by which it manifests itself. The former is sought in the cutting off of the parts from their trophic centres, and with regard to the latter it is generally supposed that the gradual developing rigidity of the muscles, the increase of the reflexes, and the later contractures depend on this degeneration. The cases, however, in which at the autopsy an extensive degeneration was found, while during life not a trace of such symptoms was present, do not speak much in favor of this view.

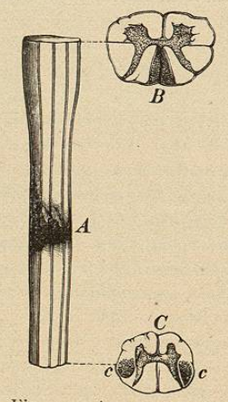

Fig. 143.—Ascending and Descending Degeneration in the Spinal Cord. *A*, primary area of degeneration (lesion). *B*, degeneration of Goll's columns (ascending). *C*, degeneration of the crossed pyramidal tract (descending). (After Gowers.)

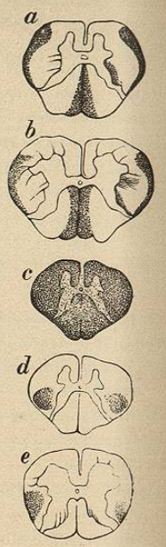

Fig. 144.—Secondary Ascending and Descending Degeneration in a Transverse Affection of the Upper Dorsal Cord. The ascending degeneration occurring in Goll's columns and the direct cerebellar tracts, the descending degeneration in the crossed pyramidal tracts. (After Strümpell.)

Lesions of the whole transverse section of the cord also produce secondary degeneration, which, however, extends not only downward (in the pyramidal tracts), but also upward—(1) in the inner segment of the posterior columns (Goll, cf. Fig. 143), and (2) in the direct cerebellar tracts (Flechsig), which are in connection with Clarke's columns (cf. Fig. 144). While this ascending degeneration is physiologically extremely interesting, as it indicates that the trophic centres of these two tracts must be situated more peripherally (as, for instance, in Clarke's columns), we are not as yet able to attribute any clinical importance to it.

III. LESIONS OF THE GRAY AND WHITE MATTER OF THE SPINAL CORD.

Charcot and Joffroy were the first to show that the large ganglionic cells of the gray anterior horns and the pyramidal tracts can be affected simultaneously by a disease which produces characteristic clinical symptoms, but it was not until Flechsig announced his discovery of the system of conducting fibres that these clinical observations became fully understood. Now we know that the disease which the French authors, following Charcot, have termed *sclérose latérale-amyotrophique*—amyotrophic (more properly myo-atrophic) lateral sclerosis—consists of a lesion of the cortico-muscular tract, which begins as a degenerative atrophy in the lumbar cord, and which, as Charcot and Marie, and more recently Rott and Mouratoff (Moscow, 1890), have pointed out, can be traced as far as the motor nerve cells of the central convolutions. Attention has already been called to the fact that, just as the nerve cells of the anterior horns, in the same way the motor nuclei of the medulla oblongata may be implicated, and thus the clinical picture of progressive bulbar paralysis develop. The two diseases are therefore analogous, and akin to them is a third—namely, the progressive spinal muscular atrophy—in which affection also the large nerve cells are diseased, as we have already pointed out above. From the nerve cells the atrophy spreads toward the periphery to the anterior nerve roots and the muscles supplied by them.

That the clinical manifestations are strictly motor and trophic, and that no sensory changes can occur, we can well understand from the anatomical distribution of the lesion. The patients at first complain of weakness in the arms and the hands, which soon interferes with their occupation. This loss of strength increases fairly rapidly, and the atrophy in the muscles of the hand—the thenar, the antithenar, and interossei—becomes more and more apparent.

The muscles of the arms also waste, more especially those of the extensor side, and the former roundness of the shoulder is soon lost owing to the atrophy of the deltoid. The triceps and other muscles also then take part in the lesion, and the helplessness of the patient, who has but little use of his upper extremities, rapidly increases. At the same time the tendon reflexes are increased, and tapping of the bones of the forearm elicits lively contractions of the muscles ("periosteal reflex").

That the so-called "jaw-jerk," which has been described by De Wattewille, is characteristic of the disease I am very much inclined to doubt, since in a number of perfectly healthy persons I found it in some present, in some absent. It certainly

does not possess any diagnostic value. This jerk may be produced by pressing down the lower jaw by means of a broad paper-cutter and tapping the latter with a percussion hammer near the teeth. The lower jaw will then respond with a contraction of the muscles of mastication.

In a relatively short time the paralysis of the upper extremities becomes so complete that not even the slightest motion is possible, and gradually contractures develop (by preference in the wrist and elbow joint). In the lower extremities the same changes may be noted, but they make their appearance later and do not reach such a high degree. Here, too, we have first weakness, difficulty in walking, and general awkwardness in making movements, then rigidity and stiffness of the muscles, enormously increased patellar reflexes and ankle clonus, later on total immobility and contractures in hip, knee, and ankle joints.

A case in one of my wards, a woman thirty-four years of age, has been for two years without power of motion, and is so entirely deprived of the use of her four extremities that without assistance she is unable to make even the slightest motion with either fingers, hands, arms, toes, feet, or legs. The disease goes on to invade the motor nuclei of the medulla oblongata, and hence is produced difficulty in swallowing, which ultimately amounts to a total inability to get food down, and the patient dies of starvation. At other times a disturbance of the respiratory apparatus may bring about a fatal issue. It is exceptional that the whole course of the disease comprises a period of more than two or three years. The diagnosis is not always easy, though it is not difficult to differentiate the disease from progressive muscular atrophy if its duration and the condition of the reflexes are borne in mind. But it is not always possible to decide between this and hysterical conditions—for example, the hysterical amyotrophia—as Charcot showed shortly before his death (Arch. de Neurologie, 1893, xxv, 74). Of the cause of the disease, as well as of effectual means wherewith to combat it, we are equally ignorant.

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While the diseases of the cord which we have studied so far were confined to certain systems of fibres—in other words, were "system diseases"—the affection now to be considered does not present this peculiarity, but the process which affects the gray as well as the white matter is more or less widely extended over the cross-section of the cord, forming a small number of large or numerous small foci. In other words, the disease is what we call "asystemic" or diffuse. It is an inflammation of the cord, which according to its course is called an acute or chronic myelitis, and to which the name transverse myelitis has also been given.

Pathological Anatomy.—Anatomical changes may in such cases be scarcely demonstrable even though the severest paralytic symptoms may have existed during life. This is more especially true in cases of spinal paralysis due to pressure, occurring in consequence of disease of the vertebræ. Here we must assume that even moderate pressure is capable of bringing about a break in conduction without any destruction of nerve elements. Usually in cases where changes can be demonstrated we find a diminution in the size and an atrophy of the nerve fibres. The axis cylinders may appear swollen and may have lost their myeline sheath. The nerve cells, which are not affected until later, become shrunken and lose their processes. According to Friedmann, the degeneration begins in a circumscribed portion of the cell, secondarily the nucleus and the processes degenerate, and finally the whole cell shrinks or disintegrates (Neurol. Centralbl., 1891, 7; cf. also Fürstner and Knoblauch, Arch. f. Psych., 1891, xxiii, 1). While thus the nerve tissue undergoes disintegration the supporting tissue increases, the meshes of the neuroglia become

broader, and in it are seen the cells of the supporting tissue first described by Deiters, which, owing to their numerous processes, have also been called spider cells. In the meshes of the neuroglia reticulum compound granular corpuscles are found which have taken up the fat and disintegrated nerve substance. These are leucocytes, and in turn undergo, sooner or later, destruction. The vessels are dilated and changes are seen in their walls, consisting of thickening or hyaline degeneration. In cases where this process has run its course in a comparatively short time the cord is found at the autopsy to be soft and of a grayish-red color, whereas if the process has been slow the cord appears, in consequence of the increase of the supporting tissue, hardened, or, as we say, "sclerosed."

Macroscopically, little is to be seen. At the most some portions may, when the cord is put into Müller's fluid for the purpose of hardening it, look light yellow, while others are dark green. The former are the diseased parts, which can not become stained because the myeline sheaths, which are turned green by chromium, are absent. With this exception all information about the pathological changes must be derived from the microscopical examination of fresh as well as of hardened sections.

According to the location of the process we distinguish a dorsal myelitis, the most common; a lumbar myelitis, the rarest; and a cervical myelitis, a relatively frequent form. In the first and second the upper extremities are entirely intact, while they are implicated if the process is situated in the cervical cord.

Symptoms.—It is very natural that the clinical manifestations of myelitis should, on the whole, very much resemble those which we have learned to recognize in the "system-diseases," and, as a matter of fact, almost all that will be described has already been said. Here, as there, we have to do with motor, sensory, and trophic disturbances, with changes in the reflexes and symptoms referable to the bladder and rectum. The motor disturbances may consist of symptoms of paralysis and irritation. The former are usually the more prominent of the two, and weakness of the legs, which sooner or later amounts to complete palsy, is one of the chief symptoms of a myelitis. As a rule, both legs are about equally affected—paraplegia; sometimes one retains its strength longer than the other, according to the extent to which the pyramidal tracts

are diseased. If not the legs but the arms are paralyzed, the lesion is situated in the cervical cord. The symptoms of irritation consist of twitchings, which occur sometimes spontaneously, sometimes as the result of slight stimulation of the skin. In many instances the removal of the bedclothes and the change of temperature resulting therefrom are sufficient to cause quite protracted clonic spasms of one or both legs. This and similar phenomena seem to be of reflex origin.

The sensory changes are less regularly met with and are of less importance than the motor disturbances. There are indeed cases where they are almost entirely absent, or where they at least do not annoy the patient or do not become marked until relatively late in the course of the disease. They consist mostly of paræsthesias, numbness, formication, also of decrease in sensibility, which may amount to a complete anæsthesia, varying in extent and situation. Actual pains, which are sufficient from their duration and intensity to cause much suffering to the patient, and which are so commonly seen, as we shall learn, in tabes, belong in this disease to the exceptions. In fact, we may say that they are usually absent, or, at any rate, not at all severe. If we are able to detect sensory changes on the trunk itself, the level up to which these extend gives us valuable indications as to the seat of the myelitis. If it is in the lumbar cord, sensibility is intact above the navel; if in the lower dorsal, above the middle of the sternum. Sensory changes in the neck and upper extremities indicate the seat to be in the cervical cord. The more prominent the sensory disturbances and the pains, the greater is the extent to which the gray matter of the posterior horns and the posterior columns participates in the inflammation or degeneration.

Trophic disturbances appear when the trophic centres—that is, the ganglia of the anterior gray horns—are diseased. Thus, if we are able to demonstrate atrophy, with reaction of degeneration in the legs, this denotes a lesion of the gray anterior horns in the lumbar cord, while the same condition in the arms indicates a disease of the anterior horns in the cervical cord. The electrical examination should never be omitted in such cases, because it may happen that the legs present a certain degree of atrophy without the presence of any reaction of degeneration. This atrophy is, then, purely the result of disuse—the atrophy of inactivity. Other trophic disturbances or vaso-motor changes in the skin are not the rule. Herpes

and urticarial eruptions, slight œdema and changes in the sweat secretion occur, but possess neither diagnostic nor prognostic value.

One symptom remains still to be mentioned, because it is rarely wanting, but rather plays an important *rôle* in myelitis, and causes endless annoyance and discomfort to the patient—namely, the bed-sores which occur in the sacral region, and become the more extensive the less the care exercised in the nursing and for the cleanliness of the patient. This is one of the most important trophic disturbances, and one which, even with the most careful attention, can not in all cases be avoided.

The condition of the skin as well as the tendon reflexes depends (1) on the state of the reflex arc in the spinal cord, (2) on the state of the fibres coming from the brain, which have probably an inhibitory function. If the reflex arc is normal, but the conduction of the inhibitory fibres interrupted, then the corresponding reflex is increased, while if the reflex arc is diseased the reflex is lost, no matter whether the conduction of the inhibitory impulses be intact or not. This holds for the skin as well as tendon reflexes. Therefore in cases of lumbar myelitis not only the skin but also the tendon reflexes are diminished or lost in the lower extremities. Those concerned are the patellar reflex, the reflex arc of which corresponds to the cord between the second and fourth lumbar nerves; the tendo-Achillis reflex, the arc of which corresponds to the first sacral nerve; the cremasteric and abdominal reflexes which have their arc at the level of exit of the first lumbar and a portion of the cord between the fourth and seventh dorsal nerves respectively. On the other hand, in a dorsal or cervical myelitis a marked increase of the tendon and skin reflexes of the lower extremities takes place, because the (supposed) inhibitory influences are cut off.

A symptom which, perhaps, causes the patient himself more annoyance than any other is the disturbance in the functions of the bladder, which in a myelitis is hardly ever totally absent. At first there is some difficulty in micturition, which may end in complete retention, so that the patient can not void his urine, but requires to be catheterized. In the later stages of the disease, however, the urine is passed involuntarily, there being either a constant dribbling (incontinentia urinæ) or from time to time an involuntary evacuation of the bladder. In

either case the patients can not dispense with a portable urinal. Occasionally there is a painful burning sensation when the urine is passed (ischuria) so that the patient dreads every evacuation of the bladder. As might be expected, cystitis frequently develops in these cases, partly owing to the length of time that the urine remains in the bladder, partly owing to the frequent use of the catheter. The rectal symptoms consist either of a most obstinate constipation, or, if the sphincter ani becomes paralyzed, of incontinence of fæces (incontinentia alvi), which aggravates to a very serious extent any bed-sore that may be present. For the localization of the myelitic process neither bladder nor rectal symptoms can be used. They are always present at whatever level the lesion may be.

Ætiology.—Of the ætiology of myelitis little is known. It seems justifiable, however, to divide the causes into those which act chemically and those which act mechanically, the former being either of an infectious or of a toxic nature. That infectious diseases may produce myelitis is shown by the fact that it occurs occasionally after diphtheria and gonorrhœa (Leyden, cf. lit.), more frequently after small-pox, and also during the course of syphilis, and that the influence of poisons may at least favor the development of myelitis has been upheld since the action of arsenic, of mercury, and of lead, and the symptomatology of the resulting intoxications have been more accurately studied. Leyden has recently published studies upon the relation between grave anæmias and some forms of chronic myelitis; Eisenlohr, upon the connection of primary atrophy of the mucous membrane of the stomach and intestines and myelitis.

Among the mechanical causes the most important is pressure, which can be exerted upon the cord by structures surrounding it, as happens, for instance, in spinal meningitis and meningeal tumors. Of greater importance in this connection is the chronic caries of the vertebræ (malum Pottii), spondylarthrocace, the tubercular spondylitis, and carcinoma of the vertebræ (cf. Figs. 145 and 146), in which either the dislocated (diseased) vertebræ themselves or the caseous and inflammatory products which are found between the dura and the bone may exert a compressing influence. That there are still other causes which may give rise to myelitis we do not deny; we would only mention bodily fatigue and exposure to cold, but these are infinitely rarer. On the other hand, there exists not