The anterior tibial group of the leg and the extensors of the thigh are most frequently affected. The psoas, on the other hand, is rarely affected, even in the most extensive paralysis.

In the upper extremity the deltoid is most frequently paralyzed.

The sphincters of the bladder and rectum are never affected. In this respect we have an important difference between this form of paralysis and that following transverse myelitis. It is true that in some very young children the general disturbance with which the disease is ushered in may give rise to a few involuntary passages, but this is only temporary, and will not be present when the paralytic stage has set in. After the sixth to the ninth month has passed, we can no longer look forward to spontaneous recovery in the paralyzed muscles. Muscles which have not improved until then are apt to remain paralyzed, and go on to the third stage of the disease, the atrophic stage, in which extreme atrophy of the paralyzed muscles is the most prominent symptom. It must not be supposed, however, that the paralyzed muscles do not atrophy until six or more months after they have become the seat of the paralysis. On the contrary, atrophy of the fibres of the paralyzed muscles can be found with the microscope very soon (two or three weeks) after the paralysis has set in, and several authorities claim to have observed atrophy, evident to the naked eye, as early as the third week after the paralysis. In my own cases I do not remember to have observed any evident atrophy earlier than three months after the first occurrence of the paralysis. The cause of this atrophy is, as we have already seen under the head of pathology, not simple disuse of the paralyzed limb, but a cutting off of the trophic influence of the nerve cells of the anterior gray horns of the spinal cord; this results in a disturbance of the nutrition of the muscle tissue as well as organic degeneration of the nerve fibres. Disuse of the paralyzed limb undoubtedly aids in producing a diminution in the size of the paralyzed member. As a result of these causes we have a very extensive atrophy of the paralyzed muscles—so complete that frequently the configuration of the bones, with their anatomical points, can be felt or even seen with startling distinctness. Often all muscular tissue seems to have disappeared, leaving only the bones, connective-tissue bands, and skin. As already explained, however, these atrophic members in the last stages of atrophy may undergo a pseudohypertrophy, owing to the deposit of fat in place of the atrophied muscle fibres. In those muscles which undergo recovery after a certain amount of atrophy has taken place, the restoration of volume in the recovered muscle does not directly follow upon restoration to power. The muscle is only gradually restored to its former size some time after power has returned.

Atrophy is not limited to the paralyzed muscles, but affects the bones and ligaments, and even the blood-vessels; the extent and character of the atrophic changes have already been discussed. There is also atrophy of the skin; it is apt to be more delicate and thinner; it is of finer texture, and paler in color at first, although later on it becomes cyanotic in color, owing to the diminished blood supply in the paralyzed limb. This diminished blood supply is due to atrophy of the blood-vessels and the disappearance of the finer capillaries. The muscular coat of the arteries is atrophied, giving rise to a diminished arterial tension. In consequence of this diminished blood supply the surface temperature of the paralyzed member becomes greatly diminished. Fluctuation in temperature is very great, especially when the body has been exposed to cold. Thus the difference between the healthy and paralyzed limbs may be 20°, 30°, or even 40° F. It is almost impossible to keep the paralyzed limbs warm; friction and artificial heat serve to restore warmth for a time, but, unless frequently applied, they do very little good. As a result of the lowered temperature and slowness of circulation, the skin sometimes becomes the seat of ulcerations, which are very slow to heal, and are exceedingly painful. These ulcers affect only the scarf skin, and consist of a desquamation of the epidermis, the derma remaining unaffected. As part of the symptomatology of the paralytic and atrophic stage of the disease, we must devote some space to the discussion of the accompaniments and results of both of these conditions, namely, the deformities so characteristic of the final stages of this affection.

Third Stage.—Deformities Resulting from Infantile Spinal Paralysis.—A careful analysis of the deformities resulting from infantile paralysis will warrant their classification into two groups:

1. Deformities due to trophic changes in the limbs.
2. Deformities depending on the paralysis of muscles.

1. Deformities Due to Trophic Changes. In discussing the symptomatology of this disease, it was seen that, owing to destruction or interference with the trophic centres in the cord, all growth of the parts supplied by the nerves passing from the affected region was arrested, and the development which the affected tissues had attained before the time of seizure was diminished, the tissues retrograding and undergoing degenerative changes. The deformities of this group, then, are due, first, to cessation of growth; second, to retrograde metamorphosis. Cessation of growth is in every direction, and results in shortening and attenuation of the affected member as compared with the condition of the limb in health. Retrograde metamorphosis or atrophy will also cause deformity, either by shortening, or, more commonly, by attenuation; so that lack of growth, aided and reinforced by atrophy and degeneration, results in deformity by producing shortening or attenuation, or both together. From a consideration of the function of the upper, as compared with the lower, extremity of the body, it is evident that deformity due to shortening is of far more importance when it affects the lower extremity than when it affects the upper. Not only is it essential that the two limbs should be of equal length in order that the patient may walk and stand without deformity, but the proper performance of the functions of other parts of the body depends upon the lower extremities being of equal length. For, the trunk being supported by the two lower extremities acting as pillars, the shortening of one of these pillars as compared with the other will result in a corresponding lowering or sagging of that side of the body; the pelvis will become oblique, the normal side being on a higher plane; and were it not that conservative nature seeks to rectify the result of the disease, we should see these patients walking and standing with the body bent over to the side of the shortened leg. To maintain equilibrium the patient is compelled to throw the body to the opposite side, giving rise to a functional lateral curvature of the spine, which, in its turn, again in the effort to maintain equilibrium, is modified by a secondary curve. We have, then, as a result of the shortening of the lower extremity, deformity in walking and functional lateral curvature.

Atrophy and attenuation are of less importance here than in the upper extremity, disability rarely resulting from this cause alone; while, on the other hand, shortening is of less importance in the upper extremity than in the lower; for one arm or hand may be shorter than the opposing member, but were it not for the paralysis which accompanies the shortening, it would be of little importance. On the other hand, atrophy is of great importance in its effects upon the upper extremity. An atrophic shoulder will sag downward, producing even, in some cases, a lateral curvature high up. But, what is more common, the head of the humerus becoming atrophied, and the hand having no resistance below, as is the case with the foot, we have a resulting subluxation, which in itself would interfere with the use of the arm. In one of my cases this subluxation in every direction resulted from the slightest motion, and was an important element in the treatment of the case. Although, in the limits of this article, I consider it more profitable to indicate the *principles* which govern the production of deformities which result from infantile spinal paralysis, rather than minutely to catalogue all of these, I cannot

here overlook a seemingly minor deformity which results from the muscular atrophy characteristic of this disease. I refer to the undue prominence of the articular extremities of the bones in cases in which the muscular atrophy has become extreme. This deformity is so marked in some cases as to lead the patients to believe that the joints are dislocated or are the seat of osteophytic growths. Although this undue prominence of the extremities of the bones does not interfere with locomotion, yet it is of great importance in the treatment of these cases; for a careless application of rigid orthopedic apparatus often results in the formation of callosities over these bony surfaces which are painful to the patient, and when once formed are difficult of removal.

The second group of deformities which result from this disease are by far the most important; they are the deformities depending upon the paralysis of muscles. Inasmuch as individual cases differ greatly as to the extent of the paralysis and the number of muscles involved, so also the variety of deformities resulting from infantile paralysis includes almost every form of inability and perverted motor activity. But before considering these deformities it will be proper to consider the laws upon which their production depends. This subject has been studied by Volkmann in the first of the "Klinische Vorträge." Although inability is the immediate result of paralysis, deformity is not the consequence of the paralysis itself, and does not occur until a later stage of the affection is reached, when it is found that permanent contractures have taken place, of which the paralyzed muscles are generally, but by no means exclusively, the seat. As to the cause of these contractures which so deform the already paralyzed limb, many explanations have been advanced. The oldest and most popular is that of unopposed muscular action. This explanation rests upon the theory that all muscles in their quiescent state are the seat of a reflex tonicity. That is to say, it is supposed that the higher centres of the brain and spinal cord are constantly sending out innumerable and minute impulses in response to constant and unconscious peripheral excitation. This constant state of contractility is known as the reflex tonicity of the muscles, and, being constantly present in all of the voluntary muscles, the parts moved by them are kept at rest except when the will or reflex causes throw excess of energy into any single group. The principle is similar to that by which the heavenly bodies are enabled to retain their places in the universe, although constantly acted upon by centrifugal and centripetal forces.

Reasoning from this assumption, it is supposed that as soon as any group of muscles is cut off from the motor centres in the cord, the opponents, being still the seat of the normal muscular tonus, undergo contracture because the equilibrium has been destroyed. Were such the explanation, however, we should expect immediately after the paralysis to see the remaining healthy muscles become permanently contractured. Volkmann did great service in combating this theory, although he has gone to the opposite extreme and proposed one almost equally untenable. He found that in children with infantile paralysis of all of the muscles of the lower extremity, in which the muscular groups on the anterior as well as those on the posterior portion, or calf of the leg, were paralyzed, the tendo Achillis was the seat of contracture, and the foot assumed the equinus position, just as though the anterior tibial group alone had been paralyzed and the calf muscles had remained intact. Again, in cases of talipes equinus, immediately after the performance of tenotomy of the tendo Achillis, if the child were placed in a chair with the legs pendent, the foot, instead of assuming the calcaneus position, in obedience to the unopposed action of the anterior tibial group, is found to place itself in the equinus position; this is explained by the action of the force of gravity upon the foot. On the strength of both of these observations he explains the production of the deformity in many cases by the action of the force of gravity, claiming that the position which this force compels the paralyzed limb to assume, as the patient sits or stands, will be the position in which contractures will most frequently fix the limb. But on consideration we find that, even in the extremity on which the force of gravity is most influential, namely, the foot, this theory is not substantiated; for which one of us has not seen an acquired talipes calcaneus with toes raised and heels bulbous and boring the ground? The study of a recent case of facial paralysis will help to clear up this subject.

It will be found that, after the paralysis has occurred, as soon as the patient has made the first use of the non-paralyzed side, the face is slightly drawn to the healthy side. I say slightly, not so completely as it would necessarily be if the cause of the deviation were the unopposed action of the healthy side, but just enough to make it noticeable. This deviation, I think, can be explained in the following manner: The first facial motor impulse that occurred immediately after the paralysis had taken place found only the healthy side of the face ready to act. It contracted, and as soon as the motor impulse ceased the tissues returned to their relaxed condition; this passive relaxation is almost, if not quite, sufficient to restore the tissues to the position they occupied before contraction. The residual work necessary to complete the restoration the opposing muscles do for each other, and since in our supposititious case the opponent was paralyzed, the healthy muscles had almost, but not quite, returned to their normal position. In other words, the origin and insertion of the muscles have been brought slightly nearer to each other than they should be normally in the condition of rest with increased activity. After a time the shortening will become more marked, and the muscle will assume the contractured appearance so familiar in deformities following long-standing paralysis. The paralyzed muscles, in order to adapt themselves to the contracture and shortening of the healthy muscles, become passively stretched, for it is evident that every time the activity of the healthy muscles is called into play the paralyzed opponent is stretched and becomes longer, just as the healthy muscle becomes shorter. The contracture or shortening of the healthy muscles, which is primarily the result of the use of the healthy muscles, is increased by, and at the same time the cause of, the elongation which the paralyzed muscles undergo. I conceive that the only contractures which are properly the result of the action of gravity are those in which all the muscles of the limb are paralyzed, and in these cases there is no question of the non-applicability of the theory of muscular tonicity. There is another form of contracture of which the paralyzed muscles themselves are sometimes the seat; this is the result of the final stage of atrophy which the paralyzed muscle undergoes. Then the muscular fibres disappear, the muscles proper being replaced by connective-tissue bands; these contract, and when such contractures occur they counteract other previous contractures of the non-paralyzed muscles.

It follows, then, that paralysis of muscles causes deformity in one of two ways: first, by mere inability resulting from the paralysis; second, by the resulting contractures in the healthy or paralyzed muscles.

Deformities of the Lower Extremities.—Extensive paralysis, involving all of the muscles of the lower extremity, will produce a deformity well described as "flail-like." Here the lower extremity is so flaccid, and the patient has so little command over it, that in walking (with crutches) it swings as loosely as a flail; and yet, however complete the paralysis may be, careful examination will show here and there a muscular group of which the patient has retained the use. The psoas magnus is frequently preserved, so that the thigh can be flexed slightly, although the leg cannot be extended. Another very common deformity at the hip-joint is inability to extend the thigh because of a contractured psoas. In these cases the hip-joint cannot be extended even to a right line with the body; in cases in which the rectus femoris and tensor vaginæ femoris are preserved this deformity is increased in degree. The prominent

tensor vaginæ femoris appears to be so important a factor in the production of the deformity that the effect of the contractured psoas is overlooked. Hence disappointment in the operative and mechanical treatment. When deformity occurs at the knee-joint it is generally due to contracture of the outer and inner hamstring tendons. In these cases the muscles which extend the knee are paralyzed, the ligaments of the knee-joint are weakened by disease, and hence, when the patient attempts to rest the weight of the body upon the knee-joint, it closes up like a jack-knife. When the muscles on both the extensor and flexor side of the thigh are paralyzed, it is impossible to use the knee-joint in standing or walking, and the inability is almost as great as in flail leg. The most common seat of the deformities resulting from this disease is undoubtedly the ankle and mediotarsal joints; in other words, the joints involved in the various forms of club-foot. The forms of paralytic club-foot conform more or less closely to the classical varieties. The order of their frequency is, in my experience, as follows: (1) talipes equino-varus; (2) talipes calcaneo-valgus; (3) talipes equinus; (4) talipes calcaneus.

Paralysis of both the anterior and posterior calf muscles is apt to result in the equino-varus deformity, that being the position in which the foot is for the most part held. When the anterior tibial group alone is paralyzed, the deformity is apt to be equinus or equino-varus, for reasons already stated. Paralysis of the peronei alone will result in varus. Talipes equinus and equino-varus are often accompanied by contraction of the plantar fascia. This is particularly apt to be the case if the tibialis anticus is preserved, while the rest of the anterior group is paralyzed. Occasionally one or two divisions of the extensor communis digitorum remain normal, the rest being paralzyed; the result is contracture of the preserved tendon, and hyperextension of the first phalanx of the toes supplied by these tendons. In such cases the shoe of the patient presses upon these prominent phalanges, and produces excoriations, thickening, and bunions, which can often be cured only by tenotomy. When the interossei are paralyzed we have a condition analogous to "main en griffe" of the hand, the first phalanx and all of the toes being extended and the ball of the foot prominent. Dr. N. M. Shaffer has described, under the head of "Non-deforming Club-Foot," a condition which may be characterized as an incomplete talipes equino-varus; this condition often results from infantile paralysis.

Paralysis of the posterior tibial group, including the gastrocnemius and soleus, will result in calcaneus or calcaneo-valgus, contracture taking place in the anterior group owing to the use of the healthy muscles in a limited arc. These patients walk with the toes raised and the heel on the ground; this pressure on the heel results in the formation of incurable callosities of the skin, and inflamed bursæ over the os calcis. Deformities of the upper extremities, although far less important than those of the lower extremities, are equally important when we take into consideration the disability which they cause. I have already referred to the inability caused by paralysis of the muscles about the shoulder-joint and the laxity of the ligaments in these cases. But, in addition to the disability, the pectoral muscles and latissimus dorsi become shortened, owing to paralysis of the deltoid, and the arm is held firmly adducted.

Contractures occasionally are found at the wrist and elbow-joints, and their production is explicable on the principle already discussed.

In the trunk the most important deformity that can occur is lateral curvature. Not the form due to the shortening of one of the lower extremities, but a paralytic curvature—that is to say, one due to paralysis of the large or extrinsic muscles of the back, or the deep intrinsic muscles of the vertebræ. When the large muscles are paralyzed the corresponding healthy muscles of the opposite side become shorter and cause a bending of the spinal column, composed as it is of flexible segments, to the healthy side, the convexity of the curve looking toward the paralyzed side. If the curve be low down, a secondary or compensatory curve will appear high up. If the primary curve be high up, no secondary curve will be present. This is the result of the necessities of equilibrium. When the deep or intrinsic muscles of the spine are paralyzed, we have, as a result, rotatory lateral curvature, the most deforming condition the orthopedic surgeon has to deal with. The limits of this paper will not admit of the discussion of the cause of the so-called idiopathic cases of lateral curvature; but of the fact that a large number of cases of lateral curvature are the result of infantile spinal paralysis there is no doubt.

Diagnosis of Muscles Affected.—In examining a case of infantile spinal paralysis for the purpose of discovering what muscles are affected, I adopt a plan somewhat as follows: If the patient is able to walk, he should be made to do so while undressed; the movements of the various joints should then be noted and compared with those of the healthy side; any dragging, inversion, or eversion of the leg should be observed. The patient should then assume the recumbent position, and each muscle in turn must be tested as to its activity, beginning with the psoas; the leg may be raised, and the patient requested to maintain it in the raised position; or, if he is intelligent enough, the patient may be required to perform the action which will bring out the muscle under examination. In infants we are aided in this examination by a condition peculiar to them, by which the limb can be placed in any desired position, and will be retained there if the necessary muscular power is preserved, only gradually falling back to the relaxed state. This peculiarity has been compared to a similar condition present in patients under the influence of catalepsy. Finally, the patient should be examined by the most reliable and accurate test of infantile spinal paralysis, namely, the electric current. In this examination we make use of both the galvanic and faradic currents. We test the degree and kind of excitability of the nerves and muscles to both currents, and compare the result with similar tests made upon corresponding healthy muscles. In speaking of the pathology of this disease it will be remembered that the changes which the nerves and muscles undergo were thoroughly discussed. These changes were of the kind known as degenerative changes. They are not the result of all forms of paralysis, but occur only in those cases in which the lesion which is the cause of the paralysis cuts off the connection between the gray matter (anterior horns in the spinal cord, bulbar nuclei in the case of cranial nerves) and the nerves originating therefrom and the muscles supplied by them. The gray matter exerts upon the nerves and muscles what is known as a trophic influence, and it is the loss of this rather mysterious trophic influence which results in the degeneration of the nerves and muscles thus deprived. Paralysis alone does not cause degenerative atrophy, as is shown in cases of cerebral paralysis, in which the nerves and muscles, although deprived of motor power, still retain their connection with the so-called trophic centres in the gray horns of the cord.

Muscles and nerves which are the seat of degenerative atrophy give rise to peculiar electric reactions, which serve to distinguish muscles paralyzed by anterior-horn or nerve-trunk lesions. If nerves and muscles paralyzed by infantile spinal paralysis are examined five weeks or more after the paralysis has occurred, it will be found that the farado-nervous excitability is very much diminished or, later on, entirely absent. Farado-muscular excitability disappears a little later than that of the nerves. Faradic contractility of muscles is in reality the expression of the faradic contractility of the nerves which ramify throughout the muscle, so that, as soon as the faradic contractility of the nerves is lost, that of the muscles supplied by the nerves is also lost, even if the former are not yet advanced in degenerative changes.

The galvanic current yields even more striking changes. When such a current is passed through a normal nerve the muscles supplied by the nerve will contract. This contraction will be greater at the closing of the negative pole than at the *closing* of the positive pole; or, expressed in formula, this reaction will be K.C.C. > A.C.C.

The *opening* contraction will be A.O.C. > K.O.C.

In a nerve paralyzed by anterior poliomyelitis these reactions will become

K.C.C. < A.C.C., or
K.C.C. = A.C.C.

The opening contraction, if present at all, will also be reversed, A.O.C. < K.O.C.

The contraction will also be altered modally; that is to say, while the normal reaction is quick and sharp, the degenerative reaction is slow and wavering. All of these changes are known as qualitative changes; the former serial, the latter modal qualitative changes.

There is, furthermore, a quantitative change. That is to say, while a certain strength of galvanic current may be needed to produce a certain contraction in a normal muscle, a much stronger current will be needed to produce a similar reaction in the degenerated nerve or muscle. What has been said of the galvanic reaction in the nerves applies also to the muscles supplied by them.

These qualitative and quantitative galvanic changes, together with the diminution or absence of faradic contractility, make up what is known as the reaction of degeneration, and it is a most valuable diagnostic agent for the recognition of muscles affected by infantile spinal paralysis.

Differential Diagnosis.—This disease is so frequent in infancy, and has been so well studied in the past few years, that, in any given case of paralysis in children, the physician, as a rule, thinks of poliomyelitis as a possible condition. It is fortunate that we have in the symptomatology of this disease much that is positive and characteristic, so that it is possible to arrive at a positive diagnosis in almost every case.

We have to distinguish this affection in children from (1) other forms of paralysis, whether cerebral, spinal, or peripheral in their pathology; (2) paralysis following acute disease, such as diphtheria, meningitis (cerebro-spinal), etc.; (3) paralysis due to toxic action of poisons, such as arsenic; (4) pseudoparalysis of rickets and marasmus; (5) deformities of congenital origin, such as club-foot and congenital dislocation of the hip; (6) atrophy and disability due to inflammatory joint disease.

Patients paralyzed from cerebral lesions present the following points of difference from those suffering from this disease: First, the history of the onset of the paralysis is different. It is that of apoplexy or of embolism occurring rapidly, accompanied by repeated convulsions, and, above all, there is the fact that the face and tongue, as well as the limbs, are affected. On examination we find a paralysis corresponding to this history; we may have (with right-sided paralysis) aphasia in older children. There is frequently loss or diminution of sensibility, the tendon reflexes are exaggerated, while in anterior myelitis these are either absent or diminished in the tendons of the paralyzed muscles, and clonus may be present. Even if the muscles of the face have recovered, these symptoms will still distinguish cerebral from spinal paralysis. Furthermore, the limbs are well nourished and not atrophied, the surface temperature is not diminished, and the contractures, if existing, are spastic in variety and present in limbs which have not lost contour through atrophy. What is most important, however, are the results obtained from an electrical examination. In these cerebral cases faradism gives good reactions in the paralyzed muscles, while galvanism gives equally good reactions, the formulas being unchanged either in quantity or in quality. On the other hand, in cases of poliomyelitis anterior we have loss of faradic contractility, while galvanism shows the reaction of degeneration in the paralyzed muscles. The electrical examination, together with the history, should certainly yield sufficient data for a positive diagnosis. It is furthermore found that children paralyzed from brain lesions are apt to show a diminution of mental power, which is never present in the patient suffering from poliomyelitis.

Diseases of the spinal cord resulting in paralysis affect directly or indirectly either the anterior gray horns, together with other portions of the cord, or do not affect these horns at all, as, for instance, in secondary sclerosis of the lateral columns. Of the first class we have transverse myelitis, compression myelitis of Pott's disease, and most probably progressive muscular atrophy, and pseudo-hypertrophic paralysis. Here also belong cases of hemorrhage into the spinal cord. Here electrical examination, although of some, is not of so great utility as in the differentiation from cerebral paralysis. In transverse myelitis we have a paralysis of sensation as well as of motion. There is paralysis of the sphincters, there is the appearance of bedsores, while the history shows no regression of the paralysis, and the presence of long-continued fever. In the myelitis, or rather meningitis with myelitis as a secondary affection, of Pott's disease, the anterior horns of gray matter may or may not be involved, giving rise to various forms of paralysis. When the anterior horns are involved we shall have paralysis (paraplegia) it is true, but we shall also have the symptoms of vertebral caries, reflexes not abolished, but often increased, and paralysis of the bladder. Faradic contractility is generally preserved, although when the affection of the cord is very extensive it may gradually disappear. Progressive muscular atrophy is not frequent in children, and when it does occur the gradual and progressive nature of the paralysis serves to distinguish it from poliomyelitis. There is also a peculiar "cut-out appearance" characteristic of the atrophied muscles in progressive muscular atrophy, the atrophied portions being at first isolated and sharply bounded by well-developed muscles. In pseudohypertrophy of the muscles the gradual hypertrophy which accompanies the paralysis is sufficient to distinguish this disease. In the last stages of atrophy in cases of infantile paralysis, as we have already stated, there may be a deposit of fat in the atrophied muscles, but this occurs so rarely and so long after the paralysis has set in, that the history also will be a certain guide. Hemorrhage into the anterior horns of the spinal cord, when affecting these portions of the cord alone, can with difficulty be differentiated; when it affects other portions of the cord the corresponding symptoms will be sufficient to localize the lesion.

Of diseases of the spinal cord which do not affect the anterior gray horns, the most commonly met with in children is spastic paralysis (Erb) or tetanoid pseudo-paralysis (Seguin). These cases were formerly classed with those of infantile spinal paralysis, although the two diseases have scarcely a symptom in common. What has already been said of the differential diagnosis from cerebral paralysis will also apply here, the cord lesion in both cases being a sclerosis of the crossed pyramidal tracts. We have then, here, the absence of the degeneration reaction to both currents, the increased reflexes, the spastic contractures, the absence of atrophy, and sometimes diminished intelligence.

In cases of peripheral paralysis the physical symptoms, those obtained both by the electric currents and by examination, are exactly similar to those present in infantile spinal paralysis. The only important point of difference is that sensation, as well as motion, is apt to be involved in the paralysis of the affected nerve. When the patient is too young for satisfactory tests of sensation to be made, the diagnosis is extremely difficult.

In diphtheria we have the history of the disease (if the diphtheria has not escaped the attention of the patient's friends), the involvement of the muscles of the palate and throat generally, and finally the rapid recovery. We may also have ataxia and paralysis of the eye muscles. The faradic contractility is preserved.

From the paralysis of cerebro-spinal meningitis, the history and what has already been said of cerebral and spinal paralysis will serve to make the diagnosis.

Arsenic and other metallic poisons as a cause of paralysis in children must be extremely rare. I have never seen a case. E. C. Seguin has described several cases in