times useful. No drugs have the slightest influence upon an acute myelitis, and even in subjects with well-marked syphilis neither mercury nor iodide of potassium is curative. Tonic remedies, such as quinine, arsenic, and strychnia, may be used in the later stages. When the muscles have wasted, massage is beneficial in maintaining their nutrition. Electricity should not be used in the early stages of myelitis. It is of no value in the transverse myelitis in the dorsal region with retention of the nutrition in the muscles of the leg.

The treatment of acute infantile paralysis has a bright and a dark side. In a case of any extent complete recovery cannot be expected; on the other hand, it is remarkable how much improvement may finally take place in a limb which is at first completely flaccid and helpless. The following treatment may be pursued: If seen in the febrile stage, a brisk laxative and a fever mixture may be given. The child should be in bed and the affected limb or limbs wrapped in cotton. As in the great majority of cases the damage is already done when the physician is called and the disease makes no further progress, the application of blisters and other forms of counter-irritation to the back is irrational and only cruel to the child.

The general nutrition should be carefully maintained by feeding the child well, and taking it out of doors every day. As soon as the child can bear friction the affected part should be carefully rubbed; at first once a day, subsequently morning and evening. Any intelligent mother can be taught systematically to rub, knead, and pinch the muscles, using either the bare hand or, better still, sweet oil or cod-liver oil. This is worth all the other measures advised in the disease, and should be systematically practised for months, or even, if necessary, a year or more. Electricity has a much more limited use, and cannot be compared with massage in maintaining the nutrition of the muscles. The faradic current should be applied to those muscles which respond. The essence of the treatment is in maintaining the nutrition of the muscles, so that in the gradual improvement which takes place in parts, at least, of the affected segments of the cord the motor impulses may have to deal with well-nourished, not atrophied muscle fibres.

Of medicines, in the early stage ergot and belladonna have been warmly recommended, but it is unlikely that they have the slightest influence. Later in the disease strychnia may be used with advantage in one or two minim doses of the liquor strychninæ, which, if it has no other effect, is a useful tonic.

The most distressing cases are those which come under the notice of the physician six, eight, or twelve months after the onset of the paralysis, when one leg or one arm or both legs are flaccid and have little or no motion. Can nothing be done? A careful electrical test should be made to ascertain which muscles respond. This may not be apparent at first, and several applications may be necessary before any contractility is

noticed. With a few lessons an intelligent mother can be taught to use the electricity as well as to apply the massage. If in a case in which the paralysis has lasted for six or eight months no observable improvement takes place in the next six months with thorough and systematic treatment, little or no hope can be entertained of further change.

In the later stage care should be taken to prevent the deformities resulting from the contractions. Great benefit results from a carefully applied apparatus.

III. Acute and Subacute Polio-myelitis in Adults.

An acute polio-myelitis in adults, the exact counterpart of the disease in children, is recognized. A majority, however, of the cases described under this heading have been multiple neuritis; but the suddenness of onset, the rapid wasting, and the marked reaction of degeneration are thought by some to be distinguishing features. Multiple neuritis may, however, set in with rapidity; there may be great wasting and the reaction of degeneration is sometimes present. The time element alone may determine the true nature. Recovery in a case of extensive multiple paralysis from polio-myelitis will certainly be with loss of power in certain groups of muscles; whereas, in multiple neuritis the recovery, while slow, may be perfect.

The subacute form, the *paralysie générale spinale antérieure subaiguë* of Duchenne, is in all probability a peripheral palsy. The paralysis usually begins in the legs with atrophy of the muscles, then the arms are involved, but not the face. Sensation is, as a rule, not involved.

IV. Acute Ascending (Landry's) Paralysis.

Definition.—An advancing paralysis, beginning in the legs, rapidly extending to the trunk and arms, and finally, in many cases, involving the muscles of respiration. It presents a remarkable similarity in its symptoms to certain cases of polyneuritis, with which it is now grouped by many writers.

Etiology and Pathology.—The disease occurs most commonly in males between the twentieth and thirtieth years. It has sometimes followed the specific fevers. An elaborate study of 93 cases collected from the literature has been made by James Ross, who concludes that in etiology, symptoms, course, and termination it conforms to a peripheral neuritis. Neuwerk and Barth have reached a similar conclusion. In their case an interstitial neuritis was found in the nerve roots, but the peripheral nerves were normal. On the other hand, cases have been reported of rapidly ascending paralysis in which the periphral nerves and nerve roots were unaffected. In a case of eleven days' duration recently studied by Hun, the lesions were certainly too slight to account for the advancing and wide-spread paralysis, and, with our present knowledge, Hun is cor-

rect in stating that "acute ascending paralysis—defined so as to exclude all cases in which the sensory symptoms are prominent, or in which well-marked bulbar symptoms are not present—must therefore be regarded as a clinical entity for which no corresponding lesion has as yet been discovered." It is not improbable that some toxic agent is responsible for the symptoms.

Symptoms.—Weakness of the legs, gradually progressing, often with tolerable rapidity, is the first symptom. In some cases within a few hours the paralysis of the legs becomes complete. The muscles of the trunk are next affected, and within a few days, or even less in more acute cases, the arms are also involved. The neck muscles are next attacked, and finally the muscles of respiration, deglutition, and articulation. The reflexes are lost, but the muscles neither waste nor show electrical changes. The sensory symptoms are variable; in some cases tingling, numbness, and hyperæsthesia have been present. In the more characteristic cases sensation is intact and the sphincters are uninvolved. Enlargement of the spleen has been noticed in several cases. The course of the disease is variable. It may prove fatal in less than two days. Other cases persist for a week or for two weeks. In some instances recovery has occurred, but in a large proportion of the cases the disease is fatal.

The *diagnosis* is difficult, particularly from certain forms of multiple neuritis, and if we include in Landry's paralysis the cases in which sensation is involved, distinction between the two affections is impossible. We apparently have to recognize the existence of a rapidly advancing motor paralysis without involvement of the sphincters, without wasting or electrical changes in the muscles, without trophic lesions, and without fever—features sufficient to distinguish it from either the acute central myelitis or the polio-myelitis anterior. It is doubtful, however, whether these characters always suffice to enable us to differentiate the cases of multiple neuritis.

IV. CHRONIC AFFECTIONS OF THE SPINAL CORD.

I. Spastic Paraplegia.

Definition.—Loss of power with spasm of the muscles of the lower extremities.

While clinically spastic paraplegia, or, as it is sometimes called, *tabes dorsalis spasmodique*, is a well-defined, readily recognizable affection, etiologically and anatomically it presents marked differences, and various groups must be separated, all of which present, however, the combination of spasm with loss of power. As the pyramidal tracts are involved, the term lateral sclerosis is sometimes used as the equivalent of spastic paraplegia. I shall consider the following forms:

(1) **Secondary Spastic Paralysis.**—After any tranverse lesion of the cord, whether the result of slow compression (as in caries), chronic myelitis, the pressure of tumor, chronic meningo-myelitis, or multiple sclerosis, degeneration takes place in the pyramidal tracts below the point of disease. The legs soon become stiff and rigid, and the reflexes increase. It happens occasionally, as Bastian has shown, that in compression paraplegia the limbs may be flaccid without increase in the reflexes—*paraplegie flasque* of the French. The condition of the patient in these secondary forms varies very much. In chronic myelitis or in multiple sclerosis he may be able to walk about, but with a characteristic spastic gait. In the compression myelitis, in fracture, or in caries, there may be complete loss of power with rigidity.

(2) **Primary Spastic Paraplegia.**—This is believed to depend upon a primary sclerosis of the lateral or pyramidal tracts. Clinically it is common to meet with cases in adults, particularly in syphilitic subjects, who have pains in the back, perhaps a girdle sensation, and a gradually developing, progressive spastic paraplegia. It may be impossible from the history or the physical examination to determine whether the condition is secondary to a transverse myelitis or a meningo-myelitis, or whether the lesion is a primary degeneration of the pyramidal tracts. The question is still debated whether a primary lesion of the lateral tracts ever takes place, or whether, in such instances, there is not always some lesion of the motor cells in the anterior horns. Cases may persist for years without any atrophy. In other instances there are signs of involvement of the posterior columns as well, forming the condition of ataxic paraplegia, which will be considered separately. So far as I know, the only case which is claimed to demonstrate the existence of a primary lateral sclerosis is that of Dreschfeld's, which occurred in 1881.

The *symptoms* of spastic paraplegia are very distinctive. The patient complains of feeling tired, of stiffness in the legs, and perhaps of pains of a dull aching character in the back or in the calves. There may be no definite loss of power, even when the spastic condition is well established. In other instances there is definite weakness. The stiffness is felt most in the morning. In a well-developed case the gait is most characteristic. The legs are moved stiffly and with hesitation, the toes drag and catch against the ground, and, in extreme cases, when the ball of the foot rests upon the ground a distinct clonus develops. The legs are kept close together, the knees touch, and in certain cases the adductor spasm may cause cross-legged progression. On examination, the legs may at first appear tolerably supple, perhaps flexed and extended readily. In other cases the rigidity is marked, particularly when the limbs are extended. The spasm of the adductors of the thigh may be so extreme that the legs are separated with the greatest difficulty. In cases of this extreme rigidity the patient usually loses the power of walking. The nutrition is well maintained, the muscles may be hypertrophied. The reflexes are greatly