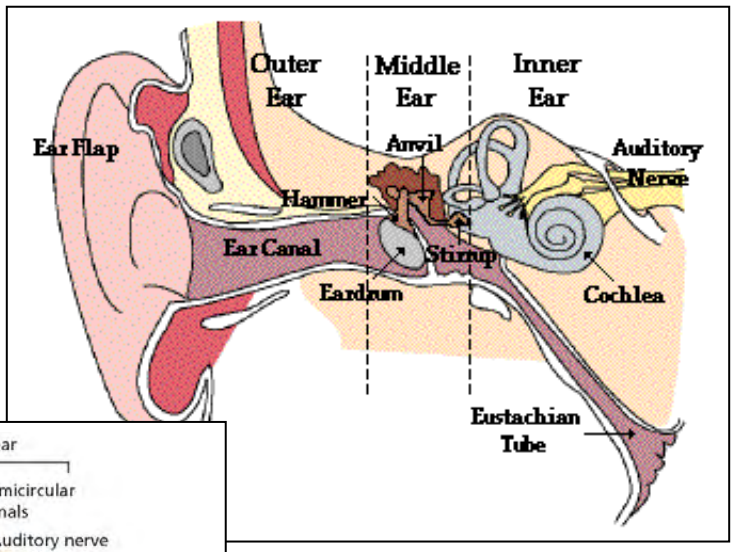
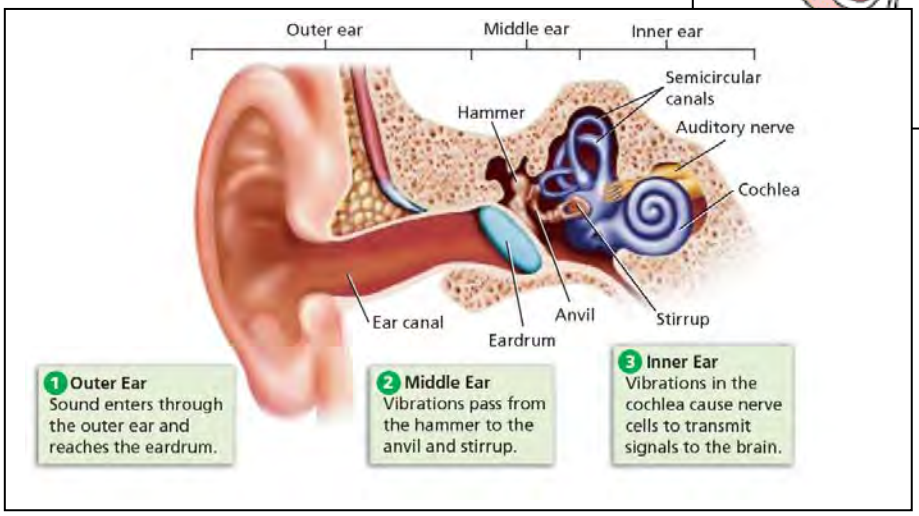
EARS – HEARING AND BALANCE/EQUILIBRIUM

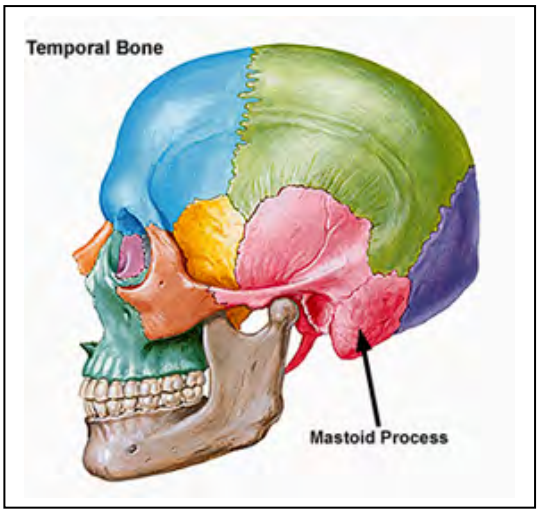
Parts of the ear:

- external, middle, inner
- The **mastoid process** of the temporal bone of the skull contains air-filled sinuses, called air cells, promoting conduction of sound from external to middle ear

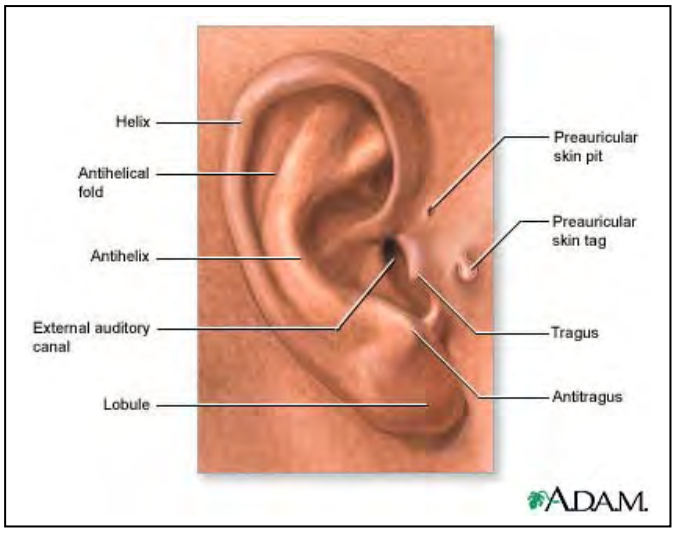


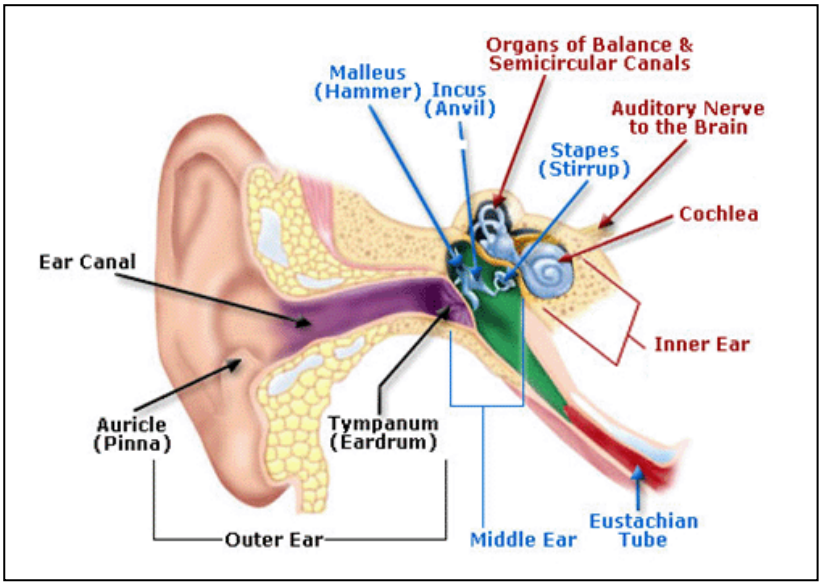


Temporal bone & mastoid process



External Ear





Outer ear:

- pinna (auricle)
- external auditory canal (EAC)
- tympanic membrane (eardrum)

Middle ear:

- eardrum (tympanic membrane)**(TM)**
- tympanic cavity (in temporal bone)
- containing three **ossicles** (small bones) that transmit **vibration**

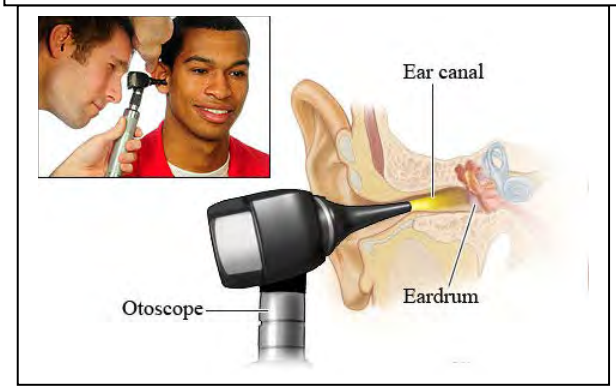
Inner ear: structures and nerve pathways for both HEARING and BALANCE/EQUILIBRIUM

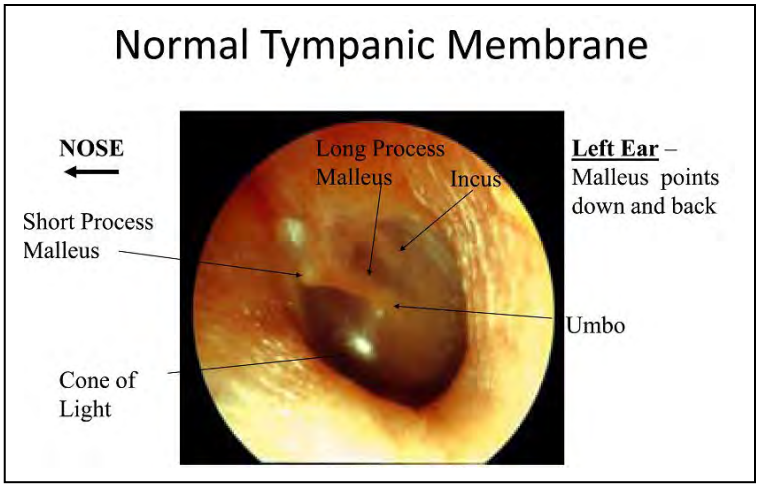
- Hearing:
  - **Cochlea** contains the **Organ of Corti** with **hair cells** (receptors for sound)
  - Air pressure force of sound transformed to nerve signals carrying sound information via CN VIII (auditory nerve) → auditory complex in **temporal lobe of brain**
- Balance/Equilibrium:
  - The **vestibule**
  - Multiple structures that recognize orientation of body in 3D space and send this information via CN VIII (auditory nerve) → **brainstem**

More on the ossicles (ear bones):

- **malleus, incus, stapes**
- Stapes presses on **oval window** (membrane of inner ear) which causes fluid of inner ear to move
- The malleus bone is seen on **otoscopy** since part of the malleus bone called the **umbo** presses on the tympanic membrane (eardrum **landmark** on physical exam)

Great site on otoscopy: University of Bristol:
https://www.entbristol.co.uk/otoscopy/

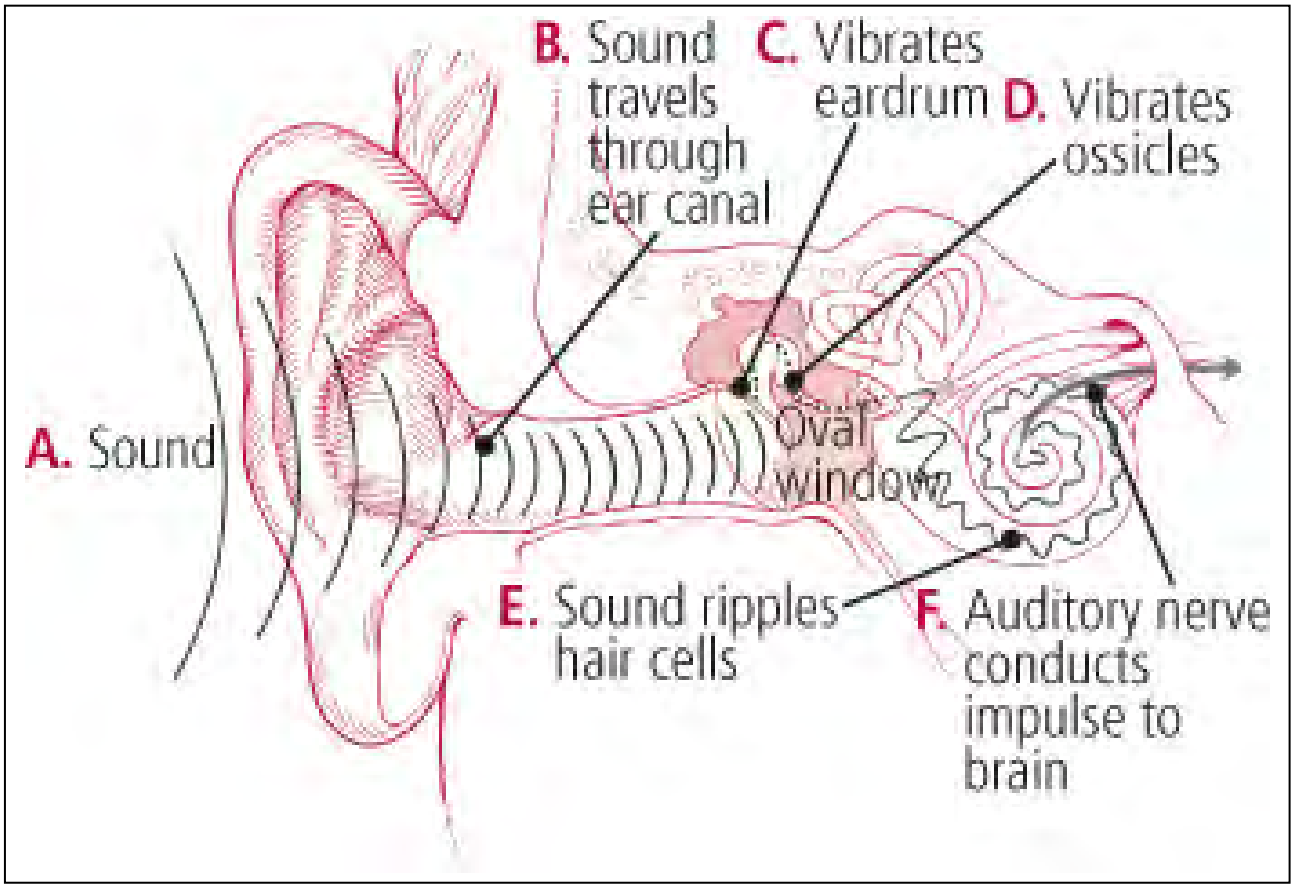




Eustachian tube:

- connects the middle ear to the throat
- opens with yawn or swallow to equalize air pressure to allow tympanic membrane vibration
- **Clinical:**
  - Can be blocked and/or inflamed with pharyngitis, tonsillitis
  - The epithelium is continuous with throat

Sound transmission: conduction of sound through **AIR** and **BONE**



Physics of sound – combination of pitch & loudness:

- Pitch (frequency):
  - refers to the frequency of sound waves
  - higher or lower pitch (soprano voice is higher, bass voice is lower)
  - usually we hear HIGHER pitch sounds better
- Loudness (amplitude):
  - more hair cells stimulated & more rapidly
  - usually we hear LOUDER sounds better
  - louder or softer – measured in **decibels**
  - **Decibel:**
    - a 10-fold increase in sound amplitude is "1 bel", and 0.1 bel = 1 decibel
    - our ears distinguish between 10 decibel divisions of sound

(Pictures from: http://www.audiology.org/)

Pitch (sound wave frequency):

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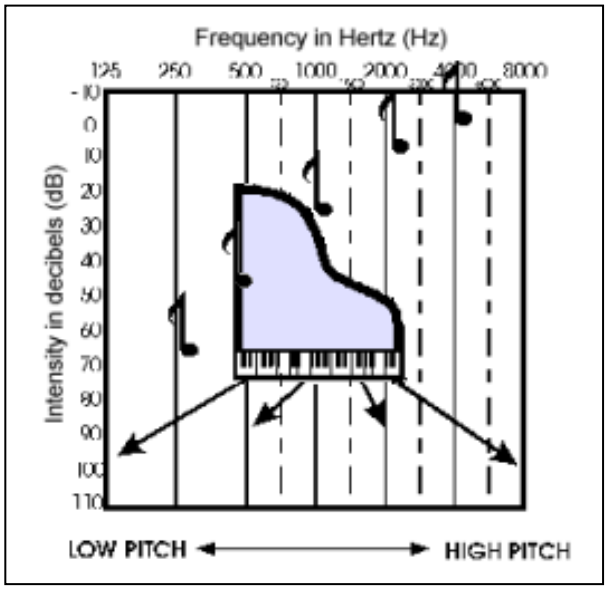
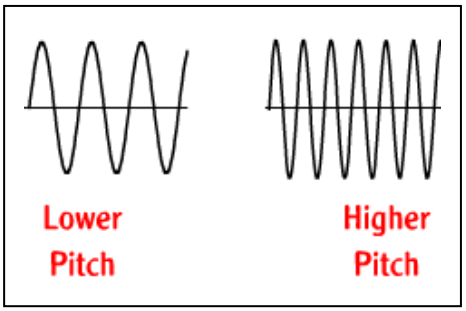
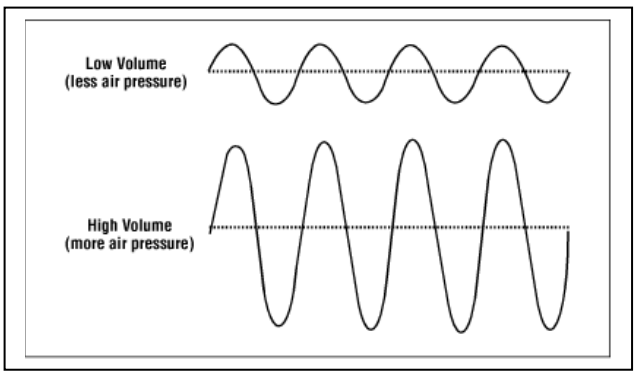
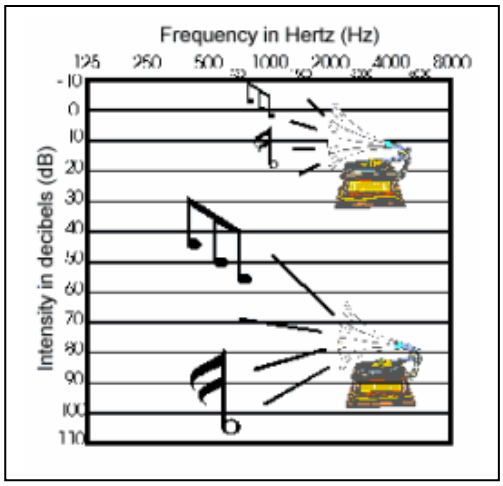




Loudness (sound wave amplitude):

- more hair cells stimulated & more rapidly at higher decibels





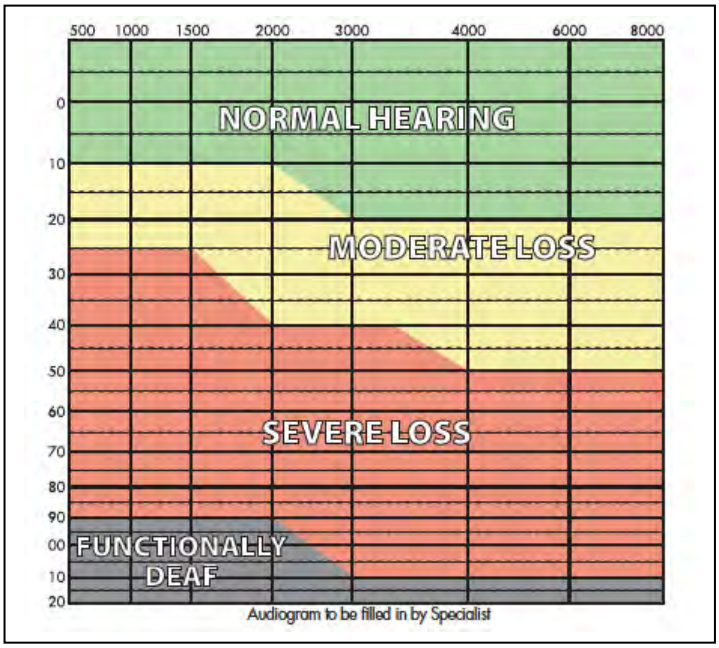
Adaptation of hearing:

- Retrograde (backwards) pathways from auditory cortex to cochlea provide for inhibition of sound transmission to the brain
- Permits for selective hearing:
  - Pick out one sound out of many and reduce sound intensity
  - Example: focus on hearing one instrument out of the whole orchestra

Directionality of hearing: Able to also distinguish direction of sound

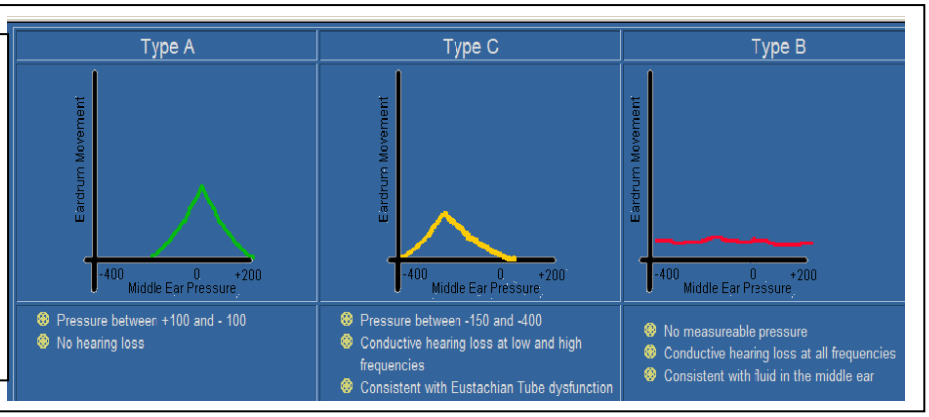
Audiogram – clinical measurement of hearing:

- "pure tone" audiometry
- determines ability to hear pitch as well as loudness
- helps distinguish ability to understand speech



Tympanometry:

- Measure the ability of the tympanic membrane to move with air pressure
- Can diagnose conditions affecting the middle ear (such as otitis media infection)
- Can help distinguish different types of **hearing loss (deafness)**



Sensory loss – DEAFNESS:

- Three **organic** types – nerve deafness (sensorineural deafness), conductive deafness, mixed
- There is also "functional" deafness – unable to hear due to psychiatric problem
- Nerve deafness (sensorineural):
  - damage to cochlea (organ of Corti), auditory nerve, or CNS afferebt neural circuits
  - Therefore, BOTH air AND bone conduction are diminished
  - Damage of specific parts of the cochlea leads to specific frequency loss deafness
- Conductive deafness:
  - Outer or middle ear dysfunction affecting air conduction only
  - Bone conduction is left intact
  - Patient tends to speak softly since their own voice is amplified (bone conduction)
  - Can be as simple as **impacted cerumen** (ear wax) or damage to middle ear (otosclerosis – scarring from repeated infections or hereditary type)
- Mixed deafness: components of both types

Hearing loss (deafness) & speech?

- Any hearing loss for any period of time will **interfere with speech acquisition in the infant/child**
- EARLY diagnosis permits evaluation/treatment and implementation of speech-language services

Newborn hearing screening – Early Hearing Detection & Intervention (EHDI):

- Cannot "ask" the infant if they can hear, must rely on technology
  - **otoacoustic emissions (OAE) testing** (mandated by most states)
  - **auditory brainstem response (ABR)(also called brainstem evoked response, BAER)**
- See American Speech-Language & Hearing Association (ASHA): http://www.asha.org/advocacy/federal/ehdi/ (lots of information on types of tests, state laws)
- This website also has good information on preventing hearing loss

Presbycusis: **"presby-" = aging**

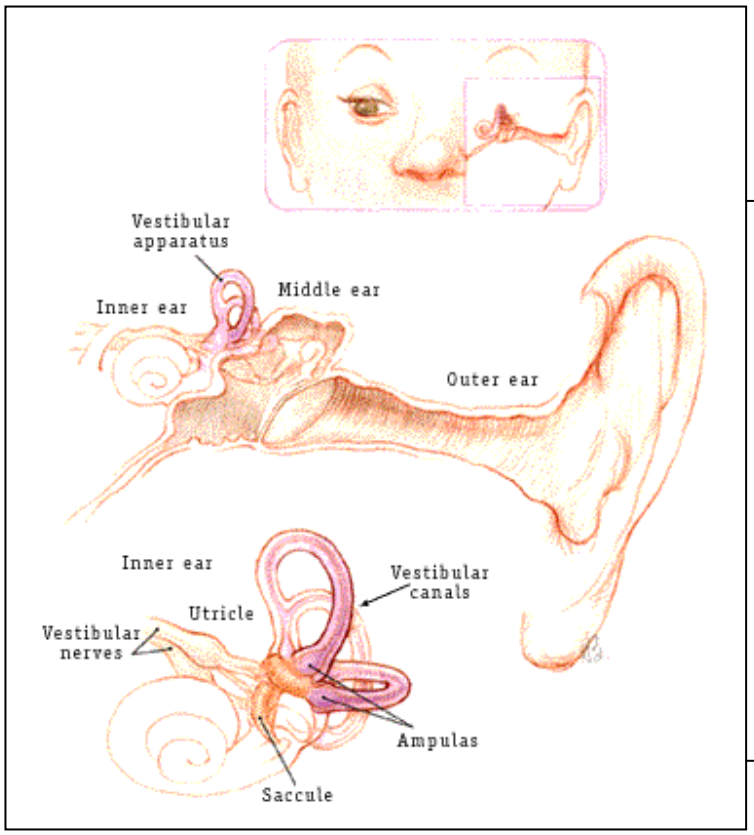
- **high frequency sound deafness** – particularly difficult for speech discrimination
- also affects hearing consonants such as s, sh, and f

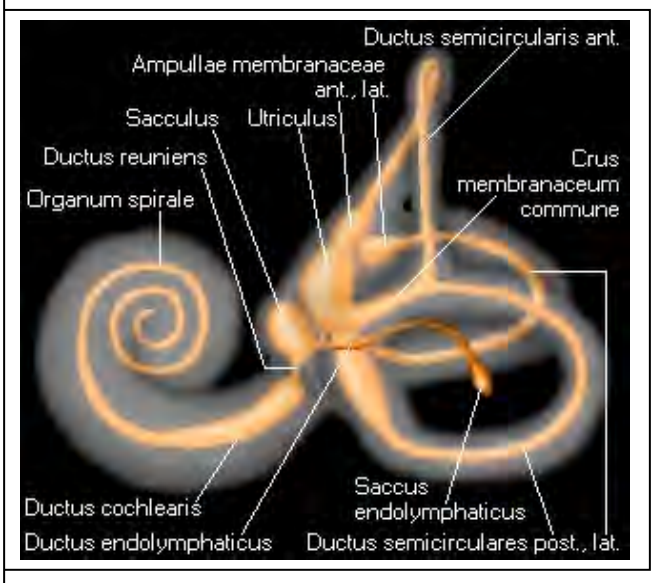
Sound deafness:

- **low frequency pitch loss due to exposure to loud sounds**
- prevent hearing loss by AVOIDING such exposure: http://www.asha.org/public/hearing/Noise-and-Hearing-Loss-Prevention/

Balance & equilibrium:

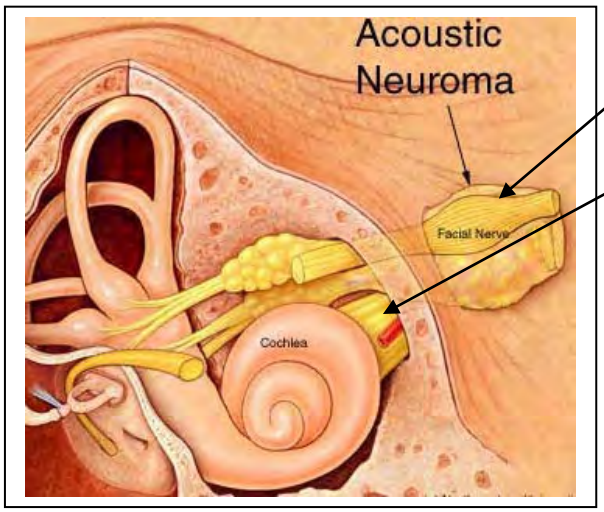
- Maintaining balance – standing upright (don't fall down – many receptors, including visual cues)
- Equilibrium – sense of where we are in 3D space
- **Video**: https://www.youtube.com/watch?v=dSHnGO9qGsE
- Inner ear balance structures:
  - The vestibular complex – receptors for equilibrium containing the **osseous labyrinths** (bony mazes and chambers filled with perilymph fluid and receptors) and the **membranous labyrinths** (follows the shapes of the bones and filled with endolymph fluid and receptors)
  - **Vestibule:**
    - Membranous sacs (saccule & utricle) with hair cells that lie under a layer of crystals made of calcium carbonate called **otoconia (ear rocks, otoliths)** – crystal movement with head tilting stimulate hair cells and give information about head in space in relation to **gravity** (are you up or down?)
  - **Semicircular canals:**
    - Project from vestibule and detect head motion forward/backward, up/down, left/right
    - Stereocilia move in the endolymph to detect head rotation information





Conditions affecting both hearing AND balance:

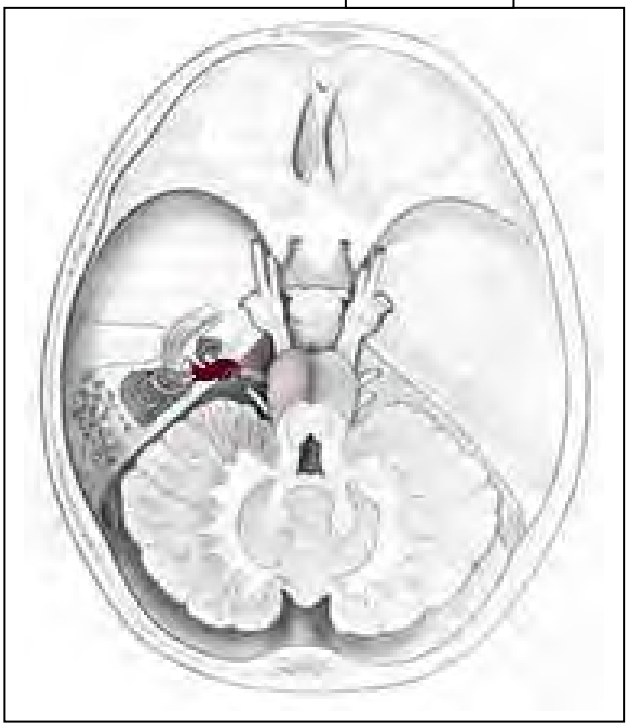
- Meniere's Disease:
  - TRIAD of progressive hearing loss, tinnitus, and vertigo
  - Final result may be permanent hearing loss & severe functional disability
  - Very detailed overview: http://www.entnet.org/content/menieres-disease
- Acoustic neuroma (also called neurinoma, vestibular schwannoma):
  - Tumor of CN VIII (vestibulo-coclear nerve, auditory nerve)
  - This CN is actually two nerves that run alongside each other (vestibular for balance, cochlea for hearing) and can be referred to as simply the "auditory" or "acoustic" nerve
  - Runs from the inner ear through the inner auditory canal (IAC)
  - It is benign but may grow rapidly if caused by neurofibromatosis (NF2)
  - Symptoms of hearing loss, tinnitus, vertigo, disequilibrium, sense of fullness/pressure in ears, facial numbness/paralysis (if pressing on CN VII Facial Nerve)
  - Evaluation: MRI of IAC, BAER (brainstem evoked response)
  - Treatment: surgery, stereotactic radioablative therapy



Acoustic neuroma pressing on CN VII (facial nerve)

CN VIII (acoustic nerve)(origin of tumor)

Tumor in IAC



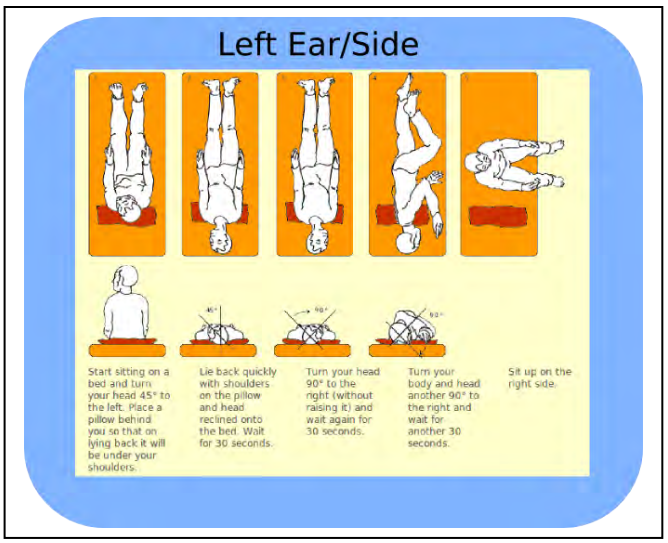
Dizziness:

- Labyrinthitis:
  - Inflammation causing inner ear dysfunction
  - Symptoms of vertigo and nausea/vomiting
- BPPV:
  - 20% of dizziness is due to benign paroxysmal positional vertigo (BPPV)
  - Usually the middle aged to older person (50% of dizziness in older persons due to BPPV)
  - Symptoms of dizziness (vertigo), lightheadedness, imbalance, nausea
  - Usually precipitated by positional head change related to gravity
  - Can also occur with head injury, whiplash, migraine
  - **Pathophysiology:** debris collection in inner ear
  - **Treatment:** Epley maneuver reposition otoconia
- Other causes:
  - Drug toxicity, viral illness (vestibular neuritis), Meniere's disease, idiopathic

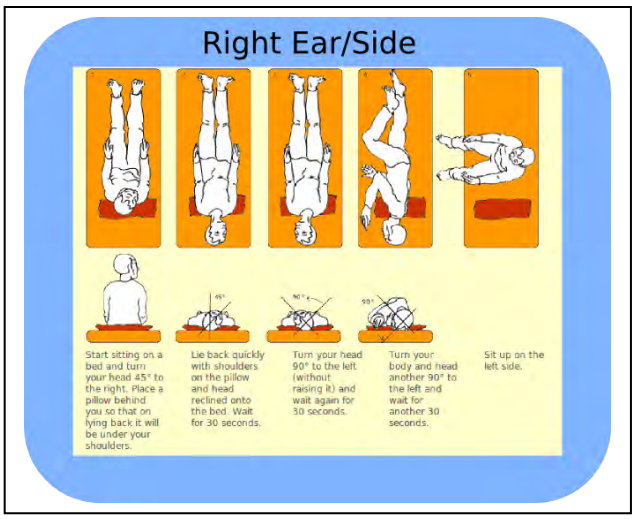
Epley maneuver for BPPV:

- (many) YouTube videos – a good one: http://www.youtube.com/watch?v=7ZgUx9G0uEs
- Maneuvers thought to reposition the otoconia
- Can be done by clinician or by patient (self treatment) – keep eyes open during treatment (http://www.tampabayhearing.com/epley.php)
- More on Epley maneuver for benign paroxysmal positional vertigo (BPPV):
  - http://www.hopkinsmedicine.org/neurology_neurosurgery/specialty_areas/vestibular/conditions/benign_paroxysmal_positional_vertigo.html
  - http://vestibular.org/understanding-vestibular-disorders/treatment/canalith-repositioning-procedure-bppv
  - http://www.activator.com/wp-content/uploads/Home%20Epley%20Handouts.pdf
  - http://www.dailymotion.com/video/xjzdqj_epley-maneuver-how-to-perform_school

Left side vertigo treatment



Right side vertigo treatment



EYE (VISION)

VISION SIMULATORS let you see what the patient sees!

Some Vision Simulators Online: shows how your vision changes with common eye conditions

1) VARIOUS: http://www.ohiolionseyeresearch.com/research/simulations/
2) LOW VISION: http://webaim.org/articles/visual/lowvision
3) COLORBLIND: http://www.webexhibits.org/causesofcolor/2.html
4) BABY VISION: http://tinyeyes.com/
5) VARIOUS: http://www.richmondeye.com/simulations-of-eye-disorders/ AND http://www.cnib.ca/en/your-eyes/eye-conditions/eye-connect/Pages/EyeSimulator.aspx

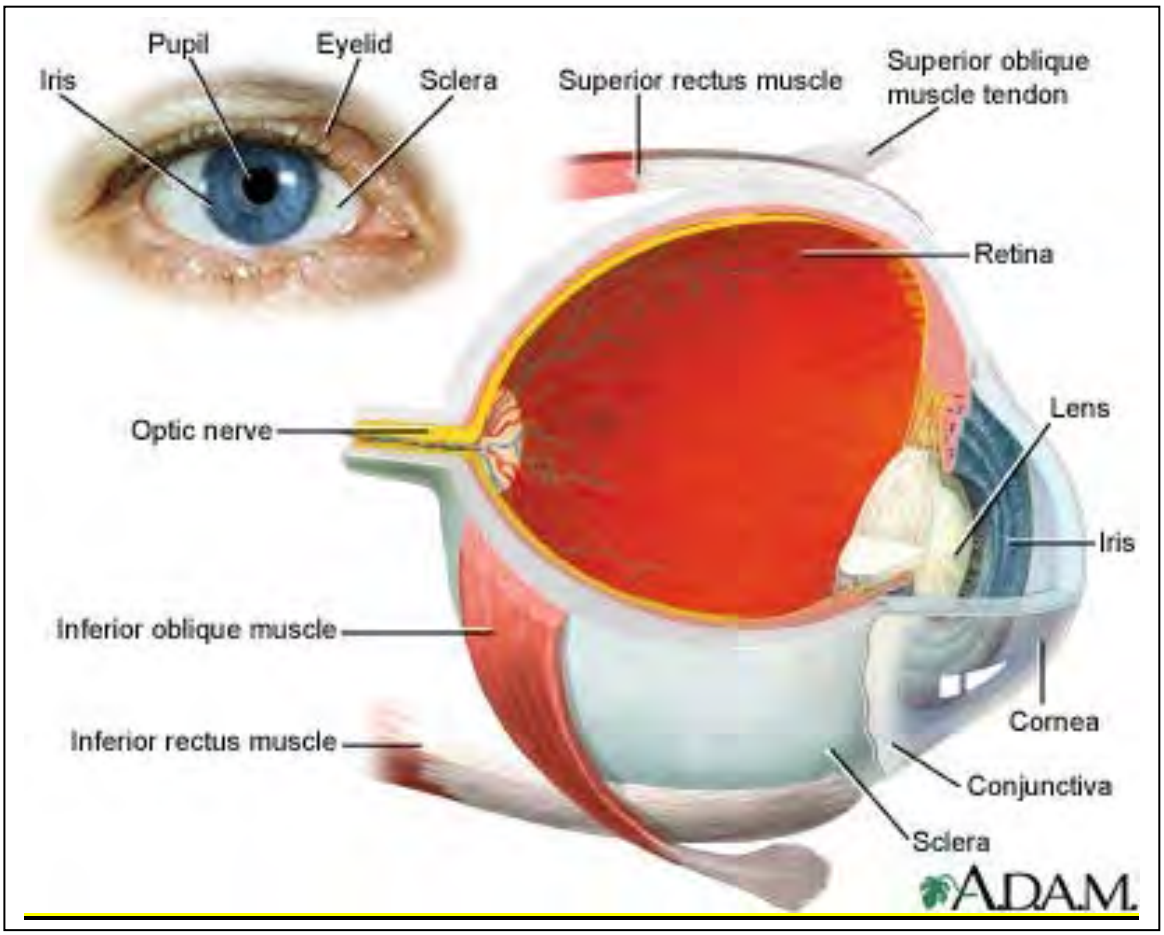
Clinical Eye Exams:

1. **Great pix:** The "Eyes Have It" at http://www.kellogg.umich.edu/theeyeshaveit/index.html
2. EOM, PERRLA, Accommodation, etc: http://www.neuroexam.com (check Cranial Nerves for vision & extra-ocular muscle movement)
3. INTERACTIVE EOM: http://anatomyresources.hsc.wvu.edu/ReillyWeb/MEP1144/Unit6/images/ExtraocularSkeletalMuscle.swf (THIS IS A **MUST** VISIT!!! WHAT NERVES CONTROL WHICH MOVEMENTS???)

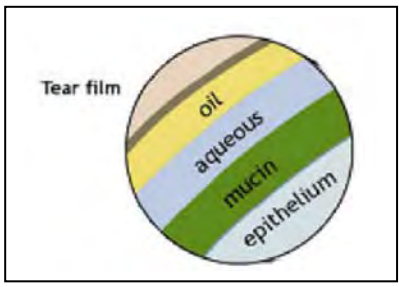
Eye Procedures:

1) Go to: https://coastaleyegroup.com/ and click on "Procedures" tab
2) LASIK & more: http://www.allaboutvision.com/

Eye structures



EXTERNAL EYE STRUCTURES:

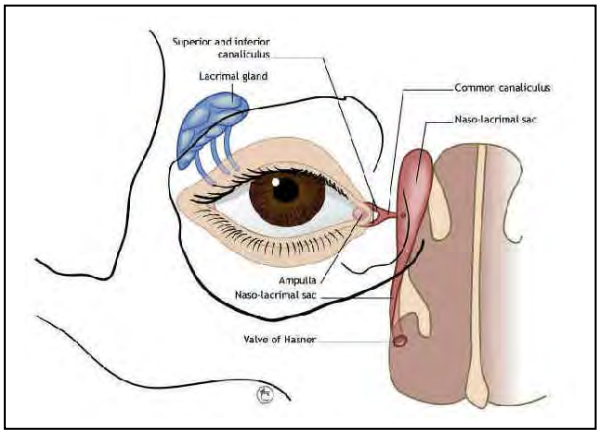


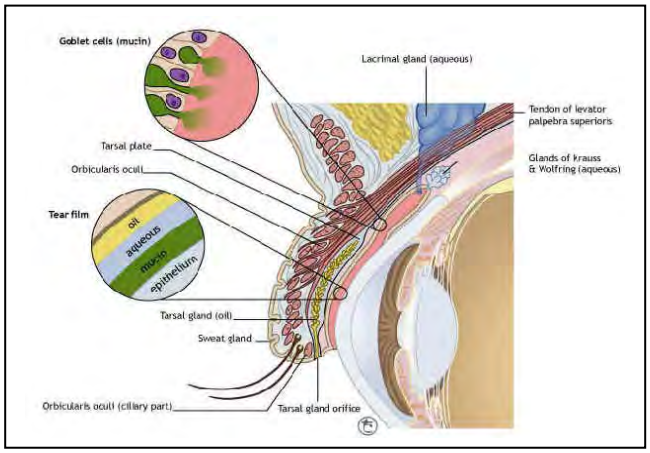
Structures:

- eyelids (palpebrae)
- conjunctiva
- lacrimal apparatus

Review the lacrimal system: Lacrimal gland, Tear ducts

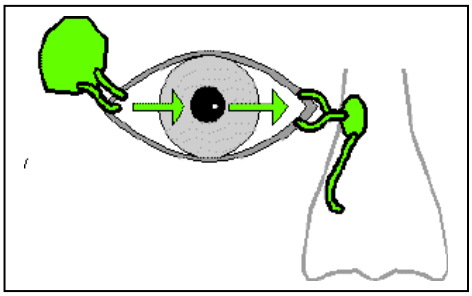
- Formation of tears ("tear film")
- Aqueous (from lacrimal gland), mucin (from goblet cells), oil (from tarsal gland)

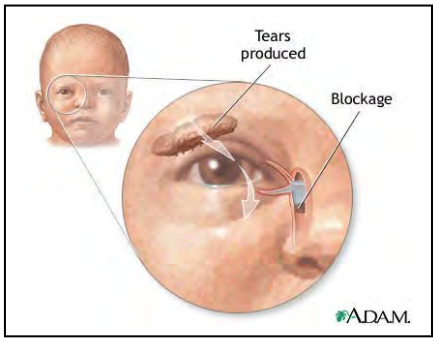




Lacrimal gland:

- lacrimal gland lies at upper lateral area above eyelid, tears go into the eye from the upper outer canthus
- Then tears drain out of the eye at the inner canthus via drainage holes (lacrimal punctum, upper & lower) and lacrimal canaliculi (upper & lower)
- The canaliculi drain into the lacrimal sac and then out of the nasolacrimal duct
- This can be blocked, especially in childhood





Subconjunctival hemorrhage:

- minor trauma, strain, sneeze, cough (& bleeding dyscrasias)
- Blood extravasates beneath conjunctiva, usually reabsorbs within two weeks without treatment

Conjunctivitis: (inflammation of conjunctiva)

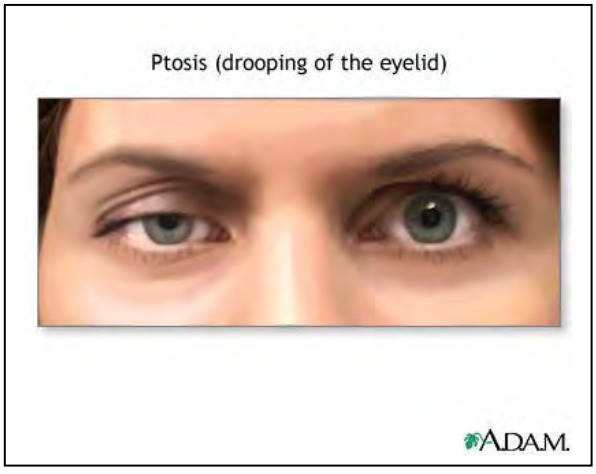
- May be bacterial, viral, allergic, chronic
- Trachoma
  - *Chlamydia trachomatis* -- leading cause of preventable blindness worldwide
  - cause of neonatal conjunctivitis from birth canal *Chlamydia*
  - seen in warm weather from swimming pool infections
  - neonatal eyedrops – treat any possible *Chlamydia* or gonococcal infections
- Kawasaki Syndrome:
  - children
  - red eyes, strawberry tongue, cracked/red lips, fever & abdominal pain, rash
  - admission to hospital to prevent **complication** of cardiac artery vasculitis and aneurysm
  - See: http://kidshealth.org/en/parents/kawasaki.html

Pink eye = conjunctivitis

Disorders of palpebrae (eyelids):

- Blepharitis:
  - bacterial, seborrheic
- Hordeolum (stye):
  - external sebaceous glands or internal meibomian oil-secreting glands
- Chalazion:
  - chronic enlargement from occlusion of meibomian oil-secreting gland
  - may need systemic antibiotics & surgical lancing
- Entropion (inversion) and extropion (eversion):
  - From aging or scarring.
  - Scarring can occur due to surgery for basal cell carcinoma (BCC)
  - Usually require blepharoplasty (plastic surgery on eyelid)
- Ptosis – drooping of lid:
  - Disorder of sympathetic nervous system (e.g. Horner's syndrome)
  - Myasthenia gravis

Ptosis



Entropion

Blepharitis

CORNEA & LENS:

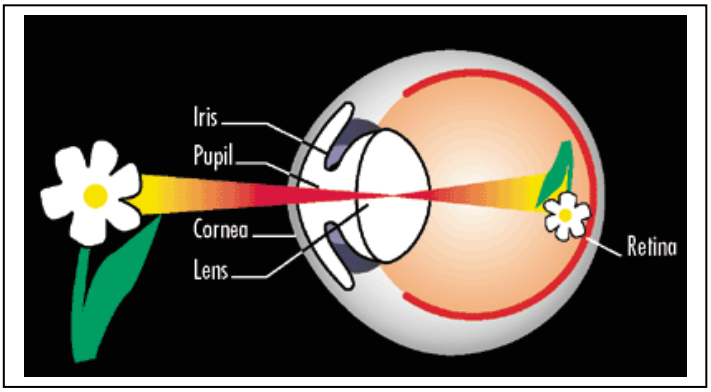
Selected corneal pathologies:

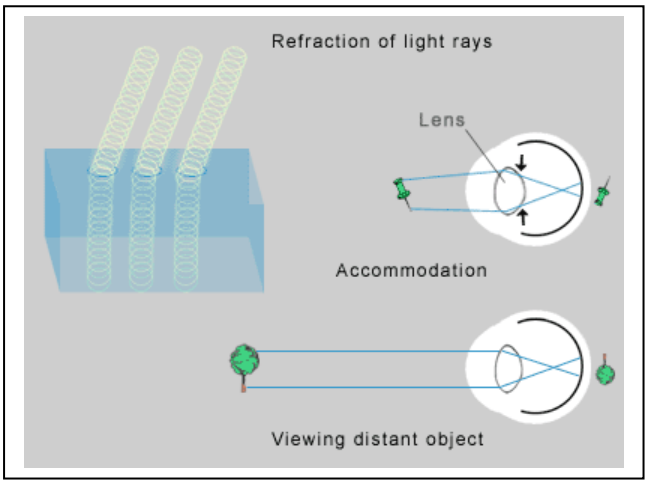
- **ulceration** (infection)
- **scarring** (herpes simplex keratitis)
- **astigmatism**

The focal point image and refraction:

- Focal point: the place that a lens will create the sharpest image after convergence of the light rays (where all the light rays come together in one spot)
  - Due to the bending of light through our eye structures, the focal image will be **upside down and reversed** and should land on the **retina**
- Lens of the eye: changes shape to focus the image to the best of its ability
  - The lens capsule is elastic, and ligaments attach to pull it into shape, done by the **ciliary muscle**
- Refraction:
  - If light passes through an *angled* interface then an *angled* light beams will exit the other side
  - **Convex lens (bulges outward at center):**
    - focuses light rays (**convergence**) towards a focal point.
    - The thicker the lens is in the middle, the greater the refractive power, and the closer to the lens will appear the focal point
    - This is measured in **diopters**
  - **Concave lens (scooped inward at center):**
    - causes rays to fan out (**divergence**)
  - Corrective lenses (eyeglasses) correct any defects by bending light rays
- Visual Acuity: ability to discriminate two close points
  - **fovea centralis of retina** responsible for this
- Determination of distance:
  - use of **stereopsis** (binocular vision – two eyes)
  - and **moving parallax** (movement of head compares close vs. distant objects)

Focal point landing on retina, image upside down & reversed





Nervous system control of lens focusing:

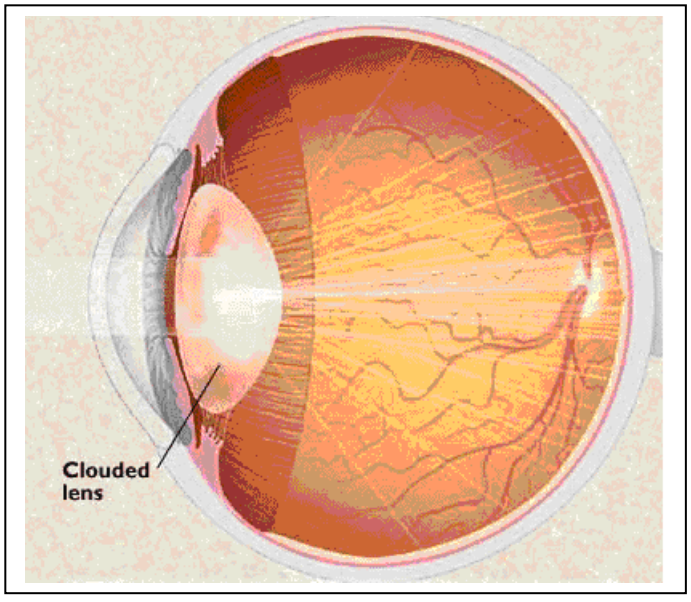
- Near Vision:
  - **Parasympathetic system**
  - contraction of the ciliary muscle, bulging the lens into a more convex shape, allows us to see near objects
  - in addition, the eyes converge (move towards the nose) and the pupils constrict
- Distance vision:
  - **Sympathetic system**
  - Relaxation of ciliary muscle, elongating the lens to see further objects (emmetropia)
  - In addition, eyes are aligned straight ahead and pupils dilate to allow more light in

Presbyopia:

- aging causes lens to be larger and thicker, less elastic, can't focus on near OR far objects **("non-accommodating")** and remains focused permanently at almost constant distance
- this is "short-arm syndrome" – arm is too short for a person to focus on reading materials (time for bifocals)

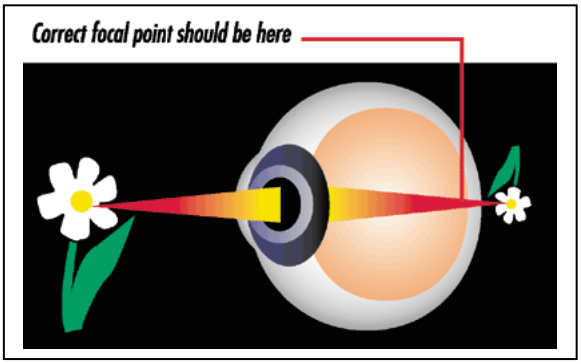
Cataracts of the lens:

- cloudy vision, "looking through a waterfall (cataract)"
- cloudy or opaque area or areas in lens
- **Causes:**
  - Xrays, infrared heat (damaged lens proteins)
  - DM: especially after acute elevations of plasma glucose → glycosylation of lens proteins with every hyperglycemic spike
  - uveitis
  - corticosteroids & other drugs

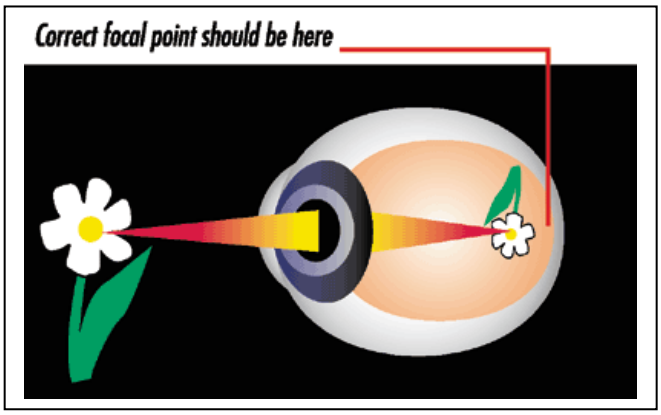


Refractive Errors:

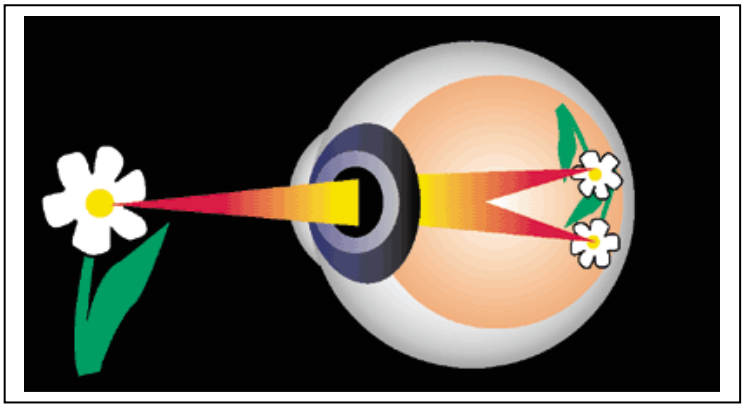
- **Hyperopia (farsightedness):**
  - eyeball too short or lens too weak (image focal point behind retina)
  - can adapt by accommodation (lens contraction), but lose even for far objects when aged due to lens inelasticity



- **Myopia (nearsightedness):**
  - eyeball too long or excessive refractive power of lens (image focal point in front of retina)
  - no way to accommodate (can't relax lens beyond normal relaxed state)



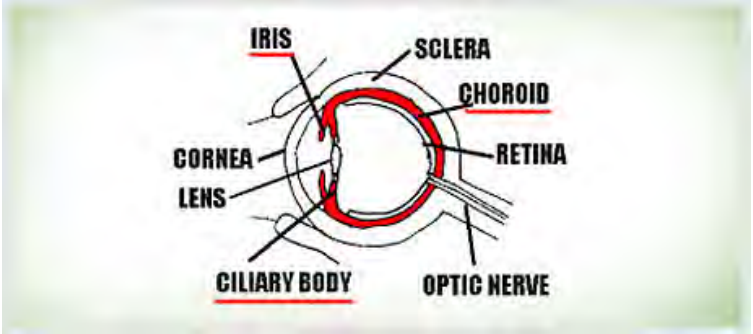
- **Astigmatism:**
  - abnormal curvature of the cornea causes non-parallel focus of image
  - Additional spherical lenses are used to correct for the incorrect light ray path



Thanks to the John A. Moran Eye Center of the University of Utah for the “daisy” pictures

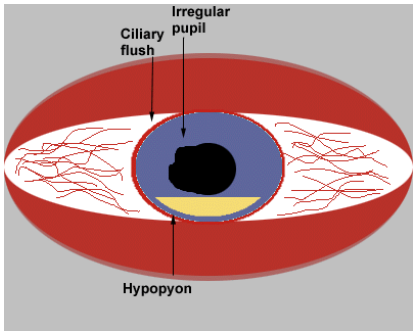
UVEAL TRACT: pigmented tissues of eyes

Uveal tract:
The IRIS, CILIARY BODY, CHOROID



Uveitis: a serious illness.

- **Causes:** retinitis, such as RA, different CT diseases, CMV, histoplasmosis, TB, syphilis, sympathetic ophthalmoplegia, sarcoid, lymphoma
- **Photophobia** with **visual impairment** and a **red eye**
- On exam **limbic flush**
  - perilimbal flush -- injection adjacent to limbus where iris meets sclera
  - pupillary miosis
- Needs URGENT ophthalmic care!

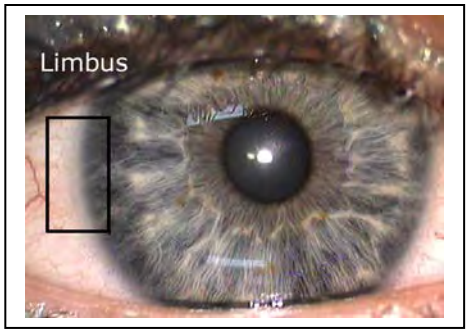


Irregular pupil due to adhesions between lens & cornea.

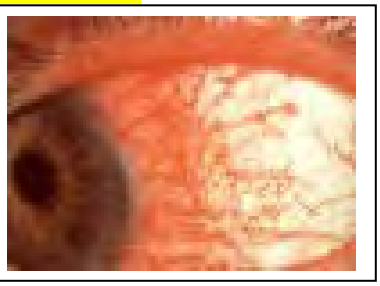
Ciliary **(limbic) flush** is seen.

Hypopyon may be seen.

Normal limbus



Limbic flush from uveitis



Evaluation of the RED EYE:

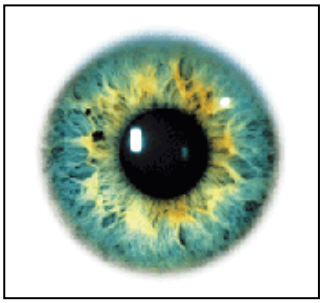
- Differential diagnosis
  - IS IT CONJUNCTIVITIS OR IRITIS/SCLERITIS?
  - Conjunctivitis is usually bacterial or viral, rarely photophobia
  - **Iritis/scleritis** associated with **UVEITIS** (inflammation of pigmented parts of the eye)
    - Usually auto-immune, serious, can result in loss of vision
    - Often has **photophobia**
    - **LIMBIC FLUSH** means the "redness" of the eye includes the limbus

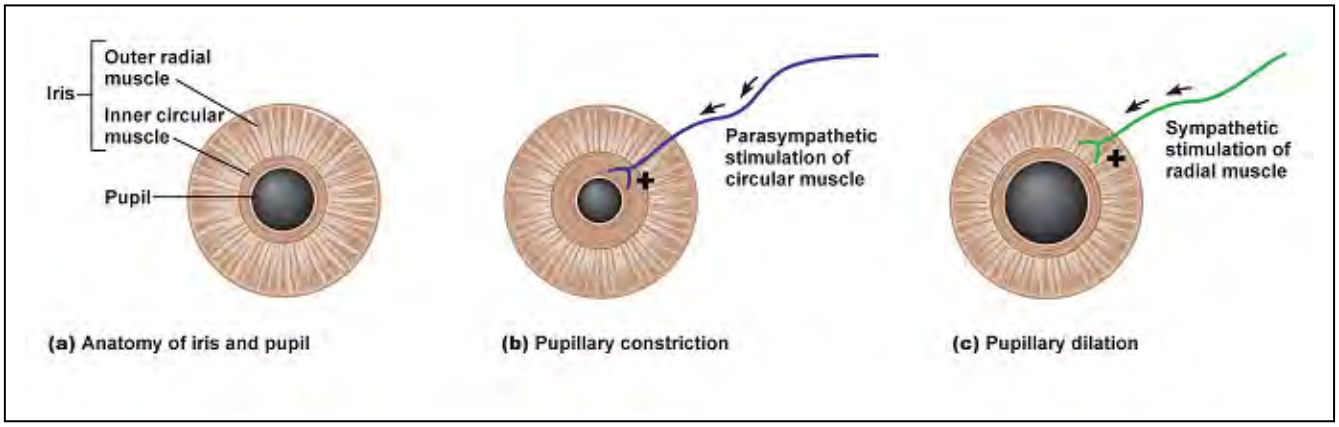
Clinical feature	Suggests
Severe eye aching	Iritis, keratitis, acute angle-closure glaucoma, scleritis, orbital cellulitis, cavernous sinus thrombosis (CST)
Prominent photophobia	Iritis, keratitis
Impaired vision	Iritis, keratitis, acute angle-closure glaucoma, orbital cellulitis, CST
Cloudy cornea	Keratitis, acute angle-closure glaucoma
Corneal opacification	Keratitis - chemical or infectious
Circumcorneal conjunctival injection	Iritis, keratitis
Cloudy anterior chamber	Iritis
Pain on eyeball palpation	Scleritis (+++), orbital cellulitis, CST
Proptosis	Orbital cellulitis, CST, posterior scleritis
Impaired, or painful, extraocular eye movements	Orbital cellulitis
Fever, toxic appearance	Orbital cellulitis (+), CST (++)
Hyperpurulent discharge from an "angry" eye	Gonococcal conjunctivitis/endophthalmitis
Prominent nausea and vomiting	Acute angle-closure glaucoma
Small, irregular, poorly-reactive pupil	Iritis
Fixed mid-dilated pupil	Acute angle-closure glaucoma
Increased intra-ocular pressure	Acute angle-closure glaucoma, iritis (secondary complication)
History of connective tissue disease, or granulomatous disease	Iritis, scleritis

THE PUPILS AND AUTONOMIC REFLEXES:

Pupillary diameter:

- If the pupil is very small, all light rays come in towards center of lens and don't lose focus (better "depth of focus")
- A smaller pupil may correct for poor vision
- Pupillary diameter changes in response to:
  - amount of light entering (mydriasis = larger; miosis = smaller)
  - accommodation to near or far vision – also involves the pupillary size
  - varies from 1.5 mm to 8.0 mm

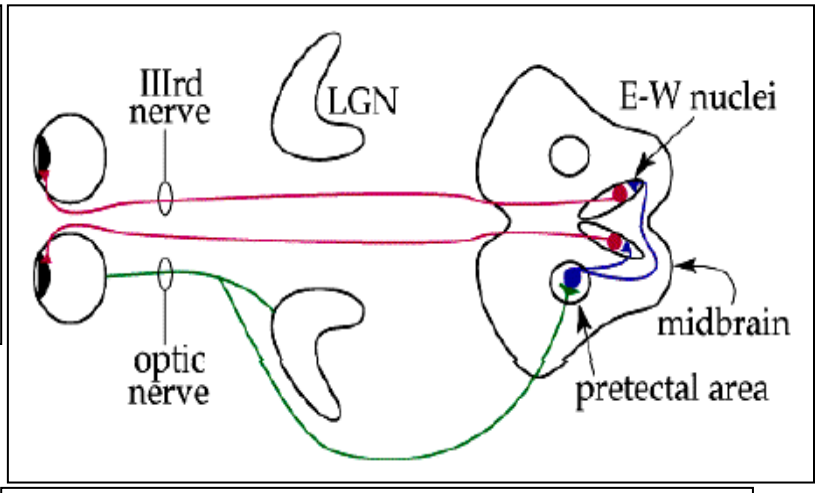


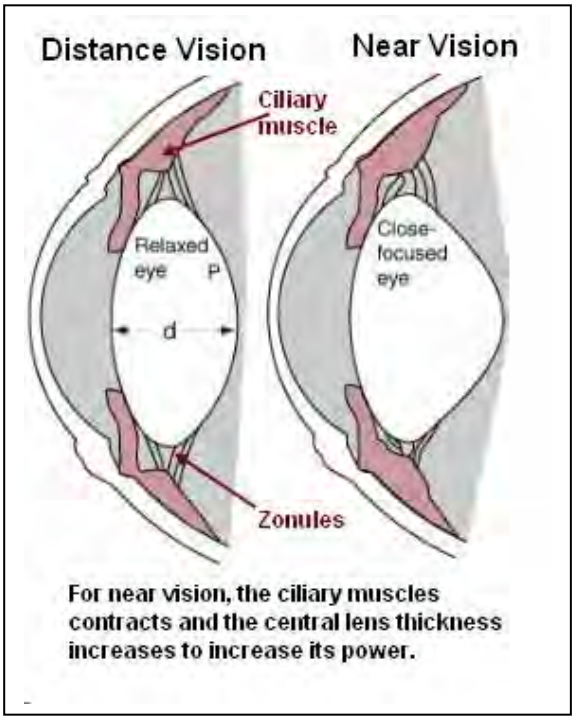


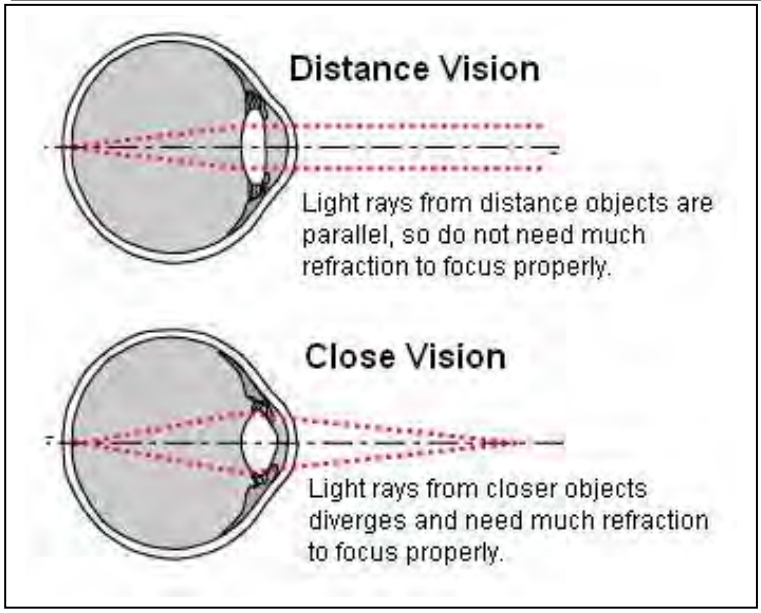
ANS control of focus:

- Near Vision: **Parasympathetic system**
  - **Miosis** occurs to reduce pupillary aperture (reflex inhibited in the dark – can't read in the dark)
    - Contraction of **circular muscle** (sphincter muscle)
  - **Pathway:** Edinger-Westphal nucleus (CN III visceral nucleus) → ciliary ganglion behind the eye → postganglionic parasympathetic neurons → ciliary nerves (lens focus, iris sphincter for constriction of pupil) & extra-ocular muscles (accommodation)
  - **Lens** bulges into a more convex shape due to contraction of the ciliary muscle
  - **EOM** for eyeballs to **converge** (move towards the nose)
- Distance vision: **Sympathetic system**
  - **Mydriasis** occurs to widen pupillary aperture
    - Radial fibers of the iris contract
    - Relaxation of circular (sphincter) muscle
  - **Lens** elongates due to relaxation of ciliary muscle
  - In addition, eyes are aligned straight ahead
  - **Pathway:** T-spine sympathetic outflow chain → superior cervical ganglion → postganglionic sympathetic neurons → along carotid artery and smaller brain arteries → to finally innervate radial fibers of iris, some extraocular muscles, weak ciliary muscle innervation, innervation of upper eyelid muscle to keep it elevated while awake

Light enters the retina; afferents via optic nerve to the pretectal area. After synapsing, interneurons send afferents to the **Edinger-Westphal nuclei** on both sides of the midbrain. Efferents from the EW nucleus travel via **CN III** to cause pupillary constriction to light.







The ocular exam:

- **Gross inspection:**
  - pupillary response to light and accommodation
  - extra-ocular muscle movements (EOM)
  - conjunctiva, sclera, pupil
  - position on head, distance between eyes,
  - eyelids, lashes
- **Ophthalmoscope:**
  - light cast onto patient's retina is reflected back to observer.
  - the focus (lens) wheel corrects for refractive error of the observer.
- **Slit lamp:**
  - precise evaluation from cornea in, including lens, uveal tract (pigmented tissues)
- **Pupillary response:**
  - Pupils equal size (if not, **anisocoria**, which may be normal variant or evidence of nerve palsy)
  - With light source, approach eye from temple and look for pupillary reaction to light (contraction, miosis) and then as the light is moved away, look for pupil opening again (mydriasis)
  - This is the **direct** reaction (shining onto that pupil & seeing constriction)
  - Now shine the light on the OTHER eye – both pupils constrict – this is the **consensual** reaction (the OTHER pupil constricts to light)
- **Accommodation:**
  - eyes follow your finger towards their nose **(convergence)** and also the **pupils constrict**
- **"PERRLA"** – pupils equal, round, reactive to light & accommodation
- *HEENT exam **videos:***
  - From OPETA: http://www.veoh.com/list/c/clinicalexamination
  - From UVA: http://www.med-ed.virginia.edu/courses/pom1/pexams/HEENT/
- *HEENT & EYE exam **videos**:*
  - from OPETA: http://www.veoh.com/list/c/clinicalexamination
  - from Loyola: http://www.lumen.luc.edu/lumen/MedEd/MEDICINE/PULMONAR/PD/eye.htm
  - from Med-Ed VA: http://www.med-ed.virginia.edu/courses/pom1/pexams/HEENT/
- *Direct ophthalmoscopy:*
  - Direct ophthalmoscopy: http://careers.bmj.com/careers/advice/view-article.html?id=354
  - Approach to funduscopic exam video: https://stanfordmedicine25.stanford.edu/the25/fundoscopic.html
  - Welch-Allyn resource: https://www.welchallyn.com/content/dam/welchallyn/documents/sap-documents/LIT/80012/80012038LITPDF.pdf
- *HEENT Physical exam:*
  - http://meded.ucsd.edu/clinicalmed/head.htm (head/neck)
  - http://meded.ucsd.edu/clinicalmed/eyes.htm (eye)

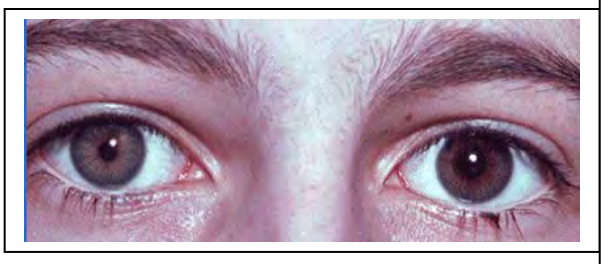
Abnormal pupils and pathology:

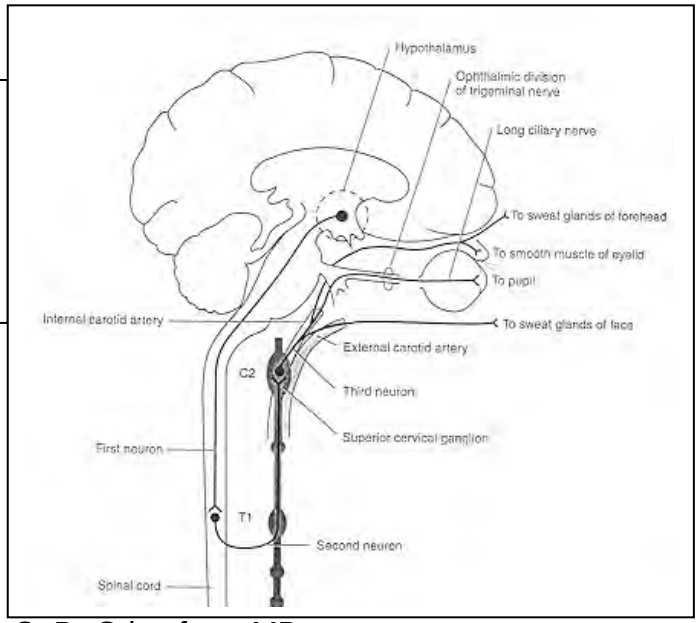
Argyll-Robertson (AR) Pupil:

- chronically partially constricted pupil and failure to respond to light
- indicates CNS disease
  - syphilis, alcoholism, encephalitis from any cause (e.g., HIV)
- seen in both eyes (**bilateral**)
- Destruction of afferent loop to the E-W nucleus results in loss of normal input
- **Exam:**
  - Continued stimulation of the E-W nucleus through convergence (accommodation) will constrict the pupils further →
  - light reflex is gone but accommodation reflex remain
  - pupil is small
- Indicates CNS disease -- syphilis, alcoholism, encephalitis from any cause (e.g. HIV).
- Seen **bilaterally**

Horner's Syndrome:

- due to damage to the cervical sympathetic chain:
  - remember the sympathetic ouflow is thoracic-lumber from the spinal cord
  - in order for the face to receive innervation, the thoracic outflow must send nerve fibers up from the chest into the neck and then to the face
  - any compression or damage along the way will prevent sympathetic innervation on that side of the face
  - usually due to a tumor (e.g., lymphoma) or other mass in the neck
- One-sided (**unilateral**) and **ipsilateral** (same side as damage/pathology):
  - **facial flushing:** blood vessels on that side of face & head become persistently dilated (sympathetic arteriolar constriction is not present)
  - **partial ptosis of upper lid:** while awake, the upper lid is kept open by sympathetic stimulation – this is now gone
  - **miosis (constriction of pupil):** lack of pupillary dilation from radial fibers
  - **anhydrosis (absence of thermoregulatory sweating):** on forehead & face — sweating cannot occur on that side of the face & head due to lack of sympathetic stimulation of sweat glands



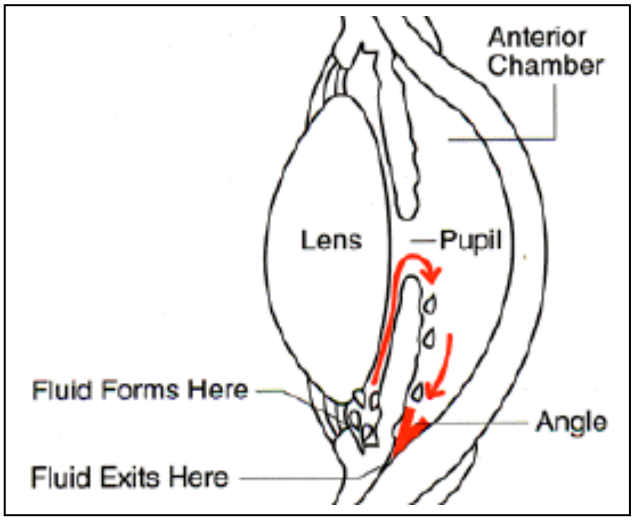


FLUID SYSTEMS OF THE EYES:

- Fluid aqueous humor in front of lens
- Gelatinous vitreous humor behind lens

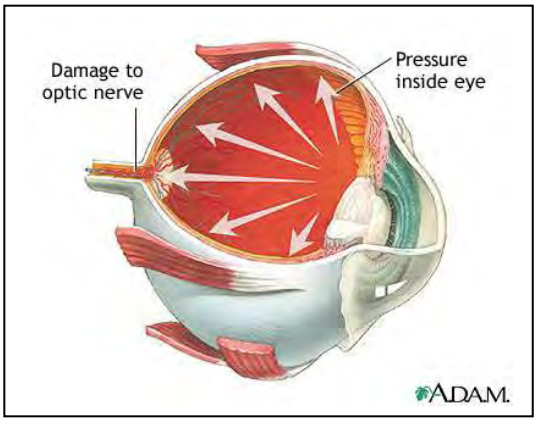
Aqueous Humor:

- Intraocular pressure (IOP): this fluid regulates total volume & pressure of the eye
- Produced by ciliary body behind the iris
- Flows from behind iris, through pupil, into anterior chamber
- Leaves the eye via angle between iris & cornea (anterior chamber), through meshwork of cell (trabeculae) into canal of Sclemm and then into venous system

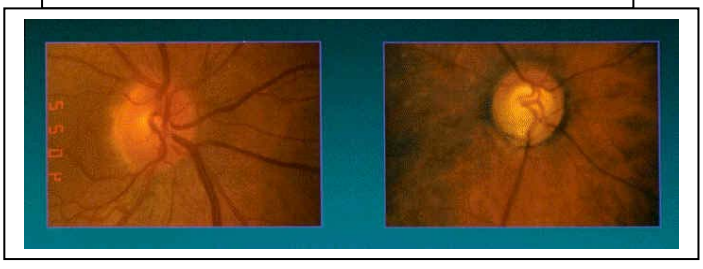


Glaucoma:

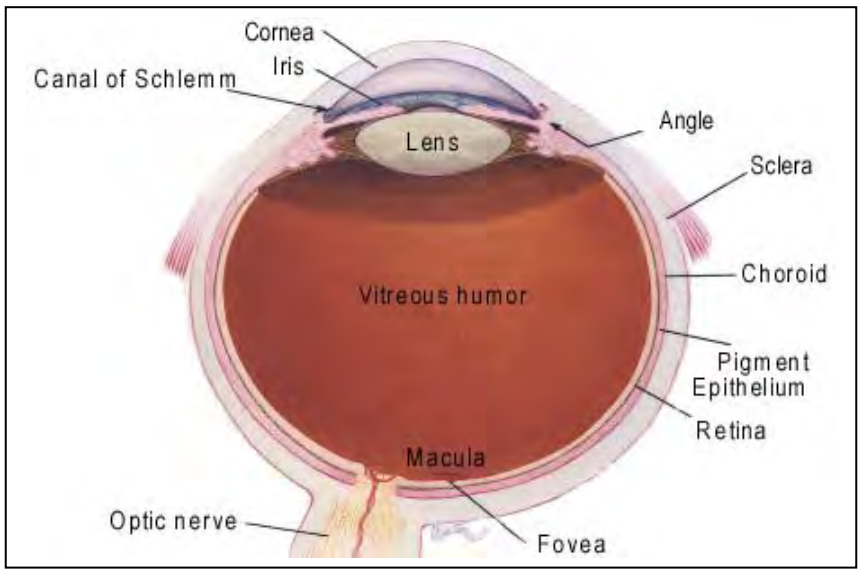
- Elevated Intra-ocular pressure (IOP): can cause blindness
- Average pressure 15 mm Hg (12-20 mm Hg normal)
- Elevations of IOP damage retina & optic nerve (above 20 mm Hg)
- **Chronic open-angle glaucoma (asymptomatic):**
  - Outflow is slowed by trabeculae
  - NO SYMPTOMS – must be tested for eye pressure to detect
  - Most common, over age 30, familial, gradual, DM, blacks > whites
- **Acute angle-closure glaucoma (symptoms):**
  - Iris acutely blocks outflow (angle of outflow is blocked)
  - Very symptomatic, acute, often unilateral attack, with GI symptoms and headache
- **Congenital glaucoma**

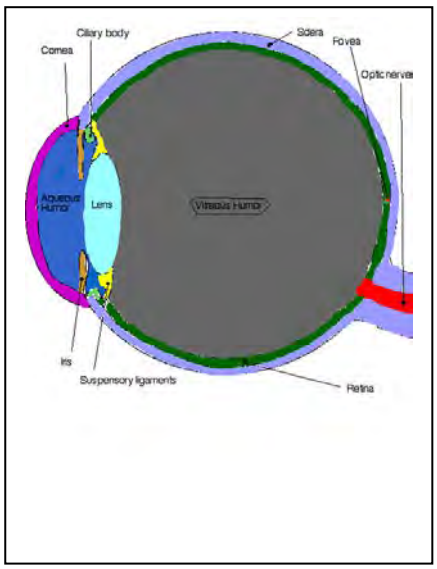


Normal disc (left) and cupping of disc (right) in glaucoma as seen on retinal exam. The cupping looks as though the disc is bulging out at you .



Vitreous humor:

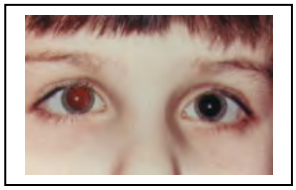




Vitreous hemorrhage:

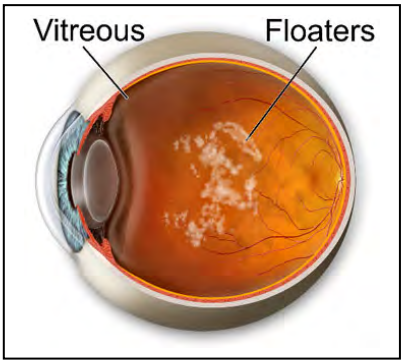
- shining a light on the eye & there is a "*black reflex"* seen on ophthalmoscopy
  - instead of a normal "red reflex" of the retina flashing back at you, due to the hemorrhage there is an abnormal "black" reflex" (the retinal flashback is blocked by the blood)
- Etiology: Trauma, DM, retinal tears, retinal detachment
- Requires IMMEDIATE care due to complications: Can coagulate and cause worse damage, needs immediate evaluation & treatment

Normal red reflex and abnormal black reflex



"Floaters": "spots before the eyes"

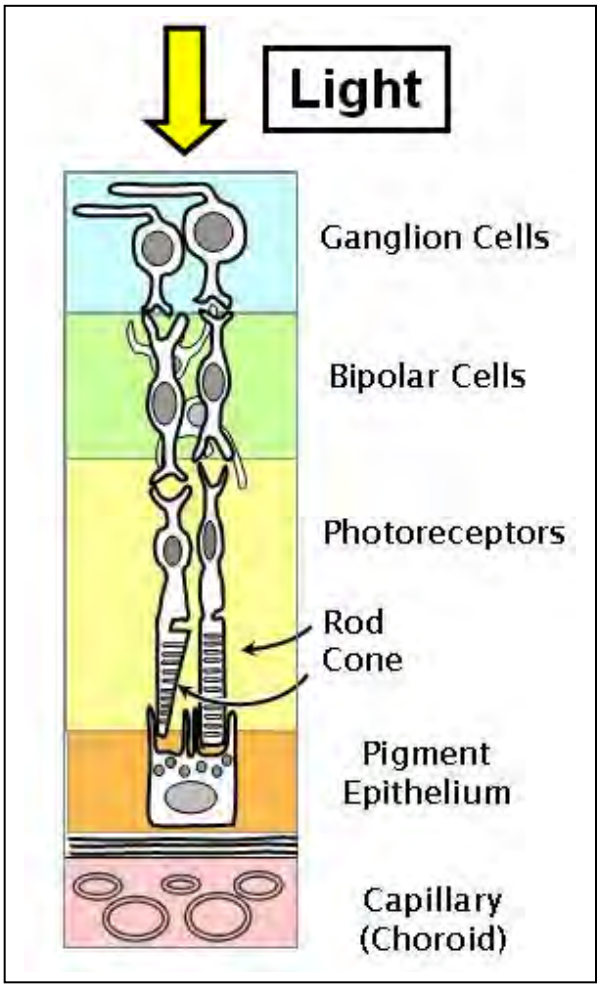
- Temporary contraction of vitreous gel and separation from retina, or cells/debris floating in the vitreous humor
- **One possible serious condition**
  - rarely they signal **retinal detachment**
  - Persistent floaters, especially if accompanied by light flashes, need **retinal evaluation** since it can be a **detached retina**

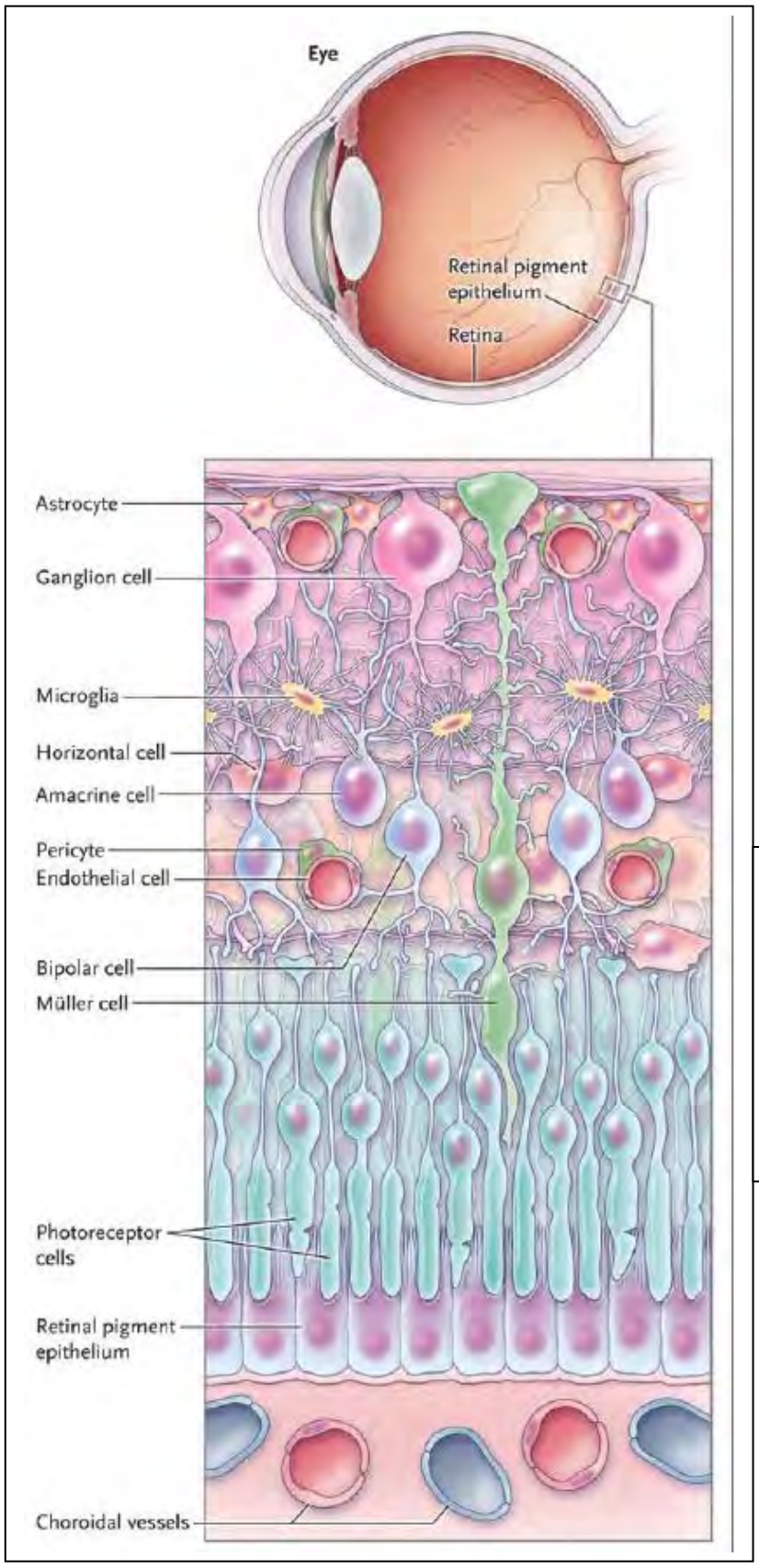


RETINA: visual photochemistry, color vision, dark adaptation

Layers of rods & cones: (more on the photochemistry of vision below)

- **Rod and cone cells** are the receptors that absorb light to create visual sense
  - Contain pigment that absorbs photons of light energy and then activates nerve fibers sending visual sense to the CNS
- Underneath these receptors is a pigmented layer (uveal tract)
  - black background to prevent scattering of light (acts as a mirror)
  - pigment also stores **vitamin A** and sends this to the rods/cones to manufacture the light absorbing pigments
- **Albinism:**
  - albino individuals **lack melanin pigment** and therefore are usually legally blind
- Retina is arranged in layers so normally, several layers of cells lie over the rods & cones
  - In the **fovea centralis**, these layers are pulled over to the side, so light falls directly on the cones.

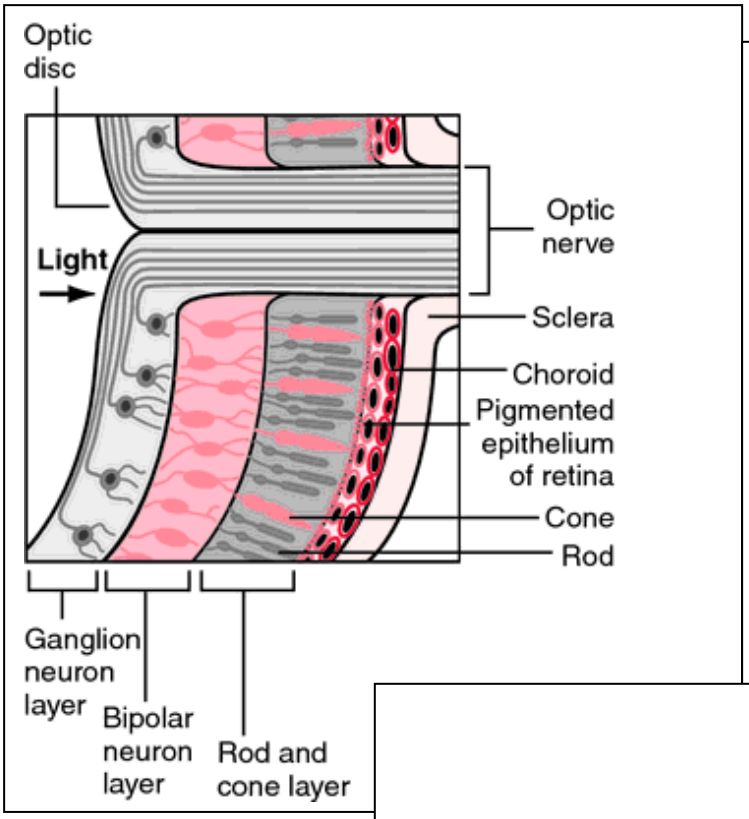


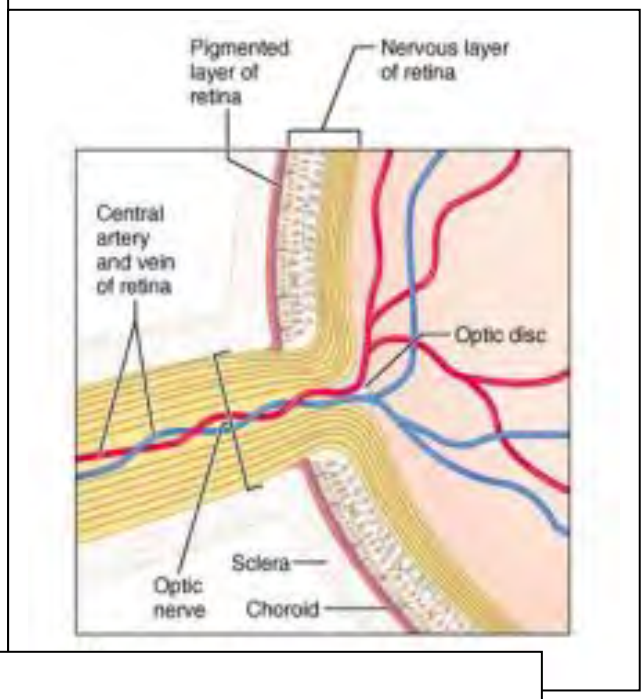


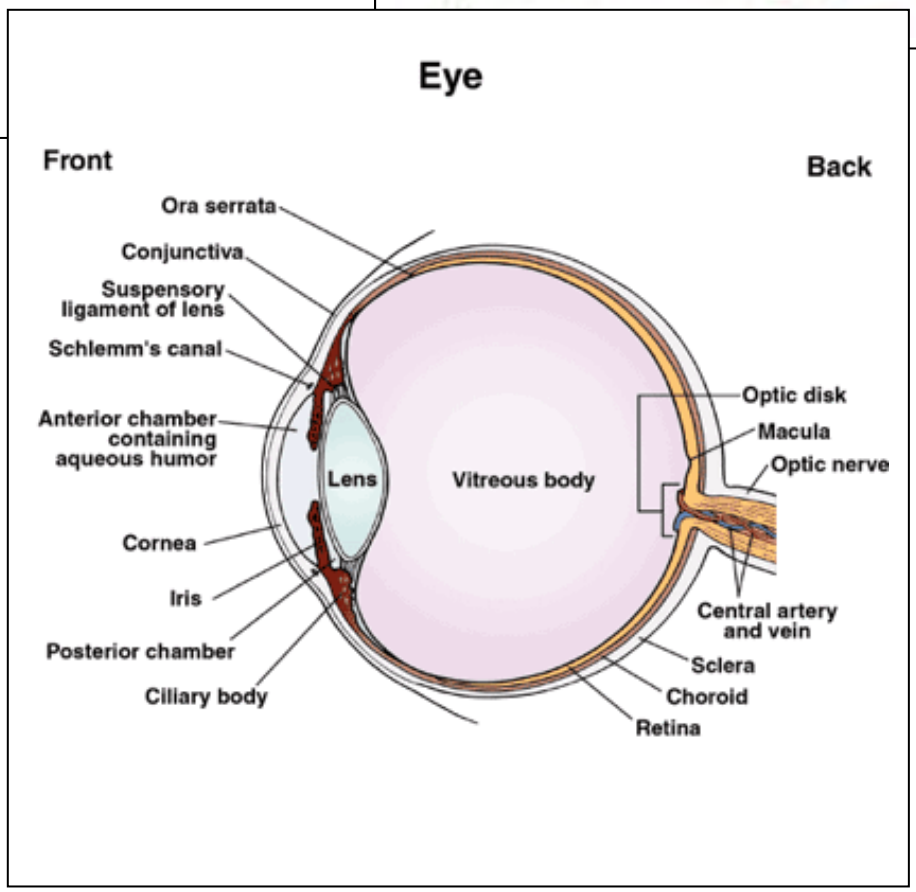
Microanatomy of retinal neurovascular unit.
From: Antonetti DA. Diabetic retinopathy. *N Engl J Med* 29 March 2012;366(13):1227-39. (with permission)

Blood supply of retina:

- **central retinal artery (end artery)** – if occluded, no alternate supply
  - supplies blood to retina, independent of other blood supply to the eye
  - outer edges of retina also get nutrition from choroid diffusion (attached to choroid, which is vascular tissue between the retina & sclera)
- Since this is an **end artery**, so occlusion (e.g., thrombosis) means death to the retina

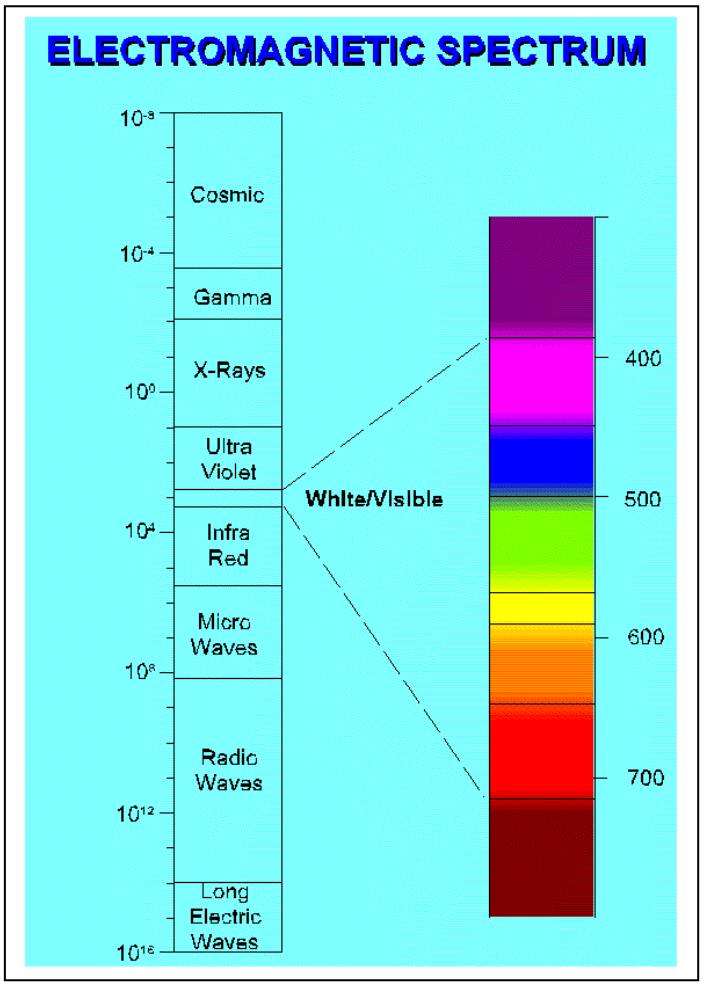


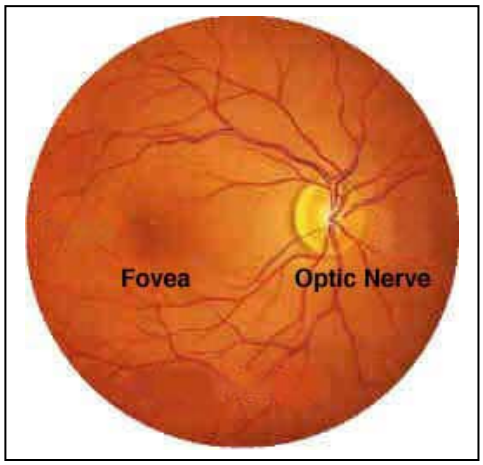




Photochemistry of Vision:

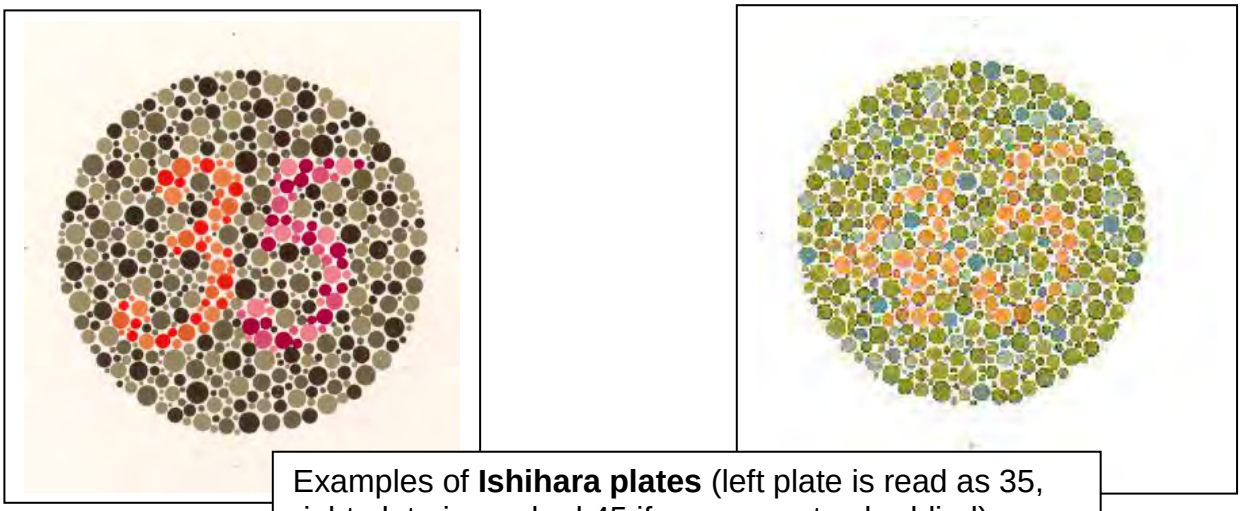
- light energy (photons) hitting the "light pigments" (proteins called opsins + color pigments)
- causes depolarization of the nerve fibers leading from the eye to the CNS (CN II Optic Nerve)
- Color pigments:
  - Rhodopsin (in rods) – excited over entire white light spectrum (can't differentiate colors – so gives us "black & white" vision)
  - Photopsin (in cones) – gives us color vision AND better visual acuity
  - Pigments are based on **vitamin A**
- Rods:
  - More sensitive – needs less light to see: **scotopic vision (twilight vision)**
  - Not as good visual acuity (see less well in darkness)
- Cones:
  - Less sensitive – need more light to be activated but also give us color vision: **photopic vision (color vision in bright light, good acuity)**
  - Normally, light is seen across an entire spectrum from 400 nm to 700 nm.
  - White light is a combination, and green/yellow light is right in the middle.
    400 (violet)--450 (blue) -- 500 (green) -- 550 (yellow) -- 580 (orange) -- 600 (red)
  - The eye distinguishes colors by using cones at different ends of the spectrum
    - cone pigments are blue-sensitive, red-sensitive, & green-sensitive
- **Brain interpretation of color:**
  - brain cells called **blobs** in the primary visual cortex can adjust for different lights
  - computes color constancy by comparing known colors in a scene to other items





Color blindness:

- **Protonope:**
  - missing red cone
  - can't distinguish red/green due to total lack of cones at this end of the spectrum (X-linked recessive)
- **Deuteronope:**
  - missing green cones (still has blue & red)
  - can't distinguish greens well but can see more colors over the entire spectrum since both ends of the spectrum are still represented
  - Very rarely, blue cones are missing
- Use **ISHIHARA TESTING**
  - determine normal color vision or which type of color blindness is present
  - **GREAT SITE**
    - Automated Ishihara colorblind testing online, go to: http://colorvisiontesting.com/ishihara.htm



Examples of **Ishihara plates** (left plate is read as 35, right plate is read ad 45 if you are not color-blind)

Light & Dark Adaptation:

- **Dark adaptation:**
  - bright light degrades light pigments, darkness allows them to reform
  - rods get progressively better at adapting to low-light conditions – the longer you are in the dark, the better your vision (takes about an hour for full dark adaptation)
  - any bright light destroys the adaptation
  - Red light & dark adaptation:
    - If only red cones are excited, this still allows dark adaptation since most of the rods not excited at the far red end (600 nm)
    - Red light allows you to read, etc. at night, while the rest of the eye can remain dark adapted for good night vision
    - Used in plane cockpits & expensive cars
- **Peripheral night vision:**
  - Rods are found more at the periphery, so lateral gaze in low light allows light to fall on the rods, which will "see" the light source better
  - stars in the night sky, for example
- **Pupillary size:** Pupil widens to increase light gathering
- **Purkinje shift:** loss of color vision in the dark (cones can't work)
- **Night Blindness:**
  - not enough vitamin A
  - causes rhodopsin deficiency

Macular Degeneration:

- affects central portion of the retina, including fovea centralis
- seen in age-related blindness, may also occur in uncontrolled diabetes
- may be hereditary

Papilledema (choked disk):

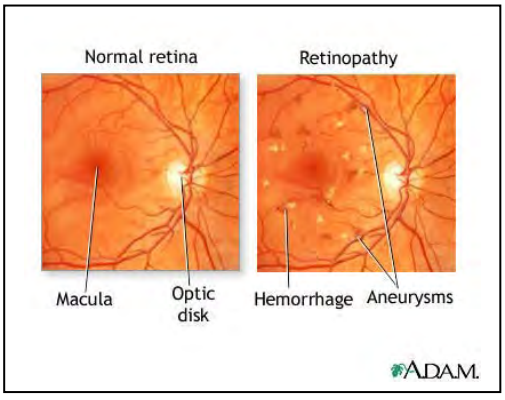
- edema of CN II (optic nerve) head (on retina)
- due to increased intracranial pressure (ICP)

Retinal detachment:

- neural retina pulls away from the pigment retina layers
- can live for days and be reattached due to retinal artery supply

Diabetes Mellitus:

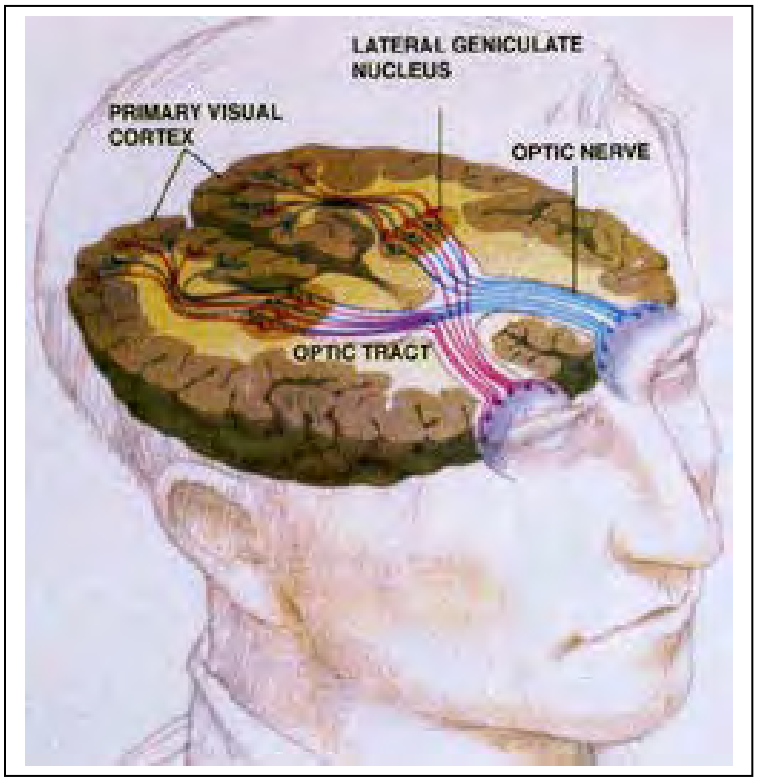
- **retinopathy** with abnormal growth of vessels (proliferative retinopathy), obscuring vision
- Other retinal findings (hemorrhages & exudates, with macular degeneration from edema)



Hemorrhages are the red lesions, exudates are the white lesions. Abnormalities in the vessels are also seen (aneurysms, AV nicking)

CENTRAL NERVOUS SYSTEM (CNS) VISUAL PHYSIOLOGY:

- optic nerves from each eye lead back to the CNS

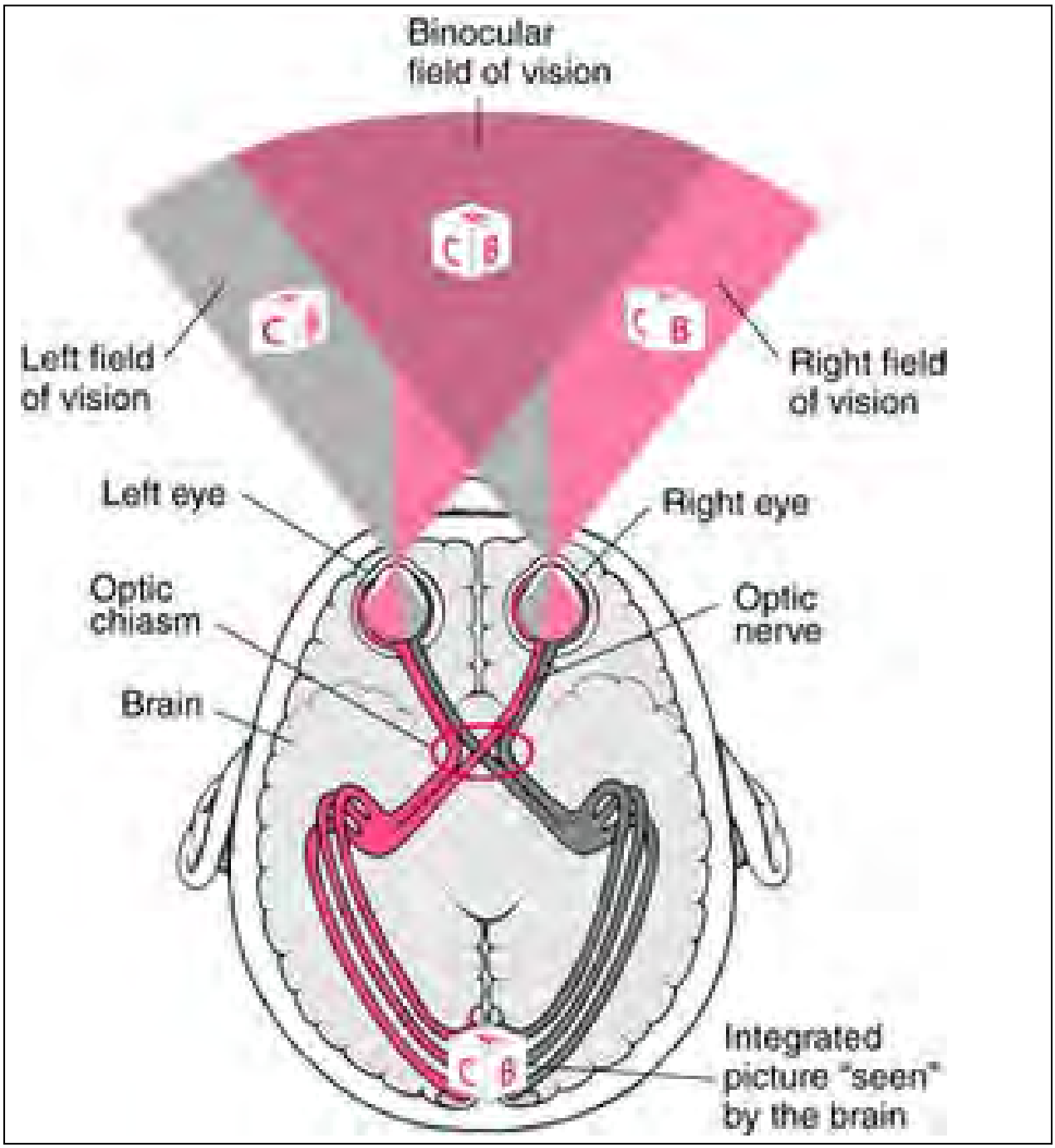


Nerve pathway:

- **Optic Chiasm:**
  - crossing point -- right below the pituitary
  - the nasal (medial) fibers from each retina cross over to the opposite side of the brain, to form the optic tracts
- **Dorsal lateral geniculate nucleus:**
  - fibers synapse here and signals are screened ("gated") & processed
- **Primary visual cortex:** occipital lobe
- **Secondary visual cortex:** all round the primary visual cortex – process the "meaning" of images
- **Other visual processing:**
  - **hypothalamus:** circadian rhythm & release of hormones
  - **pretectal nucleus:** reflex eye focus on important objects & pupillary reflex
  - **superior colliculus:** tracking of moving objects
  - **thalamus:** behavioral response to visual cues
- **Field of vision:**
  - visual field mapping and perimetry
- **Physiological (normal) blind spot:**
  - caused by the optic disc (no rods or cones in retina where nerves exit)
  - just lateral to central vision

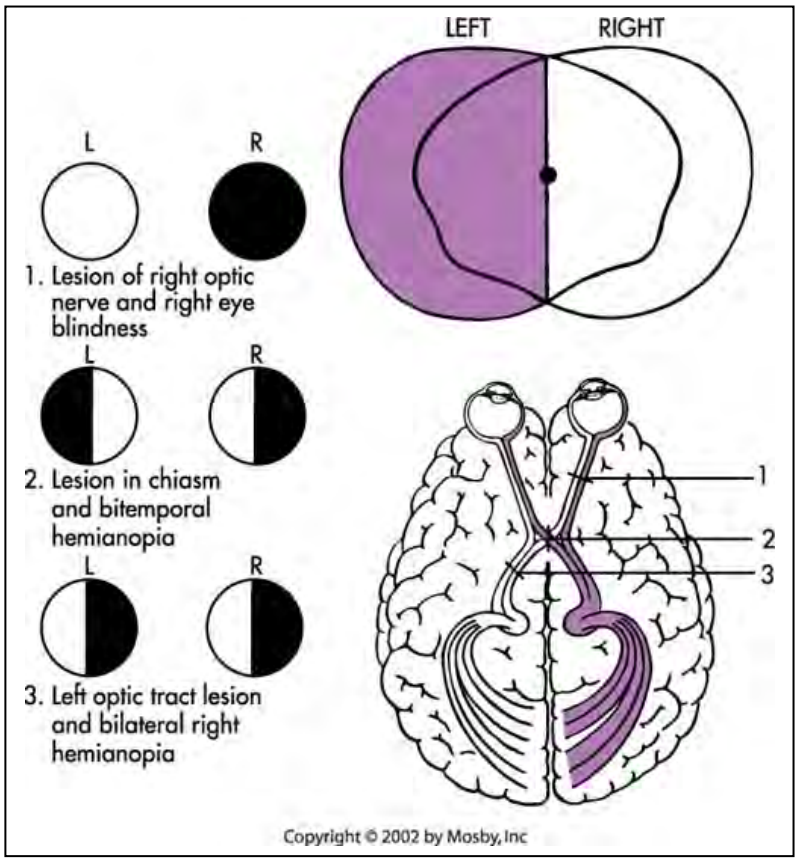
Binocular vision:

- Visual fields testing: http://www.neuroexam.com/neuroexam/content.php?p=18
- **Video:**
  - http://library.med.utah.edu/neurologicexam/html/cranialnerve_normal.html#06
  - scroll down to VISUAL FIELD testing



Visual Field Deficits (anopsia):

- **bitemporal hemianopsia:**
  - destruction of the optic chiasm, such as from pituitary tumor
  - nasal fibers are destroyed, and the temporal field is therefore gone due to image inversion on the retina
  - sideswipe their cars while parking **("tunnel vision")**
- **homonymous hemianopsia:**
  - destruction of an optic tract on one side after the optic chiasm,
  - complete loss of vision in inner half of one eye and outer half of the other eye
- **central sparing:**
  - thrombosis of posterior cerebral artery
  - destroys visual cortex except for part of the foveal visual area
- **unilateral blindness:**
  - lesion of optic nerve and/or central retinal artery occlusion
  - e.g., from thrombosis
- **Amaurosis fugax:**
  - transient visual loss
  - often seen in multiple sclerosis and temporal arteritis



Scotomata:

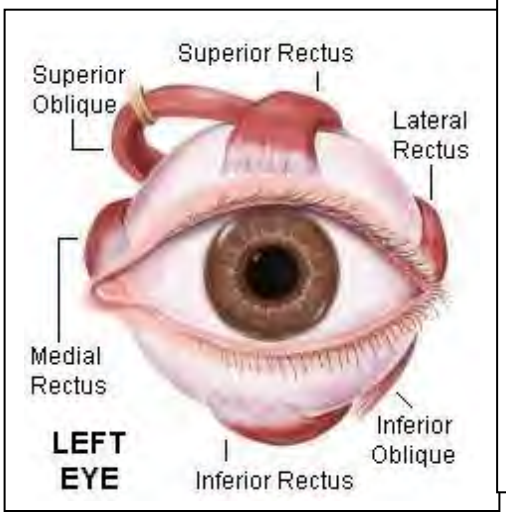
- **non-physiologic blind spots** (pathological)
- indicates severe ophthalmic or CNS illness
- **Damage to optic nerve:**
  - pressure (e.g glaucoma), toxins (lead, cigarette smoking)
  - infections (toxoplasmosis), degeneration (retinitis pigmentosa with abnormal deposition of melanin)
  - optic nerve inflammation (retrobulbar neuritis as seen in MS)

EXTRA-OCULAR MUSCLES (EOM) EYE MOVEMENTS: three pairs of muscles.

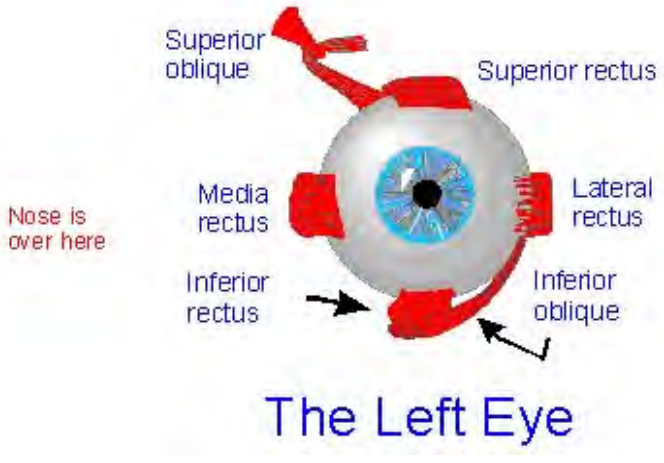
- Movements and muscles: (rectus muscle, plural recti)
  - side to side (medial & lateral recti)
  - upward and downward (superior & inferior recti)
  - rotation (superior & inferior obliques)
- Reciprocal innervation: one set of muscles relaxes when the other set is contracted.
- Oculomotor nerves:
  - **CN III → superior, inferior and medial recti**
  - **CN IV → superior oblique**
  - **CN VI → lateral rectus**
- Impact on vision:
  - Upright visual field – superior oblique upward & outward rotation
- Coordination:
  - Visual cortex causes **fusion** of both eyes to converge or diverge together
- Fixation & locking:
  - Voluntary fixation (choose where to look – prefrontal cortex)
  - Involuntary fixation now takes over to keep us looking there
- Opticokinetic movements:
  - When moving, eyes jump from spot to spot and with blend the images

http://anatomyresources.hsc.wvu.edu/ReillyWeb/MEP1144/Unit6/images/ExtraocularSkeletalMuscle.swf

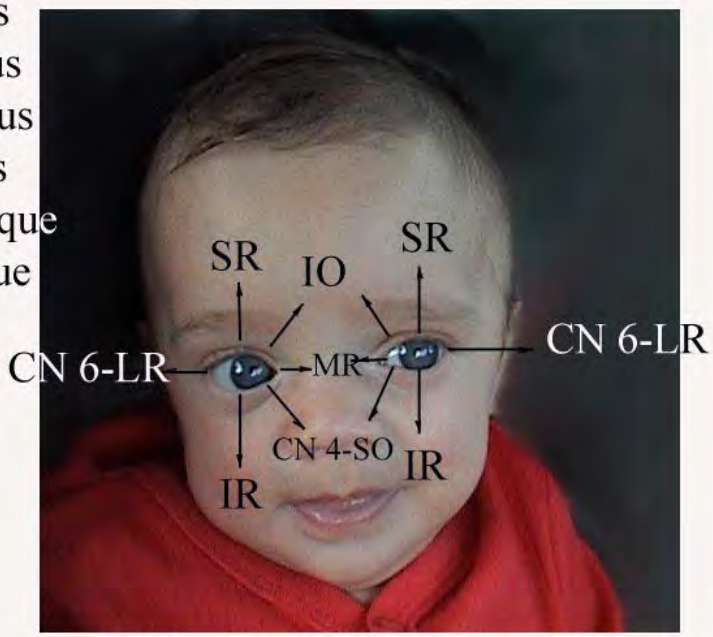
THIS IS A **MUST VISIT!!!**
WHAT NERVES CONTROL WHICH MOVEMENTS??? Play with the website.



- Medial rectus moves the eye towards the nose
- Lateral rectus moves the eye away from the nose
- Superior rectus moves the eye up
- Inferior rectus moves the eye down
- Superior oblique rotates the eye so that the top of the eye moves towards the nose.
- Inferior oblique rotates the eye so that the top of the eye moves away from the nose



LR - Lateral Rectus
MR - Medial Rectus
SR - Superior Rectus
IR - Inferior Rectus
SO - Superior Oblique
IO - Inferior Oblique



With head stationary, eyes follow object from center to periphery

Strabismus:

- Also called squint, cross-eyedness
- Lack of fusion of eyes to visual coordinates
- Abnormal conjugate fusion patterns develop in childhood – may be idiopathic, due to CNS disease, due to thyroid disease
- Symptoms of **diplopia** (double vision)
- **Lazy eye:**
  - If one eye is used all the time, the other eye loses neuronal connections and becomes blinded (lazy eye)
  - if forced to work (blindfold working eye) in childhood, can regain vision

The two most common types of strabismus—**esotropia and exotropia**:

OD (Right Eye) Esotropia

OD (Right Eye) Exotropia

Nystagmus:

- Involuntary unilateral or bilateral rhythmic movement of the eyes, at rest or with movement
- Due to imbalance in coordination, especially involving vestibular nuclei
- Seen in drug use (especially illicit drugs)

Gaze Palsies:

- can't perform conjugate eye movement in one direction
- Usually due to cerebrovascular disease (stroke)
- May also cause paralysis of upward gaze (tumor, stroke)

OTHER:

Amblyopia:

- dimness or reduction of vision but nothing wrong with refraction or obvious problems with the eye
- Usually found in chronic illness
  - DM, renal failure, toxins (alcohol, smoking)
  - Extreme age

TASTE:

Function:

- able to separate lethal from nutritious foods
- select foods that are needed by the body for current metabolic needs

Fabulous Website: (Many pictures from: Cardiff University, Wales, UK, tutorial by Tim Jacob)

- Taste: http://www.cardiff.ac.uk/biosi/staffinfo/jacob/teaching/sensory/taste.html

Taste sensation: all of these contribute to taste sensation →

Contributors to taste:

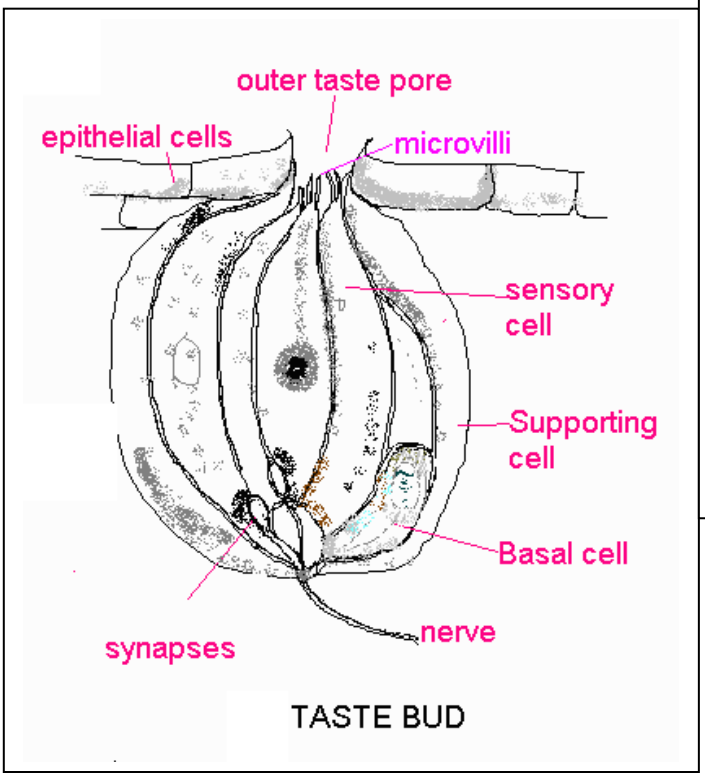
- **smell**
- **texture of food**
- **toxins** (e.g., pepper which stimulates pain fibers)

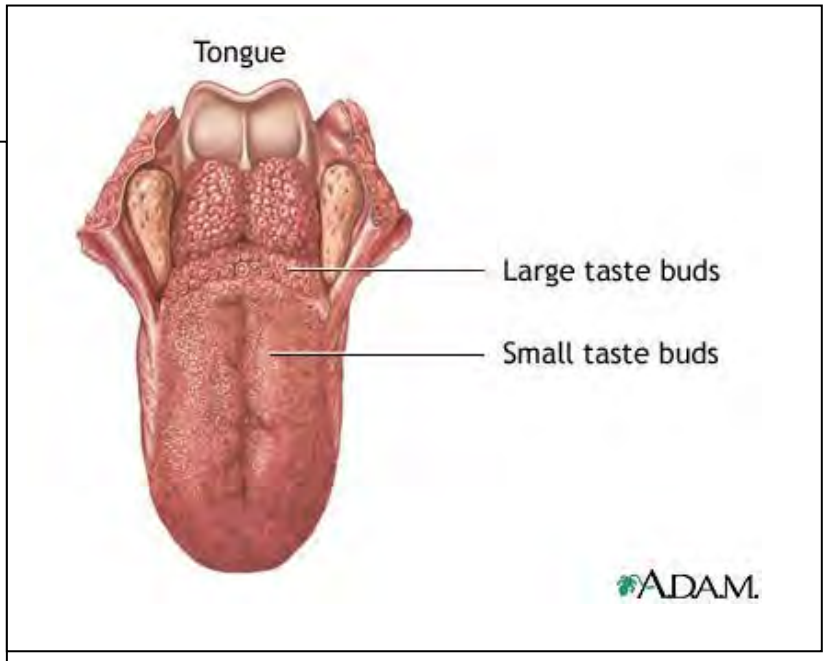
Taste receptors:

- Chemical taste receptors in **taste bud cells**, which line the **taste pores** that go down into the tongue like crevasses
- **Any compound we taste must be dissolved in water, so it trickles down into the pore and then hits a receptor on the taste bud cell**
- Located from the papillae of tongue all the way down to the epiglottis & proximal esophagus

Taste ligands:

- **electrolytes**
- **organic molecules**

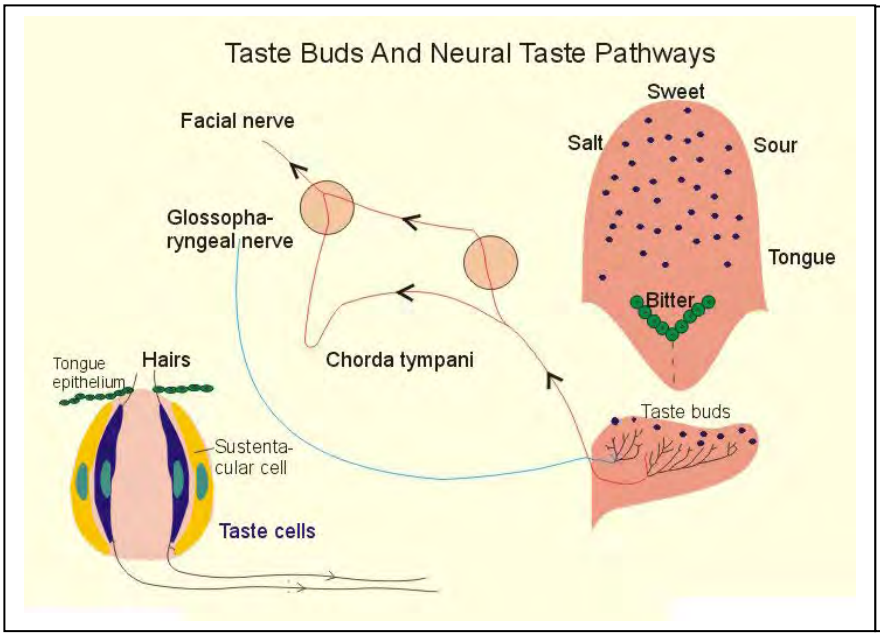




Primary sensations of taste:

- chemical receptor sensations combine to form the primary sensations of taste
- **salt** (ionized salts, anions & cations)
- **sour** (acids -- H^+ ion concentration)
- **sweet** (multiple chemicals including sugar, alcohols, ketones, esters, amino acids, proteins, halogenated acids, salts of heavy metals)
- **bitter** (multiple organics, especially nitrogen based and alkaloids, such as medications)
  - Most sensitive (lowest threshold) is for bitter taste (protective function)
- **Umami** (Japanese for "deliciousness" – a "meaty" taste)

Taste blindness: hereditary lack of receptors for that ligand



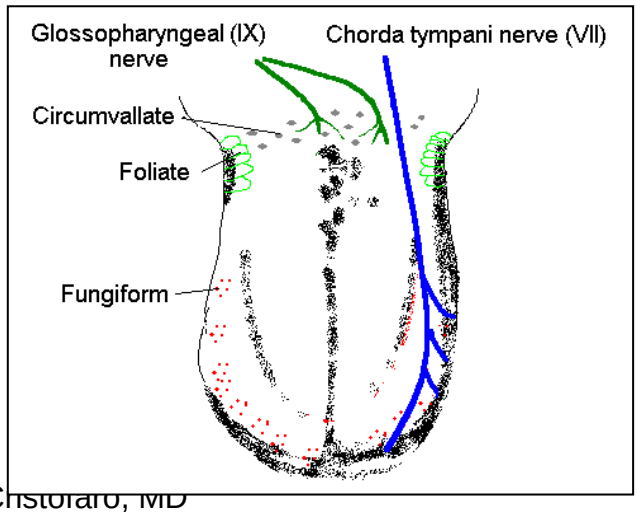
This "taste map" is probably not as accurate as was previously thought -- the arrangement of the taste buds is more scattered throughout the tongue.

Afferent neural pathways:

- *Anterior 2/3 of tongue & pharynx* carried by CN V → chorda tympani → facial nerve (CN VII) → tractus solitarius in brain stem
- *Back of tongue & mouth* carried by CN IX (glossopharyngeal) → tractus solitarius in brain stem
- *Base of tongue* →CN X vagus → tractus solitarius in brain stem
- → the **thalamus** → travel to the **parietal cortex** (cerebral cortex taste center)

Adaptation to taste:

- In the CNS
- Not at the level of the taste buds



Hypogeusia** or **ageusia:

- Can result from damage to afferent nerves carrying specific sensations
- Damage to CN IX (glossopharyngeal for posterior tongue) → loss of bitter sense
- Damage to CN VII (facial for anterior 2/3 of tongue) → loss of sour, sweet, salt senses

Parageusia:

- taste **incorrectly perceived** (unpleasant)
- chemotherapy, age, brain trauma

Presbygeusia:

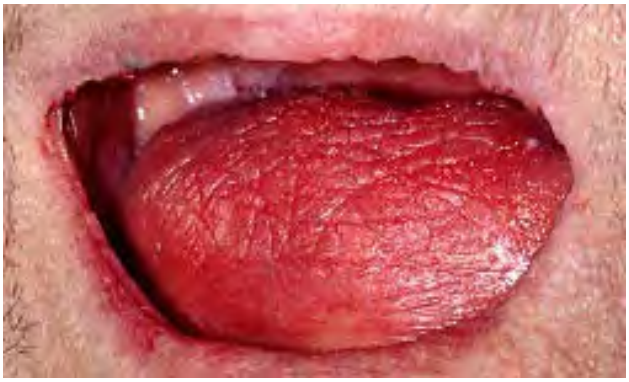
- reduced taste perception in aging
- decreased papillae, saliva, amylase
- weight loss and nutritional deficiencies

Chemical composition of saliva: necessary for taste – need fluid to dissolve taste ligands

- **pH:** pH 7.0 (neutral) to slightly alkalotic (pH 8.0)
- **Mucins:** lubricate food & protect oral mucosa.
- **Digestive enzymes:**
  - start the digestive process
  - secretory **zymogen** granules are released from acinar cells into the ducts
- **Immunologic:**
  - contain **IgA**, **lysozyme** (attacks bacterial cell walls)
  - **lactoferrin** (binds iron & is bacteriostatic
  - **proline-rich proteins** (bind toxins & protect toot enamel).
- **Sialography** can be done to map the salivary glands (imaging)

Xerostomia:

- deficiency of saliva causes **xerostomia –** extreme dryness of mouth & tongue
  - dental caries
  - dry cracked mucosa
  - adverse changes in taste
  - adverse changes in swallowing & speech
  - worsened heartburn of GERD (if present)



Xerostomia: note cracking and fissuring of lips and tongue

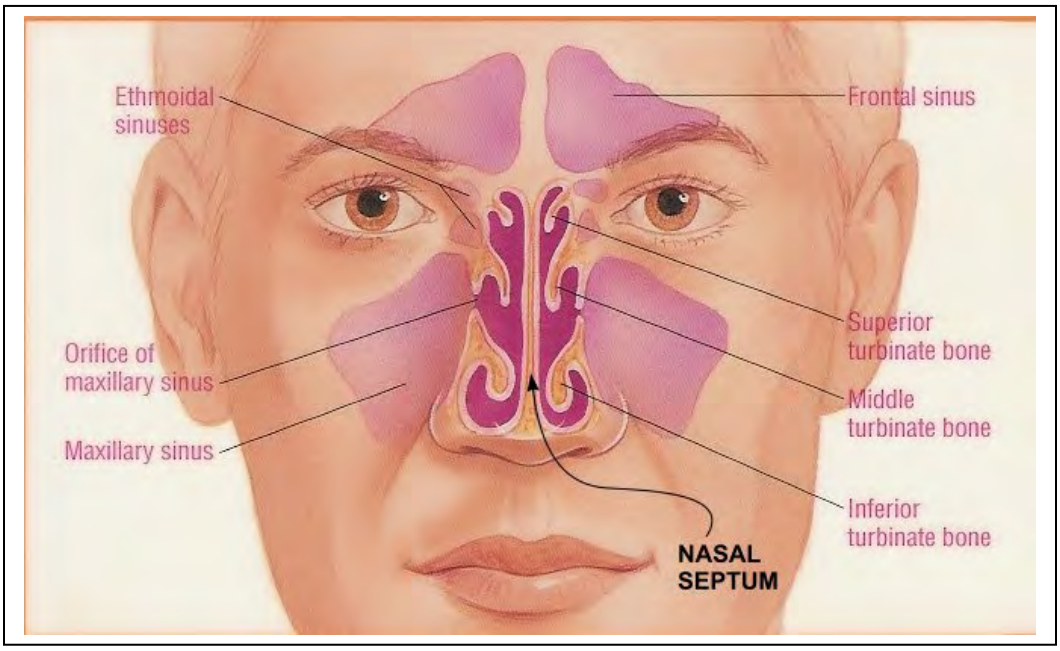
OLFACTION (SMELL):

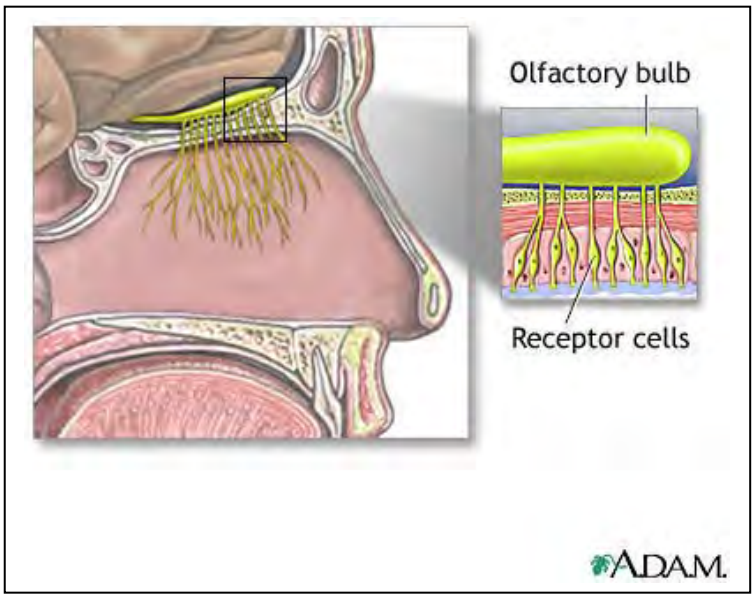
***Really Great* weblink:**

- Lots of fun stuff from Prof. Jacob at Cardiff Univ – physiology AND pathophysiology
- http://www.cardiff.ac.uk/biosi/staffinfo/jacob/teaching/sensory/olfact1.html

Structures:

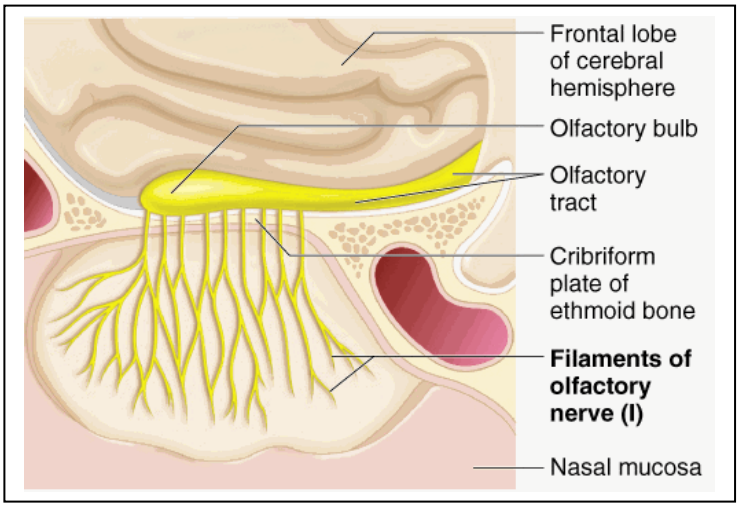
- **Olfactory membrane:**
  - covers superior part of each nostril
  - folded over **superior turbinate** and portion of **middle turbinate**
- **Olfactory cells:**
  - nerve cells of CNS origin
  - have specialized **olfactory hairs (cilia)** projecting into mucus coating nasal cavity
- **Glands of Bowman:** secrete **mucus** onto surface of olfactory membrane





Function:

- Odorants – ligands for smell:
  - a substance that is volatile, water soluble and lipid soluble
  - must travel in air, pass thru mucus
- Excitation of receptors and nerve pathways:
  - **Odorant** binds to receptor on ciliary membrane of olfactory cell
  - CN I (olfactory nerve) to the **olfactory bulb**
  - To various locations in brain (a lot in the temporal area of brain) for processing of smell
- Adaptation:
  - smell can extinguish within one minute – occurs in CNS
- Primary sensations of smell:
  - so far have identified 7 pirmary types of smells: camphoraceous, musky, floral, peppermint, ethereal, pungent, putrid
  - there are probably really over 1,000 – since our "primary smells" are probably combinations of all these separate receptors that are sensitive to different odorants



Odor blindness: hereditary lack of receptors for that particular odorant

Hyposmia or anosmia

- usually from nasal inflammation
  - smoking, infection, rhinitis
- unilateral = olfactory bulb or nerve tract damage (e.g., tumor)

Olfactory hallucinations:

- temporal lobe seizures
- schizophrenia
- sometimes migraine ("aura")

Parosmia:

- abnormally perceived smell
- seen in severe **depression**
- also seen in sinusitis