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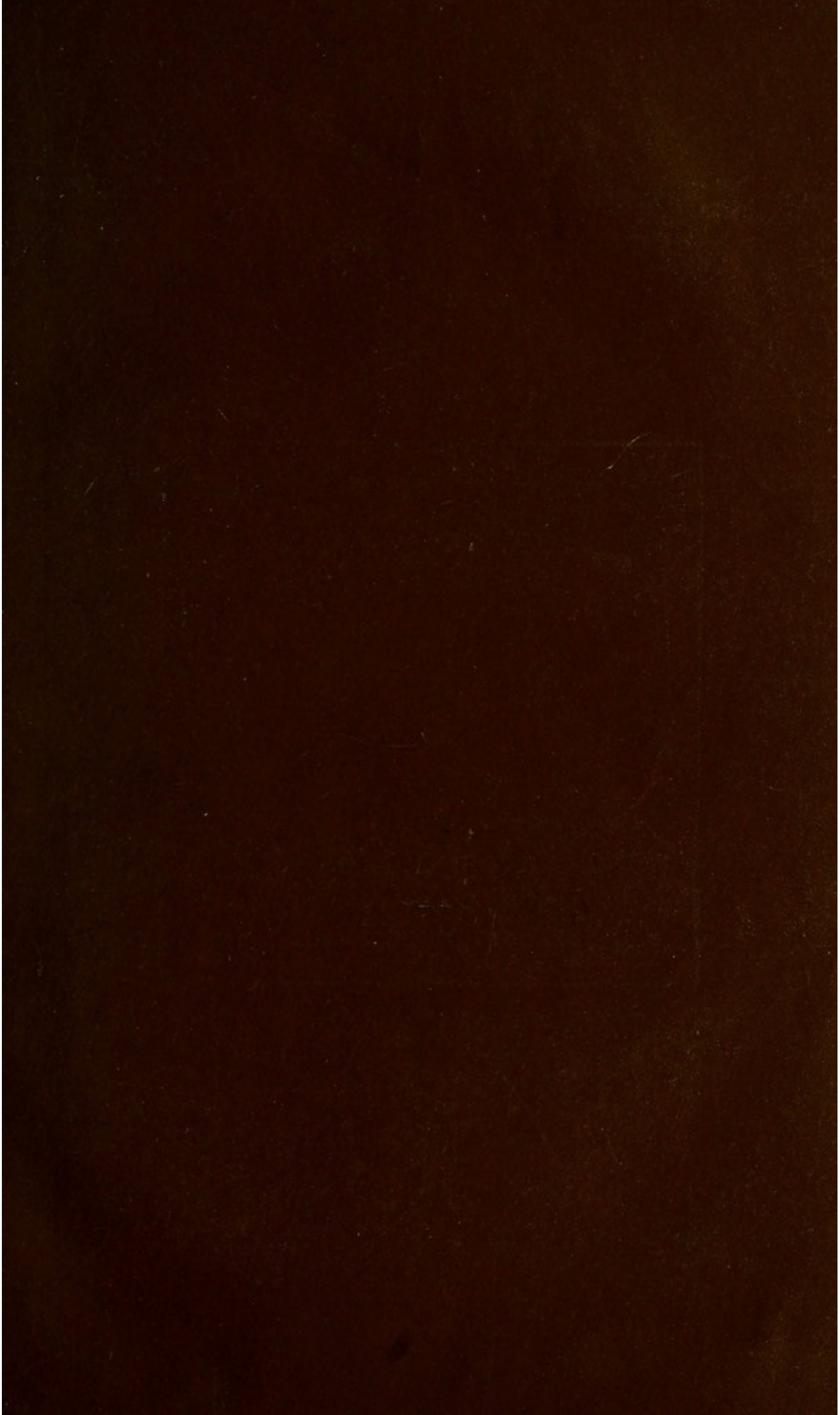
Myelitis
of
The Anterior Horns

Seguin.



M. F. Lincoln.

MYELITIS

OF

THE ANTERIOR HORNS

OR

SPINAL PARALYSIS OF THE ADULT AND CHILD

BY

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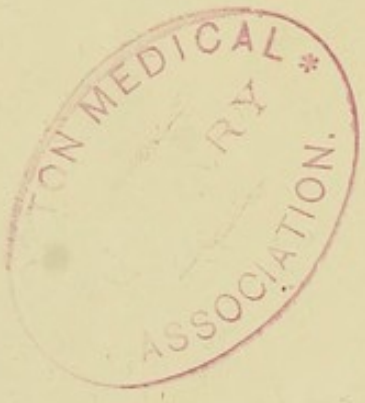
NEW YORK

G. P. PUTNAM'S SONS

182 FIFTH AVENUE

1877.

H. P. Bowditch, M.D.
1/28/82.



PREFACE.

On the fifth of November, 1874, I read before the New York Academy of Medicine an essay entitled: Spinal Paralysis of the Adult; Acute, Sub-acute, and Chronic. This, together with a reprint of a clinical lecture upon Infantile Spinal Paralysis, was reprinted and bound for private distribution only.

Numerous demands for the essay, and the fact that physicians everywhere were contributing new cases of the disease, and were disposed to admit the identity of the affection in adults and children, led me to wholly re-write the essay and offer it to the profession.

As now presented the essay embodies more cases of my own, and all the cases I have been to collect; and it is so constructed as to treat chiefly of myelitis anterior in the adult, and to refer in every chapter to the disease as it occurs in childhood.

I trust that the plan which I have followed in the study of the subject, viz., an attempt to apply the inductive method, will meet the approval of my professional brethren.

E. C. Seguin.

New York, *February 15th*, 1877.

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MYELITIS OF THE ANTERIOR HORNS.

CHAPTER I.

HISTORICAL CONSIDERATIONS.

WITHIN the last few years the attention of physicians has been drawn to a form of myelitis which had previously escaped observation.[1] The rarity of this affection has, I now think, been much exaggerated, and in proof of this I present in this essay a much greater number of cases of the disease than I did in the former one. Furthermore, I now frequently hear of cases observed by specialists and general practitioners.

The observers who have placed on record cases of the disease occurring in adults, or have written more extensively about it, have failed to agree in the matter of nomenclature; and the following names have been employed by them to designate the affection:

Acute anterior spinal paralysis (Duchenne), subacute general anterior spinal paralysis (Duchenne). Spinal paralysis of the adult (Meyer, Charcot, Gombault, Hammond). Myelitis of the anterior horns

[1] Writing as late as 1868, so experienced a physician as Prof. J. Russell Reynolds, of London, stated in a lecture upon infantile spinal paralysis, that he had seen the same symptom group in pregnancy, but that, "apart, however, from pregnancy I do not know of any disease in the adult which exhibits the clinical history now detailed." The Lancet, 1868, II, p. 35.

(Dujardin-Beaumetz). Acute spinal paralysis of adults (Petitfils, Eisenlohr, Erb). An affection of adults like infantile spinal paralysis (M. Bernhardt). Poliomyelitis anterior (Kussmaul, Frey). Atrophic paralysis of adults (Leyden).

In my paper I used the two denominations, spinal paralysis of the adult, and inflammation of the motor tract of the spinal cord, acute, sub-acute, and chronic.

For reasons which will be given when I come to treat of the pathology of the disease, I have now abandoned the former name as utterly ambiguous and indefinite, and have adopted a synonym of the second denomination. I shall speak of the disease, with Dujardin-Beaumetz, as MYELITIS OF THE ANTERIOR HORNS. Although the term POLIOMYELITIS ANTERIOR is one which may always sound pedantic for Anglo-Saxon ears, it should be retained as the Græco-latin equivalent.

Myelitis of the anterior horns may be, as I shall show, acute, sub-acute, and chronic; generalized or diffused, and localized; it may occur at almost any period of life; it may be a primary disease of the spinal cord and be complicated with other lesions, and it may arise secondarily as a complication of pre-existing disease of the spinal cord or brain.

It appears to me that the recognition and exact determination of this form of myelitis constitute quite as great a progress in pathology as the definition of sclerosis of the posterior columns (progressive locomotor ataxia).

Duchenne[1] (de Boulogne) in the last edition of his work on electrization, claims that he recognized the sub-acute form of this affection as early as 1847; and that in 1853 he gave to the profession the first sketch of its

[1] De l'Electrisation localisée, 3me. édition. Paris, 1872, pp. 437, 459–461.

symptoms. Concerning the acute form he writes (p. 437): "I had long thought that the symptomatology of the atrophic palsy of childhood, whose anatomical character is mainly acute atrophy of the anterior cells of the spinal cord, was not met with in adults; but having met with the same symptomatology in the latter, I have naturally concluded that this paralysis must be produced by the same lesion. This led me to give this palsy the name of acute anterior spinal palsy of the adult, or palsy by atrophy of anterior cells."

Moritz Meyer[1] in his work on medical electricity, described spinal palsy in the adult, and recognized its analogy with infantile spinal paralysis; and gave two interesting cases. As early as 1863, Prof. Charcot,[2] of Paris, was consulted by a patient bearing the remains of an attack of acute spinal paralysis. Charcot at that time expressed the opinion (in his notes) that the case was like the cases of infantile spinal palsy: "If we leave out the condition of age, the case of Mr. L. might be by all its characters likened to the myopathic paralysis of children." Speaking in 1872, Charcot[3] says: "With reference to the spinal paralysis of adults and general spinal paralysis (Duchenne), we have as yet no definite teaching from pathological anatomy. Judging by their symptoms, it is at least very probable that these affections are connected with a lesion of motor nerve cells. The spinal paralysis of adults recalls that of children by the almost sudden accession of palsy, by the tendency to retrocession which is shown

[1] Electricity in its Relations to Practical Medicine. Translated by Dr. Hammond. New York, 1869, pp. 229–242. (German third edition, 1868).

[2] Charcot's private notes cited by Petitfils, Considérations sur l'atrophie aiguë des cellules motrices. Paris, 1873, pp. 92–'7.

[3] Leçons sur les maladies du système nerveux, Paris, 1872–'3, p. 63.

at a given moment, by the quickly developed loss of electro-muscular reaction to the faradic current in the palsied muscles, and, lastly, by the rapid atrophy which these muscles undergo."

Dujardin-Beaumetz,[1] in his aggregation thesis, indorses Duchenne's views in regard to the semeiology and probable pathological anatomy of the disease. Gombault,[2] in the early part of 1873, published a case of acute spinal palsy of the adult with post mortem examination. He found lesions in the spinal cord which were those anticipated by Duchenne, Charcot, and others. Charcot's and Meyer's cases were republished in 1873, in a thesis by Petitfils.[3] In 1874 several contributions to the literature of the subject appeared. Bernhardt[4] wrote a short essay on the subject, basing his remarks upon one case without autopsy. Frey,[5] a pupil of Kussmaul, of Freiburg, reported three cases of the disease with remarks showing that myelitis of the anterior horns was a disease well-known in the clinic of Freiburg. In the autumn of the same year Kussmaul[6] reported another case.

In this country the subject was mentioned in an appreciative manner by Dr. Mary Putnam Jacobi[7] in

[1] De la myélite aiguë, Paris, 1872, p. 51.

[2] Note sur un cas de paralysie spinale de l'adulte suivi d'autopsie. Arch. de phys. normale et pathologique, 1873, pp. 80–87.

[3] Op. cit, pp. 71–83.

[4] Ueber eine der spinalen Kinderlähmung ähnliche Affection Erwachsener. Arch. f. Psych. u. Nervenkrankheiten, 1874, p. 370.

[5] Ueber temporare Lähmungen Erwachsener, die der temporären Spinallähmung der Kinder analog sind, und von Myelitis der Vorderhörner anszugehen scheinen. Berlin. klin. Wochenschrift, 1874, nos. 1–3.

[6] Frey, Ein Fall von subacuter Lähmung Erwachsener. Wahrscheinlich Poliomyelitis anterior subacuta. Ibid. No. 44–45 (p. 549, and p. 566).

[7] Pathology of Infantile Paralysis. American Journal of Obstetrics, May, 1864, p. 21. [Read before the New York County Medical Society, Dec. 22, 1873].

an erudite paper upon paralysis in childhood. In 1875, Dr. D. F. Lincoln,[1] of Boston, placed a case on record, and Profs. Cornil and Lépine,[2] of Paris, detailed at length a most important case with a thorough autopsy. It is upon this case and Gombault's that our positive knowledge of the pathological anatomy of the disease rests. Leyden[3] in the second volume of his admirable treatise upon diseases of the spinal cord, treats of the subject very briefly, and does not appear to fully appreciate its importance.

In an article[4] written later in the same year he discusses very ably the pathological anatomy of the affection as it occurs in adults and in children.

A very important contribution is that of Prof. W. Erb, of Heidelberg,[5] in which he announces the discovery of the reaction of degeneration (*entartungsreaction*) in myelitis of the anterior horns. He gives several interesting cases.

During the present year Dr. Wm. A. Hammond[6] has issued a new edition of his treatise upon diseases of the nervous system, and inserted an exhaustive and able chapter upon spinal paralysis of the adult. He allows twenty-one pages to the subject, and classifies

[1] A case of Spinal Paralysis in an Adult, resembling the so-called Infantile Paralysis. Boston Med. and Surg. Journal, 1875, March 25.

[2] Sur un cas de paralysie générale spinale antérieure sub-aiguë, suivi d'autopsie. Gazette Médicale de Paris, 1875, p. 127.

[3] Klinik der Rückenmarks-Krankheiten, Berlin. 1875, Bd. ii. p. 196.

[4] Beiträge zur pathologischen Anatomie der atrophischen Lähmung der Kinder und der Erwachsener. Archiv f. Psych. und Nervenkrankheiten, VI. p. 271.

[5] Ueber acute Spinallähmung (Poliomyelitis anterior acuta) bei Erwachsener, und über verwandte spinale Erkrankungen. Arch. f. Psych. u. Nervenkrankheiten, V, p. 758.

[6] A Treatise on Diseases of the Nervous System, 6th ed. New York, 1876, p. 470, et seq.

the disease under the general head of inflammation of the anterior tract of gray matter of the spinal cord, or inflammation of motor and trophic nerve-cells, placing it between infantile spinal paralysis and pseudo-hypertrophic spinal paralysis.

For my own part I can say that in November, 1871, I clearly recognized the affection as illustrated in case XVII. This girl, with her wasted left leg, I often pointed out to the resident medical staff and to visitors as an example of infantile spinal palsy occurring in an adult. In the spring of 1874 I had the opportunity of devoting a part of a clinical lecture at the College of Physicians and Surgeons to a man affected with the disease, and then indicated to the class the nosological place of acute and sub-acute inflammation of the anterior motor tract of the spinal cord. On November 5, same year, I read a paper[1] upon the subject before the New York Academy of Medicine.

As regards the growth of our knowledge relative to myelitis of the anterior horns occurring in children (infantile spinal paralysis), I will not do more than subjoin a reference to a number of books and essays devoted to its study.[2]

It has been known by the following synonymous names :

[1] Spinal Paralysis of the Adult; acute, sub-acute, and chronic. (Inflammation of the motor tract of the spinal cord), Trans. of the New York Academy of Medicine, vol. ii., 1876, p. 43, et seq.

[2] Heine, Spinale Kinderlähmung, Stuttgart 1860.

Laborde, De la paralysie (dite essentielle) de l'enfance. Paris, 1864,

C. B. Radcliffe, Art. in Reynolds' System of Medicine. Lond. 1868, vol. ii. p. 661.

Duchenne, Électrisation localisée, 1872, p. 381.

Leyden, Klinik der Rückenmarks-Krankheiten, ii. p. 552.

Hammond, Diseases of the Nervous System, 1876, p. 451.

E. C. Seguin, Infantile Spinal Paralysis : A Clinical Lecture, N. Y. Medical Record, 1874, p. 25.

Infantile spinal paralysis (Heine Charcot, Hammond, myself). Essential paralysis of childhood (Rilliet and Barthez). Myogenic paralysis (Bouchut). Essential paralysis of children (Niemeyer). Infantile paralysis (C. B. Radcliffe, Adams, Mary Putnam Jacobi, and others). Atrophic paralysis of children (Duchenne, Leyden, and others).

CHAPTER II.

RELATION OF CLINICAL FACTS.

Duchenne's cases under the title of Acute Anterior Spinal Paralysis.

CASE I.[1] Female aged twenty-two years. Fever; pain in muscles and down vertebral column, severe in cervical region; numbness in fingers; loss of voluntary movements. Sensibility preserved; micturition and defecation done normally. Fever ceased on fourth day, leaving palsy of all limbs, which in part disappeared spontaneously. Over six months after, patient exhibited palsy with atrophy of right tibialis anticus, right deltoid, infra-spinatus, biceps, interossei, and thenar muscles, and of the left serratus magnus, and flexor muscles of fingers. Improved by faradization.

CASE II.[2] Male aged forty-two years. In 1848, an effort accompanied by a cracking sensation in the back, and followed by severe pain extending into the limbs, with numbness in, and, later, palsy of both lower extremities. Paraplegia was spontaneously cured in two months. In 1869, without cause, fever and general paralysis appeared, followed by rapid wasting of fore-arms and hands, and of some muscles of the lower extremities. Improvement in four months. When

[1] Duchenne, De l'électrisation localisée, 3me. ed. 1872. Obs. lxix, p. 438.
[2] Duchenne op. cit., obs. lxx. p. 439.

seen by Duchenne, showed atrophy of hands, of extensor muscles of wrist, and of anterior tibial muscles. At no time was there any palsy of the bladder or sphincter ani.

CASE III.[1] Male aged twenty-one years. Lay naked on snow and fainted away. Afterward, had chill, fever, and delirium, with consequent general paralysis. Sensibility perfect; no trouble with bladder or sphincter ani. When seen, fifteen years later, showed atrophy of all of the muscles of the right leg, and of nearly all those of the left. The upper extremities exhibited wasting of the deltoid and muscles of hand on the right side, of the serratus magnus on the left.

CASE IV.[2] Female aged eighteen years. Hard work out of doors. Fever set in, lasting eight days, with pain in the cervical part of the spine and in the limbs: numbness in the latter. There was also palsy of arms and legs, with preservation of sensibility; bladder and intestines normal. Residue,—— atrophic palsy of right upper extremity. Improvement in palsy occurred spontaneously in the second month.

Duchenne's cases entitled Sub-Acute General Anterior Spinal Paralysis.

CASE V.[3] Male aged fifty-five years. Gradually developed palsy of the lower limbs with wasting of muscles; no affection of the bladder. In a year extension of atrophic palsy to upper limbs and trunk; weakness of the masticatory muscles, and slow speech. Slight anæsthesia in lower limbs, none elsewhere.

[1] Duchenne, op. cit., obs. lxxi, p. 440.
[2] Duchenne, op. cit., obs. lxxv, p. 445.
[3] Duchenne, op. cit., obs. lxxxi, p. 461.

Autopsy (1848) showed nervous centres healthy to the naked eye.

CASE VI.[1] Female aged ———. Gradually developed palsy of right arm, both legs, and, lastly, left arm; speech and deglutition difficult. When seen, loss of electro-muscular contractility in many muscles of back and limbs; much wasting of muscles of limbs. Sensibility and the functions of the bladder unaffected.

CASE VII.[2] Male aged fifty-one. Gradually developed palsy (descending) of all the limbs, with numbness and slight anæsthesia, but no trouble with bladder. Spontaneous partial recovery thirteen months later. When seen, five months later, presented atrophy and palsy of many muscles of forearms and hands.

CASES VIII AND IX.[3] By Moritz Meyer. The two Barons von H., twin brothers, well-built, fine, large men, uniformly healthy, in their eighteenth year simultaneously fell sick with the measles. This, having run an apparently favorable course, was followed in both with a paralysis of the legs, inducing a constantly increasing emaciation of those parts. Meyer states that he saw these men six years later, and minutely describes the wasting of many muscles of the lower limbs, and the consequent deformities. He adds that the sensibility of the skin and muscles was perfectly preserved. There was reduction of electro-muscular contractility in the wasted muscles.

[1] Duchenne, op. cit., obs. lxxxii, p. 463.

[2] Duchenne, op. cit., obs. lxxxiii, p. 464.

[3] Moritz Meyer, Electricity in its Relations to Practical Medicine. New York, 1869, pp. 229–242.

Professor Charcot's cases.

CASE X.[1] Male aged thirty-one years, seen by Charcot in 1863. In 1859 paralysis appeared, preceded by pain in the left side; weakness of left lower extremity, without numbness; in twenty-four hours complete paraplegia and anæsthesia. No numbness or spasm; no affection of sphincter ani or bladder. In next month there occurred wasting of some of the palsied muscles, and lowering of temperature in parts. Later there remained some patches of anæsthesia in legs, sides, and right axilla. In three months great improvement occurred (moxas to spine, iodide of potassium internally). When examined, showed atrophy of left natis, leg, and foot; of left lower abdominal muscles; of anterior part of right thigh. Atrophied muscles have lost electro-muscular contractility. Skin over wasted muscles colder than elsewhere. Charcot recognized similarity to infantile palsy.

CASE XI.[2] Male aged ———, seen by Charcot in 1871. In February, 1871, severe dysentery, followed in eight days by weakness of upper extremities, and complete palsy of the lower. Palsy with complete flaccidity of muscles. After a few days lower limbs were cold. The arms recovered soon; and in eight days imperfect walking was possible. Since, progressive improvement. When seen, no anæsthesia existed; the anterior part of thighs are wasted and flabby, and have lost electro-muscular reaction. Never any palsy of sphincter ani or of bladder.

[1] Petitfils, Considérations sur l'atrophie aiguë des cellules motrices, Paris, 1873, p. 72.

[2] Petitfils, op. cit., p. 78.

Case XII.[1] Male aged thirty-five years, seen by Charcot in October, 1871. In the month of August preceding, this man suffered from malaise for four days; on fifth day, right arm was feeble and tremulous. Later, on the same day, right arm paralyzed; then left leg, right leg, and left arm. Accompanying fever, with delirium lasting one week. At time of consultation there is noted wasting of right arm and left leg; no anæsthesia; formication from time to time. Never any rectal or vesical symptoms; nor bed-sore.

Case XIII.[2] By Prof. Cuming, of Belfast. Male aged forty years. Exposure to cold; numbness in hands; next day walking difficult. On fourth day, complete palsy of all limbs, without anæsthesia. Later, spasms, and lancinating pains in lower limbs. Gradual return of voluntary movements in three months. Cure with *main-en-griffe*. No bed-sore; no palsy of bladder or of sphincter ani.

Case XIV.[3] By Gombault. Female aged sixty-seven years. On January 1, 1867 (when sixty-two years old), in one day experienced great numbness, followed by palsy of all limbs. Sensibility normal; respiration, deglutition, and cerebral functions not impaired; no bed-sore; no palsy of bladder or of sphincter ani. Pain in back at beginning, and for months afterwards. Was brought to the hospital in fifteen days; had sensibility, but was completely palsied. No contracture of palsied limbs. After two years began to improve

[1] Petitfils, op. cit., p. 78.

[2] Dublin Quarterly Journal of Medicine, 1869, p. 471.

[3] Sur un cas de paralysie spinale de l'adulte, suivi d'autopsie. Archives de phys. norm. et pathol., 1873, pp. 80–87.

gradually; and in three and a half years was able to walk a little with the help of a stick. Upper limbs improved first. Examination in 1872 shows slight wasting of arms and forearms, much atrophy of muscles of hands, thenar eminences, and interossei: *main-en-griffe*. Extensors of hands very weak. Muscles of arm and forearm show fibrillary contractions. Much atrophy of many muscles of legs. Loss of electro-muscular reaction in hands and extensor group of fore-arm; lessened reaction in whole of arm and forearm. Reaction diminished in muscles of lower limbs. No numbness, or anæsthesia, or pain. Death July 19, 1872. [For details of autopsy see page 82.]

Case XV.[1] By M. Bernhardt. Male aged thirty-five years. Exposure to cold while perspiring, after exhausting diarrhœic discharges. In the course of forty-eight to seventy-two hours an extensive paralysis was developed, affecting the muscles of the extremities, unaccompanied by spasms, or by cerebral symptoms. There was no fever. In a few days the palsy of the extremities became absolute, and so remained for months. The affected muscles rapidly lost their electro-muscular excitability, at least for the faradic current, and much muscular atrophy ensued. Yet sensibility remained undiminished, the bladder and the sphincter ani were not paralyzed, no bed-sores appeared. The respiratory muscles escaped. Reflex movements were difficult to excite. Spine not tender; some spontaneous pain in back. Some return of movement in fingers in about three weeks, and afterward progressive improvement. Patient able to walk alone only after eleven

[1] Ueber eine der spinalen Kinderlähmung ähnliche Affection Erwachsener. Arch. f. Psych. u. Nervenkrankheiten, 1874. p. 370.

months. At an early stage pains occurred in all the limbs.

Bernhardt refers to two other cases brought to his notice by Profs. Traube and Westphal; but the details given are insufficient to justify their acceptance.

Case XVI. Personal. Male aged twenty-four years. Seen February 16, 1871. In early life was a healthy and temperate boy; never had syphilis. In 1865 was run away with on horseback, striking a tree with his left arm and side of head. Fell from the horse and remained unconscious for several minutes. Had no subsequent cerebral symptoms. In August of the same year, after having been repeatedly wet through while at work upon his farm, he noticed that his legs tingled and felt numb, and that his feet were heavy, so that he easily stumbled. In the course of three months the difficulty in walking was so great that he took to his bed. At that time he had no constriction around the body, no loss of feeling, no palsy of the bladder or of the rectal sphincter, no spasmodic movements. All the limbs tingled and felt numb, and were absolutely paralyzed. He had pain in the lower part of the back, and between the shoulders. Head was always clear, and the special senses normal. Took strychnia for a time without any effect, good or bad. In the spring of 1866 (?) began to sit up in bed, and in a chair. In the summer of the same year walked with help, and has since progressively improved. No bed-sore at any time. Examination shows nothing abnormal in any part above the legs. The thighs are strong; the legs are swung as wholes, the feet not moving in flexion. This is owing to extreme atrophy of the anterior tibial group of muscles. The extensors of feet act fairly, and

are but little below their normal size. The plantar muscles are evidently atrophied. No deformity exists. Sensibility in all its modes is perfect in lower extremities. The anterior tibial muscles do not respond to a strong faradic current, though it is "felt down to the bone." There is some sense of weight in lower part of back, and slight tenderness over lower lumbar vertebræ. At one time sexual power was abolished; it is now normal. This patient returned to his home in North Carolina, and has not been heard of since. My notes indicate that at the time of examining the patient I looked upon the case as one of congestion of the spinal cord.

After the publication of the original edition of this essay, I learned that Case X. of the essay was identical with the preceding case: Prof. Wm. A. Hammond having seen the patient two days after me. Dr. Hammond reported the case in full[1] in 1874, as one of progressive muscular atrophy, but in the last edition of his Treatise[2] he considers it as one of spinal paralysis of the adult.

Case XVII. Personal. Unmarried female aged twenty years. Admitted to the Epileptic and Paralytic Hospital, Blackwell's Island, in the author's service, November, 1871. The patient presents a paralyzed and extremely atrophied left leg, and gives the following imperfect history: The trouble began nine months ago, suddenly during sleep, with painful contractions. She then gradually (?) lost power in the left leg; no other limb being affected. The patient cannot state

[1] Clinical Lectures on Diseases of the Nervous System. New York, 1874, p. 147.

[2] A Treatise on the Diseases of the Nervous System. Sixth Edition. New York, 1876, p. 477.

how long a time elapsed between the first symptoms and the discovery of palsy. She adds that on the day before the attack, her left leg felt quite cold and a little numb; and that her menses were suppressed. No cause is apparent—no hereditary influence, injury, or syphilis. Examination: left foot is drawn in moderate *pes equinus*, with inward inclination. No voluntary movements below the knee. The patient's answers to the æsthesiometer test are unreliable; sensibility to painful impressions is somewhat impaired, that to temperature preserved; tickling is felt equally on both feet. Pressure shows tenderness over lumbar vertebræ; no spontaneous pain. The right calf measures 26.9 c. in circumference, the left 23.7 c. There is absolute loss of electro-muscular contractility in all the muscles of the left leg. The limb is very cold and its circulation feeble. I frequently called the attention of the resident staff and of friends to this remarkable case, as one of the same kind as that which, occurring in the early years of life, we call infantile spinal paralysis.

The subsequent notes need not be reproduced. No treatment did any good; the girl remained in the hospital without any active symptom, until Oct. 3, 1873, when she went away. She was then employed as a help in the wards of the Convalescent Hospital, on Hart's Island, and was there much exposed to cold.

The second attack, of which the patient gives a good account, came on late in December, 1873. Had pains "like rheumatism" in right leg; there was a feeling of pins and needles in the limb; numbness extending above the knee. She is positive that on the fourth day the right leg was completely paralyzed. No symptoms in wasted left leg. No bed-sore, and no affection of bladder or rectum. Re-admitted to the Epileptic

and Paralytic Hospital March 3, 1874, with palsy and atrophy of both legs; no acute symptoms.

During the spring and summer of 1874 the patient gradually lost strength in the thighs, most in the right. She also exhibited a variety of interesting visceral disturbances, consisting of amenorrhœa lasting two and three months; the menses then appearing with much pain, the blood being abundant and clotted; there was also pain in the back and lower abdomen. On many days during this period the urine had to be drawn off with a catheter, and it was often bloody, exhibiting a heavy mucous deposit, and containing albumen. The microscope showed only leucocytes and a variety of epithelial cells—there being probably both pyelitis and cystitis. After the middle of September she was better, and did not need the catheter; palsy unchanged.

Re-examined October 25, 1874. Patient, when she first came in this year, walked ill with a crutch and a stick; is now able to walk with two sticks (result of education). Cannot stand or walk without help. Patient is a stout and healthy girl, exhibiting nothing abnormal above the hips. Both lower extremities are extensively palsied and much wasted. The left leg (first attacked, in 1871) shows no voluntary movement below the knee, with exception of slight separation of the toes. As the patient lies on the bed she is able to raise the extended limb as a whole; but the strength at the knee-joint is small. The thigh is thin and flabby; the leg is the seat of extreme atrophy, and looks just like the same part in cases of infantile spinal paralysis, there being apparently only connective tissue and fat around the bones, the skin being bluish and very cold to the touch. The right lower extremity (paralyzed in 1873) is in a very similar though less

extreme state. All voluntary movements are possible with the foot, though they are feebly performed. The limb, as a whole, cannot be raised from the bed, and flexion at the knee-joint is weak. The quadriceps extensor femoris muscle is wholly paralyzed; the flexors of the thigh upon the body act feebly; the adductors fairly well. Both feet lie extended and adducted; toes flexed. The right leg is, like the left, extremely wasted, bluish, and quite cold. Sensibility to contact, pain, and temperature, is preserved in both limbs. Tickling is felt, but produces no reflex movements in palsied parts. Electro-muscular reaction (to both currents) is lost in the atrophied muscles of both limbs. At present the urine is passed normally. The patient's arms, shoulders, and chest, are large and rounded, standing in remarkable contrast to the dwindled legs. There have been no bed-sores, and no spinal epilepsy.

Circumference of right thigh (lower third)	31.5 c.
" " left " "	30.5 "
" " right calf	24.0 "
" " left calf	21.6 "
" " forearms	25.0 "

On a healthy girl (non-palsied) of nearly the same proportions as the patient, the following measurements were obtained:

Circumference of right calf	35.0 c.
" " left calf	34.5 "
" " forearms	24.0 "

The patient having been in bed some time, well covered up, has a thermometer placed between the great and second toes of each foot for three minutes, with the following results: Right side, 29° C., left side, 30° C.

CASE XVIII. Personal. Male aged about forty years, an American, and a bar-tender by occupation. Seen at South Shaftsbury, Vermont, in consultation with Drs. Rogers and Morgan, January 3, 1874.

In September, 1873, patient caught a severe "cold;" had pain in the bones, obstinate constipation, excessive vomiting; also severe cough with much expectoration. Relief was obtained by purging, after symptoms had lasted three weeks. On the second day of this attack he had suppression of urine; on the third day his face and feet were swollen, and this swelling lasted three or four weeks. At the end of the first week he experienced coldness in the legs as high as the knees; not beginning in any small part, most marked in calves. This coldness was objective as well as subjective, and lasted ten days, during which time there was no numbness, and only a doubtful stiffness of feet; he was up every day. Relieved by strychnia. About Oct. 16, was again at work, standing in a damp and cold bar-room. He had a feeling as if feet were "clumpy" (heavy?). Had slight numbness in thumb, index, and medius of both hands; enough to prevent writing. Late in the month feet again became cold and heavy, and in the course of two days the vomiting returned, lasting four days. The physician who treated him in Troy said that he had not Bright's disease. Patient returned to South Shaftsbury about Oct. 23. His feet were then weaker than ever, and he could barely walk up a slight hill. His brother-in-law, Dr. Rogers, thinks that the patient then had a degree of anæsthesia in the feet; he could still walk without a stick. The legs rapidly became weaker, so that in two weeks he could barely stand. November 6, patient ceased going down stairs, and a week later his legs were insensible

to all impressions but severe pain; limbs lost in bed. There was complete palsy of parts below ankles (muscles of leg paralyzed); movements of thighs good; no reflex movements or contracture; no disorder of rectum or bladder. Suffered much from a painful throbbing in soles of feet, most in right foot. Had slight fever. About Nov. 15, muscles of legs and feet began to waste. During the second week of November the median distribution in fingers became anæsthetic; he could use fingers supplied by ulnar nerves. The hands and forearms wasted rapidly. There was emaciation (almost atrophy) of thighs and arms. December 1, a degree of sensibility was discovered in the fingers, and in the feet a week later. Since there has been gradual improvement in voluntary movement and sensibility. The wasting has, however, continued to increase until now. On Dec. 21, motion was observed in toes. Has had no bed-sore; no sensation of constriction about the waist or elsewhere. In the last few days has had consciousness of location of all parts of lower limbs except toes. Throughout, the right side has been the more affected. Has had no cerebral symptoms.

Examination: No objective symptoms about head. The movements at shoulders and elbows are good. Patient grasps three kilo. with the right hand, and five kilo. with the left. The fingers appear normal except that their second and third phalanges cannot be extended. Opposes thumb fairly well, though he cannot make O with thumb and index. Interossei, though much wasted, still act a little. Thenar muscles (especially the opponens) are much atrophied. No deformity of hands while at rest. Co-ordinates fairly well. Æsthesiometer shows anæsthesia; its points being distinguished when separated by eight to fourteen mm. on

tips of fingers supplied by the median nerve, and by five to eight mm. on the parts supplied by the ulnar nerve.

The toes are in forced flexion. As patient lies he can raise legs high up, and bend knees well; can move all joints except those of great toes; though the movement of the other toes is hardly perceptible. There is only slight wasting of thighs, but the legs are much shrunken, especially in front. The right calf measures 25.25 cent. in circumference, the left calf 24.5 cent. The skin below the ankles is decidedly anæsthetic, slight contact being hardly perceived; the feet and legs are hyperalgesic, there being in the same parts a retardation in the perception of pain of from five to eighteen seconds. The temperature of the legs was warm until two weeks ago, since which time they have been cooling. After the first attack of vomiting had no sweating, except a very little about the head, until sensibility began to return nearly four weeks ago, when perspiration appeared everywhere. During illness, absence of erections until very lately. Throbbing pain in balls of feet has nearly ceased. The muscles of the legs respond faintly to faradism, with the exception of the extensor proprius pollicis which does not contract: all the muscles of the hand respond, those of the thenar eminences poorly. With galvanism all these muscles contract under an interrupted current of from twelve to twenty elements of Stöhrer. The left leg (less palsied) shows contractions with less faradic current and more galvanic current; while the contrary is true of the more palsied right leg. The optic disks are a little hazy (?), especially the left. Urine normal.

The patient has never had syphilis, was never injured, has never committed excesses in alcohol, tobacco, or

with women. His occupation of bar-tender obliged him to stand in a damp basement from five o'clock A. M. to midnight.

Diagnosis: Inflammation of the anterior horns of the spinal cord. I advised tonics, galvanism to muscles at present; to be followed in a few weeks by faradism.

On Feb'y. 28, Dr. Rogers wrote me that the patient "steadily improved since the time you saw him. He is now able to walk about his room by sliding a chair before him. Can raise himself from his chair by taking hold of a chair with his hands. His legs about the calves are now an inch and a half larger than when you were here. Sensation is I think much better." I since (autumn of 1874) learned that this patient wholly recovered.

CASE XIX. Personal. Male aged forty-seven years; an American, inventor by occupation, seen March 23, 1874. Patient is a very large, healthy man, who has worked hard at mechanical problems. In the last two or three years has had hæmorrhages from the lower bowel, and various dysæsthesiæ about the head; consisting in pain in upper part of neck and back of head, in a sense of pressure on the top of, and behind the head, etc., without impairment of intellect.

In October, 1873, he had a severe cold, which was followed by much coughing. The time of beginning of the paralytic symptoms is difficult to determine with accuracy, because numbness and paresis crept upon him so gradually. A few days before November 10, he had complained of a sense of coldness (not objective cold according to both patient and his wife), in his feet and legs. The numbness began in that week, appearing at about the same time in the ends of the upper and

lower extremities. On that day he went to Boston on business; he could walk with difficulty with the help of a cane, and the support of his son; he could fully dress himself. On Nov. 13, he returned, having more paresis, and remained at home. At this early date there was swelling of the feet. On Nov. 14, the legs and forearms were quite weak; the legs swollen and glossy; and the seat of subjective cold. Nov. 17, he could not leave his bed, or sit up in it. There was much formication extending up to the knees, and half-way up the forearms. No symptom about the head or eyes, no affection of bladder or of rectal sphincter. The feet were anæsthetic (?), though legs were not lost in bed. There were no reflex movements; no marked wasting of limbs; no fever; the œdema continued, the hands being a little swollen; the urine was "thick." He had a partial constricting band extending over the lower ribs on the right side, and a sense of tightness just above lines of numbness in lower part of thighs. He was dry-cupped and leeched on the spine. January, 1874: about the middle of this month more intense formication ushered in recovery. After a few days sensibility returned in part of feet, our patient was able to perform some movements. Toward the middle of February he began to sit up in bed, and in a chair; gaining daily. The numbness receded toward the extremity of the limbs; the feet remained a little swollen. Since that time he has gradually improved; he now walks a little with the help of a crutch and stick.

During the course of the illness there were some changes in the intensity of the symptoms from day to day, but not great ones. He had diffused spinal pain; and when at the worst, had a localized pain between the shoulders, relieved by cupping. There occurred some

degree of wasting in the parts below the knees. The subjective coldness continued until the numbness had almost ceased. In the second month of illness, and from time to time since, he had numbness in distribution of left superior maxillary nerve upon the face. The head-symptoms, which together with irritability of temper, had annoyed him so long, disappeared during illness.

Examination: Patient's general condition fairly good. Walks with a cane, without dragging or jerking legs; step is that of simple weakness. No facial symptoms. Moves arms in all directions. Movements of hands are good, except extension, which, almost complete for the fingers of the right hand, is far from good on the left side, especially for the thumb and index. Palmar muscles not wasted, only emaciated. Sensibility of hands to contact and pain good. Grasp equals 26 kilo. with the right hand, 16 kilo. with the left. A thermometer placed for three minutes in the fold between the thumb and palm shows 35.6° C. in the right hand, and 36.1° C. in the left. Co-ordinates well.

Lower extremities: All movements are possible, except that left foot is but slightly movable; the left toes can hardly be extended; those of the right foot move a little more. The toes are somewhat contracted in flexion. No wasting of any group of muscles in legs; co-ordination good. Sensibility to contact is normal as far as ankle, dull below that point, very dull on toes. The toes are the seat of numbness. The soles are sensitive. Can stand for a moment without cane and with eyes closed. Has no sense of constriction anywhere. Electrical examination shows very great loss of electro-muscular reaction to faradism in palsied muscles; almost complete in left extensor indicis, and in extensors of toes and flexors of foot on both sides; not

much reaction in calves of legs. The various muscles contract well under galvanism.

I advised the use of strychnia in small doses, to be increased; the application of galvanism and faradism; and exercise, with daily friction and passive movement.

It should be added that, when I examined the patient there was no œdema of the feet, and that the urine, although dense, was free from any sign of disease of the kidney.

A letter received from a member of the patient's family, on Oct. 24, 1874, speaks of Mr. H. as having almost wholly recovered; walking freely with the help of a stick, and carrying on his business.

Case XX. Personal. Male, aged twenty-three years, single, an American. Seen at Worcester, Mass., with Dr. Charles H. Davis, on June 29, 1874. Was a healthy, strong, and sober youth. In November, 1871, he went west, to Cincinnati, where he committed sexual excesses, though without contracting venereal disease. He then went out to Missouri, where he was employed as clerk, division superintendent, etc., being much exposed to the elements. In midwinter, "about two months after reaching Missouri," patient had an attack called by physicans "choreic palsy"; all his limbs being paretic and his walk like that of a drunken man. He had numbness in all his limbs; there was no palsy of the rectal sphincter or of the bladder; he could sit up, and walk with help. In the course of about two or three months (early in summer of 1872) he was cured of all except numbness and weakness of the left hand, by chloride of arsenic (?). About the end of this period of convalescence he had a week of sickness, caused by malarial intermittent.

In September, 1872, with his left hand still numb and weak, he went to Fond-du-Lac, Wis., settling there as a store-keeper. He had to travel somewhat, and had much financial responsibility. During the winter, strychnia was given him for the cure of his weak left hand, but he then grew worse, and the second attack of palsy developed.

He returned East, very much paralyzed in all his limbs, in May, 1873, the disease having made rapid progress in the preceding three weeks. When received at home, toward the close of May, his right extremities were quite powerless; those of the left side could still be used; and imperfect walking was possible when he was supported. He could sit up on the edge of the bed or in a chair. He then walked and swallowed well; had no strabismus, or impairment of sight. He had the sensation of a band around the body at the umbilicus, and this feeling afterward extended higher up.

There was much numbness in all the extremities; anæsthesia was doubtful. There was slight muscular atrophy. No sign of spinal epilepsy (jerking or stiffness of palsied parts). Constipation was quite obstinate; no palsy of bladder, though he was forced to empty the viscus quickly after a sense of distention appeared. During the summer the patient grew progressively worse; in the autumn he could not walk with help, and later on, sitting up in bed became impossible.

About November 27, 1873, there appeared external strabismus of the left eye and diplopia; also paresis (?) of the facial muscles.

In January, 1874, a degree of spontaneous improvement was observed. The patient was once more able to sit up on the edge of his bed, and could put his hands

up to his head, especially the left; the strabismus disappeared, though the left pupil remained wide.

In February, Mr. B. again lost ground, nearly complete palsy existing; speech was lost quite rapidly (not suddenly); swallowing became difficult, and strabismus reappeared.

June 29, 1874. In the last two months patient has lain in bed almost completely paralyzed in limbs, face, eyes, tongue, and throat; the chest-walls and diaphragm escaping. The muscles of the extremities have steadily wasted, and deformities have appeared in hands and feet. The difficulty of deglutition has been in the case of solids, fluids not having been regurgitated through the nose. At times has been anæsthetic (?) to pinching in feet and legs. Has had a good deal of spinal epilepsy (tonic and clonic spasms in the palsied parts following any irritation). Has had a sense of tightness about the chest and belly. Has of late passed urine and fæces involuntarily, though rarely unconsciously. Much muscular wasting. Has often complained of dim as well as of double vision.

Examination: Patient lies quite helpless on his back; when he is turned spinal epilepsy appears in the whole body. The possible voluntary movements are slight motion of the left great toe, and of left fingers. His sisters say that the day before he could raise both hands a little from the bed. Eyes are moved imperfectly, and in a quasi-ataxic way in every direction except that prohibited by palsy of the third nerve. There is no ptosis, but the pupil is widely open. The ophthalmoscope shows the fundus without marked lesion, though the disks are, perhaps, abnormally white. The face is a mask, though the upper facial muscles act fairly, and patient can purse lips. He cannot whistle, or make

any articulate sound—he utters only a low grunt. The tongue can be protruded only as far as the teeth; it lies undeviated in the floor of the mouth, atrophied and much shrivelled (folds longitudinal) in its anterior half. The intercostal muscles and the diaphragm act well; respirations are fairly deep—twenty-four per minute. No bronchitis has occurred.

The state of sensibility is difficult to examine, because patient can make few signs. Pinching is surely felt everywhere. The muscles of the palsied parts are very much wasted; those of the tongue, forearms, legs, face (?) especially. The hands show the deformity known as *main-en-griffe*. The thenar eminences are much atrophied. In the feet there is partial *pes equinus* with toes flexed. No ulceration has appeared anywhere. The atrophied limbs are cool (the thermometer standing at 31.6° C. in the shaded room). All the atrophied muscles respond, though feebly, to the faradic current.

Diagnosis: Myelitis or degeneration of the anterior horns of gray matter of the spinal cord; the motor tract being involved from the third cerebral nerve down, with probably recent extension of myelitis to deeper parts of the cord at some points.

October 20, 1874. A letter from Dr. Francis, of Worcester, states that the patient is substantially in the same condition as in June; the only change being some dementia (?).

Case XXI. Case in the practice of my friend Dr. T. A. McBride, of this city; seen in consultation October 23, 1874. Male, aged twenty-eight years. Has in the last few years led a very fast life, drinking a great deal, and committing sexual excesses. Some time ago had a soft chancre, but never had any secondary symp-

toms. Has beginning pulmonary phthisis. From August to date has had more or less subacute articular rheumatism; knees and ankles most involved.

Ten days ago (October 13), patient first noticed numbness in feet, gradually extending up to knees. Had some cramp-like pains in legs. Two days later, he observed that the tips of all fingers were numb; those supplied by the median nerve being most affected. First paresis occurred one week ago, three days after first numbness. He noticed that the left great toe could not be extended; since has had a dragging and staggering gait, and has remained in bed. To-day he discovers that extensor muscles of fingers are weak; the left middle finger dropping much below the level of the others during extension. Has no pain in the back; no rectal or vesical symptoms. He complains much of coldness of the legs.

Examination shows patient to be a nervous, rather delicate subject, with so much pulmonary disease as to give a nearly uniform daily fever of .8° C. He lies in bed, but is able to sit up, and can move his legs in every direction, though feebly. The left great toe and left middle finger cannot be fully extended. Extension of the hands and fingers is incomplete and weak. Flexion of feet upon legs is weakly performed; the strength at the knee-joints is reduced. The anterior aspect of both legs, and the extensor surface of both forearms are evidently wasted. Tested with the faradic current, the anterior tibial muscles (those of the right side more) show diminished reaction; the left extensor proprius pollicis not responding at all. The extensor muscles on the forearm have also lost much of their excitability, those of the left side especially. Sensibility is not impaired in the fingers. In the lower extremities, below

the middle of the legs, there is marked anæsthesia to simple contact and to the æsthesiometer, with some errors in localization of impressions. Pain and temperature are perceived everywhere, though the former sort of impression is perceived only after a retardation of several seconds, and the sensations persist for several seconds. The right calf measures 28 c. in circumference, the left 27.5 c. The patient has "lost his legs" a few times in the last two or three days. There have been no reflex movements, and it is difficult to provoke them now; no affection of the bladder or sphincter ani; no threatening of bed-sore; no cerebral symptoms; no weakness of thoracic and abdominal muscles. The patient's legs are cold, objectively and subjectively. He has no feeling of constriction around any part of the body. The spine is not tender.

I diagnosed subacute spinal paralysis, and advised counter-irritation to the back, and the internal administration of Squibb's fluid extract of ergot in large doses.

On November 1, I again saw the patient. He has in some respects improved. The anæsthesia of the lower limbs has decreased, and there is almost no retardation of impressions. Some feeling of tight band about knees at times. The legs have lost about .5 c. in circumference, and voluntary movements are as before. Fibrillary contractions are abundant in the anterior tibial muscles. The upper extremities are worse than ten days ago. The numbness extends up to second joint of fingers, and on the radial side of each index finger there is much tactile anæsthesia. General condition is rather better. No affection of inspiratory or expiratory muscles; no vesical or rectal weakness; no bed-sore. Advised to apply dry cups to the back, and continue Squibb's fluid extract of ergot in doses of ℥j.

a day, with quinia sulph. gr. v, in the morning; nutritious food, cod-liver oil, and stimulants.

On December 9 I saw the patient again with Dr. McBride. Sensibility is perfectly restored, except on the radial border of the left index finger, where slight anæsthesia remains. All voluntary movements are now possible. The muscles are firmer; the patient has gained flesh; his calves measure 29.25 c. This patient was severely dry cupped, and for awhile took as much as ʒj of Squibb's fluid extract of ergot with enough belladonna to affect his throat slightly. No unpleasant symptoms attended the taking of such large doses of ergot. No advance has occurred in the pulmonary affections.

In a few weeks after the above visit Dr. McBride reported that all paralytic symptoms had passed away.

Case XXII.[1] By Lucas Championière. A woman aged twenty years, was admitted to the service of Prof. Jaccoud, February 29, 1870. It was learned that eighteen months previously, a few days after confinement, she experienced a sudden complete paralysis of all the limbs. There was some pain, not well described by patient. She was a long time confined to her bed in a state of general paralysis. Recovery took place in arms, left leg, and lastly in right leg, very slowly and not perfectly. There subsequently occurred a relapse of palsy in the left leg, and this has persisted.

Examination on admission shows that there is extreme paralysis of the left lower extremity; the extensors of toes only acting, and that feebly. The paralyzed muscles are very much atrophied, and have completely

[1] Cited by Hallopeau, Sur les myélites chroniques diffuses. Archives Générales de Medecine, 1871–1872, p. 70. Obs. G.

lost their electro-muscular contractility. Walking is impossible. Sensibility is preserved. There is some dull pain in the palsied limb. A little improvement was brought about by the use of sulphur baths and of the faradic current.

The patient died of typhoid fever in October, 1870. On post mortem examination, the muscles of the left lower extremity, except those of the anterior part of the thigh and leg, were found pale and fatty; all the muscular fibres being small, and some of them filled with granulations. The nerve going to the gracilis muscle contained many empty nerve sheaths, and numerous fatty granulations. Some of the anterior nerve roots were small and grayish.

The spinal pia mater was injected; the superficial part of the spinal cord seemed normal. Sections made through the cervical region and the upper two-thirds of the dorsal region showed nothing abnormal. Sections through the cord just above its lumbar enlargement and in parts lower down showed the left anterior horn of gray matter to be dark brown in color. In the middle of the enlargement both anterior horns were brownish and diffluent.

Case XXIII.[1] By Prof. Kussmaul. A man aged twenty-eight years, previously healthy, was attacked August 8, 1864, with fever which lasted several days. He experienced weakness in the legs, pain in the back radiating to the epigastrium and sacrum; and later had pain in shoulders, arms, and chest. On the fourth day there appeared paralysis of all the extremities and of the

[1] Frey, Ueber temporäre Lähmungen Erwachsener, die den temporären Spinallähmung der Kinder analog sind, und von Myelitis der Vorderhörner auszugehen scheinen. Berlin. klin. Wochenschrift, 1874, p. 14.

back. Atrophy rapidly set in and was great. The dorsal pain soon ceased, but that in the limbs lasted more than eight weeks.

On October 8 patient was admitted to the clinic at Freiburg. The cerebral functions were well performed; there was no disturbance of sensibility, no bed-sore had appeared although the patient had lain two months on a hard bed, and the rectum and bladder performed their functions well. There was extensive muscular atrophy, affecting many muscles of the shoulders, spinal region, hips, and lower limbs, and there was everywhere diminution of faradic reaction. In some muscles this reaction was absolutely lost, as well as the galvanic. Pressure on muscular masses caused pain.

Faradism, warm baths, and nutritious food brought about recovery of all the muscles except those of the left calf and the serratus magnus. The left leg remained blue and cold.

A portion of the left calf excised showed muscular fibres in a state of marked granular and waxy degeneration, the capillaries fatty, and the tunica media and the adventitia of small arteries thickened.

This case was considered by Kussmaul at the time of its first publication,[1] as one of myopathic disease.

Case XXIV.[2] By Prof. Kussmaul. A previously healthy girl eighteen years of age, experienced lassitude, pain in the back and legs, after refrigeration in July, 1870. Patient states positively that there were no cerebral symptoms; no chill or fever; no sense of constriction about the body. The severe pain in the back and legs passed away in three weeks. In the course of eight

[1] Deutsches Archiv f. klin. Medicin, 1866, Bd. I., p. 506.

[2] Frey, loco cit. p. 15.

days complete palsy of both legs occurred without affection of the rectum or bladder, and without disorder of sensibility. Rapid wasting of the muscles of the legs occurred. No material change took place until patient's admission to the Freiburg clinic in June, 1871.

Examination then showed good general health; no symptoms of cerebral disease; no disorder of any internal organs; no palsy of sphincter ani or of bladder; no trace of bed-sore. Both legs are symmetrically atrophied and are cold. Sensibility normal; deep pressure on muscles is felt, but is not painful; on the calves a difference of one hundred grammes in weight is readily distinguished. Tickling soles of feet produces reflex movements. Feet lie in equino-varus position. A few voluntary movements are possible in the legs. Faradic reaction is diminished or lost in various muscles. The effect of galvanism is not noted. Much atrophy of extensor muscles of spine. No atrophy or palsy above level of epigastrium.

Systematic electrical treatment did no good to this patient.

Case XXV.[1] By Prof. Kussmaul. A factory woman, aged thirty-three years, the mother of one child, was seized, while at her usual occupation, on November 29, 1872, with severe pains in both arms, soon followed by sharp pain in the hip and legs. There was neither head nor back-ache; no chill or fever on this first day. The next morning there were the same pains and general weakness. December 1, the only complaint is of cutting pain in left shoulder and great weakness. On the morning of December 2 the patient was found with a red face, an expression like that of typhoid fever. She

[1] Frey, op. cit., p. 15, et p. 28.

complained of severe headache, dizziness, stupor, and of great weakness: she had fever. Her temperature was 38.8° c., pulse 100. In the evening, prostration was greater, the temperature was 40° c., pulse 120. December 3, patient slept, but still complains of headache and prostration. Morning temp. 39.2° c., pulse 100. Evening temp. 39.4° c., pulse 120. December 4, symptoms about the same; there are still redness and turgescence of the face, stupor, but less headache. No special symptoms of meningitis or typhoid fever. Morning temp. 38.3° c., pulse 108. Evening temp. 39.4° c., pulse 126. December 5, no complaint except of lassitude; urine passed in bed during the past twenty-four hours. Patient is placed in a tepid bath and a cold effusion made on the head, with effect of immediately dispelling the stupor. Morning temp. 38.4° c., pulse 92. Afternoon temp. (before bath) 39.3° c.; evening temp. 38.4° c., pulse 120. December 6, slept well; head is clearer. Morning temp. 38° c., pulse 92. Evening temp. 39° c., pulse 120, respiration 28. December 7, much better; no cerebral symptoms; morning temp. 37.4° c., pulse 106; evening temp. 38° c., pulse 110. Urine non-albuminous.

On and after December 8 fever is absent, but the pulse remains high, ranging from 84 to 100. Internal functions are well performed; bladder and rectum act well. It is now discovered, to the great surprise of the physicians, that patient is much paralyzed in all her extremities and in the hips. Sensibility is nowhere impaired; but complaint is made of pain in the calves and in shoulders.

A careful examination on December 14, showed no disturbance of function about the head, face or neck. Sensibility is everywhere preserved. Tickling soles of

feet produces reflex movements in the lower extremities. There is no trace of bed-sore, and the rectum and bladder are not palsied. There is extensive partial paralysis of the arms and legs, with contracture of the gastrocnemii. These muscles show fibrillary contractions. Galvanic and faradic reaction is much impaired in the lower extremities, very little in the upper. There is no evident muscular atrophy.

After December 21, a degree of wasting showed itself in the muscles of the calves of the legs, especially the right, and by December 30, was marked. In March, 1873, the left deltoid is stated to be much atrophied.

Under galvanism and supporting treatment improvement began and continued. At the beginning of March patient could walk without canes. On July 10, 1873, great improvement is noted; much improved faradic and galvanic reaction, even in gastrocnemii and left deltoid. Patient cured.

Case XXVI.[1] By Prof. Kussmaul. A well-grown girl aged seventeen years; not inheriting any tendency to nervous disease. In March, 1872, an unexplained attack of mydriasis occurred, cured by physostygma. First menstruation at fifteen. Patient in summer did light work in the fields, and in winter house-work and sewing. In March, 1873, weakness of the hands showed itself without cause. This weakness was especially noticed while knitting. No other symptoms at that time. During the summer hands grew stronger, but in September they rapidly became weaker; and the paresis invaded the forearms and arms, so as to make

[1] Frey, Ein Fall von sub-acuter Lähmung Erwachsener,—warscheinlich Poliomyelitis anterior subacuta. Berlin. klin. Wochenschr. 1874, p. 549, p. 566.

elevation of arms impossible. In the second half of September weakness appeared in the legs and back. There also appeared numbness and formication in arms and legs, but no true pain. Movements of head and neck were unimpaired; respiration, circulation, defecation and micturition were well done. No general symptoms.

By the end of September the maximum degree of paralysis was reached, and continued for about two months. During this time there gradually occurred atrophy of the muscles of the hands (especially the thenar and hypothenar groups), those of the forearms and legs. The arms, thighs, and nates did not undergo appreciable wasting.

Patient was admitted to Prof. Kussmaul's service in January, 1874, after having been treated by galvanism for six weeks by Dr. Schmidt. She was already improving. Examination showed no fever, no abnormal sensations, no disorder of respiration, circulation, and defecation. There is much paresis of arms, legs, and back. Marked atrophy of some dorsal interossei, of thenar and hypothenar muscles has occurred. In paretic and atrophied muscles there is great diminution of galvanic and faradic excitability. In a few muscles of the hand the strongest current produces no contraction.

On February 28 patient is again examined, and is made the subject of clinical remarks. Much improvement has taken place: patient can walk for half an hour, and ascend stairs; she can do delicate work (writing) with her hands. Has lordosis when standing; the plumb-line from shoulders dropping outside of sacrum. Forearms are bluish and cold. There is still an enormous diminution of electrical excitability. Sensibility normal, except that electrical sensibility is much dimin-

ished in forearms, legs, hands and feet. Rectum and bladder act well. Reflex movements of limbs normal. Menses regular. Electrical treatment continued. At the beginning of July patient is discharged about well; but it is remarkable that some muscles which are now under the control of the will have not recovered their excitability to either current.

The case was considered by Kussmaul and Frey to be one of myelitis anterior subacuta, or subacute spinal paralysis of the adult.

Case XXVII.[1] By Prof. Erb. A man, aged forty years, was much exposed to dampness. On September 5, 1873, there appeared severe cutting pains in the back and both legs, quickly followed by marked paralysis of both legs. On the same day there appeared chill, fever, delirium at night; and during three days retention of urine. Sensibility was in no wise impaired. In two months pains ceased, but muscles remained very tender to pressure, and began to waste. Gradual partial recovery of voluntary power. No bed-sores.

Examined May 12, 1874. Extensive atrophic paralysis of both legs. They are cold and livid. Patient can stand alone, and closing eyes does not cause staggering. In the wasted muscles there is great reduction and even loss of reaction to the faradic current, when applied directly to the muscular masses or through the motor nerves. Galvanism produces reaction in most of the muscles and nerves when applied in the same way. Reaction of degeneration in left leg. Active treatment produced only a little improvement.

Case XXVIII.[2] By Prof. Erb. A man aged twenty-

[1] Arch. f. Psych. u. Nevrenkrankheiten, V. p. 767. [2] Loco cit., p. 771.

two years. At the age of fourteen severe headache, fever, and delirium. In the third night during subsidence of fever patient discovered that both his feet were paralyzed. During the day an incomplete paraplegia was developed. There was numbness in the legs, but no loss of sensibility. In the course of six weeks improvement began.

Examined December, 1866. Patient exhibits atrophic paralysis of both legs, most marked on left side; the muscles on the anterior part of the left thigh and the left calf are extremely wasted. Sensibility is perfect. In wasted muscles the faradic current produces only traces of contractions, the right tibialis anticus not reacting at all. Similar results are obtained with galvanism. Galvanic treatment continued for a few weeks did no good.

Case XXIX.[1] By Prof. Erb. A girl aged sixteen years. Four months previously, after refrigeration during menstruation, was seized with headache, fever, pain and stiffness in the right leg. When she attempted to get up, in five weeks, she discovered that the right leg was completely paralyzed. She had not had numbness or formication, and sensibility was in no wise impaired. Gradual improvement was produced by baths and faradization.

Examined January 7, 1869. Patient's left leg is the seat of paresis and paralysis, and many of its muscles are atrophied. The muscles on the front of the leg have suffered most. Sensibility is perfect; function of bladder well performed; no alteration of nutrition of skin; menses regular. Faradism produces only slight contractions in the muscles of the right calf, none at all in

[1] Loco cit., p. 773.

the tibialis anticus and extensor digitorum communis. With galvanism, nearly the same result is obtained; except that feeble and slow contractions can be obtained by direct galvanization of the tibialis anticus, and ext. digit. com. A long-continued treatment brought about recovery in all the muscles, except the tibialis anticus.

CASE XXX.[1] By Prof. Erb. A man aged thirty-four years, came under observation June 3, 1868. Four weeks previously, he was suddenly taken ill, with chill, fever, headache, vertigo, constipation, and great general prostration. These symptoms passed away in a few days, but there soon followed a second similar attack. Toward the end of the second week, patient noticed that his right arm was paralyzed, and that its joints were painful. There was no other palsy, though he was so weak that he could not walk without help for some time. The right arm remained paralyzed. No affection of bladder; no disorder of sensibility.

When examined, the right shoulder and upper arm are seen to be smaller than the left. Deltoid, biceps, triceps, and coraco-brachialis, are entirely paralyzed. Other muscles of arm and forearm, are more or less paretic; no fibrillary contractions. Faradic and galvanic contractility is more or less reduced in all the palsied muscles. When seen five months later, marked atrophy had occurred, especially in the arm and shoulder, and the electrical reactions were unchanged.

CASE XXXI.[2] By Prof. Erb. A man aged thirty-seven years; seen Feb'y. 6, 1867. He states that in the summer of 1863, he felt a weakness in the left foot, and

[1] Loco cit. p. 777.

[2] Loco cit. p. 788.

for eight days had pain in the left hip. There was also occasional back-ache, and later there were spasms and shooting pains in both legs. In 1865, weakness appeared also in the right foot. He had no formication, and it was only last winter that he began to feel numbness in the toes. Frequently, the feet were œdematous toward evening; the walk was slow and laborious, and he could not jump. Of late the arms have been weak. The functions of the bladder and rectum have been well performed.

Examination shows partial paraplegia; no staggering when eyes are closed. The muscles of the anterior crural region are fairly active; those on the posterior aspect of the thigh paretic. Below the knees, the anterior tibial muscles are very paretic, and the peronei completely paralyzed. All the palsied muscles are flabby and atrophied. Sensibility to contact is normal in the palsied parts, but impressions of pain and temperature are not well perceived on the dorsum of the feet; and the same parts are numb. Faradic contractility is preserved in the muscles of the thighs, but reduced and even lost in those of the legs. Galvanism yields almost the same results, except that in some parts the contractions obtained are slow and feeble. Galvanic treatment continued nearly six months removed the anæsthesia and numbness, and brought about some improvement in the paralyzed muscles.

Prof. Erb considers this case to be one of poliomyelitis anterior chronica; a class which he thinks future research may justify.

Case XXXII.[1] By Professors V. Cornil and R.

[1] Cornil et Lépine, Sur un cas de paralysie générale spinale antérieure sub-aiguë, suivi d'autopsie. Gazette Médicale, 1875, p. 127.

Lépine. The patient, a man aged twenty-seven years, was admitted to the service of Prof. Sée, May 7, 1873. The antecedents of the patient are good; there being no hereditary predisposition to paralysis, and no syphilitic infection. It is learned that in childhood the patient had an attack of transitory paralysis (?) of both lower extremities. During the siege of Paris he was greatly exposed to cold, and in December, 1870, there appeared weakness in the right lower limb, with fibrillary contractions in its muscles, but no pain. In September (1871?) the same symptoms showed themselves in the left leg. In May, 1872, there was much greater weakness of legs; patient needing the aid of two canes to walk. In the winter of 1872–'3 the sacro-lumbar muscles became affected, and patient was obliged to remain in bed. The arms were not involved until January, 1873.

On admission: general condition is good; the "great functions" are well performed, and there is no loss of virile power. Arms and legs are paretic—the arms less so. There is general relaxation; no pain; sensibility is perfect everywhere. The paralyzed muscles are generally and uniformly atrophied, but the sub-cutaneous fatty layer is well-preserved. No marked deformities exist. In all the palsied muscles faradic contractility is diminished: in a few quite lost. Some fibrillary contractions are visible. Bits of paralyzed muscular tissue examined show slight granular deposit with preservation of striation in fibres.

The paralysis gradually increased, and in May (1873?) dysphagia and other symptoms of lesion of the medulla oblongata showed themselves. Death occurred through asphyxia on November 23, 1874.

The autopsy was made twenty-four hours after death.

The cerebrum and cerebellum appeared absolutely healthy. The spinal cord also seemed healthy to the naked eye except at its lowest part, where for a distance of 8 cent. above the filum terminale it was visibly softened. There was also atrophy of the anterior roots of spinal nerves. After hardening in chromic acid, microscopic examination showed that the ganglion cells in the anterior horns had undergone degeneration, leading in many parts of the lumbar and cervical enlargements, to their disappearance. There was also some diffused myelitis in the lumbar enlargement and slight descending degeneration. [For further details of the autopsy see p. 84].

Case XXXIII.[1] By Soulier. A man aged fifty-seven years, a weaver by occupation, was admitted to hospital September 11, 1874. He had never had syphilis. Twenty days before admission, he had noticed difficult micturition. In eight days, his legs became weak, first the right. On and after the eleventh day he was obliged to remain in bed. Once he had "cramp" in one leg. The articulations of the lower extremities were painful. September 8, right arm became weak, and soon after the left. On admission, shows no impairment of speech; pupils are normal; swallowing a little difficult; arms are weak; legs are quite paralyzed. Reflex movements can be provoked. No fibrillary contractions are seen; and sensibility is preserved. There is prickling in the legs. Faradic reaction is lost in palsied muscles. Later, dysphagia became greater, micturition more difficult, and the anal sphincter weak (there was not retention of urine, or positive incontinence of fæces). The actual

[1] Observation de paralysie ascendante aiguë avec guérison. Lyon Médical, 1875, No. 6.

cautery was repeatedly applied to the spinal region; extract of belladonna, and iodide of potassium (ʒss to ʒj per diem) were administered internally. Until September 24, there was fever sufficient to cause an elevation of one and two degrees C., each day. A number of joints in the lower extremities were swollen and tender, and one leg was swollen; no bed-sore. September 19, moved his toes. Afterward, gradual improvement occurred. On November 13, patient leaves hospital quite well. No mention is made of atrophy of muscles in this interesting observation.

Case XXXIV.[1] By Dr. D. F. Lincoln. A male, aged forty-nine years, of previous good health; without history of injury, syphilis, or excesses. His father and sister had died of cerebral hæmorrhage. On August 6, 1873, numbness appeared in legs; going away and returning several times, with at last weakness. In the course of the afternoon, almost complete paraplegia developed. Two days after, when first seen by Dr. L., there were noted: no paralysis of any muscles about the head, no cerebral disturbance; vision, hearing, and speech natural.

The muscles of the neck and limbs, except below the knees, were in a condition of semi-paralysis; but there was no absolute paralysis of any muscle. Below the knees full power remained. Paresis greater on the left side. Numbness had disappeared; there was no sense of constriction, no muscular twitching; reflex movements difficult to produce. Sensibility in various modes normal in hands, nearly so in feet. No pain or tenderness anywhere. The bladder and rectum acted normally, swallowing and respiration well performed.

[1] A case of Spinal Paralysis in an adult, resembling the so-called Infantile Paralysis. Boston Med. and Surg. Journal, 1875. p. 339.

On the fifth day, an electrical examination was made. There was found diminution or absolute loss of reaction to the faradic current in the palsied muscles, and marked diminution of reaction to galvanism. Gradually muscular wasting appeared in various paralyzed muscles, especially in the balls of the thumbs.

Under electrical treatment and gradual exercise, recovery took place in the course of six months.

Case XXXV.[1] By Lemoine. A woman aged forty years. Admitted to hospital December 5, 1874. Her previous health had been good. Four months before admission she noticed prickling in legs and feet, and experienced some pains (not fulgurating) in various parts of the legs. They soon became weak, and grew weaker. She had fever with evening exacerbation. Eight days before admission walking became impossible. On admission there is complete paraplegia; the muscles are flabby and much atrophied; the feet extended and in the valgus position. Sensibility is normal. No reflex movements can be produced. There is complete loss of reaction to the faradic current in the paralyzed muscles. Bladder and rectum are normal. December 8, right arm is weak. December 12, catheter used; right arm is weaker; left arm feeble. December 15, patient can not turn in bed; right arm is completely paralyzed, left nearly so; there is loss of faradic reaction in nearly all the muscles of the four limbs. Sensibility, bladder, and rectum normal. No bed-sore. December 16, speech and deglutition are difficult. At midday severe burning and tearing pain in both heels. The treatment up to this time had been

[1] Paralysie spinale de l'adulte: guérison. Lyon Médical, 1875, No. 15.

expectant. To-day the actual cautery is extensively applied to the spinal region from the vertebra prominens to the sacrum. December 20, improvement; pains have ceased, left arm is stronger. January 5, the right foot (last palsied) shows slight movements; arms are much stronger.

From this time recovery slowly advanced and became complete. At the time of last note, patient is well except that her muscles are still small, and she is easily fatigued.

Case XXXVI. Communicated by Dr. George M. Beard. A married woman aged thirty-five years (seen May 25, 1872), was attacked in the preceding winter with a feeling of numbness in both lower limbs. At that time she passed much of each day in the basement of her house; which basement was somewhat damp and cold, and the weather at that time was most severe. Previous to the occurrence of numbness she had been in good health, and there was no history of nervous disease in her family. At the same time with the numbness, attacks of vomiting came on. The limbs below the knees were quite cold. About four weeks after these symptoms (the precise time not ascertainable) the patient was obliged to take to her bed on account of a gradual loss of power over the lower limbs. At that time there was no affection of the bladder or rectum, and no other symptoms, as far as I could learn, beyond a feeling of cold and numbness, vomiting, and gradual loss of control of the lower limbs.

I found her in bed, able to move her lower limbs with some difficulty, but unable to stand upright, even with assistance. The muscles of both legs were atrophied; about equally so on both sides. There was no

anæsthesia, but rather hyperæsthesia. The patient still complained of coldness in the legs. There was no loss of the sense of pain, no bed-sore, although the patient had been so long in bed, most of the time upon her back. The sphincter ani was unaffected, but of late there had been involuntary flow of urine.

Examination with the faradic current showed very great loss of farado-contractility in the muscles of both legs, both above and below the knees; very strong currents, however, applied over the motor points, caused moderate contraction of the tibialis anticus and peronei muscles, and only a trace of contraction in the quadriceps extensor femoris. Examination with the galvanic current showed also diminution of galvano-contractility. There was no tenderness or pain in any part of the back, no exaggerated reflex action, and no muscular contractions.

The left arm was somewhat weakened, but the muscles were not seriously affected. Treatment consisted mainly of applications of galvanism, which, up to the time of making last notes of the case, had produced some improvement. The patient soon passed from under Dr. Beard's observation.

Case XXXVII.[1] By Prof. E. Leyden. A man aged thirty years, observed in the clinic at Königsberg, in December, 1865. A sharp febrile illness, during the early part of October, attended by severe headache and delirium, was followed in eight days from its beginning, and two days after its cessation, by weakness of the right arm, soon followed (same day) by weakness of the right leg. The next day, there was paralysis of all extremities; but fever had not returned, and there were

[1] Klinik des Rückenmarks-Krankheiten. Berlin, 1875. Bd. II. p. 198.

no cerebral symptoms. On the eighth day of the paralysis, the bladder became paralyzed, and the catheter became necessary. There never was pain or disorder of sensibility.

The patient attributes his disease to a cold swimming bath.

The examination by Leyden, showed absence of palsy about the face; paretic and partially atrophied extremities. Faradic contractility is much diminished in all affected muscles, and in many quite lost. Electrical treatment and systematic bathing, only partially restored the patient; when last seen, there were still atrophy of muscles in the upper extremities, and secondary contractures at the shoulder and knee joints. Leyden also relates at length, under the same head,[1] a remarkable case which I cannot look upon as one of anterior myelitis. After severe and repeated attacks of headache, the right limbs *suddenly* became paralyzed, without loss of consciousness. When examined ten months afterward, there was found paresis of the right arm and leg, with marked muscular atrophy and diminution of reaction to the faradic current, with slight passive contracture. There was no sign of palsy about the face, and the bladder was unaffected. Leyden looked upon the case (in the notes) as one of double hæmorrhage in the spinal cord; clots having formed in the right half of the anterior horns in the cervical and lumbar enlargements.

In view of the facts (1) that patients often fail to notice paralysis of the face in common hemiplegia, (2) that there was contracture, and (3) that Leyden's examination was made ten months after the appearance of paralysis, I am disposed to consider the case as one

[1] Op. cit. p. 199.

of cerebral hæmorrhage (patient aged forty years), followed by a descending secondary degeneration extending to the anterior gray horn on the right side of the spinal cord. This form of secondary or deuteropathic atrophy, after cerebral lesion has been observed by Charcot.[1]

Case XXXVIII.[2] By Prof. W. A. Hammond. Rose P., aged twenty-seven years, seen in the autumn of 1870. At the time, Dr. Hammond looked upon the case as one of spinal congestion. In May, 1870, menses suppressed; in July, deep dull aching pain in both legs, followed soon by severe pain in the back. There followed loss of motility, numbness, and anæsthesia, in both legs; and in two months she was unable to walk. At first there was constipation, but this was followed by incontinence of the rectum, lasting two weeks. Numbness in hands and fingers, as well as in legs. The palsy rapidly extended to the upper extremities. Intensity of palsy varied from time to time; was worse after rising in the morning. On examination, both legs are paretic, right weaker. Tactile sensibility is greatly impaired in both lower extremities, more on thighs. Hands weak, left weaker. Prickling and numbness still present in all extremities. Bowels constipated; bladder normal. Electro-muscular contractility and sensibility are greatly diminished. No band around waist. No reflex movements in legs. Muscles of legs wasted; the temperature of palsied parts is lowered. No syphilis.

Treatment by means of electricity and ergot, brought about a complete cure.

[1] Leçons sur les maladies du système nerveux, Paris, 1875, p. 55. Note.

[2] A Treatise on the Diseases of the Nervous System, Sixth edition, N. Y. 1876, p. 475.

Dr. Hammond next relates the history of the same patient, whose case I have numbered XVI.

Dr. Hammond also gives[1] brief abstracts of two cases, which, at the time of observation, he considered as examples of antero-lateral sclerosis. The histories given are so meagre that they cannot well be used in this essay. The symptoms were, gradually developed paralysis with moderate wasting of muscles; little or no anæsthesia; absence of palsy of the bladder. Another case shortly mentioned, was remarkable in the fact that the gradual palsy and atrophy appeared at first in one lower extremity. Ultimately, the paralysis became generalized, the medulla oblongata affected, and the patient died of asphyxia.

Case XXXIX.[2] By Prof. Hammond. A man, aged thirty-five, after rising one morning, felt a slight degree of weakness in both lower extremities, which gradually increased, so that by night he was unable to stand. The next morning he felt a similar weakness in both arms, and in a few hours was deprived of their use. When seen on fourth day, there was complete paralysis of all four limbs. There were no aberrations of sensibility, no paralysis of the bladder or sphincters, no motor spasms. Reflex excitability was abolished in all the paralyzed parts, and the electro-muscular contractility was greatly diminished, especially in the muscles of the legs. There had been slight fever. No history of syphilis.

On fifth and succeeding days there was observed a difficulty in respiration and deglutition, and in the movements of the tongue. On seventh day sudden double facial palsy.

[1] Op. cit. pp. 478 et 479.

[2] Op. cit. p. 485.

The treatment consisted in the administration of the iodide of potassium and ergot; the latter in doses of two drachms four times daily. Galvanism and faradism were also used. There was slight atrophy of the muscles of the calves.

Improvement began on the ninth day, the muscles of the face and throat acting better. The patient gradually grew better, and at the end of a year was perfectly well.

Case XL.[1] By Prof. Hammond. A man aged forty-five years, observed in April, 1875. Some months before placing himself under Dr. Hammond's care paralysis had begun in the right leg, and gradually ascending, involved the right arm. When seen by Dr. Hammond there was general paralysis, very pronounced in the limbs of the right side, in the throat, larynx, and tongue; slightly marked in the limbs of the left side. Reflex movements could not be excited; electro-muscular reaction, was greatly impaired on the right side, and lessened on the left. There was no facial paralysis; no sphincter or bladder trouble; no bed-sores; no derangement of sensibility; no pains, and no muscular spasms. No mental disturbance, except great emotional weakness. A laryingoscopic examination by Dr. Clinton Wagner, showed partial palsy of the vocal cords.

Iodide of potassium and fluid extract of ergot, were administered internally, and faradism was applied to the throat, tongue, and extremities. Improvement soon began, and by the first of June, patient was able to stand and support himself with his arms. In July, paralysis of the left lower extremity suddenly appeared. Recovery

[1] Op. cit., p. 486.

again obtained by use of above means, and of hypodermic injections of strychnia. At time of last note, the muscles of the legs were still much atrophied.

Case XLI.[1] By Courty. A man aged twenty-eight years, admitted to Val-de-Grace hospital, in the service of Prof. Villemin, September 24, 1875. The patient, a saddler by occupation, had not suffered from any noteworthy disease; though he had had tremulous hands since 1871. During February, 1875, and the succeeding months, there were over-work and anxiety. In May, there was a sense of painful fatigue in legs, compared by patient to sensation caused by severe marching. In July, there occurred an attack of fever, lasting three days; during which the calves were the seat of special pain. On August 20, without cause, he had chills, headache, fever, and insomnia, at the same time pain, made worse by any movement, in legs and abdominal muscles. The fever lasted a number of days, during which the patient was able to walk, but with difficulty; there was neither numbness nor twitching in the legs. Early in the morning of August 25, as the fever subsides, patient discovers that his left leg is almost powerless. August 26, fever has ceased. The left lower extremity is completely paralyzed. The left arm, (which in the preceding forty-eight hours, had been the seat of pain) is partially palsied. No disorder of sensibility, no alteration of nutrition. August 27, micturition is difficult, possible only with pain and effort when patient is standing. No history of disease of bladder or urethra. This slow micturition coincided for ten days with constipation. Once a catheter had to be introduced; and

[1] Note sur un cas de myélite aiguë des cornes antérieures. Gaz. Médicale, 1876, Nos. 18 and 23.

it may here be stated, that the partial inaction of the bladder lasted six weeks. From Sept. 1 to 20, the paralysis of the two left extremities remained about the same. After September 25, improvement in power of left arm, and disappearance of cervical and scapular pain.

On admission to Val-de-Grace, September 24, examination of the palsied left lower extremity with the faradic current, shows loss of reaction in the posterior muscles of thigh and leg; very strong currents producing only slight contractions in the anterior femoral and tibial regions. In the left upper extremity, there is slight diminution of contractility to the current. On the right side, all the muscles respond well. The diagnosis of spinal paralysis of the adult is made. To have applications of the faradic current slowly interrupted. October 10, slight improvement; patient can walk with two sticks; urinates freely. November 1, no material change; left lower extremity still powerless; contractility as above. Ordered weak galvanic currents, sulphur baths, hydrotherapy. December 1, in bed, the left lower extremity can be moved and raised. December 5, examination with faradic current shows slight contractility in anterior cervical and tibial muscles, none in posterior muscles of thigh and legs; diminished contractility in muscles about left shoulder and upper arm. Sensibility is perfect in all its modes, the palsied muscles are very painful to pressure. No hyperæsthesia of skin on right side. No articular or neural pains. Atrophy is marked in palsied lower extremity, especially in thigh. Left upper extremity a little wasted. The palsied parts are colder (numerous measurements taken with proper precautions) than the same parts on the right side; the axilla by .2° C., the anterior crural region by .6° C., the

calf by 1.3° C. If both lower extremities are exposed to the cold, the differences increase to 3° and 4° C. The left hand is more tremulous than the right. January 1876, no marked change in motility, muscular hyperæsthesia, muscular irritability, etc. Left foot and leg become very bluish when placed in warm water, and immediately after the bath, the left foot is warmer than the right. In other words, the paralyzed parts become more easily congested than the healthy parts, and remain longer congested. The left pulse gives a tracing indicating diminished vascular elasticity. These experiments demonstrate, according to Dr. Courty, that there is vaso-motor paresis on the palsied side. Nothing is said of reaction of muscles to galvanism. June 1, the hyperæsthesia has almost completely disappeared from the paralyzed parts; the atrophy has increased somewhat, all movements have become easy and strong, and patient has resumed work. The left leg is still colder than the right, and the left foot swells toward night.

Case XLII.[1] By J. Déjérine. A single woman aged twenty-six years, was admitted to the hôpital Saint-Louis, December 16, 1875. She could give no information as to time of syphilitic infection, and stated that her symptoms had appeared only two months previously. She had then noticed a gradually increasing weakness of the lower limbs; and on admission this is so marked that she can hardly walk a few steps without support. She has experienced some sharp pains in the lower extremities; and an eruption has lately appeared. A short time after admission walking became impossible, and patient was confined to her bed.

[1] Atrophie musculaire et paraplégie daus an cas de syphilis maligne précoce. Arch. de Phys. norm. et pathol. 1876, p, 430.

Examination January 15, 1876. — Pallor and cachexia; marked emaciation, alopecia, and syphilitic ecthyma on body and face. Has lately had severe pains in the legs, in calves and toes, more severe at night. The lower limbs are much wasted, walking and standing are impossible. While in bed feeble movements of legs and thighs are possible. Atrophy and paresis are uniformly distributed in lower limbs. Feet are in extreme extension, and motion of toes is lost. Reflex movements exaggerated. Reaction to faradism much impaired. Sensibility impaired; to contact there is slight anæsthesia on dorsum of feet and on leg; temperature is well perceived. Insteps and calves are hyperalgesic. Respiration well performed. February 20. —Since last note iodide of potassium has been given in doses increased to ʒj. Sensibility has remained the same; voluntary power has improved a little in the last few days. There is incontinence of fæces, and cachexia is about the same. No bed-sore.

March 1, a few days ago pains appeared in the arms and fingers; latter have been stiff and clumsy. March 15, lower limbs as before; upper extremities weak, and showing incoordination. March 25, arms are so weak that patient can not feed herself. April 8, lower limbs have gained strength; upper limbs perhaps less weak. Atrophy of legs still extreme. April 18, patient dies with symptoms of pulmonary disease.

Autopsy shows spinal cord and its membranes healthy to the naked eye. The muscles of the lower limbs appear pale and degenerate, and the smaller nerves are grayish. [For details of lesions found on microscopic examination see p. 87].

It seems surprising that M. Déjérine should report the above case under a semeiological denomination.

While discussing its pathology he admits its almost perfect resemblance to certain cases of spinal paralysis of the adult, but refuses to recognize it as such because of the almost chronic appearance of the symptoms, and because the "sphincters were paralyzed." The former objection is not valid because it is well-established that myelitis of the anterior horns may show itself under various degrees of acuity, and the second is not borne out by the notes of the case. In fact nothing is said of the "sphincters," and the only allusion to them is in the note made February 20, when it is stated that there is "paresis of the rectum, æcal incontinence." Nowhere is it stated that the function of the bladder was impaired. M. Déjérine doubts the syphilitic nature of the spinal lesion.

Case XLIII. Personal. Mrs. H—d, aged about thirty-five years, was very ill in the spring of 1870, with cerebro-spinal meningitis. The spinal symptoms predominated, and during convalescence it was discovered that Mrs. H. had extensive paralysis with atrophy of many muscles; she could move neither hands nor feet. There were no symptoms indicating a cerebral lesion; no anæsthesia; no difficulty in micturition. The skin of the hands and feet was hyperæsthetic. For a long time Mrs. H. suffered from stiffness of and a burning sensation in the spine.

In November, 1870, I first saw the patient, and found paralysis and atrophy of the muscles of the lower limbs below the knees, of forearms and hands. The muscles of the thighs, shoulders, and upper arm, were weak, but not much wasted. The muscles spoken of as atrophied, did not respond to the faradic current, and only a few of them gave galvanic reaction. There was cutaneous

hyperæsthesia; no anæsthesia. Systematic treatment by means of galvanism was at once begun, and, besides, the limbs were rubbed, manipulated, and bathed. During the first year, treatment was continued until June, when Mrs. H. could sit in a chair, raise her arms, flex and extend hands, and use muscles of thighs. Below the knees, there was a slight amount of voluntary contraction in the muscles of the calves. No motion in feet or hands. The feet and legs were cold, and their circulation very sluggish.

Treatment was resumed in November, 1871, and continued until June, 1872. In January, the tendo Achillis was cut on both sides, and the legs put in plaster, for the relief of talipes equinus, and to give galvanism a chance of acting on the anterior tibial muscles. On the fourth day after these muscles were relieved from tension, galvanism produced distinct contractions in them, for the first time. After a few weeks a light braced shoe was made, and Mrs. H. began to walk, at first with crutches, then with a cane, and in the last three years without support. The forearms and hands also progressively improved, and writing became possible.

The treatment (galvanism, friction, passive movements) was continued every winter, until 1875. During 1873 and 1874, there was still slight burning and stiffness in the spinal column, especially high up. The general health remained uniformly good. At the time of the cessation of treatment in June, 1875, Mrs. H. walked quite well with light steel braces for the ankles, but without a cane. Above the knees the muscles were normal; below them there was still marked atrophy, and contractions could only be obtained by strong galvanic interruptions. The muscles of the calves, which for three years had reacted to the will, still yielded no

faradic reaction. Sensibility of feet perfect. The muscles of the forearms were normal in size, power and reactions; those of the hand all showed some power, and a degree of reaction to both currents; although wasting was still marked, and "main-en-griffe" was still present, Mrs. H.'s handwriting was quite as good as before her illness. Sensibility of hands perfect.

In October, 1875, Mrs. H. went to Europe, and there under the advice and treatment of Professor Charcot and Dr. Joffroy has, I learn, gained still more.

During the first two years of my observation of this case I looked upon it as one of spinal meningitis with effusion involving the anterior roots of the spinal nerves, but in the last two years I have been led to consider that during the attack of cerebro-spinal fever, there occurred myelitis of the anterior horns. In this sense the case might be classed with some of those of paralysis with wasting following acute disease (Westphal, S. G. Webber and others).

Case XLIV. Brought to my clinic, September 23, 1876, by Dr. ——,[1] of Williamsburgh. A man aged twenty years, of good constitution, and fully developed frame. During the past spring and summer patient suffered from pain and soreness, and hyperæsthesia of the limbs. This pain was neither neuralgic nor rheumatic. About two months ago legs became weak, and have since gradually become paralyzed to a marked degree. Within a short time after paresis of the legs, the arms and hands became weak. Œnema of the hands and feet appeared. The bladder and rectum have not

[1] The gentleman who brought me this very interesting case will, I hope pardon me for not giving his name. I did not know it, and he has made no report since.

been paralyzed; and the only muscles of the trunk which seem to have suffered are the abdominal, as evidenced by weak voice, and imperfect cough. There is neither history nor evidence of syphilitic infection.

Examination yields the following results. There is no paralysis about the head; pupils are right; tongue points straight. Inspiration is well done; expiration is weak. Upper extremities are moderately paralyzed; some voluntary movements can be done by all muscular groups except extensors of hand. Left arm more palsied than the right. The lower limbs are more affected; lying on his back, patient cannot raise heels from the bed with extended legs. Left side weaker than the right. No voluntary movements of toes or foot on the left side, slight motion on the right side. Thigh muscles weak. No paralysis of bladder or sphincter ani. Denies having or having had any numbness. Muscular masses are everywhere tender, even in hands and feet. Sensibility to contact dull, but not abolished; pulling hairs or pricking well felt on legs and hands. Has some spinal pain, chiefly in lower part. There is slight œdema of hands and feet. No trace of bed-sore, faradism (strong currents) gives no trace of reaction in extensor group of forearms; flexors respond feebly but distinctly. There is no response in any of the muscles of the legs.

Test with galvanism. Extensor muscles of right forearm, right leg, anterior tibial group and peronei respond slightly to 30 cells of Stöhrer's battery.

Case XLV. Personal. My friend, Dr. W. H. Geddings, of Aiken, South Carolina, consulted me by letter June 30, 1876. As his observation is concise and exact, I prefer to give his letter verbatim. His age is

thirty-eight years. . . . . "From January to the middle of April I was engaged in extensive practice. Several times in the course of the winter I was disturbed at night by pain in the back, which I attributed at the time to a defective mattress, and indeed after changing the bed the pain recurred but once. About the middle of April I began to experience a feeling of lassitude which after a few days was followed by a soreness along the back of the thigh; the sensation being very much like that which one would have after a long ride. I continued, however, to do hard work until, finally after the loss of rest for three consecutive nights, I was taken with a chill on the evening of April 26. Fancying that it was a bilious attack, quite common in this section, and to which I myself am subject, I took a dose of calomel and opium, with a saline draught in the morning, without experiencing relief.

"On April 27 and 28, nothing worthy of note except increasing weakness and pain in the back, recurring every afternoon about 4 P. M., accompanied with nervousness. During this period and for the succeeding eight days there was a rise of temperature to 38° 8 C., without any corresponding increase in the frequency of the pulse. On the 29th there was paralysis of the detrusor (retention of urine) which lasted until the 6th of May. About the same time I remarked increased difficulty in locomotion, and discovered a few days later that it was due to paralysis of the flexors of the left lower extremity. The feeling of restlessness and the neuralgic pains continued to recur every afternoon, and became so intolerable that I was compelled to resort to hypodermic injections of morphine. These always afforded prompt relief . . . Among other symptoms at this period of the disease I noticed frequently a feel-

ing as though an electric current was suddenly passed through the body, at times so severe as to wake me from sleep, and cause me to start. Later in the course of the disease the neuralgic pains extended to the upper extremities, accompanied by a feeling of numbness. I neglected to state above, that the neuralgic pains had extended to the right leg without, however, causing any impairment of motion other than that produced by general debility. About the 28th of May there was some slight improvement, but this was followed in a few days by a recrudescence during which the paralysis of the flexors of the left leg became more marked. Since then there has been a slow improvement, and to-day (June 30), the state of affairs is as follows:

"Emaciation marked everywhere, except in the face; pain in the back reduced to a slight aching in the sacrum; very little pain, and that only at times in the left leg, no pain in right leg; slight numbness in the anterior half of the right foot; no pain in the upper extremities. Electro-motor contractility, present everywhere *except in the flexors of the left leg.* I can walk, but with great difficulty, across the room . . . Iodide of potassium was tried, but disagreed with the stomach after a few doses. . . Solid extract of ergot was tried for a while, but without any appreciable effect. . . Morphine has been reduced from one grain in twenty-four hours, to a quarter grain. . . The only other treatment I have had, has been the application of small blisters along the lower part of the spine. I am now applying the induced current to the left limb, with some advantage to the flexors of the thigh, but without any appreciable effect on those of the feet and toes. Pulse 72; temperature normal. No anæsthesia as tested with a pair of ordinary compasses."

Two physicians who examined Dr. G. pronounced the case to be one of "myelitis confined to the lowermost portion of the cord."

In my reply I expressed the opinion, subject to certain negative answers from the doctor, that he had subacute myelitis of the anterior horns, and that he would recover. I advised the use of Squibb's fluid extract of ergot, more counter-irritation to the spine, and the galvanic current to the paralyzed muscles.

The following categorical answers to my questions, made the diagnosis quite certain, by excluding common central localized myelitis.

"1. There is no very marked numbness, but a feeling of prickling and stiffness, sometimes confined to the feet, at other times extending up the back of the legs. There is also a similar sensation in both hands and forearms.

2. No anæsthesia to pricking, heat, or slightest touch.

3. There is paralysis of the flexors of the toes, and weakness of the extensors of the left leg.

4. Atrophy of muscles, but not very extensive. I have no means of measuring the strength of grasp, but find that the right hand is weaker than the left.

5. Muscles of the thigh (left) respond to both currents. The flexors of left leg to the galvanic current only; the short flexors only at times, and then with not less than twenty cells. There is marked want of sensibility to the galvanic current, in parts of the left foot.

6. No feeling of constriction.

7. No projection of vertebræ.

8. There is occasional jerking of the different extremities, as if electricity were passed through them.

9. Nothing abnormal about the rectum; urine passes

freely (no retention since first week of the disease). Erections occur at intervals.

10. No fibrillary contractions in the weak muscles.

11. I have never had specific disease.

The treatment was carried out, and the doctor came north. The sea-voyage did him a great deal of good, and he returned home in October nearly well. Owing to my absence from town, I failed to see him. A few days ago (Nov. 6), I had the pleasure of receiving a letter from him, in which he states that he is entirely restored, and has resumed practice.

NOTE.—I would add that I have met with one more case of myelitis of the anterior horns in an adult, but not having full notes of the case I do not include it in the category of cases. The patient, a man about forty-five years of age, I saw in consultation with Dr. Edward F. Schwedler of this city, last July. For many months this man suffered from atrocious neuralgia of the left lower extremity, of a sciatic type, and gradually many muscles of that limb had wasted away. There had been some neuralgia in the right thigh previous to the consultation, and I have since learned from Dr. Schwedler that during the summer atrophy also showed itself in that leg.

In my manuscript I had included an interesting case of "acute spinal paralysis" by Eisenlohr (Arch. f. Psych. u. Nervenkrankheiten, Bd. V, p. 219); but after careful consideration I concluded to strike it out. The absence of muscular atrophy and the preservation of electro-muscular contractility are sufficient reasons for my doubting (with Prof. Erb) that this case was one of poliomyelitis anterior.

CHAPTER III.

THE SYMPTOMS STUDIED ANALYTICALLY.

I shall now proceed to make a detailed study of the symptoms exhibited by the forty-five cases[1] related in the preceding pages, and shall do it in accordance with the following scheme:

1. Disorders of Movements { Paralysis. Spasms. Reflex capacity.
2. Disorders of Sensibility { Anæsthesia. Morbid Sensations. Hyperæsthesia.
3. Disorders of Nutrition { Muscular Atrophy. Local Temperature. Œdema. Arthritis. Cystitis. Eschars.
4. General Symptoms { General Temperature. Pulse.
5. Electro-muscular Contractility.
6. Special Symptoms { Vomiting, Delirium.
7. The Course of the Disease.

[1] In this analysis I do not refer to the symptoms as exhibited in the infantile form; they are elaborately studied by Heine and by Laborde: see p. 6.

I. Disorders of Movements.

a. Paralysis. This to a greater or lesser degree was present in all the cases: 100 per cent.

The paralysis was distributed as follows: There was general paralysis, or paralysis of all the extremities, in twenty-nine instances, or 64.5 per cent.: Cases i, ii, iii, iv, v, vi, vii, xi, xii, xiii, xiv, xv, xviii, xix, xxi, xxii, xxiii, xxv, xxvi, xxxii, xxviii, xxxiv, xxxvii, xxxviii, xxxix, xl, xlii, xliii, and xliv.

There was paraplegia, or paralysis of both lower extremities, in eleven instances, or 24.5 per cent.; cases v (for one year), viii, ix, x, xvi, xvii, xxiv, xxvii, xxviii, xxxi, and xxxvi.

There was hemiplegia, or paralysis of the extremities on one side of the body, in two instances, or 4.5 per cent.; cases xl (for a time) and xli. In cases xviii and xxxiii, one side of the body was more paralyzed than the other.

There was monoplegia, or paralysis of one extremity, in four instances, or 8.9 per cent.; cases xxix (right leg), xxx (right arm), xxxii (right leg for awhile alone), and xlv (left leg).

In some cases of general paralysis there was, besides, palsy of other muscles.

The muscles of Deglutition in cases xx, xxxii, xxxv, and xl, or 8.8 per cent.

The muscles of the Tongue in cases xx, xxxix, and xl; or 6.6 per cent.

The muscles of the Back in cases xxiii, xxiv, and xxxii; or 6.6 per cent.

The muscles of Respiration in cases xxxii (causing death by asphyxia), and xxxix; or 4.5 per cent.

The muscles of the Face in cases xx, and xxxix; or 4.5 per cent.

The muscles of the Vocal Cords in cases xix (?), and xl; or 4.5 per cent.

The muscles of the Eyeballs in case xx; or 2.2 per cent.

The paralysis was quickly generalized (in twenty-four hours or less) in eighteen instances, or 40 per cent; cases i, ii, iii, iv, x, xii, xiv, xv, xxii, xxiii, xxv, xxvii, xxviii, xxix, xxx, xxxiv, xxxvii, and xxxix.

The paralysis was gradually developed (forty-eight hours or more) in eighteen instances, or 40 per cent; cases v, vi, vii, xvi, xvii, xviii, xix, xxi, xxv, xxxi, xxxii, xxxiii, xxxiv, xxxvi, xxxviii, xli, xlii, and xliv.

The paralysis was ascending, involving first the legs then the arms, in twelve instances, or 26.6 per cent; cases v, xvi, xviii, xix, xxi, xxxii, xxxv, xxxviii, xxxix, xl, xlii, and xliv.

The paralysis was descending, affecting first the arms, and then the legs, in five instances, or 11.1 per cent; cases vi, vii, xii, xxvi, and xxxvii.

The mode of appearance of paralysis, is not mentioned in cases viii, ix, xx, xxiv, and xliii.

An incurable residue of paralysis remained, affecting various parts of the body, in the following cases:

In a number of muscles of the upper and lower extremities, in eight instances, or 17.7 per cent.; cases i, ii, iii, vi, xiv, xx, xxxvii, and xliii.

In the muscles of both legs in eight instances, or 17.7 per cent.; cases viii, ix, xvi, xxiv, xxvii, xxviii, xxxi, and xl.

In the muscles of both thighs, in case xi.

In some muscles of the forearms and hands, in cases vii, and xiii.

In muscles of the right arm and left leg, in case xii.

In muscles of the right upper extremity, in cases iv, and xxx.

In muscles of one leg, in cases xxii, and xxix.

The Bladder and Rectum performed their functions in an absolutely perfect manner, in thirty instances, or 68.8 per cent.; cases i, ii, iii, iv, v, vi, vii, x, xi, xii, xiii, xiv, xv, xvi, xviii, xix, xx, xxi, xxiii, xxiv, xxv, xxvi, xxix, xxx, xxxi, xxxiv, xxxix, xl, xliii, and xliv.

There was partial or temporary paralysis of these organs in ten instances, or 22.2 per cent.; cases xvii, xxvii, xxxiii, xxxv, xxxvi, xxxvii, xxxviii, xli, xlii, and lv.

In cases xxxiii, and xli, micturition was difficult, and the anal sphincter was weak, without being actually paralyzed.

In cases xxvii, and xxxv, the catheter was used for two or three days.

In case xlv, there was retention of urine for eight days, at the beginning of the attack.

In case xxxviii, there was incontinence of fæces for two weeks, early in the attack. In this case also, the catheter was used on the eighth day. The retention ceased, but at what time is not stated.

In case xxxvi, it is stated that several months after the beginning of paralysis, there was involuntary flow of urine.

In case xvii, there was occasional retention during several months in the third year of the disease.

Nothing is said of the state of the bladder and rectum in five instances: cases viii, ix, xxii, xxviii, and xxxii. The organs were probably not paralyzed.

From the foregoing, we may draw the conclusion that in myelitis of the anterior horns in the adult, there is no tendency to the occurrence of complete and last-

ing paralysis of the bladder (and rectum) which forms so prominent a symptom of common central myelitis, diffused or localized.

b. Spasm.

This form of disorder of movements, may best be studied under the following heads:

Fibrillary contractions, were noted in only four instances, or 8.8 per cent: cases xiv, xxi, xxv, and xxxii.

Tremor occurred in the hands and arms, in cases xii, and xli, or 4.4 per cent.

Local cramps were experienced in five instances, or, 11.1 per cent.; cases xiii, xvii, xxv, xxxi, and xxxiii.

Spinal epilepsy was developed late in case xx, (probably after extension of myelitis to deeper parts of cord).

Incoördination in the movements of the arms, was noted once; in case xliii.

c. Reflex capacity. This was examined in numerous cases.

It was exaggerated in case xlii.

It was present in three instances, or 6.6 per cent; cases xxiv, xxvi, and xxxiii.

It was difficult to excite in three instances, or 6.6 per cent; cases xv, xxi, and xlv.

It was lost in five cases, or 11.1 per cent.; cases xvii, xviii, xxxv, xxxviii, and xl.

In the following ten instances, or 22.2 per cent, it is stated that there was "no spasm:" cases x, xiv, xv, xvi, xviii, xix, xxi, xxxvi, xxxix, and xl.

II. Disorders of Sensibility.

There was no anæsthesia in twenty-nine instances, or 64.4 per cent.; cases i, iv, vi, viii, ix, xii, xiv, xv, xvi, xvii, xxii, xxiv, xxv xxvii, xxviii, xxix, xxx, xxxii, xxxiii, xxxiv, xxxv, xxxvi, xxxvii, xxxix, xl, xli, xliii, xliv and xlv.

a. Anæsthesia. This was noted in twelve instances, or 26.6 per cent.; cases iii, v, vii, x, xviii, xix, (?) xx (?) xxi, xxvi, xxxi, xxxviii, and xliii. It is worth while to note the particulars.

In cases xix, and xx, the existence of anæsthesia was doubtful; if present, it must have been slight and temporary.

In case xxxviii, tactile sensibility was much impaired, but only temporarily.

In case xlii, there was slight tactile anæsthesia or dorsum of feet.

In case iii, sensibility was a little dull in extremities.

In case xxi, there was marked anæsthesia to finer tests, but only for a while.

In case xxxi, it is stated that impressions of pain and temperature were not well perceived, and sensibility was a little dull in hands and feet.

In case v, there was slight anæsthesia of the lower limbs.

In case viii, there was slight temporary anæsthesia.

In case x, there were patches of anæsthetic skin in legs, sides, and axilla.

In case xviii, there was at one time great anæsthesia; when seen, this was slightly marked in extremities, and disappeared.

In case xxvi, sensibility was normal, except that electricity was not well felt in extremities.

It consequently appears that in no case, was there severe and lasting anæsthesia, co-extensive with the paralysis; and that in the great majority of cases, the loss of feeling was slight and temporary.

Painful impressions made on paralyzed parts, were retarded by several seconds in their transmission to the sensorium, in cases xviii (5 to 8 seconds), xix, and xxi.

b. Morbid sensations or paræsthesiæ.

Numbness or formication occurred (nearly always at the commencement of the disease) in twenty-one instances, or 46.6 per cent.; cases i, ii, iv, vii, xiii, xiv, xvii, xviii, xix, xx, xxi, xxvi, xxviii, xxxi, xxxiii, xxxiv, xxxv, xxxvi, xxxviii, and xlv.

There was no numbness in eight instances, or 17.7 per cent.; cases xxiv, xxvii, xxix, xxx, xxxvii, xxxix, xl, and xli.

The sensation of a constricting band was experienced in cases xix, xx, and xxi.

The sensation of subjective cold was felt in cases xix and xxxvi (?).

The sensation of an electric current passing through the body was felt in case xlv.

c. Pain is reported as present, without specification of locality, in seven instances, or 15.5 per cent.; cases xvii, xviii, xxii, xxiii, xxv, xxxv, and xxxviii.

The pain was clearly muscular in five instances, or 11.1 per cent.; cases i, xv, xxxvii, xxxviii, and xl.

It was sharp and neuralgic (in the limbs) in six instances, or 13.3 per cent.; cases xiii, xxvii, xxix, xxxi, xlii, and xlv.

Spinal pain was noted in thirteen instances, or 28.8 per cent.; cases i, ii, iv, xiv, xvi, xix, xxiii, xxvii, xxx, xxxviii, xliii, xliv, and xlv.

Pain was experienced in the joints in cases xxx and xxxiii.

There was a paroxysm of burning and tearing pain in the heels in case xxxv.

There was a throbbing pain in the soles of the feet in case xviii.

There was no pain in four instances, or 8.8 per cent.; cases xxi, xxxii, xxxix, and xl.

d. Hyperæsthesia. There was cutaneous hyperæsthesia in three instances, or 6.6 per cent.; cases xxxvi, xlii, and xliii.

Muscular hyperæsthesia (tenderness on pressure), was present in five instances, or 11.1 per cent.; cases xxiii, xxvii, xl, xlii, and xliv.

III. Disorders of Nutrition.

a. Muscular Atrophy. This was great in twenty-five instances, or 55.5 per cent.; cases i, ii, iii, iv, v, vi, vii, viii, ix, xiii, xiv, xv, xvi, xvii, xxii, xxiii, xxiv, xxvii, xxviii, xxx, xxxi, xxxvi, xl, xlii, and xliii.

It was moderate in fifteen instances, or 33.3 per cent.; cases xi, xii, xviii, xx, xxi, xxvi, xxix, xxxii, xxxiv, xxxv, xxxvii, xxxviii, xxxix, xli, and xlv.

It is not mentioned in case xxxiii, and it does not appear to have been present in cases xix, and xliv.

The wasting was temporary in ten instances, or 22.2 per cent.; cases xv, xviii, xxi, xxvi, xxxiv, xxxv, xxxviii, xxxix, xli, and xlv.

It was permanent (causing deformity) in thirty-seven instances, or 60 per cent.; cases i, ii, iii, iv, v, vi, vii, viii, ix, x, xi, xii, xiii, xiv, xvi, xvii, xxii, xxiv, xxvii, xxviii xxix, xxx, xxxi, xxxvi, xxxvii, xl, and xliii.

b. Bed-sores or ulcerations of the skin did not occur in any case, although several patients (as cases xxiii and xxxvi) lay a long time flat upon their backs.

c. Cystitis was observed in only one of the forty-five cases, viz.: case xvii.

d. Arthritis (arthropathy) affecting the small points of the hands and feet was noticed in two instances or 4.4 per cent.; cases xxx (?) and xxxiii.

e. Œdema. In four instances, or 8.8 per cent., there occurred temporarily, dropsical swelling of various parts. In case xviii the face and feet were swollen; in case xix

the legs and hands; in case xli the left foot (late in the disease), and in case xliv the hands and feet.

f. The temperature and circulation in the paralyzed parts were often modified; the parts being cold and bluish.

In eleven instances, or 24.4 per cent. the palsied parts were cold; cases x, xi, xvii, xxi, xxiii, xxiv, xxvi, xxxvii, xxxviii, xlii, and xliii.

In two instances the parts were reported to have been cool; cases xvii and xx.

In five instances, or 11.1 per cent., the circulation in the palsied parts was stated to be feeble and sluggish; cases xvii, xxiii, xxiv, xxvii, xli, and xliii.

In twenty-seven instances, or 60.0 per cent., no mention is made of these symptoms; cases i, ii, iii, iv, v, vi, vii, viii, ix, xii, xiii, xiv, xv, xvi, xix, xxii, xxv, xxxii, xxxiii, xxxiv, xxxv, xxxvii, xxxix, xl, xlii, xliv, and xlv.

IV. General Symptoms.

a. General bodily Temperature.

Fever was present during a part of the illness in eighteen instances, or 40 per cent.; cases i, ii, iii, iv, xii (?), xviii, xxi (?), xxiii, xxv, xxvii, xxviii, xxix, xxx, xxxv, xxxvii, xxxix, xli, xlv.

Fever is said to have been absent in the following instances, or 6.6 per cent.; cases xv, xxiv, and xxvi.

Fever is not mentioned in eighteen observations or 40 per cent.; cases v, vi, x, xiii, xiv, xvi, xvii, xix, xx, xxii, xxxi, xxxii, xxxiii, xxxiv, xxxvi, xlii, and xliv.

In six instances, or 13.3 per cent., an acute and quite surely febrile disease preceded the development of palsy: in cases viii and ix measles, in case xi dysen-

tery, in cases xviii and xix a "severe cold," and in case xliii, cerebro-spinal meningitis.

The following particulars are given about the fever in various cases.

In cases xviii and xxxix, there was only slight fever; in case xi, the dysentery preceding the paralysis was probably accompanied by fever; in case xxi, the slight rise of temperature observed may have been caused by pulmonary disease; in case xxvii, fever and paralysis appeared the same day, in cases i and xxviii, the fever ceased on the fourth day, in cases xxxvii and xli, it lasted six days, in case iv, it lasted eight days, in cases xxiii and xxxv, it was present several days, and in the latter showed evening exacerbations, in case xlv, it lasted ten or twelve days, in case xxviii, it lasted five weeks (?); and in case xxx, there were two attacks of fever, with the first no palsy, while nearly two weeks after the beginning of the second, paralysis appeared.

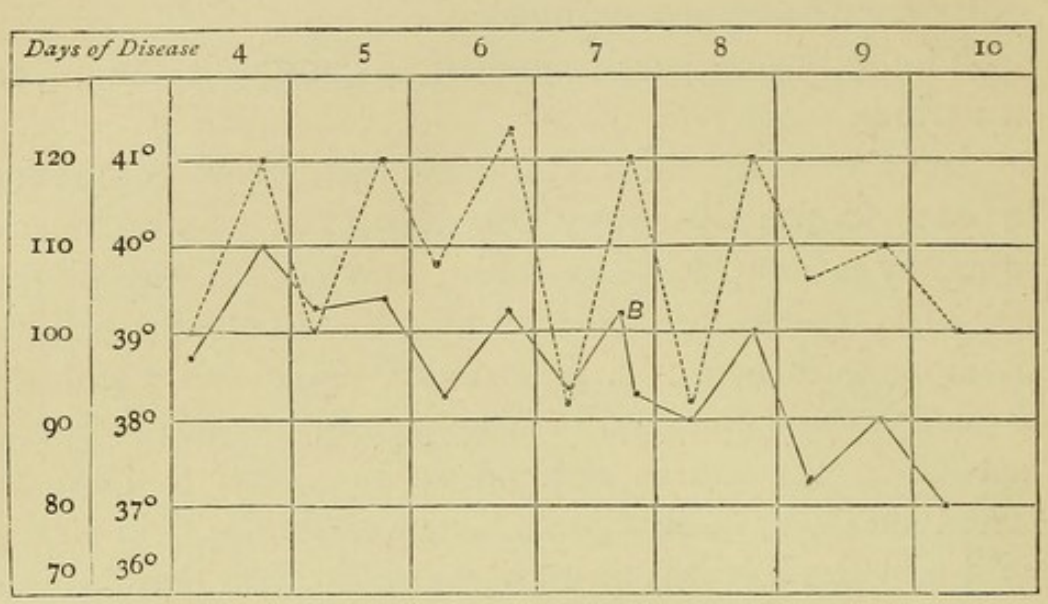
The thermometer[1] was used in cases xxv and xlv. It was not employed in other cases mainly because they came under the physician's observation only after the febrile stage had passed away.

In case xlv the measurements were not taken regularly enough to allow of tabulation, but indicated only a moderate fever ranging up to 38.8° c.

In case xxv, the fever was accurately studied, and from the figures given I have been able to make up the following table of temperature and pulse curves:

[1] It is much to be hoped that, hereafter, physicians will accurately observe the pulse and temperature of patients whom they may suspect of being about to have myelitis of the anterior horns, be they adults or children. Such records may do much to clear up the pathology of the disease.

Acute myelitis anterior—Kussmaul, case XXV.



Pulse line dotted; temperature line dark: B. bath.

This table portrays a mild fever of the continued type, terminating by a lysis showing morning remissions. The highest temperature recorded in this (or any other case) was 40° c.

It seems probable, from a consideration of the histories of acute febrile cases of myelitis of the anterior horns occurring in children as well as in adults, that the initial fever is generally of this type. In many infantile cases the fever lasts only one night, or a day and a night, and is rarely witnessed by the physician.

b. The Pulse. In the cases related above there is much contradiction as to the pulse-rate, even during the febrile stage. In case xxv, the pulse ranged very high, its excursions being disproportionate to the temperature curve. It should, however, be borne in mind that the patient was a woman. In other cases it is stated that there was no increase of pulse-rate to correspond with the rise of temperature. In very many cases nothing is said of the symptom.

V. Electro-muscular Contractility.

In many of the forty-five cases constituting the basis of this essay the contractility of the paralyzed muscles was tested with the faradic and galvanic currents, in some cases with a great degree of accuracy. From these examinations important conclusions, both for diagnosis and prognosis, can be drawn.

a. Reaction of the palsied muscles to faradism. This was lost in some of the paralyzed muscles and diminished in others in twenty-seven instances, or 60 per cent.: cases i, v, vi, vii, x, xi, xiv, xv, xvi, xvii, xxii, xxiii, xxvi. xxvii, xxviii, xxix, xxx, xxxi, xxxii, xxxiv, xxxv, xxxvi, xxxvii, xli, xliii, xliv, and xlv.

It was only diminished in seven instances, or 15.5 per cent.; cases xviii, xix, xx, xxi, xxiv, xxv, and xlii.

It is stated that electro-muscular contractility (kind of current used not specified) was much diminished in five instances, or 11.1 per cent.; cases viii, ix, xxxviii, xxxix, and xl.

No mention is made of this test in six instances, or 13.3 per cent.; cases ii, iii, iv, xii, xiii, and xxxiii.

b. Reaction of the palsied muscles to galvanism. This was lost in three instances, or 6.6 per cent.; cases xvii, xxiii, and xxvi.

It was diminished in eleven instances, or 24.2 per cent.; cases xxv, xxvii, xxviii, xxix, xxx, xxxi, xxxiv, xxxvi, xliii, xliv, and xlv.

It was well-preserved in cases xviii, and xix.

This preservation a degree of contractility under galvanism which I clearly made out in several of my own cases (though I did not state the reactions in a sufficiently precise manner) constitutes an approach to the reaction of degeneration (*entartungsreaction*) which

is observed after section of motor nerves. Prof. Erb,[1] in an interesting paper has announced the existence of this reaction in cases of myelitis anterior in the adult, and refers to Salomon,[2] as having already discovered the same in cases of myelitis anterior in children (infantile spinal paralysis).

c. A peculiarity worth noting is that in two instances, during convalescence, muscles responded to the impulse of the will but not to the electric current; in case xli, there being no response to faradism, and in case xxvi, none to both currents.

VI. Special Symptoms.

a. Delirium was present, with fever, in cases iii and xxxiii.

b. Stupor, so marked as to give the patient the aspect of one suffering from typhoid fever, in case xxv.

c. Vomiting, in cases xviii, xxxii, and xxxvi.

The Course of the Disease was:

Acute febrile in fifteen instances, or 33.3 per cent.; cases i, ii, iii, iv, xi, xii, xxi, xxiii, xxv, xxvii, xxviii, xxix, xxxvii, xli, and xlv.

Acute non-febrile in ten instances, or 22.2 per cent.; cases x, xiii, xiv, xv, xvii, xxii, xxiv, xxxiii, xxxiv, and xxxix.

Sub-acute febrile in five instances, or 11.1 per cent.; cases viii, ix, xxx, xxxv, and xliii.

Sub-acute non-febrile in four instances, or 8.8 per cent.; cases vii, xviii, xix.

Chronic in ten instances, or 22.2 per cent.; cases v, vi, xvi, xxvi, xxxi, xxxii, xxxvi, xl, xlii, and xliv.

Unknown in case xx.

[1] Ueber acute Spinallähmung (Poliomyelitis anterior acuta) bei Erwachsener. Arch. f. Psych. u. Nervenk. V. p. 758.

[2] Zur Diagnose und Therapie einige Lähmungsformen im kindlichen Alter. Jahrb. f. Kinderheilk. I. p. 370. 1868.

CHAPTER IV.

THE SYMPTOMS STUDIED SYNTHETICALLY.

If the various symptoms examined in detail in the last chapter are considered in connection with the various cases related in Chapter II, the following groups of symptoms or types of anterior myelitis may be admitted as true to nature, and as sufficient to include all cases, in adults and children.

I. Acute myelitis of the anterior horns, or poliomyelitis anterior acuta.

a. Febrile form. With or without premonitory symptoms, rarely with a chill, the patient develops a fever of a moderate continued type, which runs its course in from two to ten days. At the same time there are symptoms of irritation of the nervous system, such as pain in the head, back, and limbs, numbness in the extremities. There may be delirium or even stupor, and the physician may well be embarrassed in his diagnosis, and be disposed to look upon the attack as febricula, or typhoid fever, or meningitis. During the fever or at its close, paralysis of one or more limbs is discovered; one arm may be paralyzed, or all the extremities may be weak. The encephalic nerves are unaffected, the muscles of respiration are not paralyzed, and the bladder and rectum perform their functions well. The bladder may be paretic for a day or two. The paralyzed parts are relaxed and free from spasm. Electrical examination will,

even in the first few days, show diminished contractility in the paralyzed muscles. A degree of tactile anæsthesia may exist; but never absolute anæsthesia coextensive with the paralysis. In the course of a few days or of a week or two there occurs a spontaneous recession of paralysis, which persists in one or two limbs for weeks or months, or else wholly disappears. In this period atrophy of the paralyzed muscles becomes marked, and may go on to apparent disappearance of the muscular tissue. The temperature of the permanently paralyzed parts falls, and the circulation in them is sluggish. Electrical examination now shows absolute loss of reaction to both currents applied to the nerves supplying the palsied muscles, and only galvanic reaction (often altered in quality) in the muscles themselves. Almost always no anæsthesia remains at this stage.

In children nearly all the cases of myelitis of the anterior horns assume this type, though running their course in a much shorter time. One night or twenty-four hours of sharp fever with, possibly, delirium and convulsions, is followed by paralysis and muscular atrophy as above. The retrocession of paralysis is more marked. In children there is frequently general hyperæsthesia, and of course no complaints of numbness or special spinal pain are made. The bladder is never paralyzed. The electrical reactions are similar, and so are the appearances of those limbs which are to remain paralyzed and atrophied.

b. Non-febrile form. Pain in the back and limbs, numbness or formication in the extremities, temporary retention of urine, and rapidly developed paralysis of the limbs, and even of the throat and respiratory muscles, usher in the disease. In less than a week nearly

all the voluntary muscles may be paralyzed. There may be partial anæsthesia. Retrocession of paralysis from some of the parts often occurs, and muscular atrophy sets in as in the febrile form. The electrical reactions are similar. The paralysis may appear first in the feet and gradually ascend, in such a way as to merit the name of ascending paralysis; but, on the other hand, it is often descending. Or, the paralysis may from the start affect only one limb.

In young children this form is also frequent. A child of two or four years of age is put to bed well, and without the occurrence of any febrile illness in the night, flaccid paralysis of one or more limbs is discovered in the morning. Muscular atrophy and refrigeration ensue, and the electrical reactions are as above.

II. Subacute myelitis of the anterior horns, or poliomyelitis anterior subacuta.

a. Febrile form. The fever runs a longer period than one week, in some cases several weeks, and with or without numbness, or neuralgic pains, paralysis is developed. There may in these cases be a slight and temporary paresis of the bladder, and the sensibility of the paralyzed parts may be dull.

b. Non-febrile form. After a period of lameness or simple debility lasting a few weeks, accompanied in some cases by numbness, paralysis appears. In both forms the palsy may involve considerable parts of the body, and even threaten life by asphyxia, likewise.

I do not know that this grade of myelitis of the anterior horns has been observed in children.

In both forms of subacute myelitis of the anterior horns the paralyzed muscles show reduced or abolished reaction to the faradic and galvanic currents, and it is probable that future research will show the occurrence

of the reaction of degeneration in these cases as well as in acute ones.

III. Chronic myelitis of the anterior horns, or poliomyelitis anterior chronica.

In adults this rare grade of poliomyelitis is characterized by a long early stage of debility, or paresis, by marked numbness or pain in the parts which gradually become paralyzed. In one case (not recorded in Chapter I) which I had the opportunity of seeing with Dr. Schwedler of this city, severe sciatica with coldness and numbness of the foot and leg preceded the appearance of palsy and atrophy by several months. In such chronic cases atrophy and paralysis proceed hand in hand, and an appearance deceptively like that of progressive muscular atrophy is the result. The diseases are to be distinguished by attention to the distribution of the paralysis and atrophy, and by a careful study of the electrical reactions.

In the child this chronic development of myelitis of the anterior horns is very rare. I have met with but one case, that of a boy eleven years of age who during a period of five or six years had developed an almost general atrophic paralysis. The paralyzed muscles showed greatly reduced, and even abolished electrical excitability. I am indebted to my friend Dr. N. M. Schaffer, surgeon to the New York Orthopedic Hospital, for the privilege of observing this patient.

CHAPTER V.

PATHOLOGICAL ANATOMY.

In discussing the question of the pathological anatomy of myelitis of the anterior, I shall proceed in the same manner as in the study of its symptoms, by a rigid process of induction from ascertained facts.

In the first place I shall lay before the reader the detailed results of post-mortem examinations made in some of the cases related in Chapter II.

In the second place I shall present a brief synopsis of the numerous autopsies made in cases of myelitis of the anterior horns occurring in children.

Thirdly I shall institute an inquiry into the results of post-mortem examinations in a variety of diseases of the brain and spinal cord in which atrophy of muscles was present as a symptom; whether as a capital symptom or as one of secondary importance.

It is to be hoped that by proceeding in this manner a logical conclusion may be reached as to the relation existing between morbid states of the spinal cord and atrophic paralysis or muscular atrophy.

I. Autopsies in cases of myelitis of the anterior horns occurring in adults.

A post-mortem examination was obtained in five of the forty-five cases recorded in this essay.

Case I. (case v. p. 9). The autopsy was made in 1848, by Empis, who reported that the nervous centres were apparently healthy. Lebert made a microscopical

examination of the paralyzed and atrophied muscles, and found some of them in a state of fatty degeneration. This examination was made at a time when the pathological histology of the nervous system was yet unborn, and its results are consequently worthless.

CASE II. (case xxii, p. 31), by Lucas Championière. The paralyzed muscles were found pale and fatty; their fibres being small, and many of them filled with granulations. Some of the anterior nerve-roots were small and grayish, and a nerve supplying one of the paralyzed muscles had undergone degeneration and atrophy. The spinal cord was not examined *secundum artem*, but to the naked eye sections through it just above its lumbar enlargement and in parts lower down showed the left anterior horn of gray matter to be dark brown in color. In the middle of the enlargement both anterior horns were brownish and diffluent. These appearances are certainly very suggestive of degeneration of the anterior horns in that part of the spinal cord connected with the paralyzed muscles.

CASE III. (case xiv, p. 12.) The spinal cord was examined after hardening in chromic acid; sections cut from the cord being prepared by Clarke's method. Throughout the whole length of the organ the white columns and the posterior gray horns were found normal; in other words, the lesion found was restricted to the anterior horns. It is best to quote Gombault's description, *verbatim:*

"As regards the alteration of the ganglion cells, it exhibited the same characters as are met with in the progressive atrophy of these cells. Although the degeneration of the cells is everywhere marked, it is, nevertheless, possible to follow it in its various stages

of development in one section. Close to cells which seem quite normal, others are seen containing a small amount of yellow pigment. In other cells this is so abundant as to surround the nucleus and nucleolus, though these structures are still visible. At this stage the cells tend to assume a globular shape. In a still more advanced degree of degeneration, the processes of the cells are shriveled, or are even absent; the nucleus disappears, and the only thing left of the cell is a small, rounded body filled with yellow granules, and surrounded by a thickish envelope which is stained by the carmine. In some cells which still retain processes, these latter structures may be traced as continuous with the stained envelope. The alteration is diffused, it has affected cells here and there, and a number of these bodies must have disappeared, since in some sections from the cervical region it is not possible to count more than fifteen or twenty cells. The external posterior group (of the anterior horns), in the cervical and lumbar enlargements seem to have been attacked by preference. Throughout the whole anterior gray matter there are altered cells. The lowest part of the cervical region seems to have suffered the most. The cells which do not exhibit yellow pigmentation appear to bear some trace of the lesion which must have affected them at some former period. They have undergone, for the most part, a reduction in size; and there are very few measuring 0.066 mm—a size below the average for ganglion cells of this region."

The medulla oblongata was found to be healthy, except that a few cells of the hypoglossal nucleus were granular. Some anterior roots were in part atrophied. Sections of the nerve-trunks of the arm showed some

small patches of sclerosis—the majority of the fibres aud bundles of nerves being healthy.[1]

In brief the lesion found in the apparently normal spinal cord of a patient having had typical acute anterior myelitis, was granular degeneration of the ganglion cells of the anterior horns.

Case IV. (case xxxii, p. 41.) The autopsy was made on November 24, 1874, nearly twenty-four hours post-mortem.

To the naked eye the sciatic nerves, the brain, and all of the spinal cord except its lowest, 8 cent. appeared normal. This lowest part of the cord was softened; and the white and gray substances could not be distinguished.

[1] In a recent article (Beobachtungen und Untersuchungen über die Krankheiten des centralen Nervensystems, Arch. f. Psych. u. Nervenkrankheiten. Ed. vi, 3, p. 765, 1876, [at p. 810 et seq.]) Prof. C. Westphal, of Berlin, has attempted to show that Gombault's case is worthless as an illustration of the lesions of myelitis anterior because, (*a*) pigmented granular ganglion cells such as Gombault found in his patient's anterior horns are to be met with in aged persons, and are not strictly speaking pathological; (*b*) the diminution in the size of ganglion cells was caused by hardening in chromic acid.

This criticism appears to me insufficient and partial, for the reason that Westphal takes no cognizance of Gombault's positive statements that (*a*) numbers of cells were wanting in their accustomed places, and that (*b*) cells could be seen showing every degree of granular pigmentary degeneration from that to which Westphal refers, and which is recognized by all neurologists as quasi-normal, to that degree of change in which all that remains of a ganglion cell is a globular mass of pigment granules. In the second place Westphal's criticism upon Gombault's estimation of the size of altered ganglion cells is insufficient because no one knowing the method of work pursued by Charcot and his pupils can doubt that Gombault compared the cells in the sections of the cord of his patient, with those in sections of healthy cords which had been treated in exactly the same manner: *i. e.*, hardened in chromic acid, and subsequently mounted according to Clarke's method. Consequently, while admitting that it is possible that Gombault had an exaggerated opinion of the significance of moderate granular change in ganglion cells, I believe that there was a positive and pathological degeneration of the anterior horns in his case.

Histological examination of the spinal cord in the fresh state. Preparations from the softened part of the organ showed a whitish, pulpy mass containing great numbers of granular bodies, many of which are heaped muff-like about the blood vessels. A preparation from the centre of this softened tissue exhibited numerous normal ganglion cells with their processes. In the higher portions of the cord, where the gray and white substances were distinguishable to the naked eye, there were found some few ganglion cells, and a number of granular bodies round about the vessels.

The nerve roots throughout were small; anterior much the smaller, grayish, and semi-translucent; while the posterior roots were white and opaque. The microscope showed that these atrophied nerve-roots (fresh) were deprived of myelin, but were not filled with granulations. The connective tissue of these roots was embryonic.

After the spinal cord had been hardened in chromic acid, numerous sections were cut from it, and examined after preparation with carmine, etc. The nervous tissue did not harden well, and at the time of making the report it was impossible to make sections from the pons Varolii or the medulla oblongata.

Sections from the lowest (softened) part of the cord showed want of distinction between the gray and white substances of the organ. The anterior gray matter was much disfigured, and devoid of ganglion cells. Some good cells lay to one side of and behind the enlarged and choked central canal. In some sections no gray matter could be found.

Sections made through the lumbar enlargement showed the anterior horns of about normal size, but quite deprived of ganglion cells in their anterior and

internal parts. The outer and posterior masses of ganglion cells in the anterior horns, and the other ganglion cells in the posterior half of the organ were normal and well-preserved. The posterior horns were normal.

In the dorsal region the anterior horns were nearly everywhere unequal, and of different shapes; ganglion cells were more atrophied on the right side. Granular bodies had nearly disappeared through the process of preparation. There were very few ganglion cells in the anterior horns. The cells of the posterior vesicular columns were everywhere preserved. In the altered parts of the anterior horns there were fibroid tissue with lacunæ, and thickened bloodvessels.

In the cervical enlargement the alterations were about the same. There was found sclerosis or chronic inflammation of the gray substance of the anterior horns, with atrophy of many of their ganglion cells, thickened bloodvessels, enlarged perivascular spaces filled with blood or granular bodies.

Throughout the cord the lateral columns were sclerosed. This lesion was spread out a good deal around the anterior horns in the cervical region, but lower down it diminished in extent; and in the dorsal and lumbar parts of the cord it occupied only the posterior half of the lateral columns. It exactly resembled the degeneration occurring below a transverse section of the cord, and it seemed to be secondary to the atrophy of the anterior ganglion cells.

In brief the lesions in this case were granular degeneration of the ganglion cells of the anterior horns, a degree of diffused sclerosis of the anterior gray matter, and secondary descending degeneration of the lateral columns.

CASE V. (case xlii, p. 54.) The following is a summary of the results of the histological examination:

Muscles. The atrophied, yellowish muscles show simple atrophy of muscular fibres, with multiplication of the nuclei within the sarcolemma.

Nerves. The nerves supplying many muscles appear grayish to the naked eye. Small muscular nerves are removed at the moment of making the autopsy, acted upon by osmic acid, and then stained with picro-carminate of ammonia. Preparations from these nerves show that the myelin is broken up into drops and granulations, and that it is very scanty. The sheaths of Schwann are wrinkled in some places, and in others distended by masses of granular myelin, while between the masses of altered myelin there extends a substance colored yellow by the picro-carminate of ammonia. In other words the nerve fibres are yellowish bands of irregular diameter, distended here and there by black masses of myelin. The nuclei of the sheath of Schwann are much more numerous than in the normal state. Hardly any normal fibres are seen in the various preparations, and some of the altered fibres are composed only of the sheath of Schwann collapsed and wrinkled. The cylinder axis can not be found. The inter-fibrillar, the inter-fascicular, and the peri-fascicular connective tissue is the seat of proliferation. The above lesions are exactly similar to those which constitute the signs of the Wallerian degeneration, as we see it in animals two to three months after section of a nerve trunk.

The anterior roots of the spinal nerves contain a considerable number of similarly altered fibres.

Spinal cord. This organ is examined after two months' hardening in dilute chromic acid, by the usual

(Clarke's) process. The white matter of the spinal cord is healthy throughout, and so are the posterior vesicular columns of Clarke. In the anterior horns of the lumbar enlargement the ganglion cells are found very much diseased, and reduced in number. Even in the lower part of the enlargement it is seen that many ganglion cells are wanting, and this reduction in number increases as we approach the middle of the lumbar swelling. The disappearance of cells affects all parts of the anterior horns about equally, *i. e.*, no one group of cells is especially wasted. Many of the remaining ganglion cells exhibit various degrees of degeneration. Very few cells are larger than normal (the swelling of acute myelitis, Charcot). Many cells are diminished in volume, unnaturally globular, and their processes are broken off or difficult to make out. Granular matter appears in abnormal amount in the protoplasm round about the nucleus, and in some places all that remains of a cell is a nucleus environed by a small amount of granular protoplasma. In quite a number of cells a sort of cystic degeneration has occurred; suitable magnifying powers showing one or more vacuoles in the substance of the ganglion cells. The contents of these vacuoles is transparent, and does not much refract light; its nature is unknown. The neuroglia of the anterior horns is not the seat of any active change. The alterations of ganglion cells described above can be traced upward, in diminishing degree, as high as the eighth and ninth dorsal nerves. In the original article the text is supplemented by a colored lithographic plate illustrating the lesions found in the nerves and ganglion cells.

II. Autopsies in cases of myelitis of the anterior

horns occurring in children; so-called infantile spinal paralysis.

These post-mortem examinations have now become so numerous that the only way to introduce them into this small essay is in tabular form. Below will be found a table embodying the main features in twenty-nine cases. The first of the series are old, and were examined before physicians were acquainted with the mode of preparation of nervous tissues for microscopical examination. With one exception (No. 16, by Prof. Hammond) all autopsies made since 1865 (No. 13, by Prévost) show substantially the same results; in all there was granular degeneration and disappearance of ganglion cells of the anterior horns related to the paralyzed limbs, and in many of them there were, besides, myelitis of the gray and white substances, and descending degeneration of the antero-lateral columns. THE ONLY LESION FOUND IN ALL THE SIXTEEN CASES EXAMINED IN THE LAST TWELVE YEARS, IS ATROPHY OF THE GANGLION CELLS OF THE ANTERIOR HORNS.

The other lesions may be variously estimated. The sclerosis of the lateral columns observed in several instances is quite probably the descending degeneration which follows any severe lesion of the motor tract, cerebral or spinal: it exists only below the focus of disease in the nervous centres. Such being the probable explanation of the sclerosis of the lateral columns, it may seem remarkable that no ascending degeneration of the posterior median columns has yet been observed in poliomyelitis anterior. Its absence is probably due to the fact that the central and posterior gray matter is uninjured in this disease. As evidences of myelitis may be cited condensed patches of tissue, the formation of cavities, the presence of small hæmorrhagic infarcti in the gray matter.

AUTOPSIES IN CASES OF "INFANTILE SPINAL PARALYSIS."

No.	*Date.*	*Authors.*	*Age at Onset.*	*Age at Autopsy.*	*Mode of Onset.*	*Parts Paralyzed.*	*Lesions of the Nervous System.*	*Lesions of Muscles.*	*Bibliography.*
1	1825	Hutin.	7	49	Convulsions.	Both legs.	Atrophy of lower end of cord, and of its nerve roots.	Not stated.	Cited by Heine, Spinale Kinderlähmung. 1860. p. 151.
2	1842	Longet.	....	8	Not stated.	Right leg.	Atrophy of nerve roots of right sciatic nerve.	Idem.	Longet, Anat. et Phys. du Système Nerveux. 1842. I, p. 358.
3	1849	Fliess.	....	....	Idem.	Upper extremities.	Congestion of meninges over cervical enlargement of cord.	Idem.	Cited by Laborde, Paralysie de l'entance. 1864. p. 113.
4	1850	Rilliet et Barthez.	....	....	Idem.	One arm.	None (microscope not used).	Idem.	Gaz. Méd. de Paris. 1850. p. 681.
5	1850	Idem.	....	....	Idem.	Both legs.	Idem (idem).	Idem.	Idem.
6	1855	Behrend.	1	6	Idem.	Right lower extremity.	Chronic spinal meningitis.	Fibres small; not fatty.	Cited by Heine, op. cit., p. 150.
7	1863	Von Recklinghausen.	....	....	Idem.	Both legs.	Tubercles in cord.	Not stated.	Deutsche Klinik, Jan. 31, 1863.
8	1863	Cornil.	2	49	Unknown.	Idem.	Amyloid degeneration and atrophy of white columns of cord	Fibres fatty.	Gaz. Méd. de Paris. 1864. p. 290.

9	1863	Bouvier.	1	2	Idem.	All the limbs.	Sclerosis of antero-lateral columns of cord.	Fibres very small and granular; not fatty.	Cited by Duchenne fils, in Arch. Gén. de Méd. 1864. II, pp. 205-9.
10	1863	Henri Roger.	....	....	Idem.	Not stated.	Idem.	Not stated.	Idem.
11	1864	Laborde.	8 mos.	2	Febrile.	Both legs.	Idem; cells normal.	Fibres small; not degenerated.	Laborde, op. cit., p. 104.
12	1864	Laborde et Cornil.	1	2	Idem.	Idem.	Cortical sclerosis of cord; sciatic neuritis; cells normal.	Fibres small and granular; not fatty.	Idem, p. 109.
13	1865	Prévost.	....	78	Unknown (infantile?)	Left lower extremity.	Atrophy of cells of left anterior horn of cord; atrophy of left antero-lateral column.	Fibres fatty.	C. R. Soc. de Biologie. 1865. p. 215.
14	1866	Echeverria.	3	10	Febrile.	Right limbs.	Cells of anterior horns filled with granular pigment; diffused myelitis.	Fibres granular, not fatty.	Echeverria, On Reflex Paralysis. N.Y. 1866. p. 29.
15	1866	Idem.	2	2½	Febrile diarrhœa.	Both legs.	Nerve cells granular; diffused myelitis; nerves shrunken.	Fibres fatty.	Idem, p. 33.
16	1867	Wm. A. Hammond.	....	....	Palsy of four years' standing.	Left lower extremity.	Small clot in cord.	Not stated.	Journal of Psychological Med. N. Y. 1867. p. 51.
17	1868	Lockhart Clarke.	1	32	After cow-pox inoculation.	Upper extremities.	Atrophy of nerve cells of anterior horns of cervical enlargement: central myelitis.	Idem.	Med.-Chir. Trans. Vol. LI, p. 249. Lond. 1868.
18	1870	Charcot et Joffroy.	7	40	Sudden: non-febrile.	All the limbs.	Atrophy of cells of anterior horns: atrophy of antero-lateral columns; slight myelitis.	Fibres small but not degenerated.	Arch. de Phys. norm. et pathol. Paris. 1870. p. 135.

AUTOPSIES IN CASES OF "INFANTILE SPINAL PARALYSIS." (*Continued.*)

No.	*Date.*	*Authors.*	*Age at Onset.*	*Age at Autopsy.*	*Mode of Onset.*	*Parts Paralyzed.*	*Lesions of the Nervous System.*	*Lesions of Muscles.*	*Bibliography.*
19	1870	Parrot et Joffroy.	....	3	Unknown.	Left lower extremity.	Atrophy of cells of left anterior horn; atrophy of left antero-lateral column and nerve roots.	Idem.	Idem, p. 310.
20	1870	Vulpian.	....	69	Idem.	Right lower extremity.	Atrophy of cells of right anterior horn; some sclerosis of white columns.	Fibres small; striated, finely granular (fatty?)	Idem, p. 316.
21	1871	Roger et Damaschino.	1½ ?	2	After dysentery.	Left upper extremity.	Atrophy of cells of left anterior horn; sclerosis of antero-lateral columns; central myelitis and softening; left anterior nerve roots small.	Fibres small, with fatty granules, and numerous sarcolemma nuclei.	Gaz. Méd. de Paris. 1871. p. 480.
22	1871	Idem.	2	2½	After variola.	Both legs.	Atrophy of nerve cells of anterior horns; myelitis evidenced by sclerosis of antero-lateral columns, and central spots of softening.	Fibres small; in part striated; with much granulo-fatty deposit.	Idem, p. 505.
23	1871	Idem.	2	3	Febrile.	Both legs and left side of back.	Atrophy of cells of anterior horns; patches of softening in gray matter; sclerosis of antero-lateral columns.	Idem.	Idem, p. 541.
24	1871	Müller.	4	35	Fall from bed (?)	Right leg; double club-foot.	Atrophy of cells of anterior horns in lower lumbar enlargement, especially on right; sclerosis of white columns.	Fatty.	Beiträge zur pathol. Anat. u. Phys. des Rückenmarks. Leipzig. 1871. p. 15.

25	1873	Lancereaux et Pierret.	2	18	.....	Left arm.	Atrophy of cells of left anterior horn; atrophy and sclerosis of left half of cord.	Not stated.	Cited by Petitfils, Atrophie aigue des cellules motrices. Paris. 1873. p. 33.
26	1873	Roth.	1	2	Febrile.	Both legs.	Atrophy of cells of anterior horns of lumbar enlargement, and central myelitis; atrophy of anterior nerve roots.	Not stated.	Virchow's Archiv. 1873. Bd. LVIII, H. 2, p. 263.
27	1875	Leyden and Von Recklinghausen.	9 mos.	21 mos.	Sudden.	Both legs; left more.	Atrophy of nerve cells in anterior horns (left more) in lumbar enlargement. A trace of same lesion in cervical region. Slight atrophy of antero-lateral columns.	Simple atrophy of muscles, with moderate fatty infiltration.	Archiv. of. Psych. u. Nervenk. VI. 1875. p. 271, obs. II.
28	1875	Leyden and Von Recklinghausen.	4	58	Sudden.	Both legs; left more.	Atrophy of lower anterior roots; anterior horns atrophied, nerve cells gone, especially in lumbo-dorsal regions. Very numerous amylaceous corpuscles in gray matter.	Various degrees of fatty infiltration, even to complete loss of striation.	Idem, obs. III.
29	1875	Leyden and Friedländer.	3	20	Sudden.	Left arm and right leg.	Cervical region; left ant. horn small, cells shrunken; left lateral and post columns in gray degeneration. Lumbar region: right ant. horn atrophied, with few cells; right lateral column sclerosed. Some chronic meningitis.	Fibres small; moderately granular; separated by much fatty tissue.	Idem, obs. IV.

N. B. I do not include in the above table one case by Allbutt (Lancet 1870, ii. p. 84) and Obs I. of Leyden's paper because of evident traumatism.

The lesions found in the wasted muscles in these cases consisted of simple atrophy, of granular degeneration, and of fatty degeneration of muscular fibres, and there was often found multiplication of the nuclei of the sarcolemma.

III. Pathological anatomy of other cases in which muscular atrophy is a symptom.

a. Progressive muscular atrophy. This affection closely resembles the chronic form of myelitis of the anterior horns, and a paragraph will be devoted to their differential diagnosis further on. Thanks to the researches of J. Lockhart Clarke,[1] Charcot, Hayem and many others, it is now well-established that the central lesion in this disease is granular degeneration and disappearance of the ganglion cells of the anterior horns related to the wasted muscles.

b. Labio-glosso laryngeal paralysis. In this disease, which consists of paralysis and atrophy of certain facial, intra-buccal, pharyngeal, and laryngeal muscles, Charcot[2] has discovered a similar degeneration and disappearance of the ganglion cells forming the nuclei of the hypoglossal, facial and spinal accessory nerves in the floor of the medulla oblongata. E. R. Hun,[3] of Albany, and others have verified the correctness of the observation.

c. Muscular atrophy complicating cases of locomotor ataxia (sclerosis of the posterior columns of the spinal cord). In 1870, Dr. Pierret,[4] under the direction

[1] British and Foreign Medico-Chirurg. Review, 1862, II., p. 215.

[2] Archives de phys. normale et pathol. 1870, pp. 247–260.

[3] Labio-Glosso-Laryngeal Paralysis. American Journal of Insanity, 1871, p. 194.

[4] Sur les altérations de la substance grise de la mœlle épinière dans l'ataxie locomotrice, considerées dans leurs rapports avec l'atrophie musculaire qui complique quelquefois cette affection. Arch. de phys. norm. et pathol. 1870, pp. 599–617.

of Prof. Charcot examined the spinal cord in a case of this sort, and found that in a part of the cervical enlargement corresponding to the wasted arm, the sclerosis had extended from the posterior columns of the spinal cord into its anterior horn, destroying the mass of ganglion cells known as the external lateral group.

d. Muscular atrophy occurring in cases of common hemiplegia of cerebral origin. Charcot[1] has had the opportunity of examining the spinal cord in a case of this kind, and he discovered that there was sclerosis, with degeneration and disappearance of ganglion cells of the anterior horn on the same side as the paralysis; this morbid process being an extension of the descending degeneration which is usually limited to the lateral column.

e. Muscular atrophy in chronic diffused central myelitis. In this complex affection, which results in disorganization of the central part of the cord with formation of a plug or of a distended cavity, the anterior gray matter is encroached upon, its ganglion cells undergo granular degeneration and disappear—the externally apparent symptom of this lesion being muscular atrophy, which goes, in these cases, along with anæsthesia, akinesis, bed-sores, etc.[2]

From the numerous facts classified in the three categories just enumerated, it appears to me that we can safely and correctly draw one general conclusion, and frame a pathological law:

Muscular atrophy, whether localized or generalized,

[1] Leçons sur les maladies du système nerveux. Paris, 1872. P. 55, footnote.

[2] Consult Schüppel, Ueber Hydromyelus. Archiv der Heilkunde, 1865, p. 289.

Hallopeau, Etude sur les méylites chroniques diffuses. Archives gén. de médicine, 1871, II., pp. 277, 435, 565; 1872, I., pp. 60, 191.

depends upon a destructive lesion of ganglion cells in the anterior horns of the spinal cord; except where it is caused by what is strictly speaking peripheral paralysis, or a lesion of a nerve.

Or we may invert the proposition, and say that a destructive lesion of ganglion cells of the anterior horns of the spinal cord produces atrophy of the muscles dependent upon the affected cells for innervation.

As far back as 1868, Prof. Charcot, in a clinical lecture delivered at the Salpétrière in Paris, announced that the relation between granular degeneration of motor nerve cells and muscular atrophy was that of cause and effect. This proposition Charcot has since maintained, and he and others have steadily accumulated evidence to support it.

From the data presented above we may draw, besides the principal law, the following secondary proposition:

In myelitis of the anterior horns occurring in adults and in children the chief and constant lesion is destructive degeneration of ganglion cells.

There now remains for discussion the following important question:

Is the lesion in spinal paralysis of adults and children, a myelitis strictly speaking, or is granular degeneration of the ganglion cells the primary and essential pathological factor?

It appears to me that we do not yet possess the elements necessary for the solution of this problem. There are data of three categories which tend to somewhat contradictory conclusions.

1. Autopsies in cases of myelitis anterior in children all tend to prove the existence of lesion of the neuroglia of the anterior horns as well as of degeneration of their

ganglion cells. In some cases this lesion was sclerosis, in others patches of softening. Besides, there was secondary degeneration of the lateral columns in many cases. These cases would seem to indicate that myelitis of the anterior horns was the essential phenomenon. Against this conclusion it has been observed that in several instances the autopsy was not made until after the lapse of several or many years, and some of the alterations might be secondary; but on the other hand quite similar lesions existed in the spinal cord in cases 15, 21, 22, 23, 26, and 27, in which the post-mortem examinations were made not long (from six months to one year) after the onset of the disease.

2. Among adults, in autopsies III. and V., (cases xiv and xlii) nothing was found except degeneration of ganglion cells of the anterior horns; in the former case by granular change, in the latter by the formation of vacuities in the cell and shrinkage. These cases would seem to show that degeneration of the ganglion cells of the anterior was the primary and essential lesion.

3. The results of treatment, as will be seen in Chapter VIII, speak strongly in favor of the inflammatory theory of the disease. Such medicines as ergot and iodide of potassium seem to exert a favorable effect upon the morbid process, and the application of sharp counter-irritation has in several instances done much to check the disease.

In conclusion it appears to me that the weight of evidence in the very incomplete argument which can be made now upon this new question in pathology, is in favor of the existence of severe congestion and myelitis of the anterior horns in this disease. Future research and progress may modify this view, and may even cause us to admit varieties (pathologically speaking) of poliomyelitis anterior.

CHAPTER VI.

DIAGNOSIS.

a. The positive diagnosis of myelitis of the anterior horns is to be made by a careful consideration of the symptoms as grouped together in Chapter IV. In very general terms it may be said that an atrophic paralysis with rapid loss of faradic reaction in the palsied parts, is myelitis of the anterior horns. Of course this does not include cases in which a mixed nerve has evidently been injured, producing peripheral paralysis.

b. Differential diagnosis. This needs to be made from a number of spinal and other affections.

1. Peripheral paralysis. In this case when there is doubt as to the nature of the injury received, a diagnosis can be arrived at by carefully determining what muscles are paralyzed. In a peripheral paralysis the atrophic paralysis is strictly in the range of distribution of the motor filaments of the injured nerve. Mothers have presented children at my clinic with the statement that the paralysis had been caused by a blow, or by the penetration of a needle near a great nerve, and I have easily arrived at the correct diagnosis of poliomyelitis acuta by the above rule, by the want of chronological agreement between the reception of the injury and the appearance of paralysis, and by the absence of the symptoms of traumatic neuritis (neuralgia, anæsthesia, alterations of nutrition). A few months ago it might have

been added that the discovery of the reaction of degeneration in the paralyzed muscles might serve to designate peripheral paralysis, but we now know, thanks to the patient researches of Prof. Erb,[1] of Heidelberg, that this morbid electrical reaction is likewise present in myelitis of the anterior horns.

2. Paralysis a frigore, rheumatic paralysis wrongly so-called. It appears very doubtful to me whether these cases of limited atrophic paralysis (deltoid, serratus magnus) are not after all instances of very circumscribed myelitis of the anterior horns. The clinical aspects of the two affections are wonderfully similar, and fresh electrical and pathological researches will be necessary to convince me of their non-identity.

3. Atrophy following neuralgia. In many cases belonging to this category there is only moderate wasting of the muscles of the affected part, and these muscles present simply diminished faradic and galvanic reactions. In some of these, the atrophy is to be attributed to enforced or instinctive rest, and no error in diagnosis is possible. But I have seen several individuals who after having suffered from severe neuralgic pains (in shoulders, upper arms, or thighs) have developed a true atrophic paralysis with loss of faradic contractility. These I am now inclined to look upon as instances of chronic central myelitis with involvement of the anterior horn. In the moderate atrophy accompanying chronic sciatica (the wasting due to prolonged vascular spasm according to most authorities) there is no marked impairment of faradic contractility.

4. Spinal congestion. Very many of the symptoms of this affection are exactly like those of myelitis of the

[1] Ueber acute Spinallähmung (Poliomyelitis anterior acuta) bei Erwachsener. Arch. f. Psych. u. Nervenk., v., p. 758. 1875.

anterior horns; rapidly developed general, or gradually ascending paralysis without marked anæsthesia or palsy of the bladder, being observed in each disease. But in congestion there are never positive atrophy and loss of faradic reaction in the paralyzed muscles.

5. Acute ascending paralysis. In the former edition of this essay[1] I expressed the opinion that this disease, while resembling in many respects myelitis of the anterior horns, was different from it. Dr. William A. Hammond in the last edition of his Treatise considers this view as erroneous, and claims that the two diseases are identical.[2] Without entering into a discussion of the reasons for Dr. Hammond's opinion, I may say that I am fortified in the reiteration of my view by the conclusions recently reached by Prof. C. Westphal.[3] This high authority after a thorough study of three cases of acute ascending paralysis expresses his belief that there are three strongly marked points of difference between the two diseases. 1. In acute ascending paralysis asphyxia though affection of the medulla oblongata is frequent; 2. In this disease the paralyzed muscles do not lose their faradic contractility; and, 3. In numerous autopsies, including those in his own three cases, no lesion of the central nervous system has been found. To repeat: In acute ascending paralysis we have a rapidly extending akinesis, involving the respiratory muscles, and rapidly (one to three weeks) causing death by asphyxia. Muscular atrophy is absent and the paralyzed muscles do not lose their faradic contractility. I may add that this recently acquired knowledge should

[1] P. 29. [2] Op. cit. p. 482.

[3] Beobachtungen u. Untersuchungen über die Krankheiten des centralen Nervensystems. Ueber einige Fälle von acuter tödlicher Spinallähmung. Arch. f. Psych. u. Nervenk., vi., 3, p. 765. 1876.

be applied to the criticism of some of the older cases of so-called acute ascending paralysis, and such as exhibited loss of faradic contractility in the paralyzed muscles should be transferred to the category of myelitis of the anterior horns.

6. Central diffused myelitis. In the immense majority of cases there is no reason for confusing this disease with myelitis of the anterior horns. Central diffused myelitis produces besides paralysis and (at times) muscular atrophy, great anæsthesia co-extensive with the paralysis, and the bladder is paralyzed in a positive manner. Besides, the course of the disease is usually very chronic.

7. Hæmatomyelia, or hæmorrhage into the gray matter of the spinal cord. This affection produces sudden paralysis (paraplegic or general) just as occurs in very acute non-febrile myelitis anterior, but in addition we have extreme anæsthesia, and palsy of the rectum and bladder. In a few days great muscular atrophy may set in, and the muscles may lose their faradic contractility, as in poliomyelitis.

8. Progressive muscular atrophy. The sub-acute non-febrile and chronic forms of myelitis of the anterior horns may simulate muscular atrophy. It should be borne in mind that in the latter disease the progress of atrophy is much more gradual and irregular than in the former. In progressive muscular atrophy a muscle perishes by bundles of fibres, while in chronic myelitis anterior the whole muscle gradually wastes. Besides, in progressive muscular atrophy faradic contractility is preserved in the wasting muscles as long as any muscular tissue remains, while in myelitis anterior whole muscles show diminished or lost contractility. Still there can be no doubt that the two diseases are very

closely allied, the only difference between them being, probably, that in progressive muscular atrophy the degeneration of ganglion cells of the anterior horns takes place cell by cell and very gradually in each cell, whereas in myelitis anterior groups of cells primarily or secondarily undergo a destructive change.

In the Nosological Scale, myelitis of the anterior horns, in adults and children, is closely related, on the one hand to spinal congestion and acute ascending paralysis, and on the other to progressive muscular atrophy and labio-glosso-laryngeal paralysis.

CHAPTER VII.

ÆTIOLOGY.

Analysis of the forty-five cases recorded in Chapter II. yields the following data.

1. Predisposing causes.

a. Sex. Of the forty-five cases, thirty, or 66.6 per cent., were males, and fifteen or 33.3 per cent., females. In the infantile form of poliomyelitis anterior the same proportion does not hold good, and children of both sexes are about equally liable.[1] This difference is probably owing to the fact that at the age when children suffer from myelitis boys are not more exposed than girls.

b. Age. The forty-five cases may be classified as follows with respect to age. Thirteen, or 28.8 per cent., were from twenty to twenty-nine years of age; eleven, or 24.4 per cent., were from thirty to thirty-nine years old; eight, or 17.7 per cent., were from forty to forty-nine years; seven, or 15.5 per cent., were from fourteen to eighteen years old; three were fifty-five to fifty-seven years of age; one was over sixty; and in two cases ages are not stated. The extreme ages were fourteen years (case xxviii, by Erb), and sixty-seven years (case xiv, by Gombault). It would therefore seem as if poliomyelitis were a disease of the best time of life, the period be-

[1] Laborde, De la paralysie (dite essentielle) de l'enfance. Paris, 1864. p. 98.

tween eighteen and forty years. In children, considerable statistics[1] indicate that by far the greater number of cases occur between the ages of one and two years; the disease being very rare after the fourth year.

c. With respect to Inherited Predisposition, Occupation, Temperament, etc., we have no data worth writing down. It may be added that among children, the strong and healthy ones are the usual victims.

II. Exciting causes.

In twenty-one cases, or 46.6 per cent., no cause or preceding pathological state is given: cases i, v, vi, vii, x, xii, xix, xx, xxiii, xxv, xxvi, xxviii, xxx, xxxi, xxxiii, xxxiv, xxxv, xxxvii, xxxix, xl, and xliv.

In two instances the disease followed measles: cases viii and ix.

In one instance it developed after cerebro-spinal meningitis: case xliii. Once it followed dysentery: case xi. Once it occurred after confinement: case xxii.

In one instance, case xxxviii, the menses were suppressed before the disease appeared.

In one instance, case xlii, myelitis of the anterior horns occurred during the height of secondary syphilis.

In one case, No. xxi, sexual and other excesses, "a fast life," preceded the disease.

In case xli, overwork and anxiety were antecedent circumstances.

In one instance, case ii, atrophic paralysis set in after an effort during which the back was strained.

Finally, in twelve instances, or 26.6 per cent., exposure to cold and dampness is distinctly referred to as a cause: cases iii, xiii, xiv, xv, xvi, xvii, xviii, xxiv, xxvii, xxix, xxxii, xxxvi.

[1] Duchenne, De l'électrisation localisée. 3me ed. Paris, 1872, p. 417. Laborde, op. cit., p. 97.

We must conclude that the last-named agencies, cold and dampness are the only exciting causes we know of myelitis anterior in the adult. Consequently it would appear that myelitis of the anterior horns is often a paralysis a frigore. It seems very doubtful that syphilis can be a cause. Dr. Déjérine in commenting upon case xlii, admits that there probably was a mere coincidence of syphilis and lesion of the anterior horns.

As regards poliomyelitis occurring in children, we have even less positive ætiological knowledge. It appears that in many cases peripheral irritations, such as teething, intestinal disorder, etc., have produced the disease. Probably, cold is a cause of myelitis anterior in young patients, as well as in older ones.

CHAPTER VIII.

TREATMENT.

THE treatment of poliomyelitis may be divided into two parts. The treatment of the acute stage; and that of the late stages of acute cases, or of chronic cases.

I. Management of acute myelitis anterior. If there be fever this may be reduced by sponging, digitalis, or salicylic acid. Judging by the records of temperature now in our possession the bodily temperature is never so high as to call for the cool bath. It is to be remembered that in some cases there are, in addition to the fever, no symptoms indicating impending local disease. When these are present, or in the non-febrile acute cases, we should at once proceed to apply counter-irritation to the spinal region, and to administer ergot, belladonna, and iodide of potassium.

a. Counter-irritation. This may be done by means of dry cups, blisters, tincture of iodine, or the actual platinum cautery. In two of my own cases (xxi and xlv) dry cups were employed at my suggestion, and the same means seem to have done good to other patients. Tincture of iodine and blisters are both open to the objection that they produce a teasing pain, and by making the skin sore, open the way for the development of an eschar. On the whole I much prefer the actual cautery. It is simple in its application, and not very painful; it does not produce blisters (not once in twenty or thirty times

in my experience); it brings about a strong revulsion as evidenced by a wide area of hyperæmia round about the burn; and, lastly, it has seemed to exert a remarkable influence over the disease, as in cases xxxii and xxxv. The platinum button, heated to whiteness by a compound blowpipe (air and burning gas) arrangement, is to be lightly applied in streaks over the spinal region, from two to eight strokes being made at each application. I do not favor the use of localized anæsthesia as advised by Dr. Hammond,[1] because the pain of the application is not difficult for even delicate women, to endure, and more especially because I believe that by inducing anæsthesia of the sensory nerves of the part to be cauterized we greatly reduce the central effect of the cutaneous irritation. We do not thoroughly understand the modus operandi of counter-irritation, but so far as we now know, it affects centripetal (sensory) nerves and through them modifies in some way the molecular (chemical) activity of the anatomical elements of the nervous centre (or other organ), or modifies its vascular supply by acting on vaso-motor centres or nerves. Hence the procedure with local anæsthesia appears to me highly unphysiological.

b. Ergot should, I think, be our chief reliance in the treatment of these cases, assuming that the first stage is one of hyperæmia or of lesion complicated with hyperæmia. I have been in the habit of prescribing Squibb's fluid extract of ergot in doses of ℥xx or ℥xxx four times a day, gradually increased until the patient take ʒij four times a day. This has been my method in cases of myelitis of the anterior horns, and spinal congestion as well. It is only rarely that nausea is produced after the first day or two, and I have never seen

[1] Diseases of the Nervous System. 1876. P. 489.

any evil effect from the taking of ℥j or ℥jss of the extract per diem. Ergot was administered in cases xxi, xxxviii, xxxix, and xl. In case of marked repugnance to the taste of the fluid extract, ergotin (Merck's) may be substituted in doses of gr.v or x or more, four times a day, exhibited in capsules; or we might employ Merck's new dialyzed extract of ergot hypodermically. With the last named preparation I have had no experience, and can not speak of its relative value and strength.

c. Belladonna is indicated in poliomyelitis anterior as in other inflammations of the spinal cord. It probably causes more or less permanent contraction of the small arteries of deep organs. I usually give belladonna in the shape of the alcoholic extract, in doses of gr.$\frac{1}{4}$ or $\frac{1}{3}$ three or four times a day; just enough to produce slight dryness of the fauces. This medicine and the ergot should be continued in full doses until the symptoms (especially numbness and pain) diminish, when the doses may be gradually reduced.

d. Iodide of potassium. This medicine has been administered in several cases, (x, xxxiii, xxxix, xl, and xlii) and apparently with such good effect that I am bound to speak of it as one of the drugs to be used, although the rationale of its action is not by any means clear. It certainly does not do good by curing any syphilitic taint, as it can not be shown that syphilis is ever a cause of myelitis of the anterior horns. As regards the use and mode of administration, I see no objection to giving the iodide at the same time with ergot and belladonna, except that it may interfere with our study of the effect of these drugs. The doses should be moderate, from gr.x to ℥j three times a day, dissolved in water alone, or with a little carbonate of ammonia.

Electricity should, at this period, be very gently and sparingly used, if at all. I am of the opinion that in acute and sub-acute febrile cases it ought not to be employed until all symptoms of irritation pass away; and in non-febrile acute and sub-acute cases it should be employed when the progress of the disease seems arrested by internal treatment. By a gentle application of electricity, I mean the use of a descending galvanic current of from six to ten Stöhrer or Leclanché elements, the anode to be placed on the spinal column, and the cathode applied labile (stroking) on the weak muscles. Any attempt to produce strong muscular contractions by the galvanic or faradic current, is to be condemned in acute cases, except once for the purpose of making the diagnosis certain.

Of course, minor indications arising in the acute stage of myelitis anterior, such as temporary retention of urine, constipation, unusual prostration, etc., should be met by appropriate means.

II. Management of the later (residual) stage of myelitis of the anterior horns, and of chronic myelitis of the anterior horns.

a. In the later stages of acute and sub-acute poliomyelitis we have to deal with a more or less limited atrophic paralysis, and it is to the removal of this that our therapeutical agencies should be directed. The signs of active diseases in the spinal marrow are wanting, and we may diminish or suspend the antiphlogistic medication which has been employed heroically in the first period.

For the cure of the muscular atrophy and its consequent deformity and impaired function, we employ electricity, baths, rubbing and exercise, orthopedic appliances, and medicines.

1. As regards Electricity, the general rule that we must employ the current which produces contractions in the palsied muscles, is to be observed in this as well as in other affections. As a matter of fact it is found that the faradic current produces no effect upon the muscles which have suffered the most from myelitis of the anterior horns, and we are therefore led to employ galvanism. If any paretic muscles do respond to faradism this should be applied to them, at first gently.

Galvanism, or the continuous current, is to be applied as follows: the positive pole or anode to be placed upon the spinal column near the affected extremity, or upon the nervous trunks of that extremity. For example, to galvanize a paralyzed leg, the anode may be put upon the lumbar vertebræ, or upon the sciatic nerve in the thigh. The cathode is then to be applied to the muscle; first to the motor nerve of the muscle, and, if that produces no effect (the current of from eight to twenty cells being in circuit), it is then to be placed upon the muscle itself; either laid upon it, or applied labile (by strokes upon the belly of the muscle). When the cathode of a sufficiently strong current is thus applied labile we nearly always obtain a degree of muscular contraction, slow and imperfect, however. If the cathode be placed upon the muscle or motor nerve of the muscle and left stationary (stabile), it is necessary to close and open the circuit by means of a mechanism attached to the battery or to one of the electrodes. A slow contraction ensues, even in very much atrophied muscles, at the opening and closing of the circuit; often stronger at the opening (reaction of degeneration).[1] I

[1] For full details concerning the reactions of paralysed muscles, consult Ziemssen's Cyclopedia of the Practice of Medicine, vol. X. Diseases of the Nervous System, by Prof. W. Erb. p. 271 and p. 424.

very much like for these applications a hollow hard rubber electrode [1] containing a simple switch apparatus for interrupting and reversing the current. By means of this electrode (so-called rheodegos), it is possible to obtain contractions with a milder current than would be required with simple electrodes. This application should not be made too severely; it is quite unnecessary to obtain great muscular contractions; and each muscle should not be stimulated for more than one or two minutes. I usually allow three minutes for the galvanization of a muscular group, and from five to ten minutes for that of a whole extremity. I would repeat that no more current should be employed than is necessary to produce muscular contraction; and often the number of cells in circuit has to be reduced after a few minutes of application.

The faradic current should be tried from time to time, and when muscles have so far recovered as to respond to its stimulus, it should be regularly applied after galvanism, and ultimately applied alone.

The above rules for the application of the galvanic and faradic currents are, it appears to me, quite as applicable to cases of myelitis of the anterior horns in children, as to cases in the adult.

It occasionally happens (see case xliii) that muscles will not respond to galvanism because they are unnaturally stretched by the preponderating action of their antagonists. In such cases, unless some special surgical reasons contra-indicate interference, tenotomy should be performed upon the contracted muscles, and the limb kept straight by a plaster dressing or a mechanical contrivance. Besides, the suggestion of Dr.

[1] Made by the Galvano-Faradic Manufacturing Co., New York.

John Van Bibber,[1] of Baltimore, to apply an elastic band to relieve the tension of the paralyzed muscle, may be used to advantage.

2. Baths. Local hot and cold douches have been recommended by all authorities as calculated to improve the nutrition of paralyzed and atrophied muscles, and I am disposed to admit their efficacy. The best combination is to apply cold first and heat afterward. Cold alone does very well, as succeeding its constricting action on the bloodvessels of the part, we obtain the secondary effect of vaso-dilatation; the reaction so-called. In case xliii the application of galvanism was apparently made more efficacious by immersing the legs in hot water for a few minutes before the electrization. The addition of mustard or other rubefacient to the water is hardly called for.

With general baths I have had no experience in this disease. From my observations upon the treatment of common myelitis and sclerosis of the posterior columns of the cord by means of sulphur baths, I should be disposed to hope something from their use in poliomyelitis. The natural sulphur springs may be resorted to, but nothing can be better than the steam or hot-air sulphur baths which may be had in the Russian bath establishments of all large cities. The effect of these baths is, I take it. not merely to improve the circulation in peripheral parts, but also to favorably affect the circulation and nutrition of the central nervous system.

3. Rubbing and exercise. The rubbing should be of the kind termed massage, viz., a kneading and manipulation of the muscles rather than a mere friction. Each atrophied muscle should be carefully worked over

[1] On the Treatment of Paralyzed Muscles by Elastic Relaxation. Trans. of the American Neurological Association, vol. I. 1875, p. 244.

with the operator's thumbs and fingers for one or two minutes. On the general principle that over-use and too great stimulation do harm to highly organized tissues, I am in favor of using massage and galvanism at different times; one in the morning and the other in the afternoon, or these powerful means should be used on alternate days.

Exercise should not be other than passive until after all symptoms of irritation of the spinal cord have disappeared; and then active movements should be regulated in amount and kind by the physician. A patient who has a moderate degree of atrophy, pretty uniformly distributed over one or two limbs may I believe take exercise quite freely, in proportion to his general strength, but where, as in children especially, the atrophy is localized in one muscular group in such a way as to destroy the balance of forces about an important joint (foot, or knee) active exercise should be very limited until a proper supporting apparatus is applied, or until a degree of recovery takes place. I have certainly seen persons of all ages with atrophic paralysis making painful attempts to walk about, when every step caused greater muscular disintegration than could be replaced by any system of treatment.

4. Orthopedic apparatus. It is out of my province to discuss the shape and materials of which they should be constructed, and to give minute directions for their application. I may, however, be allowed to express my views of the principles upon which all such contrivances should be constructed when they are to be aids to treatment. In the first place they should be light, as light as can be strong; secondly they should be so applied as to cause no pressure upon nerves or important blood-vessels; thirdly, they should nearly always combine elas-

tic force with simple support. With the application of apparatus after treatment is abandoned, in the incurable stage of myelitis anterior, I have naturally nothing to do.

5. Medicines. I have very little faith in the efficacy of drugs in the residual stage of poliomyelitis. Barwell has recommended hypodermic injections of strychnia in the infantile cases, and Heine administered it internally many years before him. I should, in the present state of our knowledge, give no medicines in these cases except tonics (iron, cod-liver oil, small doses of phosphorus or strychnia), and nutritious food. This, with life out-of-doors, and, in the case of adults, freedom from cares and mental labor, will I think do something toward remedying the mischief done in the spinal cord during the early period of the disease.

b. The treatment of chronic poliomyelitis anterior. We have as yet too little experience with this form of the affection to enable me to lay down rules for its management. I should be disposed, reasoning by analogy, to apply counter-irritation to the spine, to administer ergot and iodide of potassium carefully, and to employ the galvanic current very gently to the wasting muscles. Almost complete physical rest should be observed, and everything done to favor general nutrition.

CHAPTER IX.

PROGNOSIS.

Analysis of the forty-five cases of myelitis anterior in the adult which form the basis of this essay yields the following data:

Of the forty-five patients, five, or 11.1 per cent, died; cases v, xiv, xxii, xxxii, and xlii.

Of these only two lost their lives directly through poliomyelitis; case v, by exhaustion, and case xxxii, by asphyxia. The three remaining cases died from other causes.

Of the forty patients who survived, twelve, or 26.6 per cent., recovered perfectly; viz., cases xv, xviii, xix (?) xxi, xxv, xxvi, xxxiii, xxxiv, xxxv, xxxviii, xxxix, and xlv.

In twenty-seven patients, or 60.0 per cent. partial recovery, *i. e.* with incurable residuum of atrophic paralysis, took place. The residuum varied in extent from atrophy of one muscle to that of entire limbs. Such was the issue in cases i, ii, iii, iv, vi, vii, viii, ix, x, xi, xii, xiii, xvi, xvii, xx, xxiii, xiv, xxvii, xxviii, xxix, xxx, xxxi, xxxvi, xxxvii, xl, xli, xliii.

In case xliv, the issue had not been learned at time of writing.

With respect to the connection between perfect recovery and the type of the disease, it may be said that some of each type recovered entirely.

As regards the issue in myelitis anterior in children

I have no exact statistics to present to the reader. From a careful survey of the literature of infantile spinal paralysis, and from the results of my own experience, I believe that perfect recoveries are even more rare, proportionately, in children than in adults.

a. In general terms we may, therefore, conclude that poliomyelitis anterior, whether acute, sub-acute, or chronic, occurring at any period of life, is very rarely dangerous to life, but usually leaves behind it impairment of the function of some limb; corresponding no doubt with a permanent lesion in the anterior horns of the spinal cord.

b. The prognosis in individual cases under different circumstances.

1. During the height of the acute or sub-acute attack, our prognosis must be guarded. The invasion of muscles in the throat and mouth and about the trunk, renders the chances of recovery small.

2. During recovery from an acute or sub-acute attack of myelitis anterior, our hope of absolute recovery must be based upon the absence of extreme localized atrophy, and upon the preservation of a degree of reaction to the two electrical currents. The presence of the reaction of degeneration should make us cautious in expressing a favorable prognosis.

3. In chronic myelitis anterior, no anticipation of a favorable issue is justified until all symptoms of central irritation (pains and numbness) have passed away, and until the electrical reactions of atrophied muscles are observed to approach the normal formulæ.

4. Children who have myelitis of the anterior horns, are usually brought to the physician months after the onset of the disease, bearing one or more atrophied limbs, and we are asked to say if the child will get

well. In a lecture upon infantile spinal paralysis[1] (1874) I expressed myself as follows: "One authority says 'if the muscles can be made to contract with either the induced or the primary currents, the cure is merely a matter of time and patience,' but I am afraid that this is rather a sanguine expectation. I should give a very guarded prognosis, under these circumstances, in all cases having lasted beyond a year." Experience since then has made me even more skeptical as to the value of this guide in prognosis. We now know that the muscles whose motor nerves have been severed in such a way as to preclude all chances of recovery will yield galvanic reaction for much more than a year after the operation. The reaction is a proper muscular reaction, and has nothing to do with the innervation of the muscle, or the state of the nervous centres. I am now observing a patient in whom Dr. C. K. Briddon, of this city, removed a large neuroma of the musculo-spiral nerve, including a long piece of the nerve, and in whom probably no recovery can take place; and in this patient's forearm, sixteen months after the operation, I still get marked, though slow and vermicular contractions, with the current of from twenty to thirty large Leclanché elements. I am at present inclined to the opinion that unless cured in two or three months, children with myelitis anterior have very little prospect of perfect recovery.

Of course the finding of some degree of reaction to faradism in atrophied muscles gives a rational basis for a good prognosis.

In other words, I believe that the prognosis in adults and children depends upon a factor which we can only indirectly determine, I mean the amount of injury done

[1] New York Medical Record, January 15, 1874.

to ganglion cells of the anterior horns. We have not as yet, and probably a long time will elapse before we shall have, the elements for saying that ganglion cells may be regenerated, or even that they may be purged of accumulations of granular matter; and when we shall know this we may be yet unable to apply the knowledge to individual cases in such a way as to give us as sure a prognosis as we now have in many visceral diseases.

CONCLUSIONS.

1. Careful study of a large number of cases of disease shows that there is an affection common to all ages of life, characterized by atrophic paralysis of one or more limbs, rarely of other parts, loss of faradic reaction, and diminution or loss of galvanic reaction in the paralyzed muscles; by remarkable freedom from anæsthesia and retention of urine, and absence of bed-sores; and by temporary numbness and referred pains.

2. The Course of the disease in children is nearly always acute; in about fifty per cent. of cases acute and febrile. In adults it runs an acute febrile, an acute non-febrile, a sub-acute febrile, a sub-acute non-febrile, or a chronic course. The last-named type of the disease is rare; excessively so in children.

3. In adults and in children the Lesion of the spinal cord consists in myelitis of the anterior horns, with atrophy (through granular degeneration) of the ganglion cells of this part. The motor cells constituting the trophic centre (Waller's law) of nerve trunks are destroyed, and the resulting atrophic paralysis greatly resembles that of peripheral paralysis.

4. The Causes of myelitis of the anterior horns are: Predisposing; age and sex. In childhood the two sexes are about equally liable to the disease, and are more frequently attacked when from one to four years of age. Adult males are much more often affected than women; and the best years of life, twenty to thirty-five years, are

those during which the disease occurs. Exciting causes. The only one that we have good reason to believe effectual is the impression of cold or dampness ; the affection is often, consequently, a paralysis *a frigore.*

5. The Treatment consists in the heroic use of antiphlogistic means, such as counter-irritation to the spinal region, the internal administration of ergot, belladonna, iodide of potassium, during the acute and subacute stages of the disease; and of the judicious use of galvanism, faradism, massage, douches, tonic and supporting treatment, and orthopedic apparatus during the stage of residual atrophy.

6. The Prognosis is very good as regards life, but unfavorable with respect to function of paralyzed parts ; very few patients recover perfectly from myelitis of the anterior horns.

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