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ON

PARALYSIS OF HANDS AND FEET

FROM

DISEASE OF NERVES.

BY

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(*Read before the Medico-Chirurgical Society of Edinburgh, 2d March* 1881.)

PRINTED BY OLIVER AND BOYD, EDINBURGH.

MDCCCLXXXI.

REPRINTED FROM THE EDINBURGH MEDICAL JOURNAL FOR APRIL 1881.

ON

PARALYSIS OF HANDS AND FEET FROM DISEASE OF NERVES.

THE interest of physicians in the study of diseases of the nerves, as distinguished from those of the nerve centres, has recently been on the increase; and certain cases which have during the past year come under my observation are so well fitted to throw light upon one department of the subject, that I think it desirable to bring them under the notice of the Society.

CASE I.—J. I., a clerk, æt. 52, was admitted to Royal Infirmary 18th Feb. 1880, complaining of weakness, stiffness, and pains in hands and feet, which had lasted for six weeks. His father had died from hemiplegia at the age of 85. He had himself been temperate, and lived under healthy conditions. He had had no important disease prior to the commencement of the illness for which he sought advice. He stated that in the beginning of December 1879 he fell down a stair and was considerably bruised and shaken. On the following day had some rheumatic pains, which came and went during the succeeding week. About ten days or a fortnight later he began to experience a feeling of tingling and numbness in his hands and feet, with stiffness in the joints of the hands and a difficulty in grasping and in walking, swelling of the feet and hands, and severe cutting pains both in the hands and the feet. All these symptoms gradually increased in severity until admission.

On admission he was pale, but not cachectic-looking. Temp. in right axilla 99°·8, in left 99°·2. There was no important abnormality excepting in the nervous system.

Sensory Functions.—He complained of severe cutting pain of an intermittent character in hands and feet, increased on pressure, especially of palmar or plantar surfaces, and aggravated by movements of the body, such as lying over on his side or bending his back. With the pain there was a feeling of numbness, but the tingling sensation of which he had previously complained had dis-

appeared. There was no girdle-pain, formication, or giddiness. Sensibility to touch was greatly diminished in both hands, slightly so in the forearms, normal above the elbows. It was also greatly diminished and delayed in both feet, and to a less extent in the legs. Sensibility to heat and tickling were correspondingly affected. The perception of painful impressions was delayed, but felt acutely, a distinct interval elapsing between pinching the hand and its perception; but a slight pinch was felt to be very painful, and the pain persisted longer than normal, and set up considerable reflex movements. The muscular sense in hands and feet, as tested by pointing and by the application of weights, was diminished, but the sense of pressure still more so. Sight, hearing, taste, and smell were all normal.

Motor Functions.—The organic reflex functions were normal; the skin reflex was absent when slight stimulus was applied to soles, but exaggerated and tending to become general and painful when the stimulus was strong. Patellar tendon reflex was lost. Voluntary motion was greatly impaired in hands and feet, all the groups of muscles being weakened, but those of the fingers and toes completely paralyzed. The muscles responded to some extent to the interrupted current, and in proportion to the degree of paralysis the result was feebler.

Vasomotor and Nutritive Functions.—There was glossiness of the skin over the backs of the fingers, and some appearance as of bruises over the toes, but there was no œdema. The muscles were diminished in volume. The acute pain which attended passive movements of the hands and wrists, as well as of the feet and ankles, was not associated with crackling or other evidence of changes of joints. There were no fibrillary twitchings of the muscles.

Cerebral and Mental Functions were normal.

The *Cranium and Spine* were also normal.

The treatment at first was by means of the liq. extract of ergot (℥xx. every four hours), and afterwards by the application of the interrupted current to the paralyzed part. A degree of improvement soon manifested itself, and on May 26th, three months after admission, the condition was as follows:—Pain had disappeared; numbness continued, but to a less extent. It was more marked in the right than the left hand. Sensibility to all kinds of impressions correspondingly improved. Motor power, although still diminished, greatly better. The muscles of the affected districts much wasted; and though the pain had disappeared from the joints, there was a good deal of stiffness.

On *July 15th* the sensibility was almost normal. The patient could distinguish the weight and pressure of a half-sovereign from that of a sixpence put into his hand. Skin and patellar tendon reflex were normal. Voluntary motion is slowly returning, the patient being able now to write his name with a little effort, and to stand, although not yet to walk.

On 23d September the sensory functions were normal, the skin and patellar tendon reflex rather exaggerated. Voluntary movements of hands were almost completely normal, except in respect of flexion of the distal phalanges. Patient could walk about quite well, but moved the feet *en masse*, the toes playing imperfectly, although he could move them when sitting. The skin was better nourished, the nails much curved. The muscles of the arms and legs, which had been greatly wasted, were gradually recovering their normal volume.

Case II.—J. H., æt. 51, a weaver, employed in the Blind Asylum, was admitted to the Royal Infirmary 11th June 1880, complaining of weakness and numbness in hands and feet, with pain in back. He had been ill for twelve days. Family and personal history were unimportant. The patient stated that on the 6th of June he had, after sleeping for a couple of hours on a sofa, felt on rising that he was chilly, and had a numbness and weakness in both legs and feet, as if they were "sleeping." The following day the symptoms continued, but he managed to work, although feeling the extremities cold and with "pins and needles." During the following day a further increase of discomfort took place, so that on the next he was unable to go to work. On the 11th he was able to walk from the Blind Asylum to the Infirmary, a distance of about a quarter of a mile, but with considerable difficulty and great uneasiness, and the effort was followed by a feeling of intense fatigue. Some days after the feet had become affected the hands became involved in a precisely similar manner.

On admission there was nothing wrong except in connexion with the nervous system.

Sensory Functions.—Pain was felt in the small of the back on the slightest movement, but the pain was not severe. He complained of feelings of coldness, formication, and numbness in the feet and tips of the fingers. Sensibility to touch was distinctly impaired in the parts in which the abnormal sensations were localized. The right hand and foot were worse than the left. Sensibility to heat and cold distinctly diminished; to pain, natural. The muscular sense and the special senses were normal.

Motor Functions.—The organic reflexes were natural. The skin reflex absent in right foot, diminished in left. Patellar tendon reflex absent in both legs. Passive motion of all the limbs normal. Voluntary motion was lost in the toes, very imperfect at the ankles, normal at the knees and hips. The hands, particularly the right, were much weakened. The movements of the wrists, fingers, and thumbs, although feeble, were not lost. The patient could neither walk nor stand unsupported. Co-ordination seemed impaired. He could not write, and had the greatest difficulty in buttoning an easy button.

Vasomotor and Nutritive Functions.—The feet, and to some

extent the hands, were œdematous. The muscles of the affected parts were flabby and undergoing atrophy.

The Cerebral and Mental Functions were natural, and the spine exhibited no curvature nor pain on percussion. He was ordered one drachm of liquid extract of ergot three times a day. During the following fortnight there was a good deal of constipation, and somewhat later a girdle-pain was complained of round the abdomen. In the latter part of July it was occasionally necessary to draw off the urine by catheter.

On the 12th of August there was some pain round the abdomen and a feeling as of a tight bandage over both feet. The sensibility to touch diminished in the feet. Moderate heat was perceived correctly but slowly; great heat was perceived instantaneously. Pain of other kinds was felt naturally; special senses natural. Organic reflex functions interfered with in respect of micturition and defecation, the former apparently by spasm, but less troublesome than before. Skin reflex of soles diminished. Patellar tendon reflex lost in both legs. Voluntary motion slightly improved in the legs; in the hands now practically restored.

During September improvement went on. Towards the end of it he was able to walk a few steps with the aid of another patient. In the end of October he was able to walk a distance of an eighth of a mile. Since that time he has been steadily improving. During the later stages of the case strychnia was substituted for the ergot which had been used at first.

Case III.—G. F., æt 31, hotel-keeper, admitted to Royal Infirmary 8th November 1880, complaining of general weakness, pain, stiffness, and loss of power in hands and legs, and confused vision. He was a German by birth, had been employed in various hotels, and had recently become proprietor of a hotel and got married. Early in August he noticed a weakness in the legs, and a dancing of objects before his eyes. In September pain came on in the legs. It was of a prickling character, and gradually increased. Towards the end of October a similar feeling came on in the fingers, and afterwards the hands, the feeling being accompanied with loss of power and stiffness, the symptoms also becoming gradually more intense.

On admission it was found that there was nothing important wrong except in the nervous system.

Sensory Functions.—He had pain of a tingling character in both legs, from the knee to the dorsum of the foot, with numbness, a feeling of cold in the toes and plantar surfaces; so also in the hands, but to a less extent. There was no girdle-pain, formication, or giddiness. Sensibility to touch was diminished in the legs below the knees, and in the hands. The transmission of impressions was delayed, and he had difficulty in localizing them. Sensibility to heat, tickling, and pain were all diminished. Muscular sense,

normal in the hands, appeared almost absent in right foot, and diminished in left. Sight appeared normal, but he complained of objects dancing before the eyes. There was no nystagmus. The other special senses were normal.

Motor Functions.—Organic reflex functions normal. The skin reflex absent in soles. The cremasteric reflex and that of the trunk well marked. The patellar tendon reflex absent in both legs. Voluntary motion was greatly impaired in legs and hands. The fingers remained in a semi-flexed position. Electric sensibility and irritability much diminished in legs and forearms, specially the extensor muscles. Attempt to use the muscles produced great pain.

Vasomotor and Nutritive Functions were normal.

Cerebral and Mental Functions were somewhat impaired. He was drowsy ; memory imperfect. Although he conversed tolerably well, he made mistakes, such as calling every day Sunday. The cranium and spine were normal.

On the 17th some degree of improvement is reported to have existed, but a few days later the patient was seized with croupous pneumonia of both lungs, of which he died on 4th December.

It was clear that these three cases closely resembled one another, that they were, in fact, examples of one malady. The clinical features which seemed most important were, *the co-existence of symptoms referable to the sensory, the motor, and the trophic functions of the nerves, the localization of the symptoms in the feet and hands, the intensity being greatest at the most distal points, and the affection corresponding to certain districts of the extremities, and not to the distribution-areas of particular nerves.*

On endeavouring to arrive at a diagnosis of the lesion causing these symptoms, it was evident that it must be referred either to the nerves, the spinal cord, or the brain. Now, it was certain that no known facts warrant our believing that any individual part of the brain determines all kinds of innervation in individual parts of the body, and still less that lesion of any part should be capable of producing symptoms so curiously distributed. If we were to assume a cerebral origin, we should have to conclude that the lesion was bilateral, and that in several points of both hemispheres lesions existed. It was thus certain that the disease could not be regarded as cerebral. It might have been of spinal origin, and due to some affection of the cord at a point corresponding to the origins of nerves for the feet and the hands; but here again it was manifest that the lesion must be assumed to be bilateral and sufficiently extensive on each side as to involve sensory, motor, and trophic structures, but at the same time to affect these only so as to produce symptoms involving the ultimate distribution of certain nerves, and the different nerves in such proportion as to mark off precisely certain areas bounded by lines encircling the limb. Moreover, it would be necessary to assume that the lesion

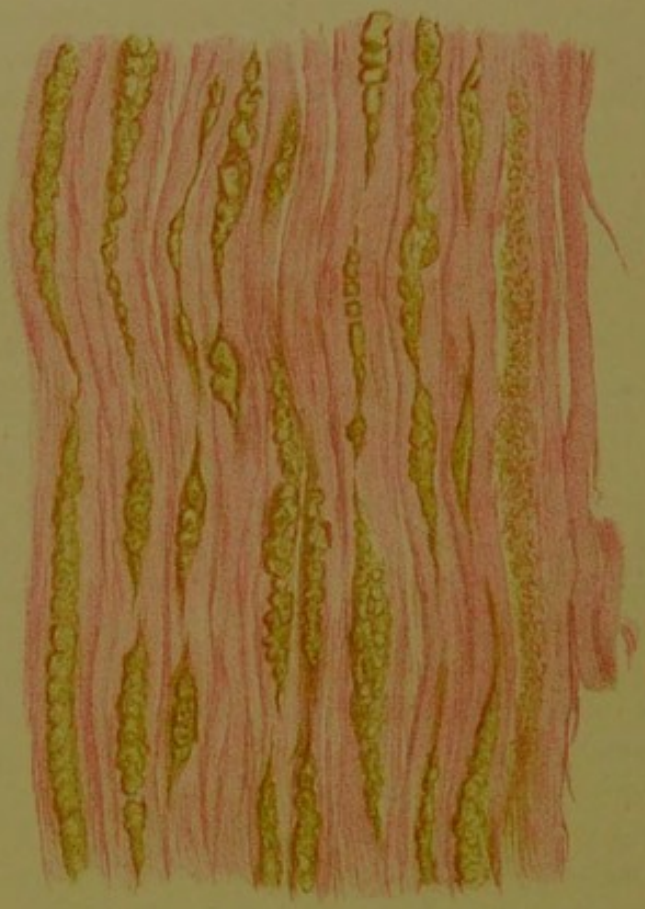
affecting the hands was so distributed as not to interfere with the transmission of sensory impressions from lower parts of the body upwards, nor of motor impulses from the cerebrum downwards to certain muscles, which conclusions it is impossible to regard as consistent with our present knowledge of the physiology of the cord. Moreover, the exact distribution of the symptoms in another respect forbade our accepting this hypothesis, because, in one of the cases, the affection of sensation and of motion was most intense upon the same side of the body, while, according to the law discovered by Brown-Sequard, and illustrated in a vast number of clinical cases, disease of one-half of the cord produces paralysis of motion on the one side and of sensation on the other. It was thus apparent that this view also could not be accepted. By a process of exclusion, then, I was led to conclude that the *nerves themselves must be the seat of disease*, and a consideration of the symptoms afforded direct evidence of this. What but a nerve lesion could have induced so curiously distributed an abnormality of all the functions?

The diagnosis was established after a study of the first two cases. One of them was demonstrated to the class of clinical medicine as an example of peripheral paralysis, and the third was at once recognised as another example of the same disease. It seems to me that the clinical features, even apart from pathological evidence, should satisfy any competent authority as to the nature of the disease; but the unfortunate occurrence of acute double pneumonia, with its fatal termination, in the third of my series of cases, gave us the opportunity of ascertaining the pathological changes, and the results of the examination, as reported by Dr Hamilton, are the following:—

On post-mortem examination both lungs were found to be much congested, the lower lobes in a state of red hepatization. The brain weighed 3 lbs. The vessels of the dura mater contained a medium amount of blood. The arachnoid space at the vertex was very œdematous. The left vertebral artery was smaller than the right; the left measured one line, the right two, in diameter. It looked like a congenital alteration, there not being any local cause. The medulla oblongata, in the neighbourhood of the Corpora Olivaria, was slightly indurated; otherwise it appeared healthy. Most of the nerves of the extremities, the spinal cord, and the brain were retained, and carefully hardened in Müller's fluid and spirit.

The brain was carefully examined by a series of perpendicular large sections, but no gross lesion, further than a little distention of the fifth ventricle, existed.

The spinal cord in the highest cervical and lower dorsal regions was healthy, but in the cervical enlargement, and to a less extent in the lumbar, there were what appeared to be certain tracts of secondary degeneration. They were extremely sharply defined at the margin, but very faint, and affected only the columns of Goll and the outermost part, particularly towards the back of the lateral columns.



Longitudinal Section of Portion of Right Median Nerve (300 Diameters).—Drawn from nature by Mr John Thomson.

The median, ulnar, and tibial nerves showed more notable change than any other part. Looked at with a low power in transverse section, certain bundles of nerve fibres appeared to be totally destroyed from what looked like fatty degeneration; others were only partially destroyed, while some were comparatively healthy. In the ulnars the degeneration was less evident than in the medians. In the medians there was hardly a sound bundle of fibres.

Under a higher power (300 diam.) the degeneration presented the following characters (see Plate):—It began by a swelling of the axis cylinder, so as to form a number of fusiform bodies in the course of the nerve tube. These, when they had reached a certain size, divided into a number of rounded bodies varying in size, but mostly considerably larger than a leucocyte. They were perfectly homogeneous, and resembled ordinary colloid bodies in every respect. The whole course of the axis cylinder thus underwent degeneration. When the colloid masses were set free they mostly underwent fatty degeneration, forming compound granular corpuscles. In some strands of the fibres the whole of the axis cylinders were destroyed, and nothing but a quantity of fibrous tissue remained. Two cords of the brachial plexus were examined. In neither was there any evidence of the disease as seen in the lower nerves of the limb. Most of the bundles of nerve fibres appeared normal. One or two tracts may have been a little abnormal, but there were none of the axis cylinder changes as in the nerves of the forearm. Any change there may have been would in all probability be secondary, and not of the same nature as the primary alteration in the peripheral parts of the nerves. Both sciatic nerves seemed to be healthy. There were certainly none of the axis cylinder alterations found lower down. There seemed, however, to be an unusually great quantity of fat between the bundles of nerve fibres. The only piece of muscle which was retained was attached to the brachial plexus. It was perfectly healthy, the striæ upon it unusually well marked.

The first question which we have to settle is, whether the result of the autopsy confirmed the diagnosis which we had established of paralysis from disease of nerves. Manifestly there was no affection of the brain; but the cord was distinctly diseased, and it might be held that that disease was the cause of the symptoms. Observe its distribution. It was situated only in part of the cervical enlargement, and to a less extent in the lumbar enlargement, and affected the columns of Goll on both sides, and also the most superficial and posterior part of the lateral columns, including part of what Flechsig terms the direct cerebellar tracts. It did not affect any part of the gray matter, neither were the nerve roots involved. This localization is distinctive of what is known as one of the forms of Turck's secondary degenerations.

I may be permitted briefly to remind the members of the Society

of the leading facts connected with these secondary degenerations. They are changes in certain strands of fibres in the brain and spinal cord which are found to follow upon diseases of the brain and cord. They are of two groups, the descending and the ascending; and sometimes we can trace, in connexion with a localized lesion in the cord, the descending below and the ascending above the seat of lesion. In descending lesions following upon disease of one hemisphere of the brain the changes manifest themselves in what are known as the pyramidal fibres, which are situated partly in the lateral column of the opposite side of the cord (the crossed pyramidal fibres) and partly in the anterior columns of the same side (the direct pyramidal fibres). In cases of descending degenerations secondary to lesion of the cord both sides are commonly affected. In ascending secondary degenerations, on the other hand, the columns of Goll are constantly affected, and along with them certain of the superficial or external fibres of the lateral columns. The lesion in our case occupied precisely these situations, and I have therefore no hesitation in accepting the view that the changes are purely secondary, and could not explain the symptoms manifested by our patient. But it is worth while to inquire what was the cause of the secondary degenerations. Are they to be referred to the nerves? I cannot doubt that they are; and yet it is most interesting to observe that it cannot have been propagated by direct continuity of morbid process, for it was the lower stretches of the nerves that were found affected, the cords of the brachial plexus and the sciatic showing, so far as Dr Hamilton could ascertain, but little change.

As to the nerves themselves, the lesion was extremely distinct. It involved a large proportion of the nerve fibres of the affected portions of nerves, and consisted in a breaking up of the axis cylinder; but to the naked eye the nerve appeared quite normal, the affected parts of the median and radial being undistinguishable from the unaffected parts farther up. A consideration of these facts must satisfy every competent authority that the diagnosis in our fatal case was amply justified. If this be so, I think it will scarcely be doubted that in the other two cases in which, on the same grounds, the same diagnosis was established we are entitled to conclude that the view taken was also correct.

Much remains still to be learned regarding the details and the course of the anatomical change. I do not think it at all likely that the process originated in muscle and spread thence to nerves seeing that the sensory changes were manifest before the motor. I greatly regret that we are not able to say anything at present with regard to the condition of the nerve endings, but from all the facts I am satisfied that the process had originated in the periphery of the nerves and spread upwards.

It is a question full of interest, though not at present susceptible

of satisfactory answer, What are the conditions which induce the process? Is it cold? Is it some poison introduced from without? Is it some poison generated within the system? or may the process owe its origin to any of these? The fact that the disease originates simultaneously, or almost simultaneously, in both the hands and in both the feet seems to point to some constitutional cause; and although in the meantime we must leave the question of its nature unsolved, it is well that we should state it to ourselves, so that we may be less likely to neglect an opportunity of clearing it up.

The occurrence of peripheral paralysis is, of course, familiar to every practitioner, but it is generally in connexion with the three following groups that we are accustomed to think of it. We are all familiar with such peripheral paralyses as that of the portio dura of the seventh cranial nerve (Bell's paralysis), or the oculo-motor paralysis involving ptosis and strabismus. We also know well the forms of local paralysis resulting from pressure on nerve trunks, as, for example, when a patient has fallen asleep in such an attitude as leads to pressure upon a particular nerve or group of nerves, and awakes with the limb paralyzed as to sensation and motion. Again, we are more or less familiar with cases in which, from some unexplained cause, an individual nerve becomes paralyzed, and for a longer or shorter time its function continues in abeyance; but it is clear that the cases which I have described belong to a quite different category from these, and I must acknowledge that to me at least they were not definitely known until the study of these cases defined them to me.

On referring to the literature of nervous disease, I find very few cases corresponding to it accurately described. In the admirable works of Wilks, Hammond, Rosenthal, Eulenburg, Hamilton, Leyden, and Erb, I have failed to find satisfactory clinical accounts of the disease, although there are indications in most of these works that cases more or less analogous have come under the notice of the authors. In the periodical literature of recent years, however, similar cases are to be found, and I think it may be well briefly to refer to those to which I have had access. The earliest references to cases of this description are those of M. Duménil of Rouen, published in the *Gazette Hebdomadaire* for 1864 and 1866. The first of his observations was made on the case of a man, æt. 71, who was suddenly seized with numbness in arms and feet, followed by paralysis, anæsthesia, muscular atrophy, etc. After death the brain, spinal cord, and nerve roots were found to be normal, while the nerves themselves were the seat of extensive degeneration. Duménil points out the close analogy between this condition and the general spinal paralysis of Duchenne. In his second paper on the subject the same writer gives one case with similar symptoms in great detail, in which he found, after death, extensive degeneration of nerves, anterior nerve roots, and

spinal cord. He believes that the facts he gives warrant him in concluding that "a class of spontaneous peripheral paralyses exists due to atrophy of the nerves. The morbid process which produces these atrophies is inflammatory in its nature in many, if not in all cases. These paralyses may affect motion and sensation with equal intensity, either simultaneously or successively, and in the latter case motion appears to be affected by reflex action. . . . . The morbid process which produces these symptoms may ascend and reach the spinal cord, where it leaves distinct traces."

Almost with certainty we may conclude that a case which M. Bablon[1] describes in some detail was of the same nature as those which form the subject of the present communication. Bablon, however, gives it as a case of acute ascending paralysis. In doing so he apparently errs.

In 1876 Eichhorst published[2] details of a case which he diagnosed as "acute progressive neuritis." The patient, a woman æt. 66, was suddenly seized with complete paralysis of the left superficial peroneal nerve, ushered in by severe boring pains, and accompanied by feelings of numbness, formication, profuse sweating, and followed by complete anæsthesia. Other nerves of arms and legs were subsequently seized upon by the morbid process, so that at length the patient lay completely paralyzed in both upper and lower extremities. Complete blindness set in forty-eight hours before death. At the autopsy it was found that while the brain and cord were normal, the peripheral nerves were extensively degenerated.

A case described by Eisenlohr[3] in 1879 resembles those I have already detailed to the Society, in that while brain and cord were found after death to be normal, the nerves were the seat of degenerative changes. It differs, however, in this important particular, viz., that no anæsthesia existed over the affected regions, but, on the contrary, there was hyperæsthesia of the skin on the soles of the feet. In other respects the symptoms closely resemble those of our cases.

In 1879 Joffroy published[4] a most important contribution to the history of neuritis, which contains records of various cases. The most important of these was that of a woman, æt. 33, suffering from phthisis. On admission to the hospital it was seen that her lower limbs were paralyzed. This paralysis gradually spread upwards, but there was no abnormality in the sensibility to touch, heat, or pain. The movements of her hands were not ataxic, but she had lost all sense of position in the lower limbs, could not point to her toe or heel when her eyes were shut, or even know whether her legs were crossed or not. About twenty days after admission her arms became similarly affected. She died of phthisis, and on post-mortem examination the brain and cord were found to

[1] *Gaz. Hebd.*, Dec. 1864. [2] Virchow's *Archiv.*, Bd. 69.
[3] *Centralblatt f. Nervenheilkunde*, 1879.
[4] *Archives de physiologie*, vol. vi. p. 172.

be healthy, while the nerves were degenerated. Joffroy points out and lays weight upon the fact that the sensibility was unaffected, and argues in favour of the parenchymatous nature of the inflammatory process, for in the case of interstitial neuritis the inflammation would not have attacked only the motor fibres of a mixed nerve, leaving the sensory fibres untouched. It is, however, difficult to allow M. Joffroy his premises, for it may be asked whether the sensibility could be perfect in the legs when she was unable to tell whether they were crossed over one another or not without looking at them.

In an able and most valuable paper published by Professor Leyden in the *Zeitschrift für Klin. Medizin* in the end of last year, he gives a general *résumé* of the subject and a detailed account of two cases in which he had opportunity of making post-mortem examinations. The first was in a sailor, æt. 28, who was admitted to the wards in February 1878. He had had fever and sharp tearing pains for some days in arms and legs, apparently proceeding from knees and elbows. The soft parts below knees and elbows were swollen and white, very sensitive on pressure. In the fingers and toes was a painful feeling of formication and diminution of sensibility. Use of the limbs was markedly diminished—a kind of lameness. After some days the fever diminished, also the fulness and sensitiveness, but instead there came distinct muscular atrophy, especially in the extensor muscles of the arm. In the course of some months the legs got quite well, but the arms got worse —claw-like hands suggesting lead-poisoning, while the muscles showed the reaction of degeneration. Patient died of atrophy of kidney a year after illness commenced. The nerves of the affected part were found to be in a state of sclerotic atrophy. The anterior spinal roots were normal, as was also the cord and the nerves of the lower extremities. He gives in great detail another case of a merchant who, two years after the commencement of certain other nervous symptoms, became affected with paralysis of motion and sensation in hands and feet, corresponding, as he remarks, to the subacute spinal paralysis of Duchenne. The autopsy showed degenerative multiple neuritis, and no affection of the spinal cord itself beyond a little swelling of some of the ganglion cells here and there, with vacuoles.

Such are, I believe, the only cases on record corresponding to those which I have brought before you.

From the facts observed in the cases which I have recorded, taken along with those recorded by others, and with some which, looking back, I now regard as examples of this condition, I find data for drawing up a clinical history of the disease, which seems to me quite distinctive. Its commencement is usually acute and attended by more or less fever. While all the functions of the nerves speedily become affected, it is in connexion with

the sensory functions that the first changes manifest themselves. Sometimes there is acute pain, but oftener a numbness or peculiar tingling sensation in the affected parts, closely resembling the feeling popularly known as sleeping of the limb —a feeling which is more like this from the circumstance that action of the muscles or pressure upon the skin induces the uneasy or painful sensation commonly known as pins and needles. Along with this there is a distinct diminution of sensibility. Touch is felt indistinctly; two points so far removed from one another as to be distinguished in the healthy conditions are no longer distinguished, and the patient may have the greatest difficulty in localizing the impression which he feels. Sensory impressions are conducted slowly, and at the same time contact is felt painful. These feelings may begin simultaneously in the fingers and in the toes, or may affect first the one and then the other. It seems to be usually in both hands and both feet simultaneously, but not necessarily in an equal degree.

To these sensory changes motor symptoms speedily become superadded. At first there is mere paresis, and affecting the most distal parts; but the paresis spreads up the limb from one group of muscles to another, and as it does so the intensity of the process deepens in the parts first affected. The organic reflexes are very rarely affected, although when the disease spreads upwards it is sometimes found that those connected with the bladder, and perhaps the bowel, are involved. The skin reflexes are modified in proportion to the diminution of sensibility in the parts, being sometimes entirely absent, sometimes absent on slight stimulation, but present even in an exaggerated degree when a strong stimulus is applied. The plantar reflex is often altered, while those higher in the body are natural. The tendon reflex, and especially the patellar tendon reflex, appears to be early and completely lost in these cases. Ankle clonus may be present after the patellar tendon reflex is lost. With regard to voluntary motion, it is found that in some of the muscles it is absolutely lost; in others it is diminished to a greater or less extent. Within a week of the commencement of the seizure it may be found that the patient has no power of flexing or extending the toes and fingers, but retains power of movement of the ankles or the wrist. A week or two later these movements also have become lost, and ere long perhaps the legs and arms in their whole extent are absolutely helpless. There is no special interference with the co-ordinating functions. With regard to the reaction of degeneration, I have not been able to satisfy myself in the cases which I have observed.

The vasomotor and trophic changes manifest themselves most distinctly in the muscles which undergo atrophy—much more rapidly than would be the case in simple motor paralysis. The colour and texture of the skin, also, sometimes change from the normal, patches of congestion or of blueness appearing here and

there, and glossiness manifesting itself, especially in the fingers. The nutrition of the nails also becomes altered. In several of the cases I have seen a very distinct degree of œdema not referable to any other than a nervous cause. The intelligence is perfect, and sleep is satisfactory except in so far as it may be disturbed by pain. There is no alteration of the condition of the spine or cranium.

The process may take some weeks, or perhaps months, to arrive at its full development. After a time it appears usually to become arrested, but at first no improvement is manifested. Gradually, however, the patient begins to notice some improvement. His pains or uneasy feelings diminish, sensitiveness to impressions increases, and he begins to feel that his power over the muscles is returning. It is in the upper part of the limb that improvement first sets in. It gradually passes downwards, until at length there is complete recovery. The process may occupy a period of from two to six or more months.

The recovery from such a condition as this seems to me most remarkable. It would appear to one examining the lesion as if it must necessarily permanently destroy the nerve; and yet we must assume that recovery does actually take place, and that in no inconsiderable proportion of the cases. But what is even more remarkable is, that the process may recur in the same individual and in the same parts time after time. I have met with at least one case in which this apparently occurred three successive times within a few years.

In some cases, probably, the disease extends to involve the cord, and perhaps to pass into myelitis. In Case II. it seemed, from the girdle-pain and the bladder symptoms, that this was to some extent the case, for we must either assume that it was so, or that the nerves of these parts were affected in the same way as those of the hands and feet; and in Case III., as I have shown you, some extension of the disease to the cord existed, although merely of the kind which is described as secondary degeneration. In the second case recorded by Duménil distinct changes were present; and in one of Leyden's cases there was a little swelling of some of the ganglion cells in the cord, with vacuole formation. It remains to be ascertained from observation of future cases whether this is a frequent result or not.

The rapidly fatal result of the pneumonia in Case III. shows that the superaddition of any acute disease must be regarded as most formidable where this disease exists. Should I meet with another case of the kind, I should investigate most carefully the condition of the nerves supplying the inflamed part.

But while recovery occurs in a considerable proportion of cases, it is not the invariable result. In some the process induces permanent atrophy of the nerves, with consequent paralysis. In others extension of the disease may take place, and vital nerves or vital nerve centres becoming affected, death must follow. Further

observation will show the proportions in which these various results occur.

I have no doubt that it will occur to many of you that probably in this disease we find the means of explanation of cases which we have regarded vaguely as spinal congestion, slight myelitis, or such like; and I expect that it will prove that cases of this kind are not very uncommon. I, at least, on looking back, can recall some in which I think I should now find the explanation by reference to this process.

If it be true that in some instances fibres of all kinds in mixed nerves may be thus affected, it seems reasonable to believe that in individual mixed nerves either the motor fibres alone, or the sensory fibres alone, might be the seat of change. And if so, we should expect to find in the former a clinical history closely resembling Landry's acute ascending paralysis; in the latter, certain forms of neuralgia with loss of sensibility.

It remains for me to mark off the clinical history thus sketched from those proper to some other affections with which, at first sight, it might be confounded. It resembles at first sight and in some respects the Acute Ascending Paralysis to which I have just referred, which was first described in 1859 by Dr Landry. In it there is paralysis commencing at the distal parts and spreading upwards, sometimes terminating in recovery, sometimes extending to vital centres and so proving fatal. In the cases which do prove fatal no lesion has been found. The disease which I am bringing before you to-night differs from it in that it affects the sensory as well as the motor functions, and exhibits well-marked pathological changes. But the processes may, on further examination, turn out to be related to one another in the way I have just suggested. It also very naturally suggests the form of disease which was described in 1853 by Duchenne under the name of General Spinal Paralysis, and which he afterwards spoke of as Anterior Spinal Paralysis, acute and subacute, in which the lesion was more accurately defined by Charcot and Joffroy, and which has been termed by Kussmaul and Erb, Poliomyelitis anterior, by Westphal, Acute Atrophic Spinal Paralysis of adults, and by Eulenburg, Acute and Subacute Spinal Paralysis of adults. In that disease the lesion is situated in the anterior horns of gray matter, and the clinical features are well defined. Commencing generally acutely with some degree of febrile disturbance, there is developed paresis of the limbs, which rapidly passes into paralysis with speedy wasting of the muscles. It spreads from the legs upwards. The same process may occur in the arms; it may, indeed, begin in the two places simultaneously gradually advancing in the trunk as the cord becomes more extensively affected. The process may extend to vital structures and prove fatal, or it may become arrested and complete recovery take place, but throughout the course of the disease there is no affection

of the sensory functions. Peripheral Paralysis of the kind we are describing differs from it in that it affects the sensory as well as the motor and trophic functions, and that it is so distinctly ascending in the limb it involves, while pathologically the changes are essentially different; and thus, although one of the most careful and distinguished of living workers in neurology, Professor Leyden of Berlin, draws special attention to the resemblance between them, it is yet clear that they are to be readily distinguished from one another.

There should be little tendency to associate this disease with Acute Transverse Myelitis, a malady in which the whole of the strands of the cord are diseased at the same point, for in that disease it commences to manifest itself at once at all points below the seat of disease, and does not spread upwards, as in our malady; moreover, in myelitis there is a marked tendency to alterations of the organic reflexes, to sloughing of skin, alkalinity of urine, and vesical catarrh, none of which occur in the malady we are considering.

Certain forms of diphtheritic paralysis more or less closely resemble the disease under consideration; but they may be readily distinguished by the fact that there is no history of any throat affection, no tendency to paralysis of laryngeal or pharyngeal muscles, nor is the distribution of the malady at all like what is seen in post-diphtheritic cases. It is interesting to remember in this connexion that in some cases of diphtheritic paralysis Charcot and Vulpian have found a distinct lesion of the nerves of the affected parts, and that this has been confirmed by others. It will be interesting to ascertain how close the resemblance between them may be.

One of the main points of interest in connexion with this disease is the light which it appears fitted to throw upon some obscure questions in nervous pathology; among these I may mention Locomotor Ataxia. You are all aware how frequently that disease is heralded by passing nervous attacks of various kinds, sometimes paralytic, affecting one nerve or some particular branch of a nerve, and generally after a time disappearing, but sometimes persisting. Such paralyses may be best explained by assuming them to be due to this disease. But, again, girdle-pains often appear as an early symptom, and may be also quite reasonably referred to such a malady, for the girdle-pain is usually associated with hyperæsthesia, and while it sometimes entirely disappears, it sometimes also produces a permanent anæsthesia of the region affected. Another very distressing symptom is that which is known as "lightning pains," which may be due to disease of the sensory fibres; and in cases of locomotor ataxia it must have struck many observers how strangely pain, hyperæsthesia, and anæsthesia may come and go in certain parts in a way that seems scarcely explicable unless on the hypothesis of local nerve change. In

regard to the eye changes, the temporary and passing amauroses, and other symptoms connected with special sense, I may refer to some facts recorded by Dr Althaus.[1] Having referred to the optic neuritis as being well known in association with Locomotor Ataxia, he gives cases in which the olfactory and auditory nerves were the seat of disease, resulting in alteration or destruction of the functions of these nerves.

It is interesting to know that while, from clinical considerations, I was led to this view of these symptoms, Dr Hamilton has been led in the same direction by his pathological observations. It has, indeed, long been known that atrophy of cranial nerves occurs in the course of locomotor ataxia, but what I have brought before you to-night is fitted to clear up our conceptions on the matter.

Another point of practical interest is the light that this may be fitted to throw on the beneficial effects of nerve-stretching. The demonstration of such changes as these may be followed one day by proof that some allied change exists in the axis cylinder of nerves in the cases which are benefited by nerve-stretching. And taking together what we have said regarding locomotor ataxia and this plan of treatment, the thought will naturally occur that we may herein find the explanation of the marvellous results of nerve-stretching recently described as having occurred in that disease.

With regard to treatment I cannot as yet speak very positively. Certainly strychnia seems injurious in the early stage and beneficial in the later, while ergot of rye seems to be useful in the early periods. It remains to be seen whether nerve-stretching is applicable or not at any stage. Many remedies may be useful for relieving pain — quinine, salicylic acid, salicylate of soda, morphia. During the period of advance the patient should be kept at rest. When the acute stage has passed, friction, electricity, passive and then active exercise, should be carefully tried.

[1] Althaus "On the Pathology of Peripheral Nerve Disease," *American Journal of the Medical Sciences*, 1879.

EDINBURGH : PRINTED BY OLIVER AND BOYD.