IN THE NAME OF GOD
Lid Laceration
Conjunctival Hemorrhage

a) No therapy is necessary

b) Usually resolve in 7-12 days.
Subconjunctival Hemorrhage
Corneal Abrasion
Abrasions

Many small abrasions can be managed with antibiotic drops or ointment alone, if extensive needs patching.
2) Traumatic mydriasis
Iris Sphincter Rupture
3) Iridodialysis

Separation of the iris from ciliary body

a) Small iridodialysis requires no treatment

b) Large dialysis may causes polycorin and monocular diplopia necessitating early surgical repair.
Cyclodialysis

a) Separation of the ciliary body from scleral spur.

b) Can cause aqueous hyposecretion and chronic hypotony and macular edema.

c) Closure may be attempted by using Argon laser, diathermy, cryotherapy or direct suturing.
Hyphema

a) Microscopic

b) Macroscopic

Total hyphema (eight-ball) if no secondary complication prognosis is good.
Traumatic Hyphema

Result from injury to the vessels of the peripheral iris or ant. Ciliary body
A Rebleeding complication

1) Glaucoma
2) Optic atrophy
3) Corneal blood staining
Rebleeding

1) Occurs most frequently between 2 and 5 days after injury.

2) 50% develop elevated IOP
Fig. 9.15
Small hyphaema (blood in anterior chamber) — characteristic fluid level of the blood.

Fig. 9.16
Hyphaema filling more than half the anterior chamber.

Fig. 9.17
Hyphaema filling the entire anterior chamber complicated by secondary glaucoma.
Treatment

1) Protective shield
2) Moderate restriction of physical activity
3) Control IOP
4) Anti fibrinolytic agents reduces the incidence of rebleeding.
Anti fibrinolytic

a) Tranexamic acid

b) Aminocaproic acid (amicar)
   50mg/kg q4h x5 days up to 30g/day rebleeding from 20-33% → 7.1% (112 patients)

C) Oral corticosteroid (↓ rebleeding)
Surgery
Timing is controversial

1) Immediate surgery with earliest detection of corneal blood staining

2) Uncontrollable tension
1) Removal of the entire clot is neither necessary nor wise
2) Intraocular diathermy may also be employed
3) Clot removal with vitrectomy can be done
Conjunctival laceration

In general conjunctival laceration do not need to be sutured
Foreign bodies

1) Extraocular

2) Intraocular
If AC extension present it should be removed in a sterile operating room.
Iron foreign body

If embedded in the cornea for more than a few hours, an orange rust ring results.
Corneal Foreign Body
Intraocular foreign body
Fig. 9.7  Siderosis bulbi of right eye caused by retained iron particle in eye.

Fig. 9.8  Iron particle in vitreous.

Fig. 9.9  Siderosis bulbi of right eye. Iris colour changed to brown (same patient as in Fig. 9.7).

Fig. 9.10  Left eye normal (same patient as in Fig. 9.7).
Therapy

1) Removal of foreign body

2) Cycloplegic and antibiotic drop
Abrasions

Many small abrasions can be managed with antibiotic drops or ointment alone, if extensive needs patching.
Penetrating and perforating trauma
Tests in perforating eye trauma

1) CT scan  2) X ray  3) BUN and creatinine

2) HIV & HBS

3) MRI especially for organic foreign objects this should never be used in a metallic foreign object.
Corneal perforation

1) Put eye shield
2) Avoid administering topical medications
3) IV antibiotics such as tobramycin with clindamicin or vancomycin
4) Tetanus prophylaxis.
Figure 8.2  Two consequences of corneal ulceration:
(a) a corneal abscess has resulted from severe pseudomonas infection; (b) a small central ulcer is associated with a hypopyon. A level of pus is seen in the anterior chamber.
Soil – contaminated retained intraocular foreign bodies

Risk of bacillus endophthalmitis, this organism can destroy the eye within 24 hours.

IV and or intravitreal therapy should be started, usually clindamycin or vancomycin.
Globe rupture
Corneoscleral laceration

1) Restore the integrity of the globe
2) Restore vision
3) If NLP enucleation should not exceed than 14 days to incite sympathetic ophthalmia.
Corneoscleral laceration

Subconjunctival injection and intravitreal antibiotics such as vancomycin 1 mg and amikacine 200 ug may be used.
Corneal Laceration
Secondary repair
intraocular trauma

Removal of IOFB
Iris repair
Cataract extraction
Mechanical vitrectomy
IOL insertion
The primary goal of initial surgical repair of corneoscleral laceration is to restore the integrity of the globe.
The secondary goal, which may be accomplished at the time of the primary repair is to restore vision through repair of both external and internal damage to the eye.
Post up management

Prevention of infection:
IV antibiotics are usually continued for 3-5 days
Topical antibiotics are generally used for about 7 days.
Retained IOFB require attention the risk of bacillus endophthalmitis. The organism can destroy the eye within 24 hours.
Intravitreal antibiotics such as Vancomycin 1mg Amikacin 200 µg may be used after contaminated wounds involving the vitreous
Suppression of inflammation
Massive fibrinous response may respond well to a short course of systemic prednisone
Management of IOFB

Fe

Cu

Wood

Glass
If the prognosis for vision in the injured eye is hopeless and the patient is at the risk for sympathetic ophthalmia, enucleation must be considered.
Corneal and scleral repair

Cornea with 10.0 nylon suture

The scleral wound is closed with 9.0 nylon 8.0 silk suture.
Iv and or intravitreal therapy should be considered
The advantage of delaying enucleation for a few days far outweigh any advantage of primary enucleation. This delay, which should not exceed 14 days, thought necessary for an injured eye to incite sympathetic ophthalmia.
Primary enucleation should be used only in a devastating injury so severe that restoration of the anatomy is impossible.
The advantage of delaying enucleation for a few days far outweigh any advantage of primary enucleation. This delay, which should not exceed 14 days, is thought necessary for an injured eye to incite sympathetic ophthalmia.
Very posterior lacerations benefit from effective physiologic tamponade by orbital tissue and are best left alone.
Corneal suture that do not loosen spontaneously are generally left in place for at least 3 months.
Lens dislocation and cataract
Fig. 9.18
Iridodialysis — iris torn at the root.

Fig. 9.19
Dislocated lens.

Fig. 9.20
Commotio retinae (traumatic oedema of macula) with typical curved choroidal tear, temporal to macula.
Traumatic Cataract
Blow out fracture
Fig. 9.21
Right lower lid haematoma and oedema with subconjunctival haemorrhage.

Fig. 9.22
Multiple lacerations of lid and face by glass fragments in motor-car accident (without seat belt). Cornea was also lacerated.

Fig. 9.23
Right blow-out fracture with limited elevation of right eye (right pupil dilated with mydriatics).
Orbital Wall Fracture
Figure 10-107. Subconjunctival emphysema in a patient with a medial orbital wall fracture who blew his nose.
Vitreous Hemorrhage
Optic Disc Hemorrhage
Traumatic extraocular nerve palsy
Eye trauma
First life
then sight
Trauma is one of the most important causes of ocular morbidity in childhood.
Visual loss in children

a) Amblyopia

b) Strabismus

c) Trauma
Playing with other children
Sports
Fire works
BB gun
Injecting needles
Results
Total cases was 100.
Age
Less than 15 years
Mechanism of trauma:

23% glass 13% knife, 11% wood 8% stone, 45% others.
Traumatized eye

61% right, 39% left
Injured boys outnumber girls by a factor of 2.5 children 3-6 years old have the highest incidence compared with other age groups.

Right eye outnumbers left eye by a factor of 1.5.
Trauma
INTACS rings
Chemical injuries

1. Mild irritation
2. Complete destruction of the ocular surface epithelium and corneal opacification, loss of vision and rarely loss of the eye.
Offendings:

1. Solid
2. Liquid
3. Powder
4. Vapor
Materials:

1. Household alkaline agents eg: cleaners, amonia, detergents, disinfectants

2. Industry: chemicals, solvents, lye and acids
The most severe chemical injuries are caused by strong alkalis and acids.
Alkali Burns

Strong alkalis raise the PH of tissues and cause saponification of fatty acids in cell membranes and cellular disruption.
Alkali Burns

Once the epithelium is damaged, alkali solutions readily penetrate the corneal stroma and they rapidly destroy the collagen fibers.
Strong alkaline may also penetrate into the anterior chamber and produce tissue damage and intense inflammation.
Alkali Burns

The limbus contains the putative corneal epithelial stem cells, when these are damaged, the denuded surface of the cornea is often resurfaced by neighboring conjunctival epithelium and causes conjunctivalization of the cornea and vascularization, inflammation and persistent and recurrent epithelial defeats.
Alkali Burns
Intraocular chemical penetration usually causes cataract and glaucoma.
In the most severe cases, phthisis of the globe may occur.
Acid Burns

Acids denature and precipitate proteins in tissues they contact.
Acid Burns

Acidic solutions tend to cause less severe tissue damage than alkaline solutions.

Because of the buffering capacity of tissues, as well as the barrier to penetration formed by precipitated protein.
Therapy of chemical injuries to the eye

The most important step is immediate and copious irrigation of the ocular surface with water or normal saline solution. Topical anesthetic should be instilled.
Therapy

It should be continued until the PH of the conjunctival sac normalizes. The conjunctival PH can be checked easily with a urinary PH strip.
Therapy

Severe chemical injuries can be approached by performing a paracentesis of anterior chamber removing 0.1-0.2ml of aqueous humor and reforming the chamber with B.S.S to normalize the anterior chamber PH.
the next phase of therapy should be decreasing inflammation, controlling IOP, limiting keratolysis, and promoting reepithelialization of the cornea.
Corticosteroids are excellent inhibitors of PMN function, and intensive topical steroids administration is recommended for the first 2 weeks following chemical injuries.
The steroid should be markedly reduced after 2 weeks, because of the ability of steroids to inhibit wound healing and potentiate infection.
A deficiency of calcium in the plasma membrane of the PMNs inhibits their ability to degranulate, and both tetracycline and citric acid are potent chelators of extracellular calcium. Therefore, oral tetracycline has theoretical benefit for inhibiting PMN-induced collagenolysis.
Ascorbic acid is believed to promote collagen synthesis in the alkali-burned eye because ascorbic acid is required as a cofactor for this synthesis.
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It is recommended that the patient receive 2 grams of oral ascorbic acid (vitc) per day.
For epithelial healing

A bandage contact lens or tarsorrhaphy may be beneficial.
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For epithelial healing
Limbal transplants

From uninvolved fellow eyes of patients limbal stem cell transplantation may be performed as soon as 2 weeks after injury if no signs of corneal epithelialization have appeared by that time.
Acute primary angle-closure Glaucoma

Is a condition that occurs when top sizes rapidly as a result of relatively sudden blockage of trabecular meshwork by iris.
Signs

1. High IOP
2. Middilated, sluggish and often irregular pupil
3. Corneal epithelial edema
4. Shallow AC
Trauma

1. Penetration
2. Perforation
3. Laceration
Penetration

1. A penetrating wound passes into a structure
2. A perforating wound passes through a structure
Treatment

1. Antibiotic
2. Repair: while studies have not documented any disadvantage in delaying the repair of an open globe for up to 36 hours, intervention ideally should occur as soon as possible.
Treatment

Bacillus endophthalmitis can destroy the eye within 24 hours.
Endophthalmitis

The term is usually associated with an infectious endophthalmitis.
Signs and Symptoms
The most common signs of endophthalmitis are decreased vision, anterior chamber reaction (hypopyon) and vitritis.
Signs and symptoms

Pain is a variable symptom, patient may have endophthalmitis without significant pain.