When they occur after the disease has lasted a long time, they often heal quite rapidly under suitable treatment. Mild cystitis is so common in myelitis that it can hardly be regarded as aggravating the prognosis; but in severe cases, especially if the bladder is completely paralyzed, there is always danger of the production of acute interstitial nephritis. The latter affection is almost inevitably fatal at an early period, though in some cases it seems to pass into a stage of quiescence.

The prognosis as regards recovery is also so much less favorable the greater the degree of implication of the gray matter, as evidenced by rapid atrophy of the paralyzed muscles and by the presence of the reaction of degeneration in these parts. But it must also be remembered that even the most marked atrophy of the muscles and complete abolition of their electrical irritability do not entirely preclude the possibility of a very satisfactory degree of recovery. I have had under observation a patient in whom the limbs were completely paralyzed and wasted to the extreme, and the reaction of degeneration was present except in those muscles in which no reaction could be obtained (even the intercostals were partly paralyzed); and yet this patient, at the end of a year and a half, was able to walk about and to perform her duties in such a satisfactory manner that she requested her discharge from the hospital.

The occurrence of a spastic condition of the lower limbs constitutes an almost insuperable obstacle to any further improvement. The degree of motor power which is acquired in these cases, however, is usually quite considerable, and the patients are often able to move about quite freely, unless the rigidity of the limbs is excessive.

Pott's paraplegia generally offers a favorable prognosis, unless the vertebral caries itself, or its sequelæ, constitute a serious menace to life. Improvement in apparently hopeless cases sometimes comes on quite suddenly, and the patient may recover even from a number of severe attacks. In these cases there is always danger of a relapse so long as the tendon reflexes remain exaggerated.

The prognosis of chronic myelitis is always grave. Even chronic compression myelitis, which, of the different forms of the disease, presents the most favorable outlook, rarely terminates in recovery. Unless the slow course of the spinal inflammation is interrupted by acute exacerbations, the patient may linger for years. The longest lease of life is enjoyed in those cases of transverse myelitis in which the disease does not extend up the cord. The greatest danger in these cases arises from the development of cystitis and secondary interstitial nephritis.

In the syphilitic forms of chronic myelitis we are sometimes able to secure a fair degree of recovery, especially if treatment is begun before the disease has extended across the entire section of the cord. But even here it will be found that complete recovery rarely takes place.

It is held by many writers that cases of railway spine, which is probably due to chronic myelitis and meningomyelitis, usually recover; but this statement does not agree with the experience of the majority of unbiassed observers.

Treatment.—The most important indication is to secure as absolute rest in bed as is possible under the circumstances. As soon as any symptoms which suggest the onset of myelitis make their appearance, the patient should take to bed and remain in the recumbent position. The wants of nature should be attended to with the aid of the bedpan, in order to avoid, as much as possible, movement during the acts of urination and defecation. Rest is, in my opinion, by far the most important remedial agent at our command. It is often difficult to determine at what period it is best to allow the patient to begin to exercise the paralyzed limbs, but it is always better to err on the safe side. I have seen very marked improvement in a number of apparently hopeless cases in which this plan had been faithfully carried out for a period of a year to a year and a half.

Contracture of the tendo Achillis is a very common sequel of all forms of myelitis, and it has been suggested that the pressure of the bedclothes upon the feet may exercise some influence in the production of this unfortunate symptom. In order to obviate this, the bedclothes may be suspended on a barrel hoop placed across the lower part of the bed. In a considerable number of cases, however, the contracture will develop despite this precaution.

Next to rest, cleanliness is all-important, because the neglect of cleanliness is very apt to result in the production of bedsores. This usually necessitates the most careful nursing. The external genitalia and gluteal regions must be frequently washed, and thoroughly but gently dried. At the slightest indication of the development of a bedsore, an air-ring should be placed under the buttocks, care being taken that the joints of the ring are not sharp. If the evidences of dermatitis are not relieved speedily in this way, the patient should be placed on a water-bed.

The bladder must be carefully looked after. Catheterization must be resorted to at once, as soon as the bladder is no longer thoroughly emptied. The greatest precautions must be taken to keep the catheter thoroughly aseptic in order to prevent the introduction of germs. Even a soft-rubber catheter must be introduced very gently, because the mucous membrane of the bladder is very apt to be congested, and bleeds readily under such conditions.

Counter-irritation is useful in very many cases. During the first stages of an acute myelitis the application of dry cups along the spinal column may be attended with some benefit. Vigorous counter-irritation with fly blisters is indicated at a later period, but these do not promise much improvement after the disease has become stationary. In this connection I may mention that in a case of chronic myelitis in which all hope of recovery had long been abandoned, I noticed that a remarkable degree of permanent improvement followed the development of a large carbuncle on the back.

The actual cautery is also recommended. I have never seen any benefit from its use, except in rare cases of compression-myelitis. In such cases, however, it sometimes acts with remarkable rapidity.

Among other non-medicinal agents may be mentioned baths and electricity.

As a rule, the patient cannot tolerate extremes of temperature. I usually recommend a sitz bath at a temperature of 100° to 110° F., in which the patient remains for from fifteen minutes to half an hour. If spastic phenomena are well marked, the bath may be as hot as can be borne by the patient. When the spastic phenomena are not prominent, the patient may receive a full bath at a somewhat lower temperature. It is well in such cases to follow the bath with rubbing and massage.

Electricity, when faithfully and persistently applied, is often productive of excellent results. It is employed either as a spinal current or directly to the paralyzed or contractured parts. When applied to the spine one large electrode should be placed over the site of disease, the other on some indifferent part of the body or spine, and they should be held constantly in position for from three to five minutes. The direction of the current seems to be immaterial. Much better results are obtained from applying the current directly to the paralyzed parts. In the majority of cases the galvanic current is preferable, but the faradic current may be used when the muscular contractility remains normal. The current should be merely strong enough to produce visible contraction of the paralyzed parts. The constant stabile galvanic current is used in the treatment of contractures, the electrodes being applied directly over the nerves supplying the contracted muscles. At the same time the interrupted galvanic or faradic current may be applied to the antagonists. In my own experience very little, if any, benefit has been derived from spinal galvanization; but, on the other hand, I have obtained most excellent results from the patient, persistent use of local electrization.

This even holds true of cases of marked contractures of the lower limbs, but a good deal will then be gained by the electrical treatment combined with tenotomy of the contractured parts.

Medicinal treatment does not play a great part in the management of this disease. Ergot has been highly recommended in the acute stages, and hypodermic injections of ergotine have been employed at the onset of hemorrhagic myelitis. I have never seen the slightest advantage from its use, and have abandoned it altogether. Iodide of potassium has also been recommended very highly, and Dr. Gibney speaks in the most favorable terms of its administration in large doses (even ʒ i.–ij., t.i.d., in children), in the compression myelitis of Pott's disease. It is very difficult to form an opinion upon the value of this agent in myelitis, though it has seemed to me to exercise a certain amount of beneficial effect. But I have employed it so faithfully, and with such little effect, in Pott's paraplegia, that I am compelled to deny any special influence in this form of the disease. Weir Mitchell has recommended suspension in this disease, but further experience has not shown that this measure possesses much value.

Strychnine is the only medicinal agent which has seemed in my hands to have a decided influence on the course of the disease. This drug may be administered as soon as the acute symptoms have ceased to advance, and it is by no means contraindicated by the occurrence of spastic symptoms. The dose may be raised gradually from gr. $\frac{1}{48}$ to gr. $\frac{1}{32}$, t.i.d., and may be given continuously for months.

So long as any improvement is observed, the patient should be kept as quiet as possible. Later, gentle exercise may be allowed, but the patient must not be permitted to tire himself. Sexual intercourse is particularly to be avoided. Even after the condition of the patient has been stationary for a long time, benefit is sometimes derived from another course of treatment with electricity, counter-irritation, and strychnine.

Leopold Putzel.

SPINAL-CORD DISEASES: POLIOMYELITIS ANTERIOR, ACUTE AND SUBACUTE.—Under this name we recognize a form of paralysis which occurs, as the name implies, most frequently in young children, especially during the period of first dentition. It is by far the most common form of paralysis to which children are subject and presents a well-marked clinical picture. A healthy child is suddenly found to have lost the use of one or more extremities, or of some individual muscle of an extremity. This loss of power may or may not have been preceded or accompanied by febrile disturbance; occasionally convulsions have ushered in the paralysis, but never is the latter accompanied by loss of sensibility. Any of the voluntary muscles of the limbs or trunk may be affected, but the functions of the bladder and rectum are left undisturbed. Such is a general description of the onset of the disease. Soon, however, many of the affected muscles begin to recover their power; this recovery may be almost complete, the paralysis being finally limited to a few individual muscles; or, on the other hand, the greater part of the muscles originally affected may remain paralyzed, and later on undergo atrophy and degeneration. In this atrophy and retardation of growth the bones take part, giving rise to shortening of the affected extremity, while other deformities occur as a result of the unopposed action of the healthy antagonists of the disabled muscles.

Synonyms are numerous, and owe their variety to the views held by different observers as to the nature of the disease, before its pathology had been established. They are: Infantile spinal paralysis; Spinale Kinderlähmung (Jacob von Heine); Paralysie spinale; Paralysis infantilis spinalis; Infantile paralysis; Kinderlähmung; Paralysie infantile; Paralysie des petits enfants; Essential idiopathic or functional paralysis of children; Paralysie essentielle de l'enfance (Rilliet and Barthez); Essentielle Kinderlähmung; Dental paralysis (Underwood, 1784); Poliomyelitis anterior acuta; Acute inflammation of the anterior gray horns of the spinal cord (Kussmaul); Tephromyélite antérieure aiguë (Charcot); Acute atrophic spinal paralysis; Paralysie atrophique graisseuse de l'enfance (Duchenne); Regressive paralysis (Barlow); Myelitis of the anterior horns (E. C. Seguin). These various terms were intended by their originators to indicate what appeared to be prominent clinical or pathological characteristics of the disease. Without entering upon the question as to the relative value of a clinical, as compared with a pathological, designation of diseases, there is no doubt that, of all the terms applied to the affection, the best of the first class is that adopted by Heine (Spinale Kinderlähmung, or infantile spinal paralysis). It is true that here even the word spinal would indicate, in a general way, the anatomical location of the lesion which is the cause of the clinical manifestations; but, inasmuch as it serves to distinguish this form of paralysis from other paralyses of cerebral origin, we can pardon this slight offence against unity in terminology in consideration of the completeness with which it serves to distinguish the disease. Of the purely pathological terms we should prefer poliomyelitis anterior acuta, as indicating the seat of the lesion, the term acute being especially applicable to the disease as it occurs in children. Seguin's myelitis of the anterior horns purposely omits the term "acute," in order to include the subacute and chronic cases of the disease which sometimes occur in adults. The designation essential or idiopathic paralysis can certainly no longer be applied to a condition which has so well defined a pathology as poliomyelitis anterior; and it seems inexcusable that so excellent a pathologist as Niemeyer should have retained this inaccurate term in the seventh edition of his work, published in 1867, at a time when the gross lesion had already been demonstrated.

Historical Account.—This disease has in all probability affected children for hundreds of years, without being differentiated by medical writers from other forms of paralysis. As early as 1784, however, Michael Underwood[1] described a form of paraplegia which occurred in teething children, and especially in those suffering from bowel troubles, which in its clinical history resembles the disease now known as infantile spinal paralysis. In 1836 John Badham,[2] of London, described a similar affection occurring in children, which he considered cerebral in origin. Notwithstanding these publications, the condition did not receive general clinical recognition until, in the publication of J. von Heine's[3] great monograph, entitled "Beobachtungen über Lähmungszustände der unteren Extremitäten und deren Behandlung," 1840, the symptom group of the affection was clearly defined and this form of paralysis distinguished from other forms of paralysis occurring in children. The picture of the affection in question, which Heine gives us in the very first edition of his work, is so accurate in detail that later clinicians had but little to add to the symptomatology of the disease. But, although no objective or subjective manifestation of the disease escaped the notice of this accurate observer, yet he remained ignorant as to the true pathogenesis of the affection. So logical a mind, however, could not but suspect that the disease was the result of an actual lesion of the nervous system, and accordingly we find him indicating the spinal cord as the most probable seat of the lesion. In the second revised edition of his work[4] he terms the disease Spinale Kinderlähmung, or infantile spinal paralysis. Duchenne, de Boulogne,[5] in 1855, besides accurately describing the affection, took a great stride in the direction of precision in diagnosis as to the character and extent of the paralysis, by discovering the relation which the faradic current bears to muscles which are the seat of this paralysis. Here, then, the first stage in the history of the disease may be said to have been completed. But, although clinically the affection was established upon a firm basis, its pathology was yet unknown. Rilliet[6] and Barthez,[7] basing their statement upon negative findings in cases of this disease upon the autopsy table, considered the affec-

tion primarily a muscular one, and hence termed it essential paralysis of children, the term essential referring to the idiopathic or non-organic nature of the paralysis. Although this view gained many followers, and some of eminence, yet it was not long before positive findings upon the autopsy table exploded this theory as to the pathogenesis of the disease. In 1863 von Reinecker and von Recklinghausen [8] published the autopsy of a case in which both lower extremities had been the seat of infantile spinal paralysis. Upon a macroscopic and minute examination of the cord it was found that the ganglion cells of the anterior gray horns and the nerve fibres of the antero-lateral columns of that portion of the cord which gave origin to the nerve fibres supplying the paralyzed extremities had undergone atrophy and degeneration (*Deutsche Klinik*, January 31st, 1863). The accuracy of these pathological data was confirmed by similar findings by Cornil,[9] Bouvier and Laborde,[10] Prevost,[11] Lockhart Clarke,[12] and later on by Charcot [13] and Joffroy,[14] Vulpian,[15] Roger and Damaschino,[16] and others.

We cannot close this brief historical account of the affection without reference to the studies of Erb,[17] as to the reactions produced by the galvanic current upon the muscles which are the seat of this spinal paralysis. Finally, we should refer to the identity of this affection, as far as the pathology is concerned, to a similar but more chronic affection, occurring in adults, which was first mentioned by Duchenne fils [5] in 1872, but not thoroughly studied until E. C. Seguin,[18] in 1877, devoted an admirable monograph to the subject. In the last few years many writers have sought to ascribe an infectious character to this disease. This has been based not so much upon bacteriological or pathological findings, but upon the fact that the disease has been found to occur in occasional epidemics, one specially reported by Medin.

Other observers, like Marie, Strümpell, Seligmüller, have reported smaller epidemics. Some have reported the occurrence of the disease in several members of the same family.

Pathology.—The anatomical seat of the primary lesion of this form of paralysis is the spinal cord. This has been positively determined by numerous autopsies. It consists of an inflammatory process, acute in character, affecting in the first instance the anterior horns of gray matter. The lesion is at first diffuse, but as soon as the acuteness of the inflammatory process subsides it becomes limited in its longitudinal extent to circumscribed portions of the cord, in which even at the outset the inflammation had been most intense; this is most apt to be about the cervical and lumbar enlargements. At these points further changes soon occur, the most important of which are destruction and atrophy of the large multipolar ganglion cells situated in the anterior cornua.

This destruction and atrophy are not limited to the cells, but involve the nerve fibres in this region, and even the cells and nerve fibres of the posterior horns may become involved; sometimes the lesion extends into the white substance of the antero-lateral columns. As a result of the primary lesion in the ganglionic cells of the anterior gray cornua, we have secondary changes taking place in the nerves which take their origin from the affected portion of the cord, and in the muscles to which these nerves are distributed. These changes are atrophic and degenerative in character.

Spinal Cord.—I know of no autopsies showing the condition of the spinal cord during the acute stage of infantile paralysis, inasmuch as the disease of itself is rarely, if ever, fatal. We can therefore make no positive statement as to the condition of the nerve centres during this acute stage; we can conclude, however, from our findings later on, that the disease is, in the first instance, an acute parenchymatous inflammation affecting chiefly the ganglion cells of the anterior horns of gray matter in the cord (Charcot).[19] When the autopsy has been made within two years after the onset of the disease, but slight changes can be observed in the cord by the naked eye, although the microscope reveals decided and constant alterations. These changes are typically shown in a case examined by Dr. Fred. Taylor, the particulars of which were presented to the London Pathological Society. It was that of a girl, three years of age, who suddenly became paralyzed, at the age of fifteen months, in the left leg. The paralysis was supposed by the friends to be the result of a blow; the principal symptoms observed at the time of the onset were fever and pain in the left lower extremity, followed by paralysis, and, later on, the development in the paralyzed member of the ordinary clinical phenomena of infantile spinal paralysis. The child was treated by galvanism for five or six months, and the paralysis somewhat improved; but at the age of three years death resulted from bronchopneumonia. Post-mortem examination of the spinal cord showed a diminution in the size of the transverse section of the left half of the lumbar region of the cord. This diminution was most marked anteriorly, although also observable in the left posteror horn of gray matter. The anterior roots of the spinal nerves at this level were smaller than the corresponding roots on the right side. The posterior nerve roots were unaffected. On microscopical examination of a section taken from this portion of the cord it was found that there were but few ganglion cells in the left anterior cornu, and even those remaining were ill-defined and smaller in size, paler in color, and possessing fewer and shorter processes than those of the right side. The absence of these cells was most marked in the external and median groups of ganglion cells.

The nerve fibres passing from the anterior root to the cornu of the affected side were diminished in number and size. The main substance of the anterior horn consisted of a very dense and felt-like tissue, made up of matted fibres, "an increase and condensation of the normally open and spongy basis-structure of the part." In this felt-like structure the remaining nerve cells were embedded; no granular corpuscles were visible. The white matter of the left antero-lateral column was found denser, the connective tissue being increased; while the nerve tubules were smaller than usual and deficient in axis cylinders.

This case shows the most prominent changes found in the cord in cases in which the autopsy has been made comparatively early. Occasionally areas of softening are found, more or less well defined, in different portions of the anterior horns, and at such places there is generally a total absence of proper nerve tissue, ganglion cells, and nerve fibres; but instead there is a low form of connective tissue, filled with large numbers of leucocytes and simple nuclei. In these recent cases the blood-vessels are found dilated. The cord may be affected at any level, although the cervical and lumbar regions are most frequently the seat of permanent lesion. The severity of the inflammatory process varies greatly, so that in some situations there is almost complete recovery, while in other places there is utter destruction, degeneration, and atrophy of nerve cells and fibres. The ganglion cells have been found in all stages of various forms of degeneration; many of them are entirely destroyed, and those that remain are found in a condition of extreme atrophy, having suffered great contraction and diminution in size, the cell processes, and frequently their nuclei, being destroyed. Sometimes the atrophy is of the pigmentary variety. In a case reported by Dr. Humphreys,[20] of Manchester, the number of the polar cells remaining in the affected portion of the anterior horns was compared with the number in the corresponding healthy portion of the cord, and it was found that while on the healthy side there were fifty-two ganglion cells, on the diseased side there were but thirteen.

Should the autopsy be made, as more frequently happens, many years after the original lesion has taken place, we would find changes similar in character to those described, but further advanced in a retrograde direction. The changes can be admirably studied in a collection of autopsies reported by Seguin in his work, and quoted and amplified by Seligmüller in his article on this subject in Gerhard's large work on "Diseases of Children." These changes are now readily appreciated

by the unaided eye. The affected portion of the gray anterior horn is apt to be markedly shrunken and atrophied; sometimes even the posterior gray horns will be found slightly smaller than on the opposite side. The white columns, and especially the antero-lateral columns, are found atrophied and sclerosed, sometimes to a limited extent on a level with the lesion, at other times to some distance above and below the lesion; indeed, the transverse section of the cord on the affected side will show a general diminution in size as compared with that of the healthy side. The anterior nerve roots at the level of the lesion are apt to present a grayish, translucent appearance and a diminution in size.

The microscope also shows the more complete atrophic changes which the nerve tissue has undergone; the diseased portion of the atrophied anterior horn is found to contain but few or no multipolar ganglion cells, and even those remaining are shrunken, with their processes atrophied, and found to have undergone various stages of granular and pigmentary degeneration. These cells have been replaced by connective tissue which has become contracted and hardened.

The changes in the white columns are apt to be less marked, although, as already stated, there is some diminution in the size of the antero-lateral column, to a greater or less extent, above and below the lesion. These changes can scarcely be termed sclerosis. They consist of a certain degree of hyperplasia and contraction of the connective tissue, and an increase of the nuclei of the neuroglia. Amyloid corpuscles are also found. The anterior nerve roots undergo a more decided atrophy than the antero-lateral columns; the number of their medullated nerve fibres is decidedly diminished. Many of them have lost their myelin, leaving only the axis cylinders, and, as a whole, the nerve root has become thinner and of a grayish and translucent appearance. The diseased foci in the gray matter are apt to be quite sharply defined, although at the onset of the disease large portions of the cord, above and below the areas of softening, are found congested; these portions, however, are not the seat of destructive changes, and they recover as soon as the inflammatory process subsides. This probably corresponds to the regression which occurs in the symptoms, thus leaving only those muscles paralyzed which derive their nerve supply from the softened areas.

All of these degenerative changes in the cord which I have described as characteristic of poliomyelitis anterior acuta take their origin in an inflammatory process in the vascular (arterial) system of the spinal cord. Such inflammatory changes are found even in the large blood-vessels of the cord, even in the walls of the arteria spinalis anterior and its branches (Socki). Manfredi, Widal and Besançon, and others have produced degenerative changes in the anterior horns of the spinal cord of animals by injections of pure cultures of various microbes. Widal was successful in producing the lesion in seven out of one hundred and seventeen rabbits which he had inoculated with streptococci. Thonot and Masselin believe that the coli bacilli can produce the lesion.

Schultz (1901), in a recent case of this disease in which the onset resembled meningitis, but the subsequent history and, months later, autopsy showed it to be a case of poliomyelitis anterior acuta, obtained by lumbar puncture the spinal fluid and isolated therefrom the meningococcus of Weichselbaum-Jaeger. The occurrence of this disease as a complication or sequel of the acute infectious diseases, such as measles and scarlet fever, would lead one to suspect that this form of inflammation of the cord, like the types of cerebro-spinal meningitis, may be the result of a mixed infection of the primary disease infection and the various pus organisms. Some observers have been led to believe that the toxins of various infections in distant parts of the body may be carried to the cord, and set in motion the inflammatory changes in the blood-vessels which result in the degenerative changes in the anterior horns of the cord.

Changes in the Paralyzed Muscles.—Within a few weeks after the paralysis has taken place the paralyzed muscles become the seat of characteristic pathological alterations; not only do they become atrophied and smaller in size, owing to disuse, but they undergo active degeneration. Portions of such muscles can be obtained for examination by means of an apparatus like Duchenne's trocar, so that the various stages of degeneration can be studied. The first stage in the degeneration is, undoubtedly, ordinary atrophy of the muscle fibres. The fibres are narrower and diminished in diameter, the transverse as well as the longitudinal striations are finer and apparently more numerous. This apparent increase in the number of the transverse striations is due to the diminished thickness of the contractile discs. These phenomena are seen in muscles that have become atrophied from disuse, and are not distinctive of atrophy due to spinal-cord lesions.

In infantile spinal paralysis, however, the muscles soon begin to show further changes. Certain portions of the degenerating muscles begin to lose their striations entirely; here and there we find contractile discs replaced by granular matter, until the whole fibre has become to a great extent granular. This granular matter is probably protein in character, being at first soluble in acetic acid and insoluble in ether; later on, however, the granules are found to be insoluble in acetic acid and soluble in ether, which shows that the protein granules have been replaced by fatty matter. Here and there will be found pigment granules. The muscle corpuscles are found to have increased in number, and in fibres which have undergone a complete granular fatty degeneration we shall find no trace of proper muscular tissue, the fibres being replaced by these muscle corpuscles embedded in a mass of fat granules surrounded by the sarcolemma. This may be termed complete muscular degeneration, and is the inevitable fate of muscles which have not recovered in the earlier stages of ordinary atrophy. Some of the muscles thus degenerated become the seat of further changes. The connective-tissue corpuscles found in the endomysium and perimysium increase largely in number, and form themselves into fibrous connective-tissue bands, which compress the degenerated muscular fibres; gradually the fat is absorbed, and what was formerly muscular tissue has become a firm fibrous cord. This change is known as cirrhosis of the muscles, and does not occur until long after the muscles have undergone hopeless degeneration. These fibrous bands are apt to undergo contracture, which results in structural shortening and deformity. Rarely the connective-tissue corpuscles, contained in the fibrous bands, in their turn become distended with fat, thus giving rise to a deceptive fulness instead of the previous atrophy. This second fatty deposit may be so great as to lead to a diagnosis of pseudohypertrophic paralysis. When such a deposit of fat does occur it is in infantile paralysis of long standing. The ligaments and bony structures also take part in the general lowering of vitality which characterizes the paralyzed limb. The bones do not grow with anything like the vigor of the corresponding bones on the healthy side. This retarded growth results in diminution in the length as well as in the thickness of the bone; the prominences, ridges, etc., are less marked; the bone is more readily bent; closer examination shows that the compact tissue is relatively diminished, while the cancellous tissue is coarser and has scattered through it a larger number of fat cells. The bones are thus softer, but microscopically no changes in the structure of the bone tissue are found. All this results in a weakening as well as a shortening of the bone, and, necessarily, of the limb. Seeligmüller considers this bony change mostly the result of the disuse which is necessarily connected with the paralyzed member; the fact, however, that these atrophic bone changes are noticed in limbs in which the paralysis is insufficient to interfere with the adequate use of the member, would militate against this view, apart from the fact that the changes indicated seem to be far more radical than would be accounted for by ordinary functional atrophy due to disuse.

Partly in consequence of this bone atrophy, and partly as a result of the relaxation of the ligaments and muscles, the joints are apt to become altered. In extreme cases the ends of the bones which form the joint are not in contact, but hang apart from each other, so that the finger can almost invaginate the skin and ligaments between the joint surfaces. As a result, the articulation admits of abnormal mobility—motion resulting in subluxation of one joint surface upon the other. These changes are most readily observed where a muscle which passes over a joint is more or less completely paralyzed; thus, when the deltoid is paralyzed in the arm, the shoulder-joint is apt to show the above-mentioned changes very markedly. No pathological changes have been observed in any other organs of the body.

Etiology.—From modern investigations we are led to believe that poliomyelitis anterior is an infectious disease occurring sporadically, although rarely also in epidemics. Whether the disease has for its causative pathological agent a specific germ or its toxin, or whether it can be produced as a result of the activity of various germs, such as the pus organisms, etc., is not yet determined. That the disease is an infection is most probable, and from this starting-point all other factors in etiology are to be viewed as subordinate. Poliomyelitis anterior acuta is no exception to the rule in the obscurity of its etiological chapter. We know little or nothing as to the causes which produce the disease. We must, therefore, satisfy ourselves with an inquiry as to the causal relation borne by certain conditions, which precede the appearance of the paralysis, to the more or less acute inflammatory process in the anterior horns of the spinal cord. Before proceeding to this study there are certain factors which can be eliminated, and thus limit the field of inquiry.

Infantile spinal paralysis is by far the most frequent form of paralysis to which children are subject. Jacob von Heine found, among 192 paralyzed children, 158 suffering from this disease.

The disease in its acute form is peculiar to childhood, and is most frequent in the first, second, and third years of infant life. Duchenne the younger has noted one case occurring at the early age of twelve days. Seeligmüller has noted the oldest of his seventy-one cases as six years, but almost ninety per cent. of all cases reported were under three years of age. Sex is not a predisposing cause, the sexes being affected with about equal frequency.

The disease is found all over the civilized world, so that climate can scarcely be considered as exerting a predisposing influence, nor does the season of the year make any difference, cases occurring indiscriminately at all seasons. Wharton Sinkler (*Amer. Journal of Med. Sciences*, April, 1878) states, however, that forty-seven out of fifty-seven cases occurred in the warm months of the year.

Heine has stated that the disease most frequently affects strong, healthy children; and, indeed, one who sees many of these cases cannot fail to have observed many large, robust little patients whose otherwise handsome physique has been deformed by an attack of poliomyelitis anterior. But, on the other hand, most observers find that the majority of cases occur in children who are weak and delicate, even if a strumous constitution cannot generally be affirmed of them.

One of the most frequent causes assigned by mothers for this disease is "catching cold," particularly exposure to the direct action of a "draught." Or another very common cause, as stated by the parents, is a fall from the arms of the nurse or from a chair. Whether a traumatism of this kind can give rise to infantile spinal paralysis proper is questionable. It is conceivable that spinal paralysis, resulting from hemorrhage into the gray matter of the cord, may follow a traumatism; but this is not poliomyelitis anterior, although the clinical symptoms may be those of the latter disease, and it may be impossible to differentiate these cases from those of inflammatory origin.[20]

The disease being one of the first dentition period, it is natural to look to conditions connected with this process for etiological factors in its production.

There is no question that the process of dentition throws the nervous system of the child into a state of exalted irritability, during which eclampsia, tetany, and other neuroses are commonly observed. These nervous phenomena are chiefly conditions of exalted nervous irritability, in which it is conceivable that the gray ganglion cells of the cord take part. From this irritated condition it is only a step to inflammation. Henry Kennedy has called this disease dental paralysis (*Dublin Quarterly Journal*, vol. ix., February and May, and vol. xxii., August and November). The restlessness of children while teething, the disturbed nutrition, want of sleep, and febrile disturbances common to this period of life, all are sufficient to render the spinal cord more sensitive to slight active causes, such as refrigeration, etc.

It may be objected, however, that all of these influences would affect the spinal cord as a whole, while in this disease only the anterior gray columns of the spinal cord are primarily affected. This might be explained by reference to the fact that motor areas of the cord in infants are much more in demand and further developed than the sensory tract. Infants are in constant motion, the extremities are thrown about whether in health or disease; during the waking hours infants constantly move and throw about the limbs and body; as a result the centres for these movements are supplied with an excess of blood, and hence are more prone to take on inflammatory processes.

Acute febrile conditions are commonly assigned as causes for this disease. It must not be forgotten, however, that the disease is in very many cases ushered in by fever, which is a symptom of the inflammation, and cannot be considered an etiological factor. But there are many cases which occur during the course of, or follow upon, the acute exanthemata—scarlet fever, measles, and typhoid fever. Such cases must be carefully distinguished from postdiphtheritic paralysis, the more so since diphtheria often complicates scarlet fever, measles, etc. Duchenne and Seeligmüller report two cases following upon vaccination.

In many of the cases careful inquiry into the family history of the patient will reveal the existence of nervous affections in other members of the same family—either paralysis, convulsions, or insanity. Thus I have seen infantile spinal paralysis of the leg in a little girl, and paralysis of the upper arm in a younger brother of the first patient.

Symptomatology.—In discussing the symptoms of this disease it is perhaps wise to take up in order the characteristics of the invasion, the appearance of the paralysis, and lastly, the deformities and contractures resulting from the paralysis.

First, the Stage of Invasion.—Although various prodromal symptoms have been laid down in the text-books as preceding the onset of the disease, yet none of these appears to me to be peculiar to this affection, even in slight degree. The children are irritable, slightly feverish, and suffer from loss of appetite; the bowels are constipated or relaxed; some of the little patients complain of pains in the back and limbs. One observer[21] has noted that these patients refuse to walk, or, if they walk, are easily made tired and wish to be carried. I myself have not noticed the latter symptom as part of the history of my cases, and have found very many cases—indeed, the large majority of my cases—in which no prodromal symptoms at all occurred. Cases may be divided, as regards the character of the invasion, into two classes: First, those in which the onset is shown by high fever, with or without convulsions; second, those in which the stage of invasion is entirely absent, the patient becoming suddenly paralyzed.

Cases belonging to the first class will be found to have either the febrile disturbance or the nervous symptoms most prominent. The patient is suddenly attacked by high fever, 100° to 102° or 103° F., rarely above; there are

great thirst and restlessness. At the beginning of this fever there may occur a convulsion lasting a short time, and often followed by others, or the fever may be slight; but the child lies in a state of semi-coma for a few days, from which it gradually rallies, when the friends find one or more groups of muscles, or one or more limbs, paralyzed. The fever may last from a few hours to two or three, or even more, days; but, as a rule, it continues only a few days, after which it subsides, and the parents believe the child to be on the road to recovery. Then suddenly paralysis is discovered to have taken place. If the paralysis is slight, it may not be noticed until the initial fever has long passed, so that the parents will assure the physician that there was no fever before the onset of the paralysis.

In cases occurring during the course of convalescence from measles or some other acute febrile affection, the paralysis is supposed by the friends to be due to the general disease.

In many of the cases, in addition to the fever, with or without convulsions or semi-coma, the patients suffer from diarrhœa and often from vomiting. Hyperæsthesia of the limbs and other portions of the body may also occur. There may be frequent micturition, but there is never paralysis of the bladder or loss of sensation.

The cause of the initial fever would appear to be the acuteness of the inflammatory process in the cord. W. Vogt,[22] however, considers the lesion in the cord a result, rather than a cause, of the fever. The fever is not a constant accompaniment of this disease; it was present in about one-half of my cases. Frequently parents do not notice the fever, which may occur at night, when the child is asleep. When the little one awakes in the morning the paralysis is observed, although the fever, if only of a few hours' duration, may have been overlooked. The antecedent fever is noted, however, in the cases in which it is of longer duration. It has been known to last for twelve days; but in most cases it is of less than two days' duration, and commonly lasts only twenty-four hours. The temperature rarely rises above 103°, or, at most, 104°, and even these figures are exceptionally high. We have no good thermometric records; the diagnosis being never possible before paralysis has set in, and the fever has, as a rule, come to an end, although cases have been reported in which the fever lasted after the paralysis was complete.

Convulsions occur, in the opening stage of this disease, in many cases in which fever is present, and also in some in which there is no rise of temperature. This would seem to prove that the convulsions are not dependent upon the cerebral hyperæmia produced by the fever. It is, furthermore, not probable that these attacks, which are epileptiform in character, are due to lesions in the spinal cord, for the muscles which become convulsed are not only those that are afterward found to be paralyzed, but impartially those of the whole body. Some writers, however, state that the convulsive movements are more violent in those muscles which afterward become paralyzed. The attacks vary in duration, number, and frequency. Frequently only a single convulsion ushers in the disease; at other times the patient is thrown into a status epilepticus lasting some hours, and it is conceivable that in this condition the patient may die, the diagnosis of poliomyelitis not having been made. On the other hand, the convulsion may be so slight as to be entirely overlooked, and, if it take place during the night, the fever as well as the convulsion may be entirely overlooked. The recovery from the convulsion may be complete, or the child may fall into a stupor, and even coma, from which it only emerges when all acute symptoms have disappeared. Frequently the little patient has no convulsions, but still is restless and trembling, or apathetic and drowsy. Either of these conditions may be present in the opening stage of the disease, with or without the fever.

Another symptom, to which we must call attention, is one which accompanies all febrile conditions, but is here particularly prominent, namely, hypersensibility—the patient cannot be touched without feeling pain—he cannot be moved without crying out. Some writers have stated that this exalted sensibility is most marked in the spinal column. My own experience does not show that the little patients ever complain of a sensitive spine. On the contrary, I have found these children very easily handled, and the spine very flexible, this fact forming one of the data for the differential diagnosis between this disease and Pott's paraplegia, although patients may have passed through the initial stage of the malady with more or less of the above symptoms, or so little of them as to escape the observation of the friends; yet the

Second, or paralytic stage, is never omitted, and is, in fact, "the disease" (Rilliet and Barthez). The paralysis may develop very suddenly, the child being paralyzed almost as rapidly as though from apoplexy, or it may develop more slowly, the paralysis gradually extending in the course of one, two, or three days. In the first class of cases a large number of the voluntary muscles of one or more limbs become paralyzed simultaneously; in the second class of cases the paralysis extends from one group to another until the maximum paralysis has been attained. Sometimes the paralysis is so complete that the patient lies without being able to move a muscle. At other times the paralysis is so slight that it is unnoticed until the function of the paralyzed member is interfered with.

However complete the paralysis at first may be, yet it is characteristic of this disease that after a short time (one or more days) many of the paralyzed muscles begin spontaneously to recover their power; this is known as the regression of the paralysis. Spontaneous recovery from the original paralysis may be complete in certain individual muscles, although entire recovery for all the muscles never occurs. Many writers state that in most cases the height of the paralysis is attained at the end of twenty-four hours. My experience leads me to give a longer period for the development of the palsy, certainly not longer than a few days, when the paralysis will cease to extend. Regression, however, begins in many cases as early as the second day, continues rapidly for the first few weeks, then much more slowly, although even up to four or five months the muscles may still undergo spontaneous recovery of power. Any power restored after that is due either to the good effects of treatment or to the use of the limb. However complete the restoration of some of the paralyzed muscles may be, it may be positively affirmed that complete recovery in all the muscles affected by infantile paralysis is impossible.

Muscles affected: All the voluntary muscles of the body may be the seat of this paralysis, except those of the head (Seeligmüller reports one case in which the muscles supplied by the facial were affected), eyes, ears, mouth, pharynx, and larynx. On the other hand, the sterno-cleido-mastoid has been found affected, the muscles of the trunk, including those of the back and abdomen, and, above all, the muscles of the extremities.

The paralysis may affect the back, so that the patient cannot sit or stand; the neck, so that the head falls forward or to the side, and it may affect one or more extremities, giving rise either to crossed paralysis (one arm and the opposite leg), or paraplegia (all four extremities or the two lower extremities), both upper extremities, or the arm and leg of the same side (hemiplegia).

Most frequently only one extremity is affected. Next in order of frequency comes paralysis of both lower extremities, while crossed paralysis and hemiplegia are much rarer. The upper extremity is less frequently affected than the lower, while paralysis of all four extremities is extremely rare, but three cases having been reported in the literature (see Seeligmüller).

It is extremely rare for a limb to remain totally paralyzed, nor do we find in any two cases an exactly similar distribution of the paralysis.

Certain groups of muscles will be found to have escaped the paralysis, and sometimes even portions of the same muscle will retain power, while the rest of the muscle is paralyzed.