any one of these acts are similar to those underlying the simple spinal reflex, and the same lesions arresting it may arrest these acts. But the result of such arrest is more serious, for, in the case of the bladder or rectum, retention or passive incontinence of urine or fæces may follow. And if the inhibitory impulses from the brain to these centres are cut off, the voluntary control over these mechanisms is impaired, and the acts cannot be initiated voluntarily, and active incontinence may result. The location of the bladder and rectal mechanism is in the lower sacral region. Hence, when this part is the seat of a lesion, or is cut off from the brain by a lesion at a higher level in the motor tract, incontinence, either active or passive, or retention, may result.

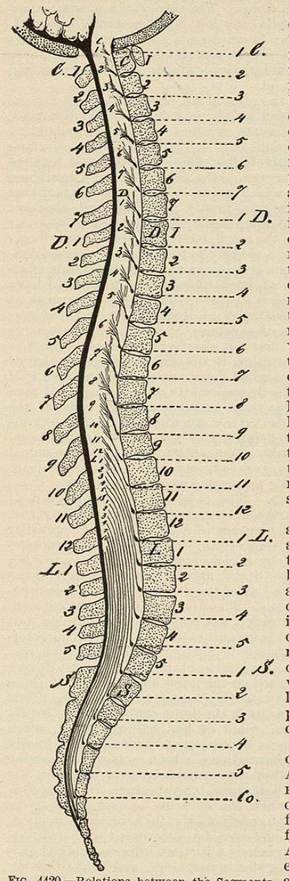

FIG. 4420.—Relations between the Segments of the Spinal Cord and their Nerves and the Bodies of the Vertebræ. (Gowers.)

A part of the automatic mechanism of respiration is governed by the cervical and dorsal regions of the cord, and is interfered with in disease in those regions. Lesions of the upper cervical region paralyze the diaphragm and thus cause death.

DISTURBANCE OF VASO-MOTOR AND TROPHIC FUNCTIONS of the cord may occur from various forms of lesion. Anterior poliomyelitis produces atrophy of the muscles paralyzed, and a sufficient affection of the vaso-motor system to cause objective, as well as subjective, coldness in the limb; and when the lesion lies deep in the anterior horn, an arrest of development of the bones of the limb affected. General myelitis is usually associated with a tendency to bedsores upon the parts exposed to pressure, which cannot be avoided by the most scrupulous cleanliness, and to cystitis, and these are ascribed to a disturbance of trophic impulses to the skin and bladder. Posterior sclerosis is sometimes associated with trophic changes, such as perforating ulcers, joint affections (Charcot's arthropathies), and eruptions on the skin. In a few cases of leprosy serious lesions of the posterior gray horns have been observed. In general myelitis there is a partial vaso-motor paralysis, indicated by cyanosis, sluggish circulation, œdema, and coldness, with abnormal sweating in the paralyzed parts. But any definite statement regarding the exact localization of vaso-motor or trophic functions in the spinal cord cannot be made as yet. And recently many vaso-motor and trophic symptoms, formerly supposed to be due to spinal lesions, have been found to be produced by disease in the peripheral nerves. It is, however, established that trophic lesions are most frequently observed when the gray matter of the spinal cord in the vicinity of the central canal, including the vesicular column of Clarke, is the part diseased; or when all sensation is cut off from the paralyzed limbs by a transverse lesion.

The regulation of urinary excretion is presided over by a centre in the medulla, and the nerve tract thence to the liver and kidneys is traced through the cervical region of the spinal cord to the first dorsal segment, where it enters the sympathetic chain of ganglia. A lesion in the lateral column of the cervical cord, by involving this tract, may cause a vaso-motor paralysis of either the liver or the kidneys. In the former case diabetes mellitus is produced; in the latter, diabetes insipidus results. It is therefore necessary, in lesions of the spinal cord, to examine the amount and constituents of the urine.

In any case of spinal disease in which it is desirable to localize accurately the lesion, it is suggested that a written summary of the symptoms be compared with the table of localization of the functions of the cord, when it will become evident, by contrasting the normal with the abnormal conditions, what part of the cord is affected. As Bramwell justly observes, "the essence of the clinical examination of the spinal cord consists in the systematic and separate examination of each spinal segment, by observing the motor, sensory, reflex, vaso-motor, and trophic conditions of its body area." Such an examination will lead to accurate diagnosis of local lesions.

But one point remains to be mentioned, that is, the relation of the various segments of the cord to the bodies and spines of the various vertebræ. As the cord extends only to the level of the second lumbar vertebra, its various segments do not lie opposite to the vertebræ from which they are named. The accompanying diagram of Gowers (Fig. 4420) displays the mutual relation between the segments and their nerves, and the bodies of the vertebræ, and no further description is needed.

M. Allen Starr.

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[1] For a full account of the grouping of these cells, see Localization of the Functions of the Spinal Cord, by M. A. Starr, American Journal of Neurology and Psychiatry, vol. iii., pp. 443 *et seq.*—Ross: Diseases of the Nervous System, p. 829.
[2] Gaskell: Journal of Physiology, 1886.
[3] See article on *Brain, Diagnosis of Local Lesions in the.*—The Motor Tract, in Vol. II. of this HANDBOOK.
[4] Schultze, F.: Ueber Secondäre Degeneration im Rückenmarke. Arch. f. Psych., xiv., from which article the figures are taken.
[5] For the anatomy of this motor tract, see the article referred to above, in Vol. II. of this HANDBOOK.

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SPINAL-CORD DISEASES: FRIEDREICH'S (HEREDITARY) ATAXIA.—The condition known as Friedreich's or hereditary ataxia was first described by Friedreich in 1861, and a detailed account published by him in 1863, with three autopsies. In 1876 he published a further paper on the subject, with a description of three new

cases. From this time on, through the investigations of Schultze, Rütimeyer, Dejerine and others, the affection came to be clearly recognized as an hereditary disease of childhood, affecting chiefly the spinal cord, and characterized by a type of ataxia hitherto undescribed. W. Everett Smith in 1885 published an important paper on the subject of "hereditary or degenerative ataxia," in which he described six cases in one family, with an autopsy. He was able at that time to collect fifty-seven cases from the literature. In 1890 Ladame made a critical digest of the subject, published in translation in *Brain*, in which he summarized the knowledge up to that year, and gave full bibliographical references. Nine autopsies only had been recorded, and five of these were in Friedreich's own cases. Oscar Richardson has recently described the post-mortem findings in a second case from the family reported in 1885 by W. Everett Smith.

Pathological Anatomy.—The spinal cord has usually been found small and somewhat imperfectly developed, which is in accordance with the apparently hereditary

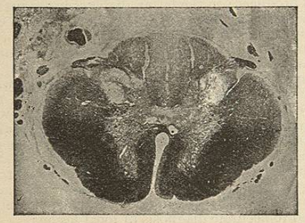

Fig. 4421.—Friedreich's Ataxia.

character of the disease. The alterations first described by Friedreich consisted in a degeneration of the dorsal tracts, atrophy of dorsal roots, and certain changes, slight in degree, in several peripheral nerves. Later study has shown that wider areas of the white matter are involved than was at first supposed, and that the gray matter also takes part in the degenerative process, though to a much less marked degree. Degeneration of the dorsal tracts to a very considerable extent is constant, with a probable constant accompaniment of degeneration of dorsal nerve roots, giving an appearance wholly analogous to tabes, and leading to the assumption that the primary sensory neurone, as such, is involved. The direct cerebellar tract is degenerated; Gowers' tract and Lissauer's bundle may be. Various observations have been made regarding the motor pyramidal tracts, and it is still in dispute whether they are in themselves involved as neurone systems in the same way that the dorsal tracts are. Degeneration in the region of the pyramidal tracts decreases from below upward, and disappears (Leyden-Goldscheider) at the lower level of the oblongata. In Richardson's case, the pathological anatomy of which he has described and which he has given me the opportunity of studying, the following lesions of the white matter were definite: degeneration of dorsal columns throughout the cord, and of dorsal nerve roots in the lumbar region; degeneration of pyramidal tracts, of somewhat lessening intensity toward the upper portions of the cord, including the uncrossed tracts in the cervical region; degeneration of direct cerebellar tracts, and in less degree of the region of Gowers' antero-lateral ascending tracts (see Fig. 4421).

In the gray matter the cells of Clarke's columns have, in certain cases, shown degenerative changes, along with the myelinated fibres of that nucleus. Alterations in other portions of the gray matter of the cord have been described, but are of somewhat doubtful character. Very few observations on the peripheral nerves have been made, but certain degenerations have been described which would be in accord with the theory of a neurone degeneration. The type of lesion in the cord is similar to that found in other sclerotic processes, an overgrowth of neuroglia following a greater or less degree of destruction of myelinated fibres. The theory of a primary overgrowth of neuroglia has not been generally accepted. Alterations in the blood-vessels in degenerated areas occur, and also have been described in the pia and nerve roots, but no characteristic significance is to be attached to the changes found.

The cause of the foregoing anatomical alterations has, in general, been sought in a defect of development, of hereditary character, leading to early degenerative changes in the spinal cord. The distribution of these changes in the relatively few cases examined post mortem has led certain observers to the assumption of a combined systemic disease, which gains weight from the fact that the disease occurs as a family affection, and does not apparently depend upon faulty blood states or vascular conditions within the cord itself. Certain cases, however, do not show a sharply systematized degeneration, although the lesions are always of a quasi-systemic character. The study of Richardson's case, to which I have already alluded, leads to the conclusion that neurones, as systems, are involved, though it cannot be said with certainty that groups of neurones, of which our knowledge is as yet deficient, may not also be degenerated. In support of this assumption is the distinct degeneration of dorsal nerve roots and of direct pyramidal tracts, as well as the characteristic degenerations of the recognized neurone systems. The most satisfactory conception of the disease, therefore, is that in congenitally defective nervous systems early degenerations of a systemic or quasi-systemic character take place, chiefly limited to the spinal cord, which progress exceedingly slowly, ultimately leading to characteristic motor and sensory disorders.

Symptomatology.—The most conspicuous feature of the disease is a characteristic inco-ordination, which is best described as a combination of a tabetic and a cerebellar ataxia. The gait is uncertain, slow, highly inco-ordinate, and accompanied by a considerable degree of swaying from side to side. Static ataxia is well marked in the extremities and head after the disease has progressed beyond its initial stages. The Romberg sign (swaying with the eyes closed) is much less constant than in tabes, but has been described in certain cases. A further characteristic motor disturbance is peculiar involuntary, chorea-like movements involving the head, which persist during rest, but are increased on intended movement. At times a definite, so-called intention tremor may develop. True paralyses do not occur in the long course of the disease, except in the late, more or less helpless stage, when weakness of muscle groups may develop, and finally paraplegia with contractures, which renders locomotion impossible. Another very constant motor disorder, but not one of the earliest signs, is nystagmus, which is usually not present when the eyes are at rest, but may be elicited by fixation, particularly in a lateral direction. This sign should, however, be interpreted with caution. Disturbance of speech is a further important sign; it is slow, difficult, irregular in utterance, and hard to understand. The tongue is tremulous and is the seat of twitching movements, suggesting, in conjunction with the speech disorder, disturbances of co-ordination similar to those observed in the extremities and possibly bearing some analogy to multiple sclerosis.

For reasons not easy of explanation the sensory sphere suffers in very slight degree, in marked contrast to tabes. With but few trifling exceptions muscle sense and skin sensibility, as well as the special senses, have been found unimpaired. The occasional occurrence of sharp pains and abnormal subjective disorders of sensibility in the extremities are of interest only because of their rarity. In view of the constant extensive degeneration of sensory areas in the cord, and the high degree of inco-ordination early developed in the disease, this lack of objectively demonstrable sensory disorders must be regarded as one of the striking peculiarities of the disease. An appeal

to vicarious or certainly added function in neurones remaining intact in an affection of very gradual progression, may be suggested by way of explanation.

The superficial skin reflexes, the pupil, bladder, and rectal reflexes show essentially no alteration, whereas the loss of knee-jerk is constant in all well-developed cases. Trophic and general vaso-motor disturbances have seldom been observed, and the sexual function remains unimpaired. A frequent occurrence, which has not received as yet a satisfactory explanation, is a deformity of the foot of the nature of a talipes equinus, or equino-varus, often with an elevated arch, shortening of the foot as a whole, development of so-called claw-foot, with particularly strong dorsal flexion of the great toe. Scoliosis is also an occasional accompaniment. Cerebral symptoms do not occur, except vertigo, and in the later stages of the disease a general impairment of the mental faculties.

Diagnosis, Prognosis, and Course.—The symptoms, —early ataxia, loss of knee-jerk, choreic movements, club-foot, disorders of speech, nystagmus, with progressive helplessness, beginning before the sixteenth year—point unmistakably to Friedreich's ataxia. With the possible exception of so-called cerebellar ataxia, tabes, and multiple sclerosis, the differential diagnosis from other organic cord affections should present no difficulties. The grouping of symptoms given above is usually well marked and is unique.

The course of the disease is steadily progressive, beginning in childhood and lasting for from twenty to forty years or even longer, death ultimately being due, in many cases, to intercurrent disease, or to cystitis or decubitus, induced by the cord changes.

Etiology.—The actual cause of the disease remains obscure. The facts that it occurs in children before or at the age of puberty, that cases have frequently been observed in the same family, though by no means constantly, that the cord, post mortem, gives indications of faulty development, have led to the suggestion that it is due to hereditary influences. Further than this nothing of value has been found. An appeal to syphilis, alcohol, or various neuroses or psychoses in ancestors does little to elucidate the matter. Nor is it profitable in a disease of this character to lay stress upon possible exciting causes. As in all disease, there is a predisposition, which is rather the statement of a self-evident fact than an explanation, so long as we remain in complete ignorance of what constitutes predisposition. The fact of importance is that in certain families the affection has appeared in several members. As given by Gowers, sixty-five cases were distributed in nineteen families, and ten occurred in one family. Sporadic cases are probably more frequent than is ordinarily supposed. It has been a frequent observation that many cases occur in one generation in families in which the parents or ancestors were not victims of the disease—so-called indirect inheritance. The affection is, therefore, to be regarded as one of the group of "family diseases."

Treatment.—In the present state of our knowledge, treatment must remain essentially unavailing, except as directed toward the amelioration of symptoms. Systematic exercises (Frenkel), avoidance of overexertion, careful attention to general hygiene, good food, and fresh air, with such drugs as are symptomatically required, must constitute our main reliance. *E. W. Taylor.*

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SPINAL-CORD DISEASES: HEMORRHAGE IN THE CORD.—(Synonyms: Hæmatomyelia; spinal apoplexy.) Hemorrhage in the spinal cord is very rare in comparison with hemorrhage in the brain. When it occurs it is usually of small extent. This is not extraordinary when the size of the organ and the firmness of its connective-tissue sheath, and the low pressure in the spinal arteries, are considered. As a rule, the clot in the spinal cord is long and narrow. It destroys a considerable part of the cord at one segment, and extends into the segments above and below, injuring these to a less extent. The clot is usually found in the gray matter of the cord, most frequently in the anterior horns, then in the posterior horns, and rarely in the white columns. Sometimes the surface of the cord is broken and the blood infiltrates the pia mater. Occasionally a large number of small clots are found at different levels. As the patient rarely dies at once of hemorrhage the clot is usually found in a state of decomposition, and the cord around it is infiltrated with blood cells, pigment granules, and hæmatin crystals. If the hemorrhage is capillary—as sometimes occurs—it is detected by the presence of pigment and crystals among the degenerated spinal elements. Around the clot the spinal cord is usually found in a state of softening, which is red in recent cases, and white when the process has been a long one. At a post-mortem examination the question sometimes arises whether the condition found is a myelitis with secondary hemorrhage, or a hemorrhage with secondary myelitis. In the former case the microscopic examination shows a greater preponderance of granular corpuscles and leucocytes, a greater degree of degeneration in the nerve cells, and a greater extent of connective-tissue growth. Secondary degenerations upward and downward from the segment destroyed, and secondary degenerations in the motor nerves from the segment affected to the muscles, are observed after spinal hemorrhage as after myelitis. The meninges are rarely involved.

Etiology.—Spinal hemorrhage is met with in males more frequently than in females, and in youth and middle age most often. The predisposing causes are chronic changes in the general arterial system, hemorrhagic diathesis, purpura, and myelitis of the spinal cord. The exciting causes are injuries to the vertebral column and cord and extreme muscular effort, also the sudden exposure to a marked change of atmospheric pressure. A number of cases of cervical hemorrhage have occurred from sudden bending forward of the neck.

Symptoms.—As a rule, the symptoms begin suddenly, and the patient is seized in a moment with complete paraplegia and intense pain in the back at the level of the hemorrhage, and shooting pains in the body and stiffness of the spine. A spinal apoplexy usually comes without any warning, after a sudden effort. When premonitory symptoms have existed for a few days it is probable that the case is one of acute myelitis. The extent of the paralysis depends upon the level of the lesion. (See article on *Spinal-Cord Diseases: Diagnosis of Local Lesions in the Cord.*) If it is located in the lumbar or dorsal region, the lower half of the body only is involved; if in the cervical region, the arms are affected as well. The hemorrhage is rarely so very limited as to affect one extremity only, but it is not infrequently the case that the symptoms are more marked on one side, and in some cases the symptoms of a Brown-Séquard type have occurred. The paralysis is total, no voluntary motion is possible, the limbs are relaxed and never rigid. The muscles do not atrophy or present any change in the electric reactions, excepting those which are supplied with nerves from the segment which is destroyed by the hemorrhage. The condition of the reflex action depends upon the seat of the lesion: it is suspended at the level of the hemorrhage, and increased in the segments below it. If the hemorrhage is in the cervical region there is an atrophic flaccid paralysis of the arms and a spastic paralysis of the legs. In such a case there may be ventro-pupillary symptoms also.

If the hemorrhage is confined to the central gray matter and the patient survives, the symptoms may resemble those of syringomyelia. The bladder and rectum are uniformly paralyzed, and various forms of incontinence of urine and fæces result, dependent upon the seat of the lesion. If this is situated high up in the

cord, the viscus may empty itself occasionally, as the need arises, unconsciously to the patient and independently of his effort or control. If the lesion is low down (below the eleventh dorsal segment), there is usually retention of urine and fæces, or complete relaxation of the sphincters. Cases sometimes occur of hemorrhage into the lower sacral region and conus terminalis from injuries; and in such cases there may be paralysis of the bladder and rectum only, with a small heart-shaped area of anæsthesia about the anus and buttocks. Vaso-motor paralysis accompanies the voluntary paralysis, and results in cyanosis and coldness of the paralyzed limbs, and predisposes the parts to the development of bedsores. The latter appear, very soon after the hemorrhage, on the prominent parts of the body which are subjected to pressure; voluntary movements to relieve such pressure, or reflex movements for the same purpose, being impossible on account of the lack of power and of sensation, for complete anæsthesia and analgesia exist in the paralyzed limbs from the outset. Pain in the back, which occurs at the time of hemorrhage, does not usually persist. The danger in these cases is from the occurrence of bedsores, or of cystitis, and consequent infection from these sources, with the development of septic fever. If the hemorrhage involves the respiratory centres in the upper cervical region, sudden death takes place. This is rare.

The symptoms described are those of a severe hemorrhage, sufficient to destroy one or more segments of the cord. As a rule these symptoms gradually subside as the pressure of the clot is removed and the only permanent symptoms are those which are due to the actual destruction of the spinal elements, viz., limited atrophic paralysis and localized anæsthesia. Dissociated anæsthesia, viz., a loss of the senses of pain and temperature with preservation of the sense of touch, is not an uncommon result in the parts below the segment affected. If the clot is a small one, however, the symptoms may be more limited and less serious, partial anæsthesia and localized paralysis, with atrophy, being the result. This, however, is the exception. Capillary hemorrhages give rise to widespread symptoms, which so closely resemble those of diffuse myelitis that differential diagnosis is impossible.

The PROGNOSIS is very bad in spinal apoplexy, for a destruction of the spinal elements cannot be recovered from. The patients either die suddenly, or die of complications, or linger on for months with all the symptoms of chronic myelitis. In the lighter cases they may recover sufficiently to get about, but some evidences of the hemorrhage in the form of local paralysis or areas of anæsthesia always remain.

The DIAGNOSIS is usually easy, the sudden onset of total paraplegia being characteristic. In meningeal hemorrhage the paralysis is less marked than the spasms, and anæsthesia is rare. In poliomyelitis anterior there is marked constitutional disturbance, with fever, before the paralysis begins, and anæsthesia is not present. In acute central myelitis the onset is more gradual, the symptoms develop successively, they extend gradually to other parts, and fever is usual.

TREATMENT cannot reach the disease, but the patient should be kept absolutely quiet in the prone posture in bed, ice should be applied to the spine, and ergot should be given freely. The subsequent treatment resolves itself into care of the patient, and such measures as are used in the management of a case of chronic myelitis.

M. Allen Starr.

SPINAL-CORD DISEASES: LATERAL SCLEROSIS.—(*a*) PRIMARY LATERAL SCLEROSIS.—(Synonyms: Spastic spinal paralysis; Tabes dorsal spasmodique.)

Definition.—The disease is characterized by a gradually progressing paralysis, which begins in the lower extremities, is accompanied by greatly exaggerated tendon reflexes, muscular rigidity, and contractures, and is not attended by sensory, trophic, or visceral symptoms. The anatomical basis is supposed to be a primary sclerosis of the antero-lateral columns.

Etiology.—We know very little of the causes of the disease. It occurs chiefly in adults, more frequently in men than in women. Exposure, excessive venery, syphilis, trauma, infection, lead poisoning, etc., have been assigned as causes in individual cases. Some cases occurring near Rome, several of which were in one family, seemed to be due to the effects of a leguminous article of diet, *lathyrus cicera*. In quite a number of instances heredity was the chief factor in causation; the disease occurring distinctly as a family disease, having been found in a number of generations, with often a number of cases in the same generation. For instance, in one of Strümpell's cases cases the grandfather, father, two uncles, and a brother were supposed to have been similarly affected; in one of Spiller's cases fourteen, in five generations, were believed to have had the disease. In some of Strümpell's cases the disease appears to have begun in early, in others in late adult life. In Spiller's and a number of other reported cases it began in early childhood. In most of these cases the disease appears to have been very slow in its progress.

Symptoms.—The disease begins with weakness in one or both lower extremities. There is an undue sense of fatigue on exertion, and an objective examination shows a slight paresis. There is at the same time some difficulty in walking, this being at first most noticeable on rising in the morning. As the paresis increases, motor irritation symptoms are soon manifested. These are at first slight clonic or tonic spasms of the affected muscles. They are most likely to occur when the patient is fatigued, are easily evoked by active or passive movements of the limbs, but often come on in the middle of the night. The tendency to muscular spasm—brought on by either a voluntary or a passive movement, or in a reflex way—increases to such an extent that complete muscular rigidity and contractures of the limbs occur. This condition antagonizes every action of the patient, makes his voluntary efforts altogether futile, and, therefore, causes the paralysis to appear more complete than it really is. The rigidity can usually be overcome, in early stages of the disease, by slow persistent pressure, but when it becomes excessive it resists powerful efforts. The usual position of the rigidly contracted limbs is that of extension at the knees, the feet in the equino-varus position, and the thighs firmly pressing against one another. Their immobility is often interrupted by clonic spasms. Occasionally the limb is drawn for a short time into another position.

A striking and usually an early symptom is the exaggeration of the deep reflexes. The patellar tendon reflex is greatly exaggerated; muscular contractions can be elicited by striking any of the tendons—for instance, the inner or outer hamstrings, where tendon reflexes cannot be elicited, as a rule, in health,—and even tapping over the periosteum will produce like manifestations. But the most striking of these phenomena is the ankle clonus, rapid and rhythmical clonic contractions taking place when the foot is sharply flexed, and continuing as long as the foot is held in a flexed position. When the reflexes are much exaggerated the clonic contractions, on evoking the ankle clonus, may not be limited to the ankle, but may extend to all the muscles of the extremity. The ankle clonus also becomes a disturbing element in walking, as every time the body rests on the toes in progressing forward there is a tendency to its production. The most notable change in the superficial reflexes is the presence of the Babinsky toe phenomenon.

The gait is very characteristic. As the legs are weak and stiff the feet cannot be freely lifted, and, when moving forward, they sweep the floor, making an almost characteristic scraping sound. At the same time, in order that the foot can be brought forward, it makes a wide outward sweep. The gait is sometimes further impaired by clonic spasms of the muscles—those representing the foot clonus, as just described—and these spasms may cause a temporary halt in walking, or may make that act altogether impossible. Walking is more difficult on an up or down grade, and on an uneven surface, than on

the level and on an even surface. It usually becomes easier after the patient has walked for a while. The gait just described is that observed when the spastic symptoms are already well marked. At a later period, when there is complete rigidity of the legs, walking is impossible.

The disease slowly extends upward, involving the muscles of the abdomen, back, and upper extremities. The latter are usually affected to a less degree than the lower extremities, though exaggerated reflexes and muscular tension are associated with the paresis. The upper extremities may even be rigidly contracted, in which case the position is usually one of slight flexion and pronation of the fore-arm, and strong flexion of the wrists and fingers, the arm being pressed firmly against the body.

In typical cases there are no further symptoms on the part of the nervous system. The sensibility is intact, the functions of the bladder and rectum are normal, there are no trophic changes in muscles or skin, and no special changes in the electrical reactions.

The progress of the disease is usually very slow. Though the patient be altogether bedridden and without power of motion, the general health need not suffer. Unless there be some complication he may live to old age.

The symptoms of spastic spinal paralysis are found in Little's disease, a congenital paralysis usually due to disease which has developed during fœtal life, or which is the result of injury to the brain in parturition. The brain disease, in these cases, is usually shown by such conditions as strabismus, speech disturbances, idiocy or imbecility, epilepsy, and the like. But in some instances there are no cerebral symptoms; the disease appears to be purely spinal and, therefore, belongs to this category. The pathological state, however, is likely to be due to a developmental defect. A definite determination of this question must await future pathological findings. Improvement in such cases is not uncommon.

The following case, at present under the writer's observation, illustrates this class of cases: I. J——, a boy eleven years of age, did not learn to walk as other children. Prior to that time nothing abnormal was detected. The spastic condition is now very marked. As he stands he rests altogether on his toes—the heels cannot be brought to the ground—and the thighs are pressed firmly together. When he tries to walk, which he can do only if well supported, the thighs rub each other and the legs cross. When the patient sits the feet and lower legs sway in the air. The rigidity of the limbs is such that much force is necessary to make any passive movements. The Babinsky phenomenon is present, and all the deep reflexes are exaggerated. The ankle clonus, as well as those of the Achilles and patellar tendons, can be elicited. The intellect, sensation, and the functions of the bladder and rectum are unimpaired, and there is no evidence of the involvement of the upper extremities or of the cranial nerves. The disease, therefore, appears to be limited to the motor tracts of the cord.

Morbid Anatomy and Physiology.—Symptoms like those of spastic paralysis had been observed in connection with various diseases, but Erb was the first to describe this as a separate and distinct disease, whose basis he believed to be a primary sclerosis of the antero-lateral columns, especially the pyramidal tracts. His views have been quite generally accepted, but yet, it must be acknowledged, there has not been much positive evidence to substantiate them. In a number of post-mortem examinations, where this disease was diagnosed during life, lesions in the brain, tumors in the medulla oblongata, diffuse sclerosis, etc., were found. In some instances, nevertheless, the disease appeared to be a primary sclerosis of the lateral columns. But, whether or not a primary disease be usually found, it is probable that the pathological changes in the lateral columns produce the symptoms of this disease, for when spastic symptoms are found with multiple sclerosis, diffuse myelitis, etc., the lateral columns are found to be affected.

The paresis or paralysis is explained by the destruction of nerve fibres in the pyramidal tracts, the tracts conveying voluntary motor impulses. The motor irritation symptoms cannot be so satisfactorily explained. Charcot believed them to be due to dynamic changes in the large ganglion cells, these being placed in a state of irritation by the degenerated nerve fibres of the pyramidal tracts. Hughlings Jackson supposed that in these cases the influence of inhibiting centres in the brain had been removed by the destruction of the pyramidal tracts, and that the excessive motor manifestations were due to the absence of this influence.

Diagnosis.—The symptoms of spastic paralysis may appear with multiple sclerosis, transverse or diffuse myelitis, brain lesions, etc. In order to establish the diagnosis in these cases we must look for the further symptoms of these various diseases, as, for example, the indications of involvement of the gray matter and posterior columns in transverse myelitis; optic atrophy, nystagmus, intention tremor in multiple sclerosis, etc.

Especially in multiple sclerosis there may for a long time be no other symptoms than those of spastic paralysis. For that reason, when the latter symptoms are present, one should always be on the watch for other symptoms, symptoms that may indicate the presence of another disease.

It is to be remembered that this disease is one of long duration, that post-mortem examinations have been made only many years after its inception. In those cases, therefore, in which other changes have been found than those in the lateral columns—in Gowers', Goll's, the cerebellar tracts, etc.—the primary disease may have been only in the lateral columns. At present we can only make a diagnosis of spastic spinal paralysis. We cannot be certain, during life, that the disease in the lateral columns is primary, or that it is altogether limited to that part of the cord.

Prognosis.—When uncomplicated the disease does not appear to shorten life. It is sometimes capable of improvement, and even cures have been reported.

Treatment.—The treatment applicable in these cases is that usually adopted in locomotor ataxia and other forms of chronic disease of the cord. Rest in bed for a number of weeks, especially when there seems to be an exacerbation of the disease, may produce amelioration of the symptoms. Hydrotherapy, electricity, massage, applications of the thermo-cautery along the spine, the administration of various drugs, are among the remedial measures that may be used. In the spinal form of Little's disease tenotomy and other orthopedic measures, together with rest, have sometimes produced good results.*

(*b*) AMYOTROPHIC LATERAL SCLEROSIS.—For both the description of the symptoms and knowledge of the anatomical character of this disease we are chiefly indebted to Charcot. Though somewhat akin to the disease just described, it presents striking differences both in its symptomatology and in its morbid anatomy. It occurs chiefly in persons of middle age, but has been observed in children.

Symptoms.—The disease usually begins with slowly progressing paralysis of the upper extremities, which is soon accompanied by atrophy and fibrillary contraction of the paralyzed muscles. Subsequently muscular rigidity and contractures occur, the arms assuming the position found in spastic paralysis, viz., partly flexed at the elbows and pressed against the body, forearms pronated, hands and fingers strongly flexed. These contractures may remain even when the muscles are almost completely atrophied. Usually, after the lapse of a number of months the lower extremities become involved. In them the manifestations are mostly like those of spastic paralysis—paralysis, exaggerated tendon reflexes, muscular rigidity, contractures,—while little or no atrophy of the muscles is observed. The electrical reactions are altered

* Since this article was written Mills and Spiller have reported to the American Neurological Association a case in which there appeared to be a primary sclerosis limited to one pyramidal tract. The pathological findings are also given in the report.