



[Return to Neuroscience](#)

SENSORY SYSTEMS

[Download a copy of this study guide](#)

- [Somatic Sensation](#)
 - [Vision](#)
 - [Hearing](#)
 - [Taste and Smell](#)
 - [Sleep and Wakefulness](#)
 - [The Hypothalamus](#)
 - [Higher Cortical Function](#)
 - [The Limbic System](#)
-



[Return to top](#)

SOMATIC SENSATION

TRANSDUCTION: The process by which a physical stimulus is converted into a neural signal and sent to the CNS.

- **Trigger Zone:** The threshold of stimulus, in the sensory receptor, at which an action potential is generated. Some sensory receptors are more sensitive than others.
- **MODALITY SPECIFICITY:** Any particular sensory unit is most sensitive to only one modality.
 - There are four broad classes of somatic stimuli:
 - Tactile Sensation (Pressure, Cold)
 - Proprioception (body position and movement)
 - Thermosensation
 - Pain
 - The modality to which a receptor is sensitive is called the **adequate stimulus** for the receptor.
 - The specific modality is the one that triggers the receptor at the *lowest threshold potential*. Other modalities may also trigger the receptor, but at much higher potentials.
- **Paradoxical Cold** is an exception to the Modality Specificity rule.
 - Sometimes heat may be perceived as cold, because it triggers cold fibers rather than warm fibers.
 - Normally warm fibers are triggered by an increase in temperature, and cold fibers by a decrease in temperature.

FIBER DIAMETER AND MODALITY SPECIFICITY:

- **Class II (A-beta) Fibers: Cutaneous Sensation**
 - Fibers terminate in *specialized nerve endings* such as Merckel's Disks and Pacinian

- Corpuscles.
- **ASPHYXIA:** These fibers are most sensitive to asphyxia and to physical insult, because they are the largest of the sensory fibers.
 - Anesthesia: These fibers are the *last* to be blocked by anesthesia -- they are the largest fibers.
 - **Class III (A-delta) Fibers:** *Fast Pain*, crude touch, temperature sensation.
 - Fibers terminate in *free nerve endings*.
 - **FAST PAIN:** Pin-prick pain; it is the first pain you will feel when pricking your finger.
 - These fibers are sensitive to intense mechanical stimulation (such as a puncture) and temperature.
 - Purpose = these fibers cause us to quickly withdraw away from a dangerous stimulus.
 - **Class IV (C) Fibers:** *Slow Pain*, crude touch, temperature sensation.
 - Fibers terminate in *free nerve endings*.
 - **SLOW PAIN:** Throbbing pain, which evokes the troublesome affective experience of pain.
 - These fibers respond to visceral *noxious stimuli* -- either mechanical, heat, or chemical.
 - Purpose = these fibers cause us to immobilize the body part so it can heal.
 - **ANESTHESIA:** *Slow pain fibers are the most sensitive to local anesthesia. Anesthesia blocks small-diameter fibers before large-diameter.*
 - Asphyxia: These fibers are the *last* to be blocked by asphyxia, as they are the smallest fibers.

CLASS	DIAMETER VELOCITY	ELECTRICAL STIMULATION	SENSATION
II (A-beta)	Relatively large diameter Fast conduction velocity	Lowest threshold i.e. first to be stimulated	Cutaneous Sensation
III (A-delta)	Small diameter Slow conduction velocity	Medium threshold	Fast Pain Crude Touch Some temperature
IV (C)	Smallest diameter Slowest conduction velocity	Highest threshold i.e. last to be stimulated	Slow Pain Crude Touch Some temperature

FREE NERVE ENDINGS: Pain and temperature both end in free nerve-endings in the skin.

SPECIALIZED NERVE ENDINGS: They mediate tactile sensation: flutter, vibrations, and pressure.

- **MEISSNER'S CORPUSCLES:** They mediate the sensation of **flutter** -- localized, slow vibrations.
 - **Rapidly-Adapting, Phasic** response: The receptor shows *Adaptation* in that it stops firing after the same stimulus has been present for a while. It "blocks" out the stimulus once the

- stimulus becomes old news.
- Anatomical Distribution: Found in *Glabrous* (non-hairy) skin, as in palm of hands.
 - The nerve fiber loses its myelin sheath before entering the corpuscle.
 - Serves the same purpose that hair follicles serve in hairy skin, i.e. sensation of flutter.
- **PACINIAN CORPUSCLES:** They mediate the sensation of **vibration**.
 - **Rapidly-Adapting, Phasic** response: The receptor stops firing after the stimulus has been present for a while.
 - **STRUCTURE:** It is like an **onion**.
 - If you remove the layers of the onion, then the *receptor becomes slowly-adapting*. The onion-layers thus serve the purpose of adaptation -- they make it so the underlying nerve fiber is only discharged temporarily.
 - The onion layers also *filter out the low-frequency stimuli*, such that Pacinian Corpuscle has frequency specificity for **high frequency vibrations**.
- **MECKEL'S DISKS:** Mediate sensitivity to **pressure**.
 - **Slowly-adapting, Tonic** response. The nerve continues to discharge as long as the stimulus remains. So, you continue to feel pressure as long as the pressure is still there.

ADAPTATION: "A decrease in neural response to sustained stimulation." Meissner's and Pacinian corpuscles both show adaptation.

- **PHASIC RESPONSE** refers to the fact that the receptor will fire only when there is a *change in stimulation*.
- **TONIC RESPONSE:** Refers to continual firing of the receptor when a continual stimulus is present. No change in stimulus is necessary to maintain firing.

DORSAL-COLUMN MEDIAL-LEMNISCAL PATHWAY: *Proprioception and Discriminative Touch* run parallel with each other but actually have separately named paths.

- **PATHWAYS:** For spinal components (not trigeminal). *These fibers enter through the medial branches of the Dorsal Horn.*
 - **DISCRIMINATIVE TOUCH:** Discriminative Touch fibers are **Group II (A-beta)** fibers.
 - First-Order: Nucleus Gracilis (medial, S4-T5) and Cuneatus (Lateral, C1-T4) carry first-order neurons.
 - Thalamic Relay: **VPL, Ventral Posterolateral Nucleus** of Thalamus.
 - Somatosensory Cortex: **Area 3b**
 - So, *Area 3B receives 3rd-order neurons originating from specialized receptors (Meckel's Disks), via Group II fibers*
 - **PROPRIOCEPTION:** Conscious Proprioception receptors are **1A-SPINDLE FIBERS**
 - Evidence: Joint position and movement can still be perceived following anesthesia to the joint capsule or following replacement with a prosthetic joint. Thus, the proprioception fibers don't lie within the joint.
 - First-Order: **Nucleus Z** (S4-T5) and **External Cuneate Nucleus** (C1-T4)
 - Thalamic Relay: **VPS, VENTRAL POSTERIOR SUPERIOR Nucleus** of Thalamus.
 - Somatosensory Cortex: **Area 3a**
 - So, *Area 3A receives IIA Spindle Afferents for proprioception signal.*
- **SOMATOTOPIC ORGANIZATION**
 - Spinal Cord: Sacral is most medial and Cervical is most lateral. As you move up the cord, sacral segments enter the cord first, and higher up segments enter right "on top of," i.e. lateral to, the sacral segments.

- THALAMUS: Somatotopic Organization is essential reversed.
 - VPM: Trigeminal, i.e. head, is *most medial* in medial nucleus.
 - VPL: Spinal is more lateral.
 - VPS: The VPS lies above both of the other nuclei, and it maintains the organization of head = medial, and sacral = lateral
- SOMATOSENSORY CORTEX: There are *four relatively complete maps* of the body: 3a, 3b, 1, and 2.
 - Sacral (lower body) is most medial, at the top of the somatosensory cortex.
 - Cervical (upper body) is most lateral, at the temporal pole of the somatosensory cortex.
- DISTORTED REPRESENTATION: Finger tips, lips, and tongue get a disproportionate amount of cortex, because they are the most sensitive sensory organs.
- DESCENDING SENSORY Connection: Descending *Sensory* fibers go from Sensory Cortex -----> Thalamus -----> Dorsal Column Nuclei. They may serve a role in adaptation or filtering repetitive stimuli, but function is unsure.
- LESIONS:
 - **Tabes Dorsalis:** Secondary to Syphilis; lesion of dorsal columns. Patients show deficits in proprioception and discriminative touch, but not pain and temp.
 - **Transsection of Dorsal Columns:** Results in large increase in two-point discrimination.
 - **Destruction of S1 (Somatosensory Cortex):** Expected sensory deficits result.

Labelled Line Theory: There are separate pathways for each modality of sensation, and these all run into the CNS in a parallel fashion.

- **CORTICAL COLUMNS:** Each column contains layers that represent *different modalities*, but they all came from the *same region of the body*.
 - So, neurons in the same "layer" or **lamina** of the cortex will exhibit the same modality specificity.

RECEPTIVE FIELDS: The area of skin which, when appropriately stimulated, causes a neuron to discharge.

- The smaller the receptive field, the more sensitive is the sensory ability. Smaller receptive fields mean higher acuity.
- *Proprioceptive and Discriminative Touch (DC-ML) fibers have smaller receptive fields than pain and temp (anterolateral) fibers.*
- **TWO-POINT THRESHOLD:** The minimum distance, on the skin, at which two pin-points can be distinguished. The smaller the two-point threshold, the higher the *tactile acuity*.
 - Fingertips and lips have smallest two-point-thresholds (~2 mm), while trunk has much larger threshold (~60 mm)
- Small receptive fields correspond to **high innervation density** and a disproportionately large amount of somatosensory tissue in the CNS.

PHANTOM LIMB SYNDROME: People who have had a severed limb still retain sensation that the limb is there (proprioception) when other parts of the body are stimulated, such as the face.

- This is due to **plasticity** of neurons in the CNS. The CNS neurons that used to supply the missing limb are near the face, so they can get stimulated when face is stimulated.

HIERARCHICAL PROCESSING: As one ascends through the CNS, more complex types of sensations are processed.

- **Area 3a and 3b** (aka S1) are the first recipients of sensory information.
- **Area 1 and 2** (aka S2 (?)) receive input from Areas 3a and 3b. Thus 1 and 2 are higher up in the processing of somatic sensation.
 - For example, Areas 1 and 2 can discriminate selectivity of movement of a finger across the skin, whereas area 3 cannot.
- **AREA 2:** It is unique in that it receives a *convergence of multiple modalities*. Both Proprioceptive and Tactile input can arrive at the same fibers.
 - This convergence of function allows for Area 2 to perform **Stereognosis** (identifying objects by touch). *Area 2 lesion results in severe deficit in this specific ability.*

NEGLECT SYNDROME: Lesion of posterior Temporo-parietal area. **Extinction** occurs in the Neglect Syndrome. Extinction is failure to recognize a specific stimulus (either visual, somatic, and/or auditory) on one side of the body, contralateral to the lesion.

PAIN:

- Terms:
 - **Nociceptive Stimuli:** Stimuli that produce pain.
 - **Analgesia:** A condition in which nociceptive stimuli are not perceived as painful.
- Two components of Pain: *The two components of pain are separable by drugs.*
 - The sensation of pain itself.
 - The *affective component* of pain in which it is perceived to be painful or unpleasant.
 - **Morphine** separates these two components such that patients still feel the pain but they do not find it to be unpleasant or "painful."
- **PAIN-PRODUCING STIMULI (PPS):** Chemicals that are involved in transduction of *slow-pain* fibers.
 - **POTASSIUM:** High extracellular K⁺ is indicative of tissue damage and is therefore painful.
 - **BRADYKININS:** Tissue injury -----> proteolytic enzymes into the extracellular fluid -----> react with gamma globulins to create Bradykinins.
 - *Bradykinin is one of the most painful substances known.* It activates C-Fiber terminals.
 - Bradykinins causes vasodilation.
 - Bradykinins causes the production of **Prostaglandin E2 (PGE₂)**, which serves to enhance the sensitivity of painful C-Fibers.
 - Aspirin will block this prostaglandin synthesis, wouldn't ya know??
 - **HISTAMINE: Substance-P**, released by C-fibers, causes Mast Cells to release Histamine.
 - Histamine can also activate C-fiber terminals.
- Neurotransmitters used in Anterolateral System:
 - GROUP III (A-delta) NEUROTRANSMITTER: **Glutamate**
 - GROUP IV (C-FIBER) NEUROTRANSMITTERS: C-Fibers have two neurotransmitters which cause vasodilation when released on vessels.
 - **Substance-P:** Increases capillary permeability, perhaps via NO.
 - **Calcium Gene-Related Peptide (CGRP):** It enhances the vasodilatory effects of Substance-P.
- ANTEROLATERAL SYSTEM PATH:: Group-III (Fast Pain) and IV (slow-Pain) fibers enter the spinal cord through the *lateral portion* of the Dorsal Root over the **Tract of Lissauer** -----> Ascend one or two segments -----> Synapse in **Substantia Gelatinosa** -----> CROSS -----> Ascend in Anterolateral tract.
 - **NEOSPINOTHALAMIC PATHWAY:** Fast pain (III) and temperature sensation.
 - Second order neurons terminate in the **VPL** of Thalamus.
 - These neurons are modality specific, have high thresholds of stimulation, and have

- small receptive fields -- all things expected for fast, sharp, localized pain.
- **MARGINAL ZONE:** *Lamina I and V* of the Dorsal Horn. This is the outermost and innermost layers of the Anterolateral System.
 - *Fast Pain only* goes through the Marginal Zone.
 - The fibers split into layers in the Tract of Lissauer.
- **PALEOSPINOTHALAMIC PATHWAY:** Receives Slow-Pain (IV) fibers, plus some Fast-Pain.
 - Second order neurons terminate in **Intralaminar Nuclei** of Thalamus.
 - Lesion of the Intralaminar Nuclei will relieve chronic pain.
 - These fibers mediate, chronic deep pain, but not cutaneous pain.
 - *Lamina II and III* carry the Slow Pain fibers -- the middle two layers of Anterolateral System.
- **Anterolateral Cordotomy:** Sectioning the anterolateral cord on the *contralateral side* in order to relieve intractable pain.
- Targets of Anterolateral Pathway:
 - **Spinoreticular Pathway:** Also for modulation of pain (see below)
 - **Spinotectal Pathway:** Also involved in pain control; orientates our response to painful stimuli.
 - **Spinothalamic Pathway:** The primary pathway for pain transmission to Thalamus.

TRIPLE RESPONSE OF LEWIS (AXON REFLEX):

- **Wheal:** Localized raised area resulting from vasodilation from local irritants.
- **Flare:** Reddened area surrounding the wheal.
 - It is a *axon-axon reflex* that does not go through the CNS. Local Nociceptive fibers are stimulated, and they send messages to neighboring fibers to cause a "flare" of vasodilation around the original wheal.
- **Capsaicin** is a peppery substance that causes the wheal-and-flare response locally. Applied continually, it will *desensitize the C-Fibers* to local allergens and can thus be used as a topical analgesic.
- **HYPERALGESIA:** Enhanced sensitivity to pain occurs in the region following the Wheal and Flare response.

THALAMIC (CENTRAL PAIN) SYNDROME: Spontaneous pain, and exaggerated responses to pain stimuli, resulting from a *vascular lesion in the Thalamus*.

- Triad of related symptoms:
 - Spontaneous Pain
 - Non-Injurious stimuli (light touch, movement) are perceived as painful.
 - Hyperalgesia: aggravated pain response.
- Originally, it was thought that only the Thalamus produced these symptoms, but it is now known that a lesion anywhere along the pain pathway (such as anterolateral cordotomy) can produce the symptoms.

Mechanisms of ANALGESIA:

- **GATE CONTROL THEORY:** Transmission of pain information can be modified by descending CNS large-fibers. *Endogenous activity in large-fiber pathways can block pain.*
 - After you bump your head, rubbing it can help it. When you rub your head you are stimulating large fiber pathways.
 - **Spinotectal and Spinoreticular Pathways:** These are ascending pathways that in turn lead

to *inhibition of pain transmission in the dorsal horn*. These pathways are an endogenous way of modulating pain.

- Alternative pathway for pain fibers: Nociceptive stimuli -----> **Periaqueductal Gray** of Midbrain and **Periventricular Gray** of Thalamus -----> Nucleus Raphe Magnus of the Reticular Formation.
- **Serotonergic Pathway:** From **Nucleus Raphe Magnus** the signal goes back down to Dorsal Horn of Spinal Cord -----> synapse with interneuron in Substantia Gelatinosa at all levels -----> inhibit the pain signal *at the point of entry into the spinal cord*.
- **Noradrenergic Pathway:** There is also a Noradrenergic pathway that has a modulatory effect on pain.
- *Stimulus-Produced Analgesia* can occur from electrical stimulation of the **periaqueductal gray**. Again, this analgesia has its effect by inhibiting pain transmission in Dorsal Horn.

- **OPIOIDS:**

- Inject very small amount of Morphine into one of two CNS regions to cause profound Analgesia:
 - Periaqueductal Gray
 - Dorsal Horn
- Opioid Receptors: **Enkephalins** and **Endorphins** are the endogenous ligands for these receptors. Opiates also bind to them but with higher potency.
 - **Opioid -Receptors** are found in Periaqueductal Gray.
 - **Naloxone** is an antagonist to this receptor.
- STRESS-INDUCED ANALGESIA: Extreme stress (Epinephrine) can induce analgesia so that a person can perform actions that would normally be painful. The action is not perceived as painful until after the stressful event is over.

RADICULAR PAIN: Pain localized to the dermatome of a dorsal root.

- Injury to a single Dorsal Root will not usually produce Anesthesia, because there is overlap between dermatomes.
- *Paresthesia* (tingling, etc.) is common however, and Radicular Pain often occurs with Paresthesia.

REFERRED PAIN: Visceral injury will send afferent pain information on the same nerves that also serve a cutaneous region. Because the brain is more used to getting sensory input from the cutaneous region of the nerve, the CNS will interpret the pain as originating from the cutaneous region.



[Return to top](#)

VISION

CONVEX LENS: Converging rays. They shorten the focal length and can be used to correct for farsightedness.

- *Positive Focal Length* -- focal point is in front of the lens.

CONCAVE LENS: Diverging rays. They lengthen the focal length and can be used to correct for nearsightedness.

- *Negative focal length* -- focal point is behind the lens.

LENS POWER: It indicates how much the lens can converge or diverge the light rays. *The stronger the power, the shorter the focal length.* A short focal length means the light is being bent a lot.

$$P \text{ (diopters)} = \frac{1}{\text{Focal Length (meters)}}$$

EMMETROPIE EYE: Normal vision, in which the light rays form an image on the retina.

- The natural eye has *two convex lenses* which serve to focus the light on the retina.
 - **Cornea**
 - **Crystalline Lens**
- ***The image on the retina is a "real" image -- upside down and inverted.***
- **ACCOMMODATION:** A **relaxed** lens is relatively flattened and lets you focus at a distance. **Accommodation** increases the curvature of the lens to focus for near vision.
 - Relaxed lens lets you focus at 63 mm from the surface of eye.
 - **Cycloplegics** are topical drugs you can put on the eye to inhibit the accommodation reflex and cause the lens to relax.
 - **PRESBYOPIA:** The loss of accommodation ability occurs with age. Children have up to 14 diopters of power to accommodate, while old adults may have only 1 or 2 diopters, or none.

MYOPIA, NEARSIGHTEDNESS: You can see things up close just fine but have difficulty seeing into the distance.

- The image forms in front of (*anterior to*) the retina.
- Corrective Lens: **Diverging (Concave) Lens**, to lengthen the focal length and move the image back a little so it forms on the retina.
- Other corrective procedures:
 - **Radio Keratotomy (RK):** Use fine diamond scalpel is used to make radial incisions in cornea, in order to flatten the cornea itself.
 - **Photorefractive Radio Keratotomy (PRK):** UV laser is used to blow off pieces of the cornea, a bit at a time.

HYPEROPIA, FAR-SIGHTEDNESS: You can see things far away just fine but have difficulty seeing things up close.

- The image forms behind (*posterior to*) the retina.
- Corrective Lens: **Converging (Convex) Lens**, the shorten the focal length and move the image forward a little so it forms on the retina.
- Other corrective procedures:
 - **Laser Keratothermoplasty:** Surgery that increases the curvature of lens.

ASTIGMATISM: A problem in which the *cornea has two different radii of curvature (one horizontal and one vertical) and two different focal points.* Essentially, the cornea is deformed such that the horizontal and vertical focal lengths don't match up with each other.

- **Torus** is donut-shaped thingie that has two radii of curvature: R_1 is the radius of the donut itself, and R_2 is the cross-sectional radius of the donut.
- Corrective Lens: **CYLINDRICAL LENS.** *The cylindrical lens is curved only in the horizontal plane -- not in the vertical plane.*
 - There is *no power in the vertical direction.*
 - Regular lenses can't be used because the lens would correct for the problem in one direction,

only to cause a distortion in the other direction.

- After correction, the patient may still be highly myopic. This can be further corrected with a normal spherical lens.

VISUAL ACUITY: The minimal angular resolution of the eye.

- **beta** = *the minimum angle of resolution of two dots*. The minimum subtended angle at which a patient can perceive two dots as distinct.
 - **Minutes** of arc are used to measure this angle, where 60 minutes = 1 degree. Degrees are too coarse of a measurement.
- Limit to Visual Acuity: The ultimate (theoretical) limit to visual acuity are the photo-receptors of the eye.
 - In the fovea (finest visual acuity), photoreceptors are separated by about **3 microns**, which corresponds to a theoretical minimum subtended angle of **0.6 minutes**. This theoretical minimum is never attained due to some **diffraction** of light in the eye.
- Factors that affect Acuity:
 - Pupil size. Small pupils result in better visual acuity.
- **SNELLEN EYE CHART:** Made of strokes and gaps, which in standard form should be separated by 5 units from each other.
- **20 / 20 (x / y) vision:**
 - **NUMERATOR:** The distance at which the patient stands from the chart. It is basically always 20!
 - **DENOMINATOR:** The *distance at which the letter would subtend 1 minute of arc for this patient*. This is equivalent to the distance at which an emmetropic eye could view the letter.
 - Due to the above, the relationship between minutes of arc and the Snellen Eye Chart is that the **distance = (20)(minutes of arc)**.
 - So, if you had 20 / 300 vision, then your minimum visual acuity would be 15 minutes of arc.

OPTICAL AXIS: Straight through the lens. It is slightly medial to the Visual Axis, which is where the fovea is located.

THE RETINA: From the back of the eye (outer limit of eye) to the inner most layer...

- **RETINAL EPITHELIUM:** Not officially part of the retina.
 - **Choroid** -- Not officially part of the retina. It is the blood supply to the retina.
 - **Retinal Pigmented Epithelium** -- Not officially part of the retina, support and recycling of rhodopsin disk-membranes.
- **PHOTORECEPTOR LAYER**
 - **OUTER SEGMENT:** Photoreceptor Cells, containing the photosensitive elements.
 - **INNER SEGMENT LAYER:** Continuation of Photoreceptor Cells, containing the mitochondria and other support organelles.
- **OUTER LIMITING MEMBRANE** really isn't a membrane, but consists of tight junctions between the cytoplasmic extensions of Muller Cells. It contains **MULLER CELLS** which are Retinal Glial cells.
 - Muller cells have their end-feet on both the outer and inner limiting membranes.
 - They regulate the extracellular K⁺ concentration in the environment.
 - They contain *Glutamine Synthetase*, necessary for metabolism of Glutamate, which is the main excitatory neurotransmitter in the visual system.
- **OUTER NUCLEAR LAYER:** Contains the *cell-bodies* of the photoreceptors.
- **OUTER PLEXIFORM LAYER:** Contains *synaptic connections* between the photoreceptor-cells

and the integrating neurons (amacrine and horizontal cells).

- **INNER NUCLEAR LAYER:** Contains the *cell-bodies of the integrating cells*. There are three integrating cell types:
 - Amacrine Cells
 - Horizontal Cells: Process lateral information.
 - Bipolar Cells: *This is the basic second-order neuron*. The "Standard Synapse" is photoreceptor cell -----> bipolar cell -----> ganglion cell.
 - Muller Cells: This cell extends almost the entire length of the retina.
 - Their tight junctions form the **external limiting membrane** on the outer surface of retina.
 - They extend all the way through retina, and parts actually lie on the internal limiting membrane.
- **INNER PLEXIFORM LAYER:** Contains synaptic connections between the integrating cells and the Ganglion Cells.
- **GANGLION CELL LAYER:** Contains the cell-bodies of the **Ganglion Cells**. They're afferent fibers make up the optic tract.
- **NERVE FIBER LAYER:** Unmyelinated axons of Ganglion cells.
- **INTERNAL LIMITING MEMBRANE:** Separates the neural retina from the Vitreous Body.

OPTIC DISK: Region of retina where the optic nerve and blood vessels enter. The optic disc is a blind-spot, but it is in a different part of the visual field for each eye so normally (with both eyes open) the blind-spot is not evident.

- You can see the Optic Disc through the Ophthalmoscope, and this is where you go to test for vascular problems or CNS problems, such as **Papilledema** which indicates CSF buildup.

FOVEA: Contains no vessels, no inner-nuclear layer, no ganglion cell layer, and *no rods*. Just a high concentration of **cone-photoreceptors**.

PRIMARY PHOTO TRANSDUCTION:

- **THE ROD CELL:** Specialized for low-acuity vision in the dark.
 - Rod cells are sensitive to blues and purples in the spectrum.
- **THE CONE CELL:** Specialized for high-acuity vision in the light.
 - **Red Cone Cell:**
 - **Protanomalous** means deficiency of red pigment.
 - **Protanopia** means total red color blindness.
 - **Green Cone Cell:**
 - **Deuteranomalous** means deficiency of green pigment
 - **Deutanomopia** means total green color blindness
 - **Blue Cone Cell:**
 - **Tritanomalous** means deficiency of blue pigment (rare)
 - **Traitanomopia** means total blue color blindness (rare)
- **OPSINS:** Rod cells contain Rhodopsin and Cone-cells contain Cone-Opsins.
 - Photon of Light CHANGES THE CONFIGURATION of **RETINAL** from **11-cis-Retinal** -- -----> **all-trans-Retinal**. This is the chemical basis for photo transduction.
 - The *trans*-Retinal then escapes the cone cells and goes back to the epithelium where it will get recycled.
- **IN THE DARK:** The rod-cell is depolarized.
 - High levels of cGMP are in the rod-cells.
 - **Na⁺-Ca²⁺-Channels** are open. The channel accommodates both Na⁺ and Ca²⁺, although

- Na⁺ is the major ion to come in.
- The cell is depolarized as Na⁺ continually comes in, and is pumped back out via Na⁺/K⁺-ATPase located in the inner segment.
- The cell is releasing excitatory neurotransmitter (**glutamate, aspartate**) onto the secondary neurons.
- IN THE LIGHT: The rod-cell becomes hyperpolarized.
 - cGMP gets cleaved to GMP by phosphodiesterase.
 - The Na⁺-Channels close.
 - Na/K-ATPase quickly restores the cell to what we normally think of as resting potential -- cell is hyperpolarized.
 - The cell stops releasing neurotransmitter.

SECONDARY CELLS: They receive signals from the photoreceptors.

- Bipolar Cells:
 - **ON BIPOLAR CELLS:** The CENTER of the cell depolarizes in the light.
 - This requires a **sign-reversal** of the membrane potential, since they receive a hyperpolarizing signal from the photoreceptors.
 - It will detect a light spot with a dark background.
 - **OFF BIPOLAR CELLS:** The CENTER of the cell hyperpolarizes in the light.
 - This does not require a sign-reversal. The membrane potential remains the same.
 - It will detect a dark spot with a light background.
- **CENTER-SURROUND INHIBITION:** The region immediately surrounding the center of the cell provides **contrast**, which makes it easier for the brain to interpret the visual stimuli.
 - When the center is stimulated, the SURROUND is *inhibited* by horizontal cells
 - When the SURROUND is stimulated, this indicates that light is nearby but not directly on the center. Thus the center is *inhibited* by horizontal cells.
 - **HORIZONTAL CELLS** are inhibitory interneurons that provide **lateral inhibition** to surrounding regions: Photoreceptor -----> Excitatory to Horizontal Cell -----> Inhibitory to the *neighboring* photoreceptor and bipolar regions (i.e. the "surround" area of visual field).
- **GANGLION CELLS:** Each Ganglion Cell has a **Receptive Field Center** which corresponds to specific Bipolar Cells, which in turn corresponds to specific photoreceptor cells.
 - **ON GANGLION CELLS:** They are stimulated by the light, i.e. by On Bipolar Cells.
 - **OFF GANGLION CELLS:** They are stimulated by the dark, i.e. by Off Bipolar Cells.
 - **CENTER-SURROUND ORG:**
 - If you shine a light in the center of an ON-GANGLION's receptive field, then it will be completely turned.
 - If you shine a light neatly around the periphery of an ON-GANGLION's receptive field, then it will be *completely turned off*.
 - The converse, of course, hold for the Off-Ganglion cells.
- X and Y SYSTEM is another way to divide up all the cells in the retina, according to their visual function.
 - **X-SYSTEM (P)** is specialized for high visual acuity and color information.
 - P stands for Parvocellular Layer of the Lateral Geniculate Nucleus.
 - **Y-SYSTEM (M)** is specialized for quick detection of motion, such as turning your head to glance at motion in the periphery.
 - M stands for Magnocellular Layer of the Lateral Geniculate Nucleus.
 - This system utilizes the **Amacrine Cells** which turn on with sudden changes in visual field, i.e. motion.

VISUAL FIELDS:

- Visual Field Organization with Retina:
 - **NASAL HALF:** The axons of the nasal (medial) half of the retina cross at the optic chiasm and go into the CNS via the contralateral optic tract.
 - **TEMPORAL HALF:** The axons on the temporal (lateral) half of the retina do not cross at the optic chiasm and go into the CNS on the ipsilateral optic tract.
 - **LEFT VISUAL FIELD:** Corresponds to the *nasal half* of your left retina and the *temporal half* of your right retina.
 - *All information in the left visual field winds up in the right hemicortex.*
 - **RIGHT VISUAL FIELD:** Corresponds to the *nasal half* of your right retina and the *temporal half* of your left retina.
 - *All information in the right visual field winds up in the left hemicortex.*
- VISUAL FIELD DEFICITS:
 - Terms:
 - **Homonymous:** The same half of each visual field is affected when either eye is tested.
 - This generally happens with *central lesions*, such as in the Occipital Lobe, or a complete loss of the Lateral Geniculate Nucleus.
 - **Heteronymous:** Opposite visual fields are affected when either eye is tested.
 - This happens with Pituitary Tumor.
 - **Hemianopia:** Loss of vision in one half of the visual field.
 - **Quadrantanopia:** Loss of vision in one quarter of the visual field.
 - **TUNNEL VISION:** No vision in the periphery, or in the *temporal visual fields*.
 - A **pituitary tumor** can impinge on the medial fibers of the optic chiasm -- the fibers that cross. That would be the *nasal halves* of each retina, corresponding to the temporal visual fields. Thus pituitary tumor can result in tunnel vision.
 - This lesion would be called a **BITEMPORAL HETERONYMOUS HEMIANOPIA**.
 - You can see tunnel vision happen with *infertility* because of the pituitary tumor.
 - **SUPERIOR QUADRANTANOPIA:** MEYER'S LOOP Unilateral damage to the ventral aspect of the Lateral Geniculate Nucleus, which contains a representation of the superior visual field, headed toward the inferior bank of the Calcarine Sulcus.
 - Meyer's Loop is the visual track going from LGN to the Visual Cortex. It travels around lateral edges of the lateral ventricles.
 - **HOMONYMOUS HEMIANOPIA WITH MACULAR SPARING:** Lesion to the Optic Radiations or Visual Cortex on one side.
 - Macular Sparing means that central (macular) vision still remains, but the rest of the vision is gone. Why this occurs is unknown.

LATERAL GENICULATE NUCLEUS:

- **MAGNOCELLULAR LAYER:** Layers 1-2. These layers process **Y-Cell** information from the retina -- sudden motion detection.
 - This is the *Where* System, telling you where things are in space. These neurons will ultimately project to the Parietal Lobe to help you orient "where" you are in your visual world.
- **PARVOCELLULAR LAYER:** Layers 3-6. These layers process **X-Cell** information from the retina -- visual acuity and color.
 - This is the *What* System. These neurons will ultimately send projections to Temporal Lobe (Wernicke's area, perhaps?) to help you identify what things are.
- Orientation: Different LGN layers process information from different eyes. All of the information

from the retina is kept in separate layers in the LGN, so it can be sent to distinct parts of visual cortex.

- Layers 1, 4, 6 process the *contralateral* eye.
- Layers 2, 3, 5 process the *ipsilateral* eye.
- Horizontal Orientation: Generally, information in each visual field is represented in contralateral LGN. As you move more laterally in the visual field, its representation moves more laterally in the contralateral LGN.

PRIMARY VISUAL (STRIATE) CORTEX: Area 17.

- Visual Field Organization: Each occipital lobe processes the contralateral visual field.
 - UPPER QUADRANT of each visual field is on the **lower bank** of the Calcarine Sulcus.
 - LOWER QUADRANT of each visual field is on the **upper bank** of the Calcarine Sulcus.
 - **Fovea:** The region of highest visual acuity is represented by the *most posterior (caudal) part of the Visual Cortex, and it has the largest (disproportionate) representation of neural tissue.*
- **MEYER'S LOOP: Optic Radiations** are the visual track going from LGN to the Visual Cortex. They form Meyer's Loop as they travel around the lateral aspect of the lateral ventricles.
 - Again, Meyer's Loop lesion leads to visual-field deficit of the contralateral quadrant.
 - Because of its anatomical arrangement, *damage to the temporal lobe can cause superior homonymous quadrantanopia.*
 - For similar reasons, *damage to the parietal lobe can cause inferior homonymous quadrantanopia.*
- CORTICAL COLUMNS: Layers of the Striate Cortex, from Pia to White Matter, are arranged into columns. All visual neurons enter the Visual Cortex through **Layer IV** of the cortex.
 - **ORIENTATION COLUMNS:** *All neurons in the same column will process will same visual-orientation information.*
 - For example, in the same column, all neurons may process vertically oriented lines or horizontally oriented lines, but not both.
 - **EYE-DOMINANCE COLUMNS:** *Any particular column will contain exclusively information from the left eye or the right eye.*
 - Left and Right-Eye Columns alternate with each other, so that a left eye-column of one orientation is right next to the right eye column for the same orientation.
 - **HYPER COLUMNS:** *The combination of the Left-Eye Column, Right-Eye Column, plus all possible Orientation-Columns for a single part of the visual field.*
 - Each hyper column contains a complete visual representation for a particular part of the visual field.
- **CYTOCHROME BLOBS:** Color processing. These blobs are present in each of the hyper columns, too.

EXTRA STRIATE CORTEX: Visual Association cortices.

- **Area V2:** The primary extra striate cortex; it receives lots of info from VI, the Striate Cortex. It processes depth information.
- **Area V5:** Processes information about motion.
- **Area V4:** Involved in **color** processing. It receives color information from V2.
- **Area IT:** Part of the *temporal lobe* involved with recognition of faces.
 - Lesion in this area results in **Prosopagnosia**, failure to recognize familiar faces.

Other Retinal Projections: Retinal projection that go somewhere other than the LGN.

- **Accessory Optic Nuclei:** Sensitive to movement of large visual fields and involved with the nystagmus reflex.
 - **Suprachiasmatic Nuclei:** Part of Hypothalamus; it receives light information in order to influence circadian rhythms.
 - **Superior Colliculus:** Involved with movement of eyes relative to visual stimuli and **foveation**, focusing, on a moving object.
 - There is a sensory map in the superficial layer that has 1:1 correspondence with a motor map in the deep layer. So, when one part of visual field is stimulated, the corresponding motor part directs the eyes to the visual stimulus.
 - **Pretectum:** Direct and consensual pupillary light reflexes. It receives bilateral information from the eyes, and projects bilaterally to the Edinger Westphal (CN III) nucleus.
-



[Return to top](#)

HEARING

Measuring Frequency and Volume:

- **SOUND-PRESSURE LEVEL (SPL):** A measure of the intensity (volume) of sound in Watts / m^2 . It is related to decibels logarithmically: a tenfold change in SPL corresponds to a linear increase of 20 decibels.
 - If the SPL is one million times (10^6) stronger, then the number of decibels is $6 \times 20 = 120$ decibel increase.
- **Threshold:** The least amount of stimulus energy (SPL) required for the ear to register a sound.
 - Threshold is *frequency dependent*. Some frequencies have a lower threshold (are more easily heard) than other frequencies.
 - Lowest threshold occurs at **1 - 3 kHz** frequency, which is the optimal pitch at which humans hear.
 - **Frequency Threshold Curve** shows the threshold SPL at different frequencies. The thresholds get higher (i.e. more difficult to hear) at high frequencies.
 - Because of this arrangement, *Sensorineural hearing loss will show a hearing-deficit at high frequencies before it will show it at low frequencies.*
- **DYNAMIC RANGE OF HEARING:** The range of *volume* that the human ear can detect without incurring damage. The range at 3 kHz is **0 - 120 dB**
- **AUDIOGRAM:** A frequency-threshold curve, in which the *deficit from normal* is graphed at each frequency range.

EXTERNAL EAR, **Pinna:**

- It *resonates* to increase the SPL of the sound *between 1 and 3 kHz*.
- It plays a role in *sound localization* -- its shape aids us in determining where a sound is coming from.

MIDDLE EAR: Air-filled cavity. Malleus -----> Incus -----> Stapes

- **Impedance Mismatching:** The middle ear bones amplify the sound vibrations from the **tympanic membrane** to the **oval window**.
- This amplification of sound gives us a **15 dB advantage**. *Removal of middle-ear ossicles does not*

result in deafness, but rather results in a 15dB hearing loss.

- **Tensor Tympani** and **Stapedius** contract reflexively in response to high intensity sound.
- Middle Ear Pathologies:
 - **Otitis Media:** Watch it with children. It will severely impair their language acquisition if they are chronically hard of hearing during those formative years.
 - **Otosclerosis:** Conductive Hearing Loss. Stapes no longer vibrates properly against oval window due to abnormal bone growth.

COCHLEA:

- **MODIOLUS:** The central "shaft" of the Cochlea, around which it screws.
 - *The modiolus contains the Cochlear Nerve (VIII)*
 - **SPIRAL GANGLION:** The starting point of the VIIIth nerve, it is in the Modiolus at the base of the Spiral Lamina.
- **SCALA MEDIA:** Endolymph fluid similar in composition to intracellular fluid (high in K⁺ and low in Na⁺). The Scala Media contains the sensory hair cells and the **Organ of Corti**
 - **TECTORIAL MEMBRANE** is *inside the scala media*, right on top of the hair cells.
 - FNXN: **Shearing Force** is created by movement of the Tectorial Membrane across the hair cells. Sound waves cause the tectorial membrane to move.
 - The Tectorial Membrane tends to move in an opposite direction as Basilar Membrane. This aids in the shearing force.
 - **SOUND TRANSDUCTION:** *This shearing force transduces the mechanical sound wave into an electrical stimulus.*
 - **STRIA VASCULARIS:** Highly vascular epithelium forming one wall of the Scala Media. It secretes endolymph into the Scala Media.
- **SCALA TYMPANI:** Perilymph fluid. In cross section, it is the section below each Scala Media, below the Basilar Membrane.
 - **BASILAR MEMBRANE:** It separates the Scala Media from the Scala Tympani. It forms the base of the Scala Media.
 - *The Organ of Corti, containing sensory hair cells, lies on the basilar membrane.*
 - **SOUND TRANSDUCTION:** *The Basilar Membrane moves upward, toward the Scala Vestibuli, to initiate the shearing of the Tectorial Membrane.*
- **SCALA VESTIBULI:** Perilymph fluid. In cross section, it is the section above each Scala Media, above the Vestibular Membrane.
 - **VESTIBULAR (REISSNER'S) MEMBRANE:** Very delicate membrane separating the Scala Media from the Scala Vestibuli.
 - The Scala Vestibuli is continuous with the **Oval Window**. It therefore conducts sound waves, through perilymph, toward the apex of the Cochlea.
 - **HELICOTREMA:** A hole at the *apex* of the cochlea. It connects the Scala Vestibuli to the Scala Tympani
 - It allows the Basilar Membrane to resting position in the event of constant pressure on the membrane.

ORGAN OF CORTI: It is located in the *Scala Media*, atop the Basilar Membrane.

- **INNER HAIR CELLS:** They are closer to the Modiolus. They are very tightly held into place.
 - **STRUCTURE:**
 - There is usually only one single row of inner hair cells. They have stereocilia that brush up against the Tectorial Membrane.
 - They are situated such that the **stereocilia** are in *endolymph* while the rest of the hair

cell is in *perilymph*.

- FNXN: These cells are the **primary sound receptors**. They respond to shearing movements of the Tectorial Membrane.
- Inner Hair Cells send primary **VIIIth Afferents** into the CNS, via the Spiral Ganglion.
- **OUTER HAIR CELLS:** There are more of them, located laterally, away from the tectorial membrane.
 - **STRUCTURE:** They have more room to breathe -- there is open space laterally.
 - They are in multiple rows.
 - Apex of the cells is embedded in **Reticular Lamina**. *This layer forms the ionic border between endolymph and perilymph.*
 - FNXN: These cells can move the *basilar membrane* and can "tune" the frequencies of the basilar membrane.
 - They receive **OLIVOCOCHLEAR BUNDLE (OCB) FIBERS** from the Superior Olivary Nucleus. This is efferent innervation that provides *both positive and negative feedback* to the hair-cell apparatus. It will affect *how sensitive the tectorial membrane is to mechanical transduction*.
 - **POSITIVE FEEDBACK:** The OHC's serve to *amplify* quiet sounds by making the Tectorial Membrane more sensitive to shearing.
 - **NEGATIVE FEEDBACK:** The OHC's serve to *dampen* loud sounds by making the Tectorial Membrane less sensitive to shearing.
 - The Outer Hair Cells also help in **frequency selectivity**. Loss of OHC's results in a **flattened tuning curve** -- VIIIth nerve afferents become less frequency-selective in the tones they carry.
 - **DISEASE:** Outer Hair Cells are subject to disease a lot. Commonly, the OHC's will be missing in a person but the inner hair cells will still be intact. Sensorineural hearing loss, but not total deafness, results.
 - Disease -----> Less Frequency Selectivity
 - Disease -----> *The patient is more sensitive to loud sounds.*
- **OTOACOUSTIC EMISSION:** Energy emitted by the Outer Hair Cells, which is a real sound and can be detected by the Cochlea. When such emissions are detected, Objective Tinnitus results.
 - **Objective Tinnitus:** Ringing in ear when a sound wave is actually present, being generated internally.
 - **Subjective Tinnitus:** Ringing in ear when no sound wave is present.

FREQUENCY SELECTIVITY: Sound is mapped to different parts of the Cochlea according to frequency. Each part of the cochlea is most sensitive to a small range of frequencies, i.e. it has the lowest threshold.

- **BASE:** Outer part of Cochlea transduces high frequency waves.
 - It is stiff and narrow, helping it to detect high frequencies.
- **APEX:** Inner part of Cochlea transduces low frequency waves.
 - Flaccid and broad, helping it to detect low frequencies.
- *Both systems are also energy dependent.* Frequency Selectivity still occurs with anoxia, but it isn't as well tuned.
- **Tuning Curve:** If you were to plot the frequency of sounds traveling on any particular VIIIth fiber, you would find that individual VIIIth afferents are also frequency selective, according to which hair cells they innervate.

ENDOLYMPHATIC POTENTIAL: +80mV Potential. The Scala Media is *positive* with respect to the hair cells, and with respect to the perilymphatic compartments.

- **DEPOLARIZATION:** Inner Hair Cell Stereocilia move toward Kinocilium -----> Open K⁺-Channels -----> **K⁺ enters the hair cells from the scala media** -----> depolarization and nerve firing result.
- **POTASSIUM:** It is unusual for this flow of K⁺ to result in depolarization. There is a large (+140mV) driving force for K⁺ to enter the cell, however.
- **Glutamate:** The IHC's probably use glutamate as an excitatory neurotransmitter to excite **VIIIth afferents** headed into the CNS.

HEARING LOSS:

- **Acoustic Neuroma:** The most common type of *CNS* hearing loss, i.e. damage to the VIIIth nerve itself.
 - **Bell's Palsy** is common secondary symptom, as VIIIth nerve neuroma can also compress the VIIth nerve in the inner ear.
- **Conduction Hearing Loss:** Otosclerosis or Otitis Media.
 - **WEBER'S TEST** can be used to distinguish conductive hearing loss from sensory hearing loss. Apply a vibrating tuning fork to the skull of a patient who has hearing loss in one ear.
 - If Conductive Hearing Loss: The sound will be louder to the patient in the *deficient ear*. Conduction through skull bypasses the conductive hearing loss problem./
 - If Sensorineural Hearing Loss: The sound will seem louder in the *other ear* -- the ear that doesn't have the sensorineural loss.
- **Sensorineural Hearing Loss:** The most common type of hearing loss. Usually the problem is the hair cells, and the VIIIth nerve fibers are left intact.
 - **CAUSES:**
 - Genetic, perinatal
 - High intensity sounds -----> IHC + OHC damage
 - Ototoxic Drugs: Aminoglycoside antibiotics, loop diuretics, some chemotherapeutic agents.
- **Presbyacusia:** Loss of hearing with age.

COCHLEAR IMPLANTS: Put in an implant that can provide direct stimulation to VIIIth-Nerve afferents. This can be done even if there is hair-cell deafness.

- This can restore some hearing but not usually speech perception. Best case scenario is speech perception is restored with practice.

MENIERE'S DISEASE:

- **TRIAD OF SYMPTOMS:** Patient will get ringing in ear, then a "fullness" in ear, then hearing loss will drop off, then vertigo.
 - **Tinnitus** (ringing in ear)
 - **Fluctuating Hearing Loss**
 - **Episodic Vertigo**
- **ETIOLOGY:** Idiopathic, Traumatic, Post-Syphilis, Viral
- **PATHOPHYSIOLOGY:**
 - **TRAUMA:** Damage to the Endolymphatic Sac; you can't absorb endolymph fluid -----> fluid overload.
 - **Vestibular Membrane ruptures** from buildup of inner fluid and pressure.
 - **Hair Cell Toxicity** then occurs from mixing of endolymph and perilymph fluids. Some cell death. They think the membrane can heal itself, hence resultant hearing loss is only

temporary.

- TREATMENT: Most common treatment is medicine designed to decrease the amount of inner ear fluid.
 - Salt balance / diuretics play a big role in treatment.
 - *Meclizine-Antivert, Valium, and Compazine* can all be used as Vestibular suppressants.
 - SURGICAL: Only if they don't respond well to medicine.
 - **Endolymphatic Shunt:** A surgical method that enhances the fluid-resorption of endolymphatic fluid.
 - **Vestibular Nerve Section** (not preferred) in the case of severe Vertigo. The problem is that this treatment is only symptomatic.
 - **Labyrinthectomy:** Removal of semicircular canals, resulting in complete destruction of *all of VIII* -- both Vestibular and Cochlear.
 - **Oscillopsia** is a terrible visual side-effect where people bounce up and down.
- PROGNOSIS: *Progressive, untreated Meniere's disease leads to irreversible hearing loss.* Early treatment is therefore essential.

CENTRAL AUDITORY PATHWAYS:

- Primary Sound Transmission: VIIIth Nerve -----> **Cochlear Nucleus** -----> Ventral and Dorsal Cochlear Nuclei -----> **Inferior Colliculus** where it SYNAPSES
- Secondary Sound Transmission: Inferior Colliculus -----> **Brachium of the Inferior Colliculus** ----> **Medial Geniculate Body** -----> **Primary Auditory Cortex** (Area 41).
- SOUND LOCALIZATION: The process of figuring out where in space a sound is originating from.
 - **SUPERIOR OLIVARY COMPLEX** receives **binaural** (both ears) input, processes it, and sends a modifying signal to the Inferior Colliculus indicating the source of sound.
 - **Lateral Superior Olive:** Sensitive to intensity differences between the two ears.
 - **Medial Superior Olive:** Sensitive to temporal (time) differences of signals coming in between the two ears.
 - **DUPLEX THEORY OF SOUND LOCALIZATION:** States that sound localization occurs by two different mechanisms, according to the frequency of the sound.
 - HIGH FREQ (greater than 3 kHz): The head casts a shadow on the sound wave, causing it to have a *different intensity* in each ear, which enables the auditory system to localize the source of the sound.
 - LOW FREQ (less than 3 kHz): The sound waves are traveling slow enough that they reach each ear at *different times*, enabling the Auditory System to localize the source of the sound.
- **Superior Colliculus** (not Inferior): Plays a role in sound processing, in paying attention to peripheral space (same role as in visual processing). The auditory information and visual information are parallel to each other and occupy different layers in the same cortical columns within the Superior Colliculus.

AUDITORY EVOKED POTENTIALS: Place electrodes on head to measure the actual electrical activity generated by signals in the Auditory pathway.

- It is used as a substitute for normal hearing tests when a hearing test isn't possible: examples = infants, invalids, **malingeringers** who behave like they are deaf when in fact they can hear.
- Can be used to diagnose some demyelinating diseases, such as Multiple Sclerosis, by studying the waveforms that are evoked in the auditory pathway.



[Return to top](#)

TASTE AND SMELL

TASTE BUDS:

- Papillae:
 - Circumvallate Papillae: Located on the border between anterior 2/3 and posterior 1/3 of tongue. They contain the highest concentration of taste receptors.
 - INNERVATION: The **Glossopharyngeal Nerve (CN IX)** which goes back to the **Petrosal Ganglion**
 - Foliate Papillae: Located on the sides of the tongue and toward the back. Sparsely populated with taste buds.
 - INNERVATION: **Facial Nerve (Chorda Tympani)** which goes back to the **Geniculate Ganglion**.
 - Fungiform Papillae: Most abundant. Located on Anterior 2/3 of tongue.
- Taste Bud Structure: *Regeneration of Gustatory cells occurs*, so if you burn your tongue the sensation of taste will return!
 - Receptor Cells have microvilli and are connected by tight junctions.
 - Supporting Cells are immature receptor cells.
 - Basal Cells are stem cells that produce new supporting cells, then new receptor cells.
 - Basal Cells -----> Supporting Cells -----> Receptor Cells
- INNERVATION:
 - Anterior 2/3: **Facial Nerve (VII)** -----> **Geniculate Ganglion**
 - Posterior 1/3: **Glossopharyngeal (IX)** -----> **Petrosal Ganglion**
 - Gustatory fibers on the **Epiglottis** are innervated by the **Vagus (X)** -----> **Nodose Ganglion**
 - **SOLITARY NUCLEUS** in **ipsilateral** brain steam receives afferents from all of the above.
- DEFICITS:
 - Conductive Taste Loss occurs with lack of saliva. Saliva, dissolving the chemicals in food, is required for taste.
 - Neural Taste Loss can occur, but it is rare because there are so many taste fibers.

ENSEMBLE ENCODING: Taste is perceived as a combination of the four basic tastes.

- **Bitter:** Back of the tongue
 - Uses IP_3 -----> Ca^{+2} as a **second messenger**.
- **Sour:** Posterior sides of the tongue.
 - Uses H^+ to block K^+ efflux -----> **direct depolarization**
- **Sweet:** Tip of the tongue
 - Uses **cAMP** as a **second messenger**
- **Salty:** Anterior sides of the tongue
 - Opens Na^+ -**Channels** -----> **direct depolarization**

OLFACTION:

- **Olfactory Cells:** They are actually, themselves, **neurons**. They are original in that regard.
 - **Basal Cells:** They differentiate directly into Olfactory Receptor cells, without the supporting cell intermediate.

- *Olfactory Neurons Regenerate.* Every time a new neuron is born, it must find its way through the **Cribriform Plate** and into the CNS.
- ENSEMBLE ENCODING: The system is even less organized than the taste system.
 - **Specific:** Each receptor cell binds to specific chemical stimuli.
 - **Sensitive:** Some receptors take an extraordinarily small amount of chemical stimuli to be activated.
- TRANSDUCTION: *There is no synapse between the olfactory cells and the CNS. The olfactory receptors go straight to the CNS.*
 - Depolarization of the receptor occurs by a second messenger system: G-Protein -----> cAMP
- OLFACtORY BULB: Olfactory Receptors travel up through the Cribriform Plate to form the Olfactory Bulb.
 - SYNAPSE occurs in Olfactory Bulb, in a **GLOMERULUS** which are little balls of neuropil.
 - **Mitral Cells** and **Tufted Cells**: They get the synapses. A single one of these cells may receive up to 1000 olfactory receptors.
 - **Periglomerular Cells** and **Granule Cells** are inhibitory interneurons in the Glomerulus. They may serve an adaptation (desensitization) role -- to ignore frequent stimuli.
- OLFACtORY TRACTS: The axons of the second-order Mitral and Tufted Cells.
 - They then branch to form the **Medial** and **Lateral Olfactory Stria**.
- CNS OLFACtORY PROJECTIONS: The Olfactory Stria generally go through the Limbic System or Paleocortical System -- not through the Thalamus.
 - **Anterior Olfactory Nucleus:** Part of the Olfactory Tract.
 - **Septal Nuclei:** They send signals -----> *Hypothalamus*.
 - **Piriform Cortex:** Also called Paleocortex. They send signals -----> *Entorhinal Cortex and Amygdala*.
 - **Amygdala:** Limbic. Involved in affective components of odor.
 - **Entorhinal Cortex:** Limbic. They send signal to -----> *Hippocampus* where it connects odors to **memories**, so that a smell is associated with a memory.
 - **Olfactory TuberclE:** Base of the Olfactory Tract, part of Paleocortex. They send signal -----> **Medial Dorsal Nucleus of Thalamus**. This is conscious perception of odors.
 - **ORBITOFRONTAL CORTEX:** It receives the signals from the Thalamus and is responsible for the conscious perception of odor.



SLEEP AND WAKEFULNESS

ELECTROENCEPHALogram (EEG): Electrodes are placed in five general regions bilaterally, over the five cortices.

- The EEG measures only cortical activity -- not subcortical activity.

Rhythm	Dominant Freq	Amplitude	State of Arousal
Beta	20 Hz	<i>Low Amplitude</i>	Alertness

	<i>High Frequency</i>		REM Sleep
Alpha	10 Hz high frequency	High	Alertness but with eyes closed; Relaxed wakefulness
Theta	3 - 7 Hz	High	Slow-Wave Sleep
Delta	0.5 - 3 Hz <i>Low Frequency</i>	High	Slow-Wave Sleep

SLEEP STAGES:

- **Stage 1:** Stage 1 and REM show the same patterns: **beta-waves**
- **SLOW-WAVE:** Low sympathetic tone, regular breathing, normal muscle tone.
 - **Stage 2:** Transitional sleep.
 - **Stage 3:** Deep sleep, **theta waves**
 - **Stage 4:** Deep sleep, **delta waves**
- **REM:** Rapid-Eye Movement.
 - Rapid eye movements, very low muscle tone (very still), high cortical activity (beta waves), **penile erection** is hallmark sign of REM sleep.
 - **Dreaming.** People aroused out of REM sleep remember their dreams vividly.
 - There is *active cortical inhibition of spinal muscles* thus resulting in low muscle tones. If it weren't for this active inhibition, then the subject would probably sleep walk or move during this period of sleep.
- Normal Sleep Progression: 1, 2, 3, 4, 3, 2, REM, 2, 3, 4, 3, 2, REM, 2, 3, 4 ... etc.
 - As the evening progresses:
 - Each period of REM becomes longer.
 - Deep Sleep (3 and 4) becomes shorter until it no longer occurs.
 - Arousal usually occurs out of REM sleep
- *Sleep is an active process. It is not simply the absence of consciousness.*

Alpha Waves: Relaxed Wakefulness with the eyes closed.

- The recording is most pronounced over the parietal and occipital lobes, and is least prevalent over the frontal lobe.

Synchronized -vs- Desynchronized:

- **Synchronized Waves** characterizes slow-wave sleep. Synchronous discharge of neurons.
 - **RETICULAR NUCLEUS of Thalamus** is one of the major source of synchrony in the brain. It projects neurons onto *itself* and releases **GABA** as an inhibitory neurotransmitter to cause synchronization of discharge.
- **Desynchronized Waves:** Neurons don't discharge at same time. Characteristic of arousal.

Definitions:

- **Sleep:** Normal, physiological alteration in consciousness and unconsciousness, which is freely reversible with appropriate stimulation.
- **Coma:** A state of irreversible unconsciousness.

- **Concussion:** Brief loss of consciousness after a blow to the head, with no permanent ramifications.
- **Syncope:** Massive, widespread anoxia of cortical neurons. Fainting.
 - It happens from a hyperactive Vagus nerve that stops the heart from pumping enough blood to the brain. Fainting relieves the hyperactivity of the Vagus, and everything return to normal.

CONSCIOUSNESS: Awareness of environment and self. It involves two systems.

- **RETICULAR ACTIVATING SYSTEM (RAS):** It is responsible for the **arousal** aspect of Consciousness.
 - Activity of Reticular formation and some brainstem nuclei, which involve the **Reticular Formation and Diffuse Thalamic Nuclei**
- **CEREBRAL CORTEX:** It is responsible for the **content** aspect of consciousness.
- Anatomical Lesions that can produce coma:
 - **Posterior Fossa:** All of the subcortical structures, caudal to the Tentorium Cerebelli. A lesion to the posterior fossa will damage the Reticular Formation and can thus result in coma.
 - **Uncal Herniation** can press up against Tegmentum causing coma.
 - Metabolic Encephalopathy.
- Tonic -vs- Phasic Modes of Consciousness:
 - **TONIC MODE:** Characteristic of Wakefulness and REM sleep.
 - *High frequency (beta), continuous discharge of action potentials.*
 - Na^+ channels cause the action potentials.
 - Tonic mode has a resting potential that is just a few millivolts depolarized from phasic mode.
 - **PHASIC MODE:** Characteristic of Slow-Wave sleep.
 - *Rhythmic, low frequency discharge of action potentials:* An AP followed by 100 to 200 msec of silence.
 - Ca^{+2} channels cause these action potentials.
 - Phasic mode is just a few millivolts hyperpolarized from Tonic Mode.
- Neurohormonal Systems of Arousal and sleep:
 - **CHOLINERGIC SYSTEM:** Raises the resting potential of thalamic and cortical neurons by a few millivolts and is related to the RAS.
 - **NUCLEUS BASALIS (of Meynert):** Basal forebrain.
 - **NORADRENERGIC SYSTEM:** Raises the resting potential of thalamic and cortical neurons by a few millivolts and is related to the RAS.
 - **LOCUS CERULEUS,** in the Midbrain, releases NorE.
 - **SEROTONERGIC SYSTEM:** Related to *sleep* and to the pain-control system.
 - Serotonin induces slow-wave sleep and comes from two-sources:
 - (Pain Control Pathway): Periaqueductal Gray -----> **Nucleus Raphe Magnus** --> Dorsal Horn of Spinal Cord to inhibit pain. This pathway is also involved in causing sleep.
 - **Preoptic Area (POA) of Hypothalamus:** It also releases Serotonin.

EPILEPSY: *Two or more unprovoked seizures.*

- **SEIZURE:** Abnormal behavior resulting large amplitude hypersynchronous neuronal discharge.
- **ETIOLOGIC CLASSIFICATION of SEIZURES:**
 - **Acute Symptomatic:** *Non-Epileptic* seizures caused by some explainable, reversible condition such as *hypernatremia* (high blood Na^+).

- By definition, acute symptomatic seizures are *reversible*.
- **Remote Symptomatic:** Epileptic seizures caused by physical or metabolic trauma, such as an automobile accident, which resulted in incurable Epilepsy.
 - By definition, *remote symptomatic seizures are irreversible*.
- **Idiopathic:** Genetic or unexplained Epilepsy.
- BEHAVIORAL CLASSIFICATIONS OF SEIZURES:
 - **Generalized:** *At Onset*, a seizure that starts by virtually all neurons of the cortex synchronously discharging.
 - **Tonic-Clonic:** Alternative behavior of stillness (tonic) followed by rocking and jerking (clonus).
 - **Abscence:** Brief
 - **Partial:** *At Onset*, a seizure that starts by a localized region of cortex discharging.
 - **Secondary Generalization:** Partial seizures can and often do spread to become generalized seizures. But, they are classified by *how the seizure starts*.
 - **Simple Partial:** The patient has full recollection of the seizure. The patient does not have retrograde or anterograde amnesia surrounding the event.
 - **Complex Partial:** The patient does not recall the seizure or the events immediately preceding it. Retrograde and possible Anterograde amnesia.
- **Dilantin** is a drug that is effective in treating partial seizures.

SLEEP APNEA:

- SYMPTOMS: Excessive daytime sleepiness, resulting from waking up many times during the night due to obstructed airways.
 - Hypercapnia, Hypoxia, Compensated Respiratory Acidosis, morning headaches.
 - Complications: Cor Pulmonale (Right-Heart Failure), Pulmonary Hypertension, Systemic Hypertension
 - Inordinately loud snoring
- Types:
 - **Obstructive Sleep Apnea:** Sleep Apnea due to obstructed or collapsed upper airways. Essentially a respiratory problem.
 - Most common type: 98% of cases.
 - Respiratory effort is still evident: expanded chest, etc.
 - Snoring only occurs with this form.
 - **Central Sleep Apnea:** Sleep Apnea due to a loss of the CNS drive to breathe -- i.e. sleep somehow inhibits the Phrenic Nerve from firing on the diaphragm.
 - This is much rarer and is essentially a *neurological* problem.
 - Snoring is absent.
- Treatment:
 - **Continuous Positive Airway Pressure (CPAP):** Physically keep upper airways open, for obstructive apnea.
 - Tracheostomy, or procedures to surgically widen the space in the nasopharynx are also options.
 - **Medroxyprogesterone** can be given to *women* to stimulate breathing in the case of central apnea.



[Return to top](#)

THE HYPOTHALAMUS

Anatomy of Hypothalamus: The Hypothalamus forms the walls of the **Third Ventricle**.

- Borders:
 - **Optic Chiasm:** Anterior-Ventral border
 - **Anterior Commissure:** Posterior-Rostral border.
 - **Mamillary Complex:** Forms the Ventral-Caudal border. It is very easy to see on the gross-brain but we don't know its function.
- BLOOD SUPPLY:
 - **Hypothalamic Arteries:** They come off the Circle of Willis.
 - **Superior Hypophyseal Artery:** Supplies the basal hypothalamus (**median eminence**) and then goes on to supply the **Adenohypophysis** (anterior pituitary).
 - So, the anterior pituitary gets blood that came directly from the hypothalamus. It receives no direct arterial blood of its own.
 - **Inferior Hypophyseal Artery:** It supplies the **Neurohypophysis** (posterior pituitary).
 - So, the posterior pituitary gets fresh arterial blood, rather than blood that has come from hypothalamus.
 - **Hypophyseal Vein:** The neurohypophysis dumps its contents (oxytocin, ADH) out of hypothalamic nerves, and into the hypophyseal vein, where it then makes its way into the general circulation.

CONNECTIONS to Hypothalamus: Most of the neural connections with the Hypothalamus are part of the Limbic System.

- **MAMMOTHALAMIC TRACT:** Mamillary Complex (posterior part of Hypothalamus) -----> *Anterior Thalamus*
 - These fibers are heavily myelinated and constitute a major nerve tract.
- **FORNIX:** *Hippocampus* -----> Hypothalamus (Pre-Optic Area) -----> Mamillary Bodies
- **STRIA TERMINALIS:** *Amygdala* -----> Hypothalamus
 - This tract also goes from Amygdala to other area of the brain.
- **MEDIAL FOREBRAIN BUNDLE:** *Locus Ceruleus* of Brainstem -----> Hypothalamus
 - This is the only place in the brain where **Norepinephrine** is used as a Neurotransmitter. All the nerves come from the Locus Ceruleus.
 - The Locus Ceruleus also sends NorE neurons to other locales, but the Medial Forebrain Bundle is the one that goes to the Hypothalamus.

POSTERIOR PITUITARY PATHWAY: **Paraventricular Nucleus** -----> **Superoptic Nucleus** -----> **(Arcuate Nucleus** -----> **Infundibulum**) -----> **Neurohypophysis**

- **MAGNOCELLULAR NEURONS:** Neurons that follow this path and make the hormones Oxytocin + ADH.
 - These neurons have larger cell bodies.
- **PARVICELLULAR NEURONS:** These neurons have smaller cell bodies and make the small peptides (releasing factors) that control the adenohypophyses.

NUCLEI, PARTS, and FUNCTIONS OF THE HYPOTHALAMUS:

- **Mamillary Bodies:** The most caudal part of the hypothalamus.
- **Pre-Optic Area:** The most anterior part of the Hypothalamus, near the optic chiasm.
- **Paraventricular Nucleus:** Above the pre-optic area, surrounding the third ventricle on either side.

The sites of origin of the Neurohypophyseal hormones.

- **Superoptic Nucleus:** Near the paraventricular nucleus. *The sites of origin of the neurohypophyseal hormones* (Oxytocin, ADH).
- **Suprachiasmatic Nucleus:** Right above the optic chiasm, it regulates circadian rhythms.
- **Arcuate Nucleus:** Sits right above the pituitary gland. All pituitary factors pass through this nucleus before reaching the pituitary.
 - **Median Eminence** is next to Arcuate Nucleus, and is where a lot of the neurons terminate on blood vessels.
- **Ventral Medial Nucleus:** Important in feeding behavior and emotional behavior.
 - Lesion of this nucleus can result in Rage, as Amygdala becomes dominant to Hypothalamus.
 - This nucleus contains **the satiety center** which tell you when you are full.
- **Dorsal Medial Nucleus:**

Functions of Hypothalamus:

- ENDOCRINE: Hypothalamus is the ultimate homeostasis machine.
- AUTONOMIC: Hypothalamus plays important roles in regulating autonomic system.
- EMOTION: Hypothalamus does not originate emotions, but it integrates them. Emotion has two mental components.
 - **Cognition:** An awareness of what is going on in your environment.
 - **Conation:** The urge to act on feelings.
 - RAGE EXPTS: Lesion of *Ventral Medial Nucleus* results in uncontrolled rage. This is because there is a BALANCE between Hypothalamus and Amygdala in controlling emotional behavior
 - **Amygdala** dominant (as in above) -----> RAGE results.
 - **Hypothalamus** dominant (as in a lesioned Amygdala) -----> PLACID, FLAT behavior results.
 - **KLÜVER-BUCY SYNDROME** results when both Amygdala and Hypothalamus are lesioned.
 - Extremely docile, eat a lot, hypersexual.
 - SEX BEHAVIOR: **Androgens** play primary role in regulating sexual behavior. At least that's true in animals. Humans, it may be less true as higher cortical functions are more dominant.
 - There are androgen-receptors in both hypothalamus and amygdala.
- MOTIVATIONAL STATES
 - FEEDING:
 - **Lateral Hypothalamus** is the HUNGER CENTER. Stimulate it and you eat.
 - **Ventromedial Nucleus** is the SATIETY CENTER. Stimulate it and you feel full.
 - It has some **CCK-Receptors**, and this may play role in satiety. There are also some local CCK neurons, and they don't know whether the CCK is originating locally or from the digestive system.
 - THIRST: Two primary determinants of thirst. **Subfornical Organ**, in the wall of third ventricle, is the primary thirst-inducer.
 - *Tissue Osmolarity:* High osmolarity in hypothalamus stimulates the sensation of thirst. Subfornical organ has **osmole receptors** to sense this.
 - *Blood Volume:* Low blood volume stimulates thirst. Subfornical organ has **Angiotensin II receptors** to sense low blood volume.
 - Fever and dry mouth also have an effect.
- TEMPERATURE REGULATION: The hypothalamus contains the thermostat for regulating our body temperature.
 - **Anterior Hypothalamic Area** is the Warm Sensitive area. *It tries to get rid of heat*

(sweating and vasodilation)

- Loss of the Anterior Hypothalamus result in Hyperthermia.
 - **Posterior Hypothalamic Area** is the Cold Sensitive area. *It tries to create more heat (shivering)*
 - FEVER: Bacterial infection releases endotoxins that causes release of interleukins by circulating blood cells, especially **IL-1**. In the brain, IL-1 causes the hypothalamus to reset the thermostat to a higher level.
 - CIRCADIAN RHYTHMS: Mediated by Suprachiasmatic Nucleus.
 - Temperature regulation: You have higher temperature in morning, and while you're awake, than when you are sleeping.
 - **Melatonin** has a circadian rhythm: It is released at night, during sleep, but not during day.
 - **ACTH / Cortisol** has circadian rhythm.
-



[Return to top](#)

HIGHER CORTICAL FUNCTION

ALLOCORTEX: All cortical structures below the **Rhinal (Collateral) Sulcus**. It contains the following (The **neocortex** is everything else)

- Hippocampus
- Parahippocampal Gyrus
- Entorhinal Cortex

CORTICAL NEURONS:

- **Pyramidal Cells** have long axons that project to other lobes of the brain or to subcortical structures.
 - They are excitatory neurons and use **glutamate** as neurotransmitter.
- **Fusiform Cells** have long axons
- **Granule (Stellate) Cells** are interneurons with short axons.
 - These neurons project to neighboring neurons in the same region of the brain.
 - These interneurons can be either excitatory (glutamate) or inhibitory (GABA)

CORTICAL LAMINA: Layers of the Cerebral Cortex

- Layers I-III: SUPRA GRANULAR LAYERS, most superficial layers.
 - Cortico-cortical inputs (associations) terminate primarily in one of these layers.
- **LAYER IV: GRANULAR LAYER**. It contains granule cells.
 - *Thalamic projections* (subcortical information) terminates primarily in this layer.
- Layer V: Output in this layer projects primarily to the Brainstem.
- Layer VI: Deepest layer, next to white matter. Output in this layer projects primarily to Thalamus.

CYTOARCHITECTURE:

- **GRANULAR CORTEX:** The primary sensory regions of the cortex, which cytologically contains granules: Somatosensory (Brodmann 1-3), Visual (17), and Auditory (41).
- **AGRANULAR CORTEX:** The motor regions of the cortex (4), which do not contain granules.

BRODMANN'S AREAS	
Area 3a, 3b, 1, 2	Primary and secondary somatosensory cortices
Area 17	Primary Visual (Striate) Cortex
Area 41	Primary Auditory Cortex
Area 42	Secondary Auditory Cortex (Wernicke's Area)
Area 4	Primary Motor Cortex
Area 6	Premotor Cortex
Area 8	Frontal Eye Fields
Area 5, 7	Posterior Parietal Cortices

ASSOCIATION CORTICES: The sign of higher intelligence. There are three association cortices.

- **POSTERIOR PARIETAL ASSOCIATION CORTEX:** The convergence of somatosensory, visual, and auditory association cortices. This is basically the area where we process special-sensory stimuli.
- **LESIONS = Neglect Syndrome, Astereognosias, Aphasias.**
- **PREFRONTAL ASSOCIATION CORTEX:** Short-Term Memory, foresight, and judgment.
 - SHORT-TERM MEMORY EXPT: Lesion in this area in monkeys, and they cannot select food under an opaque cup that was just placed there ten seconds ago.
 - **Dopamine** is a prominent
- **LIMBIC ASSOCIATION CORTEX:** Memory, olfaction, and emotion.
 - **Cingulate Cortex** Lesion -----> **Sham Rage** in monkeys. Motor actions of rage without provocation.
 - **Orbitofrontal Cortex** Lesion -----> Decrease normal aggressiveness and emotional response.
 - **Frontal Lobotomies**, historically, were attempts to flatten behavior by lesioning the orbitofrontal cortex.
 - **Inferior Temporal Lobe:** This portion of limbic association cortex is concerned with long-term memory. Stimulation of this region resulted in vivid hallucinations of past experiences.
 - **KLÜVER-BUCY-SYNDROME:** Lesion of the *inferior temporal lobe* results in hypersexuality, oral exploration, and excessive hunger in monkeys.

CORTICAL HEMISPHERES:

- **DOMINANT HEMISPHERE:** Is defined as that one which contains specialized language areas, **Wernicke's Area**. Wernicke's area will be *larger* on the dominant side.
 - The relationship between hemispheric dominance and handedness is complex. They often correspond but not always.
 - For right or left-handed individuals, the left hemisphere is most often dominant, then codominance. Right-hemisphere dominance is least common.
- **SODIUM AMYTAL TEST:** Test to see which hemisphere is dominant. Put this barbiturate into each of the Carotid Arteries, and have the subject count down from 10. When you have injected it

- into the dominant hemisphere, the subject will stop counting.
- **CORPUS COLLOSECTOMY:** Split the interconnections between left-brain and right-brain, such that left and right-brain function cannot communicate. If a subject is presented an apple in exclusively one visual field or the other:
 - RIGHT VISUAL FIELD -----> Left Visual Cortex -----> the subject is able to verbally identify the object as an apple.
 - LEFT VISUAL FIELD -----> Right Visual Cortex -----> The subject could not identify it as an apple, even though he knew it was, because he had no access to language acquisition.
 - The subject was still able to pick it out from among different objects or identify it in other non-verbal ways.
 - **NON-DOMINANT (RIGHT) HEMISPHERE:**
 - Contains lots of association areas
 - Concerned with spatial-perceptual orientation
 - Facial recognition.

Language:

- **APHASIAS:** Disturbances of language production, comprehension, or both.
 - This is not a strict motor problem, but one where problem lies in higher cortical centers.
 - Some strictly motor problems:
 - **Dysarthria:** Inability to articulate language. Strict motor deficit.
 - **Dysphonia:** Lack of control over vocal cords. Strict motor deficit.
 - **WERNICKE'S APHASIA:** Deficit in comprehension of language.
 - Empty Speech: Verbal output is fluent but it is meaningless.
 - **BROCA'S APHASIA:** Deficit in forming meaningful language.
 - Verbal Output is possible but it is not fluent.
 - Verbal comprehension is normal.
 - **CONDUCTION APHASIA:** Damage to the Arcuate Fasciculus, which interconnects Broca's and Wernicke's areas.
 - Speech comprehension is good, but incorrect words may be used.
 - Impaired writing.
- Language Areas:
 - **Wernicke's Speech Reception Area (42):** Speech comprehension.
 - Posterior Superior Temporal Lobe
 - **Broca's Motor Speech Area:** Speech formation; formation of fluent, meaningful speech.
 - **ARCUATE FASCICULUS:** The pathway that interconnects the two above.
- **WERNICKE-GSCHWIND MODEL:** Describes the neural pathway by which a person reads a word and then vocally says what the word is. The pathway goes in the following order:
 - *Visual Stimulus -----> -----> Visual Cortex -----> Visual Association Cortex -----> Angular Gyrus (in Parietal Association cortex):* This step turns the written word into an auditory signal. Language is stored primarily as an auditory signal.
 - *Angular Gyrus -----> Wernicke's Area:* Interpretation of what the auditory signal means
 - *Wernicke's Area -----> Arcuate Fasciculus -----> Broca's Area:* speech formation signals are generated
 - *Broca's Area -----> Facial part of motor cortex:* The word is vocalized.
- **ALEXIA:** Inability to read, resulting from damage to **Angular Gyrus**.
- **APROSODIA:** Deficit in singing, intonation, or inflection of voice. A flat affect to the voice.
 - This results from a lesion to the *speech-area on the non-dominant side*, which is responsible for emotional inflection of voice.
- **AGRAPHIA:** Inability to write. Also from damage to **angular gyrus**.
 - This can occur when visual symbols cannot gain access to the language system.

- **APRAXIA:** Inability to perform learned motor movement, such as a saluting or flipping a coin.
 - This occurs from a Disconnection Syndrome and involves disruption of pathways going from *Wernicke's Area* to *Premotor Cortex*.

LEARNING AND MEMORY:

- **REFLEXIVE MEMORY:** Remembering how to do something, like riding a bike.
 - **CONDITIONING:** Classical condition is a form of reflexive memory. Pavlov's dog salivates when he hears the bell rings, after constantly being presented with a bell followed by food.
 - **Conditioning Stimulus:** The bell
 - **Unconditioned Stimulus:** The food
 - **CEREBELLAR NUCLEI** have been discovered to play a role in reflexive memory.
 - Operant Conditioning: Conditioning of behaviors based on the consequences (reward / punishment).
 - **DECLARATIVE MEMORY:** Conscious memory and recall.
 - **TWO-STAGE MODEL** of Memory Processing: Memories are formed by putting information in **short-term memory** into **long-term memory**.
 - **HIPPOCAMPUS** is known to perform the function of consolidating short-term memory into long-term memory.
 - Lesion of Hippocampus causes **anterograde amnesia**, inability to remember things after the point of injury.
-



[Return to top](#)

THE LIMBIC SYSTEM

Anatomical Structures of the Limbic System:

- **NEOCORTEX:** Two structures located lateral, rather than medial, to the Sylvian Sulcus.
 - **DORSOLATERAL CORTEX:** *Pre-Frontal Association Cortex*.
 - INPUT: Dorsomedial Nucleus of the Thalamus.
 - OUTPUT: It projects to the Entorhinal Cortex
 - FNXN: It has marginal association with the Limbic System. It is involved in motivation and in the planning of emotions.
 - **ORBITOFRONTAL CORTEX:** *It is involved in the motor events associated with emotion.*
 - INPUT: Dorsomedial Nucleus of the Thalamus.
- **ALLOCORTEX:**
 - **CINGULATE GYRUS:** PALEOCORTEX (most primitive)
 - FNXN: Integral part of Papez' Circuit
 - INPUT: Anterior Nucleus of the Thalamus.
 - **PARAHIPPOCAMPAL GYRUS:** PALEOCORTEX (most primitive)
 - **HIPPOCAMPAL FORMATION:** The only structure that is exclusively part of limbic system. Locale is at the base of the Temporal Lobe.
 - Order of Signal: *Entorhinal Cortex -----> Dentate Nucleus -----> Hippocampus*
 - **HIPPOCAMPUS:** Receives input directly from Dentate Gyrus
 - Structure: Three layers containing **Pyramidal Cells**
 - Molecular Layer: Receives input from Dentate Nucleus
 - Pyramidal Cell Layer: Pyramidal cell bodies.

- Polymorphic Layer: Output through Fornix or to other Hippocampal structures.
- **Anoxia:** *The hippocampus is extraordinarily sensitive to Anoxia.*
- **OUTPUTS:** The hippocampus has several outputs.
 - Back to Neocortex: *Consolidation of Short-Term Memory into Long-Term Memory.*
 - Fornix -----> To Hypothalamus: Modulate autonomic / hypothalamic behavior.
 - Septum: A way station for information headed to the hypothalamus.
- FNXN: Hippocampus general function is to ***consolidate short-term memory into long-term memory***
 - LESION of only hippocampus results in anterograde amnesia. Patient would be no longer able to remember new things, but previous memories would remain intact.
- **DENTATE NUCLEUS:** Receives input directly from Entorhinal Cortex
 - Structure: Three layers containing **Granule Cells**
 - Molecular Layer: Outer layer, receives input from Entorhinal Cortex
 - Granule Cell Layer: Granule Cell bodies
 - Polymorphic Layer: Inner Layer, sends output to the Hippocampus
- **SUBICULUM:**
- **ENTORHINAL CORTEX:** In Papez' The primary input to the Hippocampus.
 - INPUT: *It receives direct olfactory input, as well as association input for the other senses.*
 - OUTPUT: *The Dentate Nucleus.*
- **AMYGDALA:** Attaches the subjective components of emotions and feelings to autonomic components. Amygdaloid Nuclei:
 - **CORTICOMEDIAN GROUP:** Processes Olfactory Information
 - *Corticomedian Group -----> Hypothalamus*
 - *Corticomedian Group -----> Stria Terminalis -----> Septum*
 - **BASOLATERAL GROUP:**
 - *Basolateral Nuclei <=====> Neocortex*
 - *Basolateral Nuclei -----> Dorsomedial Nucleus of Thalamus -----> Pre-Frontal Cortex*
 - *Basolateral Nuclei -----> Nucleus Accumbens*
 - **CENTRAL NUCLEUS:** Main cells that provide info to the hypothalamus.
 - Some info also sent to brainstem nuclei in Pons.
 - **TWO MAIN OUTPUTS:** *The Hypothalamus and Stria Terminalis.*
- **DIENCEPHALON:**
 - **FORNIX:** The tract over which the Hippocampus sends information to the Hypothalamus.
 - **SEPTUM:** A way station between the Hippocampus and Hypothalamus.
 - INPUT: Receives input from the Amygdala and Hippocampus.
 - OUTPUT: Sends info to the Hypothalamus
 - **Nucleus Raphe Magnus** (Serotonin) and **Locus Ceruleus** (Norepi) synapse on the Septum in a "diffuse" way and can therefore influence its output.
 - **ANTERIOR NUCLEUS, THALAMUS:**
 - OUTPUT: It sends output to the Cingulate Gyrus, in Papez' Circuit.
 - **MEDIAL DORSAL NUCLEUS, THALAMUS:**
 - OUTPUT: It sends output to the neocortical structures, Orbitofrontal and Dorsolateral Cortices.
 - INPUT: Receives motor information from the Ventral Globus Pallidus of Striatum.

- **HYPOTHALAMUS:**
- **STRIATUM (BASALGANGLIA):** Modulates motor activity in the Limbic System.
- **NUCLEUS ACCUMBENS:**
 - CLOSED CIRCUIT: *Nucleus Accumbens -----> Globus Pallidus -----> Dorsomedial Nucleus of Thalamus -----> Orbitofrontal Cortex*
 - This circuit modulates the motor behaviors associated with emotion.
 - It is referred to as a "closed circuit"

PAPEZ CIRCUIT: The fundamental interaction between *higher sensory input* from the cortex and *more primitive functions* from the sub-cortex and the rest of the body.

- **POSTERIOR PARIETAL ASSOCIATION CORTEX:** Information that enters Papez' Circuit originates from the sensory association cortex (combination of visual, auditory, somatosensory)
 - **OLFACTION:** Olfactory information, on the other hand, enters the Limbic System directly.
- **INCOMING SIGNAL:** *Association-Cortex -----> Cingulate Gyrus -----> Hippocampal formation -----> Entorhinal Cortex (synapse) -----> Hippocampus*
- **OUTGOING SIGNAL:** *Hippocampus -----> Fornix -----> Mamillary Bodies -----> Mammillothalamic Tract -----> Anterior Nucleus of Thalamus*

KLÜVER-BUCY SYNDROME: Hypersexual horniness. *Lesion of the Amygdala and Hypothalamus.*

SCHIZOPHRENIA: A splitting of the cognitive with the emotional aspects of behavior.

- **DOPAMINE:** Two Dopaminergic systems are implicated in Schizophrenia.
 - **MESOLIMBIC SYSTEM:** Provides Dopaminergic neurons to the Nucleus Accumbens.
 - **MESOCORTICAL SYSTEM:** Projects dopaminergic neurons to PreFrontal Cortex.
- **POSITIVE SYMPTOMS:** *Overproduction of Dopaminergic Mesolimbic Neurons -----> over expression of limbic system*
 - This problem could be treated with **dopamine antagonists**
 - Positive Symptoms = hallucinations and uncontrolled emotions
- **NEGATIVE SYMPTOMS:** *A decrease in dopaminergic output of the mesocortical system -----> pre-frontal cortex.* They think this may be involved.
 - This problem could theoretically be treated with dopamine itself, but that treatment doesn't work.
 - Negative Symptoms = lack of motivation, lack of planning, lack of foresight



[Return to top](#)

Copyright 1999, Scott Goodman, all rights reserved