Trauma to the Eye

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Trauma to the eye represents approximately 3% of all emergency department visits in the United States. Rapid assessment and examination following trauma to the eye is crucial. A thorough knowledge of potential injuries is imperative to ensure rapid diagnosis, to prevent further damage to the eye, and to preserve visual capacity. Although the eye represents only 0.3% of the total surface area on the human body, loss of vision in one or both eyes has been classified as a 24% or 85% whole-person impairment or disability, respectively [1].

History and physical examination

The general principles of the routine ocular examination also pertain to an examination in the setting of trauma, but certain aspects of the examination deserve special attention. Triage and registration personnel should be instructed regarding the urgency of eye injuries and of the need for simultaneous treatment and triage. One always should “take a step back” when considering eye injuries and assess the entire patient. Life- or limb-threatening injury should be addressed initially. In addition, concomitant injury to the brain, spinal cord, or facial bones is common and should be ruled out in the appropriate setting. Historical details should include the timing, mechanism, and location of injury. If there is penetrating trauma, one should learn the energy and type of material involved; organic matter has a higher rate of infection, and metal sometimes can cause a reaction in the vitreous. In pediatric eye trauma, obtaining a history can be difficult; reluctance of the caregiver to provide basic historical information should raise concern for abuse [2]. Visual symptoms such as change in vision, floaters, flashing lights, pain, discharge, or diplopia should be noted. Prior ocular

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history such as previous visual impairment, tetanus status, and surgical history should be recorded. In addition one should ascertain if protective eye-wear was used.

Physical examination should be performed in a systematic manner to avoid missed injury. The examination can be accomplished in an external to internal fashion. Throughout the examination, care should be taken to avoid placing pressure on the globe, because pressure can cause herniation of intraocular contents if the globe is ruptured. Initially, one should examine the head, scalp, face, and periorbital tissues to assess for lacerations, lid edema, foreign bodies, or sensory deficits (such as infraorbital hypoesthesia in the setting of orbital blowout fractures). Tenderness to palpation around the orbital rim or step-offs should be noted as well. One should look for exophthalmos, enophthalmos, or deformity of the external eye structures. Visual acuity should be tested before manipulation of the eye. Both near and far vision of each eye should be tested separately, with the other eye occluded if possible. Visual acuity should be measured with the patient wearing spectacle correction or using a pinhole occluder if spectacles are not available. A Snellen eye chart or standardized near reading card should be used. Any written material or an intravenous bottle may be used. If the patient is unable to read, one can assess vision by having the patient count the examiner’s displayed fingers, detecting hand motion, and indicating light perception or lack of light perception. The conjunctivae should be examined for blood, chemosis (swelling), foreign bodies, and exposed tissue. Examination of the cornea should include fluorescein staining under cobalt blue light to look for irregularities and foreign bodies. Next, the internal structures, including the iris and pupil, should be inspected, noting size, shape, symmetry, and reaction to light. In addition to direct pupillary response, one should assess consensual response using the “swinging flashlight” test. When an afferent pupillary defect is present, both pupils will dilate when the affected eye is exposed to the light source. This defect should be differentiated from pharmacologic or traumatic mydriasis, which is more specifically poor pupillary constriction and is more evident in bright settings. In mydriasis the pupil of the nonaffected eye constricts briskly. Intraocular pressure measurements help differentiate glaucomatous etiologies from other causes of a red painful eye with decreased visual acuity. Elevated intraocular pressure measurements also may indicate the need for emergent procedures such as canthotomies. Devices that can measure intraocular pressure are adequate; even palpation on an anesthetized cornea can give useful diagnostic information. Fundoscopy should be performed, first noting the presence of a red reflex. Decreased intensity of the red reflex may indicate the presence of a cataract, vitreous hemorrhage, or a large retinal detachment. Slit-lamp examination should be performed when possible to assess for injury to the anterior chamber, cornea, iris, and lens. If a slit-lamp examination cannot be performed, examination with a penlight may be used to look for visible hyphema, obvious laceration, or a shrunken-appearing globe.
Imaging techniques

Plain films

Plain film radiographs of the orbits and sinuses are rarely used for diagnosis in orbital trauma. When performed, various views can provide information regarding the orbits and sinuses, such as the presence of an orbital wall or facial bone fracture or opacification of the sinuses. The conventional Caldwell’s and Waters’ views have moderate sensitivity in detecting orbital fractures: 73% to 78% for fractures of the orbital floor, 71% for fractures of the medial orbital wall, and 64% for fractures of the ethmoid-maxillary plate [3].

CT scan

The CT scan is considered the reference standard imaging modality in the diagnosis of mid-face fracture and orbital trauma. Orbital fractures are commonly missed in patients who have concomitant head trauma, making it imperative that one maintain a high suspicion for orbital injury [4]. The advent of thin-slice helical CT with coronal reconstructions has demonstrated improved image quality and reduced radiation to the lens [5]. The sensitivity of CT scan for orbital fractures ranges from 79% to 96%, with a lower sensitivity when evaluating the infraorbital rim [6]. Vegetable or organic foreign bodies, which increase the risk of endophthalmitis, may not be visualized on CT scan.

Ultrasound

Ocular ultrasound can be a useful tool for the emergency physician when evaluating trauma to the eye. Trauma frequently results in considerable soft tissue swelling, making it difficult to retract the lids and examine the eye fully. Ultrasound can evaluate noninvasively for lens dislocation, globe rupture, retrobulbar hemorrhage, intraocular foreign body, and retinal detachment (Fig. 1). Visualization of periorbital gas on ultrasound may prompt the physician to evaluate further for orbital fracture if not previously considered [7]. One study reported that ocular ultrasound performed by emergency physicians had a sensitivity of 100% and a specificity of 97.2% for identifying ocular pathology [8]. Care should be taken to place minimal pressure on the lid when performing the study, especially if globe rupture has not yet been ruled out. Ultrasound is contraindicated if there is high suspicion of rupture.

MRI

MRI is of limited usefulness in the acute stages of ocular trauma and should not be performed if there is any concern that a metallic intraocular foreign body is present. If there is concern regarding an organic foreign body, MRI may help further differentiate this foreign body from soft tissues when compared with a CT scan.
Blunt trauma to the orbit

Periorbital tissues

Contusion

Periorbital contusion and swelling may be the most prominent initial features in patients presenting to the hospital following trauma to the orbit. The appearance of the ecchymosis and swelling can be dramatic and make examination of the orbit challenging (Fig. 2). The emergency physician always must attempt to examine the structures underlying the swollen

Fig. 1. Ultrasound demonstrating intraocular foreign body. (Courtesy of Andreas Dewitz, MD, Boston, MA.)

Fig. 2. Contusion of the eye and adnexa. (From Boruchoff SA. Anterior segment disease: a diagnostic color atlas. Philadelphia: Elsevier; 2001. p. 204; with permission.)
eyelids. Examination of the underlying tissue may be aided with a Desmarres retractor, which will help avoid global pressure and damage to underlying structures. It is important to keep in mind that periorbital contusion may indicate related significant injury (eg, bilateral raccoon eyes may indicate basilar skull fracture). A study of 600 patients who had sustained significant head trauma found that 58.3% of patients who had isolated blepharohematoma on examination were found to have an orbital fracture on CT scan [9]. Treatment includes head elevation, cold compresses, and reassurance. Complete resolution typically takes 2 to 3 weeks.

*Orbital fractures*

Orbital wall fractures were first termed “blowout’ fractures” in 1957 by Converse and Smith. Orbital blowout fractures are those that occur within the bony orbit, usually along the medial walls and/or the floor, but the orbital rims are intact. There are three proposed theories regarding the mechanism of a blowout fracture. The first is the hydraulic theory, which postulates that when orbital pressure is increased, the globe decompresses through the weakest part of the orbit. The globe-to-wall contact theory was postulated in 1943 by Raymond Pfeiffer. This theory states that when the globe becomes displaced posteriorly in trauma, it strikes the wall, causing a fracture. The third theory, proposed by Le Fort and Lagrange in 1917, is that of buckling: the posterior movement of the orbital rim causes fracture along the medial wall or floor of the orbit [10].

Blowout fractures account for approximately 11% of fractures involving the orbit [11]. Following a blowout fracture, contents may herniate into the maxillary sinus (with orbital floor fractures), or into the ethmoid sinus (with medial wall fracture). A blowout fracture should be suspected when a patient presents with trauma to the globe and soft tissue swelling. Patients may complain of swelling following nose blowing, diplopia, or epistaxis. On examination one may find periorbital ecchymosis, subcutaneous emphysema, restricted extraocular movements, enophthalmos or exophthalmos, ptosis, or anesthesia in the distribution of the infraorbital nerve. The most common limitation in extraocular movements is restriction of upward gaze caused by the entrapment of the inferior rectus muscle. Muscle contusion or cranial nerve disruptions also may be the cause of abnormal extraocular motility, however (Fig. 3). Decreased sensation thought to be related to infraorbital nerve injury can be verified further by testing sensation on the ipsilateral upper gum. It is of utmost importance to remember to inspect the entire globe, because associated injury may occur in 10% to 25% of patients who have orbital floor fractures [11].

The diagnosis of orbital fractures is made most often using CT scan. Although rarely performed, plain-film radiographs of the orbits and sinuses may demonstrate the classic teardrop sign, which may signify orbital contents herniating into the maxillary sinus. In addition one can see air fluid levels or opacification of the sinuses that may indicate a blowout fracture.
CT scan of the orbits is extremely sensitive for identifying fractures. In addition ultrasound is a promising tool that can be used to identify orbital fractures. Periorbital gas seen on ultrasound may indicate an underlying orbital fracture [7].

Orbital fractures are not considered an ophthalmologic emergency unless there is visual impairment or globe injury. Surgical repair is indicated for patients who have persistent diplopia or cosmetic concerns (enophthalmos) and in general is not performed until swelling subsides 7 to 10 days following injury [12].

Patients should be discharged with instructions to use ice compresses and should be cautioned to avoid nose blowing and Valsalva maneuvers and to sneeze with their mouths open. The routine use of prophylactic antibiotics following orbital fracture is controversial and may not be indicated in all cases [13]. Orbital cellulitis is a rare complication following orbital fracture. Risk factors for developing orbital cellulitis include fracture adjacent to an infected sinus and nose blowing. Prophylactic antibiotics are recommended if adjacent sinusitis is present [14].

Patients should be instructed to return if they experience intense eye pain, changes in vision, proptosis, or a tense globe. These complaints should raise concern for compressive orbital emphysema or retrobulbar hemorrhage.

**Retrobulbar hemorrhage**

Traumatic hemorrhage into the retrobulbar space may result in acute visual loss [15]. Hemorrhage into the potential space surrounding the globe may occur following blunt trauma because of injury to the orbital vessels. It is important to remember that the orbit is an enclosed space bound laterally and posteriorly by bony walls, superiorly and inferiorly by the orbital...
septa, and anteriorly by the globe and inelastic orbital septum. In a small series of patients nondisplaced fractures of the orbital walls were found to be associated with retrobulbar hematoma [16]. This condition is rare following displaced fractures, however, because the blood will decompress into the sinuses [17]. Hemorrhage can lead to an acute increase in intraorbital pressure, which then is transmitted to the optic nerve and globe, resulting in central retinal artery occlusion and optic nerve ischemia. Clinical signs and symptoms include proptosis, limitation of extraocular movements, visual loss, afferent pupillary defect, and increased intraocular pressure. Diagnosis can be confirmed by CT scan, but treatment never should be delayed while waiting for imaging.

Early recognition and decompression is key to preserving vision and warrants emergent ophthalmologic consultation. Treatment of increased intraocular pressure can be attempted with topical beta-blockers or intravenous mannitol or carbonic anhydrase inhibitor. Lateral canthotomy can be a vision-saving procedure and is indicated in patients who have a history of trauma and marked periorbital edema with visual loss, severe proptosis, diffuse subconjunctival hemorrhage, or an afferent papillary defect. Lateral canthotomy is performed by applying a small, straight clamp to the lateral canthus, aiming inferiorly and laterally toward the conjunctival sac after local anesthesia administration (lidocaine 1% with epinephrine). The clamp is left in place for 15 seconds to 2 minutes. After removal of the clamp there is an impression in the tissue. A 1-cm incision is made along this impression using iris scissors. If pressures remain elevated, the lateral canthal tendon should be transected as well by aiming scissors inferolaterally. Transecting the inferior lateral canthal tendon should result in the lower lid pulling easily away from the lid margin (Fig. 4). Despite the decompression of high intraorbital pressure, usually only a small amount of blood is expressed with the release of the hematoma.

Fig. 4. Orbit following lateral canthotomy. Note the lower lid pulling easily away from lid margin. (Courtesy of John Lee, MD, Boston, MA.)
Anterior chamber

Traumatic hyphema

Traumatic hyphema is caused by disruption of blood vessels in the iris or ciliary body causing blood to extravasate into the anterior chamber (Fig. 5). Hyphemas are classified from grade 0 to 4, based on the percentage of the anterior chamber that is filled with blood. Microhyphemas are grade 0 and represent circulating red blood cells that can be detected only by slit-lamp examination. The grading system then progresses from grades 1 (less than one fourth to one third of the anterior chamber) through 4 (total anterior chamber filled with blood). An “eight-ball hyphema” refers to an anterior chamber that is entirely filled with a black-appearing clot. Hyphema may occur after blunt or penetrating trauma, and more than 50% have been documented as being sports related [1].

In traumatic hyphema, it is important to obtain a complete past medical history specifically addressing bleeding disorders such as hemophilias and Von Willebrand’s disease and hemoglobinopathies such as sickle cell anemia or sickle trait. Patients also should be questioned regarding the use of medications that may affect coagulation, including warfarin and aspirin. The symptoms of hyphema include pain, photophobia, and blurring of vision. Lethargy or somnolence can be associated with isolated traumatic hyphemas but always should raise concern for a concomitant head injury. On examination one frequently can see a hyphema on gross inspection with the patient sitting upright (causing the blood to layer in the aqueous fluid). In addition slit-lamp examination should be performed to assess for associated injury and further quantify the hyphema. Traumatic miosis or mydriasis may be present as well and should be differentiated from an afferent papillary defect. This distinction can be done by the swinging flashlight test, as described elsewhere in this issue. An afferent papillary defect will cause paradoxical dilatation in the affected eye and the unaffected eye, whereas traumatic mydriasis will result in constriction of both pupils (although limited in the affected eye). An afferent pupillary defect should raise concern for an optic nerve or posterior pole injury.

Fig. 5. Hyphema. (Courtesy of D. Wagner, MD, Washington, DC.)
The most common complication of hyphema is rebleeding, which occurs 2 to 5 days following injury when the initial clot retracts and loosens. Re-bleeding occurs in approximately 22% of patients [1]. Rebleeding is more common in those who have visual acuities of 20/200 on presentation, initial hyphema of more than one third of the anterior chamber, more than 1 day’s delay in obtaining medical attention, and elevated intraocular pressure at initial presentation [1,18]. Other complications include corneal blood staining (in approximately 5% of patients), elevated intraocular pressure, and synechiae formation between the iris and the cornea or the lens posteriorly. Elevated intraocular pressure facilitates corneal blood staining. As the pressure in the anterior chamber increases, the ability of the corneal endothelial cells to transport fluid out of the stroma is impaired. The cornea swells, and the endothelium becomes leaky, forming gaps large enough for the hemoglobin to enter the corneal stroma. Patients who have sickle cell anemia or who are trait positive have a higher rate of complications associated with hyphema. The acidic and hypoxic nature of the anterior chamber leads to sickling of the red blood cells, which can cause increased intraocular pressure and decreased aqueous humor output. Patients who have a history of sickle cell trait have rates of rebleeding up to 64% [1].

Treatment for hyphema should be directed at minimizing secondary hemorrhage and reducing the incidence of secondary glaucoma. Recent literature indicates that hospitalization versus outpatient management, moderate ambulation versus strict bed rest, or treatment with unilateral or bilateral eye patches has no significant effect on the incidence of secondary hemorrhage or final visual outcome [1]. In addition, outpatient treatment has been shown to be cost effective [19]. Hospitalization has been recommended for patients who have rebleeding, elevated intraocular pressure, positive sickle cell trait or anemia, hyphemas greater than 50%, or decreased vision, for noncompliant patients, and in suspected child abuse. The disposition of these patients always should be discussed with an ophthalmologist. Patients who are discharged from the hospital must undergo daily examinations by an ophthalmologist to assess for increased size of hyphema or other complications.

Supportive therapy includes elevation of the head to 30° while at rest. In addition, eye patching with a metal shield provides protection from further ocular injury and often is recommended until the hyphema resolves. The shield must have holes or be made of clear plastic so that patients can monitor their vision, because decreased vision is the earliest symptom of rebleeding. Topical application of cycloplegics has not been shown to have significant therapeutic effects but can increase patient comfort and facilitate the examination of the posterior segment. Topical corticosteroids have been shown to reduce intraocular inflammation and decrease the incidence of secondary hemorrhage [20]. Topical and systemic antifibrinolytics, such as aminocaproic acid (ACA) and tranexamic acid (not available in United States), have been shown to decrease secondary hemorrhage significantly [18]. Aminocaproic acid is an antifibrinolytic agent that prevents the conversion of
plasminogen to plasmin and therefore delays clot dissolution and theoretically decreases the risk of rebleeding. Studies have shown that topical ACA is as effective as systemic ACA and avoids the side effects typically associated with administration such as nausea, vomiting, and hypotension [1]. These agents should be avoided in pregnant patients, patients who have renal or hepatic dysfunction, or those at risk for thromboembolic disease. Intracameral tissue plasminogen activator currently is under investigation and is reserved for large clots of prolonged duration or malignant elevation of intraocular pressure. In addition, elevated intraocular pressure (> 24 mm Hg) can be treated with topical beta-blockers and carbonic anhydrase inhibitors such as acetazolamide. Acetazolamide lowers pH, causing increased sickling, and should be avoided in pediatric patients or those who have sickle cell trait or anemia. Methazolamide is the preferred agent in this situation [21]. Mannitol may be administered for severely elevated intraocular pressure (> 35 mm Hg). Nonsteroidal anti-inflammatory medications and aspirin should be avoided because of the increased risk of rebleeding. Surgery usually is reserved for delayed complications such as corneal staining and persistently elevated intraocular pressures but often is performed early in patients who have sickle cell anemia or trait.

Subconjunctival hemorrhage

Subconjunctival hemorrhage is caused by the rupture of small subconjunctival blood vessels. Although patients may report a prior sneezing or coughing episode or a Valsalva maneuver, some patients may have no recollection of the preceding events. Patients often seek care because of the dramatic appearance. On examination a painless, smooth, bright-red area is noted over the bulbar conjunctiva and is sharply demarcated at the limbus. Visual acuity should be normal. If there is preceding blunt or penetrating trauma, underlying scleral penetration should be considered. Pain on extraocular movements and bloody chemosis also should prompt suspicion for injury to the globe. Subconjunctival hemorrhage should always be distinguished from chemosis: a hemorrhage is flat and smooth, whereas chemosis presents as erythema with edema, and the area is raised. Bilateral or recurrent subconjunctival hemorrhage may require a work-up for bleeding diathesis. Treatment of subconjunctival hemorrhage consists of reassurance and local cold compresses for 24 hours. Subconjunctival hemorrhages heal spontaneously in 2 to 4 weeks.

Injury to the iris and ciliary body

Traumatic iridocyclitis (uveitis)

Blunt injury to the globe may contuse and inflame the iris and ciliary body, resulting in ciliary spasm. Patients may complain of photophobia, blurred vision, and eye pain. Examination reveals conjunctival injection, specifically ciliary flush, cells, and flare in the anterior chamber and a small,
poorly dilating pupil. Cells and flare are best seen in the slit lamp with high magnification and very bright light. This condition is self limited; symptomatic treatment consists of paralyzing the iris and ciliary body with long-acting cycloplegic agents such as homatropine 5% for a period of 7 to 10 days. If there is no improvement after 5 to 7 days, prednisolone acetate 1% may be used to decrease inflammation in consultation with an ophthalmologist. Topical steroids should be withheld in patients who have a corneal epithelial defect. Resolution typically occurs within 1 week.

_Traumatic mydriasis and miosis_

Blunt injury to the orbit may damage the iris sphincter. Bruising and irritation of the sphincter leads to constriction of the pupil (miosis). Small tears to the sphincter muscle may result in dilatation or mydriasis. In the setting of significant head trauma and altered mental status, one must rule out a cranial nerve palsy and brain herniation. Treatment is supportive, and the condition often resolves spontaneously.

_Iridodialysis_

Traumatic iridodialysis is a tearing of the iris root from the ciliary body leading to formation of a “secondary pupil” (Fig. 6). This injury may also cause a hyphema. Large tears can be a cause of monocular diplopia. Urgent ophthalmologic consultation is warranted when iridodialysis causes a hyphema or decreased visual acuity. Surgical repair may be indicated for persistent monocular diplopia.

_Acute glaucoma_

Acute glaucoma can occur following trauma. The underlying pathophysiology is narrowing of the anterior chamber or disruption of outflow of
aqueous humor, either secondary to red blood cells (hyphema), formation of a trabecular meshwork scar, or lens dislocation. Posttraumatic glaucoma is associated with advanced patient age, lens injury, poor baseline visual acuity, and inflammation of the anterior chamber [22]. Patients who have these characteristics should be followed closely by an ophthalmologist to assess for development of glaucoma. Acute rises in intraocular pressure should be treated aggressively with miotics (contraindicated in eyes following cataract surgery or lens extraction), topical drops including beta-blockers, alpha agonists, prostaglandin analogues, acetazolamide, or mannitol.

**Injury to the lens**

**Subluxation and dislocation**

Blunt trauma to the eye results in a sudden compressive deformation of the globe, displacing the cornea and anterior sclera posteriorly with a compensatory expansion of the globe in the equatorial direction. This trauma can result in damage to the lens zonule fibers, which are responsible for holding the lens in place, causing lens dislocation or subluxation. Following complete disruption of the lens zonules, the lens may dislocate posteriorly or, less commonly, into the anterior chamber (Fig. 7). Diagnosis of this injury is based on history and physical examination. Symptoms include blurring of the vision or monocular diplopia and distortion when the lens remains partially in the visual axis. The lens may be visualized when displaced into the anterior chamber or may be viewed after pupillary dilatation when dislocated posteriorly. Iridodonesis, a tremor of the iris after rapid eye movements, may be a helpful finding associated with posterior dislocations [23]. An anteriorly dislocated lens may cause acute angle closure glaucoma, which may be a vision-threatening complication. Predisposing factors for lens dislocation include Marfan’s syndrome, homocystinuria, and sphero- phakia [23]. Lens subluxations and dislocations should be referred to an

![Fig. 7. Traumatic lens dislocation. (From Harlan JB, Pieramici DJ. Evaluation of patients with ocular trauma. Ophthalmol Clin North Am 2002;15:159; with permission.)](image-url)
ophthalmologist for surgical repair. Repair should be performed on an emergent basis if the lens is obstructing the flow of aqueous humor, leading to elevated intraocular pressure.

**Cataract formation**

The stroma of the lens normally are sequestered in a relatively dehydrated environment by the lens capsule. If the lens capsule is disrupted by either blunt or penetrating trauma, the stroma may absorb fluid, swell, and become cloudy (Fig. 8), obstructing the outflow of aqueous humor and leading to acute glaucoma. Traumatic cataracts may occur acutely or develop over weeks to months. Bilateral cataracts may develop after lightning strike or electrical injury [24]. There is no effective prevention, and definitive treatment requires lens replacement.

**Globe injury**

**Globe rupture**

Globe rupture always should be considered when evaluating a patient who has sustained blunt trauma or a penetrating injury, because it is a major cause of monocular blindness. More than 90% of these injuries are preventable, with trauma from violent behavior accounting for a large portion [25]. Ruptures are most common at the insertions of the intraocular muscles or at the limbus, where the sclera is thinnest. Diagnosis of a ruptured globe can be obvious if intraocular contents are visualized, but occult rupture can be difficult to diagnose. Critical signs and symptoms include decreased visual acuity, severe bullous subconjunctival hemorrhage (involving 360° of the bulbar conjunctiva), a deep or shallow anterior chamber, and limitation of extraocular motility. Other signs include low intraocular pressure (although pressure may be normal or increased), an irregularly shaped pupil (peaked toward the wound), iridodialysis, exposed uveal tissue (which appears brownish-red), or vitreous hemorrhage (Fig. 9).

![Fig. 8. Traumatic cataract and iridodialysis. (From Harlan JB, Pieramici DJ. Evaluation of patients with ocular trauma. Ophthalmol Clin North Am 2002;15:159; with permission.)](image)
Once the diagnosis of globe rupture is made or suspected, further manipulation of the globe should be deferred. Treatment includes emergent ophthalmologic consultation, placement of a protective eye shield, antiemetics (to avoid further increasing intraocular pressure during a Valsalva maneuver), analgesics, and systemic antibiotics to prevent endophthalmitis. Any maneuver that may increase intraocular pressure should be avoided. A CT scan should be obtained to evaluate for the presence of orbital and intraocular foreign bodies. CT scan evaluation alone is only 75% sensitive for diagnosis of globe rupture and therefore should be used as a complement to physical findings [26]. Ultrasonography and indirect ophthalmoscopy may be helpful in further evaluation but should be deferred to the ophthalmologist. The administration of succinylcholine for airway management is controversial. There have been anecdotal reports of expulsion of intraocular material with succinylcholine use, but more recent case studies do not support this finding [27]. The current recommendation for patients who require rapid airway management with concomitant penetrating ocular injury is rapid-sequence intubation with succinylcholine following pretreatment with a nondepolarizing and sedating agent.

Globe luxation

Luxation of the globe is a rare condition that results from an extreme form of trauma. Many years ago it was thought to be caused by gouging during fights and historically has been a complication of forceps delivery in obstructed labor [28]. Now it is seen more commonly after great forces are applied to the head in an anteroposterior direction (eg, in a motor vehicle crash), but there have been case reports documented in which luxation was caused by penetrating trauma. The most commonly injured and avulsed extraocular muscle is the medial rectus. The status of the optic nerve is clinically important. Injury to the optic nerve may occur from the forward propulsion of the globe with traction on the nerve or may be related to a sudden rise in intraocular pressure.

Fig. 9. Ruptured globe. (From Kanski JJ. Clinical diagnosis in ophthalmology. Philadelphia: Mosby Elsevier; 2006.)
Acute care consists mainly of protecting the eye from further damage. This protection can be accomplished with a cup or other hard protective device. In addition, early reduction with topical anesthesia can be attempted, because it will minimize the amount of traction placed on the optic nerve. Globe reduction may not be possible in the emergency department because of the swelling of the eyelids and surrounding tissues, necessitating intraoperative reduction under general anesthesia [28]. Spontaneous and voluntary globe luxation has been reported during the insertion of contact lenses and may be more common in diseases that cause proptosis such as Grave’s disease and orbital tumors. Calming the patient and having the patient relax the eyelids will make it easier for spontaneous repositioning to occur.

Posterior segment

Vitreous hemorrhage

Vitreous hemorrhage occurs when blood enters the normally avascular vitreous space, which is filled with a clear gelatinous material. This space is bordered anteriorly by the lens, posteriorly and laterally by the retina, and laterally by the ciliary body. Although most causes of vitreous hemorrhage are nontraumatic (diabetic retinopathy, sickle cell disease, posterior vitreous detachment, retinal vein occlusion, leukemia), trauma accounts for 12% to 31% (depending on study population) and is the most common cause of vitreous hemorrhage in younger patients [29,30]. Vitreous hemorrhages can be associated with retinal tears, avulsed retinal veins, or subarachnoid hemorrhage. Vitreous hemorrhages in an infant should prompt consideration of shaken baby syndrome, admission, and an in-depth search for other traumatic injuries. Vitreous hemorrhages may be associated with traumatic and atraumatic subarachnoid hemorrhage (Terson’s syndrome). The theory is that increasing intracranial pressure results in increased retinal vein pressures, causing rupture and vitreous hemorrhage [31]. Patients who have Terson’s syndrome have a poorer prognosis than patients who have subarachnoid hemorrhage without vitreous hemorrhage.

Symptoms of vitreous hemorrhage include floaters (small hemorrhage), cobwebs, shadows, a smoky haze, or loss of vision (larger hemorrhage). Physical examination may reveal a decreased or absent light reflex, loss of fundus detail, or floating debris but may be normal in the undilated eye. An indirect fundoscopic examination should be performed if the direct examination is normal. In the preretinal hemorrhages of shaken baby syndrome, layering of blood with a meniscus may be seen on the retina.

Traumatic vitreous hemorrhage mandates emergent ophthalmologic consultation, because 11% to 44% of vitreous hemorrhages are associated with retinal tears [32]. Ultrasound may be helpful if the fundus is obscured. Discharge instructions include limited physical activity and head elevation when sleeping. Nonsteroidal anti-inflammatory drugs and aspirin should be
avoided, unless the benefit outweighs the risk (i.e., in patients who have unstable angina or other clinical indications). Definitive treatment depends on the underlying cause and may include laser, cryotherapy, or scleral buckling in cases of retinal detachment. Most hemorrhages resolve spontaneously within a few weeks to months.

**Chorioretinal injury**

Trauma is the most common cause of retinal detachment in children and is responsible for about 10% of detachments in the general population [33]. Symptoms of retinal tears or detachments include floaters from bleeding and flashing lights from stimulation of retina neurons. Isolated tears and detachments do not cause pain. Visual field defects or decreased visual acuity may be present as well. Retinal detachment generally progresses slowly following injury, occurring weeks to months later. Fundoscopic examination may reveal a hazy, gray membrane of the retina billowing forward (Fig. 10), but small peripheral tears may not be visualized on direct ophthalmoscopy. Indirect ophthalmoscopy should be performed on every patient in whom the diagnosis of retinal detachment or tear is being considered. Approximately one third of retinal detachments are not diagnosed until at least 6 weeks after trauma [34].

Management of retinal tears or detachment begins with urgent ophthalmologic consultation. Treatment consists of either head elevation or supine positioning, depending on whether the tear is superior or inferior. Surgical correction consists of scleral buckling, pars plana vitrectomy, or photocoagulation. The timing of surgical correction is based on the size of the retinal detachment and the degree of macular involvement.

![Fig. 10. Retinal detachment. (From Kanski JJ. Clinical diagnosis in ophthalmology. Philadelphia: Mosby Elsevier; 2006.)](image)
Commotio retina

Commotio retina, also known as “Berlin’s edema,” may occur after recent blunt ocular trauma. Studies have demonstrated this injury to be present in 9% to 14% of blowout fractures [11]. The term “commotio retina” literally means retinal contusion in Latin and is caused by disruption of the retinal photoreceptors. The term “Berlin’s edema” is actually a misnomer, because no edema is present (Fig. 11). Patients may complain of decreased visual acuity or may be asymptomatic if the macula is not involved. Examination reveals a confluent area of retinal whitening. Blood vessels can be seen distinctly below the whitened area. Commotio retina can occur anywhere on the retina, but it usually is maximal in the area opposite the traumatic blow. There is no specific treatment for this condition, because it usually resolves spontaneously in approximately 2 weeks. Serial examination is necessary to ensure that retinal tear or detachment has not occurred.

Penetrating ocular injury

Periorbital tissues

Conjunctival lacerations

Lacerations of the bulbar conjunctiva are commonly associated with intraocular foreign bodies or underlying sclera perforation, so a ruptured globe must be ruled out. Conjunctival lacerations may be seen as a conjunctival defect, exposure of Tenon’s capsule, or orbital fat. Slit-lamp examination can help differentiate superficial from deep lacerations. Small, superficial lacerations (< 1 cm) require no suturing and generally heal rapidly. Lacerations that are greater than 1 cm may be repaired by an

Fig. 11. Commotio retina is visualized as a patchy, gray-white opacification of the retina. (Courtesy of D. Wagner, MD, Washington, DC.)
ophthalmologist using 6-0 to 8-0 absorbable suture. Treatment with prophylactic antibiotic ointment or drops is recommended for all patients who have conjunctival lacerations [35].

**Laceration of the eyelid**

Lacerations of the eyelid should prompt concern for a penetrating globe injury or foreign body. Emergency physicians can manage simple horizontal or oblique partial-thickness lid lacerations. Closure should be performed with interrupted 6-0 or 7-0 nylon sutures. Sutures should be removed in 3 to 5 days. Lacerations that require immediate referral to an ophthalmologist for repair include those that involve the lid margins, the canalicular system, or the levator or canthal tendons, loss of tissue, or lacerations through the orbital septum. A laceration through the orbital septum should be suspected if orbital fat protrudes through the wound [35].

**Globe injury**

**Corneoscleral laceration and puncture wounds**

Signs of corneal perforation include loss of anterior chamber depth, blood in the anterior chamber, and a teardrop-shaped pupil caused by iris prolapse through the corneal laceration. Small corneal lacerations may be difficult to diagnose. If corneal laceration is suspected, one must inspect the entire cornea while taking care to not put excessive pressure on the globe. Corneal lacerations occur most often on the inferior aspect of the globe because of Bell’s phenomenon, the reflex upward rotation of the globe during blinking in response to potential foreign body penetration. Suspicion that aqueous humor is leaking from the wound can be confirmed by Seidel’s test, which is performed by applying fluorescein dye over the area of concern and noting a stream of yellow dye on slit-lamp examination (Fig. 12).

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Fig. 12. Corneal laceration following repair. *(Courtesy of John Lee, MD, Boston, MA.)*
Once a laceration is suspected, a protective cover should be placed on the eye, and prophylactic antibiotics should be administered. Ophthalmologic consultation should be obtained when there is concern for a full-thickness laceration. Partial-thickness lacerations that are not widened can be treated with cycloplegics, topical antibiotics, and a pressure patch. Lacerations that require repair are performed in the operating room.

*Intraocular foreign body*

An intraocular foreign body is present in 18% to 41% of open globe injuries. The diagnosis should be suspected based on history [36]. Hammering (especially of metal on metal) is the most common mechanism, responsible for 60% to 80% of cases (Figs. 13 and 14). In developed countries this type of injury now occurs more commonly in the home than in the workplace. It is crucial to identify the material of the foreign body because it will influence treatment decisions. Specifically, iron-containing foreign bodies can cause siderosis, brownish discoloration of the iris, and yellow cataracts, and copper may lead to cheirosis, a rapidly developing sterile endophthalmitis. Lead-containing products may cause lead poisoning if left in place.

An intraocular foreign body should be considered strongly in penetrating globe injuries. Visual acuity generally is decreased, but normal visual acuity does not rule out the presence of a foreign body. Additional warning signs on examination consist of localized corneal edema, hemorrhage over the sclera, a nonsurgical hole in the iris, or a cloudy lens. CT has largely replaced plain-film radiography in the diagnosis of an intraocular foreign body.  

![Fig. 13. Intraocular foreign body located near the retina. (Courtesy of D. Wagner, MD, Washington, DC.)](image-url)
body and has a sensitivity ranging from 65% (for a foreign body < 0.06 mm³) to 100% (for foreign bodies > 0.06 mm³) [36]. Other modalities for diagnosis include ultrasound (relatively contraindicated in suspected globe penetration), in which sensitivity varies greatly based on clinician experience [36], and MRI, which is limited because of concern for secondary intraocular damage if the foreign body is metallic. Treatment consists of intravenous antibiotics to prevent endophthalmitis. Vitrectomy should be performed without delay on patients who have signs of endophthalmitis and for foreign bodies that consist of vegetable matter or copper or those that occur in the rural setting and may be contaminated with soil. In a series of patients from the military population, rates of endophthalmitis did not increase if foreign body removal was delayed [37]. Preventive measures include encouraging proper eye protection for all patients.

Orbital foreign body

Patient history often is the first clue to the diagnosis of an orbital foreign body and also can indicate the most likely material (Fig. 15). As with intraocular foreign bodies, it is important to discern if the foreign body is metallic or organic in nature. The location of the foreign body can be confirmed with an orbital CT scan (best for metallic foreign bodies) or by MRI and orbital ultrasound, which may help better locate organic material. Foreign bodies that can be left in the orbit include BBs, metallic shrapnel fragments, and bits of metal that are not copper. Small metallic fragments generally are well tolerated in the orbit, and leaving them in place may cause less harm than a deep exploration. Organic foreign bodies and objects that are protruding through the skin or penetrating the brain or the sinuses should be removed in the operating room. Inorganic foreign bodies that require special consideration and possible removal include objects containing copper, lead, and iron [38]. Incomplete extraction has been documented when attempted in the emergency department. It is important to assure complete

Fig. 14. Large plastic foreign body from fireworks injury. (From Mester V, Kuhn F. Intraocular foreign bodies. Ophthalmol Clin North Am 2002:15;239; with permission.)
removal, especially of organic material, because it is associated with a high risk of infection [39]. It is common for metallic fragments to migrate with time in the orbit. Patients who present with orbital pain after injury by shrapnel injury or another foreign body may require removal even years after the injury.

**Delayed complications**

*Endophthalmitis*

Posttraumatic endophthalmitis, or inflammation of the deep structures of the eye, is a devastating complication of open globe injury, occurring in 0% to 13% of patients [40]. Infection may occur in hours to months following ocular trauma. Symptoms consist of pain, photophobia, and visual loss and often are hard to distinguish from other posttraumatic infections. Pain or visual loss out of proportion to the clinical condition should prompt consideration of endophthalmitis. Examination may also reveal nonspecific signs including hypopyon, conjunctival erythema and edema, and lid swelling. The pathogens causing endophthalmitis, in order of prevalence, include *Staphylococcus epidermidis*, Bacillus spp, and Streptococcus (which is particularly prevalent in the pediatric population) [41]. Bacillus causes up to 46% of endophthalmitis cases seen in a rural population because of contaminated soil or farming equipment [41]. Systemic and topical antibiotics against the common causative pathogens are the standard of care for prevention of posttraumatic endophthalmitis, although there have been no randomized, clinical trials. Prophylactic intravitreal antibiotics should be administered only by a trained ophthalmologist. Risk factors for developing posttraumatic endophthalmitis were thought to include delayed primary repair, delay in initiating therapy, breach in the lens capsule, and retained intraocular foreign body [40]. Experience with penetrating eye injury during Operation Iraqi Freedom, however, found that delayed removal of intraocular foreign

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Fig. 15. External photograph demonstrating obvious ocular/orbital foreign body. (From Harlan JB, Pieramici DJ. Evaluation of patients with ocular trauma. Ophthal Clin North Am 2002:15;158; with permission.)
bodies did not increase the rate of endophthalmitis if systemic and topical antibiotics were administered [42]. Definitive treatment consists of vitrectomy in addition to antibiotics. Topical or systemic corticosteroid therapy is controversial. Early recognition and prompt treatment of Bacillus infection increases the chances of improved visual outcome.

**Sympathetic ophthalmia**

Sympathetic ophthalmia is an inflammation that occurs in the uninjured (sympathizing) eye weeks to decades following the initial insult to the injured eye. It also may occur following uncomplicated intraocular surgery. It is thought to be caused by an autoimmune response to the release of the uveal contents into the vitreous humor of the injured eye. This release of tissue leads to autoimmune destruction in the uninjured eye, potentially causing severe bilateral vision loss. Sympathetic ophthalmia is rare, occurring in 0.2% to 1% of patients following trauma and in 0.001% of patients following surgical procedures. Symptoms are similar to a nongranulomatous uveitis with blurry vision, photophobia, decreased visual acuity, and tearing. Examination may reveal keratic precipitates, papillitis, and subretinal exudates. These exudates may coalesce at the posterior pole, producing an area of exudative retinal detachment and, over time, scarring of the posterior pole. Treatment consists of high-dose oral corticosteroids. If the inflammation cannot be controlled, other immunosuppressive agents, including cyclosporine, chlorambucil, or azathioprine, may be used. Sympathetic ophthalmia can be prevented if the hopelessly injured eye with no visual potential is enucleated within 14 days of trauma. The role of enucleation following development of sympathetic ophthalmia is controversial and should be based on the visual acuity of the injured eye. It remains unclear if enucleation of the injured eye improves prognosis of the sympathizing eye. If the eye retains some visual function, it probably should not be removed.

**Burns**

**Acid and alkali exposure**

Chemical burns are worrisome because of their ability to affect multiple ocular structures profoundly and potentially cause blindness. Alkali-containing agents, such as oven and drain cleaners (potassium hydroxide), lime in plaster (calcium hydroxide), fertilizers and sparklers (ammonium hydroxide), and high-concentration bleach (sodium hypochlorite), are particularly damaging because they are both lipophilic and hydrophilic and can penetrate cell membranes rapidly (Fig. 16). Deployment of airbags causes release of sodium hydroxide, which can lead to an alkaline chemical keratitis [34]. Ocular damage results from saponification of cell membranes and cell death along with destruction of the extracellular matrix. Acidic agents, such as hydrofluoric acid (used in industrial etching and alkylation of high-octane gasoline), battery acid (sulfuric acid), and hydrochloric acid, generally cause
less damage than alkaline agents because many corneal proteins bind acid and act as a buffer. The coagulated tissue functions as a barrier and prevents further penetration of the acid.

The Roper-Hall classification of chemical burns can be used; for simplicity, however, burns can be classified as mild-to-moderate burns and severe burns. The most important differentiating feature is limbal and corneal ischemia. On examination of mild-to-moderate burns one can see focal areas of conjunctival chemosis and hyperemia as well as mild eyelid edema and anterior chamber reaction. First- and second-degree burns may be noted on the periorcular skin. Severe burns are characterized by conjunctival blanching and pallor [43]. Corneal edema or opacification can make it difficult to evaluate the anterior chamber. Early sequelae of chemical burns occurring 1 to 3 days after exposure include elevated intraocular pressure caused by damage of the trabecular meshwork and corneal edema. Long-term complications include perforation, scarring, and neovascularization of the cornea, adhesions of the lids to the globe (symblepharon), glaucoma, cataract, and retinal damage.

Treatment should be initiated immediately after exposure by irrigating the eyes with copious amounts of fluid, preferably saline or lactated Ringer’s solution, for at least 30 minutes. A Morgan lens may be used if there is no particulate material. (See the article entitled “Eye Exposures” in this issue for a discussion of eye irrigation techniques, including the Morgan lens). Five to 10 minutes after completing irrigation, litmus paper should be placed in the inferior cul de sac. Irrigation is continued until a neutral pH, 7.0 to 7.4, is reached. pH is measured before adding topical anesthetic to the eye, because most anesthetics have an acidic pH and may lower the measured pH artificially. Foreign body removal, by sweeping the fornices with a cotton swab, should be performed as well, with special attention to the conjunctival fornices, because material that is crystallized may cause a persistently elevated pH. Additional treatment consists of cycloplegia.
(using scopolamine 0.25%), topical antibiotic ointment, and topical steroids if significant inflammation of the cornea or anterior chamber is present. Phenylephrine should be avoided because it causes vasoconstriction. If elevated intraocular pressure is noted, anti-glaucoma medications such as acetazolamide should be initiated. Oral vitamin C (ascorbic acid) stimulates collagen production and therefore has a theoretical benefit [43]. These patients require close follow-up, and some may require hospital admission for further irrigation or monitoring.

Miscellaneous irritants, solvents, and detergents

Unknown exposures should be treated initially as though they were an acid or alkali exposure. The eye should be irrigated immediately. Detergents generally cause only a mild conjunctival irritation, but more irritating substances can cause denudation of the cornea and inflammation of the anterior chamber. After the eye is thoroughly irrigated, it should be treated like a corneal abrasion with topical erythromycin ointment. Exposure to aerosol products may lead to intraocular foreign body from the propellant. Compounds that are found in personal protective devices such as mace and pepper spray are treated in the same fashion as other chemical injuries.

Special consideration should be given to cyanoacrylate adhesive (‘‘super-glue’’) exposure. These glues are rapid setting and harden quickly on contact with moisture. If the eyelids are glued together, an attempt should be made to separate them with gentle traction. Lids that are sealed shut in normal anatomic position that cannot be separated with gentle traction can be left alone, allowing time for the super glue to dissolve over the next few days. Misdirected lashes and hardened glue may cause corneal defects and irritation. These defects should be treated like corneal abrasions. Attempts to dissolve the adhesive with other agents should be avoided. Ophthalmologic consultation should be obtained for these exposures [43].

Thermal burns

Thermal burns are more frequently seen on the eyelids than on the globe because of reflex blinking and Bell’s phenomenon. Superficial burns may be treated with irrigation and topical antibiotic ophthalmic ointment. Second- or third-degree burns of the eyelid warrant ophthalmologic consultation. Although patients frequently cover the face, hot liquid splashes and cigarette ashes may cause a superficial corneal epithelial injury and are treated like a corneal abrasion. There have also been documented cases of air bag–related thermal burns caused by failure of the inflation mechanism [34].

UV keratitis

Exposure to UV light can occur from tanning booth exposure, from high-altitude environments, or from welding. The UV light causes direct corneal epithelial damage. Patients give a history of exposure approximately 6 to 10 hours before developing symptoms. Symptoms include moderate-to-severe
ocular pain, foreign body sensation, red eye, tearing, photophobia, and blurred vision. Examination reveals decreased visual acuity and diffuse punctate lesions on fluorescein staining, with a sharp demarcation at the lower lid where the eye was protected. Treatment consists of cycloplegic drops, topical antibiotic ointment, and oral analgesics.

Prevention

Wearing protective eyewear often can prevent injuries to the globe. A study of patients in the US military who had sustained eye injury found that only a small percentage of them were wearing protective glasses at the time of injury [37,42]. In addition, many sports-related eye injuries could be prevented by wearing the recommended protective equipment. Air bag deployment, although associated with a 20% reduction in the incidence of fatal and severe injuries after frontal and near-frontal automobile collisions, increases the risk of orbital fractures or other ocular trauma [44].

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References