CHAPTER 36

Sensory Localization

Diminution or loss of sensation may occur because of lesions involving the peripheral nerves, nerve roots, spinal cord, brainstem, or higher centers of the brain, as may abnormal sensations, such as pain or paresthesia. Localization depends on the pattern and distribution of the sensory abnormality.

The primary modalities may be impaired because of disease involving peripheral nerve, spinal root, or sensory pathways within the central nervous system (CNS). When the primary modalities are normal in a particular body region, but the cortical modalities are impaired, a parietal lobe lesion may be responsible. When some primary modalities are involved more than others, the sensory loss is said to be "dissociated." The pathways conveying pain and temperature (the spinothalamic tracts) run in a different location than the pathways conveying touch, pressure, position, and vibration (the posterior columns, dorsolateral funiculus, and medial lemniscus). After running divergently through much of their central course, the sensory pathways converge again as they approach the thalamus and remain together in the thalamocortical projections. When the pathways are close together, such as in the peripheral nerve, spinal root, or thalamus, disease processes tend to affect all primary modalities to an approximately equal degree. When the pathways are remote from each other, such as in the spinal cord and brainstem, a disease process may affect one type of sensation and not another, producing dissociated sensory loss. A common example of dissociated sensory loss is lateral medullary stroke, or Wallenberg's syndrome. There is a very characteristic pattern of sensory loss, which only involves pain and temperature and completely spares light touch. The pain and temperature loss involves the ipsilateral face, because of involvement of the spinal tract of cranial nerve V, and the contralateral body, because of damage to the lateral spinothalamic tract, sparing the light touch pathways that are running in the midline in the medial lemniscus. A classic but not common cause of dissociated sensory loss is syringomyelia. The pain and temperature sensory fibers crossing in the anterior commissure are affected; light touch sensory fibers running in the posterior columns are well removed from the site of the pathology and remain intact. As a result, syringomyelia characteristically causes sensory loss to pain and temperature with preservation of light touch. Anterior spinal artery stroke is another example of dissociated sensory loss. The infarction involves the anterior two-thirds of the cord, sparing the posterior columns, which are perfused by the posterior spinal arteries. The patients have dense motor deficits and dense sensory loss to pain and temperature but normal touch, pressure, position, and vibration. Patients with Brown-Séquard syndrome have extreme dissociation of modalities, with loss of pain and temperature on one side of the body and loss of touch, pressure, position, and vibration on the other side of the body.

In contrast, disease processes affecting a peripheral nerve trunk or a spinal root tend to involve all of the sensory fibers traveling in that nerve or root. The sensory loss involves all modalities, but not necessarily to the same degree. Occasionally, generalized polyneuropathies may have a predilection for large or small fibers and can cause some differential involvement of pain and temperature as opposed to touch and pressure. These neuropathies are uncommon and tend to be generalized. When there is marked sensory dissociation affecting one body region, the pathology is virtually always going to be in the CNS, specifically in those regions where the different sensory pathways run in widely divergent locations.

The other consideration in elucidating the cause of sensory loss, in addition to the modalities involved, is the distribution of the abnormality. Deficits in a "hemi" distribution obviously suggest CNS disease, likely involving either the cortex or the thalamus. Crossed deficits, affecting the face on one side and the

body on the opposite side, suggest brainstem disease. Deficits involving both sides of the body below a certain level (e.g., T5) suggest spinal cord disease. A spinal cord level with "sacral sparing" suggests intraparenchymal spinal cord pathology rather than a myelopathy due to external pressure. Deficits due to generalized peripheral nerve disease typically involve the most distal body regions in a "stocking-glove" distribution. Sensory loss due to dysfunction of a peripheral nerve, nerve root, or nerve plexus follows the innervation pattern of that particular structure. Figure 36.1 depicts some of the commonly seen patterns of sensory loss. In hemidistribution sensory loss, there is a certain amount of side-to-side crossing or overlap of innervation along the anterior midline, which is greater on the trunk than on the face. Because of this midline overlap, organic sensory loss usually stops short of the midline, while nonorganic sensory loss may "split the midline" (see "Nonorganic Sensory Loss," below). Sacral sensation is not tested as part of a routine neurologic examination. In some instances, sensation in the saddle distribution should be examined (e.g., when a conus medullaris or cauda equina lesion is a possibility; when there is evidence of a myelopathy; or when there is bladder, bowel, or sexual dysfunction).

Sensory function and motor activity are interdependent, and severe motor disabilities may occur because of impaired sensation. This is particularly evident with parietal lobe lesions, but motor dysfunction may also occur with lesions involving the posterior roots, peripheral nerves posterior columns of the spinal cord, or the other central sensory pathways.

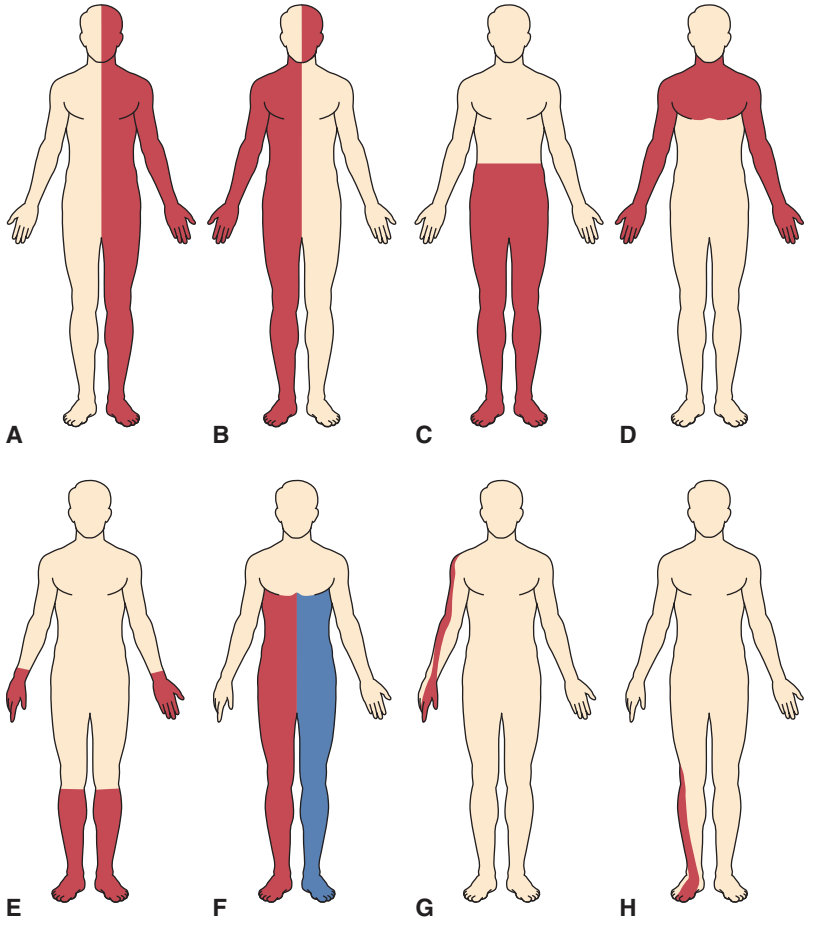
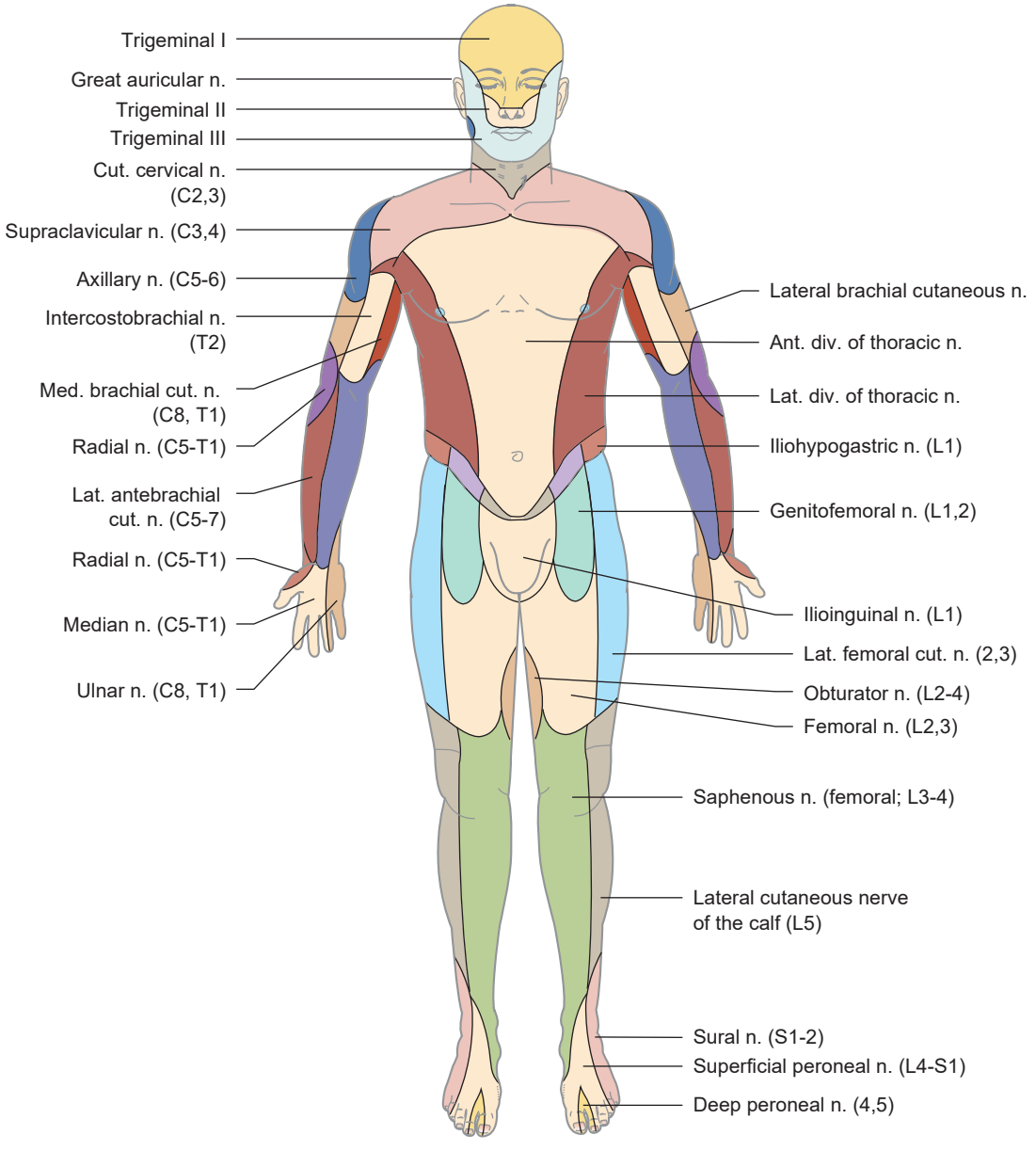


FIGURE 36.1 Some common patterns of sensory loss. **A.** Hemisensory loss due to a hemispheric lesion. **B.** Crossed sensory loss to pain and temperature due to a lateral medullary lesion. **C.** Midthoracic spinal cord level. **D.** Suspended, dissociated sensory loss to pain and temperature due to syringomyelia. **E.** Distal, symmetric sensory loss due to peripheral neuropathy. **F.** Crossed spinothalamic loss on one side with posterior column loss on the opposite side due to Brown-Séquard syndrome. **G.** Dermatomal sensory loss due to cervical radiculopathy. **H.** Dermatomal sensory loss due to lumbosacral radiculopathy.

Conversely, motor dysfunction may affect sensory discrimination. When equal weights are placed in a patient's hands, she may underestimate the weight on the side with cerebellar dysfunction and overestimate it on the side with extrapyramidal dysfunction.

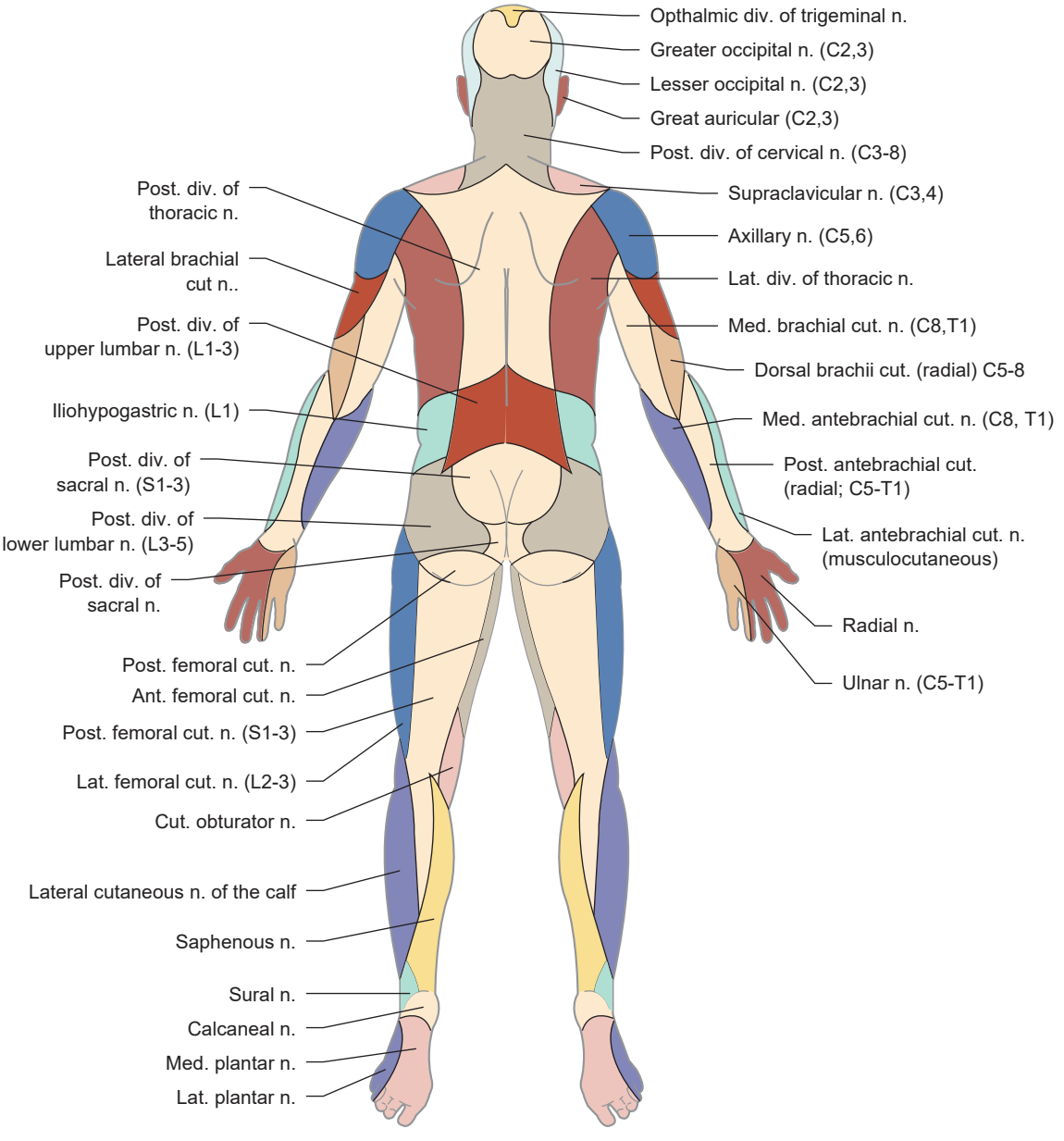
Diminution or perversion of sensation may occur with pathology involving the *sensory receptors*, but this does not often arise in primary neurologic illnesses. Pain and pruritus due to skin irritation, traumatic denudements, and burns may result from abnormalities of the receptors or the nerve filaments to them, and decreased sensation in callosities and scars may result from involvement of the end-organs and smaller filaments.

In *focal peripheral neuropathies*, the area of sensory abnormality corresponds to the distribution of the specific involved nerve. The areas of skin supplied by various nerves are shown in Figure 36.2. Within



A

FIGURE 36.2 The cutaneous distribution of the peripheral nerves. **A.** On the anterior aspect of the body. *(continued)*



B

FIGURE 36.2 *(Continued)* **B.** On the posterior aspect of the body.

the involved area, all sensory modalities are affected to a greater or lesser degree. Sensory distributions may vary slightly from individual to individual, and the mapped area may not correspond precisely to a published text or atlas. An excellent source for a pictorial/graphic demonstration of peripheral nerve distributions is http://www.neuroguide.com/nerveindex.html. Figure 36.3 demonstrates some of the variability in the cutaneous supply of the superficial radial nerve.

The demonstrable area of pain and temperature loss is typically smaller than the area of light touch loss, and smaller than the published peripheral nerve or dermatome distributions. The deficit to light touch usually corresponds more closely to a nerve distribution than the pinprick loss. In a patient with a focal

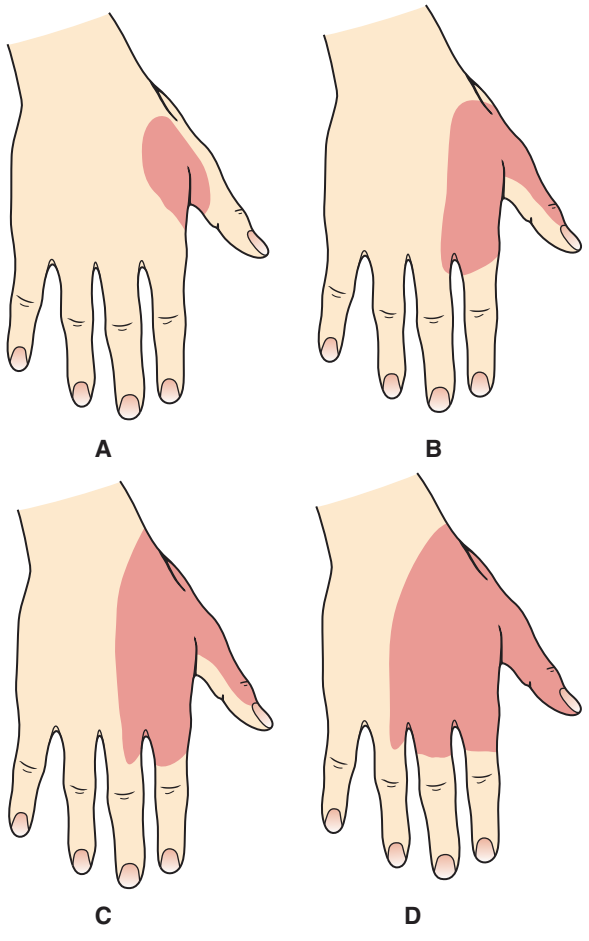


FIGURE 36.3 Variations in the cutaneous distribution of the radial nerve. **A.** Frequent distribution. **B.** Typical distribution. **C.** Frequent distribution. **D.** Anesthesia beyond the usual limit. (Modified from Tinel J. *Nerve Wounds*. Rothwell F [trans]. New York, William Wood & Co., 1917.)

nerve or root lesions, it may be possible with careful testing to identify a dense zone of severe sensory loss, corresponding to the area of autonomous supply, surrounded by areas of milder sensory loss in the zone of overlap with adjacent nerves (Figure 36.4). Occasionally, there is spread of sensory loss beyond the field of an injured nerve. Patients may have allodynia or hyperpathia in the area of sensory loss. The sensory and other neurologic abnormalities associated with lesions of specific nerves are described in Chapter 46. The sensory examination is important in the diagnosis of peripheral nerve injuries and in the evaluation of progress in nerve regeneration. In disorders of the brachial and lumbosacral plexus, sensory loss follows the same principles as in focal neuropathy but is localized to some plexus component, for example, lateral shoulder in upper brachial plexopathy and medial forearm and hand in lower brachial plexopathy.

In *generalized peripheral neuropathies*, vibration is often the first modality affected, but in severe cases, all exteroceptive, proprioceptive, and combined modalities are impaired. Some generalized peripheral neuropathies are purely sensory and some purely motor, but most are sensorimotor. Most axonopathies are length dependent, and the distribution of sensory loss usually involves predominantly the distal segments, causing a stocking-glove distribution of blunted sensation. However, margins of the involved area may be poorly demarcated, with no sharp border between the normal and hypesthetic areas. When severe, length-dependent axonopathies cause sensory loss in a strip over the anterior trunk because of involvement of intercostal nerves (shield or cuirass pattern). Even more severe dying back may cause "beanie cap" sensory loss, or global sensory loss, sparing only a strip in the posterior midline. Leprosy sensory loss may be limited to acral, temperature-dependent regions. Axonopathies produce length-dependent reflex loss; ankle jerks first and then more proximal reflexes disappear as the disease progresses.

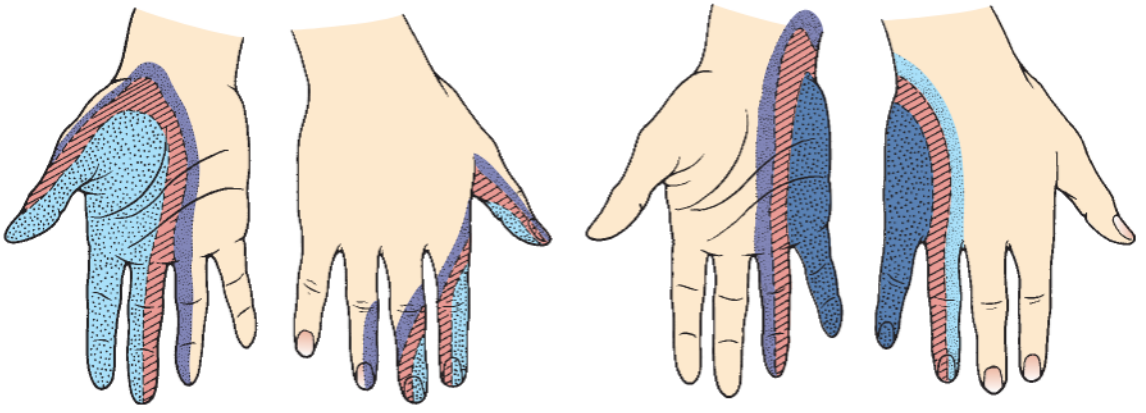


FIGURE 36.4 Transition in sensory changes with lesions of the median and the ulnar nerve. The smallest area is completely anesthetic, the next area has decreased sensation, and the surrounding area has only slight decrease in sensation. (Modified from Tinel J. *Nerve Wounds*. Rothwell F [trans]. New York, William Wood & Co., 1917.)

Demyelinating neuropathies usually cause only slight sensory loss, and the reflexes are lost globally. Rare neuropathies, for example, Tangier's disease and porphyria, have a predilection for short fibers.

Some generalized neuropathies have a predilection to involve predominantly large or small fibers. Large fiber sensory neuropathies include uremia, Sjögren's syndrome, vitamin B_{12} deficiency, certain toxins (pyridoxine, cisplatin, metronidazole), and some cases of diabetes mellitus (pseudotabes). Small fiber neuropathies include amyloidosis, hereditary sensory autonomic neuropathy, and some cases of diabetes mellitus (pseudosyringomyelia). Large fiber neuropathies are typically associated with reflex loss and, when severe, with motor involvement. Small fiber neuropathies typically produce burning pain with no motor loss and preserved reflexes. Peripheral nerve disease may also cause paresthesias, or pain that is either constant or lancinating in character. The nerves themselves may be sensitive and tender to palpation, and there may be pain on brisk stretching of the affected nerves and increased susceptibility to ischemia. There sometimes is hyperalgesia or allodynia in the involved area, even though the sensory threshold is raised.

Disease of the *dorsal root ganglia* (*DRG*), or corresponding cranial nerve ganglia, is also associated with sensory changes. The DRG may be affected by autoimmune processes, causing degeneration and inflammation of the neurons. Patients have the subacute onset of pain, paresthesias, and sensory loss, which affects large more than small fibers. Strength is preserved, but reflexes disappear. There is often a disabling sensory ataxia, which may be accompanied by pseudoathetosis. Cerebrospinal fluid protein is frequently increased. Although classically a remote effect of small cell carcinoma of the lung, sensory neuronopathy is associated with a number of other conditions, including pyridoxine intoxication, Sjögren's syndrome, and lymphoma. In herpes zoster, there is severe, lancinating pain in the distribution of the affected ganglia. The now rare tabes dorsalis causes impairment of deep and superficial pain sensation. Transient, spontaneous "lightning" pains may develop.

Lesions of the *nerve root*, most often due to compression, are accompanied by diminution or loss of sensation, pain, or paresthesias, but the distribution is segmental and corresponds to the involved dermatome (Figure 36.5). As with focal neuropathy, in compressive radiculopathy, the touch deficit is larger and often corresponds better to the published dermatome than the pinprick deficit. Pain may be either constant or intermittent and is often sharp, stabbing, and lancinating. It is increased by movement, coughing, or straining. There may be either hypalgesia or hyperalgesia. Examination may disclose root compression signs (see Chapter 47). Because of dermatome overlap, sensory changes may be difficult to demonstrate if only one root is involved.

With lesions of the *spinal cord* and *brainstem*, impairment of one or more modalities of sensation, or perversions of sensation in the form of either pain or paresthesias, may develop. Different sensory patterns may occur with myelopathy, for example, transverse syndrome, central cord syndrome, posterior column syndrome, Brown-Séquard syndrome,

anterior cord syndrome, or conus medullaris syndrome (see Chapters 24 and 47). With a transverse cord syndrome, the area of sensory involvement may involve all levels below the lesion, but occasionally, the sensory level is well below the level of the lesion; a sensory level on the trunk has been reported in lesions of the lower brainstem. Band-like radicular pain, paresthesias, or sensory loss may occur at the level of the lesion. Sacral sparing may be seen with intramedullary lesions. Spondylotic compression of the cervical spinal cord may cause glove-distribution sensory loss in the hands. Sensory loss is usually dissociated, with impairment of certain modalities and sparing of others. Because of the redundancy of the touch pathways, pain and temperature testing may be more useful than tactile sensation in evaluating CNS disease. Testing for the ability to detect the direction of skin movement above and below the level of the lesion and searching for a vibratory level may be helpful. Suspended dissociated sensory loss occurs in syringomyelia.

Lesions high in the cervical spinal cord and in the medulla may impair kinesthetic sensation in the upper extremities more than in the lower. As a result of the disturbance of proprioceptive sensations and a raised threshold for cutaneous senses, there may be stereoanesthesia, a term occasionally used when the difficulty results from infracerebral lesions, which is difficult to differentiate from astereognosis. Extinction and even autotopagnosia may be present with such lesions. Patients with pontine, medullary, or spinal cord lesions occasionally experience "central" pain. Lhermitte's sign, sudden electric-like or painful sensations spreading down the body or into the back or extremities on flexion of the neck due to involvement of the posterior columns, may occur with focal lesions of the cervical cord, multiple sclerosis, or other degenerative processes.

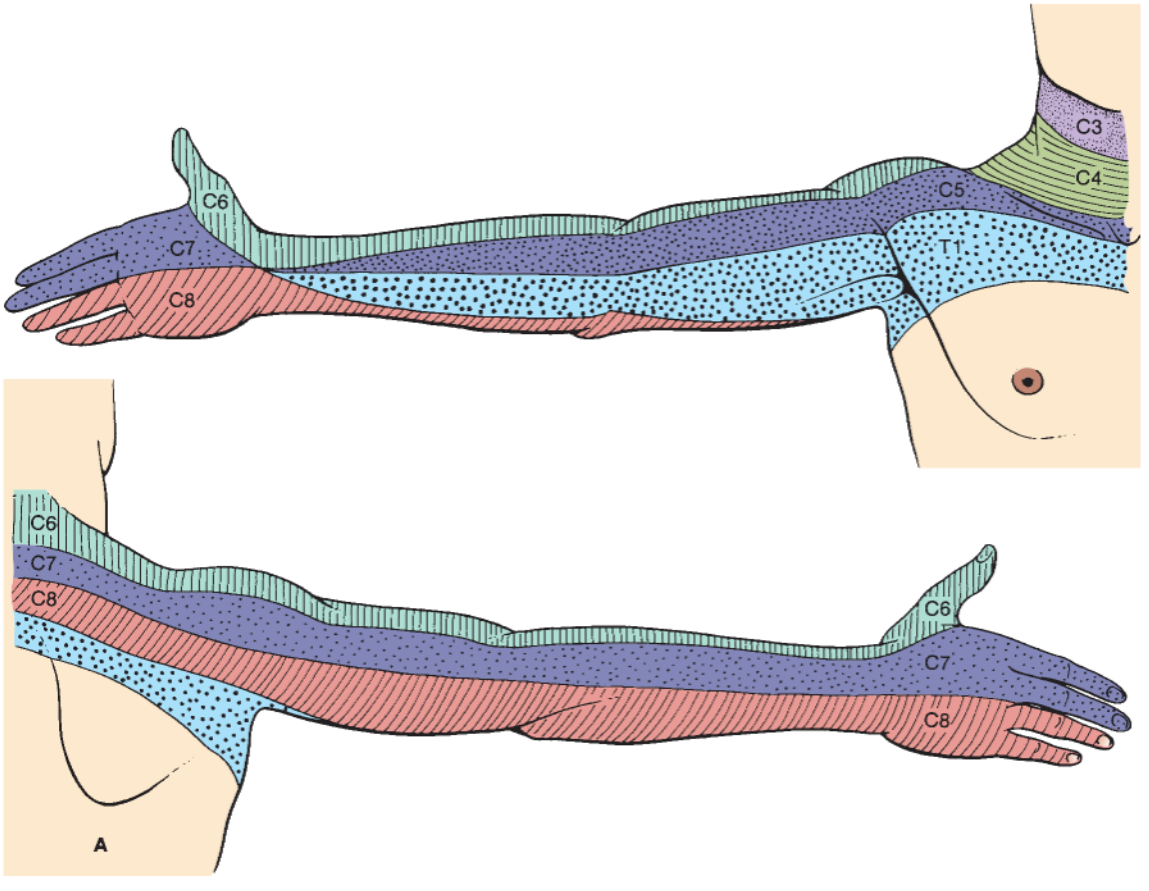


FIGURE 36.5 The segmental innervation. **A.** The upper extremity. *(continued)*

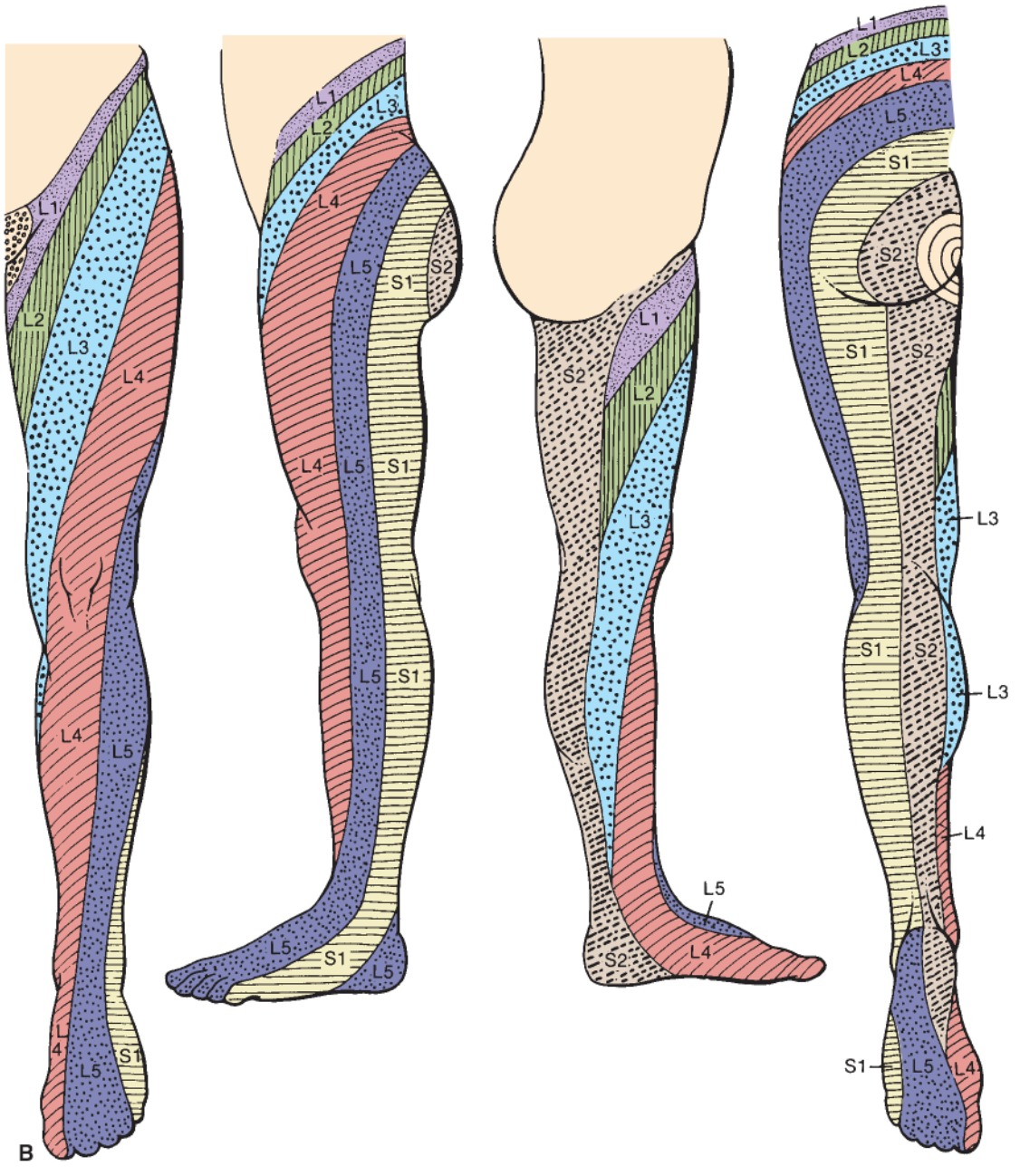


FIGURE 36.5 *(Continued)* **B.** The lower extremity.

The pattern of sensory return with recovering spinal lesions is variable; the impairment may recede downward in a segmental manner; the return may start in the sacral distribution and ascend, or there may be a gradual recovery of function over the entire affected area. Pressure sensation returns first and its recovery is usually the most complete, followed, in turn, by tactile, pain, cold, and heat sensibilities.

Sensory impulses that enter consciousness for interpretation by the parietal cortex must first pass through the *thalamus*. The thalamus is thought to be the end-station for pain, heat, cold, and heavy contact, where sensory impulses produce a crude, uncritical