

SOMATOSENSORY PATHWAYS

The somatosensory systems' anatomy in this module will review the major somatosensory pathways including the posterior columns-medial lemniscal pathway, spinothalamic tract and other anterolateral pathways, and somatosensory cortex. In addition, brainstem and spinal cord mechanisms of pain modulation will be addressed. Finally, the organization of the thalamus, serving as the major relay for sensory and other information traveling to the cortex will be reviewed.

Main Somatosensory Pathways. The term **somatosensory** refers to bodily sensations of touch, pain, temperature, vibration, and proprioception (limb or joint position sense). The **posterior column-medial lemniscal pathway** conveys proprioception, vibration sense, and fine, discriminative touch. The **anterolateral** (or **ventrolateral**) **pathways**, include the **spinothalamic tract** and other associated tracts, convey pain, temperature sense, and crude touch. Since some aspects of touch sensation are carried by both pathways, touch sensation is not eliminated completely in isolated lesions to either pathway.

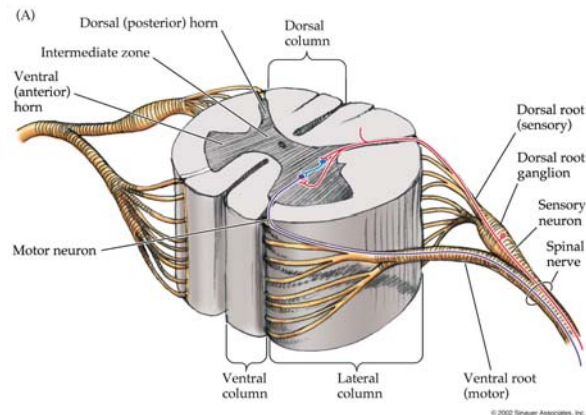
There are 4 types of sensory neuron fibers which are classified according to axon diameter. These different fiber types have specialized peripheral receptors that subserve different sensory modalities. Larger-diameter, myelinated axons conduct faster than smaller-diameter or unmyelinated axons. From largest to smallest diameter and conduction velocity, there are called A-alpha, A-beta, A-gamma, and unmyelinated C's. They are detailed below along with their diameter, receptor type, and the sensory modality the serve.

TABLE 7.2 Sensory Neuron Fiber Types					
NAME	ALTERNATE NAME	FIBER DIAMETER (μm)	MYELINATED	RECEPTORS	SENSORY MODALITIES
A-α	I	13–20	Yes	Muscle spindle	Proprioception
				Golgi tendon organ	Proprioception
A-β	II	6–12	Yes	Muscle spindle	Proprioception
				Meissner's corpuscle	Superficial touch
				Merkel's receptor	Superficial touch
				Pacinian corpuscle	Deep touch, vibration
				Ruffini ending	Deep touch, vibration
A-δ	III	1–5	Yes	Hair receptor	Touch, vibration
				Bare nerve ending	Pain
C	IV	0.2–1.5	No	Bare nerve ending	Temperature (cool)
				Bare nerve ending	Pain
				Bare nerve ending	Temperature (warm)
				Bare nerve ending	Itch

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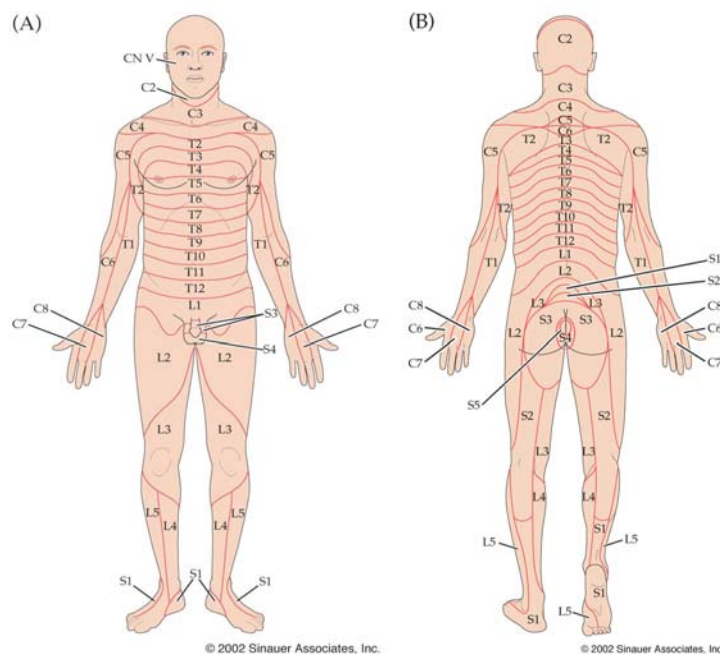
Sensory neuron cell bodies are located in the **dorsal root ganglia** (see figure below). Each dorsal root ganglion cell has a stem axon that bifurcates, resulting in one long axon that conveys sensory information from the periphery,

and a second axon that carries information to the spinal cord through the dorsal nerve roots.



A peripheral region innervated by sensory fibers from a single nerve root level is called a **dermatome**. Dermatomes for the different spinal levels form a map over the surface of the body. Sensation for the face is provided by the trigeminal nerve (CN V), while most of the remainder of the head is supplied by C2. The nipples are usually at the T4 level, while the umbilicus (“belly button”) is at approximately T10.

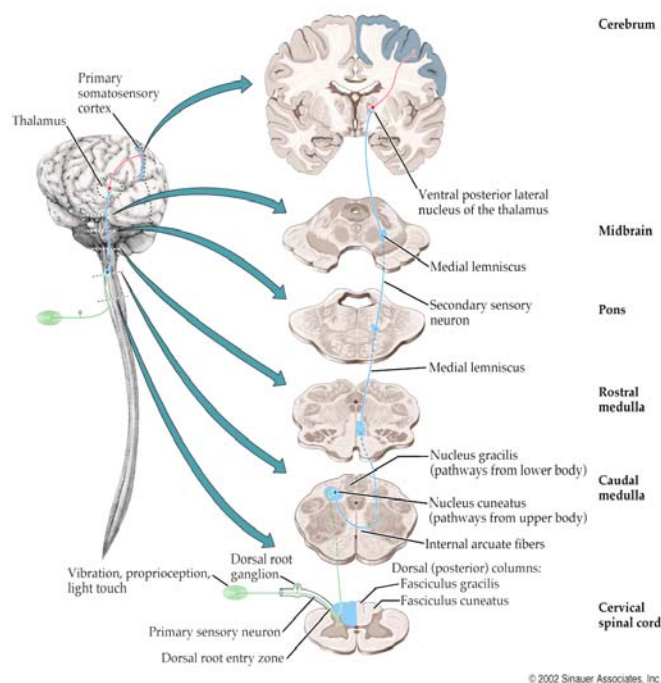
The shoulder, arms, and hands are represented in C5 through T1. The L4 and L5 representation extends over the anteriomedial shin and foot to the big toe. S1 and S2 innervate the back (dorsum) of the legs, and S3, S4, and S5 innervate the perineal area in a saddle-like distribution. The distribution of dermatomes is given in the figure below.



Just as our knowledge that the corticospinal tract crosses over at the pyramidal decussation helps us localize lesions, it is equally important to know

the points of decussation of the two major somatosensory pathways. The course of the two main somatosensory pathways will, therefore, be reviewed below.

Posterior Column-Medial Lemniscal Pathway. Large-diameter, myelinated axons carrying information about proprioception, vibration, and fine touch enter the spinal cord via the dorsal root entry zone. Most of these axons then enter the **ipsilateral posterior columns** to ascend all the way to the posterior column nuclei in the medulla (see figure below). The most medial posterior column is called the **fasciculus gracilis** which carries sensory information from the legs and lower trunk. The more lateral **fasciculus cuneatus** carries information from the trunk above about T6, and from the arms and neck.



The first order neurons travel from the sensory receptor in the periphery, into the spinal cord, and then travel all the way up the cord in the posterior columns (fasciculus gracilis and cuneatus) to synapse onto second-order neurons in the **nucleus gracilis** and **nucleus cuneatus** located in the medulla (see figure above).

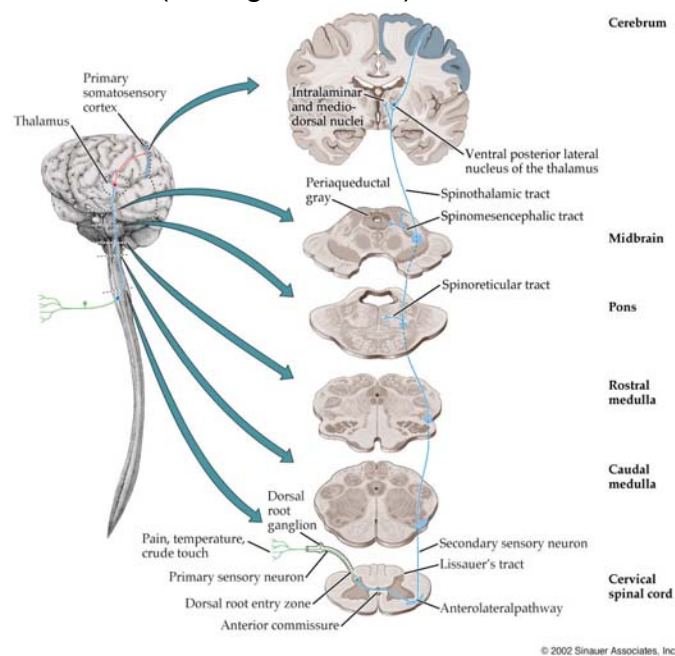
Axons of these second-order neurons decussate as the **internal arcuate fibers** and then form the **medial lemniscus** on the other side of the medulla. The next major synapse occurs when the medial lemniscus axons terminate in the **ventral posterior lateral nucleus (VPL)** of the thalamus. The neurons of VPL then project through the posterior limb of the internal capsule in the **thalamic somatosensory radiations** to reach the **primary somatosensory cortex** in the postcentral gyrus (see figure above).

An analogous pathway called the **trigeminal lemniscus** conveys touch and vibration sense for the face via the **ventral posterior medial (VPM) nucleus** of the thalamus to the primary somatosensory cortex. Synaptic inputs to the

primary somatosensory cortex from both the face and body occur mainly in **cortical layer IV**.

Spinothalamic Tract & Other Anterolateral Pathways. Smaller-diameter and unmyelinated axons carrying information about pain and temperature sense also enter the spinal cord via the dorsal root entry zone. However, in contrast to the posterior columns, these axons make their first synapses immediately in the gray matter of the spinal cord.

Second-order anterolateral sensory neurons in the central gray cross over in the spinal cord **anterior (ventral) commissure** to ascend in the anterolateral (ventrolateral) white matter (see figure below).



The next major synaptic relay is, again, in the thalamus which projects to the primary somatosensory cortex (Brodmann's areas 3, 1, 2) via the posterior limb of the internal capsule (see figure above). Pain and temperature sensation for the face is carried by an analogous pathway called the **trigeminothalamic tract**.

The anterolateral pathways consist of three tracts: the spinothalamic, spinoreticular, and spinomesencephalic tracts. The **spinothalamic tract** is the best known and mediates discriminative aspects of pain and temperature sensation, such as location and intensity of the stimulus. Like the posterior column-medial lemniscal pathway, the main relay for the spinothalamic tract is the **ventral posterior lateral nucleus (VPL)** of the thalamus. There are also some spinothalamic projections to other thalamic nuclei, including **intralaminar thalamic nuclei** and **dorsal-medial nucleus**.

The **spinoreticular tract** within the anterolateral pathways is a phylogenetically older pain pathway responsible for conveying the emotional and

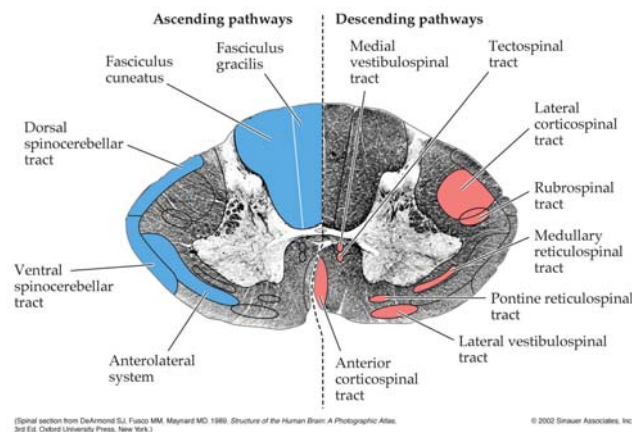
arousal aspects of pain. The spinoreticular tract terminates on the medullary-pontine reticular formation, which in turn projects to the **intralaminar thalamic nuclei (centromedian nucleus)**. Unlike the VPL which projects specifically in a somatotopic fashion to primary somatosensory cortex, the **intralaminar nuclei** project diffusely to the entire cerebral cortex and are thought to be involved in behavioral arousal.

The **spinomesencephalic tract** projects to the midbrain periaqueductal gray matter and the superior colliculi. The **periaqueductal gray** participates in central modulation of pain (see below for a more detailed discussion).

The anterolateral pathways also convey **crude touch** in addition to pain and temperature sensation, and thus, can help maintain sense of touch when damage to the posterior columns occurs.

To summarize the functions of the three tracts that make up the anterolateral pathway, if you step on a tack with your foot, your spinothalamic tract enables you to realize “something sharp is puncturing the sole of my foot”; your spinothalamic intralaminar projections and spinoreticular tract cause you to feel “ouch! That hurts!”; and your spinomesencephalic tract leads to pain modulation, allowing you eventually to think “aah, that feels better.”

The location of the spinal cord sensory and motor spinal cord pathways are depicted in the figure below (including some pathways not yet covered). Note the ascending sensory pathways are shown on the left side, and the descending motor pathways are shown on the right side, of the figure although in reality both sides mediate both sensory and motor functions.



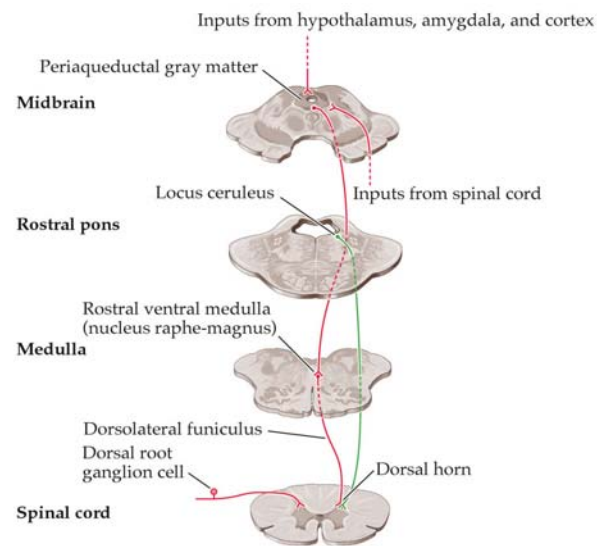
Somatosensory Cortex. From the thalamic VPL (body & neck) and VPM (face) nuclei, somatosensory information is conveyed to primary somatosensory cortex in the postcentral gyrus (Brodmann’s areas 3,1,2). Like primary motor cortex, primary somatosensory cortex is somatotopically organized, with the face represented most laterally and the leg most medially. Information from primary somatosensory cortex is conveyed to the **secondary somatosensory cortex (SII)** located with the Sylvian (lateral) fissure, along its superior margin in a region

called the parietal operculum. SII is also somatotopically arranged. Further processing of somatosensory information occurs in association cortex of the **superior parietal lobule**, including Brodmann's areas 5 and 7. Lesions of primary and association somatosensory regions produce characteristic deficits referred to as **cortical sensory loss**.

Central Modulation of Pain. Pain modulation involves interactions between local circuits at the level of the spinal cord dorsal horn and distal modulatory inputs.

In a mechanism called, **gate control theory**, sensory inputs from fast, large-diameter, non-pain A-beta fibers reduce pain transmission through the dorsal horn by blocking (or getting there first) the slower, unmyelinated C pain fibers. Thus, for example, transcutaneous electrical nerve stimulation (TENS) devices work to reduce chronic pain by activating A-beta fibers. This is also why shaking your hand after striking your thumb with a hammer temporarily helps relieve the pain.

The **periaqueductal gray** receives inputs from the hypothalamus, amygdala, and cortex, and inhibits pain transmission in the dorsal horn of the cord by stimulating release of serotonin (5-HT) from the raphe nuclei in the **rostral ventral medulla** (RVM) (see figure below). The RVM also sends inputs to the locus ceruleus, which in turn sends noradrenergic (NE) projections to modulate pain in the dorsal horn of the cord (see figure below).



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The Thalamus (meaning “inner chamber” in Greek) is an important processing center located in the middle of the brain. Nearly all pathways that project to cerebral cortex do so via synaptic relays in the thalamus. Although the thalamus is a major sensory relay station, it also conveys nearly all other inputs to the cortex, including motor inputs from the cerebellum and basal ganglia, limbic inputs, and widespread modulatory inputs involved in behavioral arousal

and sleep-wake cycles. The thalamus will be discussed fully in this section because of its importance in sensory processing.

The thalamus is part of the **diencephalon**, together with the hypothalamus and epithalamus. The thalamus is just rostral to the midbrain. In horizontal sections, the thalami are visible as deep gray matter structures, shaped somewhat like eggs, with their posterior ends angled outward, forming an inverted V.

The thalamus is divided into a **medial nuclear group**, **lateral nuclear group**, and **anterior nuclear group** by a Y-shaped white matter structure called the **internal medullary lamina**. Nuclei located within the internal medullary lamina itself are called the **intralaminar nuclei**. Finally, the **thalamic reticular nucleus** (not to be confused with the reticular nuclei in the brainstem) forms an extensive but thin sheet enveloping the lateral aspect of the thalamus. There are three main categories of thalamic nuclei:

1. Relay nuclei
2. Intralaminar nuclei
3. Reticular nucleus

Relay nuclei. Most of the thalamus is made up of relay nuclei, which receive inputs from numerous pathways and then project to the cortex. In addition, relay nuclei receive massive reciprocal connections back from cortex. Projections of relay nuclei to cortex may be localized to specific cortical regions or more diffuse.

Specific thalamic relay nuclei. Projections from thalamus to the primary sensory and motor areas tend to be the most localized. These specific relay nuclei lie mainly in the lateral thalamus. All sensory modalities, with the exception of olfaction, have specific relays in the lateral thalamus en route to their primary cortical areas. For example, somatosensory pathways relay in the ventral posterior lateral (**VPL**) and ventral posterior medial (**VPM**) nuclei before projecting to cortex. The major thalamic relay nuclei are summarized in the table below.

TABLE 7.3 Major Thalamic Nuclei (Part 1)

NUCLEI ^a	MAIN INPUTS ^b	MAIN OUTPUTS	DIFFUSENESS OF PROJECTIONS TO CORTEX ^c	PROPOSED FUNCTIONS
RELAY NUCLEI				
Lateral nuclear group				
Ventral posterior lateral nucleus (VPL)	Medial lemniscus, spinothalamic tract	Somatosensory cortex	+	Relays somatosensory spinal inputs to cortex
Ventral posteromedial nucleus (VPM)	Trigeminal lemniscus, trigeminothalamic tract, taste inputs	Somatosensory and taste cortex	+	Relays somatosensory cranial nerve inputs and taste to cortex
Lateral geniculate nucleus (LGN)	Retina	Primary visual cortex	+	Relays visual inputs to cortex
Medial geniculate nucleus (MGN)	Inferior colliculus	Primary auditory cortex	+	Relays auditory inputs to cortex
Ventral lateral nucleus (VL)	Internal globus pallidus, deep cerebellar nuclei, substantia nigra pars reticulata	Motor, premotor, and supplementary motor cortex	+	Relays basal ganglia and cerebellar inputs to cortex
Ventral anterior nucleus (VA)	Substantia nigra pars reticulata, internal globus pallidus, deep cerebellar nuclei	Widespread to frontal lobe, including prefrontal, premotor, motor, and supplementary motor cortex	+++	Relays basal ganglia and cerebellar inputs to cortex
Pulvinar	Tectum (extrageniculate visual pathway), other sensory inputs	Parietotemporo-occipital association	++	Behavioral orientation toward relevant visual and other stimuli

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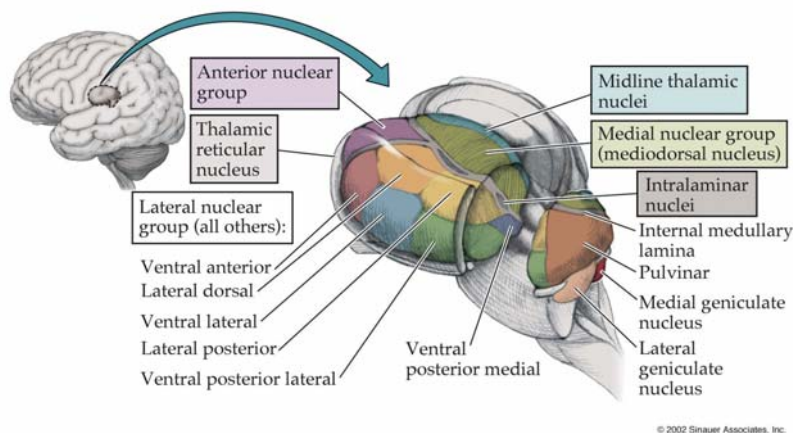
TABLE 7.3 Major Thalamic Nuclei (Part 2)

NUCLEI ^a	MAIN INPUTS ^b	MAIN OUTPUTS	DIFFUSENESS OF PROJECTIONS TO CORTEX ^c	PROPOSED FUNCTIONS
Lateral dorsal nucleus	See anterior nucleus	—	++	Functions with anterior nuclei
Lateral posterior nucleus	See pulvinar	—	++	Functions with pulvinar
Ventral medial nucleus	Midbrain reticular formation	Widespread to cortex	+++	May help maintain alert, conscious state
Medial nuclear group				
Mediodorsal nucleus (MD)	Amygdala, olfactory cortex, limbic basal ganglia	Frontal cortex	++	Limbic pathways, major relay to frontal cortex
Anterior nuclear group				
Anterior nucleus	Mammillary body, hippocampal formation	Cingulate gyrus	+	Limbic pathways
Midline thalamic nuclei Paraventricular, parataenial interanteromedial, intermediodorsal, rhomboid, reuniens (medial ventral)	Hypothalamus, basal forebrain, amygdala, hippocampus	Amygdala, hippocampus, limbic cortex	++	Limbic pathways

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As may be seen in the table above, visual information is relayed in the ***lateral geniculate nucleus (LGN)***, and auditory information is relayed in the ***medial geniculate nucleus (MGN)***. Motor pathways leaving the cerebellum and basal ganglia also have specific thalamic relays in the ***ventral lateral nucleus (VL)*** en route to premotor and supplementary motor areas. Some limbic pathways have fairly specific cortical projections, such as those carried by the ***anterior nuclear group*** to the anterior cingulate gyrus.

Widely projecting (nonspecific) thalamic relay nuclei.



There are two important nonspecific thalamic relay nuclei: the **dorsomedial nucleus** and the **pulvinar**.

Diffuse relays of limbic inputs occur in the **dorsomedial nucleus**, as well as in the midline and intralaminar thalamic nuclei. The dorsomedial nucleus forms a large buldge lying medial to the internal medullary lamina and serves as the major thalamic relay for information traveling to the frontal association cortex.

The **pulvinar** is a large, pillow-shaped nucleus that occupies most of the posterior thalamus (see figure above). It takes visual and other sensory inputs and relays then to large regions of parietal, temporal, and occipital association cortex involved in behavioral orientation (attention) toward relevant stimuli.

Intralaminar Nuclei lie within the internal medullary lamina (see figure above) and receive inputs from numerous pathways and have reciprocal connections with cortex. Their main inputs and outputs come from the basal ganglia via the **centromedian nucleus** among others. The **rostral intralaminar nuclei** relay inputs from the ascending reticular activating system (RAS) to the cortex and play an important role in maintaining the alert, conscious state.

Reticular Nucleus forms a thin sheet surrounding the lateral portions of the thalami and are just medial to the internal capsule. The reticular nucleus is the only thalamic nucleus that does not project to cortex. It receives inputs mainly from other thalamic nuclei and the cortex and projects back to the thalamus. The reticular nucleus consists of an almost pure population of inhibitory GABAergic neurons and is thus suited to its role to inhibit or modulate thalamic output to cortex, thereby altering state of alertness and attention.

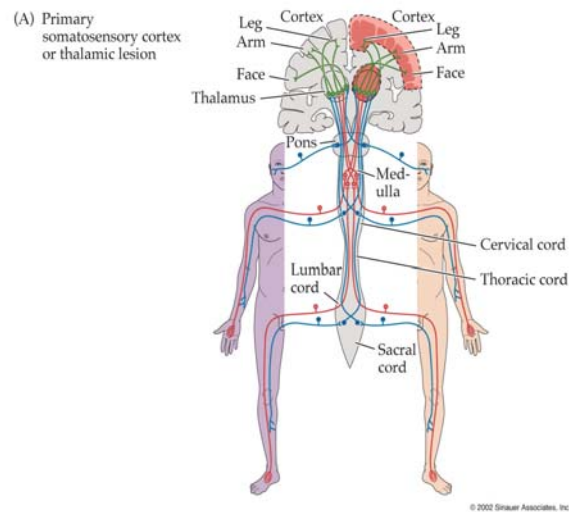
KEY CLINICAL CONCEPTS – SENSORY

Paresthesias are abnormal positive sensory experiences. The character and location of paresthesias may have localizing value. In posterior column-medial lemniscus lesions, patients commonly describe a tingling, numb

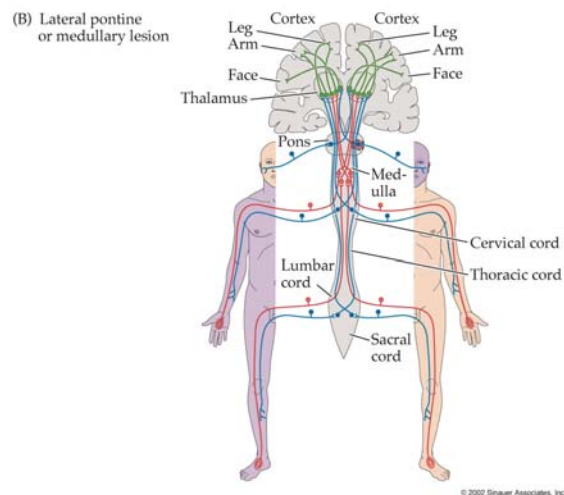
sensation, a feeling of a tight bandlike sensation around the trunk or limbs, or a sensation similar to gauze on the fingers when trying to feel objects.

Lesions of the anterolateral pathways often result in sharp, burning, or searing pain. Lesions of the parietal lobe in primary sensory cortex may cause contralateral numbness and tingling or pain. Lesions of the thalamus can cause severe pain.

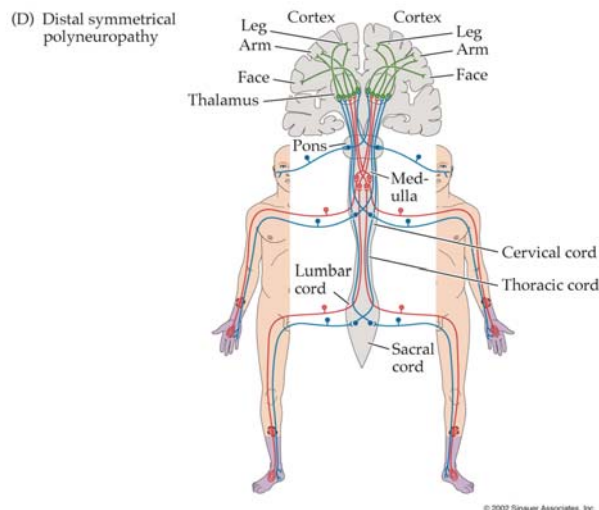
Patterns and localization of sensory loss. Sensory loss may be caused by lesions anywhere in the somatosensory pathways. Some common sites of lesion and associated sensory loss are shown below in the figures where lesions are shown in red and regions of sensory loss are shown in purple.



Lesions of primary sensory cortex and/or VPL and VPM nuclei of thalamus will result in a contralateral sensory loss in one-half the body (depicted in figure above). All sensory modalities may be involved, sometimes with no motor deficit.

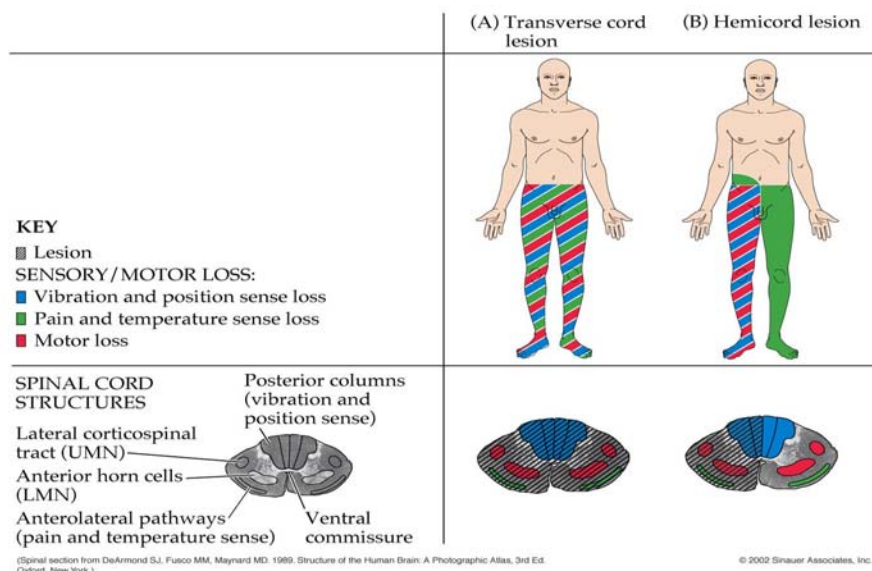


Lesions in the lateral medulla or pons can cause a lateral medullary syndrome where the lesion involves both the anterolateral pathways and the spinal trigeminal nucleus on the same side. It causes loss of pain and temperature sensation in the contralateral body, but loss of pain and temperature in the ipsilateral face (see figure above).



Distal symmetrical polyneuropathies can cause bilateral sensory loss in a “glove and stocking” distribution in all sensory modalities (see figure above). In peripheral neuropathies, the lesions involve specific nerves or nerve roots. These peripheral nerve lesions also frequently cause lower motor neuron-type weakness.

Spinal cord syndromes. The sensory and motor deficits that accompany spinal cord lesions may be used to localize the lesion. Examples are given here, but refer to the book (page 280) for a more complete discussion of these syndromes.



In Figure A above, there are sensory and motor deficits at and below the level of the spinal cord lesion which involves all pathways.

In Figure B above, damage to the lateral corticospinal tract causes *ipsilateral* upper motor neuron-type weakness and interruption of the posterior columns causes *ipsilateral* loss of vibration and joint position sense. Interruption

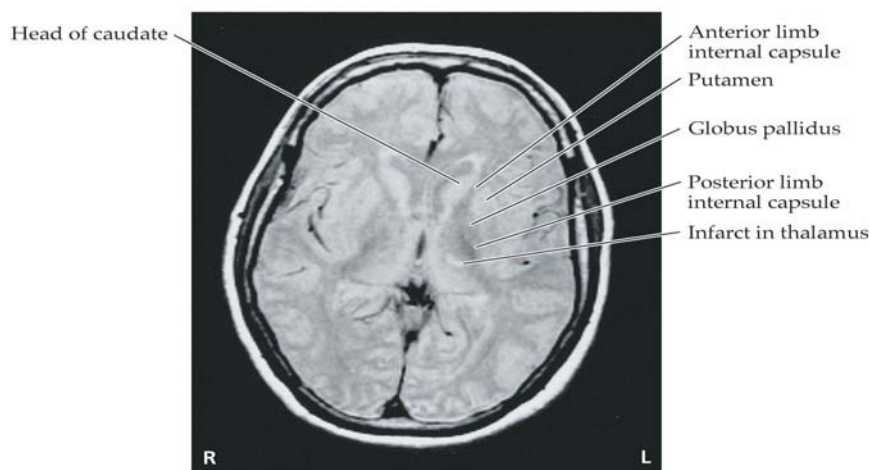
of the anterolateral systems, however, causes *contralateral* loss of pain and temperature sensation. See page 280 for other common syndromes.

CLINICAL CASE – SENSORY

CASE 1. A 62 year-old woman came to a medical clinic because of 2 days of numbness of her right face, arm, and leg. The patient awoke 2 days ago with decreased sensation in her right face and arm “as though they were asleep.” There were no difficulties with language, motor skills, or vision. She also noticed a loss of sensation in her right foot. Past medical history was significant for hypertension and cigarette smoking. Her father had a stroke at age 64.

Neurologic examination: Normal mental status. Cranial nerves were normal except for decreased pinprick, temperature, and light touch sensation in the right face. Motor exam was normal. Reflexes = normal. Coordination = normal. Gait = normal. Sensory exam = decreased pinprick, temperature, light touch and vibration sense on the right half of the body.

These circumscribed deficits are sometimes seen in conversion disorders, but may also be caused by small lesions of the thalamus involving the VPL and VPM nuclei of the thalamus. Thus, the most likely location of this lesion is in these nuclei in the left thalamus. Given the sudden onset of symptoms and history of hypertension, smoking and family history of stroke, the most likely diagnosis is an ischemic infarct of the left thalamus.



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This was confirmed by a brain MRI (see above) showing increased T2 signal in the lateral thalamus consistent with a lacunar infarct.

Her numbness gradually improved and was completely gone 5 days after first coming to medical attention. Neurological work-up for embolic causes of the infarction was negative. She was started on medicine to better control her hypertension as well as aspirin and Coumadin for stroke prevention.