ON A CASE

OF

CONCUSSION-LESION,

WITH

EXTENSIVE SECONDARY DEGENERATIONS OF THE SPINAL CORD,

FOLLOWED BY GENERAL MUSCULAR ATROPHY.

BY

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The following case presents many distinct points of interest. In the first place, because from a concussion not more severe than might occasionally be experienced in a Railway accident, the most unmistakeable, and even extensive, lesions of the spinal cord were produced at the time, and recognised after the patient's death. Then, though these primary and other extensive secondary lesions of the spinal cord were recognisable with the greatest ease after the organ had been immersed for a period in chromic acid, yet at the time of the autopsy itself, when the organ was in the fresh condition, no morbid appearances were detected even by careful scrutiny, and that for reasons which I shall hereafter be able fully to explain.

Much interest also attaches to the distribution of the areas of secondary degeneration, on account of the bearing which this has upon the physiological anatomy of the spinal cord; and also to the histological nature of the changes produced, since these serve not a little to elucidate the real nature of cerebral or spinal *ramollissement*. And lastly, the gradual supervention of a general muscular atrophy in conjunction with the wasting of a portion at least of the great sympathetic system, lends an additional interest to the consideration of this important case.

The man on whose spinal cord these observations have been made was a patient of my colleague Mr. Haynes Walton, and I am much indebted to him for his kindness in placing the organ entirely at my disposal for examination. It is much to be regretted that we are unable to give more perfect and accurate notes of the case, and that the clinical history is very imperfect. What I have been able to ascertain concerning the patient is derived from some early notes on the case by Mr. Tatham, late house-surgeon to St. Mary's Hospital, supplemented by other particulars communicated by Mr. Haynes Walton himself.

History.—Jeremiah C—, æt. 26, admitted into the accident ward of St. Mary's Hospital, July 7th, 1866; about a week ago was sleeping on the top of an unfinished hay-rick, twenty-five feet in height, and whilst asleep rolled off, falling on his back. He found himself, at once, unable to move, and was conveyed to the Barnet Union, where he remained, till, at his own request, he was removed to this hospital.

State on admission.—He lies on his back; his legs are motionless, but he retains sensation in them, although he is unable to state the exact number of fingers touching him at a time. Can raise his thighs a little when he makes a great effort; the toes are dropped and point helplessly downwards; toes twitch when soles of feet are tickled. The right arm is also partially paralysed; the wrist is dropped, he is unable to move the fingers, and he can only just raise the

limb from the bed. The bladder is paralysed, and the urine ammoniacal. The bowels were moved soon after the accident, but have not been opened since. The breathing is purely diaphragmatic, the intercostal spaces falling in at each inspiration: number of respirations per minute, 32. Has a little bronchitis, and has a great difficulty in coughing up the phlegm. Pulse 88, soft. Temperature in axilla 97° Fahr. Already there is a large bed-sore over sacrum four or five inches in diameter. He complains of soreness and stiffness in the neck, and of slight pain in the neighbourhood of the first and second dorsal vertebræ; but no fracture or displacement can be detected there. (Mr. Haynes Walton writes, "he was raised in bed for me to examine his back, but there was no mark, nor any tenderness except on the upper part of the dorsal region; but this was really very slight, considerable pressure and percussion being scarcely complained of.") Ordered:—Ol. Ricini ʒss, statim (by means of which bowels were moved—he himself being quite conscious of the fact, though unable to exercise any voluntary control); full diet, with 4 oz. of port; to be placed on a water-pillow, and the bed-sore to be dressed with glycerine.

July 8th.—Has not slept well, being much troubled with startings in the legs (the first time since the accident). In other respects, in the same state as when admitted.

9th to 13th.—The startings are very troublesome—does not sleep much.

14th.—Startings chiefly confined to the right leg; is able to move the toes of the left foot a little.

17th.—A small slough has come away from the sore, granulations healthy.

21st.—Is gradually gaining a little power in the extensors of the right forearm; can move the fingers a little. The urine now dribbles away from him, even when there are but a few ounces in the bladder; complains of pain in the lower part of the abdomen and along the urethra: there has been a slight purulent discharge from the urethra for the last day or two.

24th.—Seems rather low, the voice is weak, and his speech

drawling. States that his voice is completely changed since the accident. Takes food pretty well; has 6 oz. of port wine in addition to brandy.

August 1st.—Complains of pain in right leg; nothing can be seen to account for it. There is no tenderness at any part of the spine, even when heavy pressure is made.

5th.—Hips and shoulders are a little rubbed; sore on back healthy. Is gaining a little more power over the right hand. Seems to be improving in general health, and is much more cheerful than he was. Is now propped up in bed at his own request.

18th.—Is gaining more power in his left leg; often complains that his legs pain him. Sore over sacrum much healthier.

Through a change of house-surgeons the notes unfortunately cease at this stage. Mr. Haynes Walton tells me, however, that though in the early part of August the patient could move his right arm a little, yet this very soon began to undergo a rigid contraction, which steadily increased till the wrist almost touched the shoulder. Up to this time, also, he was only ordinarily thin, but he soon began to waste perceptibly day by day, in spite of a nourishing diet with plenty of stimulants. For the last two or three months, also, he was often sick and vomited his food. The bronchial tubes became loaded with thick mucus, and at times he seemed likely to suffocate, because he had such difficulty in expectorating. For a long time he passed his urine involuntarily, but afterwards he could retain it, though he suffered great pain during its passage, and latterly blood came away with it. Bowels were never moved without the aid of castor oil. Throughout, sensation seemed to be scarcely, if at all, impaired in the paralysed or other parts of the body. From the first to the last he was on a water bed, and everything was done to prevent other bed-sores from forming, but in vain. His position was continually changed and adjusted, until he became too weak to bear the fatigue which this induced. His mind was clear till the end. He was literally dying for weeks. His appetite got less and less, whilst his

desire for stimulants increased. During the last seven weeks he took daily 2 oz. of brandy, 6 oz. of port, and two bottles of stout with two pounds of beef jelly. He died on December 31st, 1865.

Autopsy thirty-six hours after death.—Body emaciated to a most extreme degree; large sore over sacrum; no scar or appearance of former wound higher up in back; no irregularity of vertebral spines; thighs and knees rigidly flexed; right elbow rigidly flexed.

Brain presented no abnormal appearance. After removing *vertebral* arches, these appeared perfectly natural—there was no displacement or irregularity in any part. The *spinal cord* was in no way compressed. On cutting open dura mater, the vessels on surface of cord were seen to be large and turgid with blood. No wasting or alteration of shape was observed in any part, and on section in the upper cervical region, through middle of brachial enlargement, and in various parts of dorsal and lumbar regions, no morbid change was detected; the internal vessels were also somewhat turgid with blood, but the consistence of the organ was good, and the sections to the naked eye presented a healthy appearance. *Pericardium* dry. *Heart* healthy, small, weight 7 oz.; right cavities containing semifluid blood. No fat on surface. Both *pleuræ* dry and free from serum. *Left lung*, old adhesions about apex; deposits of tubercle and superficial puckering also in this situation; a few small granular patches of tubercle in other parts of lung; weight 14 oz. *Right lung* much more firmly adherent and more solidified about apex; also containing tubercle scattered through other parts; weight 19 oz. *Peritoneum* dry. Areolar tissue around organs also quite dry and tough, so as to cause some difficulty in removal of kidneys. *Liver* large; very pale; moulded to shape of abdomen; tissue more resistant than is ordinarily the case with fatty liver; weight 50 oz. *Spleen* healthy, somewhat small; weight 5 oz. *Kidneys*, both organs in much the same condition; large, very pale, and considerably congested. The congestion was well seen on surface, when capsules, which could be stripped off freely,

were removed. In each organ also there were two or three small calculi of a pale colour, and about the size of peas, situated together with purulent-looking fluid in some of the calices; weight of right 8 oz., and of left 7 oz. *Intestines* (not opened) appeared healthy.

The fact that no naked-eye appearances of disease could be detected in the cord at the post-mortem examination, seeing that extensive deviations from the normal structure were subsequently found to exist, is a subject of much interest in connection with the numerous instances in which pathological changes have been looked for in this organ, and have not been recognised. I may add that Dr. Sieveking and Mr. Haynes Walton were both present at the time of the post-mortem examination, and were also unable to detect anything abnormal in the appearance of the organ, when sections were made in various parts. I call attention to this fact particularly, because it is a very important one to be borne in mind, and because it is the rule rather than the exception in cases of secondary degeneration of the spinal cord. Dr. Bouchard, the author of a most valuable memoir on this subject, writes,[1] "Je dois dire dès l'abord que ces dégénérations secondaires de la moelle échappant le plus souvent à un examen même attentif." As far as the present case is concerned it is easy enough to comprehend why the extensive pathological changes were not recognised by a naked-eye examination of the cord in its fresh state. The diseased tracts did not differ in colour from the healthy nerve-substance, neither did their consistence differ from that of adjacent parts; and, except in one limited portion of the cervical region of the cord (through which particular part no section happened to have been made at the time of the autopsy), a most careful subsequent examination has revealed no loss of symmetry in any part of this organ. With the absence of the ordinary naked-eye characters of disease—with no deviation from the normal consistence, colour, or symmetry of the organ—it is not so much a subject of wonder that pathological changes, complying with these conditions, should

[1] 'Archiv. Générales de Médecine,' 1866, p. 273.

escape detection, even after a careful examination. The occurrence of cases of this kind should, however, strongly impress upon us the necessity of not passing a too hasty verdict upon organs, in which we have failed at an ordinary post-mortem examination in recognising well-marked structural changes, such as might have been expected to have been present, considering the nature of the symptoms presented during life. In this case, I examined, almost immediately after the autopsy, portions of the nervous tissue taken from various parts of the cord, with the aid of the microscope, and at once found that all parts of the organ—from the lumbar enlargement up to the medulla oblongata—contained variable quantities of well-developed granulation corpuscles, or "compound inflammatory globules," as they were unfortunately named by Gluge. Having thus satisfied myself as to the existence of morbid changes in the organ, I cut segments of it off and put them aside to harden in a solution of chromic acid. After immersion for a certain time in this fluid, the areas of degeneration became most easily recognisable by the naked eye, on the surface of freshly-cut segments of the cord, owing to the fact that the diseased tissue, though hardened in the same way as other parts, had not become stained by the chromic acid. Thus the diseased tracts preserved their original dead white colour, whilst the adjacent healthy nerve-tissue had been stained to the usual extent, and presented a yellowish-brown hue. By this difference in colour alone, and even by a naked-eye examination, the distribution and extent of the various areas of degeneration, which I am about to describe, could be easily detected throughout the whole extent of the cord which was thus preserved. It is much to be regretted that I did not preserve the whole organ, since there is every reason to believe that some important lesions must have been situated in a part of the cord occupying the mid-cervical region, which was not kept. Not realising the full importance of preserving the whole of the organ at the time, I put aside, for subsequent examination, only the medulla oblongata attached to about one half of the upper portion of the spinal cord, the brachial enlargement, portions

from the dorsal region, and the whole of the lumbar enlargement, and it was only when too late to rectify the mistake that I discovered how important it would have been to have retained the whole of the cervical portion of the cord. Although the absence of certain portions of this is most unfortunate, still, in the portions preserved, sufficient primary lesions and secondary degenerations have been detected to make this case one of extreme interest.

Before describing the situations of the lesions, and the extensive secondary degenerations resulting from them, it will be well, I think, for me to give a few explanations as to the cause and nature of the latter, since our knowledge concerning this change may still be considered a recent acquisition, and one, therefore, not perfectly familiar to all readers.

It is to Dr. Augustus Waller[1] more especially that we are indebted for our knowledge of the changes which take place in the distal portions of divided nerves; also for important information concerning the different effects produced when the anterior and the posterior roots respectively of the spinal nerves are cut; as well as for the practical application of this knowledge, whereby we are provided with an accurate method of investigating the distribution of nerve-fibres, even in the central nervous system itself. These lines of investigation have been followed out and extended principally by Schiff, and by MM. Phillipeaux and Vulpian.[2] The order of the changes taking place in the fibres of the distal extremity of a cut nerve are these: about the fifth day after section, the nerve-fibres may be seen, with the aid of the microscope, to present the first visible deviations from their normal condition; their contents become slightly cloudy, and the borders of the fibres are less sharply defined—deviations so slight that they can only be recognised by comparison with

[1] 'Nouvelle Méthode Anatomique pour l'investigation du Système Nerveux' (Lettre à 'Acad. des Sciences du 23 Nov., 1851). Bonn, 1852.

[2] "Sur la Régénérat. des nerfs separés des centres nerveux" (Mem. de la Soc. de Biolog., 1859, p. 343). 'La Physiolog. Génér. et Comp. du Syst. Nerv.' Paris, 1866, p. 236.

other healthy fibres. By the eighth day, however, changes are much more easily recognisable; the medullary sheath has become obviously opaque, the double contour of the fibre on each side has become irregular and interrupted in places, owing to a kind of strangulation of the medullary matter; whilst by the tenth day, or even sooner, this strangulation has gone on to actual segmentation of the white substance into portions of various sizes. During the succeeding days the segmentation still progresses; the original fragments of myeline breaking up into successively smaller and smaller portions, which finally assume a more or less spherical form and fatty aspect. These are contained within the sheath of each nerve-fibre, and completely conceal the axis cylinder. After a month or six weeks the segmentation has become more complete, the medullary matter being reduced to small globules; whilst after two or three months only granulations so fine can be seen within the nerve-fibre, that they resemble "une poussière qui remplirait la gaine conjonctive." At last these granulations disappear, and we arrive at the ultimate change: the sheath of Schwan collapses, and folds upon itself and upon the axis cylinder so completely, that the nerve-fibres are scarcely distinguishable as such. Having become grey also from the disappearance of the white matter, a bundle of such altered nerve-fibres under the microscope can scarcely, at first sight, be discriminated from a bundle of fibrous tissue. Dr. Waller believed that the axis cylinder disappeared altogether, and that in cases where a restoration of function took place in the cut nerve, this was due to an actual new production of nerve-fibres amongst the débris of the old. The observations of Schiff, however, and of MM. Phillipeaux and Vulpian, show that in this respect Waller was wrong. Each of these observers had recognised the existence of the axis cylinder, after an interval of more than six months, and they maintain that when the functions of a nerve so altered are restored, this is brought about not by the generation of nerve-fibres, but by the re-formation of the myeline within the shrivelled sheaths, and around the comparatively unaltered axis cylinders of the original nerve-fibres. Thus

much concerning the actual changes in the individual fibres; but Phillipeaux and Vulpian also state that in the experiments which they made upon dogs, they found the atrophied fibres of cut nerves much more difficult to separate from one another, by a teazing process, than were the healthy fibres of an uninjured nerve. This they attributed to the fact that the fine connective tissue, which normally exists in small quantity between the fasciculi of a nerve, had become hypertrophied, and consequently more tenacious.

When Waller (after having ascertained the various changes which take place in the distal portions of cut nerves) turned his attention to the effects of section of the anterior and posterior roots of the spinal nerves, he soon made known important results as to the *direction* which this degeneration takes in sensory and motor nerves respectively; and he came to the conclusion that such atrophic changes were due to the severance of the connection between portions of the nerve-fibres and their ganglionic attachment—to the interruption, in short, of some controlling nutritive power which is normally exercised over the whole length of each nerve-fibre by the nerve-cell at one of its extremities, and from which it proceeds. This influence is usually exercised in the direction of the physiological action of the fibre; thus, when the anterior roots of the spinal nerves were cut, Waller found that the fibres in the proximal portion of the cut roots retained their normal structure, whilst those in the distal portions (from the cut extremities to their peripheral distributions) underwent the changes above described. When the posterior or sensory roots of the spinal nerves were cut, on the other hand, the distal extremities in connection with the ganglia of the posterior roots preserved their healthy structure, whilst the fibres of the proximal portions underwent the atrophic change, and by this means could be traced ascending for various distances in the posterior columns of the cord, and finally losing themselves in the grey matter. From this, Waller concluded that the nerve-cells presiding over the nutrition of the motor nerve-fibres were situated in the grey matter of the cord, whilst those for the nerves of sensation

were situated in the ganglia of the posterior roots—thus showing one definite function at least for these bodies.

Other observations have tended to show that the course of secondary degenerations generally in the antero-lateral columns of the cord may be said to be downwards, whilst in the posterior columns it is just the reverse. The exception to this rule will be stated hereafter. After what has been said, it would be needless to insist upon the important aid which degenerations of this kind afford, in enabling us to determine some of the most difficult problems in connection with the anatomy of the nervous system, by facilitating our tracing the distribution of nerve-fibres through plexuses or complex organs, such as the spinal cord. This method seems second to none for the accuracy of its results, and when used experimentally it has been named by Vulpian, in honour of its discoverer, the "Wallerian method" of investigation.

The above explanations as to the effects produced by sections of nerves, sufficiently explain the nature of secondary degenerations of nerve-tissue, since the process is perfectly similar in each case; and this enables me now to say a few words concerning the history of such degenerations. Cruveilhier was the first to discover descending secondary degenerations as a result of lesions of the brain.[1] These he recognised in the cerebral peduncles, in the pons, and in the medulla oblongata, though he did not succeed in tracing them into the cord. To Türck, of Vienna, is due the honour of having first called attention to these lesions in the spinal cord, in an important memoir[2] which was presented to the Academy of Sciences of that city in 1851, that is to say, in the same year that Waller made known the most important results of his experiments on the degeneration of cut nerves. Whilst, two years later,[3] Türck presented to the same academy another memoir in which he analysed thirteen cases of secondary

[1] 'Anatomie Pathologique,' livraison xxxii, p. 15.

[2] 'Compt. rend. de la sect. de Mathémat. et Sciences Nat. de l'Acad. des Sc. de Vienne,' Mars, 1851.

[3] 'Compt. rend. de l'Acad. des Sciences de Vienne,' t. xi, p. 93, Juin, 1853.

degeneration following cerebral lesions, and twelve others resulting from primary lesions of the spinal cord itself. Notwithstanding the important nature of these communications they appear to have attracted but slight attention, since such lesions are scarcely mentioned in Rokitansky's great work, and are entirely passed over by Lebert. Nearly at the same time that Türck made known the results of his researches, solitary cases were recorded by Schroeder Van der Kolk, and by MM. Charcot and Turner.[1] In 1859 M. Gubler wrote a memoir[2] on secondary degenerations of the brain, and since this time various instances of such changes in the brain and spinal cord have been recorded in the *Comptes rendus de la Société de Biologie* of Paris and in the *Bulletins de la Société Anatomique* of the same city, by MM. Charcot, Velpeau, Cornil, and Bouchard. Leyden[3] has also published a remarkable case of secondary degeneration of the spinal cord, as a result of compression from Pott's curvature of the spine; and lastly, Bouchard has recently published an admirable memoir[4] on the whole subject, to which I have been much indebted, and in which he treats of secondary degenerations of the spinal cord, resulting severally from lesions of the brain, primary lesions of the cord itself, and lesions of the posterior roots of the spinal nerves. To this work I shall frequently refer. So far as I have been able to ascertain, the case which forms the subject of the present communication is the first that has been described in Great Britain or Ireland.

These secondary degenerations, which occupy a certain extent of the columns of the cord, either throughout the whole or a considerable length of the organ, develop themselves rapidly, and also simultaneously, in the different parts of the diseased tracts. This might have been imagined

[1] "Exemple d'Atrophie cérébrale avec Atrophie et déformation dans un moitié du Corps" ('Compt. rend. de la Soc. de Biolog.,' 1852, p. 19).

[2] 'Archiv. Gén. de Méd.,' t. ii, p. 31, 1859.

[3] 'Die graue Degenerat. des hintern Ruckenmarksstrenge,' Berlin, 1863.

[4] 'Archiv. Gén. de Med.,' 1865, and subsequently republished in a separate form.

from what has been already said concerning the changes taking place in cut nerves, and seeing that such degenerations involve a definite series of tissue changes, it will be easily understood that the microscopical characters, and even the naked-eye appearances, of the diseased tracts vary in different stages of the retrograde process. According to Bouchard, secondary degenerations consist of and include the following changes:—1. An atheromatous alteration of the capillaries, and the formation of granulation-corpuscles in the degenerating tissue; 2. The alteration and finally the disappearance of a more or less considerable number of nerve-fibres; 3. The formation of connective tissue which substitutes itself in the place of the atrophied nerve-fibres. It will be seen further on that I accept Bouchard's account of the first two kinds of changes only with certain qualifications. It will be recognised also that the last two changes are precisely those which take place in the distal extremity of a cut nerve, if my interpretation of the nature of the second change be accepted instead of that given by Bouchard. He speaks not only of the alteration, but also of the disappearance of the affected nerve-fibres, and says, "Je puis ajoûter qu'il ne m'a jamais été permis, de retrouver les cylindres d'axe dénudés dans les cas de dégénération secondaire, et aucune observateur n'a constaté cette persistence." To which I can only reply, that when portions of the secondarily degenerated tissue of a spinal cord have been treated with a solution of soda, tinted with carmine, thoroughly teazed with needles, and have then been finally mounted in glycerine, nothing is more easy than to demonstrate the axis cylinders of the nerves (the extremities of which have become tinted) closely enveloped by their shrivelled sheaths. The axis cylinder certainly does *not* disappear; it remains persistent in the same way as it has been demonstrated to do in the cut nerve, and the hypertrophy of the interstitial connective tissue in the degenerating columns of the cord also has its parallel in the growth of the same elements between the wasting fibres of the nerve which has been severed from its central connections. The occurrence of crowds of granulation-corpuscles in the degenerating tracts of the

spinal cord is very characteristic of this species of change; it was to their presence that Türck principally called attention as the result of his microscopic examination; it is owing to their presence that we are enabled to trace with comparative ease the extent and distribution of the areas of degeneration; whilst, lastly, it is owing to the different proportions in which these are met with, in different stages of the disease, that the affected columns vary in appearance. During the first five or six months, at least, after the setting in of these changes, the granulation-corpuscles are most thickly sown through the degenerated tracts, which either retain the dead white colour of the healthy columns, or are tinted of the faintest yellowish hue. Up to this time, too, there is not the slightest shrinking —the wasting of the nerve-fibres has been exactly compensated by the formation of granule-corpuscles and the hypertrophy of the neuroglia on interstitial connective tissue. In the later stages we may get an actual shrinking or loss of substance, owing to the gradual absorption of the granulation corpuscles and the consolidation of the fibrous tissue. In proportion as such changes occur so also do we get alterations of colour: the dead white appearance is gradually supplanted by a greyish, or semi-transparent bluish grey aspect of the diseased columns.

Microscopical examination of the cord.—A transverse section of the hardened cord, through the upper part of the cervical enlargement (apparently corresponding with the interval between the fifth and sixth cervical nerves), showed a large rupture extending obliquely from before backwards across the grey matter of the right side, as well as a considerable shrinking in the antero-posterior direction, and a loss of symmetry of the same side of the cord (Fig. 7, Plate X). On examining this transverse section under a low power and by reflected light, it was seen that not only were the inner adjacent portions of each anterior column opaque white (as could be ascertained by the naked eye), from the presence of an innumerable quantity of granulation-corpuscles, but that bodies of the same kind were scattered more sparingly throughout other portions of the white matter of the cord,

and also in the situation of the grey matter on the right side. On the left side, the outline of the grey matter was quite distinct, and its section presented a normal appearance; whilst on the right side, on account of the large rupture extending completely through the grey matter, together with the opacity of the tissue from the granulation-corpuscles around, the outline of the grey matter could not be detected at all. From the examination of thinner sections which were prepared by Lockhart Clarke's method,[1] so as to make them transparent, and were then inspected by transmitted light, it was at once ascertained that, in those regions where granulation-corpuscles had been seen most abundantly in the sections submitted to reflected light, there was a great diminution in the number of healthy nerve-fibres, and a great increase in the quantity of interstitial connective tissue. It was seen, moreover, that the edges of the ruptured nerve tissue contained a large amount of connective tissue, and that the surrounding nerve matter contained also an increased quantity of this element. From the appearance presented by the section, it seems almost certain that originally the rent in the grey matter on the right side had been most extensive, but that at the time of death a certain amount of repair had taken place, owing to the development of connective tissue, by the contractile properties of which, also, this half of the cord had been drawn in, and so had lost its symmetry.

I may as well state at once that, in the examination of this and other sections of the cord, I have always found the atrophy of nerve-fibres and the hypertrophy of connective tissue, existing in any particular part, in direct proportion to the number of granulation-corpuscles; and that in all the figures I have endeavoured to represent the relative number of granulation-corpuscles by the greater or less intensity of the dotted shading.

In a section one quarter of an inch below the last (Plate X, fig. 8) there was still a slight want of symmetry on the right side of the cord, though it was much less than in the region just above it. There was no longer any rupture to

[1] And also by methods of my own which will be described in the third number (November, 1867) of the 'Journal of Anatomy and Physiology' (Cambridge).

be seen in the same direction as in the last figure, but a smaller one in a direction almost at right angles to it, also extending through the grey matter, and continued outwards and forwards almost to the surface by a band of cicatricial fibrous tissue. The degeneration of the inner parts of the anterior columns was now about equal in amount on each side, and more uniform in depth of shading over different parts of their surface. Two rounded and very dark patches of degeneration were seen posterior to the rupture, and somewhat lighter ones at different parts of the periphery of this half of the cord. Over other portions of it granulation-corpuscles were scattered more sparingly, and the outline of the grey matter on this side still could not be detected. The number of granulation-corpuscles in the left half of the cord was somewhat less than in the last section: the grey matter on this side still presented a healthy appearance. In sections of the cord one quarter of an inch below the last (Plate X, fig. 9) the symmetry of its two sides was almost perfect, and the outline of the grey matter could now be detected in its right half, although more than one half of its surface was occupied by a large and almost square patch of dead white tissue which was made up of granulation-corpuscles, thickly sewn amongst interlacing bands of new fibrous tissue. And in transparent sections, there could be seen, in several parts of the circumference of this patch, the sheaths of blood-vessels filled with amorphous granules of blood-pigment, of a dark olive-yellow colour (Plate XI, fig. 21), whose presence clearly indicated an original rupture of blood-vessels in this situation. The degeneration of the anterior columns is now seen most distinctly to be broader, and to present a more regular border on the right than on the left side. A considerable amount of opacity from granulation-corpuscles is seen in the white matter surrounding the right half of the grey substance, and also over nearly the whole of this half of the cord. The right half, on the contrary, with the exception of the anterior column, is seen to be freer from granulation-corpuscles than it was in the last section; a few only being seen in the inner and posterior part of the lateral column.

In sections one third of an inch below the last (Plate X, fig. 10), the opaque patch in the grey matter of the right side no longer existed; almost the whole of this half of the cord was considerably clouded, and the outline of the grey matter could scarcely be detected, whilst running through its entire extent in an antero-posterior direction was another irregular rupture across the grey matter. In the anterior part of the grey matter of the left side there was a shorter and much smaller rupture, with a slight opacity round it, and in the whole of this half of the cord, more especially around the posterior grey cornu, there was a sparing distribution of granulation-corpuscles. The anterior columns were in much the same condition as in the last section. In sections one third of an inch lower down (Plate X, fig. 11) the rupture through the grey substance on the right side no longer existed; this had resumed its healthy appearance. In the centre of the grey matter of the left side, however, two small solutions of continuity were seen. The anterior columns presented much the same appearance as before, but the remainder of the right half of the section was much clearer than it is represented in the last figure—the principal area of degeneration being a somewhat semicircular one in the posterior part of the lateral column. A few corpuscles were seen also, on this side, in the part of the posterior column next the grey matter, and in the lateral column in front of what Clarke calls the *tractus intermedio lateralis*. A few granulation-corpuscles were also scattered over the left half of the section, but they were very sparse, and only formed a distinct aggregation in the posterior part of the lateral columns, in a situation corresponding with the much larger and more marked area of degeneration in the opposite half of the cord. In sections made two thirds of an inch below the last, and just below the cervical enlargement (Plate X, fig. 12), no ruptures are seen in any part; the large and well-marked areas of degeneration in the anterior columns are somewhat different in shape, the peripheral portions being broader and more extended towards the lateral columns, whilst that on the right still continues rather more extensive than the one on the left side. There

are a very few thinly-scattered granules in the anterior part of the right lateral column, whilst in the posterior portion there is a continuation of the area seen in the last section, which now has an obtuse wedge-like shape, with its apex at the *tractus intermedio lateralis*, and its base, which becomes gradually less defined, falling short of the periphery of the cord, so as to leave a strip of healthy tissue on its outer side. In the corresponding region of the opposite half of the cord, there are some scattered granulation-corpuscles as in the last section, but the grey matter and other parts of the white substance present a healthy appearance. This arrangement continued for some distance, and was found to prevail with but slight variation, even in the mid-dorsal region (Plate X, fig. 13), where we see a continuation of the alteration in shape of the areas of degeneration in the anterior columns, such as we saw commencing in the last section. At the periphery, these patches now extend fairly as far as the lateral columns. The scattered granules in the anterior part of the right lateral column have disappeared, whilst the area in its posterior part has again become more semicircular, and its outer boundary almost reaches the periphery in this region. The few scattered granules in the corresponding area of the opposite side exist as in the last section, whilst the remaining parts have a healthy appearance.

In the lower part of dorsal region, one inch above the lumbar enlargement (Plate X, fig. 14), there was an even more appreciable alteration in the shape of the anterior areas of degeneration, their outline having become more like what it was in the lower cervical region (Plate X, fig. 10), owing to the resumption of the bluntly-rounded shape of the posterior or central extremities of these areas, which had become acutely wedge-shaped in the mid-dorsal region and contiguous parts, and also to the greatly diminished extent of the peripheral expansion of these areas. In the outer part of each, not far from its external extremity, there is a distinct notch, owing to the encroachment of healthy fibres on the diseased areas. The patch in the posterior part of the right lateral column is very distinct still, and now undoubtedly extends

quite to the surface of the cord. The scattered corpuscles in the corresponding region of opposite side are rather fewer in number, but are also situated rather more externally than they were in the sections above. The gradual wearing out of the diseased fibres and the diminution in the number of granulation-corpuscles were well seen in sections lower down, made through various parts of the lumbar enlargement, and have been represented in Plate XI, fig. 15, showing a section through the upper part of lumbar enlargement, and one inch below the last; in fig. 16, which represents a section made three eighths of an inch lower still; and in fig. 17, which was copied from a section made through the lower third of the lumbar enlargement, three fourths of an inch from the commencement of the *filum terminale*. In these it will be seen that the disease disappears first, most notably from the outer parts of the patches in the anterior columns, although at the same time a thinning-out of the diseased fibres takes place also in the inner parts of the same areas of degeneration. In the posterior part of the lateral columns, also, the atrophied fibres and granulation-corpuscles are gradually replaced by healthy nerve-fibres.

From this description it will be seen that the principal one of the original lesions or ruptures of the cord was situated in the upper part of the cervical enlargement, though there is every reason to believe that one or two other important lesions must have been situated in the portion of cord immediately above this, which was unfortunately not preserved. The large oblique rupture represented in Plate X, fig. 7, gradually diminished in extent, and one quarter of an inch lower down became continuous with another smaller rupture in an opposite direction (Plate X, fig. 8). Opposite this first section there had been the greatest damage to the cord, which had been followed by a certain amount of shrinking on the injured side. From the fact that already degeneration was well marked in the inner part of each anterior column, it is almost certain that there must have been another lesion higher up, which had severed the connection between these nerve-fibres and their ganglionic cells. It will be seen,

however, that in this first section the area of degeneration is more extensive in the left than the right anterior column, whilst an inspection of all the figures below will show that the area of degeneration in the right anterior column, instead of being less than that in the left, is decidedly more extensive. This is readily accounted for by the fact that the rupture in the section represented in Plate X, fig. 7, extends into the anterior column of the right side and must have torn across many of its fibres.

In Plate X, fig. 9, there is represented a large square patch in the midst of the grey matter of the right side; this is a section through a diseased portion of the grey matter which was found to extend for a short distance above and below. Now, secondary degeneration, such as we see in the white columns of the cord, never exists in the grey matter, and the limited extent of the patch would also serve to place it in another category. From the fact that in different parts of its circumference, in different sections, I have seen blood-vessels, or rather the sheaths of blood-vessels, such as I have represented in Plate XI, fig. 21, perfectly loaded with altered blood-pigment, it seems most probable that several of the small blood-vessels supplying this portion of the grey matter had been ruptured by the original concussion, leading to effusions of blood into their sheaths and hence obliteration of the vessels themselves from external pressure. The vascular supply to this portion of nerve-tissue being cut off or seriously diminished, the tissue underwent a process of softening, which, at the period of the man's death, showed itself in the stage of repair; it was then made up, for the most part, of well-developed interlacing bundles of fibrous tissue, thickly sown with granulation-corpuscles. In all the sections such as are represented by the first three figures I have described, there was a certain irregular amount of degeneration of the fibres around the right half of the grey matter, but no well-marked area of degeneration in the posterior part of the lateral column.

In Plate X, fig. 10, we again find another distinct rupture completely through the grey matter of the right side; the original of this extended upwards and downwards for a length

of half an inch. A very small rupture also existed in the grey matter of the opposite side. On each side of the posterior half of the rupture on the right side there were considerable patches of degenerated tissue, the inner being in the outer part of the posterior columns (which I shall speak of hereafter), whilst the outer was in the posterior part of the lateral column. Seeing that a well-defined area of degeneration is to be recognised in all the remaining sections in this same posterior part of the lateral column, and that this place corresponds with the situation occupied by the fibres of that portion of the left anterior pyramid which decussates, it would seem almost certain that this area of degeneration is due for the most part to a solution of continuity of these very fibres which have been well named, collectively, by Bouchard, the "*faisceau encéphalique croisé ou externe.*" It would seem, also, almost certain, since no such area is to be recognised in Plate X, figs. 7, 8, or 9, that this band of fibres must have been torn across by the extensive rupture shown in fig. 10, and that in this situation (somewhere about the middle of the cervical enlargement) these fibres must have been situated fairly in the grey matter of the right side, and have been just about to emerge into the posterior part of the lateral column. Thus we get most valuable anatomical evidence as to the place of emergence from the grey matter of this bundle of fibres. In the lower part of the cervical enlargement and in the upper dorsal region the area of degeneration does not extend to the surface of the cord; in the mid-dorsal region and thence downwards, however, the outermost fibres are situated quite at the surface of the cord.[1]

[1] This is in accordance also with the experience of Bouchard, who, towards the end of his memoir, speaking of a case in which the secondary degeneration or sclerosis of the lateral column of the cord was extremely well marked, says:—"Je dois dire que la sclérose atteignait la meninge vers le milien de la region dorsale, au lieu de former une bandelette complétement entourée par la substance blanche saine. J'avais donc été trop absolu dans la première partie de ce travail en disant qu'aucune fibre du faisceau encéphalique croisé n'arrivait au contact de la pie-mère."

The other cerebral fibres situated in the cord—in addition to the band in the posterior part of each lateral column which is formed by the decussating fibres of the pyramids—are lodged in the inner part of each anterior column. This band, which Bouchard names "*faisceau encéphalique direct ou interne,*" seems to be made up by those fibres of the anterior pyramids which do not decussate. This conclusion has been arrived at from the fact that, in cases of extensive cerebral lesion leading to secondary degeneration of the spinal cord, the diseased tract can be traced downwards through the crus cerebri on the same side as the lesion; through the corresponding half of the pons, the pyramid of the same side; and, lastly, along the inner part of the anterior fissure of the cord on the same side, and the posterior part of the lateral column on the opposite side. From the observations made in the present case it would seem, as I have above pointed out, that this decussating band only reaches the lateral column opposite the middle of the cervical enlargement. And inasmuch as in no case of secondary degeneration, according to Bouchard, has there been noted any alteration of the roots of the spinal nerves, it is presumed that the degenerated fibres do not pass into them, but that they terminate gradually at different levels by passing into the grey matter of the cord, and there being continuous with certain of the large nerve-cells of the anterior cornu. The number of fibres in this lateral band gradually diminishes in the lower parts of the cord, though certain of them may be traced, as in the present case, even into the lower part of the lumbar enlargement. But, whilst Bouchard admits this also, he says, with regard to the direct encephalic fibres in the cord, that the longest of them do not extend farther than the middle of the dorsal region. This dictum seems directly contradicted by what we have seen in the present case, where the degeneration of the inner part of the anterior columns only wears itself out most gradually in descending to the lower part of the lumbar region, probably from the gradual passage of its fibres into the grey matter. But this thinning-out of the diseased fibres is not perceptible at all until we get

to the lower dorsal region, and the diminishing areas can be traced with the greatest ease, even into the lower third of the lumber enlargement.

In addition to the bands of encephalic fibres occupying the inner part of the anterior column and the posterior part of the lateral, each antero-lateral column is made up of a number of *commissural* fibres which belong entirely to the spinal cord, and which also have their nutritive centres at their upper extremities. These occupy the whole intermediate space between the two bands of encephalic fibres, and their office seems to be that of longitudinal commissures between groups of cells in the grey matter, situated slightly above one another. Bouchard names them "short commissural fibres;" and he speaks of certain others, much fewer in number, and situated just in front and to the outer side of the external encephalic band (also having their nutritive centres above), which extend for a much greater distance through the cord. These he names "long commissural fibres."[1] Concerning the latter class of fibres, the present case cannot be said to furnish any evidence either positive or negative; but many of the scattered granulation-corpuscles in figs. 7–11 (Plate X), doubtless mark the situation of atrophied short commissural fibres, which, at a level just below the cervical enlargement, have all disappeared with the exception of a few in the anterior part of the right lateral column (fig. 12, Plate X): in the next section represented these also have vanished.

In this, as in other cases of secondary degeneration of the spinal cord, no atrophy either of the anterior or of the posterior nerve roots could be detected.

We now come to the consideration of the degenerations of the posterior columns in the upper cervical part of the cord, and in the medulla oblongata. These belong to another category; they are all ascending degenerations. None of the original lesions which lead to these exist in the portions of the cord about to be examined. These must have been situated in that portion of the cord (below what is about to be described and above the lesion already described in the upper part of the

[1] Loc. cit., p. 570.

cervical enlargement) which was, most unfortunately, not preserved. Inasmuch as nothing can be said, therefore, with regard to the origins of these degenerations, all I can do will be to describe their situations as they ascend through the upper part of the cord and the medulla oblongata. They occupy the posterior columns principally, but also exist in the outer and posterior part of each lateral column.

It has been known for some time that in the most external part of the posterior portion of the lateral columns above the middle of the dorsal region, there are a certain number of fibres whose nutritive cells are situated in the grey matter of the cord at their lower extremities, and which consequently degenerate in an ascending direction. Some of these fibres have been traced upwards through the corpora restiformia into the cerebellum. Areas of degeneration due to the implication of such fibres may be seen in figs. 6–1, Plate IX, marked *c*, *c'* They are seen to extend very far forward on the right side of fig. 6. They diminish rather gradually upwards, and an inspection of figs. 2 and 1 show that they occupy the outer and anterior part of each restiform body, and are situate just behind the dentate nuclei of the olivary bodies. The precise collocation of the fibres seems to vary somewhat as they ascend, judging from the different shapes presented by the same tract of degeneration at different levels.

In the posterior columns of the cord, secondary degenerations always take an ascending direction. These columns seem to be made up of a mixture of fibres, not distinctly separated from one another, part of which, as in the antero-lateral columns, are commissural, whilst the others are continuations of the posterior roots of the spinal nerves. Of these last, some travel but a very short distance amidst the other fibres of the posterior columns and then throw themselves into the grey matter, whilst the remainder travel for long distances in the posterior columns before losing themselves in the same substance. Although these ascending degenerations were pointed out by Türck, our knowledge concerning them is still very indefinite. Concerning the different kinds of fibres in the posterior columns, Bouchard says:—"On

peut demontrer cette proposition en comparant la forme de la dégénération ascendante dans les cas de compression des racines, et de compression de la moelle elle-même. Quand la dégénération succède à une lésion des racines, elle est circonscrites sur les coupes par une portion d'ellipse, la convexité de la courbe etant au avant, et ses deux extrémités reposant sur la face postérieure de la moelle; le tissu extérieur à cette ligne est parfaitment sain. Quand il y a compression de la moelle elle-même ses fibres radicales ascendantes sont atteintes sur un point de leur parcours et vout se dégénérer au-dessus du point comprimé; cependant la figure que présente la dégénération, sur les coupes de la moelle, n'est pas la même. Au lieu d'un segment d'ellipse, on a un triangle dont la base est sur la face postérieure de la moelle, le sommet vers la commissure. C'est que la dégénération porte également sur d'autres fibres qui ont leur centre trophique à leur extrémité inférieure dans la substance grise do la moelle. Ce sont des fibres médullaires propres, comme celles que nous avons indiquées dans les cordons antero-latéraux."[1]

In the present case, as before mentioned, I can say nothing with regard to the precise nature of the lesions leading to the ascending degenerations of the posterior columns. This is the more to be regretted, as they are so remarkably circumscribed and symmetrical. Sections through the lowest part preserved of the portion of spinal cord presenting these lesions, displayed what has been represented in fig. 6, Plate IX. This section is from the upper third of the cervical region of the cord, exactly $2\frac{1}{2}$ inches from the point of the fourth ventricle. Four areas are seen, constituting two almost perfectly symmetrical pairs, one of which is situated in each posterior column. The most internal patches (a, a') have an elongated elliptical form, whose more rounded anterior extremities occupy contiguous portions of the tissue skirting the posterior median fissure in its middle third, whilst their sharply-pointed posterior extremities, almost reaching the surface, slightly diverge from one another. The outer patch of each pair (b, b') reaches forwards as far as the grey

[1] Loc. cit., p. 574.

commissure, and occupies most of the anterior third of the boundaries of the posterior median fissure. From this situation each patch extends backwards and slightly outwards, closely skirting the internal areas and terminating somewhat short of the surface. Of these the area on the right side is somewhat the larger, and terminates in a small process almost at right angles, the two together looking not unlike a man's leg and foot. Following up, first of all, the areas *a*, *a'*, through sections of the lower half of the medulla oblongata (figs. 5, 4, 3, Plate IX), we see that in this situation these areas occupy the greater part of the posterior median columns of the cord. They have become almost triangular also from the widening out of their posterior extremities, and they have extended forwards, on each side of the median fissure, as far as the grey commissure, and have consequently caused a lateral displacement of the outer areas, which, in the lower section, themselves occupied this situation. In figs. 4 and 3, also, it will be seen that the number of diseased fibres has most perceptibly diminished, and that more particularly in the centre of each patch, where small areas occupied by healthy fibres may be perceived. In fig. 2, which represents a section of the medulla immediately above the point of the fourth ventricle, the posterior median columns having as usual become greatly increased in size, still show a few granulation-corpuscles chiefly scattered through their most superficial portions. The diseased fibres have evidently much decreased in number in this situation, and further than this I have not traced them. In looking now to the outer pair of patches of degeneration (*b*, *b'*), we find them, in figs. 5, 4, and 3, evidently diminishing in extent and intensity; we find also that they occupy that portion of the posterior column which in the medulla oblongata goes by the name of *processus cuneatus*, and we see, by fig. 3, that they are situated immediately on the inner side and behind the grey cornua of this same fasciculus. Tracing them onwards in fig. 2, we find them pushed further away to the side, owing to the development of the posterior median columns, though still occupying the same position with regard to the above-named

grey cornua. In fig. 1 the diseased fibres are more scattered; they are now completely in the lateral region of the medulla, forming part of the *corpora restiformia*, and in these bodies they doubtless proceeded onwards, together with the atrophied fibres of the outer and posterior parts of the lateral columns (*c*, *c'*), into the cerebellum, though I have myself not followed them further than the situation represented in fig. 1, as the parts above were not preserved.

A small area of degeneration on the right side marked *d* may be traced through figs. 5, 4, 3 and 2, Plate IX; further than this it could not be traced, though the fibres composing it may have passed also into the right corpus restiforme, and gone to form part of those occupying the area marked *b'* in fig. 1.

As I am unable myself to say anything concerning the anatomical relationships of the fibres entering into the formation of these tracts of degeneration in the posterior columns, I cannot do better than quote from Bouchard's memoir what seems to bear directly on the subject. Speaking of the fibres which come from the lower half of the cord, he says :—"Ces fibres qui se prolongent jusqu'à la partie supérieure de la région cervicale sont toutes logées, dans l'epaisseur des faisceaux greles et des pyramides postérieures." It would seem almost certain, therefore, that many of those which form the tracts of degeneration marked *a*, *a'* in my figures must be of this kind. He then adds :—"Les fibres qui naissent de la moitié supérieure de la moelle ne paraissent pas se mélanger aux precedentes, de sorte que les faisceaux sensitifs du membre inférieur et ceux du membre supérieur resteraient, isolés les uns des autres, séparés par les sillons intermédiaires posterieurs. En effet, dans un cas de compression de la moelle à la partie supérieure de la region dorsale, L. Türck a vu la dégénération occuper la partie externe des cordons posterieurs. Malheureusement il n'a pas fait de coupes dans l'épaisseur de bulbe ni de la protubérance, de telle sorte que la démonstration anatomo-pathologique de la continuation d'une partie des cordons postérieurs à travers les corps restiformes fait complètement défauts." The fibres entering into

the tract marked *b, b'* in my case seem to correspond to some of those last mentioned by Bouchard, whether or not it be correct that they have their origin in the upper part of the dorsal region. In the present case, also, they have been fairly traced upwards through the lower part of the medulla, into the corpora restiformia, thus confirming, in a new way, that anatomical distribution of some of the fibres of the posterior columns which has been previously taught to exist by anatomists.

Having now described the various morbid appearances met with in the spinal cord, I shall give a somewhat more minute account of the actual histological components of the diseased patches. These are of three kinds, viz.: 1. Atrophied nerve-fibres; 2. New connective-tissue elements; 3. Granulation-corpuscles.

Atrophied nerve-fibres can be detected in all parts of the degenerated columns of the cord, and it seems difficult to understand how they escaped the observation of Bouchard and others. According to Bouchard, however, this has been the case. They are most easily detected after a portion of this tissue has been treated with a solution of caustic soda for a short time, then tinted with carmine and mounted in glycerine. The axis cylinder exists surrounded by the shrivelled and folded hyaline sheath of Schwann, the medullary matter having all disappeared (fig. 20 d, Plate XI). The axis cylinders vary much in size, just as they do in the healthy nerves; and it is owing to the fact that more or less of their extremities become stained with the carmine that the atrophied nerve-fibres can be so readily discovered after the tissue has remained for some hours in a carmine solution. Here and there also in the midst of the diseased tissue we meet with nerve-fibres in a healthy condition (fig. 19 and fig. 20, *e*, Plate XI).

New *connective-tissue elements* exist in the greatest abundance in the sclerosed columns. Its various nuclei and cells may also be best seen after treatment with soda, carmine, and glycerine. This large quantity of connective tissue is derived from the hypertrophy of the neuroglia or normal ele-

ments of this kind which enter into the formation of the cord. In the healthy organ it forms a very delicate fibrous framework, in whose trabeculæ—gradually becoming finer as we approach the central grey matter of the cord—the nerve-fibres are lodged. Fig. 18, Plate XI, represents the normal appearance of a portion of one of the anterior columns of the cord, and shows the delicate nature of the reticulum of connective tissue; whilst fig. 19 shows the appearance of one of the diseased anterior columns magnified to the same extent. The right half of this figure exhibits the appearance presented by a very thin section when mounted in glycerine; the most striking feature being the number of large rounded or ovoidal granulation-corpuscles, each surrounded and enclosed by a rim of connective tissue containing an abundance of nuclei. The left half of the figure represents the appearance of a similar thin section mounted in Canada balsam after saturation with turpentine: here the granulation-corpuscles, being made up of molecular fat, have been dissolved, and the fibrous alveolæ in which they were lodged are fully displayed. The two methods of investigation are, therefore, most valuable. Here and there in the midst of the diseased tissue a healthy nerve-fibre may be seen. Such fibres, either having escaped rupture by the original lesion, or having issued from the grey matter below its level, still remain entire, and in connection with their nutritive cells. When portions of the diseased tracts after tinting with carmine have been teased with needles, and are then submitted to a high magnifying power, the connective tissue is found to be made up of the finest and most delicate fibres, closely beset with more or less spherical nuclei varying in size from that of a small granular speck up to $\frac{1}{2000}''$ diameter. Some of these elements have been represented in fig. 20, Plate XI, where in addition to the fine fibres which seem to have dot-like nuclei attached to them at intervals, other somewhat coarser fibres are seen in connection with more faintly tinted, flatter cells (*c*, *c*, *c*); these vary in size and shape, and seem to be connected also (as at *c'*) with the finer fibres and dot-like nuclei. These cells contain a few rather large granules in their interior, but no distinct

nucleus. That marked *c''* strongly resembles a multipolar nerve-cell, with the exception that it contains no nucleus. There can be no question, however, that it is precisely similar in nature to the others marked *c*, *c*, *c*, and situated as they all were in the midst of the anterior white columns of the cord, it seems only possible for us to look upon them as connective-tissue elements. The large nuclei were apparently unconnected with fibres, and all intermediate sizes could be traced between them and the small dot-like forms. They existed in the greatest abundance, and seemed to represent only different ages of one and the same element. All alike became deeply stained with carmine, whilst the other description of cells of which I have spoken were only tinted of a delicate rose colour.

Granulation-corpuscles seem always destined to be misunderstood. They were formerly brought prominently forward by Gluge, who looked upon them as the products of an inflammatory process; and it was in great part owing to the prevalent reception of this doctrine that ordinary softenings of the brain, in which such corpuscles invariably occur, have been so long and erroneously regarded as necessarily inflammatory in nature. The real facts are, however, as Virchow and other pathologists have lately insisted, that such elements may be met with in any place where cells exist which are gradually losing their vitality. Almost all tissues that are falling into decay, therefore, whether physiological or pathological, may exhibit such structural elements; and whether we meet with them in the brain or spinal cord, in the lungs from degeneration of its epithelium, in the kidney from changes in the same element, or in cancerous or other cellular tumours—in all cases we may safely assume that they are formed from cells whose vitality is gone, or fast going—from cells which are gradually undergoing a process of retrograde fatty metamorphosis, preliminary to a complete molecular disintegration. I think there is no evidence whatever in proof of the assumption that such bodies originate by the gradual aggregation of molecules originally separate, and I should scarcely have considered it necesssary to make these observations now had not Bouchard, in his valuable memoir,

assigned two modes of origin to the granulation-corpuscles which are so abundant in secondary degeneration of the spinal cord, both of which are, I believe, alike untenable. He seems to think that, as a rule, they are formed by the aggregation of fat-granules resulting from the molecular disintegration of the myeline of the nerve-tubes; though he suggests that some of them may also result from "la transformation granulo-graisseuse de gouttes de myéline."[1] Not to speak of the undoubted process by which bodies of this kind originate in other organs, and therefore the probability that they are produced by a similar process in this, we consider that a microscopical examination of the elements in question sufficiently disproves the theories of Bouchard, inasmuch as around most of the smaller granulation-corpuscles a very thin envelope may be detected—the original cell-wall greatly distended; whilst after tinting with carmine we can distinguish with the greatest ease, in the interior of each, and more or less covered with granules, a large spherical or ovoid nucleus, very similar to those which are found so abundantly in the free condition amidst the connective tissue fibres. This appearance I have represented in fig. 20, *a* (Plate XI), and from it I think we are almost bound to conclude that these bodies result from the fatty degeneration and repletion of nucleated cells. From what precise cells they originate, however, does not seem certain, and in the cord which I have examined the process of their formation could not be traced. It represented too late a stage of the degeneration, and in it these corpuscles were all fully formed. I should fancy from the character of the contained nucleus that the cell must have been developed around one of the original free spherical nuclei, which, perhaps, soon began to undergo a process of fatty degeneration. This, however, must be left a matter of doubt for the present.

Vessels.—In this as in other species of degeneration of nerve-tissue, the capillaries and small vessels assume what has been called an atheromatous appearance. The actual change, however, is not one of atheroma, neither is it

[1] Loc. cit., p. 284.

situated in the walls of the vessels at all. The appearance results from an accumulation of fatty elements around the vessel, though within its so-called lymphatic sheath. This Bouchard also imagined to be the real condition, although he did not feel quite sure upon the subject.[1] I, however, ascertained by an examination of some of the vessels taken from the cord in its fresh condition, that they were closely enveloped and their sheaths distended by fattily degenerated nuclei.[2] The nuclei were doubtless originally produced by a proliferation of those lining the inner surface of the perivascular sheath, and when in a state of fatty degeneration they almost exactly resemble some kinds of small pus-corpuscles. Whilst these exist in notably increased abundance within the sheaths of the vessels, many of the nuclei which also exist in the outer substance of the lymphatic sheath are much enlarged, and filled with fat-particles.

The nerve-cells in the uninjured grey matter throughout the cord presented a normal appearance. In no part of its extent were atrophied or pigmentarily degenerated nerve-cells met with.

In estimating the probable course and sequence of the changes met with in this degeneration it would seem to be as follows: the primary or initial changes commence in the nerve-fibres, and the others are more or less direct consequences of these. Thus, when a number of nerve-fibres have been severed from their nutritive ganglionic centres, and the process of degeneration of the white matter which has been described is in progress (whilst no obstruction or mechanical impediment to the flux of blood through the part exists), it seems evident that a redistribution of the nutritive pabulum amongst the elements of the tissues becomes inevitable. For, the nerve-fibres which form such a large part of the bulk of

[1] Loc. cit., p. 287.

[2] It seems to me almost certain that many of the smaller areas of granular degeneration, which have been described by Lockhart Clarke in his examinations of the cord in cases of tetanus and other morbid states, are in reality produced by this same kind of distension of the lymphatic sheath around the vessels.

the tissue, are no longer in a condition of nutritive activity, they do not select fresh material from the blood, and consequently a much larger share of pabulum is at the disposal of the intervening connective tissue. Being supplied with an excess of nutritive fluid, and, as the nerve-fibres atrophy, having more space at its disposal, it is not to be wondered at that hypertrophy of the connective tissue follows. The lymphatic sheaths of the blood-vessels have also an increase of nutritive fluid at their disposal, and this may be the first stimulus leading to the increased proliferation from their lining nuclei which has been spoken of. Their subsequent fatty degeneration, and also that of some of the newly formed connective tissue-cells, leading to the production of granulation-corpuscles, may be partly due to an instability of constitution in the elements themselves (owing to their rapid and irregular formation), and partly, as suggested by Bouchard, owing to the great abundance around them of the products of the retrograde metamorphosis of the nerve-tubes, which may still further disorder their nutrition. That these causes may be instrumental in bringing about the fatty change seems probable, from the fact that after a time the loaded condition of the vascular sheaths gradually diminishes. Granulation-corpuscles also after a time seem no longer to be produced; though when once formed of course they take some time to disappear, as this can only be brought about by molecular disintegration and absorption. These bodies are, therefore, to be found for a considerable time after the commencement of the process of degeneration, but it is now well known to those who have studied the subject that after some months the number of these bodies to be found in the diseased tracts gradually diminishes, in proportion to the length of time from their first formation; so that after the lapse of eighteen months or two years they may have all disappeared, leaving in the now shrunken tracts only atrophied nerve-fibres, and greatly hypertrophied connective tissue.

From what has been said it will be seen how nearly allied the process of cerebral or spinal softening is to that of these secondary degenerations. The regressive changes in the

nerve-tubes, the production of granulation-corpuscles, and the so-called atheromatous condition of the vessels, is the same in both cases, and in both alike the termination is in a development of new consecutive tissue.[1] In the case of secondary degenerations the change is initiated by an inability of the divided nerve-tubes to carry on their own nutrition—the pabulum being at hand they are unable to appropriate it; whilst in most cases of cerebral *ramollissement* the series of changes is more rapid, owing to their being brought about by some diminution of the proper nutritive supply either from disease or obstruction of the vessels belonging to the part.

Comments on symptoms, &c.—From what has been made known of the condition of the spinal cord we find that, as was actually the case during life, there was no reason for expecting any impairment of *sensation* in the paralysed parts. Sensory impressions are conveyed to the brain principally, if not entirely, through the central grey matter, and it has been abundantly proved that such local lesions of the grey matter, as I have shown to exist in this case on the right side, are by no means sufficient to stop the transmission of sensory impressions; at the most they could only bring about a degree of retardation in the rapidity of their transmission. There was in this case imperfect *paralysis* of the right arm and of both lower extremities, and the examination of the cord has shown such important lesions in the upper and middle parts of the cervical enlargement on its right side, as fully to account for this paralysis of the right arm, whilst the degeneration of the anterior columns and of the posterior parts of the lateral columns showed that the encephalic fibres in these situations were functionally inert; and inasmuch as this condition of degeneration extended downwards into the lower part of the lumbar enlargement (although gradually diminishing in extent), we have an explanation also of the partial paralysis of the lower extremities.

[1] Reynolds's 'System of Medicine,' vol. ii, article "Non-Inflammatory Softening."

Although we have no clinical records of a difference in the amount of power possessed over the two extremities, there is reason to believe from the much greater extent of the degeneration in the posterior parts of the right lateral column that the capability of voluntary movement must have been less on this side than on the left. The *startings* of the limbs—more especially of the right lower extremity—which was complained of after the first ten days and for some time subsequently, was doubtless to be attributed to the irritation of healthy nerve-fibres proceeding to the limbs, owing to the reparatory process at the seat of the original lesions in the cervical region. The secondary degenerations which must have been making progress at that time in the anterior and lateral columns, could not of themselves cause any symptoms, since the mere fact of the degeneration taking place in them was of itself evidence that they were functionally inert and cut off from their physiological and nutritive centres. The state of *rigid contraction* which the muscles of the right arm subsequently assumed, appears to have commenced about two months after the accident; beginning almost imperceptibly and gradually increasing, as it is stated to do by Bouchard.[1] It is certainly a question of some difficulty to ascertain the exact cause of this late rigidity coming on in paralysed parts. Some hold that it is entirely due to changes in the paralysed muscles themselves; others, such as the late Dr. Todd, ascribe to it a cerebral origin, and believe it to result from the irritation produced by the contraction and cicatrization of a brain lesion. Bouchard, however, is not a believer in either of these explanations, and thinks that in all cases, whether the paralysis be of cerebral or of spinal origin, the cause of late rigidity in the paralysed muscles is to be ascribed to changes taking place in the spinal cord itself. He believes that now we know of the invariable existence of secondary degeneration of the spinal cord in cases of apoplexy depending upon lesions of the corpora striata, optic thalami, or pons, we need no longer look for the cause of this late rigidity in the brain itself; but that it is explicable in the

[1] Loc. cit., p. 292.

same way as in cases of paralysis due to original lesions of the spinal cord, from a consideration of the natural progress of the secondary degenerations in this organ. He thinks, in fact, that this important symptom is due to an irritation of adjacent healthy fibres, when the new growth of connective tissue taking place in the diseased tracts, comes to press upon or encroach amongst the healthy fibres. The suggestion is ingenious and deserving of consideration, though there seem difficulties in the way of its acceptance. Time will not permit of my discussing these questions, however, and I must pass on to the next symptom—that of *pain* in the paralysed limbs. Although only slightly mentioned in the clinical record, I call attention to it now, since MM. Charcot and Cornil[1] have lately made some investigations as to the cause and treatment of this, which is oftentimes a most distressing symptom. They believe it to be due to a kind of hypertrophic neuritis in the affected limb, since they have found the nerves increased in volume, more vascular than natural, and having a notable increase in the thickness of their connective tissue envelopes. Whether this change is due to mere functional inertia or is more directly dependent upon the secondary degenerations of the cord is not known. It seems, however, to have an evident relation to the pains above mentioned, and, according to M. Charcot, these are not only increased by pressure along the course of the nerves, but are often soothed by the application of a blister in this situation.

The *respiration* was described as being diaphragmatic when the patient was admitted into the hospital, but it seems probable, from the condition of the cord, as well as from the length of time that the man lived without fatal engorgement of his lungs, that there was by no means complete paralysis of the thoracic and abdominal muscles of respiration. Their action was doubtless much impaired, and hence amongst other things the extreme difficulty the man had in expectorating the mucus with which his bronchial tubes were loaded for some time before his death. Fortunately, the lesions of

[1] 'Compt. Rend. de la Soc. de Biolog.,' 1863.

the grey matter did not extend quite so high as the origins of the phrenic nerves, and the diaphragm was therefore unaffected. Had it been otherwise—even though one side only were affected—death would, in all probability, have been very rapid.

It now only remains for me to say a few words concerning the *general muscular atrophy*, which commenced about two months after the accident, and which in the remaining four months of the man's life had reduced him almost to a skeleton. This atrophy following paralysis has been frequently noticed, and not unfrequently it has been confounded with 'progressive muscular atrophy,' which should, however, be regarded as a distinct disease. Unfortunately, having my attention so much attracted to the spinal cord at the time of the post-mortem, I altogether omitted to make any special examination of the atrophied muscles, though I did take out and put into chromic acid for subsequent examination the great semilunar ganglia of the sympathetic system. A careful inspection of them and a comparison with others removed from patients dying of different diseases, enables me to say that these ganglia were undoubtedly atrophied: they were scarcely as much as one third of their usual size, and whilst all other parts of the body were remarkable for the almost total absence of fat, on making thin sections of these bodies and then placing them under the microscope, certainly a larger proportion of thin fluid fat was seen than is usually met with in such sections. The ganglion-cells seemed to contain rather more than their usual amount of pigment; they were more highly refractive also than in other sections with which I compared them, and the nucleus and nucleolus, which are usually so apparent in these cells, could scarcely be distinguished in one out of twenty of the ganglion-cells, in sections of portions of the sympathetic belonging to our patient. These were the only abnormal conditions that I was able to detect in the semilunar ganglia, and other parts of the sympathetic system were not examined.

These changes met with in a limited part of the sympathetic system, although not very decided, will, I hope, be

sufficient to attract increased attention to the condition of the sympathetic system in other cases of paralysis followed by muscular atrophy. In addition to the muscular atrophy in this case, there were other conditions which might have been dependent upon a disease of the sympathetic system; since the post-mortem examination revealed an abnormal condition of the liver, apparently due to fatty degeneration, tubercle in the lungs, and disease of the kidneys; whilst during life there was constant vomiting. If the muscular atrophy and degeneration of viscera were in reality due to a morbid condition of the sympathetic in this case, we should have to look upon this as secondarily affected, and as a result of the primary disease of the spinal cord; whilst in progressive muscular atrophy, M. Jules Simon, a late able writer on the subject,[1] maintains that some abnormal condition of the sympathetic system is the starting-point of the disease, which secondarily affects the muscular system and some of the anterior roots of the spinal nerves. In this view he is supported more or less entirely by Dumenil, Schneevoogt, Remak, Bärwinkel, Jaccond, and Professor Trousseau; and MM. Schneevoogt,[2] and Jaccoud[3] have published most important cases in which unmistakeable fatty degeneration of the sympathetic system was met with. It is only fair to add, however, that some still adhere to Cruveilhier's doctrine of the dependence of this disease upon a disease of the anterior roots of the spinal nerves; whilst others, such as Duchenne, Virchow, and Aran believe the disease to be a primary one of the muscles themselves.

I may say, in conclusion, that the prognosis does not seem quite hopeless in cases of secondary degeneration of the spinal cord, even after the supervention of late rigidity in the paralysed muscles, since M. Bouchard has seen a cure result in five such cases.[4] These were all cases of complete

[1] 'Nouv. Dict. de Méd. et de Chirurg.,' vol. iv, 1866, article "Atrophie Musculaire Progressive."

[2] Nederlandisch, 'Lancet,' 1854, and Schmidt's 'Jahrb.,' 1855.

[3] 'Mém. de la Soc. Méd. des Hôpitaux,' November, 1864.

[4] Loc. cit., p. 297.

paraplegia, from compression of the cord due to Potts' curvature. He says: "Dans 4 cas la sensibilite et le mouvement ont reparu avec touté leur intégrité; dans 1 seul, les mouvements, sans avoir recouvré leur entière liberté, permettent cépendant à la malade de marcher. Dans ce cas la paraplégie était flasque; dans les autres elle s'accompagnait de contracture." "On peut donc, en conclure que les tubes nerveux de la moelle peuvent se régénérer comme ceux des nerfs périphériques, non seulement chez l'enfant, mais encore chez l'adulte et lors même que les faisceaux dégénérés, ont été déjà le siége d'un travail d' hypergénèse des éléments nucléaires."

If what I have already stated concerning the persistence in the degenerated tracts of the axis cylinders of the nerves be borne in mind, it seems probable that the repair in these cases is brought about in the same manner as when it occurs in peripheral nerves. Here MM. Phillipeaux and Vulpian have convinced themselves that the restoration of function is due to the reproduction of myeline around the persistent axis cylinders of the nerve-fibres rather than to the production of entirely new fibres, as it was formerly imagined. Notwithstanding the evidence afforded by the recovery of the patients above mentioned, and his knowledge of the manner in which restoration of function was brought about in divided nerves, Bouchard imagined that the atrophied nerve-fibres entirely disappeared in the spinal cord; but my statement that the axis cylinders of the fibres are easily recognisable in the disease columns, though resting upon evidence indubitable to myself, receives additional confirmation from these clinical facts.

DESCRIPTION OF PLATES IX, X, AND XI.[1]

PLATE IX.

Fig. 6.—Transverse section through upper cervical part of spinal cord ($2\frac{1}{3}$″ below point of fourth ventricle), showing two pairs of almost symmetrical and well-defined areas of degeneration in posterior columns of cord (a, a' and b, b'), and also unequal areas of degeneration in the lateral columns (c, c'), In addition there is a very slight patch of degeneration in each anterior column near the points of exit of anterior nerves.

Fig. 5.—Section $1\frac{1}{4}$″ above last, which is on a level with the lower boundary of the medulla oblongata and of the decussation of the pyramids. Letters of reference same as in last figure, pointing to more limited areas of degeneration, with the addition of d, another small patch situated just beneath the neck of the right "grey tubercle of Rolando."

Fig. 4.—Section through medulla $\frac{1}{2}$″ above last and $\frac{7}{12}$″ below point of fourth ventricle. Letters of reference same as in last figure. The median patches of degeneration marked a, a' have become wider, and now contain some healthy fibres in their midst. They are seen very distinctly, by this figure and the next, to occupy the posterior median columns of the cord.

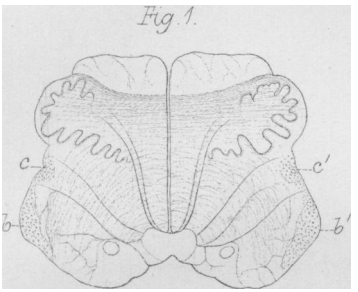
Fig. 3.—Section through medulla $\frac{1}{3}$″ above last, and $\frac{1}{4}$″ below the point of the fourth ventricle. Decussation of pyramids not represented in this or other figures. Letters of reference same as in Fig. 5, pointing to gradually waning areas of degeneration. The single area, d, on right side, is now seen to intervene between the much enlarged "grey tubercle of Rolando" and the cornu of the "processus cuneatus." The areas a, a' have increased in width with the posterior columns, and contain much more of healthy tissue in their midst.

Fig. 2.—Section through medulla just above point of fourth ventricle. The widened posterior median columns in this situation are almost composed of healthy tissue, and show only a very slight cloudiness externally. The areas b, b' and c, c' are more limited and much less obvious, and the same is the case with the area d, on the outer side of the cornu of the processus cuneatus.

Fig. 1.—Section of medulla higher up through the middle of olivary bodies. The areas a, a' have disappeared with the posterior median columns; the area d has also disappeared; whilst the areas b, b', now occupying the restiform bodies, have become rather lateral than posterior; they are much fainter and more diffuse. The areas c, c', in the anterior borders of the restiform bodies, have also nearly disappeared.

[1] Each of the sections through the medulla oblongata and the spinal cord is represented twice its natural size.

Fig. 1.



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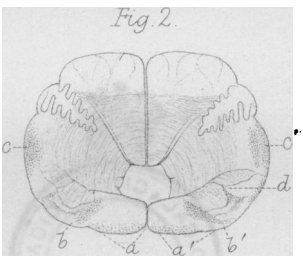

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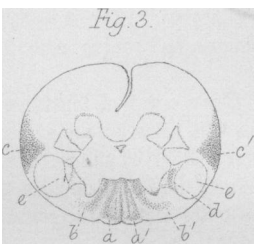
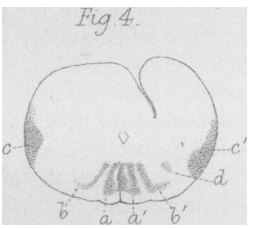


Fig. 4.



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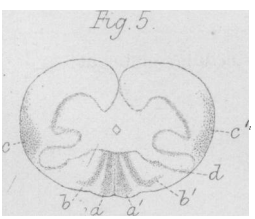

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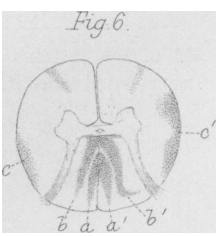

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Plate X.

Fig. 7.—Section through upper part of cervical enlargement, showing a large oblique rupture through the grey matter and a part of the right anterior column, with a contraction and want of symmetry in this portion of the cord. The anterior columns on each side, more especially left, have undergone degeneration, and other parts of the cord present granulation-corpuscles either in groups or sparsely scattered. The grey matter on left side is unaffected.

Fig. 8.—Section $\frac{1}{4}''$ below last. Want of symmetry on right side still apparent, though less marked. A smaller rupture having a different direction seen in this section, which is continuous externally with a fibrous cicatrix extending forwards and outwards to surface. Well-marked degeneration in grey matter immediately behind rupture. With the exception of anterior columns other parts of section are freer from degeneration than last.

Fig. 9.—Section $\frac{1}{4}''$ below last. Symmetry almost perfect; no ruptures. Large square patch of diseased tissue in grey matter of right side. Considerable degeneration of fibres around grey matter on this side.

Fig. 10.—Section $\frac{1}{3}''$ below last. Showing a large irregular rupture extending through the whole length of the grey matter of the right side and much degeneration of tissue around it. On the left side also there is an increase in the number of granulation-corpuscles and a very slight solution of continuity in the grey matter.

Fig. 11.—Section $\frac{1}{3}''$ below last. Grey matter of right side now entire and pretty healthy. Much less degeneration of white columns also on this side, and (leaving out of consideration inner parts of anterior columns which are much the same as in last sections) what there is occupies principally a definite part of the lateral column. Two very slight ruptures in grey matter on left side, and a very slight amount of degeneration also in posterior part of left lateral column.

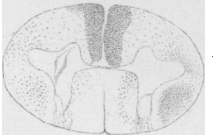
Fig. 12.—Section just below cervical enlargement, $\frac{2}{3}''$ below last. Showing alteration of shape of areas of degeneration in anterior column, a well-marked area in the posterior part of right lateral column, and a much fainter one on the left side. No lesions of grey matter, and other portions of section healthy, with the exception of a very slight amount of degeneration in the anterior part of right lateral column.

Fig. 13.—Section through mid-dorsal portion of cord, showing still further alteration in shape of anterior areas of degeneration, with evident approximation of the right posterior area to the periphery of the cord.

Fig. 14.—Section through the lower dorsal portion of cord, 1″ above the lumbar enlargement, showing another alteration of the shape of anterior areas. Posterior areas on each side now extending to surface of cord.

Fig. 7.

Fig. 11.



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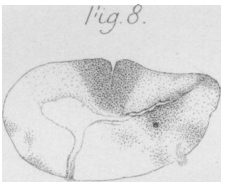

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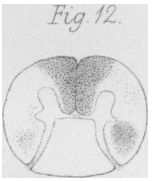

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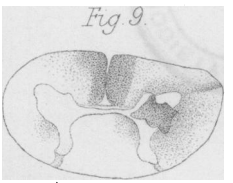

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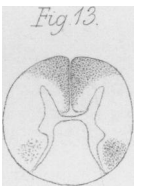

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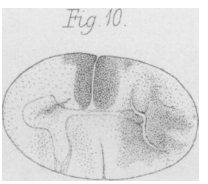

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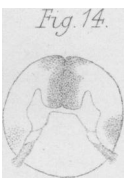

Plate XI.

Figs. 15, 16, 17.—Sections through the upper part of the lumbar enlargement, $\frac{3}{8}''$ below this last, and through the lower third of the lumbar enlargement, or $\frac{3}{4}''$ from the commencement of the *filum terminale*. These show the gradual way in which the areas of degeneration terminate, and therefore the positions occupied by the longest of the degenerated fibres.

Fig. 18.—The appearance presented by a section through a healthy portion of one of the anterior columns of the cord, showing the sections of different sized nerve-fibres with intervening partitions of delicate fibrous tissue. Magnified 150 diameters.

Fig. 19.—The appearance of a section through a diseased portion of one of the diseased anterior columns. The right half of the figure gives the appearance of a thin section when mounted in glycerine, showing the abundance of large granulation-corpuscles separated from one another by richly nucleated connective tissue, whilst the left half shows the appearance of a similar section after immersion in turpentine and Canada balsam, when the granulation-corpuscles have been dissolved out and the fibrous alveoli in which they were situated are more plainly displayed. Here and there also in each half the sections of unaltered nerve-fibres are seen, and in the whole figure the sections of three blood-vessels are shown, whose walls are considerably thickened. Magnified 150 diameters.

Fig. 20.—Highly magnified representation of the different kinds of elements met with in the secondarily degenerated columns, as they appear after tinting with carmine:—*a, a, a,* granulation-corpuscles of different sizes, each having in its interior a well-marked nucleus; *b, b, b,* free spherical or ovoidal nuclei, such as exist in the greatest abundance, and of all sizes; *c, c, c,* branched cells of various shapes and sizes, which become much more faintly tinted with carmine than the preceding nuclei; *c'* shows the apparent connection between these two kinds of elements such as may be seen occasionally; *c''*, one of the largest and most extreme forms of these cells, closely resembling a nerve-cell; *d*, one of atrophied nerve-fibres consisting only of the axis cylinder and the delicate sheath of Schwann, which is closely wrapped round it; *e*, one of healthy nerve-fibres from midst of diseased tract. Magnified 400 diameters.

Fig. 21.—Appearance presented by one of the vessels just outside the large area of degeneration in the grey matter represented in Fig. 9. It is rather the sheath of the vessel which is seen loaded with amorphous blood-pigment of a dark yellowish olive colour and marking the site of a previous effusion of blood.

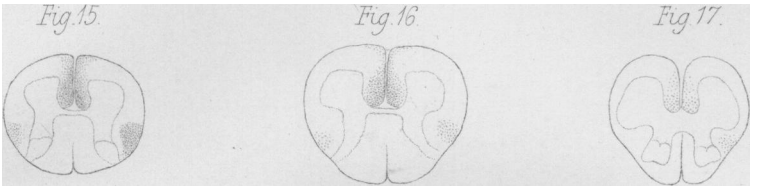
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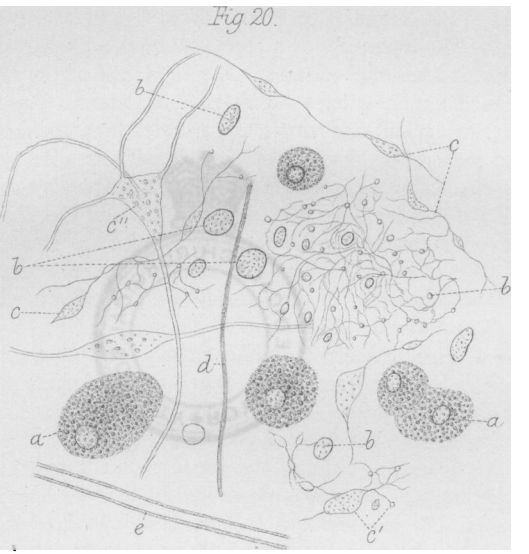
Fig. 16.
Fig. 17.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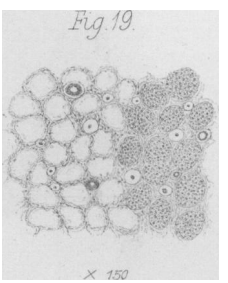
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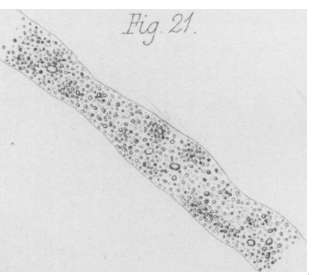
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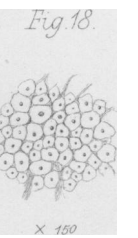
Fig. 18.
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