A contribution to the study of myelitis / by S.G. Webber.

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TO

THE STUDY OF MYELITIS.

BY

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A CONTRIBUTION TO THE STUDY OF MYELITIS.

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Dr. Seguin, in his paper on Spinal Paralysis, referring to the lesion found in Gombault's case, says, "We can not avoid concluding that the spinal paralysis of adults, in any one of its forms, depends upon degeneration of anterior ganglion cells. Whether this degeneration of the ganglion cells is the *only* lesion in spinal paralysis, is a question to be solved only by future autopsies."

The following case proves, I think, that other parts than the anterior ganglion cells are affected.

I was asked, May 9th, 1874, by Dr. Tarbell, of Boston, to see a patient in consultation. I found a young man, C. A. G., 24 years old, who gave an account of his condition as follows: In March, 1873, while at work at his trade as a mason, he fell about fifteen or twenty feet, striking his hip on an iron kettle, and cutting the right side of his forehead. He was laid up for a few days, but was soon out again, and thought nothing of the accident. He did not remember having any pain or unusual sensation till July. He then noticed a weakness in his left knee, and soon the left leg gave out altogether, and he went on crutches. After some time, the right thumb became weak, then the right leg, then both hands and arms. At no time had he pain or any unusual sensation, excepting, the evening of my visit, a headache. The only symptoms acknowledged were increasing weakness and wasting, the wasting not appearing till

after the weakness was noticed. At first there was much fibrillary trembling in the muscles of the arms and legs, but that had ceased and could not be excited. Micturition was rather difficult and the bowels were costive.

When seen, there was no voluntary control over the left leg, and only a slight motion was possible in the right thigh. Both arms were enfeebled, the left the more so. There was wasting of the thenar eminence and interossei in both hands, and of nearly all the muscles of the legs. There was no reflex action on tickling the soles of the feet, nor on forcibly flexing the feet, but the latter caused pain from stretching the tendons. Sensation remained very acute in the hands, and quite so in the legs, though it was not tested so carefully in the latter as in the hands. He had a nasal tone to his voice, and had a little trouble in swallowing; there was no choking nor regurgitation through the nose. He had also attacks of dyspnœa. The eyes and facial muscles were perfectly normal. There was not, and never had been, pain on motion, and none on twisting his body; and there was no spontaneous pain. There was no tenderness of the spine, no pain from the application of hot or of cold water—ice was not tried.

I did not see him again until nearly a month later, when, Dr. Tarbell being away, I was called. June 7th, it is reported that he had been growing weaker, and two or three days previously he had a pain in his left side. The dyspnœa had gradually increased in severity without any violent attacks of suffocation. Both hands were weaker. He had "nervous spells" every now and then, and had to be raised up; he turned red in the face, had a pricking, burning sensation in the chest, which was red also. I saw the remains of this, consisting in a faint red eruption, scattered in irregular patches somewhat resembling the eruption of scarlet-fever. For two weeks drink had regurgitated through his nose. He found it hard to swallow, and at times choked. There was much greater difficulty in talking. Bowels were constipated; micturition was natural. Pulse 78, resp. 25. There was no facial paralysis, and the tongue was protruded straight. He gradually sank, and on June 9th, 3 A.M., died.

Autopsy, 8 P.M. I was assisted by Drs. Lincoln and Put-

nam. There was nothing abnormal noticed in either brain or medulla oblongata. The cord was not congested, the membranes and bones seemed normal. The lumbar enlargement felt unusually firm, firmer than the rest of the cord. In the cervical enlargement, the right anterior cornu seemed larger than the left. In the dorsal region, the white columns on section bulged out, and projected beyond the gray substance. In one section in the upper dorsal region, was a semi-translucent spot in the antero-lateral column; the side was not noted.

Other organs.—On the surface of the lungs were small hemorrhagic spots. Otherwise they were healthy. The heart, kidneys, spleen, and liver, all seemed normal.

Only the rectus femoris on the left was examined, and that was of a very pale color.

Microscopic Examination.—*Muscles.*—The portion of the rectus femoris, which was the only muscle examined, showed nearly all forms of degenerative changes, except the hypertrophied, amyloid, or waxy change. Fibres were seen reduced to an accumulation of fatty granules, kept in place by the sheath of the fibre; others showed fat granules scattered through the fibre; in some the transverse striæ were more or less completely preserved, in others they could not be seen. On some there was a multiplication of nuclei; on others, there were no nuclei. Some showed a waxy degeneration, but were not hypertrophied. The accompanying drawing, made with a camera lucida, shows the changes seen in three fibres.

Nervous System.—*Spinal Cord.*—The *lumbar* and *dorsal* region did not harden well, and no satisfactory sections could be made. In the upper dorsal region, one section showed that changes seen in the cervical region were present. Possibly this portion had been accidentally crushed after removal, as by mistake the cord was thrown in with some soiled cloths after the autopsy.

In the *cervical* region, the outline of the gray substance varied from the normal. On the left, it was nearly natural; on the right, the anterior cornu was irregular in outline; in the posterior cornu, and to its outside, was an exudation which was strongly colored by carmine. The pia mater did not appear

to be materially changed, but its smaller vessels were rather distended with blood.

Nerve-Cells.—While in the section from the upper dorsal region only a solitary cell or two could be found in the anterior cornua, in the cervical enlargement many more were to be seen. Yet, compared with the normal number, they were very few, and of those which remained not one came up to the healthy standard in regard to size, the number of processes, or the appearance of their contents. They were, with a few exceptions, much below the average size of healthy cells; most had not more than one process, and in many this was wanting. Several still contained a nucleus and nucleolus, but the contents were more coarsely granular than in health, without any increase of pigment. The cells occupying the regions named by Clarke the vesicular columns and the tractus intermedio lateralis, had suffered to a similar degree; those of the posterior cornua had undergone less change, yet were rather fewer in number and decidedly less in size, with less numerous processes, yet they had quite generally retained their nuclei.

The *vessels* were nearly or quite normal, excepting an increase of nuclei in their external or perivascular sheath. No thickening of their walls could be recognized. Around many of the vessels was an exudation which had hardened by the action of the chromic acid, was strongly tinted by carmine, and had an amorphous, sometimes slightly granular, appearance. This exudation was confined chiefly to the gray substance, and especially the right posterior cornu. Wherever the exudation was only small in extent, it was in close relation to a bloodvessel; when, however, it was extensive, a bloodvessel was not always to be seen near it. Because of its relation to bloodvessels in so many cases, I mention it in connection with them; for I believe it to be a true exudation from them, and not merely broken-down nerve-tissue.

It is not easy to describe the condition of the *white columns*. The *nerve-fibres* were much changed, indeed scarcely any were normal. The spaces occupied by them were clearly to be seen, but instead of being filled with the medullary sheaths and axis cylinders, there was nothing to be seen but a homogeneous mass not colored by carmine; occasionally an axis cylinder

could be seen ; more frequently a small amount of coagulated substance, possibly the medullary sheath, or the degenerated axis, was seen in the centre or on one side of the space formerly occupied by the nerve-fibre.

Judging from the number of nuclei of the *neuroglia* to be seen in specimens stained by logwood, I should think this number was not increased excepting in limited areas, where they were grouped together in much larger number than usual, and where possibly was a more advanced stage of degeneration, perhaps the beginning of a softening. The fibrous tissue of the neuroglia was not apparently increased in amount, but the outlines were less clearly and sharply defined than in normal specimens, and then they were in sections at a higher level.

Around the *central canal* was a limited multiplication of nuclei in the section from which this description is taken, but by no means so great as is often seen in health. The whole of the gray and white commissures were strongly tinted by carmine. In other sections there was so much distortion and softening that the central canal could not be seen, and the posterior cornua were illy defined.

At a higher level, above the cervical enlargement, and again about one fourth of an inch below the olivary bodies, the changes were less marked. There were nerve-cells in small numbers in the anterior cornua, the nerve-fibres were much changed in the white substance, but a large proportion were furnished with an axis cylinder. The exudation was to be seen scattered through the gray substance chiefly, only occasionally in the white columns. Specimens put up in glycerine, under a low power, showed this distribution better than those which had been rendered transparent.

Just below the apex of the calamus scriptorius there were several spots of exudation, anterior to the central canal. These sections showed in the neighborhood of these spots ; and in the region occupied by the cells of the spinal accessory, a large number of spider-like cells, that is, cells with a small amount of protoplasm surrounding a nucleus, and a very large number of fine processes, running in various directions.

At the lower part of the calamus scriptorius, just above its

apex, was to be seen an interesting change in the hypoglossal nucleus. The nerve-cells were few in number; many had an appearance as if filled with an amorphous material with few granulations, and those few crowded to one side, either without a nucleus, or, when present, it was elongated and pushed to one side. There were many swollen axis cylinders. These I at first thought were changed capillaries, but on careful examination I saw that they were solid, were not furnished with nuclei; one or two could be traced to nerve-cells; they were confined strictly to the nucleus and the track of the nerve passing from that toward the periphery; also there were no anastomoses. They were more or less varicose, some were twisted like a cork-screw. At a higher level were numerous cells, but atrophied, globular, and with few or no processes, very different from the normal cells of the hypoglossal.

Nerves.—A posterior root in the lumbar region showed a large number of fibres with axis cylinders, but instead of being near the centre of the fibre they were mostly excentric; the myaline was coagulated, and in some cases there was an exudation of an amorphous material, which seemed to have pushed both the medullary sheath and axis cylinder to one side; sometimes this exudation was in the centre of the medullary sheath. On specimens put up in Canada balsam, this exudation seemed to be structureless, and was not tinted; possibly it had been dissolved out by the method of preparation. In many fibres there was no axis cylinder, but the centre of the sheath was occupied by a small coagulated mass, which did not stain from carmine, the sheath remaining as large as, or larger than usual. A very large proportion of the space was filled with small circles of connective tissue, inclosing a free space about as large as an axis cylinder, with amorphous, homogeneous contents, which were not stained by carmine.

The *anterior root* showed a more extensive change. There were only a few axis cylinders remaining—not more than eight or ten in each bundle of fibres. The rest of the section was composed entirely of the empty sheaths appearing as circles or masses of a granular appearance under low power,

which under higher power were seen to be a combination of very small circles and nuclei.

In the *crural nerve* there were sections of nerve-fibres, much more healthy than in the posterior roots, also fibres from which the axis cylinders had entirely disappeared. There were none of the masses of granular appearance already referred to. The fibres were either nearly healthy or seemed to have reached the last stage of degeneration.

The *ganglion* on the *posterior root* showed some changes. The cells were present in the normal number, the contents of the cells varied. Some contained masses of pigment, grouped at one side or one end; in others, the granules were spread more uniformly over the whole cell, and they were then less deeply colored. In those cells in which the pigment was massed together, the nucleus and nucleolus could generally be clearly seen. In those where the pigment was more evenly distributed, the nucleus could less frequently be seen. The tinting of the nucleus by carmine varied much in different cells in the same section; in some it was only faintly tinted, and had a clear, well-defined boundary, with a clearly-defined nucleolus. In other cells it was deeply tinted, its edge being less well defined, and the nucleolus not visible. The number of comparatively healthy nerve-fibres was quite large, in striking contrast with the anterior root, seen also beside the ganglion.

The connective tissue of the ganglion was decidedly granular, corresponding with the change described by Lubimoff; but this change was not so great as I have seen it in another case.

The changes found in this case were such as are due to inflammation and destruction of the nervous elements of the cord, its fibres, and its ganglion cells. The most interesting and perhaps as important a part of the examination as any, is that of the hypoglossal nucleus. The cells seemed to be passing through the change which led finally to their disappearance. I am not sure that in the present case this change can be proved to be clearly inflammatory; for it was the extension of a diseased action, from one part to another part con-

tiguous; as, however, the change at a slightly lower level was doubtless inflammatory, I suppose we may consider the whole of it so.

Can this case be included in any subdivision? The autopsy shows that all the nervous elements of the cord were implicated. It was a case of purely parenchymatous inflammation; the connective tissue or neuroglia was not materially affected; there was no sclerosis.

Is this case properly included under the subdivision Spinal Paralysis, as described by Dr. Seguin? The symptoms as given by him are: "Dysæsthesia, and slight temporary anæsthesia, paresis, and akinesis; both these symptoms affecting the extremities, and in rare cases the face, eyes, tongue, and throat; not affecting the respiratory muscles, nor those of the back and abdomen, nor the bladder, nor the sphincter ani. Muscular atrophy in the paralyzed parts; loss of electro-muscular contractility (to faradic current) in the atrophied muscles." "The important negative characters of this affection are: Absence of palsy of the bladder, or of the sphincter ani, or of respiratory muscles. No bed-sores, no great and extensive anæsthesia, no spinal epilepsy." (Pp. 27, 28.)

In the present case, the symptoms agree very nearly with the above list, excepting that the patient did not acknowledge to dysæsthesia until toward the close of life; also death took place from implication of the respiratory muscles. The difficult micturition was only temporary; there was no paralysis of the sphincter ani, but rather constipation, though how great is not mentioned. The electrical reaction was not tested, but it is not unreasonable to say that in muscles so extensively degenerated the faradic reaction must have been lost. This case belongs as properly under the class referred to as many which are given by Dr. Seguin, certainly as properly as Case XXI., which I saw in June to August, 1873.

Judging from this case, and from others in which there was no autopsy, Dr. Seguin's XXI., for instance, I can not look upon these cases as primarily due to lesion of the anterior cornua exclusively, and atrophy of the nerve-cells there situated.

I by no means deny that the anterior ganglion cells may

be diseased and other portions of the cord suffer comparatively little. But where there is so much disturbance of sensation as is found in most of these cases, and especially where numbness or other changes in sensation are noticed before the motor disturbance, it seems to me most reasonable to consider the primary lesion as more general, and that other parts are implicated also. A congestion of the cord would be found to explain all the earlier symptoms. I saw a case awhile since which strongly resembles these cases, excepting that it was comparatively slight, and the patient recovered in the course of two or three months.

A young lady was employed for about two months superintending the preparation of a wedding outfit. About the time of the marriage, she went to church and was chilled, and afterward felt chilly. Very soon after her marriage, she began to have pain in her arms and legs, in the muscles and joints. There seemed to be no rheumatic element. The pain was not sharp, but the sensation as if the parts were "asleep." She soon after fell back on the bed on trying to stand. Had a sensation as if a wide band was tied around the upper part of her arm and middle of the thigh. She could move her feet and legs better than the hands. Moving the legs caused no pain, but turning over or moving the arms caused pain. After about two weeks, she could walk, but only slowly, and was very feeble. There were no cerebral symptoms, the bowels and bladder acted naturally. When seen there was still pain in the arms, but the sensation of touch remained intact, and seemed normal in the feet. I subsequently heard that she entirely recovered. It seems to me I was justified in the diagnosis of congestion of the spinal cord and its membranes.

In comparing this case with those of spinal paralysis, the rapid subsidence of the symptoms and early recovery are the chief differences. If from some natural or acquired cause the motor cells are of lessened vitality, it is easy to understand that more or less of them may be permanently destroyed, also other parts of the cord may react less vigorously than in health, and so such changes follow as were found in the fatal case just reported.

In the cases reported by Dr. Seguin, there was numbness

or a sense of coldness or other abnormal feeling observed, either at the same time or previous to the loss of motor power, in XVII., XVIII., XIX., XX.; and in XXI., the patient himself told me distinctly that numbness was the first symptom; and in XXII., it preceded the paresis by about three days.

On looking over my notes, I find the records of nine patients not yet noticed, who had symptoms of spinal paralysis. This excludes cases which might be considered doubtful, as being perhaps cases of sclerosis, or cases of traumatic origin, excepting one case where the person was in the habit of lifting heavy weights. In six of these nine, numbness or pain or tingling were noticed either before or at the same time with the weakness. In two, numbness was mentioned, but the date at which it was noticed is not given. In one, there was pain in the back and shoulders, but no numbness before the motor weakness.

This case of spinal paralysis differs from the following only in the fact that in it the central gray matter is more severely affected, and the cells of the anterior cornua are affected; whereas, in the second case, the most extensive changes are found on the periphery and in the white substance. The second case is less complete clinically, and the condition of the muscles was not examined after death. The patient was treated at the City Hospital under Dr. Sinclair, who has kindly allowed me to make use of the case. I made the autopsy while I was pathologist of the hospital.

M. M. W., a carpenter, æt. 40, entered the City Hospital December 27th, 1869, under Dr. Sinclair. He had had two attacks of gonorrhœa, but never chancres nor skin eruption. The previous spring, he was troubled for some time with sore throat, severe pain in the forehead and back of the head, and he became quite deaf and was dizzy; he also had pain in his ankles, which were swollen. He was relieved, and left the hospital February 7th, 1870. July 1st, 1870, he again entered, and was again under Dr. Sinclair. Between February and July, he had frequent attacks of vomiting, also much pain shooting through his right eye-socket to the back of his head. For the last two months, he had been gradually losing power

over his legs. The disturbance in them began with numbness and a sense of formication. Sensation was apparently good, reflex action was absent. There was retention of urine, fæces passed involuntarily. There was a bed-sore over the left trochanter. He failed steadily, and died August 12th. If I remember rightly, I was told at the autopsy that the paralysis advanced steadily, and some days at least before his death the arms were affected.

I made the autopsy six hours after death. The viscera were all healthy. There was no pressure on the cord from any morbid growth; it was normal in size. The dura mater was more congested in the middle dorsal region than elsewhere. The gray matter of the lower part of the cord was much congested. There seemed to be no softening of one part more than another; its consistency was about normal. The brain seemed healthy.

An examination of the nerves of the lumbar and sacral plexuses in a fresh condition, showed that some nerve-fibres had undergone degeneration.

Just below the cervical enlargement the cord was considerably injured in removal. This part could not be examined microscopically; also a part of the dorsal portion; just below the part injured had changed so much that it did not harden satisfactorily, and sections could not be made sufficiently thin to show well under the microscope.

There were found, on examination, meningitis, myelitis, secondary degeneration above and below the softened portion, an exudation about the vessels in certain regions, and a very general disease of the vessels.

In the upper cervical, in that portion immediately above the cervical enlargement, in the upper dorsal and the lower dorsal, and in the lumbar portions, were found many corpora amylacea. In all these regions the distribution of these bodies was nearly the same. They were very small, but were clearly brought out by sulphuric acid and solution of iodine. They were found on the circumference extending entirely around the cord; the posterior columns were more deeply sprinkled by them, especially the central portions bordering the posterior fissure. At the posterior roots, they were also numerous, less

at the anterior roots and the anterior columns. In the cervical region, they were more numerous in the anterior columns than they were at other heights. In the dorsal region, they were more numerous in the lateral columns; and in the lumbar region, there were very few in the antero-lateral columns, but they were collected around the posterior roots and the posterior fissure. In all regions, occasionally one could be seen near the central canal. They generally were found near bloodvessels or in the thicker fibrous septa which run into the cord from the pia mater. A few were near the floor of 4th vent. in the medulla.

At about the middle of the cervical enlargement, were found many spots of exudation, especially in the gray matter. One extending a considerable distance longitudinally was just at the bottom of the anterior fissure, another occupied a part of the central canal, others were found around the bloodvessels near the centre of the cord, others in each posterior cornu, around the bloodvessels, and a few small faint spots were seen in the posterior white substance. None were found in the anterior or lateral columns nor among the cells of the anterior cornua, only at the roots of these cornua.

These spots were finely granular, seemed to separate the proper nerve-tissue, the fluid from which they were formed forcing its way in between the fibres. Under a high power, the larger one showed a striated appearance, the markings being arranged almost concentrically, the centre of the large spot showing a tendency to fatty degeneration. At the end of a large spot, away from any bloodvessel, numerous blood-globules were seen; generally, however, these did not show, either not being present, or being fused together so that the outline of the globule could not be distinguished. These spots were brightly tinted by carmine; the carmine also brought out and showed distinctly the presence of this exudation around numerous small vessels where it would otherwise not have been suspected; just enough being present among the other elements to give a light and uniform pinkish tint. Acetic acid, potassa, sulphuric acid and iodine, glycerine, spirits turpentine, and water, had no effect upon these spots of exudation.

These same spots of exudation were seen also in the lumbar

region, but less extensive than in the cervical, and in the upper part of the medulla oblongata.

The pia mater throughout was thickened, and in its substance were numerous nuclei or pus corpuscles; these were also to be seen in the fibrous tissue following the vessels and fissures, and in the substance of the cord. The layer of the cord next the pia mater contained no nerve-fibres, was uniformly tinted by carmine.

The nuclei in the perivascular sheaths of the vessels of the pia mater and cord, medulla oblongata and pons were greatly increased in number.

Many of the nerve-fibres of the cord were destroyed, but in many also the axis cylinder could be seen. The fibres of the neuroglia seemed swollen and and not clearly defined.

The cord had also undergone secondary degeneration, which does not, however, show so well as it probably would had there not been so many other changes. By careful examination and repeated sections, I saw that this degeneration followed the course first pointed out by Türk. Above the part which was injured in removing, the central portions of the posterior columns were affected; in the upper dorsal these parts, the lateral columns and the anterior columns, not quite to the border of the section, and also the central gray substance, were softened. Lower still in the dorsal region, nearly the whole cord was softened. At about the middle of the dorsal region, below the softened portion, the posterior columns were free, except a narrow border of secondary change just along the edge of the right posterior cornu; the left lateral column was changed throughout, except a narrow portion at the outer edge; the right lateral column was changed in its posterior two thirds, not quite to the gray substance. The anterior columns were changed toward the central gray substance, leaving a small border not affected in them—the change was greatest on the right. In the lower dorsal region, the anterior and posterior columns were not affected. The lateral columns were changed, the change not extending to the gray matter nor to the outer edge, and being greatest on the left. In the upper part of the lumbar enlargement, the change was confined to about the centre of the lateral columns. Toward the lower part of the

lumbar region, it was still less, confined to the centre of the lateral columns, and very slight on the right. It gradually disappeared first from the right.

In the lumbar region, several roots of nerves were examined. The posterior roots had undergone apparently considerable degeneration, also the anterior roots, though in these more nerve-fibres had retained their normal appearance. The pia mater surrounding the nerve-roots and the bloodvessels connected with them, showed the same increase of nuclei, or presence of pus corpuscles, as were seen around the cord. Beyond the dura mater this change was not noticed.

In the spinal ganglia many of the nuclei of the connective tissue were granular. The nerve-cells were strongly pigmented, their nuclei were more deeply stained by carmine than is usual in healthy cells.

These two cases represent two classes of spinal disease. Clinically, there is a difference, in that in one there was bed-sore, retention of urine, and involuntary discharge of fæces. The silence in regard to the condition of the muscles in the second case may be supposed to show that there was no marked wasting. Unfortunately, at the autopsy I did not examine them. Pathologically, the difference was very great. In the first case, the anterior cornua and their nerve-cells were much diseased; the membranes and the neuroglia of the white substance comparatively little. In the second case, the anterior cornua and cells were scarcely touched; the neuroglia of the white substance, especially at the periphery, and the pia mater, were extensively changed. In the first case, the anterior roots were more affected than the posterior; in the second, the reverse was the case. It is interesting to notice the almost complete destruction of the nerve-fibres of the anterior roots in the first case, where the cells of the anterior cornua were destroyed; whereas, in the second case, in which those cells were nearly healthy, the nerve-fibres of the anterior roots were also nearly healthy. In the second case, the ganglion of the posterior root was more affected than in the first case. The difference in the condition of the bloodvessels in the two cases is of especial significance. In the second case, the nuclei of the perivascular sheath were much increased, and the walls of the vessels

were thickened; this was especially marked by contrast in the nerve-trunks. In the first case, the walls of the vessels in the bundles of fibres of the crural nerve were of normal thickness. In the second case, in the sacral plexus the walls of the vessels were much thicker than usual.

Is this difference sufficient to warrant the application of a distinctive name? It seems to me it would be convenient to have such a name for cases wherein the anterior cornua and their cells are so seriously affected, especially as they may be clinically distinguished. That proposed by Frey is satisfactory—*Poliomyelitis anterior*—provided it is recognized that the changes are not absolutely confined to that region, but that the white substance also may be invaded.

Infantile paralysis and spinal paralysis in the adult have been associated. Clinically there are marked differences. In the former, the origin is more sudden, the paralysis sooner attaining its greatest development, the retrocession of symptoms occurs earlier and is more decided, the subsequent course is more universally without relapse, the resulting atrophy and consequent deformity more decided, the general health of the patient much better; the disease shows no tendency to extend to other parts after the first onset. The changes after death can hardly be compared, from the fact that there have been few autopsies among adults, and most of those after infantile paralysis have been made late in life.

Probably the primary lesion in both is congestion. The variation in the symptoms and in the subsequent changes may be explained by the comparatively undeveloped condition and embryonic nature of the infantile spinal cord. There is not time to explain more fully my views in regard to this difference.

As to "acute ascending paralysis," the sooner that name is dropped the better; for it expresses no pathological condition, and only assists in perpetuating the idea that there is no organic disease in such cases. It is, in truth, a cover for imperfect knowledge, and tends, like all such names, to hinder advance in the study of nervous lesions.

EXPLANATION OF FIGURES.

Fig. 1. Degenerated muscular fibres from Case I. *a*, fatty degeneration; *b*, a few fat globules scattered through the atrophied fibre, multiplication of nuclei in the sheath; *c*, multiplication of nuclei, only a few of the transverse striæ visible.

Fig. 2. Healthy hypoglossal nucleus, showing the large multipolar cells.

Fig. 3. Hypoglossal nucleus from Case I. This section was taken from very nearly the same level as the preceding. Both are equally magnified. *a*, atrophied cells; *b*, enlarged axis cylinders; *c*, cells nearly of normal size.

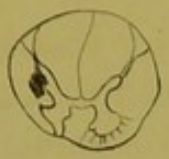
Fig. 4. Section of part of an anterior root from lumbar region in Case I. *a*, one fibre nearly healthy; *b*, circles formed by the neurilemma containing neither axis, cylinder, nor medullary sheath; *c*, masses of small circles and nuclei, arranged in indistinct groups, a little larger than nerve-fibres. Other nuclei are seen scattered irregularly over the section. *d*, two bloodvessels in the fibrous septum dividing these bundles of fibres from others not represented.

Fig. 5. Section of part of an anterior root from the lumbar region in Case II. *a*, nerve-fibres containing only coagulated medullary sheath without axis cylinder; *b*, fibres in which the axis cylinder and medullary sheath are pushed to one side; *c*, small circles, probably degenerated fibres without axis cylinders; *d*, an enlarged fibre.

Fig. 6. Section of part of a posterior root from the lumbar region in Case II.

Fig. 7. Section of an artery with a small vein at its side, from a section of a nerve from the sacral plexus in Case II. *a*, artery showing the nuclei and perhaps the sections of muscular fibres toward its inner layer, the thickened nearly homogeneous layer external to this, and the nuclei in the external layer; *v*, a very small vein. A group of nuclei will be seen around the vein and another at the opposite side of the artery; the nerve-fibres also show much degeneration.

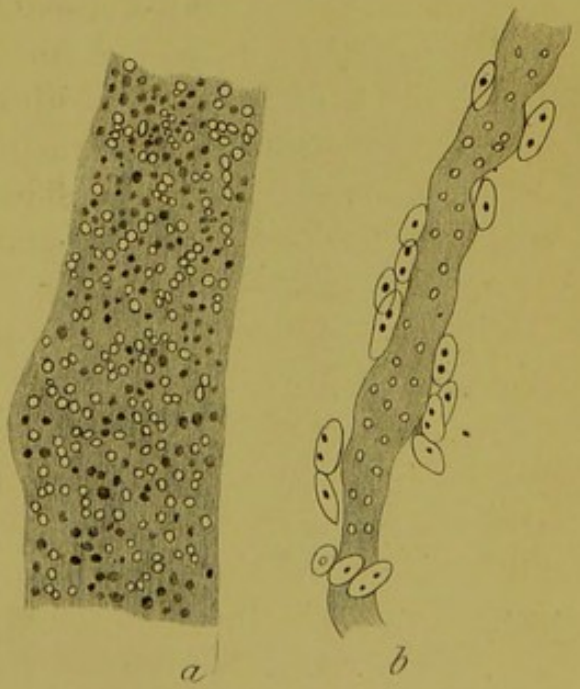
Figs. 4, 5, 6, and 7 are magnified equally. All the figures were drawn with a camera lucida.



Out line of cord and grey substance in middle cervica enlargement. Case I. The dark shaded portion is the seat of the exudation.

Fig 3.

Fig. 2.



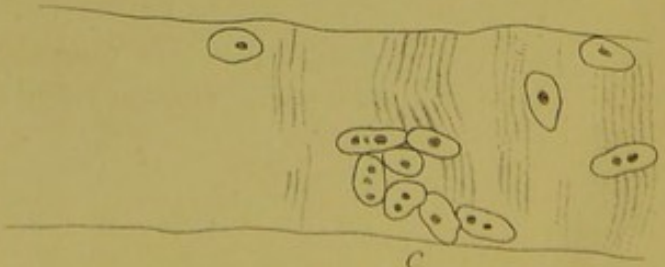


Fig. 1.

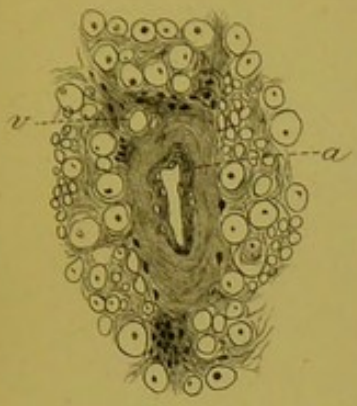


Fig. 7.

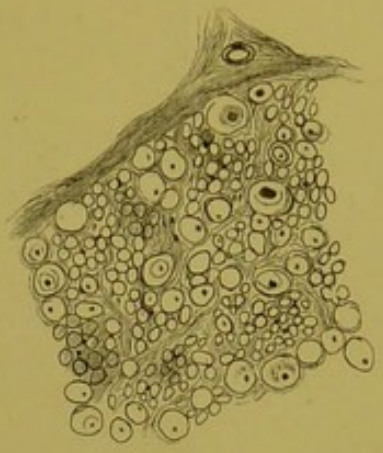


Fig. 6.

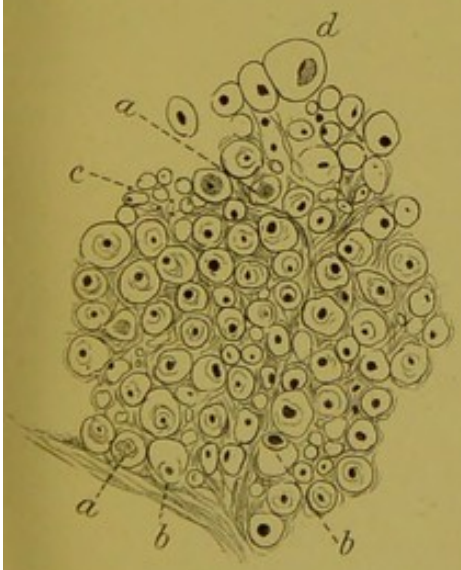


Fig. 5.

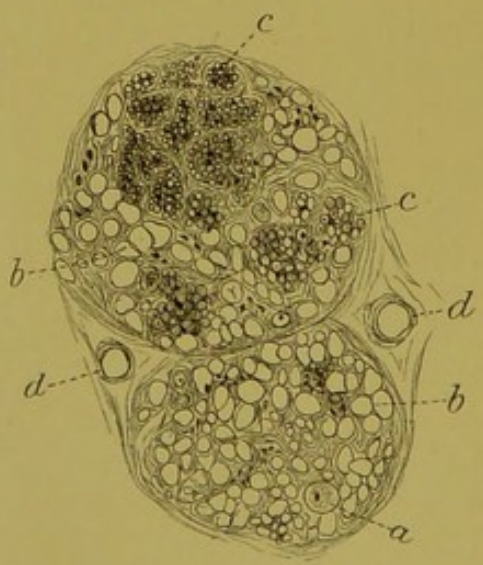


Fig. 4.