Lecture 4:
Somatosensation: Touch & Pain
Osher Series

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Lecture Outline

1) Somatosensation
   - sensory receptors in the skin (joints/muscles)
   - segments of the spinal cord
   - somatosensory cortex in the brain

2) Phantom limbs

3) Pain

4) IF THERE’S TIME -> Vestibular System
THE SENSES

Electromagnetic Sense
(1) Vision (Spring 2011……. Remember?)

The Mechanical Senses
(2) Audition (Spring 2011….. Remember?)
(3) Somatosensation (TODAY)
(6?) Vestibular Sensation (Maybe today)

Chemical Senses: Molecules that Enter Your Body
(4) Olfaction (Smell)
(5) Gustation (Taste)
SOMATOSENSATION: sensation of the body/skin

Sensory Neuron (i.e., “Receptor”) Types

1) **Tactile**: response to being touched ("light" and "deep" touch)
   - **Ruffini ending**, **Meissner’s corpuscle**, **Pacinian corpuscle**
   - *The axons from these receptors are myelinated!*

2) **Pain**: response to noxious stimulus

3) **Temperature**: response to cold/hot
   - *The axons for these neurons are thinly myelinated or not myelinated!*
   - *That’s why pain comes later than touch!*

4) **Proprioception**: response to position or mechanical movement of muscle/joint
   - “Golgi Tendon Organ”

For #1 and #4: Mechanical pressure bends/stretches the neural membrane, opens Na+ channels -> depolarization -> ACTION POTENTIAL.
Somatosensory Connections

SOMATOSENS: Sensory Neurons in Skin $\rightarrow$ Spinal Cord $\rightarrow$ Brain

(MOTOR SYSTEM: Brain $\rightarrow$ Spinal Cord $\rightarrow$ Motor Neurons $\rightarrow$ Muscles)

Spinal Cord Segment

Pain Reflex Arc

(more later)
31 Spinal Cord segments

**Dermatome:** skin area that provides input to a single **Sensory Nerve** (1 sensory nerve = 1 DRG = 1 spinal cord segment)

What about Motor Nerves? **Myotomes**
For this course, don’t worry about the different pathways to the brain for the different types of sensory neurons, although I will show the pain pathways.

Sensory Neurons in Skin -> Spinal Cord -> Brain
(Somatosensory Cortex)

Sensory “Homunculus”

(a) Somatosensory cortex
What Explains Phantom Limbs?

Two requirements

1) The area of cortex that originally received input from the lost body part (e.g., the \textbf{hand}) is still \textit{intact}.

2) Activity in that area still "represents" that part of body.

If you can \textsc{activate} that area of cortex $\rightarrow$ phantom limb

So, \textsc{how} can it be activated?

\begin{itemize}
\item a) spontaneous activity
\item b) cut nerves from the \textsc{hand} still function, but now are stimulated by the wrist (i.e., nerve endings are now in wrist)
\item c) cortical "hand" area gets taken over by inputs from "face"
\end{itemize}

\textit{(next slide)}
c) cortical “hand” area gets taken over by inputs from “face” (Ramachandran et al, UCSD)

I like this “Homunculus” better!
Invoked by harmful stimulus: cut, chemical irritation, intense heat or cold

3 Levels of Pain (keep in mind for drugs that alleviate “pain”):
1) *Sensation* of Pain (mediated by Sensory Neurons)
2) *Perception* of Pain (“unpleasant” vs. “neutral” vs. “pleasant”)
3) *Response* to Pain
   (distracters could mask the response: *adrenaline* and *ice cream*)
PAIN: Substance P
Substance P is the Neurotransmitter used by “pain” sensory neurons. Released onto neurons in the Spinal Cord and Brain!

** But, where is the PAIN PERCEIVED?!!?

Capsaicin: found in Jalapeño peppers promotes release of Substance P from tongue through CN #5 to brain
* And used in topical ointments
Body’s Own “Analgesic” Response to Pain

Gate Theory (Wall, 1965): mechanism that *inhibits* pain

.... *continuous pain is unnecessary*

**Endorphins (peptide NTs)**

(one type is called “Enkephalin”):

Attaches to endorphin (opiate) receptors on axon terminals of pain afferents, which inhibits or limits *release* of Substance P (limits “sensation” of pain)

Mediated by descending projections from the brain to the *Spinal Cord*.

**NOTE:** “Endorphin receptor” (“Opiate Receptor”) discovered because *morphine* (a type of opiate) binds to it (we’ll come back to morphine soon).

“Endorphin” means *endogenous* morphine
ENDORPHINS are also released (IN THE BRAIN) in response to **pleasant stimuli**

- **Acupuncture:** “Euphoria”
  (and/or relief from pain, if using it to relieve pain)

- **Running:** “Runner’s High”

**NALOXONE:** Endorphin Receptor Antagonist
Analgesic DRUGS

Imagine that you cut your leg. What causes the pain to last? (despite endorphin “gating”!)

- Injury produces **Prostaglandins** (PG) and **Inflammation** (part of healing/clotting process)……

PGs and Inflammation increase the sensitivity of Pain Sensory Neurons, allowing them to continue to respond

2 places of drug action:
1) Works “directly” at the site of injury

*Aspirin, Ibuprofen:* Anti-inflammatory and inhibit formation of PGs

*Tylenol (Acetaminophen):* Mechanism largely unknown

*Topical Drugs that contain Capsaicin:* Deplete Substance P
2) Works “indirectly” in the CNS (not the site of injury)

*Opiates*: agonist for *endorphin receptors*

  e.g., Morphine, Heroine, Demerol (cross BBB)

Change:
(1) “Sensation”  
  (inhibit substance P release from sensory neurons in spinal cord)

(2) “Perception” (euphoria)

Opiates help for dull, but not sharp, pain!
Vestibular System (balance system)

THREE fluid-filled semicircular canals in each **Inner Ear** (for 3 planes)

And the fluid-filled **Saccule and Utricle**

As head turns, otoliths in the fluid bend cilia on **hair cells** in these structures

Hair cells project to Vestibular Nerve Cells ….

…whose axons project (as part of the 8th cranial nerve) to the **Vestibular Nucleus** in the brain stem
Vestibular Ocular Reflex (VOR)

..... No need to know this figure in detail

Sometimes you need to *suppress* your VOR
When that fails -> Sea Sickness
Did mother nature intend for this?