Chapter 25: Numbness, Tingling, and Sensory Loss

Stephen L. Hauser

INTRODUCTION

Normal somatic sensation reflects a continuous monitoring process, little of which reaches consciousness under ordinary conditions. By contrast, disordered sensation, particularly when experienced as painful, is alarming and dominates the patient's attention. Physicians should be able to recognize abnormal sensations by how they are described, know their type and likely site of origin, and understand their implications. **Pain is considered separately in Chap. 13.**

POSITIVE AND NEGATIVE SYMPTOMS

Abnormal sensory symptoms can be divided into two categories: positive and negative. The prototypical positive symptom is tingling (pins and needles); other positive sensory phenomena include itch and altered sensations that are described as pricking, bandlike, lightning-like shooting feelings (lancinations), aching, knifelike, twisting, drawing, pulling, tightening, burning, searing, electrical, or raw feelings. Such symptoms are often painful.

Positive phenomena usually result from trains of impulses generated at sites of lowered threshold or heightened excitability along a peripheral or central sensory pathway. The nature and severity of the abnormal sensation depend on the number, rate, timing, and distribution of ectopic impulses and the type and function of nervous tissue in which they arise. Because positive phenomena represent excessive activity in sensory pathways, they are not necessarily associated with a sensory deficit (loss) on examination.

Negative phenomena represent loss of sensory function and are characterized by diminished or absent feeling that often is experienced as numbness and by abnormal findings on sensory examination. In disorders affecting peripheral sensation, at least one-half of the afferent axons innervating a particular site are probably lost or functionless before a sensory deficit can be demonstrated by clinical examination. If the rate of loss is slow, however, lack of cutaneous feeling may be unnoticed by the patient and difficult to demonstrate on examination, even though few sensory fibers are functioning; if it is rapid, both positive and negative phenomena are usually conspicuous. Subclinical degrees of sensory dysfunction may be revealed by sensory nerve conduction studies or somatosensory-evoked potentials.

Whereas sensory symptoms may be either positive or negative, sensory signs on examination are always a measure of negative phenomena.

TERMINOLOGY

Paresthesias and dysesthesias are general terms used to denote positive sensory symptoms. The term *paresthesias* typically refers to tingling or pins-and-needles sensations but may include a wide variety of other abnormal sensations, except pain; it sometimes implies that the abnormal sensations are perceived spontaneously. The more general term *dysesthesias* denotes all types of abnormal sensations, including painful ones, regardless of whether a stimulus is evident.

Another set of terms refers to sensory abnormalities found on examination. *Hypesthesia* or *hypoesthesia* refers to a reduction of cutaneous sensation to a specific type of testing such as pressure, light touch, and warm or cold stimuli; *anesthesia,* to a complete absence of skin sensation to the same stimuli plus pinprick; and *hypalgesia* or *analgesia*, to reduced or absent pain perception (nociception). *Hyperesthesia* means pain or increased sensitivity in response to touch. Similarly, *allodynia* describes the situation in which a nonpainful stimulus, once perceived, is experienced as painful, even excruciating. An example is elicitation of a painful sensation by application of a vibrating tuning fork. *Hyperalgesia* denotes severe pain in response to a mildly noxious stimulus, and *hyperpathia*, a broad term, encompasses all the phenomena described by hyperesthesia, allodynia, and hyperalgesia. With hyperpathia, the threshold for a sensory stimulus is increased and perception is delayed, but once felt, it is unduly painful.

Disorders of deep sensation arising from muscle spindles, tendons, and joints affect proprioception (position sense). Manifestations include imbalance (particularly with eyes closed or in the dark), clumsiness of precision movements, and unsteadiness of gait, which are referred to collectively as *sensory ataxia*. Other findings on examination usually, but not invariably, include reduced or absent joint position and vibratory sensibility and absent deep tendon reflexes in the affected limbs. The Romberg sign is positive, which means that the patient sways markedly or topples when asked to stand with feet close together and eyes closed. In severe states of deafferentation involving deep sensation, the patient cannot walk or stand unaided or even sit unsupported. Continuous involuntary movements (*pseudoathetosis*) of the outstretched hands and fingers occur, particularly with eyes closed.

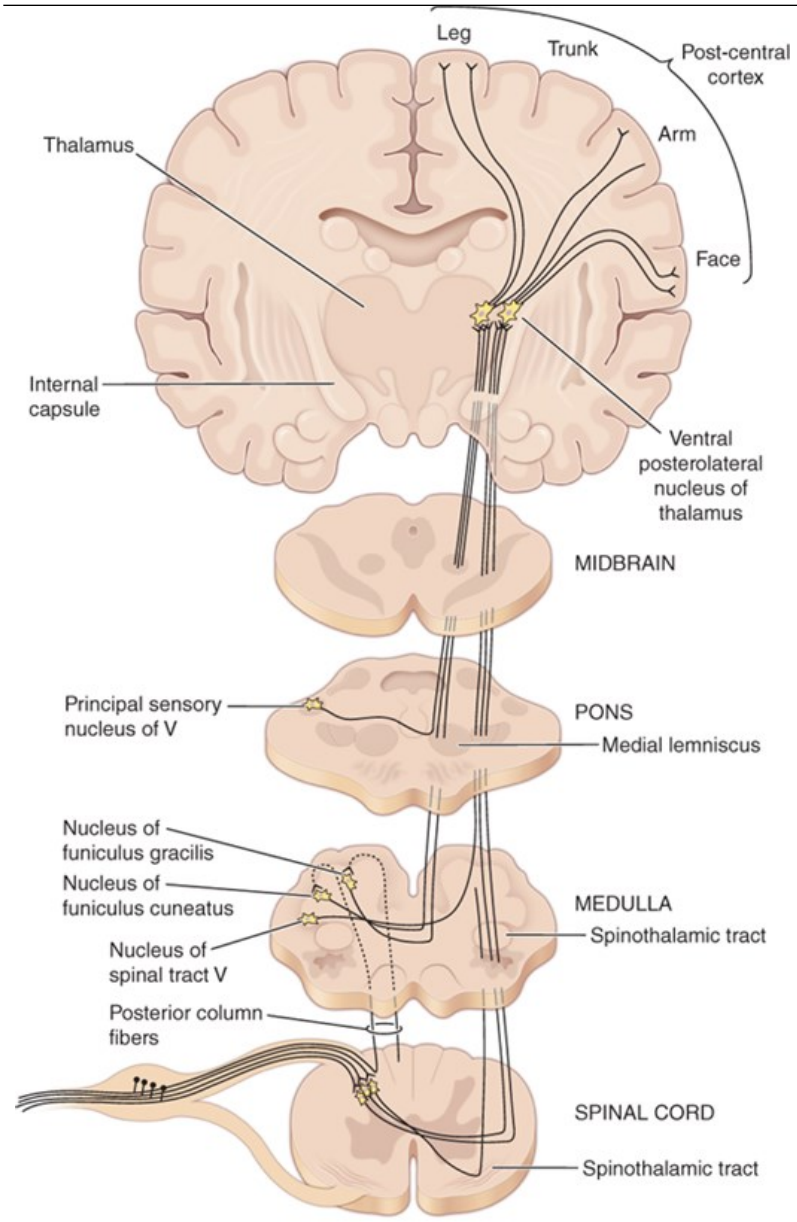
ANATOMY OF SENSATION

Cutaneous receptors are classified by the type of stimulus that optimally excites them. They consist of naked nerve endings (nociceptors, which respond to tissue-damaging stimuli, and thermoreceptors, which respond to noninjurious thermal stimuli) and encapsulated terminals (several types of mechanoreceptor, activated by physical deformation of the skin or stretch of muscles). Each type of receptor has its own set of sensitivities to specific stimuli, size and distinctness of receptive fields, and adaptational qualities.

Afferent peripheral nerve fibers conveying somatosensory information from the limbs and trunk traverse the dorsal roots and enter the dorsal horn of the spinal cord **(Fig. 25-1)**; the cell bodies of first-order neurons are located in the dorsal root ganglia (DRG). In an analogous fashion, sensations from the face and head are conveyed through the trigeminal system (Fig. 441-2). Once fiber tracts enter the spinal cord, the polysynaptic projections of the smaller fibers (unmyelinated and small myelinated), which subserve mainly nociception, itch, temperature sensibility, and touch, cross and ascend in the opposite anterior and lateral columns of the spinal cord, through the brainstem, to the ventral posterolateral (VPL) nucleus of the thalamus and ultimately project to the postcentral gyrus of the parietal cortex and other cortical areas **(Chap. 13)**. This is the *spinothalamic pathway* or *anterolateral system*. The larger fibers, which subserve tactile and position sense and kinesthesia, project rostrally in the posterior and posterolateral columns on the same side of the spinal cord and make their first synapse in the gracile or cuneate nucleus of the lower medulla. Axons of second-order neurons decussate and ascend in the medial lemniscus located medially in the medulla and in the tegmentum of the pons and midbrain and synapse in the VPL nucleus; third-order neurons project to parietal cortex as well as to other cortical areas. This large-fiber system is referred to as the *posterior column–medial lemniscal pathway* (lemniscal, for short). Although the fiber types and functions that make up the spinothalamic and lemniscal systems are relatively well known, many other fibers, particularly those associated with touch, pressure, and position sense, ascend in a diffusely distributed pattern both ipsilaterally and contralaterally in the anterolateral quadrants of the spinal cord. This explains why a complete lesion of the posterior columns of the spinal cord may be associated with little sensory deficit on examination.

FIGURE 25-1

The main somatosensory pathways. The spinothalamic tract (pain, thermal sense) and the posterior column–lemniscal system (touch, pressure, joint position) are shown. Offshoots from the ascending anterolateral fasciculus (spinothalamic tract) to nuclei in the medulla, pons, and mesencephalon and nuclear terminations of the tract are indicated. *(Reproduced with permission from AH Ropper, MA Samuels: Adams and Victor's Principles of Neurology, 9th ed. New York, McGraw-Hill, 2009.)*



Source: Joseph Loscalzo, Anthony Fauci, Dennis Kasper, Stephen Hauser, Dan Longo, J. Larry Jameson: Harrison's Principles of Internal Medicine, 21e

APPROACH TO THE PATIENT WITH CLINICAL EXAMINATION OF SENSATION

The main components of the sensory examination are tests of primary sensation (pain, touch, vibration, joint position, and thermal sensation) **(Table 25-1)**. The examiner must depend on patient responses, and this complicates interpretation. Further, examination may be limited in some patients. In a stuporous patient, for example, sensory examination is reduced to observing the briskness of withdrawal in response to a pinch or another noxious stimulus. Comparison of responses on the two sides of the body is essential. In an alert but uncooperative patient, it may not be possible to examine cutaneous sensation, but some idea of proprioceptive function may be gained by noting the patient's best performance of movements requiring balance and precision.

TABLE 25-1

Testing Primary Sensation

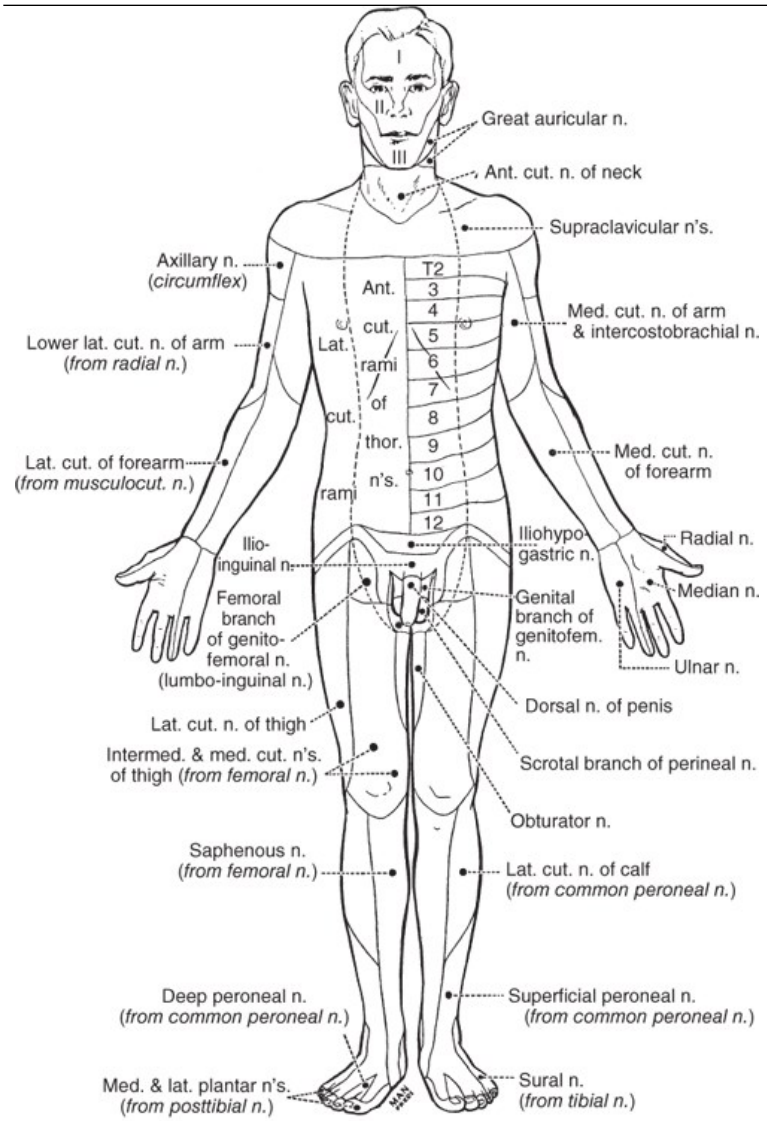
SENSE	TEST DEVICE	ENDINGS ACTIVATED	FIBER SIZE MEDIATING	CENTRAL PATHWAY
Pain	Pinprick	Cutaneous nociceptors	Small	SpTh, also D
Temperature, heat	Warm metal object	Cutaneous thermoreceptors for hot	Small	SpTh
Temperature, cold	Cold metal object	Cutaneous thermoreceptors for cold	Small	SpTh
Touch	Cotton wisp, fine brush	Cutaneous mechanoreceptors, also naked endings	Large and small	Lem, also D and SpTh
Vibration	Tuning fork, 128 Hz	Mechanoreceptors, especially pacinian corpuscles	Large	Lem, also D
Joint position	Passive movement of specific joints	Joint capsule and tendon endings, muscle spindles	Large	Lem, also D

Abbreviations: D, diffuse ascending projections in ipsilateral and contralateral anterolateral columns; Lem, posterior column and lemniscal projection, ipsilateral; SpTh, spinothalamic projection, contralateral.

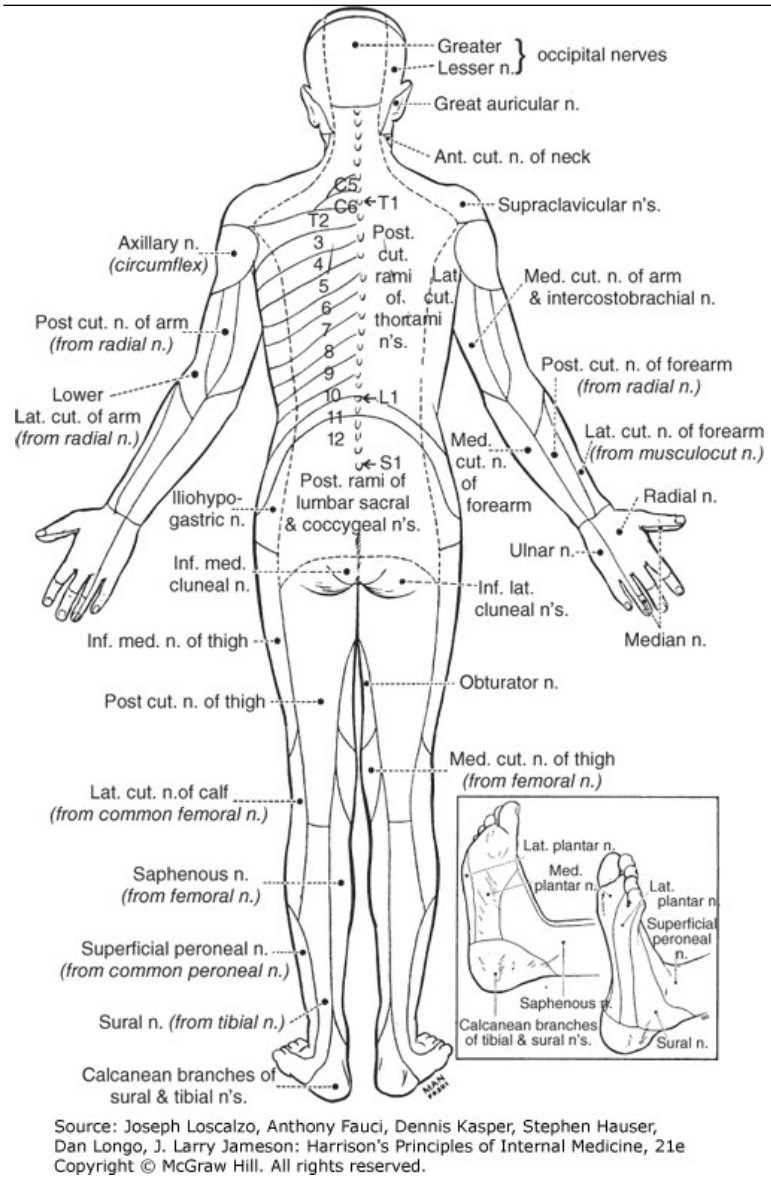
In patients with sensory complaints, testing should begin in the center of the affected region and proceed radially until sensation is perceived as normal. The distribution of any abnormality is defined and compared to root and peripheral nerve territories **(Figs. 25-2 and 25-3)**. Some patients present with sensory symptoms that do not fit an anatomic localization and are accompanied by either no abnormalities or gross inconsistencies on examination. The examiner should consider in such cases the possibility of a psychologic cause (see "Psychogenic Symptoms," below). Sensory examination of a patient who has no neurologic complaints can be brief and consist of pinprick, touch, and vibration testing in the hands and feet plus evaluation of stance and gait, including the Romberg maneuver **(Chap. V6)**. Evaluation of stance and gait also tests the integrity of motor and cerebellar systems.

FIGURE 25-2

The cutaneous fields of peripheral nerves. *(Reproduced with permission from W Haymaker, B Woodhall: Peripheral Nerve Injuries, 2nd ed. Philadelphia, Saunders, 1953.)*



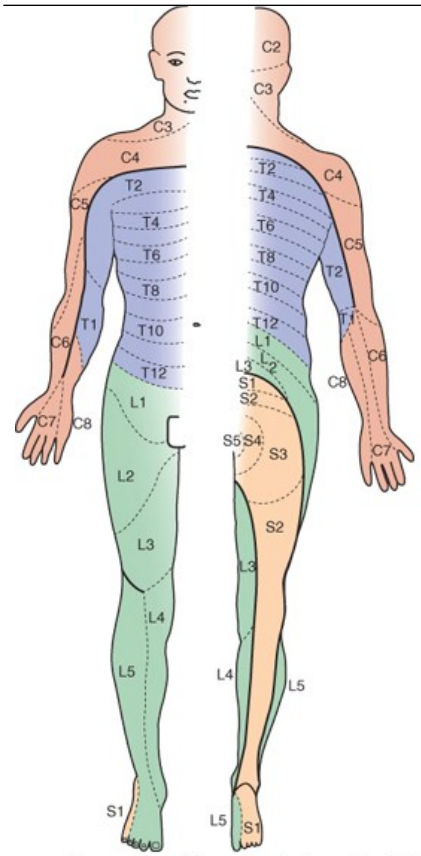
Source: Joseph Loscalzo, Anthony Fauci, Dennis Kasper, Stephen Hauser, Dan Longo, J. Larry Jameson: Harrison's Principles of Internal Medicine, 21e



Source: Joseph Loscalzo, Anthony Fauci, Dennis Kasper, Stephen Hauser, Dan Longo, J. Larry Jameson: Harrison's Principles of Internal Medicine, 21e

FIGURE 25-3

Distribution of the sensory spinal roots on the surface of the body (dermatomes). *(Reproduced with permission from D Sinclair: Mechanisms of Cutaneous Sensation. Oxford, UK, Oxford University Press, 1981 through PLS Clear.)*



Source: Joseph Loscalzo, Anthony Fauci, Dennis Kasper, Stephen Hauser, Dan Longo, J. Larry Jameson: Harrison's Principles of Internal Medicine, 21e

Primary Sensation

The sense of pain usually is tested with a clean pin, which is then discarded. The patient is asked to close the eyes and focus on the pricking or unpleasant quality of the stimulus, not just the pressure or touch sensation elicited. Areas of hypalgesia should be mapped by proceeding radially from the most hypalgesic site. Temperature sensation to both hot and cold is best tested with small containers filled with water of the desired temperature. An alternative way to test cold sensation is to touch a metal object, such as a tuning fork at room temperature, to the skin. For testing warm temperatures, the tuning fork or another metal object may be held under warm water of the desired temperature and then used. The appreciation of both cold and warmth should be tested because different receptors respond to each. Touch usually is tested with a wisp of cotton, minimizing pressure on the skin. In general, it is better to avoid testing touch on hairy skin because of the profusion of the sensory endings that surround each hair follicle. The patient is tested with the eyes closed and should respond as soon as the stimulus is perceived, indicating its location.

Joint position testing is a measure of proprioception. With the patient's eyes closed, joint position is tested in the distal interphalangeal joint of the great toe and fingers. The digit is held by its sides, distal to the joint being tested, and moved passively while more proximal joints are stabilized—the patient indicates the change in position or direction of movement. If errors are made, more proximal joints are tested. A test of proximal joint position sense, primarily at the shoulder, is performed by asking the patient to bring the two index fingers together with arms extended and eyes closed. Normal individuals can do this accurately, with errors of 1 cm or less.

The sense of vibration is tested with an oscillating tuning fork that vibrates at 128 Hz. Vibration is tested over bony points, beginning distally; in the feet, it is tested over the dorsal surface of the distal phalanx of the big toes and at the malleoli of the ankles, and in the hands, it is tested dorsally at the distal phalanx of the fingers. If abnormalities are found, more proximal sites should be examined. Vibratory thresholds at the same site in the patient and the examiner may be compared for control purposes.

Cortical Sensation

The most commonly used tests of cortical function are two-point discrimination, touch localization, and bilateral simultaneous stimulation, and tests for graphesthesia and stereognosis. Abnormalities of these sensory tests, in the presence of normal primary sensation in an alert cooperative patient, signify a lesion of the parietal cortex or thalamocortical projections. If primary sensation is altered, these cortical discriminative functions usually will be abnormal also. Comparisons should always be made between analogous sites on the two sides of the body because the deficit with a specific parietal lesion is likely to be unilateral.

Two-point discrimination can be tested with calipers, the points of which may be set from 2 mm to several centimeters apart and then applied simultaneously to the test site. On the fingertips, a normal individual can distinguish about a 3-mm separation of points.

Touch localization is performed by light pressure for an instant with the examiner's fingertip or a wisp of cotton wool; the patient, whose eyes are closed, is required to identify the site of touch. *Bilateral simultaneous stimulation* at analogous sites (e.g., the dorsum of both hands) can be carried out to determine whether the perception of touch is extinguished consistently on one side (*extinction* or *neglect*). *Graphesthesia* refers to the capacity to recognize, with eyes closed, letters or numbers drawn by the examiner's fingertip on the palm of the hand. Once again, interside comparison is of prime importance. Inability to recognize numbers or letters is termed *agraphesthesia*.

Stereognosis refers to the ability to identify common objects by palpation, recognizing their shape, texture, and size. Common standard objects such as keys, paper clips, and coins are best used. Patients with normal stereognosis should be able to distinguish a dime from a penny and a nickel from a quarter without looking. Patients should feel the object with only one hand at a time. If they are unable to identify it in one hand, it should be placed in the other for comparison. Individuals who are unable to identify common objects and coins in one hand but can do so in the other are said to have *astereognosis* of the abnormal hand.

Quantitative Sensory Testing

Effective sensory testing devices are commercially available. Quantitative sensory testing is particularly useful for serial evaluation of cutaneous sensation in clinical trials. Threshold testing for touch and vibratory and thermal sensation is the most widely used application.

Electrodiagnostic Studies and Nerve Biopsy

Nerve conduction studies and nerve biopsy are important means of investigating the peripheral nervous system, but they do not evaluate the function or structure of cutaneous receptors and free nerve endings or of unmyelinated or thinly myelinated nerve fibers in the nerve trunks. Skin biopsy can be used to evaluate these structures in the dermis and epidermis.

LOCALIZATION OF SENSORY ABNORMALITIES

Sensory symptoms and signs can result from lesions at many different levels of the nervous system from the parietal cortex to the peripheral sensory receptor. Noting their distribution and nature is the most important way to localize their source. Their extent, configuration, symmetry, quality, and severity are the key observations.

Dysesthesias without sensory findings by examination may be difficult to interpret. To illustrate, tingling dysesthesias in an acral distribution (hands and feet) can be systemic in origin, for example, secondary to hyperventilation, or induced by a medication such as acetazolamide. Distal dysesthesias can also be an early event in an evolving polyneuropathy or may herald a myelopathy, such as from vitamin B_{12} deficiency. Sometimes, distal dysesthesias have no definable basis. In contrast, dysesthesias that correspond in distribution to that of a particular peripheral nerve structure denote a lesion at that site. For instance, dysesthesias restricted to the fifth digit and the adjacent one-half of the fourth finger on one hand reliably point to disorder of the ulnar nerve, most commonly at the elbow.

Nerve and Root

In focal nerve trunk lesions, sensory abnormalities are readily mapped and generally have discrete boundaries (Figs. 25-2 and 25-3). Root ("radicular") lesions frequently are accompanied by deep, aching pain along the course of the related nerve trunk. With compression of a fifth lumbar (L5) or first sacral (S1) root, as from a ruptured intervertebral disk, sciatica (radicular pain relating to the sciatic nerve trunk) is a common manifestation **(Chap. 17)**. With a lesion affecting a single root, sensory deficits may be minimal or absent because adjacent root territories overlap extensively.

Isolated mononeuropathies may cause symptoms beyond the territory supplied by the affected nerve, but abnormalities on examination typically are confined to expected anatomic boundaries. In multiple mononeuropathies, symptoms and signs occur in discrete territories supplied by different individual nerves and—as more nerves are affected—may simulate a polyneuropathy if deficits become confluent. With polyneuropathies, sensory deficits are generally graded, distal, and symmetric in distribution **(Chap. 446)**. Dysesthesias, followed by numbness, begin in the toes and ascend symmetrically. When dysesthesias reach the knees, they usually also have appeared in the fingertips. The process is nerve length–dependent, and the deficit is often described as "stocking glove" in type. Involvement of both hands and feet also occurs with lesions of the upper cervical cord or the brainstem, but an upper level of the sensory disturbance may then be found on the trunk and other evidence of a central lesion may be present, such as sphincter involvement or signs of an upper motor neuron lesion **(Chap. 24)**. Although most polyneuropathies are pansensory and affect all modalities of sensation, selective sensory dysfunction according to nerve fiber size may occur. Small-fiber polyneuropathies are characterized by burning, painful dysesthesias with reduced pinprick and thermal sensation but with sparing of proprioception, motor function, and deep tendon reflexes. Touch is involved variably; when it is spared, the sensory pattern is referred to as exhibiting *sensory dissociation*. Sensory dissociation may occur also with spinal cord lesions **(Chap. 442)**. Large-fiber polyneuropathies are characterized by vibration and position sense deficits, imbalance, absent tendon reflexes, and variable motor dysfunction but preservation of most cutaneous sensation. Dysesthesias, if present at all, tend to be tingling or bandlike in quality.

Sensory neuronopathy (or ganglionopathy) is characterized by widespread but asymmetric sensory loss occurring in a non-length-dependent manner so that it may occur proximally or distally, and in the arms, legs, or both. Pain and numbness progress to sensory ataxia and impairment of all sensory modalities over time. This condition is usually paraneoplastic or idiopathic in origin **(Chaps. 94 and 445)** or related to an autoimmune disease, particularly Sjögren's syndrome **(Chap. 361)**.

Spinal Cord

(See also Chap. 442) If the spinal cord is transected, all sensation is lost below the level of transection. Bladder and bowel function also are lost, as is motor function. Lateral hemisection of the spinal cord produces the Brown-Séquard syndrome, with absent pain and temperature sensation contralaterally and loss of proprioceptive sensation and power ipsilaterally below the lesion (see Figs. 25-1 and **442-1**); ipsilateral pain or hyperesthesia may also occur.

Numbness or paresthesias in both feet may arise from a spinal cord lesion; this is especially likely when the upper level of the sensory loss extends to the trunk. When all extremities are affected, the lesion is probably in the cervical region or brainstem unless a peripheral neuropathy is responsible. The presence of upper motor neuron signs **(Chap. 24)** supports a central lesion; a hyperesthetic band on the trunk may suggest the level of involvement.

A dissociated sensory loss can reflect spinothalamic tract involvement in the spinal cord, especially if the deficit is unilateral and has an upper level on the torso. Bilateral spinothalamic tract involvement occurs with lesions affecting the center of the spinal cord, such as in syringomyelia. There is a dissociated sensory loss with impairment of pinprick and temperature appreciation but relative preservation of light touch, position sense, and vibration appreciation.

Dysfunction of the posterior columns in the spinal cord or of the posterior root entry zone may lead to a bandlike sensation around the trunk or a feeling of tight pressure in one or more limbs. Flexion of the neck sometimes leads to an electric shock–like sensation that radiates down the back and into the legs (Lhermitte's sign) in patients with a cervical lesion affecting the posterior columns, such as from multiple sclerosis, cervical spondylosis, or following irradiation to the cervical region.

Brainstem

Crossed patterns of sensory disturbance, in which one side of the face and the opposite side of the body are affected, localize to the lateral medulla. Here a small lesion may damage both the ipsilateral descending trigeminal tract and the ascending spinothalamic fibers subserving the opposite arm, leg, and hemitorso (see "Lateral medullary syndrome" in **Fig. 426-7**). A lesion in the tegmentum of the pons and midbrain, where the lemniscal and spinothalamic tracts merge, causes pansensory loss contralaterally.

Thalamus

Hemisensory disturbance with tingling numbness from head to foot is often thalamic in origin but also can arise from the anterior parietal region. If

abrupt in onset, the lesion is likely to be due to a small stroke (lacunar infarction), particularly if localized to the thalamus. Occasionally, with lesions affecting the VPL nucleus or adjacent white matter, a syndrome of thalamic pain, also called *Déjerine-Roussy syndrome*, may ensue. The persistent, unrelenting unilateral pain often is described in dramatic terms.

Cortex

With lesions of the parietal lobe involving either the cortex or subjacent white matter, the most prominent symptoms are contralateral hemineglect, hemi-inattention, and a tendency not to use the affected hand and arm. On cortical sensory testing (e.g., two-point discrimination, graphesthesia), abnormalities are often found but primary sensation is usually intact. Anterior parietal infarction may present as a pseudothalamic syndrome with contralateral loss of primary sensation from head to toe. Dysesthesias or a sense of numbness and, rarely, a painful state may also occur.

Focal Sensory Seizures

These seizures generally are due to lesions in the area of the postcentral or precentral gyrus. The principal symptom of focal sensory seizures is tingling, but additional, more complex sensations may occur, such as a rushing feeling, a sense of warmth, or a sense of movement without detectable motion. Symptoms typically are unilateral; commonly begin in the arm or hand, face, or foot; and often spread in a manner that reflects the cortical representation of different bodily parts, as in a Jacksonian march. Their duration is variable; seizures may be transient, lasting only for seconds, or persist for an hour or more. Focal motor features may supervene, often becoming generalized with loss of consciousness and tonic-clonic jerking.

Psychogenic Symptoms

Sensory symptoms may have a psychogenic basis. Such symptoms may be generalized or have an anatomic boundary that is difficult to explain neurologically, for example, circumferentially at the groin or shoulder or around a specific joint. Pain is common, but the nature and intensity of any sensory disturbances are variable. The diagnosis should not be one of exclusion but based on suggestive findings that are otherwise difficult to explain, such as midline splitting of impaired vibration, pinprick, or light touch appreciation; variability or poor reproducibility of sensory deficits; or normal performance of tasks requiring sensory input that is seemingly abnormal on formal testing, such as good performance with eyes closed of the finger-to-nose test despite an apparent loss of position sense in the upper limb. The side with abnormal sensation may be confused when the limbs are placed in an unusual position, such as crossed behind the back. Sensory complaints should not be regarded as psychogenic simply because they are unusual.

TREATMENT

Management is based on treatment of the underlying condition. Symptomatic treatment of acute and chronic pain is discussed in **Chap. 13**. Dysesthesias, when severe and persistent, may respond to anticonvulsants (carbamazepine, 100–1000 mg/d; gabapentin, 300–3600 mg/d; or pregabalin, 50–300 mg/d), antidepressants (amitriptyline, 25–150 mg/d; nortriptyline, 25–150 mg/d; desipramine, 100–300 mg/d; or venlafaxine, 75–225 mg/d).

ACKNOWLEDGMENTS

The editors acknowledge the contributions of Michael J. Aminoff to earlier editions of this chapter.

FURTHER READING

Brazis P et al: *Localization in Clinical Neurology*, 7th ed. Philadelphia, Lippincott William & Wilkins, 2016.

Campbell WW, Barohn RJ: *DeJong's the Neurologic Examination*, 8th ed. Philadelphia, Wolters Kluwer, 2020.

Waxman S: *Clinical Neuroanatomy*, 29th ed. New York, McGraw Hill Education, 2020.