Lecture 12 (Feb 14th):
VESTIBULAR & SOMATOSENSATION
Lecture Outline

1) Vestibular System

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

3) Phantom limbs

4) Pain (may not get through all of this)

Don’t need to know Chemical Senses (Smell & Taste)
THE SENSES

Electromagnetic Sense
(1) Vision √

The Mechanical Senses
(2) Audition √
(3) Somatosensation (TODAY)
(6?) Vestibular Sensation (TODAY)

Chemical Senses: Molecules that Enter Your Body
(4) Olfaction (Smell)
(5) Gustation (Taste)

(7?) Interoception (THE NEXT HOT TOPIC)
Hunger, thirst, fatigue, sexual arousal, temperature, pain, muscle tension, muscle soreness, itch, sensual touch, and perhaps, feelings of acceptance (Craig, 2015).
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 (and hair cells get depolarized)

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

**NOTE:** The 8th cranial nerve ALSO contains axons from the *auditory nerve cells*, which receive from the hair cells on the basilar membrane and project to the Cochlear Nucleus in the brainstem.
Vestibular Ocular Reflex (VOR)

….. No need to know this figure (but just appreciate how cool all this is!)

Sometimes you need to suppress your VOR

When that fails -> Sea Sickness
SOMATOSENSATION: sensation of the body/skin

Sensory Neuron (or “Sensory 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 from these receptors 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
(These Na+ channels are STIMULUS-gated.)
Somatosensory Connections

Sensory Neurons/Receptors in Skin (axons travel together as the “Sensory Nerve”) -> Spinal Cord -> Brain

……MOTOR SYSTEM: Brain -> Spinal Cord -> Motor Neurons (axons travel together as the “Motor Nerve”) -> Muscles

Spinal Cord Segment
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 mention the pain pathways later today.
What Explains Phantom Limbs?

Two requirements

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

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

If you can ACTIVATE that area of cortex -> phantom limb

So, HOW can it be activated?

a) spontaneous activity
b) cut nerves from the HAND still function, but now are stimulated by the wrist (i.e., nerve endings are now in wrist)
c) cortical “hand” area gets taken over by inputs from “face” (next slide)
c) cortical “hand” area gets taken over by inputs from “face” (Ramachandran et al, UCSD)

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

FUNCTION?

3 Levels of Pain (keep in mind for drugs that alleviate “pain”):
1) Sensation of Pain (mediated by Sensory Neurons)
2) Perception (Emotion) of Pain (“unpleasant” vs. “neutral” vs. “pleasant”)
3) Response to Pain
   (distracters could mask the response: adrenaline and ice cream)
I don’t expect you to know any of this in detail.
PAIN: Substance P

Substance P (and Glutamate) are the Neurotransmitters used by “pain” sensory neurons.

But, where is the PAIN PERCEIVED?!?

**Capsaicin**: found in Jalapeño peppers: promotes release of Substance P from sensory neurons of the tongue.
Body’s Own “Analgesic” Response to Pain

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

…. *continuous pain is unnecessary*

**Endorphins (peptide NTs)**

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
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 or endocannabinoids

*Tylenol (Acetaminophen)*: Mechanism largely unknown
2) Works on the pain “signal” in the Nervous System

A) *Topical Drugs that contain Capsaicin:*
   Deplete Substance P in sensory neurons

B) *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” (reappraisal, euphoria)