**TABLE 8.1** (*continued*)

| NERVE | MUSCLE(S) | FUNCTION OF THE MUSCLE(S) | NERVE ROOTS[a] |
|---|---|---|---|
| | Flexor hallucis longus | Flexes great toes, aids plantar flexion | L5, **S1**, **S2** |
| | Small foot muscles | Cup sole | S1, S2 |
| Superficial peroneal nerve | Peroneus longus | Plantar flexes and everts foot | L5, S1 |
| (branch of sciatic nerve) | Peroneus brevis | Plantar flexes and everts foot | L5, S1 |
| Deep peroneal nerve | Tibialis anterior | Dorsiflexes and inverts foot | L4, L5 |
| (branch of sciatic nerve) | Extensor digitorum longus | Extends phalanges, dorsiflexes foot | **L5**, S1 |
| | Extensor hallucis longus | Extends great toe, aids dorsiflexion | **L5**, S1 |
| | Peroneus tertius | Plantar flexes foot in pronation | L4, **L5**, S1 |
| | Extensor digitorum brevis | Extends toes | L5, S1 |
| Superior gluteal nerve | Gluteus medius | Abducts and medially rotates thigh | **L4**, **L5**, S1 |
| | Gluteus minimus | Abducts and medially rotates thigh | **L4**, **L5**, S1 |
| | Tensor fasciae latae | Abducts and medially rotates thigh | **L4**, **L5**, S1 |
| Inferior gluteal nerve | Gluteus maximus | Extends, abducts, and laterally rotates thigh, extends lower trunk | **L5**, **S1**, S2 |

*Source:* Modified and reproduced with permission from Devinsky O and Feldmann E. 1988. *Examination of the Cranial and Peripheral Nerves*. Churchill Livingstone, New York.

[a]Bold text indicates the most important nerve roots, where applicable.

KEY CLINICAL CONCEPT

## 8.1 DISORDERS OF NERVE, NEUROMUSCULAR JUNCTION, AND MUSCLE

A variety of disorders can affect the peripheral nervous system at multiple levels. This text focuses on neuroanatomical localization, so in this chapter and in Chapter 9 we concentrate mainly on localized disorders of the spinal nerve roots, nerve plexuses, or individual nerve branches. Here, we will place these disorders in the wider context of peripheral nervous system disease so that a more complete differential diagnosis can be formulated.

Disorders of the peripheral nervous system can often be distinguished from central nervous system dysfunction by the anatomical pattern of sensory or motor deficits (see KCC 6.3 and KCC 7.3; Table 8.1). In addition, presence of lower motor neuron signs (see KCC 6.1) such as atrophy, fasciculations, decreased tone, or hyporeflexia suggests peripheral nervous system dysfunction, as do paresthesias in a peripheral nerve distribution (see KCC 7.1). When the location of lesions in the central versus peripheral nervous system remains uncertain based on the history and physical examination, diagnostic tests such as neuroimaging studies (see Chapter 4), blood tests, CSF analysis, and electrodiagnostic studies (see KCC 9.2) can be helpful. Disorders of the peripheral nervous system can be produced by a large number of mechanical, toxic, metabolic, infectious, autoimmune, inflammatory, degenerative, and congenital causes that are beyond the scope of this text (see the References at the end of this chapter for additional details). We will now briefly discuss several common disorders of nerve, neuromuscular junction, and muscle.

### *Common Neuropathies*

**Neuropathy** is a general term meaning nerve disorder. The site of pathology can be in the axons, myelin, or both and can affect large-diameter fibers, small-diameter fibers, or both. Usually, neuropathies affect both sensory and

motor fibers in the nerve, although one or the other may be preferentially involved. Damage can be reversible or permanent. The location of neuropathy can be focal (**mononeuropathy**), multifocal (**mononeuropathy multiplex**), or generalized (**polyneuropathy**). Neuropathy affecting the spinal nerve roots is called **radiculopathy**, which we will discuss in greater detail in KCC 8.3. Like neuropathies, motor neuron disorders (see KCC 6.7) can also cause lower motor neuron–type weakness but motor neuron disorders do not cause sensory involvement.

Important causes of neuropathy include diabetes; mechanical causes; infectious disorders such as Lyme disease, HIV, CMV, varicella-zoster virus, or hepatitis B (see KCC 5.9); toxins; malnutrition; immune disorders such as Guillain–Barré syndrome; and hereditary neuropathies such as **Charcot-Marie-Tooth disease**, among others. We will only discuss a few of the more common causes of neuropathy here.

**Diabetic neuropathy** is produced by a number of mechanisms, including compromise of the microvascular blood supply of the peripheral nerves (other possible mechanisms include oxidative stress, autoimmunity, and neurotrophic and biochemical disturbances). The most common pattern of diabetic neuropathy is **distal symmetrical polyneuropathy**, which results in a characteristic **glove and stocking** pattern of sensory loss (see Figure 7.9D). Mononeuropathies are also relatively common in diabetes. Acute diabetic mononeuropathy can affect any cranial or spinal nerve but is most common in CN III and the femoral and sciatic nerves. Onset is usually fairly sudden, and sensorimotor deficits in the nerve distribution may be accompanied by painful paresthesias. There is often partial or complete recovery over the course of weeks to months after onset.

Mechanical causes of nerve injury include **extrinsic compression**, **traction**, **laceration**, or **entrapment** by intrinsic structures such as bone or connective tissue. Mild mechanical disruption of a nerve causes **neurapraxia**, temporary impairment of nerve conduction that usually resolves within hours to weeks. More severe injury can interrupt the axons, leading to **Wallerian degeneration** (degeneration of axons and myelin) distal to the site of injury. As long as the structural elements of the nerve are intact, **axonal regeneration** may occur at a rate of about 1 mm/day (a little more than 1 in./month). Occasional long-term complications include incomplete or aberrant reinnervation and the **complex regional pain syndrome**. Complex regional pain syndrome **Type 1**, also called **reflex sympathetic dystrophy**, is more common and follows an injury without specific nerve damage, while **Type 2**, also called **causalgia**, follows damage to a specific nerve. Both types are characterized by intense local burning pain accompanied by edema, sweating, and changes in skin blood supply. In some situations, when peripheral nerves are severed or otherwise disrupted they can be reanastomosed surgically. In addition, some entrapment syndromes may be amenable to surgical decompression. Painful paresthesias associated with neuropathies of all causes are often treated with medications such as anticonvulsants, serotonin-norepinephrine reuptake inhibitors, or tricyclic antidepressants. Common mechanical neuropathies are discussed further in KCC 8.3 and KCC 9.1.

**Guillain–Barré syndrome**, also known as **acute inflammatory demyelinating polyneuropathy** (**AIDP**), is an important form of neuropathy caused by immune-mediated demyelination of peripheral nerves. Onset typically occurs 1 to 2 weeks following a viral illness, *Campylobacter jejuni* enteritis, HIV infection, or other infections. Presentation is with progressive weakness, **areflexia**, and tingling paresthesias of the hands and feet, with motor involvement typically much more severe than sensory involvement. Symptoms usually reach their worst point 1 to 3 weeks after onset; recovery occurs over

many months. Diagnosis is based on typical clinical presentation, cerebrospinal fluid (CSF) demonstrating **elevated protein** without a significantly elevated white blood cell count, and EMG/nerve conduction studies compatible with **demyelination** (see KCC 9.2). Recovery occurs more quickly when patients are treated with **plasmapheresis** or **intravenous immunoglobulin** therapy. In severe cases, patients require intubation and mechanical ventilation. Autonomic dysfunction can be prominent in some cases, requiring careful monitoring. With good supportive care and immune therapy, the majority of patients enjoy complete or near-complete recovery, although about 20% of patients have some residual weakness 1 year after onset.

### *Common Disorders of the Neuromuscular Junction*

Impaired neuromuscular transmission can lead to motor weakness without sensory deficits. Causes include myasthenia gravis, neuromuscular blocking agents and other drugs, Lambert–Eaton myasthenic syndrome (usually paraneoplastic), and botulism.

**Myasthenia gravis** is an immune-mediated disorder in which there are circulating antibodies against the postsynaptic nicotinic acetylcholine receptors at the neuromuscular junction of skeletal muscle cells. The disorder can sometimes be accompanied by other autoimmune phenomena such as hypothyroidism, lupus, rheumatoid arthritis, and vitiligo. Myasthenia gravis has a bimodal age-related onset, with onset in the second or third decades more common in women and onset in the sixth or seventh decades more common in men. The prevalence is 50 to 125 cases per million. Clinical features include generalized symmetrical weakness, especially of proximal limb muscles, neck muscles, the diaphragm, and eye muscles. Involvement of bulbar muscles can cause facial weakness, a nasal-sounding voice, and dysphagia (see KCC 12.8). Reflexes and sensory exam are normal. Characteristically, **weakness becomes more severe with repeated use of a muscle** or during the course of the day. About 15% of cases have weakness involving only the extraocular muscles and eyelids, a condition called **ocular myasthenia**.

Diagnosis of myasthenia gravis is based on clinical features, and several diagnostic tests, including the ice pack test, repetitive nerve stimulation, measurement of anti-acetylcholine antibodies or muscle specific receptor tyrosine kinase (MuSK) antibodies, single fiber EMG, and chest CT or MRI. The **ice pack** test, which can be performed in patients with ptosis, is administered by placing a bag of ice on the closed eyelids for 2 minutes, and reevaluating for improvement in the ptosis (possibly due to reduced cholinesterase function at lower temperatures). Formerly, the **Tensilon test**, using a short-acting acetylcholinesterase inhibitor (edrophonium) was administered at bedside while observing clinical effects on involved muscles. Commercial manufacture of this agent was discontinued in 2008, however, making future availability of this test uncertain. Clinical response to intermediate-acting acetylcholinesterase inhibitors, such as neostigmine, may also be diagnostically helpful in some cases. Compound motor action potential measurement (see KCC 9.2) with **repetitive nerve stimulation at a rate of 3 per second** often produces a characteristic decrement in amplitude in myasthenia and is considered positive if there is a decrement greater than 10%. Single-fiber EMG is more sensitive (about 90%) but is not specific for myasthenia.

**Anti-acetylcholine receptor antibodies** (**AchR-Ab**) are positive in about 85% of cases of generalized myasthenia but in only about 50% of cases of ocular myasthenia. About half of the patients with generalized myasthenia who are AchR-Ab negative have positive serology for **muscle specific receptor tyrosine kinase antibodies** (**MuSK-Ab**).

About 12% of patients with myasthenia have a **thymoma**, a tumor of the thymus gland, and many others have thymic hyperplasia, so CT or MRI of the chest should be performed. In addition, testing for associated conditions such as thyroid disease and other immune disorders is appropriate.

Myasthenia gravis is treated by immune therapy. Anticholinesterase medications are also helpful to relieve symptoms. **Pyridostigmine** (**Mestinon**) is a long-acting cholinesterase inhibitor, with onset of action beginning about 30 minutes after oral administration and duration of about 2 hours. Patients' doses are individually titrated but should not ordinarily exceed about 120 mg every 3 hours, since excess anticholinesterase can actually worsen weakness. Most patients in the age range of adolescence to 60 years are treated surgically with **thymectomy** (whether a thymoma is present or not), as this usually leads to improvement by unclear mechanisms, likely involving a reduced autoimmune response. Use of thymectomy outside this age range or in patients with ocular myasthenia is more controversial but has been used in some cases. Thymectomy should be performed at a time when patients are relatively clinically stable in order to minimize complications in the perioperative period. Short-term immunotherapy with **plasmapheresis** or **intravenous immune globulin** (**IVIg**) can be helpful, particularly when patients are in **myasthenic crisis** requiring intubation, experiencing other severe worsening in symptoms, or in preparation for elective surgery. Longer-term **immunosuppressive agents**, including steroids, azathioprine, mycophenolate, and cyclosporine, are also typically prescribed.

### *Common Muscle Disorders*

Muscle disorders, or **myopathies**, produce weakness that is typically more severe proximally than distally, without loss of sensation or reflexes. Common causes of myopathy include thyroid disease, malnutrition, toxins, viral infections, dermatomyositis, polymyositis, and muscular dystrophy. **Dermatomyositis** and **polymyositis** are immune-mediated inflammatory myopathies. The blood creatinine phosphokinase (CPK) is typically elevated, and electromyography (EMG) studies (see KCC 9.2) are compatible with myopathy. In dermatomyositis there is a characteristic violet-colored skin rash, typically involving the extensor surface of the knuckles and other joints. Although numerous other forms exist, **Duchenne muscular dystrophy** is the most common form of muscular dystrophy. Transmitted by X-linked inheritance, it affects male children and causes progressive proximal weakness. The abnormal protein (dystrophin) has been identified, providing hope for a cure in the near future. ■

KEY CLINICAL CONCEPT

## 8.2 BACK PAIN

Back pain is one of the most common reasons that people seek medical attention. In this chapter we focus on back pain caused by nerve root disorders; however, it is important to briefly review causes of back pain in general. Table 8.2 is a partial list intended to emphasize the diverse nature of conditions that can be associated with back pain. Many of the diagnoses listed can be elucidated on the basis of a careful history and physical exam. Musculoskeletal causes are most common in all age groups. However, in individuals with onset of back pain over age 50, a neoplasm should be suspected. Back pain in a younger person that worsens with exertion and improves with rest is usually caused by a musculoskeletal problem, including disc herniation in some cases (see KCC 8.3). Symptoms and signs of a radiculopathy (see

**TABLE 8.2 Differential Diagnosis of Back Pain**

| | |
|---|---|
| **TRAUMA/MECHANICAL** | Disc herniation; spondylolysis; vertebral fracture; arthritis; muscle strain/ligament sprain; soft tissue injury |
| **VASCULAR** | Spinal arteriovenous malformation; spinal cord infarct; subarachnoid hemorrhage; spinal epidural hematoma |
| **INFECTIOUS/ INFLAMMATORY/ NEOPLASTIC** | Osteomyelitis; arachnoiditis; spinal epidural abscess; myositis; cytomegalovirus radiculitis; muscle aches in viral illness; Guillain–Barré syndrome; primary or metastatic neoplasms (extradural, extramedullary, or intramedullary) |
| **DEGENERATIVE/ DEVELOPMENTAL** | Scoliosis; degenerative joint disease; amyotrophic lateral sclerosis |
| **REFERRED/OTHER (NON-NEUROLOGIC)** | Normal pregnancy; ectopic pregnancy; menses; urinary tract infection; pyelonephritis; renal stone; retroperitoneal abscess; retroperitoneal hematoma; retroperitoneal tumor; pancreatitis; aortic aneurysm; aortic dissection; angina; myocardial infarction; pulmonary embolism |

KCC 8.3) should be sought. Back pain in any age group that progressively worsens or does not improve over time should be evaluated with appropriate imaging studies (usually an MRI of the spine). In addition, one should never neglect to evaluate bowel, bladder, and sexual function in patients with back pain, so that irreversible loss of function can be prevented (see KCC 7.2 and KCC 8.4). Several clarifying definitions for degenerative disorder of the spine are given in Table 8.3. ■

KEY CLINICAL CONCEPT

## 8.3 RADICULOPATHY

Sensory or motor dysfunction caused by pathology of a nerve root is called **radiculopathy**. (Neuropathies in general are discussed in KCC 8.1; radiculopathy is a specific subtype involving nerve roots.) Radiculopathy is often asso-

**TABLE 8.3 Clarifying Definitions for Degenerative Disorders of the Spine**

| DISORDER | DEFINITION |
|---|---|
| **SPONDYLOLYSIS** | A general term for degenerative disorders of the spine. From the Greek *spondylos*, meaning "vertebra." |
| **SPONDYLOLYSIS** | Fractures that appear in the interarticular portion of the vertebral bone, between the facet joints (see Figure 8.2A). *Lysis* means "loosening" in Greek. |
| **SPONDYLOLISTHESIS** | Displacement of a vertebral body relative to the vertebral body beneath it. Includes **anterolisthesis** or **retrolisthesis**, meaning "anterior" or "posterior" displacement of the upper vertebral bone, respectively. Anterolisthesis often coexists with spondylolysis. In Greek, *olisthesis* means "slipping and falling." |
| **OSTEOPHYTES** | Bony spurs that form on regions of apposition between adjacent vertebrae because of chronic degeneration. From the Greek *osteo*, meaning "bone," plus *phyton*, meaning "plant" or "outgrowth." |
| **SPINAL STENOSIS** | Congenital or acquired narrowing of the spinal canal. |

**TABLE 8.4 Common Causes of Radiculopathy**

| |
|---|
| Disc herniation |
| Osteophytes |
| Spinal stenosis |
| Trauma |
| Diabetes |
| Epidural abscess |
| Epidural metastases |
| Meningeal carcinomatosis |
| Nerve sheath tumors (schwannomas and neurofibromas) |
| Guillain–Barré syndrome |
| Herpes zoster (shingles) |
| Lyme disease |
| Cytomegalovirus |
| Idiopathic neuritis |

ciated with a burning, tingling pain that **radiates** or shoots down a limb in the dermatome of the affected nerve root (see Figure 8.4). There may be loss of reflexes and motor strength in a radicular distribution (see Tables 3.4–3.7 and 8.1). Chronic radiculopathy can result in atrophy and fasciculations (see KCC 6.1). Sensation may be diminished if a single dermatome is involved, but because of overlap from adjacent dermatomes, sensation is usually not absent. Testing with pinprick is more sensitive than touch for detecting radicular sensory loss. Relatively mild or recent-onset radiculopathy can cause sensory changes without motor deficits. T1 radiculopathy can interrupt the sympathetic pathway to the cervical sympathetic ganglia (see Figure 6.13), resulting in Horner's syndrome (see KCC 13.5). Involvement of multiple nerve roots below L1 can result in a cauda equina syndrome (see KCC 8.4).

Common causes of radiculopathy are listed in Table 8.4. The most common cause by far is intervertebral **disc herniation**, which occurs when part or all of the nucleus pulposus extrudes through a tear in the annulus fibrosus, often causing root compression (see Figures 8.2C and 8.3). It usually occurs without any recent history of traumatic injury, but it can occasionally be caused or exacerbated by trauma. Disc herniation as a cause of radiculopathy is common for the **C6**, **C7**, **L5**, and **S1 nerve roots** and less common at other levels. Lumbosacral radiculopathies are about two to three times as common as cervical radiculopathies. Thoracic disc herniations are less common, since this region of the spinal column is less mobile and fixed by the rib cage. Patients with intervertebral disc herniation typically present with back or neck pain, as well as sensorimotor symptoms in a radicular distribution. As the spine degenerates over time, bony **osteophytes** form (see Table 8.3). Osteophytes, together with disc material, may contribute to narrowing of the intervertebral foramina or may protrude more centrally into the canal, causing spinal stenosis and chronic injury to the spinal cord (myelopathy).

The **straight-leg raising test** can be helpful in the diagnosis of mechanical nerve root compression in the lumbosacral region (Figure 8.5A). In this test the patient lies supine and the examiner slowly elevates the patient's leg at an increasing angle to the table while keeping the leg straight at the knee

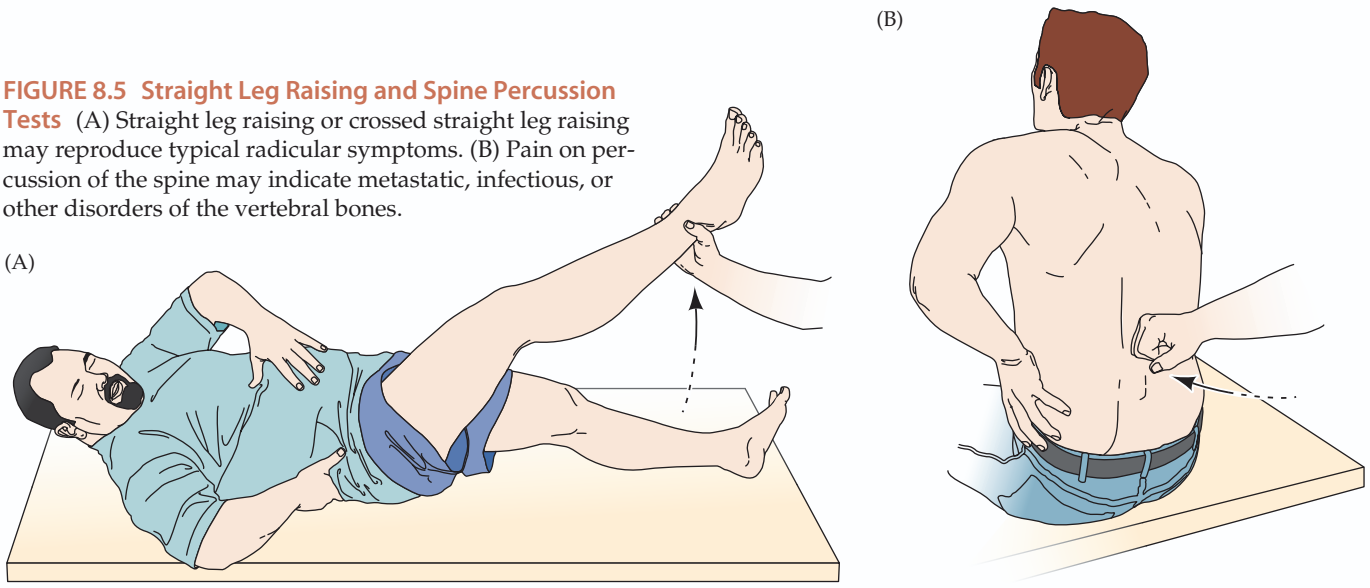



**FIGURE 8.5 Straight Leg Raising and Spine Percussion Tests** (A) Straight leg raising or crossed straight leg raising may reproduce typical radicular symptoms. (B) Pain on percussion of the spine may indicate metastatic, infectious, or other disorders of the vertebral bones.

joint. This provides traction on the nerve roots, and the test is considered positive if it reproduces the patient's typical radicular pain and paresthesias. A response to less than 10° or more than 60°F of straight-leg raising is probably not caused by root compression. In the **crossed straight-leg raising test**, elevating the asymptomatic leg causes typical symptoms in the symptomatic leg. The crossed straight-leg raising test has a specificity of over 90% for lumbosacral nerve root compression. Radicular symptoms may also be increased by the Valsalva maneuver (e.g., coughing, sneezing, straining). In cervical radiculopathy, radicular symptoms may be increased by flexing or turning of the head toward the affected side, likely because of increased narrowing of the intervertebral foramina by these movements. Pain on **percussion of the spine** (Figure 8.5B) may indicate metastatic disease, epidural abscess, osteomyelitis, or other disorders of the vertebral bones, although this sign can be absent in these conditions.

Back pain that is persistent, progressively worsens, or occurs in an older individual, in a patient with prior history of neoplastic disease, or where there is a possibility of epidural abscess, should always be evaluated with a neuroimaging study. An MRI of the spine is usually the test of choice (see Chapter 4). It is important, however, to carefully interpret the MRI in the context of the history and physical examination, since incidental disc bulges and other degenerative changes of the spine are common findings even in individuals without symptoms. In some cases, CT-myelography (see Chapter 4) can help define abnormalities that are not well visualized on MRI. When diagnostic uncertainty remains, EMG and nerve conduction studies (see KCC 9.2) may be helpful.

Other causes of radiculopathy are listed in Table 8.4. **Spinal stenosis**, meaning "narrowing of the spinal canal," can arise congenitally; gradually, as the result of degenerative processes; or by a combination of both factors. **Lumbar stenosis** may result in **neurogenic claudication**, in which bilateral leg pains and weakness occur with ambulation. **Cervical stenosis** can cause a mixture of radicular and long tract signs. Trauma produces radiculopathy through root compression, traction, or **avulsion** of nerve roots off the spinal cord. Diabetic neuropathy can occasionally involve nerve roots, particularly at thoracic levels, producing abdominal pain. **Epidural metastases** most commonly occur in the vertebral bodies, but they can extend laterally to compress nerve roots. Spread of cancer cells such as adenocarcinoma, lymphoma, medulloblastoma, and glioblastoma within the cerebrospinal fluid can involve the nerve roots.

Many causes of radiculopathy are similar to those causing neuropathy in general (see KCC 8.1) but may have an increased tendency to involve the nerve roots. For example, some autoimmune disorders, such as **Guillain–Barré syndrome**, have a predilection for nerve roots. Reactivation of latent **varicella-zoster virus** (chickenpox virus) in dorsal root ganglia produces the painful blistering lesions of **herpes zoster**, or **shingles**. These occur in a dermatomal distribution, associated with sensory and, less commonly, motor loss in the affected nerve roots. Herpes zoster is most common in thoracic dermatomes but can occur anywhere. Treatment with oral antiviral agents such as valacyclovir, famciclovir, or acyclovir can shorten the duration of blistering lesions. Severe pain, referred to as postherpetic neuralgia, can persist after the blistering eruption and is shortened by treatment with antiviral treatment. When herpes zoster occurs in the ophthalmic division of the trigeminal nerve, it can threaten vision, so prompt treatment is critical. **Lyme disease**, a tick-borne illness caused by the spirochete *Borrelia burgdorferi,* can cause radiculopathies. **Cytomegalovirus polyradiculopathy** can be seen in patients with HIV infection, most commonly in the lumbosacral

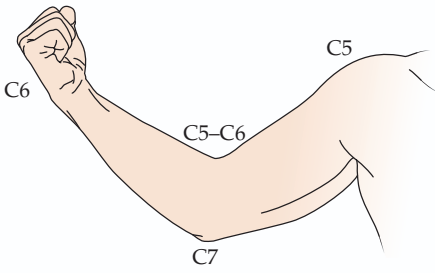



**FIGURE 8.6 Three Roots to Remember in the Arm** C5 mediates arm abduction at the shoulder; C5 and C6 mediate flexion at the elbow and the biceps reflex; C6 mediates wrist extension; C7 mediates elbow extension and the triceps reflex (see also Table 8.5).

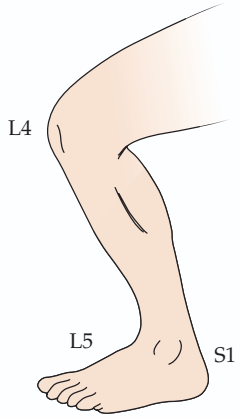



**FIGURE 8.7 Three Roots to Remember in the Leg** L4 mediates leg extension at the knee and the patellar tendon reflex; L5 mediates dorsiflexion at the ankle; S1 mediates plantar flexion at the ankle and the Achilles tendon reflex (see also Table 8.6).

roots. A milder form of radiculopathy can also be caused by HIV itself. Dumbbell-shaped nerve sheath tumors, such as **schwannomas** and **neurofibromas** (in neurofibromatosis), can occasionally occur in a neural foramen, producing radiculopathy. ■

## Simplification: Three Nerve Roots to Remember in the Arm

For practical purposes, the most clinically important nerve roots in the arm are **C5**, **C6**, and **C7**. It is important to be familiar with the reflexes and the motor and sensory functions associated with these nerve roots, as summarized in Figure 8.6 and Table 8.5. When examining patients, it is helpful to have memorized at least one muscle that gets its major innervation from each of these three nerve roots. In addition to the nerve roots listed in Table 8.5, it is also worth knowing that C8 radiculopathy accounts for about 6% of cervical radiculopathies, is usually caused by C7–T1 disc herniation, and is associated with weakness of the intrinsic hand muscles and decreased sensation in the fourth and fifth digits and the medial forearm. About 20% of all cervical radiculopathies involve two or more cervical levels.

## Simplification: Three Nerve Roots to Remember in the Leg

The most clinically important nerve roots in the leg are **L4**, **L5**, and **S1**. Reflexes and the motor and sensory functions associated with L4, L5, and S1 are summarized in Figure 8.7 and Table 8.6. As with the cervical nerve roots, it is helpful, when examining patients, to have memorized at least one muscle that gets its major innervation from each of these nerve roots.

KEY CLINICAL CONCEPT

## 8.4 CAUDA EQUINA SYNDROME

Impaired function of multiple nerve roots below L1 or L2 is called **cauda equina syndrome**. If the deficits begin at the S2 roots and below, there may be no obvious leg weakness. Sensory loss in an S2 to S5 distribution (see Figure 8.4) is sometimes called **saddle anesthesia**. Involvement of the S2, S3, and S4 nerve roots can produce a distended atonic bladder with urinary retention or overflow incontinence (see KCC 7.5), constipation, decreased rectal tone,

**TABLE 8.5 Three Important Nerve Roots in the Arm**

| NERVE ROOT | MAIN WEAKNESS[a] | REFLEX DECREASED[a] | REGION OF SENSORY ABNORMALITY[b] | USUAL DISC INVOLVED | APPROXIMATE PERCENTAGE OF CERVICAL RADICULOPATHIES |
|---|---|---|---|---|---|
| C5 | Deltoid, infraspinatus, biceps | Biceps, pectoralis | Shoulder, upper lateral arm | C4–C5 | 7% |
| C6 | Wrist extensors, biceps | Biceps, brachioradialis | First and second fingers, lateral forearm | C5–C6 | 18% |
| C7 | Triceps | Triceps | Third finger | C6–C7 | 46% |

[a]See Figure 8.6.
[b]See Figure 8.4.

**TABLE 8.6 Three Important Nerve Roots in the Leg**

| NERVE ROOT | MAIN WEAKNESS[a] | REFLEX DECREASED[a] | REGION OF SENSORY ABNORMALITY[b] | USUAL DISC INVOLVED | APPROXIMATE PERCENTAGE OF LUMBOSACRAL RADICULOPATHIES |
|---|---|---|---|---|---|
| L4 | Iliopsoas, quadriceps | Patellar tendon (knee jerk) | Knee, medial lower leg | L3–L4 | 3%–10% |
| L5 | Foot dorsiflexion, big toe extension, foot eversion, inversion | None | Dorsum of foot, big toe | L4–L5 | 40%–45% |
| S1 | Foot plantar flexion | Achilles tendon (ankle jerk) | Lateral foot, small toe, sole | L5–S1 | 45%–50% |

[a]See Figure 8.7.
[b]See Figure 8.4.

fecal incontinence, and loss of erections. It is essential to detect and treat cauda equina syndrome promptly to avoid irreversible deficits. Cauda equina syndrome can sometimes be difficult to differentiate from **conus medullaris syndrome**, in which similar deficits occur as the result of a lesion in the sacral segments of the spinal cord (see Figure 8.1). **Causes of cauda equina syndrome** include compression by a central disc herniation (see Figure 8.3C), epidural metastases, schwannoma, meningioma, neoplastic meningitis, trauma, epidural abscess, arachnoiditis, and cytomegalovirus polyradiculitis. ■

KEY CLINICAL CONCEPT

## 8.5 COMMON SURGICAL APPROACHES TO THE SPINE

Most patients with radiculopathy caused by disc herniation recover within a few months without surgery. Indications for urgent surgery include the rare instances in which cord compression or cauda equina syndrome occurs. Semiurgent surgery is indicated in patients with progressive or severe motor deficits or in the occasional patient with intolerable, medically intractable pain. Elective surgery is contemplated when a clear radiculopathy is present and conservative measures such as rest, physical therapy, and traction have been tried for 1 to 3 months but were ineffective.

In the cervical spine, surgical options include a **posterior approach** with **laminectomy**, meaning removal of the lamina over affected levels (see Figure 8.2B), combined with **discectomy** to remove herniated disc material, and **foraminotomy** to widen the lateral recess through which the nerve root passes just before it exits the intervertebral foramen. An **anterior approach** can also be used in the cervical spine. In this procedure, an incision is made in the anterior neck and the dissection is carried down to the vertebral bodies. The anterior approach provides direct access to the discs without traversing the spinal canal and also allows mechanical **fusion** of adjacent vertebral bodies, usually using a bone graft. An anterior approach is also often favored in cases of thoracic disc herniation, which is rare. In the lumbar spine, a posterior approach is generally used. Sometimes a variety of hardware is implanted to increase mechanical stability. ■

# CLINICAL CASES

## CASE 8.1 UNILATERAL NECK PAIN AND TINGLING NUMBNESS IN THE THUMB AND INDEX FINGER

### CHIEF COMPLAINT

A 30-year-old man came to his physician's office because of 4 weeks of left-sided neck and arm pain and tingling.

### HISTORY

Previous history was unremarkable except for some minor sports injuries. These included a skiing accident 2 years ago in which he struck the left side of his neck and had local pain lasting about 3 weeks, and a backward fall during a softball game 1 year ago in which he struck his occiput without loss of consciousness but had some confusion for about 30 minutes. Four weeks ago he awoke one morning with severe **left neck and shoulder pain with tingling radiating down into the first and second fingers of the left hand** (thumb and index finger). The symptoms improved slightly after a few days but then recurred about 4 days ago, making sleep difficult. Over-the-counter pain medications helped but did not eliminate the pain. He did not notice any weakness, numbness, change in bowel or bladder function (see KCC 7.5), or Lhermitte's sign (see KCC 7.1).

### PHYSICAL EXAMINATION

***Vital signs:*** T = 98°F, P = 72, BP = 140/80.

***Neck:*** Supple; no tenderness.

***Lungs:*** Clear.

***Heart:*** Regular rate.

***Abdomen:*** Soft.

***Extremities:*** Normal.

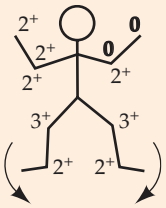
***Neurologic exam:***

***MENTAL STATUS:*** Alert and oriented × 3. Normal language. Gave detailed history.

***CRANIAL NERVES:*** Intact.

***MOTOR:*** Normal tone. 5/5 power throughout, except for **$4^+$/5 power in the left biceps, brachioradialis, and wrist extensors**.

***REFLEXES:***




***COORDINATION:*** Normal on finger-to-nose and heel-to-shin testing.

***GAIT:*** Normal. Tandem gait normal. No Romberg sign.

***SENSORY:*** Intact light touch, vibration, and joint position sense. **Mildly decreased pinprick sensation in the left first and second fingers. Two-point discrimination 4–5 mm in the left index finger**, compared to 3 mm in the right index finger (using a ruler and the ends of a paper clip).

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

## Discussion

The key symptoms and signs in this case are:

- **Left neck and shoulder pain with tingling radiating down into the first and second fingers, accompanied by decreased pinprick and discriminative sensation**
- **Weakness of the left biceps, brachioradialis, and wrist extensors**
- **Absent left biceps and brachioradialis reflexes**

The patient has a typical left C6 radiculopathy (see KCC 8.3; Figures 8.4, 8.6; Tables 8.1, 8.5). It is very likely that the diagnosis in this setting is a left C5–C6 disc herniation (see Table 8.4 for less common causes).

## Clinical Course and Neuroimaging

The patient underwent a cervical spine MRI (Image 8.1A,B, page 338), which confirmed a left C5–C6 disc herniation. He was referred to a neurosurgeon to discuss possible surgery to relieve the nerve root compression. The pa-

tient opted to wait for a few more weeks and to undergo surgery if his symptoms did not resolve. He used a hard cervical collar for traction, avoided physical exertion, and continued taking nonsteroidal anti-inflammatory pain medication. One month later, his symptoms had resolved completely. His exam was normal except for a diminished left biceps reflex and barely detectable left biceps weakness, which continued to improve with physical therapy.

**REVIEW EXERCISE**

Using Figure 8.3A and Image 8.1A,B, explain why a C5–C6 disc herniation usually causes a C6 radiculopathy.

## CASE 8.2 UNILATERAL OCCIPITAL AND NECK PAIN

### MINICASE

A 74-year-old man with a past history of bladder carcinoma developed **left-sided occipital and neck pain** over the course of 2 weeks. Exam was normal except for **questionable altered sensation over the left occipital area**. Head CT and cervical X-rays were normal. A bone scan and MRI of the cervical spine were therefore done.

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

### Discussion

The key symptoms and signs in this case are:

- **Pain and sensory changes over the left occipital area**

The combination of pain and sensory loss in this territory suggests a peripheral nerve lesion. Sensation in the occipital scalp is provided by C2 (see Figure 8.4), which gives rise to the greater and lesser occipital nerves.

The most likely *clinical localization* is left C2 nerve root or left cipital nerves. **Occipital neuralgia** (similar to trigeminal neuralgia; see KCC 12.2) is a relatively common cause of unilateral occipital pain, which is sometimes accompanied by altered sensation. Given the patient's history of bladder cancer, another possible diagnosis is epidural metastasis compressing the left C2 nerve root or left occipital nerves. Less likely possibilities include degenerative disease of the spine or the other diagnoses listed in Table 8.4.

### Clinical Course and Neuroimaging

A **cervical spine MRI** (Image 8.2, page 339) was done, revealing a left cervical mass involving C2. The patient had a CT-guided needle biopsy of the mass, and pathology revealed metastatic transitional cell bladder carcinoma. Radiation therapy was instituted, but the patient gradually deteriorated and was ultimately referred for hospice care.

## CASE 8.3 UNILATERAL SHOULDER PAIN AND WEAKNESS

### MINICASE

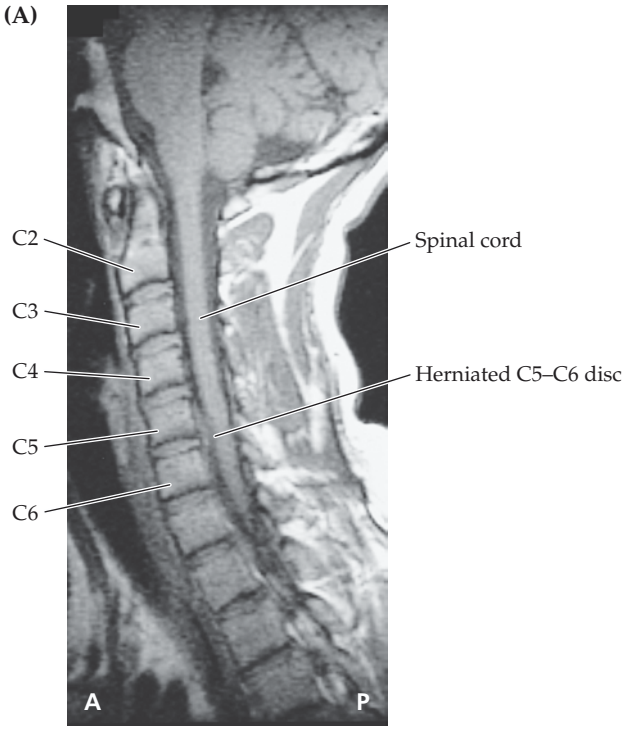
A 50-year-old man with a past history of multiple high school and college football injuries was in a motor vehicle accident and developed **left shoulder pain and numbness** that occasionally radiated down the left arm into the thumb and was increased by neck extension. Exam was normal except for **4/5 deltoid power on the left** and **decreased pinprick sensation in the left shoulder**.

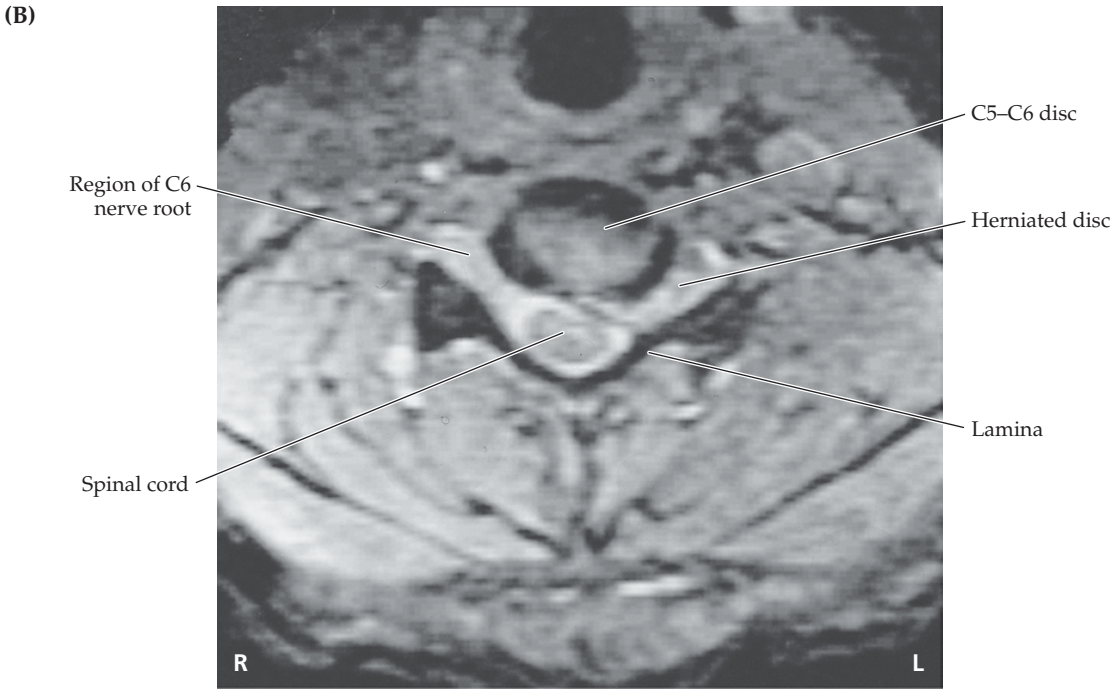
### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

## CASE 8.1 UNILATERAL NECK PAIN AND TINGLING NUMBNESS IN THE THUMB AND INDEX FINGER

**IMAGE 8.1A,B Herniated C5–C6 Intervertebral Disc Obliterating Left C6 Neural (Intervertebral) Foramen**
MRI of the cervical spine. (A) Sagittal T1-weighted image showing herniated C5–C6 intervertebral disc. (B) Axial T2-weighted image at level of herniated C5–C6 intervertebral disc showing that the disc obliterates the left C6 neural foramen.



## Discussion

The key symptoms and signs in this case are:

- **Left shoulder pain and decreased sensation**
- **Left deltoid weakness**

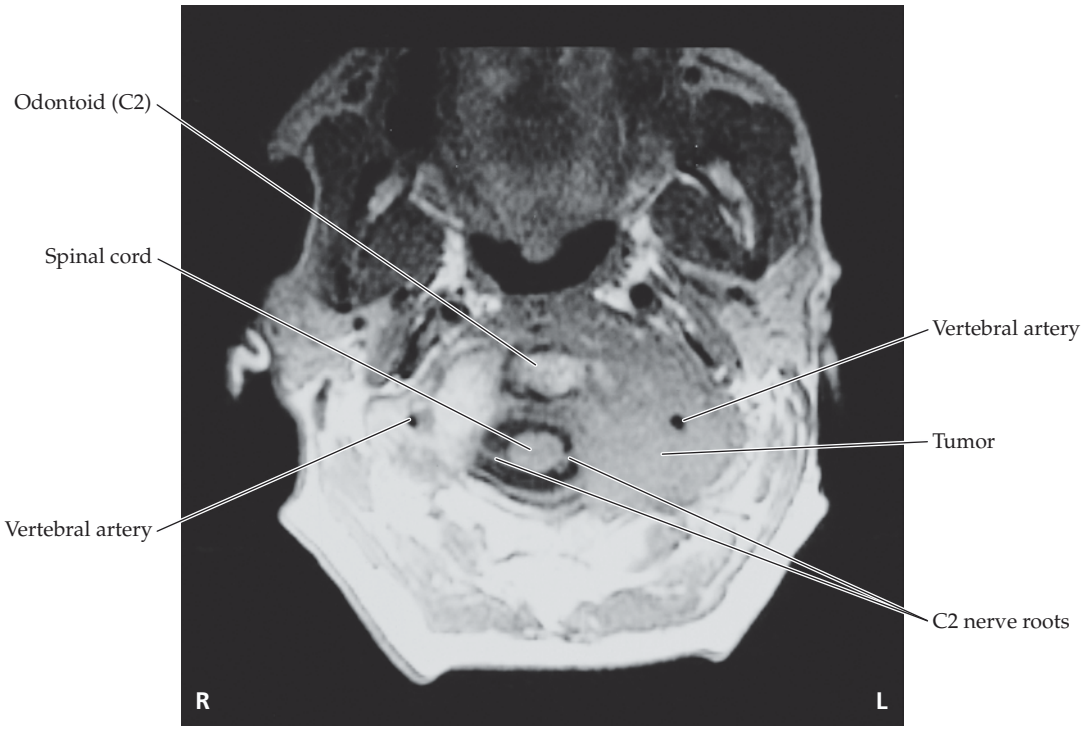
Sensation to the shoulder and motor innervation of the deltoid muscle is provided by the axillary nerve, which gets its predominant supply from the C5 nerve root (see Table 8.1; Figure 8.6). The most likely diagnosis is therefore a left C5 radiculopathy caused by C4–C5 disc herniation or osteophytes. Other, less likely causes of a C5 radiculopathy are listed in Table 8.4. An axillary neuropathy should also be considered (see Table 9.1). In addition, another diagnosis to be considered is rotator cuff tear, an injury to tendons and ligaments that can cause weakness of abduction and external rotation at the shoulder. However, such an injury would not explain this patient's sensory changes.

## Clinical Course

The patient underwent a cervical spine MRI, which demonstrated bony osteophytes (see Table 8.3) causing narrowing of the intervertebral neural foramina at C4–C5 (not shown). He was taken to the operating room for laminectomy and decompression of the neural foramina (see KCC 8.5) and had a good postoperative recovery.

# CASE 8.2 UNILATERAL OCCIPITAL AND NECK PAIN

**IMAGE 8.2 Metastatic Bladder Carcinoma Encasing Left C2 Nerve Root**
Axial T1-weighted MRI of the cervical spine. Compare to Figure 4.12A.



## CASE 8.4 BLISTERS, PAIN, AND WEAKNESS IN THE LEFT ARM

### MINICASE

A 68-year-old man awoke one morning with a painful, blistering rash on his left shoulder and arm, accompanied by left arm weakness and numbness that progressed over the next week. He was eventually **unable to raise his left arm** but had good hand strength. He visited his family doctor, and on exam he had reddish blisters, some of which were scaled over, in the distribution shown in Figure 8.8. In addition, he had **2/5 left deltoid strength**, **3/5 strength in the left arm external rotation**, **and 4/5 strength in the left biceps and brachioradialis**. **Left biceps and brachioradialis reflexes were absent**, while other reflexes were $2^+$. There was **decreased pinprick sensation in the same distribution as the rash** (see Figure 8.8). The remainder of the exam was unremarkable.

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

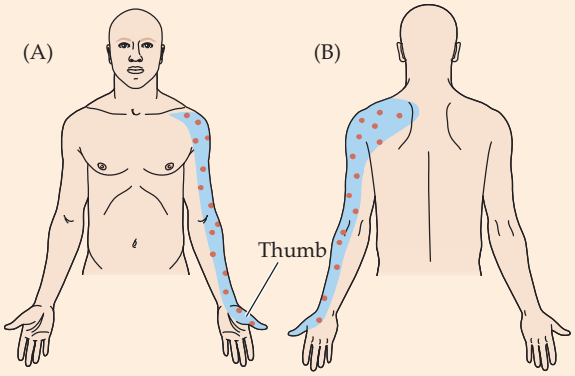



**FIGURE 8.8 Region of Skin Blistering and Decreased Pinprick Sensation** Compare to Figure 8.4.

## Discussion

The key symptoms and signs in this case are:

- **Blistering rash and decreased pinprick sensation in the left shoulder and arm**
- **Weak left deltoid, arm external rotation, biceps, and brachioradialis, with absent left biceps and brachioradialis reflexes**

The herpetic skin lesions, together with sensory loss in a left C5–C6 distribution, make the most likely diagnosis herpes zoster (shingles) of the left C5 and C6 nerve roots (see KCC 8.3). Although more common in thoracic dermatomes, herpes zoster can occur in other dermatomes as well and can occasionally cause weakness. The muscle weakness and reflex loss in this patient are also consistent with C5 and C6 involvement, although C5 seems to be more severely affected, since wrist extension (C6) was not weak (see Tables 8.1, 8.5; Figure 8.6).

## Clinical Course

A lumbar puncture was done. The cerebrospinal fluid (CSF) was normal except for the presence of 224 white blood cells per cubic millimeter (normal is 0–5; see KCC 5.9, 5.10; Table 5.7) with 89% lymphocytes. Viral cultures from CSF and from the skin lesions were negative; however, a polymerase chain reaction (PCR) test on CSF was positive for varicella-zoster virus. The patient was treated with intravenous and, later, oral acyclovir. A cervical spine MRI was negative, as was careful examination for cranial nerve involvement. When the patient was seen 3 months later in follow-up, the rash had resolved. The pain and weakness in his left arm were improving but still a significant problem.

## CASE 8.5 UNILATERAL SHOULDER PAIN AND NUMBNESS IN THE INDEX AND MIDDLE FINGERS

### MINICASE

A 37-year-old physician had sudden onset of sharp **pain in her right shoulder**, followed by tingling and numbness radiating down her arm into her hand. She noticed that her **right second and third fingers felt numb**. She tried to comb her hair, but her right arm kept bending at the elbow, causing her hand to fall onto her head. She was fairly certain of the diagnosis at this point and scheduled an appointment with an orthopedic colleague. Her exam was notable only for **3/5 strength in the right triceps**, **$4^{+}$/5 strength in the right finger extensors, an absent right triceps reflex**, **and decreased sensation to pinprick and light touch in the right second finger and lateral half of the third finger** (Figure 8.9).

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

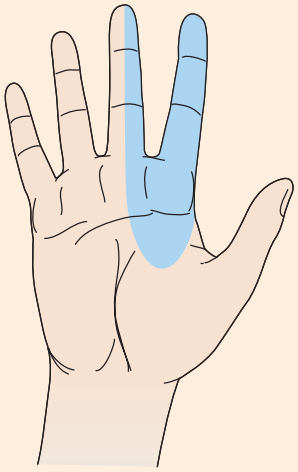


**FIGURE 8.9 Region of Decreased Sensation**
Compare to Figure 8.4.

## Discussion

The key symptoms and signs in this case are:

- **Weakness of the right triceps and finger extensors, with absent right triceps reflex**
- **Right shoulder pain with paresthesias and decreased sensation in the right second finger and lateral half of the third finger**

The weakness, reflex loss, and decreased sensation in this patient are consistent with a right C7 radiculopathy (see Tables 8.1, 8.5; Figures 8.4, 8.6). Note that, as mentioned previously, dermatomal patterns can vary slightly; however, C7 radiculopathy usually affects the middle finger and may involve the index finger as well. The most likely diagnosis is right C7 nerve root compression caused by right C6–C7 disc herniation. Other, less common possibilities are listed in Table 8.4.

## Clinical Course

An MRI of the cervical spine revealed a C6–C7 disc herniation impinging on the right C7 nerve root (see Image 8.1A,B for a similar scan at a different level). She initially elected to pursue conservative therapy, but continued to have pain and developed some atrophy of the right triceps muscle. Therefore, she underwent a partial laminectomy with a C6–C7 discectomy (see KCC 8.5). Postoperatively, her triceps strength recovered fully, although she continued to have some pain and numbness in the right arm and hand over the next few months.

## CASE 8.6 UNILATERAL NECK PAIN, HAND WEAKNESS, AND NUMBNESS IN THE RING AND LITTLE FINGERS

### MINICASE

A 34-year-old cardiothoracic surgeon developed **left neck and shoulder pain, with numbness and tingling radiating down the ulnar aspect of his arm into the fourth and fifth fingers**. On exam he had some **weakness of the intrinsic muscles of the left hand** and **decreased sensation to pinprick and light touch over the left fourth and fifth fingers** (Figure 8.10). The remainder of the exam was normal.

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

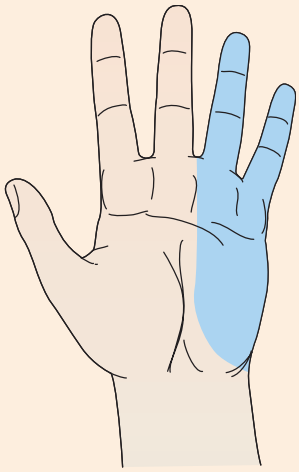


**FIGURE 8.10 Region of Decreased Sensation**
Compare to Figure 8.4.

## Discussion

The key symptoms and signs in this case are:

- **Weakness of the intrinsic muscles of the left hand**
- **Left neck and shoulder pain, with paresthesias and decreased sensation in the left fourth and fifth fingers**

It would have been helpful for localization if additional details of the motor exam had been provided. Weakness of hand intrinsic muscles (lumbricals, interossei) can be caused by lesions of the ulnar nerve, median nerve, lower trunk of the brachial plexus (see Chapter 9), C8, or T1 nerve roots (see Table 8.1). In addition, the distribution of abnormal sensation in this patient is consistent with a lesion of the ulnar nerve, lower trunk of the brachial plexus (C8, T1; see Chapter 9), or C8 nerve root (see Figure 8.4). Neck and shoulder pain suggests a radiculopathy. Therefore, a left C8 radiculopathy caused by leftward C7–T1 disc herniation is the most likely diagnosis (see also Table 8.4); however, an ulnar neuropathy or lower brachial plexus lesion should also be considered.

## Clinical Course

An MRI showed a C7–T1 disc herniation (see Image 8.1A,B for a similar scan at a different level). At the time of laminectomy, some free disc fragments were found compressing the left C8 nerve root and were removed. Postoperatively, the patient's pain was resolved, and his hand strength recovered fully.

## CASE 8.7 PAIN AND NUMBNESS IN THE MEDIAL ARM

### MINICASE

A 66-year-old executive had been suffering for 2 years with **pain and numbness in his left shoulder and medial arm**. Exam was notable for **decreased sensation to light touch in the left medial arm and forearm** (Figure 8.11) and was otherwise unremarkable. Several MRIs and a CT myelogram suggested possible neural compression at multiple levels, including C6–C7, C7–T1, and T1–T2.

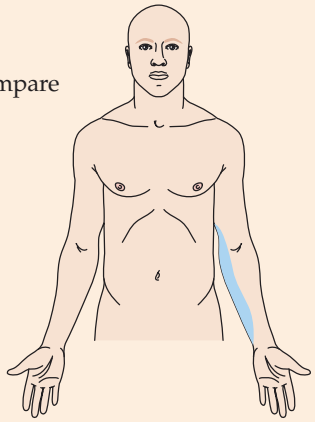


**FIGURE 8.11 Region of Decreased Sensation** Compare to Figure 8.4.

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

## Discussion

The key symptoms and signs in this case are:

- **Left shoulder pain, with decreased sensation in medial arm and forearm**

This patient had sensory changes in the left T1 dermatome (see Figure 8.4). Thoracic radiculopathy, though uncommon, can occasionally be seen as a result of disc herniation or other disorders listed in Table 8.4. It should be noted that this patient had sensory findings only, with no motor involvement (see Table 8.1), which can occur with incomplete nerve root lesions (see KCC 8.3). It should also be noted that Horner's syndrome, which sometimes can be seen in T1 radiculopathy, was not present in this case.

## Clinical Course

Because the imaging studies did not provide a definite level, yet the patient had refractory pain, he was taken to the operating room for laminectomy and exploration of the left C7, C8, and T1 nerve roots. At the time of surgery the left T1 nerve root was found to be compressed by T1–T2 disc fragments, which were removed. This case illustrates that MRI or CT findings suggesting nerve root compression need to be interpreted in the context of clinical symptoms and signs, since asymptomatic radiological abnormalities are common. Postoperatively, the patient's pain improved markedly.

## CASE 8.8 LOW BACK PAIN RADIATING TO THE SOLE OF THE FOOT AND THE SMALL TOE

### CHIEF COMPLAINT

Following an accident, a 38-year-old man developed difficulty walking and low back pain radiating to the lateral sole of his left foot.

### HISTORY

The patient was working on a road when he was injured by an explosion. He suffered severe burns requiring plastic surgery. In addition, he experienced low back pain with **numbness and "pins and needles"** running down his left leg into the **sole and lateral aspect of the left foot, including the small toe**. He had some trouble walking, mostly because of pain, but he also noticed **difficulty standing on his toes with the left foot**. He denied changes in bowel, bladder, or erectile function.

(*continued on p. 344*)

## CASE 8.8 (*continued*)

### PHYSICAL EXAMINATION

***Vital signs:*** T = 98°F, P = 80, BP = 112/80.

***Neck:*** Supple.

***Lungs:*** Clear.

***Heart:*** Regular rate with no murmurs, gallops, or rubs.

***Abdomen:*** Normal bowel sounds; soft.

***Extremities:*** Normal.

***Dermatologic:*** Multiple scars on face and arms.

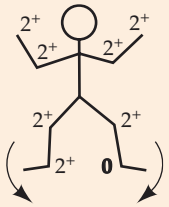
***Neurologic exam:***

***MENTAL STATUS:*** Alert and oriented × 3. Fluent language.

***CRANIAL NERVES:*** Intact.

***MOTOR:*** 5/5 power throughout, except for **4/5 power in the left gastrocnemius and hamstrings** (semitendinosus, semimembranosus, and biceps femoris).

***REFLEXES:***




***COORDINATION:*** Normal on finger-to-nose testing.

***GAIT:*** Slow and painful. **Unable to stand on toes of left foot.**

***SENSORY:*** Intact except for **decreased light touch and pinprick sensation in the left lateral calf, left lateral foot including the small toe, and sole of the left foot** (Figure 8.12).

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

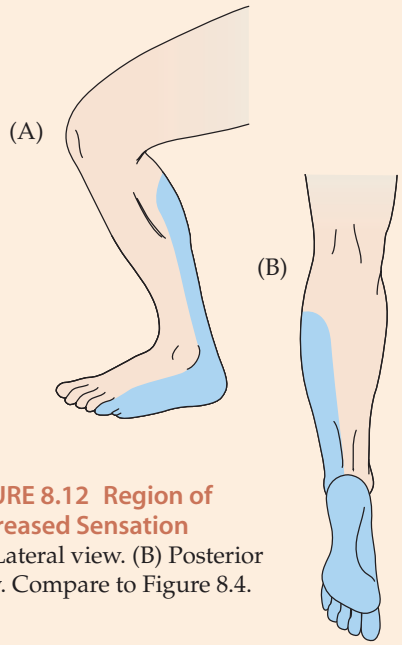



**FIGURE 8.12 Region of Decreased Sensation**
(A) Lateral view. (B) Posterior view. Compare to Figure 8.4.

## REVIEW EXERCISE

Trace the course of the L4, L5, and S1 nerve roots in Image 8.8A–G (see also Figure 8.3B,C) to demonstrate why an L4–L5 disc herniation usually causes an L5 radiculopathy and an L5–S1 disc herniation usually causes an S1 radiculopathy.

## Discussion

The key symptoms and signs in this case are:

- **Weakness of the left gastrocnemius and hamstrings, with absent left Achilles tendon reflex**
- **Paresthesias and decreased sensation in the left lateral calf, lateral foot including the small toe, and sole**

The weakness, reflex loss, and sensory changes in this patient are consistent with a left S1 radiculopathy (see Tables 8.1, 8.6; Figures 8.4, 8.7). The most likely diagnosis is a left posterolateral L5–S1 disc herniation compressing the left S1 nerve root (see also Table 8.4 for other possibilities).

## Clinical Course and Neuroimaging

A **spine MRI** was performed and showed a left L5–S1 disc herniation (Image 8.8A–G, pages 346–347). The patient's symptoms did not improve, and he was therefore taken to the operating room for a laminectomy, which revealed a herniated L5–S1 disc with a free disc fragment compressing the left S1 nerve root in the lateral recess. The fragment was removed, and the patient did well until 1 year later, when he had recurrent pain in the same distribution and mild calf weakness. Repeat MRI showed scar tissue surround-

ing the left S1 nerve root. This could not be treated surgically, and he was therefore treated with pain medications and local steroid injections with only partial relief.

**RELATED CASE.** Image 8.8H (page 348) shows an example from a different patient of a myelogram (see Chapter 4) demonstrating bilateral L5 nerve root compression by a herniated L4–L5 disc.

## CASE 8.9 UNILATERAL THIGH WEAKNESS WITH PAIN RADIATING TO THE ANTERIOR SHIN

### MINICASE

A 76-year-old man suffered for 1 year with **relentless pain and numbness radiating from his right buttock down the anterior thigh into the shin. Exam was notable for $4^-$/5 right quadriceps strength, $4^+$/5 right iliopsoas strength, absent right patellar reflex, and decreased pinprick sensation in the right shin and medial calf** (Figure 8.13).

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

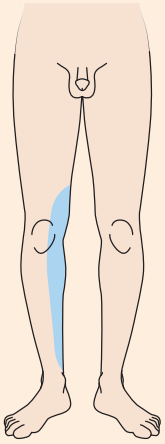


**FIGURE 8.13 Region of Decreased Sensation** Compare to Figure 8.4.

## Discussion

The key symptoms and signs in this case are:

- **Weakness of the right quadriceps and iliopsoas, with absent right patellar reflex**
- **Paresthesias and decreased sensation in the anterior thigh, shin, and medial calf**

The pattern of weakness, reflex loss, and sensory changes is compatible with a right femoral neuropathy or L4 radiculopathy (see Tables 8.1, 8.6; Figures 8.4, 8.7; see also Table 9.3). **Lesions of the femoral nerve can sometimes be distinguished from an L4 radiculopathy by testing for weakness of thigh adduction**, which may be present in L4 radiculopathy but not femoral neuropathy (see Table 8.1). Unfortunately, thigh adduction testing was not documented in this patient. An L2 or L3 radiculopathy could also be considered in this patient; however, these radiculopathies do not usually produce sensory changes extending below the knee, and they are also much less common than an L4 radiculopathy. The most likely diagnosis is therefore right femoral neuropathy or right posterolateral L3–L4 disc herniation compressing the right L4 nerve root (see also Table 8.4 for other possibilities).

## Clinical Course

Interestingly, rather than an L3–L4 posterolateral disc herniation, this patient's MRI (not shown) revealed a herniated right L4–L5 disc extending far upward and laterally to compress the right L4 nerve root (review Figure 8.3C). Following laminectomy and removal of the herniated disc material, the patient had complete resolution of the pain, and his right leg strength improved.

## CASE 8.8 LOW BACK PAIN RADIATING TO THE SOLE OF THE FOOT AND THE SMALL TOE

**IMAGE 8.8A,B L5–S1 Posterolateral Disc Herniation Compressing Left S1 Nerve Root in the Lateral Recess** T1-weighted MRI of the spine. (A) Parasagittal view slightly to the left of midline, showing herniated L5-S1 intervertebral disc. (B) Parasagittal view, farther to the left of midline, showing neural foramina. Note that the foramen for the L5 root (L5–S1) is not obstructed.

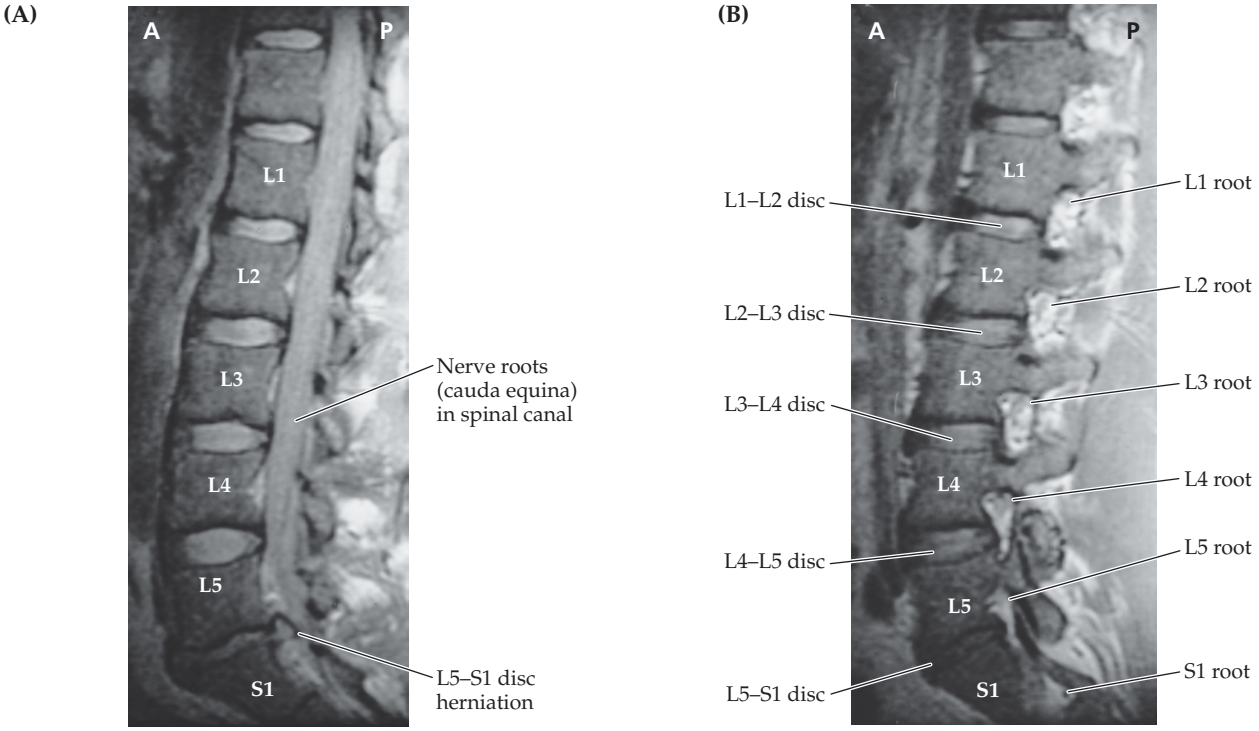
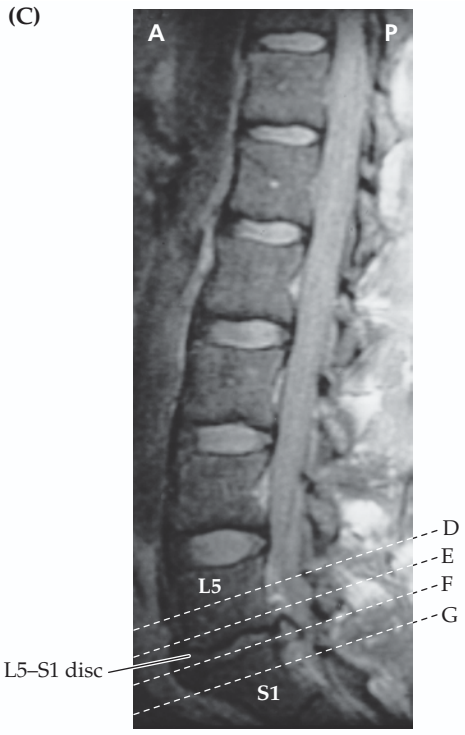
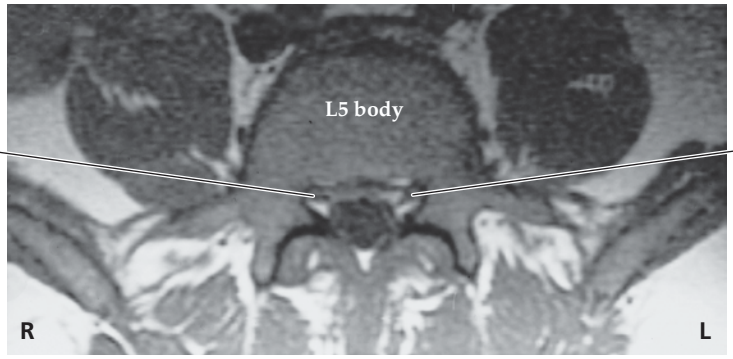



**IMAGE 8.8C–G Axial Sections Showing Posterolateral Disc Herniation Compressing Left S1 Nerve Root in the Lateral Recess** T1-weighted MRI of the spine. (C) Midsagittal view showing herniated L5–S1 intervertebral disc (as in Image 8.8A), with levels of axial sections in D–G indicated. Sections D–G proceed from rostral to caudal. (D) Axial section at level of L5 vertebral body showing L5 nerve roots in lateral recesses. (E) Axial section at level of L5 neural foramen, above the level of the L5–S1 intervertebral disc (compare to Figure 8.3B,C). (F) Axial section at level of L5–S1 intervertebral disc showing herniation of disc into left lateral recess compressing left S1 nerve root (compare to Figure 8.3B,C). (G) Axial section at level of S1 body showing S1 nerve roots in lateral recess below the level of compression.

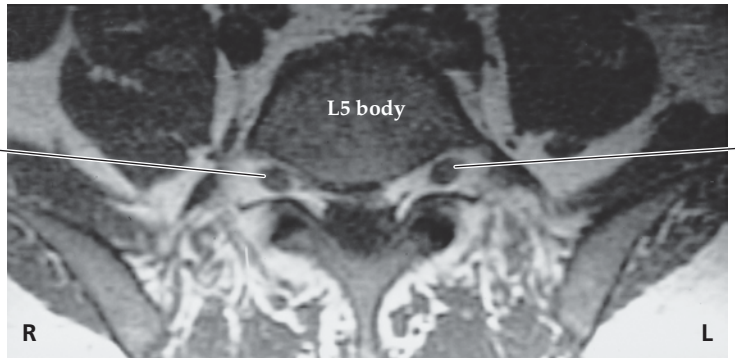


## CASE 8.8 (*continued*)

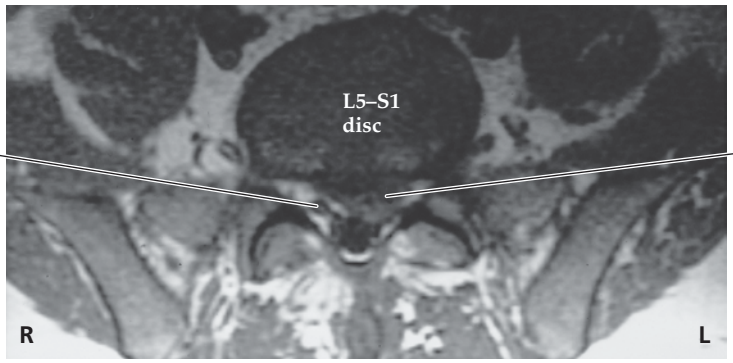
(D)



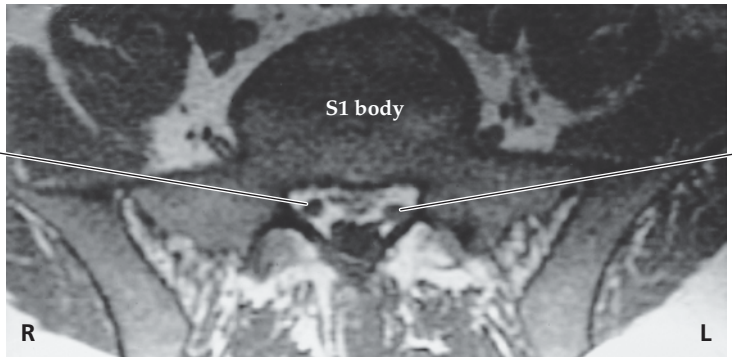

(E)




(F)

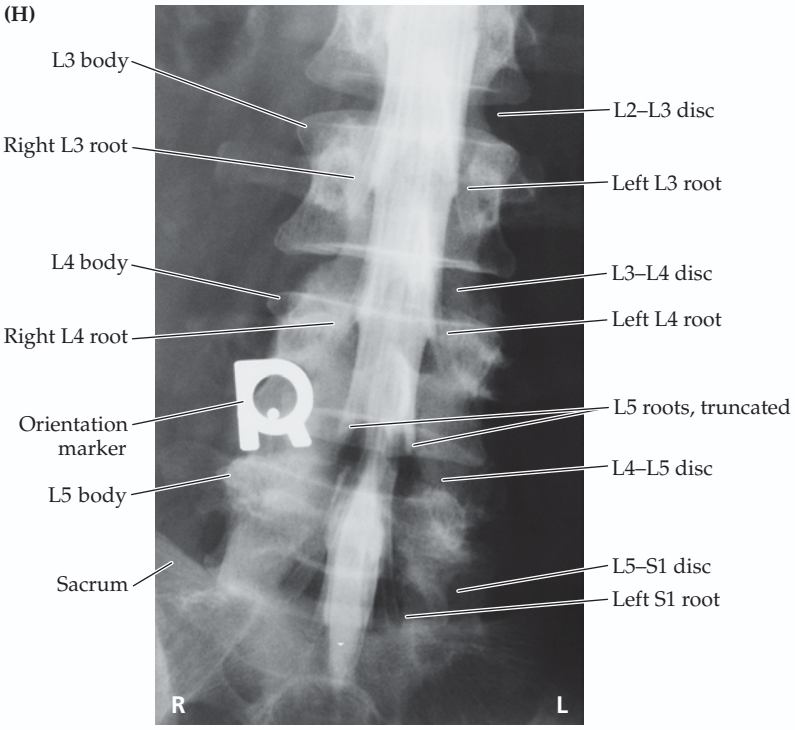



(G)



## CASE 8.8 *RELATED CASE*

**IMAGE 8.8H Example of Myelogram Showing Bilateral L5 Nerve Root Compression at the Level of the L4–L5 Intervertebral Disc** An anterior–posterior plain X-ray film is shown after introduction of myelographic contrast material into the subarachnoid space (see Chapter 4; see also KCC 5.10). The normal L3, L4, S1, and S2 nerve root sleeves can be visualized in relation to vertebral bones (compare to Figure 8.3). The L5 nerve root sleeves are truncated bilaterally because of L4–L5 intervertebral disc herniation.




## CASE 8.10 LOW BACK PAIN RADIATING TO THE BIG TOE

### MINICASE

A 57-year-old man with low back pain for over 20 years tripped over a door ledge and had a sudden increase in **right-sided low back pain radiating down his leg to the right big toe**. He had some difficulty walking because of the pain, causing him to visit the emergency room several times over the next 3 months, where his exam was notable for **3/5 power in the right extensor hallucis longus and tibialis anterior**, **$4^{+}$/5 power in the right foot invertors and evertors**, normal reflexes, and **decreased pinprick sensation in the right anterolateral calf and dorsum of the foot** (Figure 8.14). **Straight-leg raising** (see Figure 8.5A) beyond 30° on the left side had no effect, but on the right side **reproduced the patient's usual pain**.

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

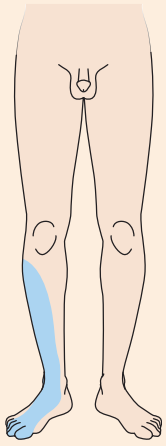


**FIGURE 8.14 Region of Decreased Sensation** Compare to Figure 8.4.

## Discussion

The key symptoms and signs in this case are:

- **Weakness of the right extensor hallucis longus, tibialis anterior, and right foot invertors and evertors**
- **Pain radiating to the right big toe reproduced by straight-leg raising, with decreased sensation in the anterolateral calf and dorsum of the foot**

This patient has typical radicular pain, sensory loss, and weakness compatible with a right L5 radiculopathy (see Tables 8.1, 8.6; Figures 8.4, 8.7). A peroneal nerve palsy (see Tables 8.1, 9.3) can also produce similar decreased sensation and foot drop but does not cause painful paresthesias with straight-leg raising. In addition, lesions of the peroneal nerve can sometimes be distinguished from an L5 radiculopathy by testing for weakness of foot inversion, which may be present in L5 radiculopathy but not in peroneal nerve palsy (see Table 8.1).

The most likely diagnosis is therefore right posterolateral L4–L5 disc herniation compressing the right L5 nerve root (see also Table 8.4 for other possibilities).

## Clinical Course

An MRI showed a herniated L4–L5 disc compressing the right L5 nerve root (see Image 8.8F for a similar scan at a different level). Surgery was discussed with the patient; however, he was lost to follow-up.

# CASE 8.11 SADDLE ANESTHESIA WITH LOSS OF SPHINCTERIC AND ERECTILE FUNCTION

### CHIEF COMPLAINT

A 39-year-old man came to the emergency room with 10 days of bilateral gluteal pain, numbness, and sphincteric dysfunction.

### HISTORY

Ten days prior to admission the patient was doing heavy labor with concrete when he coughed and felt a sudden "pop" followed by sharp **pain in the gluteal region bilaterally**. The pain was only partly relieved by over-the-counter pain medications. During the following days he noticed that he **had no erections**, even upon awakening. In addition, he noticed a **loss of sensation over his genitals and buttocks**. When he sat down it felt as though he was "on air" because he could not feel the seat. He also became **constipated** and did not have any bowel movements for 10 days, despite frequent attempts. **Urination was also difficult**, and when he felt discomfort from bladder distention, he applied pressure over his lower abdomen to initiate flow. Because of increasing problems with urinary retention, he finally came to the emergency room.

### PHYSICAL EXAMINATION

***Vital signs:*** T = 98.6°F, P = 60, BP = 130/80, R = 16.

***Neck:*** Supple with no bruits.

***Lungs:*** Clear.

***Heart:*** Regular rate with no murmurs, gallops, or rubs.

***Abdomen:*** Normal bowel sounds; soft. **Firm, distended bladder palpable in lower abdomen above pubic bone.**

***Extremities:*** No edema.

***Rectal:*** **Rectal tone flaccid.**

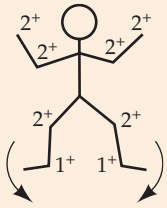
***Neurologic exam:***

***MENTAL STATUS:*** Alert and oriented × 3. Normal language.

***CRANIAL NERVES:*** Intact II–XII.

***MOTOR:*** Normal bulk and tone. 5/5 power throughout.

***REFLEXES:*** **No anal wink. Only trace bulbocavernosus reflex** (see Table 3.7). Cremasteric reflex was present.




***COORDINATION:*** Normal on finger-to-nose and heel-to-shin testing.

***GAIT:*** Normal.

(*continued on p. 350*)

## CASE 8.11 (*continued*)

***SENSORY:*** **Decreased pinprick and light touch sensation in a saddle distribution, including the genitals, perianal area, buttocks, and upper posterior thighs** (Figure 8.15). Pinprick, light touch, vibration, and joint position sense were normal in all other areas.

***POSTVOID RESIDUAL VOLUME (see KCC 7.5):*** The patient was catheterized after attempting voluntary urination, and **1300 cc** of urine were obtained (normal volume is less than 100 cc).

### LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in **bold** above, where is the lesion? What is the most likely diagnosis?

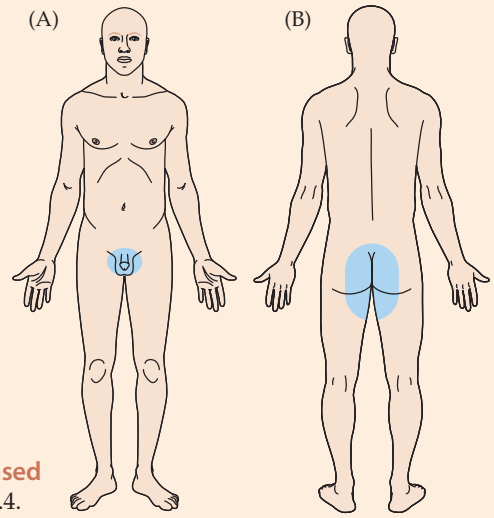



**FIGURE 8.15 Region of Decreased Sensation** Compare to Figure 8.4.

## Discussion

The key symptoms and signs in this case are:

- **Pain in the gluteal region bilaterally with loss of sensation in a saddle distribution over the genitals and buttocks**
- **Constipation, urinary retention, loss of erections, loss of rectal tone, no anal wink, and loss of bulbocavernosus reflex**

Impaired bowel, bladder, and sexual function can be caused by bilateral lesions of the cerebral hemispheres, spinal cord, conus medullaris, cauda equina, or peripheral nerves (see KCC 7.5). The intact cremasteric reflex suggests that function of the L1–L2 nerve roots is preserved (see Table 3.7), and the normal lower extremity strength suggests preserved function down through S1. Meanwhile, the region of pain and sensory loss is in the bilateral S2 through S5 or coccygeal dermatomes (see Figure 8.4), suggesting a lesion of the lower cauda equina or conus medullaris.

The most likely *clinical localization* is: cauda equina S2 through S5 roots, or conus medullaris.

Given the sudden onset of symptoms, a central disc herniation is likely as the diagnosis. Since the lower nerve roots are located more medially in the cauda equina (see Figure 8.3C), a central disc herniation tends to compress the lower nerve roots. For other, less likely causes of cauda equina syndrome in this patient, see KCC 8.4. Possible lesions of the conus medullaris include intrinsic tumors such as ependymoma or astrocytoma; metastatic lesions; demyelinating processes; and sarcoidosis.

## Clinical Course and Neuroimaging

The patient immediately underwent a **spinal CT/myelogram** (Image 8.11A–C, pages 352–353), which showed an L5–S1 central disc herniation compressing the cauda equina. An urgent laminectomy was therefore performed. A large mass of disc material was found compressing the thecal sac from the ante-

rior aspect at the L5–S1 intervertebral level. Following decompression, the patient's pain improved, and he regained some gluteal sensation; however, he still had urinary retention and required intermittent catheterization at the end of his 11-day hospital stay.

## Additional Cases

Other chapters include related cases for the following topics: **peripheral nerve disorders** (Cases 9.1–9.14); **distal symmetric polyneuropathy** (Cases 6.5 and 10.3); and **cranial neuropathy** (Cases 12.2–12.7, 13.1–13.3, and 13.5). Other relevant cases can be found using the **Case Index** located at the end of this book, and new cases are also available through the **Online Review and Study Guide**.

## Brief Anatomical Study Guide

1. In this chapter we have discussed the segmental innervation of the body provided by **dorsal sensory** and **ventral motor nerve roots** that exit the spinal cord at **cervical**, **thoracic**, **lumbar**, **and sacral** levels (see Figure 8.1A) and fuse to form mixed spinal nerves (see Figure 8.1B). Because the vertebral bones outgrow the spinal cord during development, the lower roots continue **below the L1 or L2 vertebral bones** as the **cauda equina** (see Figure 8.1). The sensory regions innervated by nerve roots are called **dermatomes** (see Figure 8.4), and motor territories of nerve roots are called **myotomes**.
2. The most common cause of nerve root dysfunction, or **radiculopathy**, is **intervertebral disc herniation** at the cervical or lumbosacral levels (see Figures 8.2 and 8.3). The nerve root involved usually corresponds to the **vertebral body below the level of the herniated disc**. For example, an L5–S1 disc herniation usually causes an S1 radiculopathy.
3. The **three most clinically relevant arm and leg nerve roots are C5**, **C6**, **and C7**, **and L4**, **L5**, **and S1**, **respectively**. A summary of the sensory and motor functions of these nerve roots is provided in Tables 8.5 and 8.6 and Figures 8.6 and 8.7.