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Emergency Management of Traumatic Eye Injuries

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The tremendous physical and psychological sequelae of vision loss make it one of the most feared disabilities. Ocular complaints account for 3-6% of emergency department (ED) visits, with traumatic conditions accounting for two-thirds of these visits.[1,2] Emergency physicians are expected to recognize and manage all forms of vision-threatening ocular emergencies. Patients with vision-threatening ocular injuries often have multiple comorbidities, as in the case of multiple trauma patients, and emergency physicians often are forced to prioritize the management of these various injuries. Once an ocular injury is diagnosed, emergency physicians must be familiar with the criteria and the need for urgency for ophthalmologic evaluation of specific injuries.

In this review, the authors highlight key elements of ocular anatomy, patient history, and physical examination essential to the diagnosis and management of traumatic ocular injuries. They review current imaging modalities used in the evaluation of traumatic eye injuries, and discuss specific indications, limitations, and potential hazards of ocular imaging. They conclude with a comprehensive review of the diagnosis and emergent management of the more common ocular injuries seen in the ED. (See Table 1.)

— The Editor

Introduction

Anatomy. Globe anatomy may be divided into anterior and posterior segments. *(See Figures 1a and 1b.)* The anterior segment consists of the cornea, anterior sclera, conjunctiva, aqueous humor, iris, lens, and the anterior and posterior chamber spaces. The posterior segment consists of the vitreous body, retina, choroid, posterior sclera, and optic nerve. Additional anatomical areas include adnexal structures (eyelid, lacrimal gland, and canalicular system) and the bony orbit.

The cornea is comprised of highly organized, dense collagen layers, and is approximately 500-600 microns thick.[3,4] The sclera consists of less strictly oriented collagen lamellae, which give the sclera its opaque, white appearance. Anteriorly, the conjunctiva, the tenon capsule, and the underlying episcleral vasculature cover the sclera. Posteriorly, the sclera ultimately fuses with the

coverings of the optic nerve. The anterior segment may be divided into an anterior chamber (the space between the cornea and iris) and the posterior chamber (the space between the iris and lens). The anterior and posterior chambers are filled with transparent, protein-free aqueous humor, which provides oxygen and nutrients to the cornea and lens. The ciliary body is responsible for aqueous humor production, while the trabecular meshwork is responsible for its egress from the anterior chamber. Normal intraocular eye pressure (IOP) is 10-20 millimeters of mercury (mmHg). This pressure serves to maintain the structural integrity of the eye without causing any barometric optic nerve injury. The posterior segment's vitreous cavity is filled with approximately 4 mL of vitreous humor. The vitreous humor is a transparent gel composed of water, hyaluronic acid, and mucopolysaccharides.

Evaluation of the Ocular Trauma Patient

History. A detailed history aids in the assessment of injury severity and guides subsequent patient evaluation. If the initial history reveals that a patient has suffered a chemical injury to the eye, treatment should be initiated immediately. Further history and physical examination should be deferred, or performed in concert with immediate, copious irrigation of the eyes. The history should establish the pre-morbid visual acuity. Information about a patient's use of corrective lenses, ophthalmologic medications, tetanus status, and previous eye surgery should be obtained.[5-7] Even minor trauma may lead to corneal or scleral rupture in patients with a history of prior ocular surgery. In cases of blunt trauma, the nature, force, and direction of impact of the offending object are important factors for determining the extent of injury. If the patient reports exposure to high-speed projectiles (such as close proximity to pounding metal on metal), the physician should consider the possibility of an intraocular foreign body.[7] When faced with a penetrating injury, it is important for the emergency physician to ascertain the composition of the potentially retained foreign body. Certain foreign bodies elicit an intense inflammatory reaction or lead to infection within the globe, while others are well-tolerated. Patients involved in automobile accidents with airbag deployment may suffer a variety of blunt ocular injuries, and may sustain alkaline chemical exposures as well.[5]

Physical Exam. With few exceptions, the physical exam should be performed in a systematic manner to reduce the chance of missing injuries. The primary elements of the examination are visual acuity, external examination of the orbit and adnexal structures, ocular motility, pupillary exam, visual field testing, anterior segment examination (including slit lamp exam), posterior segment evaluation (including direct ophthalmoscopy), and IOP measurement. These primary elements will be discussed later. A more comprehensive discussion of how to perform a complete ocular exam may be found in an excellent article by Juang and Rosen.[6]

Visual Acuity. Visual acuity, the vital sign of the eye, should be the first step in virtually all ophthalmologic exams and never should be excluded. Although often overlooked in the setting of ocular trauma, visual acuity is an important predictor of visual outcome.[8] A review of cases involving vision-threatening conditions misdiagnosed by emergency physi-

Table 1. Analysis of 1158 Cases of Ocular Trauma Presenting to a Casualty Department[2]

DIAGNOSIS	NUMBER	PERCENT
Corneal foreign body	405	35.0%
Corneal abrasion	229	20.0%
Superficial foreign body	162	14.0%
Chemical exposure	89	7.7%
Radiation injury	64	5.5%
Periorbital contusion	59	5.1%
Conjunctival trauma	55	4.8%
Hyphemia	28	2.4%
Lid laceration	26	2.3%
Traumatic iritis	16	1.4%
Thermal burns	12	1.0%
Globe perforation	11	1.0%
Vitreal hemorrhage	2	0.2%

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Figure 1a. The Eye

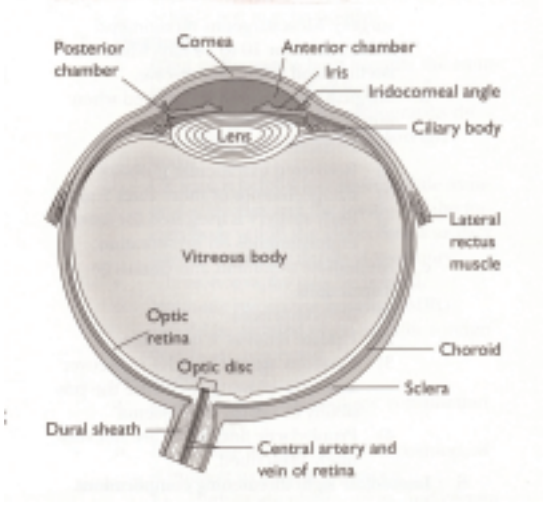

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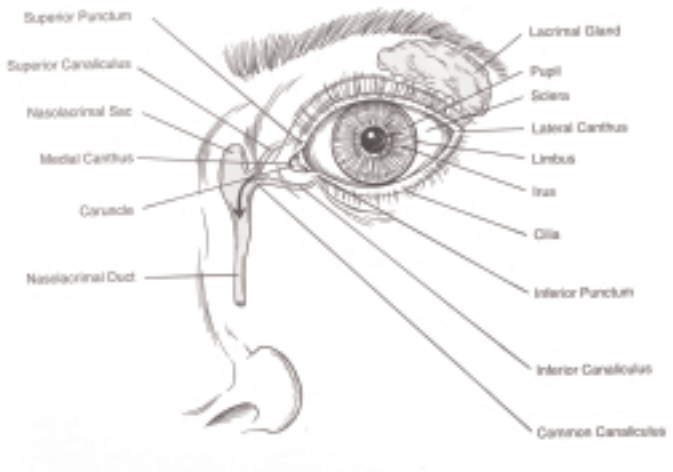
Figure 1b. The Eye and Adnexa



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cians revealed that visual acuity documentation frequently was absent from the medical record.[9] Visual acuity should be determined independently in each eye. The best-corrected visual acuity should be obtained using, if necessary, the patient's glasses, pinhole testing, or even a hand-held ophthalmoscope. Topical anesthetics may facilitate visual acuity testing in patients with acute eye pain and blepharospasm. If an eye chart is unavailable, other forms of typed print, such as a magazine, may be substituted. If a patient is unable to visualize typed print, visual acuity should be recorded by having the patient count fingers at a specified distance, having him detect hand motion, and by determining light perception (or lack thereof). An E chart or picture chart may be used with cooperative but illiterate patients and preschool children.[5,6] Visual acuity deficits found after correcting for refractive error mandate further evaluation.

Orbit and Adnexal Structure Examination. The physician should observe the orbit for the presence of any asymmetry. Proptosis (protruding eye) may suggest an orbital compartment syndrome, while enophthalmos (sunken eye) could indicate a globe rupture or an orbital fracture. These conditions are assessed best by looking from above the head downward toward the eyes.[5] Ptosis may indicate a third- or seventh-nerve palsy, levator palpebrae injury, or post-traumatic Horner's syndrome (miosis, ptosis, and anhidrosis). The physician should palpate the zygoma and orbital rim, noting any deformity, point tenderness, or subcutaneous emphysema. Periorbital subcutaneous emphysema is highly suggestive of an orbital or nasal antrum fracture. The eyelids should be assessed for movement, swelling, ecchymoses, lacerations, margin integrity, or occult foreign bodies. Medial eyelid lacerations should be evaluated for injuries to the lacrimal drainage system (canaliculi). The presence of fatty tissue within a lid laceration suggests orbital penetration with fat prolapse. The lower eyelid may be pulled down and the upper eyelid everted over a cotton swab to rule out adherent conjunctival foreign bodies.[10] The lid should not be manipulated if globe rupture is suspected. Foreign bodies should be removed only if the entire foreign body can be visualized, as one end may have penetrated the globe or orbit. A traumatized eye may exhibit blepharospasm or blepharedema, making the examination difficult. Edematous eyelids may be retracted manually; this should be followed by an application of a topical anesthetic. In cases of marked blepharedema, use of a Desmarres retractor or folded paper clip may be necessary to retract the eyelid.

Ocular Motility. The corneal light reflex should be at the same relative position on each cornea and the patient should be able to move his eyes through the six cardinal positions of gaze (left, right, up and right, up and left, down and right, down and left). Limited extraocular motility may indicate the presence of orbital fractures, cranial nerve injury, extraocular muscle injury, or restriction of globe motility from intraorbital edema or blood. It is important to determine whether patients complaining of diplopia are suffering from monocular vs. binocular diplopia. Diplopia that persists when the uninjured eye is covered suggests a refractive error (monocular diplopia). Monocular diplopia most often is the result of a corneal irregularity, lens abnormality, iridodialysis, or retinal detachment. Diplopia that resolves with occlusion of either eye is suggestive of a defect in coordinated eye movement (binocular diplopia). Binocular diplopia most often is the result of orbital fractures, cranial nerve injury, extraocular muscle injury, or orbital compartment syndromes.

Pupillary Examination. An examination of the pupil and iris should note size, shape, symmetry, and reaction to light. Pupil size should be recorded in millimeters. Blunt trauma may cause traumatic miosis (constricted pupil) or mydriasis

(dilated pupil). A teardrop-shaped pupil is suggestive of globe rupture, with the apex of the teardrop pointing to the rupture site.[5] Each pupil should be assessed for direct and consensual response to light stimulation (pupillary light reflex). Patients should be screened for an afferent pupillary defect (APD) utilizing the swinging light test. *(See Figure 2.)* In the setting of trauma, an APD is likely the result of an optic nerve injury, retinal injury, or extensive vitreal hemorrhage.

Visual Field Testing. Visual field testing will detect disorders affecting the retina, optic nerve, optic chiasm, and visual cortex. Regardless of visual acuity, patients with visual complaints always should be screened for visual field defects. Confrontational visual fields are measured one eye at a time, and can detect segmental retinal detachments (often a horizontal defect) or even intracranial pathology (usually a vertical defect). Care must be taken to ensure that the non-examined eye of the patient is covered completely.

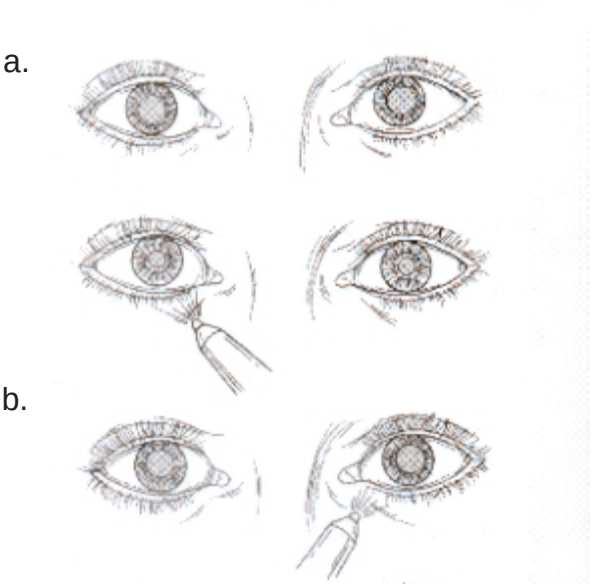
Anterior Segment Examination. General inspection and slit lamp examination will detect injuries to the conjunctiva, sclera, cornea, iris, lens, and anterior chamber. The conjunctivae and the sclera should be examined for injection, bleeding, lacerations, chemosis (swelling), exposed tissues (darkly pigmented uveal tissues), and foreign bodies. The presence of bloody chemosis or circumferential subconjunctival hemorrhage suggests open-globe injury. The cornea should be examined for lack of clarity, surface irregularities, and foreign bodies. Fluoroscein staining of the cornea is helpful in diagnosing corneal epithelial defects. Removal of contact lenses prior to the application of fluoroscein will prevent permanent staining of the contact lenses. In cases of possible globe penetration, a Seidel test should be performed. The iris is inspected for color, irregularities, tears, and evidence of prior surgical procedures. Inspection of the lens should reveal it to be clear and centered immediately behind the pupil. Traumatic subluxation of the lens often manifests as a dark crescent moon in the center of the pupil. The depth and contents of the anterior chamber require evaluation. Excessively shallow or deep anterior chambers (when compared with the opposite eye) suggest the possibility of globe rupture. The anterior chamber may reveal proteinaceous debris (flare), red blood cells (hyphema), or purulent exudate (hypopyon). Flare resembles dust specks floating in a bright, sunlit room, whereas the red blood cells that form a microscopic hyphema will look like brown dust.[6]

Posterior Segment Examination. Direct ophthalmoscopy will allow visualization of the anterior and posterior chambers, the retina, and the optic nerve. A fundoscopic exam should begin by documenting the status of the red reflex. A diminished red reflex suggests corneal edema, a cataract, vitreous hemorrhage, or a large retinal detachment. Dilation of the pupils with a topical mydriatic (e.g., 2 drops of 1% tropicamide) or cycloplegic (2 drops of 1% cyclopentolate) may facilitate the fundoscopic examination. Patients should be screened for contraindications to pupillary dilation; contraindications may include patients with significant head trauma, suspicion of globe rupture, or history of angle-closure glaucoma. A complete pupillary exam should be performed prior to initiating pupillary dilation. All patients with possible posterior segment injuries should be referred to an ophthalmologist for a complete posterior segment evaluation. Few emergency physicians possess the skills and tools (such as an indirect ophthalmoscope) to perform a complete, 360-degree retinal examination.

Intraocular Pressure Measurement. The IOP may be measured using a Schiotz tonometer or tonopen, or by applanation tonometry. Normal IOP ranges from 10 mmHg to 20 mmHg. It is important to exclude elevated IOP in the presence of hyphema or retrobulbar hemorrhage. IOP measurements should be deferred in cases of suspected open-globe injury. If a low IOP (< 5 mmHg) is documented in a patient with ocular trauma, an open-globe injury should be suspected.[11]

Ocular Trauma Imaging Techniques. Plain radiography, computed tomography (CT), ultrasound (US), and magnetic resonance imaging (MRI) have been used in the evaluation of orbital trauma and the search for foreign bodies. CT scanning is the study of choice in the evaluation of orbital and ocular trauma, and is more sensitive than plain films in the detection of foreign bodies.[12-14] In settings where CT is unavailable, conventional plain films may be used to screen for metallic foreign bodies, fractures, and sinus injury.

Figure 2. Afferent Pupillary Defect



The "swinging flashlight test" reveals an afferent pupillary defect of the left eye. **2a** shows a normal pupillary response; in **2b**, the test is positive when the affected pupil dilates in response to light.
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Standard CT examination should include both axial and direct coronal imaging; thin cuts (1 mm) are obtained for specific indications, such as localizing foreign bodies or viewing the optic canal. Contrast administration is not necessary. CT detects globe rupture, orbital fractures, intraocular and intraorbital foreign bodies, retro-orbital and intraocular hemorrhage, lens dislocation, and chorioretinal injuries.[9,15] Plastic, glass, cement, and wood are less radiopaque and are more difficult to detect with CT.[14]

When ocular examination is obscured by an opaque medium (corneal clouding or intraocular blood), US may provide detailed intraocular anatomic definition,[16] and may detect the presence of a retained foreign body (> 0.2 mm), retinal detachment, choroidal rupture, ruptured globe, vitreous hemorrhage, and orbital hemorrhage.[12,15] Because the transducer applies pressure to the globe, US should be avoided in cases of suspected open-globe injury.

MRI serves as a useful adjunct to CT imaging and is the procedure of choice for orbital soft-tissue evaluation.[12] MRI is the gold standard imaging technique for detecting optic nerve injury, and also can detect choroidal detachment, subperiosteal hemorrhage, vitreous hemorrhage, and non-metallic foreign bodies.[16,17] MRI is contraindicated in patients with metallic intraocular or intraorbital foreign bodies because of the risk of further injury from moving these objects.[18]

Anterior Segment Injuries

Corneal Epithelial Defects. Corneal epithelial defects, including corneal abrasions, are among the most common ocular injuries emergency physicians encounter.[2] A corneal epithelial defect may result from minor mechanical trauma, improper use of contact lenses, or intense exposure to ultraviolet light. Corneal epithelial defects present with eye pain, redness, foreign body sensation, tearing, photophobia, and blepharospasm. If a corneal defect is large or lies in the visual axis, it may diminish visual acuity. Infants with corneal epithelial defects may present with excessive crying.[19]

Application of a topical anesthetic (e.g., 2 drops of 0.5% proparacaine) to the eye of a patient with a presumed corneal epithelial defect will be both therapeutic and diagnostic. The topical anesthetic provides patients immediate relief from their corneal epithelial defect-related pain and supports the diagnosis of a corneal injury.[20] Topical anesthetics are not for repetitive use, as they suppress protective ocular blink reflexes, have been linked to delayed corneal healing, and may cause a toxic chemical keratitis.[21] The diagnosis of a corneal epithelial defect is made by visualizing a corneal defect that enhances under cobalt blue light following fluoroscein staining. The size (measured in millimeters), shape, and location of the abrasion should be documented. Multiple linear, corneal, epithelial defects (i.e., ice-rink sign) are suggestive of a foreign body adherent to the conjunctiva under the upper or lower eyelids (palpebral conjunctiva).

The primary objectives of corneal epithelial defect care have been to provide patients with analgesia and minimize the chance of bacterial superinfection. Analgesics such as acetaminophen or hydrocodone may be given orally or in the form of topical, non-steroidal agents (e.g., 1 drop qid ketoralac tromethamine ophthalmic solution 0.5%).[22,23] Despite the lack of supporting clinical evidence, cycloplegics and prophylactic antibiotic therapy have been the mainstay of corneal epithelial defect care. Short-acting cycloplegics (cyclopentolate) alleviate ciliary spasm and commonly are recommended to reduce the pain associated with corneal epithelial defects. In non-contact lens users, topical erythromycin ophthalmic ointment (tid) or polymyxinB/trimethoprim ophthalmic drops (qid) have been advocated.[24,25] Ointments offer better barrier function between the eyelid and corneal defect than drops do, but tend to blur vision. In contact lens users, there is a higher incidence of Pseudomonal species superinfection, hence antibiotic therapy should provide anti-Pseudomonal coverage (e.g., 0.3% tobramycin ophthalmic drops, 2 drops every 4 hours).[26,27] Contact lens users should be instructed to avoid wearing contact lenses until their defects have completely healed.

The practice of pressure patching eyes with corneal epithelial defects has no merit. Several prospective, randomized, controlled trials have failed to show any benefit from patching eyes in improving rate of healing or reduction of pain.[28-33] The practice of routine tetanus prophylaxis for uncomplicated corneal epithelial defects also has been challenged. Researchers recommended against the routine use of tetanus prophylaxis for uncomplicated corneal epithelial defects.[34] There is sufficient evidence to support the administration of tetanus prophylaxis for cases of open-globe injury.[35,36]

Most patients with corneal epithelial defects should be re-evaluated in 24 hours. In general, an uncomplicated corneal epithelial defect will have healed in 2-3 days, and long-term visual deficits are uncommon. Patients with extensive corneal epithelial defects affecting their visual axis warrant ophthalmologic evaluation within 24 hours of their injuries. This subset of corneal epithelial defect patients is at substantial risk for visual deficits should they develop any corneal infection and resultant corneal scarring. Any progression of pain or visual distortion should be interpreted with concern and result in immediate re-evaluation.

Corneal and Conjunctival Foreign Bodies. Patients with corneal or conjunctival foreign bodies may present with a foreign body sensation, pain, conjunctival injection, photophobia, tearing, blepharospasm, and blurred vision. Inquiry should be made about the possibility of a high-speed projectile mechanism of injury, as this is suggestive of an intraocular foreign body. Patient evaluation involves administering a topical anesthetic (which should provide immediate pain relief) and a detailed slit lamp exam. The majority of foreign bodies are very superficially embedded and easily visible on slit lamp examination. Foreign bodies that contain iron may produce a corneal stromal rust ring. Associated findings may include blepharedema, conjunctival injection, corneal infiltrate, and a mild anterior chamber reaction. If perforation is

not suspected, the upper and lower lids should be everted and the fornices swept to remove any additional conjunctival foreign bodies. Irrigating the affected eye with either normal saline or lactated Ringer's solution may rinse out particulate matter that escaped detection on physical examination.

A foreign body should be removed only after assessing its depth using a slit lamp. After topical anesthesia, an initial attempt at removal may be made with a sterile stream of saline or moistened cotton-tip applicator. If this fails, foreign body removal may be attempted using a commercial eye spud or 25-gauge needle, while utilizing slit lamp magnification. The patient must be cooperative and keep the eye still during removal. The removal of foreign bodies imbedded deep within the corneal stroma should be performed by ophthalmologists. Immediate removal of corneal foreign bodies is preferred, as this will minimize infection risk and foreign body-mediated corneal epithelial toxicity. A negative Seidel test following foreign body removal should be documented. A patient with a residual rust ring should be referred to an ophthalmologist. Ophthalmologists typically delay the removal of deep rust rings, as the rings soften and migrate to a more superficial location with time.[25] Aftercare following foreign body removal typically involves a cycloplegic, a topical antibiotic such as erythromycin ointment, and re-examination in 24 hours. Prophylactic antibiotics typically are prescribed for 3-4 days, until complete healing of any residual epithelial defects occurs.

Conjunctival Laceration. Patients with conjunctival lacerations will present with a history of ocular trauma. Symptoms include eye pain, foreign body sensation, and conjunctival injection.

The sclera is examined to exclude a scleral laceration or subconjunctival foreign body. Physical exam reveals a conjunctival laceration upon white light examination. The damaged conjunctiva typically folds over on itself at the site of the laceration, and an unobstructed view of exposed white sclera is noted. Varying degrees of conjunctival and subconjunctival hemorrhages may be noted. The damaged region of conjunctiva will fluoresce green following fluoroscein staining and cobalt blue light examination.

Patients with small conjunctival defects (smaller than 1 cm) typically will heal without surgical intervention. Patients with conjunctival defects larger than 1 cm often will need surgical repair.[37] Conjunctival laceration repair is best deferred to ophthalmologists. The repair typically is performed using 8-0 vicryl interrupted sutures. Aftercare involves topical antibiotic administration (e.g., erythromycin) for 4-7 days, until the affected area has healed. All patients with conjunctival lacerations deemed in need of surgical repair require ophthalmologic evaluation within 24 hours of injury.

Hyphema. Hyphema and microhyphema consist of varying degrees of anterior chamber blood. They typically represent bleeding from the iris root or ciliary body.[38] Patients typically present with a history of ocular trauma, diminished vision, and varying degrees of pain. Penlight examination will reveal larger hyphemas, while smaller hyphemas and microhyphemas will require a slit lamp evaluation for diagnosis. Physical findings include anterior chamber reaction, layering of red blood cells, or a clot. A microhyphema represents red blood cells suspended in the anterior chamber; layering of blood is absent. Hyphemas are graded based on the percentage of the anterior chamber that is filled with blood. *(See Table 2.)* The focus of the ocular exam is to quantitate the extent of the hyphema and exclude additional ocular injuries. Even small hyphemas may indicate significant intraocular injury. For instance, corneal epithelial defects and iris, lens, posterior segment, globe, and orbital injuries often accompany traumatic hyphemas.[39] The presence of hyphema in the absence of trauma should prompt investigation for an underlying blood dyscrasia (e.g., leukemia), coagulopathy, or melanoma of the iris.

Table 2. Hyphemia Scale

GRADE	HYPHEMIA SIZE
0	Microhyphemia (no layering of blood, circulating blood cells only)
1	Less than 33% of anterior chamber
2	Involvement of 33-50% of anterior chamber, but less than total hyphemia
3	Greater than 50% of anterior chamber, but less than total hyphemia
4	Total hyphemia ("eight-ball" or "blackball" hyphemia)

A well-documented secondary complication of hyphema is delayed hemorrhage.[38,39] This typically occurs within 2-5 days of the injury. It is believed to result from clot degradation and retraction. The incidence of secondary hemorrhage depends on the extent of the initial hyphema, treatment rendered, and patient population. Rebleed rates of 3-9% have been documented in patients with hyphemas who underwent outpatient treatment.[39,40] Additional complications of hyphemas include corneal staining, synechiae formation (adhesions between the iris and cornea anteriorly and lens posteriorly), glaucoma (secondary to the obstruction of aqueous outflow by blood cells), and optic nerve atrophy. The larger the hyphema, the worse the prognosis. The chance of recovering visual acuity of 20/50 or better is 75-90% with grade I hyphemas, 65-70% with grade II hyphemas, and 25-50% with grade III and IV hyphemas.[41,42]

All hyphema and microhyphema patients should have emergent ophthalmologic consultation. There is considerable controversy regarding the utility of hospitalization, bed rest, topical and systemic medications, and indications for surgical intervention in hyphema management.[43-45] The objectives of hyphema treatment include rapid resolution of the hyphema, patient comfort, and avoiding secondary complications. Traditional hyphema treatment involves bed rest with the head of

the bed elevated at least 30° (to facilitate settling of blood cells in the inferior anterior chamber), and eye rest (e.g., avoid reading). This may be performed at home by compliant patients.[39] Placement of an eye shield may prevent further ocular trauma. Patients should be instructed to avoid platelet-inhibiting agents such as aspirin. Hospitalization has been recommended in cases of extensive hyphemas (greater than 50%), elevated IOP, secondary hemorrhage, underlying blood dyscrasias (e.g., sickle cell disease), or non-compliant patients.[46,47]

Prevention of rebleeding has been a focus of hyphema treatment, and a variety of medications including estrogens, corticosteroids, and anti-fibrinolytics have been utilized for this purpose. Studies addressing the efficacy of these medications have produced mixed results.[48,49] Medical treatment of hyphema with miotics, mydriatics, cycloplegics, steroids, and antifibrinolytic agents should occur only following consultation with an ophthalmologist. African-American hyphema patients with sickle cell disease are at very high risk for hyphema-related complications.[50,51] They are intolerant of many medications used in hyphema treatment (e.g., carbonic anhydrase inhibitors), and often require additional therapeutic maneuvers such as aminocaproic acid drops. Surgery usually is reserved for delayed complications of hyphema, such as corneal staining, failure of a clot to resolve, or persistently elevated IOP, but may be considered earlier, as when dealing with a sickle cell disease patient.

Iris and Ciliary Body Injury. Blunt eye trauma can lead to irritation of the iris and ciliary body and incite an inflammatory reaction in the anterior chamber known as traumatic iritis (iridocyclitis). Symptoms include deep eye pain, photophobia, and blurred vision. Symptoms often are delayed, typically beginning 24-48 hours after injury. Evaluation should include a complete slit lamp exam, fundoscopic exam, and documentation of IOP. The key physical finding is a mild to severe anterior chamber reaction (cells and flare). Additional findings include: perilimbal injection (ciliary flush); photophobia; consensual photophobia (pain in the affected eye when light is shone in the opposite eye); and occasionally, decreased vision. Other clues to the diagnosis include continued patient discomfort after instillation of a topical anesthetic and pain with accommodation.[20,52] Treatment regimens include cycloplegic drops and topical steroids, which should be administered only in conjunction with ophthalmologic consultation. Patients with isolated traumatic iritis and no other associated injuries have a good prognosis.

Blunt eye trauma may result in injury to the iris, causing either pupillary constriction (traumatic miosis) or dilation (traumatic mydriasis).[53] Treatment is supportive, as these conditions either will resolve spontaneously or remain indefinitely if the damage to the iris is permanent. It is important to differentiate traumatic mydriasis from an APD. The pupil in traumatic mydriasis is dilated and poorly reactive to both direct and contralateral pupillary light exposure. A pupil with an APD will constrict normally when the contralateral pupil is exposed to light (consensual light reflex). Blunt eye trauma can avulse the iris from its root, resulting in a separation of the iris from the sclera (iridodialysis). Iridodialysis may create a second pupil and result in monocular diplopia. These patients eventually may require surgery to correct any cosmetic or persisting visual defect. All patients with iris or ciliary body injuries warrant ophthalmologic evaluation within 24 hours of their injuries.

Post-Traumatic Glaucoma. Glaucoma may present as an early or late complication of ocular trauma.[38] Acute post-traumatic glaucoma may occur secondary to impaired trabecular meshwork flow as a result of inflammatory cells or blood in the anterior chamber.[41,48] Occasionally, angle recession (narrowing) may occur as a result of trabecular meshwork displacement. Pupillary block and trabecular meshwork obstructions are the mechanisms by which hyphemas cause acute glaucoma. Patients typically complain of decreased vision, colored halos around lights, eye pain, headache, nausea, and vomiting. Critical signs include an elevated IOP (> 22 mmHg) and corneal edema. Additional physical findings include conjunctival injection and a minimally-reactive and mid-dilated pupil. All patients should have immediate ophthalmologic evaluation. Typical medical treatment of acute post-traumatic glaucoma involves topical beta blockade (e.g., ophthalmic timolol drops), carbonic anhydrase inhibitors (e.g., acetazolamide), osmotic agents (e.g., mannitol), and central alpha agonists (e.g., apraclonidine). Post-traumatic glaucoma patients failing medical management may be surgical candidates, per the discretion of the consulting ophthalmologist.

Lens Subluxation and Dislocation. Patients with lens subluxation or dislocation present with a history of ocular trauma, distorted vision, monocular diplopia, and pain. Critical findings include a displaced lens on direct ophthalmoscopy, phakodonesis (quivering of the lens), and iridodonesis (quivering of the iris). Additional findings include lens cataract, acute pupillary block glaucoma, and acquired myopia. Partial disruption of the lens' zonular fibers will result in lens subluxation. The lens is off-center, yet still remains partly within the pupillary aperture. When the lens' zonular fibers are disrupted completely, the lens becomes dislocated and is no longer seen through the pupillary aperture. A complication of lens dislocation is acute pupillary block glaucoma.[54] The dislocated lens may prevent aqueous humor from flowing from the posterior chamber through the pupil into the anterior chamber, where it normally exits the eye at the iridocorneal angle. Patients with lens subluxation or dislocation, with or without acute IOP elevations, need emergent opthalmologic consultation. Post-traumatic lens cataracts may occur following injuries that disrupt the lens capsule.[54]

Posterior Segment Injuries. Posterior segment ocular injuries may involve the vitreous body, retina, choroid, posterior sclera, or optic nerve. Injuries to posterior segment structures are vision-threatening. Blunt and penetrating forces may cause direct damage to the posterior segment in several ways. These include direct tissue damage, injury at a site distant to

the impact site (contracoup injury), and globe deformation.[55]

Patients presenting to the ED with a history of a visual deficit that cannot definitively be ascribed to anterior segment pathology should have a posterior segment examination by an ophthalmologist. While emergency physicians should be facile at performing a dilated fundoscopic examination, definitive diagnosis or exclusion of posterior segment injuries (through the performance of a 360-degree indirect ophthalmosopic retinal exam) is beyond their scope of practice.

Closed-globe posterior segment injuries are discussed in greater detail in the following section. Open-globe injuries that involve posterior segment structures are covered in the miscellaneous section of this manuscript.

Vitreous Cavity Hemorrhage. Vitreous cavity hemorrhage may occur from a variety of mechanisms. These include iris injuries, ciliary body trauma, vitreous body detachment, retinal vessel injury, and choroidal rupture.[56] Patients with vitreal hemorrhages may present complaining of varying degrees of vision loss and pain. Extensive vitreal hemorrhages may cloud the macula and obscure the red reflex. These patients may present with profound loss of vision, perhaps having light perception only. Additional physical findings may include a relative APD. Patients with less extensive vitreal hemorrhages may complain of floaters, cobwebs, and hazy or blurred vision.

Visualizing blood within the vitreal cavity with direct ophthalmoscopy makes the diagnosis of vitreous cavity hemorrhage. It is important to document a normal IOP in patients with posterior segment hemorrhages, as they may develop acute glaucoma. Vitreal hemorrhage treatment involves bedrest with the head of the bed elevated, a protective eye shield, analgesics (avoiding aspirin and non-steroidal anti-inflammatory drugs), and immediate ophthalmologic consultation. All vitreal hemorrhages in the setting of ocular trauma should be assumed to be secondary to retinal injury until proven otherwise. These patients require a dilated fundoscopic evaluation with an indirect ophthalmoscope to exclude concurrent peripheral retinal or choroidal injury. Patients with large posterior segment hemorrhages (opaque media) often require US scans to exclude a retinal detachment. A majority of isolated vitreal hemorrhages are treated expectantly and resolve spontaneously.

Retinal Injuries. Ocular trauma may result in a variety of retinal injuries, including retinal breaks, retinal dialysis, retinal detachments, and chorioretinal rupture (sclopetaria).[57]

Patients with retinal injuries present with a history of ocular trauma (may be remote) and symptoms including floaters, photopsia (flashes of light), a curtain or shadow moving over the visual field, and varying degrees of vision loss. The key diagnostic findings are evident only after detailed dilated fundoscopic examination. Peripheral retinal breaks initially may be asymptomatic, only later to lead to retinal detachment. Emergency physicians must maintain a high suspicion for retinal injuries in patients with significant ocular trauma, as morbidity with delayed treatment of these injuries is high.

Definitive diagnosis of these injuries is well beyond the scope of a majority of emergency physicians. Emergency physicians are responsible for identifying patients with a mechanism compatible with retinal injury or symptoms suggestive of retinal injury and providing them with appropriate ophthalmologic consultation. A variety of acute interventions, including laser therapy, scleral buckling, and cryopexy, may prevent a retinal break from progressing to an extensive retinal detachment. These must be performed within 24-72 hours of injury to maximize their rate of success.[55]

Commotio retinae (Berlin's edema) was first described by Berlin and is an example of a closed-globe retinal injury resulting from a contracoup mechanism.[58] Commotio retinae is characterized by a transient whitening of the deep sensory retina following ocular trauma. The exact etiology of the retinal opacification is unclear, but it no longer is thought to be the result of retinal edema.[59] Patients present with varying degrees of vision loss. Patients with macular involvement will have more severe vision loss. Fundoscopic examination will reveal focal retinal opacification. The fundoscopic changes and visual symptoms often resolve in several days in milder cases, but may persist in more severe cases. There is no specific treatment for commotio retinae.

Traumatic Optic Neuropathy. Blunt or penetrating ocular trauma may damage the optic nerve (traumatic optic neuropathy). Patients with traumatic optic neuropathy typically note an abrupt and profound loss of vision and present with an APD. The optic disc often appears normal upon initial fundoscopic examination; optic disc pallor may take several weeks to develop following the injury. Optic nerve injury may be detected by utilizing a test of relative brightness — a bright light shown on both eyes will be interpreted by the patient as dimmer in the eye with the injured optic nerve.[60] Similarly, bright red objects shown to each eye independently appear gray or washed-out when viewed by the eye with optic nerve injury (red desaturation). The underlying etiology of nerve injury may be nerve compression, transection, or ischemic injury.[61] Traumatic optic neuropathy is typically a diagnosis of exclusion and is made only after other causes of severe vision loss, such as severe retinal pathology, are excluded by an ophthalmologist. Immediate evaluation involves ruling out alternative etiologies of vision loss, as in the use of orbital imaging to exclude optic nerve compression. Some ophthalmologists may initiate a high-dose glucocorticosteroid treatment regimen, while others may attempt surgical decompression of the optic nerve if impingement is suspected.[62]

Adnexal and Orbital Trauma

Eyelid Trauma. All patients with eyelid trauma warrant careful ocular examinations. Globe injury should be suspected in cases of full-thickness lacerations, puncture wounds to the eyelid, or orbital septal lacerations with orbital fat prolapse. Eyelid injuries that involve the levator aponeurosis of the upper eyelid or superior tarsus muscle may result in ptosis. The repair of partial thickness, non-marginal eyelid lacera-

tions is within the scope of emergency medicine physician practice. These lacerations may be repaired utilizing the standard technique and 6-0 or 7-0 non-absorbable suture repair. Lacerations that extend through all layers of the eyelid should be referred to an ophthalmologist, as these injuries are a sign of potentially more serious injury such as globe penetration. Aftercare consists of topical antibiotic ointment for 48-72 hours and suture removal in 4-5 days. Tetanus status should be updated as needed. Any laceration involving the lid margin, orbital septum with fat prolapse, levator apparatus, or the lacrimal drainage system (punctum, canaliculi, or common duct) should be referred to an ophthalmologist for management. These injuries pose a high risk for complications, including disfiguring lid notching and epiphora.[63] A sterile gauze pad moistened with sterile saline placed over the wound will prevent desiccation pending immediate ophthalmologic consultation.

Orbital Compartment Syndrome. An acute orbital compartment syndrome (OCS) may be defined by an acute elevation of intraorbital pressure with resultant ocular dysfunction. Orbital compartment syndromes have been described in multiple clinical settings. Emergency medicine physicians will most likely encounter post-traumatic retrobulbar hemorrhages leading to an OCS.[64,65] Orbital compartment syndromes have been documented following blepharoplasty, retrobulbar anesthesia, orbital and sinus surgery, orbital fractures with intraorbital emphysema, and spontaneous subperiosteal and retrobulbar hemorrhages.[66-72] Orbital compartment syndromes also may occur as the result of chronic and progressive disease processes (e.g., malignant exophthalmos).[73] Though the exact mechanisms by which acute orbital compartment syndromes result in blindness remain speculative, prompt recognition and treatment is needed if irreversible vision loss is to be avoided.[74-76] Intraorbital pressure elevation leads to irreversible optic nerve damage, retinal ischemia, or both. Experimental studies have demonstrated that irreversible ischemic injury to the retina may occur within 90 minutes of vascular insufficiency.[77-80]

The globe is located within the bony orbit of the eye and is encased in a fascial envelope. The medial and lateral canthal tendons provide structural fixation of the eyelids to the orbital rim. The lateral canthal tendon (LCT) is located posterior and inferior to the lateral canthal fold. It originates from the lateral tarsal plates and attaches approximately 9.7 mm (standard deviation [SD] ± 0.8 mm) below the frontozygomatic suture at the lateral orbital tubercle.[81] Immediately superficial to the LCT is Eisler's pocket, a collection of adipose tissue. The check ligament of the lateral rectus muscle is located posterior to the LCT. Because the orbit is a closed space, any rise in intraorbital contents (e.g., retrobulbar hematoma) or orbital volume loss (e.g., orbital fracture) will result in an elevation of intraorbital pressure. The globe itself may partially accommodate some of the elevation in intraorbital pressure by prolapsing forward. This will result in ocular pain and proptosis. The intraorbital pressure rises dramatically as the orbit approaches maximal distension. This rise in intraorbital pressure may result in chemosis, elevated intraocular pressures, and pupillary sphincter dysfunction. An ophthalmoplegia may arise when ocular muscles or the nerves innervating them are damaged.

The diagnosis of an OCS is based on clinical signs and symptoms. Patients typically present with ocular pain, proptosis, afferent pupillary defects, and diminished vision. Additional clinical findings include chemosis, elevated intraocular pressure, mydriasis, diminished retropulsion of the affected globe to direct manual pressure, ophthalmoplegia, and fundoscopic signs of retinal ischemia (rare). Once the diagnosis of an acute OCS is made, immediate lateral canthotomy and cantholysis is indicated within one hour of injury and ocular dysfunction [82-85] *(See Table 3.)* The primary therapy for an acute OCS is surgical intervention. Medical interventions aimed at reducing intraocular pressures (including mannitol, acetazolamide, topical beta-blockers, etc.) should be considered adjunctive therapy. The elevated intraocular pressures in OCS merely reflect elevated intraorbital pressures. Therefore, any attempts to decrease intraocular pressures will not reliably reduce orbital compartment pressures. Refractory cases of OCS necessitate emergent deep orbital-wall decompression.[70] Deep orbital-wall decompressions are operative techniques that are best reserved for surgical specialists (head and neck specialists, for example). Adequate follow-up for these patients, with ophthalmologic consultation, is necessary. Patient's canthotomy site and LCT repair may be performed in a delayed fashion once the orbital compartment syndrome resolves.

Other Injuries

Open-Globe Injuries. Open-globe injuries may result from blunt trauma (causing globe rupture) or penetrating trauma (resulting in corneosclereal lacerations and punctures). Globe rupture is a full-thickness wound of the eye wall caused by a blunt object.[86] The blunt impact results in a momentary increase of the IOP, and the eye wall gives out at its weakest site, usually the corneoscleral junction or the insertion of the extraocular muscles.[53] Corneoscleral lacerations and punctures result from penetrating eye injuries and should be suspected in any patient with a penetrating facial injury in proximity to the eye.

Patients with open-globe injuries typically complain of eye pain, redness, swelling, or decreased vision. A normal visual acuity can occur, and does not exclude the diagnosis. The physical exam should begin with careful inspection of the globe. Pressure on the globe should be avoided, as this may result in expulsion of intraocular contents. Though many open-globe injuries are readily apparent with a penlight exam, others may be very subtle, requiring high-power magnification for diagnosis. Physical exam findings of open-globe injuries include an irregular or eccentric pupil (commonly teardrop-shaped if iris prolapses through the laceration), hemorrhagic chemosis (conjunctival edema), localized areas of erythema

Table 3. Orbital Compartment Syndrome Treatment

EQUIPMENT

Local anesthetic (e.g., 1% or 2% lidocaine with epinephrine); mosquito hemostat; iris scissors; tissue forceps; gauze pads

LATERAL CANTHOTOMY AND CANTHOLYSIS

1. Ensure adequate patient sedation and analgesia.
2. Position patient supine and begin irrigating the lateral canthal fold region with sterile irrigating solution.
3. Utilizing sterilizing technique, inject 1 cc of local anesthetic subcutaneously along the lateral canthal fold region. Anesthetize the tissue extending laterally from the canthal fold up to the orbital rim. Exercise caution to avoid inadvertant needle puncture to the globe.
4. Insert mosquito hemostats at the lateral palpebral fissure, with one blade anterior to and one blade posterior to the lid. Advance the tips of the hemostat laterally, until the orbital rim is encountered; clamp and compress the intervening tissue for at least 30 seconds (to minimize any ensuing bleeding).
5. Remove the hemostat and utilize the iris scissors to cut all tissue layers (from the skin down to the bulbar conjunctiva) along the lateral canthal fold, down to the lateral bone orbital rim (**lateral canthotomy**).
6. Retract the lower lid margin outward with a pair of hemostats or tissue forceps. This will facilitate identification of the LCT, which is located just posterior and inferior to the lateral canthal fold.
7. Dissection of the conjunctiva and fascial tissues with hemostats or iris scissors will be necessary to identify the LCT. A pocket of adipose tissue (Eisler's pocket) will be encountered beneath the superficial fascial planes. The LCT lies just posterior to this adipose tissue collection. Once identified, the LCT must be completely severed (**lateral cantholysis**).

and conjunctival injection, an unusually deep or shallow anterior chamber, and hyphema.[10] Additional findings may include limitation of extraocular movement, enophthalmos, vitreal hemorrhage, and chorioretinal injury. Corneoscleral lacerations commonly are located on the inferior aspect of the globe, due to the reflex upward-rotation of the globe while blinking (Bell's phenomenon). A positive Seidel's test, the streaming of fluorescein from a focal site on slit lamp exam, confirms the leakage of aqueous humor from an open-globe injury. Although a low IOP (< 5 mmHg) suggests the diagnosis of an open-globe injury, tonometry should be avoided in any patient with a potential open-globe injury.[87]

Examination by the ED Physician of a Suspected Open-globe Injury. The injured eye should be covered with a Fox shield, and immediate ophthalmologic consultation obtained. Liberal use of antiemetics, analgesics, and sedation may minimize expulsion of the intraocular contents and may be vision-saving. Patients should not have anything to eat or drink, have their tetanus status addressed, and receive a dose of broad-spectrum prophylactic antibiotics. Suggested parenteral regimens include ceftazidime (1 g every 8 hours) and vancomycin (1 g every 12 hours) for 48 hours following open-globe injuries. Ciprofloxacin (400 mg IV every 12 hours) may be substituted for ceftazidime in the penicillin-allergic patient. This combination provides broad-spectrum coverage against gram-positive and -negative organisms, including *Bacillus* species. CT scanning of the orbit should be obtained if a retained foreign body is possible.

The use of depolarizing paralytic agents such as succinylcholine to facilitate endotracheal intubation in the setting of globe rupture is controversial. Since succinylcholine administration results in a transient rise in IOP, its use in the setting of an open-globe injury theoretically may lead to expulsion of ocular contents.[88,89] Despite this theoretical consideration, the use of succinylcholine in open-globe injury patients is commonplace.[90] One group used succinylcholine during rapid sequence intubation of 100 open-globe injured patients and reported no expulsion of ocular contents.[91] The few reports of ocular content expulsion associated with succinylcholine administration are in the form of poorly documented personal communications published in the 1950s.[92,93]

With the exception of small (< 1 mm), self-sealing corneal lacerations, most open-globe injuries will require surgery. The prognosis for final visual outcome is related to numerous factors. These include the initial presenting visual acuity, injury mechanism, and location and extent of injury.[94] The presence of an APD, lens injury, vitreous hemorrhage, or retained intraocular foreign body is associated with a worse visual outcome. Post-traumatic endophthalmitis is a devastating complication of open-globe injuries. Injuries involving intraocular foreign bodies, organic matter, and those with delayed presentation are at highest risk for developing endophthalmitis.[95-98]

Patients with unilateral penetrating globe injuries are at risk for developing a severe inflammatory uveitis (sympathetic ophthalmia) in the opposite, unaffected eye. Sympathetic ophthalmia is thought to arise from sensitization of the immune system to uveal antigens.[99] These patients typically complain of bilateral eye pain, redness, photophobia, and decreased vision. Symptoms may develop weeks to years after the inciting event. The earliest signs may be a loss of accommodation or a mild uveitis in the uninjured eye. Evidence suggests that the risk of sympathetic ophthalmia is significantly reduced if the injured eye is removed within 14 days of the initial injury.[99,100] Therefore, any severely traumatized eye

with no chance of recovering vision should be considered for enucleation. Once sympathetic ophthalmia is established, enucleation is no longer therapeutic and treatment revolves around the use of immunosuppressive therapy.

Intraocular Foreign Body. A patient with an intraocular foreign body typically will complain of eye pain and decreased vision, and report eye injury as the result of a high-speed projectile. Unfortunately, not all cases present in this manner. Intraocular foreign bodies should be suspected with any sanding, drilling, grinding, or hammering activity prior to eye injury. Metal splinters from hammering and glass splinters from shattering glass may enter the eye painlessly.[101] The initial ocular examination may appear deceptively benign, revealing only slight erythema and local discomfort. Visual acuity is typically decreased, but can be normal. Some corneal or scleral perforations are difficult to detect. Additional findings suggestive of an intraocular foreign body include conjunctival chemosis, hyphema, localized cataract, iris injury, pupillary asymmetry, vitreous hemorrhage, decreased IOP, or an aqueous humor leak (positive Seidel test). Impaled intraocular foreign bodies, such as a knife or wire, should be removed in the operating room.

Suspicion of an intraocular foreign body necessitates obtaining an orbital CT scan. CT is helpful for identifying intraocular injuries and delineating the position of foreign bodies within the orbit. While small pieces of wood, glass, or plastic may not be seen on CT, the vast majority of intraocular foreign bodies will be visualized. MRI may be a useful adjunct but should be avoided when metallic foreign bodies are suspected.[18] Neither CT nor MRI alone or in combination will detect all intraocular foreign bodies.[102]

Patients with intraocular foreign bodies warrant emergent ophthalmologic consultation. These patients typically are hospitalized and given nothing by mouth in anticipation of surgery. They should be fitted with eye shields, and their tetanus status should be updated as needed. Broad-spectrum prophylactic antibiotics are the standard recommendation in the setting of penetrating trauma, although there are no clinical studies supporting the efficacy of this intervention. Antibiotic choice and route of administration should be discussed with the consulting ophthalmologist. Intraocular foreign body management depends on the composition and location of the foreign body within the eye.[102,103] The reactivity of intraocular foreign bodies is highly variable. Wood, vegetable matter, and metals (i.e., iron, copper, and steel) typically incite an intense inflammatory reaction when left in the eye. BBs and pellets typically are composed of both lead and iron and are poorly tolerated intraocular foreign bodies.[104] These warrant emergent surgical extraction by the consulting ophthalmologist. Inert foreign bodies (e.g., glass, lead, plaster, rubber, silver, and stone) often are left in the eye if they are minimally symptomatic.[105] Many inert foreign bodies may cause toxicity by virtue of a coating or chemical additive. With prompt recognition and improved surgical techniques, the prognosis for penetrating orbital injuries has greatly improved. Unfortunately, due to associated chorioretinal injuries, visual outcome following BB and pellet gun injuries is uniformly poor.[106]

Patients found to have an intraorbital foreign body with no involvement of the globe have a varied course. Complications of orbital foreign bodies include proptosis, diplopia, cellulitis, or a chronic draining fistula. Removal of such foreign bodies is left to the discretion of the consulting ophthalmologist. Once again, the more reactive objects (wood, vegetable matter, copper, steel, and iron) are poorly tolerated, while inert objects that are minimally symptomatic may best be treated with a nonoperative approach.

Post-Traumatic Endophthalmitis. Endophthalmitis (intraocular infection) in the setting of ocular trauma is associated with a poor prognosis.[96,107] The precise incidence of post-penetrating ocular trauma endophthalmitis is difficult to define. Culture-proven post-traumatic endophthalmitis has been documented in 2.4-30% of cases.[97,108,109] Patients typically have a history of ocular trauma (most commonly penetrating) and complain of progressive loss of vision and pain. Diagnostic features include decreased visual acuity, anterior segment reaction with or without hypopyon, conjunctival injection, blepharedema, and chemosis. The most common organisms include *Staphylococcus epidermidis*, *Bacillus* species, Streptococcal species, and *Staphylococcus aureus*.[97,107] Less common pathogens include gram-negative bacteria, anaerobes, and occasionally fungal species.[96] Many infections are polymicrobial.

Emergent management involves systemic, topical, and intraocular antibiotic therapy. Topical broad-spectrum antibiotics, such as topical vancomycin and gentamycin or tobramycin, are applied in an alternating fashion every 30 minutes. Broad-spectrum systemic antibiotics, such as cefazolin, gentamycin, and clindamycin, are administered parenterally. Emergency physicians need to be aware of the high incidence of endophthalmitis secondary to *Bacillus* species.[96,97,107] *Bacillus* species infections may lead to irreversible vision loss in as little as 24 hours. They often are resistant to penicillin and cephalosporin antibiotic therapy. Emergency ophthalmologic consultation should be immediately obtained in all cases of suspected endophthalmitis. Patients are typically hospitalized, and preparations are made to have intraocular antibiotic therapy and possible surgical vitrectomy performed. Culturing intraocular fluid obtained by diagnostic paracentesis makes the definitive diagnosis of ophthalmitis. Additional diagnostic testing may include an orbital CT scan or US to exclude a retained intraocular foreign body.

Chemical Eye Injuries. Chemical eye injuries are a true ophthalmologic emergency. Ocular acid or alkali exposure may result in extensive damage to the cornea and anterior segment with resultant permanent visual impairment. While the cornea is often the area of focus, additional areas such as the eyelids and adnexal structures often are damaged. In general, alkali burns are more severe than acid burns.[110] Alkali burns cause liquefactive necrosis, saponify corneal proteins, and initiate corneal collagen destruction. Acid burns cause injury

Table 4. Ocular Chemical Injury Severity

GRADE	PHYSICAL EXAM	PROGNOSIS
1	Corneal opacification and limbal ischemia absent	Excellent
2	Cornea is hazy, but iris details visible. Ischemia of less than one-third of the limbus	Good
3	Total loss of corneal epithelium, stromal haze obscures iris details. Ischemia of one-third to one-half of the limbus	Poor
4	Cornea is opaque, with no view of the iris or pupil. Ischemia affects more than one-half of the limbus.	Dismal

through coagulation necrosis and tend to precipitate corneal proteins, thereby limiting their toxicity. Alkali injuries occur more frequently than acid injuries due to their presence in a variety of household cleaning agents and home construction materials.[111]

No time should be wasted on additional history and physical examination once a chemical eye injury is documented. Immediate therapy should be instituted. The initial treatment for a majority of ocular chemical injuries is immediate, copious irrigation.[112] There is no therapeutic difference between ocular irrigation of chemical injuries with normal saline, normal saline with bicarbonate, lactated Ringer's, and balanced salt solutions.[113] The lids should be retracted during irrigation and the stream of irrigating fluid should be directed onto the globe and conjunctival fornices. The fornices should be swabbed to remove any chemical particulate matter. Topical anesthesia will facilitate irrigation. Irrigation with a minimum of one liter of irrigating solution is recommended, and irrigation should continue until the pH of the tear film obtained at the inferior conjunctival fornix is neutral.[114] It may be difficult to return the pH to 7 precisely, but a general trend toward that number should be documented. Litmus paper or the pH indicator of a urine dipstick may be used for pH determination, testing several minutes after irrigation is ceased to allow for pH equilibration. No attempt to neutralize chemicals with either acids or alkalis should be made.

A theoretical concern with immediate ocular irrigation occurs in the setting of metallic sodium, metallic potassiumm or yellow or white phosphorous ocular exposure.[112] Irrigation of these chemicals has the potential to initiate more chemical injury. Despite this concern, authors still recommend copious irrigation of all chemical eye injuries, even when exposure to metallic sodium, metallic potassium, or yellow or white phosphorous is known.

Chemical eye injury severity is related to the type of chemical, surface area of contact, depth of chemical penetration, and duration of chemical eye contact. The ultimate prognosis correlates with the loss of corneal clarity and degree of limbal ischemia. Examination should assess the severity of chemical injury. *(See Table 4.)* If an epithelial defect is highly suspected but none is found on initial fluoroscein staining, the procedure should be repeated. Sloughing of the entire corneal epithelium may have occurred. This will expose the underlying Bowman's membrane, which is slow to take up fluoroscein. The prognosis for chemical eye injuries is linked to the initial injury severity. Patients with mild injuries (grade I) may be treated with a cycloplegic (although phenylephrine should be avoided due to its vasoconstrictor effect), a topical antibiotic such as erythromycin, and oral pain medication. These patients should be seen by an ophthalmologist daily until a healing trend is documented. Patients with moderate to severe injuries (grades II-IV) warrant emergent ophthalmologic assessment and, likely, hospitalization. Inpatient treatment will focus on lysis of adhesions, minimizing infection potential, and treatment of iritis and any elevated IOP. Long-term complications of chemical injuries include corneal scarring and neovascularization, adhesions of the lids to the globe (symblepharon), glaucoma, cataracts, retinal injury, and globe injury.

Airbag Injuries. While airbags have saved countless lives, they also have been implicated in causing a broad spectrum of injuries.[116] Airbags have caused periorbital abrasions, chemical keratitis, corneal epithelial defects, lens subluxations and cataracts, hyphemas, and globe ruptures.[117,118] Sodium azide is used as fuel to inflate deployed airbags; this process releases minute amounts of sodium hydroxide as a by-product.[119] Hence, airbag-related ocular trauma victims should be screened for alkaline-induced chemical eye injuries. Airbag-related ocular trauma victims deserve careful ocular examination, including pH measurement, slit lamp examination, fluoroscein staining, IOP measurement, and a funduscopic examination. Patients with acute visual deficits will need emergent posterior segment examination by an ophthalmologist.

Cyanoacrylate Glues. As cyanoacrylate dermal adhesive use has increased, emergency physicians have been faced with cyanoacrylate-associated ocular complications. The household superglues are monomers, while the medical superglues are higher alkyl derivatives with lower tissue toxicity.[120] Household superglues may cause a chemical keratitis upon corneal contact. Medical superglues typically bond with dry surfaces only, so they tend to congeal in the lower conjunctival fornix and cause a traumatic keratopathy.[121] A majority of the complications involve eyelid margin adhesions (ankyloblepharon) and occasional corneal cyanoacrylate precipitates. Anecdotal treatment of these complications involves placement of petroleum-based products, such as erythromycin ointment, bacitracin ointment, or petroleum jelly, onto the affected area. This serves to degrade the cyanoacrylate precipitate. Phenol should not be used for ocular cyanoacrylate removal, as it will cause a chemical keratitis. Forceful retraction of glued eyelids should be avoided, as it may compound the injury. Serious, long-term complications of ocular cyanoacrylate exposure have not been published.[121,122]

Globe Luxation. Traumatic globe luxation is an unusual but dramatic injury that occurs only with significant trauma to the globe. The globe can be avulsed entirely and lie in a position external to the orbit. Visual impairment with traumatic globe luxation may result from an optic neuropathy as well as a variety of associated anterior or posterior segment injuries. Obtain emergent ophthalmologic consultation in all cases of traumatic globe luxation. Early reduction is recommended to reduce traction on the optic nerve and may be achieved by applying gentle pressure to the globe while retracting the eyelids. Edema, retrobulbar hemorrhage, and loss of orbital integrity often make reduction outside of the operating room very difficult.

Shaken-Baby Syndrome. Shaken-baby syndrome is a proposed mechanism of child abuse that involves intracranial and intraocular bleeding with an absence of external cranial trauma. It is thought to arise from a whiplash mechanism, and is predominately seen in children younger than 2 years of age.[123-125] Ocular findings include preretinal, subretinal, intraretinal, and vitreal hemorrhages that typically are bilateral. The diagnosis is made when ocular findings are found in concert with the appropriate clinical history of abuse. The findings of retinal hemorrhages in an infant, while suggestive of shaken-baby syndrome, are not specific for abuse. Conditions such as trauma, blood dyscrasias, vasculitis, and meningitis have been associated with similar fundoscopic findings.[123] An underlying blood dyscrasia should be excluded, as children with idiopathic thrombocytopenic purpura (ITP) or leukemia often present with multiple ecchymoses and often initially raise the suspicion of abuse. The treatment of shaken-baby syndrome is supportive care.

Conclusion

Ocular trauma has the potential for devastating complications. Emergency physicians must have a comprehensive understanding of ocular anatomy, pertinent patient history, and physical examination findings which will affecy the decision to obtain ocular imaging and ophthalmologic consultation. A careful history and a meticulous physical examination will identify the majority of serious eye injuries and guide appropriate evaluations.

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CME Questions

To earn CME credit for this issue of Trauma Reports, *please refer to the enclosed Scantron form for directions on taking the test and submitting your answers.*

1. The most important aspect of the eye exam in the traumatized eye is:
   A. visual field testing.
   B. fundoscopic exam.
   C. measurement of intraocular pressure.
   D. assessment of extraocular movement.
   E. visual acuity.

2. Which of the following usually is most difficult to detect with CT?
   A. Globe rupture
   B. Wood foreign bodies
   C. Retro-orbital hematoma
   D. Lens dislocation
   E. Orbital fractures

3. Which of the following statements about corneal epithelial defects is correct?
   A. Contact lens wearers are at lower risk for *Pseudomonas* superinfection.
   B. Pressure patching reduces pain and speeds healing.
   C. Topical anesthetics reduce pain and support the diagnosis.
   D. Topical analgesics should be avoided.
   E. Patients with abrasions involving their visual axis may follow up after 72 hours.

4. Which of the following patients with hyphema may be treated as an outpatient?
   A. Microhyphema with an open globe injury
   B. Grade 1 hyphema (< 33%) in a 10-year-old
   C. Grade 2 hyphema (33-50%) with an elevated IOP
   D. Grade 3 hyphema (> 50%) with signs of rebleeding
   E. Grade 4 hyphema (total) in a cooperative patient

5. Which of the following statements concerning posterior segment injuries is correct?
   A. Injuries to the posterior segment structures are rarely vision-threatening.
   B. Vitreous cavity hemorrhage may be detected by tests for relative brightness or red desaturation.
   C. Ultrasound may be useful in excluding retinal detachments in a patient with vitreous cavity hemorrhages.
   D. Most emergency physicians are adequately trained to definitively diagnose retinal injuries.
   E. Traumatic optic neuropathy is suggested by floaters or a curtain or shadow moving over the visual field.

6. Patients with orbital compartment syndrome may present with which of the following?
   A. Pain
   B. Propotosis
   C. Afferent pupillary defect
   D. Diminished vision
   E. All of the above

7. Which of the following concerning open globe injuries is correct?
   A. A normal visual acuity excludes the diagnosis.
   B. Documentation of a decreased intraocular pressure is imperative prior to calling the ophthalmologist.
   C. Most penetrating eye injuries will seal on their own.
   D. Leakage of clear fluid (a positive Seidel test) confirms the diagnosis.
   E. A pressure patch should be placed while awaiting ophthalmologic evaluation.

8. The most important initial action in the treatment of chemical exposures to the eye is:
   A. checking visual acuity.
   B. determining the offending chemical.
   C. determining the pH of the conjunctival fornix.
   D. copious irrigation.
   E. neutralizing the offending chemical with an acid or base.

9. Ptosis in a patient with ocular trauma may indicate a:
   A. third nerve palsy.
   B. seventh nerve palsy.
   C. levator palpebrae injury.
   D. post-traumatic Horner's syndrome.
   E. All of the above

10. Ultrasound (US) may be used in all of these situations *except*:
    A. open globe injury.
    B. detection of retained foreign body.
    C. detection of orbital hemorrhage.
    D. detection of retinal detachment.
    E. detection of choroidal rupture.

In Future Issues: Pediatric Fluid Resuscitation