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Eye trauma is a common chief complaint among emergency department (ED) pediatric patients. Although many eye injuries can be prevented by appropriate supervision of children's activities and the appropriate use of protective eyewear, eye injuries remain an important cause of visual loss in children. Specific attention to a thorough physical examination is mandatory in the evaluation of pediatric eye trauma, and may include visual acuity, pupillary reactions, external examination, ocular motility, visual field testing, slit-lamp examination, fluorescein staining, funduscopic examination, and intraocular pressure (IOP) when indicated. Since a pediatric eye examination might be challenging, certain tricks of the trade are presented to aid in the physical examination of the patient with a potential eye injury.

Following complete ocular evaluation, many eye injuries can be managed on an outpatient basis with ophthalmologic follow-up and appropriate medical management. Certain eye injuries warrant emergent ophthalmologic consultation, including high-grade hyphema, penetrating globe injury, suspected or known open globe injury, retrobulbar hematoma, or any eye injury resulting in significant vision loss. Prompt evaluation, treatment, and referral are indicated to reduce incidence of long-term complications, which may include deformity, impaired muscle function, and vision loss.

—The Editor

Keeping an 'Eye' on the Pediatric Patient: An Update on Eye Trauma

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Introduction

It is estimated that 2.4 million eye injuries occur annually in the United States, accounting for 0.2% of ED visits; approximately one third of eye injuries occur in pediatrics.[1,2] Ocular trauma is one of the most significant causes of vision loss in pediatric patients. Many eye injuries can be prevented by appropriate supervision of children's activities and the appropriate use

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of protective eyewear during sports. The majority of injuries occur in the home, followed by public places such as schools or athletic facilities.[3] Injuries often occur related to consumer products, such as sports equipment, household cleaning chemicals, toys, and furniture.[4] Eye injuries are the most common type of injury from fireworks in the pediatric population.[5] The most common diagnoses include corneal abrasion, blunt trauma, and corneal foreign body.[6,7]

Eye injuries are often isolated injuries, many of which can be managed on an outpatient basis; 2%–3% of patients require inpatient hospital admission.[8] Importantly, many eye injuries are also associated with major trauma, particularly in patients with facial fractures, facial contusions, or basilar skull fractures.[9,10]

Examination of the Injured Eye in the Pediatric Patient

Examining the eye of an injured child may pose a significant challenge. Most patients older than 3 years of age can be successfully examined using interactive distracting techniques and parental assistance; rarely, a papoose board or sedation may be required for an adequate examination. Topical anesthesia may aid in comfort during the eye examination. After collecting a comprehensive history, the physical examination of the eye should include these elements: visual acuity, pupillary reactions, external examination, ocular motility, visual field testing, slit-lamp examination, fluorescein staining, IOP, and funduscopic examination.[11] However, certain aspects of a thorough eye examination are age-specific, other areas are independent of age, and most depend on patient cooperation. Remember to perform the more uncomfortable portions of the exam last.

Visual acuity is the vital sign of the eye, and its measurement is essential in the evaluation of eye trauma. The only acceptable delay is in the case of chemical exposure to the eye, which requires immediate irrigation. Visual acuity in the neonate, infant, and toddler is performed by assessing pupillary reaction to light. A light source should be used 1–3 feet away; the ability to track and fixate on light determines adequate visual acuity. Steady fixation is considered roughly equivalent to 20/40, unsteady fixation is equivalent to 20/100, and inability to fixate is equivalent to 20/400. By the age of 2–3 years, formal visual acuity testing is performed using a Snellen chart, Allen chart (pictures), or rotating "E" chart at 20 feet. Testing should always be done with correction (if possible) or with a pinhole device if the patient is myopic. A pre-made pinhole device will correct most refractive errors to at least 20/30. If a pinhole device is not available, a quick alternative can be made using an 18-gauge needle and an index card; a metal eye shield with multiple small holes can also be used. If a refractive error exists beyond 20/30, it should not be attributed to simple refractive error. When individuals are unable to read the first line (20/200), shorten the distance to 10 feet and have them read the top line (10/200). Continued inability to read any letters will require the use of counting fingers and recording the distance (e.g., able to count three fingers at a distance of three feet). If the patient is unable to count fingers, then attempt to see if the patient can visualize gross hand motion or perceive light.[12,13]

Pupil examination should include size, shape, symmetry, and reaction to light. A unilateral dilated pupil raises concern for compression of the third nerve, which may be a sign of uncal herniation, especially when associated with traumatic head injuries. If this finding is present, the physician should perform a more detailed neurological assessment, as there are other causes of unequal pupils, such as anisocoria or direct pupillary injury. A teardrop pupil should alert the emergency physician that ocular perforation has occurred. After a penetrating injury to the eye, the pupillary margin and portions of the iris are drawn into the penetrating wound, causing a teardrop appearance with the apex pointing to the injury. During the pupillary exam, if you suspect a penetrating injury to the eye, avoid any pressure to the eye. Examination of the eye should include evaluation for an afferent pupillary defect. Normal pupils will constrict and accommodate equally when a bright light is shown on the pupil (direct) and light shown on the opposite pupil (consensual light reflex). If the light reflex is diminished, a swinging flashlight test will discriminate between an afferent (retina, optic nerve) injury or efferent (third nerve or papillary muscle) injury. With an injury to the afferent tract, the pupil constricts consensually but not directly, while an efferent injury prevents both direct and consensual

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pupillary reflex; the unaffected eye maintains both responses.[13]

External examination of the eye includes observing for proptosis (suggestive of retrobulbar hematoma) and enophthalmos (suggestive of ocular rupture or blow-out fracture). The eyelids should be examined for lacerations, contusions, and punctures. The inner aspects of the eyelids should be examined, looking for foreign bodies and subconjunctival hemorrhage. The cornea, sclera, and conjunctiva should be examined by gross inspection as well as by slit lamp examination. The cornea should be examined after the patient has been upright for several minutes to allow visualization of a hyphema, if present. Evaluate the conjunctiva to detect lacerations, foreign bodies, or underlying scleral perforations.

Ocular motility should be assessed and documented. If a patient is unable to move the eye in a certain direction, concerns for entrapment of a muscle from an orbital blow out fracture, direct muscle injury or central nervous problem should be considered. If the complaint is diplopia after trauma, evaluation should assess whether the diplopia is monocular (typically lens displacement [after a lens surgery]) or binocular (entrapment, muscle injury, or central nervous system injury).

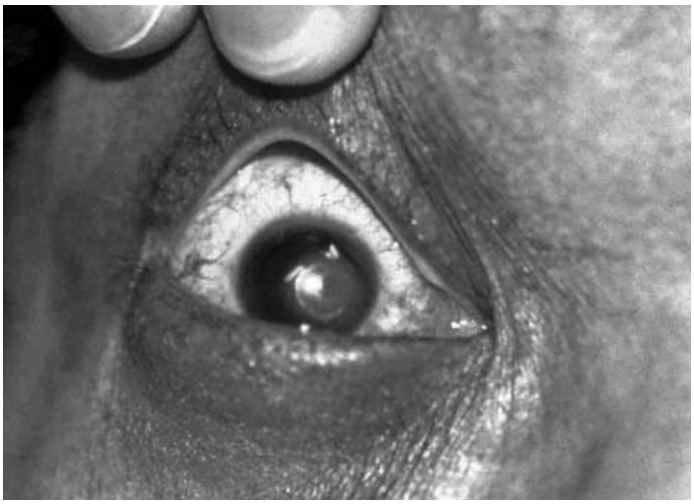
Visual field testing can be brief and simply involve the four major quadrants of vision; formal visual field testing can be done at a later time. Visual field testing is important to perform especially if the history suggests the possibility of retinal detachment or vitreous hemorrhage (e.g., new floaters or flashes of light after eye trauma). The patient should cover one eye; position yourself at eye level and hold your fingers halfway between yourself and the patient. Ask the patient to look at your nose and slowly bring fingers from the outside in, making sure the patient does not look away from your nose and towards the fingers. Binocular visual field loss is typically neurological in nature and not an ophthalmologic issue.

Slit lamp examination in older children is performed using the same technique as for adults. Younger children may feel more comfortable sitting on a parent's lap for the examination. Some tricks to having younger children more cooperative with the exam is to coax them into seeing something fun in the slit lamp (e.g., favorite cartoon character). Have parents place their chin in the device first to reassure the child, and tell them that they will see a pretty blue light. When examining infants, have parents sit and support the child's bottom with one hand, while the other hand rests on the back of the infant's head. If the child is not cooperative, a Wood's lamp may be an alternative to diagnosing corneal defects; however, Wood's lamp examination does not allow the detail of inspection afforded by slit lamp examination.

IOP evaluation is used to assess the significance of some eye injuries. IOP testing is contraindicated in suspected globe perforation, corneal abrasion, and foreign bodies. IOP evaluation is useful in patients with simple hyphemas, when ocular rupture is not suspected. IOP can be measured with a portable tonometer, or using slit lamp applanation tonometry. Normal IOP is 12 mmHg and increases by an estimated 1 mmHg for every decade of age over 40. Errors in tonometry can easily be made by placing too much pressure on the eye, blinking, or crying, all of which may artificially elevate IOP.

Finally, to view the posterior portions of the eye and retina, funduscopic examination is indicated. To assist in good visualization of the optic nerve and retina, have lights in the room turned off, and turn the intensity of the ophthalmoscope to its lowest setting and smallest aperture. While examining infants, making creative noises and clicking sounds will enhance your success. Toddlers may be afraid of the ophthalmoscope, but allowing them to see it up close and possibly hold it may take away some anxiety. Ideally, funduscopic examination is best performed after dilation. Pupils should not be dilated if serial neurologic exams are to be performed or if acute angle closure glaucoma is suspected.

Figure 1. Corneal Abrasion from a Fingernail Injury



Photograph courtesy of David Effron, MD.

Corneal Abrasions

Corneal abrasions are one of the most common eye injuries, and may occur from a variety of mechanisms, including projectiles, foreign bodies, direct trauma, fingernails, contact lenses, bee stings, and others.[14,15] Symptoms of corneal abrasion may include pain, tearing, blepharospasm, photophobia, or foreign body sensation.

Examination should include evaluation of the location, depth, and surface area of the abrasion, using fluorescein staining and slit lamp examination. *(See Figure 1.)* Lid eversion should be performed to exclude a retained foreign body. Current treatment includes pain control, topical antibiotics, topical analgesics, and cycloplegic agents. Eye patching has no demonstrated benefit and is no longer recommended for corneal abrasions.[16-19] Patching decreases depth perception and was shown in children to result in difficulty with ambulation.[19]

A topical antibiotic is indicated to prevent bacterial infection, although antibiotics have not been shown to improve outcomes. Appropriate choices may include sulfacetamide; a topical aminoglycoside; a topical quinolone, such as ciprofloxacin or ofloxacin drops; erythromycin ointment; polymyxin/trimethoprim drops; or numerous others. For contact lens wearers, a fluoroquinolone or aminoglycoside is preferred. Topical analgesics such as dicolfenac (Voltaren) and ketorolac (Acular) have shown a beneficial effect in reducing pain in corneal abrasions. A meta-analysis of five randomized, controlled studies showed a reduction of pain on a visual analog scale.[20] Patients using topical nonsteroidal anti-inflammatory drugs (NSAIDs) had greater relief of pain and used fewer oral analgesics and narcotics; however, an increase in initial transient stinging was commonly noted as an adverse effect.[20,21] The use of a cycloplegic agent, such as homatropine, or atropine, will relieve pain resulting from ciliary spasm. Home use of topical anesthetics should be avoided, due to negative effects on healing and increased risk of recurrent injury. Tetanus toxoid is routinely recommended for patients without updated immunization status;[22] however, a search of the literature has not identified any cases of clinical tetanus developing from a simple corneal abrasion.[23]

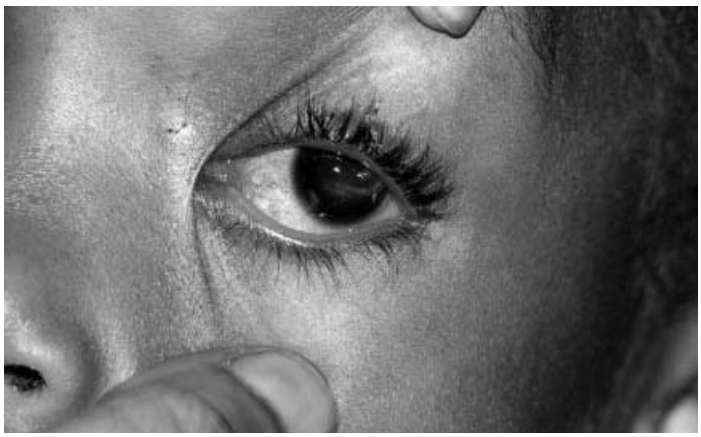
Most corneal abrasions heal well within 24 hours. Rarely, complications may occur, including delayed healing, scarring, infection, vision loss, or missed retained foreign body. Close follow-up is essential to ensure appropriate healing and relief of symptoms. In particular, injuries resulting from vegetable or botanical matter, or contact lens injury, are at higher risk for *Pseudomonas* infection and require close follow-up.

Corneal Foreign Bodies

A great variety of intraocular foreign bodies have been described, including metal fragments, wood, plastic, and others. Once determined that the injury to the eye is a simple foreign body and resultant corneal abrasion, removal of the foreign body should be performed under slit lamp visualization to provide consistent and stable removal of the offending irritant.[24]

Corneal foreign bodies can often be safely removed in the ED. An initial attempt to remove an irritant from the pediatric eye may be performed with a moistened cotton-tipped swab. If this effort is unsuccessful, the use of a needle is indicated to remove the foreign body. It is important to reassure the patient and parents that the needle does not go into the eye, but merely rests on the surface. Attach a standard 18-gauge needle to a 3 mL syringe for stability. Some prefer to bend the needle shaft 30° to facilitate the approach to the eye. The foreign body can then be gently lifted off the surface of the cornea. Once the foreign body is dislodged, use a moist cotton swab to remove it from the surface of the eye, if necessary. If a rust ring remains following removal of a metallic foreign body, this may also be removed in the ED with either the needle or a burr. It is also acceptable to schedule ophthalmology follow up for removal within 24–48 hours.[25]

Figure 2. Globe Rupture Resulting from a Stick Injury



Photograph courtesy of David Effron, MD.

Following foreign body removal, reexamine the eye for signs of ocular penetration. Pay particular attention for Seidel's sign, a leak of fluid that appears to be a dark stream of fluid on top of a green fluorescein background. After the foreign body is removed and signs of ocular perforation are excluded, the injury may be treated as a simple corneal abrasion.

Penetrating Ocular Injuries

Common causes of penetrating ocular injuries include glass, BBs, metallic fragments, toys, sticks, and wood/plastic particles that have become projectiles.[24,26] *(See Figure 2.)* Bony fracture fragments may also cause intraocular foreign bodies or injury.[27] Most injuries could have been avoided if proper safety equipment, mainly safety glasses, were worn.[28] Factors associated with worse prognosis include object with higher mass, or objects with a blade shape, as opposed to disc, cylinder, or sphere shapes.[29] Clinical factors predictive of poor outcome include visual acuity worse than 20/200, pupillary abnormalities, and hyphema. Children are at higher risk for suffering open globe injuries than adults; open globe injuries are the leading cause of monocular blindness in children world wide.[30] Associated injuries may include corneal injury, scleral laceration, retinal detachment, or numerous other injuries. Thus, whenever a penetrating injury to the eye is suspected, a complete ocular examination should be conducted, and ophthalmologic consultation considered.[31] Penetrating injuries may present immediately, or be delayed with complications arising up to years following the injury.[32]

Terms relevant to penetrating eye injuries include:

Laceration: A defect in the cornea or sclera caused by a sharp object;

Rupture: A disruption of the cornea or sclera caused by indirect forces or agents of blunt trauma, including low-velocity missiles such as BBs;

Perforation: Any injury that traverses the full thickness of the sclera; and

Table 1. Management of Perforating Eye Injuries
1. Document visual acuity
2. Protect eye with metal shield
3. Do not touch the eye or attempt tonometry
4. Do not instill any drops
5. Obtain imaging of the orbits (CT scan)
6. Administer systemic antibiotics
7. Administer tetanus prophylaxis
8. Administer antiemetics to prevent rise in intraocular pressure
9. Obtain immediate ophthalmologist consultation

Table 2. Pitfalls in the Management of Perforating Eye Trauma
• Intraocular foreign bodies may be missed in asymptomatic patients with normal vision.
• Negative plain radiographs or CT do not exclude the possibility of an intraocular foreign body.
• A negative Siedel test does not rule out full-thickness corneal laceration (may simply be result of self-sealing wound).
• All patients with suspected ocular perforation require emergent ophthalmology consultation.

Double perforation: Any injury that enters the eye, traverses the intraocular cavity, and exits the sclera on the opposite side.

The anatomy of the bony orbit protects the eye from most oblique and posterior injuries. Smaller missiles and sharp objects are often capable of entering the area within the orbital rim increasing the likelihood of piercing the globe. Therefore, ocular penetration must be suspected whenever there has been a laceration, puncture, or disruption of the eyelids or orbital bone, or periorbital ecchymosis.[33] In many cases, high-velocity projectiles that are small may penetrate the eyelid/globe and cause little pain or visual disturbance. The cornea may seal over the entry portal, leading to a paucity of eye findings. Intraocular foreign bodies should be considered in all penetrating eye injuries. If a penetrating globe injury is suspected, no pressure should be exerted on the eye because of risk of expelling intraocular contents. Perform a complete neurologic exam and obtain a thorough history of the events, keeping in mind that children may not tell the entire truth for fear of negative consequences. A detailed ocular exam should include visual acuity testing; pupillary testing; examination of the cornea, sclera, and anterior chamber with slit lamp, when possible; and funduscopic exam. Signs of possible perforation, such as bloody chemosis of the sclera, collapse and hemorrhage in the anterior chamber, pupil and iris irregularities, positive Seidel's test, and traumatic cataract should be sought and noted when present. Seidel's test is administered by instilling fluorescein into the affected eye directly at the site of the suspected perforation, and examining under slit lamp for a bright green stream of fluid resulting from the outflow of aqueous humor. If examination is unable to be adequately performed due to emotional distress or poor cooperation, suspicion of a penetrating injury justifies an evaluation under anesthesia in the operating room, where the nature and extent of the injury may be adequately addressed. *(See Table 1.)*

All penetrating eye injuries or suspected penetration warrant radiographic imaging. Although plain radiographs may serve to reveal the size and number of metallic foreign bodies, the exact three-dimensional orientation of the foreign body with respect to the globe is not delineated. Plain films also are not adequate for full evaluation of adjacent bony structures, sinuses, soft tissue, and the evaluation of non-metallic objects that may be readily seen on computed tomography (CT). A randomized, retrospective study of the accuracy of CT in evaluating open globe injuries has shown a sensitivity of 75% and specificity of 95% for picking up such injuries in the absence of clinical exam or historical findings.[34] It is therefore recommended that patients with suspected intraorbital foreign bodies undergo CT with 1.5–3.0 mm cuts with coronal reconstruction when available. *(See Table 2.)*

Ultrasonography may also be helpful in the identification of intraorbital foreign bodies, although suboptimal sensitivity suggests that additional imaging should be performed if suspected clinically.[35] In addition, intracranial foreign body may be associated with perforating ocular injuries, and intracranial imaging should be considered.[36]

Hyphema

Hyphema, blood in the anterior chamber, is a common condition seen after blunt ocular trauma in pediatric patients, and results from rupture of the iris or ciliary body blood vessels.[13,37] Hyphema is more common among males, particularly ages 15–18 years. Examples of trauma resulting in hyphema include blunt trauma, projectiles, or explosions, including specific examples of fists, balls, motor vehicle collisions, rocks, airbags, sticks, pellet guns, toys, tools, and numerous others.[38,39] Hyphema usually occurs from a tear in the anterior portion of the ciliary body. Hyphema should be observed after the patient has been sitting upright for several minutes, to allow layering and visualization of the hyphema. Intraocular pressure may be elevated. Other associated eye injuries may occur, including corneal injuries (26%–40% incidence), posterior segment injuries, and rebleeding. Hyphemas are classified according to amount of hemorrhage.

Complications of hyphema may include corneal blood staining, vision loss, secondary glaucoma, secondary hemorrhage, and optic nerve atrophy. Factors related to poor outcome include grade of hyphema, intraocular hypertension, time for hemorrhage absorption, and associated posterior segment lesions.[40] *(See Table 3.)* Good visual recovery occurs in only 35% of patients with total hyphema.

Corneal blood staining occurs in 2%–11% of patients with hyphema, and occurs more commonly among higher-grade

Table 3. Classification of Traumatic Hyphema

GRADE	FREQUENCY	PROGNOSIS OF 20/50 OR BETTER
Microhyphema	6%–28%	75%–90%
I	44%–77%	75%–90%
II	3%–20%	65%–70%
III	1%–14%	25%–50%
IV	1%–8%	25%–50%

Adapted from Brandt MT, Haug RH. Traumatic hyphema: A comprehensive review. *J Oral Maxillofac Surg* 2001;59:1462-1470.

Table 4. Management of Hyphema

- Elevation of head to 30 degrees
- Rigid eye shield
- Topical antibiotics
- Topical corticosteroids
- Cycloplegics
- Avoid antiplatelet agents
- Treat elevated IOP (if indicated)

hyphemas. Corneal blood staining may lead to vision loss or amblyopia. Increased IOP is common; about one-third of patients with hyphema have increased IOP.

Secondary hemorrhage is associated with increased morbidity, including corneal blood staining, secondary glaucoma, optic nerve atrophy, and vision loss. Secondary hemorrhage typically occurs 2–7 days after the primary injury, and the incidence is higher in patients with poorer initial visual acuity, large hyphema (more than 33% of anterior chamber), delayed medical attention, initial elevated IOP, and use of antiplatelet medications.[41] It has been estimated that secondary hemorrhage occurs in 5%–22% of patients with hyphema.[41]

Outpatient management is feasible for most patients with low-grade hyphema.[40] Although traditional management in the past included strict bed rest, sedation, and eye patching, these treatments have not demonstrated improved outcomes. Hospital admission is usually not necessary and is not associated with improved outcome, although admission may be indicated for patients with secondary hemorrhage, markedly elevated IOP, sickle cell disease, hemophilia, or vision loss; in cases of child abuse; or for noncompliant patients.[42] Elevation of the head to 30° aids in hyphema clearance and facilitates the diagnosis of secondary hemorrhage. Vigorous physical activity should be avoided. The use of eye patching is controversial. Patching may improve comfort and reduce eye movement, although some authors argue that patching may raise the eye temperature and promote bacterial growth. Typically, topical antibiotics, cycloplegic agents, and topical corticosteroids are indicated. *(See Table 4.)* Other treatments remain controversial, and may include topical cycloplegics, systemic steroids, topical or systemic antifibrinolytics, topical beta-blockers, and carbonic anhydrase inhibitors. Surgical treatment, including paracentesis, hyphectomy, clot irrigation, or trabeculectomy, are indicated only for uncontrolled elevated IOP, corneal staining, or large hyphemas.

Table 5. Chemical Eye Burn Classification

GRADE	APPEARANCE	PROGNOSIS
I	Hyperemia, opacification	Complete recovery
II	Chemosis, opacification	Complete recovery
III	Severe chemosis, opacification, tissue damage	Scarring, visual loss
IV	Extensive necrosis, chemosis, opacification	Scarring, visual loss

Thermal and Chemical Burns

Chemical burns to the eye occur as a result of direct exposure to caustic chemicals. Common sources include household cleaning agents, automotive cleaners, swimming pool chlorine, battery acid, and other chemicals. Chemical burns are classified by tissue damage that is associated with prognosis. *(See Table 5.)*

Alkali burns are often more extensive than corresponding acid burns. Alkali rapidly penetrates the cornea and leads to increased pH of the aqueous, causing liquefaction necrosis, with extensive loss of corneal epithelium. Widespread tissue damage may follow rapid rise in pH.

Acid burns may also be severe; however, the coagulation necrosis initiated by acid injury limits the extent of tissue injury in many cases. Following acid exposure, protein coagulation in the corneal epithelium produces a barrier to deeper penetration of acid. Hydrofluoric acid is an exception to this mechanism and may result in extensive corneal penetration and injury.

Chemical burns should be managed with immediate and copious irrigation, which improves prognosis and outcome.[43,44] Effective irrigation may require topical anesthesia and/or systemic sedatives. Rapid initiation of irrigation is more important than the precise irrigant solution used.[45] Irrigation may be performed with a variety of readily available solutions. Normal saline (NS, 0.9%) has a pH 4.5–6.0 and may be associated with mild discomfort. Alternative irrigants include water (readily available, but hypotonic), lactated Ringer's solution (pH 6.2–7.5), buffered NS with pH adjusted to 7.4 with sodium bicarbonate, and Balanced Salt Solution Plus (BSS Plus), with a pH of 7.4.[46] The use of an intraocular irrigation lens may improve the contact of the irrigant with the cornea. If the pH of the chemical causing injury is unknown, pH testing should be done to ascertain the acidity of the substance. Following alkali burns, regular pH assessment with litmus paper should be performed intermittently until the pH normalizes. Acid burns should be treated with irrigation for 15–20 minutes; alkali burns may require several hours of copious irrigation.

Table 6. Management of Chemical Burns to the Eye

- Immediate and copious irrigation until pH normalizes
- Foreign body removal (if indicated)
- Debridement of devitalized tissue
- Lubricants
- Artificial tears
- Topical steroids
- Topical antibiotics
- Management of increased intraocular pressure (if indicated)

Thermal burns are less common, but may occur as a result of splash injuries, cigarettes/cigars, fireworks, matches, lighters, curling iron contact, or other mechanisms. The eye is the most common body site injured by fireworks among pediatric patients, followed by injury to face and hands.[5] Thermal burns may be extensive and are proportionate to the time of contact and temperature of the burn surface. Often eye protective mechanisms spare significant eye injury, including the blink, tears, bony orbit structure, and shielding of the face by hands and arms.

Following either chemical or thermal burns, a complete eye examination and assessment should be performed, including visual acuity, IOP, and slit lamp examination. If devitalized tissue or foreign bodies are present, debridement and removal should be performed. Following irrigation for both chemical and thermal burns, supportive measures during the healing process for both chemical and thermal burns should include ocular lubricants, artificial tears, topical corticosteroids, and topical antibiotics. *(See Table 6.)*[47-49] If IOP is elevated, appropriate therapy should be instituted. Appropriate pain management may include systemic NSAIDs or opioids. Ophthalmologic consultation is indicated for most injuries.[50] Surgical management may be indicated for significant necrotic tissue or severe injury.

Blunt Eye Trauma

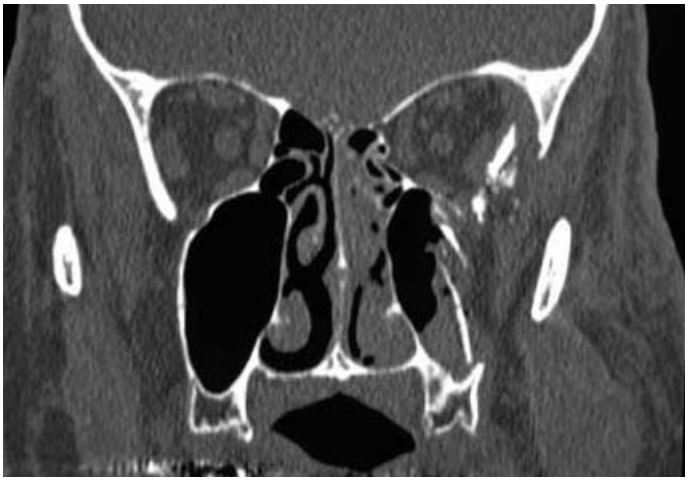
Blunt injuries to the eyes range from a spectrum of simple contusion to retinal detachment or globe rupture. The most common cause of blunt trauma to the eye in children is sporting and recreational events, which make up more than 59% of blunt injuries to the eye.[2] Basketball, baseball, water sports, and racquet sports account for most injuries.[51] The extent of the damage to the eye is dependent of the size, velocity, hardness of the object, and the amount of direct force placed on the eye. It is well established that penetrating injuries to the eye have a poorer prognosis than blunt injuries; however, serious blunt injuries can cause a significant intraocular disruption that may cause vision-threatening injuries that are critical to be recognized and referred.[8] There are two major blunt ocular injury patterns that every emergency physician should be aware of: direct globe injury and orbital fracture. A direct blow to the globe is usually caused by smaller objects that are able to bypass the bony orbital rim and strike the eye directly. Forces that directly hit the eye cause a rapid compression of the globe anterior–posteriorly and corresponding expansion/dilation of the middle of the globe. The transmission of these forces produces extensive tearing and stretching forces, causing several types of injuries.

When the eye is struck, the anterior chamber is compressed, and the pupil is forced to dilate rapidly. The iris may tear, causing rupture of sphincter papillae, which can lead to traumatic mydriasis. Force distributed directly to the peripheral iris can cause the iris to disinsert from its root (iridodialysis). A traumatic cataract may be seen when compressive forces damage the lens. Alternatively, the lens can be dislocated by the tearing of its insertion at the zonules. Signs of lens subluxation or dislocation include blurry vision and monocular diplopia. Damage to any of the structures in the anterior chamber can cause bleeding leading to hyphema. The importance of finding a hyphema on physical exam is that it is an indicator of serious ocular injury and that structures in the anterior as well as posterior segments are likely to be damaged. As forces are distributed beyond the lens, injuries to the posterior segment are possible. The vitreous humor is attached to the retina and large stresses are able to disinsert the retina, leading to retinal detachment. The choroid may be stretched and torn; due to the vascular nature of this structure, choroidal rupture may occur, presenting as subretinal blood on funduscopic exam. Finally, commotio retinae may be seen after stretching and edema of the retina. This localized injury is identified as a whitish discoloration of the retina, on funduscopic exam, caused by edema of the photoreceptor cells in the retina. Significant complications may arise as a result of blunt trauma, including vision loss, disfiguration, secondary glaucoma, or traumatic optic neuropathy.[52] General treatment for blunt ocular injuries include prophylactic eye shield, pain control and ophthalmologic consultation. Guidelines for immediate referral include globe rupture or suspected rupture, pupil defect with dense periorbital hematoma, hyphema, significant vision loss, or subconjunctival hemorrhage (may be masking globe rupture). Blunt injuries that may require ophthalmologic consultation within 24 hours include minor reduced visual acuity, retinal hemorrhages, eyelid lacerations, and blowout fractures.

Orbital Wall Fractures

Orbital fractures are typically caused by blunt impact by objects larger than the orbital rim or by impacts with flat surfaces, including falls and motor vehicle collisions Seven bones of the skull come to form the orbit: maxilla, zygoma, lacrimal, ethmoid, sphenoid, palatine, and frontal. The term "blow-out fracture" refers to buckling of the orbital floor, following intense intraorbital pressure associated with blunt trauma, often protecting the eye from more serious internal injury.[53] Entrapment of periorbital tissues, commonly including muscle, may occur as a result of phase differences of the movement of various orbital structures.[54] Orbital entrapment occurs more commonly in pediatrics, thought to be due to the pliable nature of the orbital floor. The most common area to fracture is the orbital floor and medial wall due to thinner bone in these regions. Orbital roof fractures

Figure 3. Computed Tomography of Left Intraorbital Fracture



Photograph courtesy of Ann Dietrich, MD.

make up about 5% of injuries. Children younger than 7 years are more prone to these injuries. The orbital roof is a very thin structure, and frontal sinuses are not fully pneumatized until approximately age 7 years. Blunt trauma that impacts the upper portion of the orbit is dissipated by the frontal sinus. Orbital roof fractures may communicate with the brain and result in more serious injuries, including intracranial hemorrhage and leakage of cerebrospinal fluid. Symptoms of an orbital fracture include periorbital ecchymosis, diplopia, hypoesthesia in V_2 (lower eyelid, cheek and upper lip) distribution, pain with ocular movement (especially vertical movement), and intraorbital emphysema on plain radiograph. Symptoms of nausea and vomiting may be associated with inferior rectus entrapment and poor outcome.[55] Diagnosis is made by radiographic imaging with CT or magnetic resonance imaging (MRI). *(See Figure 3.)* Plain radiographs of the orbits have a high false negative or non-diagnostic rate ranging from 30%–50%. CT is often more widely available, but also carries the risks associated with significant radiation exposure.[56,57] MRI typically shows better resolution and causes no radiation exposure, but may require sedation to perform and is less readily available. High-resolution MRI imaging with an orbital coil shows promise in the diagnosis of pediatric orbital fractures.[58] Limitations of orbital radiography should be noted, as fractures and entrapment may be missed by radiography alone.[59,60]

Once the diagnosis is made, two management schema are available — surgical or non-surgical. Three specific findings are of particular concern, including diplopia (entrapment), restriction of extraocular movements, and enophthalmous > 2 mm. Careful ophthalmologic examination is essential, as 26% of patients with orbital fractures have associated ocular injuries.[61] Most orbital fractures requiring surgical intervention are repaired within two weeks.[53] The initial management of orbital blowout fractures includes treatment of the area with ice for 48 hours, nasal decongestants, broad spectrum antibiotics, elevation of head of bed while sleeping, avoidance of aspirin, avoidance of nose-blowing, and ophthalmologic consultation. Consideration for early consultation should be made if mechanism of injury and physical exam suggest a serious intraocular injury.

Retrobulbar Hemorrhage

Retrobulbar hemorrhage is a true emergency that may require urgent intervention to preserve vision. This condition is effectively a compartment syndrome within the orbit and must be managed with the same, if not more, haste as any other compartment syndrome. The time until the oxygen-sensitive tissues of the retina are irreversibly damaged is 60 minutes.[62] This sight-threatening injury typically arises from orbital bleeding following a non-displaced fracture of the orbital wall. As with other compartment syndromes, the natural progression of the condition is increased pressure around the globe, resulting in reduced retinal perfusion, compression of ciliary vessels, stretching of the optic nerve, and exophthalmos.[63] In most cases, development of retrobulbar hemorrhage occurs within a few hours of injury, but case reports have shown that delayed retrobulbar hemorrhages may appear after the initial injury.[64] Retrobulbar hemorrhage must be treated immediately if there is evidence of vision loss. Clinical signs include pain, proptosis, loss of vision, and presence of an afferent pupillary defect. In the unconscious patient, a tense, proptosed globe and a dilated pupil may be all that is apparent in the presence of a retrobulbar hemorrhage. CT scanning of the brain and orbits will help identify orbital wall fractures and retrobulbar blood if the diagnosis is in question. The management of a retrobulbar hemorrhage is surgical and emergent ophthalmologic consultation is warranted. Emergent lateral canthotomy can be performed in the ED as a temporizing measure, until formal decompression can be performed under general anesthesia. Once the lateral canthus is incised and the canthal tendon cut, the globe can move forward, allowing increased retrobulbar volume. Other medical treatments of potential benefit include intravenous steroids, acetazolamide, and mannitol. Definitive treatment is surgical decompression and repair.

Eyelid Lacerations

Eyelid lacerations are commonly seen in pediatric patients, and are often due to falls, motor vehicle collisions, or direct impact. Evaluation should assess potential globe injury and should include visual acuity, slit lamp examination, IOP measurement, and facial nerve function. Potential bony fractures should be ruled out by examination and radiographs, if indicated. *(See Figure 4.)*

Simple eyelid lacerations can be successfully repaired in the ED. Superficial wounds not involving the lid margins can be closed with single or two-layer closures with small suture material. Slight eversion of wound edges should reduce scar forma-

Figure 4. Multiple Ocular Injuries from Dog Bite

Injuries include tear duct injury and facial lacerations.

Photograph courtesy of David Effron, MD

tion. The dermal layer should not be under tension. Complex wounds, including tarsal plate involvement, canaliculi involvement, lid margin involvement, or scleral involvement may require ophthalmologic or plastic surgery consultation.[65] Debridement of devitalized tissue may be indicated. Tetanus toxoid should be administered if indicated.

Child Abuse

Certain eye findings may be indicative of child abuse. Any injury which does not fit the characteristic pattern and severity of the described injury should heighten suspicion of possible abuse. Intraocular hemorrhage occurs rarely with accidental head trauma and is commonly associated with shaken-baby syndrome, particularly when seen in association with subdural hemorrhage, abnormal mental status, or seizures.[66,67] Bilateral subconjunctival hemorrhages may be seen in infants as a result of asphyxia.[68]

Conclusions

Eye trauma is a common chief complaint among ED pediatric patients. Many eye injuries can be prevented by appropriate supervision of children's activities and the appropriate use of protective eyewear during sports. Detailed physical examination is indicated for the evaluation of pediatric eye trauma, and should include visual acuity, pupillary reactions, external examination, ocular motility, visual field testing, slit-lamp examination, fluorescein staining, funduscopic examination, and IOP (when indicated). Most eye injuries can be managed on an outpatient basis with ophthalmologic follow-up and appropriate medical management. Certain eye injuries warrant emergent ophthalmologic consultation, including high-grade hyphema, penetrating globe injury, suspected or known open globe injury, retrobulbar hematoma, or any eye injury resulting in significant vision loss.

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b.) Describe the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

CME Questions

101. What is the most essential aspect of the physical examination of the injured eye?
    A. Intraocular pressure
    B. Ocular motility
    C. Pupillary response
    D. Visual acuity
    E. Visual field testing

102. A 5-year-old child presents with a corneal abrasion resulting from being hit in the eye with a tree branch. What type of secondary infection poses a threat to uncomplicated healing?
    A. *Aeromonas hydrophilia*
    B. *Clostridium perfringens*
    C. Methicillin-resistant *Staphylococcus aureus*
    D. *Pseudomonas aeruginosa*
    E. *Streptococcus pneumonia*

103. A 15-year-old male presents with a corneal abrasion resulting from prolonged contact lens wear. What type of secondary infection poses a threat to uncomplicated healing?
    A. *Aeromonas hydrophilia*
    B. *Clostridium perfringens*
    C. Methicillin-resistant *Staphylococcus aureus*
    D. *Pseudomonas aeruginosa*
    E. *Streptococcus pneumonia*

104. What is the preferred method of removal of corneal foreign bodies?
    A. Removal with an 18-gauge needle
    B. Removal with a moistened cotton-tipped swab
    C. Removal with an optical burr
    D. Outpatient removal within one week by ophthalmologist

105. Which of the following intraocular medications should be administered to patients with suspected perforating globe injury:
   A. Antibiotics
   B. Cycloplegics
   C. Pilocarpine
   D. Nonsteroidal anti-inflammatory drugs
   E. No intraocular medications should be administered.

106. What is the priority management issue for a patient who presents with suspected alkali burn to the eye?
   A. Assessment of visual acuity
   B. Measurement of intraocular pressure
   C. Irrigation
   D. Ophthalmic antibiotic drops
   E. Slit lamp examination with fluorescein

107. What acid is associated with extensive corneal injury?
   A. Hydrofluoric acid
   B. Hydrochloric acid
   C. Sulfuric acid
   D. Carboxyl acid
   E. Oxoacetyl acid

108. What clinical finding suggests the need for urgent surgical consultation for orbital blowout fracture?
   A. Photophobia
   B. Periorbital ecchymosis
   C. Grade I hyphema
   D. Corneal abrasion
   E. Diplopia

109. What ED procedure should be performed for vision loss secondary to retrobulbar hemorrhage?
   A. Lateral canthotomy
   B. Measurement of intraocular pressure
   C. Seidel's test
   D. Anterior chamber paracentesis
   E. Closed globe massage

110. What eye injury is commonly associated with nonaccidental child injury?
   A. Hyphema
   B. Intraocular hemorrhage
   C. Chemical burn
   D. Retrobulbar hemorrhage
   E. Corneal abrasion

Answers: 101. D, 102. D, 103. D, 104. B, 105. E, 106. C, 107. A, 108. E, 109. A, 110. B

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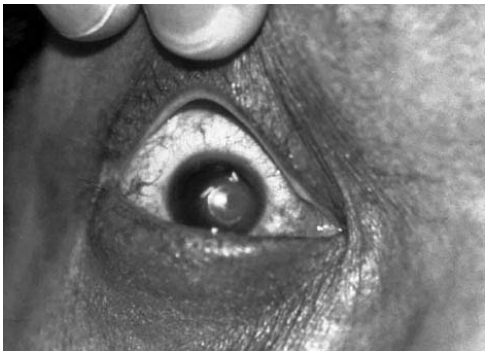
The Practical Journal of Pediatric Emergency Medicine

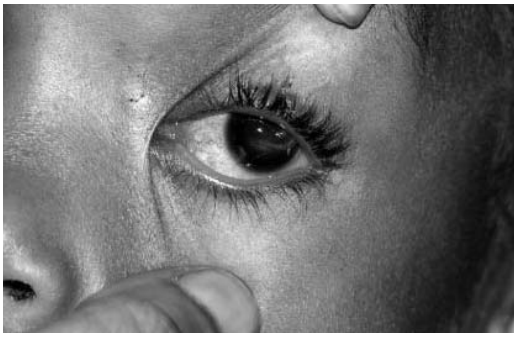
Eye Injuries

Corneal Abrasion from a Fingernail Injury



Photograph courtesy of David Effron, MD.

Globe Rupture Resulting from a Stick Injury



Photograph courtesy of David Effron, MD.

Management of Perforating Eye Injuries

1. Document visual acuity
2. Protect eye with metal shield
3. Do not touch the eye or attempt tonometry
4. Do not instill any drops
5. Obtain imaging of the orbits (CT scan)
6. Administer systemic antibiotics
7. Administer tetanus prophylaxis
8. Administer antiemetics to prevent rise in intraocular pressure
9. Obtain immediate ophthalmologist consultation

Pitfalls in the Management of Perforating Eye Trauma

- Intraocular foreign bodies may be missed in asymptomatic patients with normal vision.
- Negative plain radiographs or CT do not exclude the possibility of an intraocular foreign body.
- A negative Siedel test does not rule out full-thickness corneal laceration (may simply be result of self-sealing wound).
- All patients with suspected ocular perforation require emergent ophthalmology consultation.

Classification of Traumatic Hyphema

GRADE	FREQUENCY	PROGNOSIS OF 20/50 OR BETTER
Microhyphema	6%–28%	75%–90%
I	44%–77%	75%–90%
II	3%–20%	65%–70%
III	1%–14%	25%–50%
IV	1%–8%	25%–50%

Adapted from Brandt MT, Haug RH. Traumatic hyphema: A comprehensive review. *J Oral Maxillofac Surg* 2001;59:1462-1470.

Management of Hyphema

- Elevation of head to 30 degrees
- Rigid eye shield
- Topical antibiotics
- Topical corticosteroids
- Cycloplegics
- Avoid antiplatelet agents
- Treat elevated IOP (if indicated)

Chemical Eye Burn Classification

GRADE	APPEARANCE	PROGNOSIS
I	Hyperemia, opacification	Complete recovery
II	Chemosis, opacification	Complete recovery
III	Severe chemosis, opacification, tissue damage	Scarring, visual loss
IV	Extensive necrosis, chemosis, opacification	Scarring, visual loss

Management of Chemical Burns to the Eye

- Immediate and copious irrigation until pH normalizes
- Foreign body removal (if indicated)
- Debridement of devitalized tissue
- Lubricants
- Artificial tears
- Topical steroids
- Topical antibiotics
- Management of increased intraocular pressure (if indicated)

Computed Tomography of Left Intraorbital Fracture

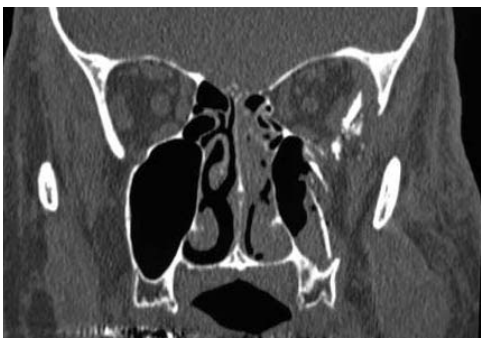


Image courtesy of Ann Dietrich, MD

Multiple Ocular Injuries from Dog Bite

Injuries include tear duct injury and facial lacerations.

Photograph courtesy of David Effron, MD

Supplement to *Pediatric Emergency Medicine Reports*, November 2009: "Keep an 'Eye' on the Patient: Update on Pediatric Eye Trauma." *Author:* **Catherine A. Marco, MD, FACEP**, Professor, Department of Surgery, Director of Medical Ethics Curriculum, University of Toledo College of Medicine, Toledo, OH. *Peer Reviewer:* **Dennis Hanlon, MD, FAAEM**, Vice Chairman, Emergency Medicine, Allegheny General Hospital, Pittsburgh, PA. *Pediatric Emergency Medicine Reports' "Rapid Access Guidelines."* **Senior Vice President and Group Publisher:** Don Johnston. **Editor-in-Chief:** Ann Dietrich, MD, FAAP, FACEP. **Associate Publisher:** Coles McKagen. **Managing Editor:** Allison Weaver. For customer service, call: **1-800-688-2421.**

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Traumatic brain injury (TBI) is a leading cause of mortality in the United States and represents over half of trauma related deaths.[1,2] Approximately 1.4 million people suffer from a TBI each year.[3] (See Figure 1.) Of these, 1.1 million are evaluated in the emergency department (ED) and subsequently discharged home while another 235,000 are admitted. Of those admitted, approximately 50,000 die annually. Those 14 years and younger accounted for 435,000 ED visits, 37,000 hospitalizations, and more than 2,600 deaths. Males are 1.5 times more likely than females to sustain TBI. Those between the ages of 0–4 years and 15–19 years have the highest TBI-related ED visits, however, adults older than 75 years have the highest rates of TBI-related hospitalization and death.[3] The authors comprehensively review the presentation and management of patients with TBI.

— The Editor

Traumatic Brain Injury

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Introduction

By the year 2030, the number of persons older than age 65 will double relative to 2000, representing almost 20% of the nation's total population.[4] As the population ages, so does the number of elderly people who are admitted to trauma centers with TBI. Older age has been well recognized as an independent predictor of worse outcome after TBI, even with relatively minor head injuries.[6,7]

The Centers for Disease Control and Prevention estimates currently there are at least 5.3 million Americans who have long-term or life-long disability as a result of TBI,[8] with an additional 80,000–90,000 added annually.[3] In 2000, the estimated direct and indirect medical costs including injury-related work loss, disability and lost income from pre-mature death as a result of TBI was $60 billion.[9]

Falls are the leading cause of TBI with motor vehicle collisions (MVC) second. *(See Figure 2.)* However, for those age

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Dr. Dietrich (editor in chief), Drs. Menaker and Scalea (authors), and Lipman (peer reviewer), and Ms. Behrens (nurse reviewer) report no relationships with companies related to this field of study.

15–19, MVCs are the leading cause of TBI.[3] Overall, MVCs result in the greatest number of all TBI-related hospitalizations, while firearm use is the leading cause of death related to TBI.[3,10]

Pathophysiology

The cranium is a rigid vault that contains three compartments: brain tissue, blood, and cerebrospinal fluid (CSF); the pressure in the skull exerted by these elements is termed intracranial pressure (ICP). According to the Monroe-Kellie doctrine, the intracranial volume is a fixed space and additional collections, such as mass lesions or hemorrhage, must be accompanied by a decrease in another compartment, or ICP will increase. Various intrinsic mechanisms exist to prevent increases in ICP after trauma. These include shunting of the CSF to the spinal subarachnoid space, increasing CSF absorption and shunting venous blood out of the skull.[11] However, eventually this compensation fails and ICP rises.

Cerebral perfusion pressure (CPP) is used as an estimate of cerebral blood flow (CBF), which is difficult to accurately measure clinically. CPP is the difference between the mean arterial pressure (MAP) and the ICP (CPP = MAP - ICP). Normal ICP is 0–10 mmHg, and therefore CPP closely relates to the MAP. Under normal conditions, when CPP is between 50 mmHg and 150 mmHg, the brain has the ability to autoregulate to maintain a constant CBF. However, after TBI, autoregulation fails and the relationship between CPP and CBF becomes linear.[12] This results in increased CBF (and ICP) as CPP increases.

TBI results from both direct and indirect forces. Direct injury is a result of the initial insult and typically occurs at site of impact. Indirect forces are a consequence of the acceleration/deceleration of the brain tissue within the skull and cause injury on the opposite side of impact (coup-contrecoup injury). In addition, this acceleration/deceleration mechanism can cause shearing of neurons, causing diffuse axonal injury (DAI).

TBI may further be classified as primary and secondary injury. Primary injury is a result of the initial trauma, and occurs at the time of injury. These injuries are irreversible, and thus efforts must be aimed at injury prevention. However, after TBI, a cascade of cellular events begins that leads to what is known as "secondary injury." It is the leading cause of in-hospital death after TBI.[13] Most secondary brain injury is the result of cerebral edema with a resultant increase in ICP and decrease in CPP leading to ischemia.[14] As autoregulation fails, CBF and CPP become directly proportional. The increase in ICP lowers the threshold of systemic blood pressure for cerebral ischemia. Secondary injury should be differentiated from secondary insults. The former is a direct result of the primary injury. The latter is a result of discrete, sometimes iatrogenic processes independent of the primary injury.

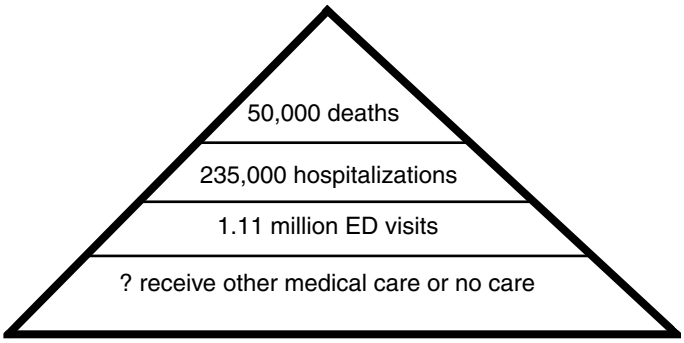
Hypoxia and hypotension are two examples of secondary insult. They often occur in patients with TBI prior to the arrival of pre-hospital providers or arrival to the hospital and are independently associated with significant increases in morbidity and mortality.[15] A single episode of hypotension has been shown to double mortality rates in patients with TBI.[15] Clinical decisions should attempt to minimize secondary insult and improve clinical outcome.

Intracranial Hematomas and Contusions

Subdural Hematoma. Subdural hematoma (SDH) is a result of sudden acceleration/deceleration tearing of the bridging veins. Blood accumulates between the dura matter and the arachnoid, and radiographically it appears as a crescent- or sickle-shaped collection. *(See Figure 3.)* Brain atrophy is common in the elderly and alcoholics, making them more susceptible to SDH. Although the blood accumulates slowly, due to it venous origin, SDH is associ-

Figure 1. TBI in the United States Annually

At least 1.4 million traumatic brain injuries (TBIs) occur in the United States each year:*



*Average annual numbers, 1995-2001.

Source: Langlois JA, Rutland-Brown W, Thomas KE. Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2004.

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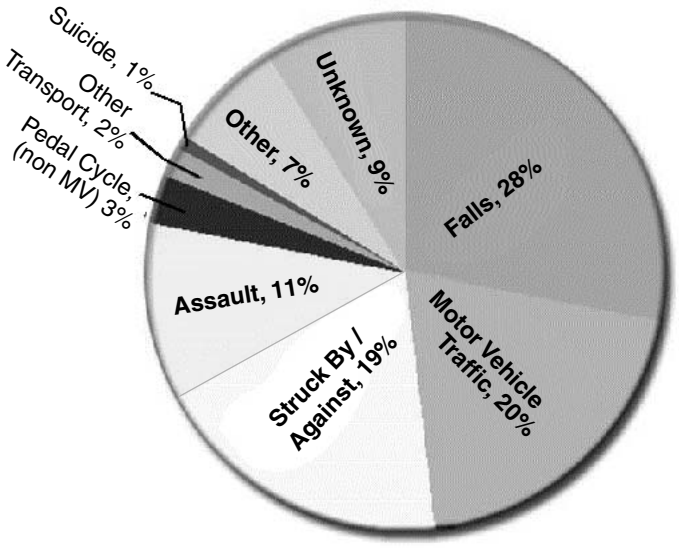
Figure 2. Causes of TBI



Source: Centers for Disease Control and Prevention (CDC), National Center for Injury Prevention and Control. Traumatic brain injury in the United States–A report to Congress. Atlanta (GA): Centers for Disease Control and Prevention; 1999.

ated with more brain parenchymal damage, as opposed to other types of TBI, and thus has worse outcome. However, early surgical evacuation, within four hours of injury, decreases mortality.[16]

SDH accounts for approximately 25%–30% of TBI[17] and can be classified as acute (< 48 hours), subacute (2–14 days) or chronic (> 14 days). Patients with an acute SDH will often present unconscious or with lateralizing signs on exam. (i.e., a unilateral, non reactive, dilated pupil) More chronically, these patients may have a gradual decline in consciousness. Others may have balance issues that will often lead to presentation to the ED. Radiographically acute SDH appear as hyperdense (white) lesions, while subacute and chronic SDH are isodense and hypodense, respectively.

Epidural Hematoma. Epidural hematomas (EDH) typically result from tearing of the middle meningeal artery associated with temporal bone fractures. Blood accumulates between the skull and the dura and gives a biconvex-, lens-, or football-shape collection on CT scan. *(See Figure 4.)* They are more common in younger people and are rare in the elderly and those younger than 2 years of age. In the elderly, the dura is tightly adhered to the skull; thus, blood does not accumulate in this space.

The classic description of a patient with an EDH is someone who loses consciousness immediately after TBI and then awakens to a normal state of consciousness. As the EDH continues to expand, the patient once again loses consciousness. This "lucid interval"' in reality occurs in only 20%–30% of patients.[17] EDHs are not parenchymal injuries, and thus rapid diagnosis and treatment is imperative to prevent herniation and improve outcome.

Subarachnoid Hemorrhage. Traumatic subarachnoid hemorrhage (tSAH) results from injury to arteries surrounding the subarachnoid space. This leads to buildup of blood between pia mater and the arachnoid. On CT scan, blood is seen in the basal cisterns, and hemispheric sulci and fissures. *(See Figure 5.)* The blood is spread diffusely and does not cause a mass effect. Posttraumatic SAH is, however, a marker of severity of injury. Multiple studies have demonstrated those with tSAH on admission CT scan have poorer outcome compared to those who do not have tSAH[18-20]; this is likely related to the initial mechanical damage, as opposed to delayed secondary injury.[19]

Much like aneurysmal SAH, up to 20% patients with tSAH may develop vasospasm.[21] This sometimes leads to hydrocephalus from decreased CSF absorption when the arachnoid villi get obstructed by blood product degradation.[21] Temporary ventriculostomy placement or a permanent ventriculo-peritoneal shunt is sometimes required.[22]

Intracerebral Hematoma/Contusion. Intracerebral contusions result from the brain parenchyma's impact with the skull. *(See Figure 6.)* Typically they are found in the frontal and temporal regions of the brain. Coup contusions occur at the site of impact, where as contrecoup injuries occur at the opposite side of impact. Contusions often evolve over time and increase in size on subsequent CT scans. As they increase in size, the mass effect and cerebral edema worsen and patients often deteriorate.

Diffuse Axonal Injury. Diffuse axonal injury (DAI) is a result of shearing of the axons in the white matter and brainstem during sudden deceleration. These injuries often are not dramatic on the initial CT scan, but clinically the patient has significant neurologic deficits.

Clinical Classification of Traumatic Brain Injury

Glasgow Coma Scale. The Glasgow Coma Scale (GCS) was first described in 1974.[23] The scale was developed to standardize a neurological scoring system and allow reliable inter-observer evaluation of patients with TBI. GCS is best evaluated after resuscitation, as hypoxia, hypotension, hypothermia, or hypoglycemia may cause a decreased mental status unrelated to TBI. The scale is based on three aspects of neurologic function: eye opening, verbal functioning, and motor function. *(See Table 1.)* The best response from each section is added together for a final score ranging from 3 to 15. For those who are intubated, the verbal scale can be replaced with "T," and thus the best response becomes an "11T."

A single score is insufficient to determine the extent of injury, and serial examinations should be performed. Patients who are intubated cannot be assessed for the verbal component, and those chemically paralyzed cannot be assessed for any of the three categories. In addition, those with severe facial trauma may not be physically able to open their eyes due to edema. Patients with a GCS of 13–15 are classified as having mild TBI, 9–12 as moderate, and 3–8 as severe. However, some consider a GCS of 13 to be a moderate TBI.

Mild Traumatic Brain Injury. Mild traumatic brain injury (mTBI), often called a concussion, is one of the most common neurologic disorders and accounts for approximately 80% of TBI. It is defined as an injury to the head from blunt trauma or accelerated/decelerated forces with one or more of the following associated conditions: transient confusion, disorientation or impaired consciousness; memory loss around the time of injury; and/or loss

Figure 3. Subdural Hematoma

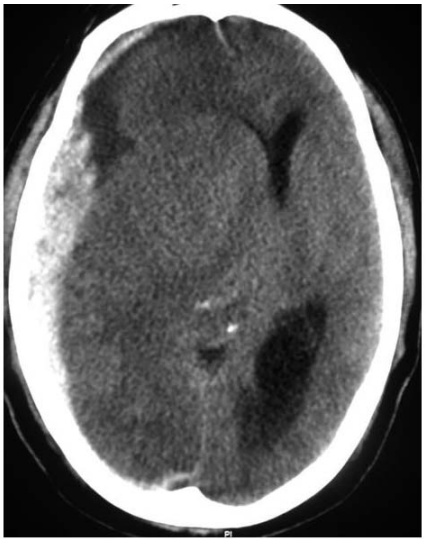


Figure 4. Epidural Hematoma

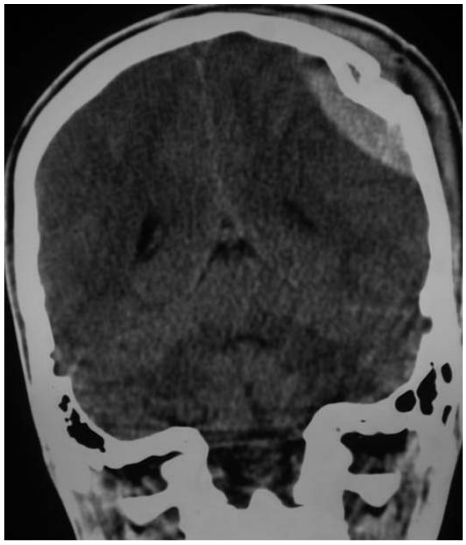


Figure 5. Subarachnoid Hemorrhage

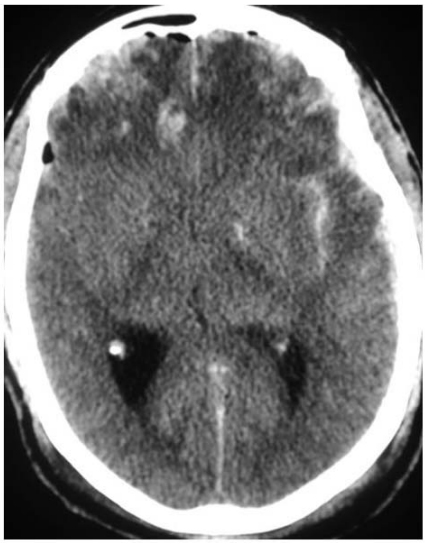
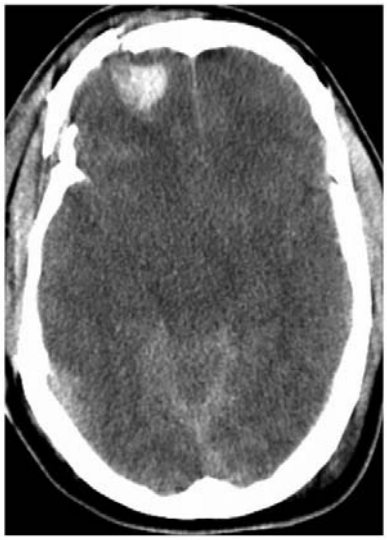


Figure 6. Intracerebral Contusion



of consciousness for less than 30 minutes.[24] Seventy-five percent of all patients with TBI have mTBI, at a cost of nearly $17 billion a year.[25,26] Although most patients with mTBI recover fully, up to 15% of patients may have persistent disabling problems.[27]

Moderate Traumatic Brain Injury. Moderate injuries account for approximately 10% of patients with TBI. Although not often studied, those with moderate TBI often have abnormalities on CT scan and frequently need surgical intervention. In 1982, Rimel and colleagues reported on a series of patients with moderate TBI as defined by GCS.[28] Thirty percent of patients who had a CT scan performed had a mass lesion, and more than 90% of those with a mass lesion had an operative intervention. In 1992, Stein and Ross also demonstrated an increased risk of intracranial lesions and need for operative intervention in a cohort of moderate TBI patients.[29] Thirty percent of their patients with moderate TBI had CT abnormalities; 20% required surgical intervention.

Severe Traumatic Brain Injury. Severe TBI accounts for the remaining 10% of brain injuries. The mortality is considerably higher and the functional recovery is considerably lower in those with severe TBI as compared to those with mild and moderate TBI. The primary management goals for patients with severe TBI center on identifying mass lesions, treating other life-threatening injuries, and preventing secondary brain insult. The remainder of this article will predominantly focus on those with severe traumatic brain injury.

Pre-hospital Care

The pre-hospital management of TBI is critical to maximizing good outcome and the care provided by EMS affects the outcome of a patient with TBI.[30]

Establishing a definitive airway, if necessary; cervical spine immobilization; intravenous access; and controlling hemorrhage

Table 1. Glasgow Coma Scale

SCORE	EYE OPENING	MOTOR	VERBAL
6	—	Follows commands	—
5	—	Localizes (cross midline)	Alert and oriented
4	Spontaneous	Withdraws	Disoriented conversation
3	To voice	Flexes arms (decorticate)	Nonsensical speech
2	To pain	Extends arms (decerebrate)	Moan/unintelligible speech
1	No response	No response	No response

Table 2. Risk Factors for Post-traumatic Seizures

- Glasgow coma score < 10
- Cortical contusion
- Depressed skull fracture
- Subdural hematoma
- Epidural hematoma
- Intracerebral hematoma
- Penetrating head wound
- Seizure within 24 hours of injury

are primary EMS goals. Once a possible TBI is identified, EMS must triage the patient to a facility capable of definitive care. In a large, prospective study, Härtl and colleagues demonstrated a 50% increase in mortality for patients with TBI who were not transported directly to a center that could provide definitive care.[31] Helicopters have been shown to rapidly transport patients over long distances, allowing for earlier definitive care.[32] Davis and colleagues demonstrated that aeromedical response teams appear to improve outcomes in patients with moderate to severe TBI.[33]

Hypoxia at any time will also increase mortality.[15,34] In 2007, The Brain Trauma Foundation (BTF) recommended that any patient with severe TBI who is unable to maintain an adequate airway or pulse oximeter of greater than 90% despite supplemental oxygen should have an airway secured.[35] However, there is very little objective data supporting prehospital airway management. In fact, many studies suggest that outcomes are worse in TBI patients who are intubated prehospital.[36-40] Longer scene times, hyperventilation after intubation, or persistent hypoxia during failed intubations have all been suggested as explanations for worse outcomes. Some authors have suggested a survival benefit from prehospital intubation when performed by more experienced providers, including physicians and flight nurses.[33,41,42]

During initial evaluation and transport by EMS, it is imperative to avoid hypotension. However, the ideal intravenous fluid has not been determined.[43-45] Isotonic fluids are used most commonly. Solutions containing glucose can worsen cerebral edema, and thus should not be used. Hypertonic saline expands intravascular volume, thus stabilizing blood pressure. Smaller volumes of hypertonic saline can be used, thus minimizing potential complications such as extravascular fluid accumulation, cerebral edema, and increased ICP. Although some studies have demonstrated a survival benefit from hypertonic saline in patients with severe TBI in the pre-hospital phase, long-term neurological benefit has yet to be established.[43-45]

ED Evaluation and Management

Upon the patient's arrival at the ED, multiple simultaneous events occur. Beginning with airway, breathing, and circulation, the emergency physician needs to rapidly assess for other life-threatening injuries. For those in a small hospital setting without immediate neurosurgical consultation, steps to facilitate transfer to a tertiary care center should begin early. In addition, the past medical history of the patient, such as anti-platelet or anticoagulation therapy, may elucidate additional risk factors for TBI or other injuries.

Airway. In patients with TBI, hypoxia worsens outcome. Thus, early intubation in the ED seems wise. Those with a GCS of < 9 warrant endotracheal intubation for definitive airway protection. All trauma patients should be considered to have a cervical spine injury until proven otherwise. Thus, cervical spine precautions need to be used during intubation.

During laryngeal manipulation and tracheal intubation, increases in blood pressure and heart rate may cause an increase in ICP. Lidocaine is believed to blunt these increases in ICP during intubation. In addition, lidocaine is thought to reduce cerebral blood flow, cerebral vascular resistance, cerebral metabolism as well as catecholamine release, minimizing ICP elevation during intubation.[46-48] However, a 2001 systematic literature review by Robinson and Clancy failed to demonstrate a reduction in ICP or improved neurological outcome when using lidocaine as a pretreatment during rapid sequence intubation in patients with TBI.[49]

Controversy also exists as to whether lidocaine usage worsens outcome. In a 1990 study by Asfar and Abdulla, the addition of lidocaine to thiopental and succinylcholine caused a significantly lowered blood pressure compared to those who did not receive lidocaine.[50] Others, however, have demonstrated no hemodynamic effects of lidocaine.[46-49] Some authors believe this drop in blood pressure from the lidocaine is related to the co-administration of the thiopental, an agent known to drop mean arterial blood pressure.[51] Lidocaine's ability to blunt the sympathetic response with the use of thiopental may explain the significant drop in blood pressure.[51]

Other pretreatment medications include fentanyl, which not only provides analgesia but is usually not hemodynamically compromising. However, in patients who are not adequately resuscitated, fentanyl may cause hypotension. In addition to fentanyl, defasciculating doses of a non-depolarizing neuromuscular blocking agent, such as rocuronium (0.06 mg/kg), vecuronium (0.01 mg/k), or even pancuronium (0.01 mg/kg) can be used.

Multiple induction agents can be used during intubation. The ideal agent should both blunt increases in ICP and lower mean arterial blood pressure. Etomidate (0.3 mg/kg) has minimal cardiovascular effects and is an ideal choice. However, recent controversy has suggested that etomidate may be associated with adre-

nal suppression.[52,53] A short-acting barbiturate, such as thiopental (3–5 mg/kg) is an alternative choice. If a patient presents hypotensive, a lower dose of thiopental (0.5–1 mg/kg) is recommended. Agents such as ketamine are traditionally not recommended, as they can increase ICP.

Breathing. Traditional management of patients with TBI once included hyperventilation to an arterial $PaCO_2$ < 25 mmHg. Hyperventilation causes cerebral vasoconstriction, thus lowering cerebral blood flow (CBF) and ICP.[54] A 1995 survey indicated that 83% of trauma centers were using hyperventilation as a tool to lower ICP.[55] However, after TBI, patients have 50% less CBF than normal people, and thus, lowering cerebral blood even further can cause cerebral ischemia. Multiple studies have demonstrated that prophylactic hyperventilation increases cerebral ischemia with a resultant worsening of outcome in patients with TBI.[56,57] Currently, the BTF does not recommend prophylactic hyperventilation and further suggest it be used only as a temporizing measure for the reduction of ICP.[58] Currently, common practice is to maintain an arterial $PaCO_2$ at 35 mmHg to 40 mmHg.

Circulation. Delayed resuscitation may improve outcomes in the patient with penetrating trauma.[59] However, this may not be wise after TBI as a single episode of systolic blood pressure below 90 mmHg doubles mortality.[15,60] No literature to date supports an ideal fluid in the ED for the resuscitation of a patient with TBI. In 2007, a post hoc analysis of the SAFE trial demonstrated the use of albumin in patients with TBI was associated with higher mortality rates.[61] The authors suggest that saline is preferable to albumin in the resuscitation of the patient with TBI.

Hypertonic saline provides prompt restoration of volume, and also increases serum osmolarity and decreases cerebral edema, thus lowering ICP.[62,63] As a result, osmotherapy has become an important tool in the management of elevated ICP after TBI.[64] However, there is very limited human data supporting outcome benefits from its use. There are also some side effects with the use of hypertonic saline, including renal failure, osmotic demyelinating syndrome, and rebound increases in ICP.

Mannitol provides an additional option for patients with TBI and elevated ICP. Mannitol is a plasma expander that reduces the hematocrit and subsequent blood viscosity, resulting in increased cerebral blood flow and cerebral oxygen delivery.[65-68] Mannitol also causes a delayed (15–30 minutes), pure osmotic diuretic effect.[65] This can cause hypotension, and so the patient's volume status must be carefully monitored. Currently, the BTF recommends mannitol (0.25 g/kg to 1 g/kg) for the control of elevated ICP. In addition, the panel recommends its use prior to ICP monitoring in patients with signs of transtentorial herniation or neurological deterioration not attributed to extracranial causes.[69]

Neurologic Assessment

It is imperative to obtain an accurate neurologic exam in every trauma patient with suspected TBI. One should attempt an accurate GCS prior to intubation to better communicate with neurosurgical consultants the patient's presenting condition. Hypothermia, hypoglycemia, drugs administered during pre-hospital transport, and other factors can affect GCS.

In addition to GCS, pupil size, reactivity, and anisocoria, as well as motor and brainstem function, must be assessed. Patients with fixed and dilated pupils have a high likelihood of a surgically amenable lesion, usually an ipsilateral hematoma with uncal herniation requiring emergent evacuation. However, previous eye surgery (e.g., cataract), or ocular trauma may also cause pupil dilation. In addition, hypoxia or hypotension should be corrected, as they can also cause pupil abnormalities.

Diagnostic Imaging

CT scanning rapidly identifies life-threatening, space-occupying lesions in need of emergent evacuation. However, rising healthcare costs and concerns for unnecessary exposure to radiation have raised questions about who needs head CTs. Haydel and colleagues evaluated patients with GCS 15 and a history of loss of consciousness or amnesia and concluded that any patient with headache, vomiting, age greater that 60, intoxication, deficit in short-term memory, physical evidence of trauma above the clavicles, or seizure (New Orleans Criteria) warranted a head CT.[70] Absence of all seven predictors had 100% negative predictive value. In 2001, Stiell and colleagues developed a clinical decision rule for those with mTBI that included five high-risk factors: failure to reach GCS of 15 within two hours; suspected open skull fracture; any sign of basal skull fracture; more than two episodes of vomiting; or age greater than 65 years.[71] Two additional medium-risk factors (amnesia before impact > 30 minutes, and dangerous mechanism of injury) were also included. The high-risk factors were associated with 100% sensitivity for predicting the need for surgical intervention and would eliminate 68% of CTs performed. The medium risk factors had a 98.4% sensitivity and 49.6% specificity for predicting clinically important brain injury and would eliminate the need for 46% of CTs. In 2005, the NEXUS II investigators identified eight variables (evidence of skull fracture, scalp hematoma, neurologic deficit, altered level of alertness, abnormal behavior, coagulopathy, persistent vomiting, and age 65 years or older) that were independently and highly associated with intracranial injuries.[72] The decision instrument had a sensitivity of 98.3% and negative predictive value of 99.1%.

Based on a comprehensive literature review, in 2008 the American College of Emergency Physicians' clinical policy on neuroimaging in patients with mTBI recommended that a head CT be obtained in patients with either loss of consciousness or posttraumatic amnesia with one or more of the following: headache, vomiting, age greater than 60 years, drug or alcohol intoxication, deficit in short-term memory, physical evidence of trauma above the clavicles, posttraumatic seizure, GCS less than 15, focal neurologic deficit, or coagulopathy.[73] The committee recommended a head CT in patients with no loss of consciousness or posttraumatic amnesia if there is a focal neurologic deficit, vomiting, severe headache, age ≥ 65 years, sign of a basilar skull fracture, GCS less than 15, coagulopathy, or dangerous mechanism of injury.

Patients with moderate and severe TBI, specifically severe TBI, should have a non-contrast CT scan of the head performed as early as possible. Patients should be adequately resuscitated and life-threatening injuries (e.g., respiratory compromise, active

hemorrhage, tension pneumothorax, open-book pelvis) should be addressed prior to obtaining the CT scan.

Magnetic resonance imaging (MRI) has no role in the initial evaluation of patients with suspected TBI. CT is a superior imaging modality for the evaluation of acute hemorrhage as well as bony abnormalities of the skull associated with TBI. The length of time it takes to obtain an MRI and distance from the ED in many institutions makes it unsuitable during the initial evaluation.

ICP Monitoring: Indications, Methods, and Treatment Threshold

Many patients with moderate or severe head injuries will require ICP monitors after TBI. Although placement of these monitors is beyond the scope of most emergency physicians, knowing the indications allows for more sophisticated communication with consultants or transfer facilities. In addition, longer stays in the ED now require emergency physicians to begin treatment of elevated ICPs.

Current recommendations for ICP monitoring include any patient with severe TBI that has a CT abnormality, including hematoma, contusion, edema, herniation, or compressed basal cistern;[74] and any patient with severe TBI who has a normal CT scan and at least two of the following: age greater than 40 years, unilateral or bilateral posturing, or systolic blood pressure less than 90 mmHg on arrival.

There are a number of monitoring devices available for measuring ICP. The two most commonly used are an intraventricular catheter (IVC) and a parenchymal monitor ("Bolt"). An IVC not only measures ICP, but also provides cerebrospinal fluid drainage to help control elevated ICP. However, the placement of an IVC is a more invasive procedure than the placement of a parenchymal monitor, and complications are more common.

Currently there are no large, randomized trials that determine the optimal ICP threshold. The largest prospective study involved 428 patients and determined that the incidence of morbidity and mortality was strongly related to ICP control when 20 mmHg was used as the treatment threshold.[75] Other studies have suggested 15–25 mmHg represents the optimal treatment threshold.[76-78] The BTF currently recommends beginning treatment of ICP if they are above 20 mmHg[79] with the goal of maintaining CPP between 50 mmHg and 70 mmHg.[80] Furthermore, the BTF recognizes clinical worsening or CT scan findings, such as acute pupillary abnormalities or herniation respectively, can occur with ICPs below the recommended treatment threshold of 20 mmHg and warrant treatment regardless.[79]

Additional Medical Management Therapies

The primary goal of medical management is to control ICP while maintaining CPP, and to ensure adequate oxygenation and hemodynamic stability to minimize secondary insult. While many patients have elevated ICP due to the primary injury, other factors, including agitation, pain, seizure activity, fever, and patient positioning, may also affect ICP.

Sedation. Propofol has become a popular sedative for patients with TBI. Its rapid onset and short half-life allow clinicians to obtain reliable neurologic exams to determine any change in mental status. Propofol decreases cerebral metabolism and oxygen demand, and thus is believed to be neuroprotective. However, little data has demonstrated its benefit in lowering ICP. In addition, complications, including propofol infusion syndrome (PIS), hyperkalemia, metabolic acidosis, and rhabdomyolysis, can occur. Although first identified in children, PIS can occur in adults, as well. Caution must be taken when high doses (> 5 mg/kg/hr) are used for an extended period of time (> 48 hours).[81,82] If not recognized, PIS can lead to cardiovascular collapse and death.

Anticonvulsants. Post traumatic seizures (PTS) have been classified as *early* (within the first seven days after injury) and *late* (after seven days post-injury).[83] PTS increases metabolic demand and ICP, and thus worsens injury; however, early PTS has not been shown to affect outcome. Risk factors have been identified that increase the risk of seizures after TBI. *(See Table 2.)* Early studies suggest that prophylactic phenytoin decreased the incidence of PTS. A 1990 study by Temkin and colleagues confirmed phenytoin's ability to decrease early PTS, but it did not prevent late PTS.[84] Valproate therapy has not been shown to be more effective that phenytoin in preventing early PTS. Nor has it been shown to prevent late PTS more effectively either. Due to a trend towards higher mortality with the use of valproate, its use for preventing PTS over phenytoin cannot be recommended.[85] More recently, levetiracetam has been shown to be as effective as phenytoin in preventing early PTS.[86] Compared to phenytoin, levetiracetam has fewer pharmacokinetic interactions and does not require drug level monitoring.

No studies have demonstrated that prophylactic anticonvulsant therapy will decrease the incidence of late PTS. Currently, anticonvulsant therapy is indicated for the first seven days following injury. In addition, patients who have late PTS should be treated according to standard protocols for those with new-onset seizure.[87]

Positioning. It is common practice to elevate the head above the heart in patients with TBI. Multiple studies have demonstrated lowering of ICP with head elevation.[88-90] Clinicians must rule out lumbar and thoracic spine injuries as well as pelvic fracture prior to raising a patient's head. In the interim, reverse Trendelenburg can be used to elevate the head.

Steroids. Steroids are commonly used to prevent cerebral edema in patients with various types of brain tumors. In the early 1970s, steroids were commonly used in patients with TBI. Early studies demonstrated no significant difference in outcome with use of steroids in patients with TBI.[91,92] However, a 2004 study was terminated early, after more than 10,000 patients were enrolled, because of a significantly increased risk of death in patients treated with steroids after TBI.[93] Thus, based on the current existing literature, steroids should not be used in the setting of TBI.

The Elderly TBI Patient

By the year 2030, the number of persons older than age 65 will double relative to 2000, representing almost 20% of the nation's total population.[4] In persons 65 years and older, TBI is responsible for more than 80,000 ED visits annually.[3] In 2006, more than

$2.8 billion was spent on treating TBI in those older patients.[5] Falls are the leading cause (51%) of TBI in elderly patients, while MVCs are a distant second (9%).[3]

As many as 73% of elderly TBI patients may have at least one co-morbid condition, compared to only 29% of younger patients.[94] Treatment of some of these chronic conditions includes the use of aspirin, clopidogrel, and warfarin, increasing the risk of TBI in the elderly. In a study by Ohm and colleagues, those patients taking either aspirin or clopidogrel or both had two and half times higher mortality rates than those not on antiplatelet therapy.[95] A study by Lavoie et al found that 9% of the older patients with TBI were taking warfarin pre-injury, and it was associated with more severe TBI and a higher rate of mortality.[96] TBI patients on warfarin have demonstrated a five times higher mortality than those similarly injured who were not on warfarin.[97] Others, however, have shown that pre-injury warfarin use in the elderly had no effect on mortality in trauma patients.[98,99]

For patients taking aspirin or clopidogrel, limited options exist to reverse the antiplatelet effects. Aspirin irreversibly inhibits platelet activity for the lifetime of the platelet. However, as aspirin has a relatively short half-life, transfusion of new platelets should be minimally affected by the patient's aspirin consumption. On the other hand, clopidogrel has a long half-life and will affect newly transfused platelets up to seven days after the last dose. Consultation with a neurosurgeon as well as hematology is required for optimal treatment of patients with active bleeds on the CT scan and those going to the operating room

Patients taking warfarin require immediate reversal. Warfarin, a vitamin k antagonist, inactivates clotting factors II, VII, IX, and X. Fresh-frozen plasma (FFP) has traditionally been used to restore these factors. Using a reversal protocol of 2 units of uncrossed FFP and 10 mg of intravenous vitamin K followed by an additional 2 units of cross-matched FFP, Ivascu et al demonstrated decreased time for reversal initiation from 4.3 hours to 1.9 hours, with a subsequent improvement in mortality from 48% to 10%.[100] However, many EDs do not have readily available, prethawed FFP, making rapid reversal difficult. In addition, large volumes of FFP are often required to fully reverse warfarin. In the elderly, this risks pulmonary edema and volume overload.

Alternatives to FFP do exist. Recombinant factor VIIa (rFVIIa) rapidly and effectively treats mild to moderate coagulopathy following injury.[101,102] In addition, rFVIIa decreases the time to neurosurgical intervention (144 vs. 446 minutes) and decreases the use of blood products without increasing the rate of thromboembolic complications.[103] While the initial cost of rFVIIa is high, its use in severely injured patients with TBI can significantly decrease their total charges and costs of hospitalization.[104] Although not considered standard of care, rFVIIa offers a rapid and cost-effective treatment option for patients with TBI and coagulopathy.

Other alternatives include vitamin K, prothrombin complex concentrates (PCC), and cryoprecipitate. The administration of vitamin K may not be helpful in the initial immediate reversal; however, it may help lessen the rebound effect when the FFP or rFVIIa wears off.[100] Limited evidence suggests that intravenous and oral administration of vitamin K are equivalent for warfarin reversal while subcutaneous administration is inferior.[105] PCCs are human plasma derived and undergo viral inactivation. They contain vitamin-K-dependent coagulation factors II, VII, IX, and X. Recent evidence suggests that PCCs may be an effective alternative to FFP for the reversal of warfarin anticoagulation in the acute setting.[106] Currently, PCCs are not commercially available in the United States.

Elderly trauma patients on warfarin with mild TBI and no radiographic evidence of intracranial pathology may be susceptible to delayed hemorrhage and clinical deterioration. Some authors suggest that an initial screening CT of the head as well as interval follow up imaging is unnecessary in this patient population, while others recommend admission and observation for a minimum of 24– 48 hours.[107-109] When clinicians elect not to image or admit patients for close monitoring, they must ensure a strong and reliable social support with 24-hour observation at home. A repeat CT scan should be done for any change in the patient's neurologic exam.

Hypothermia

Hypothermia is the most powerful neuroprotective method in animal models of TBI.[110] Therapeutic hypothermia was common practice in many medical centers in the 1960s and early 1970s however by the late 1970s fell out of favor because of increase rates of infection, especially pneumonia as well as a lack of randomized controlled trials in humans demonstrating efficacy. More recently, a renewed interest in hypothermia for the treatment of severe TBI has emerged. Although studies have demonstrated mild hypothermia's ability to decrease ICP and improve outcome, there is no data demonstrating clear outcome benefit.[111-113]

Serum Markers

Many serum biomarkers have been studied to help predict outcome in patients with TBI. The best studied is S100B. Multiple studies have shown that elevated levels of S100B after TBI are predictive of poor outcome.[114,115] However, a lack of a well-defined cutoff value, as well as the inability in many institutions to obtain timely laboratory results, has precluded widespread use of 100SB. Other markers including neuron-specific enolase and glial fibrillary acidic protein have been studied as possible predictors of outcome after TBI. However, further studies for all biomarkers are needed to help better identify and predict outcome after TBI.

Conclusion

TBI continues to be a leading cause of mortality in the United States. The emergency medicine physician's primary management goals for patients with severe TBI must center on identifying mass lesions, treating life-threatening injury, and preventing secondary brain insult. It is imperative that hypoxia and hypotension be minimized in patients with TBI, as a single episode of either is associated with a statistically significant increase in both morbidity and mortality.

Additionally, as the population ages, so will the number of elderly patients with TBI. Many of these patients are taking either

antiplatelet or anticoagulation medications, or both. Rapid identification of these patients is essential to attempt to reverse the effects of the medications and prevent clinical deterioration. Platelet and FFP transfusions are the traditional treatment options. Alternatives include vitamin K, rFVIIa, and PCC. However, further studies are needed to ensure efficacy and safety.

Finally, research into the benefits of hypothermia in brain injury, as well as serum markers, including S100B and NSE, in predicting outcome after TBI currently are underway.

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CNE/CME Objectives

Upon completing this program, the participants will be able to:

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

CME / CNE Instructions

Physicians and nurses participate in this CME/CNE program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided to receive a letter of credit.** When your evaluation is received, a letter of credit will be mailed to you.

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CME / CNE Questions

1. Which of the following is an example/are examples of secondary insult?
   A. Hypoxia
   B. Epidural hematoma
   C. Hypotension
   D. Both A and C
   E. All of the above

2. Cerebral perfusion pressure is equal to:
   A. Mean arterial pressure + intracranial pressure
   B. Mean arterial pressure - intracranial pressure
   C. Cerebral blood flow + intracranial pressure
   D. Cerebral blood flow - intracranial pressure
   E. Mean arterial pressure + cerebral blood flow

3. A single episode of hypotension in patients with traumatic brain injury has been shown to:
   A. have no effect on mortality.
   B. double mortality.
   C. triple mortality.
   D. decrease mortality by 10%.
   E. decrease mortality by 25%.

4. The "lucid interval" has classically been associated with:
   A. subdural hematoma.
   B. subarachnoid hemorrhage.
   C. diffuse axonal injury.
   D. epidural hematoma.
   E. None of the above

5. Many studies suggest pre-hospital intubation worsens outcome in patients with traumatic brain injury because of:
   A. longer scene times.
   B. hyperventilation by prehospital providers.
   C. hypoxia due to failed intubations.
   D. All of the above
   E. None of the above

6. Which of the following is *not* associated with lidocaine administration during rapid sequence intubation in patients with traumatic brain injury?
   A. Reduced cerebral blood flow
   B. Reduced cerebral vascular resistance
   C. Reduced cerebral metabolism
   D. Minimized ICP elevation
   E. Increased catecholamine response

7. A single dose of etomidate during rapid sequence intubation has been associated with:
   A. Neutropenia
   B. Thrombocytosis
   C. Adrenal suppression
   D. Thyrotoxicosis
   E. All of the above

8. According to the NEXUS II study, which of the following variables has/have been independently and highly associated with intracranial injuries?
   A. Scalp hematoma
   B. Intermittent vomiting
   C. Age 65 years or older
   D. A and B
   E. A and C

9. All of the following are associated with propofol infusion syndrome *except*:
   A. Hyperkalemia
   B. Metabolic acidosis
   C. Rhabdomyolysis
   D. High-dose infusion
   E. None of the above

10. Which of the following is *not* currently commercially available in the United States for the reversal of warfarin in patients with traumatic brain injury?
   A. Vitamin K
   B. rFVIIa
   C. Fresh-frozen plasma
   D. Prothrombin complex concentrates (PCC)
   E. None of the above

Answers: 1. D; 2. B; 3. B; 4. D; 5. D; 6. E; 7. C; 8. E; 9. E; 10. D

In Future Issues: **Eye Trauma**