ON NERVOUS SYMPTOMS AND MORBID CHANGES IN THE SPINAL CORD

IN

CERTAIN CASES OF PROFOUND ANÆMIA.

BY

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IN 1887, Professor Lichtheim described at the Congress für innere Medicin¹ in a paper "Ueber Pathologie und Therapie der perniciösen Anämie" three cases which presented the usual clinical symptoms of that disease. But in addition to these symptoms all of them had also signs pointing to an affection of the spinal cord. In the first case these signs were rigidity, weakness, and ataxy in the legs, with paræsthesiæ. The knee-jerks were present but diminished, and the pupils were normal. The second patient had similar symptoms; but in the third the symptoms differed somewhat: there were lancinating

¹ 'Neurologisches Centralblatt,' 1887, p. 236.

pains and the knee-jerk was lost, so that the resemblance clinically, at least as far as spinal symptoms were concerned, was to a case of commencing tabes.

In this last case there was no post-mortem examination. In the other two, however, distinct changes were found in the spinal cords after death. These were almost complete degeneration of the columns of Goll, with similar but slighter affection of the pyramidal tracts. Besides these changes there were small foci of degeneration in other parts of the lateral columns and in the anterior columns. Lichtheim thus early regarded the changes as resulting from a toxic blood condition.

The next communication on this subject was from the same observer, when at Heidelberg¹ in 1889 he described the conditions which his pupil Minnich had found in the spinal cords of patients who had died from pernicious anæmia, but in whom no symptoms of spinal disease were present. In none of the cases examined was the cord absolutely normal. In half the cases there were small sclerotic foci, which on microscopical examination were found to be the result of hæmorrhages. In the other half of the cases there was distinct degeneration in the posterior columns, differing in intensity in the different cords. The degeneration was not restricted to the posterior columns, but was most evident in these. In all of them Clarke's column, Lissauer's tract, and the posterior roots were absolutely normal. The view was again expressed that the condition was the result of a toxic blood-state, and the changes resulting in the nervous system from another blood-state, namely, diabetes, were referred to as analogous. The analogy has been further strengthened by the more recent researches of Williamson, who found degeneration in the posterior columns of the cord in three cases of diabetes mellitus.²

A further very exhaustive examination of Lichtheim's cases was carried out by Minnich, and to those previously

¹ 'Neur. Cent.,' 1889, p. 662.

² 'Brit. Med. Journ.,' 1894, i, p. 398.

described other cases were added.¹ Six cases were described, in all of which post-mortem examination was obtained. In all of them profound anæmia was present, with retinal hæmorrhages and motor weakness, sometimes in all limbs, sometimes only in the lower. In two the knee-jerk was exaggerated, in one it was described as present, in another diminished, and in two absent. In one in which the knee-jerk was absent there was no reaction in the pupils to light, but they reacted during accommodation. One exhibited no marked signs of spinal affection. Of the others, all except one showed ataxy and complained of subjective sensations, while most had actual sensory impairment in the limbs, and weakness of the bladder. Lightning pains do not seem to have been present in any.

The spinal cords in all the cases showed similar degeneration, chiefly marked in the posterior columns and in one confined to them. In the others there was also degeneration in the lateral columns, and in two also in the anterior columns; while in all the cases the posterior roots, the grey matter and the peripheral nerves were described as free from changes.

Minnich² supplemented his work by an examination of the cords in five cases of pernicious anæmia, the subjects of which had not developed symptoms of affection of the cord before death. The examination was undertaken in the hope that the commencing changes in the spinal cord might be recognised. Two sets of changes were found. The first were capillary hæmorrhages with apparently miliary sclerosis resulting from them. The second set of changes was present in three cases, and consisted in slighter coloration of the posterior columns throughout the cord after hardening with bichromate salts, and to some degree of the anterior and lateral columns in certain parts. The cord as a whole was brittle, possibly as a result of its cedematous condition.

¹ 'Zeitschr. f. klin. Med.,' xxi, 1892.

² Ibid., xxii, 1893.

Similar changes were found in three cases of chronic jaundice, in one case of leukæmia, and in one of tumour of the inferior vermis of the cerebellum, that is, in the cords of patients who had become much reduced in consequence of prolonged illness. The author regarded it as not impossible that dropsical changes in consequence of some illness might be the starting-point of a true degenerative affection of the spinal cord.

Van Noorden¹ next described a case in which the prominent symptoms were anæmia, paræsthesiæ in the hands and feet, impaired sensibility in the legs, motor weakness in the arms and legs, with spontaneous jerking in the former, and finally complete inability to walk. The knee-jerks were lost. In the cord there is said to have been complete degeneration of the posterior columns except of a part next the posterior roots, and also in the lateral columns of the cord. The posterior roots were free from change, and there was no shrinking of the cord, such as occurs in cases of tabes.

Eisenlohr² also had a patient, a man of fifty-nine, suffering from pernicious anæmia, who developed motor weakness and paræsthesiæ with, later, loss of control over the sphincters. There was no obvious ataxy and no pupil change, but the knee-jerks were lost. He found degeneration in the posterior columns of the cord, and scattered foci also in the lateral columns.

Two cases of this affection have also been published by Nonne.³ The first was that of a man of forty-eight, suffering from pernicious anæmia, who developed nervous symptoms, numbness and weakness of the lower extremities, ataxy, girdle sensation, and loss of control over the sphincters. There was no pupil change, and the condition of the knee-jerk varied, being at first diminished, then lost, and later again distinctly present. In the second patient, a man of fifty-seven, uncertainty in

¹ 'Charité Annalen,' 1891.

² 'Deut. med. Wochenschr.,' 1892.

³ 'Archiv f. Psych.,' xxv, p. 2, 1893.

walking, occasional girdle sensation, and numbness in the hands and feet had been present for some months before the anæmia was obvious. When this became profound the motor weakness increased, but ataxy was no longer present. There was incontinence of urine, and the knee-jerks, which previously had been diminished, were now increased. The pupil reflex was not affected.

The cords in both cases showed changes in the posterior columns especially, but also in the lateral and anterior. The areas in these two latter columns seemed to be formed to some extent at least by the coalescence of foci of comparatively recent degeneration. The area of the posterior column contiguous to the posterior roots, the posterior roots themselves, and Lissauer's tract were unaffected.

A very interesting case was recently published by Dr. Bowman,¹ being that of a patient who was under the care of Dr. Hughlings Jackson. The patient was a woman of fifty-three, and on admission to hospital she was found to have great weakness but no actual paralysis, and no change in the reflexes. She was very anæmic. She improved very much, and was able after three months' treatment with arsenic to leave the hospital and to go to a convalescent home. She was, however, readmitted after nine months, and she then had been unable to walk for a month, and there had been occasional incontinence of urine. There was great weakness and ataxy in the arms, while the legs were rigid and semi-flexed, and spasmodic jerkings in them were painful and frequent. Sensibility in the lower limbs was also impaired. The knee-jerks were active and the pupil reflex was normal. There was marked anæmia; and this gradually became more and more profound, the ataxy in the arms increased, the legs became more rigid and drawn up, and the anæsthesia on them deepened. Numerous retinal hæmorrhages developed, the patient passed into a delirious condition, and she gradually sank and died.

¹ 'Brain,' vol. xvii, 1894, p. 201.

In the cord, changes were found similar to those described in the previous cases referred to, namely, marked degeneration in the posterior columns, with extensive changes as well in the lateral and anterior. The posterior roots and the grey matter were normal, but the blood-vessels in the most degenerated parts were much altered, the walls of the arterioles being greatly thickened and their lumen much reduced—in some instances occluded.

Such are the cases which in the course of my work on this subject have come under my notice as cases in which spinal symptoms and anæmia of the type known as pernicious were recognised as associated. In all the cases I have mentioned a strikingly similar condition of the spinal cord was present. But I have also met in the course of my investigations with certain other facts which show that a similar condition of the cord has been noticed in other cases, whilst its close association with, perhaps dependence on, the anæmia which was also present, had not been recognised. It is mentioned by Nonne, in the paper to which I have referred, that Leichtenstern described in 1884 two cases of what he called *tabes* associated with anæmia. In neither case was there any history or suspicion of syphilis, and no gastric or other crises. In 1891 Dr. James Putnam, of Boston,¹ described “a group of cases of system sclerosis of the spinal cord, associated with diffuse collateral degeneration occurring in enfeebled persons past middle life, and especially in women.” Of such cases he had seen eight, and in four instances had been able to obtain post-mortem examinations. The symptoms generally consisted in a progressive impairment of both sensory and motor functions of all four extremities, associated after a time with general wasting of muscles and emaciation. The fatal cases ran their course in about two years, in one case four years. In three cases marked inco-ordination of movement was present, and in one lancinating pains. In three the knee-jerk was exaggerated and ankle-clonus was present; in the other case

¹ ‘Journal of Nervous and Mental Diseases,’ February, 1891.

the state of this reflex is not mentioned. Of the eight fatal cases, six were women past middle life, and almost all were in a condition of considerable debility. In several, obstinate diarrhoea was a prominent symptom. Several of the patients are also said to have had lead in the urine. Anatomically morbid changes were found in both motor and sensory areas of the cord. In one the medulla was examined and found normal. In the white columns two sets of changes were present, both degenerative,—one of old standing, the other recent and more acute, chiefly around the older degenerated areas. In the grey matter there was disintegration of nerve-cells. Unequivocal signs of acute inflammation were absent. Slight thickening of the pia was present in three cords, and change in the peripheral nerves was found in the one case in which they were examined.

Dr. Putnam also gives a short clinical account of four other cases in which similar symptoms were present. The illness usually began with paræsthesiæ of the extremities, weakness and inco-ordination usually followed, and finally paraplegia and death. In all there was great weakness with emaciation, and in some at least profound anæmia. In two the state of the knee-jerks is not noted, in the other two it was absent.

I think there can be little doubt that these cases described by Dr. Putnam are to be placed in the same category as those to which I have alluded. The gradual onset of symptoms—weakness and paraplegia, the association of those with, in all cases presumably, and in some certainly, a greater or less degree of anæmia, in some profound anæmia, the gradual increase in the weakness leading to complete paraplegia, the variability in the reflexes, in some exaggerated, in some diminished or absent, characterise the histories and clinical condition in both classes of cases. And the actual post-mortem findings, if not identical, present at least very striking resemblances. There is the same wide-spread sclerosis; the same parts of the cord are picked out; there is the same tendency

to limitation to the similarly situated comparatively small areas in the lumbar region. The changes described in the grey matter in most of Dr. Putnam's cases, however, are not present in the others I have referred to ; but, on the other hand, in at least one of these the changes were very slight.

Professor Sir T. Grainger Stewart described¹ the case of a patient who had spent most of his life in New Zealand. He had lived freely, but had never been intemperate. In January, 1889, he had noticed some tingling in the tips of the fingers of both hands. This was most marked after exertion, and was aggravated by cold. He also became shaky and unsteady, his knee-jerks were exaggerated, and ankle-clonus was obtained on both sides. Voluntary power was impaired, especially in regard to fine movements ; writing had become difficult ; and there was a certain awkwardness in his gait. There were no vaso-motor or trophic changes, but the patient was thin and anæmic. His psychical condition was unaltered. He became gradually worse, the anæmia deepened, and there was greater difficulty in walking. Sensibility also became diminished. He spent the winter at Mentone, but was confined to his room nearly all the time on account of severe diarrhoea. On his return he was more anæmic, there was diminished sensibility, and the difficulty in getting about was much increased. The knee-jerks were still exaggerated, and ankle-clonus was present.

Sir T. Stewart referred this case to the same category as Dr. Putnam's, and I would venture to think that both it and Dr. Putnam's cases belong to the same group as those I have just described.

Dr. Bulloch² described, under the title "Hyaline Degeneration of the Spinal Cord," a very interesting case, of which the following is a brief summary :—A man of fifty-one, a farm labourer of temperate life, and with nothing of significance in his previous history, was admitted to hos-

¹ 'Brit. Med. Journ.,' June 6th, 1891.

² 'Brain,' 1892, p. 411.

pital complaining of shortness of breath and swelling of the feet and face, which had come on about two months previously. He was well nourished but profoundly anæmic, of lemon-yellow colour, with œdema of both feet and ankles, and also of the face. Examination of the blood revealed great paucity of red corpuscles and change in their form and pigmentation; flame-shaped hæmorrhages were present in both retinæ, but no optic neuritis. He was rather awkward and unsteady when he got up, but this was attributed to the great weakness which was present. He also had occasional delusions. He was treated for the anæmia with Fowler's solution, and improved. While at a convalescent home he was the subject of delusions, but was easily managed, and here gained strength, so that he was able to walk to hospital to report himself on more than one occasion. In the third week of his stay he was observed to be ataxic in his gait. Romberg's symptom was present, and there was paresis of the legs, which increased so much as to necessitate his return to hospital. A day or two after admission the paresis of the legs had become complete paralysis. The limbs were flaccid, and the superficial and deep reflexes were abolished. Weakness and inco-ordination now affected the upper limbs, so that he became unable to feed himself. Sensibility was also diminished, he became apathetic and listless, and passed urine and fæces involuntarily. He had several severe attacks of diarrhœa, he became gradually worse both physically and psychically, bedsores formed, and he died of exhaustion.

No appearances indicative of the profound anæmia are mentioned as being visible in the organs. The liver is described as congested and nutmeg-like in appearance. The spinal cord was softer than usual, and between the third and fifth cervical nerves diffuent and altered in contour. On section here a cavity was seen, which at its lower part was more of the nature of a fissure extending deeply into the substance of the cord, so as almost to divide it into two. After being hardened, the cord was

cut, and sections at different levels showed the presence of what is described as "hyaline matter" in various situations. The pia was thickened, and a layer of this hyaline material was under it, and this had, at different levels, replaced the nervous tissue in various parts of the white columns. In the cervical region it was most abundant in the posterior columns, and was present also in the lateral column. It was also abundant in the grey matter, and no cells could be found. In the dorsal region this degeneration was less extensive, being present only in the posterior columns. The parts of these along the margins of the posterior horns were healthy. No cells could be found in the grey matter. In the lumbar region the change was confined to a small triangular area on each side of the posterior median septum. A few ganglion-cells could be seen in the anterior horns. Below the fourth lumbar nerve no degeneration in the posterior columns was recognisable.

The vessels were thickened and distended with blood-corpuscles, and the septa were enlarged and homogeneous.

This case has also striking similarities to the cases already mentioned. There is the profound anæmia, evidently of the pernicious variety, the symptoms of disease of the spinal cord, and the degeneration in certain areas, similar at least in distribution to that found in Minnich's and the other cases. The chief difference is that in this, as in Putnam's cases, degenerative changes in the cells of the grey matter are described, while in Minnich's and the other no such change was present.

I shall now describe two cases which I have observed, for permission to publish which I am indebted to the kindness of Dr. Hughlings Jackson and Dr. Gowers.

CASE 1.—S. A. P—, æt. 50, married for twenty-two years, laundress, admitted to the National Hospital for the Paralysed and Epileptic, Queen Square, February 12th, 1891, under the care of Dr. Hughlings Jackson, on account of numbness in the legs and difficulty in walking.

The history was that her present illness began with numbness in the forefinger of the left hand twelve months before admission. A month later there was a tingling sensation along the ulnar border of the left hand. The outer side of the left leg became similarly affected, and afterwards the right arm and leg became numb and stiff. She stated that she had also suffered from slight darting pains in the left knee on several occasions about three months before admission. There was no trouble with the bowels, and no vomiting or gastric disturbance. For eleven months she had had difficulty in walking on account of unsteadiness and weakness. There had also been some weakness in the hands, so that she could not write so well as usual. There had been no impairment of vision. During the nine months before her admission her friends had frequently remarked on the brown discoloration of the skin. For the six months immediately before admission she had been troubled with frequency of micturition.

Her health previous to the illness for which she sought advice had been good, with the exception of an attack of what is called "congestion of the liver" four years before admission. She made a good recovery from this. She had two children, aged respectively twenty and twenty-one, both healthy. She had had no miscarriages.

There was nothing important or significant in her family history. Her father died at seventy-two, her mother at sixty,—the latter of some liver disease, it is said, and the former from some unknown cause. She had two brothers, who had both died; the cause of death was not known in either case. She had three sisters healthy. There was no history to be obtained of any family disease.

On admission she looked fairly well nourished, but had a peculiar yellowish-brown colour, which suggested a possibility of Addison's disease. Examination of the flexures at the groin and knee, and also of the axillæ, failed, however, to reveal any abnormal degree of pigmentation in these parts. She walked with difficulty and uncertainty, and the weakness seemed to be greater in the left leg than in the right.

There was unsteadiness in standing, which was distinctly increased on covering the eyes. The knee-jerks were present, that on the right side being more active than usual, while that on the left side could only just be elicited. There was no sensory impairment to be made out, except that the sense of position in regard to the left foot seemed to be defective. The special senses were all normal, and no changes were observed in the optic discs or fundi. There was no albumen or sugar in the urine.

During the next few months the patient became gradually weaker, and by the end of the year was unable to stand. The urine also had become albuminous, and there was retention, necessitating the daily use of the catheter. The knee-jerks were very feeble on both sides.

On January 15th, 1892, it was found that sensibility to touch on the legs was much dulled, especially on the outer sides; incontinence of urine was now present, and neither knee-jerk could be obtained.

On January 24th it was observed that in the left upper limb peculiar spontaneous movements were present, consisting in ulnar flexion at the wrist alternating with relaxation. There were also movements of extension of the proximal phalanges, rhythmical in character, associated with irregular wave-like movements affecting the fingers, not simultaneously, but one after another irregularly. There were also slight movements of extension at the elbow synchronous with those at the wrist, and there seemed to be some movement at the shoulder, although no distinct contraction could be seen in the muscles there. The frequency of the wrist movements was about seventy per minute. They were best seen when the limb was slightly supported at the wrist, and tended to become more irregular when the hand was allowed to lie free.

In the right arm there were also constant movements, not of the same character, but more of the nature of coarse tremor. This hand was also "dropped."

These movements gradually decreased in force and extent, but remained present until the middle of June,

—that is, for about five months. By this time the legs had become extremely flexed at both hips and knees, and voluntary movement was entirely abolished. There was marked sensory impairment in the legs. The movements of the arms now gradually ceased, and those limbs became contracted. The contraction of the legs became extreme, bedsores and cystitis developed, pain was very great, and rendered the use of morphia necessary. The patient became extremely anæmic, gradually sank, and died on the 26th November, 1892.

At the necropsy which was made on the following day the body was found much emaciated, and both arms and legs were contracted to an extreme degree at all the joints, and fixed. As regards the viscera generally, it need only be said that all the organs were extremely pale, and the liver fatty. No change was found in the supra-renal capsules. The cord and brain were preserved; and the former, after being hardened in Müller's fluid, was examined at several different levels. The sections were stained by the Weigert-Pal method, and the following conditions observed. In the cervical region (Fig. 1) there was very extensive sclerosis in the anterior, lateral and posterior tracts of the cord. In the direct pyramidal tract on the right side there was, as the figure shows, a considerable part from which the fibres had entirely disappeared. On the left side, in a corresponding position, there was also a patch of sclerosis, but much smaller in extent. In the lateral regions of the cord the change was as nearly as possible symmetrical, and consisted in an almost complete disappearance of fibres in the region of the direct cerebellar and the crossed pyramidal tracts. On each side there was a small zone of comparatively normal nervous tissue on the outer side of the posterior horn. In the posterior region of the cord also there was a zone of fibres on the inner side of the posterior horns on each side. That on the right was more extensive than that on the left. At each side the fibres were abundant near the horn, gradually fading towards the middle line,

and there was a large area on each side of the posterior median fissure practically free from normal fibres.

In the dorsal region the sclerosis, as shown in the figure (Fig. 2), was similar to that just described, both in degree and extent. Both anterior columns were affected, and in one at least the sclerosis was more extensive than the region of the anterior or direct pyramidal tract. In the lateral and posterior columns the sclerosis was similar to that in the cervical region. Here also, both on the outer and inner side of each posterior horn, there was a small marginal zone of fibres which preserved the hæmatoxylin stain.

In the lumbar region (Fig. 3) the sclerosis was much less extensive, and was confined to the lateral and posterior regions. In the former the degenerated area occupied the position of the crossed pyramidal tract on each side. It was almost symmetrical, and on each side there was a considerable zone of healthy fibres on the outer side of each posterior horn. In the posterior region the degenerated fibres were most numerous in the central region on each side, but immediately to each side of the posterior median fissure there was a small area of stained, apparently normal fibres. The inner or central third also of each posterior column had only a few degenerated fibres. The anterior columns presented no change.

The three figures furnish representations of typical sections in the three chief divisions of the cord.

The grey matter in all the different regions seemed to be normal, except that in the cervical region the cells were perhaps wanting in clearness and definiteness of outline. In the lumbar region there was no change from the normal.

CASE 2.—A. W—, æt. 43, admitted to the same institution on 22nd September, 1891, under the care of Dr. Gowers.

In the beginning of 1888 the patient began to suffer from a tired feeling in different joints. In June, 1890,

he accidentally cut off his right index finger. This was very soon followed by very great weakness, accompanied with vomiting and diarrhoea, and a sensation of "pins and needles" in the feet. He was at this time in Canada, and he came to this country, and after a few months he returned well, and 25 lbs. heavier. At this time he was able to walk five or six miles.

Soon after his return, however, his legs began to grow gradually more stiff. In 1891 he suffered from an "attack of piles," with vomiting and diarrhoea. He woke one morning with a dead feeling in his legs, and a sensation of intense coldness in the abdomen. Since that time he had had no control over his legs, and had been quite unable to walk. He had also had a little difficulty in passing his water, but no pains or sickness, or difficulty in breathing. During the fortnight before admission he suffered from a feeling of constriction round his waist.

His health previous to the time alluded to above had been excellent. He had had quinsy twice, but no venereal disease, not even exposure to the possibility of it. His family history was important, because, although he had four sisters living and healthy, his father died of cancer at the age of fifty-six, and a brother at twenty-eight of "poorness of blood."

His condition on admission can be described briefly. He was a tall, spare, and somewhat pale man, with slight ataxy in the arms and considerable in the legs. There was no sensory impairment, and no change in the electrical reactions of the muscles. The knee-jerk could not be elicited, but the pupils were equal, and reacted normally to light and during accommodation. His condition did not improve, and by the beginning of December the anæmia was very obvious. An examination of the blood showed the corpuscles to be 25 per cent., and the hæmoglobin to be 10 per cent. of the normal. There was some reason to doubt the accuracy of the latter observation, because a week later the corpuscles and hæmoglobin were each 20 per cent. of the normal.

By this time there was impaired sensibility both for touches and pricks, below the knee on the inner side, and on the front and inner side of the thigh, specially on the right limb. On the outer side of the leg and thigh sensibility was good. A few days later (December 17th) hæmorrhages were observed in both retinæ, and subcutaneous ones on each heel. The patient gradually sank, and died on December 24th.

At the necropsy, which was carried out on the same day, no obvious morbid condition of the viscera was found except the extreme pallor, and in the liver obvious fatty change. The brain and spinal cord were hardened in Müller's fluid, and the latter was carefully examined in several different regions. In the cervical region (Fig. 4) considerable degeneration was revealed in different parts. In the anterior columns there was well-marked degeneration on each side of the median fissure, more extensive on the left side than on the right. In the lateral columns there was very extensive marginal sclerosis, the change extending inwards, especially in the region of the crossed pyramidal tracts. The degeneration was almost symmetrical on the two sides. In the posterior columns there was very intense sclerosis, especially in the columns of Goll peripherally. Towards the centre the degeneration was less intense, and outwards on the inner side of each posterior horn there was an area of apparently normal tissue.

In the dorsal region (Fig. 5) the sclerosis was well marked in each anterior column, and similar in extent and intensity, less marked but distinct in the lateral columns, especially the pyramidal tract, and very intense in the posterior columns on each side of the median fissure, although immediately to each side of this there was a small area of less degenerated tissue. In the lumbar region (Fig. 6) the sclerosis was much less intense, and was confined to the lateral and posterior columns. In the former there was a mere patch on each side in the pyramidal tract; in the latter more extensive sclerosis, especially on each side

of the posterior median fissure. But as in the dorsal region, so also in the lumbar, there was immediately on each side of the median fissure a line of less degenerated fibres, and on each side posteriorly, internal to each posterior horn also, there was a bounding layer of comparatively undegenerated fibres.

In all these different regions the grey matter showed no change. The cells were numerous, their outline clear, and they showed no detectable alteration from the normal.

From a consideration of all these cases, it seems to me that we have in them no mere accidental association of anæmia and changes in the spinal cord. The anæmia was a prominent feature in all, and the changes in the cord were so similar and so constant that they must be regarded as in some way dependent on a similar cause. A striking illustration of this occurs incidentally in an observation of Leyden's. This author, in the course of a paper on "*Chronische Myelitis u. System-Erkrankungen im Rückenmark*," mentions a case of what he calls chronic myelitis, and figures the sections at different levels of the cord. The case was that of a patient who had been in a railway accident, and came under observation suffering from distinct symptoms of spastic paralysis. This condition remained stationary for several months, and then rapidly grew worse. He became pale, and symptoms of pernicious anæmia with characteristic changes in the blood were developed. He gradually sank and died, and a comparison of the conditions found in his cord with those in my cases and in the others referred to shows an almost identical distribution of sclerosis.

The question now arises, What determines the changes in the spinal cord? Are they the result of anæmia? is the anæmia the result of them? or are they both the result of the same cause? In most of the cases the anæmia seems to have preceded the spinal symptoms. In one of Nonne's, however, the spinal symptoms are expressly stated to have been present before the anæmia, so it seems most

natural to regard the anæmia and the spinal cord changes with their symptoms as both resulting from a common cause. Such a cause we should most naturally look for in some toxic blood condition, having regard especially to the bilateral symmetry of the changes, and to the constancy with which certain tracts were affected and others spared, so that sections at certain levels in one spinal cord are scarcely to be distinguished from sections at the same level in another (see Figs. 3 and 6). But while I believe that this is the chief cause of such changes as I have described, I think that there may be, in addition to this degeneration, other minor changes, probably the result of that tendency to the occurrence of hæmorrhages which is so marked a feature of pernicious anæmia. This tendency to hæmorrhage is most marked in the retina, but it is also manifested elsewhere, and, as I have already indicated, occurred in certain of the cases to which I have alluded ; and more particularly in the cases of pernicious anæmia without symptoms of spinal cord affection, investigated by Minnich, hæmorrhages, or small foci of degeneration which had apparently resulted from hæmorrhage, have been found in the spinal cord. One very peculiar symptom which was present in the first case which I observed, namely, the curious rhythmical persistent movement of the left arm and hand, and to a less degree of the right, may possibly be accounted for by some such change ; and it may have a little interest and perhaps significance if I mention that at the time of their occurrence the movements were spoken of by Dr. Hughlings Jackson as bearing a certain resemblance to those occurring in canine chorea. As is well known, small hæmorrhages and foci of inflammation are found in the cord of the dog in that disease. I think, therefore, that we may have two factors in the production of the cord changes : (1) the degeneration determined by a blood-state ; and (2) degeneration resulting from actual hæmorrhage, and it may be some slight focal inflammation. That a toxic condition of blood may at least be associated with spinal

cord changes, and probably causally, we have other evidence of, even if we do not regard *tabes dorsalis* itself as of this nature. I have already mentioned the analogy which Lichtheim brought forward of diabetes, universally recognised as a condition in which a poison is present in the blood, and which is frequently associated with symptoms of degenerative disease in the nervous system. When Lichtheim wrote, Williamson's researches were not yet published, but it is now proved by those that sclerosis in the posterior columns of the cord may be associated with diabetes mellitus, and is probably a result of the blood-state. We also have in pellagra a disease probably the result of a chemical poison produced in damaged maize, and characterised by sclerosis of the cord, which offers many points of similarity to the condition to which I have directed attention.

I think, therefore, that it is evident that in certain cases we have a condition of profound anæmia associated with spinal symptoms; and that these symptoms vary in different cases according to the relative amount and intensity of the sclerosis affecting different parts of the cord: that, for example, when the posterior columns are mostly affected we have ataxic symptoms with loss of the knee-jerk predominant, whereas if the changes are most intense in the lateral columns the clinical symptoms more closely resemble those of spastic paraplegia. But it is striking that in the great majority of cases ataxy is so constantly noted, and that, correspondingly, the most constant change in the cord is one of the posterior columns. It remains for future observation to show what relation this condition may have to the group of cases which Dr. Gowers has described under the name of ataxic paraplegia, and to certain others which have hitherto been regarded as cases of chronic myelitis.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 117.)

DESCRIPTION OF PLATES II AND III.

On Nervous Symptoms and Morbid Changes in the Spinal Cord in
Certain Cases of Profound Anæmia (Dr. JAMES TAYLOR).

PLATE II.

Representations of sections of the spinal cord at different levels in Case 1.

FIG. 1 shows the sclerosis as it affected the anterior, lateral, and posterior columns in the cervical region.

FIG. 2 shows the sclerosis similar in distribution in the dorsal region.

FIG. 3 illustrates the much less extensive sclerosis in the lumbar region, confined to the lateral and posterior columns.

PLATE III.

Illustrations of sclerosis in different regions of the cord in Case 2.

FIG. 4 shows the distribution of the sclerosis in the cervical region.

FIG. 5 shows the distribution of the sclerosis in the dorsal region.

FIG. 6 shows the distribution of the sclerosis in the lumbar region.

Comparison of Fig. 6 (Plate III) and Fig. 3 (Plate II) indicates an almost identical distribution of sclerosis in the two cases.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.