

ous bands of fibrous tissue, and is stripped off from the proper capsule with the greatest difficulty. Such a condition probably produces no symptoms.

**Symptoms.**—There may be intense pain, aggravated by pressure, in the lumbar region. In other instances, the onset is insidious; there is no pain in the renal region, but on the first examination signs of deep-seated suppuration may be detected. On the affected side there is usually pain, which may be referred to the neighborhood of the hip-joint or radiate down the thigh and be associated with retraction of the testis. Sometimes the pain is referred even to the knee-joint, as in hip-disease. The patient lies with the thigh flexed, so as to relax the psoas muscle, and in walking throws, as far as possible, the weight on the opposite leg. According to Gibney, the patient keeps the spine immobile, assumes a stooping posture in walking, and has great difficulty in voluntarily adducting the thigh.

There may be pus in the urine if the disease has extended from the pelvis or the kidney, but in other forms the urine is clear. When pus has formed there are usually chills with irregular fever and sweats. On examination, deep-seated induration is felt between the last rib and the crest of the ilium. Bimanual palpation may reveal a distinct tumor mass. Edema or puffiness of the skin is frequently present.

The diagnosis of perinephric abscess is usually easy, and in any case when doubt exists the aspirator needle should be used. We cannot always differentiate the primary forms from those due to perforation of the kidney or of the bowel. This, however, makes but little difference, for the treatment is identical. It is usually possible by the history and examination to exclude disease of the vertebra. In children the condition is often mistaken for disease of the hip-joint, but the pain is higher, and there is an entire absence of fulness and tenderness over the hip-joint itself.

From whatever cause produced, the indications for treatment are identical—early, free, and permanent drainage.

## SECTION VIII.

### DISEASES OF THE NERVOUS SYSTEM.

#### I. DISEASES OF THE NERVES.

##### I. NEURITIS (*Inflammation of the Nerve Fibres*).

Neuritis may be *localized* in a single nerve, or *general*, involving a large number of nerves, in which case it is usually known as *multiple neuritis* or *polyneuritis*.

**Etiology.**—*Localized neuritis* arises from (a) cold, which is a very frequent cause, as, for example, in the facial nerve. This is sometimes known as rheumatic neuritis. (b) Traumatism—wounds, blows, direct pressure on the nerves, the tearing and stretching which follow a dislocation or a fracture, and the hypodermic injection of ether. Under this section come also the professional palsies, due to pressure in the exercise of certain occupations. (c) Extension of inflammation from neighboring parts, as in a neuritis of the facial nerve due to caries in the temporal bone, or in that met with in syphilitic disease of the bones, disease of the joints, and occasionally in tumors.

*Multiple neuritis* has a very complex etiology, the causes of which may be classified as follows: (a) The poisons of infectious diseases, as in leprosy, diphtheria, typhoid fever, small-pox, scarlet fever, and occasionally in other forms; (b) the organic poisons, comprising the diffusible stimulants, such as alcohol and ether, bisulphide of carbon, and naphtha, and the metallic bodies, such as lead, arsenic, and mercury; (c) cachectic conditions, such as occur in anæmia, cancer, tuberculosis, or marasmus from any cause; (d) the endemic neuritis or beri-beri; and (e) lastly, there are cases in which none of these factors prevail, but the disease sets in suddenly after overexertion or exposure to cold.

**Morbid Anatomy.**—In neuritis due to the extension of inflammation the nerve is usually swollen, infiltrated, and red in color. The inflammation may be chiefly perineural or it may pass into the deeper portion—*interstitial neuritis*—in which form there is an accumulation of lymphoid elements between the nerve bundles. The nerve fibres themselves may not appear involved, but there is an increase in the nuclei of



may be only hyperæsthesia with soreness and stiffness of the limbs; in some cases, increased sensitiveness with anæsthesia; in other instances the sensory disturbances are slight. The clinical picture is not to be distinguished, in many cases, from Landry's paralysis; in others, from the subacute myelitis of Duchenne. James Ross concludes from an analysis of all the reported cases of the former disease that it coincides with multiple neuritis in general etiology, symptoms, and course. On the other hand, Hun, in a very thorough study of a recent case of Landry's paralysis, concludes that it is a separate and distinctive disease.

The course is variable. In the most intense forms the patient may die in a week or ten days, with involvement of the respiratory muscles or from paralysis of the heart. As a rule in cases of moderate severity, after persisting for five or six weeks, the condition remains stationary and then slow improvement begins. The paralysis in some muscles may persist for many months and contractures may occur from shortening of the muscles, but even when this occurs the outlook is, as a rule, good, although the paralysis may have lasted for a year or more.

(2) *Recurring Multiple Neuritis*.—Under the term *polyneuritis recurrens* Mary Sherwood has described from Eichhorst's clinic two cases in adults—in one case involving the nerves of the right arm, in the other both legs. In one patient there were three attacks, in the other two, the distribution in the various attacks being identical. There has recently been at my clinic a somewhat similar case—a man, aged thirty-one, who had, two and a half years ago, widespread paralysis, and who now has a second attack.

(3) *Alcoholic Neuritis*.—This, perhaps the most important form of multiple neuritis, was described in 1822 by James Jackson, Sr., of Boston, whose account of it is very graphic. Wilks recognized it as alcoholic paraplegia, but the starting point of the recent researches on the disease dates from the observation of Dumenil, of Rouen. Of late years our knowledge of the disease has extended rapidly, owing to the researches of Huss, Leyden, James Ross, Buzzard, and Henry Hun. It occurs most frequently in women, particularly steady, quiet tipplers. Its appearance may be the first revelation to the physician or to the family of habits of secret drinking. The onset is usually gradual, and may be preceded for weeks or months by neuralgic pains and tingling in the feet and hands. Convulsions are not uncommon. Fever is rare. The paralysis gradually sets in, at first in the feet and legs, and then in the hands and forearms. The extensors are affected more than the flexors, so that there is wrist-drop and foot-drop. The paralysis may be thus limited and not extend higher in the limbs. In other instances there is paraplegia alone, while in the most extreme cases all the extremities are involved. In rare instances the facial muscles and the sphincters are also affected. A case with this distribution recovered in my wards last year. The sensory symptoms are very variable. There are cases in which there are numbness and tingling only, without

great pain. In other cases there are severe burning or boring pains, the nerve trunks are sensitive, and the muscles are sore when grasped. The hands and feet are frequently swollen and congested, particularly when held down for a few moments. The cutaneous reflexes as a rule are preserved. The deep reflexes are usually lost.

The course of these alcoholic cases is, as a rule, favorable, and after persisting for weeks or months improvement gradually begins, the muscles regain their power, and even in the most desperate cases recovery may follow. The extensors of the feet may remain paralyzed for some time, and give to the patient a distinctive walk, the so-called *steppage* gait, characteristic of peripheral neuritis. It is sometimes known as the pseudotabetic gait, although in reality it could not well be mistaken for the gait of ataxia. The foot is thrown forcibly forward, the toe lifted high in the air so as not to trip upon it. The heel is brought down first and then the entire foot. It is an awkward, clumsy gait, and gives the patient the appearance of constantly stepping over obstacles. Among the most striking features of alcoholic neuritis are the mental symptoms. Delirium is common, and hallucinations with extravagant ideas, resembling somewhat those of general paralysis. In some cases the picture is that of ordinary delirium tremens, but the most peculiar and almost characteristic mental disorder is that so well described by Wilks, in which the patient loses all appreciation of time and place, and describes with circumstantial details long journeys which he has recently taken, or tells of persons whom he has just seen.

(4) *Multiple Neuritis in the Infectious Diseases*.—These have been already referred to, particularly in diphtheria, in which it is most common. The peripheral nature of the lesion in these instances has been shown by post-mortem examination. The outlook is usually favorable and, except in diphtheria, fatal cases are uncommon. Multiple neuritis in tuberculosis, diabetes, and syphilis is of the same nature, probably due to toxic materials absorbed into the blood.

(5) *Arsenical and Saturnine Neuritis*.—The arsenical neuritis is not common; only a single instance of it has come under my observation. No case to my knowledge has followed the use of Fowler's solution in my ward or dispensary practice, although I am in the habit of giving in chorea and anæmia doses which might be regarded as excessive. The most common causes are accidental poisoning, as in the cases reported by Mills. In a case of E. G. Cutler the patient got the arsenic from green-paper tags, which he was in the habit of putting in his mouth. The general symptoms are not unlike those of alcoholic paralysis; the weakness of the extensors is marked and the *steppage* gait characteristic. The neuritis due to lead will be discussed in the consideration of lead poisoning. The special involvement of the motor nerves and the great frequency of the occurrence of wrist-drop are the peculiarities of this form.

A similar form of neuritis is caused by the bisulphide of carbon.



(6) *Endemic Neuritis; Beri-beri*.—This is a widely spread disease in parts of India, and in China and Japan. To Sheube and Baelz are due the credit of determining its true nature. It is probably due to a micro-organism. Food appears also to have a large share in its causation and it has been attributed to a fish diet. Some have thought it might be due to the presence of parasites in the intestines, but there are no grounds for this belief. There are several types of cases. In the acute pernicious form the nervous phenomena are not so marked. There are fever, anæmia, and general anasarca. In another group of cases there are numbness, loss of tendon reflexes, areas of anæsthesia, and muscular atrophy and anasarca. In other cases the paralysis and atrophy are the most prominent symptoms and the clinical picture is that of a rapidly progressing multiple neuritis with sensory and motor disturbances. The mortality varies from three or four to fifty per cent. Great difference of opinion still prevails concerning the cause of the disease. Special interest has been aroused in the subject in this country, owing to the fact that J. J. Putnam has described a similar disorder among the New England fishermen who frequent the Grand Banks. It occurs in epidemic form, and has, as prominent symptoms, general cedema, shortness of breath, and sensory disturbances with paralysis. In other instances, the paralysis is more extensive and proves fatal. In 1881 and 1889 there were epidemics among the crews of vessels fishing in this region. Birge describes eleven cases which occurred on one vessel in a crew of thirteen, two of whom died. One patient of this crew I saw with F. C. Shattuck, in the Massachusetts General Hospital, with the well-marked symptoms of multiple neuritis. The disease also exists in the West Indies, whence cases have come to this country (Seguin).

**Diagnosis.**—The electrical condition in multiple neuritis is thus described by Allen Starr: "The excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost and then the muscles contract to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and thus far it is quite pathognomonic of neuritis. For in anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion."

"The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found and the negative pole produces stronger contractions than the positive pole. A loss of faradic irritability and a marked decrease in the galvanic irritability of

the muscle and nerve are therefore important symptoms of multiple neuritis." \*

There is rarely any difficulty in distinguishing the alcohol cases. The combination of wrist and foot drop with congestion of the hands and feet, and the peculiar delirium already referred to, is quite characteristic. The rapidly advancing cases with paralysis of all extremities, often reaching to the face and involving the sphincters, are more commonly regarded as of spinal origin, but the general opinion seems to point strongly to the fact that all such cases are peripheral. The less acute cases, in which the paralysis gradually involves the legs and arms with rapid wasting, simulate closely and are usually confounded with the subacute atrophic spinal paralysis of Duchenne. The diagnosis from locomotor ataxia is rarely difficult. The *steppage* gait is entirely different from that of tabes. There is rarely positive incoördination. The patient can usually stand well with the eyes closed. Foot-drop is not common in locomotor ataxia. The lightning pains are absent and there are no pupillary symptoms. The etiology, too, is of moment. The patient is recovering from a paralysis which has been more extensive, or from arsenical poisoning or has diabetes.

**Treatment.**—Rest in bed is essential. In the acute cases with fever, the salicylates and antipyrin are recommended. To allay the intense pain morphia or the hot applications of lead water and laudanum are often required. Great care must be exercised in treating the alcoholic form, and the attendant must not allow himself to be deceived by the statements of the relatives. It is sometimes exceedingly difficult to get a history of spirit-drinking. In the alcoholic form it is well to reduce the stimulants gradually. If there is any tendency to bed-sore an air-bed should be used or the patient placed in a continuous bath. Gentle friction of the muscles may be applied from the outset, and in the later stages, when the atrophy is marked and the pains have lessened, massage is probably the most reliable means at our command. Contractures may be gradually overcome by passive movements and extension. Often, with the most extreme deformity from contracture, recovery is, in time, still possible. The interrupted current is useful when the acute stage is passed.

Of internal remedies, strychnia is of value and may be given in increasing doses. Arsenic also may be employed, and if there is a history of syphilis the iodide of potassium and mercury may be given.

## II. NEUROMATA.

Tumors situated on nerve fibres may consist of nerve substance proper, the true neuromata, or of fibrous tissue, the false neuromata. The true

\* Lectures on Neuritis, Medical Record, New York, 1887.