nical congestion. I cannot but think that the damage to the cord would often be lessened, were the early treatment of these cases conducted on the principles which we apply to acute inflammation elsewhere.

Should not the internal treatment in the early stage be rather that for the morbid process than for its seat; be that for an internal inflammation rather than for a disease of the spinal cord, and therefore consist of aperients, diuretics, and agents which act on the vascular system, rather than nerve remedies? During the stage of recovery, there seems no reason to doubt that nervine tonics do some good; and on the value of electricity and skilled rubbing there is a fair agreement of opinion. When the disease was regarded as purely muscular, it was thought that electricity actually cured it in some cases. The proof that the seat of the disease is the spinal cord renders this opinion extremely improbable. Beyond doubt, the application of the voltaic or constant current to the muscles (to which their fibres will still respond, although insensitive to faradisation) improves their nutrition, lessens their wasting, and keeps them in a better condition to respond to any nerve-power over them which may ultimately be regained by the recovery of damaged cells and regeneration of degenerated fibres. Thus the ultimate state of the patient is better than it would be without electricity. It is desirable, therefore, to use such a current as shall cause slight visible contraction in the muscles. Unfortunately, children are often so much frightened by even a very weak application, that the needful strength cannot be employed without causing an amount of emotional disturbance, which may not unnaturally be regarded as doing the child more harm than the electricity does good. Here, again, we have sometimes to be content with a compromise. A weaker current than will cause contraction has probably some influence on muscular nutrition; and I think the best rule is to employ such a strength as the child will bear without much emotional disturbance, whether we can thus get muscular contraction or not. If care be taken to avoid alarming the child at the commencement, a current of some strength can often thus be employed.

Is the influence of electricity confined to the muscles? Does it aid the recovery of the spinal cord and nerves? Some believe that it does, and urge therefore that, in treating the muscles, one pole should be placed upon the spine; and the method is, on *à priori* grounds, a reasonable one. But the tendency of the disease to spontaneous improvement renders it extremely difficult to form an opinion on this point. For myself, after a careful comparative trial of the two methods of applying electricity, from the cord to the muscles, or to the muscles only, I have been unable to observe any superior advantage in the former method, which seems, even when a large spinal electrode is employed, to disturb children more than the application to the muscles only. At the same time, beyond the increased discomfort, there is no objection to the application of one pole to the spine.

How soon should electrical treatment be commenced? I cannot think it desirable to apply the voltaic current to the spine within one month of the onset; but it may certainly be applied to the muscles, with care, much sooner, at the end of ten days or a fortnight when the "reaction of degeneration" has become distinct.

ON SOME POINTS IN THE DIFFERENTIAL DIAGNOSIS OF INTRACRANIAL DISEASE, GENERAL PARALYSIS OF THE INSANE, AND TABES DORSALIS.

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PATHOLOGICAL and clinical experience during the past ten years, in reference to nervous disease, have convinced me that, in the pathology of the general paralysis of the insane, and in the pathology of tabes dorsalis, we have an association of changes which are still open to a vast deal of investigation.

The relationship of spinal to cerebral lesions in the general paralysis of the insane has been noted for many years; but I have failed to discover any sound pathological deductions as to why the two conditions should coexist to the extent which is invariably found. We are greatly indebted to the valuable labours of Westphal, Voisin, and Mickle, for much information upon this subject. To M. Pierret we are also indebted for bringing before the profession the head-symptoms in tabes, and the important part which the trigeminal nerve takes, by its reflex influence, in the production of certain transitory pareses of the muscles of the eye, of the face, and of mastication; and also the head-pains, which are sometimes met with, but which must not be confounded with the chronic inflammatory changes of the brain

which are found in the course of the general paralysis of the insane. I have seen such cases relegated to the class of tabes, when one might as fairly have classed them with cases of gross intracranial disease, or even of general paralysis, and where, in fact, the tabes was merely a manifestation of a more or less diffuse process of degeneration, extending over the entire nervous system, although the objective signs were by comparison more marked in the posterior columns of the cord. Erb says: "Naturally, in all such cases, the importance of the spinal becomes subordinate to that of the cerebral disease." I must admit that I have had several cases under my care, where, in the initial stage (and this is extremely important from a prognostic point of view), I have been unable to give a decided opinion as to whether my patient would remain tabetic, or whether he would suddenly merge into the insane paralytic. I will give an example.

Some time ago, I was called in consultation, by my friend Mr. Willis, of Hornsey Lane, to see a patient who might fairly be said to be suffering from tabes dorsalis. He had the signs, subjective and objective, of this disease: the flying lightning-like pains, which he called rheumatic; the dysæsthesia and anæsthesia of the lower limbs; absence of knee-reflex; the Argyle-Robertson pupil; and inability to maintain the equilibrium of the body with the eyes closed; and, in addition to these, he suffered from M. Pierret's head-symptoms—namely, pains in the head involving the regions supplied by the trigeminal nerve; but there is this noteworthy fact to remember, that, during these attacks of pain, the temperature of the head was always increased. Again, his *morale* had changed, and this is not common to tabes; for, instead of being as he was formerly, an even-tempered man, he had become liable to attacks of uncontrollable excitement, and during these attacks the articulation became so markedly defective that he could scarcely be understood; but at ordinary times there was no tremor either of the tongue or facial muscles. He was a strongly built, florid-complexioned, muscular man, aged 32, without any nervous history.

I will now describe another case which has a direct bearing upon the subject. John H., aged 48, came to consult me, at the Hospital for Epilepsy and Paralysis, on December 20th, 1880. He was a florid-complexioned, strongly built, muscular man. He had suffered from shooting lightning-like pains about the body for the past ten or twelve years; they affected chiefly the anterior and the inner surfaces of the thighs and legs; they would last for five minutes, or for six, twelve, or twenty-four hours. He said these pains were not so bad as they used to be. He thought "they had given place to weakness". At times, he had burning pains of the same character in the penis, which migrated to the rectum; and these were accompanied with great desire both to urinate and to defæcate. His mental powers had failed, so that he was unable to carry on his business. When any person was talking to him, he not only forgot what he was talking about, but forgot the presence of the person to whom he was speaking. For some time, he had been unable to preserve his equilibrium, and he would roll about in walking, and his friends would accuse him of being drunk. When at the theatre, he became very emotional, and was readily excited to cry or laugh, according to the nature of the performance. Upon examining the cranial nerves, the following signs were noted. The first nerve was normal; as to the second nerve (the examination was made by Mr. Mackinlay), the discs were pale, the left one most decidedly so; the arteries small. There was no retinal change, or change elsewhere in the fundus of the eye. Right eye, vision = $\frac{2}{3}$, and C. $\frac{2}{3}$ $\frac{2}{3}$; left eye, vision $\frac{2}{3}$, and C. $\frac{2}{3}$ $\frac{2}{3}$. With the best eye, he could read Jäger 1 at a foot without glasses; the pupils were unusually small. There was no marked limitation of the field of vision in either eye; the pupils did, however, contract very slowly and sluggishly when exposed to light. The third, fourth, and sixth nerves were normal. There was marked anæsthesia and analgesia throughout the range of the fifth nerve. The lateral movements of the jaw were performed with some slowness and difficulty. He was unable to whistle or send forth a full current of air. When he talked, the mouth became filled with froth. The labials were frequently reduplicated: he pronounced "Papa's performing pony" in this way, "Pap-p-p-a perf-f-f-f-forming pony"; and, during articulation which required the exercise of labial and lingual co-ordinating power, the labial and zygomatic muscles acted without purpose. When he stood with the eyes closed, he became unsteady, and said that he felt trembling all over. The knee-reflex was scarcely appreciable. There was no ankle-clonus; no reflex of the feet. The cutaneous sensibility of the trunk was fairly normal. Muscular response was normal, both of sensation and motion, to the continuous and to the faradaic currents. There were deep-seated anæsthesia and analgesia of the lower limbs. From his own evidence, it appeared that on January 6th he completely lost his reason; and he remained in this state for a fortnight, and after this he began to improve. On July 8th, 1881, he again presented himself at the hospital,

greatly improved in every way. His memory, reasoning power, and articulation were almost perfect; there was no tremor of the lips or facial muscles, no tremor of the tongue nor overflow of saliva; yet there was the same evidence of spinal myosis, and the sharp lancinating pains continued to worry him. He had been taking large doses of the iodide of potassium with evident benefit. I need scarcely say that, in all human probability, this man will ere long become the subject of confirmed general paralysis, which was preceded by the symptoms of tabes about twelve years ago.

The following case, from a clinical and pathological point of view, is of exceptional interest, and shows how the signs of ataxy may exist, due to sclerosis of the posterior columns of the spinal cord in connection with pathological changes in the brain which are common to the general paralysis of the insane, without the patient presenting during life those objective psychical manifestations which usually are to be found in this disease. I do not assume that this case is in any way unique; but it is undoubtedly a typical connecting link in a certain chain of evidence—I mean the evidence of association between pathological changes in the brain and spinal cord of tabes on the one hand, and general paralysis of the insane on the other hand.

W. B., aged 34, was sent to consult me by Mr. Mackinlay, at the Hospital for Paralysis and Epilepsy, Regent's Park, in November 1880. There was no family history of brain-disease, neither was there any clear evidence of syphilis. He had been employed as a dock-labourer, and enjoyed excellent health until the end of 1879, when the left eyelid began to droop, and the sight of the left eye began to fail, and so did the sight of the right eye; and, after this, he saw double. In the month of July 1880, he began to suffer from severe headaches; the pain was of a shooting character, and extended from the forehead to the occiput, and sometimes from one temporal region to the other; and the pains would sometimes come on suddenly, and depart suddenly, but they were invariably associated with an increase of temperature. Soon after the pains in the head began, his gait became unsteady, and his fellow workmen accused him of being continually drunk. It is a point of interest to note that he never felt giddy during locomotion, but, when lying in bed, it seemed to him as though the bed were continually moving round and round; and, when sometimes standing upon his legs, his body revolved against his will from left to right. At times, he suffered from great stiffness of the muscles of the back of the neck, and the head would be forcibly retracted. The following notes of his actual state were made at the time of his admission into the hospital. "The cranial development is regular, and not unduly prominent in any direction. When the skull is percussed, he experiences no pain, but, some months ago, the scalp was so tender, that he could not bear it to be touched. I am indebted to Mr. Mackinlay for the following ophthalmoscopic examination. Right eye: discs white (a little bluish); punched out in appearance; no cupping; arteries very small indeed (like fine hairs); veins about normal size; choroid thin; tension normal; vision equal $\frac{2}{3}$ partly, and Jäger 4 at 8 inches (minim type). Left eye: disc dead white and punched out; veins tortuous, arteries very small indeed; tension normal; vision absolutely nil. The pupils, moderate in size, rather larger in the right eye than in the left, do not respond, even to very strong light. Slight ptosis of left lid. There are no marked objective signs of any other cranial nerves being involved. He complains of numbness at the tips of the fingers, but there are no other subjective symptoms of defective sensation. On the objective side, there is a marked retardation in the conduction of impressions from the periphery to the nervous centres, and more especially in the lower limbs, both in reference to pain and sensation. All voluntary movements are co-ordinate when he is lying down, but, when standing or walking, there is defective volitional co-ordinating power. There is complete absence of knee-reflex, and no reflex movements are excited when the soles of the feet are tickled. The sphincter muscles of the bladder and of the rectum are unaffected. There is a marked tendency to depression, and he is readily induced to cry and to laugh. Sometimes (but not often), there is defect of articulation; otherwise, there is neither tremor of the tongue nor of the facial muscles."

These signs and symptoms deserve consideration; for I maintain now, as I maintained then, that we had clear evidence of gross intracranial change, which involved the cerebellum; and I am quite willing to admit with all candour, that I even thought my patient had a tumour of the cerebellum. Dr. Buzzard, who saw the case, thought there was no question of doubt about its being a case of tabes, pure and simple; and my friend Dr. Boyd, with his vast experience and great mental acumen in all such matters, suggested "general paralysis of the insane". In about three months from this time, the man died, with no other marked signs or symptoms than those just narrated, excepting the signs of a more or less acute general paralysis, into the details of which it is not necessary to enter.

At the *post mortem* examination, which was kindly made for me by my then colleague Dr. Sturge, the following notes were made. The dura mater was much thickened, especially over the vertex and the anterior lobes. There was much meningeal congestion and diffuse chronic arachnitis, with considerable excess of fluid; adhesions existed between the membranes and the cortex. The convolutions were particularly noticeable over the frontal gyri, and the gyri of the temporo-sphenoidal lobes, as well as the ascending parietal convolutions. The grey cortex was materially altered and wasted and softened in the parts just mentioned, and the white substance of the hemispheres was excessively vascular. At the base of the brain, marked changes had taken place, but more particularly was this the case in the optic nerves; they were shrivelled and hardened, and looked something like dried gelatine; this was seen more clearly towards their ends, for, where they wound round the thalamus, the structures appeared to be normal. There was the same condition of arachnitis at the base that there was over the vertex of the brain. The cerebellum was healthy, but there were some adhesions of a slight character over its surface. The floor of the fourth ventricle was remarkably granular; and I may here mention that this condition is common in the brain of the general paralytic. The spinal cord presented slight chronic inflammatory thickening of the membranes, but no adhesions. I am indebted to my friend Dr. W. B. Kesteven for the following careful microscopic examination. Beginning at the conus medullaris, and going up through the spinal cord to the cervical region, there was marked sclerosis of the posterior columns, extending slightly into the lateral columns. Between the posterior cornua, the columns had a darker aspect than in healthy cords, owing to an increase of the connective tissue and its fibrillation, which encroached upon the nerve-fibres, compressing them and their axis-cylinders, so that, in a transverse section of these portions, the translucent sheath and coloured axis-cylinders presented an opaque aspect. These portions of the cord took dyes more deeply than the healthy parts. The texture of the cornua also was much denser than in the normal state. Their cells had not undergone any alteration of size, but in many places they were slightly granular, and their nuclei appeared to me enlarged. The cells of the anterior horns were distinctly undergoing fuscous degeneration in varying degrees, as shown by their only partially taking the carmine dye. The roots of the nerves were, in many instances, to some extent atrophied. The blood-vessels were tortuous, and had become dilated by repeated distension, so that large perivascular spaces were seen in which the cut ends of vessels were observed to be thickened. In many of the smaller vessels were thrombi and emboli. These lesions were most obvious in the cervical region and in the medulla oblongata. The appearance of sclerosis decreased upwards, until it had disappeared in the medulla, where, as in the anterior horns of the cervical region, the cells seemed to be the principal seat of the morbid changes, very marked in those of the olivary bodies, and in the nuclei of the vagus. In the medulla oblongata were scattered a few colloid bodies. The central canal throughout its whole length was obliterated by nuclei and corpuscles. The fibres of the optic nerve and commissures were very much atrophied, whilst their blood-vessels were enlarged and much dilated.

Such, then, are the notes, clinical and pathological, of a case which, I hold, presents features common alike to the general paralytic and tabetic. I have no time here to analyse these especial features, but, in the following remarks, the drift of my ideas will be elucidated.

(To be continued.)

NOTES OF A CASE OF ACUTE ASCENDING PARALYSIS.

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THIS disease is one of very great rarity. In 1859, Dr. Landry described a peculiar form of paralysis, which he termed "Paralysie ascendante aiguë" (*Gaz. Hebdom.*, 1859, Nos. 30, 31); and yet not more than thirty cases are to be found recorded, and some even of these are doubtful examples of that disease. This lack of instances is the more noticeable, since the last decade is one marked in the history of medicine for the very great interest taken, and the rapid advances made, by many skilful observers in diseases of the nervous system.

Of the etiology and pathology of acute ascending paralysis, it may be truly said we have no knowledge. It seems to have no morbid anatomy. The most careful and experienced observers—such as Vulpian, Hayem, Westphal, Pelligrino, Levi, and MM. Déjérine and Goetz—