ceived for a second or two or may be delayed for as much as ten seconds. The pain felt may persist. A curious phenomenon is the loss of the power of localizing the pain. For instance, if the patient is pricked on one limb he may say that he feels it on the other (allocheiria), or a pin-prick on the foot may be felt in both feet. The muscular sense becomes much impaired and the patient no longer recognizes the position in which his limbs are placed. This may be present in the pre-ataxic stage.

Reflexes.—As mentioned, the loss of the knee-jerk is one of the earliest symptoms of the disease. Occasionally a case is found in which it is retained. The skin reflexes may at first be increased, but later are usually involved with the deep reflexes.

Special Senses.—The eye symptoms noted above may be present, but, as mentioned, ataxia is rare with atrophy of the optic nerve.

Deafness may develop, due to lesion of the auditory nerve. There may also be attacks of vertigo. Olfactory symptoms are rare.

Visceral Symptoms.—Among the most remarkable sensory disturbances are the tabetic crises, severe paroxysms of pain referred to various viscera; thus laryngeal, gastric, nephralgic, rectal, urethral, and clitoral crises have been described. The most common are the gastric and laryngeal. In the former there are intense pains in the stomach, vomiting, and a secretion of hyperacid gastric juice. The attack may last for several days or even longer. There may be severe pain without any vomiting. The attacks are of variable intensity and usually require morphia. Paroxysms of rectal pain and tenesmus are described. They have not been common in my experience. Laryngeal crises also are rare. There may be true spasm with dyspnœa and noisy inspiration. In one instance at least the patient has died in the attack.

The sphincters are frequently involved. Early in the disease there may be a retardation or hesitancy in making water. Later there is retention, and cystitis may occur. Unless great care is taken the inflammation may extend to the kidneys. Constipation is extremely common. Late in the disease the sphincter ani is weakened. The sexual power is usually lost in the ataxic stage.

Trophic Changes.—Skin rashes may develop in the course of the lightning pains, such as herpes, œdema, or local sweating. Alteration in the nails may occur. A perforating ulcer may develop on the foot, usually beneath the great toe. Onychia may prove very troublesome.

The arthropathies or joint lesions affect chiefly the knees. They are unquestionably associated with the disease itself, and not necessarily a result of trauma. The condition, known as Charcot's joint, is anatomically similar to that of chronic arthritis deformans. The effusion may be rapid and there may be great disintegration and destruction of the cartilages and bones, leading to dislocation and deformity. Pus was present in a well-marked Charcot's joint in a patient of C. K. Mills at the Philadelphia Hospital. Spontaneous fractures may occur. Among other trophic

disturbances may be mentioned atrophy of the muscles, which is usually a late manifestation, but may be localized and associated with neuritis. In any very large collection of cases many instances of atrophy are found, due either to involvement of the anterior horns or to peripheral neuritis.

Cerebral Symptoms.—Hemiplegia may develop at any stage of the disease, more commonly when it is well advanced. It may be due to hæmorrhagic softening in consequence of disease of the vessels or to progressive cortical changes. Hemianæsthesia is sometimes present. Very rarely the hemiplegia is due to coarse syphilitic disease.

Dementia paralytica frequently exists with tabes, and it may be extremely difficult to determine which has been the primary affection. In a majority of the cases the locomotor ataxia has preceded the symptoms of general paresis. In other instances melancholia, dementia, or paranoia develop.

(*c*) **Paralytic Stage.**—After persisting for an indefinite number of years the patient gradually loses the power of walking and becomes bedridden or paralyzed. In this condition he is very likely to be carried off by some intercurrent affection, such as pyelo-nephritis, pneumonia, or tuberculosis.

The Course of the Disease.—A patient may remain in the pre-ataxic stage for an indefinite period, and the loss of knee-jerk and the gray atrophy of the optic nerves may be the sole indications of the true nature of the disease. In such cases incoördination rarely develops. In a majority of cases the progress is slow, and after six or eight years, sometimes less, the ataxia is well developed. The symptoms may vary a good deal; thus the pains, which may have been excessive at first, often lessen. The disease may remain stationary for years; then exacerbations occur and it makes rapid progress. Occasionally the disease seems to be arrested. There are instances of what may be called acute ataxia, in which, within a year or even less, the incoördination is marked, and the paralytic stage may develop within a few months. The disease itself rarely causes death, and after becoming bedridden the patient may live for fifteen or twenty years.

Diagnosis.—In the pre-ataxic stage the combination of lightning pains and the absence of knee-jerk is distinctive. The association of progressive atrophy of the optic nerves with loss of knee-jerk is also characteristic. The early ocular palsies are of the greatest importance. A squint, ptosis, or the Argyll-Robertson pupil may be the first symptom, and may exist with the loss only of the knee-jerk. Loss of the knee-jerk alone, however, does occasionally occur in healthy individuals.

The diseases most likely to be confounded with locomotor ataxia are: (1) *Peripheral Neuritis.*—The pseudo-tabetic gait of arsenical, alcoholic, or diabetic paralysis is quite unlike that of locomotor ataxia. In these forms there is a paralysis of the feet and the leg is lifted high in order that the toes may clear the floor. The use of the word tabes in this con-

nection should no longer be continued. If in any doubt, the absence of the lightning pains and eye symptoms and the history will suffice in the majority of cases to make the diagnosis clear. In diphtheritic paralysis the early loss of knee-jerk and the associated eye symptoms may suggest tabes, but the history, the existence of paralysis of the throat, and the absence of pains render a diagnosis easy.

(2) *Ataxic Paraplegia.*—Marked incoördination with spastic paralysis is characteristic of the condition which Gowers has termed ataxic paraplegia. In a majority of the cases this affection is distinguished also by the absence of pains and of eye symptoms.

(3) *Cerebellar Disease.*—The cerebellar incoördination has only a superficial resemblance to that of locomotor ataxia; the knee-jerk is present, there are no lightning pains, no sensory disturbances; while, on the other hand, there are headache, optic neuritis, and vomiting.

(4) Some *acute affections* involving the posterior columns of the cord may be followed by incoördination and resemble tabes very closely. In a case recently under my care, the gait was characteristic and Romberg's symptom was present. The knee-jerk, however, was retained and there were no ocular symptoms. The condition had developed within three or four months, and there was a well-marked history of syphilis. Under large doses of iodide of potassium the ataxia and other symptoms completely disappeared.

(5) *General Paresis.*—In some cases this offers a serious difficulty. In the first place, in general paresis, tabetic symptoms often develop; on the other hand, there are cases of locomotor ataxia in which, toward the end, there are symptoms of general paresis. Cases of unusually acute ataxia with mental symptoms belong, as a rule, to the former disease. The question will be considered under general paresis.

(6) Visceral crises and neuralgic symptoms may lead to error, and in middle-aged men with severe, recurring attacks of gastralgia it is always well to bear in mind the possibility of tabes, and to make a careful examination of the eyes and of the knee-jerk.

Prognosis.—Complete recovery cannot be expected, but arrest of the progress is not uncommon and a marked amelioration of the symptoms is frequent. Optic-nerve atrophy, one of the most serious events in the disease, has this hopeful aspect—that incoördination rarely follows and the progress may be arrested. The optic atrophy itself is occasionally checked. On the whole, the prognosis in tabes is bad. The experience of such men as Weir Mitchell, Charcot, and Gowers is distinctly opposed to the belief that locomotor ataxia is ever completely cured.* No such instance has come under my personal observation.

Treatment.—To arrest the progress and to relieve, if possible, the symptoms are the objects which the practitioner should have in view. A

* For a study of the reputed cures, see L. C. Gray, N. Y. Medical Journal, Nov., 1880.

quiet, well-regulated method of life is essential. It is not well, as a rule, for a patient to give up his occupation so long as he is able to keep about and perform ordinary work. I know tabetics who have for years conducted large businesses, and there have been several notable instances in our profession of men who have risen to distinction in spite of the existence of this disease. Excesses of all sorts, more particularly *in baccho et venere*, should be carefully avoided. A man in the pre-ataxic stage should not marry.

Care should be taken in the diet, particularly if gastric crises have occurred. To secure arrest of the disease many remedies have been employed. Although syphilis plays such an important *rôle* in the etiology, it is universally acknowledged that neither mercury nor the iodide of potassium have as a rule the slightest influence over the tabetic lesions. To this there is but one exception—when the syphilis is comparatively recent; when the symptoms develop within two years of the primary infection, there is then a possibility of arrest by mercury and iodide of potassium. However, they do not always relieve. In two cases of very rapidly progressing tabes following syphilis this medication was of no avail. Not only is an anti-mercurial treatment of no benefit in the majority of cases of locomotor ataxia, but my experience tallies with that of Gowers in that it may even hasten the progress of the disease. Of remedies which may be tried and are believed by some writers to retard the progress, the following are recommended: Arsenic in full doses, nitrate of silver in quarter-grain doses, Calabar bean, ergot, and the preparations of gold.

The treatment by suspension introduced a few years ago has already been practically abandoned. Good effects certainly have followed in a few cases, but it was unreasonable from the outset, either on therapeutic or scientific grounds, to hope that by such a measure permanent changes could be induced in the pathological condition. The benefits were due in great part to suggestion and to psychical effects. In any case it must be used with caution.

For the pains, complete rest in bed, as advised by Weir Mitchell, and counter-irritation to the spine (either blisters or the thermo-cautery) may be employed. The severe spells which come on particularly after excesses of any kind are often promptly relieved by a hot bath or by a Turkish bath. A prolonged course of nitrate of silver seems in some cases to allay the pains and lessen the liability to the attacks. I have never seen ill effects from its use in the spinal scleroses. Antipyrin and antifebrin may be employed, and occasionally do good, but their analgesic powers in this disease have been greatly overrated. Cannabis indica is sometimes useful. In the severe paroxysms of pain hypodermics of morphia or of cocaine must be used. The use of morphia should be postponed as long as possible. Electricity is of very little benefit. For the severe attacks of gastralgia, morphia is also required. The laryngeal crises are rarely dangerous. An application of cocaine may be made during the spasm, or a few whiffs of chloroform may be given, or nitrite of amyl. In all cases of tabes

with increased arterial tension the prolonged use of nitroglycerin, given in increasing doses until the physiological effect is produced, is of great service in allaying the neuralgic pains and diminishing the frequency of the crises. Its use must be guarded when there is aortic insufficiency. The special indication is increased tension. The bladder symptoms demand constant care. When the organ cannot be perfectly emptied the catheter should be used, and the patient may be taught its use and how to keep it thoroughly sterilized.

III. Hereditary Ataxia (*Friedreich's Ataxia*).

In 1861 Friedreich reported six cases of a form of hereditary ataxia, and the affection has usually gone by his name. Unfortunately, *paramyoclonus multiplex* is also called Friedreich's disease; so it is best, if his name is used in connection with this affection, to term it Friedreich's ataxia. It is a very different disease in many respects from ordinary tabes. It may or may not be hereditary. It is really a family disease, several brothers and sisters being, as a rule, affected. The 143 cases analyzed by Griffith occurred in 71 unrelated families. In his series inheritance of the disease itself occurred in only 33 cases. Various influences in the parents have been noted; alcoholism in only 7 cases. Syphilis has rarely been present. Of the 143 cases, 86 were males and 57 females. The disease sets in early in life, and in Griffith's series 15 occurred before the age of two years, 39 before the sixth year, 45 between the sixth and tenth years, 20 between the eleventh and fifteenth years, 18 between the sixteenth and twentieth years, and 5 between the twentieth and twenty-fifth years.

The *morbid anatomy* shows an extensive sclerosis of the posterior and lateral columns of the spinal cord. The periphery, and the cerebellar tracts are usually involved. The recent observations of Déjérine and Letulle are of special interest, since they seem to indicate that the change in this disease is a neurogliar (ectodermal) sclerosis, differing entirely from the ordinary spinal sclerosis. According to this view, Friedreich's disease is a gliosis of the posterior columns due to developmental errors.

Symptoms.—The ataxia is unlike the ordinary form. The incoördination begins in the legs, but the gait is peculiar. It is swaying, irregular, and more like that of a drunken man. There is not the characteristic stamping gait of the true tabes. Romberg's symptom may or may not be present. The ataxia of the arms occurs early and is very marked; the movements are almost choreiform, irregular, and somewhat swaying. In making any voluntary movement the action is overdone, the prehension is claw-like, and the fingers may be spread or overextended just before grasping an object. The hand frequently moves about an object for a moment and then suddenly pounces upon it. There are irregular, swaying movements, some of which are choreiform, of the head and shoulders. There is present in many cases what is known as static ataxia, that is to say, ataxia of quiet action—irregular, slow movements of the fingers or the hands while at rest.

Sensory symptoms are not usually present. The reflexes may be lost. In Griffith's table they were abolished in 91 cases.

Nystagmus is a characteristic symptom. Atrophy of the optic nerve rarely occurs. A striking feature is early deformity of the feet. There is talipes equinus, and the patient walks on the outer edge of the feet. The big toe is flexed dorsally on the first phalanx. Lateral curvature of the spine is very common.

Trophic lesions are rare. As the disease advances paralysis comes on and may ultimately be complete. Some of the patients never walk.

Disturbance of speech is common. It is usually slow and scanning; the expression is often dull; the mental power is, as a rule, maintained, but late in the disease becomes impaired.

The *diagnosis* of the disease is not difficult when several members of a family are affected. The onset in childhood, the curious form of incoördination, the early talipes equinus, the position of the great toe, the scoliosis, the nystagmus, and scanning speech make up an unmistakable picture. The disease is often confounded with chorea, with the ordinary form of which it has nothing in common. With hereditary chorea it has certain similarities, but usually this disease does not set in until after the thirtieth year.

The disease lasts for many years and is incurable. Care should be taken to prevent contractures.

IV. Syringo-myelia.

Definition.—A gliomatous new formation about the central canal of the spinal cord, with cavity formation.

The disease has attracted a good deal of attention within the past few years, and has a definite clinical interest since cases can now be diagnosed.

Etiology and Morbid Anatomy.—Syringo-myelia must be distinguished from dilatation of the central canal—hydromyelus—slight grades of which are not very uncommon either as a congenital condition or as a result of the pressure of tumors. The cavity of syringo-myelia has a variable extent in the cord, sometimes existing in the entire length, but in many cases involving only the cervical and dorsal regions or a more limited area. It is usually in the posterior portion of the cord and extends into one posterior cornu. The transverse section may be oval or circular or narrow and fissure-like. It varies at different levels. The condition is now regarded as a *gliosis*, a development of embryonal neurogliar tissue in which hæmorrhage or degeneration takes place with the formation of cavities.