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Spinal Cord Anatomy and Localization

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ABSTRACT

PURPOSE OF REVIEW: This article focuses on clinically relevant teaching points in spinal anatomy and localizing the lesion in myelopathy.

RECENT FINDINGS: The principles underlying spinal cord lesion localization are well established, but improvements in MRI and the discovery of pathologic antibodies associated with causes of transverse myelitis distinct from multiple sclerosis, such as aquaporin-4 IgG and myelin oligodendrocyte glycoprotein IgG, have assisted in diagnosis.

SUMMARY: The spinal cord has a highly organized neuroanatomy of ascending and descending tracts that convey sensory, motor, and autonomic information. Using integration of clues from the patient's history and neurologic examination, the effective clinician can distinguish spinal cord from peripheral nerve or brain pathology, often determine the level and parts of the spinal cord affected by a lesion, and focus on a likely diagnosis. The advent of MRI of the spine has revolutionized investigation of spinal cord disorders, but an important place for strong clinical acumen still exists in assessing the patient with a myelopathy.

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INTRODUCTION

A thorough understanding of the anatomy of the spinal cord is of value to the neurologist because patients can present with an array of symptoms and signs referable to the spinal cord, and the capacity to distinguish cord pathology from brain or peripheral nerve pathology facilitates early diagnosis and avoids unnecessary investigations. Early diagnosis is particularly important because the consequences of delayed diagnosis of spinal cord pathology can be devastating, with the potential for permanent disability.

The neuroanatomy of the spinal cord is somatotopically and segmentally organized, with tracts and pathways that transmit sensory information from organs and peripheral receptors to the brain and motor information from the brain to internal and peripheral effector organs. This means that sensory or motor deficits in one area of the body can often be deduced to be due to damage in a specific area of the spinal cord.

The localization of lesional pathology is an essential skill for the neurologist and helps with differential diagnosis and targeted investigation. For the most part, spinal cord lesions are associated with upper motor neuron signs. The approach to a patient with a suspected myelopathy involves ascertaining the

motor and sensory level of the lesion. Certain patterns of signs may lead a neurologist to diagnose a spinal cord syndrome, such as a Brown-Séquard syndrome accompanying a partial transverse myelitis, which may provide a clue to the etiology of the underlying disease process.

The advent of MRI has revolutionized the diagnosis of myelopathies to the extent that specific diagnoses can often be made with MRI alone. Nevertheless, not all myelopathies are associated with MRI lesions, and, occasionally, subtle spinal cord lesions may be missed or difficult to appreciate on MRI. MRI can also identify unrelated pathologies or red herrings that may divert attention away from the actual diagnosis, which means that the clinical history and examination must be used in conjunction with MRI or other adjunctive investigations to establish a diagnosis.

This article focuses on clinically relevant neuroanatomy of the spinal cord and explains how neurologic symptoms and signs relate to the anatomy to give clues that enable the correct localization of spinal cord pathology.

ANATOMY OF THE SPINAL CORD

The spinal cord is a central nervous system (CNS) structure that arises from the inferior medulla of the brainstem above the C1 vertebra and runs in the spinal canal to terminate at the L1-L2 level of the lumbar spine (**FIGURE 1-1**¹). It is covered in the meninges, which are contiguous with the meninges of the brain; from the innermost layer to the outermost layer, the meninges are the pia, arachnoid, and dura mater. The spinal cord is broadly divided longitudinally into the cervical, thoracic, and lumbar regions, with the sacral spinal cord region that represents the very lower aspect of the spinal cord referred to as the conus medullaris. A modified fibrous termination of the pia mater called the filum terminale extends from the conus medullaris caudally to the coccyx, fusing at its lower end with the dura mater, where it anchors the entire spinal cord to the vertebral column.^{2,3}

Enlargements in the cervical region and lumbar regions reflect increased numbers of neurons in these regions to supply the upper and lower limbs, respectively (**FIGURE 1-1**). Pairs of sensory and motor nerve roots emerge at distinct spinal levels throughout the length of the cord.³ Sensory nerve roots emerge dorsally from the cord and motor nerve roots emerge ventrally before they meet to form mixed motor and sensory spinal nerves. The nerve roots are numbered C1 through C8 when they arise from the cervical cord and T1 through T12 when they arise from the thoracic cord. The lumbar L1 nerve roots emerge just below the T12 nerve roots. The lowermost nerve roots (lumbar, L2 through L5; sacral, S1 through S5; and the coccygeal nerve) fan out distally from the lower cord and are referred to as the cauda equina (Latin for horse's tail). Critically, only the sacral nerve roots emerge from the conus medullaris. The lowermost nerve roots inhabit the CSF-filled subarachnoid space that is formed because the pia-covered spinal cord terminates at L1-L2, but the arachnoid and dura continue to the S2 vertebral level. The subarachnoid space is clinically relevant as the site from which CSF can be collected in a lumbar puncture without risk to the spinal cord, as it is typically performed at the L3-L4 or L4-L5 level.⁴

Cross-sectional Anatomy of the Spinal Cord

The spinal cord is highly somatotopically organized, and its function is to transmit motor, sensory, and autonomic information.^{2,3} The neurons of the

KEY POINT

- Lumbar puncture is typically performed at the L3-L4 or L4-L5 level.

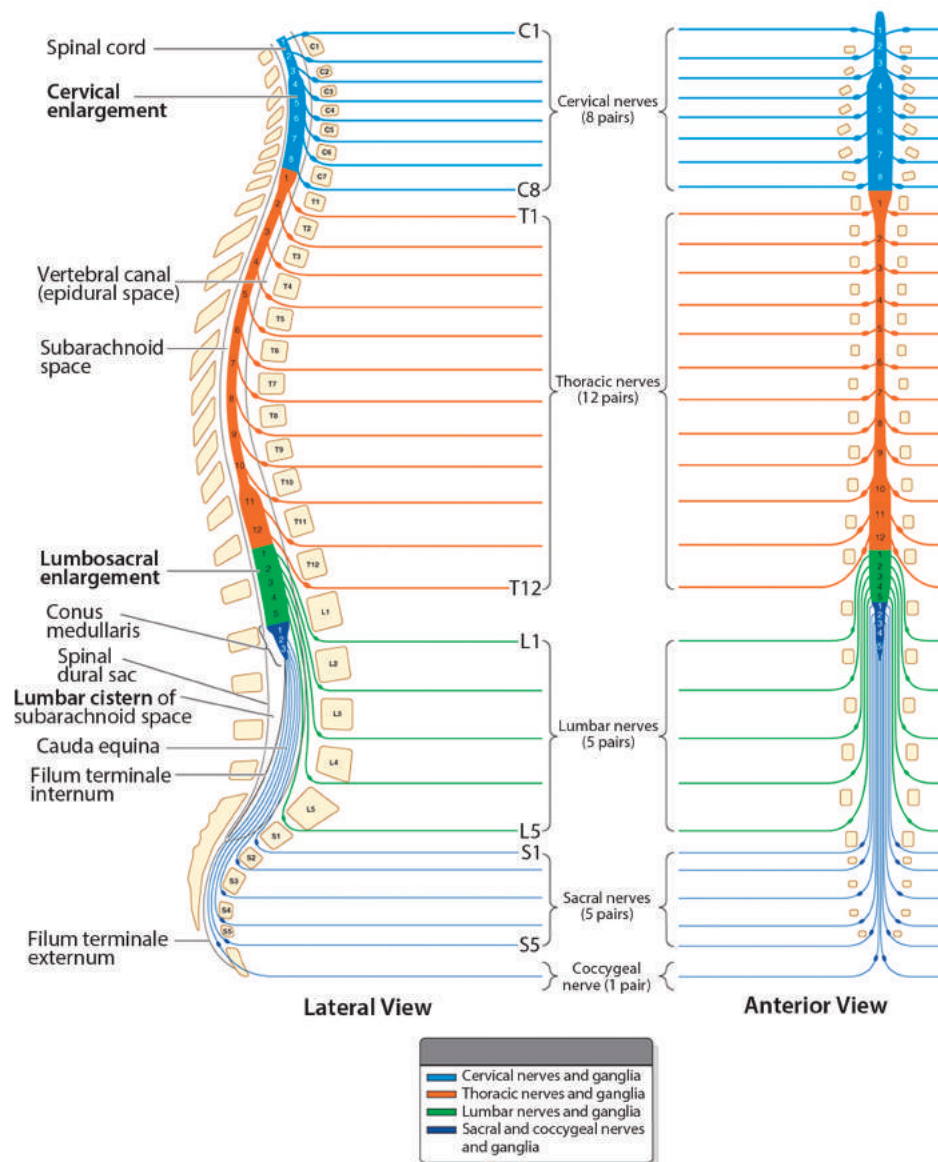


FIGURE 1-1

The gross anatomy of the spinal cord and adjacent spinal nerve roots.

Reprinted with permission from Moore KL, et al, Wolters Kluwer/Lippincott Williams & Wilkins.¹

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spinal cord are located centrally in an H-shaped or butterfly-shaped area known as the central gray matter (**FIGURE 1-2**). The central gray matter is connected across the midline by the gray commissure, within which lies the central canal of the cord. The anterior and posterior columns of the gray matter are usually referred to as the ventral and dorsal horns. The ventral horns contain alpha and gamma motor neurons and interneurons. Somatic motor neurons travel in the medial motor columns, where they innervate the axial muscles of the body, or in the lateral motor columns, where they innervate the upper and lower limbs. The dorsal horns are where somatic and visceral afferent (sensory) fibers enter the cord. Their cell bodies are in adjacent dorsal root ganglia. The intermediate gray

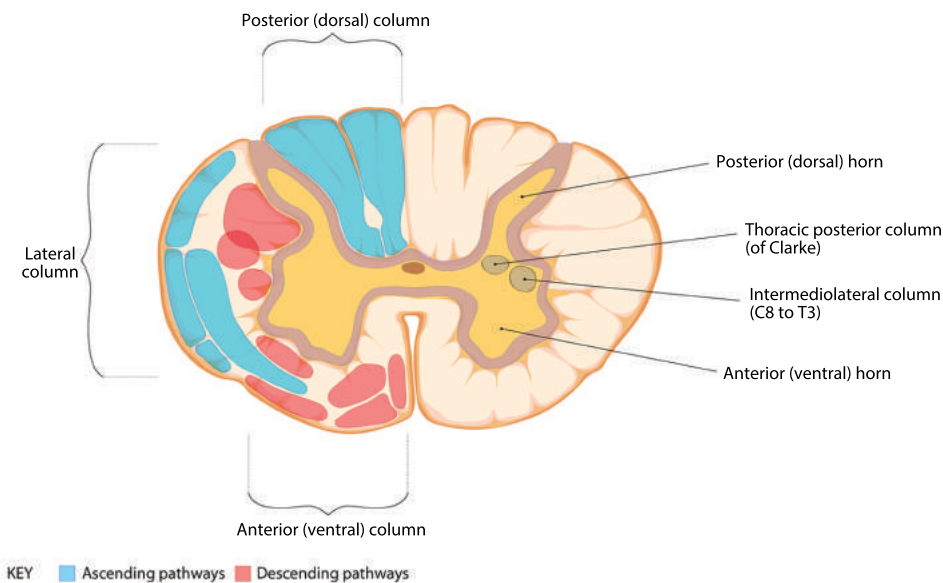


FIGURE 1-2

Cross-sectional anatomy of the gray and white matter tracts of the spinal cord.

Reprinted with permission from Cho TA, Continuum (Minneapolis Minn).³ © 2015 American Academy of Neurology.

KEY POINTS

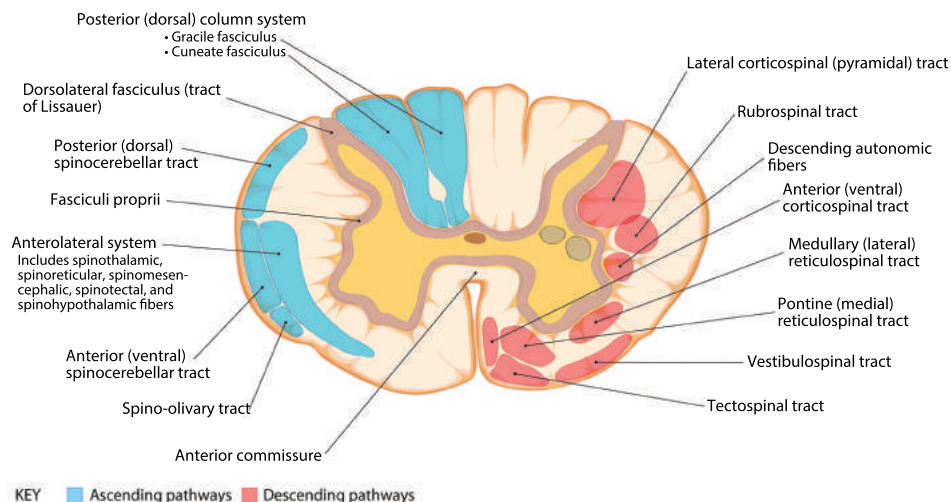
- The butterfly-shaped area of the spinal cord in cross section is known as the central gray matter.
- The descending motor pathways in the cord before the anterior horns are called upper motor neurons, and those of the anterior horns and somatic motor nerves are called lower motor neurons.

layer between the ventral and dorsal horns is where autonomic preganglionic cells lie, divided into intermediolateral and intermediomedial groups; the preganglionic sympathetic neurons are located from C8 through L1. The preganglionic parasympathetic neurons are located from S2 through S4.

The white matter of the spinal cord surrounds the gray matter and is composed of myelinated axons. Principal white matter tracts of clinical significance include the descending motor pathways of the corticospinal (pyramidal) tract, which run laterally and anteriorly (ventrally) in the cord throughout its length (FIGURE 1-3).⁵ These convey somatic motor information arising from the contralateral cerebral cortex, which decussates in the anterior medulla just before entering the cervical spinal cord. The descending motor pathways of the corticospinal tract synapse with the anterior horns cells that convey motor information via the motor nerve roots and then via somatic (peripheral) motor neurons to a target muscle. The descending motor pathways in the cord before the anterior horns are called upper motor neurons, and those of the anterior horns and somatic motor nerves are called lower motor neurons.

Additional descending motor pathways referred to as extrapyramidal tracts arise from the brainstem and convey involuntary information important in different aspects of motor control (FIGURE 1-3).⁵ Important extrapyramidal tracts include the rubrospinal tract, which transmits information to the spinal interneurons to maintain balance; the reticulospinal tract, which transmits motor information from the reticular formation in the brainstem; the tectospinal tract, which assists in coordinating head and eye movements; and the vestibulospinal tract, which transmits balance information to the extremities from the vestibular system.

Somatosensory information in the form of temperature, pinprick sensation, and touch is carried anterolaterally in the cord via the spinothalamic (also known

**FIGURE 1-3**

Cross-sectional anatomy of the principal white matter tracts of the spinal cord.

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as the anterolateral) tracts (**FIGURE 1-3**).³ This information is referred to the contralateral thalamus of the brain as the sensory nerve roots entering via the two dorsal horns synapse with second-order neurons. The axons of the second-order neurons ascend for two to three vertebral segments above where they enter the cord and then decussate across the midline. Proprioception, vibration, and touch are carried in the dorsal columns of the cord to the ipsilateral dorsal column nuclei of the medulla. The parts of the dorsal columns transmitting information from the lower limbs are referred to as gracile and those from the upper limbs are referred to as cuneate.

The spinocerebellar tracts (dorsal and ventral) are ipsilateral to the cerebellum and run laterally in the cord, transmitting proprioceptive information from muscles, tendons, and joints of the lower limbs. Cerebellar information from the upper limbs is conveyed by tracts arising from the external cuneate nucleus of the caudal medulla. White matter tracts are also involved in the transmission of information related to autonomic nervous system control of bowel and bladder function and reflexes.

The spinal cord also has the capacity to produce a motor response that is independent of the brain when triggered by external stimuli by way of spinal reflexes.⁶ The components of the monosynaptic spinal reflex arc include primary sensory afferents arising from peripheral stretch receptors synapsing directly in the spinal cord on alpha motor neurons that make up the efferent pathway to a muscle and therefore mediate a final motor response. The classic example in clinical practice is the patellar reflex, which occurs when stretch receptors in the patellar tendon are activated by the tendon hammer of a clinician causing reflex contraction of the quadriceps femoris muscle by way of a monosynaptic spinal reflex. More complex polysynaptic reflexes involving an integrated response to sensory input across multiple spinal segments to produce a motor response also occur as a result of spinal interneurons between afferent and efferent pathways. A polysynaptic reflex underlies the startle response to an unexpected touch to the trunk or limbs.

Vascular Supply of the Spinal Cord

The vascular supply of the spinal cord has clinical importance. The anterior spinal artery runs ventrally along the length of the cord and supplies blood to the anterior two-thirds of the spinal cord (including the corticospinal tracts), with the remaining dorsal one-third of the spinal cord (including the dorsal columns) supplied by the paired posterior spinal arteries. All three of these arteries emerge as branches of the vertebral arteries at the cervical-cranial junction, but some of their blood supply also comes from the thyrocervical trunk via the cervical arteries. Separate radiculomedullary arteries also arise from the thyrocervical trunk to supply the cervical spinal cord sequentially at the different levels at which they enter the vertebral canal. More caudal radiculomedullary arteries emerge directly from the aorta to supply the thoracolumbar cord. The radiculomedullary artery that anastomotically supplies the anterior spinal artery between T9 and T12 in most individuals is the great anterior radiculomedullary artery, or artery of Adamkiewicz (FIGURE 1-4).⁸ The artery of Adamkiewicz is responsible for supplying blood to the anterior cord from its point of entry in the lower thoracic cord down to the conus medullaris.

The venous drainage of the spinal cord is from the dorsal spinal and ventral spinal veins into the internal and external venous plexus adjacent to the dural sac and vertebral bodies. From there, venous blood is transported to the dural venous sinuses.⁹

IMPORTANCE OF THE CLINICAL HISTORY

Whether neurologic symptoms are the result of a myelopathy or brain or peripheral nervous system pathology and the precise location of the lesion in the cord can often be determined by an accurate history and examination combined with knowledge of the somatotopic and segmental anatomy of the spinal cord (TABLE 1-1).¹⁰ Clues to a spinal cord lesion in the history include bilateral sensory

KEY POINTS

- The monosynaptic spinal reflex is caused by activation of peripheral stretch receptors transmitting an impulse along primary sensory afferents that synapse directly on alpha motor neurons, causing a final efferent motor response.
- The artery of Adamkiewicz is the large radiculomedullary artery that supplies the anterior spinal artery between T9 and T12 in most individuals.
- The somatotopic organization of the spinal cord allows determination of the approximate or, in some cases, precise level of a spinal cord lesion.

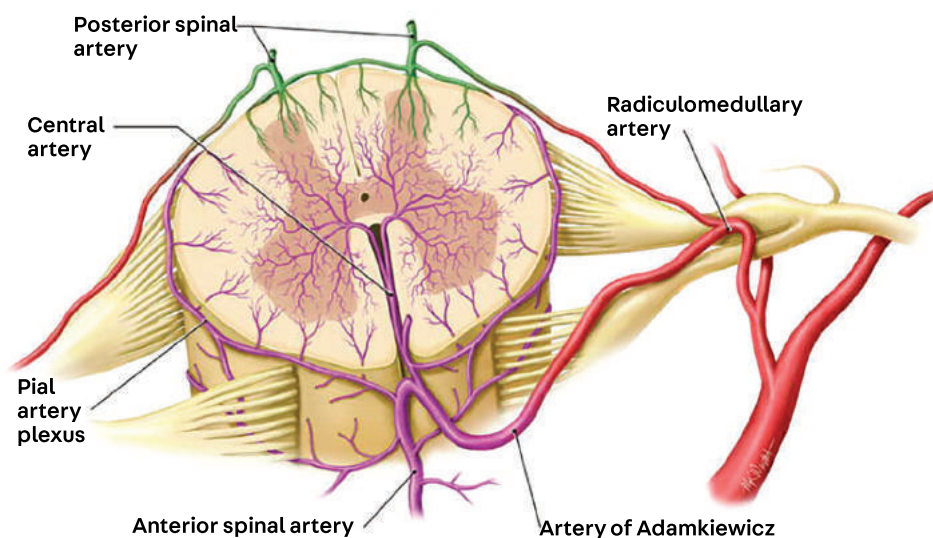


FIGURE 1-4

The arterial supply of the spinal cord.

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and/or motor symptoms in the limbs, particularly if concomitant sphincter disturbance is present. Patients may report stiffness in the legs or sustained (tonic) or rhythmic (clonic) spasm. Other clues include a history of neck or back pain associated with neurologic symptoms when there is extrinsic compression on the cord, such as due to a cervical or thoracic vertebral metastasis, but intrinsic spinal cord pathology typically does not have associated neck or spine pain. Bladder symptoms, sensory symptoms, and pain that precede the development of weakness favor an extrinsic (compressive) myelopathy over an intrinsic cord lesion. Particular care should be taken in patients with a history of known metastatic cancer or risk factors for cancer (eg, cigarette smoking) that may suggest myelopathy due to evolving metastatic spinal cord compression, which is a neurologic emergency.

The Uhthoff phenomenon occurs when patients with a demyelinating disease (such as multiple sclerosis [MS]) develop a recrudescence of symptoms attributable to a CNS demyelinating lesion with an increase in body temperature, such as when febrile, during exercise, or when ambient temperatures are hot (eg, in the shower). Other features that may be present include a feeling as if a tight

TABLE 1-1

Clues to a Spinal Cord Lesion From the History

General symptoms

- ◆ Bilateral greater than unilateral sensory and/or motor symptoms
- ◆ Bladder, bowel, and/or sexual dysfunction
- ◆ Stiffness in the legs
- ◆ Neck and/or back pain in association with neurologic symptoms, particularly if the patient had preceding trauma or if pain is exacerbated by neck flexion or extension
- ◆ Lhermitte or Uhthoff phenomenon
- ◆ Sensory level across the trunk (often more reliable as a symptom than a sign)
- ◆ Tight band around the trunk or torso
- ◆ Neurogenic claudication (suggests cauda equina pathology rather than a lower cord lesion)
- ◆ Sensory ataxia (can also occur with peripheral nervous system disorders)
- ◆ Dyspnea when lying flat (C3-C5 lesion)

Time course of symptoms

- ◆ **Acute**
 - ◇ Trauma
 - ◇ Vascular (infarction, hemorrhage)
- ◆ **Subacute**
 - ◇ Demyelination
 - ◇ Space-occupying lesion (eg, epidural abscess)
- ◆ **Chronic or progressive**
 - ◇ Primary or secondary progressive multiple sclerosis
 - ◇ Hereditary spastic paraparesis
 - ◇ Motor neuron disease (amyotrophic lateral sclerosis, primary lateral sclerosis)

band is around the chest or torso. When this is present in patients with MS, it is commonly referred to as the *MS hug*.

A careful history is also vital in trying to establish the level of the lesion in the spinal cord. Cervical cord lesions tend to affect the upper limbs but can affect all four limbs or, more rarely, just the lower limbs. When present, the Lhermitte phenomenon (defined as an electrical pain or tingling brought on by neck flexion that runs from the neck down the back and sometimes into the arms or legs) is a clue to cervical myelopathy most commonly due to a demyelinating lesion, but it may be encountered with other etiologies, including cervical spondylotic myelopathy. Cervical cord lesions can disrupt the origin of the phrenic nerves and cause diaphragmatic paralysis, hence the medical school mnemonic “C3, 4, 5 keeps the diaphragm alive.” Patients with diaphragmatic paralysis typically report dyspnea when lying flat, but severe cervical cord injury can result in respiratory failure¹¹ and severe autonomic dysfunction (refer to the section Spinal Shock Versus Neurogenic Shock). Thoracic lesions typically cause sensory and/or motor symptoms in the lower limbs.

Painful cramping or weakness in the legs that presents after walking a set distance and improves with rest (neurogenic claudication) is a feature of clinically significant lumbar spinal canal stenosis causing intermittent compression of cauda equina nerve roots; patients may report doing better when the spine is in flexion, which widens the lumbar canal (eg, when bent forward leaning over a shopping cart).¹⁰ Sensory ataxia is less specific to spinal cord pathology but is nevertheless a common manifestation of cord disease caused by dorsal column dysfunction and can arise from cervical, thoracic, or even lumbar cord or cauda equina lesions.

Ascending numbness with preserved (or increased) reflexes is usually secondary to myelopathy (most often sensory myelitis of MS) but is frequently mistaken for peripheral nervous system involvement. Indeed, if a patient mentions altered sensation or numbness from a particular level on the trunk that extends downward (often noticed in the shower or when swimming), it is a clue to a spinal sensory level.

Bladder and bowel dysfunction can also relate to lesions in the cervical, thoracic, or lumbar cord; conus medullaris; or cauda equina. Sphincter disturbance arises because normal voluntary voiding of the bowel and bladder relies on afferent feedback of a sense of fullness in these organs via the spinal cord to the brain. When this feedback is interrupted, a neurogenic bowel and bladder can develop. Spinal injury can lead to either a flaccid or spastic bladder, with a range of symptoms, including urinary frequency, urgency, incontinence, and urinary retention due to lack of coordination between the detrusor muscle of the bladder (which contracts during normal micturition) and the urinary sphincter (which relaxes during normal micturition).¹² Similarly, bowel dysfunction from spinal cord injury can lead to constipation or fecal urgency and incontinence with a dilated anal sphincter. Male and female sexual dysfunction often accompanies neurogenic bowel and bladder symptoms and commonly manifests as erectile dysfunction, retrograde ejaculation, and anorgasmia in males and vaginal dryness and anorgasmia in females.¹²

The demographic profile of the patient and a detailed history can also help to generate a relevant differential diagnosis list. Older patients are more likely to experience a spinal cord infarct than younger patients, and MS is 3 times more common in females than males.¹³ A history of trauma raises the possibility of a

KEY POINTS

- The Uhthoff phenomenon commonly occurs when patients with multiple sclerosis experience an exacerbation of symptoms with an increase in body temperature.
- Spinal injury can lead to either a flaccid or spastic bladder, with a range of symptoms, including urinary frequency, urgency, incontinence, and urinary retention due to a lack of coordination between the detrusor muscle of the bladder and the urinary sphincter.
- The patient's previous medical history and the temporal onset of neurologic symptoms can be used to narrow the differential diagnosis of a spinal cord lesion.

compressive myelopathy; therefore, a history of trauma to the neck or back preceding neurologic symptoms should always prompt consideration of spinal cord injury either directly (traumatic myelopathy) or indirectly (eg, spinal cord watershed infarction secondary to vertebral artery dissection).

The time course of myelopathic symptoms is also informative. Acute symptoms suggest trauma or a vascular cause, such as infarction or hemorrhage; subacute symptoms may suggest demyelination or an extrinsic compressive cause (eg, epidural abscess); and an insidious onset with chronic progression could indicate a progressive form of MS, hereditary spastic paraparesis, or primary lateral sclerosis.¹⁴ Relapsing symptoms could indicate MS or other

TABLE 1-2

Clues to a Spinal Cord Lesion From the Examination

Upper motor neuron signs

- ◆ Spasticity
- ◆ Hyperreflexia (including Hoffman sign and crossed adductor response)
- ◆ Clonus
- ◆ Pyramidal weakness
- ◆ Upgoing (extensor) plantar response

Paraparesis or quadriparesis

Inverted biceps or brachioradialis/supinator reflex

- ◆ Testing of the biceps or brachioradialis/supinator reflex (C5-C6) may lead to finger flexion instead of flexion of the elbow from loss of the biceps jerk (C5-C6) or brachioradialis (C5-C6) and spread to the finger flexors (C8); suggests a lesion at C5-C6 (lower motor neuron damage at the lesion and upper motor neuron damage below)

Involvement of multiple contiguous dermatomes

Absent abdominal reflexes (not always reliable)

Gait disturbance

- ◆ Stiff legs
- ◆ Circumduction
- ◆ Scissoring
- ◆ Sensory ataxia with positive Romberg sign (can also occur with peripheral nervous system disease)

Spinal shock or neurogenic shock

Spinal cord syndrome

- ◆ Posterior cord syndrome
- ◆ Central cord syndrome
- ◆ Anterior cord syndrome
- ◆ Brown-Séquard syndrome
- ◆ Transverse (complete or partial) myelitis
- ◆ Cauda equina syndrome
- ◆ Conus medullaris syndrome

inflammatory demyelinating disorders, such as aquaporin-4 IgG–seropositive neuromyelitis optica spectrum disorder (NMOSD) or myelin oligodendrocyte glycoprotein (MOG) IgG–associated demyelination.¹⁵

Purely motor symptoms might indicate spinal cord infarction, particularly if acute in onset, with a common etiology being embolization from the artery of Adamkiewicz to the anterior spinal artery affecting the ventral corticospinal tracts in the thoracic spine at a point usually between T9 and T12. Unsteadiness when the eyes are closed (eg, in the shower) is highly suggestive of dorsal column pathology such as from subacute combined degeneration of the spinal cord due to vitamin B₁₂ deficiency or copper deficiency. Fever in the context of neurologic symptoms referable to the spinal cord should initiate investigations for diskitis, spinal epidural abscess, or vertebral osteomyelitis, particularly if the patient has a history of systemic infection (eg, endocarditis) or is an IV drug user.

CLINICAL EXAMINATION FINDINGS

The neurologic examination is essential for lesion localization in a patient with a suspected myelopathy (**TABLE 1-2**).¹⁰ Typically, patients will have upper motor neuron signs, such as spasticity, hyperreflexia, and clonus, or pyramidal weakness characterized by weakness of shoulder abduction, elbow extension, wrist extension, and finger extension and the interossei in the upper limbs. Patients may also have weakness of hip flexion, knee flexion, and ankle dorsiflexion in the lower limbs. Upper motor neuron signs develop consequent to loss of descending inhibitory pathways in the spinal cord, leading to hyperexcitability.¹⁶ It is not uncommon to see young patients with hyperreflexia and nonsustained clonus without significant neurologic pathology, so these signs cannot always be relied upon as indicators of CNS disease; however, the likelihood of upper motor neuron pathology is increased if hyperreflexia occurs in the company of other upper motor neuron signs, such as an extensor plantar response or pyramidal weakness, or both. Occasionally, patients with spinal cord pathology will have a normal clinical examination (eg, asymptomatic spinal cord lesions in MS).

The reflexes most often assessed in the neurologic examination are the biceps jerks, triceps jerks, and supinator (brachioradialis) reflexes in the upper limbs and knee jerks, ankle jerks, and plantar responses in the lower limbs.¹⁷ Reflexes are graded as 0 or absent, 1+ when present but reduced, 2+ when normal, 3+ when brisker or more exaggerated than normal (hyperreflexia), and 4+ when hyperreflexia is accompanied by clonus. Clonus is a rhythmic involuntary movement most often elicited at the ankle, where a sudden sharp dorsiflexion of the ankle by the examiner results in rhythmic plantar flexion of the foot.¹⁶ Clonus may fatigue after a few beats (nonsustained) or occur indefinitely (sustained), with the latter usually indicating a more significant CNS insult.

Other reflexes can also be checked and are often helpful in providing further information if doubt exists regarding hyperreflexia, including deltoid and pectoral reflexes and finger jerks in the upper limbs and adductor reflexes in the lower limbs. A positive Hoffman sign (or reflex) occurs when the tip of the middle finger is flicked downward, causing the thumb of the hand to flex and adduct; this can indicate a cervical myelopathy.¹⁸ Spread of reflexes into the fingers may cause the fingers to flex when the biceps jerk or supinator reflex is being elicited in the setting of hyperactive finger flexors. An inverted brachioradialis/supinator reflex

KEY POINT

● The likelihood of upper motor neuron pathology is increased if hyperreflexia occurs accompanied by other upper motor neuron signs, such as an extensor plantar response or pyramidal weakness, or both.

(C5, C6) occurs when an attempt to elicit the biceps jerk leads to flexion of the fingers rather than elbow flexion because of a lesion at the C5 and/or C6 level. Injury to the C5 and/or C6 nerve root leads to an absent brachioradialis jerk (ie, no elbow flexion or supination), and spinal cord damage below that level leads to hyperactivity of the finger flexor (C8) jerks and finger flexion from spread when the adjacent brachioradialis tendon is struck.¹⁹ An inverted biceps reflex can similarly occur from a lesion at this location when loss of the biceps jerk is associated with spread to the hyperactive triceps, causing elbow extension rather than the expected flexion. Cervical spondylosis is the most common etiology of these inverted reflexes in the upper limb. The presence of a crossed adductor reflex (contraction of both hip adductors when testing a reflex at the ipsilateral knee) from either or both lower limbs is considered an upper motor neuron sign.

Superficial abdominal reflexes are cutaneous reflexes that can be tested in the four quadrants of the abdomen by stroking the skin (eg, with an orange stick) in the direction of the umbilicus, which causes reflex contractions of the abdominal wall. The upper abdominal reflexes are supplied by T9 through T11 and the lower reflexes by T11 and T12. The superficial abdominal reflexes are absent when a spinal cord lesion above the segmental level is present, but their clinical relevance is limited as it can be difficult to elicit abdominal reflexes in patients who are obese, have undergone abdominal surgery, are pregnant, or are elderly, and they may be absent in 15% of healthy individuals.²⁰ The Beever sign, which is also nonspecific, refers to upward displacement of the umbilicus when the neck is flexed on getting up from a reclining position; it occurs because of relative weakness of the lower abdominal muscles compared to the upper abdominal muscles.²¹ The Beever sign can indicate a thoracic cord lesion between T10 and T12 but is also seen in fascioscapulohumeral muscular dystrophy.

An extensor plantar response (positive Babinski reflex) is a major clue, particularly if bilateral, as it suggests involvement of the corticospinal tracts in the spinal cord, whereas an isolated unilateral extensor plantar response could be caused by either ipsilateral cord or contralateral brain pathology. The plantar response is elicited by gently scraping along the lateral border of the foot and then across the ball of the foot to the middle metatarsophalangeal joint; it is considered abnormal in adult patients when extension (rather than flexion) of the great toe is seen, with or without fanning of the other toes.²² It is worth mentioning that spinal cord pathology can still be present even with a flexor plantar response, so the sign is more helpful in localizing cord pathology when extensor rather than when flexor.

A dermatome is the area of skin supplied by the sensory nerve root of a single spinal segment (FIGURE 1-5).²³ Knowledge of the dermatomes can help the clinician establish the level of a lesion involving sensation in the nervous system. If a spinal cord lesion is suspected, a dedicated search to try to determine the spinal sensory level of the lesion should be conducted, including over the anterior and posterior trunk.¹⁰ Anatomic landmarks, such as the position of the nipples (which correspond to approximately the T4 dermatomal level) and the umbilicus (which corresponds to the T10 level), can greatly assist the clinician. Light touch, pinprick, and temperature sensation can all be tested over the trunk anteriorly or posteriorly. It is worth remembering that the C4 dermatome abuts the T2 dermatome on the chest. A spinal cord lesion affecting the spinothalamic tract(s) may cause anesthesia below the lesion. The most caudal dermatome of normal pain and temperature sensation on the trunk (truncal sensory level) occurs two

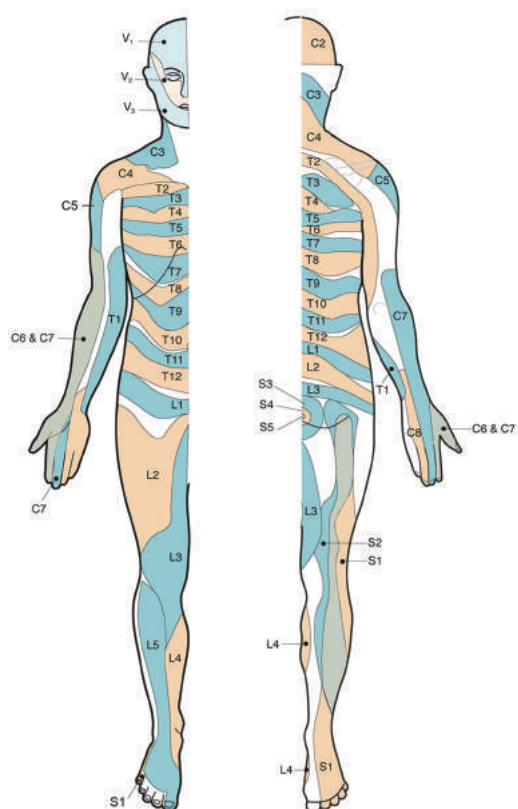


FIGURE 1-5

The human dermatomes. Evidence-based dermatome map representing the most consistent tactile dermatomal areas for each spinal dorsal nerve root found in most individuals, based on the best available evidence. The dermatomal areas shown are not autonomous zones of cutaneous sensory innervation since, except in the midline where overlap is minimal, adjacent dermatomes overlap to a large and variable extent. Blank regions indicate areas of major variability and overlap.

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in normal resting muscle tone arising from upper motor neuron pathology.¹⁶ The term *paresis* is used to denote weakness, whereas *plegia* is used to denote absence of any voluntary movement. Patients with a lesion involving the bilateral corticospinal tracts may be paraplegic (no voluntary movement in either leg) or quadriplegic/tetraplegic (have no movement in any of their four extremities) with severe cervical cord lesions. Patients with spinal cord disease may have a spastic paraparesis (with weakness but some preserved voluntary movement) and an accompanying spastic gait with a tendency for the legs to veer laterally from the midline during each stride (circumduction); they may also exhibit scissoring, in which the feet cross in front of each other during walking.¹⁹ A spastic monoparesis (weakness but some preserved voluntary movement) can occur when patients have unilateral corticospinal tract pathology, but contralateral pathology in the brain also should be considered.

to three vertebral segments below the level of the actual lesion in the spinal cord because the spinothalamic tracts ascend as they decussate. This anatomic arrangement means that a lesion affecting the spinothalamic tract of the right hemicord only will cause impaired temperature and pinprick sensation on the left trunk and lower limb below the level of the cord lesion. A sensory level is most reliable if the patient has described altered sensation at that level during the history. Involvement of multiple contiguous dermatomes tends to favor spinal cord pathology over radiculopathy, particularly if it occurs in the context of other upper motor neuron signs. A spinal motor level can be determined by ascertaining which muscles are affected by weakness and relating that to knowledge of the myotomes, which are defined as the group of muscles innervated by a single spinal nerve.

Patients with significant spinal cord pathology will often have an abnormal gait. As in other parts of the examination, the location of the lesion determines the way in which the gait will be abnormal. Spasticity is usually present and is defined clinically as a velocity-dependent increase

KEY POINTS

- The C4 dermatome abuts the T2 dermatome on the chest.
- A truncal sensory level is defined as the highest dermatomal area of normal sensation to pinprick and temperature on the trunk.
- A lesion affecting the spinothalamic tract of the right hemicord only will cause impaired temperature and pinprick sensation on the left trunk and lower limb two to three vertebral levels below the level of the cord lesion because the spinothalamic tracts ascend as they decussate.
- The term *paresis* is used to denote weakness, whereas *plegia* is used to denote absence of any voluntary movement.

Lesions involving afferent pathways in the cord, particularly the dorsal columns, give rise to a sensory ataxia; a positive Romberg sign (marked unsteadiness when standing with the feet together and the eyes closed) and pseudoathetosis (involuntary writhing movements more pronounced with eyes closed from loss of joint position sensation in the hands) may be seen.¹⁹

A coexistent stocking-glove peripheral neuropathy causing lower motor neuron signs (hyporeflexia or areflexia, wasting, fasciculations, hypotonia) can mask the upper motor neuron signs of a spinal cord lesion, so it may be appropriate to image the spinal cord in selected patients if a clinical possibility of a myelopathy exists, despite an absence of upper motor neuron signs.

SPINAL SHOCK VERSUS NEUROGENIC SHOCK

Spinal shock occurs when hyperacute or acute injury (particularly trauma) to the spinal cord results in flaccid areflexia below the level of the lesion, and the absence of upper motor neuron signs can lead to diagnostic confusion. The signs of spinal shock may last from days to weeks. Neurogenic shock occurs in acute trauma or occasionally in acute nontraumatic myelopathies above the level of T6, which leads to loss of sympathetic tone below the lesion causing hypotension and unopposed vagal activity leading to bradycardia.²⁴ The hypotension may require IV fluids, inotropes, and vasopressors or combinations thereof, whereas the bradycardia may require atropine or temporary pacing.

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia occurs when patients have injury above T6, leading to an exaggerated sympathetic nervous system response to sensory stimuli (eg, urinary retention, urinary tract infection, or constipation) below the level of injury. A reflex response causes a strong sympathetic response, leading to constriction of splanchnic blood vessels and arterial hypertension. Carotid baroreceptors trigger descending inhibitory signals to reduce sympathetic tone via the spinal cord, but a signal cannot reach most sympathetic outflow levels because of the cord lesion at or above T6. A further compensatory parasympathetic vagal response tries to reduce blood pressure by lowering the heart rate. In addition to hypertension and bradycardia, the excessive sympathetic tone below the lesion results in pale cool extremities with piloerection. Above the lesion, the parasympathetic activity results in pupil constriction, flushing, and sweating. Autonomic dysreflexia is managed by treating the offending stimulus.

SPINAL CORD SYNDROMES

Spinal cord syndromes are classic examination findings that can suggest the site or nature of a spinal cord lesion. (TABLE 1-2) (FIGURE 1-6²⁵).²⁶

Central Cord Syndrome

A central cord syndrome usually occurs as a result of a neck hyperextension cervical spinal cord injury, leading to damage to medial structures in the cord. A characteristic pattern of distal greater than proximal weakness in the upper limbs (predominantly the hands from damage to the anterior horn cells at that level) ensues, and the upper limbs are more affected than the lower limbs. The upper motor neuron pathology can lead to bladder dysfunction and priapism (in males). Classically, a capelike area of sensory loss to pain and temperature occurs over the upper trunk and arms at the level of the lesion as the decussating

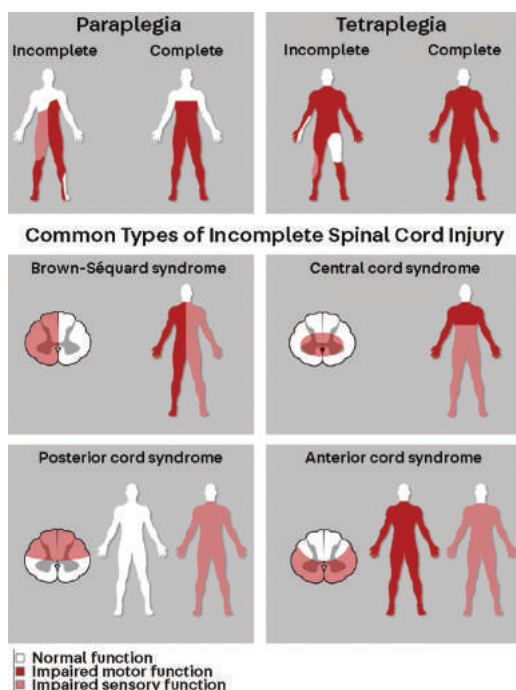


FIGURE 1-6

Spinal cord syndromes occurring from lesions above the conus medullaris. Paraplegia arises from lesions of the thoracic cord affecting motor pathways, and tetraplegia arises from similar lesions of the cervical cord. When transverse myelopathy or transection of the cord occurs, sensation below the level of the lesion is also affected. For simplicity, the other spinal cord syndromes are depicted as resulting from a cervical cord lesion.

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often, loss of pain and temperature sensation below the level of the lesion. Proprioception and vibration are preserved as the dorsal columns run posteriorly in the cord. Anterior cord syndromes classically occur with anterior spinal artery infarction, which is recognized as an iatrogenic complication resulting from cross clamping of the aorta intraoperatively.

Posterior Cord Syndrome

A posterior cord syndrome occurs when pathology of one or both dorsal columns of the spinal cord is present. The prototypic cause is vitamin B₁₂ deficiency, leading to subacute combined degeneration of the cord in which the lateral columns are also involved concurrently. Patients may present with sensory ataxia and have proprioceptive deficits on examination as well as impaired appreciation of vibration. In the case of vitamin B₁₂ deficiency, peripheral neuropathy, cognitive deficits, and megaloblastic anemia may also be seen. Other causes of posterior cord syndromes in the absence of a mass lesion or extrinsic compression on MRI include nitrous oxide abuse (causing vitamin B₁₂ deficiency), neurosyphilis, MS, and copper deficiency. For more information on the effects of vitamin

spinothalamic tract fibers are affected; this is called a suspended sensory level because the sensory loss is “suspended” or “hangs” on the trunk without involving the sacrum or lower limbs.²⁷ This is often seen with a central cord lesion due to syringomyelia (**CASE 1-1**). A patchy loss of pain, temperature, and light touch can be seen below the level of the lesion. Relative preservation of sacral and lower limb sensation occurs because the lower extremity spinothalamic tracts run more laterally than those of the upper extremities and so are not affected early. This finding can also be helpful in distinguishing an intraaxial lesion (within the cord) from an extraaxial lesion (intradural or extradural). Impairment of spinothalamic sensation without involvement of dorsal column sensation, as seen in this syndrome, is an example of dissociated sensory loss.

Anterior Cord Syndrome

In an anterior cord syndrome, damage to the ventral cord results in motor weakness and,

KEY POINTS

- Spinal shock occurs when hyperacute or acute injury (particularly trauma) to the spinal cord results in flaccid areflexia below the level of the lesion.
- Neurogenic shock occurs due to acute pathology above the level of T6, which leads to loss of sympathetic tone below the lesion causing hypotension and unopposed vagal activity leading to bradycardia.
- Autonomic dysreflexia occurs when patients have injury above the T6 level, leading to an exaggerated sympathetic nervous system response to sensory stimuli below the level of the lesion (eg, bladder filling).
- A central intraaxial spinal cord lesion often causes sensory symptoms and signs in the upper limbs and trunk before the lower limbs and sacral regions (called a suspended sensory level). This is because the lower extremity spinothalamic tracts run more laterally than those of the upper extremities and so take longer to be affected by an expanding central cord lesion.

deficiencies and nitrous oxide use, refer to the article “Metabolic and Toxic Myelopathies” by Natalie Elizabeth Parks, MD,²⁸ in this issue of *Continuum*.

Brown-Séquard Syndrome

Brown-Séquard syndrome refers to ipsilateral upper motor neuron motor weakness in one lower limb (and rarely proprioceptive loss) with spinothalamic pain and temperature loss in the contralateral lower limb. It is caused by a lesion that involves the lateral half of the cord, affecting the corticospinal, spinothalamic, and, sometimes, proprioceptive tracts on that side. Patients will have a truncal sensory level to pain and temperature on the contralateral side of the lesion two or three segments below the level of the lesion. Causes include MS, unilateral cord compression due to spinal degenerative disease, or hemitransection of the cord due to a knife injury or other trauma.

CASE 1-1

A 50-year-old woman presented with a 13-day history of sensory disturbance in the upper limbs, pain in the shoulders, and weakness in the hands following a flulike illness. She had no relevant past medical history.

Examination revealed mild weakness and wasting of the intrinsic muscle of the hands, with impaired temperature and pain sensation throughout the upper limbs and upper trunk in a capelike distribution. Upper limb reflexes were reduced. Lower limb motor and sensory examination was normal, and sacral function was intact. Lower limb reflexes were normal. Plantar responses were downgoing.

Routine blood tests and nerve conduction studies were normal. MRI of the cervical spine (FIGURE 1-7) showed slight descent of the cerebellar tonsils into the foramen magnum, with a cervical cord syrinx extending from C4 to T2. She underwent foramen magnum decompression surgery that resulted in improvement in her symptoms.

COMMENT

The differential diagnosis in this patient included an acquired demyelinating peripheral neuropathy such as Guillain-Barré syndrome because of the red herring of the flulike illness, distal sensory and motor symptoms, and reduced reflexes, at least in the upper limbs, but the wasting of the intrinsic muscles of the hands suggested a more chronic process. The downgoing plantar responses did not immediately implicate spinal cord pathology but the key was the capelike distribution of pain and temperature loss in the upper trunk, which would not be present in chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) and was more in keeping with a central cord syndrome. Bilateral involvement of the upper limbs and sparing of the lower limbs with sphincter preservation favored an intrinsic rather than an extrinsic cord lesion (keeping in mind that compressive myelopathies affecting the central cord can sometimes present with this pattern) and helped to localize the lesion to the cervical cord. The wasting in the hands and reduced reflexes in the upper limbs are not upper motor neuron signs but likely occurred in this case as a result of the large syrinx chronically impinging on the anterior horn cells.

Transverse Myelitis

The term *transverse myelitis* can result in confusion as by definition it should involve the entire transverse diameter of the spinal cord, yet it is often stratified clinically into complete and partial forms. A partial transverse myelitis (typically encountered in MS) refers to spinal cord inflammation in which symptoms and signs occur that are attributable to only a portion of the cord axially rather than involving the entire transverse diameter. A complete transverse myelitis (typically encountered with aquaporin-4 IgG-seropositive NMOSD) is attributable to spinal cord inflammation involving its entire cross section, usually resulting in severe bilateral sensory and motor dysfunction. Transverse myelitis also can be defined radiologically by the length of the T2-hyperintense lesion on sagittal sequences, which is probably more useful diagnostically than the clinical definition. Involvement may either affect a short segment of the cord (fewer

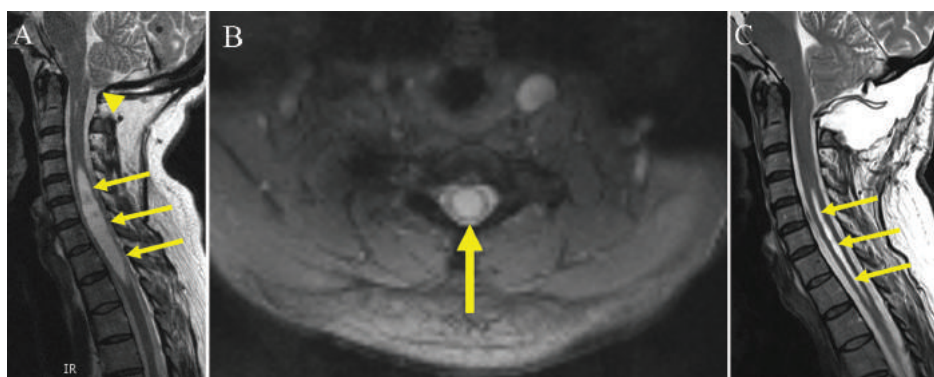


FIGURE 1-7

Imaging of the patient in [CASE 1-1](#), who presented with a central cord syndrome due to cervical syringomyelia. **A**, Sagittal T2-weighted MRI of the cervical spine shows cerebellar ectopia at the level of the foramen magnum (*arrowhead*) with a T2-hyperintense lesion of similar signal intensity to CSF in the central cord from C4 to T2 (*arrows*), consistent with a syrinx. **B**, Axial T2-weighted MRI confirms that the syrinx involves the central cord. **C**, Sagittal T2-weighted MRI of the cervical spine after foramen magnum decompression shows significant improvement in the caliber of the syrinx.

KEY POINTS

● A partial transverse myelitis refers to spinal cord inflammation in which symptoms and signs occur that are attributable to only a portion of the spinal cord in cross section rather than involving the entire transverse diameter. A complete transverse myelitis is attributable to spinal cord inflammation involving its entire cross section.

● Patients with a longitudinally extensive transverse myelitis should be tested for aquaporin-4 IgG and myelin oligodendrocyte glycoprotein IgG.

than three vertebral segments in length) on sagittal images, which is typical of MS, or may be longitudinally extensive (extending three or more vertebral segments in length), which is characteristic of aquaporin-4 IgG-seropositive NMOSD.^{13,15} A variety of causes of transverse myelitis exist. Some have argued for a new classification of spinal cord inflammatory disorders that removes the term transverse from its definition as a large proportion of inflammatory myelopathies do not involve the entire transverse dimension of the spinal cord. For more information on transverse myelitis, refer to the article “Myelitis and Other Autoimmune Myelopathies” by Sebastian Lopez Chiriboga, MD, and Eoin P. Flanagan, MBBCh,²⁹ in this issue of *Continuum*.

Cauda Equina Syndrome

Cauda equina syndrome refers to distal greater than proximal weakness and sensory disturbance in the lower limbs accompanied by impairment of sphincter control, including weakness of the anal sphincter, with areflexia of the ankle jerks but preserved knee jerks. Patients may report saddle anesthesia. The most common cause is compression of the cauda equina by a structural lesion, such as a prolapsed lumbosacral disk or tumor, but the syndrome can occur due to infiltration of the cauda equina by neoplasm or involvement by infection or granulomatous disease. For more information on cauda equina syndrome, refer to the article “Disorders of the Cauda Equina” by Samantha LoRusso, MD,³⁰ in this issue of *Continuum*.

Conus Medullaris Syndrome

The upper level of the conus medullaris is not well defined. Although the corresponding spinal cord segments of the conus medullaris are typically from S1 through S5, the classic conus medullaris syndrome usually encompasses lesions of the cord as rostral as L2; therefore, the clinical picture is of varying degrees of mixed upper and lower motor neuron signs in both lower limbs. The patient may report back pain, and the examination may reveal distal greater than proximal weakness, brisk knee jerks, and absent ankle jerks, with saddle and lower limb numbness; bladder, bowel, and sexual dysfunction is particularly common in these patients.

CONCLUSION

When a patient reports neurologic symptoms that are potentially attributable to the spinal cord or cauda equina, a careful history and examination may provide numerous clinical clues that, together with a thorough knowledge of spinal neuroanatomy, can assist with lesion localization and suggest a differential diagnosis.

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