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

may find one large focus or several smaller ones. These foci exhibit the appearance of parenchymatous degeneration with swelling, or pronounced vascular changes, or there may be softening, with or without hemorrhage.

In some places the white substance shows marked swelling of the axis cylinders and medullary sheaths; the latter may have a vesicular appearance and stain poorly with Weigert's solution. The swollen axis cylinders, on transverse section, form partly homogeneous, dark-stained bodies, or pale, finely granular bodies. Marchi's method of staining may show black coloration of the swollen fibres, indicating beginning fatty changes. Longitudinal sections show that the axis cylinders break up into smaller segments, and these pieces are often enclosed in swollen medullary substance; if the latter has undergone fatty degeneration, an appearance of granular corpuscles may be simulated. The neuroglia is also involved; the fine fibrillæ, which separate the individual nerve fibres, are swollen into thick, homogeneous bands, which often take a deeper stain; the glia cells are often swollen and enlarged. In addition, we find arterial hyperæmia, dilatation of the small arteries and veins, homogeneous or slightly granular masses of exudation in the interstitial tissue, masses of leucocytes in the walls of the vessels or free in the tissues, and more or less numerous granular corpuscles. When the gray matter is involved, the ganglion cells exhibit interesting changes. These begin with swelling of the granules capable of staining, or with a finely granular distribution of the tigroid substance. Next come changes in the cell body as a whole, viz., homogeneous swelling or degeneration of the cell and its processes, shrivelling of the cell, changes in pigmentation, vacuolar degeneration, finally complete disappearance of certain cells or groups of cells. The nuclei also exhibit signs of degeneration; they stain more deeply, grow smaller and darker, are irregular or fissured, and finally coalesce completely with the body of the cell.

In a second group of cases, the vascular changes are most prominent. The microscope shows that the chief change is a small-cell infiltration, most marked around the vessels and following them along their course. The cellular infiltration occurs chiefly in the lymph sheaths of the blood-vessels, but it also extends into surrounding parts and into the meshes of the neuroglia; sometimes even the pericellular spaces around the ganglion cells are filled with such small-cells. We also find hyperæmia and serous infiltration of the tissues, with secondary conditions of swelling. Not infrequently the evidences of degeneration of the nerve cells and fibres are found to extend diffusely over large areas, while the infiltration occurs only in scattered foci. In other cases, however, the swelling and degeneration are not alone less widespread than the cellular infiltration, but may even be confined to the foci of infiltration, while the remaining tissues are almost unchanged. Sometimes the nerve fibres and ganglion cells are well preserved, even within the zone of infiltration, and evidently are only affected at a late period of the disease. This indicates that the inflammatory process is first manifested in the walls of the vessels. More or less extensive hemorrhages not infrequently accompany the infiltrations. Granular corpuscles in the meshes of the neuroglia become more numerous the longer the process lasts. Even in cases which have lasted only a few weeks we sometimes find the walls of the blood-vessels thickened by cellular proliferations and the lumen diminished by thickening of the intima (endarteritis obliterans).

In a third group of cases the chief feature is a condition of softening, due to infiltration with granular corpuscles. The foci may have the consistence of porridge, or they may even be diffluent on section, leaving vacuolæ. The softening spots consist almost entirely of granular corpuscles, together with detritus of the nerve tissues. In sections which have been stained with Weigert's "medullary-sheath stain," the softened spots appear as brownish-yellow or colorless patches. The vessels show even more pronounced changes than in the previous group. Thrombi are found in some of the vessels, and these may be responsible for a part of the softening. There are also hemorrhagic forms of inflammatory softening; in the slightest grades capillary hemorrhages are found in the softened spots and surrounding parts, and give rise to a speckled appearance.

As a matter of course there are numerous transitions between the three forms just described. A transverse lesion is followed regularly by ascending and descending degenerations.

Acute Disseminated Myelitis.—In this form of disease the various foci exhibit a pronounced vascular character; small vessels form the centre, around which are grouped the swelling and degeneration of the tissues, as well as the infiltration with round cells and granular corpuscles. Larger foci involve several vessels, but even here we find distinct signs of vasculitis, viz., distention of the vessels with blood, small-cell infiltration of the walls of the perivascular and adventitial lymph sheaths, and deposit of granular corpuscles. In older cases there are not infrequently thickenings of the walls of the vessels, especially of the tunica intima. The infiltration with granular corpuscles may be so dense and the degeneration of the tissues so pronounced that the appearances of softening are produced; in other cases hemorrhages are present.

Purulent Myelitis.—Abscess of the cord is of rare occurrence. Infection-producers may enter the cord in three ways: after injury to the cord with direct infection from the outside, through the lymphatics, or through the blood-vessels. If the bacteria enter through the blood-vessels, they may be simply carried along by the current of blood or they may be conveyed in emboli. The latter give rise, in the nervous tissues, to suppurative softening. Apart from traumatic purulent myelitis, the disease is most frequent after suppurative spinal meningitis.

Chronic Myelitis.—Histologically the diseased parts of the cord exhibit either degeneration and sclerosis or a condition of softening, with or without degeneration and proliferation of the glia.

In the former event we find swelling and segmentation of the axis cylinders, fatty degeneration of the medullary sheaths, the formation of hyaline bodies and corpora amylacea. The disappearing parenchyma is replaced by proliferating neuroglia tissue. The blood-vessels present cellular infiltration of their walls (especially of the adventitia, but sometimes of the media and intima), thickening of the walls, adhesion and obliteration of their lymph sheaths, and often dilatation of the perivascular lymph spaces. In some cases there are no decided changes in the vascular apparatus, but the myelitic foci have peculiar relations to the vessels, inasmuch as we often find more or less changed vessels at the centre of the diseased foci, or tracts of degeneration follow the course of the vessels.

The second form of chronic myelitis is that of softening. It is difficult to decide whether the spots of softening are the result of an inflammatory process or of a simple disturbance of circulation. If the softening is owing to infectious or toxic causes, the process may continue as long as these causes are active. It must also be remembered that softening often causes disturbance of circulation in its vicinity, and that the neighboring parts are in a condition of more or less marked œdematous swelling. Hence the softening may continue even after the toxic or infectious agent has ceased action.

With regard to the secondary degenerations of the cord, which are found after myelitis, we refer to other articles in the present series.

Diagnosis.—Acute myelitis must be distinguished from meningitis. The latter affection is very much rarer than acute myelitis, apart from those cases in which it is associated with inflammation of the cerebral pia matter. In meningitis the pains in the back and limbs, the cramps in the muscles, and the hyperæsthesia, are much more marked than in myelitis, and usually persist for a much longer time. Paralytic symptoms are not pronounced, and the bladder and rectum, as a rule, are unaffected. In myelitis, on the other hand, the initial irritative symptoms are often entirely absent, or, in the very large majority of cases, soon give place to anæsthesia and motor paralysis. With the exception of certain cases of inflammation of the cervical cord, fever is usually absent or slight in myelitis; in meningitis there may be decided febrile movement.

In two cases I have seen acute myelitis mistaken for acute articular rheumatism—once in a child suffering from compression-myelitis dependent upon Pott's disease, another time in an adult with extremely acute dorsal myelitis. In both patients the joints of the lower limbs were very tender on pressure, and in the adult there was high fever. In the former, however, examination of the spine revealed a well-marked kyphosis, the loss of power was disproportionate to the apparent severity of the pain, and the tendon reflexes were greatly exaggerated. In the latter the patient also suffered from rapid pulmonary phthisis, which explained the high fever; the pains were not confined to the joints, but were also marked in the muscles, and the lower limbs were anæsthetic (anæsthesia dolorosa).

Hysteria is sometimes mistaken for myelitis, especially when it occurs in young females; or, *vice versa*, myelitis is sometimes, though less frequently, regarded as hysterical paraplegia. The occurrence of other undoubted evidences of hysteria is an important point in diagnosis; but too much stress should not be laid upon this feature, since it must be remembered that hysterical individuals are also liable to be attacked by myelitis. The etiology should also be taken into consideration, and, in the absence of any of the causes previously mentioned, the diagnosis of myelitis in a young female should be looked upon with suspicion. In hysterical cases, however, the evidences of serious organic lesions of the cord are always wanting. There is no pronounced wasting of muscles, no change in their electrical excitability, no loss of power of the bladder and rectum. On careful observation, moreover, it is often found that the apparent paralysis varies in different positions of the body, and that there are sudden changes in motility which remain inexplicable on the theory of an organic disease of the cord.

The diagnosis from hemorrhage of the cord is usually easy, except in cases of hemorrhagic or apoplectiform myelitis, when the symptoms have come on with extreme rapidity. Hemorrhage into the spinal cord is extremely rare, except as the result of injury. The symptoms of motor paralysis attain their utmost severity at once, and then, unless the hemorrhage is situated high in the cervical region, begin to improve slowly up to a certain point. With the exception of the suddenness of the onset, and the previous occurrence of traumatism, there is really no means of differentiating spinal hemorrhage from acute myelitis. In the majority of cases, indeed, the hemorrhage is followed by myelitis, either as the result of the injury to the elements of the cord from the traumatism, or as the outcome of the irritation excited by the clot.

The diagnosis of acute disseminated myelitis is extremely difficult and often impossible. Great importance attaches to the etiology of the disease, as almost all the reported cases have followed traumatism or an infectious or toxic condition. When the process results in paraplegia a probable diagnosis can be made if ocular symptoms are marked, or if there is an early development of bulbar or cerebral symptoms (scanning speech, tremor, hallucinations, mental obtuseness, etc.). When the disease is manifested by an acute ataxia, an important diagnostic feature is the preservation or even exaggeration of the tendon reflexes. Like the paraplegic variety, the ataxic form is also attended by bulbar and cerebral symptoms.

In the majority of cases of compression myelitis the primary lesion is caries of the vertebræ, and, as a general thing, the deformity of the spine is so pronounced that there can be very little doubt with regard to the diagnosis. In Pott's paraplegia, moreover, the exaggeration of the cutaneous and tendon reflexes may precede other symptoms for a considerable period, and may persist after other evidences of myelitis have disappeared. In addition, the sensory symptoms are usually much less pronounced than those in the motor sphere, and may even be entirely absent, despite complete paralysis of the limbs. In a few cases, however, particularly when the patient has been confined to bed on account of some other disease, spinal deformity may not be noticed, and the myelitis is then apt to be regarded as primary.

When compression myelitis is secondary to cancer of the vertebræ, the recognition of the nature of the primary disease usually depends on the presence of a primary cancer in some other part of the body, on the violence of the pains in the paralyzed parts (especially if associated with anæsthesia), and on the presence of a deformity of the spine which seems to be due to an irregular enlargement of the vertebral arches or lateral masses. It must not be forgotten, however, that cancer of other parts of the body may be associated with ordinary vertebral caries. In a case of this kind recently under my observation, I made the diagnosis of compression myelitis from vertebral cancer, but the autopsy proved that the disease had been an ordinary Pott's paraplegia.

Diagnosis.—Railway spine, when due to chronic myelitis or meningomyelitis, may prove a source of great difficulty in diagnosis, especially because the element of malingering so often comes into play in this condition. When this factor can be excluded, the diagnosis is based on the slow development, at a variable period after the injury, of symptoms which point to disseminated focal lesions of the cord, viz., irregularly distributed paralyses, various paræsthesiæ, pain in the spine and limbs, disturbances of micturition, localized atrophies of muscles. A depressed and irritable condition of the mental faculties, with loss of memory, is quite common in this condition.

The diagnosis of chronic compression-myelitis is made in the same way as that of the acute form, except that the symptoms develop much more slowly. Acute exacerbations are particularly apt to develop in this form of the disease.

Chronic myelitis can be distinguished from the persistent paraplegia left over after an acute attack only by the history of the onset of the illness. When, for any reason, the patient is unable to furnish the previous history, the diagnosis is impossible.

Tumors of the spinal cord or its membranes are rare, and their differential diagnosis from chronic myelitis is extremely difficult. The clinical history of the former consists essentially of symptoms of irritation of the nerve roots, followed by those of compression of the cord. In some cases, however, the latter precede the former. The compression of the nerve roots sometimes lasts a long time before the evidences of compression of the cord make their appearance. Probably the most important point in the differential diagnosis is the great variation which is sometimes noticed in the severity of the symptoms. In addition, it is found not infrequently that the first evidences of compression of the cord occur in the form of spinal hemiplegia, owing to the fact that the tumor often develops first in one lateral half of the cord.

Chronic myelitis is not apt to be mistaken for hysterical paraplegia, because the latter disease begins much more abruptly than the former.

The diagnosis of the localization of the inflammation in the cord depends upon the grouping of symptoms, for which we refer the reader to the section on clinical history.

Prognosis.—Complete recovery from acute myelitis, with the exception of the compression-myelitis of Pott's disease, is a comparatively rare result. Incomplete recovery is by far the most frequent termination, although a fatal issue is by no means uncommon. Other things being equal, the disease is more apt to end fatally the higher the location of the lesion in the cord. If the centre for the phrenic nerve has been attacked, as shown by paralysis of the diaphragm, recovery is extremely rare. The occurrence of bedsores is likewise of grave import, particularly if they develop at an early period.

When they occur after the disease has lasted a long time, they often heal quite rapidly under suitable treatment. Mild cystitis is so common in myelitis that it can hardly be regarded as aggravating the prognosis; but in severe cases, especially if the bladder is completely paralyzed, there is always danger of the production of acute interstitial nephritis. The latter affection is almost inevitably fatal at an early period, though in some cases it seems to pass into a stage of quiescence.

The prognosis as regards recovery is also so much less favorable the greater the degree of implication of the gray matter, as evidenced by rapid atrophy of the paralyzed muscles and by the presence of the reaction of degeneration in these parts. But it must also be remembered that even the most marked atrophy of the muscles and complete abolition of their electrical irritability do not entirely preclude the possibility of a very satisfactory degree of recovery. I have had under observation a patient in whom the limbs were completely paralyzed and wasted to the extreme, and the reaction of degeneration was present except in those muscles in which no reaction could be obtained (even the intercostals were partly paralyzed); and yet this patient, at the end of a year and a half, was able to walk about and to perform her duties in such a satisfactory manner that she requested her discharge from the hospital.

The occurrence of a spastic condition of the lower limbs constitutes an almost insuperable obstacle to any further improvement. The degree of motor power which is acquired in these cases, however, is usually quite considerable, and the patients are often able to move about quite freely, unless the rigidity of the limbs is excessive.

Pott's paraplegia generally offers a favorable prognosis, unless the vertebral caries itself, or its sequelæ, constitute a serious menace to life. Improvement in apparently hopeless cases sometimes comes on quite suddenly, and the patient may recover even from a number of severe attacks. In these cases there is always danger of a relapse so long as the tendon reflexes remain exaggerated.

The prognosis of chronic myelitis is always grave. Even chronic compression myelitis, which, of the different forms of the disease, presents the most favorable outlook, rarely terminates in recovery. Unless the slow course of the spinal inflammation is interrupted by acute exacerbations, the patient may linger for years. The longest lease of life is enjoyed in those cases of transverse myelitis in which the disease does not extend up the cord. The greatest danger in these cases arises from the development of cystitis and secondary interstitial nephritis.

In the syphilitic forms of chronic myelitis we are sometimes able to secure a fair degree of recovery, especially if treatment is begun before the disease has extended across the entire section of the cord. But even here it will be found that complete recovery rarely takes place.

It is held by many writers that cases of railway spine, which is probably due to chronic myelitis and meningomyelitis, usually recover; but this statement does not agree with the experience of the majority of unbiassed observers.

Treatment.—The most important indication is to secure as absolute rest in bed as is possible under the circumstances. As soon as any symptoms which suggest the onset of myelitis make their appearance, the patient should take to bed and remain in the recumbent position. The wants of nature should be attended to with the aid of the bedpan, in order to avoid, as much as possible, movement during the acts of urination and defecation. Rest is, in my opinion, by far the most important remedial agent at our command. It is often difficult to determine at what period it is best to allow the patient to begin to exercise the paralyzed limbs, but it is always better to err on the safe side. I have seen very marked improvement in a number of apparently hopeless cases in which this plan had been faithfully carried out for a period of a year to a year and a half.

Contracture of the tendo Achillis is a very common sequel of all forms of myelitis, and it has been suggested that the pressure of the bedclothes upon the feet may exercise some influence in the production of this unfortunate symptom. In order to obviate this, the bedclothes may be suspended on a barrel hoop placed across the lower part of the bed. In a considerable number of cases, however, the contracture will develop despite this precaution.

Next to rest, cleanliness is all-important, because the neglect of cleanliness is very apt to result in the production of bedsores. This usually necessitates the most careful nursing. The external genitalia and gluteal regions must be frequently washed, and thoroughly but gently dried. At the slightest indication of the development of a bedsore, an air-ring should be placed under the buttocks, care being taken that the joints of the ring are not sharp. If the evidences of dermatitis are not relieved speedily in this way, the patient should be placed on a water-bed.

The bladder must be carefully looked after. Catheterization must be resorted to at once, as soon as the bladder is no longer thoroughly emptied. The greatest precautions must be taken to keep the catheter thoroughly aseptic in order to prevent the introduction of germs. Even a soft-rubber catheter must be introduced very gently, because the mucous membrane of the bladder is very apt to be congested, and bleeds readily under such conditions.

Counter-irritation is useful in very many cases. During the first stages of an acute myelitis the application of dry cups along the spinal column may be attended with some benefit. Vigorous counter-irritation with fly blisters is indicated at a later period, but these do not promise much improvement after the disease has become stationary. In this connection I may mention that in a case of chronic myelitis in which all hope of recovery had long been abandoned, I noticed that a remarkable degree of permanent improvement followed the development of a large carbuncle on the back.

The actual cautery is also recommended. I have never seen any benefit from its use, except in rare cases of compression-myelitis. In such cases, however, it sometimes acts with remarkable rapidity.

Among other non-medicinal agents may be mentioned baths and electricity.

As a rule, the patient cannot tolerate extremes of temperature. I usually recommend a sitz bath at a temperature of 100° to 110° F., in which the patient remains for from fifteen minutes to half an hour. If spastic phenomena are well marked, the bath may be as hot as can be borne by the patient. When the spastic phenomena are not prominent, the patient may receive a full bath at a somewhat lower temperature. It is well in such cases to follow the bath with rubbing and massage.

Electricity, when faithfully and persistently applied, is often productive of excellent results. It is employed either as a spinal current or directly to the paralyzed or contractured parts. When applied to the spine one large electrode should be placed over the site of disease, the other on some indifferent part of the body or spine, and they should be held constantly in position for from three to five minutes. The direction of the current seems to be immaterial. Much better results are obtained from applying the current directly to the paralyzed parts. In the majority of cases the galvanic current is preferable, but the faradic current may be used when the muscular contractility remains normal. The current should be merely strong enough to produce visible contraction of the paralyzed parts. The constant stabile galvanic current is used in the treatment of contractures, the electrodes being applied directly over the nerves supplying the contracted muscles. At the same time the interrupted galvanic or faradic current may be applied to the antagonists. In my own experience very little, if any, benefit has been derived from spinal galvanization; but, on the other hand, I have obtained most excellent results from the patient, persistent use of local electrization.

This even holds true of cases of marked contractures of the lower limbs, but a good deal will then be gained by the electrical treatment combined with tenotomy of the contractured parts.

Medicinal treatment does not play a great part in the management of this disease. Ergot has been highly recommended in the acute stages, and hypodermic injections of ergotine have been employed at the onset of hemorrhagic myelitis. I have never seen the slightest advantage from its use, and have abandoned it altogether. Iodide of potassium has also been recommended very highly, and Dr. Gibney speaks in the most favorable terms of its administration in large doses (even ʒ i.–ij., t.i.d., in children), in the compression myelitis of Pott's disease. It is very difficult to form an opinion upon the value of this agent in myelitis, though it has seemed to me to exercise a certain amount of beneficial effect. But I have employed it so faithfully, and with such little effect, in Pott's paraplegia, that I am compelled to deny any special influence in this form of the disease. Weir Mitchell has recommended suspension in this disease, but further experience has not shown that this measure possesses much value.

Strychnine is the only medicinal agent which has seemed in my hands to have a decided influence on the course of the disease. This drug may be administered as soon as the acute symptoms have ceased to advance, and it is by no means contraindicated by the occurrence of spastic symptoms. The dose may be raised gradually from gr. $\frac{1}{48}$ to gr. $\frac{1}{32}$, t.i.d., and may be given continuously for months.

So long as any improvement is observed, the patient should be kept as quiet as possible. Later, gentle exercise may be allowed, but the patient must not be permitted to tire himself. Sexual intercourse is particularly to be avoided. Even after the condition of the patient has been stationary for a long time, benefit is sometimes derived from another course of treatment with electricity, counter-irritation, and strychnine.

Leopold Putzel.

SPINAL-CORD DISEASES: POLIOMYELITIS ANTERIOR, ACUTE AND SUBACUTE.—Under this name we recognize a form of paralysis which occurs, as the name implies, most frequently in young children, especially during the period of first dentition. It is by far the most common form of paralysis to which children are subject and presents a well-marked clinical picture. A healthy child is suddenly found to have lost the use of one or more extremities, or of some individual muscle of an extremity. This loss of power may or may not have been preceded or accompanied by febrile disturbance; occasionally convulsions have ushered in the paralysis, but never is the latter accompanied by loss of sensibility. Any of the voluntary muscles of the limbs or trunk may be affected, but the functions of the bladder and rectum are left undisturbed. Such is a general description of the onset of the disease. Soon, however, many of the affected muscles begin to recover their power; this recovery may be almost complete, the paralysis being finally limited to a few individual muscles; or, on the other hand, the greater part of the muscles originally affected may remain paralyzed, and later on undergo atrophy and degeneration. In this atrophy and retardation of growth the bones take part, giving rise to shortening of the affected extremity, while other deformities occur as a result of the unopposed action of the healthy antagonists of the disabled muscles.

Synonyms are numerous, and owe their variety to the views held by different observers as to the nature of the disease, before its pathology had been established. They are: Infantile spinal paralysis; Spinale Kinderlähmung (Jacob von Heine); Paralysie spinale; Paralysis infantilis spinalis; Infantile paralysis; Kinderlähmung; Paralysie infantile; Paralysie des petits enfants; Essential idiopathic or functional paralysis of children; Paralysie essentielle de l'enfance (Rilliet and Barthez); Essentielle Kinderlähmung; Dental paralysis (Underwood, 1784); Poliomyelitis anterior acuta; Acute inflammation of the anterior gray horns of the spinal cord (Kussmaul); Tephromyélite antérieure aiguë (Charcot); Acute atrophic spinal paralysis; Paralysie atrophique graisseuse de l'enfance (Duchenne); Regressive paralysis (Barlow); Myelitis of the anterior horns (E. C. Seguin). These various terms were intended by their originators to indicate what appeared to be prominent clinical or pathological characteristics of the disease. Without entering upon the question as to the relative value of a clinical, as compared with a pathological, designation of diseases, there is no doubt that, of all the terms applied to the affection, the best of the first class is that adopted by Heine (Spinale Kinderlähmung, or infantile spinal paralysis). It is true that here even the word spinal would indicate, in a general way, the anatomical location of the lesion which is the cause of the clinical manifestations; but, inasmuch as it serves to distinguish this form of paralysis from other paralyses of cerebral origin, we can pardon this slight offence against unity in terminology in consideration of the completeness with which it serves to distinguish the disease. Of the purely pathological terms we should prefer poliomyelitis anterior acuta, as indicating the seat of the lesion, the term acute being especially applicable to the disease as it occurs in children. Seguin's myelitis of the anterior horns purposely omits the term "acute," in order to include the subacute and chronic cases of the disease which sometimes occur in adults. The designation essential or idiopathic paralysis can certainly no longer be applied to a condition which has so well defined a pathology as poliomyelitis anterior; and it seems inexcusable that so excellent a pathologist as Niemeyer should have retained this inaccurate term in the seventh edition of his work, published in 1867, at a time when the gross lesion had already been demonstrated.

Historical Account.—This disease has in all probability affected children for hundreds of years, without being differentiated by medical writers from other forms of paralysis. As early as 1784, however, Michael Underwood[1] described a form of paraplegia which occurred in teething children, and especially in those suffering from bowel troubles, which in its clinical history resembles the disease now known as infantile spinal paralysis. In 1836 John Badham,[2] of London, described a similar affection occurring in children, which he considered cerebral in origin. Notwithstanding these publications, the condition did not receive general clinical recognition until, in the publication of J. von Heine's[3] great monograph, entitled "Beobachtungen über Lähmungszustände der unteren Extremitäten und deren Behandlung," 1840, the symptom group of the affection was clearly defined and this form of paralysis distinguished from other forms of paralysis occurring in children. The picture of the affection in question, which Heine gives us in the very first edition of his work, is so accurate in detail that later clinicians had but little to add to the symptomatology of the disease. But, although no objective or subjective manifestation of the disease escaped the notice of this accurate observer, yet he remained ignorant as to the true pathogenesis of the affection. So logical a mind, however, could not but suspect that the disease was the result of an actual lesion of the nervous system, and accordingly we find him indicating the spinal cord as the most probable seat of the lesion. In the second revised edition of his work[4] he terms the disease Spinale Kinderlähmung, or infantile spinal paralysis. Duchenne, de Boulogne,[5] in 1855, besides accurately describing the affection, took a great stride in the direction of precision in diagnosis as to the character and extent of the paralysis, by discovering the relation which the faradic current bears to muscles which are the seat of this paralysis. Here, then, the first stage in the history of the disease may be said to have been completed. But, although clinically the affection was established upon a firm basis, its pathology was yet unknown. Rilliet[6] and Barthez,[7] basing their statement upon negative findings in cases of this disease upon the autopsy table, considered the affec-