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

are either directly invaded by the gliosis, or are destroyed by hemorrhage, or at times they undergo atrophy at a distance from the process—perhaps as a result of inherent weakness. The ventral portion of the posterior columns —the ventral field of the posterior columns; the dorso-medial sacral bundle of Obersteiner; Schultze's comma tract—most frequently suffers. The pyramidal tracts may either be directly invaded or they may undergo degeneration from pressure of the growth at a higher level of the cord.

Bulbar Lesions.—Pathological changes occur in the median line or laterally, especially along the entering vagus roots. The cavities are as a rule small—mere slits rather than holes. Those lying near the median line are due to developmental anomalies, while the lateral rifts and cavities have their origin in vascular changes, in inflammatory processes. The cavities are lined with glia, but usually there is no distinct evidence of proliferation. The vessels are enlarged and present in unusual number. The central cavity when present is lined with ependyma. The lateral lesions may involve the nerves from the twelfth to the fifth; the twelfth, tenth, ninth, and descending roots of the fifth are most often implicated. The process ends at the pons. The fillet is frequently destroyed. The process is nearly always predominantly unilateral.

In judging of pathological findings one must remember the possibility of various artefacts. Tears may occur along the posterior horn and commissure and in the gray substance from pressure and crushing during removal of the cord (Kolisko). Cavities may result from putrefactive gas-formation, and changes may go on in the specimen while it remains in the hardening fluid if this is not frequently changed during the first days. Van Gieson has called attention to the various artefacts in an exhaustive article.

Etiology.—Not infrequently the disease seems to have direct relation with trauma of some sort. According to Minor a central hæmatomyelia due to trauma is often the starting-point of the abnormal process. Perhaps hemorrhages due to trauma during birth act in the same way (Schultze). It is plausible to think that, in the case of the developmental anomalies described above, the incitement to glia proliferation may be given by trauma; experimental evidence favors such a view (Schmaus). The theory has been advanced that even peripheral trauma or a panaritium, etc., may, with the predisposition already mentioned, and through the agency of an ascending neuritis, be able to start the process of proliferation; this supposition has little probability. Different infections, especially typhoid and influenza, may act like trauma in certain cases and supply the initial irritation leading to later tumor-growth. Excessive temperature changes, particularly exposure to cold, seem occasionally to act in the same way. Syphilis may lead to cavity-formation through vascular and meningeal lesions, but has little influence in the production of the progressive changes which characterize syringomyelia. Leprosy plays no rôle as a causal factor. The relations of trauma to the disease are most important from a medico-legal standpoint. Most often the trauma is an accident in the already developed disease. Rarely, in congenitally predisposed subjects, it may be the primal cause of the process. Not infrequently it leads to decided exacerbation of the quiescent disease. Most often its effects are disastrous, due to peculiarities of the disease and not to severity of the trauma (trophic joints, spontaneous fractures, etc.).

Symptomatology.—From the character of the pathological lesions it is plain that symptoms must be protean and capable of almost endless combination. In the most usual localization of the process in the cervical enlargement the picture is fairly typical. Lightning pains in one or both arms, or paræsthesiæ, especially sensations of burning or coldness, or "burning cold," not infrequently precede striking changes. Atrophy of hand or arm muscles is a marked feature; it may be unilateral. The small hand muscles are most often involved; the process may begin in the shoulder group. Usually when atrophy is well marked, sensory changes can be demonstrated. These at first are found over small areas of the hands, arms, or shoulders, and there may be only a moderate diminution of temperature perception, not a complete loss. Vaso-motor disturbances and trophic changes of varied kind may be noted in the skin, subcutaneous tissues, bones, and joints. The knee-jerks are increased; if the process involves one arm predominantly the knee-jerk on the same side will be the livelier. Later on, contractures may occur; the atrophy progresses; unilateral bulbar symptoms may develop; spastic paraplegia becomes marked; further trophic changes are seen; sensory changes cover wide areas and increase in intensity; the sphincters may be affected.

If the cavity-formation involves the dorsal and lumbar cord, pain and paræsthesiæ will be felt in corresponding nerve roots. The muscle-atrophy will affect the adductors, quadriceps extensor, peroneus group, the back extensors, perhaps the glutei and other muscles of the calves. The knee and Achilles reflexes are usually increased. Sensory and trophic disturbances are found in the lower extremities or in the trunk. The sphincters are frequently affected.

When the lumbo-sacral cord is involved there is atrophy of the glutei, knee flexors or foot muscles—often unilateral. Sensory changes are demonstrated in the feet, perineum, bladder. The sphincters are usually affected early, but they may escape. Trophic changes are often peculiar. The leg and foot may remain small or may be unduly large; bone and joint changes, panaritium, malum perforans, are frequent. The patellar as well as the Achilles jerks are usually increased; a weighty sign is loss of the Achilles reflexes with normal knee-jerk.

Association with congenital hydrocephalus or spinal deformities, spina bifida, are common.

Symptoms in Detail.—I. *Sensory Disturbances.*—The essential characteristics of the sensory changes were well described in the first publications of Kahler and Schultze. Laehr, Hahn, Dejerine, Brissaud, Schlesinger, and von Soelder have studied in detail the peculiarities of distribution. Most characteristic is the so-called "syringomyelia dissociation"—the essential preservation of touch and sense of pressure, position, and movement, and more or less complete loss of the sense of pain and the temperature sense. At times only the temperature sense is involved or the perception of heat may be lost and that of cold preserved, or *vice versa*. At times only extremes of temperature are confused and moderate degrees are well recognized (Dejerine). The distribution of the sensory loss follows the rules of segmental innervation (Laehr) in nearly all cases; in the extremities the loss is in bands folllowing the axis of the limb; over the trunk the changes occur in zones about the body. Even in the trigeminus the segmental type prevails (von Soelder). In certain cases, however, the so-called "geometrical" distribution of Charcot holds good; the analgesia or thermanæsthesia affects an entire hand, or the entire arm, or the arm and part of the trunk, etc.—"sleeve," "cuff" "waistcoat," forms of Charcot. This form exists without complicating hysteria (Brissaud, Schlesinger). Even within these plaques Laehr has demonstrated a tendency to segmental distribution of varying degrees of analgesia or thermanæsthesia.

In many cases tactile sensibility is normal, in others there are losses over small areas. Deep sensation may at times be involved, owing to the existence of posterior-column lesions. Ataxia is not infrequent. Stereognosis is often affected. Surface sensation and deep sensation are not necessarily affected to an equal degree; there may be loss of pain in deep structures, as bones and joints, while the skin sensations over these parts may be normal; usually, however, they are involved simultaneously. Frequently there is loss of testicular pain on pressure. The mucous membranes are commonly involved.

Pain is common and often severe; it may be constant, boring, not infrequently lancinating; it is felt oftenest in the arms and upper trunk; with high seat of the lesion

there may be occipital neuralgia or obstinate pain in the distribution of the trigeminus. Paræsthesiæ are usually present, often a feeling of cold or heat, or a mixed sensation of "burning cold." The pains and paræsthesiæ may persist in regions in which there is objective loss of sensation. The subjective sensations are important in diagnosis as they appear early and often call attention to atrophy and objective sensory changes. The paræsthesiæ of the temperature sense may be extreme and may lead to grave injury through the patient's ungoverned use of counteracting heat or cold. Analgesia and thermanæsthesia depend on lesions of the central gray matter and adjacent conducting paths. It must be remembered that dissociation of sensory qualities may not be an attribute of syringomyelia alone; it is frequent in hysteria and may occur in brain or spinal-cord lesions or in affections of peripheral nerves.

II. *Motor Phenomena.*—Atrophy is the symptom of chief import. It involves the arms most frequently, is often unilateral. The small muscles of the hand are most constantly affected. In early stages there may be the type of a peripheral median or ulnar lesion, manifesting itself in the form of the ape hand or the claw hand; combinations of the two are not infrequent. More frequent is the simultaneous atrophy of all the small hand muscles, the Aran-Duchenne type. The process may skip the forearm and spring to the shoulder group. At times all muscles undergo extreme wasting. The shoulder muscles may be attacked first; frequently individual bundles undergo atrophy, while others remain normal or become hypertrophied. The lower third of the trapezius is commonly involved, the upper part very rarely. The process is seldom symmetrical. The thorax muscles may be involved irregularly—primarily or after the arms. The intercostal muscles and diaphragm are usually spared. The lower extremities, as a rule, follow the arms in point of time. The quadriceps and other muscles of the calf of the leg (foot extensors) are the common seats of atrophy. Various forms of club-foot occur. At times atrophy is masked by fat or diffuse œdema.

Fibrillary twitching is frequent; it may be an early symptom and is often felt by the patient. Tremor, choreiform twitching, and other spontaneous movements occur; they may or may not be limited to atrophic muscles, and are often associated with paræsthesiæ or pain. Tonic cramps occur most often in the lower extremities, but they may involve many muscles and simulate at times hysterical seizures. Gradations toward myotonia have been observed, muscles becoming rigid on exposure to cold. The myotonic reaction has been observed. Contractures are frequent in later stages and may lead to great distortion. The gait shows usually a spastic paraplegia; ataxia is not infrequent; cerebellar gait is rare. All forms of electric reactions may be demonstrated. Often there is only quantitative loss; at times the reaction of degeneration may be found in separate muscle bundles; at times complete reaction of degeneration is present.

III. *Trophic Changes.*—The manifold trophic disturbances of syringomyelia speak rather for separate trophic nerves and centres. Trophic changes may exist without demonstrable loss of sensation. It is difficult to classify the diverse skin lesions that may occur. Peculiarities of sweat secretion are common: hyperidrosis, anidrosis, unilateral or regional sweating, sweating in response to cold and not to heat. Hyperidrosis may be an early symptom, its distribution may correspond to the sensory changes.

Scars of injuries and burns, panaritium (often painless), eczema and deep fissures, blisters, erythema, urticaria, and pemphigus are frequently seen.

Thickening of the skin of the palms and of the fingers may be extreme. Firm œdema of the hands and the peculiar thickening of the hands—the *main succulente* of Marinesco—are not uncommon.

Scleroderma has been noted in a few cases; Raynaud's symptom-complex is rare, gangrene not uncommon. Bedsores may develop in acute cases. Keloid is common. The nails are often thick, brittle, and deformed. Aside from the presence of a tuft of hair over a spina bifida or a spina bifida occulta, an overgrowth of hair rarely occurs.

Joint lesions are most important. They may be the earliest symptom; eighty per cent. affect the upper extremities. Pain may be a prodrome, but usually the developed affection is painless—a striking feature. The change in the joint may come on suddenly, with large effusion or with marked deformity and grating. The changes are due to trophic and not to mere mechanical influences. Atrophic and hypertrophic forms occur. Extreme deformity may result. The finger, elbow, and shoulder joints are the usual seats of trophic change. The lower extremities are more rarely involved. Exostoses about the joints and ossification of muscles are not infrequent. Suppuration may occur. The lesions develop spontaneously or under the influence of trauma, which may be slight.

Fractures of bones may occur spontaneously or with insufficient cause. There is a great preponderance of forearm fractures. Healing may be long delayed and incomplete. Exostoses may form. At times there is overgrowth of the bones as a whole or enlargement of an entire extremity, or of the hand or foot. Usually the enlargements are partial and irregular. They may develop quickly and be associated with inflammation. Hitzig described a case with unilateral bulbar lesions and hypertrophy of the face on the same side. I have seen great enlargement of an arm due to recurrent lymphangitis from panaritium infection.

Two cases of spontaneous tendon rupture have come under my observation: in one there was rupture of the long head of the biceps of the right (affected) arm; in the other, rupture of the patellar tendon in a lumbo-sacral type of the disease.

Thorax Deformities.—Bernhardt was the first to call attention to the frequency of scoliosis and kyphoscoliosis. Scoliosis is important as a symptom; it may occur early and independently of muscle weakness. The kypho-scoliosis occasionally leads to great deformity. It is most frequent in the dorsal region. Sometimes there is great tenderness of the spine, which may be limited to the extent of the deformity or of the process within the cord. Oppenheim regards the scoliosis as a congenital anomaly in certain cases.

Spina bifida and spina bifida occulta are not uncommon.

The *thorax en bateau* is the name given by Marie to a peculiar depression of the upper part of the sternum; it is often associated with subluxation of the clavicles forward.

Hemiatrophy of the face has been reported and may or may not be associated with lesions of the sympathetic.

In this connection may be mentioned the peculiar irregular or "crooked" look of many patients afflicted with syringomyelia. This seems to depend on irregularities of head, face, and thorax, on irregular thickening of soft parts, and on irregular innervation of facial muscles. It is important as emphasizing the influence of congenital anomalies in the pathogenesis of the disease.

IV. *Reflexes.*—The arm reflexes are often absent, and when present they follow the atrophy or the affections of the joints. Increased knee-jerks form a cardinal symptom. Clonus is frequent; unilateral increase is common; even with lumbo-sacral localization the knee-jerk is curiously persistent and usually increased. Absent knee-jerk is found in association with tabes or nerve-root lesions or destruction of lumbar centres. The superficial reflexes are not often absent; abdominal and cremaster reflexes are frequently increased. The Babinski reflex is not constant; its absence is not against syringomyelia, even when there are marked spastic symptoms.

V. *Sphincter Disturbances.*—The bladder is not generally affected early and may never be involved. Sensibility of the mucous membrane may be lost and yet the function be perfect. Difficulty of starting the urine is the usual symptom in the beginning; later there may be incontinence. Diabetes may occur in bulbar lesions. Pollakiuria is frequent and may be bothersome.