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Eye Emergencies

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Editor's Note—*Eye emergencies are a common reason for patients to present to the emergency room or their primary care physician. This article presents a review of the ophthalmologic exam, the most common conditions requiring emergent ocular evaluation, and guidelines for diagnostic testing, emergent treatment, and referral when needed.*

A "red eye" is a common presenting symptom. The etiology can be as benign as a subconjunctival hemorrhage or as sight threatening as angle closure glaucoma or endophthalmitis. Most cases of red eye can be diagnosed by examination alone and appropriate treatment instituted. Angle closure glaucoma and endophthalmitis require emergent treatment and possibly surgery in order to preserve vision.

Patients with ocular trauma require thorough examination in order to make a precise diagnosis on which to base treatment. Special attention is required for injuries possibly involving foreign body penetration or perforation of ocular structures, and for blunt trauma that may have caused occult rupture of the globe. Chemical burns are true ophthalmologic emergencies that require immediate ocular lavage.

Acute non-traumatic visual loss can arise from any area of the visual system. The most common etiologies are due to optic nerve or retinal disorders. Some causes, such as central retinal artery occlusion, require immediate treatment to try and preserve vision.

Diplopia can signal an acute neurologic emergency (such as third nerve palsy from posterior communicating artery aneurysm), the first manifestations of multiple sclerosis, or an isolated microvascular event.

This article reviews the common causes for patients presenting for emergency eye care, the examination, diagnostic tests needed to make an accurate diagnosis, and guidelines for treatment—especially for those conditions that require emergent treatment in order to preserve vision.

Ocular Examination

The eye examination can be broken down into the eight parts listed in Table 1.

For both medical and legal reasons, visual acuity should be

checked on every patient who presents with an eye problem. This is usually done for distance vision with a standard acuity chart (Snellen) and recorded for first the right eye and then the left eye. A near card can also be used and is especially helpful for bedridden patients. Acuity can be checked both with and without correction, although the most important piece of information is usually the "best corrected" acuity. If the patient did not bring or does not have glasses, a pinhole occluder can give a good approximation of corrected acuity.

The external examination should look for signs of trauma, lid disease, proptosis, etc.

Pupillary examination is important, especially in patients with significant loss of vision. The swinging flashlight test is used to check for a relative afferent pupillary defect.[1]

Extraocular movements (EOMs) are checked by having the patient follow an object while holding their head still. Look especially for any evidence of muscle palsy or restriction. Defer EOM testing if an open globe injury is suspected.

A confrontation visual field is performed by presenting two objects (usually both hands of the examiner) in front of each eye separately and having the patient state whether they can count the fingers on each hand. This is especially helpful for picking up hemianopic field defects.

Biomicroscopy of the anterior segment of the eye is best performed using a slit lamp. A direct ophthalmoscope with the +10 lens in place also works well. Look especially for abnormalities of the cornea, anterior chamber (i.e., hyphema), and iris.

Intraocular pressure is tested unless an open globe is suspected. A Goldmann type applanation tonometer is best. The portable Tonopen (Mentor) also works well in the emergency setting.

Fundoscopic examination is best performed through dilated pupils (use 1% tropicamide and 2.5% phenylephrine). Carefully document any dilating drops given in patients with head trauma.[2] A direct ophthalmoscope gives a good view of the optic nerve, macula, and vessels. Indirect ophthalmoscopy is needed for the peripheral retinal exam.

Table 1. The Ocular Examination

- Visual Acuity
- External-lids, lashes, lacrimal, periorbital
- Pupils
- Extraocular motility
- Visual fields
- Biomicroscopy
- Intraocular pressure
- Fundoscopy

Red Eye

A red eye can signify a sight-threatening process or a benign self-limited disease. Examination findings which help to separate various etiologies are listed in Table 2.

Common non-traumatic causes of red eye, along with recommended treatments, are listed below. (Traumatic causes are given in a later section.)

Subconjunctival hemorrhage. Usually spontaneous onset with no pain or decreased vision. Exam shows solid, bright red patch on bulbar conjunctiva with sharp borders. If the patient has any history of trauma, look for further injury. Treat by observation. If there are multiple recurrences or other signs of excessive bleeding, check clotting parameters.[3]

Allergic reactions. The patient has symptoms of itching, burning, and watering. There is often a history of systemic allergic problems. Exam shows injected conjunctiva, possibly chemosis, and watery discharge. Treat by cool compresses, removal of offending allergen, and medications if necessary. Naphazoline and others are available over the counter. Histamine blockers, such as levocarbastine, help acute symptoms. Mast cell inhibitors (lodoxamide tromethamine, cromolyn) help prevent future attacks. Olopatadine hydrochloride combines both histamine blockade and mast cell inhibition.

Viral infections (non-herpetic). Patients often have a history of exposure to "pink eye" or concurrent upper respiratory infection. Exam shows preauricular adenopathy, watery discharge, possible conjunctival membrane. The cornea may have grayish round subepithelial lesions. The patient may have eyelid edema, especially with adenovirus. Treatment for most

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Table 2. Red Eye Findings

Discharge

Clear or watery discharge with allergic processes, superficial abrasions, and viral infections. Yellowish or purulent discharge most common with bacterial conjunctivitis.

Preauricular adenopathy

Seen with most viral infections, including herpes simplex. Gonococcus only bacterial conjunctivitis with a preauricular node.

Membrane

Fibrinous membrane over palpebral conjunctiva most commonly seem with adenoviral or herpetic viral infections, streptococcus or gonococcus bacterial infections, and with chemical burns.

viral conjunctivitis is cool compresses and observation (adenoviral infections can take 2-3 weeks to clear and are highly contagious). An antibiotic, such as erythromycin ointment 1-2 times/day, can be used as prophylaxis against secondary bacterial infection. Topical steroids can help the acute symptoms but tend to prolong the course, especially of corneal infiltrates.[4]

Herpes Simplex. This usually presents with pain or foreign body sensation and decreased vision. Exam may show a tender preauricular node, corneal dendrite that stains with fluorescein (acute HSV) or hazy, edematous patch in cornea (disciform HSV). Treatment is topical trifluridine eight times/day for 5-7 days until dendrite heals. Debridement of the dendrite with a cotton-applicator at the slit lamp may also help hasten healing.[5] The ability to differentiate between HSV keratitis and conjunctivitis is important. Prompt referral to an ophthalmologist is recommended.

Bacterial Conjunctivitis. This presents with a history of yellowish discharge and eyelids mattered together upon awakening. It can be mild to severe. Hyperacute or extremely severe cases of purulent conjunctivitis are often due to gonococcus, which is also the only bacterial conjunctivitis to have preauricular adenopathy. Examination shows mild-to-severe conjunctival injection and purulent discharge. Most cases of bacterial conjunctivitis are mild and self limited. Treatment should be with an earlier generation antibiotic such as 10% sodium sulfacetamide qid for 5-7 days.[6] Overuse of later generation broad spectrum antibiotics (such as the topical flouroquinolones) should be avoided because of the emerging problems of resistance.[7] Non-responding infections should be cultured and treatment adjusted based on culture results. Gonococcal infection requires systemic treatment (ceftriaxone),[8] ocular lavage, and topical erythromycin, along with appropriate reporting and treatment of partners.

Bacterial Keratitis. Patients have a history of photophobia, pain, and decreased vision. It is more common in soft contact lens wearers, especially if lenses are worn overnight. The patient may have had a predisposing insult to corneal epithelium, such as an abrasion from mild trauma or misdirected eyelashes. Examination shows marked conjunctival injection, whitish ulcer in the superficial cornea, anterior chamber reaction, and occasionally hypopyon. Get ophthalmology consultation for culture and scraping of cornea and possible admission to the hospital. Treatment is usually started with hourly, around-the-clock, broad spectrum topical antibiotics. One of the flouroquinolones (oflaxacin or ciprofloxacin) is usually used initially with adjustment of medications based on culture results.[9] Homatropine 5% bid will help to reduce ciliary spasm and pain.

Fungal Keratitis. The symptoms are similar to bacterial keratitis. The exam shows a more lacy appearance to the corneal infiltrate and needs to be referred to an ophthalmologist. There is often a history of superficial corneal trauma from plant material. Treat patients with natamycin drops after obtaining cultures. Fungal ulcers may require surgical debridement.

Endophthalmitis. History of recent (usually within 48-72 hours) intraocular surgery or trauma. There is usually a sudden decrease in vision to hand motions or light perception levels. Exam shows marked conjunctival injection, discharge, possible eyelid edema, hazy cornea, and marked anterior chamber cellular reaction with hypopyon. The fundus is often not visible secondary to vitreous inflammation. Endophthalmitis is a true ophthalmic emergency and requires immediate evaluation and treatment by an ophthalmologist. Treatment consists of vitreous tap or vitrectomy, intravitreal antibiotics and possibly steroids, and intensive topical antibiotics. Prognosis depends on the causative organism and the length of time to treatment.[10]

Eyelid Infections (Preseptal Cellulitis). There is a history of spontaneous onset or recent minor lid trauma. Exam shows an erythematous and edematous eyelid. The globe is normal to mildly injected. Vision, extraocular motility, pupils, and optic nerve head are usually normal. Look for a hordeolum (infection around base of eyelash) or chalazion (enlarged meibomian gland) as the inciting event. Use warm compresses qid if evidence of chalazion. If there is early infection and no material for Gram's stain, treat empirically with amoxicillin/clavulanate 250-500 mg po tid in adults and appropriate adjusted dose in children.[11] More severe cases may require incision and drainage of eyelid and treatment with appropriate IV antibiotics based on Gram's stain and culture. Careful differentiation must be made between preseptal and orbital cellulitis.

Orbital Cellulitis. Patients give a history of pain, decreased vision, and possible diplopia. They may have sinusitis (especially ethmoiditis), recent orbital trauma, or recent dental surgery. Examination shows swollen, erythematous lids, mild to severe proptosis, limitation of EOMs, decreased vision, and possible afferent pupillary defect. Workup includes a CT scan (look for signs of sinusitis, subperiosteal abscess, and orbital abscess) and appropriate cultures. Patients are admitted and given broad spectrum coverage with IV antibiotics (Clindamycin and Ceftazidime). ENT consultation should be obtained.[12] In diabetics with ketoacidosis and immunocompromized patients, consider fungal infection (mucormycosis), which is life threatening and requires immediate treatment with IV, Amphotericin B, surgical debridement, and possibly hyperbaric oxygen.[13]

Dacryocyctitis. Patients give a history of purulent dis-

charge and pain over the medial canthal region. Exam shows edema and erythema over region of the lacrimal sac and often reflux discharge through the puncta with pressure over the lacrimal sac. Treat by taking cultures and starting oral antibiotics (amoxicillin/clauvulanate 250-500 mg po tid or cephalexin 500 mg po q 6 h). Definitive treatment is surgical dacryocystorhinostomy.[14]

Uveitis or Inflammation of the Uveal tissue (Iris and Choroid). The most common emergent presentation is as iritis. A history of pain, photophobia, and decreased vision is given. Exam shows ocular injection, especially at the corneal scleral limbus and whitish deposits on the posterior cornea (keratic precipitates). Flare and cells are seen in the anterior chamber with a tangential slit beam. A hypopyon may be present and there may be dilated vessels on the iris. Funduscopic exam is needed to rule out posterior uveitis. Intraocular pressure may be elevated from inflammatory cells or decreased from ciliary body inflammation. Often no laboratory workup needed acutely for isolated, primary iritis. Treat with intensive topical steroids (Prednisolone acetate 1% every hour) and cycloplegics (atropine 1% or homatropine 5% bid).[15] Ophthalmologic follow-up is required for steroid taper and further diagnostic work-up, especially if any evidence of posterior (choroidal) involvement.

Acute Angle-Closure Glaucoma. Patients usually present with sudden onset of redness, decreased vision, intense pain, possibly nausea, and emesis. This is more common in farsighted patients. Examination shows marked ocular injection, especially at the limbus. The cornea is edematous. The pupil may be mid-dilated and non-reactive. The intraocular pressure is elevated, often markedly to 60 or 70 mmHg. The fellow eye is also at significant risk for angle closure and can be examined for narrow angles at the slit lamp. Angle closure is a true ophthalmologic emergency that requires immediate treatment. Ocular massage should be done immediately as this can sometimes break the attack. Topical aqueous suppressants (timolol, brominidine, dorzolamide) are placed in the eye. Miotics, such as pilocarpine 2%, are given. Oral osmotics like 50% glycerin are given to help decrease intraocular pressure. As soon as possible, a surgical peripheral iridectomy (laser or incisional) is performed. Prophylactic peripheral iridectomy is usually placed in the fellow eye within 1-2 weeks.[16]

Carotid Cavernous Fistula. There are two main types, low flow and high flow. The low flow type is usually found in elderly, hypertensive patients without a history of trauma. They present with a red eye secondary to dilated episcleral vessels, elevated intraocular pressure, and may have proptosis. Treatment is by control of blood pressure and observation or, in more severe cases, interventional radiology. High flow types are usually found in younger patients following blunt or penetrating head trauma. The vision may be decreased. There is marked ocular vascular engorgement externally and internally, and usually marked exophthalmos. This requires emergent treatment. Interventional radiologic treatment methods are usually used with attempted embolic closure of the fistula.[17]

Table 3. Common Chemicals Involved in Eye Injuries

Common Substances	Compound	Class
Battery acid	Sulfuric acid (H_2SO_4)	Acid
Bleach	Sulfurous acid (H_2SO_3)	Acid
Cement and mortar	Lime ($Ca(OH)_2$	Alkali
Chrome plating solution	Chromic acid (Cr_2O_3)	Acid
Drain cleaner	Lye (NaOH)	Alkali
Glacial acetic acid	Acetic acid (CH_3COOH)	Acid
Glass and tile cleaners	Ammonia (NH_3)	Alkali
Glass frosting acid	Hydrofluoric acid	Acid
Fertilizers	Ammonia (NH_3)	Alkali
Hydrochloric acid	Hydrochloric acid	Acid
Industrial cleaners	Sulfuric acid	Acid
Plaster	Lime ($Ca(OH)_2$)	Alkali
Sparklers and firecrackers	Magnesium hydroxide	Alkali
Vinegar	Acetic acid (Ch_3COOH)	Acid
Whitewash	Lime ($Ca(OH)_2$)	Alkali

Traumatic Visual Loss

Chemical burns. Exposure of the eye to chemicals is a true ocular emergency. Prompt and vigorous lavage is crucial to successful management. IV solutions, such as Ringers or normal saline attached to a large bore IV tubing, can be used to irrigate the eye at a rate of 500 cc/30 minutes. The eye should be kept open and the topical anesthesia using proparacaine or tetracaine is recommended to facilitate the irrigation. Sometimes, special irrigating contact lenses are used but this is not necessary. Great care must be taken to have the patient move the eye in all directions and careful inspection of the fornices must be undertaken during irrigation. Five minutes after cessation of irrigation, the pH of the eye should be checked and the range should be from pH 6.8-7.4. If pH is outside of this range, further irrigation is necessary.

A complete history of the incident with identification of the chemical and duration of exposure prior to irrigation is crucial. (*See Table 3.*) Acids cause surface proteins to precipitate and coagulate, confining the chemical to the surface of the eye and limiting the damage. The most common causes of acid injury are sulfuric, sulfurous, hydrofluoric, acetic, chromic, and hydrochloric acid.[18] Alkalis saponify and solubilize cell membranes, allowing penetration into the eye and resulting in significant deep damage to the eye. The most common causes of alkali injury are ammonia, lye, potassium hydroxide, magnesium hydroxide, and lime.[18] For all chemical injuries, a thorough examination of the lids with eversion of the lids to examine the fornices for particular matter is important. Calcium hydroxide particles may be more easily removed with a cotton tip applicator soaked in EDTA. Slit lamp exam of the cornea and conjunctiva with fluorescein is necessary to check for staining defects in the cornea. The intraocular pressure should be checked using the tonopen or Goldmann applanation tonometer. Prompt referral to an ophthalmologist is recommended at this point. If the IOP is elevated, Diamox 250 mg tablet and one drop of a topical beta blocker such as timoptic 0.5% will help decrease pressure. One drop of cycloplegic agent (scopolamine 0.25%) and topical antibiotic ointment (erythromycin) must be given with oral medications for pain control.

Eyelid Lacerations

Trauma to the eyelids and orbit can pose a severe threat to eyesight. The history must include relevant information about blunt vs. sharp objects, type of object, and velocity of the object. Thorough examination of the eyelids, globe, motility, and palpation of the orbital rim is important. Presence of lid lacerations requires careful cleaning of the wound and determination of tissue loss. Unless the patient comes with tissue in a plastic bag, tissue loss is rare, even in a complex lid laceration with multiple full thickness laceration with significant exposure of the globe. Great care must be taken not to discard any tissue or excise what appears to be macerated tissue from the surface of the lid. If the laceration is lateral to the puncta, chances of canalicular laceration are remote. If the laceration is medial to the puncta and includes the lid margins, there is a possibility of canalicular laceration. For all lacerations involving the lid margins and/or medial lid, either an ophthalmologist or skilled facial plastics specialist should close the wound, taking care to maintain the integrity of the canalicular system with stents. The repair of lid lacerations should be considered urgent and, in most cases, a delay of 12-36 hours does not alter the surgical result.[19] Lid lacerations lateral to the puncta can be closed easily with 6.0 silk or nylon suture superficially and 6.0 vicryl for deeper structures such as the tarsal plate. Care should be taken to horizontally close lacerations as often as possible, because vertical closure can result in cicatricial ectropion with exposure and drying of the eyes. Prophylactic antibiotics are indicated if given within three hours of injury, but since the lids are highly vascular, the chances of infection are reduced and the majority of lids do well.[20]

Orbital Fractures

If examination of the orbital walls with gentle palpation reveals crepitus, subcutaneous air, or a severe restriction to ocular motility, further evaluation with a CT scan of the orbit is indicated to rule out orbital fractures.[21] Orbital fractures are commonly seen with blunt trauma to the orbit. Careful evaluation of the eye by an ophthalmologist prior to surgery is important and presence of diplopia with restriction of motility needs to be determined. Differentiating between a paretic muscle and an entrapped muscle that is restricting motility (leash effect) is important. Timing of repair is controversial, with some experts suggesting initial aggressive surgical repair[22,23] and others suggesting delay of repair until the orbit has been re-evaluated in 10-14 days.[24] Inferior orbital fractures need only be repaired if there is diplopia within the central 20° of gaze, significant enophthalmos, or recession of the globe into the maxillary sinus. Too often, surgery is performed immediately, not allowing time for orbital edema to subside so that a better evaluation of motility can be performed.

Intraorbital Foreign Body

If an orbital foreign body is noted on CT scan or on x-ray, proper history to determine type of foreign body is extremely important. If the foreign body is composed of organic matter, such as wood or vegetable matter, it must be removed to prevent orbital cellulitis. Foreign bodies made from copper are not well tolerated and also should be removed. Copper alloys from brass and bronze are fairly well tolerated and inert substances, such as glass, plastic, iron, lead, steel, and aluminum, are well tolerated and can be left in the orbit. BBs and shotgun pellets are typically made of 80-90% lead and 10-20% iron.

Corneal Foreign Bodies

Corneal foreign bodies are the most common workplace injury, accounting for nearly 35% of all eye injuries at the workplace.[25] There is usually a history of grinding wheel work or metal upon metal contact. Thorough examination of the cornea and the anterior chamber with a slit lamp is imperative to rule out intraocular foreign bodies. Careful examination of the conjunctiva under the upper and lower lids up to the fornix is necessary to remove particles trapped in the lid conjunctiva. Superficial foreign bodies may be removed by irrigation but foreign bodies embedded in the cornea need to be removed at the slit lamp using a 25-gauge needle or foreign body spud. Residual rust and material must be removed with a foreign body bur. If the foreign body is in the visual axis, it would be

Table 4. Commonly Encountered Intraocular Foreign Bodies

Class of Foreign Body	Activity	Management
Copper	Severe inflammatory	Remove immediately
Glass	Inert	Remove
Iron	Severe inflammatory response	Remove immediately
Lead	Mild inflammatory	Remove
Nickel	Mild inflammatory	Remove
Organic—wood, plants, and cilia	Severe inflammatory	Remove immediately
Plastic	Inert	Remove
Steel	Severe inflammatory	Remove immediately
Stone	Inert	Remove

prudent to refer to an ophthalmologist and have them remove the foreign body to limit size of the scar and, thus, decrease risk of vision loss. After removal, a cycloplegic agent (Cyclogyl 1%) and a broad spectrum antibiotic ointment is given to prevent corneal infection until the defect is healed.

Intraocular Foreign Bodies

Patients with a history of trauma with a sharp object or high-speed missile are at high risk for corneoscleral laceration and possible intraocular foreign body. Signs, such as a shallow anterior chamber, subconjunctival hemorrhage, hypotony, hyphema, cataract, and decreased vision can all point to a ruptured globe. Orbital CT scans with thin slices are the gold standard for detection of intraocular foreign bodies. Presence of an intraocular foreign body on CT scan requires an emergent referral to an ophthalmologist. The globe should not be manipulated excessively and an eye shield should be placed. Substances, such as copper, iron, steel, and organic material, need to be removed immediately because of the risk of toxicity to the retina or endophthalmitis. (*See Table 4*.) Patients should undergo a pars plana vitrectomy and removal of the foreign body.[26] Prognosis for vision is poor—especially if the foreign body has imbedded in the retina because of the risk of proliferative vitreoretinopathy (PVR).[27] In patients with PVR, significant scar tissue develops on the retinal surface and can result in a tractional retinal detachment with poor visual outcomes. All patients with an intraocular foreign body will have a corneal or scleral laceration and will need surgical repair of the laceration. (*See Corneal and Scleral Lacerations, page 253*.)

Corneal Abrasions and Erosions

In patients complaining of severe pain, erythema with edema of the eyelids, and photophobia, one must strongly consider corneal abrasion in the differential diagnosis. The patient may indicate a history of trauma, contact lens wear, herpes infection, or acute onset of pain upon awakening. Patients should be evaluated with the slit lamp using both regular light and cobalt blue light with fluorescein dye in a topical anesthetic. If an epithelial defect exists, it will take up the dye and fluoresce yellow against the blue background illumination. The examiner should have the patient blink his eyelid. If there is pooling of the dye without an abrasion, the yellow fluorescein will be displaced. With an abrasion, the region of yellow fluorescein will remain fixed on the cornea. Care must be taken to differentiate between a corneal abrasion and the dendrite seen in *Herpes keratitis*. Treatment with broad spectrum antibiotic drops (sulfacetamide 10% or polytrim) and cycloplegic drops (Cyclogyl 1%) are recommended. Currently, there are significant questions as to the benefits of a pressure patch over no patch.[28,29] If patients are not patched, topical nonsteroidal agents, such as diclofenac or ketorolac, are recommended for pain control.[30,31] Contact lens-associated abrasions need gram negative coverage with an aminoglycoside and a cycloplegic without patching. These abrasions need to be carefully monitored to ensure that a corneal ulcer does not occur.

Patients with a history of acute onset of pain and redness upon awakening frequently have a history of a previous large corneal abrasion. In these patients, the adhesion complexes between the epithelium and the basement membrane have been damaged, resulting in recurrent corneal erosions. Slit lamp exam reveals either a full thickness epithelial defect or an epithelial irregularity with mobile epithelium. The epithelium needs to be debrided and treatment is the same as for a corneal abrasion. After the epithelium is healed, copious lubrication is necessary for 6-8 weeks—especially lubricating ointment at night. If erosions recur, than surgical intervention to increase adhesion complexes is necessary.

Conjunctival Lacerations

Sometimes, patients with mild pain, red eye, foreign-body sensation, and a history of trauma will only have conjunctival laceration on careful examination of the globe. Exploration of the site and possible CT scan of the orbit may be indicated to rule out rupture of the globe. The laceration limited to the conjunctiva will rapidly heal and may need antibiotic ointment for 4-7 days. Large laceration (> 1.5 cm) may be sutured but most need no surgical repair. These patients should be re-examined within one week if the laceration is large.

Corneal and Scleral Lacerations

Lacerations of the globe can occur due to blunt trauma, missiles, or sharp objects. The sclera and cornea are usually resistant to blunt trauma but, with sufficient force, scleral rupture can occur. Compressive forces rarely cause rupture at the impact site, but ruptures actually occur at a remote site where the sclera is the thinnest. The sclera is the thinnest at the junction of the cornea and the sclera (limbus) and posterior to the insertion of the rectus muscles. Usually, scleral ruptures occur in the superonasal and superotemporal quadrants, are solitary, and extend from the limbus to the equator of the globe. Laceration due to missiles and sharp objects occur mostly in the front of the eye and can involve both the cornea and sclera. Signs of rupture include hyphema, cataract, vitreous hemorrhage, visual acuity of light perception or no light perception, ocular hypotony, and subconjunctival hemorrhage. If a laceration of the cornea and sclera is suspected, place a shield over the eye and, if one is not available, fashion one with the bottom of a styrofoam cup and obtain an ophthalmology consult immediately. Do not attempt to finish the exam as this may cause further extrusion of ocular contents. An orbital CT scan to rule out intraocular foreign bodies is highly advisable, and prompt surgical repair with proper ophthalmic follow-up is needed. Injuries greater than 20 mm in length and injuries caused by blunt trauma or missiles generally have a poor visual prognosis.[32] After primary repair of the lacerations, vitreoretinal surgery to re-attach the retina or remove retinal traction has greatly improved visual outcomes.[33]

Traumatic Iritis and Hyphema

The four phases of blunt injury are compression, decompression, overshooting, and oscillations.[34] Anterior-posterior compression results in equatorial expansion, shortening of the visual axis, and posterior displacement of the lens and iris. Extreme stretching of the ocular tissue results in specific types of injury to iris, trabecular meshwork, ciliary body, lens, retina vitreous, choroid, sclera, and optic nerve. The net result can be a breakdown in the blood-aqueous barrier and the blood-retinal barrier. Depending on the extent of the damage, it can result either in traumatic uveitis or in traumatic hyphema.

If the patient complains of pain, photophobia, and tearing, a careful slit lamp examination must be done to check for anterior flare (protein) and cells (white blood cells). Since the signs for uveitis can be subtle, referral to an experienced eyecare provider is preferred.

Flare and cell in the anterior chamber indicates traumatic uveitis, which runs a brief and benign course.[35] Cycloplegics drops (Cyclogyl 1% qid) and prednisone drops (Prednisolone acetate 1% qid) for one week will reduce the inflammation and recheck of the eye in one week is recommended.

Patients with blood in the anterior chamber or on slit lamp exam have a hyphema. These patients deserve a thorough examination by an ophthalmologist to rule out possibility of a ruptured globe. There is a high risk of rebleed within five days of the initial trauma and elevated intraocular pressures. Historically, patients were hospitalized for hyphema, but there appears to be no significant difference in rebleeding rates and clinical outcomes between hospitalized patients and those treated at home.[36,37] Close outpatient follow-up and strict bedrest is necessary to prevent rebleeds.[38] Cycloplegia with Atropine 1% bid and topical steroids (prednisolone acetate 1% qid) are given to enhance patient comfort but have not been shown to prevent rebleed or improve outcome. Antifibrinolytic agents, such as aminocaproic acid, can be used both systemically or topically to stabilize the clot for five days and decrease the rate of rebleed.[39-41] Patients with hyphema should be examined every day for five days post-trauma and then two days after cessation of aminocaproic acid to check for rebleed and elevated intraocular pressures. Approximately 5% of patients with hyphema will develop uncontrollable intraocular pressures, prolonged clot duration, and corneal blood staining. These patients will need surgical intervention to remove clot mass from the anterior chamber. Great care must be taken to avoid vision loss from uncontrolled glaucoma or corneal bloodstaining and pressure control is vital.

Retinal Edema, Retinal Tears, and Vitreous Hemorrhage

Traumatic forces can cause violent movement of the vitreous away from the retina, resulting in a coup or contrecoup lesion in the retina. Types of injuries most often encountered are retinal tears and nontearing retinal injury resulting in retinal edema or retinal hemorrhages.[42] Retinal edema, called Berlin's edema or commotio retinae, can be seen in the retinal periphery or the posterior pole. If it involves the macula or fovea, the patient will usually complain of decreased vision because of photoreceptor disruption.[43] Direct ophthalmoscopy may reveal a whitish gray central retinal lesion that may be difficult to note without a dilated exam. Patients that have sustained blunt trauma to the retina do not need emergent surgical intervention but need to be monitored by an ophthalmologist because they may develop sequelae, such as retinal pigment epithelium atrophy or macular hole.[44] If a macular hole develops, surgical intervention to repair the hole should be considered. Retinal hemorrhages should be observed and will be reabsorbed with time. They usually do not cause chronic vision loss.

Occasionally, patients will complain of decreased vision and there is a poor view of the retinal. If the vitreous is detached from the retina and a retinal vessel is broken, bleeding can occur in the vitreous cavity making it impossible to evaluate the retina with ophthalmoscopy. If there is no rupture of the globe, vitreous hemorrhages need to be monitored carefully with diagnostic ultrasound called B-scan to rule out retinal tears or retinal detachment. Patients should be monitored monthly until the hemorrhage resolves. Some retina specialists would perform a vitrectomy to improve vision and avoid other complications sooner than six months if the vitreous hemorrhage persists.

Traumatic retinal tears most commonly occur at the anterior edge of the retina, at its junction with the ciliary body, and are called retinal dialysis.[45] Often, the retinal dialysis is not visible at the time of injury due to its location and it may be overlooked on subsequent exams. Many retinal detachments associated with retinal dialysis are first diagnosed more than one year after the injury. The inferotemporal and superonasal quadrants are most frequently involved.[46] Careful history and fol-

low-up examination might prevent progression to a retinal detachment. If a retinal tear is seen, laser photocoagulation or freezing therapy will stabilize the retina. Patients who have a history of previous trauma, flashing lights, and a grey curtain over the eye are at high risk for a retinal detachment. Prompt referral to an ophthalmologist for an indirect ophthalmoscopic examination might prevent further delay in diagnosis and surgical repair. Visual prognosis is good when retinal breaks or detachments are diagnosed and repaired within six weeks of surgery.[47] The patient may undergo placement of air in the globe, scleral buckle, or pars plana vitrectomy for repair of retinal detachment.

Lens Subluxation/Dislocation

Compressive forces from trauma can cause dehiscence of zonules supporting the lens. Decentration of the lens with some partial zonular dehiscence is called subluxation. Total zonular disruption can lead to lens dislocation into the vitreous cavity or into the anterior chamber. Usually, the lens capsule is intact and there is minimal inflammation. In the majority of cases, observation and a thorough eye exam with refraction for visual rehabilitation is sufficient. However, in cases of subluxed lenses with the edge of the lens in visual axis or dislocated lenses with angle closure glaucoma, surgical intervention is necessary. Although many ophthalmologists still use intracapsular lens extraction, pars plana lens removal has had promising results.[48]

Optic Nerve Trauma

Patients with an injury to the optic nerve may have decreased visual acuity, visual field deficits, or a relative afferent pupillary defect. Examination of the pupils with the swinging flashlight test must be performed, but sometimes because of corneal opacities, hyphema, or lid edema, it is difficult to assess the injured eye. In this instance, checking the consensual response in the other eye will help the examiner determine the status of the nerve. Trauma to the nerve may be direct, such as in penetrating injuries due to projectiles or objects that lacerate the nerve. Orbital fractures and high velocity projectiles can also cause severe contusion of the nerve. The first due to compression by bone fragments and the latter due to dissipation of significant energy in close proximity to the nerve. Increased tissue pressure from orbital air or retrobulbar hemorrhage can cause optic nerve compression and tractional forces on the globe can completely avulse the optic nerve from the eye.[49,50] Usually, disc edema, hemorrhage, or avulsion can be seen with ophthalmoscopy.

Indirect trauma can occur when there is no initial ophthalmoscopic evidence of injury to the eye. Many times, the severity of injury is not proportional to the amount of vision loss and it can occur immediately or is delayed. Concussive forces shear axons at the lamina cribrosa at the entry of the nerve into the eye. Optic atrophy with pale optic nerve is then seen 3-6 weeks later. Generally, the prognosis for recovery of vision is poor in traumatic optic nerve injuries, but IV corticosteroids given for 3-5 days have been shown to be beneficial. Treatment should be immediately initiated.

Non-Traumatic Visual Loss

Acute, non-traumatic visual loss presents special diagnostic challenges. Important historical points include time of onset, quality and severity of visual loss, mono- or binocularity, duration of loss, and associated ocular and systemic diseases. It is helpful to think through the globe structure from anterior to posterior to sort out the various etiologies of acute non-traumatic visual loss. Corneal causes include corneal erosions (can be spontaneous or associated with corneal epithelial dystrophy's or bullous kerotapathy), infectious keratitis, or toxic reactions to chemicals or medications. Anterior chamber causes include acute iritis and spontaneous hyphema. Lenticular causes include cataract (usually posterior subcapsular type that can progress fairly rapidly and be "suddenly" noted by patient). Vitreous causes include spontaneous vitreous hemorrhage (often in diabetics with proliferative retinopathy) or pars planitis (idiopathic inflammation of vitreous usually found in young adults).

Retinal and optic nerve diseases are probably the most common causes of acute non-traumatic visual loss. (*See Table 5.*) Central retinal artery occlusion requires immediate ophthalmologic treatment to attempt to preserve vision.

Optic Nerve Disorders

Optic nerve dysfunction causes decreased visual acuity, color vision, and almost always presents with an afferent pupillary defect. The optic nerve can appear swollen, congested, and possibly pale, or it can look normal if the insult is retrobulbar. Optic neuritis is seen in younger patients (15-45 age group). Examination will show the above findings and there may be pain with extraocular movements. Vision can fall to low levels and then usually recovers, although often not completely. Treatment is based on results of the optic neuritis treatment trial. Oral steroids should not be used. High dose IV steroids may help hasten recovery in severe cases.[55] Anterior ischemic optic neuropathy (AION) is more common in older patients. It is found in both arteritic (age > 55) and non-arteritic (age 45-65) forms. The arteritic form is associated with giant cell arteritis. Patients may have profound loss of vision in one eye and may have an associated central retinal artery occlusion. If untreated, the second eye has a significant chance of also losing vision. Patients usually have temporal artery tenderness and systemic symptoms, such as jaw claudication, weight loss, and fever. Westergren sedimentation rate (> 47) and c-reactive protein level (> 2.45 mg/dL) can help with the diagnosis.[56] If a patient has a clinical presentation consistent with giant cell arteritis, high-dose systemic steroids should be started immediately and a temporal artery biopsy should be arranged. The non-arteritic form of anterior ischemic optic neuropathy is often found in patients with underlying systemic hypertension or diabetes mellitus. There is no proven treatment for non-arteritic AION.

Anisocoria

Anisocoria, or a difference in pupil size, can be a benign physiological state or a sign of a serious, life-threatening intracranial disease. Important historical points are any recent trauma, any previously noted anisocoria, any foreign material in eyes, and any headache, diplopia, or other neurological symptoms. An efficient, accurate decision tree is shown in Figure 1. Obtain ophthalmologic consultation if any doubt

exists as to the cause of the anisocoria. Any anisocoria in conjunction with a third nerve palsy ("blown pupil" on the ipsilateral side) requires emergent neuroradiologic workup and possible emergent neurosurgical intervention if an intracranial aneurysm is found.[57]

Diplopia

Diplopia, or double vision, can be separated into binocular diplopia (present only with both eyes open) or monocular diplopia (still present with one of the eyes closed). Other important historical points are time of onset, whether the diplopia is intermittent or constant, directions of gaze in which diplopia worsens, antecedent trauma, and any previous ocular surgeries, especially strabismus surgeries. During the examination, pay special attention to the pupils and extraocular motility exam. Occlude first the right eye, then the left eye to determine if any shifts or correcting movements occur. Alternating the occluder between the eyes rapidly can also help to elicit any abnormalities. Categorize the ocular alignment as normal, esotropic, exotropic, or vertical misalignment. Carefully check the full range of extraocular movements to determine if any visible muscle palsy is present. If the patient notes diplopia is worse or only present in certain directions of gaze, this can be a good clue of what muscles are malfunctioning. True monocular diplopia is usually caused by an abnormality of the cornea, lens, or central retina. A complete dilated examination is usually required to find the cause. Children and young adults can have sixth nerve palsies following head trauma or a viral illness, or it may signify an intracranial process. Aggressive workup with neuroimaging is indicated.[58] Acute third nerve palsies with pupil involvement in either young adults or the elderly may represent the effects of a posterior communicating artery aneurysm. Neuroradiologic workup and multiple sclerosis can present in young adults with any pattern of EOM involvement that can have a high degree of variability. Elderly patients often have sixth or fourth nerve palsies secondary to a microvascular insult. These patients often have hypertension, diabetes, or generalized atherosclerotic vascular disease. If

Table 5. Retinal Causes of Acute Non-Traumatic Visual Loss

	Age-related Macular Degeneration	Central retinal Artery Occlusion	Central Retinal Vein Occusion	Retinal Detachment
History	metamorphopsia to sudden loss of central vision, usually elderly	amaurosis fugax episodes, sudden loss of vision, middle age to elderly	rapid (over several days) to sudden loss of vision, usually elderly but can be seen in young adults	dark shadow over vision, often preceded by flashes and floaters
Visual Acuity	small decrease to loss of central vision	usually severe loss to count fingers or no light perception	moderate to severe loss	normal to severe loss depending on macular status
Pupils	no afferent defect	large afferent defect on affected side	often have afferent defect	afferent defect in relation to size of detachment
Retina	drusen (yellow spots), pigment clumping, hemorrhage	milky, swollen retina with cherry red spot over fovea, may see hollenhorst plaque	widely scattered retinal hemorrhage and whitish retinal infarcts	elevated, whitish, billowy retinal tissue—may see tear in retina
Optic Nerve	not affected	usually pale, may be swollen	swollen, congested vessels with hemorrhage	usually normal—may be obscured by detachment
Emergency Treatment	referral for fluoroscein angiogram within 24-48 hours	immediate ocular massage, IOP anterior chamber paracentesis	treat any IOP elevation	referral to ophthalmologist for repair
Long-Term Treatment	laser treatment for eligible lesions, long-term prognosis poor[18]	if not reversed acutely prognosis very poor; watch for neovascularization[19]	close observation for neovascular complications, possible laser tx[20]	pneumatic retinopexy or scleral buckling or vitrectomy used to repair[21]

they have no other neurological findings and a history of a predisposing condition, they can be observed for 3-4 months.[59] If they have other neurological findings, worsen, or have no resolution after 3-4 months, perform an MRI. Acute onset of diplopia or worsening diplopia in patients with Graves disease can signal new activity of the disease. This may require emergent evaluation and treatment (i.e., radiation, orbital decompression, systemic steroids) if decreased vision, increased IOP, or signs of optic nerve compression are present.

Summary

Patients who present with eye emergencies can have a large variety of pathology, ranging from benign to sight and life threatening. Accurate, timely diagnosis is needed and this requires careful slit lamp and ophthalmoscopic examination by the physician to determine whether an emergency eye consult is necessary. In patients with red eye, the physician must be able to rule out conditions that are site threatening prior to treating a patient for conjunctivitis. In a traumatic vision loss after an initial evaluation, the patient should be promptly referred to an ophthalmologist. In patients with suspected rupture of the globe or intraocular foreign body, one must obtain an orbital CT scan, place an eye shield, and let the ophthalmologist determine the extent of the injury. Sight threatening mistakes are usually made only when physicians attempt to manage ocular emergencies without the benefit of an experienced eye care specialist.

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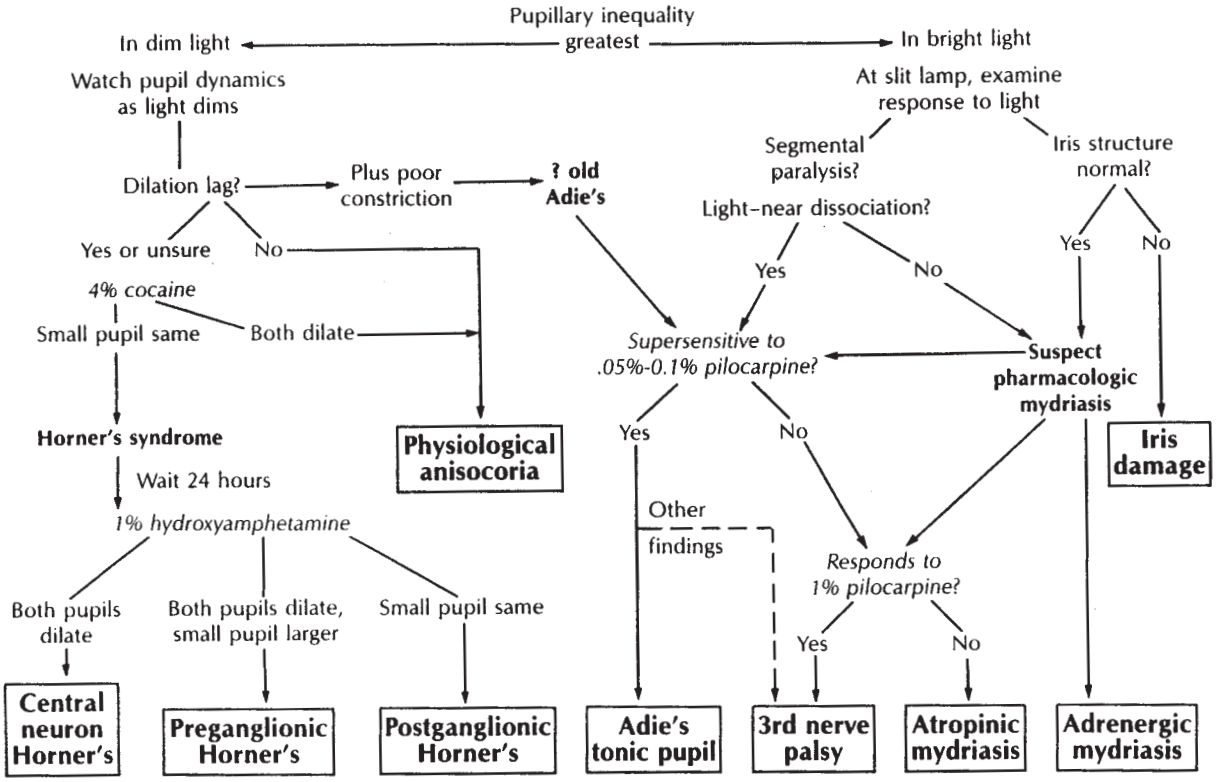
Figure 1. Anisocoria Flowchart



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Physician CME Questions

62. Which of the following chemicals will penetrate deeper into the globe and cause more damage?
    a. Fertilizer
    b. Bleach
    c. Vinegar
    d. Hydrochloric acid

63. Which of the following is *not* true about hyphemas?
    a. Rebleeding occurs most frequently in the first five days.
    b. Aminocaproic acid decreases the risk of rebleed.
    c. Patients must be hospitalized.
    d. Rebleed with increased IOP can cause corneal bloodstaining.

64. With regard to ocular and adnexal lacerations, which of the following are true?
    a. Marginal lid lacerations medial to the perimeter could possibly involve the canaliculus.
    b. Most conjunctival lacerations do not require surgical repair.
    c. Scleral lacerations due to blunt trauma occur most often at the limbus or in the area posterior to the insertion of the rectus muscles.
    d. All of the above

65. Orbital fractures need to be repaired if there is:
    a. diplopia in central 20° of the eye.
    b. ptosis of the globe.
    c. significant enophthalmos.
    d. All of the above

66. Traumatic retinal detachments are most often:
    a. seen with retinal flap tears due to vitreous separation
    b. diagnosed at the time of injury
    c. in the inferotemporal and superonasal quadrant
    d. in the superotemporal and inferonasal quadrant

67. Pre-auricular adenopathy is found in conjunction with many viral eye infections and with which of the following bacterial infections?
    a. Staphylococcus
    b. Streptococcus
    c. Gonococcus
    d. Pseudomonas
    e. None of the above

68. Which of the following conditions require emergent treatment?
    a. Central retinal artery occlusion
    b. Ocular chemical burn
    c. Angle closure glaucoma
    d. Endophthalmitis
    e. All of the above

69. Which of the following retinal causes of visual loss most commonly presents with no afferent pupillary defect?
    a. Central retinal artery occlusion
    b. Retinal detachment
    c. Central retinal vein occlusion
    d. Age-related macular degeneration
    e. None of the above

70. The workup of children and young adults with acute sixth nerve palsies should include:
    a. patching of one eye to relieve their diplopia.
    b. observation to watch for resolution of palsy secondary to viral illness.
    c. westergren sedimentation rate and c-reactive protein levels.
    d. urgent neuro-imaging to rule out a compressive etiology.
    e. cerebral angiography to rule out posterior communicating artery aneurysm.

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