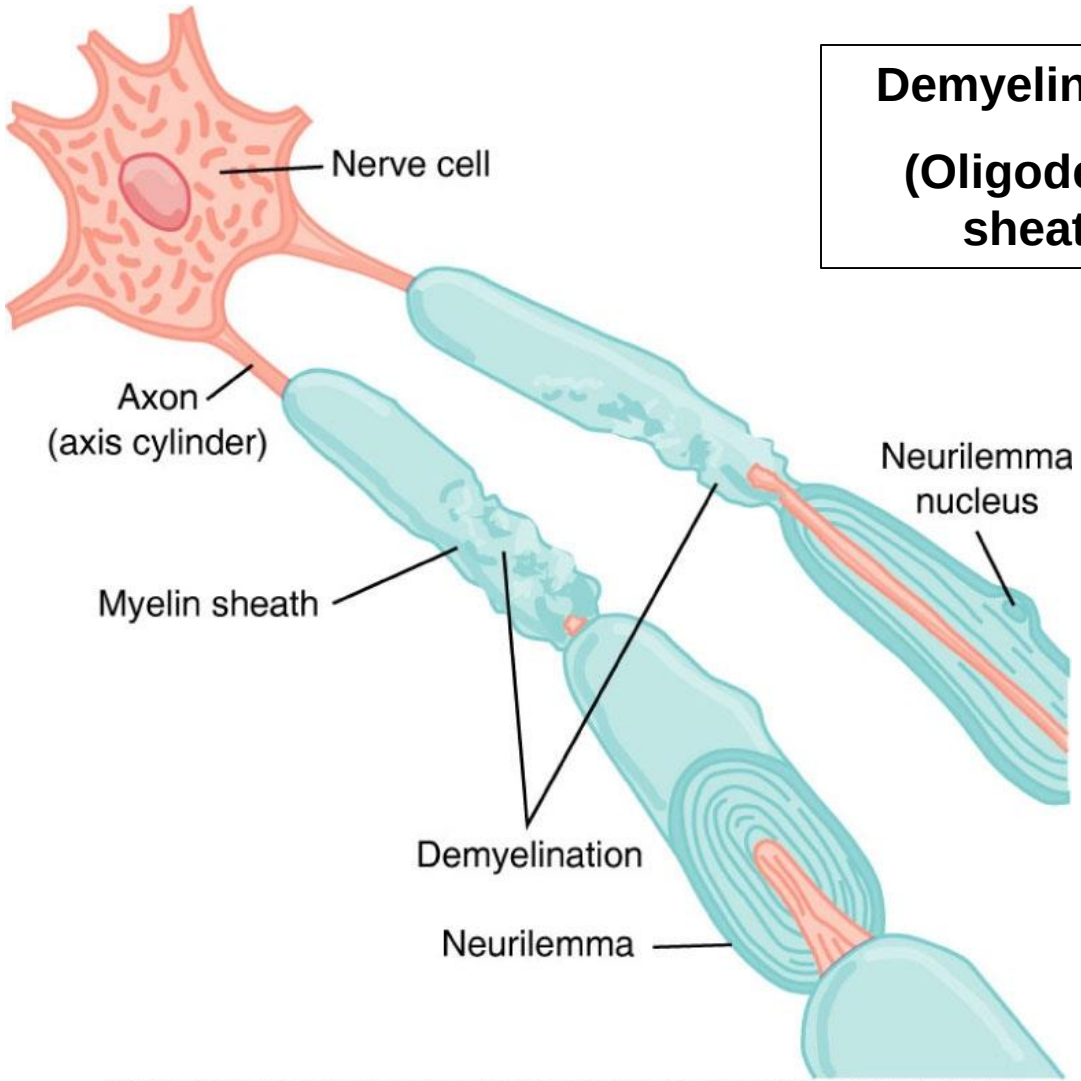
Lecture 7C
(PNS and Spinal Cord Disorders)
Multiple Sclerosis
Guillain-Barre Syndrome
Spina Bifida
ALS
Bell Palsy
Spinal Cord Injury

Multiple Sclerosis

Etiology

- Autoimmune disorder that results in damage to the myelin sheath of **CNS neurons (Oligodendrocytes form the myelin sheath in CNS neurons.)** in the brain and spinal cord.
- About 400,000 Americans have MS.
- Diagnosis is usually made at age 20-50 years; more common in females than in males
- More common in people of northern European descent who live above the 37th parallel, or spent their first 15 years in northern regions.
- Viral or toxin exposure may trigger attacks in genetically predisposed individuals.
- About 15% of affected individuals have an affected relative.
- Certain MHC alleles are more common in patients with MS.

Multiple Sclerosis



From Black JM, Hawks JH, Keene AM: Medical-surgical nursing: clinical management for positive outcomes, ed 6, Philadelphia, 2001, Saunders, p 2013.

Demyelination of CNS Neurons in MS

(Oligodendrocytes form the myelin sheath around CNS neurons.)

Multiple Sclerosis

Pathogenesis

- Structures affected: brain, cranial nerves, corticospinal tracts (upper motor neurons), cerebellar tracts
- **Lower** motor neurons are **not** affected.
- Both humoral and cellular autoimmune factors are involved
- Some remyelination occurs, but it is slow and partial.

Clinical Manifestations

- Extremely variable-motor, sensory, cerebellar, bowel, bladder, cognitive, and emotional effects.
- Some forms exhibit periods of **exacerbation and remission**.
- Exacerbated by heat, infection, trauma, and stress.
- Relapse after pregnancy is common.

Multiple Sclerosis

Diagnosis and Treatment

- No conclusive test for MS.
  - MRIs may show presence of demyelination (plaques).
  - Lab tests may show lymphocytosis and elevated IgG in CSF.
  - Diagnosis is one of exclusion.
  - CNS lesions that are **disseminated in time and space** is one criterion for diagnosis. That means lesions in one location heal over time, but more lesions occur, over time, in different locations.
- There are four clinical types of MS based on the course of the disease. The most common form (85%) is **relapsing-remitting.** Patients with this form of MS, experience acute relapses with periods of remission that last months or years.
- Anti-inflammatory drugs, immune-modulating drugs, and immunosuppressive drugs may be helpful.
- There is no cure.

Guillain-Barre Syndrome

Etiology and Pathogenesis

- Autoimmune inflammatory demyelination of PRIMARILY LOWER MOTOR NEURONS.
- Causes “nontraumatic paralysis”
- Occurs most often in elderly
- Often follows infection, inoculation, or surgery
- Immunologic, but the mechanism is unknown.
  - Aggregates of lymphocytes (B and T) and macrophages are found at the sites of demyelination.
  - Anti-myelin antibodies are found in plasma and CSF.

Guillain-Barre Syndrome

Clinical Manifestations

- Weakness/paralysis starts in the legs and spreads to the arms and face; respiratory muscles may be affected.
- Peak severity of disability occurs in 10 to 14 days.
- Sensory and autonomic function are affected to a lesser extent
- CSF contains elevated protein levels.
- Lab studies and imaging are used to rule out other conditions.
- Majority of patients experience spontaneous recovery; 10%-20% have mild disability.

Guillain-Barre Syndrome

Treatment

- Plasmapheresis (filtering the blood plasma) to remove anti-myelin antibodies is helpful.
- Intravenous immunoglobulin is also helpful in preventing infections in those with compromised immune systems.
- Patients may require intensive care for ventilation and circulatory support.

Amyotrophic Lateral Sclerosis (ALS)

Etiology/Pathogenesis

- Progressive degeneration of BOTH THE UPPER AND LOWER MOTOR NEURONS.
- About 12,000 Americans have ALS, more common in men than women
- Death due to respiratory failure usually occurs within 3-5 years of diagnosis. Stephen Hawking was an exception to this rule!
- The majority of cases are sporadic, but 5%-10% are familial.
- Mutations linked to a familial form are in the gene that codes for **superoxide dismutase 1 (SOD1)**, an enzyme that destroys oxygen free radicals.
- Other genetic defects suggest neuronal problems with RNA processing, protein recycling, neuron structure or glial cell defects.
- High levels of **glutamate** are found in the CSF of some ALS patients.

Amyotrophic Lateral Sclerosis (ALS)

Clinical Manifestations

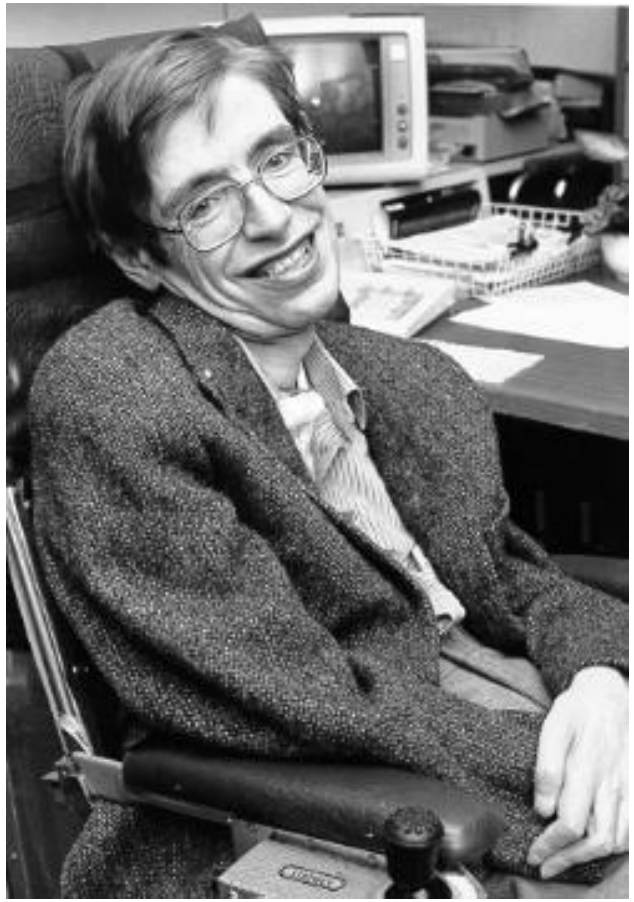
- Early signs: twitching, cramping, and stiffness in the hands and upper limbs
- Later signs: weakness in muscles of swallowing, speech, and respiration, severe **muscle atrophy.**
- Death from **respiratory failure** (atrophy of respiratory muscles) normally occurs in 3 to 5 years.
- Most patients **retain sensory and cognitive** function (Lou Gehrig and Stephen Hawking).
- Electromyelogram (EMG), nerve conduction studies, MRI, and blood tests are used to rule out MS, CNS tumors, HIV, and Lyme disease.

Treatment

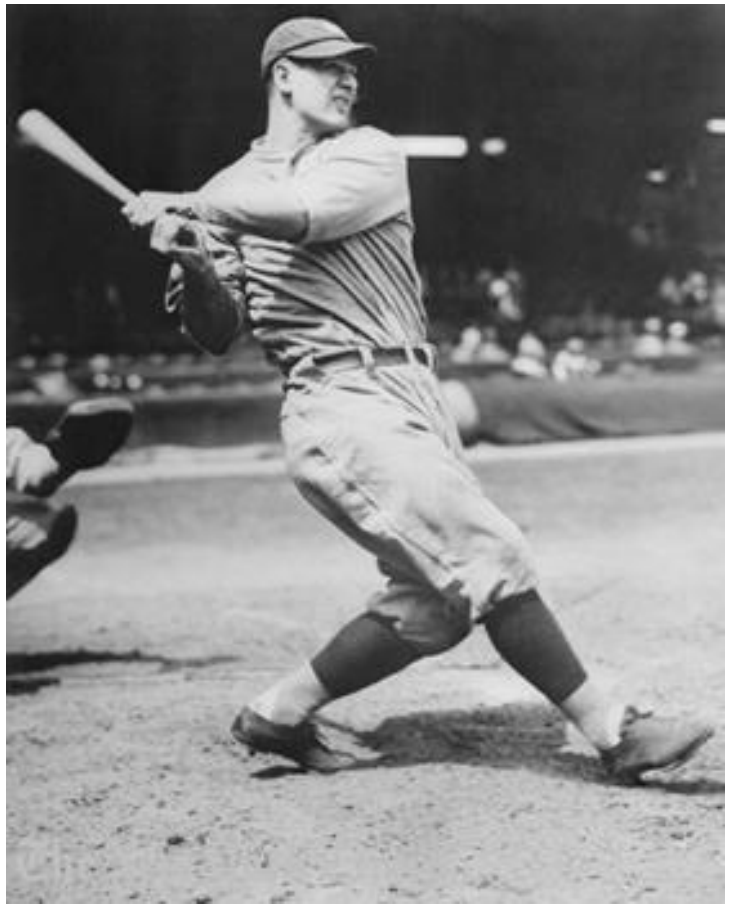
- The only treatment is the drug, riluzole (Rilutek), a **glutamate inhibitor.**
- It is not a cure but may delay the need for mechanical ventilation.

Amyotrophic Lateral Sclerosis (ALS)

Stephen Hawking



Lou Gehrig



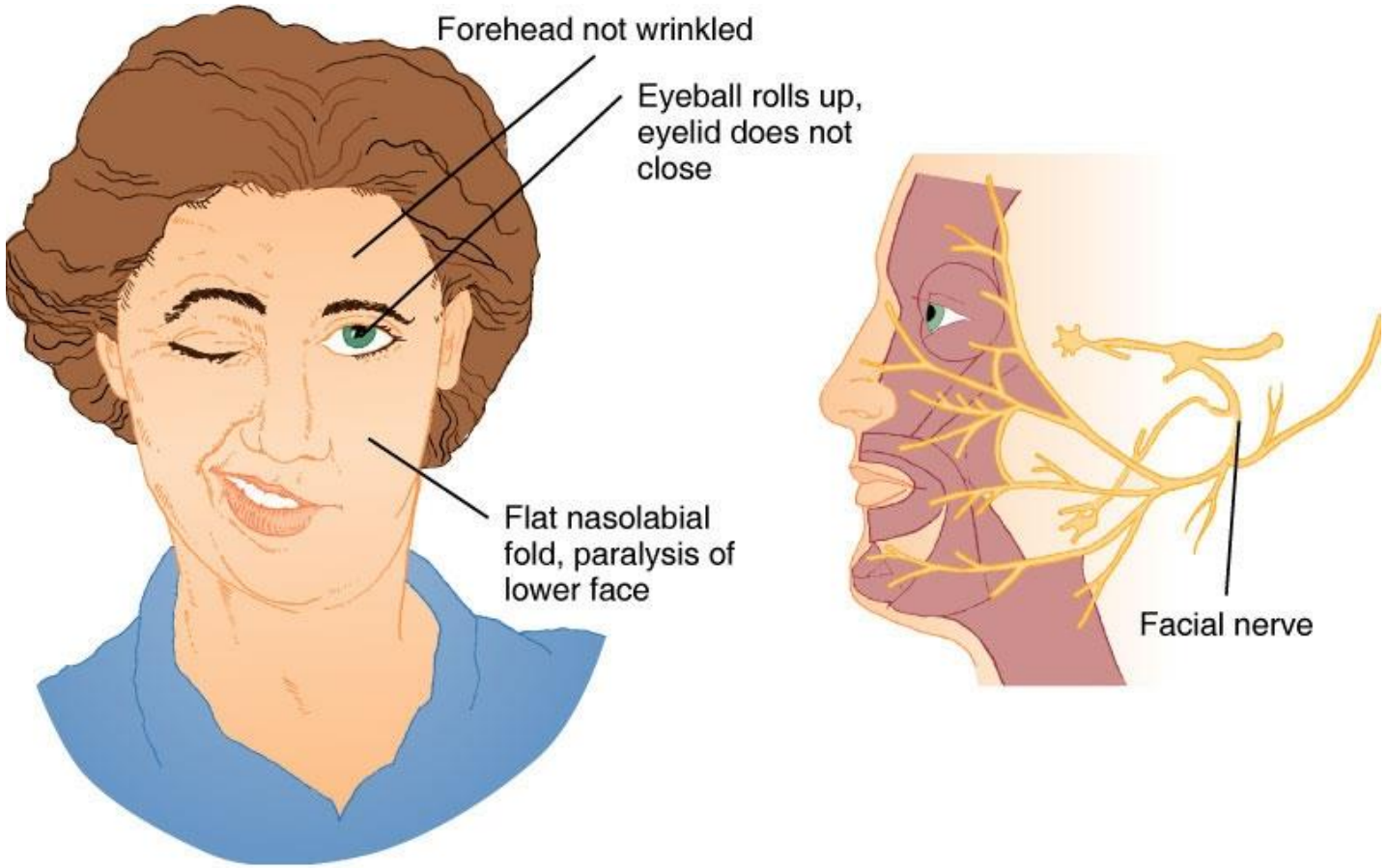
Bell Palsy

Etiology

- Acute paralysis or paresis due to inflammatory injury to cranial nerve VII (facial).
- The incidence peaks in the 40s.
- Evidence is mounting that the cause is viral infection.
- Antibodies to the **herpes simplex and herpes zoster** virus have been found in some patients with Bell palsy.
- Other causes of facial paralysis (stroke, otitis media, tumor, trauma) must be ruled out.

Bell Palsy

Bell Palsy: Left Facial Nerve



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Bell Palsy

Clinical Manifestations

- Symptoms develop rapidly over 24 to 48 hours
- **Unilateral** facial weakness with facial droop, diminished eye blink
- **Hyperacusis**-heightened sensitivity to sound
- Decreased **lacrimation** (tear production) may injure the cornea of the eye.
- Patients complain of a heavy sensation in the face and **decreased sense of taste**.
- Posterior auricular (behind the external ear) pain may be present.

Bell Palsy

Treatment

- Prevention of corneal damage is vital.
- Drops, ointment, and eye patching may be necessary.
- Most patients recover spontaneously in within 3 weeks.
- Corticosteroids plus anti-viral meds have been used with success.
- About 15% are left with residual disability

Spina Bifida

Etiology and Pathogenesis

- A neural tube developmental defect; the bones of the vertebral column fail to close completely around the spinal cord.
- The spinal cord and its meninges may or may not protrude through the vertebral column.
- Cause is unknown, but both genetic and environmental factors contribute
- Risk factors: alcohol, folic acid deficiency, congenital Rubella infection, advanced maternal age, maternal obesity, maternal diabetes
- Maternal use of the anti-acne drug, Accutane, and certain anticonvulsant drugs are also risk factors.

Spina Bifida

Clinical Manifestations

- **Spina bifida occulta:** No cyst formation occurs.
- **Spina bifida cystica:** A cyst protrudes from the vertebral column defect. There are two types of cysts. Both may be diagnosed prenatally by ultrasound or alpha fetoprotein testing.
  - **Meningocele-**The cyst is meningeal sac filled with CSF; occurrence in the cervical, thoracic and lumber regions is equally likely.
  - **Myelomeningocele-**The cyst is a meningeal sac (in the lumbar or sacral region) filled with CSF and contains spinal cord neural tissue.
- Delivery by C-section reduces risk of cyst leakage and damage to the spinal cord. Surgical closure is attempted soon after delivery. Closure *in utero* is sometimes done.
- **Permanent neurological damage** usually occurs: motor and sensory defects below the defect, bowel/bladder dysfunction, scoliosis, hydrocephalus, seizures.
- Growth causes tension on the cord and possible tethering.

Spina Bifida

Treatment, Prevention and Prenatal Testing

- Treatment depends on location and severity of the defect.
- **Folic acid** supplementation before and during pregnancy has been shown to decrease the occurrence of neural tube defects.
- **It is recommended that all women of childbearing age take 0.4 mg of folic acid daily.**
- Alpha fetoprotein is produced by the liver and the yolk sac of the fetus during pregnancy. Elevated AFP in the mother's blood is considered an indicator of neural tube defects like spina bifida. **AFP testing** is fairly routine during pregnancy. NOTE: AFP is an indicator of neural defects, but **not a cause** of neural tube defects.

Spinal Cord Injury

Etiology

- Greatest incidence between 20 and 24 years
- Three to four times more likely in males
- Motor vehicle accidents are the most common cause.
- Other causes: gunshot wounds, falls, recreational accidents, birth injuries, herniated discs or bone spurs related to aging.

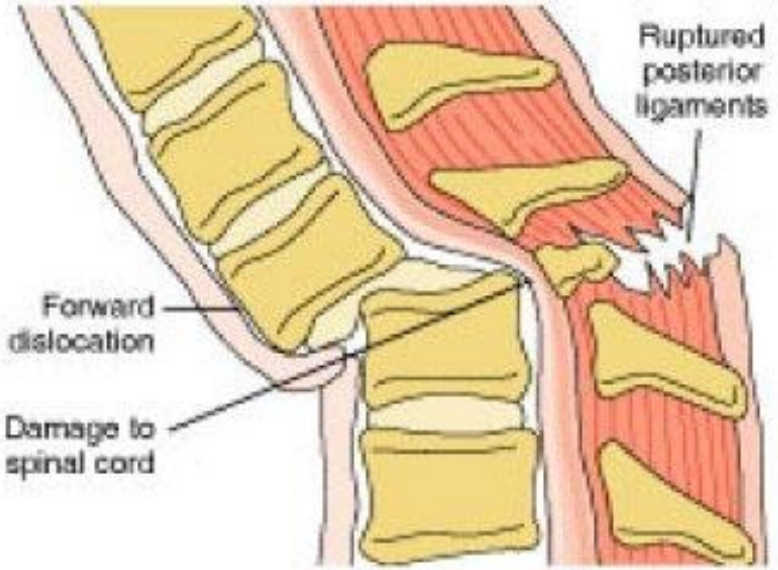
Pathogenesis

- Hyperflexion, hyperextension, or compression of the spinal cord.
  - **Hyperflexion** injury is the most unstable and serious injury.
  - **Hyperextension** is the most common.

Spinal Cord Injury

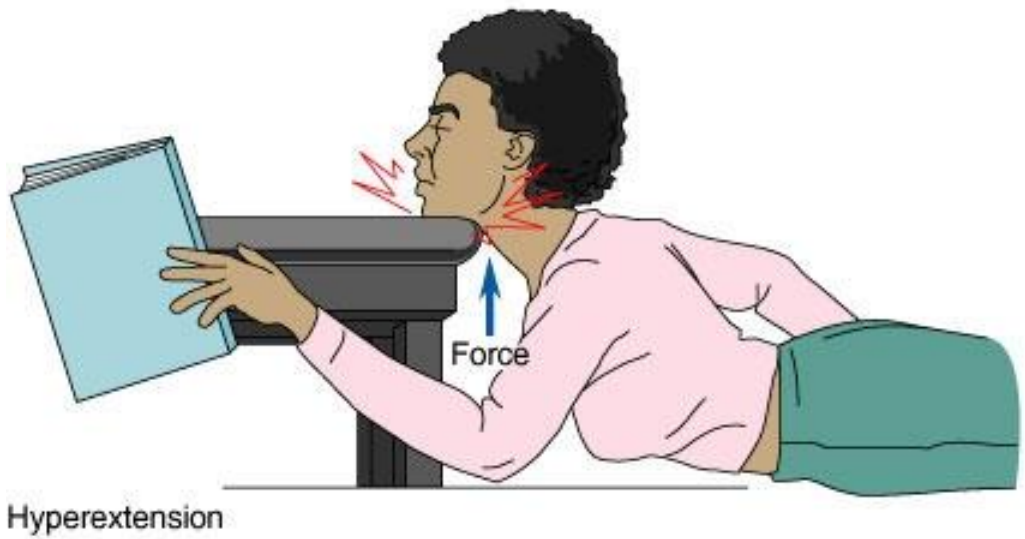
Hyperflexion:
Most Dangerous

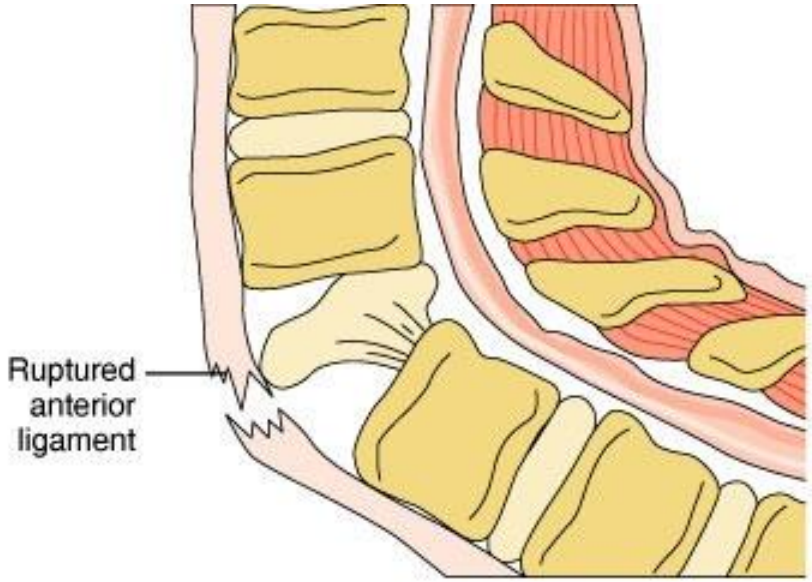
Flexion



Spinal Cord Injury

Hyperextension:
Most Common

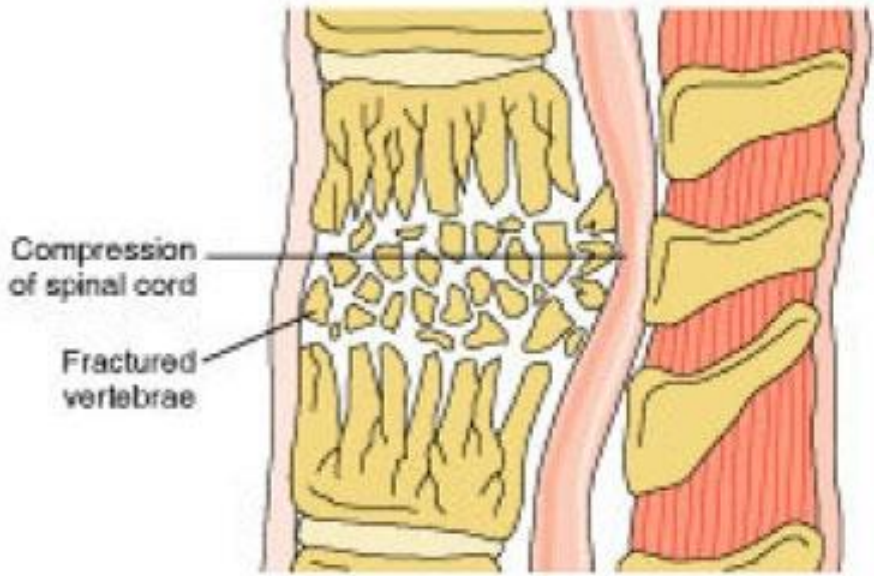




Spinal Cord Injury

Compression

Compression



Spinal Cord Injury

Pathogenesis

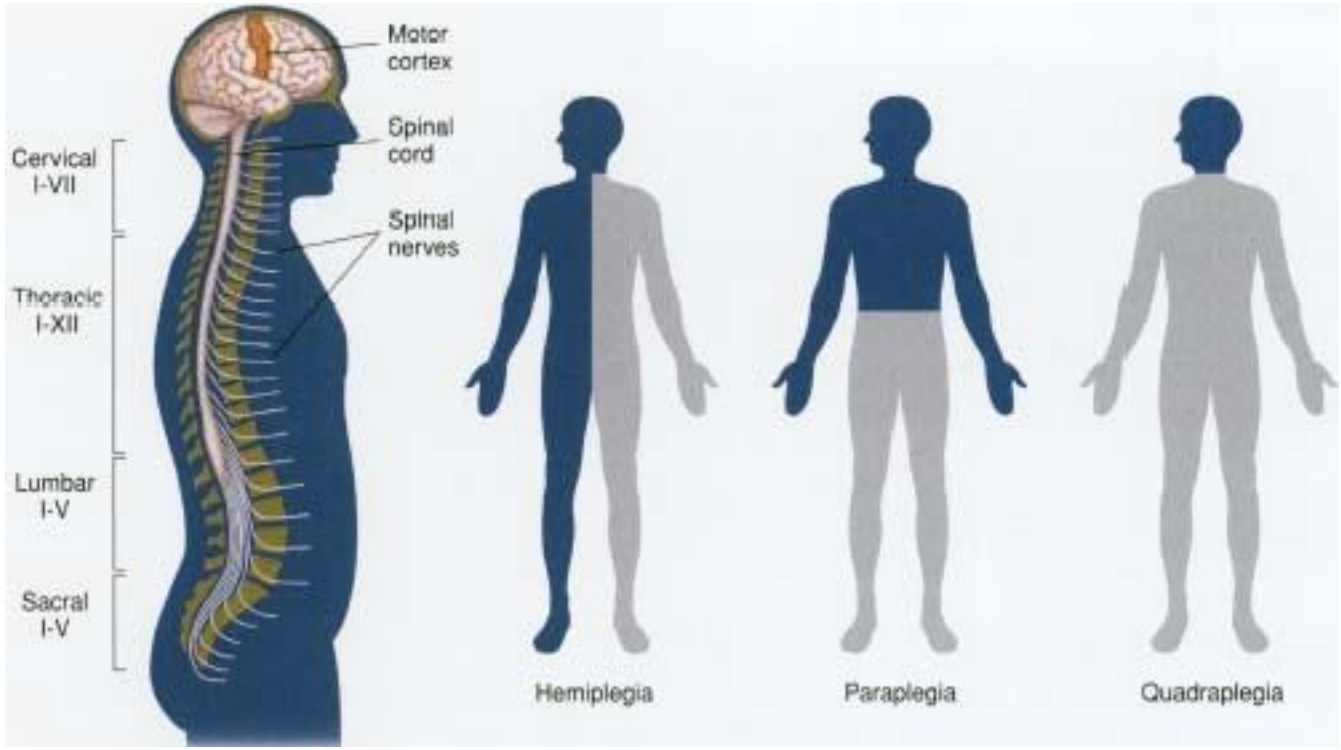
- Primary injury triggers secondary inflammatory injury:
  - Oxygen free radicals
  - Calcium overload-mediated cell changes (excessive glutamate release by neurons, activation of damaging enzymes in neurons)
  - Glutamate excitotoxicity
  - Autoregulation of capillary bed perfusion is lost leading to ischemia and further cell damage and inflammation.
- High dose **corticosteroids** (methylprednisolone) may be given during the first 24 hours after injury to minimize secondary injury.

Spinal Cord Injury

Clinical Manifestations

- **Spinal Shock**
  - Complete lack of function (motor, sensory and reflexive) below the site of injury; usually lasts **less than 24 hours**.
  - Flaccid paralysis
  - Loss of bowel and bladder control
  - Spinal shock is not “true” shock. Hypotension isn’t present.
- **Return of Reflex Function**
  - Signals end of spinal shock
  - Spastic paraplegia or spastic hemiplegia develops signaling early recovery of upper motor neurons. Reflexes are exaggerated=**hyperreflexia**
  - Bowel and bladder may regain reflexive function.
- **Return of Full Function**
  - Difficult to predict, may or may not occur; takes time

Spinal Cord Injury

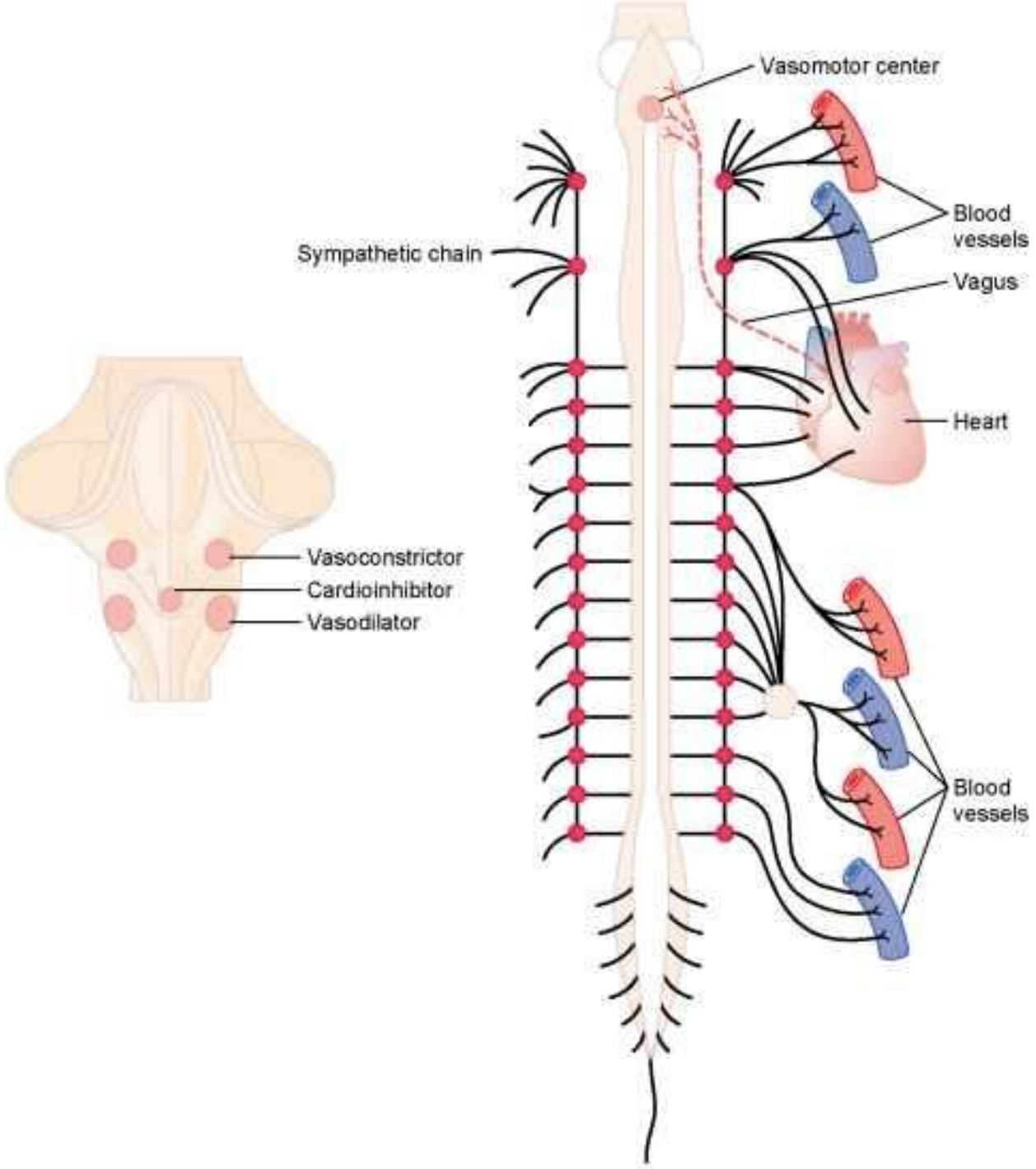


Spinal Cord Injury

Complications of Spinal Cord Injury

- **Neurogenic Shock**
  - If the spinal cord injury is above T6 a severe **drop in blood pressure (true shock)** may add still further to the ischemia.
  - Neurogenic shock is due to loss of **sympathetic nervous system** activity below the level of injury.
  - Sympathetic spinal nerve fibers that constrict blood vessel wall smooth muscle (in all areas except the head) to maintain proper blood pressure exit the spinal cord below T6. Without their function (without **"sympathetic tone"**) blood vessels dilate and blood pressure drops causing **hypotension.**
  - **Bradycardia** (a slow heart rate) and the **inability to sweat** below the level of injury may also occur due to loss of sympathetic nerve fiber function.
  - **Intensive care** is required to maintain blood pressure.

Most of the body's blood vessels are served by **sympathetic** fibers in spinal nerves that exit the spinal cord below T6.
Most blood vessel walls do not receive any **parasympathetic** innervation.

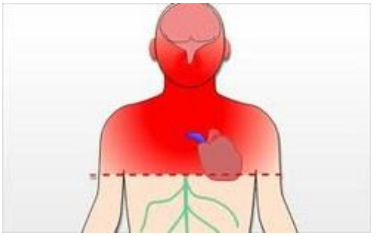


Spinal Cord Injury

Complications of Neurogenic Shock

- **Autonomic Dysreflexia**
  - May occur if injury is above T6 and **AFTER spinal shock** has resolved
  - Initiated by activation of visceral or cutaneous pain receptors below injury level
  - A full bladder or constipation are common triggers.
- Afferent pain impulses travel up the spinal cord as far as the site of injury to cause a **sympathetic response to acute pain**.
- **Reflexive** efferent sympathetic impulses in spinal nerves result in **vasoconstriction** in vessels served by spinal cord regions below the site of injury (This affects most of the body's blood vessels.).
- The baroreceptors (pressure receptors)in the aorta and carotid arteries are stimulated by the resulting increase in blood pressure. (Baroreceptors are innervated by cranial nerves, not spinal nerves.)
- The homeostatic **baroreceptor response** is triggered—the body attempts to lower BP.

Spinal Cord Injury



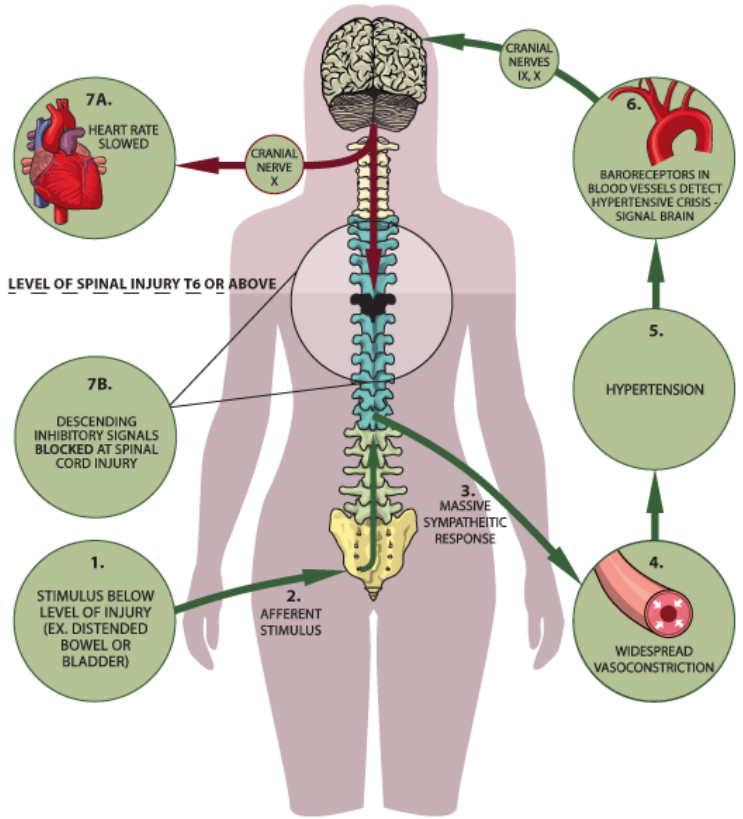
Complications of Neurogenic Shock

- **Autonomic Dysreflexia cont.**
- The baroreceptors (BP receptors) are located in the aortic arch and the carotid arteries. Cranial nerves IX and X carry afferent impulses from there to the cardiac and vasomotor centers in the medulla of the brain stem, so they aren't affected by the spinal cord injury.
  - Efferent parasympathetic fibers in cranial nerves X (**vagus)** cause **bradycardia** (slow heart rate).
  - Efferent fibers in **spinal nerves** located **above the level** of the injury cause **vasodilation and flushing** by decreasing sympathetic impulses to vascular smooth muscle.
  - **Systemic blood pressure remains** high because most body blood vessels remain constricted due to the abnormal reflex activity.
- **Intracranial hemorrhage** may occur due to the high BP.
- Painful stimuli must be removed.
- **Adrenergic receptor blockers** help by reducing the binding of norepinephrine to its receptors.

Spinal Cord Injury

Summary: Mechanism of Autonomic Dysreflexia

1. Afferent pain impulses climb to T5/T6.
2. Sympathetic spinal reflexes produce efferent impulses that cause vasoconstriction in blood vessels that receive spinal output **below** the level of the injury.
3. The resultant **high systemic blood pressure** stimulates the baroreceptors. Baroreceptors send impulses to the vasomotor and cardiac centers in the medulla. The body tries, unsuccessfully, to lower the BP by slowing the heart rate and dilating blood vessels.
4. Parasympathetic impulses from the medulla slow the heart rate. Vasodilation occurs, but only in vessels that receive spinal output above the injury site. Flushing occurs in the skin above the injury. BP stays high!



Lecture 7D
(Disorders of the Special Senses)
Equilibrium Disorders
Hearing Disorders
Visual Disorders

Equilibrium

The Inner Ear: Special Sensory Organs of Equilibrium

The **semicircular canals** contain sensory organs (cristae ampullares) that detect changes in **angular** motion.

The **vestibule** contains sensory organs (maculae) in two structures (utricle and saccule) that detect changes in **linear** motion.

Sensory impulses from the sensory organs of equilibrium are carried in the vestibular portion of **cranial nerve VIII (vestibulocochlear) to the vestibular nuclei** in the brain stem.

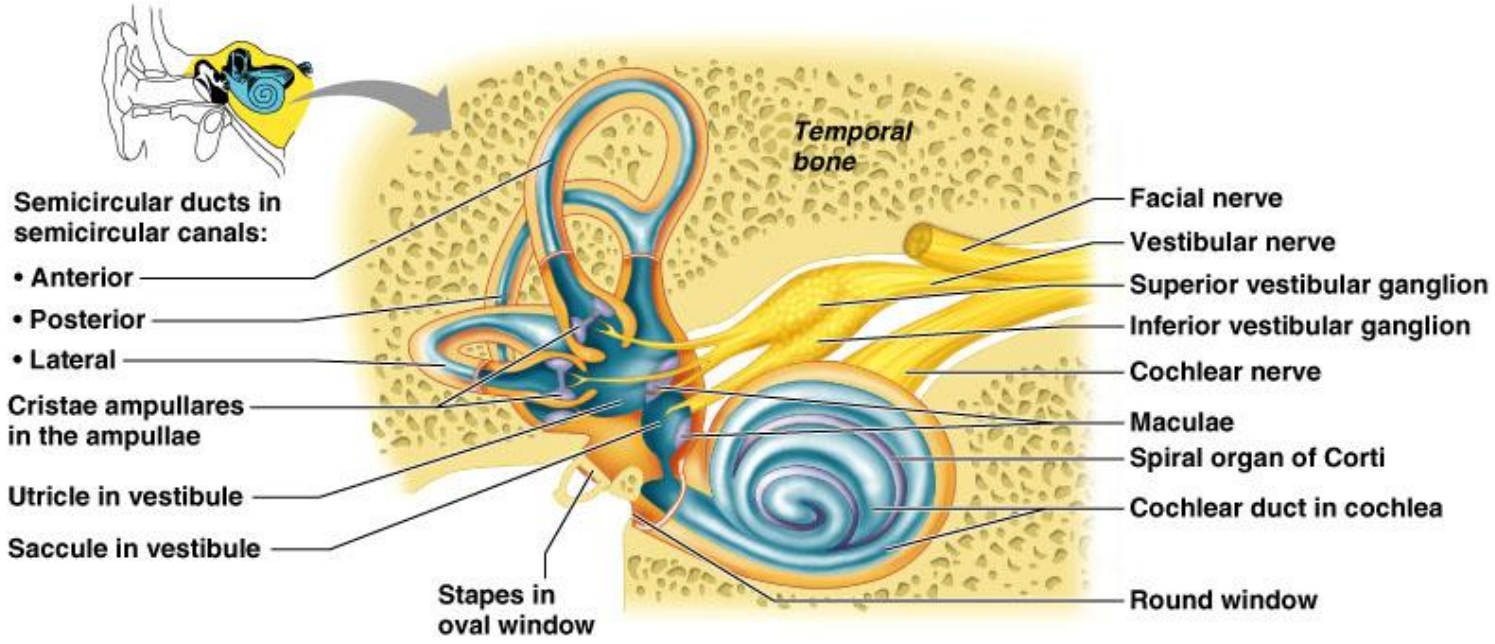
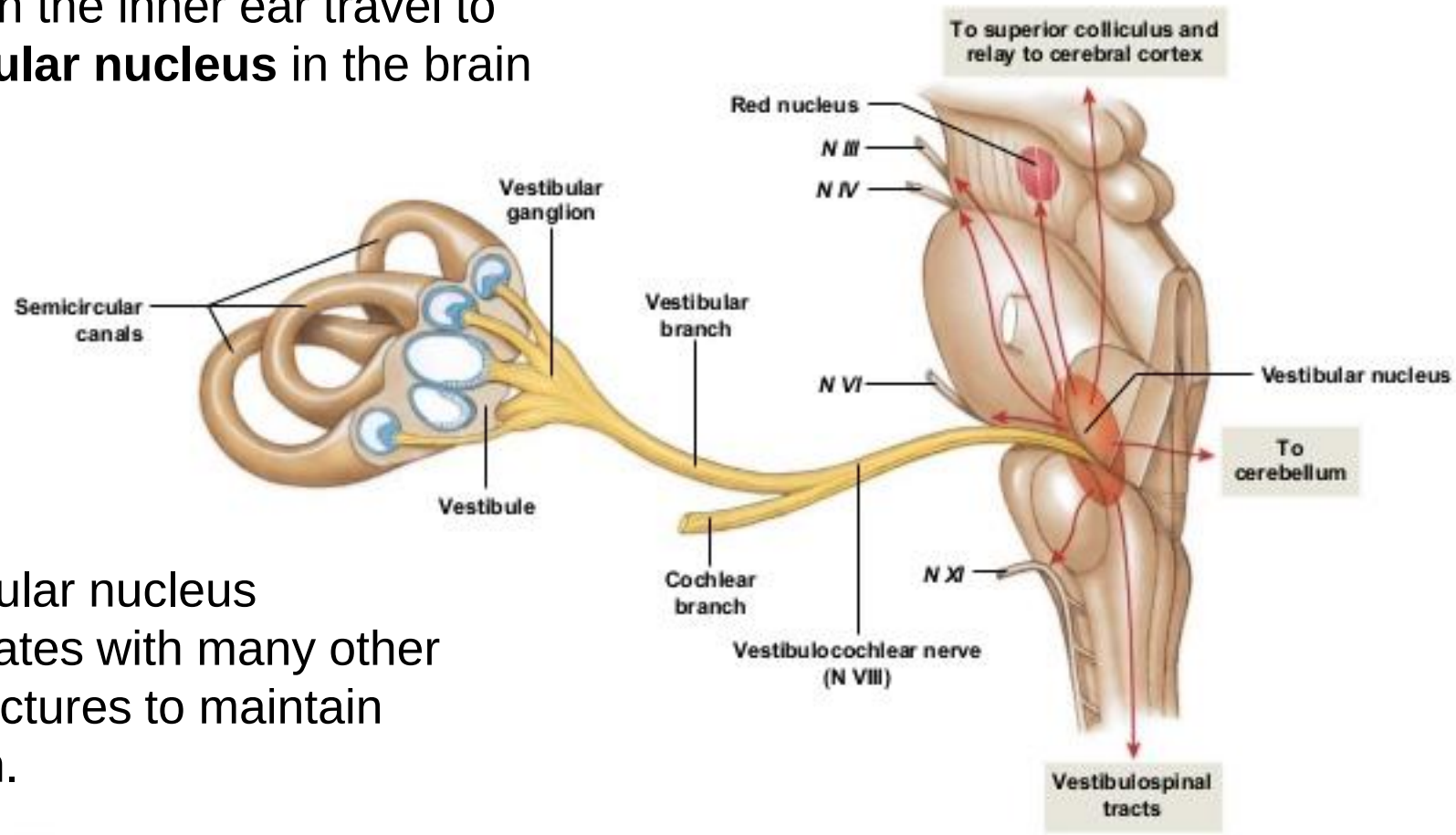


Figure 18.16 Neural Pathways for Equilibrium Sensations

Impulses from the equilibrium receptors in the inner ear travel to the **vestibular nucleus** in the brain stem.



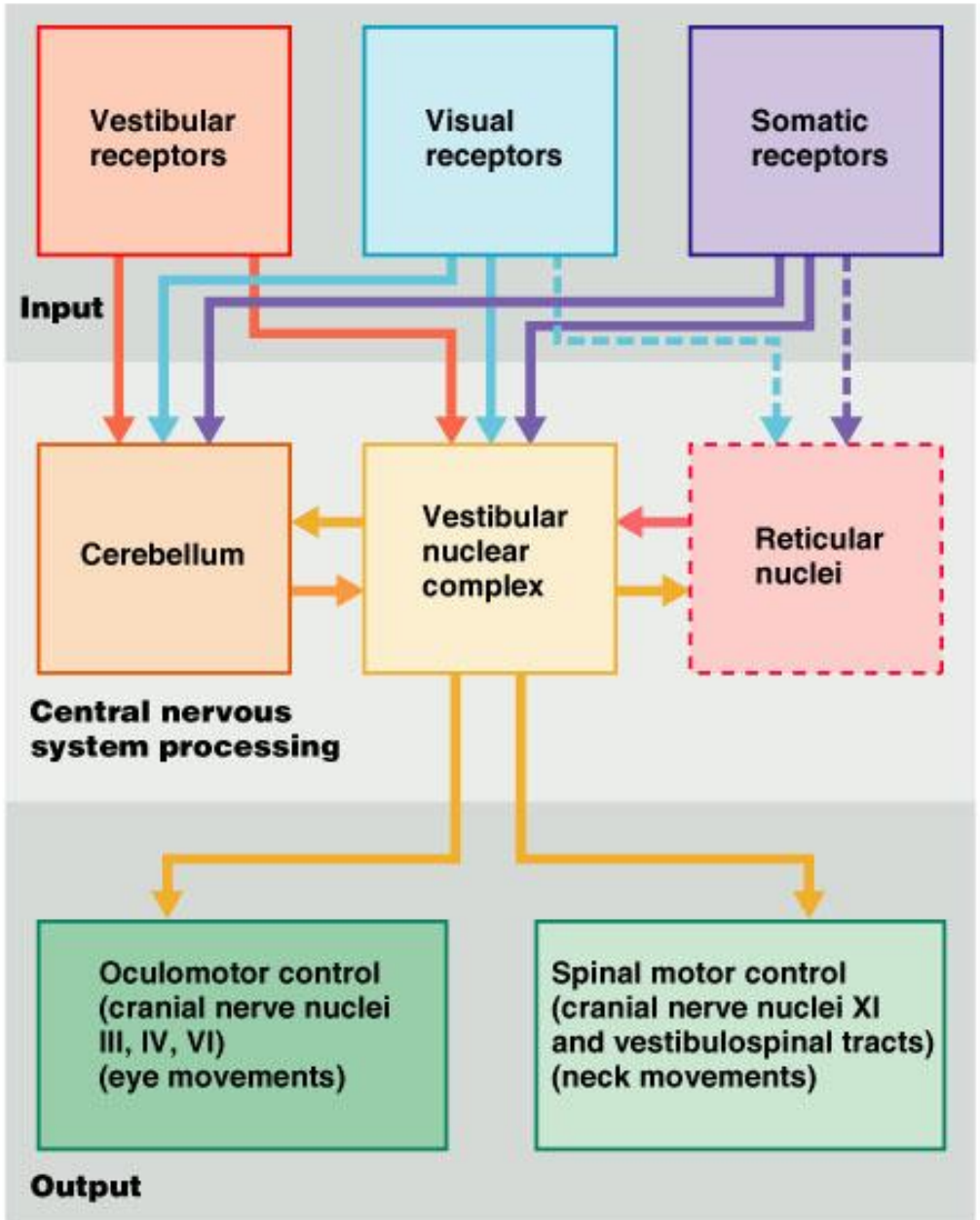
The vestibular nucleus communicates with many other neural structures to maintain equilibrium.

Equilibrium

Maintaining Equilibrium

- The **vestibular nuclei** play a central role in equilibrium processing. These nuclei receive **sensory input** from the inner ear, the eyes and proprioceptors in muscles and joints throughout the body.
- **CNS processing** involves **two-way** communication between the **vestibular nuclei in the brainstem** and both the **cerebellum** and **reticular nuclei in the thalamus** .
- **Motor output from the vestibular nuclei** is aimed at making adjustments in head and eye position in order to maintain a visual focus. The skeletal muscles receiving the motor output are the extrinsic eye muscles via cranial nerves III, IV and V (oculomotor, abducens, trochlear) and neck muscles via the accessory nerve (XI) and via motor fibers from the **vestibulospinal tract**.

The Equilibrium Pathway

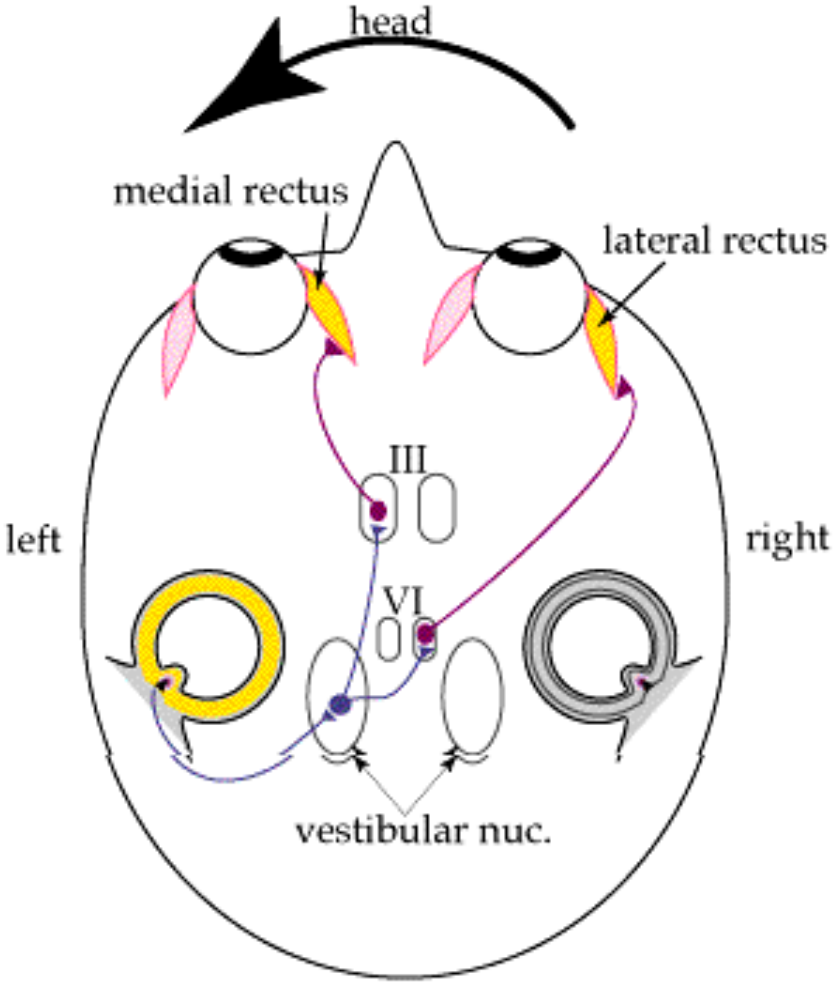


Equilibrium

Remember the Oculovestibular Reflex?

If you move your head to the left, you will excite the left horizontal semicircular canal, and inhibit the right horizontal semicircular canal.

To keep your eyes fixed on a stationary point while turning your head to the left, you must contract the right lateral rectus muscle (via the abducens nerve-IV) and the left medial rectus muscle (via the oculomotor nerve-III) so your **eyes will move to the right.**



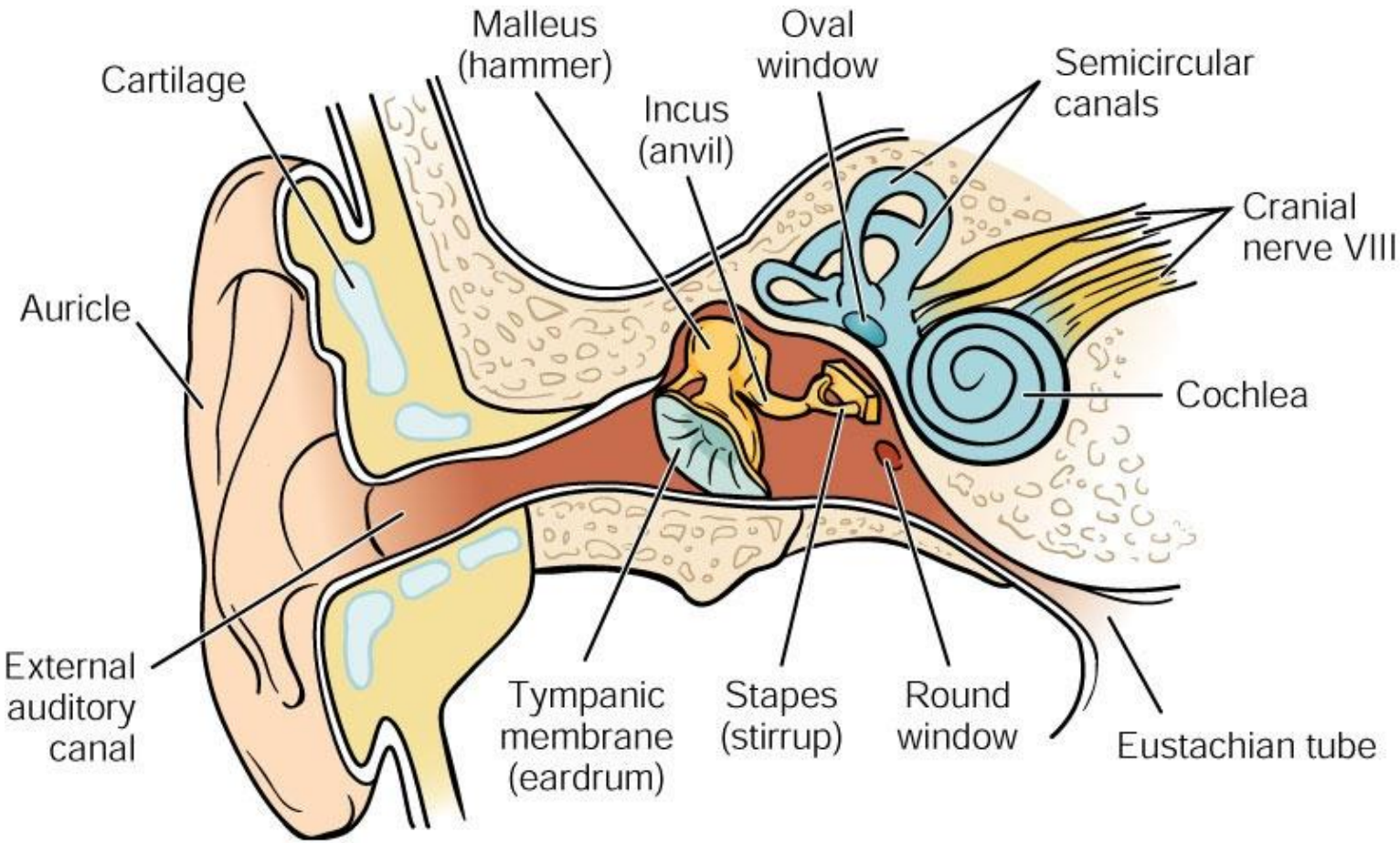
Equilibrium Disorders: Vertigo

- **Vertigo** is a sensation of movement when no movement is occurring or an exaggerated sense of movement.
- It may be accompanied by nausea, vomiting, pallor, sweating or nystagmus.
- Vertigo may be caused by abnormalities in the inner ear or anywhere along the neural pathway that controls equilibrium.
- Alcohol, anticonvulsants and sedatives may cause vertigo.
- It is a symptom in diseases such as MS and Meniere's disease and also in migraine and psychogenic disorders.

Hearing

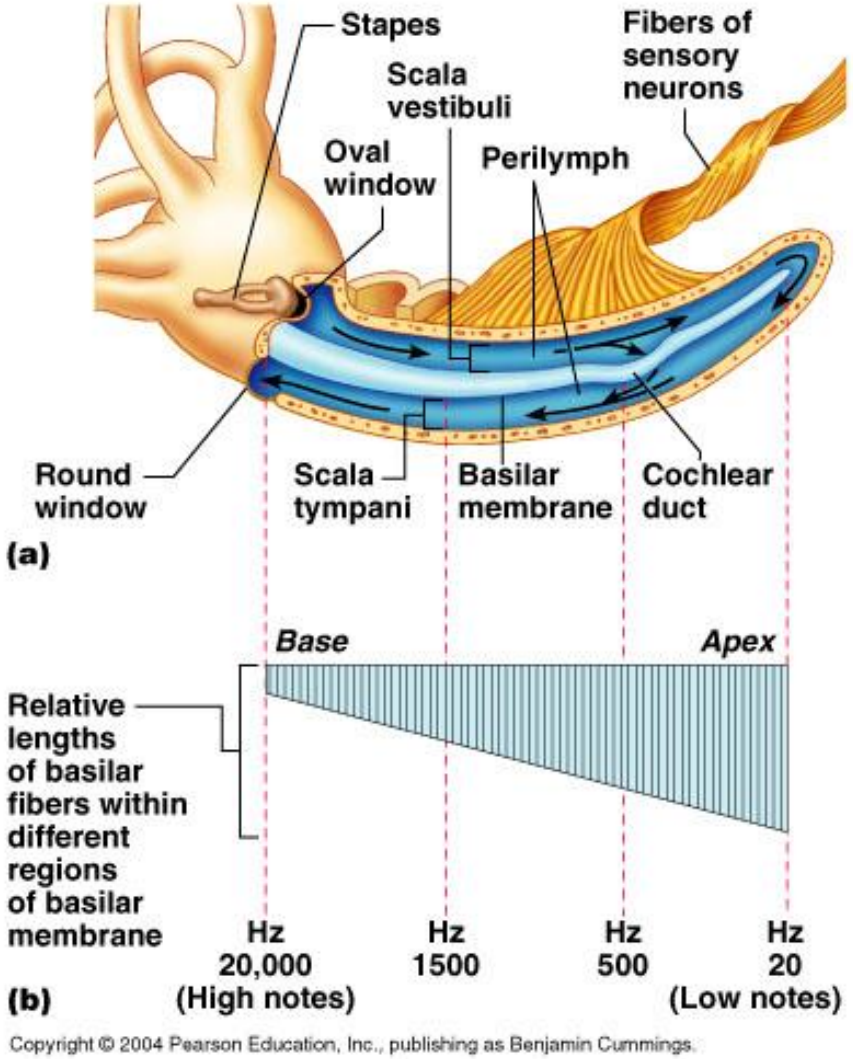
Hearing involves all three regions of the ear:

external, middle, inner



Hearing

Sound wave vibrations are transmitted from the stapes to the oval window to the perilymph of the scala vestibuli of the cochlea, then through the endolymph of the cochlear duct to the basilar membrane causing **hair cells in the Spiral Organ of Corti in the cochlear duct to depolarize.** Sensory impulses from the hair cells are carried in the cochlear portion of cranial nerve VIII (vestibulocochlear) to the cortex of the temporal lobe.



Conductive Hearing Loss

Etiology

- Sound waves cannot reach the cochlea.
- Four Common Causes:

1. **Obstruction**-cerumen (earwax) impaction or foreign body occlusion (stones, peas, beans, pieces of wood or paper, insects, usually placed there by kids).
2. **Mass Loading**-middle ear effusion due to infection (otitis media) causes pressure on the tympanic membrane.
3. **Stiffness Effect**- due to otosclerosis, resorption of bone followed by formation of new sponge-like bone on and around the ossicles of the middle ear.
    - Otosclerosis of the footplate of the stapes (**stapedial fixation**) decreases sound transmission to the cochlea
    - If otosclerotic bone enters the cochlea, permanent sensorineural hearing loss may occur.
    - Family history, autoimmunity, infection increase risk.
4. **Discontinuity-**disruption of the connection between any two ear ossicles

Conductive Hearing Loss

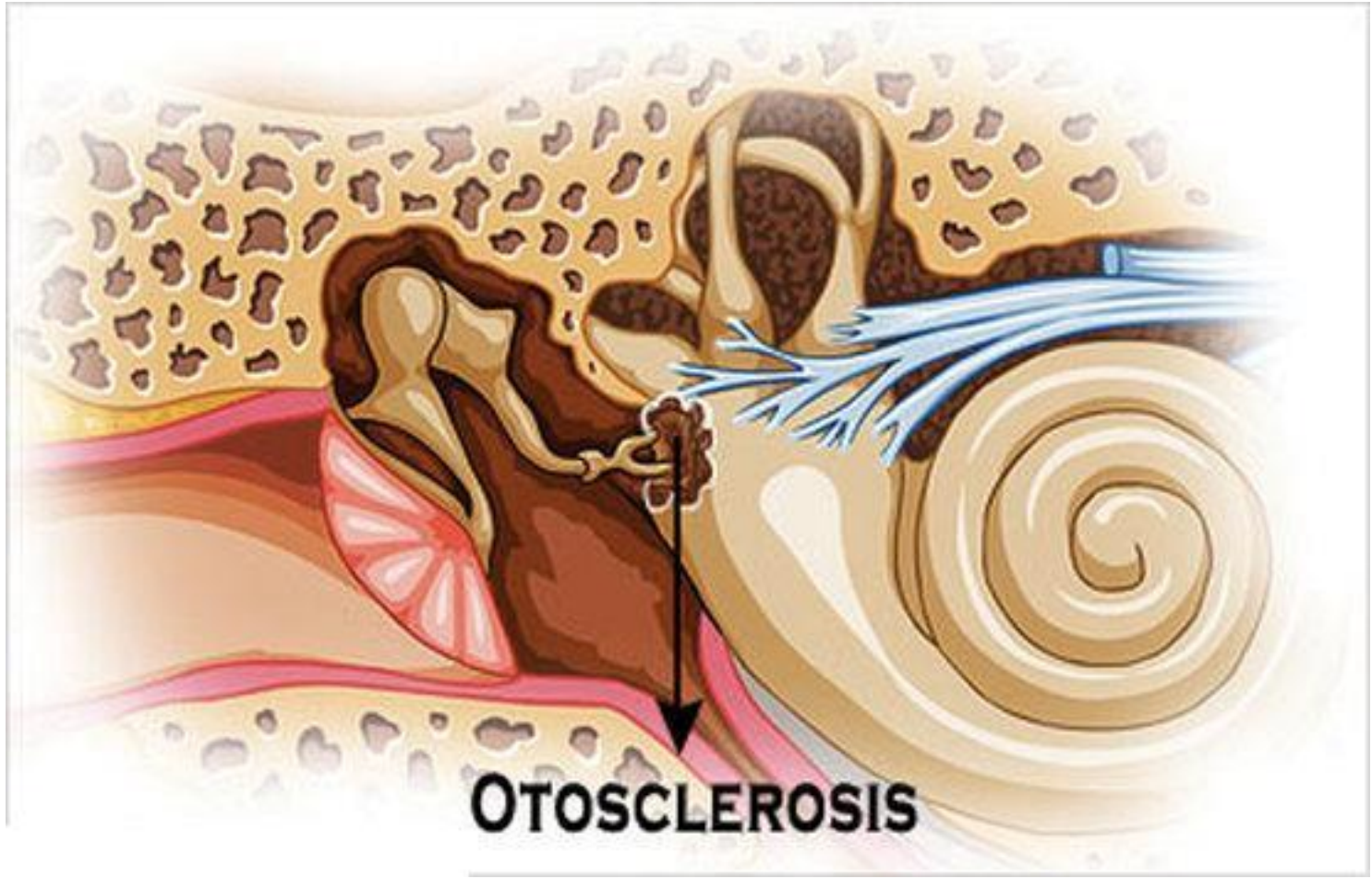
Clinical Manifestations

- Patients **hear best in a noisy environment** as opposed to a quiet one.
- Often unilateral unless the cause is otosclerosis. Otosclerosis is usually bilateral.
- Tinnitus (“ringing in the ears”) is often present.
- Speech discrimination is preserved unless the cochlea is involved.

Treatment

- Removal of cerumen or foreign body
- For otosclerosis—surgical removal of the stapes and replacement with a prosthesis.

Conductive Hearing Loss

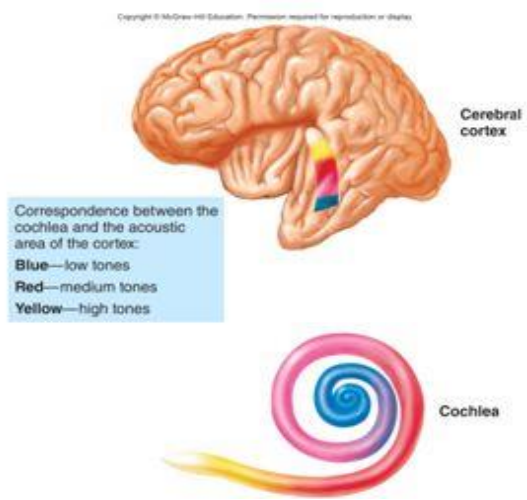


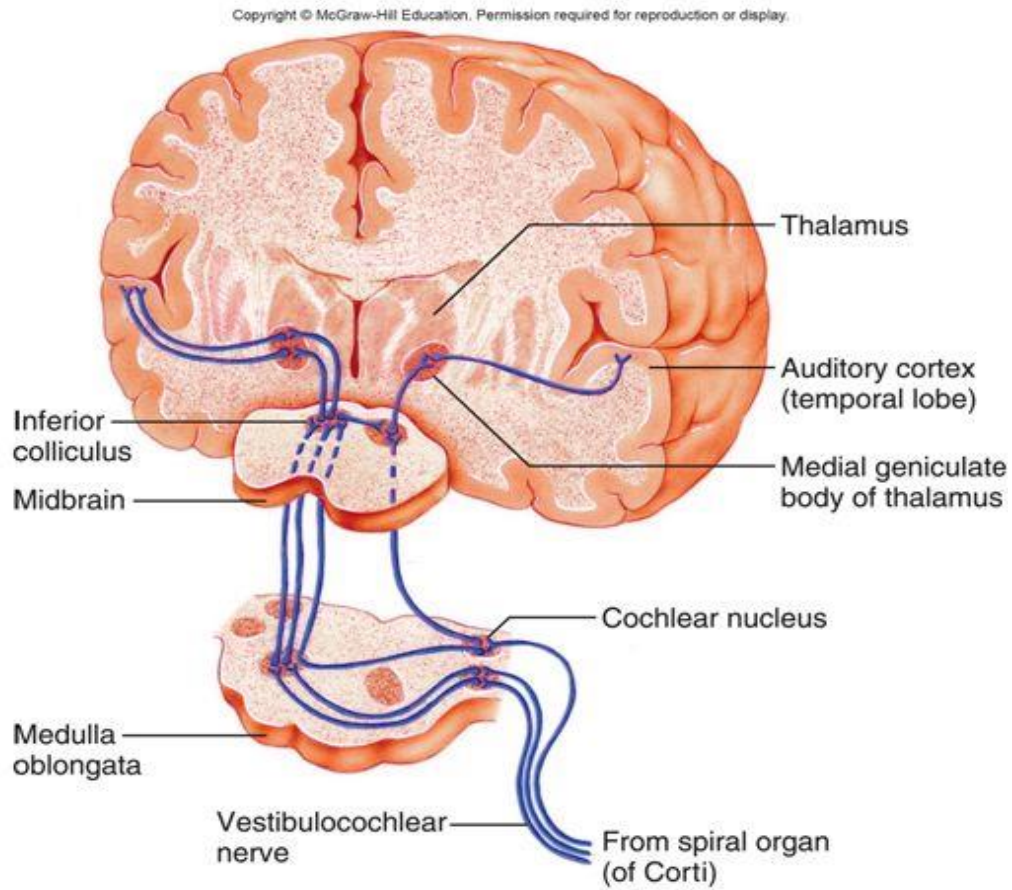
Neural Pathways for Hearing

Sensory neurons from the vc nerve synapse in the medulla oblongata

neurons extend from medulla to midbrain

neurons extend from midbrain to auditory cortex of temporal lobe





NOTE that MOST fibers, but not all, decussate to the contralateral cerebral cortex of the temporal lobe.

Sensorineural Hearing Loss

Etiology

- Damage to the **hair cells of the cochlea or the neural pathway of audition** prevents the neural impulses required for hearing from being generated or from being transmitted to the cortex for perception.
- Common Causes:
  - **Ototoxic drugs** damage hair cells of the cochlea.
    - Antibiotics, high dose aspirin, anti-malarial drugs, cancer chemotherapy.
    - Effect may be unilateral, tinnitus is common.
  - **Trauma due to loud sound**: firearms, personal stereo systems, power tools, occupations as firefighting, construction, agriculture, manufacturing, transportation, and the military.
    - Hearing loss due to loud sound is usually **bilateral.**
    - Inability to hear **higher** frequencies (speech) occurs first=*threshold shift.*

Sensorineural Hearing Loss

- When the noise source is removed normal hearing will return if no permanent damage has been done.
- If permanent damage has occurred the *threshold shift* remains in place.
- Loudness is measured in decibels (dB).
  - Conversation at 3 feet=55 dB
  - Lawn mowers and motorcycles=90 dB
  - Rock concerts=140 dB
- Sounds **exceeding 85 dB** are considered injurious.

- **Head Trauma**
  - Especially if bleeding from the ears or temporal bone fracture occurs.
- **Infection**
  - **Mumps**
  - **Measles (rubeola or rubella)**

Sensorineural Hearing Loss

Clinical Manifestations

- Inability to discriminate the speech of others, especially in a noisy environment.
- Patients usually complain more about **tinnitus** (the perception of sound in the absence of sound—ringing in the ears) than hearing loss.
- Sensorineural hearing loss is often permanent, at least to some degree.

Treatment

- Prevention is the best treatment.
- Hearing aids may help.

Presbycusis

- Presbycusis is hearing loss associated with the **aging** process.
- It has a gradual bilateral onset.
- **High-pitched loss first** (speech discrimination) and middle and low tones may be involved in time.
- Four categories have been theorized.
  - **Sensory**-degeneration of cochlear hair cells (receptor cells) and supporting cells is the most common cause.
  - **Neural**-loss of neurons between the cochlea and the CNS or within the CNS
  - **Metabolic**-atrophy of the cochlear wall
  - **Mechanical**-due to changes in middle ear structures
- Hearing aids and life style adjustments can dramatically improve the quality of life.

Meniere Disease

Etiology and Pathogenesis

- Accumulation of **endolymph** (the fluid in the membranous labyrinth of all three areas of the **inner** ear), usually unilateral. Both **equilibrium and hearing** are usually affected.
- The **scala media (aka cochlear duct)** of the cochlea may rupture causing the cochlea to degenerate.
- Linked to allergy, infection, head trauma, metabolic derangements, and chronic stress.
- Onset is usually in the fourth decade.

Clinical Manifestations

- Acute attacks of tinnitus, sensorineural hearing loss, vertigo, nausea, vomiting
- Hearing loss is usually in the **low** tones.
- Nystagmus may occur during acute attacks of vertigo.

Meniere Disease

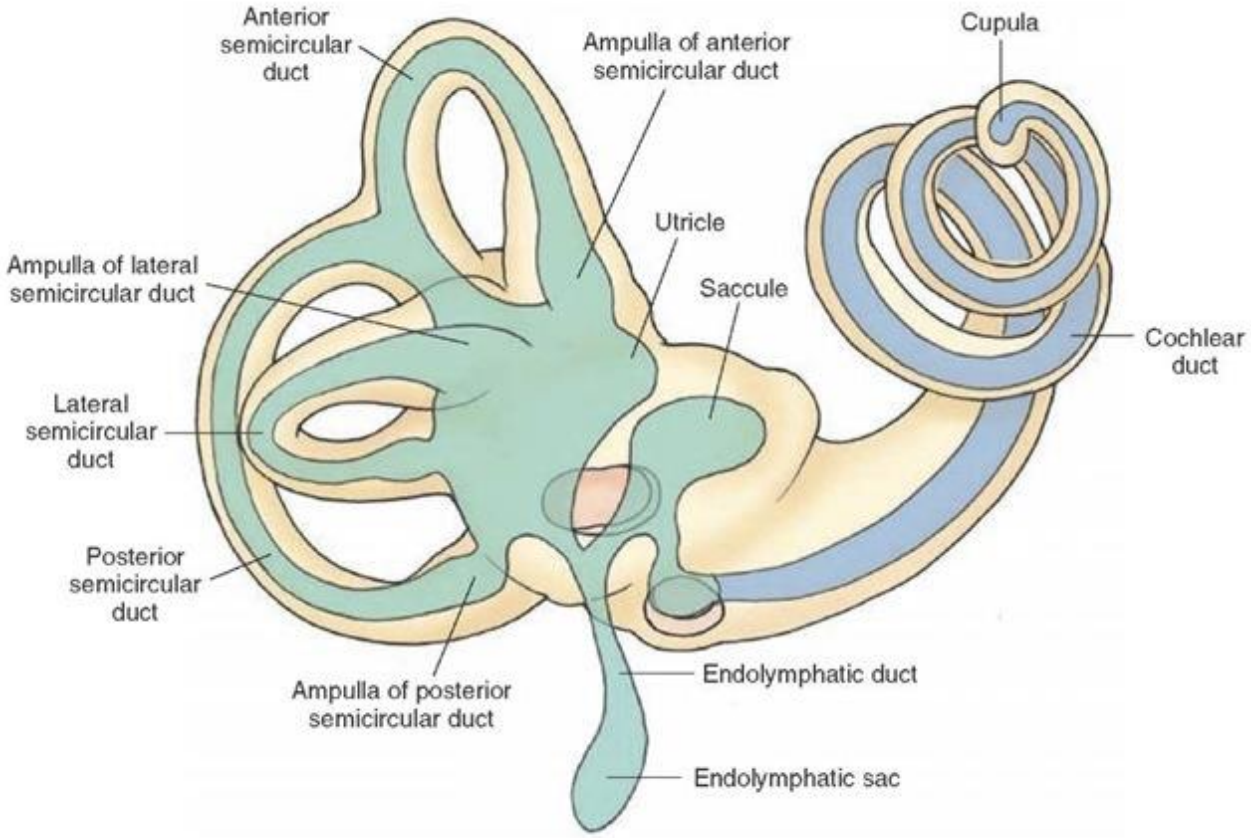
Inner Ear Anatomy Notes:

*The areas shown in green and blue (membranous labyrinth) contain **endolymph.**

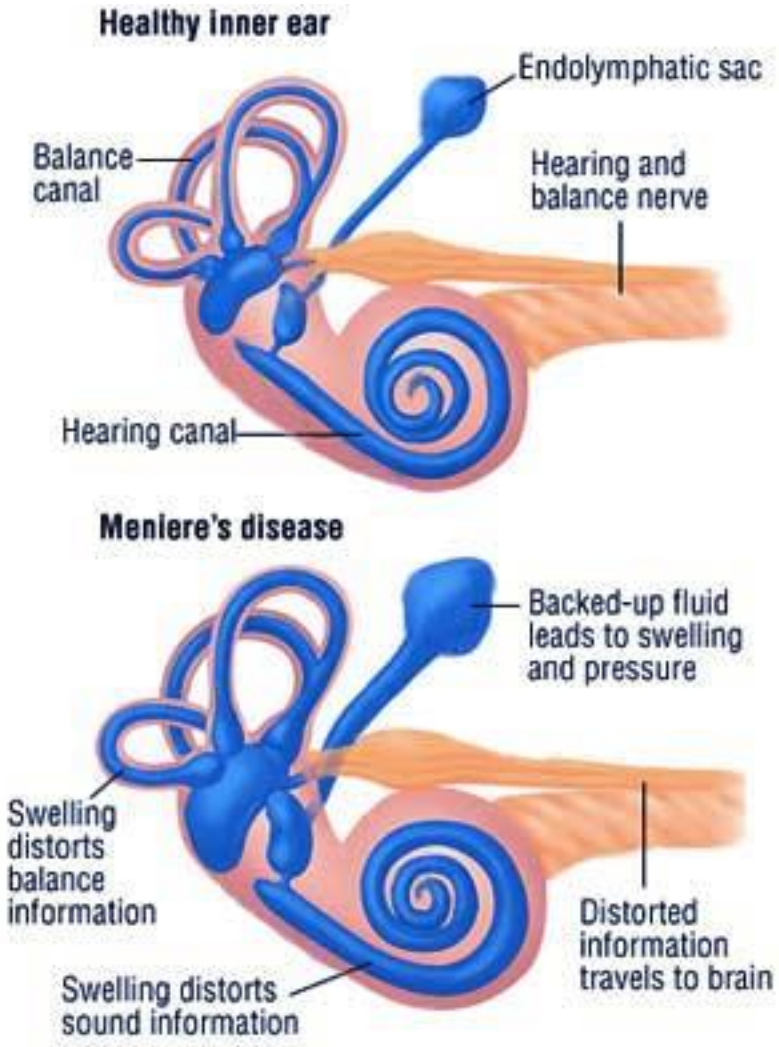
*The tan area contains **perilymph.**

*The utricle and saccule are the two parts of the **vestibule.**

*The **cochlear duct** is also known as the scala media.



Meniere Disease



Meniere Disease

Diagnosis

- Aided by electrophysiologic testing (auditory brainstem responses and electrocochleography) and audiometry.
- Caloric testing (irrigation with warm or cold water) often reveals **loss of a nystagmus response** on the affected side.

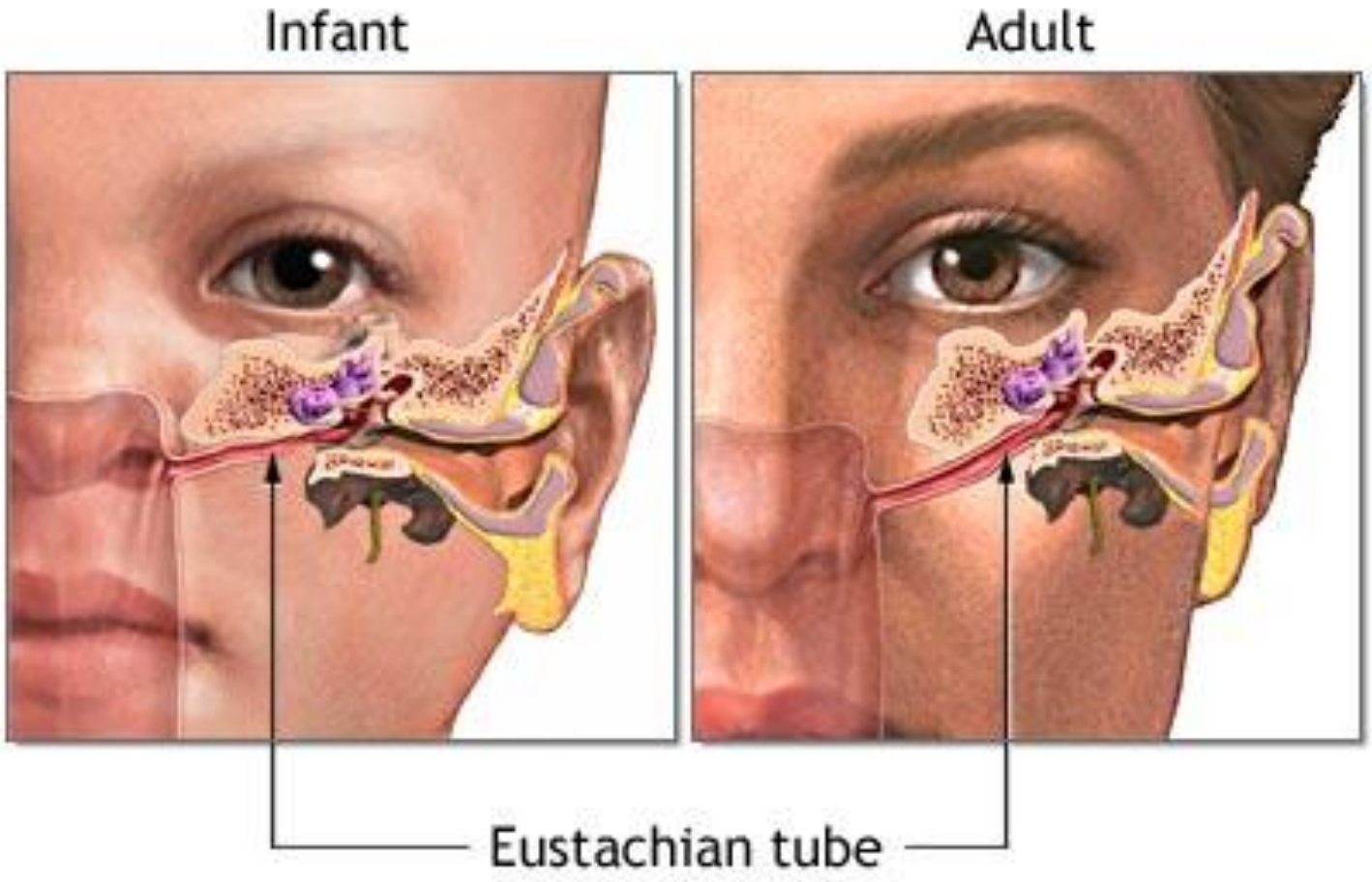
Treatment

- Antiemetics, anticholinergics, bed rest, and sedation.
- Between attacks diuretics and a low-sodium diet may help to reduce endolymph volume.
- Cessation of smoking and caffeine use are recommended.
- Surgical interventions include **shunt placement** to drain excess endolymph, ablation of parts of the cranial nerve, and destruction of the membranous labyrinth of the affected ear.

Otitis Media

- Infection of the **middle ear:**
- It is the most common reason for children to visit a medical facility. .
- A child's eustachian tube is short and horizontally positioned and thus
  - upper respiratory tract infections are more likely to access the middle ear.
  - inflammatory fluids in the middle ear are more prone to collection and less prone to drainage.
- As the skull develops, the eustachian tube develops a more vertical angle, so otitis media is much less common in adults.
- Risk factors: pacifier use, second-hand smoke, propped bottles, daycare
- Two forms: acute and chronic

Otitis Media



Acute Otitis Media

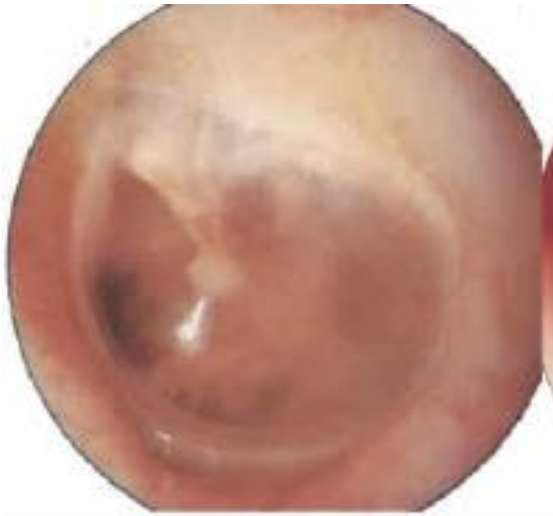
Etiology and Clinical Manifestations

- Sudden onset of ear pain with upper respiratory tract infection, short duration; usually bacterial, but may be viral
- Otoscopic exam shows reddened tympanic membrane with poor mobility and bulging.
- Tympanic membrane rupture is possible.

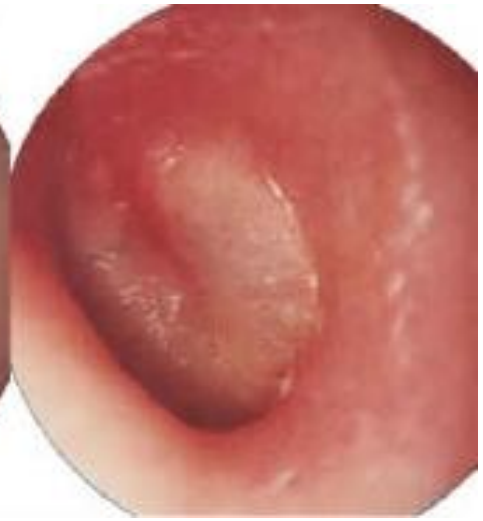
Treatment

- 14-day course of antibiotics
- Follow-up exam is important.
- **Myringotomy**, surgical placement of tubes through the tympanic membrane, is used to treat recurrent, acute otitis media.
- Complications-mastoiditis, meningitis, osteomyelitis, facial paralysis.

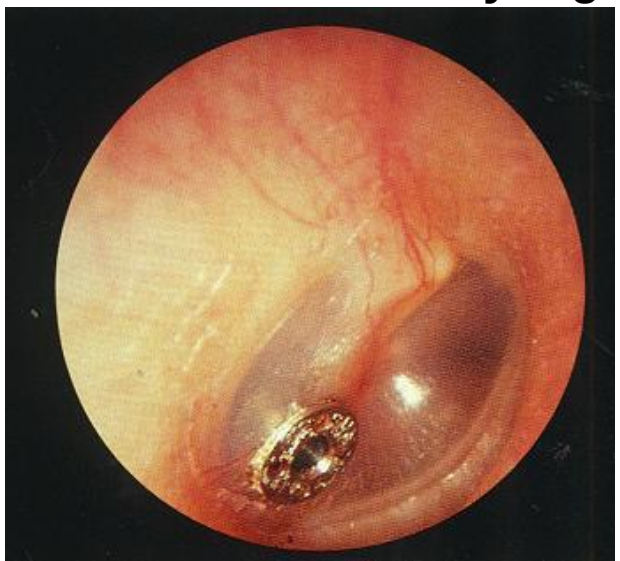
normal tympanic membrane



otitis media tympanic membrane

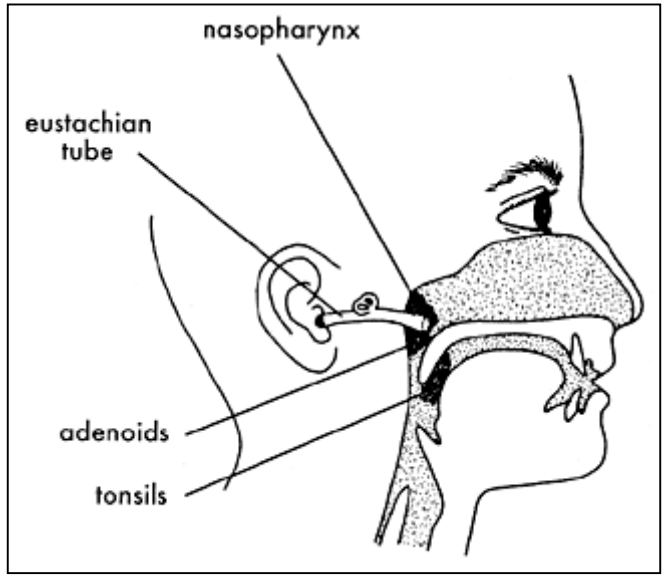


tympanic membrane with myringotomy tube

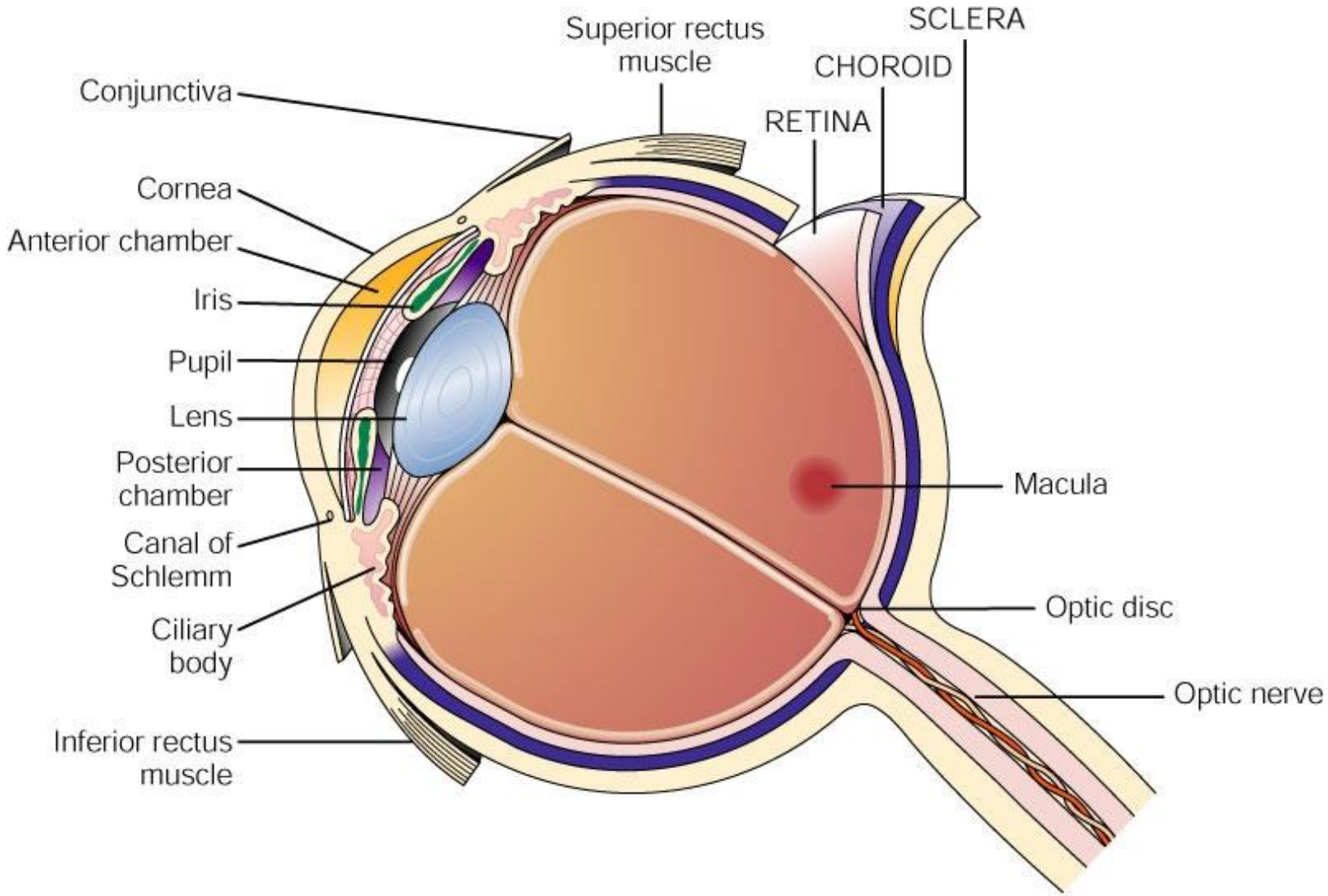


Chronic Otitis Media

- Infection of **more than 12 weeks with irreversible damage**
- The hallmark of chronic otitis media is purulent discharge.
- Pain is **uncommon** and conductive hearing loss may occur.
- Surgical interventions: myringotomy, removal of inflamed tonsils that may be blocking the outlet of the eustachian tube.



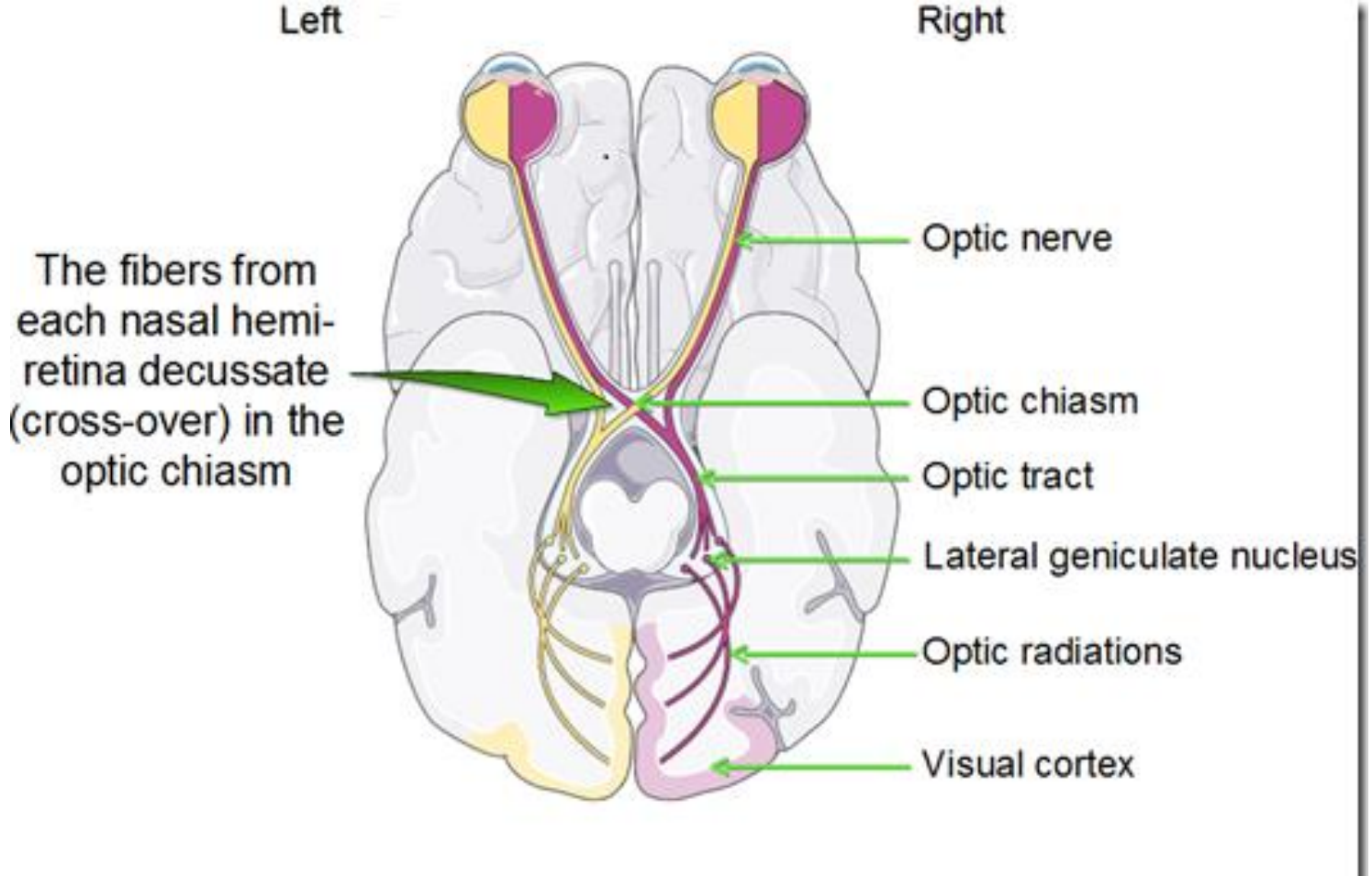
The Eye



The Eye

- Light is the stimulus for sight.
- The light receptors of the eye (**rods and cones**) are part of the **retina**, the innermost layer of the lateral and posterior eye. The **macula** of the retina is the area of **highest visual acuity**. It contains a high concentration of cones.
- Before reaching the retina light passes through several **transparent** structures/substances: cornea, anterior chamber (contains aqueous humor), posterior chamber (contains aqueous humor), lens, vitreous humor.
- The iris is a smooth muscle structure that partially separates the anterior and posterior chambers. It has a central opening, the pupil.
- Impulses from the rods and cones are carried in the **optic nerve** and **optic tracts** to the **lateral geniculate nucleus** of the **thalamus** and then to the **cerebral cortex** of the **occipital lobe.**

The retina is divided in half functionally: the **nasal** half of each retina is located closest to the nose, while the **temporal** half of each retina is located closest to the temporal bone. Note that the optic nerve fibers from the **nasal half** of each retina decussate at the **optic chiasm**. Each occipital cortex receives input from both the left eye and the right eye.



Visual Disorders

- Visual Field Blindness
- Refraction Disorders
- Strabismus
- Cataracts
- Diabetic Retinopathy
- Macular Degeneration
- Retinal Detachment
- Glaucoma

Visual Field Blindness

Total Blindness-complete lack of form or light perception; NLP (no light perception)

Legal Blindness (3 Etiologies)

- NLP
- Visual acuity is 20/200 or worse (20/20 is normal.) in the best eye with the best correction possible.
- Visual field is reduced to 20° or less.

Etiology and Pathogenesis

•Loss of all or part of a visual field or fields is due to injury somewhere in the neural pathway of vision (retina to occipital cortex).

•May be due to tumors, vascular lesions, stroke, or demyelinating lesions in the neural pathway for vision.

Visual Field Blindness

Etiology and Pathogenesis

- **Monocular visual field loss** is due to damage to the retina or optic nerve.
- **Bilateral visual field** loss is due to damage at or distal to the optic chiasma
  - **Bitemporal hemianopsia**
    - Loss of vision in the **temporal fields** of both eyes
    - Due to lesions in the **optic chiasma** (often due to pituitary tumors)
  - **Homonymous hemianopsia**
    - Loss of vision in the temporal field of one eye and the nasal field of the other eye.
    - Due to lesions **beyond the optic chiasma**
    - Common in **stroke**

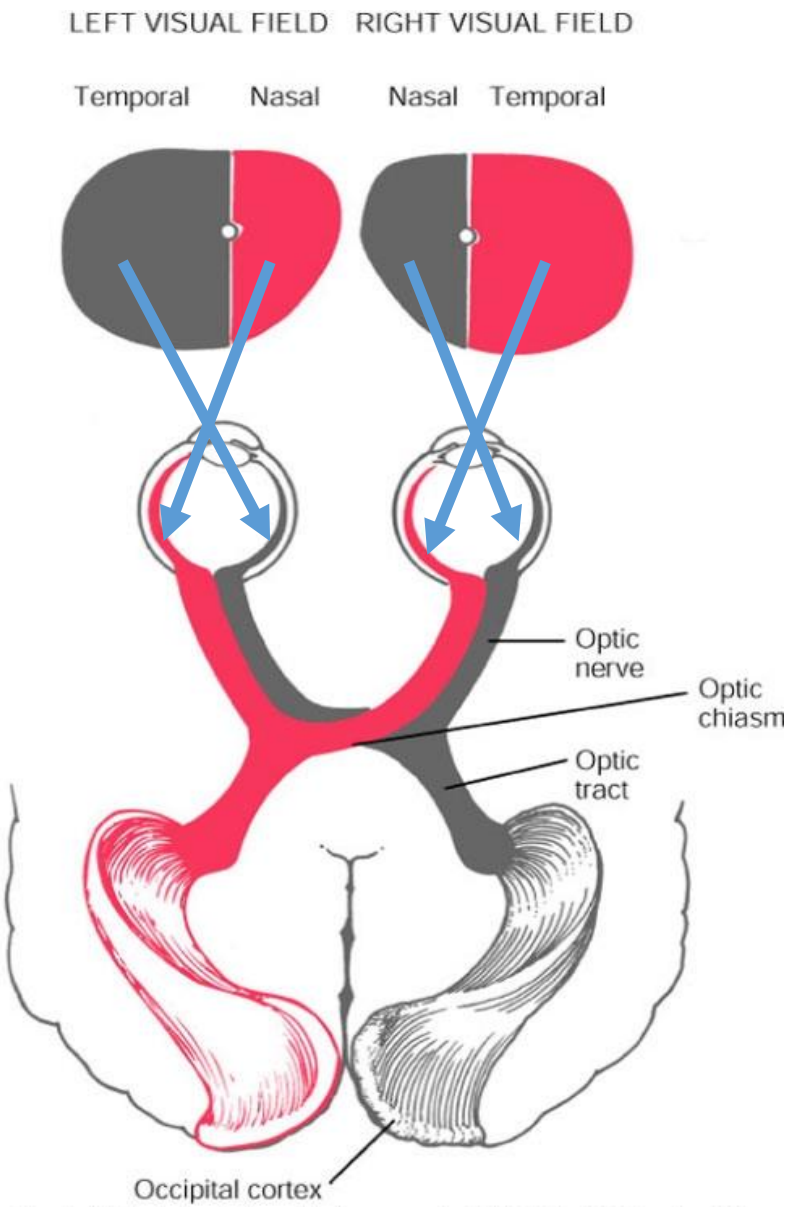
Treatment

- Management of the underlying cause
- Adaptation of the patient's environment to the visual loss

Due to the curvatures of the cornea, the lens the retina:

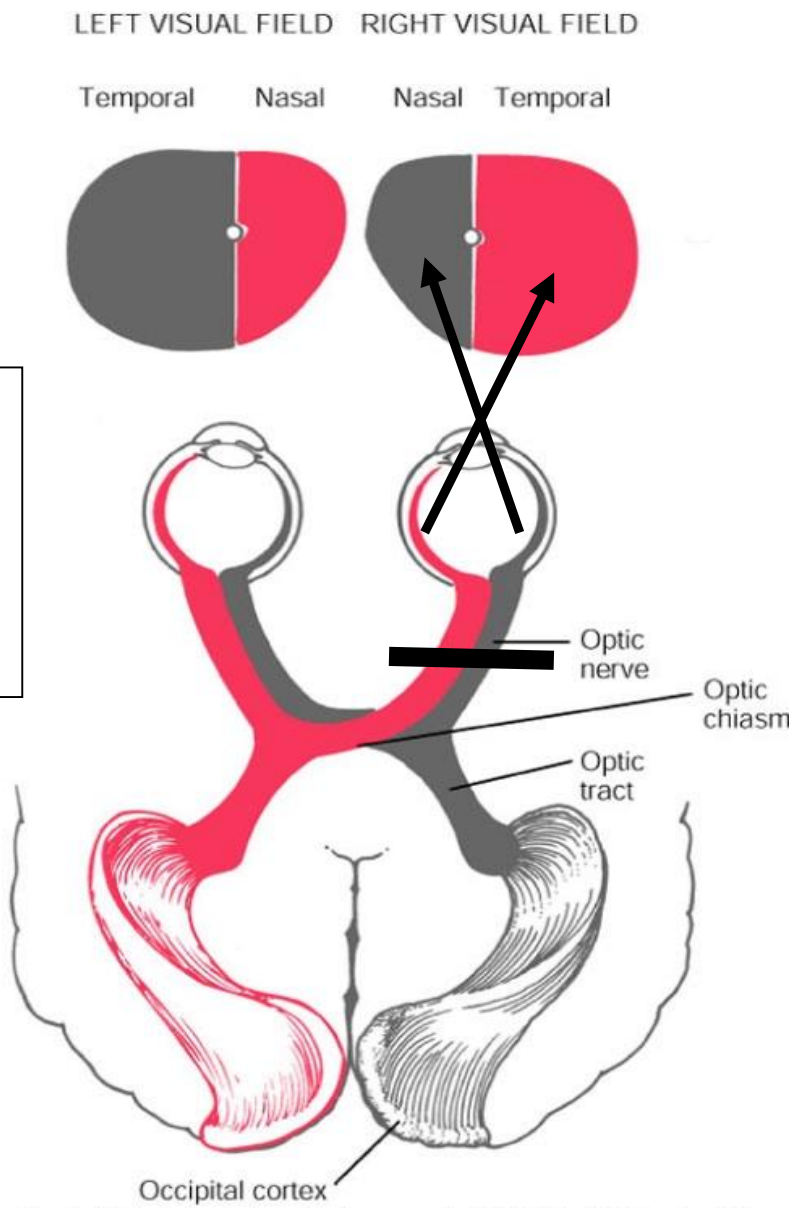
The light stimuli from the **temporal** region of the visual field are received by the **nasal** region of the retina.

The light stimuli from the **nasal** region of the visual field are received by the **temporal** region of the retina.



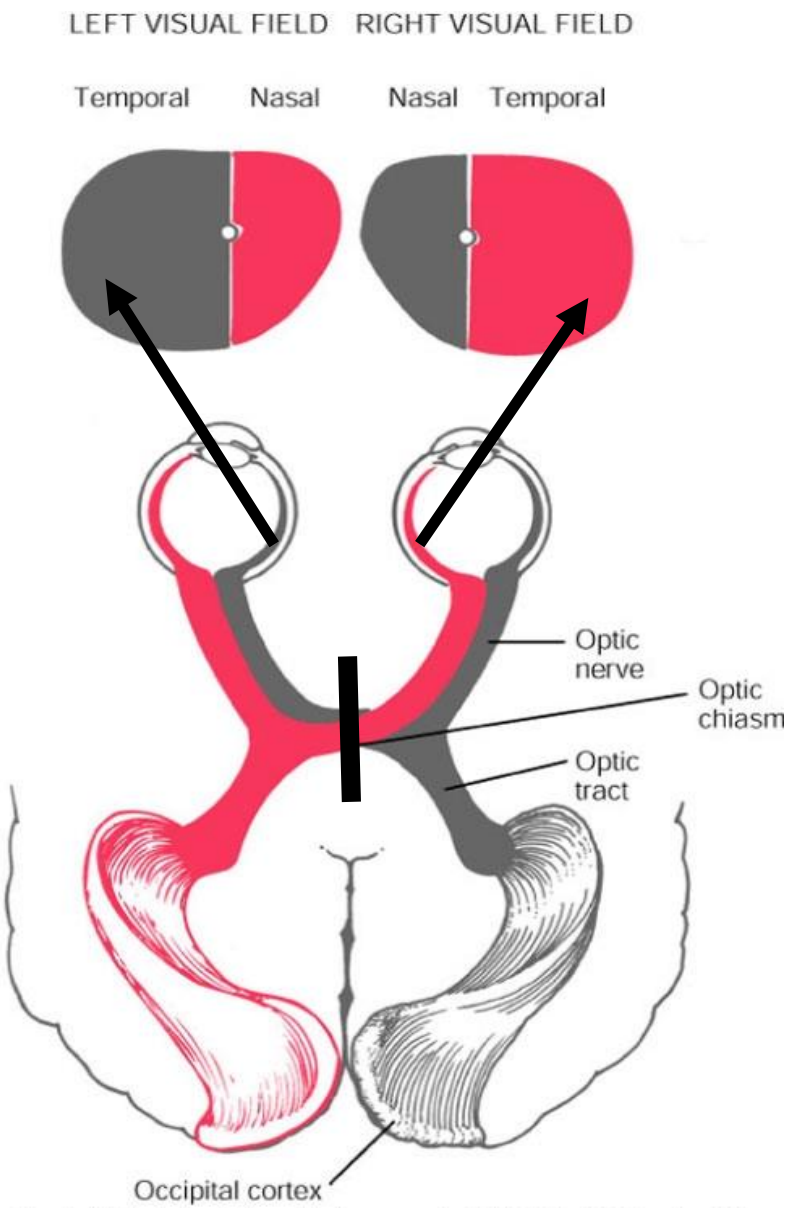
From Jarvis C: Physical examination and health assessment, ed 4, Philadelphia, 2004, Saunders, p 301.

Right optic nerve damage affects vision in the nasal and temporal regions of the visual field of the right eye.



From Jarvis C: Physical examination and health assessment, ed 4, Philadelphia, 2004, Saunders, p 301.

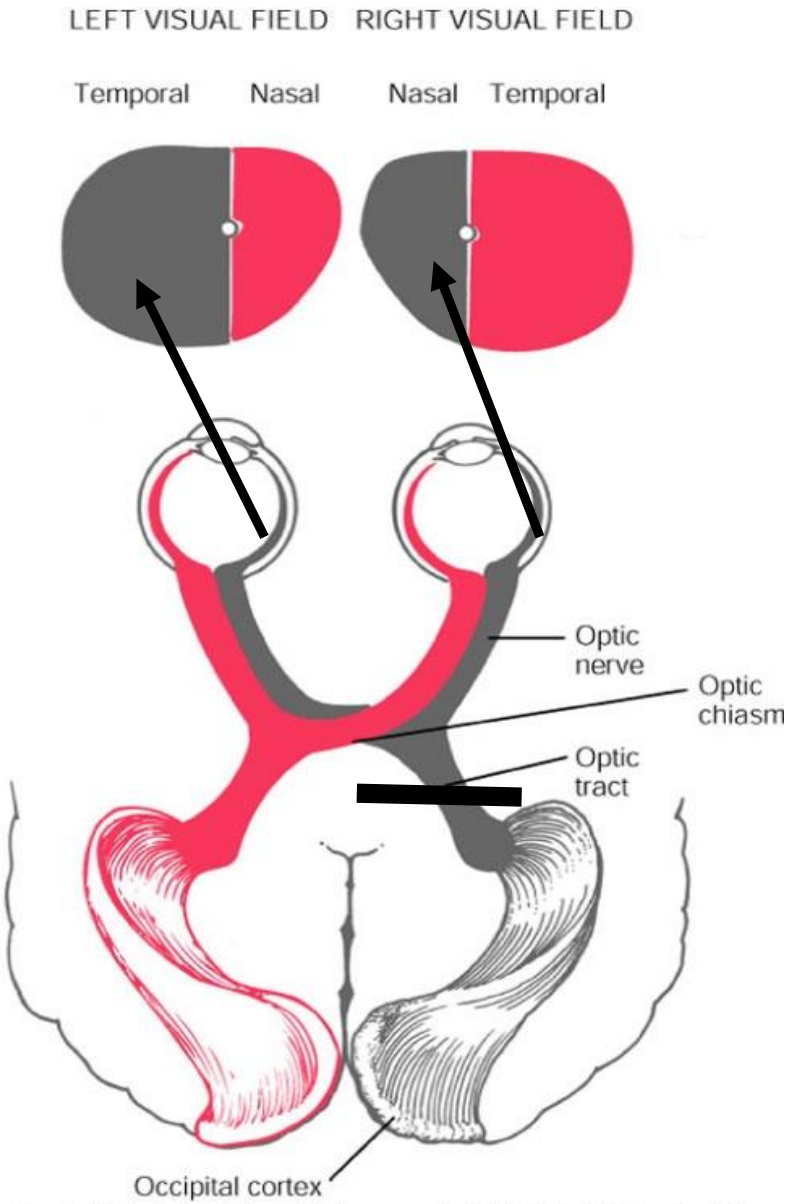
Optic chiasm damage affects vision in the **temporal** regions of the visual fields of both eyes**:** **bitemporal (bipolar) hemianopsia** (blindness in the temporal region of the left and right visual fields).



From Jarvis C: Physical examination and health assessment, ed 4, Philadelphia, 2004, Saunders, p 301.

Right optic tract damage affects vision in the **left** half of each visual field (**nasal** region of the visual field of the right eye and the **temporal** region of the visual field of the left eye): **left homonymous hemianopsia** (blindness in the left side of both visual fields).

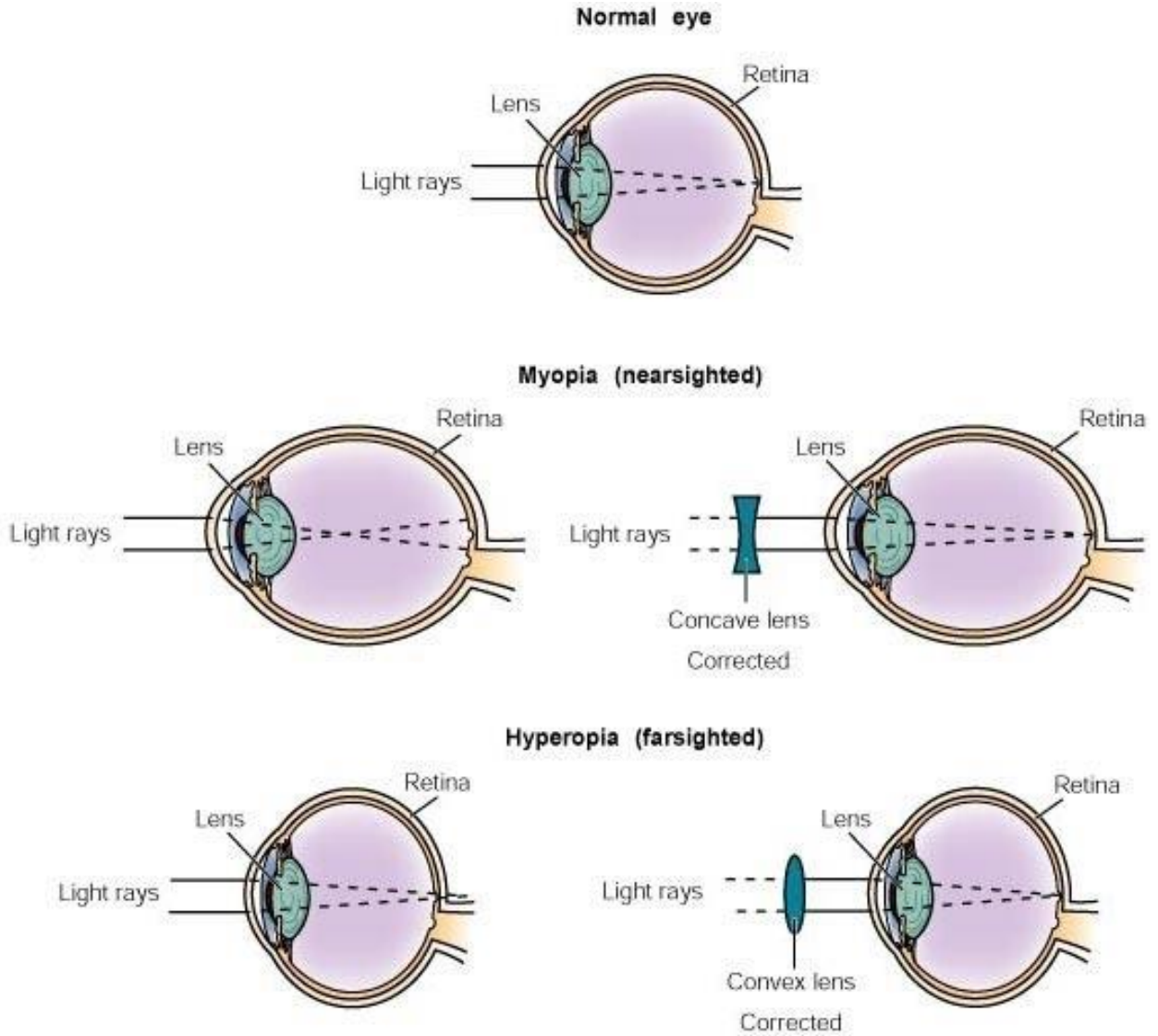
A similar situation exists for damage to the **left** optic tract, **right homonymous hemianopsia** (blindness in the right side of both visual fields).



From Jarvis C: Physical examination and health assessment, ed 4, Philadelphia, 2004, Saunders, p 301.

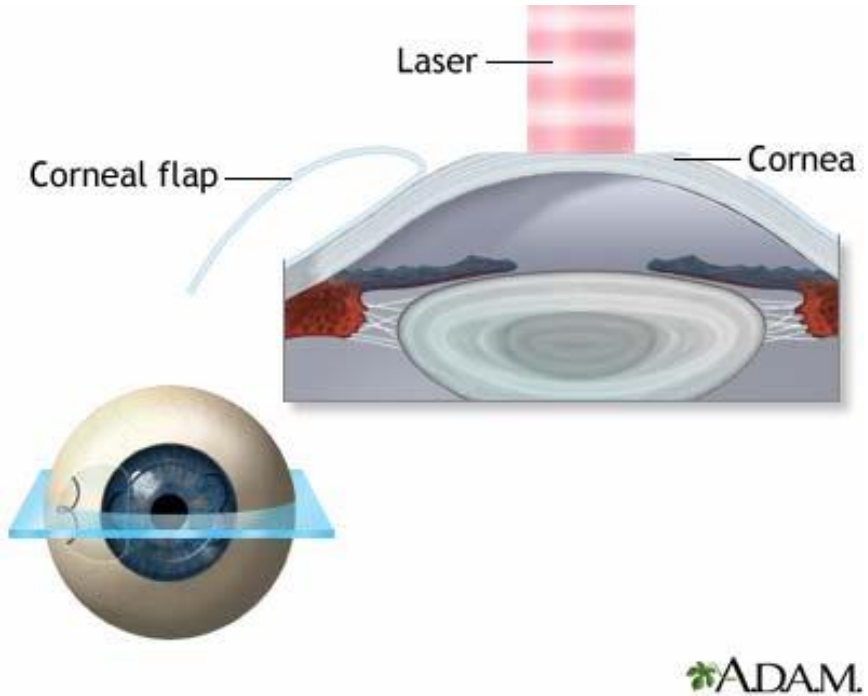
Refraction Disorders

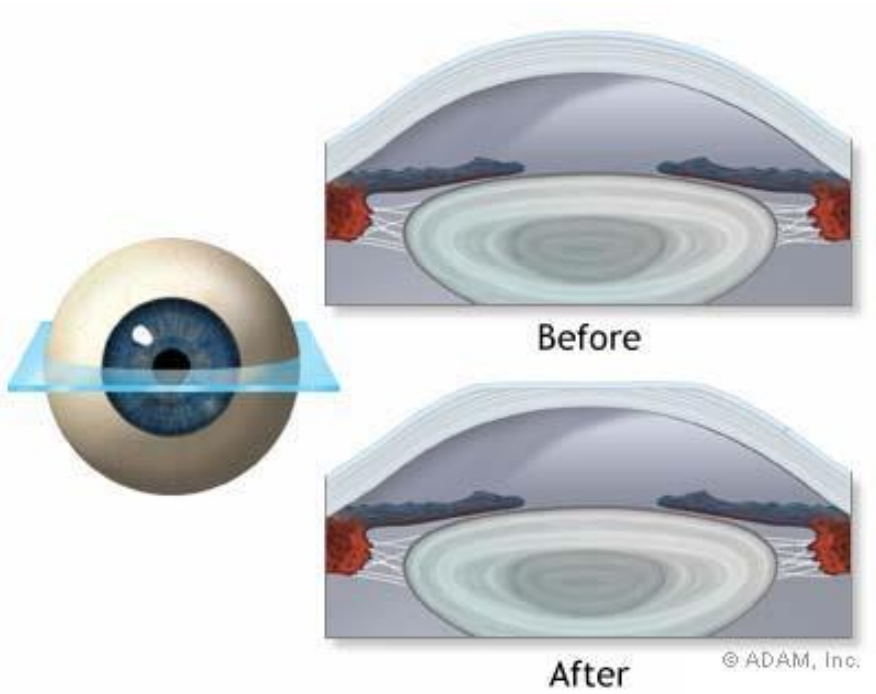
- **Myopia or nearsightedness**-eyeball is too long
  - Image focus is **in front** of the retina
  - **Distant** objects are not seen clearly
  - **Concave** corrective lenses moves focus backward to the retina
- **Hyperopia or farsightedness**-eyeball is too short
  - Image focus is **behind** the retina
  - **Near** objects are not seen clearly
  - **Convex** corrective lenses moves focus forward to the retina
- **Presbyopia**-the loss of accommodation by the lens.
  - Accommodation is the ability of the lens to change shape in order to focus light from near objects. This causes a form of **farsightedness** (hyperopia).
  - Age-related; loss first noticed about age 40; “reading glasses” needed
  - **Convex** corrective lenses
- **Astigmatism**-irregularity in the shape of the cornea or lens
  - Corrected with lenses that are formed with the opposite curvature.



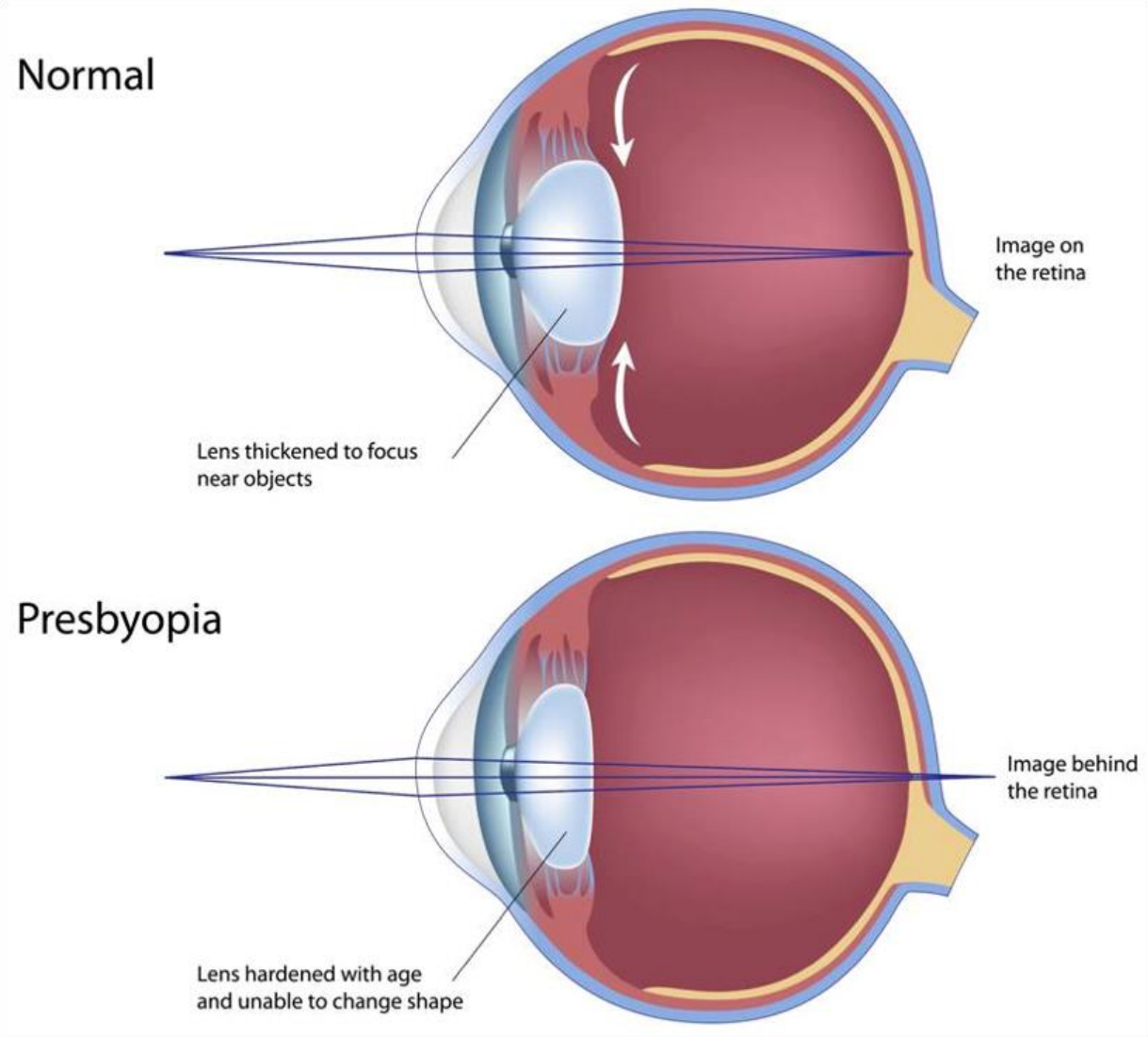
Remember “LMN”
Long eyeball
Myopia
Nearsightedness

Laser Eye Surgery
(Correction for Myopia)





Refraction Disorders



Strabismus (“Cross-Eyedness”)

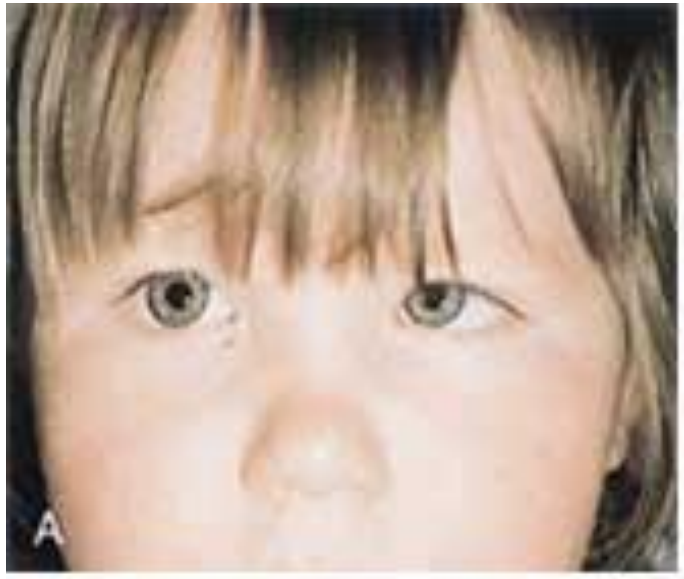
Etiology and Pathogenesis

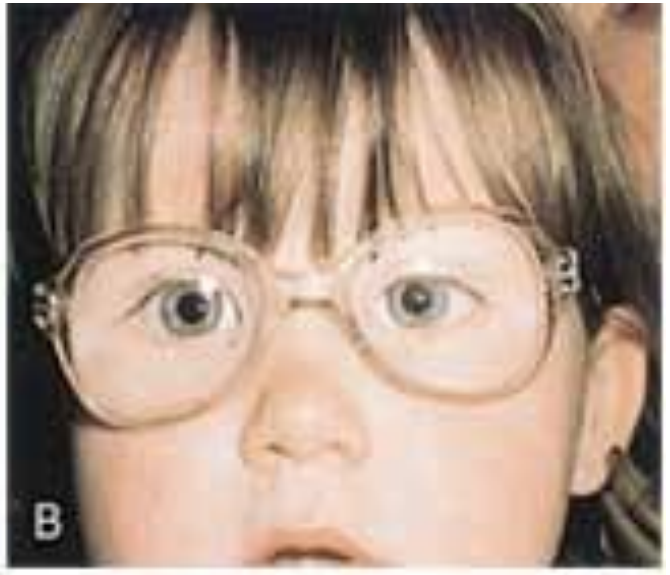
- Eyes cannot fixate simultaneously. Refraction may be normal.
- About 4% of children under 6 years are affected
- Due to weak extrinsic eye muscles on one side
- Eyes appear misaligned
- Squinting, frowning when reading, closing one eye to see
- Difficulty manipulating and picking up small objects
- Dizziness and headache

Treatment

- Occlusion therapy—patching the good eye to force use of the weak eye muscles
- Surgery on the extrinsic eye muscles
- Extrinsic eye muscle exercises

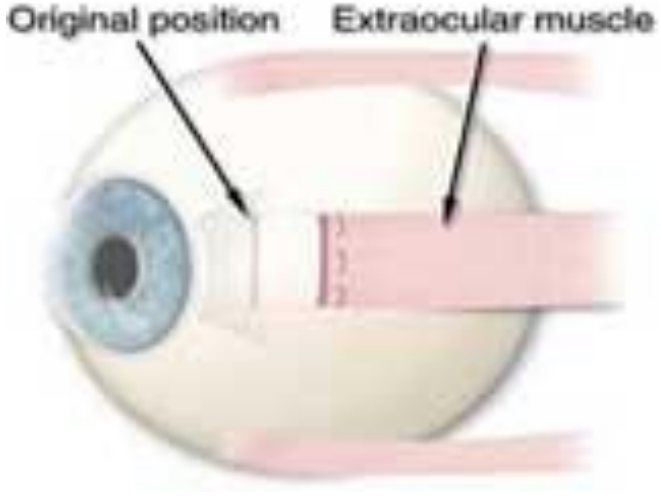
Surgical Correction for Strabismus

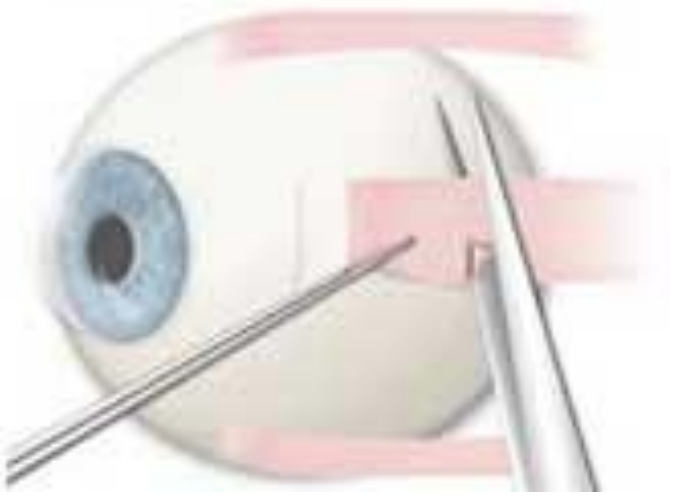




Muscle Recession Procedure

Muscle Resection Procedure





Cataracts

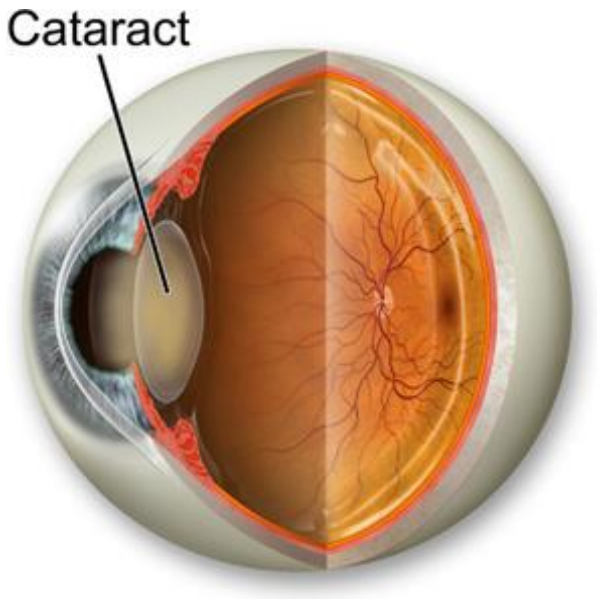
Etiology and Pathogenesis

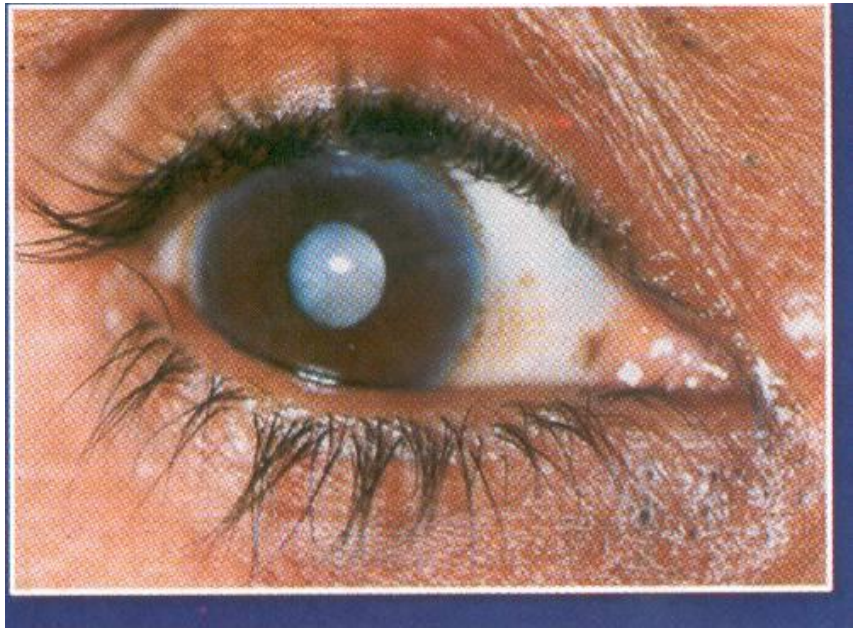
- Clouding of the lens
- Affects more than half of Americans age 65 and older
- Other factors: trauma, Down syndrome, intrauterine Rubella infection, diabetes mellitus, hypoparathyroidism, alcoholism, smoking, steroid drugs, overexposure to ultraviolet light
- Causes increased glare at night, blurred vision, altered color perception.
- If the cataract is in the central portion of the lens, vision will be best in dim light when the pupil is dilated.
- Diagnosed with an ophthalmoscope, pupil appears opaque, retina cannot be visualized.

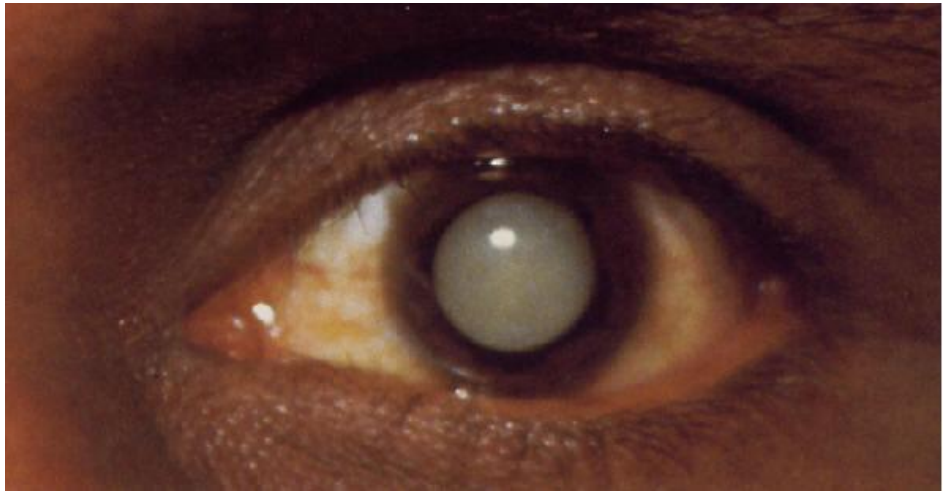
Treatment

- Removal of the affected lens and replacement with a prosthesis.
- The procedure is done on an outpatient basis.

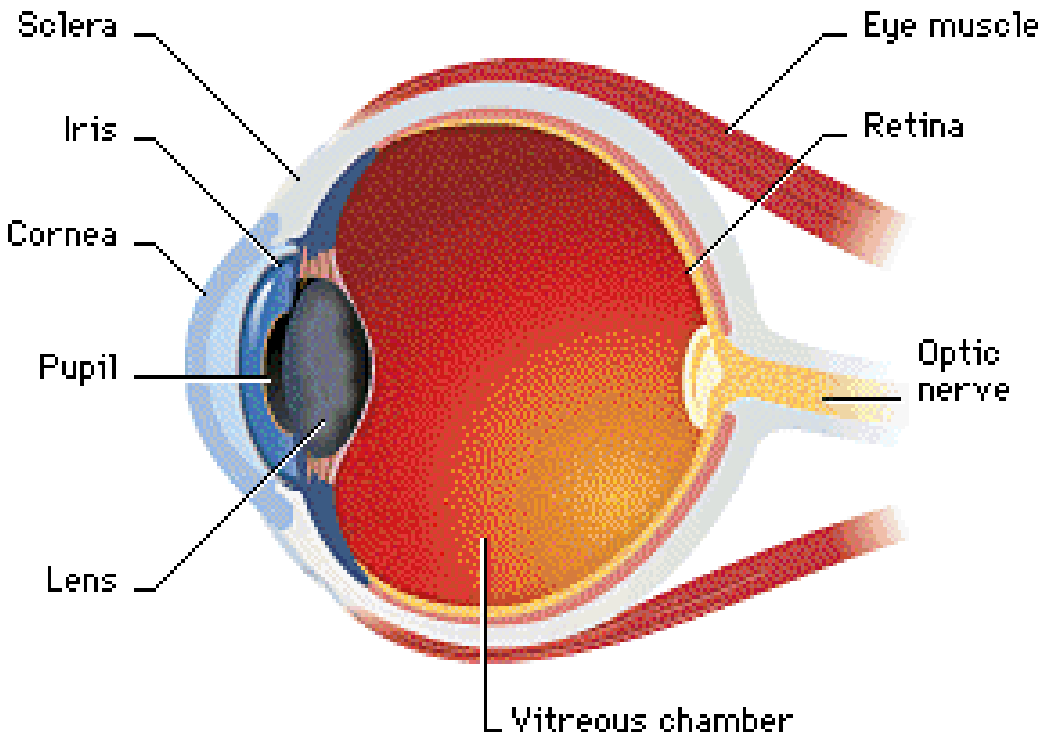
Cataracts

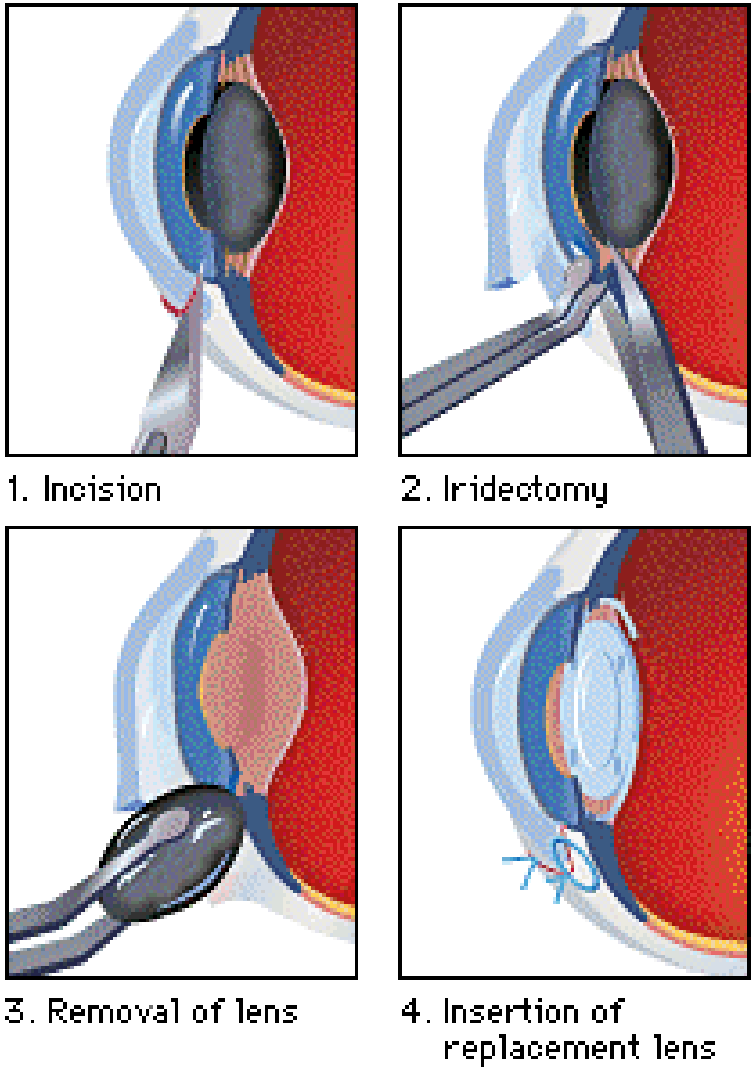






Cataract Surgery





Diabetic Retinopathy

Etiology and Pathogenesis

- The leading cause of blindness in the US.
- The retinal capillaries are unable to transport sufficient oxygen and nutrients to the retina.
- The result is tissue hypoxia and ischemia.
- Diagnosis is made by visual acuity testing, retinal angiography, and ophthalmologic exam.
- There are two categories of the disorder
  - **Nonproliferative**
  - **Proliferative**

Diabetic Retinopathy

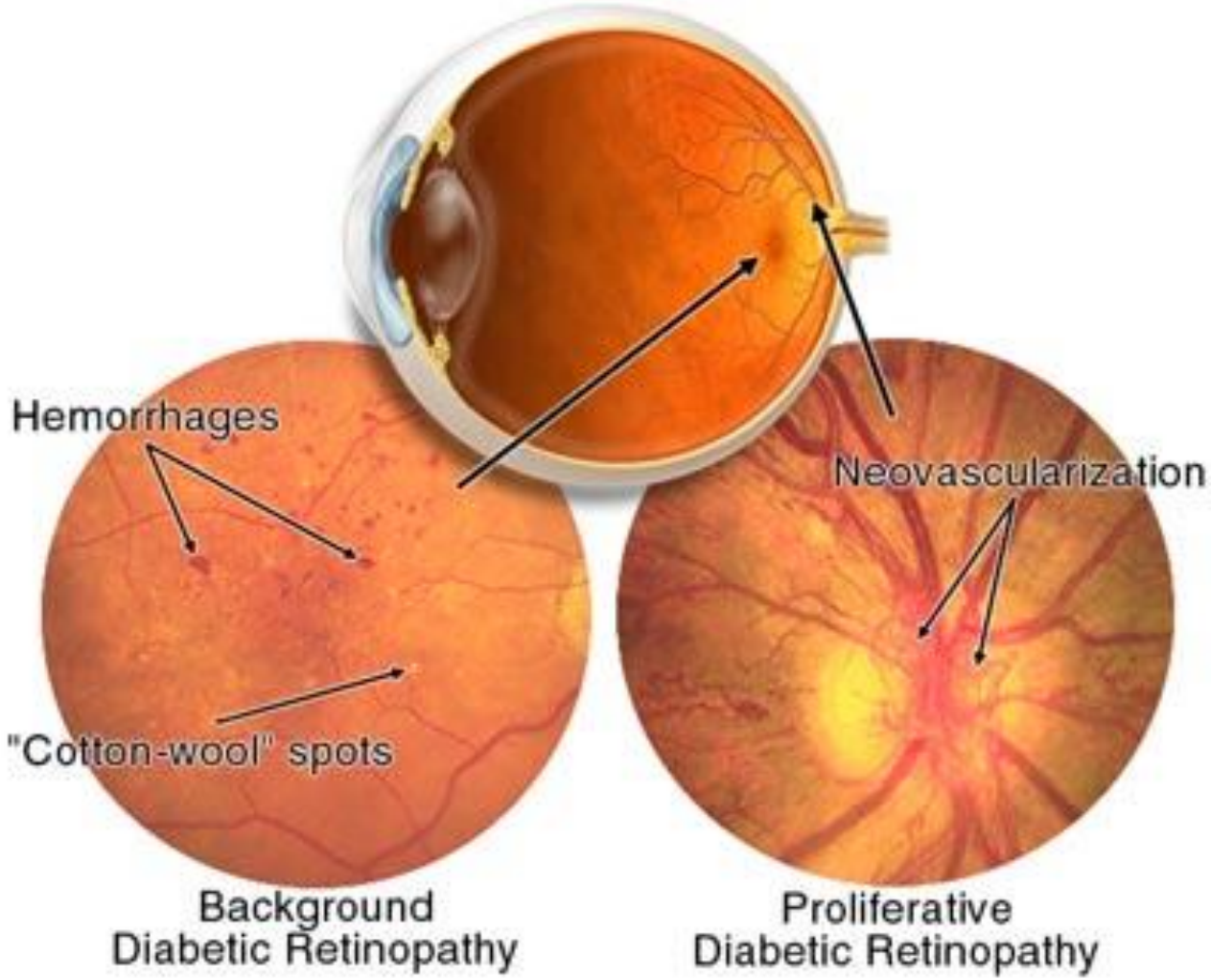
Nonproliferative (aka Background) Diabetic Retinopathy

- Involves the **dilation of retinal veins**, formation of microaneurysms, and retinal hemorrhage. Ischemia results.
- "Cotton wool spots" (infarctions in the retinal nerve fibers) occur.
- Retinal edema occurs. If edema is in the area of the **macula**, visual acuity is affected.
- Laser treatments may be used to prevent further loss of vision

Proliferative Diabetic Retinopathy

- Involves development (proliferation) of **new abnormal blood vessels** in response to loss of retinal blood flow and ischemia.
- Vision is affected in two ways.
  - The abnormal vessels are prone to leakage of blood into the vitreous humor disrupting the pathway of light.
  - The abnormal vessels grow out into the vitreous humor, pulling the retina with them and possibly causing detachment.
- Management-surgical intervention and laser treatments to prevent blindness.

Diabetic Retinopathy



Age-Related Macular Degeneration (AMD)

Etiology

- Progressive degeneration of the retina from the **macula** outward.
- The leading cause of permanent visual loss in the elderly.
- The cause is unknown. The result is loss of **centra**l field vision.
- Risk factors: Female gender, family history, smoking, high serum cholesterol, cardiovascular disease, and significant sun exposure

Diagnosis

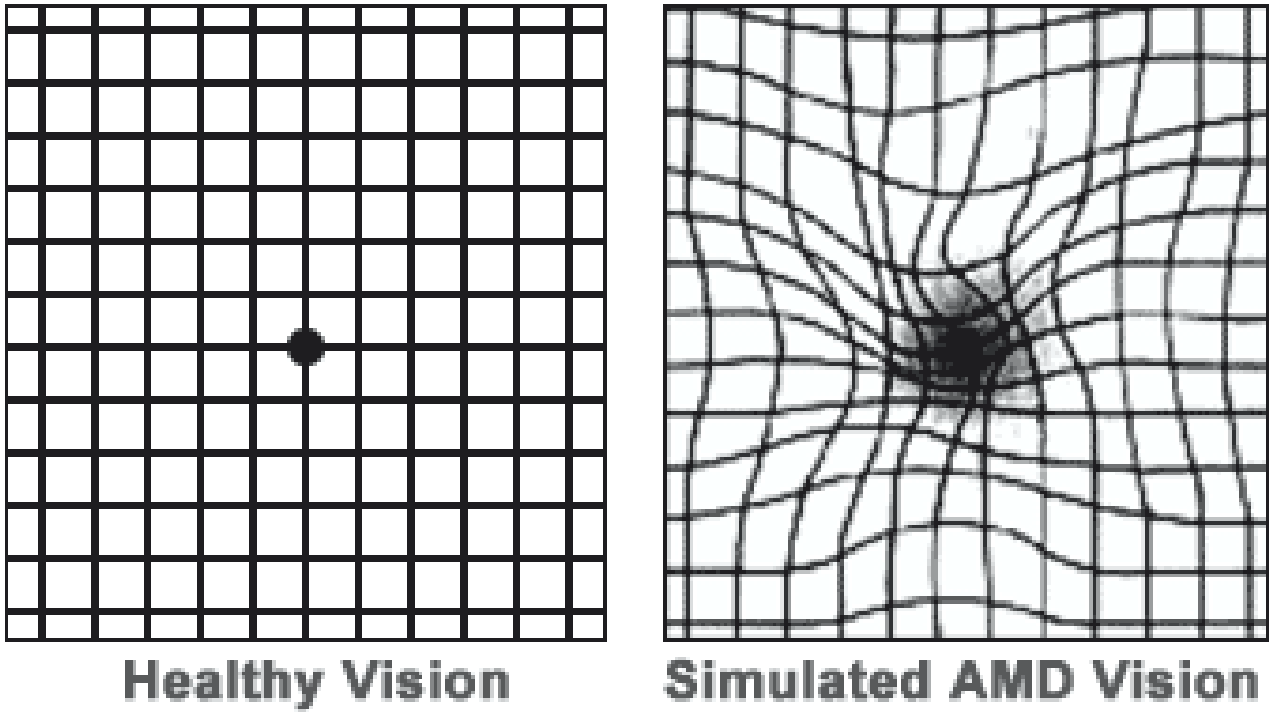
- Visual acuity tests
- Dilated retinal exam
- Use of the Amsler grid: lines appear wavy and areas of the grid may appear to be missing
- Fluorescein angiography will show changes in **choroid blood vessels.** The choroid is the tissue layer behind the retina.

Treatment

- Antioxidant/zinc vitamin supplements to slow progression
- Laser treatments in the case of vascular changes

Age-Related Macular Degeneration (AMD)

Amsler Grid



Two Forms of AMD

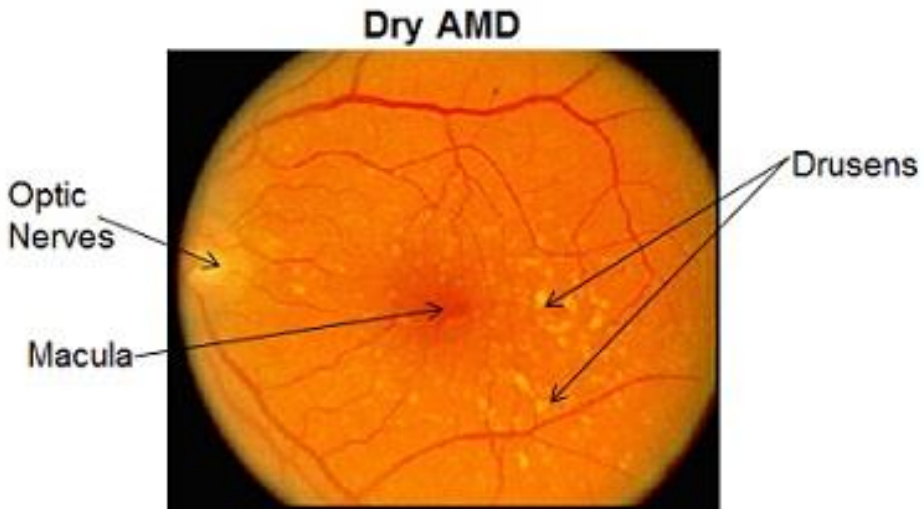
Dry (Atrophic) AMD

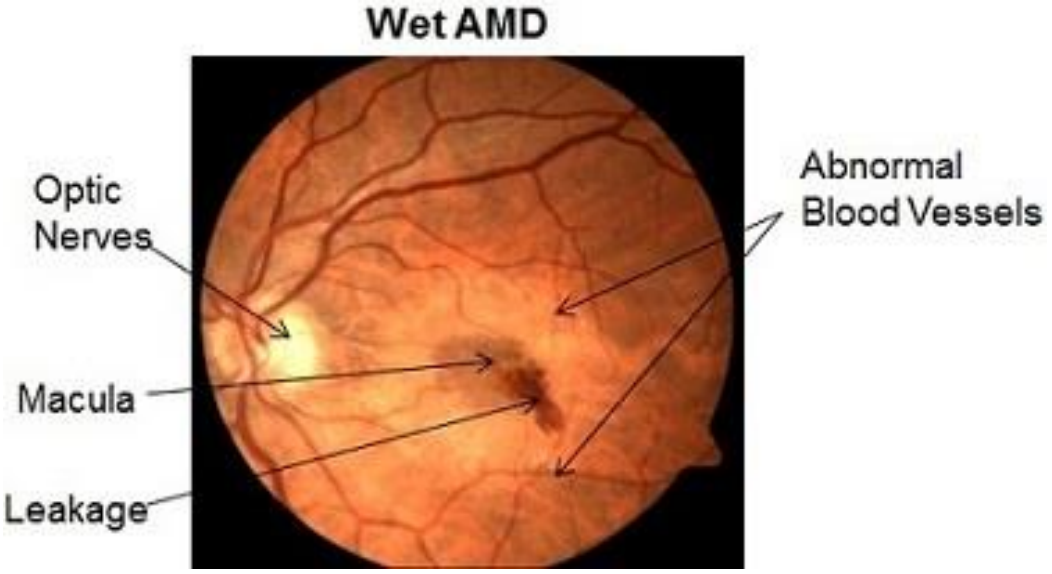
- 90% of all AMD patients have this type.
- Degeneration of the outermost, pigmented layer, of the retina and the choroid layer of the eyeball. It does not involve bleeding into the retina (dry).
- Ophthalmoscope exam reveals subretinal yellow deposits=**drusens**
- Affects just one eye initially, but spreads to the other.
- Initially there is blurred vision and decreased ability to see detail.
- With progression the area of vision loss becomes larger and darker.

Wet (Exudative) AMD

- Rapid onset and affects vision more severely.
- Blood vessels invade the retina and bleed into it.
- Subretinal fluid collections often lead to retinal detachment and neovascularization.

Macular Degeneration





Retinal Detachment

Etiology and Pathogenesis

- Usually spontaneous, may be secondary to head trauma
- Predisposing factors-eye tumors, myopia, cataract extraction, wet AMD.
- Tearing of the retina allows vitreous humor to flow behind the retina causing progressive detachment and visual loss.

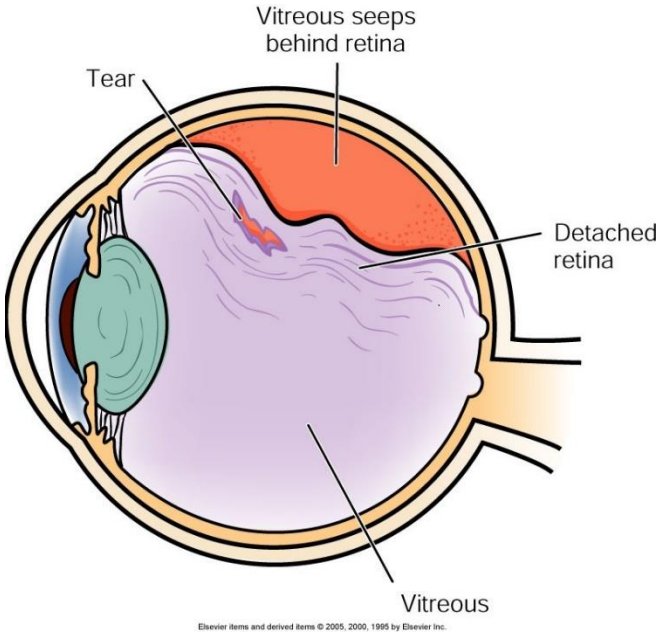
Clinical Manifestations

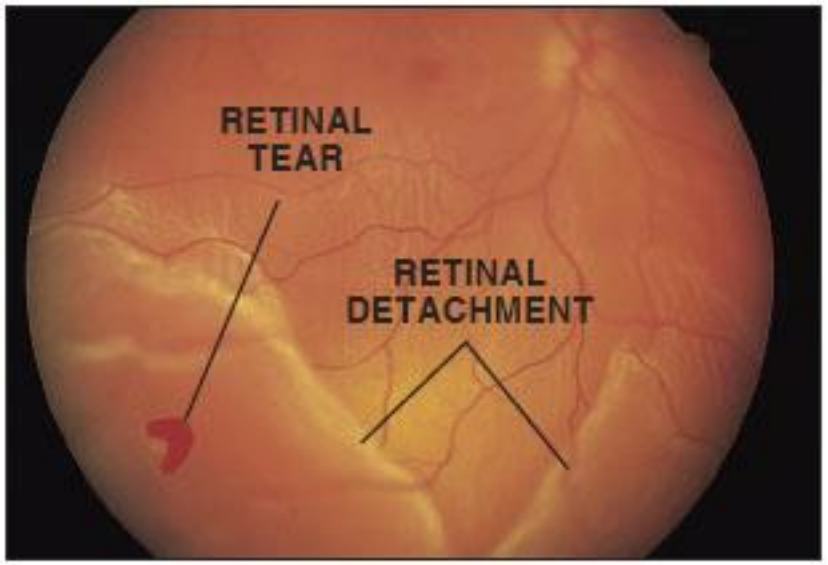
- Sudden appearance of floating spots in the visual field
- Flashes of light when the eye moves
- Blurring of vision as if a curtain is being pulled down over the eye.
- Vitreous hemorrhage may also occur.
- Diagnosis is made by examining the retina with an ophthalmoscope.
- Retina appears to hang in the vitreous like a gray cloud.

Retinal Detachment

Treatment

- Closing retinal tears so that reattachment can occur.
- Surgery is common. It followed by days of lying still to allow healing.
- 80% of uncomplicated cases are resolved with one procedure.

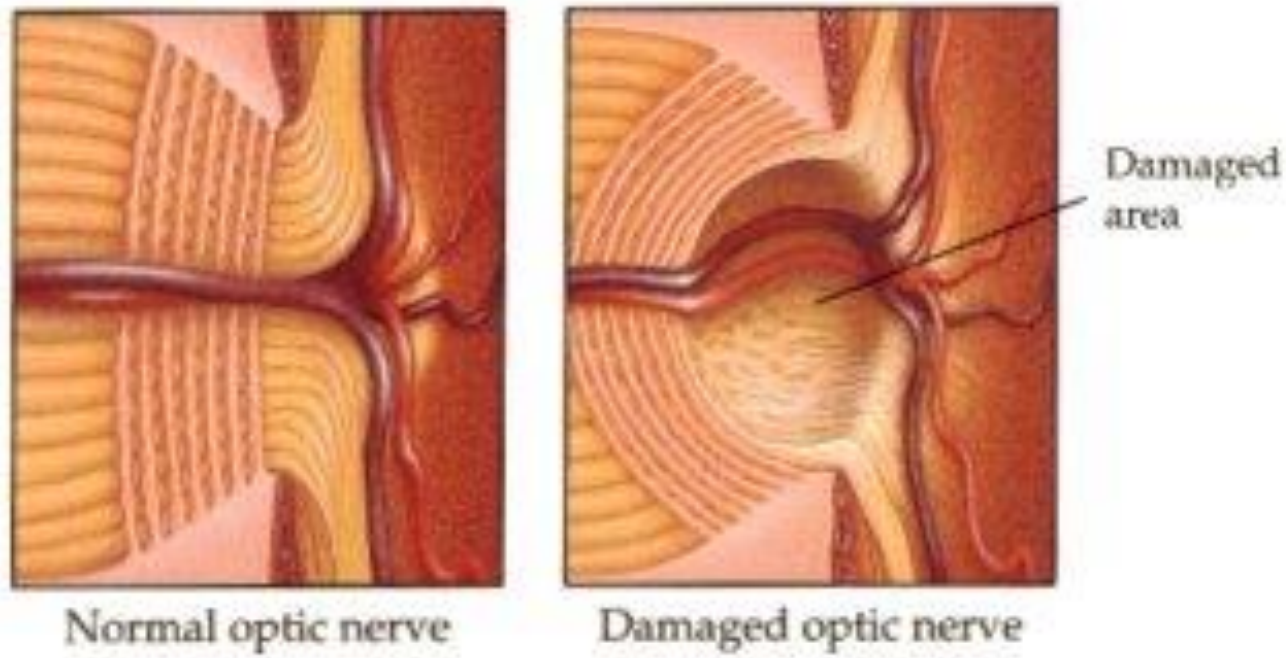




Glaucoma

- Increased production or decreased drainage of **aqueous humor** causes increased intraocular pressure and progressive loss of vision.
- Fluid pressure on the optic nerve and retina decreases blood flow and leads to ischemic loss of function.
- A progressive loss of the visual field results beginning at the periphery of the field (**“tunnel” vision**). Vision is best at the center of the visual field.
- More common in the elderly, African Americans, diabetics, those with myopia, and those with a family history.
- There are two main types of glaucoma:
  - **Closed Angle Glaucoma**
  - **Open Angle Glaucoma**

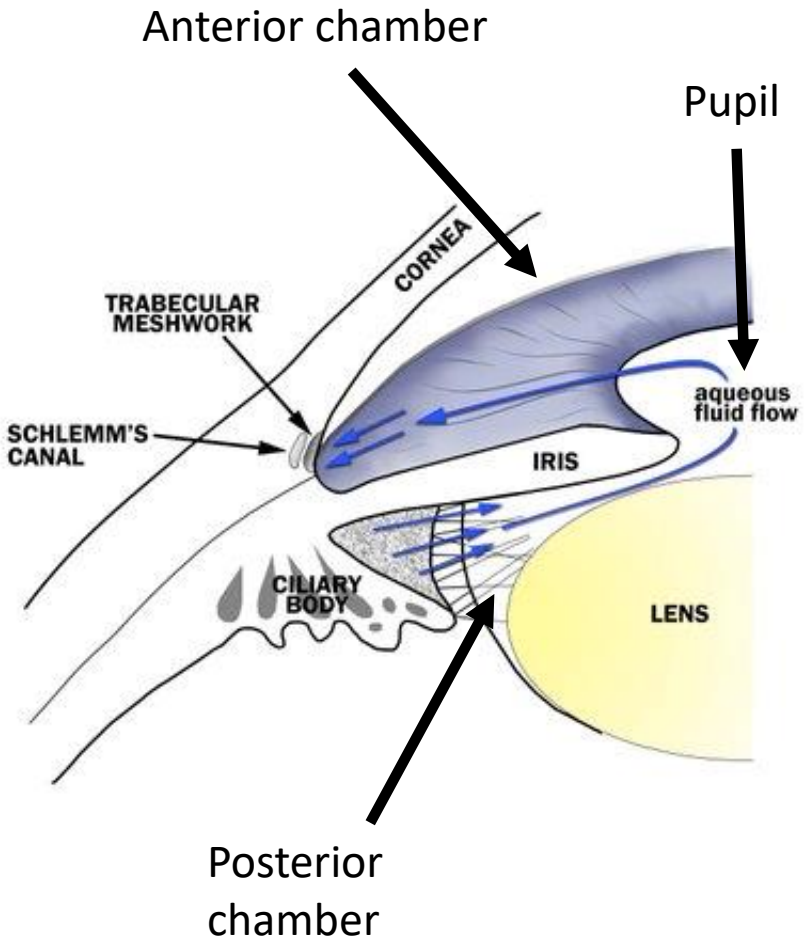
Effect of Glaucoma



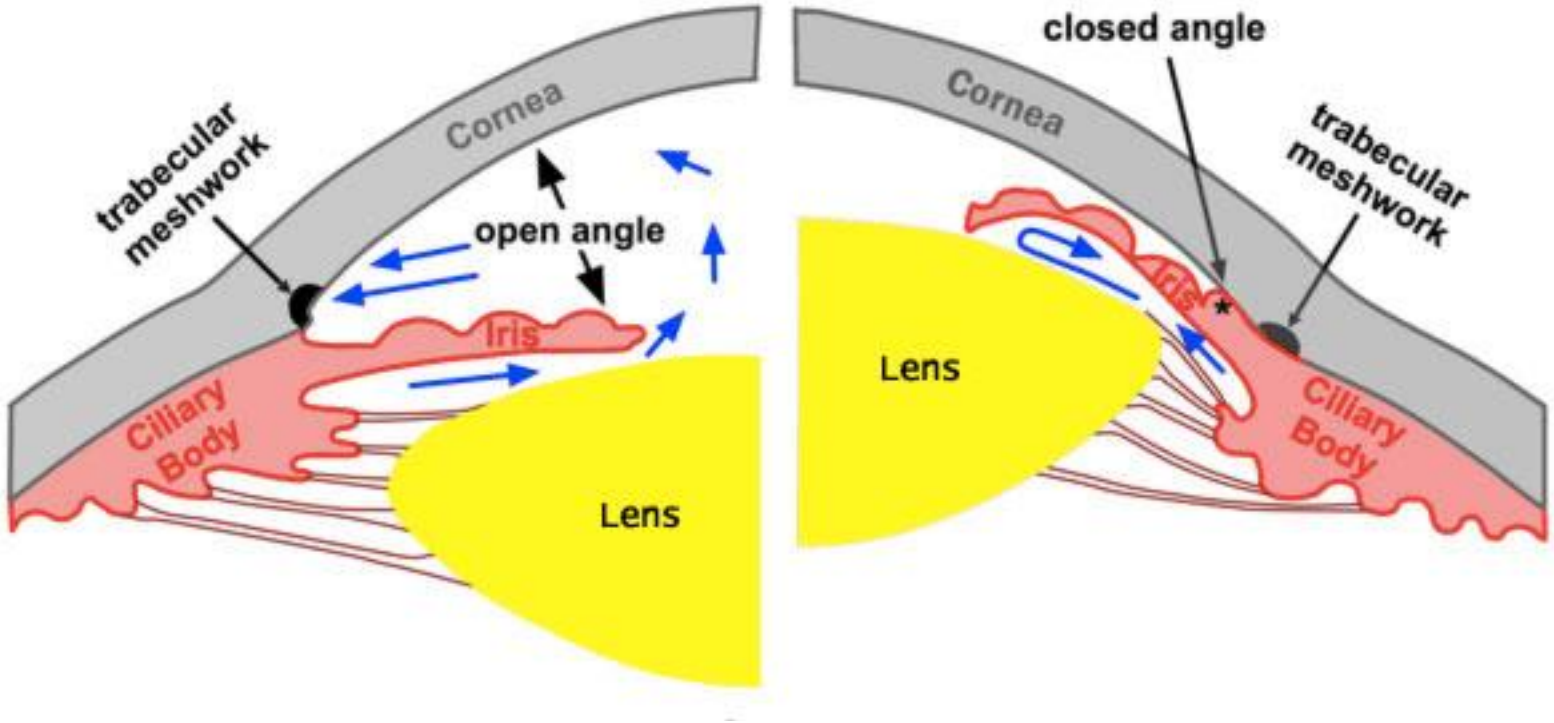
"Tunnel Vision"

Aqueous humor is produced by the capillaries of the **ciliary body** that encircles the lens. The ciliary body contains smooth muscle that can cause the lens to change shape when focusing light on the retina.

Aqueous humor circulates between the eye and the blood. It flows forward from the **posterior chamber** through the **pupil** into the **anterior chamber** and then drains through the trabecular meshwork into the **Canal of Schlemm** at the periphery of the iris. From there it enters the **blood** via the **aqueous veins.**

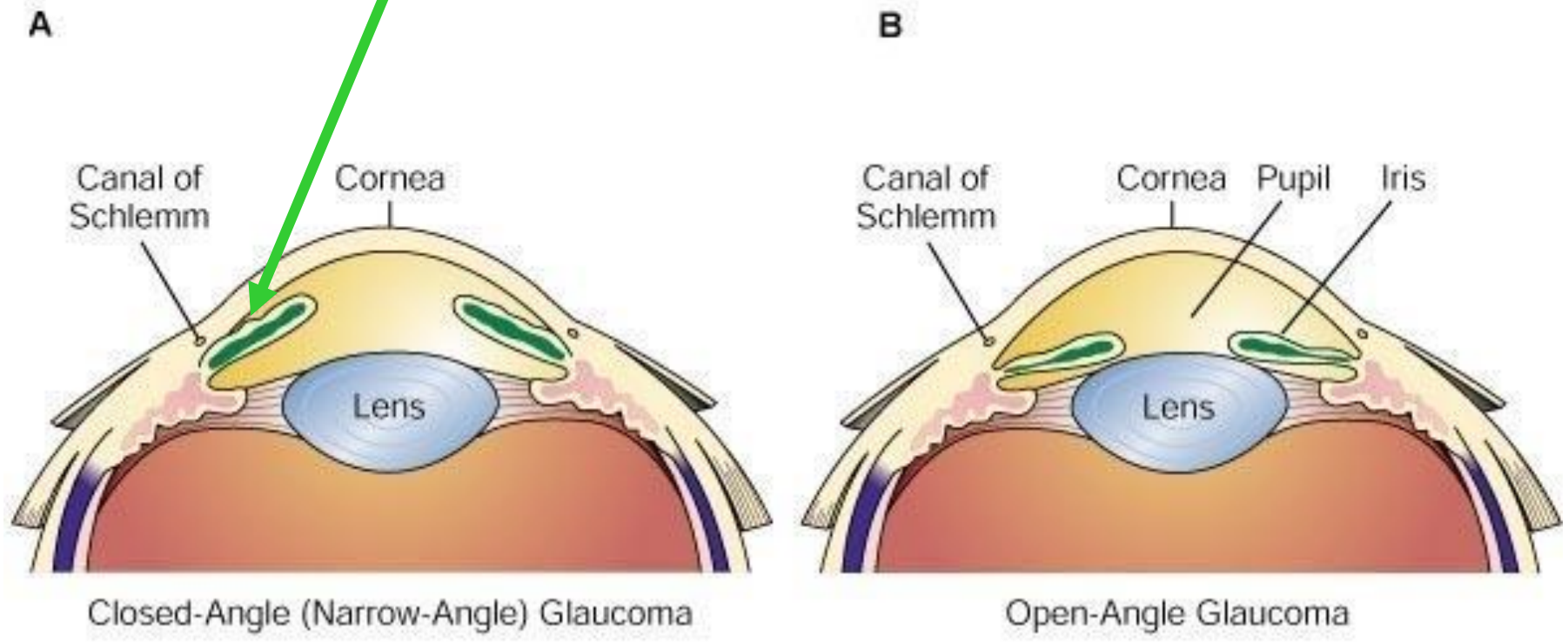


Glaucoma


trabecular meshwork
Cornea
open angle
Iris
Ciliary Body
Lens
closed angle
Cornea
trabecular meshwork
Iris
Lens
Ciliary Body

The angle between the cornea and the iris is too narrow. Drainage into the Canal of Schlemm is obstructed.

Too much aqueous humor is produced, but drainage is not blocked.



Closed Angle Glaucoma

Etiology and Pathogenesis

- A build-up of aqueous humor behind the iris pushes it forward decreasing the angle between the iris and the cornea. Drainage of aqueous humor into the Canal of Schlemm is blocked.
- Eye pain, nausea, vomiting, blurred vision with halos, redness of the eye, a steamy cornea, and a dilated pupil not reactive to light.
- It is an EMERGENCY situation! It can cause permanent blindness within days of onset.

Diagnosis

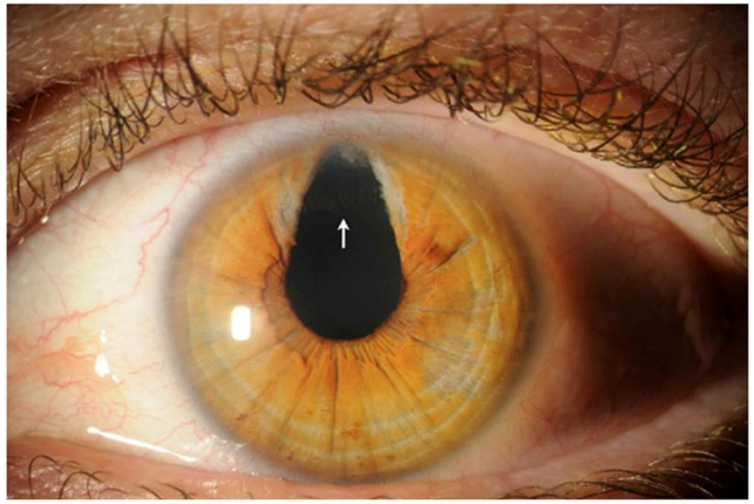
- Intraocular pressure measurement
- Ophthalomoscopic exam of the optic disk
- Central field vision testing

Treatment

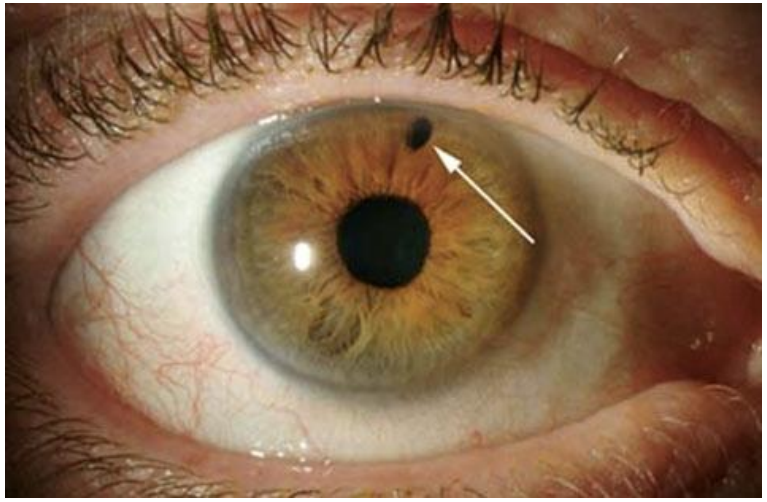
- Drugs to diminish production of aqueous humor.
- **Laser iridectomy (removing part of the iris) or iridotomy** (making a hole or holes in the iris) to affect a permanent cure.

Closed-Angle Glaucoma Treatment

Iridectomy



Iridotomy



Open-Angle Glaucoma

Etiology and Pathogenesis

- 90% of all glaucoma cases
- Due to overproduction of aqueous humor.
- Onset is insidious, dull eye pain or inability to distinguish colors.

Diagnosis

- Intraocular pressure measurement
- Ophthalomoscopic exam of the optic disk
- Central field vision testing.

Treatment

- Eye drops containing the **β-adrenergic receptor blocker, timolol**, to reduce aqueous humor production.
- Miotics (**pilocarpine**) are useful in **constricting the pupil** so that the ciliary muscle pulls on the trabecular meshwork of the canal of Schlemm and increases the drainage of aqueous humor.
- Laser surgery aimed at the trabecular meshwork may also be done.

QUIZ 7CD

- COMPLETE QUIZ 7CD.
- THEN PREPARE FOR EXAM 7.

EXAM 7

- COMPLETE EXAM 7.
- THEN PREPARE FOR YOUR FINAL EXAM.