SPINAL CORD

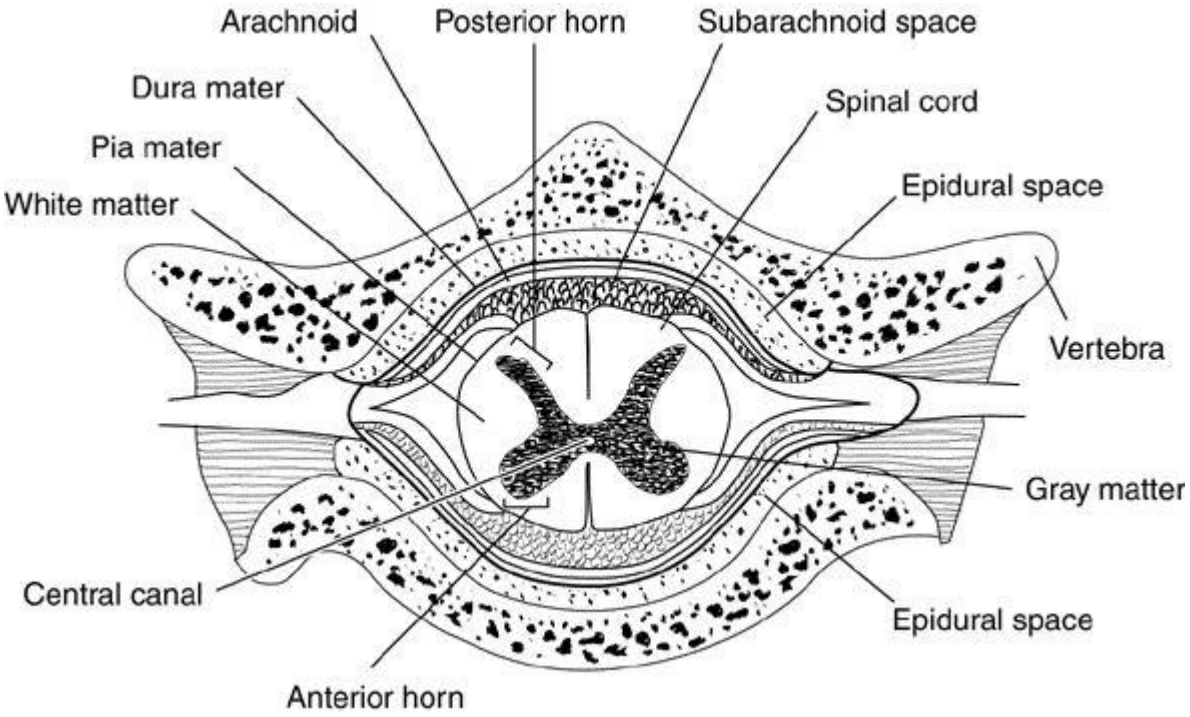
The spinal cord is an elongated, nearly cylindrical portion of the nervous system that is continuous with the medulla above, roughly at the level of the foramen magnum, and ends in a conical extremity, the conus medullaris. The average length of the cord is 42 cm in women and 44.7 cm in men. In normal adults the cord ends between the twelfth thoracic vertebra (T_{12}) and the lower third of the third lumbar vertebra (L_3). The usual level of termination is the lower third of the first lumbar vertebra (L_1).

During the process of maturation the vertebral column elongates more than the spinal cord, and in the adult the spinal cord is shorter than the vertebral column and the spinal nerves course downward before making their exit through the intervertebral foramina. Those that originate in the lower part of the spinal cord become more and more oblique in their downward descent. The lumbar and sacral nerves descend almost vertically to reach their points of exit; these are designated *the* ***cauda equina***. Consequent to this developmental change, there is a discrepancy between the segments of the spinal cord and the level of the spinous processes of the vertebrae. In the upper cervical area the cord level is about one segment higher than the corresponding spinous process; in the lower cervical and thoracic areas there is a difference of about two segments, whereas in the lumbar region there is a difference of almost three segments This elevation in the level of the caudal tip of the cord relative to the vertebral column is spoken of as ***the ascensus of the cord***. The ascensus is sufficiently far along even in premature infants that a lumbar puncture needle can be inserted between the lower lumbar vertebrae without fear of injuring the cord, unless a congenital anomaly tethers the cord, preventing the ascensus. The spinal cord is slightly flattened in an anteroposterior direction. Cervical and lumbosacral enlargements are seen on its surface at the level of C_5-Th_1 (C_{III}-C_{VIII} vertebrae) and L_1-S_2 (T_X-T_{XII} vertebrae) respectively.

An *anterior median fissure* and a *posterior median sulcus* divide it into two symmetrical halves. In the cervical region only, a shallow dorsal intermediate sulcus separates the dorsal columns into the *fasciculus gracilis* and the *fasciculus cuneatus*. The entry line of the dorsal root filaments is indicated by a shallow *dorsolateral sulcus*; that of the ventral roots, by a *ventrolateral sulcus*. The cord is divided into longitudinal segments or columns by these external features. A *dorsal column* is demarcated by the dorsal median sulcus and the dorsolateral sulcus. A *lateral column* extends from the dorsolateral sulcus to the ventrolateral sulcus along the line of attachment of the ventral roots. A *ventral column* extends from the ventrolateral sulcus to the anterior median fissure.

Caudally, the cord tapers to form the *conus medullaris*, from which the fourth and fifth sacral and coccygeal nerves arise. The cord continues caudally from the conus as a thin filament, the *filum terminale*, which ultimately fuses with the dura at the *dural cul-de-sac* at about S_2. The ilium is surrounded by the longitudinally coursing nerve roots that run from the lumbosacral region down to exit through their proper inter-vertebral or sacral foramens. The lumbosacral nerve roots are called the ***cauda equina***.

The spinal cord is surrounded by *pia mater*, *arachnoid*, and *dura mater*. The pia mater is a delicate membrane that closely invests the spinal cord. The arachnoid is a transparent membrane that is close to the inner surface of the dura, but fine strands extend to the pia. The dura mater is a strong, fibrous membrane, penetrated by the nerve roots, which forms a firm, tubular sheath. It is separated from the wall of the vertebral canal by the ***epidural space***, which contains areolar tissue and a plexus of veins. *The* ***subdural space*** is a potential space containing a small amount of fluid. *The* ***subarachnoid space***, which extends to about the level of the second sacral vertebra, is a well-defined cavity containing cerebrospinal fluid. The dentate ligaments extend along the lateral surface of the spinal cord, between the anterior and posterior nerve roots, from the pia to the dura mater. They suspend the spinal cord in the vertebral canal.



Attached to the spinal cord are *31 pairs of nerves*: 8 cervical (C), 12 thoracic (T), 5 lumbar (L), 5 sacral (S), and one or more (1-3) rudimentary coccygeal pairs. The spinal nerves are formed by the union of a dorsal and a ventral root. This union occurs at the intervertebral foramens. In most instances, C_1 consists only of a ventral root.

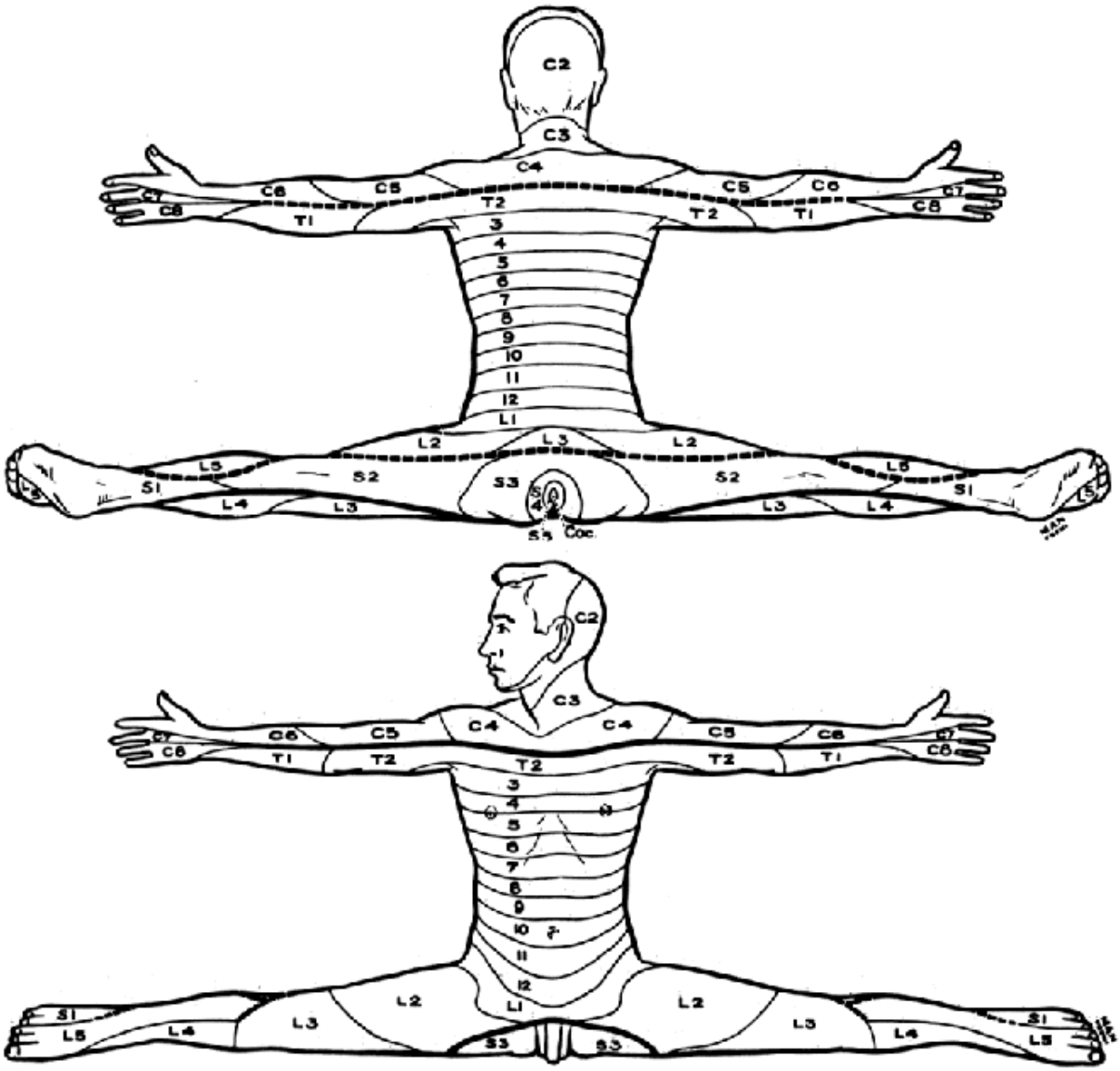
The numeration system and the relationship of the spinal nerves and roots to the vertebrae must be understood for lesion localization. The C_1 nerve root exits above the C_1 vertebra and the C_8 root exits below C_7. Thus, there are eight cervical nerve roots but only seven vertebrae. While the spinal cord and vertebral levels roughly correspond in the cervical region, the ascensus of the cord causes an increasing downward angulation of the more caudal roots.

INTERNAL STRUCTURE

On cross section the spinal cord is seen to be divided into an H-shaped core of gray matter and a surrounding white matter. The superior limbs of the H are called the *dorsal horns* and the inferior, the *ventral horns*. This gray matter consists largely of neurons and glia, while the white matter consists of nerve fibers and glia. Glial supporting tissue extends a short distance into the nerve roots to meet the Schwann cells and the collagenous supporting tissue. Within the center of the gray matter and running throughout the entire length of the cord is a minute remnant of the ***central canal*** consisting of a single layer of ependymal cells.

SEGMENTAL INNERVATION OF SKIN AND MUSCLE

Reflecting the subdivision of the developing embryo into somites is the arrangement of the spinal nerves. Each somite receives its own spinal nerve. The neuraxis itself is not segmented, but a spinal cord segment is defined as the length of cord between the rostralmost filaments of one dorsal root and the rostralmost filaments of the next root below. In the thoracic region the spinal nerves retain their original simplicity, with one nerve going to each somite without anastomosing with its neighbor. At the level of the arm and leg, the somites have undergone a dramatic renovation. Since the original nerve supply from the proper spinal segment is retained, the spinal nerves have to rearrange themselves by anastomosing in the brachial and lumbosacral plexuses to reach their original somite territory. The dermatomes, myotomes, and sclerotomes derived from each somite do not always correspond in position. Thus, the area of sensory innervation does not always overlie the muscles innervated by the same segment. On the whole, the sensory pattern is more regular than the motor pattern



The segmental innervation of muscle provides a basis for lesion localization. The exact segmental details differ from observer to observer but hold in main outline

SYMPTOMS, SIGNS, AND LOCALIZATION OF SPINAL CORD LESIONS

The spinal cord is essential to the regulation and administration of various motor, sensory, and autonomic activities of the body. By means of its segmentally arranged spinal nerves and its nuclear centers, it receives impulses at various levels and carries them to motor cells in the same or adjoining segments for distribution to appropriate muscles. Thus it provides for reflex action and governs motor activity. By means of its descending pathways from higher centers, it regulates and inhibits spinal cord reflexes and motor activity. Through its ascending pathways, it conducts impulses from the extremities, trunk, and neck to higher centers and to consciousness. It also has a regulatory and administrative action over various visceral activities. In disease of the spinal cord any or all of these functions may be affected, and one may often localize spinal cord lesions in both the transverse and the longitudinal planes by the neurologic examination.

Lesions of the spinal cord are characterized by sensory, motor, and autonomic changes. The re-suiting symptoms depend upon the location and extent of damage to various functional elements, and often upon the type of damage and the rapidity with which the lesion develops. If there is involvement of the ***posterior roots*** or of the sensory cells in the ***posterior horns*** of the gray matter, there are segmental sensory changes; there may be either a loss of certain or all varieties of sensation in the dermatomes supplied by the involved segments, or irritative

phenomena such as pain and paresthesias. If there is involvement of the ***ascending pathways***, loss of sensation, principally of the pain, temperature, and proprioceptive modalities, develops below the lesion. There is often a dissociation of sensation, with loss of some varieties but sparing of others. If there is involvement of the ***anterior horn cells*** or of the ***anterior roots***, there is a ***lower motor neuron (or peripheral) paralysis*** in the myotomes supplied by the involved segments, occasionally with either fasciculations or muscle spasm. If there is involvement of the ***descending motor pathways***, either corticospinal, extrapyramidal, or vestibulospinal, there are changes in motor power and tone below the level of the lesion ***(central paralysis)***. If there is involvement of the ***intermediolateral cell group*** of the gray matter, the neuraxes of these cells, or the descending autonomic pathways, there are changes in autonomic function. Alterations of the reflexes, ataxia, disturbances of gait, dysfunction of the sphincters, and other abnormalities of function are all secondary to either isolated or combined motor, sensory, or autonomic involvement.

A lesion at the C_8 and Th_1 segments interrupts the sympathetic innervation of the face, by affecting either the descending pathway from the hypothalamus to the spinal cord or the peripheral connections and thus may cause a Horner's syndrome (unilateral miosis, ptosis, and ipsilateral hemifacial anhidrosis).

TYPES OF SPINAL CORD LESIONS

There are many varieties of spinal cord lesions. Transverse syndromes are characterized by complete interruption of the continuity of the spinal cord; there is loss of all motor, sensory, and autonomic function below the level of involvement. Incomplete transverse lesions are followed by loss of function of certain portions of the cord; there may be dysfunction of onehalf, one-quarter, or a certain portion or segment of the cord. Syndromes of the gray matter show segmental loss of function of certain cell groups. Disease of the ***anterior horn*** is followed by a segmental flaccid paralysis (anterior poliomyelitis, progressive spinal muscular atrophy); disease of the ***posterior horn***, by segmental sensory changes (commonly exteroception ims impaired while proprioception remains intact) and loss of muscle stretch reflexes as afferent part of the reflex arc is damaged; disease of the ***lateral horn***, by autonomic changes (e.g., Horner syndrome if lesion affects C_8-Th_1 segments) disease of the ***gray commissure***, by dissociation of sensation: loss of pain and temperature sensation with spared tactile sensation and proprioception (syringomyelia).

Syndromes of the white matter cause interference with the ascending and descending pathways. In disease of the ***dorsal funiculi*** there is interference with the ascending proprioceptive or related impulses that results in loss of proprioception and thus in sensitive ataxia. In disease of the ***lateral columns***, the lateral corticospinal, lateral spinothalamic, or other tracts may be affected, that bring about central palsy of muscles on side of the lesion just below its level, and loss of pain, temperature and tactile sensation on the level of 2-3 segments below the lesion. System disease is characterized by dysfunction of anatomically and functionally related systems of cells or fibers (spinocerebellar degenerations, posterolateral sclerosis, amyotrophic lateral sclerosis). Disseminated disease is manifested by patchy involvement, with many lesions of a focal nature (multiple sclerosis). Diffuse disease shows widespread involvement but some cells or fibers may be affected more than others.

Complete Transection

Complete transection of the spinal cord, whether of traumatic, neoplastic, vascular, or other origin, causes isolation of the segments below the level of the lesion. The upper portions of the cerebrospinal axis function normally, but motor, sensory, and autonomic functions are lost distal to the lesion. The term ***transverse myelitis*** is often used for syndromes that result in complete transection, even though the process may not be inflammatory in origin; in most cases the term transverse ***myelopathy*** is more appropriate.

If a transverse lesion is abrupt in onset, the state of spinal shock or ***diaschisis*** occurs. There is flaccid paralysis together with loss of all types of sensation, absence of autonomic function, and areflexia below the level of the lesion. Spinal shock usually lasts for about 3 or 4

weeks, after which the reflexes gradually return and become exaggerated, pathologic reflexes appear, muscle tone becomes increased, and the nature of the bladder and rectal dysfunctionbecomes altered. If infection supervenes, however, in the form of a severe urinary tract involvement or infected decubiti, the period of spinal shock is prolonged. In transverse myelopathy there is a general loss of all types of sensation below the uppermost level of the lesion, and the lowest level of preserved sensation corresponds to the dermatome supplied by the lowest intact segment. This is most apparent and most clearly delineated with the exteroceptive sensations, especially the superficial pain and temperature modalities. There is loss of these sensations below the level of the lesion, or within one or two segments of the level of the lesion. There may be hyperesthesia at the level of the lesion. The level for tactile sensation is seldom clearly delineated; proprioceptive sensations are also lost, but it is difficult to demonstrate a specific level for these.

After the initial period of flaccidity, the musculature below the level of the lesion becomes spastic, or sometimes spastic and rigid. There is increased tone, with increased resistance to passive movement. The muscle stretch reflexes return and then become hyperactive. The superficial reflexes do not return. Corticospinal tract responses may appear, but they do not do so as characteristically in complete as in incomplete transverse syndromes. There may be an exaggeration of sweating and piloerection below the level of the lesion, with changes in skin temperature and cutaneous vascular function. The bladder, first atonic and distended, with retention and overflow incontinence, becomes small and contracted; the patient develops a reflex type of neurogenic dysfunction. There may be priapism. With the development of these changes, reflexes of spinal automatism appear. These may be elicited by nociceptive stimuli up to the level of the lesion. They are usually of the uniphasic, or flexor, variety, and frequently are accompanied by the mass response with urination, defecation, and sweating below the level of the lesion.

The characteristic position in complete transverse lesions is one of flexion of the lower extremities. The paralysis and sensory loss are symmetric and total; voluntary power does not return; vasomotor and sphincter disturbances are evident; corticospinal tract responses are usually minimal, but defense responses are definite; there is a marked tendency toward the development of decubiti. Inasmuch as the response is mainly one of flexion, the extensor reflexes may be difficult to elicit. With transverse lesions of long duration there may be metabolic alterations, including increased excretion of proteins, fall in serum protein, increase of potassium and decrease of sodium and chlorides in the blood and tissue fluids, hypercalciuria, osteoporosis, testicular atrophy, gynecomastia, altered urinary excretion of 17-ketosteroids, and orthostatic hypotension.

No functional regeneration has been observed in man following complete transverse lesions. With incomplete transection, however, or gradual compression of the cord, there may be recovery of function.

The Brown-Sequard syndrome

The Brown-Sequard syndrome follows the lateral hemisection of the cord. Ipsilateral features include upper motor neuron paralysis of the parts distal to the lesion and loss of tactile discrimination, position sense, and vibration in these parts. The major contralateral feature is loss of pain and temperature discrimination distal to the lesion because of crossing of the spinothalamic tracts which mediate these modalities. Usually the level for pain and temperature loss is one or several segments below the level of the lesion. At the level of the lesion, some segmental motor or sensory signs may occur if the lesion damages dorsal or ventral roots. A quadrantic lesion of the spinal cord that involves the dorsal quadrant causes ipsilateral loss of proprioceptive sensation and corticospinal paresis below the level of the lesion. Involvement of the ventral quadrant causes an ipsilateral segmental flaccid paralysis, due to involvement of the anterior horn cells, together with a contralateral loss of pain and temperature sensations below the level of the lesion.

Pure hemisection or quadrantic lesions of the cord are rare; incomplete or partially bilateral involvements are more common.

Table . Signs of the spinal cord lesion

A. The level of the lesion **C1-C5**: _ spastic palsy below the lesion (tetraplegia) _ flaccid palsy of muscles of head and neck and **diaphragm** _ sensory loss below the lesion _ upper neuron type of bladder dysfunction **C5-T1 (cervical enlargement)**: _ flaccid palsy of hands _ spastic palsy of legs _ sensory loss below the lesion _ upper neuron type of bladder dysfunction _ Horner's syndrome **T1-T12**: _ spastic palsy of legs _ sensory loss below the lesion _ upper neuron type of bladder dysfunction _ trophic changes in lower part of the body and legs **L1-S2 (lumbar enlargement)**: _ flaccid palsy of legs _ sensory loss in legs and saddle (paranesthesia) _ upper neuron type of bladder dysfunction	**L4-S2 (epiconus)**: _ flaccid palsy of posterior group of muscles of the leg, absent Achilles reflex _ sensory loss in shin, foot, buttocks and saddle (paranesthesia) _ upper neuron type of bladder dysfunction **S3-Co (conus)**: _ sensory loss in saddle region _ lower neuron type of bladder dysfunction _ trophic changes in sacral region **cauda equina (L2-S5)**: _ flaccid palsy in legs _ sensory loss in legs, buttocks and saddle _ lancinating pain _ tension symptoms _ lower neuron bladder dysfunction **B. The site of the lesion** **anterior root**: _ segmental flaccid palsy **posterior root**: _ segmental sensory loss _ lancinating pain _ no reflexes _ tension symptoms **spinal nerve**: _ segmental flaccid palsy _ segmental sensory loss _ lancinating pain _ tension symptoms

THE BLADDER DYSFUNCTION

Although incontinence of bladder or bowel is common with lesions at any level of the spinal cord, it is especially frequent with lumbosacral or cauda equina lesions. Therefore, careful assessment of bladder and bowel function is indicated whenever a spinal lesion is suspected.

Normal urination (as well as defecation) is a complicated act requiring first of all the perception of fullness of the bladder. Bladder sensation is conveyed to the cord via the hypogastric and pelvic splanchnic nerves (*nervi erigentes*). Pain sensation may be mediated through both of these nerves; however, the sensation of fullness appears to depend primarily on the pelvic nerves. The puclendal nerve, which supplies the voluntary urethral sphincter muscle, does not appear to be involved in these bladder sensations. The afferent fibers for the sensation of fullness apparently enter the cord at several levels, up to approximately Th_7. Thus, if all sensation of fullness is lost, and the lesion is central, it is above Th_7. For a central lesion of the cord to abolish the sensation of fullness, it has to be extensive, involving much or all of the cord cross section. Probably pathways in both ventrolateral and posterior columns have to be interrupted. Bladder musculature has tonicity, rhythmicity, and is able to retain without discomfort either small or large amounts of urine; contractions can be stimulated either reflexly or voluntarily. Vesical function is a complex mechanism that involves both the autonomic and the voluntary nervous systems, and disorders of bladder function may follow lesions of the paracentral lobule, hypothalamus, descending pathways in the spinal cord, pre- or postganglionic parasympathetic nerves, or pudendal nerve. It is difficult to differentiate between disorders of function that are entirely autonomic in origin and those with associated voluntary nervous system involvement; these will be considered together. The various disturbances of vesical function may be appraised by cystometry.

The bladder acts as a reflex organ and contracts in response to a stretch reflex. The afferent impulses are carried to the sacral portion of the spinal cord, and stimulation of efferent centers causes contraction of the detrusor muscle and relaxation of the internal sphincter. There is probably a center in the lumbar spinal cord that produces a contraction of the internal sphincter and allows distention of the bladder and retention of urine. In the baby the bladder is purely reflex in function, but with the maturation of the cerebral cortex and the completion of myelination an inhibitory control over this reflex is developed, with voluntary regulation of the external sphincter. For normal micturition the parasympathetic arc, sympathetic arc, and spinal pathways must be intact, and cerebral inhibition and control of the external sphincter must be normal.

Normal urination requires the volitional ability to increase the intra-abdominal pressure by contracting the abdominal and chest muscles and diaphragm while at the same time relaxing the skeletal muscle sphincter of the urethra. Sacral cord reflexes acting through the parasympathetic innervation via the pelvic splanchnic nerves contract the smooth muscle of the bladder wall and the detrusor muscle unguards the urethra.

The sympathetic innervation of the bladder plays no essential role in urination. Urination is halted by contraction of the skeletal muscles of the bladder floor. Automatic urination can be accomplished solely by reflex mechanisms, even if all suprasegmental control has been eliminated by complete cord transection.

In the incontinent patient with a neurologic lesion, the clinician has to decide, as in any motor disturbance, whether the lesion involves the upper motor neuron mechanisms or is in the micturition centers in S_2 to S_4, or in the afferent or efferent connections of the reflex arc.

Basically, neurologic lesions of the cord or reflex arc affect urination in one of four ways:

1. interruption of descending pathways from the brain, thus an upper motor neuron lesion;
2. interruption of lower motor neurons of skeletal and smooth muscles or their reflex centers in the sacral cord;
3. interruption of afferent fibers from the bladder, or
4. combinations of 1 to 3.

1. Cord lesions rostral to the sacral segments may compromise ***upper motor neuron*** control of urination. The effect of the upper neuron interruption on the bladder depends on several factors, e. g., the acuity of the lesion and the effect on the voluntary sphincter. In the case of acute transverse lesions rostral to the sacral segments, the bladder may be flaccid and hypotonic during the stage of spinal shock. In most chronic lesions, the bladder ultimately becomes spastic and «hyperreflexic» in accordance with the usual effects of upper motor neuron lesions on motor function. The bladder empties automatically in response to minimal distention by urine. Since the bladder never distends to its full capacity, it ultimately contracts. The patient urinates frequently and involuntarily and voids only small quantities of urine. Although a small, contracted automatic bladder is the usual end stage of transverse cord lesions, in some instances the bladder may become huge and distended. This usually occurs when the voluntary sphincter is very spastic, preventing reflex emptying. While an automatically emptying bladder can be suspected from the history, it is confirmed by cystometrograms. Cystometrograms show a small filling capacity, early contractions, and reflex emptying long before the bladder reaches its usual capacity.

2. In the case of ***lesions of the lower motor neurons*** which innervate the bladder wall, the bladder becomes hypotonic and contracts sluggishly, or it may be paralytic. Sensation may be preserved, but the bladder wall distends and becomes very thin. Residual urine and bladder capacity are huge. Cystometrograms show a low intravesical pressure and absence or weakness of reflex contractions.

3. ***Deafferentiation*** of the bladder results in a huge, distended sac which may contain 1,000 to 1,500 ml of urine. The bladder wall becomes hypertrophic. The patient has no sensation of fullness. The absence of afferent impulses makes urination difficult or impossible to initiate.

The patient has overflow incontinence and huge quantities of residual urine. The principal types of neurogenic bladder dysfunction are the following.

The uninhibited neurogenic bladder shows the least variation from the normal. This type of bladder dysfunction occurs in mental deficiency, cerebral palsy, enuresis in adults, early diffuse brain damage, cerebral lesions affecting the dominant hemisphere, hemiplegia, and early multiple and posterolateral sclerosis. It is characterized by a more or less infantile type of reaction. There is a loss of the cortical inhibition of reflex voiding, while tone remains normal, so that when the bladder is distended it contracts in response to the stretch reflex. There is frequency, urgency, and incontinence not associated with dysuria. Hesitancy may precede urgency. Bladder sensation is usually normal. Several rhythmic uninhibited contractions of the detrusor may take place before bladder capacity is reached and the final emptying contraction occurs. These contractions coincide with the patient's awareness of an urge to void, but micturition does not take place until a contraction occurs that is of sufficient intensity to empty the bladder; then the patient urinates precipitously. Up to this point, however, normal voluntary control is possible. There is no residual urine.

The reflex neurogenic bladder occurs with widespread disease of the spinal cord in which both the descending autonomic tracts to the bladder and the ascending sensory pathways are interrupted above the sacral segments of the cord. Thus, it occurs in patients with transverse myelitis, advanced multiple sclerosis, neoplasms, and traumatic and vascular lesions sufficiently extensive to cause a functional transection of the spinal cord. Extensive brain lesions may also cause the development of a reflex neurogenic bladder. All sensation to the bladder is lost; the patient cannot feel heat, cold, or distention. The bladder capacity is small. Micturition is reflex and involuntary. The patient cannot either initiate or stop urination in a normal way, and micturition is precipitous. There are rhythmic uninhibited contractions during filling. The patient may become aware of the presence of a full bladder by autonomic reflexes activated by distention of the bladder- these include sweating, pilomotor phenomena, increased spasticity, and a feeling of fullness in the abdomen—and he may be able to initiate urination by pinching the skin in the perineal region or pressing over the abdomen or bladder. The bladder may empty as part of the mass reflex of spinal automatism. The residual urine volume is variable.

The autonomous neurogenic bladder is one without external innervation. It is caused by lesions of the sacral portion of the spinal cord, the conus medullaris or cauda equina, the motor or sensory roots of the second, third, and fourth sacral nerves, or the peripheral nerves. It occurs with neoplastic, traumatic, inflammatory, and other lesions of the conus medullaris, cauda equina, or sacral nerve roots, and with congenital anomalies such as spina bifida. There is destruction of the parasympathetic supply. Sensation is absent and there is no reflex or voluntary control of the bladder; contractions occur as the result of stimulation of the intrinsic neural plexuses within the bladder wall. There are no sustained contractions of the detrusor as a whole, and no emptying contractions. During filling, however, there are minor inherent contractions of individual muscle groups, and at the height of one of these there may be emptying, which is never complete. There may be a high intravesical pressure, and the amount of residual urine is large, but the bladder capacity is not greatly increased. On neurologic examination saddle anesthesia and absence of the bulbocavernosus reflex are found. Patients with this type of bladder dysfunction may have incontinence on coughing or straining. Voiding is usually brought about by increasing the intra-abdominal pressure, and there may be dribbling as a result of the high intravesical pressure. The desire to void is made known by abdominal discomfort.

Treatment consists of teaching the patient to empty his bladder as completely as possible by manual external pressure and increased intra-abdominal pressure.

The sensory paralytic bladder, also known as ***the atonic neurogenic bladder***, is found with lesions that involve the posterior roots or posterior root ganglia of the sacral nerves or the posterior columns of the spinal cord. This type of bladder is present in patients with tabes dorsalis, posterolateral sclerosis, multiple sclerosis, diabetic autonomic neuropathy, and related conditions. Sensation is absent, and there is no desire to void. There may be distention, dribbling,

and difficulty both in initiating micturition and in emptying the bladder. There is low intravesical pressure and a large capacity, with absence of waves of contraction, and a large amount of residual urine. Voiding may be brought about by straining. There is incontinence of an overflow type. There is a large amount of residual urine. The bulbocavernosus reflex is absent. Bethanechol is helpful in treatment.

The motor paralytic bladder develops when the motor nerve supply to the bladder is interrupted. Among the etiologies are poliomyelitis, polyradiculoneuritis, neoplasm, trauma, and congenital anomalies. The bladder distends and decompensates, but sensation is normal. The patient complains of painful distention and inability to initiate urination. If the condition becomes chronic the symptoms are similar to those of obstructive uropathy. No contractions of the detrusor are observed and the patient cannot initiate micturition. The residual urine and bladder capacity vary with each individual. Saddle sensation is normal and the bulbocavernosus reflex may or may not be present.

With spinal cord lesions, especially those of severe degree and sudden onset, such as traumatic myelopathies, there is at first marked urinary retention during the period of spinal shock. Reflex activity is absent; the bladder is atonic and may become markedly distended, with overflow incontinence. Later it becomes autonomous in function, owing to reflex contraction from the plexuses in the bladder wall. If the patient develops a spastic paraplegia, there will be a reflex bladder with small capacity and precipitate micturition. The paralyzed bladder not infrequently becomes infected, and chronic infection leads to contraction of the viscus and often to continual dribbling. There may be calculus formation.

With cerebral lesions there may be incontinence at the onset of the lesion, owing to abolition of inhibitory control, or there may be retention with distention of the bladder and overflow incontinence. The mental apathy associated with frontal lobe lesions may lead to involuntary micturition. Vesical dysfunction may be associated with disease of either the corticospinal or extrapyramidal motor systems. Both urinary retention and incontinence may have a psychogenic origin. There are many causes, both organic and nonorganic, for nocturnal enuresis in childhood and adolescence.

The determination of the condition of the bladder is as much a part of the neurologic examination in the patient suspected of having a cord or lumbosacral lesion as is the examination of the somatic motor and sensory systems. The first step in the analysis of bladder function is to palpate and percuss the suprapubic region. A huge bladder is easily detected by these means. Next the residual urine is determined and consideration is given to anchoring a catheter. Further evaluation of bladder size and physiology involves cystometrograms and radiographic contrast studies of bladder and renal function.