CHAPTER 104 Spinal Cord Disorders

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PERSPECTIVE

Spinal cord disorders encompass a wide range of pathologic entities and affect all age groups. Some spinal cord disorders may have catastrophic outcomes if not recognized early in the clinical course. The ultimate neurologic outcome with many of these disorders may depend on expeditious recognition in the emergency department (ED), with appropriate initial investigations, neuroimaging, management, and consultation for definitive therapy. Diagnosis of these disorders may be extremely challenging, and certain disorders may mimic other disease processes until late in the clinical course, when there is clear neurologic impairment. As with many disease processes affecting the nervous system, correct diagnosis and appropriate management require knowledge of the anatomic organization of the spinal cord and skill in taking the history and in performing the neurologic examination.

This chapter generally is concerned with processes affecting the spinal cord and its vascular supply, as well as processes compressing the spinal cord. Direct trauma and mechanical instability of the spinal column are discussed in Chapter 40.

PRINCIPLES OF DISEASE

Anatomy

In adults, the spinal cord is approximately 40 cm long and extends from the foramen magnum, where it is continuous with the medulla oblongata, to the body of the first or second lumbar vertebra. Similar to the brain, the spinal cord is covered by three meningeal layers: the inner pial layer, the arachnoid, and the outer dural layer. At its lower end, the spinal cord tapers into the conus medullaris, where several segmental levels are represented in a small area. The lumbar and sacral nerve roots form the cauda equina as they descend caudally in the thecal sac before exiting the spinal canal at the respective foramina. The non-neural filum terminale runs from the tip of the conus and inserts into the dura at the level of the second sacral vertebra.

Two symmetrical enlargements of the spinal cord contain the segments that innervate the limbs. The *cervical enlargement* (cord level C5 to T1) gives rise to the brachial plexus and subsequently to the peripheral nerves of the upper extremity. The *lumbar enlargement* (L2 to S3) gives rise to the lumbosacral plexus and peripheral nerves of the lower extremity. The space surrounding the spinal cord within the spinal canal is reduced in the area of the enlargements, potentially leaving the cord more vulnerable to compression in these regions. At each segmental level, anterior (ventral) and posterior (dorsal) roots arise from rootlets along the anterolateral and posterolateral surfaces of the cord. At each level, the anterior root conveys the outflow of the motor neurons in the anterior horn of the spinal cord, and the posterior root contains sensory neurons and fibers that convey sensory inflow.

The arterial supply of the spinal cord is derived primarily from two sources. The single anterior spinal artery arises from the paired vertebral arteries. This anterior spinal artery runs the entire length of the cord in the midline anterior median sulcus and supplies roughly the anterior two thirds of the spinal cord. Blood supply to the posterior third of the spinal cord derives from the smaller paired posterior spinal arteries. The anterior and the posterior spinal arteries receive segmental contributions from radicular arteries, the largest being the radicular artery of Adamkiewicz, which typically originates from the aorta between T8 and L4. The venous drainage of the cord largely parallels the arterial supply.

The internal anatomy of the spinal cord is divided into central gray matter, which contains cell bodies and their processes, and surrounding white matter, where the ascending and descending myelinated fiber tracts are located. These fiber tracts are organized into discrete bundles, with the ascending tracts conveying sensory information and the descending tracts conveying the efferent motor impulses and visceral innervation.

For clinical purposes, neuroanatomy of the spinal cord may be greatly simplified, as depicted in Figure 104-1. Major ascending sensory tracts are represented on the right side of the figure, with motor tracts on the left side. The posterior columns carry afferent ascending proprioceptive and vibratory information on the ipsilateral side of the cord to the area stimulated; decussation of these fibers occurs in the medulla so that contralateral cortical representation ultimately occurs. In a portion of the lateral column of white matter, the lateral spinothalamic tract conveys afferent information about pain and temperature. (Tracts are named with their point of origin first—the spinothalamic tract, for example, arises in the spinal cord and travels to the thalamus.) The tract is laminated so that sacral fibers are represented most laterally. Crossing of fibers from this tract occurs near the level of entry of the spinal nerve; a cord lesion affecting one lateral spinothalamic tract results in decreased or absent pain and temperature perception below the level of injury on the contralateral side of the body.

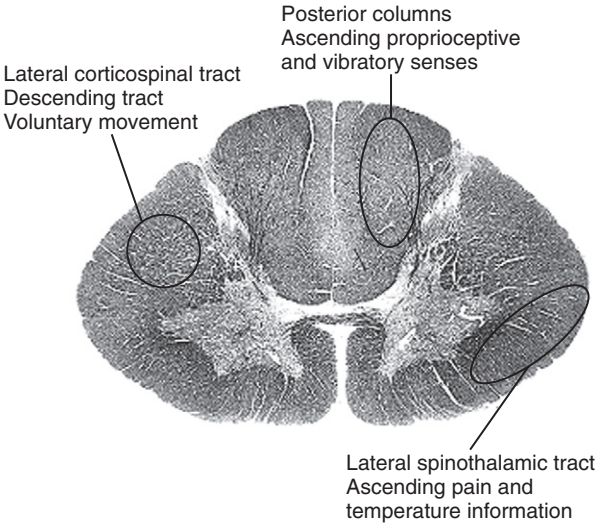


Figure 104-1. Simplified spinal cord anatomy showing clinically essential motor and sensory tracts. (Photomicrograph courtesy of John Sundsten, Digital Anatomist Project, University of Washington.)

For clinical purposes, the major descending motor tract is represented in the lateral corticospinal tract (which, as the name implies, originates in the cortex and flows toward the spinal cord). This tract also is anatomically organized, with efferent motor axons to the cervical area located medially and the sacral efferent axons located laterally. Decussation of this descending tract occurs in the medulla. The cell bodies of the lower motor neurons (anterior horn cells) are in the ventral portion of the gray matter of the spinal cord.

CLASSIFICATION OF SPINAL CORD SYNDROMES

The anatomic organization of the spinal cord lends itself to a corresponding anatomic-pathophysiologic classification of cord dysfunction. Any of the different anatomic syndromes may be the final clinical picture of a variety of clinical processes either intrinsic or extrinsic to the spinal cord. The syndromes frequently exist in partial or incomplete forms.

Complete (Transverse) Spinal Cord Syndrome

Complete spinal cord lesions may occur as either acute or subacute pathologic processes. A *complete* spinal cord lesion is defined as a total loss of sensory, autonomic, and voluntary motor innervation distal to the spinal cord level of injury. Reflex responses mediated at the spinal level, such as muscle stretch ("deep tendon") reflexes, may persist, although they also may be absent or abnormal. Autonomic dysfunction may manifest with hypotension (neurogenic shock) or priapism. The most common cause of the complete transverse cord syndrome is trauma, although this anatomic syndrome is nonspecific as to etiology.[1,2] Other causes of acute complete cord syndrome include infarction, hemorrhage, and entities causing extrinsic compression. Of patients in whom complete transverse syndromes develop and persist for more than 24 hours, functional recovery does not occur in 99%.[3,4] Any evidence of cord function below the level of injury denotes a partial rather than a complete lesion. Signs such as persistent perineal sensation ("sacral sparing"), reflex rectal sphincter tone or voluntary rectal sphincter contraction, or even slight voluntary toe movement suggest a partial cord lesion, which usually carries a better prognosis than a complete lesion.[1]

Spinal shock refers to the loss of muscle tone and reflexes with complete cord syndrome during the acute phase of injury. The intensity of the spinal shock increases with affected spinal cord level.[5] Spinal shock typically lasts less than 24 hours but has been reported occasionally to last days to weeks.[5,6] A marker of spinal shock is loss of the bulbocavernosus reflex, which is a normal cord-mediated reflex that also may be preserved in complete cord lesions. The bulbocavernosus reflex involves involuntary reflex contraction of the anal sphincter in response to a squeeze of the glans penis or a tug on the Foley catheter. The termination of the spinal shock phase of injury is heralded by the return of the bulbocavernosus reflex; increased muscle tone and hyper-reflexia follow later.[1,5,6]

Incomplete Spinal Cord Lesions

Incomplete spinal lesions are characterized by preservation of function of various portions of the spinal cord. Of all incomplete spinal lesions, most can be classified generally as one of three clinical syndromes: central cord syndrome, Brown-Séquard syndrome, or anterior cord syndrome (Table 104-1).

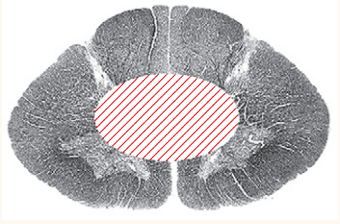
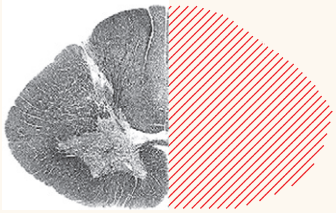
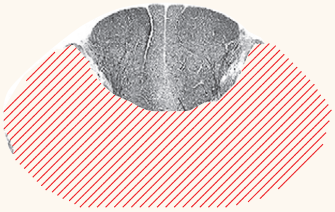
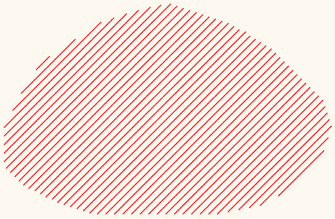
Central Cord Syndrome

Central cord syndrome, first described by Schneider and colleagues in 1954, is the most prevalent of the partial cord syndromes.[7,8] Because of the anatomic organization of the spinal cord, a central cord injury is characterized by bilateral motor paresis, with upper extremities affected to a greater degree than lower extremities, and distal muscle groups affected to a greater degree than proximal muscle groups. Sensory impairment and bladder dysfunction are variable features. At times, burning dysesthesias in the upper extremities may be the dominant feature.[9] Central cord injury affects the central gray matter and the central portions of the corticospinal and spinothalamic tracts. It is caused most often by a hyperextension injury, with the postulated pathomechanism being squeezing or pinching of the spinal cord anteriorly and posteriorly by inward bulging of the ligamentum flavum. The most common cause of such injuries is a fall, followed in frequency by a motor vehicle crash.[7,8] The result is contusion to the spinal cord, with the central portion being most affected. This injury classically occurs in elderly individuals with degenerative arthritis and spinal stenosis in the cervical area, but may affect any patient with cervical canal narrowing of any etiology (e.g., congenital narrow canal as seen in achondroplasia or canal narrowing from disk protrusion or tumor). The prognosis with central cord syndrome depends on the degree of injury at presentation and patient age.[10,11] In patients younger than 50 years of age, more than 80% regain bladder continence, and approximately 90% return to ambulatory status. In patients older than 50, only 30% regain bladder function, with approximately 50% regaining the ability to ambulate.[11]

Brown-Séquard Syndrome

Brown-Séquard syndrome, first described in 1846 by the one physician for whom it is named,[12] is the result of an anatomic or functional hemisection of the spinal cord. Usually associated with penetrating injuries,[13] Brown-Séquard syndrome also may be seen with compressive or intrinsic lesions. The syndrome has been reported in association with spinal cord tumors, spinal epidural hematoma, vascular malformations, cervical spondylosis, degenerative disk disease, and radiation injury and as a

Table 104-1 Spinal Cord Syndromes

SYNDROME	SENSORY	MOTOR	SPHINCTER INVOLVEMENT
Central cord syndrome 	Variable	Upper extremity weakness, distal > proximal	Variable
Brown-Séquard syndrome 	Ipsilateral position and vibration sense loss Contralateral pain and temperature sensation loss	Motor loss ipsilateral to cord lesion	Variable
Anterior cord syndrome 	Loss of pin and touch sensation Vibration, position sense preserved	Motor loss or weakness below cord level	Variable
Transverse cord syndrome—complete 	Loss of sensation below level of cord injury	Loss of voluntary motor function below cord level	Sphincter control lost
Conus medullaris syndrome	Saddle anesthesia may be present, or sensory loss may range from patchy to complete transverse pattern	Weakness may be of upper motor neuron type	Sphincter control impaired
Cauda equina syndrome	Saddle anesthesia may be present, or sensory loss may range from patchy to complete transverse pattern	Weakness may be of lower motor neuron type	Sphincter control impaired

complication of spinal instrumentation.[13] The syndrome in its pure form is characterized by ipsilateral loss of motor function and proprioception or vibration, with contralateral loss of pain and temperature sensation, below the spinal cord level of injury. Because fibers associated with the lateral spinothalamic tract ascend or descend one or two spinal cord segments before crossing to the contralateral side, ipsilateral anesthesia (pain and temperature modalities) may be noted one or two segments above the lesion, although this observation is variable. Most patients with Brown-Séquard syndrome incur only partial sensory and motor impairment, and the classic pattern is not seen.[11,13,14] Brown-Séquard syndrome carries the best prognosis of any of the incomplete spinal cord syndromes. Fully 80 to 90% of patients with Brown-Séquard syndrome regain bowel and bladder function, 75% regain ambulatory status, and 70% become independent in their activities of daily living.[11]

Anterior Cord Syndrome

Anterior cord syndrome is characterized by loss of motor function, pinprick, and light touch below the level of the lesion, with preservation of posterior column function, including some touch, position, and vibratory sensation. Although most reported cases of anterior spinal syndrome follow aortic surgery,[15] the syndrome also may occur after severe hypotension, infection, myocardial infarction, vasospasm from drug reaction, and aortic angiography.[16] The anatomic lesion may be the result of a cervical hyperflexion injury resulting in a

cord contusion or from protrusion of bony fragments or herniated cervical disk material into the spinal canal. Rarely, it is produced by laceration or thrombosis of the anterior spinal artery or a major radicular feeding vessel.[11] Patients present with the characteristic neurologic findings noted earlier. Functional recovery varies; most improvement occurs over the first 24 hours, but little improvement is expected thereafter.[4] Although anterior cord lesions from ischemia usually are incomplete, patients without motor function at 30 days have little or no likelihood of regaining any motor function by 1 year.[15,17] Overall, only 10 to 20% of patients with this entity regain some muscle function, and even in this group, there is little power or coordination.[11]

Conus Medullaris Syndrome and Cauda Equina Syndromes

The separation of conus medullaris and cauda equina lesions in clinical practice is difficult because the clinical features of the disorders overlap. Additionally, a combined lesion may occur that masks clear clinical symptoms or signs of either an upper or a lower motor neuron type of injury. The conus medullaris is the terminal end of the spinal cord, located at approximately the L1 level in adults. The conus medullaris syndrome may involve disturbances of urination (usually from a denervated, autonomic bladder that manifests clinically with overflow incontinence) and sphincter impairment or sexual dysfunction. Sensory involvement may affect the sacral and coccygeal segments, resulting in saddle anesthesia. Pure lesions of the conus medullaris are rare.[18] Upper motor neuron signs, such as increased motor tone and abnormal reflexes, may be present, but their absence does not exclude the syndrome. The conus medullaris syndrome can be caused by central disk herniation, neoplasm, trauma, or vascular insufficiency. Because the conus is such a small structure, with lumbar and sacral segments represented in a small area, a lesion usually causes bilateral symptoms. This finding may help distinguish lesions of the conus from lesions of the cauda equina, which often are unilateral.[18]

The *cauda equina* (Latin for "horse's tail") is the name given to the lumbar and sacral nerve roots that continue on within the dural sac caudal to the conus medullaris. Not a true "cord syndrome," cauda equina syndrome represents dysfunction at the level of nerve roots, but the anatomic clustering of nerve roots with the lumbar dural sac allows injury to several nerve roots to occur simultaneously. The etiologic lesion in the cauda equina syndrome usually is a midline rupture of an intervertebral disk, most commonly at the L4-5 level. Tumors and other compressive masses also may cause the syndrome. As in the conus medullaris syndrome, patients generally present with progressive symptoms of fecal or urinary incontinence, impotence, distal motor weakness, and sensory loss in a saddle distribution. Muscle stretch reflexes also may be reduced. The presence of urinary retention is the most consistent finding, with a sensitivity of 90%.[19] Low back pain may or may not be present.

■ CLINICAL FEATURES

History

Weakness, sensory abnormalities, and autonomic dysfunction are the cardinal manifestations of spinal cord dysfunction. The tempo and degree of impairment often reflect the disease process. Past medical history is vital because a history of coagulopathy or other systemic processes may be elicited. A history of cancer should suggest the possibility of metastatic disease. Recent trauma raises the possibility of vertebral fracture or disk protrusion.

Physical Examination

The physical examination pertinent to spinal cord dysfunction involves testing in three areas: (1) motor function, (2) sensory function, and (3) reflexes. Each component is best tested with the anatomic organization of the spinal cord in mind to help determine the level of the spinal cord dysfunction.

Motor Function

Testing of motor function encompasses examination of muscle bulk, tone, and strength. Muscle bulk is easily examined in large motor groups, such as the thigh or calf muscles, the biceps, or the triceps. Inspection of the intrinsic hand muscles also may be helpful for determining muscle bulk; wasting may be evident as hollowed or recessed regions of the hand. Decreased mass, asymmetry, or fasciculations should be noted. Tone is tested with repeated passive knee, elbow, or wrist flexion, with the examiner feeling for abnormally increased or decreased resistance. Rapid pronation-supination of the forearm is another useful method to assess tone. Increased tone may indicate spasticity or an upper motor neuron lesion, whereas decreased tone corresponds with lower motor neuron, motor endplate, or muscular problems. Finally, motor strength is graded in the upper and the lower extremities. Motor grading for the neurologic examination is relatively straightforward. Scored on a scale of 0 to 5, neuromuscular functioning is graded as follows:

0: No firing of the muscle is present.
1: The muscle fires but is unable to move the intended part.
2: The muscle is able to move the intended part with gravity eliminated.
3: The muscle is able to move the intended part against gravity.
4: The muscle is able to move the intended part, but not at full strength.
5: Full muscular strength is present.

A rectal examination is performed to assess voluntary sphincter contraction, resting tone, and, as described previously, the bulbocavernosus reflex.

Sensory Function

Sensory testing requires a cooperative patient and an attentive examiner. The spinal cord–related modalities that may be clinically useful in the ED setting include testing for pinprick, light touch (contralateral lateral spinothalamic tract), and proprioception (ipsilateral posterior column). Assessment of the patient's response to pinprick, light touch, and proprioception in all four extremities is necessary if a neurologic injury is suspected. Testing of sacral dermatomes may be an important part of the examination in some patients. As previously noted, sacral sparing is an important finding indicating that spinal cord dysfunction may be incomplete. The sensory fibers from sacral dermatomes are more peripherally located in the ascending fiber bundles; central or partial cord lesions may ablate sensation in the extremities yet allow some perception of sensation in the sacral area.

Reflexes

Muscle stretch ("deep tendon") reflexes may be tested rapidly at the bedside. Responses are graded on a scale of 0 to 4+, with

2 being normal. Hyperactive reflexes suggest upper motor neuron disease (affecting the neurons or their outflow from the brain or spinal cord), as do sustained clonus and a Babinski's sign. The absence of these reflex changes does not constitute evidence that a myelopathy is not present. In fact, one small series noted a low incidence of extensor planar responses, as well as a lack of hyper-reflexia, in patients presenting to the ED with acute or progressive cord compression or myelopathies.[20] Reflexes also may be diminished or absent when sensation is lost, or when spinal shock is present, or when lower motor neuron disease is present. Diseases of muscles or neuromuscular junctions also may decrease reflexes. In acute cord injury, reflexes may be diminished in the acute phase. The bulbocavernosus reflex may be helpful in this assessment.

DIAGNOSTIC STRATEGIES

Historical or physical examination findings that suggest spinal cord dysfunction prompt further investigations. The basic strategy is to detect or exclude extrinsic compressive lesions or other potentially treatable entities. Magnetic resonance imaging (MRI) has changed the diagnostic approach to patients with suspected spinal cord dysfunction. Plain radiographs and computed tomography (CT) scans may show bone and some soft tissue abnormalities. Conventional radiographs and CT scans are required in patients with trauma or suspected bone involvement by tumor or degenerative processes, but MRI shows many of these abnormalities and defines the spinal cord as well as the soft tissue structures associated with it. Tissue damage patterns within the cord, such as hemorrhage and edema, also may be detected with MRI. CT myelography may be able to answer some of these questions in patients in whom implanted metal precludes MRI but generally does not yield the same level of detail. After imaging studies exclude compressive lesions or other masses affecting the spinal cord, the possibility of inflammatory or demyelinating disorders remains, and lumbar puncture may be useful in diagnosis.

DIFFERENTIAL CONSIDERATIONS

The prime principle in management of spinal cord dysfunction is to consider and exclude potentially treatable problems. The clinical assessment of spinal cord dysfunction is limited to detecting weakness, sensory alterations, sphincter dysfunction, and perhaps reflex abnormalities. Pain in the back may be present depending on the pathologic process but generally is not helpful in formulating a list of considerations for the differential diagnosis. Because potential functional loss and impact on quality of life are great, the detection of a process for which some intervention is possible assumes great importance. A likely diagnosis of spinal cord infarction may be entertained, but the pursuit of a treatable process, such as spinal cord compression from an epidural hematoma, should be seriously considered.[21] This discovery process may involve specialty consultation or obtaining studies not readily available in many ED settings, such as MRI. As a general rule, liberal use of consultation and imaging is recommended when the possibility of spinal cord dysfunction is considered. The history may suggest a specific cause and will guide the tempo of investigation. The caveat is that spinal cord diseases may mimic many other disease processes, and neither the history nor physical examination may allow diagnosis until appreciable neurologic dysfunction has developed.

The picture of a *complete* transverse spinal cord syndrome with paraplegia, sensory loss at a clear anatomic level, and sphincter dysfunction cannot be fully simulated by other anatomic lesions. *Incomplete* or evolving spinal cord syndromes may be imitated by other disease processes. It is always prudent to focus the differential diagnosis on anatomic considerations—the classic "where is the lesion?" approach (Table 104-2). Progressive lower extremity weakness and sensory alteration may represent cord dysfunction but could reflect an intracranial vertex mass with bilateral cortical dysfunction. Ataxia may be a finding in cerebellar disease but also has rarely been reported as an isolated finding with spinal cord compression. Another example is rapidly progressive paralysis in a patient with areflexia and quadriplegia; ascending paralysis (Landry-Guillain-Barré syndrome) at times may mimic an acute cord lesion.

Generally, pathologic processes involving the spinal cord may be divided into processes affecting the cord or its blood supply primarily, such as demyelination, infection, or infarction, and processes that compress the cord, most often originating outside the dura (Box 104-1). *Myelitis* is a comprehensive term for spinal cord inflammation with dysfunction, and the potential causes are legion. The clinical presentation often is similar across the variety of entities that may cause cord compression. The tempo of the process may yield a different clinical picture. In chronic compression, muscle wasting and abnormal reflexes may be present, whereas both of these may be lacking in acute compression. A neurologic deficit in concert with back pain strongly suggests a spinal cord lesion, necessitating prompt investigation to identify a specific cause. Atypical presentations for these lesions are the rule, and additional diagnostic studies should be pursued as appropriate.

Table 104-2 Clinical Characteristics of Neuromuscular Diseases

	HISTORY	STRENGTH	DTR	SENSATION	WASTING
Myelopathy	Trauma, infection, cancer	Normal to decreased	Increased	Normal to decreased	No
Motor neuron disease (ALS)	Progressive difficulty with swallowing, speaking, walking	Decreased	Increased	Normal	Yes
Neuropathy	Recent infection Ascending weakness	Normal or decreased Distal > proximal	Decreased	Decreased	Yes
Neuromuscular junction disease	Food (canned goods) Tick exposure Easy fatigability	Normal to fatigue	Normal	Normal	No
Myopathy	Thyroid disease Previous similar episodes	Decreased Proximal > distal	Normal	Normal	Yes

ALS, amyotrophic lateral sclerosis; DTR, deep tendon reflex.

BOX 104-1 NONTRAUMATIC ETIOLOGIES OF SPINAL CORD DYSFUNCTION

Processes Affecting the Spinal Cord or Blood Supply Directly
- Multiple sclerosis
- Transverse myelitis
- Spinal arteriovenous malformation/subarachnoid hemorrhage
- Syringomyelia
- HIV myelopathy
- Other myelopathies
- Spinal cord infarction

Compressive Lesions Affecting the Spinal Cord
- Spinal epidural abscess
- Spinal epidural hematoma
- Diskitis
- Neoplasm
  - Metastatic
  - Primary CNS

HIV, human immunodeficiency virus; CNS, central nervous system.

MANAGEMENT

Just as the clinical manifestations of spinal cord dysfunction are nonspecific with respect to etiology, the treatment for many of the disease entities often is nonspecific. Steroid administration has been recommended as therapy in spinal cord trauma, although this use of steroids has been seriously questioned in the literature.[22-24] Steroids also have been used with many nontraumatic causes of cord compression, despite the lack of rigorous clinical studies supporting this use. Radiation treatment is recommended for cord compression by tumor. Surgical consultation for decompression may be considered, although the indications for surgery and timing of surgery are controversial.

A specific diagnosis is needed to guide therapy. Accordingly, involvement of appropriate consultants and discussion of what may be understudied therapies are suggested.

SPECIFIC DISEASE PROCESSES

As noted earlier, spinal cord disorders may be grouped into lesions resulting from processes intrinsic to the cord and vasculature and lesions causing extrinsic compression. The order of the following discussion roughly corresponds with the organization of Box 104-1.

Intrinsic Cord Lesions

Multiple Sclerosis

Principles of Disease. *Demyelination* denotes a disease process with the prominent feature of partial or complete loss of the myelin surrounding the axons of the central nervous system. Multiple sclerosis (MS) is the most common example of such a process; spinal cord involvement may dominate the clinical picture.

Clinical Features. Central nervous system lesions that are "scattered in time and space" are the hallmark of MS. The demyelinated segments do not transmit action potentials normally, resulting in a wide variety of spinal cord–related abnormalities, depending on the location and extent of the demyelination. In addition to patchy motor and sensory deficits, patients with MS may complain of bladder dysfunction or tremor or demonstrate evidence of a transverse partial or complete cord syndrome mimicking a compressive spinal lesion.[25,26] The history may include a previous episode of optic neuritis or transient visual problems. Spinal cord lesions in MS primarily involve the lateral corticospinal tracts, the posterior columns, and the lateral spinothalamic tracts. Motor system dysfunction is the most frequent manifestation of MS involvement of the spinal cord, usually as a result of lesions in the lateral corticospinal tracts.

The examination of patients with MS often reveals paresis, increased muscle tone, hyper-reflexia, clonus, and a Babinski's response. Spinal cord involvement also may result in dysautonomias.

Diagnostic Strategies. Spinal MRI is the diagnostic imaging modality of choice because it can exclude cord compression and show lesions suggesting MS.[26-28] Cranial MRI may be helpful in showing other central nervous system lesions. Cerebrospinal fluid (CSF) testing for myelin basic protein and oligoclonal bands also is a diagnostic option, but no CSF abnormalities are entirely specific for MS.[29,30] Oligoclonal bands in the CSF may aid in the diagnosis, but they are significant only if not present in the serum as well.[29]

Differential Considerations. Considerations in the differential diagnosis include systemic lupus erythematosus, Lyme disease, neurosyphilis, human immunodeficiency virus (HIV) myelopathy, and other disorders.

Management. MS exacerbations may be treated with high-dose methylprednisolone followed by a tapering dose of prednisone. Corticosteroids have been shown to be useful in shortening the time required for recovery from an exacerbation of MS.[26] Consultation and referral to a neurologist usually are indicated. Immunosuppressive therapy in patients with the chronic progressive form of the disease has met with variable success.[25,26] Because numerous disorders can mimic MS, the definitive diagnosis of this disease usually is not made in the ED.[31]

Transverse Myelitis

Principles of Disease. *Acute transverse myelitis* refers to acute or subacute spinal cord dysfunction characterized by paraplegia, a transverse level of sensory impairment, and sphincter disturbance. It is relatively rare, with a reported annual incidence of 1 case per 1.3 million population. The presentation may be mimicked by compressive lesions, trauma, infection, or malignant infiltration. The exact pathogenesis is unknown, although it is noted to follow viral infection in approximately 30% of patients and commonly is termed "postinfectious myelitis."[32] Other postulated etiologic categories include infectious, autoimmune, and idiopathic.[33,34] No apparent cause for acute transverse myelitis is identified in 30% of the patients.[32] Progression of symptoms usually is rapid, with 66% of the cases reaching maximal deficit by 24 hours.[35] Symptoms may progress, however, over days to weeks. The thoracic cord region is affected most often by this process (60 to 70%)[35]; the cervical spinal cord is rarely affected.[36]

Clinical Features. In addition to motor, sensory, and urinary disturbances, patients with acute transverse myelitis may complain of back pain and may have low-grade fever, raising concern for spinal epidural abscess. As with MS, the examination may reveal weakness progressing to paresis, hypertonia, hyper-reflexia, clonus, and a Babinski's response. Spinal cord involvement also can result in dysautonomias.

Diagnostic Strategies. Evaluation for acute transverse myelitis is done primarily with emergent MRI to exclude compressive