NAME: OKOR PRECIOUS EIKHOMUN

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**General Organization of Somatosensory Pathways**

**Sensory pathways** consist of the chain of neurons, from receptor organ to cerebral cortex, that are responsible for the perception of sensations.

**Common Anatomical Features**

Somatosensory stimuli activate a chain of neurons starting with the peripheral first-order (1°) afferent and ending in the cerebral cortex

Within each somatosensory pathway,

* The 1° afferent is a pseudounipolar neuron that has its cell body located in a peripheral (spinal or cranial) ganglion. It has a peripheral axon that forms or innervates somatosensory receptors and a central process that synapses with 2° afferent neuron(s) in a spinal cord or brain stem nucleus.
* The 2° afferent may synapse with 3° afferent neurons in the spinal cord or may ascend the neuraxis to synapse with 3° afferent neurons in the thalamus.
* There is a decussation (i.e., axons crossing the midline to the opposite side of the spinal cord or brain stem) in each somatosensory pathway below the level of the thalamus.
* All somatosensory pathways include a thalamic nucleus. The thalamic neurons send their axons in the posterior limb of the internal capsule to end in the cerebral cortex.
* Most somatosensory pathways terminate in the parietal lobe of the cerebral cortex.
* Each somatosensory pathway is named after a major tract or nucleus in the pathway.
* In general, conscious perception of sensory stimuli requires the involvement of neurons in the thalamus and cerebral cortex. For example, electrical stimulation of a structure in pathways connecting muscle and joint receptors to the cerebellum (e.g., electrical stimulation of the anterior spinocerebellar tract) will not produce a sensation of limb movement, as these pathways do not include the thalamus or cortex. In contrast, electrical stimulation of a structure in the posterior column-medial lemniscal pathway (e.g., electrical stimulation of the medial lemniscus) may result in a sensation of limb movement, as this pathway includes the thalamus and terminates in the cerebral cortex.

**Peripheral Somatosensory Axons**

The morphology of the **peripheral somatosensory axon** is related to the receptor it innervates or forms and to the sensory information it carries (Figure 4.2).

* The Group I and II 1° afferent axons, which form the muscle/tendon receptors and carry body proprioceptive information, have the largest diameter and the thickest myelin of all the somatosensory 1° afferent axons.
* The Type C 1° afferent axons, which form free nerve endings and carry dull pain, deep pain, crude touch or warm/hot information, are the smallest 1° afferent axons and are unmyelinated.
* The Type Aδ1° afferent axons, which form free nerve endings and carry sharp pain or cool/cold information, are thinly myelinated and larger than the Type C axons.
* The Type Aβ 1° afferent axons, which form encapsulated endings in skin and joints or hair follicle endings or Merkel disks in skin, are myelinated and have diameter less than Group I afferents and greater than the Type Aδ 1° afferent axons.
* The morphology of the peripheral somatosensory axon is also related to the conduction velocity of the action potentials generated by the axon.
* The conduction velocity of an axon is determined by electrically stimulating the axon and recording the time (latency) it takes the electrically elicited action potential to reach a recording electrode (Figure 4.3). The distance traveled from the electrical stimulating site to the recording site divided by the latency provides the conduction velocity of the axon.
* As discussed in earlier chapters, the larger and more heavily myelinated the axon, the greater its conduction velocity (Figure 4.3). Consequently, the 1° afferent axons carrying information required for fine motor control and rapid reflex responses (i.e., those forming body proprioceptors) conduct action potentials rapidly, whereas those carrying information about body and object temperature conduct action potentials at a much slower rate.
* The whole nerve potential or compound action potential (CAP) is recorded extracellularly from an electrically stimulated nerve and is the sum of the signals produced by each of the individual action potentials of the axons forming the nerve. (Figure 4.3) The mixed nerve (afferent and efferent axons) compound action potential has three prominent peaks that are called A, B and C. The conduction velocity of an axon determines the axon's contribution to the compound action potential peaks. Specifically, the faster the axon conduction velocity, the shorter the latency of axon response and the greater the axon's contribution to the shorter latency peaks (e.g., compare columns CAP Peak and Conduction Velocity in Table I). The axons contributing to a given compound action potential peak (e.g., peak A) are named according to the peak name (e.g., Type A axon). When the relative amplitudes of the peaks differ from those generated by "normal" nerves, the types of damaged axons can be assessed by determining which peaks are abnormal. Consequently, the compound action potential is used clinically to detect nerve damage and to monitor the progress of the regeneration of damaged nerves.
* Somatosensory Receptors and their Peripheral Axons**Receptor TypeAxon3 GroupCAP PeakConduction VelocityAxon DiameterInformation Processed**Muscle Spindle: Annulospiral endings1aAα70-120 m/sec1-20 μMMuscle length and velocity
* Muscle Spindle:  
  Flower Spray endings
* IIAβ 30-70 m/sec6-12 μMMuscle lengthGolgi Tendon OrganIbAα70-120 m/sec12-20 μMMuscle tensionJoint: PacinianIIAβ30-70 m/sec6-12 μMJoint movementJoint: RuffiniIIAβ30-70 m/sec6-12 μMJoint angleJoint: Golgi Tendon OrganIIAβ30-70 m/sec6-12 μMJoint torqueMeissner corpuscleIIAβ30-70 m/sec6-12 μMTouch, flitter or movementPacinian corpuscleIIAβ30-70 m/sec6-12 μMVibrationRuffini corpuscleIIAβ30-70 m/sec6-12 μMSkin stretchHair follicleII & IIIAβ & Aδ10-70 m/sec2-12 μMTouch movementMerkel complexIIAβ30-70 m/sec6-12 μMFine touchFree Nerve endingsIIIAδ5-30 m/sec1-6 μMSharp pain or cool/coldFree nerve endingsIVC0.5-2 m/sec<1.5 μMDull or aching pain, or touch or warm

For historical reasons, the terminology based on axon conduction velocity (Group I, II, III and IV) is used for afferent and efferent axons innervating muscles and tendons. And the terminology based on the compound action potential (Type A, B or C) is used for afferent axons innervating the skin, joints and viscera.

Note that the fastest conducting somatosensory 1° afferents (Group Ia) innervate skeletal muscle and the slowest (C-fibers) form the receptors of the pain systems. While one might expect painful, tissue damaging stimuli to have priority over all other somatosensory stimuli, the afferent information required to control the reaction to the painful stimuli are conveyed by the faster conducting muscle and joint afferents. Even afferents providing more exact information about the location of a cutaneous stimulus, the Aβ axons, conduct at a faster rate than the Aδ and C axons carrying information about painful stimuli.

**Somatotopic Organization**

Somatosensory neurons are topographically (i.e., spatially) organized so that adjacent neurons represent neighboring regions of the body or face. This organization is preserved by a precise point-to-point somatotopic pattern of connections from the spinal cord and brain stem to the thalamus and cortex. Consequently, within each somatosensory pathway there is a complete map (spatial representation) of the body or face in each of the somatosensory nuclei, tracts, and cortex.

**Somatosensory Pathways**

The sensory information processed by the somatosensory systems travels along different anatomical pathways depending on the information carried. For example, the posterior column-medial lemniscal pathway carries discriminative touch and proprioceptive information from the body, and the main sensory trigeminal pathway carries this information from the face. Whereas, the spinothalamic pathways carry crude touch, pain and temperature information from the body, and the spinal trigeminal pathway carries this information from the face.

**Medial Lemniscal Pathway: Body Discriminative Touch and Proprioception**

The posterior (dorsal) column - **medial lemniscal pathway** (i.e., the medial lemniscal pathway) carries and processes discriminative touch and proprioceptive information from the body. It is important to keep in mind that within the medial lemniscal pathway, the afferents carrying discriminative touch information are kept separate from those carrying proprioceptive information up to the level of the cerebral cortex.

The peripheral axons of the 1° afferents are myelinated, large or medium diameter axons. Each axon travels via a posterior root, spinal nerve and peripheral nerve to skin, muscle or joint- where it forms or innervates a somatosensory receptor.

The 1° medial lemniscal afferent peripheral process that end in the

* skin, are Aβ axons that branch to innervate hair follicles or Merkel’s cells or form Meissner, Pacinian or Ruffini corpuscles.
* joints, are Aβ axons that branch to form encapsulated endings similar to the Ruffini and Pacinian corpuscles and Golgi tendon organs.
* muscle, are Group I and II axons that branch to terminate in muscle spindles (Ia and II axons) or Golgi tendon organs (Ib axons).

The 1° medial lemniscal afferent central axons

* join a posterior root, enters the spinal cord, and ascends to the brain stem in the posterior column of the spinal cord (Figure 4.5).
* of coccygeal to mid-thoracic posterior roots (i.e., up to T7) ascend the spinal cord in the ipsilateral gracile fasciculus.
* of the upper thoracic (level T6 and above) and cervical roots collect in the ipsilateral cuneate fasciculus.
* of the gracile and cuneate fasciculi are collectively called the posterior funiculus or posterior column.
* ascends the spinal cord in the posterior funiculus up to the medulla without synapsing or decussating (i.e., without crossing the midline to the contralateral half of the spinal cord).

In the medulla,

* the 1° afferents in the gracile fasciculus synapse in the gracile nucleus
* the 1° afferents in the cuneate fasciculus synapse in the cuneate nucleus.
* the axons of the gracile and cuneate nuclei (2° afferents) pass anteriorly and decussate to form the medial lemniscus, contralateral to their cells of origin.
* above the level of the gracile and cuneate nuclei, each half of the body is represented contralaterally (e.g., left half of body in right medial lemniscus) within the medial lemniscal pathway.

The 2° medial lemniscal afferents

* ascend the brain stem in the medial lemniscus to the diencephalon.
* terminate in the ventral posterolateral (VPL) nucleus of the thalamus.
* carrying cutaneous information terminate in the core of the VPL.
* carrying proprioceptive information terminate in the surrounding shell of the VPL.

The axons of the VPL 3° afferent neurons

* travel in the posterior limb of the internal capsule.
* terminate in the postcentral gyrus and posterior paracentral lobule of the parietal lobe.

The postcentral gyrus and posterior paracentral lobule

* are called the primary somatosensory cortex.
* are the primary cortical receiving areas of the somatosensory system.

*The lower part of the body (foot and leg) are represented in the posterior paracentral lobule, whereas the upper body (chest, arm, and hand) are represented in the upper postcentral gyrus*

The action potentials ascend the spinal cord via the central process of the 1° afferent in the fasciculus gracilis of the posterior column until they reach the medulla. In the medulla, the action potentials initiate the release of neurotransmitter from the 1° afferent axon terminals onto 2° afferents within the gracile nucleus. The 2° afferent generates action potentials that are conducted by its axons, which decussate to form the medial lemniscus. These action potentials are conducted by the 2° afferent axon contralateral to their site of origin and contralateral to the foot where the stimulus was applied. The action potentials ascend to the thalamus where they initiate the release of neurotransmitter from the 2° afferent axon terminals. They release neurotransmitters onto the 3° afferents in the core of the VPL of the thalamus. The action potentials generated by the 3° VPL afferents are conducted by their axons, which travel in the posterior limb of the internal capsule, to the posterior paracentral lobule of the parietal cortex. These action potentials initiate the release of neurotransmitter from the 3° afferent axon terminals onto cortical neurons and initiate the higher-order processing of the stimulus information generated by the Meissner corpuscle. The point-to-point connections within the pathway provide the basis for a somatotopic map that is used to locate the area of contact with the stimulus and for modality specific information used to identify the stimulus as tactile and from a Meissner corpuscle.

**Main Sensory Trigeminal Pathway: Face Discriminative Touch and Proprioception**

The**main sensory trigeminal pathway** carries and processes discriminative touch and proprioceptive information from the face. Consequently, it is the cranial homologue of the medial lemniscal pathway.

The cranial 1° main sensory trigeminal afferent neurons

* peripheral processes are located in the trigeminal (predominantly), facial, glossopharyngeal and vagus nerves.
* form mechanoreceptors in the skin, mucous membranes, muscles and joints of the face. The relationship between receptor type formed and the axon Type/Group are similar to those of the medial lemniscal 1° afferents.
* have pseudounipolar cell bodies in the cranial ganglia of the trigeminal, facial, glossopharyngeal and vagus nerves
* send their central axons to the brain stem.
* synapse in the main sensory trigeminal nucleus (2° afferents).

The main sensory trigeminal 2° afferent axons

* decussate immediately on leaving the main sensory trigeminal nucleus.
* join the contralateral ventral trigeminal lemniscus.
* above the level of the main sensory trigeminal nucleus (i.e., the mid pons), carries information about the contralateral face (i.e., the right ventral trigeminal lemniscus carries information about the left side of the face).

The 2° main sensory trigeminal afferents in the ventral trigeminal lemniscus

* ascend to the diencephalon.
* terminate in the ventral posteromedial (VPM) nucleus of the thalamus.

The axons of the 3° main sensory trigeminal afferents (VPM neurons)

* travel in the posterior limb of the internal capsule.
* end in the postcentral gyrus of the parietal lobe.

The **postcentral gyrus**

* is part of the primary cortical receiving area of the somatosensory system.

*The face is represented in the lower half of the postcentral gyrus* A Merkel receptor in the left cheek is stimulated, and its 1° afferent generates action potentials that are conducted by the 1° afferent Ab axon, past its pseudounipolar soma, into the brain stem. The 1° afferent central process conducts the action potentials into the pons where they initiate the release neurotransmitter from the 1° afferent axon terminals. The neurotransmitter is released onto 2° afferents within the main sensory trigeminal nucleus. The 2° afferent generates action potentials that are conducted along its axon, which decussates in the pons to join the ventral trigeminal lemniscus. These action potentials are conducted by the 2° afferent axon contralateral to their site of origin and contralateral to the site where the stimulus was applied. The action potentials ascend to the thalamus where they initiate the release of neurotransmitter from the 2° afferent axon terminals. They release neurotransmitters onto the 3° afferents in the core of the VPM of the thalamus. The action potentials generated by the 3° VPM afferents are conducted by their axons, which travel in the posterior limb of the internal capsule, to the postcentral gyrus of the parietal cortex. These action potentials initiate the release of neurotransmitter from the 3° afferent axon terminals onto cortical neurons and initiate the higher-order processing of the stimulus information generated by the Merkel cell. The point-to-point connections within the pathway provide the basis for a somatotopic map that is used to locate the area of contact with the stimulus and for modality specific information used to identify the stimulus as tactile and from a Merkel cell.

There is a minor proprioceptive component for the jaw in cranial nerve V that has 1° afferent cell bodies located in the mesencephalic trigeminal nucleus. The peripheral axons of these afferents travel in the mandibular branch of the trigeminal nerve and end in the jaw muscles and joint. The central processes of most of these afferents end in the trigeminal motor nucleus that controls the muscles of the jaw. Few synapse in the main sensory trigeminal nucleus.

**Neospinothalamic Pathway: Body - Sharp Prickling Pain and Cool/Cold**

The **neospinothalamic pathway** carries and processes sharp, pricking pain and dropping temperature (cool/cold) information from the body .The pain information carried by the neospinothalamic pathway is well localized and the sensations are the short lasting “fast” or “first” pain elicited by tissue-damaging cutaneous stimuli. The neospinothalamic pathway is also characterized by somatotopic representation, which allows for accurate localization of the painful stimulus.

Recall that there are multiple spinal pathways processing pain information. Most of the ascending afferents of the spinal pain pathways travel with the neospinothalamic afferents in a fiber tract called the "spinothalamic tract" or "anterolateral spinothalamic tract". Elements of these other pain pathways will be mentioned below to help you understand how pain sensations may remain after damage to the neospinothalamic pathway.

The 1° neospinothalamic afferents

* have Type Aδ peripheral axons that form free nerve endings in skin, muscles and joints.
* have central processes that enter the spinal cord.
* synapse in the posterior marginal nucleus (2° afferents) of the posterior horn.

The 2° neospinothalamic afferent axons

* decussate in the spinal cord anterior white commissure.
* form the lateral part of the spinothalamic tract in the lateral funiculus.

*Note that the fibers in the lateral spinothalamic tract are contralateral to their cells of origin and contralateral to the body area they represent.*

The crossed 2° neospinothalamic afferent axons

* ascend the spinal cord and brain stem as part of the spinothalamic tract.
* travel with other pain (archispinothalamic) afferents that leave the spinothalamic tract and terminate in the brain stem as:
  + spinoreticular fibers that end in the reticular formation of the brain stem.
  + spinomesencephalic fibers that end near the periaqueductal gray of the midbrain.
* travel with other pain (paleospinothalamic) afferents to the diencephalon where
  + the neospinothalamic afferents terminate in the ventral posterolateral (VPL) nucleus of the thalamus.
  + the paleospinothalamic afferents terminate in the intralaminar nuclei of the thalamus.

The spinothalamic afferent axons from the thalamus

* travel in the posterior limb of the internal capsule.
* VPL (i.e., the 3° neospinothalamics) end in the postcentral gyrus and posterior paracentral lobule of the parietal lobe.
* Intralaminar nuclei (i.e., paleospinothalamics) end in the insula and rostral cingulate gyrus.

The postcentral gyrus and posterior paracentral lobule are

* the neospinothalamic pathway termination sites.
* the primary cortical receiving areas for sharp, cutting pain information.
* not the exclusive cortical receiving area for pain information.

The insula and rostral cingulate gyrus

* are the archispinothalamic and paleospinothalamic pathways' termination sites.
* receive dull and deep pain information.
* are responsible for poorly localized, longer lasting pain sensations and add the emotional (i.e., unpleasant) features to these sensations.

The action potentials enter the spinal cord via the central process of the 1° afferents to initiate the release neurotransmitter from the 1° afferent axon terminals onto 2° afferents within the posterior marginal nucleus. The 2° afferent generates action potentials that are conducted by its axon, which decussates in the anterior white commissure of the spinal cord. The crossed 2° neospinothalamic afferent axons form the lateral component of the spinothalamic tract. The action potentials conducted by the crossed 2° afferent axon are contralateral to their site of origin and contralateral to the foot where the stimulus was applied. The action potentials ascend to the thalamus where they initiate the release of neurotransmitter from the 2° afferent axon terminals. They release neurotransmitters onto the 3° afferents in the VPL of the thalamus. The action potentials generated by the 3° VPL afferents are conducted by their axons, which travel in the posterior limb of the internal capsule, to the posterior paracentral lobule of the parietal cortex. These action potentials initiate the release of neurotransmitter from the 3° afferent axon terminals onto cortical neurons and initiate the higher-order processing of the stimulus information generated by the free nerve ending. The point-to-point connections within the pathway provide the basis for a somatotopic map that is used to locate the area of contact with the stimulus and for modality specific information used to identify the stimulus as a sharp pinprick.

**Spinal Trigeminal Pathway: Face Pain, Temperature and Crude Touch**

The **spinal trigeminal pathway** carries and processes crude touch, pain and temperature information from the face Consequently, it is the cranial homologue of the spinothalamic pathways i.e., homologous to all the spinothalamic pathways, the archi-, paleo- and neo-spinothalamic pathways. As in the spinothalamic pathways, the afferents carrying crude touch information are kept separate from those carrying temperature information and from others carrying pain information. Also the trigeminal afferents carrying sharp, cutting pain information are segregated from those carrying dull, burning pain and deep aching pain information.

The 1° spinal trigeminal afferents

* are located in the same nerves and ganglia as those of the main sensory trigeminal pathway.
* have Aδ and C peripheral axons that form free nerve endings in the dura and face.
* on entering the brain stem, form the spinal trigeminal tract.

The spinal trigeminal tract

* extends from mid pontine levels (the level of entry of trigeminal nerve) down to C1 of the spinal cord.
* consists of spinal trigeminal 1° afferent axons (predominantly of the trigeminal nerve).
* 1° afferents synapse in the spinal trigeminal nucleus (2° spinal trigeminal afferents).

The 2° spinal trigeminal afferent axons

* decussate and form the ventral trigeminal lemniscus contralateral to their cells of origin.
* ascend in the ventral trigeminal lemniscus as crossed 2° spinal trigeminal afferents.
* travel with afferents that leave the ventral trigeminal lemniscus as trigeminoreticular fibers, which terminate in the brain stem reticular formation.
* are joined by the crossed 2° main sensory trigeminal afferents at mid-pons.
* travel with afferents that leave the ventral trigeminal lemniscus as trigeminomesencephalic fibers, which terminate near the midbrain periaqueductal gray.
* terminate in the VPM and in the intralaminar nuclei of the thalamus.

Multiple thalamic nuclei process information in this pathway

* the VPM processes sharp pricking pain.
* the intralaminar nuclei processes other poorly localized sensations of dull, burning pain, deep, aching pain, temperature and crude touch.

The 3° spinal trigeminal afferent axons from the thalamus:

* travel in the posterior limb of the internal capsule.
* end in multiple areas of the cerebral cortex.

The spinal trigeminal pathway terminates in multiple cortical areas:

* the 3° VPM axons end in the primary somatosensory cortex, which provides for accurate localization in the face area of the source of the sharp, pricking pain.
* the intralaminar nuclei axons terminate in the cingulate gyrus and insula of the cerebral cortex, which provide for poorly localized sensations of dull and aching pain, temperature and crude touch.

The 1° afferent central process bypasses the main sensory trigeminal nucleus and descends the brain stem in the spinal trigeminal tract. The action potentials are conducted in this descending tract to the spinal trigeminal nucleus, where they initiate the release neurotransmitter from the 1° afferent axon terminals. The neurotransmitter is released onto 2° afferents within the spinal trigeminal nucleus. The 2° afferent generates action potentials that are conducted along its axon, which decussates to form the ventral trigeminal lemniscus. These action potentials are conducted by the 2° afferent axon contralateral to their site of origin and contralateral to the cheek where the stimulus was applied. The action potentials ascend to the thalamus where they initiate the release of neurotransmitter from the 2° afferent axon terminals. They release neurotransmitters onto the 3° afferents in the VPM. The action potentials generated by the 3° VPM afferents are conducted by their axons, which travel in the posterior limb of the internal capsule, to the postcentral gyrus of the parietal cortex. These action potentials initiate the release of neurotransmitter from the 3° afferent axon terminals onto cortical neurons and initiate the higher-order processing of the stimulus information generated by the free nerve ending. The point-to-point connections within the pathway provide the basis for a somatotopic map that is used to locate the area of contact with the stimulus and for modality specific information used to identify the stimulus as a sharp pinprick.

The Nerve Roots and Ganglia Associated with the Somatic and Visceral Afferent Pathways**Nerve RootGangliaSomatic InnervationVisceral Innervation**Spinal Cord: Sacralposterior root: S5 to S1buttocks, back of leg and foot, genitalslower pelvic region, e.g., RectumSpinal Cord: Lumbarposterior root: L5 to L1lower back, hip, pelvic area, side and front of leg and footleg and pelvic region, e.g., bladderSpinal Cord: Thoracicposterior root: T12 to T1trunk (abdomen, back, and chest), part of arm

lower roots: Lower abdomen (e.g., kidney, colon, appendix)

middle roots: Upper abdomen (e.g., stomach, liver, gall bladder)

upper roots: Chest (e.g., diaphragm, esophagus, lung, heart)

Spinal Cord: Cervicalposterior root: C8 to C2shoulder, arm, hand, fingers, neck and back of headminor to blood vessels and sweat glands of upper body and extremitiesCranial Nerve: Vagus Nerve

jugular (superior)

nodose (inferior)

back of ear, external auditory canal and dura

none

throat, thoracic and abdominal viscera

Cranial Nerve: Glossopharyngeal

superior (jugular)

petrosal (inferior)

back of ear (minor), ear drum, middle ear

ear drum, middle ear, Eustachian tube, tonsil, pharynx, soft palate and posterior tongue

none

carotid body and sinus

Cranial Nerve: Facialgeniculateskin of earminor - Parotid glandCranial Nerve: Trigeminalsemilunarface, eye, oral and nasal cavities, and meningesnone