A study of the paths of secondary degeneration in a case of injury of the cervical spine / by Arthur V. Meigs.

A STUDY

OF THE

PATHS OF SECONDARY DEGENERATION

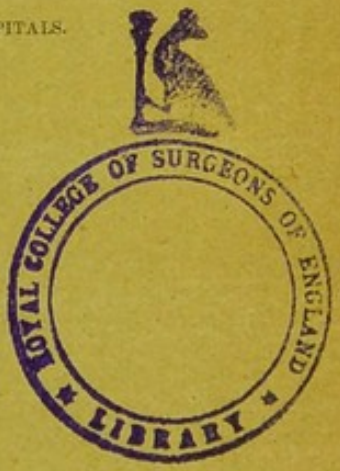
IN A CASE OF

INJURY OF THE CERVICAL SPINE.

BY

ARTHUR V. MEIGS, M.D.,

PHYSICIAN TO THE PENNSYLVANIA AND CHILDREN'S HOSPITALS.



REPRINTED FROM THE TRANSACTIONS OF

THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, MARCH 5, 1890,

AND FROM

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES,

AUGUST, 1890.

A STUDY OF THE PATHS OF SECONDARY DEGENERATION IN A CASE OF INJURY OF THE CERVICAL SPINE.

By ARTHUR V. MEIGS, M.D.,
PHYSICIAN TO THE PENNSYLVANIA AND CHILDREN'S HOSPITALS.

[Read March 5, 1890.]

Although at first sight it might appear that one case of any given disease is like another, and, therefore, that isolated cases are hardly worth placing upon record, yet the one I propose to relate presented features that are not so common as to render them trite, and, at the same time, showed the incorrectness of statements which common acceptation has caused to be looked upon as facts. The subject of the results of spinal injuries has long occupied a large share of the attention of surgeons; and neurologists and anatomists have been much occupied of late in studying the upward and downward paths of the secondary degenerations which follow injuries of the spine, whether traumatic or the result of the processes of disease; for, beside the interest which always attaches to the study of pathology, it being an acknowledged fact that the more perfectly we understand the morbid processes of any disease the more competent we are to treat it, anatomists have learned that from a study of the course of the secondary degenerations they can best follow the paths of the nerve fibres from their origin in the brain to their final termination in skin, muscle, or elsewhere, according to what their special function may be.[1]

[1] My most hearty thanks are due to Dr. Packard, my colleague at the Pennsylvania Hospital, who placed the case at my disposal and under whose charge the patient was while in the ward, and to Dr. W. D. Green, the resident physician who made the post-mortem examination and prepared the history for me.

Henry B., thirty-five years of age, a sailor by occupation, and born in England, was admitted to the surgical ward of the Pennsylvania Hospital August 20, 1888, and died September 15th of the same year. During heavy weather at sea, ten days before his admission, he was struck by a wave and dashed against the bulwarks striking the back of the head and neck against the rail. It was found at once that he had lost all sensation and power of motion from the clavicles downward, and from the time of the accident he had retention of urine and incontinence of feces. When admitted to the hospital there was a large bedsore upon the back, entire loss of sensation below the clavicles, and abolition of the reflexes. Examination of the urine gave negative results. The temperature varied between 100° and 103⅘° F. There were no marks of violence nor signs of fracture or luxation of the vertebræ. The treatment consisted of the administration of iodide of potassium, the use of a water-bed, and a poultice upon the chest. Even upon his admission there were some coarse mucous *râles* to be heard upon examination of the lungs, and this condition gradually increased until the lungs were full of *râles*, and he became unable to expel the mucus; abdominal tympany came on and he became comatose and died seemingly of heart-failure.

Post-mortem examination showed that there was neither luxation nor fracture of the spine, but a small extra-dural hemorrhage into the spinal canal at the level of the seventh cervical vertebra.

In regard to the condition of the cord it will be necessary to give some details of the methods employed for its examination and preparation for microscopical study. When first removed the dura mater was slit up upon both the anterior and posterior aspects to expose the pia and nerve-roots and the substance of the cord cut transversely at many points, the sections being made at intervals of from an inch to an inch and a half. Though it was then examined with ordinary care nothing abnormal was noticed, and it was placed in Müller's fluid for preservation and hardening. After the tissue had been a few weeks in the fluid, when removed for examination it was at once obvious that it presented marked evidence of disease. When the lower part of the cord was looked at, a fresh transverse section having been made, it was seen that the gray matter was stained of the yellow tint produced by Müller's fluid and that the greater portion of the white matter had the usual greenish color, but there was in the antero-lateral region upon both sides a large spot which was roundish

when the cord was looked at, as already stated, in horizontal section. This area was of a yellow hue, the shade of color being as nearly as possible the same as that taken by the gray horns. Upon cutting across the cervical portion—and it must have been at about the level of the seventh vertebra, the precise relation of the parts to the vertebræ was unfortunately not preserved, the nerve substance was seen to have lost all apparent uniformity of geographical arrangement, no distinction between gray and white matter could be discovered. The cord when thus seen presented a surface somewhat rough and irregular—and was of an almost even dirty yellow color, presenting at no point the greenish shade which is taken by the white portion of normal spinal cord. At all points, the cord below this area was natural, so far as naked-eye appearances went, except for the irregularly round spot in the antero-lateral columns which was everywhere distinctly visible, extending down into the lumbar region, where it was perhaps more evident even than above, and for two comma-shaped spots of yellow in the postero-external columns (see Plate I., Fig. 2 *a*) in the upper dorsal section cut a short distance below the area of injury. Unfortunately but little of the cord above the position of disintegration was preserved, but what there was showed perfectly distinctly that the yellowish spot in the antero-lateral columns was absent, but that a portion (see Plate I, Fig. 1 *a*) of the posterior columns was of the same yellow color—thus showing ascending degeneration above the area of injury.

Pieces were taken for microscopical study from the region of destruction in the cervical portion; a short distance below this area in the upper dorsal portion; from the mid-dorsal, and from the lumbar region. Two sets of these were prepared, the one by the celloidin method and stained with Weigert's reagents, the other in paraffine and stained with carmine, and carmine and sulpho-indigotate of potash. A third set were prepared according to the method of Schultze, which has been described in the *American Monthly Microscopical Journal*, December, 1889, by Dr. George A. Piersol, who pre-

pared these sections for me—the staining material being carminate of soda. Sections were also prepared and stained by the Schultze method of a piece of the cord, a very short distance above the area of degeneration, from the cervical enlargement. Teased preparations were also made both of tissue taken directly from Müller's fluid, and, after being stained in carminate of soda and these mounted, some in glycerin and some in balsam; of portions of the antero-lateral (crossed) pyramidal tract which had been carefully dissected out, and of the anterior white substance which was still healthy, for comparison.

It will probably be best to begin the description of the histological conditions observed with the disintegrated area, and afterward to consider the secondary degenerations passing up and down the cord.

The sections were taken as nearly as can be judged from about the region of the seventh cervical vertebra. The large artery at the edge anteriorly presented some thickening of the intima, which was more marked upon one side than the other; others at the posterior surface also showed moderate increase of thickness of intima from nuclear proliferation. Veins at the posterior surface presented evidence of inflammation, and one contained a well-organized laminated clot occluding its calibre. The greater portion of the tissue of the cord itself had undergone complete disorganization, but the extent of this and its geographical distribution will be better understood by an examination of Plate II., Fig. 1 than by any description. Toward the periphery anteriorly a thin fringe of tissue remains, and posteriorly the whole of the posterior columns—except a small portion (constituting perhaps one-sixteenth to one-eighth of the area) near the commissure and quite a large area of the lateral column—upon one side are in a condition quite or nearly normal. In both these areas the nerve-fibres can be readily distinguished, and even the axis-cylinders are sharply outlined and well defined. The rest of the tissue, including thus a small part of the anterior portion of the posterior columns and much the greater part of the anterior white substance, with the whole of the gray matter, is completely

disorganized and is made up of the so-called fat-granule cells, swollen and distorted nerve-fibres, corpora amylacea, large nucleated cells staining very red with carmine, and much space, apparently empty, which was probably filled with colloid material or liquid, for if it had not been so the tissue would have collapsed instead of hardening in Müller's fluid. At the junction of the uninjured tissue with the disintegrated portion everywhere there is a boundary layer of material, greater or less in extent, which stains very bright red with carmine, showing the condition of cell activity which is to be looked for at the periphery of areas undergoing any stage of the process of inflammation.

In order to facilitate and make clearer the description of what was observed in regard to the condition of the cord above and below the area of destruction which has been described, two sets of diagrams were made (see Plate I.). These represent, upon the one hand, the paths of degeneration as their outlines would have been described from a macroscopical examination of the tissue alone after it had been hardened in Müller's fluid—for, as has already been stated, no abnormality was noticed when the fresh cord was examined; and, upon the other, these same paths as outlined from a careful microscopical study of the state of the nerve-fibres and other elements made from very good sections cut and stained in several different ways and from teased preparations.

Sections made a short distance above the area of disintegration, which, as already stated, was at about the level of the seventh cervical vertebra, showed that the tissue was not anywhere in as good histological condition as that taken below that region and further away from the area of transverse myelitis. Though the greater part of the nerve-fibres were natural looking, there were at all portions of the cord at this level scattered fibres which were more or less degenerated. The position in which the fibres are in the best state of preservation is the lateral (crossed) pyramidal tracts. An examination of the two diagrams (Plate I.) gives the best understanding of

the apparent geographical distribution, both macroscopical and microscopical, of the paths of secondary degeneration.

It is evident that the gross appearances are very misleading, but this matter will be discussed later. Microscopical examination shows that in this particular case the whole of the posterior columns had undergone some degeneration, the greater portion, and that toward the centre lying nearest to the commissure in particular, having been almost entirely destroyed, there being hardly any normal fibres remaining, while a rather narrow band at the posterior edge showed only a partial destruction of the tissue—it consisting of nerves whose outlines were sharp and distinct—and many others showing partial destruction of the axis cylinders, some of these being swollen or fatty, or, again, very granular looking and stained intensely red with carmine. Some of these latter were undoubtedly axis cylinders, swollen, disintegrated, and inflamed, for in preparations that had been stained with carminate of soda and then teased out into shreds (see Plate II., Fig. 3) many nerve-fibres could be seen in which the axis cylinders were very red, distorted to all sorts of shapes, and granular. In places the irregular swellings were so great as to be as large as the diameter of the myelin sheath.

Toward the inner sides of the lateral (crossed) pyramidal tracts the areas of degeneration abutted directly against the posterior gray cornua, there being positively no band of healthy or even partially healthy tissue separating them.

In the anterior white columns, extending around at the periphery from the posterior gray cornua well toward the anterior, and involving the whole of the direct cerebellar tracts and what Gowers describes as the antero-lateral ascending tracts, were upon either side belts of tissue which had undergone partial degeneration, the degree of change being about the same as that of the most posterior part of the posterior columns. The area occupied by the degeneration can be better understood from an examination of the diagram (Plate I.) than by any description. Almost directly opposite the lateral branch of the anterior gray cornua upon one side, the red

stained spots that have been mentioned were larger and more numerous than anywhere else—their appearance is well represented in Plate II., Fig. 2,—and the teased preparation (Plate II., Fig. 3) proves that they are changed axis cylinders.

In the gray matter some of the large multipolar cells were seen to be round and granular in appearance and no branches could be seen, nor nuclei; while others, again, were sharp in outline, the nuclei distinct, and several branches remained attached to them. Whether these appearances were due to disease or not cannot be positively stated.

In the sections from the upper dorsal cord a short distance below the area of transverse myelitis, and in those from the mid-dorsal and lumbar portions, the degeneration in the lateral (crossed) pyramidal tracts was very evident, and its appearance, as compared with the healthy tissue from the anterior portion, is most graphically shown in Plate III. The nerve-fibres are swollen, in many instances the axis cylinders having disappeared, or, again, they are distorted and irregular in outline, and a very marked feature is the overgrowth and increase of the neuroglia, which has, at the same time, lost much of its regularity of arrangement.

The strands seem to be irregularly thick, have lost their sharpness of outline, and no longer present the natural appearance of running in a regular radial manner inward from the pia mater from which they start. The diagrams (Plate I.) show better the areas of degeneration than any verbal description. It may be seen how different are the impressions of the geographical distribution of these areas to be derived from the microscopical and macroscopical appearances, which latter are very misleading. The large comma-shaped spots in the posterior columns in the sections taken from the upper dorsal portion do not appear upon microscopical examination as areas of degeneration at all, the only change which corresponds being a distinct, but not very great, increase of thickness of the neuroglia, which is stained very red by carmine, and a separation of the fibres as if by effusion. A noticeable feature is, that in all the sections taken from below the area of destruc-

tion—to a slight degree in those from the upper dorsal region and markedly in the mid-dorsal and lumbar regions—the nerve-fibres in the posterior columns toward the commissure, though in a good state of preservation, are very much separated as though by the effusion of some fluid or colloid material. This could not have been the result of any faulty technique, as it is plainly to be seen in sections prepared in three entirely different ways. In several of these sections cut below the area of transverse myelitis, there was a strong suspicion of the existence of slight degeneration in the anterior (direct) pyramidal tracts, but it was not absolutely certain, and, therefore, not figured in any of the diagrams. With reference to the extent of the degeneration of the lateral pyramidal tracts, an examination of the diagrams shows that, as their geographical relation was determined by microscopical study in the sections from the upper dorsal region, the direct cerebellar tracts were left untouched, and a little more than is commonly described as belonging to this region, for the bands of healthy tissue at the periphery extended well back to the posterior gray cornua, the degenerated matter nowhere coming in contact with the enveloping pia mater, while at the inner side the degeneration extended flatly up against the posterior gray cornua, leaving no healthy tissue in what is described by Gowers as the lateral limiting layers. In sections from the mid-dorsal region the areas of degeneration were closely parallel to those last described. In the lumbar region, however, the conditions differed markedly (see Plate I., Figs. 4 *a* and 4 *b*), both from those found in sections taken from portions of the cord above and from the macroscopical appearances. The areas of total degeneration were separated from both the periphery and the posterior gray cornua by bands in which some slight change had taken place, but in which the greater part of the fibres were still in a good state of preservation. The spinal nerves, where any of them are included in the sections, exhibit marked degeneration; the degree of this is greater in the sections from the lumbar and mid-dorsal regions than in those from the upper dorsal, though still unmistakable

in the latter area. There are also large clear spaces between the bundles of fibres where the bloodvessels lie, as though there had been distention of the lymph sheaths. The character of degeneration of these nerves presents a marked contrast with that in the cord itself, looking as though a mere shrinkage and wasting of the tissue had occurred as a consequence of disuse, very different from what is seen in the cord, which exhibits all the appearances of a more or less active inflammation—in brief, the one looks as if it was a secondary and remote consequence of something that had occurred far away, the other like an active process going on where it is seen. A marked feature in the sections taken from these three regions below the area of transverse myelitis is, that the columns of degeneration in the lateral pyramidal tracts in the lumbar sections cover a much larger area than they do in the mid-dorsal, and fully as large as in the upper dorsal, this being contrary to what is said commonly to occur—that the areas of degeneration became progressively less as the region of original injury becomes more distant.

Peculiar interest, perhaps, attaches to this case, in that the use of the Schultze method of staining made it possible to study the paths of degeneration and their precise degree and geographical outlines with much more precision than would have been possible by older methods. The Weigert method and very good paraffine preparations stained with carmine, and carmine and sulpho-indigotate of potash, gave results far inferior to those obtained with the Schultze stain, carminate of soda.

The degree of degeneration was slight as compared with that commonly found in long-standing cases of tabes or other forms of spinal sclerosis, which the Weigert method demonstrates so beautifully. The Weigert method, so far as the study of transverse sections of the white substance of the spinal cord is concerned, depends upon the black or bluish-black color taken by the myelin sheaths, and this prevents in healthy tissue any accurate study of the state of the axis cylinders, which are so closely surrounded by the dark-colored

material that their outlines are not distinct. In old cases of spinal sclerosis the myelin, as well as the axis cylinders, has disappeared or undergone so much change as to be no longer capable of giving the characteristic color; and, therefore, the method demonstrates most graphically the degenerated areas. In this case, though, the degenerative processes had not progressed so far, the man having lived about five weeks only after his injury, and the changes had taken place principally in the axis cylinders, leaving the myelin still in sufficiently good condition to take the characteristic color; and therefore, though the changes were sufficiently great for the Weigert method to show their presence plainly, it entirely failed to reveal the geographical outlines and extent, as shown by the other preparations.

The question whether these secondary degenerations of the cord are inflammatory, is one which it would seem possible only to answer in the affirmative, if the definition of Burden Sanderson of what constitutes inflammation be received. In his classical article,[1] he defines it as follows; "By the 'process of inflammation' I understand the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality. With reference to their origin, all inflammations may be comprised in two classes—extrinsic and intrinsic."

This definition is very comprehensive, and would seem to be as good a one as the subject to be defined admits of; the prime difficulty, however, remains—which is, to decide in individual instances among the very long "succession of changes" which occurs, often extending in an unbroken line to regions far remote from the original seat of injury; at what point precisely to draw the line, and say, upon one side the changes are truly inflammatory, and on the other are secondary and non-inflammatory. Certainly no one would pretend to call paralysis of a limb an inflammation, though caused directly by some

[1] Holmes's Surgery, vol. v., page 729.

inflammation of the spine which produced at the original seat effects the inflammatory character of which no one would pretend to deny. In the individual case being dealt with, a careful examination of the nature of the changes which occurred seems to make it sufficiently clear that those in the spine itself were truly a part of the process of inflammation, while those outside of this tissue, beginning therefore with the spinal nerves, were secondary, and would be more correctly described as atrophic than inflammatory.

The difference of the appearances in the two positions is most striking, in the spine the axis cylinders and sheaths being greatly and irregularly swollen (see Plates), as though from an active inflammatory process extending upward and downward from the original seat of injury throughout the cord, while the spinal nerves, when changed at all, are shrunken so as to be much reduced in size—as though they were merely dried up from disuse—none of the commonly accepted signs of inflammation being present. The process of change was an unbroken one, beginning at the seat of original injury as an active inflammation and extending upward—how far, unfortunately, cannot be known, as the condition of the brain was not studied—and downward through the cord to the spinal nerves, and doubtless much further if it had been sought for, with lessening intensity until it ceased to be any longer properly named an inflammation, but became an atrophy.

The fact is a curious one—and its cause will probably be learned in the future, perhaps from a careful study and fuller understanding of the circulation in the parts—that the change from inflammation to atrophy is quite abrupt, and that the line is at the point of separation of nerve from cord.

It will not be amiss once more to call attention to Plate II., Figs. 2 and 3, showing, as they do, such very great swelling of the axis cylinders, and to recall to attention the fact that no condition in any wise parallel to this was found in the spinal nerves, which, although in the nature of things, they must have undergone their changes later than those in the cord, exhibited only alteration which could be described as atrophic;

and to emphasize the conclusion, which would seem a necessary one, that the so-called secondary degenerations of the spinal cord are in truth more correctly to be described as direct extensions of inflammation.

What part of the changes at the seat of injury was due to the direct effect of the blow producing rupture, hemorrhage, and immediate destruction of the tissue, and how much to the disturbance of the circulation and consequent failure of nutrition, it is quite impossible to say, but it would seem certain that the disintegration instantaneously produced must have been very great, for there was absolute loss of function, as evidenced by the complete loss of sensation and motion from the instant that the blow was received.

So far as the ascending and descending secondary degenerations are concerned, a study of their geographical relations and extent—and a very correct understanding of these may be had from the diagrams—brings to light several features of interest. It may be premised that, though the paralysis was absolute from the reception of the injury, the transverse myelitis was not; a large part of the posterior columns was structurally little, or not at all, injured, and two bands of tissue in the anterior white substance (see Plate II., Fig. 1) also were made up of nerve-fibres natural in appearance. A most important point which the study of this case demonstrates is, that any deductions drawn from an examination of the macroscopic appearances alone would lead to very false conclusions with regard to the paths and extent of the secondary degeneration, though the naked-eye appearances of disease were so manifest, after the tissue had been in Müller's fluid, that they could not have been overlooked by anyone. It is in the highest degree likely—nay, almost certain—that in the past erroneous conclusions in regard to the paths of degeneration have been recorded, owing to observers having trusted to the gross appearances alone, and to the fact that by older methods of preparation and staining it was almost impossible to obtain sections which would show the condition of all the nerve-fibres throughout whole sections down to the minutest details, as by

Schultze's method. Beyond question, it is difficult, or even impossible in some instances, to determine the presence or absence of disease by a naked-eye examination alone of fresh spinal cord. Soaking in Müller's fluid for a week or too will very likely bring to light diseased appearances which cannot fail to be recognized, but the study of this case demonstrates that, if the color-changes thus produced be exclusively relied upon, and it be concluded that all areas are degenerated in which the white substance stains of a yellow color instead of the greenish hue generally taken by healthy white matter, and that all parts taking the greenish tinge are healthy, a grave error will be made. The diagrams (Plate I.) show, upon the one hand, the outlines of the yellow-staining spots in the white substance drawn from a naked-eye examination, and, upon the other, the actual areas occupied by degenerated nerve-fibres; the appearances presented, as is readily seen, do not by any means correspond. In the sections from the cervical swelling above the myelitis area (Plate I., Figs. 1 *a* and 1 *b*), the whole of the posterior columns was more or less degenerated, a narrow band at the peripheral portion being slightly so, while that toward the centre had undergone complete disintegration, very few axis cylinders being distinguishable. This is, perhaps, no more than might have been looked for, as the section was cut so short a distance above the region of almost total destruction, and if other sections could have been had from regions still higher, it is likely that the degeneration might have been found confined to the postero-median (Goll's) column, as is said usually to take place.

The degeneration of the whole of the direct cerebellar tracts, and of a portion, at least, of the antero-lateral ascending tracts (as described by Gowers), is what usually occurs in ascending degeneration, and, as has already been said, it was in about the area of junction of these two tracts that were found the largest number of the greatly-swollen axis cylinders; the disease-process seeming here to be very fresh and active.

The diagram Plate I., Fig. 1 *a* represents the area of

degeneration, as shown by macroscopic examination alone, and it may be seen that it is misleading, as it makes it appear that there was no degeneration of the posterior portion of the posterior columns at all, and none of the anterior part of the postero-external, nor any of the direct cerebellar or antero-lateral ascending tracts.

Figs. 2 *a* and 2 *b* represent the appearances, macroscopical and from microscopic examination, below the region of destruction, being of the upper dorsal region. This, as represented in Fig. 2 *b*, shows that the degeneration was of the lateral pyramidal tracts and lateral limiting layers alone. The appearances, as studied macroscopically, are very different—it would seem as if (Plate I., Fig. 2 *a*) the lateral limiting layers had remained healthy, while the greater portion of the direct cerebellar tracts appeared to be involved by the degenerative process; further, there appeared two comma-shaped spots in the postero-external columns, which microscopic examination failed to demonstrate at all—the only change, as has already been stated, that was found in this area being a general separation of the nerve-fibres, as though by some effusion and overgrowth of the neuroglia. This comma-shaped downward degeneration is alluded to and figured by Gowers,[1] but by him is represented as being of much less extent than in my case. It is very strange, and at present inexplicable, that it should have appeared merely as a color change and that no degeneration of the fibres should have been found, but only a separation and connective tissue increase.

Plate I., Figs. 3 *a* and 3 *b* show that though macroscopically it appeared as if the columns of degeneration extended externally directly up to the periphery, microscopic examination showed quite a wide band of tissue (the direct cerebellar tracts) which had remained healthy; the lateral limiting layers were involved.

In Plate I., Figs. 4 *a* and 4 *b*, quite a different appearance is presented: macroscopically, the degeneration seemed to extend

[1] Diseases of the Nervous System, vol. i., pages 118 and 120.

to the periphery, but to leave the lateral limiting layer healthy; microscopic examination showed that the degeneration in truth occupied the whole area outside the posterior horns, but that at the centre it was great in degree, while the lateral limiting layers and direct cerebellar tracts, though degenerated, were so in a less degree. It is curious that nowhere was there any positive descending degeneration of the direct pyramidal tracts, though some of the sections presented appearances suspiciously like it, and, contrary to what is stated to be the usual condition, the degeneration in the lateral (crossed) pyramidal tracts did not become less in extent from above downward as the distance from the seat of injury became greater. On the contrary, the degenerated areas in the lumbar sections, besides being as large as they were higher up the cord, presented, if anything, a still greater degree of overgrowth of the neuroglia. It is worthy of note that sections prepared by the Weigert method, though they exhibited distinctly the presence of degeneration, did not outline its extent nor show the histological condition of the component parts very satisfactorily. This was probably due to the fact that it produces its effect by staining the nerve sheaths of a more or less dark color, thus necessarily concealing, to a greater or less extent, the outlines of the axis cylinders.

In conclusion, it may be well to summarize the points of interest of the case, and to emphasize the lessons it teaches.

First. The use of the Schultze method of staining in addition to the older ones rendered possible a closer study and more complete understanding of the disease than could otherwise have been had.

Second. The suggestion that the changes in the cord would be more properly described as inflammatory than atrophic, as they are usually called, is worthy of notice and consideration. The condition of the bloodvessels described in the sections from the area of transverse myelitis and that of the connective tissue elements would seem to bear this out.

Third. The enormous size of the axis cylinders, greatest in the antero-lateral ascending tracts above the area of myelitis

—a thing the Weigert method did not reveal and which was only made plain by the Schultze stain—would seem to be one of the earliest changes to take place, the white substance of Schwann remaining apparently unaltered and the large axis cylinders being in the midst of nerve-fibres apparently healthy.

Fourth. The question is an interesting one, and at present entirely unanswerable: What portion of the changes at the area of myelitis was at once produced by the original violence, and how much came on afterward as a consequence of the hemorrhage and disturbance of the circulation and consequent failure of nutrition?

Fifth. The separation of the nerve-fibres in the posterior columns, apparently from interstitial effusion, is curious and worthy of attention. There can be no doubt, either, of its existence below the area of injury throughout the dorsal and in the lumbar cord, for it was plainly evident in sections prepared in three different ways, and cannot, therefore, justly be attributed to any fault in technique.

Sixth. It is worthy of reiteration that the columns of degeneration, contrary to what seems to be universally accepted as always the case, did not become smaller from above downward, for they were larger in the sections from the lumbar than from the mid-dorsal region.

Seventh. The fact that the actual paths of degeneration, as outlined by careful study of the microscopical condition of the tissue, were so different from the areas occupied by the color-changes, the only guide to their understanding macroscopically, is a most important one.

Eighth. It is noticeable, too, that the paths of degeneration do not occupy and confine themselves strictly to the areas usually described. This proves either that the ordinary descriptions are somewhat incorrect, or that there is much variability of each case from every other one.

Ninth. The absence of any degeneration of the nerve-fibres in the areas occupied by the comma-shaped yellow spots in

the sections from the upper dorsal region is not easy to explain.

Tenth. In considering the paths of upward degeneration it is curious that the whole of the posterior columns should have been involved, when it is recollected that in the region of greatest destruction, the posterior columns were the portion in the best state of preservation, the nerve-fibres being destroyed only in a very small portion of the front part. This does not tally very well with the doctrine that the change is a degenerative one, and that the disease creeps along the nerve-fibres in the direction of their function.

Eleventh. From the surgical aspect the case is interesting, especially in connection with the question of railway injuries, as demonstrating how complete may be the destruction of the nerve tissues without any bone lesion whatever, whether luxation or fracture.

DESCRIPTION OF PLATES.

PLATE I., × 4 diameters. In Figs. 1, 2, 3, 4 *a* the areas covered with dots are the degenerated regions as determined from macroscopical examination alone, the dotted areas being the portions of the white matter which had a yellowish color, instead of green like the healthy portion of the white matter. On the other hand, and in strong contrast, Figs. 1, 2, 3, 4 *b* represent the true areas of degeneration as determined by careful microscopical examination of sections.

Fig. 1 *a*. Cervical portion, above the area of transverse myelitis. The dotted area represents the ascending degeneration as determined by the color changes produced after soaking in Müller's fluid. It occupies the posterior columns alone, and of them leaves untouched the whole of the posterior portion next the periphery, and most of the anterior portion of the postero-external columns.

Fig. 1 *b*. Same region as Fig. 1 *a*, but the dotted areas here represent the ascending degeneration as outlined by careful microscopical study of sections. The heavier dots represent much degenerated regions, the lighter dots those less so. The whole of the posterior columns is degenerated, the anterior portion more so than the posterior, and degeneration extends around at the sides in the antero-lateral columns, occupying the regions called the direct cerebellar and the antero-lateral ascending tracts.

Fig. 2*a*. Upper dorsal portion, below the area of transverse myelitis. The dotted portions are the regions of descending degeneration as outlined from the gross appearances after soaking in Müller's fluid. There are the comma-shaped spots in the posterior columns and degeneration of the crossed pyramidal tracts. The macroscopic examination made it appear that the lateral limiting layers were not involved, while the degeneration did occupy all, or nearly all, of the direct cerebellar tracts.

Fig. 2*b*. Same region as Fig. 2*a*, but the dotted areas representing the descending degeneration as determined by microscopical study of sections. The crossed pyramidal tracts involved together with the lateral limiting layers, while the direct cerebellar tracts are free from change. No degeneration of the posterior columns.

Fig. 3*a*. Mid-dorsal region. The descending degeneration here as outlined from the gross appearances seems to occupy the crossed pyramidal tracts and to extend flatly up against both the periphery and the posterior gray horns, occupying, therefore, both the lateral limiting layers and the direct cerebellar tracts, if the latter extend so far posteriorly in this region.

Fig. 3*b*. Same region as Fig. 3*a*. The descending degeneration as mapped out from microscopical examination. It exists in the crossed pyramidal tracts and lateral limiting layers alone. As there is a wide portion of healthy tissue between the column of degeneration and the periphery, the direct cerebellar tract is certainly not involved.

Fig. 4*a*. Lumbar region. The degeneration here, as determined from examination of the color changes, seems to occupy the crossed pyramidal tracts and to extend quite up to the periphery externally, but to leave the lateral limiting layers uninvolved.

Fig. 4*b*. Same region as Fig. 4*a*. The descending degeneration as outlined from microscopical examination. There is here a central column of greatly degenerated tissue (represented by the heavier dots) surrounded at both sides and posteriorly by a less degenerated portion (represented by the lighter dots), which fills up the whole of that portion of the antero-lateral white substance lying between the periphery externally and the posterior horns internally. This column of degeneration is much more extensive than is the one in the dorsal region (see Fig. 3*b*).

PLATE II.

Fig. 1, × 10 diameters, stained with borax carmine. The region of transverse myelitis. The tissue almost all destroyed except about half of the posterior portion of the posterior columns and a band of the antero-lateral white substance at the posterior edge, and a still narrower one at the anterior edge upon the right hand in the picture.

Fig. 2, × about 400 diameters, stained with carminate of soda. Taken from the cervical portion above the region of transverse myelitis. The picture shows greatly swollen axis cylinders (the large black spots), and was taken from

the antero-lateral white column near the periphery of the cord at about the junction of the direct cerebellar tract with the antero-lateral ascending tract.

Fig. 3, × 400 diameters, stained with carminate of soda, and the tissue teased to isolate single nerve-fibres. The picture shows a nerve sheath and axis cylinder, the latter irregularly swollen and granular in appearance. This was from the crossed pyramidal tract in the upper dorsal portion of the cord.

Plate III.

Fig. 1, × 225 diameters, stained with carminate of soda. This picture was taken from the anterior region (the anterior ground fibres) of a section of the upper dorsal portion of the cord, and shows the typically healthy appearance of the nerve-fibres and connective tissue.

Fig. 2, × 225 diameters, stained with carminate of soda. From the same section as Fig. 1, but a picture of the crossed pyramidal tract showing descending degeneration in a high degree of development. The axis cylinders have in most places disappeared, and the arrangement of the connective tissue is distorted, having lost its radiating appearance. The contrast between these two pictures, which were taken from different regions of the same section, is a most striking demonstration of the more obvious differences in appearance between the healthy and diseased portions of tissue.

The pictures were all made by photographic process, and without retouching, from beautiful negatives made directly from the sections by Dr. George A. Piersol, except the diagrams (Plate I.) which was drawn by Dr. B. A. Randall, and Fig. 3, Plate II., which is a pen-sketch by Dr. Allen J. Smith. Without the assistance of these gentlemen to represent graphically what cannot be adequately described in words, my paper would have been almost valueless.

DISCUSSION.

Dr. Wharton Sinkler: I have heard this paper with a great deal of interest. It is one of great value, as are all studies and researches in regard to the pathological changes taking place in the cord, especially when we consider that nearly all our recent advances in our anatomical knowledge of the cord has arisen from a study of pathological changes in it. In the sixth edition of Dr. Hammond's work, published only little more than ten years ago, it is stated, in regard to a large portion of the white matter of the cord, that its anatomy and pathology are unknown. It is only in the past few years that what we now know of these regions of the cord has been discovered. The areas of degeneration shown in this case are not absolutely in accord with those hitherto described, but, in the main, they are. We can understand why, if there was an inflammatory condition, these degenerated

areas in the crossed pyramidal tracts may have become larger as we depart from the seat of injury, instead of becoming smaller, as other observers have stated.

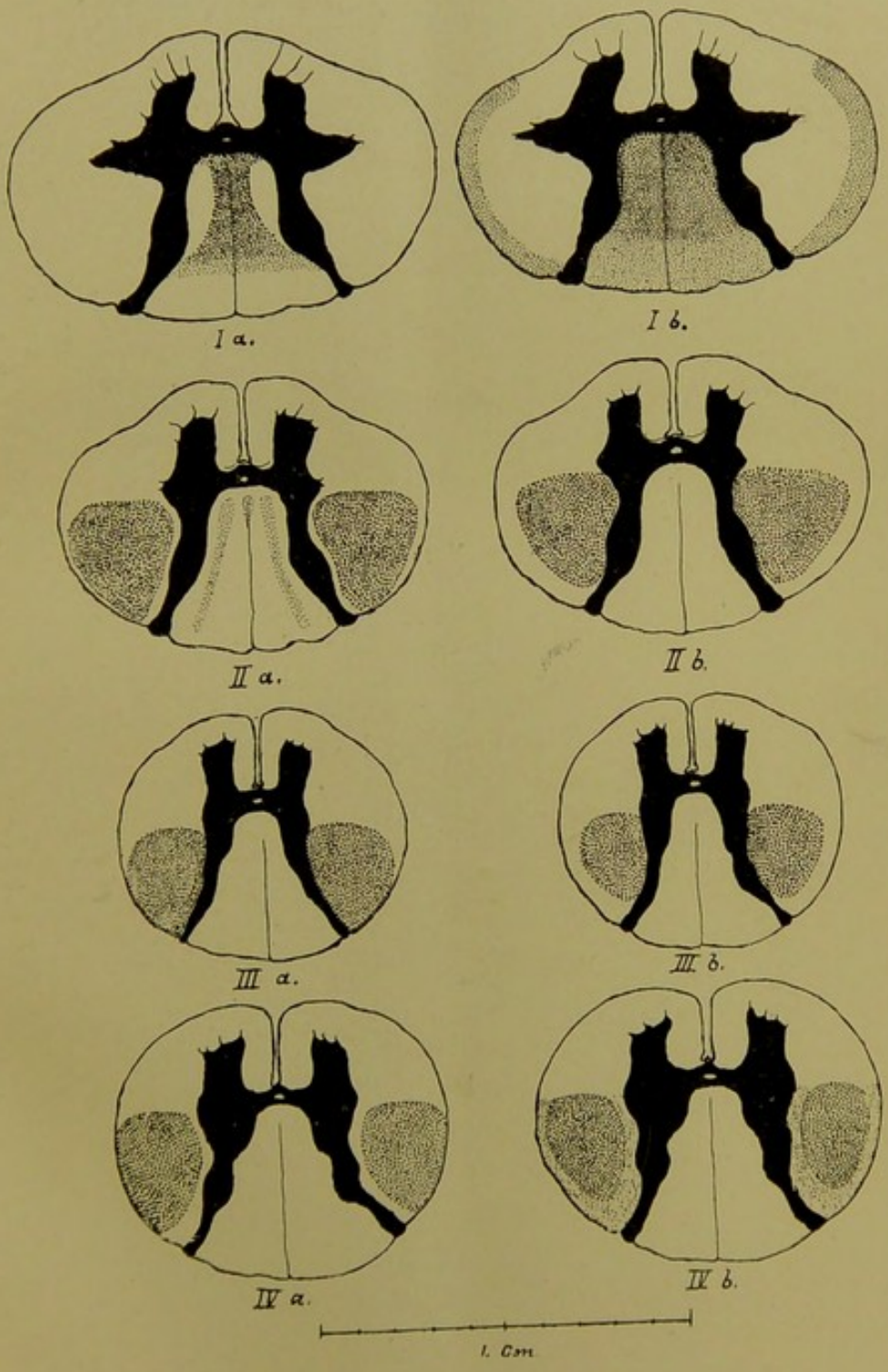
There are certain practical points of interest in connection with this case. One is in regard to the gross appearances. When the cord was exposed there was no unusual appearance of the spinal cord, although such extreme degeneration was found to have taken place on making a section. This is of importance in connection with operations upon the spinal cord. When the laminæ have been removed, it is sometimes impossible to say whether or not the cord presents a normal appearance.

Another practical point is the absolute failure that would have followed an operation in this case. In the light of other experiences, there was certainly a great temptation to operation to determine whether there was any pressure from displacement of the vertebræ. In many cases where operation has afforded good results, there have been no more evidences of pressure than were present in this case.

The absence of all the reflexes is interesting and rather difficult to understand. It would have been of value to have had the reflexes more carefully studied. There is no mention made whether or not the reflexes were studied with the aid of reinforcement. In some cases where the tendon reflexes are absent, they may be developed by reinforcement.

Dr. Francis Dercum: This case is certainly thoroughly worked up, and is exceedingly interesting. I am inclined to think that the difference in results from those ordinarily found may be due to the fact that the primary lesion was a very gross one, and one in which all the structures at the seat of injury were more or less involved. There was distinct inflammation, and this may account for the increased size of the lateral tracts in the lumbar cord. Another thing is the appearance of nerve-fibre in teased preparations. I understood the operator to state that the axis cylinders changed early. In the classical experiments of Ranvier the axis cylinders were the last to change. This subject is, of course, still open, and there is a great field, especially in the study of secondary degeneration after apoplexy, in which to determine whether or not diminution of the degenerated area increases the further we go down the cord.

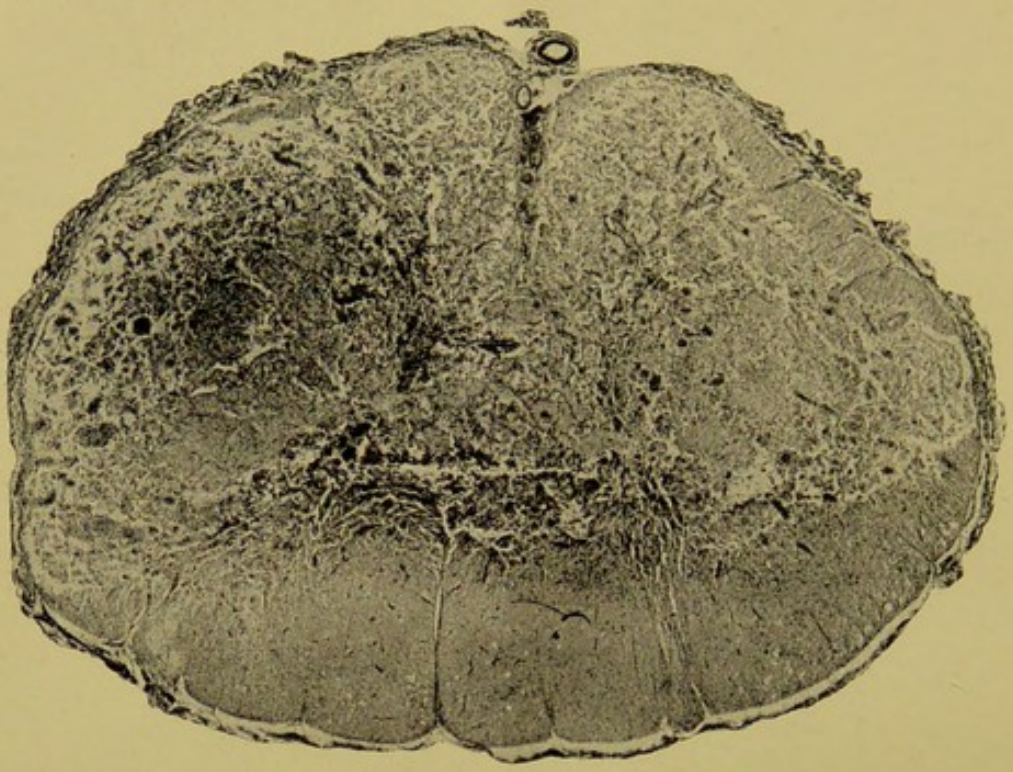
PLATE I.



× 4 diameters.

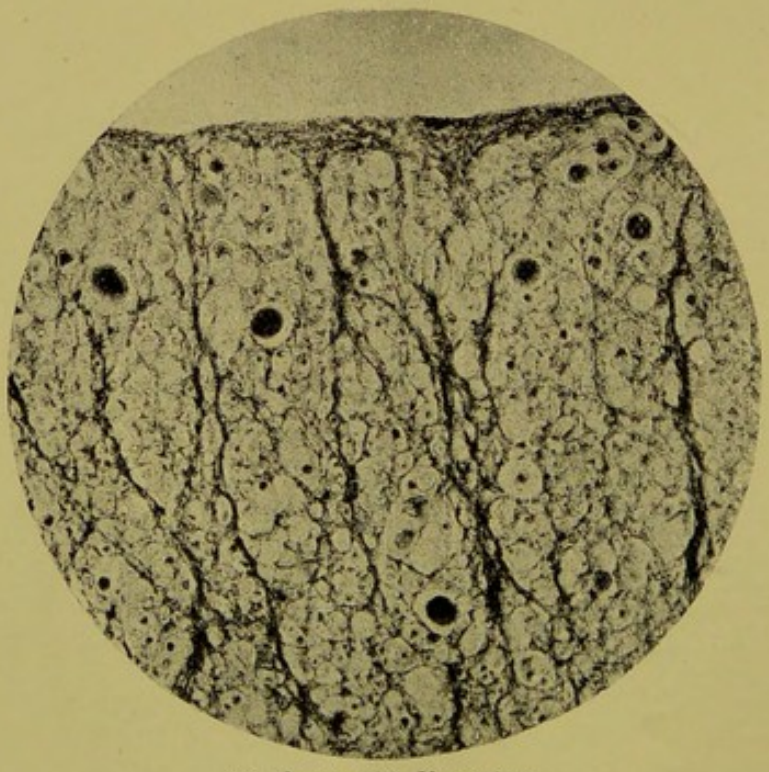
PLATE II.

FIG. 1.



× 10 diameters.

FIG. 2.



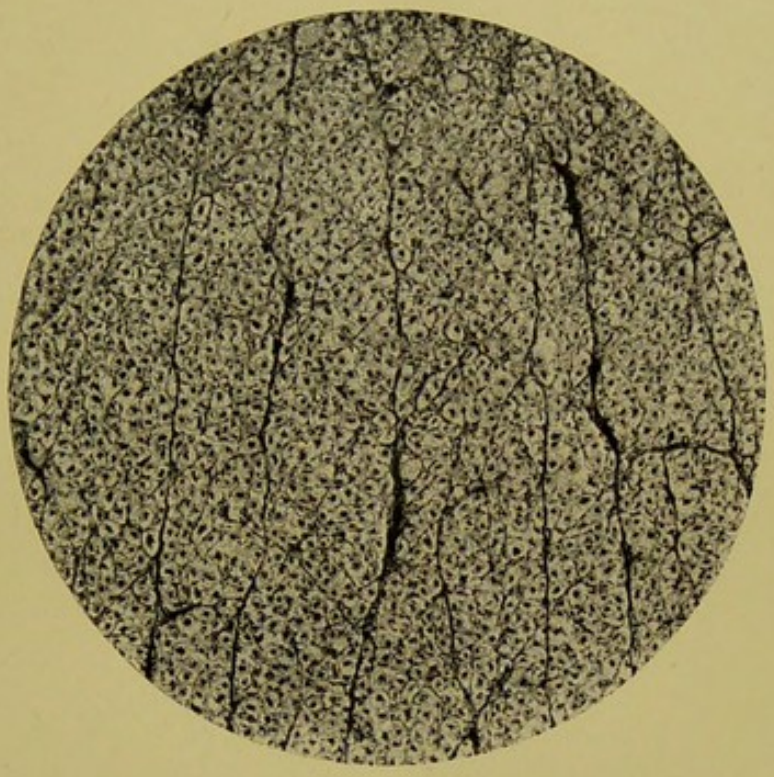
× about 400 diameters.

FIG. 3.

× 400. 1/10 millimeter

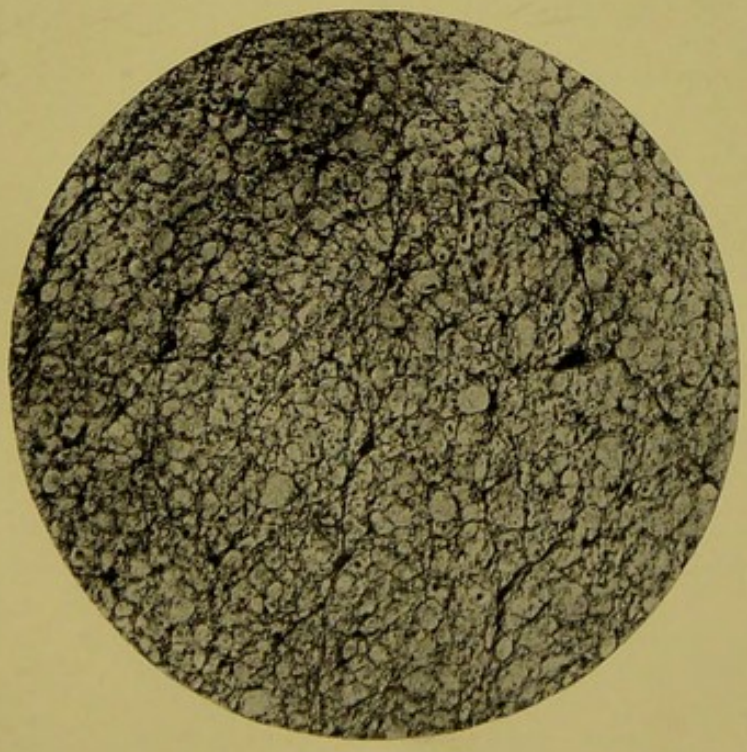
PLATE III.

FIG. 1.



× 225 diameters.

FIG. 2.



× 225 diameters.