ON PERIPHERAL NEURITIS.¹

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THE subject of this discussion, which I have been asked to open, is "Peripheral Neuritis." By *neuritis* is, of course, meant inflammation of nerves, and by the qualifying term, "peripheral," I presume it is intended to limit the discussion to disease in the course of the nerves after they have passed beyond the intervertebral and cranial foramina. The subject, as thus defined, is so wide, that I will take it upon myself to limit the discussion still further. It would hardly lead to any profitable result were we, at one sitting, to take into our consideration the phenomena which attend inflammation of individual nerves, including sensory, motor, and mixed nerves, and the nerves of special sense. It seems to me, therefore, desirable to restrict the field of our discussion to that very important group of inflammation of nerves in which a larger and larger number of the trunks are successively invaded, the disease beginning at the periphery and extending towards the centre. This disease, variously called progressive multiple neuritis, polyneuritis multiplex, and other names, invades the body as a rule in a symmetrical manner as regards the two sides, although the phenomena may be more pronounced on one side than on the other. Now this is one of the chief characteristics of those diseases that are caused by a poison circulating in the blood, and there can be no doubt whatever, that in at least the majority of cases, this affection is caused by a poison. The best known disease of this group is probably lead paralysis, and there is no room for doubt that it, at any rate, is caused, directly or indirectly, by the introduction into the system of a poison from without. It is also known that arsenic, and probably copper and other metallic salts, give rise to a paralysis of the same general type as lead paralysis.

The second variety of this disease is typified by the paralysis which results from alcoholic excess. In this variety may be included the paralysis caused by the fumes of bisulphide and oxide of carbon, and probably also, the severe pains in the limbs caused by the abuse of chloroform and chloral. The same type of paralysis has also been met with in advanced cases of diabetes. The third variety of this affection results from animal poisons, and of these diphtheritic paralysis is probably the best known. The remarkable cases of universal paralysis recorded by Dr. Buzzard, as occurring in syphilitic subjects, is now acknowledged by him to belong to the group of peripheral neuritis, and these may be placed in the same class as diphtheritic paralysis. This form of paralysis occurs as a signal, not only of diphtheria and syphilis, but also of small-pox, scarlet-fever, measles, typhoid, typhus, intermittent fevers, and dengue. Pitres and Vaillard have recently brought together many cases as occurring in the course of tuberculosis, while the essential morbid changes in anæsthetic leprosy, and beriberi, are now found to be a peripheral neuritis. It is also probable that pemphigus, herpes, and other cutaneous diseases are caused by neuritis of sensory nerves.

It is very probable that peripheral neuritis is sometimes caused by the poison of rheumatism. I have myself seen paralysis of the extensors of the forearm, and loss of the patellar tendon reactions, but without manifest paralysis in the lower extremities, in a young girl who had suffered from an attack of acute rheumatism, and was at the time suffering from an attack of acute rheumatism which proved fatal. A case of peripheral paralysis is recorded by Freund, which supervened without any known cause; but the facts that the body was bathed in sweat at an early period of the disease, and that the patient was suffering at the time from endocarditis, rendered it probable that the neuritis was of peripheral origin.

The muscular feebleness and wasting, principally limited to the extensors, which invariably accompanies chronic rheumatoid arthritis, is also most probably caused by neuritis of the neighbouring nerves; and, quite recently, profound morbid changes have been discovered by Pitres and Vaillard in the nerves in such cases. In a certain number of cases, however, no assignable cause for the disease could be ascertained. The patient may have been exposed to severe degrees of cold; but there was nothing in the degree of exposure or in the family history to account for the initiation of so grave a disorder as the one under consideration. It must, therefore, be admitted in the meantime that there may be cases of idiopathic multiple neuritis.

It is quite impossible for me, in the time at my disposal, to give anything like a complete description of the manifold symptoms of the

different varieties of this disease, and consequently I shall only endeavour to seize upon the prominent features which give character to the affection, and which help to differentiate it from allied disorders.

The *motor disturbances* consist of a more or less widely distributed paralysis, which, we shall soon see, attacks by preference certain groups of muscles. I have made inquiries of patients to ascertain whether the paralysis was preceded by active spasm; and some of them, especially alcoholic subjects, asserted that they had suffered greatly from "cramps" in the legs; but, on cross-examination, it generally turned out that the so-called "cramps" are more likely to have been severe shooting pains from irritation of sensory nerves. On the whole, I never could assure myself that the paralysis was in any case preceded by active spasms in the affected muscles.

With regard to the *nature* of the paralysis in neuritis, it is always of the atrophic variety. The muscular masses emaciate and soften, undergo rapid wasting, while they are soft and flaccid, and no tension is provoked in them by passive movements of the limb. The cutaneous reflexes are very variable. The reflex of the sole is generally absent when the lower extremities are implicated, as in alcoholic paralysis; but the cremasteric and abdominal reflexes are generally preserved, and are often said to have been exaggerated. In a case recorded by Fischer, the reflex of the sole was present, but it was delayed in transmission. The mechanical irritability of the muscles is also often said to have been exaggerated, while it is lost in some cases, probably in all cases of advanced atrophy. Probably the most generally valuable test of the nature of the paralysis is to be found in the state of the tendon-reactions, and especially of the reaction of the patellar tendon when the lower extremities are attacked. In all the recent records of cases of alcoholic, diphtheritic, and other forms of peripheral neuritis affecting the lower extremities, the patellar tendon reaction has been examined, and found absent, with a few notable exceptions which deserve special consideration. In a case of alcoholic paralysis recorded by Dr. Buzzard in his recent valuable work on *Peripheral Neuritis*, tickling of the soles of the feet is said to have given rise to "very extensive reflexes," and the patellar tendon reactions were at first "notably exaggerated;" and a similar exaggeration of both these reactions was observed in a case of alcoholic paralysis recorded by Dettinger. In the case of rheumatic peripheral multiple neuritis recorded by Freund, exaggeration of the patellar tendon reactions was also observed; and a blow on the tendon was followed by tremors of the limb, which increased in intensity for about a quarter of a minute, and then ceased. These reactions were, however, lost a few days before the termination of the case, which ended fatally. In one of Oppenheim's cases of alcoholic paralysis, in which the patient made a complete recovery in three months, the knee-jerks were present at first; the reaction became feebler and feebler, and finally disappeared, first on the right, then on the left side; but it reappeared on both sides in the course of a few days.

In lead-poisoning it is not uncommon to find that the knee-jerks are absent, even in cases in which the muscles of the lower extremities are not appreciably involved in paralysis. If my memory serves me well, I think I remember that Dr. Broadbent remarked at one of our medical meetings in Manchester, upon the occasional absence of the knee-jerks in cases of lead-poisoning, in which the paralysis was strictly limited to the extensors of the forearm, and I was then able, from my own observations, to confirm the statement. Since that time, however, I have met with several cases of double-wrist drop, from lead-poisoning, in which the patellar tendon reactions were very lively. In a case of extensively distributed paralysis from lead-poisoning, recently under the care of my colleague, Dr. Morgan—who, with his usual kindness and consideration, has given me permission to mention the case—the patellar tendon reactions were greatly exaggerated. In addition to a widely diffused paralysis, involving all the muscles of the upper extremities, this patient is the subject of a double facial paralysis, which involves the upper as well as the lower branches of both nerves. In the lower extremities, the common extensors of the toes and the peroneal group are completely paralysed, while the tibialis anticus, the muscles of the calf, and those of the thighs, are relatively spared.

Tickling of the sole of the foot gives rise to repeated contractions of the tibialis anticus muscle, and to extensively distributed contractions of the muscles of the thighs. The mechanical irritability of the quadriceps muscle is increased, and, as already stated, the patellar tendon reactions are very decidedly exaggerated. Probably the most remarkable part of the case is that, on electrical examination, the quadriceps femoris muscles manifest the partial reaction of degeneration, showing that the anterior crural nerves are slightly implicated in the disease. In this case there are no sensory phenomena, and not the slightest grounds for suspecting disease of the spinal cord or brain.

¹ An Introduction to a Discussion in the Section of Pathology at the Annual Meeting of the British Medical Association at Brighton.

The patient is a painter by trade ; he has suffered from colic ; has had a previous attack of paralysis of the extensors of the arms, from which he recovered, and has a well marked blue line on the gums. I mention these cases to show that although absence of the patellar tendon reaction is a most valuable sign of peripheral neuritis, yet the evidence it affords is not absolute, and can only be relied upon when taken in conjunction with the other symptoms, and all the circumstances of the case.

This remark applies with equal force to the electrical examination. In by far the majority of cases, the electrical test affords conclusive evidence of the nature of the paralysis. In aggravated cases, the affected nerves and muscles manifest a complete loss of faradic irritability, and the qualitative changes of the galvanic current, which constitute the reaction of degeneration. In many cases, probably in most, the irritability of the nerves and muscles to the faradic current is simply lowered ; but, in the majority of cases, the galvanic test gives the degenerative reaction. It is important to observe, however, that in a few cases of peripheral neuritis, such as one recorded in the *Lancet* of August 7th, by Dr. Saundby, the normal formula to the galvanic current was maintained, and although, in such cases, the electrical irritability is somewhat lowered in the affected nerves and muscles, yet a relative degree of lowering does not suffice to lead us by itself to an absolute conclusion.

The distribution of the paralysis varies greatly according to its cause, but in all varieties of the disease the extensors of the limbs manifest a proclivity to become predominatingly, although by no means exclusively, affected. The usual form of lead-paralysis consists of a double wrist-drop, caused by paralysis of the extensors of the forearm, and when the lower extremities are attacked, the most common form for the paralysis to assume is double ankle-drop. In these varieties of lead-paralysis, the supinator longus is spared in the upper extremity, and the tibialis anticus often, but not always, in the lower extremity. But although double wrist-drop, and, more rarely, double ankle-drop, are the most common varieties of lead-paralysis, yet, in some cases, the paralysis is widely distributed over the muscles of the extremities, trunk, and even the face, while the laryngeal muscles have occasionally been implicated.

In poisoning from alcohol and bisulphide of carbon, and in the animal poisons generally, and even in arsenical poisoning, the lower extremities are usually the first to be attacked. The paralysis begins by a double ankle-drop, in which the proper extensor of the big toe and the tibialis anticus are attacked with as much frequency as the common extensors of the toes and the peroneal group. The curving downwards of the big toe into the sole forms, indeed, a prominent feature of the ankle-drop of alcoholic paralysis, and is in marked contrast to the hyper-extension of the toe, which is so generally seen in the various forms of spastic paraplegia.

In alcoholic paralysis, the extensors of the forearm are attacked soon after those of the leg, and, indeed, when the latter are found paralysed, it is generally found that the former are so feeble that the patient is unable to maintain his hand extended upon the forearm in the prone position when a slight degree of pressure is applied to the back of the hand. After the extensors of the leg and forearm, the next muscles to be attacked are the quadriceps in the thighs, and the triceps in the upper arm, and, after them, the muscles of the calf and the flexors of the forearm, with the small muscles of the foot and hand. If the paralysis advances further, the remaining muscles of the thigh and upper arm become paralysed, and those of the trunk are so feeble that the patient is unable to sit up, or even to turn himself in bed.

In the remarkable cases of universal paralysis recorded by Dr. Bazzard as occurring in syphilitic subjects, the muscles of the face were implicated along with almost all the other muscles of the body. The face is always very expressionless in aggravated cases of alcoholic paralysis ; and Hun reported a case in which the muscles of one side of the face were paralysed ; but paralysis of the external ocular muscles, generally of the external recti muscles, has been observed by Hillier, Lilienfeld, and Schulz, and nystagmus has been noted by Oppenheim and Vierordt.

In a case of idiopathic neuritis observed by Freund, the pupils were dilated, the left more than the right ; and, in a case of alcoholic paralysis reported by Oppenheim, the light reflex was lost in the left eye, but the pupil reacted to accommodation, while, in the right eye, both reactions were lost ; but it is probable that these changes might have been independent of the alcoholic poisoning. Diptheritic paralysis generally begins in the muscles of the soft palate, and, after these, the external and internal muscles of the eye are invaded, and, after a considerable interval of time, the lower extremities are attacked, but the upper extremities are seldom much affected.

The predominance of the paralysis in the extensors imposes upon the extremities very characteristic attitudes. The wrist-drop of lead-paralysis causes the origins and insertions of the flexors of the forearm to be approximated ; and, consequently, these muscles undergo so much of adapted shortening as to make it appear, to a superficial observer, that paralysis of the extensors is accompanied by an active spasm of their antagonists. The same principle applies to the other joints. The ankle-drop in the lower extremities approximates the origins and insertions of the muscles of the calf, and the foot is consequently maintained in a position of talipes equinus, or equino-varus, according as the tibialis anticus muscle is or is not implicated along with the other anterior muscles of the leg. In one case of alcoholic paralysis, reported by Dr. Buzzard, the muscles of the calf had become so contracted that the patient was unable to bring his heels to the ground, although he was still able to stand and walk a little. When the muscles of the upper arm are attacked, in alcoholic paralysis, the triceps is relatively more paralysed than the flexors, and consequently the forearm is maintained flexed at right angles to the arm, and cannot be passively extended without causing great pain, by putting the shortened flexors on the stretch ; and, on the patient being asked, under these circumstances, to move his arm, the flexors of the arm and forearm, and the supinators, enter into contraction ; while the extensors and pronators remain unmoved, and, consequently, the deformity of the extremity is rendered still more marked. The forearm is drawn to an acute angle with the arm and supinated ; the flexion of the wrist on the forearm is still further exaggerated, and the fingers are drawn slightly towards the palm, so that superficial examination might lead one to suppose that the attitude was altogether caused by an active spasm, and the case then bears some degree of likeness to tetany. In the lower extremities, when the extensors of the thigh are implicated, the leg becomes partially flexed on the thigh, and the thigh on the body. In most cases, the angle formed at the knee-joint is not more acute than 120° ; but, in a case reported by Lancereaux, flexion was carried to so great an extent, that the muscles of the calves were applied to those of the thighs. In alcoholic paralysis, the patient generally lies in bed midway between the dorsal and lateral positions, but the position is more lateral in the lower extremities and pelvis than in the upper part of the body ; and, consequently, the legs lie on their side on the bed, with the thigh slightly flexed on the pelvis, the leg on the thigh, and the feet so much extended that their dorsal surfaces form, as Brissaud remarks, a continuous plane with the front of the leg ; while the toes, and especially the great toes, are curled downwards into the sole of the foot.

The deformities at the wrist-joint, and to a less extent at the ankle, tend to become permanent by adhesions taking place between the tendons and their sheaths, owing to an inflammation which attacks the flexor as well as the extensor tendons. Into the question of whether this inflammation is set up by the mechanical malposition of the tendons, or directly by the poison, I shall not enter ; it will suffice at present to notice that, when once this inflammation occurs, it tends to render permanent the various deformities of the joints.

The sensory disorders which accompany the paralysis need not detain us long. One of the chief characteristics of the usual form of lead-paralysis is the absence of any disorder of sensibility, but sensory disturbances are generally present when the paralysis is widely distributed. In the other forms of multiple neuritis, the paralysis is preceded by numbness and tingling pains in the hands and feet. The patients often suffer from paroxysms of lancinating pains, which are liable to become peculiarly distressing and teasing at night. In addition to these pains, patients complain of a distressing feeling of burning of the feet ; and, in some patients, according to Dr. Dreschfeld, the contact of any object gives rise to a sensation of cold. Cutaneous hyperæsthesia is occasionally present, but, in alcoholic paralysis, hyperæsthesia of the skin, even when present, is overshadowed by an intense hyperæsthesia of the muscles, which is so great as to evoke loud cries from the patient on the slightest pressure, and is at times so exaggerated that the weight of the bed-clothes is intolerable to the patient, and the feet have to be protected by cradles (Glyn). In the more advanced stages of the disease, the cutaneous hyperæsthesia gives place to more or less anaesthesia, sensory conduction is retarded, the tactile sense is diminished, and Remak's double sensations are sometimes observed. These sensory disorders remind one of the sensory symptoms in locomotor ataxy, and, to make the likeness more complete, the subjects of alcoholic paralysis sometimes complain of constrictive pains about the joints, and even a feeling of tightness round the chest, while swaying movements of the body occur when the eyes are closed. The subject of the disorders of the special senses, especially of the optic nerves—which may be present in the various forms of peripheral neuritis—is so wide that I dare not enter upon it.

The vaso-motor disorders consist of redness of the hands and feet, and oedema of the ankles and of the backs of the feet and hands. The body is often bathed in a profuse sweat. The pulse is in most cases accelerated, rarely beating under 120, and sometimes being as frequent as 160 in the minute. A case of alcoholic paralysis has, however, been reported by Oppenheim, in which the pulse, which was very irregular, beat only forty-eight in the minute; but, as the patient improved under treatment, the pulse rose in frequency, and the action of the heart became regular.

Peripheral neuritis may begin with a febrile movement, and there is occasionally a considerable elevation of temperature before death in fatal cases, but it probably depends upon some complication.

The trophic disorders consist of glossy skin, papulous and herpetic eruptions, local asphyxia, and even gangrene of the extremities has been met with, as in cases recorded by Lancereaux and Myrtle.

Incontinence of urine is rare, and of feces still rarer, but both symptoms have occasionally been observed (Francott, Schulz). Sexual impotence is probably very common, and, in paralysis from bisulphide of carbon, the impotence which is always present is said to be preceded by satyriasis. Bed-sores are almost never present, but, in the case of paralysis recorded by Fox in *Brain*, a small bed-sore was situated on the left buttock. The psychical symptoms are also very interesting, but I shall pass them over.

Diagnosis.—The first disease for which progressive multiple neuritis is likely to be mistaken is chronic poliomyelitis. In both diseases, there is a widely distributed paralysis, with atrophy and the usual degenerative electrical reactions of the affected muscles. The neuritic affection in its generalised forms is, however, usually accompanied by well-marked sensory phenomena, which are absent in the spinal disease. The order in which the muscles are attacked is also different in the two diseases. When the spinal disease pursues an ascending course, the paralysis begins in the muscles of the feet, and gradually invades in succession the muscles of the leg, the flexors of the thighs, the extensors of the thigh, the gluteal muscles, and the muscles of the trunk. Subsequently, the intercostal muscles show signs of weakness, and one of the most important signs of all is to be found in the fact that the small muscles of the hand, which are innervated by the first dorsal nerves, are attacked before the extensors of the wrist and fingers, which are innervated chiefly by the seventh cervical nerves. If, then, we meet with a case of widely diffused atrophic paralysis beginning in the muscles of the lower extremities, and attacking the extensors of the forearm before the small muscles of the hand, the disease is not likely to be of spinal origin; and, again, if in a case of atrophic paralysis the diaphragm is attacked before the intercostal muscles, the disease is not a spinal one; and when the paralysis begins in the upper extremities and pursues a descending course; if, then, the anterior extensors of the leg are paralysed before the gluteal muscles and the muscles supplied by the anterior crural and obturator nerves, the disease is not a spinal one.

Acute cases of peripheral neuritis are liable to be mistaken for Landry's paralysis, and there can be no doubt that some, at least, of the cases described under the name of acute ascending paralysis really belong to the category of peripheral neuritis. The cases of ascending paralysis described by Myrtle and Fox, for instance, are undoubtedly examples of alcoholic paralysis. Of the remaining cases of acute ascending paralysis, some, like the cases of Schulz and Schultze, are caused by an acute myelitis or meningo-myelitis, and others are caused by an acute bulbar myelitis, but a few remain which are of obscure origin, and the pathology of which is unknown, inasmuch as no anatomical changes of any kind have been discovered after death. If we meet, therefore, with a rapidly ascending paralysis, with loss of tendon-reactions, and probably of the cutaneous reflexes, but without being accompanied by marked sensory disorders, muscular wasting, or a degenerative reaction on electrical examination, we should regard it, in the present state of our knowledge, as an example of Landry's paralysis, while an acute ascending paralysis, which is accompanied by marked sensory disturbances, muscular wasting, and degenerative reaction on electrical exploration, and in which the extensors of the forearm are attacked before the flexors we should place in the category of peripheral neuritis.

The most interesting, as well as the most difficult problem in diagnosis arises from the close resemblance between tabes dorsalis and cases of widely distributed paralysis from peripheral neuritis. I will not dwell upon all the signs by which genuine tabes may be distinguished from the cases now very unfortunately named pseudo-tabes, such as the chronic character of the former affection and the comparatively acute course pursued by the latter, and the nature of the eye-symptoms in ataxy in comparison with those met with in neuritis, but I will proceed at once to show that an attentive examination of

the gait in the two affections will generally suffice to discriminate the neuritic disease from genuine tabes.

Let us suppose that one foot has just been moved forwards, and is planted on the ground in front of the other. By a contraction of the abductor muscles of the thigh, the body is now inclined to that side, so that the line of gravity passes through the middle of the arch of the foot. The heel of the foot in the rear is, at the same time, raised and now the limb is ready to be moved forwards. In normal locomotion the anterior muscles of the leg enter into contraction, and when slight dorsal flexion of the foot is made, the toe clears the ground, and the limb swings forwards with a pendulum movement, by its own weight, and not by muscular action. In pseudo-tabes, however, the toes, instead of being elevated, as in normal walking, by a slight dorsal flexion of the foot, drop lower as the foot is raised from the ground, and consequently, before the limb can be moved forwards, the heel must be elevated to an unusual degree, so that a person looking from behind sees, at each step, more of the sole of the foot than in health. During the forward movement of the foot, the necessary elevation of the toes is obtained by the thigh being flexed to an unusual degree on the pelvis, and the leg on the thigh, so that a person standing by the side of, or in front of the patient, will observe that with each forward step the knee is raised very high, while the toe drops; this elevation of the knee impresses upon the gait that characteristic which Charcot has aptly compared to the carriage of a high stepping horse, while the depression of the toe gives to the gait an appearance which has led Schulz to compare it with the walk of a dancing master. When once this peculiar gait is observed in a patient, he ought to be placed sitting on a chair, with his feet flat on the ground, and thus asked to elevate the toes of each foot in succession. If he is unable to perform this action, the disease is almost certainly not locomotor ataxy. With regard to the course of peripheral multiple neuritis, I may state briefly, that in 48 cases arising from various causes collected by me, the disease proved fatal no less than 26 times. Of the fatal cases, death was caused 8 times from some complication like granular kidney and broncho-pneumonia, and 18 times from respiratory paralysis, paralysis of the diaphragm being expressly mentioned in the majority of these last cases. The duration of the fatal cases, counting from the first appearance of pronounced paralysis, varied, with two exceptions, from nine days to five months. Of the cases which recovered, the majority were able to walk fairly well in about four months from the commencement of treatment, but a case is recorded by Fischer, in which the patient is said to have only made a fair recovery at the end of two years, and one is reported by Buzzard, in which the patient made a good recovery at the end of three years of continuous treatment.

Morbid Anatomy.—In one of the cases of idiopathic peripheral neuritis, reported by Leyden, the musculo-spinal nerves were found thickened for about 5 centimetres of their length by a deposit of fat. The nerve-sheath was thickened, and the primitive bundles held only a comparatively small number of medullated fibres, the medullary sheaths were absent and destitute of nuclei, and what remained of the fibres was constituted by thickened axis-cylinders. The vessels in the fatty tissue were surrounded by a considerable quantity of granular yellow pigment, showing, according to the author, the existence of a previous hæmorrhagic infiltration. The spinal cord and nerve roots were normal. In lead paralysis the changes described by the earlier pathologists, consisted of softening, and superficial hæmorrhages of the brain, but as the methods of microscopical examinations of nervous tissues had become perfected, attention was directed to the spinal cord and peripheral nerves. Vulpian observed vitreous degeneration and atrophy of the ganglion cells of the anterior horns of the spinal cord in one case, and in another he found neuritis of the anterior roots of the cervical region of the cord. Minor changes in the ganglion cells of the anterior horns, such as vitreous degeneration and vacuolation, have also been reported by Monako, Zuckerkandl, Oppenheim, and Robertson of Manchester, but other competent observers have failed to discover any evidence of disease in the spinal cord. Disease of the peripheral nerves has been observed by Gombault and Charcot, Westphal, Vulpian, Friedlander, Déjerine, Kussmaul, and Maier, Eisenlohn, Dreschfeld, and Moritz, Robertson, Oppenheim, and others, so that now there is amongst the most trustworthy pathologists an almost unanimous consensus of opinion that the essential lesion in lead paralysis is to be found in the peripheral nerves. This last remark applies equally to alcoholic paralysis. Such observers as Lancereaux, Moeli, Dreschfeld, Déjerine, Hadden, and Francott, have failed to discover any evidence of disease in the spinal cord in cases of alcoholic paralysis, but they have found marked changes in the peripheral nerves. In diphtheritic paralysis, minor changes, indicative of spinal meningitis, have been reported by Déjerine and by Pierret,

while Vulpian described a rarefaction of the posterior and external portion of the spinal cord, but the opinion, first advanced by Charcot and Vulpian, that the essential change is to be found in the peripheral nerves, is now gaining ground with pathologists to the exclusion of all other views. In kaki or beri-beri, the peripheral nerves have been found diseased by Raelz and Scheube.

Now, upon the question of the nature of the lesion, whether it is inflammatory or simply degenerative, a parenchymatous or an interstitial neuritis, I do not feel myself competent to speak. Eichhorst and Hadden believe the changes observed by them to have indicated a perineuritis, while most other authors believe that the disease begins in the nerve-fibres themselves. In one of the cases reported by Dr. Grainger Stewart, which was most probably an example of alcoholic paralysis, the changes observed in the nerves were regarded by Professor Hamilton, now of Aberdeen, to have indicated a simple degenerative neuritis, and the same view has been adopted by Moeli and others.

In some reported cases, the affected nerves have been found much swollen, and, in a case recorded by Desnos and Pierret, the posterior tibial nerve is said to have been nearly as thick as the little finger, and such a condition could hardly have been produced by a simple degenerative neuritis. It is hardly probable that an active parenchymatous neuritis would lead to such a remarkable degree of swelling of the nerve. That a parenchymatous neuritis has been met with in some cases of this affection scarcely admits of doubt. By inducing chronic lead-poisoning in guinea-pigs, Gambault produced inflammatory changes in the nerves of such a character as to lead him to name the lesion segmental periaxillary neuritis. In this condition, a segment lying between two of the nodes of Ranvier is found diseased, while those on the other side of these nodes remain healthy, and several segments may be affected in the course of one fibre. Dr. Dreschfeld met with this condition in the nerves in a case of alcoholic paralysis, and it may be assumed that it indicates a parenchymatous neuritis. A very valuable observation has just been made by Gambault in cases of alcoholic paralysis. At a certain elevation of the diseased nerve, he found the endoneurium thickened, and the nuclei of the endoneurium and of the sheath of Schwann much increased in number, while the medullary sheaths and the axis-cylinders were simply attenuated, probably by compression. Below this level, the changes observed indicated simply the ordinary Wallerian degeneration. It is therefore possible that, at a certain level of the nerve, a perineuritis may be observed, and that the fibres below this level, being separated from the trophic centres, undergo the usual descending degeneration.

With regard to the localisation of the lesion, this much is certain; the paralysis, which is present in these diseases, being of the atrophic variety, the lesion must implicate the ganglion cells of the anterior horns of the spinal cord, the fibres which connect them with the muscles, or the muscles themselves. The sensory disorders which are present, render it certain that the disease is not a purely muscular one, and, consequently, the lesion must be situated in the grey matter of the spinal cord, or in the fibres connecting the motor ganglion cells with the muscles. We have just seen that morbid anatomists are declaring in favour of the opinion that the disease is situated in the peripheral nerves rather than in the spinal cord.

Let us now see whether the clinical phenomena can be explained on this view, as well as, or better than on the assumption of the disease being of spinal origin. Some pathologists find great difficulty in adopting the neural theory, because the motor paralysis is often, as in the usual form of lead-paralysis, unaccompanied by any sensory disturbances. But, it is a general law that inflammation and injuries of mixed nerves give rise to a degree of motor paralysis which is out of all proportion with the sensory phenomena. In disease of the musculo-spiral nerve, caused by compression and exposure of its trunk as it winds round the humerus, the extensors and supinators of the forearm remain paralysed for four or five months; but the anaesthesia which accompanies motor paralysis is only slight or transitory. We often speak as if the spinal nerves of the body were all mixed nerves throughout their whole course, and it seems to be forgotten that, for nearly half their length, they are either purely sensory or purely motor. In the musculo-spiral nerve itself, the sensory branch—the radial—is given off above the elbow; and, as in the ordinary form of lead-paralysis, the supinator longus is always spared, it is clear that the lesion does not attain to the level at which the sensory branch would become affected. Similar remarks apply, although not to the full extent, to the nerves which supply the extensors of the leg. The common extensors of the toes and the peroneal group, and the tibialis anticus, may be separately paralysed without the sensory branch being much implicated; but, when all the anterior muscles of the

leg are implicated, the lesion has most probably reached the level of the external popliteal nerve, and in a case of lead-paralysis under me, in which all these muscles were implicated, the external surface of the legs and the dorsum of each foot were more or less anaesthetic.

The next argument brought against the theory of peripheral neuritis is that the muscles are paralysed in groups, according as they are associated in their actions, and not according to the peripheral distribution of the nerves. But the extensors of the forearm—which are the muscles usually attacked in lead-paralysis—form a functional group, which is innervated by one nerve, namely, the musculo-spiral, and consequently disease of that nerve would explain the paralysis as readily as disease of the spinal centre.

But what Remak calls the upper arm type of lead-paralysis, in which the deltoid, biceps, brachialis anticus and the supinator longus are attacked, is more difficult to explain on the peripheral neuritis theory, inasmuch as these muscles are innervated by three nerves, namely, the circumflex, the musculo-cutaneous, and branches from the musculo-spiral. It is now known that these muscles are paralysed by injury or disease of the fifth cervical root just before it joins the brachial plexus. This form of paralysis was first described by Erb, and, in a case observed by me a short time ago, the motor affection was accompanied by relative anaesthesia of the skin of the outer surface of the arm, and of the external border of the forearm and hand as far as the metacarpo-phalangeal joints of the thumb and index finger. Now in a case of the upper arm type of lead-paralysis recently under me, the paralysis was also accompanied by a considerable degree of anaesthesia on the outer surface of the upper arm, and even although it could not be traced below the elbow, the combination of symptoms present could be better explained on the supposition of a neuritis localised in the fifth cervical root immediately above its point of junction with the brachial-plexus than by a lesion in the grey matter of the cord. These considerations help to remove the main objections which have been advanced against the neuritic theory, but time will not allow me to enter upon the positive objections which might be urged against the spinal theory.

SOME POINTS IN THE PATHOLOGY OF MULTIPLE NEURITIS.¹

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MODERN observation has been rewarded by the discovery of an increasing number of cases in which more or less pronounced symptoms of paralysis have been found to be dependent upon changes in the peripheral nerves, the cerebro-spinal centres being usually free from any sign of lesion. In speaking of an "increasing" number of such cases, it is not implied that many more examples of the kind occur now than formerly, but that, as observation is on the alert, a larger number of cases is differentiated from the category of central organic lesions with which these instances were formerly confounded. The writer has so recently published a work upon this subject,² that he does not consider it necessary on the present occasion to do more than refer in the briefest and most general manner to the clinical and pathological aspects of multiple neuritis, but proposes to present for consideration a few remarks bearing chiefly upon the nature and pathogenesis of the disorder.

Multiple neuritis has been observed in connection with many conditions which are provisionally assumed to be associated with its etiology. Amongst these may be mentioned certain toxic influences, lead, arsenic, alcohol, infective disorders, as leprosy, diphtheria, syphilis, beri-beri, variola, enteric, recurrent and intermittent fever, tubercle, whilst other examples are associated with the not easily classed conditions of cold, rheumatism, diabetes. But besides these there would seem to be a somewhat large number of cases in which it is not possible at present to discover any antecedent which can properly be considered a cause. I shall recur presently to this very interesting part of the subject.

It is not intended to assert that the various influences mentioned produce examples of disease exclusively affecting the periphery of the cerebro-spinal nerves, the great centres themselves being invariably intact. Such is manifestly not the case. Although, for example, a large number of observations has shown disease limited to the peripheral nerves in the course of leprosy and of lead-poisoning, yet examples of these disorders have occurred in which the spinal cord itself

¹ Read in the Section of Pathology at the Annual Meeting of the British Medical Association in Brighton, introductory to a discussion on Peripheral Neuritis.

² *On Some Forms of Paralysis from Peripheral Neuritis*. Churchill. 1886.