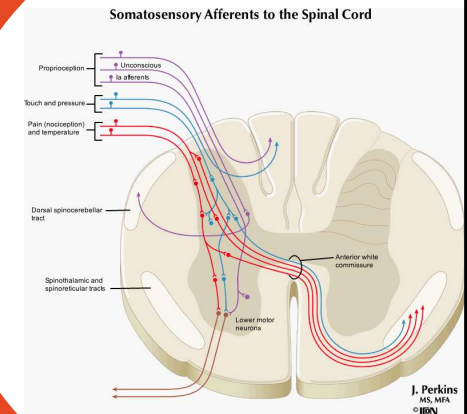


The Somatosensory Systems From The Body



Learning Objectives

1. Describe the classification of sensory nerve fibers according to the fiber diameter and conduction velocity.
2. Indicate the sensory modalities transmitted by the DCML pathways and the type of peripheral receptors involved in this system.
3. Describe the trajectory of the DCML pathway from origin to termination in the cerebral cortex.
4. Indicate the sensory modalities transmitted by the ALS pathways and the type of peripheral receptors involved in this system.
5. Describe the trajectory of the ALS pathways from origin to termination in the cerebral cortex.
6. Indicate the location and name of the cortical areas where somatosensory information from the DCML and ALS pathways is processed.
7. Correlate the effects of a DCML lesion at all CNS levels with clinical findings.
8. Correlate the effects of ALS fiber damage at all CNS levels with clinical exam findings.
9. Briefly describe the pathophysiology of pain.
10. Describe the descending pain-modulating pathway including origin, trajectory in the brainstem and termination on dorsal horn sensory neurons
11. Apply your acquired knowledge to solve clinical cases - we will discuss this objective in the lab.

Sensation And Perception

Sensation is

"...the ability to recognize, transduce, encode and perceive information about the exterior world or the inside of our body."

Perception is

... the conscious experience that the CNS elaborates from a sensation. Perceptions are creations of our brains out of sensory experiences

Sensation And Perception

What is a sensory system?

A sensory system is a collection of parallel fibers transmitting a particular type of sensation from the body surface or the viscera to the CNS.

What defines a sensory system?

A particular type of peripheral receptor, the sensory fibers to the CNS and the area of the cerebral cortex where the processing of this information takes place.

Coding A Sensation

There are several fundamental attributes of a sensory experience encoded and recognized by the nervous system

Modality indicates the type of sensation and is defined by the type of energy transmitted and the specialized receptor involved. This specificity is called the **labeled line principle**

Location is represented by the specific place on the body or viscera where the receptors were activated by the stimulus



Coding A Sensation

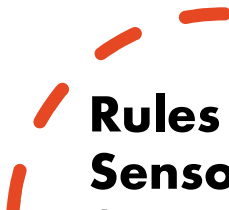
Intensity of a stimulus is signaled by the receptor potential amplitude which is an indication of the total amount of energy delivered to the receptor by the physical stimulus

Timing or duration of a stimulus is encoded by the changes in firing pattern of a neuron and / or when firing of a neuron starts or ends



All sensory systems share 3 common steps

- The presence of a physical stimulus
- A set of events transforming the stimulus into nerve impulses
- A response in the form of a perception or a conscious experience of the sensation



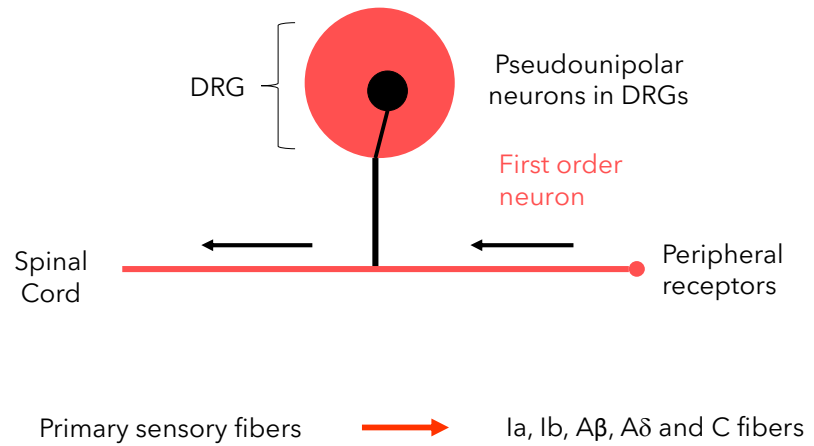
Rules Of Sensory Systems

- ① The sensory receptor captures the stimulus and **transduces** the energy into electrical signals (receptor potentials) outside CNS
- ② The cell body of origin of the primary afferent fiber is the **first neuron of the pathway**, always located in a sensory ganglion in the PNS pseudounipolar
- ③ There is no synapses in a sensory ganglion
- ④ The first synapse of the pathway occurs in the CNS between the **second neuron of the pathway** and the primary afferent fiber
- ⑤ After the second neuron of the pathway, all or some of the secondary afferent fibers usually decussate

ALS - decussate at or around level of where the secondary sensory neuron is DCL is in medulla

Sensory Fibers Origin

The primary sensory fiber which originates from a pseudounipolar (first order) neuron in a sensory ganglion transmits the captured information to the CNS



no synapse here just where cell bodies live

Fiber Size And Velocity

There are 2 systems for the classification of nerve fibers:

- Based on conduction velocity
- Based on measured fiber diameter

stick with numbered system

A, B, C system - based on **conduction velocity**

- **A** fibers are the fastest heavily myelinated sensory and motor fibers. They are subdivided into $A\alpha$, $A\beta$, $A\gamma$, $A\delta$ with $A\delta$ being the slowest and less myelinated of the A group **C** fibers are non-myelinated
- I, II, III and IV system - measured **fiber diameter**
- I, II and III are myelinated fibers and IV are non-myelinated

Fiber Size And Velocity

OBJ. # 1

- **A α** fibers - motor fibers innervating skeletal muscle fibers
- **A γ** fibers - motor fibers innervating intrafusal fibers (muscle spindle)
- **B** fibers - preganglionic autonomic fibers
- **C** fibers are non-myelinated
- **Ia** fibers - sensory afferents from muscle spindle
- **Ib** fibers - sensory afferents from Golgi tendon
- **II** fibers are similar to **A β** - sensory afferents from encapsulated skin or visceral receptors
- **III** fibers are similar to **A δ** fibers - sensory, small myelinated afferent fibers transmitting thermal and nociceptive information
- **IV** fibers are non-myelinated, transmit nociceptive information primarily pain perception

The Sensory Receptors

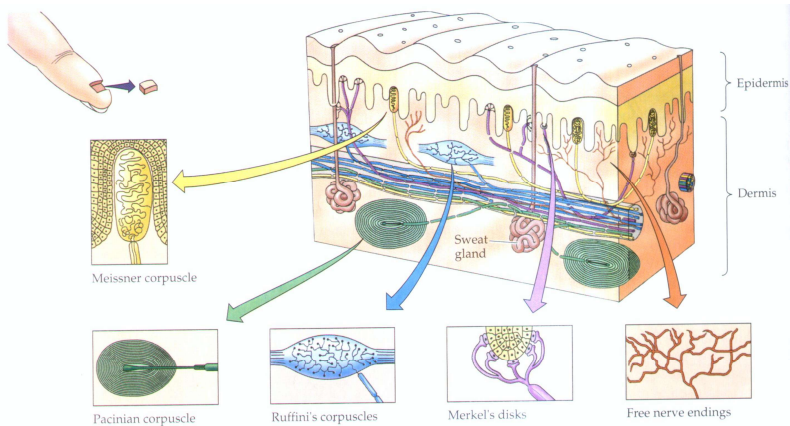
OBJ. # 2

All sensory systems originate with the sensation being captured by a peripheral receptor

Nociceptors

Thermoreceptors

Mechanoreceptors



end sensory organs not the nerve themselves but intertwined with nerve fibers

Free nerve endings are mostly associated with nociception, the perception of pain.

Mechanoreceptors

Cutaneous mechanoreceptors

- Encapsulated or non-encapsulated
- Slowly adapting or rapidly adapting

Encapsulated receptors

- Pacinian corpuscle (rapidly adapting)
- Ruffinian ending (slowly adapting)
- Meissner corpuscle (rapidly adapting)

Non-encapsulated receptors

- Merkel endings (slowly adapting)
- Some are free nerve endings

I won't be asking you to memorize these different receptor types, though I can't guarantee you won't need to know them for the boards.

Meissner's corpuscle – encapsulated, Abeta myelinated sensory fiber, present in non-hairy skin and responsible for a large component of discriminative touch. Rapidly adapting

Merkel's discs – non-encapsulated, Abeta myelinated sensory fiber, slowly adapting, present in non-hairy and hairy skin – allows for sensation of continuous contact.

Ruffini's endings – encapsulated, slowly adapting, present in deep skin and joint capsules

Free nerve endings are non-encapsulated, always small unmyelinated fibers, responsible for many types of nociception, as well as itching and tickling.

non-hairy skin: palms and soles of feet

The Dorsal Column Medial Lemniscal Pathway

OBJ. # 2

The sensory modalities conveyed by this system are:

- Discriminative touch
- Vibration
- Proprioception

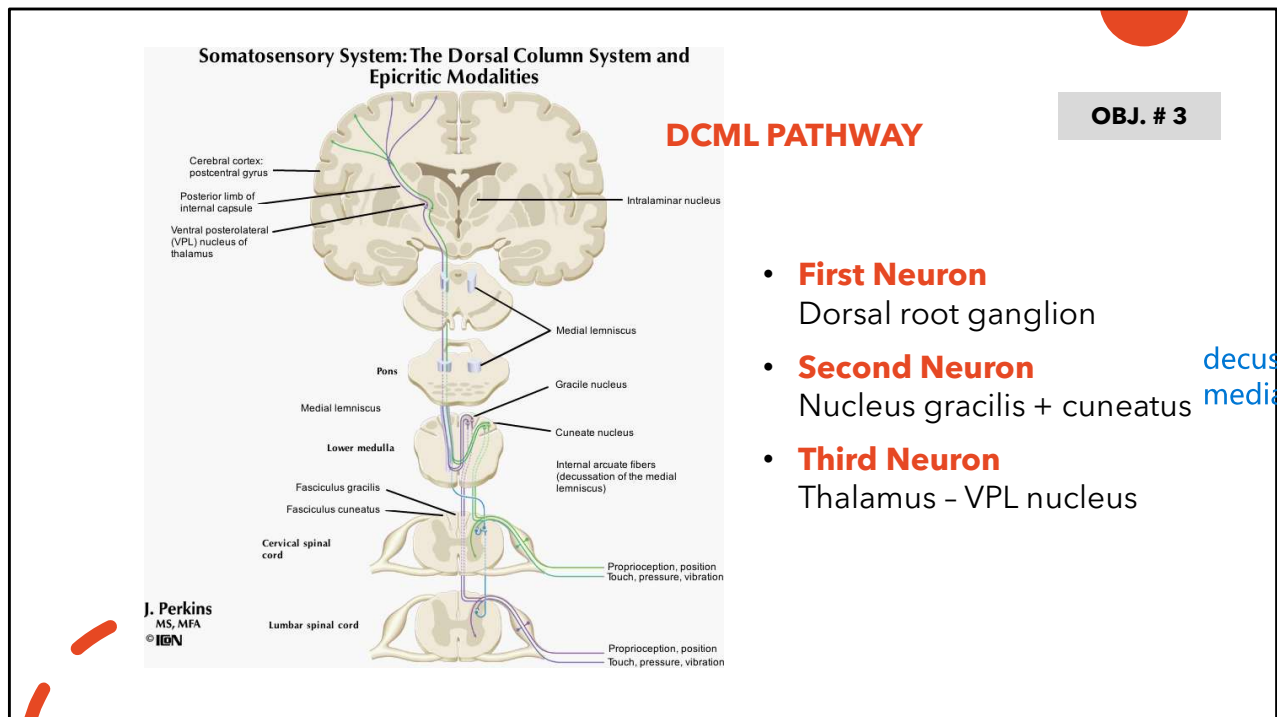
} **FROM THE BODY**

The peripheral receptors involved in this system are:

- Mechanoreceptors - Skin
- Proprioceptors - Muscles and tendons



Proprioception is the sense of body-space position



Here's the DCML pathway in it's entirety

decussation in lower medulla

The Antero-lateral System

OBJ. # 4

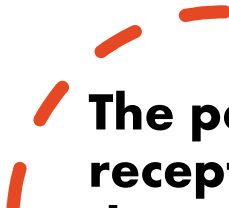
The sensory modalities conveyed by this system are:

- Non discriminative touch
- Thermal sensation
- Nociception - Pain

FROM THE BODY

- The spinothalamic tract
- The spinoreticular tract
- The spinomesencephalic tract
- The spinohypothalamic tract

ALS



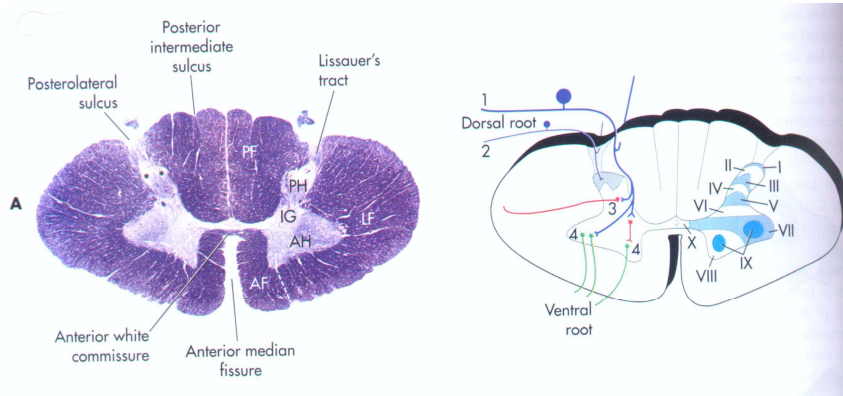
The peripheral receptors of the ALS system

OBJ. # 4

- Most of the peripheral receptors are non-encapsulated terminal axon branches - **free nerve endings**
- Free nerve endings are classified as: **mechanoreceptors, thermoreceptors, chemoreceptors and polymodal nociceptors**
- 2 types of afferent fibers are associated with the transmission of pain: **A-δ thinly myelinated and C fibers non-myelinated**
- Mechanoreceptors transmit innocuous sensation mostly through A- δ fibers
- Thermoreceptors transmit innocuous thermal sensation through A- δ and C fibers
- Nociceptors transmit through A- δ and C fibers

Spinal Cord Laminae Of Rexed

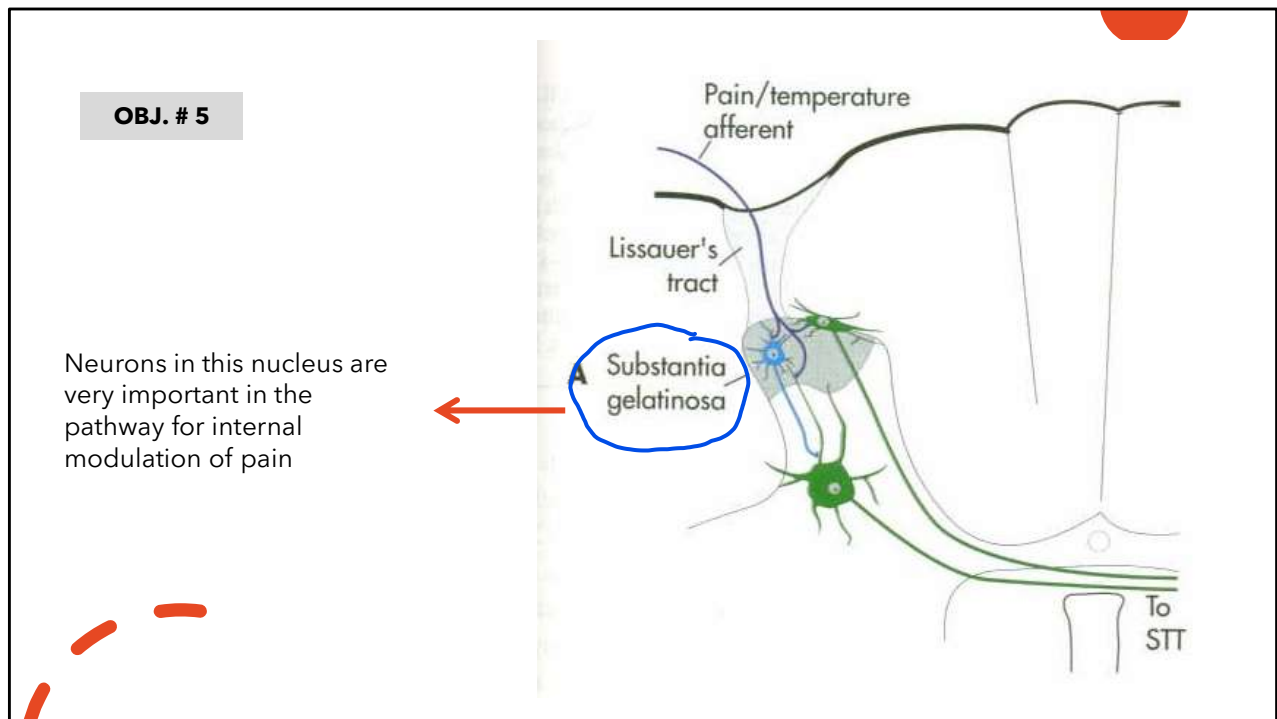
OBJ. # 5



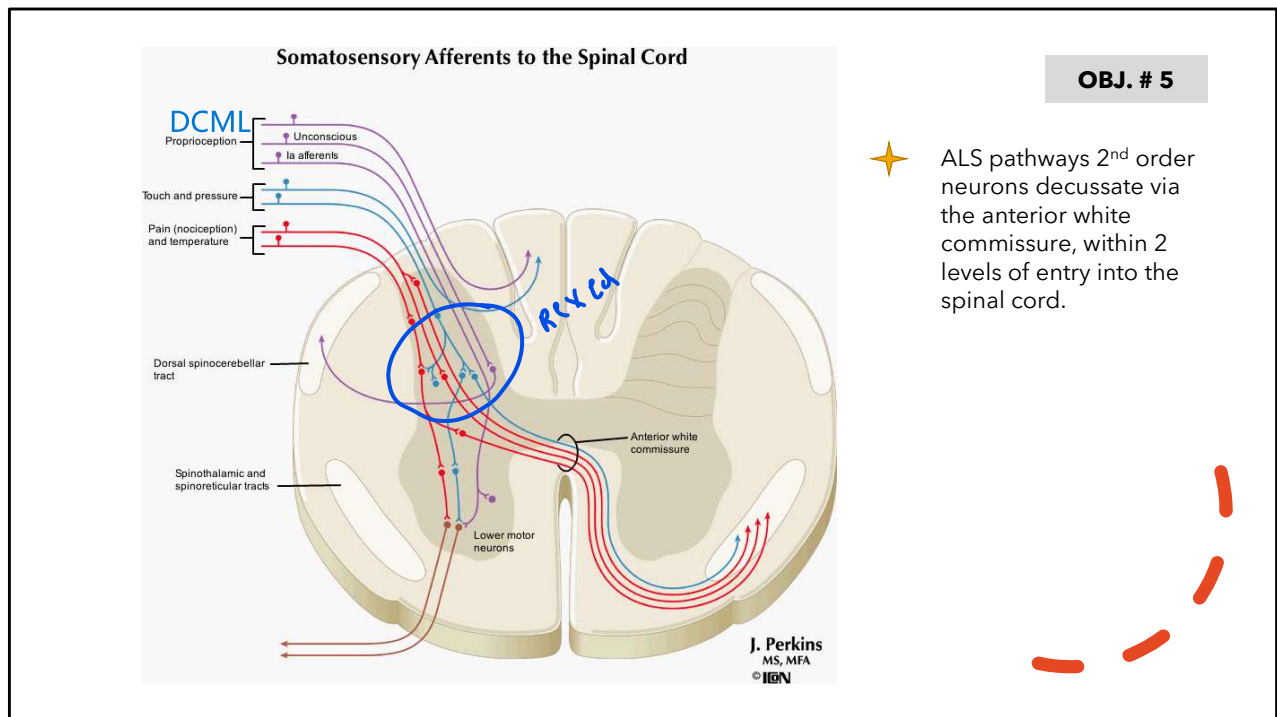
- Primary afferent fiber synapse in dorsal horn with **second order neuron**
- Fibers cross the midline in the anterior white commissure

Think of the laminae like nuclei that exist in a column the length of the spinal cord. This is the first synapse in the ALS sensory pathways and the location of the second order neuron cell bodies.

synapse almost immediately once enter spinal cord in rexed



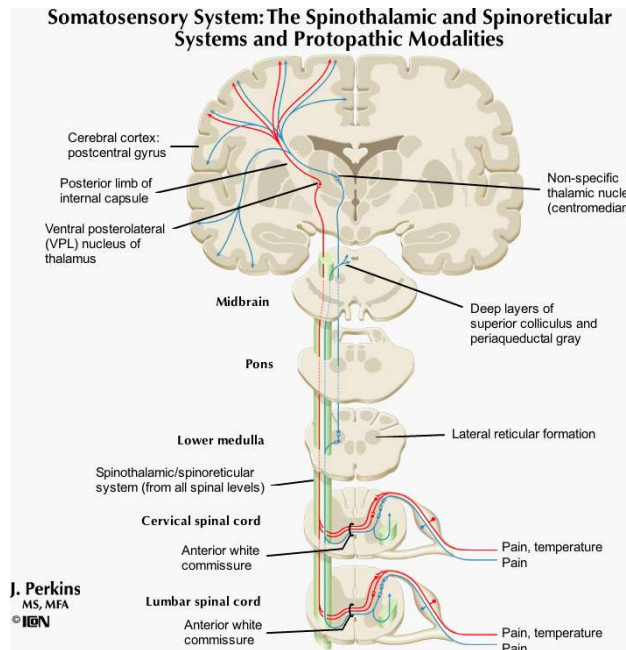
Some fibers need to ascend or descend a level to get to their 2nd order targets, this structure is called Lissauer's tract.
before synapsing



* ALS pathways 2nd order neurons decussate via the anterior white commissure, within 2 levels of entry into the spinal cord.

The ALS General Pathway

OBJ. # 5



In through the dorsal root, decussate in the anterior white commissure, travel up through the spinothalamic tract to the ventral posterolateral nucleus of the thalamus, then projecting to the cortex.

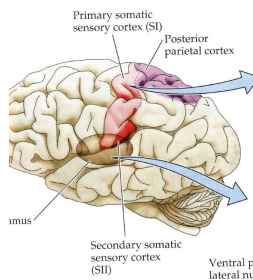
Another component of the ALS pathway is the spinoreticular pathway, which we won't be discussing in great detail. It is a 4 order pathway, which projects contralaterally to the reticular formation as well as the periaqueductal grey and superior colliculus. The reticular formation projects to the centromedian nuclei of the thalamus, which then project diffusely throughout the entire cortex. Felt to play a role in deeper types of pain and the emotional components of pain.

DCML and ALS project into VPL of thalamus

OBJ. # 6

Thalamocortical Fibers

Thalamo-cortical fibers from
both DCML and ALS systems
terminate in 3 cortical areas



Primary sensory cortex (SI)
Brodman's 3,2,1



Secondary sensory cortex (SII) some



Parietal association cortex,
Brodman's 5 and 7 some

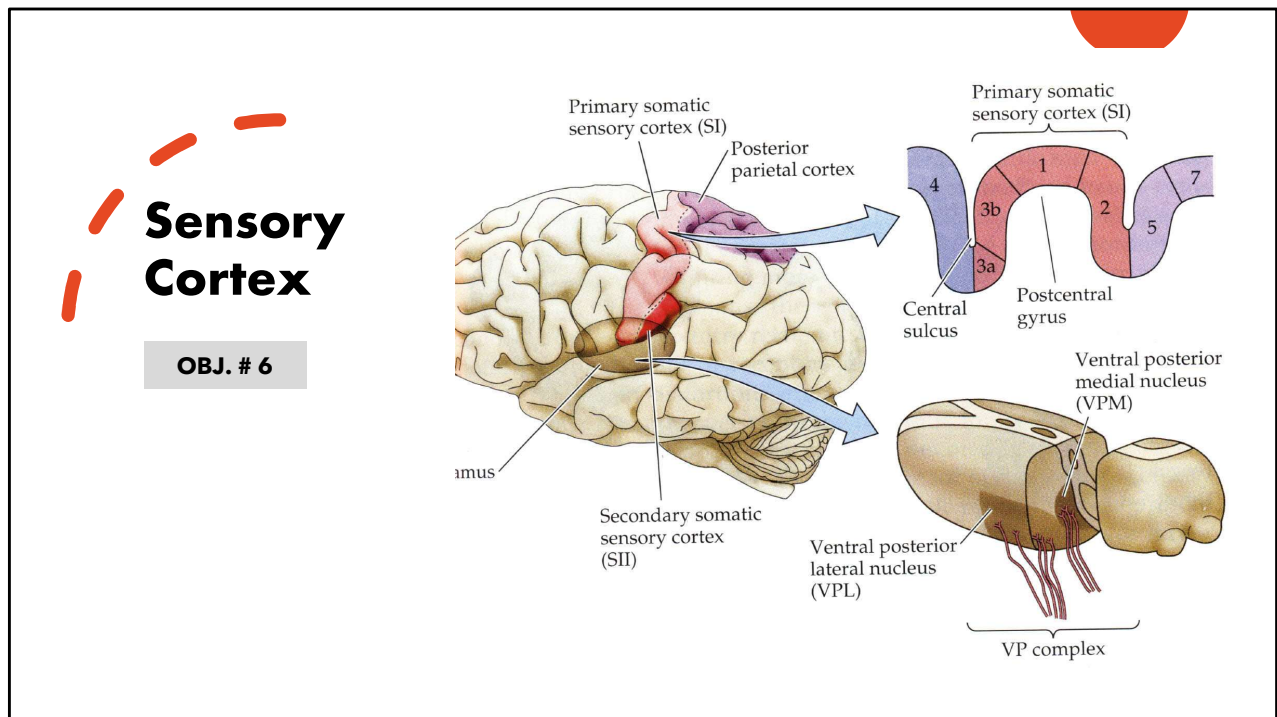


Diagram showing the somatosensory and posterior parietal cortices.

Thalamic fibers project mostly to S1, but some project to SII and to the parietal association cortex. The flow of information is primarily from S1->S2->association cortex

An association cortex refers to an area of cortex that processes information from multiple sensory systems. Also called multimodal cortex or heteromodal cortex.

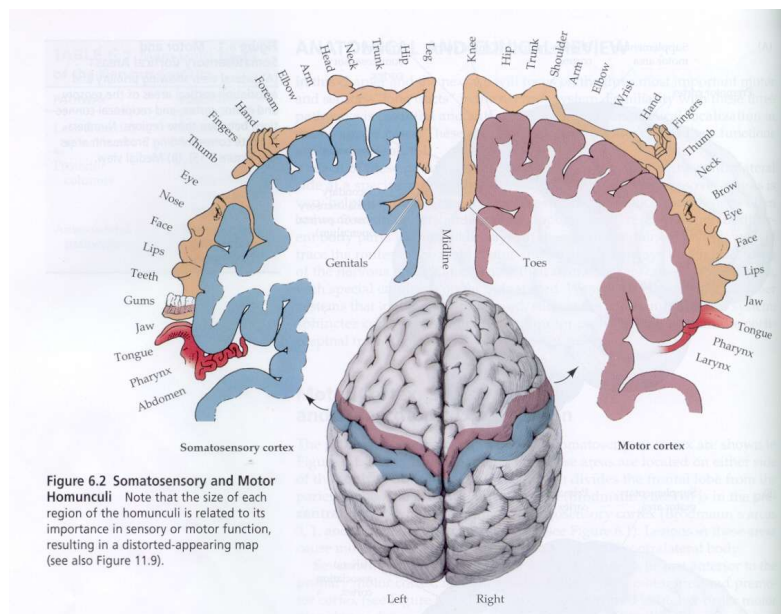
[not directly receiving outside information](#)

There is some division of labor within the primary sensory cortex. For instance the most anterior portions (3a) are involved in motoring of muscle tendon and joint stretch receptors. They project to the motor cortex for feedback of the control of muscle movement. Moving posteriorly, the neurons respond to progressively more complex types of sensory stimuli with the most posterior neurons only responding to stimuli moving in particular directions.

[particular type of touch rather than presence of touch](#)

OBJ. # 6

Sensory Cortex Somatotopic Organization



Quick review of the somatotopic organization that we spoke about in the previous lecture. Now we can pay more attention to the somatosensory cortex.



Clinical Correlations

OBJ. # 7

- Damage to DCML pathways, along their ascending trajectory **above decussation**, produces:
 - loss of touch, vibration, and proprioception from the body **contralateral** to the side of the lesion
- Damage to Dorsal Column pathways, in the **spinal cord**, produces the same deficits as above, but on the **same side** of the lesion
- **Positive phenomena:**
 - Paresthesias: tingling, prickling, crawling, burning sensations
 - hyperesthesia: exaggeration of any sensory modality

Now let's talk about what these systems mean for a patient.

*always know the point of decussation, it is the key to answering questions about clinical deficits.

For instance, if you know that the DCML pathways decussate in the medulla, you will know that a lesion anywhere above the medulla involving the DCML pathway will result in contralateral sensory loss to DCML modalities.

decussation is lower medulla



Clinical Correlations

OBJ. # 7

- Damage to the **somatosensory cortex** produces:
- Discreet loss of touch, vibration, and proprioception **contralateral** to the side of the lesion. However, a patient can sometimes crudely localize the sensations on a particular hand or leg.
- With cortical damage the patient can lose more sophisticated abilities such as:
 - The ability to judge weights of objects *have patient describe something in their hand*
 - The ability to judge shapes or forms of objects - **astereognosis**
 - The ability to judge texture of materials
- A lesion in the somatosensory association areas (5 & 7) produce:
 - Loss of the ability to recognize objects and forms felt on the opposite side of the body
 - Loss of most of the perception of the patient's own body or body parts on the opposite side - **neglect** or **extinction**

*patient's fully ignoring a side or side of world
may only respond to one side when testing sensation*

Clinical Correlations

OBJ. # 8

decussates as it enters or 1 above or 1 below

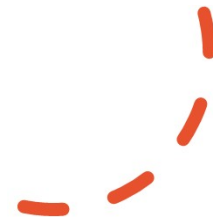
- Damage to the ALS pathways along their ascending trajectory **above decussation**, produces:
 - Loss of thermal, deep touch and pain sensation from the body **contralateral** to the side of the lesion
- Damage to the ALS pathways in the spinal cord produces:
 - loss of thermal, deep touch and pain sensation from the body **contralateral** to the side of the lesion below the lesion level
- Positive phenomenon: **PAIN**
- SENSORY LEVEL = When there is a spinal cord lesion, the sensory deficit level (dermatome) found on the patient's body is helpful to localize the spinal cord **level of damage**

low c- spine lesion = contralateral deficits below the lesion but ipsi at or around lesion

Clinical Correlations - Cortical Processing

OBJ. # 8

- With cortical damage, pain is not affected in intensity or quality, but it becomes poorly localized. [patient less able to express where pain is originating](#)
- The cortical perception of pain involves several cortical areas.
- Beyond the somatosensory cortex, the insular cortex and cingulate gyrus have a predominant role.
- The insular cortex may be a place where the sensory, affective, and cognitive components of pain are integrated.



Pathophysiology of PAIN

OBJ. # 9

- Positive symptom of ALS pathways: **PAIN**
- The International Association for the Study of Pain (IASP) defines pain as “an unpleasant sensory and emotional experience which is primarily associated with tissue damage or described in terms of such damage or both”
- **FUNCTION OF PAIN:** Alert mechanism to signal that something is wrong. Pain is a perception constructed by the brain and it is not always based on an observable injury
- Pain is produced by tissue injury, inflammation or necrosis
- The injury could be mechanical, chemical or thermal (cold / heat)
- Persistent pain could either be **nociceptive** or **neuropathic** pain
- **Central pain** is produced by damage to central pathways, the spinal cord or the thalamic nuclei involved in pain processing

hemi-body pain from damage to thalamus

pain = unpleasant sensory or emotional experience

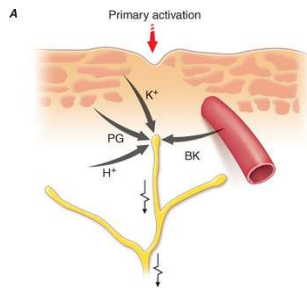
Pathophysiology of PAIN

OBJ. # 9

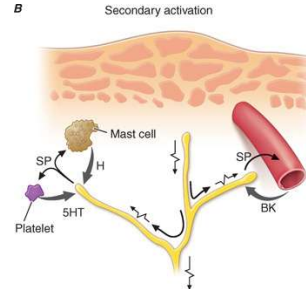
- Injured cells release intracellular components into the extracellular matrix, amongst them K⁺ which depolarizes nociceptors
- Other chemical mediators are also released: hydrogen ions, prostaglandins, leukotrienes, histamine, serotonin, and bradykinin
- These chemical mediators trigger an inflammatory process (See picture in next slide)
- Pain is then perpetuated by inflammation and vasodilatation
- Sensitization of pain receptors leads to **hyperalgesia and allodynia**
- Pain can also be also **referred** from visceral structures to the body surface



extreme sensitivity to pain and sensation of pain when otherwise non-painful stimulus is given (exaggerated pain)



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: Harrison's Principles of Internal Medicine, 18th Edition: www.accessmedicine.com
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OBJ. # 9

Events leading to activation, sensitization, and spread of sensitization of primary afferent nociceptor terminals.

- A. Direct activation by intense pressure and consequent cell damage. Cell damage induces lower pH (H^+) and leads to release of potassium (K^+) and to synthesis of prostaglandins (PG) and bradykinin (BK). Prostaglandins increase the sensitivity of the terminal to bradykinin and other pain-producing substances.

- B. Secondary activation. Impulses generated in the stimulated terminal propagate not only to the spinal cord but also into other terminal branches where they induce the release of peptides, including substance P (SP). Substance P causes vasodilation and neurogenic edema with further accumulation of bradykinin (BK). Substance P also causes the release of histamine (H) from mast cells and serotonin (5HT) from platelets.

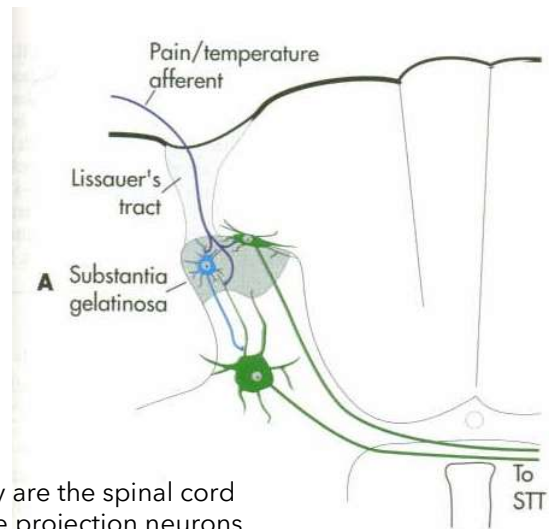
OBJ. # 10

Modulation Of Pain Perception

The body possess intrinsic mechanisms to modulate the transmission of pain.

- The **Descending Pain Modulating Pathway**

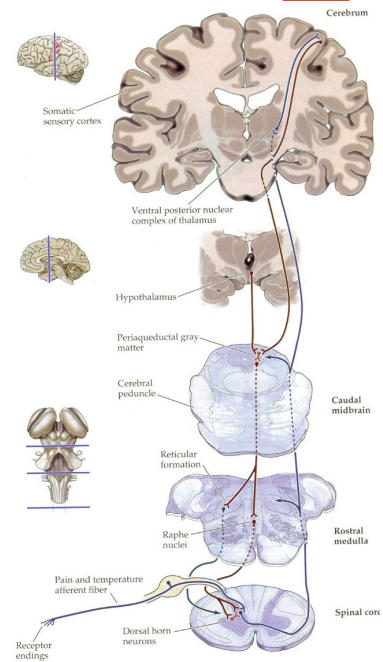
The targets of the descending pain pathway are the spinal cord neurons in the substantia gelatinosa and the projection neurons that transmit pain through the spinothalamic tract

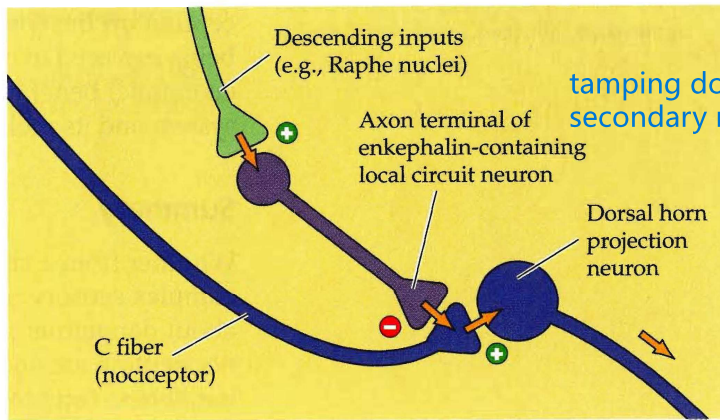
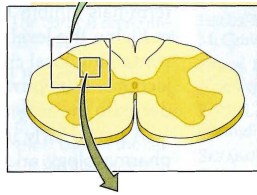


Modulation Of Pain Perception

OBJ. # 10

- Modulation in the spinal cord through descending modulatory fibers using different neurotransmitters
- Endogenous opioid peptides:
 - Enkephalins
 - Endorphins
 - Dynorphins
- Norepinephrine
- Serotonin
- Glycine
- GABA





tamping down response of
secondary neuron

Descending Pain Modulation Pathway