The Spinal Cord
A Review of Functional Neuroanatomy

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KEYWORDS
- Spinal cord anatomy • Dorsal columns • Corticospinal tract
- Lateral spinothalamic tract • Central cord syndrome • Brown-Sequard
- Compressive myelopathy

KEY POINTS
- The spinal cord is located within the spinal canal but does not extend the entire length of the vertebral canal.
- The spinal cord is incompletely divided into 2 halves by a deep anterior median fissure and a shallow posterior median sulcus.
- The anatomic organization of ascending and descending pathways in the spinal cord often allows precise longitudinal and transverse localization of the pathologic process.
- Disproportion between the length of the spinal cord and the length of the bony spinal vertebral column gives rise to longitudinal localization discrepancies.

The spinal cord controls the voluntary muscles of the trunk and upper and lower extremities and receives sensory input from these areas of the body. It is anchored to the dura by the denticulate ligaments and extends from the medulla oblongata to the lower border of the first lumbar vertebra. A basic knowledge of spinal cord anatomy is essential for the interpretation of and understanding of pathologic findings. The blood supply of the spinal cord is discussed elsewhere in this issue. In this article, anatomic structures are correlated with relevant clinical signs and symptoms.

GROSS ANATOMY OF SPINAL CORD

The spinal cord is located within the spinal canal but does not extend the entire length of the vertebral canal. The spinal cord length is about 45 cm in men and 43 cm in women and its width ranges from 1.27 cm in cervical and lumbar regions to 64 mm in the thoracic region. There are 20 or 21 pairs of denticulate ligaments that constitute a surgical landmark for the anterolateral segment of the spinal cord. Caudally, the...
spinal cord is anchored by filum terminale, which is a nonneural membrane arising from the conus medullaris and descending to attach to the coccyx (Fig. 1).

The spinal cord has 31 segments (8 cervical, 12 thoracic or dorsal, 5 lumbar, 5 sacral, and 1 coccygeal), each of which (except the first cervical segment, which has only a ventral root) has a pair of dorsal and ventral roots and a pair of spinal nerves. There are no sharp boundaries between the segments within the cord, but the cervical and lumbar enlargements, giving rise to nerve roots for arms and legs, respectively, are clearly apparent. Each dorsal and ventral root joins in the intervertebral foramina to form a spinal nerve. Outside the spinal cord and just proximal to its junction with the ventral root, the dorsal root has an oval enlargement, the dorsal root ganglion, which contains sensory neurons (Fig. 2A). The neurons of the sensory ganglia are pseudounipolar nerve cells (ie, the cell body is in the ganglion where a solitary nerve process divides into a long process coming from the periphery as the receptor and

Fig. 1. The spinal cord is shown in situ to indicate its relationship to vertebral and other bony structures. Drawing by Frank Netter, MD. (Netter illustration from www.netterimages.com. © Elsevier, Inc. All rights reserved.)
a much shorter process entering the spinal cord). There are no synapses in the dorsal root ganglia. Spinal nerves leave the vertebral canal through the intervertebral foramina. The first cervical nerve emerges from the vertebral canal between the occipital bone and the atlas; the eighth cervical nerve emerges between the seventh cervical (C7) and the first thoracic (T1) vertebrae. All spinal nerves caudal to C7-T1 exit beneath the corresponding vertebrae.

Spinal cord and vertebral column levels do not correspond because of their differences in rates of growth during embryonic development. In the upper cervical region

Fig. 2. (A) Anterior view of the cord showing dorsal and ventral roots of the spinal cord and formation of the spinal nerves and their dorsal and ventral rami as well as the rami communicantes. Drawing by Frank Netter, MD. (B) Posterior view of the cord indicating the meningeal layers, denticulate ligaments, and dorsal root ganglia. Drawing by Frank Netter, MD. (Netter illustration from www.netterimages.com. © Elsevier, Inc. All rights reserved.)
the cord segment corresponds to the like-numbered vertebral body. From C5 to C8 the spinal cord level is 1 level higher than the corresponding vertebral body. Thus, the fifth cervical vertebral body corresponds to the level of the sixth spinal cord segment. In the upper thoracic region, the vertebral spinal process is 2 segments above the corresponding cord segment. In the lower thoracic and upper lumbar regions, the difference between the vertebral and cord level is 2 or 3 segments so that a spinal cord sensory level at T9 would correspond to a pathologic abnormality at the sixth or seventh thoracic vertebral body. Sacral cord levels correspond to vertebral T12-L1 levels. These longitudinal discrepancies should be taken into account when a sensory level is found during clinical examination (see later discussion and Fig. 3). As the nerve roots descend to their corresponding vertebral level, they become more oblique from rostral to caudal so that the lumbar and sacral nerves descend almost vertically to reach their points of exit (see Fig. 1). The lumbosacral roots below

![Spinal Segmental Sensory Innervation](image)

**Fig. 3.** Spinal segmental sensory innervation. (From Squire LR, Bloom FE, McConnell SK, et al. Fundamental neuroscience. 2nd edition. New York: Academic Press; 2003. p. 678; with permission. Copyright © 2002, Elsevier Science (USA), All rights reserved.)
the termination of the spinal cord form the cauda equina. These spinal roots surround the filum terminale and are organized such that caudal-most segments are most centrally located, but this somatotopic organization is not usually of much value in clinical localization.

MENINGES

Dura mater, arachnoid, and pia mater are the membranes covering the spinal cord from the most superficial layer to that closest to the spinal cord, respectively (see Fig. 2B). The outer of the 2 cranial dural layers serves as the cranial periosteum and at the foramen magnum the inner layer separates to descend as the dural sleeve of the spinal cord. The epidural space separates dura from the vertebral canal and contains a layer of fat that can be useful as a magnetic resonance imaging landmark. A network of epidural veins called Batson’s plexus is also in the epidural space and is believed to be relevant to the spread of infection or metastatic cancer. The subdural space is a potential space between the dura mater and the arachnoid space. The subarachnoid space is a clinically important and relatively wide space filled with cerebrospinal fluid separating the arachnoid from the pia mater surrounding the spinal cord. Together the arachnoid and pia are called the leptomeninges.

LONGITUDINAL DIVISIONS

The spinal cord is incompletely divided into 2 halves by a deep anterior median fissure and a shallow posterior median sulcus (Fig. 4). The anterior median fissure contains a double fold of pia mater, and its floor is the anterior (ventral) white commissure. More laterally is the anterolateral sulcus, marking the exit of the ventral roots. The posterior median sulcus is a shallow septum, on either side of which lies the posterolateral sulcus, the site of the posterior nerve roots entry. Three funiculi divide the white matter of the cord. The posterior funiculus lies between the posterior median sulcus

Fig. 4. The spinal cord at the eighth cervical segmental level indicating external landmarks of spinal cord, funiculi, dorsal root fiber somatotopic organization at the root entry zone, and Rexed’s laminae. (Adapted from Nolte JK, Angevine JB. The human brain in photographs and diagrams. St Louis, MO: Mosby; 2005. p. 11; with permission.)
and the dorsal roots. The lateral funiculus lies between the dorsal and ventral roots and the anterior funiculus lies between the anterior median fissure and the ventral roots (see Fig. 4).

**SPINAL ROOTS AND NERVES**

The dorsal roots consist of several types of afferent fibers somatotopically organized such that the largest diameter fibers are medial to the smaller ones (see Fig. 4). These largest afferent fibers (Ia and Ib) are heavily myelinated fibers that conduct the afferent limb of muscle stretch reflexes and carry information from muscle spindles and Golgi tendon organs. Medium-sized fibers (A-beta) convey impulses from mechanoreceptors in skin and joints. Small, thinly myelinated A-delta and C-type unmyelinated fibers convey noxious and thermal sensation.

The anterior root bundles constitute the motor output from the spinal cord. These anterior root bundles are large-diameter alpha motor neuron axons to the extrafusal striated muscle fibers, and gamma motor neuron axons, which supply the intrafusal striated muscle spindles as well as preganglionic autonomic motor fibers. After the posterior and anterior roots join to form spinal nerves, they divide into a smaller primary dorsal ramus and a larger primary ventral ramus (see Fig. 2B). The dorsal ramus consists of a medial sensory branch and a lateral motor branch to supply the skin and paraspinal muscle at the segmental level. The ventral ramus is much larger and contributes to the brachial and lumbosacral plexuses as well as to segmental branches such as intercostal nerves.

**INTERNAL ANATOMY OF THE SPINAL CORD: GRAY MATTER**

The gray matter of the spinal cord is an H-shaped structure in the transverse section of the spinal cord with 2 symmetric halves connected by a narrow bridge or commissure composed of gray and white matter through which runs the central canal (see Fig. 4). The central canal is a continuation of the fourth ventricle. Lined with ciliated columnar epithelium, it is filled with cerebrospinal fluid. Encircling the columnar epithelium is a band of neuroglia, the substantia gliosa. The ratio of gray substance to white substance varies markedly at different levels of spinal cord. In the thoracic levels the amount of gray matter is less than that in the cervical and lumbosacral enlargements.

An imaginary coronal line through the central canal divides the gray matter into anterior and posterior columns (or anterior and posterior horns). The anterior horn contains alpha and gamma motor neurons. In the thoracic region, the posterolateral portion of the anterior column is called the lateral (intermediolateral) column. This lateral column contains preganglionic cells for the autonomic nervous system in the thoracic and upper lumbar areas. From T1 to L2 spinal segments, preganglionic sympathetic neurons within the intermediolateral gray column give rise to sympathetic axons, which leave the spinal cord through the anterior roots and travel to the adjacent sympathetic ganglia through white rami comminucantes. All spinal nerves contain gray rami communicantes; however, white rami communicantes are only found from the T1 to L2 levels. Parasympathetic preganglionic neurons arise from the spinal segments S2, S3, and S4 within the intermediolateral gray column. These neurons also leave the spinal cord through ventral roots and, after projecting to the viscera, synapse on postganglionic parasympathetic ganglia. The posterior column or horn is directed posterolaterally and is separated from the posterolateral sulcus by an important thin layer of white matter, the tract of Lissauer.
ARCHITECTURAL LAMINATION OF THE SPINAL CORD GRAY MATTER

Rexed’s cytoarchitectural studies of the spinal cord gray matter are the basis of the conventional gray matter division into the 10 eponymic laminae enumerated as Rexed’s laminae I to X. Laminae I to IX are arranged from dorsal to ventral, whereas Lamina X consists of a circle of cells around the central canal (see Fig. 4, also diagrammed in Fig. 5). Some of the laminae correspond to specific cell types with functional significance. Thus, Lamina II corresponds to the substantia gelatinosa and plays an important part in transmission of fibers subserving thermal and painful sensations. Lamina VII occupies the intermediate gray zone and contains Clarke’s nucleus. Lamina IX contains the largest spinal cord cells, the alpha motor neurons that supply skeletal extrafusal muscle fibers.

THE SPINAL CORD WHITE MATTER

Descending Fiber Systems

The corticospinal tract, arising from the precentral motor cortex, is the largest and most significant descending tract of the human spinal cord (Fig. 6).1 Almost 90% of the corticospinal tract fibers decussate in the lower medulla to form the lateral corticospinal tract, whereas 8% of the nondecussating descending fibers form the anterior corticospinal tract and 2% of noncrossing fibers generate the uncrossed lateral corticospinal tract. The uncrossed fibers of the anterior corticospinal tract may cross to the contralateral side via the anterior white commissure to project to interneurons and lower motor neurons of the contralateral side. These fibers provide synaptic input to neurons responsible for the control of axial body and proximal limb movements. There are also fibers in the corticospinal tract that project to the dorsal gray column and function as modulators of sensory afferent information, allowing the brain to process pain stimuli selectively.

The rubrospinal tract consists of fibers that originate from the contralateral red nucleus in the brainstem and descend just anterior to the corticospinal tract, projecting to interneurons that modulate proximal, largely flexor movements of the upper limb (Fig. 7).

Fig. 7 also shows several other ventral descending tracts that work together to modulate responses to changes in body posture and position. The vestibulospinal fasciculus consists of lateral and medial tracts, both of which arise from brainstem vestibular nuclei and, via interneurons that project to alpha motor neurons, provide excitatory input for extensor and antigravity muscles to control tone and posture. Reticulospinal tracts also arise from extensor-biased upper motor neurons and modulate muscle spindles. The tectospinal tract, derived from the contralateral superior colliculus in the midbrain tectum, crosses to the opposite side and descends in the contralateral ventral longitudinal bundle, providing neural input to ventral gray interneurons. This anatomic tract mediates reflex head turning movements in reaction to sudden auditory, visual, and tactile stimuli.

Ascending Fiber Systems

All afferent axons have their primary neurons in the dorsal root ganglia. The level of decussation varies among ascending systems. However, as a general rule, the ascending axons synapse before decussating to the contralateral side.

The dorsal column tract is responsible for the transmission of sensations of vibration, proprioception (position sense), and 2-point discrimination from the skin and joints.2 The fasciculus gracilis is located medially and transmits sensation from the lower half of the body, whereas the fasciculus cuneatus, which is
located more laterally at levels rostral to T5, carries proprioceptive input from the upper thorax through the back of the head. The gracile and cuneate fasciculi terminate in the gracile and cuneate nuclei of the lower medulla. The axons of these second-order neurons decussate as the internal arcuate fibers and travel rostrally as the medial lemniscus to the ventral posterolateral thalamic nucleus (Fig. 7A).

The spinothalamic tract mediates sensation of pain and temperature. These sensory modalities are transmitted by small-diameter axons, which, after entering the cord via the dorsal root entry zone, divide into short ascending and descending branches that run longitudinally in Lissauer’s tract for 1 to 2 segments and then synapse in the dorsal horn. The postsynaptic fibers cross to the opposite side of the spinal cord anterior to the central canal in the ventral white commissure and continue rostrally within the anterolateral funiculus (see Fig. 7B). The spinothalamic tract consists of a ventral tract that conveys light touch sensation, and a lateral tract that conveys pain and temperature sensation. Somatotopic organization of the spinothalamic tracts is the opposite of the dorsal column tracts: rostral (upper limb) regions are represented medially, whereas more caudal regions are in the lateral aspect of the spinothalamic tract. The spinothalamic tract projects to ventral posterolateral and intralaminar thalamic nuclei.

The spinoreticular tract courses ipsilaterally without decussating within the ventrolateral portion of the spinal cord ending in the reticular formation of the brainstem from which it projects to intralaminar thalamic nuclei and then to the limbic system. This tract has a regulatory role in the sensation of pain, especially deep, chronic pain as well as the emotional reaction to and memory of painful stimuli. The difficult treatment problem of chronic pain from myelopathic disease processes may derive from dysfunction of these (and other) afferent fibers.

The spinocerebellar pathway provides cerebellar inputs via 2 spinocerebellar systems that terminate on the ipsilateral cerebellum, the dorsal spinocerebellar tract arising from Clarke’s nucleus at levels T1-L2 and its upper body equivalent, the ventral spinocerebellar (or cuneocerebellar) tract. These tracts convey unconscious proprioceptive information from Golgi tendon organs and muscle spindles to coordinate smooth execution of movements.

Fig. 5. The somatotopic organization of the spinal cord, as depicted here schematically at a cervical level, explains clinical syndromes, such as (1) the hemicord (Brown-Sequard) syndrome; (2) isolated tractopathies or preferential loss of specific sensory modalities; (3) extramedullary compressive lesions causing ascending symptoms caused by the superficial location of more caudal pathways; (4) intramedullary lesions producing descending sensory symptoms often with sacral sparing; (5) early central cord syndromes affecting the spinothalamic tract fibers crossing through the anterior commissure, giving “suspended” pain and temperature sensory loss. Gray matter is divided into groups of neurons that form layers in dorsal and ventral horns. Termination of large and small afferent axons in the cord varies by depth and the 2 groups enter the cord separately with little overlap. The 4 medial motor systems, represented schematically in the figure, terminate largely on interneurons that project bilaterally controlling movements that involve multiple segments. Thus unilateral lesions of the medial motor systems, unlike those of the lateral corticospinal tract, do not produce obvious clinical deficits. Ap, Appendicular; Ax, axial; C, cervical; Ex, extensor; Fl, flexor; L, lumbar; S, sacral; T, thoracic. (Adapted and modified from Squire LR, Bloom FE, McConnell SK, et al. Fundamental neuroscience. 2nd edition. San Diego: Academic Press; 2003. p. 681; with permission. Copyright © 2002, Elsevier Science (USA), All rights reserved.)
The anatomic organization of ascending and descending pathways in the spinal cord described earlier often allows precise longitudinal and transverse localization of the pathologic process. Such localization not only directs diagnostic imaging procedures efficiently to the appropriate site but also offers early etiologic clues because many disease processes preferentially produce specific patterns of spinal cord dysfunction. For example, “tractopathies” such as dorsal column dysfunction occurs with syphilis and B12 or copper deficiency, whereas isolated spinothalamic tract dysfunction raises the suspicion of a paraneoplastic disorder. However, localization is infrequently entirely etiologically specific. Thus, Brown-Sequard syndrome can occur after trauma, or in the setting of tumor, demyelinating disease, and other intrinsic cord processes. At times, falsely localizing or misleading signs and symptoms may distract the clinician from the appropriate area of the cord. For example, isolated lower limb vibratory sense loss can be caused by an exclusively cervical process such as demyelination that involves fasciculus gracilis selectively.

The practical 5-step approach to spinal cord localization and triage outlined below takes advantage of neuroanatomic features to exclude the most emergent situations of compressive myelopathy.

**STEP 1**: Do symptoms and signs suggest the disease in the spinal cord?

Certain configurations classically suggest spinal cord pathologic abnormalities, but, as depicted in Fig. 8, there are a large number of signs and symptoms that, while possibly localizable to the spinal cord, could reflect disease in other parts of the nervous system as well. Perhaps the most confusing are paresthesias that present roughly symmetrically. These paresthesias could reflect polyneuropathy or dorsal
column pathologic abnormalities. Lower limb paraparesis without sensory signs could be a sign of a parasagittal meningioma.

**STEP 2: Is the problem a compressive myelopathy?**

Although all spinal cord problems are in a sense emergencies capable of causing irreparable damage to critical structures, some myelopathies dictate more rapid intervention than others. The most important early clinical distinction involves compressive versus noncompressive syndromes. Most compressive syndromes will reveal abnormalities on magnetic resonance imaging of the spinal cord/column.

**Compressive Myelopathies**

Compressive myelopathies may stem from extradural or intradural processes. Tumors and trauma are major causes of compressive myelopathies. In general, extradural neoplasms are more aggressive malignancies such as metastatic disease, whereas intradural neoplasms are more indolent tumors such as meningiomas or neurofibromas. Clinical clues to the presence of compressive myelopathies include the following:

1. Radicular and vertebral pain
2. Early upper motor neuron (UMN) signs, rare lower motor neuron (LMN) signs except possible root involvement at level of compression
Is the problem localizable to the spinal cord?

**Signs strongly suggestive of Spinal Card**
- Suspended band of sensory loss
- Sensory level on torso
- Spinal tract crossed findings (pyramidal on one side and contralateral spinothalamic)
- Dissociated sensory loss conforming to cord syndrome (syringomyelia, anterior spinal artery)
- Root plus longtract signs (sarcoïdosis, spondylosis)
- Isolated tractopathy (anterior horn, posterior column, spinothalamic)
- Lhermitte’s sign
- Urinary retention

**Signs consistent with, but not diagnostic of Spinal Cord**
- Bilateral symmetric sensory loss with normal reflexes (consider polyneuropathy)
- Paraparesis without sensory signs (consider parasagittal location)
- Hyporeflexia (consider polyneuropathy)
- Unilateral or bilateral upper motor neuron signs (consider brain or brainstem)
- Ascending sensory loss (consider AIDP or CIDP)
- Exertional worsening of symptoms (consider vascular, MS, or lumbar stenosis)

**Signs NOT suggestive of Spinal Cord**
- Monoparesis of arm
- Cranial nerve deficits (other than V)
- Pure lower motor neuron signs
- Paratonia (frontal lobe), Stiffness rather than spasticity (SPS, PD)
- Dysarthria, dysphagia, brisk jaw reflex (brainstem or ALS)
- Weakness with normal sensation and reflexes (MG)
- Proximal muscle weakness (watershed or myopathy)

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Fig. 8. General diagnostic guidelines to localize the lesion to spinal cord. Isolated urinary retention would be an unusual initial spinal cord symptom and also raises consideration of bifrontal pathologic abnormality or mechanical or pharmacologic obstruction, such as prostatic hypertrophy or anticholinergic toxicity. AIDP, acute inflammatory demyelinating neuropathy; ALS, amyotrophic lateral sclerosis; CIDP, chronic inflammatory demyelinating polyneuropathy; MG, myasthenia gravis; MS, multiple sclerosis; PD, Parkinson disease; SPS, stiff person syndrome. (Modified from Squire LR, Bloom FE, McConnell SK, et al. Fundamental neuroscience. 2nd edition. New York: Academic Press/Elsevier; 2003. p. 38; with permission.)
3. *Ascending* paresthesias (because the caudal-most dermatomes are represented superficially in the cord, see Fig. 7)
4. Brown-Sequard syndrome

**NONCOMPRESSIVE MYELOPATHIES**

Noncompressive myelopathies include most neuropathologies that initiate in the intramedullary compartment although noncompressive and intramedullary are not synonymous terms, as significant inflammatory or infectious conditions can expand the cord.\(^4\)

Clinical clues of a noncompressive myelopathy include the following:

1. Radicular and bone pain uncommon
2. Dysesthetic pain: the patient will use words to describe associated paresthesias, such as burning, tingling, or tightness
3. Late UMN with diffuse LMN at level of process
4. Dissociated sensory loss (spinothalamic dysfunction with spared dorsal columns or vice versa)
5. *Descending* sensory loss with sacral sparing because of the somatotopic organization of spinothalamic fibers (see Fig. 7)
6. Brown-Sequard syndrome is less common than in compressive myelopathy, but can be seen with varicella zoster virus infection\(^5\) or multiple sclerosis (MS)
7. Early sphincter involvement in conus medullaris lesions with saddle anesthesia

**STEP 3:** If the lesion is likely to be in the spinal cord, longitudinal localization will be based primarily on dermatomal levels.

Fig. 3 shows anterior and posterior views of the dermatomes. Because spinothalamic fibers ascend over 2 cord levels crossing in the anterior commissure, lesions are often located several levels above the rostral-most clinical signs. Key localizing signs at various longitudinal levels are shown in Fig. 9.

As described earlier in this article, disproportion between the length of the spinal cord and the length of the bony spinal vertebral column gives rise to longitudinal localization discrepancies. In the adult, the spinal cord ends at L1 level, but the roots descend in the cauda equina so that the L4 anterior root emerges from the 4th spinal lumbar segment opposite the vertebral level T12 and exits between the L4 and L5 vertebrae. Neoplastic or other disorders such as arachnoiditis that involve both the conus and the cauda equina produce a sometimes confusing set of signs that suggest pathologic abnormalities lower than the actual process. Any process below the L1 vertebral body will not impinge on the cord. Thus compressive lesions at the vertebral L4 level do not always dictate the same degree of urgency as similar pathologic processes such as a disc or tumor at T4.

The C1-C7 roots exist above their respective vertebral bodies. The C8 root is “extra” and exits between C7 and T1. Therefore, starting at the T1-T2 level, the remaining roots exit below the like-numbered vertebral body (see Fig. 9). At the lumbar level, however, the root affected also may be the next lower one. For example, an L4-L5 disc may impinge on the L5 root if it protrudes midline and on the L4 root if the disc emerges more laterally. There are no discs between the sacral spinal segments.

**STEP 4:** Once the longitudinal level is localized, transverse cord localization may help with the etiologic diagnosis.

Fig. 7 illustrates a composite diagram of tracts and nuclei at a cervical cord level and explains the occurrence of specific spinal cord syndromes. These syndromes and representative etiologies are summarized in Table 1.
Fig. 9. In addition to dermatomal sensory motor and reflex changes, some specific signs of localizing value can help to concentrate attention on a specific level of the spinal cord. AJ, ankle and knee jerk (muscle stretch reflex); LMN, lower motor neuron. Beevor's Sign: Upward retraction of abdomen when abdominal wall is stroked occurs when upper abdominal reflexes are present (T6-9) but lower reflexes (T9-11) are lost with lesion at approximately T10. (Modified from Squire LR, Bloom FE, McConnell SK, et al. Fundamental Neuroscience. 2nd edition. New York: Academic Press; 2003. p. 38; with permission.)
Complete Transverse Cord Involvement

All sensory and motor as well as autonomic pathways are interrupted. There is diminished sensation in all dermatomes 1 or 2 levels below the area of structural damage. A period of areflexia with spinal shock may occur immediately after disease onset. Common causes include transverse myelitis, MS, and trauma.

Hemicord (Brown-Sequard) Syndrome

Injury to the lateral corticospinal tract causes ipsilateral weakness, and ipsilateral loss of vibration sense causes posterior column damage. Contralateral spinothalamic signs are present.

Central Cord Syndrome

Small lesions damaging spinothalamic fibers crossing in the ventral commissure cause bilateral “suspended” sensory loss to pain and temperature often involving the lateral aspect of upper limbs. Sacral sparing occurs until late because these fibers are laterally represented in the cord. A larger central lesion produces pain and temperature loss in a cape distribution. Damaged anterior horn cells produced lower motor
neuron deficits at the level of the lesion with upper motor neuron signs, as listed below. Dorsal column function may be spared. Common causes include cord contusion hyperextension neck injuries, neuromyelitis optica, syringomyelia, and intrinsic cord tumors such as astrocytoma or ependymoma.

**Posterior Cord Syndrome**

Loss of vibration and proprioception below the level of the lesion are hallmarks of this syndrome. If the lesion is confined to posterior columns, tendon reflexes and strength may be normal. The somatotopic organization of the dorsal columns makes it mandatory to scan the entire cord because localized fasciculus gracilis lesions may give rise to the potentially misleading finding of isolated lower limb sensory disturbance. Conditions producing selective dorsal column pathologic abnormalities include MS, B12 and copper deficiency, chemotherapy toxicities, paraneoplastic sensory neuronopathy (anti-Hu), and tabes dorsalis. In addition to sensory ataxia, trophic changes and Charcot joints may be observed.

**Anterior Cord Syndrome**

Pain and temperature loss occur 2 levels below the lesion, and LMN signs are seen at the level of the lesion. Weakness is prominent. Atrophy and fasciculations may be seen in longer duration disease. Dorsal columns may be spared, particularly with anterior spinal artery syndrome. Common causes are anterior spinal artery infarction, trauma (disc herniation), spinal muscular atrophy, MS, poliomyelitis, and West Nile virus.

**Anterior Horn Cell and Pyramidal Tract Disease**

A combination of UMN and LMN signs is seen. There is sparing of sensation and sphincter function. Additional signs and symptoms may include muscle cramping, bulbar involvement, and pseudobulbar involvement. Amyotrophic lateral sclerosis and variants are the likely causes.

**Myeloneuropathy**

Myelopathic signs as well as signs of a polyneuropathy are found. There is a wide variety of possible etiologies, including deficiency states such as B12, copper, and vitamin E, sarcoidosis, Sjogren syndrome, and infections such as human immunodeficiency virus and human T-lymphotropic virus infections as well as numerous genetic conditions including adrenomyeloneuropathy, metachromatic leukodystrophy, and mitochondrial disorders.6

**STEP 5**: Once having satisfactorily localized the problem to the cord in both longitudinal and transverse dimensions, the clinician should return to consider further the possibility of falsely localizing signs and problematic presentations. Five potentially misleading sets of symptoms are summarized below.

**Exertional Symptoms**

Both spinal cord and noncord etiologies may give rise to exertional limb symptoms. For example, lumbar stenosis and claudication on a vascular basis may cause worsening of back and lower limb pain. Exertional symptoms also occur in the setting of dural fistulas, producing venous congestion of the cord. Heat-related exacerbation of symptoms in demyelinating disease could also be construed an exertion-related spinal cord syndrome.
Ipsilateral Shoulder Weakness and Lower Cranial Nerve Problems with Spasticity

Lesions in the foramen magnum interrupt decussating pyramidal tracts to the legs, which cross below those of the arms. Crural paresis or weakness of the lower limbs may be an early sign of foramen magnum pathologic abnormality or the syndrome, which is often accompanied by suboccipital and shoulder pain, may begin with ipsilateral shoulder and arm weakness followed by leg weakness and then contralateral arm involvement in a “round-the-clock” pattern. The weakness can begin in any limb.

Cranial Nerves and Spinal Cord Syndromes

Because the descending tract of the trigeminal subserving pain descends as far as C4, lesions in the upper cord may be accompanied by facial symptoms. At the foramen magnum, lower cranial nerves IX-XII may be involved along with spinal cord structures.

Referred Pain

Visceral afferent fibers to cell bodies from T1 to L2 or L3 in dorsal root ganglia accompany sympathetic efferents in spinal nerves. Most of these afferents carry information about visceral function (distortion or inflammation of organs) that may be interpreted as pain. Visceral pain, in contrast to somatic pain, is usually poorly localized and referred to an area of the body surface corresponding to the dermatome innervated by that spinal segment. Thus, arm pain may reflect cardiac ischemia or, conversely, a sense of tightness in the chest in an MS patient may suggest acute pulmonary disease but reflect active demyelination at a thoracic spinal cord segment. Typical patterns of dermatomal distribution of referred pain are summarized in Table 2.

Ascending Sensory Symptoms

As discussed earlier, compressive lesions may cause ascending sensory deficits. Sensory symptoms of acute inflammatory demyelinating polyneuropathy can ascend as well, however. Clear-cut UMN signs exclude acute inflammatory demyelinating polyneuropathy, but in the early “spinal shock” phase of a spinal cord process, these may

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Symptoms are predominantly pain, deep, aching unless otherwise indicated</th>
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<tr>
<td><strong>Referred Symptom Patterns</strong></td>
<td><strong>Diaphragm</strong></td>
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<tr>
<td><strong>Heart</strong></td>
<td>T1–T4</td>
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<tr>
<td><strong>Lungs</strong></td>
<td>T3–T10</td>
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<tr>
<td><strong>Stomach</strong></td>
<td>T6–T9</td>
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<td><strong>Gallbladder</strong></td>
<td>T7–T8</td>
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<td><strong>Duodenum</strong></td>
<td>T9–T10</td>
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<td><strong>Appendix</strong></td>
<td>T10</td>
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<tr>
<td><strong>Reproductive organs</strong></td>
<td>T10–T12</td>
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<tr>
<td><strong>Kidney and ureter</strong></td>
<td>L1–L2</td>
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Visceral pain differs from somatic pain in that it is poorly localized and referred to an area of the body surface. Visceral afferents and somatic pain fibers at specific cord levels converge on the same spinothalamic tract pathways, sometimes giving rise to diagnostic confusion.

not be present. When localization is in doubt but a spinal cord process is possible, full spinal cord imaging is always a prudent investigative approach.

REFERENCES