



CASE XVIII.

Left leg palsied in 1871—right leg in 1873, acutely.

SPINAL PARALYSIS OF THE ADULT;

ACUTE, SUBACUTE, AND CHRONIC.

(*INFLAMMATION OF THE MOTOR TRACT OF THE
SPINAL CORD.*)

A PAPER READ BEFORE THE NEW YORK ACADEMY OF MEDICINE,
NOVEMBER 5, 1874.

BY

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COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK.

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By E. C. SEGUIN, M. D.,

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MR. PRESIDENT, AND FELLOWS OF THE ACADEMY :

I desire in this essay to call your attention to a rare form of myelitis, one whose existence has been revealed to us only within the last few years, by the progress of pathological anatomy and physiological analysis. The few observers who have recorded cases of this disease have given it different names, the chief of which are as follows :

Acute anterior spinal paralysis (Duchenne).

Subacute general anterior spinal paralysis (Duchenne).

Spinal paralysis of adults (Meyer, Charcot, Gombault).

Myelitis of the anterior horns (Dujardin-Beaumetz).

Acute spinal paralysis of adults (Petitfils).

An affection of adults like infantile spinal paralysis (M. Bernhardt).

I prefer the names of acute, subacute, chronic spinal paralysis of adults ; or, inflammation of the motor tract of the spinal cord. I have adopted the latter name for reasons which will be fully given when I come to discuss the pathological anatomy of the disease. The first name is a semeiological denomination, the second a pathological one ; both being useful in the early stage of our knowledge of a disease, the second being the one which should remain as a part of nosology.

Before relating the cases upon which this paper is based, it were well to give a short historical sketch of the subject.

Duchenne¹ (de Boulogne), in the last edition of his work on electrization, claims that he recognized the subacute form of this affection as early as 1847; and that in 1853 he gave to the profession the first sketch of its symptoms. Concerning the acute form, he writes (p. 437) that he "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 that 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,² in his work on medical electricity, describes spinal palsy in the adult, and recognizes its analogy with infantile spinal paralysis, giving two interesting cases. As early as 1863, Prof. Charcot,³ 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, Charcot⁴ says: "With reference to the *spinal paralysis of adults*, and the *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.

¹ Duchenne, *De l'Électrisation localisée*, 3me édition, Paris, 1872, pp. 437, 438, 459-461.

² Moritz Meyer, *Electricity in its Relations to Practical Medicine*. By Dr. Hammond. New York, 1869, pp. 229-242. (German third edition, 1868.)

³ Charcot's private notes cited by Petitfils, *Considérations sur l'atrophie aiguë des cellules motrices*. Paris, 1873, pp. 72-77.

⁴ Charcot, *Leçons sur les maladies du système nerveux*, Paris, 1872-'73, p. 63.

The spinal paralysis of adults recalls that of children by the almost sudden accession of palsy, by the tendency to retrocession, which is shown 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 this 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. Upon this excellent observation our view of the pathology of these cases rests. Charcot's cases and Meyer's were republished in 1873, in a thesis by Petitfils.³ During the present year the only contribution to the literature of the subject has been an article by M. Bernhardt,⁴ of Berlin, based upon one case without autopsy. In this country the subject is mentioned in an appreciative way by Dr. Mary Putnam Jacobi⁵ in a paper, remarkable for erudition, upon paralysis in childhood, published this year.

For my own part, I can say that in November, 1871, I clearly recognized the affection as illustrated in Case XVIII. This girl, with her wasted left leg, I often pointed out to the resident staff and to visitors as an example of infantile spinal palsy in the adult. Last spring I had occasion to lecture clinically upon a man affected with this disease, and then pointed out to the class the nosological place of acute and subacute inflammation of the anterior motor tract of the spinal cord.

¹ Dujardin-Beaumetz, *De la myélite aiguë*. Paris, 1872, p. 51.

² Gombault, Note sur un cas de paralysie spinale de l'adulte suivi d'autopsie. *Archives de physiologie normale et pathologique*, 1873, pp. 80-87.

³ Petitfils, *op. cit.*, pp. 71-83.

⁴ M. Bernhardt, Ueber eine der spinalen Kinderlähmung ähnliche Affection Erwachsener. *Arch. f. Psychiatrie u. Nervenkrank.*, 1874, p. 370.

⁵ Pathology of Infantile Paralysis. *American Journal of Obstetrics and Diseases of Children*, May, 1864, p. 21. [Read before the New York County Medical Society, December 22, 1873.]

The notes of this patient's condition have been mislaid, and his case is therefore omitted from the essay.

I shall first give abstracts of the few cases of acute and sub-acute spinal palsy which I have found recorded; giving full details only of Gombault's case with autopsy. I shall then relate my own cases, and afterward consider the semeiology of the disease—its diagnosis, its pathological anatomy, its etiology, its prognosis, and its treatment:

DUCHENNE'S Cases, under title of *Acute Anterior Spinal Palsy*.

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

CASE II. (Obs. LXX.)—Male, aged forty-two. In 1848, an effort accompanied by cracking sensation in back, and followed by severe pain, extending into the limbs, with numbness in, and, later, palsy of both lower extremities. Paraplegia cured spontaneously, in two months. In 1869, without cause, fever and general paralysis. Rapid wasting of forearms and hands. Wasting of some muscles of lower limbs. Improvement in four months. When seen by Duchenne, showed atrophy of hands, of extensors of wrists, and of anterior tibial muscles. At no time any bladder or sphincter ani palsy.

CASE III. (Obs. LXXI.)—Male, aged twenty-one. Lay on snow while naked, and fainted away. Then 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 right-leg muscles, and wasting of nearly all muscles of left leg.

Upper extremities exhibit wasted deltoid and hand-muscles on right side, and serratus magnus on left side. Sensibility a little dull in hands and feet.

CASE IV. (Obs. LXXV.)—Female, aged eighteen. Hard work out-of-doors. Fever lasting eight days, with pain in cervical spine and limbs, and numbness in latter. Palsy of both arms and of legs, with preservation of sensibility; bladder and intestines normal. Residue, atrophic palsy of right upper extremity. Improvement in palsy spontaneous, in second month.

DUCHENNE'S Cases, under title of *Subacute General Anterior Spinal Paralysis*.

CASE V. (Obs. LXXXI.)—Male, aged fifty-five. Gradual palsy of lower limbs, with wasting of muscles; no affection of bladder. In a year, extension of atrophic palsy to upper limbs and trunk; weakness of masticatory muscles, and slow speech. Slight anæsthesia in lower limbs; none elsewhere. Autopsy showed nervous centres healthy to naked eye.

CASE VI. (Obs. LXXXII.)—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 in trunk and limbs; much wasting of muscles of limbs. Sensibility and bladder functions unaffected.

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

CASES VIII. and IX. 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. These, having run an apparently favorable course, were followed, in both, with a paralysis of the legs, inducing a constantly-increasing emaciation of those

parts." He then states that he saw them six years later, and minutely describes the wasting of many muscles of the lower limbs, and the consequent deformities. He adds, "The sensibility of the skin and muscles was perfectly preserved." There was reduction of electro-muscular contractility in the wasted muscles.

CASE X.—Dr. W. A. Hammond,¹ under the head of progressive muscular atrophy, relates the following case, which I take to have been one of subacute spinal paralysis: An adult male had a first illness, characterized by electric pains in the legs, weakness, and head-symptoms (confusion of ideas, vertigo, dim vision, headache, etc.). There was also numbness of both upper and lower extremities. He partially recovered; but in May, 1867, there was a return of the head-symptoms, the electric pains, and numbness; to which were superadded cramps, fibrillary contractions in both hands and legs, with tingling and twitching. In the course of three weeks he was obliged to use crutches. From this time he noticed the atrophy of the muscles of both legs, and it has gradually extended till it has involved the muscles of the lower third of both thighs. In the legs, the gastrocnemii and solei and the extensors are almost destroyed. Condition of sensibility not stated.

CHARCOT'S Cases; first published in "*Considérations sur l'atrophie aiguë des cellules motrices.*" Par Alfred Petrifils. Paris, 1873, p. 72, *et seq.*

CASE XI.—Male, aged twenty-seven (seen by M. Charcot in 1863). In 1859 paralysis appeared, preceded by pain in left side; weakness of left lower limb, without numbness; in twenty-four hours complete paraplegia and anæsthesia. No numbness or spasm; no affection of sphincter ani or bladder. In next month wasting of some palsied muscles, and lowering of temperature in parts. Later, some patches of anæsthesia, in legs, sides, right axilla. In three months great improve-

¹ *A Treatise on Diseases of the Nervous System.* New York, 1871, p. 656.

ment (moxas to spine, Kl. internally). When examined showed atrophy of left natis, leg, and foot; of left lower abdominal muscles; of right thigh, anterior part. Atrophied muscles have lost electro-muscular contractility. Skin over wasted muscles colder than elsewhere. Charcot recognized similarity to infantile palsy.

CASE XII.—Male (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 parts of thighs are wasted and flabby, and have lost electro-muscular reaction. Never any sphincter ani or bladder palsy.

CASE XIII.—Male, aged thirty-five (seen by Charcot, October, 1871). In the month of August preceding, this man suffered from *malaise* for four days; on fifth day, right arm feeble and tremulous. Later, on same day, right arm paralyzed; then left leg, right leg, and left arm. Accompanying fever, with delirium, lasting one week. At consultation, there is noted wasting of right arm and left leg; no anæsthesia; formication from time to time. Never any rectal or vesical symptoms. No bed-sore.

CASE XIV.—By Prof. Cuming, of Belfast.¹ Male, aged forty. 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 vesical or rectal sphincter palsy.

CASE XV. By Gombault.—Female, aged sixty-seven. On 1st of January, 1867 (sixty-two years of age), in one day experienced great numbness, followed by palsy of all limbs. Sensibility normal; respiration, deglutition, and brain-functions not impaired; no bed-sore, no palsy of bladder or of sphincter

¹ *Dublin Quarterly Journal of Medicine*, 1869, p. 471.

ani. Pain in back at beginning, and for months afterward. Was brought to hospital in fifteen days; had sensibility, but was completely palsied. No contracture of palsied limbs. After two years began to improve gradually; and in three and a half years was able to walk a little with help of a stick. Upper limbs improved first. Examination in 1872 shows slight wasting of arms and forearms, much atrophy of hand-muscles, 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 forearm; 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.

CASE XVI. By M. Bernhardt.—Male, aged thirty-five. Exposure to cold while perspiring, after exhausting diarrhoeal 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 to the induced current, and much muscular atrophy ensued. Yet sensibility remained undiminished, the bladder and sphincter ani were not paralyzed, no bed-sore appeared. The respiratory muscles escaped. Reflex movements 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 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 XVII. (Personal).—Male, aged twenty-four. Seen February 16, 1871. In early life was a healthy and temperate boy; never had syphilis. In 1865 was run away with on horse-

“I have just been informed by Dr. Hammond that Cases X. and XVII. are identical, he having seen the patient two days after me. The case is reported in full by Dr. Hammond in his Clinical Lectures, p. 147.”

[E. C. S., *January 4, 1875.*]

back, striking a tree with his left arm and side of head. Fell from his horse and remained unconscious several minutes. Had no subsequent head-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 body, no loss of feeling, no bladder or rectal sphincter palsy, no spasmodic movements. All the limbs tingled and felt numb and were absolutely paralyzed. He had pain in the back, in its lower part, and between the shoulders. Head was always clear, and the special senses normal. Took strychnine 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 summer of same year walked with help, and has since progressively improved. No bed-sore at any time. Examination shows nothing abnormal in any part above 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 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 the sexual power was abolished; is now normal. This patient returned to his home in North Carolina, not being heard of since.

My notes indicate that I looked upon the case as one of congestion of the spinal cord.

CASE XVIII. (Personal).—Female, unmarried, aged twenty years. Admitted to the Epileptic and Paralytic Hospital, Blackwell's Island, service of Dr. E. C. Seguin, November, 1871. 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 affected. The patient cannot state how long a time elapsed between the first symptom 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, no injury.

Examination: Left foot is drawn up 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 the 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 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 palsy.

The subsequent history need not be reported. No treatment did any good; the girl remained in the hospital without any active symptom, and went away October 3, 1873, carrying this wasted left leg. She was 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 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, this numbness extending above the knee. She is positive that on the fourth day the right leg was completely paralyzed. No symptoms in left leg. No bed-sore, and no affection of bladder or rectum. Re-admitted to the Epileptic and Paralytic Hospital, March 3, 1874, with atrophy and palsy of both legs; no acute symptoms.

During the spring and summer this patient rather gradually lost strength in the thighs, in the right most. 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 abundant and in clots; there were also pains in the back and lower abdomen. On many days in this period the urine had to be drawn off with the catheter, and it often was 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. Since the middle of September has not required the catheter, and, with exception of palsy, has been better.

Re-examined October 25, 1874. Patient, when she first came in this year, walked ill with a crutch and stick; is now able to walk with two sticks (result of education). Cannot stand or walk without help. The 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 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 palsy, 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 knee-joint is weak. The quadriceps extensor femoris is wholly paralyzed; the flexors of the thigh upon the body act feebly; the adductors fairly. 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 movement in the palsied parts. The electro-muscular reaction of the atrophied muscles of both limbs is lost (both currents). At present, 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 “	21.5
“ forearms	25.0

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

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

The patient having been in bed some time, well covered up, has a thermometer held between the great and second toes of each foot for three minutes, with results: Right side, 84.25° Fahr.; left side, 86° Fahr.

CASE XIX. (Personal). Male, aged about forty, American, a bar-tender by occupation. Seen at South Shaftsbury, Vermont, with Drs. Rogers and Morgan, January 3, 1874.

In September, 1873, caught a severe “cold,” had pain in 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; the swelling in face lasted three to four weeks. At the end of first week he experienced coldness in 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; was up each day. Relieved by strychnia; about October 16th 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 October 23d; the feet were then weaker than ever, and he could barely walk up a slight hill. After the 24th, the vomiting moderated, and he improved generally. His feet were cold, stiff, but not numb. His brother-in-law, Dr. Rogers, thinks that patient then had a degree of anæsthesia in feet; he could still walk without a stick. The legs rapidly became weaker, so that in two weeks he could barely stand. November 6th, patient ceased going down-stairs, and a week later, legs were insensible to all impressions but severe pain; limbs lost in bed. There was complete palsy of parts below ankle (leg-muscles paralyzed); thigh-movements good; no reflex movements, or contracture; no bladder or rectum disorder. Suffered much from a painful throbbing in soles of feet, most in right foot. Had slight fever; about November 15th 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 nerve. The hands and forearms wasted rapidly. There was emaciation (almost wasting) of thighs and arms. December 8th, a degree of sensibility was discovered in feet, and in the fingers a week earlier. Since, there had been gradual improvement in voluntary movement and sensibility. The wasting has, however, continued to increase until now. On December 21st, 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 head-symptoms.

Examination: Nothing objective about head. The movements at shoulders and elbows are good. Patient grasps three

kilogrammes with right hand, five kilogrammes with the left. The fingers appear normal, except that the second and third phalanges of forefingers cannot be extended. Opposes thumb fairly well, though he cannot make O with thumb and index. Interossei, though much wasted, still act. Thenar muscles (especially the opponens) are much atrophied. No deformity while at rest. Co-ordinates fairly well. *Æsthesiometer* shows anæsthesia; the points being distinguished at distances of from eight to fourteen mm. on tips of fingers in median distribution, and at five to eight mm. on the parts supplied by the ulnar nerve.

The toes are in forced flexion. As he 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 c. in circumference, the left calf 24.5 c. The skin below ankles is decidedly anæsthetic, slight contact being hardly perceived; the feet and legs are hyperalgesic, there being at the same place 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 first attack of vomiting had no sweating except a very little about head, until sensibility began to return nearly four weeks ago, when perspiration appeared everywhere. During illness, absence of erection, until very lately. Throbbing pain in balls of feet has nearly ceased. The muscles of legs respond faintly to faradism, with exception of extensor proprius pollicis, which does not contract; all the hand-muscles respond, those of the thenar eminences poorly. With galvanism all these muscles contract under twelve or twenty elements, current interrupted. The left leg (less palsied) shows contraction with less faradic and more galvanic current; while the opposite is true of the more palsied right leg. The optic disks are a little hazy, especially the left. The patient 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 5 A. M. to midnight. The urine is normal. Diagnosis: inflammation of the anterior horns of the spinal cord. Advise tonics, galvanic current now to muscles, to be followed by faradism in a few weeks.

On February 28th, Dr. Rogers wrote me that "he has 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 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 have since learned that this patient wholly recovered.

CASE XX. (Personal).—Male, aged forty-seven, American inventor, seen March 23, 1874. A very large, healthy man, who has worked hard at scientific problems. In last two or three years hæmorrhages from lower bowel, and various dysæsthesiæ about the head. These consisted in pain in upper neck and back of head, in a sense of pressure on top of and behind head, etc., without impairment of intellect.

In October, 1873, had a severe cold, which was followed by much coughing. The time of beginning of symptoms of paralytic affection is difficult to determine with accuracy, because numbness and paresis crept upon him so gradually. A few days before November 10th he had complained of a sense of coldness (not objective, according to both patient and wife) in feet and legs. The numbness began in that week, appearing at about same time in ends of upper and lower extremities. On this 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 November 13th he returned, having more paresis, and remained at home. At this early date there was swelling of the feet. On November 14th the legs and forearms were quite weak; the legs swollen and glossy; the seat of subjective cold. November 17th, could not leave bed, or sit up in it. There was much formication extending up to the knees, and half-way up forearms. No

head or eye symptoms; no affection of bladder or of rectal sphincter. The feet were anæsthetic (?), though legs were not lost in bed. 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 constriction band extending over lower ribs of right side, and a sense of tightness just above line of numbness in lower part of thighs. He was dry-cupped and leeched over spine. January, 1874: About middle of this month more intense formication ushered in recovery. After a few days, sensibility returned in part of feet, and 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 extremity of limbs; feet remained a little swollen. Since that time 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 parts below the knees. The subjective coldness continued until the numbness had almost ceased. In second month of illness, and from time to time since, had numbness in distribution of second branch of left fifth nerve on face. The head symptoms, which together with irritability had annoyed him so long, disappeared during illness. Examination: patient's general condition fairly good. Walks with a cane, without dragging or jerking leg; 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 good; no atrophy, only general wasting. Sensibility of hands to contact and pain good. Grasp equals twenty-six kilogrammes with right hand, sixteen kilogrammes with left. A thermometer placed for three minutes in the fold between

thumb and palm shows 961° Fahr. in right hand, and 97° Fahr. in left. Co-ordinates well.

Lower extremities: all movements possible except that the left foot is but slightly movable; the left toes can hardly be extended; those of 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. Slight retardation of impressions of pain on toes. Toes are seat of numbness. The soles are sensitive. Can stand 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 muscles of calves. These various muscles contract well under the galvanic current.

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

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

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

CASE XXI. (Personal).—Male, aged twenty-three, single; American. Seen at Worcester, Mass., with Dr. Francis, June 29, 1874. Was a healthy, strong, and sober youth. In November, 1871, went West, to Cincinnati, where he committed sexual excesses, though without contracting venereal disease. He then went out to Missouri, where he was employed on railways, as division superintendent, clerk, etc., being much exposed to the elements. In midwinter, "about two months after reaching Missouri," patient had an attack, called, by physicians, "choreic palsy;" all his limbs being

paretic, and he walking like a drunken man. He had numbness in all his limbs; there was no affection of rectal sphincter or bladder; he could sit up, and walk with help; was cured of all but numbness and weakness of left hand, by chloride of arsenic (?), in the course of two or three months (this being early summer of 1872). About the end of this period of convalescence, he had a week of sickness, in bed, from malarial intermittent fever.

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

He returned East, very much paralyzed in all limbs, in May, 1873, the disease having made rapid progress in the preceding three weeks. When received at home, about the last of May, his right extremities were quite powerless—those on the left side could still be used, he walking, though very imperfectly, when supported. He could sit up on the edge of the bed, or in a chair. He then talked and swallowed well; had no strabismus or impairment of sight. He had the sensation of a band around body at umbilicus (which feeling, afterward, extended higher up). There was much numbness in all extremities; anæsthesia doubtful. There was slight muscular atrophy. No sign of spinal epilepsy (jerking or stiffness of palsied parts). Constipation quite obstinate; no bladder-palsy, though he was forced to empty the viscus quickly after 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 doubtful paresis of 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 29th.—In the last two months 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 the hands and feet. The difficulty of deglutition has been with solids; no regurgitation of fluids through the nose having occurred. Has been anaesthetic (?) to pinching in feet and legs at times. 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 chest and belly. Has of late passed urine and faeces 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 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 above bed. Eyes are moved imperfectly, and in a quasi-ataxic way, in every direction, except that prohibited by the left third-nerve palsy. There is no ptosis, but the pupil is wide. 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—uttering only a low grunt. The tongue can be protruded only to teeth; it lies undeviated in the floor of the mouth, atrophied and much shriveled (folds longitudinal) in anterior half. The intercostal muscles and the diaphragm act well; respirations fairly deep—twenty-four per minute. Patient has escaped bronchitis.

The state of sensibility is difficult to examine, because

patient can make no sign. 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 wasted. In feet, there is partial *pes equinus*, with toes flexed. No ulceration has appeared anywhere. The atrophied limbs are cool (thermometer showing 89° Fahr. in 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 cord; the motor part being involved from the third cerebral nerve downward, with probably recent extension of myelitis, to deeper parts of cord at some points.

October 20th.—A letter from Dr. Francis states that patient is in substantially the same condition as in June; the only change being some dementia.

CASE XXII.—In the practice of 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; never any secondary symptoms. Has beginning pulmonary phthisis. From August 27th to date has had more or less subacute articular rheumatism; knees and ankles most involved.

Ten days ago (October 13th) first noticed numbness in feet, gradually extending up to knees. Has some cramp-like pains in legs. Two days after (21st) observed that the tips of all fingers were numb; those supplied by the median nerve most. First paresis 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 discovers that the extensor muscles of fingers are weak, the left middle finger dropping much below the others during extension. Has had no pain in back; no vesical or rectal symptom. He complains much of coldness of legs.

Examination shows patient to be a nervous, rather delicate

subject, with so much lung-disease as to give a nearly uniform daily fever of 1.5° Fahr. 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 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 left 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 on the left side especially. Sensibility is not impaired in fingers. In the lower extremities, below the middle of the legs, there is marked anæsthesia to simple contact and to æsthesiometer-test, with some errors in localization of impressions. Pain and temperature are perceived everywhere, though the former kind of impressions is perceived after a retardation of several seconds, and the impressions 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 few days. There have been no reflex movements (it is difficult to provoke any now); no affection of the bladder or sphincter ani; no threatening of bed-sore; no cerebral symptoms; no weakness of chest and abdominal muscles. The patient's legs are cold, objectively and subjectively. He has had 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 free doses.

On November 1st I again saw the patient. He has in some respects improved. The anæsthesia of the lower limbs has decreased, there being 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 there is much tactile anæsthesia. General condition rather better. No affection of inspiratory or expiratory muscles; no bladder or rectum weakness; no bed-sore. Advised to dry cup the back and continue Squibb's ergot in doses of \mathfrak{z} ss a day, with quinia sulph. gr. v, in the morning; nutritious food, cod-liver oil and stimulants.¹

Semeiology.—I shall divide my remarks upon the symptoms recorded in the above histories into two categories: 1. I shall study the symptoms individually, or analytically; and, 2. shall attempt to decide whether the symptom-grouping is such as to justify us in admitting the name acute and subacute spinal paralysis into our nomenclature of nervous diseases.

The various symptoms noted in the above cases are naturally divisible into three groups, viz., disorders of motility, disorders of sensibility, and disorders of nutrition.

A. Disorders of Motility.—These have consisted in paresis and akinesia. In some of the Cases (I., II., III., IV., VIII., IX., XI., XII., XIII., XIV., XV., XVI., XVIII.), the loss of voluntary motion appeared well marked in an acute way; that is, within twenty-four or seventy-two hours after the first symptoms. In others (V., VI., VII., X., XVII., XIX., XX., XXI.), the palsy crept gradually upon the patient; in some of these cases so slowly as almost to deserve the designation of chronic (Cases X. and XVII.). In fact, a study of the twenty-two cases will show that every degree in rapidity of development of paralysis may be observed, between an almost sudden onset (Gombault's case, XV.), to a very gradual one (personal observation, XVII.).

¹ On December 9th, I saw this patient with Dr. McBride. Sensibility is perfectly restored, except on radial border of left index-finger, where slight anæsthesia remains. All voluntary movements are now possible. The muscles are firmer; patient has gained flesh; calves measure 29.25 c. This patient was severely dry cupped, and took for a while \mathfrak{z} j Squibb's ext. ergotæ fl. with belladonna. No advance in the pulmonary disease.

The distribution of the paresis and akinesis has been either general or paraplegic. Cases I., II., III., IV., V., VI., VII., XII., XIV., XV., XVI., XVII., XIX., XX., XXI., XXII., were instances of general paralysis, i. e., paralysis of all four extremities. Cases VIII., IX., X., XI., XIII., XVIII., were paraplegiform, i. e., the palsy affected one or both lower extremities. Case XXI. (personal) stands alone as an example of paralysis having involved that part of the motor spinal tract which is intracranial. In Cases V. and VI. the organs of deglutition and speech were partially paralyzed. The akinesis, as a rule, affected whole groups of muscles, and involved parts which are symmetrical and homologous. That is to say, the anterior muscles of the legs and the extensor group of the forearms were paralyzed together in several cases. Except in two or three instances the palsy appears to have been almost equal on each side of the body.

A striking feature to be noticed in nearly all the histories is the retrocession of paralysis, and the spontaneous return of voluntary movements in certain parts.

A remarkable immunity to palsy is shown by certain muscles; those of the neck, back, chest, abdomen, and the sphincter ani. The bladder was not paralyzed in any case (possible exception in Case XXI. and in Case XVIII., where temporary retention occurred). This limitation of the akinesis to muscles concerned in the acts of relation will prove a most important element in diagnosis.

Hyperkinesis, or spasm, was absent in almost every case. Late in the course of Case XXI., spinal epilepsy (tonico-clonic spasm of palsied parts) showed itself, and in Cases XXII. (actually under observation) X., and XV., fibrillary contractions were observed in the palsied muscles.

B. Disorders of Sensibility.—The general statement that sensibility was not permanently impaired in these cases is, I think, fair. In more than half of them no anæsthesia existed at any time (I., II., III., IV., VI., VIII., IX., XII., XIII., XIV., XV., XVI., XVII., XVIII.); in the remaining seven cases a moderate degree of loss of tactile sensibility existed, a

temporary defect in most of them. It was only in Cases XI. and XIX. that total anæsthesia of a limb, or large part of a limb, was noted; and that was temporary.

Hyperæsthesia of the palsied parts in the shape of tenderness to pressure was noted only in Cases XX. and XXII.

Dysæsthesiæ, or morbid sensations, were abundant; consisting in prickings and numbness in Cases I., II., IV., VI., X., XIII., XIV., XV., XVII., XVIII., XIX., XX., XXI. This numbness was in nearly every instance an early symptom, in many cases the earliest symptom. Another variety of dysæsthesia experienced by five patients early in the course of the disease was subjective cold in the palsied (or to be palsied) parts (Cases XVIII., XIX., XX., XXII.). The morbid sensation called constricting-band feeling, or cincture feeling, was observed only in Cases XX., XXI., and XXII., over the lower part of the thighs in the first case, around the waist (late in the disease) in the second, and over knees in the third. Pain in the limbs to be paralyzed was present at the beginning of the disease in Cases I., II., IV., X., XIV., XVI., XVII., XVIII., XIX., XXII. This pain was sometimes severe, "cramp-like" in the language of one patient; in another patient, it was a tearing pain in the soles of the feet; in another (X.) it was an "electric" pain. Spontaneous pain in the back was noted in five cases (I., II., IV., XVII., XX.), but was great only in the first. No marked tenderness of spinal region was observed in any case.

C. Disorders of Nutrition were only of one order, viz., muscular atrophy. This atrophy was developed more or less rapidly and extensively in different cases, but was recognizable in all. It affected whole muscles, or whole groups of muscles, differing in this respect from the irregular wasting called progressive muscular atrophy. In Case XXI. the tongue was much atrophied, and the facial muscles slightly. Fibrillary contractions were seen to accompany this wasting in Cases X., XV., and XXII.

Together with muscular atrophy, there occurred loss of

electro-muscular reaction to the electrical (faradic) current. This was observed very early in several cases, and was well marked even where the atrophy was but slight (Cases XX. and XXII.). In Case XVIII. the atrophied muscles did not contract even under strong galvanic interruptions. In all my own cases (XVII., XVIII., XIX., XX., XXI., XXII.) the paralyzed and wasted parts were much colder (objectively) than the healthy parts. The reduction in temperature was not as great as in the cases of infantile spinal paralysis. In the case represented in the photograph the lower limbs are very cold to the touch, and look purplish. No bed-sore occurred in any case, nor were lesions of the skin,¹ nails, or hair, noted.

Visceral complications were observed in some patients, and these may have been nutritive disorders secondary to the spinal disease. In Case XVIII., cystitis, pyelitis, and amenorrhœa, were at one time present; in Case XIX., vomiting, bronchitis, suppression of urine, and swelling of the feet without Bright's disease, were noted; in Case XX. there was œdema of the feet, legs, and hands, without albuminuria.

Fever was recorded as present at the onset in Cases I., III., IV., XIII., XIX. Cases VIII. and IX. issued out of rubeola.

From the above analysis it appears that the symptoms of spinal paralysis of the adult (acute and subacute) are remarkably constant, and we may safely give the following grouping as characteristic: Dysæsthesiæ and slight temporary anæsthesia, paresis and akinesia, both these symptoms affecting the extremities, and in rare cases the face, eyes, tongue, and throat; not affecting the respiratory muscles, nor those of the back and abdomen, nor the bladder, nor the sphincter ani. Muscular atrophy in the paralyzed parts. Loss of electro-muscular contractility (to faradic current) in

¹ In Case XXII., after my second visit (third week of palsy) there appeared marked vaso-motor paresis, as shown by the occurrence of red patches wherever pressure was made, on legs, knees, and malleoli, arms, and back; the redness lasting some time.

the atrophied muscles. A strong tendency to spontaneous retrocession of the palsy, and to spontaneous cure.

The important negative characters of this affection are: Absence of palsy of bladder, or of sphincter ani, or of respiratory muscles. No bed-sores. No great and extensive anæsthesia. No spinal epilepsy.

The course of the affection may be acute, subacute, or chronic; the majority of the recorded cases being of the first variety. In spite of this element of time, the symptom-grouping remains remarkably uniform in all varieties. In one of my own cases (XVIII.) the left leg was gradually paralyzed in 1871, and in 1873 the right leg was powerless after an illness of four days, and probably was so earlier. In Case XV. (Gombault's) the general paralysis was fully developed in one day. In my own case (XVI.) the patient's legs became weak slowly during three months' time.

Diagnosis.—These remarks upon the symptoms and course of spinal paralysis of the adult naturally lead to the consideration of its diagnosis, both positive and differential.

The positive diagnosis is to be made, and that in nearly all cases with ease, from the presence of the following symptoms, arranged in order of importance: Paresis or akinesis, affecting the muscles of relational life; extremities, tongue, face, or even eyes. Atrophy of, and loss of electro-muscular contractility (faradic) in the palsied muscles. Also from the absence of extensive or permanent anæsthesia, spinal epilepsy, bed-sores, bladder or sphincter ani palsy.

The problem in differential diagnosis will be different in cases in which the above capital symptoms appear in an (*a*) acute, (*b*) subacute, and (*c*) chronic way.

(*a*) *Acute Spinal Paralysis.*—This resembles infantile spinal paralysis in the most wonderful way; the symptoms of general systematic disturbance being much more marked in the young child (fever, delirium, convulsions). In each class of subjects the akinesis is developed in one or three days, it bears the same characters (with early loss of electro-muscular reaction), and affects the same muscles. Five adult cases

had fever at the beginning (I., III.(?), IV., XII., XVIII.). When speaking of pathological anatomy I expect to convince you that the diseases are identical. The acute spinal palsy of adults, when developed in one day, may bear a resemblance to hæmatomyelia (hæmorrhage into the spinal cord), to softening of the spinal cord, or to central myelitis (localized or diffused). From hæmatomyelia the diagnosis is to be made by the absence of great anæsthesia, and the escape of the bladder and sphincter ani from palsy. Besides, hæmatomyelia produces symptoms almost or quite *suddenly*, whereas the symptoms of the acute form of myelitis we are studying appear in a *rapid* way. Softening of the spinal cord and central myelitis (with limited or diffused lesion) give us anæsthesia, bladder-palsy, spasms in the paralyzed parts (spinal epilepsy) even at a quite early stage. In both these affections it is seldom that, as is the rule in spinal paralysis, the paralysis involves all the limbs in a short space of time; and when the palsy is thus general, in all three of these affections, the intercostal and the abdominal muscles are paralyzed.

(b.) *Subacute Spinal Paralysis*.—For a few days the physician may well be in doubt as to whether he has to do with the above disease, or with spinal congestion. In both affections there is peripheral numbness, with gradually-developing palsy; no anæsthesia, no tendency to bed-sore, no palsy of the bladder, or of sphincter ani. In congestion the respiratory muscles do not escape as completely as in spinal palsy. In a few days, however, an important element in diagnosis appears in the shape of loss of electro-muscular contractility (to faradism) in the weakest muscles of the patient with spinal paralysis. The atrophy soon following makes the differential diagnosis sure. There is an affection running its course in ten to twenty days, characterized by symptoms almost identical with those of subacute spinal palsy. There is an akinesia, without much anæsthesia, first appearing in the feet and legs, then ascending and involving the entire trunk and limbs, producing in nearly all cases death by asphyxia. It is upon this palsy of the respiratory muscles that the diagnosis of this

most fatal disease, acute ascending paralysis, is to be made from spinal paralysis. In some of the more recently-observed cases of ascending paralysis, the muscles were found to have lost their electro-muscular reaction at a very early day. At any rate, the relationship between the two diseases is very close; and of this I shall give some additional proof in the section on pathological anatomy. I should add that acute ascending paralysis is sometimes better named acute *descending* paralysis, the palsy first appearing in the arms.¹ The absence of anæsthesia, of bed-sores, of vesical and rectal palsy, of spasmodic movements in the paralyzed parts, will serve to distinguish spinal paralysis from subacute localized myelitis, and from the effects of tumors upon the spinal cord. In the last-named forms of paralysis the muscular irritability is retained or exaggerated.

Cases of subacute spinal paralysis, which develop so slowly as to merit the designation chronic (as Case XVI.), resemble in many ways progressive muscular atrophy: the appearance of the patient may be very deceptive indeed. The patient, whose photograph I have passed around, seems to have progressive muscular atrophy limited to the lower limbs. In true progressive muscular atrophy there are no paralytic symptoms in the strict sense of the word; the loss of power coincides with the wasting of the muscular substance; in spinal paralysis, weakness, even in the most chronic cases, is more prominent than atrophy. In progressive muscular atrophy the wasting affects portions of muscles, and never muscular groups, as in spinal palsy. The electro-muscular contractility is preserved in the muscles which are the seat of disorganization in progressive muscular atrophy, as long as any healthy muscular fibre remains; we see one half of a muscle responding to the faradic current, while the other half shows no reaction. In spinal paralysis the muscles lose their reaction to faradism in groups, and do so before much wasting is apparent. Again, progressive muscular atrophy is strangely apt to strike homologous parts: the arms and

¹ Duchenne, *op. cit.*, p. 446, note.

thighs wasting simultaneously, or the legs and forearms, or the upper thigh and hip at the same time as the shoulders. Fibrillary contractions are very often present in the wasting muscles of patients with progressive muscular atrophy; rarely in chronic and subacute spinal paralysis. Lastly, the course of true progressive muscular atrophy is very much more chronic than that of any form of spinal paralysis.

Pathological Anatomy.—The first thorough autopsy with microscopical examination of the spinal cord, in a case of spinal paralysis of the adult (acute form), was made by Gombault, in 1872, and published early in 1873.¹ If this single autopsy were made in a case belonging to a class of disease with less well-defined and constant symptoms than spinal paralysis, I should feel hesitation in attaching much importance to it. In view of the non-existence of this objection, and of the strong argument by analogy, which may be made out of abundant material collected by the best observers, I feel bound to ask you to believe, with me, that in this autopsy we have a basis for the determination of the pathological nature of the affection we have been studying under the semeiological denomination of spinal paralysis of the adult.

The autopsy of M. Gombault's patient revealed no lesion visible to the naked eye in the brain or spinal cord.

Histological Study.—The atrophied muscles were found to be in the various stages of granulo-fatty degeneration.

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 was found restricted to the ganglion cells of the anterior horns. It is worth while to quote Gombault's description of the lesion, *verbatim*:

“As regards the alteration of the ganglion cells, it exhibits the same characters as are met with in the progressive atrophy of these cells. Although the cell-degeneration is everywhere well marked, it is, nevertheless, possible to follow

¹ *Archives de physiologie normale et pathologique*, 1873, pp. 80–87.

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 cell-processes 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, in the cervical and lumbar enlargements, seems 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 anterior 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 the 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 arm showed some small patches of sclerosis—the majority of the tubules and bundles of nerves being healthy.

In brief, the lesion found in the apparently normal spinal cord of a patient having had typical acute spinal paralysis, WAS GRANULAR DEGENERATION OF THE GANGLION-CELLS OF THE ANTERIOR HORNS.

What is the lesion present in the nervous centres of pa-

tients dying while the subjects of muscular atrophy? Such is the line of inquiry to be pursued in order to test the value of the autopsy above related.

(a.) The disease accompanied by muscular atrophy and loss of electro-muscular reaction, which most resembles the spinal paralysis of adults, is that called *infantile spinal palsy*. For many years this was thought to be a congestive disease, or a myopathic disease, or a form of reflex paralysis. We owe to Charcot,¹ of Paris, and his pupils, the demonstration of the presence of a constant lesion in these cases; a lesion consisting *always* in granular degeneration of the ganglion cells of the anterior horns, and nearly always also in sclerosis (secondary) of the antero-lateral columns of the cord.

Since Prévost published his first autopsy in 1865,² every autopsy has yielded the same results in the hands of skilled observers and microscopists of France, Germany, and England.

(b.) *Progressive Muscular Atrophy*.—We owe, among many debts, our knowledge of the pathological anatomy of this disease to J. Lockhart Clarke, of London, who, in 1862³ and subsequent years, published the results of most carefully-made microscopical examinations of the spinal cord in cases of this affection. Hayem, Charcot and Joffroy, and others, have obtained the same results, viz., showing that the essential central lesion of progressive muscular atrophy consists in granular degeneration of the ganglion cells of the anterior horns of the spinal cord in the parts giving origin to the nerves going to the wasted muscles.

(c.) Again, in *labio-glosso-laryngeal paralysis*, a disease in which wasting of certain facial and intrabuccal muscles goes hand in hand with their paralysis, Charcot⁴ has discovered granular degeneration of the ganglion cells, forming the nu-

¹ See *Infantile Spinal Paralysis: a clinical lecture*, by Dr. E. C. Seguin. *The Medical Record* (New York), 1874, p. 25.

² Prévost, *Comptes-Rendus de la Société de Biologie*, 1865, p. 215.

³ *British and Foreign Medico-Chirurgical Review*, 1862, ii., 215.

⁴ Charcot, in *Archives de physiologie normale et pathologique*, 1870, pp. 247-260.

clei of origin of the hypo-glossal and facial nerves, in the floor of the medulla oblongata.

(d.) *Muscular Atrophy occurring as a complication in the course of various diseases of the central nervous system.*—It is a well-known fact that, in hemiplegia caused by a cerebral lesion, the muscles of the paralyzed side retain their irritability in a remarkable way; that they in fact usually respond to electric stimulation more readily than the muscles of the opposite healthy side (Marshall Hall's law). In exceedingly rare instances the paralyzed muscles have been known to lose their electro-muscular excitability, and to undergo rapid atrophy. M. Charcot¹ has had the opportunity of examining the spinal cord in a case of this kind, and he discovered that there was a sclerosis, with degeneration 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 white columns. Once in a while we see muscular atrophy complicating progressive locomotor ataxia. Pierret,² in 1870, examined the spinal cord of an ataxic patient, having wasted muscles, under Charcot's guidance, and discovered 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 the anterior horn, destroying the group of ganglion cells known as the external lateral group. Lastly, in the complex affection, which results in disorganization of the central part of the cord, with formation of a plug or 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

¹ Cited by H. Charlton Bastian, *Clinical Lectures on the Common Forms of Paralysis from Brain-Disease*. Lecture V., Part I.—*The Lancet*, 1874, p. 406.

² Pierret, "Sur les altérations de la substance grise de la moelle épinière dans l'ataxie locomotrice, considérées dans leurs rapports avec l'atrophie musculaire qui complique quelquefois cette affection."—*Archives de physiologie normale et pathologique*, 1870, pp. 599–617.

anæsthesia, akinesis, bed-sores, etc.¹ It appears to me that, in the several newly-discovered facts enumerated, viz., that the only lesion always present in infantile spinal paralysis, progressive muscular atrophy, labio-glosso-laryngeal palsy, and in various forms of "symptomatic atrophy," is a granular, pigmentary degeneration of ganglion cells of the anterior horns of the spinal cord, there is a justification of the generalization first made by Charcot. This most able observer and teacher announced, in a clinical lecture delivered at the Salpêtrière in June, 1868, that the relation between granular degeneration of motor nerve cells and muscular atrophy was that of cause and effect. This position Prof. Charcot has since maintained, accumulating fresh evidence year by year in its support. For my own part, I accept this view without hesitation. If we now confront this generalization that granular degeneration of motor nerve-cells produces muscular atrophy, with the bare fact revealed (p. 31) by Gombault's autopsy, that the only lesion in his case of acute spinal palsy was granular, pigmentary degeneration of some of the ganglion cells of the anterior horns of the spinal cord, we cannot avoid concluding that the spinal paralysis of adults, in any one of its forms, depends upon degeneration of anterior ganglion cells.

Whether this degeneration of the ganglion cells is the *only* lesion in spinal paralysis is a question to be solved only by future autopsies. It is well to remember that, in nearly every case of infantile spinal paralysis recently examined, a degree of myelitis (of gray and white matters) coexisted with the cell-degeneration. With respect to the great question, whether the change present in Gombault's case, pigmentary granular degeneration of ganglion cells, is an inflammatory or a degenerative (ischæmic) process: the elements for discussing this complicated problem are not yet in our hands; many autopsies, at different stages (or age) of the lesion, as shown in vari-

¹ See Schüppel, Ueber Hydromyelus, *Archiv der Heilkunde*, 1865, p. 289. Hallopeau, Étude sur les myélites chroniques diffuses, *Archives générales de médecine*, 1871, ii., pp. 277, 435, 565; 1872, i., pp. 60, 191.

ous clinical forms, will have to be made. I think it best to assume, as a temporary hypothesis, that there is parenchymatous inflammation in the spinal paralysis of adults (and of children), the results appearing chiefly or only in mal-nutrition and degeneration of the affected ganglion cells.

Consequently I have chosen the pathological name of inflammation of the motor tract of the spinal cord. I prefer it to anterior myelitis, because when the disease ascends above the decussation of the pyramids, it affects parts which, while constituting a part of the motor tract, lie posteriorly. The term motor (or kinesodic) tract I prefer to that of anterior horns of the spinal cord, because it applies to the upper part of the spinal axis, that part which lies in the parts called medulla oblongata, pons Varolii, and crura cerebri by anatomists. The true spinal cord, with a well-developed motor tract, extends, I believe, as far as the origin of the third cerebral nerve inclusive; and it has been shown, in Case XXI., that the symptoms of spinal paralysis may indicate disease of the whole of this physiological spinal cord.

Etiology.—The only predisposing causes, or predispositions, which are very evident on reading the cases related in the first part of this essay, are age and sex. All the patients were adults, nearly all of them of middle age. The greatest age at time of seizure was sixty-two years; the least eighteen years. I would add that the oldest child developing infantile spinal paralysis recorded in my table of cases,¹ was seven years; thus leaving a gap of eleven years of life (adolescence), in which an immunity to spinal paralysis seems to exist. As regards sex, by far the greater number of the patients (seventeen) were males. This peculiarity may be logically related to the only exciting cause which is apparent in the twenty-two cases, viz., exposure to cold, or “catching cold.” In four cases (III., XIV., XVI., XIX.), the action of cold upon the surface of the body is given as the cause of the attack by the reporters. In three other cases (IV., XV., XVII.), the efficacy

¹ *The Medical Record*, New York, January 15, 1874. Case by Charcot and Joffroy.

of refrigeration may be inferred: in the first case, the patient having worked hard out-of-doors; in the second, having been out on January 1st; in the third, having been repeatedly wet through. In Case I., an effort accompanied by strain of the muscles of the back is reported as a cause; in Cases VIII. and IX., the palsy and atrophy appeared as the boys were recovering from measles; in Case XII., dysentery seems to have preceded the palsy.

The inference that spinal paralysis in the adult is caused by refrigeration (or other irritation) of peripheral nerves, seems fair to draw. This view is supported by what little we know of the etiology of the disease as it occurs in young children (infantile spinal palsy); nearly every observer having expressed the opinion that peripheral irritation of nerves of relational or organic life (cold, worms, dentition, indigestion) determines the outbreak of spinal cord diseases.

To bring forward arguments to prove that peripheral irritation may set up organic disease in the nervous centres, that a paralysis *a frigore* (as adult spinal paralysis seems to be) may be due to a myelitis, would be to transgress the limits of this paper. I will merely refer to what two recent and authoritative writers say upon the subject.¹

Prognosis.—None of the subjects died of the paralysis, with exception (?) of Case V.; the patient whose spinal cord Gombault examined dying of an intercurrent affection while she was in the stationary stage of her spinal disease. The prognosis as regards life is therefore very good, just as in the spinal paralysis of children. The small amount of danger seems to depend upon the non-involvement of the respiratory muscles, and the continued normal action of the bladder. It is well known that the majority of paralytic patients die of asphyxia, or bronchitis, or cystitis extending upward so as to set up pyelitis. The freedom from bed-sore is also a reason for the

¹ Jaccoud, *Les paraplegies et l'ataxie du mouvement*, Paris, 1864, pp. 381-386.

E. Leyden, Ueber Reflexlähmungen. Volkmann's *Klinische Vorträge*, I. Series, No. 2, 1870.

small degree of fatality attending inflammation of the motor tract of the spinal cord. The prognosis relating to the paralysis is only moderately good. Many subjects retain one hopelessly wasted limb or more. In a few cases an almost perfect cure may be hoped for (XVI., XIX., XX., XXII.). In others the first force of the disease is so great, i. e., such a destruction of ganglion cells takes place, that not any improvement occurs in the atrophied limbs. As guides in prognosis we must rely upon the rapidity with which atrophy occurs, and upon the loss of electro-muscular reaction. Even in the most promising cases a cautious prognosis, at least one made conditional upon repeated testing of the muscles with faradism and galvanism, should be given. In Case XVIII. (subject of photograph), for example, all irritability having disappeared in the muscles of the left leg, the prognosis is absolutely bad.

Treatment.—As this essay is intended to call attention to the semeiology and pathology of spinal paralysis (inflammation of the motor tract), and as it has already grown larger than it was originally intended, I think it best to dismiss this subject in a few words. The early stage, in the acute and subacute forms, should be treated by counter-irritation to the spinal region; dry cups, actual cautery, or leeches. Ergot and belladonna should, I think, be given freely, the latter so as to produce slight throat or eye symptoms. At the same time, nutrition should be furthered and constipation prevented. Pain, if severe, may be relieved by means of liniments, or simple warm wraps. In these two forms, after the active symptoms have subsided, and in the chronic form, the treatment resolves itself into attempts to restore the nutrition and force of the muscles. This is done, as in infantile spinal paralysis, by means of the galvanic current, applied in such a way as to produce muscular contractions. When improvement takes place, the muscles reacquire the property of contracting to faradism, and this agent should then be substituted for galvanism (Case XIX.). For more details upon the subject of treatment of the remaining muscular wasting, I refer the reader to my lecture¹ upon the infantile form of the disease.

¹ *The Medical Record* (New York), January 15, 1874.

My conclusions are: There is a form of paralysis in the adult very similar to the infantile spinal paralysis of children, in its symptomatology and pathological anatomy, in its etiology and prognosis. The two affections I propose to denominate, provisionally, as follows:

Spinal paralysis or Inflammation of the motor tract of the spinal cord,	} In the adult.	{ Acute.
		{ Subacute.
	} In children.	{ Chronic.
		{ Acute.

This disease is closely related, in the nosological scale, with acute ascending paralysis on the one hand, and to progressive muscular atrophy, and labio-glosso-laryngeal paralysis, on the other.

