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.

according to the degree of muscular atrophy. In the lower extremities there may be a mere quantitative change—diminution of electrical irritability—while in the upper extremities the alteration is usually more marked, and, when the muscular atrophy advances rapidly, as often occurs in the small muscles of the hand, the typical reaction of degeneration may be found.

At a still later period bulbar symptoms, those of glosso-labio-laryngeal paralysis—atrophy of the lips and tongue, difficulty in deglutition, indistinct speech, respiratory disturbances—appear, and finally carry off the patient.

The symptoms do not always appear in the order described. Sometimes the disease begins in the lower extremities, extending thence to the arms, and finally to the cranial nerves. Sometimes the disease begins as a bulbar paralysis, though it need not, therefore, run a rapid course. The writer has at present under observation a case which began with bulbar symptoms two years ago, and now presents a typical picture of the disease. In this case the muscles supporting the head upon the trunk are, also, almost completely paralyzed.

The degree to which spastic symptoms, and to which muscular atrophy appear in both upper and lower extremities, is also quite variable, depending upon the part of the nervous system in which the morbid changes began, and upon the extent to which the gray and white nervous tissues are respectively affected.

The duration of the disease is usually from one to three years. The well-marked cases hitherto recorded terminated fatally. Death is generally caused by the bulbar symptoms.

Morbid Anatomy and Physiology.—The morbid changes are almost, if not altogether, limited to the motor central and peripheral nervous apparatus. There is sclerosis—atrophy of the nerve fibres and increase of the connective tissue—of the direct and crossed pyramidal tracts in the cord, often extending above the crossing of the pyramids in the medulla. In some instances the atrophy has been followed the whole length of the motor pathway to the central convolutions, and atrophied pyramidal cells have been found in the cortex of these convolutions. There is atrophy of the large ganglion cells of the anterior cornua, and of the cells of the nuclei of the affected cranial nerves. Certain of the cranial nerves, of the anterior roots of the spinal nerves, of the mixed nerves and of the muscles, are also found in various stages of atrophy.

As to the explanation of the symptoms, the muscular atrophy is due to the atrophy of the large ganglion cells, the bulbar symptoms to disease of the nuclei of the cranial nerves, the spastic phenomena to sclerosis of the pyramidal tracts. The preponderance of spastic or atrophic manifestations is due to preponderance of the morbid process in the white or gray matter respectively.

Diagnosis.—While the disease is still confined to the upper extremities it may be mistaken for subacute poliomyelitis anterior, or even for progressive muscular atrophy, for the weakness, fibrillary tremor, and other manifestations—atrophied thenar and hypothenar eminences, claw-hand, etc.—are very much like what is observed in the latter disease. But its progress, even when unusually slow, is more rapid than that of progressive muscular atrophy. Furthermore, spastic symptoms, exaggerated tendon reflexes, some muscular rigidity, etc., are likely to be manifest at an early period, thus aiding us to distinguish it from the aforementioned diseases.

When the disease begins in the lower extremities or as a bulbar paralysis, it is as yet impossible to make a diagnosis of amyotrophic lateral sclerosis. At times in the bulbar form, spastic phenomena—the jaw clonus—are found. When the disease has developed in its typical form, with atrophic paralysis in the upper and spastic paralysis in the lower extremities, as well as with bulbar symptoms, no mistake is possible.

Myelitis, multiple sclerosis, gliosis, syphilis, may be so localized as to simulate this disease. But usually other symptoms—sensory and trophic symptoms and symptoms on the part of the bladder and rectum—will appear and thus differentiate these diseases from amyotrophic lateral sclerosis, which has only motor symptoms.

The prognosis is sufficiently indicated in the foregoing description. Such treatment may be resorted to as is employed in other chronic diseases of the cord.

Philip Zenner.

SPINAL-CORD DISEASES: MENINGEAL HEMORRHAGE.—Spinal hemorrhage is of two varieties: extrameningeal, between the dura mater and the bones; and intrameningeal. The last named may occur between the dura and the arachnoid, or between the latter membrane and the pia mater. The extrameningeal variety is the commoner.

Etiology.—Both on this score and on that of symptomatology a good deal of error appears to have been perpetuated by writers of text-books. The explanation of this fact lies in the relative infrequency with which the disease in question occurs. Hence writers are compelled to formulate their opinions on theoretical grounds rather than on actual observation in the clinic and in the pathological laboratory. The causative importance of spinal injuries seems particularly to be overestimated. In the writer's experience traumatic hemorrhage, either within or without the spinal envelopes, of sufficient extent to produce definite symptoms, is almost too rare—even in severe spinal fractures and fracture dislocations—to demand serious consideration. In the minor spinal injuries the symptoms attributed by many writers to meningeal hemorrhage could be attributed more logically to the pinching of nerve roots or to the transitory pressure on the cord resulting from the spinal distortion produced by the injury.

Meningeal hemorrhage occurs at all ages and is more common in men than in women. In early childhood it is never spontaneous; it merely gravitates into the spinal membranes from those of the brain, as in the case of the birth palsies. It is sometimes found post mortem in severe convulsive conditions, epilepsy, chorea, tetanus, puerperal eclampsia, and strychnine poisoning. It is also found in the hemorrhagic forms of certain acute specific diseases, such as smallpox and yellow fever; rarely as an isolated phenomenon in typhoid fever. In most of these cases it is probably an agonal phenomenon and is not recognized at the time of its occurrence. Extradural hemorrhage is a rare result of the bursting of an aortic aneurism, after erosion of the bodies of the vertebræ. Within the dura hemorrhage may result from the rupture of an aneurism of the vertebral artery.

Pathologic Anatomy.—Extradural hemorrhage is usually small in amount; it comes from the venous network which lies between the dura and the bone and is found mainly posteriorly, this situation being favored by gravitation and by the fact that the space between the dura and the bone is greater posteriorly than anteriorly. In very large effusions the blood may make its way out between the intervertebral foramina, along the nerves. The cervical region forms the commonest site for this variety of hemorrhage, and the blood is usually found more or less coagulated.

In making a post-mortem examination care should be taken, as Gowers has pointed out, not to fall into the error of considering as ante-mortem hemorrhage the blood which has gravitated into the meningeal veins and is subsequently released into the retrodural space when the canal is opened.

Intrameningeal hemorrhage varies greatly in amount. In the subarachnoid form it comes usually from the pial vessels and may surround the cord for a short distance, or fill the whole subarachnoid cavity. It has even been known to force its way through the valve of Vieussens into the cerebral ventricles. The cerebro-spinal fluid is often blood-stained, a fact which may lead the observer into thinking the hemorrhage larger than it really is. In cases more than a few days old there are usually signs of meningitis, set up by the irritation of the effused blood.

Symptoms.—To produce any symptoms whatsoever the hemorrhage must be extensive. Such symptoms are

at first irritative and result mainly from the more or less complete asphyxiation of the nerves which pass through the hemorrhagic area. The first indication is, as a rule, sudden with severe pain in the back. This pain is most intense at a point corresponding to the site of the hemorrhage, but radiates up and down the spine. With this pain in the back are others of a paroxysmal character, due to the irritation of the spinal nerves involved in the hemorrhage. These vary in distribution with the level of the cord implicated. Between the paroxysms of pain all sorts of disagreeable sensations may be experienced in the regions supplied by these nerves, the result of perverted function. More or less muscular spasm usually accompanies the pain; this may affect the vertebral muscles only so far as to cause rigidity of the spine, or it may produce actual opisthotonos; for the rest, it involves in part the muscles supplied by the affected nerves, and in part the muscles supplied by the cord below the hemorrhagic focus. There is usually spasmodic retention of urine. In a short time more or less paralysis follows this stage, but there is rarely if ever a condition of complete paraplegia. As a rule the sensorium remains clear throughout; occasionally unconsciousness may develop as a result of shock, and coma or delirium may come on.

Diagnosis.—Hemorrhage into the membranes, if at all severe, is usually marked by the group of symptoms above mentioned: sudden intense pain in the back, with spasmodic pain and other phenomena indicative of perverted function in the region supplied by nerves implicated in the hemorrhagic focus, muscular spasm followed by partial paralysis, spasmodic retention of urine, etc. In hemorrhage into the substance of the cord the pain is very much less in degree and more distinctly local, the paralysis is immediate, much more extensive and persistent, and the resulting atrophy more pronounced; the sphincteric disturbance is very marked and priapism is often present; the reflexes are abolished if the hemorrhage involves the entire transverse area of the cord, and increased if the lateral columns are mainly implicated. Should the cord hemorrhage make its way into the membranes the symptoms of both lesions will be combined, but even then the chronological order of the symptoms will enable one to follow the course of events. Meningitis has a more gradual onset, and fever is present from the outset; meningitis may, however, be started up by a hemorrhage, a fact that should be borne in mind. In myelitis the pain is very rarely prominent and the irritative phenomena so common to hemorrhage are *nil* or nearly so. Tetanus is differentiated by its more gradual onset, the presence of trismus and the absence of the severe spinal pains.

Prognosis.—In stormy cases the symptoms may reach their maximum in a few hours and death may result. This is particularly true if the cervical region is the one affected, owing to the implication of the phrenic nerves. If the patient survives the shock and exhaustion which accompany the culmination of symptoms, very gradual improvement may take place, with ultimate complete recovery. The danger is that the decline of the hemorrhagic symptoms may be followed, after a few days, by the onset of those of a meningitis which may prove fatal. If, however, the meningitis be of only moderate severity, it will begin to abate at the end of a week or ten days and gradual improvement will follow; complete recovery is a matter of many months.

Treatment.—Absolute rest is a prime requisite. The patient should be laid upon an air or water mattress and made to lie upon the face or side as much as possible, in order to have the spine elevated. The bladder and rectum should receive careful attention. Early scarification beside the spine, with free letting of blood, is a logical procedure, if the anatomical connection between the venous plexuses and the veins in the postvertebral tissues be considered. This may be followed by the application of ice to the spine and the internal administration of some form of ergot. The pain should be controlled by morphine or other sedative. If acute meningitis follows the hemorrhage, the treatment for this condition elsewhere indicated should be followed. The resultant paralyses are best dealt with by electricity and massage.

Surgical interference has been advocated by many in cases in which the early symptoms progress rapidly and the patient's strength remains good. The writer's experience with spinal surgery does not lead him to a warm advocacy of this measure. *Joseph W. Courtney.*

SPINAL-CORD DISEASES: MYELITIS.—Etiology.—Myelitis is one of the rarer forms of disease of the spinal cord. With the exception of certain special varieties, it is usually a disease of early adult life, and grows more infrequent in middle life and old age. The male sex seems to be much more predisposed than the female. Thus, among thirty-three cases under my observation there were twenty males and thirteen females. This discrepancy is probably owing to the greater frequency of the exciting causes among males.

The exciting causes of myelitis are numerous. The most frequent one, in the writer's experience, has been exposure to cold and wet. The disease is observed not infrequently after a debauch, if the patient has been compelled to sleep on the damp ground or lie in the gutter, etc. It may also result from working in ditches, or even from a brief exposure to wet and cold.

Traumatism plays a much less considerable part in the etiology of this disease. The injury may be inflicted directly upon the spinal cord, as the result of gunshot and stab wounds, direct blows on the back, or even blows on the head. Injury to adjacent parts may also lead to myelitis. For example, in one of the writer's cases an injury to the back gave rise to a myositis in the erector spinæ muscle, attended with suppuration. After a long interval the pus made its way along the roots of the spinal nerves, through the intervertebral foramina, to the pia mater, where it set up an acute meningitis, and then extended to the substance of the cord, setting up myelitis. A certain amount of myelitis always attends spinal meningitis, but it is usually confined to the outer segment of the cord, and does not produce any notable symptoms.

Myelitis is also a frequent result of injuries to the spine (fractures, dislocations, etc.) which are attended with direct pressure on the cord; or it may occur in spinal concussion, when there is no evidence of injury to the external parts. In the latter form, however, the myelitis is more apt to be subacute or chronic.

Crocq reports a case of acute myelitis resulting from forced extension of the cord. So-called compression myelitis is also observed quite often, usually as the result of Pott's disease, more rarely of cancer or other tumors of the vertebræ. In the majority of cases, however, evidences of inflammation of the cord are wanting in this condition and the post-mortem appearances indicate degeneration due to interference with circulation. In some cases, however, evidences of myelitis are manifest.

In a considerable proportion of cases myelitis follows an infectious disease, being due either to the presence of bacteria in the cord or more frequently to the action of toxins circulating in the blood. Thus, cases have been reported after diphtheria, dysentery, typhoid fever, influenza, gonorrhœa, smallpox, measles. Schiff reports a case of acute hemorrhagic myelitis during the course of typhoid fever in which a fatal termination occurred eighteen hours after the onset of the spinal symptoms.

Among the rarer infectious sources of myelitis may be mentioned erysipelas, malaria, pneumonia, whooping-cough, scarlatina, puerperal sepsis, and streptococcus infection. Quite a number of cases have been reported in which a bronchiectatic cavity has been the starting-point of infection. Flatau reports a case which began several weeks after a suppuration of the antrum of Highmore.

Marinesco believes that the poison acts indirectly through the blood-vessels or directly upon the nerve tissues.

Moltschanoff found, upon injecting guinea-pigs and