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THE PATHOLOGY OF

ACUTE ASCENDING (LANDRY'S) PARALYSIS

BY

HENRY HUN, M.D.,

PROFESSOR OF DISEASES OF THE CHEST AND NERVOUS SYSTEM
IN THE ALBANY MEDICAL COLLEGE

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ACUTE ASCENDING (LANDRY'S) PARALYSIS.*

The following case of Landry's paralysis which I have recently observed seems to me not unworthy of publication, especially at this time when the attempt is being made on many sides to class this disease as one of the forms of multiple neuritis:

G. C., aged forty-five years, a salesman, unmarried, entered the Albany Hospital on April 18, 1890. His family history was good, and he, with the exception of a gonorrhœa contracted at the age of twenty-six years, had always enjoyed good health, and in especial he had never had either syphilis or rheumatism. In the winter of 1886, while skating on roller skates, he fell and struck his sacrum against the edge of a bench, in consequence of which fall he felt some soreness and stiffness in his back, which passed off entirely in a week, and which did not at any time interfere with his ordinary duties. About four months after this fall he felt a sudden "lightness" in the left leg, and a feeling as if there was a tight band about the left ankle, which feeling of constriction about the ankle has persisted up to the present time. He has had some slight pain in both legs, especially in the left, at various times, always worse before a storm; but these pains have troubled him but very little during the past year. In January, 1890, he had what his physicians called an attack of "plastic iritis" of the right eye, and since that time the sight in that eye has been somewhat impaired. Of late he has felt some stiffness in his legs and has taken walks of several miles daily "to limber himself up." About one week ago (April 11th) walking became difficult, and he felt "as if he was dragging weights on his feet." At this time he was ex-

* Read before the New York Neurological Society, April 7, 1891.

amined by Dr. H. Lyle Smith, of Hudson, who found a weakness of the muscles of the legs and diminished knee-jerks, and who prescribed fluid extract of ergot. The patient continued at work, although his walking gradually became worse, until the 15th of April, when he was obliged to go to bed, having lost completely the power of the muscles of his legs below the hips. At the same time he noticed a slight weakness of his hands and arms, and a difficulty in speech (during the past month he had noticed that his voice was somewhat husky and that mucus was excreted in excess). Since he has been confined to his bed it has been necessary to draw off his urine with a catheter, and his bowels have been constipated. The muscles of his body have become paralyzed so that he has been unable to sit up in bed. His arms have grown weaker, his voice has become more husky, and deglutition has grown difficult. His appetite has been good, and he has suffered no pain.

Physical Examination.—Well nourished. Ptosis of right eyelid, which he says is voluntary and due to impairment of vision. This impairment of vision seems to consist of diplopia, one object being to the right of and lower than the other. Decided paralysis of lower branch of left facial nerve. Tongue protruded straight, no tremor of tongue or lips, and no muscular atrophy or fibrillary contraction. Breath very offensive and tongue covered with a white coat. He can whistle with difficulty. Speech thick and indistinct. Deglutition so difficult that he has to be fed with a stomach tube. Mucus collects in the trachea and patient can eject it only imperfectly. Hearing, smell, and taste are normal. Grasp of left hand null. Grasp of right hand very feeble. Flexors and extensors of upper arm very feeble, but more powerful than those of forearm. Muscles of shoulder feeble, but more powerful than those of arm. Muscles of body paralyzed so that he can neither sit up nor move in bed. Absolute motor paralysis of both legs and thighs. Absence of plantar, cremasteric, umbilical, and patellar reflexes. No muscular tenderness, fibrillary contraction, nor atrophy. No disturbance of sensibility to tactile (tested with pin-head and cotton), thermic, or painful impressions anywhere. No retardation of conduction of pain. Bladder and rectum inactive. Urine drawn off with catheter and bowels moved by injection. Temperature and pulse normal. His treatment consisted in a drachm of iodide of potassium and half a drachm of salicylate of sodium daily.

April 20th.—Patient is growing weaker. He has more difficulty in expelling phlegm by coughing and his speech is becoming more difficult. Temperature normal, pulse 85.

22d.—Growing weaker. The left-sided facial paralysis con-

tinues unchanged, and, in trying to cough, the muscles connected with the larynx and jaw on the right side of the neck can be seen to contract decidedly, while those on the left side remain passive. To-day the muscles of both arms are much weaker, and those of the right arm and shoulder are weaker than those of the left, so that he can scarcely move the right arm at all. The muscles of the legs continue absolutely paralyzed. There is nowhere any muscular atrophy, tremor, or disturbance of any kind of sensibility, and there is no muscular tenderness. All the muscles of both legs respond decidedly and quickly to the chloride-of-silver faradaic battery (the roll being shoved into between the marks 60 and 70). The difficulty of speech and of respiration is so great that it does not seem possible that he can live twenty-four hours; the coarse mucous râles in the trachea can be constantly heard.

23d.—At 11.30 A. M. he seemed better than yesterday. The symptoms remained without change, except that his voice seemed somewhat stronger, although still very indistinct, but at 12 M. he began to fail rapidly. He first became deaf; about half an hour later he became entirely blind, a quarter of an hour later he became comatose, and about a quarter of an hour later, his respirations having become steadily slower and more difficult, and being only three to five a minute, he died, the pulse being of fair quality, although rapid, almost to the moment of death, and continuing after the respiration had ceased.

Autopsy, held Eight Hours after Death.—Thorax and abdomen not examined. Post-mortem rigidity slightly marked. Spinal cord seemed normal, except that the lumbar portion seemed to be slightly œdematous, and the outlines of the gray matter in this region a little less clear than normal. Brain seemed normal, except for a somewhat increased amount of subarachnoid fluid over both parietal regions. At junction of ascending and horizontal arm of left fissure of Sylvius was a patch of white thickened cicatricial pia mater (as large as a silver quarter of a dollar) adherent to the cerebral cortex; the convolution at this point seemed atrophied. About an inch above this point on the anterior central convolution was a small piece of bone, about a half by a quarter of an inch, firmly adherent to the pia mater. The corresponding portion of the dura mater was fenestrated, and the bone of the skull at this point was roughened and presented several deep holes filled with fresh blood exuding from the veins in the bone. The arteries at the base of the brain were not atheromatous, and the ventricles of the brain were not dilated. The nervous organs were placed in Müller's fluid, which was changed once after about three hours, and then sent immediately to Dr. Van Gieson

for examination. His examination was so very thorough that I may be pardoned in presenting it at length.

Microscopical Examination.—The brain, spinal cord, and peripheral nerves were hardened in a two-per-cent. solution of bichromate of potassium for six weeks, and subsequently in eighty per cent. alcohol, and finally in strong alcohol. The sections were stained doubly by hæmatoxylin and picro-acid fuchsin, and by Weigert's method.

The *cerebral pia mater* contains in its meshes a moderate accumulation of small round and cuboidal cells which appear to be derived partly from the connective-tissue cells of the pia mater and partly by emigration from the blood-vessels. This condition of the pia mater is quite uniform over the whole convexity of the brain, and is not more extensive over the motor zone than elsewhere.

The Cerebral Cortex.—Sections from the paracentral lobule, from the middle of the precentral and postcentral convolutions of both sides, and from the lower junction of the central convolutions on the left side show nothing abnormal about the structure or arrangement of the elements of the cortex. The large multipolar ganglion cells in the paracentral lobule and the præcentral convolution are unchanged. They are not present in the post-central convolution. The blood-vessels of the cortex are normal. Sections of the basal ganglia show nothing abnormal.

The crus, pons, and medulla are normal. The nuclei of the third, fifth, sixth, seventh, tenth, eleventh, and twelfth nerves were examined in detail for changes in the motor ganglion cells, but the latter are not changed in structure or deficient in number.

The spinal cord was divided into thirty-one segments, corresponding to the insertions of the nerve roots, and sections from each of these segments were thoroughly examined by Weigert's method, and with double staining by hæmatoxylin and picro-acid fuchsin. There are no changes in the substance of the spinal cord, except some slight alterations in the ganglion cells of the anterior horns. Some of the ganglion cells stain faintly, have a vitreous, homogeneous appearance, and appear to be swollen; but these changes in the ganglion cells are so slight, and affect so few of them in such an irregular way, that the alterations in the ganglion cells may be ascribed to the bichromate hardening, which does not preserve the ganglion cells perfectly.

To eliminate the artificial changes in the ganglion cells which might be produced by the bichromate hardening, small portions from the sixth cervical and third lumbar segments were hard-

ened in absolute alcohol, a better preservative agent for the ganglion cells. The ganglion cells in these portions hardened in alcohol are well preserved and seem perfectly normal.

The ganglion cells from the hypoglossal nucleus and from the anterior horns of the spinal enlargements were examined in the fresh condition, and no changes could be found.

The spinal pia mater contains in its meshes, especially in the dorsal region, a moderate number of small round cells. A few of the larger and smaller veins of the spinal pia mater have their walls infiltrated with small round cells (Fig. 3, A).

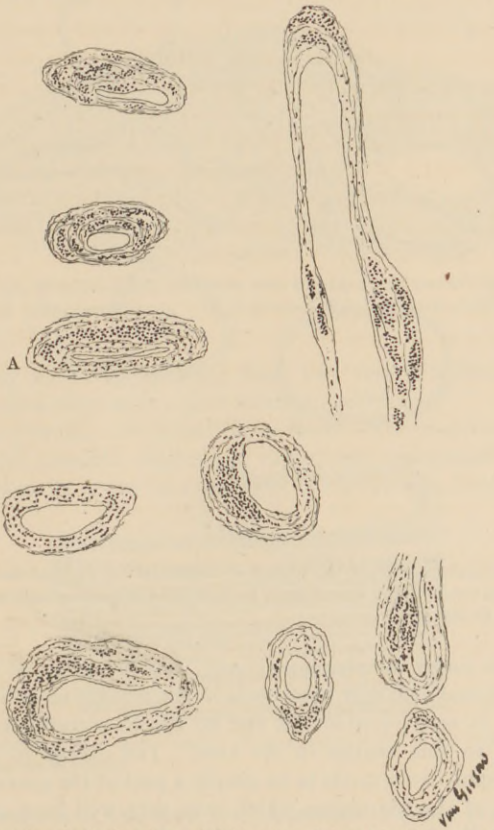

FIG. 1, showing the infiltration and thickening of the walls of the anterior spinal vein. In the section A the walls have collapsed so that the lumen is artificially closed. The lumen is nearly normal.

The anterior spinal vein and its branches in the dorsal and lower cervical regions are not only infiltrated with small round cells, but their walls are considerably thickened in places. The

lumen of the vein, however, does not appear to be narrowed. Fig. 1 shows this infiltration and thickening of the anterior spinal vein, which may be appreciated better by comparing with Fig. 2, illustrating the normal structure and thickness of the anterior spinal vein. (The lumen of the vein in Fig. 1 is very nearly normal; it looks small when contrasted with Fig. 2, but this is due to the fact that in Fig. 2 the sections were taken from a larger cord which had a correspondingly larger anterior spinal vein.)

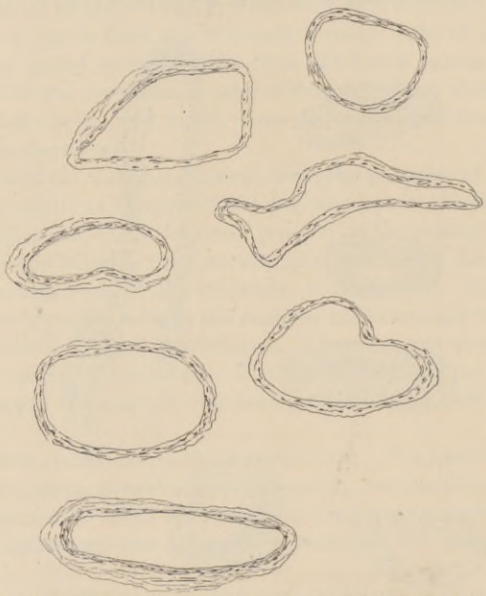

FIG. 2.—Sections showing the normal structure and thickness of the anterior spinal vein. Taken from a larger cord having a correspondingly larger vein than in Fig. 1.

How much interference, if any, this condition of the anterior spinal vein may have made in the circulation of the gray matter is very questionable, and no special significance is attached to this change in the vein. The change in the anterior spinal vein seems to be simply a part of the general infiltration of the pia mater, which may very well have occurred secondarily during the course of the disease, if not during its latter stages.

Hyaline thrombosis, recently described in Landry's paralysis by Klebs,* in the central arteries of the cord, was not found in this case. (In two places—in a small vein passing out of the

* *Deutsche medicinische Wochenschrift*, January, 1891.

hypoglossal nucleus, and in a capillary in the motor cortex—hyaline plugs were found, but the perfectly normal condition of

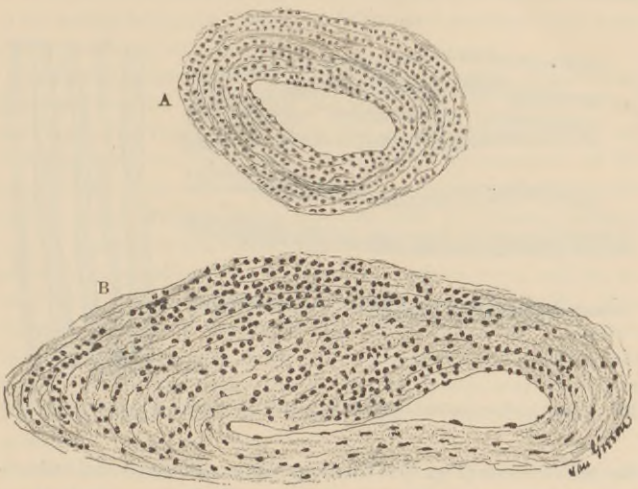

FIG. 3.—(A) Thickened and infiltrated vein accompanying one of the anterior dorsal spinal nerve roots. (B) Thickened and infiltrated anterior spinal vein.

the gray matter surrounding these two vessels indicated that the hyaline plugs were a post-mortem occurrence.)

The Cranial and Spinal Nerve Roots.—The root strands of the facial, acoustic, trigeminus, vagus, and hypoglossal nerves, and also many of the anterior roots of the cervical, lumbar, and

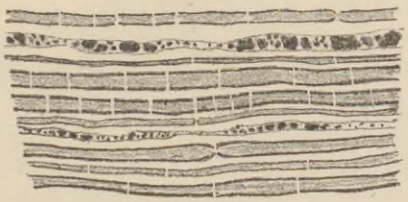

FIG. 4, showing the average number of degenerated fibers in the anterior roots of the cauda equina.

sacral regions of the cord, were hardened in osmic acid. The cranial and cervical nerve roots show no changes except those due to manipulation and imperfect preservation. There is no evidence of degeneration or neuritis in these roots. The anterior roots of the cauda equina show, in a slight degree, the changes of neuritis or degeneration of the nerve fibers (Figs. 4 and 5). The degenerated fibers are not numerous, comprising about one

tenth of the fibers of the anterior roots of the cauda equina. Transverse sections of the cauda equina show in places a considerable number of small round cells lying about and between

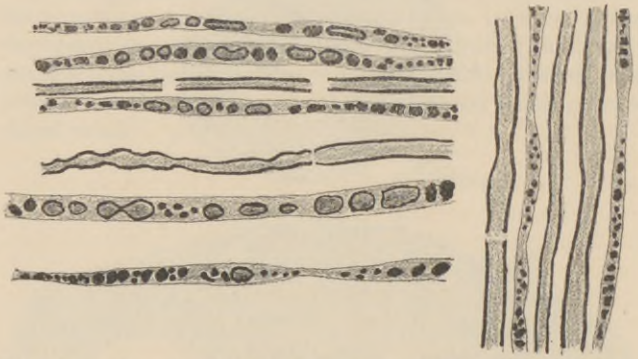

FIG. 5, from a place selected to show the maximum number of degenerated fibers.

the nerve fibers (Fig. 6). Whether the small round and branching cells of the cauda equina are actually increased to any extent is difficult to determine, and for this reason, and from the fact that Nauwerck and Barth describe an increase of cells in the cauda equina in Landry's paralysis, Fig. 6 has been drawn to show definitely the number and distribution of cells in the cauda equina. The number of cells in the cauda equina is practically normal.

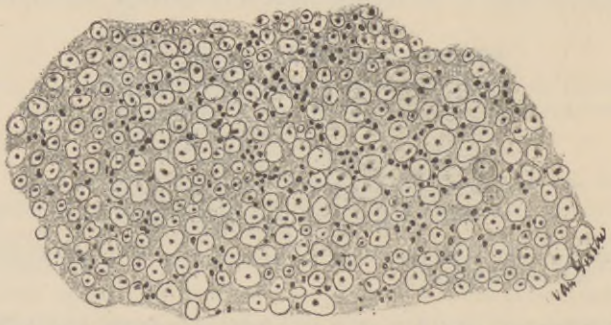

FIG. 6.—Section showing the number and distribution of the cells in the cauda equina.

The peripheral nerves examined were the median, a branch of the musculo-spiral, and the trunk of the sciatic. Sections of these nerves, stained in the same ways as the central nervous system, show nothing abnormal. Portions of all of these nerves

were also preserved in osmic acid (one-per-cent. solution) and examined, without finding any alterations.

The semi-tendinosus and biceps muscles are normal. They were examined in the fresh condition, and sections were made of the hardened muscles.

Bacteriological Examination. — Glycerin-agar plates were planted from the hypoglossal nucleus, from the motor cortex, and from the anterior horns of the spinal enlargements, and nothing grew on the plates. Cover-glass preparations of the gray matter from the same places were stained with the simple aniline dyes, with Löffler's solution, and by Gram's method, but no bacteria were found. Sections of the cord, hardened in alcohol, were stained for bacteria in various ways, and examined with the oil-immersion lens with negative results. Sections of portions of the peripheral nerves, hardened in alcohol, were carefully examined for micro-organisms, but neither the bacilli which Centanni* found in the peripheral nerves in Landry's disease, nor any other kind of bacteria, were discovered.

The results of the microscopical examinations are: A slight cerebral and spinal meningitis and infiltration of the walls of some of the veins of the spinal pia mater, and a degeneration (or neuritis) of some of the fibers of the anterior roots of the cauda equina, the nervous system in other respects being normal.

We have here, then, the case of a man who is suddenly attacked by a paresis of the legs which, in the space of four days, becomes a complete paralysis, and then the muscles of the trunk, arms, and parts supplied by the bulbar nerves become paretic in the order named, and this paresis becomes a more and more decided paralysis until death from bulbar paralysis puts an end to the further extension of the process. The paralysis was a purely motor one, the sensory nerves not being involved, and the sphincters of the bladder and rectum were not involved, although the paralysis of the abdominal muscles made the expulsion of the contents of these viscera difficult or impossible. As a result of the post-mortem examination, we have a slight cerebral and spinal meningitis of quite recent origin, a degeneration of a few of the fibers of the anterior roots of the cauda equina, and a thickening and infiltration of the walls of the anterior spinal vein.

This complex of symptoms corresponds entirely with

* *La riforma medica*, July, 1889.

the clinical picture of the disease—acute ascending paralysis—which was first clearly described by Landry * in 1859, and which has since been generally called in his honor by the name of Landry's paralysis, or by the name which Landry himself gave it, acute ascending paralysis. The only unusual symptom in the case reported was the diplopïa, a symptom which is not common in Landry's paralysis, although it has been observed by Pellegrino-Levi,† and the usual ætiological factors—*i. e.*, exposure to cold or previous occurrence of some infectious disease—were not present in this case. It is very doubtful whether the blow on the back received four years previous to his death had any connection with the disease. The slight lesions found in this case are altogether insufficient, both in extent and in intensity, to explain the severe symptoms present during life, and this case goes far to confirm the previously accepted view that acute ascending paralysis is not associated with any appreciable lesion.

It is not a desirable thing to overburden our already long list of nervous diseases with additional names, and it is a question demanding careful consideration whether acute ascending paralysis can not be regarded as a variety of some other well-recognized nervous disease. In answer to this question we must say that there are only three diseases of the nervous system which bear any close resemblance to acute ascending paralysis—viz., bulbar paralysis, myelitis of the anterior horns, and multiple neuritis.

Certainly acute ascending paralysis can be easily distinguished from bulbar paralysis, for in this latter form of disease the disturbances in respiration and deglutition are the first, or at least very early, symptoms, to which the paralysis of the arms and legs is quite subordinate, and this paralysis usually involves sensation as well as motion, while the tendon reflexes are not only present but are exaggerated. It is true that a few cases have been reported under the title acute ascending paralysis in which lesions have been found in the medulla oblongata or in the upper cervical region of the cord, but these cases were either not acute ascending

* *Gazette hebdomadaire*, 1859, pp. 472 and 486.

† *Archives générales*, 1865, i, p. 123.

paralysis at all, as in the case reported by Leyden,* or the lesions were too small to explain the symptoms, as in the case of Kümmel,† or, finally, the lesion was of a very indeterminate nature and not found in other cases, as the patches of exudative matter deeply stained by carmine found by Eisenlohr,‡ and the swollen axis cylinders and minute hæmorrhages found by Hoffmann.#

Myelitis of the anterior horns bears a closer resemblance to acute ascending paralysis, although bulbar symptoms occur very rarely in the former disease. In both diseases there is a widespread motor paralysis with abolition of reflexes and without any disturbance of sensibility or of the organic reflexes; and the attempt has been made by Petitfils,‖ Bernhardt,^ and others, to classify acute ascending paralysis as a form of myelitis of the anterior horns in which, in consequence of the rapidly fatal termination, the muscles do not exhibit the characteristic electrical reaction of degeneration during life, nor do the nerve cells in the anterior horns exhibit any definite changes in death. These assumptions were disproved by Westphal,◊ who, in several cases of relatively long duration, found neither lesions after death nor the electrical reaction of degeneration during life. Since the publication of Westphal's paper the early appearance of muscular atrophy and that of the electrical reaction of degeneration have been regarded as diagnostic symptoms, and are sufficient to distinguish clinically myelitis of the anterior horns from acute ascending paralysis; while at the autopsy of cases of myelitis of the anterior horns a definite lesion is found sufficient to explain the symptoms observed during life. Although in most cases there is no great difficulty in distinguishing these two forms of disease from each other clinically, yet some cases of myelitis of the anterior horns simulate the symptoms of acute ascending paralysis quite

* *Allg. Zeitschrift f. Psych.*, 1875.

† *Zeitschrift f. klin. Med.*, 1881, ii, p. 273.

‡ Virchow's *Archiv*, 1878, lxxiv, p. 73.

Archiv für Psychiatrie, 1884, xv, p. 140.

‖ *Considérations sur l'atrophie aiguë des cellules motrices*, Paris, 1879, p. 93.

^ *Berl. klin. Wochenschrift*, 1871, No. 47.

◊ *Archiv f. Psychiatrie*, Bd. vi, p. 765.

closely, as, for instance, the cases reported by Sudeykine * and Immermann.†

Multiple neuritis bears a very close resemblance to acute ascending paralysis, and, just as twenty years ago, when myelitis of the anterior horns was attracting great attention, the attempt was made to regard Landry's paralysis as a form of it, so, during the past five years, when multiple neuritis has been attracting much attention, the attempt has been made to regard Landry's paralysis as one of its forms, but neither attempt has been as yet successful. Certainly, to one who has observed cases of both diseases, there is a decided difference in the clinical picture of Landry's paralysis and multiple neuritis. There can be no doubt that many of the cases which have been reported under the name of acute ascending paralysis are really cases of multiple neuritis; but in every such case in which the autopsy has revealed a multiple neuritis, even of slight degree, the symptoms of bulbar paralysis were absent or very slightly marked,‡ and there was present during life either a decided disturbance of sensibility, manifesting itself by severe pain or by extensive anæsthesia,# or else a paralysis of the sphincters, ‖ or else the muscles presented a more or less well-marked electrical reaction of degeneration, and were tender on pressure and atrophied △—symptoms which form

* *Russk. Med.*, St. Petersburg, 1886. *Ctrlbl. f. klin. Med.*, 1887.

† *Poliomyelitis Anterior Acuta, Subacuta et Chronica.* By Charles Morel. Basel, 1890.

‡ Difficulty of respiration due to paralysis of the intercostal or phrenic nerves can not be regarded as a symptom of bulbar paralysis in the absence of any disturbance in deglutition and speech.

Sensory symptoms were prominent in the cases reported by Nauwerck and Barth (Ziegler's *Beiträge z. path. Anat.*, etc., Bd. v, p. 1) and Eisenlohr (*Deutsch. med. Woch.*, Sept. 18, 1890).

‖ Paralysis of the sphincters was present in the cases reported by Déjerine (*Arch. de phys. norm. et path*, 1873, p. 312, and 1876, p. 312; *Comp. rend.*, 1878, lxxxvii, No. 3; and *Recherches sur les lésions du système nerveux*, Paris, 1879) and Roth (*Corresp.-Blatt. f. schweizer Aerzte*, 1883, No 13).

△ The electrical reaction of the muscles was altered in the cases reported by Déjerine (*Arch. de physiol. norm. et path.*, 1876 p. 312; *Comp. rend.*, 1878, lxxxvii, No. 3; and *Recherches sur les lésions du système nerveux*, Paris, 1879), Eichhorst (Virchow's *Arch.*, 1876, lxix), Eisenlohr (*Deutsche med. Woch.*, Sept. 18, 1890), Gombault (*Arch. de physiologie*, v, 1873, p. 81), and Schultz and Schultze (*Arch. f. Psychiat.*, Bd. xii, p. 458).

no part of the clinical picture of the disease described by Landry.

It is true that cases of multiple neuritis present so much variation in the relative prominence of the sensory and motor symptoms, in the completeness of the electrical reaction of degeneration, and in their other symptoms, that it seems somewhat artificial to insist on separating the complex of symptoms constituting Landry's paralysis from the general class of multiple neuritis. But if we do make such a separation, we can find in medical literature some two dozen cases of well-marked Landry's paralysis, while many other cases reported under this title are cases either of multiple neuritis or are too imperfectly reported to allow of a diagnosis being made. Of these cases, six terminated in recovery;* in twelve cases the disease terminated fatally, but the peripheral nerves were not examined;† while at least six cases similar to the one which forms the basis of this paper have been reported by competent observers—such as Westphal,‡ Cornil,# Bernhardt, ‖ and Leyden △—in which during life the paralysis was a purely motor one, followed an ascending course, and extended to the medulla, there being no involvement of the sphincters nor any reaction of

* These cases terminating in recovery have been reported by Mieth (*Deutsch. med. Woch*, 1885, xi, p. 67), Rendu (*La France médicale*, 1881, ii, p. 793), Sorgenfrey (*Neurolog. Ctrlbl.*, Bd. iv, p. 198), Oppenheim (*Wien. klin. Woch.*, 1890, No. 39, iii, p. 761), Ross (*Treatise on Diseases of the Nervous System*, second ed., vol. 1. p. 905), and Eiselt (*Aertz. Bericht. des K. K. allg. Krankenhaus z. Prag* [1880], 1882, p. 53).

† These cases terminating fatally in which the peripheral nerves were not examined have been reported by Walford (*Brit. Med. Jour.*, 1853, p. 993), Chalvet (*Thèse de Paris*, 1871, p. 16), Dazet (*Revue médicale de Toulouse*, 1881, xv, p. 225), Emminghaus (*Verhand. d. phys.-med. Gesellsc. in Würzburg*, 1880, N. F. xiv, p. 17), Finny (*Brit. Med. Jour.*, 1882, vol. 1, p. 732), Kahler and Pick (*Arch. f. Psychiat.*, Bd. x, p. 313), Kümmel (*Zeitsch. f. klin. Med*, 1881, ii, p. 273), Aufrecht (*Path. Mittheil.*, Magdeburg, 1881, i, p. 170), Eisenlohr (Virchow's *Arch.*, 1878, lxxiii, p. 73), Mann (*Med. Chronicle*, 1887, vol. vi, p. 99), Féré (*Comptes rendus d. la Société de biologie*, 1888, vol. v, 3d s., p. 189), and Stair (*Med. Record*, 1882, vol. xxii, p. 355).

‡ *Arch. für Psychiatrie*, 1876, vi, p. 765.

Case of Pellegrino-Levi, reported in *Archives générales de médecine*, 1865, vol. 1, p. 132.

‖ *Berlin. klinische Wochenschrift*, 1871, No. 47.

△ *Die Entzündung der peripheren Nerven*, Berlin, 1888, p. 21.

degeneration, and in which, after death, the most careful examination of the central and peripheral nervous organs failed to reveal any lesion.

It must be confessed that most of these observations are of rather old date, and, further, it may be urged that typical cases of Landry's paralysis run such a rapid course that death occurs before the reaction of degeneration and structural changes in the nerve fibers have had time to develop. But in those cases which run a slower course the reaction of degeneration does not occur, and even at the outset of the disease cases of acute ascending paralysis differ from cases of multiple neuritis by the absence of those decided sensory disturbances which are so characteristic of the latter disease. And, finally, those very acute cases of multiple neuritis which terminate fatally in the course of a few days, as in the cases reported by Putnam * and Rosenheim,† or in a few hours, as in the case reported by Pitres and Vaillard,‡ present the well-marked lesion of a neuritis.

Two papers have recently appeared, in which the attempt is made to prove that Landry's paralysis is a form of multiple neuritis, which are so very elaborate that they demand especial notice. One of these papers was published in England by Ross, and the other in Germany by Nauwerck and Barth.

Ross # makes a comparative study of a large number of cases of acute ascending and allied forms of paralysis. Of course, it is possible—by including in a list of cases of acute ascending paralysis a number which, by their clinical course and by the lesions found at the autopsy, are evidently cases of multiple neuritis, and then making a summary of the symptoms found in all these cases—to so obliterate the lines dividing the two diseases from each other that acute ascending paralysis will resemble more and more closely multiple neuritis, and the greater the number of cases of this latter disease which are included in the list, the greater will this resemblance be. This is what Ross does in his article; but even he agrees with the first con-

* *Boston Med. and Surg. Jour.*, Feb. 4, 1889.
† *Arch. f. Psychiat.*, Bd. xviii, Hft. 3.
‡ *Arch. de physiologie*, 1887, ii, p. 150.
# *Medical Chronicle*, November, 1889, p. 102.

clusion of Nauwerck and Barth, that cases of typical acute ascending paralysis do occur in which it is impossible after death to discover any lesion.

Nauwerck and Barth,* after a long discussion of the previously reported cases of acute ascending paralysis and of some allied forms of disease, arrive at the following conclusions:

1. A typical acute ascending paralysis, with slight sensory symptoms, without implication of the sphincters, and without any diminution of the electro-muscular excitability, may prove fatal, and it may be impossible to discover any anatomical changes in either the central or the peripheral nervous system.

2. No sure proof has hitherto been afforded that the clinical picture of an acute ascending paralysis can be produced by disease of the medulla oblongata or spinal cord or of any part of the central nervous system.

3. If acute ascending paralysis is defined so as to include cases in which the loss of motor power is accompanied by more severe sensory symptoms, by affections of the sphincters, and especially by diminution or loss of the electro-muscular contractility or the reaction of degeneration, then a considerable number of observations have been recorded which warrant us in assuming a disease of the peripheral nerves alone.

4. It has not been proved that, even in the extended signification of the term, acute ascending paralysis can be caused by disease of the central nervous system.

At the close of their valuable paper Nauwerck and Barth report under the name of Landry's paralysis a case in which, after death, the medulla and spinal cord were normal, but an interstitial neuritis, without any degenerative changes in the nerve fibers,† was found in the cauda equina, and to a less degree in the anterior and posterior nerve roots throughout the spinal cord, while the bulbar nerve roots were normal. The muscles and the peripheral nerves were normal with the exception of the sciatic nerves, which were affected in the same way as the nerve roots. Their case then must

* Ziegler's *Beiträge z. path. Anat. u. z. allg. Pathologie*, Bd. v, p. 3.

† This lesion is therefore quite different from the lesion found in the case reported in this paper.

be regarded as a multiple neuritis mainly limited to the nerve roots, and one factor in the production of the neuritis may well have been the tuberculous disease which was found in the lungs. Clinically the case resembles a case of multiple neuritis, and not one of acute ascending paralysis, in that the legs were the seat of sharp pains during many weeks and were almost absolutely anæsthetic, and the arms moderately so; the neuritis of both the anterior and posterior nerve roots thus producing characteristic symptoms in the domain both of motility and sensibility. There was no difficulty in swallowing, in breathing, or in speaking, there were no other bulbar symptoms, and the motor paralysis was showing decided improvement when the patient died in collapse. The fact that the patient died in collapse, apparently due to cardiac rather than to respiratory failure, can not be taken as sufficient proof of bulbar paralysis in the absence of any previous symptom of such paralysis. It is quite as reasonable to suppose, considering the weak condition of the patient resulting from the long-continued pulmonary tuberculosis and paralysis, that the collapse was due to the hæmorrhage found after death in the lumen and in the walls of the small intestine, or even if the autopsy revealed no cause for the collapse, it does not follow that bulbar paralysis was the cause of it. This case, then, occurring in a tuberculous patient and presenting severe disturbances both of motion and sensation without bulbar symptoms, running a chronic course and showing improvement in the paralysis previous to the sudden death, may be indeed a peculiar form of multiple neuritis, but can not be regarded as a typical case of acute ascending paralysis, and does not justify the conclusion of the authors "that Landry's paralysis is to be regarded as an infectio-toxic multiple neuritis."

Finally, Klebs* attempts to refer the symptoms of Landry's paralysis to a hyaline thrombosis of the branches of the central artery of the spinal cord, because in one case he found such a hyaline thrombosis which was exclusively limited to those branches of the central artery lying on each side of the central canal and supplying the anterior horns of gray matter. This thrombosis had apparently

* *Deutsch. med. Wochenschrift*, Jan. 15, 1891.

produced a dilatation of the circumcellular spaces about the motor cells, which dilated circumcellular spaces contained small round cells and a network of coagulated material. This lesion would explain the symptoms of the disease fairly well, but it is certainly of a very indeterminate nature. It has been found in only one case, and was not present in the case reported in this paper, and Hlaval* did not find this hyaline thrombosis in a case reported by him under the title of Landry's paralysis, but which is evidently a case of myelitis of the anterior horns.

In conclusion, it appears from this somewhat hasty and imperfect review of the subject that no conclusive evidence has as yet been brought forward which justifies us in abandoning the term acute ascending or Landry's paralysis, for neither has it been shown that those cases of the disease in which no lesion has been discovered depend on faulty methods of examination, nor has any case been reported which was clinically a typical case of Landry's paralysis in which, after death, characteristic lesions were found either in the central nervous organs or in the peripheral nerves. Acute ascending paralysis (defined so as to exclude all cases in which the sensory symptoms are prominent, or in which well-marked bulbar symptoms are not present) must therefore be regarded as a clinical entity for which no corresponding lesion has as yet been discovered. That there is some change in the nervous system causing the severe symptoms can not be doubted, but this change is probably of a chemical rather than of an anatomical character. From the many points of resemblance which acute ascending paralysis bears both to myelitis of the anterior horns and to multiple neuritis, this chemical change must affect either the motor cells of the spinal cord and medulla or the fibers springing from them; and, although this chemical change is so great as to cause an entire arrest of the function of those cells or fibers, yet it leaves no trace in any altered character of cell or fiber, no more than does morphine or strychnine leave any trace in the structure of the nervous system of their fatal action.

In regard to the nature of this supposed chemical poison we know nothing. The general tendency of the present

* *Archives bohèmes de médecine*, vol. iv, No. 2, p. 270.

day is to consider it to be a ptomaine, and indeed the acute course, the fact that it often follows an infectious disease, and that it is associated with an enlargement of the spleen, make it not improbable that Landry's paralysis is a germ disease. In further support of this view is the fact that the paralytic form of rabies closely resembles Landry's paralysis, and that Baumgarten * found the anthrax bacillus in the spinal cord, and Curschmann † found the typhoid bacillus in the spinal cord in cases bearing some resemblance to, but evidently not true cases of, Landry's paralysis; and in two similar cases of so-called Landry's paralysis in which the diagnosis was, to say the least, extremely doubtful, Centanni ‡ and Eisenlohr # have each found bacteria in the central nervous organs. In neither case were the bacteria cultivated, and in a second case observed by Eisenlohr no bacteria were found. In a number of cases bacteria were looked for, as was done in the case which I report, but were not found. So that the hypothesis that Landry's paralysis is due to bacterial agency, attractive as it seems, is far from being proved, and the pathology of the disease still remains to be discovered.

* *Arch. d. Heilk.*, 1876.

† *Verhandl. des v. Congress f. innere Medicin*, 1886.

‡ Ziegler's *Beiträge zur path. Anatomie*, etc., Bd. viii, Hft. 3.

Deutsch. med. Wochenschrift, No. 38, Sept. 18, 1890.