

INFANTILE SPINAL PARALYSIS.

A CLINICAL LECTURE,

DELIVERED AT THE COLLEGE OF PHYSICIANS AND
SURGEONS, 1873.

BY

E. C. SEGUIN, M. D.

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REPORTED BY

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INFANTILE SPINAL PARALYSIS.

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BY E. C. SEGUIN, M. D.

(Reported by FRANK P. KINNICUTT, M. D.)

GENTLEMEN: I think that you may obtain a clearer idea of the importance of infantile spinal paralysis if we study it not separately, as I had at first purposed we should, but as one of the forms of paralysis to which infants are liable. At a subsequent meeting we can briefly inquire into the other varieties of infantile palsy, and you will then obtain a knowledge of a great part of the nervous pathology of infancy, and be better prepared to diagnose the most important of these affections—infantile spinal paralysis. The period of life termed infancy is that which includes the first dentition to the beginning of the seventh year. The pathology of this stage of existence is treated of separately by authors, in part because of the peculiar difficulties in diagnosis presented by children of this age, and in part because of numerous peculiarities they exhibit, such as susceptibility to the action of causes of disease, of medicines, and the exaggeration of certain groups of symptoms, such as the spasmodic element in the sphere of the nervous system. There is no essential difference, please remember, between infants and adults in the nature, course, and treatment of disease.

The following synopsis represents the principal forms of paralysis which are likely to occur in infancy, together with their leading pathogenetic factors :

¹ On November 8, 1873.

PARALYSIS IN INFANCY.

1. Hemiplegia (clot, injury, embolism, tumor in brain).
2. Paraplegia (injury, clot, inflammation of cord).
3. Peripheral paralysis (compression, etc., of nerve trunk).
4. Inhibitory paralysis (peripheral irritation).
5. Infantile spinal paralysis (atrophy of motor nerve cells).

The last of these is the disease we shall study to-day, and several examples of it are before you, affecting subjects of different ages. The term which I have adopted is one of numerous synonyms by which the disease has been known. The first systematic writer upon the subject, Heine, used a corresponding German name, *spinale Kinderlähmung*, in his second edition of 1860. French writers upon the diseases of infancy, who studied the subject very thoroughly in its clinical aspects, Rilliet and Barthez,¹ denominated the disease *paralysie essentielle de l'enfance*. Bouchut, entertaining a very singular notion of the pathology of the affection, gives it the name of *paralysie myogénique*. Niemeyer retains the term essential paralysis, considering it as a safe one to adopt during the present unsettled state of opinion upon the question of pathology. The word *essential* is used by these authors as equivalent to functional or *sine materia*. The title infantile paralysis is one employed by such authors as C. B. Radcliffe and Adams. Duchenne, in the last edition of his work on electrization, calls the disease *paralysie atrophique de l'enfance*, instead of *paralysie atrophique graisseuse de l'enfance*, as in his edition of 1860. Hammond, in his treatise upon "Diseases of the Nervous System," speaks of organic infantile paralysis.

Symptoms and Course of the Disease.—In brief, infantile spinal paralysis may be defined as an acute febrile affection, resulting in generalized paralysis, which shortly disappears from all but a limited part of the body, where the akinesia persists indefinitely without impairment of sensibility, is ac-

¹ Rilliet et Barthez, *Maladies des enfants*, tome ii., p. 545, *et seq.* Paris, 1861.

accompanied within a few weeks by atrophy of the palsied muscles, and is followed later by various deformities—the result of altered balance of power at certain joints. The anatomical lesion of the disease consists in primary (?) atrophy of the nerve cells of the anterior horns of the spinal cord (motor tract), and in secondary (?), complicating (?) myelitis.

A. Febrile Stage.—A child, and usually a healthy or even a robust child, has an indisposition, with febrile movement, evidenced by restlessness or drowsiness, hot skin and frequent pulse. The mother's attention is often called to a coexisting local affection, such as indigestion, intestinal catarrh, or morbid dentition. The last is the usual condition referred to, because the age when infantile spinal paralysis is most apt to occur (six to twenty-four months) includes nearly the whole period of dentition, and because mothers as well as many practitioners are only too willing to hold the teeth responsible for any disease which may arise during their evolution. The fever has not, to my knowledge, been studied with the thermometer. Fever has been observed in nearly all cases¹ (forty out of fifty). When parents assert that no fever was present at the outset, it must be borne in mind that a certain degree of intelligence is required to enable a person to recognize fever; that in some cases only skilled observations (thermometrical measurement) will reveal its existence; and that it may have been present for a few hours only. Besides these phenomena, there are several nervous symptoms present in a certain proportion of cases, convulsions usually not involving the facial muscles, and delirium. Hyperæsthesia is also present; but whether this is a true hyperæsthesia, or an evidence of that morbid sensitiveness which is part of the febrile state, remains uncertain.

B. Stage of Paralysis.—As the fever subsides, the mother, upon handling the child, discovers that voluntary motion has been lost in nearly the entire body. The exact period when the akinesis appears, and whether it develops slowly or sud-

¹ Laborde, De la paralysie (dite essentielle) de l'enfance. Paris, 1864, p. 3.

denly, are points not yet investigated. The paralysis is at first generalized, often involving all the limbs, very rarely the face. At the same time no impairment of sensibility is to be noted. In a short time, a few hours, the paralysis recedes from some parts and persists in others; it being very rare that more than one-half of the body remains palsied. This retrocession of the paralysis, its disappearance from some limbs, and fixation upon one or two, or upon unsymmetrical distant muscles, is highly characteristic of the disease—I might almost say pathognomonic. This fixation is usually upon parts of the lower limbs, more seldom upon the upper, and rarely upon parts of the trunk. In only one case has a muscle of the head been found definitely paralyzed—the temporal. The muscular group which is most often affected is the anterior tibial and peroneal. Owing to the immunity of the abdominal muscles, the bladder and rectum perform their functions.

C. *Third Stage*.—Inseparably connected with the definitive akinesis is the muscular atrophy, with loss of electromuscular contractility, and with deformities. The loss of irritability in the palsied muscles, as tested by the electrical current (faradic), occurs very early—at a period varying from one to six weeks. In one case atrophy and loss of electromuscular contractility were well marked on the fourth day (Laborde)¹. A very few years ago three observers independently made the very valuable observation that these atrophied muscles could be made to contract by the application of the galvanic current. Mr. Harry Lobb,² Dr. William A. Hammond,³ and Mr. J. Netten Radcliffe⁴ (from 1863 to 1865), are each entitled to the merit of making this observation in infantile palsy. The application of this reaction to the galvanic current to prognosis will be stated farther on.

¹ Laborde, *op. cit.*, p. 19.

² In a letter to the London *Medical Times and Gazette*, 1863, vol. ii., p. 682.

³ NEW YORK MEDICAL JOURNAL, December, 1865.

⁴ Reference by C. B. Radcliffe, in Reynolds's *System of Medicine*, vol. ii., article on "Infantile Paralysis," p. 665. London, 1868.

The atrophy of the muscles is both relative and positive, and is accompanied by certain histological changes, which will be detailed when we study the morbid anatomy of the disease. In the case now before you, a young man, seventeen years old, in whom infantile palsy set in at the age of twenty months, the bones of the legs, themselves much reduced in size, seem covered only by skin, and a little adipose tissue. These legs also exhibit two accompaniments of the atrophy of infantile paralysis, viz., imperfect circulation and diminished temperature. The atrophied parts are very purple, and are quite cold to the touch. The diminution of temperature has been measured by Heine,¹ and found very great; the atrophied region in one case having a surface temperature of only 14° Réaumur (63°.5 Fahr.). This is in striking contrast to the temperature of limbs palsied by a cerebral lesion.

In consequence of this akinesis, indefinitely prolonged, accompanied by wasting of the muscular tissue, there are formed a number of *deformities*. Some of these consist merely in arrested development, as indicated by the shortening of the affected limb—there occurs a relative atrophy. This shortening of a limb, a lower limb more especially, gives rise to secondary deviations in form, such as twisting of the pelvis and lateral curvature of the spine. These secondary deviations, which are the result of attempts to restore disturbed equilibrium, are called compensatory. It is important to distinguish these compensatory deformities from the primary, or truly paralytic ones, because the treatment of either kind differs radically. The positive deformities are caused, as stated in the definition, by impairment in the balance of muscular power about any articulation. For example, in the feet of one of the patients before you, you see an example of deformity which constitutes the talipes equinovarus of surgeons; toes point downward and inward. The anterior and posterior muscles of the legs are palsied and atrophied, and consequently the feet obey the laws of gravity, dropping into their position because the ankle-joint is

¹ Heine, *Signale Kinderlähmung*. Stuttgart, 1860, p. 16.

well back upon the posterior third of the foot. There is no resistance to passive movement in all directions. In other cases only one set of tibial muscles is paralyzed, and the deformities are produced by the unchecked (unantagonized) action of the healthy muscles. In the feet we may thus have talipes equinus ("the most common and important variety," Erichsen), or talipes equino-varus, or any of the varieties which you will find fully described in surgical text-books. In a case of talipes equinus of this sort we find great resistance to any attempt at overcoming the deformity; the tendo-Achillis is very tense. The muscles governing the movements of the knee-joints may be paralyzed, and in consequence we obtain analogous deviations at that joint. About the upper extremities the deformities are less striking, but are produced by the same double mechanism, obedience of parts to gravity, and unchecked action of certain muscles. The spinal deformities are nearly always of the compensatory sort: for instance, a shortened leg necessitates the inclination of the pelvis to the side on which the shortening is; and, in order to preserve the equilibrium of the body, the lumbar vertebræ bend so as to form a concavity on the opposite side, and higher up there occurs a secondary spinal curve to the side of the shortened limb. The lateral curvature is known technically as scoliosis. This is rarely due to palsy of spinal muscles, though I have now under treatment a case of this primary paralytic scoliosis. A variety of muscular disorders may cause scoliosis in individuals not affected with infantile paralysis. (Consult the various works on deformities.) Other spinal deformities, more often primary, are the bending forward of the spinal column, non-angular, called kyphosis; and the bending backward, termed lordosis. The bending forward is shown in a minor degree (quasi-pathological) in what is termed stooping—a supposed sign of studious habits. Lordosis, as first clearly pointed out by Duchenne, may be due to palsy of the abdominal muscles, or to bilateral palsy of the lower spinal extensors; in both cases the upper part of the body is thrown unnaturally backward to preserve

equilibrium. The two forms may be recognized without a minute examination of the seat of palsy, by using a plumb-line. Dropping the line from the shoulders, it will clear the sacrum in cases of lordosis due to palsy of the erectors of the spine; whereas in palsy of the abdominal muscles it will fall within the sacrum. In the latter case there is a deepening of the normal lumbar curve without positive backward projection of the upper part of the body.¹

One word more concerning the genesis of primary deformities. It is often said that, in club-foot and other deformities, the efficient cause of the deviation is spasm of the preponderating muscles. This I believe to be extremely rare, and a belief in this erroneous doctrine leads to insufficient (merely orthopedic) treatment of the deformities.

Changes in nutrition in the paralyzed parts; many of these, as observed in the disease, consist in what may be termed relative atrophy, i. e., arrested development or retarded growth, e. g., diminution in the calibre of the blood-vessels, not only of the parts affected, but extending farther into the vessels above the atrophied muscles, and even into the main trunks. In a certain number² of cases of atrophied lower extremities, a marked diminution in calibre has been found to exist in the iliacs, and even in the lower portion of the aorta. In the boy before you, with extreme atrophy of all the muscles below both knees, the whole vascular system of the lower extremities is abnormally small. It is necessary to bear in mind that no pathological change is discoverable in the walls of these vessels; it is an excellent example of simple arrested development. Again, we find the bones of the implicated regions diminished in size, the arrested development affecting rather their circumferential than their longitudinal growth, only a slight shortening being found to exist even in cases of long standing.—Let us now devote a few moments to the study of the more positive changes in nutrition, as exhibited in the

¹ Duchenne, *De l'électrisation localisée*, 3me édition, Paris, 1872, p. 498.

² Cases by Charcot and Joffroy, *Archives de physiologie normale et pathologique*, 1870.

muscles. The morbid anatomy of the muscular system in infantile spinal paralysis has been the subject of laborious and careful investigation, and the results of the latest researches tend to convince us of the existence of the different kinds of muscular degeneration, though by no means of equally frequent occurrence, in this disease. By far the most common form is : (a.) The simple atrophy, a form which almost always affects such muscles as are kept in forced repose for a long period ; it manifests itself in a shrinking of the individual muscular fibres ; a transverse section reveals the separation of the contractile substance proper from the sarcolemma ; moreover, as a rule, we find a fatty infiltration of the cells of the connective tissue. (b.) A granular degeneration is described as succeeding in order of frequency. In this form proteinaceous granules (bodies unaffected by ether, dissolved by acetic acid) are found in the fibre itself ; the striation disappears gradually, more slowly than in the simple atrophy ; as in the latter form, a fatty infiltration of the interstitial tissue also exists. (c.) A true fatty degeneration is believed to be comparatively rare. The earlier belief, that it constituted the peculiar muscular lesion in infantile paralysis, aside from its occasional occurrence, may have arisen from the almost unexceptional co-existence of the interstitial fatty changes with the forms above described.

Pathological Anatomy.—We have now to consider the morbid anatomy of the disease itself. By the earlier writers it was believed to be essentially functional in its character. The first step toward giving us a clearer conception of the true nature of the affection was made by Heine, who published the results of his observations in 1840 ; discarding the functional theory, he expressed his belief that the disease was due to a violent congestion, with perhaps a subsequent inflammation of the nervous centres. Later, cases were published by Laborde and Cornil, in which a sclerosis of the antero-lateral columns was described. In an autopsy by Echeverria, sclerosis and amyloid degeneration of the antero-lateral columns, sclerosis of the anterior nerve roots, and

brown pigment in the nerve cells, were found. In a case of von Recklinghausen's, tubercular deposit was discovered in the substance of the cord. H. Roger and Duchenne, Jr., reported two cases in which autopsy revealed atrophy of the anterior and antero-lateral columns, diminution in size of the nerve fibres, increase of the interstitial connective tissue, and the presence of numerous amyloid bodies. In an *unrecognized* case of infantile paralysis, Mr. J. Lockhart Clarke, of London, described an atrophy, a degeneration of the cells of the anterior horns.

To Prévost (a pupil of Charcot's), therefore, is due the credit of first recognizing this cell degeneration as the true lesion in this disease. In 1866 he published the report of the autopsy in which this lesion was discovered. Since that date a number of cases have been reported by Charcot and his pupils, in which a similar cell degeneration has been found. Studied with the microscope, we find that it consists in an increase of the normal pigment of the nerve cells; the latter are observed to become densely packed with pigment granules, and, finally, to wholly lose their cell character. In their place a simple granular mass, which gradually undergoes a marked diminution in size, is seen. In a certain number of cases no granular change is discoverable, the cells seemingly being subjected to a simple wasting process.

Other morbid changes, indicating a myelitis, have been described as among the lesions of infantile paralysis, e. g., a sclerosis, an increase of the interfibrillar connective substance of the medullary columns, with a marked increase in the nuclei of that substance; the formation of cavities in the gray matter, apparently through a process of liquefaction; small clots in the same substance; corpora amylacea in both the gray¹ and white matter; and, finally, what can only be described as condensed patches of tissue in the former.

The question is yet disputed whether the granular cell

¹ The theory now gaining ground in regard to the character of these bodies is, that they are due to an amyloid degeneration of the round cells of the neuroglia.

CASES OF INFANTILE SPINAL PARALYSIS.

No.	Date.	Author.	Age at Onset.	Age at Autopsy.	Mode of Onset.	Parts paralyzed.	Lesions of the Nervous System.	Lesions of Muscles.	Bibliography.
1	1825	Hutin.	7		Convulsions.	Both legs.	Atrophy of lower end of cord, and of its nerve roots.		Cited by Heine, <i>Spinale Kinderlähmung</i> , p. 151, 1860.
2	1842	Longet.	8		Not stated.	Right leg.	Atrophy of roots of right sciatic nerve.	Not stated.	Longet, <i>Anat. et Phys. du Système Nerveux</i> , I., p. 358. Paris, 1842.
3	1849	Filoss.				Upper extremities	Congestion of meninges over cervical enlargement of cord.	Idem.	Cited by Laborde, <i>Paralyse de l'enfance</i> , p. 113. Paris, 1864.
4	1850	Rilliet et Barthez.				One arm.	None. (Microscope not used.)	Idem.	<i>Gazette Méd. de Paris</i> , 1850, p. 681.
5	1850	Idem.				Both legs.	Idem. (Idem.)	Idem.	Idem.
6	1855	Behrend.	1	5		Right lower extremity.	Chronic spinal meningitis.	Fibres small; not fatty	Cited by Heine, <i>op. cit.</i> , p. 150.
7	1853	Von Recklinghausen.				Both legs.	Tubercles in cord.		<i>Deutsche Klinik</i> , 31 Jan. 1863.
8	1863	Cornil.	2	49	Unknown.	Idem.	Amyloid degeneration and atrophy of white columns of cord.	Fibres fatty.	<i>Gazette Méd. de Paris</i> , 1864, p. 290.
9	1863	Bouvier.	1	2	Idem.	All limbs.	Sclerosis of antero-lateral columns of cord.	Fibres very small and granular; not fatty.	Cited by Duchenne fils in <i>Archives Gén.</i> 1864, II., p. 205-9.
10	1863	Henri Roger.			Idem.	Not stated.	Idem.	Not stated.	Idem.
11	1864	Laborde.	2	2	Febrile.	Both legs.	Sclerosis of antero-lateral columns of cord; cells normal.	Fibres small; not degenerated.	Laborde, <i>op. cit.</i> , 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.
13	1865	Prévost.			Unknown (infantile ?).	Left lower extremity.	Atrophy of cells of left anterior antero-lateral column.	Fibres granular; not 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, <i>On Reflex Paralysis</i> , p. 29. New York, 1866.
15	1866	Idem.	2	2½	Febrile diarrhœa.	Both legs.	Nerve cells granular; diffused myelitis; nerves shrunken.	Fibres fatty.	Idem, p. 35.

16	1867	Wm. A. Hammond.		Palsy of 4 years' standing.	Left lower extremity.	ex-	Small clot in cord.	Not stated.	Journal of Psychological Med., 1867, p. 51.
17	1868	Lockhart Clarke.	1	32 After cow-pox inoculation.	Upper extremities	ex-	Atrophy of nerve cells of anterior horns of cervical enlargement; central myelitis.	Idem.	New York Med.-Chr. Trans., vol. II., p. 249. London, 1868.
18	1870	Charcot et Joffroy.	7	40 Sudden, non-febrile.	All limbs.	ex-	Atrophy of cells of anterior horns; atrophy of antero-lateral columns; slight myelitis.	Fibres small, but not degenerated.	Archives de Phys., 1870, p. 135. Paris.
19	1870	Parrot et Joffroy.	3	Unknown.	Left lower extremity.	ex-	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.	ex-	Atrophy of cells of right anterior horn; some sclerosis of white columns.	Fibres small; striated; finely granular (fat-ty?).	Idem, p. 316.
21	1871	Roger et Damaschino.	11 ⁹	2 After dysentery.	Left upper extremity.	ex-	Atrophy of cells of left anterior horn; sclerosis of antero-lateral columns; central myelitis and softening; left anterior nerve roots small.	Fibres small, with fat-ty granules and numerous sarcolemma nuclei.	Gazette Méd., 1871, p. 480.
22	1871	Idem.	2	2½ After variola.	Both legs.	ex-	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.	ex-	Atrophy of cells of anterior horns; patches of softening in gray matter; sclerosis of antero-lateral columns.	Idem.	Idem, p. 541.
24	1873	Lancereux et Pierret.	2	18	Left arm.	ex-	Atrophy of cells of left anterior horn; atrophy and sclerosis of left half of cord.	Not stated.	Cited by Pettiflis, Atrophie aiguë des cellules motrices, p. 88. Paris, 1873.
25		Roth.	1	2 Febrile.	Both legs.	ex-	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., Hf. 2, p. 263.

NOTE.—Dr. Allbutt's case (*Lancet*, 1870, II., p. 84) I reject because of evident traumatism.

degeneration is to be regarded as the primary lesion. The fact that, in one case at least,¹ the cell lesion has been observed without any concomitant myelitis, would seem to support Prof. Charcot's view that the cell degeneration is the primary and essential lesion. This is further strengthened by the recent revelations concerning the state of the nervous centres in progressive muscular atrophy; in that disease the cells of the anterior horns being found in part or wholly destroyed by a similar pigmentary degeneration, without surrounding myelitis.

Prof. Charcot was so kind as to give me some sections of the cord from the case, now classical, which he published under his own name and Joffroy's (his *interne*) in 1870. I will pass around two microscopes armed with half-inch objectives, one showing a transverse section of a normal spinal cord, the other the section from Charcot's case. Both sections are from the mid-dorsal region of the cord, a part in which the anterior horns are small and the cells few. The normal anterior horn exhibits six or eight large well-defined multipolar ganglion cells, stained red by carmine, their nuclei standing out more deeply tinged than the body of the cell. On the other hand, careful examination of the morbid section shows nothing but a somewhat condensed gray horn tissue, without one distinct ganglion cell. The granulations which probably once existed in the place of some of the cells have been dissolved by the method (Clarke's) used in preparing the section. I would also call your attention to the fact that the diseased anterior horn is shrunken and less club-shaped than the same part in the healthy section.

The *differential diagnosis* of this from other paralytic affections of infants I leave until our next meeting, when we shall review the whole group briefly.

The *prognosis* of infantile spinal paralysis is not good for many reasons. The disease is a severe one, accompanied by a serious central lesion, and is possibly incurable in a certain proportion of cases in spite of every favoring circumstance.

¹ Case by Charcot and Joffroy.

Then usually we are consulted weeks, months, and even years after the stage of atrophy and deformity has set in, and the cure is then more questionable in proportion to the period of time which has thus elapsed. Many of the deformities can be remedied, at any age, by proper orthopedic treatment; but that is not curing the disease. Until the recent (*see supra*) discovery of the reaction of atrophied muscles we had no guide in prognosis beyond time, and the appearance of the parts. Now, we know that if any contraction can be obtained by means of the galvanic current (interrupted), there is some hope of restoring the muscles to activity. One authority (Dr. Hammond)¹ 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.

I will be very brief about the treatment. The management of the first or febrile stage is a matter of uncertainty; few practitioners see the cases in this stage, and, when they do, the diagnosis of simple fever, or of fever symptomatic of some teething or intestinal disorder, is usually made. Were I to meet with such a case, and have due reason, from occurrence of delirium, convulsions, and the presence of hyperæsthesia, to suspect impending spinal palsy, I should leech the child's spine, and apply counter-irritants to the extremities. Besides, treatment indicated by the state of the mouth or bowels should be carried out. The treatment of the second stage is likewise a matter about which no rules based on solid experience can be laid down. I should favor irritating the spine and the extremities, keeping the child's bowels free, and applying electricity, in either of the forms commonly employed, to the palsied muscles.

The management of the third stage may be divided into 1. The treatment of the central lesion and of the atrophy; and 2. The curing of the deformities.

¹ A Treatise on Diseases of the Nervous System. New York, 1871, p. 692.

1. We know no means which will with certainty remedy the central lesion which I have described as existing in this disease. Strychnia and nux-vomica were prescribed by Heine and by other older authorities, but are now abandoned. The hypodermic injection of strychnia about the wasted parts has been recommended of late, and is worthy of a trial; from $\frac{1}{80}$ to $\frac{1}{15}$ grain may be injected with safety, according to the age of the patient. Of course every thing which shall tend to improve the patient's general condition (hygiene, nutritious food, cod-liver oil, exercise) will favor the reconstruction of the atrophied nerve cells.

2. The means of treating the palsy and atrophy consisted, until a few years ago, in the (nearly always vain) application of the faradic current, cold and hot douches, and the systematic friction of the atrophied muscles. These last are valuable, especially the alternate use of ice (or cold water) and hot water to produce hyperæmia. Beyond these in value comes the use of the galvanic current, which will in many cases produce good contractions in the wasted muscles. The number of cells to be used must be determined by trial; 10-20-30 elements of Stöhrer's battery may be required to obtain a reaction. The positive electrode should be placed upon the nerve trunk supplying the atrophied group of muscles, and the negative sponge upon various muscles of the group; the current being meanwhile interrupted slowly by removing and replacing the negative electrode, or (to produce maximum irritation) reversed as well as interrupted by a mechanical contrivance on the battery or in the hand. I would suggest the use of nitrous oxide gas for the purpose of avoiding the intense pain produced by a large number of cells. Especially is anæsthesia useful in first examinations when you want to base a prognosis upon the result. An advantage offered by the use of this adjunct is, that, the child being still, you are able to recognize a small muscular contraction which might be overlooked during the struggles of the suffering patient. How difficult it is to be sure that contractions occur under these circumstances, those of you who have attended in

the electrical room of the college will remember. I am led to make a remark which I should have made when speaking of the prognosis, to the effect that a safe negative prognosis cannot in my opinion be based upon a first or second galvanic examination. If you can afford the time, and your patient the money, you should ask for a number of trials, at least six, before saying that the patient cannot be cured or improved. A great difficulty in the way of proper and successful galvanization consists in the very common stretched state of the atrophied muscles. For instance, in a palsied leg with healthy posterior tibial muscles, there exists pes equinus, and in consequence the wasted anterior tibial muscles are stretched to a great degree. Now, gentlemen, I believe that this tense condition will prevent galvanic reaction for a long time, if not indefinitely; and the relief of the tension either by mechanical contrivance (shoe), or better still by a tenotomy and a shoe, is followed by success in treatment. In illustration I would cite a private case of my own, a lady suffering from symptomatic muscular atrophy consequent upon cerebro-spinal meningitis, in whom the muscles of the legs were in much the same state as the muscles in cases of infantile spinal palsy; the posterior tibial muscles having in part recovered. The anterior tibial muscles were kept tense by a strong pes equino-varus, and for several months careful galvanization (even electro-puncture) produced no reaction. At last, as a *dernier ressort*, I asked Dr. H. B. Sands to cut the tendo-Achillis in both legs and to put the limbs in plaster of Paris. This was most dexterously done, and four days after the tenotomy distinct contractions appeared under galvanization in the atrophied muscles.

The muscles, after improving in size under the above means, gradually begin to obey the will, and also respond to the faradic current. This stage in recovery should be carefully looked for, and the change of current made.

The indications for tenotomy and for mechanical treatment of the deformities are various, and really out of my province. You would do well to call in the aid of a surgeon skilled in the devising of apparatus, and make use of the instrument as

auxiliary means in promising cases. The most important use of tenotomy I take to be that referred to above, facilitating the galvanic treatment. Incurable cases, and cases in which only slight improvement can be hoped for, had better be at once sent to an orthopedic surgeon for advice and treatment.

The foregoing table of autopsies, in cases of infantile spinal paralysis, may prove useful. I believe it to be quite full.