## EYE EMERGENCIES

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1. An infant who was delivered at home and received no perinatal care is brought in 12 days post-delivery for evaluation of a purulent eye discharge and cough. Examination reveals diffuse conjunctival injection and normal pupillary reactivity. The most likely etiologic agent is:
   (a) Staph. aureus
   (b) Adenovirus
   (c) Chlamydia trachomatis
   (d) Neisseria gonorrhoea

2. A 20-yr.-old male presents with redness and irritation of his right eye with an associated discharge. Eye findings include diffuse conjunctival injection and a copious purulent discharge. Visual acuity and pupillary reactivity are normal. The most likely etiologic agent is:
   (a) Neisseria gonorrhoea
   (b) Herpes simplex
   (c) Adenovirus
   (d) Vernal conjunctivitis

3. The most appropriate therapy for a patient with conjunctivitis and corneal ulceration due to Neisseria gonorrhoea is:
   (a) Discharge to home with topical erythromycin or tetracycline ophthalmic ointment
   (b) Discharge to home with tobramycin ophthalmic ointment or drops
   (c) Hospital admission with administration of IM or IV ceftriaxone
   (d) Hospital admission with administration of IM or IV ceftriaxone plus topical erythromycin and oral erythromycin or tetracycline.

4. All of the following entities are associated with painless decrease (or loss) of vision except:
   (a) Central retinal artery occlusion
   (b) Acute iritis
   (c) Retinal detachment
   (d) Central retinal vein occlusion

5. Which of the following is associated with painless loss of peripheral vision?
   (a) Primary open angle closure glaucoma
   (b) Acute iritis
   (c) Retrobulbar neuritis
   (d) Eclipse burn
6. A patient presents with eye pain, slight blurring of vision and severe photophobia. Examination reveals a red eye with a ciliary flush, a constricted pupil and a clear cornea. Slit lamp exam reveals flare and cells in the anterior chamber. The most likely diagnosis is:
   (a) Acute angle closure glaucoma
   (b) Retrobulbar neuritis
   (c) Acute iritis
   (d) Primary open angle closure glaucoma

7. All of the following are appropriate in the treatment of acute traumatic iritis except:
   (a) A long-acting topical cycloplegic agent
   (b) Topical steroids (in consultation with an ophthalmologist)
   (c) Antibiotic ointment or drops
   (d) Ophthalmology referral

8. The mechanism of action by which timolol and acetazolamide work in treatment of acute narrow angle glaucoma is:
   (a) They pull the iris back from its anterior position, thereby opening the angle and allowing escape of aqueous humor.
   (b) They decrease the secretion of aqueous humor by the ciliary body.
   (c) They create an osmotic gradient, thereby decreasing the volume of fluid in the eye.
   (d) None of the above

9. A 77-yr.-old woman presents with a headache and sudden loss of vision in her left eye. Visual acuity is measured as bare light perception in this eye. The cornea is clear, pupillary reactivity is normal and the sedimentation rate is 60. The most appropriate therapy for this patient is:
   (a) Topical pilocarpine solution
   (b) A topical steroid agent
   (c) Ophthalmology referral for follow-up in 3 - 4 days
   (d) High-dose corticosteroids, preferably IV

10. Signs of a blowout fracture of the orbit may include all of the following except:
    (a) Infraorbital anesthesia
    (b) Pulsating exophthalmos
    (c) Subcutaneous orbital emphysema
    (d) Pain and diplopia on upward gaze
11. A patient presents with a laceration to his upper eyelid. On evaluation, you note a deep horizontal laceration toward the middle of the upper lid and associated ptosis. The globe itself does not appear to be injured. The most likely associated injury is damage to the:
   (a) Lacrimal canaliculi
   (b) Orbital septum
   (c) Levator muscle/tendon
   (d) Canthal tendon

12. Findings suspicious for and/or consistent with a ruptured globe include all of the following except:
   (a) Decreased visual acuity
   (b) Vitreous hemorrhage
   (c) Exophthalmos
   (d) A penetrating wound to the eyelid

13. The shortest acting cycloplegic agent is:
   (a) Cyclopentolate
   (b) Tropicamide
   (c) Homatropine
   (d) Atropine

14. The topical antibiotic agent associated with the development of skin sensitivity in 10 - 15% of patients is:
   (a) Sulfacetamide
   (b) Chloramphenicol
   (c) Erythromycin
   (d) Neomycin

15. Topical ophthalmic drops have color-coded caps to facilitate the identification of the various types of agents. The color cap associated with miotic agents is:
   (a) Green
   (b) Red
   (c) Yellow
   (d) White

16. The presence of a Marcus-Gunn pupil on exam indicates which of the following?
   (a) Infection
   (b) Optic nerve damage
   (c) Encephalitis
   (d) All of the above
17. A 70-year-old woman presents with obvious signs and symptoms of acute angle closure glaucoma. Her past medical history is significant for poorly controlled congestive heart failure. All of the following would be appropriate in the management of this patient except:

(a) Pilocarpine 2% solution
(b) Glycerol 50% solution
(c) Timolol 0.5% solution
(d) Acetazolamide

18. All of the following statements regarding periorbital cellulitis are accurate except:

(a) Children < 3 years old are most commonly affected.
(b) Patients present with erythema, warmth and swelling of one or both eyelids.
(c) Patients complain of pain with ocular movement and ophthalmoplegia may be present.
(d) Fever is not uncommon.

19. All of the following statements regarding the use of the Schiötz tonometer are accurate except:

(a) Its use is contraindicated in the presence of a ruptured globe.
(b) If a scale reading < 1 is obtained, the procedure should be repeated using more weight.
(c) A scale reading of 6 is consistent with a normal intraocular pressure.
(d) A scale reading of 10 is consistent with elevated intraocular pressure.

20. Which of the following conditions does not classically produce a decrease in visual acuity?

(a) Traumatic lens dislocation
(b) Retrobulbar hematoma
(c) Periorbital cellulitis
(d) Retrobulbar neuritis

21. A 25-year-old patient presents with a foreign body sensation in his left eye, photophobia and tearing. Evaluation reveals a visual acuity of 20/30, diffusely reddened eye, decreased corneal sensation and a dendritic lesion on fluorescein staining. Which of the following could produce rapid worsening of his condition and should not be prescribed by the emergency physician?

(a) A topical antibiotic
(b) A topical steroid
(c) A topical antiviral agent (in consultation with an ophthalmologist)
(d) A mydriatic agent
22. A patient who uses extended wear soft contact lenses presents with complaints of photophobia, eye pain, blurred vision and a purulent discharge. Exam reveals a corneal ulcer. The causative agent in this setting is most likely:
(a) Neisseria gonorrhea  
(b) Klebsiella  
(c) Adenovirus  
(d) Pseudomonas

23. Immediate ophthalmology consultation, hospital admission and treatment is mandatory for which of the following conditions?
(a) Orbital (postseptal) cellulitis  
(b) Herpes zoster ophthalmitis  
(c) Corneal ulcers  
(d) Acute angle closure glaucoma

24. Regarding chemical burns of the eye, which of the following statements is inaccurate?
(a) Alkali burns are usually worse than acid burns.  
(b) Alkali burns produce a liquefaction necrosis.  
(c) A neutralizing agent should be used to irrigate the eye.  
(d) Irrigation should be continued until the pH of the conjunctival fornix is neutral.

25. The most appropriate management sequence for an acid or alkali burn of the eye is:
(a) Detailed eye exam, assessment of visual acuity, copious irrigation, pH assessment  
(b) Evaluation of visual acuity, pH assessment, copious irrigation, detailed eye exam  
(c) pH assessment, copious irrigation, evaluation of visual acuity, detailed eye exam  
(d) Copious irrigation, pH assessment, evaluation of visual acuity, detailed eye exam

26. A 42-year-old male presents with painful swelling below the inner aspect of his right eye of one day duration. Exam reveals localized erythematous swelling and tearing. His visual acuity is 20/20, the remainder of his eye exam is unremarkable and he otherwise appears well. The most appropriate treatment for this patient is:
(a) Immediate incision and drainage  
(b) Admission for parenteral antibiotics  
(c) A topical antibiotic ointment  
(d) A broad-spectrum oral antibiotic and warm compresses.
27. A patient with AIDS, and a CD4 count less than 100, presents with decreased vision in his left eye. Fundoscopic exam reveals fluffy white perivascular retinal lesions associated with hemorrhage. Appropriate treatment of this patient involves immediate ophthalmology consult and:
   (a) IV acyclovir
   (b) IV steroids
   (c) IV ganciclovir or foscarnet
   (d) Topical steroids

28. The most common cause of conjunctivitis is:
   (a) Staph. aureus
   (b) Chlamydia trachomatis
   (c) Neisseria gonorrhoea
   (d) Viral

29. All of the following statements about viral conjunctivitis are accurate except:
   (a) Adenovirus is the most common offending agent.
   (b) Constitutional symptoms consistent with a viral syndrome are present in up to 50% of patients.
   (c) A follicular response of the palpebral conjunctiva and preauricular adenopathy are typical exam findings.
   (d) The associated discharge is typically mucopurulent.

30. Initial management for patients with primary open angle glaucoma usually consists of:
   (a) Topical medications to decrease intraocular pressure
   (b) Oral medications to decrease intraocular pressure
   (c) Laser or surgical therapy
   (d) Observation and close follow-up only, until symptoms develop.

Answers: 1. c, 2. a, 3. d, 4. b, 5. a, 6. c, 7. c, 8. b, 9. d, 10. b, 11. c, 12. c, 13. b, 14. d, 15. a, 16. b, 17. c, 18. c, 19. d, 20. c, 21. b, 22. d, 23. a, 24. c, 25. d, 26. d, 27. c, 28. d, 29. d, 30. a
   Use the pre-chapter multiple choice question worksheet (p. xxv) to record and determine the percentage of correct answers for this section.
I. Conjunctivitis

A. Bacterial Conjunctivitis

1. Clinical presentation: Patients usually present with redness, a gritty foreign body sensation and discharge. The discharge is mucopurulent and often results in the eyelids being matted together on awakening; a Gram stain and culture should be obtained in all cases of neonatal conjunctivitis or whenever *Neisseria gonorrhoea* conjunctivitis is suspected (see below). Exam findings include diffuse conjunctival injection, discharge and a clear cornea that does not stain with fluorescein dye; visual acuity and pupillary responses are normal. If *Neisseria gonorrhoea* is the causative agent, marked swelling, redness of the eyelids and severe chemosis (conjunctival edema) may also be present.

2. The types of bacterial conjunctivitis important for this exam are:
   a. *Staph. aureus* conjunctivitis
      (1) Onset is acute.
      (2) The discharge is typically mucopurulent in nature.
      (3) White ulcers may be seen at the limbus (“marginal ulcers”) as a result of an allergic reaction to the staphylococcal toxin.
      (4) There is no central corneal staining with fluorescein dye or cobalt blue light.
      (5) Treatment
         (a) Topical antibiotics: 10% sulfacetamide, quinolones (ciprofloxacin, ofloxacin) or aminoglycosides, trimethoprim/polymyxin (gentamicin, tobramycin) for 5 - 7 days
         (b) Good hygiene (frequent hand washing, separate hand linens)
         (c) Discontinuation of contact lens wear*
         (d) Ophthalmologic follow-up in 2 - 3 days to rule out co-existing corneal involvement.
   b. *Neisseria gonorrhoea* conjunctivitis (an ocular emergency)
      (1) Is an extremely aggressive form of conjunctivitis that can ulcerate and perforate an intact cornea within hours or days.
      (2) It is most commonly seen in newborns (ophthalmia neonatorum), typically in the first 3 days of life; it is also seen occasionally in sexually active adults.
      (3) Incubation period is 2 - 5 days and onset is hyperacute.
      (4) The discharge is characteristically purulent (and an important clue to the diagnosis).

*Contact lens-related conjunctivitis should be treated with an aminoglycoside or quinolone to cover pseudomonas.
(5) Treatment
(a) Parenteral and topical antibiotic coverage for gonorrhea:
• IM or IV ceftriaxone
• Topical erythromycin
(b) Antibiotic coverage for possible concomitant chlamydia: oral erythromycin, doxycycline or tetracycline.
(c) Frequent ocular irrigation with a saline solution
(d) Immediate ophthalmology consult and hospital admission for all but the mildest of cases
(e) Evaluation and treatment of sexual partners

B. Chlamydia Trachomatis Conjunctivitis

1. Is the leading cause of preventable blindness worldwide.
2. Is caused by an obligate intracellular parasite that produces a chronic conjunctivitis with scarring of the cornea and underside of the lids (Arlt's lines).
3. Occurs in newborns (ophthalmia neonatorum) and in sexually active adults (adult inclusion conjunctivitis). In newborns, an associated chlamydial pneumonia may also occur.
4. Signs of infection occur 5 - 14 days after inoculation/birth (compared to only 2 - 5 days with gonorrhea). Gram stain may not be helpful (lack of organisms) but an immunofluorescent antibody screen should be positive.
5. Treatment
   a. Systemic and topical antibiotic therapy*
      (1) Oral erythromycin (in children and newborns); erythromycin, doxycycline or tetracycline (in adults) plus
      (2) Topical erythromycin
   b. Evaluation and treatment of sexual partners.

C. Viral Conjunctivitis

1. Viruses are the most frequent cause of conjunctivitis... and are most commonly caused by adenovirus.
2. The infection is often unilateral initially, but usually spreads quickly (via autoinoculation) to involve both eyes.
3. Up to 50% of patients have constitutional symptoms or a viral prodromal illness consistent with a viral syndrome (fever, rhinorrhea, myalgias and preauricular adenopathy).
4. Clinical presentation: Patients classically present with redness, itching and tearing (watery discharge). Typical exam findings include diffuse conjunctival injection, a follicular response on the palpebral (lid) conjunctiva, a thin watery discharge and preauricular adenopathy. Unless an associated keratitis is present, the cornea is clear; visual acuity and pupillary reactivity are normal.

*Nearly half of infants will have concomitant chlamydial nasopharyngitis.
5. Infection with adenovirus types 8 & 19 → epidemic keratoconjunctivitis — a highly contagious eye infection.
   a. Clinical evolution: itchy irritated eyes → tender preauricular nodes (a few days later) → painful keratitis with severe photophobia and decreased visual acuity (one week later).
   b. Fluorescein staining of the cornea reveals multiple tiny pinpoint dots.

6. Treatment
   a. Antibiotic therapy — Since the symptoms and clinical appearance of viral conjunctivitis are usually inadequate in distinguishing it from bacterial conjunctivitis, most patients are treated with a topical antibiotic such as 10% sulfacetamide or trimethoprim/polymyxin.
   b. Meticulous hygiene (frequent hand washing, separate hand linens) — especially with epidemic keratoconjunctivitis (“pink eye”) — since patients may be contagious for two weeks.
   c. Cool compresses and artificial tears.
   d. Prompt ophthalmologic consultation for patients with evidence of keratitis and for consideration of a topical vasoconstrictor (such as Ophcon-A) for a few days.
   e. Steroids should be strictly avoided.

D. Vernal Conjunctivitis

1. This is one of several types of allergic conjunctivitis.

2. Most commonly affects male children; a family history of atopy is common.

3. A recurrent seasonal inflammation, vernal conjunctivitis occurs in the warmer months of the year.

4. It is characterized by intense itching, burning photophobia, chemosis (swelling of the bulbar conjunctiva), bilateral lid edema, cobblestone papillae under the upper lid and a stringy mucoid discharge.

5. Treatment
   a. A topical antihistamine preparation (such as Livostin®) or a topical decongestant/antihistamine combination (such as Naphcon-A®).
   b. A topical mast cell stabilizer (such as Alomide® or Crolom®).
   c. Cool compresses.
   d. If bacterial coinfection is suspected or the etiology of the conjunctivitis is unclear, a topical antibiotic agent should also be prescribed.

6. A potential complication is a “shield ulcer” produced from the irritated papillae and discharge; if the ulcer becomes infected or scarred, loss of vision may occur.
II. Inflammatory Disorders of the Eyelids and Cornea

A. Anterior Blepharitis

1. This is a chronic and frequently bilateral inflammation of the lid margins due to a staphylococcal infection and/or seborrheic dermatitis; itching and lid margin irritation are common symptoms.

2. Examination reveals crusty, scaly lid margins which may be erythematous and slightly swollen.

3. Treatment
   a. Gentle scrubbing of the eyelids and lashes with baby shampoo (to remove debris) followed by application of a topical antibiotic ointment to the eyelid margins — erythromycin or bacitracin — daily for 4 - 6 weeks; warm compresses and artificial tears are also helpful.
   b. If seborrheic dermatitis of the scalp and eyebrows is present, it should be treated with selenium sulfide shampoo.
   c. Cases resistant to these measures can be treated with oral tetracycline or doxycycline for 2 - 3 weeks.

B. Hordeolum (stye)

1. This is an acute infection (usually staphylococcal) of the eyelid involving either the meibomian glands, Zeis glands or Moll glands.

2. Signs and symptoms include pain, erythema, focal swelling with a tender nodule or pustule on the eyelid (usually close to the margin).

3. Treatment: although styes often point and drain spontaneously, topical antibiotic ointments (bacitracin or erythromycin) and hot compresses hasten the process.

C. Chalazion

1. This is an acute or chronic granulomatous inflammation of a meibomian gland that often evolves from an incompletely resolved hordeolum.

2. It presents as a nontender bump in the midportion of the eyelid.

3. Treatment
   a. Initial treatment is with a topical antibiotic ointment (bacitracin or erythromycin) and hot compresses.
   b. Persistent or chronic recurrent chalazions often respond to the addition of doxycycline for 2 - 3 weeks.
   c. Chalazions that persist for several weeks (or months) despite these measures may eventually require surgical incision and curettage by an ophthalmologist.
D. Dacryocystitis (Be able to identify on pictorial)

1. This is an acute infection of the lacrimal sac due to obstruction of the nasolacrimal duct.
2. It is most often seen in infants and in adults > 40 years old.
3. The most common causative organism is: *Staph. aureus* followed by *Staph. epidermidis*, *Strep.* species and *Hemophilus influenzae*.
4. Patients present with epiphora (tearing) and an acute, unilateral, painful, red swelling below the medial canthus (location of the lacrimal sac). Digital pressure applied over this site may result in the expression of purulent material from the puncta.
5. Treatment: broad-spectrum oral antibiotics (Augmentin), topical ophthalmic antibiotics, warm compresses, gentle massage of the lacrimal sac (to empty stagnant tear accumulation) and ophthalmologic referral; signs of systemic illness may require admission.

E. Corneal Ulcers (a true ocular emergency)

1. Clinical presentation: Patients present with pain, foreign body sensation, photophobia and tearing. Exam reveals conjunctival hyperemia, lid edema and a localized whitish corneal infiltrate that has a staining epithelial defect may also be associated with a hypopyon (an accumulation of white blood cells in the anterior chamber). Signs of iritis (e.g. miosis, cells and flare in the anterior chamber on slit-lamp exam) may also be present.
2. Often seen in patients who use soft contact lenses; the most common pathogen in these patients is *Pseudomonas*. Other common pathogens include *Staph.* and *Strep*.
3. Ulceration may lead to corneal melting and perforation. (*Pseudomonas* is particularly virulent, able to destroy the cornea in 24 hours or less).
4. Treatment:
   b. Hospital admission is rarely needed unless the patient is a neonate or there is extensive corneal involvement.
   c. Topical broad-spectrum antibiotics (quinolones such as ciprofloxacin or ofloxacin Q 30 - 60 minutes) or topical fortified antibiotics (cefazolin plus gentamycin).
   d. A topical cycloplegic (cyclopentolate) for associated iritis and ciliary spasm to control pain.
   e. Discontinuation of contact lens use; no patch.
F. Herpes Simplex Keratitis (Be able to identify on pictorial)

1. May be the result of primary or recurrent infection with the herpes simplex virus (usually type I). Skin and mucocutaneous lesions are more common in primary infections, whereas corneal involvement (keratitis) is more common in recurrent infections.

2. Clinical presentation: Patients present with ocular pain, a foreign body sensation, photophobia and tearing. Visual acuity may be reduced. Examination reveals a diffusely reddened eye, decreased corneal sensation and preauricular adenopathy. Slit lamp evaluation of the unstained cornea may reveal localized corneal haziness. Fluorescein staining usually demonstrates the classic dendritic (branching) pattern over the cornea in well-established, recurrent infections; in primary infections and early on in recurrent infections, however, only a superficial punctate keratitis may be seen.

3. Treatment
   a. Prompt ophthalmology consultation regarding specific treatment recommendations and follow-up is mandatory; these infections are aggressive and may lead to corneal destruction resulting in permanent disability.
   b. A topical antiviral agent such as trifluridine or vidarabine.
   c. A topical cycloplegic such as cyclopentolate for pain relief from ciliary muscle spasm if an associated iritis is present. [Note: Although topical steroids are occasionally prescribed by ophthalmologists for patients with refractory or advanced stromal keratitis, they can promote rapid progression of the infection and are, therefore, contraindicated in all other forms of herpetic keratoconjunctivitis and should not be prescribed by the EP].

G. Herpes Zoster Ophthalmicus

1. Results from reactivation of latent varicella zoster virus in the trigeminal ganglion.

2. Clinical presentation: Patients present with pain, paresthesias, tearing and a unilateral vesicular eruption in the dermatome supplied by the ophthalmic branch of the trigeminal nerve.

3. Ocular involvement can range from conjunctivitis to iritis, keratitis, corneal anesthesia or ocular muscle palsies; lesions on the tip of the nose (Hutchinson’s sign) signal nasociliary nerve involvement and a very high likelihood of ocular lesions.

4. Treatment
   a. Immediate ophthalmology consultation
   b. Oral acyclovir or famciclovir
   c. A topical broad-spectrum antibiotic to prevent secondary infection
   d. A topical cycloplegic agent and a topical steroid (in consultation with an ophthalmologist) if iritis is present
   e. Oral analgesics as needed
H. Ultraviolet Keratitis

1. Prolonged ultraviolet radiation exposure can cause damage to the corneal epithelium.

2. Typical sources of ultraviolet radiation are:
   a. Arc welding (welders’ keratitis / flash)
   b. Reflected sunlight, especially at high altitude (snow blindness)
   c. Artificial sunlight (tanning booths, sun lamps)

3. Symptoms develop 6 - 8 hours following exposure and include severe pain, photophobia, a foreign body sensation, tearing and blepharospasm. There is decreased visual acuity, conjunctival injection and a diffuse punctate keratopathy (multiple pinpoint fluorescein-stained dots on the corneal surface seen with cobalt blue light).

4. Treatment:*  
   a. A topical cycloplegic agent such as cyclopentolate or scopolamine for pain control  
   b. A topical broad-spectrum antibiotic ointment  
   c. An oral analgesic  
   d. Eye patching as needed for comfort.  
   e. Follow-up with ophthalmology in 24 hours

III. Periorbital and Orbital Cellulitis

A. Periorbital (Preseptal) Cellulitis

1. An infection confined to superficial tissues that are anterior to the orbital septum (the fascial layer that acts as a partition separating the eyelids from the orbit).

2. This is more common and generally less serious than orbital cellulitis.

3. Children (particularly those < 3 years old) are most commonly affected.

4. *Streptococci, Staphylococci* and *H. influenzae* are the most common pathogens; *H. influenzae* is more common in young children, but due to the HIB vaccine its incidence in this population is decreasing.

5. Usually arises by hematogenous spread (bacteremia) from otitis media or pneumonia or by direct extension from ethmoid sinusitis, skin infections or trauma.

6. Patients present with erythema, edema, warmth and tenderness of the eyelid, conjunctival injection and occasionally fever. However, visual acuity, eye movements and pupillary findings are normal (which helps distinguish preseptal from orbital cellulitis).

*Avoid topical anesthetics; frequent use retards healing.*
7. Laboratory evaluation should include:
   a. Blood cultures
   b. Uninfused CT of the orbit — if orbital cellulitis cannot be definitively ruled out by clinical evaluation.

[Note: Soft-tissue aspirates for gram stain and culture are no longer recommended due to risk of septum perforation with susequent risk of deeper infection.]

8. Treatment
   a. A broad-spectrum antibiotic which covers Streptococci, Staphylococci and H. influenzae. Older children and adults with early/mild periorbital cellulitis may be treated on an outpatient basis with an oral antibiotic (such as Augmentin) if follow-up in 24 hours can be assured. Adults with more advanced infections and children < 5 years of age should be admitted for parenteral antibiotics (ceftriaxone or vancomycin) due to the increased incidence of associated bacteremia and meningitis.
   b. Ophthalmology consult.

B. Orbital (Postseptal) Cellulitis

1. Infection of the tissues within the orbit posterior to the orbital septum. (Note: Preseptal cellulitis is invariably present in these patients.)

2. Affects all age groups, but is more common in children.

3. Staph. aureus, Strep. pneumoniae and H. influenzae (the bacteria associated with acute sinusitis) are the most common etiologic agents. Mucormycosis should be considered in the diabetic and immunocompromised.

4. Most cases arise from direct extension of a sinus infection (usually from the ethmoid).

5. Patients present with the same findings seen in those with periorbital cellulitis but also have chemosis, ocular pain, pain with extraocular movements, limitation of extraocular muscle function, pupillary paralysis and proptosis. Decreased visual acuity, increased intraocular pressure and loss of sensation in the ophthalmic and maxillary branches of the trigeminal nerve may also be present.

6. Laboratory evaluation should include:
   a. Blood cultures
   b. Soft-tissue aspirates for gram stain and culture
   c. Uninfused CT of the orbit and paranasal sinuses; if negative, repeat CT with contrast (looking for a subperiosteal abscess).

7. Treatment
   a. Hospital admission
   b. Broad-spectrum IV antibiotic covering gram-positive cocci and H. flu
   c. Immediate ophthalmology/ENT and infectious disease consults
8. Complications include:
   a. Visual loss
   b. Cavernous sinus thrombosis
   c. CNS involvement
   d. Osteomyelitis

IV. Acute Eye Pain (with associated decrease in visual acuity)

A. Acute Iritis (Acute Anterior Uveitis)

1. Clinical presentation: The patient presents with a painful red eye, severe photophobia and blurring of vision. He often has a prior history of iritis or similar undiagnosed pain in the past. Generally, only one eye is affected and symptoms develop over time. Physical exam reveals a constricted and sometimes irregular pupil, a ciliary flush (diffuse reddening of the sclera at the limbus) and a decrease in visual acuity. The intraocular pressure is variable; it is often decreased (due to paralysis of ciliary body function) but may be normal or increased (due to inadequate aqueous drainage). Slit-lamp exam is diagnostic and reveals flare (protein) and cells (leukocytes) in the anterior chamber. Keratic precipitates (an accumulation of WBCs on the endothelial surface of the cornea) may also be seen.

2. Helpful clues to the diagnosis include:
   a. The presence of consensual photophobia (the photophobia produced by iritis is both direct and consensual).
   b. The presence of pain unrelieved by diagnostic application of a topical anesthetic agent (unlike the superficial pain associated with a corneal abrasion which is readily relieved by the diagnostic application of a topical anesthetic agent, the deep-seated ache of iritis is not).

3. Causes
   a. Trauma (occurs 1 - 4 days post-trauma)
   b. Seronegative arthritides (idiopathic ankylosing spondylitis, Reiter's syndrome, etc.)
   c. Inflammatory bowel disease
   d. Chronic granulomatous conditions (TB, sarcoidosis, etc.)
   e. Local infection/ulcers
   f. Sexually transmitted diseases (syphilis, gonorrhea)
   g. Corneal abrasions and foreign bodies
   h. Idiopathic
4. Treatment
   a. A long-acting topical cycloplegic such as homatropine 5% or scopolamine 0.25% — to provide comfort (by eliminating ciliary spasm) and prevent formation of posterior synechiae (adhesions between the iris and lens).
   b. A topical steroid such as prednisolone acetate 1% — to relieve inflammation; consult with an ophthalmologist first and arrange follow-up in 24 hrs.

5. Complications — If the pupil is not dilated, iritis may result in formation of posterior synechiae. These adhesions produce permanent pupillary disfigurement and can lead to secondary glaucoma.

B. Acute Angle Closure Glaucoma (an ocular emergency)

1. Occurs in patients with shallow (narrow) anterior chamber angles. Narrowing of this angle (either congenital or as a result of aging) results in closer contact between the iris and lens producing resistance to flow of aqueous humor from the posterior to the anterior chamber (a relative pupillary block). When the angle closes completely, exit of the aqueous humor is prevented and the intraocular pressure increases abruptly. This condition is precipitated by stress, pupillary dilation either by medications (parasympatholytics, sympathomimetics or anticholinergics), inadvertent administration of a topical cycloplegic in a patient with a shallow anterior chamber or, more commonly, by moving from daylight to a darkened room. (Note: This is predominantly a condition of the elderly, is more common in farsighted [hyperopic] individuals, as well as in women, and typically occurs abruptly in patients with no previous history of glaucoma).

2. Diagnosis
   a. Symptoms: nausea/vomiting, pain (either localized to the eye/eyebrow or generalized as headache pain), blurred vision, halos around lights (due to corneal edema) and (occasionally) abdominal pain.
   b. Signs
      (1) Decreased visual acuity
      (2) A shallow anterior chamber
      (3) A red, congested-looking eye with a fixed, mid-dilated, non-reactive pupil, a hazy cornea and ciliary (perilimbal) injection
      (4) Rock-hard consistency of the globe on gentle palpation
      (5) Intraocular pressure > 40mmHg

3. Treatment
   a. Have the patient lie completely supine. (gravity may allow the lens to pull away from the iris.)
   b. Pharmacologic therapy — consists of the sequential administration of several different agents (which act by various means) to decrease the intraocular pressure (IOP).
(1) Topical beta-blockers (e.g. timolol), α-agonists (e.g. Iodipine) and carbonic anhydrase inhibitors (acetazolamide) — decrease the secretion of aqueous humor by the ciliary body. Dosings: timolol 0.5% 1 drop followed by Iopidine (apraclonadine)* 0.1% 1 drop and acetazolamide 500mg PO or IV.

(2) Topical steroids (e.g. prednisolone 1%) reduce inflammation. Dose: 1 drop Q 15 mins × 4, then hourly.

(3) Hyperosmotic agents**(e.g. mannitol) — decrease the volume of fluid in the eye and rapidly lower IOP (check hourly). Dose: mannitol (20% sol.) 1 - 2gms/kg IV over 30 - 60 mins.

(4) Topical miotics (e.g. pilocarpine) — pull the iris back from its anterior position, thereby reopening the angle and allowing egress of aqueous humor; they do not usually have an effect until the IOP is < 40mmHg due to pressure-induced ischemic paralysis of the iris. Dose: pilocarpine 2% sol., 1 drop Q 6 hours (when the IOP is < 40mmHg). Treat the unaffected eye prophylactically with pilocarpine 1/2% sol., 1 drop Q 6 hours since the chamber angle is usually narrow in both eyes and is also at risk for acute closure.

(5) Analgesics as needed

c. Immediate ophthalmology consult
d. Definitive treatment is surgical; bilateral laser iridotomy is performed once the acute episode is resolved and the cornea clears.

V. Acute Visual Loss

A. Central Retinal Artery Occlusion (a true ocular emergency)

1. Clinical Picture
   The patient experiences sudden, painless, monocular loss of vision due to a thrombotic plaque or embolus (more common) of the central retinal artery. Physical exam is usually normal initially but, after an hour or two, reveals a dilated pupil unreactive to direct light but with a positive consensual response to light (Marcus-Gunn pupil). Visual acuity is markedly decreased (light perception or finger counting) and funduscopic examination reveals a pale retina with a cherry-red spot in the macular area (fovea).

2. Patients are usually between 50 and 70 years of age and frequently have one of the following risk factors:
   a. Hypertension
   b. Carotid artery disease
   c. Diabetes mellitus
   d. Cardiac disease (atrial fibrillation, valvular disease)

*Acts synergistically with the beta-blocker
**Osmotic diuretics have the greatest potential for serious side effects, especially in the setting of cardiovascular or renal disease.
e. Vasculitis
f. Temporal arteritis
g. Sickle cell disease

3. Treatment
a. Must be instituted as soon as the clinical diagnosis is suspected; an occlusion that persists longer than 90 minutes usually results in permanent visual loss.
b. The aim of treatment is to restore retinal artery blood flow by dislodging the clot, dilating the artery + lowering intraocular pressure.
c. Treatment modalities include:
   (1) Intermittent digital massage of globe (5 sec. on, 5 sec. off) for 5 to 15 minutes.
   (2) Inhalation of 95% O₂ and 5% CO₂ (carbogen) 3L/min or hyper-ventilating into a paper bag for 10 minutes every hour.
   (3) Acetazolamide 500mg IV + a topical B-blocker (Timolol® 0.5%).
   (4) Immediate ophthalmology referral for paracentesis of the anterior chamber.
d. Evaluation for treatable associated disease processes is appropriate.

B. Central Retinal Vein Occlusion

1. Clinical Picture
   The patient complains of a sudden, painless, monocular decrease in vision. The visual loss is usually noted on awakening and is due to thrombosis of the central retinal vein. The pupil reacts sluggishly to light and the fundoscopic exam reveals retinal hemorrhages and tortuous retinal veins ("blood and thunder fundus"). Edema of the disc may also be present. The degree of visual loss is variable but is typically less severe than that which occurs in association with central retinal artery occlusion.

2. Risk factors include:
   a. Hypertension
   b. Diabetes Mellitus
   c. Arteriosclerosis
   d. Chronic glaucoma
   e. Vasculitis

3. Treatment — no immediate treatment is effective.
   a. Refer patients to ophthalmology for:
      (1) Confirmation of the diagnosis and
      (2) On-going monitoring for the development of complications.
   b. Identify and treat contributing factors.

4. Complications — develop in a delayed fashion:
   a. Neovascularization of the retina and/or iris
   b. Neovascular glaucoma
C. Optic Neuritis

1. Is an inflammatory process of the optic nerve; it is typically monocular in adults and binocular in children; occurs in women more than men.

2. Is characterized by reduction of central vision with preservation of peripheral vision; visual loss ranges from mild to severe (finger counting to light perception).

3. Most cases of optic neuritis are retrobulbar and do not have changes in the optic disc.

4. Clinical Picture
   The patient is usually between 15 and 45 years of age and presents with a rapidly progressive (hours to days) reduction or blurring of vision in association with ocular pain that is worsened by eye movement.

5. The presence of a Marcus-Gunn pupil (afferent pupil defect), red vision desaturation (the color red is perceived as less intense in the affected eye) and a central visual field defect confirms optic pathology. The disc may be normal (retrobulbar optic neuritis) or swollen and hyperemic (papillitis). The remainder of the eye exam is otherwise normal.

6. A clear association with multiple sclerosis exists; 25 - 65% of these patients will go on to develop MS in the ensuing years.

7. Treatment
   a. Emergent ophthalmology consult
   b. Admission for intravenous steroids (IV methylprednisolone) for 3 days, followed by oral steroids for 11 days.

D. Eclipse Burn (solar retinopathy)

1. This is caused by prolonged or accidental viewing of the sun which leads to permanent central retinal (macular) loss of vision.

2. Visual acuity is decreased and funduscopic exam demonstrates discrete retinal disruption in the macular area.

3. No treatment is effective; the patient should be referred to an ophthalmologist.
E. Retinal Detachment (an ocular emergency)

1. Involves a separation of the inner neuronal retina from the outer retinal pigment epithelial layer, usually as the result of a tear in the retina from vitreous traction.

2. Is painless and usually seen in patients with one or more risk factors:
   a. Severe myopia
   b. Older age
   c. Trauma
   d. Previous cataract surgery
   e. A family history of retinal detachment
   f. An inherited connective tissue disorder (e.g. Marfan’s syndrome)
   g. Diabetes Mellitus

3. Clinical Picture
   Early symptoms are flashing lights (as the retina begins to tear) and the presence of “spider webs” or “coal dust” across the visual field (small vitreous hemorrhages). As the retina detaches, the patient experiences a sensation of a curtain that is gradually lowering or raising in front of the affected eye. Examination with a direct ophthalmoscope may reveal an undulating, dull gray, detached retina. The tear itself often originates in the retinal periphery and can be seen with the indirect ophthalmoscope.

4. As long as the macula remains attached, the probability of preserving central retinal vision is very good.

5. Treatment
   a. Immediate ophthalmology consultation for evaluation and retinal attachment surgery is indicated.
   b. The patient should avoid vigorous activity and preferably remain at bed rest until evaluated by the ophthalmologist.

F. Vitreous Hemorrhage

1. Is a common cause of sudden painless unilateral loss of vision.

2. Greatest risk factor is proliferative diabetic retinopathy; posterior vitreous detachment and retinal vessel occlusion occur most commonly in the elderly patient.

3. Clinical Picture
   If hemorrhage is preceded by retinal detachment, the patient may experience flashes of light (photopsias). Otherwise, unilateral painless vision loss occurs (often described as haze, shadows, cobwebs or smoke). On exam, the red reflex is diminished or absent and the fundal view is hazy. Visual acuity may not be affected.

4. Treatment — Immediate ophthalmology consult.
G. Functional Blindness

1. Unilateral or bilateral blindness in the presence of normal pupillary reactions, a normal funduscopic exam and lack of an afferent pupillary defect is very suggestive of a functional problem (hysteria/conversion disorder or malingering).

2. An intact visual pathway can be confirmed with use of an optokinetic drum or strip which will elicit optokinetic nystagmus.

3. Despite the serious nature of their complaint, hysterical patients are often remarkably calm. Malingers, on the other hand, are usually overly emotional and less cooperative.

4. Treatment: ophthalmologic and psychiatric referrals.

H. Temporal (Giant Cell) Arteritis

1. Is a vasculitis of medium and large arteries that can cause optic nerve infarction and permanent visual loss. It is notorious for involving the contralateral optic nerve within hours or weeks. For unknown reasons, the incidence of this disorder appears to be rising.

2. Clinical Picture
   Patients usually present with a sudden monocular loss of vision and a unilateral temporal headache; eye pain is not typical. Up to 50% of these patients have polymyalgia rheumatica (a syndrome of aching, pain and stiffness in the proximal muscles, especially neck and shoulders); some patients may have associated neurologic symptoms that simulate a TIA or stroke. There is a female predominance and most patients are in their fifties or older. Examination reveals tender, tortuous (and sometimes pulseless) temporal arteries, visual loss, an afferent pupillary defect and a pale, swollen optic disc. Diagnosis is suggested by an elevated sed rate (>50mm/hr)* and C-reactive protein; it is confirmed by temporal artery biopsy.

3. Treatment: immediate ophthalmology (and internal medicine) consultation and administration of high-dose steroids (IV or oral). IV methylprednisolone is associated with a better prognosis.

Note: Further discussion of this topic can be found in the ENT chapter on pp. 208 - 209 and in the Neurology chapter on p. 919.

*The sed rate increases with age; a simple rule of thumb for estimating the upper range of "normal" for patients older than 50 is: age + 2 (for males) and age + 10 + 2 (for females).
VI. Nonacute Visual Loss

A. Primary Open Angle Glaucoma

1. Is the most common type of glaucoma and a major cause of slowly-progressive blindness in the U.S.

2. It is a painless condition of chronically increased intraocular pressure that ultimately damages the optic nerve. It is frequently bilateral and there is a strong familial tendency.

3. Signs and symptoms
   a. Normal appearing cornea and pupil
   b. Increased intraocular pressure ( > 21mmHg)
   c. Increased optic nerve cup/disc ratio ( > 0.4)
   d. Arcuate scotomas
   e. Gradual loss of peripheral vision with sparing of central vision

4. Treatment
   a. Unlike acute angle closure glaucoma, open angle glaucoma is a chronic, painless, nonemergent condition that does not generally present to the Emergency Dept.
   b. Patients should be referred to an ophthalmologist for evaluation and treatment.
   c. Treatment is usually with topical agents that decrease intraocular pressure by reducing the production of aqueous humor and/or increasing aqueous outflow.

B. Cytomegalovirus (CMV) Retinitis

1. Is a necrotizing retinitis that, if left untreated, results in progressive visual loss.

2. It occurs in immunocompromised patients, most notably those with advanced AIDS.

3. Clinical Picture
   Although patients can be asymptomatic early on, they typically present with scintillating scotomas, visual distortions and visual loss. Funduscopic exam reveals fluffy white retinal lesions that are usually perivascular in location and associated with hemorrhage (the characteristic “pizza pie” or “cheese and ketchup” fundus).

4. Treatment: Immediate ophthalmology referral and IV administration of ganciclovir or foscarnet.
VII. Trauma

A. Chemical Burns

1. Alkali burns (a true ocular emergency)
   a. Generally more devastating than acid burns.
   b. Examples of strong alkalis include: sodium hydroxide (lye), calcium hydroxide (lime), potassium hydroxide and ammonia.
   c. Produce a liquefaction necrosis that continues to dissolve and penetrate the tissues until the alkaline substance is removed; perforation of the globe may occur.

2. Acid burns
   a. Produce coagulation necrosis of the cornea; invasion of the acid is limited by the coagulum formed.
   b. Examples of strong acids include: sulfuric acid (battery acid), sodium hypochlorite (bleach) and toilet cleaners.

3. Treatment
   a. Immediate and copious irrigation at the scene followed by continuous irrigation in the ED using a minimum of 2 L of NS or LR per affected eye
      (1) Topical anesthetic drops should be instilled to make the patient more comfortable and tolerant of the irrigation.
      (2) Particulate matter should be removed with a moist cotton-tipped applicator.
      (3) The pH of the conjunctival fornix should be assessed periodically to monitor the progress of the irrigation:
         (a) It can be tested using nitrazine paper or a urine pH stick.
         (b) Irrigation should be continued until the pH is neutral (7.4) on two consecutive tests 10 minutes apart; alkali burns may require several hours irrigation (up to 24 hrs).
   b. Irrigation should be followed by:
      (1) A complete eye exam (including visual acuity + IOP*)
      (2) Instillation of a topical cycloplegic (e.g. cyclopentolate or scopoline) and an antibiotic ointment (e.g. bacitracin, erythromycin).
      (3) Administration of oral analgesics as needed.
      (4) Ophthalmology referral for follow-up in 24 hours.

   [Note: Severe burns, particularly those due to alkalis, require immediate ophthalmology consultation, discussion of additional treatment measures (e.g. topical steroids) and hospital admission.]

*Requires treatment if elevated.
B. Lid Lacerations

1. Once injury to the underlying globe has been ruled out, superficial lacerations can be repaired with 6-0 or 7-0 nonabsorbable suture.

2. Lacerations in the following anatomically high-risk areas require referral for repair:
   a. Lacrimal canaliculi (lacerations of the lid margin that lie medial to the lacrimal puncta)
   b. Levator muscle/tendon (deep horizontal lacerations of the upper lid) — the presence of ptosis is suggestive.
   c. Canthal tendons (penetrating wounds that transect the lateral or medial canthi)
   d. Orbital septum (deep horizontal lacerations of the upper lid) — the protrusion of fat into the laceration confirms septal perforation.
   e. Lid margins and lid lacerations with extensive tissue loss

C. Corneal Injuries

1. Corneal abrasions
   a. Patients complain of pain, foreign body sensation, tearing and photophobia. Conjunctival injection is frequently present; visual acuity may be decreased if the abrasion is large or centrally located.
   b. Instillation of a topical anesthetic agent will provide temporary pain relief and permit a better exam. [Note: Although these agents are useful diagnostically, they should NEVER be prescribed for pain on an outpatient basis; corneal toxicity and retardation of epithelial regrowth occur with repetitive use.]
   c. Always evert the lids to look for entrapped conjunctival foreign bodies; multiple fine linear abrasions of the corneal surface (“ice rink sign”) are highly suggestive of a foreign body trapped under the upper lid.
   d. Examination using a cobalt blue light source (slit lamp or Wood’s light) with fluorescein staining reveals the abraded area (it stains bright green).
   e. Treatment
      (1) A long-acting cycloplegic agent such as cyclopentolate or homatropine — to relieve ciliary spasm and reduce pain.
      (2) A topical antibiotic — to prevent secondary infection; use an agent with good gram-negative coverage for abrasions associated with soft contact lens use: a topical aminoglycoside (e.g. tobramycin, gentamycin) or a quinolone (e.g. ciprofloxacin, ofloxacin).
      (3) Eye patching
         (a) Does not appear to facilitate healing (or provide comfort) and can have detrimental effects, especially if the injury was caused by vegetable matter or the patient wears contact lenses.
         (b) Should clearly be avoided in corneal abrasions due to prolonged contact lens use. These abrasions are associated with an increased incidence of gram-negative bacterial infections (particularly pseudomonas) and ulceration. The warm, dark and relatively hypoxic environment created by patching may further predispose these abrasions to infection.
(4) Oral analgesics — are seldom needed if the eye is adequately treated with a cycloplegic agent (e.g. cyclopentolate one drop Q 6 - 8 hrs; however, if the patient cannot be redosed with a cycloplegic, then oral analgesia will become necessary.

(5) Ophthalmology referral — for re-evaluation in 24 - 36 hours

2. Corneal rust rings
   a. Corneal foreign bodies containing iron produce a rust ring which is irritating and can be difficult to remove.
   b. Apply a topical anesthetic agent and then remove the rust ring with a burr drill or a sterile needle. The burr drill is preferred because the removal can be accomplished more quickly and easily with this instrument.*
   c. Once the rust ring has been removed, apply a topical broad-spectrum antibiotic (e.g. erythromycin) and a cycloplegic agent; refer to an ophthalmologist for re-evaluation in 24 - 48 hours.
   d. If the rust ring can not be easily removed, treat as above and refer to an ophthalmologist for removal the following day.

3. Corneoscleral lacerations and perforations
   a. Because of Bell’s phenomenon,** they are commonly located in the inferior aspect of the globe.
   b. Diagnostic clues
      (1) Teardrop-shaped pupil
      (2) Flattening of the anterior chamber
      (3) Small fragments of black (iris) pigment at the wound edges
   c. Examination with the cobalt blue light source of the slit-lamp with fluorescein staining will mark the area of suspected aqueous leak; the efflux of aqueous humor will cause the fluorescein (a lime-green liquid) to flow from the laceration in a “riverlike” pattern (positive Seidel test) and is diagnostic if present.
   d. Treatment
      (1) Do NOT put any pressure on the globe (not even an IOP check with a tonopen or Schiötz tonometer).
      (2) Apply a rigid metal eye shield (NOT a patch) to protect the eye from any inward pressure.
      (3) Keep patient NPO (since many will need repair in the OR).
      (4) Provide tetanus prophylaxis as needed.
      (5) Administer:
         (a) Prophylactic IV antibiotics
         (b) Prophylactic antiemetics
         (c) Analgesics as needed
      (6) Obtain immediate ophthalmology consult.

*Rosen’s 5th edition suggests that the rust ring should be left for the ophthalmologist to remove at the 24-hour follow-up since the rust ring will migrate to the corneal surface by that time.

**Eyeball rolling upward and outward in response to eye closure.
D. Ruptured Globe

1. Suggestive findings:
   a. Teardrop-shaped pupil
   b. Bloody chemosis
   c. Decrease in visual acuity
   d. Distorted anterior chamber
   e. Vitreous hemorrhage

2. Treatment: Is the same as that for corneoscleral lacerations and punctures as described above.

E. Hyphema (Be able to identify on photograph)

1. Clinical recognition and significance
   a. Blunt ocular injury may cause bleeding into the anterior chamber (hyphema). The bleeding originates from the blood vessels of the ciliary body or iris.
   b. Although some patients with small hyphemas are asymptomatic, most complain of blurred vision, dull eye pain and photophobia.
   c. Hyphemas are graded according to the percentage of blood in the anterior chamber and range in size from microscopic (grade 0) to total (grade IV).
   d. They are most easily detected when the patient is examined in the upright (sitting) position. In this position, blood will layer out in the inferior aspect of the anterior chamber forming a meniscus.
   e. Microscopic hyphemas will go unnoticed without slit-lamp exam.
   f. A detailed eye exam should be performed to rule out associated injuries (e.g. ruptured globe); the intraocular pressure should be measured since it may be elevated due to RBCs clogging the trabecular meshwork, thus impeding aqueous outflow.

2. Treatment
   a. Bedrest with the head of the bed elevated 45°
   b. Metal eye shield
   c. Avoidance of eye movement (e.g. watching TV, reading)
   d. Analgesics as needed but no NSAIDs or ASA products
   e. Antiemetics for nausea
   f. Medications to reduce intraocular pressure (IOP) if > 30 mmHg in a normal patient or > 24mmHg in a sickle cell patient.*
   g. Immediate ophthalmology consult
   h. Administration of other meds (mydriatics, cycloplegics, steroids and antifibrinolytics) and the need for hospitalization should be decided in consultation with the ophthalmologist.
      (1) A cycloplegic/mydriatic agent is often used to dilate the pupil and avoid “pupillary play” (pupil size fluctuations due to changing ambient lighting) so as not to stretch the leaking blood vessel.
      (2) Steroids and antifibrinolytics may prevent rebleeding.
   i. Surgical evacuation may be necessary with large (grade IV) hyphemas.

*Avoid carbonic anhydrase inhibitors in sickle cell patients; they can cause RBCs to sickle in the anterior chamber which clogs the trabecular meshwork and further increases IOP.
3. Complications*
   a. Rebleeding — usually occurs 2 - 5 days following the initial bleed (when clots begin to lyse) and is the most common complication; it can cause an abrupt and sight-threatening rise in IOP.
   b. Blood staining of the corneal epithelium
   c. Secondary glaucoma
   d. Anterior and posterior synechia formation

F. Lens Subluxation and Dislocation
1. Is usually caused by blunt trauma to the globe, but can occur spontaneously or with trivial trauma in patients with Marfan’s syndrome, homocystinuria, tertiary syphilis and rheumatoid arthritis.
2. Lens subluxation produces visual distortion and monocular diplopia whereas lens dislocation causes severe blurring of vision.
3. The lens may dislocate either anteriorly into the anterior chamber or posteriorly into the vitreous cavity.
   a. Anterior dislocations — can produce acute angle closure glaucoma by mechanically blocking the egress of aqueous humor.
   b. Posterior dislocations — produce iridodonesis (a quivering of the iris after rapid eye movement).
4. With subluxations, the edge of the lens will be seen on slit-lamp exam when the pupil is dilated.
5. Treatment: obtain immediate ophthalmology consultation.
   a. Anterior dislocations require immediate repositioning or removal of the lens.
   b. Posterior dislocations are handled more conservatively, often only with refractive correction.

G. Blowout Fractures of the Orbit
1. Etiology
   Blunt trauma to the eye transmits hydraulic forces throughout the globe. The floor and medial wall of the orbit are most susceptible to blowout fractures. Orbital floor fractures can entrap the inferior rectus and inferior oblique muscles as well as fat; contusion or laceration of the infraorbital nerve may also occur. Fractures of the medial wall of the orbit can entrap the medial rectus muscle. [Orbital floor fractures are discussed below. Further discussion of medial wall fractures (which are less common) can be found in the Trauma chapter under facial injuries on pp. 487 - 488.]
2. Signs and symptoms of orbital floor fractures:
   a. Pain and diplopia on upward gaze
   b. Enophthalmos (recognizable as slight ptosis)
   c. Infraorbital nerve distribution anesthesiad. Limitation of upward gaze (produces marked diplopia in the upper visual fields and suggests muscle entrapment)
   e. Subcutaneous orbital emphysema (suggests a medial wall fracture with ethmoid disruption)

3. Be sure to perform a careful eye exam in these patients, since up to 32% of them will have concomitant injuries to the globe.

4. Imaging studies
   a. Plain x-ray findings (best visualized with a modified Waters view):
      (1) Prolapsed orbital tissue in the maxillary antrum (“tear-drop” sign)
      (2) Bony disruption of the orbital floor
      (3) An air-fluid level in the maxillary sinus
   b. Although CT scanning (with coronal and axial views) provides excellent images of these fractures, emergent scanning should be reserved for those few patients you suspect may require surgery (e.g., those with enophthalmos* or EOM entrapment).

5. Treatment — Though symptoms usually resolve spontaneously with conservative management, ophthalmology consult is still indicated to R/O associated injuries to the globe. Broad-spectrum prophylactic antibiotics and decongestants should be considered for those patients with sinus fractures. Patients with subcutaneous emphysema should be advised to avoid nose blowing as well as Valsalva maneuvers.

H. Retrobulbar Hematoma (an ocular emergency)

1. Etiology: extreme blunt or deep penetrating trauma to the eye produces orbital hemorrhage with accumulation of blood behind the globe.

2. Signs and symptoms
   a. Proptosis (may be severe)
   b. Decreased visual acuity
   c. Eye pain
   d. Limited mobility of the globe
   e. Increased intraocular pressure
   f. Afferent pupil defect (APD or Marcus-Gunn pupil)

3. Orbital CT will demonstrate the hematoma.

4. Treatment: Immediate ophthalmology consult for emergent decompression via a lateral canthotomy and anterior chamber paracentesis.

*Eye appears small due to loss of floor support with the globe falling backward and sinking lower into the orbit.
I. Intraocular Foreign Body

1. Historical clue: work that involves pounding metal on metal or using a grinding wheel; physical exam clue: an irregular pupil that is usually peaked toward the site of penetration.

2. Onset of symptoms may be delayed (24 hrs. or more)
   a. Reduced visual acuity
   b. Dull, unlocalized ocular pain

3. Radiographic confirmation and localization of the penetrating FB is indicated since these FBs may need to be surgically removed.
   a. Orbital films may demonstrate FB.
   b. Ultrasound or CT scanning are very helpful in the precise localization of intraocular FBs (or associated air). MRI also provides accurate localization but is not as readily available and cannot be used with metallic objects (could dislodge them).

4. Management
   a. Perform a Seidel test to see if an aqueous leak from the site of penetration is present. Do not attempt to check IOP if penetration of the globe is suspected.
   b. Obtain an immediate ophthalmology consult; the decision to remove an intraocular FB (or not) is based on the composition/reactivity of the object as well as its location in the eye.
      (1) Wood, vegetable matter and metals (i.e. iron, copper and steel) typically incite an intense inflammatory reaction when left in the eye. BBs and pellets are also poorly tolerated since they are composed of iron as well as lead. Emergent surgical extraction is indicated.
      (2) Inert foreign bodies (e.g. glass, lead, plaster, rubber, silver and stone) that are minimally symptomatic may be treated with a non-operative approach. However, many inert foreign bodies can cause toxicity by virtue of a coating or chemical additive that may influence management decisions.
   c. Place a metal shield over the eye, administer a broad-spectrum antibiotic IV and provide tetanus prophylaxis as needed.

J. Traumatic Iritis/Iridocyclitis

1. Clinical Picture
   Blunt ocular trauma can produce traumatic iritis/iridocyclitis. Onset of symptoms (aching pain, photophobia and reduced visual acuity) usually begin 1 - 4 days after injury. The pupil is constricted and a deep ciliary flush is usually present. Slit-lamp exam reveals cells and flare in the anterior chamber.

2. Treatment: a long-acting cycloplegic agent such as homatropine 5% (or scopolamine 0.25%), a topical steroid (in consultation with an ophthalmologist) and ophthalmology referral.
K. Traumatic Miosis and Mydriasis

1. Blunt ocular trauma can produce:
   a. Bruising and irritation of the iris sphincter → constriction and spasm of the pupil (miosis), while
   b. Tears in the iris sphincter muscle fibers → loss of constriction ability → dilation (mydriasis)

2. These defects may resolve spontaneously over a period of days to weeks or, in the case of actual iris sphincter muscle fiber damage, may become permanent.

3. Treatment: No acute treatment is indicated; however, these patients should be referred to an ophthalmologist for follow-up.

VIII. Anisocoria (Unequal Pupil Size)

A. Causes Include:

1. Normal finding - simple physiologic anisocoria (usually ≤ 1mm) occurs in up to 20% of the population.

2. Intracranial third nerve palsy - secondary to an expanding supratentorial mass/bleed or a posterior communicating artery (PCA) aneurysm.*


4. Traumatic mydriasis or miosis.

5. Uniocular drug exposure.

6. Adie’s tonic pupil — benign condition primarily affecting women 20 - 40 years of age who often present with blurred vision.


B. Helpful Diagnostic Measures

1. Compare pupil sizes in bright light and under dark conditions.
   a. With simple physiologic anisocoria, the pupil size discrepancy is the same in all levels of illumination. Furthermore, the pupils in these patients are round and respond normally to light and accommodation; associated findings (e.g. ptosis) are absent.
   b. Anisocoria that is more pronounced in the dark suggests that the smaller pupil is abnormal (e.g. Horner’s syndrome).
   c. Anisocoria that is greater in bright light suggests the larger pupil is abnormal (e.g. third nerve palsy).

*Absence of an isolated third nerve palsy does not rule out an aneurysm.
2. Inquire about prior eye surgery/trauma.

3. Consider the setting and associated signs and symptoms.
   a. Head trauma + altered level of consciousness → r/o an expanding supratentorial mass/hemorrhage
   b. Headache + third nerve palsy → r/o a PCA aneurysm
   c. Miosis + ptosis + anhydrosis → r/o Horner's syndrome

4. Ask about the use of medications.
   a. Topical parasympatholytics (e.g. atropine) or sympathomimetics (e.g. phenylephrine, cocaine)
   b. Nebulized anticholinergics
   c. Scopolamine patches

IX. Ophthalmic Medications

A. Topical Anesthetics (no cross reaction between esters and amides) — are usually packaged in bottles with white or clear caps. They are used to facilitate the examination of patients with painful ocular processes and to allow the performance of ocular procedures (FB removal, irrigation, tonometry).

1. Esters
   a. Proparacaine 0.5%
      (1) Rapid onset
      (2) Lasts about fifteen minutes
   b. Tetracaine 0.5%
      (1) More irritating than proparacaine
      (2) Has a delayed onset but lasts about thirty minutes
   c. Cocaine — an excellent anesthetic, mydriatic and vasoconstrictor but it is not used in the ED since it softens the corneal epithelium, and subsequent minor trauma can produce desquamation
   d. Fluorocaine — a premixed combination of proparacaine and fluorescein dye that has thimerosal as a preservative (which may cause a local allergic conjunctivitis in susceptible patients)
   e. Benoxinate 0.4%
      (1) Onset 1 - 2 minutes
      (2) Duration of action 15 minutes

2. Amides — Dibucaine 0.1%

Note: These agents deprive the cornea of its normal protective reflexes and, in repeated doses, will retard healing of the cornea; they should NEVER be dispensed as pain medications for outpatient use.
B. **Mydriatics and Cycloplegics** — are packed in bottles with red caps.

1. **Mydriatics** — are topical sympathomimetic agents that paralyze the iris sphincter only, thereby producing pupillary dilation (mydriasis) without affecting accommodation. These agents are used diagnostically to facilitate evaluation of the internal ocular structures.
   a. Phenylephrine 2.5% — onset 15 - 60 mins., duration 3 - 4 hrs.
   b. Cocaine — produces mydriasis but is not used for this purpose.

2. **Cycloplegics** — are topical parasympatholytic agents that paralyze both the iris sphincter (mydriasis) and the ciliary muscle (cycloplegia). They are used therapeutically in the treatment of iritis and deep corneal abrasions to relieve pain by eliminating ciliary spasm.
   a. Use of cyclopentolate or tropicamide is preferred in the presence of acute inflammation since their effective duration of action is shorter than other agents which allows repetitive dosing to keep the patient comfortable.
      (1) Tropicamide — onset 15 - 20 mins., duration 4 - 6 hrs.
      (2) Cyclopentolate — onset 30 - 60 mins., duration ≤ 24 hrs.
   b. Homatropine is useful (short term) for subacute inflammation (iritis) caused by trauma or abrasion — onset is in 30 - 40 mins., duration 2 - 3 days.
   c. Scopolamine and atropine are more effective for chronic inflammation seen post-operatively and in severe uveitis; due to the long duration of action (3 - 5 days for scopolamine, 1 - 2 weeks for atropine) their use in the ED is limited.

   **Note:** These agents are contraindicated in patients with a Hx of glaucoma, evidence of increased intraocular pressure, presence of a shallow anterior chamber or suspicion of a ruptured globe.

C. **Miotics** — are packaged in bottles with green tops. Pilocarpine (2%) is used for treatment of acute angle closure glaucoma. By constricting the pupil, it pulls the iris back from its anterior position, thereby opening the angle and allowing egress of the aqueous humor.

D. **Topical Antibiotics** — many are supplied in both solution and ointment form.
   The solution (drops) are more rapidly absorbed and require more frequent instillation (Q 2 - 4 hrs.) Ointments, by contrast, have a longer duration of action and require less frequent administration (Q 4 - 6 hrs.) but transiently blur vision when applied.

1. Erythromycin — broad-spectrum
2. Sulfacetamide — broad-spectrum
3. Bacitracin — broad-spectrum
4. Quinolones (ciprofloxacin, norfloxacin, ofloxacin) — broad spectrum; drug of choice for monotherapy in the treatment of corneal ulcers; poor strep coverage; expensive.
E. **Antivirals** — interfere with viral DNA synthesis

1. Topical antivirals — are used in the treatment of herpes simplex keratoconjunctivitis; they shorten the treatment time and improve the rate of healing. Ophthalmologic consultation should be obtained before prescribing these agents (vidarabine, trifluridine).

2. Systemic antivirals — are used in consultation with ophthalmology and internal medicine in the treatment of herpes simplex keratitis, herpes zoster ophthalmicus and CMV retinitis.
   a. Acyclovir — useful for both herpes simplex keratitis and herpes zoster ophthalmicus.
   b. Famciclovir — used to treat herpes zoster ophthalmicus; requires less frequent dosing than acyclovir; reduces the duration of postherpetic neuralgia.
   c. Foscarnet — used in the treatment of CMV retinitis
   d. Ganciclovir — CMV retinitis

F. **Topical Steroids** — are used in the treatment of iritis to reduce inflammation. In general, they should not be prescribed in the ED; if they are prescribed, this should be done in consultation with an ophthalmologist and prompt (within 24 hrs.) ophthalmology follow-up is indicated.

1. Prednisolone acetate
2. Fluorometholone
3. Dexamethasone

G. **Hyperosmotic Agents** — are used in the treatment of acute angle closure glaucoma; they decrease intraocular pressure by decreasing the volume of fluid in the eye.

1. IV mannitol (20% sol.) — works rapidly + is very effective in the ED
2. Oral glycerol (50% sol.)* — may cause nausea
3. Oral isosorbide (45% sol.)

*Avoid the use of glycerol in diabetic patients; glycerol is metabolized to glucose and can produce hyperglycemia and ketosis in these patients.
H. **Carbonic Anhydrase Inhibitors** — are used in the treatment of acute angle closure glaucoma; they act to reduce intraocular pressure by decreasing the secretion of aqueous humor by the ciliary body.

1. Acetazolamide (IV or oral)
2. Neptazane (oral)
3. Trusopt (topical)

*Note:* These agents are sulfa compounds; sickle cell patients who present with hyphema are at risk for red cell sickling in the anterior chamber, further compromising aqueous outflow and increasing intraocular pressure.

I. **Topical Adrenergic Antagonists** — are used in the treatment of acute angle closure glaucoma; they reduce intraocular pressure by decreasing secretion of aqueous humor by the ciliary body.

1. These agents are either beta-blockers or alpha agonists. They are preferred for initial management because they do not produce the side effects associated with miotics.

2. Beta and alpha agonists both decrease secretion of aqueous humor and act synergistically to decrease IOP more rapidly than when either agent is used alone.
   a. The beta agonist most commonly used is timolol (a non-selective beta-blocker), but betaxolol is cardioselective and, therefore, preferred by some clinicians; they are packaged in bottles with yellow or blue caps.
   b. The alpha agonist most commonly used is apraclonidine; it is usually packaged in bottles with white caps.

3. All topical adrenergic antagonists can cause serious systemic side effects (especially cardiopulmonary).

X. **Tonometry**

A. Is the measurement of intraocular pressure (IOP) using a calibrated device called a tonometer.

B. It is indicated whenever the diagnosis of glaucoma is considered.
C. Available Devices

1. Schiötz impression tonometer — a handheld instrument that is available in most EDs and easy to use.

2. Goldman applanation tonometer — an instrument that is used in conjunction with a slit lamp.

3. TonoPen — a handheld device that gives a digital readout of the IOP and uses disposable covers.

4. Air-puff tonometer — an instrument that has the advantage of not requiring direct eye contact.

D. Use of the Schiötz tonometer

1. The patient is reclined and a drop of a topical anesthetic agent is placed in each eye; he/she is then instructed to keep both eyes wide open and fixed on an object.

2. Using the 5.5gm weight, the Schiötz tonometer is vertically lowered onto the cornea and a reading is taken.

3. The conversion table that accompanies the tonometer is then used to translate the tonometer scale reading to millimeters of mercury. The scale readings are inversely related to IOP; the lower the scale reading, the higher the IOP.

4. Scale readings of 4 - 8 are normal; they correspond with an intraocular pressure of 21 - 10.

5. If the scale reading is low (< 4), the tonometer is removed, additional weights are added to the plunger and the process is repeated.

E. Contraindications include the presence or suspicion of a penetrating ocular injury (absolute) or an eye infection (relative).

F. Use of the TonoPen

1. Same patient prep as for the Schiötz tonometer.

2. With a latex condom covering the plunger, the digital TonoPen is vertically lowered onto the cornea to assess IOP.

3. The digital display gives the IOP in mmHg; no conversion table is necessary.
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