aortitis of the first part of the artery which may more and more be narrowing the mouths of the nutrient arteries of the heart muscle.

In dilated and flabby and probably fatty hearts, with soft, flabby, and often irregular or intermitting pulse, digitalis does admirably if the pulse is moderately fast, and even when it is slow digitalis in small doses and combined with other tonics as iron and strychnine, and continued for a long period, is often of great value. I have had admirable results in cases of that kind with a pill I learnt many years ago, probably known to some of you. It contains gr. $\frac{1}{2}$ of powdered digitalis, sulphate of iron, and sulphate of quinine, with gr. $\frac{1}{4}$ of black pepper, and grs. 3 of extract of taraxacum; this given night and morning for a fortnight, and resumed after a week's intermission, acts admirably if you give it in time, and continue it steadily in the way I have indicated. Cases of this type are met with in persons well up in life, but are not confined to them; in one sense it might be regarded as the normal "senile heart," for that happy phrase seems only to embrace one type of heart, and leaves out the heart which at every beat threatens to break the degenerated arteries, and which is equally, if not more common in senility than the weak and flabby heart, every beat of which almost threatens to be its last.

I shall not dwell upon the value of digitalis in many cases of acute febrile disease, specially in pneumonia; nor of its value in acute alcoholism with flabby heart and pulse; nor shall I touch the treatment of cardiac dyspnoea of anginous type.

I have not attempted, nor has it been my aim, to go into the wide question of other methods of treatment. I am content if I have succeeded in submitting for your consideration points, bearing upon our ordinary measures, which may tend to formulate somewhat of our knowledge on the indications for the use of these, and some of the conditions which limit their utility, and may even make their administration hurtful.

AN ADDRESS

ON THE

DIAGNOSIS OF FUNCTIONAL FROM ORGANIC DISEASE OF THE NERVOUS SYSTEM.¹

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THE TERMS "FUNCTIONAL" AND "ORGANIC."

THE subject that I have the honour to bring before you is one of the most interesting as well as one of the most difficult problems in medicine. At the very outset one is beset with the difficulty of giving any definition or limitation to the words "functional" and "organic." These as commonly used are pathological terms, but can we say that there are any salient anatomical features which are present in the one and absent in the other condition? Is organic disease of the nervous system characterised by the presence of some definite structural change and functional disease by the absence of such structural change? The general understanding with regard to the latter is that it is a disease in which the nervous system is free from any visible change. But visibility is a relative term; it depends on our eyesight, on the power of our microscopes, and to some extent on the use of staining reagents, and also—let it not be forgotten—on the thoroughness of our search.

Cases of hemiplegia, of bulbar paralysis, and many other nervous diseases in which structural changes are usually found are sometimes met with, quite apart from hysteria, where no visible alteration can be discovered in the nervous system even after a most exhaustive examination. Are such cases to be classed as examples of functional disease? Visibility in the usual meaning of the word cannot then be regarded as a distinctive sign between the two classes. Moreover, we must not forget that even where there is a gross lesion in the nervous system, such as a tumour or meningitis, the manifest symptoms are the expression of minute, probably chemical changes in nerve tissue. For example, the

convulsions produced by a worm in the intestines or by cerebral meningitis are frequently identical, and both depend on irritation of cortical cells; in the former case the irritation is of reflex origin, in the latter it is direct. The change in the cells, or in their nuclei which gives rise to the convulsions, may be the same in both cases.

These examples suggest that duration may be a test sign between functional and organic disease, but here again our search for a boundary line is thwarted, for the question naturally arises, may not definite visible structural changes begin in the brain, cord, or nerves, and, after varying degrees of development, be arrested in their progress, and ultimately completely removed? Multiple peripheral neuritis started by alcohol, lead, or other poison is a noteworthy instance. The evidence of neuritis may be profound, as shown by marked atrophic paralysis of groups of muscles and by considerable anæsthesia; the paralysis, indeed, may be almost universal and yet the patient may in time completely recover. And it is reasonable to believe that the nerves and their sheaths will at last attain a normal condition.

Furthermore, there is satisfactory evidence that gross lesions in the central nervous system become arrested in their growth and ultimately dispersed, and the patient thought to be the subject of incurable disease has made a good recovery. This statement is adequately illustrated by a reference to tumours of the brain and to locomotor ataxy. Many of us have seen cases which ended in recovery, where the presence of headache, optic neuritis, and paralytic symptoms had convinced us that a cerebral tumour was present. I can recall one such case in which the skull was trephined but no tumour found, and yet the patient gradually lost her paralytic symptoms and her headache, and from a condition of nearly absolute blindness was restored to fair though not perfect vision. Moreover, it is a common experience in making necropsies on children's brains to find caseous masses which shell out of the convolutions, and which during life must have been practically harmless. As to locomotor ataxy, cases might be mentioned where absence of knee-jerks, the presence of pains and other signs of sensory disturbance in association with slight inco-ordination and sluggish pupils have entirely passed away. One has seen, too, more advanced cases in which the most extreme ataxia of the lower limbs has been replaced by inco-ordination of much less intensity.

To my mind, the terms "functional" and "organic" as at present employed are singularly unsatisfactory. Even in a passing emotion, such as terror or anger, some change must occur in the brain—we know not whether it is merely a disturbance in the supply of blood to certain parts or whether it is a chemical change affecting for the time being the atoms and molecules of which nerve tissue is composed. Some such changes must take place at the onset of every "organic" disease and the gradations between the changes which constitute the basis of a so-called "functional" disease and those which form the anatomical substratum of so-called "organic" lesions must be infinite and the transition from one to the other imperceptible. With improvements in our methods of examining nerve tissue, the class of "functional disorders" will get smaller and smaller, and in the present state of our knowledge it would conduce to clearness if nervous diseases were simply divided into two great groups, namely: Group I, nervous disorders with known anatomical basis; Group II, nervous disorders without known anatomical basis. In my further remarks, however, I must use for the sake of convenience the terms "functional" and "organic" in their usually understood sense.

Let us now turn to the clinical side of the question and endeavour to illustrate the methods that should guide us in making a diagnosis between functional and organic disease of the nervous system.

It is a commonplace observation, and yet one ever to be borne in mind, that the first thing to do, after listening to an account of the ailment as given by the patient or his friends, is to make a thorough examination of the patient by every means at our disposal, and then having considered the history and the symptoms of the case, to try to determine, first, what part of the nervous system is affected; and, secondly, what is the nature of the affection. Is there merely a change similar to that produced by a violent emotion, and therefore

¹ Read at a meeting of the Wigan Medical Society.

likely to be temporary, or is there some definite visible structural change?

HYSTERICAL HEMIPLEGIA.

Hysterical hemiplegia may follow a violent emotion, and is common after railway accidents, but it bears no relation to the severity or site of any injury received. The onset is usually gradual, and the left side is more often affected than the right. On examining a typical case we find that the left arm and the left leg are weaker than their fellows, but by no means powerless. Thus the patient can walk and can move the various parts of the upper limb, though in a more or less feeble way. There is complete anaesthesia of the left side, involving head, face, trunk, and limbs. Every form of cutaneous sensibility, as well as common sensation in the accessible mucous membranes, muscles, bones, and other deep structures, is more or less completely lost, and even the muscular sense is often impaired or abolished. The special senses are likewise implicated; the senses of taste and smell are generally lost on the affected side, while hearing on that side is only impaired, and the affection of sight consists of a restriction of the fields of vision: for form, and especially for colour, affecting to some extent both eyes, but most markedly the eye on the anaesthetic side. Obviously these symptoms depend on some change in the right side of the brain.

If we examine the paralysed side carefully, we shall find that the face and the trunk muscles are spared. In organic hemiplegia the facial muscles, especially the lower ones, are weak on the paralysed side, and weakness of the thoracic muscles may also usually be detected by grasping the sides of the chest while the patient takes a deep breath. It is clear, then, that the whole of the right motor path is not involved, and, on a little reflection, it appears likely that the cortex is affected rather than the internal capsule, or any part of the motor path below this; for in the internal capsule all the motor fibres which proceed to the opposite side of the body are aggregated together in a very small area, whereas they diverge widely as they approach the cortex to end in the large nerve cells, which constitute the motor centres.

We assume, then, that cortical cells which preside over the movements of the left limbs are in an abnormal condition. As to the loss of sensation, cases of gross lesion teach us that hemianæsthesia may be produced either by extensive disease of the parietal region of the cortex, or by a lesion implicating the posterior part of the internal capsule. The latter is the more common site, and its destruction also leads to impairment of the special senses. The visual defect is an important test, for it differs in the two cases. When, for example, the posterior third of the right internal capsule is implicated, the patient suffers from left hemianopsia, that is, when looking straight before him he cannot see objects situated to his left. It is true that hemianopsia also results from lesions affecting the cortex of the occipital lobe, but when sight is damaged by lesions in front of the occipital lobe, as when the marginal and angular convolutions are implicated, the defect is identical with that which occurs as already mentioned in hysteria, that is, the patient can see objects, and only those which are situated in the centre of the field of vision. What is the nature of the lesion of the cortex of the right hemisphere in the case discussed? I have already said that all the symptoms mentioned, namely—hemiparesis, hemianæsthesia, loss of smell and taste, impaired hearing, and concentric restriction of the fields of vision—may result from severe cortical disease; thus I remember one case in which extensive softening of the cortex appeared to account for the symptoms enumerated. But such cases are very rare, and, as a rule, the patient is very ill, he lies for the most part in a state of stupor, and his condition is obviously a serious one.

Now, whatever the nature of the cortical affection in hysteria may be, it does not strike the observer as a dangerous one, for the patient, although often excitable, apathetic, or otherwise abnormal in manner, is usually able to converse rationally; moreover, he is not confined to bed. His complaints may be various, relating to certain head symptoms or to queer sensations down the left side, but very often he is quite unaware of the presence or of the profoundness of his anaesthesia. To make the picture complete it is necessary to add that perhaps suddenly one day all his symptoms pass away, and a careful examination reveals nothing

abnormal. But we cannot say when that day will come; the anaesthesia and paralysis may disappear in a few days, or in exceptional cases may persist during the greater part of life. Moreover, when the symptoms have gone we cannot assert that the nervous system is healthy. The after-histories of these hysterical hemianæsthetics is not sufficiently known, and we are not able to say with confidence whether they are more liable than healthy individuals or not to be attacked in the future by some affection of the nervous system. But again we ask what is the nature of the cortical change? It is impossible to say; we might as well try to explain the changes that underlie memory or the process of reasoning. One thing is certain—the matter is infinitely more complicated than some writers appear to think; and with Professor Janet we believe that it is erroneous to assume arrest, exhaustion, or functional insufficiency of the cortical centres as adequate explanations of these hysterical phenomena.

HYSTERICAL AND ORGANIC PARALYSIS.

I have briefly analysed this familiar type of hysterical paralysis in order to illustrate the method of procedure which I think ought always to be pursued in examining a nervous case. I have omitted to mention many symptoms, such as the condition of the reflexes, peculiarities as regards gait and contractures, because I did not wish to burden the description with details which had no direct bearing on the main object of the inquiry—namely, to ascertain the site of the lesion. This is the first question to be solved, and the importance of first directing our attention when investigating nervous disease to finding out what part of the nervous system is affected can scarcely be exaggerated. For hysterical manifestations not only simulate symptoms produced by organic disease, but are frequently found in association with them. When we find that an emotion is sufficient in some persons to set up hysteria, we can hardly be surprised to find that a softening or sclerosis of nerve tissue, or the development of a tumour or other lesion may initiate an outbreak of hysterical phenomena. If, then, our attention becomes concentrated on symptoms due to hysteria, we run a risk of ignoring others, a careful investigation of which would make us suspect the commencement of organic disease. That the risk is a real one is seen almost every day both in hospital and in private practice. There is, I think, at the present time far too great a tendency to diagnose forms of paralysis that do not conform to recognised types of organic disease as hysterical.

Judging from my own experience I should be inclined to say that while it is common enough to meet with hysterical manifestations in association with and frequently overshadowing symptoms indicating organic disease, it is not common to see cases of paralysis due to hysteria and to nothing else. Over and over again one has seen paralyses diagnosed as hysterical which have turned out to be symptomatic of grave organic disease. This is perhaps particularly the case with regard to paralysis affecting the lower limbs. Hence the necessity of making a complete examination. This is attained by careful observations with regard to the degree and distribution of the motor weakness and the condition of the affected muscles as to nutrition and tone.

A consideration of the nutrition and tonicity of the muscles enables us to separate the large majority of cases of paralysis into two classes. In the one class—spastic paralysis—weakness is combined with rigidity of muscular tissue, and the lesion is situated in some portion of the upper segment of the motor tract. In the other class—atrophic paralysis—weakness is combined with wasting of muscular tissue, and the lesion is situated in some portion of the lower segment of the motor tract. A third class is constituted by the remaining and comparatively few cases of flaccid paralysis, in which there is weakness but no perceptible wasting or rigidity of muscular tissue. The deep reflexes are exaggerated in spastic paralysis, diminished or abolished in atrophic paralysis, but may be quite normal in flaccid paralysis. Can hysteria give rise to each of those forms, and, if so, how is the diagnosis made? Continental writers record cases of hysterical paralysis which closely simulate cases of lateral sclerosis, chronic anterior poliomyelitis, and every variety of

paralysis known to depend on visible lesions. Their arguments in favour of such a diagnosis are largely based on the coexistence of hysterical stigmata, such as convulsive seizures, emotional disturbances, hyperæsthetic areas, etc. Such symptoms undoubtedly are often found in association with paralysis, but they do not necessarily prove that the paralysis is hysterical. That may depend on an organic lesion which has lit up a previous tendency to hysteria. We cannot be too careful to avoid being biased by the presence of hysterical manifestations, and, as I have already indicated, our first object should be to try to make out from an investigation of the paralysis itself what part of the nervous system is likely to be at fault. If we can do this, then we are justified in discussing the probable nature of the lesion.

In order to limit the discussion I will exclude cases of atrophic paralysis, and confine my remarks to the two other groups of paralysis. Now, whether the paralysis is flaccid or is associated with rigidity or contractures, two points require to be especially observed. Does paralysis or spasm affect certain groups of muscles as the flexors or extensors of a joint, or does it only affect some complex movement such as walking, all other movements being performed with normal strength? We must also observe whether the degree of paralysis or spasm varies in intensity from time to time, as tested by the force shown in resisting passive movements. If, for example, we find that a patient while able to move the legs in bed with normal power and co-ordination is unable to stand, or that he can hop or jump although unable to walk, we infer that a complex co-ordinated movement is paralysed rather than a group of muscles, and conclude that the cortex of the brain is more likely to be the part affected than the spinal cord or any other part of the nervous system. Then if there are no other symptoms, or if other symptoms are present, and these are manifestly hysterical and cannot possibly be assigned to any known organic lesion, we may feel pretty certain that the affection of the cortical cells is hysterical in nature, whatever that may be.

But there are other cases of paralysis in which the diagnosis is more difficult. For example, one leg is weaker than the other; the weakness affects principally the flexors of the ankle, so that the foot is dropped a little in walking. The knee-jerk is slightly more irritable on the affected side. But the evidence of both weakness and increased tonicity of the muscles is slight, and the patient is distinctly hysterical. Can we admit the possibility of such a condition of limb being due to hysteria, or is it an early indication of a patch of commencing myelitis or other lesion in one pyramidal tract? The answer is that the condition may be due to either hysteria or organic disease, but that, as a rule in such a case, there is some feature as regards either the position of the foot or the question of variations in the power of ankle flexion which enables us to form a correct opinion. The diagnosis is well illustrated by the two following cases:

CASE I.—A lady, aged 26, consulted me early in June, 1893. She stated that two months before she began to have numb pricking sensations all down the left side and the left leg. A little while afterwards she experienced a cold clammy feeling below the left breast, and after that had passed away she used to suffer from a peculiar dragging sensation in the left groin, and she felt as if her clothes were disarranged about the groin. About a month later she noticed one day that the toes of the left foot caught in walking, and on the next day she could hardly use the foot. This weakness lasted for about four months, and then became much less. Since Christmas, 1892, she had had "attacks" consisting of queer sensations down the left side, and weakness of the left foot nearly every fortnight, the attacks lasting about a week and then getting better. These symptoms had developed shortly after the shock she had had by finding the dead body of her brother who had committed suicide. She said that she had no pain in the leg, and, on examination, no disturbance of sensibility could be detected. The patient had the hysterical manner, but objective symptoms were limited to the left leg, and were purely motor.

All the movements of the limb at hip, knee, and ankle were slightly, but only slightly, weaker than those of the right leg, flexion of the ankle being more affected than other movements. But the posture of the left foot in walking was very peculiar. It was markedly dropped and slightly inverted, so that on standing behind her as she walked one could see the sole and the outer border of the foot. The outer side of the boot at the level of and behind the little toe region was much worn, and she told me that she had worn a hole in the outer side of a house shoe in a fortnight. The knee-jerks were increased, and the wrist and elbow-jerks also lively. There was no ankle clonus. The plantar reflex was diminished on the left foot as compared with that of the right foot. She went to Llandudno for six weeks, and under the influence of salt water baths and massage made a complete recovery; so that at the middle of August I was unable to find anything wrong with the left leg, and the left foot was normal in every respect.

The curious attitude of the foot in walking, which was

much less conspicuous when she sat in a chair, and which could not be adequately accounted for by the degree of weakness present and the absence of detectable contracture, led me to diagnose hysteria as the cause. I have not seen or heard from her since, so that one cannot be absolutely certain that the attack of paralysis was purely hysterical in origin; for, as insisted on by Dr. Buzzard, loss of power in a limb and recovery therefrom is very common during the development of disseminated sclerosis, and he records cases in which the apparent recovery lasted for four or five years, after which typical symptoms of the disease began to be manifest. This apparent recovery in disseminated sclerosis is illustrated by the following case, which also shows how difficult it may be to diagnose the presence of this disease in its early stages.

CASE II.—The patient, a woman, aged 33 years, was sent to me early in June, 1892, by Dr. G. H. Milnes, of Derby, who kindly gave me the following account:

She first consulted Dr. Milnes on April 27th for a gradually increasing lameness of the left leg, which was first noticed four years ago. Ten years ago her left arm was weak and somewhat numb, but these symptoms passed away in a few weeks. The lameness only comes on when she is tired or excited. At first she only noticed it after a walk of seven miles; now it comes on after a walk of less than half a mile, and the left toes sometimes catch the ground and cause her to stumble. For the last seven years patient had been dressmaking. Previously she was behind the counter from the age of 14, and had to endure much standing and long hours. She had scarlet fever when 4 years old, but has had no other acute illness. There is no history of syphilis, and no family history of nervous diseases. Her father is a martyr to gout; her mother died in childhood thirty-one years ago. Two years ago patient was much troubled with constipation, for which she had to take strong purgatives, and her bowels became very irritable, and sometimes she was unable to restrain the motions. Dr. Milnes found that the left foot and leg were colder than the right ones, that the knee-jerks were about normal, the left, however, being probably a little the more irritable of the two, and he thought that there was a tendency to left ankle clonus. Sensation was normal, and there were no other symptoms of disease with the exception of some deep-seated tenderness and a sense of resistance in the left iliac fossa. The latter symptoms were not removed by the administration of purgatives, and Dr. Milnes was inclined to suspect some source of pressure within the pelvis, which might possibly explain the condition of the left leg. I saw her for the first time in June. She was a healthy-looking woman, and complained of the weakness of the left leg. I found that all the movements of the limb were weaker than those of the right leg, flexion of the ankle being the most affected, but even this movement was only slightly impaired. The cutaneous sensibility of the left leg was very slightly impaired as compared with that of the right leg, the difference being most distinct on the outer side of the leg and on the sole of the foot, but even in these situations the impairment was slight. Both knee-jerks were moderately exaggerated, but I could not elicit ankle clonus. The plantar reflex was absent on the left side, doubtfully present on the right side. No other symptoms could be detected. Her manner was somewhat hysterical, and this together with the loss of the plantar reflex, made me inclined to regard the case as hysterical. Her condition remained much the same till October, when it was noticed that the left hand was weak and the left thenar eminence slightly wasted. There was now paralysis of the flexors of the left ankle and double ankle clonus. Disseminated sclerosis was suspected for the first time, although the typical symptoms were absent. There was no tremor, no nystagmus, and no disorder of speech. A note made on February 23rd, 1893, says that the weakness and wasting of the left hand are more marked than formerly; that the plantar reflex is absent on both sides, and that (curiously enough) ankle clonus, which was pronounced at the last examination, cannot now be properly obtained. In June, 1893, the right leg became affected somewhat suddenly, and gradually got weaker; a little later the functions of the bladder and rectum became affected, and every now and then the excreta were passed involuntarily. On May 24th, 1894, the following notes were taken: Patient is unable to stand or walk alone; with assistance she walks with a spastic gait, and scrapes her toes on the ground. She cannot raise her left leg from the bed or dorsiflex the left foot; she can slightly bend the right knee and hip, but not the right ankle. No atrophy of the muscles of the leg. Passive movement reveals the existence of a moderate degree of rigidity in both legs. Both knee-jerks are increased and ankle clonus is well marked on both sides. The plantar reflexes are present, but the abdominal and epigastric cannot be elicited. Cutaneous sensibility is not impaired, and patient is free from pain. Motor power in the arms is fairly good, but the grasp of the left hand is weaker than that of the right. The bladder is distended, and there is dribbling of urine and no control over the motions. Speech is normal, and neither tremor nor nystagmus can be detected. On June 4th, the patient lapsed into a semicomatose condition, and the following morning was quite unconscious. The temperature rose to 101.5°, the pulse quickened to 176, the face was flushed and perspiring, the conjunctiva injected, and there was conjugate deviation of the eyes to the right, together with retraction of the hand and twitchings of the limbs. The respirations, which were regular and 40 to the minute early in the coma, assumed later the Cheyne-Stokes type, and she died during the evening of June 6th. Post-mortem examination revealed well-marked patches of sclerosis, scattered through the brain and spinal cord, and also septic cystitis and pyelo-nephritis. A careful microscopic examination was made of the nervous system by Dr. Arnold Edward, full details of which will be found in his thesis on Disseminated Sclerosis.

With regard to the differential diagnosis of such a case in the early stages, Dr. Buzzard lays stress, as I have already mentioned, on the recovery from previous paralysis (witness the account of the left arm in the case related), and on a

shifting of powerlessness from one side to the other which he says rarely occurs in hysteria. He also regards loss of the plantar reflex as a sign in favour of hysteria. This is undoubtedly true, but this reflex could not be obtained in my case till towards the termination of the disease. In disseminated sclerosis, pallor or atrophy of the optic disc is not uncommon, but in hysteria the characteristic affection of vision is blindness on one side, the optic discs being perfectly normal.

It may be thought that I have been too anxious to point out the possibilities of mistaking organic disease for hysteria, and have ignored the converse error of diagnosing organic disease when hysteria only is present. I should be sorry to convey such an impression because one mistake is as bad as the other, and it is certainly pleasanter for all parties concerned to have the case diagnosed as one of hysteria than as one due to the presence of organic disease. But I particularly wish to lay stress on one point, and that is that, having decided that a patient is suffering from hysteria, do not let us close our minds to the possibility of future developments. An hysterical person is just as liable as, perhaps more liable than, other persons to become the subject of organic disease; also we must admit the possibility of having misinterpreted some of the symptoms; and further, the manifestations, although purely hysterical, may have been started by minute yet definite structural changes, which ere long will lead to symptoms of serious augury. Hence let subsequent examination be as thorough as the first examination, and quite unbiassed by our first diagnosis of hysteria. Only in this way can we avoid the danger of omitting to recognise signs of serious disease.

I shall never forget the case of a lady whom I attended about twelve years ago. She was a stout, robust-looking woman, of about 50 years of age. She complained of an aching pain in the back and of creeping sensations in the lower limbs and of being easily tired by short walks. Her manner was distinctly hysterical and she was very loquacious, giving graphic details of all her symptoms. A careful examination revealed nothing abnormal. There was no paralysis and no alteration in cutaneous sensibility, and the knee-jerks, though irritable, could scarcely be said to be pathologically exaggerated. I looked on the case as hysterical, and prescribed massage and tonic treatment. As there was not much improvement after a fortnight's treatment, I asked an eminent physician to see her with me. He also made a thorough examination of her case, and agreed with me that there were no signs of organic disease, and that in all probability the symptoms were purely hysterical. After the consultation I visited her from time to time, but, having now a strong bias in favour of hysteria, I did not again make a thorough examination. About ten days later I received a message to come at once, for the patient was paralysed. I found that she was suffering from complete paraplegia, and from other symptoms indicating an acute myelitis in the upper dorsal portion of the spinal cord, and she died within the week. Such an experience as that is not easily forgotten.

There is the same necessity for caution in regard to the diagnosis of what we call neurasthenia; for example, it is by no means always easy to distinguish between neurasthenia and the early stage of general paralysis, and I could well illustrate this by notes of cases from my own practice.

Again, some of the chief symptoms met with in neurasthenia—headache, vertigo, mental incapacity, and feelings of exhaustion—may constitute the premonitory symptoms of a brain tumour. Hence once more we must recognise the necessity of admitting the possibility of commencing organic disease every time we examine a patient whose case at the first investigation was diagnosed neurasthenia. Between the years 1880 and 1884 my judgment was much exercised with regard to a boy who suffered from attacks of severe headache and dizziness. He was a pale delicate-looking lad, highly sensitive, who had always shown a marked tendency to make the most of his ailment. There was a history of tubercle in the family. The headache at times was so constant and the boy looked so very ill that the question of brain tumour was often discussed. Again and again his optic discs were examined, and the condition of his nervous system was carefully investigated and by more than one physician, but always

with negative results, and the diagnosis of neurasthenia seemed to be the only possible one under the circumstances. He is now 22 years of age, and nearly blind from atrophy of his optic nerves, the result of neuritis which first declared itself about two years ago by the presence of bilateral temporal hemianopsia. The case is still obscure, inasmuch as there are no definite paralytic symptoms, but there are reasons for believing that a tumour of exceptionally slow growth exists at the base of the brain, and that it is pressing on the middle of the optic chiasma. If there is a tumour, has it existed for sixteen years, and was it responsible for the early symptoms, or did its development commence only two or three years ago, its presence and the symptoms of neurasthenia which still exist having no relation to one another?

ON THE IMPORTANCE OF THE EXAMINATION OF THE ABDOMEN DURING THE PUERPERIUM,

WITH SPECIAL REFERENCE TO THE INVOLUTION OF THE PUERPERAL UTERUS.

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THE process of involution in the human uterus is of great interest to the physiologist, owing to the amount of material absorbed in a short space of time. The exact causation has not yet been definitely ascertained, although microscopists have offered many theories. It is not, however, the purpose of this paper to deal with the intimate changes occurring in the uterine tissue, but to determine the period when the uterus has returned to such a size that the puerperal woman may without risk, *ceteris paribus*, assume the erect posture. For this purpose two methods of investigation have been used, namely, external and internal measurement:

(a) *External Measurement*—where the distance between the fundus uteri and the symphysis pubis is measured, together with the breadth from side to side at the widest part.

(b) *Internal Measurement*—by means of the uterine sound to determine the length of the cavum uteri.

To the first method few objections can be raised, but to the second the dangers of infection must always retard such a procedure, more especially where a series of observations can be made, for example, in maternity hospitals. The observations recorded in this paper were carried out by me six years ago. At that time I was unaware of the work of Serdukoff,¹ and it was not until the termination of my work that I discovered the existence of his paper. Serdukoff employed external measurement, giving his reasons against the internal method as follows:

1. From the curvature of the sound being always the same, and from the fact that we have a varying axis of the uterus corresponding to the curvature of the sound, it follows that we do not by this method ascertain the natural length of the uterus as it lies anteflexed or anteverted, but only an artificial length as it is straightened out.

2. The sound is not always easily introduced, its point is frequently to be felt with difficulty or not at all through the anterior abdominal wall and thick uterine wall, and you can never be certain that at each observation the point touches the same part of the fundus; accordingly we may have an apparent length which is less than the real one.

3. This method is not unaccompanied with danger, however carefully it may be employed.

To these I would add a fourth:

4. The diminution of the uterine cavity is not proportionate to the diminution of the uterine tissue, being much more rapid. This I have proved by examination of puerperal uteri *post mortem*.

It is clear then that the most accurate results would be obtained by combining the two methods. However, for all practical purposes the external measurement is sufficient, and on this account my observations will be confined to it.

Method.—In a series of patients the measurement of the distance between the upper border of the symphysis pubis and the centre of the fundus uteri was taken by a tape measure drawn tight. These measurements were obtained daily at the same hour, due attention being paid to the condition of the bowels and bladder. Cases in which there was evidence of septic trouble were excluded.

Reference to works on obstetrics shows that the date when

¹ *Obstetrical Journal*, vol. iii. p. 477. The paper was communicated to the Obstetrical Society of Edinburgh by the late Dr. Matthews Duncan, February 10th, 1875.