A Study of Six Hundred and Thirty-Five Cases of Infantile Paralysis.

WITH ESPECIAL REFERENCE TO TREATMENT.

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ONE HUNDRED AND THREE DEARBORN AVENUE.
CHICAGO.

A STUDY OF SIX HUNDRED AND THIRTY-FIVE CASES OF INFANTILE PARALYSIS.

WITH ESPECIAL REFERENCE TO TREATMENT

FROM THE CHILDREN'S HOSPITAL,

BOSTON.*

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The following paper deals with an analysis of 635 consecutive unselected cases of infantile paralysis seen at the Orthopedic Out-Patient Department of the Childrens' Hospital, Boston, between Jan. 1, 1897, and Jan. 1, 1908. We are indebted to the Orthopedic Department of the Hospital for permission to report the cases, and to the neurologist for the privilege of consulting the records of that department in cases common to both clinics. The investigation was begun eighteen months ago, and so far as possible the cases have been examined personally by one of us.

FUNDAMENTAL ANATOMIC CONSIDERATIONS.

We are dealing with a lesion of the motor cells of the anterior horns of the spinal cord, and, as it is obvious from the pathology that the harmful agent reaches these cells through the blood current, the relation of the arterial supply to the motor cells becomes of importance.

Spinal Motor Cells.—The spinal motor cells lie in spindle-shaped bundles in the anterior horns of the spinal cord, being chiefly located in the cervical and lumbar enlargements. The greatest extent of these groups is in the length of the spine, one group frequently reaching through two or three spinal segments, while it overlaps other groups lying above and below

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it. Whether such group represents an anatomic set of muscles or whether the cells represent a functional grouping, as held by Lapinsky, is not settled, but in any event the relations of the cells to others in the same group and to those of other groups is most complex. One center will send impulses to two or more muscles, and, on the other hand, one muscle will receive motor impulses from more than one group. This matter of the complicated origin of motor impulses becomes of great importance in considering treatment.

Blood Supply of Spinal Cord.—The blood supply of the spinal cord is derived chiefly from one anterior and two posterior spinal arteries. This anterior artery runs the length of the cord in the anterior fissure and is the chief supply of the anterior cornua. It is with the distribution of the blood supplied by the anterior spinal artery that we are chiefly concerned. From this anterior spinal artery there arise two lateral horizontal branches, one to each side, which enter the cord at different levels, about 200 in number. These are the central or sulco-commisural spinal arteries. Entering the anterior cornua, they terminate in a network after subdividing into various branches. Each of these central arteries has ascending and descending branches, covering an area in the length of the cord of from 1.5 to 2 cm. These branches are terminal arteries and do not anastomose with each other. Although they supply chiefly the anterior horns, there are branches to the bordering white matter and one posterior branch.

The terminal twigs of the central arteries do not individually supply each an especial group of motor cells in the anterior horns, but are distributed apparently without reference to the cell groups so that each cell group gets its blood supply from several branches of one central artery or from those of more than one central artery. The irregular distribution of destruction of foci in infantile paralysis is thus explained, being dependent on the fact that the lesion follows the blood course and is determined by the distribution of the arteries affected.

The main fact is that, on the whole, the groups of motor cells run in the length of the cord and have their associations in this direction, and that the blood supply is mainly horizontal. Hence, unless the lesion is very extensive, some cells in a group are likely to escape destruction. *The utilization of such remaining cells in partly destroyed groups becomes, therefore, one of the most important objects of treatment.*

ETIOLOGY.[1]

Bacteriology.—Most examinations of the fluid obtained by lumbar puncture by observers all over the world have been negative. In about thirty cases organisms have been reported as present in the last ten years, but these organisms have differed, the most constant finding being the report of fifteen cases by Geiersvold and Harbitz and Scheele,[2] where the same diplococcus was found.

Experimental Production.—Paralysis of a sudden type frequently occurs in laboratory animals, especially in young ones. Lesion of the cells in the anterior horns of the cord has been found in experimental paralysis caused by the injection of staphylococci, streptococci, colon bacilli, pyocyaneus, and other organisms in rabbits. A selective action of certain poisons for the anterior spinal cells is thus shown, and the same is true of some of the metallic poisons, such as lead and arsenic.

Epidemic Character.[3]—That the disease occurs in epidemic form is well recognized, distinct epidemics having been reported from France, Italy, Germany, Austria, Norway, Sweden, Australia and in the United States from California, Alabama, Massachusetts, Maine, New York and Vermont. New York City in the summer of 1907 experienced the most severe epidemic ever yet reported anywhere.

Contagion.—The comparatively frequent occurrence of more than one case in a family suggests the possibly contagious nature of the disease or infection from a common source.

Traumatism.—A history of trauma was given in 47 of our series where a history was obtained, and occurring in so large a proportion of the cases it seemed worthy of notice. In 32 cases there was given a history of accident followed almost immediately by paralysis, as shown in Table 1.

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1. Lovett, R. W.: General Review of Etiology. Boston Med. and Surg. Jour., July 30, 1908; Trans. Massachusetts Med. Soc., 1908.

2. The Journal A. M. A., Jan. 25, 1908.

3. Holt and Bartlett: Am. Jour. Med. Sc., May, 1908.

TABLE 1.

	No. of Cases.
A.—Slight accidents:	
Slight fall	20
Fall from cradle	3
Fall from chair	1
Fall from swing	1
Fall from carriage	3
Fall from automobile	1
Fall while walking, skating or playing	5
Dropped by nurse	1
Falling under other children	1
B.—More serious accidents:	
Fall from a third-story window	1
Fall from first story window	1
Stone fell on the head	1
Gate fell on the foot	1
Fracture of tibia	1
Total	41

In all of these cases the paralysis followed immediately or so shortly after the accident as to suggest to the parent's mind the relation of cause and effect.

In a second series of six cases (Table 2) the paralysis did not follow the accident immediately, but came on at an interval of some days.

TABLE 2.

Fall from chair	Paralysis a few days later
Fall from carriage	Paralysis a few days later
Fall from bed	Paralysis 7 days later
Fall	Paralysis 3 weeks later
"Injury"	Paralysis 1 week later
Sprain of ankle	Paralysis later

Of these 47 cases, 16 were rejected on the ground that the history was not sufficiently definite as to the traumatism, leaving 31 cases where a clearly described accident or fall preceded the paralysis immediately or at an interval of a few days.

We do not consider that our cases, suggestive as they are, establish the traumatic origin of anterior poliomyelitis, but that so large a number give a clear history of trauma preceding the paralysis we consider worthy of serious consideration in formulating the etiology. Unless one is disposed to dismiss this evidence as necessarily untrustworthy, which seems hardly justifiable, one of two suppositions seems likely: (1) Either trauma may be regarded as predisposing to infection of the spinal cord, or (2) trauma causes a disease so closely resembling anterior poliomyelitis as to be indistinguishable from it. In 234 cases of infantile paralysis reported to the State Board of Health of Massachusetts as occurring in the state in 1907, there were 35 authentic traumatic histories given by the family physician in attendance at the time after rejecting all questionable ones.[1]

The association of trauma and anterior poliomyelitis has not been wholly overlooked in literature. A case is reported by Borckhardt[4] where a child, 1 year old, was scalded in the face by boiling water. Vomiting and fever followed in a few days with paralysis, and four days later death ensued, the autopsy showing the characteristic changes of anterior poliomyelitis. The other cases reported are mostly or wholly those of adults, the class of accidents described being practically identical with those in this series. The cases are as follows: von Leyden,[5] 2 cases; Stark,[6] Perrin,[7] Meyer,[8] Flatley,[9] and Bullen,[10] one case each.

Unusual Onset.—In the analysis of the onset of our cases there separated itself a group of cases where the onset was atypical or associated with other diseases. In six cases the paralysis appeared slowly and gradually, without febrile attack. In eight cases the onset of the usual character followed exposure to cold. In one case the disease manifested itself on the day after the child had been jumping rope to excess. In thirty-five cases it was said to have followed or to have been associated with the diseases enumerated in Table 3.

TABLE 3.

	No. of Cases.
Bronchitis	3
Bilious attack	1
Teething	2
Nephritis	1
Typhoid fever	5
Scarlet fever	3
Measles	3
Pneumonia	2
Intestinal diarrhœa	8
Rheumatism	5
Diphtheria	2

The diagnosis of these cases is, of course, doubtful, as the original onset may have been mistaken for any of these diseases, and there is no means of knowing

4. Arch. de méd. des enf., October, 1902, p. 608.
5. Arch. f. Psychiat., 1876, vi., 271.
6. Neurol. Centralbl., 1904, xxiii, 14.
7. Arch. de méd. des enf., October, 1902, p. 608.
8. München. med. Wchnschr., 1901, No. 5.
9. Am. Med., 1904, viii, 956.
10. Jour. Ment. Sc., 1892, xxxviii, 71.

whether this diagnosis was made by the parents or by a competent physician.

Two further subdivisions of the general group deserve special mention. Two patients became paralyzed during an attack of boils or abscesses, which is of much interest in connection with what has been said in the bacteriology about experimental septic infection, and one patient was paralyzed in the hospital ward during recovery from an operation for appendicitis. Twenty-three cases were said to have followed cerebrospinal meningitis. It is fair to assume that the majority of these cases of so-called meningitis were merely the routine onset of the disease of rather severe character, but in six cases the character of the meningitis was established by hospital observation and the subject has already been discussed by Dr. W. N. Bullard, neurologist to the hospital.

Summary of Etiologic Evidence.—The disease is generally believed to be of infectious origin, but direct bacteriologic proof is as yet absent. The character of the onset, the epidemic distribution, the apparent contagiousness, and the experimental production of paralysis in animals all point in this direction. The fact that the disease selects by preference children in the first dentition and prevails in the summer and early fall offers a close analogy to the gastrointestinal disease and suggests a possible source of infection in the intestinal tract, possibly from some bacillus contained in milk. It may be that such a bacillus liberates a toxin which is the harmful agent and disappears.

With the evidence offered by our cases we can not be sure that the disease represents infection by one specific organism nor that it is always of infectious origin for it must be remembered that the clinical picture may represent only the reaction of the spinal cord to one of several causes, such as pyogenic organisms, the results of exposure to cold, traumatism, etc. We have not yet sufficient evidence to say whether we are dealing with a specific infectious disease or with a disease of more than one origin.

Sex.—Of the 635 patients, 334 were boys and 301 were girls.

Age.—Table 4 shows the age at onset.

TABLE 4.

Age.	No. of Cases.
Early	8
Under six months	15
6-12 months	55
1 year (second year of life)	150
2 years	128
3 years	65
4 years	40
5 years	36
6 years	18
7 years	13
8 years	8
9 years	7
10 years	6
11 years	4
12 years	1
13 years	1
Total	555
Not noted	80

Season of Onset.—Most of the cases where the date of onset was noted ocurred during the spring and autumn months, as shown in Table 5.

TABLE 5.

January	8	July	36	Spring	8
February	4	August	43	Summer	17
March	5	September	47	Autumn	13
April	5	October	39	Winter	7
May	6	November	29		
June	13	December	4		

Relation of Severity of Onset to Paralysis.—The character of the onset was tabulated with regard to the degree of paralysis following, to see what, if any, relation existed.

The onset was classed as severe when it lasted over a week, or was accompanied by unconsciousness, delirium or convulsions. It was classed as moderate when it was from a day or two to a week in duration, when the symptoms were not alarming and when the fever was moderate. It was classed as slight when the child was affected by a slight febrile attack for one or two days, when the temperature was but little elevated and the illness apparently trifling.

The paralysis was classed as severe when it was complete in one limb, when it involved more than one limb to any serious degree, or when walking on the paralyzed leg was impossible. It was classed as moderate when important groups of muscles were affected enough to make the child seriously lame, when marked deformity was present, or when it was the cause of moderate disability. It was classed as slight when only one muscle or an unimportant group was affected, when the limb was merely weak, or when serious lameness and disability were absent. Arranged in this way, the results were as shown in Table 6.

TABLE 6.

Character of Onset.	No. of Cases.	Paralysis. Severe.	Moderate.	Slight.
Severe	98	51	28	19
Moderate	84	25	44	15
Slight	62	11	28	23
No attack at onset	31	2	10	19

From this it appears that a severe onset, as defined above, is more likely to be followed by severe paralysis than by any other type, and that slight paralysis is unlikely (19 in 88). A moderate attack is most often followed by moderate paralysis, with a greater tendency to a severe than to a slight form. A slight attack shows about the same tendency to be followed by moderate or slight paralysis (11 severe cases among 62). Cases in which no onset is noted are most often followed by slight or moderate paralysis with little tendency to the severe type. The same is true of cases where the affection is attributed to traumatism.

The Distribution of the Paralysis in 628 cases is shown in Table 7. (For comparison the composite group generally quoted is reproduced at the right of the table.)

TABLE 7.

	Authors' Cases.	Duchene; Seeligmuller; Sinkler; Starr.
Both legs	130	184
Right leg	216	134
Left leg	239	133
Right arm	5	30
Left arm	5	23
Both arms alone	0	9
All four extremities	3	51
Arm and leg, same side	15	48
Arm and leg, opposite side	7	10
One arm, both legs	2	15
Abdomen with other paralysis	6	

Our figures show paralysis of one leg to be nearly four times as common as paralysis of both legs, whereas the other figures show it to be twice as common; both sets of figures show paralysis of the arm and leg of the same side to be more common than crossed paralysis. Paralysis of the abdominal muscles was recorded as present in six of our cases and probably was overlooked in the examination of others.

Affection of Individual Muscles of the Leg.—Four hundred and seventy-eight cases where the data were sufficient were tabulated as to the frequency of affection of the individual muscles in paralysis of the leg. The examination was not made by electricity, but by ascertaining whether or not the functions of a certain muscle or group of muscles could be performed in response to voluntary effort. For example, the patient was told and shown how to abduct the foot; when the motion was being attempted the finger was placed on the peroneal tendons. If the motion was properly performed, the muscle was classed as "normal"; if some contractile power was present, but if motion was imperfect, it was classed as "weak"; if no power of contraction existed it was classed as "paralyzed." For purposes of tabulation the muscles in the lower leg were divided into groups as outlined in Table 8.

TABLE 8.

Anterior	Extensor longus digitorum. Extensor proprius hallucis.	Posterior	Gastrocnemius. Flexor longus digitorum. Flexor longus hallucis.
Internal	Tibialis anticus. Tibialis posticus.	External	Peroneus longus. Peroneus brevis.

The number of cases in which the different groups of the lower leg were affected, no case being entered in two groups, is shown in Table 9.

TABLE 9.

Anterior	92	Anterior, external and internal	18
Internal	40	Posterior and internal	34
Posterior	45	Posterior and external	7
External	31	Posterior, internal and external	8
Anterior and internal	29	Tibialis anticus	95
Anterior and external	27		
Anterior, internal and posterior	14		

The chief practical lesson to be drawn from this analysis is that the internal muscles are affected more frequently than the external, and the anterior more often than the posterior. Of combinations into which the internal muscles entered, there were 238 cases of paralysis against 91 for the external muscles. In the same way there were 180 such combinations in which the anterior entered against 108 for the posterior. The quadriceps muscle of the thigh was affected alone or in combination with muscles of the lower leg, not counting cases of complete paralysis, in 305 legs, being the muscle most frequently affected in either thigh or calf. The tibialis anticus and anterior muscles of the lower leg came next in frequency, and the short toe flexors were the least likely of all to be affected, frequently persisting in legs otherwise wholly disabled.

The anterior muscles of the thigh are affected much more frequently than the posterior hamstring muscles. When only one hamstring muscle is affected it is more often the internal than the external. The sartorius muscle frequently escapes when the quadriceps femoris is paralyzed, and we have never found the sartorius muscle paralyzed alone. The motor center for the sartorius is given by Edinger as one segment higher than that of the quadriceps.[11] Except in cases of extensive thigh paralysis the sartorius muscle is likely to be found intact, a matter of some importance in the question of treatment, as it may be substituted by operation for the quadriceps.

As to the relative frequency of the affection of internal and external thigh rotators, the fact that the psoas and sartorius muscles frequently escape, makes observation of this somewhat inaccurate, for the reason that the latter act as rotators. Therefore, although our figures apparently show a more frequent paralysis of the internal than of the external rotators, and thus confirm general clinical observation, we regard it rather as indicating that the muscular balance was disturbed in that direction than that especial muscles were paralyzed. Involvement of the adductor thigh muscles is far more common than that of the abductor muscles.

Complete Paralysis.—There were 95 instances of complete paralysis of the thigh on one or both sides; 37 cases were recorded as affecting only one side. In 34 of these the paralysis was also complete in the calf of that leg. The right arm was involved once, and the abdominal muscles twice in this group. Fifty-eight cases showed complete paralysis of the lower extremity on one side and some involvement of the other. In 26 of these

11. Bau der nervösen Centralorgane, Leipsic, 1896.

the paralysis was complete in both thighs and both calves. In 31 cases the paralysis was not complete on both sides, in 10 affecting only the quadriceps of the least affected side. In one case there was no note of the muscles affected. Both arms were paralyzed in 2 cases, one arm in 3 cases, the hip was dislocated in 2 cases, and the abdominal muscles paralyzed twice in this group.

Paralysis of the Arm.—Paralysis of the upper arm was much more frequent than paralysis of the forearm, the deltoid apparently suffering most frequently, often in connection with the biceps, triceps, or scapular muscles, or with all of them. Paralysis of the forearm was comparatively uncommon.

In general, the paralysis was more often symmetrical than asymmetrical, roughly speaking, when both sides were affected. In complete paralysis of one leg it has been seen that complete or severe paralysis of the other side existed in nearly two-thirds of the cases (58 double to 37 single). When the paralysis was partial in one leg and the other was affected, the paralysis in the second leg was more likely to be external than internal if the paralysis of the first leg was external.

Deformities.—These are largely, if not wholly, unnecessary, and are of two kinds—(a) static deformities, resulting from superincumbent weight coming on structures imperfectly supported by the paralyzed or weakened muscles or by the weight of some part dragging and stretching the structures; such are valgus deformity of the foot, hyperextension of the knee, dislocation of the hip, scoliosis, dislocation of the shoulder, and some cases of talipes equinus.

The second class of deformities (b) are to be classed as muscular and result from the action of muscles whose antagonists are paralyzed or weakened. These unopposed muscles cause distortions of one or more joints by their continued pull, which is not antagonized by their paralyzed or weakened opponents, a proper muscular balance being required for joint equilibrium. Such deformities are equinus, varus and calcaneus deformities of the foot and their combinations with those cases of valgus caused by paralysis of the tibials, one or both, flexion deformity of the knee and hip, and cases of scoliosis where the muscles of one side of the spine are paralyzed or weakened. These cases are in all instances accompanied by muscular contractions on the shortened side.

It can be predicted in advance in most instances what a definite muscular paralysis will produce in the way of deformity. For example, in a flail leg unable to bear weight the constant plantar flexion of the foot will induce talipes equinus; if the patient can bear weight on a generally weakened leg a valgus will result from improper support of the foot from the tibial muscles. If the anterior muscles are paralyzed, an equinus deformity will ensue with contraction of the tendo Achillis. If the posterior muscles are paralyzed, the patient will walk on his heel and a calcaneus deformity be present.

From the study of our cases we have formulated the following table of deformities (Table 10) of the foot and their causes:

TABLE 10.

Deformity.	Resulting from paralysis of.
Varus	peronei.
Valgus	anterior tibial.
	posterior tibial.
	both tibials.
	flexor longus hallucis.
	whole leg (weakened).
	complete paralysis.
Equinus	anterior muscles, paralyzed or weak.
	complete paralysis (from dangling).
Equino varus	anterior muscles. (with persistence of flexor longus hallucis).
	anterior and external group.
	paralysis apparently complete (probably toe flexors remaining).
Equino valgus	anterior and internal muscles.
	anterior muscles and weight bearing.
Calcaneus	posterior muscles.
Calcaneo valgus	posterior muscles and one or both tibials.

PATHOLOGY.

The modern view of the pathology of anterior poliomyelitis is somewhat different from the view advanced by Charcot, who concluded that the changes in the ganglion cells were primary and that the affection was parenchymatous in character. His conclusions were drawn largely from the observation of cases of long standing in which the terminal results were evident. Of late years the study of recent cases has led to a change in the point of view, and by the majority of modern pathologists anterior poliomyelitis is now regarded as a more or less generalized inflammation of the cerebrospinal axis, and the interstitial character of the process is emphasized rather than the parenchymatous. There are still some observers who hold the original view of Charcot, and in certain recent autopsies the changes in the ganglion cells predominate, but the majority of those who have had an opportunity to examine sections of the cord in recent cases incline to the view that the poison reaches the cord through the arteries supplying it and that the changes are most evident where the blood supply is freest and that the lesions are determined very largely by the distribution of the most important blood supply; namely, the branches of the anterior spinal artery.

In the early stage of the affection there is noticed a congestion of the meninges and of that part of the gray matter supplied by the anterior spinal artery. The vessels are dilated, and the perivascular spaces and the gray matter are filled with leucocytes, small and large mononuclear lymphocytes, which occur especially in the pia and white matter, and polymorphonuclear lymphocytes which are especially evident in the gray matter. The capillaries are ruptured, and there may be extravasations in the area supplied by the blood vessels. Thrombosis of the branches of the anterior spinal artery may occur. Macroscopically, hyperemia along the vessels and fissures is evident. The ganglion cells of the anterior horns become cloudy and swollen and the nucleus may appear to be granular. Later the protoplasm no longer stains in the customary way, the cell appears swollen and loses its sharp outline, the nucleus becomes faint and some of the dendrites disappear. The grade of involvement of the nerve cell is, in general, proportionate to the nearness of the cell group to the most extensive interstitial changes.

It is probable that, from the stages described, regeneration is possible, but from the severer grade of changes to be next described it is probable that no repair of the cell is possible.

In the next stage the cell appears swollen and irregular. The nucleus is not visible; the dendrites have dropped off; the chromophile granules have lost their regular arrangement, and vacuoles may be present. In the last stage the cell body is reduced in size, perhaps being no larger than the original nucleus. It is granular, and leucocytes have penetrated the pericellular spaces and have encroached on the cell body.

There is in the interstitial tissues a stage parallel to that occurring in the gray matter, in most cases shown by a shrinkage and progressive destruction of the neuroglia. There is hyperplasia and hypertrophy of the neuroglia cells and a diminution in the size of the axone and early degeneration of nerve fibers. There is a secondary degenerative process, an atrophy of fibers in the anterior nerves connected with groups of cells already affected. There is a shrinkage and slight sclerosis in the anterior lateral columns above and below the lesion. It has been commented on by some observers that the pathologic appearances are in most cases severer than the clinical history would lead one to expect, and the examination of recent cases emphasizes this point of view still further, and it is recognized that parts which were clinically normal in function may be found on microscopic examination to be slightly affected. It has been found on microscopic examination of the pia that in the severe cases there is a widespread inflammation. Foci in the cord correspond in all cases to involvement of the pia on the same level, but inflammation of the pia may occur without the cord involvement. In the severer cases Harbitz and Scheele found an extensive inflammation of the pia over the pons, cerebellum, the cerebrum itself and also more or less inflammation of the brain substance, less intense in the medulla and pons than in the cord. In milder cases these investigators found fat granule cells in the walls of blood vessels of the medulla, occasionally in those of the basal ganglia, and in some cells in the gray cortex.

In a word, the process seems to be a widespread inflammation, with especially marked changes in the anterior cornua of the cord. Although the lesion is most marked in the anterior horns, the central gray matter is, in some cases, affected, explaining the occurrence of pain and subsequent interference with the growth of the limb, the central gray matter being especially associated with growth.

It is not unusual in severe cases to find also some involvement of the posterior horns which with the meningeal inflammation also explains the occurrence of pain in so many cases in the acute stage of the disease. Following the acute stage there is a gradual shrinkage of the entire area of the anterior horn by the formation of the scar tissue inevitably following the process described. This leads to a collapse inward of the white columns surrounding the affected area in the gray matter, and there is also an atrophy in the nerves issuing from the horns through these columns to the anterior roots. The anterior nerve roots involved are diminished in size and there is a degeneration of the fibers composing them. The widespread distribution of scar tissue and the mechanical influence of the interstitial tissue thus affected, on the neighboring cells is a factor occasionally spoken of as predisposing to further destruction of the motor cells. In old cases the outlines of the tracts are indistinct, and microscopically one finds the vessels sclerosed in all their coats and the group of ganglion cells replaced by fibrous tissue rich in nuclear cells.

In short, the cord at the affected level appears shrunken on the diseased side, the changes being particularly marked in the anterior horn. In some cases the changes in the gray matter are predominant and but little interstitial change can be found; in other cases the reverse is true. Some writers have assumed the existence of two distinct types of the affection, but, whatever the theoretical origin of the disease may be, the practical conclusion for the surgeon is, "That in the lesion of the great multipolar nerve cells of the anterior horns the striking character of the affection and its sequelæ are manifest" (Schmaus).

The changes in the affected muscles vary between severe parenchymatous and interstitial changes to a wasting of whole muscles, depending on the intensity and duration of the affection of the ganglion cells. This atrophy apparently does not begin at once, being absent in a case of Jagic[12] five days after the attack, but fatty degeneration without atrophy was found by Redlich[13] ten days after the attack. It is, however, seen from three to four weeks after the attack.[14] Later in the history are to be found fatty, waxy, and granular degeneration. In the latest stages these degenerative changes are represented by the replacement of the muscles by thin bundles or layers of connective tissue. The

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12. Wien. med. Wchnschr., 1899, pp. 9-10.
13. Wien. klin. Wchnschr., 1894, p. 287.
14. Lorenz: Die Muskelerkrankungen, Vienna, 1904, p. 574.

muscle nuclei remain normal or are increased in size. In operations on the living, where the disease has been of considerable duration, normal muscles are bright red, disused or partly paralyzed muscles are rose colored, and wholly paralyzed muscles are yellowish in color.

The pathologic conclusions presented above have been largely obtained from the works of Starr,[15] Schmaus,[16] Harbitz and Scheele,[17] Hoch,[18] Pretorius,[19] Goldscheider,[20] and Lorenz.[21]

TREATMENT.

The matter of treatment will be discussed only in general terms, for no tabulation of results in so large and varied a group of cases would be practicable or of great value.

Stage of Onset.—The stage of onset represents an acute hematogenous myelitis. Quiet in bed is essential, and beyond this it is doubtful if much is accomplished by the administration of ergot and similar drugs or by blisters or other counter irritants applied to the back. As there is a possibility that the absorption of toxins from the intestinal tract is an important factor, it would seem reasonable to empty the intestines at the onset by catharsis in all cases. From the onset paralyzed limbs should be supported in a normal position, and muscular dragging and stretching should be avoided. Pain and sensitiveness, when present, generally last only for the first few days and will not long prevent the use of supports. A foot can be supported at right angles to the leg by splints of wood, tin, or plaster of Paris. It is doubtful if the use of massage and electricity in the first days after the attack is advisable. It would seem wiser to allow from one to three weeks for nerve centers to quiet down before stimulating their peripheral connections. How long the period of rest to the limbs should be carried out can not

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15. Organic and Functional Diseases of the Nervous System. Lea Bros., Philadelphia, 2d ed.
16. Vorlesungen über die pathologische Anatomie des Rückenmarkes, Wiesbaden, 1901.
17. The Journal A. M. A., Jan. 25, 1908.
18. Hoch: Jour. Nerv. and Ment. Dis., September-October, 1905.
19. Centralbl. f. Kinderh., new series 58, 1903, p. 193.
20. Deutsch. med. Wchnschr., 1893, No. 19.
21. Die Muskelerkrankungen, Vienna, 1904.

yet be stated, but will probably be answered definitely by the results observed in the recent New York epidemic.

The Stage of Established Paralysis.—A localized hematogenous myelitis has attacked the cord and has destroyed more or less at random certain areas of spinal nerve centers. Unless the cord lesion has been extensive (for the reasons discussed in the opening section) the chances are rather against the total destruction of all the centers and associations of any large number of muscles, some centers or associations having perhaps escaped.

It is, therefore, obvious that in the stage of established paralysis, whether early or late, it is important to prevent unnecessary muscular deterioration and to utilize, so far as possible, the unaffected cells in partly affected centers.

In other words, to prevent (a) muscular stretching, (b) muscular disuse, which are both obviously harmful to the well-being of the muscles, and (c) stimulate to functional activity muscles partly paralyzed or simply disused.

It is coming to be the experience of those who deal with the later stages of infantile paralysis, and it has been particularly evident in this series of cases, that there exists in many paralyzed limbs a possible amount of muscular power that is not suspected and will not be available unless cultivated and developed. For example, it has several times been our experience in this series to examine cases of paralytic talipes equinus of long standing where there was no power in the anterior muscles of the lower leg. The tendo Achillis has been divided, and the foot put up at a right angle in plaster for three or four weeks. When this plaster has been removed, the anterior muscles have been found to functionate and from that point on to have recovered power. They had been stretched and out of balance before the operation; when properly balanced and allowed to shorten they possessed function.

The absence of function in a muscle or group of muscles does not necessarily mean paralysis, even in the later stages of the affection, as long recognized by the neurologists. This paper becomes, therefore, a plea for the more persistent attempt to use other measures in addition to braces and operations in the treatment of these cases.

(*a*) *Prevention of Muscular Stretching.*—The harmful effect of stretching muscles in paralyzed limbs was dwelt on forty years ago by C. F. Taylor,[22] but has not been sufficiently remembered. He wrote as follows:

Any position of a limb which allows the extensor muscles to become shortened must inflict a worse damage on the flexors by keeping them extended till they lose their remaining irritability and become degenerated. Now, we have another fact connected with these cases when they have arrived at the stage of deformity, viz: The difficulty of treatment consists much less in relaxing shortened muscles than in giving tone and strength to their antagonists—the lengthened and weakened ones. Indeed, it is this, in the destruction of all remaining muscular irritability, and in many instances the destruction and entire loss of the substance itself of the expanded muscle, which constitutes the principal anxiety in treating this class of deformities. As this important consideration has been many times neglected, if not entirely lost sight of, let us consider the effect of simple extension on the power and functions of muscular tissue in its healthy state.

In reference to this the following propositions are believed to be true, viz.:

First. To retain a healthy muscle in an expanded state for a certain length of time is to diminish or destroy its irritability and contractible force.

Second. To extend a muscle while in the act of contracting, that is, to overcome it, is, at once, to destroy its irritability and force.

Having shown that position of the limbs alone is sufficient to cause deformity even in the healthy subject, it only remains to inquire: Is this actually the case in infantile paralysis? The reply must be affirmative.

The importance of supporting the limb during the early stage in a correct position has been alluded to. In the stage of established paralysis orthopedic apparatus is intended not only to make locomotion possible, but to prevent deformed positions in standing and walking and with this to prevent muscular stretching. The foot should be neither in valgus, calcaneus, or varus when weight is borne, but supported in the proper plane at right angles to the leg. The knee should be neither in a flexed nor hyperextended position in weight-bearing, but straight. A very good general rule for the use of supporting apparatus is that it should be used when

22. Infantile Paralysis, Philadelphia, Lippincott, 1867.

weight-bearing induces a deformed position in the foot or knee, because such deformity not only leads to permanent distortion of the bones, but is necessarily accompanied by prolonged muscular stretching and may lead to contraction deformity of the antagonistic, not stretched muscles.

(*b*) and (*c*) *The prevention of muscular disuse and the stimulation of nervous centers* in the cord in partially destroyed groups are accomplished together by the same measures. For a muscle to lie idle and not to contract is bad for it whether it is paralyzed, or partially paralyzed, or not paralyzed at all. It will deteriorate locally and its functional cord center will not be stimulated to establish new associations or develop any latent power. But as a prerequisite to all treatment of this kind it must be assumed that the foot is properly supported by apparatus in walking, if without it it falls into a position of deformity. (The use and function of supporting apparatus is discussed in text-books on orthopedic surgery.)

Having thus supported and held the paralyzed limb in a position fit for functional use, massage and electricity are to be regarded as measures to improve the condition of the muscles and to lead toward functional use of partially paralyzed, stretched or disused muscles. Of themselves they are not curative or especially helpful, except in so far as they promote muscular well-being and lead to muscular contractibility.

Active muscular contraction or muscle training is, in general, the most universally applicable therapeutic measure at our command and one insufficiently appreciated. Having worked with massage or electricity or both, for the development of the muscle or muscles an attempt should be made as soon as it becomes possible, to get the partly affected muscular group to contract in response to a voluntary impulse, at first being satisfied with only a quiver in the fibers at the attempted movement, but later affording aid in the attempted movement by the hand, by some improvised apparatus, such as a strap or bandage, or by the use of some mechanical apparatus, such as those of the Zander system, those used so extensively in Germany, or by some simpler form. In short, the effort is to be made to make the partially affected muscles contract without being overweighted and, having learned to contract, to educate them to use more and more power until they perform useful function.

To repeat this most important point, mechanical or conservative treatment should consist not only in supporting the paralyzed limb by an adequate and acurately fitted brace, but should push on to the develoment of muscles apparently paralyzed, but really with a possibility of function.

Tendon Grafting or Anastomosis or Transfer.[23]—Although there were 120 cases of tendon transfer in the present series, they will not be analyzed here, for the reason that a report on this subject has already been made from the hospital and because the limits of the paper are such that it is impossible to consider every phase of the subject, and, as this part of the subject is adequately treated in current literature, we have chosen rather to devote our space to those aspects of the subject which have received less attention. The following practical conclusions have, however, been reached from the study of these cases:

1. It is important to remove deformity by a preliminary operation when it is present to any considerable degree and not to correct the deformity and perform the tendon transfer at one operation.

2. The operation should not be performed on very young children.

3. Periosteal implantation yields better results than when tendons are united to tendons.

4. Simple operations are more satisfactory than complicated ones.

5. It is not advisable to turn sharp corners with transferred muscles, but to secure as straight a line as possible of muscular pull from origin to insertion.

6. The substitution of small muscles for large ones is likely to be unsatisfactory, e. g., one of the peroneal muscles is rarely a satisfactory substitute for the gastrocnemius.

7. Tendons must be inserted on the stretch and the foot maintained for some weeks in a position of overcorrection.

8. The use of silk tendons has proved practicable and satisfactory.

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23. Assn. franç. de chir., 20th cong., 1907, pp. 380-574.

9. Finally, the most striking conclusion that has been impressed on us is that the after-treatment is as important as the operation, if a successful result is to be obtained.

To perform a tendon transfer, fix the leg in plaster for some weeks, until muscular atrophy has become marked, and then to allow walking unprotected is bad treatment. Unless the operated leg is massaged from the sixth week on and the newly transferred muscle is trained to its new function shortly afterward, and unless this massage and training are carried over some weeks, the best results can not be expected. No matter how good the operation, it must be followed up or the operation of tendon transfer will be regarded by the individual operator as a much overestimated procedure; if, on the other hand, he performs a sound and reasonable operation, and has his patient properly treated over a period of about six months, he will find it, in a large proportion of cases, one of the most satisfactory procedures in operative surgery. No importance can be attached to published results of tendon grafting unless there is published at the same time an account of the after-treatment. If it is lacking, poor results are explained.

The method followed at the Children's Hospital has been as follows: The plaster put on at the operation is worn for six weeks. It is then split, removed for massage, and reapplied. About two or three months after the operation a brace is applied which shall support the foot in a position to relieve strain on the transferred tendon, and muscle training is begun. Walking is allowed from three to six months after operation in this brace, when the tendon seems to possess enough power to warrant it, and the brace is discontinued when circumstances warrant it. At the Hospital Out-patient Orthopedic Clinic a department for massage and muscle training has proved a necessity and is devoted wholly to the treatment of infantile paralysis, both operative and non-operative. It has proved of such great use that it is planned to extend it in the near future and add simple mechanical appliances to amplify the work.

Arthrodesis.—The operation of arthrodesis was performed fifty times in this series, but will not be dwelt on in detail here, for the same reasons given in speak-

TABLE 11.

Name of Observer.	Variety of Paralysis.	Operation.	Result.
1. Peckham[24]	Peroneal	Int. into ext. popliteal	Marked improvement.
2. Young[25]	Valgus	Branch to ant. tib. into musc. cut.	Great improvement.
3. Taylor[26]	Equinus	Ext. into int. popliteal	More power.
4. Taylor	Equinus	Ext. into int. popliteal	Can flex foot.
5. Spiller and Frazier[27]	Ant. tibial	Ant. tib. into musc. cut	Return of normal power.
6. Spiller and Frazier	Peroneal	Ant. tib. into musc. cut	No improvement.
7. Murphy[8]	Talipes	Ext. popl. into int. popl	Improvement.
8. Murphy	Peroneal	Musc. cut into ant. tibial and tenoplasty	Time too short for report.
9. Hackenbruck[29]	Equinus	Ant. into ext. popl	No result—Keloid.
10. Hackenbruck	Equino-varus	Ant. into ext. popl	No result—Keloid.
11. Hackenbruck	Equinus	Ant. into ext. popl	Excellent result.
12. Tubby[20]	Equino-varus	Nerves of gastrocn. soleus into ext. popl	Return of movement.
13. Tubby	Equino-varus	Nerves of gastrocn. soleus into ext. popl	Return of movement.
14. Tubby	Equino-varus	Ext. into int. popl	Unsatisfactory.
15. Spitzy[31]	Anterior and exterior	Ext. into int. popl	Good result.
16. Spitzy	Obstetr. parai	Median into radial	Remarkable result.
17. Tubby	Erb-Duchenne	Ext. into int. branches of brachial plexus	No definite result.
18. Harris[32]	Deltoid	Fifth into fourth cerv. nerve	Good result.
19. Peuguiez[29]	Circumflex	Radial into circumflex	Too recent.

24. Providence Med. Jour., January, 1900, p. 50.
25. Am. Jour. Orthop. Surg., I, 27.
26. New York Med. Jour., 1906.
27. THE JOURNAL A. M. A., Jan. 21, 1905.
28. Surg., Gynec. and Obst., April, 1907.
29. Assn. franç. de chir., 20th cong., 1907, p. 444.
30. Brit. Med. Jour., 1906.
31. Ztsch. f. Orthop. Chir., 1905.
32. Tr. Clin. Soc., London, 1905.

ing of tendon transfer. The following conclusions have been reached from the study of these cases: The operation in the ankle joint is useful in properly selected cases. It has not been used in the knee. It has been both successful and unsuccessful in the hip, and fairly satisfactory in the shoulder, although here tendon transfer is advisable when possible. The use of arthrodesis in the ankle by means of silk has proved satisfactory and is preferable in children to the more destructive operation of removing the joint surface. The cutting away of the joint surface in young children is, at times, followed a few years later by a distortion of the foot into a position of varus. The latter operation should not be performed in the ankle on children much under the age of puberty.

Nerve Anastomosis.—The question of the value of nerve anastomosis is still *sub judice* and the operation was performed only once, with negative result, in the series reported. About 20 cases have been reported in the literature, which are given in Table 11, which presents the results of available recorded operations performed for paralysis of the limbs. The brilliant results reported in some of the cases would seem to promise a future for the operation.

CONCLUSIONS.

Infantile paralysis is a less formidable affection than is generally believed, partial paralysis is common, disused and stretched muscles appear to be paralyzed, but possess a possibility of function. In addition to mechanical treatment, an attempt should be made by massage, electricity and especially by muscle training to wake to activity the remaining cells in partly destroyed groups and thus to secure muscles which perform function. After tendon transfer, the development by muscle training of the transferred tendons is essential to good results, and without this the percentage of failure will be large.

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