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

white mice with gonococci toxins, that symptoms developed on the part of the nervous system, and marked changes in the cord were discovered on microscopic examination (colossal vacuolization of all the cells of the anterior and posterior horns).

It has also been claimed that myelitis may be due to tuberculosis or syphilis independently of local specific lesions.

Not a few cases have been reported as the result of acute or chronic poisoning with lead, arsenic, alcohol, or coal gas.

Formerly it was supposed that myelitis was not infrequently secondary to a cystitis or to inflammation of other parts of the urinary tract, the intervening link consisting of an ascending neuritis. At the present time we are inclined to believe that such cases are due to infection with the toxins of gonococci or streptococci.

In this connection we may mention an interesting case, reported by Dupré and Delamare, of a man, aged twenty years, with scoliosis of the dorsal spine since childhood. For ten years he had had a tuberculous process in the left upper maxilla and this was finally curetted. Two months later an apoplectiform paraplegia developed, with pronounced disturbances of tactile and thermal sensation in the legs. Atrophy of the muscles set in, with extensive bedsores and interference with micturition. After a short illness the patient died in a delirious condition. The diagnosis was tuberculous spondylitis and compression of the lumbo-dorsal cord. At the autopsy the spine was found to be intact, but there was extensive hemorrhagic pachymeningitis which encircled the lower dorsal cord like a thick ring. There were also secondary myelitic changes in various segments of the dorsal and lumbar cord. The microscope showed various stages of tuberculous inflammation of the dura. In the cord were found scattered islets in which the parenchyma and glia exhibited extensive degeneration, and there were irregular hypertrophy, with deformity of the posterior horns, and proliferation of the epithelial cells of the ependyma of the central canal.

Finally, in a considerable proportion of cases, we are unable to discover any adequate cause for the development of the disease.

Myelitis may be divided into several varieties, viz.: (1) *Transverse myelitis*, in which the entire thickness of the cord is affected, while the longitudinal involvement is slight, compared to the entire length of the cord; (2) *disseminated myelitis*, in which numerous small foci are scattered irregularly through the cord (this form is very apt to be complicated with a similar condition of the brain, really constituting an encephalo-myelitis); (3) *compression myelitis*; (4) *poliomyelitis*, in which the gray matter is the chief site of the lesion (this will be discussed in a separate article); (5) *purulent myelitis*, in which the disease terminates in the production of abscesses; (6) *chronic myelitis*.

Clinical History.—*Acute transverse myelitis* is rarely preceded by prodromal manifestations, unless this term be applied to the first symptoms of the beginning inflammation. In rare cases there is a feeling of general malaise, slight shivering sensations, or a distinct chill, and there may even be a certain degree of mild delirium; even general convulsions have been observed at the onset of acute dorsal myelitis, and still more often in cervical myelitis. Marked febrile movement is not often noticeable, however acute the inflammation may be.

The onset varies considerably in its degree of suddenness. In rare cases the disease begins as quickly as an attack of cerebral apoplexy. The patient suddenly feels a sense of weight in his lower limbs, this increases, and at the end of a few minutes the limbs are paralyzed. Or the onset extends over a week or more. At first a feeling of numbness, or of pins and needles, is noticed in the soles of the feet; this gradually spreads up the limbs, accompanied by increasing weakness of the parts, perhaps by severe pains in the muscles, particularly the calves of the legs, or by painful spasms of the muscles. Then the anæsthesia and motor paralysis continue to increase, sometimes uninterruptedly, sometimes by fits and starts, until they have attained their greatest intensity. The paralysis may be so complete that the patient is unable to move even the toes to the slightest extent.

Pain in the spinal column is not a pronounced symptom, nor do the patients, as a rule, complain much of tenderness in this region. The pains in the paralyzed muscles, sometimes in the joints, are extremely violent in certain cases, so that I have even known this condition to be mistaken for acute articular rheumatism. These pains may continue during the entire acute stage of the disease. A more constant symptom is the cincture feeling, a term applied to a painful sense of constriction around the abdomen, or thorax, varying according to the site of the disease in the spinal cord; this is usually attended with circumscribed tenderness over the corresponding portion of the spine. The cincture feeling is sometimes described by the patients as confined within very narrow limits, like those of a cord or thin ribbon; sometimes it is as broad as the hand.

In the majority of cases the pains in the limbs disappear as the paralysis becomes more pronounced, but in some instances they persist, even despite complete motor and sensory paralysis (anæsthesia dolorosa). When the pains in the limbs are severe they are usually attended with spasm or contracture of the affected muscles.

The bladder and bowels, especially the former, are usually implicated at an early period. The patient first experiences difficulty in passing water and must strain a good deal to start the flow. Finally, the inability to urinate may become complete, and the use of the catheter is necessary. Or the sphincter also becomes paralyzed, and incontinence results from overflow. The bowels are affected in a similar way, though less frequently. At first there is constipation, sometimes attended with extreme tympanites, from paralysis of the muscular coat of the bowels. Or the stools may pass away involuntarily, and, if anæsthesia is profound, this occurs without the patient's knowledge. In many cases, however, the functions of the bowels are quite undisturbed throughout the entire course of the disease. Even when voluntary evacuations are completely lost, the action of the bowels may sometimes be stimulated by reflex means, as by the passage of a catheter into the bladder, the irritation of the perineum, etc.

The cutaneous and tendon reflexes vary according to the exact site of the disease. Trophic symptoms are not an important feature in myelitis, apart from the atrophy of muscles seen in inflammation of the anterior horns of the cervical and lumbar enlargements. They are observed most frequently in the integument. Vesicular eruptions sometimes appear upon the paralyzed parts, and there may also be excessive desquamation of the outer layers of the skin. The formation of bedsores, which sometimes occur at such an early period as to indicate their trophic origin, will be considered later. We may also call attention to the fact that the changes in the urine, which are so common in this disease, are regarded by some writers as the result of trophic changes in the secreting structures of the kidneys.

Enlargement and tenderness of the knee-joints, sometimes attended with distinct effusion, occur rarely, and their interpretation is obscure. I have also found a similar "rheumatoid" condition in the ankle-joints in a few cases of myelitis.

Gowers reports a remarkable case of trophic disturbance in a patient suffering from subacute myelitis, in which the myelitic symptoms were complicated by cellulitis of the lower part of the abdomen. At the autopsy no local cause of the cellulitis could be discovered.

Vaso-motor symptoms are more frequent than the trophic changes, but possess little clinical importance. The temperature of the paralyzed parts is often changed. At first it is sometimes increased, but later, as a rule, it is decidedly lower than that of other parts of the body. Pitting of the limbs from slight subcutaneous œdema is also not infrequent.

The disease may be arrested at any period during its course, and may then terminate slowly in complete recovery. This is possible even after all the limbs are completely paralyzed, and after extreme wasting of muscles has taken place. As a general thing, however, recovery is incomplete. In the majority of cases the disease advances to a certain point, at which point it remains at a standstill, in some instances for a period of several years. In some of these cases further improvement is possible even after a long interval, during which the patient has remained *in statu quo*. Usually, however, the non-fatal cases terminate in the condition known as spastic paraplegia. The lower limbs gradually become more and more rigid, until finally each limb is moved as if it were one solid body. The feet cannot be flexed, the toes are shuffled across the floor, and the pelvis must be raised farther from the ground, in order to prevent the toes from catching in the floor. The limbs offer a decided resistance to passive motion. The tendon reflexes are often enormously increased. Ankle clonus often develops involuntarily if the patient, while sitting down, happens to bear his weight on the front part of the foot.

In much rarer cases the limbs become contractured in flexion, the thighs upon the abdomen, the legs on the thighs, the feet on the legs. At the same time the thighs are usually adducted strongly upon one another, so that one thigh is drawn forcibly across the other. In these cases bedsores are very apt to develop at the points at which the limbs come constantly in contact with one another. The patients very often suffer from intense pains in the contractured limbs, especially when an attempt is made to overcome the contracture. At other times the pains come on spontaneously in violent paroxysms, which are usually very difficult to control.

Death occurs generally from extension of the inflammation upward, and implication of the muscles of respiration; from gradual exhaustion, resulting usually from the formation of bedsores; or from disease of the kidneys consequent upon cystitis following paralysis of the bladder.

Bedsores develop over the sacrum, and are caused not alone by the protracted pressure on this part of the body, but also by soiling from urine and fæces, which is prevented with difficulty in many cases. In certain severe cases of inflammation of the lumbar enlargement bedsores may occur at a very early period, and then seem to be due directly to trophic changes in the skin.

With proper care and treatment the bedsores may remain confined to the integument, but not infrequently they extend more deeply, spread to the bones, and give rise to pyæmia; or, as I have also seen, the inflammation extends through the sacral foramina and sets up a rapidly fatal meningitis.

The occurrence of cystitis is also an important symptom. Perhaps in the majority of cases it results from the introduction of foul catheters, but it may also develop when such a cause cannot be discovered. The danger, in this condition, consists in the secondary development of acute interstitial nephritis, as the result of the introduction of bacteria along the ureter from the decomposing urine in the bladder. This complication usually proves rapidly fatal, but the writer has seen a few cases in which the post-mortem appearances warranted the belief that this morbid condition in the kidneys may subside. In such cases, however, death was the result of another attack of the same kind. But, on the whole, the renal affection seems to be a rare cause of death, even if the cystitis remains unchanged for a long time.

Close study of the symptoms enables us to localize the lesion very accurately in cases of transverse myelitis.

In myelitis of the lower part of the lumbar enlargement the motor paralysis is confined to the muscles supplied by the sciatics (gluteal region, posterior thigh group, and all the leg muscles, with the exception of the tibialis anticus). The sensory disturbances (complete or incomplete anæsthesia) extend over the entire lower limbs, with the exception of the parts supplied by the obturator and crural nerves (anterior and inner portion of thigh). Reflex movements in the paralyzed parts are abolished. The sphincter ani is paralyzed. When the lesion is a severe one the paralyzed muscles undergo atrophy, and the reaction of degeneration is observed.

In a lesion of the upper part of the lumbar enlargement the motor and sensory paralysis involves the lower limbs completely.

In a lesion of the dorsal cord more or less complete motor and sensory paralysis is observed in the lower limbs throughout, the anæsthesia extending upward to the intercostal nerves given off at the site of the lesion. The bladder and rectum are paralyzed. The muscles undergo very little or no atrophy, and the electrical reactions are unchanged. The cutaneous and tendon reflexes are increased on account of the abolition of the inhibitory influence from the brain.

In a complete transverse lesion of the cervical cord the paralysis affects all the limbs; when the transverse lesion does not cut across the entire cord the paralysis may be confined to the upper limbs (cervical paraplegia). The muscles of the trunk are also involved in these cases, and this process is a great source of danger to life on account of the resulting interference with respiration. In lesions of the lower part of the cervical enlargement the intercostals are paralyzed. If the lesion extends higher the diaphragm also becomes paralyzed, from implication of the nucleus of the phrenic nerves. As a general thing the muscles do not undergo atrophy, because the disease usually proves rapidly fatal. When the cilio-spinal centre is involved we may observe dilatation or contraction of the pupil, according as the centre in question is irritated or paralyzed.

Optic neuritis has also been observed in rare cases of myelitis of other parts of the cord (usually of the disseminated variety), but its relation to the spinal inflammation is not clear. The neuritis and myelitis probably are effects of the same underlying cause.

In lesions of the upper cervical cord the symptoms are similar to those just described; but in these cases the disease is apt to spread into the medulla oblongata, and to involve certain of the cerebral nerves. The following brief abstract will illustrate the history of acute myelitis of the upper cervical cord, extending into the medulla oblongata.

A. B—— contracted syphilis two months ago. At the present time he exhibits a roseola, and is suffering from slight sore throat. On November 12th he had sexual intercourse while standing. During the same night he complained of an uneasy feeling in the legs, and of slight numbness in the tips of both middle fingers and in the feet. On the next day he walked up five flights of stairs twice; then the legs began to feel heavy. He managed to get about his room on the following day (Saturday), but on Sunday morning was unable to rise from bed. At my first visit (November 16th, 6 P.M.) I found the patient in the following condition: Almost complete paralysis of the lower limbs; slight motion of the thighs; a little reflex action from the soles; numbness of the entire limbs. He could hold his water, but after it began to flow he was unable to check it. There was very marked paralysis of the upper limbs, especially the right limb, but most movements could be performed slowly; partial anæsthesia of the upper limbs and trunk, none of the face; voice a trifle hoarse; temperature, normal; respirations, 16; pulse, 84 and of good quality. The patient does not use the diaphragm in breathing, but can do so quite well voluntarily. He says that he sometimes has difficulty in breathing, and cannot cough as well as yesterday.

November 17th, 9 A.M.—Passed a bad night, unable to sleep at all; whenever he was about to doze off he had to struggle for breath; had one bad attack of dyspnœa which lasted three hours. No reflex from the soles of the feet; power absolutely lost in lower limbs; also worse in upper limbs, particularly the right; sensation more blunted; passed a large quantity of urine at 5 A.M.

Respiration, 20; pulse, 90; respirations interrupted occasionally by a long breath; more difficulty in breathing; still moves the diaphragm voluntarily, but not so vigorously as last night. Speech is more hoarse, the vowels are not enunciated very clearly. Slight anæsthesia of the left side of the face, paresis of the left internal rectus, also of the right side of the face. He has double vision over a small area a little to the left of the median line on the horizontal. All the right facial muscles are paretic; he is unable to close the right eye entirely.

November 17th, 8 P.M.—Condition unchanged, has slept at intervals, once for half an hour at a stretch.

November 18th, 10 A.M.—Left pupil slightly dilated, but reacts to light; left side of face not so numb; paresis of internal rectus improved; no double vision; enunciates vowels more distinctly. Paralysis of the limbs is a little more marked; no reflex from soles; urine passed freely. During the night had severe pain between the shoulders; respiration, 22; pulse, 86; diaphragm not so active, but respiration easy.

November 18th, 9 P.M.—Respiration, 33 and more labored; diaphragm weaker; pulse, 84 and weaker. Skin cool and covered with cold sweat; looks a little bluish; face flushed; patient seems excited and talks a good deal—says "he is off his nut because he has taken too much brandy"; pupils normal; speech more difficult, vowels less distinct; does not swallow so well. The patient died at five o'clock the following morning, after increasing difficulty in breathing.

Disseminated myelitis is almost always an infectious or toxic affection. In comparatively rare cases the lesions are confined to the cord, but in the majority of cases they are also found in the medulla, pons, and other parts of the brain. If the lesions in the cord are grouped in such a manner that the transverse section is involved in great part, the symptoms closely resemble those of transverse myelitis. In a considerable proportion of cases, however, an acute ataxia develops, frequently in all the limbs at the same time. The patellar reflexes, instead of being lost as they are in tabes dorsalis, may be exaggerated. The muscles exhibit loss of power, varying in degree from slight paresis to complete paralysis. The lightning pains of tabes are absent and the sphincters may be unaffected. Optic neuritis is not uncommon. When the medulla and pons Varolii are affected, as is generally the case, we may observe scanning speech, nystagmus, tremor, occasionally attacks of mental excitement, as in multiple sclerosis.

So-called *compression myelitis* is usually, as we have intimated in the section on etiology, not a real inflammation. But as this point can be decided only upon autopsy, and not by the clinical history, we will here give a brief description of this condition.

In the majority of cases compression myelitis is the result of Pott's disease and the lesion is situated in the dorsal region. The malady is often preceded by symptoms of irritation of the roots of the nerves by the diseased bone or the secondary pachymeningitis. These irritative symptoms consist of a cincture feeling around the abdomen or chest (according to the location of the vertebral lesion), or of pains in the legs; sometimes there is an eruption of herpes zoster upon the trunk or the lower limbs.

Weakness of the limbs generally begins slowly and finally advances, as a rule, to profound paralysis. This may be preceded for quite a while by marked exaggeration of the knee-jerk and ankle clonus. In the same way these symptoms may persist for a long time after the restoration of motor power.

After the motor paralysis has developed, the symptoms differ in no respect from those of ordinary transverse myelitis, except that there is usually a much greater discrepancy between motor and sensory paralysis, the latter being very often entirely absent. The paralysis exhibits very little tendency to extend beyond the parts originally involved. In a certain proportion of cases the lower limbs become contractured—the thighs flexed on the abdomen, the legs on the thighs—or shooting pains in the lower limbs become a prominent feature of the disease. To judge from his own experience, the writer is inclined to believe that this condition is particularly apt to develop when the myelitis is the result of irritation of the cord rather than of compression by morbid products.

When the compression myelitis is the result of cancer of the vertebræ, pains in the lower limbs and trunk are extremely distressing, and, if cutaneous sensation is abolished at the same time, the condition is known as anæsthesia dolorosa. Apart from the excessive violence of the pains, this paraplegia dolorosa cannot be distinguished from other forms of compression myelitis.

Purulent Myelitis.—Purulent inflammation of the spinal cord is much rarer than the similar process in the brain. On account of the protected location of the cord, injuries to the latter and hence surgical infection are uncommon. On the other hand, purulent myelitis has been observed after injuries to the vertebræ without direct lesion of the cord. In such cases the infectious bacteria must have been conveyed through the lymph channels. The disease occurs most frequently as a sequel to suppurative meningitis. It is also possible that the pus-producing germs are conveyed to the cord and meninges at the same time. A few cases have been reported as the result of metastases in remote parts of the body: for example, after abscess of the lung, putrid bronchitis, suppurative processes in the genito-urinary tract, etc.

The symptoms of the disease are generally those of a transverse myelitis, associated perhaps with unusually pronounced meningitic symptoms and evidences of profound general infection. As a rule, the diagnosis of the suppurative character of the inflammation is not very difficult after recognition of the etiological factors in the case.

Chronic myelitis is a much rarer affection than the acute or subacute form of the disease. The causes of chronic myelitis are similar to those of the acute form. It is generally held that the difference in the mode of beginning is the result of differences in the intensity of the etiological factors—for example, sudden and violent exposure will produce acute myelitis, long-continued and less severe exposure will produce chronic myelitis;—but it seems to me that this view is based on theory rather than on well-established clinical facts. In traumatic cases a single traumatism may set up an acute myelitis or an inflammation of the cord which does not make its appearance until long after the symptoms of the original injury have subsided. Perhaps there has been some spinal pain for a few days or weeks after the injury, then the patient feels entirely well; but, a few months later, the signs of slowly advancing myelitis make their appearance.

Syphilis seems to exert a considerable influence as a predisposing cause, but it is questionable whether it acts simply by making the spinal tissues more vulnerable or by the production of gross lesions in the vessels or membranes of the cord. It seems probable, however, that in a large proportion of such cases the former theory is correct, the syphilitic virus appearing to act as a potent predisposing factor, as it does in the production of locomotor ataxia.

Alcoholism also appears to exercise great influence in the production of chronic myelitis; but we must bear in mind that this vice is often connected with repeated exposure to cold and wet, and to violence, and that it is difficult to decide between the parts played by each of these factors.

The *symptoms* of chronic myelitis are very similar to those of the acute form, except that they develop very slowly and insidiously. In some instances this gradual course may be interrupted occasionally by more or less acute exacerbations. In the majority of cases the disease affects the dorsal region, and hence the symptoms are those of slowly developing paraplegia. As in the acute form, the sensory symptoms are generally much less marked than the motor phenomena. The patient complains of formication and tingling in the feet, or of a sense

of numbness in the soles, so that the floor does not feel natural in walking. This condition may last a very long time, although no change in sensation can be noticed objectively after the most careful examination. Pain is not a prominent feature, although there is often complaint of a slight girdle feeling around the abdomen. In a few cases, however, the pains are remarkable for their violence. Slight, dull pains are often felt in the back and limbs, particularly after walking for some length of time. These symptoms may last a long time before any motor weakness becomes apparent. The patient notices that he becomes more tired than usual after walking, and finally begins to drag the legs. Rigidity of the lower limbs is an early and almost constant accompaniment of the loss of power (spastic paraplegia). In many cases, indeed, I have noticed a marked development of this spastic condition before any real loss of power could be detected. At the same time the tendon reflexes are notably increased. In some cases disturbances of sensation, which can be determined by objective examination, may be entirely absent, and subjective sensory disturbances may also be extremely slight, or wanting. The symptoms are then exactly similar to those of so-called primary lateral sclerosis, and may remain in this stage for several years before the advent of other symptoms attests the spread of the disease.

In a very small proportion of cases, particularly in those which are due to syphilis, the lesion is confined to a small section of the cord, and, if limited to one-half, may give rise to the symptoms of spinal hemiplegia (paralysis of motion on one side, paralysis of sensation on the opposite side). As a general thing, however, the lesion is not confined exactly to one half of the cord, but extends somewhat into the other side, and motor and sensory symptoms are therefore observed in each limb, although the paralysis of motion is more marked in one limb, that of sensation in the other limb. In this form of the disease, sensory symptoms (anæsthesia, pains in the back and limbs) usually form an important feature.

As a general thing, the lesion spreads slowly along the cord in an upward or downward direction (usually the former), and other parts of the body become involved. The spread of the disease may be exceedingly slow, and even twenty or thirty years may elapse before the final termination. In other cases the chronic course may be interrupted by acute exacerbations. I have also seen several cases in which the symptoms of chronic myelitis appeared to merge later into those of posterior sclerosis or multiple spinal sclerosis.

Spastic symptoms are apt to be extremely severe in this form of myelitis. In the most aggravated cases the muscles are contractured to such an extent that the thighs may be flexed sharply on the abdomen and the legs flexed firmly against the thighs. The rigidity may be so pronounced that the entire body is moved as a whole when the force is applied to the feet. We then find not infrequently that the pressure of one knee upon the other gives rise to the formation of bedsores, and these are also apt to develop upon the sacral and gluteal regions. With proper care and nursing the bedsores may often be prevented, but the absolute helplessness of the patient always entails great suffering. It is a surprising fact, however, that the patient often exhibits a cheerful frame of mind during the entire course of the disease.

In a considerable proportion of cases the functions of the bladder and bowels are notably impaired, but it is found not infrequently, even in the most spastic cases, that the patients still retain considerable power over these organs.

When the sensory disturbances (paræsthesiæ and pains) are severe, the condition of the patient is truly pitiable. Unfortunately, he may remain in this state for many years until finally an intercurrent affection brings relief in death.

In concluding the clinical history of myelitis I will refer briefly to a mode of termination which has been unnoticed by the majority of writers. In a number of cases which have come under my observation the disease appeared to begin as a transverse myelitis; but the symptoms cleared up, in great part, and then merged into those of locomotor ataxia. In the following case I was fortunate enough to obtain a post-mortem examination:

J. D——, thirty-five years of age, married; entered Randall's Island Hospital on September 4th, 1883. The patient gives no evidence of syphilis; habits regular.

The present illness has lasted nine months; it began with ptosis of the right eye; shortly afterward he began to have shooting pains in the legs and arms. In a few weeks he had to quit work on account of the pains and weakness in the lower limbs. About five months ago the patient became so weak that he was unable to walk, and had trouble in holding his water.

Present condition (October 4th, 1883).—Patient walks with assistance, but has a characteristic heel gait, and must watch the movements of his feet; he reels to and fro when standing with the eyes closed.

Anæsthesia and analgesia are present, below Scarpa's triangle, in both thighs and legs; patellar reflexes are absent. Slight inco-ordination of upper limbs, and some numbness of the fingers. Partial ptosis of right eye; both pupils very much dilated, and react poorly to light; optic papillæ unchanged; special senses normal. The dynamometer is forced to forty by the right hand, to thirty-six by the left hand.

October 24th.—Had a pulmonary hemorrhage; dulness and a few moist râles over the upper lobe of left lung.

The motor power of the lower limbs improved considerably, but the inco-ordination rapidly grew worse, the limbs jerking in every direction when any voluntary movement was attempted. There was very marked emaciation, but the muscular power finally appeared to be normal compared with the amount of muscular tissue retained. The ptosis disappeared almost entirely; sight remained normal. The phthisis continued to advance, and the patient died January 7th, 1885, without the development of any fresh symptoms on the part of the nervous system.

Microscopic examination of the spinal cord showed exquisite sclerosis of the posterior columns (enormous increase of the neuroglia and of nuclei), together with a general increase of connective-tissue elements in the remaining portions of the cord, with the exception of the gray matter, which seemed to be perfectly normal. In addition, the blood-vessels of the posterior columns were very much dilated, and their walls were exceedingly thickened, particularly the adventitia, in which the nuclei were enormously increased. The nuclei around the central canal were increased in number, and there seemed to be, also, an increase of fibrous tissue in this locality. Here and there in the other columns of the cord were seen blood-vessels which also presented a considerable increase of nuclei.

Pathological Anatomy.—In a good many cases of acute myelitis no change in the structure of the cord is visible to the naked eye. Sometimes the diseased parts are softened, and their color is changed. The cord cuts less firmly than in the normal state, the cut surface projects as if the cord were too large for the enveloping pia mater, and the normal differentiation between the white and the gray matter becomes less distinct or is completely lost. The entire surface has a reddish tinge; sharply defined reddish streaks (distended blood-vessels) are often seen, particularly in the white matter; and here and there we occasionally notice minute red specks (capillary hemorrhages). In some cases these hemorrhages form the most marked change recognizable by the naked eye, and the cut surface has a bright red, or, at a later stage, chocolate color (hemorrhagic myelitis). The degree of softening varies between very wide limits. In some cases it is entirely absent, in others it is so pronounced that the diseased part of the cord, but particularly the gray matter, is diffluent and flows out like cream.

Schmaus and Sacki give the following description of the pathological anatomy of the various forms of myelitis:

Transverse Myelitis.—In this form of the disease we