Study Guide – Physiological Psychology, 2200 – Exam 3
How the Brain Works (Part I) – Somatosensory, Hearing, Vision, Motor
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General Principles across sensory systems

Sensory transducers -- physically altered by their stimulus [for example, physical vibration (pacinian corpuscle), sound wave vibration (cochlear hair cell), light (photoreceptor)] in a manner that translates external energy into a neural signal via altering ion flow. Thus is true for all sensory receptors – whether receptors are specialized neurons (SM system) or specialized cells (vision (photoreceptor), hearing (hair cell), smell (hair cell)).

Transduction and action potentials:
For touch/pain & proprioceptive (deep tissue) receptors, the receptor is part of the sensory neuron (a special modification in the dendritic ending), and can lead to action potentials directly in the sensory neuron (whose cell body (soma) is in the dorsal spinal root) as follows:

1) Touch/pain/proprorception input --> somatosensory neuron --> spinal root/spinal cord --> VPN --> S1.

For smell, (taste), hearing, and vision, the sensory receptors produce graded potentials only. For smell, taste, hearing, receptors depolarize (become more +). For vision, receptors hyperpolarize (become more -). These receptor changes can stimulate an action potential in an associated (secondary) neuron as follows:

2) Smell/molecule – olfactory hair cell --> mitral neuron --> olfactory cranial nerve --> olfactory cortex (bypasses thalamus);

3) Sound/air wave – cochlear hair cell --> spiral neuron --> auditory nerve/vestibulocochlear cranial nerve --> MGN --> A1;


Thalamus -- a major sensory way-station to the cortex. Somatosensory (touch) input projects through the ventro-posterior nucleus (VPN; somatosensory nucleus); [note -- this nucleus also relays gustatory (taste) info from the tongue, though we did not cover this system]; auditory information projects through the medial geniculate nucleus (MGN; auditory nucleus); visual information projects through the lateral geniculate nucleus (LGN; visual nucleus). Motor feedback pathways pass from proprioceptors to cerebellum and basal ganglia to the ventro-anterior nucleus (VAN, motor), back to motor cortex. There is no olfactory (smell) input thru thalamus -- probably because the system is older and primitive.

Crossing -- ascending sensory pathways are generally crossed (from one side of the body to the other side of the brain, or “contralateral” projections), but level of crossing varies. Somatosensory path crosses after ascending to the brain, while pain path crosses immediately on entry into the spinal cord. Percent of crossed fibers also varies (right visual field projects exclusively to left hemisphere, while auditory fibers are about 2/3 contralateral, 1/3 ipsilateral). Crossing explains why left-hemisphere dominant people are right handed (motor pathways cross, too).
Distinguish topographic (touch), tonotopic (sounds) and retinotopic (vision) patterns of cortical organization.

**Touch/proprioception & Pain – Somatosensory system**

*Pain and temp* – transduced by 3 types of free nerve endings (2 have unmyelinated fibers, 1 is myelinated).

*Nociceptor* = pain receptor (noxious receptor).

*Touch* – transduced by 4 types of receptors. **Pacinian corpuscle** (vibration, large receptive field, fast adapting); **Ruffini’s endings** (stretch, large border, slow adapting); **Meissner corpuscle** (touch, small receptive field, fast adapting); **Merkel’s disks** (touch, small receptive field, slow adapting).

Cutaneous (skin) stimulus intensity is coded via *differential receptor thresholds*, which means as the intensity of skin stimulation *increases* (temp, pressure, pain), *more* receptors are depolarized. (This principle holds for *all* sensory systems).

Know the **somatosensory pathway** (skin receptor, projects up dorsal (spinal) column, to the somatosensory thalamic nucleus (ventro-posterior nucleus; VPN), and then primary somatosensory cortex (SM -1)). This path *crosses midline* just before passing through the thalamus.

Understand **topographic** (point to point) mapping in somatosensory cortex.
Understand that representation in somatosensory cortex is proportional to sensory receptor populations thus highly enervated areas (fingers, lips) have greater representation in SM cortex (think of the “homunculus” with enlarged hands and lips).

**SM cortical plasticity** – you can increase cortical representation via stimulation/training, decrease representation with deprivation or loss of digit/hand etc.

Understand basic concept of laminar/columnar organization in SM cortex (in fact, all of sensory cortex). Lamina are formed early by migrating neurons (which stop in layers).

Know basic ascending pain pathway (free nerve endings (receptors), project to anterolateral pathway (crosses midline) in spinal cord, ascends front of spinal column (unlike regular SM), reticular formation, periaqueductal grey, and different target regions of cortex (frontal, cingulate, primary somatosensory)).

Understand different perceptual representations of pain – cognitive (frontal), emotional (cingulate), location (SM cortex).

In response to pain, in the descending pain pathway, periaqueductal grey releases opiates, which causes descending neurons of the raphe nucleus to inhibit incoming pain signals via serotonergic inhibition. This is different from periphery, where serotonin is an excitatory pain neurotransmitter.

**Olfactory (smell) system**

Olfactory transduction takes place at the olfactory hair cell, when an olfactory receptor is bound by a matching odor molecule (ligand), hence depolarizing the hair cell, and exciting the mitral neuron to fire.

Olfactory bulb (mitral neuron axons) project to amygdala and hippocampus (parts of limbic system) which is one reason smells can trigger strong memories.
Hearing/Auditory system

**Divisions of the ear** -- outer [ear & ear-drum (tympanic membrane)]; middle [bones & air]; inner [cochlea]. The outer ear can be reached by Q-tip. The middle ear is behind the ear-drum, and connects to sinus (thus can become infected). The inner ear contains fluid.

**Parts of the cochlea (inner ear)** -- oval window, perilymph, basilar & tectorial membranes, organ of corti (the whole part of inner ear that does the transducing and makes up most of the cochlea), hair cells (inner & outer), spiral neurons.

Understand that the vibrating *tympanic membrane* transfers acoustic signal to the bony structures, which transfer vibration to the oval window of the cochlea. The role of the bony structures is to *focus/intensify vibrations* so they can be transmitted from air (easy to vibrate) to fluid (harder to vibrate). The perilymph fluid waves created *inside* the cochlea result in sound transduction.

*Transduction at the hair cell* – hair cell stereocilia bend due to vibrations in the *basilar* membrane (which vibrates with the perilymph), while *tectorial* membrane stays still. **Bending causes depolarization of the hair cell** (open K+ ion channels), excitation causes *associated spiral neuron to fire*.

**Tonotopy** -- the basilar membrane is organized so that the base vibrates preferentially to *high* frequencies, and the tip (apex) to *low* frequencies. The hair cells for each region thus have a preferred or characteristic frequency (CF), corresponding to their *location* (and so does their spiral neuron). This *tonotopic* pattern is evident elsewhere in the auditory system including MGN (medial geniculate nucleus of thalamus) and primary auditory cortex.

**Type I spiral neurons** contact just 1 *inner* hair cell; **type II spiral neurons** contact lots of *outer* hair cells. Therefore Type I spiral neurons have a tight “tuning curve” that matches the preferred frequency (or characteristic frequency) of their hair cell.

**Encoding frequency** – tonotopy (see above; frequency bands in MGN and A1); phase-locking (multiple neurons firing in concert (volley-principle)).

**Encoding amplitude** -- firing rate (increase intensity, increase firing); spatial spread (louder sounds activate a more distributed network of cochlear hair cells).
**Auditory pathway structures** -- cochlea, cochlear nucleus, olivary nucleus, inferior colliculus, medial geniculate nucleus (MGN), A1 (primary auditory cortex), A2 (secondary auditory cortex, including Wernicke’s speech area in left A2).

**Wernicke’s area** for speech perception is in secondary auditory cortex; connects to Broca’s in frontal/motor cortex (speech production).

If an individual is deaf from early in life and uses sign language to communicate, the **auditory cortex will re-organize** to respond to visual information. In particular, this is seen with activation of Wernicke’s area in secondary cortex by ASL (or other sign language).

Based on evidence of developmental “critical periods,” coupled with the plasticity leading to re-organization (above), **cochlear implants** are useful mainly for children with congenital deafness, or adults who were deafened after learning to speak. Although experimental surgery has led to some cochlear implants in congenitally deaf adults, the evidence suggests that sudden auditory input after a lifetime in its absence is difficult/impossible to process.

**Hearing aids** can assist people with “residual hearing” (hard of hearing), by boosting the power of acoustic signals (can overcome loss of flexibility in ear-drum or middle ear from age, etc.)

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**Vision – Visual system**

Light **frequency** in visible spectrum = **color**.

Know **major parts of eye** – pupil, lens, retina, fovea, optic nerve. The lenses are controlled by muscles to focus images on the back of the retina. The **blind spot** is where the optic nerve passes through the retina (our brain “fills in” this perceptual hole for us).

Visual accuracy is called “**acuity.**” Poor acuity **usually** means the lens is not working properly and glasses are needed (far-sighted or near-sighted). Retinopathies and cataracts can also impair vision.

**Retina** is composed of photoreceptors (rods and cones), bi-polar cells and ganglion cells. The photoreceptors are actually in back so that light passes through ganglion and bi-polar layers to reach them (inverted design).

Just as spiral neurons in the **auditory** system send their axons into the auditory (cranial sensory) nerve, **ganglion cells** in the retina send their axons into the **optic (cranial sensory) nerve.** (The optic nerve is actually a bundle of ganglion cell axons).
Know the mechanics of **phototransduction** – light photon hits opsin molecule, conformation change results in reduced cGMP, this closes Na+/Ca++ ion channels, and hyperpolarizes photoreceptor. Hyperpolarization reduces transmitter output to bi-polar cell. (Note the visual transducer is the only one to **hyperpolarize** in response to stimulation, while other sensory receptors **depolarize** – we do not know why). Hyperpolarization gets translated into depolarization by the bipolar cell – when transmission from photoreceptor decreases, it stimulates the ganglion neuron into a possible action potential. 

Thus, photoreceptors (rods/cones) and bi-polar cells use **graded potentials**, and the bipolar cell reverses the hyperpolarization of the photoreceptor to depolarization. The ganglion cell is the first to send an **action potential** when activated by visual stimulus.

**Rods** -- in the retinal periphery, use rhodopsin, encode blue/green light (generally perceived in grey tones), are low acuity, and are used for night vision.
**Cones** -- concentrated in the fovea (which is why acuity is very high there), use 3 different opsins, and prefer 3 different light wavelengths (blue, green, red).

**Color blind** people are missing 1 or more kind of cone, and can only process light wavelengths with the cones they have. Some light frequencies are hard for them to discriminate. More frequent in men because it is X-linked.

Visual detail is also characterized by **frequency** (another visual meaning for this word), which represents the **level** of detail [think “high frequency = high definition TV (lots of info per inch),” while “low frequency” = simple, low-detail].

Details are also defined by **contrast** (black/ white = high contrast, shades of grey = low contrast).

Primary visual cortex (V1) shows **retinotopy**, as well as **laminar** and **columnar** organization as seen in SM cortex and auditory cortex.

Structures of the visual system respond to increasingly complex bits of information as one moves upwards. So whereas photoreceptors are “on/off,” ganglion cells respond to “fields,” and LGN and V1 neurons start to respond to specific orientations, motion, colors, etc. This principle of shifting from “point to point” representation, to more of a “Gestalt” representation (objects, faces) is a common principle of sensory systems.

Individuals who are blind from an early age & use Braille to read, show **re-organization of visual cortex** for somatosensory (SM) information. Visual cortical reading areas (next to Wernicke) respond to visual text in the sighted, but to Braille (somatosensory) text in the congenitally blind.

The fact that vision restoration of congenitally blind individuals, as adults, results in only partial (sometime minimal) vision – particularly for processing complex visual stimuli – reflects the existence of a **critical period for visual cortical development in childhood**. (Remember Hubel, Weisel & the kitten).
Motor System (covered half-lecture)

Types of muscle fibers --
- **Striated muscle** – cardiac and skeletal, contracts and relaxes in short bursts.
- **Non-striated muscle** (smooth muscle) – enervates the gut and other organs, involuntary, more sustained contractions.

*Muscle contraction* – occurs due to stimulation at the **neuromuscular junction**, where excitatory *acetylcholine* leads to *changes in ion channels (potentials)*, and the “sliding” of actin/myosin protein molecules over each other to contract muscle.

Divisions of the motor system (movement control) –
- **Pyramidal motor system** controls all voluntary movement.
- **Extra-pyramidal system** is an involuntary “stabilizing” or motor feedback system, and operates through input/feedback to cerebellum and basal ganglia, which projects back to motor cortex through VAN (other inputs as well, see below).
- **Autonomic nervous system** (sympathetic, parasympathetic, enteric) controls smooth (involuntary) muscle (& also striated cardiac muscle).

**ALS (amyotrophic lateral sclerosis) or Lou Gehrigs disease** -- progressive neurodegenerative disease that affects motor neurons in the brain and spinal cord.

*Proprioception* – sensory feedback (part of the “touch” system) regarding posture and muscle status (tells the brain where our limbs are in space) – projects mainly to cerebellum and basal ganglia, (which feed back to motor cortex via VAN thalamic nucleus); some input through VPN to SM cortex.

Know the **pyramidal motor pathway** (motor cortex to spinal nerves), which controls voluntary muscles. This pathway is fairly *direct* (for speed), and includes some of the longest axons in the body.

Know the **extra-pyramidal motor pathway**, which modifies voluntary muscle (motor) output through feedback regulation, including:
1. SM input from VPN to SM to motor;
2. Proprioceptive input mainly to cerebellum and basal ganglia, then to VAN and back to motor;
3. Vestibular input through PLN to auditory cortex and cerebellum to motor;
4. Auditory and visual orienting through MGN and LGN to A1/V1 and back to motor.

*Reflexes* – specific motor impulses generated within the spinal horn to specific stimuli (don’t pass up to the brain). Short latency, probably for survival.