Westphal, A.: Ueber die Bedeutung von Traumen und Blutungen in der Pathogenese der Syringomyelie. Archiv für Psychiatrie, Bd. 36.
Westrum, W.: Klinische Beiträge zur Kenntniss der Syringomyelie. Inaug.-Dissert., Erlangen, December, 1901.

SPINAL-CORD DISEASES: TABES.—Tabes is the term which is gradually being universally adopted to describe the disease popularly known as "locomotor ataxia." The name "ataxie locomotrice" was given to this affection by Duchenne in 1858 and is still quite popular. It is objectionable, however, because it is merely descriptive of a symptom, which at times may not appear until late in the course of the disease, or which may be entirely absent. "Posterior spinal sclerosis" is also unsatisfactory, as it refers only to the most easily discoverable lesion. "Tabes dorsalis," or "wasting of the back," was applied by Hippocrates to certain symptoms supposed to be due to venereal excesses, but was restricted to this disease by Romberg.

Tabes is the most common form of chronic organic disease of the nervous system. The pathological process underlying the affection is a parenchymatous degeneration terminating in sclerosis, which principally involves the sensory neurones. The peripheral motor neurones are also frequently implicated. In other words, it may be looked upon as a degenerative disease affecting various parts of the entire nervous system, while the most pronounced and extensive lesion is found in the posterior columns of the spinal cord. The morbid process may attack the cranial nerves and their nuclei as well as the peripheral nerves in the extremities. It may thus produce blindness from gray degeneration of the optic nerves, or paralysis of ocular muscles from degeneration of the nerve nuclei. The brain cortex does not always escape. The parts first affected in tabes are usually the fibres which originate in the spinal ganglia, *i.e.*, the fibres of the posterior roots which traverse the posterior columns.

Etiology.—In the vast majority of cases there is an antecedent history of syphilitic infection, antedating the first signs of the disease by from one to thirty-five years. In most cases the period ranges from five to fifteen years, the previous existence of syphilis being either demonstrable or admitted by the patient.

Statistics collected from various reliable sources show that from seventy to ninety-five per cent. of all tabetics give a history of previous syphilis. Hence it has been assumed by nearly all clinical investigators that tabes is one of the results of syphilis. Strümpell has expressed the view, which has been favorably received, that tabes does not arise from the syphilitic bacteria themselves, but from a toxin derived from them. It has therefore been almost universally considered as a sequel of syphilis, and has been variously characterized as a "post-," "para-," or "metasyphilitic" process. On the other hand, it is well known that only a small percentage of those who have contracted syphilis develop tabes. It cannot be consistently denied, however, that tabes also occurs without any discoverable evidence of previous syphilis. With the previous knowledge of an antecedent syphilis in a given case, we are by no means certain that the tabetic degeneration is the result of such early infection. It would seem that in most cases additional exciting or accessory causes are operative, such as exposure to cold, over-fatigue, alcoholic, sexual or other excesses, or a congenital or acquired neuropathic constitution—causes which predispose to the development of tabes, as well as to other degenerative processes, in the nervous system, in those patients who have contracted syphilis at some remote period.

Tabes has at times been considered as resulting from trauma, but in all such cases that have been investigated it has been shown that tabetic symptoms existed before the injury. It is a well-established fact, however, that the tabetic process is accelerated under such circumstances.

Symptoms.—Among the early symptoms of the disease first noticed by the patient are pains in the trunk and extremities; disturbance of the bladder; impotence; rectal pain; diplopia; ocular paralysis; failing vision; cutaneous hyperæsthesia or anæsthesia; incoördination in walking; visceral symptoms known as "crises" affecting the gastro-intestinal tract, bladder, or rectum, and occasionally laryngeal "crises."

Pain is often one of the earliest symptoms of tabes, and may antedate all other symptoms for many years. The character of the tabetic pains is as a rule pathognomonic. They are irregular in their distribution and are usually described by the patient as "shock-like," "sharp," "piercing," "cutting," or "stabbing," being rapidly repeated at the same spot, which often becomes extremely sensitive to the slightest touch. They may also resemble the painful sensation as if the muscles and bones were being crushed, or as if a piece of nerve was pulled. They may be so sudden in their onset that a strong man is surprised into a loud exclamation of pain while feeling otherwise well. They may occur every few moments for minutes, hours, or days. Although they differ essentially from true neuralgic, muscular, or periosteal pains, they are often erroneously considered as being of such origin. Hence it is a common experience for the neurologist to receive such patients with their announcement that they had previously been under treatment for rheumatism or neuralgia. In order fully to appreciate the significance of these pains, it is necessary to study this symptom carefully and minutely, for it may exist for several years before other symptoms are at all prominent. In the majority of instances, the pains most frequently affect the lower extremities. They may, however, be limited to the inframammary, intercostal, dorsal, or lumbar regions. Indeed, they are known to attack almost any part of the body. They may thus appear in the form of trigeminal facial pain or be located in the scalp. On the other hand, in some patients, pain is either entirely absent or only a slight, dull, circumscribed, and aching pain is occasionally complained of. If such pains do not appear in the early period of the disease, they are not likely to appear later.

Crises.—At almost any stage of the disease suddenly recurring paroxysmal attacks of severe gastralgia may take place, accompanied by exhaustive vomiting, which resists all ordinary methods of treatment, but seems to subside spontaneously. These attacks are known as "gastric crises." Sometimes persistent gastralgia may be the only symptom complained of. In such a case careful examination will often reveal an area of cutaneous anæsthesia in the epigastric region, thus leading to further investigation which discloses the underlying cause of the pain. Instead of these "gastric crises," there may be severe and unaccountable diarrhœa associated with violent colicky pain that may sometimes simulate an attack of renal colic. In some instances the rectum may be the seat of severe pain that is remittent in character. This pain not being relieved by ordinary means, its true nature being overlooked, surgeons have been led to the fruitless operation of forcibly stretching and paralyzing the anal sphincter and removing any hemorrhoids that perchance are present.

"Laryngeal crises" occur only in a very small percentage of cases. The most common form is true laryngeal spasm, with noisy inspiration and expiration, cough, and often considerable dyspnœa. The paroxysms may resemble those of whooping-cough or of laryngismus stridulus. Death during these attacks is extremely rare.

Incontinence or retention of urine is frequently an early symptom. Considerable effort is required in micturition, the urine flowing slowly. Quite often the bladder is not completely emptied and the residual urine may undergo decomposition and thus set up a cystitis.

Impotence may also occur as an early manifestation, but it usually develops as the disease advances.

Diplopia, ocular paralysis, or *failing vision* may be the first symptom that leads the patient to consult a physician. For several years before other symptoms are clearly manifested, or at any time in the early period of the disease, there may be frequent diplopia, or transient attacks of paralysis affecting the ocular muscles; these

attacks are of nuclear or peripheral origin. The paralyses of the external ocular muscles are often bilateral, but not symmetrical, often unilateral, and frequently affect only single muscles. Ptosis and paralysis of one or more muscles supplied by the oculomotor nerve, and abducens paralysis, are the most frequent. As a rule they develop suddenly and usually disappear after a longer or shorter period, with or without treatment. Relapses are frequent. Attention may first be drawn to the disease through a sudden third- or sixth-nerve paralysis, either partial or complete. The duration varies from a few hours to a year or more, or the paralysis may become permanent. Sometimes both eyes are affected, and a progressive and complete ophthalmoplegia may ultimately develop.

Atrophy of the optic nerve is the most serious ocular complication of tabes. It occurs in about ten or fifteen per cent. of the cases. For a long time it may be the only symptom of the disease, thus appearing several years before other characteristic phenomena are manifested. It rarely develops in the later period of the disease. The atrophic process is the result of gray degeneration of the optic nerves similar to that which attacks the posterior column of the spinal cord. The failure of sight usually commences with a peripheral limitation of the field and loss of color vision (the visual field for green contracting early); but sometimes central vision is defective from the very beginning. The atrophy is almost always bilateral, being more advanced in one eye than in the other, and ultimately progresses to complete blindness. It is rare for tabetic patients who become blind at an early stage of the disease to become ataxic later; but if the ataxia has become well pronounced, it does not always improve with the subsequent development of optic atrophy. In the majority of instances, the occurrence of optic atrophy seems to inhibit the further progress of the disease. This is a peculiar clinical fact which has never been satisfactorily explained.

Incoördination, or ataxia as it is familiarly termed, is a common symptom in many cases of tabes and most frequently affects the lower extremities. It has occasionally been noted by the patient himself, who first discovers difficulty in standing or walking with closed eyes.

As a rule, the lower extremities are affected first. When the sclerosis begins in the cervical portion of the cord, the ataxia, as well as other symptoms, may, for a longer or shorter period, be confined to the upper extremities. It is a curious clinical fact, previously mentioned, that, in cases in which optic atrophy appears, the ataxia often ceases its further progress.

Incoördination is a disturbance of the associated muscular action which is essential in the maintenance of equilibrium. Coördinate muscular action is kept under the patient's control to a certain extent by the attention and vision. As soon as the patient fails to watch his movements, or the eyes are closed, the ataxia is materially increased and may become uncontrollable. Thus, while standing with closed eyes and the feet close together, he may gradually or suddenly fall to the ground (static ataxia), being deprived of the sense of position of the legs, unless the eyes are opened. Incoördination affecting the lower extremities is productive of the characteristic tabetic gait ("locomotor ataxia"). In attempts at walking, when the condition is well pronounced, the foot is lifted from the ground at a much higher elevation than normal, and, being poised with considerable uncertainty, it is suddenly brought to the ground, striking forcibly on the heel.

The ataxia is due to an obstruction or obliteration of afferent impulses conducted from the periphery through the various sensory tracts. It therefore follows as a result of sclerosis of the posterior columns of the cord, or of degeneration of the peripheral sensory fibres in the muscles and joints, being directly conditioned by a loss of muscle and joint sensibility.

While in some cases it is often the most striking symptom, occasionally developing during the first years of the disease in conjunction with other discoverable signs, in many instances the ataxia is scarcely perceptible or demonstrable, or does not appear at all at any time during the course of the malady. Hence the designation of the affection as "locomotor ataxia" is a misnomer.

Sensory Disturbances.—Various paræsthesiæ are often complained of, such as numbness or formication in the extremities—a sensation as if the soles of the feet were resting upon rubber or fur; a band-like feeling around the body, usually at the level of the midthoracic region, as if a tight corset or belt enveloped the body. This sensation is known as "cincture feeling" or "girdle sensation." Or the patient discovers that sensibility in his legs is absent in various areas. In the upper extremities there may be numbness in the course of the ulnar nerve affecting especially the fourth and fifth fingers. In addition to the pains and paræsthesiæ described by the patient, disturbances of cutaneous sensibility are often found upon examination, in association with the loss of muscle and joint sensibility just mentioned. Impairment of sensation may be entirely absent, but in most cases of tabes some objective sensory disturbance is always found. It may vary from slight diminution of tactile sensibility to the most pronounced analgesia. The different qualities of sensation should always be tested carefully. In a large percentage of tabetics (four-fifths) there is an area of diminished sensibility to touch situated like a band about the chest in the midthoracic region and varying from three to four inches in width. This tactile loss may be associated with analgesia in the same area. A diminution in the sense of touch, pressure sense, and the sense of position of the limbs, usually appears in the early stage, while the higher degrees of anæsthesia are developed later. The temperature sense may remain unimpaired until the last stage of the disease. But analgesia is one of the earliest and commonest forms of sensory disturbance. The areas of analgesia may be distributed over the trunk and extremities. In the lower extremities it involves the sole of the foot, the heel, the toes, the anterior or lateral portions of the legs, or the inner surface of the thigh. The transmission of painful sensations is retarded in some cases, the prick of a needle being interpreted at once as a touch, and recognized as a painful sensation from three to ten or fifteen seconds later. This is generally described as "delayed conduction."

The loss of joint sensibility and of the sensibility of the deeper muscular structure occasions interference with the normal muscular "tonus" and results in deficiency or complete relaxation of muscular tension (hypotonus). This condition will explain the fact why tabetic patients submit to extreme passive movements of the extremities without complaint of pain. It may also account for the occurrence of hyperextension of the knee-joints often seen in tabetics (genu recurvatum).

Although patients often complain of weakness in the legs, it is only in exceptional instances (in which the motor neurone system becomes implicated) that such condition can be actually demonstrated. As a rule, the muscular power and resistance to passive movement are unimpaired. When muscular atrophy or actual paralysis exists, they must be considered as complications arising in the course of the disease.

The Loss of the Knee-jerk.—One of the earliest and most constant objective signs of tabes is the loss of the knee-jerk. As a rule it does not disappear suddenly. The pains may exist for a long time before the knee-jerk disappears. In the study of a large series of cases the knee-jerk symptom is found to be variable in its character. In quite a number of instances of undoubted tabes both knee-jerks may be active, but they usually disappear as the disease advances. This occurs when the upper portion of the cord is first affected. In other cases, the knee-jerks may be well marked on one side and lost on the other. Again, they may be apparently absent, but demonstrable only upon so-called reinforcement. Both may be equally feeble, or may differ in degree of weakness.

It has been claimed that in about one per cent. of healthy people the knee-jerk is absent. The correctness

of this assertion is questionable. Such an anomaly is so rare that its possibility need only be remembered. The writer has tested over one thousand healthy children from three to fourteen years of age, and the knee-jerk was demonstrable in every case. Its absence is due to an interruption in the so-called reflex arc in relation with the corresponding centre situated in the lumbar region of the cord. Much patience is often required in satisfactorily determining whether the knee-jerk is present or absent. The ordinary and customary method of testing for the knee-jerk while the person's legs are crossed, or the feet resting on the floor, will suffice when the knee-jerks are quite active. Under such circumstances the position of the limbs is immaterial. The utmost care, however, is necessary when there is any doubt as to the presence of the reaction. It will then be advisable to have the patient sit upon a high chair or upon the edge of a table, so that the feet are free from the floor. As a rule, both sides should be examined. In many instances the anticipation of the tap upon the tendon occasions involuntary rigidity of the flexor group of muscles, thus producing sufficient opposition to overcome the action of the quadriceps. Hence, before and during the examination, the patient should close his eyes, and his attention should be directed from the purpose of the examiner, either by conversation or by rapid interrogation. Or he may be directed to make some muscular effort with his hands, such as forcibly interlocking the fingers, elevating the arms, etc. This is known as "re-enforcement." With this object in view any other similar expedient may be resorted to that suggests itself to the examiner. It is never safe to state that the knee-jerk is absent, unless repeated and varied tests have been made with the clothing removed.

Reflex Iridoplegia.—Another early and most important evidence of tabes is the loss of the reflex action of the iris to light. This is familiarly known as the "Argyll-Robertson symptom," but in recent years it is more correctly described as reflex iridoplegia. It is present in about eighty per cent. of all cases. Being a phenomenon somewhat analogous to that of the loss of the knee-jerk, it is found to be just as variable in the extent of its manifestations. Its absence, however, does not exclude the diagnosis of tabes. The longer the duration of the disease, the more likely are we to find this sign. When present it is generally bilateral. In rare instances it is unilateral—*i.e.*, affecting only one eye, or the degree of reaction may differ in the two eyes. It is usually unaccompanied by any disturbance of vision. As a rule, the pupils react in convergence of the optic axes. In some cases of tabes the pupils are absolutely rigid, and do not react to light or in convergence. They may also be unequal, partly dilated or contracted. The inaction of the pupil when exposed to light is not indicative of a lesion in the spinal cord, as was erroneously supposed by many before tabes rested upon a firm pathological basis, but is the result of interference with the reflex conducting path in its course between the optic nerve, corpora quadrigemina, and oculomotorius. It is therefore due to a lesion within the brain, and may be looked upon as an almost infallible sign of central nerve degeneration involving the sphincter nuclei of the third nerve or their efferent fibres when the eyes are otherwise apparently normal.

The examination of the pupils for reaction to light requires the closest observance in its performance. This apparently simple procedure is deserving of careful attention, and certain precautions are absolutely essential in order to avoid erroneous conclusions. The following method should be pursued: Place the patient in a position facing a window. Daylight is always preferable. Instruct him to gaze steadily upon some large object at least twenty feet distant, and to keep both eyes open. The eyes are then covered or shaded either with the examiner's hands or with two pieces of dark card-board. The patient in the mean time must continue gazing in the direction just mentioned. In a few moments one eye is suddenly uncovered and exposed to the light, when in the normal state the pupil (which always dilates in darkness or subdued light) immediately contracts. The other eye is then tested in the same manner. The next step is to note if the pupils contract in convergence or accommodation. After the patient has been looking in the distance for a short time, with both eyes uncovered, he is suddenly told to look at the examiner's finger, or some small object held within two inches of the patient's nose. In the normal condition the eyeballs converge and the pupils contract. The pupillary reactions should not be tested before a gas flame, as the patient is apt to, and in fact generally does, "fix" upon the flame, thereby causing contraction of the pupils in accommodation and convergence, which may easily mislead. A very common source of error to be guarded against, which is similar in its result, is the failure to bear in mind the natural tendency of the patient to look at the examiner as soon as the eye is uncovered. Bilateral reflex iridoplegia may also be associated with myosis. While the former often exists without the latter, myosis is as a rule accompanied by loss of the pupillary light reflex. The myosis, however, is due to a lesion affecting the spinal cord and involving the so-called cilio-spinal centre situated between the fourth cervical and the second dorsal segments. As the pupil-dilating fibres pass from thence out by the rami communicantes into the cervical sympathetic, degeneration of these fibres causes the permanent contraction of the pupils.

Arthropathy.—Trophic disturbance in the bones and joints is an occasional phenomenon of tabes, but it is of sufficiently frequent occurrence (in about three or four per cent. of all cases) to command attention. This form of joint affection is known as "tabetic arthropathy," and most commonly involves the knee-joints, although the hip-joint and the large joints of the upper extremities may also be similarly affected. It has often been known to arise during the early or pre-ataxic stage of the disease. Painless swelling and complete disorganization of the joint are pathognomonic of tabetic arthropathy. It usually arises suddenly and develops rapidly. Sometimes following a trivial injury, the joint becomes swollen and the surrounding tissues œdematous, but there is complete freedom from pain, redness, or fever. The joint structures, including the ends of the bones, undergo rapid destruction.

The long bones at times become brittle and thus easily fracture either spontaneously or from a slight traumatism. Among the trophic cutaneous disorders, perforating ulcer of the foot is the most common. It is usually situated on the plantar surface of the foot, either under the base of the great toe or of the fifth metatarsal bone, or at the heel. It generally begins as a callus or corn, and is painless, even when the ulcerative process is quite deep and extensive.

Diagnosis.—The appearance of a classical case of the well-advanced disease, as described in the text-books, is a familiar picture. There is a superabundance of convincing proof to establish the fact that this, probably the commonest form of organic nervous disorder, often remains unrecognized for months or years during its early stages, although ample evidence of its presence may be discoverable by proper methods of investigation. To the most sanguine neurological mind the approach of the medical millennium does not imply the restoration to life and normal function of sclerosed and destroyed nerve structures; but if our modern therapeutic measures are to prove curative in this disease, they must be utilized before organic changes are too far advanced. Our only hope of success in the treatment of tabes rests in a correct diagnosis at the earliest possible moment. The early diagnosis of tabes—which, so to speak, is chronic from its very beginning—is of incalculable value, inasmuch as it saves the patient much misdirected and useless medication. When unrecognized tabes has existed for some time, various pronounced symptoms may appear, and, although a part of the disease, may conceal its true character. Its presence is frequently unsuspected by the attending physician until the signs are so conspicuous that they almost speak for themselves. Although static ataxia frequently occurs

in tabes, it is by no means pathognomonic. The fact that a slight swaying is of common occurrence in healthy individuals should always be borne in mind. Static incoördination is often present in hysteria or pronounced neurasthenia, during convalescence after prolonged illness, in transverse myelitis, and in polyneuritis resulting from diphtheria, alcohol, lead, arsenic, etc. Some forms of alcoholic polyneuritis are commonly mistaken for tabes on account of their resemblance to that affection (see the sections on Neuritis and Polyneuritis in the article on *Neuritis*). Upon superficial examination the presence of static ataxia with anæsthesia and absence of the knee-jerk, points to tabes. But as these symptoms are usually associated with tenderness on pressure over the affected nerve trunks, diminished muscular power or actual paralysis, and decrease or loss of faradic irritability in the affected muscles, we are enabled to exclude tabes. The absence of the knee-jerk in these cases is due to involvement of the motor fibres of the anterior crural nerve or its branches, and is usually an accompaniment of weakness or diminished resistance in the quadriceps. The loss of the knee-jerk in uncomplicated cases of tabes, however, is attended as a rule with preservation of muscular power in the extensor group, even when the disease is well advanced and there is pronounced incoördination. This peculiar feature of tabes can be easily demonstrated, and in my experience is of extreme value. When the peripheral sensory nerves are especially affected, as in certain forms of toxic polyneuritis, particularly from arsenical poisoning, the disease has been termed pseudotabes. On account of the presence of well-marked ataxia, absence of the knee-jerks, and anæsthesia, it closely resembles the true disease. A consideration of the antecedent history, mode of onset, and development will generally enable us to reach a satisfactory conclusion as to the nature of the trouble. At times the differential diagnosis between tabes and some atypical forms of polyneuritis is attended with much difficulty. On the other hand, in some tabetic patients the peripheral nerves of the lower extremities may undergo degeneration, and thus lend confusion to the otherwise stereotyped picture. I have repeatedly seen chronic transverse dorsal myelitis mistaken for tabes, owing to pain in the lower extremities, inability to stand with closed eyes, and cutaneous anæsthesia. Such an error must be ascribed to gross carelessness or neglect in the examination. The exaggerated knee-jerks with probable ankle clonus, and the loss of muscular power sometimes with atrophy, should serve to make the diagnosis clear. Some cases of cerebrospinal syphilis so closely simulate genuine tabes that their differentiation can be accomplished only after a careful study of the development and course of the symptoms. Paretic dementia and tabes are sometimes found associated, but when the mental symptoms predominate, such as changes in the patient's character, diminution of the intellectual functions, speech disturbances, attacks of unconsciousness, etc., we must favor the diagnosis of the former.

The most important subjective symptom is the peculiar paroxysmal pains, while the two principal objective signs of tabes are the absence of the knee-jerk and the loss of the pupillary light reflex. It may be safely said that the coexistence of the latter is equivalent to the diagnosis of tabes. It rarely happens that both are absent at the same time, yet such cases do occasionally occur. We must then depend upon the association of other symptoms and must watch for further developments. The presence of either one, in conjunction with the fulgurating pains, or bladder disturbance, or the areas of anæsthesia over the back, is sufficient evidence to warrant the diagnosis. When a man over thirty years of age, with a history of ancient syphilitic infection, complains of the pains characteristic of tabes, we should at once suspect its existence, whether other signs are present or not. Should these typical pains be associated with one or more of the objective symptoms, the diagnosis may be made with confidence. It is a fact well known to all neurologists, that among physicians in general the failure to recognize the presence of tabes is more common than its diagnosis. On the other hand, an incorrect interpretation of phenomena which resemble those of tabes is of frequent occurrence. Such conditions can be obviated only, first, by a clear conception of the clinical peculiarities of the disease; second, by a practical familiarity with the method of examination, combined with its application to the individual case.

Duration, Course, and Prognosis.—Tabes is not invariably progressive in character, but it is always chronic in its course, the average duration of the disease being about ten years, although in uncomplicated cases it has frequently lasted for from twenty to thirty years. In rare instances it is very rapid in its progress, terminating fatally in a few years. As the clinical types differ materially, the course and duration of the disease vary with the character and severity of the symptoms. Remissions have been known to occur, and under suitable management the disease often becomes stationary in its early stages. Occasionally, even in well-advanced cases, the amount of improvement is often very great. The development of cystitis may at times lead to fatal pyelonephritis, and falls or slight injuries may result in fractured bones or in a rapid advance of the pathological process. The characteristic pains, slight ataxia, and absence of knee-jerk and pupillary light reflex may exist for many years without any further manifestations. In some cases the symptoms are so moderate that the patient may live in comfort and be able to be about for many years, while in others the intensity of the symptoms may be so pronounced that the patient becomes bedridden in a year or two.

Treatment.—Although the tabetic degeneration cannot be cured, much can be accomplished by improving the general health of the patient, and thus, in many instances, delaying the further progress of the disease. Hence such measures as prolonged rest in bed, if necessary, judiciously applied hydrotherapy, massage, and static electricity often prove valuable. It is essential to avoid fatiguing exercise or excesses of any kind. Certain subjective symptoms may also be relieved. Objective symptoms, such as optic atrophy, reflex iridoplegia, and absent knee-jerks, always remain permanent despite any form of treatment. The effect of drugs on the tabetic process is very misleading on account of the spontaneous remissions that are known to occur in the course of the disease. Besides, the result of the administration of medicinal remedies in a given case cannot be predicted upon the basis of its beneficial influence in another patient. As the disease varies in its type in different individuals, the plan of treatment will have to be modified accordingly. The patient should be cautioned to avoid any unnecessary exposure to falls or other injuries, for the osseous structures are often brittle and fractures are easily produced. The condition of the bladder must be carefully watched and the urine frequently examined. At the first indication of decomposition of residual urine, or of any symptoms of cystitis, the bladder should be irrigated and the patient instructed in the aseptic use of the catheter.

Tabetic patients who have had syphilis, but who have never received antiluetic treatment since tabetic symptoms have developed, should be given a thorough course of active medication for several weeks. If this has already been carried out without relief, it is useless—nay, even harmful—to repeat the procedure. Not only have I never seen such patients improved by this means, but I have repeatedly noted an increase in the degree of many of the symptoms, presumably as the result of interference with the general nutrition. One difficulty in this matter lies in the possibility of mistaking cases of exudative cerebrospinal syphilis for tabes, *i.e.*, an inflammatory for a degenerative condition. When such a differentiation is not clear or is apparently impossible, the patient should be given the benefit of any reasonable doubt, and antisyphilitic drugs administered.

Pain.—If the action of the bowels is regulated and the diet and general habits of the patient are properly controlled, the frequency and severity of the tabetic pains

will be invariably ameliorated, or the attacks may subside entirely. The faradic wire brush, or linear cauterization applied over the vertebral column in the region corresponding with the posterior roots that are presumably associated with the location of the pain, often proves effective. Various anodyne liniments, or the application of an ice-bag or of hot water to the seat of the pain during the attack, give relief in some cases. When necessary, the various coal-tar products may be tried. The most useful are acetanilid and phenacetin. As a last resort codeine or morphine may be administered. During a "gastric crisis" the best method of relief is the subcutaneous injection of morphine or codeine.

Suspension.—This is often useful in selected cases, but it should never be used indiscriminately. The patient may be suspended in a Sayre apparatus for about two or three minutes twice a week. The same result (*i.e.*, stretching of the vertebral column) may be accomplished by having the patient sit on the floor, and then forcibly flex the head and trunk upon the thighs, while the lower extremities are kept straight.

Educational Exercises for Controlling the Ataxia.—This plan of treatment was introduced to the medical profession in the year 1889 by Dr. H. S. Frenkel, of Heiden, Switzerland. A large variety of exercises may be practised. They may be executed with the patient in a recumbent position; while sitting; in an erect position; and various movements may be carried out while walking.

Exercises for the Trunk and the Lower Extremities.—While recumbent: (*a*) Lying on the back, raise body into sitting position and return to recumbency. (*b*) Flex thigh upon abdomen, leg upon thigh, then extend and elevate entire extremity and slowly return to original position. (*c*) Elevate entire extremity while in extension and return (Goldscheider).

The following movements have been recommended by Dana as a modification of Frenkel's method:

"1. Sit in a chair, rise slowly to erect position, without help from cane or arms of chair. Sit down slowly in the same way. Repeat once.

"2. Stand with cane, feet together; advance left foot and return it; same with right. Repeat three times.

"3. Walk ten steps with cane, slowly. Walk backward five steps with cane, slowly.

"4. Stand without cane, feet a little spread, hands on hips. In this position flex the knees, and stoop slowly down as far as possible; rise slowly; repeat twice.

"5. Stand erect, carry left foot behind and bring it back to its place; the same with the right. Repeat three times.

"6. Walk twenty steps, as in exercise No. 3; then walk backward five steps.

"7. Repeat exercise No. 2, without cane.

"8. Stand without cane, heels together, hands on hips. Stand in this way until you can count twenty. Increase the duration each day by five, until you can stand in this way while one hundred is being counted.

"9. Stand without cane, feet spread apart; raise the arms up from the sides until they meet above the head. Repeat this three times. With the arms raised above the head, carry them forward and downward, bending with the body until the tips of the fingers come as near the floor as they can be safely carried.

"10. Stand without cane, feet spread apart, hands on hips; flex the trunk forward, then to the left, then backward, then to the right, making a circle with the head. Repeat this three times.

"11. Do exercise No. 9 with heels together.

"12. Do exercise No. 10 with heels together.

"13. Walk along a fixed line, such as a seam on the carpet, with cane, placing the feet carefully on the line each time. Walk a distance of at least fifteen feet. Repeat this twice.

"14. Do the same without cane.

"15. Stand erect with cane; describe a circle on the floor with the toe of right foot. Same with toe of left. Repeat twice.

"Between the fifth and sixth exercises the patient should rest for a few moments."

All exercises must be performed slowly and deliberately and repeated several times according to the amount of fatigue produced. Some of the principal movements have just been described. Many modifications or additions may be followed, according to the condition of the patient. For more elaborate details relating to all forms of exercises for the upper and lower extremities, the reader is referred to Frenkel's book on "The Exercise Treatment of Tabetic Ataxia."

As all tabetic patients are not ataxic, and as all ataxic tabetics are not suitable subjects for such exercises, this method of re-educating the cerebral centres for coördination will be found to have well-defined limitations. Great care and watchfulness are necessary in carrying out this plan of systematic exercise, particularly in advanced cases. Unless practised persistently and systematically, preferably under the immediate supervision of a competent attendant, the results are usually unsatisfactory. Under suitable conditions, patients who were formerly unable to stand without assistance have been enabled to walk. *William M. Leszynsky.*

SPINAL-CORD DISEASES: TUMORS.—Practically all of our knowledge of spinal-cord tumors that is of any import for clinical medicine has come in these last two decades. It is true that, over thirty years ago, von Leyden put medical literature under a great debt by his published studies of one very interesting case, and within a few years after that he succeeded in showing that the diagnosis of the location and of the nature of a spinal-cord tumor is not impossible to the careful clinician. Before this only the pathological anatomists had given any attention to tumors lying within the spinal canal. Of these, two men deserve mention, because of the thorough manner in which they studied the subject and suggested at least the possibility of progress in clinical medicine up to a point where even this obscure condition might be recognized with certainty, its progress foreseen, and possibilities of surgical interference discussed. These two men are Virchow, in Germany, and Cruveilhier, in France.

The most important practical progress in our knowledge of spinal-cord tumors was to come from England. In 1886 Gowers, having demonstrated many times that it was possible to determine exactly the location of tumors of the spinal cord, recommended that when the diagnosis was satisfactory and conclusive, operative interference should be undertaken. Some years before this Erb, at Heidelberg, had made the same suggestion, though it did not for the moment attract much attention.

Gowers' recommendation was destined to bear fruit very shortly. In 1887 Gowers and Victor Horsley, after thorough discussion of a case of spinal-cord tumor, proceeded to operation, and Horsley removed the tumor successfully, the patient recovering completely and without any serious sequelæ. This classical case deserves to be recalled as the first step in a new and brilliant chapter of surgical therapeutics. It is described in a paper by Horsley and Gowers in the *Medico-Chirurgical Transactions*, London, 1888.

The patient had suffered from pain referred to the middorsal region for four years, which was suspected at first to be due to an aneurism. Certain neurotic symptoms superadded to the pain made some observers consider the symptoms functional. The pain continued off and on for four years; then the left leg became weak and later the right, with complete loss of power. Sensation was impaired and retention of urine occurred. Absolute palsy of the legs was present just before the operation was decided on, with loss of sensation in the trunk as high as the ensiform cartilage, and severe girdle pains were felt in the sixth and seventh intercostal spaces, more on the left than on the right. From time to time the legs became rigid in extensor spasm and the reflexes were much exaggerated. There was no tenderness and

no deformity of the spinal columns. Potassium iodide had been used in vain.

At the operation the arches of the fourth, fifth, and sixth dorsal vertebræ were removed, but no tumor was found. The third and seventh arches were removed without any more success. As the patient's condition was good, his second dorsal arch was also removed, when the lower end of the tumor appeared. A portion of the first dorsal arch was cut away to facilitate its removal. It proved to be a fibromyxoma that could be easily lifted from its bed in the lateral column of the cord. The patient made a good recovery and gradually sensation and motion returned. A year later he was doing sixteen hours of work a day, standing and walking most of the time. Horsley and Gowers suggested that spinal tumors were more operable than tumors of the brain, and reported a certain number of cases collected from the literature in which operations would probably have been successful.

This demonstrated that spinal tumors were not as hopeless as had been thought. In the *American Journal of the Medical Sciences* for 1895, page 614, Dr. M. Allen Starr collected the reports of 122 cases of tumor of the spinal cord, with regard to 100 of which fairly satisfactory histories were obtainable. In all of these the diagnosis might have been made before death, and of the 100 cases there were 75, in Dr. Starr's opinion, in which the tumor could have been removed. He also reported, apart from the 127, 22 cases in which operations had been performed. In 2 of these the tumor was not found. In 1 it was impossible to remove it when found. In 11 cases death took place soon after the operation. Eleven patients operated upon recovered, and in 6 the paraplegia completely disappeared; in the other 5 there was relief of symptoms, especially of pain, and some improvement in the gait. Dr. Starr considers then that in nearly seventy-five per cent. of the cases spinal tumors can be operated upon successfully.

In the Versammlung Südwest. Deutsch. Neurologen, held at Baden, 1902, Professor Schultze gave a synopsis of 8 cases of spinal tumors in which operations had been performed. In 2 cases there were errors of diagnosis; of the remaining 6 there were 3 recoveries—2 complete, 1 partial. There were 2 deaths, shortly after operation, and 1 patient remained unimproved. This would give somewhat less than fifty per cent. of prospect of relief from operation.

Dr. Collins, in the *Medical Record* for December 6th, 1902, reports 70 cases of spinal tumor from the recent literature, 30 of which were operated upon. He concludes that spinal-cord tumors are twice as operable as brain tumors, and the results of operation are twice as successful. Fifty per cent. of intraspinal tumors are operable and one-third to one-half of these cases are benefited by operation. Of the 30 operations, 12 were completely successful, 8 were partially successful, and 10 failed to give relief, or the patient died shortly after operation. Of the 70 cases reported, 44 were almost surely operable with hope of relief; 26 were wholly beyond hope. While, then, the outlook for operation in cases of spinal tumors is quite hopeful, Dr. Collins considers that it is not as good as has been represented by previous authorities.

These reports serve to show the present status and the evolution of the surgical treatment of spinal tumors. They serve also to bring out very clearly the fact that neoplasms affecting the spinal cord are by no means hopeless. This has led to thorough discussion of the diagnostic points of spinal tumors, so as to make assurance of their presence and localization as absolute as possible for the guidance of the surgeon. Nowithstanding the progress in this matter, every year some cases are reported in which the tumor fails to be found at operation. In the very first case operated upon (Horsley's cited above) the opening in the spinal canal was made somewhat too low at first. Dr. Collins points out that recent experiences of Starr, Oppenheim, and Schlesinger show that there is danger of localizing the spinal tumor one segment too low, even at the present time. With this precaution it must be admitted that the diagnosis of spinal tumors is as certain as any other bit of internal diagnosis, and definite localization can usually be reached without serious doubt remaining.

Division of Tumors.—Tumors of the spinal cord—that is, new growths affecting the spinal portion of the central nervous system—may spring either from the nervous substance of the cord itself or from some of its coverings. These include not only the spinal membranes (that is, the meninges), but also the periosteum of the spinal canal and the bony column that acts as the support and protection for the cord. It seems better, with the authorities on this subject, to include the bony tumors of the vertebræ. All these tumors give a certain similarity of symptoms, so that we shall consider first tumors of the vertebræ, then tumors which have their origin from the periosteum or from the extradural fat-tissue, then intradural tumors which spring from the inner surface of the dura, from the arachnoid membrane, from the ligamentum denticulatum, from the nerve roots, or from the pia mater, and finally true medullary tumors, which spring from the nervous substance of the cord itself. We may say at once that by far the most important group of tumors is the intradural.

Tumors of the vertebræ have been arranged by Bruns, according to their importance, in the following order: Carcinoma, sarcoma, and osteosarcoma, as also sarcomatoid tumors with myxomatous degeneration, or fibrosarcomata, exostoses, or osteomata, especially multiple exostoses, echinoccus cysts, and finally gummata. There is a pseudo-tumor of the vertebra, due to exaggerated callous formation after fracture of a vertebra, which acts exactly like a tumor, and should accordingly be treated among them.

By far the most important and the most frequent form of tumor of the vertebræ is *carcinoma*. In the great majority of the cases the tumor is not primary, but secondary to cancer in some other part of the body. Vertebral cancer follows particularly cancer of the breast; much more rarely does it occur after stomach or uterine cancer. It is likely, then, to affect especially women, and as a matter of fact the cases of cancer of the vertebræ reported in men are extremely rare. Vertebral cancer may occur many years after an apparently successful operation for cancer of the breast. Bruns reported a case in which eight years had intervened. The late Dr. William Pepper showed to his clinic a case in which over six years had elapsed since the removal of the breast. Primary cancer of the vertebræ occurs very rarely.

As might be expected from the original seat of the tumor, secondary cancers of the vertebræ occur especially in the middle and upper dorsal region, occasionally in the lower cervical region. Primary carcinoma of the vertebræ by contrast occurs more particularly in the lumbar region. Secondary cancer does not limit itself to one vertebra, but apparently from the beginning attacks several. There are in the literature a number of cases in which practically every vertebra from the cervical region down to the sacral region had become carcinomatous. The process may spread by continuity, but it would seem that frequently each vertebral body becomes the seat of a new metastatic focus of malignant disease.

Notwithstanding the wide distribution of the carcinomatous process, the area in which there is pain on pressure or in which there are neuralgic pains due to the irritation of the growing tumor may be very small. Even when there is extensive involvement of the vertebral column, deformity may be absent. The usual deformity is a sinking together of the whole spinal column, a change which the French call *entassement*. In a number of cases patients have been distinctly decreased in height by this sinking together of the vertebræ. Often the bodies of the vertebræ become almost as thin as paper, and it is hard to understand how the patient can hold himself erect. Occasionally there are sudden dislocations of the vertebræ, and these may occur after even very slight trauma. When displacement of the vertebræ occurs, however,