

4

Spinal Cord and Ascending, Descending, and Intersegmental Tracts

CHAPTER OBJECTIVES

- To learn how injuries to the spinal cord can occur
- To understand the position of the main nervous pathways and nerve cell groups in the spinal cord as well as be able to correlate radiologic evidence of bone injury with segmental levels of the spinal cord and neurologic deficits
- To review the basic structure of the delicate spinal cord and the positions and functions of the various ascending and descending tracts that lie within it
- To make simple line drawings of each of the ascending and descending tracts, showing their cells of origin, their course through the central nervous system, and their destination

A 35-year-old man is galloping his horse when he attempts to jump over a farm gate. The horse refuses to jump, and he is thrown to the ground. His head strikes a log, and his head and neck are excessively flexed. On initial evaluation in the emergency department after he regains consciousness, he is found to have signs and symptoms of severe neurologic deficits in the upper and lower extremities. A lateral radiograph of the cervical region of the spine shows fragmentation of the body of the fourth cervical vertebra with backward displacement of a large bony fragment on the left side.

After stabilization of the vertebral column by using skeletal traction to prevent further neurologic damage, a complete examination reveals that the patient has signs

and symptoms indicating incomplete hemisection of the spinal cord on the left side.

Any medical personnel involved in the evaluation and treatment of a patient with spinal cord injuries must know the structure of the spinal cord and the arrangement and functions of the various nerve tracts passing up and down this vital conduit in the central nervous system.

Because of the devastating nature of spinal cord injuries and the prolonged disability that results, all concerned with the care of such patients must be trained to prevent any additional cord injury and provide the best chance for recovery. All medical personnel must have a clear picture of the extent of the cord lesion and the possible expectations for the return of function.

Spinal cord injuries are common and can occur as a result of automobile and motorcycle accidents, falls, sports injuries, and gunshot wounds. Spinal cord and spinal nerve damage may also be associated with vertebral fractures; vertebral infections; vertebral tumors, both primary and secondary; and herniated intervertebral discs. The student must learn the course and connections of the various tracts within the spinal cord in order to be able to diagnose and understand the treatment of cord injuries. Particular attention should be paid as to whether a specific tract crosses the midline to the opposite side of the central nervous system (CNS) or remains on the same side. If the tract does cross the midline, the level of the crossover is important.

The assessment of neurologic damage requires not only an understanding of the main nervous pathways within the spinal cord but an ability to correlate

radiologic evidence of bone injury with segmental levels of the spinal cord. The close relationship of the spinal cord to the bony vertebral column necessitates a brief review of the vertebral column before the spinal cord is considered.

BRIEF REVIEW OF THE VERTEBRAL COLUMN

The vertebral column is the central bony pillar of the body. It supports the skull, pectoral girdle, upper limbs, and thoracic cage and, by way of the pelvic girdle, transmits body weight to the lower limbs. Within its cavity lie the spinal cord, spinal nerve roots, and the covering meninges, to which the vertebral column gives great protection.

Vertebral Column Composition

The vertebral column (Figs. 4-1 and 4-2) is composed of 33 vertebrae—7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused to form the sacrum), and 4 coccygeal (the lower 3 are commonly fused). Because it is segmented and made up of vertebrae, joints, and pads of fibrocartilage called **intervertebral discs**, it is a flexible structure. The intervertebral discs form about a quarter of the length of the column.

Vertebra General Characteristics

Although vertebrae show regional differences, they all possess a common pattern (Fig. 4-2B). A **typical vertebra**

consists of a rounded **body** anteriorly and a **vertebral arch** posteriorly. These enclose a space called the **vertebral foramen**, through which run the spinal cord and its coverings. The vertebral arch consists of a pair of cylindrical **pedicles**, which form the sides of the arch, and a pair of flattened **laminae**, which complete the arch posteriorly.

The vertebral arch gives rise to seven processes: one spinous, two transverse, and four articular.

The **spinous process**, or **spine**, is directed posteriorly from the junction of the two laminae. The transverse processes are directed laterally from the junction of the laminae and the pedicles. Both the spinous and transverse processes serve as levers and receive attachments of muscles and ligaments.

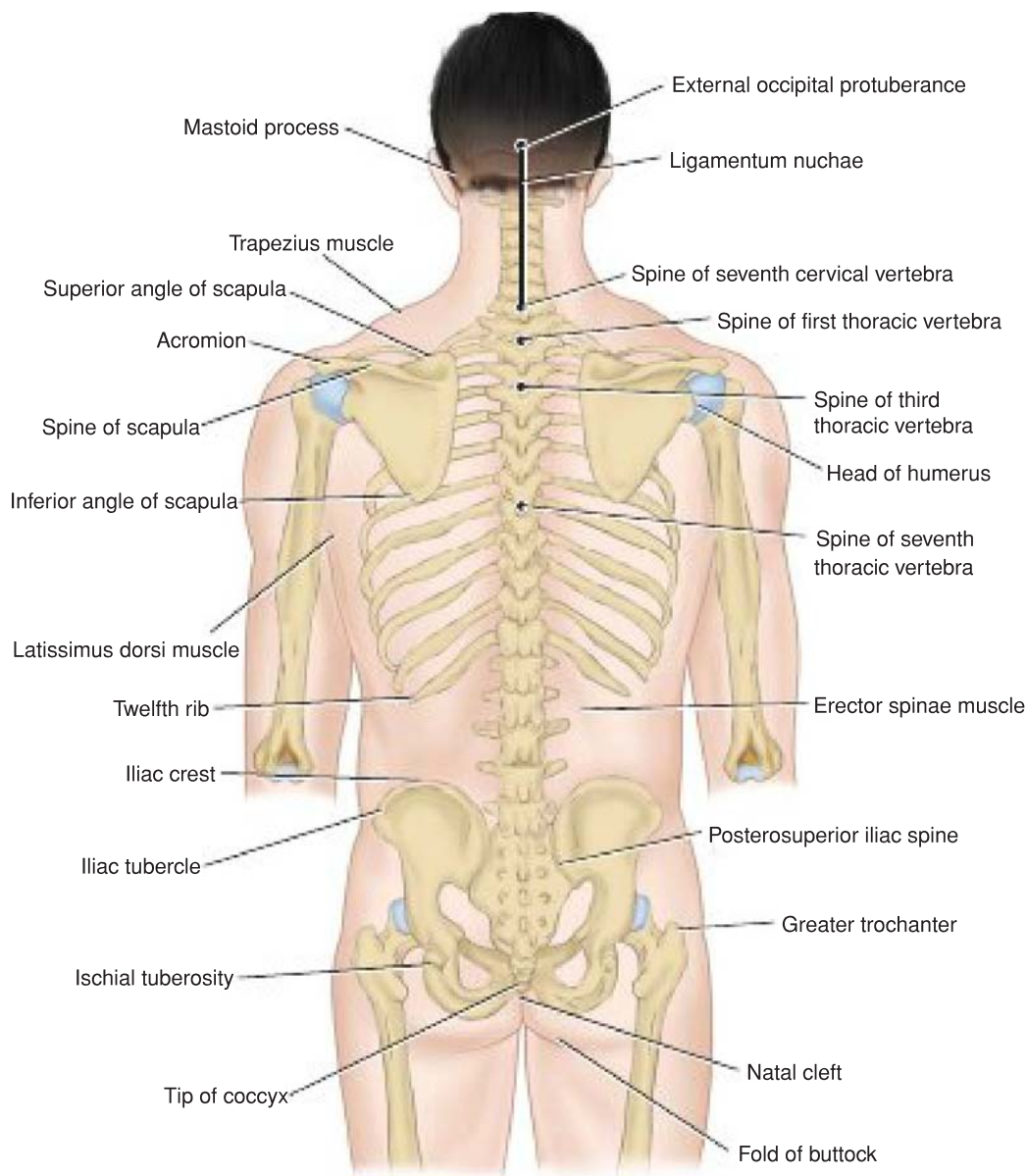


Figure 4-1 Posterior view of the skeleton showing the vertebral column. The surface marking of the external occipital protuberance of the skull, the ligamentum nuchae (solid black line) and some important palpable spines (solid dots) are also shown.

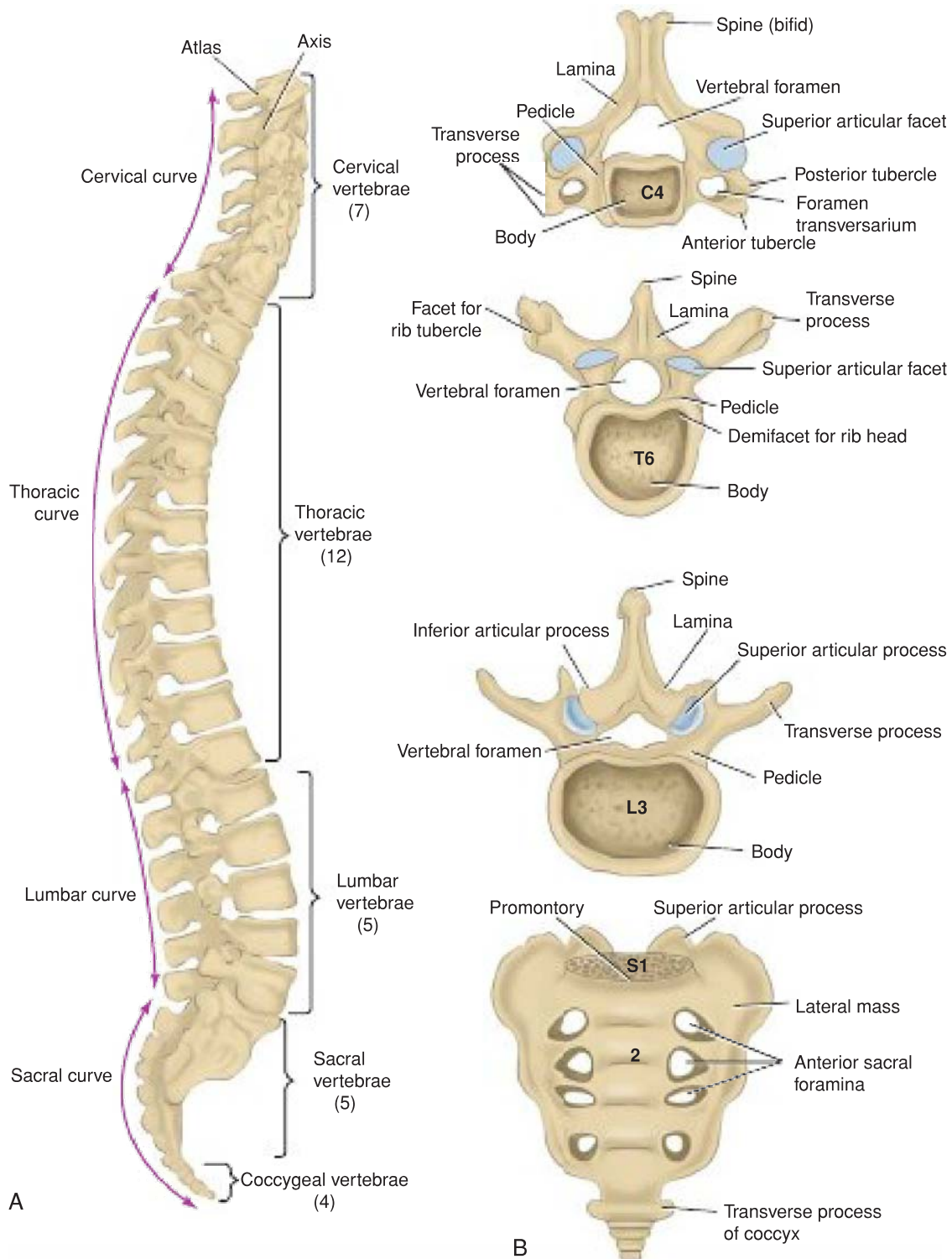


Figure 4-2 **A:** Lateral view of the vertebral column. **B:** General features of different kinds of vertebrae.

The **articular processes** are vertically arranged and consist of two superior and two inferior processes. They arise from the junction of the laminae and the pedicles. The two superior articular processes of one vertebral arch articulate with the two inferior articular processes of the arch above, forming two synovial joints.

The pedicles are notched on their upper and lower borders, forming the **superior** and **inferior vertebral notches**. On each side, the superior notch of one vertebra and the inferior notch of an adjacent vertebra together form an **intervertebral foramen**. These foramina, in an articulated skeleton, serve to transmit

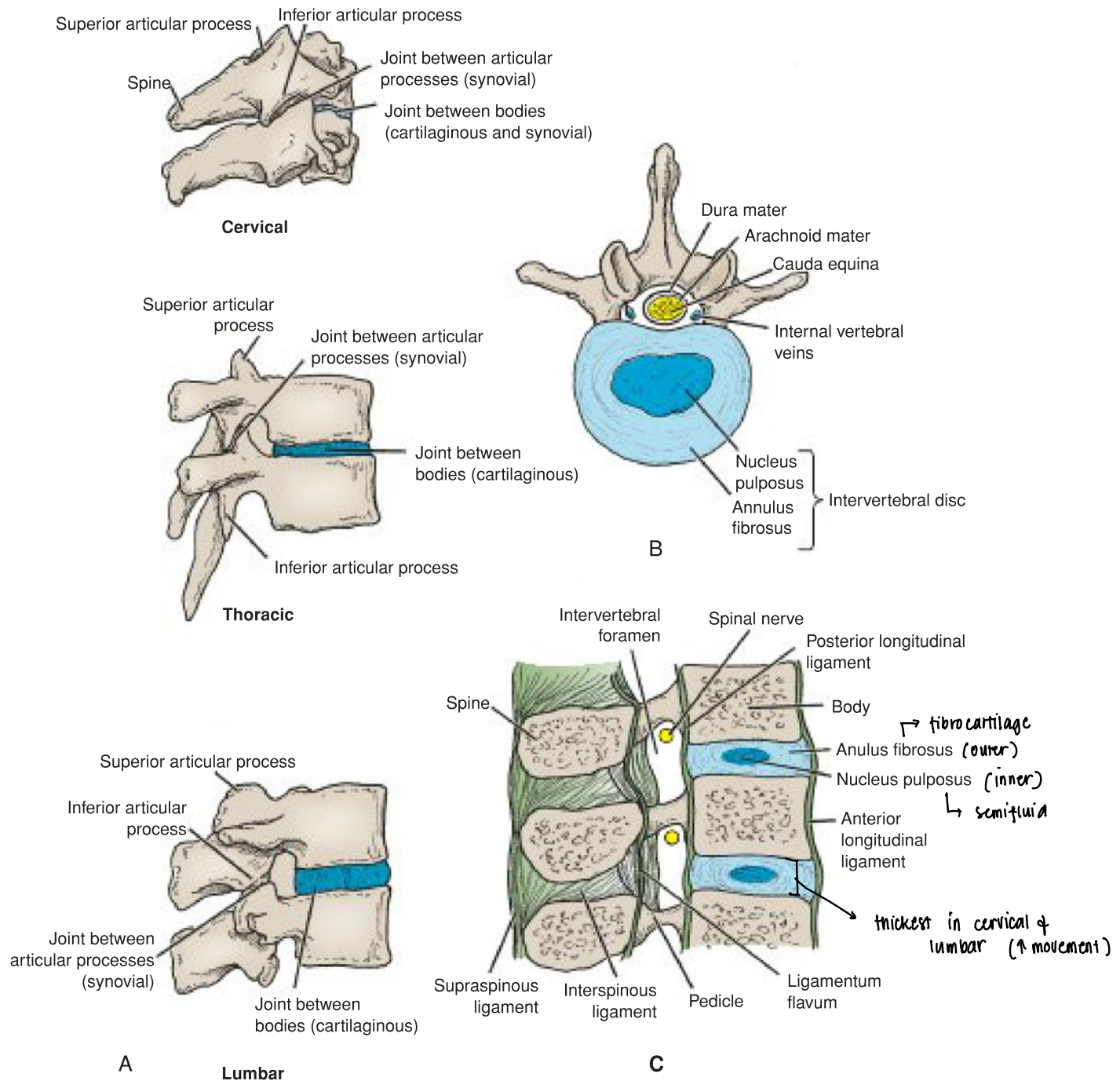


Figure 4-3 **A:** Joints in the cervical, thoracic, and lumbar regions of the vertebral column. **B:** Third lumbar vertebra seen from above showing the relationship between intervertebral disc and cauda equina. **C:** Lumbar vertebrae in sagittal section showing vertebral ligament relationships.

the spinal nerves and blood vessels. The anterior and posterior nerve roots of a spinal nerve unite within these foramina with their coverings of dura to form the segmental spinal nerves.

Vertebral Column Joints

Below the axis, the vertebrae articulate with each other by means of cartilaginous joints between their bodies and by synovial joints between their articular processes.

Joints Between Two Vertebral Bodies

Sandwiched between the vertebral bodies is an intervertebral disc of fibrocartilage (Fig. 4-3).

Intervertebral Discs

The intervertebral discs are thickest in the cervical and lumbar regions, where the movements of the vertebral column are greatest. They serve as shock absorbers when the load on the vertebral column is suddenly

increased. Unfortunately, their resilience is gradually lost with advancing age.

Each disc consists of a peripheral part, the annulus fibrosus, and a central part, the nucleus pulposus (Fig. 4-3B,C). The **annulus fibrosus** is composed of fibrocartilage, which is strongly attached to the vertebral bodies and the anterior and posterior longitudinal ligaments of the vertebral column.

The **nucleus pulposus** in the young is an ovoid mass of gelatinous material. It is normally under pressure and situated slightly nearer to the posterior than to the anterior margin of the disc. The upper and lower surfaces of the bodies of adjacent vertebrae that abut onto the disc are covered with thin plates of hyaline cartilage.

The semifluid nature of the nucleus pulposus allows it to change shape and permits one vertebra to rock forward or backward on another. A sudden increase in the compression load on the vertebral column flattens the nucleus pulposus, which is accommodated by the resilience of the surrounding annulus fibrosus. Sometimes, the outward thrust is too great for the annulus fibrosus and it ruptures, allowing the nucleus pulposus to herniate and protrude into the vertebral canal, where it may press on the spinal nerve roots, the spinal nerve, or even the spinal cord.

With advancing age, the nucleus pulposus becomes smaller and is replaced by fibrocartilage. The collagen fibers of the annulus degenerate, and, as a result, the annulus cannot always contain the nucleus pulposus under stress. In old age, the discs are thin and less elastic, and distinguishing the nucleus from the annulus is no longer possible.

Ligaments

The **anterior** and **posterior longitudinal ligaments** run as continuous bands down the anterior and posterior

surfaces of the vertebral column from the skull to the sacrum (Fig. 4-3C). The anterior ligament is wide and is strongly attached to the front and sides of the vertebral bodies and to the intervertebral discs. The posterior ligament is weak and narrow and is attached to the posterior borders of the discs.

Joints Between Two Vertebral Arches

The joints between two vertebral arches consist of synovial joints between the superior and inferior articular processes of adjacent vertebrae (Fig. 4-3A).

Ligaments

Refer to Figure 4-3C.

- The **supraspinous ligament** runs between the tips of adjacent spines.
- The **interspinous ligament** connects adjacent spines.
- The **intertransverse ligaments** run between adjacent transverse processes.
- The **ligamentum flavum** connects the laminae of adjacent vertebrae.

In the cervical region, the supraspinous and interspinous ligaments are greatly thickened to form the strong **ligamentum nuchae**.

Vertebral Joint Nerve Supply

The joints between the vertebral bodies are innervated by the small meningeal branches of each spinal nerve (Fig. 4-4). The joints between the articular processes are innervated by branches from the posterior rami of the spinal nerves; the joints of any particular level receive nerve fibers from two adjacent spinal nerves.

The atlanto-occipital joints and the atlanto-axial joints should be reviewed in a textbook of gross anatomy.

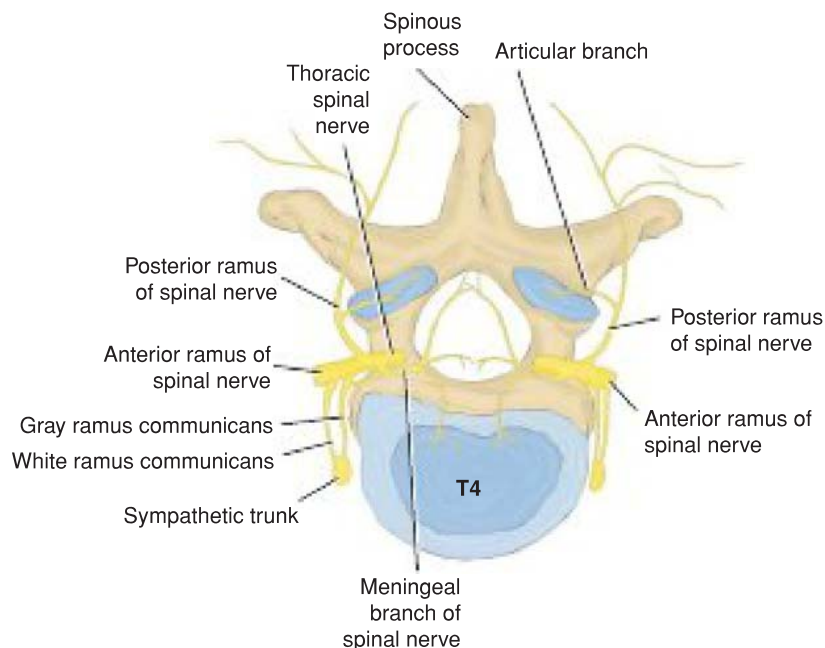


Figure 4-4 The innervation of vertebral joints. At any particular vertebral level, the joints receive nerve fibers from two adjacent spinal nerves.

SPINAL CORD

The spinal cord is roughly cylindrical in shape. It begins superiorly at the foramen magnum in the skull, where it is continuous with the **medulla oblongata** of the brain, and it terminates inferiorly in the adult at the level of the **lower border of the first lumbar vertebra**. In the young child, it is relatively longer and usually ends at the upper border of the third lumbar vertebra. Thus, it occupies the upper two thirds of the **vertebral canal** of the vertebral column and is surrounded by the three meninges, the **dura mater**, the arachnoid mater, and

the **pia mater**. Further protection is provided by the **cerebrospinal fluid (CSF)**, which surrounds the spinal cord in the **subarachnoid space**.

In the cervical region, where it gives origin to the brachial plexus, and in the lower thoracic and lumbar regions, where it gives origin to the lumbosacral plexus, the spinal cord is fusiformly enlarged; the enlargements are referred to as the **cervical** and **lumbar enlargements** (Fig. 4-5). Inferiorly, the spinal cord tapers off into the **conus medullaris**, from the apex of which a prolongation of the pia mater, the **filum terminale**, descends to attach to the posterior

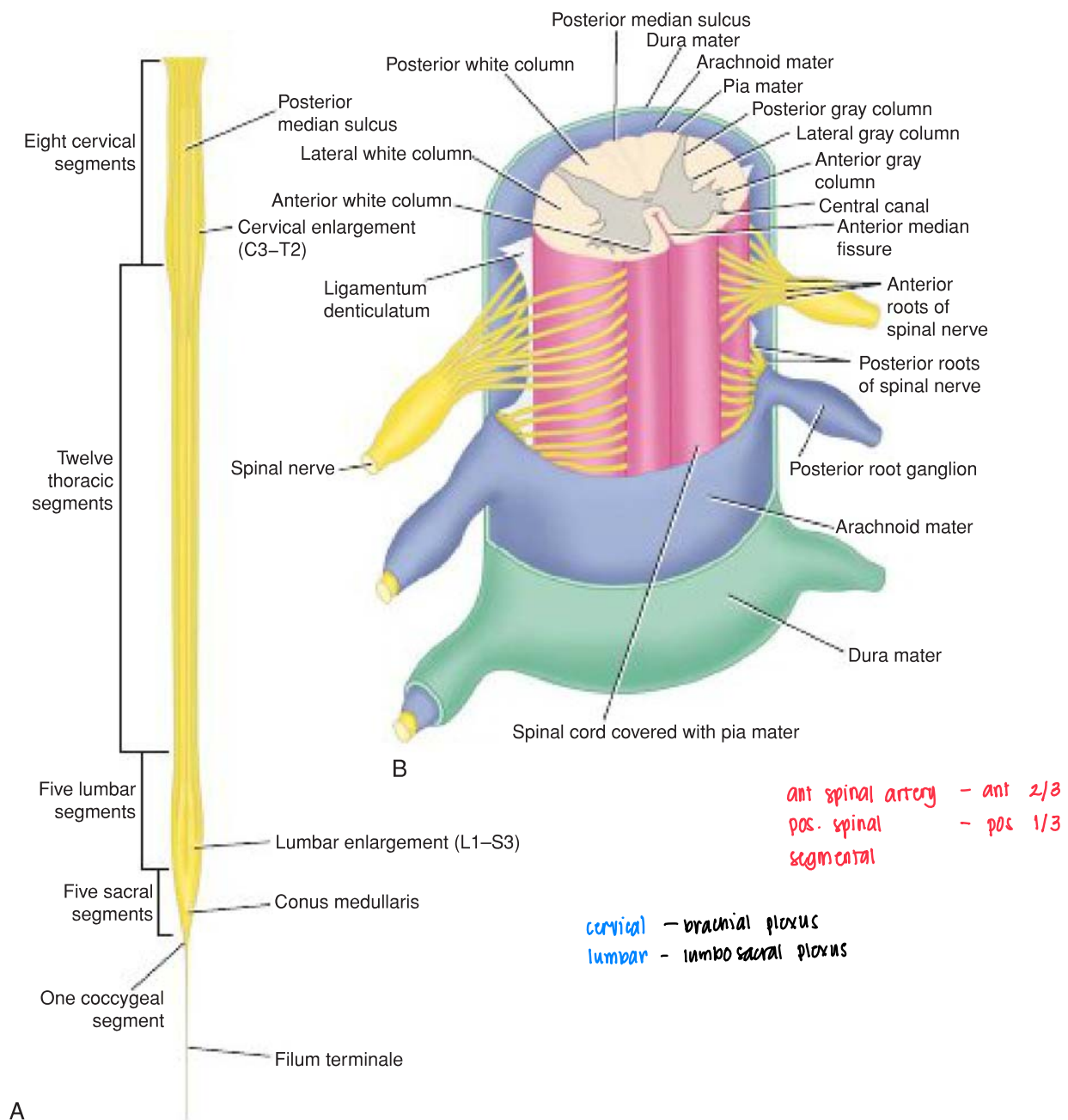


Figure 4-5 Spinal cord. **A:** Posterior view, showing cervical and lumbar enlargements. **B:** Three segments of the spinal cord showing the coverings of dura mater, arachnoid mater, and pia mater.

surface of the coccyx. The cord possesses a deep longitudinal fissure called the **anterior median fissure** in the midline anteriorly and a shallow furrow called the **posterior median sulcus** on the posterior surface (Fig. 4-5B).

Along the entire length of the spinal cord, 31 pairs of spinal nerves are attached by the **anterior** or **motor roots** and the **posterior** or **sensory roots** (Fig. 4-5B). Each root is attached to the cord by a series of rootlets, which extend the whole length of the corresponding

segment of the cord. Each posterior nerve root possesses a **posterior root ganglion**, the cells of which give rise to peripheral and central nerve fibers.

Spinal Cord Structure

The spinal cord is composed of an inner core of gray matter, which is surrounded by an outer covering of white matter (Fig. 4-6); there is no indication that the cord is segmented.

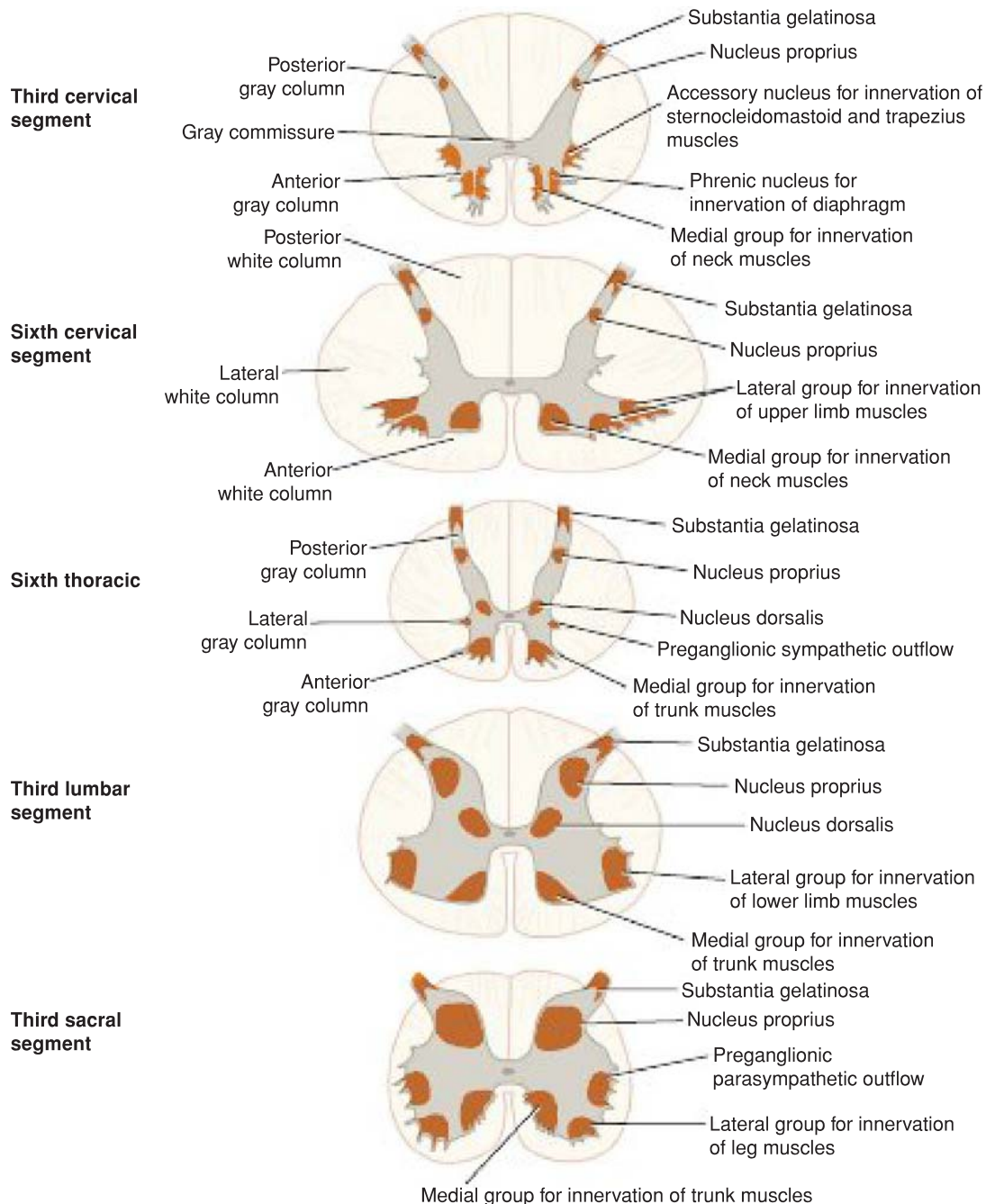


Figure 4-6 Transverse sections of the spinal cord at different levels showing the arrangement of the gray matter and white matter.

Table 4-1 Comparison of Structural Details in Different Regions of the Spinal Cord^a

Region	Shape	White Matter	Gray Matter		
			Anterior Gray Column	Posterior Gray Column	Lateral Gray Column
Cervical	Oval	Fasciculus cuneatus and fasciculus gracilis present	Medial group of cells for neck muscles; central group of cells for accessory nucleus (C1–C5) and phrenic nucleus (C3–C5); lateral group of cells for upper limb muscles	Substantia gelatinosa present, continuous with Sp.N. of cranial nerve V at level C2; nucleus proprius present; nucleus dorsalis (Clarke column) absent	Absent
Thoracic	Round	Fasciculus cuneatus (T1–T6) and fasciculus gracilis present	Medial group of cells for trunk muscles	Substantia gelatinosa, nucleus proprius, and visceral afferent nucleus present.	Present; gives rise to preganglionic sympathetic fibers
Lumbar	Round to oval	Fasciculus cuneatus absent; fasciculus gracilis present	Medial group of cells for lower limb muscles; central group of cells for lumbosacral nerve	Substantia gelatinosa, nucleus proprius, nucleus dorsalis (Clarke column) at L1–L4, and visceral afferent nucleus present	Present (L1–L2 [3]); gives rise to preganglionic sympathetic fibers
Sacral	Round	Small amount; fasciculus cuneatus absent; fasciculus gracilis present	Medial group of cells for lower limb and perineal muscles	Substantia gelatinosa and nucleus proprius present	Absent; group of cells present at S2–S4, for parasympathetic outflow

^aThe information in this table is useful for identifying the specific level of the spinal cord from which a section has been taken.

For a comparison of the structural details in different regions of the spinal cord, see Table 4-1.

Gray Matter

On cross section, the gray matter is seen as an H-shaped pillar with **anterior** and **posterior gray columns**, or **horns**, united by a thin **gray commissure** containing the small **central canal**. A small **lateral gray column** or **horn** is present in the thoracic and upper lumbar segments of the cord. The amount of gray matter present at any given level of the spinal cord is related to the amount of muscle innervated at that level. Thus, its size is greatest within the cervical and lumbosacral enlargements of the cord, which innervate the muscles of the upper and lower limbs, respectively (Figs. 4-7 to 4-10; also see Fig. 4-6).

Structure

As in other CNS regions, spinal cord gray matter consists of a mixture of nerve cells and their processes, neuroglia, and blood vessels. The nerve cells are multipolar, and the neuroglia forms an intricate network around the nerve cell bodies and their neurites.

ANTERIOR GRAY COLUMN NERVE CELL GROUPS

Most nerve cells are large and multipolar, and their axons pass out in the anterior roots of the spinal nerves

as **α efferents**, which innervate skeletal muscles. The smaller nerve cells are also multipolar, and the axons of many of these pass out in the anterior roots of the spinal nerves as **γ efferents**, which innervate the intrafusal muscle fibers of neuromuscular spindles.

For practical purposes, the nerve cells of the anterior gray column can be divided into three basic groups or columns: medial, central, and lateral (Fig. 4-6).

The medial group is present in most segments of the spinal cord and is responsible for innervating the skeletal muscles of the neck and trunk, including the intercostal and abdominal musculature.

The central group is the smallest and is present in some cervical and lumbosacral segments. In the cervical part of the cord, some of these nerve cells (segments C3–C5) specifically innervate the diaphragm and are collectively referred to as the **phrenic nucleus** (Fig. 4-7). In the upper five or six cervical segments, some of the nerve cells innervate the sternocleidomastoid and trapezius muscles and are referred to as the **accessory nucleus**. The axons of these cells form the spinal part of the accessory nerve. The **lumbosacral nucleus** present in the second lumbar down to the first sacral segment of the cord is made up of nerve cells whose axons have an unknown distribution.

The lateral group is present in the cervical and lumbosacral segments of the cord and is responsible for

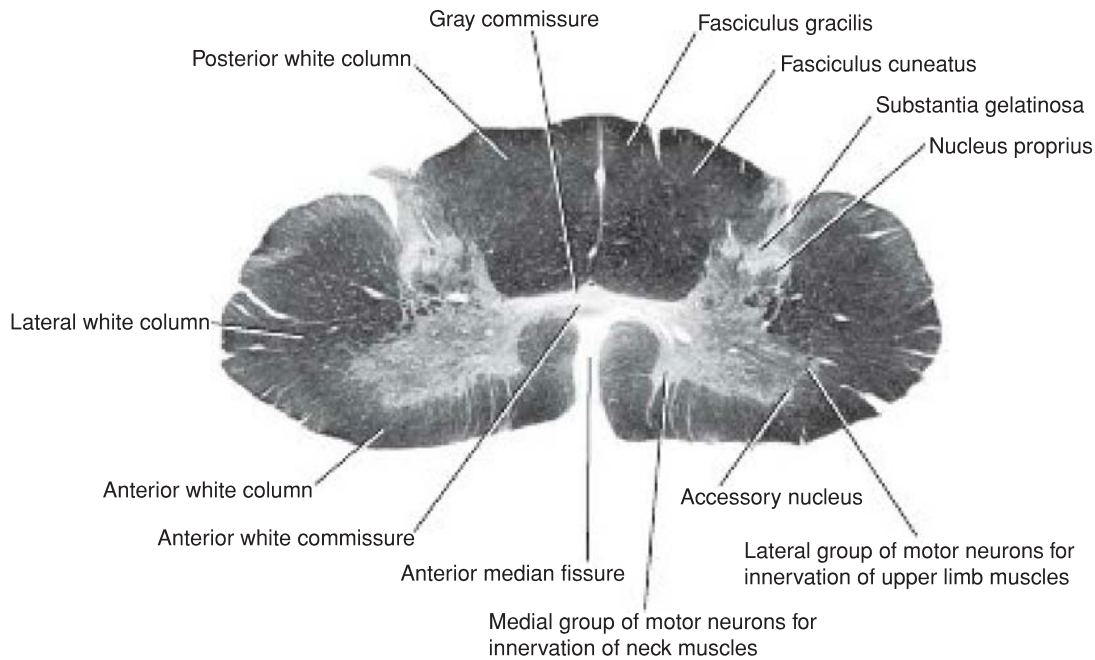


Figure 4-7 Transverse section of the spinal cord at the level of the fifth cervical segment. (Weigert stain.)

innervating the skeletal muscles of the limbs (Figs. 4-6, 4-7, 4-9, and 4-10).

POSTERIOR GRAY COLUMN NERVE CELL GROUPS

Two of the four nerve cell groups of the posterior gray column extend throughout the length of the cord; the other two are restricted to the thoracic and lumbar segments.

The **substantia gelatinosa group** is situated at the apex of the posterior gray column throughout the length of the spinal cord (Figs. 4-6 to 4-10). It is largely

composed of Golgi type II neurons and receives afferent fibers concerned with pain, temperature, and touch from the posterior root. Furthermore, it receives input from descending fibers from supraspinal levels. Pain and temperature inputs are thought to be modified by excitatory or inhibitory information from other sensory inputs and by information from the cerebral cortex.

The **nucleus proprius** is a group of large nerve cells situated anterior to the substantia gelatinosa throughout the spinal cord (Figs. 4-6 to 4-10). This nucleus constitutes the main bulk of cells present in the posterior

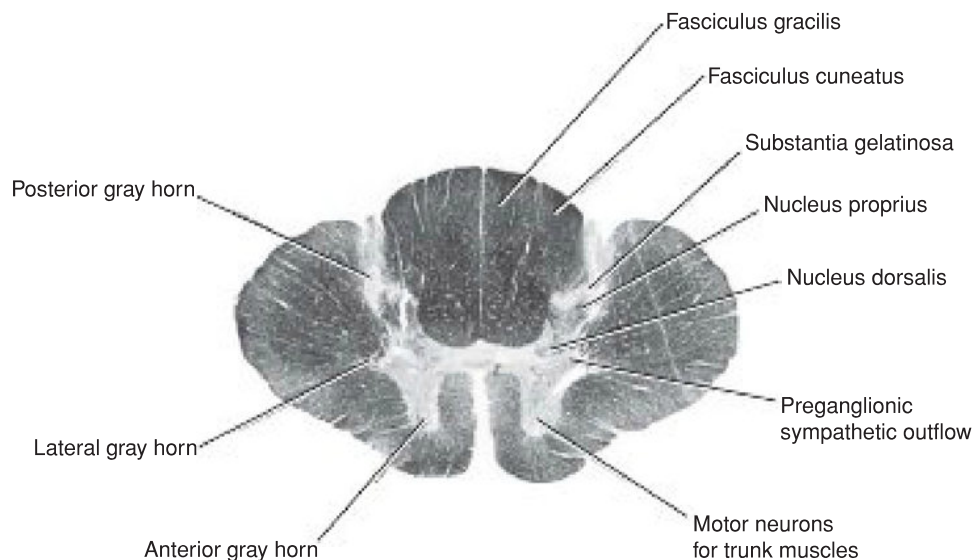


Figure 4-8 Transverse section of the spinal cord at the level of the second thoracic segment. (Weigert stain.)

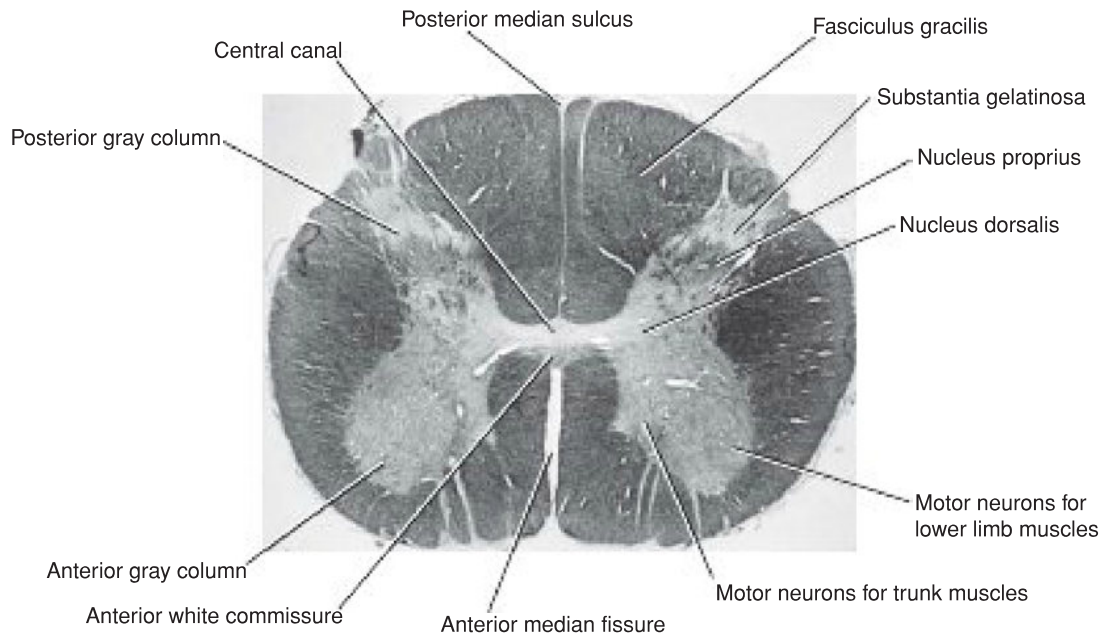


Figure 4-9 Transverse section of the spinal cord at the level of the fourth lumbar segment. (Weigert stain.)

gray column and receives fibers from the posterior white column that are associated with the senses of position and movement (proprioception), two-point discrimination, and vibration.

The **nucleus dorsalis (Clarke column)** is a group of nerve cells situated at the base of the posterior gray column and extending from the eighth cervical segment

caudally to the third or fourth lumbar segment (Figs. 4-6 to 4-9). Most of the cells are comparatively large and are associated with proprioceptive endings (neuromuscular spindles and tendon spindles).

The **visceral afferent nucleus** is a group of nerve cells of medium size situated lateral to the nucleus dorsalis; it extends from the first thoracic to

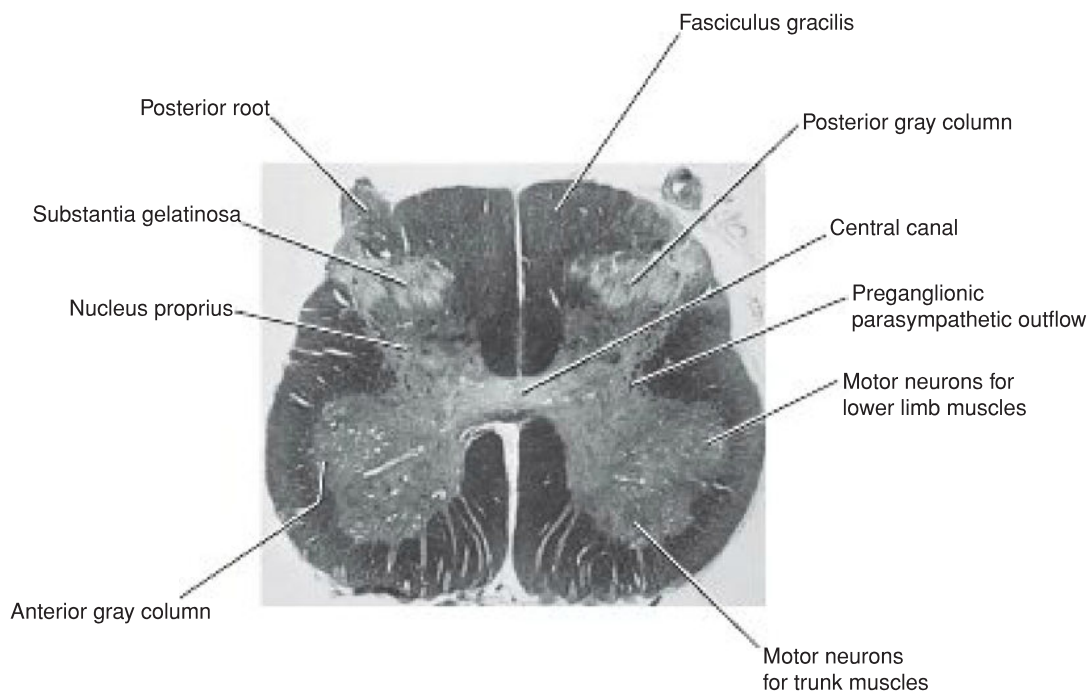


Figure 4-10 Transverse section of the spinal cord at the level of the second sacral segment. (Weigert stain.)

the third lumbar segment of the spinal cord. It is believed to be associated with receiving visceral afferent information.

LATERAL GRAY COLUMN NERVE CELL GROUPS

The intermediolateral group of cells form the small lateral gray column, which extends from the first thoracic to the second or third lumbar segment of the spinal cord (Figs. 4-6 and 4-8). The cells are relatively small and give rise to preganglionic sympathetic fibers.

A similar group of cells found in the second, third, and fourth sacral segments of the spinal cord give rise to preganglionic parasympathetic fibers (Figs. 4-6 and 4-10).

GRAY COMMISSURE AND CENTRAL CANAL

In transverse sections of the spinal cord, the anterior and posterior gray columns on each side are connected by a transverse **gray commissure**; the gray matter resembles the letter H (Figs. 4-6 to 4-10). The **central canal** is situated in the center of the gray commissure. The part of the gray commissure that is situated posterior to the central canal is the **posterior gray commissure**; the anterior part is the **anterior gray commissure**.

The central canal is present throughout the spinal cord. Superiorly, it is continuous with the central canal of the caudal half of the medulla oblongata; above this, it opens into the cavity of the fourth ventricle. Inferiorly in the conus medullaris, it expands into the fusiform **terminal ventricle** and terminates below within the root of the filum terminale. It is filled with CSF and is lined with ciliated columnar epithelium, the **ependyma**.

Thus, the central canal is closed inferiorly and opens superiorly into the fourth ventricle.

White Matter

The white matter, for purposes of description, may be divided into **anterior**, **lateral**, and **posterior white columns** or **funiculi** (Figs. 4-5 to 4-10). The anterior column on each side lies between the midline and the point of emergence of the anterior nerve roots; the lateral column lies between the emergence of the anterior nerve roots and the entry of the posterior nerve roots; the posterior column lies between the entry of the posterior nerve roots and the midline.

Structure

As in other CNS regions, the spinal cord white matter consists of a mixture of nerve fibers, neuroglia, and blood vessels. It surrounds the gray matter, and its white color is due to the high proportion of myelinated nerve fibers.

NERVE FIBER TRACT ARRANGEMENT

The arrangement of the nerve fiber tracts within the spinal cord has been deduced as the result of animal experimentation and study of the human spinal cord for degenerative nerve fibers resulting from injury or disease. Although some nerve tracts are concentrated in certain areas of the white matter, considerable overlap is present. For purposes of description, the spinal tracts are divided into ascending, descending, and intersegmental tracts, and their relative positions in the white matter are described below. A simplified diagram, showing the general arrangement of the major tracts, is shown in Figure 4-11.

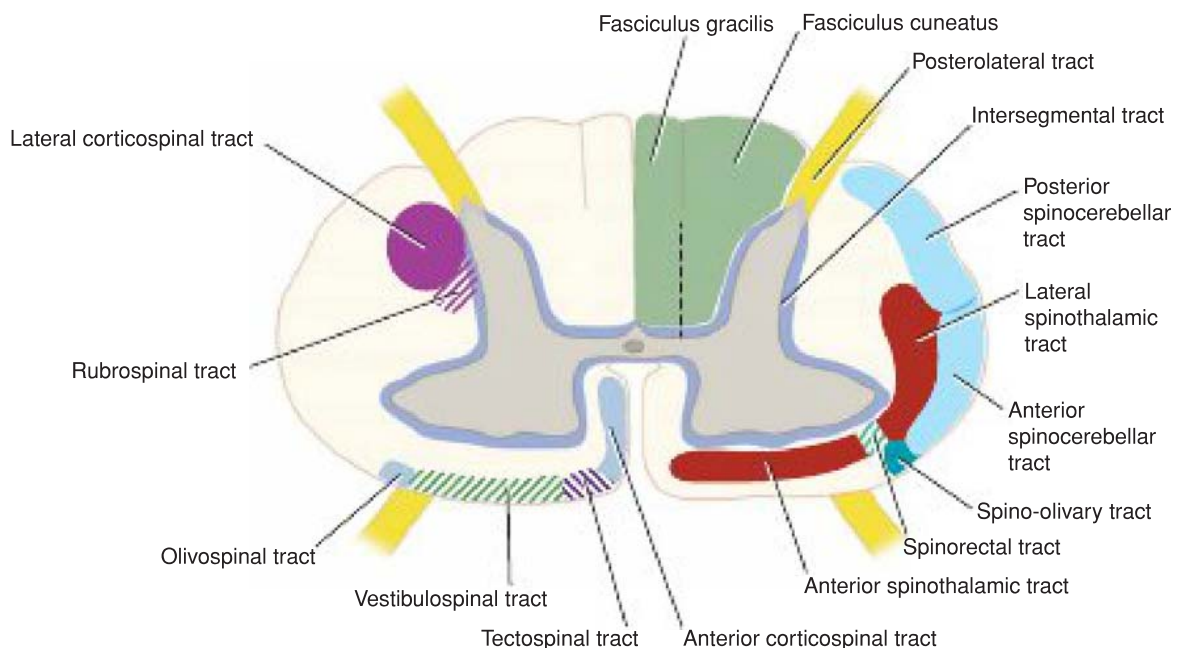


Figure 4-11 Transverse section of the spinal cord at the midcervical level showing the general arrangement of the ascending tracts on the right and the descending tracts on the left.

ASCENDING TRACTS

On entering the spinal cord, the sensory nerve fibers of different sizes and functions are sorted out and segregated into nerve bundles or **tracts** in the white matter (Fig. 4-12; also see Fig. 4-11). Some of the nerve fibers serve to link different segments of the spinal cord, while others ascend from the spinal cord to higher centers and thus connect the spinal cord with the brain. These bundles of the ascending fibers are referred to as the **ascending tracts**.

The ascending tracts conduct two types of afferent information, which may or may not reach consciousness. **Exteroceptive** information originates from

↳ from outside

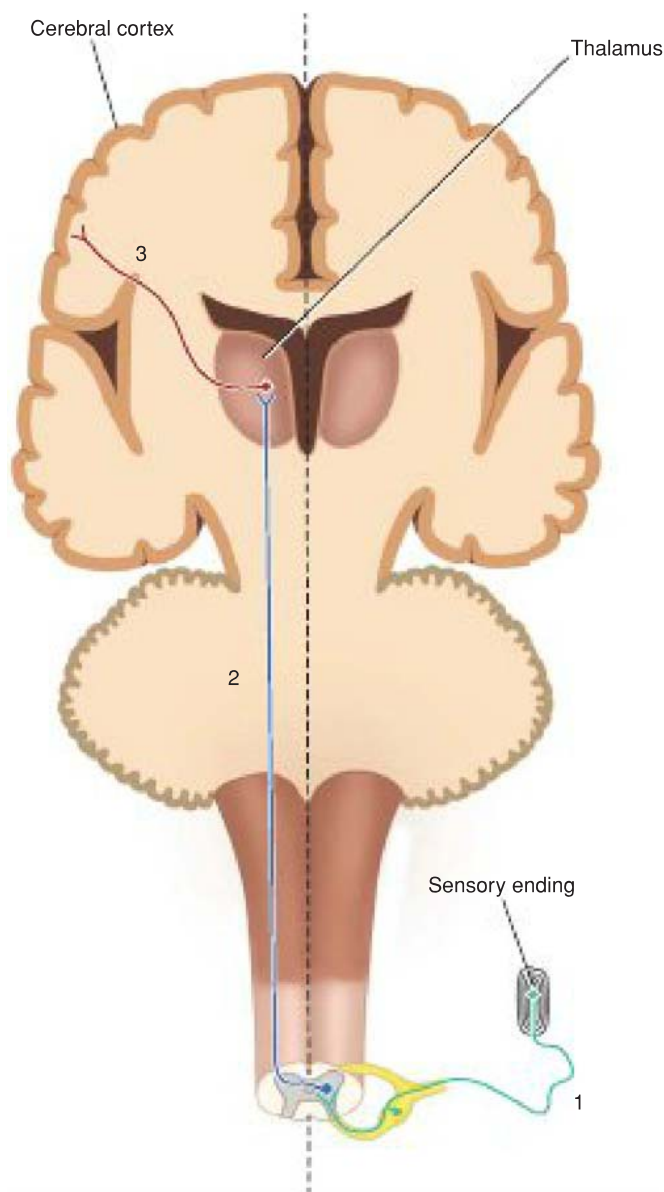


Figure 4-12 Simplest form of the ascending sensory pathway from the sensory nerve ending to the cerebral cortex. Note the three neurons involved.

outside the body, such as pain, temperature, and touch.

Proprioceptive information originates from inside the body, for example, from muscles and joints.

↳ inside originate

Anatomical Organization

General information from the peripheral sensory endings is conducted through the nervous system by a series of neurons. In its simplest form, the ascending pathway to consciousness consists of three neurons (Fig. 4-12). The first neuron, the **first-order neuron**, has its cell body in the **posterior root ganglion** of the spinal nerve. A peripheral process connects with a sensory receptor ending, whereas a central process enters the spinal cord through the posterior root to synapse on the second-order neuron. The **second-order neuron** gives rise to an axon that **decussates** (crosses to the opposite side) and ascends to a higher level of the CNS, where it **synapses** with the **third-order neuron**. The third-order neuron is usually in the **thalamus** and gives rise to a projection fiber that passes to a sensory region of the cerebral cortex. This **three-neuron chain** is the most common arrangement, but some afferent pathways use more or fewer neurons. Many of the neurons in the ascending pathways branch and give a major input into the reticular formation, which, in turn, activates the cerebral cortex, maintaining wakefulness. Other branches pass to motor neurons and participate in reflex muscular activity.

Functions

Painful and thermal sensations ascend in the **lateral spinothalamic tract**; light (crude) touch and pressure ascend in the **anterior spinothalamic tract** (Fig. 4-13). **Discriminative touch**—that is, the ability to localize accurately the area of the body touched and also to be aware that two points are touched simultaneously, even though they are close together (**two-point discrimination**)—ascends in the **posterior white columns**. Also ascending in the posterior white columns is information from muscles and joints pertaining to movement and position of different parts of the body. In addition, vibratory sensations ascend in the posterior white column. Unconscious information from muscles, joints, the skin, and subcutaneous tissue reaches the cerebellum by way of the anterior and posterior spinocerebellar tracts and by the cuneocerebellar tract. Pain, thermal, and tactile information are passed to the superior colliculus of the midbrain through the spinotectal tract for the purpose of spinovisual reflexes. The spinoreticular tract provides a pathway from the muscles, joints, and skin to the reticular formation, while the spino-olivary tract provides an indirect pathway for further afferent information to reach the cerebellum.

Pain and Temperature Pathways

The pain and thermal receptors in the skin and other tissues are free nerve endings. The pain impulses are transmitted to the spinal cord in fast-conducting

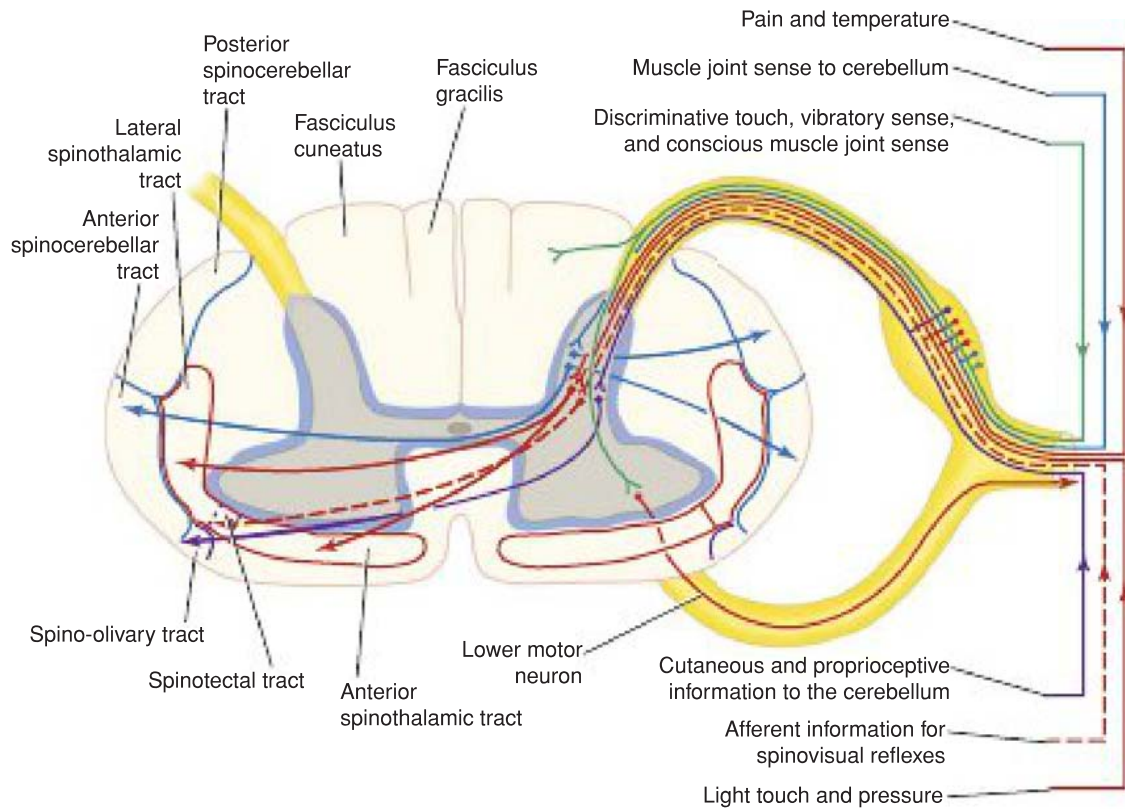


Figure 4-13 Transverse section of the spinal cord showing the origin of the main ascending sensory tracts. Note that the sensations of pain and temperature ascend in the lateral spinothalamic tract, and light touch and pressure ascend in the anterior spinothalamic tract.

δ A-type fibers and slow-conducting C-type fibers. The fast-conducting fibers alert the individual to initial sharp pain, and the slow-conducting fibers are responsible for prolonged burning, aching pain. The

sensations of heat and cold also travel by δ A and C fibers.

The main somatosensory pathways are summarized in Table 4-2.

Table 4-2 Main Somatosensory Pathways to Consciousness^a

Sensation	Receptor	First-Order Neuron	Second-Order Neuron	Third-Order Neuron	Pathways	Destination
Pain and temperature <i>lateral spinothalamic tract</i>	Free nerve endings	Posterior root ganglion	Substantia gelatinosa	Ventral posterolateral nucleus of thalamus	Lateral spinothalamic, spinal lemniscus	Posterior central gyrus
Light touch and pressure <i>anterior spinothalamic tract</i>	Free nerve endings	Posterior root ganglion	Substantia gelatinosa	Ventral posterolateral nucleus of thalamus	Anterior spinothalamic, spinal lemniscus	Posterior central gyrus
Discriminative touch, vibratory sense, conscious muscle joint sense	Meissner corpuscles, pacinian corpuscles, muscle spindles, tendon organs	Posterior root ganglion	Nuclei gracilis and cuneatus	Ventral posterolateral nucleus of thalamus	Fasciculi gracilis and cuneatus, medial lemniscus	Posterior central gyrus

^aNote that all ascending pathways send branches to the reticular activating system.

*gracilis - lower
cuneatus - upper*

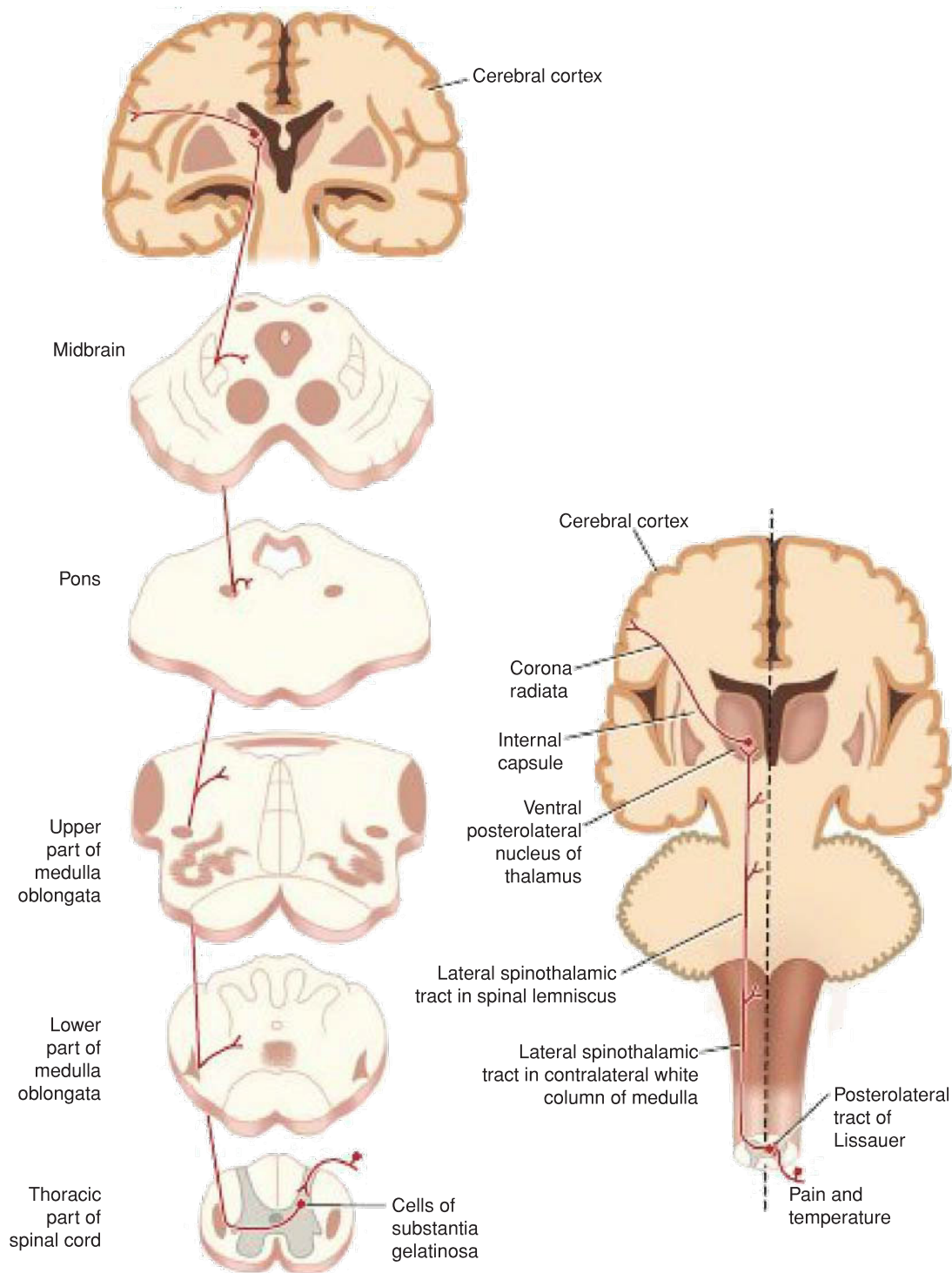


Figure 4-14 Pain and temperature pathways.

Lateral Spinothalamic Tract

Axons entering the spinal cord from the posterior root ganglion proceed to the tip of the posterior gray column and divide into ascending and descending branches (Fig. 4-14). These branches travel for a distance of one or two segments of the spinal cord and form the **posterolateral tract of Lissauer**. These fibers of the

first-order neuron terminate by synapsing with cells in the posterior gray column, including cells in the substantia gelatinosa. Substance P, a peptide, is thought to be the neurotransmitter at these synapses.

Axons of the second-order neurons now **cross obliquely to the opposite side in the anterior gray and white commissures within one spinal segment of**

the cord, ascending in the contralateral white column as the lateral spinothalamic tract. The lateral spinothalamic tract lies medial to the anterior spinocerebellar tract. As the lateral spinothalamic tract ascends through the spinal cord, new fibers are added to the anteromedial aspect of the tract. Thus, in the upper cervical segments of the cord, the sacral fibers are lateral and the cervical segments are medial. The fibers carrying pain are situated slightly anterior to those conducting temperature.

As the lateral spinothalamic tract ascends through the medulla oblongata, it lies near the lateral surface and between the inferior olivary nucleus and the nucleus of the spinal tract of the trigeminal nerve. It is now accompanied by the anterior spinothalamic tract and the spinotectal tract; together they form the **spinal lemniscus**.

The spinal lemniscus continues to ascend through the posterior part of the pons. In the midbrain, it lies in the tegmentum lateral to the medial lemniscus. Many of the fibers of the lateral spinothalamic tract end by synapsing with the third-order neuron in the ventral posterolateral nucleus of the thalamus. Crude pain and temperature sensations are probably appreciated and emotional reactions initiated here.

Axons of the third-order neurons in the ventral posterolateral nucleus of the thalamus now pass through the posterior limb of the internal capsule and the corona radiata to reach the somesthetic area in the postcentral gyrus of the cerebral cortex. The contralateral half of the body is represented as inverted, with the hand and mouth situated inferiorly and the leg situated superiorly, and with the foot and anogenital region on the medial surface of the hemisphere. (For details, see Chapter 7.) From here, the information is transmitted to other regions of the cerebral cortex to be used by motor areas and the parietal association area. The role of the cerebral cortex is interpreting the quality of the sensory information at the level of consciousness.

Pain Reception

The perception of pain is a complex phenomenon that is influenced by the emotional state and past experiences of the individual. Pain is a sensation that warns of potential injury and alerts the person to avoid or treat it.

Pain can be divided into two main types: **fast pain** and **slow pain**. Fast pain is experienced within about 0.1 second after the pain stimulus is applied; slow pain is felt 1.0 second or later after the stimulation. Fast pain is described by the patient as sharp pain, acute pain, or pricking pain and is the type of pain felt after pricking the finger with a needle. Fast pain is almost confined to the skin.

Slow pain is described as burning pain, aching pain, and throbbing pain and is produced on tissue destruction, as for example, in the development of an abscess or in severe arthritis. Slow pain can occur in any tissue of the body.

All types of pain reception take place in free nerve endings. Fast pain is experienced by mechanical or thermal types of stimuli, and slow pain may be elicited by mechanical, thermal, and chemical stimuli.

Many chemical substances have been found in extracts from damaged **tissue that will excite free nerve endings. These include serotonin; histamine; bradykinin; acids, such as lactic acid; and K⁺ ions.** The threshold for pain endings can be lowered by prostaglandins and substance P, but they cannot stimulate the endings directly by themselves.

The individual should be aware of the existence of stimuli that, if allowed to persist, will cause tissue destruction; pain receptors have little or no adaptation.

Pain Conduction to the Central Nervous System

Fast pain travels in peripheral nerves in large diameter A δ axons at velocities between 6 and 30 msec. Slow pain travels in the small-diameter C fibers at velocities between 0.5 and 2.0 msec. The fast pain impulses reach consciousness first to alert the individual to danger so that a suitable protective response may take place. Slow pain is appreciated later and lasts much longer.

Pain Conduction in the Central Nervous System

Afferent pain fibers enter the spinal cord, for example, in the posterior roots of a spinal nerve and terminate predominantly in the superficial layers of the posterior gray horn. The main excitatory neurotransmitter released by A δ fibers and C fibers is the amino acid **glutamate**. Substance P, a neuropeptide, is also released from C fibers. Whereas glutamate is a fast-acting localized neurotransmitter, substance P has a slow release and diffuses widely in the posterior horn and can influence many neurons.

The initial sharp, pricking, fast-acting pain fibers stimulate the second-order neurons of the lateral spinothalamic tract. The axons immediately cross to the opposite side of the spinal cord and ascend to the thalamus where they are relayed to the sensory postcentral gyrus. The burning, aching, slow-acting pain fibers also stimulate the second-order neurons of the lateral spinal thalamic tract in the posterior gray horn and ascend with the axons of the fast-acting pain fibers. However, most of the incoming slow fibers to the spinal cord probably take part in additional relays involving several neurons in the posterior horn before ascending in the spinal cord. The repeated arrival of noxious stimuli through C fibers in the posterior gray horn during severe injury results in an increased response of the second-order neurons. This **winding up** phenomenon is attributed to the release of the neurotransmitter glutamate from C fibers.

The fast type of pain is precisely localized. For example, if someone hits a thumb with a hammer, where the injury has occurred is clear. The slow type of pain is only poorly localized. For example, in a patient with osteoarthritis of the hip joint, the individual can only vaguely localize the pain to the hip area and not to the specific site of the disease. This may

be explained by the fact that fast pain fibers directly ascend the spinal cord in the lateral spinothalamic tract, whereas the slow pain fibers take part in multiple relays in the posterior gray horn before ascending to higher centers.

Other Terminations of the Lateral Spinothalamic Tract

As is now generally agreed, fast pain impulses travel directly up to the ventral posterolateral nucleus of the thalamus and are then relayed to the cerebral cortex.

The majority of the slow pain fibers in the lateral spinothalamic tract terminate in the reticular formation, which then activates the entire nervous system. In the lower areas of the brain, the individual becomes aware of the chronic, nauseous, suffering type of pain.

As the result of research using the positron emission tomography scan, the postcentral gyrus, the cingulate gyrus of the limbic system, and the insular gyrus are sites concerned with the reception and interpretation of nociceptor information. The postcentral gyrus is responsible for the interpretation of pain in relation to past experiences. The cingulate gyrus is involved with the interpretation of the emotional aspect of pain, whereas the insular gyrus is concerned with the interpretation of pain stimuli from the internal organs of the body and brings about an autonomic response.

Reception of pain information by the CNS is modulated first in the posterior gray horns of the spinal cord and at other sites at higher levels.

Pain Control in the Central Nervous System

Massage and the application of liniments to painful areas in the body can relieve pain. The technique of acupuncture, which was discovered several thousand years ago in China, is also beneficial in relieving pain. Low-frequency electrical stimulation of the skin also relieves pain in certain cases.

Gating Theory

Although the precise mechanism for the phenomena described above is not understood, the gating theory suggested that, at the site where the pain fiber enters the CNS, inhibition could occur by means of connector neurons excited by large, myelinated afferent fibers carrying information of nonpainful touch and pressure. The excess tactile stimulation produced by massage, for example, “closed the gate” for pain. Once the nonpainful tactile stimulation ceased, however, “the gate was opened,” and information on the painful stimuli ascended the lateral spinothalamic tract. Although the gate theory may partially explain the phenomena, the analgesia system is probably involved with the liberation of enkephalins and endorphins in the posterior gray columns.

Analgesia System

Stimulation of certain areas of the brainstem can reduce or block sensations of pain. These areas include

the periventricular area of the diencephalon, the periaqueductal gray matter of the midbrain, and midline nuclei of the brainstem. Fibers of the reticulospinal tract are believed to pass down to the spinal cord and synapse on cells concerned with pain sensation in the posterior gray column. The analgesic system can suppress both sharp pricking pain and burning pain sensations.

Recently, two compounds with morphinelike actions, **enkephalins** and **endorphins**, have been isolated in the CNS. These compounds and **serotonin** serve as neurotransmitter substances in the analgesic system of the brain, and they may inhibit the release of substance P in the posterior gray column.

Light (Crude) Touch and Pressure Pathways

These pathways are summarized in Table 4-2.

Anterior Spinothalamic Tract

Axons enter the spinal cord from the posterior root ganglion and proceed to the tip of the posterior gray column, where they divide into ascending and descending branches (Fig. 4-15). These branches travel for a distance of one or two segments of the spinal cord, contributing to the posterolateral tract of Lissauer. These fibers of the first-order neuron are believed to terminate by synapsing with cells in the substantia gelatinosa group in the posterior gray column.

Axons of the second-order neuron now **cross very obliquely to the opposite side in the anterior gray and white commissures within several spinal segments** and ascend in the opposite anterolateral white column as the anterior spinothalamic tract. As the anterior spinothalamic tract ascends through the spinal cord, new fibers are added to the medial aspect of the tract. Thus, in the upper cervical segments of the cord, the sacral fibers are mostly lateral and the cervical segments are mostly medial.

As the anterior spinothalamic tract ascends through the medulla oblongata, it accompanies the lateral spinothalamic tract and the spinotectal tract, all of which form the **spinal lemniscus**.

The spinal lemniscus continues to ascend through the posterior part of the pons, and the tegmentum of the midbrain and the fibers of the anterior spinothalamic tract terminate by synapsing with the third-order neuron in the ventral posterolateral nucleus of the thalamus. Crude awareness of touch and pressure is believed to be appreciated here.

Axons of the third-order neurons in the ventral posterolateral nucleus of the thalamus pass through the posterior limb of the **internal capsule** and the **corona radiata** to reach the somesthetic area in the postcentral gyrus of the cerebral cortex. The contralateral half of the body is represented inverted, with the hand and mouth situated inferiorly, as described previously. (For details, see Chapter 7.) The conscious appreciation of touch and pressure depends on the activity of the cerebral cortex. The sensations can be only crudely

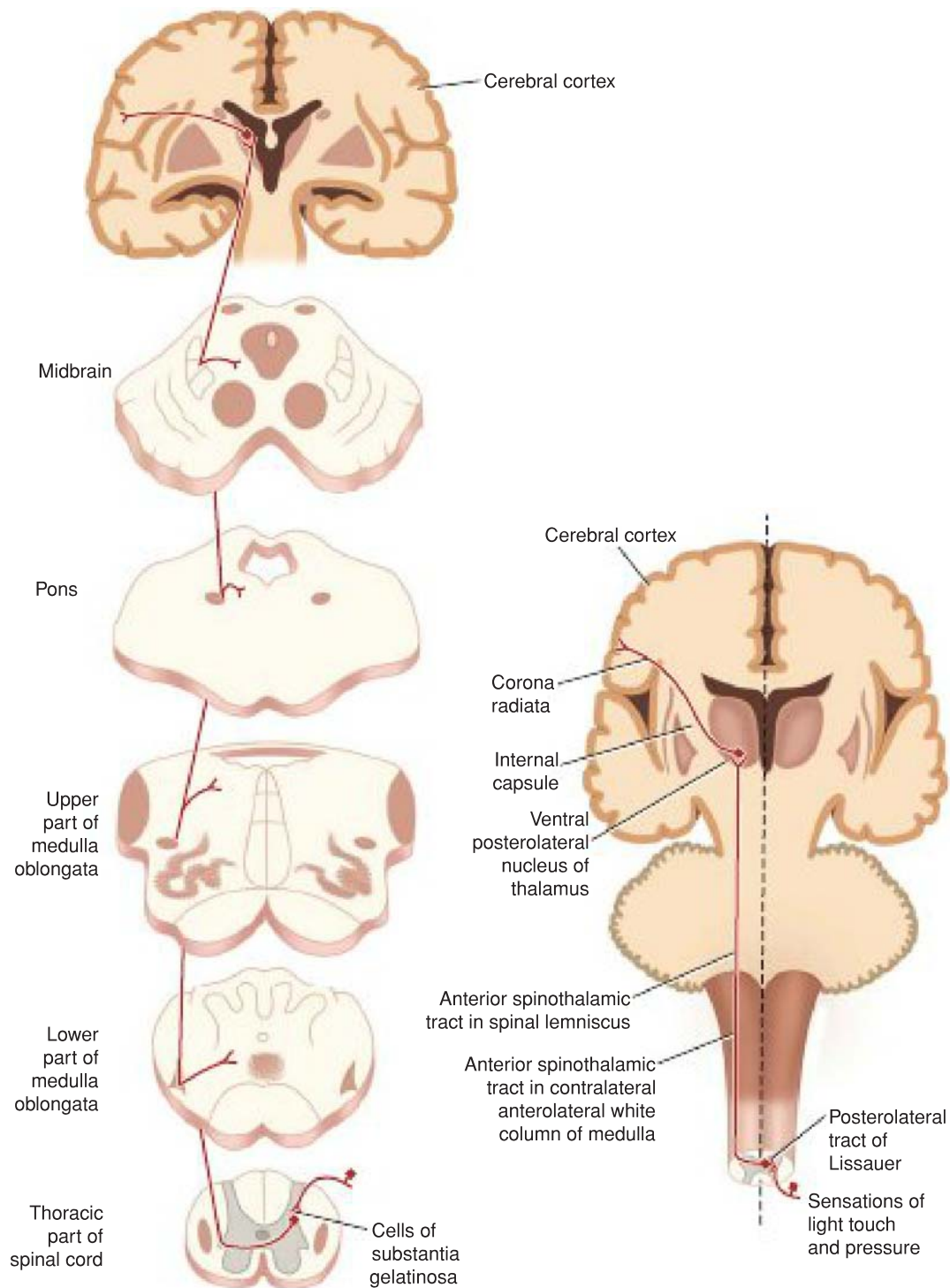


Figure 4-15 Light touch and pressure pathways.

localized, and very little discrimination of intensity is possible.

Discriminative Touch, Vibratory Sense, and Conscious Muscle Joint Sense

These pathways are summarized in Table 4-2.

Posterior White Column: Fasciculus Gracilis and Fasciculus Cuneatus

Axons enter the spinal cord from the posterior root ganglion and pass directly to the posterior white column of the same side (Fig. 4-16). Here, the fibers divide into long ascending and short descending branches. The

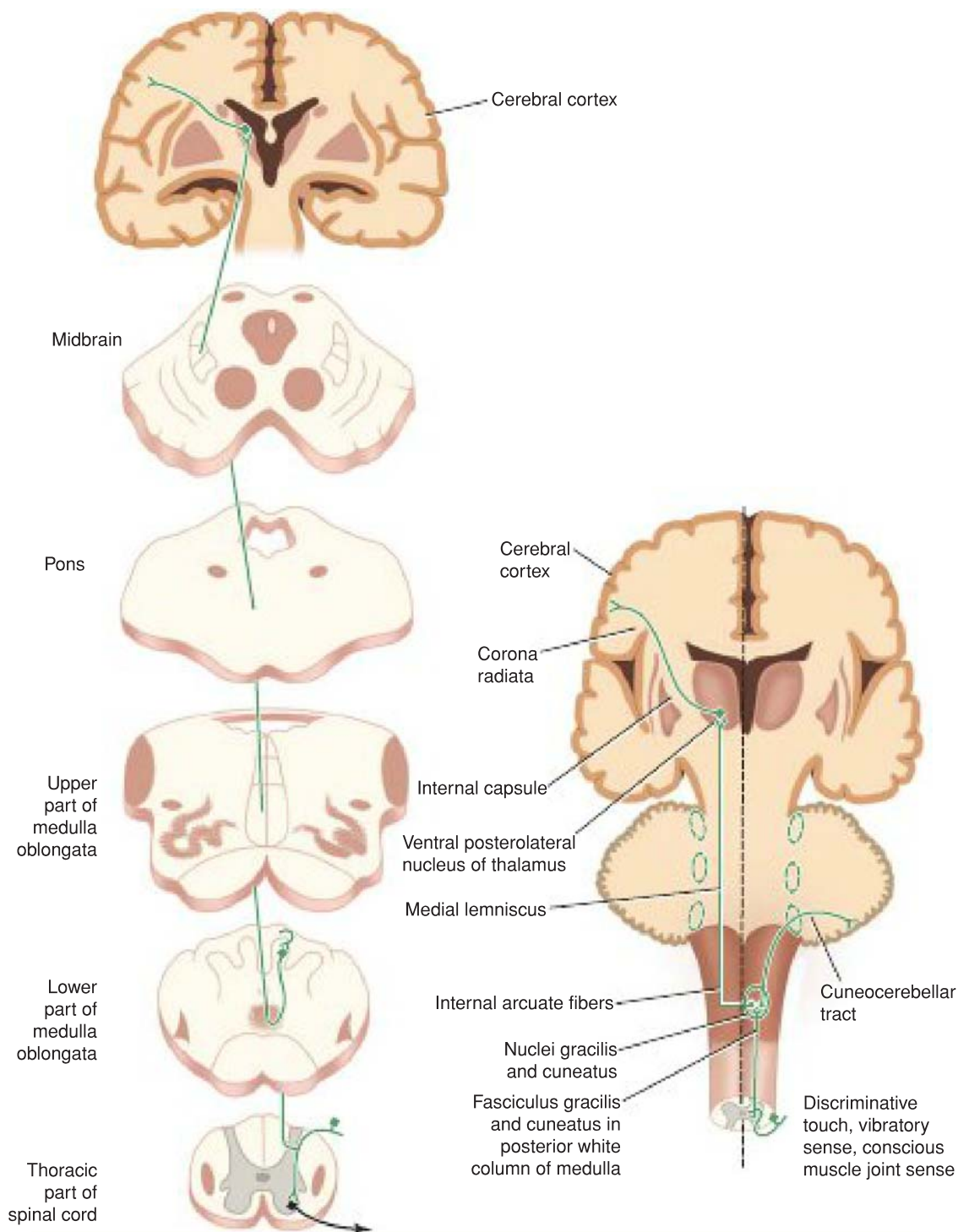


Figure 4-16 Discriminative touch, vibratory sense, and conscious muscle joint sense pathways.

descending branches pass down a variable number of segments, giving off collateral branches that synapse with cells in the posterior gray horn, with internuncial neurons, and with anterior horn cells. These short descending fibers are clearly involved with intersegmental reflexes.

Long ascending fibers may also end by synapsing with cells in the posterior gray horn, with internuncial neurons, and with anterior horn cells. This distribution may extend over numerous segments of the spinal cord. As in the case of the short descending fibers, they are involved with intersegmental reflexes.

Many of the long ascending fibers travel upward in the posterior white column as the **fasciculus gracilis** and **fasciculus cuneatus**. The fasciculus gracilis is present throughout the length of the spinal cord and contains the long ascending fibers from the sacral, lumbar, and lower six thoracic spinal nerves. The fasciculus cuneatus is situated laterally in the upper thoracic and cervical segments of the spinal cord and is separated from the fasciculus gracilis by a septum. The fasciculus cuneatus contains the long ascending fibers from the upper six thoracic and all the cervical spinal nerves.

Fibers of the fasciculus gracilis and fasciculus cuneatus ascend **ipsilaterally** and terminate by synapsing on the second-order neurons in the **nuclei gracilis** and **cuneatus** of the medulla oblongata. The axons of the second-order neurons, called the **internal arcuate fibers**, sweep anteromedially around the central gray matter and **cross the median plane**, decussating with the corresponding fibers of the opposite side in the **sensory decussation**. The fibers then ascend as a single compact bundle, the **medial lemniscus**, through the medulla oblongata, the pons, and the midbrain. The fibers terminate by synapsing on the third-order neurons in the ventral posterolateral nucleus of the thalamus.

Axons of the third-order neuron leave and pass through the posterior limb of the **internal capsule** and **corona radiata** to reach the somesthetic area in the postcentral gyrus of the cerebral cortex. The contralateral half of the body is represented inverted, with the hand and mouth situated inferiorly, as described previously. (For details, see Chapter 7.) In this manner, the impressions of touch with fine gradations of intensity, exact localization, and two-point discrimination can be appreciated. Vibratory sense and the position of the different parts of the body can be consciously recognized.

Many fibers in the fasciculus cuneatus from the cervical and upper thoracic segments, having terminated on the second-order neuron of the nucleus cuneatus, are relayed and travel as the axons of the second-order neurons to enter the cerebellum through the inferior cerebellar peduncle of the same side. This pathway is the **cuneocerebellar tract**, and the fibers are known as the **posterior external arcuate fibers**. The function of these fibers is to convey information of muscle joint sense to the cerebellum.

Muscle Joint Sense Pathways to the Cerebellum

Muscle joint sense pathways to the cerebellum are summarized in Table 4-3.

Posterior Spinocerebellar Tract

Axons entering the spinal cord from the posterior root ganglion enter the posterior gray column and terminate by synapsing on the second-order neurons at the base of the posterior gray column (Fig. 4-17). These neurons are known collectively as the **nucleus dorsalis (Clarke column)**. Axons of the second-order neurons enter the posterolateral part of the lateral white column on the **same side** and ascend as the posterior spinocerebellar tract to the medulla oblongata. Here, the tract joins the inferior cerebellar peduncle and terminates in the cerebellar cortex. Note that it does not ascend to the cerebral cortex. Because the nucleus dorsalis (Clarke column) extends only from the eighth cervical segment caudally to the third or fourth lumbar segment, axons entering the spinal cord from the posterior roots of the lower lumbar and sacral segments ascend in the posterior white column until they reach the third or fourth lumbar segment, where they enter the nucleus dorsalis.

The posterior spinocerebellar fibers receive muscle joint information from the muscle spindles, tendon organs, and joint receptors of the trunk and lower limbs. This information concerning tension of muscle tendons and the movements of muscles and joints is used by the cerebellum in the coordination of limb movements and the maintenance of posture.

Anterior Spinocerebellar Tract

Axons entering the spinal cord from the posterior root ganglion terminate by synapsing with the second-order neurons in the **nucleus dorsalis** at the base of the posterior gray column (Fig. 4-17). Most axons of the second-order neurons **cross** to the opposite side and ascend as the anterior spinocerebellar tract in the contralateral white column; a minority ascend as the anterior spinocerebellar tract in the lateral white column of the **same side**. The fibers, having ascended through the medulla oblongata and pons, enter the cerebellum through the superior cerebellar peduncle and terminate in the cerebellar cortex. Those fibers that crossed over to the opposite side in the spinal cord are believed to **cross back** within the cerebellum. The anterior spinocerebellar tract conveys muscle joint information from the muscle spindles, tendon organs, and joint receptors of the trunk and the upper and lower limbs. It is also believed that the cerebellum receives information from the skin and superficial fascia by this tract.

Table 4-3 Muscle Joint Sense Pathways to the Cerebellum

Sensation	Receptor	First-Order Neuron	Second-Order Neuron	Pathways	Destination
Unconscious muscle joint sense	Muscle spindles, tendon organs, joint receptors	Posterior root ganglion	Nucleus dorsalis	Anterior and posterior spinocerebellar	Cerebellar cortex

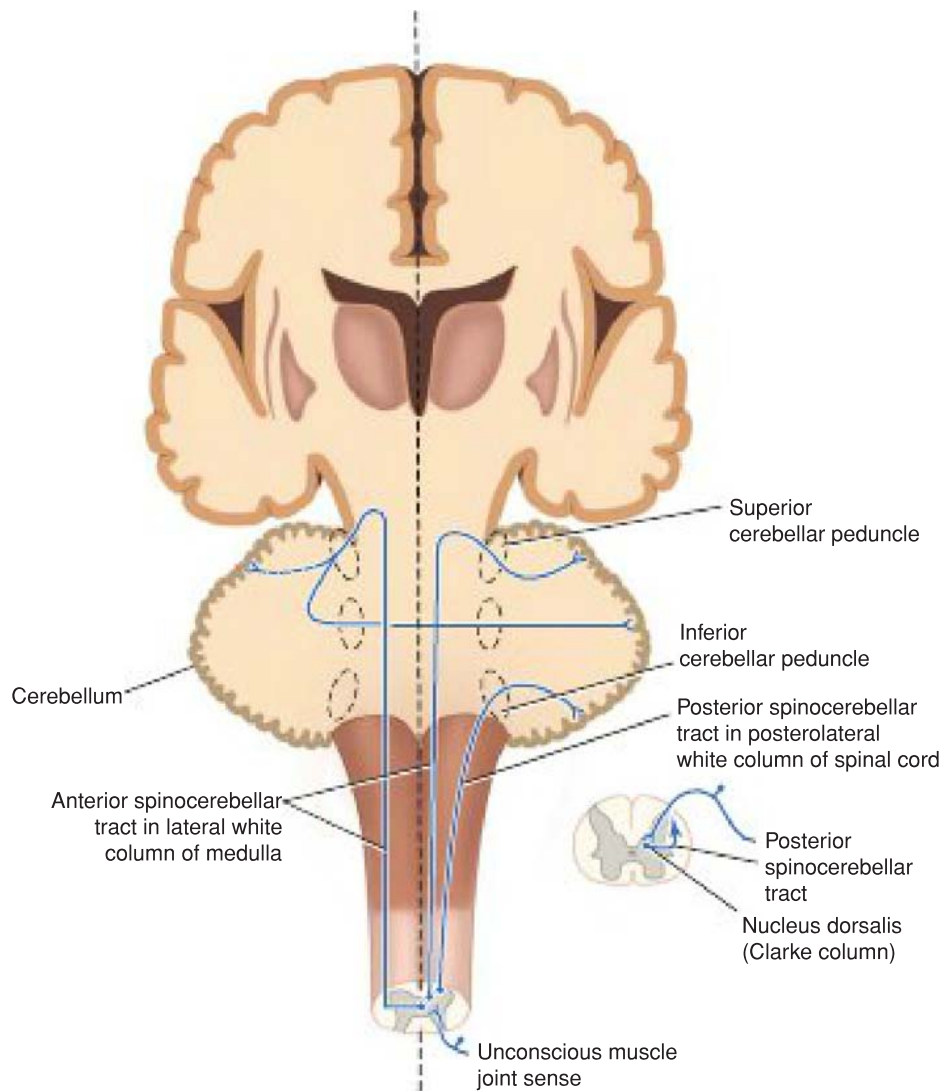


Figure 4-17 Unconscious muscle joint sense pathways to the cerebellum.

Cuneocerebellar Tract

These fibers are described on page 142. They originate in the nucleus cuneatus and enter the cerebellum through the inferior cerebellar peduncle of the **same side** (Fig. 4-16). They are known as the **posterior external arcuate fibers**, and their function is to convey information of muscle joint sense to the cerebellum.

Other Ascending Pathways

The spinotectal, spinoreticular, and spino-olivary tracts are shown in Figure 4-18.

Spinotectal Tract

Axons enter the spinal cord from the posterior root ganglion and travel to the gray matter where they synapse on unknown second-order neurons. Axons of the second-order neurons **cross the median plane**

and ascend as the spinotectal tract in the anterolateral white column lying close to the lateral spinothalamic tract. After passing through the medulla oblongata and pons, they terminate by synapsing with neurons in the superior colliculus of the midbrain. This pathway provides afferent information for spinovisual reflexes and brings about movements of the eyes and head toward the source of the stimulation.

Spinoreticular Tract

Axons enter the spinal cord from the posterior root ganglion and terminate on unknown second-order neurons in the gray matter. Axons from these second-order neurons ascend the spinal cord as the spinoreticular tract in the lateral white column mixed with the lateral spinothalamic tract. Most of the fibers are **uncrossed** and terminate by synapsing with neurons of the reticular formation in the medulla oblongata, pons, and midbrain. The spinoreticular tract provides an afferent

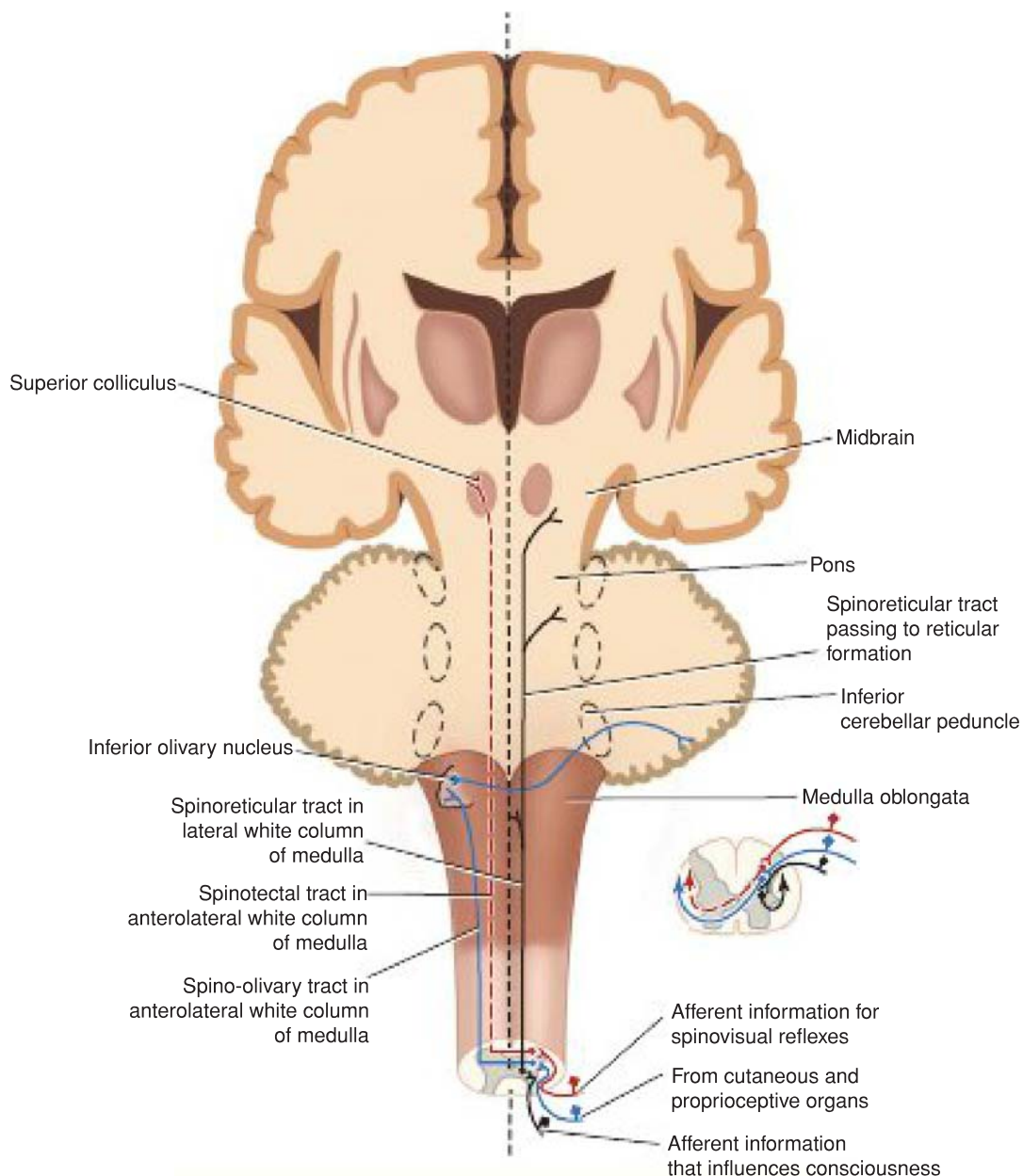


Figure 4-18 Spinotectal, spinoreticular, and spino-olivary tracts.

pathway for the reticular formation, which plays an important role in influencing levels of consciousness. (For details, see p. 204.)

Spino-olivary Tract

Axons enter the spinal cord from the posterior root ganglion and terminate on unknown second-order neurons in the posterior gray column. Axons from the second-order neurons **cross the midline** and ascend as the spino-olivary tract in the white matter at the junction of the anterior and lateral columns. The axons end by synapsing on third-order neurons in the inferior olivary nuclei in the medulla oblongata. Axons of the third-order neurons cross the midline and

enter the cerebellum through the inferior cerebellar peduncle. The spino-olivary tract conveys information to the cerebellum from cutaneous and proprioceptive organs.

Visceral Sensory Tracts

Sensations that arise in viscera located in the thorax and abdomen enter the spinal cord through the posterior roots. The cell bodies of the first-order neuron are situated in the posterior root ganglia. The peripheral processes of these cells receive nerve impulses from pain and stretch receptor endings in the viscera. The causes of visceral pain include ischemia, chemical damage, spasm of smooth muscle,

and distention. The central processes, having entered the spinal cord, synapse with second-order neurons in the gray matter, probably in the posterior or lateral gray columns.

Axons of the second-order neurons are believed to join the spinothalamic tracts and ascend and terminate on third-order neurons in the ventral posterolateral nucleus of the thalamus. The final destination of these axons is probably in the postcentral gyrus of the cerebral cortex.

Many of the visceral afferent fibers that enter the spinal cord branch participate in reflex activity.

DESCENDING TRACTS

Motor neurons situated in the anterior gray columns of the spinal cord send axons to innervate skeletal muscle through the anterior roots of the spinal nerves. These motor neurons are referred to as the **lower motor neurons** and constitute the final common pathway to the muscles (Fig. 4-19).

Lower motor neurons are constantly bombarded by nervous impulses that descend from the medulla, pons, midbrain, and cerebral cortex as well as those that enter along sensory fibers from the posterior

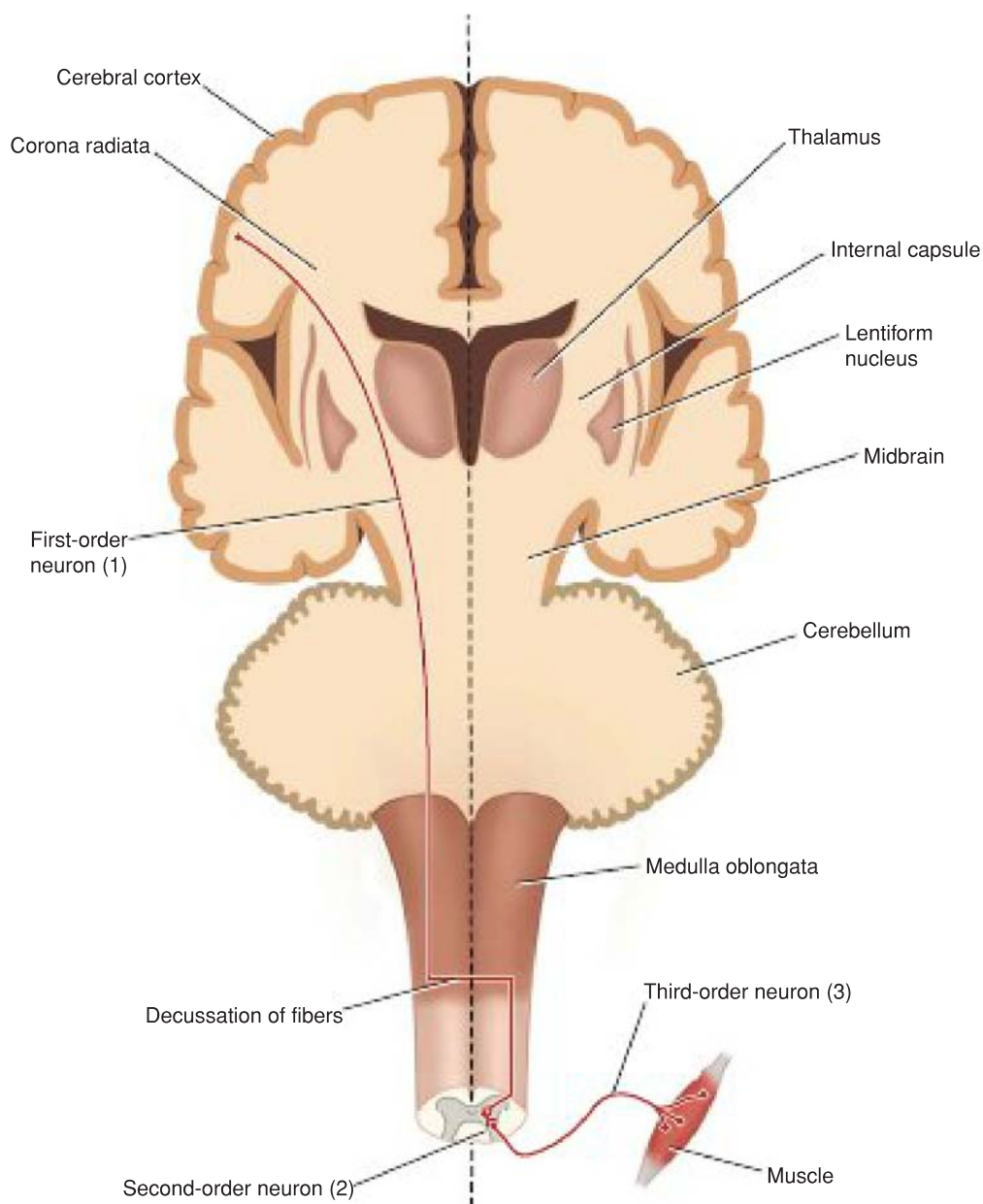


Figure 4-19 Simple form of the descending motor pathway from the cerebral cortex to the skeletal muscle. Note the three neurons involved.

Table 4-4 Main Descending Pathways to the Spinal Cord^a

Pathway	Function	Origin	Site of Crossover	Destination	Branches to
Corticospinal tracts	Rapid, skilled, voluntary movements, especially distal ends of limbs	Primary motor cortex (area 4), secondary motor cortex (area 6), parietal lobe (areas 3, 1, and 2)	Most cross at decussation of pyramids and descend as lateral corticospinal tracts; some continue as anterior corticospinal tracts and cross over at level of destination	Internuncial neurons or α motor neurons	Cerebral cortex, basal nuclei, red nucleus, olivary nuclei, reticular formation
Reticulospinal tracts	Inhibit or facilitate voluntary movement; hypothalamus controls sympathetic, para-sympathetic outflows	Reticular formation	Some cross at various levels	α and γ motor neurons	Multiple branches as they descend
Tectospinal tract	Reflex postural movements concerning sight	Superior colliculus	Soon after origin	α and γ motor neurons	?
Rubrospinal tract	Facilitates activity of flexor muscles and inhibits activity of extensor muscles	Red nucleus	Immediately	α and γ motor neurons	?
Vestibulospinal tract	Facilitates activity of extensor inhibits flexor muscles	Vestibular nuclei	Uncrossed	α and γ motor neurons	?
Olivospinal tract	??	Inferior olivary nuclei	Cross in brainstem	? α and γ motor neurons	—
Descending autonomic fibers	Control sympathetic and parasympathetic systems	Cerebral cortex, hypothalamus, amygdaloid complex, reticular formation		Sympathetic and parasympathetic outflows	—

^aNote that the corticospinal tracts are believed to control the prime mover muscles (especially the highly skilled movements), whereas the other descending tracts are important in controlling the simple basic movements. For simplicity, the internuncial neurons are omitted from this table.

roots. Nerve fibers that descend in the white matter from different supraspinal nerve centers are segregated into nerve bundles called the **descending tracts**. These supraspinal neurons and their tracts are referred to as the **upper motor neurons**, and they provide numerous separate pathways that can influence motor activity.

A summary of the main descending pathways in the spinal cord is shown in Table 4-4.

Anatomical Organization

Control of skeletal muscle activity from the cerebral cortex and other higher centers is conducted through the nervous system by a series of neurons (Fig. 4-19). The descending pathway from the cerebral cortex is often made up of three neurons. The first neuron, the **first-order neuron**, has its cell body in the cerebral cortex. Its axon descends to synapse

on the **second-order neuron**, an internuncial neuron, situated in the anterior gray column of the spinal cord. The axon of the second-order neuron is short and synapses with the **third-order neuron**, the lower motor neuron, in the anterior gray column. The axon of the third-order neuron innervates the skeletal muscle through the anterior root and spinal nerve. In some instances, the axon of the first-order neuron terminates directly on the third-order neuron (as in reflex arcs).

Functions

Corticospinal tracts (Fig. 4-20) are the pathways concerned with voluntary, discrete, skilled movements, especially those of the distal parts of the limbs. **Reticulospinal tracts** may facilitate or inhibit the activity of the α and γ motor neurons in the anterior gray columns and may, therefore, facilitate or inhibit voluntary

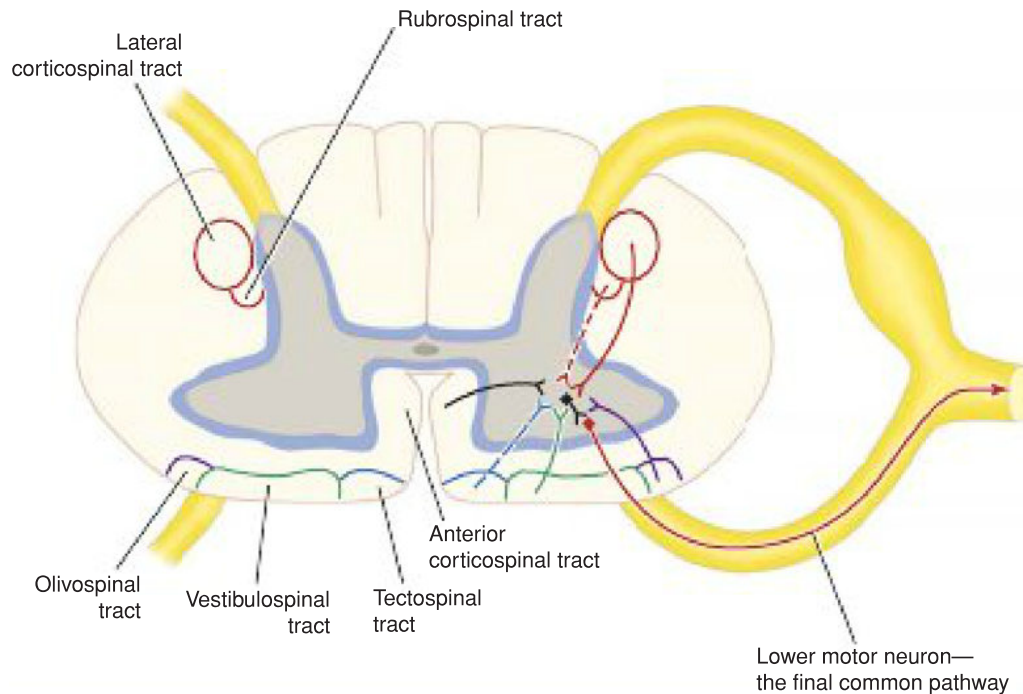


Figure 4-20 Transverse section of the spinal cord showing the termination of the descending motor tracts. Note that the existence of the olivospinal tract as a separate pathway is now in considerably doubt.

movement or reflex activity. The **tectospinal tract** is concerned with reflex postural movements in response to visual stimuli. Those fibers that are associated with the sympathetic neurons in the lateral gray column are concerned with the pupillodilation reflex in response to darkness. The **rubrospinal tract** acts on both the α and γ motor neurons in the anterior gray columns and facilitates the activity of flexor muscles and inhibits the activity of extensor or antigravity muscles. The **vestibulospinal tract**, by acting on the motor neurons in the anterior gray columns, facilitates the activity of the extensor muscles, inhibits the activity of the flexor muscles, and is concerned with the postural activity associated with balance. The **olivospinal tract** may play a role in muscular activity, but some doubt its existence. The **descending autonomic fibers** are concerned with the control of visceral activity.

Corticospinal Tracts

Fibers of the corticospinal tract arise as axons of pyramidal cells situated in the fifth layer of the cerebral cortex (Fig. 4-21). About a third of the fibers originate from the primary motor cortex (area 4), another third from the secondary motor cortex (area 6), and the final third from the parietal lobe (areas 3, 1, and 2); thus, two thirds of the fibers arise from the precentral gyrus, and one third of the fibers arise from the postcentral gyrus. These fibers do not control motor activity but influence sensory input to the nervous system. Because electrical stimulation of different parts of the precentral gyrus produces movements of different parts of the opposite

side of the body, we can represent the parts of the body in this area of the cortex. Such a homunculus is shown in Figure 4-21. Note that the region controlling the face is situated inferiorly, and the region controlling the lower limb is situated superiorly and on the medial surface of the hemisphere. The homunculus is a distorted picture of the body, with the various parts having a size proportional to the area of the cerebral cortex devoted to their control. Interestingly, most corticospinal fibers are myelinated and are relatively slow-conducting, small fibers.

The descending fibers converge in the **corona radiata** and then pass through the posterior limb of the **internal capsule**. Here, the fibers are organized so that those closest to the genu are concerned with cervical portions of the body, while those situated more posteriorly are concerned with the lower extremity. The tract then continues through the middle three fifths of the **basis pedunculi of the midbrain**. Here, the fibers concerned with cervical portions of the body are situated medially, while those concerned with the leg are placed laterally.

On entering the pons, the tract is broken into many bundles by the **transverse pontocerebellar fibers** (see Figs. 5-19 to 5-22). In the medulla oblongata, the bundles become grouped together along the anterior border to form a swelling known as the **pyramid** (hence the alternative name, **pyramidal tract**) (see Figs. 5-11 and 5-12). At the junction of the medulla oblongata and the spinal cord, most of the fibers **cross** the midline at the **decussation of the pyramids** (Fig. 4-21) and enter the lateral white column of the

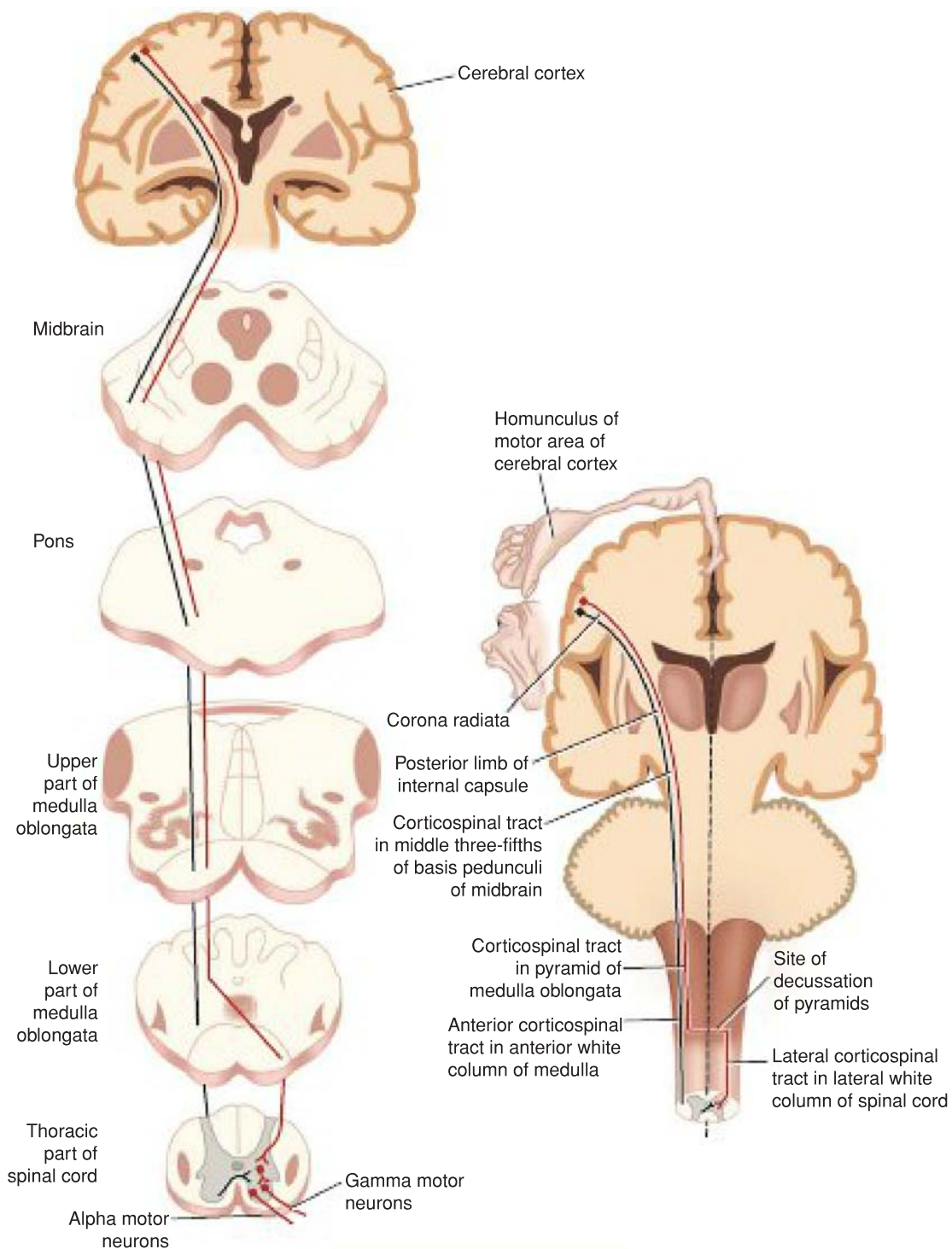


Figure 4-21 Corticospinal tracts.

spinal cord to form the **lateral corticospinal tract** (Fig. 4-20). The remaining fibers do not cross in the decussation but descend in the anterior white column of the spinal cord as the **anterior corticospinal tract** (Figs. 4-20 and 4-21). These fibers eventually **cross** the midline and terminate in the anterior gray column of the spinal cord segments in the cervical and upper thoracic regions.

The lateral corticospinal tract descends the length of the spinal cord; its fibers terminate in the anterior gray column of all the spinal cord segments.

Most corticospinal fibers synapse with internuncial neurons, which, in turn, synapse with α motor neurons and some γ motor neurons. Only the largest corticospinal fibers synapse directly with the motor neurons.

The corticospinal tracts are not the sole pathway for serving voluntary movement. Rather, they form the pathway that confers speed and agility to voluntary movements and is thus used in performing rapid skilled movements. Many of the simple, basic voluntary movements are mediated by other descending tracts.

Branches

1. Branches are given off early in their descent and return to the cerebral cortex to inhibit activity in adjacent regions of the cortex.
2. Branches pass to the caudate and lentiform nuclei, the red nuclei, and the olivary nuclei and the reticular formation. These branches keep the subcortical

regions informed about the cortical motor activity. Once alerted, the subcortical regions may react and send their own nervous impulses to the α and γ motor neurons by other descending pathways.

Reticulospinal Tracts

Throughout the midbrain, pons, and medulla oblongata, groups of scattered nerve cells and nerve fibers exist that are collectively known as the **reticular formation**. From the pons, these neurons send axons, which are mostly **uncrossed**, down into the spinal cord and form the **pontine reticulospinal tract** (Fig. 4-22). From the medulla, similar neurons send axons, which are crossed and uncrossed, to the spinal cord and form the **medullary reticulospinal tract**.

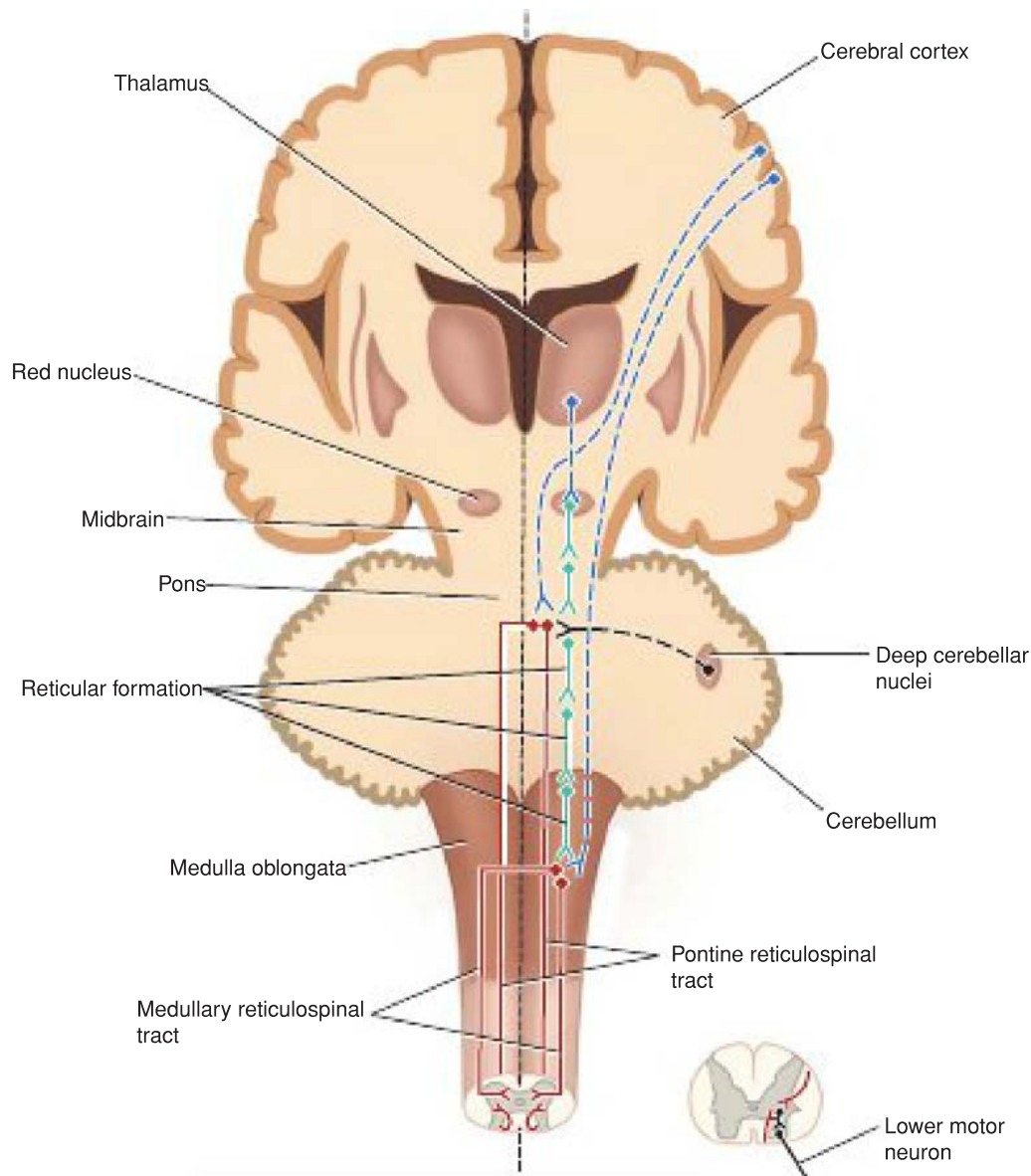


Figure 4-22 Reticulospinal tracts.

Reticulospinal fibers from the pons descend through the anterior white column, while those from the medulla oblongata descend in the lateral white column. Both sets of fibers enter the anterior gray columns of the spinal cord and may facilitate or inhibit α and γ motor neuron activity. By these means, the reticulospinal tracts influence voluntary movements and reflex activity. The reticulospinal fibers are also now thought to include the descending autonomic fibers. The reticulospinal tracts thus provide a pathway by which the hypothalamus can control the sympathetic outflow and the sacral parasympathetic outflow.

Tectospinal Tract

Fibers of this tract arise from nerve cells in the **superior colliculus** of the midbrain (Fig. 4-23). Most

of the fibers **cross** the midline soon after their origin and descend through the brainstem close to the **medial longitudinal fasciculus**. The tectospinal tract descends through the anterior white column of the spinal cord close to the anterior median fissure (Fig. 4-20). The majority of the fibers terminate in the anterior gray column in the upper cervical segments of the spinal cord by synapsing with internuncial neurons. These fibers are believed to be concerned with reflex postural movements in response to visual stimuli.

Rubrospinal Tract

The **red nucleus** is situated in the tegmentum of the midbrain at the level of the superior colliculus (Fig. 4-24). The axons of neurons in this nucleus cross

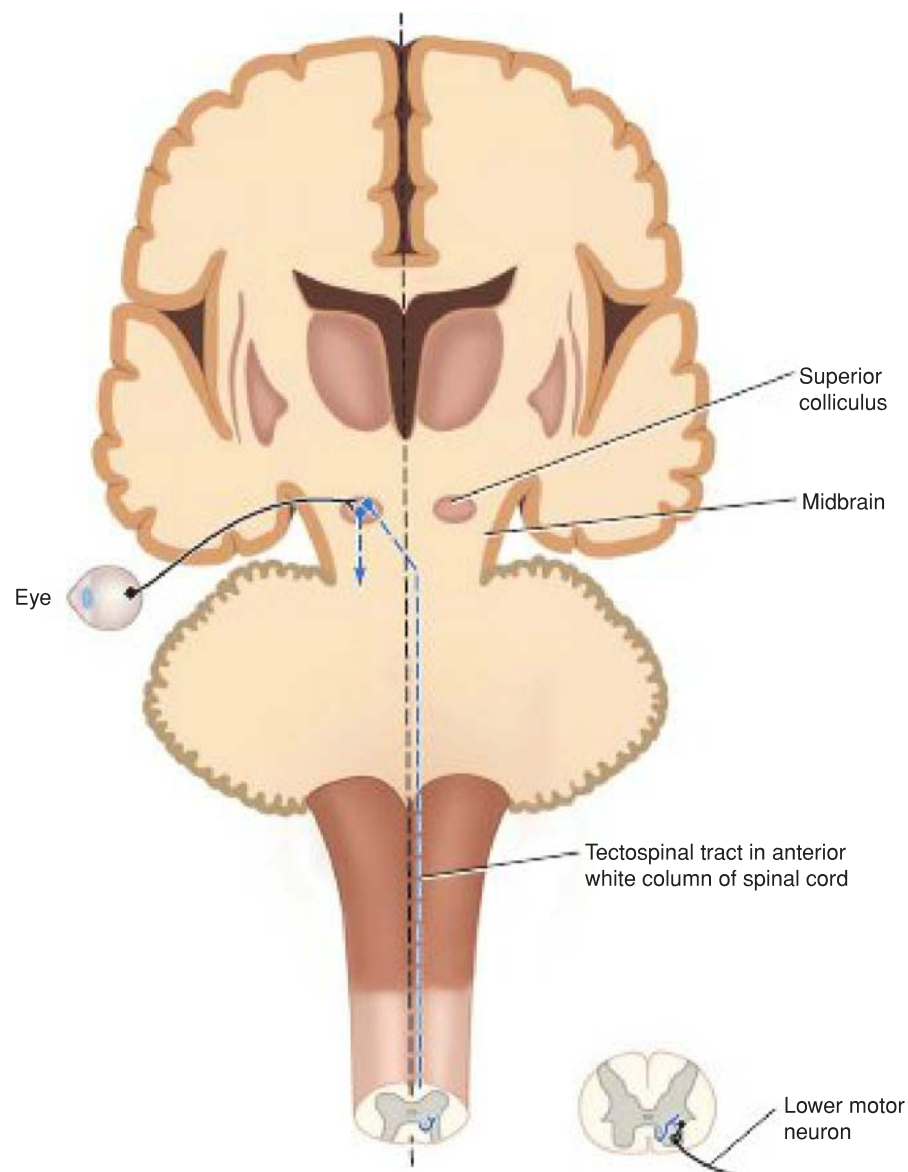


Figure 4-23 Tectospinal tract.

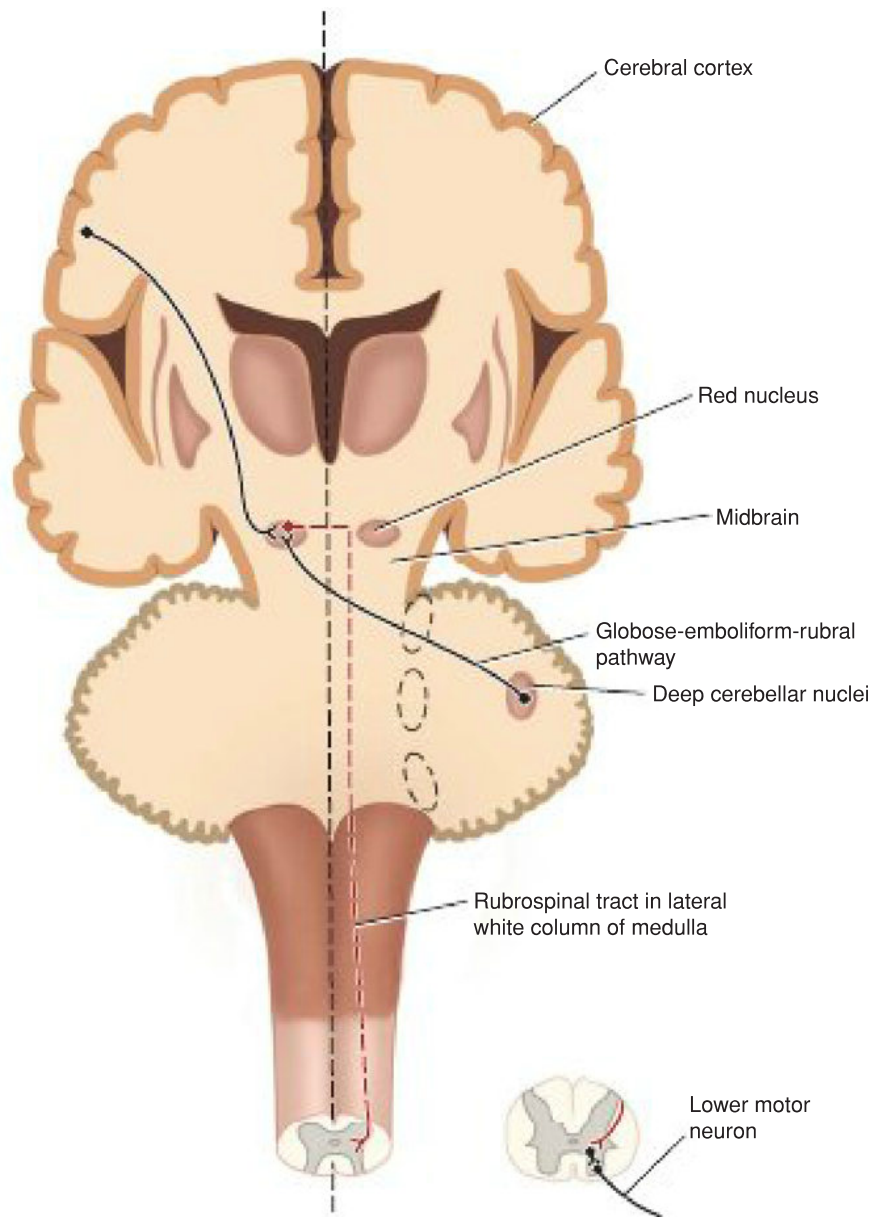


Figure 4-24 Rubrospinal tract.

the midline at the level of the nucleus and descend as the rubrospinal tract through the pons and medulla oblongata to enter the lateral white column of the spinal cord (Fig. 4-20). The fibers terminate by synapsing with internuncial neurons in the anterior gray column of the cord.

Neurons of the red nucleus receive afferent impulses through connections with the cerebral cortex and the cerebellum. This is believed to be an important indirect pathway by which the cerebral cortex and the cerebellum can influence α and γ motor neuron activity. The tract facilitates the activity of the flexor muscles and inhibits the activity of the extensor or antigravity muscles.

Vestibulospinal Tract

Vestibular nuclei are situated in the pons and medulla oblongata beneath the floor of the fourth ventricle (Fig. 4-25). The vestibular nuclei receive afferent fibers from the inner ear through the vestibular nerve and from the cerebellum. Neurons of the lateral vestibular nucleus give rise to the axons that form the vestibulospinal tract. The tract descends **uncrossed** through the medulla and through the length of the spinal cord in the anterior white column (Fig. 4-20). The fibers terminate by synapsing with internuncial neurons of the anterior gray column of the spinal cord.

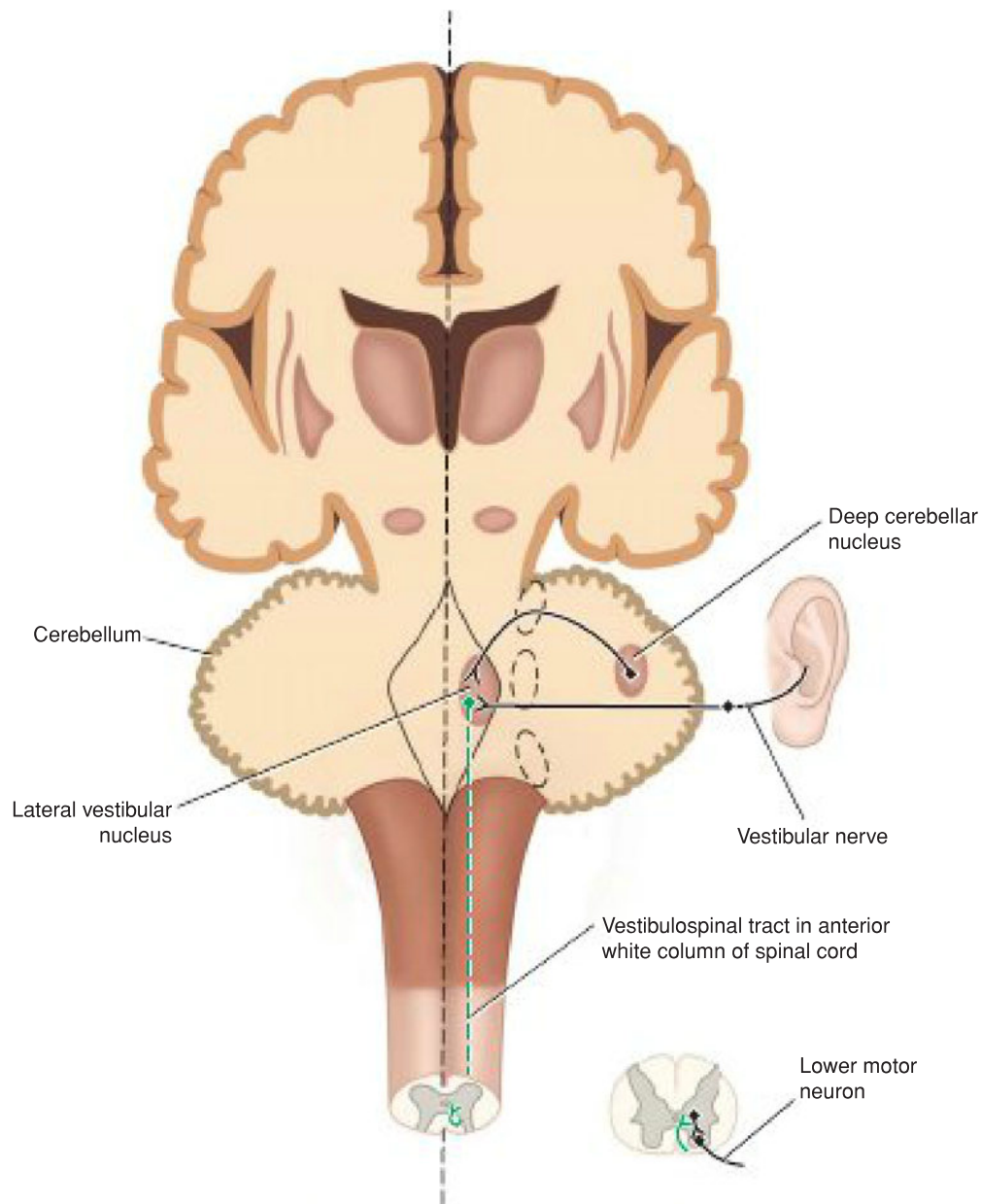


Figure 4-25 Vestibulospinal tract.

The inner ear and the cerebellum, by means of this tract, facilitate the activity of the extensor muscles and inhibit the activity of the flexor muscles in association with the maintenance of balance.

Olivospinal Tract

The olivospinal tract was thought to arise from the inferior olivary nucleus and to descend in the lateral white column of the spinal cord (Fig. 4-26), to influence the activity of the motor neurons in the anterior gray column. Many now doubt that it exists.

Descending Autonomic Fibers

The higher centers of the CNS associated with the control of autonomic activity are situated in the cerebral cortex, hypothalamus, amygdaloid complex, and reticular formation. Although distinct tracts have not been recognized, investigation of spinal cord lesions has demonstrated that descending autonomic tracts do exist and probably form part of the reticulospinal tract.

The fibers arise from neurons in the higher centers and cross the midline in the brainstem. They are

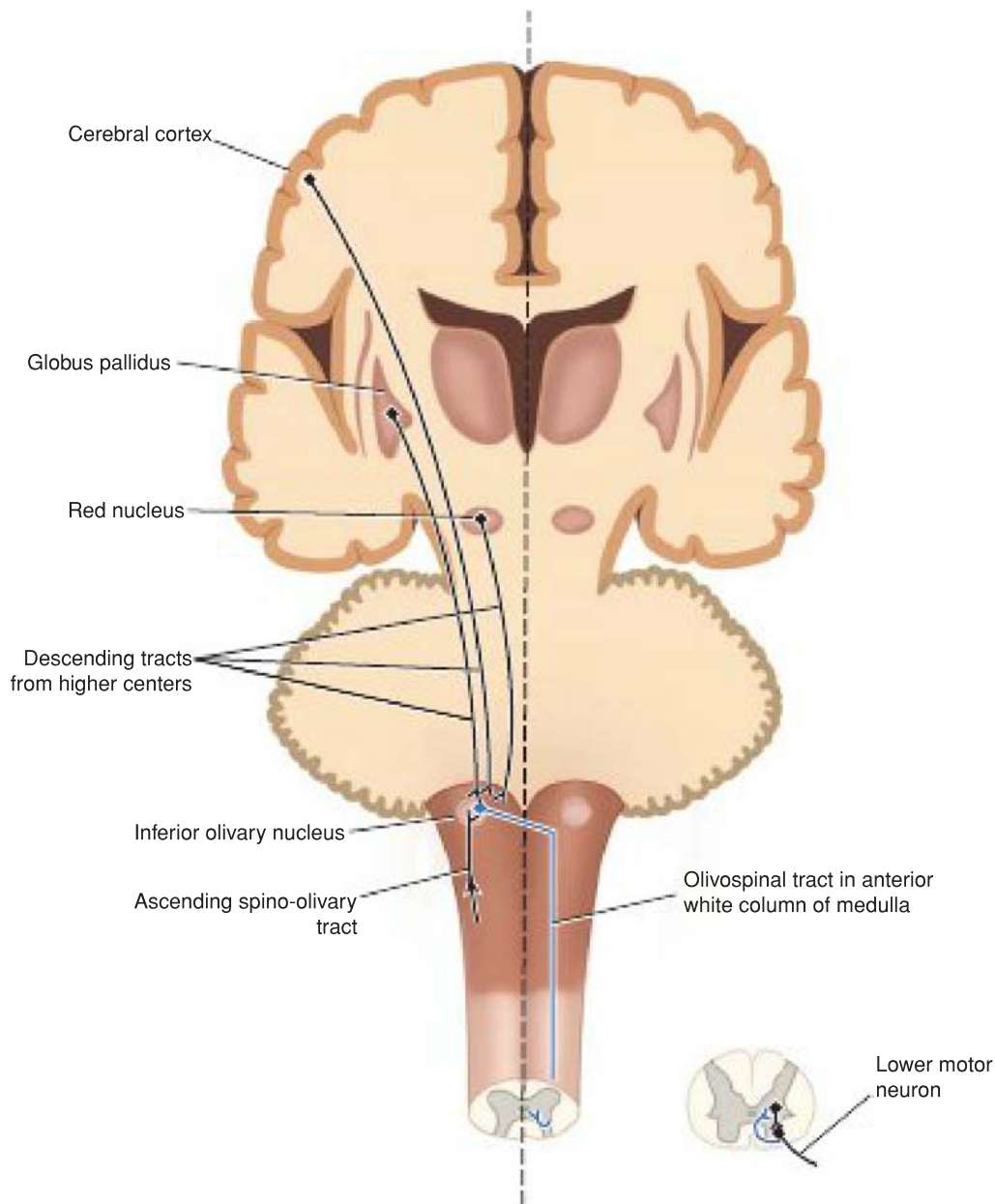


Figure 4-26 Olivospinal tract. The existence of this tract as a separate pathway is doubtful.

believed to descend in the lateral white column of the spinal cord and to terminate by synapsing on the autonomic motor cells in the lateral gray columns in the thoracic and upper lumbar (sympathetic outflow) and midsacral (parasympathetic) levels of the spinal cord.

INTERSEGMENTAL TRACTS

Short ascending and descending tracts that originate and end within the spinal cord exist in the anterior, lateral, and posterior white columns. The function of these pathways is to interconnect the neurons of different segmental levels, and the pathways are particularly important in intersegmental spinal reflexes.

Reflex Arc

A **reflex** may be defined as an involuntary response to a stimulus. It depends on the integrity of the reflex arc (Fig. 4-27). In its simplest form, a reflex arc consists of the following anatomical structures: (1) a receptor organ, (2) an afferent neuron, (3) an effector neuron, and (4) an effector organ. A reflex arc involving only one synapse is referred to as a **monosynaptic reflex arc**. Interruption of the reflex arc at any point along its course would abolish the response.

In the spinal cord, reflex arcs play an important role in maintaining muscle tone, which is the basis for body posture. The receptor organ is situated in the skin, muscle, or tendon. The cell body of the afferent neuron is

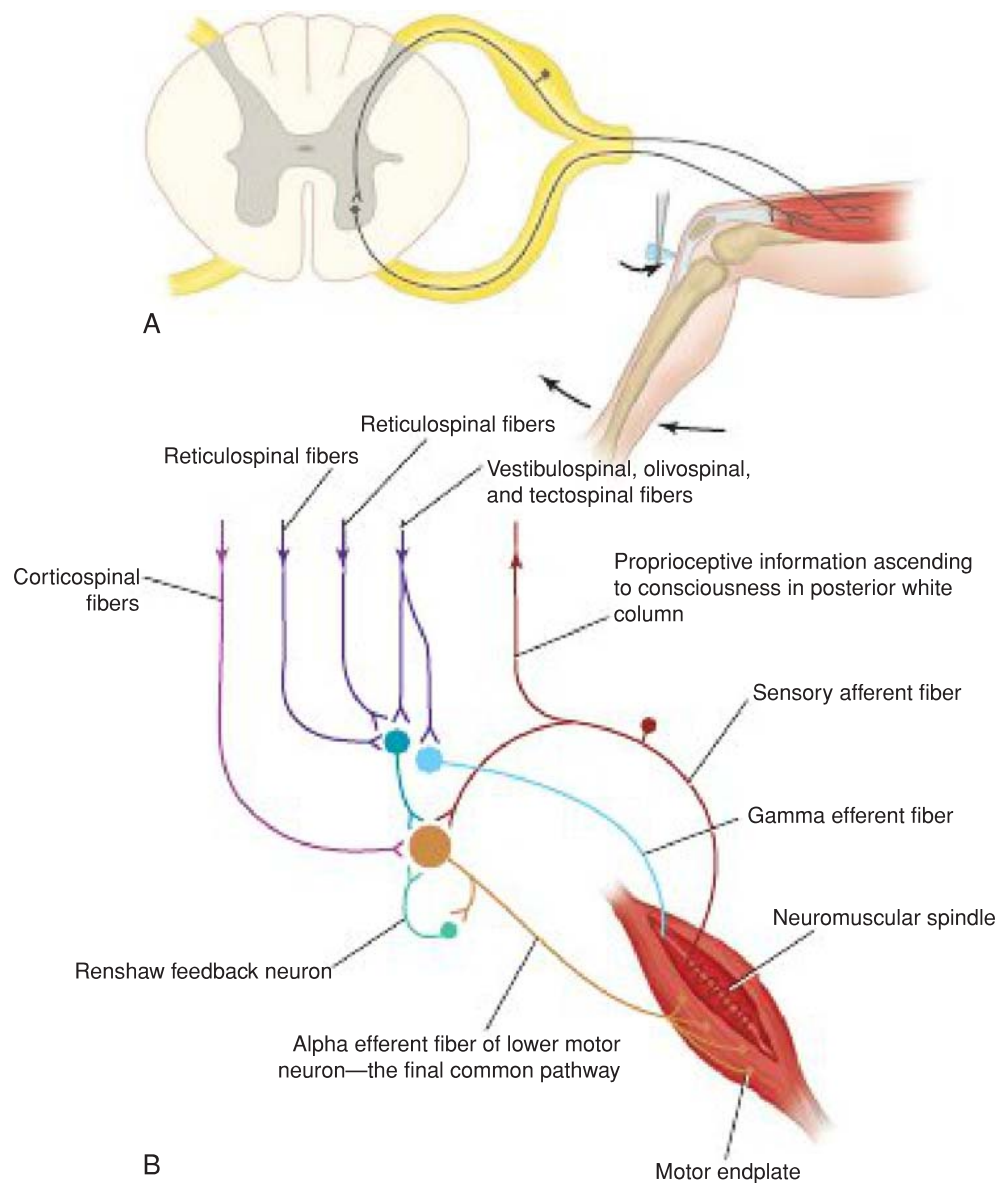


Figure 4-27 **A:** A monosynaptic reflex arc. **B:** Multiple neurons synapsing with the lower motor neuron. Note the presence of the Renshaw feedback neuron.

located in the posterior root ganglion, and the central axon of this first-order neuron terminates by synapsing on the effector neuron. Since the afferent fibers are of large diameter and are rapidly conducting and because of the presence of only one synapse, a very quick response is possible.

Physiologic study of the electrical activity of the effector neuron shows that a prolonged asynchronous discharge follows the very quick monosynaptic discharge. The reason for this later discharge is that the afferent fibers entering the spinal cord frequently branch, and the branches synapse with many internuncial neurons, which ultimately synapse with the effector neuron (Fig. 4-28). These additional neuronal circuits prolong the bombardment of the effector neurons after the initial stimulation by the afferent neuron has ceased. The presence of internuncial neurons also results in the

spread of the afferent stimulus to neurons at different segmental levels of the spinal cord.

The **law of reciprocal innervation** is important to understand in considering reflex skeletal muscle activity. Simply stated, it means that the flexor and extensor reflexes of the same limb cannot be made to contract simultaneously. For this law to work, the afferent nerve fibers responsible for flexor reflex muscle action must have branches that synapse with the extensor motor neurons of the same limb, causing them to be inhibited.

Another interesting property of spinal reflexes is that the evocation of a reflex on one side of the body causes opposite effects on the limb of the other side of the body. This **crossed extensor reflex** is demonstrated when afferent stimulation of the reflex arc that causes the ipsilateral limb to flex results in the contralateral limb being extended.

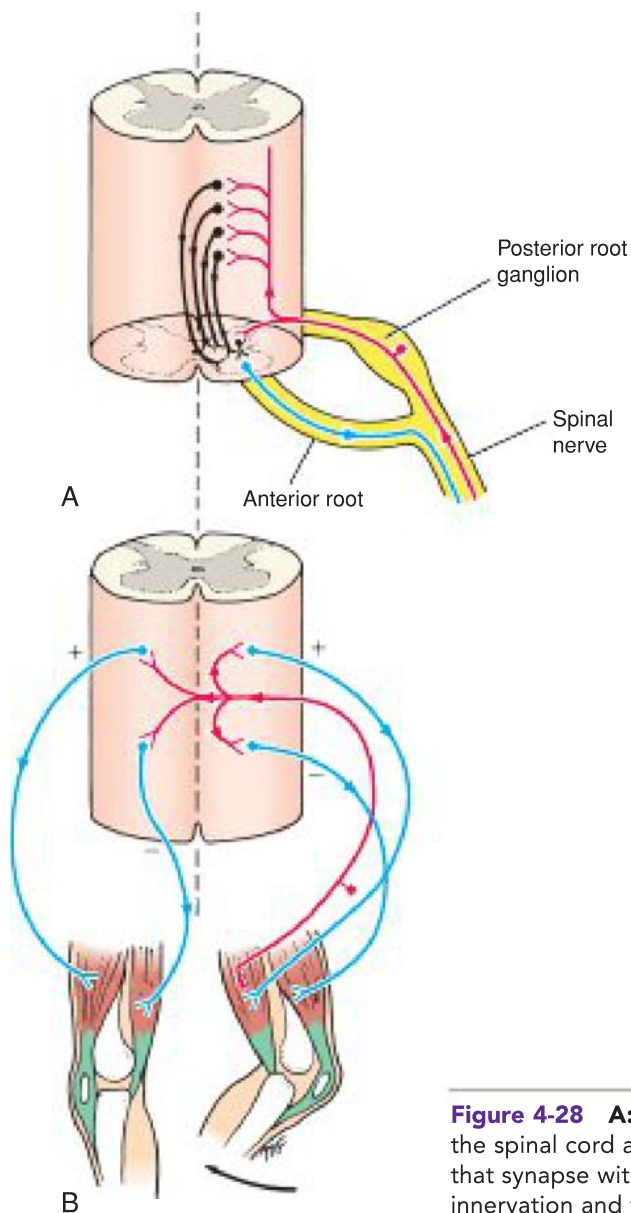


Figure 4-28 **A:** Multiple branching of afferent fibers entering the spinal cord and the presence of many internuncial neurons that synapse with the effector neuron. **B:** Law of reciprocal innervation and the crossed extensor reflex.

Influence of Higher Neuronal Centers on Spinal Reflex Activity

The spinal segmental reflex arc involving motor activity is greatly influenced by higher centers in the brain. These influences are mediated through the cortico-spinal, reticulospinal, tectospinal, rubrospinal, and vestibulospinal tracts. In the clinical condition known as spinal shock (see p. 167), which follows the sudden removal of these influences by spinal cord severance, the segmental spinal reflexes are depressed. When the spinal shock disappears in a few weeks, the segmental spinal reflexes return, and the muscle tone is increased. This **decerebrate rigidity** is due to the overactivity of the γ efferent nerve fibers to the muscle spindles, which results from the release of these neurons from the higher centers. The next stage may be **paraplegia in extension** with domination of the increased tone of the extensor muscles over the flexor

muscles. Some neurologists believe that this condition is due to incomplete severance of all the descending tracts with persistence of the vestibulospinal tract. Should all the descending tracts be severed, the condition of **paraplegia in flexion** occurs. In this condition, the reflex responses are flexor in nature, and the tone of the extensor muscles is diminished.

RENSHAW CELLS AND LOWER MOTOR NEURON INHIBITION

Lower motor neuron axons give off collateral branches as they pass through the white matter to reach the anterior roots of the spinal nerve. These collaterals synapse on neurons described by Renshaw, which, in turn, synapse on the lower motor neurons (Fig. 4-27). These internuncial neurons are believed to provide feedback on the lower motor neurons, inhibiting their activity.

Clinical Notes

General Anatomical Features of Clinical Importance

The spinal cord may be described, for practical purposes, as consisting of columns of motor and sensory nerve cells (gray matter) surrounded by ascending and descending tracts (white matter). It lies within the vertebral canal and is protected by three surrounding fibrous membranes, the meninges. It is cushioned against trauma by cerebrospinal fluid (CSF) and is held in position by the denticulate ligaments on each side and the filum terminale inferiorly. The spinal cord is segmented, and paired posterior (sensory) and anterior (motor) roots corresponding to each segment of the cord leave the vertebral canal through the intervertebral foramina.

The spinal cord is shorter than the vertebral column and terminates inferiorly in the adult at the level of the lower border of the first lumbar vertebra. The subarachnoid space extends inferiorly beyond the end of the cord and ends at the level of the lower border of the second sacral vertebra.

Because of the shortness of the spinal cord relative to the length of the vertebral column, the nerve roots of the lumbar and sacral segments have to take an oblique course downward to reach their respective intervertebral foramina; the resulting leash of nerve roots forms the cauda equina.

A spinal tap needle may be inserted into the subarachnoid space below the level of the second lumbar vertebra without damaging the spinal cord. (For details, see p. 19.)

Anterior and Posterior Nerve Root Lesions

Each nerve root has a covering of pia, arachnoid, and dura mater. The anterior and posterior roots unite in the intervertebral foramina to form the spinal nerves. Here, the meninges fuse with the epineurium of the spinal nerves. Either or both spinal nerve roots may be involved in syphilitic spinal meningitis or pyogenic meningitis. The posterior roots may be involved in tabes dorsalis and herpes zoster. Their anatomical location, both in the vertebral canal and in the intervertebral foramina, exposes them to compression from tumors of the vertebral column and to irritation from abnormal constituents of CSF, such as blood following a subarachnoid hemorrhage. A herniated intervertebral disc, a primary or secondary vertebral tumor, vertebral destruction by tumor or infection, or a fracture dislocation can press on the spinal nerve roots in the intervertebral foramina. Even severe scoliosis can compress the nerve roots.

A lesion of one posterior spinal nerve root will produce pain in the area of skin innervated by that root and in the muscles that receive their sensory nerve supply from that root. Movements of the vertebral column in the region of the lesion will heighten the pain, and coughing and sneezing will also make it worse by raising the pressure within the vertebral canal. Before loss of sensation in the dermatome occurs, the patient might experience hyperalgesia and hyperesthesia.

A lesion of an anterior root will result in paralysis of any muscle that is supplied exclusively by that root and a partial paralysis of any muscle that is supplied partially by that root. In both cases, fasciculation and muscle atrophy occur.

Clinical Significance of Ascending Tract Lamination

Within the anterolateral white column of the spinal cord, the axons of the spinothalamic tracts from the sacral and lumbar segments of the body are deflected laterally by axons crossing the midline at successively higher levels. Within the posterior white column, the axons from the sacral and lumbar segments of the body are pushed medially by the axons from higher segments of the body. This deflection of the tracts produces lamination; thus, in the spinothalamic tracts (anterolateral system), the cervical to sacral segments are located from medial to lateral, whereas in the posterior white column (medial lemniscus system), the sacral to cervical segments are located from medial to lateral. This is shown diagrammatically in Figure 4-29.

Ascending Tract Injury

The detailed information described above is of practical value in patients experiencing external pressure exerted on the spinal cord in the region of the spinothalamic tracts. It explains, for example, why patients will experience a loss of pain and temperature sensations first in the sacral dermatomes of the body and, if the pressure increases, in the other higher segmental dermatomes of the body.

Lateral Spinothalamic Tract

Destruction of this tract produces contralateral loss of pain and thermal sensibilities below the level of the lesion. Therefore, the patient will not respond to pinprick or recognize hot and cold objects placed in contact with the skin.

Anterior Spinothalamic Tract

Destruction of this tract produces contralateral loss of light touch and pressure sensibilities below the level of the lesion. Remember that discriminative touch will still be present, because this information is conducted through the fasciculus gracilis and fasciculus cuneatus. The patient will not feel the light touch of a piece of cotton placed against the skin or feel pressure from a blunt object placed against the skin.

Fasciculus Gracilis and Fasciculus Cuneatus

Destruction of these tracts cuts off the supply of information from the muscles and joints to consciousness; thus, the individual does not know about the position and movements of the ipsilateral limbs below the level of the lesion. With the patient's eyes closed, he or she is unable to tell where the limb or part of the limb is in space. For example, if you passively dorsiflex the patient's big toe, he or she is unable to tell you whether the toe is pointing upward or downward. The patient has impaired muscular control, and the movements are jerky or ataxic.

The patient also has loss of vibration sense below the level of the lesion on the same side. This is easily tested by applying a vibrating tuning fork to a bony prominence, such as the lateral malleolus of the fibula or the styloid process of the radius.

Tactile discrimination will also be lost on the side of the lesion. This is tested most easily by gradually separating the two points of a compass until the patient can appreciate them as two separate points, not as one, when they are applied to the skin surface. Tactile discrimination

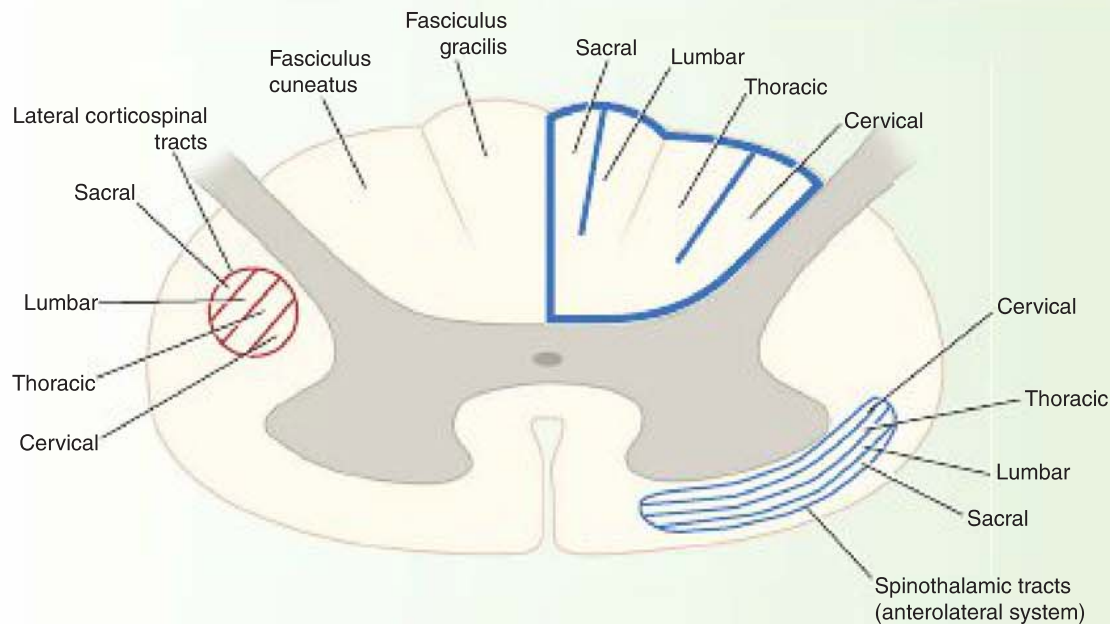


Figure 4-29 Segmental organization of the tracts in the posterior, lateral, and anterior white columns of the spinal cord.

varies from one part of the body to another. In a normal individual, the points have to be separated by about 3 to 4 mm before they are recognized as separate points on the tips of the fingers. On the back, however, the points have to be separated by 65 mm or more before they can be recognized as separate points.

The sense of general light touch would be unaffected, as these impulses ascend in the anterior spinothalamic tracts.

Note that a spinal cord lesion so localized that it affects one sensory tract only is extremely rare; usually, several ascending and descending tracts are involved.

Somatic and Visceral Pain

Somatic pain has been considered extensively in this chapter. The sense organs for somatic pain are the naked nerve endings. The initial sharp pain is transmitted by fast-conducting fibers, and the more prolonged burning pain travels in the slow-conducting nerve fibers (see p. 143).

In the viscera, special receptors, chemoreceptors, baroreceptors, osmoreceptors, and stretch receptors are sensitive to a variety of stimuli, including ischemia, stretching, and chemical damage. Afferent fibers from the visceral receptors reach the central nervous system (CNS) via the sympathetic and parasympathetic parts of the autonomic nervous system. Once within the CNS, the pain impulses travel by the same ascending tracts as the somatic pain and ultimately reach the postcentral gyrus.

Visceral pain is poorly localized and often associated with salivation, nausea, vomiting, tachycardia, and sweating. Visceral pain may be referred from the organ involved to a distant area of the body (**referred pain**).

Treatment of Acute Pain

Drugs such as salicylates can be used to reduce the synthesis of prostaglandin, a substance that sensitizes free nerve endings to painful stimuli. Local anesthetics, such as procaine, can be used to block nerve conduction in peripheral nerves.

Narcotic analgesics, such as morphine and codeine, reduce the affective reaction to pain and act on the opiate receptor sites in the cells in the posterior gray column of the spinal cord as well as other cells in the analgesic system in the brain likely by inhibiting the release of glutamate, substance P, and other transmitters from the sensory nerve endings. To minimize the side effects of morphine given by systemic injection, the narcotic can be given by local injection directly into the posterior gray horn of the spinal cord or by injection indirectly into the CSF in the subarachnoid space. Long-term cancer pain has been treated successfully by the continuous infusion of morphine into the spinal cord.

Treatment of Chronic Pain

New techniques, such as acupuncture and electrical stimulation of the skin, are now being used with success. Relief of pain can be achieved by the use of placebos in a few patients. The anticipation of the relief of pain is thought to stimulate the release of endorphins, which inhibit the normal pain pathway.

Relief of Pain by Rhizotomy or Cordotomy

Surgical relief of pain has been used extensively in patients with terminal cancer. Posterior rhizotomy or division of the posterior root of a spinal nerve effectively severs the conduction of pain into the CNS. It is a relatively simple procedure, but, unfortunately, the operation deprives the patient of other sensations besides pain. Moreover, if the pain sensation is entering the spinal cord through more than one spinal nerve, it may be necessary to divide several posterior roots.

Thoracic cordotomy has been performed with success in patients with severe pain originating from the lower abdomen or pelvis. Essentially, the operation consists of dividing the lateral spinothalamic tracts by inserting a knife into the anterolateral quadrant of the

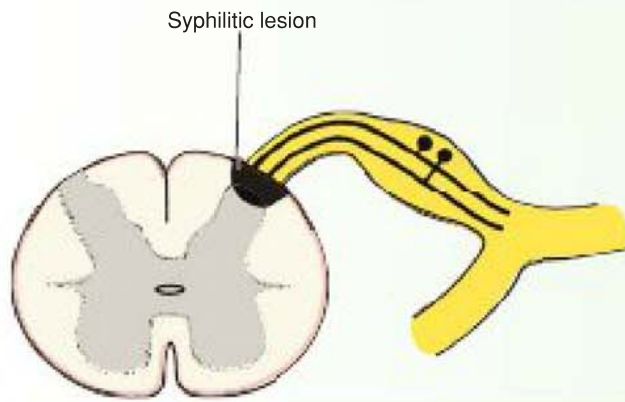


Figure 4-30 Site of a syphilitic lesion on the spinal cord.

spinal cord. Remember that the lateral spinothalamic fibers have originated in cells of the substantia gelatinosa in the opposite posterior gray column and that they cross the spinal cord obliquely and reach their tract in the white column three or four segments higher than their posterior root of entry. Cervical cordotomy has been performed successfully in patients with intractable pain in the neck or thorax.

Tabes Dorsalis

Tabes dorsalis is caused by syphilis. The organism causes a selective destruction of nerve fibers at the point of entrance of the posterior root into the spinal cord, especially in the lower thoracic and lumbosacral regions (Fig. 4-30). The following symptoms and signs may be present: (1) stabbing pains in the lower limbs, which may be very severe; (2) paresthesia, with numbness in the lower limbs; (3) hypersensitivity of skin to touch, heat, and cold; (4) loss of sensation in the skin of parts of the trunk and lower limbs and loss of awareness that the urinary bladder is full; (5) loss of appreciation of posture or passive movements of the limbs, especially the legs; (6) loss of deep pain sensation, such as when the muscles are forcibly compressed or when the Achilles tendon is compressed between the finger and thumb; (7) loss of pain sensation in the skin in certain areas of the body, such as the side of the nose or the medial border of the forearm, the thoracic wall between the nipples, or the lateral border of the leg; (8) ataxia of the lower limbs as the result of loss of proprioceptive sensibility (the unsteadiness in gait is compensated to some extent by vision; however, in the dark or if the eyes are closed, the ataxia becomes worse and the person may fall); (9) hypotonia as the result of loss of proprioceptive information that arises from the muscles and joints; and (10) loss of tendon reflexes, owing to degeneration of the afferent fiber component of the reflex arc (the knee and ankle tendon jerks are lost early in the disease).

Muscle Activity

Muscle tone is a state of continuous partial contraction of a muscle and is dependent on the integrity of a monosynaptic reflex arc (see description on p. 160). The receptor organs are the muscle spindles. The afferent neuron enters the spinal cord through the posterior root and synapses with the effector neuron or lower motor neuron

in the anterior gray column. The lower motor neuron supplies the muscle fibers by traveling through the anterior roots, the spinal nerves, and peripheral nerves.

Muscle Tone

Muscle tone is abolished if any part of that simple reflex arc is destroyed. An atonic muscle feels soft and flabby and atrophies rapidly. Normal muscle tone exhibits a certain resilience or elasticity, and, when a muscle is passively stretched by moving a joint, a certain degree of resistance is felt. Normal muscle tone depends on the integrity of the monosynaptic reflex arc described above and the control superimposed on it by impulses received through the descending tracts from supraspinal levels. Note that muscle spindles are excitatory to muscle tone, whereas neurotendinous receptors are inhibitory to muscle tone.

Voluntary Movement

Voluntary movement is initiated by the individual. A series of different muscles are made to contract for the purpose of reaching a goal. This would suggest that the descending tracts that influence the activity of the lower motor neurons are driven by information received by the sensory systems, the eyes, the ears, and the muscles themselves and are affected further by past afferent information that has been stored in the memory. Moreover, the whole process may be colored by past and present emotional input. The limbic structures appear to play a role in emotion, motivation, and memory and may influence the initiation process of voluntary movement by their projections to the cerebral cortex.

The descending pathways from the cerebral cortex and brainstem, that is, the upper motor neurons, influence the activity of the lower motor neurons either directly or through internuncial neurons. Most of the tracts originating in the brainstem that descend to the spinal cord also are receiving input from the cerebral cortex.

The corticospinal tracts are believed to control the prime mover muscles, especially those responsible for the highly skilled movements of the distal parts of the limbs. The other supraspinal descending tracts play a major role in the simple basic voluntary movements and, in addition, adjust muscle tone so that easy and rapid movements of the joints can take place.

Interestingly, the basal ganglia and cerebellum do not give rise directly to descending tracts that influence the activities of the lower motor neuron, yet these parts of the nervous system greatly influence voluntary movements. This influence is accomplished indirectly by fibers that project to the cerebral cortex and brainstem nuclei, which are the sites of origin of the descending tracts.

Pyramidal and Extrapyrarnidal Tracts

The term **pyramidal tract** is used commonly by clinicians and refers specifically to the corticospinal tracts. The term came into common usage when it was learned that the corticospinal fibers become concentrated on the anterior part of the medulla oblongata in an area referred to as the **pyramids**.

The term **extrapyramidal tracts** refers to all the descending tracts other than the corticospinal tracts.

Upper Motor Neuron Lesions

Damage to descending tracts produces a variety of clinical signs, depending on the specific location.

Corticospinal Tract (Pyramidal Tract) Lesions

Lesions restricted to the corticospinal tracts produce the following clinical signs:

1. The **Babinski sign** is present. The great toe becomes dorsally flexed, and the other toes fan outward in response to scratching the skin along the lateral aspect of the sole of the foot. The normal response is plantar flexion of all the toes. Remember that the Babinski sign is normally present during the first year of life because the corticospinal tract is not myelinated until the end of the first year of life.
2. The explanation for the Babinski sign is thought to be that, normally, the corticospinal tracts produce plantar flexion of the toes in response to sensory stimulation of the skin of the sole. When the corticospinal tracts are nonfunctional, the influence of the other descending tracts on the toes becomes apparent, and a kind of withdrawal reflex takes place in response to stimulation of the sole, with the great toe being dorsally flexed and the other toes fanning out.
3. The **superficial abdominal reflexes** are absent. The abdominal muscles fail to contract when the skin of the abdomen is scratched. This reflex is dependent on the integrity of the corticospinal tracts, which exert a tonic excitatory influence on the internuncial neurons.
4. The **cremasteric reflex** is absent. The cremaster muscle fails to contract when the skin on the medial side of the thigh is stroked. This reflex arc passes through the first lumbar segment of the spinal cord. This reflex is dependent on the integrity of the corticospinal tracts, which exert a tonic excitatory influence on the internuncial neurons.
5. **Fine-skilled voluntary movements are lost.** This occurs especially at the distal end of the limbs.

Lesions of Other Descending Tracts (Extrapyramidal Tracts)

The following clinical signs are present in lesions restricted to the other descending tracts:

1. **Severe paralysis** with little or no muscle atrophy (except secondary to disuse).
2. **Spasticity or hypertonicity** of the muscles. The lower limb is maintained in extension, and the upper limb is maintained in flexion.
3. **Exaggerated deep muscle reflexes** and clonus may be present in the flexors of the fingers, the quadriceps femoris, and the calf muscles.
4. **Clasp-knife reaction.** When passive movement of a joint is attempted, muscle spasticity produces resistance. The muscles, on stretching, suddenly give way due to neurotendinous organ-mediated inhibition.

In clinical practice, organic lesions restricted only to the pyramidal tracts or only to the extrapyramidal tracts are rare. Usually, both sets of tracts are affected to a variable extent, producing both groups of clinical signs. As the pyramidal tracts normally tend to increase muscle tone and the extrapyramidal tracts inhibit muscle tone, the balance between these opposing effects will be altered, producing different degrees of muscle tone.

Lower Motor Neuron Lesions

Trauma, infection (poliomyelitis), vascular disorders, degenerative diseases, and neoplasms may all produce a

lesion of the lower motor neuron by destroying the cell body in the anterior gray column or its axon in the anterior root or spinal nerve. The following clinical signs are present with lower motor neuron lesions:

1. Muscles exhibit **flaccid paralysis**.
2. Muscles **atrophy**.
3. Muscles **lose reflexes**.
4. **Muscular fasciculation** (muscle twitching) is seen only with slow destruction of the lower motor neuron cell.
5. **Muscular contracture** (shortening of the paralyzed muscles) occurs more often in the antagonist muscles whose action is no longer opposed by the paralyzed muscles.
6. Normally innervated muscles respond to stimulation by the application of faradic (interrupted) current, and the contraction continues as long as the current is passing. Galvanic or direct current causes contraction only when the current is turned on or turned off. When the lower motor neuron is cut, a muscle will no longer respond to interrupted electrical stimulation 7 days after nerve section, although it still will respond to direct current. After 10 days, the response to direct current also ceases. This change in muscle response to electrical stimulation is known as the **reaction of degeneration**.

Types of Paralysis

Hemiplegia is a paralysis of one side of the body and includes the upper limb, one side of the trunk, and the lower limb.

Monoplegia is paralysis of one limb only.

Diplegia is paralysis of two corresponding limbs (i.e., arms or legs).

Paraplegia is paralysis of the two lower limbs.

Quadriplegia is paralysis of all four limbs.

Relationship of Muscular Signs and Symptoms to Nervous System Lesions

The type of abnormal muscle tone resulting from nervous system damage will depend on the location of the lesion.

Hypotonia

Hypotonia exists when the muscle tone is diminished or absent. It occurs when any part of the monosynaptic stretch reflex arc is interrupted. It also occurs in cerebellar disease as the result of diminished influence on the γ motor neurons from the cerebellum.

Hypertonia

Hypertonia (spasticity, rigidity) exists when the muscle tone is increased. It occurs when lesions exist that involve supraspinal centers or their descending tracts but *not* the corticospinal tract. It also may occur at the local spinal segmental level and be produced by local excitation of the stretch reflex by sensory irritation (e.g., spasm of back muscles secondary to prolapsed intervertebral disc, spasm of abdominal muscles secondary to peritonitis).

Tremors

Tremors are rhythmic involuntary movements that result from the contraction of opposing muscle groups. These may be slow, as in **parkinsonism**, or fast, as in toxic tremors from thyrotoxicosis. They may occur at rest, as in parkinsonism, or with action, the so-called intention tremor, as seen in cerebellar disease.

Spasms

Spasms are sudden, involuntary contractions of large groups of muscles. Examples of spasms are seen in paraplegia and are due to lesions involving the descending tracts but not the corticospinal tract.

Athetosis

Athetosis are continuous, slow, involuntary, dysrhythmic movements that are always the same in the same patient and disappear during sleep. They impede voluntary movement. Athetosis occurs with lesions of the corpus striatum.

Chorea

Chorea consists of a series of continuous, rapid, involuntary, jerky, coarse, purposeless movements, which may occur during sleep. Chorea occurs with lesions of the corpus striatum.

Dystonia

Dystonia consists of frequent, maintained contractions of hypertonic muscles, leading to bizarre postures. It occurs with lesions of the lentiform nucleus.

Myoclonus

Myoclonus is a sudden contraction of an isolated muscle or part of a muscle. It occurs irregularly and commonly involves a muscle of a limb. It may be present with diseases that involve the reticular formation and the cerebellum. Normal myoclonic jerks sometimes occur in individuals as they are falling asleep and are believed to be due to a sudden temporary reactivation of the reticular formation.

Hemiballismus

Hemiballismus is a rare form of involuntary movement confined to one side of the body. It usually involves the proximal extremity musculature, and the limb involved is made to fly about in all directions. The lesion responsible occurs in the opposite subthalamic nucleus.

Acute Spinal Cord Injury

The incidence of acute spinal cord injuries in the United States is about 10,000 per year. The injury is catastrophic, because little or no regeneration of the severed nerve tracts takes place (see p. 71) and the individual is permanently disabled. Treatment has been restricted to anatomical realignment and stabilization of the vertebral column or decompression of the spinal cord. During the recovery process, the patient goes through intensive rehabilitation to optimize remaining neurologic function. Apart from improved management of medical complications, very little new therapy has been successful despite an enormous amount of research into the problem of neuronal regeneration in the spinal cord. Recently, the use of certain drugs (GM₁ ganglioside and methylprednisolone) administered to the patient soon after injury has resulted in some improvement in the neurologic deficit. Animal experiments appear to indicate that these drugs enhance the functional recovery of damaged neurons.

Chronic Compression of the Spinal Cord

If acute spinal cord injury is excluded, the causes of compression may be divided into extradural and intradural, which is further divided into those that arise outside the

spinal cord (extramedullary) and those that arise within the cord (intramedullary).

Extradural causes include herniation of an intervertebral disc, infection of the vertebrae with tuberculosis, and primary and secondary tumors of the vertebra; leukemic deposits and extradural abscesses may also compress the spinal cord. The two common extramedullary tumors are meningiomas and nerve fibromas. Intramedullary causes include primary tumors of the spinal cord, such as gliomas.

The clinical signs and symptoms are produced by an interference with the normal anatomical and physiologic functions of the spinal cord. Pressure on the spinal arteries causes ischemia of the spinal cord with degeneration of nerve cells and their fibers. Pressure on the spinal veins causes edema of the spinal cord with interference in the function of the neurons. Finally, direct pressure on the white and gray matter of the spinal cord and the spinal nerve roots interferes with nerve conduction. At the same time, CSF circulation is obstructed, and the composition of the fluid changes below the level of obstruction.

Clinical Signs

One of the earliest signs is pain, which may be local pain in the vertebra involved or pain radiating along the distribution of one or more spinal nerve roots. The pain is made worse by coughing or sneezing and is usually worse at night, when the patient is recumbent.

Interference with motor function occurs early. Involvement of the anterior gray column motor cells at the level of the lesion results in partial or complete paralysis of muscles, with loss of tone and muscle wasting. The early involvement of the corticospinal and other descending tracts produces muscular weakness, increased muscle tone (spasticity), increased tendon reflexes below the level of the lesion, and an extensor plantar response. The degree of sensory loss will depend on the nerve tracts involved. A lesion of the posterior white columns of the spinal cord will cause loss of muscle joint sense (proprioception), vibration sense, and tactile discrimination below the level of the lesion on the same side. Involvement of the lateral spinal thalamic tracts will cause loss of pain and heat and cold sensations on the opposite side of the body below the level of the lesion. A more detailed discussion of the symptoms and signs following injury to the ascending and descending tracts in the spinal cord is given on pages 163 and 166.

Because many spinal tumors are benign and can be successfully removed (provided that irreversible damage to the spinal cord has not occurred as a result of compression of the blood supply), an early accurate diagnosis is essential. The following investigations should be performed: (1) radiography of the vertebral column, including computed tomography (CT) and magnetic resonance imaging (MRI); (2) spinal tap; and (3) myelography when determining the diagnosis is difficult.

Clinical Syndromes Affecting the Spinal Cord

Myelopathy stems from a number of causes, including trauma, developmental abnormality, infection, autoimmune destruction, and genetic disease.

Spinal Shock Syndrome

Spinal shock syndrome is a clinical condition that follows acute severe damage to the spinal cord. All cord functions below the level of the lesion become depressed or lost, and

sensory impairment and flaccid paralysis occur. The segmental spinal reflexes are depressed due to the removal of influences from the higher centers that are mediated through the corticospinal, reticulospinal, tectospinal, rubrospinal, and vestibulospinal tracts. Spinal shock, especially when the lesion is at a high level of the cord, may also cause severe hypotension from loss of sympathetic vasomotor tone.

In most patients, the shock persists for less than 24 hours, whereas in others, it may persist for as long as 1 to 4 weeks. As the shock diminishes, the neurons regain their excitability, and the effects of the upper motor neuron loss on the segments of the cord below the lesion, for example, spasticity and exaggerated reflexes, appear.

The presence of spinal shock can be determined by testing for anal sphincter reflex activity. The reflex can be initiated by placing a gloved finger in the anal canal and stimulating the anal sphincter to contract by squeezing the glans penis or clitoris or gently tugging on an inserted Foley catheter. An absent anal reflex indicates spinal shock. A cord lesion involving the sacral segments of the cord would nullify this test, insofar as the neurons giving rise to the inferior hemorrhoidal nerve to the anal sphincter (S2–S4) would be nonfunctioning.

Destructive Spinal Cord Syndromes

When neurologic impairment is identified following the disappearance of spinal shock, it can often be categorized into one of the following syndromes: (1) complete cord transection syndrome, (2) anterior cord syndrome, (3) central cord syndrome, or (4) Brown-Séquard syndrome or hemisection of the cord. Clinical findings often indicate a combination of lower motor neuron injury (at the level of destruction of the cord) and upper motor neuron injury (for those segments below the level of destruction).

COMPLETE CORD TRANSECTION SYNDROME

Complete cord transection syndrome (Fig. 4-31) results in complete loss of all sensibility and voluntary movement below the level of the lesion. It can be caused by fracture dislocation of the vertebral column, by a bullet or stab wound, or by an expanding tumor. The following characteristic clinical features will be seen *after* the period of spinal shock has ended:

1. Bilateral lower motor neuron paralysis and muscular atrophy in the segment of the lesion results from damage to the neurons in the anterior gray columns (i.e., lower motor neuron) and possibly from damage to the nerve roots of the same segment.
2. In bilateral spastic paralysis below the level of the lesion, bilateral Babinski sign is present, and, depending on the level of the segment of the spinal cord damaged, bilateral loss of the superficial abdominal and cremaster reflexes occurs. All these signs are caused by an interruption of the corticospinal tracts on both sides of the cord. The bilateral spastic paralysis is produced by the cutting of the descending tracts other than the corticospinal tracts.
3. In bilateral loss of all sensations below the level of the lesion, loss of tactile discrimination and vibratory and proprioceptive sensations is due to bilateral destruction of the ascending tracts in the posterior white columns. The loss of pain, temperature, and light touch sensations is caused by section of the lateral and anterior spinothalamic tracts on both sides. Because these tracts cross obliquely, the loss of thermal and light touch sensations occurs two or three segments below the lesion distally.

4. Bladder and bowel functions are no longer under voluntary control, because all the descending autonomic fibers have been destroyed.

With a complete fracture dislocation at the L2–L3 vertebral level (i.e., a level below the lower end of the cord in the adult), no cord injury occurs. Neural damage is confined to the cauda equina, and lower motor neuron, autonomic, and sensory fibers are involved.

ANTERIOR CORD SYNDROME

Anterior cord syndrome (Fig. 4-31) can be caused by cord contusion during vertebral fracture or dislocation, from injury to the anterior spinal artery or its feeder arteries with resultant ischemia of the cord, or by a herniated intervertebral disc. The following characteristic clinical features are seen *after* the period of spinal shock has ended:

1. Bilateral lower motor neuron paralysis in the segment of the lesion and muscular atrophy is caused by damage to the neurons in the anterior gray columns (i.e., lower motor neuron) and possibly by damage to the anterior nerve roots of the same segment.
2. In bilateral spastic paralysis below the level of the lesion, the extent depends on the size of the injured area of the cord. The bilateral paralysis is caused by the interruption of the anterior corticospinal tracts on both sides of the cord. The spasticity is produced by the interruption of tracts other than the corticospinal tracts.
3. Bilateral loss of pain, temperature, and light touch sensations below the level of the lesion are caused by interruption of the anterior and lateral spinothalamic tracts on both sides.
4. Tactile discrimination and vibratory and proprioceptive sensations are preserved because the posterior white columns on both sides are undamaged.

CENTRAL CORD SYNDROME

Central cord syndrome is most often caused by hyperextension of the cervical region of the spine (Fig. 4-31). The cord is pressed on anteriorly by the vertebral bodies and posteriorly by the bulging of the ligamentum flavum, causing damage to the central region of the spinal cord. Radiographs of these injuries often appear normal because no fracture or dislocation has occurred. The following characteristic clinical features are seen *after* the period of spinal shock has ended:

1. Bilateral lower motor neuron paralysis in the segment of the lesion and muscular atrophy is caused by damage to the neurons in the anterior gray columns (i.e., lower motor neuron) and possibly by damage to the nerve roots of the same segment.
2. In bilateral spastic paralysis below the level of the lesion with characteristic sacral “sparing,” lower limb fibers are affected less than upper limb fibers because the descending fibers in the lateral corticospinal tracts are laminated, with the upper limb fibers located medially and the lower limb fibers located laterally (Fig. 4-29).
3. In bilateral loss of pain, temperature, light touch, and pressure sensations below the level of the lesion with characteristic sacral “sparing,” because the ascending fibers in the lateral and anterior spinothalamic tracts are also laminated, with the upper limb fibers located medially and the lower limb fibers located laterally, the upper limb fibers are more susceptible to damage than the lower limb fibers.

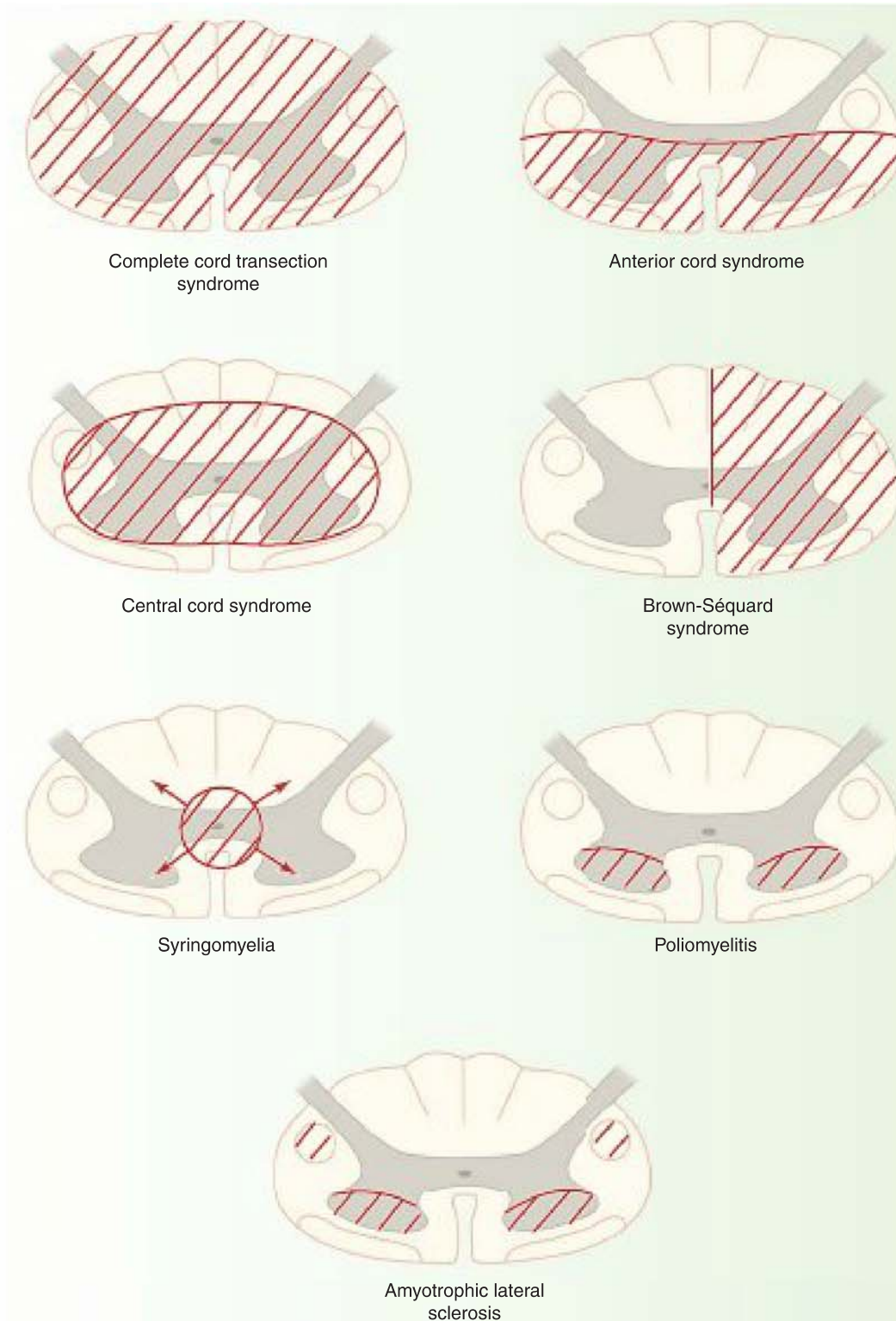


Figure 4-31 Spinal cord syndromes.

Thus, the clinical picture of a patient with a history of a hyperextension injury of the neck, presenting with motor and sensory tract injuries involving principally the upper limb, would strongly suggest central cord syndrome. The sparing of the lower part of the body may be evidenced

by (1) the presence of perianal sensation, (2) good anal sphincter tone, and (3) the ability to move the toes slightly. In patients whose damage is caused by edema of the spinal cord alone, the prognosis is often very good. Mild central cord syndrome that consists only of paresthesias of the

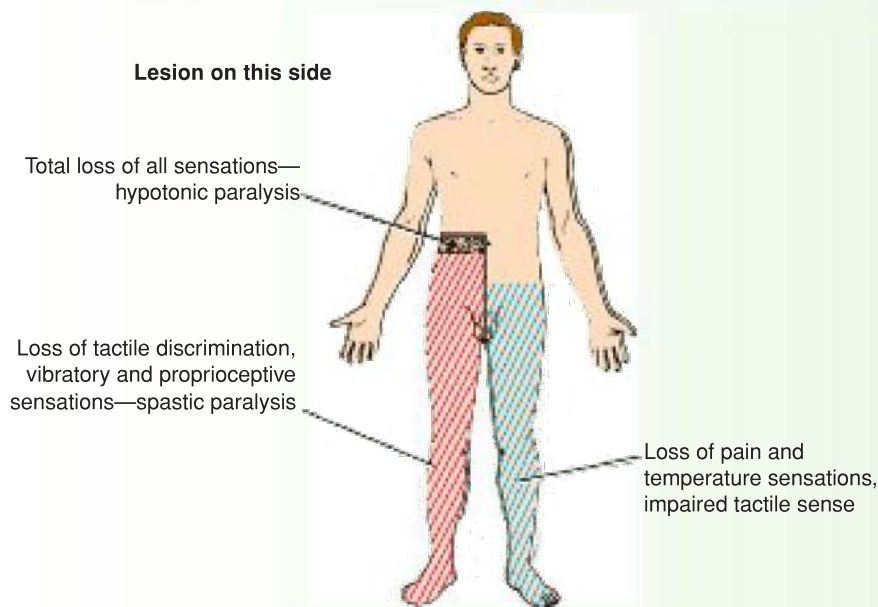


Figure 4-32 Brown-Séquard syndrome with a spinal cord lesion at the right 10th thoracic level.

upper part of the arm and some mild arm and hand weakness can occur.

BROWN-SÉQUARD SYNDROME (CORD HEMISECTION)

Hemisection of the spinal cord can be caused by fracture dislocation of the vertebral column, by a bullet or stab wound, or by an expanding tumor (Fig. 4-31). Incomplete hemisection is common; complete hemisection is rare. The following characteristic clinical features are seen in patients with complete hemisection of the cord (Fig. 4-32) after the period of spinal shock has ended:

1. Ipsilateral lower motor neuron paralysis in the segment of the lesion and muscular atrophy are caused by damage to the neurons on the anterior gray column and possibly by damage to the nerve roots of the same segment.
2. In ipsilateral spastic paralysis below the level of the lesion, an ipsilateral Babinski sign is present, and, depending on the segment of the cord damaged, an ipsilateral loss of the superficial abdominal reflexes and cremasteric reflex occurs. All these signs are due to loss of the corticospinal tracts on the side of the lesion. Spastic paralysis is produced by interruption of the descending tracts other than the corticospinal tracts.
3. Ipsilateral band of cutaneous anesthesia in the segment of the lesion results from destruction of the posterior root and its entrance into the spinal cord at the level of the lesion.
4. Ipsilateral loss of tactile discrimination and of vibratory and proprioceptive sensations below the level of the lesion are caused by destruction of the ascending tracts in the posterior white column on the same side of the lesion.
5. Contralateral loss of pain and temperature sensations below the level of the lesion is due to destruction of the crossed lateral spinothalamic tracts on the same side of the lesion. Because the tracts cross obliquely, the sensory loss occurs two or three segments below the lesion distally.
6. Contralateral but not complete loss of tactile sensation below the level of the lesion condition is caused

by destruction of the crossed anterior spinothalamic tracts on the side of the lesion. Here, again, because the tracts cross obliquely, the sensory impairment occurs two or three segments below the level of the lesion distally. The contralateral loss of tactile sense is incomplete because discriminative touch traveling in the ascending tracts in the contralateral posterior white column remains intact.

Syringomyelia

Syringomyelia, which is due to a developmental abnormality in the formation of the central canal, most often affects the brainstem and cervical region of the spinal cord. At the site of the lesion, cavitation and gliosis in the central region of the neuroaxis occurs (Fig. 4-33). The following characteristic signs and symptoms are found:

1. Pain and temperature sensations are lost in dermatomes on both sides of the body related to the affected segments of the cord. This loss commonly has a shawllike distribution caused by the interruption of the lateral spinothalamic tracts as they cross the midline in the anterior gray and white commissures. The patient commonly complains of accidental burning injuries to the fingers.
2. Tactile discrimination, vibratory sense, and proprioceptive sense are normal because the ascending tracts in the posterior white column are unaffected.
3. Lower motor neuron weakness is present in the small muscles of the hand. It may be bilateral, or one hand may suffer before the other. As the lesion expands in the lower cervical and upper thoracic region, it destroys the anterior horn cells of these segments. Later, the other muscles of the arm and shoulder girdles undergo atrophy.
4. Bilateral spastic paralysis of both legs may occur, with exaggerated deep tendon reflexes and the presence of a positive Babinski response. These signs are produced by the further expansion of the lesion laterally into the white column to involve the descending tracts.

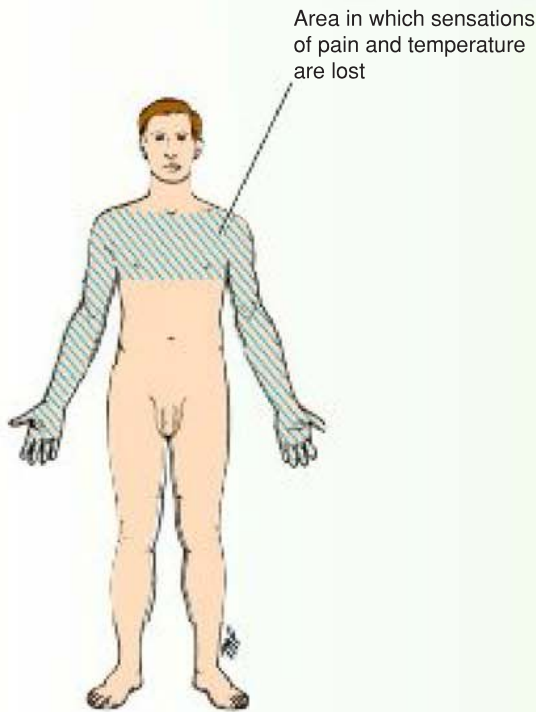


Figure 4-33 Skin area in which the sensations of pain and temperature are lost in syringomyelia.

5. Horner syndrome may be present. This is caused by the interruption of the descending autonomic fibers in the reticulospinal tracts in the lateral white column by the expanding lesion.

Poliomyelitis

Poliomyelitis is an acute viral infection of the neurons of the anterior gray columns of the spinal cord (Fig. 4-31) and cranial nerve motor nuclei. Immunization has greatly reduced the incidence of poliomyelitis, which was once a feared disease. Paralysis and muscle wasting follow motor nerve cell death. Lower limb muscles are more often affected than those of the upper limb. In severe poliomyelitis, respiration may be threatened due to the paralysis spreading to the intercostal muscles and diaphragm. Muscles of the face, pharynx, larynx, and tongue may also be paralyzed. Improvement usually begins at the end of the first week as the edema in the affected area subsides, and function returns to the neurons that have not been destroyed.

Multiple Sclerosis

Multiple sclerosis is a common disease confined to the CNS, causing demyelination of the ascending and descending tracts. It is a disease of young adults, and the cause is unknown. Autoimmunity, infection, and heredity, alone or in combination, may play a role in its etiology. A breach in the integrity of the blood-brain barrier in an individual who is genetically predisposed to the disease may be responsible. This could result in the invasion of the brain and spinal cord by some infection allowing leukocytes to enter the normally immunologically protected CNS. Inflammation

and demyelination with loss of the myelin sheath results in the breakdown of the insulation around the axons, and the velocity of the action potentials is reduced and ultimately becomes blocked. Although myelin is relatively rich in lipid (70% to 80%), it also contains proteins that play a role in myelin compaction. Many of these proteins in the myelin of the CNS differ from those in the peripheral nervous system. Experimentally, basic myelin proteins injected into animals have been shown to produce a strong immune response, and CNS demyelination occurs. Mutations in the structure of myelin protein can possibly occur and be responsible for some inherited forms of demyelination. Also possible is that autoantigens develop in multiple sclerosis.

The course of multiple sclerosis is chronic with exacerbations and remissions. Because of the widespread involvement of different tracts at different levels of the neuroaxis, the signs and symptoms are multiple, but remissions do occur. Weakness of the limbs is the most common sign of the disease. Ataxia due to involvement of the tracts of the cerebellum may occur, but spastic paralysis may also be present.

Recent research has suggested that the remissions in multiple sclerosis may in part be explained by the remodeling of the demyelinated axonal plasma membrane so that it acquires a higher than normal number of sodium channels, which permit conduction of action potentials despite the loss of myelin.

Patients who have the progressive form of the disease without remissions have substantial damage to the axons as well as the myelin. This would suggest that multiple sclerosis is not just a demyelinating disease but one with axonal pathology also.

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (Lou Gehrig disease) is a disease confined to the corticospinal tracts and the motor neurons of the anterior gray columns of the spinal cord (Fig. 4-31). It is rarely familial and is inherited in about 10% of patients. Amyotrophic lateral sclerosis is a chronic progressive disease of unknown etiology. Typically, it occurs in late middle age and is inevitably fatal in 2 to 6 years. The lower motor neuron signs of progressive muscular atrophy, paresis, and fasciculations are superimposed on the signs and symptoms of upper motor neuron disease with paresis, spasticity, and Babinski response. The motor nuclei of some cranial nerves may also be involved.

Parkinson Disease

Parkinson disease is associated with neuronal degeneration in the substantia nigra and, to a lesser extent, in the globus pallidus, putamen, and caudate nucleus. The degeneration of the inhibitory nigrostriate fibers results in a reduction in the release of the neurotransmitter dopamine within the corpus striatum. This leads to hypersensitivity of the dopamine receptors in the postsynaptic neurons in the corpus striatum, which become overactive. The characteristic signs of the disease include tremor and cogwheel rigidity (hyperkinetic activity) and difficulty initiating voluntary movements, which are slow (hypokinetic activity).

Pernicious Anemia

Pernicious anemia, a form of megaloblastic anemia, is caused by vitamin B₁₂ deficiency. The disease may produce

extensive damage to the tracts in the posterior and lateral white columns of the spinal cord as well as peripheral nerve degeneration. Widespread sensory and motor losses may be present due to involvement of the ascending and descending tracts of the spinal cord.

Vertebral Column Radiography

The views commonly used in radiography are anteroposterior, lateral, and oblique. Vertebral destruction due to tuberculosis or primary or secondary tumors of the vertebrae or fractures due to trauma usually can be revealed by radiographic examination. Erosion of the pedicles by a tumor within the intervertebral foramina may be seen. Narrowing of the space between the vertebral bodies with bony spurs because of osteoarthritic changes in adjacent vertebral bodies can also be seen.

Computed Tomography and Magnetic Resonance Imaging

CT scans of the vertebrae and joints can be obtained (Fig. 4-34). A protrusion of an intervertebral disc can be identified, and the presence of narrowing of the vertebral canal (**spinal stenosis**) can be diagnosed.

Sagittal MRI is increasingly being used to replace CT and myelography. The parts of a vertebra, the intervertebral

disc, the posterior longitudinal ligament, and meningeal sac (**thecal sac**) can easily be identified (Fig. 4-35).

Myelography

The subarachnoid space can be studied radiographically by the injection of a contrast medium into the subarachnoid space by spinal tap. Iodized oil has been used with success. This technique is referred to as **myelography** (Figs. 4-36 and 4-37).

If the patient is sitting in the upright position, the oil sinks to the lower limit of the subarachnoid space at the level of the lower border of the second sacral vertebra. By placing the patient on a tilting table, the oil can be made to gravitate gradually to higher levels of the vertebral column.

A normal myelogram will show pointed lateral projections at regular intervals at the intervertebral space levels because the opaque medium fills the lateral extensions of the subarachnoid space around each spinal nerve. The presence of a tumor or a prolapsed intervertebral disc may obstruct the movement of the oil from one region to another when the patient is tilted.

With the recent technologic advances in CT scans and MRIs, intrusive procedures such as myelography are usually not required to make a diagnosis.

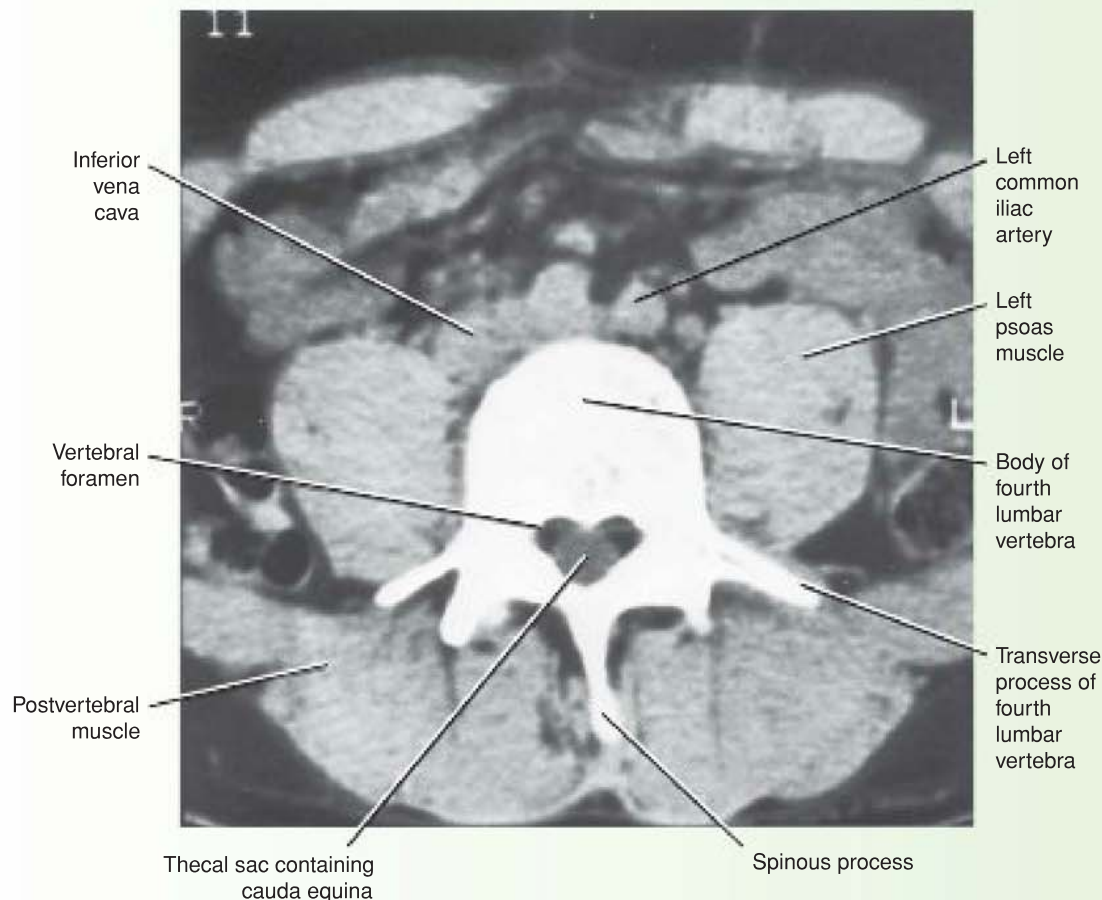


Figure 4-34 Horizontal (axial) CT scan of the fourth lumbar vertebra.

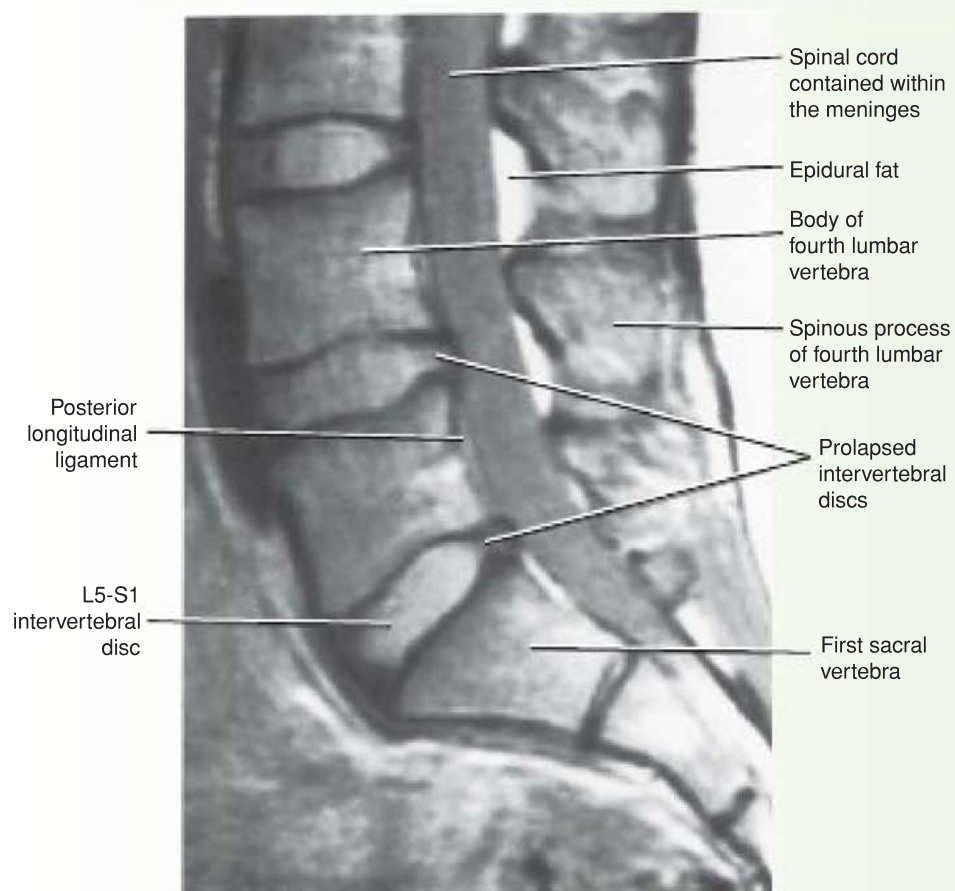


Figure 4-35 Sagittal MRI of the lumbosacral part of the vertebral column showing several prolapsed intervertebral discs. (Courtesy Dr. Pait.)



Figure 4-36 Posteroanterior myelogram of the cervical region of a 22-year-old woman.

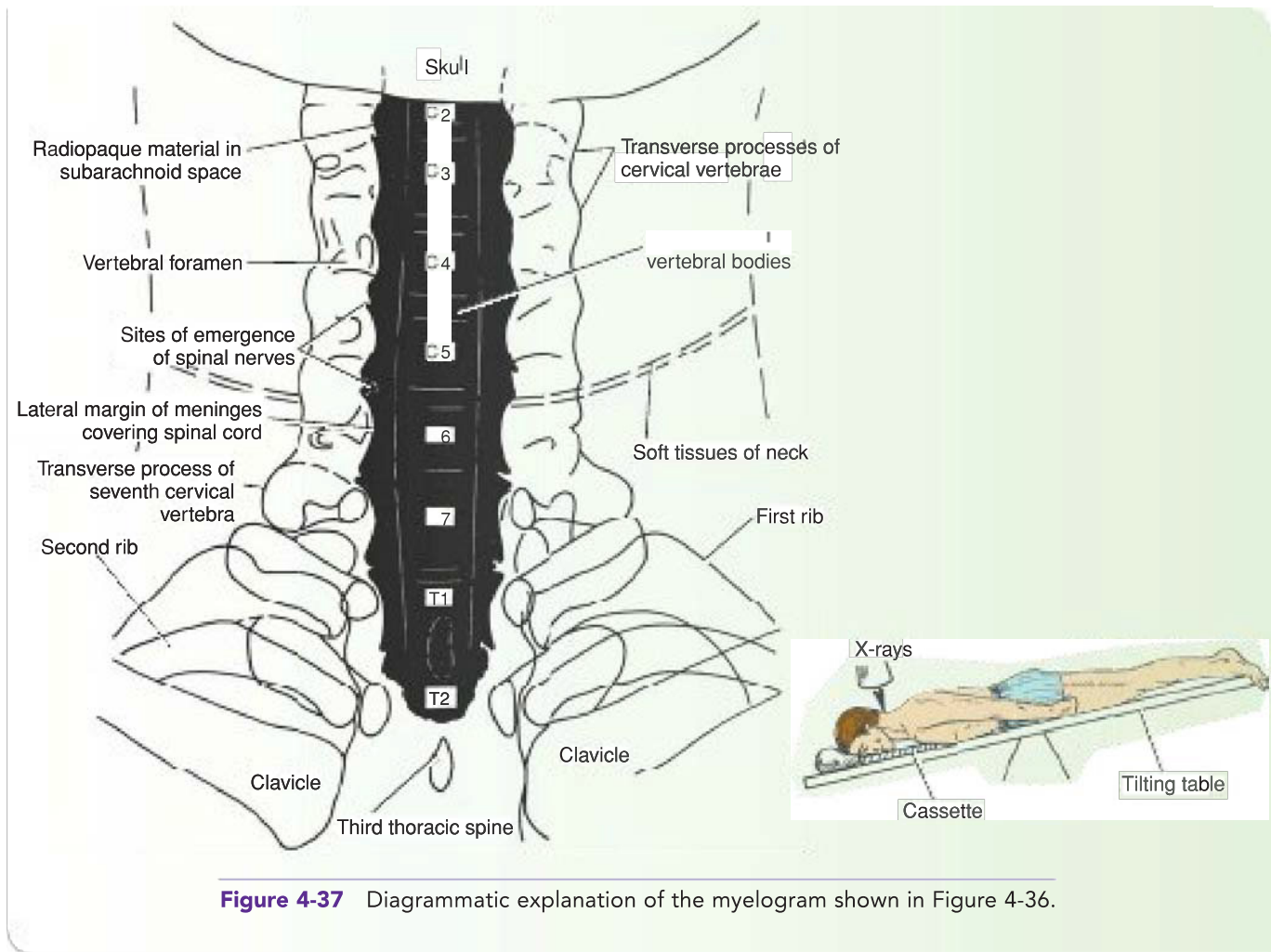


Figure 4-37 Diagrammatic explanation of the myelogram shown in Figure 4-36.

Key Concepts

Vertebral Column

- The vertebral column is composed of 33 vertebrae—7 cervical, 12 thoracic, 5 lumbar, 5 sacral (fused), and 4 coccygeal (fused).
- Because it is segmented and made up of vertebrae, joints, and intervertebral discs, it is a flexible structure.
- The vertebral arch encloses a space called the vertebral foramen. The collective vertebral foramina form the vertebral canal—the housing of the spinal cord.

Spinal Cord

- Roughly cylindrical, the spinal cord extends from the rostral end of the medulla oblongata to the eventual tapering of the cord, called the conus medullaris.
- Within the vertebral canal, the spinal cord is covered by three layers of meninges, the dura mater, arachnoid mater, and pia mater. Between the latter two, is the subarachnoid space, which is filled with CSF.
- The spinal cord is composed of an inner core of gray matter surrounded by an outer covering of white matter.
- On cross section, the gray matter is seen as an H-shaped pillar with anterior and posterior columns or horns. A lateral gray column is present in thoracic and upper lumbar segments.
- Most nerve cells in the anterior columns are large, alpha and multipolar, and their axons pass out in the anterior roots of the spinal nerves.
- The substantia gelatinosa of the posterior gray column is largely composed of neurons receiving

afferent fibers concerned with pain, temperature, and touch from the posterior root.

- The nucleus proprius is situated anterior to the substantia gelatinosa throughout the spinal cord and communicates information regarding the senses of proprioception, two-point discrimination, and vibration with the posterior white columns.
- The lateral gray column extends from T1 to L2(3) segments and contains cells that give rise to preganglionic sympathetic fibers.
- The white matter of the spinal cord can be described as anterior, lateral, and posterior columns formed from multiple myelinated nerve bundles, or tracts.

Ascending Tracts

- Pain and temperature sensations ascend in the lateral spinothalamic tract; light touch and pressure ascend in the anterior spinothalamic tract. Together, these are also referred to as the anterolateral system.
- First-order axons entering the spinal cord with pain and temperature information, synapse on second-order neurons of the substantia gelatinosa, cross obliquely to the opposite side through the anterior gray and white commissure, and ascend in the contralateral white column as the lateral spinothalamic tract. The anterior spinothalamic ascends in the contralateral anterior white column.
- As the anterolateral system tracts ascend through the medulla oblongata and pons, they form the spinal lemniscus and eventually synapse on third-order neurons in the ventral posterolateral nucleus of the thalamus.
- First-order axons carrying discriminative touch and vibration sensations enter the dorsal horn and continue traveling upward in the posterior white column as the fasciculus gracilis (sacral, lumbar, and lower thoracic spinal nerves) and fasciculus cuneatus (thoracic and cervical spinal nerves). Fibers of these fasciculi synapse on the second-order neurons of the ipsilateral nuclei gracilis and cuneatus in the medulla oblongata, respectively.
- The axons of the second-order neurons cross anteromedially as the internal arcuate fibers and ascend a compact bundle, called the medial lemniscus, until synapsing with third-order neurons in the ventral posterolateral nucleus of the thalamus.

Descending Tracts

- Motor neurons situated in the anterior gray columns of the spinal cord send axons through the anterior roots to the spinal nerves and are often referred to as lower motor neurons. This is the final pathway to the muscles.
- Corticospinal tracts are pathways concerned with voluntary movement of muscles. Axons of pyramidal cells in the motor cortex descend through the internal capsule, form the cerebral peduncles of the midbrain, then separate into smaller bundles throughout the pons. As the axons approach the medulla, they coalesce, forming the pyramid of the rostral medulla oblongata and cross caudal medulla as the decussation of the pyramids. The axons continue as the lateral corticospinal tract of the spinal cord until they synapse on the lower motor neurons of the anterior horns.
- Reticulospinal tracts may facilitate or inhibit voluntary movements or reflex activity.
- Tectospinal tracts are concerned with reflex postural movements in response to visual stimuli while vestibulospinal tracts are concerned with postural activity associated with balance.
- The rubrospinal tract facilitates the activity of flexor muscles and inhibits the activity of extensor muscles.

? Clinical Problem Solving

1. A 53-year-old widower is admitted to the hospital complaining of a burning pain over his right shoulder region and the upper part of his right arm. The pain started 3 weeks previously and, since that time, has progressively worsened. The pain is accentuated when the patient moves his neck or coughs. Two years previously, he had been treated for osteoarthritis of his vertebral

column. The patient states that he had been a football player at college and continued to take an active part in the game until he was 42 years old. Physical examination reveals weakness, wasting, and fasciculation of the right deltoid and biceps brachii muscles. The right biceps tendon reflex is absent. Radiologic examination reveals extensive spur formation on the bodies of the fourth, fifth,