Eye Trauma

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**Orbital Fractures** – see [p. TrH27 >>](http://www.neurosurgeryresident.net/TrH.%20Head%20trauma%5CTrH27.%20Facial%20Trauma%20%28frontal%2C%20orbital%29.pdf)

Document legally (in any patient with upper facial trauma): vision, range of extraocular motion, location of lid and conjunctival lacerations and foreign bodies, depth of anterior chamber, anterior chamber / vitreous hemorrhage, cataract.

Ocular trauma accounts for 8-10% of all visual impairments!

**Prehospital management** – see [p. TrH25 >>](http://www.neurosurgeryresident.net/TrH.%20Head%20trauma%5CTrH25.%20Facial%20Trauma%20%28GENERAL%29.pdf)

Hyphema

*- blood accumulation in anterior chamber.*



Classification

**Traumatic hyphema** (even small hyphema can be sign of major intraocular trauma!)

Grade 1 - occupying < 1/3 of anterior chamber

##### Grade 2 - filling 1/3-1/2 of anterior chamber

##### Grade 3 - filling > 1/2 of anterior chamber

Grade 4 - total filling.

**Spontaneous hyphema** - secondary to neovascularization, ocular neoplasms, vascular anomalies.

Clinical features

* < 50% hyphemas settle inferiorly to form **level**; 40% form definite **clot** **adherent to iris stroma**; 10% have dark **clot in contact with endothelium** (poor outcome and corneal staining).
* tear at *anterior aspect of ciliary body* is most common site of bleeding (71%).
* usual duration of uncomplicated hyphema is 5-6 days; mean duration of elevated IOP is 6 days.

Complications

#### **Elevated IOP** - may accompany hyphemas of any size (esp. with near total or total hyphemas); periods:

1. hypertonia - during acute phase of hyphema (first 24 hours after injury) - ***trabecular plugging by erythrocytes and fibrin***.
2. hypotonia (≤ normal IOP) from 2nd to 6th day - due to ***reduced aqueous production and uveitis***.
3. hypertonia - recovery of ciliary body.
4. normotonia - recovery of trabecular meshwork (disappearance of hyphema)

Glaucoma may result:

* 1. if large segments of anterior chamber angle are irreparably damaged and/or clot organization produces extensive peripheral anterior synechiae → **intractable glaucoma**.
	2. erythrocytes lose hemoglobin and become ghost cells in vitreous cavity → circulate forward into anterior chamber with resultant trabecular blockage → delayed **ghost cell glaucoma**.

#### **Secondary hemorrhage** into anterior chamber (≈ 25%) usually in first 3 days.

* due to clot lysis and retraction.
* markedly worsens prognosis.

#### **Posterior synechiae**

* secondary to iritis or iridocyclitis.

#### **Peripheral anterior synechiae**

* occurs if hyphema has remained in anterior chamber for prolonged period (> 9 days).

#### **Corneal bloodstaining**

* more likely in total hyphema that remains for at least 6 days with IOP > 25 mmHg.
* clearing of corneal bloodstains may require many months.

#### **Optic atrophy**

* due to ↑ IOP.
* for black patients (with sickle cell trait), prevention of secondary hemorrhage is critical factor!

Treatment

1. **patching** (bilateral or injured eye only).
2. **bed rest**, elevating bed head 30-45° (→ hyphema settling in inferior anterior chamber).
3. **sedation** in extremely apprehensive individual.
4. If **analgesics** are required, avoid aspirin and other NSAIDs with antiplatelet effect.
5. Topical medications:
6. aminocaproic acid(some administer orally)- prevention of recurrent hemorrhages; clot will persist in anterior chamber for increased period – so avoid in grade 4 hyphema.
7. **antiglaucomatous** medications - initiate therapy incrementally with brimonidine, followed by latanoprost and timolol; if IOP is still elevated, add *carbonic anhydrase inhibitor*.
8. **steroids** (after 3-4th day of retained hyphema) - to decrease iridocyclitis and to prevent synechiae.
9. **atropine** (indicated in grade 3-4 hyphemas) - to break pupillary block.

Any other *topical medications* lack definite evidence of advantages!!!

1. Surgical evacuation – indicated in:
2. ***grade 3-4 hyphemas*** persisting for > 4 days.
3. ***microscopic corneal bloodstaining*** (at any time); most typical early sign of corneal bloodstaining is tiny yellowish granules in posterior third of corneal stroma - surgical treatment in this early stage may prevent gross staining, and cornea may clear in 4-6 months.
4. ***IOP ≥ 50 mmHg*** for 4 days (or ≥ 35 mmHg for more than 24 hours if sickle cell trait or disease is present!)
* preferred technique is **evacuation with closed** **vitrectomy instrumentation**.
* other methodologies – paracentesis, irrigation & aspiration through small incision, clot irrigation with trabeculectomy.
* extreme care is required to *avoid any contact with iris, lens, or corneal endothelium*.

Foreign Bodies

Typical patient - 20-40 yrs male who does not wear protective eye gear.

* most injuries occur at work using various tools with ***metal striking metal*** – patient feels something enter eye with no obvious external changes; hence, incident usually is dismissed quickly.

Avoid pressure on globe!

**Conjunctival, Corneal foreign bodies**

1. Apply ***anesthetic*** and ***fluorescein staining***
2. Evert individually both lids.
3. Inspect with binocular lens (loupe) or slit lamp.
4. Remove foreign body:

**Conjunctival** → ***moist sterile cotton applicator***.

**Corneal** → ***irrigation***;

if cannot be dislodged → lift out on point of ***sterile spud / hypodermic needle*** under slit-lamp magnification.

N.B. unless **steel / iron foreign bodies** are removed immediately, they leave *rust ring on cornea* (also requires removal under slit-lamp magnification):



[Source of picture: “Online Journal of Ophthalmology” >>](http://www.atlasophthalmology.com/atlas/frontpage.jsf?locale=en)

Burr removal of metallic rust ring:



1. **Antibiotic ointment** (bacitracin/polymyxin B or sulfacetamide sodium 10%).
	* for larger foreign bodies, treatment is as for corneal abrasion (short-acting cycloplegic + antibiotic + firm patch to keep eye closed overnight).
	* corneal epithelium regenerates within 1-3 days.

N.B. *corticosteroids are* *contraindicated* (promote growth of fungi and herpes simplex virus)!!!

**Intraocular foreign bodies**

Diagnosis

Slit lamp examination:

* **entry sites**:

in cornea - disruption in smooth surface with corneal edema surrounding perforation site.

in sclera - area of conjunctival injection; darker pigmentation indicates choroidal exposure.

* examine **iris** before dilatation (disruption point?) and **lens** after dilatation (cataract?).

Dilated fundus examination reveals foreign bodies in **posterior segment**.

Fine CT with 2-mm sections can localize foreign bodies as small as 0.7 mm.

X-ray - beneficial for ***metallic*** foreign bodies.

MRI - more effective in localizing ***nonmetallic*** foreign bodies.

*Do not use if cannot exclude metallic objects*!!!

Ultrasound is useful adjunct tool to determine if object is metallic.

Treatment

Require immediate surgical removal (delay of 24 hours increases endophthalmitis risk to 13.4%):

**Inert substances** - glass, stone, plastic (may be removed at later time after initial wound is closed).

**Metals** – oxidize (*copper / iron should be removed urgently* – can cause *chalcosis* / *siderosis* - toxic to retina!!!)

**Organic material** - ↑ risk of endophthalmitis.

* systemic and topical ***antimicrobials*** are indicated; tetanus prophylaxis.
* ***minimize pressure on globe*** even in cases of self-sealing wounds.
* surgical approach varies with object location.
* removal through original entry wound is not recommended.

Object in anterior chamber

* lens is intact - constrict pupil with miotics to reduce risk of lenticular injury.
* 20-gauge rare earth **magnet** may retrieve small metallic objects; nonmagnetic objects or large magnetic objects are managed best with **intraocular forceps**.
* damaged lens should be removed (usually via phacoemulsification); concurrent IOL insertion is not performed (because calculation of intraocular lens power may not be exact).

Object in posterior chamber

* to reduce risk of intraocular content extrusion, anterior chamber paracentesis is performed to soften eye.
	1. **external approach** - via sclerotomy and electromagnet.
	2. **internal approach** – via vitrectomy; very large objects sometimes are managed best through limbal incision.

Contusions & Lacerations

**Lid contusions (black eye)**

**ice packs** to inhibit swelling - during first 24 h;

**hot compresses** to aid absorption - next day.

**Lid lacerations** → repair with fine sutures in at least three layers (to prevent notching as healing progresses):

6-0 absorbable suture for deep layer (conjunctiva and tarsus) - knots tied into wound;

5-0 chromic sutures for middle layer (orbicularis oculi muscle);

6-0 nonabsorbable suture (e.g. silk) for skin.

Traumatized lids should never be opened forcibly - injury could be aggravated!

* patch eye.
* lacerations ***involving lid margin*** or ***loss of lid tissue*** should be dealt by ophthalmologist (if injury prevents tears from keeping cornea moist → artificial tears).
* lacerations ***near medial canthus*** - danger of violating lacrimal apparatus. *see below*

**Eyebrow lacerations**

* carefully explore wound - fracture may be palpated (that does not visualize on X-ray).
* minimum debridement - eyebrow is very difficult to reconstruct.
* debridement should be done *parallel to hair follicles* (*not* perpendicular to skin, as is general rule) – to minimize bald area in repaired eyebrow:



* any defect in muscle layers is approximated with deep sutures (to prevent functional defect or depressed scar).
* during wound closure, eyebrow *borders should be aligned first* to avoid visible step-off (for this reason eyebrow should not be shaved - would destroy landmarks for accurate closure).

**Eyebrow avulsion**

- can be replaced with hair-bearing tissue from postauricular area.

* keep graft < 1 cm in width to ensure its survival.
* graft pedicled on ante­rior branch of superficial temporal artery also may be used.

**Lacrimal canaliculus laceration**

* > 50% tear volume is normally evacuated through inferior canaliculus - when this pathway is interrupted it is important that it be repaired when possible.

*any laceration in medial third of lower lid - suspect injury to inferior canaliculus.*

* diagnosis (after hemostasis):
1. place probe (e.g. small nylon or polypropylene suture) through punctum into wound.
2. insuf­flate canaliculus with air and instill sterile saline into laceration → air will bubble through saline.
3. instill fluorescein in conjunctival sac - observe dye in wound.
* treatment: **Vier stainless steel rod** (with swaged-on black silk) is passed through punctum into laceration site and then into medial por­tion of canaliculus, to align cut ends; laceration is stabilized with small chromic sutures; free end of suture of rod is tied in place to help stabilize rod and is used to retrieve it for removal; rod is left in place for 4-6 weeks.

**Trauma to globe**

Perforating injury – sclera partially torn.

Penetrating injury – sclera complete rupture.

* corneal laceration *- irregular (“teardrop”) pupil* due to iris prolapse through cornea.
* globe laceration risks **sympathetic ophthalmia**.
* emergency treatment:
1. protective rigid shield
2. analgetics
3. cyclopentolate 1% + phenylephrine 2.5%
4. antimicrobials (systemic / local - drops only, since ointment could penetrate lacerated globe!)
* corticosteroids are *contraindicated* until wounds are closed surgically.

Severe windshield injury after 7 days - iris tissue has prolapsed through corneal wound, pupil is peaked toward prolapse, marked hyperemia of conjunctiva:



[Source of picture: “Online Journal of Ophthalmology” >>](http://www.atlasophthalmology.com/atlas/frontpage.jsf?locale=en)

Traumatic optic neuropathy - impact injury to optic nerve (without concomitant facial fracture or penetrating wound) → instantaneous (rarely delayed) *permanent visual loss*

* occurs in 0.5-5% closed head injuries.
* treatment - **high-dose steroids** (≈ acute spinal cord injury).

Burns

**Lid burns** - cleanse thoroughly with **sterile saline** → apply petrolatum gauze or **antimicrobial ointment** → **sterile pressure dressing** held by elastic bandage around head until surface has healed.

**Chemical burns of cornea / conjunctiva**

Symptoms & signs – pain, hyperemic edema, eyelid burns, corneal opacification & epithelial defects, anterior chamber reaction (hazy fluid), possibly ↑ IOP.

Treatment:

* 1. **immediate copious irrigation** with water, saline, or other bland fluid; for at least 30 min; holding eyelids open [e.g. Morgan lens]; repeatedly assess pH of inferior fornix (with *litmus paper*) until it becomes neutral (recheck after 5-10 min).
	2. **anesthetize** eye with 1 drop of proparacaine 0.5% (→ oral analgetics)
	3. asses extent of injury (incl. visual acuity, slit lamp)
	4. **antibiotic ointment**
	5. pressure **patching**, artificial tears.
	6. **steroids** are helpful.
* *chemical iritis* → long-acting cycloplegic (e.g. atropine 1%).
* corneal scarring, opacification is risk.

Iridodialysis, Cyclodialysis

**Iridodialysis** - disinsertion of iris from scleral spur; elevated IOP can result from damage to trabecular meshwork or from formation of peripheral anterior synechiae (PAS).



**Cyclodialysis** - disinsertion of ciliary body from scleral spur; increased uveoscleral outflow occurs initially resulting in hypotony (later, IOP elevation can result from closure of cyclodialysis cleft).

Clinically

- asymptomatic unless glaucoma develops.

Treatment

* **sunglasses**, **contact lenses** with artificial pupil.
* ***surgical correction*** if large iridodialysis and patient symptomatic.
* if ***glaucoma*** develops → treatment similar to primary open-angle glaucoma.

N.B. avoid miotics - may reopen cyclodialysis clefts, causing hypotony; strong mydriatics may close clefts, resulting in pressure spikes.

Commotio Retinae

Etiology

- ***contrecoup injury*** (blunt trauma to globe causes shock waves which travel posteriorly and lead to disruption of photoreceptors).

Clinically

- decreased vision or asymptomatic; history of recent ocular trauma.

Funduscopy

- confluent area of retinal whitening (visual acuity does not always correlate with degree of retinal whitening).

* whitening is intracellular edema and fragmentation of photoreceptor outer segments and intracellular edema of retinal pigment epithelium; no intercellular edema.
* when occurs in ***macula*** is called **Berlin edema**.
* retinal blood vessels are undisturbed in area of retinal whitening!



Treatment

No treatment is required - clears without therapy!

Bibliography for ch. “Ophthalmology” → follow this [link >>](http://www.neurosurgeryresident.net/Eye.%20Ophthalmology%5CEye.%20Bibliography.pdf)

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