PSY 3031 Introduction to Sensation and Perception (sec 001) Spring 08

Topic outline

Approximate schedule of lectures is provided in syllabus. The order will not change, but the schedule is fluid and will adjust according to what we can cover in class.

Lecture topics and readings for the upcoming week are updated at the end of each week.
Lecture outlines are posted after the lecture is delivered.

Noteworthy (*look here for upcoming events and course announcements*):

NEW PLAN FOR FINAL EXAM.

No lecture last day of class. Review during 2nd half of class on May 6.
Final exam offered during last day of class, on **May 8**.
Exam also offered Saturday, **May 17**, 1:30pm.
60 multiple choice questions covering Lectures 18 - 27.
New plan also means NO make-up exam on May 9th.

Open me first: quick links for instructors / designers
Exam 1
Exam 2
Exam 3
Syllabus

1 Lecture 1: Introduction to the course

Reading: Goldstein, pp. 3 - 19.

- Sensation: **transduction** of physical stimulus by sensory neurons.
- Perception: interpretation of sensation. Perception is subjective and relative.
- Psychophysical methods for quantifying perception
  - Detection
    - Methods of **Limits**, Adjustment and **Constant Stimuli** (p. 13).
    - **Difference thresholds**. Weber-Fechner law: dS/S = k (p. 14).
  - **Magnitude estimation**. Steven's law: $P = kS^n$ (Fig. 1.12; p. 15 - 16).

Email:
moodle@umn.edu

- n < 1: compression
- n > 1: expansion

Lecture 2: Introduction to neural mechanisms
Reading: Goldstein, pp. 21 - 28.

- Basic properties of neurons
  - Main divisions: **dendrite**, **soma**, **axon**, **myelin** (Fig. 2.3)
  - **Membrane potential** = potential for communication (p. 25)
- Communication between neurons
  - **Action potentials** are created/carried by **ionic currents** (Fig. 2.5)
  - Information is encoded by the **rate** of action potentials (Fig. 2.6)
  - Information is chemically transmitted at **synapses** (Fig. 2.7)
- Nerves are bundles of axons
  - Sensory information travels in **cranial nerves** (the 12 nerves that enter the brainstem above the spinal cord) and **peripheral nerves**
  - **Conduction velocity** is highest in large, myelinated axons
    - Small, unmyelinated axons (C-fibers) transmit action potentials at ~1 m/s
    - The largest myelinated axons (A-alpha fibers) conduct at ~100 m/s.
- Anatomy of the nervous system(s)
  - **Central nervous system**: brain, spinal cord and retina.
    - in general cannot regenerate after injury, e.g. spinal cord injury results in paralysis
  - **Peripheral nervous system**: peripheral nerves & sensory neurons, cranial nerves.
    - generally can re-grow after injury (a cut in the finger heals, and has normal sensation).

ICE1

2 **Lecture 3: Touch**
Reading: Goldstein, pp. 305 - 318

- **Somatosensory** system: sensation throughout the body (p. 305)
  - Proprioception: balance and limb position
  - Cutaneous senses: Mechanical, Thermal, Noxious
- Mechanoreceptors: 4 primary types, we focus on 2 (Table 14.1)
  - **Merkel receptor**: slowly **adapting**, low frequency, perception of fine detail (Fig. 14.10)
    - Density is inversely related to receptive field size (Fig. 14.12)
  - **Pacinian corpuscle**: rapidly adapting, high-frequency, texture and vibration

    - Texture is perceived by a combination of Merkel and Pacinian receptors (pp 313-314; Fig. 14.15)
- **Thermal receptors**
    - Separate receptors for warm (38 - 48 deg C) and cool (20 - 35 deg. C)
        - For reference, body temperature is ~37 deg. C
    - **Non-uniformly distributed**: cold sensation may not occur on all parts of the back of your hand.
- Pathways: **spinothalamic** (thermal) and **dorsal column/medial lemniscal** (mechanical) (Fig. 14.5)
- **Thalamus**: all sensory information goes to thalamus before cortex
    - General principles (without memorizing details)
        - Somatosensory information is **somatotopically organized** like cortex
        - Different pathways segregated in **different nuclei** (sub-regions)
    - Response properties are more specialized, e.g. center-surround receptive field organization (Fig. 14.18)
- Cortical representation: **somatotopic** and non-uniform
    - Primary somatosensory cortex (S1) is on the **post-central gyrus** (Fig. 14.6)
    - Body parts with higher tactile acuity have **larger cortical representations**.
    - **Attention**. Unattended stimuli can fail to elicit neural response, even in primary somatosensory cortex (Fig. 14.21)
    - Object-selective responses in regions beyond S1, e.g. farther back in parietal cortex (Fig. 14.20)

Lecture 4: Pain

Reading: Goldstein, pp. 318 - 324

- Three types of pain
    - **Nociceptive**: mediated by cutaneous nerves responding to chemical insult or extreme heat, cold or force (Fig. 14.22a). Travel in a separate pathway from other sensory information (touch is in **ipsilateral** dorsal column - medial lemniscal path; nociception is in **contralateral** spinothalamic path, shown in Fig 14.5).
        - Ipsilateral means that the neural representation is on the side of the body on which stimulus transduction occurred.
        - Contralateral means that the neurons responding to a stimulus (or controlling an action) are on the opposite side of the body.
        - Types of nociceptors: chemical, thermal (very hot and very cold), mechanical (respond to very sharp things or very strong pressure) and **polymodal** (respond to both thermal and mechanical stimulation)
    - **Inflammatory**: mediated by prostaglandins generated in response to injury (Fig 14.22b). **Prostaglandins** are chemical messengers that initiate platelet aggregation or increased or decreased blood flow where appropriate; they also sensitize sensory neurons.
    - **Neuropathic**: caused by damage to or pressure on peripheral nerves or neurons in pain pathways (Fig. 14.22c). Examples:

        - **Carpal tunnel syndrome**: the carpal ligament puts pressure on the medial nerve, causing weakness, numbness and/or pain in the hand (particularly thumb through 4th finger).
        - **Sciatica**: pressure on the large sciatic nerve, where it goes through hip joints, causes burning or freezing pain in legs and buttocks.
- The importance of pain
    - Social cost: $100 billion / year in US; 20% of American adults report chronic pain (most commonly lower back pain).
    - Protective mechanism: lack of pain (e.g. CIPA) is life threatening.
- The experience of pain
    - **Nociception** is the sensory component; **pain** is is the affective (emotional) component
        - Fig. 14.24: decreasing intensity decreases unpleasantness, but unpleasantness can be decreased without decreasing intensity.
    - Pain pathways project to the **limbic** system (**amygdala**, hippocampus, anterior cingulate gyrus, Fig. 14.23) as well as somatosensory cortex.
- The treatment of pain
    - **Psychological factors**
        - Expectation: surgical patients who know what to expect and are told to relax experience less pain and recover more quickly (p 320).
        - Distraction: subjects looking at pleasant pictures can endure painfully cold stimuli longer than subjects looking at neutral or unpleasant pictures (Fig. 14.25).
        - Gating: although this model is still underdevelopment, Fig. 14.26 illustrates how sensory information can inhibit the pain response through T-cells in the substantia gelatinosa. Stimulation-produced analgesia illustrates this phenomennon.
    - Physiological treatments
        - **NSAIDs**. Non-steroidal anti-inflammatories inhibit the production of prostaglandins (the molecules that trigger inflammatory responses and potentiate nociception). Aspirin, Tylenol, ibuprofen (Advil) ...
        - **Opiods** and **cannabinoids**: when **endorphins** (endogenous opiods: chemicals similar to opium that the body naturally produces) dock at opiod receptors in the CNS, pain is inhibited.
            - Some opiod receptors/sites also induce pleasure.
            - Exogenous opiods (heroin, morphine) activate the same pleasure pathways and suppress pain.
            - Opiod blockers (naloxone) inhibit analgesic effects of endorphins (and are used to treat heroin addiction).
        - **Capsaicin**: a chemical produced by chile peppers that activates nociceptive neurons
            - Burning sensation, because the heat nociceptors are activated.
            - Role in nature: birds experience analgesia instead of pain when

consuming capsaicin. Mammalian intestinal tracts destroy chile seeds, while they pass through birds unharmed and are distributed.
- Applied after a topical analgesic, which blocks action potentials from traveling down sensory nerves, capsaicin overwhelms nociceptive response and causes thermal nociceptive neurons reduce the number of receptors and become less reactive. Capsaicin cream is used to treat neuropathic pain.

ICE2

Principles of Neural Science (Google books)

3 Lecture 5: Olfaction

Reading: Goldstein, pp. 330 - 338

- Olfactory sensory neurons
  - **Cilia** (Fig. 15.3) actually contact molecules
  - Receptors are embedded in membrane of the tips of the cilia (Fig. 15.4)
  - When the right molecule docks on the receptor, a signaling cascade starts in the cell, resulting in:
    - Opening of sodium channels
    - Intracellular calcium increases (which can be detected by calcium imaging, Box, pg. 332)
    - Initiation of action potential, which travels up (myelinated) axon through bone to **glomerulus** (Fig.15.4) in olfactory bulb on the base (ventral side) of the frontal lobe
  - Each neuron has one receptor type
    - There are **350 receptor types** (pg. 332)
    - **Each receptor responds to more than one molecule**, but with different strengths (Fig. 15.6)
    - **Each molecule activates more than one receptor**
    - Why different receptors respond to different molecules is not fully understood
- **Olfactory mucosa**
  - This is where olfactory neurons are located
  - Dime sized, at the top of the nasal cavity
  - Organized into Zones (Fig. 15.8)
    - Each zone contains many receptor types
    - Each receptor type is only in one zone
- **Olfactory bulb**
  - Receptors make their first synapse in clusters called **glomeruli** (singular: **glomerulus**)
  - There is an orderly arrangement of odorant responses (Fig. 15. 9)
    - Glomeruli responding to longer molecules are more anterior
    - Glomeruli responding to different functional groups are in different places

- Distributed code
- Olfactory cortex (Fig. 15.5)
  - Primary olfactory cortex is **piriform cortex**, on ventral aspect of temporal lobe
  - Secondary olfactory cortex is in **orbitofrontal cortex**

Lecture 6: Olfaction

Reading: Goldstein, pp. 327 - 330

- Predicting Scent
  - Unpredictable relationship between odorant molecular structure and individual receptor activation: can't predict pattern of receptor activation.
  - **Predictable relationship** between pattern of receptor activation, pattern of glomerular activation in olfactory bulb and odorant scent (Fig. 15.10).
- Quantifying Scent: the puzzle of olfactory quality (pgs. 330 - 331)
  - Insufficient vocabulary
  - Too many dimensions
- Experiencing Scent
  - Smell is hardwired into the limbic system
    - Smell is the ONLY sense that bypasses thalamus
    - Taste and smell offer important protective mechanisms
    - Emotional content of memory evoked by smell is stronger than same memory evoked by verbal cues
  - Recall of scent identities is generally poor (pg. 330)
  - People can improve sensitivity with practice (experience)
  - Subliminal scents (**pheromones**) mediate intra-species **aggression** and **attraction** in animals; probably humans.

ICE3

4 **Lecture 7: Taste & Flavor**

Reading: Goldstein, pp. 338 - 348, 367 - 368

- **Taste cells** are located in taste buds (saliva gets in through taste pore)
  - **Taste buds** are on **papillae**; papillae are on the tongue(pp. 339 - 340; Fig. 15.13)
    - There are 4 kinds of papillae, all of which have tactile and pain receptors.
    - The 3 kinds of papillae that have taste buds are circuvilliate, foliate and fungiform.
    - Filiform papillae give thetongue its hair-like appearance.
  - The **4 primary dimensions** of taste correspond to 4 taste receptors
    - ***Sweet*** and ***bitter*** are similar to odorant receptors in olfactory mucosa: Molecules dock on trans-membrane receptors, changing membrane potential and initiating action potential

        - ***Salty*** and ***sour*** are ionic sensors
        - The **5th aspect** of taste, ***umami,*** is detected by a receptor that responds to MSG
    - **Supertasters** (p. 341 - 342) are people with a genetic difference that means they have an extra kind of taste cell in their taste buds, one which signals a bitter sensation in response to **PROP** (6-n-propylthiouracil),and a higher density of fungiform papillae.
- Taste pathways
    - Tongue and mouth are enervated by four nerves (p. 339), in Cranial Nerves VII, IX & X
    - Cell bodies are located in **Nucleus of Solitary Tract** in the brainstem (Fig. 15.15)
    - Taste signals are relayed through **thalamus** to **frontal operculum** and **insular cortex** (primary taste area, Fig. 15.15).
- **Flavor** = Taste + Smell
- **Appetite** is more than taste & smell
    - Taste response (NST reaction) is regulated by signals such as blood sugar
    - Appetite is mediated by **orbitofrontal cortex**, which combines information from all senses (Fig. 15.22), as well as reward/desire (Fig. 15.23).
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Lecture 8: Lab day

- **Spinal reflexes** are behaviors initiated by sensation that do not require (involve) the brain
    - **Monosynaptic** or disynaptic connections in the spinal cord automatically connect **afferent** nerves to **efferent** neurons
    - These hard-wired pathways mean that one particular sensation *automatically* results in one particular action
    - Example: knee jerk.
        - Muscle spindle **proprioceptors** in the quadriceps detect that the muscle is stretching. Usually this means the leg is bending.
        - The afferent axon from the spindle proprioceptor is directly connected to the cell body of an efferent motor neuron.
        - An action potential is therefore automatically initiated in the motor neuron when the stretch receptor is activated.
        - The action potential in the efferent motor neuron axon contracts the quadriceps muscle.
- In-class demos
    - Spinal reflex
    - Cutaneous thermal receptor density
    - Taste test: chocolate
    - Tasting without the nose: juju candies and sour gummy bears
    - Supertasters
    - Braille and Tactile Acuity
    - Somatosensory adaptation (thermal)
    - Psychophysical methods: Method of Constant Stimuli and Method of Limits for

measuring Two-Point Discrimination thresholds on finger tip and forearm.

ICE4

Exam 1 Study Guide

5 Lecture 9: Balance and cue integration; exam review (2/19)

Reading: Goldstein, pp. 220 - 221

- Balance is maintained by the interaction of three sensory systems:
  - the vestibular system (see Study Guide for figures, information)
  - vision (see text for description of optic flow)
  - kinesthesis (sensation of the motions and positions of the limbs and body).
- The Vestibular System
  - The inner ear comprises the cochlea, the **saccule**, the **utricle** and the **semicircular canals**
    - Hair cells in the **maculae** of the saccule and utricle have their tips embedded in the **otolith** organ, which shifts when the head undergoes linear acceleration
    - Hair cells in the **ampullae** of the semicircular canals have their tips embedded in the **cupula**, which flexes when the head undergoes angular acceleration
  - Bending the tips of hair cells causes membrane potential change
    - Depending on the direction, deflection can generate both increases and decreases in action potential rates in sensory neurons
  - Central pathways: brainstem nuclei process vestibular information and generate motor responses
- The Visual System and Balance
  - **Optic flow** is the pattern of image motion received by the eye by stimuli in the environment when an observer moves relative to the environment (Fig. 10.10).
    - Forward motion creates an expanding pattern of flow whereas backward motion creates a contracting pattern of flow.
  - Visual cues of motion can override vestibular signals, causing humans to perceive that they are moving when in fact they are stationary, as demonstrated by the swinging room study by Lee

and Aronson (Fig. 10.9).

- Kinesthesis and Balance
  - **Automatic postural responses** to vestibular input are no longer thought to be simple reflexes, but rather they are learned motor strategies that are influenced by the specific task at hand (walking while reading versus while holding a glass of water without spilling). (see Horak, Henry & Shumway-Cook. (1997). Postural perturbations: New insights for treatment of balance disorders.)
  - Prior experience and the ability to predict the forces that will be exerted on the body allow automatic postural responses to be more efficient.
- Integration of the Vestibular, Visual and Kinesthetic Senses: Motion Sickness
  - **Motion sickness** is typically caused by real (as when riding in a car) or perceived (as when playing a video game) low frequency stimulation of the vestibular system.
    - The **sensory conflict theory** of motion sickness claims that it is caused when the central nervous system receives discrepant information from different senses about the movement of the body.
    - Another theory of motion sickness suggests that it is caused by **postural instability**. People who tend to get motion sick also sway more than people who do not get motion sick when standing inside a moving room (like in the Lee & Aronson study).

Exam 1 (2/21)

Practice exam

Practice exam: with answers

6 Lecture 10: Hearing (2/26)

Reading: Goldstein, pp. 233 - 241

- The auditory system is crucial for navigating our environment,detecting threats in our environment, and communicating with people (and lots of other things, p. 234).
- Sound waves are pressure changes (p. 235)
  - loudness is related to the amplitude of the wave (Fig. 11.2)
    - The physical amplitude of the wave is measured in units of pressure, Pascals

        - The most common unit of loudness is the sound pressure level, measured in decibels, which is a logarithmic (compressive) transformation of absolute pressure levels
            - The dB scale is inherently a relative scale. Barely audible sounds are defined as 0 dB; conversation is ~60 dB; the pain threshold is 140 dB (Table 11.1).
            - Perceived loudness is a compressive function of stimulus intensity.
                - Steven's power law, exponent < 1.
                - Fig. 11.4 is using a log transform (see Ch. 1) to convert the compressive function to a (roughly) straight line.
        - SPL (dB) = $20\log(P/P_{ref})$. See practice problems to get good at using this equation.
    - Pitch is related to frequency (the idea of a pure tone, characterized by a sinusoidal waveform, is abstract but physically realistic)
        - Lower frequency = longer wavelength = lower pitch
        - Higher frequency = shorter wavelength = higher pitch
        - A doubling in frequency is a one-octave increase in pitch
    - Complex sounds are created by summing individual pure tones (Fig. 11.8, 11.10)
- The typical human observer can hear frequencies between 20 Hz and 20,000 Hz
    - We are most sensitive around 1,000 Hz (Fig. 11.7)
        - Coincidentally, the frequencies used in speech are in this range
    - Dogs can hear above 40,000 Hz; dolphins can hear up to 150,000 Hz (sound waves also propagate more efficiently in water, but our ears are not set up to detect them efficiently)

Lecture 11: Ear and cochlea (2/28)

Reading: Goldstein, pp. 241 - 252

- The ear is divided into 3 major parts (Fig. 11.11)
    - The outer ear comprises the pinna and auditory canal.
        - Pinna collects sound and aids localization.
    - The middle ear comprises the tympanic membrane (ear drum) and the ossicles (Fig. 11.12)
        - Mechanical advantage amplifies sound (Fig. 11.14)
    - The inner ear comprises the cochlea and vestibular organs.
        - Hair cells in cochlea transduce sound
- sound pressure waves vibrate the tympanic membrane, which is amplifed by the ossicles to vibrate the oval window of the cochlea (Fig. 11.12), which sets the basilar membrane in motion (Fig. 11.19)
- The cochlea is a twirled-up cone, comprising: the fluid-filled spaces (scala tympani and scala vestibuli) on either side of the basilar membrane, on which the organ of corti is found.
    - Different parts of the basilar membrane move more in response to different frequencies

        - Higher frequencies are represented closer to the base of the cochlea, on the skinny part of the basilar membrane (Figs. 11.20 & 11.23)
    - When the basiilar membrane moves, cilia belonging to the hair cells in the organ of corti are stimulated
        - the outer hair cells have a motile response, that amplifies the vibration of the basilar membrane (Figs 11.28 & 11.29)
        - the inner hair cells change their membrane potential and transduce the physical vibration into neural action potentials
- Neurons do not send action potentials fast enough to respond to 1,000 or 10,000 Hz stimulus
    - The upper limit for a neuron is 500 - 800 impulses/sec (p. 27)
    - A population code solves this problem: different neurons respond to different cycles. Added together, the waveform is represented
    - Timing solves this problem: neurons initiate action potentials only in response to peak deflection (phase-locking, Fig. 11.34).

ICE5
Log examples
Log answers

7 Lecture 12: Central auditory processing (3/4)

Reading: Goldstein, pp. 253 - 261

- Basilar membrane as a **frequency analyzer**
    - Different parts of the membrane move more when stimulated by different frequencies (Fig. 11.19)
    - Frequency analysis converts a time-domain signal (e.g. pressure wave) into a list of what frequency components are represented at what amplitudes (and phases, or relative onset times) in the signal. Fig. 11.32 is the same information as 11.8 & 11.9)
    - Therefore, by measuring the vibration strength at different points on the basilar membrane (Fig. 11.22) you get a **Fourier transform** of the signal, which is frequency analysis.
- **Frequency channels** (Fig. 11.24)
    - In a **masking** experiment (Fig. 11.25 & 11.26), a strong pure tone only elevates **audibility thresholds** for neighboring tones.
    - Each channel has a bandwidth -- a range of frequencies that it "transmits"
        - Cat auditory nerve fibers show finite **bandwidth** (equal on a log scale, Fig. 11.24)
        - Psychophysical functions show similar bandwidth (Fig. 11.31)
- The quality, or **timbre**, of a sound is determined by ...
    - The different frequencies represented in the sound
    - The relative amplitude of those frequency components
    - The relative timing of those frequency components

- The **pitch** of a note is determined by the spacing between the harmonics (Fig. 11.44 & discussion on p. 257)
    - The **fundamental frequency** is the lowest common denominator of all the frequency components.
    - The **harmonics** are the higher frequencies, which are all multiples of the fundamental frequency (e.g. 880 Hz, 1320 Hz & 1760 Hz are all harmonics of a 440 Hz fundamental frequency).
    - Even when the fundamental is missing, we hear the fundamental pitch (which is created by a beat pattern between all the other harmonics).
- **Brainstem & midbrain nuclei** do lots of processing before sound information gets to the brain proper (primary auditory cortex on Heschl's gyrus, on the superior temproal lobe). Fig. 11.36
    - Cochlear nucleus in brainstem: signals from opposite ears are segregated
    - Superior Olivary Nucleus: signals from ears are combined; timing information
    - Inferior Colliculus: contributes to signal localization
    - Medial Geniculate nucleus: in the thalamus
- **Primary auditory cortex** has **tonotopic** maps (Fig 11.42)
    - There's a systematic progression through octaves as you move across cortex
    - **Plasticity** means that cortical representation for important frequency bands can expand with experience
    - A1 is required for pitch perception (Fig 11.43, patient A)
- Other regions of the brain are differentially involved in processing **what** (sound identification) and **where** (sound localization information) Fig. 11.38
    - Dorsal and **parietal** areas are required for localization (Fig. 11.39, subject ES)
    - Ventral and **anterior parts of the temporal lobe** are required for identification i(Fig. 11.39, JG)

Lecture 13: Auditory scene analysis (3/6)

Reading: Goldstein, pp. 265 - 273; 278 - 282

- **Deafness** (absence of sound perception) and **hearing loss** (diminished sensitivity to sound)
    - usually fall into one of two categories
        - **Sensorineural** damage: unrecoverable damage to the inner ear (hair cells, auditory nerve)
        - **Conductive**: often recoverable blockage of auditory canal or damage to the tympanic membrane
    - have many causes
        - heredity (severity of hearing loss with age can be predicted from family members)
        - trauma (e.g. punctured tympanic membrane)
        - ototoxic medicines (e.g. quinine, some chemotherapy drugs)
        - exposure to loud noise (damages inner hair cells) -- even just 85 dB if

exposure is long term (e.g. snow-blowers, highway noise ...)
    - Three examples of treatment:
        - **cochlear implant** (p. 280 - 282, Fig 11.46): for severe damage to inner ear
        - hearing aids: amplify and filter sounds to treat partial hearing loss
        - tubes in ears: either to relieve pressure through tympanic membrane, or to keep eustachian tubes open
- Sound transfer through bone: we (like dolphins) can receive auditory stimulus from sound that vibrates the basilar membrane after being transferred through our skulls instead of our auditory canals
    - A **tuning fork** can be heard when the base is pressed against the temple or mastoid bone (behind the ear), even when there is complete conductive hearing loss. This is a good diagnostic tool.
    - Hasbro Tooth Tunes toothbrush.
- Sound localization, auditory scene analysis
    - Sounds from all directions are mixed when they reach our ears, so we need tricks to figure out what came from where.
    - The coordinate system for sound localization is specified by **distance**, **azimuth** and **elevation** (Fig. 12.1).
    - There are three cues to the location of a sound source
        - **Interaural timing difference (ITD)**: if the same pitch reaches one ear before the other, we will interpret as being closer to the first ear. This is most useful for low frequencies (Fig. 12.4)
        - **Interaural level difference (ILD)**: for high frequencies, our heads cast acoustic shadows, so SPL is weaker in the more distant ear (Fig 12.5, 12.6)
        - Spectral cues (**head-related transfer function, HRTF**): the shaped of each person's pinna acts as an individualized frequency filter, that slightly changes timbre of sound, depending on the elevation (Fig. 12.7)
    - Remember the experiment in which people's ability to localize sounds with their eyes closed was measured before and after re-shaping the **pinna** (Fig 12.10).
    - Hearing indoors: we know we're indoors because the same sound reaches us several times (echoes). Lots of time and money goes into figuring out the **acoustics** of indoor spaces (Fig. 12.22).

8 Lecture 14: Speech (3/11)

Reading: Goldstein, pp. 286 - 300

- **Time-frequency analysis** shows how the frequencies that contribute to a sound change over time (Fig. 13.9)
    - This is like the "graphic equalizer" on a stereo.
    - This is what a spectrogram is: the temporal evolution of the pitch of a person's voice.
    - **Formants** are the harmonics of a person's voice (Fig. 13.3).
- **Vocal tract**: the lips, teeth, alveolar ridge, tongue, soft pallate, epiglottis, glottis (**voxal cords**) and **larynx** comprise the voxal tract -- these are the things we use to modulate our

voice when we talk (Fig. 13.2)
    - We tense or relax our vocal cords, and change the shape of our larynx, to affect the **pitch** of our voice.
    - Throat singers create a second pitch using their pharynx (behind the tongue, above the vocal cords and larynx).
- **Phonemes** (Table 13.1) are the basic unit of speech (the smallest part that will change a word's meaning when it is changed)
    - Different languages have different phonemes.
- Computers have a difficult time understanding voices, even when we have no trouble
    - **Segmentation problem**: it's not always clear where one phoneme stops and another starts (Fig. 13.5)
    - **Co-articulation problem**: formants are different for the same phoneme, depending on what phoneme precedes or follows it (Fig. 13.06).
    - **Speaker problem**: we all speak different dialects, with different pronunciations -- often depending on where we grew up or where our parents grew up (Fig. 13.07).
- Practiced speakers/listeners hear phonemes **categorically**: a continuous range of sounds can be made between "da" and "ta," but we will hear one or the other (Fig. 13.11)
    - This is a learned distinction. People in different language groups draw different phoneme boundaries.
    - **Babies** are born with the ability to hear all phonemes. At about **12 mo**. of age they lose the ability to distinguish between phonemes that don't affect word meaning.
- Specialized language areas in the brain (Fig. 13.16) were discovered originally through lesion studies; now we have non-invasive neuroimaging techniques to study how the brain processes language. *The following definitions are overly simplistic, but a useful starting framework.*
    - **Wernicke**'s area is responsible for word meaning (lexicon, or dictionary). Damage to Wernicke's area results in fluent aphasia, or word salad -- patients have no difficulty generating language, but it is often non-sensible ("colorless green ideas sleep furiously"). This part of language is called semantics.
    - **Broca**'s area is responsible for sentence structure (syntax -- how words go together). Damage to Broca's area results in very labored production of language, but meaningful words (appropriate to context) are used. Often called telegraphic speech, since patients generate a few meaningful words, as if paying by the length of the message. Phineas Gage was a famous patient with damage to Broca's area.
        - when you read silently to yourself, Broca's area is active -- a counter-intuitive result from neuroimaging studies that shows us language is an inherently spoken thing (we re-generate the words in our head to understand them)
    - **Conductive aphasia** can be difficult to diagnose because it can share symptoms with Wernicke's and Broca's aphasias, but it results from the loss of connection between crucial language areas.

Lecture 15: Music & Lab Day (3/13)

Reading: Goldstein, pp. 275 - 278

- Very brief introduction to music perception
  - Pitch perception is not absolute (climbing pitch illusion, "Incredible Audio Illusion" on YouTube.com)
  - We group pitches by proximity to hear a melody. Demonstration: interleaved violin parts in Tchaikovsky's 6th Symphony, 4th movement (http://lipscomb.umn.edu/music_cognition/)
- 8 Stations
  - **McGurk Effect:** the lips say "ga" without sound, the ears hear "ba" without sight, but watching and listening we hear "tha" or "da"
  - Virtual Barbershop demonstration of **sound localization** when stereo signals (with ILD and ITD intact) are recorded using the HRTF and played back to the correct ears
  - **Tuning Forks**: 256 Hz (middle C) transfers through the skull better than 512 Hz (an octave above)
  - Hasbro Tooth Tunes: how cool is that!
  - **Decibel** meter: snapping your fingers is 100dB if it's right close to your ear, but sound falls off with distance because the pressure waves spread out as they travel.
  - Word scramble: we use **top-down knowledge** to interpret both spoken and written language, so we only need sparse cues.
  - **Spectrogram** reading: given a series of minimal pairs starting with "b" (short words or word-like things that are different by just one phoneme), look at a spectrogram of a short word starting with "d" and guess what the speaker is saying.
  - Phonetic transcription: using Table 13.1, transcribe the phrase "linguistics is awesome" using the **International Phonetic Alphabet**

ICE6

ICE7

9 Lecture 16: Light transduction (3/25)

Reading: Goldstein, pp. 29 - 35

- Structure of the eye (Fig. 2.8)
  - Terms to know
    - **Cornea** - transparent but alive curved structure at the front of the eye, with embedded nerve endings (pain, touch and thermal sensation) that provides 80% of the focusing power of the eye
    - **Sclera**: white, hard outside of the eyeball
    - **Aqueous humor**: low viscosity fluid behind the cornea, in front of the lens
    - **Iris**: muscular, colored tissue that surrounds (and shapes) the pupil
    - **Pupil**: hole through which light enters the eye
    - **Lens**: flexible, clear substance that provides 20% of the focusing power of the eye
    - Ciliary (or lens) **muscles**: muscles that control shape (curvature) of the lens
    - **Vitreous humor**: high viscosity fluid filling the eyeball

        - **Retina**: sheet of neurons at the back of the eye
        - **Pigment epithelium**: black layer behind the retina where visual pigments are replenished
        - **Optic nerve**: collection of axons leaving the eye
        - **Blind spot**: location on the retina where there are no photoreceptors (because axons heading for the optic nerve occupy that space)
        - **Fovea**: where light from the center of gaze lands on the retina
    - Contribution of cornea to vision
        - 80% of focusing power (p. 30), but not flexible
            - if the cornea focuses too fast or the eyeball is too long, you're near-sighted
            - if the cornea does not focus strongly enough, light from far objects focuses behind the retina and is blurry: far-sighted
        - LASIK can reshape the cornea to eliminate the need for corrective lenses for near-sightedness, far-sightedness, astygmatism and other issues
        - The cornea can be replaced surgically in extreme cases.
    - **Lens**
        - Only **20% of focusing power of the eyes**, but important because it is flexible and gives us the ability to accomodate
        - **Accomodation**: the ability to focus on things that are near or far away (Fig. 2.11)
        - **Presbyopia**: "old eyes" (p. 31) the lens gets hard and can't be squished by the ciliary muscles to focus on things that are near. Eventually, your arms won't be long enough to hold reading material far enough away for your eyes to focus on it (Fig. 2.12).
        - Cataracts: crystallization of the lens scatters light, making it hard to perceive detail. Solution: replace the lens.
- The retina
    - In the eye backward, so light travels through several layers before it gets to the photoreceptors, specifically the outer segments of the rods and cones (Fig. 2.13).
    - **Rods**
        - More sensitive to light than cones
        - Not present in the fovea
        - Only one kind of visual pigment (therefore night vision is black-and-white)
    - **Cones**
        - Not as sensitive as rods
        - Concentrated in the fovea but present every where
        - Three kinds of visual pigments -- this is where we get color vision
- Light transduction
    - **Rods and cones have stacks of discs in their outer segments** (Fig. 2.19)
    - The disks are there to increase the surface area, so there can be lots of membrane area to carry photosensitive proteins
    - The photosensitive protein in a rod is rhodopsin

        - rhodopsin is a 7 transmembrane protein, with retinal attached to one of the transmembrane domains (Fig. 2.19)
        - When light (of the right wavelength, or color) hits **retinal**, it changes conformation (Fig. 2.20), acting as a switch to start an **enzyme cascade** (Fig. 2.22) in the cell, which eventually changes the rate at which rods release neurotransmitters
    - The photosensitive protein in a cone will have one of three different pigments (like retinal, but slightly different molecular structure so it absorbs optimally at a different wavelength - see Fig. 2.29 for a comparison of rod photopigment with cone photopigments).
    - After absorbing light, photopigments need to be regnerated at the pigment epithelium ... using molecules derived from Vitamin A (which is half of beta-carotene).

Lecture 17: Retina (3/27)

Reading: Goldstein, pp. 36 - 41

- Light
    - is electromagnetic radiation -- sinusoidal electric and magnetic fields that are oriented opposite to each other, out of phase (one gets large while the other gets small, then they trade off), and can propagate through a vacuum.
        - speed = wavelength x frequency
        - electromagnetic radiation propagates at a constant speed (300 million meters per second in a vacuum), so large wavelength corresponds to low frequency, and vice versa.
    - is one small region of the electromagnetic spectrum (Fig. 2.19), and is visible to us only because we happen to have molecules in our retina that absorb that range of retinas
        - a honeybee would consider UV to be visible (Fig. 2.30), where we just think of it as a sunburn hazzard.
        - we define visible light as EM radiation in the range 400 - 700 nm.
    - Even though light propagates as a continuous dance between electric and magnetic fields, the magnitude is quantized at very low light levels - a photon is the smallest amount of light that can be generated
        - A blue photon has more energy than a red photon
        - A photon in the gamma ray segment of the spectrum conveys much more energy than a photon in the visible portion ... which is much more energetic than a radio wave (which has a wavelength of tens or hundreds of meters)
- Foveal vs. peripheral vision
    - Definition of **fovea**: the region of the retina where light is focused when it comes straight through the eyeballs optical axis (Fig. 2.13)
        - The fovea contains only cones (Fig. 2.15).
        - The fovea is a slight depression in the retina, which maximizes the number of cones that can be there.

        - The fovea is the central 1 degree of visual angle.
    - Definition of **macula**: the central 5 degrees of visual angle
        - Larger than the fovea, but crucial to good visual perception
        - Macular degeneration (p. 33 and Fig. 2.18) is the loss of the cones in the macula.
            - 10% of patients age 66 - 74 have macular degeneration.
            - 30% of patients age 75 - 85.
    - The **peripheral retina** is dominated by rods, but also contains cones
        - The **blind spot** (Fig. 2.17) exists in each eye, and is created by a lack of photoreceptors at the head of the optic nerve.
- **Dark adaptation**
    - During daylight viewing, rods are almost completely **photobleached**, and cones are partially photobleached.
        - photobleaching occurs because the pigment epithelium cannot regenerate 11-cis retinal as fast as it is converted to all trans retinal (a process called isomeration) by arriving photons (Fig. 2.25 shows photobleaching of a retina with no pigment epithelium around)
    - In the dark visual pigments are replenished
        - It takes about **7 min. for cone visual pigments to replenish** (Fig. 2.24)
        - It takes **20 - 30 min. for rod visual pigments to replenish**
        - A normal observer's threshold decreases with time in 2 phases
            - for the first few minutes, the cones are more sensitive than the rods
            - after 7 or 8 minutes, the rod photopigments have replenished and the rods become the most sensitive cells in the retina
        -

Practice Exam 2

Practice Exam 2, with answers

ICE8

10 Exam 2 (4/1)

Lecture 18: Low vision research (4/3)
Reading: (see pdf linked below)

I. What is low vision?

a. Low vision can be defined as any chronic visual condition that impairs everyday activities and is not correctable by glasses or contact lenses.

b. The distinction between low vision and blindness- "Blindness" is

defined as the lack of any useful pattern vision.

c. The most common causes of low vision and blindness in the U.S. are age-related eye diseases (macular degeneration, glaucoma, and cataract).

II. Dimensions of vision loss

a. Acuity

i. Visual acuity refers to the ability of the eye to resolve fine detail.

ii. Someone with a visual acuity of 20/200 needs letters to be 10x as big or 10x closer as someone with an acuity of 20/20 to be able to be able to read them.

iii. There are several problems with using just acuity measurements to characterize low vision (Legge, 2005):

1. Standard eye charts cannot measure very low acuity.
2. Testing conditions (such as lighting) can affect measurements.
3. Acuity is not always a good predictor of how well someone can perform everyday tasks (such as reading).

b. Contrast

i. Contrast sensitivity refers to the ability to see small differences in shades of gray that distinguish one pattern feature from another.

ii. The contrast sensitivity function describes the relationship between detecting low contrast patterns with the size of patterns.

1. As the size of the pattern decreases, contrast sensitivity increases then decreases.

c. Visual Field

i. Visual field is the area that can be seen with one fixation of the eyes.

III. Common Visual Disorders (spend less time on- already covered some)

a. Age-related Macular Degeneration (AMD)

i. Dry type- yellow deposits collect in the retina, damaging the photoreceptors in the macula. This type of AMD develops gradually.

ii. Wet type- occurs because of an abnormal growth of blood vessels in the retina.

b. Glaucoma

i. Occurs when the pressure inside the eye becomes excessively high because of an obstruction of the drainage of the fluid inside the anterior chamber. The increased pressure can result in damage to the cornea or optic nerve.

ii. Damage to the optic nerve typically results in peripheral field loss.

c. Cataracts

i. Opacities in the lens of the eye that can result in blurred vision.

ii. Ostrovsky, Andalman & Sinha (2006)

1. Subject SRD had congenital cataracts until the age of 12, when she had surgery to remove the cataracts. This study showed that she was able to perform basic visual tasks, such as matching shapes and counting objects, even though she did not gain useful vision until later in life.

IV. Navigation Technology for the Visually-Impaired

a. Navigation- the ability to plan and follow routes from one location to another in an environment.

i. Not the same as obstacle avoidance.

b. Information that is important for navigation:

i. Current position in the environment

ii. The distance to a location (or the next place to turn)

iii. The direction to travel to a location

c. Vision provides useful cues (landmarks, depth cues, optic flow) to help determine current position, distance, and direction of travel. Navigation technology for the visually-impaired must convey the same information to users in a way that is practical and easy to use.

Article on low vision

11 Lecture 19: Retinal processing

Reading: Goldstein, pp. 48 - 66

- Architecture: 5 main cell types in 3 main layers. From back to front, these are:
  - Input layer: **photoreceptors** (rods and cones)
  - Processing layer: horizontal cells, amacrine cells & **bipolar** cells
  - Output layer: **retinal ganglion cells**
- **Convergence** in the retina
  - There are ~126 million photoreceptors in each eye (120M rods, 6M cones), but only 1 million ganglion cells. Which means that, on *average*, the output of 120 rods is combined to determine the response of a single ganglion cell; 6 rods -> 1 ganglion cell (**more convergence for rods than cones**).
  - In the fovea there is no convergence (1 cone -> 1 ganglion cell).
  - Convergence plays several roles
    - Increases sensitivity
    - Decreases acuity
    - Creates a network in which center-surround ganglion cell responses can be calculated.
- **Visual acuity**: the ability to see very fine detail
  - Acuity is **best in the fovea**, where there are as many ganglion cells as there are cones.
  - Acuity is best with **good light levels**
    - Better response from cones
    - **Pupil diameter is 2 - 5 mm** (instead off fully dilated~9mm which allows more off-axis light which blurs vision)
  - Acuity is best with longer exposures (longer than ~100ms)
  - Acuity is best when image is focused (contributing factors: accommodation of lens and constriction of pupil)
- Eye movements
  - A **saccade** is a fast motion of the eye, between fixations.
  - When we study a picture, we move our eyes around it -- on average every few hundred milliseconds. It's actually impossible to hold your eyes perfectly still.
  - Eye movements are often reflexive or automatic
- Center/surround receptive fields and lateral inhibition.
  - While the retinal circuits are very complicated, the effect is quite simple.

    - Neighboring cells tend to inhibit each other
        - A simple circuit with **lateral inhibition** explains **Mach bands**, the illusory appearance of light and dark lines flanking an abrupt change in brightness (like a shadow).
        - Retinal neurons have **sustained response levels**, so **excitation** can increase rate of neurotransmission, and **inhibition** can decrease the rate of neurotransmission.
    - **Definition of receptive field**: for each visual neuron, this is the region of visual space in which a change in lightness or color will cause a change in the neuron's firing rate.
    - **Retinal ganglion cells have center-surround organization**, with 4 main types
        - Transient response, excitatory center/inhibitory surround
        - Transient response, inhibitory center/excitatory surround
        - Sustained response, excitatory center/inhibitory surround
        - Sustained response, inhibitory center/excitatory surround
    - Key function: when center and surround are balanced, the RGC will not change its firing rate in response to **uniform illumination.**
- Cortical **receptive fields**
    - If you combine the receptive fields from a line of center/surround receptive fields, you get a receptive field that is an excitatory bar flanked by inhibitory bars (or vice versa, depending on whether the centers were excitatory or inhibitory.
    - This is how oriented receptive fields are created in primary visual cortex.

Lecture 20: Color perception; organization of lateral geniculate nucleus

Reading: Goldstein, pp. 142 - 159

- **Uses of color**
    - Scene segmentation; object detection; object recognition
    - Mate selection
    - Threat detection
    - Aesthetic enjoyment
- Competing theories for color vision (proposed during mid-19th century; both correct)
    - **Trichromatic theory**
        - Basic idea: any color can be matched with a combination of 3 primary colors (and it's not important exactly which three colors)
        - Remember the distinction between additive and subtractive color mixing.
    - **Opponent process theory**
        - We see colors as opponent pairs: red vs. green & blue vs. yellow.
    - Resolution lies in the retina: we have 3 kinds of photoreceptor pigments, but the circuity of the retina combines them so ganglion cells respond along a red/green axis or along a blue/yellow axis
        - Ganglion cells therefore have center/surround receptive fields sensitive to:
            - Luminance (**bright/dark**)

        - The **red/green** axis (excited by red in the center, inhibited by green in the surround ... or vice versa)
        - The **blue/yellow** axis. This circuit is created by adding the L & M responses, then subtracting the S (blue) response.
- Color deficiencies
    - True **color blindness** (the lack of color sensation) is rare, found in rod monochromats and after particular brain injuries.
    - **Color deficiency** is more common, and results from the lack of one of the cone pigments
        - **Protanopia**: no **L** (long wavelength, or red) pigment. Hard to distinguish between red and green; reds look particularly dark. A few percent of the male population; a very small fraction of a percent of the female population (since the genes for the cone pigments are on the **X chromosome**).
        - **Deuteranopia**: no **M** (medium wavelength, or green) pigment. Hard to distinguish between red and green. A few percent of the male population; tiny fraction of a percent of the female population.
        - **Tritanopia**: no **S** (short wavelength) pigment. Difficult to distinguish yellows, greens and blues. Very rare.
- **Optic chiasm**
    - Where the 2 optic nerves (each with 1 million axons) cross
    - **Half of the information from each eye crosses**
    - This way, information from the left side of visual space reaches the right side of the brain, and vice versa.
- The **lateral geniculate nucleus (LGN)**
    - A nucleus **in the thalamus** (named because of its location and shape).
    - All ganglion cell outputs make synapses here with neurons that project to the cortex.
    - Receptive cells are center/surround like the retinal ganglion cells
    - **6 layers**, distinguishing
        - **Eye of origin**: which eye the information is coming from
        - **On- or off-**receptive field: whether the center is excitatory or inhibitory
        - **Magnocellular or parvocellular** pathway: see below
- Magnocellular and parvocellular systems
    - Although the picture is more complicated, it is useful to think of 2 streams of information coming from the eyes to the brain.
        - The magno and parvo systems are named for the size of their cell bodies in the retina. **Magno for large, parvo for small**.
    - The **magnocellular** system carries information about large, fast things (**low spatial frequency information; high temporal frequency information**).
        - magnocellular system is **colorblind**
    - The **parvocellular** system carries information about small, slow things (**high spatial frequency information; low temporal frequency information**).
        - parvocellular system is **sensitive to color**.

ICE9

12 Lecture 21: Visual system organization

Reading: Goldstein, pp. 71 - 82

- Themes for organizing the visual system (Fig. 4.25)
  - Microscopic: within a visual area, how are selective responses organized?
  - Macroscopic: between visual areas, how does selectivity change?
- Visual areas vs. visual maps
  - Many areas of the brain respond to visual stimuli (e.g. Halle Berry cell in entorhinal cortex, Fig. 4.29), but a region that responds to words and ideas as strongly as it responds to visual stimuli should not be called a visual area.
  - The most stringent test for whether a region is a visual area is whether it contains a regular map of some kind of stimulus attribute, e.g. in V1, different orientations are represented in different places on cortex.
  - A looser test for "visual area" is whether the region responds better to visual stimuli than other stimuli (like sounds or words). This test is used for many higher visual areas, in the parietal and temporal cortex.
- Neuroimaging methods: non-invasive study of brain activity
  - **PET**: positron emission tomography. Requires the use of small amounts of radioactive tracers, and each data point takes a long time to acquire, but this technique gives us good information about metabolic activity, or specific neurotransmitters (e.g. maps of dopamine concentrations in the brain).
  - **EEG**: electroencephalography. This measures the electric fields in the scalp that are generated by clusters of neurons that are strongly stimulated.
  - **MEG**: magnetoencephalography. This measures the magnetic fields (perpendicular to the electric fields) that are generated by clusters of active neurons. MEG has slightly better spatial resolution (both techniques have millisecond temporal resolution), but is more difficult and much more expensive than EEG.
  - **fMRI**: functional magnetic resonance imaging.
    - MRI has been in clinical use since the '70's
    - functional MRI is the use of MRI images to detect blood flow and blood oxygenation changes in the brain, which are the result of neural activity.
      - Unlike the description in Goldstein on p. 74, MRI DOES NOT cause "the hemoglobin molecules to line up like tiny magnets."
      - MRI is sensitive to the details of the magnetic field in your head, which is created by putting you in a very strong magnetic field, and which is changed when the hemoglobin loses its oxygen and becomes paramagnetic (the free electrons on deoxyhemoglobin cause microscopic amplifications of the local magnetic field). Thus hemoglobin affects the magnetic field and the signal, but DOES NOT ROTATE.
  - Neuroimaging methods are useful, but must always be interpreted in the context of

behavior: having a picture of a brain does not make you right!

- Organization of primary visual cortex (striate cortex, or V1). *Relevant images/descriptions will be provided before the exam in a study guide. Some organizational principles are described on pg. 75 - 79.*
  - V1 is located in posterior occipital cortex
    - it is named "striate" cortex because when the tissue is stained, the dense input from the LGN shows up as a dark band in the input layers.
  - **V1 neurons respond well to short, oriented bars**
    - **Receptive fields are small**, which means neurons respond only to a small region of the visual field.
    - Like retina and LGN, the receptive field center can be excitatory or inhibitory.
    - The flanking bars are the opposite -- an excitatory center is flanked (not surrounded) by inhibitory regions.
  - V1 has **retinotopic organization**, which means that neurons that respond to neighboring regions of the visual field are located close to each other on the cortex.
    - An aside on hypercolumns (gratuitous information): V1 is therefore tessellated by hypercolumns, each representing a little region of space. A *hypercolumn* is a chunk of cortex that has every kind of selective response represented once (one column for each eye, which contains an entire pinwheel for each eye ...). At least some part of the hypercolumn will respond to any kind of stimulus in that little region of space.
      - Hypercolumns are the same size everywhere on cortex, which means that smaller regions of space are represented in the hypercolumns that represent the fovea ... receptive fields are smaller here ... and hypercolumns in the cortex that represents peripheral V1 contain neurons with larger receptive fields.
  - V1 has **cortical magnification**, which means that the region of cortex that represents stimuli in the cortex is disproportionately large.
  - V1 has **layers**. Like the rest of cortex, input layers are in the middle. Local processing and connections to other parts of the brain are in the superficial layers.
  - V1 has **columns**. Like the rest of cortex, neurons with similar response properties are clustered in columns that run throughout the depth of cortex. Inputs from the 2 eyes are segregated in **ocular dominance columns**.
  - V1 has **pinwheels**. Columns of neurons that with similar preferred orientations are generally next to each other, creating a spinning pattern of responses as stimuli of different orientations are presented.
  - V1 has **blobs**. Blobs are regions of V1, scattered with no particular regard to column boundaries, where color-selective neurons are clustered. They're named blobs because of their shape, which shows up when you stain the cortex for cytochrome oxidase, an enzyme involved in oxidative metabolism (which means that blobs consume more oxygen than other parts of cortex).
- What and where pathways. Similar to auditory stimuli, more dorsal regions of the posterior part of the brain (parietal cortex) are involved in processing information about location

("Where/how pathway" Fig. 4.13); more ventral (temporal) regions are involved in recognizing objects ("what" pathway).
  - **Magnocellular** information (colorblind neurons responding to large, fast things) tends to head up the "where/how" pathway (Fig. 4.15).
  - **Parvocellular** information (color-sensitive neurons responding to small, slow things) tends to head down the "what" pathway (description, p. 79).

Lecture 22: Visual system development

Reading: Goldstein, pp. 82 - 90, 352 - 358

- Babies are born with underdeveloped neurons, which has many consequences
  - Retinal photoreceptors have big inner segments and small outer segments, so visual information is undersampled (Fig. 16.4).
  - Cortical neurons have relatively sparse connections. The first 6 months of development witnesses a great elaboration of connections (Fig. 16.3 -- note neural density is roughly the same, but connections -- axons & dendrites -- are much richer).
- Many aspects of vision change rapidly during the first 3 months
  - **Visual acuity** is awful at birth -- 20/400 vision (legally blind). Acuity is reasonable at 3 months, but still improving.
  - **Depth perception** begins at 3 mo. (evidence from vergence -- eyes track objects as they get closer, and stereo vision (Fig. 16.11); infants understand that closer things are larger ~ 7 mo.
  - Object unity: infants understand **occlusion** (Fig. 16.16, 16.17) and Gestalt principle of common fate at ~3 mo.
- Many aspects of vision continue to change for the first several years of life
  - **Contrast sensitivity function** (Fig. 16.6 & study guide) makes good progress toward "normal" (sensitivity to high spatial frequencies increases; this is related to acuity) during the first several years of life, but does not reach adult form until ~10 years old.
  - **Hyperacuity** (the ability to perform better on a **Vernier acuity** task than predicted by normal visual acuity) develops after ~10 years.
- Some things are good from Day 1
  - **Color** vision appears the same as for adults (Fig. 16.9)
  - Infants show an immediate preference for **faces** (Fig. 16.13, 16.14), in spite of horrid visual acuity.

UWO MedsWeb: early vision

13 Lecture 23: Object perception, face perception

Reading: Goldstein, pp. 93 - 116

- **Gestalt principles**: these are not absolute rules, but common sense principles explaining why we see what we see, developed during the 1st half of the 20th century. By contrast to

structuralism (whole = sum of parts), Gestalt principles describe how the whole (our perception, or interpretation of an image) can be greater than the sum of the parts. Of the 9 in the text (p. 99 - 103), the 4 in bold are required knowledge.

- *Pragnanz*, or **simplicity**. The simplest shape is usually the right explanation for an image.
- Similarity. Things that look alike probably come from the same source.
- **Good continuation**. Contours rarely change abruptly; curves are smooth, acute angles are rare.
- Proximity. Things that are close together belong together.
- **Common fate**. Things that move together belong together.
- Meaningfulness or **familiarity**. We cluster features into familiar patterns (e.g. faces made out of rocks or branches in an image).
- Common region. Features that are circled or on the same background belong together.
- Connectedness. Connected features belong together.
- Synchrony. Features that appear and disappear at the same time belong together.

- Modern questions in vision research.
  - *How do neurons efficiently encode our environment?* Neurons tend to be **tuned** to (i.e. respond better to) features or shapes that are common in our environment. This is a consequence both of the architecture that we're born with and of our experience.
    - Low-level example: **oblique effect** (Fig. 5.30)
    - High-level example: fusiform face area (Fig. 4.19)
  - *Do we recognize objects or perform figure/ground segmentation first?* **Figure/ground segmentation** relies on low-level cues (different features, like lines oriented in different directions, Fig. 5.29 ... but NOTE that this figure exaggerates the reduced response in (b)); shape or object recognition requires high-level knowledge.
    - It's a chicken-egg problem that we haven't solved.
    - Modulation of V1 responses (Fig. 5.29) requires awareness of the shape.
    - Identification of foreground/background depends on shape recognition (Fig. 5.31).
  - *How do we recognize objects from novel viewpoints?* A computer would be flummoxed by the 3 views of the chair in Fig. 5.6. Humans aren't. There are 2 dominant theories for how we recognize shapes
    - **3D models**. Irv Biederman's geons are one instantiation of this type of theory, which holds that our object recognition areas (e.g. **LOC**) maintain ful 3d models of objects, which we can mentally rotate to compare against experiment.
    - **A set of 2D models**. Our visual experience is inherently 2 dimensional, because the entire world is projected onto the retina, so it would make sense if what we learn about objects is a series of 2D projections (Fig. 5.37). Some experiments show that our viewpoint dependence acts like we're interpolating between stored 2D memories.
  - *Where and how does the brain store and compute object models?* This is the

$100,000 question, and so far we know there are 2 pieces to the puzzle.
    - Specialized areas. The list of specialized visual areas is growing: lateral occipital complex (LOC, shapes), extrastriate body area (EBA, body parts), fusiform face area (FFA, faces), parahippocampal place area (PPA), motion-selective complex (MST or MT, motion) ... Each responds preferentially (but not uniquely) to one type of stimulus.
    - Coordination between areas.
        - FFA responds better to faces than to chairs or houses ... but it also responds to houses. Perhaps the object code is distributed within and between multiple areas.
        - Multiple copies of each piece of visual information are processed in multiple visual areas (e.g. Patient DF, with damage to the ventral stream, who could not describe the orientation of the mail slot (form recognition), but could post a letter (action) -- so clearly she could see the slot, and could tell what angle it was at ... she just couldn't describe it).

Lecture 24: Attention and stimulus selection
Reading: Goldstein, pp. 121 - 140

- Vision without attention (**inattentional blindness** -- we don't see what we're not paying attention to.)
    - **Scene gist**. In a very brief image presentation, we can't detect a lot of the details or name a lot of the objects, but we can tell if it is outdoors or indoors, and whether there are people or animals present.
        - This very rapid processing is largely attributed to the magnocellular system -- fast, but not much detail.
        - We can get the scene gist in the absence of attention
    - **Change blindness**. Many details can change without our awareness.
        - In normal experience, change is accompanied by motion, which is a very salient cue.
        - In the lab, we put white space between two images so there are no motion cues, and entire engines can disappear off of airplanes without our noticing it.
- Vision with attention (attention is the filter that decides which sensations get turned into perception).
    - What grabs our attention?
        - **Salient** image regions. When we start to look at an image, our eyes are attracted to points of high contrast or fast motion.
        - Other parts of important **objects.** Experiments investigating how attention gives our vision a performance advantage (better detection threshold) find that all parts of a cued object have an advantage over a distractor object (Fig. 6.5).
    - What does attention do for our vision?
        - Attention binds together features that belong to the same object (Fig. 6.18)

        - Without attention, people perform poorly at conjunction tasks (Fig. 6.21 & 6.22).
        - Without the parietal lobe (brain damage), people cannot perform conjunction tasks (Balint's syndrome, Table 6.1).
        - ... this doesn't mean that attention is in the parietal lobe. These are just 2 aspects of the conjunction problem.
    - Attention changes neural responses
        - Object-selective responses (e.g. IT neuron that likes parrots, Fig. 6.26, or Harrison Ford response, Fig. 5.39) are only present when the observer is aware of the stimulus.
        - Enhancement of early visual responses (e.g. figure/ground segmentation, Fig. 5.29 or simple light response, Fig. 6.24) is only present when monkey is aware of or attending to the stimulus

ICE10

Biological motion: adjustable individual

Biological motion: many examples

14 Lecture 25: Depth & Size perception

Reading: Goldstein, pp. 169 - 192

- Three types of cues for depth perception
    - **Oculomotor**: proprioceptive information from **oculomotor muscles** (which rotate the eyeballs to **converge** at a particular depth) and **ciliary muscles** (which compress the lens to change focal length)
    - **Monocular** cues: these are cues that can be perceived without stereo vision, i.e. with just one eye.
        - Occlusion: stuff gets in front of stuff
        - Relative height: things on the ground at a distance look like their base is higher
        - Relative size: things farther away are smaller
        - Perspective convergence: parallel lines look like they get closer as they get farther away
        - Familiar size: if we know how big something is, it will look farther away when it seems too small, and close to us when it seems too big.
        - Atmospheric perspective: far away things look hazy (this cue can be misinterpreted when hiking on a clear day!)
        - Texture gradient: similar to relative size, textures look finer as they recede
        - Shadows: Kersten's ball-in-a-box demo (http://gandalf.psych.umn.edu/users/kersten/kersten-lab/demos/shadows.html) illustrates that shadows are a powerful cue for relative height and a depth – as long as they're consistent with our "**light from above**" assumption (illustration at end of Ch. 6).

        - Movement parallax: as we move, things at different depths cross over each other. YouTube video: http://www.youtube.com/watch?v=Jd3-eiid-Uw
        - Accretion/deletion: when one thing moves in front of another, the amount of the thing in back that you can see gets deleted; when the front thing moves out of the way, there's accretion of the back object.
    - **Binocular cues**: stereo vision
        - **Disparity** is the relative location of images of the same object on each retina.
            - The **horopter** is an imaginary arc drawn through the thing the eyes are converged on, which traces out the location of all other objects in the 3D visual field that will land on the retinae with zero disparity.
            - In *primary visual cortex*, **disparity neurons** are tuned to the relative location of images of the same object on each retina. Some neurons are tuned to near
            - The **correspondence problem** is the problem of figuring out how to line up the images on each retina to perceive depth. It's a complex calculation and we're not sure how the brain does it.
        - 10% of the population is stereo-blind, lacking disparity neurons and unable to perceive the depth cues in **random dot stereograms** (images without any monocular cues to depth, in which 90% of observers can still see depth ... with some training ... by looking through the image to a focal plane at which the dots in the stereogram line up to provide depth-from-disparity cues).
            - **Strabismus** is the misalignment of the two eyes. Sometimes this is due to problems with oculomotor muscles, which can be cured by surgery.
            - **Amblyopia** occurs when the brain receives different images from the two eyes. This can be because of strabismus or because the optical path in one eye is much longer or shorter than the other, in which case one image will always be blurry
                - The brain discards the information from the non-dominant eye, creating monocular vision instead of binocular.
                - Usually this is not discovered in children until 3 or 4 years of age. If treated young, stereo vision can develop normally.
- Size
    - We are horrid at judging size of unfamiliar objects without depth information.
    - We are very good at perceiving constant size as an object changes depth.
        - The **Ames room** demonstrates a situation in which size constancy breaks down because depth cues are wrong.
        - The **Ponzo** illusion demonstrates how strongly our perception of size is affected by our perception of depth.

Lab Day #3

ICE11

Strabismus & Amblyopia

15 Lecture 27: Perception and action

Reading: Goldstein, pp. 215 - 230

Exam 3

ICE12

Study guide, Exam 3

Practice Exam 3

Answers, practice exam

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