2 THE SOMATOSENSORY SYSTEMS

CHAPTER OVERVIEW

The spinal cord encompasses three major ascending sensory pathways that provide ongoing feedback from essentially all of our body, with the exception of our face and parts of our head. Much, although not all, of this sensory input is consciously perceived and provides us with critical information not only about our immediate environment, but about the state and integrity of our bodies. This information is provided by two separate, but complementary systems; the **anterolateral** or **spinothalamic**, and what has been termed the **posterior column** or **lemniscal** system. The spinothalamic system allows us to perceive pain and temperature, as well as pressure or crude touch. In addition to giving us potentially useful information about these aspects of our environment, this system also provides a protective function by serving as the afferent limb for withdrawal reflexes in the presence of potentially harmful stimuli. The lemniscal system, on the other hand, allows us to make fine tactile discriminations (stereognosis), to be aware of the position and movement of our limbs in space, and is probably our primary means of detecting vibrations (other than sound). In addition to these "conscious" perceptions, other ascending pathways provide constant sensory feedback to the cerebellum that apparently bypasses conscious awareness. Such information is critical in maintaining posture and balance, especially while engaging in whole-body activities.

By the conclusion of this chapter the reader should be able to identify the major ascending pathways, both in terms of their general localization within the cord and general functional correlates. In addition, one should be able to describe the basic elements of a somatosensory examination, as well as the nature and clinical significance of abnormal somatosensory findings. Finally, having reviewed the basic anatomical structure of the spinal cord, as well as its major ascending and descending pathways, the reader should be able to identify and appreciate the anatomical basis for some of the more common spinal and peripheral nerve syndromes described in the chapter.

INTRODUCTION

In order to function effectively one needs constant sensory feedback from the external environment, as well as information about conditions that directly impact on one's body. Additionally, it is essential that one maintain awareness of the body in space (e.g., the position and/or movement of one's limbs through the time-space continuum). One also must modulate internal states or homeostatic mechanisms. These functions are accomplished through multiple afferent or sensory feedback systems, some of which operate at a conscious level, others at an unconscious level.

Some stimuli remain essentially detached from us. That is, we become aware of them only indirectly through what are referred to as telereceptors. These include the senses of hearing and vision. Olfaction may be considered a telereceptor as through this sense one can be aware of the presence of a sweet olive tree or a garbage dump from a sizable distance away. On the other hand, odor is perceived when airborne molecules of the object or substance come into direct contact with our nasal mucosa—not always a particularly comforting or pleasant thought.

Other external stimuli are perceived only when they come into direct contact with the body. This would include most tactile sensations such as touch, pressure, temperature, external painful stimuli, and vibration. However, under certain conditions, low-frequency, high-amplitude vibrations may be perceived at considerable distance from their source via pressure waves. Finally, there are those sensations that derive not from external sources or forces but rather from internal conditions or states. In contrast to the first two classes of stimuli for which one is either generally aware or potentially aware, stimuli that arise from within the body may or may not ever reach conscious awareness. In general, this probably has less to do with the nature of the stimulus than with the pathways that carry such information. Thus, awareness of the position of one's limbs may reach conscious awareness when such signals are conveyed through the dorsal columns of the cord through the thalamus and to the cortex. Signals from the same or similar peripheral receptors that synapse locally in the cord or travel through the spinocerebellar tracts never reach conscious levels, but still have a meaningful impact on certain aspects of motor coordination, maintaining muscle tone, and providing the afferent loops necessary for postural and other reflexes.

The special senses mediated by the cranial nerves will be discussed in Chapter 5. These include vision, hearing, taste, and olfaction, and what might be considered a sixth sense: awareness of one's orientation in or movement through space (vestibular). In the current chapter, the emphasis will be on general somatic perceptions or the somatosensory system. This includes the perception of simple touch, discriminative touch (stereognosis), pain, temperature, proprioception (awareness of the position of the limbs, head, or trunk), kinesthesia (awareness of movement), and vibration. While this chapter will focus primarily on those tracts that ascend in the cord, it will be noted that comparable mechanisms serve these same types of stimuli applied to the head or face. More detail about the neural pathways for the latter will be presented in Chapter 5 (The Cranial Nerves). Finally, in Chapters 9 (The Cerebal Cortex) and 10 (The Cerebral Vascular System), there will be a discussion of some of the more unique types of disturbances of somatic awareness that are peculiar to cortical lesions, namely, anosognosia (lack of awareness of sensorimotor deficits), Anton's syndrome (lack of awareness of blindness), finger agnosia (a specific type of autotopagnosia resulting in difficulty discriminating parts of one's body), and unilateral neglect (inattention to one half of the body or hemispace).

ASCENDING PATHWAYS

The ascending or afferent somatosensory pathways within the spinal cord, with the exception of those that primarily mediate position sense and stereognosis (the dorsal columns), are intermingled with descending motor pathways in the lateral and ventral funiculi (see Figure 1–2, Chapter 1).

The ascending pathways can be divided into three major, though not always totally distinct, systems:

1. The **anterolateral system,** which tends to be more primitive and polysynaptic and is primarily responsible for the sensations of pain, temperature, and crude (i.e., less well defined) or simple touch.
2. The **lemniscal system,** which is represented by the dorsal column pathways or dorsal funiculus, has fewer synaptic connections than the anterolateral pathways and conveys conscious information about position sense, kinesthesia, vibration, and the finer aspects of tactual discrimination.
3. The **spinocerebellar system,** which unlike the other two systems, does not project to the thalamus, and hence does not reach conscious awareness. The latter system, however, may carry much of the same type of information as the other two pathways, particularly position sense and kinesthesia, which are essential for coordinated motor activity.

These systems can be further divided into those areas that are supplied by the cranial nerves versus those innervated via the dorsal spinal roots (see Table 2–1). Again, the former will be covered in more detail in Chapter 5.

Before discussing each of these systems in greater detail, it might be useful to review features that they all have in common. Most of the fibers contained in these systems arise from specialized receptors or nerve endings within the skin, muscles, or tendons. Those that supply the trunk and limbs travel centrally in the peripheral nerves, along with the motor or efferent fibers. Just outside the cord, they separate from the motor fibers and enter the cord as the dorsal nerve roots. The cell bodies for these latter fibers reside outside the cord in the dorsal root ganglia. All dorsal root fibers synapse after entering the cord. Most synapse

Table 2–1. Major Somatosensory Tracts

Tracts	*Origin*	*Termination*	*Function*
Spinothalamic (anterolateral system)	Free nerve endings; specialized skin receptors	Spinal cord, posterior horn; secondary fibers to VPL[a] nuclei; tertiary fibers to cortex	Mediates perception of pain, temperature, pressure or simple touch; spinal reflexes
Dorsal columns (lemniscal system)	Specialized skin receptors; muscle and joint receptors	Nuclei cuneatus and gracilis; secondary fibers to VPL; tertiary fibers to cortex	Proprioception, kinesthetic feedback, vibration, fine tactile discrimination; spinal reflexes
Spinocerebellar (dorsal, ventral, rostral, and cuneocerebellar)	Similar to the above	Cerebellum, primarily in the vermal region	Coordination, balance via somatosensory and kinesthetic feedback to cerebellum
Ventral (crossed) and dorsal (uncrossed)[b] trigeminothalamic	Similar to the above, but in an area of the face	Trigeminal ganglion; secondary fibers to VPM[c]; tertiary to cortex	Provides feedback comparable to posterior columns and spinothalamic tracts, but from the facial area[d]

[a] Ventral posterior lateral nuclei of the thalamus.
[b] Appears to carry information from the oral cavity.
[c] Ventral posterior medial nuclei of the thalamus.
[d] See Chapters 4 and 5 for more detail.

locally, that is, at or within a few segments from where they entered the cord; however, some of the fibers of the posterior columns ascend to the upper cord before synapsing. These synapses within the cord would appear to serve multiple purposes. As noted in Chapter 1, some of the afferent neurons synapse either directly or indirectly (via interneurons) on alpha or gamma neurons and serve to maintain muscle tone, mediate spinal reflexes, and provide the potential for the modulation of, or modulation by, descending or efferent fibers. Thus both sensory input and motor output can be influenced at the spinal level. This branching and interconnection of the afferent fibers also allow for the summation of sensory input from more than one nerve rootlet. The commonly observed phenomenon of attempting to relieve sharp pain by rubbing the affected area seems to be accomplished in large part by the interaction of the two types of stimuli in the substantia gelatinosa.

Anterolateral System

The anterolateral system is represented primarily by the **lateral** and **ventral spinothalamic tracts** (Figure 2–1). While these two tracts were once described as carrying different and distinct types of sensory information, the current thinking is that the two have extensive

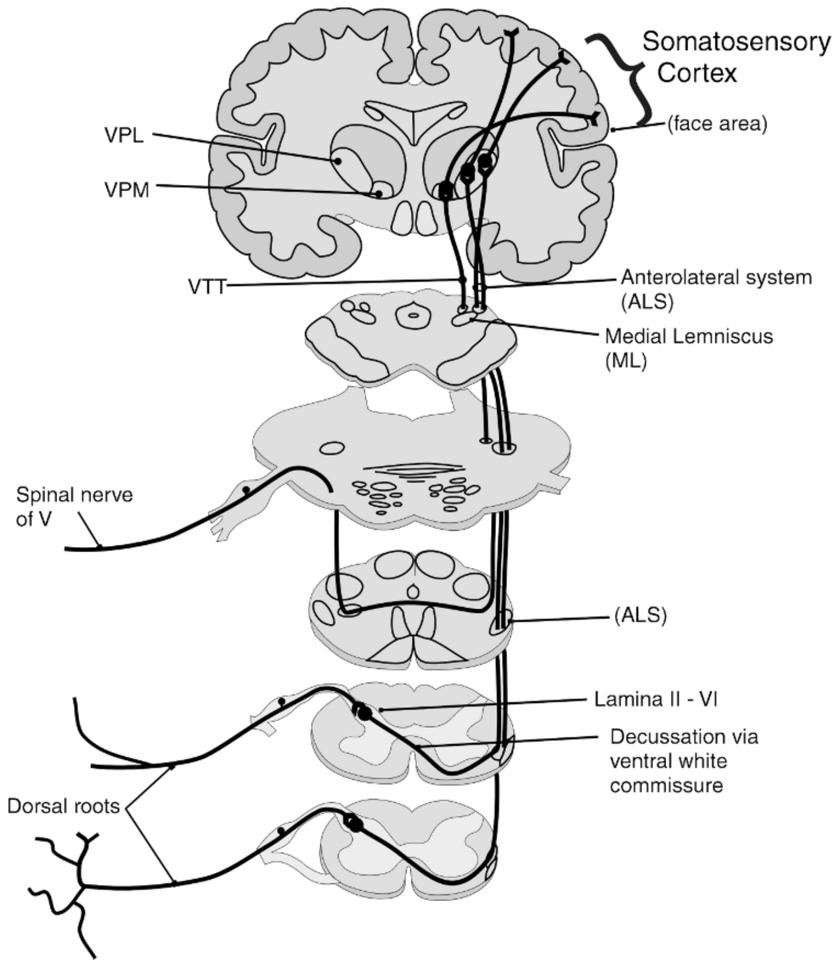


Figure 2–1. Fibers of the anterolateral (spinothalamic) system, which are important in pain and temperature perception, synapse in the dorsal horn and then cross the midline within a few segments of where they enter the cord to ascend in the lateral and ventral funiculi. Traveling in the ventrolateral and later in the dorsolateral portions of the brainstem, these second-order fibers terminate in the ventral posterior lateral (VPL) nuclei of the thalamus. From there, third-order neurons proceed to the cortex. Abbreviations: VPM, ventral posterior medial nucleus (thalamus); VPL, ventral posterior lateral nucleus; VTT, ventral trigeminothalamic tract.

functional overlap, and hence should be considered as a single system. It should be noted that the anterolateral system is most closely associated with, and probably most responsible for, the sensations of pain, temperature, and light touch, while the posterior columns (lemniscal system) are more closely associated with position sense and stereognosis. Some overlapping of functions is found within these two systems as well. In addition, some information shared by both of these systems is conveyed to the thalamus by the **lateral cervical system** (see below). One net effect of all this redundancy within the spinal cord is that unilateral lesions affecting a single tract or system unlikely will result in a total loss of the sense of touch. However, the finer aspects of tactile discrimination indeed will be generally severely compromised following lesions to the posterior columns. While loss of pain on one side of the body caudal to a lesion in the cord typically results from unilateral destruction of the anterolateral pathways (and hence, occasionally surgically produced to treat severe and otherwise intractable pain), the effect may not be permanent, again suggesting the possibility of alternate pain pathways.

Pain

The pathways for pain and temperature are difficult to separate, and hence, are usually considered together when discussed anatomically. The sensations of itching and tickling also are thought to be mediated by this system.

Unlike some of the other somatic sensations, there apparently are no specialized “pain receptors,” although some may respond chemically to specific substances released during injury to tissues. The sensation of pain probably is initiated at the periphery by small-diameter myelinated (A-delta) and unmyelinated (C) fibers. Each of these appear to be responsible for different aspects or types of pain: the former or the A-delta fibers being responsible for the perception of “fast,” “sharp,” well-localized pain, while the C fibers carry the “slow,” “dull,” aching or burning chronic type of pain that tends to be less well localized.

After entering the dorsal cord, these fibers might branch into short ascending and descending axonal processes and might travel for one or more segments in posterolateral portion of the lateral funiculus (Lissauer’s tract) before synapsing on interneurons in the posterior horn. While the neurons in lamina II, the substantia gelatinosa, are thought to play a significant role in the spinal regulation of pain perception, synaptic connections for the incoming pain fibers are established in lamina I through V of the spinal cord. While some of these incoming fibers make direct connections with the cells of origin for the anterolateral spinothalamic tracts, most probably make polysynaptic connections, including connections with interneurons in lamina VI through VIII, before ascending. Once these final synaptic connections have been made, the fibers then cross the midline in the ventral white commissure, which is located just anterior to the spinal canal in the central gray area of the cord. Once on the opposite side of the cord, these fibers ascend as part of the anterolateral spinothalamic tracts.

It again should be noted that because the initial peripheral nerves branched into both ascending and descending fibers as they entered the cord, any group of fibers might represent several cord segments entering the spinothalamic tracts. As a result of this arrangement, if a lesion occurs in the cord affecting the anterolateral tracts, the patient will demonstrate a loss or diminution of pain sensation on the opposite side of the body, beginning one or two segments below the level of the lesion. Conversely, irritation of a nerve root will result in pain (ipsilateral to the source of irritation) in the area served by the distribution of that nerve.

In addition to the spinothalamic tracts, the pathways just discussed also give rise to a large proportion of fibers that do not reach the thalamus, at least not directly. Rather, they synapse in the reticular system located in the brainstem, and hence, give rise to the spinoreticular tracts that travel in association with the anterolateral system. The possible function of the **spinoreticular tracts** will be discussed shortly.

As mentioned earlier, two general types of pain have been identified. The first is characterized by a sharp, acute, pricking, and generally well-localized type of pain. The second type has been described as pain that is slower in onset, more aching or burning in character, less well localized, and generally more persistent. It also was noted that the former seemed to be mediated primarily by the larger, myelinated A-delta fibers (fast pain), whereas smaller, unmyelinated C fibers were largely responsible for the latter or “slow pain.” However, it also appears that these two types of pain may be conveyed centrally or rostrally in somewhat separate and distinct pathways. “Fast pain” from the contralateral hemibody appears to be related to those spinothalamic fibers originating primarily in lamina I and V and projecting to the **ventral posterolateral (VPL) nucleus** of the thalamus. Comparable fibers from the contralateral face mediated by the trigeminothalamic pathway project to the **ventral posteromedial (VPM) nucleus**. These thalamic nuclei, to which the posterior columns (medial lemniscus) and related trigeminothalamic pathways also project, are topographically well organized and project in this same topographically organized fashion to the sensorimotor cortex (Brodmann's areas 3, 1, and 2). Because of the rather direct, point-to-point representation that is retained from the periphery to the cortex, this system is capable of finer discriminations of pain (or other tactile) information. Because such finer cortical discriminations represent a more recent phylogenetic development, these pain pathways have been termed the **neospinothalamic tracts**.

On the other hand, those fibers that carry information related to chronic, "slow," dull pain seem to originate more in lamina VI through VIII. These slow or dull pain pathways project primarily to the intralaminar nuclei of the thalamus, which in turn project to the cortex in a much more diffuse manner. These latter projections then do not have the same precise topographical organization as VPL or VPM and receive considerable input from the ascending reticular system. In fact, it is thought that these intralaminar nuclei probably represent the primary thalamic projection area for the spinoreticular fibers discussed above. Because they would appear to represent a more primitive system, those spinothalamic fibers that project to the intralaminar nuclei are known as the **paleospinothalamic** tracts. Fibers within this system (perhaps neospinothalamic fibers as well) also project to the posterior nuclei of the thalamus. Both the intralaminar and posterior nuclei of the thalamus project heavily to the secondary somatosensory cortex that lies in the area of the parietal operculum (an area where the inferior parietal cortex enfolds into the lateral fissure). In fact, though relatively rare, loss of affective response to painful stimuli or **pain asymbolia** has been associated most frequently with lesions in the left parietal opercular area. Thus, pain may be "perceived" in either the primary and/or secondary somatosensory cortices. There has been some suggestion that pain also may be "perceived" in some manner even in the thalamus, although it would seem unlikely that any true discriminative perception of pain could take place at this level.

In summary, the neospinothalamic tracts, which are phylogenetically more recently developed, project to those thalamic nuclei (VPL and VPM) that are topographically organized and which, in turn, project to the sensorimotor cortex in the same organized fashion. Because of this point-to-point representation, this system is better able to discriminate and localize painful stimuli. The paleospinothalamic system, along with the spinoreticular tracts, project in a more polysynaptic, diffuse manner to the intralaminar nuclei (and posterior nuclear complex). It is this latter system that seems to be responsible for the less well-defined, more diffusely localized chronic type of pain. It also is thought that this latter system may likely be the one more responsible for an individual's affective response to pain because of its connections with the limbic system.

Before leaving the discussion of pain pathways, several additional phenomena should be reviewed. The first is the notion of **referred pain**. In general, two types of referred pain might be identified, although only the first is classically defined as referred pain. An example of the former is where afferent feedback from some internal organs are perceived as painful in the distribution more or less corresponding to the segmental portion through which these fibers enter the cord. A classic example is pain radiating down the left arm in cases of myocardial infarction. The other situation in which pain might be considered as "referred" is found, for example, in dorsal root irritation. While the problem or mechanical irritant may be located in the lower lumbar region of the spinal vertebrae, the pain may be "perceived" as radiating down the leg.

A **thalamic pain** syndrome can result from a destructive lesion affecting the posterior thalamus. Such a lesion may initially produce a loss or decrease in sensation on the contralateral side of the body.

While these deficits may persist, after a time the patient may begin to experience significant and occasionally quite distressing pain on the side contralateral to the lesion. Such pain generally takes on more of the characteristics of the kind of pain mediated by the paleospinothalamic system (i.e., poorly localized, aching or burning type of pain). It often can be set off by minimal stimuli and will tend to persist and/or expand well beyond the time and space parameters of the eliciting stimulus.

Certain surgical lesions in the brain, including frontal lobotomies, frontal leukotomies, cingulotomies, thalamotomies (especially when the lesions involved some of the intralaminar or dorsomedial nuclei), and even surgical destruction of the pituitary gland, are among the sites found capable of dramatically altering one's subjective impression of, or response to, otherwise intractable pain. The problem with most of these lesions is that they also can produce other disabling behavioral problems. It is thought that perhaps the critical element in most, if not all, of these surgeries is the disconnection of critical limbic pathways (it has been hypothesized that the effectiveness of pituitary lesions is due to secondary lesions produced in the hypothalamus). In many of these "disconnection" cases, the patient may still report "feeling the pain," but being affectively "disconnected" from it. Other surgical treatments for pain include implanting devices to produce stimulation of large-diameter fibers, which in turn suppress or inhibit the firing of small-diameter C fibers (transcutaneous stimulation) or stimulation of the periaqueductal gray matter of the midbrain to stimulate the release of endorphins. Surgery also is used to disrupt the pain pathways by sectioning single nerves, the dorsal roots (**rhizotomy**), the lateral spinothalamic tract (**cordotomy**), or the posterior and intralaminar nuclei of the thalamus. Unfortunately, many of the surgical treatments may produce only temporary relief from pain.

More conservatively, treatment for pain may simply involve a variety of procedures including drugs that change the sensitivity of the peripheral nerve endings or affect the central processing of pain.

Temperature Sense

The information that allows the brain to perceive and distinguish the temperature of objects, liquids, or air as they come into contact with the skin also is carried by the anterolateral system. Similar to pain, it appears that A-delta and C fibers are responsible for conveying this information from the periphery to the spinal cord. No specific nerve endings have been identified as responding to temperature. It appears likely that, again similar to pain, these sensations are initiated through free nerve endings in the skin. The C fibers may mediate both extremes of hot or cold sensations that are perceived as "painful." The spinothalamic pathways for the perception of temperature are virtually indistinguishable from those mediating pain. Hence, loss of pain and temperature generally occur at the same time and in the same distribution.

Pain and temperature sensations in the face are mediated by the trigeminal (V) cranial nerve, specifically through the spinal trigeminal nucleus (see Chapter 5 for a more detailed description). Before entering this nucleus, however, the fibers coming into the pons travel caudally as the spinal tract of V prior to synapsing in the spinal trigeminal nucleus. The caudal portion of this nucleus is probably the relay center for most of the face, with the exception of dental pain, which is mediated through the interpolar nucleus. From these nuclei, crossed fibers ascend via the ventral trigeminal tract to terminate in the ventral posteromedial (VPM) nucleus of the thalamus. Pain sensations probably also reach the thalamus indirectly via the reticular system and are responsible for the duller, aching, chronic pain sensations of the face.

Simple Touch (Pressure)

A number of different types of cutaneous end organs have been identified that seem to play a role in the perception of touch. These are **Meissner's** and **Pacinian corpuscles; Merkel, peritichial,** and **Ruffini nerve endings**; as well as **free nerve endings**. It is not clear whether the sensory end organs that mediate the sensations of touch and pressure that travel in the anterolateral system (spinothalamic tracts) are any different in type or distribution than the ones that subserve the lemniscal (dorsal column) system, to be discussed next.

There are, however, at least two major distinctions between the two systems other than the tracts themselves:

1. In the anterolateral system, the fibers cross the midline at or near their level of entry into the cord, while those in the lemniscal system cross at the level of the medulla.
2. While both the anterolateral system and the lemniscal system are capable of mediating the sense of touch and pressure on the skin, the anterolateral system does not appear capable of fine tactile sensory discriminations, a capacity that characterizes the dorsal column or lemniscal system.

Actually, the sensory fibers that mediate touch (and probably vibration and proprioception as well) divide into three distinct ascending pathways or systems. Some ascend in the dorsal columns (lemniscal system), while others ascend ipsilaterally in the lateral cervical tract, synapsing in the lateral cervical nucleus in the upper cord. These fibers then cross the midline of the cord and ascend with the contralateral fibers of the anterolateral spinothalamic tracts. Finally, those fibers traveling in the anterolateral spinothalamic tracts follow pathways similar to those described for pain and temperature fibers. As is true of the latter nerve fibers, those conveying touch via this system typically ascend and descend several segments in the cord before crossing and ascending to the ventral posterolateral (VPL) nucleus of the thalamus. After synapsing there, these fibers, along with other somatosensory inputs, project via the posterior limb of the internal capsule to the postcentral gyrus to relay information concerning crude touch and pressure. It should be noted that while the major cortical projection site for the VPL and VPM nuclei is the primary somatosensory cortex (Brodmann's areas 3, 1, and 2), these nuclei probably project more broadly. Other cortical projections likely include the secondary somatosensory cortex in the parietal operculum and perhaps to the precentral sensorimotor cortices. As with the pathways for pain and temperature that travel in the lateral spinothalamic tract, collaterals for touch and pressure probably also go to the reticular formation and to the intralaminar nuclei of the thalamus. The intralaminar nuclei, in turn, project to a variety of structures, including other thalamic nuclei, the basal ganglia, and the limbic system and seem to play a role in general arousal or alertness.

As previously noted, because of the diversity of these pathways, including fibers that cross shortly after entering the cord (S-T tracts) and those that ascend to the medullary junction before crossing (lateral cervical tract and posterior columns), the more elementary sensations of touch are typically preserved following unilateral trauma to the cord. However, such a lesion likely would disrupt fine tactile discrimination ipsilaterally and pain and temperature perception contralaterally. The apparent presence of small-diameter afferent fibers in the ventral roots may offer one basis for the persistence of pain that can occur following sectioning of the dorsal roots. All tactile sensation below the level of the lesion, of course, would be lost following a complete transection of the cord.

Dermatomes

As noted in Chapter 1, the area of the skin represented by the pairs of dorsal roots at each spinal level (i.e., the vertebral spaces at which the nerves enter or exit) is referred

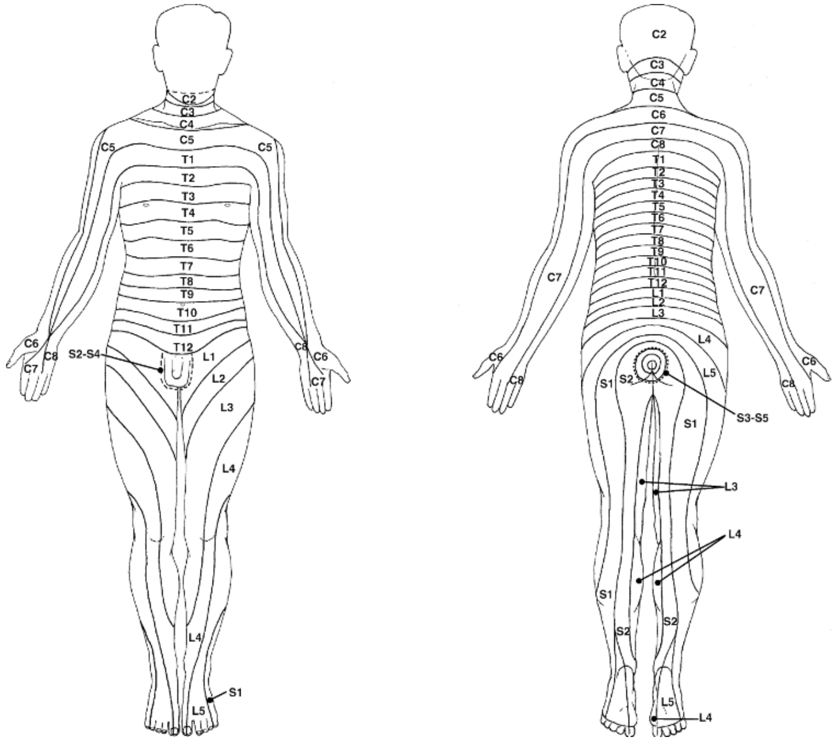


Figure 2–2. Approximate distribution of the segments of the skin represented by the various dorsal roots of the spinal cord. Considerable overlapping of adjacent segments is generally the case.

to as a **dermatome**. Figure 2–2 illustrates the distribution of the dermatomes for the spinal nerves. It should be kept in mind that the divisions shown in the illustrations are approximations and variations may be noted among various authors. Also, from a clinical standpoint, the divisions are not nearly as sharp or clear as might be implied by the diagram. There is considerable overlap between adjacent spinal segments. As a result, there may be little, if any, detectable sensory loss unless several adjacent dorsal roots are affected.

Lemniscal System

So far we have primarily focused on the anterolateral system (ventral and lateral S-T tracts), which primarily carries information regarding pain, temperature, and simple touch. In this section, the focus will be on the other major ascending system responsible for conscious perception of stimuli affecting the arms, legs and trunk, the **lemniscal** system.

The term "lemniscus" means a "ribbon" or a "band" and generically is a name that is applied to several pathways within the brainstem. The "lateral lemniscus" carries auditory fibers (see Chapter 5) and the "spinal lemniscus" results from the joining of the spinothalamic and spinotectal tracts in the medulla. The **medial lemniscus**, from which the lemniscal system derives its name, represents the ascending fibers of the posterior (dorsal) columns *after* they synapse in the nuclei cuneatus and gracilis above the spinal–medullary junction. The dorsal columns or lemniscal system represent the primary (though probably not exclusive) pathway for the perception of proprioception (position sense), kinesthesia (awareness of speed and direction of limb movement), stereognosis (fine tactual discrimination of contour, relief, texture, size, localization, etc.), and vibration sense.

Proprioception, Stereognosis, Vibratory Sense

The senses of proprioception and kinesthesia are initiated at the periphery by specialized nerve fibers. These include primary or annulospiral endings and secondary or flower-spray endings that attach to the intrafusal muscle fibers or muscles spindles, Golgi tendon organs (found where the tendon joins with the muscle), and both free and capsular (e.g., Golgi, Ruffini, and Pacinian) nerve endings in the ligaments and joints. Feedback for stereognosis is derived from the same types of specialized nerve ending in the skin (e.g., Pacinian corpuscles, Merkel's disks, Meissner's corpuscles, nerve plexus around hair follicles, and free nerve endings) that are found in the fibers which eventually ascend in the anterolateral system. Vibration probably should not be identified as a separate or special sense, but more likely represents a special condition (e.g., temporal summation) of touch. Pacinian corpuscles, because of their rapid adaptation, may play an important role in the perception of vibration.

Fibers from receptors mediating proprioception, kinesthesia, stereognosis, and vibratory sense enter directly into the dorsal columns and ascend ipsilaterally, without synapsing, to the lower medulla (Figure 2–3). The fibers from the leg ascend in the more medial portion of the dorsal columns in the fasciculus gracilis, while those fibers from the upper extremity ascend in the more lateral fasciculus cuneatus. Thus, the nerve fibers for the arm might be viewed as entering the cord after the fibers from the leg already have been laid down. These two tracts–the fasciculus gracilis and fasciculus cuneatus—terminate in the nucleus gracilis and nucleus cuneatus which are located in the lower medulla. After synapsing in these nuclei, the fibers then cross (decussate) and ascend as the medial lemniscus and terminate in the ventral posterolateral nucleus (VPL) of the thalamus. Again, comparable projections from the cranial nerves (e.g., the trigeminothalamic tract) terminate in the ventral posteromedial nucleus (VPM). From there, topographically organized projections are sent to the postcentral gyrus (BA 3, 1, 2). There probably is considerable overlap in the cortical projections of both the anterolateral and the lemniscal systems. Nevertheless, the lemniscal system's more direct and discrete topographically organized projections to the somatosensory cortex (as opposed to the more polysynaptic, diffused connections which are more characteristic of the anterolateral system) are what permit us to make fine tactile discriminations.

Facial Tactile Discrimination and Proprioception

Discrimination of tactile stimuli and proprioceptive feedback from the face is mediated by cranial nerve V. The "main sensory nucleus" of the trigeminal nucleus is the likely point of relay for this type of somatosensory information. Two tracts ascend from this nucleus. One decussates to join the medial lemniscus and terminates in the VPM nucleus of the thalamus; the other, an uncrossed tract, ascends as the dorsal trigeminal tract and terminates in a separate part of the VPM nucleus (Figure 2–3). The role of the latter is not clear, but it may relate to intraoral sensations. Proprioception for the muscles of mastication is somewhat unique. Their unipolar nerve cells are located in the mesencephalic nucleus, which is located in the upper pons and lower midbrain, rather than in a ganglion outside the CNS.

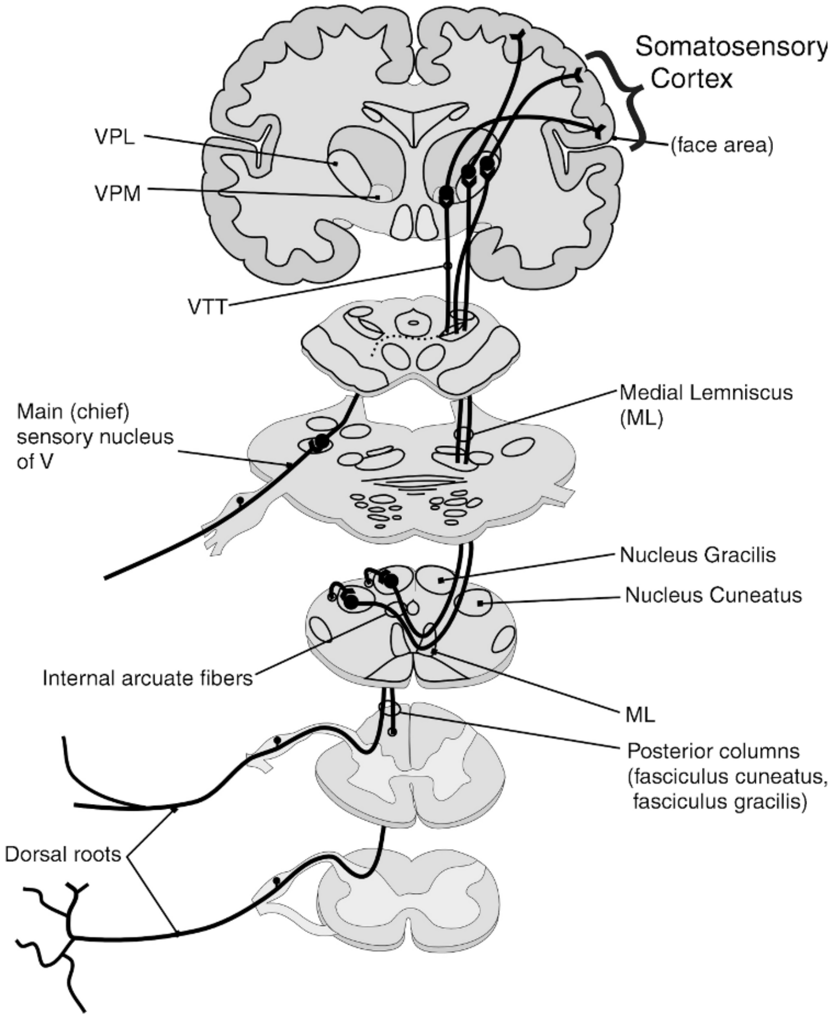


Figure 2–3. Fibers making up the lemniscal (dorsal column) system, critical for proprioception and fine tactual discrimination, enter ipsilateral dorsal funiculus without synapsing until they reach the nucleus cuneatus and nucleus gracilis in the lower medulla. Second-order neurons then cross the midline and ascend as the medical lemniscus to the VPL nuclei of the thalamus, there giving rise to third-order thalamocortical fibers.

Spinocerebellar Connections

As discussed in Chapter 1, there are many reflexes that are mediated at the spinal level. Perhaps among the most important are those that respond to noxious stimuli and those that respond to sudden changes in the antigravity muscles (necessary to maintain an upright balance).

These reflexes are executed, or at least can be executed, at the spinal level. In some instances, they may rely on a simple or monosynaptic connection between an intrafusal (proprioceptive) afferent fiber and an alpha motor neuron in the anterior horn feeding the same extrafusal muscle. At other times, although still remaining within the cord, the reflex involves several cord segments, several muscle groups, and multiple interneurons. However, for the more elaborate demands of most coordinated movements, higher-order connections must be established. Of central importance to this process are the cerebellum

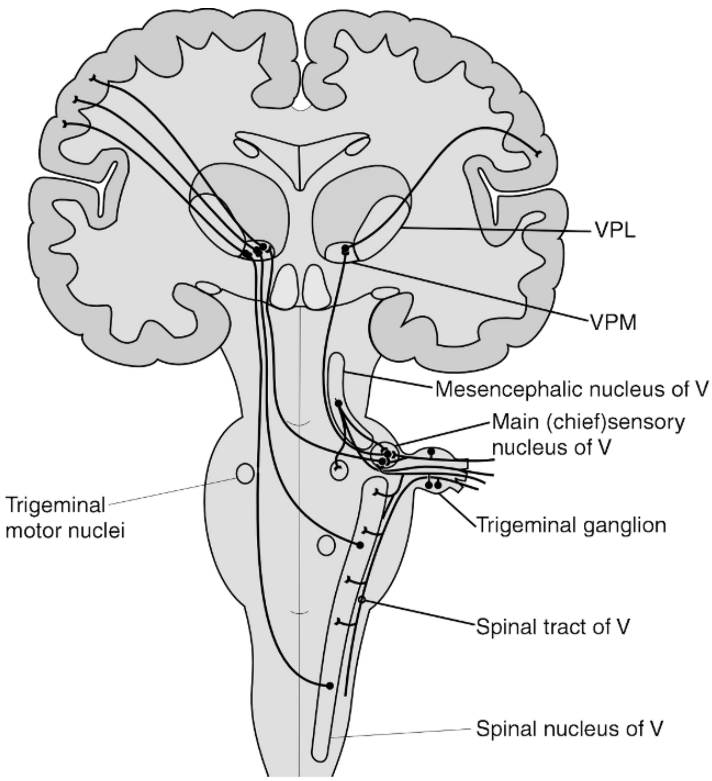


Figure 2–4. Comparable somatosensory systems mediating pain, temperature, proprioception, and fine tactual discrimination in the face are shown in this figure. Unlike the spinal pathways that synapse in the VPL, trigeminal pathways synapse in the ventral posterior medial (VPM) nuclei of the thalamus. See Chapter 5 for more detailed discussion of these pathways.

and its subsequent connections to the cortex, vestibular system, and other sensorimotor systems. Apparently there are no direct, descending connections between the cerebellum and the spinal cord. Obviously (or so it seems), what appears to be most critically needed by the cerebellum is information regarding the position of the body (both trunk and limbs) at any point in time, as well as the direction, degree, and speed of movement that is occurring. However, other types of sensory information also may be important or useful to coordinate higher-order reflex or voluntary movement. As a result of these needs, there are extensive connections between incoming somatosensory information and the cerebellum. The following is a review of these connections.

There are four major pathways by which information entering the spinal cord gets to the cerebellum. These will be discussed in greater detail in Chapter 3. For now it is important to remember that two of these pathways convey information from the lower extremities and two convey information from the upper extremities. Another point to remember is that virtually all information that goes to the cerebellum from the spinal cord remains ipsilateral, even when there is crossing of the midline by the spinocerebellar fibers (see below for the simple explanation). There are three pathways into the cerebellum: the **superior, middle,** and **inferior cerebellar peduncles**. The largest of these, the middle cerebellar peduncle, is the route by which the cortex gets information into the cerebellum. Of the pathways by which spinal information gets into the cerebellum, the majority enters via the inferior peduncle. Only one, the **ventral spinocerebellar tract**, clearly enters via the superior peduncle. The superior cerebellar peduncle, as we shall see, is also the major conduit for information leaving the cerebellum. Fibers mediating proprioception, touch, pressure, pain, and local reflex activity concerning the leg and lower torso synapse in the base of the posterior horn and then give rise to fibers that cross over to the opposite side of the cord. These then ascend to the cerebellum via the ventral spinocerebellar tract (crossed) in the lateral funiculus (Figure 2–5). However, before entering the cerebellum through the superior cerebellar peduncle (the only spinal tract that does), most of these fibers cross the midline again (i.e., double cross), so the input is still ipsilateral.

The **nucleus dorsalis of Clarke** located in the base of the dorsal horn (T-1 through L-2 or L-3) receives projections from the muscle spindles, Golgi tendon organs, and joint receptors. From there these fibers ascend, without crossing, to the cerebellum as the **dorsal spinocerebellar tract**, entering through the inferior cerebellar peduncle. This tract, which also contains information from the trunk and lower extremity, appears to be more specific in its input than the ventral spinocerebellar tract. It is primarily concerned with information regarding muscle tone, posture, and proprioceptive feedback. It enters the cerebellum through the inferior cerebellar peduncle.

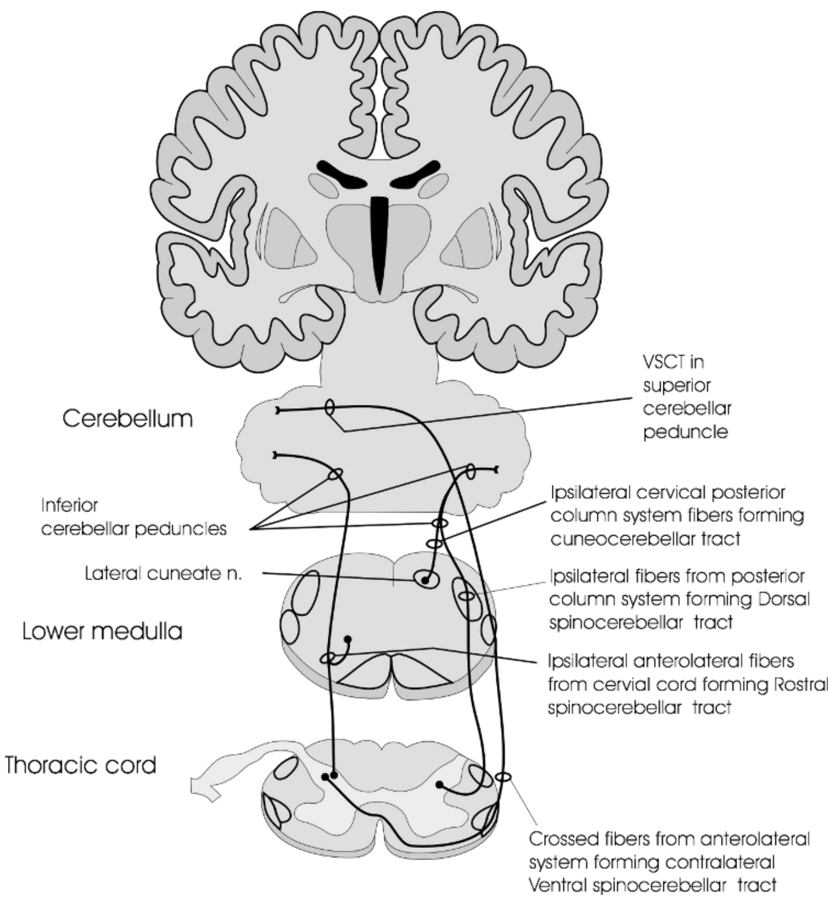


Figure 2–5. Spinocerebellar fibers shown here represent the third major group of ascending fibers in the spinal cord. Within the spinal cord itself, these consist of the dorsal and ventral spinocerebellar tract located in the lateral funiculus. Note: Spinal input to the cerebellum is primarily ipsilateral, despite the double crossing of the VSCT.

Comparable information regarding muscle tone and proprioceptive feedback from the upper extremities travels up the fasciculus cuneatus to synapse in the lateral or accessory cuneate nucleus (the upper extremity counterpart of the nucleus dorsalis). After synapsing in the accessory cuneate nucleus, the postsynaptic fibers become the **cuneocerebellar tract**. These fibers then ascend ipsilaterally and, like the dorsal spinocerebellar tract, enter the vermis of the cerebellum via the inferior cerebellar peduncle. The upper extremity counterpart of the ventral spinocerebellar tract is the **rostral spinocerebellar tract,** which originates in the nucleus centralis basalis of the cervical cord and proceeds ipsilaterally to the cerebellum.

TESTING FOR SOMATOSENSORY DEFICITS

Testing for Pain

Have the patient discriminate between the point ("sharp") and head ("dull") of a pin. You have to be careful to avoid simply tapping into the sense of touch. One should avoid using the same pin with different patients, as there is evidence that certain viruses can be transmitted in this fashion.

Testing for Proprioception

Have the patient attempt to localize his or her limb in space following passive movement by the examiner (with patients eyes closed) or indicate the state of flexion or extension of one's limb. However, perhaps the easiest and certainly one of the most sensitive and specific tests of proprioception is to passively extend or flex a digit (e.g., the great toe or a finger) while asking the patient to indicate the direction in which it is being moved. One also may check the integrity of the posterior columns by asking the patient to stand erect with the feet together. If the patient shows considerably more difficulty maintaining balance with the eyes closed than opened, this suggests posterior column compromise (Romberg's sign). If comparable difficulties are noted regardless of whether the eyes are open or closed, cerebellar disease should be suspected.

Testing for Stereognosis

Here we might ask the patient to differentiate shapes, textures, or similarly configured small objects (e.g., a paper clip versus a safety pin) by touch. The patient also may be asked to identify numbers written on the fingertip or palm of the hand (graphesthesia), to make two-point discriminations, or to localize stimuli applied to various parts of the face, limbs, or torso.

Testing for Vibration

This procedure basically calls for the application of a tuning fork (256 cps) to bony prominences of the distal upper and lower extremities. The examiner must perform trials with and without the tuning fork vibrating to assure reliability and comprehension of the test. The patient is instructed to indicate whether the tuning fork is vibrating when touching the limbs, and if it is when the vibration appears to stop. A vibratory sensory level can be determined by starting distally and working one's way proximately up the limb. The latter procedure would be important if a neurologist expects a peripheral neuropathy.

Testing for Temperature

The patient is asked to discriminate between objects of different temperatures. The examiner should ensure that the temperatures are readily discriminable, but neither is extreme. Test tubes filled with warm and cool water make reasonable testing devices.

In all cases, the examiner always should compare performances on the right versus the left side of the body. One should be alert to the possibility of cortical neglect as well as old central or peripheral injuries or disease processes that might have an effect on peripheral processes, such as peripheral neuropathy secondary to diabetes or chronic alcohol abuse.

LESIONS AND DISEASES AFFECTING THE ASCENDING AND DESCENDING TRACTS

Lesions Affecting the Posterior Limb of the Internal Capsule

Lesions restricted to the anterior portion of the posterior limb of the internal capsule normally result in a **contralateral hemiparesis** or hemiplegia (see Figure 2–6a). The distribution of weakness is distinct and characteristic of lesions of the corticospinal pathway. In the upper extremity, weakness is most pronounced in the distal extensor muscles, while in the lower extremity it is the proximal flexor muscles that are most affected. Increased deep-tendon reflexes are present on the affected side, along with pathological reflexes, such a **Babinski** or **Hoffmann's sign**. Although hypotonia may be present initially, this eventually is followed by an increase in muscle tone along with other indications of **hyperreflexia** on that side,

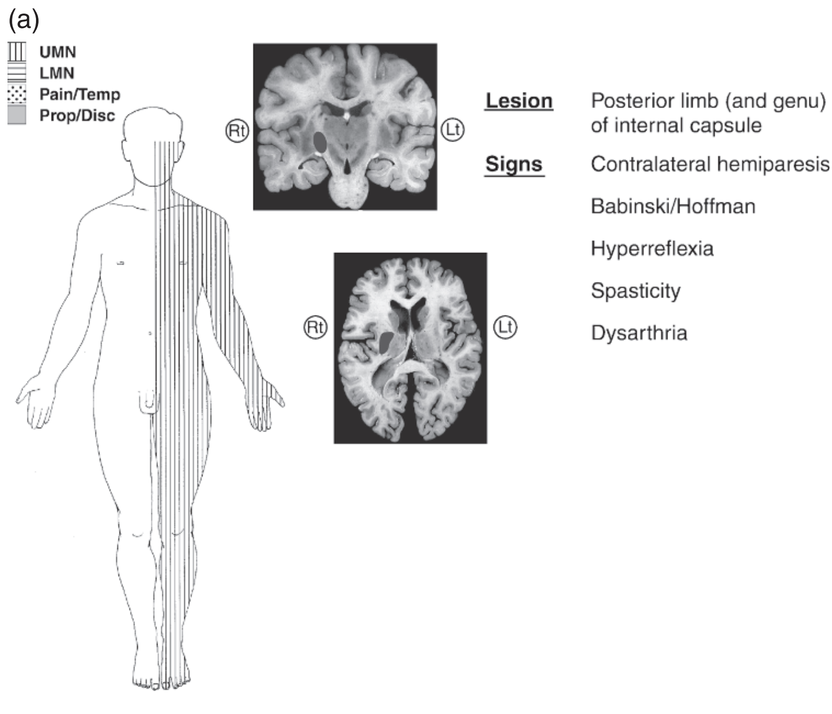
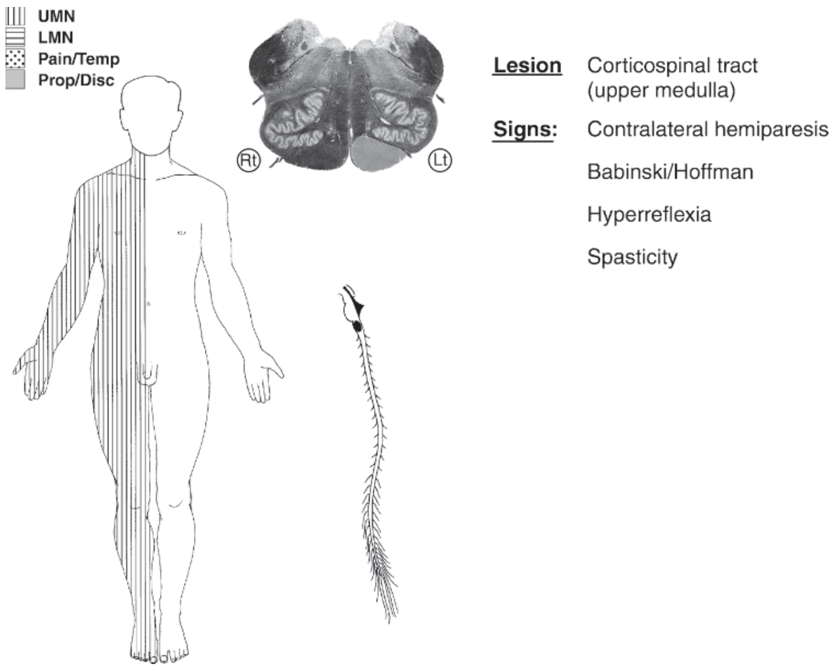
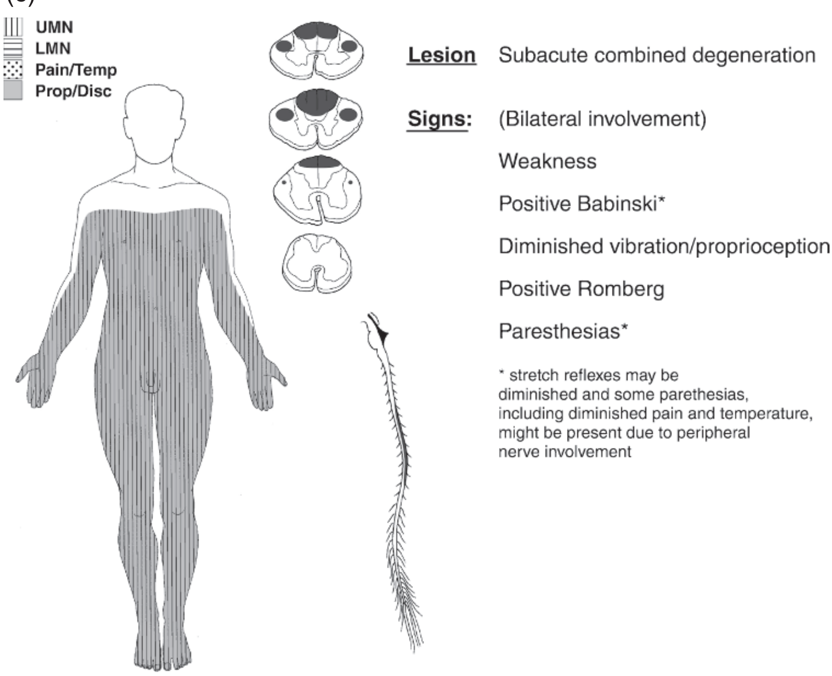


Figure 2–6. Lesions affecting sensory–motor systems. Legend: UMN, upper motor neuron deficits; LMN, lower motor neuron deficits; Pain/Temp, changes in pain and temperature; Prop/Disc, changes in proprioception, fine tactile discrimination (and vibration).

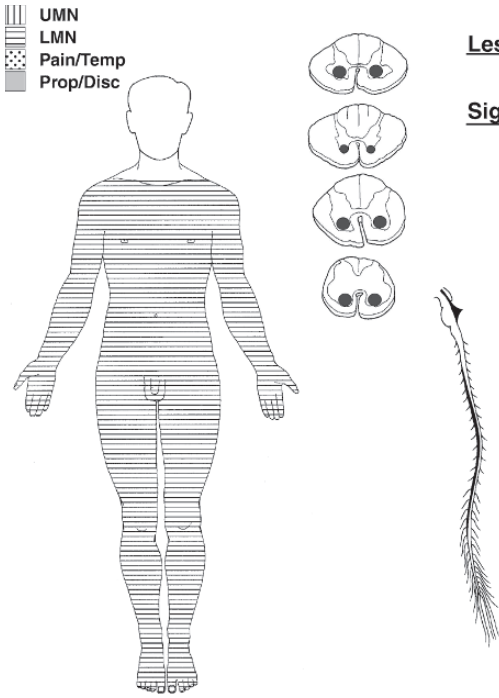
(b)


UMN
LMN
Pain/Temp
Prop/Disc
Rt
Lt
Lesion Corticospinal tract (upper medulla)
Signs: Contralateral hemiparesis
Babinski/Hoffman
Hyperreflexia
Spasticity

(c)


UMN
LMN
Pain/Temp
Prop/Disc
Lesion Subacute combined degeneration
Signs: (Bilateral involvement)
Weakness
Positive Babinski*
Diminished vibration/proprioception
Positive Romberg
Paresthesias*
* stretch reflexes may be diminished and some parethesias, including diminished pain and temperature, might be present due to peripheral nerve involvement

(d)



Lesion Anterior horn cells

Signs: (Bilateral involvement)

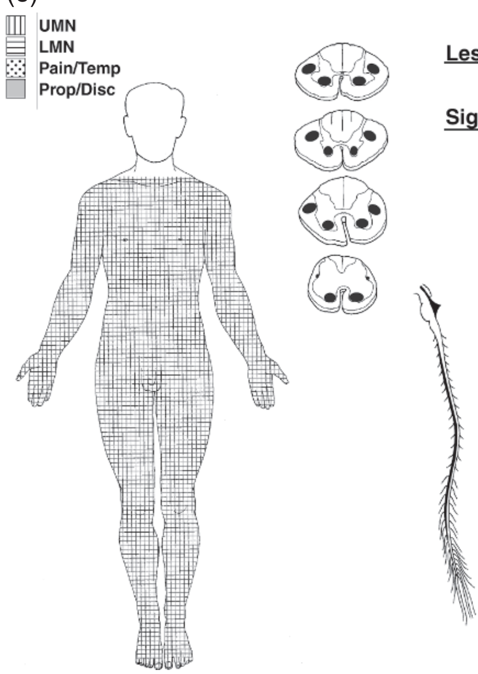
Flaccid paralysis

Absent reflexes

Fasciculations

Severe muscle atrophy

(e)



Lesion Amyotrophic lateral sclerosis

Signs: Weakness* (bilateral)

Muscle atrophy

Gait disturbance

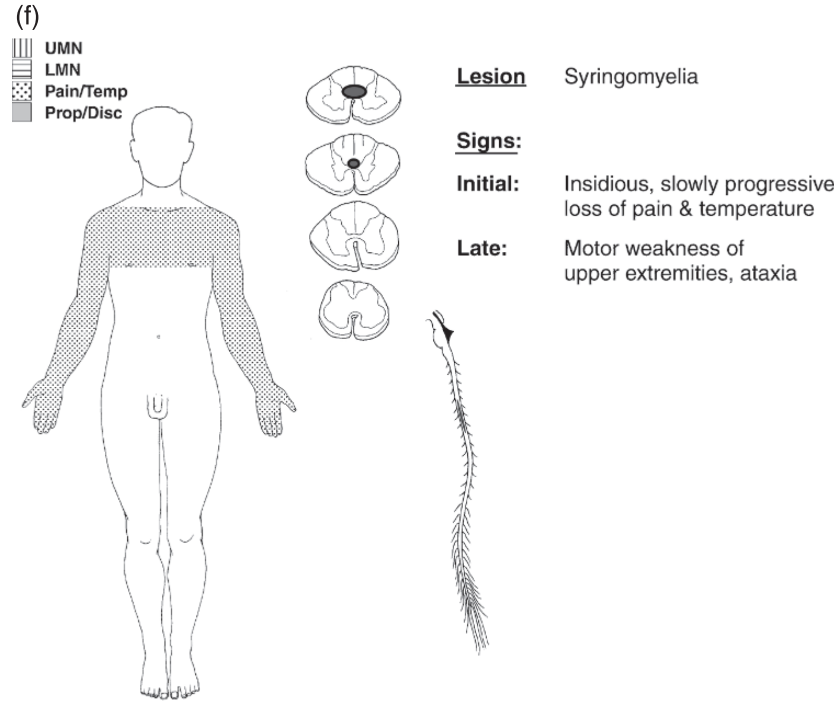
Hyperreflexia** (including Babinski, clonus)

Fasciculations

Cramping

* may start distally and unilaterally, or include dysphagia, dysarthria, respiration difficulties

** some DTR's may be diminished



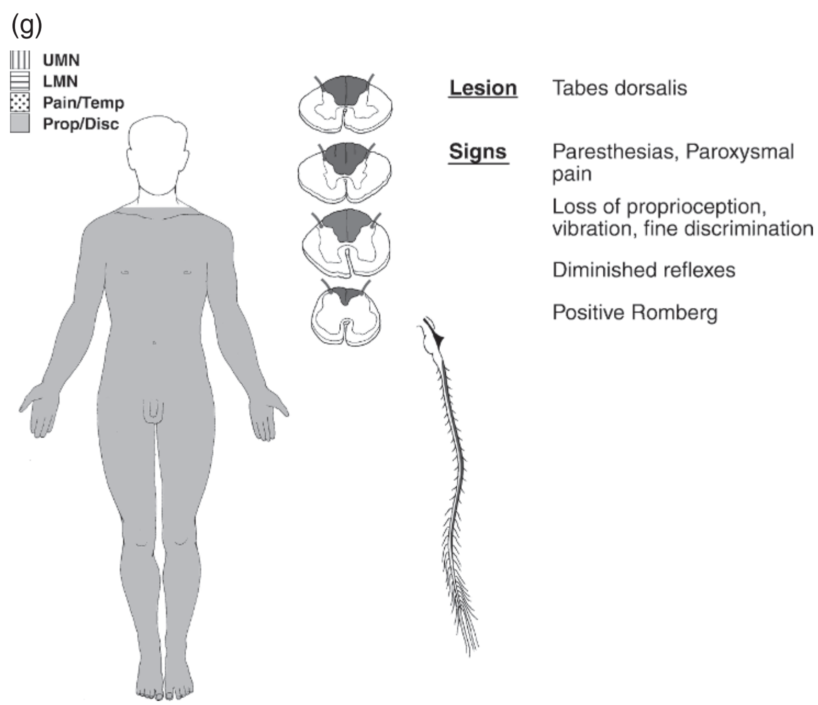
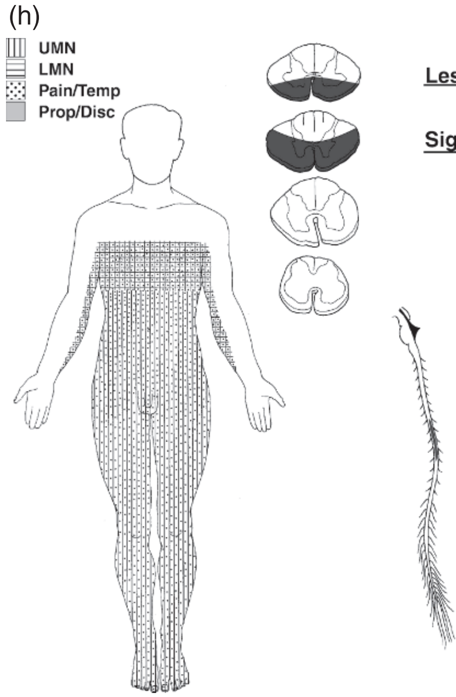
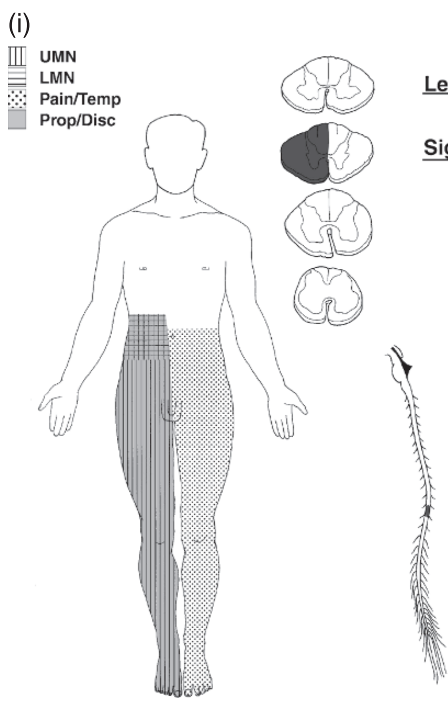


Figure 2–6. *(Continued)*



Lesion Anterior Spinal Artery Syndrome

Signs:
- Weakness/paralysis below level of lesion
- Atrophy, fasciculations (LMN) at level of infarction
- Loss of pain/temperature below level of lesion (proprioception/vibration intact)
- Acute/subacute onset
- Pain may be present



Lesion Brown-Séquard

Signs:
- Ipsilateral UMN signs below level of lesion
- Ipsilateral LMN signs at level of lesion
- Ipsilateral loss of proprioception, vibration, fine discrimination below level of lesion
- Contralateral loss of pain/temperature beginning a few segments below lesion

Figure 2–6. *(Continued)*

which might include **clonus** and clasp-knife **spasticity**. If the lesion extends toward the genu of the internal capsule, an upper motor neuron VIIth cranial nerve deficit may be present involving the contralateral face. In upper motor neuron lesions of the VIIth nerve, only the muscles of expression in the lower face are affected (see Chapter 5).

The most common cause of such lesions are lacunar infarcts secondary to hypertension (see Chapter 10) which can be relatively small in size, and hence restricted in their sphere of influence. However, since the motor pathways converge in the internal capsule, even a small lesion can have profound and broad-reaching effects topographically. As can be seen in Figure 7–5, the thalamocortical projections from the ventral posterior nuclei of the thalamus that carry somatosensory information to the cortex are in close proximity to the descending motor pathways (generally slightly posterior to the latter, with the visual projections being even more posterior in the internal capsule). Thus, if the lacunar lesion extends slightly more posteriorly, somatosensory symptoms also may be seen. Simple touch or pain usually is not affected by such lesions, but rather the more higher-order perceptual judgments normally carried out by the postcentral cortices (3, 1, 2) such as stereognosis, two-point, weight, or texture discriminations. If there is an even more extensive disruption involving the posterior limb of the internal capsule (e.g., as a result of an intracranial hemorrhage), one might see unilateral motor, somatosensory, and visual disturbances from a single lesion, although this would be a relatively rare phenomenon.

Lesions Affecting the Corticospinal Tract

Lesions that are isolated to the C-S tract and do not involve other descending tracts are relatively rare, but may occur, for example, with focal vascular disease (strokes) in the brainstem. Lesions of the corticospinal tracts in the brainstem produce the same type of deficit described above, except the face will not be involved if the insult is below level of the nucleus of the VIIth cranial nerve (Figure 2–6b). If the brainstem lesion is above the level of the decussation, cranial nerve signs may accompany it on the side opposite the hemiplegia. However, if the damage is restricted solely to the C-S tract at the medullary level, the hemiplegia and associated reflex changes are often mild and may be limited to a pronator drift and decreased fine motor skills. The presence of other descending tracts at this point (e.g., reticulospinal, vestibulospinal, and rubrospinal) likely provides sufficient muscular innervation to prevent a more dramatic loss of function.

Subacute Combined Degeneration (Posterolateral, Dorsal Column Syndrome)

Occurring in association with **pernicious anemia** (vitamin B-12 deficiency), this syndrome tends to selectively affect the posterior columns and the lateral corticospinal tracts in the cord (Figure 2–6c). Because of the involvement of the corticospinal tracts, **upper motor neuron** signs are present (weakness, spasticity, and positive Babinski). Reflexes, however, may be diminished due to peripheral nerve involvement. Because the posterior columns are affected, there will be impairments of **proprioception, stereognosis,** and **vibration sense** and a **positive Romberg** (loss of balance with the feet together and eyes closed). As a result of the peripheral neuropathy, **paresthesias** as well as some **loss of pain and temperature** may be present. The disease typically presents with a slow, insidious onset with bilateral signs or symptoms and the legs are often affected before the upper extremities. In addition, if due to a B-12 deficiency, this syndrome may be accompanied by memory loss. Early identification and treatment are essential to prevent further progression of symptoms.

Anterior Horn Cell Disease (e.g., Poliomyelitis)

Since the disease process is typically isolated to the region of the **anterior horn cells** (lower motor neurons), the presenting clinical picture is one of flaccid paralysis, absent reflexes, fasciculations, and muscle atrophy (Figure 2–6d). As the posterior roots are unaffected, sensations remain essentially intact.

Combined Anterior Horn and Corticospinal Tract Disease (ALS)

In amyotrophic lateral sclerosis (ALS), there is a combined degeneration of the pyramidal tracts and the motor cells in the cranial nerve nuclei and in the ventral horns of the cord resulting in a **combination of upper (UMN) and lower motor neuron (LMN) symptoms** (Figure 2–6e). The LMN involvement produces weakness and atrophy in some muscle groups and the UMN lesion result in spasticity and hyperreflexia in others. The cranial nerves (motor) often are affected. Fasciculations are commonly present. As this disease process is limited to the motor system, no sensory loss is experienced.

Lesion of the Central Gray Matter (Syringomyelia)

In syringomyelia, there is an expanding cavity that forms in the central gray area of the cord, usually in the area of the cervical or lumbar enlargement. The lesion primarily affects the fibers that make up the lateral spinothalamic tract as they cross the midline of the cord. Because they affect the crossing fibers from both sides, there is a bilateral loss of pain and temperature sensation at the level of the lesion, that is, typically in both upper extremities, often described as a cape-like sensory loss (Figure 2–6f). Since the fibers that enter the cord below this defect and the lateral spinal tracts made up of those fibers are unaffected, pain and temperature in the lower extremities are unaffected. Also, since the lemniscal tracts do not decussate until the midbrain, proprioception, touch, and vibratory sense are intact in both the upper and lower extremities. Signs of upper motor neuron disease, however, may be evident in the lower extremities as a result of pressure being exerted on the corticospinal tracts by the expanding cavity. Also, as the lesion expands, it also may affect the anterior horn cells, leading to motor changes in the upper extremities. These lesions are most often congenital or associated with trauma.

Posterior Column Disease

Tabes dorsalis is a lesion associated with tertiary neurosyphilis and is anatomically limited to the posterior columns and dorsal roots of the spinal cord. Therefore, early on there may be paresthesias and sharp pains, particularly in the lower extremities (Figure 2–6g). As the disease progresses, there is a loss of sensation and loss of reflexes as a result of an interruption of the reflex arc. There is also a loss of position and vibratory sense and a positive Romberg due to the loss of proprioceptive feedback due to involvement of the posterior columns. While not directly involving the lower motor neurons, the lack of sensory feedback may produce hypotonia with little or no loss of voluntary movement or strength and no major atrophic changes or fasciculations. Gait will be affected as the patient has to visually monitor what he is doing. The gait is wide-based and clinically resembles the ataxic gait seen in midline vermal cerebellar lesions. Because the mechanism is different, the gait due to posterior column disease is called a sensory ataxia.

Thrombosis of the Anterior Spinal Artery

The anterior spinal artery supplies blood to the anterior and much of the middle sections of the cord. Atrophy, flaccid paralysis, and fasciculations (lower motor neuron syndrome) are present at the level of the lesion due to the damage to the anterior horn cells (Figure 2–6h). Spastic paralysis below the lesion (paraplegia) can result from destruction of the corticospinal tract. Loss of pain and temperature bilaterally below the lesion results from damage to the spinothalamic tracts. Because of anastomoses with radicular arteries along the course of the spinal cord, the area of infarction is often limited.

Hemisection of the Cord

A lesion that more or less locally affects one lateral half of the cord produces what is commonly referred to as a **Brown-Séquard syndrome** (Figure 2–6i). This condition usually results from a traumatic lesion, such as stab or bullet wound. Upper motor neuron signs (e.g., spastic paralysis, hyperreflexia, clonus, loss of superficial reflexes, and a positive Babinski) will be present on the side of the lesion due to disruption of the corticospinal tracts. Ipsilateral loss of position and vibratory sense and tactile discrimination (astereognosis) below the level of the lesion are observed as a result of sectioning of the dorsal column. Loss of pain and temperature on the opposite side of the lesion beginning about one or two dermatomes below the lesion are present, resulting from damage to the anterolateral spinothalamic tracts.

Peripheral Lesions

The dorsal and ventral roots, along with the combined portions of the peripheral nerves, are subject to a vast array of pathology, including trauma or mechanical injury; metabolic deficiencies; toxins, autoimmune, and infectious processes; and genetic and other disorders. Depending on the particular pathology, the symptoms may be primarily motor or sensory or a combination of the two. In addition, the effects may be unilateral and limited to or restricted to a single limb or a few dermatomes, or may be bilateral or relatively diffuse. Further confusing the picture is the fact that some disease processes directly affect the muscles rather than the nerves while producing similar symptoms. On the other hand, one disease process, **myasthenia gravis**, affects not so much the muscle or the nerve, but rather the synaptic junction between the two.[1] In addition to the history and clinical examination, nerve conduction studies, electromyography, and serum analysis are often quite helpful in making these differential diagnoses.

The array of peripheral neuropathies is quite extensive and the interested reader is referred to clinical neurology texts for a more complete listing and description of these disorders (e.g., Adams, Victor, & Ropper, 1997). There are, however, certain clinical correlates that tend to be more characteristic of peripheral nerve lesions. Because except for the nerve roots, the peripheral nerves carry both motor and sensory information, muscle weakness frequently is accompanied by sensory changes (e.g., diminished proprioception, vibration, stereognosis, and/or the presence of paresthesias, pain, or a burning sensation).[2] Frequently, although not invariably, peripheral neuropathies may tend to manifest themselves distally more than proximally and may result in a "glove and stocking" type distribution affecting several or all extremities. Because they represent lower motor neuron lesions, diminished reflexes are often prominent (see Chapter 1 for review of lower-motor neuron lesions). Muscle atrophy, a common correlate of peripheral nerve damage, may not be seen in the more acute stages of the disorder. Among the more common causes of peripheral polyneuropathy in the general population are those resulting from chronic alcohol abuse and diabetes, whereas trauma and disk herniation are among the more frequent sources of more focal neuropathies.

One particularly dramatic and potentially fatal form of peripheral neuropathy is "acute post-infectious polyneuropathy," also known as Guillain-Barré syndrome. Typically developing a week or more after a viral infection (or vaccination), this syndrome primarily attacks the peripheral nerves. Both motor (progressive weakness or paralysis, decreased tone, and markedly diminished or absent deep tendon reflexes) and sensory (paresthesias, pain, and diminished proprioception, stereognosis, and vibration) symptoms are present, although the motor are generally more severe. The symptoms are usually symmetrical and frequently start in the legs, progressing to the upper extremities and cranial nerves, often sparing the eye muscles. As the respiratory muscles are commonly involved, this may prove to be a rapidly fatal condition. However, if properly ventilated during the acute stage complete recovery is common.

Long-Tract Findings in Supraspinal Lesions

The various descending motor pathways in the spinal cord have their origins in the cerebral cortex, as well as in the brainstem. Similarly, the ascending fibers mediating conscious somatosensory perception continue either uninterrupted (spinal thalamic tracts) or following synaptic connections in the medulla (posterior columns) as discreet fiber tracts throughout the length of the brainstem before synapsing in the thalamus. As a result, brainstem lesions can result in symptoms not unlike those accompanying cord lesions. While such lesions will be reviewed in greater detail in Chapter 4, for the present it should be noted that spinal cord and brainstem lesions often might be differentiated clinically. Part of this is due to where the various fiber tracts cross the midline. For example, focal lesions of the cord are more likely to be associated with ipsilateral disturbances of proprioception and stereognosis and upper motor neuron signs below the level of the lesion. By contrast, in the brainstem, focal lesions affecting the pyramidal tract also may result in changes in pain and temperature (as well as proprioception and stereognosis) on the same side as the motor deficits. Even more critically, the brainstem is the site of most of the cranial nerves and their respective nuclei. Hence, brainstem lesions that affect the major ascending or descending pathways often also result in cranial nerve findings involving the head or face, which, of course, are not expected in lesions of the cord.[3]

Endnotes

1. Unlike peripheral neuropathy, which frequently first manifests itself in the extremities, myasthenia gravis tends to initially affect the muscles of the face and head, particularly the eyelids (levator palpebrae) and extraocular muscles, and tends to improve following rest.
2. Multiple sclerosis may similarly present with focal muscle weakness and sensory changes. However, in multiple sclerosis the locus of the motor and sensory changes may be dissociable and the symptoms tend to vary more over time and (body) space than with peripheral neuropathies.
3. One notable exception is **Horner's syndrome** (constricted pupil, partial ptosis, and ipsilateral anhidrosis) which, as noted in Chapter 1, can result from lesions of the lower cervical or upper thoracic cord, disrupting sympathetic innervation of the eye and face. As will be discussed in Chapters 4 and 5, further complicating the situation is the fact that this latter syndrome also may accompany lesions of the brainstem that interrupt descending sympathetic fibers from the hypothalamus.

REFERENCES AND SUGGESTED READINGS

Adams, R.D., Victor, M., & Ropper, A.H. (1997) Principles of Neurology (Part 5. Diseases of_Spinal Cord, Peripheral Nerve, and Muscle). New York: McGraw-Hill.

Barr, M.L. & Kiernan, J.A. (1993) The Human Nervous System. Philadelphia: J.B. Lippincott Co., pp. 293–312.

Brazis, P., Masdeu, J. & Biller, J. (1990) Localization in Clinical Neurology. Boston: Little, Brown & Co., pp. 70–92.

Carpenter, M.B. & Sutin, J. (1983) Human Neuroanatomy. Baltimore: Williams & Wilkins, pp. 265–314.
Davidoff, R.A. (1989) The dorsal columns. *Neurology, 39*, 1377–1385.
DeMeyer, W. (1980) Technique of the Neurologic Examination: A programmed text. New York: McGraw-Hill, pp. 298–328.
Gilman S. & Newman, S.W. (1992) Essentials of Clinical Neuroanatomy and Neurophysiology. Philadelphia: F.A. Davis Co., pp. 55–74; 84–95.
Haines, D.E. (Ed.) Fundamental neuroscience. New York: Churchill Livingstone, pp. 219–263 (Somatosensory System I and II; Viscerosensory Pathways).
Martin, J.H. (1996) Neuroanatomy: Text and Atlas. Stamford, CT: Appleton and Lange, pp. 125–159.
Melzack, R. & Wall, P.D. (1965) Pain mechanisms: A new theory. *Science, 150*, 971–979.
Nathan, P.W., Smith, M.C., & Cook, A.W. (1986) Sensory effects in man of lesions of the posterior colums and of some other afferent pathways. *Brain, 109*, 1003–1041.
Wiebers, D.O., Dale, A.J.D., Kokmen, E., & Swanson, J.W. (Eds.) (1998) *Mayo Clinic Examinations in Neurology*. St. Louis: Mosby, pp. 255–268.
Willis, W.D. & Coggeshall, R.E. (1991) *Sensory Mechanisms of the Spinal Cord*. 2nd edition. New York: Plenum Press.