Constipation is common, but incontinence of the bowels is rare. Sexual and menstrual troubles are very rare.

VI. *Bulbar and Cranial Symptoms.*—As was stated in the previous description, the anatomical process may involve nerves from the fifth to the twelfth; other cranial nerves may be influenced indirectly by hydrocephalus, tabes, etc. Symptoms on the part of the olfactorius and auditory nerves are very rare.

Taste may be affected, but complicating hysteria may be a cause; the affection is usually unilateral.

Eye symptoms are important. Neuritis and atrophy of the nerve are rare occurrences and probably depend on hydrocephalus or actual gliomata. The pupils are often different on account of lesions of the sympathetic. The narrow pupil and narrowed opening between the eyelids, as well as the retrocession of the bulb, are found on the side chiefly affected. Kocher has shown that lesions of the medulla and upper cervical segments are as competent to produce sympathetic disturbances as are lesions of the lower cervical and first dorsal segments.

Argyll-Robertson pupils do not belong to syringomyelia; if present, they suggest complicating tabes.

Nystagmus occurs more frequently than in any other nerve lesion except multiple sclerosis.

Eye-muscle palsies may occur, but, on the whole, they are rare. They may be recurrent; the abducens is most often affected.

Trigeminus lesions are important. Violent neuralgias or paræsthesiæ may be the first symptom. Dissociation of sensation is usual, and the distribution follows segmental types. The "parietal-ear-chin" line of Kocher marks the upper level of the second cervical segment supply. As the proximal trigeminus becomes involved the first sensory loss is in the scalp and then in areas narrowing concentrically toward the nose (von Soelder, Schlesinger).

The motor fifth is hardly ever affected. The corneal reflex is very rarely absent. Occasional trophic disturbances occur, such as loss of hair or teeth; or the tears may be more abundant on the affected side. Implication of the facial is uncommon; most often the lower portion is alone affected. Often there is no reaction of degeneration.

Hemiatrophy of the tongue is a frequent and may be an initial symptom. Unilateral affections of the soft palate and larnyx are extremely important in diagnosis. In the larynx the usual lesion is paralysis of muscles supplied by one recurrent. Sensory and motor laryngeal changes may be independent of one another. It is important to remember that the laryngeal palsies may develop in an apoplectiform manner with marked vertigo and even loss of consciousness. Vertigo, however, may occur independently of bulbar lesions.

Vomiting may be periodic, occurring in attacks like regular tabetic crises. Heart symptoms are rare.

Headache is not a feature of the disease. Convulsions may rarely occur. Scanning speech has been reported in several cases.

Atypical Forms.—There may be, for long periods, the picture of a spastic paraplegia or an amyotrophic lateral sclerosis. Sensory changes may be absent for years (Bouchard). Bulbar symptoms may come on in an apoplectiform manner and for a long time they may be the only manifestations of the disease. In very rare cases there may be widespread sensory changes—anæsthesia as well as analgesia and thermanæsthesia. In the celebrated case of Spaeth and Schneppel there was practically total loss of all forms of sensation.

In 1883 Morvan published descriptions of a symptom-complex that has since been known by his name. He described the combination of painless panaritium of the fingers with great deformity and accompanied by marked loss of pain- and temperature-perception in the hands and arms. Autopsies of Joffroy-Achard, Prouff, Marinesco, Hoffman, Redlich, Schlesinger, and others have demonstrated the direct association of Morvan's symptom-complex with syringomyelia. On the other hand, the same picture may present itself in leprosy, and only a careful examination will enable one to make a differential diagnosis.

The early appearance of changes in the joints, spontaneous fractures, etc., can lead to confusion only on superficial examination.

Differential Diagnosis.—There is no doubt that syringomyelia represents a clinical if not an anatomical entity. Due attention to the combination of sensory, motor, and trophic changes will usually enable one to make a correct diagnosis. The following represent the chief conditions that may lead to confusion:

1. *Progressive Muscular Atrophy.*—Kahler and Schultze pointed out the importance of sensory changes in distinguishing syringomyelia from cases of muscle atrophy of the Aran-Duchenne type. The doubtful cases are those in which, for years, there are no sensory changes (Croeg, Dejerine). It is necessary to seek for isolated loss or diminution of temperature-perception. Increased knee-jerks, scoliosis, lesions of the sympathetic, trophic changes, paræsthesiæ, ataxia, sphincter disturbances, speak for syringomyelia.

2. *Amyotrophic Lateral Sclerosis.*—Sensory changes are rare, but they may occur (Oppenheim). Bladder symptoms, scoliosis, paræsthesiæ, unilateral bulbar symptoms, trigeminus involvement, nystagmus, are decisive for syringomyelia.

3. *Multiple Sclerosis.*—Tremor, nystagmus, scanning speech, increased knee-jerks, spastic paraplegia, ataxia, sphincter disturbances—these are symptoms that belong to both diseases. Some sensory change is not uncommon in multiple sclerosis, and even dissociation may occur (Freund, Reichel). Optic atrophy, especially atrophy of the temporal half, decides for multiple sclerosis. Marked sensory changes, muscle atrophy, trophic disturbances, scoliosis, speak for syringomyelia. Remissions may occur in either affection, but are more usual in multiple sclerosis.

4. *Progressive Muscular Dystrophy.*—If syringomyelia begins in the shoulder group of muscles atrophy is not rarely associated with partial hypertrophy, and, if cases of dystrophy are complicated with hysterical sensory changes, the two conditions will appear very much alike. Trophic disturbances, increased knee-jerks, segmental sensory distribution, bulbar symptoms, decide for syringomyelia.

5. *Meningomyelitis Luetica.*—Spastic paraplegia and dissociation of sensation may occur in both diseases. Brain symptoms and a Brown-Séquard type of motor and sensory lesions favor lues. Scoliosis and trophic changes are absent. Nystagmus is very rare.

6. *Tabes.*—There may be many symptoms in common. Strongly characteristic symptoms in either direction decide. Thus, for example, the Argyll-Robertson pupil, loss of knee-jerk, ataxia, sphincter disturbances, character of sensory loss, trophic changes in lower extremities, crises, belong to tabes; on the other hand, muscle atrophy, marked dissociation of sensation over large areas, increased knee-jerks, scoliosis, trophic changes in the arms, speak for syringomyelia.

7. *Hæmatomyelia.*—In this disease the symptoms are marked at first, but later some improvement takes place. Syringomyelia is usually steadily progressive.

8. *Plexus Affections.*—Cases have been reported of bilateral plexus palsies, of both the Erb and the Klumpke types. There may be dissociation of sensation as in syringomyelia, and the distribution may be of the segmental type. In some cases only continued observation will decide; as a rule, tenderness of the nerve trunks and of the spine is more marked in the plexus affections, there is no increase of knee-jerks, bulbar symptoms do not occur, and the losses of sensation are limited to the arms.

9. *Hysteria.*—Complications with hysteria may add to the difficulties of differential diagnosis in muscular dystrophies, atrophies, etc., as mentioned above; or hysteria may be added to or may simulate some of the symptoms of syringomyelia. In the sensory disturbances of hysteria dissociation is rarely segmental. Suggestion may lead to disappearance or transference of the sensory losses. Deep or superficial reflexes are little modified, trophic changes and atrophies are extremely rare. Bulbar changes do not occur.

10. *Central Tumors.*—(Tubercle gumma, glioma, etc.) Here symptoms are severe and advance quickly. Pain is severe; marked paræsthesiæ, especially of heat and cold, are usual; atrophies and paralyses, particularly paraplegia, come on rapidly. Ataxia and sphincter disturbances are frequent; there is often complete or partial development of the Brown-Séquard complex; if bulbar symptoms occur they are severe and run their course quickly.

11. *Pemphigus, scleroderma, Raynaud's disease, deformities due to arteriosclerosis, pellagra, and lathyrismus,* can rarely give rise to a confusion.

12. *Leprosy.*—The similarity between some forms of leprosy and syringomyelia was pointed out by Steudener in 1867 and by Langhans in 1875, but the publication of Morvan's work in 1883 first awakened general interest in the question. As described above, the Morvan symptom-complex was shown, by carefully made autopsies, to be associated with syringomyelia (Joffroy and Achard, Prouff, Redlich, Schlesinger, and others). But, subsequently, Zambaco published reports which showed that in Brittany, where Morvan had carried on his work and where leprosy was endemic, there occurred cases which merited the name of Morvan's disease, but in which the existence of leprous neuritis was absolutely proved. It is particularly these trophic forms of syringomyelia that are liable to be taken for leprosy, and it is equally true that the anæsthetic and not the tuberculous types of leprosy are confused with syringomyelia. Despite the views of some extremists, the two diseases are undoubtedly distinct. Leprosy has no influence in the production of syringomyelia, and unequivocal lesions of leprosy have never been found associated with syringomyelia; accidental combination may of course be possible. The leprous analgesia or thermo-anæsthesia depends on involvement of peripheral nerves and is usually not so widespread nor so segmentally distributed as in syringomyelia. Evidence of peripheral nerve disease is found in thickening of nerve trunks, particularly the ulna and great auricular. Bulbar lesions, increased knee-jerks, girdle sensation, sphincter disturbance, and scoliosis do not belong to the symptomatology of leprosy. On the other hand, the cutaneous nodes, widespread pemphigus, vitiligo, white scars, and, above all, the demonstration of bacilli in the blood, nasal secretion, etc., decide for leprosy. There are some cases, especially in districts where leprosy is endemic, in which a diagnosis is difficult; as autopsies prove, however, confounding of the two diseases can even then be avoided if a careful examination be made.

Course and Prognosis.—The disease is eminently chronic; cases have been known of thirty, forty, and fifty years' duration. The type of disease dependent on quick glia-proliferation (gliosis without cavity formation) runs a relatively rapid course (from three to six years), but the cases can hardly be separated clinically.

Intercurrent infections and trauma often influence the process unfavorably. Apoplectiform attacks may occur with accentuation of bulbar or spinal symptoms; marked remissions also occasionally occur. The bulbar lesions are on the whole benignant and may last for years unchanged.

Death may occur from bladder and kidney complications, from sepsis following the various trophic disturbances, and sometimes from bulbar lesions, but most often from intercurrent disease.

Treatment.—From the nature of the lesions it is impossible to expect direct results from treatment. In a few cases, due seemingly to lues, specific treatment has proved advantageous. Prophylactic measures play an important part. Such are: avoidance of trauma, of overuse of atrophic muscles; prevention of bedsores and, to a certain extent, of atrophic joints; avoidance of injury to analgesic extremities, and of exposure to injurious temperature changes; above all, great care in the avoidance of bladder complications.

Mud baths and gentle massage may help contractures.

Electricity, galvanic and static, may relieve pain or paræsthesiæ.

Panaritium and suppurating joints must be treated surgically; as a rule, wounds from surgical operations heal fairly. Pain may require phenacetin, antipyrin, pyramidon, salicylates, or morphine. Potassium iodide and mercury should be given in cases of questionable luetic origin, or iodide may be tried for a time in all cases. General tonics can only be of indirect service.

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Some of the More Important Recent Publications.

Albarran and Guillain: Ueber Blasenstörungen bei Syringomyelie. Wien. med. Woch., No. 51, 1901.

Audrey, Ch., et Dalous: Hyperkeratose circonscrite des doigts chez un syringomyelique. Journ. de mal. cutan. et syph., Paris, 1902.

Azema: Un cas de syringomyélie. Écho méd., Toulouse, 1902, xvi.

Bischofswerder: Les Nevromes intramedullaires dans deux cas de syringomyélie avec main succulente. Journ. de Neurol., Paris-Brux., 1901.—Lésions des cordons postérieurs et troubles de la sensibilité dans un cas de syringomyélie. Rev. Neurol., Paris, 1902.—Contribution à l'étude de l'anatomie pathologique de la syringomyélie. Nevromes intramedullaires. Main succulente, Paris, 1902.

Bonhoff, Hermann F.: Trauma in Beziehung zur Syringomyelitis. Inaug.-Dissert., Bonn, Lille, Juli, 1901.

de Buck et de Stella: Un cas de syringobulbie et de syringomyélie. Belgique med. Grand-Haarlene, 1902, ix.

Collier, James: A Case of Deformity of the Skull, Simulating Leontiasis Ossea, Associated with a Condition of Syringomyelia; no physical signs of syringomyelia present. (Case with autopsy.)

Cuchini: Contributo allo studio della siringomielia de origine traumatica. Gass. med. di Roma, 1902.

Fargues: Contribution à l'étude de la topographie des troubles sensitifs dans la syringomyélie, Paris, Nantes, Derouai, 1902.

Flegler, P.: Beitrag zur Casuistik der Syringomyelie und ueber die bei dieser Krankheit vorkommenden Hautstorungen.

Gangolphe: Osteoarthropathie d'origine medullaire et tuberculose. Notes sur les ostéoarthropathies syringomyeliques. Bull. Soc. de Chir. de Lyon, 1901.

Guillain, G.: La forme spasmodique de la syringomyélie. La nevrite ascendante et la traumatisme dans l'étiologie de la syringomyélie, Paris, 1902.—La nevrite ascendante dans l'étiologie de la syringomyélie. Bull. et Mem. Soc. med. de Hôp. de Paris, 1902.

Goldbaum: A New Symptom in Syringomyelia. Neurologisches Centralblatt, 1901.

Huet, E., et Cestan, R.: Sur deux cas de syringomyélie à topographie radiculaire des troubles sensitifs et des troubles moteurs. Rev. Neurol., Paris, 1902.

Hudervernig, C.: Zur Pathogenese der Arthropathien bei Syringomyelie. Neurol. Centralbl., Leipzig, 1901.

Hoedlmoser, G.: A Peculiarly Localized Arthropathy in a Case of Syringomyelia with Genital Hypoplasia. Wiener klinische Wochenschrift, 1901.

Ingebrans et Brassart, H.: Syringomyélie posttraumatique. Écho méd. du Nord., Lille, vi., 1902.

Koelichen, J.: Contribution à l'étude de la syringomyélie et de l'hydromyélie. Gaz. lek. Warszawa, 1902.

Kienboch, R.: Critik der sogennanten traumatischen Syringomyélie. Jahrbücher f. Psychiatrie, Vienna, 1892.

Launois, P., et Roy: Exostoses multiples, Syndrome syringomyélique (Amputations spontanées des doigts, thermanesthésie) chez un homme de trente et un ans hystérique et tuberculeux. Bull. et Mem. Soc. méd. de Hôp. de Paris, 1902.

Libotte: Un cas de syringomyélie au début. Journ. de Neurol., Paris-Brux., 1902, viii.

Losen, Adolf: Ein Beitrag zur Morvan'schen Krankheit (Syringomyelie). Inaug.-Dissert. Würzburg, April to September, 1901.

Meyer, Adolf: Report of a Transverse Lesion of the Median Thoracic Segments, Leaving Intact the Posterior Columns, and Causing Syringomyelic Dissociation. Journ. of Nerv. and Ment. Dis., December, 1902.

Pearce, F. S.: Syringomyelia. Medical Fortnightly, St. Louis, December, 1902.

Preobrachensky, P. A.: The Pathological Anatomy and Pathogenesis of Syringomyelia. Roussky Archiev Pathologyi, June, 1902.

Renon, L.: Des Fractures spontanées chez les syringomyéliques. Presse médicale, Paris, July, 1902.

Rosenfeld: The Distribution of the Temperature Sense in Syringomyelia. Deutsche Zeitschrift für Nervenheilkunde, 1901.

Raymond: Syringomyelia of the Medulla and Pons. Semaine médicale, September, 1901.

Stolper, P.: Syringomyelie, Gelenkerkrankung, Trauma. Curztl. Sachverstand Ztg., Berlin, 1902.

Schroefl, August: Zur Kasuistik der Syringomyelia. Inaug.-Dissert. München, October, 1901.

Sachs, Paul: Die differentialdiagnostischen Beziehungen des Morvan'schen Symptomenkomplexes zur Lepra und zur Syringomyelie. Inaug.-Dissert., Berlin, Juli, August, 1901.

Sainton and Ferrand: Syringomyelia with "Thorax en Bateau" and Trophic Disturbances Resembling Myxœdema.

Schlesinger: Die Syringomyelie, Leipzig, 1895; also 1901.

Taubert: Ueber Erythromelalgie bei Syringomyelia des Cervikal-Marks. Deutsche med. Wochenschrift, 1903.

Westphal, A.: Ueber die Bedeutung von Traumen und Blutungen in der Pathogenese der Syringomyelie. Archiv für Psychiatrie, Bd. 36.
Westrum, W.: Klinische Beiträge zur Kenntniss der Syringomyelie. Inaug.-Dissert., Erlangen, December, 1901.

SPINAL-CORD DISEASES: TABES.—Tabes is the term which is gradually being universally adopted to describe the disease popularly known as "locomotor ataxia." The name "ataxie locomotrice" was given to this affection by Duchenne in 1858 and is still quite popular. It is objectionable, however, because it is merely descriptive of a symptom, which at times may not appear until late in the course of the disease, or which may be entirely absent. "Posterior spinal sclerosis" is also unsatisfactory, as it refers only to the most easily discoverable lesion. "Tabes dorsalis," or "wasting of the back," was applied by Hippocrates to certain symptoms supposed to be due to venereal excesses, but was restricted to this disease by Romberg.

Tabes is the most common form of chronic organic disease of the nervous system. The pathological process underlying the affection is a parenchymatous degeneration terminating in sclerosis, which principally involves the sensory neurones. The peripheral motor neurones are also frequently implicated. In other words, it may be looked upon as a degenerative disease affecting various parts of the entire nervous system, while the most pronounced and extensive lesion is found in the posterior columns of the spinal cord. The morbid process may attack the cranial nerves and their nuclei as well as the peripheral nerves in the extremities. It may thus produce blindness from gray degeneration of the optic nerves, or paralysis of ocular muscles from degeneration of the nerve nuclei. The brain cortex does not always escape. The parts first affected in tabes are usually the fibres which originate in the spinal ganglia, *i.e.*, the fibres of the posterior roots which traverse the posterior columns.

Etiology.—In the vast majority of cases there is an antecedent history of syphilitic infection, antedating the first signs of the disease by from one to thirty-five years. In most cases the period ranges from five to fifteen years, the previous existence of syphilis being either demonstrable or admitted by the patient.

Statistics collected from various reliable sources show that from seventy to ninety-five per cent. of all tabetics give a history of previous syphilis. Hence it has been assumed by nearly all clinical investigators that tabes is one of the results of syphilis. Strümpell has expressed the view, which has been favorably received, that tabes does not arise from the syphilitic bacteria themselves, but from a toxin derived from them. It has therefore been almost universally considered as a sequel of syphilis, and has been variously characterized as a "post-," "para-," or "metasyphilitic" process. On the other hand, it is well known that only a small percentage of those who have contracted syphilis develop tabes. It cannot be consistently denied, however, that tabes also occurs without any discoverable evidence of previous syphilis. With the previous knowledge of an antecedent syphilis in a given case, we are by no means certain that the tabetic degeneration is the result of such early infection. It would seem that in most cases additional exciting or accessory causes are operative, such as exposure to cold, over-fatigue, alcoholic, sexual or other excesses, or a congenital or acquired neuropathic constitution—causes which predispose to the development of tabes, as well as to other degenerative processes, in the nervous system, in those patients who have contracted syphilis at some remote period.

Tabes has at times been considered as resulting from trauma, but in all such cases that have been investigated it has been shown that tabetic symptoms existed before the injury. It is a well-established fact, however, that the tabetic process is accelerated under such circumstances.

Symptoms.—Among the early symptoms of the disease first noticed by the patient are pains in the trunk and extremities; disturbance of the bladder; impotence; rectal pain; diplopia; ocular paralysis; failing vision; cutaneous hyperæsthesia or anæsthesia; incoördination in walking; visceral symptoms known as "crises" affecting the gastro-intestinal tract, bladder, or rectum, and occasionally laryngeal "crises."

Pain is often one of the earliest symptoms of tabes, and may antedate all other symptoms for many years. The character of the tabetic pains is as a rule pathognomonic. They are irregular in their distribution and are usually described by the patient as "shock-like," "sharp," "piercing," "cutting," or "stabbing," being rapidly repeated at the same spot, which often becomes extremely sensitive to the slightest touch. They may also resemble the painful sensation as if the muscles and bones were being crushed, or as if a piece of nerve was pulled. They may be so sudden in their onset that a strong man is surprised into a loud exclamation of pain while feeling otherwise well. They may occur every few moments for minutes, hours, or days. Although they differ essentially from true neuralgic, muscular, or periosteal pains, they are often erroneously considered as being of such origin. Hence it is a common experience for the neurologist to receive such patients with their announcement that they had previously been under treatment for rheumatism or neuralgia. In order fully to appreciate the significance of these pains, it is necessary to study this symptom carefully and minutely, for it may exist for several years before other symptoms are at all prominent. In the majority of instances, the pains most frequently affect the lower extremities. They may, however, be limited to the inframammary, intercostal, dorsal, or lumbar regions. Indeed, they are known to attack almost any part of the body. They may thus appear in the form of trigeminal facial pain or be located in the scalp. On the other hand, in some patients, pain is either entirely absent or only a slight, dull, circumscribed, and aching pain is occasionally complained of. If such pains do not appear in the early period of the disease, they are not likely to appear later.

Crises.—At almost any stage of the disease suddenly recurring paroxysmal attacks of severe gastralgia may take place, accompanied by exhaustive vomiting, which resists all ordinary methods of treatment, but seems to subside spontaneously. These attacks are known as "gastric crises." Sometimes persistent gastralgia may be the only symptom complained of. In such a case careful examination will often reveal an area of cutaneous anæsthesia in the epigastric region, thus leading to further investigation which discloses the underlying cause of the pain. Instead of these "gastric crises," there may be severe and unaccountable diarrhœa associated with violent colicky pain that may sometimes simulate an attack of renal colic. In some instances the rectum may be the seat of severe pain that is remittent in character. This pain not being relieved by ordinary means, its true nature being overlooked, surgeons have been led to the fruitless operation of forcibly stretching and paralyzing the anal sphincter and removing any hemorrhoids that perchance are present.

"Laryngeal crises" occur only in a very small percentage of cases. The most common form is true laryngeal spasm, with noisy inspiration and expiration, cough, and often considerable dyspnœa. The paroxysms may resemble those of whooping-cough or of laryngismus stridulus. Death during these attacks is extremely rare.

Incontinence or retention of urine is frequently an early symptom. Considerable effort is required in micturition, the urine flowing slowly. Quite often the bladder is not completely emptied and the residual urine may undergo decomposition and thus set up a cystitis.

Impotence may also occur as an early manifestation, but it usually develops as the disease advances.

Diplopia, ocular paralysis, or *failing vision* may be the first symptom that leads the patient to consult a physician. For several years before other symptoms are clearly manifested, or at any time in the early period of the disease, there may be frequent diplopia, or transient attacks of paralysis affecting the ocular muscles; these

attacks are of nuclear or peripheral origin. The paralyses of the external ocular muscles are often bilateral, but not symmetrical, often unilateral, and frequently affect only single muscles. Ptosis and paralysis of one or more muscles supplied by the oculomotor nerve, and abducens paralysis, are the most frequent. As a rule they develop suddenly and usually disappear after a longer or shorter period, with or without treatment. Relapses are frequent. Attention may first be drawn to the disease through a sudden third- or sixth-nerve paralysis, either partial or complete. The duration varies from a few hours to a year or more, or the paralysis may become permanent. Sometimes both eyes are affected, and a progressive and complete ophthalmoplegia may ultimately develop.

Atrophy of the optic nerve is the most serious ocular complication of tabes. It occurs in about ten or fifteen per cent. of the cases. For a long time it may be the only symptom of the disease, thus appearing several years before other characteristic phenomena are manifested. It rarely develops in the later period of the disease. The atrophic process is the result of gray degeneration of the optic nerves similar to that which attacks the posterior column of the spinal cord. The failure of sight usually commences with a peripheral limitation of the field and loss of color vision (the visual field for green contracting early); but sometimes central vision is defective from the very beginning. The atrophy is almost always bilateral, being more advanced in one eye than in the other, and ultimately progresses to complete blindness. It is rare for tabetic patients who become blind at an early stage of the disease to become ataxic later; but if the ataxia has become well pronounced, it does not always improve with the subsequent development of optic atrophy. In the majority of instances, the occurrence of optic atrophy seems to inhibit the further progress of the disease. This is a peculiar clinical fact which has never been satisfactorily explained.

Incoördination, or ataxia as it is familiarly termed, is a common symptom in many cases of tabes and most frequently affects the lower extremities. It has occasionally been noted by the patient himself, who first discovers difficulty in standing or walking with closed eyes.

As a rule, the lower extremities are affected first. When the sclerosis begins in the cervical portion of the cord, the ataxia, as well as other symptoms, may, for a longer or shorter period, be confined to the upper extremities. It is a curious clinical fact, previously mentioned, that, in cases in which optic atrophy appears, the ataxia often ceases its further progress.

Incoördination is a disturbance of the associated muscular action which is essential in the maintenance of equilibrium. Coördinate muscular action is kept under the patient's control to a certain extent by the attention and vision. As soon as the patient fails to watch his movements, or the eyes are closed, the ataxia is materially increased and may become uncontrollable. Thus, while standing with closed eyes and the feet close together, he may gradually or suddenly fall to the ground (static ataxia), being deprived of the sense of position of the legs, unless the eyes are opened. Incoördination affecting the lower extremities is productive of the characteristic tabetic gait ("locomotor ataxia"). In attempts at walking, when the condition is well pronounced, the foot is lifted from the ground at a much higher elevation than normal, and, being poised with considerable uncertainty, it is suddenly brought to the ground, striking forcibly on the heel.

The ataxia is due to an obstruction or obliteration of afferent impulses conducted from the periphery through the various sensory tracts. It therefore follows as a result of sclerosis of the posterior columns of the cord, or of degeneration of the peripheral sensory fibres in the muscles and joints, being directly conditioned by a loss of muscle and joint sensibility.

While in some cases it is often the most striking symptom, occasionally developing during the first years of the disease in conjunction with other discoverable signs, in many instances the ataxia is scarcely perceptible or demonstrable, or does not appear at all at any time during the course of the malady. Hence the designation of the affection as "locomotor ataxia" is a misnomer.

Sensory Disturbances.—Various paræsthesiæ are often complained of, such as numbness or formication in the extremities—a sensation as if the soles of the feet were resting upon rubber or fur; a band-like feeling around the body, usually at the level of the midthoracic region, as if a tight corset or belt enveloped the body. This sensation is known as "cincture feeling" or "girdle sensation." Or the patient discovers that sensibility in his legs is absent in various areas. In the upper extremities there may be numbness in the course of the ulnar nerve affecting especially the fourth and fifth fingers. In addition to the pains and paræsthesiæ described by the patient, disturbances of cutaneous sensibility are often found upon examination, in association with the loss of muscle and joint sensibility just mentioned. Impairment of sensation may be entirely absent, but in most cases of tabes some objective sensory disturbance is always found. It may vary from slight diminution of tactile sensibility to the most pronounced analgesia. The different qualities of sensation should always be tested carefully. In a large percentage of tabetics (four-fifths) there is an area of diminished sensibility to touch situated like a band about the chest in the midthoracic region and varying from three to four inches in width. This tactile loss may be associated with analgesia in the same area. A diminution in the sense of touch, pressure sense, and the sense of position of the limbs, usually appears in the early stage, while the higher degrees of anæsthesia are developed later. The temperature sense may remain unimpaired until the last stage of the disease. But analgesia is one of the earliest and commonest forms of sensory disturbance. The areas of analgesia may be distributed over the trunk and extremities. In the lower extremities it involves the sole of the foot, the heel, the toes, the anterior or lateral portions of the legs, or the inner surface of the thigh. The transmission of painful sensations is retarded in some cases, the prick of a needle being interpreted at once as a touch, and recognized as a painful sensation from three to ten or fifteen seconds later. This is generally described as "delayed conduction."

The loss of joint sensibility and of the sensibility of the deeper muscular structure occasions interference with the normal muscular "tonus" and results in deficiency or complete relaxation of muscular tension (hypotonus). This condition will explain the fact why tabetic patients submit to extreme passive movements of the extremities without complaint of pain. It may also account for the occurrence of hyperextension of the knee-joints often seen in tabetics (genu recurvatum).

Although patients often complain of weakness in the legs, it is only in exceptional instances (in which the motor neurone system becomes implicated) that such condition can be actually demonstrated. As a rule, the muscular power and resistance to passive movement are unimpaired. When muscular atrophy or actual paralysis exists, they must be considered as complications arising in the course of the disease.

The Loss of the Knee-jerk.—One of the earliest and most constant objective signs of tabes is the loss of the knee-jerk. As a rule it does not disappear suddenly. The pains may exist for a long time before the knee-jerk disappears. In the study of a large series of cases the knee-jerk symptom is found to be variable in its character. In quite a number of instances of undoubted tabes both knee-jerks may be active, but they usually disappear as the disease advances. This occurs when the upper portion of the cord is first affected. In other cases, the knee-jerks may be well marked on one side and lost on the other. Again, they may be apparently absent, but demonstrable only upon so-called reinforcement. Both may be equally feeble, or may differ in degree of weakness.

It has been claimed that in about one per cent. of healthy people the knee-jerk is absent. The correctness