

NWC EMSS Continuing Education
Eye and Ear Disorders and Trauma
Independent Study Materials: September 2012
Connie J. Mattera, MS, RN, EMT-P
Updates to new Ed Standards: Jennifer Dyer, R.N., B.S., EMT-P

I. Incidence of ocular trauma

- A. Approximately 1.2-2.5 million people suffer eye injuries each year in the U.S. of which 500,000 are serious. About 43% occur in the home (U.S. Eye Injury Registry).
- B. Over 40,000 are associated with some visual loss and 25,000 result in total blindness.
- C. Ocular trauma is the leading cause of visual loss in persons less than 25 years of age and the leading cause for eye-related hospital admissions.
- D. The leading causes of eye injuries include household chemicals, workshop and yard debris, battery acid, sports accidents, consumer fireworks, over-exposure to UV radiation, and inappropriate toys and games used without supervision (Am Academy of Ophthalmology, 2002).
- E. An estimated 40,000 sports-related eye injuries occur each year. About 90% are preventable by using appropriate sports-specific eyewear.
- F. Workplace and MVC ocular injury rates have reduced significantly due to application of general safety measures, more effective safety glasses, mandatory seat belt legislation and use of airbags.
- G. Specific ocular surveillance systems include the **National Eye Trauma System (NETS)** and the **United States Eye Injury Registry (USEIR)**.

II. Immediate goals in ocular injuries

- A. Protection of the intact portions of the visual system and avoidance of further injury to undamaged structures. A total loss of vision in one eye equals a 25% impairment of Visual System and a 24% impairment of Whole Man (Work Eye Injury Registry, 2002).
- B. Accurate assessment of the extent of injury and referral of the patient for immediate repair of injured tissues to prevent further damage.
- C. Institution of therapeutic measures that first achieve optimal function and secondarily achieve optimal cosmetic results.

III. Anatomy and physiology of the eye

The function of the eye depends on an exact maintenance of anatomical relationships between the eyelids, cornea, anterior chamber, lens, retina, extraocular muscles, and nerves. Permanent deficit in any of these components may result in altered visual function and potentially, loss of the eye.

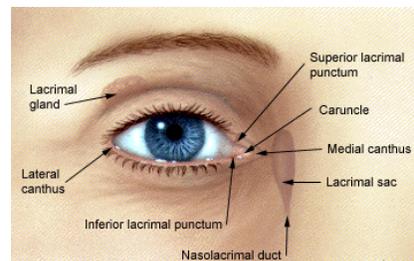
A. Periocular and orbital structures

- 1. **Bony orbit:** Provides structure and support for housing the globe. Composed of frontal, maxillary, zygoma, nasal, ethmoid, greater and lesser sphenoid, lacrimal, and palatine bones. These are joined to form a quadrilateral pyramid with a posterior apex.

Damage to these bones can cause globe injury. Since the globe and orbit are in close approximation to many other important non-ocular structures, serious ocular injury is often seen in the context of serious non-ocular injury.



2. **Eyelids:** Continuation of the facial skin and serve as protective coverings to the eyes, distribute tear film evenly to lubricate the cornea and aid in the removal of excess tears and tear film debris. Eyelids close tightly to protect the eye. Both are lined with conjunctiva.
 - a. Orbital portion
 - b. Tarsal portion: dense, fibrous, connective tissue measuring 10 mm vertically in the upper eyelid and approx. 3.6 mm in the lower lid. The tarsus is the structural support for the lid.
 - c. Fornices: superior and inferior



3. **Medial and lateral canthus** (corners of the eye): Points of attachment for upper and lower eyelids.
4. **Eyelashes:** Strong hairs that help prevent foreign particles from reaching the surface of the eye.

5. **Conjunctiva:** Thin, vascular mucous membrane that lines the posterior surface of the eyelids and covers the anterior surface of the sclera. Contains many free nerve endings and is very sensitive.



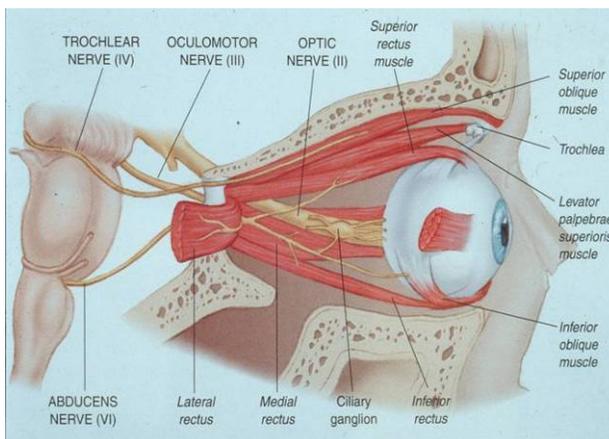
- a. Bulbar: Outer surface of sclera – white
- b. Palpebral: Lines inner surface of lids - reddish in color

6. **Glands/drainage systems**

- a. **Lacrimal gland:** Located within a depression in the frontal bone, just inside the orbit and superior and lateral to the eyeball, the lacrimal gland has a dozen or more ducts that empty into the pocket between the eyelid and the eye. It continuously secretes tears, distributed across the eye through blinking, that reduce friction, remove debris, prevent bacterial infection, and provide nutrients and oxygen to the conjunctival epithelium (Martini et al, 2008). Tears are watery, slightly alkaline, and contain *lysozyme*, an enzyme that attacks bacteria. Blinking the eye sweeps the tears to the medial canthus.
- b. **Puncta:** Orifices leading to the lacrimal drainage system in the upper and lower eyelids located about 5 mm from the medial canthus.
- c. **Sebaceous glands:** Associated with the eyelashes as with other hair follicles. Secrete a lipid-rich substance that keeps lids from sticking together.
- d. **Lacrimal caruncle:** Soft mass of tissue located at the medial canthus containing glands that produce thick secretions that contribute to the gritty deposits sometimes found after a night's sleep.

B. **Extrinsic eye (oculomotor) muscles:** Originate on the surface of the orbit and control the position of the eye.

- | | | | |
|----|-------------------|--------------------------------------|--------|
| 1. | Medial rectus: | Eye rotates toward the nose | CN III |
| 2. | Inferior rectus: | Eye looks downward | CN III |
| 3. | Inferior oblique: | Eye looks upward and to the side | CN III |
| 4. | Superior rectus: | Eye looks upward | CN III |
| 5. | Superior oblique: | Eye looks down and to the side | CN IV |
| 6. | Lateral rectus: | Eye rotates laterally away from nose | CN VI |



C. **Eyeball (Globe)**

1. Each eye is roughly spherical with a diameter of about 2.5 cm (1 inch). The length from the apex of the cornea to the point at which the optic nerve exits the sclera is approximately 24.5 mm. The globe weighs 7.5-8.0 g and has a volume of 6.5 ml. It occupies 1/5th of the orbital volume. It shares space with the extrinsic eye muscles, the lacrimal gland, the various cranial nerves and blood vessels that service the eye and adjacent areas of the orbit and face, and orbital fat that provides padding and insulation (Martini et al, 2008).

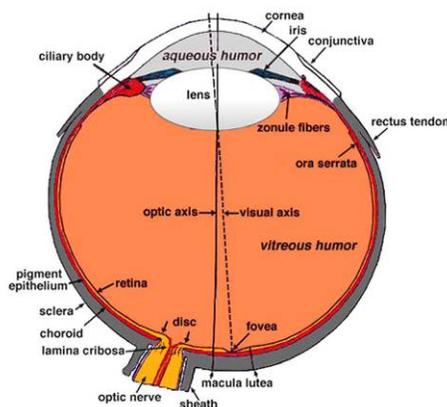


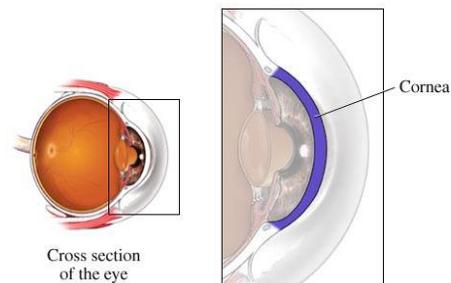
Fig. 2. Sagittal horizontal section of the adult human eye.

2. **Wall of the eye has three distinct layers or tunics**
 - a. Outer **fibrous tunic**: Outermost layer covering the eye; composed of the sclera and cornea
 - (1) Provides mechanical support and some physical protection
 - (2) Serves as an attachment site for extrinsic eye muscles
 - (3) Assists in the focusing process
 - b. Intermediate **vascular tunic**: Contains numerous blood vessels, lymphatics, and all of the intrinsic eye muscles, iris, ciliary body, and the choroid.
 - c. Inner **neural tunic** or retina
3. **Sclera**
 - a. Dense, fibrous connective tissue that contains both collagen and elastic fibers. It forms the outer layer of the globe, and is composed of epithelial cells.
 - b. Thickest over the posterior and thinnest over the anterior surface
 - c. Extrinsic eye muscles insert on its surface

- d. Its posterior surface contains nerves and small blood vessels that penetrate to reach internal structures. On the anterior surface, these vessels lie under the conjunctiva. Because the capillary network carries so little blood, there is no obvious color, so the white of the collagen fibers is visible (thus forming the white of the eye).
- e. **Functions**
 - (1) Protect the internal ocular structures
 - (2) Act as the structural skeleton of the globe

4. **Cornea**

- a. Transparent, avascular, concave dome continuous with the sclera that covers the iris (11.5 - 12.5 mm across).
- b. It ranges in thickness from 0.7 mm peripherally to 0.5 mm centrally.
- c. Collagen fibers are organized into a series of layers that permit the transmission of light through the cornea, pupil and lens to the retina. It is covered by a thin layer of epithelium overlying a basement membrane (Bowman's membrane).
- d. Innervated by the sensory limb of CN V (trigeminal nerve). Because the cornea is so richly innervated beneath the corneal epithelium, the pain of corneal injury is often greater than that of iritis or conjunctivitis. Topical anesthesia freely penetrates the cornea, but not the inner aspect of the eye. Therefore it has little effect on pain caused by deeper problems.



- 5. **Limbus:** Circular margin where the conjunctiva meets the cornea
- 6. **Iris:** Circular, contractile muscular disc that is an extension of the ciliary body that is located anterior to the lens. It contains pigment cells that produce the color of the eye and two layers of smooth muscle. The iris controls the amount of light reaching the retina by dilating and constricting its muscles to change pupillary size in response to impulses mediated by CNs II & III. When there are no pigment cells in the iris, light passes through it and bounces off its inner surface of pigmented epithelium. The eye then appears blue. In order, persons with gray, brown, or black eyes have increasing numbers of pigment cells in the body and surface of the iris (Martini et al, 2008).

7. **Pupil:** Typically is a round central opening in the iris

- a. **Parasympathetic stimulation:** Rapid reflex constriction of the pupil in response to bright light.
- b. **Sympathetic stimulation:** Slower pupillary dilation in response to a reduction in light levels

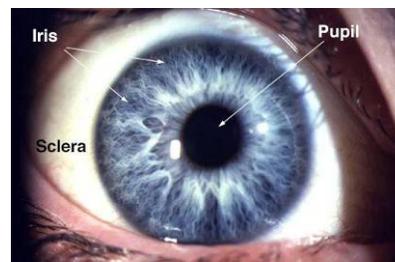
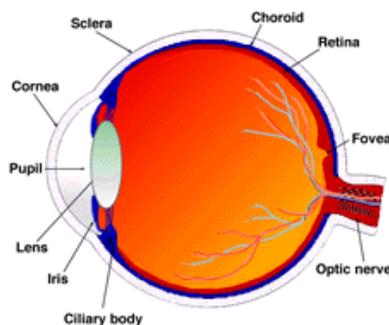


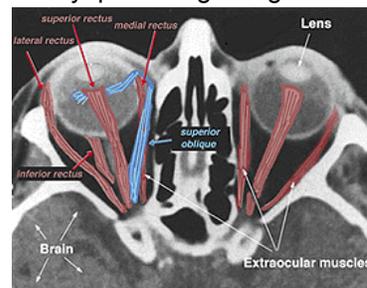
Fig. 1. View of the human eye

- 8. **Ciliary body:** Along its outer edge, the iris attaches to the anterior portion of the ciliary body which is composed primarily of a ring of ciliary muscle that projects into the interior of the eye. It begins at the junction between the cornea and sclera and extends to the anterior edge of the retina. Posterior to the iris, the suspensory ligaments of the lens attach to folds called the ciliary processes. These fibers position the lens so light passing through the pupil goes through the center of the lens. Responsible for producing aqueous humor and for changing the shape of the lens.



9. **Lens:** Disc-shaped structure approximately 9 mm in diameter and 4 mm thick, containing transparent crystalline matter, suspended immediately behind the iris and anterior to the vitreous by suspensory ligaments that connect it to the wedge-shaped ciliary body. The lens is highly elastic and contraction or relaxation of the ciliary body changes its thickness and shape, thereby permitting images from varied distances to be focused on the retina.

Its shape changes for near and far vision, becoming flatter for far vision. The ability to change shape deteriorates with age explaining need for reading glasses after age 40.

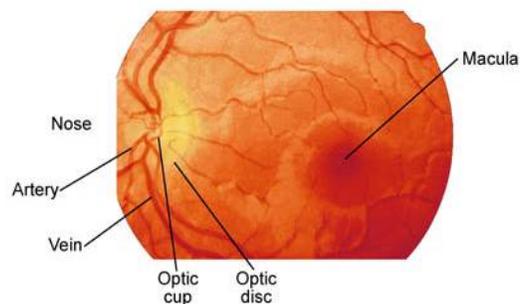


10. **Choroid:** Layer of vascular channels that separates the fibrous and neural tunics posterior to the ciliary body. This layer delivers oxygen and nutrients to the retina.

11. **Retina:** Consists of a thin outer pigment layer and a thick inner layer of neural retina. The pigment layer absorbs light after it passes through the receptor layer. The neural retina contains photoreceptors that respond to light, supporting cells and neurons that perform preliminary processing and integration of visual information, and blood vessels supplying tissues that line the posterior cavity. The sensory network transforms light impulses into electrical impulses via *rods* and *cones*. Rods do not discriminate among colors. They enable us to see in poor lighting conditions. Cones provide color vision. They give sharper, clearer images but require more intense light.

- a. **Macula lutea** (or yellow spot): Avascular region where the visual image arrives after passing through the cornea and lens. The area of greatest concentration of cones is located in the central portion, called the **fovea** (shallow depression). All color and sharpest vision better than 20:200 is located here.

Normal Retina - Left Eye
 (Photo courtesy of Dr. David Rapaport)



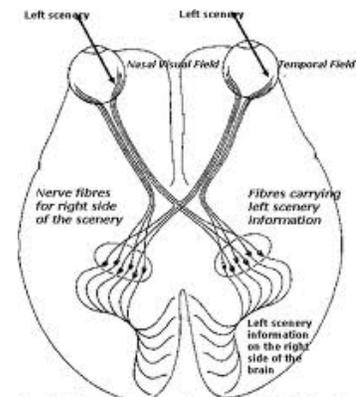
- b. **Optic disc:** A circular region just medial to the fovea. It measures 1.5 mm across and becomes the origin of the optic nerve, which is formed from the convergence of axons from 1 million retinal ganglion cells. The central depression or cup usually averages $\frac{1}{3}$ of the disc diameter. Blood vessels that supply the retina (central retinal artery and vein) pass through the center of the optic nerve and emerge on the surface of the optic disc (Martini et al, 2008). The disc has no photoreceptors or other retinal structures. Light striking this area is not perceived, so it is commonly called the **blind spot**.

- c. **Optic nerve** - Sensory impulses are transmitted through the optic nerve (CN II) to the occipital lobe for conscious interpretation. Injury to the optic nerve and retina are most responsible for permanent visual loss in cases of trauma.

d. **Refracting system:**

As light passes through the cornea and the lens, it is redirected (refracted) by the lens, so that the light focuses in the area of the rods and cones on the retina. From there, light is converted to electrical impulses, which are carried by the optic nerve to the occipital lobe for interpretation as an image.

- e. **Binocular vision:** As "light" signals are transmitted via the optic nerve on their way to the occipital lobe, they pass through the optic chiasm. Here, roughly half the nerve fibers from each eye cross over to the opposite side of the brain. Vision centers on each side of the occipital lobe interpret information separately, then combine that information so the brain "sees" one integrated image, creating the perception of depth and 3-dimensions. This merging of two images is known as binocular vision.



Visual Cortex. Information from the eyes is in bands, matching bands of scenery. Details from both eyes are matched in each band, allowing accurate integration.

- f. The **occipital lobe** houses the vision centers of the brain. The occipital lobe is responsible for processing of visual information – light/dark, shape, shadow, motion, color, and interpretation.

12. **Chambers of the eye:** The eye is hollow. The ciliary body and lens divide the interior into two **cavities**:

a. **Anterior cavity**

(1) **Divided into two chambers**

- (a) **Anterior chamber:** Extends from the cornea to the iris
(b) **Posterior chamber:** Extends from the iris to the ciliary body and lens.

- (2) **Contents:** Filled with **aqueous humor** produced by the ciliary processes and secreted into the posterior chamber. The fluid circulates within the anterior cavity, passing from the posterior to the anterior chamber through the pupil. It leaves the anterior chamber near the edge of the iris through the canal of Schlemm that returns the fluid to the venous system. The anterior cavity holds about 125 microliters of fluid. Interference with secretion, circulation, or absorption can change the pressure within the eye. Glaucoma is caused by an elevation in ocular pressure and can produce blindness by distorting the retina and the optic disc.

- b. **Posterior cavity** or *vitreous chamber* contains the gelatinous vitreous body. Helps to maintain the shape of the eye and holds the retina against the choroid.

13. **Vascular supply**

- a. **Arterial:** The ophthalmic (central retinal) artery is the main vessel that supplies blood to the globe and orbit and is the first branch off of the internal carotid after it enters the cranial cavity. Emboli from the internal carotid artery can enter the ophthalmic artery, producing visual problems. Facial and maxillary arteries feed the lower lid, medial canthus and inferior orbit.

- b. **Venous:** Blood returns through the inferior and superior ophthalmic veins. Venous draining through the cavernous sinus is an important route for the intracranial spread of infection. There are no lymphatics in the orbit, but the eye lid has a rich lymphatic system.

IV. **Assessment and management of ocular trauma**

Many patients presenting with ocular trauma have other associated trauma to the head and neck with facial and skull injuries that may delay recognition of ocular damage.

A. **Chief complaint/history of present illness:** Because the nature of eye emergencies ranges from minor to severe, history taking may be done simultaneously with the eye exam. Whenever time permits, obtaining an accurate, detailed history can aid in the rapid treatment and evaluation of the patient. Important details to include:

1. **Nature of complaint:** traumatic vs. non-traumatic
 - a. **Non-traumatic:** Usually divided into complaints of vision, appearance or sensation. Obtain details regarding the rapidity of onset, duration, intermittence and frequency. Note associated symptoms.
 - b. **Traumatic:** Blunt, penetrating, sharp or explosive; thermal, UV; time and course of events. Ask about any change in vision, and if so, was it immediate, sudden or gradual? If chemical burn, ask if it was toxic, acid or alkali. Determine any treatment rendered prior to arrival.
2. **HPI - Detailed description of symptoms:** Obtain OPQRST details about onset, provocation (what object or item caused the injury - magnetic?)/palliation, quality, region/recurrence, severity, and date and time of injury.
 - a. Visual changes from baseline: Blurred, double, or deficits in acuity or visual fields
 - b. Symptoms in one or both eyes?
 - c. **Diplopia (double vision)**
 - (1) Diplopia may indicate trauma to the globe with muscle entrapment or nerve deficit, peripheral or central
 - (2) Monocular diplopia usually indicates a refractive error in the eye itself
 - (3) Binocular diplopia, present only when both eyes are open, is the result of a deficiency in the movement of the eye
 - d. Redness
 - e. Tearing, drainage
 - f. Pain
 - g. Note associated complaints/symptoms other than decreased vision

B. **SAMPLE History**

1. **Allergies**
2. **Medications:** Regular use of eye medications or over-the-counter remedies including drops or ointments. Use of anticoagulants or aspirin. Include licit and illicit drug use, as they can affect pupillary size and reactivity.
3. **Past ocular history**
 - a. In self or family: cataracts, glaucoma. Any ocular disease or visual loss prior to trauma?
 - (1) Blurred vision that does not improve with blinking
 - (2) Diplopia
 - (3) Sectorial visual loss
 - (4) Spots before eyes, curtain over the visual field?
 - (5) Halos or rings around lights
 - (6) Photophobia

- b. **Baseline visual acuity**; ever worn glasses or contact lenses? How long?
If contacts are worn: hard, soft, extended wear? For reading, driving, distance?
 - c. History of ocular pain
 - d. Eye injury or surgery? When?
 - e. Excessive tearing, crusting, discharge?
 - f. Burning (inflammation)
 - g. Relevant occupational hazards; dust, chemicals, metalwork
 - (1) Use of protective devices
 - (2) Use of safety glasses, if traumatic injury
4. **Past medical history in self or family**
- a. Heart disease, hypertension
 - b. Diabetes
 - c. Sickle cell disease
 - d. Liver disease
 - e. Vascular disorders
5. Last oral intake; tetanus
6. **Events surrounding the incident**: Details regarding probable size, velocity, and chemical constituency of pellets or projectiles; determine treatment rendered prior to arrival.
- C. **Physical exam**
1. **Remove and store contact lenses**: Lenses must be removed as part of the trauma assessment/management, unless doing so would create risk of further injury or irritation.
 - a. **Hard contact lenses**: Remove with suction cup lens remover. Place in normal saline and label container left and right.
 - b. **Soft contact lenses**: Remove by pinching the lens together and pulling away from eye. Place immediately in 0.9 NS and label containers L and R.
 2. **Signs and symptoms**
 - a. Pain: duration, intensity, location
 - b. Visual changes from normal: Blurred, double, partial, no vision. Always check both eyes.
 - c. Anatomic disruptions: Look for obvious deformities, lacerations
 - d. Functional deficits: Is movement of globe or lid impaired?
 3. **External structures** - Inspect from top to bottom; medial to lateral canthus
 - a. **Orbit and periorbital structures**: Often, one needs only to visually inspect the patient's face. However, bony palpation of the orbital rim can detect deformities and pain. Orbital rim should be smooth, without deformities or irregularities. Inspect/palpate for the following:
 - (1) Ecchymoses, laceration
 - (2) Periorbital edema, masses, asymmetry of tissue
 - (3) Tenderness and crepitus
 - (4) Position of the eye within the orbit and in comparison with the opposite side.
 - (a) **Enophthalmos**: Relative recession (backward or downward displacement) of the globe into the bony orbit. Frequently seen after orbital fracture.
 - (b) **Exophthalmos** (eye bulging out)

- (c) The pupils should be on same horizontal plane. If one eye is lower than the other suspect orbital floor fracture. Eyes should be in line with top of ear. Is gaze focused on you?
 - (5) Globe intact? If not, what type of fluid is present? What is the shape of the eye?
 - (6) Eyebrow size and extension
 - b. **Lids: Examine closed and open**
 - (1) Symmetry
 - (2) Ecchymoses
 - (3) Laceration/lesion: Can be associated with penetrating trauma to the globe. Look for prolapse of orbital fat.
 - (4) Edema, inflammation
 - (5) Ability to spontaneously lift and close lid to cover the superior limbus of the iris by 1 mm - 3 mm
 - (a) **Ptosis:** If the lid cannot open or lift up and covers more of the iris than the other side or extends over the iris, this indicates a congenital or acquired weakness of the levator (plus Muller's) muscle or paresis of a branch of the third cranial nerve.
 - (b) If the lid cannot close, suspect trauma to CN VII which innervates the orbicularis muscle. Ex.: Bell's palsy.
 - (6) Surface growths or internal masses; never palpate an injured eye
 - c. **Lashes and sebaceous glands**
 - (1) Crusting, scaling
 - (2) Hair loss
 - (3) Pus, blood, CSF from puncta
4. **Anterior segment:** This examination includes the conjunctiva, cornea, anterior chamber, iris and lens
- a. **Conjunctiva** - Use a penlight. **Inspect for the following:**
 - (1) Degree and depth of redness/injection
 - (2) Foreign body, embedded material
 - (3) Laceration
 - (4) Chemosis or hemorrhage
 - (5) Rust ring; black defect
 - (6) Pus
 - b. **Sclera**
 - (1) Should be white. Abnormal colors: jaundiced, bluish (Marfan's syndrome), injected
 - (2) Wounds
 - (3) Black defect
 - c. **Cornea;** should be smooth, round, clear, glistening and free of lesions. Inspect for the following:
 - (1) Blood
 - (2) Clouding/opacity; could indicate acute glaucoma, edema from trauma, foreign body in the anterior chamber, or infection
 - (3) Abnormal pigment
 - (4) Irregular light reflection if irregular in size or shape
 - (5) Abrasion/laceration
 - (6) Foreign body

- d. **Anterior chamber:** should be clear and deep with the iris well separated from the posterior corneal surface. If blood (hyphema) or white cells (hypopyon) are present, serious injury has occurred.
5. **Iris:** Should be flat and smooth. In trauma, may show tears and holes. These may cause pupil irregularities. If torn, suspect ruptured globe.
6. **Lens:** Should be transparent. Not uncommon to have post-traumatic cataracts develop which if often visible on penlight exam. In severe trauma, lens may dislocate to float free in the vitreous or settle on the retina.
7. **Cranial nerve assessment**
 - a. **CN II (Optic nerve)**
 - (1) **Visual acuity:** Never delay eye irrigation for chemical burns in order to obtain visual acuity. This is vital from both the medical and legal standpoint.
 - (a) Test each eye separately; shield eye that is not being tested
 - (b) **Note their best (corrected) vision.** If they normally wear eyeglasses or contact lenses, have them wear them during the assessment.
 - (i) Can read print (near card at 14 inches)
 - (ii) If patient cannot read, see if they can count the number of fingers being held up at 12 inches.
 - (iii) If they can't count fingers, see if they can perceive hand motion 1 ft. in front of the eye.
 - (iv) If unable to see hand motion, document if they can perceive light with or without the ability to determine the direction from which it is projecting.
 - (v) No light perception (NLP)
 - (c) **Differential diagnosis of post-traumatic loss of vision**
 - (i) Lid swelling; blood or foreign material covering cornea; corneal damage
 - (ii) Hyphema; vitreous hemorrhage
 - (iii) Traumatic cataract; injury to the lens
 - (iv) Central retinal artery or vein occlusion (from markedly increased orbital pressure or embolus)
 - (v) Traumatic retinal edema and hemorrhages of retina from direct or contrecoup blow to head
 - (vi) Retinal detachment
 - (vii) Avulsion of optic nerve by trauma of lateral orbital wall or contrecoup blow to head
 - (viii) Indirect trauma to optic nerves and/or chiasm (traumatic optic neuritis)
 - (ix) Intracranial interruption of visual pathways (hemorrhage/ foreign body)
 - (x) Cortical blindness from hematoma, ischemia, or anoxia (patient may be unaware of blindness)
 - (xi) Acute (angle-closure) glaucoma precipitated by emotional trauma of recent trauma
 - (xii) Hysteria
 - (xiii) Malingering
 - (xiv) Dislocation of intraocular lens
 - (xv) Dislocation or loss of contact lens

- (2) **Visual fields (by confrontation):** Have the patient cover one eye and focus on your face. Raise one or two fingers in the upper and lower right and left visual fields and ask the patient to identify the number of fingers you have presented. Repeat the test for the other eye. The visual deficits will depend on whether there is disruption to one of the optic nerve branches before or after the optic chiasm or directly in the chiasm.
- (3) A fundoscopic exam using an ophthalmoscope is usually not performed in the field. The purpose of the exam is to inspect the major retinal vessels to the optic disc assessing for constriction, dilation, thickening, sheathing, or bright intravascular objects (atheromatous emboli). The disc is examined for color, contour, and margins. Swelling or bulging of the optic disc is called papilledema and indicates an increase in intracranial pressure.

b. **CN III (Oculomotor): Pupil size, shape (deformity or defect), equality**

- (1) Have patient look at a distant object. This prevents pupillary constriction that occurs when one looks at a near object (accommodation). Note the size and shape of each pupil. Compare one side to the other for symmetry.
- (2) Pupils should be round, symmetric, midpoint, and equal in size. Previous ocular surgery, injuries and medication can affect the pupils, so obtain an accurate history.
- (3) **Causes of pupil asymmetry**
 - (a) About 15% of the population normally has pupils of different sizes (*anisocoria*). If this is physiologic, the pupils should remain asymmetric in both bright and dim light.
 - (b) **Horner's syndrome:** Rare disorder that occurs when nerves serving the face and eyes are damaged. It is usually a result of a stroke, a tumor or an injury to the spinal cord. Symptoms include a ptosis, a small pupil (asymmetry is accentuated in dim light), and decreased sweating on the affected side of the face. Prehospital treatment is supportive.
 - (c) **Adie's pupil: (Adie's syndrome, Holmes-Adie syndrome, HAS):** Thought to be the result of a virus that causes inflammation of the neurons in the autonomic nervous system (ANS) and the area of the brain that controls eye movement. HAS affects the pupil of one eye, leaving that pupil larger than normal and slow to constrict in bright light. Patients w/ HAS may also have absent deep tendon reflexes (usually Achilles tendon). Pts may also sweat excessively (ANS). The disorder is most often seen in young women.
 - (d) Traumatic mydriasis (prolonged abnormal pupil dilation) or miosis (constricted pupil) from direct blow
 - (e) Rupture of iris sphincter
 - (f) Unilateral use of topical drugs
 - (g) Intraorbital trauma
 - (h) Acute intraocular inflammation with spasm or no tone
 - (i) Intracranial third-nerve palsy
 - (j) Iritis
 - (k) Unilateral blindness
 - (l) Stroke; pontine lesions

(4) **Pupil reactivity to light**

- (a) **Direct and consensual.** Shine a bright light source into one eye at a time from the side. Do not hold the penlight directly within the visual axis because of the accommodation reflex. Both pupils should briskly constrict and remain constricted as long as the light stimulus is present. The pupil into which the light is beamed constricts with a direct response and the opposite pupil constricts in a consensual response.
- (b) **Afferent defect** (*nice to know*): When a bright light is directed at a healthy eye, both pupils should constrict. As the light is quickly swung over to the injured eye, both pupils will widen. As light is returned to the healthy eye, both pupils will constrict. Can be performed even if only one pupil is assessed. In ambient light, if the optic nerve of one eye has been completely transected, the pupils should be equal because the consensual response of the damaged eye to the good eye will result in equal pupils. Therefore, the swinging light test is important.

c. **CN III, IV (Trochlear), IV (Abducens): Extraocular movements (EOMs):**

Assess ocular motility by having patient fix their gaze on a penlight or other object which is moved in each of the cardinal directions of gaze to evaluate nerve and muscle function. Eyes should move an equal amount in each gaze direction. Assess for range of movement, symmetry, dysconjugate gaze or pain. Many things affect eye mobility. Common causes include orbital edema, muscle entrapment and cranial nerve damage.

- (1) Conjugate gaze: Eyes move together
- (2) Dysconjugate gaze: Eyes do not move together
- (3) Gaze palsy: One eye does not move symmetrically or looks to one side
- (4) **Strabismus** (crossed eye): One or both eyes turn in, out, up or down. Usually not an acute condition-usually chronic, due to poor eye muscle control.



When an eye turns in, the child has **esotropia**. This is sometimes called being "cross-eyed."

II. **Chemical injury (burns)**

A. Apart from history, the diagnosis of chemical burn is usually based on the presence of swollen eyelids with marked conjunctival swelling and redness

B. **Etiology**

1. **Acids:** When acids come in contact with the eye, tissue proteins are released and form a protective barrier to prevent further penetration of the eye, so these types of injuries are usually limited to the external surface of the globe. Generally less serious than alkali burns because they do not cause progressive destruction of ocular tissues or collagen swelling (ex.: exploding car battery).



2. **Alkalis:** Cause breakdown of the fatty acids in the epithelial cell membranes (emulsification) causing collagen denaturation. They destroy the surface tissues, affecting the eyelid, conjunctiva and mucous secreting cells. Once the epithelium is damaged, the chemical rapidly penetrates the cornea and anterior chamber. When the pH is 8.0 or higher an alkaline chemical keratitis is present. If pH > 11.5 there is intraocular penetration. (Ex.: drain cleaners containing lye, lime, ammonia, and airbag propellant gasses such as sodium hydroxide. The higher the pH the worse the injury.)

C. **Morbidity and prognosis for eye**

1. Depends on the pH of the agent, chemical concentration, and the duration of ocular exposure. They are often very serious, because even after apparent removal of the agent, lodgment of tiny particles under the lids may continue and cause progressive damage to the eye
2. It may take 48-72 hours to make an accurate assessment of burn damage. For alkali burns, the degree of corneal opacification (clouding) and perilimbal blanching correlate with prognosis.
3. Corneal clouding occurs due to disruption of the regularly spaced collagen network by direct chemical damage, reactive edema, and damage from lysosome enzymes. Burns at the limbal region are the most unfavorable, as they produce vascular damage with extensive thrombosis and eventually ischemic necrosis.

D. **Clinical presentation: acute phase**

1. Severe pain and diminished or blurred vision
2. Conjunctival chemosis and injection
3. Possible limbal blanching
4. Localized edema
5. Denuding of corneal epithelium (corneal grafts don't take)
6. Loss of corneal transparency

E. **EMS care: Do not delay treatment** while trying to determine nature of the fluid

1. Rapidly remove contact lenses
2. Rapid visual acuity for light perception only while preparing to irrigate
3. Instill tetracaine drops if severe pain/spasm. Research by a group of New Mexico physicians suggests that a patient's response to local anesthesia may predict the complexity of a corneal lesion. Those that receive significant pain relief probably have an uncomplicated corneal lesion. Patients with a diagnosis of conjunctivitis, iritis, corneal ulcer, hyphema, glaucoma, or subconjunctival hemorrhage typically had less pain initially and got little relief from tetracaine.
4. **Immediate and profuse eye irrigation with the most immediately available neutral solution.** In the field, this is usually normal saline. Aim the stream from the inner to outer canthus. Don't attempt to neutralize the chemical agent. Evert the eyelids to flush the cul-de-sac. Continue into the hospital. Irrigation should continue until the pH has returned to normal (7.3 to 7.7). Repeat pH testing should be done 30 minutes after irrigation is stopped. If rising, suspect trapped particulate matter that is releasing additional chemicals and irrigation should be repeated.

F. **Morgan Lens** (New in National Education Standards – will not use in NWC EMSS)

1. Device used to provide eye irrigation for removal of chemical and physical irritants. It is a molded scleral lens w/ directions fins and adaptor tubing that attaches to IV tubing. The "fins" are positioned underneath the eyelids and the patient can then close his/her eyes around the lens. It allows for continuous hands-free irrigation, w/o the need to hold the eyelids open.
2. Indications:
 - a. Need for continuous eye irrigation
 - b. Chemical splash/burns to eye
 - c. Non-embedded foreign bodies
 - d. FB sensation w/ no visible foreign body
3. Contraindications
 - a. Penetrating eye injuries
 - b. Suspected or actual rupture of the globe



III. **Penetrating/perforating injuries** (open globe): Major cause of traumatic visual loss due to damage of intraocular structures caused by the force required to rupture the globe. Require immediate careful attention and prompt surgical repair at the hospital to prevent possible loss of the eye.

A. **Etiology**

1. Children: running with sharp objects in hand or unsafe playground games or toys
2. Adults: Acts of violence, injuries in home and/or the workplace

B. **Penetrating injuries** are those that cause disruption of the outer coats of the eye without interrupting the anatomic continuity of the whole eye, thus preventing prolapse of internal ocular contents. The most obvious would be a retained FB in the surface of the eye.

C. **Perforating injuries** are those that result in full penetration of the sclera or cornea. Such wounds may or may not be associated with prolapse of internal structures.

D. **Clinical assessment/presentation**

1. Pain
2. Change in pupil shape (tear-drop: iris spasm pulls pupil to site of injury)
3. Unequal pupils; may be dilated and nonreactive on affected side
4. Black defect (choroid prolapse into defect)
5. Injected sclera, subconjunctival hemorrhage
6. Leakage or prolapse of vitreous humor
7. Decreased visual acuity
8. Hyphema
9. Iris prolapse
10. Dislocated lens, traumatic cataract
11. Do not pull swollen or injured lids apart with fingers to prevent extrusion of the eye

E. **Emergency management**

1. Never remove an impaled FB; stabilize object by wrapping it with roller gauze until it would fill inside of cup; take bottom out of the cup, place over object, tape cup in place and transport as a time-sensitive patient.
2. **DO NOT** manipulate the eye, instill eye drops, or apply ointments to an open globe
3. Do not attempt to remove any blood clots, foreign bodies or tissue from the eye
4. **Eye shields:** An eye shield should be applied when pressure on the globe might result in loss of vitreous or aqueous humor. A metal shield or cup should rest on the bone of the brow and the cheek; it should not exert pressure on the eye. The patient must not be able to touch or rub the eyelids.
5. Unilateral vs. bilateral shielding: Based on local protocol. Based on recommendations from the University of Illinois Eye Trauma Center, the NWC EMSS does not shield an uninjured eye.
6. Analgesics and sedatives may be needed
7. Don't let patient bend, stoop, strain, or move his or her head suddenly
8. Keep patient NPO
9. Reduce patient's anxiety through therapeutic communication or medication

IV. **Hyphema**

A. **Definition:** Accumulation of blood in the anterior chamber

B. **Etiology:** Usually caused by severe blunt, and less often, perforating trauma to the eye resulting in the rupture of blood vessels in the anterior chamber. Bleeding occurs from the angle where the iris meets the cornea leading to hemorrhage. Bleeding is self-limited as vascular pressures and intraocular pressures equilibrate. In rare instances, hyphema may occur spontaneously as a complication of an ocular or systemic disorder.



C. **Demographics**

1. Incidence: 17-20/1000 population/year
2. M:F 3:1
3. 70% < 20 y/o
4. 60% sports related

D. **Frequency and severity (graded by hospital)**

1. 60% Grade 1: Small; $\frac{1}{3}$ of the anterior chamber (AC)
2. 20% Grade 2: Moderate; $\frac{1}{3} - \frac{1}{2}$ AC
3. 20% Grade 3: Severe; Greater than $\frac{1}{2}$ to near total AC
4. Grade 4: Total or "eight-ball" hyphema

E. **Clinical presentation**

1. Decrease in visual acuity if blood covers the pupil (25%); patient may report seeing red, moving spots that seem to drift in front of the eye
2. Note color: red indicates new bleed, brown is characteristic of older blood
3. Gravity and clotting mechanisms will eventually cause the blood to settle inferiorly, giving the hyphema a crescent shape
4. Pain, nausea, vomiting; may indicate elevation of intraocular pressure causing angle recession and acute glaucoma

F. **Clinical significance:** Suspicion of accompanying globe rupture

G. **Examination**

1. **History:** Trauma, bleeding, use of anticoagulants
2. ABCs, mental status
3. Visual acuity
4. Rule out FB, open globe, laceration
5. Evaluate iris; active hemorrhage, tears
6. Height of hyphema
7. **Emergency management:** Aimed at limiting acute complications (Acute: ocular hypertension, secondary hemorrhage)
 - a. Sit up 30° - 45° to facilitate settling of the hyphema into the inferior portion of the anterior chamber and away from the visual axis
 - b. Place shield over involved eye to help prevent rebleeding
 - c. No reading should be permitted
 - d. Sedate the patient if overly anxious
 - e. Non-aspirin containing analgesics
8. Major complication: Rebleeding 3-5 days later

V. **Lid injuries**

- A. **Etiology:** Blunt or sharp trauma to the orbital region may lead to significant functional and structural abnormalities.
- B. Types of lid injuries: lateral vs. medial to punctum
- C. **History:** Determine the time, circumstances, and type of injury, assess the possibility of FB
- D. **Clinical significance:** These may be associated with serious ocular injuries not apparent at first examination. Injuries to the lids can be serious because these structures protect the eyes and keep them moist, acting like windshield wipers to wash away foreign matter. An injured eyelid can lose its ability to cover the eye adequately, resulting in drying of the eye, infection or clouding of the normally clear cornea.
- E. **Clinical presentation:** edema, ecchymosis, asymmetrical eyelid, exposed eyeball, bleeding, and possible laceration to tear ducts.

F. **Assessment**

1. Avoid vertical or horizontal traction near the lid margin to prevent late ectropion (sagging lid)
2. Note any abnormality of eyelid integrity or contour
3. Be alert for injury to the margins, canthal tendons, lacrimal drainage system, deeper lid structures or the globe. These require special repair.
4. Document ptosis
5. Describe the amount of edema, ecchymosis, and erythema (redness and swelling)
6. Avulsion injuries may result in loss of lid skin or deeper tissue. Estimate and document the size and extent of the lesions.
7. Assess visual acuity

G. **Emergency management**

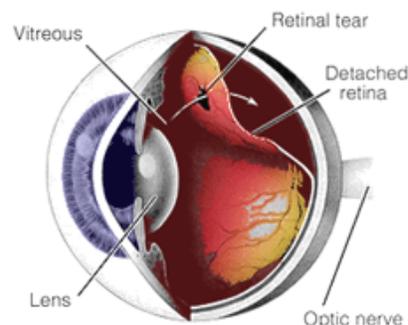
1. Cover wound with saline-moistened sterile dressing
2. Apply cold packs to decrease swelling and pain
3. Bring in all avulsed tissue, even if the viability is questioned

VI. **Retinal detachment:** Separation of the retina from its source of blood supply via tear, fold or rent. Subretinal fluid accumulates under the neurosensory layer. Minimal to moderate trauma may cause retinal detachment.

A. Etiology: 15% due to trauma

B. **Clinical presentation**

1. Painless visual field deficit or decrease in vision. May be described as a darkening/haziness or a curtain being drawn over the visual field.
2. Photopsia (**flashes of light or sparks**); spider webs
3. "**Floaters**" or black dots



C. **Emergency management**

1. Keep patient quiet and supine with both eyes patched
2. Retinal attachment can only be accomplished by surgery which is successful in about 80% of the cases

VII. **Thermal burn**

A. **Etiology:** Flash burn, fiery explosion, walks into something hot, etc. Almost always associated with facial burns. Even severely burned patients may avoid direct injury to the cornea and conjunctiva due to protection by the eyelids. Burns due to toxic chemicals, hot liquids, and molten metal behave much like alkaline burns. Iron has a higher melting point (1200° C) and causes more damage than other metals (lead, tin, zinc, melting point below 1000° C). Arc welding injuries common without protective eyewear.

B. **Clinical exam:** May require local anesthetic

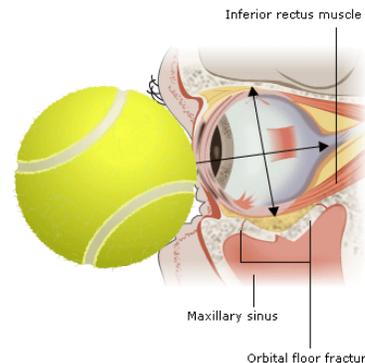
1. Visual acuity
2. Pupillary exam
3. External exam: lid spasm, edema, orbital cellulitis
4. In explosions: must R/O perforating injuries, FB
5. Cornea will slough and regenerate in 4-5 days in children; 3-4 wks in adults

C. **Treatment**

1. Cool burn; apply moist dressings to eyelids; dry dressings to other burned areas.
2. Bandage with contact lens in place. Note: the contact lens will not remain moist if lids are not functioning properly, may need to remoisten dressings.

VIII. **Orbital (blow-out) fracture**

A. **Etiology:** When blunt force is applied to the eye, the orbital contents are pushed posteriorly and intraorbital pressure suddenly rises. The orbital floor is also the roof of the maxillary sinus. It is thin and may fracture. Immediately at impact, the edges of the fracture may separate briefly then come back together. At the moment of separation, the contents of the lower orbit may prolapse into the fracture space and become trapped as the bone comes together. If the inferior rectus muscle is entrapped, the patient cannot look upward. Suspect orbital blowout fracture with adjacent facial bone fracture.



B. **Clinical presentation:** Depends on the size and location within the orbital cavity

1. Periorbital pain, pain on upward gaze, point tenderness along orbital ridge, swelling, ecchymosis, crepitus if sinus cavity is involved
2. **Abnormal EOMs:** Eyes may be aligned on downward gaze, but attempted elevation of the eyes causes double vision (diplopia) with vertical separation of images.
3. **Paralysis of upward or vertical gaze** indicates possible EOM entrapment.
4. Visual changes may result from a hematoma forming around the optic nerve or retinal damage
5. If significant orbital fat is entrapped, the eye may appear sunken (enophthalmos) if not obscured by local swelling
6. Subconjunctival air or hemorrhage; chemosis
7. Hyperesthesia or paresthesias over the infraorbital nerve distribution depending on degree of nerve stretching or tearing. Ask the patient if his or her cheek or upper lip is numb on the side of injury.
8. Facial asymmetry
9. Rim fracture may be palpable; a fracture posterior to the rim may not be palpable
10. Nosebleed (epistaxis) on the same side



C. **Emergency management**

1. Caution patient not to blow their nose
2. Apply cold pack if no globe rupture
3. May be asked to shield the eye to protect it from further damage. The NWC MD does not recommend bilateral shielding or bandaging.
4. Fentanyl as needed

IX. **Corneal/conjunctival foreign bodies**

A. **Etiology:** Typical FBs: dirt, dust, glass or cinders, or fragments that fly into the eye when working without protective goggles. If metal, identify the type of metal if possible. Iron and copper common. BB pellets etc. also common. Most FBs do not penetrate and result in ocular irritation. However, view them as a possible indicator of concurrent intraocular or intraorbital FB. This is especially important in the setting of grinding or hammering metal upon metal in the absence of protective eyewear.

B. **History:** Question patient about the use of safety goggles, the nature of the FB, and if they were in an environment where metal was striking metal

C. **Clinical assessment/presentation:** Carefully examine the eyebrows and eyelashes and remove any debris that could fall into the eye. Inspect conjunctiva and evert the lids to search for additional FB. If vertical linear abrasions are seen suspect occult FB under the lid. Observe unaffected eye first for comparison.

1. Obtain visual acuity; ask if vision is blurred or if FB is in the visual axis
2. Pain, foreign body sensation; may worsen with blinking
3. Photophobia
4. Always suspect penetration in higher velocity injuries e.g., hammer and chisel
5. Key feature is the finding of the FB, a rust ring, or both
6. Conjunctival injection, mild anterior chamber reaction
7. Eyelid edema
8. There may be a small white infiltrate around the FB if it has been in the cornea for greater than 24 hours.

D. **Emergency management**

1. Topical anesthetics: Tetracaine 0.5% 1 gtt
2. **Conjunctival FB:** Easily removed with gentle irrigation with NS or moistened cotton-tipped applicator.
3. **Corneal FB:** Requires removal under the slit lamp at the hospital
4. Check for clinically obvious **corneal abrasion**
5. Systemic analgesics; may need Fentanyl if iritis is severe

X. **Cyanoacrylate (Krazy Glue):** If patient has gotten an adhesive like Krazy glue on their lids or into their eyes, immediately irrigate with warm water for at least 15 minutes. Do not pry lids loose or try to remove residual glue in the field. Acetone or ethanol solutions should **not** be used in the eye. Mineral oil can be applied to the eyelid if available. Corneal abrasions are likely once the glue is removed from the eye.

XI. **Corneal abrasions**

A. **Etiology:** Scratch or tear in the corneal epithelium resulting from an impact, contact lens, and/or exposure to ultraviolet light. FBs can scratch the cornea at various depths. All of these objects may be contaminated and the possibility of infection should always be considered. Patients with contact lenses are at particular risk for gram negative infections such as *pseudomonas* and hospital treatment differs from a routine abrasion.

B. **Clinical presentation:** tetracaine aids in the exam if no signs of penetration

1. Pain (can be severe) and FB sensation that is worsened by blinking
2. Copious tearing (epiphora)
3. Possible decrease in visual acuity if over the pupil
4. Irregular corneal light reflex
5. Diffuse conjunctival redness
6. Photophobia
7. Lid spasm (blepharospasm)

C. **Examination**

1. A corneal abrasion may be associated with serious ocular injury. Vision should be checked and recorded.
2. Evaluate patient for retained foreign body by everting the eyelid. If the cornea has vertical linear abrasions, a FB is likely.
3. Examine the anterior chamber for the presence of a hyphema that may result from blunt trauma and may require different treatment.

D. **Emergency management**

1. Elevate head of stretcher 45 degrees; give patient something to use to absorb tears
2. Instill tetracaine per SOP
3. **Non-contact lens wearer:** May be asked to apply a pressure patch to help alleviate pain and limit eyelid movement (use is controversial and not required). See procedure manual for steps. Caution patient about loss of depth perception if patch is applied.
4. **Contact lens wearer: DO NOT PATCH** when microbial infection is a significant risk (contact lens wearers)

XII. **Subconjunctival hemorrhage**

- A. **Etiology:** Very common; caused by leaking of blood from delicate vessels between conjunctiva and sclera. Can be initiated by sneezing, coughing, or lifting heavy objects. They can also be caused by trauma to the eye or surrounding tissues.
- B. **Clinical presentation:** Blood-red eye that does not change in appearance when the patient changes position; looks terrible, but is painless. Does not change visual acuity. Non-emergency if non-traumatic origin.
- C. **Emergency management:** Usually self-limiting, spontaneously resolves in 1 to 2 weeks, however, always suspect possible ruptured globe

XIII. **U.V. radiation burns**

- A. **Etiology:** Uncommon, yet may be seen in head and neck tumors treated with radiation, those who use tanning beds with the corneas unprotected, arc welders, and repeated direct exposure to sunlight as in mountain climbers and skiers.
- B. **Clinical presentation**
 - 1. A flash exposure can cause temporary blindness from overstimulation of the retina
 - 2. Superficial punctate keratitis, corneal epithelium sloughs
 - 3. Pain, 3-6 hours after exposure
 - 4. Increased tearing, burning, eyes feel full of sand
 - 5. Superficial swelling of cornea
- C. **Emergency management**
 - 1. Patching, cold compresses
 - 2. Tetracaine eye drops to relieve pain
 - 3. 12-36 hour recovery period
 - 4. Permanent scarring is rare; long-term exposure may permanently thin the surface layer of the cornea, reducing the eye's natural defense mechanism

XIV. **Non-traumatic eye emergencies (New in the Education Standards)**

- A. **Oculomotor nerve paralysis**
 - 1. As a point of review: CN III moves the eye up, down, medially to the nose; lifts the eyelid, and changes the pupil size
 - 2. **Etiology**
 - a. Compression from edema or brain shift of the temporal lobe (uncal herniation) onto the nerve (which has its origin adjacent to the midbrain part of the brainstem) due to intracerebral hemorrhage, epidural, or subdural hematoma
 - b. Pressure into the nerve from an aneurysm arising from the posterior cerebral circulation
 - c. Cavernous sinus mass or thrombosis
 - 3. **Clinical presentation**
 - a. Dilated pupil on affected side
 - b. Ptosis
 - c. Gaze palsy to ear (as 3rd nerve is dysfunctional but CN VI is still working)
 - d. Double vision as eyes are not looking in the same direction
 - 4. **Emergency management:**
 - a. Thorough SAMPLE history and neuro exam
 - b. Consider time-sensitive etiology
 - c. Supportive care/ABCs

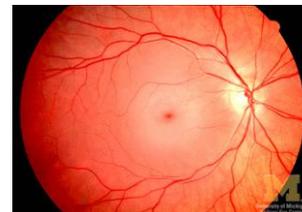
B. **Central retinal artery occlusion (CRAO): true ocular emergency**

1. Blockage of the artery by a thrombus or embolus. The retina is completely without blood as long as the artery is occluded and visual receptors in the retina will degenerate within 30-60 minutes if blood flow is not restored.

2. **Etiology**

a. Emboli (20%)

- (1) Most common = cholesterol (Hollenhorst plaque)
- (2) Heart: diseased cardiac valves; AF
- (3) Carotid: atheromatous plaques
- (4) Young patient: cardiac myxoma
- (5) Fat from bone fracture
- (6) Exogenous; i.e., IV drug use



b. Sickle cell disease

3. **Clinical presentation**

- a. Sudden, painless, nontraumatic unilateral loss of vision without apparent lesion of the eye, usually in an older person with atherosclerosis.
- b. Dilated pupil
- c. Absent direct light response; consensual light response intact

4. **Emergency management:** if treatment is not successful within thirty minutes the outcome will be marginal and permanent vision loss is likely if symptoms persist over 120 minutes. Despite the very high probability of poor outcome, treatment may be attempted up to 48 hours after onset.

- a. Supine position allows better circulation to the head and dilates retinal arteries: may increase pressure across embolus
- b. Gentle digital massage of globe over a closed eyelid for 10 seconds on, 10 seconds off during transport. Apply cardiac monitor as globe pressure may precipitate a Vagal response. Alternating massage changes intraocular pressure and may allow an embolus to flow slightly downstream to a smaller vessel and allow some vision to return.
- c. OLMC: NTG 0.4 mg SL to dilate vessels

C. **Acute elevation of intraocular pressure: Glaucoma**

1. **Definition:** Aqueous humor collects in the anterior chamber due to blockage of the drainage angle by the peripheral iris root which causes intraocular pressure (IOP) to rise with varying speed and severity. An acute elevation in IOP between 30 - 40 mmHg may damage the optic nerve and visual fields. Symptoms may appear acutely or on a chronic basis.

2. **Pathophysiology**

- a. A build up of aqueous humor in the anterior chamber causes the iris to be pushed against the drainage system, therefore obstructing outflow.
- b. Pupil dilation (mydriasis) creates a pressure differential between the posterior and anterior chambers. This pushes the peripheral iris forward, and may precipitate angle closure.
- c. Pupil constriction (miosis) can pull the peripheral iris away from the angle, often reversing the acute glaucoma. However, miotics (meds that constrict the pupil) can rotate the ciliary body (holds the lens in place) forward and may occasionally induce angle closure as well.

3. **Etiology:** Common precipitating factors
 - a. Near work - reading, sewing
 - b. Dim light; cinema, television
 - c. Fatigue
 - d. Excitement, stress, illness
 - e. Antidepressants; other mydriatics (pupil dilating agents)
 4. **Palliation**
 - a. Sleep/rest
 - b. Cessation of activity
 - c. Mild analgesics
 5. **Clinical presentation:** Closed angle (narrow-angle). When the IOP rises abruptly, the fluid pressure in the anterior chamber overcomes the corneal endothelium's ability to actively pump fluid out of the cornea. This leads to corneal edema, corneal clouding, and diminished vision. Infarction of iris tissue by the elevated pressure leads to an inflammatory response.
 - a. Pain (intense)
 - b. Blurred vision w/ decreased visual acuity
 - c. Nausea, vomiting
 - d. Vaso-vagal: bradycardia, diaphoresis
 - e. Tearing; lid edema
 - f. Anxiety, fatigue
 - g. Halos around lights
 - h. Mid-dilated pupil and minimally reactive
 - i. Oval pupil due to iris sphincter ischemia
 - j. Red eye with hazy or steamy appearing cornea with edema
 6. **Treatment**
 - a. Position patient supine to allow lens to fall backward
 - b. Transport for immediate medication at hospital
- D. **Periorbital and orbital cellulitis**
1. Etiology: Infection
 2. Clinical presentation: Red eye, fever, pain, lid edema. Orbital cellulitis will also be characterized by proptosis (globe displaced forward), impaired vision, impaired ocular movement and papilledema (bulging optic disc).
 3. Clinical significance: Orbital cellulitis is very serious and can have negative outcomes, including loss of vision, glaucoma, retinal damage and death.
 4. Associated findings: Encephalitis
 5. Emergency mgt: IMC; transport ASAP. At the hospital, the patient needs a CT scan of the orbit and brain, IV antibiotics and possible drainage in surgery.
- E. **Conjunctivitis:** Also referred to as "*pink eye*". Characterized by dilatation of the vessels of the conjunctiva, causing redness and swelling. This condition is highly contagious! Proper hygiene and hand washing can help prevent its spread.
1. **Viral:** Most common form, often seen in children, and associated with upper respiratory infection. Redness; tearing more prominent than in bacterial. Vision should be normal or near normal with either type if infection is limited to the conjunctiva. Treatment is supportive.
 2. **Bacterial:** Presents with purulent drainage, exudates ("crusty eyes") in the morning, and photophobia often caused by bacterial or Chlamydia infection. Severe or persistent pain suggests corneal involvement. EMS Treatment: supportive. Warm soaks to the lids can help loosen crusted lids.
 3. **Allergic:** Non-infectious form of conjunctivitis. Symptoms include severe pruritus, clear to white discharge and erythema. OTC antihistamine drops can provide relief.

XV. **Ocular conditions**

A. **Cataracts**

1. Protein that composes the lens clumps together and starts to cloud a small area of the lens. Over time, the cataract may grow larger, decreasing visual acuity
2. Possible causes: Smoking, diabetes, steroid use, aging, trauma
3. **Clinical presentation**
 - a. Cloudy or blurred vision
 - b. Problems with light; headlights seem too bright at night, glare from lamps or the sun, or a halo or haze around lights
 - c. Colors that seem faded
 - d. Double or multiple vision (goes away as the cataract grows)
4. Treatment: The most advanced area of medicine in the ancient world was ophthalmic surgery. Celsus (A.D. 14-37) left detailed descriptions of delicate cataract surgery using sophisticated needle syringes (LWW.com Insider - Dec. 2003). Most common approach in U.S. is extracapsular surgery. The front of the lens capsule is opened, the lens removed, and the posterior capsule left in place. Sound waves may be used to break up the cloudy lens so it can be removed through a narrow hollow tube. This is called *phacoemulsification* or phaco. An intraocular lens implant is inserted. However, if the person cannot have an intraocular lens because they are sensitive to the lens material, their eye structure is not suitable, or they have other eye diseases, they may wear soft contact lenses or cataract glasses.
5. Complications are rare but can include infection, bleeding, high pressure within the eye (intense pain, steamy cornea, vomiting), inflammation (pain, redness, swelling), and detachment of the retina. If a patient presents following cataract surgery with any of these symptoms, transport to the hospital. Strongly discourage a refusal.

B. **Color blindness:** Inability to see some colors normally

1. Causes: Most cases related to a genetic problem. One in 10 men have color blindness; it is rare in women.
2. Pigment in the cones of the retina is deficient/missing. If only one pigment is missing, the patient will have difficulty distinguishing red from green (most common) or blue from yellow. A person with the most severe form (achromatopsia) sees everything in shades of gray, and sees no color at all.
3. Signs and symptoms
 - a. Trouble seeing colors and color brightness
 - b. Difficulty distinguishing between shades of the same or similar colors
 - c. Pts w/ minor S&S may be unaware
4. Treatment: None
5. Implications: May prevent patient from entering careers requiring the ability to see colors accurately

C. **Near-sightedness (myopia):** Problems seeing far-away images clearly

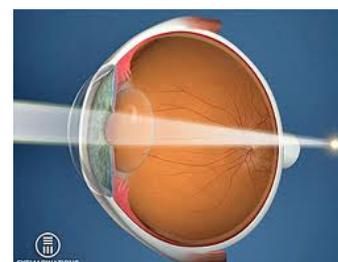
1. Myopia is an error of refraction. When light enters the eye, the light rays are not focused correctly on the retina, resulting in a blurred image.
2. Causes: Those w/ family history are more likely to develop myopia.



3. Signs and symptoms:
 - a. Objects viewed at a distance are blurred
 - b. Squinting may make far away objects appear clearer
 - c. Eyestrain
 - d. Headaches
 - e. Symptoms get worse during growth years, and slow around age 20
4. Treatment: Corrective lenses and LASIK surgery
5. Complications: Early diagnosis is important to prevent negative social and educational impacts

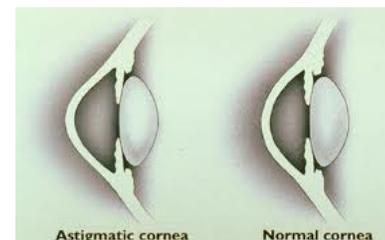
D. **Farsightedness (hyperopia):** Difficulty seeing near objects

1. Hyperopia is an error of refraction. When light enters the eye, the light rays are not focused directly on the retina, but instead, behind it.
2. Causes: May be related to a small-size eyeball or weakened ability to focus. Often present from birth. Those w/ family history of hyperopia are more likely to develop it. Common with aging.
3. Signs and symptoms:
 - a. Aching eyes
 - b. Blurred images of things viewed close-up
 - c. Crossed eyes (strabismus) in children
 - d. Eye strain
 - e. Headache while reading
4. Treatment: Corrective lenses, surgical options
5. Complications: possible risk factor for glaucoma and lazy eye



E. **Astigmatism**

1. Error of refraction, causing blurred vision and or inability to see fine details
2. Causes: changes in the length of the eye, or the shape of the lens or the cornea, creating an abnormal curvature to the eye. Abnormal curvature of the cornea makes it difficult for the eye to focus light on the retina correctly.
3. Signs and symptoms: Difficulty seeing fine details of images at any distance
4. Treatment: Corrective lenses and laser surgery
5. Complications: If uncorrected, unilateral astigmatism may result in amblyopia



F. **Amblyopia (lazy eye):** During childhood, the nerve pathway from the affected eye to the brain fails to develop because the abnormal eye sends a blurred image which is "confusing" to the brain. The brain may learn to ignore the signal from the abnormal eye.

1. Causes: Strabismus (most common cause), childhood cataracts, errors of refraction
2. Signs and symptoms:
 - a. Eyes that turn in or out
 - b. Eyes that do not appear to work together
 - c. Inability to correctly judge depth
 - d. Unilateral poor vision
3. Treatment: Correction of underlying cause; children w/ refractory errors must wear eye glasses; the normal eye is patched, forcing the brain to recognize the image from the eye w/ amblyopia. If treatment is received prior to age 5, recovery is usually complete, w/ the exception of persisting depth perception problems.
4. Complications: eye muscle problems requiring surgical correction. If treatment is delayed, permanent vision loss may result.



G. Inflammation of the eyelid

1. **Chalazion:** Small bump on the eyelid caused by a blocked oil gland
 - a. The eyelid is lined w/ tiny glands near the eyelashes that produce fluid to lubricate the eyes. When one of them becomes blocked, a chalazion develops.
 - b. Signs and symptoms:
 - (1) Eyelid tenderness
 - (2) Tearing
 - (3) Painful, swollen area on the eyelid
 - (4) Light sensitivity
 - c. Treatment: Usually heals without treatment, in approximately one month. Warm compresses may soften the hardened oils and promote drainage & healing.
 - d. Complications: If large, a chalazion may cause astigmatism as a result of pressure on the cornea (reversible w/ treatment).
2. **Hordeolum (stye):** An inflamed oil gland on the edge of the lid; appears as a red, swollen bump (like a pimple) where the lash meets the lid.
 - a. Signs and symptoms:
 - (1) Tenderness
 - (2) Tearing
 - (3) Gritty sensation
 - (4) Sensitivity to light
 - b. Treatment: Warm compresses 4 times daily. Styes may drain and heal on their own.
 - c. Complications: Styes can become chalazions if the oil gland becomes fully blocked. Infection can spread to other follicles or to the tissues around the eye (cellulitis).



- H. **Iritis:** Swelling and irritation of the iris. It may affect only one eye. Occurs mostly in young and middle aged persons and may be chronic or have an acute onset.
 1. Causes: autoimmune disorders, infection, exposure to toxins
 2. Signs and symptoms:
 - a. Blurred vision
 - b. Dark floating spots
 - c. Eye pain
 - d. Eye redness
 - e. Sensitivity to light
 3. Treatment: Dark glasses; steroid and anti-inflammatory eye drops
 4. Complications: Causes blindness if not treated. Other: cataracts; fluid collection within the retina; glaucoma; retinal detachment; vision loss. With treatment, heals in days to weeks. Tendency to relapse.
- I. **Papilledema:** Swelling of the head of the optic disc
 1. Cause: Increased ICP (associated with cerebral edema, intracranial hemorrhage, tumors, encephalitis, excessive CSF, etc.), which causes the optic nerve to swell. Swelling is usually bilateral. Requires ophthalmoscopic exam to confirm.
 2. Signs and symptoms:
 - a. Headache, made worse upon awakening, w/ coughing, breath holding, straining
 - b. Nausea and vomiting
 - c. Vision disturbances (diplopia, flickering, "graying" of vision field)
 3. Treatment: depends on the cause of the ↑ ICP
 4. Complications: If diagnosed and treated early, vision abnormalities can be avoided

XVI. **Age-related findings and conditions of vision:** Vision changes related to aging begin as early as age 40, and progress with time. Tear production diminishes. The conjunctiva thins and yellows, and the cornea yellows as well. Thus, color discrimination diminishes (esp. green, violet, and blue). With age, the size and density of the lens increases, and it becomes rigid, limiting the amount of light that passes through the eye, and decreasing the eye's ability to focus. The pupil grows smaller and less elastic, decreasing the eye's ability to dilate and constrict. As a result, older patients require longer adjusting when going from dark to light and vice versa. Impairments often limit a patient's ability to function independently, and may create safety issues. Effects of aging on vision include: reading difficulties, poor depth perception, difficulties adjusting vision in response to changes in distance, altered color perception, light sensitivity, decreased tear production, difficulty seeing well at night, and decreased visual acuity. The most common eye conditions that develop with age include the following:

A. **Macular degeneration:** Atrophy or degeneration of the macular disk

1. Age-related macular degeneration is the most common cause of legal blindness in adults, esp. the elderly. It is caused by hardening and obstruction of retinal arteries. Formation of new vasculature in the macular area interferes with central vision.
2. Two types: Dry/atrophic: atrophy of pigment epithelium; wet: formation of new blood vessels under the retina that cause leaking, hemorrhage, and scarring.
3. Signs and symptoms:
 - a. Changes in central vision (ex: black spot in the center of a page when reading)
 - b. Distortion of straight lines (due to relocation of retinal receptors)
4. Treatment: None; modification of activities as necessary and able.
5. Complications: Limitations on independence, safety.

B. **Glaucoma** (see p. 21)

C. **Cataracts** (see p. 21)

D. **Retinal detachment** (see p. 16)

XVII. **Physical Needs and Challenges:** Approximately one fifth of people over 70 years of age have some sort of visual impairment. As a result, this population is at a greater risk for falls and injuries. Vision impairment is a major contributor to disability and limitation of activity in this age group (National Center for Health Statistics, 2005). In the case of peripheral vision loss, the patient may be unaware until the loss is quite advanced, making him or her even more prone to injury. Young and older patients alike with reduced vision may be very frightened during an emergency. They may have developed a more keen sense of hearing or smell to compensate for their visual loss. Vision loss and impairment create challenges for both the patient and prehospital personnel.

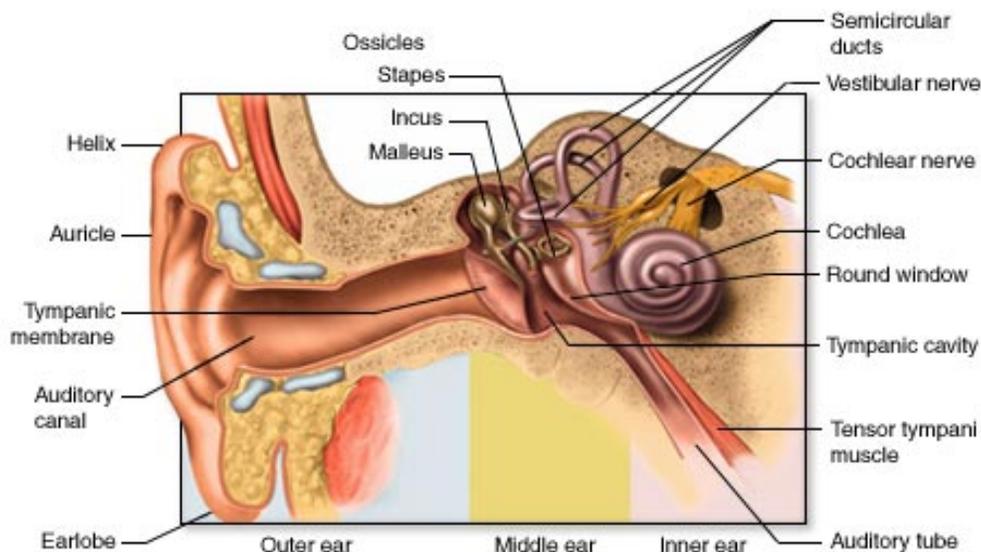
A. At-risk populations include those with:

1. Blindness
2. Degeneration of the eyeball, optic nerve, and nerve pathways
3. Chronic diseases affecting vision (DM, HTN)
4. Eye or brain injury
5. Infections affecting vision
6. Vitamin A deficiency in children
7. Glaucoma
8. Myopia, hyperopia, amblyopia
9. Optic nerve atrophy, hypoplasia
10. Retinal disease
11. Cortical vision impairment
12. Strabismus

- B. General consequences of vision loss in general include:
1. Misjudging the height of curbs, steps, etc.
 2. Difficulty reading medication labels
 3. Inability to see details (ex: button holes)
 4. Increased time needed to adjust to changes in light conditions
 5. Need for additional lighting
- C. When caring for any patient with a visual disturbance, follow these guidelines:
1. Orient the patient to whatever care or treatment is being provided
 2. "Lead" ambulatory patients instead of pushing them
 3. Explain any movements in position, such as moving the stretcher from the home to the back of the ambulance
 4. Retrieve visual aids if necessary, and bring to the hospital
 5. Be careful to announce yourself, provide your identity and the reason you are there
 6. If you move furniture in the visually impaired patient's home, be sure to put it back exactly where you found it
 7. Notify staff at the hospital so that appropriate assistance can be available (Aehlert, p. 195)
- D. Some patients with vision impairments have a guide dog to assist them. Guide dogs are highly trained companions-more than just pets! Avoid touching the dog until it is released by the patient. The patient may refuse to be transported if the dog cannot accompany him or her, or if arrangements cannot be made for the dog. Attempt to make arrangements to transport the animal to the hospital if at all possible. If that is not possible, and other arrangements cannot be made for the dog, request assistance from the police to make sure the dog is cared for.



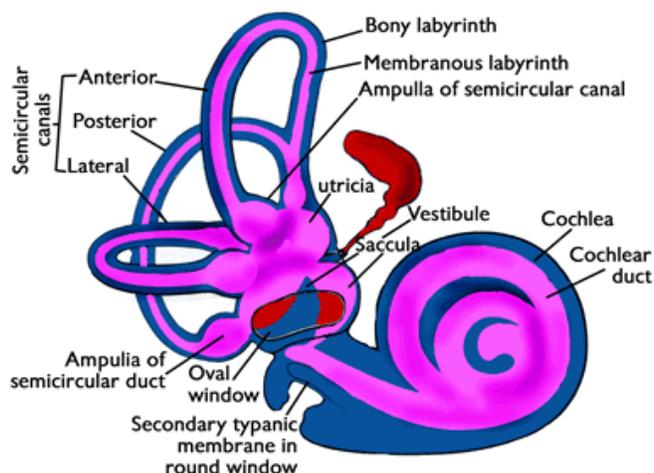
The Ear: Disorders and Emergencies –all new



- I. **Anatomy and physiology of the ear:** The ear is divided into three anatomic parts:
- A. **Outer/External ear:** collects and directs sound waves toward the middle ear.
1. **Auricle/pinna:** large cartilaginous external portion of the ear; collects sound waves and directs them to the ear canal.

2. **Ear canal:** The ear canal is considered a mucous membrane. It is lined w/ hairs and glands that secrete cerumen (ear wax), which protects against entry of foreign objects and insects, and deters growth of disease-causing agents. It directs sound to the tympanic membrane and narrows as it proceeds inward, serving to amplify the sound waves. The canal terminates at the TM, where the middle ear begins.
 3. **Tympanic membrane** (abbrev: TM): thin, oval membranous structure separating the external ear from the middle ear; protects the middle ear, conducts vibrations to the ossicles.
- B. **Middle ear:** Collects and amplifies sound waves, then transmits them to the inner ear.
1. Air-filled space in the temporal bone that contains the ossicle; connected to the inner ear by 2 covered openings – the round window and the oval window.
 2. **Eustachian tube** (auditory tube): connects the middle ear with the nasopharynx. Regulates pressure in the middle ear, protects against sound pressures and from nasopharyngeal secretions, and acts as an outlet for drainage of middle ear secretions.
 3. **Ossicles:** made up of 3 small bones: the **malleus**, the **incus**, and the **stapes**. The in and out vibrations of the TM convert sound energy into mechanical movement of the ossicles. The ossicles then act as levers to conduct vibrations to the inner ear.
 4. **Oval window:** opening between the middle ear and the vestibule, which receives vibrations from the TM. Almost completely filled by the base of the stapes. Movement of the oval window is magnified, allowing production of even very faint sounds.
 5. The middle ear is constructed to protect its delicate structures from violent movements associated with very noisy conditions. Two muscles, the tensor tympani, and the stapedius muscle, reduce the movement of the TM and the oval window, respectively.
- C. **Inner ear:** contains the sensory organ for hearing and equilibrium.
1. The receptors for equilibrium and hearing are protected by the **bony labyrinth**, whose outer walls are fused w/ the temporal bone. The **membranous labyrinth**, a collection of tubes and chambers that follow those of the surrounding outer bony labyrinth. The sensors for hearing and equilibrium are located here. The bony labyrinth is filled with **endolymph**, and the area between the two labyrinths is filled with **perilymph**.
 2. The bony labyrinth consists of 3 distinct parts. The **vestibule** houses 2 membranous sacs - the **sacculle** and the **utricle**, which contain receptors that provide sensation for gravity and linear acceleration. The chambers of the vestibule are continuous with those of the **semicircular canals**. The semicircular canals enclose semicircular ducts, which contain receptors that are stimulated by rotation of the head. Together, the vestibule and the semicircular canals are called the **vestibular complex**. The **cochlea** is a bony, spiral-shaped structure that contains the **cochlear duct** of the membranous labyrinth. Receptors in the duct provide the sense of hearing.
 3. The walls of the bony labyrinth are thick except at two points near the base of the cochlea. An opening in the bony labyrinth, the **round window**, is spanned by a thin membrane and separates perilymph in the cochlea from the air in the middle ear. The **oval window** is spanned by a membrane attached to the base of the stapes. Movement of the stapes ultimately leads to stimulation of the hearing receptors in the cochlear duct.
 4. The actual receptors in the inner ear are called **hair cells**. Each hair cell is connected to sensory neurons, and continually releases small amounts of neurotransmitter. When an external force causes the hair cells to move, secretion of neurotransmitter is either increased or decreased.

- a. **Organ of Corti:** Houses the rows of hair cells of the cochlear duct.



II. **Physiology of hearing:** Sound waves enter the ear through the auricle (pinna), and are directed down the auditory canal (ear canal) toward the tympanic membrane. Sound waves create vibration against the tympanic membrane, and that vibration is conducted to the ossicles. From there, sound waves are transmitted to the cochlear duct, which causes movement of the oval window. This movement causes fluid within the cochlea, which is lined with tiny hair follicles, to vibrate. The movement of the hairs creates nerve impulses that travel to the brain via the auditory nerve. Once the impulses arrive in the brain, they are interpreted as sound (Caroline. 2011).

III. **Physiology of equilibrium**

A. There are 2 aspects of equilibrium:

1. **Dynamic:** Helps us maintain balance when we move suddenly. These changes are monitored by **semicircular ducts** that provide information about rotational movement. Three ducts within the semicircular canal – anterior, posterior and lateral ducts – are continuous with the utricle. Each duct's sensory receptors are contained in the duct's swollen **ampulla**. Hair cells within the ampulla are embedded in a gelatinous structure. When there is movement, endolymph pushes against the gelatinous structure and stimulates the hair cells. Each of the 3 ducts responds only to its associated rotational movement – lateral, anterior, or posterior.
2. **Static:** Helps maintain posture and stability when at rest; monitored by the saccule and the utricle; provides information about position with respect to gravity and about changes in velocity. Hair cells in the utricle and saccule are also embedded in a gelatinous mass, but unlike the surface in the semicircular system, the surface contains a thin layer of densely packed calcium carbonate crystals. As a whole, this complex is known as an **otolith**. The weight of the crystals presses down on the gelatinous surface, and pushes the sensory hairs downward, or to the side, depending on the position of the head. Because the crystals are relatively heavy, when the body makes a sudden movement, the crystals lag behind. This lagging distorts the sensory hairs and alters the activity of the sensory neurons. Paired with visual information, the brain determines whether the sensations received indicate acceleration, or a change in head position.

B. Hair cells of the vestibule and semicircular canals transmit their information to the brain by way of the vestibular branch of the vestibulocochlear nerve (CN VIII).

IV. **Assessment of the ear:** This is a limited exam in the prehospital setting, usually not done unless the ear is part of the patient's chief complaint or area of injury. Inspect the outer ear structures for signs of trauma, infection (redness, heat, swelling, pain), or drainage (pus, blood, CSF).

A. External ear: Assess for intactness, symmetry, drainage, redness, heat, swelling, DCAP-BLS.

- B. **Otoscopic exam**
 - 1. Required material by new National Education Standards
 - 2. **Otoscope:** lighted, magnifying instrument used by physicians/PAs to examine the external auditory canal and the TM.
 - 3. Normal findings: The normal eardrum appears as a thin, translucent, grayish oval disk. The normal ear canal is pink and free of drainage/debris, aside from varying amounts of ear wax.
- C. Tympanic membrane: See above. Not assessed in the prehospital setting.
- D. Hearing: Whisper words or a phrase in the patient's ear; have them repeat it. Test each ear separately.
- E. **Proprioception** (ability to sense the location of body parts relative to the rest of the body) :
Assessment: With eyes closed, move thumb toward or away from the patient and see if they can correctly detect the direction of movement. One can do the same exam moving the big toe. This is particularly important with suspected spinal cord injuries as proprioception is also mediated by the posterior columns of the spinal cord.

V. **Assessment findings: ear**

- A. Hearing loss: (see "Deafness" below, p. 30)
- B. **Pain:** Assessment should include severity rating, SAMPLE history, and OPQRST elements, including pertinent negatives ("does it hurt when you swallow, cough, lie flat, etc.?). History of events leading up to the pain (scuba diving trip, recent URI, for example) is especially important. Associated symptoms may include sensations like ear popping or itching.
- C. Discharge/ or drainage: Discharge noted from the ear may be due to TM rupture (hemorrhagic otorrhea), infection (pus), or basilar skull fracture (CSF).
- D. **Tinnitus:** Tinnitus is the perception of noise (ringing, roaring, or hissing) in the ear, usually due to medication or exposure to loud noise, but may be associated with allergic reaction, Lyme disease, brain tumors, or foreign bodies in the ear. It is a symptom, rather than a disease, and often accompanies hearing loss, especially w/ age-related hearing loss. It is usually constant, and may change in intensity, but the patient is often only intermittently aware.
- E. **Vertigo:** Vertigo is the sensation of spinning, which is unrelieved by lying down and often worsens if the eyes are closed. Often, vertigo occurs in the absence of a known cause, but it is considered a problem with either the fluid in the inner ear or the bones in the middle ear. It can be debilitating as it prevents even the simplest of activities, and is often accompanied by nausea and vomiting. Vertigo is often accompanied by tinnitus.
- F. **Presence of CSF:** An indication of a basilar skull fracture involving the middle fossa bones. Thorough neuro assessment is indicated!
- G. Ruptured TM (see "Conditions of the ear" below)

VI. **Conditions of the ear**

- A. **Deafness:** We hear by way of air conduction and bone conduction of sound waves. Hearing loss affects 5-10% of the population (Martini et al, 2008). There are several different types of hearing loss:
 - 1. **Conduction deafness:** Due to blockage of sound waves' passage down the external auditory canal, damage to the TM, disruption of the ossicles, or middle ear scarring or fluid build-up. Structures involved in this type of hearing loss are those of the external and middle parts of the ear.
 - 2. **Sensorineural (nerve deafness):** Results from damage to the hair cells in the organ of Corti. Damage is usually due to very loud noise, infection, ototoxic drugs, temporal bone fracture, Meniere's disease, or aging. Structures involved include those of the inner ear, or the vestibulocochlear nerve (CN VIII).

3. **Central deafness:** Most rare type of hearing loss. Nerve impulses reach the temporal lobes, but the brain is unable to interpret the signals.
4. **Etiology of deafness**
 - a. Prolonged exposure to loud noise
 - b. Disease (ex: Meniere's disease)
 - c. Tumors
 - d. Medication (esp. aspirin)
 - e. Viral infections
 - f. Natural degeneration of cochlea, labyrinth in old age
 - g. Auditory process deficits: A deficit in the processing of information specific to auditory modality; may be worse in "unfavorable acoustic environments: and is often associated w/ listening, speech interpretation, language development, and learning difficulties. (Journal of the American Academy of Audiology)
 - h. Auditory dyssynchrony: Condition in which cochlear function is normal, but the patient is unable to decode speech and or language (Medscape, 2012)
5. **Physical needs and challenges: hearing impairments**
 - a. Be alert to signs that indicate a patient may have a hearing impairment:
 - (1) Presence of hearing aids
 - (2) Poor diction
 - (3) Inability to respond to verbal communication unless direct eye contact is made
 - b. Accommodations for hearing impaired patients
 - (1) Retrieve hearing aid or other assistive device
 - (2) Provide pen and paper as an alternative means of communication
 - (3) Speak softly and directly into the patient's ear canal in a low-pitched voice (notable, since ~ 80% of hearing loss involves inability to hear high-pitched sound)
 - (4) Speak slowly so speech is clear
 - (5) For patients w/ significant but not total hearing loss, consider allowing the patient to wear your stethoscope, while you hold the bell and speak into it
 - (6) Ask a family member or care giver to assist, especially if patient communicates in sign language
 - (7) DO NOT shout or exaggerate lip movement!
 - (8) Notify the receiving facility as soon as possible
 - c. Special considerations: hearing aids (see geriatric exam findings)

B. Foreign body

1. Presence of foreign bodies is not always outwardly obvious, and assessment is limited to visual clues. Indications of a potential foreign body include:
 - a. Redness
 - b. Swelling of the external ear structures
 - c. Bloody, purulent, or foul-smelling drainage from affected ear
 - d. Decreased hearing in the affected ear
 - e. Pain/discomfort
 - f. Sound of insect buzzing in the ear
 - g. Sensation of fullness or something in the ear
2. To visualize the external ear canal, pull the ear back and examine with a penlight.
3. Seen most often in pediatric population
4. Rarely an emergency

- a. Food and organic matter will expand in moist environment and become difficult to remove
 - b. Small batteries (ex: watches) may leak chemicals and cause burns
 5. Do not remove any impaled objects – stabilize it only. May remove foreign body if it is visible and within reach by hand.
- C. **Impacted cerumen:** Ear wax is the most common ear canal obstruction in both adults and pediatric patients. Children often develop this condition as a result of cerumen being packed down into the ear canal by inserting cotton swabs too far. In the geriatric population, cerumen is dryer than normal, and normal drainage of ear wax is often blocked by the presence of a hearing aid.
- D. **Labarynthitis**
1. The labyrinth (balance control structure in the inner ear structure) becomes irritated and swollen, often following a viral ear or upper respiratory infection. Patients experience loss of balance and dizziness (or “vertigo”: sensation of spinning in the absence of actual movement; may also include sensation of falling or tilting). Other symptoms may include tinnitus (ringing in the ears), temporary hearing loss, nausea and vomiting.
 2. Prehospital care:
 - a. Medication for nausea and vomiting
 - b. Assist patient to move to stretcher – danger of falls
- E. **Meniere’s disease**
1. Symptoms of Meniere’s disease are related to changes in fluid volume in the labyrinth. The patient usually experiences abrupt onset of vertigo, tinnitus, and hearing loss, and may be accompanied by severe nausea and vomiting. The patient may experience pressure or pain in the ear as well.
 2. Causes: environmental factors, viral infection, biological factors
 3. Management: same as labarynthitis (above)
- F. **Otitis externa:** (Swimmer’s ear). Bacterial growth in the ear canal produces redness, pain, tinnitus, and irritation. Externally, redness may be noted to the part of the ear canal visible upon gross inspection. History will usually include recent exposure to polluted water or frequent exposure to water without drying the ear. Prehospital management: supportive.
- G. **Otitis media**
1. Infection of the ear drum/middle ear, usually bacterial (between ear drum and middle ear). Usually heal in several weeks w/o treatment.
 2. More common in children and following URI
 3. Signs and symptoms
 - a. Pain
 - b. Diminished hearing
 - c. In young children, pulling or rubbing of ear, irritability; poor feeding (check for dehydration)
 - d. Fever, chills
 - e. Sleeplessness
 - f. Malaise, nausea, vomiting, diarrhea
 4. Management: supportive care; pain management if severe
 5. Risk for meningitis: infection can travel from ear structures to brain
- H. **Perforated Tympanic Membrane**
1. Hole or rupture in the eardrum, usually related to infection or trauma.

2. Etiology
 - a. Pressure trauma (diving, water skiing)
 - b. Direct blow to ear
 - c. Explosion/barotraumas
 - d. Foreign objects (ex: Q-tip)
3. Signs and symptoms
 - a. Diminished hearing
 - b. Occasionally, hemorrhagic otorrhea
 - c. Usually not painful, but may be preceded by pain if related to build-up of fluid/pus behind ear drum
4. Management
 - a. Supportive care
 - b. Pain relief if severe

VII. **Geriatric exam findings:** As people age, the TM thins and loses elasticity, resulting in dry cerumen. The ear canal narrows and stiffens, and the hairs lining it become stiff. This predisposes to cerumen impaction, which interferes w/ movement of the TM and with sound transmission. Sensory function related to aging is inevitable, since it is largely due to lack of replacement of neurons. Increases in the strength of stimuli may partially compensate, but the loss of those axons necessary for conducting sensory information is inadequate to compensate fully.

- A. **Presbycusis:** Progressive loss of hearing associated with aging. Occurs due to changes in structures of the inner ear, the auditory nerve, middle, or outer ear. Happens gradually. People w/ age-related hearing loss find it difficult to hear high-pitched sounds, and to tolerate loud sounds. These patients may have difficulty hearing conversations in high background noise conditions.
- B. **Use of hearing aids:** Note whether the patient is wearing a hearing aid. Keep in mind that some patients remove their hearing aid to sleep. These patients may seem to respond inappropriately to verbal communication. Always confirm that the patient can hear you! If the patient does use a hearing aid, be sure it is either put in place or brought along to the hospital. Be very careful to protect the device, and inform those to whom you transfer care that the patient has a hearing aid. They are very expensive and are easily damaged!
- C. General physiologic changes and their consequences: With aging, the TM thins and becomes less elastic, resulting in decreased sound transmission. Drier ear wax related to atrophy of cerumen glands makes the patient more prone to impacted ear wax. Combined with a narrowed ear canal and hairs that have grown stiff and coarse, movement of the eardrum is impaired, impairing sound transmission. Atrophy of the cochlear hair cells contributes to decreased sense of balance.

References

- Aehlert, B. (2010). Patient assessment. In, *Paramedic Practice Today: Above and Beyond*. (vol. 1) (588-634). St. Louis: Mosby
- Aehlert, B. (2010). Head, ear, eye, nose, and throat disorders. In, *Paramedic Practice Today: Above and Beyond*. (vol. 1) (674-689). St. Louis: Mosby
- Aehlert, B. (2010). Lifespan development. In, *Paramedic Practice Today: Above and Beyond*. (vol. 1) (275-295). St. Louis: Mosby
- Aehlert, B. (2010). Patients with special challenges. In, *Paramedic Practice Today: Above and Beyond*. (vol. 2) (192-291) St. Louis: Mosby
- Aehlert, B. (2010). Head and face trauma. In, *Paramedic Practice Today: Above and Beyond*. (vol. 2) (383-423). St. Louis: Mosby
- Aehlert, B. (2010). Burn injury. In, *Paramedic Practice Today: Above and Beyond*. (vol. 2) (348-382). St. Louis: Mosby
- Bausch & Lomb. (2010). *A journey through the human eye: How we see*. [On-line]. Available: <http://www.youtube.com/watch?v=gvozcv8pS3c>
- Beebe, R., Funk, D.I., Scadden, J. (2010). Head, face, and traumatic brain injury. *Fundamentals of Basic Emergency Care* (3rd ed) (722-744). Clifton Park, NY: Delmar
- Beebe, R., & Myers, M. (2012). Soft tissue injury. In, *Trauma Care & EMS Operations* (vol. III) (178-204). Clifton Park, NY: Delmar.
- Beebe, R. & Myers, M. (2012). Neck and facial trauma. In, *Trauma Care & EMS Operations* (vol. III) (58-74). Clifton Park, NY: Delmar.
- Beebe, R. & Myers, M. (2012). Water emergencies. In, *Trauma Care & EMS Operations* (vol. III) (312-326). Clifton Park, NY: Delmar.
- Beebe, R. & Myers, M. (2012). Emergency response to terrorism. In, *Trauma Care & EMS Operations* (vol. III). (422-446). Clifton Park, NY: Delmar.
- Beebe, R., Myers, M. (2012). Vehicle Rescue and Extrication. *Trauma Care & EMS Operations* (vol. III). (474-487). Clifton Park, NY: Delmar
- Beebe, R. & Myers, M. (2012). Trauma overview. In, *Trauma Care & EMS Operations* (vol. III) (2-32). Clifton Park, NY: Delmar.
- Beebe, R. & Myers, M. (2012). Traumatic brain injury. In, *Trauma Care & EMS Operations* (vol. III) (34-56). Clifton Park, NY: Delmar.
- Beebe, R. & Myers, M. (2012). Soft tissue injury. In, *Trauma Care & EMS Operations* (vol. III) (178-203). Clifton Park, NY: Delmar.
- Beebe, R. & Myers, M. (2012). Mountain medicine. In, *Trauma Care & EMS Operations* (vol. III) (298-310). Clifton Park, NY: Delmar.
- Beebe, R., Myers, M. (2012). Burn Trauma. *Trauma Care & EMS Operations* (vol. III). (206-229). Clifton Park, NY: Delmar
- Caroline, N. (2013). Diseases of the eyes, ears, nose and throat. In, *Nancy Caroline's Emergency Care in the Streets*. (vol. 2) (7th ed) (1091-1104). Burlington, MA: Jones & Bartlett Learning
- Castellari, A. & Pieramici, D. (2007). Open globe management. *Comprehensive Ophthalmology* [On-line]. Available: www.medscape.com
- Chichin85. (2007). Hearing and Balance. [On-line]. Available: <http://www.youtube.com/watch?v=vTiGskc1o48>
- Martini, F. H., Bartholomew, E. F., & Bledsoe, B. E. (2008). Equilibrium and hearing. In, *Anatomy and Physiology for Emergency Care*. (2nd ed) (351-361). Upper Saddle River: Pearson Education Inc.
- Martini, F. H., Bartholomew, E. F., & Bledsoe, B.E. (2008). The general and special senses. In, *Anatomy & Physiology for Emergency Care* (2nd ed) (336-351). Upper Saddle River: Pearson Prentice Hall.
- Mattera, C. (2008). Cranial Nerve handout. Unpublished class handout for NWC EMSS Paramedic class.
- Mattera, C. (2010). Drug Administration. Unpublished class handout for NWC EMSS Paramedic class.
- Mattera, C. (2012). Neuro Assessment in 5 Easy Steps. Unpublished class handout for NWC EMSS Paramedic class.

- Mattera, C. (2012). Ocular trauma. Unpublished class handout for NWC EMSS Paramedic Class.
- MayoClinic.com. (2011). *Horner syndrome* [On-line]. Available: <http://www.mayoclinic.com/health/horner-syndrome/DS01137>
- MayoClinic.com. (2010). *Retinal detachment*. [On-line]. Available: <http://www.mayoclinic.com/health/retinal-detachment/DS00254>
- Medscape.com. (2012). *Auditory Neuropathy*. [On-line]. Available: <http://emedicine.medscape.com/article/836769-overview#showall>
- Medscape.com. (2011). *Eustachian Tube Function*. [On-line]. Available: <http://emedicine.medscape.com/article/874348-overview#aw2aab6b6>
- MorTan Inc. *The Morgan Lens*. Unpublished document accompanying product.
- National Institute of Neurological Disorders and Stroke. (2011). *Holmes-Adie Syndrome*. [On-line]. Available: http://www.ninds.nih.gov/disorders/holmes_adie/holmes_adie.htm.
- Northwest Community Emergency Medical Services System Procedure Manual: Contact Lens Removal-Soft Lenses. (2012). Unpublished document for NWC EMSS System.
- NWC EMSS Procedure Manual: Contact Lens Removal-Hard Lenses. (2012). Unpublished document for NWC EMSS System.
- NWC EMSS Procedure Manual: Eye Irrigation. (2012). Unpublished document for NWC EMSS System.
- PubMed Health. (2010). *Astigmatism*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002010/?report=printable>
- PubMed Health. (2010). *Chalazion*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002001/>
- PubMed Health. (2011). *Color blindness*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001997/?report=printable>
- PubMed Health. (2010). *Farsightedness*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002015/>
- PubMed Health. (2010). *Hordeolum*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002004/>
- PubMed Health. (2010). *Iritis/Uveitis*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002000/>
- PubMed Health. (2010). *Nearsightedness*. [On-line]. Available: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002018/>
- Region IX Standard Operating Procedure/Standing Medical Orders. (2011). Unpublished document for Northwest Community Emergency Medical System.
- Robinett, D; Kahn, J. (2008). The physical examination of the eye. *Emergency Medicine Clinics of North America*, 26(1), 1-16. WB Saunders Co.
- Sanders, M. (2012). Review of human systems. In, *Mosby's Paramedic Text Book* (4th ed) (512-559) (139-210). St. Louis: Elsevier.
- Sanders, M. (2012). Diseases of the eyes, ears, nose, and throat. In, *Mosby's Paramedic Text Book* (4th ed) (720-739). St. Louis: Elsevier.
- Sanders, M. (2012). Trauma: burns. In, *Mosby's Paramedic Text Book* (4th ed) (1122-1145). St. Louis: Elsevier.
- Sanders, M. (2012). Secondary assessment. In, *Mosby's Paramedic Text Book* (4th ed) (512-559). St. Louis: Elsevier.
- Sanders, M. (2012). Special patient populations: Geriatrics. In, *Mosby's Paramedic Text Book* (4th ed) (1388-1395). St. Louis: Elsevier.
- Sanders, M. (2012). Head, face and neck trauma. In, *Mosby's Paramedic Text Book* (4th ed) (1146-1175). St. Louis: Elsevier.
- Shuker, S. (2008). Mechanism and emergency management of blast eye/orbital injuries. [On-line]. Available: www.medscape.com
- Warner Bros. (2010). *Any Given Sunday: The Eye*. [On-line]. Available: <http://www.youtube.com/watch?v=Fkkg2Zr3lw4>

GLOSSARY

Bell's Phenomenon:	Reflex movement upward of eyes with eyelid closure.
Chemosis:	Swelling of the conjunctiva around the cornea.
Cycloplegia:	Pharmacologic paralysis of ciliary body musculature, reducing reactive spasm.
Entropion:	Inturning of eyelid and lid margin.
Epiphora:	Tearing.
Keratitis:	Inflammation of the cornea.
Lagophthalmos:	Incomplete lid closure.
Limbus:	Transition zone between clear cornea and white sclera.
Mydriasis:	Pupil dilation (opposite of miosis).
Phthisis bulbi:	End stage non-seeing eye, with associated shrinking and disorganization of intraocular contents.
Seidel test:	Use of fluorescein to detect perforating injury; leakage of aqueous humor will create green rivulets when observed with cobalt blue or Wood's light.
Trichiasis:	Inversion of hairs about an orifice, as eyelashes that turn in and cause irritation of the cornea.
Uvea:	The middle, pigmented, vascular layer of the eye consisting of the choroid, the ciliary body, and the iris. Also called the uveal tract.
Uveitis:	Intraocular inflammation; may be anterior or posterior in location.

Eye Fun Facts:

- You blink every 2-10 seconds. As you focus on each word in this sentence, your eyes swing back and forth 100 times a second; every second the retina performs 10 billion computer-like calculations.
- The human eye can perceive more than 1 million simultaneous visual impressions and is able to discriminate among nearly 8 million gradations of color.
- Having two eyes instead of just one provides us with stereo vision and depth perception. Each of our eyes views an object from a slightly different angle. Our brains put the different images together into one three-dimensional image.
- The iris of the eye provides better identification than a fingerprint. A scan of the iris has 256 different unique characteristics. A fingerprint has only 40 (LWW.comInsider - December 2001).