fourth edition

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ORTHOPAEDICS

Principles & Their Application

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Anterior Median Fissure. This fissure is lined by an overlapping fold of pia and dips into the greater part of the anterior portion of the spinal cord.

Anterior Lateral Sulcus. This fine depression lies about midway between the anterior median fissure and the lateral margin of the spinal canal and marks the exit of the fila of the anterior roots.

ARTERIES OF THE SPINAL CORD

The spinal cord derives its blood supply from the vertebral artery and a series of spinal rami that enter the intervertebral foramina at successive levels (Color Plate 14-4).

Posterior Spinal Artery. This branch of the vertebral artery begins near the lateral margin of the medulla oblongata and descends on the dorsolateral surface of the spinal cord posterior to the spinal roots. In its downward course to the cauda equina, the posterior spinal artery receives a succession of small arterial branches that enter the spinal canal through the intervertebral foramina. These vessels and their branches anastomose freely around the posterior roots and with the corresponding vessels on the opposite side, dipping also into the substance of the spinal cord; in the midline they form the posterior central artery.

Anterior Spinal Artery. This artery is formed by the union of two branches from the terminal portion of the vertebral artery at the level of the foramen magnum. The artery descends as a single trunk on the anterior aspect of the spinal cord to the conus medullaris. It continues along the cauda equina and ends as a fine arteriole accompanying the filum terminale. At successive levels, it, too, is reinforced by spinal branches entering through the intervertebral foramina. Along its course small twigs from this artery enter the substance of the spinal cord, and in the anterior median fissure these form the anterior central artery.

Spinal Branches. These branches arise at various levels from the sacral, iliolumbar, intercostal, inferior thyroid, and vertebral arteries, which enter the spinal canal through the intervertebral foramina. Each spinal branch divides into two rami: (1) a peripheral ramus, which, after entering the spinal canal, divides into an ascending and a descending branch and then anastomoses with the one above and below to form two lateral chains on the posterior surfaces of the vertebral bodies near the junction of the pedicles and (2) a central ramus, which supplies the spinal cord and it membranes by dividing into anterior and posterior arteries that anastomose with the anterior and the posterior arteries of the spinal cord.

VENOUS DRAINAGE OF SPINAL CORD AND VERTEBRAL COLUMN

Outside and inside the vertebral canal, running along the entire length, are series of venous plexuses that freely anastomose with each other and end in intervertebral veins.

Two groups of venous plexuses are found outside the vertebral canal: (1) the anterior group, which lies in front of the vertebral bodies and receives some venous tributaries from vertebral bodies and communicates with the basivertebral and the intervertebral veins, and (2) the posterior group, which forms a network of venous plexuses spreading over the spinous processes, laminae, facets, and adjacent deep musculature. In the cervical region these veins communicate with the deep cervical occipital and cerebral veins.

The venous plexuses inside the vertebral canal lie between the dura and the inner vertebral surfaces. These veins receive tributaries from the adjacent bony structures and the spinal cord. Although they form a close network, running vertically within the spinal canal, they may be subdivided into a pair of anterior internal venous plexuses of veins, which lie on either side of the posterior longitudinal ligament and into which basivertebral veins empty, and into a single posterior internal venous plexus of veins, which lies anterior to, and on either side of, the vertebral arches and the ligamentum flavum, which anastomoses with the posterior external veins.

These plexuses form almost a series of venous rings at the level of each vertebra, found most strikingly at the foramen magnum.

Tunneling the bony structure of each vertebral body is the basivertebral vein, which has a small valvelike opening as it joins the anterior internal venous plexus.

The intervertebral veins leave the spinal cord through the intervertebral foramina in company with the intercostal, the lumbar, and the sacral veins.

The veins of the spinal cord are minute and delicate. They emerge from the anterior median fissure as the anterior central vein and from the posterior sulcus as the posterior central vein. There are also two lateral longitudinal veins, on either side of the spinal cord, and they all empty into the intervertebral veins. However, those near the foramen magnum empty into the inferior petrosal sinus of cerebellar veins.

DERMAL SEGMENTATION

Sensation from the outside world reaches consciousness through sensory impulses. Most of these are carried by afferent nerve fibers to the spinal cord and up to the brain.

The nerve fibers that carry sensation of pain, temperature, touch, vibration, position sense, and other discriminatory sensibilities have their cells of origin in the

spinal ganglia, from which fibers also arise to make up the dorsal root.

The fibers that carry the impulses of sensation from the skin, the muscles, and the joints are arranged in segments, which in simplest form are found in the thoracic region as broad bands. In the upper and the lower limbs the sensory arrangement is more complicated but follows a vertical pattern in each limb.

It has been found that if a dorsal root is sectioned, complete anesthesia of the involved dermal segment does not follow because of an overlapping of sensation by the nerves above and below the affected dermal segment. It has been further established that each sensory nerve carries impulses not only from its own dermal segment but also from the ones above and below. This overlapping of cutaneous sensation is known as metamerism.

If one is familiar with the cutaneous distribution of various nerve roots, it is possible to localize with great accuracy the site and the level of any pathologic disturbance.

A chart outlining the exact sensory dermal segments is a good reference (Fig. 14-2), but it is valuable to remember some surface landmarks that will serve as a general guide to localization:

The clavicle is supplied by C3 sensory root.
The deltoid is supplied by C5 sensory root.
The nipple area is supplied by T4 sensory root.
The intercostal margin is supplied by T7 sensory root.
The umbilicus is supplied by T10 sensory root.
The groin region is supplied by T12 sensory root.
The lateral aspect of the forearm and hand is supplied by C6 and C7 sensory roots.
The inner aspect of the forearm and hand is supplied by C8 and T1 sensory roots.
The anterior surface of the thigh and inner surface of the leg is supplied from above down by L1, L2, L3, and L4.
The outer and posterior surfaces of the legs are supplied by L5 and S1 and S2 sensory roots.
The perineum is supplied by S3, S4, and S5 sensory roots.

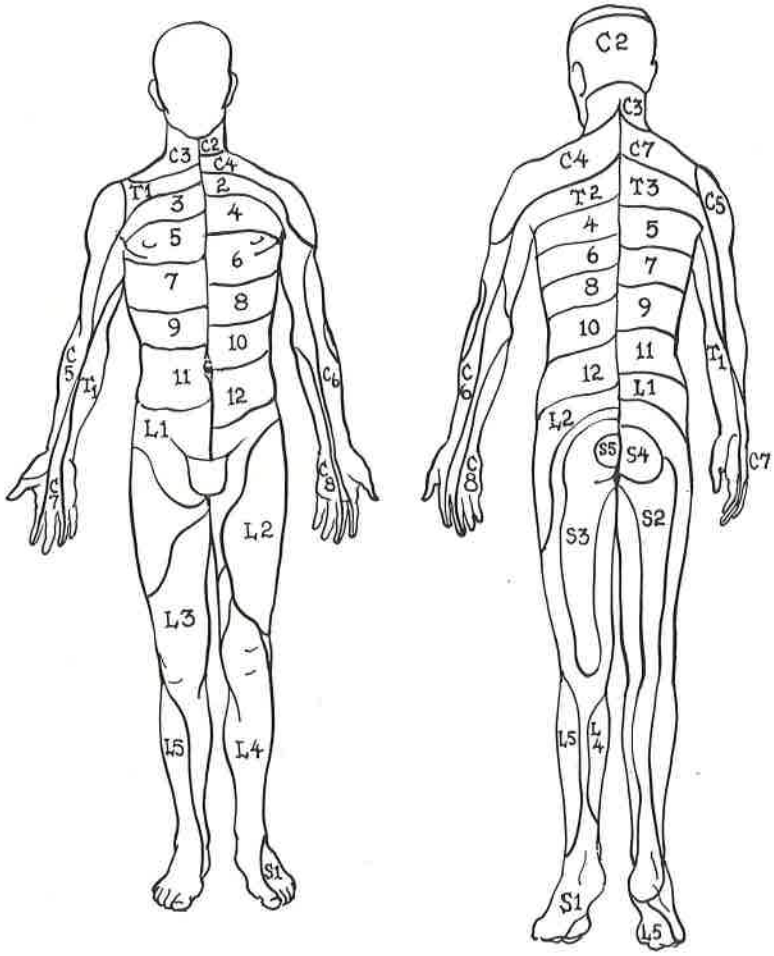


FIG. 14-2. Distribution of spinal dermatomes. Considerable overlap occurs; consequently, involvement of a single spinal segment may not be evident.

NEUROLOGIC DIAGNOSIS

Diagnosis of orthopaedic conditions requires a basic understanding of neurology.[2] A simple neurologic examination ordinarily suffices to differentiate the majority of neurologic disorders.

MOTOR FUNCTION

Disturbance of muscle power varies from paresis (weakness) and paralysis (complete loss) to hyperkinesia (increased muscular movements). The patient may be able to move a muscle; yet paresis may be demonstrated by inability to perform a movement against resistance. Monoplegia defines paralysis of a single extremity; hemiplegia is paralysis of a unilateral half of the body; diplegia or brachial paraplegia is paralysis of both upper extremities; and paraplegia is paralysis of both lower extremities. The paralysis is due to either an upper or a lower motor neuron lesion.

The upper motor pyramidal cells of the cerebral precentral cortex send fibers through the corticobulbar and the corticospinal tracts to the motor cells of the cranial nerves and of the anterior horns of the spinal cord on the opposite side. These tracts undergo partial decussation at the caudal end of the medulla before continuing distally into the spinal cord. The major portion of the fibers cross to the opposite side, forming the lateral corticospinal tract; the smaller uncrossed portion continues downward as the ventral corticospinal tract. The lower motor neurons are also under the influence of various other motor centers in the brain, located chiefly in the basal ganglia and the cerebellum.

LESIONS

Upper motor neuron lesions produce a spastic paralysis characterized by increased muscle tone, increased deep reflexes, diminished or absent superficial reflexes, and demonstrable pathologic reflexes such as the Babinski, the Oppenheim, the Gordon, and the Chaddock. These findings are explained by the removal of the inhibitory impulses of the cerebral centers.

A *lower motor neuron lesion* is characterized by a flaccid paralysis (loss of muscle tone), absent deep reflexes, muscle atrophy, and the reaction of degeneration. This type of paralysis may be produced by disease or injury to the anterior horn cells, the anterior roots, the peripheral nerves, the nerve plexuses, or the cauda equina.

Hyperkinesia is a condition of excessive, involuntary, purposeless movements.

Tremors are rhythmic, oscillating movements affecting all or groups of muscles. *Intention tremors* are characteristic of multiple sclerosis.

Tonic spasms are prolonged, intense, muscular contractions. *Clonic spasms* are rapid, repeated contractions of muscles.

A *cramp* is a tonic spasm localized to one muscle. Persisting spasm of a muscle eventually leads to its contracture. Continued spasm of a group of muscles overcoming their antagonists may cause joint contracture.

Choreiform movements are quick, uncoordinated, irregular, and arrhythmic; they are characteristic of chorea, which commonly follows rheumatic fever.

Athetosis, which is due to basal ganglia damage and often associated with hemiplegia, is characterized by a recurring series of slow, vermicular, "pill-rolling" movements of the hands.

Myotonia is a condition of increased muscle tonus that is brought on by emotion and attempts at movement.

Lesions of the corpus striatum are commonly associated with cerebral palsy. They are characterized by athetochoreic movements, rigidity, tremor, loss of associate movements, and masked facies.

Synergic movements are governed by the cerebellum. *Adiodokokinesis* is the inability to accomplish synergic movements (*e.g.*, the patient is unable to perform rapidly and alternatingly supination and pronation with both hands at the same time).

Hypotonia is a decrease of muscle tonus associated with muscle atrophy. Hypotonia also arises when lesions interrupt transmission of deep sensation (*e.g.*, in tabes dorsalis).

Complicated coordinated movements are examined by observing the manner of walking to check for *gait disorders*. Paresis will produce a slow, guarded, short-stepped, shuffling gait. Paralysis of the anterior tibial muscles, especially by an anterior horn or peripheral nerve lesion, causes a dropfoot and produces a steppage gait. To avoid tripping over the plantar flexed foot, the extremity is advanced with knee and hip hyperflexed. With spasticity, the legs are advanced slowly with shortened steps and the toes scraping the ground. Adductor tightness produces a scissors gait, by which the legs are alternately crossed. In the ataxic or tabetic gait, because of absence of deep position sense, the patient must constantly observe the placing of his feet; The hip is hyperflexed and externally rotated, and the forefoot is strongly dorsiflexed before being thrown down with the heel striking the ground first. The patient is unable to stand with eyes closed. In contrast, the cerebellar ataxic is not aided by visual assitance. The gait is stumbling, drunken, swaying from side to side, and there is a tendency to fall toward the side of the lesion.

Muscle coordination in the lower extremity may be tested by having the patient, while his eyes are closed, place the heel of one foot upon various points on the opposite leg. The upper extremities are tested by asking the patient to touch the tip of his nose with the end of the index finger.

REFLEXES

The simplest spinal reflex consists of a primary sensory and motor neuron and a synapse in the anterior gray matter of the spinal cord. It is composed of a receptor, or peripheral sensory nerve ending; an afferent conductor; a synaptic center; an efferent conductor; and the effector mechanism or muscle fibers. One or more intermediate neurons are interposed between the primary neurons. The latter may remain localized to one side of the cord (association neurons), pass to the opposite side of the cord (commissural neurons), or extend proximally or distally to complete an intersegmental reflex arc. Each reflex is contained within a definite segment of the cord (Fig. 14-3).

Deep Reflexes. Reflexes requiring stimulation of the tendons (*e.g.*, patellar, Achilles, biceps and triceps) are termed *deep reflexes.*

Superficial Reflexes. Others requiring cutaneous stimulation (*e.g.*, abdominal and cremasteric) comprise the superficial reflexes. Destruction of either limb of the reflex arc or the spinal cord segment abolishes the reflex response. If the lesion extends across the entire spinal cord segment, elimination of inhibitory impulses from cerebral centers causes an exaggeration of deep reflexes below the level of the lesion. Superficial reflex arcs involve the cerebral cortex, thereby explaining their absence in upper motor neuron lesions.

Pathologic Reflexes. Destruction of the upper motor neurons or pyramidal tracts is indicated by pathologic reflexes. The Babinski phenomenon is elicited by stroking the plantar surface of the foot. Normally, all the toes flex plantarward. A pathologic response is a dorsiflexion of the large toe. An exaggerated deep reflex is only of significance if associated with absent superficial reflexes, pathologic reflexes, and sustained clonus. Sphincter disturbances are often present, manifest by difficulty in starting the urinary stream, urinary retention, or incontinence.

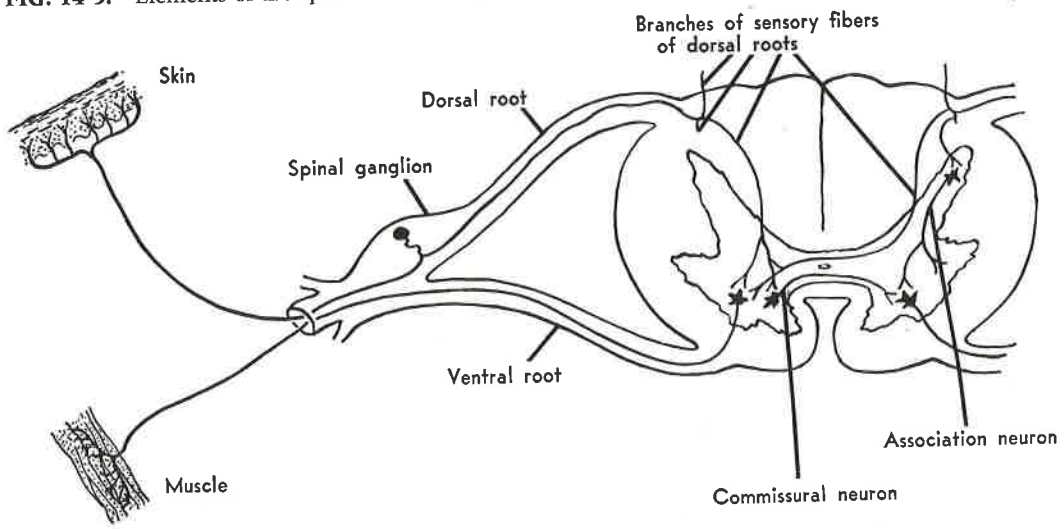
Argyll Robertson Pupil. The pupil of the eye contracts when exposed to light and when accommodating for a near object. When the pupil reflexly contracts to accommodation but not to light, it is the Argyll Robertson pupil, which is characteristic of central nervous system lues.

Reflex Centers. The centers of various reflexes of importance to the orthopaedic surgeon are:

Deep reflexes	
Biceps	C5
Triceps	C6
Radial	C7
Ulnar	T1
Patellar	L2-L3
Achilles	L5-S1
Superficial reflexes	
Upper abdominal	T8-T10
Lower abdominal	T10-T12
Cremaster	L2
Sphincteric reflexes	
Bladder	S3-S4
Anus	S3-S4

Mass Reflexes. In normal humans, the motor response to an afferent impulse is localized and specific. In lower vertebrates the response, usually a flexor spasm, is more widespread and constitutes a protective reflex. When, in

FIG. 14-3. Elements of the spinal reflex arc. (After Ranson)



the human the spinal cord is completely transected, the distal portion temporarily loses and then regains its reflex excitability. The reflex response is now primitive. Specificity of response is lost, and stimulation occasions a widespread motor reaction. For example, stroking the plantar surface of the foot produces flexion at the hip and the knee, dorsiflexion of the foot, and emptying of the bladder. This is the "mass reflex" characteristic of complete interruption in continuity of the spinal cord. The center for this reflex is located low down in the spinal cord and is independent of cerebral control.

SENSATION

Objective evidence of sensory loss defines the site of the lesion and should be compared with normal areas. Changes of sensation form an accurate indication of improvement or progression. Light touch is determined by stroking the skin with a wisp of cotton. The hair on the skin produces sensation other than light touch and should first be removed. Pressure touch is tested by use of a blunt instrument. Pricking the skin with a needle elicits superficial pain. Temperature sensation is determined by applying a test tube filled with hot water and another with cold water. Vibration sense is tested with a tuning fork.

Epicritic or discrimination sensibility is the ability to discriminate between two points. When this sensation is reduced, the points of a compass may have to be widely separated before the stimulus can be recognized as dual. Position is tested by placing a part of an extremity such as the large toe in a certain attitude and asking the blindfolded patient to describe the position. Stereognosis, the sensation of size, shape, and form, the center for which exists in the parietal lobe, is determined by placing familiar objects in the patient's hand. Disease of the posterior columns of the cord produces loss of muscle and joint sensibility in the hands and, therefore, a loss of stereognosis.

Sensations conducted through peripheral nerves consist in deep sensation, the ability to discern pressure, position, and vibration; protopathic sensation, the recognition of painful stimuli and the distinction between extremes of hot and cold; and epicritic sensation, the ability to discriminate between two points and to distinguish between finer grades of temperature.

Severance of a cutaneous nerve produces a loss of all forms of superficial sensation, touch, pinprick, two-point discrimination, and distinction between hot and cold. The sense of deep position, pressure, and vibration is preserved. Epicritic sensory loss is well defined. Protopathic sensory loss after division of a peripheral nerve is smaller because of enormous overlapping in innervation from several nerves. Therefore, in determining peripheral sensory loss, testing superficial touch with a wisp of cotton is more accurate than testing pain perception with a pin. Destruction of nerves closer to the spinal cord increases the extent of loss to painful stimuli.

Afferent sensory fibers pass through the dorsal root to enter the spinal cord. Each fiber passes to the posterior gray column, where it divides into a long ascending and a short descending branch. Thus synapses are made not only with neurons at the same level but also at other levels as high as the medulla. Through relays, afferent impulses reach the cerebral cortex and the cerebellum.

The impulses of movement, position, and vibration pass upward within the cord on the same side as their point of entry. Impulses of touch, pain, and temperature cross to the opposite side of the cord before ascending.

A transverse division of the spinal cord causes loss of sensation below the level of the lesion. At the upper level of sensory loss there exists a band of hyperesthesia due to sensory root irritation at the level of the lesion.

Sensory supply to the body is made up of a regularly spaced series of dermatomes, which correspond to spinal cord segments. If one imagines the body in the quadruped position and then intersects the body at regular intervals, beginning at the neck and ending at the coccyx, the segmental nerve distribution will be apparent. In this position the thumbs and the large toes are in a more advanced position than the small finger and toe. Therefore, the radial side of the upper extremity is represented by a higher segmental level of the spinal cord than the ulnar side, the medial side of the thigh and the leg are of a higher segmental level than the external side of the lower extremity. By finding the sensory loss in a specific dermatome, the level of the spinal cord lesion is localized.

The viscera are generally lacking in sensory fibers. Disease in a viscus supplied by a certain spinal cord segment will produce referred pain in the cutaneous distribution of that segment. Thus, gallbladder disease causes interscapular pain; subdiaphragmatic disease causes pain in the shoulder.

The type of pain should be noted. Tabes dorsalis causes lightning pains; neuritis causes a burning pain; arthritis, an aching pain and sense of stiffness; and spinal disease, a girdle pain.

THE AUTONOMIC NERVOUS SYSTEM

From various centers in the central nervous system, fibers pass through the spinal nerves by way of rami communicantes to ganglia; the latter in turn innervate viscera, glands, heart, blood vessels, and smooth muscles. The sympathetic trunk, on each side of the body, extends along the lateral aspect of the spinal column from C2 to the coccyx. It is composed of 21 to 22 ganglia: 3 cervical (superior, middle, inferior), 10 or 11 thoracic, 4 lumbar, and 4 sacral. Gray and white rami communicantes connect the sympathetic trunk with the spinal nerves. The gray rami are composed of unmyelinated fibers, originating in the sympathetic ganglia and distributed

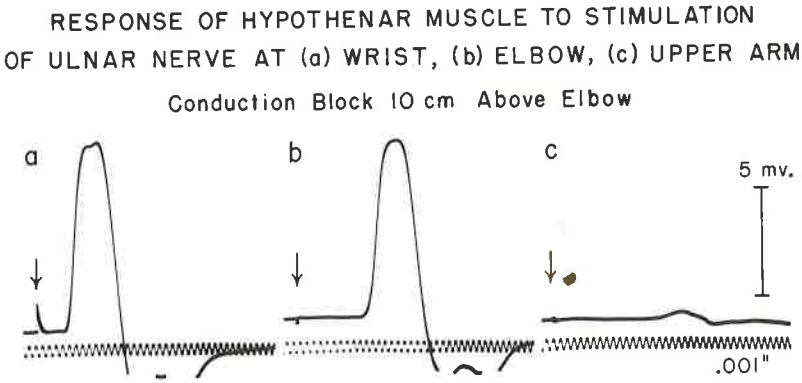


FIG. 14-7. Location of the site of block of nerve conduction. The action potential of the hypothernar muscles was recorded as an indication of the response to maximal stimulation of the ulnar nerve. A normal response occurred when the nerve was stimulated at the wrist (*a*) or at the elbow (*b*). A greatly diminished response was obtained when the nerve was stimulated 11 cm or more above the elbow (*c*). Surgical exploration revealed compression of the nerve at a point 10 cm above the elbow as the cause of paralysis of voluntary contraction. The stimulus artifact (*arrow*) designated the exact time of initiating the stimulus. Time scale is 1000 cycles/sec. (Clinical Examinations in Neurology, 4th ed. Philadelphia, WB Saunders, 1976)

may disclose changes that indicate either progress in reinnervation or advancing denervation.

The procedure also aids in localization of the point of nerve injury or disease. A normal muscle response indicates that the nerve lesion is proximal to the point of stimulation, either in the proximal portion of the nerve trunk, plexus, or root, or at a higher level in the central nervous system. If the point of nerve stimulation is placed at successively proximal points along the nerve, and a reduction of conduction time suddenly appears at a certain point of application, the lesion is situated just beyond this level (Fig. 14-7).

The portion of a nerve trunk beyond the level of a severe nerve injury, regardless of whether complete interruption of the nerve is physiologic or anatomical, remains normally excitable to nerve stimulation for 2 to 3 days after the injury; then it becomes progressively less excitable until it is completely inexcitable just prior to the appearance of fibrillation potentials in the muscle. Therefore, the electrical response within the first 10 days is no indication of the degree of nerve injury.

Motor Nerve Fibers. Conduction velocity of motor nerve fibers is reduced in peripheral neuropathies caused by either trauma or disease and is reflected on the electromyogram as an increase in the conduction time from the point of stimulation to the muscle, as an increase in the duration of the action potential of the muscle (because reduction of conduction velocity does not uniformly affect all nerve axons within the trunk), or as both of the above.

In conditions that affect the anterior horn cells, such as amyotrophic lateral sclerosis and progressive muscular atrophy, conduction velocities are usually within the normal range or only slightly below the average normal conduction velocity for the nerve tested. Marked slowing of conduction velocity to within 5% to 60% of the normal average is found only in conditions affecting the peripheral nerve. These include chronic nerve compression (*e.g.*, carpal tunnel syndrome), severe nerve injury or neuritis which is undergoing recovery, and chronic neuropathies, particularly those primarily demyelinating disorders such as Guillain-Barré syndrome and Charcot-Marie-Tooth atrophy of the neuropathic type. The low conduction velocity during reinnervation of a muscle after a severe nerve injury or neuritis is related to the small diameter of regenerating nerve fibers.

The conduction velocities at birth are about one-half of adult values, increase to adult values by 3 to 5 years of age, and then slow progressively after 20 to 30 years of age, becoming about 5 to 10 meters/sec slower by 80 years. Conduction velocities are decreased by low temperatures and insufficient circulation in the extremity.

For example, to determine the conduction velocity of the ulnar nerve in the forearm, a stimulating electrode is placed over the nerve at the wrist and the other electrode is placed over the hypothenar muscles. The distance between electrodes is determined in millimeters. The time between the stimulus artifact and the action potential on the graph is determined in milliseconds (Fig. 14-8). Next, the stimulating electrode is placed over the nerve at the elbow and the procedure is repeated. These values of distance and time for the elbow to the hand are recorded, and the values of the wrist to the hand are subtracted, yielding values from elbow to wrist. Millimeters and milliseconds are transposed to meters and seconds, yielding the conduction velocity as meters per second for the ulnar nerve from the elbow to the wrist (Table 14-1).

Afferent Nerve Fibers. Conduction in afferent nerve fibers[3] is a more sensitive indication of involvement of large myelinated fibers than are tests of conduction in motor fibers. This is determined by recording the action potential evoked in a cutaneous nerve by a maximal electrical stimulus. A small triphasic action potential, usually less than 50μv in amplitude, represents the action potential of large myelinated fibers. The nerve action potential is recorded by electrodes at standard positions along the course of the nerve. For example, in compression of the median nerve at the wrist, the stimulating electrodes are placed about the index finger (digital

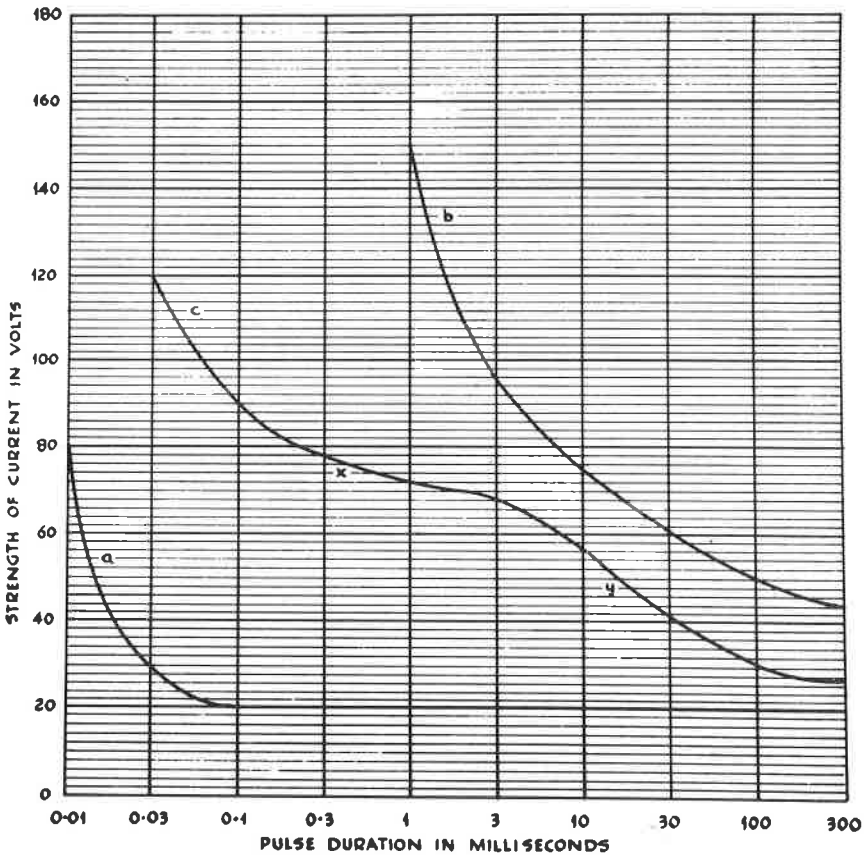


FIG. 14-10. Strength duration curves on normal (*a*), denervated (*b*), and partially innervated muscle (*c*). That part of the curve (*c*) marked *x* represents the innervated component (response of nerve) and that part marked *y* represents the denervated component (response of muscle). (Wynn Parry CB: Electrodiognosis. J Bone Joint Surg 43B:222, 1961)

LOCALIZATION OF SPINAL CORD LESION

BASIC ANATOMY

The pyramidal, or motor, tracts that have already crossed above the spinal cord descend in the lateral columns of white matter. Anterior and lateral to these fibers are the lateral spinothalamic tracts, which carry impulses of pain and temperature upward from the opposite side of the body. The posterior white columns transmit upward both deep (joint, muscle, bone) and superficial tactile sensations uncrossed.

TYPES OF LESIONS

The various types of lesions include the following.

Lower Motor Neuron Lesion. In this lesion limbs are flaccid, deep reflexes are absent, and muscles are atrophied. This lesion may be in the anterior horn, the anterior spinal root, or in a peripheral nerve. If sensory changes are absent, the peripheral nerve is excluded. Anterior poliomyelitis is the most common cause.

One must remember that a sudden traumatic lesion of the spinal cord produces at first a flaccid paralysis below the level of the lesion, but eventually spastic paralysis develops.

Upper Motor Neuron Lesion. This condition is characterized by increased muscle tone and spasticity, hyperreflexia, clonus, absent superficial reflexes, and positive pathologic reflexes. Bilateral spastic paralysis is due most commonly to cerebral palsy.

Combined Upper and Lower Motor Neuron Lesion. Spastic paralysis in the lower extremities and flaccid paralysis in the upper extremities, with a reaction of degeneration and no sensory loss, indicate a combined lesion of the anterior horns and the corticospinal tracts. The disease is amyotrophic lateral sclerosis. When this combination is associated with loss of pain and temperature sense, while tactile sense is preserved, the cause is syringomyelia or an intramedullary tumor. The central lesion destroys the centrally crossing fibers of pain and temperature, while tactile fibers ascend in the posterior columns unharmed.

Lesion of Posterior Spinal Root. This lesion involves

absence of deep reflexes, loss of all sensation, and spontaneous lightning pains. Loss of joint and muscle sense results in ataxia. The disease is due to tabes dorsalis (locomotor ataxia). The posterior columns undergo secondary degeneration.

Combined Lesion of Posterior Columns and Pyramidal Tracts. In addition to loss of joint and muscle sense and an ataxia, a spastic weakness with hyperreflexia is present. Subacute combined degeneration is due to pernicious anemia. Increased mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), macrocytosis, and achlorhydria are confirmatory.

Transverse Lesion of the Spinal Cord. At the corresponding segmental level, the deep reflex is absent; flaccid paralysis, muscle atrophy, and electrical evidence of denervation are present. Below this level, one finds spastic paralysis, hyperreflexia, absent superficial reflexes, pathologic reflexes, complete sensory loss, or sphincteric constriction with urinary retention. The cause of the pathology includes myelitis, tumor, fracture-dislocation of the spine, thrombosis, hemorrhage, abscess, and vertebral disease. When the onset is sudden, vascular or traumatic lesions are probable. Tumors and infections cause slowly progressive symptoms.

Section II
Miscellaneous Affections of the Nervous System

SPINAL CORD TUMORS

To the orthopaedic surgeon the identification of a spinal cord tumor is important as a source of pain about the spine or of pain referred to other regions. Symptoms about the chest and the abdomen may cause confusion with visceral disease. Especially important is the differentiation from other causes of pain in the upper or lower extremity, particularly intervertebral disk protrusion.

PATHOLOGY

Spinal cord tumors are intramedullary, originating from and involving the substance of the cord itself, and extramedullary, occurring in the meninges and the surrounding tissues.[12] The intramedullary tumors are most frequently ependymomas and less often multiform glioblastomas and medulloblastomas. The extramedullary tumors include meningiomas, angiomas, lipomas, glands of Hodgkin's disease, tuberculomas, syphilomas, cysts of the spinal cord, and metastatic tumors. An ependymoma of the filum terminale forms a giant-sized mass, which compresses the roots of the cauda equina. Multiple neurofibroma of von Recklinghausen's disease may form in the cauda equina. The extramedullary tumor is more frequent, and the vast majority are benign and accessible to surgery. Intramedullary tumors occur most often in the cervical and the lumbar enlargements. A glioma of the cervical enlargement may become cystic and produce a syringomyelic cavity.

CLINICAL PICTURE

Symptoms are caused by direct pressure on the cord and the nerve roots, by pressure against the opposite side of the cord that is pushed against the bony wall, and by changes in blood vessels with altered blood and spinal fluid flow.

Pain

Pain is the most frequent symptom and usually is felt in the region supplied by the involved posterior root.[13] Thus a neuralgic pain may extend down a limb or be situated about the chest or the abdomen as a girdle pain. The usual pain of an intraspinal lesion may precede any other symptom by months or years and may be constant or intermittent. It characteristically is most pronounced at rest and is reduced in intensity by exercise. It usually persists in a well-localized area because of definite nerve root involvement. Commonly, it is lancinating and aggravated by coughing, sneezing, lifting, and straining during bowel movement. It invariably awakens the patient 4 to 6 hours after he has retired and often becomes so severe as to compel the patient to walk the floor or to sleep in a sitting position.

Pain may be confined to the back, where it is generally situated about the site of the lesion. However, involvement of the cauda equina anywhere in its course from the upper lumbar canal often causes low back pain. Referred pain from the cervical area involves the upper extremity. Within the thoracic spinal canal, a lesion at a definite vertebral level will involve the dermatome several segments below. Thus, a neurofibroma at the (T5) level often causes pain in the subcostal area of the abdomen, where it may be mistaken for gallbladder disease. Cauda equina tumors cause sciatica as well as low back pain, with a ruptured disk often being diagnosed.

Involvement of a motor nerve may cause painful spasms of the muscle that it innervates.

Motor Signs

Eventually, motor signs appear in the form of muscle weakness at the level supplied by the involved segment of the cord and usually on the same side. Involvement

a malignant metastasis. Metrizamide injection helps to localize the lesion. Characteristic shadows produced by certain tumors have been described but are not reliable.

Myelography is essential for determining the presence of a space-occupying mass and localization for surgical removal. It may aid in detecting a congenital malformation such as a tight filum terminale and diastematomyelia.

Computed tomography combined with myelography will disclose an extraordinary enlargement of the spinal cord or cauda equina, an extramedullary compressive mass, and secondary erosive bone changes. A bone scan may localize a vertebral neoplastic lesion.

Importance of a General Examination

Carcinomas of thyroid, breast, prostate, and adrenal gland are prone to metastasize to the spinal column. These sites should be investigated thoroughly before exploration of the spine is attempted.

TREATMENT

Surgery aims at relief of compression of the spinal cord. The mortality rate is less than 4%. Most tumors are extramedullary and after their removal symptoms subside to a remarkable degree. Intramedullary tumors are surgically inaccessible. Even when the lesion cannot be removed, laminectomy reduces pain and prolongs life by relief of pressure. It often prevents bladder paralysis and ascending urinary infection.

CAUDA EQUINA TUMORS

Tumors of the cauda equina are especially important to orthopaedic study because they produce a subtle clinical picture that often is erroneously treated as being due to musculoskeletal disease. Failure to recognize this possibility at an early stage results in irremediable paralysis. The early symptoms are often mistakenly attributed to a lumbar disk protrusion. The following points aid in recognition and should be carefully studied and correlated with a complete neurologic examination including diagnostic procedures (*e.g.*, electrodiagnosis, myelography).

Early pain is a cardinal symptom. It develops early and continues long before neurologic signs appear. Its radiation varies: back of thighs, front of thighs, perineum, sciatic distribution, or limited to the low back.

Later symptoms are muscle weakness; flaccid paralysis; and impaired sensation of all forms, including pain and temperature sense. In the male there is impaired erection and ejaculation. Deep tendon reflexes are absent. Sphincter loss and saddle anesthesia, when it occurs early and is severe, points to a high lesion about the conus; when it develops late, the lesion is low in the cauda equina. A lesion of the epiconus typically causes early paralysis of the feet.

Symptoms suggesting tumor rather than a disk protrusion include insidious onset, spontaneous, no prior trauma; unremitting, progressive course; sphincter involvement; and constant pain, unrelieved by recumbency and severe at night.

Common presenting symptoms in order of frequency are low back pain, unilateral sciatica and numbness of legs.

Common physical findings in order of frequency are diminution of ankle jerks and sensory changes.

The myelogram reveals a space-occupying mass. There is markedly high spinal fluid total protein in 70% of tumor cases. In 30% of cases, the total protein is not elevated.

Malignant tumors include leiomyosarcoma, lymphoma, myxofibrosarcoma, metastatic carcinoma, and neurogenic fibrosarcoma. Benign lesions include neurilemmoma, ependymoma, neurofibroma, meningioma, fibrous cyst, ependymal cysts, chordoma, lipoma, and fibrosis.

FILUM TERMINALE SYNDROME

Progressive spastic paralysis (filum terminale or cord traction syndrome) can occur in Arnold-Chiari syndrome, scoliosis, and other spinal malformations. The mechanism appears to be abnormally short filum terminale, which pulls the cord distally as the vertebral column grows in length. In Arnold-Chiari syndrome, the hindbrain is pulled into the narrow foramen magnum. In scoliosis, the cord is pulled over the angulation. Typically, symptoms appear during periods of rapid growth, from 13 to 19 years of age. A shortened cauda equina is often a component of spina bifida and is responsible for restricting ascent on the conus medullaris during growth. Treatment is by sectioning the filum, which gradually improves the patient. In Arnold-Chiari syndrome, decompression of the foramen magnum produces the same result.[14,15] This latter condition consists of herniation of the hindbrain and the cerebellum through a narrowed foramen magnum. It occurs during the growth period when the spinal cord is prevented from ascending by a tight filum terminale. The constriction of the hindbrain interferes with the exit of spinal fluid from the ventricles, and hydrocephalus results in infants, with signs of increased intracranial pressure and spastic paralysis in older children. In addition, the discrepancy in growth between the spinal cord and the spinal column results in reversal in the course of the cervical spinal nerve roots, which become angulated over their point of exit at the intervertebral foramina.

When progressively increasing weakness in the lower extremities, deformities of the feet, sphincter weakness, root pains in the upper extremities, and headaches develop in an actively growing child, a tight filum

ralysis is often transient in some areas; atrophy is assymetrical, spotty, and nonprogressive.

Cervical Cord Tumor. Root pain and atrophy may be unilateral and become progressively worse. Sensory impairment affects all forms. Headache is common. Spinal fluid studies may show subarachnoid block and an increase of total protein.

Friedreich's Ataxia. Muscle atrophy affects the leg and foot, causing typical severe equinocavovarus deformity. Onset is in childhood. Ataxia, loss of proprioception, pyramidal tract signs, and nystagmus occur. The disorder is hereditary and familial.

PERIPHERAL NERVES

FUNCTION OF A PERIPHERAL NERVE

Peripheral nerve fibers conduct sensory, motor, and trophic impulses. Sensation includes coarse and light touch, pain, temperature, stereognosis, and deep tissue sense (pain, position, vibration). Motor fibers innervate muscles. Trophic fibers are supplied to all tissues, including skin, tendons, joints, and muscles.

BASIC NERVE UNIT

The motor fibers originate from neurons in the anterior horn of the spinal cord. Sympathetic neurons in the lateral columns of the gray matter give rise to vasomotor and trophic fibers. All fibers combine and emerge as myelinated fibers from the anterolateral aspect of the cord as a common white ramus. A ganglion located outside the dorsolateral aspect of the cord is connected with the latter by a dorsal gray nerve root. It contains the neurons for sensory perception.

PATHOLOGY FOLLOWING SEVERANCE OF A PERIPHERAL NERVE

All functions distal to the point of severance are interrupted. At the end of the proximal nerve segment the axons multiply and attempt to grow distally. However, a connective tissue bulblike growth envelops the end of the nerve and obstructs the path of these fibrils, which become arranged in disorderly fashion. The connective tissue and fibril growth is called a neuroma. The distal nerve segment swells to twice its original size and undergoes wallerian degeneration. This process is complete in about 1 month. Its proximal end displays only a small enlargement, consisting only of fibrous tissue. Occasionally, the connective tissue growths at the end of each segment may unite, and some of the fibrils may successfully penetrate the mass and grow distally. Partial function may thereby be restored.

SYMPTOMS AND FINDINGS FOLLOWING COMPLETE NERVE INJURY

Stereognosis, the most specialized perception of shape and texture is lost. The specialized touch corpuscles (Meissner's, Pacini's, Ruffini's) located in the hand, most particularly in the median nerve distribution, are linked with the stereognostic center on the opposite side of the brain.

Superficial sensation to touch, pain, and temperature is lost. This includes epicritic sensation (light sensation), by which two points of a compass are distinguished, and coarse sensation, such as pain.

Deep sensation to muscle and joint movements, position, deep pressure, and vibration travels mainly in motor nerves and if these nerves are injured, sensation is lost. Bunnell describes an excellent method of mapping anesthetic areas by an electric skin resistance machine consisting of a battery, a galvanometer, and two electrodes placed near one another. Anesthetic skin is electroresistant because the sweat glands are dry. Placed on normal skin, this apparatus is a detector of malingerers, since the galvanometer will show normal conductivity. A person who claims to have pain will show excellent conductivity, because pain stimulates the sweat glands. No reaction is obtained on anesthetic skin.

Loss of motor supply to a muscle results in progressive atrophy and fibrous degeneration of that muscle. A muscle is partially paralyzed when nerve severance is incomplete and is revealed by a limited amplitude of motion and decreased force against resistance.

Deep reflexes are diminished and lost.

Electric stimulation of the nerve no longer causes the muscle to contract. However, the muscle may be well stimulated directly by faradic current. This response gradually diminishes until after 2 weeks no response to faradism is obtainable. Nevertheless, the muscle continues to respond to galvanic current by a slow vermicular contraction, greater in amplitude and followed by slow relaxation. This chain of events, namely, early loss of response to faradism and increased continued response to galvanism, is known as the reaction of degeneration and is characteristic of peripheral nerve interruption. After the muscle has undergone complete fibrous degeneration, no further electric reaction is obtainable. Each muscle responds best electrically at the point at which the nerve enters the muscle. Normally, it contracts strongly to faradic current and gives a quick twitch to galvanic. (See Electrodiagnosis.)

Trophic influence is lost. All tissues in the supplied area undergo atrophy. The skin is thin and glossy, red, or cyanotic. Hair and nails are brittle. Bone is osteoporotic. Joint cartilage is thinned, and ligaments are contracted

and inflexible, resulting in decrease of motion due to contracture of the joint. Healing of wounds is slow. This picture should be differentiated from the condition of reflex sympathetic dystrophy, which is characterized by a generally painful, swollen, cold, cyanotic part and typical mottled osteoporosis.

DETERMINING THE SITE OF NERVE INJURY

The particular nerve involved is revealed by the muscles paralyzed and the area of anesthesia. The point of interruption is located by the history of accident, by the location of the nerve, and by Tinel's sign. The last is performed by percussing or tapping over the severed nerve end, causing tingling in the area of distribution of the nerve. In the course of regeneration the sign can be elicited further distally, indicating the level to which the new axons have grown.

REGENERATION OF NERVES

Severed nerves will bridge a gap of 1 cm or a little more. At operation a nerve stripped of its surrounding tissues loses its blood supply and function temporarily. Scar tissue will strangulate a nerve and obstruct peripheral growth. Repair of a peripheral nerve demands accurate approximation in exact rotation; otherwise, sensory fibers may grow down motor pathways, and vice versa, and so are wasted. Regeneration occurs at a rate of 1 mm or 2 mm/day. Motor recovery can occur after 2 or 3 years, and sensory return occurs from 3 to 5 years after nerve severance. In the arm, the radial nerve regenerates better than the median and the median, better than the ulnar. Sensation recovers before motor function. Protopathic precedes the epicritic sense. Deep sensibility returns with the epicritic sense and, finally, stereognosis occurs. The proximal portions of the anesthetic area disappear first. Tinel's sign can be elicited by tapping anywhere over the newly formed nonmyelinated axons. The sign disappears in 1 or 2 years as the axons become myelinated. The quality of sensation early after return is not normal. Paresthesia is felt in response to stimuli. Reactivation of muscles occurs later, the most proximal ones returning first. The early flicker of motion increases until a large portion of the muscle contracts, although it moves the part through a limited amplitude and is easily fatigued. Strength and coordination in movement are acquired eventually. Trophic changes progress even after nerve severance; these start to regress when sensation begins to appear. Serial electromyographic and strength duration studies will often demonstrate beginning recovery of muscle excitability to electric stimulation long before clinical signs of reinnervation become apparent. These electrodiagnostic tests can be used to follow the rate of nerve regeneration.

THE DEGREE OF NERVE INJURY

Neurapraxia is a physiological injury to a nerve. No anatomical damage is present. The paralysis is transient, sensory loss is slight, no reaction of denegeration is obtainable, and recovery is complete within a few hours to days.

Neurotmesis is complete physiological and anatomical interruption of the nerve fibers and their sheaths. Recovery generally is obtainable by surgical approximation.

Axonotmesis is interruption of nerve fibers within their sheath. The Schwann tubes remain in continuity so that spontaneous cure eventuates. It is necessary to distinguish between neurotmesis and axonotmesis to determine whether to intervene surgically.

Traction nerve injuries are often severe and are essentially a neurotmesis. Although anatomical continuity may appear to be preserved, extensive intraneural scar formation occurs. Direct compression injuries at a fracture site usually have a good prognosis for spontaneous cure. This is especially so in the case of the radial nerve. Involvement of the axillary nerve has an unfavorable prognosis. It is necessary to preserve muscle and joint function by galvanic stimulation, passive motion, and daily massage until the nerve regenerates. Inasmuch as axons grow down 1 mm/day, one can estimate the time required to reach the most proximally involved muscle (*e.g.*, the brachioradialis in case of the radial nerve). If reinnervation does not occur at the expected time, surgical exploration of the nerve should be undertaken. Electromyographic evidence of motor unit action potentials is found in the most proximal muscle some weeks before a flicker of voluntary power is ascertained clinically.

POLYNEURITIS

Polyneuritis (multiple neuritis) is applied to a painful, degenerative, often inflammatory process in a neuron and its fiber.[12] When any portion of a neuron, whether axon or cell body, is involved, the remainder of the cell invariably undergoes changes. Therefore, the painful degenerative process, regardless of its initial situation, causes functional loss of the entire unit.

Neuritis is very common and is caused by many conditions. The principal causes are listed in Table 14-2 under those causing mononeuritis and others causing polyneuritis. Neuritis of a single nerve most often stems from local causes, which are described under their respective sections. The following discussion applies mainly to multiple neuritis.

PATHOLOGY

In most forms of polyneuritis, a noninflammatory degeneration of the peripheral nerves takes place. The

neuritis) in diabetes, which is probably due to sudden arterial occlusion within the nerve and which causes permanent paralysis. The femoral and anterior tibial nerves are most frequently affected by mononeuritis.

The outlook for polyneuritis in the diabetic is good, with improvement taking place over several weeks to a few months. Occasionally, burning pain about the heels may persist as a very distressing symptom and keeps the patient awake at night.

The symptoms of occlusive arterial disease (intermittent claudication, numbness, rest pain) should not be confused with those of diabetic polyneuritis.

Treatment consists of control of severe painful spasms by use of narcotics, bed rest, and warm moist packs. The administration of various components of vitamin B in large amounts seems to shorten the course and lessen the intensity of the pain. The diabetes should be well controlled.

CHEMICAL POLYNEURITIS

Lead Neuritis

Lead poisoning differs from other forms of polyneuritis in causing involvement of the central nervous system, especially the anterior horn cells. Painters, plumbers, plasterers, and typesetters are predisposed. Cosmetics, hair dyes, and many other substances contain lead.

The degenerative neuritis is limited to motor nerves, especially those to the extensors of the hand and the forearm. However, the forearm flexors and the deltoid finally are involved. Usually, the brachioradialis and sometimes the extensor and the abductor of the thumb are spared. When the lower extremity is involved, the peroneals are more likely to be affected than the anterior tibial. The course is chronic, and the outlook is good when the offending agent is removed.

Arsenical Polyneuritis

The source of arsenic includes medications, wines, and insecticides. Symptoms resemble those of alcoholic polyneuritis. Pain in the limbs and numbness of the hands and the feet are followed by wristdrop and footdrop. The muscles atrophy rapidly. Cutaneous anesthesia, pains, and hyperesthesias are more severe than in alcoholic polyneuritis, and mental symptoms are rare.

Associated features of arsenical poisoning include abdominal pain, vomiting and diarrhea, skin pigmentation, keratoses, and herpes zoster.

The diagnosis is established by recovering arsenic from the urine and the hair.

Recovery requires 2 to 3 years.

Mercurial Polyneuritis

Polyneuritis due to mercury poisoning resembles other forms of polyneuritis, but renal damage and gingivitis are additional findings.

Alcoholic Polyneuritis

This type of polyneuritis occurs in patients who are chronically addicted to alcoholic intake for many years. Because their dietary intake is also inadequate, the cause appears to be a combination of the toxic effect of alcohol and a nutritional deficiency. Moreover, most patients suffer from achlorhydria, with loss of intrinsic factor and malabsorption, which may explain the frequently associated anemia and posterior column involvement in the spinal cord. The condition is best described as an alcohol–vitamin deficiency polyneuritis, but the actual pathogenesis is unclear. It is the most common form of polyneuritis and occurs about five times more frequently in men than in women.

Symptoms develop slowly over weeks to months, but they may develop rapidly over a few days. The first symptoms are pain in the legs and paresthesias in the hands and feet. These are soon followed by weakness of the legs, dropfoot, and ataxia. Unless the condition is recognized and the process halted by treatment, the legs gradually become paralyzed and muscle weakness spreads to involve the trunk and upper extremities. In extreme cases, optic neuritis, extraocular paralysis, and weakness of facial muscles and those of other cranial nerves develop. Muscle weakness is greatest in the distal portions of the extremities, and the extensors are more severely affected than the flexors. Anesthesia and hypoesthesia are most pronounced over the distal parts of the extremities, whereas cutaneous sensibility is preserved elsewhere. Proprioception is impaired. The nerve trunks and muscles are tender. The deep tendon reflexes disappear, especially in the legs. Plantar and abdominal skin reflexes are unresponsive. Drying, scaling, and pigmentation of the skin on the back of the hands and wrists, and swelling of the ankles, suggests a pellagralike component.

Korsakoff's syndrome is a combination of polyneuritis and a mental state characterized by confusion, disorientation, loss of memory, and a tendency to confabulate. Most often it is associated with alcoholic polyneuritis, but it may occur with other forms of polyneuritis and, in alcoholics, can occur in the absence of polyneuritis. Signs of liver damage and convulsive seizures are not infrequent. The mortality rate in untreated cases is about 50%, with death occurring within the first few weeks after the symptoms become severe. Recovery is slow and incomplete.

The course of alcoholic polyneuritis is prolonged, especially if paralysis has already developed before treatment is instituted. In uncomplicated cases, the mortality is low but is increased proportionate to the degree of cerebral involvement.

The pathology, as in the majority of other forms of polyneuritis, is mainly a noninflammatory degeneration of the peripheral nerves. In the initial stages there are swelling and fragmentation of the myelin. As the destructive process advances, the axis cylinders also become involved, and the axons undergo disintegration. Retro-

grade changes are found in cells of the anterior horns (axonal reaction). In chronic cases, the posterior funiculi show degeneration of the ascending fibers.

Treatment. Thiamine and vitamin B complex given intravenously in large doses greatly reduce the mortality. The medical regimen aims at gradual withdrawal of alcohol and increasing the diet, which is supplemented by vitamins. Correction of the anemia and malabsorptive defect is essential.

Orthopaedic treatment consists of splinting of the extremities during the acutely painful stage to prevent stretching of the paralyzed muscles. Physical therapy includes warm moist packs and passive range of motion exercises. Later, when voluntary movements begin to return, increasing active exercises, electrical stimulation, and muscle training help to restore strength and coordination. Orthoses and walking aids may be necessary for months. Recovery may take as long as 2 years.

INDIVIDUAL PERIPHERAL NERVES

MERALGIA PARESTHETICA

The lateral femoral cutaneous nerve arises from the posterior divisions of the second and third lumbar nerves. It makes its appearance at the lateral border of the psoas and passes obliquely across the iliacus to the anterior-superior iliac spine, where it proceeds beneath the inguinal ligament to enter the anterolateral aspect of the thigh.

Any syndrome manifesting numbness, paresthesias, and pain over the lateral and anterolateral aspect of the thigh suggests an inflammatory, degenerative lesion of the lateral femoral cutaneous nerve and is designated meralgia paresthetica (lateral femoral cutaneous neuropathy). The sensations experienced are described variously as burning, tingling, hyperesthesia, numbness, or severe pain. Often pain occurs after activity or direct pressure against the thigh and is relieved by rest. Reduction of tactile sensation is demonstrable over the lateral aspect of the thigh.

In most instances, the condition is idiopathic. Some cases are due to kinking and constriction of the nerve at its point of emergence from the pelvis. Normally, the nerve courses beneath the inguinal ligament and in front of the sartorius muscle. Instead, the lateral femoral cutaneous nerve may penetrate between two fasciculi of the inguinal ligament. Consequently, when the hip is fully extended, the nerve is compressed by the posterior fibers of the ligament (Fig. 14-12).

Treatment. Symptoms may persist for many months but almost invariably subside. If pain is persistent and intolerable and is relieved by injection of a local anesthetic about the nerve at its point of emergence from the pelvis, surgical relief may be accomplished by division of the portion of the inguinal ligament lying posterior to the nerve or by removal of the nerve.

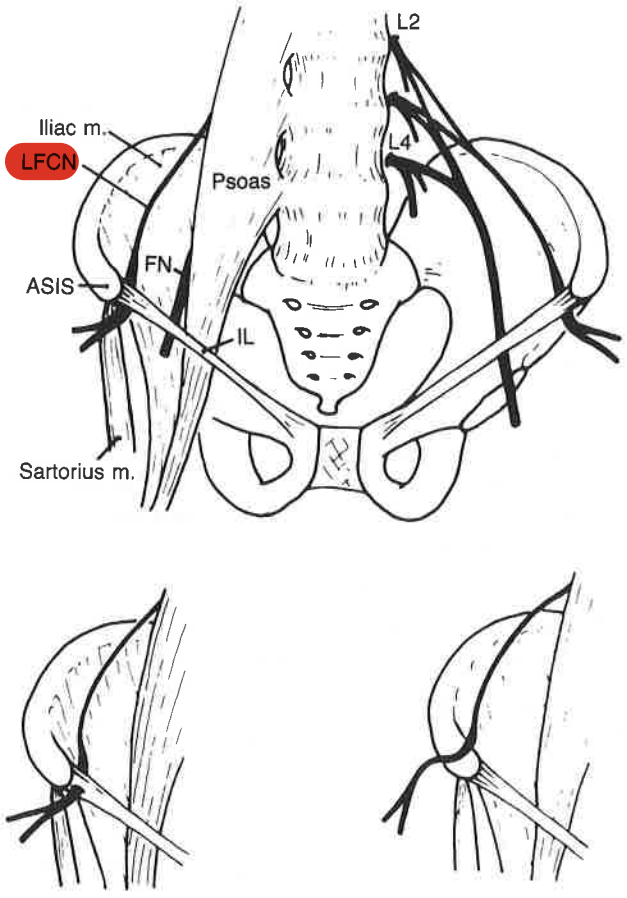
Otherwise, the lesion presumably must be located proximal to this point. It is essential to seek pathology lying along the route of the nerve, whether intraspinal, retroperitoneal, abdominal, or pelvic.

AXILLARY NERVE

Anatomy

The axillary nerve springs from the posterior cord of the brachial plexus and runs alongside the radial nerve behind the axillary artery, separating the latter from the subscapularis muscle on the floor. At the lower border of the muscle, it leaves the radial nerve and turns posteriorly, in company with the posterior humeral circumflex artery, through the quadrangular space to the

FIG. 14-12. Lateral femoral cutaneous nerve (LFCN) of the thigh. (*Top*) Normal course of LFCN passing under the inguinal ligament medial to the anterior superior iliac spine (ASIS). (*Bottom left*) LFCN passing through a split in the inguinal ligament. (*Bottom right*) LFCN astride the iliac crest lateral to the ASIS. (*FN*, femoral nerve; *IL*, inguinal ligament) (Edelson JG, Nathan H: Meralgia paresthetica. Clin Orthop 122:255, 1977)



shortening is preferable because the bone is deeply situated beneath the musculature where deformity due to the bone graft and overlap of fragments is not visible, delayed union or nonunion is rare, and thigh muscles quickly recover their strength. The femur may be osteotomized obliquely, overlapped, and held by screws. A bone graft ensures union. If loss of length is obviously confined to the tibia, shortening of the tibia may be preferable. A step-cut osteotomy of the tibia is performed. The removed segment of bone is used as a bone graft about the osteotomy site. A comparable segment of fibula is also removed.

Section IV
Cerebral Palsy

Cerebral palsy is a state of muscular dysfunction that results from injury or disease of the upper motor neurons at the level of the cerebral cortex or throughout the course of their fibers within the brain. The loss of inhibitory control results in excessive impulses emanating from lower motor neurons. Lesions of the cerebellum are included, causing symptoms of ataxia and incoordination.

ETIOLOGY

The chief factors include the following:

Brain Injury at Birth. Mechanical causes are poorly applied forceps, excessive uterine contractions, excessive traction on the neck (ruptured vein causing intracranial hemorrhage), and sudden pressure change caused by precipitous extrusion of the fetus. Prematurity is a contributing factor because the delicate vessels beneath the anterior fontanelle are easily ruptured and the leg areas of the motor cortex are damaged by hemorrhage, with consequent spastic hemiplegia. Prolonged anoxia may be due to excessive use of analgesics and anesthetics, the umbilical cord wrapped around the neck of the fetus, or tracheal obstruction. The nerve cells are highly susceptible to anoxia.

Congenital Brain Defects. Disease in mothers during the first 3 months of pregnancy is associated with a high incidence of congenital anomalies. Diffuse and symmetrical brain involvement produces a clinical syndrome characterized by ataxia and athetosis.

Inborn errors of metabolism, mainly related to metabolism of certain amino acids and glucose, produce widespread brain damage, resulting in mental retardation as well as symptoms of cerebral palsy.

Rh factor. The severe jaundice of erythroblastosis fetalis damages the basal ganglia, resulting in athetosis.

Postnatal causes. These include encephalitis, convulsions, and head trauma.

CLINICAL PICTURE

A history is often obtained of a difficult birth or of illness during the early months of pregnancy. The infant is late in sitting up, standing, walking, and talking. The face is expressionless and may exhibit grimacing and drooling. Speech is difficult. Motion is clumsy, slow, jerky, and uncoordinated. The shoulder is adducted and rotated internally, the elbow flexed, the forearm pronated, the wrist flexed and deviated ulnarward, the fingers flexed, and the thumb adducted into the palm. The hip may be flexed, adducted, and rotated internally; the knees flexed; the ankle plantar flexed; the foot in equinovarus; and the toes flexed. The child must be supported under the armpits when standing. The lower extremities are held tightly pressed together or crossed in scissorslike fashion, and the heels cannot be brought down to the floor when standing. The gait is uncoordinated. Mass movements occur (*i.e.*, attempts to move one portion of an extremity throws all the other muscles into a state of spasm). Passive attempts to move a joint are resisted by a spastic group of muscles that may respond reflexly by a strong sustained contraction. Clonus is often seen. The deep reflexes are hyperactive, and pathologic reflex response is obtained. Athetosis may gradually appear within the first 2 years. Mental retardation becomes evident during the second and the third years. The clinical picture varies with the location and extent of the lesion.

EVALUATION OF MENTAL STATUS

About 70% of cerebral palsy patients have a mentality within normal limits. The speech difficulty should not be interpreted as a sign of mental deficiency. Rather, the response to tests made on several occasions should determine the degree of intelligence. Often a child becomes aware of his physical handicap and inability to play with other children, and so development of mental faculties is retarded.

CLINICOPATHOLOGIC TYPES

The location of the lesion determines the predominating clinical symptoms and findings. Although the following specific types are identified, a mixture of types is usual, but the most outstanding symptoms generally classify the offending lesion.

Cerebral Cortex Lesion

Premotor Area. Spasticity results and is evidenced by increased muscle tone, exaggerated deep tendon reflexes, clonus, pathologic reflexes, and the stretch reflex. The

stretch reflex is elicited by stretching that muscle by passively bending or extending the joint over which the muscle acts. The spastic muscle will react by an abnormally strong contraction.

Motor Area. Flaccidity results and is evidenced by decreased muscle tone, diminished deep tendon reflexes, and abnormal elongation of the muscle, which can be stretched without evoking a stretch reflex. A flaccid muscle must be differentiated from a normal muscle, which is weak as a result of stretching and lack of use. A cerebral flaccid muscle may be made to contract by "confusion" on resisted contraction of another muscle. For example, cerebral flaccid dorsiflexors of the foot will contract when active flexion of the hip is resisted.

Hemorrhage varies in extent and location. It may affect both motor and premotor areas so that a mixture of spastic and flaccid muscles result. If the lesion involves the vertex, the legs are affected; if lower on the cortex, the arms are affected; if on the dominant side of the brain, the speech area is affected.

Basal Ganglia Lesion. Athetosis results, as evidenced by irregular, arrhythmic, involuntary movements, chiefly of the hands, that are accentuated by voluntary effort and emotion and subside during sleep. The face is expressionless, but involvement of facial muscles causes constant grimacing and twitchings. In his effort to control involuntary movements, the patient increases muscle tension, which should not be confused with spasticity. The stretch reflex is absent. Athetosis when controlled in one region by braces or surgical fixation will reappear in another area of the extremity.

Cerebellar Lesion. Characteristic signs of cerebellar dysfunction include ataxia, loss of sense of balance, muscle incoordination, adiadochokinesia, nystagmus, and dizziness. The usual cause is a congenital defect or, less commonly, a hemorrhage at birth. Often the ataxia improves spontaneously as the patient learns voluntary control of balance.

Diffuse Brain Damage (Prolonged Anoxia, Multiple Petechial Hemorrhages, Encephalitis). Generalized rigidity of muscles results and is manifest by loss of muscle elasticity and a "lead pipe" resistance to passive flexion and extension of a joint. The degree of rigidity varies from time to time. No true stretch reflex is present, nor are the deep reflexes hyperactive. Usually the mentality is deficient. Neurectomy is valuable for true spasticity but is of no value for rigidity.

TREATMENT

REHABILITATION PROGRAM

About one third of patients are feeble-minded, and another third are crippled severely and irremediably. These latter patients require institutional care. The remainder can be rehabilitated by reeducation, which includes training in balance and posture, locomotion, relaxation, rhythmic exercises, and speech.

At first only fundamental active motion is taught. This means acquiring the earliest primitive motion of the infant, who at first reaches out with one hand to grasp an object and later uses both hands. First, one leg is kicked, later, both legs. These voluntary rudimentary exercises are performed before more complicated motions. The aim should be toward developing the weak antagonists of spastic muscles. At the same time the spastic muscles are stretched repeatedly but gently to avoid exciting the stretch reflex. Exercises are performed rhythmically and with increased speed to develop coordination. This can be effected by having the child relax on the floor and perform movements to the accompaniment of music. Constant repetition enables the patient eventually to develop these actions without interference by the stretch reflex.

TEMPLE FAY METHOD

The Temple Fay method is aimed at the development and the organization of automatic spinal reflexes, as observed by Sherrington and Babinski in the decerebrate animal, and the ultimate coordination of these reflexes with what remains of higher cortical control.[177] At first, the patient is considered an amphibian (*i.e.*, one who because of partial or complete loss of cortical control must depend on the midbrain). It is usually simple to teach the fundamental motion excited at this level.The child is laced prone with the chin forward on a polished floor. First, the arm and the leg of one side is made to flex, the extended thumb pointing toward the face, which is turned toward the hand. As the limbs are extended outward and downward, the limbs of the opposite side flex, the head rotating toward that side. A regular rhythm of alternating movement is kept up to the accompaniment of music until a definite pattern is developed. The original jerky movements become replaced by a smooth series of regular muscular contractions. Once this homolateral or amphibian pattern is well developed, the child progresses to the reptilian or next stage of evolution, characterized by the crossed or contralateral pattern of movements. As the right arm flexes and the head rotates toward that side, the opposite leg flexes. Eventually, these crossed movements can be performed without passive assistance. After these motions are established, further advanced patterns are developed through stages of creeping and crawling to independent walking, feeding, and writing.

Contractures develop early in the presence of spasticity. Foot equinus is common and can be prevented by repeated stretching of the Achilles tendon and wearing a night brace to maintain dorsiflexion. The brace must be worn throughout the growth period because the taut calf muscles do not keep pace with the growth in length

by sympathetic interruption is often obtained. The cessation of sweating and increase in cutaneous electric resistance in a part is proof that the sympathetic trunk has been anesthetized. These tests are useful when no vasodilatation is demonstrable. Autonomic blocking agents, such as tetraethylammonium chloride, are of no value because they effect a varying degree of vasodilation and in effective doses often cause a fall in blood pressure.

TECHNIQUE OF SYMPATHETIC BLOCKS

Injection of a local anesthetic along the side of the spinal column blocks the nerve roots at their point of emergence from their foramina and the sympathetic chain (Figs. 20-22 through 20-25). This is known as a paravertebral block. By blocking one nerve and its corresponding sympathetic ganglion, one visceral and somatic segment of the body is anesthetized (segmental block). The technique of paravertebral block is such that it is almost impossible to block the nerve root without similarly affecting its rami to the sympathetic trunk. On the other hand, the sympathetic ganglion may be blocked without affecting the nerve root. The following description pertains to thoracic and lumbar paravertebral blocks, the technique being varied as indicated to obtain sympathetic blocks. For the upper extremity, the lowest cervical and the upper three thoracic ganglia are infiltrated. All four lumbar ganglia must be blocked to deprive the lower extremity of sympathetic innervation.[66]

Landmarks. In the thoracic spine, the emerging nerve lies at the level of the tip of the spinous process of the vertebra directly above it. For example, the foramen of the first thoracic nerve will be found directly opposite the tip of the spinous process of the seventh cervical vertebra.

In the lumbar spine, the foramen is directly opposite the center of its corresponding spinous process.

The foramen in the thoracic spine lies about 1¼ inches deep to the posterior surface of the transverse process. In other words, when the needle point contacts the posterior surface of the transverse process and then is directed deeply below the inferior margin of the trans-

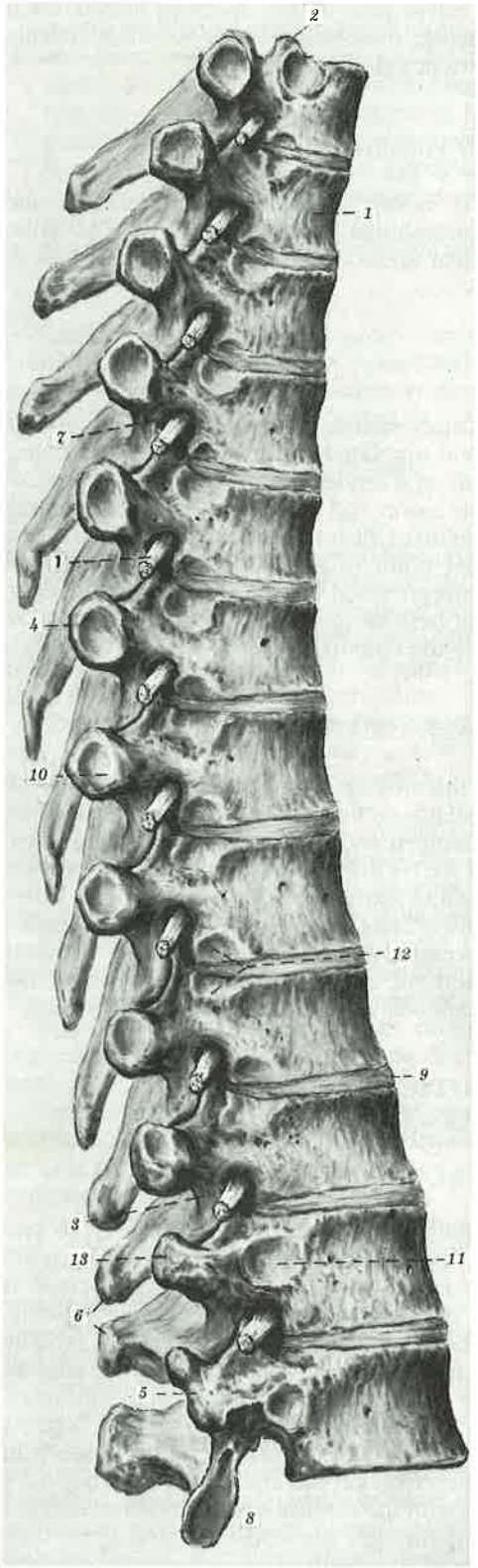


FIG. 20-22. Thoracic vertebrae as seen from the right side.
Nerve: (*1*) spinal nerve emerging from the intervertebral foramen
Tissues: (*1*) body, (*2*) superior articular surface, (*3*) lamina, (*4*) transverse process, (*5*) mamillary process, (*6*) spinous process, (*7*) intervertebral foramen, (*8*) inferior articular surface, (*9*) intervertebral cartilage, (*10*) facet on transverse process for articular part of tubercle of rib, (*11*) facet for head of rib, (*12*) demifacet for articular head of rib, (*13*) no facet on transverse process. (Southworth JL, Hingson RA, Pitkin WM (eds): Pitkin's Conduction Anesthesia. Philadelphia, JB Lippincott, 1946)

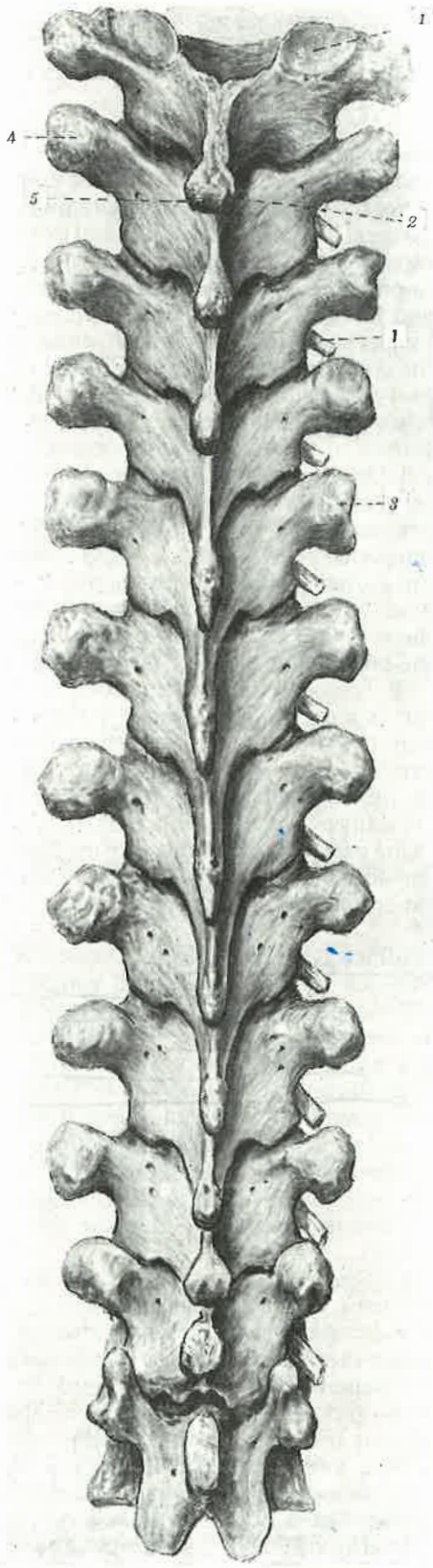


FIG. 20-23. Posterior view of the thoracic vertebrae; spinal nerves emerge from the foramina on the right side.

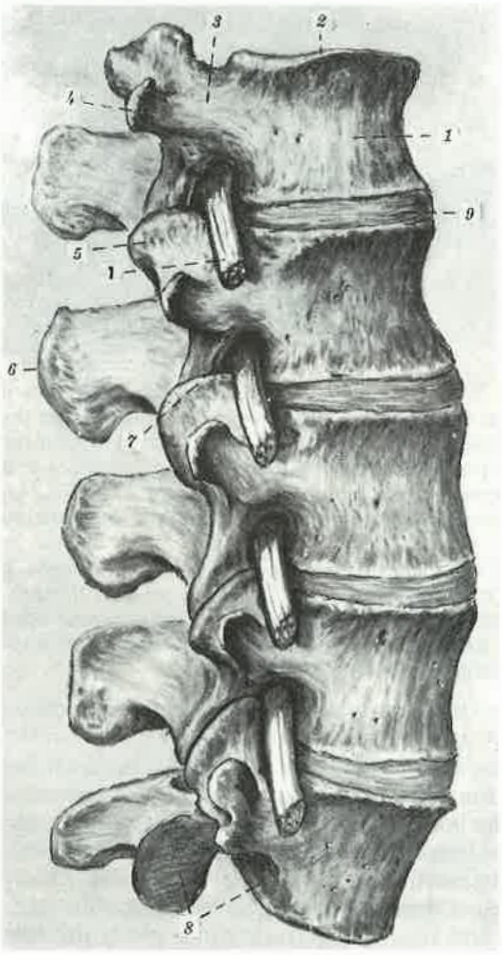
Nerve: (*1*) spinal

Tissues: (*1*) superior articular surface, (*2*) lamina, (*3, 4*) transverse processes, (*5*) spinous process. (Southworth JL, Hingson RA, Pitkin WM (eds): Pitkin's Conduction Anesthesia. Philadelphia, JB Lippincott, 1946)

FIG. 20-24. Lumbar vertebrae as seen from the right side.

Nerve: (*1*) first lumbar nerve emerging from the intervertebral foramen

Tissues: (*1*) body, (*2*) superior articular surface, (*3*) lamina; (*4*) transverse process, (*5*) mamillary process, (*6*) spinous process, (*7*) intervertebral foramen, (*8*) inferior articular surface, (*9*) intervertebral cartilage. (Southworth JL, Hingson RA, Pitkin WM (eds): Pitkin's Conduction Anesthesia. Philadelphia, JB Lippincott, 1946)



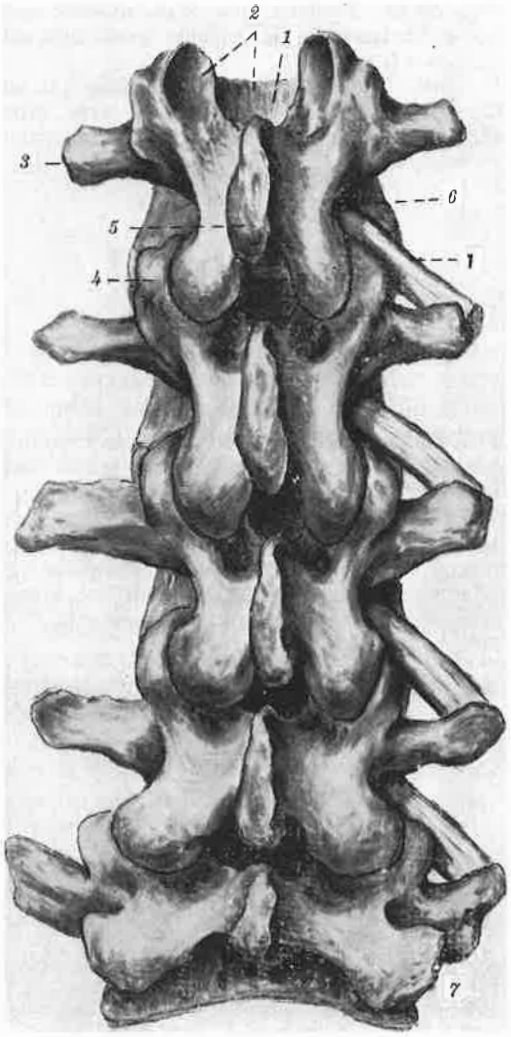


FIG. 20-25 Lumbar vertebrae, posterior view. Note that in blocking near the midline (1½ inches) as described in the text the needle glides off the inferior border of the transverse process to touch the posterior surface of the body of the vertebra at or near the intervertebral foramen. (see Figure 20-27).

Nerve: (*1*) first lumbar nerve emerging from the intervertebral foramen

Tissues: (*1*) body, (*2*) superior articular surface, (*3*) transverse process, (*4*) mamillary process, (*5*) spinous process, (*6*) intervertebral foramen, (*7*) inferior articular surface. (Southworth JL, Hingson RA, Pitkin WM (eds): Pitkin's Conduction Anesthesia. Philadelphia, JB Lippincott, 1946)

verse process, it is inserted to a depth of 1¼ inches before it contacts the nerve, the foramen, or the posterolateral aspect of the body of the vertebra. In the lumbar spine, the distance from the transverse process to the foramen is about one fourth of an inch less.

In the upper thorax, the ganglia lie beneath the necks of the ribs and near the thoracic roots, along the sides of the vertebral bodies. In the lumbar area, the ganglia lie a little deeper along the lateral surfaces of the vertebral bodies. The right sympathetic chain is covered anteriorly by the vena cava, and to the left lies the aorta.

Needle Insertion. The patient is placed in the lateral recumbent position with knees flexed on the abdomen and head bent forward, with the chin on the chest to make the spinous processes more prominent posteriorly. A wheal is made 1½ inches lateral to the selected spinous process. (In the thoracic spine, the landmarks for the upper three sympathetic ganglia are the seventh cervical and first and second thoracic spinous processes; in the lumbar spine, the first to fourth spinous processes identify the correspondingly numbered lumbar ganglia.) A 3½- or 4-inch blunt-pointed needle is inserted through the wheal inward and medially until it contacts the posterior surface of the transverse process (Figs. 20-26 and 20-27). Then it is withdrawn slightly and directed downward at an angle of 15° to 20°, tilted laterally 15° to 20°, and advanced again to pass below the inferior margin of the transverse process. It is advanced farther until it contacts the posterior surface of the body of the vertebra at the outer border of the intervertebral foramen. Contact with the nerve root may evoke paresthetic sensations. This is the position for injecting the nerve root.

If the needle angulation is reduced, that is, the needle hub is moved slightly nearer to the midline, the needle may be made to advance farther along the side of the vertebral body to the sympathetic chain. If the injection of the sympathetics is proper, it will be evidenced clinically by vasodilatation, redness, warmth, and dryness of the part. When infiltration of the first thoracic ganglion spreads to involve the inferior cervical ganglion, Horner's syndrome will be produced.

Stellate Ganglion Block. Occasionally, it is desirable to block the inferior cervical ganglion. This ganglion, when combined with the first thoracic ganglion, is known as the stellate ganglion. The inferior cervical ganglion lies immediately in front of the transverse process of the seventh cervical vertebra and in front of the first rib and just behind the vertebral artery. It may be approached in the same manner as described for paravertebral block of the first thoracic nerve and first thoracic ganglion. Occasionally, it may be easier, particularly in the obese patient, to approach it from the side (Figs. 20-28 and 20-29).

Technique. A point is selected on the lateral aspect of the neck even with the transverse process of the sixth cervical vertebra, if it is palpable, or a fingerbreadth above the seventh cervical spinous process. Anterior to the trapezius the needle is inserted and directed downward and medially at an angle of about 90° with the midline to touch the transverse process of the seventh cervical vertebra. Then the needle is withdrawn slightly and advanced more anteriorly until it contacts the body of the vertebra, where the injection is made. Proper response is indicated by Horner's syndrome and vasodilatation and dryness of the upper extremity.