

Introduction to Sensation and Perception

INTRODUCTION TO SENSATION AND PERCEPTION

STUDENTS OF PSY 3031 AND EDITED BY DR. CHERYL OLMAN

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INTRODUCTION

This book was created by the students of PSY 3031: Sensation and Perception, as a class project, because there is no existing open-source textbook for S&P. Content is, for the most part, re-used and re-mixed from existing open-source materials from Psychology and Anatomy and Psychology textbooks.

We wanted to create a resource with a stronger neuroscience foundation than your average psychology textbook, with strong links between physiology and perception. The final product will always be a work in progress, but hopefully a useful collection of materials to support college-level courses that want to understand how human physiology supports human perceptual experiences.

The course has two over-arching themes or guiding principles, both of which rest on the basic understanding that perception is an interpretive act, which means that our perceptions are sometimes only loosely based on our sensory experiences:

- Our brains shape our environment: there are many things that we simply do not perceive because we are not prepared to perceive them.
- Our environments shape our brains: color categories and phonetic boundaries are just two examples of how our conscious access to sensory information is limited by the culture we grew up in.

SECTION I

BASICS: NEUROSCIENCE AND PSYCHOPHYSICS

This first chapter is a mixture of two kinds of background: an introduction to psychophysical methods, which are the tools that psychologists use to quantify perceptual behaviors, and a review of neuroscience principles, such as action potentials and the distinction between the central nervous system and the peripheral nervous system. The fact that this introductory chapter is a blend of background from two fields is a good analogy for the entire book: our study of Sensation and Perception will rest in equal parts on methods for understanding human behavior and methods for understanding neural networks.

This chapter was created by: Cheryl Olman.

1.

SENSATION VERSUS PERCEPTION

Learning Objectives

Distinguish between sensation and perception

Distinguish between top-down and bottom-up contributions to perception

What does it mean to sense something? Sensory receptors are specialized neurons that respond to specific types of stimuli. When sensory information is detected by a sensory receptor, **sensation** has occurred. For example, light that enters the eye causes chemical changes in cells that line the back of the eye. These cells relay messages, in the form of action potentials (as you learned when studying biopsychology), to the central nervous system. The conversion from sensory stimulus energy to action potential is known as transduction.

You have probably known since elementary school that we have five senses: vision, hearing (audition), smell (olfaction), taste (gustation), and touch (somatosensation). It turns out that this notion of five senses is oversimplified. We also have sensory systems that provide information about balance (the vestibular sense), body position and movement (proprioception and kinesthesia), pain (nociception), and temperature (thermoception).

While our sensory receptors are constantly collecting information from the environment, it is ultimately how we interpret that information that affects how we interact with the world. **Perception** refers to the way sensory information is organized, interpreted, and consciously experienced. Perception involves both **bottom-up** and **top-down** processing. Bottom-up processing refers to the fact that perceptions are built from sensory input. On the other hand, how we interpret those sensations is influenced by our available knowledge, our experiences, and our thoughts. This is called top-down processing.

One way to think of this concept is that sensation is a physical process, whereas perception is psychological. For example, upon walking into a kitchen and smelling the scent of baking cinnamon rolls, the sensation is the scent receptors detecting the odor of cinnamon, but the perception may be “Mmm, this smells like the bread Grandma used to bake when the family gathered for holidays.”

Although our perceptions are built from sensations, not all sensations result in perception. In fact, we often don't perceive stimuli that remain relatively constant over prolonged periods of time. This is known as sensory **adaptation**. Imagine entering a classroom with an old analog clock. Upon first entering the room, you can hear the ticking of the clock; as you begin to engage in conversation with classmates or listen to your professor greet the class, you are no longer aware of the ticking. The clock is still ticking, and that information is still affecting sensory receptors of the auditory system. The fact that you no longer perceive the sound demonstrates sensory adaptation and shows that while closely associated, sensation and perception are different.

There is another factor that affects sensation and perception: **attention**. Attention plays a significant role in determining what is sensed versus what is perceived. Imagine you are at a party full of music, chatter, and laughter. You get involved in an interesting conversation with a friend, and you tune out all the background noise. If someone interrupted you to ask what song had just finished playing, you would probably be unable to answer that question.

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2.

PSYCHOPHYSICAL METHODS

Learning Objectives

Be able to diagnose whether a given experiment measures an absolute threshold, a difference threshold, or is a magnitude estimation experiment

Be able to describe a couple of different methods of estimating a threshold

Know what a subliminal message is

Know Weber's law (also called Weber-Fechner law)

The sensitivity of a given sensory system to the relevant stimuli can be expressed as an absolute threshold. Absolute threshold refers to the minimum amount of stimulus energy that must be present for the stimulus to be detected 50% of the time. Another way to think about this is by asking how dim can a light be or how soft can a sound be and still be detected half of the time. The sensitivity of our sensory receptors can be quite amazing. It has been estimated that on a clear night, the most sensitive sensory cells in the back of the eye can detect a candle flame 30 miles away (Okawa & Sampath, 2007). Under quiet conditions, the hair cells (the receptor cells of the inner ear) can detect the tick of a clock 20 feet away (Galanter, 1962).

It is also possible for us to get messages that are presented below the threshold for conscious awareness—these are called subliminal messages. A stimulus reaches a physiological threshold when it is strong enough to excite sensory receptors and send nerve impulses to the brain: This is an absolute threshold. A message below that threshold is said to be subliminal: We receive it, but we are not consciously aware of it. Over the years there has been a great deal of speculation about the use of subliminal messages in advertising, rock music, and self-help audio programs. Research evidence shows that in laboratory settings, people can process and respond to information outside of awareness. But this does not mean that we obey these messages like zombies; in fact, hidden messages have little effect on behavior outside the laboratory (Kunst-Wilson & Zajonc, 1980; Rensink, 2004; Nelson, 2008; Radel, Sarrazin, Legrain, & Gobancé, 2009; Loersch, Durso, & Petty, 2013).

Methods for estimating thresholds

When we design experiments, we have to decide how we're going to approach a threshold estimation. Here are three common techniques

- **Method of Limits.** The experimenter can increase the stimulus intensity (or intensity difference) until the observer detects the stimulus (or the change). For example, turn up the volume until the observer first detects the sound. This is intuitive, but it is subject to bias — the estimated threshold is likely to be different, for example, if we start high and work down vs. start low and work up.
- **Method of Adjustment.** This is very much like the Method of Limits, except the experimenter gives the observer the knob: “adjust the stimulus until it's very visible” or “adjust the color of the patch until it matches the test patch.”
- **Method of Constant Stimuli.** This is the most

Absolute thresholds are generally measured under incredibly controlled conditions in situations that are optimal for sensitivity. Sometimes, we are more interested in how much difference in stimuli is required to detect a difference between them. This is known as the just noticeable difference (jnd) or difference threshold. Unlike the absolute threshold, the difference threshold changes depending on the stimulus intensity. As an example, imagine yourself in a very dark movie theater. If an audience member were to receive a text message on her cell phone which caused her screen to light up, chances are that many people would notice the change in illumination in the theater. However, if the same thing happened in a brightly lit arena during a basketball game, very few people would notice. The cell phone brightness does not change, but its ability to be detected as a change in illumination varies dramatically between the two contexts. Ernst Weber proposed this theory of change in difference threshold in the 1830s, and it has become known as Weber's law: The difference threshold is a constant fraction of the original stimulus, as the example illustrates.

Weber's law is approximately true for many of our senses — for brightness perception, visual contrast perception, loudness perception, and visual distance estimation, our sensitivity to change decreases as the stimulus gets bigger or stronger. However, there are many senses for which the opposite is true: our sensitivity increases as the stimulus increases. With electric shock, for example, a small increase in the size of the shock is much more noticeable when the shock is large than when it is small. A psychophysical researcher named Stanley Smith Stevens asked people to estimate the magnitude of their sensations for many different kinds of stimuli at different intensities, and then tried to fit lines through the data to predict people's sensory experiences (Stevens, 1967). What he discovered was that most senses could be described by a power law of the form

$$P \propto S^n$$

where P is the perceived magnitude, \propto means “is proportional to”, S is the physical stimulus magnitude, and n is a positive number. If n is greater than 1, then the slope (rate of change of perception) is getting larger as the stimulus gets larger, and sensitivity increases as stimulus intensity increases. A function like this is described as being expansive or supra-linear. If n is less than 1, then the slope decreases as the stimulus gets larger (the function “rolls over”). These sensations are described as being compressive. Weber’s Law is only (approximately) true for compressive (sublinear) functions; Stevens’ Power Law is useful for describing a wider range of senses.

Both Stevens’ Power Law and Weber’s Law are only approximately true. They are useful for describing, in broad strokes, how our perception of a stimulus depends on its intensity or size. They are rarely accurate for describing perception of stimuli that are near the absolute detection threshold. Still, they are useful for describing how people are going to react to normal every-day stimuli.

reliable, but most time-consuming. You decide ahead of time what levels you are going to measure, do each one a fixed number of times, and record % correct (or the number of detections) for each level. If you randomize the order, you can get rid of bias.

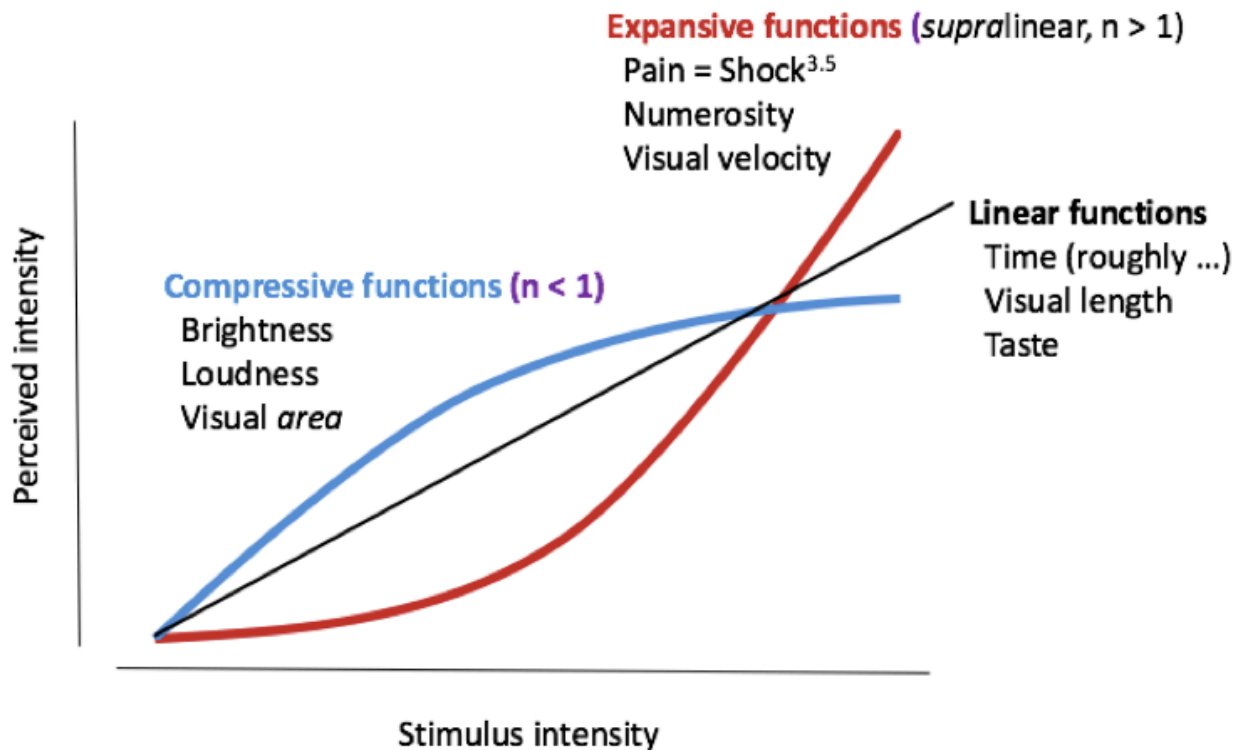


Fig. 2.1. Different sensory systems exhibit different relationships between perceived magnitude and stimulus intensity. Sometimes, it makes the most sense to discount or ignore increases in stimulus intensity above a certain point; compressive sensory modalities with a power-law exponent less than 1 accomplish this. Other times, we need heightened sensitivity to stimuli with increased intensity; expansive sensory modalities described by a power law with exponent greater than 1 accomplish this. Not all perception is non-linear, however: some senses are best described by a linear relationship between stimulus and perception.



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References:

- Galanter, E. (1962). Contemporary Psychophysics. In R. Brown, E. Galanter, E. H. Hess, & G. Mandler (Eds.), *New directions in psychology*. New York, NY: Holt, Rinehart & Winston.
- Kunst-Wilson, W. R., & Zajonc, R. B. (1980). Affective discrimination of stimuli that cannot be recognized. *Science*, 207, 557–558.
- Nelson, M. R. (2008). The hidden persuaders: Then and now. *Journal of Advertising*, 37(1), 113–126.
- Okawa, H., & Sampath, A. P. (2007). Optimization of single-photon response transmission at the rod-to-rod bipolar synapse. *Physiology*, 22, 279–286.
- Radel, R., Sarrazin, P., Legrain, P., & Gobancé, L. (2009). Subliminal priming of motivational orientation in educational settings: Effect on academic performance moderated by mindfulness. *Journal of Research in Personality*, 43(4), 1–18.
- Rensink, R. A. (2004). Visual sensing without seeing. *Psychological Science*, 15, 27–32.
- Stevens, S. S. (1957). On the psychophysical law. *Psychological Review* 64(3):153—181. PMID 13441853

3.

PSYCHOMETRIC FUNCTIONS

Psychometric functions describe stimulus/response relationships.

Learning Objectives

- Understand why many psychometric functions are S-shaped
- Know why maximum performance is rarely 100%
- Know why minimum performance can be anything between 0% and 50%
- Understand what the slope of a psychometric function indicates

Put together, the concepts from the previous section teach us that perception can be quantified. A graph that shows an observer's response as a function of the stimulus level or intensity is called a psychometric function. Psychometric functions can have many shapes, but the most common and most interesting is the S-shaped curve that represents an observer's behavior around a threshold (this works for either absolute or difference thresholds).

Many psychometric functions are S-shaped because the transition from “can't tell” to “can tell” is not instantaneous. Because (neural) responses are variable (both bottom-up and top-down effects have variability), observers don't give the same answer to every stimulus.

- The “ceiling” is the best performance for the most detectable stimuli. Sometimes this is not 100% because people make mistakes in answering (“finger errors”: they hit the wrong button on accident).
- The “floor” is the worst performance. If you're doing a two-alternative forced-choice task, the worst a person can do is 50% (guessing). If you're doing a simple yes/no task (presenting a stimulus on every trial, and the participants says “yes” if they detect it), the floor is 0%.
- The middle is the most interesting part. This is where a person begins to detect the changes, but not all the time.

- You determine the threshold by picking a performance criterion (e.g., 80% correct); the threshold is the stimulus value (intensity or difference) that resulted in criterion performance.

One thing we want to learn from a psychometric function is whether a person's detection (or discrimination) threshold is high because the average neural response to the stimulus is weak or because there is a lot of variability in the responses. Thresholds are higher when response doesn't depend very strongly on stimulus. Thresholds are also higher when responses are highly variable — even a big change isn't reliably detected. The slope of the psychometric function (for a single stimulus level) indicates the level of perceptual noise, or the variability of the responses. So when comparing different population groups, and one tends to have higher thresholds than the other, we can use the slope to tell whether the difference is caused by reduced response amplitude or increased noise (Park, 2017; Reynolds, 2009).

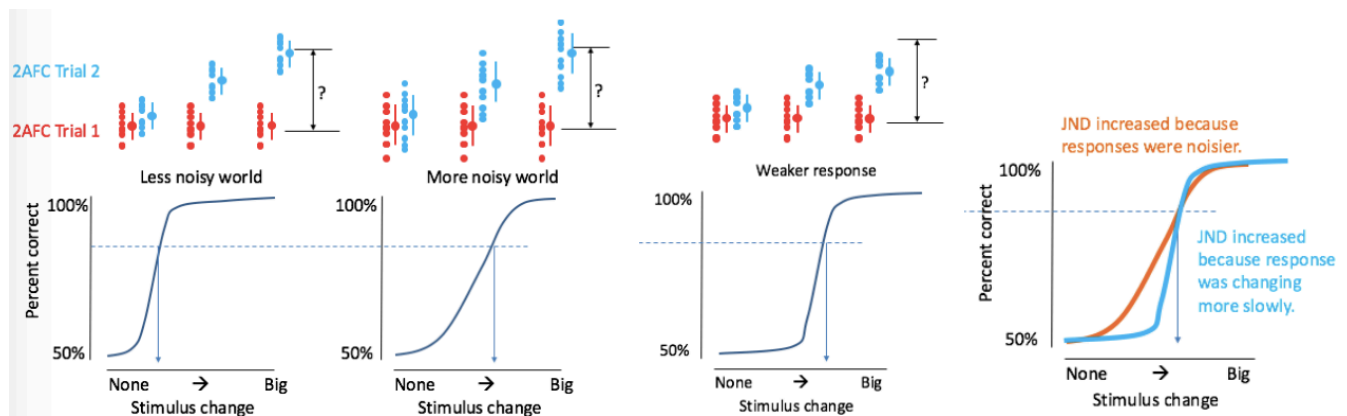


Figure 3.1. Detection thresholds are reached when stimulus-induced response changes are large compared to the variability in the response.

If you would like to learn more here is another creative commons source covering psychometric functions or check out this recorded lecture.



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References:

Park WJ, Schauder KB, Zhang R, Bennetto L, Tadin D (2017). High internal noise and poor external noise filtering characterize perception in autism spectrum disorder. *Sci Rep.*7(1):17584. Quantification of how well people detect patterns in noisy backgrounds tells us that autism is associated with increased internal noise and decreased ability to filter out external noise.

Reynolds, JH and Heeger DJ (2009). The normalization model of attention. *Neuron* 29;61(2):168-85. Studying how the psychometric function shifts when people pay attention to or ignore a stimulus revealed one of the neural mechanisms by which attention improves performance.

NEUROSCIENCE REVIEW

Learning Objectives

Make sure that you're familiar with each of these concepts – this introductory section recaps background vocabulary to support other learning goals.

If you have yet to take a neuroscience class, you will want to read — thoroughly — the very excellent Chapter 12 from the OpenStax Anatomy and Physiology textbook. This Neuroscience section is composed of excerpts from that chapter to highlight just the key concepts most relevant to a study of sensation and perception.

Nervous tissue is composed of two types of cells, neurons and glial cells. Neurons are the primary type of cell that most anyone associates with the nervous system. They are responsible for the computation and communication that the nervous system provides. They are electrically active and release chemical signals to target cells. Glial cells, or glia, are known to play a supporting role for nervous tissue. Ongoing research pursues an expanded role that glial cells might play in signaling, but neurons are still considered the basis of this function. Neurons are important, but without glial support they would not be able to perform their function.

Parts of a Neuron

The main part of a neuron is the cell body, which is also known as the soma (soma = “body”). The cell body contains the nucleus and most of the major organelles. But what makes neurons special is that they have many extensions of their cell membranes, which are generally referred to as processes. Neurons are usually described as having one, and only one, axon—a fiber that emerges from the cell body and projects to target cells. That single axon can branch repeatedly to communicate with many target cells. It is the axon that propagates the nerve impulse, which is communicated to one or more cells. The other processes of the neuron are dendrites, which receive information from other neurons at specialized areas of contact called synapses. The dendrites are usually highly branched processes, providing locations for other neurons to communicate with the cell body.

Information flows through a neuron from the dendrites, across the cell body, and down the axon. This gives the neuron a polarity—meaning that information flows in this one direction.



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Types of Neurons

There are many neurons in the nervous system—a number in the trillions. And there are many different types of neurons. They can be classified by many different criteria. The first way to classify them is by the number of processes attached to the cell body. Using the standard model of neurons, one of these processes is the axon, and the rest are dendrites. Because information flows through the neuron from dendrites or cell bodies toward the axon, these names are based on the neuron's polarity.

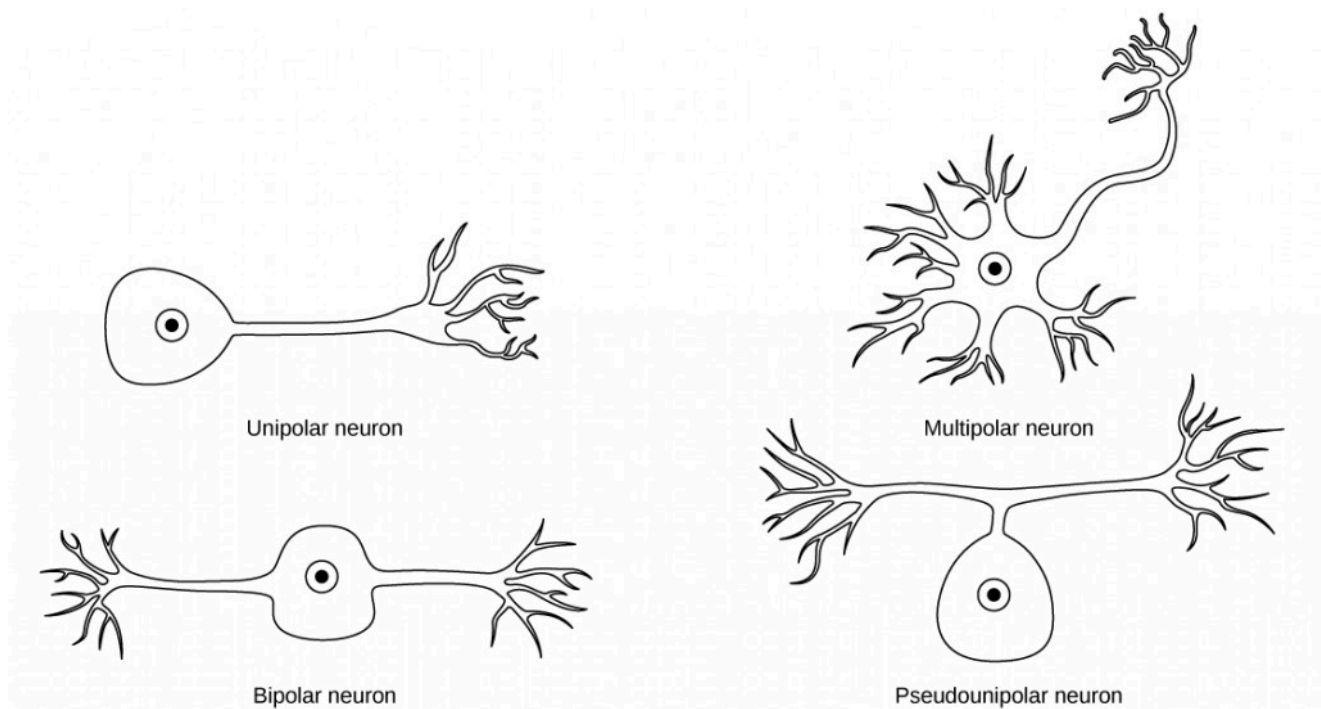


Figure 4.2. Neurons are broadly divided into four main types based on the number and placement of axons: (1) unipolar, (2) bipolar, (3) multipolar, and (4) pseudounipolar (Credit: Figure 16.5, Opentext Concepts of Biology, CC BY 4.0, <https://opentextbc.ca/biology/chapter/16-1-neurons-and-glia-cells/>. No modifications).

Unipolar cells have only one process emerging from the cell. True unipolar cells are only found in invertebrate

animals, so the unipolar cells in humans are more appropriately called “pseudo-unipolar” cells. Invertebrate unipolar cells do not have dendrites. Human unipolar cells have an axon that emerges from the cell body, but it splits so that the axon can extend along a very long distance. At one end of the axon are dendrites, and at the other end, the axon forms synaptic connections with a target. Unipolar cells are exclusively sensory neurons and have two unique characteristics. First, their dendrites are receiving sensory information, sometimes directly from the stimulus itself. Secondly, the cell bodies of unipolar neurons are always found in ganglia. Sensory reception is a peripheral function (those dendrites are in the periphery, perhaps in the skin) so the cell body is in the periphery, though closer to the CNS in a ganglion. The axon projects from the dendrite endings, past the cell body in a ganglion, and into the central nervous system.

Bipolar cells have two processes, which extend from each end of the cell body, opposite to each other. One is the axon and one the dendrite. Bipolar cells are not very common. They are found mainly in the olfactory epithelium (where smell stimuli are sensed), and as part of the retina.

Multipolar neurons are all of the neurons that are not unipolar or bipolar. They have one axon and two or more dendrites (usually many more). With the exception of the unipolar sensory ganglion cells, and the two specific bipolar cells mentioned above, all other neurons are multipolar. Some cutting edge research suggests that certain neurons in the CNS do not conform to the standard model of “one, and only one” axon. Some sources describe a fourth type of neuron, called an anaxonic neuron. The name suggests that it has no axon (an- = “without”), but this is not accurate. Anaxonic neurons are very small, and if you look through a microscope at the standard resolution used in histology (approximately 400X to 1000X total magnification), you will not be able to distinguish any process specifically as an axon or a dendrite. Any of those processes can function as an axon depending on the conditions at any given time. Nevertheless, even if they cannot be easily seen, and one specific process is definitively the axon, these neurons have multiple processes and are therefore multipolar.

Neurons can also be classified on the basis of where they are found, who found them, what they do, or even what chemicals they use to communicate with each other. Some neurons referred to in this section on the nervous system are named on the basis of those sorts of classifications. For example, a multipolar neuron that has a very important role to play in a part of the brain called the cerebellum is known as a Purkinje (commonly pronounced per-KIN-gee) cell. It is named after the anatomist who discovered it (Jan Evangelista Purkinje, 1787–1869).

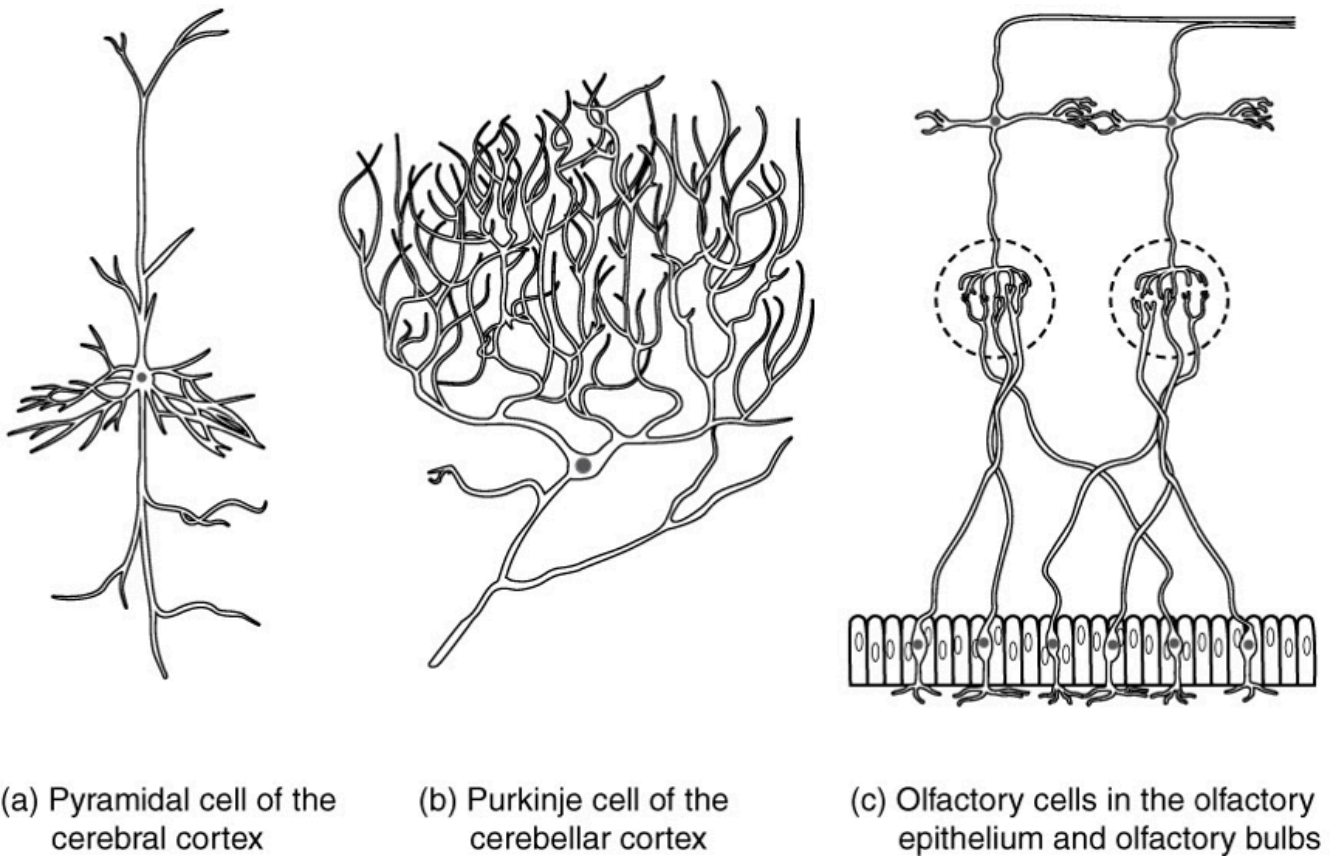


Figure 4.3. Example Neuron Classifications. Three examples of neurons that are classified on the basis of other criteria. (a) The pyramidal cell is a multipolar cell with a cell body that is shaped something like a pyramid. (b) The Purkinje cell in the cerebellum was named after the scientist who originally described it. (c) Olfactory neurons are named for the functional group with which they belong. (Credit: Figure 12.10, OpenStax Anatomy and Physiology)

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OpenStax, Anatomy and Physiology Section 12.2 Nervous Tissue

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5.

THE CENTRAL AND PERIPHERAL NERVOUS SYSTEM

Learning Objectives

Understand the layout of the CNS and PNS to support future learning about different sensory systems

Know which nervous system is most likely to heal after injury

The nervous system can be divided into two major regions: the peripheral nervous system and the central nervous system. The peripheral nervous system consists of everything else. It has been compared to the power plant of the nervous system. It is like a system that collects information and sends commands. The PNS is also divided into two systems, the autonomic nervous system and the sensory-somatic nervous system. Both will be talked about in more depth later on. The central nervous system also known as the CNS consists of the brain and spinal cord. The spinal cord transmits information for the brain to process. The brain is contained within the cranial cavity of the skull, and the spinal cord is contained within the vertebral cavity of the vertebral column. It is a bit of an oversimplification to say that the CNS is what is inside these two cavities and the peripheral nervous system is outside of them, but that is one way to start to think about it. In actuality, there are some elements of the peripheral nervous system that are within the cranial or vertebral cavities. The peripheral nervous system is so named because it is on the periphery—meaning beyond the brain and spinal cord. Depending on different aspects of the nervous system, the dividing line between central and peripheral is not necessarily universal.

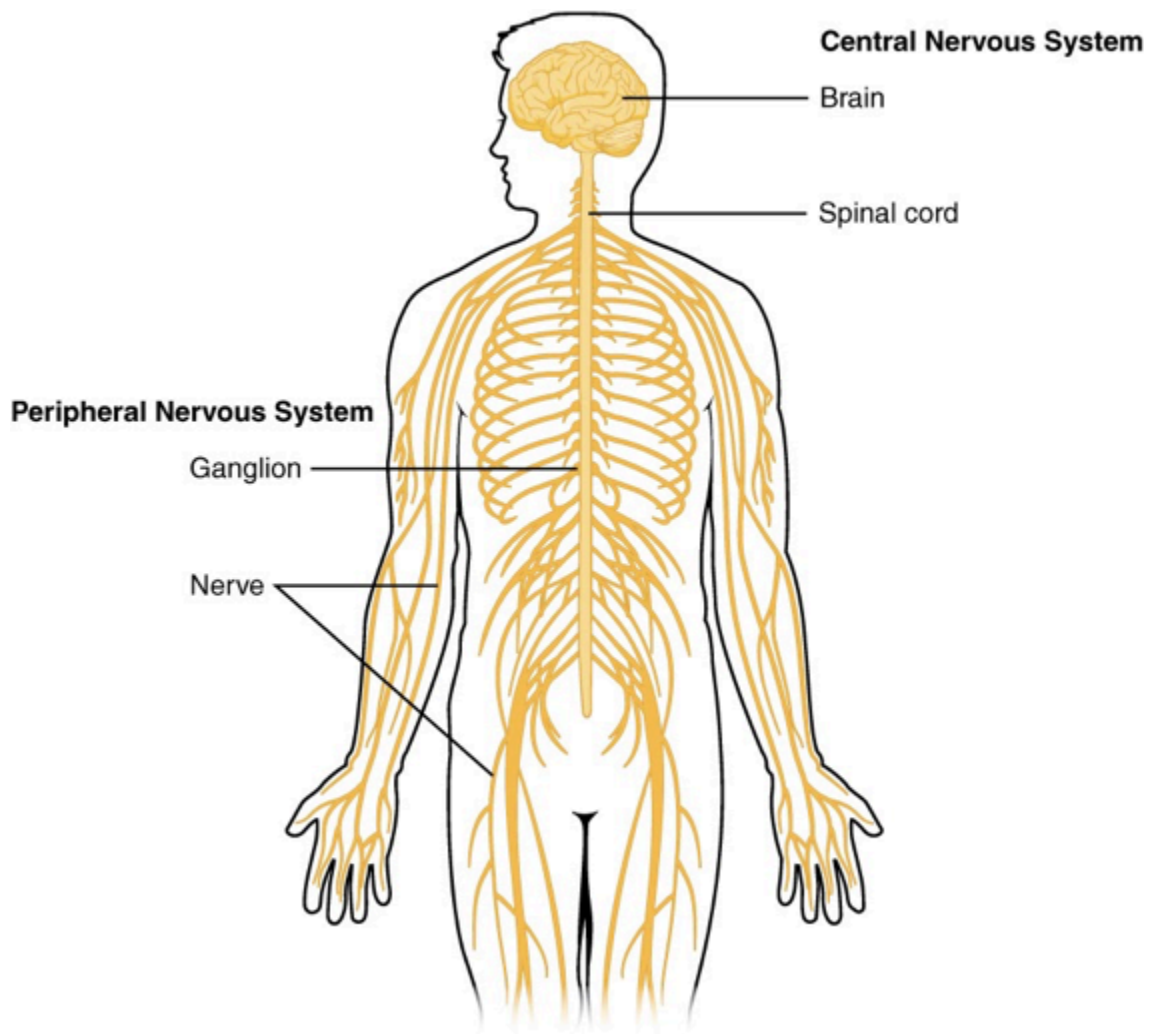


Figure 5.1. Central and Peripheral Nervous System The structures of the PNS are referred to as ganglia and nerves, which can be seen as distinct structures. The equivalent structures in the CNS are not obvious from this overall perspective and are best examined in prepared tissue under the microscope. (credit: OpenStax Anatomy and Physiology, chapter 8.3).

As a general rule, neurons in the central nervous system cannot regenerate or grow back after injury. The most obvious example of this is paralysis after spinal cord injury. Peripheral neurons, on the other hand, often do regrow after injury. For example, after a deep cut on the skin, the area around the cut is often numb for a long time after the cut looks like it has healed. But slowly — after several weeks or months — the sensation comes back. It is not yet known why neural regeneration is so different in the central vs. peripheral nervous system, and many research groups who want to help patients with spinal cord injuries or strokes are trying to discover what inhibits growth in the CNS and encourages growth in the PNS (Tsintou, 2020).

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References:

Tsintou, M., Dalamagkas, K., Makris, N. (2020). Taking central nervous system regenerative therapies to the clinic: curing rodents versus nonhuman primates versus humans. *Neural Regen Res.* 2020 Mar;15(3):425-437. doi: 10.4103/1673-5374.266048.

6.

ACTION POTENTIALS

Learning Objectives

Distinguish between action potentials and graded potentials

Understand that action potentials are all-or-nothing events — size doesn't matter, rate does

Understand that both the rate and timing of action potentials have a random component

The Membrane Potential

The electrical state of the cell membrane can have several variations. These are all variations in the membrane potential. A potential is a distribution of charge across the cell membrane, measured in millivolts (mV). The standard is to compare the inside of the cell relative to the outside, so the membrane potential is a value representing the charge on the intracellular side of the membrane based on the outside being zero, relatively speaking.

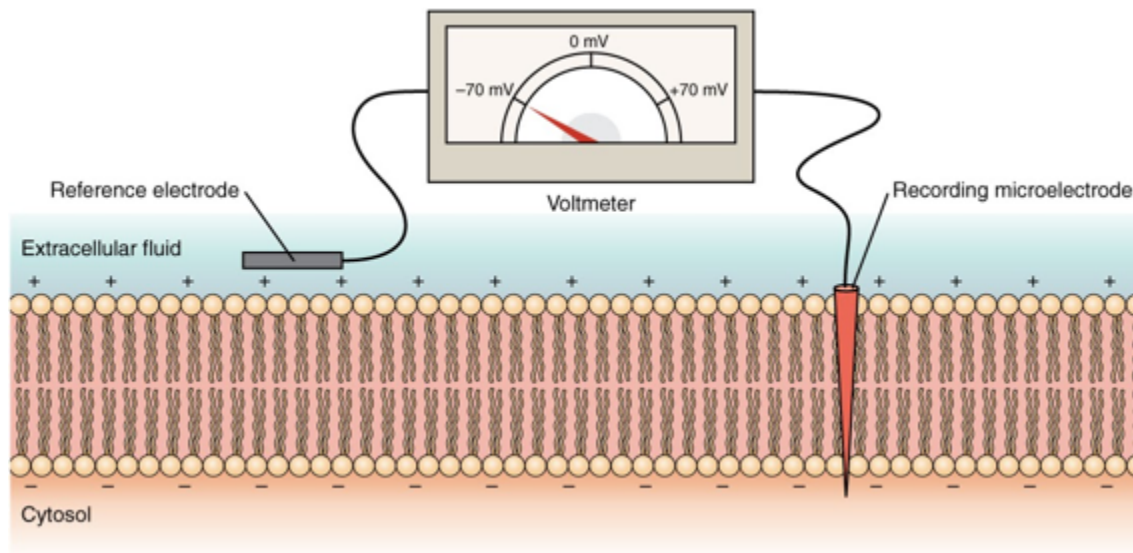


Figure 6.1. Measuring Charge across a Membrane with a Voltmeter A recording electrode is inserted into the cell and a reference electrode is outside the cell. By comparing the charge measured by these two electrodes, the transmembrane voltage is determined. It is conventional to express that value for the cytosol relative to the outside. (credit: OpenStax Anatomy and Physiology, <https://openstax.org/books/anatomy-and-physiology/pages/12-4-the-action-potential>)

Action Potentials vs. Graded Potentials

Resting membrane potential describes the steady-state of the cell, which is a dynamic process that is balanced by ion leakage and ion pumping. Without any outside influence, it will not change. To get an electrical signal started, the membrane potential has to change.

Each neuron, and each section of each neuron, has a specific distribution of ion channels in the cell membrane that determines how the membrane potential can change. Axons have the right distributions and types of Na^+ and K^+ channels to support action potentials, which are all-or-nothing events (Figure X). In dendrites, on the other hand, and in specialized sensory neurons like the photoreceptors in the retina, graded potentials happen instead of action potentials (Figure X). Unlike action potentials, graded potentials can be different sizes and can be positive or negative. Therefore, graded potentials are analog signals while action potentials are digital signals.

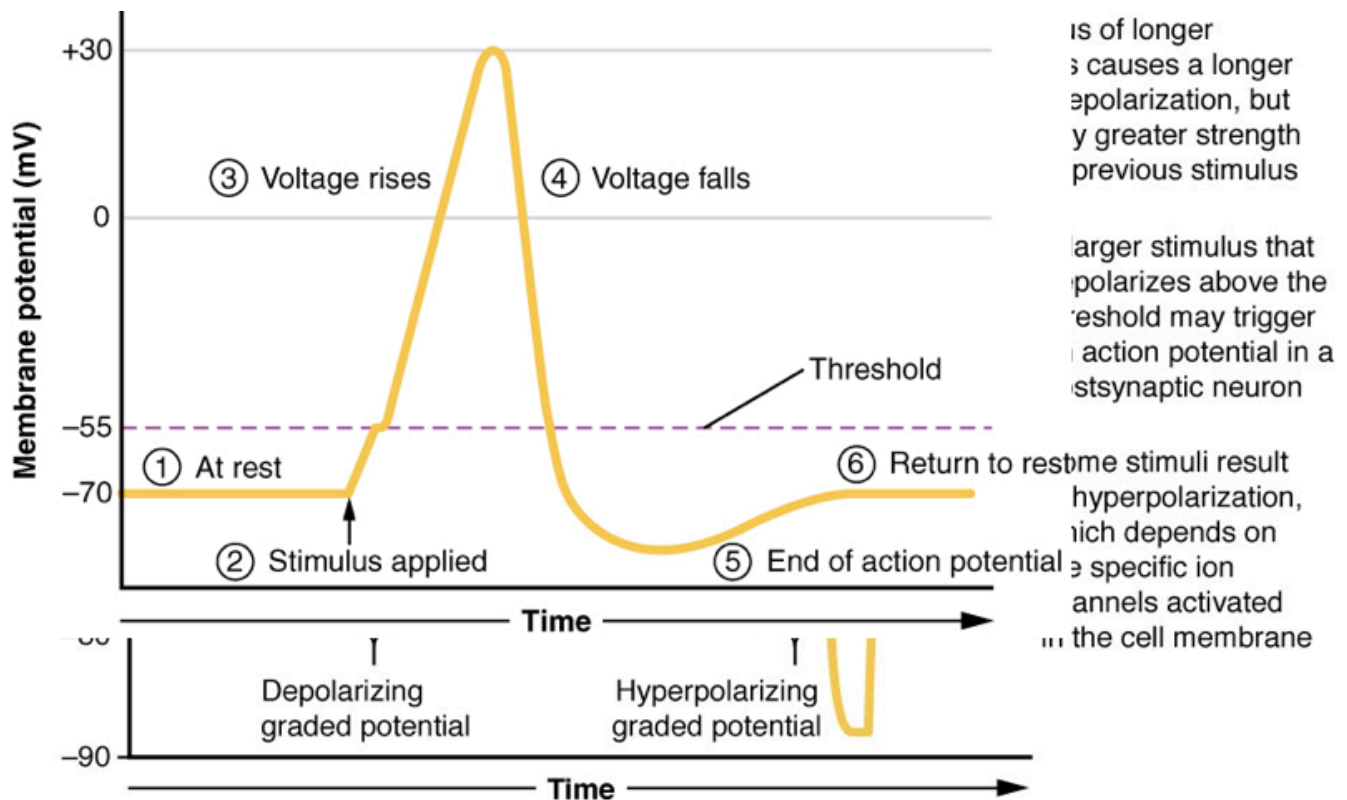


Figure 6.2. Stages of an Action Potential Plotting voltage measured across the cell membrane against time, the events of the action potential can be related to specific changes in the membrane voltage. (1) At rest, the membrane voltage is -70 mV. (2) The membrane begins to depolarize when an external stimulus is applied. (3) The membrane voltage begins a rapid rise toward +30 mV. (4) The membrane voltage starts to return to a negative value. (5) Repolarization continues past the resting membrane voltage, resulting in hyperpolarization. (6) The membrane voltage returns to the resting value shortly after hyperpolarization. (credit: OpenStax Anatomy and Physiology, Section 12.4 The Action Potential)

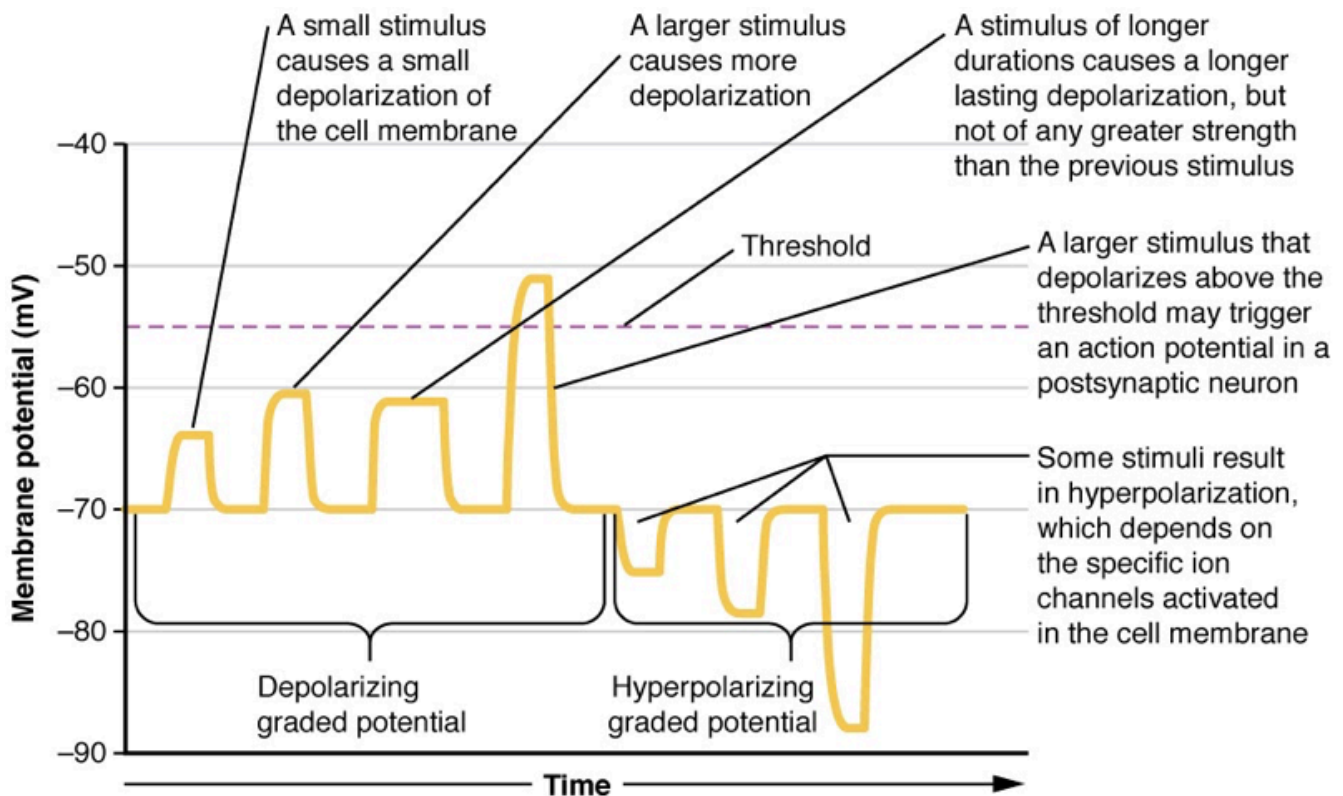


Figure 6.3. Graded potentials are temporary changes in the membrane voltage, the characteristics of which depend on the size of the stimulus. Some types of stimuli cause depolarization of the membrane, whereas others cause hyperpolarization. It depends on the specific ion channels that are activated in the cell membrane. (credit: OpenStax Anatomy and Physiology, Section 12.5 Communication Between Neurons)

For the unipolar cells of sensory neurons—both those with free nerve endings and those within encapsulations—graded potentials develop in the dendrites that influence the generation of an action potential in the axon of the same cell. This is called a generator potential. For other sensory receptor cells, such as taste cells or photoreceptors of the retina, graded potentials in their membranes result in the release of neurotransmitters at synapses with sensory neurons. This is called a receptor potential.

A postsynaptic potential (PSP) is the graded potential in the dendrites of a neuron that is receiving synapses from other cells. Postsynaptic potentials can be depolarizing or hyperpolarizing. Depolarization in a postsynaptic potential is called an excitatory postsynaptic potential (EPSP) because it causes the membrane potential to move toward threshold. Hyperpolarization in a postsynaptic potential is an inhibitory postsynaptic potential (IPSP) because it causes the membrane potential to move away from threshold.

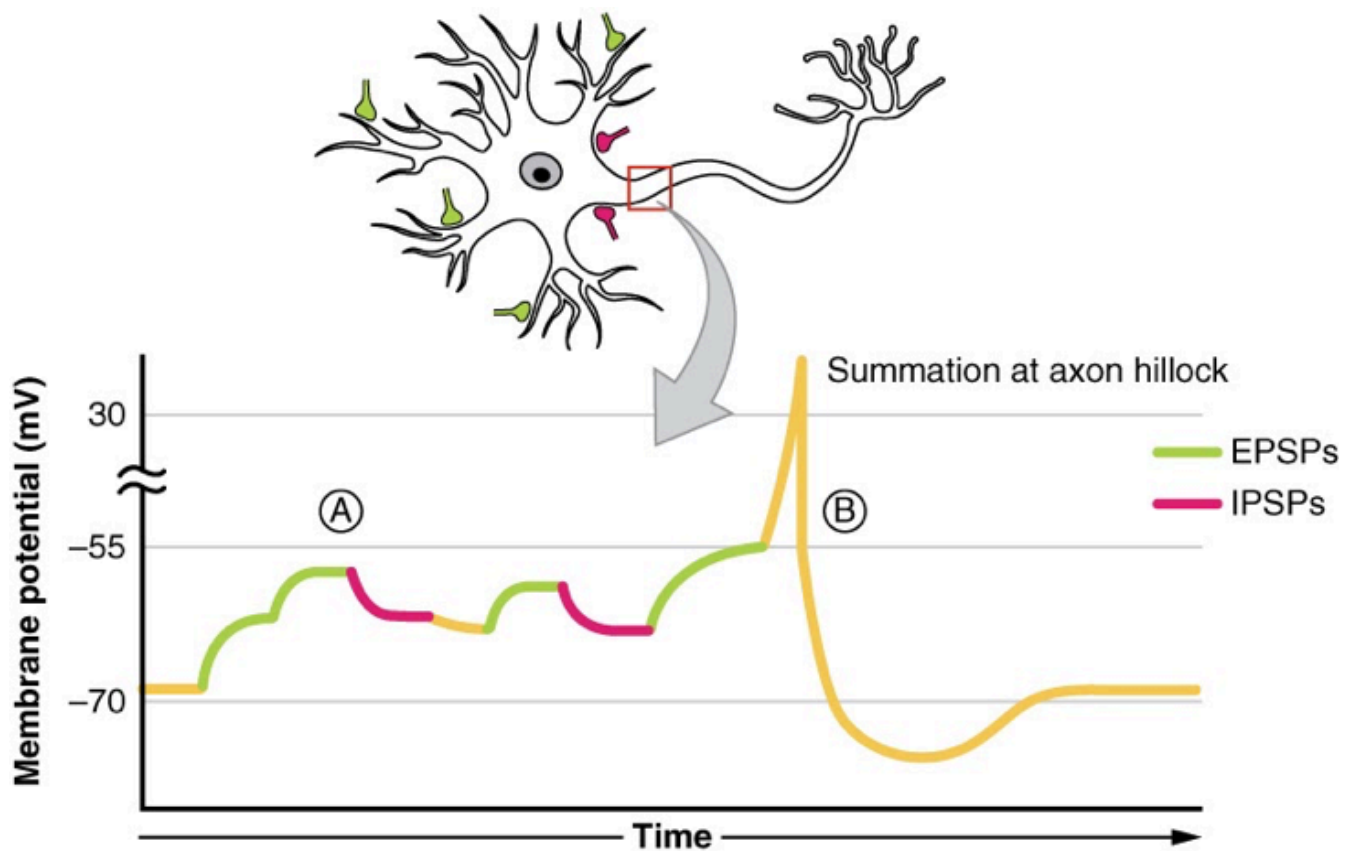


Figure 6.4. Postsynaptic Potential Summation. The result of summation of postsynaptic potentials is the overall change in the membrane potential. At time A, several different excitatory postsynaptic potentials add up to a large depolarization. At time B, a mix of excitatory and inhibitory postsynaptic potentials result in a different end result for the membrane potential.

Variability in Action Potential Rates and Times

Given the complex mechanisms that generate action potentials, it is no wonder that there is a bit of randomness in the process. A sensory neuron will not respond in the exact same way every time a stimulus is present. On average, a more intense stimulus will result in stronger graded potentials and, after summation, a higher rate of action potentials. But on each trial — each time the stimulus is applied — the rate will not be exactly the same, and the timing of all the spikes in the spike train will not be the same. Sometimes, there are systematic differences. For example, neurons adapt to stimuli, and respond more weakly after repeated trials. However, on top of this, there are also random differences. In order to support perception, neural networks must evolve efficient strategies for making perceptual decisions even in the presence of sensory noise.

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7.

SYNAPSES

Learning Objectives

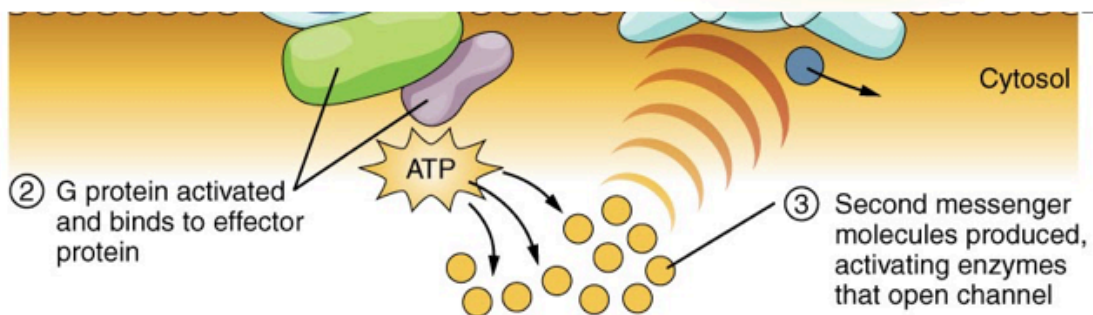
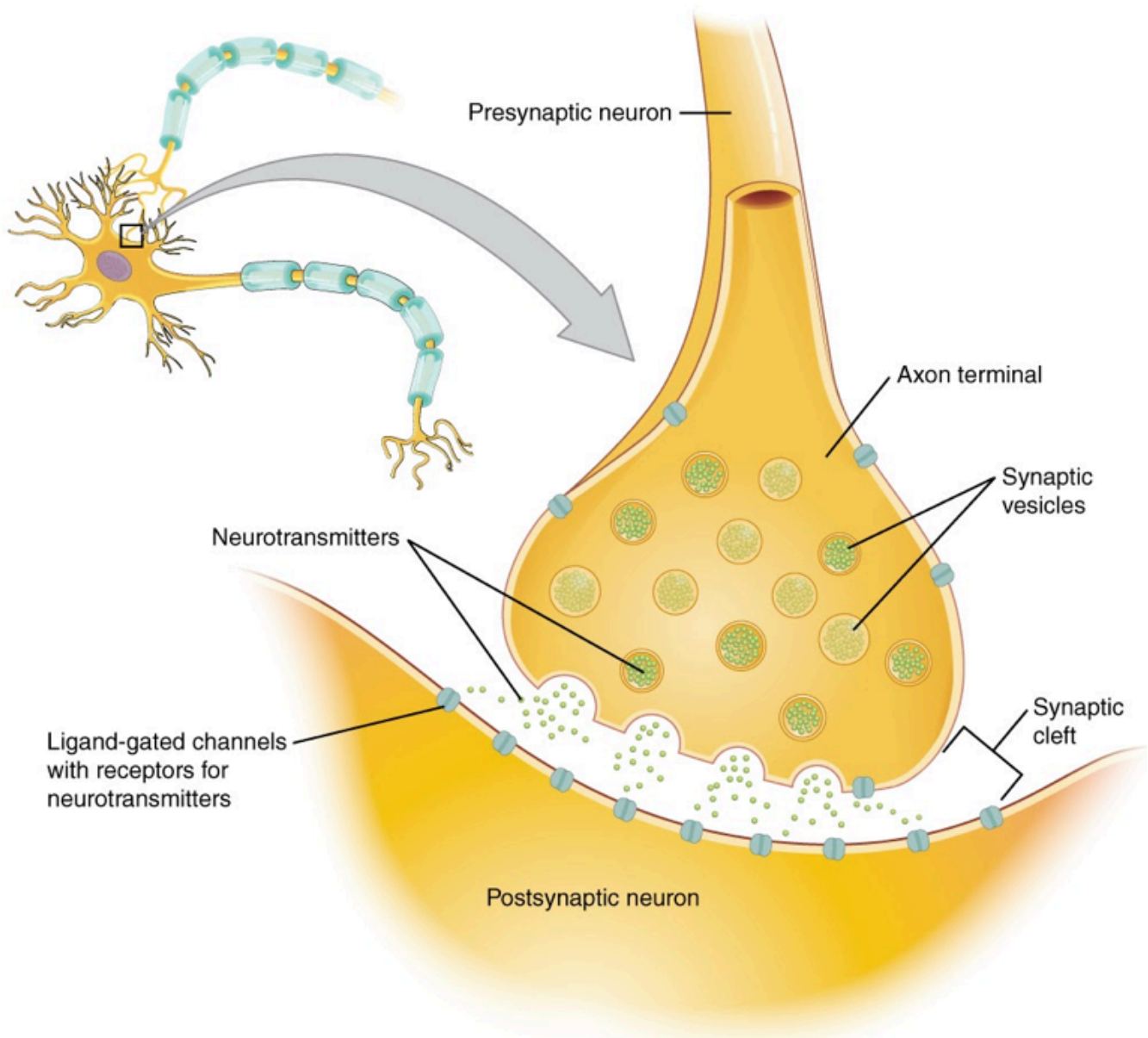
Know that a synapse is a gap between two neurons where communication happens chemically

Understand that the effect of a given neurotransmitter is determined by the post-synaptic receptors

The synapse or “gap” is the place where information is transmitted from one neuron to another. Synapses usually form between axon terminals and dendritic spines, but this is not universally true. There are also axon-to-axon, dendrite-to-dendrite, and axon-to-cell body synapses. The neuron transmitting the signal is called the presynaptic neuron, and the neuron receiving the signal is called the postsynaptic neuron. Note that these designations are relative to a particular synapse—most neurons are both presynaptic and postsynaptic. There are two types of synapses: chemical and electrical.

When an action potential reaches the axon terminals, voltage-gated Ca^{2+} channels in the membrane of the synaptic end bulb open. The concentration of Ca^{2+} increases inside the end bulb, and the Ca^{2+} ion associates with proteins in the outer surface of neurotransmitter vesicles. The Ca^{2+} facilitates the merging of the vesicle with the presynaptic membrane so that the neurotransmitter is released (a process called exocytosis) into the small gap between the cells, known as the synaptic cleft.

Once in the synaptic cleft, the neurotransmitter diffuses the short distance to the postsynaptic membrane and can interact with neurotransmitter receptors. Receptors are specific for the neurotransmitter, and the two fit together like a key and lock. One neurotransmitter binds to its receptor and will not bind to receptors for other neurotransmitters, making the binding a specific chemical event.



(b) Indirect activation involves a prolonged response, amplified over time

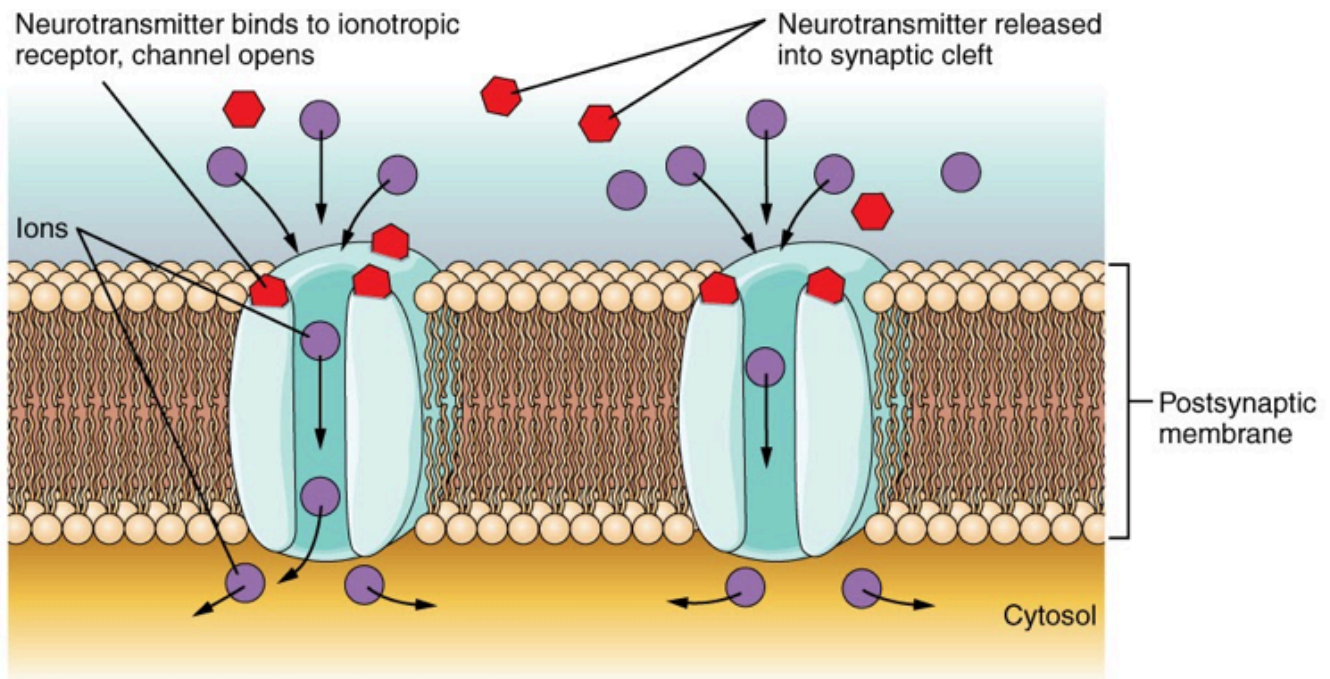
Figure 7.1. The Synapse The synapse is a connection between a neuron and its target cell (which is not necessarily a neuron). The presynaptic element is the synaptic end bulb of the axon where Ca^{2+} enters the bulb to cause vesicle fusion and neurotransmitter release. The neurotransmitter diffuses across the synaptic cleft to bind to its receptor. The neurotransmitter is cleared from the synapse either by enzymatic degradation, neuronal reuptake, or glial reuptake. (Credit: OpenStax Anatomy and Physiology, Section 12.5

Communication Between Neurons)

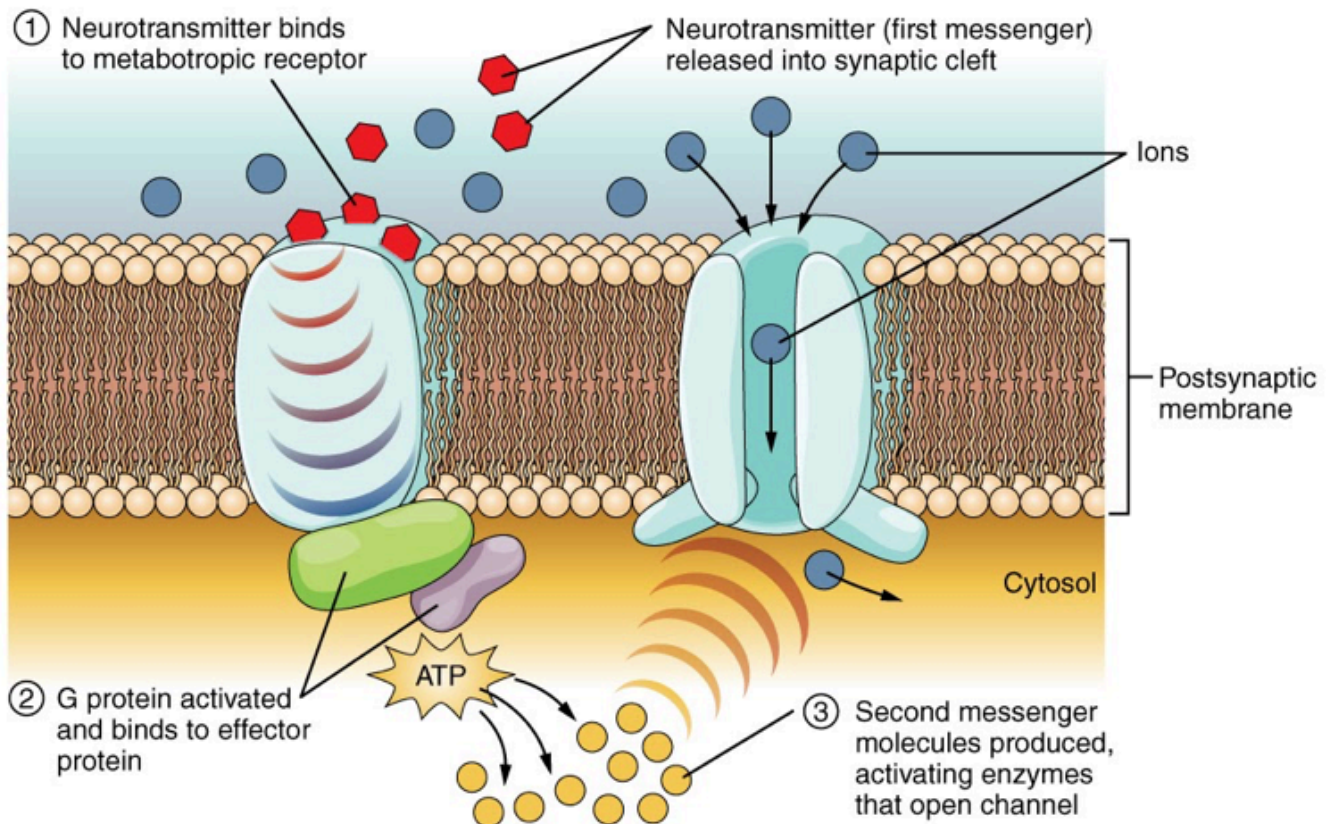
The effect of a neurotransmitter on the postsynaptic element is entirely dependent on the receptor protein. First, if there is no receptor protein in the membrane of the postsynaptic element, then the neurotransmitter has no effect. The depolarizing or hyperpolarizing effect is also dependent on the receptor. For example, when acetylcholine binds to the nicotinic receptor, the postsynaptic cell is depolarized. This is because the receptor is a cation channel and positively charged Na^+ will rush into the cell. However, when acetylcholine binds to the muscarinic receptor, of which there are several variants, it might cause depolarization or hyperpolarization of the target cell.

The amino acid neurotransmitters, glutamate, glycine, and GABA, are almost exclusively associated with just one effect. Glutamate is considered an excitatory amino acid, but only because Glu receptors in the adult cause depolarization of the postsynaptic cell. Glycine and GABA are considered inhibitory amino acids, again because their receptors cause hyperpolarization.

Glutamate is interesting because it can cause excitation by activating ionotropic receptors, but it can also activate metabotropic receptors that act on the post-synaptic cell in ways other than immediately changing the membrane potential. Ionotropic receptors are fast-acting and have (relatively) short-lived consequences. Metabotropic receptors, on the other hand, can start intracellular processes with effects as far-reaching as changing the regulation of genes. Glutamate's ionotropic receptors are NMDA, AMPA and kainate (named after the other molecules that also activate them). Glutamate's three metabotropic receptors are called mGluRs and can either increase or decrease the excitability of the post-synaptic cell.



(a) Direct activation brings about immediate response



(b) Indirect activation involves a prolonged response, amplified over time

Figure 7.2. Receptor Types (a) An ionotropic receptor is a channel that opens when the neurotransmitter binds to it. (b) A metabotropic receptor is a complex that causes metabolic changes in the cell when the neurotransmitter binds to it (1). After binding, the G protein hydrolyzes GTP and moves to the effector

protein (2). When the G protein contacts the effector protein, a second messenger is generated, such as cAMP (3). The second messenger can then go on to cause changes in the neuron, such as opening or closing ion channels, metabolic changes, and changes in gene transcription. (Credit: OpenStax Anatomy and Physiology, Section 12.5 Communication Between Neurons)

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8.

CONDUCTION VELOCITY AND MYELIN

Learning Objectives

Understand what myelin is

Understand how myelin increases conduction velocity

Myelin

The insulation for axons in the nervous system is provided by glial cells, oligodendrocytes in the CNS, and Schwann cells in the PNS. Whereas the manner in which either cell is associated with the axon segment, or segments, that it insulates is different, the means of myelinating an axon segment is mostly the same in the two situations. Myelin is a lipid-rich sheath that surrounds the axon and by doing so creates a myelin sheath that facilitates the transmission of electrical signals along the axon. The lipids are essentially the phospholipids of the glial cell membrane. Myelin, however, is more than just the membrane of the glial cell. It also includes important proteins that are integral to that membrane. Some of the proteins help to hold the layers of the glial cell membrane closely together.

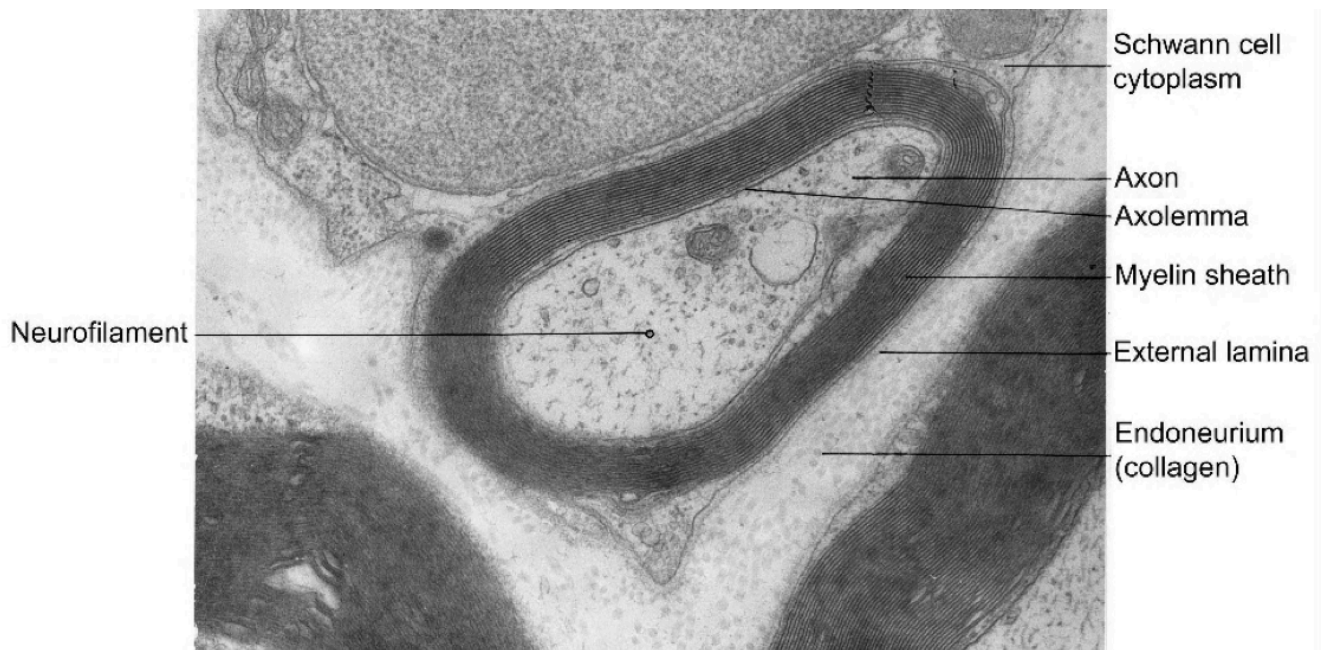


Figure 8.1. Cross-section of a myelinated axon. Myelinating glia wrap several layers of cell membrane around the cell membrane of an axon segment. A single Schwann cell insulates a segment of a peripheral nerve, whereas in the CNS, an oligodendrocyte may provide insulation for a few separate axon segments. EM $\times 1,460,000$. Visit Webscope for a chance to zoom interactively on the image and see more detail. (Micrograph provided by the Regents of University of Michigan Medical School \copyright 2012)

Conduction velocity

Myelination improves the conduction velocity — the speed with which action potentials travel — in axons. Axon diameter also affects conduction velocity: fatter axons carry action potentials faster. So there are two factors that determine how fast an action potential travels down an axon: diameter and myelination (Suzuki, 2010). Axon diameter and myelination are correlated: the skinniest axons (< 1 micron in diameter) are also unmyelinated (often called C-fibers; a good example is found in the neurons that carry pain and temperature information from the skin to the spinal cord). They carry action potentials the most slowly, about 1 meter/second (walking speed). Axons fatter than 1 mm in diameter are generally myelinated. The fattest myelinated axons can carry action potentials faster than 100 m/s — race car speeds! Mechanical (pressure) sensors in our skins and motor neurons have fat, myelinated axons so they can do their jobs rapidly.

Disorders of nervous tissue

Several diseases can result from the demyelination of axons. The causes of these diseases are not

the same; some have genetic causes, some are caused by pathogens, and others are the result of autoimmune disorders. Though the causes are varied, the results are largely similar. The myelin insulation of axons is compromised, making electrical signaling slower.

Multiple sclerosis (MS) is one such disease. It is an example of an autoimmune disease. The antibodies produced by lymphocytes (a type of white blood cell) mark myelin as something that should not be in the body. This causes inflammation and the destruction of the myelin in the central nervous system. As the insulation around the axons is destroyed by the disease, scarring becomes obvious. This is where the name of the disease comes from; sclerosis means hardening of tissue, which is what a scar is. Multiple scars are found in the white matter of the brain and spinal cord. The symptoms of MS include both somatic and autonomic deficits. Control of the musculature is compromised, as is control of organs such as the bladder.

Guillain-Barré (pronounced gee-YAN bah-RAY) syndrome is an example of a demyelinating disease of the peripheral nervous system. It is also the result of an autoimmune reaction, but the inflammation is in peripheral nerves. Sensory symptoms or motor deficits are common, and autonomic failures can lead to changes in the heart rhythm or a drop in blood pressure, especially when standing, which causes dizziness.

Exercises

1. Conduction Velocity and Myelin Review Quiz
 - A. What type of cell provides insulation for axons in the nervous system?
 - B. Astrocytes
 - C. Glial Cells
 - D. Ependymal Cell
 - E. Microglia
2. Match the specific cell with the nervous system that it insulates.

A. CNS	__ Schwann Cells
B. PNS	__ Oligodendrocytes

3. What facilitates the transmission of electrical signals along the axon?
 - A. Myelin Sheath
 - B. Axolemma
 - C. Glial Cell
 - D. Neurofilament
4. What type of structure is responsible for holding the layers of the Glial Cell membrane closely together?
 - A. Nucleus
 - B. Proteins
 - C. Ribosomes
 - D. Cell Membrane
5. As axon diameter increases, conduction velocity...
 - A. Decreases
 - B. Increases
 - C. No Change
6. Axon diameter and myelination have what type of relationship?
 - A. Inverse
 - B. Direct
7. In people who have Multiple Sclerosis, what type of white blood cell produces antibodies that mark myelin as something bad?
 - A. Monocytes
 - B. Neutrophils
 - C. Basophils
 - D. Lymphocytes
8. Match the disease with the nervous system that it affects
 - A. Guillan-Barre Syndrome __PNS
 - B. Multiple Sclerosis (MS) __CNS

Answer Key:

1. B
2. A is Oligodendrocytes; B is Schwann Cells
3. A
4. B
5. B
6. B

7. D

8. A is PNS; B is CNS

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Adapted by: Cheryl Olman

References:

Susuki, K. (2010) Myelin: A Specialized Membrane for Cell Communication. *Nature Education* 3(9):59.

9.

ACTIVE LEARNING EXERCISE - PSYCHOPHYSICS

This Google slide show has instructions for completing an experiment that demonstrates how our sensitivity to change depends on how strong the original stimulus is.



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SECTION II

SOMATOSENSATION

Somatosensation is the sensory experience of one's body. The most obvious sensory experiences are the sense of touch (e.g., pressure, warmth, vibration) and pain (burns, cuts, pinches). More subtle senses are proprioceptive senses — the sense of where parts of our body are in the world and relative to each other. Proprioception will be addressed in the next chapter, so this chapter will just address the senses of touch and pain.

This chapter was created by:

Khadija Abdi, Andrew Barnard, Cale Berkoff, Kimberly Cardinal, Lucinda Carter, Annie Chen, Devyn Compisi, Stacey Dai, Yizi Deng, Tu Do, Shawn George, Safiya Haille, Hanna Hoyt, Zuohan Huang, and Hannah Moser

10.

OVERVIEW OF SOMATOSENSATION

Learning Objectives

Somatosensation includes: touch (mechanical and thermal), pain (mechanical, thermal, chemical) & proprioception (sense of self — the topic of the next chapter).

Know the primary categories of cutaneous senses: Mechanical, Thermal, Noxious.

Somatosensation (Touch)

Somatosensation is considered a general sense, as opposed to the special senses discussed in this section. Somatosensation is the group of sensory modalities that are associated with touch, proprioception, and interoception. These modalities include pressure, vibration, light touch, tickle, itch, temperature, pain, proprioception, and kinesthesia. This means that its receptors are not associated with a specialized organ, but are instead spread throughout the body in a variety of organs (Fig.2.1.1). Many of the somatosensory receptors are located in the skin, but receptors are also found in muscles, tendons, joint capsules, ligaments, and in the walls of visceral organs.

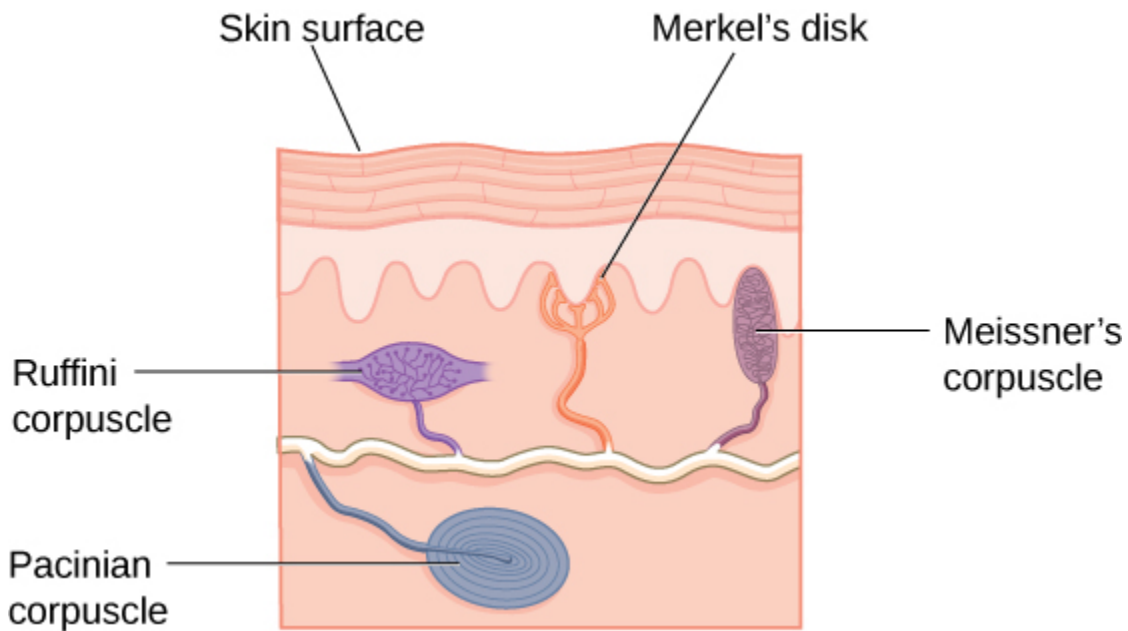


Figure 2.1. Sensory Receptors. There are many types of sensory receptors located in the skin, each attuned to specific touch-related stimuli. (Provided by: Rice University. License: CC-BY 4.0.)

Two types of somatosensory signals that are transduced by free nerve endings are pain and temperature. These two modalities use thermoreceptors and nociceptors to transduce temperature and pain stimuli, respectively. Temperature receptors are stimulated when local temperatures differ from body temperature. Some thermoreceptors are sensitive to just cold and others to just heat. Nociception is the sensation of potentially damaging stimuli. Mechanical, chemical, or thermal stimuli beyond a set threshold will elicit painful sensations. Stressed or damaged tissues release chemicals that activate receptor proteins in the nociceptors. For example, the sensation of heat associated with spicy foods involves capsaicin, the active molecule in hot peppers. Capsaicin molecules bind to a transmembrane ion channel in nociceptors that is sensitive to temperatures above 37°C. The dynamics of capsaicin binding with this transmembrane ion channel is unusual in that the molecule remains bound for a long time. Because of this, it will decrease the ability of other stimuli to elicit pain sensations through the activated nociceptor. For this reason, capsaicin can be used as a topical analgesic, such as in products such as Icy Hot™.

If you drag your finger across a textured surface, the skin of your finger will vibrate. Such low frequency vibrations are sensed by mechanoreceptors called Merkel cells (figure x), also known as type I cutaneous mechanoreceptors. Merkel cells are located in the stratum basale of the epidermis. Deep pressure and vibration is transduced by lamellated (Pacinian) corpuscles, which are receptors with encapsulated endings found deep in the dermis, or subcutaneous tissue. Light touch is transduced by the encapsulated endings known as tactile (Meissner) corpuscles. Follicles are also wrapped in a plexus of nerve endings known as the hair follicle plexus. These nerve endings detect the movement of hair at the surface of the skin, such as when an insect may be

walking along the skin. Stretching of the skin is transduced by stretch receptors known as bulbous corpuscles. Bulbous corpuscles are also known as Ruffini corpuscles, or type II cutaneous mechanoreceptors.

Other somatosensory receptors are found in the joints and muscles. Stretch receptors monitor the stretching of tendons, muscles, and the components of joints. For example, have you ever stretched your muscles before or after exercise and noticed that you can only stretch so far before your muscles spasm back to a less stretched state? This spasm is a reflex that is initiated by stretch receptors to avoid muscle tearing. Such stretch receptors can also prevent over-contraction of a muscle. In skeletal muscle tissue, these stretch receptors are called muscle spindles. Golgi tendon organs similarly transduce the stretch levels of tendons. Bulbous corpuscles are also present in joint capsules, where they measure stretch in the components of the skeletal system within the joint.

Most of your non-proprioceptive somatosensory neurons are cutaneous sensory neurons in your skin. They fall into 3 categories: Mechanical, Thermal, Noxious. Most cutaneous receptors are pseudo-unipolar neurons, with cell bodies in the dorsal root ganglia. The dorsal root ganglia (singular: ganglion; plural: ganglia) are lumps of nervous tissue next to the spinal cord that house the cell bodies of somatosensory neurons. To review the different types of receptors, you can watch this seven minute Khan Academy video linked here and included below.



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11.

THERMAL RECEPTORS

Learning Objectives

Know the difference between thermal sensation and thermal nociception.

Know that thermal sensation is carried on thin, unmyelinated axons.

Understand what it means that thermal receptors are not uniformly distributed

Thermal touch refers to the perception of temperature of objects in contact with the skin. When the hand makes contact with an object, the temperatures of the object and the skin change at a rate that is determined by the thermal properties of the object and skin and their initial temperatures. On the basis of these changes in temperature, people can identify the material composition of objects, for example, whether the object is made from copper or wood. These sensations, however, are not the same as the feeling of being burned by extreme heat (boiling water), or extreme cold (touching dry ice). This experience of pain in relation to temperature is called thermal nociception and involves a different set of distributed receptors.

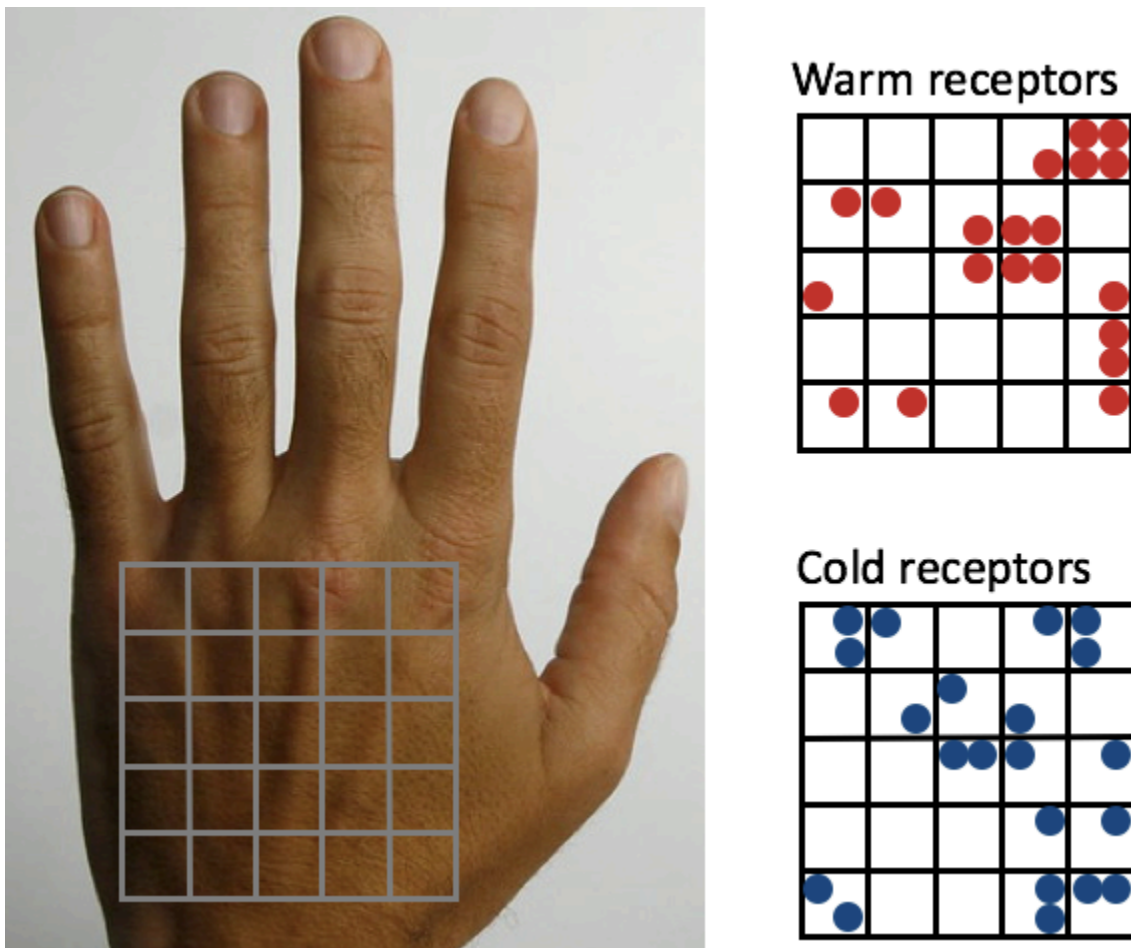


Figure 2.2. Thermal Receptors. Thermal receptors are sparsely distributed in the skin. Some regions of the skin have no thermal sensation for non-damaging (non-noxious) stimuli. (credit: Nate Steiner. Provided by: Flickr. License: CC0 1.0. Adapted by: Cheryl A. Olman.)

The number and density of thermoreceptors in the skin has been measured by placing small warm and cold stimulators on the skin and recording the sites at which a person detects a change in temperature. The locations at which a thermal stimulus is detected are known as warm and cold spots and are assumed to mark the receptive fields of underlying thermoreceptors (Fig.2.2.1). Warm and cold spots are only a few millimeters in diameter, and are distributed independently. There are more cold spots than warm spots, and the density of spots varies across the body. For example, on the forearm it is estimated that there are approximately 7 cold spots and 0.24 warm spots per 100 mm². In addition to differences in the distribution of cold and warm thermoreceptors across the skin surface, the two types of receptor differ with respect to the conduction velocities of the afferent fibers that convey information from the receptor to the central nervous system. Cold afferent fibers are myelinated and so are much faster than unmyelinated warm afferent fibers with conduction velocities of 10-20 m/s as compared to 1-2 m/s for warm fibers. As would be expected from these differences in conduction velocities, the time to respond to a cold stimulus is significantly shorter than that for a warm

stimulus. Additionally, the Khan Academy video linked here and included below provides an additional explanation about thermal receptors and thermal nociceptors.



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Exercises



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12.

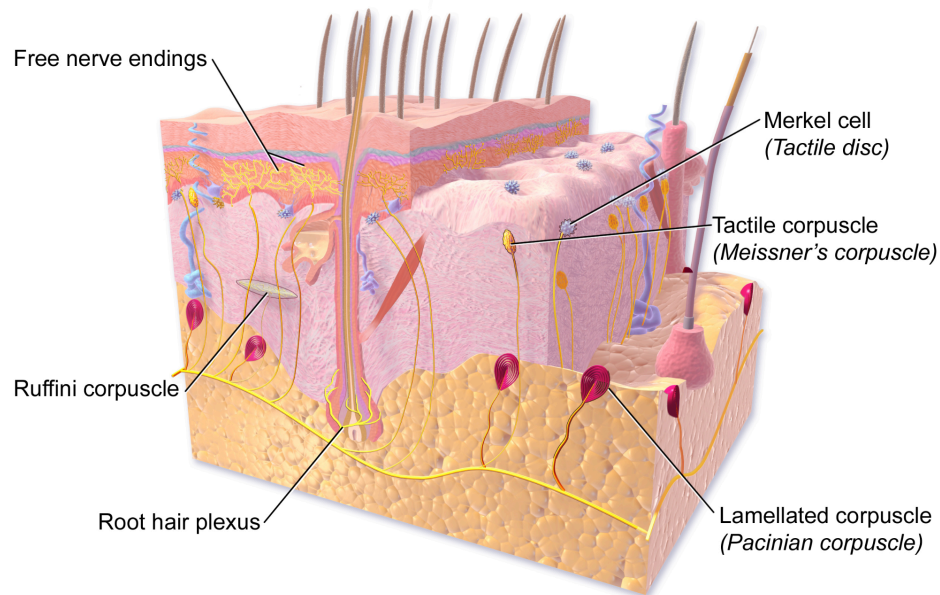
MECHANORECEPTORS

Learning Objectives

Knows the four types of mechanoreceptors in the skin

Know what role the two corpuscles and two nerve endings play in the skin

Know the different rates of adaptation in cutaneous mechanoreceptors



Tactile Receptors in the Skin

Fig.2.3.1. Mechanoreceptors are distributed at different depths and respond to different temporal frequencies and types of pressure. (Credit: Blausen.com Staff. Provided by: Wikipedia. License: CC-BY 3.0)

In glabrous (hairless) skin, there are four principal types of mechanoreceptors, each shaped according to its function (Fig.2.3.1). The tactile corpuscles (also known as Meissner corpuscles) respond to light touch, and adapt rapidly to changes in texture (vibrations around 50 Hz). The bulbous corpuscles (also known as Ruffini endings) detect tension deep in the skin and fascia. The Merkel nerve endings (also known as Merkel discs) detect sustained pressure. The lamellar corpuscles (also known as Pacinian corpuscles) in the skin and fascia detect rapid vibrations (of about 200–300 Hz).

Cutaneous mechanoreceptors respond to mechanical stimuli that result from physical interaction, including pressure and vibration. They are located in the skin, like other cutaneous receptors. They are all innervated by A β fibers, except the mechanoreceiving free nerve endings, which are innervated by A δ fibers. Cutaneous mechanoreceptors can be categorized by morphology, by what kind of sensation they perceive, and by the rate of adaptation. Furthermore, each has a different receptive field.

- The **Slowly Adapting type 1 (SA1) mechanoreceptor**, with the Merkel corpuscle end-organ,

underlies the perception of form and roughness on the skin.[1] They have small receptive fields and produce sustained responses to static stimulation.

- The **Slowly Adapting type 2 (SA2) mechanoreceptors**, with the Ruffini corpuscle end-organ, respond to skin stretch, but have not been closely linked to either proprioceptive or mechanoreceptive roles in perception.[2] They also produce sustained responses to static stimulation, but have large receptive fields.
- The **Rapidly Adapting (RA) or Meissner corpuscle end-organ mechanoreceptor** underlies the perception of flutter[3] and slip on the skin.[4] They have small receptive fields and produce transient responses to the onset and offset of stimulation.
- The **Pacinian corpuscle or Vater-Pacinian corpuscles or Lamellar corpuscles** [5] underlie the perception of high frequency vibration.[3] [6] They also produce transient responses, but have large receptive fields.

Here is a quick five minute video also linked here which covers the four different kinds of mechanoreceptors:



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Wikipedia, Mechanoreceptor

URL: <https://en.wikipedia.org/wiki/Mechanoreceptor#Types>

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References:

- [1] Johnson KO, Hsiao SS (1992). "Neural mechanisms of tactual form and texture perception". Annual Review of Neuroscience. 15: 227–50. doi:10.1146/annurev.ne.15.030192.001303. PMID 1575442.
- [2] ^ Torebjörk HE, Ochoa JL (December 1980). "Specific sensations evoked by activity in single identified

sensory units in man". *Acta Physiologica Scandinavica*. 110 (4): 445–7. doi:10.1111/j.1748-1716.1980.tb06695.x. PMID 7234450.

[3]a b Talbot WH, Darian-Smith I, Kornhuber HH, Mountcastle VB (March 1968). "The sense of flutter-vibration: comparison of the human capacity with response patterns of mechanoreceptive afferents from the monkey hand". *Journal of Neurophysiology*. 31 (2): 301–34. doi:10.1152/jn.1968.31.2.301. PMID 4972033.

[4]^ Johansson RS, Westling G (1987). "Signals in tactile afferents from the fingers eliciting adaptive motor responses during precision grip". *Experimental Brain Research*. 66 (1): 141–54. doi:10.1007/bf00236210. PMID 3582528.

[5]^ Biswas A, Manivannan M, Srinivasan MA (2015). "Multiscale layered biomechanical model of the pacinian corpuscle". *IEEE Transactions on Haptics*. 8 (1): 31–42. doi:10.1109/TOH.2014.2369416. PMID 25398182.

[6]^ Biswas A, Manivannan M, Srinivasan MA (2015). "Vibrotactile sensitivity threshold: nonlinear stochastic mechanotransduction model of the Pacinian Corpuscle". *IEEE Transactions on Haptics*. 8 (1): 102–13. doi:10.1109/TOH.2014.2369422. PMID 25398183.

13.

SOMATOSENSORY PATHWAYS TO THE BRAIN

Learning Objectives

Know why the dorsal root ganglion is important to understanding somatosensation.

Know what the thalamus is

Know what “center-surround antagonism” means and how it distinguishes thalamic receptive fields from cutaneous receptive fields.

Most cutaneous receptors are pseudo-unipolar neurons, with cell bodies in the dorsal root ganglia. The dorsal root ganglia (singular: ganglion; plural: ganglia) are lumps of nervous tissue next to the spinal cord that house the cell bodies of somatosensory neurons. For mechanoreceptors, the first synapse for information on the way to the brain is in the brainstem. After that, information crosses over to the other side of the body, makes another synapse in the thalamus, and then projects to the contralateral postcentral gyrus.

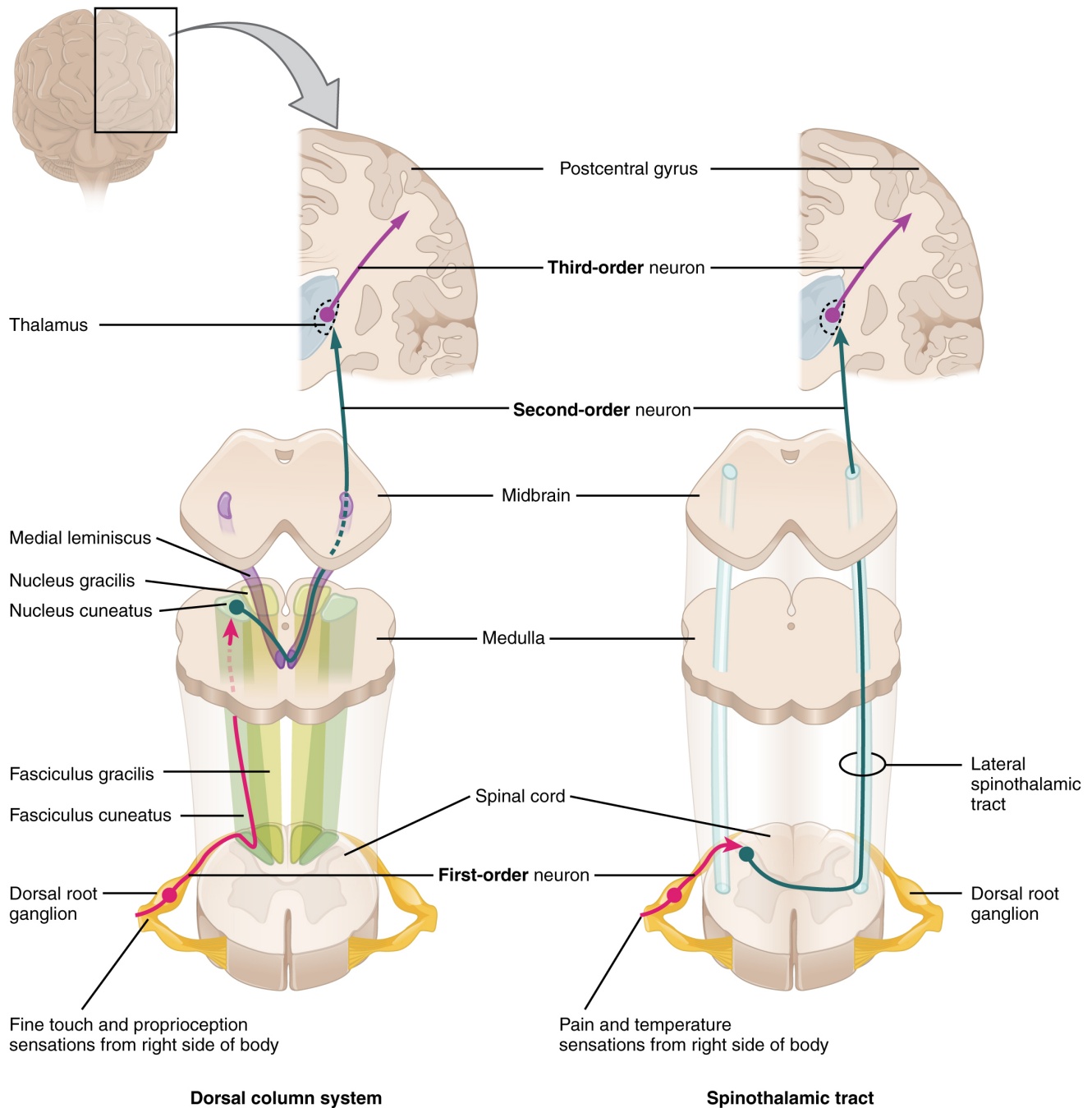


Figure 2.4.1. Ascending pathways for touch and pain information. OpenStax College, CC BY 3.0 <<https://creativecommons.org/licenses/by/3.0/>>, via Wikimedia Commons.

The thalamus is in the middle of the brain, at the top of the brainstem. Almost all sensory information goes to thalamus before the cortex. The thalamus likely helps regulate which stimuli or parts of our environment we pay attention to.

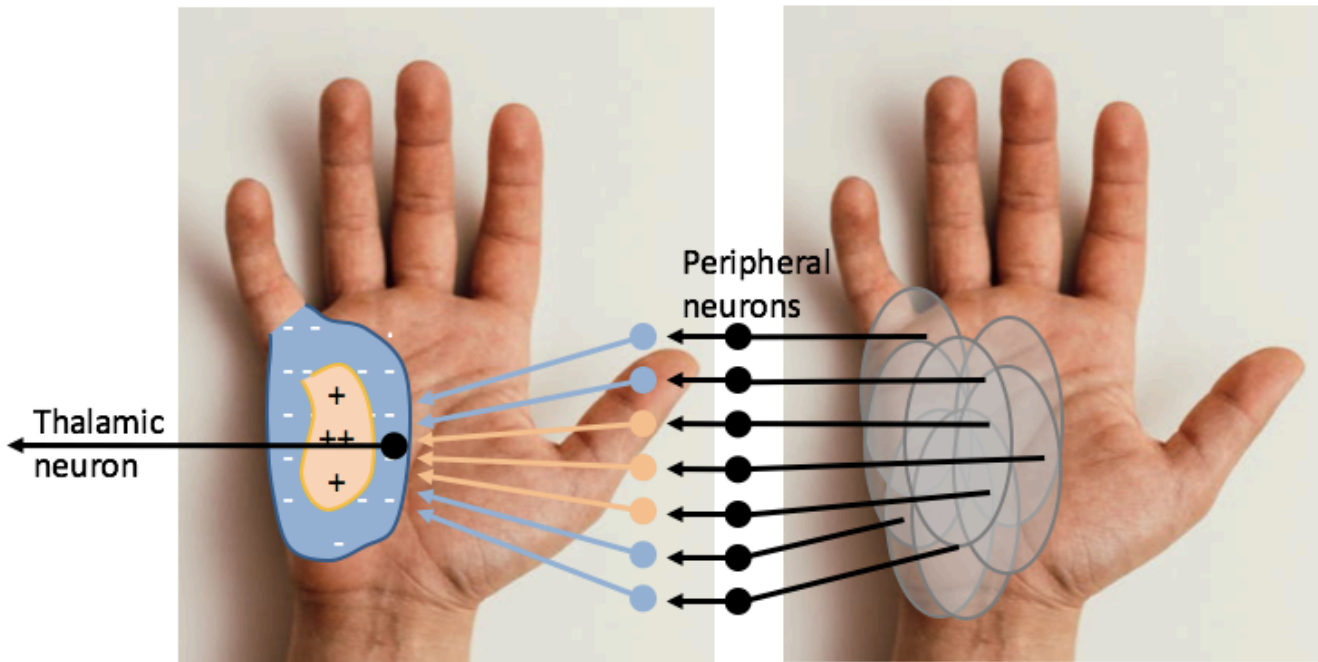


Fig. 2.4.2. Center-surround receptive fields of thalamic neurons. Signals from multiple peripheral cutaneous receptors converge in the thalamus and are combined with excitation and inhibition to create receptive fields that are sensitive to small stimuli and edges. (credit: Juan Pablo Serrano Arenas. Provided by: Pexels. License:CC-BY. Adapted by: Cheryl A. Olman)

Center-surround receptive fields are created by addition and subtraction in the thalamus. Each neuron in the thalamus gets input from many cutaneous sensors, which have overlapping receptive fields. Because some synapses are excitatory and some are inhibitory, this convergence of inputs shapes the receptive field of the thalamic neuron (Fig.2.4.2). Having an inhibitory ring around an excitatory center makes the thalamic neurons respond very little to large stimuli, but small stimuli, which hit just the center of their receptive fields and don't stimulate the entire inhibitory surround, elicit strong responses.

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14.

SOMATOSENSORY REPRESENTATIONS IN THE BRAIN

Learning Objectives

Know what the somatosensory homunculus is

Know where you can find primary sensory cortex, and what is different about the neural responses in primary sensory cortex and nearby regions in parietal cortex that also respond when you touch an object

Be able to describe the effect of attention on neural responses.

The cerebral cortex maintains sensory topography in particular areas of the cortex that correspond to the position of the receptor cells. The somatosensory cortex provides an example in which, in essence, the locations of the somatosensory receptors in the body are mapped onto the somatosensory cortex. This mapping is often depicted using a sensory homunculus.

The term homunculus comes from the Latin word for “little man” and refers to a map of the human body that is laid across a portion of the cerebral cortex. (Fig.2.5.1) below shows the body parts in proportion to the amount of brain space allocated them.

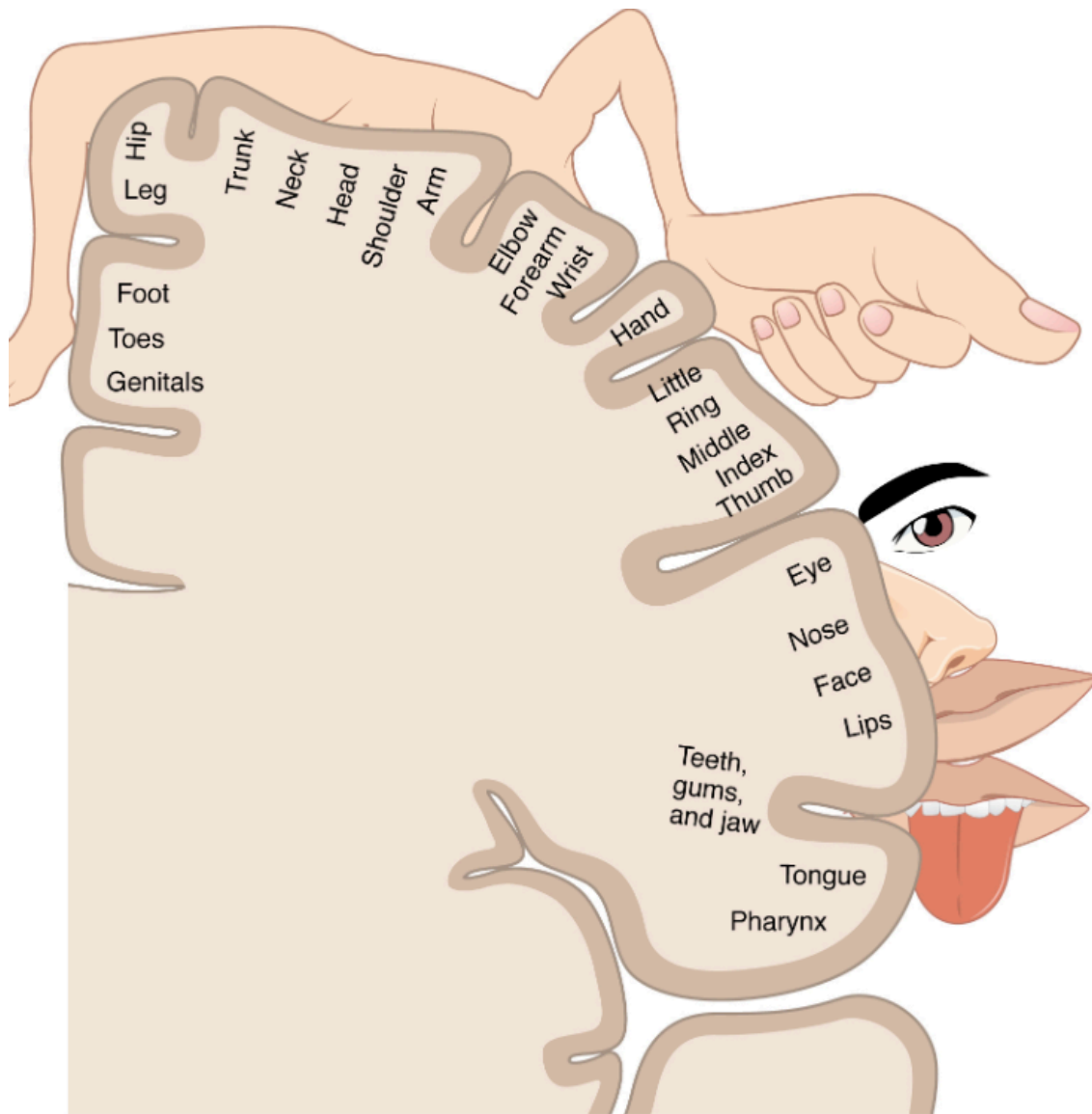


Fig.2.5.1. Sensory homunculus used to depict a “topical” version of the body in the brain. (Provided by: Openstax. License: CC-BY)

Primary somatotopic representation (S1) is on the postcentral gyrus. It is a distorted map (body parts with high receptor density get more territory). Some senses that are controlled by the primary sensory cortex are touch, thermal information, orientation and direction. Regions in parietal cortex outside S1 respond to more complex features such as object-selective responses.

Unattended stimuli can fail to elicit neural response, even in primary somatosensory cortex. But the effects of attention are stronger outside S1.

Watch the video linked here and included below to learn more about somatosensory representations in the brain!



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15.

THE GOOD THINGS ABOUT PAIN

Learning Objectives

Understand why pain is important to our health

Be able to define and describe CIPA.

Pain is an unpleasant experience that involves both physical and psychological components. Feeling pain is quite adaptive because it makes us aware of an injury, and it motivates us to remove ourselves from the cause of that injury. In addition, pain also makes us less likely to suffer additional injury because we will be gentler with our injured body parts.



Fig. 2.6.1. Sad Child. Pain teaches us our limits. Without pain this baby may not learn to not bite their thumb which can lead to future injuries. (Credit: George Hodan. Provided by: Public Domain Pictures. License: CC0)

However, there are some individuals born without the ability to feel pain. This very rare genetic disorder is known as congenital insensitivity to pain (aka CIPA or congenital analgesia). While those with congenital analgesia can detect differences in temperature and pressure, they cannot experience pain. As a result, they often suffer significant injuries. Young children have serious mouth and tongue injuries because they have bitten themselves repeatedly. CIPA is an inherited disease in which people lack pain in the mouth and limbs. Generally results in early death, due to either overheating (the A in CIPA is anhidrosis, an inability to sweat to cool oneself) or to repeated injury (e.g., chewing off your tongue and lips, twisting the skin off your hand trying to open a jar). CIPA happens when you get 2 defective copies of the SCN9A gene, which codes for a sub-unit of the Na⁺ channels in nociceptors. This results in these receptors to not form properly and a failure to send pain signals to the brain.

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References:

Gerbrich E. van den Bosch, Martin G. A. Baartmans, Paul Vos, Jan Dokter, Tonya White, Dick Tibboel
Pediatrics May 2014, 133 (5) e1381-e1387; DOI: 10.1542/peds.2013-2015.

ITCH

Learning Objectives

Know how neurons initiate a sensation of itching

Be able to describe an experiment that tests whether itch is a sensation other than pain

Itch was not originally identified as its own sensory modality. Instead, researchers thought itch was the brain's interpretation (perception) of mild or subthreshold stimulation of nociceptors. However, specific receptors for itch have been identified, which means that itch is a unique sensation. For example, capsaicin activates receptors on peripheral nerve endings to induce pain, while histamine activates different receptors on a subset of capsaicin-responsive nerve endings to induce itch (LaMotte, 2014).

Unique, itch-selective neurons have been discovered and characterized as having unmyelinated axons with free nerve endings, not unlike thermal and mechanical nociceptors. In a microneurography experiment, researchers isolated the unmyelinated axons of 56 different neurons in the legs of healthy volunteers (Schmelz, 1997). None of the axons carried spontaneous action potentials, and 13 did not respond to high heat or strong pressure, ruling them out as mechanoreceptors or thermal nociceptors. However, when histamine was slowly injected into the skin, the neurons that had been non-responsive started sending action potentials. The rate of action potentials correlated with the participants' reports of the severity of the itch, and when the action potentials started fading away, so did the sensation of itch (see Fig.2.7.1).

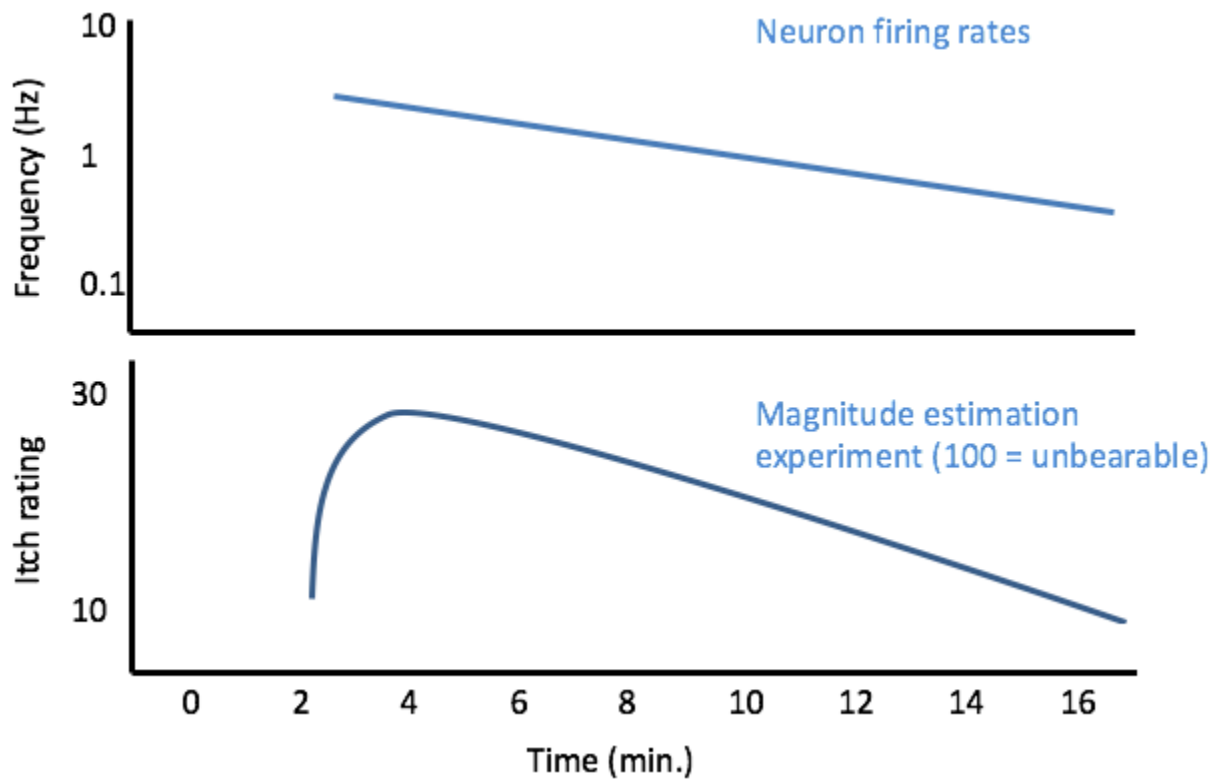


Fig. 2.7.1. The frequency of C-fiber action potentials elicited by application of the itch-causing chemical histamine (top) is similar in time-course and intensity to the perception of histamine-induced itch reported by human participants on a visual analog scale (VAS) (bottom) (credit: Schmelz, 1997)

It is still unknown exactly how itch signals are coded separately from pain signals in the central nervous system. It is widely accepted that there does not exist a labeled line for itch, as itch-responsive neurons are a subset of pain-responsive neurons in the spinal cord and cortex. Instead, it is likely that itch is mediated by a population code whereby specific activation of itch-selective neurons within the population of pain-responsive neurons results in the perception of itch (LaMotte, 2014).

Author: Cheryl Olman

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References:

Schmelz et al (1997). Specific C-Receptors for Itch in Human Skin. *J. Neuroscience* 17(20):8003-8008

LaMotte, Dong, and Ringkamp (2014). Sensory neurons and circuits mediating itch. *Nature Reviews: Neuroscience*. 15: 19-31.

17.

CATEGORIES OF PAIN

Learning Objectives

- Be able to define nociceptive pain and give an example
- Be able to define inflammatory pain and give an example
- Be able to define neuropathic pain and give an example

There are three types of pain.

Nociceptive pain is mediated by cutaneous receptors which detect heat, cold, severe force and chemical insult

- Medical term that describes pain from physical or potential damage to the body
- Examples: pain felt from sports injuries, dental procedures, or arthritis

Inflammatory pain happens when immune responses activate nociceptors in response to injury. Pain associated with tumors and post-injury swelling are examples of inflammatory pain.

- An increased sensitivity due to an immune inflammatory response
- Examples: pain, redness, or swelling caused by a cut, bump, and any injuries ranging from minor to major

Neuropathic pain is caused by damage to the nervous system itself. Examples: carpal tunnel syndrome, sciatic nerve pain

- Associated with abnormal sensations or pain from non-painful stimuli
- Examples: phantom limb, neuralgia, diabetic neuropathy, central pain syndrome



Fig. 2.8.1. Advil Pills. Advil is a common over the counter drug that helps with inflammatory pain. (Credit: Tony Webster. Provided by: Flickrriver. License: CC-BY 2.0)

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18.

CAPSAICIN

Learning Objectives

Know how Capsaicin causes pain

Be able to describe how Capsaicin treats pain

Capsaicin is the oil-soluble chemical that is found in hot chile peppers (it's what makes them hot!!).



Fig. 2.9.1. Chili Peppers. Chilies and other spicy peppers like these contain the chemical capsaicin. This chemical causes the pain we feel after eating spicy foods (and is the reason why it lingers for so long!). (Credit: John Lambeth. Provided by: Pexels. License: CC-BY)

In the body, there are two types of somatosensory signals that are transduced by free nerve endings (pain and temperature). These two modalities use thermoreceptors and nociceptors to transduce temperature and pain stimuli, respectively. Nociception is the sensation of potentially damaging stimuli. Nociceptive pain is mediated by cutaneous (essentially skin) receptors that detect heat, cold, severe force, and chemical insult.

So, when someone eats spicy foods (containing capsaicin), the Capsaicin molecules activate polymodal nociceptors that respond to heat and capsaicin, effectively creating a pain sensation. (Most nociceptors respond to noxious mechanical stimuli (painful pressure, squeezing or cutting the tissue), noxious thermal stimuli (heat or extreme cold), and chemical stimuli and are therefore called polymodal)

The dynamics of Capsaicin are unusual in that the molecule remains bound for a long time. Because of this, Capsaicin decreases the ability of other stimuli to elicit pain sensations through the activated nociceptor. For this reason, capsaicin can be used as a topical analgesic, such as in products such as Icy Hot™.

To learn more, watch this Ted Talk linked here and included below.



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19.

PAIN PATHWAYS IN THE BRAIN

Learning Objectives

Know the similarities and differences in pain vs. touch pathways from the periphery to the brain

Understand how the cortical targets of pain projections explain why it is such a good motivator

There's a few main similarities and differences to be aware of regarding pain vs. touch pathways:

The dorsal column system is primarily responsible for touch sensations and proprioception, whereas the spinothalamic tract pathway is primarily responsible for pain and temperature sensations. They are similar in that they both begin with dorsal root ganglion cells, as with most general sensory information.

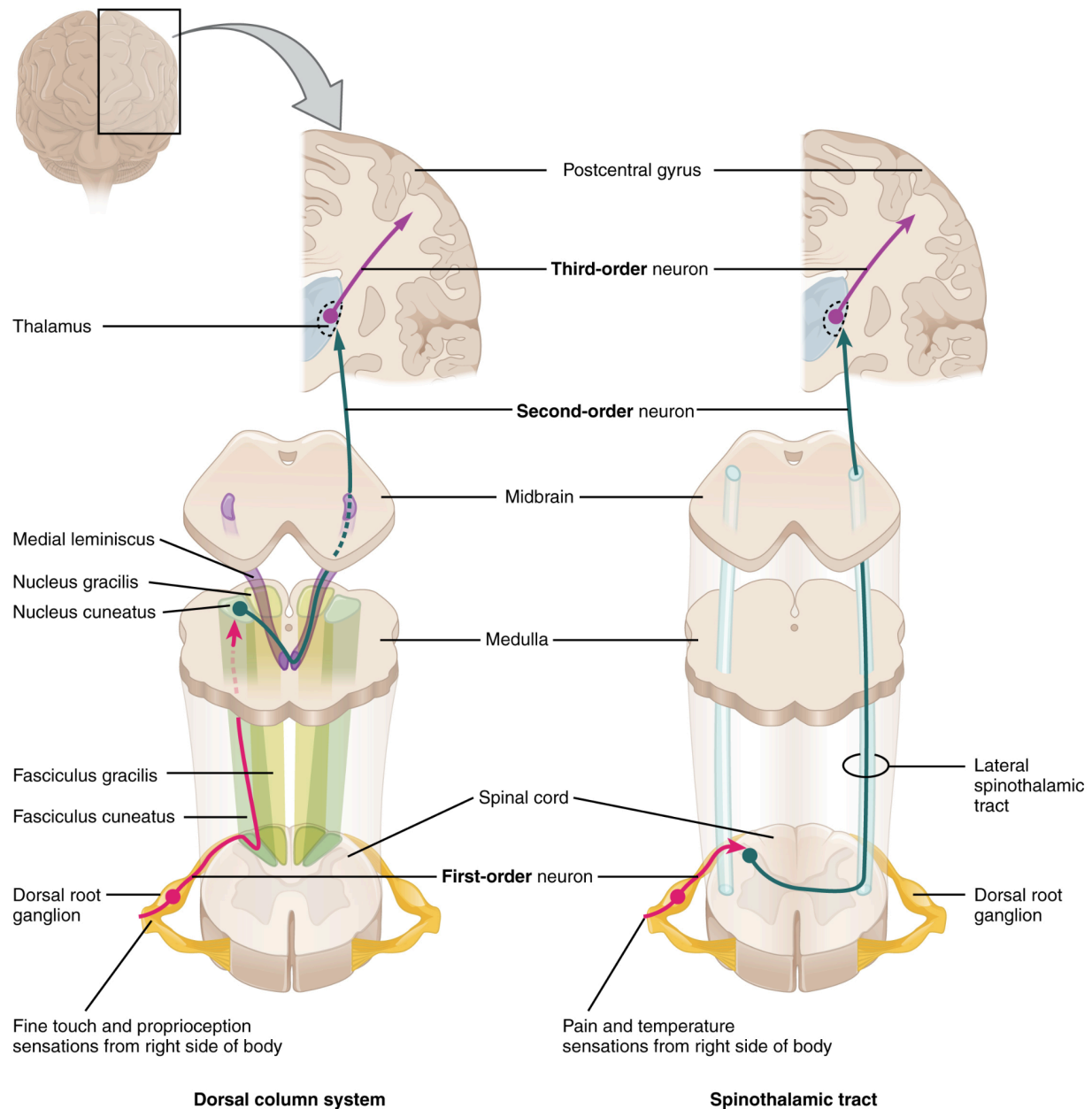


Fig. 2.10.1. Ascending sensory pathways of the spinal cord. The dorsal column system and spinothalamic tract are the major ascending pathways that connect the periphery with the brain. (Provided by: Openstax. License: CC-BY 4.0)

The dorsal column terminates neuron one in the nuclei of the medulla, then separates into two component tracts, targeting specific fibers. The fasciculus gracilis (contains axons from the legs and lower body) targets the nucleus gracilis, while the fasciculus cuneatus (contains axons from the upper body and arms) targets the nucleus cuneatus.

The spinothalamic tract neurons extend their axons to the dorsal horn, where they synapse with the second neuron. Axons from these second neurons then decussate within the spinal cord and ascend to the brain and enter the thalamus, where each synapses with the third neuron in its respective pathway. In the dorsal column

system, this decussation takes place in the brain stem; in the spinothalamic pathway, it takes place in the spinal cord at the same spinal cord level at which the information entered.

Not only does pain project to the contralateral primary somatosensory cortex, but it does to the limbic system as well. If you want to motivate a strong reaction from someone, the limbic system is a good target!

Check out this Crash Course video linked here and included below to learn more!



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PHYSIOLOGICAL TREATMENTS FOR PAIN

Learning Objectives

Be able to give 2 examples of drugs or treatments that work outside the central nervous system, and describe how they work

Be able to give 2 examples of drugs or treatments that work inside the central nervous system, and describe how they work

Two examples of drugs/treatments that work OUTSIDE the central nervous system

- Capsaicin: Capsaicin molecules bind to a transmembrane ion channel in nociceptors that is sensitive to temperatures above 37°C. The dynamics of capsaicin binding with this transmembrane ion channel is unusual in that the molecule remains bound for a long time. Because of this, it will decrease the ability of other stimuli to elicit pain sensations through the activated nociceptor. For this reason, capsaicin can be used as a topical analgesic, such as in products such as Icy Hot™.
- NSAIDs: Non-steroidal anti-inflammatory drugs (NSAIDs) such as Advil and Motrin reduce pain because they inhibit the synthesis of prostaglandins. High levels of NSAIDs reduce inflammation

Two examples of drugs/treatments that work INSIDE the central nervous system

- Opioids: An opioid is one of a category of drugs that includes heroin, morphine, methadone, and codeine. Opioids have analgesic properties; that is, they decrease pain. Opioids are prescribed less frequently than NSAIDs due to their risk of causing an addiction. Opioids are most addictive when you take them in ways that aren't recommended such as crushing them up and snorting it.
- Stimulus-produced analgesia: mechanical stimulus can suppress pain by targeting inhibitory neurons in the spinal cord.



Fig.2.11.1. Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly prescribed by healthcare providers for treating symptoms of pain. (credit: Sgt. Mark R. W. Orders-Woempner. Provided by: Air Force Medical Service. License: CC0)

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Mayo Clinic. (2018). *How opioid addiction occurs*. <https://www.mayoclinic.org/diseases-conditions/prescription-drug-abuse/in-depth/how-opioid-addiction-occurs/art-20360372>

PSYCHOLOGICAL TREATMENTS FOR PAIN

Learning Objectives

Understand how giving patients an accurate understanding of what to expect improves their outcomes

Understand how distracting people from their pain affects their experience

Be able to describe how an experiment with hypnosis showed how we can dissociate pain from suffering

Expectation, or giving patients an ‘accurate’ understanding of what to anticipate from treatment, is shown to improve their outcome in relation to pain relief. In an article written by Jennifer M Weiss, M.D. in *Psychology Today* (2018), it is stated that people expecting complete pain relief are going to be let down if doctors do not begin to manage the expectations of the outcome to their patients. Because of this, complete and total pain relief is not something most patients attain because expectations may be too high and not accurate.

Distraction, or thinking about something positive, is also shown to decrease the severity of pain in patients. In an article by Malcolm H. Johnson, “How Does Distraction Work in the Management of Pain?” (2005), he makes an argument that the distraction approach to relieve pain could be looked at as a competition between exogenous and endogenous information processing and that a patient’s perception of pain is suppressed by consciously focused attention to non-pain stimulus or stimuli. However there is a trade-off; the efficiency of the distraction can be affected by the qualities of the distractor, the qualities of the pain experience being suppressed, and factors related to individual differences.



Fig. 2.12.1. The doctor is singing to the baby, distracting her while she administers the shot. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-SA 4.0)

Effectiveness of hypnosis to decrease sensitivity to pain is known as hypo-analgesia. A meta-analysis, conducted in 2000 of 18 published studies by psychologists Guy Montgomery, PhD, Katherine DuHamel, PhD, and William Redd, PhD, showed that 75% of clinical and experimental participants with different types of pain obtained substantial pain relief from hypnotic techniques. These findings support that hypnosis is likely to be effective for most people suffering from diverse forms of pain unless the patient fails to respond to it or shows a strong opposition against it.

Another possible pain treatment may be therapeutic vibrations. Watch the video below and linked here to learn more!



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References:

Weiss, J. (2018, December 23). Expectations Instead of Medications: Managing Patient Pain. Retrieved May 27, 2020, from <https://www.psychologytoday.com/us/blog/women-in-medicine/201812/expectations-instead-medications-managing-patient-pain>

Johnson M. H. (2005). How does distraction work in the management of pain?. *Current pain and headache reports*, 9(2), 90–95. <https://doi.org/10.1007/s11916-005-0044-1>

Hypnosis for the Relief and Control of Pain. (2004, July 2). Retrieved May 27, 2020, from <https://www.apa.org/research/action/hypnosis>

Olman, C. A. (2017). PSY 3031 Outline. Retrieved May 27, 2020, from <http://vision.psych.umn.edu/users/caolman/courses/PSY3031/>

ACTIVE LEARNING EXERCISE - 2-POINT DISCRIMINATION

This slide show has instructions for measuring the tactile acuity of your fingertip and palm, using the Method of Constant Stimuli. In the process, you will create a psychometric function for each body part, and you will use it to determine what your two-point discrimination threshold is. A low threshold means you can distinguish two points that are very close to each other, which means you have good acuity.



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SECTION III

PROPRIOCEPTION: BALANCE AND PHANTOM LIMBS

One theme that unites the three topics covered in this chapter — balance, phantom limbs, and prosthetics — is proprioception. Proprioception is our sense of our own body. We need it to stay balanced, it's gone awry when we're experiencing phantom limbs, but current technological innovations are actually taking advantage of our ability to perceive phantom limbs to create active prosthetic limbs that can be thought-controlled!

Another idea that we rely on to understand how our brain processes balance and perceives phantom and prosthetic limbs is cue combination. Cue combination is an idea that crops up again and again as we study perception. The idea is this: while it's simplest to talk about one thing at a time — just visual perception or just auditory perception — the reality is that we're always experiencing multiple senses at the same time. As I type, I both feel and hear the keys on my keyboard; as I talk, I feel myself speak and I hear myself speak. Our brains are always integrating cues from multiple senses as they try to figure out what's going on in the world.

Cue conflict is related to cue combination: it is what happens when the cues we're getting from different senses don't fit together. What do we do when we get conflicting information about our environment? In the case of motion sickness, we feel ill. If we hear a delayed copy of our own voice as we try to speak, we stop being able to speak correctly. Or, in the case of the rubber hand illusion, our brains might just decide to believe a reliable cue and ignore the conflicting cue.

This chapter was created by Iqra Ismail, Drew Kampmeier, Nika Khadem, Kyle Knudsen, Nathaniel Luers, Anna Maunu, Mehraz Mirhosseini, Ryleigh Orning,

KINESTHESIA AND PROPRIOCEPTION

Learning Objectives

Understand the relationship between the terms kinesthesia and proprioception

Be able to explain how the knee-jerk reflex works

Proprioception is the sense of the relative positioning of neighboring parts of the body, and the sense of the strength of effort needed for movement. It is distinguished from exteroception, by which one perceives the outside world, and interoception, by which one perceives pain, hunger, and the movement of internal organs. A major component of proprioception is joint position sense (JPS), which involves an individual's ability to perceive the position of a joint without the aid of vision. Proprioception is one of the subtler sensory systems, but it comes into play almost every moment. This system is activated when you step off a curb and know where to put your foot, or when you push an elevator button and control how hard you have to press down with your fingers.

Kinesthesia is the awareness of the position and movement of the parts of the body using sensory organs, which are known as proprioceptors, in joints and muscles. Kinesthesia is a key component in muscle memory and hand-eye coordination. The discovery of kinesthesia served as a precursor to the study of proprioception. While the terms proprioception and kinesthesia are often used interchangeably, they actually have many different components. Often the kinesthetic sense is differentiated from proprioception by excluding the sense of equilibrium or balance from kinesthesia. An inner ear infection, for example, might degrade the sense of balance. This would degrade the proprioceptive sense, but not the kinesthetic sense. The affected individual would be able to walk, but only by using the sense of sight to maintain balance; the person would be unable to walk with eyes closed. Another difference in proprioception and kinesthesia is that kinesthesia focuses on the body's motion or movements, while proprioception focuses more on the body's awareness of its movements and behaviors. This has led to the notion that kinesthesia is more behavioral, and proprioception is more cognitive.

Reflexes combine the spinal sensory and motor components with a sensory input that directly generates a motor response. The reflexes that are tested in the neurological exam are classified into two groups. A deep tendon reflex is commonly known as a stretch reflex and is elicited by a strong tap to a tendon, such as in the knee-jerk reflex. A superficial reflex is elicited through gentle stimulation of the skin and causes contraction of the associated muscles.

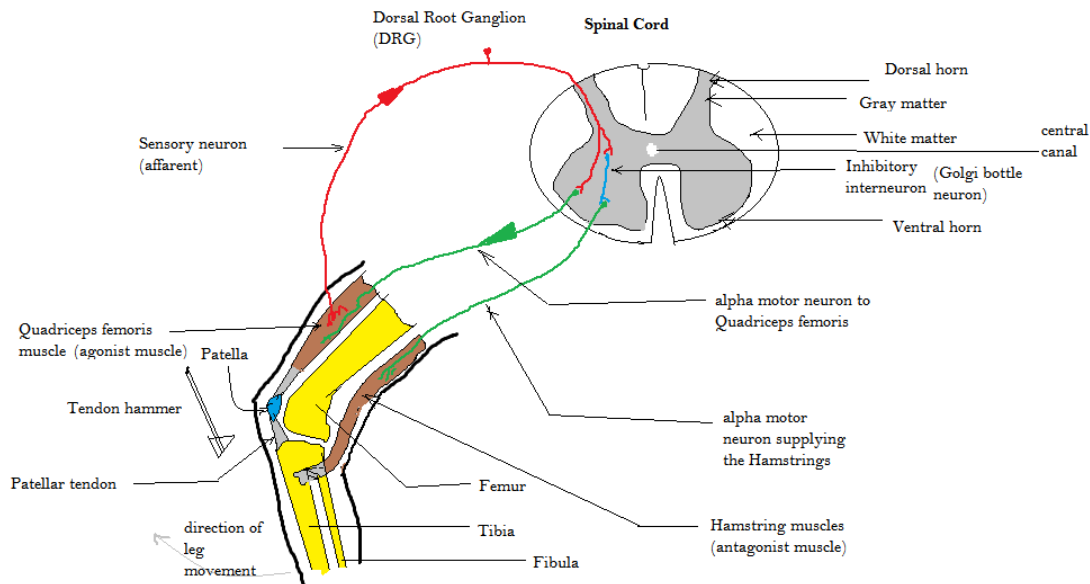
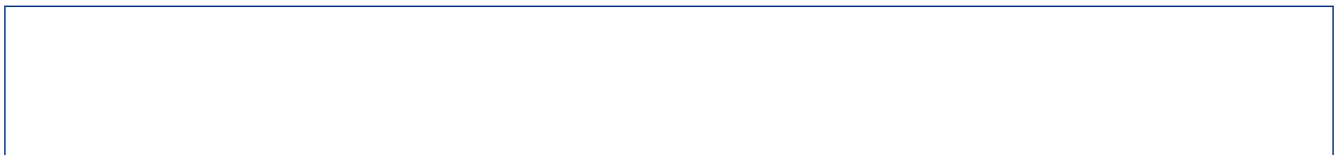


Fig.3.1.1. Knee-jerk Reflex. This illustrates the patellar reflex, also known as a myotatic reflex, the stretch reflex, and the knee-jerk reflex.(Credit: Amiya Sarkar. Provided by: Wikipedia License: CC-BY-SA 4.0.)

Knee-jerk reflex, also called patellar reflex, sudden kicking movement of the lower leg in response to a sharp tap on the patellar tendon, which lies just below the kneecap (Fig.3.1.1). One of the several positions that a subject may take for the test is to sit with knees bent and with one leg crossed over the other so that the upper foot hangs clear of the floor. The sharp tap on the tendon slightly stretches the quadriceps, the complex of muscles at the front of the upper leg. In reaction, these muscles contract and the contraction tends to straighten the leg in a kicking motion. Exaggeration or absence of the reaction suggests that there may be damage to the central nervous system. The knee jerk can also be helpful in recognizing thyroid disease.

If you want to learn more about reflexes, watch this youtube video or follow this link to OpenStax Textbook chapter on Sensory and Motor Exams.





One or more interactive elements has been excluded from this version of the text. You can view them online here: <https://pressbooks.umn.edu/sensationandperception/?p=504#oembed-1>

Exercises



An interactive H5P element has been excluded from this version of the text. You can view it online here: <https://pressbooks.umn.edu/sensationandperception/?p=504#h5p-3>

Reflection

Take a minute to answer these questions in your notes:

1. In a few sentences, describe the relationship between the terms kinesthesia and proprioception.
2. In a few sentences, please explain how the knee-jerk reflex works.

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Encyclopaedia Britannica, Knee-Jerk Reflex

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PHANTOM LIMBS

Learning Objectives

- Know what a phantom limb is.
- Be able to describe three possible reasons that people experience phantom limbs or phantom pain.
- Be able to describe how mirror boxes are used to treat phantom limb pain.

In this section, the focus of learning is to understand more about phantom limbs. Very few people have personal experience with phantom limbs because it requires for a limb to be absent. There are numerous causes of this ranging from car accidents where someone loses a limb to a birth defect where a child is born with only one arm or leg. Now, I think everyone is wondering, what is a phantom limb? A phantom limb is the sensation of feeling various things in a limb that is not there. For example, a tingling sensation in an area where a limb no longer exists.

There are multiple complications that could be caused in experiencing sensations in limbs that are no longer existent. One of the main complications is phantom pain, which is a painful sensation on phantom limbs. There are three different ways that an individual could experience phantom pain. The first is called Neuroma, which is a physical source of pain caused by scarring on nerve endings. Sometimes, neuroma can be treated by surgery to remove the growth/tumor on the nerve, although the neuroma might re-grow after surgery, so the treatment is not always effective. The treatments for Cortical causes of phantom pain are less obvious. The first Cortical cause comes to negative sensory deprivation. For example, if the last thing a person felt on their arm before it got amputated was pain, the person's brain could be stuck in that state. The second Cortical cause is about sensory reorganization and the issue here is that nerves from an amputated limb could grow back and infiltrate a different area of the body causing pain.



Fig. 3.2.1. Navy Cmdr. (Dr.) Jack Tsao shows Army Sgt. Nicholas Paupore how to perform mirror therapy to treat phantom pain in his amputated right leg Jan. 15 at Walter Reed Army Medical Center in Washington, D.C. Commander Tsao conducted the first clinical trials in mirror therapy and said he hopes to advance the study to bring relief to amputees from Iraq and Afghanistan. Commander Tsao is the associate professor of neurology at the Uniformed Services University of the Health Sciences at Bethesda, Md. (Credit: Donna Miles. Provided by: American Forces Press Service. License: CC-BY)

There is new technology being used, though, that can help people struggling from phantom pain with Cortical causes and it's called mirror boxes. Mirror boxes are treatments that rewire the brain to portray a lack of sensory responses as not being painful. It allows a person's brain to not think of an absence of sensory responses as pain and it allows the person to be at ease rather than feeling pain.

Exercises

1. What is a phantom limb?

- A. An arm or leg of a person
 - B. A person who had had a limb amputated
 - C. Limbs that are not present (due to amputation or congenital condition) but still perceived
 - D. A figment of the imagination
2. What is neuroma?
- A. Inflammation or scar tissue on a nerve
 - B. Damage, disease, or dysfunction of one or more nerves especially of the peripheral nervous system that is typically marked by burning or shooting pain
 - C. The branch of medicine or biology that deals with the anatomy, functions, and organic disorders of nerves and the nervous system
 - D. Differing in mental or neurological function from what is considered typical or normal
3. How can neuroma be treated?
- A. Prescribed medications
 - B. Do nothing
 - C. Tell your doctor
 - D. Surgery
4. What are the two cortical causes of phantom pain?
- A. Pessimistic interpretation and mechanical sensation
 - B. Sensory reorganization and neuroma
 - C. Pessimistic interpretation of sensory deprivation and sensory reorganization
 - D. Phantom limbs and pain
5. What are mirror boxes?
- A. A reflective surface, now typically of glass coated with a metal amalgamation that reflects an image
 - B. A neuron that fires when an animal acts and when the animal observes the same action performed by another
 - C. A condition that causes a person to feel the sensations of being touched on the opposite side of their body when they see another person being touched
 - D. Promising treatments that train the brain to interpret sensory responses, or the lack of sensory responses, as not painful

Answer Key:

- 1. C
- 2. A
- 3. D

4. C
5. D

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Adapted by: Nathaniel Luers

ACTIVE PROSTHETIC LIMBS

Learning Objectives

Be able to explain what targeted muscle innervation is, and why it is done

Be able to describe at least 2 ways that people build Brain/Machine Interfaces

Prosthetic limbs have been around for a long time, and mechanical improvements are continuously being made. Active prosthetic limbs are exciting, although they are difficult to control without sensory feedback. First we will talk about two approaches for controlling active prosthetic limbs (since directly wiring into the nervous system isn't an option, due to reasons of complexity, vulnerability and stability).

The first approach, targeted muscle reinnervation, provides control signals, once patients re-learn how their stump muscles map to bionic limb parts. In targeted muscle innervation, a physician surgically re-routes a motor nerve so the neurons grow out and innervate a muscle, creating little twitches that sensors can pick up. The sensors then decode the user's desired action from the pattern of twitches in the re-innervated muscle. In simpler terms, targeted muscle reinnervation gives severed nerves something to do and somewhere to go.

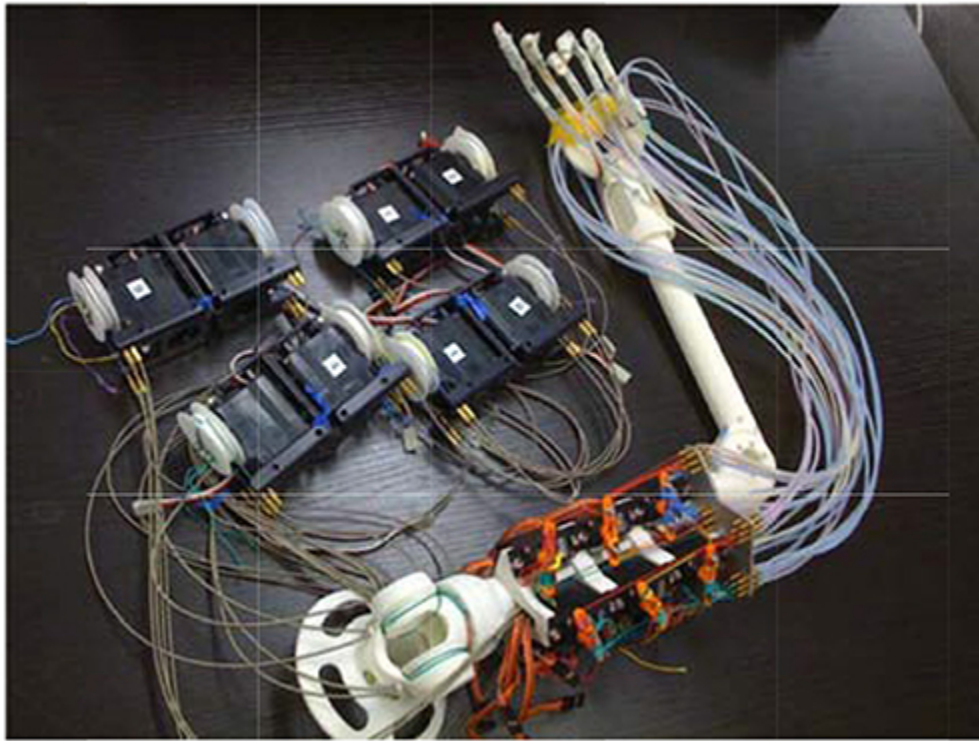


Figure 3.3.1: This figure shows a prosthetic arm with an interference-based wire-driven mechanism. (Credit: Morishita S, Sato K, Watanabe H, Nishimura Y, Isa T, Kato R, Nakamura T and Yokoi H (2014) Brain-machine interface to control a prosthetic arm with monkey ECoGs during periodic movements. *Front. Neurosci.* 8:417. doi: 10.3389/fnins.2014.00417. License: CC-BY 4.0.)

The second approach for controlling active prosthetic limbs is called Brain-Machine Interface. When direct access to peripheral nerves is not an option, electrophysiological signals from the brain can be used to control the bionic limbs. These signals can be detected either invasively or non-invasively. Invasive approaches allow for higher success in prosthetic limb control, but the degeneration and necrosis limit the long-term use. This obstacle led researchers to develop non-invasive methods, such as electroencephalography (EEG) electrodes. EEG electrodes are placed on the scalp. The electrodes record the brain signals and are sent to a computer that determines what motion the user wants to accomplish. Another way to build a Brain-Machine Interface for prosthetic limbs are electrocorticography (ECoG) electrodes, which is an invasive signal detection method involving the use of a surface electrode on the cerebral cortex under the dura mater. It is important to note that this approach has long-term stability with low clinical risk.

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Brain-machine interface to control a prosthetic arm with monkey ECoGs during periodic movements.

Authored by: Morishita S, Sato K, Watanabe H, Nishimura Y, Isa T, Kato R, Nakamura T and Yokoi H

Provided by: Frontiers in Neuroscience

Url: <https://doi.org/10.3389/fnins.2014.00417>

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Adapted by: Sayla Hendry

References:

Lebedev, M. A. & Nicolelis, M. A. L. (2006). Brain-machine interfaces: past, present and future. *TRENDS in Neurosciences*, 29(9):536.

26.

TARGETED SENSORY RE-INNervation

Learning Objectives

Be able to explain why sensory feedback is necessary to control an active prosthetic limb.

Be able to describe at least 1 method of providing sensory feedback



Fig.3.4.1 Targeted muscle reinnervation provides control signals, once patients re-learn how their stump muscles map to bionic limb parts.
(Credit: RAEng_Publications, Provided by: Pixabay License: CC0)

Targeted sensory reinnervation is a method by which skin near or over the targeted muscle is denervated, then reinnervated with afferent fibers of the remaining hand nerves. This allows an amputee to better control their active prosthetic limb. Therefore, when this piece of skin is touched, it provides the amputee with a sense of the missing arm or hand being touched. While active prosthetic limbs are exciting, they are difficult to control without this sensory feedback. Without sensory feedback, an amputee will not be able to gauge how much force

to apply on an object. So, someone may be in for a much more aggressive handshake than they originally planned. Or an amputee may accidentally crush their can of Pepsi when they pick it up. An additional issue involving sensory feedback is detecting temperature. An amputee has no sense of burning heat or freezing cold without some form of sensory feedback. This can be quite dangerous if they pass the object to their other hand for example.

In providing the necessary sensory feedback by delivering temperature and force cues to adjacent tissue, targeted sensory reinnervation lets the brain learn to interpret the sensations as coming from the prosthetic arm. This approach takes advantage of the same mechanisms that let phantom limbs live in the brain. For example, while receiving an alcohol rub on his chest after the surgery, a patient described a sensation of being touched on the pinky. The explanation for this phenomenon is that, since his subcutaneous fat was removed during surgery, his chest skin was denervated. Thus, the afferent nerve fibers regenerated through the pectoral muscle, reinnervating the skin over the muscle. Since then, areas of the pectoral muscle have been mapped to parts of the arm and hand according to the patient's description of touch sensations he felt.

With this discovery, a team of doctors set out to perform nerve transfer surgery specifically aimed to reinnervate sensory feedback. A piece of skin near or over the targeted muscle was denervated, thus the afferent nerve fibers were allowed to reinnervate the skin. This technique has been dubbed "transfer sensation", and it has the potential of providing useful sensory feedback, such as pressure sensing, to help the patient judge the amount of force to be exerted.

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Adapted by: Kori Skrypek Wikipedia, Targeted Reinnervation

URL: https://en.wikipedia.org/wiki/Targeted_reinnervation#:~:text=Targeted%20sensory%20reinnervation%20is%20a,arm%20or%20hand%20being%20touched.

Targeted_reinnervation#:~:text=Targeted%20sensory%20reinnervation%20is%20a,arm%20or%20hand%20being%20touched.

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27.

VESTIBULAR TRANSDUCTION

Learning Objectives

Distinguish the roles of the semicircular canals, the saccule, and the utricle

Describe how hair cells transduce linear or angular acceleration

Explain why some people get the spins when they've consumed a lot of alcohol

The vestibular system is responsible for detecting head motion and maintaining balance. The sensory organs that support balance are in the inner ear.

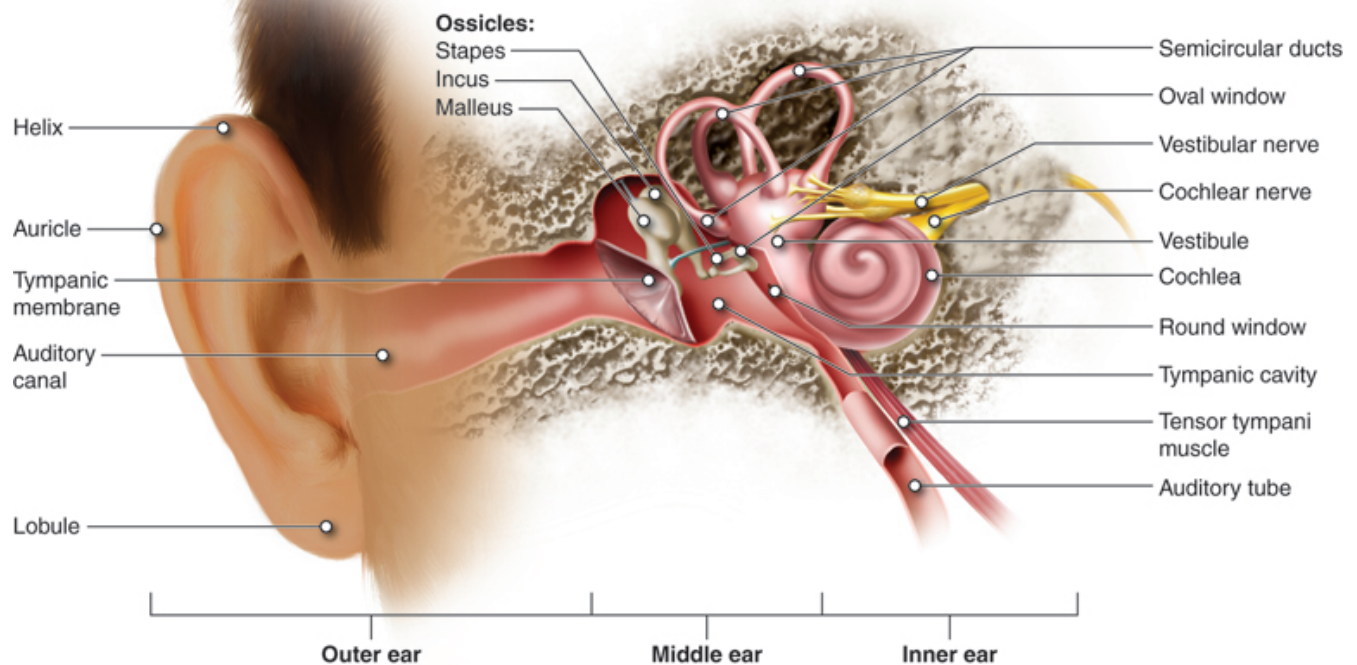


Figure 3.5.1. Anatomy of the Ear. The outer ear is the auricle and ear canal through to the tympanic membrane. The middle ear contains the ossicles and is connected to the pharynx by the auditory tube. The inner ear is the cochlea and vestibule which are responsible for hearing and equilibrium, respectively. This work by Cenvio is licensed under a Creative Commons Attribution 3.0 United States (<http://creativecommons.org/licenses/by/3.0/us/>).

In the vestibular system, there are three semicircular canals and two otolithic receptors, the saccule and utricle. Semicircular canals transduce, or convert, the physical energy of rotational angular accelerations into neural impulses while the utricle and saccule transduce the physical energy of linear accelerations into neural impulses. The three semicircular canals lie perpendicular to each other.

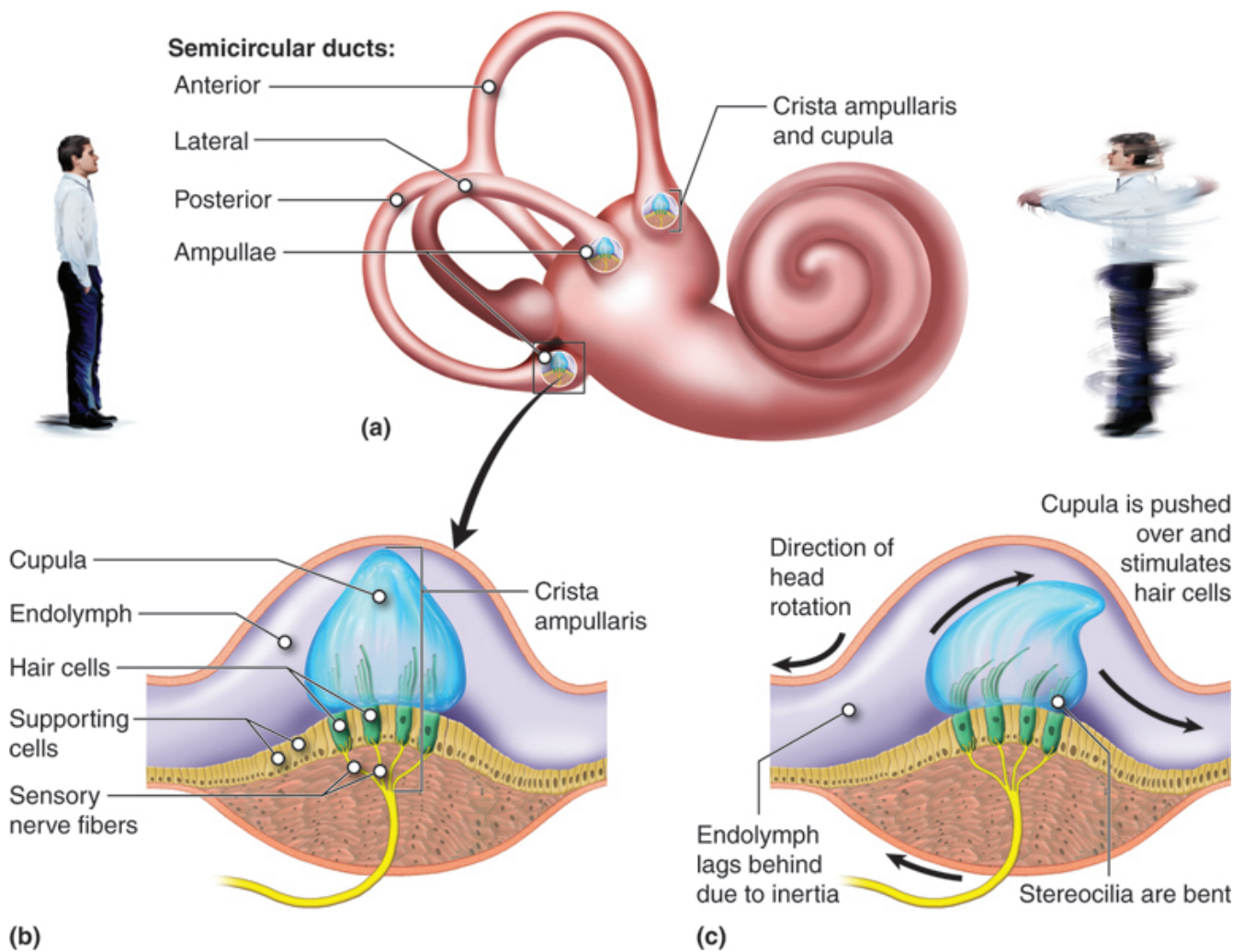


Figure 3.5.2. Structure and Function of the Semicircular Canals. The three canals each have an ampulla containing a crista ampullaris and cupula (a). When the head is stationary, the cupula, and embedded stereocilia, are not bent (b). When the head rotates in the same plane as one of the canals, the fluid in the canal (endolymph) lags, leading to bending of the stereocilia in the cupula, which initiates nerve impulses. This work by Cenveo is licensed under a Creative Commons Attribution 3.0 United States (<http://creativecommons.org/licenses/by/3.0/us/>).

The utricle senses motion in the horizontal plane, and the saccule senses motion in the vertical plane. All together the canals and the otolithic receptors respond to head motion and maintain static head position relative to gravity in all directions in 3D space.

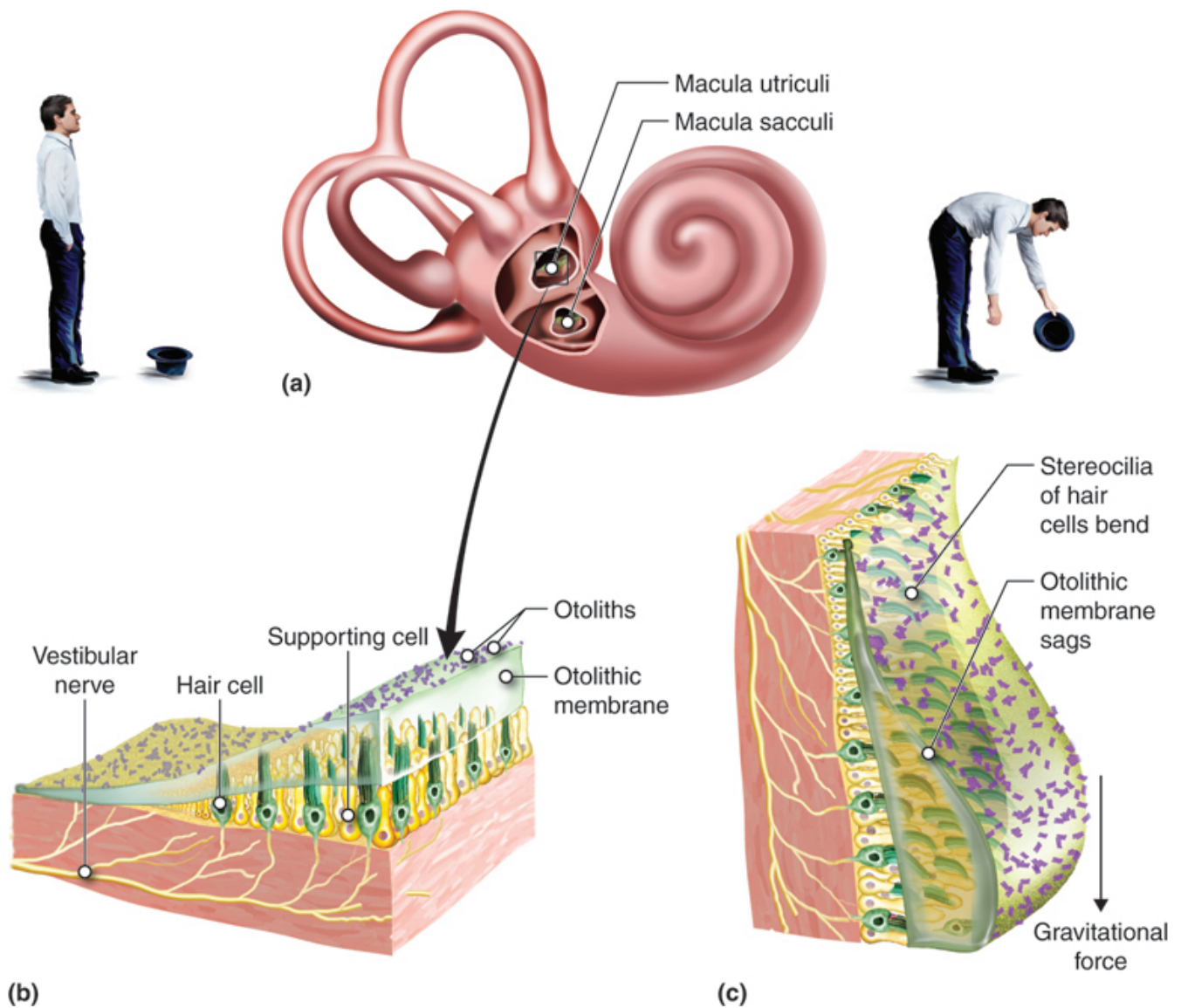


Figure 3.5.3. Structure of the Maculae. The macula utriculi (macula of the utricle) lies horizontally while the macula sacculi lies vertically (a). If the head is tilted, the dense otolithic membrane will cause the stereocilia of the hair cells to move from the straight position (b) to the bent position (c), sending signals to the central nervous system that the head has been tilted forward. This work by Cenvéo is licensed under a Creative Commons Attribution 3.0 United States (<http://creativecommons.org/licenses/by/3.0/us/>).

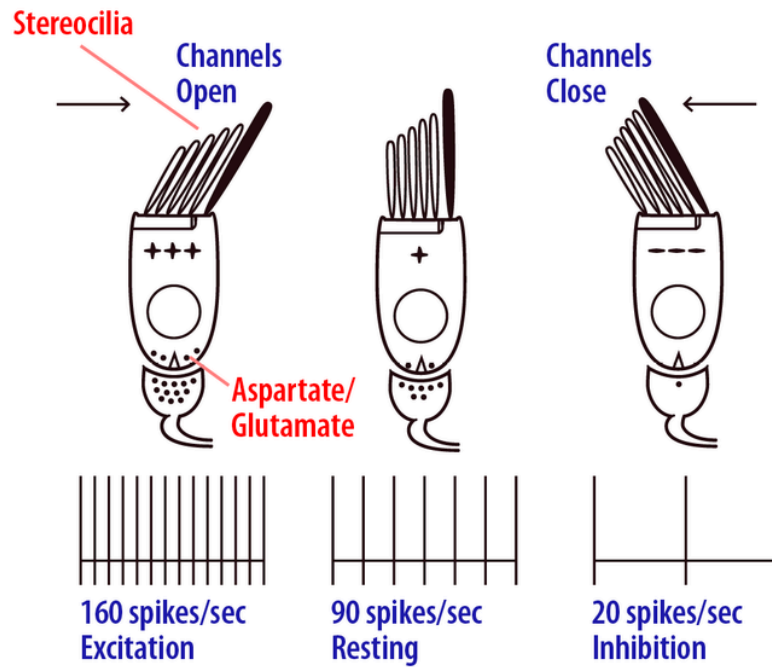


Fig. 3.5.4. Stereocilia in the semicircular canals, organized shortest to tallest, open or close the ion gated channel. (Provided by: Noba. License: CC-BY)

Within semicircular canals, an enlarged region called the ampulla holds hair cells that are able to respond to rotational movement. These cells have stereocilia, or actin-based protrusions required for hearing and balance, that project outward into a gel called the cupula. These stereocilia are staggered so the shortest protrusions are on one side of the cell while the taller protrusions are on the opposite side (Fig.3.5.1). When the head rotates, the fluid endolymph in the semicircular canals lags behind the head motion and pushes the cupula in the opposite direction of the head movement. If the cupula pushes the stereocilia toward the tall end, the ion gated channels open which depolarizes the cell and increases the release of neurotransmitters. Conversely, the cell becomes hyperpolarized when the stereocilia are pushed towards the shorter end. Since left and right ear semicircular canals have opposite polarity, by comparing the movements of the stereocilia, the sensory nerve fibers are able to send signals that allow the brain to detect head rotation. The otolith receptors have stereocilia that extend into a gelatin membrane that is covered by a layer of calcium carbonate crystals known as the otoconia. The otoconia are displaced by linear accelerations, not by fluid movement like in the semicircular canals. When otoconia move, they bend the hair cells and open or close the ion gated channels. The hair cells in the otolith layer are organized differently than those in the semicircular canals. The tallest stereocilia are pointed toward the center of the utricle and away from the center of the saccule.

Alcohol can cause a person to get the spins because alcohol interferes with the vestibular system. At least one semicircular canal is affected. Alcohol has a lighter density than the fluid in the semicircular canal, endolymph, and when alcohol is consumed, the alcohol changes the buoyancy of the endolymph. The cupula is more prone to shifting because of the lighter density of the endolymph and this shifting of the cupula causes the hair cells

to shift and send signals. Linear acceleration results in a shift in the cupula of the semicircular canals, and is interpreted as angular acceleration. This process can start at a fairly low blood alcohol content (BAC) and is a major reason why it is illegal to drive with a BAC of 0.08 or above.

The Khan Academy video linked here and included below provides some additional information about the vestibular system:



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NOBA, The Vestibular System.

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VISUAL CONTRIBUTIONS TO BALANCE

Learning Objectives

Be able to define optic flow

Understand what information optic flow provides that the vestibular system cannot

The utricle and the saccule sense head position, and the semicircular canals sense head movement, but balance is affected by more than just vestibular information. Both proprioceptive information (pressure sensors and kinesthetic information), and visual information become integrated and contribute to our sense of balance. Yet there is no place in the cerebral cortex that has been discovered thus far as being dedicated to interpreting balance. There are, however, vestibular nuclei in the brainstem (near 4th ventricle and brainstem) that receive vestibular information as well as proprioceptive and optic flow information.

Optic flow is the perceived visual motion of objects as the observer moves relative to them. To an observer driving a car, a sign on the side of the road would move from the center of his vision to the side, growing as he approached. If he had 360 degree vision, this sign would proceed to move quickly past his side to his back, where it would shrink. This motion of the sign is its optic flow.



Fig.3.6.1. Optic flow gives these planets the illusion of passing by as the image approaches the point of expansion. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY SA 4.0)

This allows a person to judge how close he is to certain objects, and how quickly he is approaching them. It is also useful for avoiding obstacles: if an object in front of an observer appears to be expanding but not moving, he is probably headed straight for it, but if it is expanding but moving slowly to the side, he will probably pass by it. Since optic flow relies only on relative motion, it remains the same when he is moving and the world remains still, and when he is standing still but everything he can see is moving past him. These properties have made the concept useful for robot designers writing visual navigation routines. It also appears to be used by certain insects, especially flying ones, where a large optic flow (indicating a quickly approaching obstacle) triggers muscles to move away.

When moving in a particular direction, an observer's optical flow field expands from a singular point, called the focus of expansion (Fig.3.6.1). This point of expansion tells us our heading; this is the one place in our visual field where there is no motion when we're moving (because we're moving straight toward it).

Optic flow thus provides us with information concerning coordinated motion in the visual field. Whereas semicircular canals respond to rotations and otolithic organs (utricle, saccule) sense linear accelerations, thus contributing to our sense of balance, optic flow tells us about our motion when we're not accelerating (i.e., when our vestibular and kinesthetic information is useless).

Exercises

1. True or False: Balance is only affected by vestibular information.
2. True or False: Optic flow applies when you are moving, and the world remains still, rather than when you are still, and the world is moving.
3. True or False: Optic flow helps someone determine how quickly an object is moving towards them.
4. Vestibular nuclei in the brainstem receive what kind of information?
 - A. Proprioceptive Information
 - B. Vestibular information
 - C. Optic Flow Information
 - D. All of the above
5. One example of optic flow is blank:
 - A. When you are able to detect an object from your peripheral vision
 - B. When you sit in front of a non-moving wall and stare at it
 - C. When you're sitting in a car and a train passes by, and the world around you (trees, buildings, etc.) appears to be moving backwards
 - D. When you lose your balance after you close your eyes and spin in a circle
6. Optic flow is necessary for balance because blank:
 - A. When we are not accelerating, our vestibular and proprioceptive information is useless
 - B. It sends signals to our brain when our eyes get fatigued, causing imbalance
 - C. It provides us with information concerning where light is coming from in the visual field
 - D. Its purpose is to determine what size a non-moving object is when the object is 5 feet or closer

Answer Key:

1. False.
2. False.
3. True.
4. D.
5. C.
6. A.

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Psychology Wiki, Optic Flow.

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References:

Huston, S. J., & Krapp, H. G. (2008). Visuomotor Transformation in the Fly Gaze Stabilization System. *PLoS Biology*, 6(7). doi: 10.1371/journal.pbio.0060173

VERTIGO

Learning Objectives

- Know how to define vertigo
- Describe the mechanism by which excess alcohol consumption results in vertigo

Vertigo is a condition in which a person has the sensation of moving or of surrounding objects moving when they are not.^[1] Often it feels like a spinning or swaying movement.^[2] This may be associated with nausea, vomiting, sweating, or difficulties walking. It is typically worse when the head is moved. Vertigo is the most common type of dizziness.

The most common disorders that result in vertigo are benign paroxysmal positional vertigo (BPPV), Ménière's disease, and labyrinthitis. Less common causes include stroke, brain tumors, brain injury, multiple sclerosis, migraines, trauma, and uneven pressures between the middle ears.^{[3][4]} Physiologic vertigo may occur following being exposed to motion for a prolonged period such as when on a ship or simply following spinning with the eyes closed.^{[5][6]} Other causes may include toxin exposures such as to carbon monoxide, alcohol, or aspirin.^[7] Vertigo typically indicates a problem in a part of the vestibular system. Other causes of dizziness include presyncope, disequilibrium, and non-specific dizziness.

Benign paroxysmal positional vertigo is more likely in someone who gets repeated episodes of vertigo with movement and is otherwise normal between these episodes.^[8] The episodes of vertigo should last less than one minute.^[2] The Dix-Hallpike test lowers a patient quickly to a supine (lying on your back) position and typically produces a period of rapid eye movements known as nystagmus in this condition. In Ménière's disease there is often ringing in the ears, hearing loss, and the attacks of vertigo last more than twenty minutes. In labyrinthitis the onset of vertigo is sudden and the nystagmus occurs without movement. In this condition vertigo can last for days.^[2] More severe causes should also be considered. This is especially true if other problems such as weakness, headache, double vision, or numbness occur.

Dizziness affects approximately 20–40% of people at some point in time, while about 7.5–10% have vertigo. About 5% have vertigo in a given year; it becomes more common with age and affects women two to three times more often than men.^[9] Vertigo accounts for about 2–3% of emergency department visits in the developed world.

Alcohol-Induced Spins

Alcohol-induced “spins” are a form of vertigo. Elevated blood alcohol content causes an increase in the density of the endolymph in the semi-circular canals, which throws the mechanics of the inner ear off. The cupula floats a bit, stimulating neurons that normally signal rotation. Thus, the sensation of spinning. Many people report that spins are worse when their eyes are closed. With your eyes open, your sense of balance has additional information (i.e., the lack of optic flow) to anchor your understanding of the world.

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Spins#:~:text=The%20alcohol%20apparently%20causes%20the,in%20addition%20to%20rotational%20acceleration.

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References

1. POST, RE; DICKERSON, LM (2010). “DIZZINESS: A DIAGNOSTIC APPROACH”. *AMERICAN FAMILY PHYSICIAN*. **82** (4): 361–369. PMID 20704166. ARCHIVED FROM THE ORIGINAL ON 2013-06-06.
2. HOGUE, JD (JUNE 2015). “OFFICE EVALUATION OF DIZZINESS”. *PRIMARY CARE: CLINICS IN OFFICE PRACTICE*. **42** (2): 249–258. DOI:10.1016/J.POP.2015.01.004. PMID 25979586.
3. WICKS, RE (JANUARY 1989). “ALTERNOBARIC VERTIGO: AN AEROMEDICAL REVIEW”. *AVIATION, SPACE, AND ENVIRONMENTAL MEDICINE*. **60** (1): 67–72. PMID 2647073.
4. BUTTARO, TERRY MAHAN; TRYBULSKI, JOANN; POLGAR-BAILEY, PATRICIA; SANDBERG-COOK, JOANNE (2012). *PRIMARY CARE – E-BOOK: A COLLABORATIVE PRACTICE* (4 ED.).

- ELSEVIER HEALTH SCIENCES. P. 354. ISBN 978-0323075855. ARCHIVED FROM THE ORIGINAL ON 2017-09-08.
5. FALVO, DONNA R. (2014). *MEDICAL AND PSYCHOSOCIAL ASPECTS OF CHRONIC ILLNESS AND DISABILITY* (5 ED.). BURLINGTON, MA: JONES & BARTLETT LEARNING.
P. 273. ISBN 9781449694425. ARCHIVED FROM THE ORIGINAL ON 2015-07-02.
 6. WARDLAW, JOANNA M. (2008). *CLINICAL NEUROLOGY*. LONDON: MANSON.
P. 107. ISBN 9781840765182. ARCHIVED FROM THE ORIGINAL ON 2015-07-02.
 7. GOEBEL, JOEL A. (2008). *PRACTICAL MANAGEMENT OF THE DIZZY PATIENT* (2ND ED.). PHILADELPHIA: LIPPINCOTT WILLIAMS & WILKINS. P. 97. ISBN 9780781765626. ARCHIVED FROM THE ORIGINAL ON 2015-07-02.
 8. KERBER, KA (2009). "VERTIGO AND DIZZINESS IN THE EMERGENCY DEPARTMENT". *EMERGENCY MEDICINE CLINICS OF NORTH AMERICA*. **27** (1): 39–50. DOI:10.1016/J.EMC.2008.09.002. PMC 2676794. PMID 19218018.
 9. NEUHAUSER HK, LEMPERT T (NOVEMBER 2009). "VERTIGO: EPIDEMIOLOGIC ASPECTS" (PDF). *SEMINARS IN NEUROLOGY*. **29**(5): 473–81. DOI:10.1055/S-0029-1241043. PMID 19834858.

MOTION SICKNESS

Learning Objectives

Understand what sensory cues are in conflict when people get motion sick

Be able to explain how postural instability is related to motion sickness

Although a number of conditions can produce motion sickness, it is generally thought that it is evoked from a mismatch in sensory cues between vestibular, visual, and proprioceptive signals (Yates, Miller, & Lucot, 1998). For example, reading a book in a car on a winding road can produce motion sickness, whereby the accelerations experienced by the vestibular system do not match the visual input. However, if one looks out the window at the scenery going by during the same travel, no sickness occurs because the visual and vestibular cues are in alignment.

Sea sickness, a form of motion sickness, appears to be a special case and arises from unusual vertical oscillatory and roll motion. Human studies have found that low frequency oscillations of 0.2 Hz and large amplitudes (such as found in large seas during a storm) are most likely to cause motion sickness, with higher frequencies offering little problems. At frequency oscillations of 2 Hz or above motion sickness ceases completely, although discomfort and injury can still occur. It's possible that high frequency oscillations happen so quickly that the vestibular system doesn't have time to properly function to send the proper signals to the brain, so no motion sickness occurs as the visual and vestibular cues are not clashing, but much more research is needed to determine a proper explanation to this phenomena.



Fig.3.7.1. Coast Guard. Lifeboat A common treatment of motion sickness is Dramamine, which helps to reduce the sensitivity of input from your vestibular system to the rest of your body. (Credit: Mike Baird. Provided by: Flickr. License:CC-BY 2.0)

It is often difficult to predict who will experience motion sickness and who won't. One thing that predicts motion sickness is postural instability. Normally, the muscles in our legs and our core body muscles automatically contract and relax, in response to sensory cues from proprioceptors in the muscles and tendons and pressure sensors on our feet, to automatically adjust our posture when the floor tilts. However, if the floor is moving in an unpredictable pattern, it is more difficult for us to keep stable and we sway. Tom Stoffregen, at the University of Minnesota, has shown that the amount a person sways in response to unpredictable motion of the floor predicts whether or not that person will experience motion sickness. The nature of any causal link between postural stability and motion sickness is not yet known; nor do we know whether this connection is mediated by cue conflict like the above examples. But it is one more clue to help us understand why people experience motion sickness.

Exercises

1. The sensory cues in conflict when someone experiences motion sickness are vestibular, visual, and _____.
 - A. Proprioceptive
 - B. Haptic
 - C. Auditory
 - D. Environmental
2. What frequency are you more likely to get sea sick while experiencing?
 - A. A low frequency and small amplitudes
 - B. A high frequency and small amplitudes
 - C. A low frequency and large amplitudes
 - D. A high frequency and large amplitudes
3. The muscles in our legs and core automatically _____, because of the sensory cues from _____.
 - A. Stay relaxed; proprioceptors
 - B. Contract and relax; proprioceptors
 - C. Stiffen up; visual cues
 - D. Strain themselves; auditory cues
4. True or False: The amount a person sways in response to unpredictable movements on the floor will determine if they experience any motion sickness.

Answer Key:

1. A
2. C
3. B
4. True

Authored by: Cheryl Olman

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NOBA, The Vestibular System.

Provided by: Baylor College of Medicine

URL: <https://nobaproject.com/textbooks/psychology-as-a-biological-science/modules/the-vestibular-system>

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References:

Bonnet, C. T., Faugloire, E., Riley, M. A., Bardy, B. G., & Stoffregen, T. A. (2006). Motion sickness preceded by unstable displacements of the center of pressure. *Human movement science*, 25(6), 800-820.

Villard, S. J., Flanagan, M. B., Albanese, G. M., & Stoffregen, T. A. (2008). Postural instability and motion sickness in a virtual moving room. *Human factors*, 50(2), 332-345.

Yates, B. J., Miller A. D., & Lucot, J. B. (1998). Physiological basis and pharmacology of motion sickness: An update. *Brain Res Bull*, 47, 395–406.

31.

ACTIVE LEARNING EXERCISE - CUE COMBINATION

We are learning that our brain spends a lot of time guessing about the structure of the world. All we have are a few sensations, and we have to put them together to understand what objects are out there. This exercise shows us how our brain uses inference to create perception.



One or more interactive elements has been excluded from this version of the text. You can view them online here: <https://pressbooks.umn.edu/sensationandperception/?p=2344>

SECTION IV

TASTE AND SMELL

Both taste and smell are considered chemosensations: ways we sense chemicals. The “chemicals” we’re sensing are molecules that float through the air (smell) or come from the things we put in our mouths (taste). Taste and smell are closely related — they combine to produce our perception of flavor, and they rely on receptors (neurons) that actually come into contact with the environment outside our body. The rest of our sensory neurons are protected from the world by a layer of skin or some other tissue. Because they’re protected, there is no process for regeneration, and once a neuron in your eyeball or inner ear dies, it doesn’t get replaced. But olfactory receptors and taste neurons both take a lot of abuse and new cells are born throughout our lives to replace the ones that die.

This chapter was created by Shawna Ratanpal, Rowan Sexton, Kori Skrypek, Brock Sorensen, Samantha Strubing, Barbora Tomancova, Savannah Vasek, Alex Wallace, Jacob Weckwerth, Maria Xiong, Peter Yong, and Grace Zellner.

OLFACTORY ANATOMY

Learning Objectives

Be able to locate the olfactory epithelium, the olfactory bulb, and the olfactory cortex

Be able to describe an olfactory neuron and explain what a cilium is

Understand that each olfactory neuron expresses only one type of receptor, and know approximately how many types of olfactory receptors there are

Like taste, the sense of smell, or olfaction, is also responsive to chemical stimuli. The olfactory receptor neurons are located in a small region within the superior nasal cavity (see Figure below). This region is referred to as the olfactory epithelium and contains bipolar sensory neurons. Each olfactory sensory neuron has dendrites (also called cilia) that extend from the apical surface of the epithelium into the mucus lining the cavity.

Cilia are small hair-like structures that can be found protruding from the olfactory receptor cells dendrite. The surface of these cilia is covered with olfactory receptors, also known as odorant receptors. An odorant will dissolve into the mucus of the olfactory epithelium and then bind to an olfactory receptor. Olfactory receptors can bind to a variety of odor molecules, with varying affinities. The difference in affinities causes differences in activation patterns resulting in unique odorant profiles.

As airborne molecules are inhaled through the nose, they pass over the olfactory epithelial region and dissolve into the mucus. These odorant molecules bind to proteins that keep them dissolved in the mucus and help transport them to the olfactory dendrites. The odorant–protein complex binds to a receptor protein within the cell membrane of an olfactory dendrite. These receptors are G protein–coupled, and will produce a graded membrane potential in the olfactory neurons.

The nasal epithelium, including the olfactory cells, can be harmed by airborne toxic chemicals. Therefore, the olfactory neurons are regularly replaced within the nasal epithelium, after which the axons of the new neurons must find their appropriate connections in the olfactory bulb. These new axons grow along the axons that are already in place in the cranial nerve.

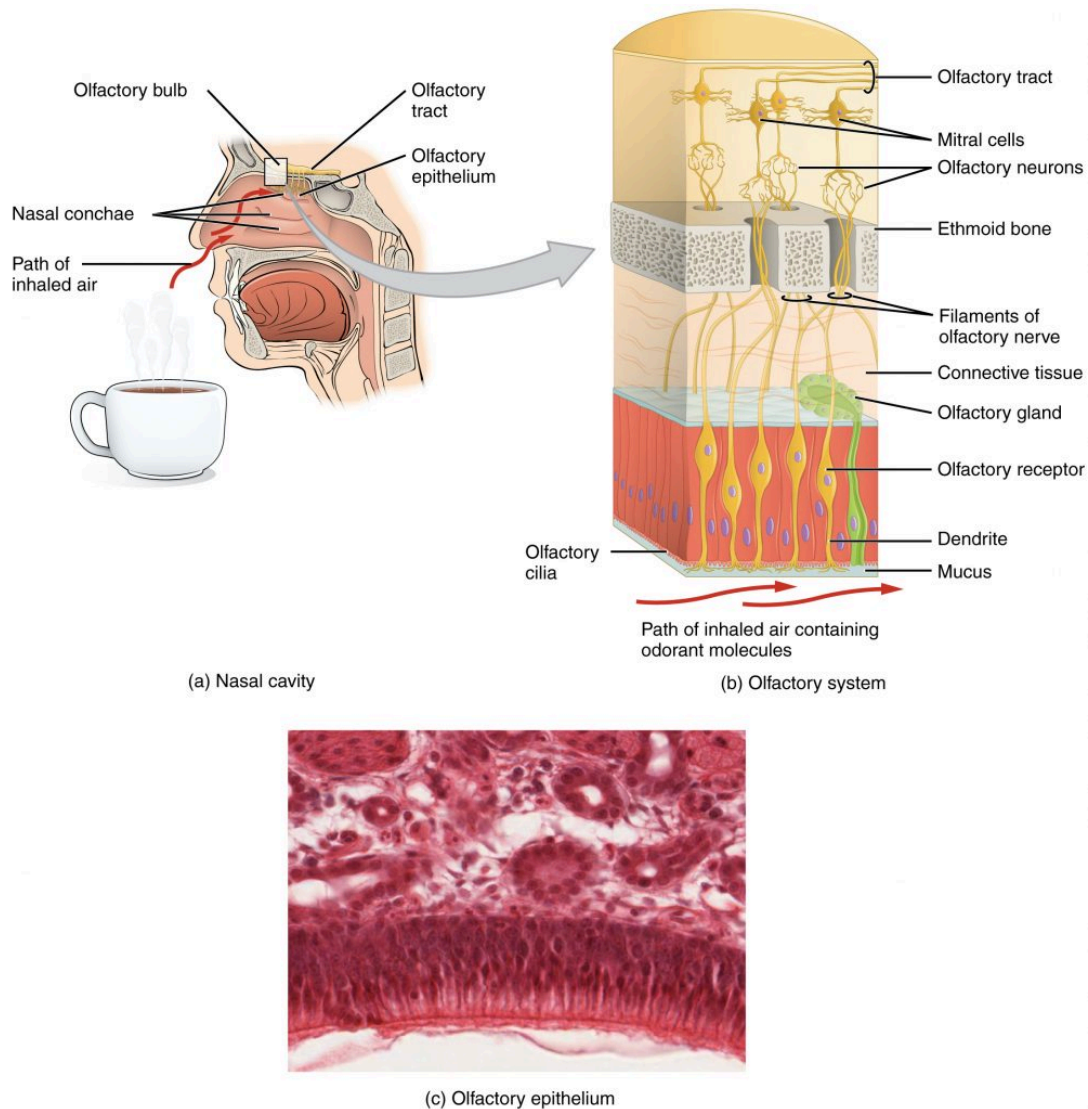


Fig.4.1.1. The Olfactory System. (a) The olfactory system begins in the peripheral structures of the nasal cavity. (b) The olfactory receptor neurons are within the olfactory epithelium. (c) Axons of the olfactory receptor neurons project through the cribriform plate of the ethmoid bone and synapse with the neurons of the olfactory bulb (tissue source: simian). (Credit: Micrograph provided by the Regents of University of Michigan Medical School © 2012. Provided by: OpenStax. License: CC-BY)

The axon of an olfactory neuron extends from the basal surface of the epithelium, through an olfactory foramen in the cribriform plate of the ethmoid bone, and into the brain (Fig.4.1.1). The first olfactory synapse in the brain occurs in a glomerulus in the olfactory bulb. A glomerulus is a cluster of synapses that collects information from olfactory neurons expressing a single type of receptor. There are about 350 different types of olfactory receptors, so there are about 350 different types of glomeruli spread throughout the olfactory bulb.

Each receptor responds with different strengths to different kinds of molecules, so the activity level of a single type of olfactory receptor neuron (and therefore an individual glomerulus) does not predict what something will smell like. But the pattern of responses across the different glomeruli in the olfactory bulb provides a code from which you can decipher what something smells like to an individual.

The group of axons called the olfactory tract connect to the olfactory bulb on the ventral surface of the frontal lobe. From there, the axons split to travel to several brain regions. Some travel to the cerebrum, specifically to the primary olfactory cortex that is located in piriform cortex (Fig.4.1.2) in the inferior and medial areas of the temporal lobe. Other axons from the olfactory bulb project to structures within the limbic system and hypothalamus, where smells become associated with long-term memory and emotional responses. This is how certain smells trigger emotional memories, such as the smell of food associated with one's birthplace. Smell is the one sensory modality that does not synapse in the thalamus before connecting to the cerebral cortex. This intimate connection between the olfactory system and the cerebral cortex is one reason why smell can be a potent trigger of memories and emotion.

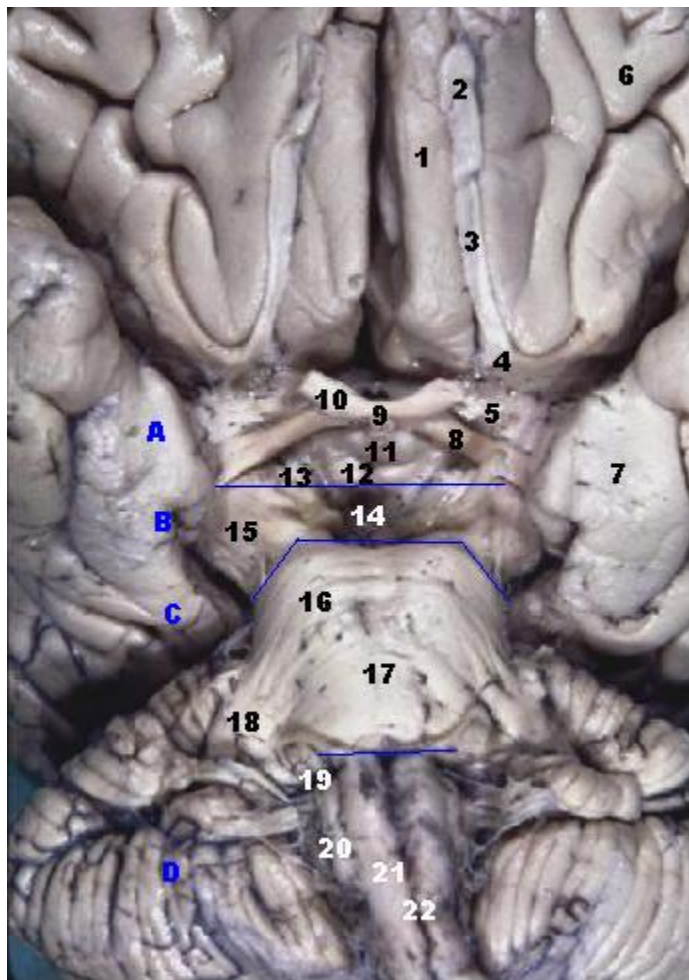


Fig.4.1.2. Location of Piriform Cortex. 1: Orbitofrontal gyrus. 2: Olfactory bulb. 3, 4: Olfactory tract. 7: Parahippocampal gyrus. 8, 9, 10: Optic tract, chiasm and nerve, respectively. Piriform cortex is just anterior to the parahippocampal gyrus, adjacent to the amygdala. (Credit: John A Beal, PhD. Provided by: Wikipedia. License: CC-BY 2.5)

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Wikipedia, Olfactory Receptor Neuron

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EXPERIENCING SCENT

Learning Objectives

Be able to describe the paradoxical relationship between scent and memory

Know the distinction between detection and recognition of a scent.

Know whether or not it is a myth that females have a better sense of smell than males

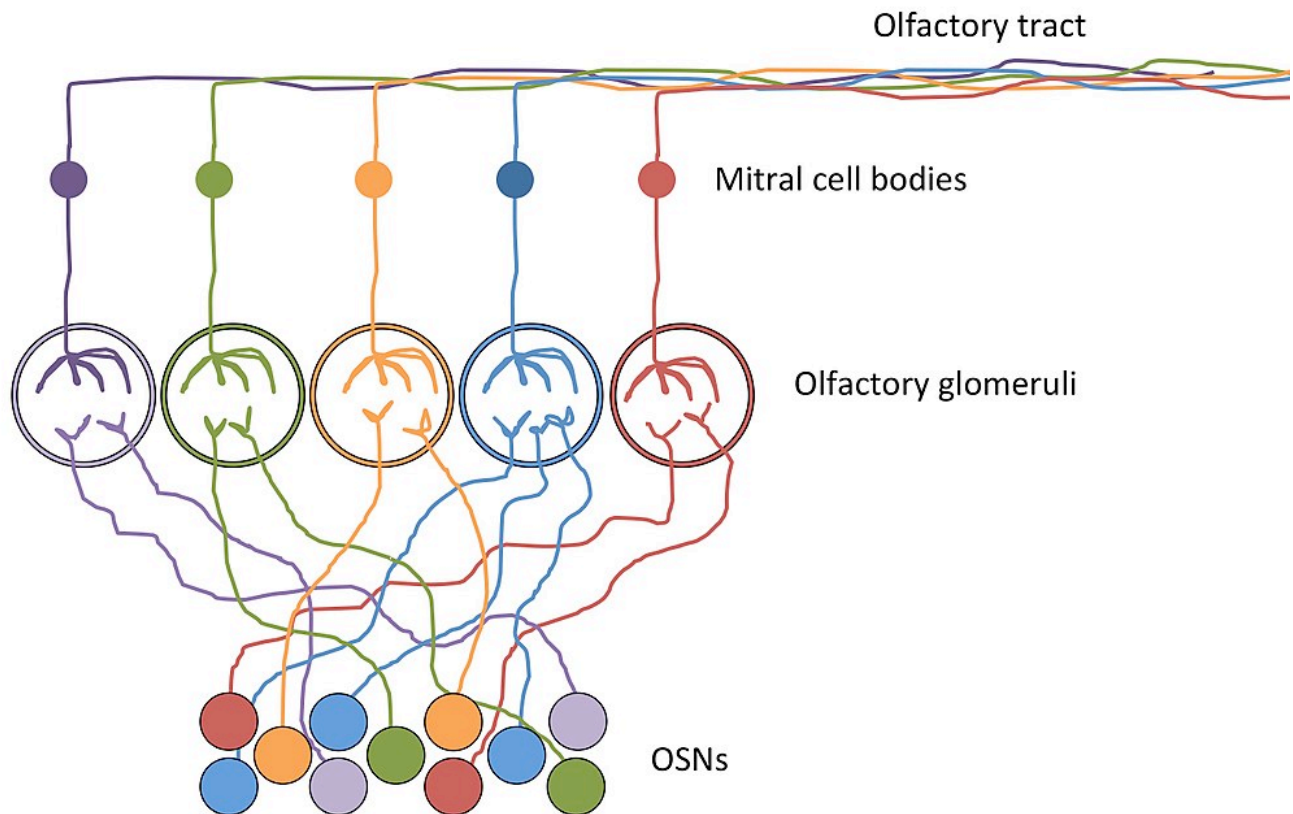
The strong projections from the olfactory tract to piriform cortex and other regions associated with memory (e.g., parahippocampal gyrus) and emotion (e.g., the amygdala) help us understand why smells readily evoke memories, and why the emotional content of smell-evoked memories is often stronger than other memories (Willander, 2007). However, the relationship is one-directional. While smells evoke memories, we're not always good at recognizing scents.

When studying detection of scents, there are two kinds of absolute thresholds that can be measured: detection thresholds and recognition thresholds. To measure detection thresholds, the participant is simply answering the question “can you smell something?” To measure recognition thresholds, the participant is answering the question “what can you smell?” Recognition thresholds are typically about 3 times larger than detection thresholds (Van Gemert, 2003).

Have you ever heard that women have a better sense of smell than men? Olfactory anatomy is the same for both sexes, but there is some experimental evidence that females perform slightly better at standardized smell tests than males (Sorokowski, 2019). The effect size is very small; differences in performance are likely related to experience (some bias might occur in testing if one sex is more likely to have experience with test scents than others) and possibly sex hormones.

Predicting how something is going to smell to someone is difficult — each molecule activates several different receptors, and each receptor might respond to several different molecules. In addition, most things we smell have several different molecules in them, each of which is going to activate several different receptors. So the olfactory code that goes to the brain is complicated, and each of us has a different set of experiences

that means our brain interprets these patterns differently. This interpretation is done in piriform cortex, on the bottom of the temporal lobe (not far from the parts of the brain responsible for forming memories!). We can't look at a single molecule and predict what it is going to smell like to someone. But we can look at the pattern of responses across mitral cells (which get their information from bundles of synapses called glomeruli), and if the pattern is the same for two scents (for example, in the figure below, lots of action potentials from the orange mitral cell, and a few from the blue one), then the person smelling the two scents will say they smell the same.



Mapping of olfactory information from olfactory sensory neurons (OSNs) to mitral cells. A glomerulus is a cluster of synapses. Each mitral cell receives input from OSNs with the same molecular receptor type. This amplifies sensitivity. Noodle brain, CC BY-SA 4.0, via Wikimedia Commons.

Authored by: Samantha Strubing and Cheryl Olman
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Sorokowski, P., Karwowski, M., Misiak, M., Marczak, M. K., Dziekan, M., Hummel, T., & Sorokowska, A. (2019). Sex differences in human olfaction: A meta-analysis. *Frontiers in psychology, 10*, 242.

Willander, J., & Larsson, M. (2007). Olfaction and emotion: The case of autobiographical memory. *Memory & cognition, 35*(7), 1659-1663.

Van Gemert, L. J. (2003). Odour thresholds. Compilations of odour threshold values in air, water and other media. Oliemans, Punter & Partners BV, Utrecht.

PHEROMONES

Learning Objectives

Know what the vomeronasal gland is and whether or not people have one

Be able to cite arguments for and against the presence of pheromone communication in humans

The vomeronasal organ (VNO, or Jacobson's organ) is a tubular, fluid-filled, olfactory organ present in many vertebrate animals that sits adjacent to the nasal cavity. It is very sensitive to pheromones and is connected to the nasal cavity by a duct. When molecules dissolve in the mucosa of the nasal cavity, they then enter the VNO where the pheromone molecules among them bind with specialized pheromone receptors. Upon exposure to pheromones from their own species or others, many animals, including cats, may display the flehmen response (Fig.4.3.1), a curling of the upper lip that helps pheromone molecules enter the VNO. Humans do not have a vomeronasal organ.



Fig.4.3.1. Tiger Nose. The flehmen response in this tiger results in the curling of the upper lip and helps airborne pheromone molecules enter the vomeronasal organ. (Credit: "Chadh"/Flickr. Provided by: OpenStax. Modified by: OpenStax. License: CC-BY)

A pheromone is a secreted chemical signal used to obtain a response from another individual of the same species. The purpose of pheromones is to elicit a specific behavior from the receiving individual. Pheromones are especially common among social insects, but they are used by many species to attract the opposite sex, to sound alarms, to mark food trails, and to elicit other, more complex behaviors. Even humans are thought to respond to certain pheromones called axillary steroids. These chemicals influence human perception of other people, and in one study were responsible for a group of women synchronizing their menstrual cycles (McClintock, 1971, but also see Strassman, 1999). The role of pheromones in human-to-human communication is not fully understood and continues to be researched.

Pheromonal signals are sent, not to the main olfactory bulb, but to a different neural structure that projects directly to the amygdala (recall that the amygdala is a brain center important in emotional reactions, such as fear). The pheromonal signal then continues to areas of the hypothalamus that are key to reproductive physiology and behavior. While some scientists assert that the VNO is apparently functionally vestigial in humans, even though there is a similar structure located near human nasal cavities, others are researching it as a possible functional system that may, for example, contribute to synchronization of menstrual cycles in women living in close proximity.

Is the effect associated with olfaction ever hard-wired? Pheromones are said to be olfactory molecules that evoke specific behaviors. Googling “human pheromone” will take you to websites selling various sprays that are supposed to make one more sexually appealing. However, careful research does not support such claims in

humans or any other mammals (Doty, 2010). For example, amniotic fluid was at one time believed to contain a pheromone that attracted rat pups to their mother's nipples so they could suckle. Early interest in identifying the molecule that acted as that pheromone gave way to understanding that the behavior was learned when a novel odorant, citral (which smells like lemons), was easily substituted for amniotic fluid (Pedersen, Williams, & Blass, 1982).

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References:

McClintock, M. K. (1971). Menstrual synchrony and suppression. *Nature*, 229(5282), 244-245..

Strassmann, B. I. (1999). Menstrual synchrony pheromones: cause for doubt. *Human Reproduction*, 14(3), 579-580.

THE STRUCTURE OF THE TONGUE

Learning Objectives

Know the difference between a taste bud and a papilla.

Be able to name the different kinds of papillae.

Gustation is the special sense associated with the tongue. The surface of the tongue, along with the rest of the oral cavity, is lined by a stratified squamous epithelium. Raised bumps called papillae (singular = papilla) contain the structures for gustatory transduction. There are four types of papillae, based on their appearance: circumvallate, foliate, filiform, and fungiform (Fig 4.4.1). Within the structure of the papillae are taste buds that contain specialized gustatory receptor cells for the transduction of taste stimuli. These receptor cells are sensitive to the chemicals contained within foods that are ingested, and they release neurotransmitters based on the amount of the chemical in the food. Neurotransmitters from the gustatory cells can activate sensory neurons in the facial, glossopharyngeal, and vagus cranial nerves.

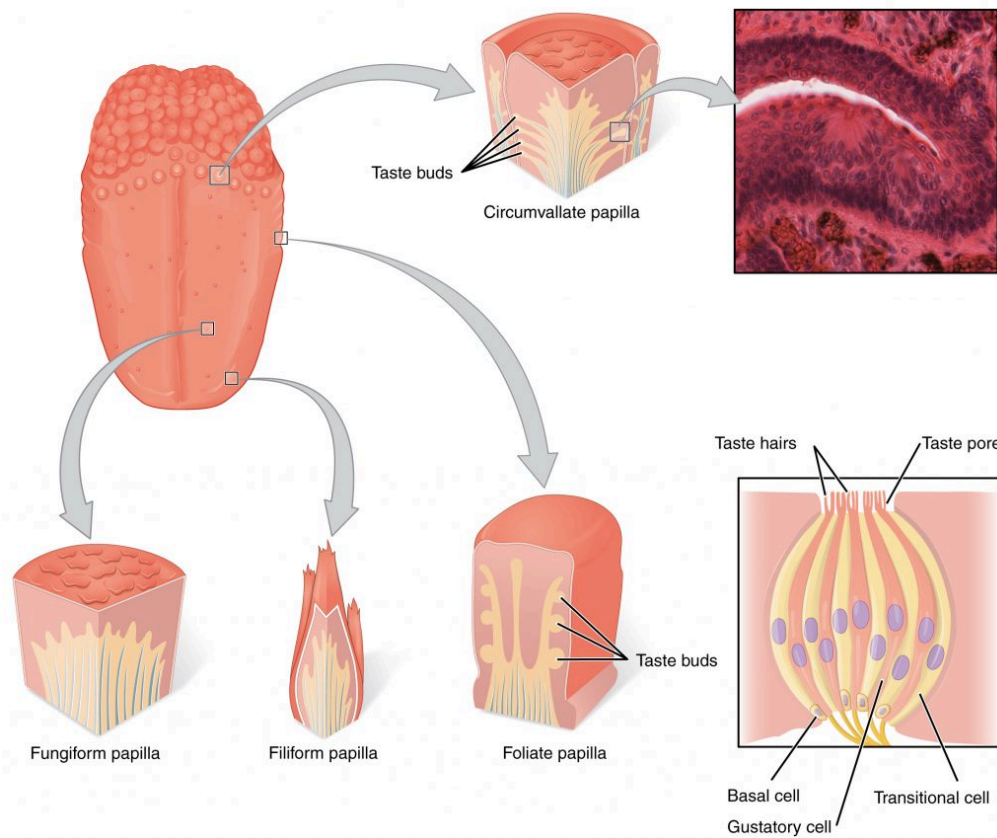


Fig.4.4.1. Tongue. The tongue is covered with small bumps, called papillae, which contain taste buds that are sensitive to chemicals in ingested food or drink. Different types of papillae are found in different regions of the tongue. The taste buds contain specialized gustatory receptor cells that respond to chemical stimuli dissolved in saliva. (Credit: Micrograph provided by the Regents of University of Michigan Medical School. Provided by: Openstax. License: CC-BY)

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36.

THE DIMENSIONS OF TASTE

Learning Objectives

Be able to describe 6 different dimensions of taste.

Know which 2 dimensions of taste rely on the detection of single ions rather than entire molecules

Dimensions of Taste

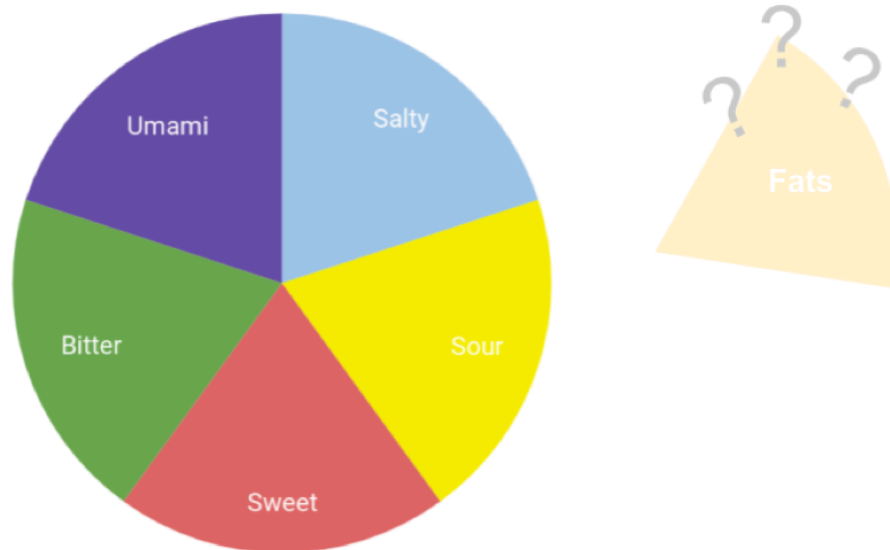


Fig.4.5.1. Flavors. While scientists haven't all come to the conclusion that fats are a dimension of taste, there are many valid arguments to be made in its case. Here we see a visualization of the agreed-upon five dimensions: Salty, Sour, Sweet, Bitter, and Umami, with Fats separate but still relevant. (Credit: Rowan Sexton. Provided by: University of Minnesota. License: CC-BY)

Only a few recognized sub-modalities exist within the sense of taste, or gustation. Until recently, only four tastes were recognized: sweet, salty, sour, and bitter. Research at the turn of the 20th century led to recognition of the fifth taste, umami, during the mid-1980s. Umami is a Japanese word that means “delicious taste,” and is often translated to mean savory. Very recent research has suggested that there may also be a sixth taste for fats, or lipids.

Salty taste is simply the perception of sodium ions (Na^+) in the saliva. When you eat something salty, the salt crystals dissociate into the component ions Na^+ and Cl^- , which dissolve into the saliva in your mouth. The Na^+ concentration becomes high outside the gustatory cells, creating a strong concentration gradient that drives the diffusion of the ion into the cells. The entry of Na^+ into these cells results in the depolarization of the cell membrane and the generation of a receptor potential.

Sour taste is the perception of H^+ concentration. Just as with sodium ions in salty flavors, these hydrogen ions enter the cell and trigger depolarization. Sour flavors are, essentially, the perception of acids in our food. Increasing hydrogen ion concentrations in the saliva (lowering saliva pH) triggers progressively stronger graded potentials in the gustatory cells. For example, orange juice—which contains citric acid—will taste sour because it has a pH value of approximately 3. Of course, it is often sweetened so that the sour taste is masked.

The first two tastes (salty and sour) are triggered by the cations Na^+ and H^+ . The other tastes result from food molecules binding to a G protein–coupled receptor. A G protein signal transduction system ultimately leads to depolarization of the gustatory cell.

Sweet taste is the sensitivity of gustatory cells to the presence of glucose dissolved in the saliva. Other monosaccharides such as fructose, or artificial sweeteners such as aspartame (NutraSweet™), saccharine, or sucralose (Splenda™) also activate the sweet receptors. The affinity for each of these molecules varies, and some will taste sweeter than glucose because they bind to the G protein–coupled receptor differently.

Bitter taste is similar to sweet in that food molecules bind to G protein–coupled receptors. However, there are a number of different ways in which this can happen because there are a large diversity of bitter-tasting molecules. Some bitter molecules depolarize gustatory cells, whereas others hyperpolarize gustatory cells. Likewise, some bitter molecules increase G protein activation within the gustatory cells, whereas other bitter molecules decrease G protein activation. The specific response depends on which molecule is binding to the receptor. One major group of bitter-tasting molecules are alkaloids. Alkaloids are nitrogen containing molecules that are commonly found in bitter-tasting plant products, such as coffee, hops (in beer), tannins (in wine), tea, and aspirin. By containing toxic alkaloids, the plant is less susceptible to microbe infection and less attractive to herbivores. Therefore, the function of bitter taste may primarily be related to stimulating the gag reflex to avoid ingesting poisons. Because of this, many bitter foods that are normally ingested are often combined with a sweet component to make them more palatable (cream and sugar in coffee, for example). The highest concentration of bitter receptors appear to be in the posterior tongue, where a gag reflex could still spit out poisonous food.

Umami taste is often referred to as the savory taste. Like sweet and bitter, it is based on the activation of G protein–coupled receptors by a specific molecule. The molecule that activates this receptor is the amino acid

L-glutamate. Therefore, the umami flavor is often perceived while eating protein-rich foods. Not surprisingly, dishes that contain meat are often described as savory. For example, foods that have a strong umami flavor include broths, gravies, soups, shellfish, fish (including fish sauce and preserved fish such as maldive fish), tomatoes, mushrooms, hydrolyzed vegetable protein, meat extract, yeast extract, cheeses, and soy sauce.

Fat taste elicited by the digestive products of fat (fatty acids) is yet to be confirmed; however, a growing body of evidence from humans and other animal species provides support for this proposition. In support for a functional significance of fat taste, differences in taste sensitivity for fat appear to predict certain dietary behaviours, i.e. decreased sensitivity to fat taste is associated with an increased consumption of fat, and this has been reported in both animal and human studies. Moreover, sensitivity to fat can be modulated by the diet, i.e. consumption of a high-fat diet appears to maximise the body's capacity for fat absorption, with no changes in appetite, suggesting that such changes may accompany or encourage excess fat intake and obesity. These data propose a direct role of the taste system in the consumption and preference of high-fat foods, which may be linked to the development of obesity given that differences in BMI have also been linked to oral fatty acid sensitivity. The mechanism allowing for increased consumption of fat is proposed to be via satiety or fullness signals, as associations in both taste and digestive responses to fat have been reported. The next 5 to 10 years should reveal, conclusively, whether fat can be classified as the sixth taste, but no matter what, there appears to be a functional significance to oral chemosensing of fats.

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BioMed Central, Is fat the sixth taste primary? Evidence and implications

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Wikipedia, Umami

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References

Keast, R.S., Costanzo, A. Is fat the sixth taste primary? Evidence and implications. *Flavour* 4, 5 (2015).
<https://doi.org/10.1186/2044-7248-4-5>

SUPERTASTERS

Learning Objectives

Be able to describe what it means to be a non-taster, a taster, or a supertaster

Know what is the typical distribution of tasters and supertasters in a population

Supertasters 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) or PTC (Phenylthiocarbamide).

A supertaster is a person who is able to taste things to a more extreme level. This is a result from the extra number of fungiform papillae on the tongue. With this extra amount of papillae, the individual is able to taste things better and more potently. A non-taster would be a person with much less fungiform papillae on their tongue, resulting in a lesser sense of taste. A taster is what is considered in between a non-taster and a supertaster.

In the 1960's Roland Fischer was the first to link the ability to taste PTC to food preference and body type. Today, PROP has replaced PTC in taste research because of a faint sulfurous odor and safety concerns with PTC. Most estimates suggest that 25% of the population are nontasters, 50% are medium tasters, and 25% are supertasters. Women are more likely to be supertasters, as are those from Asia, South America, and Africa. Female supertasters have a lower body mass index and predominant cardiovascular health. This can be due to the fact that supertasters do not have a high predilection for sweet or high-fat foods.

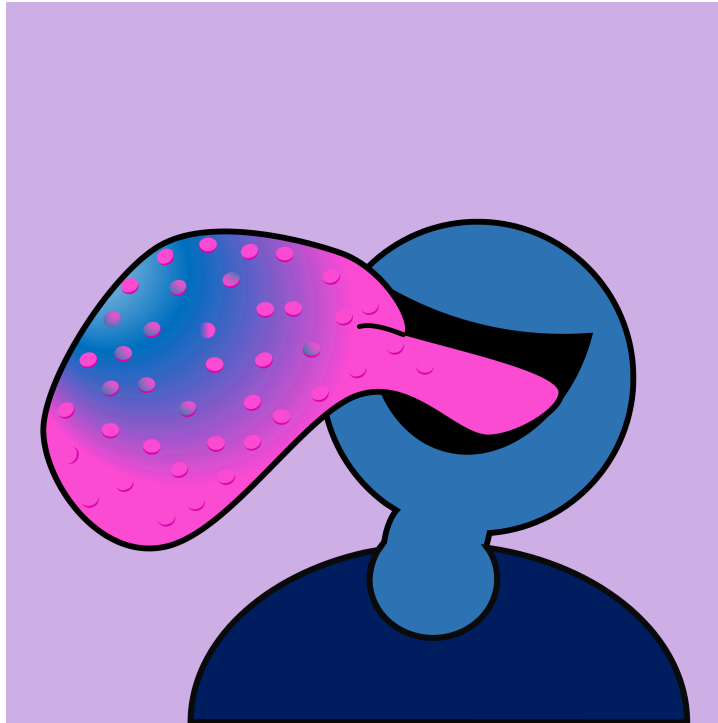


Fig.4.6.1. In this figure, you can visualize the difference between a supertaster and a nontaster. The supertaster has many more papillae than the nontaster evidently. You can see why someone who is a supertaster would be more picky in their eating choices, as they have an overwhelming amount of senses reacting to what they put in their mouth. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

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Wikipedia, Supertaster

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38.

TASTE PATHWAYS

Learning Objectives

Know where taste information first reaches the brain.

Know where the nucleus of the solitary tract is and what role it plays in relaying taste information to the brain.

Taste buds are formed by groupings of taste receptor cells. Receptor cells protrude into the central pore of the taste bud. Chemical changes within sensory cells, from the taste molecules binding onto the taste receptors, will result in neural impulses that transfer to the brain through other nerves. Where these neural impulses transfer to will depend on where the receptor is located, such as the posterior two thirds of the tongue. Taste information is transmitted to the medulla, thalamus, limbic system, and to the gustatory cortex, which is tucked underneath the overlap between the frontal and temporal lobes (Maffei, Haley, & Fontanini, 2012; Roper, 2013).

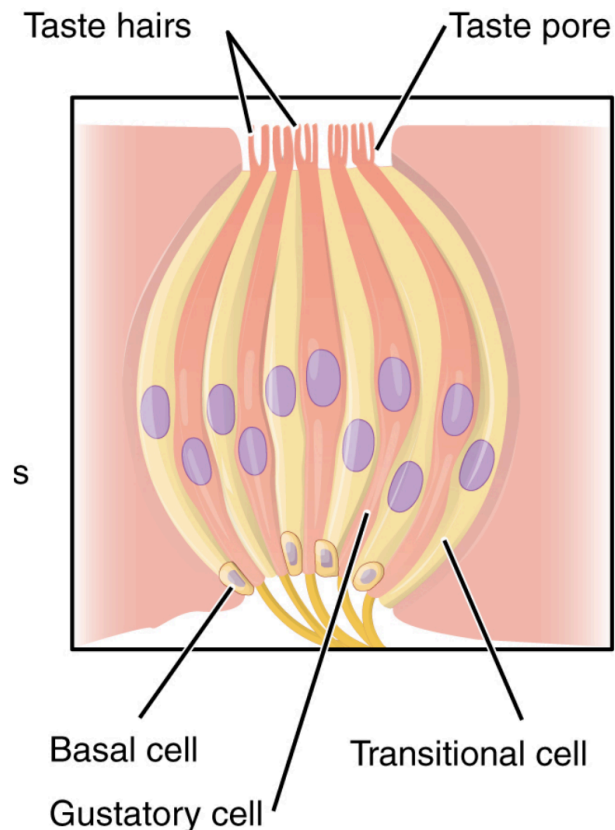


Figure 4.7.1. Taste Buds. Taste buds are composed of multiple taste receptor cells. Unlike olfactory neurons, taste cells do not have axons. Taste buds contain gustatory cells, basal cells, and transitional cells. The gustatory cells contain hair like cells called microvilli that extend through the taste pore in the taste buds. These taste cells transmit information by using secondary afferent neurons to make synaptic contact with taste cells to transmit information to the thalamus.(Credit: Cheryl Olman, own photo. CC-BY-4.0)

Due to the fact that taste cells have no axons, secondary afferent neurons with cell bodies in the nucleus of the solitary tract, which is located in the medulla of the brain stem, make synaptic contact with taste cells. Depending on where these cells and neurons come from, they will terminate in specific parts of the solitary nucleus. After termination at the solitary nucleus, the new information will relay data to the thalamus. In addition, information from the thalamus is transported to the frontal operculum and insular cortex, which is the primary taste area of the brain.

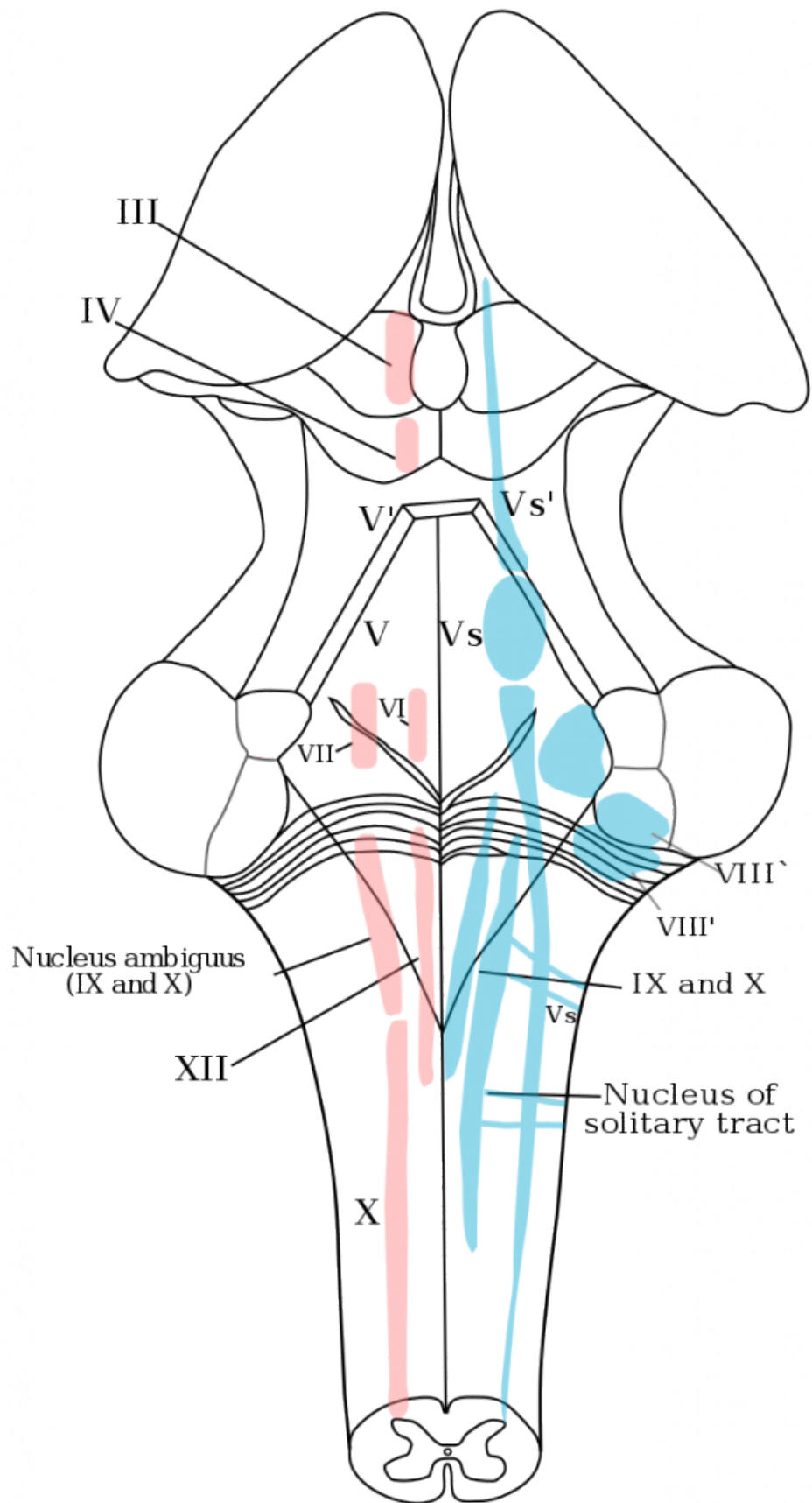


Fig.4.7.2. Taste Data. Location of the nucleus of the solitary tract and insular

cortex (primary gustatory cortex). The NST is very low in the brainstem. It is an important site for the modification of taste information either by blood glucose (which inhibits responses) or by feedback from cortical regions like the orbitofrontal cortex. From the NST, taste information continues to the thalamus. (Credit: Mcstrother. Provided by: Wikipedia. License: Public Domain)

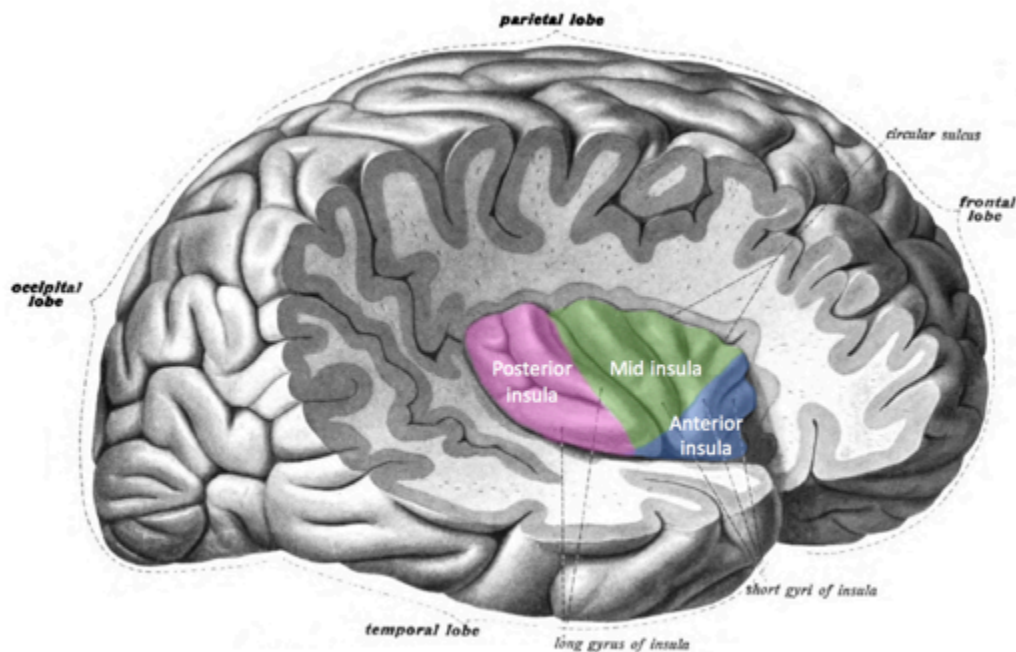


Fig.4.7.3. The insular cortex is tucked behind frontal and temporal cortex, which is cut away in this illustration. Insular cortex is involved in more than just our sense of taste; it receives input from all over the brain with information about how our body is doing. The dorsal (top) part of the mid-insula is where taste information from the thalamus is represented in the brain. (Credit: Schappelle. Provided by: Wikimedia Commons. License: CC-BY SA 4.0)

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FLAVOR

Learning Objectives

Understand how the sense of smell contributes to our perception of flavor.

Understand why infants have appetitive responses to sweetness and aversive responses to bitterness.

Understand how taste and smell are affected by aging.

Taste (gustation) and smell (olfaction) are considered chemical senses because both have sensory receptors that respond to molecules in the food we eat and/or in the air we breathe. There is a pronounced interaction between our chemical senses in the orbitofrontal cortex (OFC). When we describe the flavor of a given food, we are really referring to both gustatory and olfactory properties of the food working in combination.

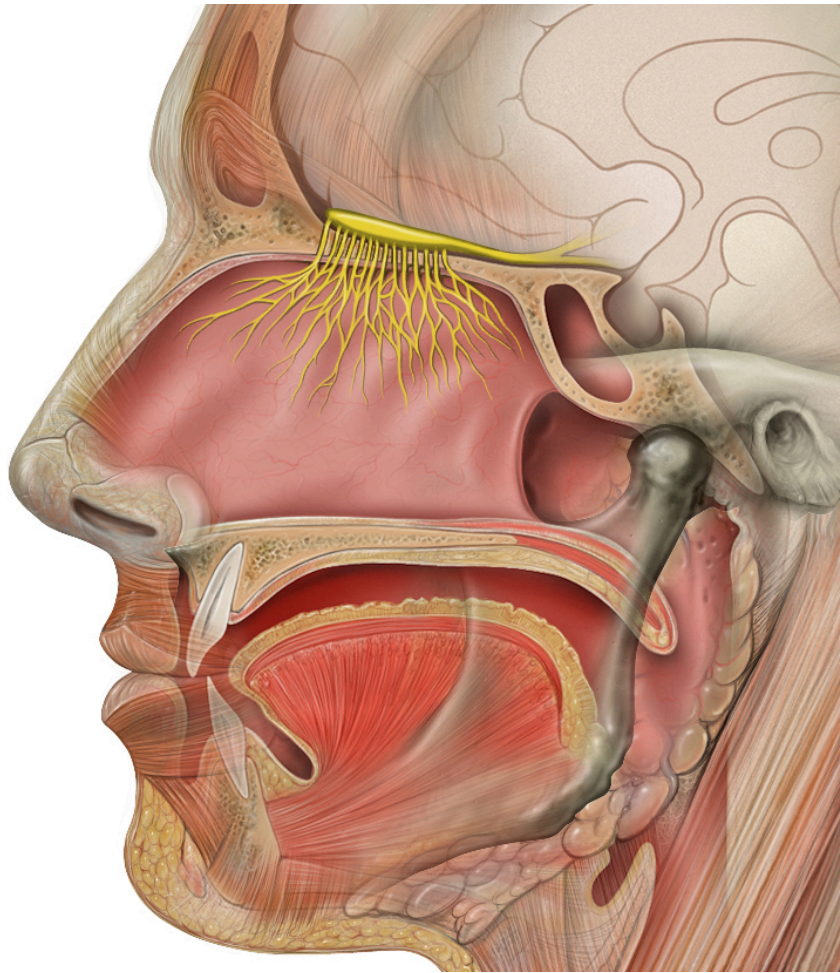


Fig.4.8.1. Scent in Flavor. Retronasal olfaction reaches the nose through the opening at the back of the throat. If you have ever started laughing while drinking water, you may recall it going up in your nose. This pathway is how the water would have gotten there. (Credit: Patrick J. Lynch. Provided by: Wikipedia. License: CC-BY)

When we chew and swallow food, the odorants emitted by the food are forced up behind the palate (roof of the mouth) and enter our noses from the back; this is called “retronasal olfaction.” Ortho and retronasal olfaction involve the same odor molecules and the same olfactory receptors; however, the brain can tell the difference between the two and does not send the input to the same areas. Retronasal olfaction and taste project to some common areas where they are presumably integrated into flavor. Flavors tell us about the food we are eating.

The pleasure associated with sweet and salty and the displeasure associated with sour and bitter are hard-wired in the brain. Newborns love sweet (taste of mother’s milk) and hate bitter (poisons) immediately. The receptors mediating salty taste are not mature in humans at birth.. Sour is generally disliked (possibly the brain’s way of protecting against tissue damage from acid). This hard-wired effect is the main characteristic of taste and is the reason why we only classify these four qualities as “basic tastes. Our sense of taste evolves across our lifetime as we learn to enjoy all kinds of flavors in finding out which are indeed safe to eat. While the

number of taste buds does not decline until after 75 years of age, there is a general decrease in taste perception as part of the aging process. Other factors that may contribute to the reduction in taste in older persons are a decrease in the volume of saliva secreted, an increase in the viscosity of saliva, and the formation of fissures and furrows on the tongue.

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APPETITE

Learning Objectives

Be able to describe how orbitofrontal cortex impacts appetite.

Understand why it matters that blood sugar inhibits responses in the nucleus of the solitary tract.

Understand why texture affects appetite.

The control of appetite and food intake can be divided into “homeostatic” and “non-homeostatic” control. The alteration in consumption of food that follows sensing of energy balance forms the basis of the homeostatic control of appetite: following a meal, appetite is suppressed, whereas, following significant energy expenditure, we feel hungry. Postprandial satiety signals include changes in circulating concentrations of nutrients and gut hormones.

Key areas include the amygdala, hippocampus, insula, striatum, and orbitofrontal cortex (OFC), although this list is by no means exhaustive. It is now widely accepted that, with regard to appetite control, homeostatic and non-homeostatic systems do not function independently; instead, there is extensive cross-modulation between them, along with a complex integration of inputs before a final decision is made regarding food consumption.

Glucose-sensing vagal and sympathetic afferents arise from the liver and gastrointestinal tract; these convey information to the nucleus of the solitary tract (NTS). Unlike their sympathetic sensory counter-parts arising near the portal vein, vagal glucose sensors probably do not contribute the counter-regulatory response. Carotid body glucose sensors may also contribute to the counter-regulatory response. Information is relayed from NTS to the dorsal motor nucleus of the vagus (DMV) which provides parasympathetic drive to the pancreatic islets and via the parabrachial nucleus (PBN) to supramedullary structures.

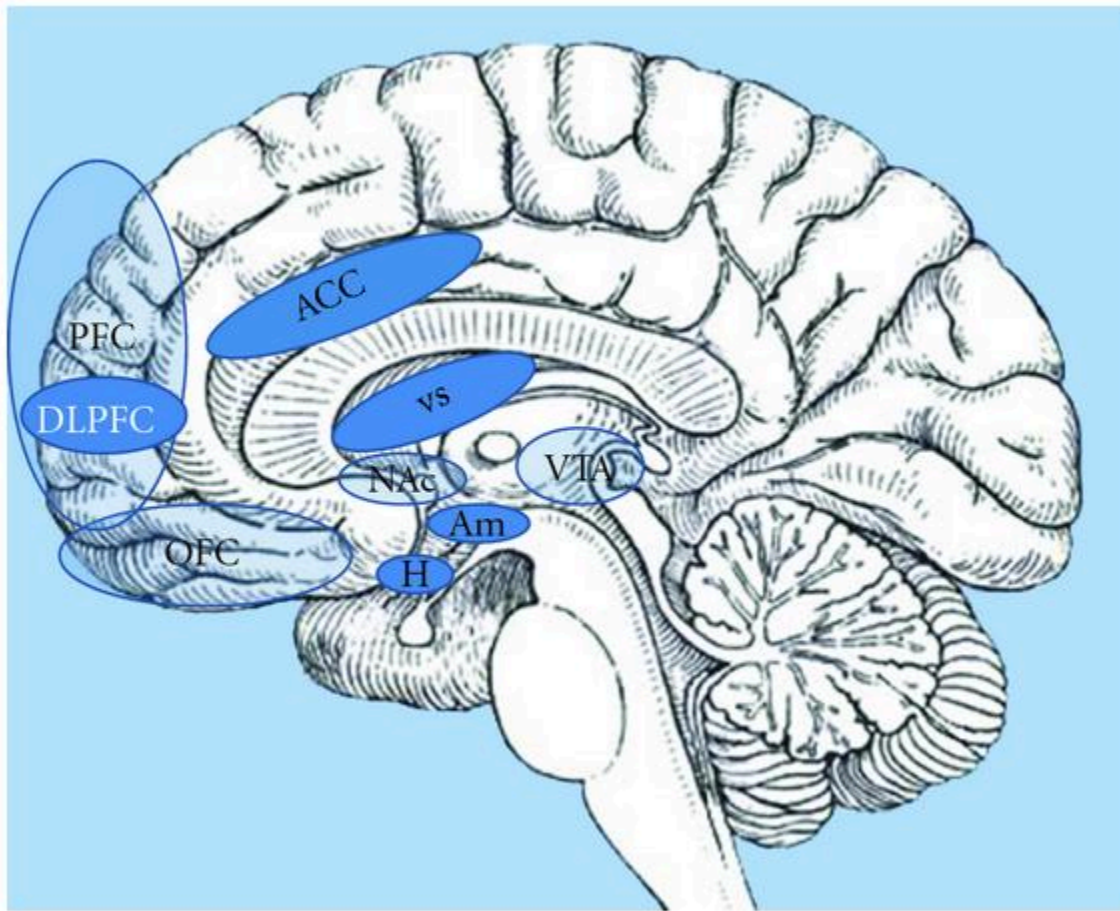


Fig.4.9.1. All of the brain that deal with appetite, nucleus accumbens (Nac): anticipatory reward processing; ventral tegmental area (VTA): numerous dopaminergic projections to other limbic areas; ventral striatum (VS): motivation reward; expectancy and novelty processing; anterior cingulate cortex (ACC): decision making; orbitofrontal cortex (OFC): reward encoding; prefrontal cortex (PFC): translation of external and internal cues into behavioural responses; dorsolateral prefrontal cortex (DLPFC): self-control. (credit: Akila De Silva et al, 2012)

Texture affects appetite on the basis of complexity. Now what exactly does complexity mean in regards to food texture? A food with a complex texture, like a salad with berries and nuts, will create more neural responses while chewing. (It often also takes longer to chew as well.) So your brain feels like it has eaten more, leaving you feeling full after eating less. On the other side of the coin, it is easier to eat more food with little texture. For example, a smoothie with the same amount of spinach, berries, and nuts as the salad from before would be easier to finish due to its lower textural complexity.

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“The Use of Functional MRI to Study Appetite Control in the CNS,” Akila De Silva et al.

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“Neural pathways that control the glucose counterregulatory response,” Anthony J. M. Verberne et. al

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Reference

Larsen DS, Tang J, Ferguson LR, James BJ. Increased textural complexity in food enhances satiation. *Appetite*. 2016;105:189-194. doi:10.1016/j.appet.2016.05.029

41.

ANOSMIA

Learning Objectives

Be able to define anosmia and give two examples of why it happens.

Understand why anosmia is dangerous.

Sometimes, certain conditions can lead to the loss of sense of smell. Anosmia, also known as smell blindness, is the loss of the ability to detect one or more smells. Anosmia may be temporary or permanent. It differs from hyposmia, which is a decreased sensitivity to some or all smells.

The loss of olfactory sensation is generally caused by the loss of the olfactory nerve, such as by blunt force trauma. When the frontal lobe of the brain moves relative to the ethmoid bone, the olfactory tract axons may be sheared apart. Therefore, professional fighters often experience anosmia because of repeated trauma to the face and head.

In addition, certain pharmaceuticals, such as antibiotics, can cause anosmia by killing all the olfactory neurons at once. If no axons are in place within the olfactory nerve, then the axons from newly formed olfactory neurons have no guide to lead them to their connections within the olfactory bulb. There are temporary causes of anosmia, as well, such as those caused by inflammatory responses related to respiratory infections or allergies (Fig.4.10.1).

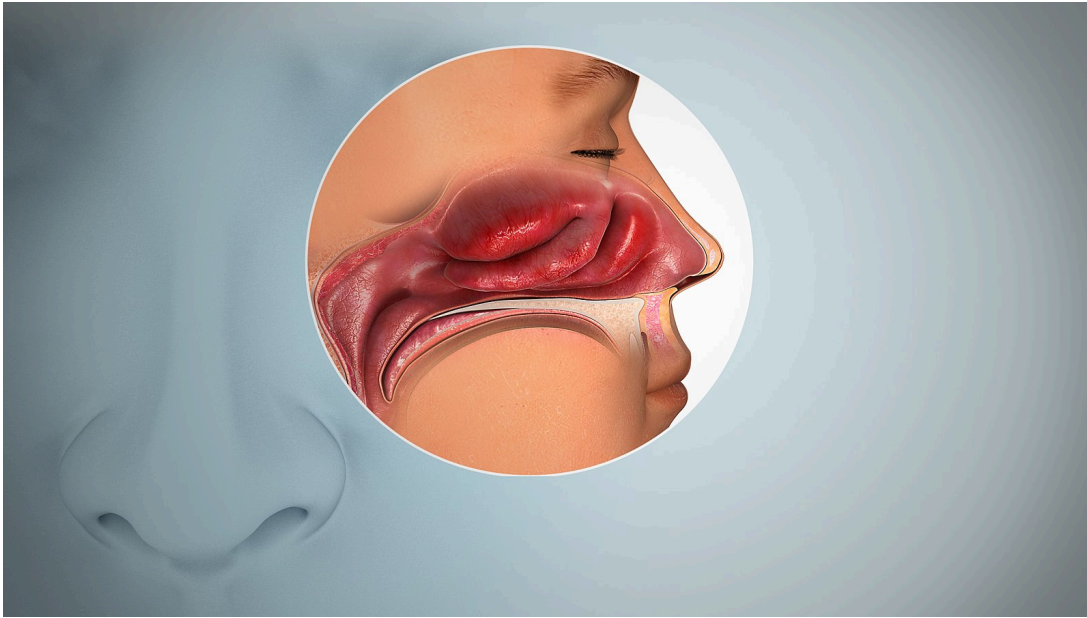


Fig.4.10.1. Anosmia Causes. Inflammation of the nasal membranes blocks air flow causing temporary loss of smell. This is common when you have a cold or allergies. (Credit: Manu5. Provided by: Wikimedia Commons. License: CC-BY SA 4.0)

Anosmia can be a dangerous condition, as smell is used frequently when detecting unsafe situations. For example, individuals suffering from anosmia may be unable to detect the smell of smoke or dangerous gases, which would normally alert them to a dangerous scene. This may also affect the individuals taste, as their odor receptors may no longer activate when eating. Further, the ability of olfactory neurons to replace themselves decreases with age, leading to age-related anosmia. This explains why some elderly people salt their food more than younger people do. However, this increased sodium intake can increase blood volume and blood pressure, increasing the risk of cardiovascular diseases in the elderly.

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Wikipedia, Anosmia

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References:

Melinte, V., & Sarafoleanu, C. (2019). Congenital anosmia: a case report. *Romanian Journal of Rhinology*, 9(35), 161–164. doi: 10.2478/rjr-2019-0020

42.

ACTIVE LEARNING EXERCISE - FLAVOR

This one is super easy to do and kind of fun, although it only generates qualitative data, not quantitative data. So to generate a figure for a lab report, you can just illustrate the kind of food you ate, or your reaction to it with and without your sense of smell.



One or more interactive elements has been excluded from this version of the text. You can view them online here: <https://pressbooks.umn.edu/sensationandperception/?p=2347>

SECTION V

THE MECHANICS OF HEARING

We'll cover the auditory senses in 3 chapters. This first one addresses the physics of sound as well as the anatomy, mechanics, and neural systems in the inner ear. It will include the perceptual and physical characteristics of sound including loudness and amplitude, pitch and frequency, timbre and quality, what are the three parts of ear and their specific functions, and how sound transduction happens in the inner ear.

This chapter was created by Elhan Ahmed, Margaret Kaufman, Cassie Kiehl, Izzy Kosobud, Collin Martin, Nicole McCue, Robert Moss, Mona Osman, Minh Phan, Han Phung.

43.

USES OF SOUND

Learning Objectives

Be able to describe at least 3 uses of sound

Understand what the sound waves are (pressure changes)

Know the relationship between everyday sound waves and atmospheric pressure (unit in Pascal)

The physical phenomenon of sound is a disturbance of matter that is transmitted from its source outward. Hearing is the perception of sound, just as seeing is the perception of visible light. On the atomic scale, sound is a disturbance of atoms that is far more ordered than their thermal motions. In many instances, sound is a periodic wave, and the atoms undergo simple harmonic motion. Thus, sound waves can induce oscillations and resonance effects.

For example, a speaker produces a sound wave by oscillating a cone, causing vibrations of air molecules. It vibrates at a constant frequency and amplitude, producing vibrations in the surrounding air molecules. As the speaker oscillates back and forth, it transfers energy to the air, mostly as thermal energy. But a small part of the speaker's energy goes into compressing and expanding the surrounding air, creating slightly higher and lower local pressures. These compressions (high-pressure regions) and rarefactions (low-pressure regions) move out as longitudinal pressure waves having the same frequency as the speaker—they are the disturbance that is a sound wave. (Sound waves in air and most fluids are longitudinal, because fluids have almost no shear strength. In solids, sound waves can be both transverse and longitudinal.)

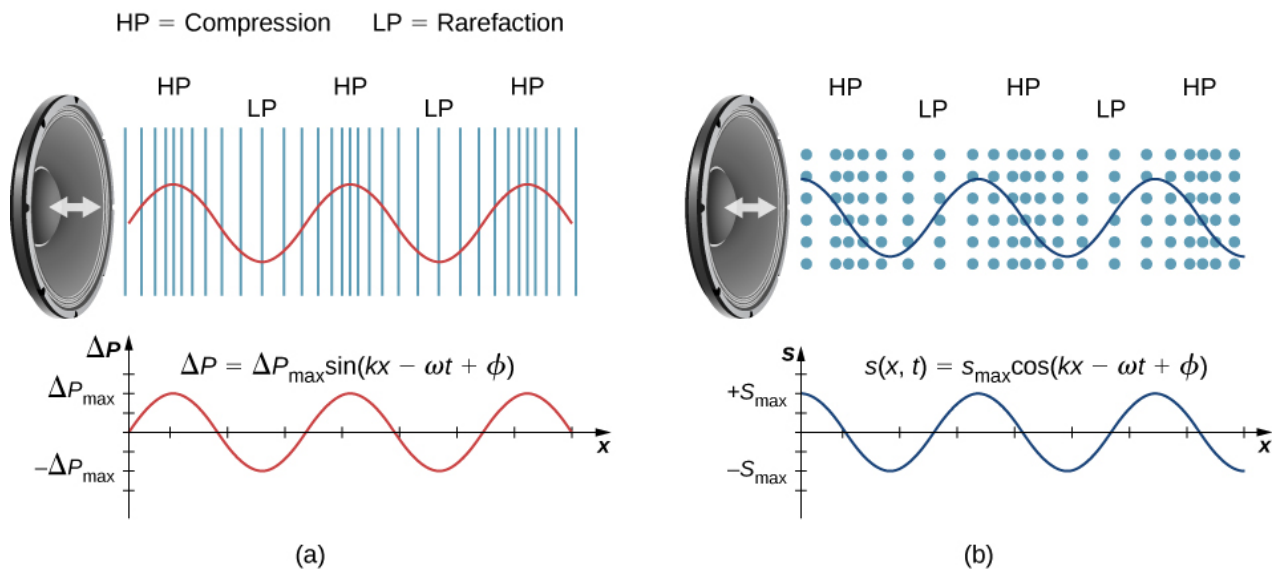


Figure 5.1.1. Sound pressure waves. (a) A vibrating cone of a speaker, moving in the positive x -direction, compresses the air in front of it and expands the air behind it. As the speaker oscillates, it creates another compression and rarefaction as those on the right move away from the speaker. After many vibrations, a series of compressions (high pressure, HP) and rarefactions (low pressure, LP) moves out from the speaker as a sound wave. The red graph shows the gauge pressure of the air versus the distance from the speaker. Pressures vary only slightly from atmospheric pressure for ordinary sounds. Note that gauge pressure is modeled with a sine function, where the crests of the function line up with the compressions and the troughs line up with the rarefactions. (b) Sound waves can also be modeled using the displacement of the air molecules. The blue graph shows the displacement of the air molecules versus the position from the speaker and is modeled with a cosine function. Notice that the displacement is zero for the molecules in their equilibrium position and are centered at the compressions and rarefactions. Compressions are formed when molecules on either side of the equilibrium molecules are displaced toward the equilibrium position. Rarefactions are formed when the molecules are displaced away from the equilibrium position. (Credit: Openstax. License: CC BY 4.0)

Sound waves are pressure changes, usually in air. Compression and rarefaction describe the regions of high and low pressure, respectively, that form when something vibrates and starts a sound wave. The pressure changes propagate (travel) at a rate of 340 m/s (1100 ft/s) in air; 1500 m/s in water.

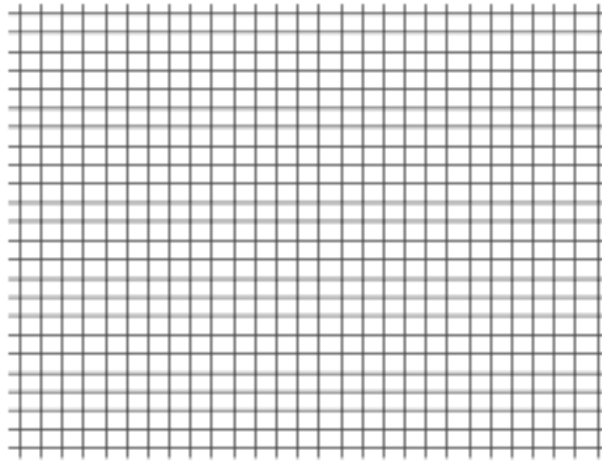


Fig.5.1.2. Sound Waves. Sound travels through the air and other matter as longitudinal waves (also called compression or pressure waves). These waves have points of compression and rarefaction which push the waveforms forward. (Credit: Christophe Dang Ngoc Chan. Provided by: Wikimedia Commons. License: CC-BY SA 3.0)

The Pascal is the standard unit for pressure (force per area); for reference atmospheric pressure (at sea level) is 101 kPa. An express subway train generates pressures of ~ 2 Pa, and conversational speech generates sound waves with intensities of approximately 20 millipascals (mPa). So the sounds we hear are tiny modulations of the air pressure.

Now how do we interpret these pressure waves? Our auditory system converts pressure waves into meaningful sounds. This translates into our ability to locate sounds in nature, to appreciate the beauty of music, and to communicate with one another through spoken language.

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LOUDNESS AND LEVEL

Learning Objectives

Be able to describe what physical characteristic of sound predicts perceived loudness

Know the unit of loudness (decibel) and sound pressure level

Be able to explain the relationship between perception loudness and stimulus intensity

The most direct physical correlate of loudness is sound intensity (or sound pressure) measured close to the eardrum. However, many other factors also influence the loudness of a sound, including its frequency content, its duration, and the context in which it is presented. Some of the earliest psychophysical studies of auditory perception, going back more than a century, were aimed at examining the relationships between perceived loudness, the physical sound intensity, and the just-noticeable differences in loudness (Fechner, 1860; Stevens, 1957).

The loudness of a given sound is closely associated with the amplitude of the sound wave. Higher amplitudes are associated with louder sounds. Loudness is measured in terms of decibels (dB), a logarithmic unit of sound intensity. A typical conversation would correlate with 60 dB; a rock concert might check in at 120 dB. A whisper 5 feet away or rustling leaves are at the low end of our hearing range; sounds like a window air conditioner, a normal conversation, and even heavy traffic or a vacuum cleaner are within a tolerable range. However, there is the potential for hearing damage from about 80 dB to 130 dB: These are sounds of a food processor, power lawnmower, heavy truck (25 feet away), subway train (20 feet away), live rock music, and a jackhammer. The threshold for pain is about 130 dB, a jet plane taking off or a revolver firing at close range (Dunkle, 1982).

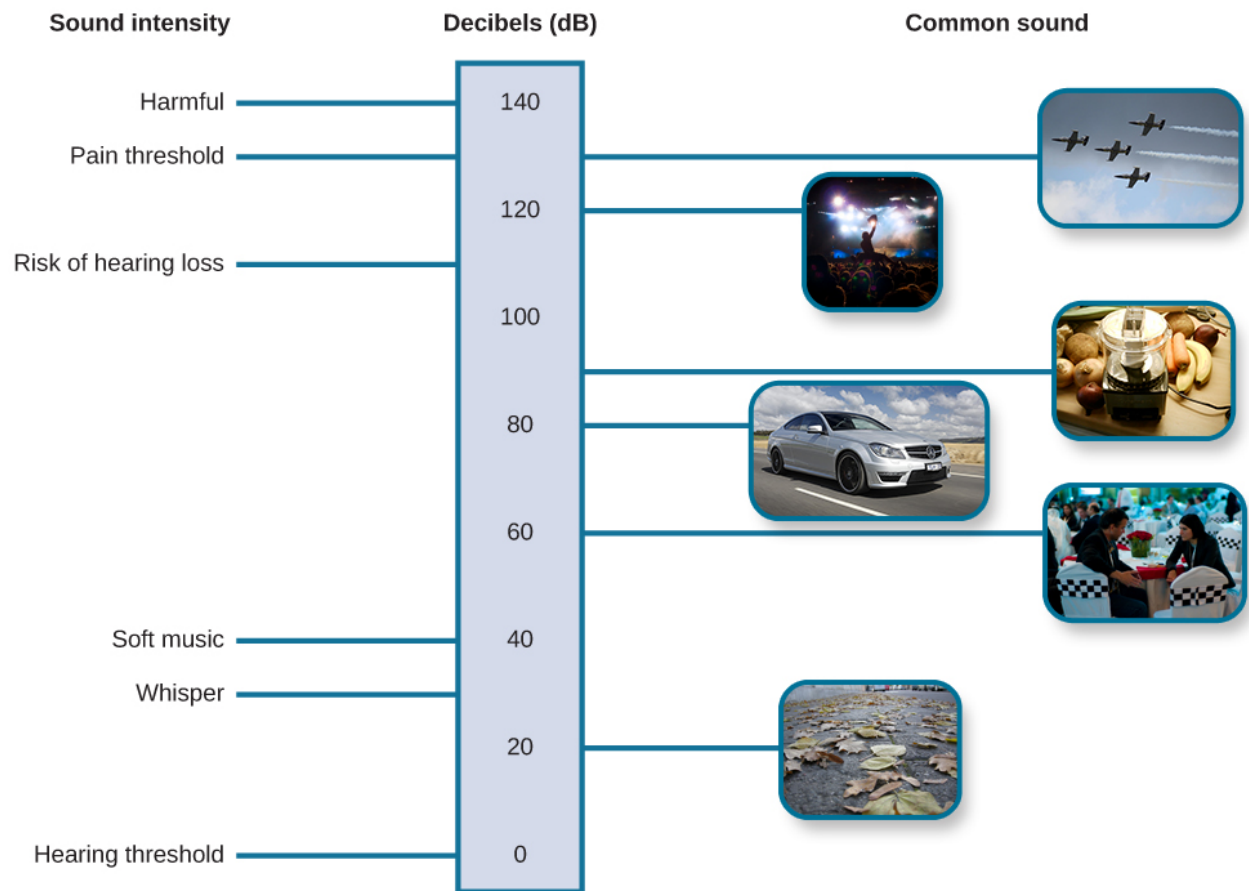


Fig.5.2.1. Loudness Scales. This figure illustrates the loudness of common sounds. (credit “planes”: modification of work by Max Pfandl; credit “crowd”: modification of work by Christian Holmér; credit “blender”: modification of work by Jo Brodie; credit “car”: modification of work by NRMA New Cars/ Flickr; credit “talking”: modification of work by Joi Ito; credit “leaves”: modification of work by Aurelijus Valeiša. Provided by: Openstax. License: CC-BY SA 4.0)

The decibel (dB) quantifies sound pressure level (SPL). A decibel is a relative measure — a loudness relative to another loudness. To calculate Sound Pressure Level, we use a reference sound intensity of 20 micropascals. It is very useful to remember that an increase in SPL of 20dB represents a 10-fold increase in the amplitude of the pressure wave.

$$\text{SPL} = 20\log(P/P_0) = 10\log(P^2/P_0^2) = 10\log(I/I_0)$$

Decibels are calculated by the following equation: $\text{SPL} = 20\log(P/P_0)$. Intensity is the square of the pressure. Intensity is power. So another way of writing the equation above is $\text{SPL} = 10\log(P^2/P_0^2) = 10\log(I/I_0)$. Note: A decibel is 10 bels; a bel is $\log(I/I_0)$. A 20Pa (120dB) sound is about 1 Watt of energy.

Perception of loudness vs. stimulus intensity is a compressive function. The use of a logarithmic function

(decibels) to characterize sound pressure level makes it a little hard to see. If you plot perceived loudness against dB, you get a function that is essentially linear, maybe even scooping up a bit. However, when you plot perceived loudness against the amplitude of the pressure wave, or even against intensity (pressure squared), you see that our perception of loudness is compressive: the louder a sound is, the bigger the change needs to be before we notice it. The Active Learning Exercise at the end of this chapter lets you do a magnitude estimation experiment to discover your own psychometric function for describing your perception of loudness as a function of sound pressure level (although the speakers on our devices often have some non-linearities, so we'd have to use carefully calibrated equipment to get an exact answer).

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45.

PITCH IS FREQUENCY

Learning Objectives

Know the physical characteristic of sound waves that pitch is related to and its unit

Be able to describe the relationship between pitch, frequency, wavelength

Visual and auditory stimuli both occur in the form of waves. Although the two stimuli are very different in terms of composition, waveforms share similar characteristics that are especially important to our visual and auditory perceptions. In this section, we focus on the physical properties of the auditory waves. Two physical characteristics of a wave are amplitude and wavelength. The amplitude of a wave is the height of a wave as measured from the highest point on the wave (peak or crest) to the lowest point on the wave (trough). Wavelength refers to the length of a wave from one peak to the next (Fig.5.3.1). Wavelength is directly related to the frequency of a given waveform. Frequency refers to the number of waves that pass a given point in a given time period and is often expressed in terms of hertz (Hz), or cycles per second. Longer wavelengths will have lower frequencies, and shorter wavelengths will have higher frequencies.

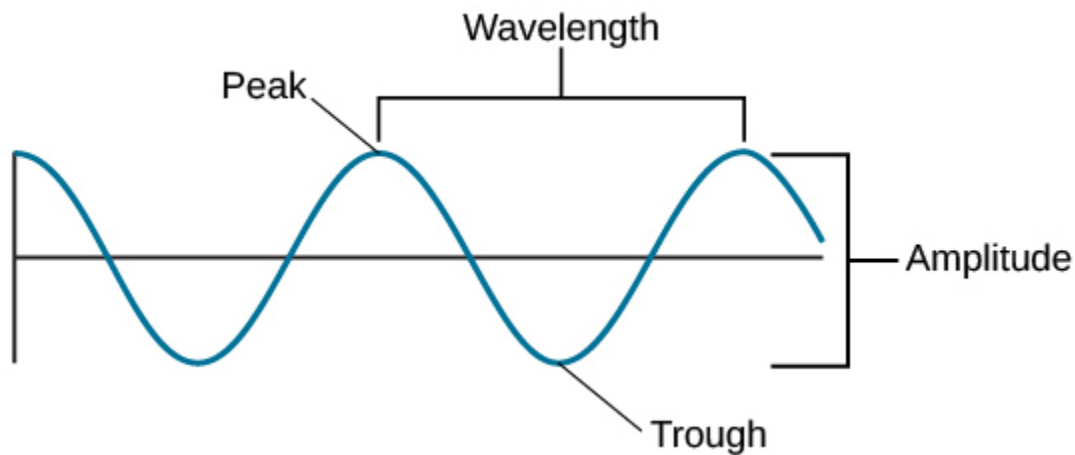


Fig.5.3.1. Sound Wave. Diagram of the critical points to measure a wavelength. (Provided by: OpenStax. License: CC-BY 4.0)

The frequency of a sound wave is associated with our perception of that sound's pitch. High-frequency sound waves are perceived as high-pitched sounds, while low-frequency sound waves are perceived as low-pitched sounds. The audible range of sound frequencies is between 20 and 20000 Hz, with greatest sensitivity to those frequencies that fall in the middle of this range. Different organisms have different auditory sensitivity. For example, chickens have a very limited audible range, from 125 to 2000 Hz. Mice have an audible range from 1000 to 91000 Hz, and the beluga whale's audible range is from 1000 to 123000 Hz. Our pet dogs and cats have audible ranges of about 70–45000 Hz and 45–64000 Hz, respectively (Strain, 2003).

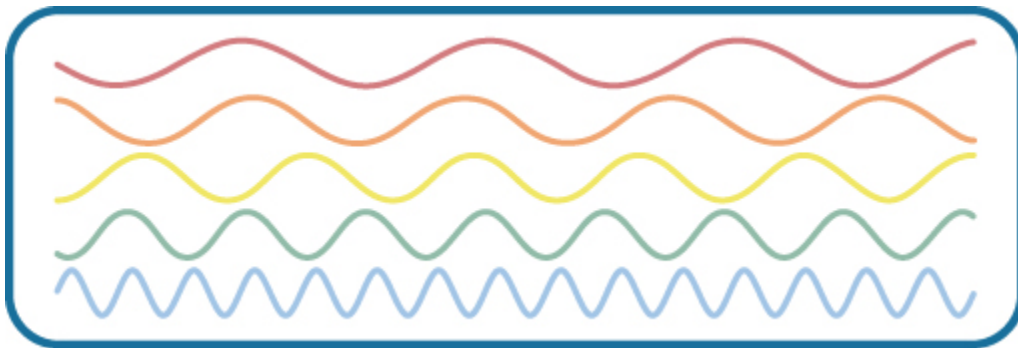


Figure 5.3.2. Differing Wavelengths/Frequencies. This figure illustrates waves of differing wavelengths/frequencies. At the top of the figure, the red wave has a long wavelength/short frequency. Moving from top to bottom, the wavelengths decrease and frequencies increase. (Provided by: OpenStax. License: CC-BY 4.0)

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Adapted by: Cassie Kiehl

INTERFERENCE AND COMPLEX TONES

Learning Objectives

Be able to explain the difference between constructive and destructive interference

Be able to define the fundamental tones and harmonics in complex tones

Constructive interference is when two waves superimpose (place an object over another object) and the resulting wave has a higher amplitude than the previous waves. Destructive interference is when two waves superimpose and cancel each other out (like adding a positive and negative number), leading to a lower amplitude. While pure constructive and pure destructive interference do occur, they require precisely aligned identical waves. The superposition of most waves produces a combination of constructive and destructive interference, and can vary from place to place and time to time. Sound from a stereo, for example, can be loud in one spot but quiet in another. Varying loudness means the sound waves add partially constructively and partially destructively at different locations. A stereo has at least two speakers creating sound waves, and waves can reflect from walls. All these waves superimpose. An example of sounds that vary over time from constructive to destructive is found in the combined whine of airplane jets heard by a stationary passenger. The combined sound can fluctuate up and down in volume as the sound from the two engines varies in time from constructive to destructive.

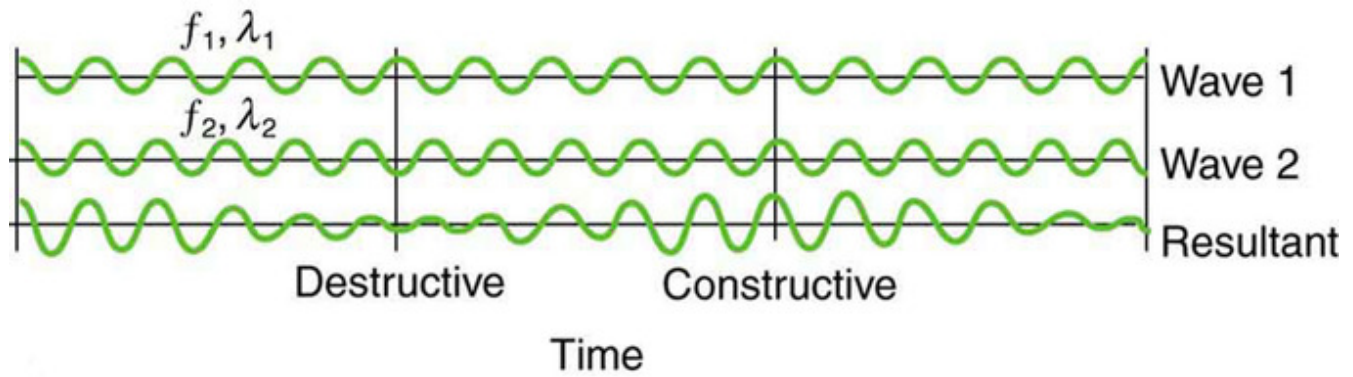


Fig.5.4.1. Wave Interference. The bottom wave is the sum of the two wavelengths above. Each of the top two waves has a slightly different frequency, so sometimes their peaks line up and sometimes they don't. Notice how two peaks meet in the constructive case, while a peak and trough meet in the destructive case. (Provided by: Lumens. License: CC-BY SA 4.0)

Pitch is essentially the perceptual correlate of waveform periodicity, or repetition rate: The faster a waveform repeats over time, the higher is its perceived pitch. The most common pitch-evoking sounds are known as harmonic complex tones. They are complex because they consist of more than one frequency, and they are harmonic because the frequencies are all integer multiples of a common fundamental frequency (F_0). For instance, a harmonic complex tone with a F_0 of 100 Hz would also contain energy at frequencies of 200, 300, 400 Hz, and so on. These higher frequencies are known as harmonics or overtones, and they also play an important role in determining the pitch of a sound.

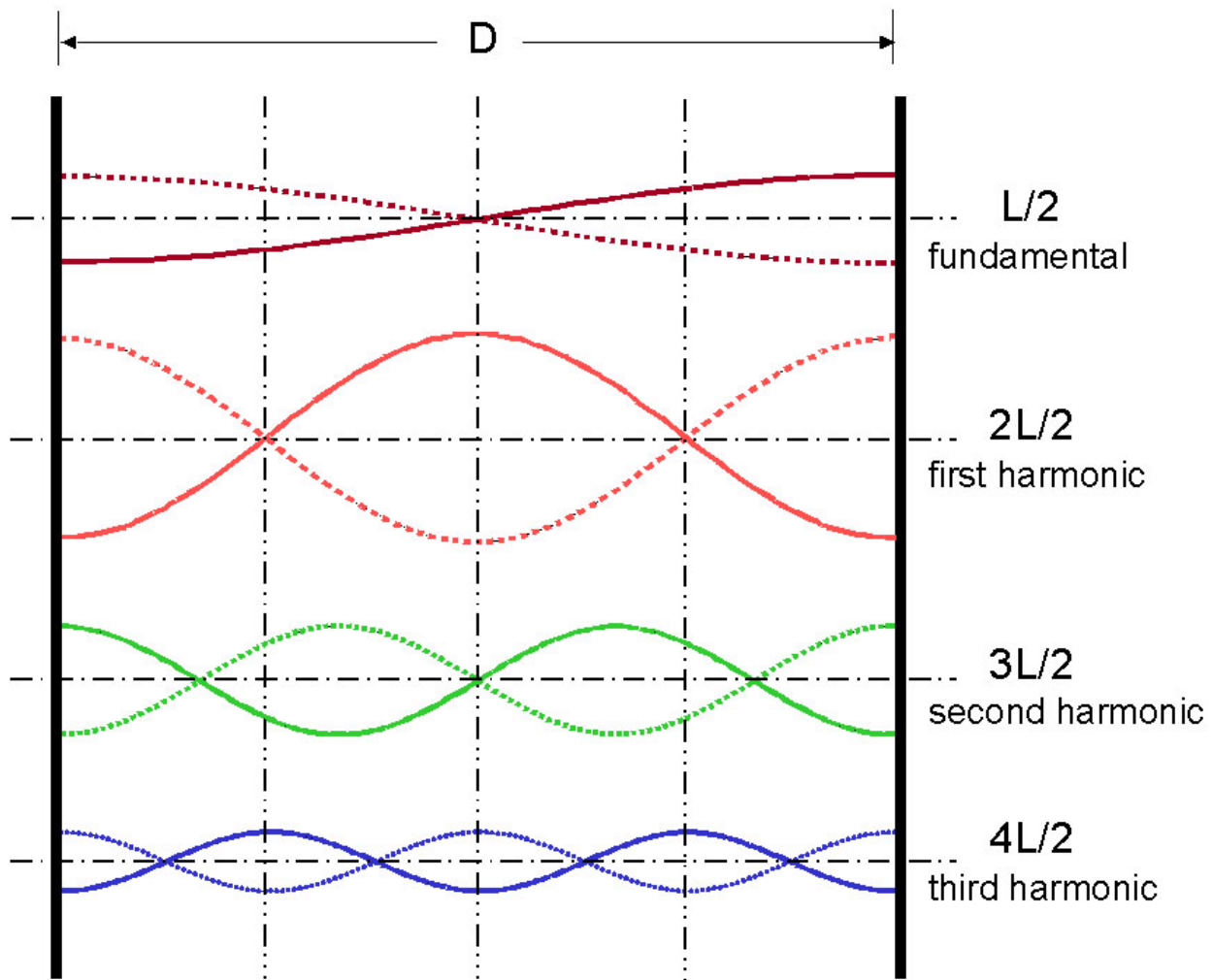


Figure 5.4.2. Fundamental frequency and first 3 harmonic overtones. The fundamental frequency, shown in red, is the lowest frequency of sound. The frequencies of harmonic overtones, shown in orange, green, and blue, are higher integer multiples of the fundamental frequency. Together, these frequencies are called harmonic complex tones. (Credit: Buesching, Fritz, Harmonics, CC-BY-SA-2.0, <https://commons.wikimedia.org/wiki/File:Harmonics.jpg>)

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Lumen Learning, Boundless Physics, Interactions with Sound Waves

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NOBA, Hearing

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47.

AUDITORY SENSITIVITY FUNCTION

Learning Objectives

Be able to describe the frequency range of human hearing

Know the range of frequencies at which human hearing is typically most sensitive

Be able to explain the typical fundamental frequency of speech

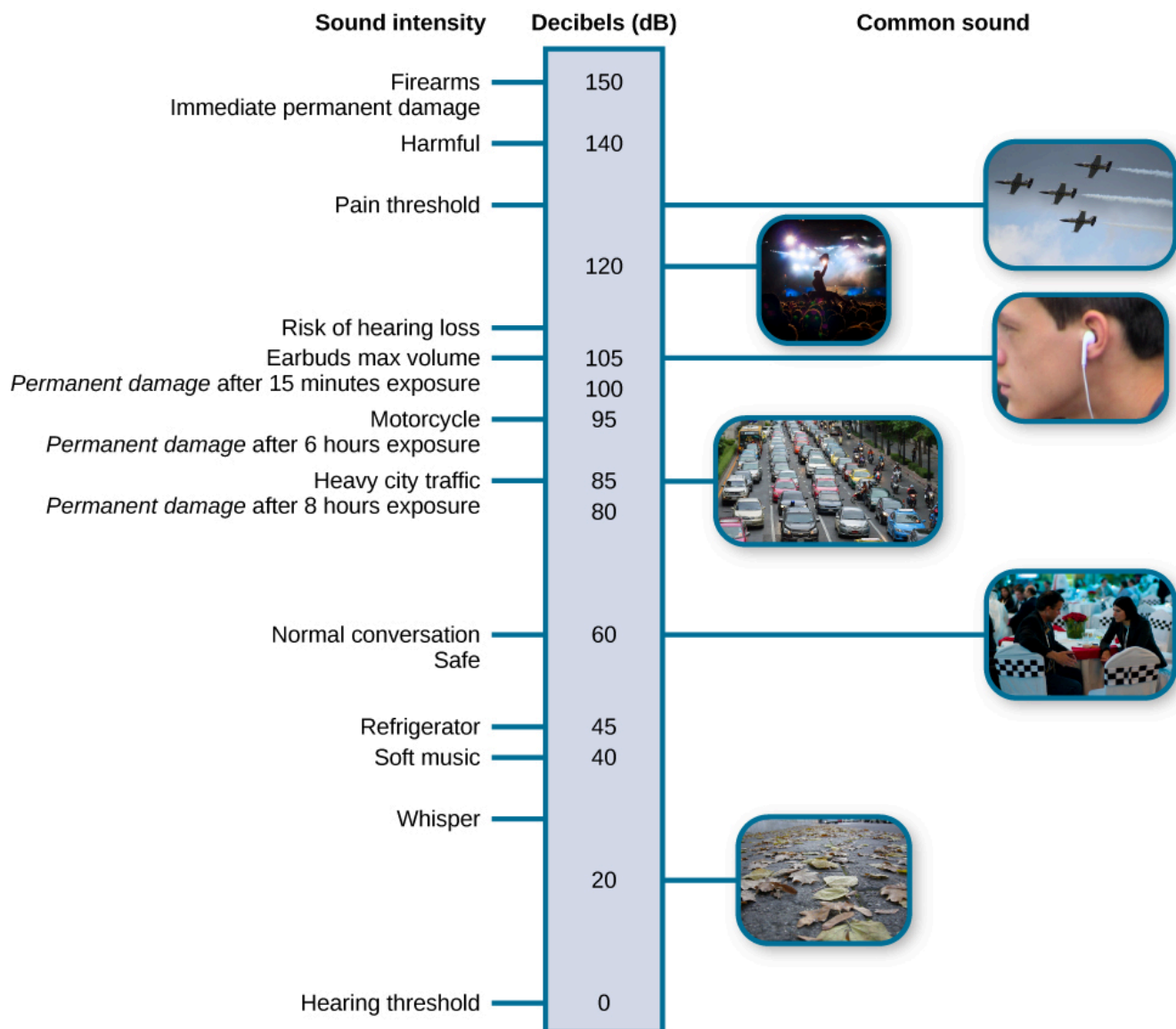


Fig 5.5.3. This figure illustrates the loudness of common sounds. (Provided by: OpenStax. License CC-BY SA)

Like light waves, the physical properties of sound waves are associated with various aspects of our perception of sound. The frequency of a sound wave is associated with our perception of that sound's pitch. High-frequency sound waves are perceived as high-pitched sounds, while low-frequency sound waves are perceived as low-pitched sounds. The audible range of sound frequencies is between 20 and 20000 Hz, with greatest sensitivity to those frequencies that fall in the middle of this range.

The unit of the loudness of sound is the decibel (dB). For example, an ordinary conversation is approximately equal to 60 decibels (dB), and a rock concert is approximately equal to 120 decibels (dB). These are all examples of different levels of sound loudness in daily life. The lowest of the human hearing range is approximately equal to a whisper at a distance of 5 feet. This level of sound loudness is almost the smallest sound in the acceptable range.

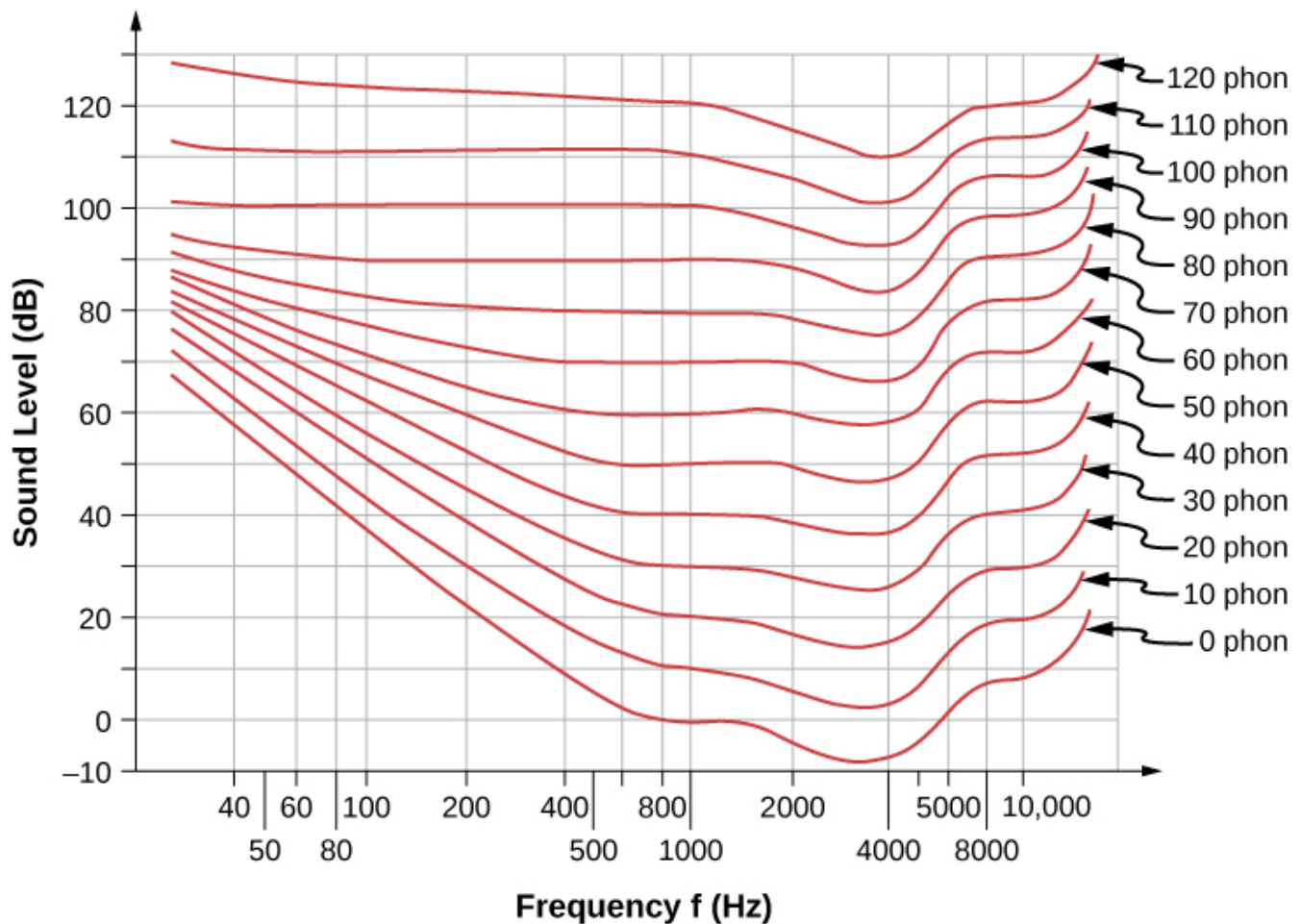


Fig.5.5.1. The relationship of loudness in phons to intensity level (in decibels) and intensity (in watts per meter squared) for persons with normal hearing. The curved lines are equal-loudness curves—all sounds on a given curve are perceived as equally loud. Phons and decibels are defined to be the same at 1000 Hz. (Provided by: OpenStax. License CC-BY SA)

We are most sensitive to sounds in the range of 1,000 – 3,000 Hz, where important information in conversational speech tends to be. Musical notes occupy a smaller range, from about 28 Hz to just over 4,000 Hz. The voiced speech of a typical adult male will have a fundamental frequency from 85 to 180 Hz, and that of a typical adult female from 165 to 255 Hz. Thus, the fundamental frequency of most speech falls below the bottom of the voice frequency band.

Dogs can hear above 40,000 Hz; dolphins can hear up to 150,000 Hz. Additionally, chickens have a very limited audible range, from 125 to 2000 Hz. Mice have an audible range from 1000 to 91000 Hz, and the beluga whale's audible range is from 1000 to 123000 Hz. Our pet dogs and cats have audible ranges of about 70–45000 Hz and 45–64000 Hz, respectively (Strain, 2004).

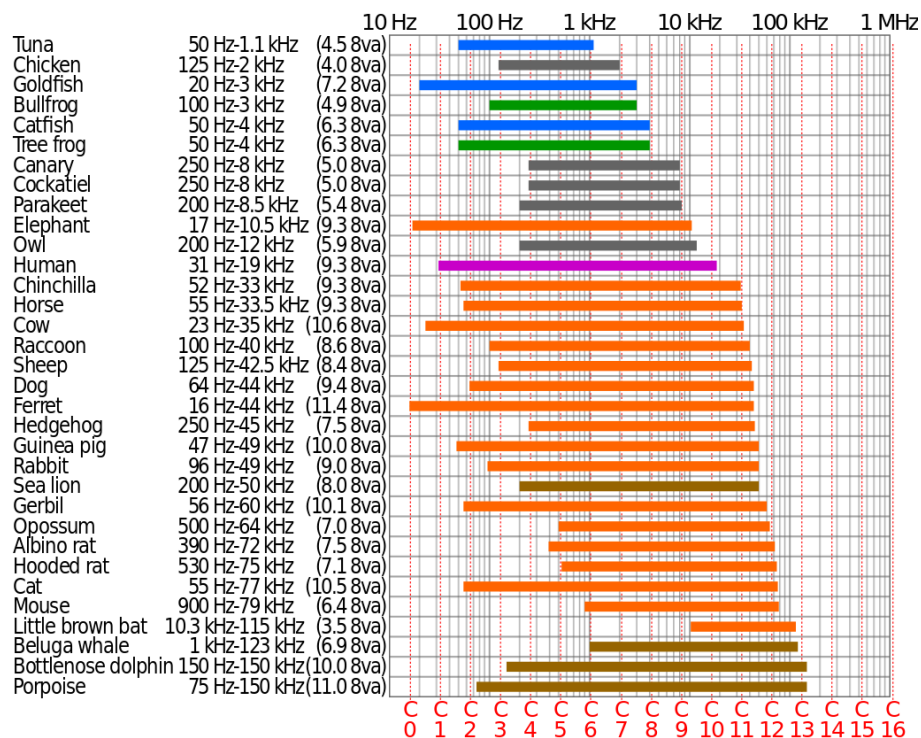


Fig.5.5.2. Animal and humans hearing frequency range. (Credit: Cmglee. Provided by: Wikimedia Commons. License: CC-BY SA 3.0)

Strain GM. Deafness prevalence and pigmentation and gender associations in dog breeds at risk. Vet J. 2004 Jan;167(1):23-32. doi: 10.1016/s1090-0233(03)00104-7. PMID: 14623147.

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OpenStax, Psychology Chapter 5.2 Waves and Wavelengths

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URL:https://en.wikipedia.org/wiki/Voice_frequency#:~:text=The%20voiced%20speech%20of%20a,frequency%20band%20as%20defined%20above.

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TIMBRE

Learning Objectives

Know the difference between simple sounds and normal real-world sounds (complex sounds)

Be able to describe what timbre characterizes

There are two categories of sound covered in this section: simple and complex. Simple sounds are also called pure or simple tones because they consist of a single frequency, but the intensity of that frequency can vary. An example of a simple tone is a tuning fork because the sound it produces is almost entirely only one tone for musical instruments to match. Simple tones do not characterize normal real-world sounds because real-world sounds are more complicated. In everyday life, we hear sounds that consist of the second type of sound: complex sounds (or complex tones). A complex tone comprises two or more simple tones. Complex tones make up the vast majority of sounds we hear day-to-day. Some examples of complex sounds include voices and music.

The tone that has the lowest frequency in the complex tone is called the fundamental frequency, and the other tones are known as overtones or harmonics. The fundamental frequency is what we perceive as being the pitch of a sound. Because the fundamental frequency is the perceived pitch we hear in a complex tone, we say the complex tone is characterized by its fundamental frequency. For example, if a complex tone had three overtones of varying frequencies that were a multiple of 100 Hz, and a fundamental frequency of 100 Hz, the perceived pitch of this sound would be 100 Hz.

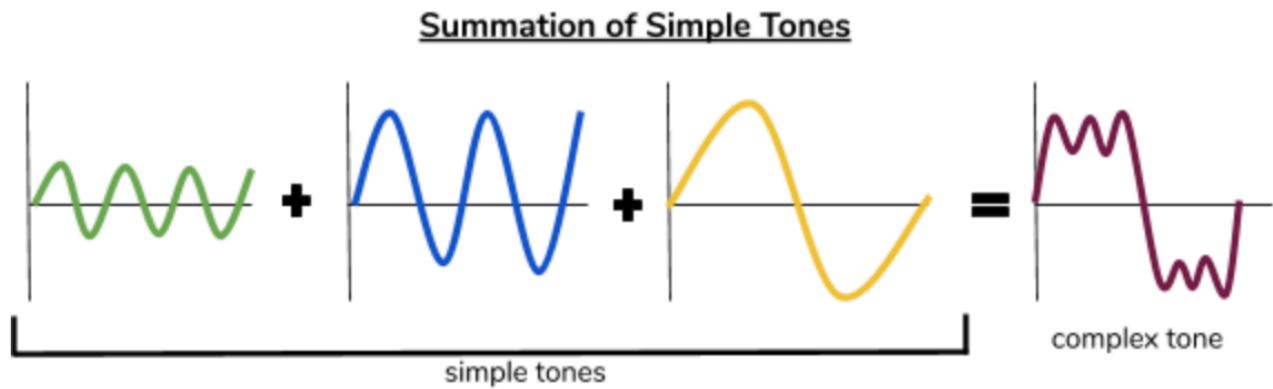


Fig.5.6.1. Synthesis of multiple pure tones of varying frequencies to create a complex tone when summed together. Two or more simple tones combined is enough to create a complex tone wherein the lowest frequency represents the fundamental frequency. (Credit: Nicole McCue. Provided by: University of Minnesota. License: CC-BY 4.0)

Timbre is the quality of complex tones produced. The timbre of a sound depends on its waveform, which varies with the number, frequency, and relative intensity of the overtones that are present. Different waveforms are produced by synthesizing (combining) different pure tones of various frequencies and intensities. For this reason, the timbre of a sound varies with the characteristics of the overtones. Two different musical instruments can be playing the same note but have different timbres because they have different complex tones despite having the same fundamental frequency.

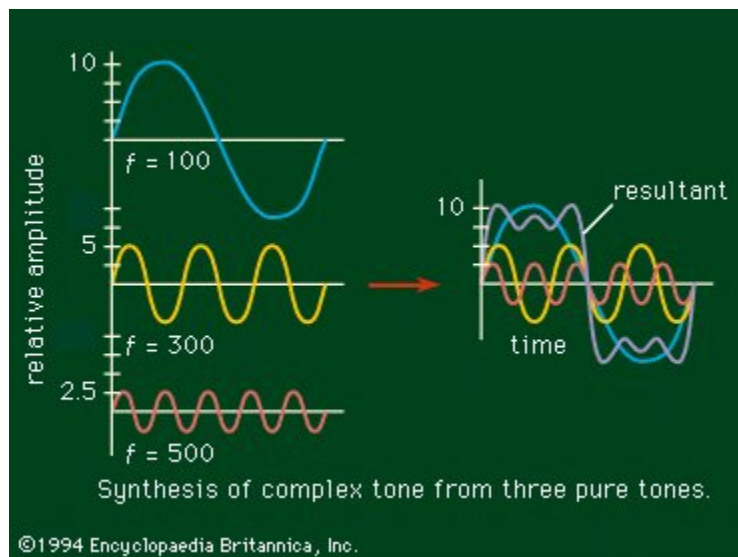


Fig 5.6.2 The illustration shows the waveform that results when pure tones of frequencies 100, 300, and 500 hertz (cycles per second) and relative amplitudes of 10, 5, and 2.5 are synthesized into a complex tone. At the right is the resultant which would be considered a timbre. (Credit: Britannica, T. Editors of Encyclopaedia (2018, February 1). Timbre. Encyclopedia Britannica. <https://www.britannica.com/science/timbre>)

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Encyclopaedia Britannica, Tone

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Encyclopaedia Britannica, Timbre

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49.

THREE DIVISIONS OF THE EAR

Learning Objectives

Be able to describe what the outer ear comprises and its function

Be able to describe mechanics of the middle ear and how to amplify sound waves

Know what impedance and stapedius reflex mean

The ear can be separated into multiple sections. The outer ear includes the pinna, which is the visible part of the ear that protrudes from our heads, the auditory canal, and the tympanic membrane, or eardrum. The middle ear contains three tiny bones known as the ossicles, which are named the malleus (or hammer), incus (or anvil), and the stapes (or stirrup). The inner ear contains the semicircular canals, which are involved in balance and movement (the vestibular sense), and the cochlea. The cochlea is a fluid-filled, snail-shaped structure that contains the sensory receptor cells (hair cells) of the auditory system.

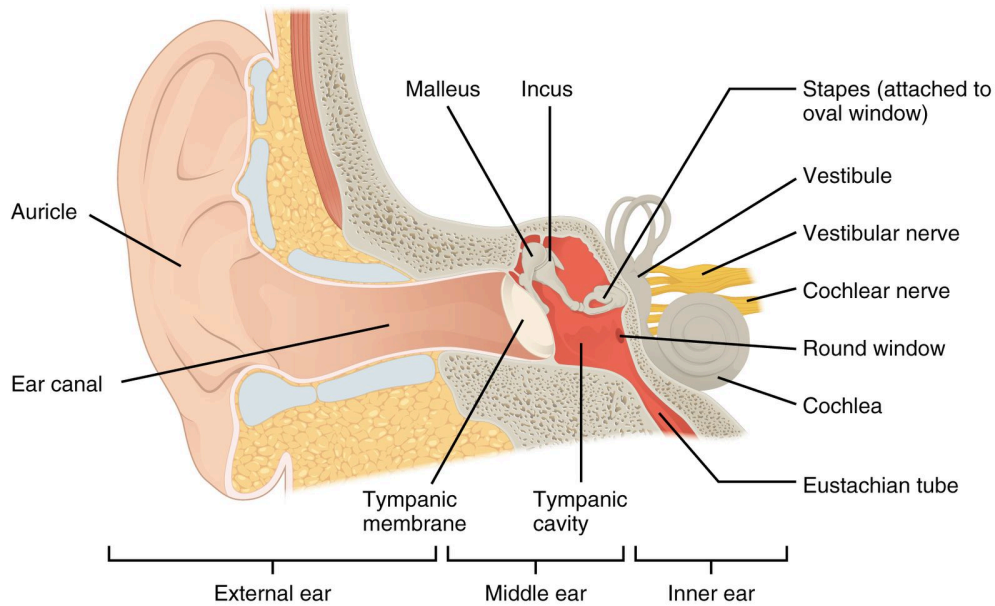


Fig. 5.7.1. The anatomy of the ear can be separated into three sections as seen above. Note that the semi-circular canals are not labeled above. They are the three grey loops above the cochlea. (Credit: OpenStax. Provided by: Wikimedia Commons. License: CC-BY 4.0)

Sound waves travel along the auditory canal and strike the tympanic membrane, causing it to vibrate. This vibration results in movement of the three ossicles. As the ossicles move, the stapes press into a thin membrane of the cochlea known as the oval window. As the stapes presses into the oval window, the fluid inside the cochlea begins to move, which in turn stimulates hair cells, which are auditory receptor cells of the inner ear embedded in the basilar membrane (more on the basilar membrane in the next section).

Stapedius reflex means that the stapedius muscle connected to the ossicles will tighten up to reduce the amplification when we're exposed to loud sounds. But it's not that effective and the muscle reflex is too slow to protect us from abrupt sounds like gunshot. And the muscles adapt after a while. Impedance refers to the ability of different objects to hinder sound. People will leverage the ossicles (smallest bones in the body) and the fact that the tympanic membrane is larger than the oval window amplifies sound waves. The primary reason sound pressure waves need to be amplified is that the energy is being transferred from air (outer ear) to liquid (inner ear/cochlea). Air and water have different impedance, which means air is easier to move with air pressure waves than water.

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THE INNER EAR

Learning Objectives

Be able to explain what the inner ear comprises (cochlea and vestibular organs)

Know the basilar membrane in inner ear and whether high frequencies are at the base or the apex

The inner ear includes the cochlea, encased in the temporal bone of the skull, in which the mechanical vibrations of sound are transduced into neural signals that are processed by the brain. The cochlea is a spiral-shaped structure that is filled with fluid.

One of the most important principles of hearing—frequency analysis—is established in the cochlea. In a way, the action of the cochlea can be likened to that of a prism: the many frequencies that make up a complex sound are broken down into their constituent frequencies, with low frequencies creating maximal basilar-membrane vibrations near the apex of the cochlea and high frequencies creating maximal basilar-membrane vibrations nearer the base of the cochlea. This decomposition of sound into its constituent frequencies, and the frequency-to-place mapping, or “tonotopic” representation, is a major organizational principle of the auditory system, and is maintained in the neural representation of sounds all the way from the cochlea to the primary auditory cortex.

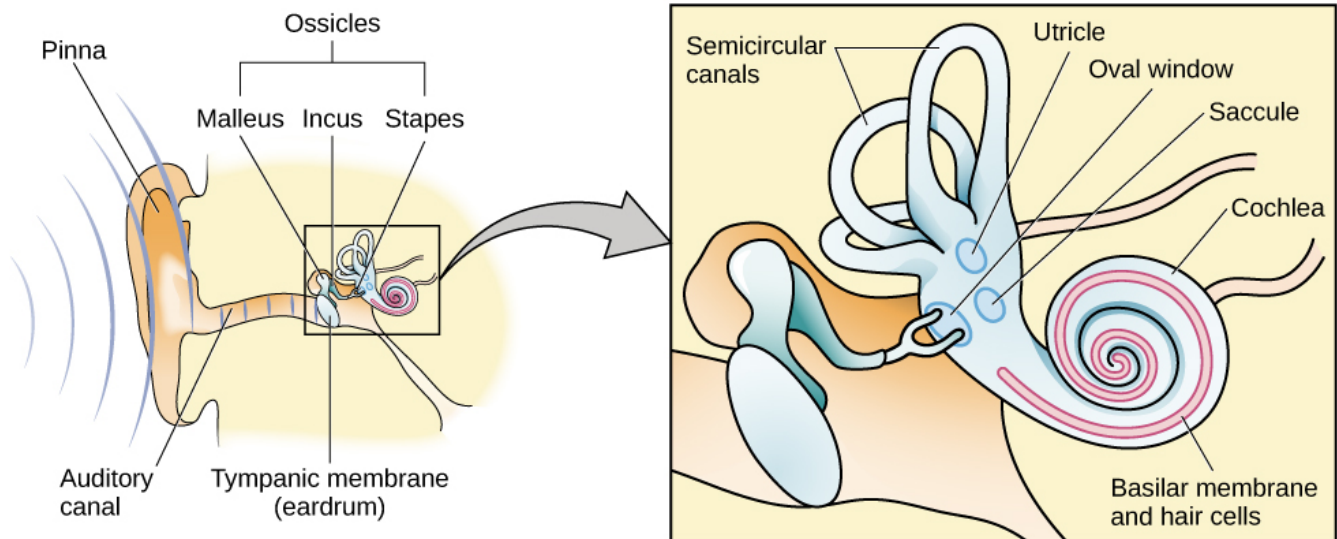


Fig.5.8.1. The ear is divided into outer (the pinna and the tympanic membrane), middle (the three ossicles: malleus, incus, and stapes), and the inner ear (Provided by: OpenStax. License: CC-BY 4.0)

The place theory of pitch perception suggests that different portions of the basilar membrane are sensitive to sounds of different frequencies. More specifically, the base of the basilar membrane responds best to high frequencies and the tip of the basilar membrane responds best to low frequencies. Therefore, hair cells that are in the base portion would be labeled as high-pitch receptors, while those in the tip of basilar membrane would be labeled as low-pitch receptors (Shamma, 2001).

References:

Shamma S. On the role of space and time in auditory processing. *Trends Cogn Sci*. 2001 Aug 1;5(8):340-348. doi: 10.1016/s1364-6613(00)01704-6. PMID: 11477003.

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51.

INNER AND OUTER HAIR CELLS

Learning Objectives

Be able to explain how sound transduction happens from inner hair cells to brainstem

Be able to describe the motile response (electromotility) of the outer hair cells

The inner hair cells are the sensory neurons. We have about 3500 of them, running the whole length of the basilar membrane. Each hair cell connects to about 10 auditory nerve endings! These auditory neurons live in the Organ of Corti, which rides on the basilar membrane and houses one row of inner hair cells and three rows of outer hair cells. The top of the Organ of Corti is the tectorial membrane. When the basilar membrane waves, the tectorial membrane shifts sideways, relative to the basilar membrane, and stimulates the hair cells. This causes the hair cells to shift and release neurotransmitters. The actual motion of the hair cells are quite tiny. If a hair cell were as tall as the Eiffel tower, then the displacement of the cilia would only be 1 cm.

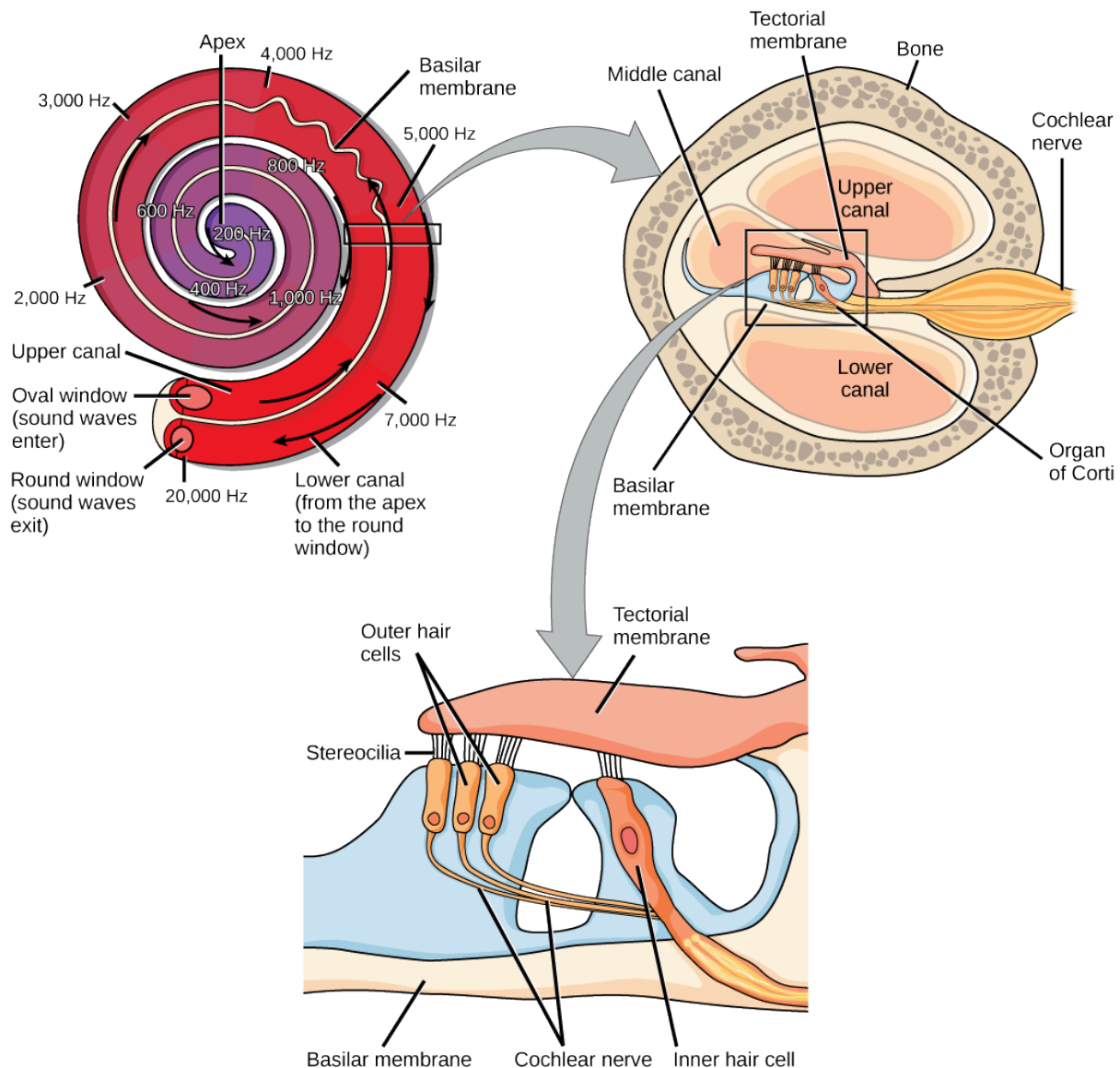


Fig.5.9.1: Structures and Motion of Sound Transduction

In the human ear, sound waves cause the stapes to press against the oval window. Vibrations travel up the fluid-filled interior of the cochlea. The basilar membrane that lines the cochlea gets continuously thinner toward the apex of the cochlea. Different thicknesses of membrane vibrate in response to different frequencies of sound. Sound waves then exit through the round window. In the cross section of the cochlea (top right figure), note that in addition to the upper canal and lower canal, the cochlea also has a middle canal. The organ of Corti (bottom image) is the site of sound transduction. Movement of stereocilia on hair cells results in an action potential that travels along the auditory nerve. (credit: Openstax, <https://openstax.org/books/biology-ap-courses/pages/27-4-hearing-and-vestibular-sensation>, CC-BY-4.0).

The motile response (electromotility) of the outer hair cells amplifies the vibration of the basilar membrane and sharpens frequency tuning. Prestin is the name of the molecule that makes the cell move. This feedback loop actually makes the ear generate sounds as well! “Otoacoustic emissions” can be measured: these are sounds

coming out of your auditory canal. They can be used as hearing screening in newborn babies: play a sound, and measure echo coming back out. If you don't measure them, maybe it's just a plugged up middle ear. Noticing hearing loss early is crucial for assisting language development (most common treatment is a cochlear implant, but sign language and hearing aids are also used). After all, outer hair cells are most susceptible to damage and their loss is often the root of hearing damage — you lose amplification for quiet sounds, and you stop being able to “hear out” sounds ... separate frequencies.

As hair cells become activated, secondary afferent neurons with cell bodies in the spiral ganglion pick up neurotransmitters from inner hair cells and send signals along the auditory nerve to the brain. Auditory information is shuttled to the inferior colliculus (in the brainstem), the medial geniculate nucleus of the thalamus, and finally to the auditory cortex in the temporal lobe of the brain for processing. Like the visual system, there is also evidence suggesting that information about auditory recognition and localization is processed in parallel streams (Rauschecker & Tian, 2000; Renier et al., 2009).

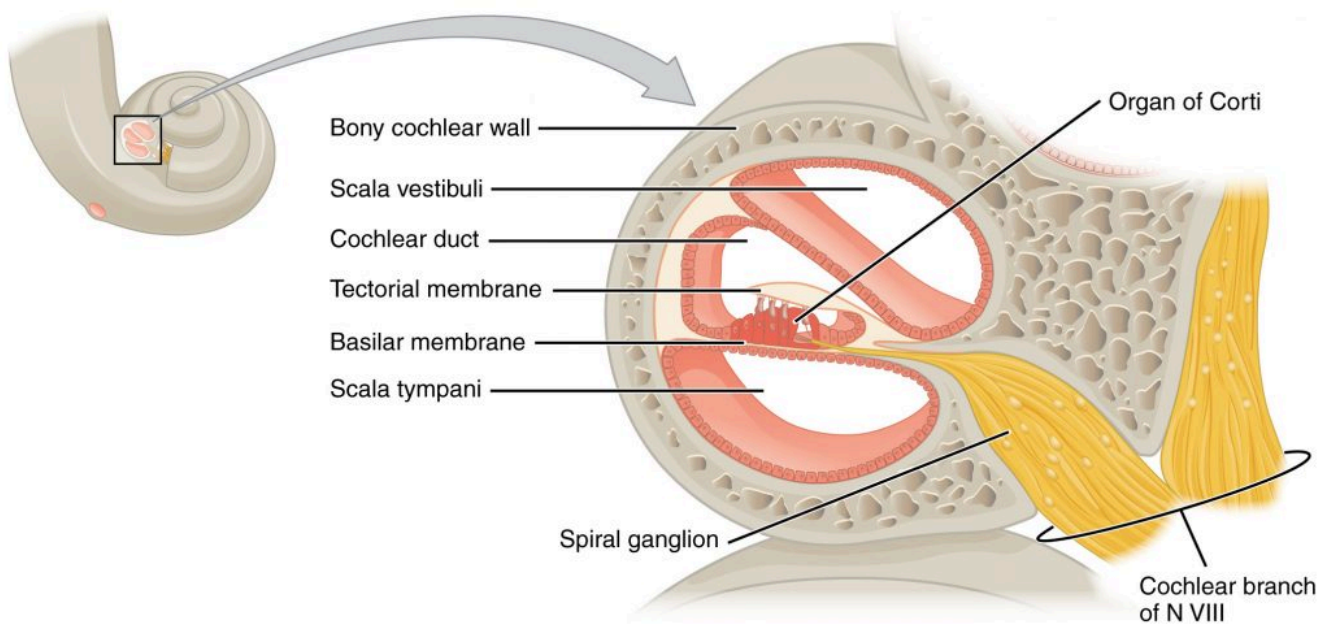


Fig.5.9.2. Cross Section of the Cochlea. The three major spaces within the cochlea are highlighted. The scala tympani and scala vestibuli lie on either side of the cochlear duct. The organ of Corti, containing the mechanoreceptor hair cells, is adjacent to the scala tympani, where it sits atop the basilar membrane. (Provided by: OpenStax. License: CC-BY 4.0)

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PLACE CODING AND TIME CODING

Learning Objectives

Be able to explain how to encode sound frequency (combination of place and time coding)

Know what role time coding plays when encoding sound

Be able to describe why place coding is necessary

Different frequencies of sound waves are associated with differences in our perception of the pitch of those sounds. Low-frequency sounds are lower-pitched, and high-frequency sounds are higher pitched. Several theories have been proposed to account for how the auditory system differentiates various pitches.

First, time coding of pitch perception asserts that frequency is coded by the activity level of a sensory neuron. This would mean that a given hair cell would fire action potentials related to the frequency of the sound wave. Different neurons respond to different cycles of sound; when action potentials fire, they fire at the same place in the cycle. Added together, the population of the different neurons as a whole represents the entire waveform. While this is a very intuitive explanation, we detect such a broad range of frequencies (20–20,000 Hz) that the frequency of action potentials fired by hair cells cannot account for the entire range. There is a point at which a cell cannot fire any faster (Shamma, 2001).

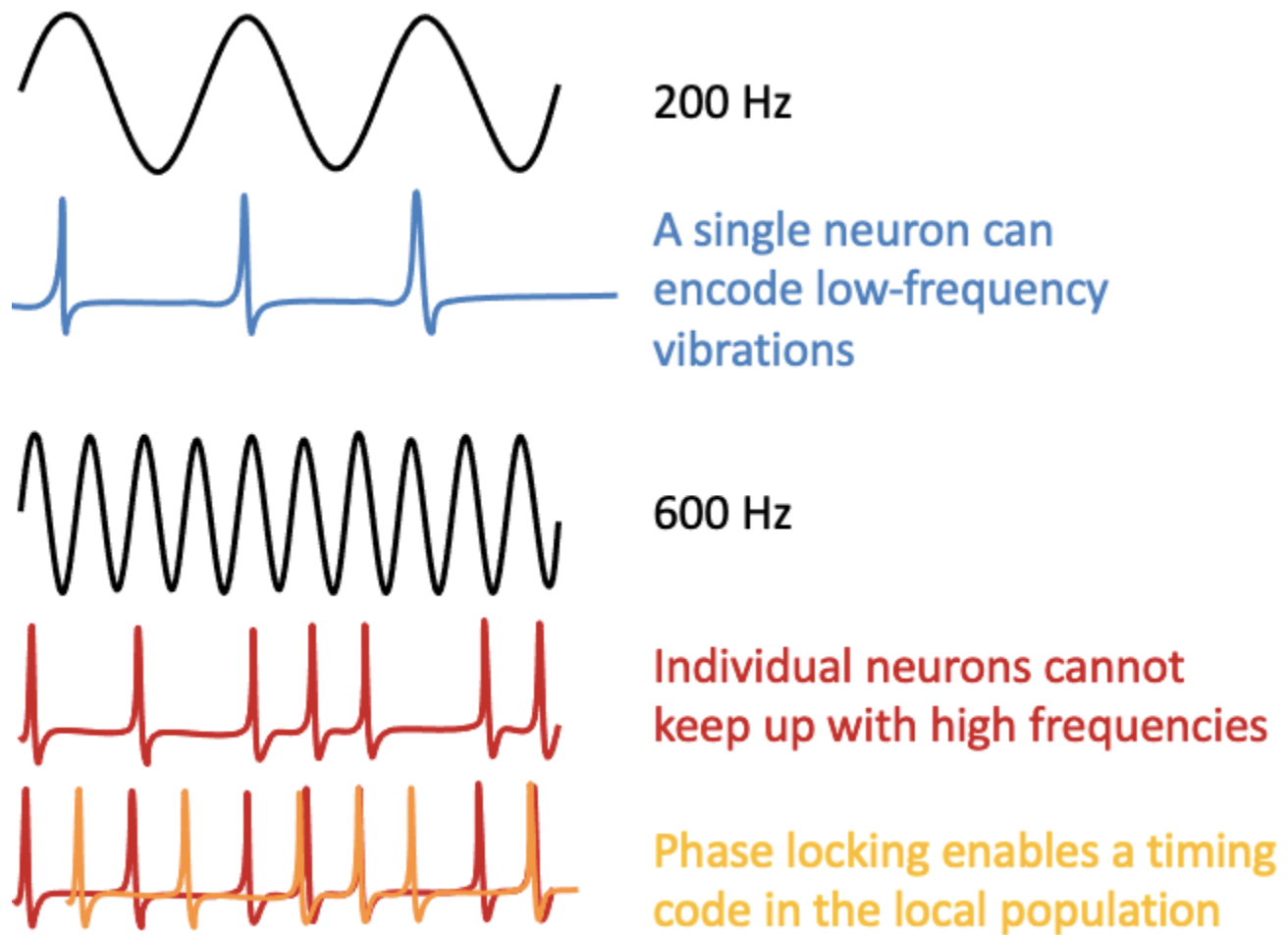


Figure 5.10.1. The timing code is created by phase locking. (Credit: Cheryl Olman. Provided by: University of Minnesota. License: CC BY 4.0)

Place coding comes from the fact that different portions of the basilar membrane are more sensitive to sounds of different frequencies. Hair cells that are in the base portion of the basilar membrane would be labeled as high-pitch receptors, while those in the tip of basilar membrane would be labeled as low-pitch receptors (Shamma, 2001).

In reality, both place coding and the timing code contribute to pitch perception. At frequencies up to about 4000 Hz, it is clear that both the rate of action potentials and place contribute to our perception of pitch. However, higher frequency sounds are encoded using place cues (Shamma, 2001).

Place Coding

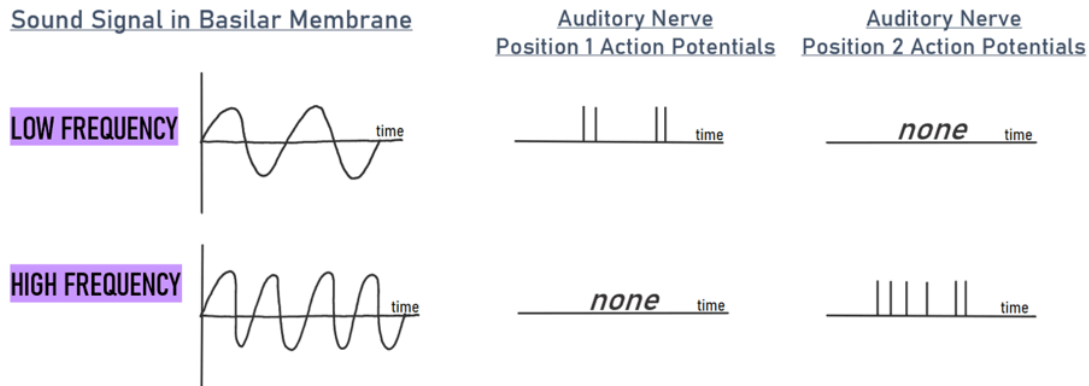


Fig.5.10.2. For low frequencies received in the basilar membrane, a small amount of action potentials are fired at a particular region designated for low frequencies, and vice versa for high frequencies. Because there are designated positions for low and high frequencies, the nerve position will not fire any action potentials for the contrasting frequency signal (Credit: Han Phung. Provided by: University of Minnesota. License: CC BY 4.0).

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ACTIVE LEARNING EXERCISE: LOUDNESS



One or more interactive elements has been excluded from this version of the text. You can view them online here: <https://pressbooks.umn.edu/sensationandperception/?p=2381>

SECTION VI

HEARING LOSS AND CENTRAL AUDITORY PROCESSING

This chapter will cover two very different topics. The first topic is hearing loss — for people who are born with a sense of hearing, environmental factors lead to hearing damage for everyone, and some people experience injury or illness that decreases or eliminates their sense of hearing. For people who are born without hearing, cochlear implants make hearing possible, but whether or not to join the Hearing community is often a hard choice for an individual.

The second topic is central auditory processing — the neural pathways, neural circuits, and anatomical locations that process sounds once the auditory nerve relays auditory signals from the inner ear.

This chapter was created by Sherwina Adams, Andrew Bartlett, Aliciana Bezdicek, Olivia Chareunrath, Zhijian Chen, Jacia Christiansen, Grace Cumming, Ananda Davenport, Josef Dewitt, Nicole Engelhart, Maida Fazlic, Wendy Geronimo, and Kelvin Goebel.

CONDUCTIVE HEARING LOSS

Learning Objectives

Be able to describe the possible causes of conductive hearing loss

Understand the impact of it in daily life

Know what the mastoid bone is and how can it be used to distinguish conductive hearing loss from sensorineural hearing loss

Conductive hearing loss is less common but more likely to be fixable. It is caused by damage to mechanical structures of the outer ear (e.g., extraordinary wax build-up or over-production of skin; both of these are uncommon) or the middle ear (e.g., ruptured tympanic membrane, which produces 30-40dB loss; fluid due to infection; damaged, missing or ossified ossicles).

The most obvious effect is that you can't hear quiet sounds. But hearing loss is more complicated than that! People with hearing loss have more difficulty understanding speech in noise (can't "hear out" the words). And the fact that different frequencies are affected differently and dynamic range is compressed ("loudness recruitment") means people with hearing loss have a hard time enjoying music.

Many people suffer from conductive hearing loss because of age, genetic predisposition, or environmental effects. With conductive hearing loss, hearing problems are associated with a failure in the vibration of the eardrum and/or movement of the ossicles. These problems are often dealt with through devices like hearing aids that amplify incoming sound waves to make vibration of the eardrum and movement of the ossicles more likely to occur.

Conductive hearing loss can be distinguished from sensorineural hearing loss by measuring hearing through bone conduction: you put a speaker against the mastoid bone behind the ear, and if hearing through bone is normal, or there's an "air-bone gap" (hearing is better through bone than air), then at least part of the loss is conductive.

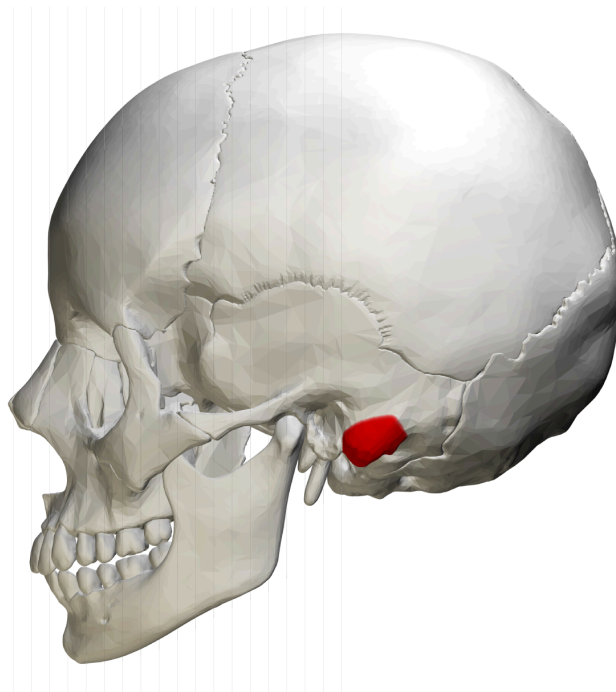


Figure 6.1.1. Mastoid process – lateral view. Conductive hearing loss can be tested by placing a speaker on the mastoid bone. (Credit: Database Center for Life Science (DBCLS). Provided by: Wikimedia Commons License: CC BY SA)

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55.

SENSORINEURAL HEARING LOSS

Learning Objectives

Know the possible causes of sensorineural hearing loss and indicate the most common cause

Understand what temporary threshold shift (TTS) is

Understand the impact of loss of outer hair cells

Sensorineural hearing loss is generally permanent (and unfortunately more common than conductive hearing loss). Sensorineural hearing loss is caused by damage to neurons (most commonly outer hair cells, but also inner hair cells and the auditory nerve) due to disease or exposure to loud noises. Frequencies in the 1000-5000Hz range are most strongly affected by environmental damage. Hearing loss is often preceded by a temporary threshold shift (TTS), which usually consists of a roughly 40dB loss after exposure to loud noise followed by a return to a normal hearing threshold within 48 hours.

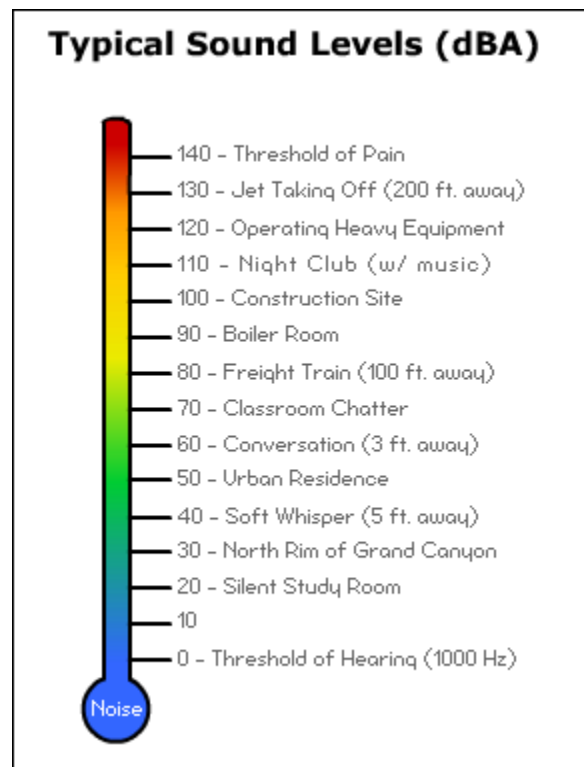


Fig.6.2.1. Decibel Levels. Here are some examples of noises at specific decibels. (Provided by: Occupational Safety and Health Administration. License: PDM)

Most sensorineural hearing loss is caused by the loss of outer hair cells. Damage to the stereocilia on the outer hair cells has three effects:

- Loss of the motile response blunts the frequency tuning, so sounds get mushed together.
- Loss of the amplification that outer hair cells provide at low sound levels results in an elevation of detection thresholds
- Loudness recruitment: Sensitivity to quiet sounds is reduced, but sensitivity to loud sounds is significantly increased, meaning that sounds are only tolerable in a small range

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AGE-RELATED HEARING LOSS

Learning Objectives

Know the frequencies that are most affected by age-related hearing loss

Know what term can be used instead of “age-related hearing loss” for this condition

Be able to discuss possible sex differences

Hearing tests are performed over a range of frequencies, usually from 250 to 8000 Hz, and can be displayed graphically in an audiogram shown below. Audiograms are used to measure adults by raising your right or left hand (or push a button) to depict when you hear a sound by your right ear or left ear while wearing headphones. The hearing threshold is measured in dB relative to the normal threshold, so that normal hearing registers as 0 dB at all frequencies. Hearing loss caused by noise typically shows a dip near the 4000 Hz frequency, irrespective of the frequency that caused the loss and often affects both ears. The most common form of hearing loss comes with age and is called presbycusis—literally elder ear. Such loss is increasingly severe at higher frequencies, and interferes with music appreciation and speech recognition.

High frequencies (above ~5kHz) are also affected by aging. It appears that men experience greater hearing loss. Perhaps it is because men are historically subjected to louder environments; we won't know until a new generation ages. In one study of males and females on Easter Island, where roads and jack hammers and other common auditory insults from the urban environment are absent, elderly males did not show greater hearing loss than elderly females. But this study is not broadly accepted or replicated. Otoacoustic emissions are stronger in females than in males. So we know there are some differences between genders, but we do not know how much these differences are caused by biological factors or social factors or how they may play out in day-to-day hearing.

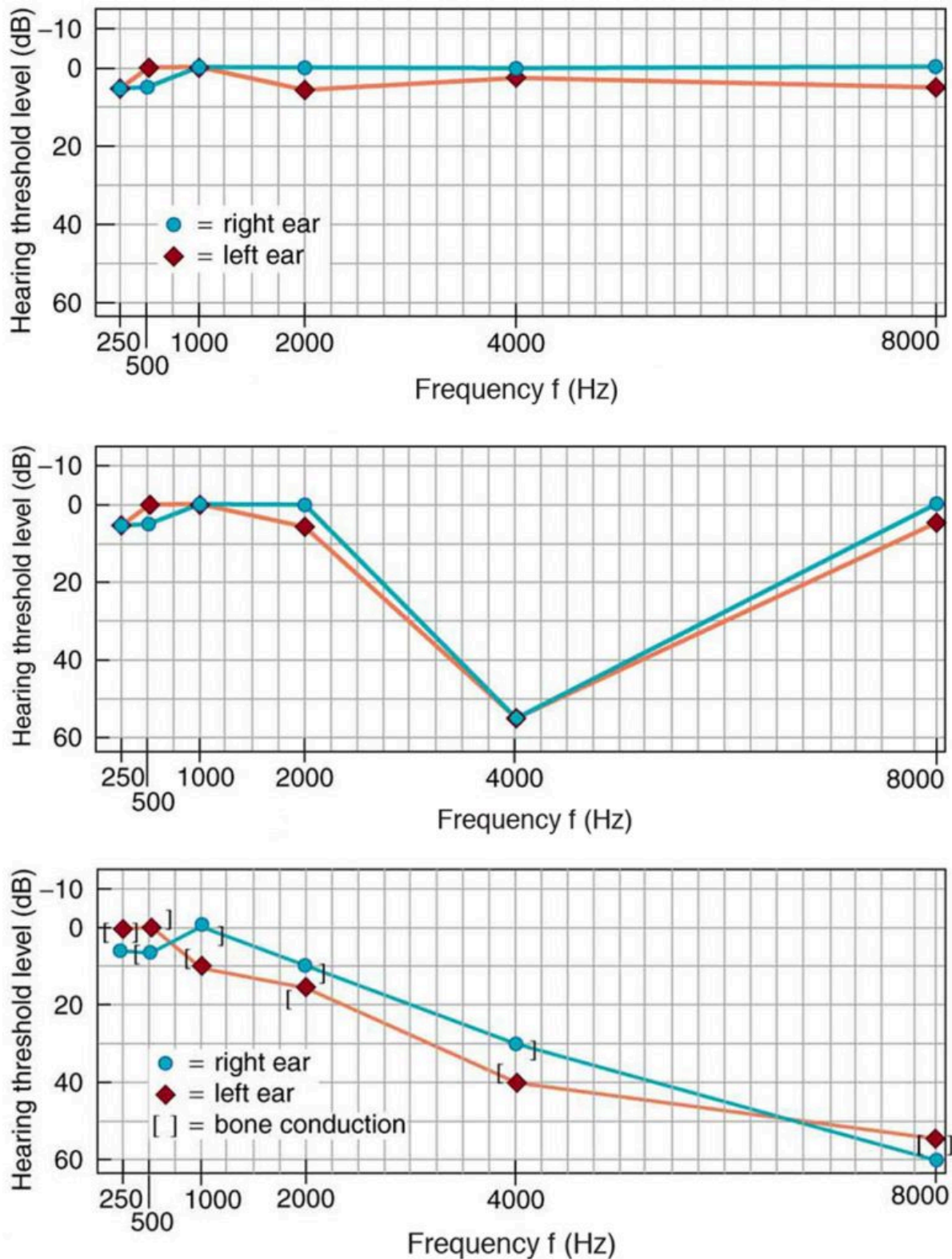


Fig.6.3.1. Hearing Threshold level vs. Frequency. Audiograms showing the threshold in intensity level versus frequency for three different individuals. Intensity level is measured relative to the normal threshold. The top left graph is that of a person with normal hearing. The graph to its right has a dip at 4000 Hz and is that of a child who suffered hearing loss due to a cap gun. The third graph is typical of presbycusis, the progressive loss of higher frequency hearing with age.

Tests performed by bone conduction (brackets) can distinguish nerve damage from middle ear damage. (Provided by: OpenStax. License: CC BY 4.0)

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OpenStax, College Physics for AP Courses Chapter 17.6 Hearing

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Adapted by: Aliciana Bezdicek and John Bates

TINNITUS

Learning Objectives

Be able to describe what it is and why it may happen

Be able to discuss the possible long-term impact of it

Tinnitus is phantom noise (“ringing”) in the ears. When sensorineural loss occurs, the brain is missing sensory input ... the brain imagines it’s hearing noise. This is just like a phantom limb. So tinnitus is generally a sign of sensorineural damage, although it is not necessarily accompanied by hearing loss.

Tinnitus is not a disease but a symptom resulting from a range of underlying causes that can include ear infections, foreign objects or wax in the ear, nose allergies that prevent (or induce) fluid drain and cause wax build-up. Tinnitus can also be caused by natural hearing impairment (as in aging), as a side-effect of some medications, and as a side-effect of genetic (congenital) hearing loss. However, the most common cause for tinnitus is noise-induced hearing loss.

Tinnitus can be perceived in one or both ears or in the head. It is usually described as a ringing noise, but in some patients it takes the form of a high pitched whining, buzzing, hissing, screaming, humming, tingling or whistling sound, or as ticking, clicking, roaring, “crickets” or “tree frogs” or “locusts,” tunes, songs, beeping, or even a pure steady tone like heard in a hearing test. (1) It has also been described as a “wooshing” sound, as of wind or waves. (2) Tinnitus can be intermittent or it can be continuous in which case it can be the cause of great distress. In some individuals, the intensity of tinnitus can be changed by shoulder, head, tongue, jaw, or eye movements. (3) Click on this link to try out a simulation of what one with tinnitus may experience hearing. Scroll down to the “Sounds of Tinnitus” section, and be sure to keep your volume low/medium.



Fig.6.4.1. Tinnitus can be experienced as ticking, clicking, beeping, buzzing screaming, whining and tingling. It can be very distressing. (Credit: Alexander Krivitskiy, Thomas Bormans, and Anh Nguyen. Edited by: Jarod Davis. Provided by Unsplash. License: CC-BY)

Prevention involves avoiding exposure to loud noise for longer periods. (4) If there is an underlying cause, treating it may lead to improvements. (5) Otherwise, typically, management involves psychoeducation or counseling as talk therapy. (6) Sound generators or hearing aids may help some. (4) As of 2013, there were no effective medications. (5) It is common, affecting about 10–15% of people. (6) Most, however, tolerate it well, and it is a significant problem in only 1–2% of people. (6) The word tinnitus comes from the Latin tinnire which means “to ring”. (5)

Besides being an annoying condition to which most people adapt, persistent tinnitus may cause anxiety and depression in some people. (7)(8) Tinnitus annoyance is more strongly associated with the psychological condition of the person than the loudness or frequency range. (9)(10) Psychological problems such as

depression, anxiety, sleep disturbances, and concentration difficulties are common in those with strongly annoying tinnitus. (11)(12) 45% of people with tinnitus have an anxiety disorder at some time in their life. (13)

Exercises

1. Although tinnitus is not a disease, it is a symptom resulting from a range of causes, such as ear infections, foreign objects in the ear, genetic hearing loss, noise-induced hearing loss, etc., and there are methods used to prevent it. Which of the following is the most common preventative measure to take in avoiding tinnitus?
 - A. Hearing aids
 - B. Attend psychoeducation and/or talk therapy.
 - C. Limiting exposure to loud noise for long periods of time
 - D. Avoid taking aspirin and Tylenol.
2. As of 2013, there are no effective medications for Tinnitus, but what does end up occurring with most people who have Tinnitus?
 - A. Most people experience anxiety disorders.
 - B. Most people experience depression.
 - C. Most people have a significant problem with handling it.
 - D. Most people tolerate it well.
3. One who experiences tinnitus may notice a phantom noise in a quiet environment daily. Although a phantom noise can be ignored, what is a common sound that people typically hear when they are living with tinnitus?
 - A. Ringing
 - B. Heart beat
 - C. Rhythmic drums
 - D. Tree frogs
4. Tinnitus can be perceived in ____ ears and is usually described as a ____ noise.
 - A. One ear only, dinging
 - B. One or both ears, chirping
 - C. One or both ears, ringing
 - D. Both ears, beeping
5. Tinnitus annoyance is more strongly associated with the ____ condition of the person than the loudness or frequency range. 45% of people with tinnitus have a(n) ____ disorder at some

time in their life.

- A. Sensorineural, sleeping
 - B. Sensorineural, anxiety
 - C. Psychological, sleeping
 - D. Psychological, anxiety
6. A youthful individual listens to music quite loud in their car on their thirty minute drives to work, five days a week, without ear protection. They have recently noticed that throughout their daily life, they hear a phantom ringing noise. This particular individual also has several relatives who wear hearing aids, due to their old age. Worried about their potential hearing damage, the individual goes to the doctor to get their hearing checked out, and they are told that they are experiencing tinnitus. The source of their tinnitus is most likely due to which of the following?
- A. Age-related hearing loss
 - B. Genetic predisposition to hearing loss
 - C. Ear-wax buildup
 - D. Exposure to loud noise for long periods of time
7. One who has tinnitus may experience phantom noises, such as ringing, whining, buzzing, tingling, whistling, etc. Moreover, the intensity of tinnitus can be changed by what type of movements of the body?
- A. Head movements only
 - B. Shoulder, head, jaw, tongue, and/or eye movement
 - C. Eye, head, tongue, and jaw movements
 - D. Head and jaw movements only
8. Below is a list of some types of hearing damage. Which of the following forms of hearing damage listed can stem from a side-effect of some medications?
- A. Age-related hearing loss
 - B. Tinnitus
 - C. Hidden hearing loss
9. One gets psychological testing done, and their results come back with depression, anxiety, and concentration difficulties. They have also complained about hearing a ringing noise constantly throughout the day, irritating them greatly, and they cannot ignore it. It turns out that they have a form of tinnitus that affects what percent of people?
- A. 1-2%
 - B. 45-50%
 - C. 10-15%

10. Tinnitus, which is phantom noise or “ringing” in the ears, is a common symptom of underlying causes that can include ear infections, foreign objects or wax in the ear, nose allergies that prevent fluid drain, natural hearing impairment with aging, noise-induced hearing loss, or a side-effect of medication or of genetic hearing loss. That being said, what is happening in the brain, on a neurological level, when one experiences Tinnitus?
- A. The brain is missing sensory input, i.e. the brain imagines hearing noise.
 - B. The hair cells in the ear are moving, even when there is not a sound wave to be heard, causing one to believe that they are hearing noise.
 - C. The nerves in the ear, such as the vestibulocochlear nerve, are damaged, and they are retrieving sound from the last source that they remember prior to injury, creating a phantom noise.

Answer Key:

- 1. C
- 2. D
- 3. A
- 4. C
- 5. D
- 6. D
- 7. B
- 8. B
- 9. A
- 10. A

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References

1. ↑ RNID.org.uk: Information and resources: Tinnitus: About tinnitus: What is tinnitus
2. ↑ MedlinePlus Encyclopedia 003043
3. ↑ Simmons R, Dambra C, Lobarinas E, Stocking C, Salvi R. (2008). Head, Neck, and Eye Movements That Modulate Tinnitus. *Semin Hear.* 29(4):361-370. PMID 19183705
4. “Tinnitus”. NIH – National Institute on Deafness and Other Communication Disorders (NIDCD). 6 March 2017. Archived from the original on 3 April 2019. Retrieved 20 September 2019.
5. Baguley, D; McFerran, D; Hall, D (Nov 9, 2013). “Tinnitus” (PDF). *The Lancet.* 382(9904): 1600–07. doi:10.1016/S0140-6736(13)60142-7. PMID 23827090. Archived (PDF) from the original on 2018-04-11.
6. Langguth, B; Kreuzer, PM; Kleinjung, T; De Ridder, D (Sep 2013). “Tinnitus: causes and clinical management”. *The Lancet Neurology.* 12 (9): 920–30. doi:10.1016/S1474-4422(13)70160-1. PMID 23948178.
7. Andersson G (2002). “Psychological aspects of tinnitus and the application of cognitive-behavioral therapy”. *Clinical Psychology Review.* 22 (7): 977–90. doi:10.1016/s0272-7358(01)00124-6. PMID 12238249.
8. Reiss M, Reiss G (1999). “Some psychological aspects of tinnitus”. *Perceptual and Motor Skills.* 88 (3 Pt 1): 790–92. doi:10.2466/pms.1999.88.3.790. PMID 10407886.
9. Baguley DM (2002). “Mechanisms of tinnitus”. *British Medical Bulletin.* 63: 195–212. doi:10.1093/bmb/63.1.195. PMID 12324394.
10. Henry JA, Meikele MB (1999). “Pulsed versus continuous tones for evaluating the loudness of tinnitus”. *Journal of the American Academy of Audiology.* 10 (5): 261–72. PMID 10331618.
11. Henry JA, Dennis KC, Schechter MA (2005). “General review of tinnitus: Prevalence, mechanisms, effects, and management”. *Journal of Speech, Language, and Hearing Research.* 48 (5): 1204–35. doi:10.1044/1092-4388(2005/084). PMID 16411806.
12. Davies A, Rafie EA (2000). “Epidemiology of Tinnitus”. In Tyler, RS (ed.). *Tinnitus Handbook.* San

Diego: Singular. pp. 1–23. OCLC 42771695.

13. Pattyn T, Van Den Eede F, Vanneste S, Cassiers L, Veltman DJ, Van De Heyning P, Sabbe BC (2015). “Tinnitus and anxiety disorders: A review”. *Hearing Research*. 333: 255–65. doi:10.1016/j.heares.2015.08.014. PMID 26342399

HIDDEN HEARING LOSS

Learning Objectives

Be able to discuss the impact of exposure to loud noises related to this condition

Be able to describe the impact in behavioral thresholds

Hidden hearing loss wasn't reported before 2009 (Kujawa et al, J. Neurosci 19:14077-14085) but it is rapidly garnering broad attention. Even when the hair cells are not damaged and behavioral thresholds recover completely after loud noise exposure, 50% of the synapses are gone! But hearing thresholds as measured clinically and reported in audiograms are still the same. The technical name for hidden hearing loss is **cochlear synaptopathy**.

To explore this finding of noise-induced primary neuropathy further, and to clarify interpretation, the observations were repeated for an exposure that produced only temporary threshold shift (TTS) in fully adult animals (Kujawa and Liberman, 2009). In this work, mice from the same inbred strain were exposed to a band of noise placed in the region of best threshold sensitivity. The noise was titrated in level and duration to produce a large, acute threshold shift (30–40 dB at 24 h), but one that recovered by 2 weeks, without hair cell loss. Immunostained cochlear whole mounts and plastic-embedded sections (Fig. 6.5.1), imaged by confocal and conventional light microscopy, were assessed to quantify hair cells, cochlear neurons, and synaptic structures providing the communication conduits. Hair cell-based distortion product otoacoustic emissions (DPOAEs) and neural-based auditory brainstem responses (ABRs) or compound action potentials (CAPs) of the auditory nerve were used to assess the peripheral consequences of the noise on function (Fig. 6.5.1).

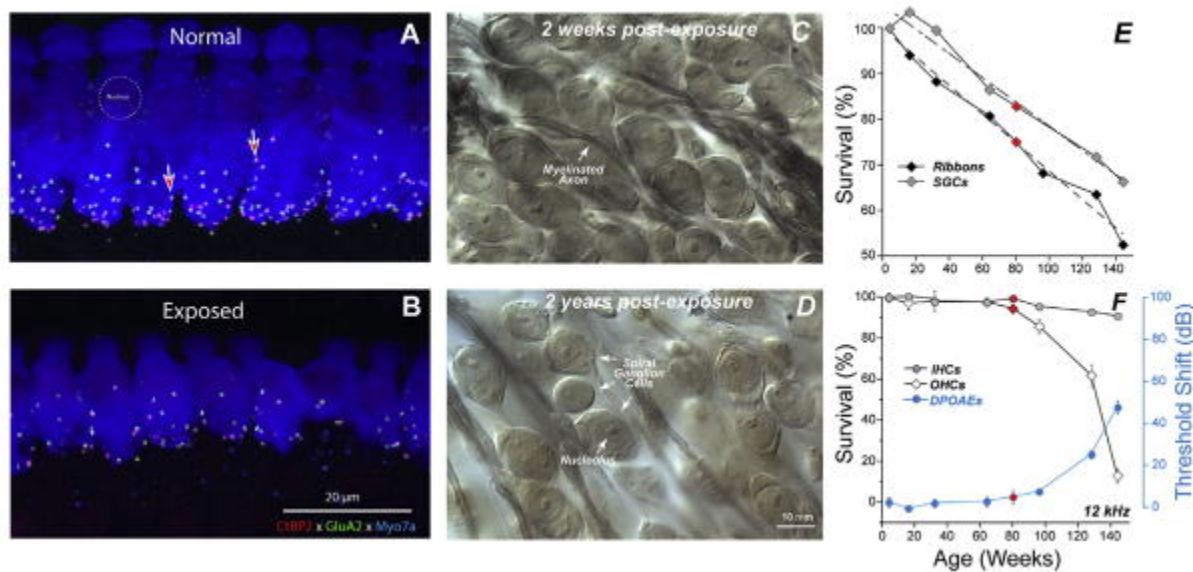


Fig.6.5.1. Noise-induced and age-related loss of synapses and SGNs. Evaluating synaptopathy by triple-staining cochlear whole mounts for a pre-synaptic marker (CtBP2-red), a postsynaptic marker (GluA2-green) and a hair cell marker (Myosin VIIa-blue). Confocal z-stacks in the IHC area from a control (A) and a noise-exposed mouse (B), 2 wks post exposure. Light micrographs of osmium-stained plastic sections from noise-exposed ears, 2 wks (C) or 2 yrs (D) post exposure. Exposure in B and D was 8-16 kHz, 2 h, 100 dB SPL, delivered at 16 wk to CBA/CaJ mice. (E) In aging ears from the same inbred strain, synaptic counts at IHCs decrease steadily from 4 to 144 wks and parallel ganglion cell loss follows whereas, (F) threshold loss begins comparatively later and accelerates beyond 80 wks, mirrored by accelerating loss of OHCs. IHC loss is trivial at any age. Red symbols flag 80 wk data points for all measurements. After Kujawa and Liberman 2006, 2009; Sergeyenko et al. (2013). (Cedit: M. Charles Liberman and Sharon G. Kujawa, Noise-induced and age-related loss of synapses and SGNs, CC BY 4.0)

Presaging the ganglion cell losses, results of these studies revealed an acute loss of synapses between IHCs and the peripheral terminals of the spiral ganglion neurons that contact them (Kujawa and Liberman, 2009). Although thresholds recovered, by design, and no hair cells were lost, IHC synaptic losses were greater than 40% in basal cochlear regions, when assessed 24 h post noise, and were stable 2 and 8 weeks later. Losses were proportional in magnitude and cochlear location to the SGC loss observed in the previous series, suggesting that this interruption of IHC-to-neural communication set the stage for neurodegeneration.

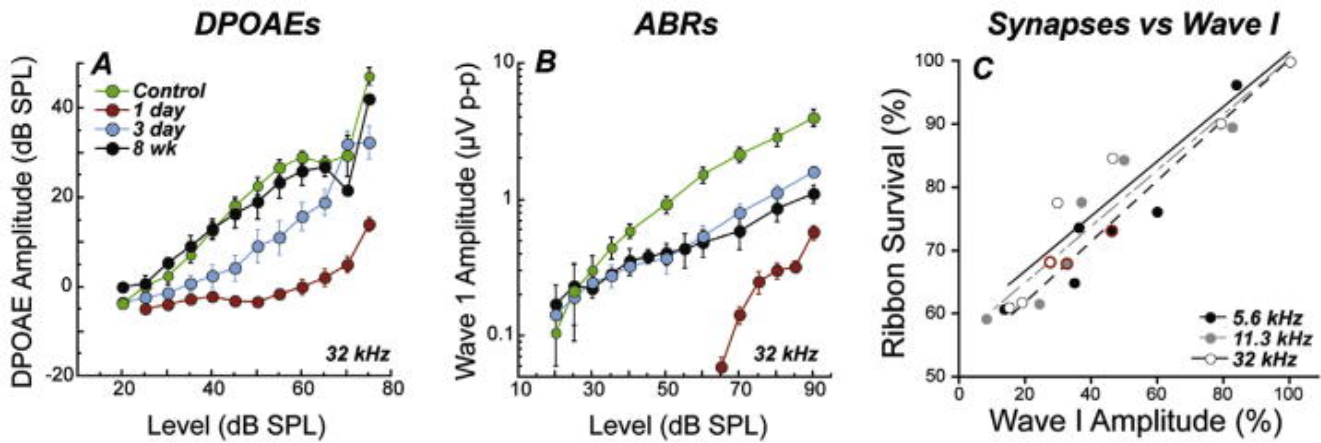


Fig6.5.2 Response amplitudes and synapse counts. Permanent reductions in ABR, but not DPOAE amplitudes in ears with recovered thresholds after noise. Shown are DPOAE (A) and ABR wave 1 (B) response growth functions in the region of maximum acute TTS 1 d and 2 wk after exposure (as in Fig. 1) to 16 wk CBA/CaJ mice; unexposed controls shown for comparison. Neural response amplitude declines are proportional to synaptic and neural losses in aging CBA/CaJ, where synapses are plotted vs mean wave 1 amplitudes (at 80 dB SPL in 4–128 wk animals (C). Panels A,B from Fernandez et al., 2015; Panel C from Sergeyenko et al., 2013. (credit: M. Charles Liberman and Sharon G. Kujawa, Response amplitudes and synapse counts, CC BY 4.0)

For an additional explanation about hidden hearing loss, take a peak at the video link here and included below.



One or more interactive elements has been excluded from this version of the text. You can view them online here: <https://pressbooks.umn.edu/sensationandperception/?p=479#oembed-1>

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National Center for Biotechnology Information, "Cochlear synaptopathy in acquired sensorineural hearing loss: Manifestations and mechanisms" by M. Charles Liberman and Sharon G. Kujawa

Provided by: Hearing Research

URL: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5438769/>

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References:

Kujawa, S. G., & Liberman, M. C. (2009). Adding insult to injury: cochlear nerve degeneration after “temporary” noise-induced hearing loss. *Journal of Neuroscience*, 29(45), 14077-14085.

Liberman, M. C., & Kujawa, S. G. (2017). Cochlear synaptopathy in acquired sensorineural hearing loss: Manifestations and mechanisms. *Hearing research*, 349, 138-147.

PREVENTION OF HEARING LOSS

Learning Objectives

Understand the best ways to prevent hearing loss

Be able to discuss the possible treatments for children who get frequent ear infections

Be able to describe what ototoxic means, and its relation to hearing loss

The best way to prevent hearing loss is to take necessary precautions when one is exposed to loud sounds. These precautions include wearing ear plugs. Be nice to your future self and wear them at concerts and loud sporting events or when operating power tools. If you don't like the muffled sound, buy some fancy ones that attenuate all frequencies equally. While we all lose hearing with age (98% of 80 year-olds have hearing loss), a lot of that is because of exposure to loud noises over the lifetime. Additionally, hearing loss can be due to simple illnesses, such as the flu. This means that getting a flu shot, as well as other vaccines, can be a preventative measure when it comes to hearing loss. Lastly, in today's world, earbuds are used frequently. It is advised that a person uses caution when listening to music through earbuds, and limits the volume so as not to damage the ear.



(a)



(b)

Figure 6.6.1. Environmental factors that can lead to conductive hearing loss include regular exposure to loud music or construction equipment. (a) Rock musicians and (b) construction workers are at risk for this type of hearing loss. (Provided by: OpenStax. License: CC BY 4.0. (a): modification of work by Kenny Sun; (b): modification of work by Nick Allen)

Ear infections can also lead to hearing loss. If this happens occasionally, it can be easily medicated. For children who get frequent ear infections, ventilation tubes can be implanted in the tympanic membrane. The tubes relieve pressure from infection to avoid larger tear in membrane.

Some medicines are ototoxic. An ototoxic medication attacks the inner ear and auditory nerves. The resulting hearing loss can be temporary or permanent. So be prudent when using Aspirin or other NSAIDs, and be careful with your antibiotics. Overuse or an overdose can result in these ototoxic hearing loss.

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US Department of Health and Human Services

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60.

HEARING AIDS

Learning Objectives

Understand hearing aids are most effective in what cases of hearing loss and why

Know the main features of hearing aids

Be able to discuss the prevalence of hearing aids

Hearing aids amplify sound, so they rely on functioning hair cells. Hearing aids do not restore normal hearing when used to treat sensorineural loss, because the effects of neural damage are so complicated. Given the mechanical nature by which the sound wave stimulus is transmitted from the eardrum through the ossicles to the oval window of the cochlea, some degree of hearing loss is inevitable. With conductive hearing loss, hearing problems are associated with a failure in the vibration of the eardrum and/or movement of the ossicles. These problems are often dealt with through devices like hearing aids that amplify incoming sound waves to make vibration of the eardrum and movement of the ossicles more likely to occur.



Fig.6.7.1. Hearing aids can sit on the inside, or outside of the ear so they are less obvious to the people around. (Credit: Mark Paton. Provided by: Unsplash. License: CC-BY)

Modern hearing aids have, in addition to basic sound amplification, these features: amplitude compression (mimicking the natural compressive response of the inner ear, which you lose when you lose OHC; this reduces Loudness Recruitment), and noise reduction — a filtering algorithm that tries to take out background noise. Noise reduction improves comfort, but doesn't improve speech intelligibility ... and the algorithm doesn't know which sound you're trying to pick out, and might reduce the wrong sound. Current research is trying to use brain-steering: an EEG signal can detect which speech sound you're paying attention to, and this can be used to control amplification. Maybe someday.

Only about a quarter of the people who would benefit from hearing aids actually use them. Adoption rates are higher (30-40%) in countries like Norway where insurance covers hearing aids. These low rates may be due to the \$2k-\$3k price tag that insurance often may not cover. Another thing that keeps adoption rates down is that they really don't help in all situations. On the bright side, new FDA regulations are coming out that might create over-the-counter hearing aids and bring down the price!

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COCHLEAR IMPLANTS

Learning Objectives

Understand the conditions in which cochlear implant can be an option

Understand how cochlear implants are implemented

When the hearing problem is associated with a failure to transmit neural signals from the cochlea to the brain, it is called sensorineural hearing loss. One disease that results in sensorineural hearing loss is Ménière's disease. Although not well understood, Ménière's disease results in a degeneration of inner ear structures that can lead to hearing loss, tinnitus (constant ringing or buzzing), vertigo (a sense of spinning), and an increase in pressure within the inner ear (Semaan & Megerian, 2011). This kind of loss cannot be treated with hearing aids, but some individuals might be candidates for a cochlear implant as a treatment option. Cochlear implants are electronic devices that consist of a microphone, a speech processor, and an electrode array. The device receives incoming sound information and directly stimulates the auditory nerve to transmit information to the brain.

A cochlear implant is very different from a hearing aid. Hearing aids amplify sounds so they may be detected by damaged ears. Cochlear implants bypass damaged portions of the ear and directly stimulate the auditory nerve. Signals generated by the implant are sent by way of the auditory nerve to the brain, which recognizes the signals as sound. Hearing through a cochlear implant is different from normal hearing and takes time to learn or relearn. However, it allows many people to recognize warning signals, understand other sounds in the environment, and understand speech in person or over the telephone.

Children and adults who are deaf or severely hard-of-hearing can be fitted for cochlear implants. Use of a cochlear implant requires both a surgical procedure and significant therapy to learn or relearn the sense of hearing. Not everyone performs at the same level with this device. The decision to receive an implant should involve discussions with medical specialists, including an experienced cochlear-implant surgeon. The process can be expensive. For example, a person's health insurance may cover the expense, but not always. Some individuals may choose not to have a cochlear implant for a variety of personal reasons. Surgical implantations

are almost always safe, although complications are a risk factor, just as with any kind of surgery. An additional consideration is learning to interpret the sounds created by an implant. This process takes time and practice. Speech-language pathologists and audiologists are frequently involved in this learning process. Prior to implantation, all of these factors need to be considered. As of December 2012, approximately 324,200 registered devices have been implanted worldwide. In the United States, roughly 58,000 devices have been implanted in adults and 38,000 in children. (Estimates provided by the U.S. Food and Drug Administration [FDA], as reported by cochlear implant manufacturers.)

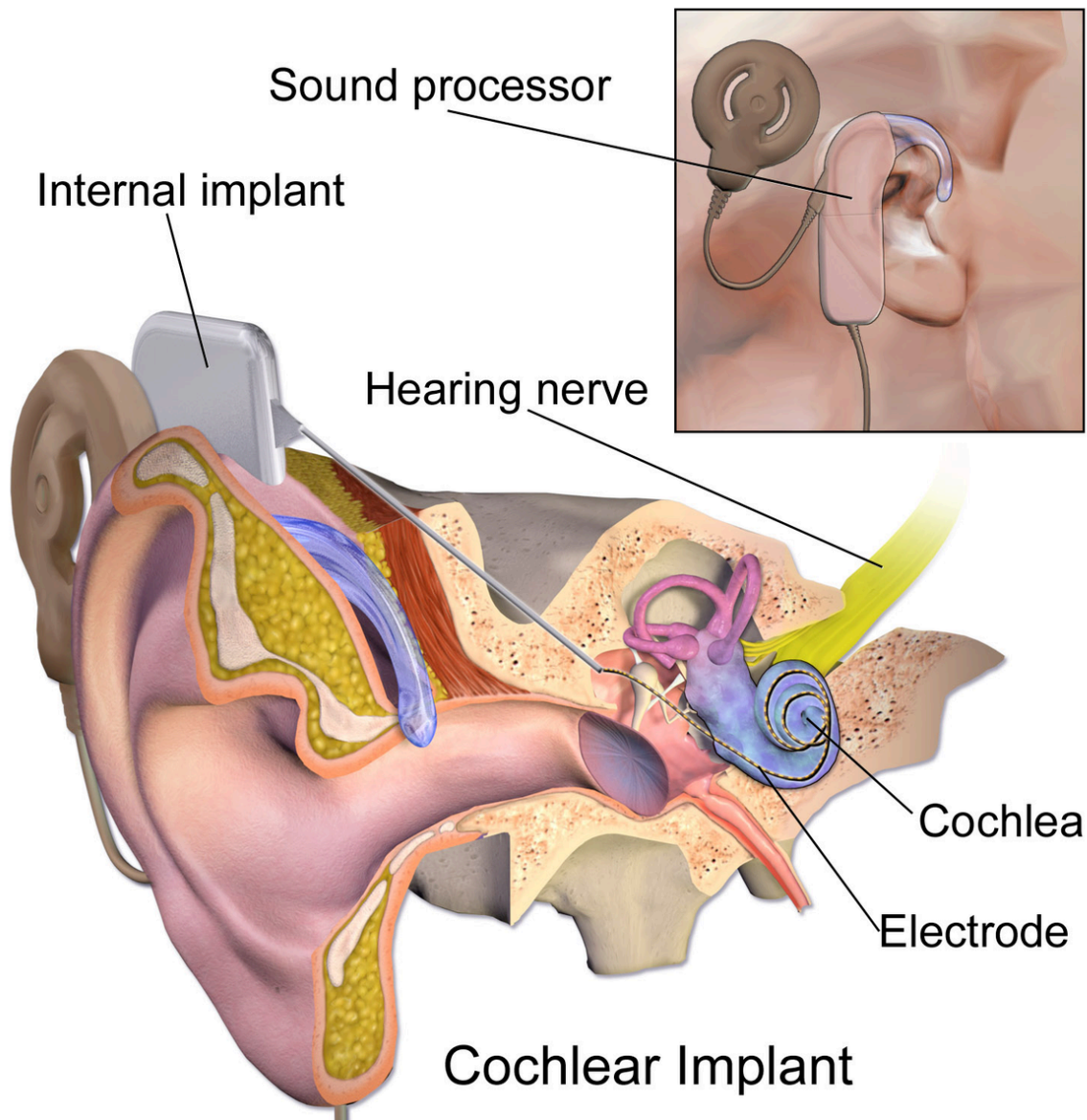


Figure 6.8.1. Simplified image of a cochlear implant. Includes the technology of the implant, as well as the associated ear regions that are involved. (Credit: Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". Provided by: Wikijournal of Medicine. License: CC BY 3.0)

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National Institutes of Deafness and Other Communication Disorders (NIDCD), Cochlear Implants

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Adapted by: Yichen Dai

CRITICAL BANDS AND MASKING

Learning Objectives

Be able to discuss the evidence why masking happens only in similar frequencies

Understand the implications of motion of the basilar membrane

Understand what place coding is

One of the most important principles of hearing—frequency analysis—is established in the cochlea. In a way, the action of the cochlea can be likened to that of a prism: the many frequencies that make up a complex sound are broken down into their constituent frequencies, with low frequencies creating maximal basilar-membrane vibrations near the apex of the cochlea and high frequencies creating maximal basilar-membrane vibrations nearer the base of the cochlea. This decomposition of sound into its constituent frequencies, and the frequency-to-place mapping, or “tonotopic” representation, is a major organizational principle of the auditory system (place coding), and is maintained in the neural representation of sounds all the way from the cochlea to the primary auditory cortex. The decomposition of sound into its constituent frequency components is part of what allows us to hear more than one sound at a time.

Masking is the process by which the presence of one sound makes another sound more difficult to hear. We all encounter masking in our everyday lives, when we fail to hear the phone ring while we are taking a shower, or when we struggle to follow a conversation in a noisy restaurant. In general, a more intense sound will mask a less intense sound, assuming that the frequency content of the sounds overlap. In psychophysical studies, a tone only masks, or elevates thresholds for, nearby tones. This is taken as evidence that frequencies that are widely separated are processed separately. In other words, the activity in the cochlea produced by a masking sound “swamps” that produced by the target sound, making the target sound harder to perceive.

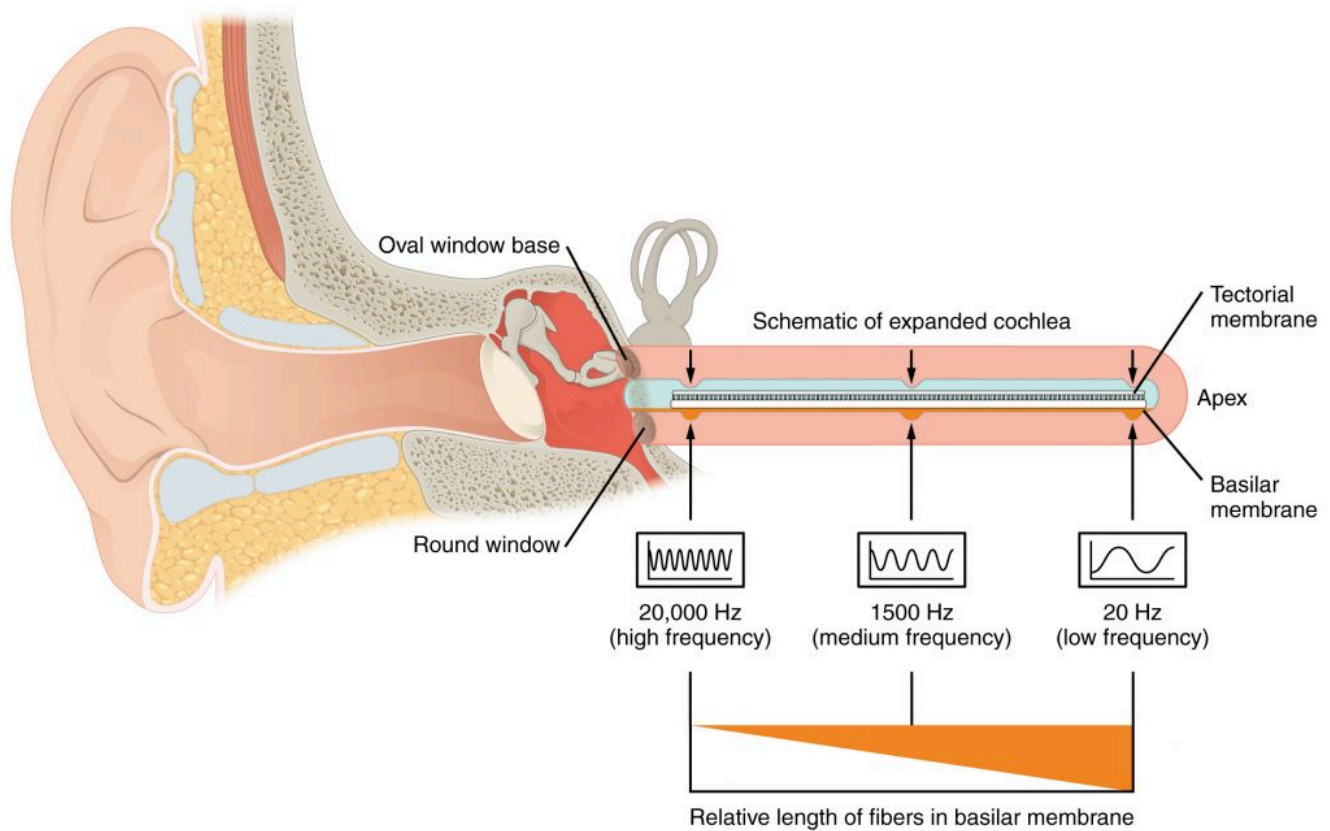


Fig. 6.9.1. Frequency coding in the cochlea, showing the change in characteristic frequency from base to apex. (Provided by: Wikipedia. License: CC BY 4.0)

The physical origin of this phenomenon is likely the motion of the basilar membrane. Support for this idea comes from the upward spread of masking — interference is stronger in one direction than the other, and this matches asymmetries in how the basilar membrane moves. Because of the way that filtering in the cochlea functions, low-frequency sounds are more likely to mask high frequencies than vice versa, particularly at high sound intensities. The loss of sharp cochlear tuning that often accompanies cochlear damage leads to broader filtering and more masking—a physiological phenomenon that is likely to contribute to the difficulties experienced by people with hearing loss in noisy environments (Moore, 2007).

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63.

AUDITORY PATHWAYS TO THE BRAIN

Learning Objectives

Know the three different brainstem nuclei

Understand the role of medial geniculate nucleus

A sensory pathway that carries peripheral sensations to the brain is referred to as an ascending pathway, or ascending tract. The sensory pathway of audition ascends through three brainstem nuclei. Audition begins by traveling along the vestibulocochlear nerve, which synapses with neurons in the cochlear nuclei of the superior medulla. The cochlear nuclei receives information from the cochlea. The input received in the cochlear nuclei is only from one side of the body.

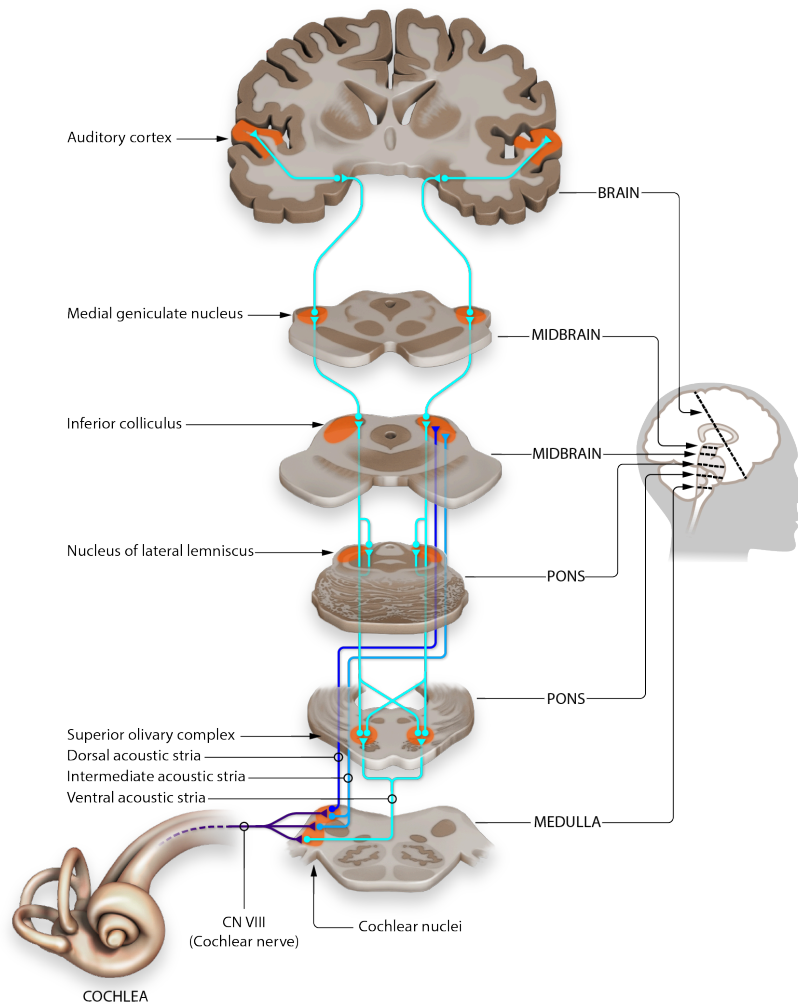


Fig.6.10.1. Schematic figure of the auditory neural pathway. The auditory pathway starts at the cochlear nucleus, then the superior olivary complex, and inferior colliculus, and finally medial geniculate nucleus. The information is decoded and integrated by each relay nucleus in the pathway and finally projected to the auditory cortex.

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The next part of the ascending process of auditory information in the brainstem is the superior olivary nucleus. The superior olivary nucleus takes the information from the cochlear nucleus and begins the process of interpreting and combining information. This begins the process of sound localization through estimating time difference and intensity between each ear (Christov, Nelson, & Gluth 2018).

After the superior olivary nucleus, auditory processing continues on to a nucleus called the inferior colliculus (IC). The inferior colliculus (IC), a midbrain structure that integrates the vast majority of ascending auditory information and projects via the thalamus to the auditory cortex. The IC is also a point of convergence for corticofugal (information from the cerebral cortex) input and input originating outside the

auditory pathway. Pathophysiological changes in the IC can alter all aspects of auditory perception and aid in localization of sound.

The medial geniculate nucleus of the thalamus then receives the auditory information from the three brainstem nuclei. The information is integrated and the medial geniculate nucleus projects that information to the auditory cortex in the temporal lobe of the cerebral cortex.

Additionally, feel free to take a look at this great video!



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Adapted by: Nicole Engelhart

“Tinnitus-related changes in the inferior colliculus,” Joel I. Berger and Ben Coomber

Provided by: Frontiers in Neuroscience

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Adapted by: Nicole Engelhart

References

Christov, F., Nelson, E. G., & Gluth, M. B. (2018). Human Superior Olivary Nucleus Neuron Populations in Subjects With Normal Hearing and Presbycusis. *Annals of Otology, Rhinology & Laryngology*, 127(8), 527-535.

PRIMARY AUDITORY CORTEX

Learning Objectives

Know where Heschl's gyrus is

Be able to describe what tonotopic maps in A1 are

Heschl's gyrus is a part of the temporal lobe, and the primary auditory cortex (A1) is located on it – in the superior temporal cortex. The A1 contains tonotopic maps. The word “tonotopic” means that neurons that respond to similar frequencies are close to each other in the cortex. On a fine scale, this is like orientation pinwheels. Retinotopy, the mapping of visual input from the retina to neurons, is an example of this on a more coarse scale. Tonotopic maps can shift with experience. For example, monkeys who get really good at discrimination sounds in a particular frequency band grow fatter cortical representations of that frequency. Additionally, experienced musicians tend to have elaborated A1 maps.

Primary auditory cortex

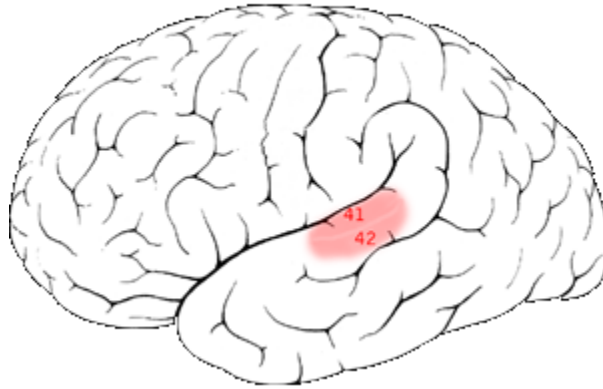


Fig. 6.11.1. The primary auditory cortex (PAC) is found on the posterior superior temporal gyrus and portions of the planum temporale and Heschl's gyrus. The PAC is located in Brodmann areas 41 and 42. (Provided by: OpenStax. License: CC-BY 4.0)

It is important to note that we don't simply perceive the tones represented in the A1. For example, we perceive the missing fundamental in a series of harmonic tones. The A1 is necessary for pitch perception, but not duration information. It is not sufficient for pitch perception, as damage in inferior temporal cortex affects our ability to identify tones. The A1 is surrounded by a region of cortex called the Belt area. Neurons in the belt area respond to the combinations of frequency and details of timing that define more complex characteristics of sound.

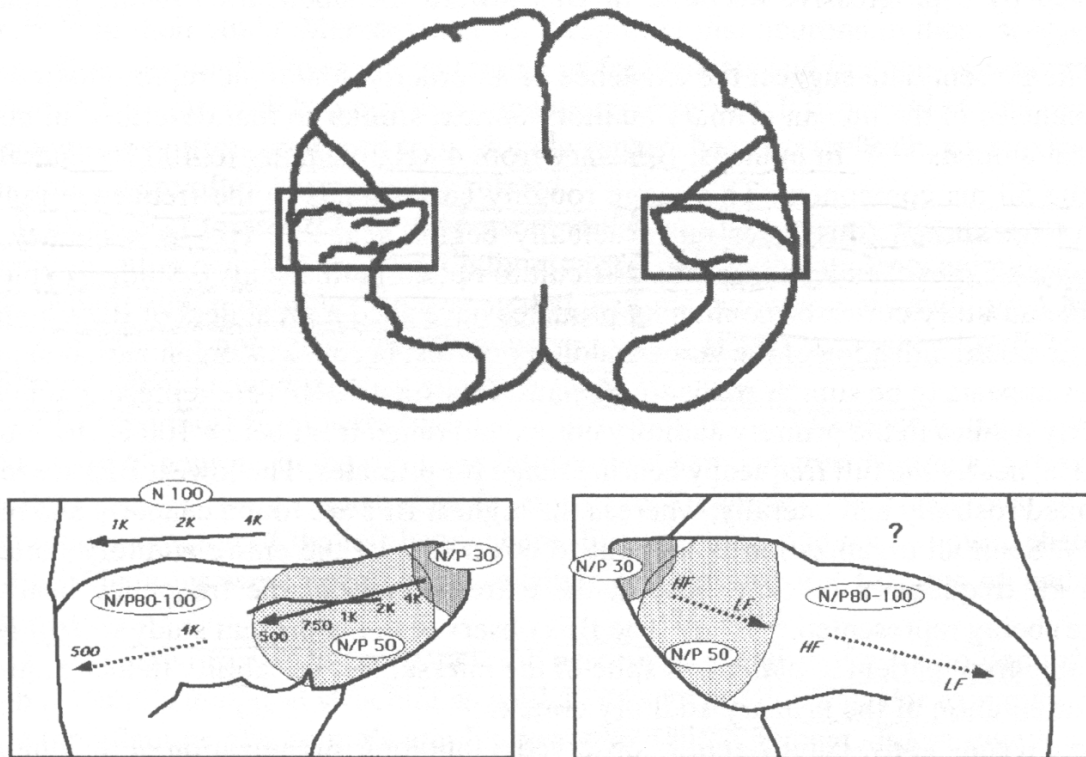


Fig.6.11.2. This figure shows a schematic representation of the tonotopic organization of the auditory cortices of both the left and right hemispheres. Low frequencies (500 HZ) are represented laterally or closer to the surface of the cortex and high frequencies represented medially or closer to the center of the brain. (Provided by: OpenStax. License: CC-BY 4.0)

Here is a video that provides an additional overview on tonotopy!



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Adapted by: Maida Fazlic

PITCH PERCEPTION

Learning Objectives

Understand why pitch perception is different than only tone presentation

Be able to describe what brain regions play a role in pitch perception

Pitch plays a crucial role in acoustic communication. Pitch variations over time provide the basis of melody for most types of music; pitch contours in speech provide us with important prosodic information in non-tone languages, such as English, and help define the meaning of words in tone languages, such as Mandarin Chinese. Pitch is essentially the perceptual correlate of waveform periodicity, or repetition rate: The faster a waveform repeats over time, the higher is its perceived pitch. The most common pitch-evoking sounds are known as harmonic complex tones. They are complex because they consist of more than one frequency, and they are harmonic because the frequencies are all integer multiples of a common fundamental frequency (F_0). For instance, a harmonic complex tone with a F_0 of 100 Hz would also contain energy at frequencies of 200, 300, 400 Hz, and so on. These higher frequencies are known as harmonics or overtones, and they also play an important role in determining the pitch of a sound. In fact, even if the energy at the F_0 is absent or masked, we generally still perceive the remaining sound to have a pitch corresponding to the F_0 . This phenomenon is known as the “pitch of the missing fundamental,” and it has played an important role in the formation of theories and models about pitch (de Cheveigné, 2005). We hear pitch with sufficient accuracy to perceive melodies over a range of F_0 s from about 30 Hz (Pressnitzer, Patterson, & Krumbholz, 2001) up to about 4–5 kHz (Attneave & Olson, 1971; Oxenham, Micheyl, Keebler, Loper, & Santurette, 2011). This range also corresponds quite well to the range covered by musical instruments; for instance, the modern grand piano has notes that extend from 27.5 Hz to 4,186 Hz. We are able to discriminate changes in frequency above 5,000 Hz, but we are no longer very accurate in recognizing melodies or judging musical intervals.

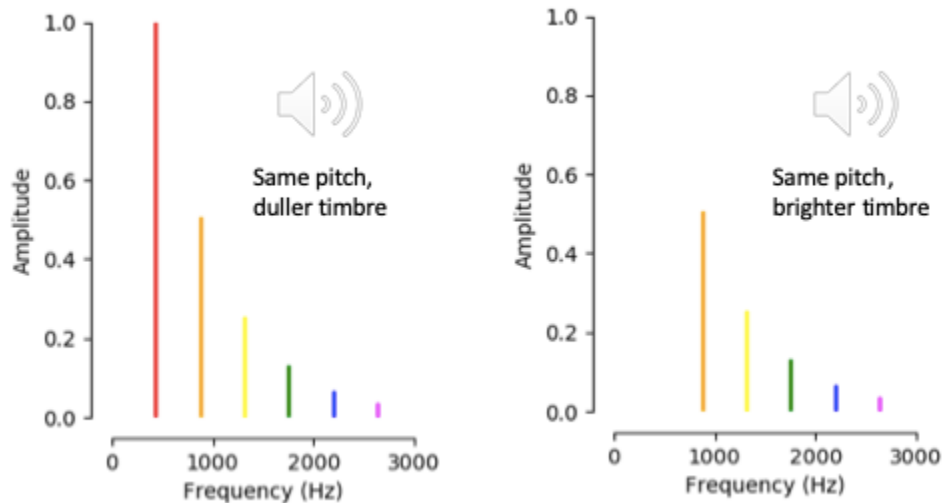


Figure 6.12.1 The case of the missing fundamental. The sounds represented by the 2 (Fourier) spectra have the same pitch (you would hum the same note to match them) because the spacing of the harmonics determines the perceived pitch, not the presence of the fundamental frequency. The leftmost plot corresponds to the first sound file below; the right plot corresponds to the second. (Provided by Cheryl Olman, University of Minnesota. License: CC BY 4.0)



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It turns out that primary auditory cortex (A1) is necessary but not sufficient for pitch perception. If you have damage to A1, you lose the ability to identify pitches. But the neural responses in A1 are not enough to predict what pitch someone will perceive: if you experience brain damage in inferior temporal cortex, you lose the ability to recognize tones.

For more information about pitch perception, here is a video about psychoacoustics.



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Oxenham, A. J. (2020). Hearing. In R. Biswas-Diener & E. Diener (Eds), *Noba textbook series: Psychology*. Champaign, IL: DEF publishers. Retrieved from <http://noba.to/jry3cu78>

References:

- Attneave, F., & Olson, R. K. (1971). Pitch as a medium: A new approach to psychophysical scaling. *American Journal of Psychology*, 84, 147–166.
- Oxenham, A. J., Micheyl, C., Keebler, M. V., Loper, A., & Santurette, S. (2011). Pitch perception beyond the traditional existence region of pitch. *Proceedings of the National Academy of Sciences USA*, 108, 7629–7634.
- Pressnitzer, D., Patterson, R. D., & Krumbholz, K. (2001). The lower limit of melodic pitch. *Journal of the Acoustical Society of America*, 109, 2074–2084.
- de Cheveigné, A. (2005). Pitch perception models. In C. J. Plack, A. J. Oxenham, A. N. Popper, & R. Fay (Eds.), *Pitch: Neural coding and perception* (pp. 169–233). New York, NY: Springer Verlag.

66.

SOUND IDENTITY VS. LOCATION

Learning Objectives

Know what “what” and “where” pathways represents

Be able to describe their technical names

Be able to discuss evidence for how these pathways were discovered

Beyond the cortical representation of the A1 and the belt area lies the “what” and “where” pathways of the ear. Through modern neuroimaging studies as well as lesion studies, we know sound identity is represented in a more ventral extended network whereas sound location is represented in a more dorsal extended network. The following paragraph is from the OpenStax textbook on visual processing, but the general principles apply to auditory processing, as well!

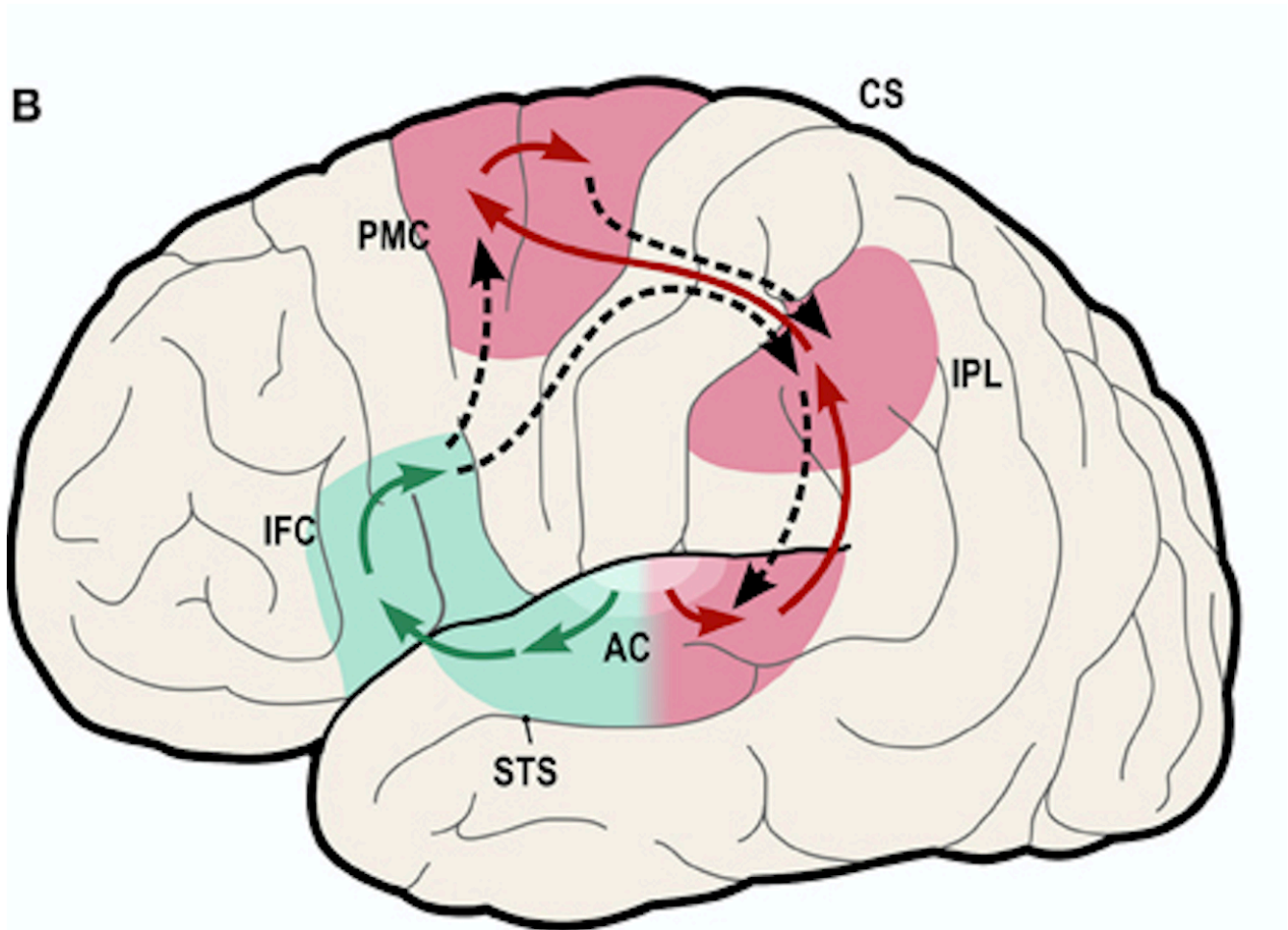


Fig.6.13.1 Model of dual-stream auditory processing in the primate brain, from Rauschecker (2011). Dorsal (red) and ventral (green) auditory pathways are shown in the human brain. Solid arrows indicate ascending projections from auditory cortex, while dashed arrows indicate reciprocal projections back to the auditory cortex. AC, auditory cortex; AL/CL, anterolateral/caudolateral superior temporal gyrus; CS, central sulcus; DLPFC, dorsolateral prefrontal cortex; IFC, inferior frontal cortex; IPL, inferior parietal lobule; IPS, inferior parietal sulcus; PFC, prefrontal cortex; PMc, premotor cortex; STS, superior temporal sulcus; VLPFC, ventrolateral prefrontal cortex. (credit: Aniruddh D. Patel, and John R. Iversen. Provided by: Wikipedia. License: CC BY 3.0 Modifications: Modified to only show panel B, the human brain).

The ventral stream identifies visual stimuli and their significance. Because the ventral stream uses temporal lobe structures, it begins to interact with the non-visual cortex and may be important in visual stimuli becoming part of memories. The dorsal stream locates objects in space and helps in guiding movements of the body in response to visual inputs. The dorsal stream enters the parietal lobe, where it interacts with somatosensory cortical areas that are important for our perception of the body and its movements. The dorsal stream can then influence frontal lobe activity where motor functions originate.

Authored by: Cheryl Olman

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67.

ACTIVE LEARNING EXERCISES - HEARING LOSS AND MASKING



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SECTION VII

HEARING IN COMPLEX ENVIRONMENTS

This final chapter on the auditory senses covers the more complex problems of hearing in a busy world. One topic is Auditory Scene Analysis: you can never close your ears, so you're constantly bombarded with the sounds around you, but how do you make sense of where those sounds are coming from? A second topic is speech production and speech processing. Understanding spoken language is a remarkable auditory skill.

This chapter was created by Naomi Beutel, Jingyi Chen, Chandni Jaspal, Cameron Kennedy, Rebecca King, Rachel Lam, Jin Yong Lee, Lilly McLaughlin, Zamzam Mo'Allin, Samira Moalim-Yusuf, Autumn Nelsen, and Abby Popp.

SPATIAL HEARING

Learning Objectives

Understand why auditory localization is hard

Be able to describe the coordinate system for auditory scene analysis: azimuth, elevation and distance

Auditory localization is hard for many reasons. The human ear has to work really hard to make sense out of auditory information that is mixed in with many other sounds at the same time. It requires being able to determine where sound is coming from. This one task is extremely difficult, but also a crucial part of hearing. Without that, humans would likely make many mistakes about the auditory information coming in. There is a coordinate system that helps humans to do this, which includes azimuth, elevation, and distance.

Azimuth can be explained as the angle to the right or left of what is straight ahead, moving in an arc parallel to the ground. (The median plane is a vertical plane cutting through your head, perpendicular to “straight ahead”.) This helps to identify if sound is coming from the left or right side.

Elevation is the exact opposite of azimuth. It is the angle above or below the horizontal plane, which helps to identify if sound is coming from above or below.

Distance is the more obvious part of the system. It is how far or near the sound is to a person, which helps to determine the source of the sound as well.

The coordinate system above works together in order for humans to get the most information out of sound. But, another central part of spatial hearing is monaural and binaural hearing, which is one eared versus two eared hearing. This is a key component in being able to determine where sound is coming from. Hearing with one ear can be beneficial in identifying where sound is coming from because it tells someone which side of the body the sound is coming from. Hearing with two ears is beneficial as well because this can give one a sense of if the sound is coming from above, below, directly in front of, or behind the person.

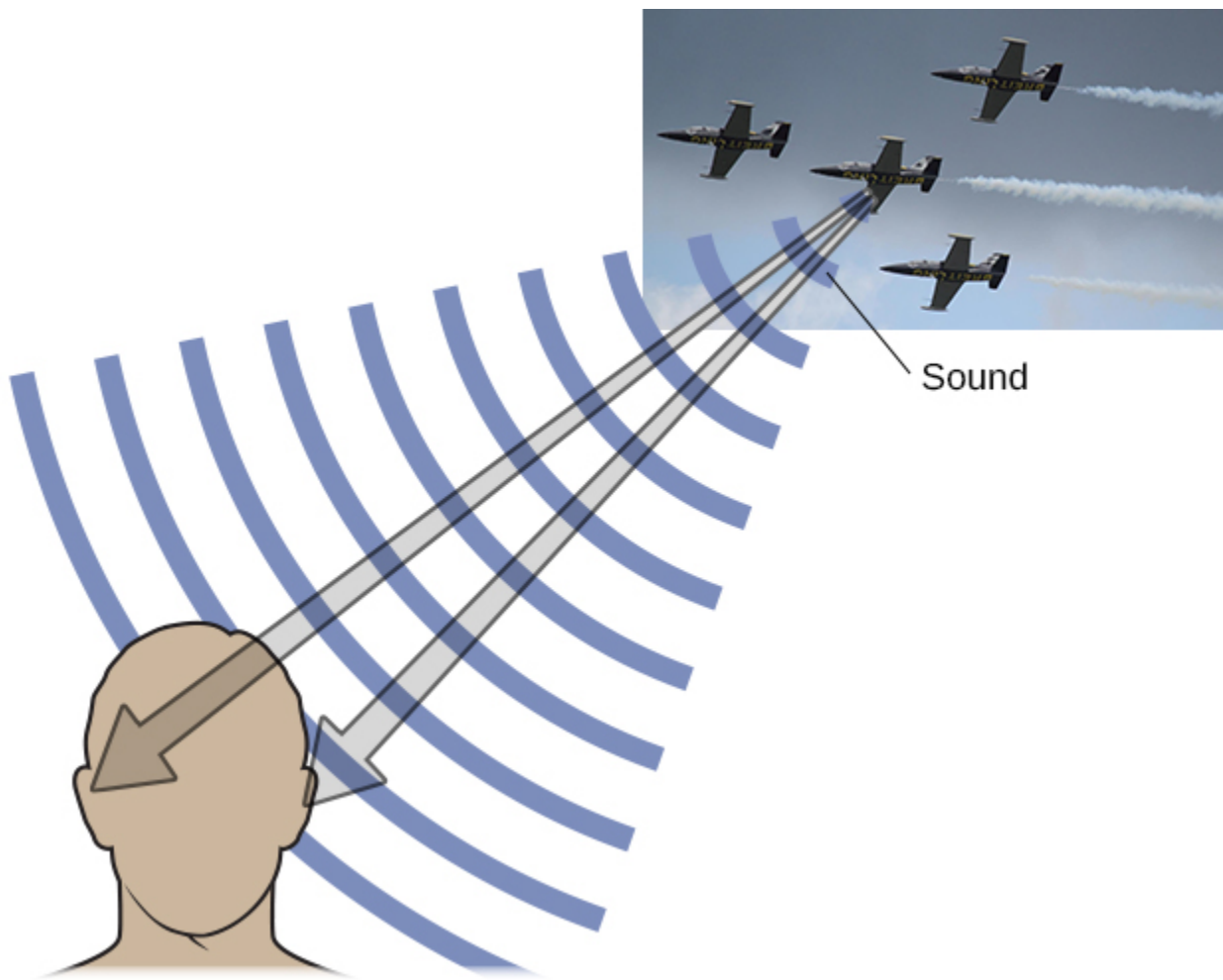


Fig 7.1.1. Sound localization. Localizing sound involves the use of both monaural and binaural cues. The image above demonstrates how the human ear determines where the source of sound is coming from by showing that the sound hits one ear first, giving the person the idea that the sound is above and to the left. (Provided by: OpenStax. License: CC BY 4.0. Modification of work by Max Pfandl)

Exercises

1. What part of the coordinate system helps discern if a sound is to the left, right or middle?
 - A. Azimuth
 - B. Elevation

- C. Horizontal
 - D. Distance
2. What part of the coordinate system helps discern if a sound is coming from above or below?
- A. Distance
 - B. Azimuth
 - C. Vertical
 - D. Elevation
3. What part of the coordinate system deciphers how far or near a sound is?
- A. Length
 - B. Elevation
 - C. Distance
 - D. Azimuth

Answer Key:

- 1. A
- 2. D
- 3. C

Authored by: Naomi Beutel

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69.

INTERAURAL TIME DIFFERENCE

Learning Objectives

Know interaural time difference (ITD) is a binaural cue and why

Understand neurons in which area are sensitive to ITD (the first place that signals from two ears meet)

Our ability to locate sound sources in space is an impressive feat of neural computation. The two main sources of information both come from a comparison of the sounds at the two ears. The first is based on interaural time differences (ITD) and relies on the fact that a sound source on the left will generate sound that will reach the left ear slightly before it reaches the right ear. Although sound is much slower than light, its speed still means that the time of arrival differences between the two ears is a fraction of a millisecond. The largest ITD we encounter in the real world (when sounds are directly to the left or right of us) are only a little over half a millisecond. With some practice, humans can learn to detect an ITD of between 10 and 20 μs (i.e., 20 millionths of a second) (Klump & Eady, 1956).

For the mathematically inclined: If the extra distance to the far ear is 23 cm (0.23 m) across, and sound is traveling at 340 m/s, this timing difference is at most $0.23/340 = 0.000676$ seconds or 0.676 milliseconds or 686 microseconds. That's tiny! The smallest difference the ears can notice is 10 microseconds!

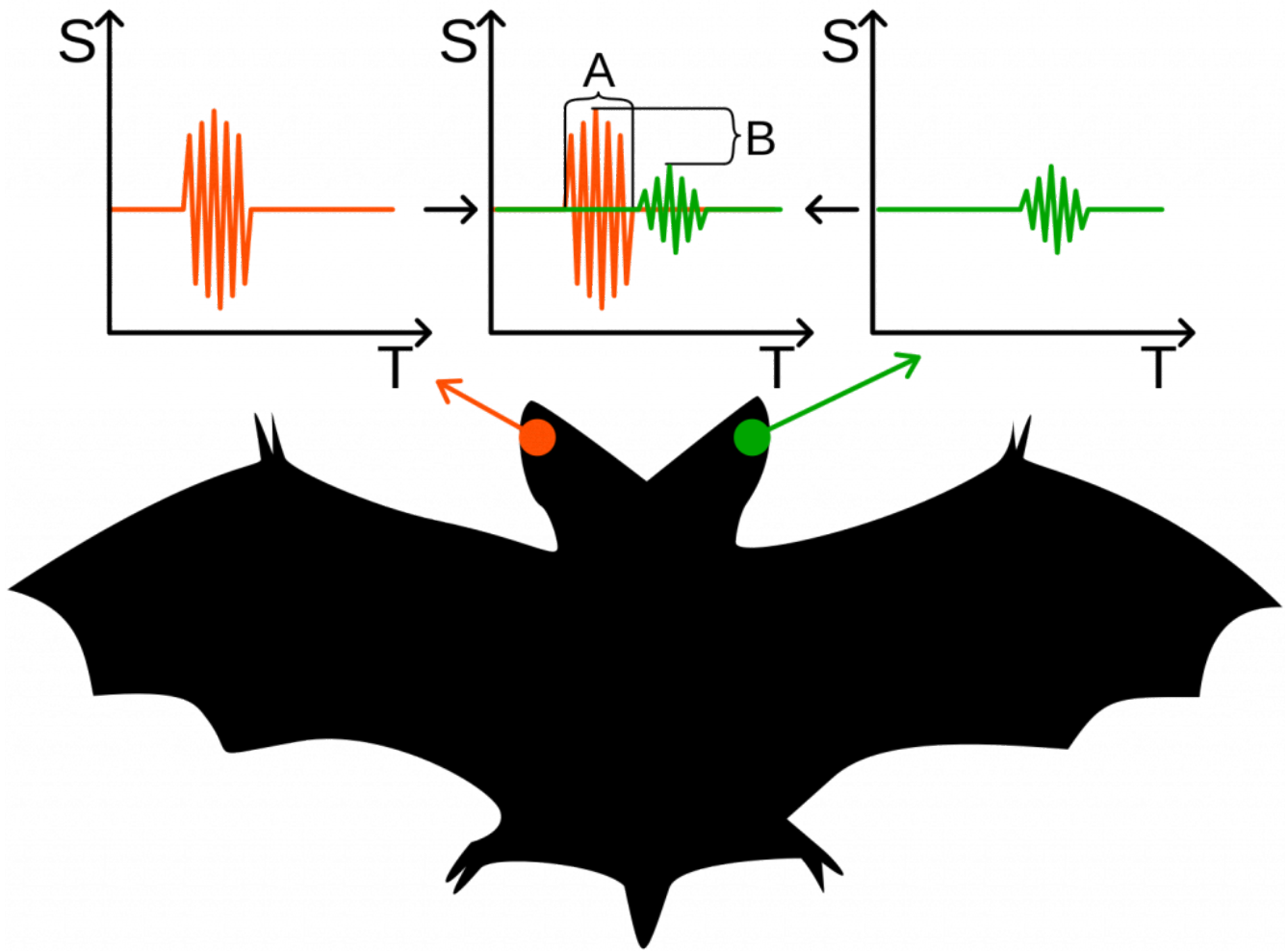


Fig. 7.2.1 Interaural time difference and interaural intensity level. This interaural time difference is the difference between the onset times or (A). The interaural level difference is indicated by (B). (credit: Shung and derivative work: Marek M. Provided by: Wikimedia Commons. License: PDM.)

When sound waves hit human ears, a signal is created by both ears independently, which is how the time difference is determined. These signals are sent into the brain and first meet in the superior olivary complex, which due to its purpose, is incredibly sensitive. Experimental Psychologist and acoustical scientist Lloyd A. Jeffress proposed a theory stating that there are different detectors for particular ITDs in the brain and each is excited maximally when triggered, which leads to the placement of sound. In 1988, the “coincidence counter circuit” predicted by Jeffress was experimentally discovered in the barn owl. However this specific map of coincidence detectors was undetectable in guinea pigs, leading to the conclusion that optimal coding strategies for the time differences depend on head size as well as frequency of sound. In further support of this hypothesis, it was experimentally determined that for humans, whose heads are large enough to homogeneously distributed coincidence detectors, also depends on frequency. If a sound is above 400 Hz the distribution is optimal, but if below 400 Hz, the use of distinct sub- populations are optimal (Harper. & McAlpine 2004).

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Adapted by: Joseph Hemenway

70.

INTERAURAL LEVEL DIFFERENCE

Learning Objectives

Know interaural level difference (ILD) is a binaural cue

Be able to explain why ILD is useful for high-frequency sounds only (frequency-dependent)

Understand the difference between ILD (higher frequency) and ITD (below about 1500Hz)

If a sound comes from an off-center location, it creates two types of binaural cues: interaural level differences and interaural timing differences. Interaural level difference refers to the fact that a sound coming from the right side of your body is more intense at your right ear than at your left ear, and vice versa, because of the attenuation of the sound wave as it passes through your head.

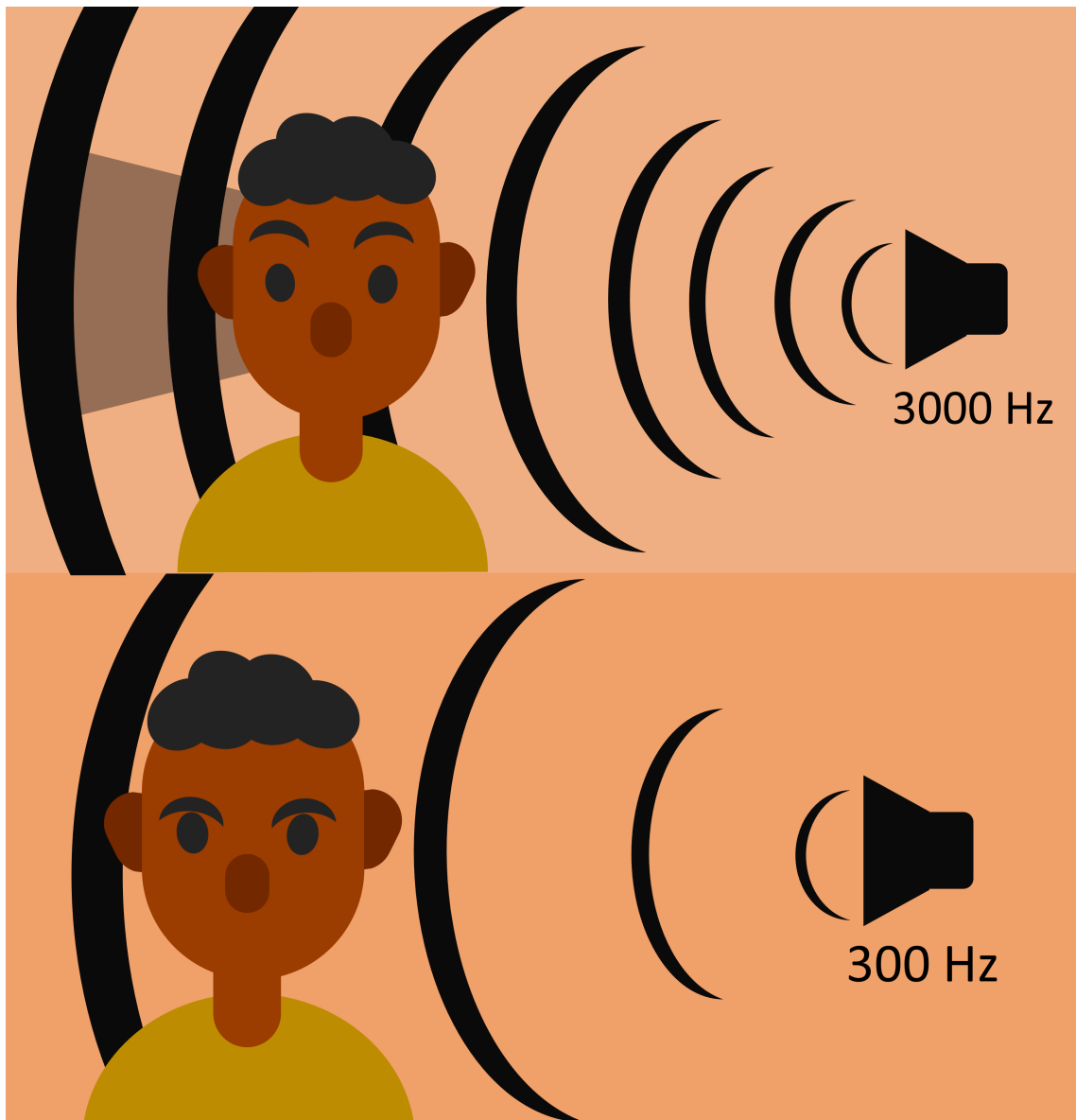


Fig.7.3.1. ILD. The interaural Level binaural cue only happens with high frequencies. In the image above, the person only experiences an acoustic shadow produced by this cue with a high frequency, but not a low one (<1000 Hz). (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

Interaural level difference (ILD) is a binaural cue for high-frequency sounds only. High frequency sounds have short wavelengths, so the head casts an acoustic shadow and sounds are quieter in the ear away from the sound. Below about 1000Hz, there is no ILD because the head is small compared to the wavelength of the air pressure perturbation...the sound sweeps on by without really noticing the head. ILDs can be as big as 20dB for some frequencies; they depend both on frequency and on the direction that sounds are coming from. ILDs are more useful at higher frequencies; ITDs stop being useful at about 1500Hz.

Interaural level difference refers to the fact that a sound coming from the right side of your body is more intense at your right ear than at your left ear because of the attenuation of the sound wave as it passes through

your head. Interaural timing difference refers to the small difference in the time at which a given sound wave arrives at each ear. Certain brain areas monitor these differences to construct where along a horizontal axis a sound originates.

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HEAD-RELATED TRANSFER FUNCTION

Learning Objectives

Know head-related transfer function (HRTF) is a monaural cue related to pinnae and why HRTF can provide information about elevation

Understand that people can learn new HRTF but still have their old HRTF

Know what cone of confusion is

The head-related transfer function (HRTF) is an auditory function that gives information about elevation. This function is monaural, meaning it takes place in each individual ear. By using information from both ears, one should be able to roughly identify the elevation of an external auditory cue. The speeds and intensities of the auditory cue are compared and contrasted at each ear, giving enough information for the brain to perceive the sound at the correct spot.

Each of us has a customized HRTF because of the shape of our pinnae: different frequencies are attenuated differently at different elevations. Thus, the HRTF provides information about elevation. It's a complicated function and one that we've learned after years of living in our heads. A study by Hofman et al. (1998) changed the shape of subjects pinnae and discovered:

1. Subjects lost the ability to determine the elevation of sounds when the first received some fake pinnae.
2. Subjects learned a new HRTF after a few weeks.
3. Subjects still had their old HRTF (were immediately accurately determining elevation) when the artificial pinnae were removed.

There's a thing called a cone of confusion, which is the cone-shaped region pointing out from the side of your head in which ITD and ILD are the same for all locations. This often leads to front/back confusions as well

as left-right confusions (although less often). Elevation information provided by the HRTF is needed to break this up. Head movements have also been shown to help break up the cone of confusion.

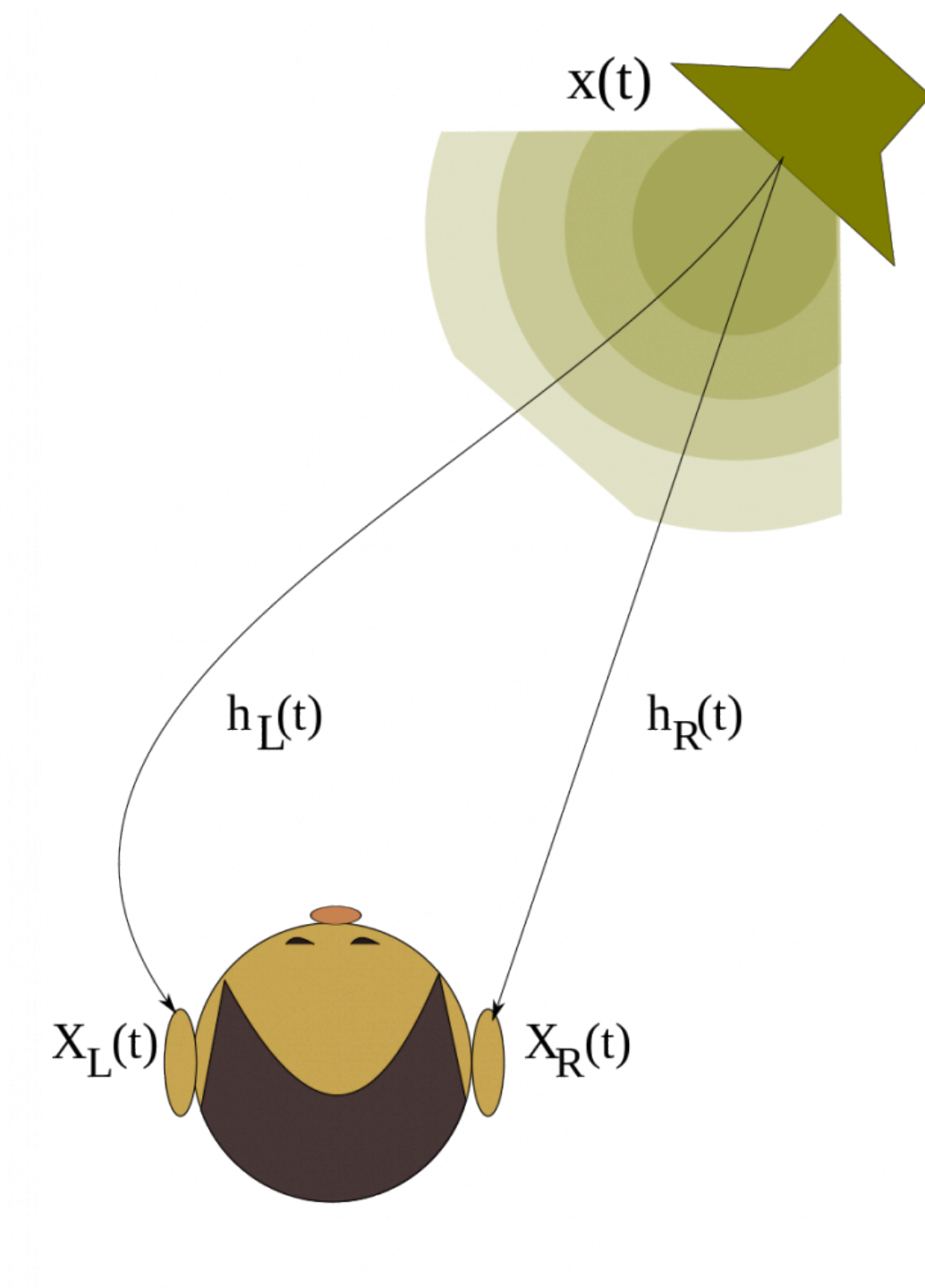


Fig 7.4.1. Each person has an individualized head-related transfer function, which helps with detection of elevation information. The above image shows how there are multiple factors that go into auditory perception. The height of the sound's location and the speed at which it is received at each ear are important factors. (Credit: Soumyasch. Provided by: Wikipedia. License: CC-BY 4.0)

Exercises

1. Which one of the five senses comes into play when we need to identify the elevation of an external auditory cue?
 - A. Hearing (Ears)
 - B. Seeing (Eyes)
 - C. Touch (Hands)
 - D. Taste (Tongue)
 - E. Smell (Nose)
2. True/False: We all have the same HRTF and HRTF provides information about why we lose our sense of smell as we age?
3. Which one of the following correctly defines cone of confusion?
 - A. Cone of confusion is what causes confusion after a concussion
 - B. Cone of confusion is the cone-shaped region pointing out from the side of your nose in which ITD and ILD are the same for all locations
 - C. Cone of confusion is the cone-shaped region pointing out from the side of your head in which ITD and ILD are the same for all locations

Answer Key:

1. A (Explanation: You need to use the information from both ears to evaluate external auditory cues.)
2. False (Explanation: We all have customized HRTF because of the shape of our pinnae. And HRTF provides information about elevation.)
3. C

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Adapted by: Cameron Kennedy & Kori Skrypek

INDOOR SPACES

Learning Objectives

Understand how echo can help us determine the layout of our environment

Be able to define the reverberation time

An **echo** is the reflection of sound from its original source. Like clapping in a hallway; the clap you hear after being the echo. It works similarly to sound localization that would be used to locate a plane in the air. But instead of experiencing the direct interaction between the sound's source and our ears, we utilize the reflection of the sound against surrounding materials and the delay this causes for the sound to reach us.

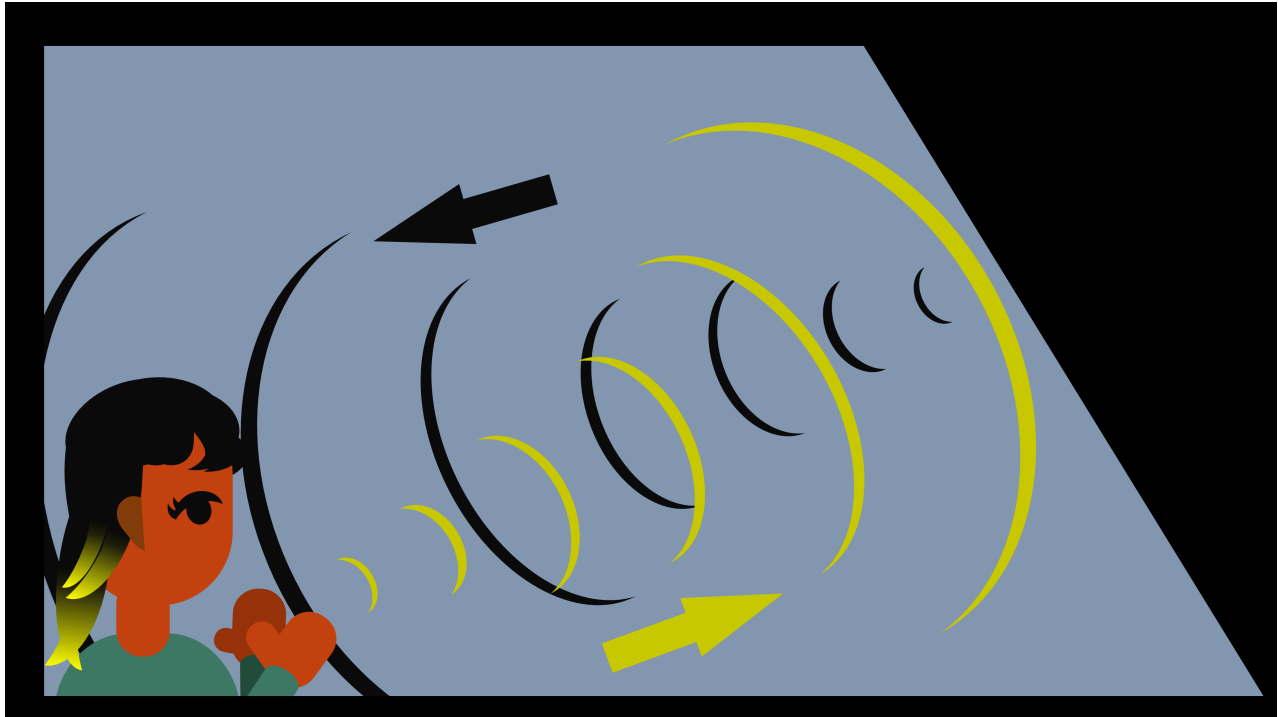


Fig. 7.5.1. Eco chamber. An echo occurs when sound reflects of an object from the original source. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

Echo also helps to determine the relative size of a space and the compositions of the materials within it. This perception is a result of **reverberation time**: the time it takes for a sound to decay 60 decibels from its original loudness. This is usually measured in terms of amplitude. Different materials can extend reverberation (ex. hardwood, metal) or muffle it (ex. foam, carpet) by absorbing more or less of the sound.

The sensation of echo and use of reverberation can be combined with monaural and binaural cues to create a more complex experiences of our surroundings through detailing sensory information, like hearing a myriad of musical notes in a concert that aren't actually being played individually.

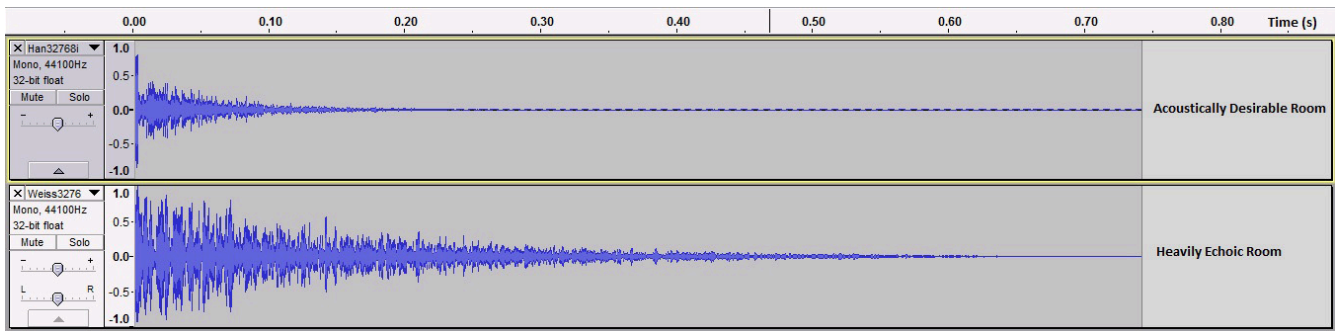


Fig 7.5.2. Reverberation time is longer in rooms with hard surfaces. The figure above shows the difference in reverberation time in two different rooms, one more prone to echo. This illustrates the difference between an echo and reverberation pretty well, essentially an echo is one reflection of sound and reverberation is many different reflections of sound. The more a room echoes the longer it will reverberate (which can also hint at the size of the room).

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Exercises

1. What does an echo help determine?
 - A. Relative sound of a space
 - B. Relative size of a space
 - C. Relative shadow of a space
 - D. Relative location of a space

Correct answer: B) relative size of a space

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DISTANCE PERCEPTION

Learning Objectives

Be able to describe what the direct-to-reverberant ratio is

Know how expectation let us understand distance

Imagine an angry customer at a retail store yelling that he can't find the swim shorts he wants in his size (the ones with the smiling sharks). You're annoyed, but you can tell that 28 year old is far away from you just by how loud he is to you. So you steer clear of the children's swim section. This is considered direct-to-reverberant. When we listen to someone that is the direct source, the reflection of the sound to us allows us to gain the location and distance of said object. The direct-to-reverberant sound is the ratio of a sound's reverberation time to when we hear it. The ratio also includes the amount of that reflected sound we are not picking up and is lost.

Our expectations about sounds helps us understand the distance of the sound we are hearing. For instance, we know a whisper is quiet, so if we were to hear the whisper loudly, we would know the distance of the sound is shorter and closer to us compared if a whisper was more quiet, it's further away from us. This goes the same with louder sounds as well. For example, we know that train noises are loud, so when we hear the train more quietly, we know the train or the source of the sound is much further away.



Fig. 7.6.1. We perceive things that are typically loud as far away when they sound quieter than we expect. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

Exercises

1. True or False: If someone is yelling from a far away distance, they can sound more quieter than we normally expect them to.
2. According to expectation, if the sound of someone whispering is loud, you expect them to be:
A. far away from you
B. close to you
3. True or False: Humans use sounds as cues for distance, the louder a sound is, the closer it is.

Answer Key:

1. True
2. B
3. True

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SOUND SEGREGATION

Learning Objectives

Be able to describe how to use primitive strategies to separate sounds (bottom-up strategies)

Be able to explain schema-based segregation strategies (top-down strategies)

Know the cocktail party problem

It is rare that we hear just one sound at a time. Usually, we're hearing several things — a bird chirping, a car driving by, a conversation on the sidewalk — and we have to separate them from each other in order to make sense of them. This is called sound segregation. We have several tools in order to do this.

Schema-based strategies are top-down. A schema is essentially a structure in our brain that holds and organizes the information that we have obtained growing up. Schema-based strategies are essentially using prior knowledge to locate and understand the distance of the sound. Primitive strategies, on the other hand, are bottom-up strategies: reflexive strategies that help us group sounds together based on similarity or location..

First, there is the primitive auditory stream segregation, where the brain groups the sound perceptually to form a consistent representation of the object from the sound it makes. A good example of this is when we hear an orchestra. As they play, we separate sounds with similar features (ex. the blare of trumpets) from non-like sounds (ex. the whisper of flutes). Grouping sounds by timbre like the trumpet/flue example is one primitive strategy. Other primitive strategies are grouping by pitch or grouping by location.



Fig.7.7.1. Listening to Music. We use Primary Auditory Stream Segregation, to identify the melody of a song by its individual notes. Our Schema-Based Analysis will tell us whose voice we hear and the exact instruments being used. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

The other process we can use to identify the information given from a mixture of sounds is the schema-based analysis. This is a top-down strategy in which the brain matches the sensory signal from the knowledge stored in the memory. An example of this is when you hear a noise at the park and recognize it as a birdsong, where your schema of what a particular bird sounds like helps you pick out the notes of that one birdsong from all the rest.

Even with the help of primitive strategies like grouping by similarity and schema-based strategies like recognizing familiar voices, we still have to work to focus on an interesting sound in a complex auditory environment. Imagine talking to someone in a loud environment and trying to hear what they are trying to say. We are able to hear the sound of interest (the other person voice) by focusing intently on it. Picking out a certain sound like in this example is known as the “cocktail party problem.” The cocktail party problem is a hard problem to solve and requires selective attention.

Exercises

1. The cocktail party problem, where an individual listens to a target sound more while in a noisy environment, works by:
 - A. making background noise less interesting to the individual listening
 - B. subconsciously increasing attention to a sound
 - C. making the individual instinctively leave a noisy environment so that they can listen more clearly
 - D. subconsciously tuning out background noise

Answer: B

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SPEECH PRODUCTION

Learning Objectives

Understand the separate roles of respiration, phonation, and articulation

Know the difference between a voiced and an unvoiced sound

The field of phonetics studies the sounds of human speech. When we study speech sounds we can consider them from two angles. Acoustic phonetics, in addition to being part of linguistics, is also a branch of physics. It's concerned with the physical, acoustic properties of the sound waves that we produce. We'll talk some about the acoustics of speech sounds, but we're primarily interested in articulatory phonetics, that is, how we humans use our bodies to produce speech sounds. Producing speech needs three mechanisms.

- The first is a source of energy. Anything that makes a sound needs a source of energy. For human speech sounds, the air flowing from our lungs provides energy.
- The second is a source of the sound: air flowing from the lungs arrives at the larynx. Put your hand on the front of your throat and gently feel the bony part under your skin. That's the front of your larynx. It's not actually made of bone; it's cartilage and muscle. This picture shows what the larynx looks like from the front:

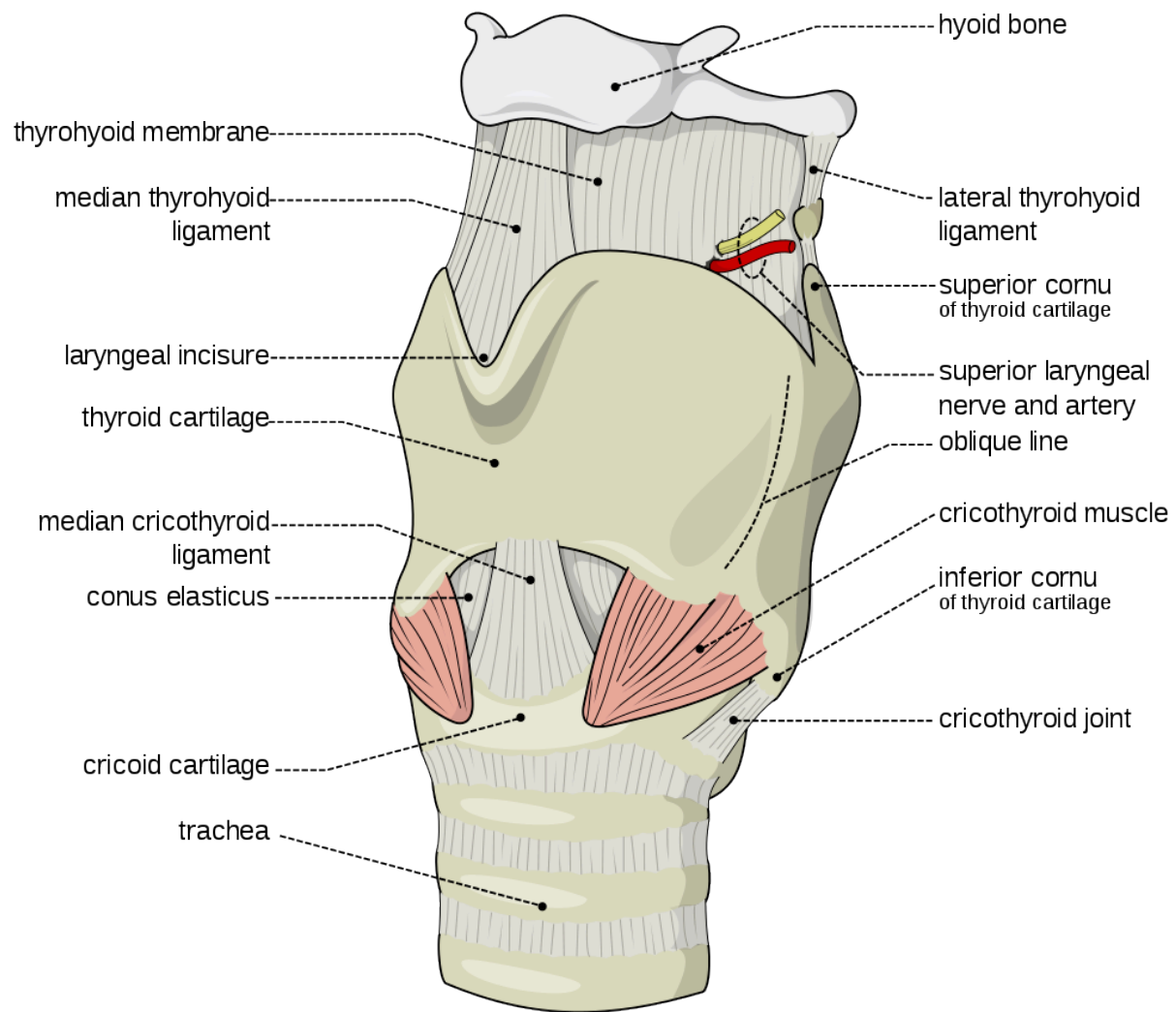


Fig 7.8.1 Front-view of Larynx. The larynx is also called the voice box and is where sound is produced when we speak. (Credit: Olek Remesz Provided by: Essentials of Linguistics. License: CC BY-SA 2.5-2.0-1.0)

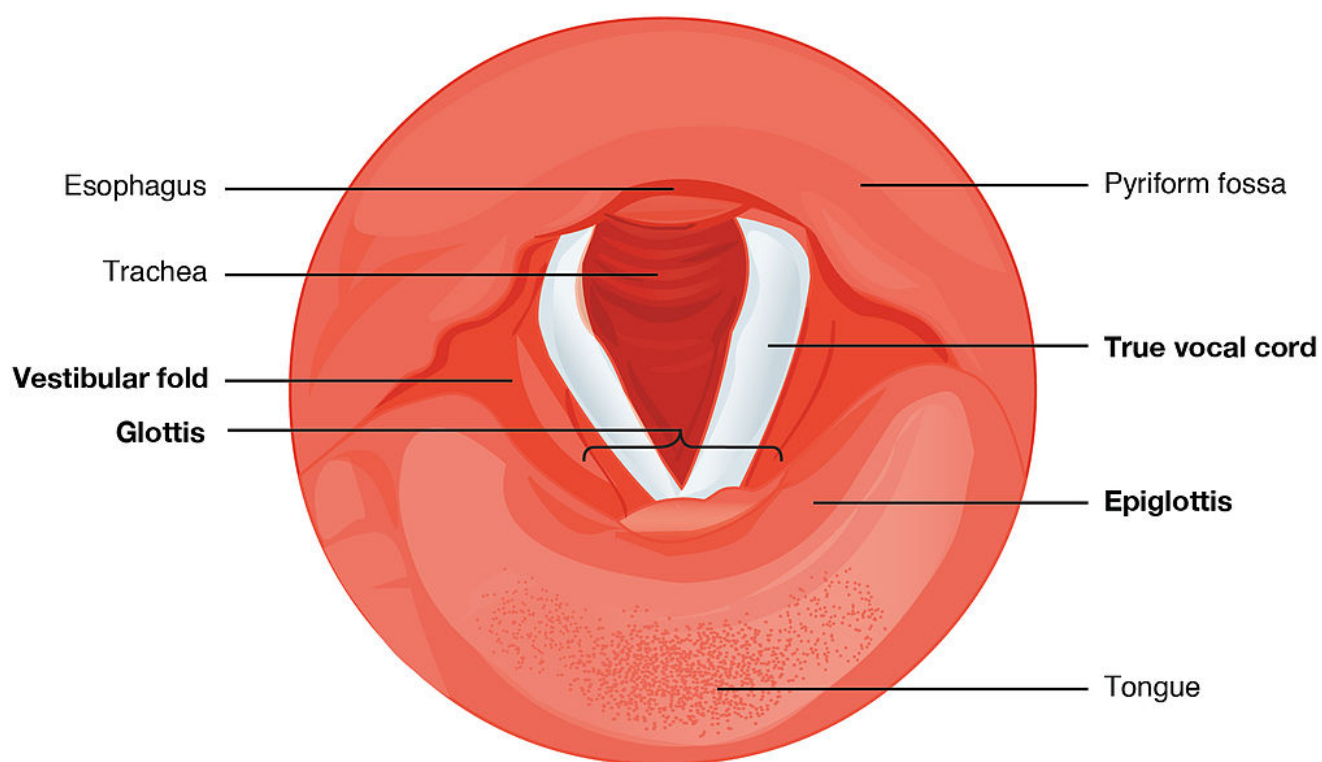


Fig 7.8.2 A view down a person's throat. The vocal cords vibrate to produce voiced sounds. (Provided by: OpenStax College. License: CC BY 3.0)

Larynx

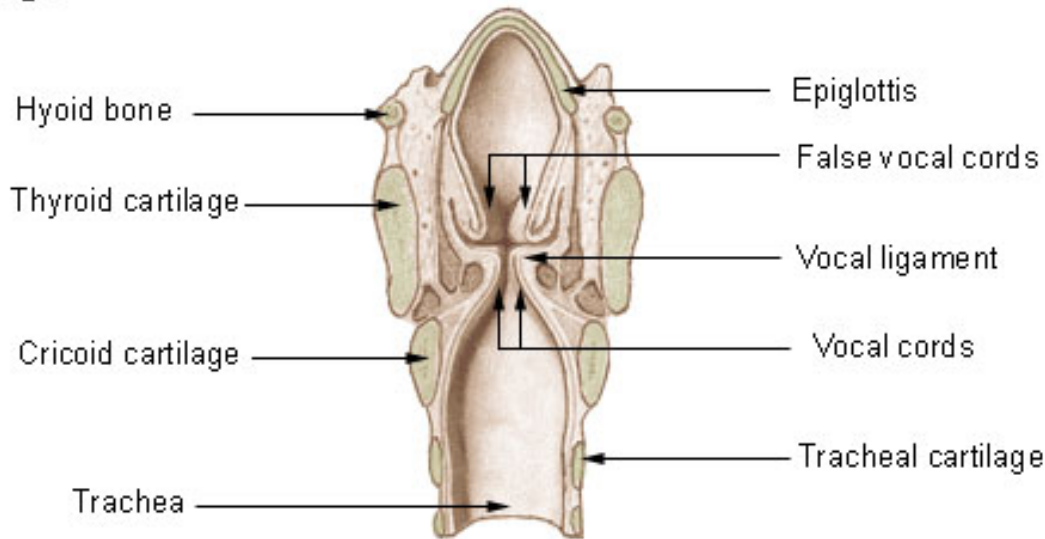


Fig. 7.8.3 Here is a cross section of the larynx in anatomical position. When humans speak, muscles in the larynx close the vocal cords. The air from lungs goes out through the Larynx, producing vibrations and sounds.

(Credit: Provided by: Wikipedia, License: Public Domain)

What you see here is that the opening of the larynx can be covered by two triangle-shaped pieces of skin. These are often called “vocal cords” but they’re not really like cords or strings. A better name for them is vocal folds. The opening between the vocal folds is called the glottis.

We can control our vocal folds to make a sound. I want you to try this out so take a moment and close your door or make sure there’s no one around that you might disturb:

First I want you to say the word uh-oh. Now say it again, but stop half-way through, Uh-. When you do that, you’ve closed your vocal folds by bringing them together. This stops the air flowing through your vocal tract. That little silence in the middle of uh-oh is called a glottal stop because the air is stopped completely when the vocal folds close off the glottis. Now I want you to open your mouth and breathe out quietly, haaaaaaah. When you do this, your vocal folds are open and the air is passing freely through the glottis. Now breathe out again and say aaah, as if the doctor is looking down your throat. To make that aaaah sound, you’re holding your vocal folds close together and vibrating them rapidly. When we speak, we make some sounds with vocal folds open, and some with vocal folds vibrating. Put your hand on the front of your larynx again and make a long SSSSS sound. Now switch and make a ZZZZZ sound. You can feel your larynx vibrate on ZZZZZ but not on SSSSS. That’s because [s] is a voiceless sound, made with the vocal folds held open, and [z] is a voiced sound, where we vibrate the vocal folds. Do it again and feel the difference between voiced and voiceless. Now take your hand off your larynx and plug your ears and make the two sounds again with your ears plugged. You can hear the difference between voiceless and voiced sounds inside your head.

There are three crucial mechanisms involved in producing speech, and so far we've looked at only two (listed as the first two below):

- Energy comes from the air supplied by the lungs.
- The vocal folds produce sound at the larynx.
- The sound is then filtered, or shaped, by the articulators.

The oral cavity is the space in your mouth. The nasal cavity, obviously, is the space inside and behind your nose. And of course, we use our tongues, lips, teeth and jaws to articulate speech as well. In the next unit, we'll look in more detail at how we use our articulators.

So to sum up, the three mechanisms that we use to produce speech are:

- respiration at the lungs,
- phonation at the larynx
- articulation in the mouth.

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Essentials of Linguistics, Chapter 2.1 How Humans Produce Speech by Catherine Anderson

URL: <https://essentialsoflinguistics.pressbooks.com/chapter/2-2-how-humans-produce-speech/>

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Adapted by: Lilly McLaughlin

76.

SPECTROGRAMS

Learning Objectives

Be able to describe what spectrogram (time-frequency plot) is

Know the definition of formants and transitions

Time-frequency analysis shows up a lot. For speech, when we visualize the evolving frequency composition of sounds, it is called a spectrogram. Spectrograms are used extensively in the fields of music, linguistics, sonar, radar, speech processing, seismology, and others. Spectrograms of audio can be used to identify spoken words phonetically, and to analyse the various calls of animals. For music, this is the graphic equalizer on a stereo. On spectrograms (time-frequency plots) of speech, there are bands of power at different frequencies. These are formants. As the speaker changes the sound it is making, the power bands swoop up and down. These are transitions.

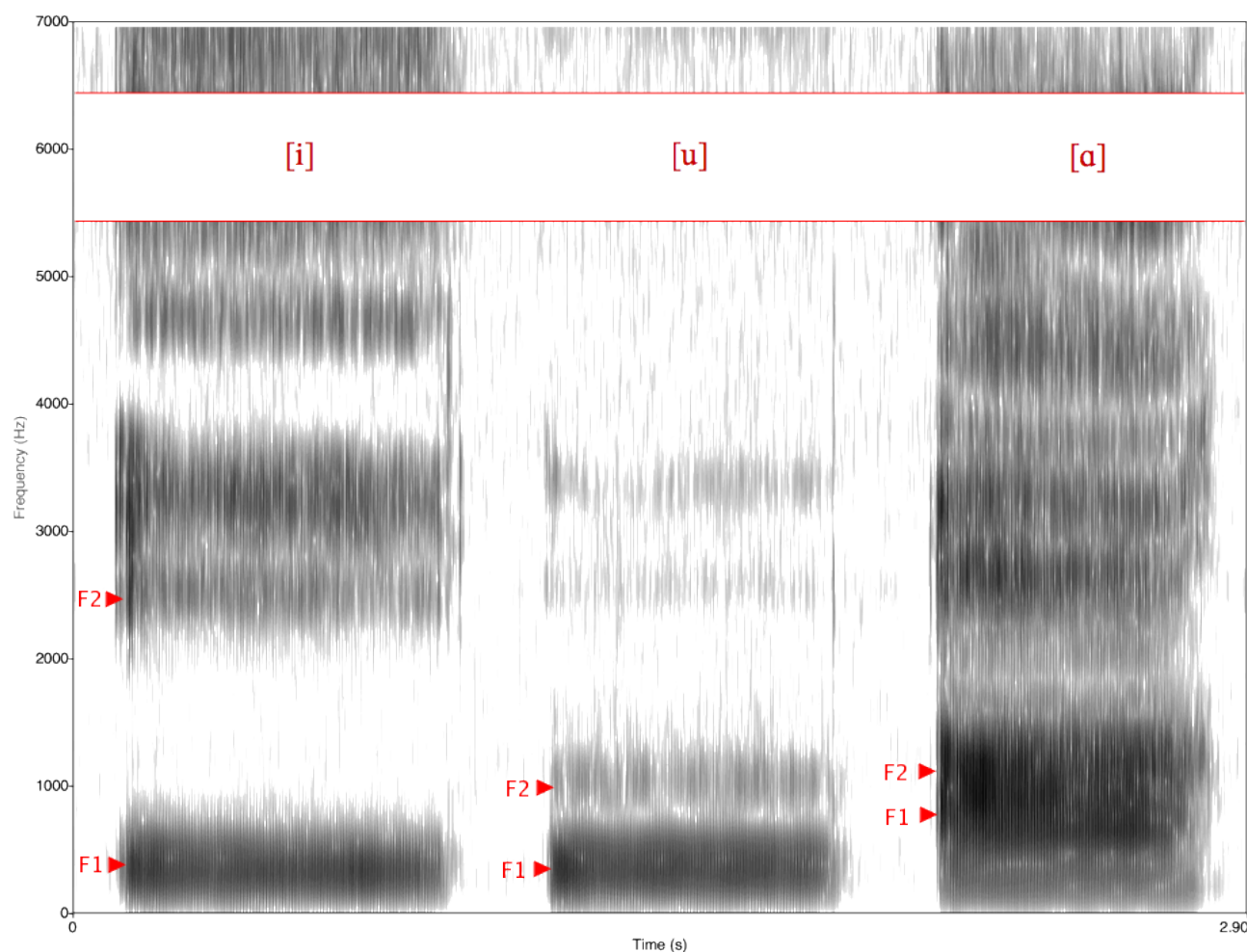


Fig 7.9.1 Spectrogram showing the vowels [i], [u] and [a]. By analyzing the frequency content of voice sounds, as a function of time, we can see different bands of power (formants) that define different vowels. (Provided by: Wikimedia Commons. License: CC BY 2.0)

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CATEGORICAL PERCEPTION

Learning Objectives

Understand what the phoneme is and changing a phoneme will change the meaning of a word

Be able to explain categorical perception with examples

Know learning language means we learn to create phonetic boundaries and hear phonemes

Phonemes are distinct units of sound that you put together to create a word. When you change a **phoneme** in a syllable or a word, you change the meaning of the word. We're used to think of words as things made out of letters and syllables. A phoneme is not necessarily either of those. The phoneme is the sound we make; sometimes there are several phonemes in a single syllable, like the word "tie" — it's one syllable, but it has 3 phonemes /taɪ/. You make the /t/ when your tongue comes up against your teeth, you make the /a/ as you lower your jaw and make space between the tongue and the roof of your mouth, and you make the /ɪ/ (sounds like "ee") by changing the position of your tongue again. Below is a chart of consonants that English speakers use. Check out <https://ipa.typeit.org/> if you want a tool that will let you type phonemes you can't find on the keyboard.

24 Consonant Phonemes / Graphemes

Phoneme (sound)	Examples	Graphemes (written patterns)		Phoneme (sound)	Examples	Graphemes (written patterns)	
/b/	ban <u>ana</u> , bu <u>bb</u> les	b	bb	/s/	<u>s</u> un, mou <u>se</u>	s	ss, ce, se, c, sc
/c/	<u>c</u> ar, du <u>ck</u>	c	k, ck, q, ch	/t/	<u>t</u> urtle, litt <u>le</u>	t	tt
/d/	<u>d</u> inosaur, pudd <u>le</u>	d	dd,	/v/	<u>v</u> olcano, hal <u>ve</u>	v	ve
/f/	<u>f</u> ish, giraff <u>e</u>	f	ff, ph, gh	/w/	<u>w</u> atch, que <u>en</u>	w	wh, u
/g/	<u>g</u> uitar, gogg <u>le</u> s	g	gg,	/x/	fo <u>x</u>	x	
/h/	<u>h</u> elicopter	h		/y/	yo-yo	y	
/j/	<u>j</u> ellyfish, fridg <u>e</u>	j	g, dge, ge	/z/	<u>z</u> ip, pleas <u>e</u>	z	zz, ze, s, se
/l/	lea <u>f</u> , be <u>ll</u>	l	ll, le	/sh/	sh <u>oe</u> s, televi <u>si</u> on	sh, ch, si, ti	
/m/	<u>m</u> onkey, ham <u>me</u> r	m	mm, mb	/ch/	<u>ch</u> ildren, stit <u>ch</u>	ch, tch	
/n/	<u>n</u> ail, kn <u>o</u> t	n	nn, kn	/th/	moth <u>er</u>	th	
/p/	<u>p</u> umpkin, pup <u>pe</u> t	p	pp	/th/	<u>th</u> ong	th	
/r/	rai <u>n</u> , <u>w</u> rite	r	rr, wr	/ng/	sin <u>g</u> , an <u>kle</u>	ng, n	

 Regular Alphabet Letters and Sounds

 Advanced Letter Patterns and Sounds

[@K-3TeacherResources.com](https://www.k-3teacherresources.com)

Fig. 7.10.1 Every language creates units of sound called phonemes to communicate. Here is a chart of english consonant phonemes . Different languages draw boundaries at different locations (one example being Korean, which recognizes a third phoneme between /b/ and /pa/ that English speakers don't hear). (Credit: attanatta, provided by: flicker, license: CC BY 2.0.)

When you're around somebody who speaks a different language than you, you obviously can't understand what is being said. However did you know that you actually hear different sounds than what the native speaker hears? As a bilingual person, it's hard for me to distinguish different sounds such as "Th" and "Ph" sounds, as native English speakers can easily determine the difference, Somali speakers can't; these sounds appear the same. This happens because of a brain trick called categorical perception. Categorical perception is a phenomenon indicating that certain stimuli (especially speech) are perceived categorically rather than continuously despite a continuous variation in their form. This occurs when items that range along a continuum are perceived as being either more or less similar to each other than they really are because of the way they are classified. A classic example shortens the VOT (voice onset time) to change the /t/ sound in /tar/ ("die") to a /d/ ("die"). Even though the VOT is changed continuously across a wide range, the listener perceives only two different sounds: "die" on one side of the phonetic boundary and "tie" on the other side. Since nothing in between would make sense, we don't hear it!

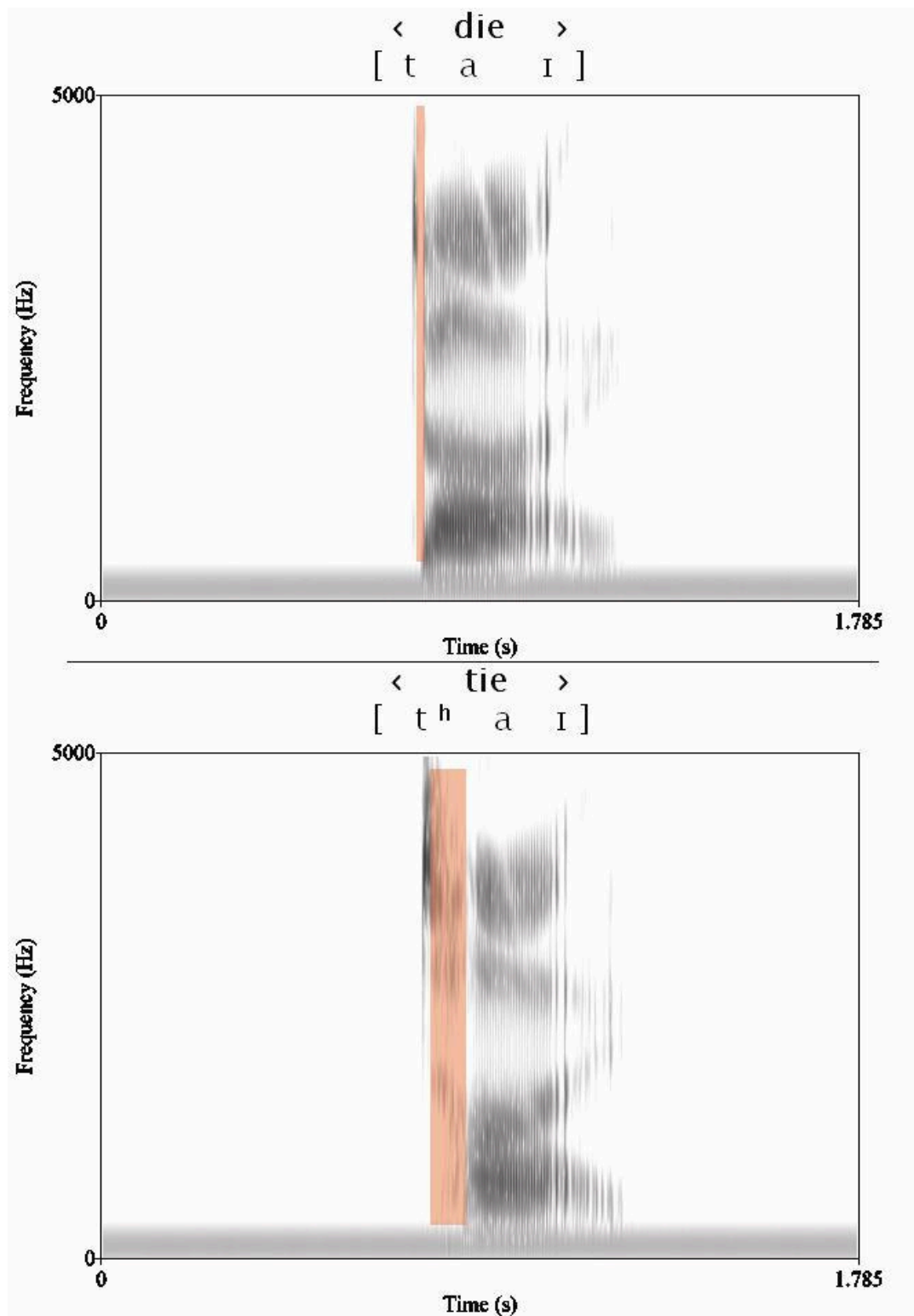


Fig 7.10.2 Voice onset time. The difference in the phonemes of “die” and “tie” shown on a spectrogram. (Provided by: Wikimedia Commons. License: PDM)

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Adapted by: Samira Moalim-YusufOpenStax, Speech Perception

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URL : <https://cnx.org/contents/bwyYFEPa@10/Speech-Perception>

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UNDERSTANDING SPEECH

Learning Objectives

Be able to describe why speech is hard to understand: the segmentation problem, co-articulation problem, speaker problem

Know what the McGurk effect is

Difficulty in segmenting phonemes — where does one stop and another start? Anyone who has learned a second language knows how hard it is to know where one word stops and another starts! People who study speech call this the segmentation problem.

Variability due to co-articulation — phonemes look/sound slightly different in different contexts. In a spectrogram, phonemes are characterized by formants (bands of constant sound during vowels) and transitions (onsets and offsets of formants, usually associated with consonants). A single phoneme will look very different on a spectrogram, depending on what syllable, word or phrase it is part of.

Variation in speaker styles — we all speak at different speeds, slur things together, etc. Human listeners employ a lot of social and contextual cues (e.g., visual cues) to figure out what people are saying. Computers do not have access to this information.

The McGurk effect shows how we use visual cues (cue combination: auditory plus visual cues) to ignore the variability and figure out which phoneme is which. In other words, what we see affects what we hear.

To learn more about the McGurk Effect, watch the video linked here and included below.



One or more interactive elements has been excluded from this version of the text. You can view them online here: <https://pressbooks.umn.edu/sensationandperception/?p=432#oembed-1>

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LANGUAGE CORTEX

Learning Objectives

Know aphasia is a loss of language skills due to cortical damage

Be able to distinguish 3 primary kinds of aphasia: expressive, receptive and conduction aphasia

Distinguish functions of the Wernicke's area and Broca's area in speech perception and reading

Aphasia is an inability to comprehend and formulate language because of damage to a specific brain region. The main causes of aphasia is a cerebral vascular accident (stroke) or any sort of head trauma. However aphasia can also be a result of brain tumors, brain infections, or a neurodegenerative disease. There are three primary kinds of aphasia, which include: expressive, receptive and conduction aphasia.

Expressive aphasia, also known as Broca's aphasia, is characterized by the partial or full loss of ability to produce language. This includes spoken, manual, or written language. Although expressive language can be extremely difficult, the ability to understand and comprehend language remains intact. Individuals with this type of aphasia usually will be able to read, however, since the ability to produce language is impaired, writing is usually limited because of the brain's inability to produce language in the form of writing.

Receptive aphasia, also known as Wernicke's aphasia, is characterized in which individuals have difficulty understanding written and spoken language. However, other intellectual abilities are completely fine. This type of aphasia also causes an individual's speech to be hard to understand. What this means is they can speak in correct grammatical and syntactical sentences, but it doesn't make a lot of sense. Because Wernicke's aphasia affects the brain's ability to make sense of words, reading and writing are often severely impaired.

Conduction aphasia is characterized by impairment with repetition. These individuals are able to express themselves relatively well, with only some word-finding issues, but have extreme difficulty repeating phrases, especially when they increase in length and complexity. This type of aphasia is much more rare than the other two types of aphasia listed above.

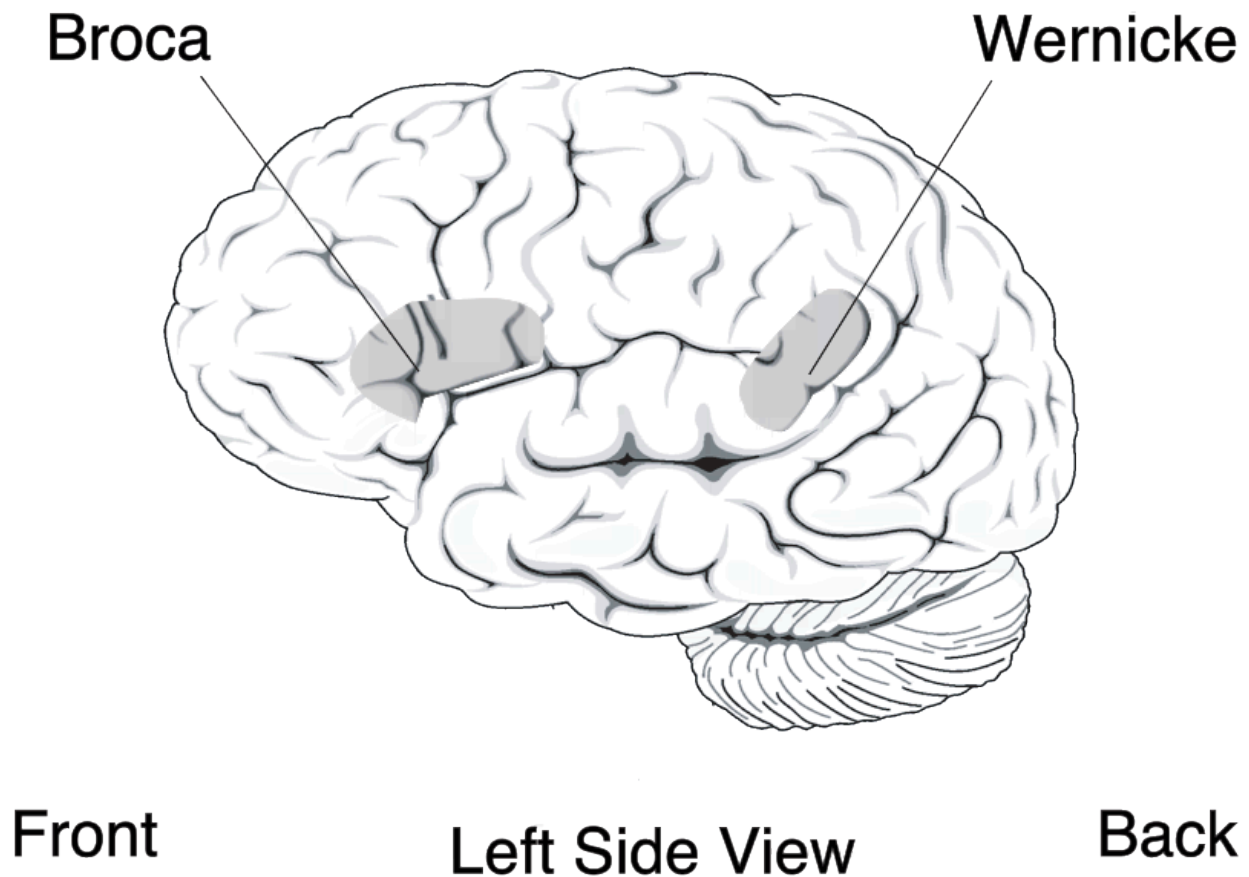


Fig 7.12.1 Regions involved in speech recognition and production. The figure above shows where Broca's and Wernicke's areas are located and where damage would be localized. (Provided by: Wikimedia Commons, License: PDM)

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Wikipedia, Aphasia

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Expressive_aphasia#:~:text=Expressive%20aphasia%2C%20also%20known%20as,aphasia%20will%20exhibit%20effortful%20speech.

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80.

ACTIVE LEARNING EXERCISES - SPEECH



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SECTION VIII

LIGHT AND EYEBALLS

As with hearing, we'll start out the section on visual perception with discussions of the physics of light and the anatomy of the eyeballs. In this chapter, we will be discussing the basics of our visual perception from the physics of light, the path of light through our eyes, to how the light is transcribed in our retina. We will also learn about the complexity of our retina regarding the retinal network, different retinal cells, and their functions.

This chapter was created by Nura Ahmed, Ran Rice, Elena Shest, Jeeho Sohn, Kathryn Taterka, Joyce Thao, Sebastian Vile, Sarah Warden, Savannah Whisenhunt, Dheeraj Uppal, and Yeemeng Yang.

81.

PHYSICS OF LIGHT

Learning Objectives

Being able to describe the basic properties of light

Know what factors determine the color of an object

Basic Properties of Light

We define light as the electromagnetic rays that interact with the photoreceptors in our eyes. A honeybee would have a different definition of light, because it can see ultraviolet rays (200-400nm), where we just think of UV as a sunburn hazard.

Light is a form of the electromagnetic radiation. Electromagnetic radiation is sinusoidal electric and magnetic fields that are oriented at right angles (orthogonal) to each other, out of phase (one gets large while the other gets small, then they trade off), and can propagate through a vacuum. Like sound, all electromagnetic radiation travels at the same speed (300 million meters per second in a vacuum), and $\text{speed} = \text{wavelength} \times \text{frequency}$. These waves come in a wide variety of wavelengths called the electromagnetic spectrum. The portion of the electromagnetic spectrum that we call light is the portion that has wavelengths between 400 and 700 nanometers.

A property of light is that it is quantized which means it can only take on specific values. Even though light propagates as a continuous dance between electric and magnetic fields, the energy is quantized – a photon is the smallest amount of light that can be generated or transmitted.

Factors that Determine Color

Light falling on the retina causes chemical changes to pigment molecules in the photoreceptors, ultimately leading to a change in the activity of the RGCs (retinal ganglion cells). Photoreceptor cells have two parts, the

inner segment and the outer segment. The inner segment contains the nucleus and other common organelles of a cell, whereas the outer segment is a specialized region in which photoreception takes place.

There are two types of photoreceptors—rods and cones—which differ in the shape of their outer segment. Another difference between rods and cones is the light levels. Rods are in control of vision at night and cones take the role of vision during the day. Not only that but, rods have lower visual sharpness and cones have higher visual sharpness. This is because rods have a sensitivity to light intensity that is higher compared to cones. In addition to that, there are three cone photopigments, called opsins, which are each sensitive to a particular wavelength of light. The wavelength of visible light determines its color. The pigments in human eyes are specialized in perceiving three different primary colors: red, green, and blue. The color of an object is determined by the wavelength that the object reflects and the color of the light source.

outer segments are short, tapered shapes with folds of membrane in place of the discs in the rods. (b) Tissue of the retina shows a dense layer of nuclei of the rods and cones. (Provided by: OpenStax License: CC-BY 4.0.)

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Schwiegerling, J. (2004). Field Guide to Visual and Ophthalmic Optics, SPIE Press, Bellingham WA.

Retrieved from: https://spie.org/publications/fg04_p04_photoreceptors?SSO=1

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82.

EYEBALL ANATOMY

Learning Objectives

- Being able to describe different parts of a human eyeball
- Know which parts of the eyeball are involved in accommodation
- Know what's a blind spot on the retina, and where it's located.

Structure of the Eye

The Cornea is the transparent but alive curved structure at the front of the eye, with embedded nerve endings (pain, touch and thermal sensation). It has 80% of the focusing power of the eye but is not flexible. The Sclera is the white, hard outside of the eyeball. The aqueous humor is the low viscosity fluid behind the cornea, in front of the lens. The Iris is the muscular, colored tissue that surrounds (and shapes) the pupil. The Pupil is the hole through which light enters the eye. The Lens is the flexible, clear substance that provides 20% of the focusing power of the eye. The Ciliary (or lens) muscles are the muscles that control the shape (curvature) of the lens. The Vitreous humor is the high viscosity fluid filling the eyeball. The Retina is the sheet of neurons at the back of the eye. The Fovea is where light from the center of gaze lands on the retina. The Pigment epithelium is the black layer behind the retina where visual pigments are replenished.

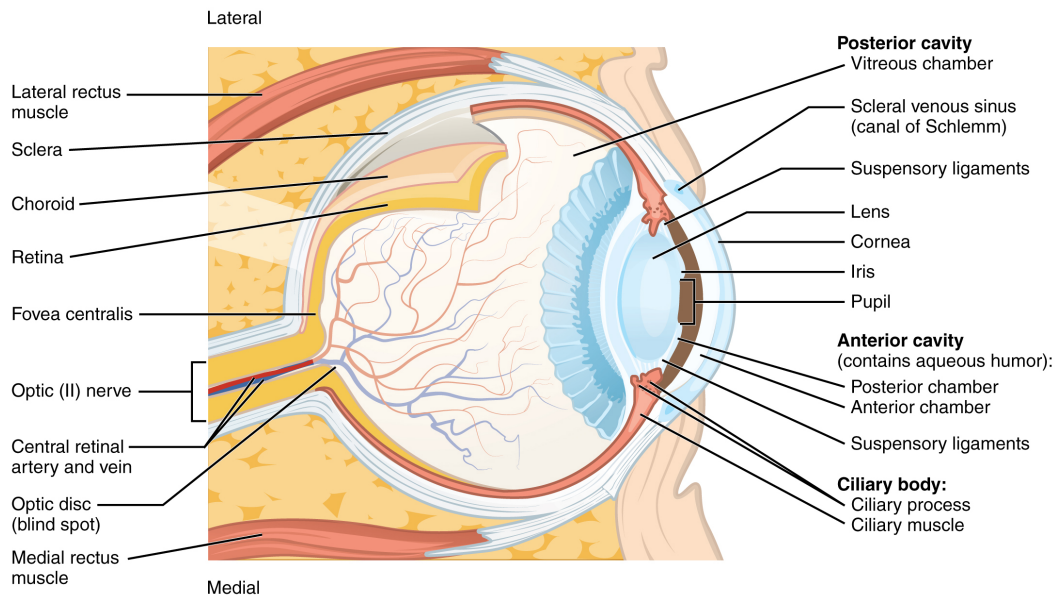


Fig.8.2.1. The Structure of the Eye The sphere of the eye can be divided into anterior and posterior chambers. The wall of the eye is composed of three layers: the fibrous tunic, vascular tunic, and neural tunic. Within the neural tunic is the retina, with three layers of cells and two synaptic layers in between. The center of the retina has a small indentation known as the fovea. (Provided by: OpenStax. License: CC-BY 4.0)

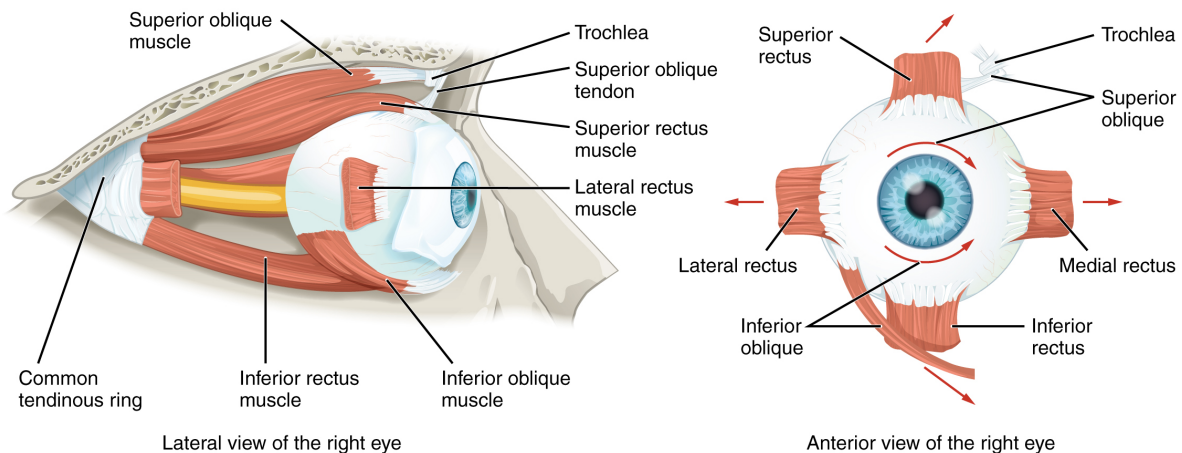


Fig.8.2.2. Extraocular Muscles. In this diagram we see the different muscles in place around the eye that help move the eye within the orbit. (Provided by: OpenStax . License: CC-BY 4.0)

20% of the focusing power of the eye is in the lens. Although it is only 20% of the focusing power it is important because it is flexible and gives us the ability to accommodate. Accommodation is the ability to focus on things that are near or far away. Presbyopia is when 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. Cataracts are the crystallization of the lens that scatters light, making it hard to perceive detail.

The Blind spot is located on the retina where there are no photoreceptors (because axons heading for the optic nerve occupy that space). Your brain fills in the hole with a copy of what's around the blind spot. The axons that pass through the retina where the optic nerve begins and where there are no photoreceptors at the very back of the eye. This is what creates a "blind spot" in the retina, and a corresponding blind spot in our visual field.

Exercises



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Adapted by: Ran Rice

83.

NEAR- AND FAR-SIGHTED EYES

Learning Objectives

Know what are the causes of myopia and hyperopia

Being able to describe how the focus point is affected by myopia and hyperopia

Know what are the current treatments for myopia and hyperopia (what kinds of glasses, surgery)

Causes of Myopia and Hyperopia

Myopia (near-sightedness) is characterized by blurring of objects viewed at a distance, and is commonly the result of abnormal elongation of the eyeball – which causes the refractive image formed by the cornea and the lens to fall in front of the photoreceptors of the retina. It occurs when the axial length of the eye is too long, and light is focused in front of the photoreceptors. Hyperopia (far-sightedness) occurs when the axial length of the eye is too short, and light is focused behind the photoreceptors. This makes close-by objects appear blurry.

Effects on the Focus Point

The cornea determines whether you are near-sighted or far-sighted because it has 80% of the focusing power of the eye and is not flexible. If the cornea focuses too fast or the eyeball is too long, you are near-sighted (myopic). If the cornea does not focus strongly enough, light from near objects focuses behind the retina and is blurry, meaning you are far-sighted (hyperopic).

Treatment Options

There are multiple ways to treat both myopia and hyperopia. In the case of myopia, affixing a negative (concave or diverging) lens over the eye pushes the focal place of the visual image behind the retina inducing an increase in axial elongation and a more myopic refraction. With hyperopia, a positive (convex or converging) lens over

the eye pushes the focal plane of the visual image in front of the retina, inducing a decrease in axial elongation and a more hyperopic refraction. Besides glasses and contact lenses, another option is surgery. LASIK and LASEK can reshape the cornea to eliminate the need for corrective lenses for near-sightedness, far-sightedness, and other issues.

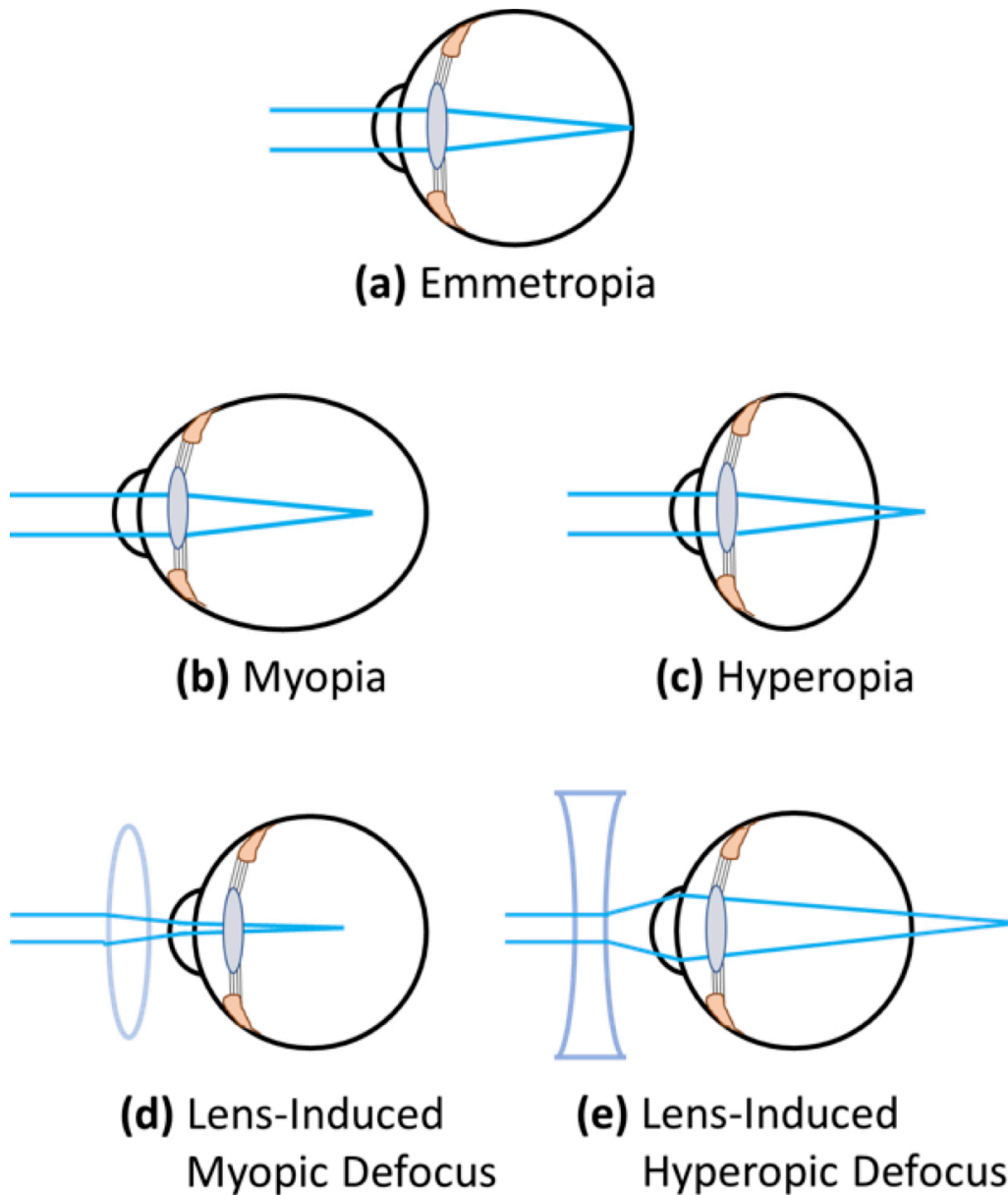


Fig.8.3.1. Exaggerated representation of simple refractive errors caused by abnormal eye growth. The figures above give a visual representation of how the focal point in both myopic and hyperopic eyes are shifted and how correction lenses work to adjust the eye. (Provided by: Webvision. License: CC-BY-NC 4.0)

Exercises

1. ____ is characterized by blurring of objects viewed at a distance
 - A. Myopia (near-sightedness)
 - B. Myopia (far-sightedness)
 - C. Hyperopia (far-sightedness)
 - D. Hyperopia (near-sightedness)
2. The ____ determines whether you are near-sighted or far-sighted because it has 80% of the focusing power of the eye and is not flexible.
 - A. iris
 - B. cornea
 - C. lens
 - D. sclera
3. Which of the following can NOT treat Myopia/Hyperopia
 - A. LASIK
 - B. LASEK
 - C. Contact lenses
 - D. Blue-Light glasses

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Adapted by: Elena ShestWebvision: The Organization of the Retina and Visual System, The Science Behind Myopia by Brittany J. Carr and William K. Stell

URL: <https://webvision.med.utah.edu/book/part-xvii-refractive-errors/the-science-behind-myopia-by-brittany-j-carr-and-william-k-stell/>

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PRESBYOPIA

Learning Objectives

Know what's the cause of presbyopia

Know what are the current treatment options for presbyopia

Being able to describe another common eye problem that comes with age

Presbyopia is a condition associated with the aging of the eye that results in progressively worsening ability to focus clearly on close objects.[1] Symptoms include difficulty reading small print, having to hold reading material farther away, headaches , and eyestrain .[1] Different people will have different degrees of problems.[1] Other types of refractive errors may exist at the same time as presbyopia.[1] Presbyopia is a normal part of the aging process.[1] It occurs due to hardening of the lens of the eye, causing the eye to focus light behind rather than on the retina when looking at close objects.[1] It is a type of refractive error along with nearsightedness, farsightedness, and astigmatism.[1] Diagnosis is by an eye examination.[1] Treatment is typically with eyeglasses.[1] The eyeglasses used have higher focusing power in the lower portion of the lens.[1] Off the shelf reading glasses may be sufficient for some.[1] Contact lenses may also occasionally be used.[2]

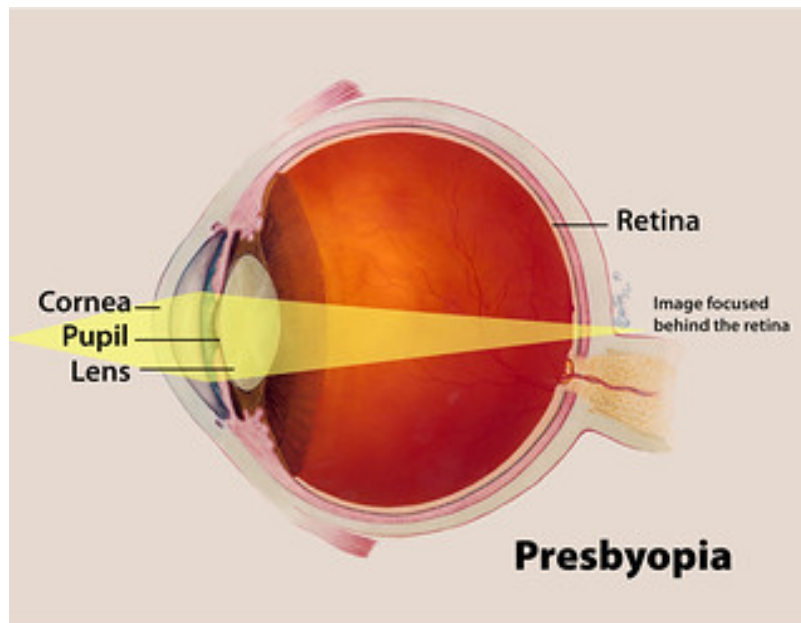


Fig.8.4.1 Presbyopia. As we age, it becomes harder for our eyes to focus an image on the retina. In this diagram, it is shown that the image comes into focus behind the retina. (credit: National Eye Institute, National Institutes of Health., Flickr, License: CC BY 2.0)

Age-related macular degeneration (AMD), the leading cause of worldwide blindness in the elderly, is a bilateral ocular condition that affects the central area of the retina known as the macula. Clinically, AMD is classified into the nonexudative “dry” or atrophic form and the exudative “wet” or neovascular form. More severe vision loss is typically associated with the “wet” form that occurs in about 15% of all patients with AMD but up to 20% of legal blindness from AMD is due to the “dry” form [3]. The typical clinical sign of “dry” AMD is pigment disruption and drusen (small yellowish deposits) in the retina. Drusen may be small “hard” (small with discrete margins) or “soft” (larger with indistinct edges). They lie between the RPE and an adjacent basement membrane complex known as Bruch’s membrane (BM). The designation of exudative or “wet” AMD implies that fluid, exudates and/or blood are present in the extracellular space between the neural retina and the RPE (i.e. the subretinal space) and/or, as in the case of RPE detachments, between the RPE and Bruch’s membrane (i.e. the sub-RPE space).

Here is a quick refresher video on how the eye works.



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Wikipedia, Presbyopia

URL: <https://en.wikipedia.org/wiki/Presbyopia>

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Webvision: The Organization of the Retina and Visual System, Age-related macular degeneration (AMD)

Authored by: Gregory S. Hageman, Karen Gaehrs, Lincoln V. Johnson and Don Anderson

URL: <https://webvision.med.utah.edu/book/part-xii-cell-biology-of-retinal-degenerations/age-related-macular-degeneration-amd/>

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Adapted by: Kori Skrypek

References

1. FACTS ABOUT PRESBYOPIA". *NEI*. OCTOBER 2010. ARCHIVED FROM THE ORIGINAL ON 4 OCTOBER 2016. RETRIEVED 11 SEPTEMBER 2016.
2. PÉREZ-PRADOS, ROQUE; PIÑERO, DAVID P; PÉREZ-CAMBRODÍ, RAFAEL J; MADRID-COSTA, DAVID (MARCH 2017). "SOFT MULTIFOCAL SIMULTANEOUS IMAGE CONTACT LENSES: A REVIEW". *CLINICAL AND EXPERIMENTAL OPTOMETRY*. **100** (2): 107–127. DOI:10.1111/CXO.12488. PMID 27800638.
3. Sunness JS. The natural history of geographic atrophy, the advanced atrophic form of age-related macular degeneration. *Mol Vis*. 1999;5:25. [PubMed]

THE RETINAL NETWORK

Learning Objectives

Being able to describe the different layers and cell types in a retina

Know what are the differences between rods and cones

The retina is composed of several layers and contains specialized cells for the initial processing of visual stimuli. The photoreceptors (rods and cones) change their membrane potential when stimulated by light energy. The change in membrane potential alters the amount of neurotransmitter that the photoreceptor cells release onto bipolar cells in the outer synaptic layer. It is the bipolar cell in the retina that connects a photoreceptor to a retinal ganglion cell (RGC) in the inner synaptic layer. There, amacrine cells additionally contribute to retinal processing before an action potential is produced by the RGC. The axons of RGCs, which lie at the innermost layer of the retina, collect at the optic disc and leave the eye as the optic nerve (Fig.8.5.1). Because these axons pass through the retina, there are no photoreceptors at the very back of the eye, where the optic nerve begins. This creates a “blind spot” in the retina, and a corresponding blind spot in our visual field.

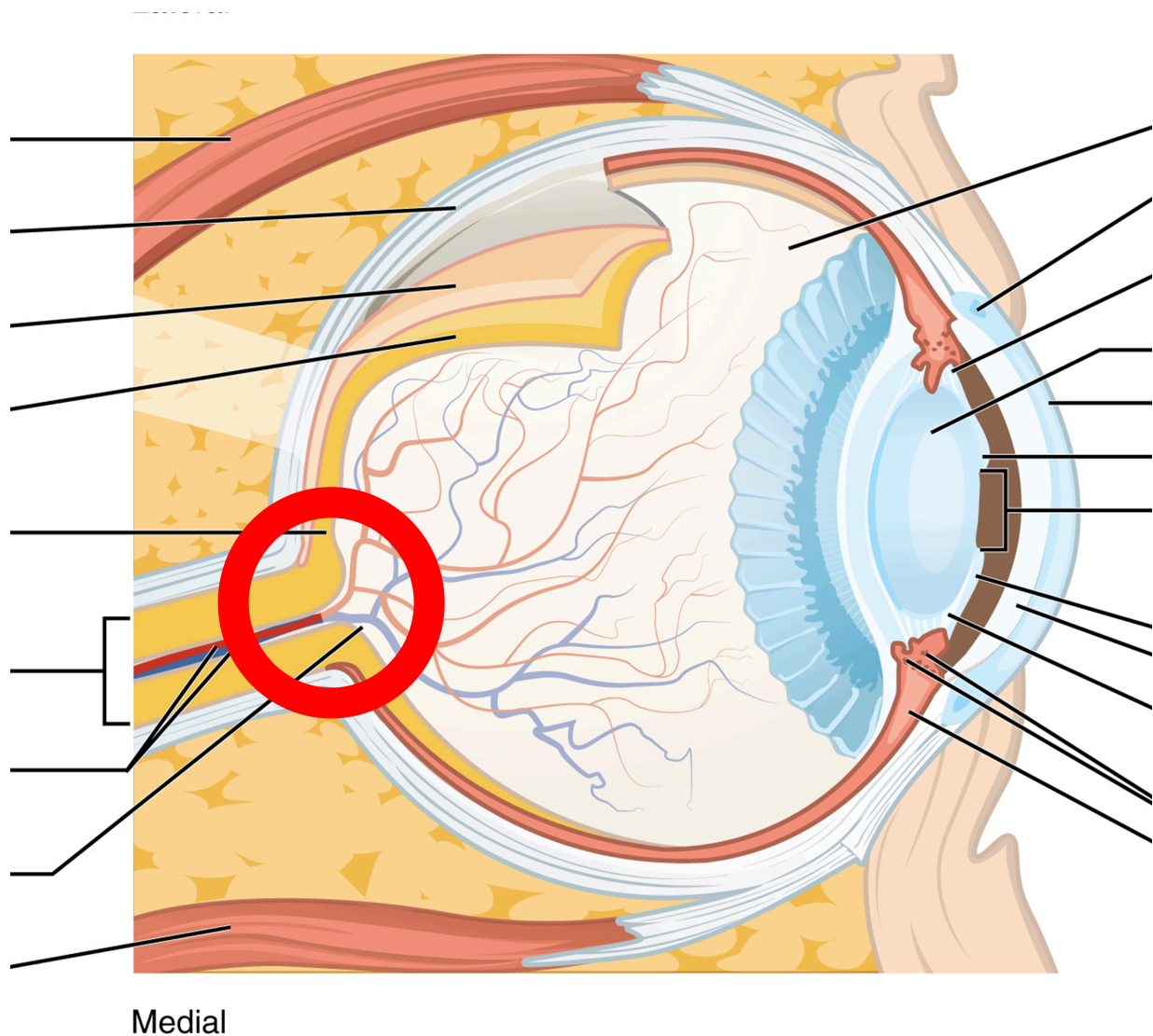


Fig.8.5.1. Structure of the Eye. The area circles in red is the blind spot, it is where sensory data from the eye begins its journey to the brain via the optic nerve. (Adapted by: Jarod Davis. Original image Provided by: OpenStax . License CC-BY 4.0)

The retinal surface includes two types of photoreceptors called rods and cones based on their shape (Fig.8.5.2). Rods are very sensitive to light and serve as the basis for vision in the dark, however visual acuity is poor in comparison to during daylight. Since rods are monochromatic (i.e., respond to only one color) and the information cannot be combined with information from cone receptors, very limited color perception is possible in the dark. There are three types of cones activated during daylight, each enabling perception of color on limited sections of the visible part of the electromagnetic spectrum: those responding to short (bluish), medium (greenish), and long (reddish) wavelengths.

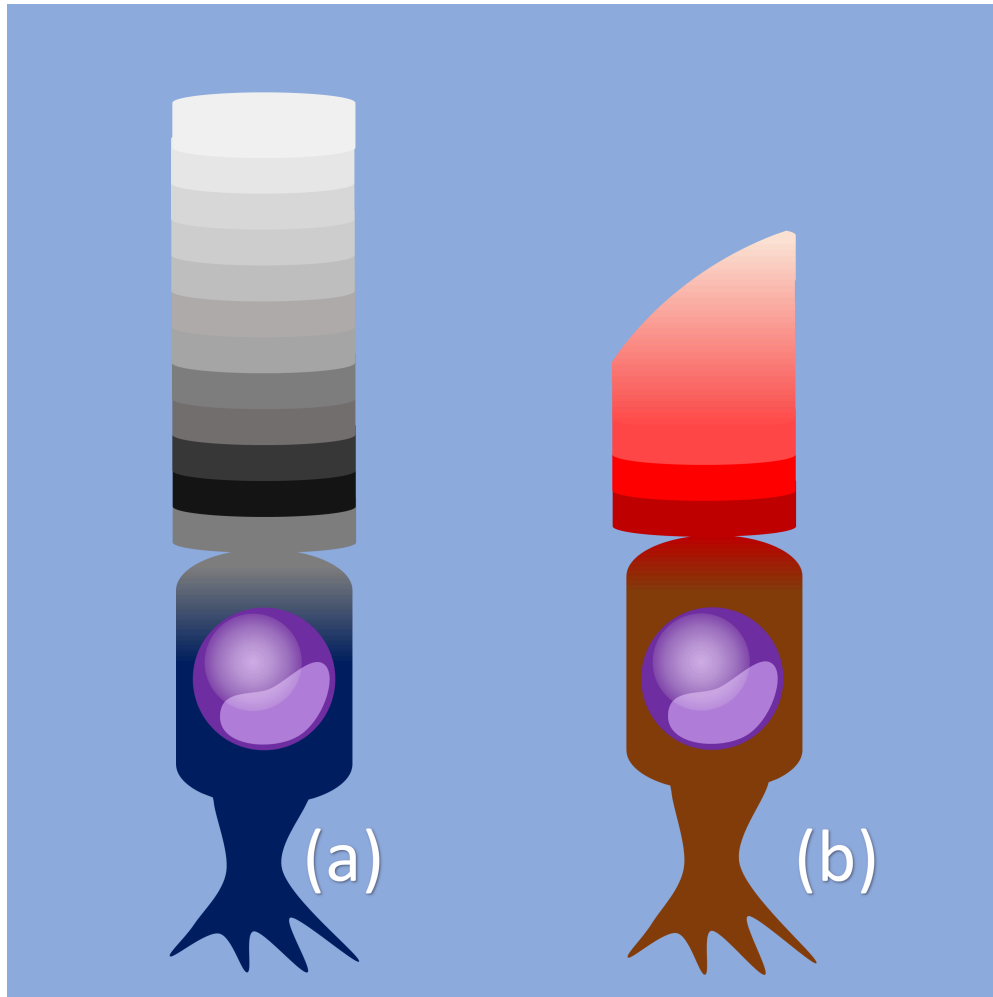


Fig.8.5.2. Rods and Cones. Rods(a) are very sensitive photoreceptors. They do not produce signals for color. Cones(b) are less sensitive than rods, but produce signals relating to a particular color associated wavelength. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-NC-ND)

The outer segments of the rods and cones are mixed together to tessellate the back half of the eyeball and catch light from everywhere, with two exceptions: In the fovea, there are only cones. In the blind spot there are no photoreceptors, because all of the axons are leaving the eye.

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perception

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Adapted by: Kathryn TaterkaPsychology by Jeffrey C. Levy, Vision

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Adapted by: Kathryn Taterka

86.

LIGHT TRANSDUCTION

Learning Objectives

Being able to describe how light transduction is achieved on a retina

Know why the role of the pigment epithelium in the light transduction process.

How do we experience light? When light reaches the back of the eye, it enters the retina. The retina is composed of many layers and contains specialized cells known as photoreceptors. There are two types of photoreceptors: rods and cones. Rods are sensitive to vision in low light and do not sense the color of light. Whereas, cones are sensitive to brighter light and allow us to perceive color in normal lighting.

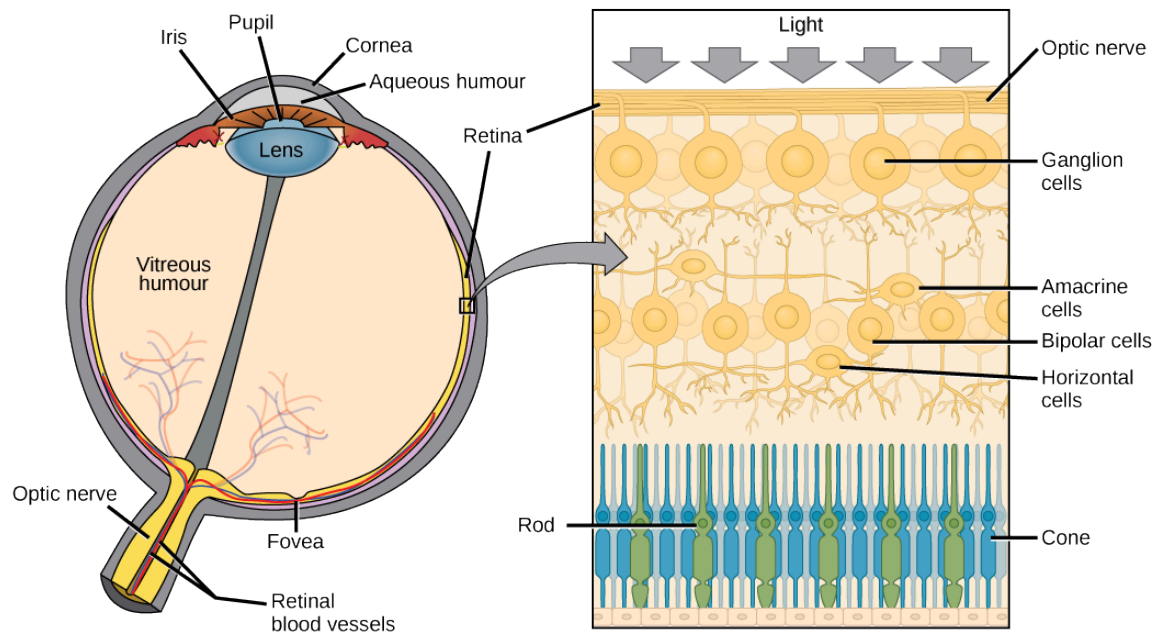


Figure 36.17 (a) The human eye is shown in cross section (b) A blowup shows the layers of the retina. (credit: OpenStax Biology, figure 36.17, CC-BY 4.0, <https://openstax.org/books/biology/pages/36-5-vision>. no modifications.)

Light transduction happens in the outer segments of the rods and cones. This means that light travels through several layers (ganglion cells, bipolar and amacrine cells) before it does anything! Light falling on the retina causes chemical changes to pigment molecules in the photoreceptors, ultimately leading to a change in the activity of the rods and cones. When light (of the right wavelength/color) hits 11-cis retinal, it changes conformation (to all-trans), acting as a switch to start an enzyme cascade in the cell, which eventually changes the rate at which photoreceptors release neurotransmitters.

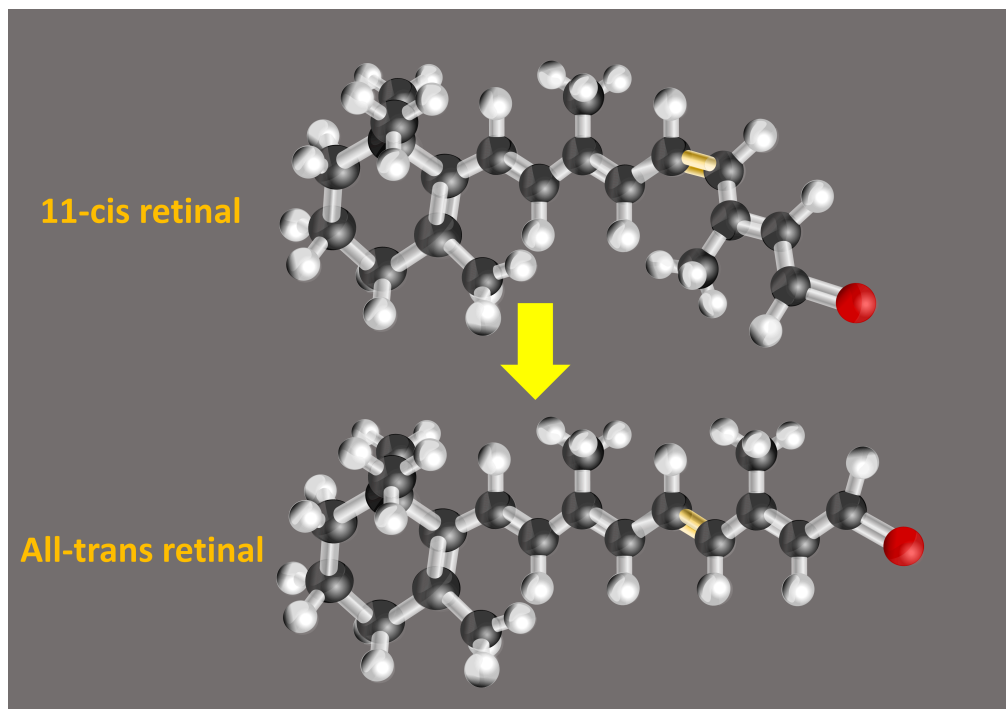


Fig. 8.6.1. In response to light, retinal changes confirmation from 11-cis retinal to all-trans retinal. (credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-NC4.0)

After absorbing light, all-trans retinal needs to be turned back into 11-cis retinal. So it breaks off its opsin, finds its way to the pigment epithelium, gets bent back into shape, and finds its way back to an opsin. This is why photoreceptor outer segments need to be close to the pigment epithelium, which is the back layer behind the retina where visual pigments are replenished

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87.

ECCENTRICITY

Learning Objectives

Know the idea of eccentricity in degrees, and how big is our fovea and macula.

Being able to describe the density of cones and rods from the fovea to the periphery.

Know the visual acuity in our foveal vision and peripheral vision.

In a normal-sighted individual, the lens will focus images perfectly on a small indentation in the back of the eye known as the fovea, which is part of the retina, the light-sensitive lining of the eye. The fovea contains densely packed specialized photoreceptor cells. These photoreceptor cells, known as cones, are light-detecting cells. The cones are specialized types of photoreceptors that work best in bright light conditions. Cones are very sensitive to acute detail and provide tremendous spatial resolution. They also are directly involved in our ability to perceive color.

Although the macula comprises only 4% of the retinal area and 10% of the entire visual field, it is responsible for the majority of useful photopic vision. The fovea lies at the center of the macula and is approximately 2mm in diameter. The fovea is the central 1 degree of the visual field; the macula is the central 5 degrees of visual angle. The fovea contains the highest density of cone photoreceptor cells and is the only region of the retina where 20/20 vision is attainable. We have high acuity (ability to see fine detail) in the fovea because there is little convergence: basically one photoreceptor for every output ganglion cell. Acuity is also improved by high light levels (smaller pupil, ideally 2-5mm), longer exposure times, and appropriate focusing of the image by cornea and lens.

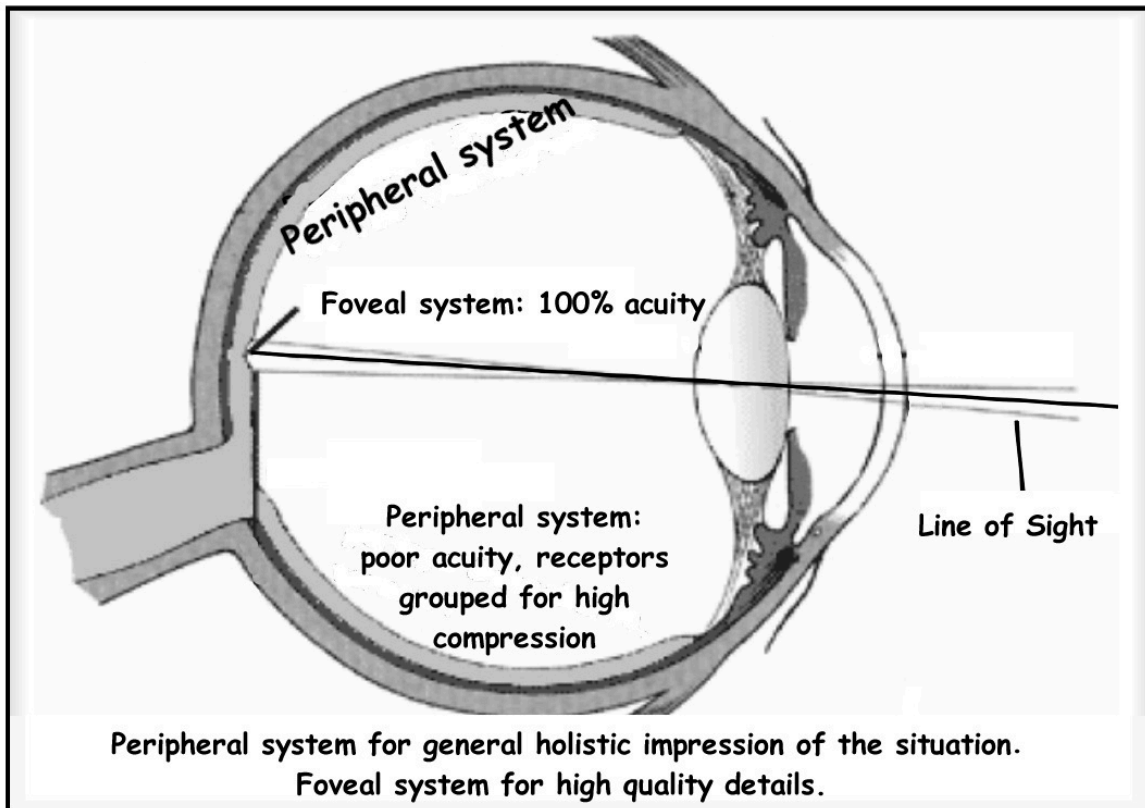


Fig. 8.7.1 The eye as a double system: the foveal system delivers 3 to 4 high quality images per second (via the line of sight). The peripheral system delivers up to 90 blurred images per second. (credit: Hans-Werner Hunziker, Provided by: Wikimedia Commons, License: CC BY SA.)

While cones are concentrated in the fovea, where images tend to be focused, rods, another type of photoreceptor, are located throughout the remainder of the retina. Rods are specialized photoreceptors that work well in low light conditions, and while they lack the spatial resolution and color function of the cones, they are involved in our vision in dimly lit environments as well as in our perception of movement on the periphery of our visual field.

The peripheral retina is dominated by rods, but also contains cones. We have low acuity in the periphery because there is strong convergence: many rods map to a single output ganglion cell. (Rods are more sensitive to light than cones.) Convergence (each ganglion cell pools responses from multiple photoreceptors) increases sensitivity but decreases acuity. There is lots of convergence for rods; much less convergence for cones.

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(AMD)

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88.

SACCADES

Learning Objectives

Know what the saccades are.

Know why we need saccades.

Being able to view objects such as pictures is a knowledgeable part of the task of seeing. Viewing things is achieved by the orientation of the eyes. This means that you are directing the visual axes to point to a new location. Large movements used when viewing large objects involve coordinated motion from the eyes, head, and body. Small movements used to view smaller objects such as pictures are viewed with just the eyes. The smaller movements are known as saccades. Saccades refers to rapid jerk-like movements of the eyes between fixation points (Fig.8.8.1). Most saccadic movement is involuntary and does not provide us with useful information until fixated on a particular point.

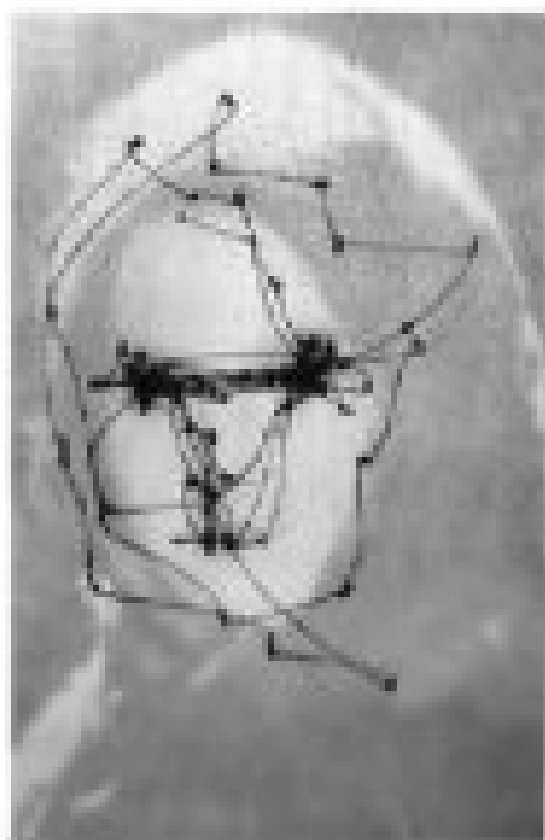


Fig.8.8.1. Visual Scan Path. The image above shows the 'scan path' after looking at the picture for 2 minutes.(Provided by: Scholarpedia. License: CC-BY-NC-SA)

Saccades can be produced either voluntarily or involuntarily. The production of saccades is important to our eye health because they allow for us to direct the fovea onto an object or region of interest. This then enables our mind to begin a high-acuity detail analysis of said object or region.

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Scholarpedia, "Human saccadic eye movements" by John Findlay, University of Durham, UK & Robin Walker, Department of Psychology, Royal Holloway, University of London. UK

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89.

DARK ADAPTATION

Learning Objectives

Being able to describe different stages of dark adaptation (time duration, luminance levels...etc)

Know the differences between our scotopic and photopic vision

Dark adaptation refers to the ability of both rod (scotopic) and cone (photopic) mechanisms to recover sensitivity in the dark following exposure to bright lights. The first rapid recovery is attributed to the cones and the later recovery to the rods.

Let's Break It Down: A Timeline

- **First Few Minutes:** The rods and cones both become more sensitive during the first few minutes. However, after 5-10 minutes, the cones reach their maximum sensitivity. This is known as the rod-cone break because it's the point where rods become more sensitive than cones.
- **5-10 Minutes:** The rods will continue to become more sensitive over the next few hours as they regenerate a receptor protein called rhodopsin. After 7 or 8 minutes, the rod photopigments have replenished enough that the rods become the most sensitive cells in the retina, and sensitivity continues to improve for another 13-22 minutes (20-30 total) while the rod visual pigment finishes replenishing.
- **30 Minutes:** At this point, the rhodopsin has mostly been replenished, so you should be about 90% dark-adapted.

Rhodopsin, a photopigment, plays a large role in dark adaptation because it's so sensitive. However, light causes it to deteriorate rapidly, in a process called photobleaching. Photobleaching occurs when the pigment epithelium cannot regenerate 11-cis retinal as fast as it is converted to all-trans retinal. During daylight viewing, rods are essentially completely photobleached, and cones are partially photobleached.

So if you're trying to get dark-adapted, it's crucial to avoid light – it can undo hours of dark adaptation in

seconds. All the rhodopsin you have built up over the previous 30+ minutes disappears, and it will take time for your retina to replenish it.

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Webvision: The Organization of the Retina and Visual System, Light and Dark Adaption by Michael Kalloniatis and Charles Luuz

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90.

CENTER-SURROUND ANTAGONISM IN RECEPTIVE FIELDS

Learning Objectives

Know what's the center-surround receptive field

Being able to describe the differences between an ON-center and OFF-center receptive field.

Know what types of retinal cells have the center-surround receptive fields.

The definition of a visual receptive field is the region of visual space in which a change in lightness or color will cause a change in the neuron's firing rate. Almost all receptive fields have structure — different changes in different parts of the receptive field will have different effects on the neuron's response. A key function of this receptive field structure: neurons only respond to edges. When center and surround are balanced, the RGC (retinal ganglion cell) will not change its firing rate in response to uniform illumination.

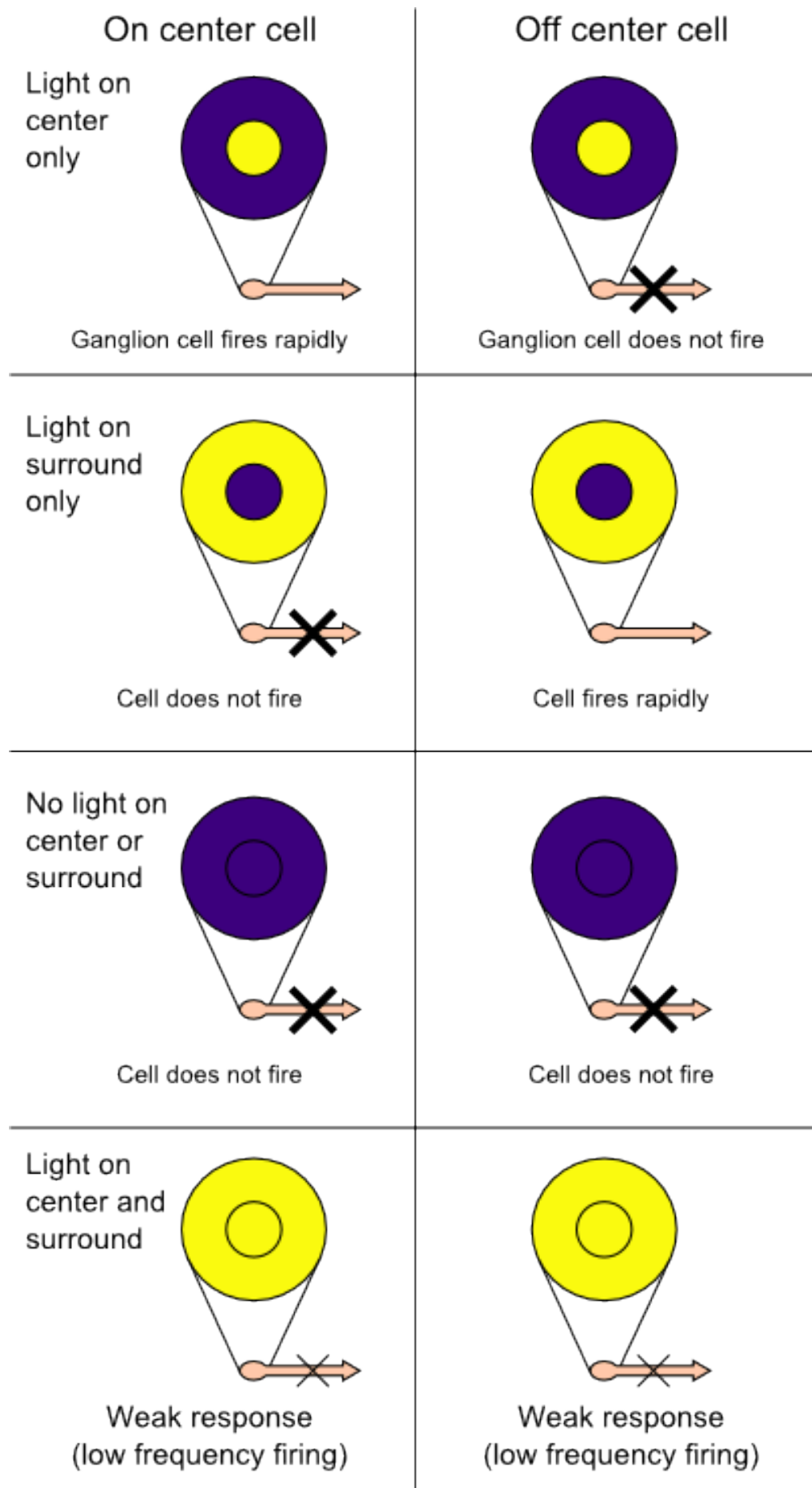


Fig.8.10.1. Caption. In this figure we can see that retinal ganglion cells have the center-surround receptive fields and that they respond to light differently on the center only, surround only, both center and surround, or on neither center nor surround. (Provided by: Wikimedia Commons, License: CC-BY-4.0)

Most neurons in the retina and thalamus have small receptive fields that have a very basic organization, which resembles two concentric circles. This concentric receptive field structure is usually known as center-surround organization. On-center retinal ganglion cells respond to light spots surrounded by dark backgrounds like a star in a dark sky. Off-center retinal ganglion cells respond to dark spots surrounded by light backgrounds like a fly in a bright sky.

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Alonso & Dr. Yao Chen, SUNY State College of Optometry

URL: http://www.scholarpedia.org/article/Receptive_field

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LATERAL INHIBITION

Learning Objectives

Be able to describe what lateral inhibition is.

Be able to give at least one example/illusion caused by lateral inhibition.

Lateral inhibition is a secondary signal sent by a neuron to reduce the responses of neighboring neurons when firing information. In vision this can be observed through a neuron that is responding to differences in light, which will fire one signal of information about light to the next level in vision while a second signal is sent to neighboring neurons to suppress their firing rate. This suppression of response in neighboring neurons helps to pinpoint visual information by exaggerating differences in light.

There are four basic types of lateral inhibition:

1. Transient response with an excitatory center and inhibitory surrounding, or a shorter response in the retinal ganglion cells (RGC) with a receptive field that has a high firing rate in the center and low firing rate in the surrounding part of the receptive field.
2. Transient response with an inhibitory center and excitatory surrounding, or a shorter response in the RGC with a receptive field that has a low firing rate in the center and high firing rate in the surrounding part of the receptive field.
3. Sustained response with an excitatory center and inhibitory surrounding, or a longer response in the RGC with a receptive field that has a high firing rate in the center and low firing rate in the surrounding part of the receptive field.
4. Sustained response with an inhibitory center and excitatory surrounding, or a longer response in the RGC with a receptive field that has a low firing rate in the center and high firing rate in the surrounding part of the receptive field.

Lateral inhibition can explain Mach bands, or the illusion that light and dark lines exist adjacent to sudden changes in brightness, and the gray dots that appear between intersections in the Hermann grid illusion (Fig.8.11.1).

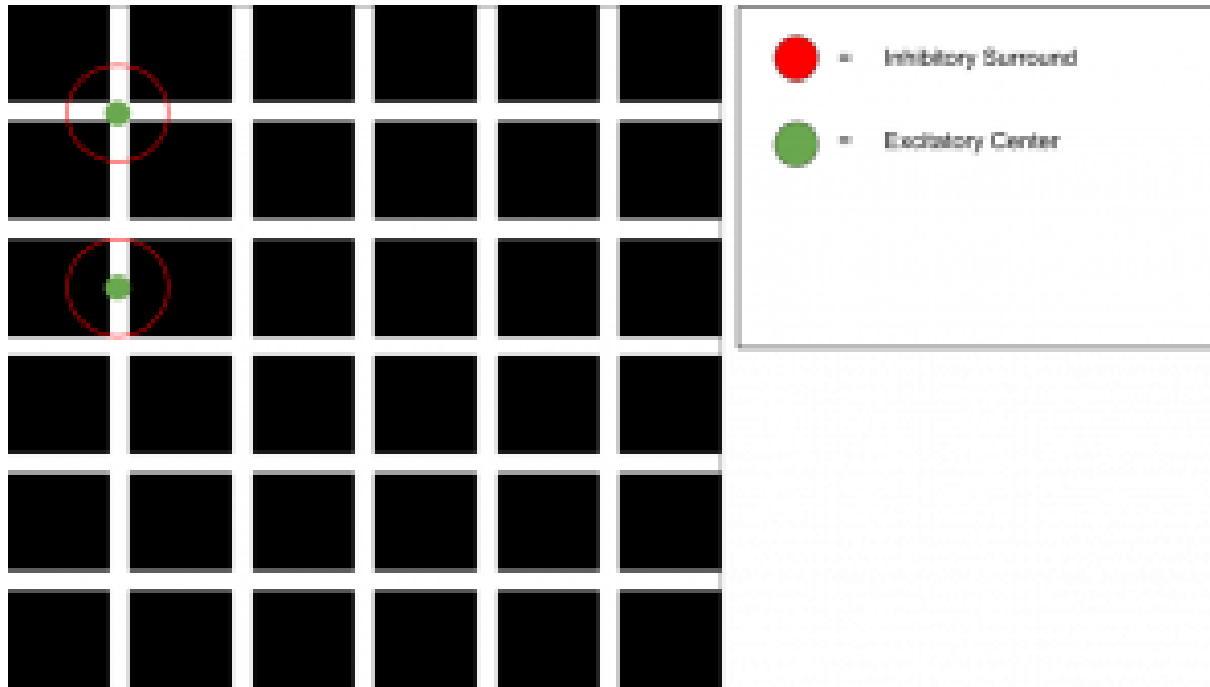


Fig. 8.11.1. Hermann Grid Illusion. The Hermann grid illusion is the perception of gray dots at each intersection of white lines when not directly looking at the intersection. The bottom circle shows that when looking at lines between intersections, the inhibitory surround is mostly picking up black, making the white line appear brighter due to the contrast in light. The top circle also has black in the inhibitory surround which causes the intersection to appear brighter. However, since there is less black in the top circle than the bottom, there is more emphasis on the brightness between intersections, causing the illusion of gray dots. (Provided by: Wikimedia Commons. License: CC-BY-SA 4.0)

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92.

ACTIVE LEARNING EXERCISES

This slide show has activities and links to activities that will reinforce concepts of presbyopia, the blind spot, lateral inhibition, and chromatic adaptation.



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SECTION IX

VISION LOSS AND V1

Again following the example of our section on Hearing, the 2nd chapter of the vision section contains discussion of leading causes for blindness worldwide and in the United States, and treatments, when they're available. Then we continue on to talk about the neural pathways and structures that relay visual information to the brain, and how visual information is represented in the primary visual area of the brain.

This chapter was created by McKenzie Roberts, Ayane Shimasaki, Kayla Spychalla, Ava Stende, Ryan Taufen, Kia Thompson, Katya Tomlin, Ramla Warsame, Risa Ya, Jingyi Zhao

93.

LOW VISION

Learning Objectives

Know the definition of low vision and legal blindness

Know the prevalence of low vision

Being able to explain why we are still expecting more low vision people in our society given that we have better and more treatments for many eye diseases.

Low vision is constituted by the visual acuity in one's best eye being worse than 20/60 even with correction. Low vision occurs in 2% of the worldwide population (124 million people).

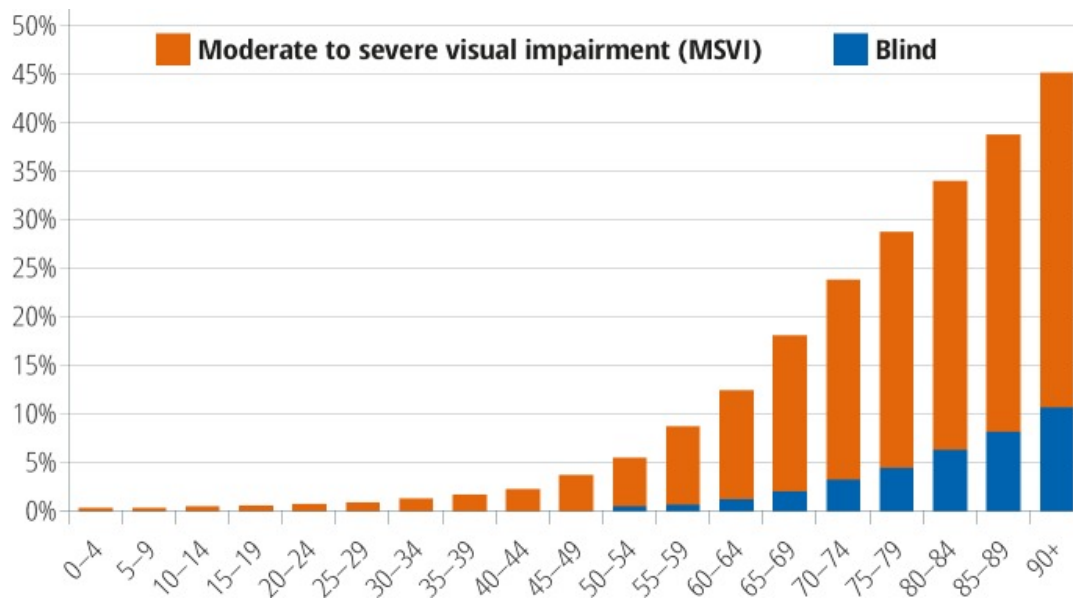


Figure 9.1.1. The global prevalence of blindness and low vision (moderate to severe visual impairment) in women. (PubMed Central, Figure 2, CC BY-NC, <http://ncbi.nlm.nih.gov/pmc/articles/PMC5820628/figure/F6>. No Modifications.)

For one to be constituted as legally blind, the visual acuity of their best eye must be worse than 20/200 (or only 20 deg. visual field.). 0.6% of the worldwide population is blind.

With the youngest of the baby boomers hitting 65 by 2029, the number of people with visual impairment or blindness in the United States is expected to double to more than 8 million by 2050, according to projections based on the most recent census data and from studies funded by the National Eye Institute, part of the National Institutes of Health. The data tells us that we should prepare for the surge in visual impairment by implementing further nationwide screening in order to identify people with correctable vision problems and early signs of eye disease. Early detection and intervention could be one of the best options in our attempt to avoid a significant proportion of avoidable vision loss (Fig.9.1.1). Once low vision or blindness occur, they are usually permanent, making early detection and intervention one of the top priorities in our attempt to avoid a significant proportion of avoidable vision loss.

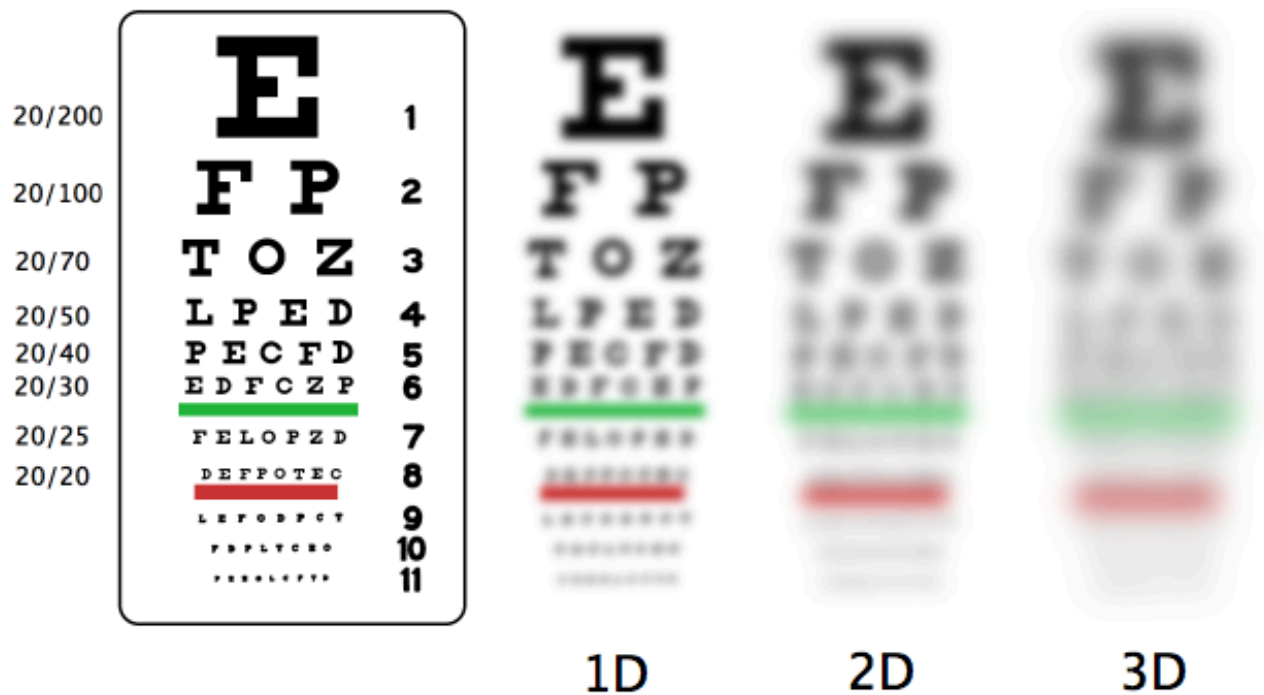


Fig.9.1.1. The Snellen Chart. The typical Snellen chart, originally developed by Dutch ophthalmologist Herman Snellen in 1862, used in most optometry offices to measure visual acuity. The leftmost image displays how the Snellen Chart appears to a typically sighted person. The three images to the right correspond roughly to the visual experiences of those with various levels of low vision or blindness. (Provided by: Wikipedia. License: CC-BY-SA 4.0.)

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Adapted by: Mckenzie RobertsCONTENT SHARED PREVIOUSLY

National Eye Institute, "Visual impairment, blindness cases in U.S. expected to double by 2050"

URL: <https://www.nei.nih.gov/about/news-and-events/news/visual-impairment-blindness-cases-us-expected-double-2050>

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Adapted by: Mckenzie Roberts

Reference

Varma, R et al, “Visual impairment and blindness in adults in the United States: Demographic and Geographic Variations from 2015 to 2050,” JAMA Ophthalmology, DOI:10.1001/jamaophthalmol.2016.1284 Pubmed

CAUSES OF VISION LOSS

Learning Objectives

Know what are the major causes of low vision in the US.

Being able to identify what parts of the eye are affected by cataracts, macular degeneration, and glaucoma

Being able to describe the impact of different diseases on our visual function. e.g. visual acuity, visual field. and dark adaptation.

The leading causes of blindness and low vision in the United States are primarily age-related eye diseases such as age-related macular degeneration, cataract, diabetic retinopathy, and glaucoma. Other common eye disorders include amblyopia and strabismus.

Most cataracts are caused by normal changes in your eyes as you get older. When you're young, the lens in your eye is clear. Around age 40, the proteins in the lens of your eye start to break down and clump together. This clump makes a cloudy area on your lens — or a cataract (Fig.9.2.1). Over time, the cataract gets more severe and clouds more of the lens.

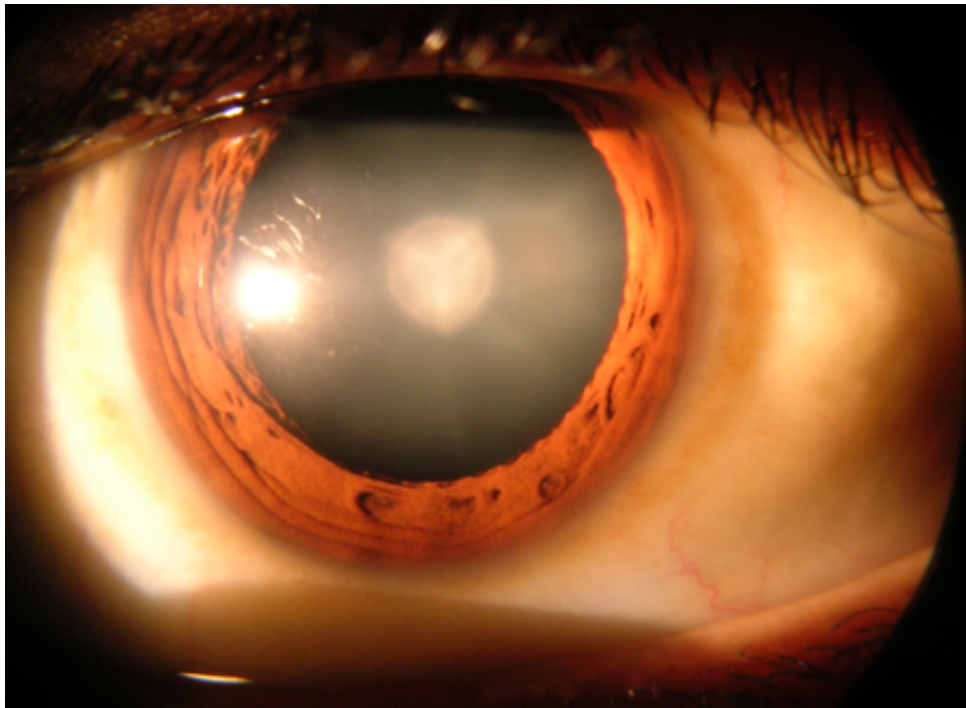


Figure 9.2.1. Cataract. A magnified image of a cataract, it can be identified by the cloudy appearance in the pupil.(Provided by: Wikipedia. License: CC-BY SA 3.0)

Macular degeneration, often called **age-related macular degeneration (AMD)**, is an eye disorder associated with aging and results in damaging sharp and central vision. AMD affects the macula, the central part the retina that allows the eye to see fine details. There are two forms of AMD—wet and dry.

Wet AMD is when abnormal blood vessel behind the retina start to grow under the macula, ultimately leading to blood and fluid leakage. Bleeding, leaking, and scarring from these blood vessels cause damage and lead to rapid central vision loss.

Dry AMD is when the macula thins over time as part of aging process, gradually blurring central vision. Over time, as less of the macula functions, central vision is gradually lost in the affected eye.

AMD doesn't cause complete blindness, but losing your central vision can make it harder to see faces, drive, or do close-up work like cooking or fixing things around the house.

AMD happens very slowly in some people. Even if you have early AMD, you may not experience vision loss for a long time. For other people, AMD progresses faster and can lead to central vision loss in one eye or both eyes.

As AMD progresses, many people see a blurry area near the center of their vision. Over time, this blurry area may get bigger or you may see blank spots. Things may also seem less bright than before.

Some people may also notice that straight lines start to look wavy. This can be a warning sign for late AMD.

Glaucoma is a group of diseases that can damage the eye's optic nerve and result in vision loss and blindness. Glaucoma occurs when the normal fluid pressure inside the eyes slowly rises.

At first, glaucoma doesn't usually have any symptoms. That's why half of people with glaucoma don't even know they have it.

Over time, you may slowly lose vision, usually starting with your side (peripheral) vision — especially the part of your vision that's closest to your nose. Because it happens so slowly, many people can't tell that their vision is changing, especially at first.

But as the disease gets worse, you may start to notice that you can't see things off to the side anymore. Without treatment, glaucoma can eventually cause blindness.

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National Eye Institute, Cataracts

URL: <https://www.nei.nih.gov/learn-about-eye-health/eye-conditions-and-diseases/cataracts>

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Center for Disease Control and Prevention, Common Eye Disorders and Diseases

URL: <https://www.cdc.gov/visionhealth/basics/ced/index.html>

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MACULAR DEGENERATION

Learning Objectives

Being able to describe the cause of macular degeneration

Know what are the two main types of age-related macular degeneration

Age-related macular degeneration (AMD), the leading cause of worldwide blindness in the elderly, is a bilateral ocular condition that affects the central area of the retina known as the macula. The macula accounts for almost 10% of the entire visual field. Thus, lesions developing in this region can have a major impact on visual function.

The two main types of age-related macular degeneration are dry and wet. Age-related macular degeneration is generally thought to progress along a continuum from dry AMD to wet AMD. Dry AMD is progressive, with gradual loss of visual function that may span over many years. The typical clinical sign of dry AMD is the pigment disruption and drusen (small yellowish deposits in Fig.9.3.1) in the retina. Approximately 10-15% of all AMD patients will eventually develop the wet form. Wet AMD implies that fluid, exudates and/or blood are present in the extracellular space between the neural retina and the RPE (i.e. the subretinal space) and/or, as in the case of RPE detachments, between the RPE and Bruch's membrane (i.e. the sub-RPE space). Wet AMD can advance to severe central vision loss.



Fig.9.3.1 Dry Macular Degeneration. A color fundus photograph from an individual with dry AMD. They have numerous small and intermediate-sized drusen visible in the macular region (oval).(Provided by: Webvision. License: CC-BY 2.0)

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Webvision: The Organization of the Retina and Visual System, Age-related macular degeneration (AMD)

Authored by: Gregory S. Hageman, Karen Gaehrs, Lincoln V. Johnson and Don Anderson

URL:<https://webvision.med.utah.edu/book/part-xii-cell-biology-of-retinal-degenerations/age-related-macular-degeneration-amd/>

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PREVENTION AND TREATMENT FOR VISION LOSS

Learning Objectives

Know what are the preventions for some common eye diseases, such as glaucoma, macular degeneration, and diabetic retinopathy.

Being able to list some treatment options for some common eye diseases. such as cataract. glaucoma, macular degeneration, and retinitis pigmentosa.



Fig. 9.4.1 There are more ways to protect your eyes than sunglasses. (credit: Max Pixel, License CC0.)

To preserve eyesight for as long as possible, it is very important to know how to prevent eye disease of any kind, and it is helpful to know when and how to seek treatment.

Glaucoma, a group of optic neuropathies characterized by progressive degeneration of retinal ganglion cells (RGCs), represents the leading cause of irreversible blindness in the developed world. Glaucoma is generally identified by abnormal regulation of intraocular pressure (IOP) and/or pathological mechanosensitivity of ocular cells. Current treatments are limited to lowering and stabilizing IOP, indicating that glaucoma is principally a disease of ocular mechanotransduction. In order to prevent glaucoma, it is important to detect pressure build up that has been caused by the blockage of flow of aqueous liquid before the resulting pressure damages the head of the optic nerve.

There are two types of macular degeneration, wet and dry. Dry is less severe and slowly progressing. The best way to prevent macular degeneration is with early detection. This early detection permits injection of anti vascular drugs to keep elaboration of blood vessels from blocking vision. Besides treatment with anti vascular drugs, gene therapy may be a promising treatment for some forms of macular degeneration.

If diabetes goes untreated, it can begin to harm your vision. This is referred to as diabetic retinopathy. In order to prevent this from occurring, it is best to control the diet and blood sugar. This in turn can stall peripheral degeneration.

For eye diseases that have genetic causes, like retinitis pigmentosa (RP), there is no one treatment. Researchers are working hard to discover effective treatments. An NEI-sponsored clinical trial found that a daily dose of 15,000 international units of vitamin A palmitate modestly slowed the progression of the disorder (RP) in adults. An artificial vision device called the Argus II has also shown promise for restoring some vision to people with late-stage RP. The Argus II, developed by Second Sight with NEI support, is a prosthetic device that functions in place of lost photoreceptor cells. Although it does not restore normal vision, in clinical studies, the Argus II enabled people with RP to read large letters and navigate environments without the use of a cane or guide dog.

A cataract is a cloudy area in the lens of your eye. No matter what type of cataract you have, the treatment is always surgery. During cataract surgery, the doctor removes the clouded lens and replaces it with a new, artificial lens (also called an intraocular lens, or IOL). Early on, you may be able to make small changes to manage your cataracts. A new prescription for eyeglasses or contact lenses can help you see better with cataracts early on.

Exercises

1. You have noticed your peripheral vision closest to your nose has begun to decline. What disease may you have and how do you treat it?
 - A. Glaucoma and it can be treated by lowering intraocular pressure.
 - B. Retinitis pigmentosa and it can be treated by lowering intraocular pressure.
 - C. Glaucoma and it can be treated by surgery, removing clouded lens and replacing it with a new artificial one.
 - D. Cataract and it can be treated by surgery, removing clouded lens and replacing it with a new artificial one.

Answer: A

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Webvision: The Organization of the Retina and Visual System, What is Glaucoma? Authored by: David Križaj

URL: <https://webvision.med.utah.edu/book/part-xii-cell-biology-of-retinal-degenerations/what-is-glaucoma/>

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National Eye Institute, Retinitis Pigmentosa

URL: <https://www.nei.nih.gov/learn-about-eye-health/eye-conditions-and-diseases/retinitis-pigmentosa>

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National Eye Institute, Cataracts

URL: <https://www.nei.nih.gov/learn-about-eye-health/eye-conditions-and-diseases/cataracts>

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SENSORY SUBSTITUTION

Learning Objectives

Know some sensory substitution

Be able to describe examples of sensory substitution options available for reading and navigation.

Sensory substitution is the process of using a different sense (such as touch) to replace or makeup for the lack of another (commonly sight). Often technology is required to make this process more feasible and provides an alternative to those who lack the ability to experience sensations in the usual way.

There are many tools for visually impaired people on the market that can help them live more independent lives. Here are a few...

One example is Microsoft Seeing AI (<https://www.microsoft.com/en-us/ai/seeing-ai>). The app can be applied in different contexts, such as reading, scene recognition, and social interaction. It can transcribe printed text into audio by using the phone camera. People can also use it to recognize people's faces, currency, and some common scenes. It's an application of current developments of computer vision.

Another approach is the service of remote assistants, such as Aira (<https://aira.io/>). Users who subscribe to the service can connect with a trained assistant on their phones. The agent can see through a user's phone camera and offer requested assistance, such as reading mails or bills or navigation. Many public spaces in the US have adopted this service. For example, people with visual impairment can access this service in the MSP airport and many Target stores without further charges.

VoiceOver is also a popular accessibility function built in Apple products, such as iPhones, iPads and Macs. It can help users read texts on their devices.

These technologies cannot replace white canes or guide dogs or orientation training for the visually impaired, but they can provide more accurate information about their surrounding environment and the elements they interact with on a daily basis.

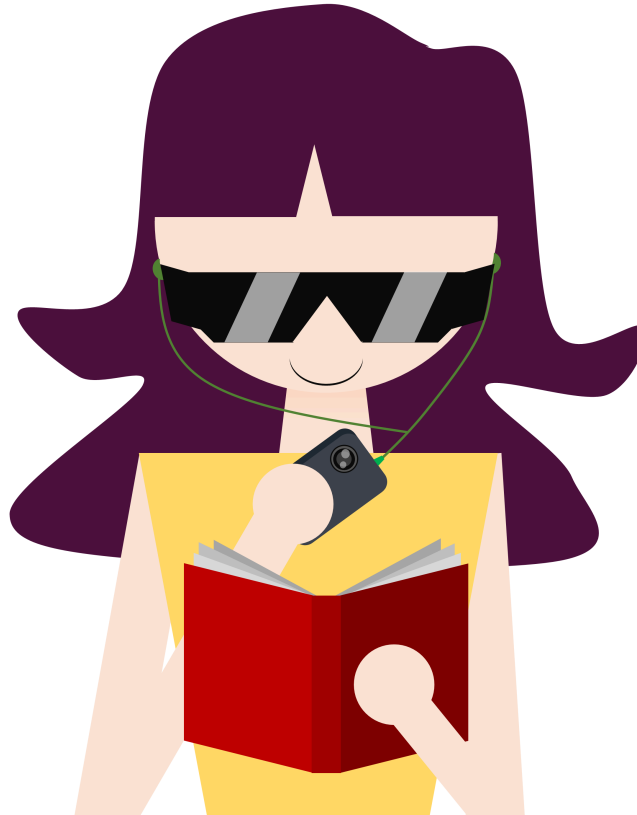


Fig.9.5.1. Blind British Girl Reading With Microsoft Seeing AI: Using her phone, this girl is using Microsoft AI to read a book. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-NC 4.0)

If you want to know more about sensory substitution, watch this video or take a look at this website.



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Adapted by: Jarod Davis

98.

ACTIVE LEARNING EXERCISE: BRAILLE



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99.

MAGNOCELLULAR AND PARVOCELLULAR PATHWAYS

Learning Objectives

Know the differences between the magnocellular and parvocellular pathway

Know how the M and P pathway are organized in LGN

There are two visual nuclei in the thalamus: the lateral geniculate nucleus (LGN) and the pulvinar, which we won't talk about. Neurons in the LGN have receptive fields that are center/surround like the retinal ganglion cells. The LGN has 6 layers, segregating inputs/outputs according to: The 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 (more details below); the names come from the fact that the retinal ganglion cells have large (magno) or small (parvo) cell bodies. It's useful to think of two streams of information coming from the eyes to the brain.

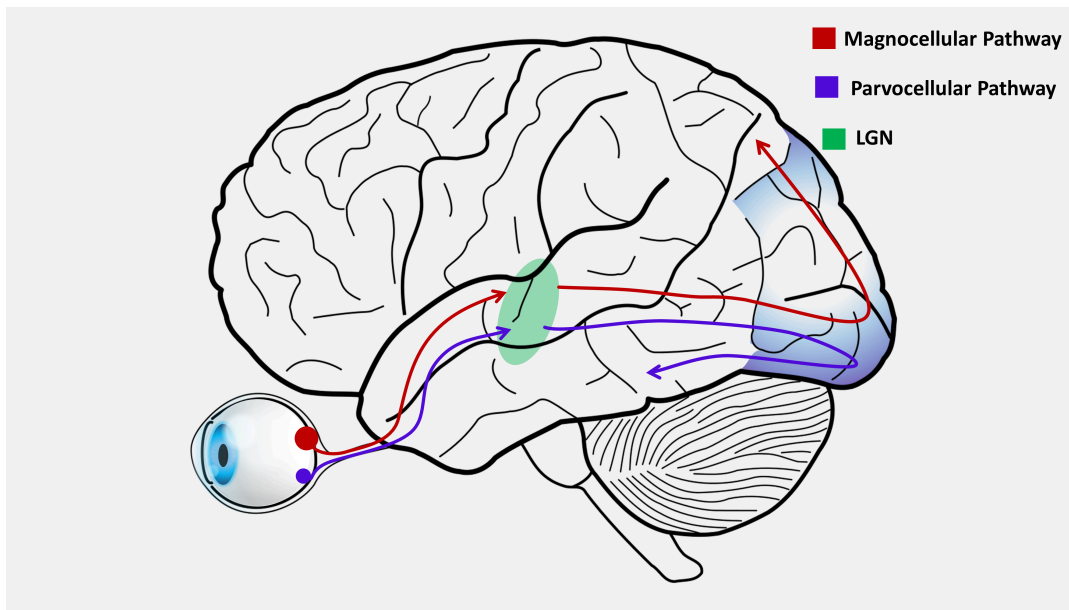


Fig. 9.7.1. Think of the Magno and Parvocellular pathways as 2 streams of different information that the brain combines and relates. (Credit: Jarod Davis. Provided by: University of Minnesota. Original image from: Wikipedia Commons. License: CC0)

The Magnocellular pathway carries information about large, fast things (low spatial frequency; high temporal frequency) and is colorblind. The Parvocellular pathway carries information about small, slow, colorful things (high spatial frequency information; low temporal frequency information).

Type	Size*	Source / Type of Information	Location	Response	Number
M: Magnocellular cells	Large	Rods; necessary for the perception of movement , depth, and small differences in brightness	Layers 1 and 2	rapid and transient	?
P: Parvocellular cells (or "parvicellular")	Small	Cones; long- and medium-wavelength ("red" and "green" cones); necessary for the perception of color and form (fine details).	Layers 3, 4, 5 and 6	slow and sustained	?
K: Koniocellular cells (or "interlaminar")	Very small cell bodies	Short-wavelength "blue" cones.	Between each of the M and P layers		

Figure 9.7.2 In humans the LGN is normally described as having six distinctive layers. The inner two layers, (1 and 2) are magnocellular layers, while the outer four layers, (3,4,5 and 6), are parvocellular layers. An additional set of neurons, known as the koniocellular layers, are found ventral to each of the magnocellular and parvocellular layers (credit: Wikipedia, https://en.wikipedia.org/wiki/Lateral_geniculate_nucleus, CC-BY-SA 3.0).

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Adapted by: Katya Tomlin

VISUAL PROSTHETICS

Learning Objectives

- Know how the retinal and cortical implant work.
- Know what are the requirements for the retinal and cortical implant.
- Know what's the limitation of current retinal and cortical implant.

Retinal implants take images from an external video camera which sends electric signals to an array implanted in the eye, bypassing damaged photoreceptors to kick-start retinal cells that are still viable.

A cortical implant is a subset of neuroprosthetics that is in direct connection with the cerebral cortex of the brain. Certain types of cortical implants can partially restore vision by directly stimulating the visual cortex.

Minimally, a patient must have an intact ganglion cell layer in order to be a candidate for a retinal implant. Optimal candidates for retinal implants have retinal diseases, such as retinitis pigmentosa or age-related macular degeneration. Other factors, including the amount of residual vision, overall health, and family commitment to rehabilitation, are also considered when determining candidates for retinal implants.

Cortical visual prostheses are important to people who have a completely damaged retina, optic nerve or lateral geniculate body, as they are one of the only ways they would be able to have their vision restored.

Limitations to the implants:

Both prosthetic solutions have a resolution problem: to provide even low vision, we would need to stimulate neurons with a spatial precision of 100 microns, and right now, the precision is measured in millimeters. So the current goal is to restore useful visual experiences to people who have none. Both approaches share the problems of biocompatibility and stability: the salt-water environment of the body is hostile to electronics, and scar tissue degrades performance of devices over time.

Retinal implants need to contend with the fact that the eye moves rapidly and the retinal sheet is delicate. Cortical implants have more neural territory to work with, but permanent brain implants bring risks of infection, inflammation, and other complications you don't want in your brain.

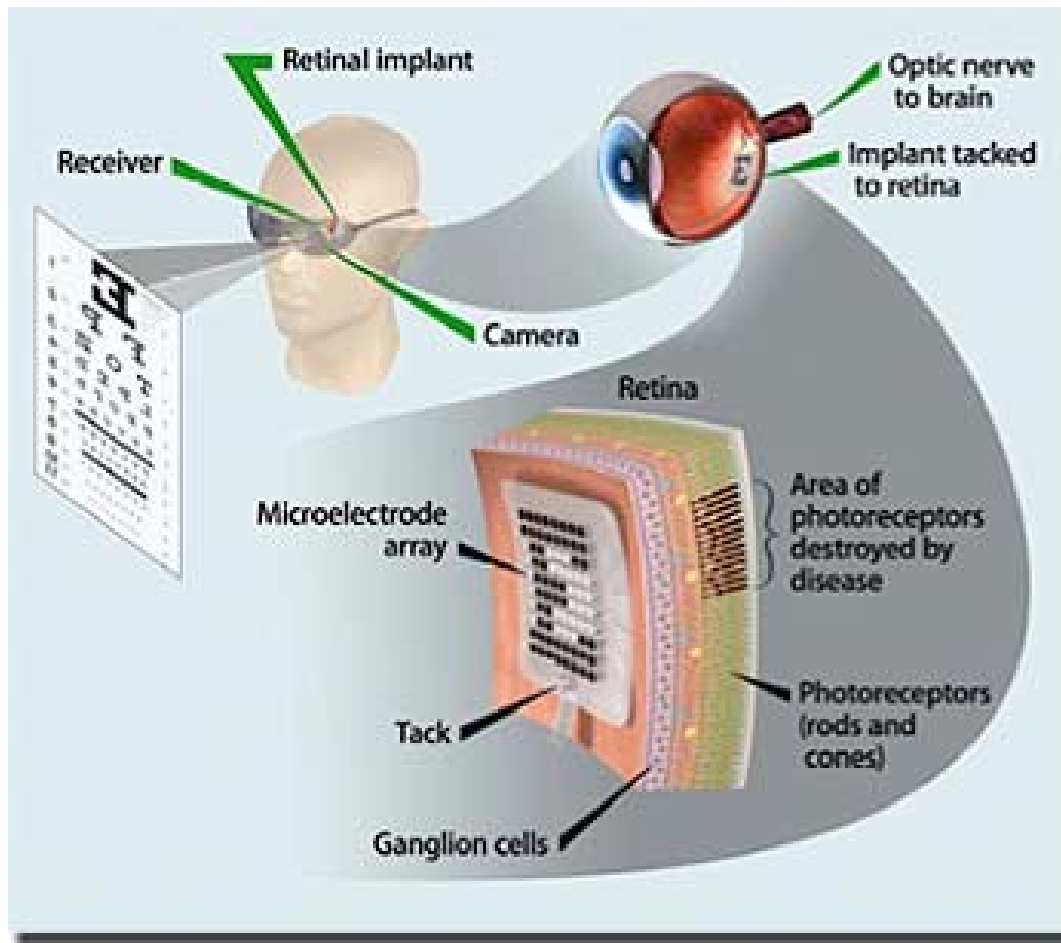


Fig.9.6.1. How the retinal implant works. (Provided by: Lawrence Livermore National Laboratory. License: CC BY-NC-SA..)

If you want to know more about retinal prosthesis systems, take a look at this additional creative commons source!

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Lawrence Livermore National Laboratory, "Retinal implant seen as 2010 tech breakthrough"

URL: <https://www.llnl.gov/news/retinal-implant-seen-2010-tech-breakthrough>

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Wikipedia, Cortical implant

URL: https://en.wikipedia.org/wiki/Cortical_implant#cite_note-Fernandes-4

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Wikipedia, Retinal implant

URL: https://en.m.wikipedia.org/wiki/Retinal_implant

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RETINOTOPIC ORGANIZATION OF V1

Learning Objectives

Know where is our primary visual cortex (V1).

Know how the V1 neurons are organized according to their receptive fields in visual space (retinotopic organization)

Primary visual cortex (V1) is located in the calcarine sulcus, has retinotopic organization and cortical magnification. The calcarine sulcus is in the occipital cortex, on the medial aspect. If you dissect away the white matter, the gray matter can be laid out like a sheet (flat). When you do this to V1, it is roughly U-shaped, and we use this visualization a lot because it helps us to identify how the V1 cortex is organized.

Retinotopic organization means that neurons with receptive fields close together in visual space have cell bodies close together in the cortex. Here's an interactive visualization of how the visual world is mapped to V1. What it shows is signals from each retina are sent to V1 on the opposite side of the brain. Essentially, the image is flipped in our brain as signals from our left eye are processed and mapped by the right side of V1 and vice versa. Furthermore, the neurons which help map the image are spatially located in corresponding regions in V1. For example, a stimulus in the center of one's right hand visual field will be mapped by the center neuron in the V1 region on the left side.

Area V1 has retinotopic organization, meaning that it contains a complete [map of the visual field | visual map] covered by the two eyes. In most species V1 is considered to have a single map of the visual field, but in cats it contains two of them: one for area 17 and one for area 18.

Neurons in area V1 are classically divided into two types: simple and complex, based on the structure of their receptive field. In simple cells, receptive fields have separate ON and OFF subregions. ON and OFF subregions differ in their responses to the onset of stimuli on a gray background: ON subregions respond white bars, and OFF subregions respond to black bars. In complex cells, instead, ON and OFF regions are superimposed, i.e. every location in the receptive field responds both to white and black bars.

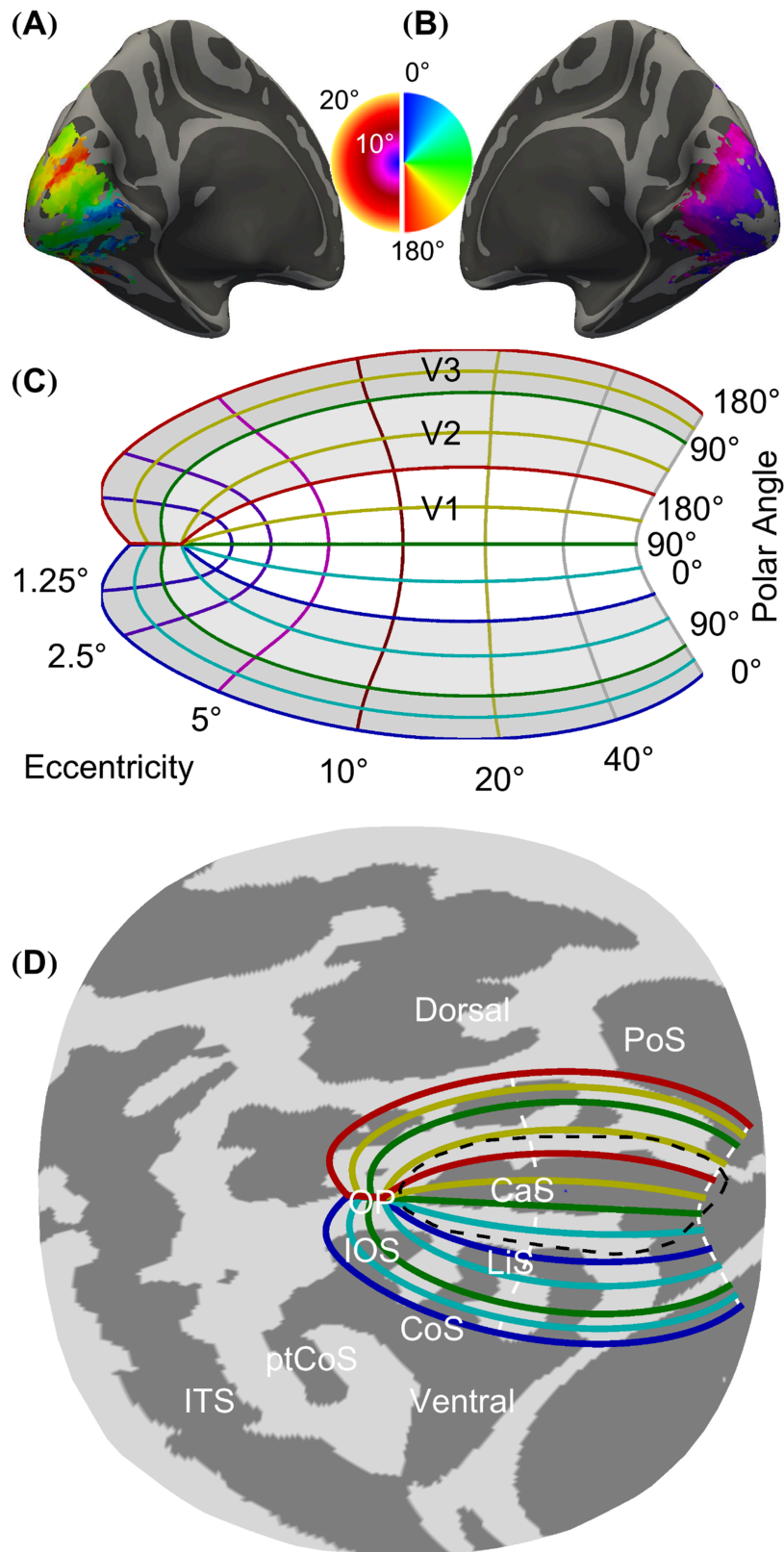


Fig.9.8.1. The Retinotopic Organization of the Visual Cortex Modeled. (C) The algebraic model of retinotopic organization. V1, V2, and V3 are colored

white, light gray, and dark gray, respectively. (Credit: NOAH BENSON, OMAR BUTT, DAVID BRAINARD, & GEOFFREY AGUIRRE. Provided by: Figshare. License: CC-BY 4.0)

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Scholarpedia, "Area V1" by Matteo Carandini, University College London, London UK

URL: http://www.scholarpedia.org/article/Area_V1

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Adapted by: Frank Wiest-Puchala Cheryl Olman PSY 3031 Detailed Outline

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CORTICAL MAGNIFICATION IN V1

Learning Objectives

Being able to describe what's cortical magnification in V1.

Know how receptive field sizes differ between foveal and peripheral vision in V1

Cortical magnification describes how many neurons in an area of the visual context are 'responsible' for processing a stimulus of a given size as a function of visual field location. In the center of the visual field, corresponding to the center of the fovea of the retina, a very large number of neurons process information from a small region of the visual field. If the same stimulus is seen in the periphery of the visual field (i.e. away from the center), it would be processed by a much smaller number of neurons. The reduction of the number of neurons per visual field area from foveal to peripheral representations is achieved in several steps along the visual pathway, starting already in the retina.

The receptive field of a visual neuron comprises a two-dimensional region in visual space whose size can range from a few minutes of arc (a dot in this page at reading distance) to tens of degrees (the entire page). The receptive field size increases at successive processing stages in the visual pathway and, at each processing stage, it increases with the distance from the point of fixation (eccentricity).

Retinal ganglion cells located at the center of vision, in the fovea, have the smallest receptive fields and those located in the visual periphery have the largest receptive fields. The large receptive field size of neurons in the visual periphery explains the poor spatial resolution of our vision outside the point of fixation (other factors are photoreceptor density and optical aberrations). To become aware of the poor spatial resolution in our retinal periphery, try to read this line of text while fixating your eyes in a single letter. The letter that you are fixating is being projected at the center of your fovea where the receptive fields of retinal ganglion cells are smallest. The letters that surround the point of fixation are being projected in the peripheral retina. You will notice that you can identify just a few letters surrounding the point of fixation and that you need to move your eyes if you want to read the entire line of text.

Visual receptive fields are sometimes described as 3-dimensional volumes in visual space to include depth in addition to planar space. However, this use of ‘receptive field’ is less common and it is usually restricted to cortical neurons whose responses are modulated by visual depth.

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Wikipedia, Cortical magnification

URL: https://en.wikipedia.org/wiki/Cortical_magnification

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Scholarpedia, “Receptive field” by Dr. Jose-Manuel Alonso & Dr. Yao Chen,
SUNY State College of Optometry

URL: http://www.scholarpedia.org/article/Receptive_field

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References:

Born, R.T., Trott, A., Hartmann, T. (2014, October 16). Cortical Magnification Plus Cortical Plasticity Equals Vision? National Center for Biotechnology Information. Retrieved April 7, 2020
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4400204/>

103.

COLUMNS AND HYPERCOLUMNS IN V₁

Learning Objectives

Know how the V₁ neurons are organized according to eye of origin

Know how the V₁ neurons are organized according to their orientation preferences (depth vs surface)

Know what V₁ hypercolumns are.

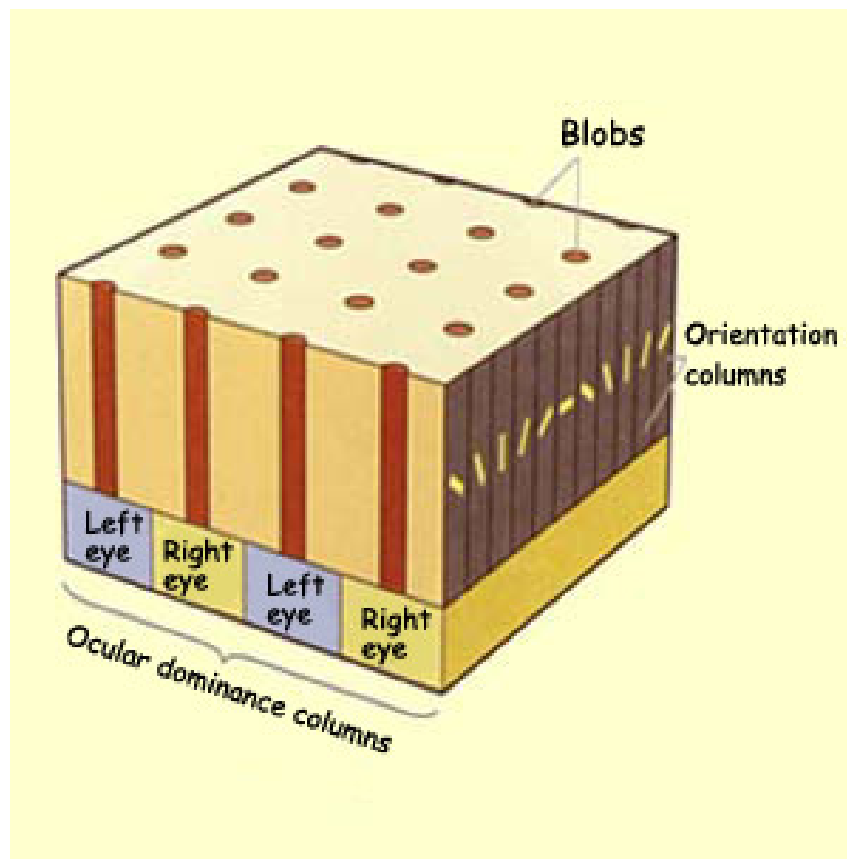


Figure 9.2. Hypercolumn diagram. Ocular dominance columns are segregated into left and right eye inputs. Orientation columns are neurons that get excited at different orientations and a cluster of these is called a pinwheel. Blobs are color selective and for every pinwheel there is a blob (credit: McGill: The Brain from Top to Bottom, Figure of hypercolumns, Copyleft <https://copyleft.org/>, https://thebrain.mcgill.ca/flash/a/a_02/a_02_cl/a_02_cl_vis/a_02_cl_vis.html. No modifications).

Columnar organization is an important property of primary sensory and motor cortices. The gray matter has 2 important directions: across the surface and through the depth.

- If you sample neural responses as you move through the depth, they stay the same (approximately). For V1,
- If you sample neural responses as you move across the surface, they change. For V1, this means that, as you move across cortex, you find neurons with different orientation preferences. This means that, as you move down through cortex, you find neurons with the same orientation preferences.

Left and right eye inputs are segregated into ocular dominance columns. This segregation is strongest in the input layer (4), so when we look for ODCs, we look in the middle of cortex. A cluster of orientation columns is called a pinwheel; there's an orientation pinwheel for each eye. Some V1 neurons are color-blind; some

are color-selective. Color-selective neurons live in blobs (regions of cortex that stain dark when you stain for cytochrome oxidase, because they're metabolically rich); there's a blob for each pinwheel.

A cortical column is a group of neurons in the cortex of the brain that can be successively penetrated by a probe inserted perpendicularly to the cortical surface, and which have nearly identical receptive fields. Neurons within a minicolumn (microcolumn) encode similar features, whereas a hypercolumn “denotes a unit containing a full set of values for any given set of receptive field parameters”.

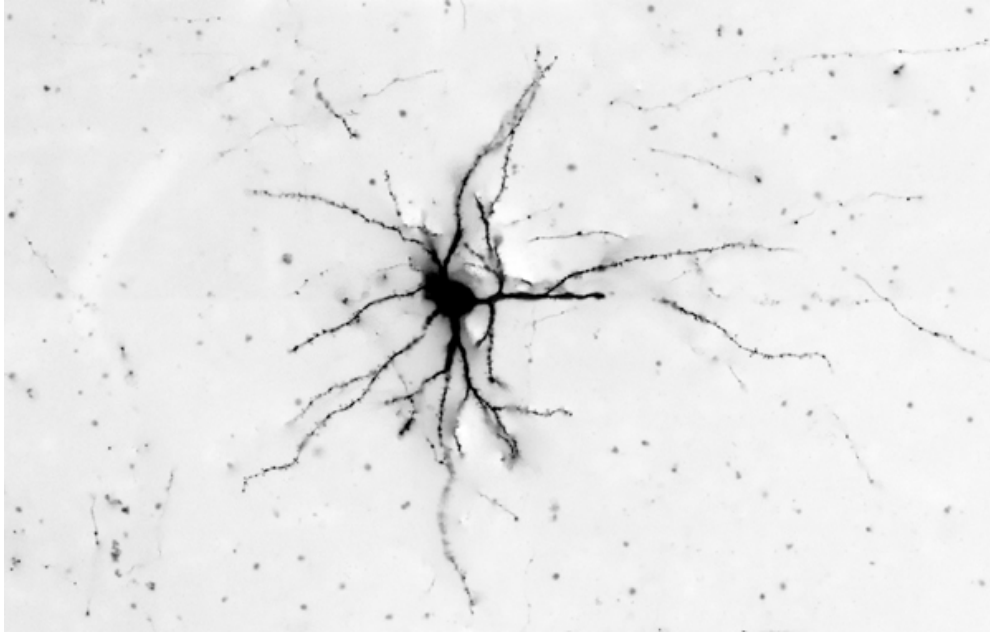


Fig.9.10.1. fluorescently tagged primary visual cortex neuron. A simple neuron from a light microscope capture. (Provided by: Webvision: The Organization of the Retina and Visual System. License:CC BY-NC)

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URL: https://en.wikipedia.org/wiki/Cortical_column

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104.

ACTIVE LEARNING EXERCISE: ORIENTATION SELECTIVITY



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SECTION X

COLOR, DEPTH, AND SIZE

For many people, color is a salient aspect of the visual world — it helps us separate objects from the background and it helps us detect danger. But not everybody can see color, and 5-10% of the population has “anomalous” color vision. This chapter discusses the uses of color, the causes of color blindness, the ways that color is encoded in the visual system, and finally, how flexible and context-dependent our perception of color is.

Size and distance are two things that are very hard to estimate for the visual system, because they are interrelated. How do you know if you’re looking at a small object, close to you, or a big object, far away? Distance is difficult for us to estimate, visually, because the 3D world is projected onto our 2D retina, and the brain has to use every clue it can find to figure out what the correct 3D interpretation of the world is.

This chapter was created by: Mohamed Ahmed, Molly Baugh, Francel Colon-Acosta, Jarod Davis, Kaelyn Dezell, Lucas Gaffney, Madelynn Gibbons, Trevor Graham, Katherine Hebig, Noah Hjelle, and Wanlin Hu

USES OF COLOR

Learning Objectives

Be able to discuss at least three uses of color

Understand the possible cause of true color blindness

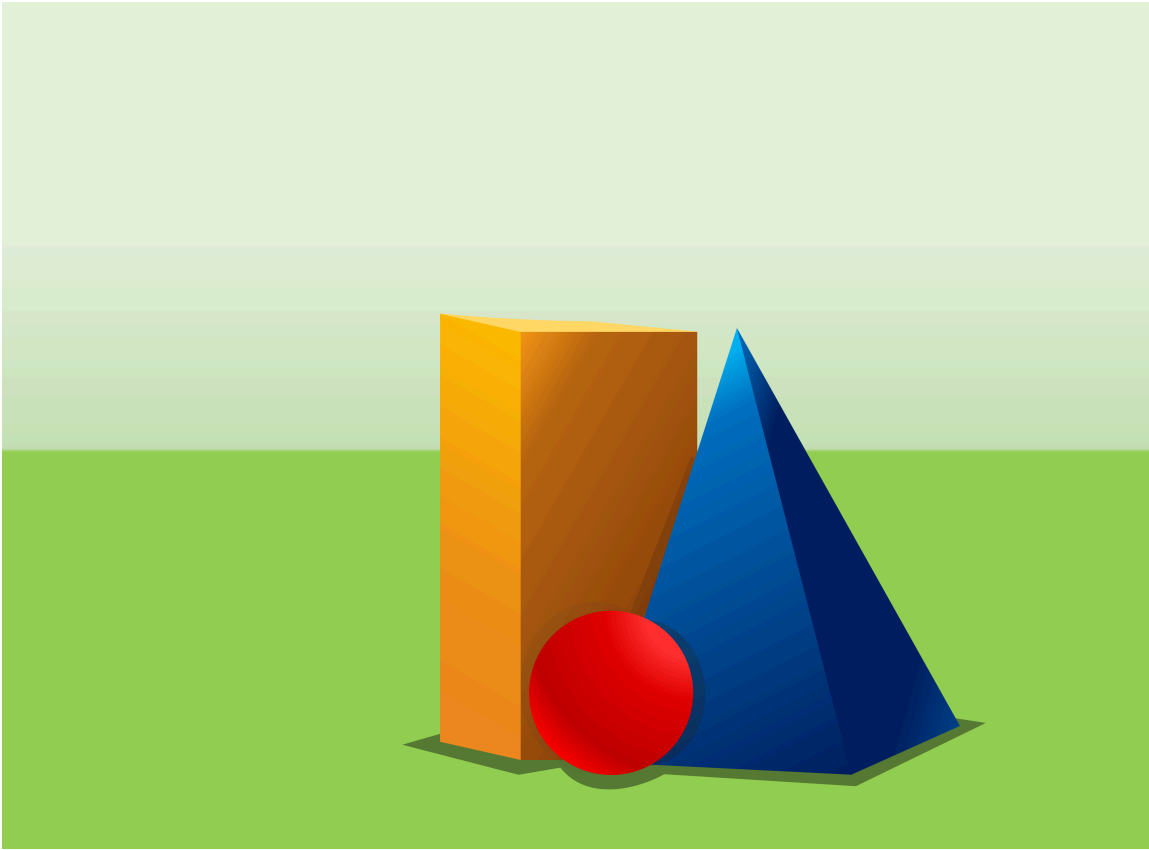
Color is a visual tool that allows us to more easily navigate the outside world. We use it in various ways every day and tend not to think twice about it. Here are a few to think about:

Object Detection and Recognition

Color allows us to notice and identify objects in a space. Some colors, like red and green, catch our attention more readily, and so are used more often when the desired outcome is your attention. Take a red ball. The ball's redness makes it easy to detect (Fig.10.1.1). Or the redness of an octagonal sign ignites recognition that it is a stop sign. It would likely not illicit a reaction to stop if one day it was blue, as we have learned to identify stop signs with red.

Scene Segmentation

Color also supports our ability to separate objects in a space. Take the red ball again. We are aware that the red ball is separate from the orange block and the blue pyramid because they are not the same color (fig.10.x.1). This difference in hue makes it easier for us to recognize the shapes as individuals and analyse them as such. Their color also allows us to separate the shapes from the green background.



10.1.1. Recognition and Segmentation. The colors in this image make it easy to separate the elements within it. The colors also help decipher what each object is based off the descriptions in the paragraph above. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

Health and Mating

Color is a primary tool used across cultures and animal species to identify the health of an individual. Someone with greenish skin may be seen as unwell, and potentially in need of medical attention. Culturally, some cultures perceive skin of a particular tones as a symbol of good health, with makeup being used in some ways to show sexual viability.

For non-human animals, the use of color not only indicates health but also acts as a tool to attract suitable mates. The peacock is one of the most common examples; the males carrying large fans of multicolored feathers.

Aesthetics

Color also helps us identify things that are pleasing to the eye. Certain color combinations succeed well in this, particularly at high saturations, evoking perceptions of beauty or perfection. Not all color combinations do this, however.



Fig.10.1.2. desirable color combinations produce aesthetically pleasing imagery. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-SA NC 4.0)

Color can help us in many other ways too, such as identifying dangerous plants and animals (A yellow and red spider is way more threatening than a light brown one); food finding (yellow bananas are normal, teal ones probably aren't edible); and space illusions (Color on a ceiling makes a room feel smaller).

Color deficiencies can have a strong influence on how a person experiences color. Often these deficiencies are confused with “color-blindness”. However, color blindness is a complete lack of color sensation. Instead, the individual sees only in grayscale. This is a rare condition characterized by nonfunctional cones, or even a complete lack of cones altogether (The person only has rods). This can come as a result of traumatic brain injuries (such as oxygen deprivation).



Fig.10.1.3. People with colorblindness do not experience color through vision. They only see in black and white/grayscale (right). (Credit: Mark Harpur. Provided by: Unsplash. Altered by: Jarod Davis. License: CC-BY)

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Adapted by: Jarod Davis

TRI-CHROMATIC VS. COLOR OPPONENT PROCESSING

Learning Objectives

- Be able to describe Trichromatic theory of vision
- Be able to describe Opponent process theory
- Understand the reconciliation in the retina

The trichromatic theory states that our cones allow us to see details in normal light conditions, as well as color. We have cones that respond preferentially, not exclusively, for red, green and blue (Svaetichin, 1955). This trichromatic theory is not new; it dates back to the early 19th century (Young, 1802; Von Helmholtz, 1867). This theory, however, does not explain the odd effect that occurs when we look at a white wall after staring at a picture for around 30 seconds. Try this: stare at the image of the flag in Fig.10.2.1. for 30 seconds and then immediately look at a sheet of white paper or a wall. According to the trichromatic theory of color vision, you should see white when you do that. Is that what you experienced? As you can see, the trichromatic theory doesn't explain the afterimage you just witnessed. This is where the opponent-process theory comes in (Hering, 1920). This theory states that our cones send information to retinal ganglion cells that respond to pairs of colors (red-green, blue-yellow, black-white). These specialized cells take information from the cones and compute the difference between the two colors—a process that explains why we cannot see reddish-green or bluish-yellow, as well as why we see afterimages. Color blindness can result from issues with the cones or retinal ganglion cells involved in color vision.

Reconciliation between these two theories lies in the retina. We have 3 kinds of photoreceptor pigments, but the circuitry of the retina combines them so ganglion cells respond along a red/green axis or along a blue/yellow axis.



Fig.10.2.1. Stare at the center of the Canadian flag for fifteen seconds. Then, shift your eyes away to a white wall or blank piece of paper. You should see an “after image” in a different color scheme. (Provided by: General Psychology. License CC-BY-NC-SA)

Here are two Audiopedia videos on trichromatic theory and opponent process theory:



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Noba project, General Psychology: An Introduction, Chapter 4: Sensation and Perception

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COLOR DEFICIENCY

Learning Objectives

Be able to describe what color deficiency is

Understand the difference between true color blindness and color deficiency

Be able to describe three types of color deficiency: Protanopia, Deuteranopia and Tritanopia

True color blindness (the lack of color sensation) is rare. It can be found in rod monochromats (people with only rods) and/or as a result of some brain injuries (color-sensitive blobs are most sensitive to oxygen deprivation). Color deficiency, however, is more common, and results from the lack of one of the cone pigments. The three different types of color deficiency include:

Protanopia: There is no L (long wavelength, or red) pigment. This makes it hard to distinguish between red and green making reds look particularly dark. This affects a few percent of the male population and a very small fraction of a percent of the female population (since the genes for the cone pigments are located on the X chromosome).

Deuteranopia: There is no M (medium wavelength, or green) pigment. This makes it hard to distinguish between red and green. It affects a few percent of the male population and a tiny fraction of a percent of the female population.

Tritanopia: There is no S (short wavelength) pigment. This makes it difficult to distinguish yellows, greens and blues. This kind of color deficiency is very rare.

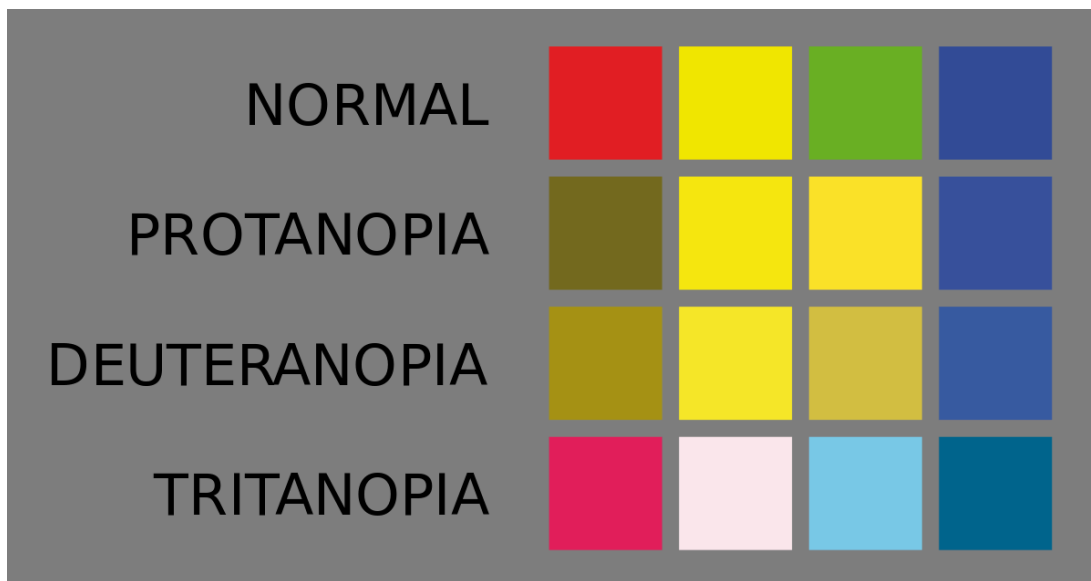


Fig.10.3.1. Different Types of Color Deficiency. This figure shows how individuals with different types of color deficiencies see colors. (Credit: Dr. Douglas Keene. Provided by: Wikimedia. License: CC-BY SA 4.0)

Watch this video to learn more about colorblindness!



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Adapted by: Katherine Hebig

SIMULTANEOUS CONTRAST

Learning Objectives

Understand the simultaneous contrast phenomenon

Be able to give an example for this phenomenon (e.g. examples from visual illusions)

Simultaneous contrast is the following phenomenon: a gray patch looks lighter when it's next to a darker patch. This demonstrates how fluid our perception of lightness really is.

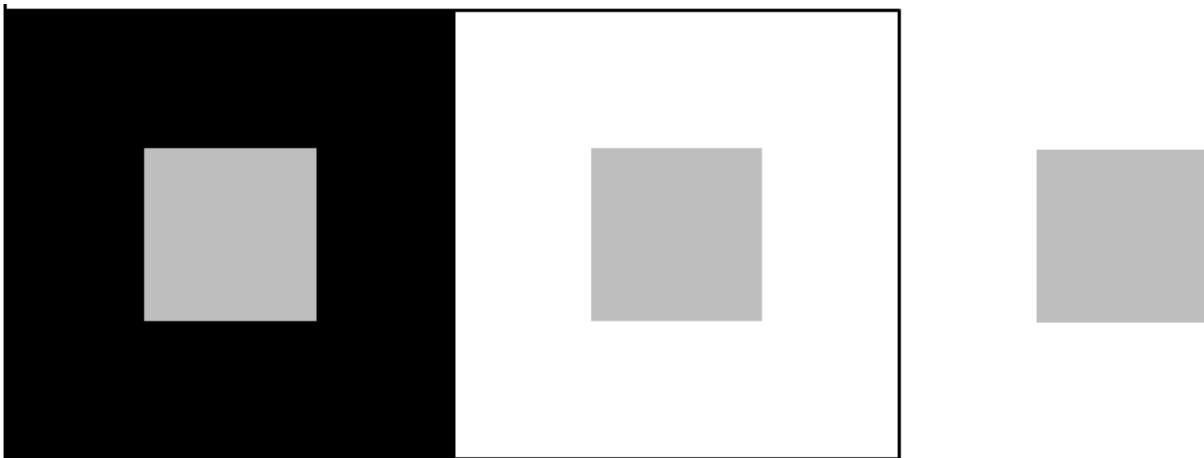


Fig.10.4.1. Simultaneous contrast. A square looks lighter when it's on a dark background. For some observers, the distant boundary might make the middle square look different from the far right square. (Credit: Cheryl A. Olman. Provided by: University of Minnesota. License: CC-BY 4.0)

Often, lateral inhibition is used as an explanation for simultaneous contrast. But White's illusion (below) shows that this is an inadequate explanation — the rectangles with their long sides against a white background look lighter than the rectangles with their long sides against a dark background. So we know that some other,

top-down effect is at play in shaping your lightness perception. There are many ways of generating context effects that create lightness illusions. What is important to remember is that our sense of lightness, brightness, and color is easily swayed by context.

Neural responses representing luminance boundaries are more credible than neural responses representing uniform patches for two reasons. First, adaptation makes it impossible to make absolute lightness judgments, so most of our perception is based on contrast and comparisons between two things. Second, the center/surround receptive field structure of our retinal and thalamic visual neurons provides weak responses to uniform fields and strong responses at boundaries. This is why we are very susceptible to lightness illusions: we know that our ability to judge lightness is weak, and we are easily swayed by what we see at boundaries and what we believe about overall scene illuminance.

A) White's illusion



B) Adding context breaks White's illusion



Fig.10.4.2. White's illusion. A) A rectangle surrounded by black bars looks darker than a rectangle surrounded by white bars. This is the opposite of simultaneous contrast. It probably happens because the rectangles 'belong' to the bars that they're on (surface grouping), so the gray bar that looks darker is dark by comparison to the white bar that it 'belongs' to. Contrast along the edges is bottom-up processing; 'belonging' is top-down processing. B) You can break White's illusion by letting the gray bars overlap, so they belong to each other. (Credit: Cheryl Olman. Provided by: University of Minnesota. License: CC-BY 4.0)

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COLOR AND LUMINANCE CONSTANCY

Learning Objectives

Be able to explain the main three things that contribute to our ability to experience an object as being the same color

Understand the differences/similarities between color and lightness constancy

There are three main aspects of vision that allow the consistent perception of color in objects independent of illumination:

1. Chromatic adaptation – when the light source contains a disproportionate amount of light in one section of the spectrum, the responses of the relevant photoreceptors are suppressed, shifting our perception away from the dominant wavelength.
2. Local context – we compare all the colors in a scene to normalize our perception. This process takes place at a conscious and unconscious level.
3. Prior knowledge – certain objects characterized by a distinct color will be perceived as this color despite differential illumination.

Light constancy concerns the relative reflectance of an object independent of illumination. Color constancy deals with the perception of color independent of illumination.

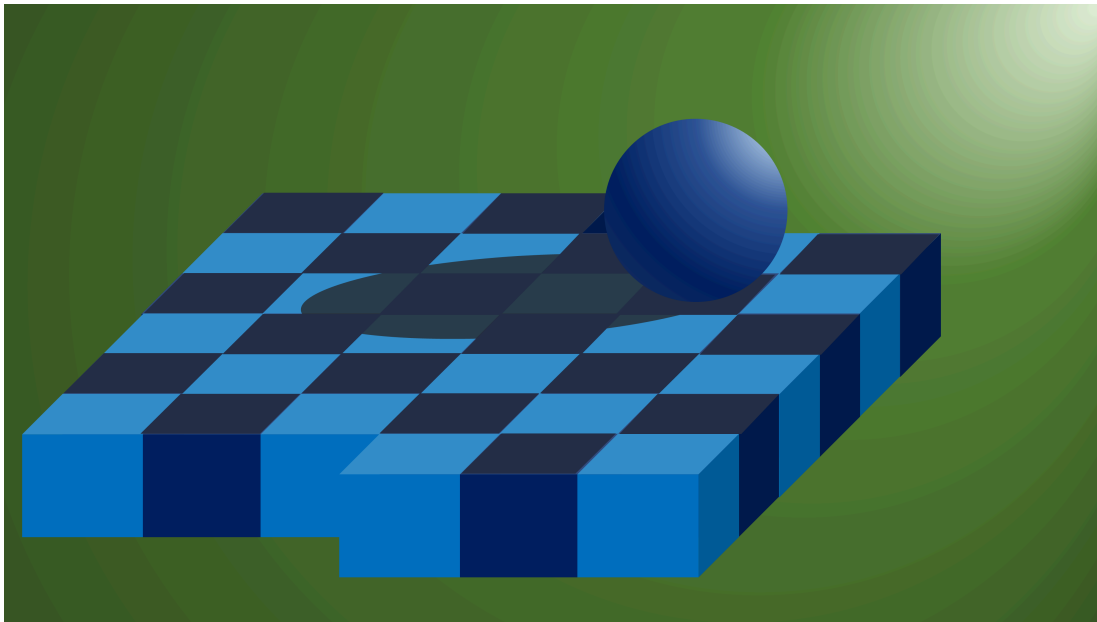


Fig.10.5.1. Color Consistency. Image shows how squares with the same luminance appear to be different colors due to environmental shadows (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY SA 4.0)

To learn more, take a look at this video about color and light constancy with great examples!

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Adapted by: Trevor Graham

110.

OCULOMOTOR AND MONOCULAR DEPTH CUES

Learning Objectives

Understand what oculomotor depth cues are

Be able to briefly describe what oculomotor muscles and ciliary muscles do

Be able to list and explain monocular depth cues (at least 6)

Oculomotor depth cues are proprioceptive information from oculomotor muscles and ciliary muscles. Oculomotor muscles are the muscles that rotate the eyeballs for them to converge at a depth (fig.10.6.1). Ciliary muscles are the muscles that change the focal length by compressing the lens of the eye.

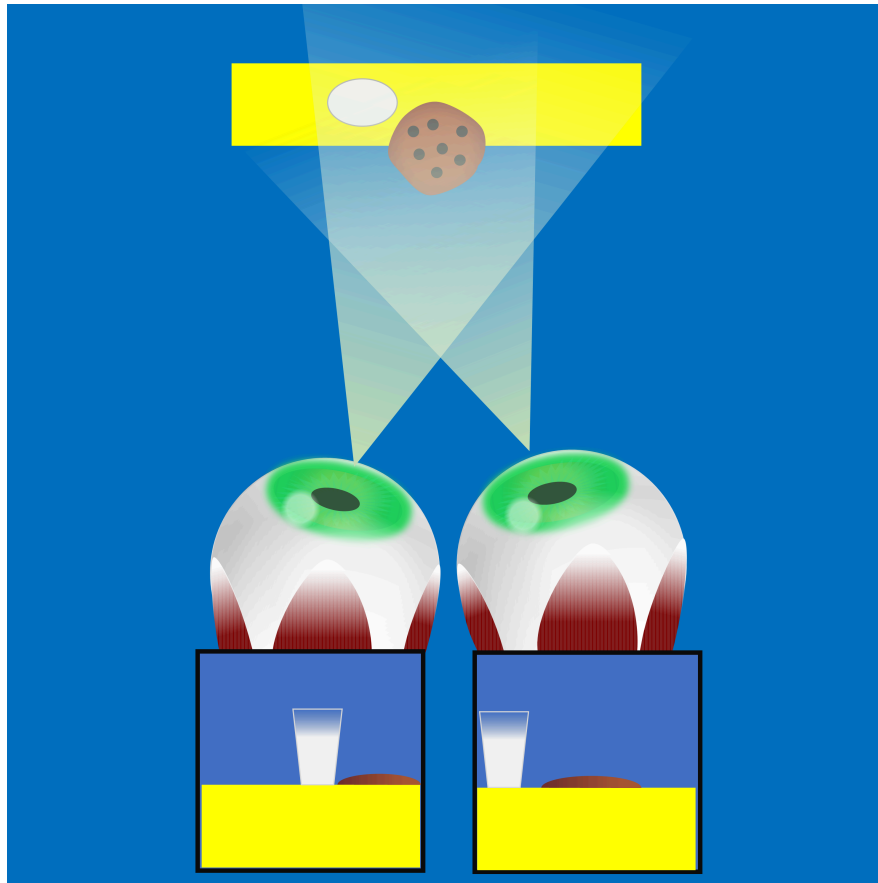


Fig. 10.6.1. *Oculomotor Depth Cues*. These two eyes have been adjusted by their oculomotor muscles to provide depth cues about the cookie that's about to fall off the table! (Credit: Jarod Davis Provided by: University of Minnesota. License CC-BY-NC-ND)

Monocular depth cues are depth cues that are able to be perceived without both eyes. Some monocular depth cues include, but are not limited to:

1. Relative Height: Things at a distance look like their base is higher
2. Relative Size: Objects farther away from other objects are smaller (fig.10.6.2)
3. Occlusion: Things will get in front of other things
4. Shadows: Relative height and depth
5. Texture Gradient: Textures look finer as they draw back
6. Atmospheric Perspective: Things that are far away look hazy or out of focus

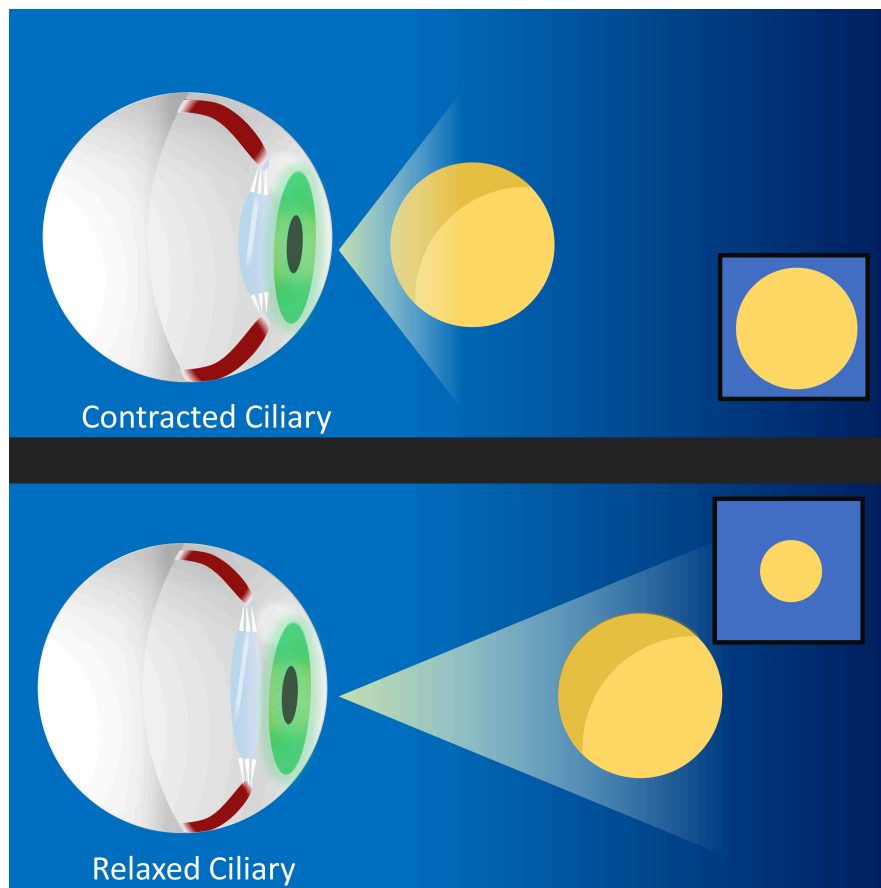


Fig.10.6.2. *Monocular Depth Cues*. The ciliary muscles of this eye provide depth cues based on relative size of the ball. (Credit: Jarod Davis Provided by: University of Minnesota. License CC-BY-4.0)

Take a peak at these websites to learn more about monocular and binocular oculomotor cues.

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Adapted by: Madelynn Gibbons

111.

STEREO DEPTH CUES

Learning Objectives

Be able to describe the concepts: disparity, vergence and accommodation

Understand what horopter is

Understand the disparity neurons in primary visual cortex

Stereo depth cues or binocular depth cues are when the photoreceptors or movements of both eyes are required for depth perception. Our ability to perceive spatial relationships in three-dimensions is known as depth perception. With depth perception, we can describe things as being in front, behind, above, or to the side of other things. We use a variety of cues in our visual field to establish our sense of depth. Some of these are binocular cues are disparity, vergence, and accommodation. An example of a depth cue is binocular disparity, the slightly different view of the world that each of our eyes receives. Vergence refers to the equal movements of the eyes to a certain point. Accommodation refers to the focus of your eyes on distant or near objects.

Accommodation and vergence usually match: your eyes are verged and focused on the thing you're looking at, and that point defines the horopter. The horopter is an imaginary circle drawn through the point where the eyes are converged on and back to the eyes, which traces out the location of all objects in the three-dimensional visual field that will land on the retinae with zero disparity. Things that are closer to you than the horopter have negative (crossed) disparity and things beyond the horopter have positive disparity. In the primary visual cortex (V1), disparity neurons are tuned to the general area of images of a similar object on every retina. Some neurons are tuned to near; some to far.

In this video, you can learn more about how accommodation works.



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AMBLYOPIA AND STRABISMUS

Learning Objectives

Understand what strabismus and amblyopia are

Understand the results of growing up with these conditions

Strabismus is a disorder in which both eyes do not line up in the same direction. Therefore, they do not look at the same object at the same time. The most common form of strabismus is known as “crossed eyes.” The oculomotor muscles around each eye allow for both eyes to focus on the same object. When someone has strabismus, these muscles do not work together which causes the eyes to look in different directions. When this occurs, two different images are sent to the brain — one from each eye. This confuses the brain. In children, the brain may learn to ignore or suppress the image from the weaker eye. If the strabismus is not treated by training or surgery, the eye that the brain ignores will never see well and the brain will rely solely on the image from the stronger eye. This loss of vision in one eye is called amblyopia. In most children with strabismus, the cause is unknown and the problem is present at or shortly after birth.

Amblyopia, also called lazy eye, is a type of poor vision that happens in just one eye. It can result from strabismus or anisometropia. Amblyopia develops when there’s a breakdown in how the brain and the eye work together, and the brain can’t recognize the sight from one eye. Normally, the brain uses nerve signals from both eyes to see. But if an eye condition makes vision in one eye worse, the brain may try to work around it. It starts to “turn off” signals from the weaker eye and rely solely on signals from the stronger eye. This creates monocular visions instead of binocular. Amblyopia starts in childhood, and it’s the most common cause of vision loss in kids. However, early treatment works well and usually prevents long-term vision problems. If caught and treated early, stereo vision can develop normally (Fig.10.8.1).



Fig.10.8.1. Treatment for Amblyopia. This figure shows a child wearing an eye patch attempting to cure amblyopia. Using an eye patch helps to strengthen the non-dominant eye and forces the brain to receive images from that visual field.
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A.D.A.M. Inc., MEDLINE, Strabismus

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URL: <https://medlineplus.gov/ency/article/001004.htm>

License: PDMNational Eye Institute, Amblyopia (Lazy Eye)

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URL: <https://www.nei.nih.gov/learn-about-eye-health/eye-conditions-and-diseases/amblyopia-lazy-eye>

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113.

BINOCULAR RIVALRY

Learning Objectives

Be able to describe what binocular rivalry is

Be able to give an example under what conditions binocular rivalry may happen

Binocular Rivalry is a visual phenomenon characterized by oscillation between two different perceptions of a constant stimulus (Bradley, 2012). This can happen through (a) one image being read by both eyes, or (b) two different things being read by either eye.

The first instance (a) is often associated with optical illusions; “Does Figure X below say Green, or does it say Blue?” Images like these can be interpreted as two different things. The mind jumps between one or the other as we can only consciously attend to one of these interpretations at a time (Carter 2006). The interpretation that you are focusing on at the time is considered the dominant perception, while the other is, temporarily, the suppressed perception (Carter, 2006).

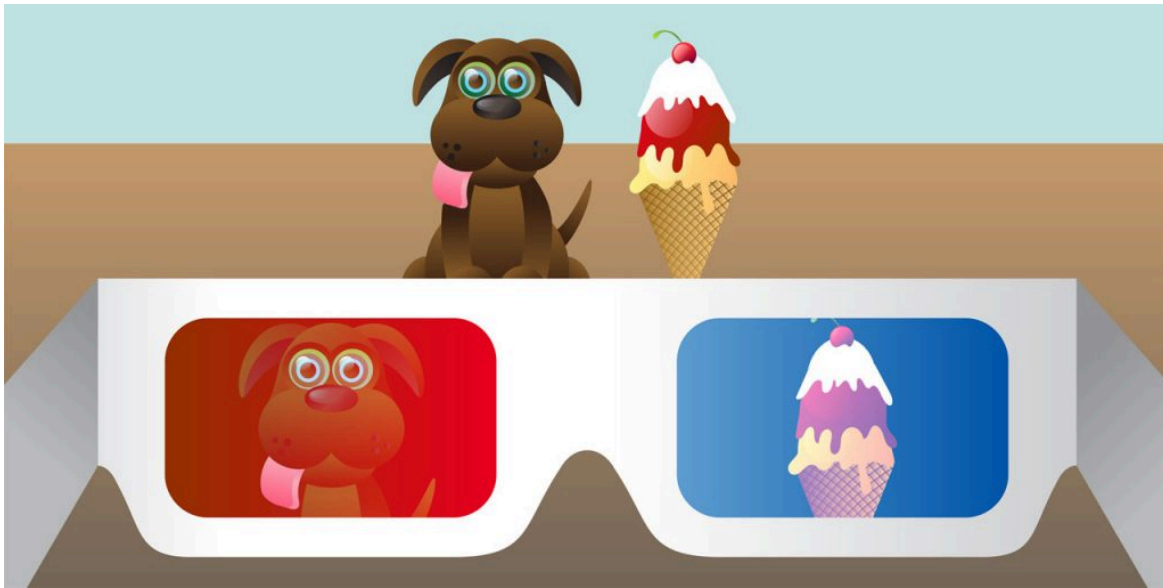


Fig. 10.9.2 This is an image of a dog and ice cream cone viewed through special glasses. The person using these special glasses would see the image of the ice cream cone and dog switching back and forth, rather than a red dog and blue ice cream cone. This is called binocular rivalry because the two (“bi”) different inputs to the eyes (“ocular”) compete (or, “rival”) for dominance. (Credit: Rajiv Madipakkam A, Ludwig K, Rothkirch M and Hesselmann G (2015) Now You See it, Now You Don’t: Interacting with Invisible Objects. CC-BY 3.0).

The second instance of binocular rivalry (b) is where 2 different things are read by either eye. This is often experienced when looking out of the corner of one eye. When you do this, you are able to “see through” your nose. This is because the two images from both eyes are combined. As one image is interpreted as dominant, it precedes over the other image; hence your nose appears as a ghostly outline.

Authored by: Jarod Davis

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References:

Binocular Rivalry, Olivia Carter, 2006. <http://visionlab.harvard.edu/Members/Olivia/tutorialsDemos/Binocular%20Rivalry%20Tutorial.pdf>

Binocular rivalry for beginners, Bradley N. Jack, 2012. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485850/>

STEREO DISPLAYS

Learning Objectives

Understand how 3D stereo stimuli are created

Be able to explain why some people cannot see 3D displays

Stereo Displays are 3D displays that are able to transmit depth perception to a person's vision. The 3D stereo stimuli is when there are separate images presented to a person's left and right eye in order to recreate a normal visual disparity when viewing an image with both eyes. Most stereo display systems do require the observer to wear glasses. One example is polarized glasses which are used in theaters such as 3D Imax. These glasses have filters that isolate the left and right eyes much like the red/blue filters but retain all the color integrity of the images. Another example is anaglyph glasses which uses color filters for isolating the left and right eye images.

About 5% of people cannot see 3D effects at all because they do not have stereo vision. If someone is unable to see the 3D effect at all, they may be stereo-blind. They are someone who is flat-viewer or lacks depth perception. The most common reasons for stereo-blindness can be medical disorders that prevent the eyes focusing and/or aligning correctly or loss of vision in one eye.

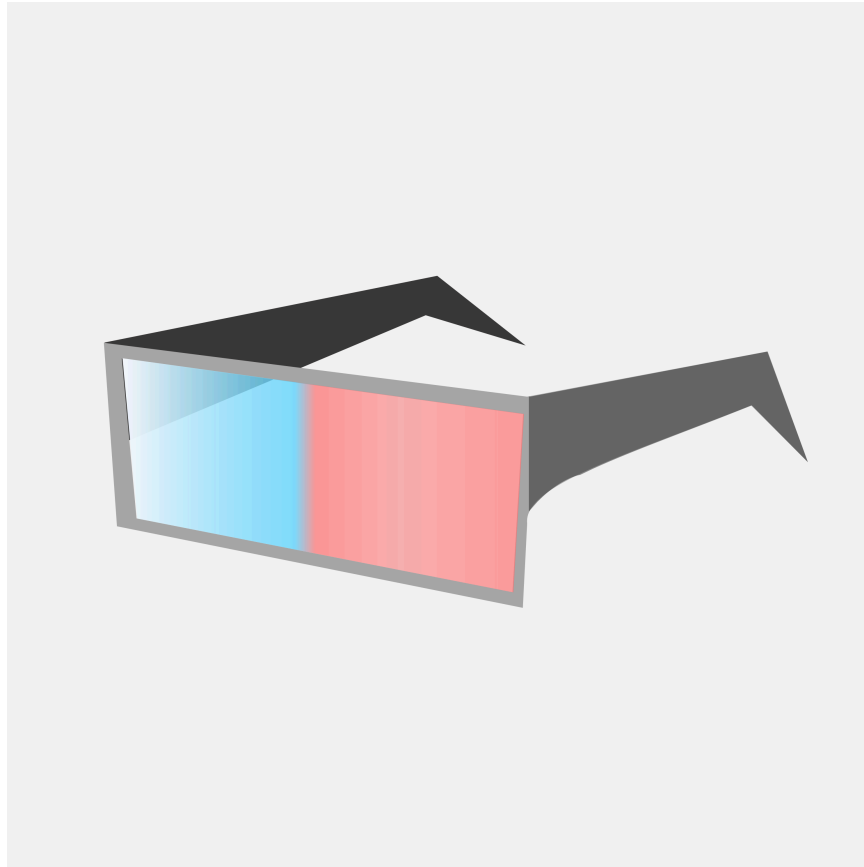


Fig.10.10.1. 3D Glasses. 3D Glasses with red and blue lenses filter two different images through either eye for a “pop out affect” when the images are combined in the brain. (credit: Jarod Davis. Provided by: University of Minnesota. License CC-BY-NC-ND)

Learn more about stereoblindness by watching the video linked here and included below.



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References:

Owen, D. (2010, September 1). Stereo-Blind: People who can't see 3D. Retrieved from

<https://www.mediacollege.com/3d/depth-perception/stereoblind.html>

Stereo basics. (2001).WorldViz. Retrieved from

<https://docs.worldviz.com/vizard/latest/>

index.htm#Frequently_asked_questions.htm%3FTocPath%3DReference%7C_____29

115.

SIZE/DISTANCE RELATIONSHIPS

Learning Objectives

Understand what Emmert's law is

Understand the concept of after-image in the context of size-distance relationship

Emmert's law demonstrates how retinal size is determined by a combination of object size and viewing distance. If you look at a bright object on a dark background (or vice versa), then close your eyes, you see a ghostly after-image. If you then look at a screen close to you, it looks like a relatively small ghost. But if you look at a screen far away, it looks like a big ghost.

After-image is a visual illusion causing retinal impressions to retain a phantom-like image of a previously present stimulus. The afterimage can appear to be “positive” or similar in brightness and/or color to the original image, or “negative” which would show colors complementary and less bright than that of the original image. A common example of an afterimage is the square of light one might see after a camera flashes. Afterimage is believed to be caused by continued activity in the visual system after a stimulus has been removed.

It's actually really hard to guess how far away a visual object is. As with many other visual problems, our visual system usually solves this one easily. However, images close to the eye and far from the eye arrive in the same place in the retina.

Our brains have learned that retinal size is not an indication of actual object size, so we're always reaching for other cues to figure out how big something is.



Fig.10.11.1. Size and distance. This mother and her child look closer than the people farther up the stairs.
(Credit: Dmitry Anikin. Provided by: Unsplash. License: CC-BY-4.0)

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Encyclopaedia Britannica, Afterimage

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SIZE ILLUSIONS

Learning Objectives

Understand the concept of retinal size and why it is not reliable for perceiving size

Be able to give at least 2 examples where size perception is strongly rely on depth cues (e.g. Ponzo illusion)

Emmert's law demonstrates how retinal size is determined by a combination of object size and viewing distance. It's actually really hard to guess how far away a visual object is. As with many other visual problems, our visual system usually solves this one easily. However, images close to the eye and far from the eye arrive in the same place in the retina.

Our brains have learned that retinal size is not an indication of actual object size, so we're always reaching for other cues to figure out how big something is.

The Ames room and Ponzo illusion demonstrate situations in which size perception breaks down because depth cues are strong.

An Ames room is a distorted room used to create an optical illusion of relative sizes (fig.10.12.1). Upon viewing people or objects within an Ames room, there is a loss of normal perspective. As a result of the optical illusion created by the distorted room, a person standing in one corner appears to the observer to be significantly larger than a person standing in the opposite corner while the room appears to be a normal rectangular shape. This is taken to indicate the significant role past experience has on our interpretation of our perceived world.

The Ponzo illusion is an optical illusion where a pair of converging lines distorts the perception of two identically sized lines. Like most visual and perceptual illusions, the Ponzo illusion helps neuroscientists study the way the brain and visual system perceive and interpret images. Artists have also utilized the illusion to great effect in their works.

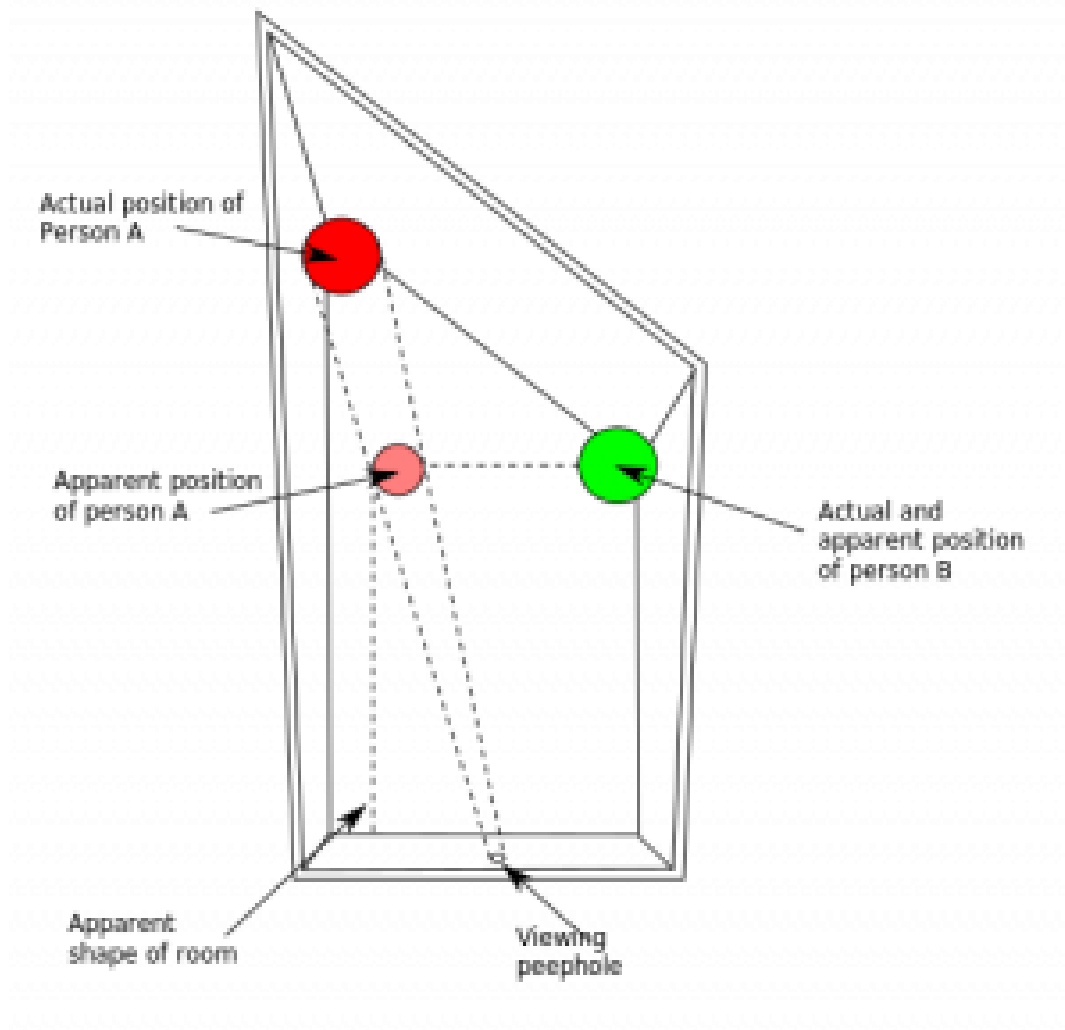


Fig.10.12.1. Ames room. This is an example of how a distorted room creates an optical illusion. (Provided by: The New World Encyclopedia. License: CC-BY-SA.)

Learn more about the Ames Illusion by watching this video!



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New world encyclopedia, Ames Room

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117.

ACTIVE LEARNING EXERCISES: INFERENCE GRAPHS



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SECTION XI

VISUAL DEVELOPMENT AND OBJECT RECOGNITION

In recent years, computer algorithms have started catching up to human observers' skill at recognizing objects, which is to say, correctly categorizing parts of an image according to uses or identities. But object definitions are not clear cut; they tend to be rather operational (defined by the utility of the situation). This chapter discusses the development of the visual system — what is the normal trajectory for developing visual skills — and the types of experiments that visual scientists have done to try to define objects and characterize object recognition skills.

This chapter was created by: Jaclyn Bowen, Tessa Rossini, Hannaan Shire, John Taylor, Hannah Thormodsgaard, Ashlynn VanHorn, Jamie Wahout, Ariyanna Watts, and Elton Wong.

118.

INFANT ACUITY

Learning Objectives

Being able to describe the developmental changes of visual acuity in humans

Know what are the causes of the developmental changes of visual acuity

Being able to describe the developmental changes of contrast sensitivity function and hyperacuity

Developmental Changes of Visual Acuity in Humans

The womb is a dark environment void of visual stimulation. Consequently, vision is the most poorly developed sense at birth and time is needed to build those neural pathways between the eye and the brain. Newborn visual acuity is about 20/400, which means that an infant can see something at 20 feet that an adult with normal vision could see at 400 feet. Thus, the world probably looks blurry to young infants.

The visual system is basically functional by 1 year of age. Depth perception begins at 3 months (evidence from vergence — eyes track objects as they get closer, and stereo vision). Infants understand that closer things are larger at about 7 months.

Causes of Developmental Changes of Visual Acuity

The primary cause of poor visual acuity is that humans are born with underdeveloped neurons:

- Retinal photoreceptors have big inner segments and small outer segments, so visual information is undersampled.
- Some sources say color vision is normal; some say it isn't.
- Cortical neurons have relatively sparse connections. The first 6 months of development witnesses a great elaboration of connections (note neural density is roughly the same, but connections — axons &

dendrites — are much richer).

The Developmental Changes of Contrast Sensitivity Function and Hyperacuity

Many aspects of vision continue to change for the first several years of life. The contrast sensitivity function (detection threshold as a function of spatial frequency) makes good progress toward “normal” (peak sensitivity: 4 cycles/degree) during the first several years of life, but does not reach adult form until about 10 years old. Hyperacuity (the ability to perform better on a Vernier acuity task than predicted by normal visual acuity) develops after about 10 years. This skill requires an extrapolation of straight lines to achieve hyper-resolution.



Fig.11.1.1. Baby Eyes. Babies are legally blind when they are born. (Credit: Damir Spanic. Provided by: Unsplash. License: CC-BY-4.0.)

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DEVELOPMENT OF OBJECT VISION

Learning Objectives

Know that infants have full color vision and face perception at birth

Being able to describe the developmental changes of depth perception and object perception

Vision is the most poorly developed sense at birth. Newborns typically cannot see further than 8 to 16 inches away from their faces (which is about the distance from the newborn's face to the mother/caregiver when an infant is breastfeeding/bottle-feeding). When viewing a person's face, newborns do not look at the eyes the way adults do; rather, they tend to look at the chin – a less detailed part of the face.

Newborns have difficulty distinguishing between colors, but within a few months they are able to discriminate between colors as well as adults do. Due to their poor visual acuity, infants look longer at checkerboards with larger squares rather than the boards with many smaller squares. This behavior can actually be observed in a lab setting through eye tracking experiments. Eye tracking is when an observer tracks the gaze of an individual and is sometimes accompanied by measuring the amount of time during which that individual spends looking at one visual stimulus versus another. Thus, toys for infants are sometimes manufactured with black and white patterns rather than pastel colors because the higher contrast between black and white makes the pattern more visible to the immature visual system (Fig.11.2.1).

By 2 or 3 months, they will seek more detail when exploring an object visually and begin showing preferences for unusual images over familiar ones, for patterns over solids, for faces over patterns, and for three-dimensional objects over flat images. Sensitivity to binocular depth cues, which require inputs from both eyes, is evident by about 3 months and continues to develop during the first 6 months. By 6 months, the infant can perceive depth perception in pictures as well (Sen, Yonas, & Knill, 2001). Infants who have experience crawling and exploring will pay greater attention to visual cues of depth and modify their actions accordingly (Berk, 2007).



Fig. 11.2.1. Color and Patterns in Infant Toys. An infant is playing with a toy with different patterns and colors, where the black and white pattern on the toy is most likely more visible to the infant due to color contrast. (Provided by: Pixabay. License: CC-BY-4.0.)

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120.

NEUROIMAGING

Learning Objectives

Know what are the four main neuroimaging methods (PET, EEG, MEG, fMRI)

Know how these four methods work

Know what are the pros and cons of using different methods.

Neuroimaging exists as a way for scientists to monitor brain activity. There are four major neuroimaging methods that are commonly used by scientists; Positron Emission tomography (PET), Electroencephalography (EEG), Functional magnetic imaging (fMRI) and Magnetoencephalogram (MEG).

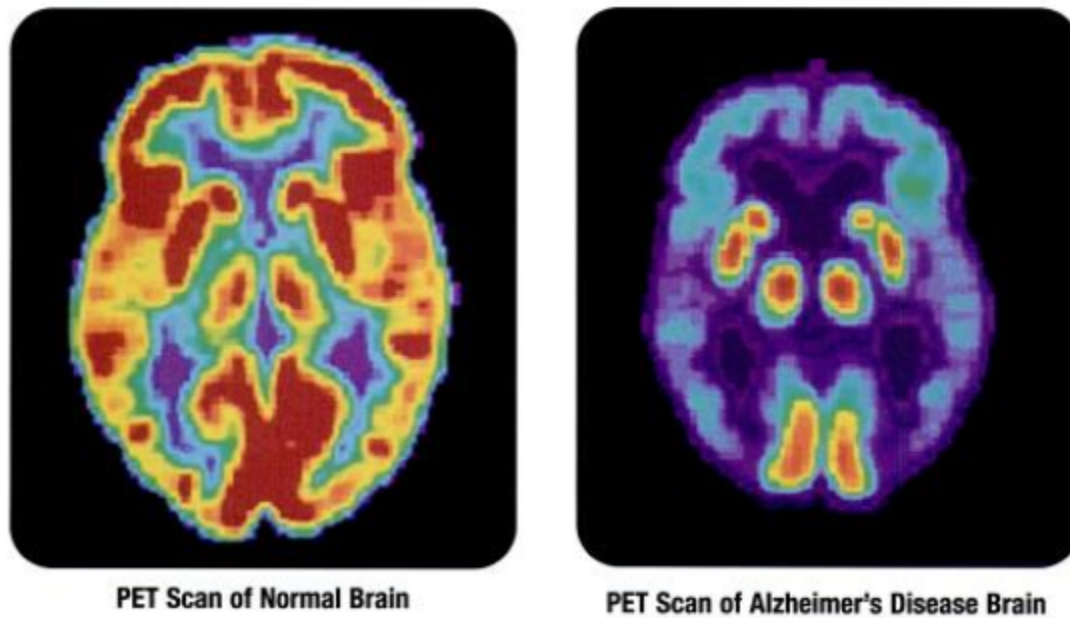


Fig. 11.3.2. An example of PET scan of a normal brain and an Alzheimer's disease brain. (Provided by Wikimedia Commons. License: CC Zero)

Positron emission tomography (PET) scans create pictures of the living, active brain. An individual receiving a PET scan drinks or is injected with a mildly radioactive substance, called a tracer. Once in the bloodstream, the amount of tracer in any given region of the brain can be monitored. As brain areas become more active, more blood flows to that area. A computer monitors the movement of the tracer and creates a rough map of active and inactive areas of the brain during a given behavior (Fig.11.3.1).

In some situations, it is helpful to gain an understanding of the overall activity of a person's brain, without needing information on the actual location of the activity. Electroencephalography (EEG) serves this purpose by providing a measure of a brain's electrical activity. An array of electrodes is placed around a person's head. The signals received by the electrodes result in a printout of the electrical activity of his or her brain, or brainwaves, showing both the frequency (number of waves per second) and amplitude (height) of the recorded brainwaves, with an accuracy within milliseconds. Such information is especially helpful to researchers studying sleep patterns among individuals with sleep disorders.

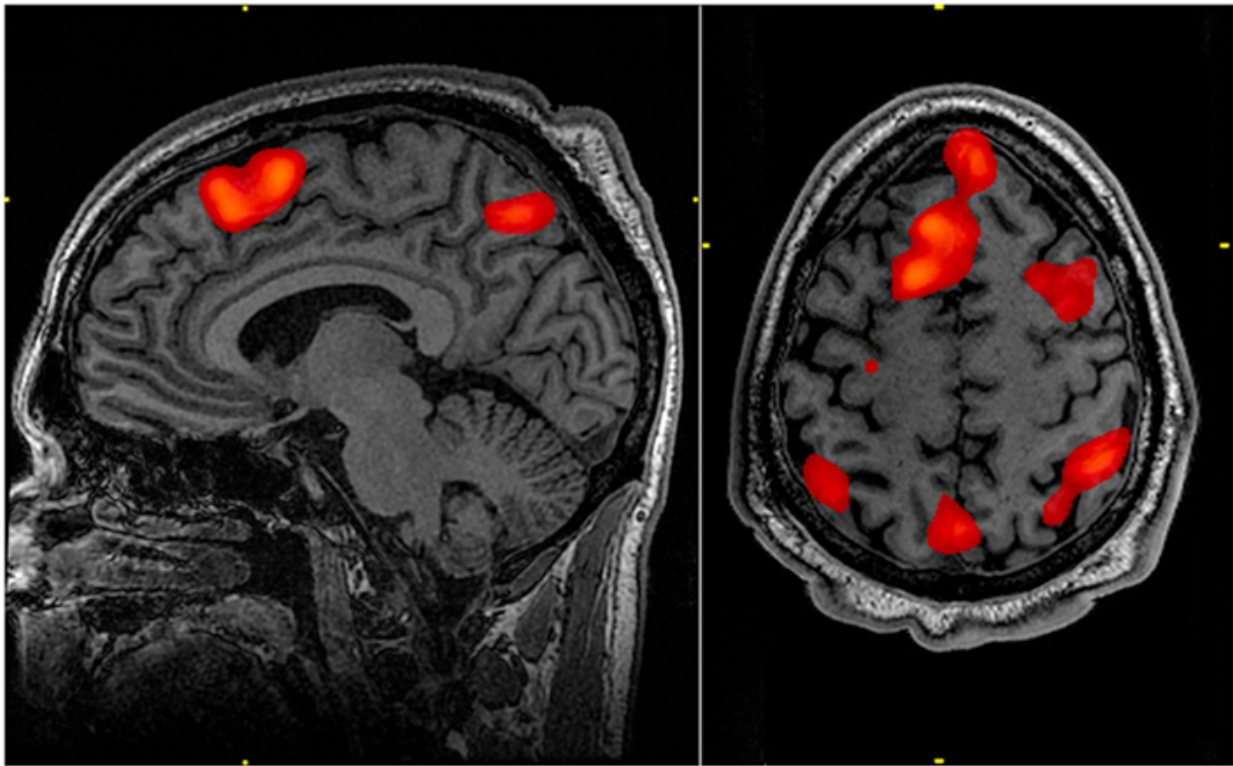


Fig. 11.3.1. An example of a fMRI brain scan during working memory tasks. (Provided by Wikimedia Commons. License: CC Zero.)

Functional magnetic resonance imaging (fMRI) operates on the same principles, but it shows changes in brain activity over time by tracking blood flow and oxygen levels (Fig.11.3.1).

Magnetoencephalography (MEG) is another technique for noninvasively measuring neural activity. The flow of electrical charge (the current) associated with neural activity produces very weak magnetic fields that can be detected by sensors placed near the participant's scalp (Fig.11.3.1). The number of sensors used varies from a few to several hundred. Due to the fact that the magnetic fields of interest are so small, special rooms that are shielded from magnetic fields in the environment are needed in order to avoid contamination of the signal being measured.

There are many pros and cons to using each method. The fMRI provides more detailed images of the brain's structure, as well as better accuracy in time, than is possible in PET scans. The fMRI also provides more detailed images of the brain's structure, as well as better accuracy in time, than is possible in PET scans. One major advantage of EEG is its temporal resolution. Data can be recorded thousands of times per second, allowing researchers to document events that happen in less than a millisecond. MEG analytic strategies are nearly identical to those used in EEG. However, the MEG recording apparatus is much more expensive than EEG, so MEG is much less widely available.

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SPECIALIZED VISUAL AREAS

Learning Objectives

Know what dorsal and ventral pathways in visual cortex are.

Know what are the differences between the dorsal and ventral pathways (anatomical, functional).

Being able to list some brain regions in both pathways.

Complex computations relating to vision, those that eventually allow you to have a visual experience of the world, all happen in the cortex. The first stop in the cortex is at the primary visual cortex (also known as V1). Here, the “reconstruction” process begins in earnest: based on the contrast information arriving from the eyes, neurons will start computing information about color and simple lines, detecting various orientations and thicknesses. Small-scale motion signals are also computed (Hubel & Wiesel, 1962). The computation of motion and aspects like color, are what give rise to the ventral and dorsal pathways of vision.

Visual-recognition areas are located along the ventral pathway of the brain, and terminate around the temporal lobe (the What pathway). Information that is funneled through this pathway is focused on identifying what a specific object is. Identification relies on multiple brain structures. The fusiform face area specializes in identifying objects for which fine discriminations are required, like faces. There is even a brain region specialized in letter and word processing, called with visual word form area. These are just a few important brain regions in the ventral pathway of vision in the brain.

Brain regions along the dorsal pathway, near the parietal lobe (Fig.11.4.1), (or Where-and-How pathway) will compute information about self- and object-motion, allowing you to interact with objects, navigate the environment, and avoid obstacles (Goodale and Milner, 1992). Some major structures in this pathway include the medial superior temporal region and basic parietal regions, both of which play a major role in determining optic flow.

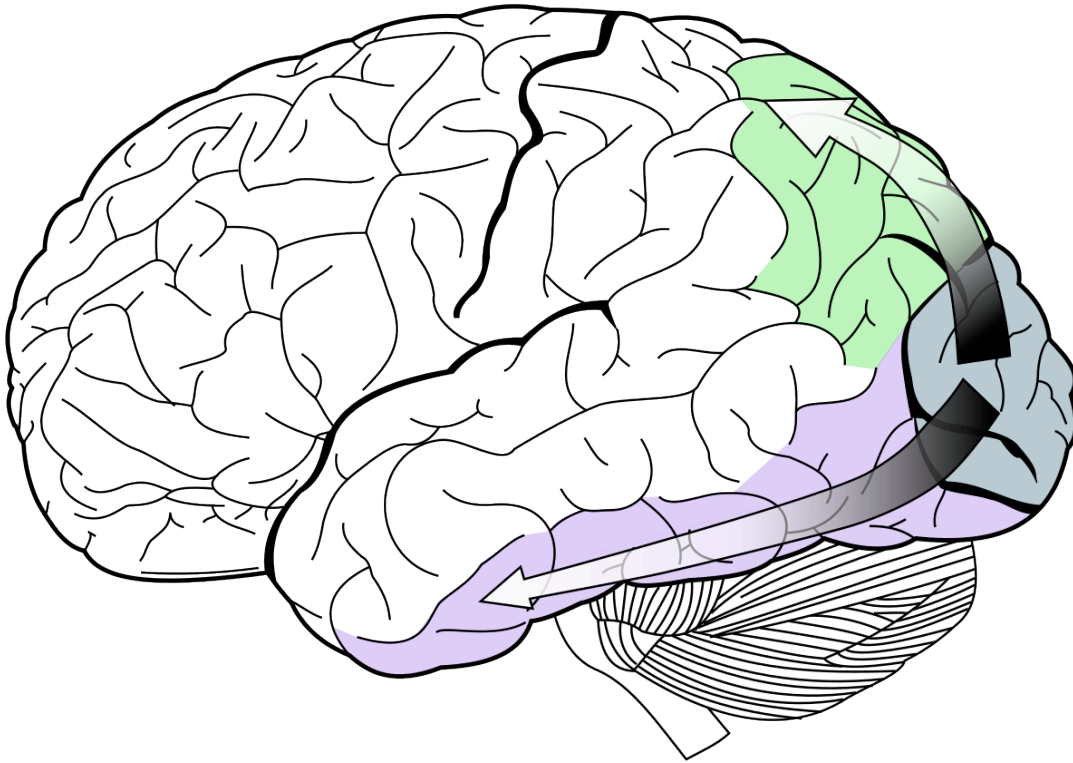


Fig. 11.4.1. Brain Areas for Dorsal and Ventral Streams of Vision. The dorsal stream (green) and ventral stream (purple) are shown. They originate from a common source in the visual cortex. (Credit: Selket. Provided by: Wikimedia Commons. License: CC-BY-SA 3.0.)

Exercises

1. Which answer fills in the blanks the best? The _____ pathway (also known as the Where-and-How pathway) will compute information about self- and object- motion to allow you to navigate and interact with the environment while the _____ pathway (also known as the What pathway) will compute information to identify what a specific object is.
A. Ventral; Dorsal
B. MT; MST
C. Dorsal; Ventral

D. MST; MT

Answer: C

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122.

MOTION PROCESSING: MT AND MST

Learning Objectives

Know where are MT (medial temporal area) and MST (medial superior temporal area)

Know the functions of these two areas.

Know which pathway (dorsal or ventral) do these two areas belong to

The medial temporal (MT) and medial superior temporal (MST) areas are examples of the specialized visual areas in the brain. Specifically, MT and MST have the function of detection of motion in the brain. These medial temporal areas reside toward the bottom of the brain, in the dorsal stream of the visual cortex, and play a role in both visual perception and visual memory. (Zu, Khan)



Fig.11.5.1. An individual with a lesion affecting their MT would be unable to perceive motion. For example a moving ceiling fan would appear as stagnant frames. (Credit: WxMom. Provided by: Flickr. License: CC-BY-NC-SA 2.0)

The “motion pathway” is believed to be the stream in which a special role of representing motion signals takes place, beginning with the parasol cells in the retina and continuing through cortical areas V1 and to MT. MT is known to be the area which receives the input from direction selective neurons in area V1. Direction selective basically means that they have a strong response when an object or field of random dots move in one direction, but they respond little to the other direction. The neurons in MT detect coherent motion in patches. That info from MT is then sent to MST to help put together coherent motion from around the scene to detect optic flow. (Wandell, Brian)

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123.

FFA AND VWFA

Learning Objectives

Know where are FFA (fusiform face area) and VWFA (visual word form area)

Know the functions of these two areas.

Know which pathway (dorsal or ventral) do these two areas belong to

While we memorize a few regions as examples of specialized functions, it is more important to remember that every visual stimulus is processed by every visual region. Neurons selective to particular aspects of an image will be most responsive. Visual experience is the sum of all of these responses, and these regions provide simultaneous, multi-dimensional representations of our complex visual world.

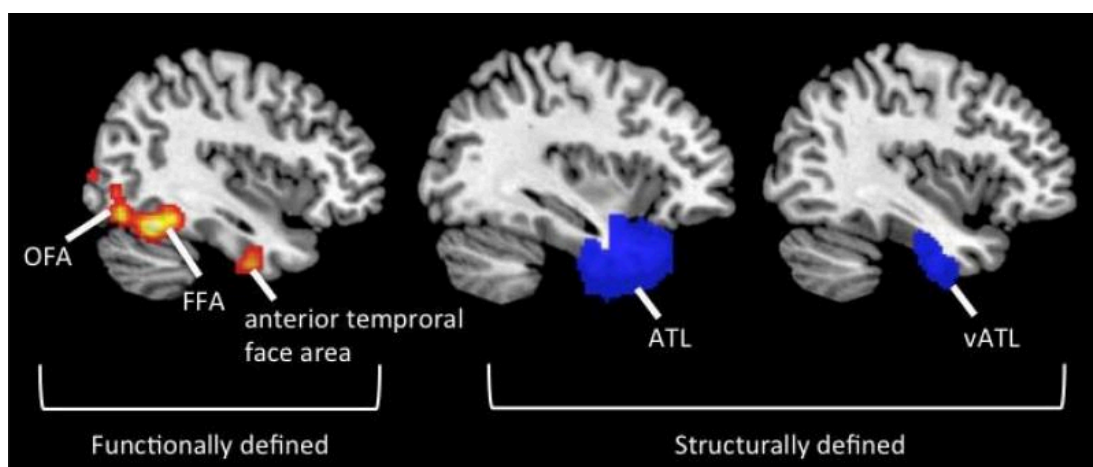


Fig. 11.6.1. shown below shows the regions and extent of the FFA (shown in blue) and the VWFA (shown in red). (Provided by: US National Library of Medicine. License: CC0)

The FFA is located in the ventral stream on the ventral surface of the temporal lobe on the lateral side of the fusiform gyrus. This area of the brain has been specialized for facial recognition.

The visual word form area (VWFA) is a functional region of the left fusiform gyrus and surrounding cortex (right-hand side being part of the fusiform face area) that is hypothesized to be involved in identifying words and letters from lower-level shape images, prior to association with phonology or semantics.[1][2] Because the alphabet is relatively new in human evolution, it is unlikely that this region developed as a result of selection pressures related to word recognition per se; however, this region may be highly specialized for certain types of shapes that occur naturally in the environment and are therefore likely to surface within written language.[1] This area is also apart of the ventral stream.

Similar to auditory stimuli, more dorsal regions of the posterior half of the brain (parietal cortex) are involved in processing information about location (“Where/how pathway”); more ventral (temporal) regions are involved in recognizing objects (“what” pathway). Magnocellular information (colorblind neurons responding to large, fast things) tends to serve up the “where/how” pathway. Parvocellular information (color-sensitive neurons responding to small, slow things) tends to head down the “what” pathway.

Again ... a car zooming by is represented in the dorsal and ventral visual streams: we need information about both identity and location. And this information needs to be integrated. There is a massive white matter bundle (fasciculus) connecting dorsal and ventral regions. This paper by Jason Yeatman et al. about the history of the vertical occipital fasciculus is great:

Yeatman JD, Weiner KS, Pestilli F, Rokem A, Mezer A, Wandell BA (2014). The vertical occipital fasciculus: a century of controversy resolved by in vivo measurements. *Proc Natl Acad Sci U S A*. 111(48):E5214-23.

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URL:<https://en.wikipedia.org/wiki/>

Fusiform_face_area#:~:text=Anatomical%20terminology,gyrus%20(Brodmann%20area%2037).

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URL: https://en.wikipedia.org/wiki/Visual_word_form_area

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124.

PERCEPTION IS AMBIGUOUS

Learning Objectives

Being able to describe some of our visual experiences that illustrate the problem of ambiguity.

Show at least one example of Bistable illusions and describe the ambiguities in that example.

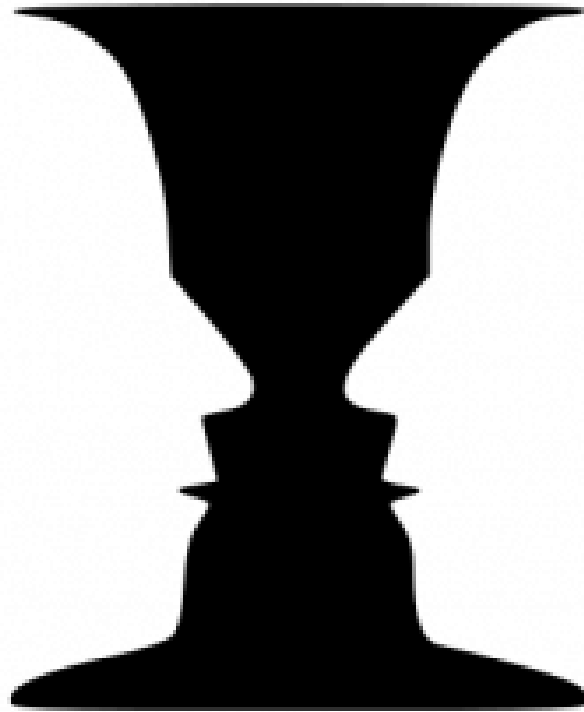


Fig.11.7.1 The famous “face and vase” bistable illusion. (Credit: Alan De Smit. Provided by: Wikimedia Commons. License: CC-BY-2.0.)

Visual perception is often ambiguous. Our perception of a scene is an interpretation of the retinal images. The exact mechanisms that our visual brains use to represent and wrestle with uncertainty are open questions. Here are some visual experiences that illustrate the problem of ambiguity:

- Figure/ground segmentation. Low-level features (lines, edges, textures) provide clues about what belongs with what, but high-level interpretation (shape, scene layout) is also needed to separate foreground and background.
- Vase/Face: when equal evidence exists for multiple interpretations, we experience spontaneous switching between perceptual states. Perhaps this is because feedback has not amplified a “winning” response in V1. For example, refer to the figure above.
- Aperture problem. When we only see part of an image, we can perceive line segments as moving either horizontally or vertically.
- Shading and light.

- Light from above: when a scene is ambiguous, our perception of shape relies on the assumption that light is coming from above and casting shadows downward.
- Shape from shading: our perceptions of lighting, lightness, and shape are interrelated.

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GESTALT PRINCIPLES

Learning Objectives

Being able to explain the six Gestalt Principles, e.g. Pragnanz, similarity, etc.

Know what Gestalt Principles can help us with understanding visual perception.

The first gestalt principle is the figure-ground relationship which we piece the world visually into figure then ground. Figure explains the object that our field focuses on, while ground is our background information. The ability that we interpret our sensory information is based on what is perceived as figure and what is perceived as ground (Peterson & Gibson, 1994; Vecera & O'Reilly, 1998). The second principle that we tend to use is similarity in which we use our visual information and interpret it into groups. For example, when we see a picture with various shapes, we tend to group shapes that are similar to one another rather than different shapes. We might also use the principle continuity where we tend to see the stimuli perceived in a smoother continuous way compared to a discontinuous way. In a figure that has dotted lines, we tend to see the straighter line compared to a line that is more jagged. Another principle that we use is proximity where we organize and group things together in a way where it is meaningful in terms of perception. This means that in the visual information that we perceive, things that are closer to each other are more likely to be grouped together (Fig.11.8.1). Also, we organize what we perceive into complete objects rather than incomplete, which is the closure principle. When we see a square that may be fragmented, we are more likely to perceive it as a complete square. The last principle that we tend to use is pragnanz which coincides with the principles continuity and closure where we perceive objects as concise or meaningful. This means that certain information that is familiar to us makes it harder to be perceived as something different.

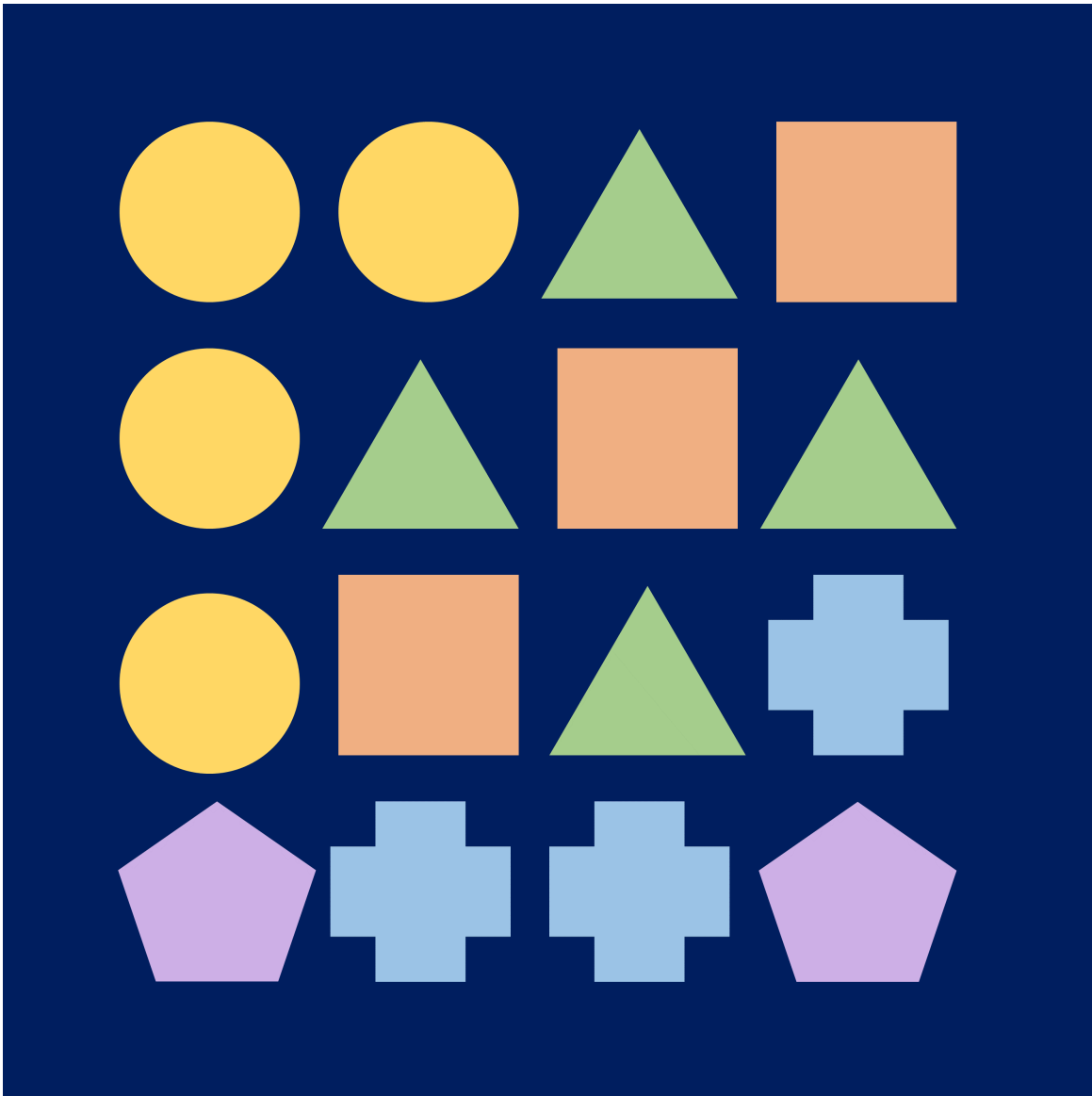


Fig. 11.8.1. Gestalt Shapes. We use Gestalt Principles to organize these shapes in varying ways. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-NC-ND)

Gestalt's principles can help us understand visual perception because one of the required processes that is within our vision is form. We create forms out of sensation components based on gestalt. The word "gestalt" stems from the meaning of patterns and perceiving information as a meaningful, organized whole. However, it considers the idea of the whole is different from the sum of the included parts. This means that our brain is able to create a predictable perception that is more than the sum of its sensory inputs. The part that is predicted is translated into sensory information which allows us to organize it.

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BAYESIAN INFERENCE

Learning Objectives

Know what is Bayesian inference

Know why Bayesian inference is important in visual perception

Bayesian inference comes from Thomas Bayes (1702-1761) who described how inference occurs. Inference based on the probability of an event occurring, using the probability of an event or observation: $p(A|B) \times p(B) = p(B|A) \times p(A)$. We calculate the probability of something developed from prior experience $p(S)$. Bayesian inference can be understood by looking at the Boston Dynamic Big Dog (Fig.11.9.1). Since it appears to be a dog, we use prior experience multiplied by the generative model (living things move) to infer that it is alive.

Visual perception is affected by a combination of things. The human brain uses the image it's being given to calculate the posterior likelihood that it is seeing a specific scene. This is done by using the formula; $p(S|I) = p(I|S) \times p(S) / p(I)$ where S is the scene and I is the image. $p(I|S)$ is called the generative model. This is the probability that the scene will generate a given image. $p(S)$ is the probability of a particular scene which comes from prior experience. $p(I)$ is the probability of the image. This image will not change, most likely, based upon how the brain interprets the scene so when calculating the posterior likelihood of perceiving a specific scene it can be dropped from the calculation. This leads to $p(I|S)$ being proportional to $p(I|S) \times p(S)$. That is to say the probability that a scene will generate a particular image increases or decreases depending on how familiar the person viewing it is. An example of this can be seen when viewing the Boston Dynamic Big Dog (Fig.11.9.1). If, for example, somebody were to kick the dog the viewer would have a negative emotional reaction. This is because the viewer perceives the dog as alive. This is because even though they've never seen an animal that looks like the dynamic big dog it still moves like a living thing. The generative model ($p(I|S)$) asks the probability of the movement being generated by a living thing. Even though the animal looks nothing like a real dog the movement overrides it and the human brain will perceive it as a living organism.



Fig. 11.9.1. Big Dog Robot. Bayesian inference is used to explain how we empathize for a robot dog. (Credit: Lance Cpl. M. L. Meier. Provided by: Wikimedia Commons. License: CC-BY-4.0.)

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Adapted by: Elton Wang and Cole Lachmiller

127.

LATENT VARIABLES

Learning Objectives

- Understand what a latent variable is
- Understand what conditioning is

The latent variable is the unobserved variable that, if you knew its value, would allow you to explain the data that you can actually observe.

Let's say for example that you are sitting in a chair facing a blank wall. Behind you is some object and you're asked to infer what it is. Sometimes a light behind that object is turned on, creating a shadow on the wall that you can see.

The latent variable in this case is the object you can't see, but wish to know what it is. When there is no shadow, all you can do is wildly guess. But even with no shadow, your guess is somewhat informed by your understanding of the world. You might guess that it is an apple before you guessed that it's a snow leopard, since apples are pretty common and snow leopards are rare. This is the **prior**: how likely any potential latent variable is to occur in general.

Conditioning is when you are given a shadow caused by the latent object. So, what is the object behind you *given* this shadow?



Fig. 11.10.1 The shadow provides data you can use to guess what the latent variable is. (Credit: Author.Title.License.URL)

Now this significantly reduces your search space. You see that the shadow is shaped like a human, so that more or less rules out a lot of things, like the snow leopard. But it doesn't rule out *everything* else. It could actually be a pile of trash that is placed in such a way that it makes a shadow that's in the general shape of a human.



Fig.11.10.2 You would need more data (e.g., a different viewpoint) to correctly guess the latent variable.
(Credit: Author.Title.License.URL)

This is where **likelihood** comes in. In this example likelihood would be how likely the particular shadow you are seeing would be caused by a given latent variable (i.e. if the latent variable were a human, how likely would it be that you would observe this particular shadow?). Humans are likely to cause such a pattern, piles of trash are not. This is why the prior and the likelihood are both needed. Maybe the prior for trash is high (there are probably more piles of trash in the world than there are humans), but piles of trash are not likely to create the shadow that you are seeing. These two things combined give you the **posterior**.

Now if you were given more samples, for example if the object was rotated to cause a new shadow, you might have to update your posterior. Maybe when the object is rotated, the shadow becomes formless, in which case the posterior for pile of trash suddenly increases significantly. But maybe the shadow looks like the profile of a human. Human is still a good explanation of the shadow.

The brain's impossible job is to infer what is out in the world (latent variable) using only patterns of photons hitting the retina. In the case of a hierarchical bayesian model of vision in the brain, this sort of conditional inference is repeated at every level of the brain, with the posterior at one level acting as the input/evidence to the one above it. And so in this case the latent variable takes on the form of different visual features that cause other visual features at different levels of abstractness. That is, V1 is inferring the latent cause of the activity in LGN (perhaps certain edges caused that pattern in LGN), V2 is inferring the latent cause of the edges encoded in V1 (perhaps a certain shape caused those edges encoded in V1), and so on all the way up to object category or beyond. Each area is describing the latent variable that would explain the pattern of activity that it is receiving from the area below it and it does this describing in terms of the types of features that it encodes.

A generative model of imagery is reversing this direction of inference. It would be like starting with knowing what the object behind you was, and inferring what kind of shadow it would make.

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SECTION XII

PERCEPTION AND ACTION

What is the point of perceiving things if we cannot interact with them in some way? Perception is rarely a passive experience; it is almost always shaped by some kind of goal. Our perception is also shaped by the fact that we really can't process our entire visual experience. We selectively attend to subsets of our experience and process that; much of our visual world is not encoded. What we attend to depends on our goals at the moment, so our actions shape our perception and our perception shapes our action.

This chapter is being created by Andrea Duong, Megan Hulke, Hae Kun Jung, Peter Kennedy, Joey Kloncz, Joshua Lee, Sophia Masica, Persephone Mo, Umulkheye Mohamed, Kim Tuy Ngo, Blake Paulson, and Annika Nur-Ayeisha Ututalum.

128.

MOTION

Learning Objectives

- Know the importance of motion in scene segmentation
- Be able to define the aperture problem and optic flow
- Know visual area MT/MST are sensitive to motion coherence and MST is responsive to optic flow

Scene segmentation is best put as breaking down the scene you are looking at to see the different parts of it. The importance motion has in scene segmentation includes motion parallax as a depth cue; motion for breaking camouflage; motion for grabbing attention. Motion helps us pick out things in a scene that otherwise would blend in if it were still; similarly, someone sitting across a room full of people would probably blend in with everyone else in the room, but if they were the only ones waving at you they would stand out.

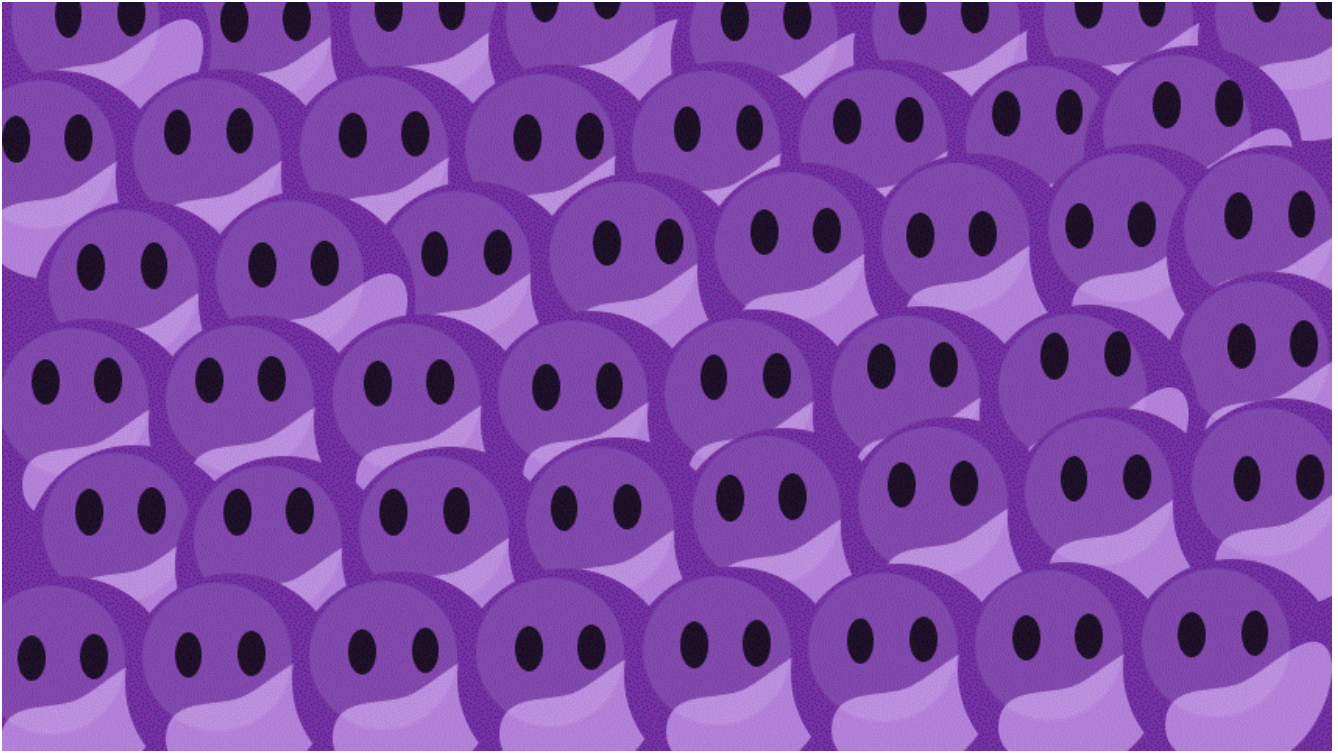


Fig.12.11. Take a look at these cells. You'll notice the ones that blink and move stand out in the crowd.
(Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY-4.0)

The aperture problem refers to when the direction of motion of straight lines is ambiguous because you can't see the ends or corners; in other words if something (the aperture) is in the way of what we are viewing, it can distort the movement we are seeing by making it appear to be stagnant.

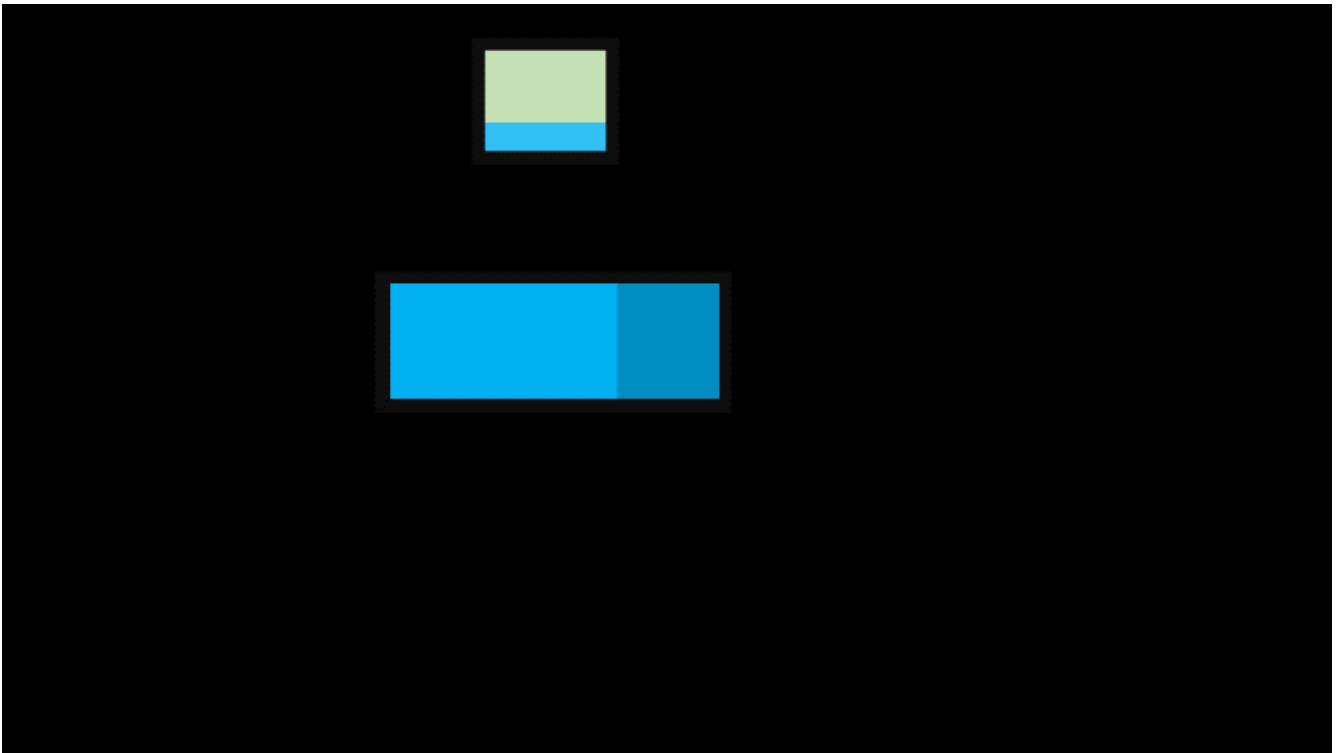


Fig. 12.1.2. Aperture Problem. Notice that if you watch the block through the larger square, it seems as though the block is not moving, since its lines go with the block's direction of motion. But the smaller square shows obvious motion, as its lines do not follow the direction of the block's motion. (Credit: Jarod Davis. Provided by: University of Minnesota. License: CC-BY 4.0)

Visual area MT responds to local motion which is closest to us. MST likes optic flow, which is motion that's coordinated across the entire scene and has a heading, or a point of convergence much like a car driving towards the horizon and looking increasingly smaller as it goes. Check out this cool site with moving examples of optic flow!

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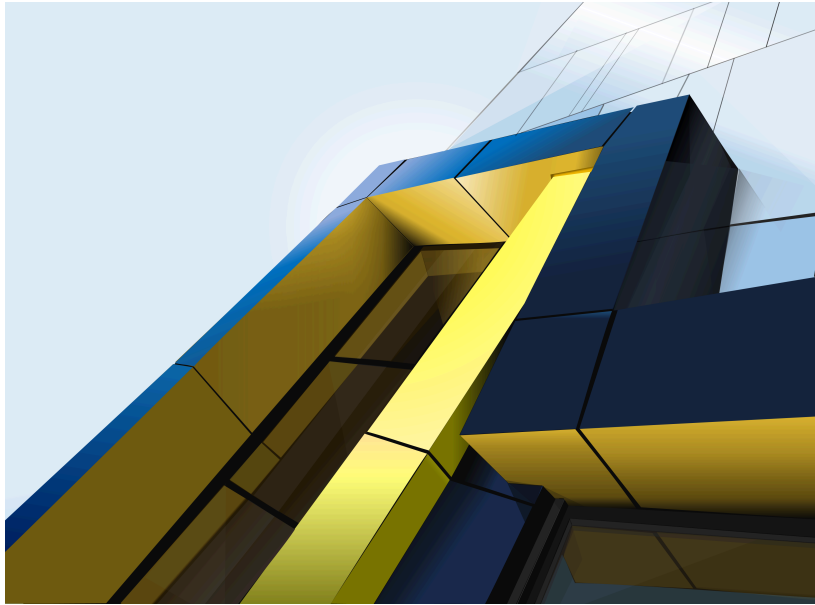
PRE-ATTENTIVE VISION

Learning Objectives

- Know without attention, what we can perceive and what we will miss when first directing our eyes.
- Be able to describe what bottom-up salience and scene gist mean.
- Know subsequent saccades are directed by a combination of salience and top-down knowledge.

A **scene gist** is a brief scenery, sometimes without attention, the brain processes to obtain the essence of what the scenery is about. It can let you determine with a short glance of the scenery whether you are indoors/outdoors, an animal/person, a mountain/sea, and basic geometries. Without attention, we can perceive the scene gist of our surroundings, but not any details that may be perceived upon closer attention.

Bottom-up salience is defined as the degree of difference between a stimulus and its neighbors. Many objects are observed along with its neighboring objects. The brain observes the target object's orientation and separates the object from the neighboring objects with differing orientations.



12.2.1. Bottom-Up Salience. While looking at this image, your brain separates the blocks as individual elements based on their relative orientations. (Credit Jarod Davis. Provided by: University of Minnesota. Based off William Digneault Original image. License: CC-BY 4.0)

The **subsequent saccades** are directed by a combination of salience and top-down knowledge. The salience influences saccades more from individual features of objects and contrasting luminescence, intensity, etc. The top-down knowledge influences saccades depending on what your goals are for looking at the scene (A person, building, etc).

Exercises

1. Which two phrases fill in the blanks most properly? While looking at a scene with bottom-up salience, the brain focuses more on _____, whereas with top-down knowledge, the brain focuses more on _____.
 - A. Gathering all the information; the colors and shapes in the scene
 - B. Individual features; the surrounding context
 - C. What you are expecting to see; what you are not expecting to see

D. The bottom of what you are seeing; the top of what you are seeing

Answer: B

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ATTENTIVE VISION

Learning Objectives

- Know attention can be deployed several ways.
- Be able to define feature-based, location-based and object-based attention.

Attention can be deployed several ways and is required for conjunction search tasks.

Not everything just jumps out at you.

- Basic features, like color, orientation, direction of motion serve as natural segmentation cues
- More complex tasks, like “find the T in with the L’s”, require attention
- Parietal cortex appears necessary for this.

Loads of psychophysical and electrophysiological experiments demonstrate three strategies:

- Feature-based attention: you can look for a particular feature anywhere
Ex. Look for something that is black, metal, and has a diamond design.
- Spatial attention: you can look in a particular place for anything
Ex. Look at the top right of the photo
- Object-based attention: you can deploy attention along an object, even if it’s partially occluded
Ex. Look at the street lamps on the bridge in the photo.



Fig.12.3.1. The figure above is an example of the FIT Theory; however, it serves as a good timeline of when pre-attentive and attentive vision are being used. (Credit: MrBazoun. Provided by: Wikimedia. License: CC-BY SA 3.0)

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CONJUNCTION AND BINDING

Learning Objectives

- Know what conjunction search task is
- Know attention is required for feature binding, relating to the parietal cortex

Conjunction search task is a process of identifying a desired object when it is in close proximity to objects that share some of its same features. Humans engage in conjunction search tasks every day. For example, looking for a specific pencil in a pouch full of them. Every pencil shares similar features to the one you are looking for: the length, the color, the sharpness, etc. To find the one you want, you must systematically critique each one, matching its features to the feature combination you want.

In order to do this, however, binding has to occur. Binding is a feature of attention that involves associating the individual characteristics of an object with each other. Using the figure below, this entails associating the shape (square) with its color (red) and recognizing it as the features of one thing (a red square) rather than two distinct things (red and a square).

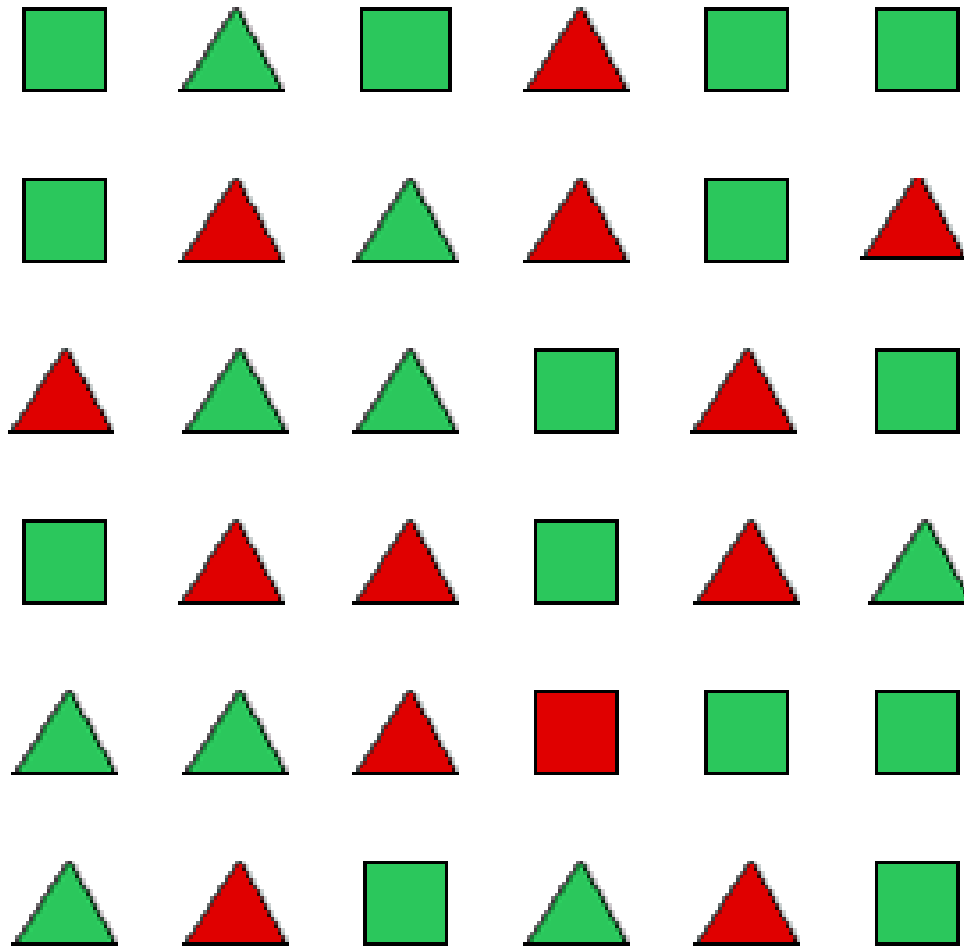


Fig.12.4.1. This picture is perfect for explaining feature conjunction in a visual search task. When looking at this picture and if someone told you to pick out the red square it would take you a longer time to look for it than if they told you to look for a red triangle.(Credit: Head. Provided by: Wikimedia. License: CC0)

The mental process of conjunction searching, and binding are facilitated by the parietal cortex. This area in the brain plays a key role in exercising attention, which is crucial for binding and conjunction searching. Without attention, you would not be able to identify the pencil you were looking for because they would all look too similar. In fact, you might even grab the wrong pencil because you associated the red of the pencil you wanted with a pencil near it that is not the same color!

The perceptions derived from conjunction and binding are not always accurate which can be illustrated in baseball. In baseball, curveball pitches can produce an illusion to the batter as a result of accidental feature binding (feature blurring) in peripheral vision. This gives the batter the wrong impression of the ball's trajectory if the batter does not look directly at the ball.

In this video, you can learn more about the binding problem.



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Adapted by: Joey Kloncz & Jarod Davis

References:

[1] Kesner, R. (2012). "Parietal lesions produce illusory conjunction errors in rats". *Frontiers in Integrative Neuroscience*. doi:10.3389/fnint.2012.00022.

132.

INATTENTIONAL BLINDNESS

Learning Objectives

- Be able to define change blindness
- Know object-selective neurons fail to respond many things that go unattended
- Explain that our interaction with objects is not limited by conscious perception

Change blindness refers to the perceptual phenomenon that the observer fails to identify when there is a change in a visual stimulus. We fail to perceive so many things that go unattended. Classic change blindness demos, like this one with static images or this one with real people show how much of a scene we do not perceive. If you don't have some kind of bottom-up salience cue (motion, color contrast ...), you need some top-down attention allocation if you're going to be aware of something. This relates to how object-selective neurons fail to respond to many things that go unattended. For example, a major source of image motion is eye motion: we're making a saccade every few hundred milliseconds. How does our brain handle that? Saccadic masking: our brains blank out all the wild motion created by saccades. It would be impossible for our neurons to respond to every motion created by saccades.



Fig.12.5.1 In this figure, an example of change blindness is shown through the use of two similar pictures. For the picture on the left, it represents what the participant is able to see through their visual perspective. The picture on the right depicts what they actually see. The key takeaway from this representation is that the tree branch present in the left side picture fails to be identified by the viewer due to the emphasis on other details. It is very common that the swaying of a tree branch goes unattended when our eyes focus on greater details such as the world monument in the center or the immense body of water behind it. Other visual phenomena that many of us instantly may have missed are the people and the vertical building in the background that occupy the left photo are nowhere to be seen for the other one.

Watch this video on the Monkey Business Illusion to learn more about inattentional blindness!



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Adapted by: Joshua Lee

PERCEPTION AND ACTION

Learning Objectives

- Be able to explain how perception guides action with example
- Be able to describe how action changes perception with example
- Know acting on unconscious sensory information is possible

The relationship between perception and action is best described by a diagram with an arrow pointing both ways. Perception selects targets for action and helps us correct errors as we execute actions. Broadly speaking, there are 2 kinds of actions: navigation (moving around our environment) and reaching/grabbing.

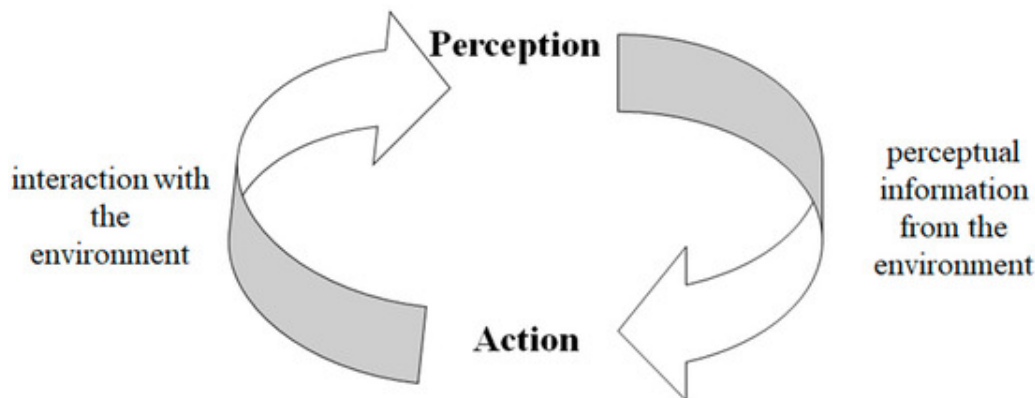


Fig.12.6.1. This caption shows the relationship between perception and action which is described as a cycle or arrows pointing back and forth. An example of this cycle is when you reach out for a bottle sitting on a slanted surface: Banks et al (2000) showed that your sense of touch updates your visual estimates of the surface slant. (Provided by: MDPI. License: CC-BY 4.0)

Acting on unconscious sensory information is possible. There are a few dramatic examples of action without perception. For example a patient, DF, could mail letters through a slot but couldn't tell you what the

orientation of the slot was, or patient TN, who was completely blind but could navigate cluttered hallways. TN's case make it obvious that visual information gets to the non-visual regions of the brain (parietal cortex) even when V1 is not working.

One line of research suggests that reaching and grasping behaviors are not always affected by visual illusions. For example, even though the Ebbinghaus size illusion is very strong, if people pinch together their fingers like they're going to grasp the center disk, they grasp more accurately than their conscious reports of perceived size (Haffenden and Milner, 1998). Similarly, healthy controls accurately grasp a center object affected by the rod-and-frame illusion, which is the phenomenon that the orientation of a line is altered by surrounding lines or grating (Dyde, 2000). These studies suggest that all of us maintain a “raw copy” of sensory information, separate from the version that we're consciously aware of (which has been shaped by inference), to guide action. However, there is contradictory research indicating that motor planning (Craje, 2008) and reaching behaviors (Dyde, 2000) can be impacted by illusions. So it is an open and interesting question: when is our behavior controlled by illusions, and when does our motor system ignore illusions?

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References:

Craje, C., van der Kamp, J., & Steenbergen, B. (2008). The effect of the “rod-and-frame” illusion on grip planning in a sequential object manipulation task. *Experimental brain research*, 185(1), 53–62. <https://doi.org/10.1007/s00221-007-1130-x>

Dyde RT, Milner AD (2002) Two illusions of perceived orientations: one fools all of the people some of the time; the other fools all of the people all of the time. *Exp Brain Res* 144:518–527

Ernst, M., Banks, M. & Bühlhoff, H. Touch can change visual slant perception. *Nat Neurosci* 3, 69–73 (2000). <https://doi.org/10.1038/71140>

Haffenden AM, Goodale MA. The effect of pictorial illusion on prehension and perception. *J Cogn Neurosci*. 1998;10(1):122-136. doi:10.1162/089892998563824

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MIRROR NEURONS

Learning Objectives

- Know mirror neurons located in premotor cortex (frontal lobe)
- Be able to explain the function of mirror neurons

Mirror neurons represent a distinctive class of neurons that discharge both when an individual executes a motor act and when he observes another individual performing the same or a similar motor act. These neurons were first discovered in monkey's brain. In humans, brain activity consistent with that of mirror neurons has been found in the premotor cortex, the supplementary motor area, the primary somatosensory cortex, and the inferior parietal cortex.

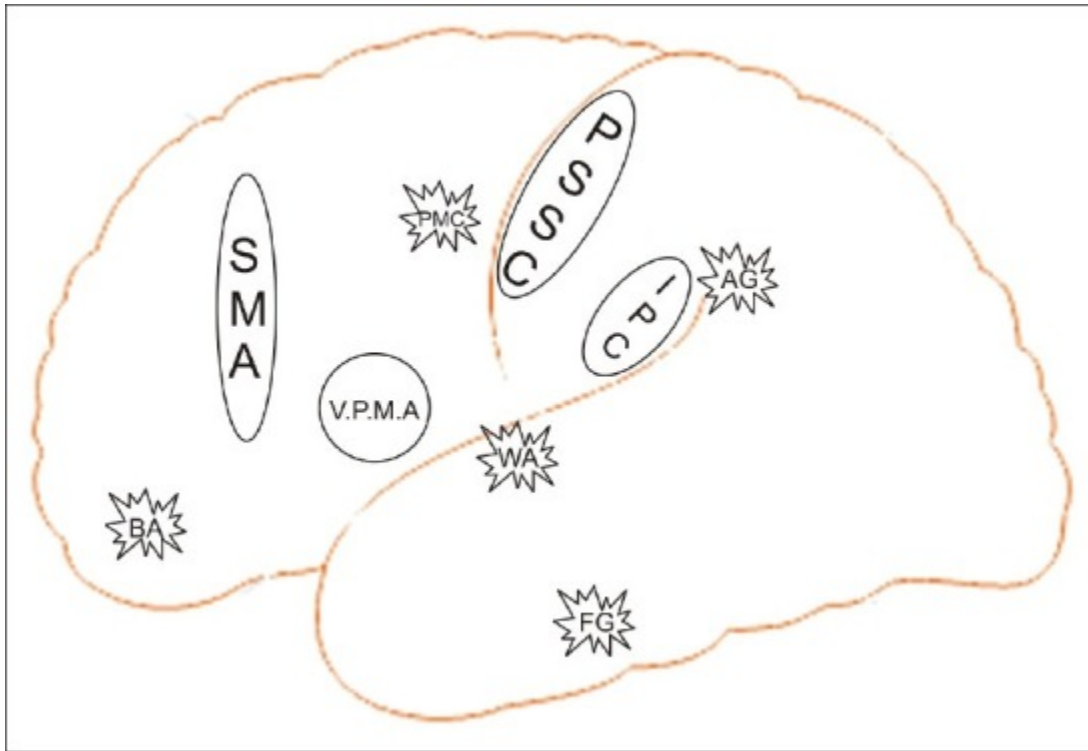


Fig. 12.7.1. The mirror neuron system in the human brain. (1) SMA: Supplementary motor area, (2) PSSC: Primary somato sensory cortex, (3) IPC: Inferior parietal cortex, (4) VPMA: Ventral premotor area, neurons having mirror properties, BA: Broca's area, WA: Wernicke's area, FG: Fusiform Gyrus, AG: Angular gyrus, PMC: Primary motor cortex. (Provided by Acharya and Shukla, JNSBM. License: CC BY 3.0).

What might be the functional role of the mirror neuron system? A series of hypotheses such as action understanding, imitation, intention understanding, and empathy have been put forward to explain the functional role of the mirror neurons. In addition to these, it has also been suggested that the mirror neuron system represents the basic neural mechanism from which language evolved.

The question, however, of what is the function of the mirror neuron system is probably an ill-posed question. Mirror neurons do not have a unique functional role. Their properties indicate, rather, that they represent a mechanism that maps the pictorial description of actions carried out in the higher order visual areas onto their motor counterpart. This matching mechanism may underlie a variety of functions. More details about specific functions served by mirror neurons are in this Scholarpedia article.

Learn more about mirror neurons in the video linked here and included below.



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Acharya, S., & Shukla, S. (2012). Mirror neurons: Enigma of the metaphysical modular brain. *Journal of natural science, biology, and medicine*, 3(2), 118–124. <https://doi.org/10.4103/0976-9668.101878>

Giacomo Rizzolatti and Maddalena Fabbri Destro (2008) Mirror neurons. *Scholarpedia*, 3(1):2055.

135.

NAVIGATION

Learning Objectives

Be able to define navigation

Be able to describe the location and function of the brain region most commonly associated with navigation

The brain region most strongly implicated in the landmark recognition skills necessary for navigation is in parahippocampal cortex. A recent neuroimaging study found that patients who had received thermal lesions in parahippocampal regions (inferior ventral temporal regions close to the hippocampus) were less capable of remembering the configuration (relative location) of objects, but were not impaired in identifying the objects (Bohbot, 2015). This is the kind of research that implicates parahippocampal cortex in navigation, one skill that bridges the gap between perception and action.

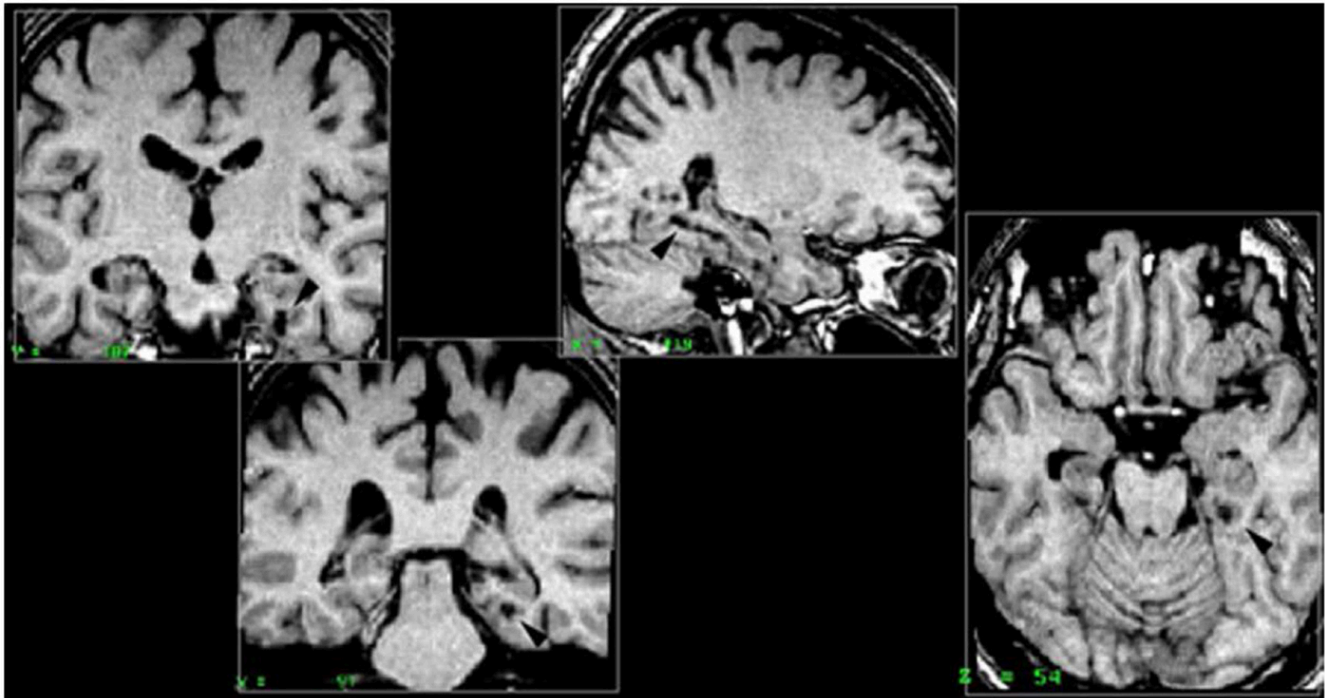


Fig. 12.8.1. Right parahippocampal lesion. MRI section in a coronal plane, zooming-in on the lesion to the right parahippocampal cortex (RPH) of patient PP (indicated with the arrow). This section was taken 30 mm posterior to the anterior commissure ($Y = -30$) in Talairach standard stereotaxic space. (Provided by: Bohbot et al., *Frontiers in Human Neuroscience*. License: CC BY.)

One of the most famous studies is a brain imaging study of London taxi drivers, which found that taxi drivers have larger PHCs than a control population (Maguire, 2006). The difficulty with interpreting the original results, of course, is that a causal relationship cannot be established — maybe people with big PHC regions are good at learning complicated road networks, or maybe learning all those twists and turns and landmarks actually makes your PHC grow. The research group followed up their initial study with a longitudinal study (following cab drivers from when they started training until they learned the streets, and comparing them against a control population). The second study was actually able to measure an increase in the size of posterior hippocampal cortex as cab drivers learned the streets (Woollett, 2011).

Here is an additional video about London taxi drivers.



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Bohbot VD, Allen JJ, Dagher A, et al. Role of the parahippocampal cortex in memory for the configuration but not the identity of objects: converging evidence from patients with selective thermal lesions and fMRI. *Front Hum Neurosci*. 2015;9:431. Published 2015 Aug 3. doi:10.3389/fnhum.2015.00431

References:

Bohbot VD, Allen JJ, Dagher A, et al. Role of the parahippocampal cortex in memory for the configuration but not the identity of objects: converging evidence from patients with selective thermal lesions and fMRI. *Front Hum Neurosci*. 2015;9:431. Published 2015 Aug 3. doi:10.3389/fnhum.2015.00431

Maguire E.A., Woollett K., Spiers H.J. London taxi drivers and bus drivers: a structural MRI and neuropsychological analysis. *Hippocampus*. 2006;16:1091–1101.

Woollett K, Maguire EA. Acquiring “the Knowledge” of London’s layout drives structural brain changes. *Curr Biol*. 2011;21(24):2109-2114. doi:10.1016/j.cub.2011.11.018

CURVE BALL ILLUSION

Learning Objectives

- Be able to explain what the curveball illusion is
- Know curveball illusion is an example of when it's better not to update action plans with new perceptual information

The human visual system does not treat all parts of an image equally: the central segments of an image, which fall on the fovea, are processed with a higher resolution than the segments that fall in the visual periphery. Even though the differences between foveal and peripheral resolution are large, these differences do not usually disrupt our perception of seamless visual space. The curveball illusion represents a motion stimulus in which the shift from foveal to peripheral viewing creates a dramatic spatial/temporal discontinuity.

In the curveball illusion, a disk descends vertically from the top center of the screen to the bottom center, while motion inside the disk is from right to left. If an observer tracks the disk foveally (looking directly at the disc), the disk appears to descend vertically; however, if an observer shifts his/her gaze to the right so that the disk falls in the visual periphery, the disk appears to descend at an slanted angle. This shift also occurs when a baseball player will hit a baseball; the player will initially place the ball within their foveal vision as it is thrown but ultimately switch to their peripheral vision to strike the ball.

As a result of this illusion, batters often report that the flight of the ball undergoes a dramatic discontinuous shift in position as the ball nears home plate, also called the “break.” The curveball’s “break” is a perceptual illusion caused by the inability of peripheral vision to maintain separate representations of different motion signals, and gaze shifts between foveal and peripheral vision during the curveball’s flight. The discontinuity is therefore the result of new perceptual information from the change in the neural response at the transitional moment when the image of the ball is transferred from the fovea to the periphery (or vice versa).

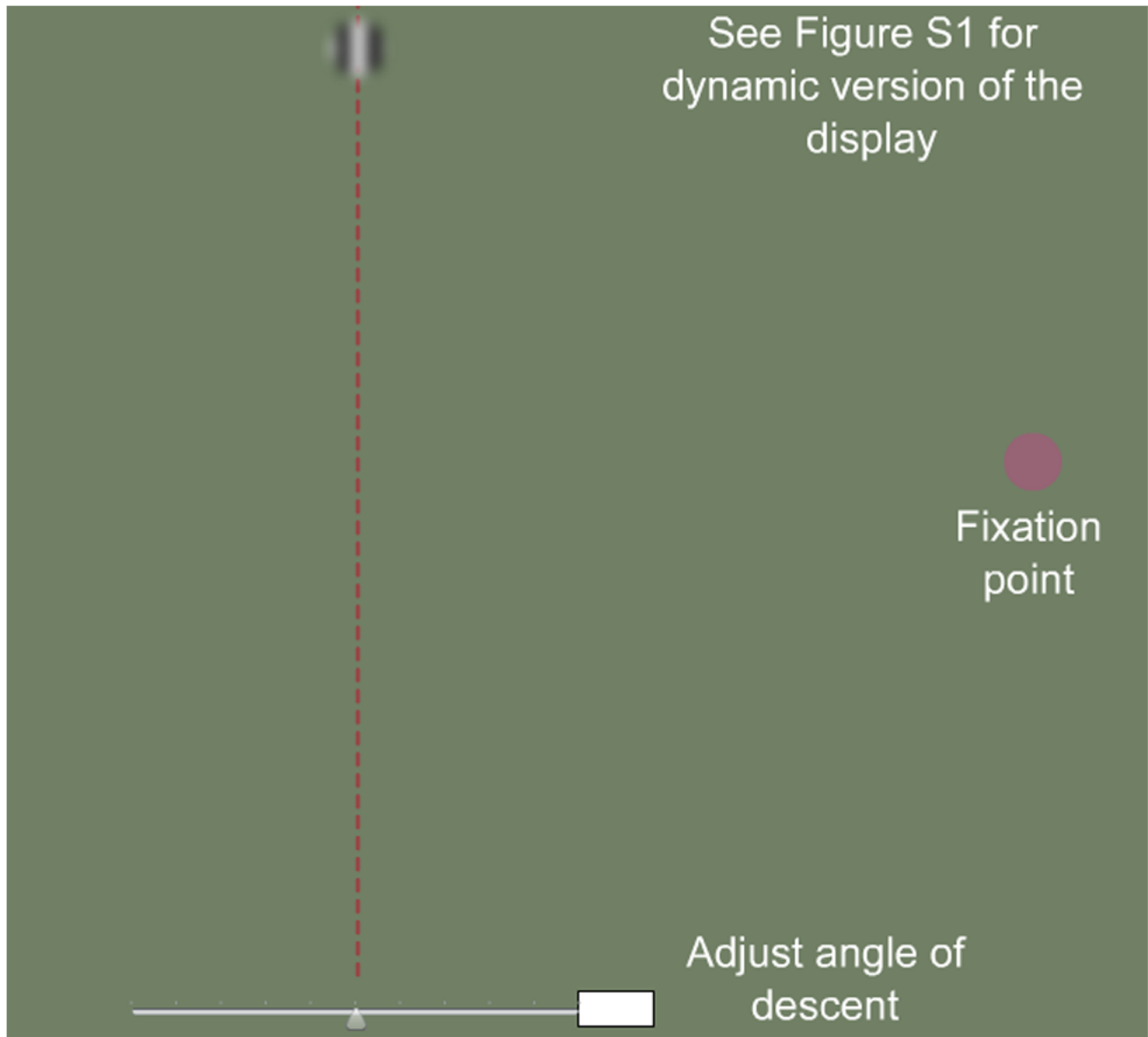


Fig. 12.9.1. The curveball illusion. Above is an example of the curveball illusion. The red dotted line represents the path of the disc when viewed with foveal vision, and the straight black line represents the change in the path of the disc when viewed with peripheral vision. [Click here to experience the curveball illusion in real time.](#) (Provided by Shapiro et al., PLoS ONE. License: CC BY).

The Curveball Illusion involves “feature blurring” — position and direction of motion get blurred together in confusing ways in peripheral vision, where receptive fields are large. An illusion that works off of similar principles is the Rotating Snakes illusion. That one has a very cool possible explanation related to contrast-dependent action potential latencies in V1 (how long it takes a neuron to respond to a stimulus depends on how strong the stimulus is).

Test your visual acuity with this video of the curveball illusion!



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Shapiro A, Lu Z-L, Huang C-B, Knight E, Ennis R (2010) Transitions between Central and Peripheral Vision Create Spatial/Temporal Distortions: A Hypothesis Concerning the Perceived Break of the Curveball. PLoS ONE 5(10): e13296. <https://doi.org/10.1371/journal.pone.0013296>

137.

SYNESTHESIA

Learning Objectives

- Be able to describe what synesthesia is
- Be able to describe color/grapheme synesthesia

Synesthesia is a condition in which individuals experience atypical responses to certain types of stimuli, in addition to the typical responses elicited by those stimuli. For example, a synesthete may perceive tastes when seeing certain shapes or might perceive colors when seeing achromatic letters. Synesthesia comes in many forms, covering a wide range of sensory interactions both cross-modally and within a single modality.

It will be useful to highlight some characteristics of synesthesia that serve to distinguish it from other perceptual phenomena, such as visual imagery and certain forms of imagistic memory. There are three such characteristic features: (1) automaticity, (2) reliability, and (3) consistency. First, there is ample evidence that synesthetic associations are automatic in nature. They are typically produced outside the intentional control of the individual and cannot be directly inhibited. Second, it is typically the case that synesthetes reliably experience synesthetic responses when presented with triggering stimuli. Finally, although there is variability across synesthetes, synesthetic associations within an individual appear to remain relatively consistent over time in that the same types of stimuli (e.g., specific auditory tones) tend to elicit the same types of synesthetic responses (e.g., specific colors).

Grapheme-color synesthesia is a condition characterized by enduring and consistent associations between letter/digits and colors. For people with grapheme-color synesthesia, letters or digits have fixed enduring conscious color associations. For example, the letter A might be red, B might be blue, C might be yellow and so on. In the terminology of the literature, letters and digits are the “inducers” of grapheme-color synesthesia, and the color itself (i.e., the synesthetic experience) is the “concurrent”.



Figure 12.1.1. Different synesthetes may link the letter 'A' to the color red and the letter 'B' to the color blue and so on. (Credit: Flickr)

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Synesthesia: a colorful word with a touching sound?

Authored by: Myrto I. Mylopoulos and Tony Ro

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License: CC BY 4.0A longitudinal study of grapheme-color synesthesia in childhood: 6/7 years to 10/11 years

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