adults in which paralysis resulted from acute arsenical poisoning, and which simulated anterior myelitis.

Cases in which rickets or marasmus have so disabled the lower extremities as to interfere with their use in locomotion are easily distinguished by the fact that there is no true paralysis; these muscles can be used, but walking is impossible, owing to tenderness and softness of the bones in the rachitic patients, and the weakness of the muscles of patients suffering from marasmus.

Congenital club-foot can be distinguished from paralytic cases by the absence of the characteristic electric reaction, and of other symptoms of infantile spinal paralysis.

The disability produced by congenital dislocation of the hips may be mistaken for paralysis, but a careful examination of the hip-joints, together with the peculiar gait and the absence of all the symptoms of this disease (infantile spinal paralysis), will serve as aids in making a diagnosis. Finally, inflammatory disease of the joints, especially of the hip, may give rise to atrophy and disability which might cause a suspicion of the presence of infantile paralysis.[23] When the history of joint disease is taken into consideration, together with the pain and swelling accompanying this condition, it will be hardly possible to be in error for any long period.

Treatment.—Inasmuch as there are no characteristic and pathognomonic signs by which a diagnosis can be made in the inceptive stage of the disease, before the paralysis has occurred, our treatment is necessarily one of symptoms and not rational. We treat, in other words, the fever, the convulsions, and the general conditions that may be present. But as soon as the disease itself can be diagnosticated by the presence of paralysis of motion in one or more members, the loss of reflex action, and the retention of sensibility in the paralyzed muscles, then a rational treatment can be instituted. We can attempt a derivative action to the skin by counter-irritation applied either locally to the spinal column, or to the whole of the cutaneous surface. As local counter-irritants in robust subjects, cupping and other forms of local blood-letting have been tried. Iodine may be painted along the whole length of the spinal column, or fly-blisters may be applied over the portion of the cord where the lesion is supposed to be located. General derivation to the skin may be accomplished by a strong mustard bath, repeated frequently in the course of a day. These baths, by reducing temperature, will serve to render the patient more comfortable, and will also exert a beneficial action upon the central disease. A brisk purgative may aid in reducing the congestion of the cord, and under any circumstances will benefit the general condition of the patient. Ice applications to the spine are indicated in cases in which the fever is an important factor in the treatment of the patient. The drugs which may be useful in this stage are those which antagonize the febrile movement, and those which are commonly termed antispasmodic. Of the former class are antifebrin and antipyrin, in doses sufficient to reduce temperature; among the latter are the bromides and chloral, which, together with the antipyretics, will generally succeed in stopping the convulsions. Ergot has been especially recommended by Althaus. He uses Bonjean's ergotine in watery solution (Julius Althaus: "On Infantile Paralysis," London, 1878, p. 51). The dose varies according to age under five years; from 0.01 to 0.02 gm. is injected once or twice a day. The treatment is continued until the temperature has become normal.

During the paralytic stage of the affection the treatment is well understood. Here the indications are, first, to treat the myelitis and thus promote and aid the regression of the paralysis; second, to treat the paralysis.

For the inflammation in the cord we can do but little. Ergot is given with supposed benefit. But the natural course of the inflammatory process is to destroy the ganglion cells in the gray anterior horns; when this has taken place, it is difficult to conceive how any internal medication can restore the destroyed nerve elements. It is claimed, however, and perhaps with some propriety, that those portions of gray matter which are only the seat of congestion can be benefited by internal medication; hence the ergot. With the same object in view counter-irritants to the spine are also used, such as iodine painted externally, blisters and cupping, either wet or dry. Dry cups applied to the spine on a level with the supposed lesion are better than wet cups. For obvious reasons they are also better than blisters.

Electricity is the curative agent in this affection. It may be applied locally to the spine, and also to the paralyzed muscles. Both galvanism and faradism are recommended. In my opinion, however, galvanism is more rational in theory and more effective in practice. Central galvanization of the spine is performed by placing one large flat electrode over the site of the lesion and the other over the farther extremity of the spinal column. An ascending (irritating) current should be employed, of strength sufficient to be felt by the patient; generally from twelve to sixteen elements Leclanché are sufficient. The duration of the applications should not exceed five minutes, and they should be repeated once every second day.

The application of electricity locally to the paralyzed muscles is the most effective use of this agent in this disease. Under the head of deformities we have already discussed its importance as a diagnostic agent. By the use of faradism alone we can recognize whether a patient has been the subject of a spinal-cord or peripheral-nerve lesion, or of a cerebral lesion. In advanced cases of atrophy from this disease the reaction to faradism is entirely destroyed; in these cases, therefore, the application of faradism as a therapeutic agent is certainly useless. I never apply this current until the muscles have sufficiently recovered to show a slight reaction when it is applied. Very few muscles, however—with the exception of those which are entirely destroyed—lose their excitability to galvanism, although the reaction present is generally that of degeneration. Galvanism, therefore, applied to the paralyzed muscles and nerves, causes both to become excited. This stimulus is undoubtedly beneficial, and persevering treatment with galvanism, continued for months and even years, is frequently successful in restoring muscles that were hopeless, to all appearances, when the treatment began. I apply the current by the aid of the interrupting electrode to the paralyzed muscles, and the other pole, generally the cathode, to some indifferent point. Applications to a limb should not exceed three minutes in duration.

Treatment of Deformities.—The principal therapeutic means at our command in the treatment of the deformities are tenotomy and mechanical appliances. Under the latter head I also include massage.

It has long been a question whether it is permissible to cut the tendons of the contractured muscles, and thus virtually paralyze all of the remaining healthy muscles of the limb. But, as is often the case, we find here that what would theoretically appear to be unwarranted, is found, in practice, to be sound and proper treatment. Careful consideration will show that the above objection is only apparently valid. Tenotomy disables a muscle only for a short time. The tendon soon reunites and the muscle is enabled to perform its functions to far better advantage than before. The strained position of the limb is relieved and the patient is enabled to use a joint which was entirely disabled before tenotomy was performed. Conservative surgeons prefer to stretch a contractured tendon, provided it admits of stretching. But such stretching, unless maintained for a very long period, is apt to be of but temporary utility; for a relapse is sure to occur when extension has been remitted. Where time, then, is important, tenotomy is certainly preferable to extension. I do not believe that the argument that tenotomy weakens a muscle is tenable. The active portion of a muscle is certainly not the tendon, but the muscular fibre. Tenotomy results in lengthening the tendon, the muscular fibres retaining their integrity. If healing has been properly looked to, the united tendon will not stretch more than is proper for the restoration of the joint to its

proper function. Where, however, the contracture is yielding, traction will readily accomplish all that is necessary, and should be preferred to tenotomy. It will be impossible, in the limits of this article, to describe all of the instruments used for purposes of extension of different muscles. For talipes equinus I know none better than Dr. Shaffer's "extension shoe," the working of which has been thoroughly described in a paper read before the Academy of Medicine. For varus, Dr. Shaffer has devised an equally effective "lateral-extension shoe." In calcaneus the chief indication is to raise the heel, the contracture rarely requiring extension or tenotomy. For these cases, as in all cases of paralytic club-foot, the shoe that is worn daily should have inside, covering the sole, a steel plate, to keep the sole firm and to prevent the shoe yielding itself to the deformed position of the foot. If the case be one of equinus, the heel should be fixed to the sole of the shoe by means of a strap passing over the instep. In all cases the shoe should be laced and made so as to open to the toes. The paralyzed muscles in many of these cases can be replaced, to a certain extent, by properly applied elastic straps, to act as artificial muscles. Contracture at the knee-joint should be overcome by tenotomy or extension. If the muscles about the knee-joint do not retain sufficient power to enable the patient to stand or walk with some firmness, we can virtually ankylose this joint by an apparatus in which a rigid bar passes from the hip or thigh down to the foot, without a movable joint at the knee; or a joint may be made at the knee, which, being guarded by an automatic spring, becomes rigid as soon as the patient assumes the standing position. One important principle in the application of apparatus for these deformities must not be lost sight of, and that is, that all apparatus must be so attached to the limb or body that the healthy muscles may be able to control or carry it, while it aids and takes the place of the paralyzed member. On the mechanical treatment of lateral curvature we will say nothing, the field being too large for the limits of this article. In treating the deformities of the upper arm and hand the principles already laid down will serve as a guide.

In the final stages of the disease, when contracture and paralysis have destroyed the function of limbs, and flail joints have rendered utterly useless whole extremities, certain surgical procedures will benefit the patient very greatly. Thus a healthy contractured muscle, which is useless owing to paralysis of its opponent, may have its tendon implanted at or near the insertion of the paralyzed muscle, and thus to a certain extent be enabled to perform the function of that muscle. This operation, known as tendon grafting, was first performed with good result by Nicolodoni in 1882. Goldthwait, of Boston, in 1896 reported a series of very interesting cases of this type. Arthrodesis has been useful in converting a flail leg into an efficient prop, much superior to a splint. Such ankylosis of joints is useful in bringing a useless joint into better position than that in which it has been left by the paralysis and resulting contractures.

Acute Poliomyelitis Anterior in Adults.

There is no doubt that a condition resembling, in its course and symptomatology, the same disease as it occurs in children may affect adults, although far more rarely than children. This has been evident from cases reported since that of Moritz Meyer in 1872 ("Die Electricität in ihrer Anwendung auf praktische Medicin," 2te Auflage, 1861) to the present time. Many of the cases reported as cases of acute poliomyelitis anterior in adults were in reality cases of polyneuritis without sensory symptoms. Indeed, a multiple neuritis of anterior spinal nerve roots would produce a condition simulating poliomyelitis anterior acuta in its symptomatology almost perfectly. The adults in whom this disease may occur are rarely more than thirty years of age. The disease is apt to affect adults who have recovered from an acute infectious disease, such as measles and scarlet fever. One case was reported as following an attack of rheumatism. Another case was supposed to be due to an exposure to cold after active physical exercise. The adult cases occur far more rarely than the infantile cases.

The symptomatology is almost identical with the same disease in children. Pain in the limbs preceding the occurrence of the paralysis is a symptom which has been reported in most of the cases. This pain disappears as soon as the paralysis occurs. The paralysis has the characteristics described in discussing infantile spinal paralysis. Its distribution in adults is characterized by the same lack of uniformity as in children. It is regressive. In adults, however, complete spontaneous recovery is believed to be possible. There is absence of patellar reflex. There are no sensory disturbances except the temporary pain just referred to. One or more muscles, groups of muscles, or extremities may be involved. There is sometimes backache. There are no motor disturbances of the rectum or bladder. Atrophies and degenerations occur as in the same disease in children. Similar electrical manifestations can be demonstrated in the paralyzed muscles. The reactions to galvanism and faradism are the same as in the infantile form of the disease. Deformities are not so apt to occur as a result of contractures in paralyzed muscles and their opponents, owing to absence of the factor of growth, which has such an important influence in disturbing the equilibrium between the healthy and paralyzed muscles of an extremity in young children suffering from this form of paralysis.

The diagnosis of this affection in adults is difficult, because the paralysis of poliomyelitis anterior acuta resembles that due to other pathological lesions to which adults are more often liable. Polyneuritis has already been mentioned. However, fever, followed by a rapidly developing motor paralysis, acute in its onset, which when it has reached its height begins to improve, together with the characteristic electrical symptoms, should enable the physician to make a diagnosis, at least as soon as the fever has subsided and the paralysis has become stationary.

The pathology of this disease in adults is the same as when it occurs in children.

The treatment is that which we have indicated in the same condition when it occurs in children, both in the stage of onset and in the chronic stage.

Poliomyelitis Anterior Subacuta et Chronica.

This form of poliomyelitis anterior is also generally found in adults; it is very rare. The condition resembles multiple neuritis in its symptomatology in a general way, and doubtless most of the cases reported clinically as poliomyelitis anterior subacuta were in reality cases of multiple neuritis. Yet cases have been reported in which post-mortem findings demonstrate that subacute or chronic poliomyelitis anterior is an actual entity, although a rare condition.

As etiological factors we find first metallic poisoning. In these cases this condition may be present alone or it may be accompanied by a toxic neuritis of peripheral nerves. The condition has also been seen during pregnancy. One case reported gave a history of an attack of infantile paralysis during infancy, followed in adult age by a gradually developing chronic poliomyelitis.

Pathologically the findings are similar to those in poliomyelitis anterior acuta, which have been recounted in the earlier portion of this article. Some of these cases, however, while they show degenerative changes with atrophy and sclerosis of the gray anterior horns, show also degenerations in the crossed pyramidal tracts and anterior nerve roots. One case reported by Oppenheim also showed systematic degeneration of the columns of Burdach in the cervical and dorsal regions, together with atrophy of the posterior nerve roots. One case has even shown an extension of the process to the columns of Clarke, and the posterior horns were also slightly involved, in addition to the changes in the anterior horns.

The root of the hypoglossus has also been found involved.

The atrophic changes in the peripheral muscles and nerves, as well as those occurring in the bones, while less marked, are similar to those found in poliomyelitis of children.

The symptoms are as follows: A paresis developing with a paralysis of a single limb or muscle is followed, in a week or month or year, by a gradually developing paresis or paralysis of another limb or of other limbs. This gradually extending, paralysis may continue, at intervals of longer or shorter periods, to involve more muscles until a large portion of the voluntary muscular system is affected, although one case has been reported in which there were bulbar symptoms. The muscles that are the seat of the paralysis gradually undergo atrophy, and give the complete and partial degeneration reaction found in poliomyelitis anterior acuta. The paralysis keeps extending for a year or more, and then may become stationary for the remainder of the life of the patient. In this form of the disease, unlike the acute form, there is observed little or no spontaneous improvement in the paralysis, while in infantile paralysis it is the rule that the paralyzed muscles recover, to a greater or less extent, spontaneously shortly after (*i.e.*, a few days or weeks after) the paralysis has set in. In the subacute and chronic form of the disease there is no spontaneous improvement.

Death generally occurs through some intercurrent disease, such as pneumonia, or, in rare cases, by paralysis of the nerves of respiration.

The differential diagnosis rests upon the absence of bladder and rectal symptoms. The diminished or lost patellar reflex; the absence, with rare exceptions, of sensory symptoms; the atrophy of the paralyzed muscles and limbs, are also important symptoms of the disease.

Progressive muscular atrophy can be differentiated from this condition by the fact that the paralysis in poliomyelitis subacuta and chronica precedes the atrophy, while in progressive muscular atrophy the atrophy precedes the paralysis. From multiple neuritis the condition is distinguished by the absence of sensory symptoms both subjective and objective. From amyotrophic lateral sclerosis it can be distinguished by the absence of the contractures and exalted patellar reflex which is characteristic of this affection.

The treatment consists of electricity, massage, baths, orthopædic apparatus, and drugs. The rules laid down, under acute infantile paralysis, for the employment of these agents in the treatment of the disease hold good here.

Orthopædic apparatus should be so constructed as to give rest and firmness to the paralyzed limb, by fixation of incompetent joints, etc., and to cause healthy muscles and limbs to do the work of those that have been paralyzed. Of the drugs that may be used, intramuscular injections of strychnine are the best. Galvanism is the form of electricity most applicable. It should be used in accordance with the directions already laid down.

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SPINAL-CORD DISEASES: SPINAL HEMIPLEGIA. (Synonym: Brown-Séquard's Paralysis.)—This term is applied to a group of symptoms produced by disease or injury of a lateral half of the transverse section of the cord. The array of symptoms to be mentioned become more or less clearly defined according to whether the entire half-section of the cord is affected or only a part thereof. The clinical picture was first described by Brown-Séquard, the description being based upon the results of experiments on animals as well as upon clinical and pathological observations in man. The conclusions of Brown-Séquard have not always been accepted, but Oppenheim has recently called special attention to the fact that, while experimental investigations have varied greatly in their results, accumulated clinical experience altogether upholds the clinical picture which he described.

Briefly stated, the result of lesion of one-half of the cord is motor paralysis of one-half the body below the seat of, and on the same side as, the lesion, and loss of sensation to a corresponding extent on the other side. *The motor paralysis is on the side of the lesion, the sensory paralysis is on the opposite side.* The extent of the paralysis depends on the height of the lesion; if it is in the cervical region, the upper extremity as well as the trunk and lower extremity is affected; if it is at a lower level, the manifestations will be limited to a corresponding part of the body. If the lesion be in the lower lumbar or sacral region, the motor and sensory symptoms will be on the same side, for at this level the sensory fibres have not yet crossed over to the other side of the cord.

The anæsthesia of the opposite side does not involve all qualities of sensation. The senses of pain and temperature are chiefly affected. In well-marked cases these do not escape. Tactile sensation is often unaffected, and, when impaired, is affected about as often on the side of the lesion as on the opposite side. On the other hand, the muscular sense is always impaired on the paralyzed side, that is, on the side of the lesion. Another symptom allied to the latter, ataxia, is often found on the same side when the motor paralysis is slight or when it has begun to improve.

Another notable sensory symptom is hyperæsthesia on the side of the lesion, that is, the side of motor paralysis. In well-marked cases this is nearly always present, though it may be slight in degree. This area of hyperæsthesia is often bordered above by a narrow zone of anæsthesia, the latter due to injury of the posterior spinal roots on the same side. On the other hand, injury of the anterior spinal roots (also of the anterior cornua) may be the cause of atrophy of the paralyzed muscles corresponding to the level of the lesion. Otherwise the paralysis usually assumes a spastic form with increased tendon reflexes and the appearance of the Babinsky phenomenon.

Not infrequently there is, temporarily, vaso-motor paralysis with some elevation of temperature on the side of the lesion. According to the seat of the lesion there may be vesical, rectal, or oculo-pupillary symptoms. Trophic symptoms have been observed in a few instances, sometimes on the side of the lesion, sometimes on the opposite side.

To recapitulate: The symptoms on the side of the lesion are motor paralysis, loss of muscular sense, and hyperæsthesia; on the opposite side, loss or impairment of the sense of pain and the sense of temperature. Tactile sensation is as likely to be affected on the one side as on the other, and, at least in old cases, more likely not to be affected at all. Temporary vaso-motor paralysis, and, very frequently, ataxia are found on the side of the lesion.

The physiological explanation of the symptoms is as follows: The motor fibres in the cord are in the antero-lateral column on the same side as the muscles with which they are in relation, their decussation occurring at a higher level, viz., in the medulla. The nerve fibres for muscular sense are in the posterior column, also on the same side, their decussation not occurring until they have reached the brain. On the other hand, the fibres which convey the sense of pain and that of temperature enter the cord through the posterior root and then pass in one of the commissures to the other side, ascending in the opposite antero-lateral column (it is generally believed in Gowers' column) to the brain. There is much greater uncertainty as to the paths for tactile sensation. Mann believes, and Oppenheim accepts this theory, that any nerve fibres which convey centripetal impulses—that is, impulses from the periphery toward the brain—may convey tactile sensations, though they are commonly conveyed by paths in the posterior columns. This theory readily explains the varying conditions of tactile sensation in these cases. The ataxia is probably due to lesion of the posterior column. The hyperæsthesia has as yet not been very satisfactorily explained.

The lesions producing this array of symptoms are often of traumatic origin, such as gunshot wounds, or wounds with sharp instruments. The lesions due to disease are most frequently those of spinal syphilis. In some instances hemorrhage limited to one side, in others pressure or destructive effects of a tumor, again a circumscribed myelitis or sclerosis, etc., may be the cause of the clinical picture. In some of these instances, especially those of trauma and hemorrhage, the early symptoms are much more marked than the later ones. The vaso-motor paralysis is usually but transient. The motor paralysis is likely to improve. The paralysis that remains is usually of the flexors of the hip and knee and of the dorsal flexors of the foot. Ataxia is likely to appear as the paralysis disappears. If tactile anæsthesia exists it is also usually an early symptom, disappearing at a later period. The disappearance of symptoms is in part due to the absorption of effusions and exudations and lessening of pressure, in part perhaps to functions assumed by the other half of the cord, or even to healing and resumption of functions of affected nervous tissues.

In cases of progressive lesions we may find double Brown-Séquard paralysis—first the symptoms of lesion of one-half of the cord, and then those due to the extension of the lesion to the other side. *Philip Zenner.*

SPINAL-CORD DISEASES: SYRINGOMYELIA.—It is impossible to give a satisfactory definition of a disease with such protean symptomatology and diversity of pathological findings. In general the name is given to a process of new growth in the gray substance, or to intramedullary cavities of varied origin, marked clinically by disturbances of motor, sensory, and trophic nature; by frequent bulbar symptoms, by almost constant reflex changes; usually by extreme chronicity. Reports of cavity formation in the spinal cord date back to a description of Étienne in 1564. Morgagni and Santorini recounted a case in 1740. Numbers of descriptions date from the early part of the last century. Calmeil in 1828 pointed out the influence of developmental anomalies. Ollivier first used the term "syringomyelia," though his conception of the cavity-formation needed later correction. A little later it was recognized that the pathological condition syringomyelia not infrequently was associated, clinically, with muscle atrophies (Landau, Nonat, Lenhossék, Gull). This association did not escape the acumen of Duchenne de Boulogne: "I found in a good part of my cases that electrocutaneous as well as ordinary cutaneous sensibility was affected. This anæsthesia is often so marked that the patient does not feel the strongest current or the ferrum candens."

Up to the publications of Kahler and Schultze in 1882 few clinical facts had been collected, but anatomical views had become sifted and well formulated. Five chief views maintained that the cavities resulted from: (1) a dilated central canal (Stilling, Waldeyer); (2) retrogressive changes in foci of chronic myelitis (Hallopeau, Charcot, Joffroy); (3) molecular degeneration due to vascular changes (Lockhart Clarke); (4) destructive metamorphosis of tumor masses (Grimm, Simon, Schultze); (5) certain developmental anomalies (Virchow, Leyden). In 1882 simultaneous publications of Kahler and Schultze quickened clinical interest and formulated rules of diagnosis. Since then the literature has become immense; the magnificent monograph of Schlesinger in 1901 represents the sum of our knowledge of the disease and has been freely drawn upon in the preparation of this article.

PATHOLOGICAL ANATOMY.—The cord may be hard to remove on account of marked kyphoscoliosis. There may be pachymeningitis, or pia or nerve-root thickening. The cord may seem flat and thin, the medulla small; or it may appear swollen in certain regions, especially in the neighborhood of the cervical enlargement; on section there may be cavities varying greatly in shape, vertical extent, and diameter. Frequently there are no macroscopical cavities, but irregular reddish-gray tumors in the gray substance. The medulla oblongata is often involved in the tumor- or the cavity-formation. The process may be limited to one side or to a small part of the cord; or it may extend throughout the cord and medulla oblongata. Usually the formation of tumors or of cavities involves the gray substance about the central canal. The anterior commissure is most often intact. One or both posterior horns may be attacked, or the ventral portion of the posterior columns; if the destruction advances, a large part of the gray matter disappears and the whole of the posterior and a large portion of the lateral columns become involved. The cavity walls may be smooth or ragged, may be lined by ependyma or closed by a proliferation of the glia. There may be evidence of old hemorrhage. As a rule, all changes are most marked in the cervical cord.

Microscopically, the tumor masses consist of glia cells and fibrils. The cavities are lined by a firm membrane that at times shows an epithelial covering. The tumors spring from the central part of the cord, the posterior horns, the posterior commissure, etc., and spread through the gray substance toward the posterior columns along the posterior median septum. The ultimate cause of the growth is to be sought for in certain developmental anomalies of the central canal or of the glia. The central canal may be unduly large or markedly irregular with diverticula. It may fail properly to develop and be marked by persistence of the dorsal process which is a characteristic at a certain stage of fœtal growth; or this diverticulum may be cut off and may persist as a dorsal canal seemingly independent of the central canal (Leyden). At other times nests of embryonic glia cells (ependyma) lie scattered in the neighborhood of the central canal or along the dorsal line of closure about the posterior median septum; and these may spontaneously, or under influence of some irritation (trauma), begin to proliferate and give rise to the glia tumors or gliosis (Hoffman). The cavity-formation may depend on many different processes—on dilatation or diverticula of the central canal, softening after trauma, hemorrhage or inflammation, destruction of tumor masses, retrogressive changes in glia proliferations (the common syringomyelia). But cavities and cysts after hemorrhage, myelitis, disintegration due to trauma, are rarely to be reckoned with the changes known as syringomyelia; they are stationary and not progressive. Another element is necessary, viz., some congenital disposition of the cord—an inherent tendency of the ependyma or glia cells to proliferate. Developmental anomalies of the whole central nervous system or only of the central canal and its neighborhood are found, therefore, in most cases of syringomyelia; anomalies of vessels and of the vascular connective tissues are also common and contribute to further proliferation and later cavity-formation.

Next to the posterior horns and posterior commissure the anterior horns and the posterior columns are the parts of the cord most affected. The cells of the anterior horns