Illustrations of some less-known forms of peripheral neuritis, especially alcoholic monoplegia, and diabetic neuritis / by Thomas Buzzard.

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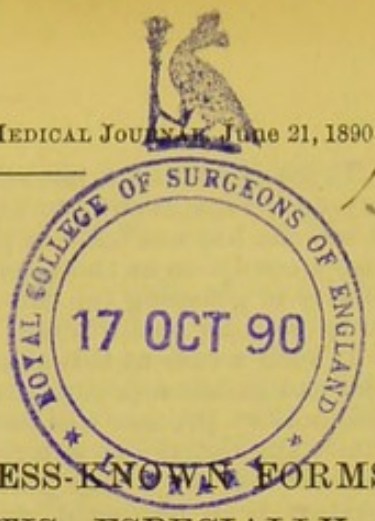

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ILLUSTRATIONS OF SOME LESS-KNOWN FORMS OF PERIPHERAL NEURITIS, ESPECIALLY ALCOHOLIC MONOPLEGIA, AND DIABETIC NEURITIS.[1]

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As is now widely known, it is in the form of multiple neuritis that the toxic influence of alcohol is very prone to be exerted on the nervous system. Cases of alcoholic paralysis of this kind are not at all uncommon, and occur more frequently in women than in men, and occasionally even in females of good social position.

Let me first very briefly sketch a case of alcoholic polyneuritis, as it most commonly presents itself when seen in consultation.

The patient is lying in bed, unable to stand, able to flex the thighs upon the pelvis fairly well, and possibly also to bend the knees, but with still greater difficulty. The feet are "dropped," that is, they lie flaccidly in a position of over-extension, and the patient is unable, when requested, to dorsal-flex them. The knee-jerks are absent. The muscles of the legs, especially those on the anterior surface below the knee are probably atrophied, and are found to yield no response to induced currents of electricity. The arms are thin and the thenar and hypothenar eminences may be found atrophied. There is more or less "wrist drop," so that the patient presents the appearance of one suffering from lead palsy. The extensor muscles in the forearm, as well as the intrinsic muscles of the hand, may not contract to faradic currents.

On the sensory side we may expect to hear of pains, which are often of lightning character, coming and going in sudden darts, like stabs of a knife or the boring of a gimlet, and quite recalling those which are characteristic of tabes dorsalis. Or they may be described as gnawing, or burning, or pricking. They are usually most pronounced in the lower extremities. It is commonly found that great tenderness of the muscles is complained of when these are grasped by the hand. The patient herself will sometimes describe a sensation of aching in the muscles, and very commonly indeed a feeling of "numbness," "deadness," or "pins and needles," which is referred especially to the hands and feet. More or less cutaneous anæsthesia is found in the feet and legs. As a general rule the functions of the bladder are not disordered, and there is no tendency to bedsores.

[1] Read at a meeting of the East Surrey District of the South-Eastern Branch of the British Medical Association.

There is usually a remarkable loss of memory, the patient often saying for instance that she has been out for a walk on that day, when she has not left her room for many weeks. There is often also a condition as though of complacent indifference and incapability of grasping the full meaning of her condition, which seems to be peculiar to a state of chronic alcoholism.

If such a case as this should give the opportunity of *post-mortem* examination (a comparatively rare exception) a subacute inflammatory process will be found affecting the nerve trunks, especially the radial and anterior tibial or the peroneal. The nerves will seldom show any particular change to the naked eye, but, having been hardened and submitted to section, evidence of parenchymatous degeneration is discovered in them, together with more or less interstitial neuritis.

In females affected with alcoholic neuritis I have observed now, during many years, that the catamenia are almost always suppressed, and often for many months during the illness. I do not attempt an explanation of this. It seems to me a curious and interesting fact when taken in conjunction with the well-known complete, or almost complete, immunity of the functions of the bladder and rectum.

Although in the majority of cases it is the nerves of the extremities which appear to bear the brunt of the lesion, yet I have seen paralysis of the respiratory muscles and of those subserving deglutition, and in one instance almost complete paralysis of the external muscles of the eyeballs. The pneumonia which occasionally terminates the scene in cases of this kind is very likely due to lesion of the vagus.

I have given an illustration of the more usual symptoms in a case of multiple neuritis due to alcohol. It is necessary to mention exceptional conditions which are found in certain cases. There may be no pains, and but very little or indeed no disturbance of cutaneous sensibility. It seems that in some rare instances it is almost entirely the efferent fibres which suffer. There is often a very considerable amount of œdema of the feet and legs, and the hands may look puffy and sodden. Indeed, I have seen one case in which the brunt of the lesion appeared to fall mostly on the vasomotor system of nerves, and give rise to œdema. In some cases it is chiefly a tottering ataxic gait which is noticeable. This often precedes the paralytic state, which may and often does arrive quite suddenly.

Although a dropped condition of the feet is so common as to be highly characteristic of alcoholic paralysis, and should always suggest inquiries into the history, yet I have lately seen a few cases in which the symptom was not present. In these, it is true, the amount of paralysis of the lower extremities was not very great, but as the loss of power in the anterior tibial muscles is commonly an initiatory as well as a very characteristic symptom, it seems worth while to note that it may occasionally fail to occur, at least early in the case.

A case which I have seen this winter illustrates some of these exceptional conditions. A gentleman who had greatly exceeded began in September to lose power in the knees and ankles, at the same time complaining of pains in the muscles of his legs. He managed to attend office for a month, then one day when dressed and ready to start, his legs gave way under him and he could not get up. He then began to lose power in the hands, which swelled and were numbed. They remained bad for a short time only. When I saw him two months after his attack he had not been able to walk or even to stand. His legs were swollen, he could not

dorsal-flex the feet. There were no pains nor anæsthesia nor tenderness of muscles. The knee jerks were absent. He made a rapid recovery, and in three months was almost well. When I last saw him both knee-jerks had returned.

The lower extremities as a rule are more severely affected than the upper. It occasionally happens, however, that when the patient comes under observation recovery has taken place in all except one limb, and then there is a chance, unless care be taken, of a mistake in the diagnosis of the case. A lady was seen by me in consultation in April, 1888. She was suffering from loss of power in the left hand. She had no complaint to make of her legs or of the right arm. There was wasting of the thenar and hypothenar muscles of the left hand with loss of faradic reaction. The skin was red and polished looking. The catamenia had been absent for some months.

There was exquisite sensitiveness to touch at the tips of the fingers and in the palm, and if the wrist-joint were moved it caused her excruciating pain. The hand and arm presented the helpless look of a fractured limb. The fingers were white and puffy, the nails overgrown because she could not bear to have them cut. Great tenderness was experienced on pressing the median nerve at the wrist. The right thumb, and two fingers were sensitive at the tip, and the hand was weak; it had been still more helpless than when I saw it.

In January the patient had begun to feel as if she had "rheumatism," and her fingers became over-sensitive. At that time she was very weak.

In February her knees would give way, so that she fell more than once on her wrists. At that time she was walking with the help of a stick or someone's arm on account of "rheumatism" in her knees. She dragged her feet. She was treated with induced electrical currents, and the hand gradually improved.

On May 3rd, after the hand had been placed in hot water for the purpose of faradism, it became purplish red and swollen. I have often noticed this to happen in cases of neuritis. The cuticle will often scale off.

On May 26th, adhesions in the wrist and finger-joints were broken down under chloroform. Under faradism, massage, and passive movements she slowly improved, and I heard subsequently that the arms had got quite well. It should be said that there was a distinct history of excessive drinking in this case.

In the following case neuritis (presumably from alcohol) was confined from the first to the district of a single muscle.

A gentleman, aged 29, was sent to me by his medical attendant with paralysis of the index, middle, and, to a less extent, of the ring finger of the right hand, which had been observed for three weeks. The patient was a paroxysmal drinker, and had just come out of a debauch of several weeks. The hand presented just the appearance of an early stage of wrist-drop from lead. There was no blue line, and no history of exposure to that metal. The faradic excitability of the extensor communis of the right arm was lessened and A S Z > K S Z. The hand felt numbed. There had been no pain in the arm. There was no sign of lesion in the other arm or in the lower extremities. The knee-jerks were present and equal. Under treatment, chiefly by abstinence and with slight application of electric currents, he quite recovered in six or seven weeks.

In another example the lesion was confined to the brachial plexus of one side.

A gentleman, aged 22, came to me from a medical man in the

country. His right arm hung helpless. It was exquisitely tender to the touch, full of pain, and presented a sodden appearance. Pains had begun in the arm some weeks previously. For some two or three years, I was informed, his habits had been most intemperate, and he had been on the verge of *delirium tremens* when the attack of brachial neuritis began. At first its occurrence appeared to check the development of the symptoms of delirium, but when he returned home he was in a half maniacal condition which prevented an exact examination of the arm being made. He had been then treated with salicylates, bromides, iodides, arsenic, and quinine. Under antipyrin, grains 25, and extract of Indian hemp, grain ¼, three times a day, he soon began to have less pain and tenderness. In a fortnight he looked very much better, and was free from pain. I heard later that he had quite recovered.

A female patient was brought to me on account of loss of power in the right hand and greatly impaired vision. I found the right grasp diminished in power. The two middle fingers were especially weak, and tended to drop. There was no cutaneous anæsthesia. She had suffered from what she called "rheumatism"—sharp shooting pains in the feet and across the instep for years, but had had no pain in the arms. In the course of the last six years she had had on three occasions an epileptic fit. The difficulty of vision was peculiar. At first it strongly suggested hemiopia. Looking at the word "holding," she could read "hold" only. After a pause and apparently readjusting her gaze, she saw the remainder of the word. Facing me, she could see my right eye clearly, my left but very indistinctly. Yet on holding up my fingers, and testing the field of vision, I could discern no impairment of the extent to which she could see to her right and left. It appeared, therefore, that she had a scotoma just to the right of the centre of the visual field. Her tongue appeared slightly inclined to her right. This last symptom, coupled with the suggestion of hemiopia, paresis of the right arm, and the history of fits, might easily lead to a diagnosis of intracranial disease. But close investigation did not confirm this.

The extensor communis muscle of the right arm showed distinct lowering of excitability to both faradism and galvanism, but especially to the latter. This could not result from an intracranial lesion. It pointed to an affection of the "spinal" nervous system, and was most probably due to peripheral neuritis. (The word "spinal" connotes, of course, the spinal nerves as well as the cord.) There was a history of alcoholism. I was disposed to refer the scotoma to periaxial neuritis of the optic nerve. The patient recovered completely in a few weeks.

I saw quite recently in consultation a lady, aged 64, who a fortnight previously had entirely lost the use of the right arm. Investigation of the cause of this monoplegia excluded a traumatic origin. The tendon reflexes in the other arm and in the legs were good. In the right arm they were absent. There was total inability to move any muscle of the right upper extremity, including the shoulder, together with anæsthesia of the hand, which decreased shortly above the wrist. Pain of excruciating character, especially bad at night, was described as affecting the right arm. It was as though the "limb were being torn out of its socket on a rack." I had not an opportunity of testing the faradic excitability of the muscles, which were very flaccid, but the other symptoms are sufficient to show that the lesion was probably a neuritis of the brachial plexus. The patient was described as not having been sober for a year past.

These examples, I have thought, would prove interesting and valuable, as tending to show what has not yet, so far as I know, been advanced respecting the influence of alcohol, that its effects may appear, not only in the more common form of a generalised paralysis of the extremities, but in lesion of a peculiarly localised character. If I am right in the view that these are examples of localised neuritis due to alcohol, it is evident that we shall have in future to bear this possibility in mind when we meet with neuritis of single nerves or plexuses, of the cause of which we do not find a ready explanation.

Let us turn now to peripheral neuritis arising from other causes than the influence of alcohol. A week or two after I had published a work on *Paralysis from Peripheral Neuritis* the following case came under my observation at the hospital. It is one of multiple neuritis occuring in a patient affected with diabetes mellitus:—

J. K., aged 55, carpenter, applied to the National Hospital for the Paralysed and the Epileptic on June 30th, 1886, suffering from loss of power in the lower extremities, which he ascribed to working in the wet. He had been quite well until the previous September, when, after working daily for several weeks out-of-doors in the cold and wet, he began to get pain and tenderness in the front of the right thigh, which extended presently down the leg to the foot. The pain was very severe, and there was so much weakness that his knee often gave way and let him down. A month after the right the left leg was similarly attacked, and in three months from the first symptoms he could not walk at all. About this time he noticed also numbness and "pins and needles" in both legs, especially in the soles of the feet, and the pains were severe and continuous. The symptoms continued till the end of February, when the pains became less severe, and he became able to walk upon crutches. The improvement in his lower limbs continued, but towards the end of May he began to have a little tingling and slight numbness in the tips of his fingers. At no time was there any difficulty with his bladder, but soon after the commencement of his illness he was troubled with great thirst and noticed that he passed much urine, and, moreover, wasted in flesh a great deal.

On examination, the legs were found thin; they could be flexed readily upon the thighs, but extension was imperfectly performed. There was no power of dorsal-flexing the feet upon the legs. There was tenderness on pressure in the soles of both feet, and he complained of tingling and numbness from the feet up to the knees—more in the feet, ankles, and front of the legs than in the calves. There was much loss of cutaneous sensibility in the feet and lower part of the legs. The knee-jerks were absent. There was no response to the strongest induced currents in the anterior tibial group of muscles, and this painful application could be borne without inconvenience in that situation, whilst lower down it was only felt as pressure. It was remarkable, I may note by the way, that some immediate return of voluntary power in dorsal-flexing the feet followed this application, although the electric current itself caused no muscular contraction. Examination of the urine showed that it contained a large quantity of sugar.

The man was admitted as an in-patient a fortnight afterwards. Examination then showed the plantar reflex fair on the left, but hardly to be obtained on the right sole; the gluteal fair on the left, not well marked on the right; the cremasteric also better on the left than the right side. The abdominal and epigastric reflexes were present on each side. There was a ready and normal

response to induced currents in the muscles of the hands and arms, but still no reaction in the anterior tibial group; the calf muscles responded, though not perfectly, and the muscles of the thighs also responded to the induced current. A strong galvanic current was required to cause contractions in the anterior tibial muscles, but the response took place on the right side to twenty-eight cells with the positive-pole closure, as against thirty-four cells with the negative-pole closure. On the left side, too, the positive-pole closure contraction was more marked than the negative. On the other hand, in the right gastrocnemius only twenty-two cells were required with the negative pole, as against thirty-two with the positive. A fortnight later a very strong faradic current produced contractions in the anterior tibial group, but a much weaker current was sufficient to act upon the peroneal muscles.

When he left the hospital at the end of August, KSZ > ASZ in the anterior tibial group. There was still great loss of cutaneous sensibility in front of the legs.

Examination of the urine showed that it was passed in large quantities and of high specific gravity, as the following figures will indicate:—

July 25th, 74 ounces in the 24 hours, specific gravity 1045
„ 26th, 79 „ „ „ „ „ 1043
„ 27th, 94 „ „ „ „ „ 1042
„ 28th, 84 „ „ „ „ „ 1042

The urine contained sugar in abundance.

The patient was shown to the Harveian Society in November, 1886. After remaining as an out-patient during the winter and spring, he was again admitted into hospital in June, 1887, still complaining of great loss of power in both lower limbs, the movements of which were feeble. There was drooping of the toes of the left foot. Both legs were slightly wasted. The knee jerks and plantar reflexes were absent. There was continuous pain in both legs and feet, and some but less numbness than formerly. Sensation to touch was diminished below the knees. In the same region there was slight tenderness on pressure.

On the outer side of the right foot, just below the external malleolus, was a deep ulcer, the skin around it and on the outer side of the foot generally being much congested. This had begun as a small swelling, which had gradually increased in size, burst, and discharged a small amount of pus.

On the sole of the left foot, just below the metatarso-phalangeal joint of the great toe, was a cicatrix due to a similar condition which had arisen in connection with a corn in that situation in the February preceding.

Tactile sensation was diminished in both hands, and the patient complained of slight pain in both hands and the left forearm. There was pain in all the fingers of the left hand, and in the little and ring finger of the right hand. There was loss of power in both hands, especially in the left, and the power of flexing the elbow was much feebler on the left than on the right side. There was wasting of the thenar and hypothenar eminences of both hands, and of the interossei (especially of the first) in both hands; also slight wasting of the left forearm.

From the middle to the end of July the diet consisted in toast, green vegetables, meat, eggs; no potatoes, sweets, beer, rice, or other starchy food. The quantity of urine averaged 57 ounces per diem, and by the fermentation process the amount of sugar was estimated at an average of 25 grains per ounce of urine.

From the end of July to the middle of August soft bread was

substituted for toast, with the result that the urine averaged 83 ounces per diem, with 35 grains of sugar per ounce of urine.

From the middle of August until the middle of October gluten bread replaced the soft bread previously taken. The quantity of urine fell to an average of 54 ounces per diem, with only 16 grains of sugar per ounce of urine. *Pari passu* with this decrease of glycosuria, the patient gained weight, the ulcer healed, and when he left the hospital, on November 3rd, his condition generally was described as much improved. I have since lost sight of him.

When I first saw this man in the out-patient room I recognised his case as one of a class to which (in order to distinguish them from those resulting from disease of the cord) I have suggested the name of neuritic paraplegia. The basis or ultimate cause of the neuritis did not at once appear evident, as there was no alcoholic history. It was in an endeavour to discover this, and influenced by the man's description of thirst and wasting, that I examined the urine for sugar. Just at that time the occasional dependence of peripheral neuritis upon diabetes had been announced abroad, and I was much interested in meeting with a case in point. The occurrence of perforating ulcers in this case is very interesting. From this, together with the absence of knee-jerks, and the pains, the case might readily, but for the discovery of the sugar and the existence of muscular atrophy, have been set down at first sight to one of tabes dorsalis, with which it had much in common.

In another case, to which I need only very briefly refer, symptoms of pains and weakness of the extremities, with absence of knee-jerks, had been ascribed to tabes. In that instance I discovered a large quantity of sugar, and under a partially restricted dietary there was remarkable improvement in the symptoms and the knee-jerks returned. The symptoms in this case had never attained the severity observed in the preceding one. There had been no considerable loss of power and the muscles showed no atrophy. In another, seen recently in consultation, a diabetic patient suffered from excruciating pain in the lower extremities with loss of knee-jerks. In the following example the paralysis and atrophy were strongly pronounced and much more localised than in the first example.

A gentleman, aged 60, was sent to me in October, 1887, with the left arm hanging helpless and its muscles wasted. He told me that he was suffering from diabetes and had had much gout. Sugar had been discovered six years, and he passed about 15 grains to an ounce. Albumen had been observed eighteen months, and it measured about a twentieth. The son of a gouty father, he had always drunk beer and port wine, and began to show gout at 40 years of age. His appearance was that of a man who had lived freely. His heart sounds were feeble, and he had an aortic systolic *bruit*. He passed in the twenty-four hours about 6 pints of urine, usually of a specific gravity of 1025. This was on a partially restricted dietary. On ordinary diet it would rise to 1040 or more. He was taking six ounces of whisky daily, besides some wine. His weight, which had been formerly 18 st. 10 lbs., was then about 14 stones.

Examination showed wasting of all the muscles of the left shoulder and arm, except the flexors of the wrist and fingers. In these only reaction to the induced current persisted, although it was diminished. In the wasted muscles there was none. There was much œdema of the hand, and the sensibility of the skin of the arm was much diminished. Only the left shoulder and arm

appeared to be affected. The atrophy had been preceded by great gnawing pain about the shoulder, with shooting down the arm, which had begun, according to the patient's account, about a month previously. There were no knee-jerks.

When I saw this patient five months later, he could lift the shoulder and elbow, which he had not been able to do when he was first examined, but electrically there was no improvement. There was no reaction to the strongest induced current in the muscles of the shoulder, arm, and extensor side of the forearm; the flexors of the wrist and fingers required also a strong current. The skin of the shoulder of the right side was but little sensitive to induced currents, and he had been complaining of pain in that situation.

In June, 1889, I again saw this patient in consultation at his own house. He was then suffering from congestion of the base of the left lung. Both his arms were now very incapable.

I recently saw a female whose gait was shambling, and who could scarcely use her hands. There was a history of pains in the feet, gradually extending up the legs, of a sharp character, from which she had suffered for three years. She had no knee-jerks. Pupils reacted to light. There was anæsthesia below the knees. She could dorsal-flex the feet, but the legs were very weak. Her memory was good. There was no appearance or history of alcoholism. I suggested to her medical attendant to examine the urine for sugar. He found it of specific gravity 1042 and highly charged with sugar.

It appears from a case recently published by Dr. Althaus that the neuritis in diabetes may be limited to a single nerve.