

OPHTHALMOLOGY

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| | | | |
|--|-----------|--------------------------------------|-----------|
| COMMON COMPLAINTS | 3 | CONJUNCTIVA | 14 |
| Common Ocular Problems in the Elderly | | Pinguecula | |
| Common Ocular Problems in Contact Lens Wearer | | Pterygium | |
| THE OCULAR EXAMINATION | 5 | Subconjunctival Hemorrhage | |
| Vision Assessment | | Conjunctivitis | |
| Visual Fields | | Bacterial Conjunctivitis | |
| Pupils | | Viral Conjunctivitis | |
| Anterior Chamber Depth | | Chlamydia Conjunctivitis | |
| Extraocular Muscles | | Allergic Conjunctivitis | |
| External Examination | | Giant Papillary Conjunctivitis (GPC) | |
| Slit-Lamp Examination | | Vernal Conjunctivitis | |
| Tonometry | | SCLERA | 16 |
| Ophthalmoscopy/Fundoscopy | | Episcleritis | |
| OPTICS | 7 | Scleritis | |
| Emmetropia | | Scleromalacia Perforans | |
| Refractive Errors | | Blue Sclerae | |
| Myopia | | Staphyloma | |
| Hyperopia | | CORNEA | 17 |
| Astigmatism | | Foreign Bodies | |
| Presbyopia | | Corneal Abrasion | |
| Anisometropia | | Recurrent Erosions | |
| Refraction | | Corneal Ulcers | |
| Refractive Eye Surgery | | Herpes Simplex Keratitis | |
| THE ORBIT | 9 | Herpes Zoster | |
| Exophthalmos (Proptosis) | | Keratoconus | |
| Enophthalmos | | Arcus Senilis | |
| Preseptal Cellulitis | | Kayser-Fleischer Rings | |
| Orbital Cellulitis | | THE UVEAL TRACT | 19 |
| LACRIMAL APPARATUS & LYMPH NODES .. | 10 | Uveitis | |
| Lymph Nodes | | Anterior Uveitis | |
| Lacrimal Apparatus | | Posterior Uveitis | |
| Keratoconjunctivitis Sicca (Dry Eyes) | | GLOBE | 20 |
| Epiphora (Tearing) | | Endophthalmitis | |
| Dacryocystitis | | LENS | 21 |
| Dacryoadenitis | | Cataracts | |
| LIDS AND LASHES | 12 | Dislocated Lens | |
| Lid Swelling | | | |
| Ptosis | | | |
| Trichiasis | | | |
| Entropion | | | |
| Ectropion | | | |
| Hordeolum | | | |
| Chalazion | | | |
| Blepharitis | | | |
| Xanthelasma | | | |
| Lid Carcinoma | | | |

OPHTHALMOLOGY ... CONT.

| | | | |
|---|-----------|---|-----------|
| RETINA AND VITREOUS | 22 | OCULAR MANIFESTATIONS OF | 32 |
| Vitreous | | SYSTEMIC DISEASE | |
| Vitreous Hemorrhage | | HIV | |
| Retina | | Other Systemic Infections | |
| Central Retinal Artery Occlusion (CRAO) | | Diabetes Mellitus (DM) | |
| Branch Retinal Artery Occlusion | | Multiple Sclerosis (MS) | |
| Central Retinal Vein Occlusion (CRVO) | | Hypertension | |
| Retinal Detachment (RD) | | Amaurosis Fugax | |
| Retinitis Pigmentosa | | Hyperthyroidism/Graves' Disease | |
| Roth Spots | | Connective Tissue Disorder | |
| Age-Related Macular Degeneration (ARMD) | | Giant Cell Arteritis | |
| | | Sarcoidosis | |
| BLURRED OPTIC DISC MARGINS | 25 | STRABISMUS | 34 |
| Drusen | | Tropia | |
| Myelinated Nerve Fibres | | Phoria | |
| | | Paralytic Strabismus | |
| GLAUCOMA | 26 | Non-Paralytic Strabismus | |
| Primary Open Angle Glaucoma | | | |
| Primary Angle Closure Glaucoma | | PEDIATRIC OPHTHALMOLOGY | 36 |
| Secondary Open Angle Glaucoma | | Amblyopia | |
| Secondary Angle Closure Glaucoma | | Leukocoria | |
| Normal Pressure Glaucoma | | Nasolacrimal System Defects | |
| Congenital Glaucoma | | Ophthalmia Neonatorum | |
| | | Rubella | |
| PUPILS | 28 | OCULAR TRAUMA | 37 |
| Pupillary Light Reflex | | Blunt Trauma | |
| Dilated Pupil: Differential Diagnosis | | Penetrating Trauma | |
| Constricted Pupil: Differential Diagnosis | | Chemical Burns | |
| Relative Afferent Pupillary Defect (RAPD) | | Hyphema | |
| | | Blow Out Fractures | |
| NEURO-OPHTHALMOLOGY | 30 | Sympathetic Ophthalmia | |
| Visual Field Defects | | | |
| Bitemporal Hemianopsia | | OCULAR EMERGENCIES | 38 |
| Homonymous Hemianopsia | | DRUGS WITH OCULAR TOXICITY | 39 |
| Internuclear Ophthalmoplegia (INO) | | Topical Ocular Diagnostic Drugs | |
| Nystagmus | | Glaucoma Medications | |
| | | Topical Ocular Therapeutic Drugs | |
| INTRAOCULAR MALIGNANCIES | 31 | OCULAR DRUG TOXICITY | 40 |
| Malignant Melanoma | | REFERENCES | 40 |
| Retinoblastoma | | | |
| Metastases | | | |

COMMON COMPLAINTS

Persistent Loss of Vision

- gradual (weeks to months)
 - refractive error
 - cataracts
 - diabetes
 - macular degeneration
 - glaucoma (chronic)
 - intracranial compressive lesion
 - tumour infiltration
 - toxic degeneration
- acute (minutes to days)
 - vascular/ischemia
 - cortical blindness
 - central retinal vein/artery occlusion (CRVO/CRAO)
 - vitreous hemorrhage
 - optic neuropathy
 - giant cell (temporal) arteritis
 - anterior ischemic optic neuropathy (AION)
 - optic neuritis
 - multiple sclerosis (MS)
 - papillitis or retrobulbar neuritis
 - retinal detachment
 - acute glaucoma
 - acute iritis

Transient Loss of Vision (Amaurosis Fugax)

- transient ischemic attack (TIA), microemboli
- migrainous spasm of artery
- hypertension

Floaters

- physiologic; vitreous syneresis
- vitreous hemorrhage
- retinal detachment

Flashing Lights

- vitreous traction
- retinal tear/detachment
- migraine

Ocular Pain

- corneal abrasion, corneal ulcer, foreign body
- acute angle closure glaucoma
- acute uveitis
- scleritis, episcleritis
- optic neuritis
- differentiate from ocular ache: eye fatigue/asthenopia

Photophobia (Light Sensitivity)

- iritis
- meningitis, encephalitis
- light dispersion by mucus, lens, corneal opacities
- retinal degeneration
- acute glaucoma

Diplopia (Double Vision)

- binocular diplopia: strabismus, CN paresis (III,IV,VI), muscle entrapment
- monocular diplopia: dislocated lens, cataract, corneal scar

COMMON COMPLAINTS ... CONT.

Red Eye

Table 1. Differential Diagnosis of Red Eye

| | Conjunctivitis | Acute Iritis | Acute Glaucoma | Angle Closure Keratitis |
|--------------------|---|-----------------------------|----------------------------|----------------------------|
| Discharge | Bacteria: pus Virus: serous Allergy: mucous | No | No | Profuse tearing |
| Pain | No | ++ (tender globe) | ++++ With nausea | +++ With blinking |
| Photophobia | No | ++++ | + | ++ |
| Vision | Normal | Reduced (cloudy aqueous) | Reduced (corneal edema) | Varies with site of lesion |
| Pupil | Normal | Smaller | Fixed in mid dilation | Same or smaller |

Clinical Pearl

- All red eyes are not necessarily conjunctivitis.

Table 2. Additional Features of Red Eye

| | Conjunctivitis | Acute Iritis | Acute Angle Closure Glaucoma |
|-----------------------------------|---------------------------------|----------------------|------------------------------|
| Injection | Palpebral+bulbar (limbal palor) | Ciliary flush | Diffuse |
| Intraocular Pressure (IOP) | Normal | Lower | Increased |
| Ant.chamber | Normal | Cells/flare | Shallow |
| Cornea | Normal | Keratic precipitates | Steamy |
| Other | Preauricular node (if viral) | Synechia | Nausea/vomiting |

- other causes of red eye
- adnexal and lacrimal system
 - hordeolum/stye
 - chalazion
 - blepharitis
 - dacryocystitis
 - canaliculitis
 - dacryoadenitis
 - preseptal cellulitis
 - orbital cellulitis
 - conjunctiva
 - subconjunctival hemorrhage
 - pterygium, pinguecula
 - sclera
 - episcleritis
 - scleritis
 - cornea
 - abrasion
 - ulcer
 - foreign body
 - endophthalmitis

COMMON COMPLAINTS ... CONT.

COMMON OCULAR PROBLEMS IN THE ELDERLY

- cataracts
- age-related macular degeneration (ARMD)
- epiphora
 - overflow of tears
 - due to dry eyes, entropion, ectropion, trichiasis
- neoplasia
- vascular disease: CRVO, CRAO, giant cell arteritis
- dry eyes
- ptosis

COMMON OCULAR PROBLEMS IN CONTACT LENS WEARER

- corneal abrasion
- superficial punctate keratitis
- giant papillary conjunctivitis
- sterile infiltrates (immunologic)
- infected ulcers (*Pseudomonas*, *S. aureus*)

THE OCULAR EXAMINATION

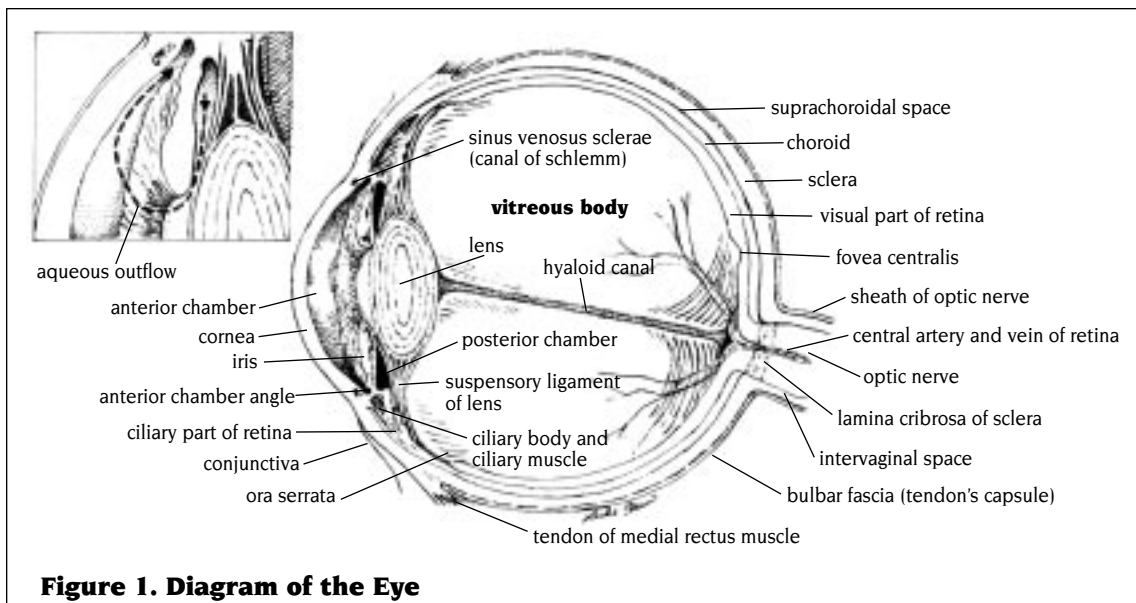


Figure 1. Diagram of the Eye

Drawing by Luke Itani

VISION ASSESSMENT

- always note best corrected vision first (e.g. with glasses if possible), especially in emergency room
- test both near and distance vision
- pinhole test will improve vision with most refractive errors

Visual Acuity (VA) Far

- Snellen Fraction = $\frac{\text{testing distance (usually 6 metres or 20 feet)}}{\text{smallest line patient can read on the chart}}$
e.g. 20/40 -2 (missed two letters of 20/40 line)
- OD = RIGHT EYE, OS = LEFT EYE, OU = BOTH EYES
- Hierarchy for low vision - Snellen acuity - count fingers - hand motion - light perception - no light perception
- Legal blindness is 20/200 or < 10 degrees of visual field in better eye
- Minimum visual acuity to operate an automobile is 20/40 in the better eye

Visual Acuity (VA) Near

- use pocket vision chart (e.g. Rosenbaum)
- record number and testing distance (usually 30 cm)
e.g. J2 @ 30cm

Visual Acuity for Children, Illiterate Adults or Dysphasics with CVA

- Sheridan-Gardiner matching test (most 4 year olds able to do it)
- illiterate "E" chart (patient states direction of "E")

Infant Visual Acuity

- 6-12 months - 20/120
- 1-2 years - 20/80
- 2-4 years - 20/20

VISUAL FIELDS

- test visual field quadrants grossly by confrontation
- automated field testing (e.g. Humphrey, Goldmann), or Tangent Screen

PUPILS

- examine pupils with respect to
 - equality, size, shape
 - reactivity to light (both direct and consensual)
- test for relative afferent pupillary defect (RAPD)
 - use "swinging flashlight test" (use ophthalmoscope with "+4" setting especially with dark brown iris)
- test pupillary constriction portion of near reflex
 - bringing object from far to near results in
 - lens accommodation
 - eye convergence
 - pupil constriction
 - must use reduced illumination or pupillary constriction will be produced

ANTERIOR CHAMBER DEPTH

- shine light tangentially from temporal side
- shallow = $> 2/3$ of nasal iris in shadow

EXTRAOCULAR MUSCLES

Alignment

- examine in primary position of gaze (e.g. straight ahead)
- Hirschberg test (shine light into patient's eyes from 30 cm away)
 - corneal light reflex should be symmetric and near centre of each cornea
- strabismus testing as indicated - see Strabismus section

Movement

- examine movement of eyeball through the nine diagnostic positions of gaze (with six muscles responsible for extra-ocular movement (EOM))
- determine if diplopia is present in any position of gaze
- observe for nystagmus (horizontal and vertical)

EXTERNAL EXAMINATION

- the four Ls
 - lymph nodes
 - lacrimal apparatus
 - lids
 - lashes

SLIT-LAMP EXAMINATION

- systematically examine all structures of the anterior segment
 - lids and lashes, including upper lid eversion if necessary
 - conjunctiva and sclera
 - cornea
 - anterior chamber
 - iris
 - lens
- also examine with
 - fluorescein staining: (water-soluble dye stains de-epithelialized cornea green), with cobalt blue filter
 - Rose Bengal dye (stains devitalized corneal epithelium)
- can examine structures in the posterior segment with special lenses (78D, 90D)

THE OCULAR EXAMINATION ... CONT.

TONOMETRY

- measurement of intraocular pressure
- normal range is 8-21 mm Hg
- commonly measured by
 - indentation (Schiotz or Tonopen)
 - applanation (Goldmann) – gold standard
 - non-contact (air puff)
- use topical anesthetic for Schiotz, Goldmann, Tonopen

OPHTHALMOSCOPY/FUNDOSCOPY

- examination of the anterior segment
 - fluorescein and cobalt blue filter and +20 lens
 - corneal opacity
 - cataract
 - the red reflex
- examination of the posterior segment of the eye
 - vitreous
 - optic disc (colour, cup, margins, cup/disc ratio)
 - nasal retinal vessels
 - retina, macula (temporal)
- best with pupillary dilatation (e.g. tropicamide) (see Table 6)
- contraindications to dilatation
 - narrow anterior chamber angles
 - neurologic abnormality requiring pupillary evaluation
 - iris supported anterior chamber lens implant (square pupil)

OPTICS

- main refractive mechanisms are: cornea (2/3), lens (1/3)

EMMETROPIA

- no refractive error
- image of distant objects focused on the retina without accommodation

REFRACTIVE ERRORS (see Figure 2 and Figure 3)

- distant light is not focused on retina, without accommodation
- three types: myopia, hyperopia, and astigmatism

MYOPIA

- "nearsightedness"
- to remember: LMN (**L**ong eyeball is **M**yopic, requiring negative/concave lens, and is **N**earsighted)

Pathophysiology

- globe too long relative to refractive mechanisms or refractive mechanisms too strong
- image of distant object falls in front of retina without accommodation → blurring of distant vision

Presentation

- usually presents in 1st or 2nd decade, stabilizes in 2nd and 3rd decade;
rarely begins after 25 years except in diabetes or cataracts
- blurring of distance vision
- near vision usually unaffected

Complications

- retinal degeneration and detachment
- chronic open angle glaucoma
- complications not prevented with refractive correction

Management

- correct with concave negative (–) spectacles or contact lenses, which diverge light rays
- refractive eye surgery - see below

HYPEROPIA

- "farsightedness"
- hyperopia may be developmental, or may be due to any cause which shortens the eyeball
- to quantitate hyperopia, cycloplegic drops are used to prevent accommodation

Pathophysiology

- globe too short or refractive mechanisms too weak
- image of distant object falls behind retina without accommodation
- person will accommodate to try to bring image onto retina

Presentation

- ❑ youth: usually do not require glasses (still have sufficient accommodative ability to focus image on retina), +/- accommodative esotropia (see Strabismus section)
- ❑ 30s: blurring of near vision due to decreased accommodation, may need reading glasses
- ❑ > 50s: blurring of distance vision due to severely decreased accommodation

Complications

- ❑ angle closure glaucoma, particularly in later life as lens enlarges

Management

- ❑ when symptomatic, correct with convex positive (+) lenses, which converge light rays
- ❑ refractive eye surgery - see below

ASTIGMATISM

- ❑ light rays not refracted uniformly in all meridians
- ❑ due to non-spherical surface of cornea or non-spherical lens (e.g. football shaped)
- ❑ regular astigmatism: curvature is uniformly different in meridians at right angles to each other
- ❑ irregular astigmatism: distorted cornea, due to injury or keratoconus (cone-shaped cornea)

Management

- ❑ correct with cylindrical lens, toric contact lens, arcuate keratotomy or refractive eye surgery (see below)

PRESBYOPIA

- ❑ decreased ability of eye to accommodate with aging (decrease in lens elasticity, NOT a refractive error)
- ❑ experienced by emmetropes as well as patients with refractive errors
- ❑ normal decline in near vision with age (> 40 years) with distance spectacles in place

Presentation

- ❑ if initially emmetropic, starts holding things further away to read, but distance vision unaffected
- ❑ if initially myopic, remove distance glasses to read
- ❑ if initially hyperopic, symptoms of presbyopia occurs earlier; the hyperope needs distance glasses in later decades

Management

- ❑ correct vision with convex positive (+) lens for reading
- ❑ reading lens will blur distance vision; options are half-glasses or bifocals

ANISOMETROPIA

- ❑ difference in refractive error between eyes
 - second most common cause of amblyopia in children

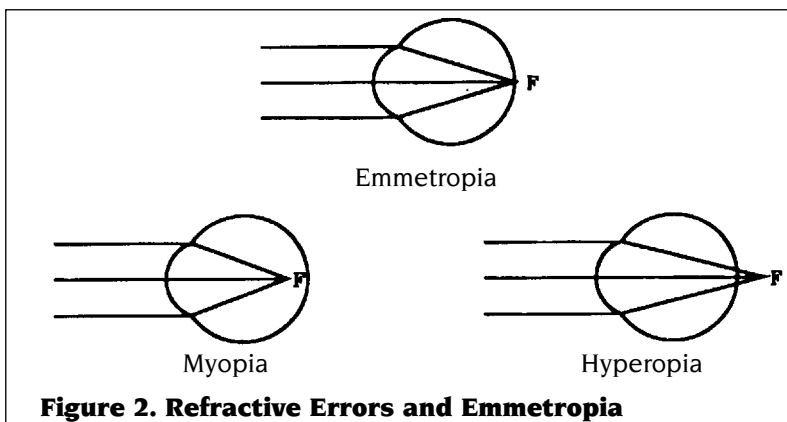


Figure 2. Refractive Errors and Emmetropia

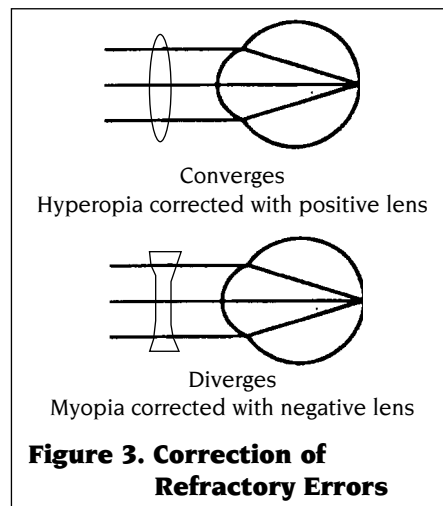


Figure 3. Correction of Refractive Errors

REFRACTION

- technique of determining the lenses needed to correct the optical defects of the eye (ametropia)
- two techniques used
 - 1) Flash/Streak Retinoscopy
 - refractive error determined objectively by use of retinoscope and lenses
 - 2) Manifest (Acceptance)
 - subjective trial of lenses used to refine retinoscopy findings
 - a typical lens prescription would contain
 - sphere power in D (diopters)
 - cylinder power in D to correct astigmatism
 - axis of cylinder (in degrees)
 - add (reading lens) for presbyopes
 - e.g. $-1.50 + 1.00 \times 120$ degrees, add +2.00

REFRACTIVE EYE SURGERY

- permanently altering the refractive properties of the cornea
- used for correction of myopia, hyperopia, and astigmatism
- most commonly using excimer laser system
- ablation of the corneal surface - photorefractive keratectomy (PRK)
- ablation of the stroma below a flap of corneal tissue (LASIK)

THE ORBIT

EXOPHTHALMOS (PROPTOSIS)

- eyeball protrusion

Etiology

- hyperthyroidism: Graves' disease (unilateral or bilateral, the most common cause in adults)
- orbital cellulitis (unilateral, most common cause in children)
- primary or secondary orbital tumours
- orbital/retrobulbar hemorrhage
- cavernous sinus thrombosis or fistula
- sinus mucoceles

Diagnosis

- exophthalmometer (Hertel): measure at lateral canthi
- CT head

ENOPHTHALMOS

- retracted globe
- often due to "blow-out" fracture (see Ocular Trauma section)

PRESEPTAL CELLULITIS

- infection of soft tissue anterior to orbital septum

Etiology

- stye, chalazion, acute meibomitis
- insect bite
- secondary to lid laceration or lacrimal system inflammation

Symptoms and Signs

- tender and erythematous lids
- may have adenopathy and fever
- normal VA, pupil, EOMs
- no exophthalmos or RAPD

Management

- topical and systemic antibiotics
- warm compress

ORBITAL CELLULITIS

- inflammation of orbital contents posterior to orbital septum
- common in children, but also in the aged and immunocompromised

Etiology

- secondary to sinusitis, facial and/or tooth infections and trauma

THE ORBIT ... CONT.

Symptoms and Signs

- RED FLAG – ophthalmoplegia (because of pain on ocular movement)
- lids swollen shut, chemosis (conjunctival swelling)
- exophthalmos
- decreased VA
- RAPD due to optic disc edema may occur
- adenopathy and fever

Management

- admit, IV antibiotics, blood cultures and orbital CT
- serious complications exist e.g. cavernous sinus thrombosis, meningitis and blindness
- surgical evacuation of abscess if it develops

Table 3. Differences Between Preseptal and Orbital Cellulitis

| Finding | Preseptal Cellulitis | Orbital Cellulitis |
|----------------------|----------------------|--------------------------------|
| Fever | Present | Present |
| Lid edema | Moderate to severe | Severe |
| Chemosis | Absent or mild | Moderate or marked |
| Proptosis | Unusual | Present |
| Pain on eye movement | Absent | Present |
| Ocular mobility | Normal | Decreased |
| Vision | Normal | Diminished vision +/- diplopia |
| RAPD | Absent | May be seen |
| Leukocytosis | Minimal or moderate | Marked |
| Adenopathy | Absent | May be seen |
| ESR | Normal or elevated | Very elevated |
| Additional findings | Skin infection | Sinusitis; dental abscess |

LACRIMAL APPARATUS AND LYMPH NODES

LYMPH NODES

- temporal conjunctival lymphatics drain to preauricular and parotid nodes
- nasal conjunctival lymphatics drain to submandibular nodes
- enlarged nodes significant for infectious etiology, especially viral or chlamydial conjunctivitis

LACRIMAL APPARATUS

- tear film made up of three layers
 - an outer oily layer, secreted by the meibomian glands
 - a middle watery layer, constant secretion from conjunctival glands and reflex secretion by lacrimal gland with ocular irritation or emotion
 - an inner mucous layer, secreted by conjunctival goblet cells

KERATOCONJUNCTIVITIS SICCA (DRY EYES)

Etiology

- with aging, tear production normally decreases
- lid abnormality (ectropion), decreased blinking e.g. CN VII palsy
- systemic diseases: rheumatoid arthritis, Sjögren's syndrome, sarcoidosis, amyloidosis, leukemia, lymphoma
- vitamin A deficiency, especially in malnourished areas
- post-cataract surgery
- medications: anticholinergics, diuretics, antihistamines

Symptoms and Signs

- dry eyes, red eyes, blurred vision, overflow tearing

Diagnosis

- slit-lamp exam: decreased tear meniscus, decreased tear break up time (BUT), superficial punctate keratitis (SPK)
- revealed by fluorescein staining, rose bengal staining
- Schirmer test: measures tear quantity on surface of eye in 5 minute time period (< 10 mm of strip wetting in 5 minutes is considered a dry eye)

Complications

- erosions and scarring of cornea

Management

- artificial tears and ointments
- punctal occlusion
- tarsorrhaphy (sew lids together)
- treat underlying cause

EPIPHORA (TEARING)

Etiology

- emotions
- ocular irritation/inflammation (including entropion, trichiasis)
- blocked tear outflow (ectropion; punctal, canalicular or nasolacrimal duct (NLD) obstruction)
 - aging
 - infection (dacryocystitis)
 - rhinitis
 - in infants: failure of NLD opening
- paradoxical lacrimation (crocodile tears)
 - excessive tearing while eating
 - sequelae to Bell's palsy, due to aberrant regeneration of CN VII
 - nerve fibres that formerly went to the salivary glands regrow to the lacrimal gland

Diagnosis

- history and observation of lids
- fluorescein dye put in eye, examine for punctal reflux by pressing on canaliculi
- irrigate through punctum into nose, noting resistance/reflux

Management

- lid repair for ectropion/entropion
- punctal irrigation
- NLD probe (infants)
- tube placement: temporary (Crawford) or permanent (Jones)
- surgical: dacryocystorhinostomy (DCR) = joining the lacrimal sac to the nasal mucosa, thus restoring lacrimal drainage

DACRYOCYSTITIS

- infection of the lacrimal sac
- usually due to obstruction of the nasolacrimal duct
- usually very young children (due to failure of NLD opening) or in persons > 40 years old
- commonly associated with *S. aureus* or rarely β -hemolytic streptococcus infection in acute setting; *S. pneumoniae* in chronic infections

Symptoms and Signs (see Colour Atlas OP1)

- tearing and discharge
- inflammation, pain, swelling over lacrimal sac at inner canthus
- pressure on the lacrimal sac may extrude pus through the punctum
- in the chronic form, tearing may be the only symptom

Management

- warm compresses, nasal decongestants
- in newborns - gentle massage over sac, +/- probing
- in adults - local or systemic antibiotics +/- irrigation (refer to ophthalmologist)
- if chronic, treatment is surgical: dacryocystorhinostomy (DCR)

DACRYOADENITIS

- very rare, usually children
- inflammation of the lacrimal gland (outer third of upper eyelid)
- acute causes: (infectious etiology) mumps, measles, influenza in children, gonorrhoea in adults
- chronic causes: lymphoma, leukemia, sarcoidosis, tuberculosis

Symptoms and Signs (see Colour Atlas OP9)

- pain, swelling, redness of the outer region of the upper eyelid

Management

- +/- systemic antibiotics
- incision and drainage if required

LIDS AND LASHES

LID SWELLING

Etiology

- commonly due to allergy, with shrivelling of skin between episodes
- dependent edema on awakening (e.g. CHF, renal or hepatic failure)
- orbital venous congestion due to mass or cavernous sinus fistula
- dermatochalasis: loose skin due to aging
- lid cellulitis, hypothyroidism (e.g. myxedema), trauma (e.g. bruising), chemosis
- adenoviral conjunctivitis

PTOSIS

- drooping of upper lid > 2 mm below the superior corneal margin

Etiology

- congenital: very rare
- CN III palsy
 - complete: eye is down and out, mydriasis (pupil dilation) (e.g. external compression)
 - incomplete: pupil-sparing (e.g. diabetes mellitus)
- Horner's syndrome
 - ptosis, miosis, anhidrosis
 - loss of sympathetic innervation causing Muller muscle paralysis
 - see Pupils section for causes
- myasthenia gravis (see Neurology Chapter)
 - easy fatiguability, with ptosis and diplopia
 - diagnose with Tensilon test
- myogenic: disinsertion or dehiscence of levator aponeurosis
 - most common cause of acquired ptosis in geriatrics
- pseudoptosis (e.g. dermatochalasia, enophthalmos, contralateral endocrine exophthalmos)
- trauma, infection (e.g. cellulitis)
- mechanical
 - eyelid prevented from opening completely by mass or scarring

Management

- treat underlying cause
- diabetic CN III palsy may resolve spontaneously
- surgical correction if interferes with vision or for cosmesis

TRICHIASIS

- eyelashes turn inward causing corneal irritation +/- ulceration
- patient complains of irritation, tearing, mucous discharge

Management

- pluck eyelash, electrolysis, surgery to remove eyelash +/- destroy hair follicles

ENTROPION

- lid margin turns in towards globe
- most commonly affects lower lid
- symptoms: tearing, dry eye, cosmetic
- may cause abrasions with secondary corneal scarring

Etiology

- involutional (aging)
- scar contraction (burns, surgery)
- orbicularis oculi muscle spasm
- congenital

Management

- lubricants, evert lid with tape, surgery

ECTROPION

- eversion of lower lid
- often bilateral
- symptoms: dry eye, tearing, cosmetic
- may cause exposure keratitis

Etiology

- weak orbicularis oculi (aging)
- CN VII palsy
- scarring (burns, surgery)
- mechanical (tumour, herniated fat)

Management

- upward massage with ointment or surgery
- lubricants

HORDEOLUM 'STYE'

- infection of the glands of the eyelid, usually with *S. aureus*
- painful, red swelling of lid
- internal
 - acute infection of meibomian gland (Meibomitis)
 - chalazion in chronic stage
- external
 - styne, pimple
 - acute infection of hair follicle or of glands of Zeis or Moll

Management

- warm compresses, lid care
- topical antibiotics
- cellulitis may develop (necessitates systemic antibiotics)
- usually resolves in 2-5 days

CHALAZION (see Colour Atlas OP10)

- due to chronic sterile granuloma of a meibomian gland (following acute meibomitis), usually pointing towards the conjunctiva
- painless, often subsides
- differential diagnosis: basal cell carcinoma, sebaceous cell adenoma, meibomian gland carcinoma
- differentiate from hordeolum by absence of acute inflammatory signs

Management

- warm compresses
- surgical incision and curettage if needed

BLEPHARITIS (see Colour Atlas OP11)

- chronic inflammation of lid margins
- symptoms: redness of lid margins, scaling and discharge with misdirection or loss of lashes, burning and/or itching of lids
- most common types
 - *S. aureus*
 - ulcerative, with dry scales
 - seborrheic
 - no ulcers, with greasy scales
- association with allergy, acne rosacea, seborrheic dermatitis

Complications

- recurrent chalazia
- conjunctivitis
- keratitis
- corneal ulceration and neovascularization (late sequelae from trichiasis)

Management (*S. aureus*)

- warm compresses and lid hygiene
- cleansing with dilute baby shampoo
- topical or systemic antibiotics as needed

XANTHELASMA

- lipid deposits in histiocytes in dermis of lids
- frequently near inner canthus, may be bilateral
- appears as pale, slightly raised yellowish patches or streaks
- sometimes associated with increased serum cholesterol
- of more concern in young; common in elderly

Management

- surgical removal for cosmesis only, recurrences common

LID CARCINOMA

Etiology

- basal cell carcinoma (95%)
 - spread via local invasion
 - rodent ulcer, indurated base with pearly rolled edges
- squamous cell carcinoma (5%)
 - spread via local invasion, may also spread to nodes and metastasize
 - ulceration, keratosis of lesion
- sebaceous cell carcinoma (rare)
 - can present as non-resolving blepharitis (unilateral)
 - highly invasive, metastasize

Management (see Dermatology/Plastic Surgery Chapter)

- surgical excision
- irradiation optional for basal cell, sebaceous cell
- biopsy to confirm diagnosis

CONJUNCTIVA

- a mucous membrane/epithelium
- bulbar conjunctiva: covers sclera to the corneal limbus
- palpebral conjunctiva: covers inside of lids

PINGUECULA

- yellow nodule of hyaline and elastic tissue
- commonly deep to conjunctiva adjacent to the limbus
- associated with sun and wind exposure
- common, benign, usually nasal

Management

- does not require treatment, surgical excision rarely indicated
- if ulcerated, topical antibiotics may be required

PTERYGIUM (see Colour Atlas OP8)

- fleshy triangular encroachment of epithelial tissue onto the cornea (extension of pinguecula)
- usually nasal and bilateral
- associated with wind, ultraviolet light exposure

Management

- surgery if threatens visual axis, causes irritation, or for cosmesis
- may recur (10% with conjunctival grafts) and require repeated surgery
- may decrease recurrence with conjunctival autograft or mitomycin C (anti-neoplastic) drops

SUBCONJUNCTIVAL HEMORRHAGE

- blood beneath the conjunctiva
- painless, normal vision
- causes: idiopathic, valsalva, trauma, bleeding disorders
- if bilateral and recurrent, rule out blood dyscrasias, hypertension, HIV, Kaposi sarcoma

Management

- reassurance as it resorbs in 2-3 weeks, discourage rubbing

CONJUNCTIVITIS

Etiology (see Table 4)

- tired or dry eyes
- allergy: pollutants, wind dust
- infection: bacterial, viral, chlamydial, fungal, rickettsial, parasitic
- chemical
- irradiation associated with systemic disease
- immune reaction e.g. giant papillary conjunctivitis especially in contact lens wearer
- secondary to dacryocystitis or canaliculitis

Symptoms and Signs

- gritty sensation, purulent discharge, crusts on lids upon awakening
- inflamed and injected conjunctiva usually with limbal pallor
- follicles (common)
 - pale lymphoid elevations of the conjunctiva
 - found in viral and chlamydial conjunctivitis
- papillae
 - vascularized elevations of the palpebral conjunctiva (contain PMNs)
 - found in giant papillary conjunctivitis (GPC) and vernal conjunctivitis

BACTERIAL CONJUNCTIVITIS – ‘ACUTE PINK EYE’ (see Colour Atlas OP13)

- often has a purulent white-yellow discharge, and less commonly papillae
- causes: *S. aureus*, *S. pneumoniae*, and *H. influenzae*
- may also be due to *N. gonorrhoea* (in neonates and sexually active people), *Chlamydia* is the most common cause in neonates
- classification
 1. Hyperacute: *N. gonorrhoea*, *N. meningitis*
 2. Acute: *H. egyptius*, *S. pneumoniae*
 3. Subacute: *H. influenzae*

Management

- topical antibiotics
- systemic antibiotics if indicated
- course – self-limited – 10-14 days if no treatment, 1-3 days with treatment

VIRAL CONJUNCTIVITIS (see Colour Atlas OP14)

- watery discharge (mucopurulent common), follicles, subepithelial corneal infiltrates may occur at 10-14 days
- associated with cold symptoms, (recent upper respiratory tract infection (URTI) history)
- preauricular node often palpable and tender
- initially unilateral, often progresses to the other eye
- cause: adenovirus

Management

- self-limiting – (7 – 10 days or longer)
- adenovirus is highly contagious therefore proper hygiene is very important

CHLAMYDIAL CONJUNCTIVITIS

- caused by *Chlamydia trachomatis* (various serotypes)
- affects neonates on day 3-5, sexually active people
- causes trachoma, inclusion conjunctivitis, lymphogranuloma venereum (L1, L2, L3)

Trachoma

- severe keratoconjunctivitis
- leading cause of blindness in the world
- papillae and follicles on superior palpebral conjunctiva
- conjunctival scarring leads to entropion, causing trichiasis, corneal abrasions +/- ulceration and scarring
- keratitis leads to superior vascularization (pannus) and corneal scarring
- treatment: systemic tetracycline

Inclusion Conjunctivitis

- follicles with occasional keratitis
- most common cause of conjunctivitis in newborns
- prevention: topical erythromycin at birth
- treatment: topical tetracycline and systemic erythromycin

ALLERGIC CONJUNCTIVITIS (see Colour Atlas OP15)

- intermittent
- chemosis/injection with itching and burning
- mucous discharge, lid edema, palpebral conjunctival papillae

Management

- avoid irritants
- cold compresses
- topical medications: decongestant/antihistamines (e.g. Albalon-A), mast cell stabilizer (e.g. Opticrom) combinations (e.g. Patanol), NSAIDs (e.g. Acular), steroids (not used in primary care)

GIANT PAPILLARY CONJUNCTIVITIS (GPC)

- immune reaction to mucous debris on lenses in contact lens wearers
- large papillae form on superior palpebral conjunctiva

Management

- decrease wearing time, clean lenses thoroughly
- switch to disposable lenses or hard lenses
- topical mast cell stabilizer
- if refractory to treatment stop using contacts

VERNAL CONJUNCTIVITIS

- allergic condition, seasonal (warm weather)
- large papillae on superior palpebral conjunctiva may cause corneal abrasions
- occurs in first decade, may last for many years

Management

- topical and/or systemic antihistamines
- topical NSAIDs, mast cell stabilizer, steroids (not in primary care)

SCLERA

- sclera is the white fibrous outer protective coat of the eye
- continuous with the cornea anteriorly and the dura of the optic nerve posteriorly
- made of avascular collagen, biochemically similar to joint cartilage, thus rheumatoid conditions may affect sclera and episclera
- episclera is a thin layer of vascularized tissue covering the sclera anteriorly

EPISCLERITIS

- usually unilateral; simple or nodular
- non-specific immune response to irritants

Etiology

- mostly idiopathic
- associated with rheumatoid arthritis (RA), gout, Sjögren's syndrome, SLE, herpes zoster, tuberculosis, syphilis or coccidioidomycosis in one third of cases
- more frequent in women than men (3:1)

Symptoms and Signs (see Colour Atlas OP16)

- localized, elevated, segmental hyperemia, pink/purple-coloured globe
- pain, swelling, tenderness, photophobia, lacrimation
- topical vasoconstrictor reduces redness
- lasts for weeks, typically recurs

Management

- generally self limited
- chilled artificial tears
- topical corticosteroids for 3-5 days if painful (prescribed and monitored by ophthalmologist)

SCLERITIS

- uni- or bilateral; sectoral, diffuse, nodular or necrotizing
- anterior scleritis: engorgement of vessels deep to conjunctiva
- may cause scleral melt, corneal ulceration, secondary glaucoma
- posterior scleritis: may cause exudative retinal detachment
- usually with episcleral involvement

Etiology

- over half are a manifestation of systemic disease
 - autoimmune e.g. SLE, RA
 - granulomatous e.g. TB, sarcoidosis, syphilis
 - metabolic e.g. gout, thyrotoxicosis
 - infectious e.g. *S. aureus*, *S. pneumoniae*, *P. aeruginosa*, HSV, herpes zoster
- chemical or physical agents e.g. thermal, alkali or acid burns
- idiopathic

Symptoms and Signs (see Colour Atlas OP17)

- severe pain and tenderness, more severe than in episcleritis
- pain is best indicator of disease progression
- conjunctival injection/scleral engorgement
- bluish-red hue (in contrast to brighter red of episcleritis)
 - topical vasoconstrictors will decrease redness of episcleritis, facilitating examination of scleritis
- +/- decrease in visual acuity

Management

- topical steroids, not used in primary care (may thin sclera)
- systemic NSAIDs or steroids

SCLEROMALACIA PERFORANS

- associated with severe rheumatoid arthritis (rare)
- thinning of the sclera may lead to uveal dehiscence and globe rupture with minor trauma
- prognosis poor, avoid steroids
- very gentle examination of eye (Schiotz tonometry contraindicated)

BLUE SCLERAE

- rare, uveal pigment seen through thin sclera
- associated with: collagen vascular diseases (CVD) (e.g. osteogenesis imperfecta, Ehlers-Danlos syndrome, Marfan's syndrome), prolonged use of corticosteroids
- may occur in normal newborns

STAPHYLOMA

- localized ballooning of thinned sclera – anteriorly, equatorially or posteriorly
- seen in rheumatoid arthritis, high myopia, glaucoma, trauma

CORNEA

- function
 - transmission of light
 - refraction of light (2/3 of total refractive power)
 - barrier against infection, foreign bodies
- transparency due to avascularity, uniform structure and deturgescence
- 5 layers: epithelium, Bowman's membrane, stroma, Descemet's membrane, endothelium
- extensive sensory fibre network (V1 distribution); therefore abrasions and inflammation (keratitis) are very painful
- two most common corneal lesions: abrasions and foreign bodies

FOREIGN BODIES (see Colour Atlas I5)

- RED FLAG -foreign body behind lid may cause multiple vertical epithelial abrasion due to blinking
- tearing, photophobia, foreign body sensation
- detected with fluorescein staining viewed under cobalt blue light of ophthalmoscope or slitlamp

Complications

- scarring, infection, rust ring, secondary iritis

Management

- remove under magnification using local anesthetic and sterile needle or refer to ophthalmologist (depending on depth and location)
- topical antibiotics
- cycloplegia (paralysis of ciliary body = loss of accommodation) if iritis already present
- no pressure patch if from contact lens wear
- must see the next day and check for secondary iritis and infection

CORNEAL ABRASION

Etiology

- trauma (e.g. fingernails, paper, twigs), contact lens

Symptoms and Signs (see Colour Atlas OP2)

- pain on blinking, conjunctival injection, tearing, photophobia (after 24-48 hours)
- de-epithelialized area stains green with fluorescein dye
- pain relieved with topical anesthetic

Complications

- infection, ulceration, recurrent erosion, secondary iritis

Management

- topical antibiotics
- +/- short acting topical cycloplegics (rest iris for comfort - prevents secondary iritis and posterior synechiae)
- patch affected eye for comfort only if abrasion is large
- NEVER patch abrasion secondary to contact lens wear (prone to *Pseudomonas* infection)
- NEVER give topical analgesics (except to facilitate examination) - impedes epithelial healing (unless needed to facilitate examination)
- systemic analgesics as needed
- most abrasions clear within 24-48 hours
- bilateral occlusion gives fastest healing

RECURRENT EROSIONS

- localized area of superficial corneal edema +/- de-epithelialization where the epithelium fails to properly adhere to the underlying Bowman's membrane, therefore epithelial cells detach easily

Etiology

- previous injury with incomplete healing
- corneal dystrophy
- spontaneous, idiopathic

Symptoms and Signs

- patient usually awakes with pain in morning when poorly adherent epithelium is dislodged during first eye opening of the day
- examination reveals localized epithelial irregularity

Management

- as for corneal abrasion
- bandage contact lens (to protect corneal epithelium), bed rest
- topical hypertonic 2% or 5% sodium chloride solution to dehydrate epithelial edema
- anterior stromal needle puncture or YAG laser for chronic recurrence
- excimer laser superficial keratectomy

Table 4. Corneal Abrasion vs. Corneal Ulcer

| | Abrasion | Ulcer |
|-------------------|-----------------------|------------------------|
| Time course | Acute (hours) | Subacute (days) |
| History of trauma | Yes | Yes |
| Cornea | Clear | White, necrotic |
| Iris detail | Clear | Obscured |
| Corneal thickness | Normal | May have crater effect |
| Extent of lesion | Limited to epithelium | Extension into stroma |

CORNEAL ULCERS

Etiology

- secondary to corneal abrasions, conjunctivitis, blepharitis, usually bacterial, rarely viral or fungal
- contact lens use (50% of ulcers)
- marginal ulcers are mostly due to staphylococcal toxins from associated blepharitis and various autoimmune disorders
- central ulcers are most serious because they can perforate or scar

Symptoms and Signs

- RED FLAG - pain NOT relieved with topical anesthetic
- pain, photophobia, tearing, decreased visual acuity (if central ulcer)
- area of thinning with infiltrative base
- surrounding corneal edema, conjunctivitis
- +/- hypopyon (pus in anterior chamber)

Complications

- corneal perforation
- infection of globe - endophthalmitis

Management

- OCULAR EMERGENCY - refer to Ophthalmology service
- culture first
- topical antibiotics every hour
- must treat vigorously to avoid complications

HERPES SIMPLEX KERATITIS

- usually HSV type 1
- may be triggered by stress, fever, UV light, immunosuppression

Symptoms and Signs (see Colour Atlas OP3)

- pain, tearing, foreign body sensation, redness,
- may have visual acuity loss
- corneal hypoesthesia
- occasional cold sore on lip, vesicles on skin
- dendritic lesion seen in corneal epithelium with fluorescein staining + cobalt blue illumination

Complications

- geographic ulcer (delicate denritic lesions) may often arise
- corneal scarring (can lead to loss of vision)
- chronic interstitial keratitis due to penetration of virus into stroma
- secondary iritis

Management

- epithelial debridement
- NO STEROIDS initially - may exacerbate condition
- antivirals such as topical trifluridine (Viroptic), or oral acyclovir (Zovirax)
- ophthalmologist must exercise caution if adding topical steroids for chronic keratitis or iritis

HERPES ZOSTER KERATITIS

- CN VI territory

Symptoms and Signs (see Colour Atlas OP18)

- neuralgia-type pain (vesicular skin eruption ~ 2 weeks)
- ocular tearing, pain and photophobia
- corneal hypoesthesia
- Hutchinson's sign: if tip of nose involved (nasociliary branch of VI), eye involvement likely (75% chance)

Complications

- corneal keratitis, ulceration, perforation and scarring
- uveitis
- glaucoma secondary to trabeculitis
- muscle palsies (rare) due to CNS involvement
- occasionally severe post-herpetic neuralgia

Management

- oral acyclovir, valcyclovir or famcyclovir +/- cycloplegic agent
- topical steroids as indicated for keratitis, iritis (prescribed by an Ophthalmologist)

KERATOCONUS

- bilateral central thinning and bulging (ectasia) of the cornea to form a conical shape
- associated with Descemet's and Bowman's membrane folds
- onset between 1st and 3rd decade
- results in irregular astigmatism, corrects poorly with glasses
- apical scarring
- blurring of visual acuity is the only symptom

Management

- contact lens initially if spectacle correction unsatisfactory
- penetrating keratoplasty (corneal transplant) 90% successful
- post-operative complications: endophthalmitis, graft rejection, graft dehiscence

ARCUS SENILIS

- hazy white ring in peripheral cornea, < 2 mm wide, clearly separated from limbus
- common, bilateral, benign corneal degeneration due to lipid deposition, part of the aging process
- may be associated with hypercholesterolemia if age < 50 years
- no associated visual symptoms, no complications
- no treatment necessary

KAYSER-FLEISCHER RINGS

- rare
- pigmented ring 1-3 mm wide, located in the peripheral cornea
- due to copper pigment deposition in Descemet's membrane
- associated with Wilson's disease (hepatolenticular degeneration)

Management

- penicillamine to chelate copper

THE UVEAL TRACT

- uveal tract = iris, ciliary body, and choroid
- vascularized, pigmented middle layer of the eye

UVEITIS

- may involve one or all three parts of the tract
- idiopathic or associated with autoimmune, infectious, granulomatous, malignant causes
- should be managed by an Ophthalmologist

IRITIS

- inflammation of iris
- usually unilateral

Etiology

- usually idiopathic
- connective tissue diseases: juvenile rheumatoid arthritis (JRA), ankylosing spondylitis (AS), Reiter's syndrome, inflammatory bowel disease (IBD)
- infectious: syphilis, Lyme disease, toxoplasmosis, TB, HSV, herpes zoster
- other: sarcoidosis, trauma

THE UVEAL TRACT ... CONT.

Symptoms and Signs (see Colour Atlas OP4)

- ocular pain, tenderness of the globe, PHOTOPHOBIA, decreased visual acuity, brow ache (ciliary muscle spasm)
- ciliary flush (perilimbal conjunctival injection), miosis
- anterior chamber cells (WBC in anterior chamber due to anterior segment inflammation) and flare (protein precipitates in anterior chamber secondary to inflammation)
- occasionally keratic precipitates (clumps of cells on corneal endothelium)
- iritis typically reduces intraocular pressure though severe iritis may cause an inflammatory glaucoma

Complications

- inflammatory glaucoma
- posterior synechiae
 - iritis leading to iris bombe (posterior iris adheres to anterior lens capsule entrapping aqueous in posterior chamber) – angle closure glaucoma
 - indicated by an irregularly shaped pupil
- anterior synechiae (rare): adhesions of iris to cornea → glaucoma
- cataracts
- band keratopathy (with chronic iritis)
 - superficial corneal calcification keratopathy
- macular edema with chronic iritis

Management

- dilate pupil to prevent formation of posterior synechiae and to decrease pain from ciliary spasm
- topical, subconjunctival, or systemic steroids
- systemic analgesia
- medical workup may be indicated to determine etiology

POSTERIOR UVEITIS

- inflammation of the choroid

Etiology

- bacterial: syphilis, tuberculosis
- viral: herpes simplex virus, cytomegalovirus in AIDS
- fungal: histoplasmosis, candidiasis
- parasitic: toxoplasma, toxocara
- immunosuppression may predispose to any of the above infections
- autoimmune: Behcet's disease
- malignancies: metastatic lesions, malignant melanoma

Symptoms and Signs

- decreased visual acuity
- floaters
- frequently there is no conjunctival or scleral injection
- vitreous cells and opacities
- hypopyon formation

Management

- retrobulbar, or systemic steroids if indicated (e.g. threat of vision loss)

GLOBE

ENDOPHTHALMITIS

- most commonly a postoperative complication of cataract surgery, or due to post-penetrating injury to eye, but also bloodstream dissemination from elsewhere

Symptoms and Signs (see Colour Atlas OP6)

- very painful, red eye with circumlimbal flush
- anterior chamber cells, hypopyon
- reduced vision, extreme photophobia

Management

- OCULAR EMERGENCY: immediate admission to prevent loss of eye
- vitreous tap and/or vitrectomy
- intravitreal, topical, IV antibiotics

LENS

- consists of an outer capsule surrounding a soft cortex and a firm inner nucleus

CATARACTS

- lens opacity
- most common cause of reversible blindness

Etiology

- increased age is the most common cause
 - includes nuclear sclerosis, cuneiform (spoke-like), cortical and posterior subcapsular cataracts
- congenital
 - presents with leukocoria
 - treat promptly to prevent amblyopia
- juvenile onset: diabetes mellitus, metabolic abnormalities (e.g. Wilson's disease, galactosemia, homocystinuria)
- medication toxicity
 - steroids: posterior subcapsular cataracts
 - phenothiazines: anterior subcapsular cataracts
 - hypocalcemia: zonular cataracts (rare)
- inflammatory: uveitis
- radiation, UV light
- traumatic: typically leaf-shaped or rosette cataracts, may see subcapsular cataracts

Symptoms and Signs (see Colour Atlas OP7)

- gradual, progressive, painless decrease in visual acuity
 - "second sight" phenomenon: only in cataracts due to nuclear sclerosis
 - cataract increases power of lens causing artificial myopia - patient may read without reading glasses
- halos around lights at night, double and triple images
- diagnose by slit-lamp exam and by eliciting red reflex with direct ophthalmoscope
- if severe enough may not be able to see fundus on ophthalmic exam

Management

- surgical extraction of the cataract
- phacoemulsification
 - more common
 - remove cataract nucleus and cortex, leaving the posterior capsule and a peripheral rim of anterior capsule
 - less commonly by extracapsular nuclear expression and leaving the posterior capsule behind
 - leaving an intact posterior capsule confines the vitreous to the posterior segment, thereby reducing the incidence of retinal detachments and macular edema
 - allows for posterior chamber intraocular lens, lowering risk of hyphema, glaucoma and corneal endothelial damage
 - approximately 5-30% get an "after-cataract" (opacification of posterior capsule) which is treated with YAG laser capsulotomy
- intracapsular: (now rarely used)
 - remove whole lens with the cataract still in the capsule
 - use with anterior chamber intraocular lens, glasses, or contact lenses
- post-operative complications: retinal detachment, endophthalmitis, opacified posterior capsule, corneal edema secondary to endothelial damage, macular edema

Indication for Surgery

- absolute indications: trauma, congenital cataracts
- relative indications: age related (elective surgery when cataract interferes with daily living)

DISLOCATED LENS 'ECTOPIA LENTIS'

Etiology

- associated with Marfan's Syndrome, Ehlers-Danlos type VI, homocystinuria, syphilis, lens coloboma (congenital cleft due to failure of ocular adnexa to complete growth)
- traumatic

Symptoms and Signs

- decreased visual acuity
- may get unilateral diplopia
- iridodonesis (quivering of iris with movement)
- direct ophthalmoscopy may elicit abnormal red reflex

Complications

- cataract, glaucoma, uveitis

Management

- surgical correction +/- lens replacement

RETINA AND VITREOUS

VITREOUS

- clear collagen-containing gel that fills the posterior segment of eye
- normally firmly attached to optic disc and pars plana and apposed to the retina
- commonly liquefies with age (syneresis)
- when syneresis occurs, remaining vitreous gel can collapse on itself and lift away from retinal surface (posterior vitreous detachment)
- posterior vitreous detachment can cause vitreous hemorrhage, retinal tears and retinal detachment if abnormal vitreoretinal adhesions are present (see Retinal Detachment and Vitreous Hemorrhage sections)
- vitreous floaters
 - shadow of a mobile vitreous opacity cast upon the retina
 - must rule out retinal tears or hemorrhagic diseases
 - harmless if no serious pathology

VITREOUS HEMORRHAGE

Etiology

- diabetic retinopathy (most common cause)
- retinal detachment/tear
- retinal vein occlusion
- posterior vitreous detachment
- trauma

Symptoms and Signs

- sudden loss of visual acuity
- may be preceded by many floaters and/or flashes of light
- ophthalmoscopy: retina not visible due to blood in vitreous

Management

- ultrasound to rule out retinal detachment
- expectant: in non-urgent cases (e.g. no retinal detachment) blood resorbs in 3-6 months
- surgical: vitrectomy, retinal detachment repair
- retinal endolaser to possible bleeding sites/vessels

RETINA

- sensory component of eye
- made up of photoreceptor, nerve cells and pigment epithelial layer
- macula: area rich in cones, most sensitive area of retina and is darker due to lack of retinal vessels and thinning of retina in this region
- fovea: centre of macula, responsible for the most acute, fine vision
- optic disc: normally reddish-orange, with central yellow cup (normal cup/disc (C:D) ratio is < 0.5), retinal artery and vein pass through cup

CENTRAL RETINAL ARTERY OCCLUSION (CRAO)

Etiology

- emboli from carotid arteries or heart (e.g. arrhythmia, endocarditis, valvular disease)
- thrombus
- temporal arteritis

Symptoms and Signs (see Colour Atlas OP19)

- sudden, painless (except in temporal arteritis), unilateral loss of vision
- relative afferent pupillary defect (RAPD)
- fundoscopy
 - cherry red spot (macula), retinal pallor
 - boxcars (segmentation of blood in arteries), narrowed arteries
 - non-pulsatile empty veins
 - Hollenhorst plaques (small glistening cholesterol emboli, located commonly at bifurcation of retinal arteries, originating from the carotid arteries or the aortic arch)

Management

- OCULAR EMERGENCY: attempt to restore blood flow within 2 hours
- if < 2 hours
 - massage the globe
 - decrease intraocular pressure
 - topical β -blockers
 - inhaled oxygen – carbon dioxide mixture
 - IV Diamox (carbonic anhydrase inhibitor)
 - IV Mannitol (draws fluid from eye)
 - anterior chamber paracentesis (carries risk of endophthalmitis)

BRANCH RETINAL ARTERY OCCLUSION (BRAO)

- only part of the retina becomes ischemic resulting in loss of a visual field
- more likely than a CRAO to be embolic; search for source
- management: if < 2 hrs since onset of symptoms, ocular massage to dislodge embolus if VA is affected

CENTRAL RETINAL VEIN OCCLUSION (CRVO)

- an uncommon cause of blindness in the elderly
- choriocapillaris protects retina from ischemia

Predisposing Factors

- hypertension
- arteriosclerotic vascular disease
- diabetes mellitus
- glaucoma
- hyperviscosity e.g. polycythemia rubra vera, sickle-cell disease, lymphoma, leukemia, macroglobulinemia
- any condition that slows venous blood flow

Symptoms and Signs (see Colour Atlas OP20)

- painless, unilateral, gradual or sudden visual loss
- relative afferent pupillary defect (RAPD)
- retina engorged with blood, "ketchup retina", "blood and thunder"
- swollen disc, dilated veins, marked flame-shaped hemorrhages
- cotton wool spots in resolving phase
- two fairly distinct groups
 - venous stasis/non-ischemic retinopathy
 - VA approximately 20/80, no RAPD
 - mild hemorrhage, few cotton wool spots
 - resolves spontaneously over weeks to months
 - may regain normal vision if macula intact
 - hemorrhagic/ischemic retinopathy
 - usually older patient with deficient arterial supply
 - VA approximately 20/200, reduced peripheral vision, RAPD
 - more hemorrhages, cotton wool spots, congestion
 - poor visual prognosis

Complications

- occurs in 33% of cases
- degeneration of retinal pigment epithelium
- liquefaction of vitreous
- neovascularization of retina and especially iris (secondary rubeosis), which can lead to secondary glaucoma
- iritis

Management

- retinal laser photocoagulation to reduce neovascularization

RETINAL DETACHMENT (RD)

- fluid collects between the neurosensory retina and the underlying retinal pigment epithelium
- three types
- rhegmatogenous
 - most common type of RD
 - indicates that the detachment originally started with a hole or tear in the retina
 - tears may be caused by posterior vitreous detachment, trauma or iatrogenically
 - fluid goes through the hole and lifts the neuroretina off the pigment layer and choroid
 - more likely to occur spontaneously in high myopes, or after ocular surgery/trauma
- tractional
 - found in proliferative retinopathies such as diabetes, CRVO, sickle cell disease, retinopathy of prematurity (ROP) and ocular trauma
 - results from vitreal preretinal traction (due to vitreal, epiretinal or subretinal membrane) pulling the sensory retina away from the underlying pigment epithelium
- exudative
 - may be due to choroidal tumour, metastatic tumour, uveitis
 - caused by local or systemic conditions that damage the retinal pigment epithelium which allows passage of the choroidal fluid into the subretinal space

Symptoms and Signs (see Colour Atlas OP24)

- sudden onset
- flashes of light
 - due to mechanical stimulation of the retinal photoreceptors
- floaters
 - hazy spots in the line of vision which move with eye position, due to drops of blood in the vitreous (blood vessels torn as the retina tears)
- curtain of blackness (late)
 - darkness in one field of vision when the retina detaches in that area and function is reduced
- decreased vision
 - visual acuity dramatically drops if the macula becomes detached
- relative afferent pupillary defect (RAPD) may be seen

RETINA AND VITREOUS ... CONT.

Management

- prophylactic: if a symptomatic tear (flashes or floaters) is detected, it can be sealed off with laser therapy or cryotherapy, thereby preventing progression to detachment
- therapeutic
 - rhegmatogenous retinal detachment:
 - scleral buckling (retinal break is mounted on sclera indented by an explant)
 - pneumatic retinopexy (intraocular injection of air or an expandable gas in order to tamponade the retinal break while the chorioretinal adhesion forms)
 - both treatments used in combination with localization of the retinal break and treatment with diathermy, cryotherapy or laser to create adhesion between the pigment epithelium and the sensory retina
 - intraocular injection of silicone oil in cases of recurrent retinal detachments
 - tractional retinal detachment:
 - vitreoretinal surgery (may involve vitrectomy, membrane removal, scleral buckling and injection of intraocular gas)
 - exudative
 - treatment of underlying cause
- complications: loss of vision, vitreous hemorrhage, recurrent retinal detachment
- a retinal detachment should be considered an emergency, especially if the macula is still attached
- prognosis for visual recovery varies inversely with the amount of time the retina is detached and whether the macula is attached or not

RETINITIS PIGMENTOSA

- hereditary degenerative disease of the retina
- degeneration of rod cells and retinal atrophy
- areas of peppery/"bone-spicule" pigmentary degeneration scattered through mid- and peripheral retina
- symptoms: night blindness, often beginning in adolescence, with expanding ring scotoma (abnormal blind spot) leading to blindness
- no satisfactory treatment

ROTH SPOTS

- flame-shaped hemorrhages with central white fibrin thrombosis
- seen in
 - subacute bacterial endocarditis (SBE)
 - leukemia (hemorrhage and extravasation of leukocytes)
 - severe anemia

AGE-RELATED MACULAR DEGENERATION (ARMD)

- leading cause of blindness in people > 65 years old
- 10% of people > 65 years old have some degree of ARMD
- female > male

Non-Neovascular (Non-Exudative/"Dry") ARMD

- most common type of ARMD - 90% of cases
- drusen: pale, yellow-white deposits of membranous vesicles and collagen deposited between the retinal pigment epithelium (RPE) and Bruch's membrane (area separating inner choroidal vessels from RPE)
 - no visual loss unless atrophy of overlying RPE and photoreceptors
- RPE atrophy: coalescence of depigmented RPE, clumps of focal hyperpigmentation

Neovascular (Exudative/"Wet") ARMD

- 10% of ARMD, yet 80% results in severe visual loss
- choroidal neovascularization: drusen predispose to breaks in Bruch's membrane, subsequent growth and proliferation of choroidal capillaries
 - may get serous detachment of overlying RPE and retina, hemorrhage and lipid precipitates into subretinal space
- disciform scarring
 - an elevated subretinal mass due to fibrous metaplasia of hemorrhagic retinal detachment
 - causes severe central visual loss

Risk Factors

- female
- increased age
- family history
- smoking

Symptoms and Signs

- variable amount of progressive visual loss
- metamorphopsia (distorted vision characterized by straight parallel lines appearing convergent or wavy)

RETINA AND VITREOUS ... CONT.

Investigations

- Amsler Grid: commonly held at normal reading distance with glasses on, assesses macular function
- fluorescein angiography: see neovascularization, leaks

Management

- non-neovascular ARMD
 - monitor
 - low vision aids e.g. magnifiers, closed-circuit television
 - query – anti-oxidants
- neovascular ARMD
 - laser photocoagulation for neovascularization
 - 50% of choroidal neovascularization cannot be treated initially
 - no definitive treatment for disciform scarring
 - focal laser for macular edema
 - photodynamic therapy with verteporfin: IV injection of verteporfin followed by low intensity laser to area of choroidal neovascularization;

Treatment of Age-Related Macular Degeneration with Photodynamic Therapy (TAP) Study Group indicate that for selected patients with subfoveal lesions in ARMD with predominantly classic choroidal neovascularization, verteporfin treatment can reduce the risk of moderate vision loss for at least 1 year; this therapy cannot stop or reverse vision loss in all patients with ARMD; investigations are ongoing

BLURRED OPTIC DISC MARGINS

DRUSEN

- German, plural for "granules"
- "giant drusen": hyaline deposits at disc margin and in disc itself, commonly producing field defects
- more common form is deposited in Bruch's membrane (area separating inner choroidal vessels from retinal pigment epithelium)
- seen with increasing age, retinal and choroidal degeneration and as a primary dystrophy
- with drusen alone, vision is normal or near normal

MYELINATED NERVE FIBRES

- a variant of normal in which the retinal nerve fibres are myelinated anterior to the cribriform plate
- appear as white streaks extending from the cup and occasionally in retina remote from disc

Table 5. Differential Diagnosis of Blurred Optic Disc Margins

| | Papilledema (see Colour Atlas OP11) | Optic Neuritis | Ischemic Neuropathy | Central Retinal Vein Occlusion (CRVO) |
|------------------------------|---|---|--|--|
| Age | Any | < 50 | > 50 | > 50 |
| Etiology | Increased intracranial pressure (ICP) | Idiopathic, associated with MS | Idiopathic, vascular, Giant Cell Arteritis (GCA) | Idiopathic |
| VA | Normal | Reduced | Reduced | Reduced |
| Other sign or symptom | Bilateral, enlarged blind spot, neurologic findings (e.g. headache) | Unilateral, pain on eye movement, RAPD, reduced color vision, +/- MS findings | Altitudinal field loss, +/- GCA findings (e.g. jaw claudication, headache, tender scalp) | Unilateral, hypertension, diabetes, increased viscosity, arteriosclerotic vascular disease |
| Fundoscopy findings | Swollen disc, hemorrhage, dilated retinal veins | Hyperemic swollen disc, normal if retrobulbar neuritis | Pale, swollen disc, flame hemorrhage | "Blood and thunder", swollen disc, venous engorgement, retinal hemorrhage |
| Investigation | CT head | Visual field (VF), CT (if atypical VF or if no improvement in 6 weeks) | ESR | Fluorescein angiography, medial evaluation |
| Treatment | Neurosurgery consult, treat underlying cause | Steroids | High dose steroids (for GCA) | Panretinal laser photocoagulation steroids |

GLAUCOMA

Definition

- progressive optic neuropathy involving characteristic structural changes to optic nerve head with associated visual field changes
- commonly associated with high intraocular pressure (IOP)

Background

- aqueous is produced by the ciliary body and flows from the posterior chamber to the anterior chamber through the pupil, and drains into the episcleral veins via the trabecular meshwork and the canal of Schlemm (see Figure 1)
- an isolated increase in IOP is termed ocular hypertension (or glaucoma suspect) and these patients should be followed because ~10% if 20-30 mmHg; 40% if 30-40 mmHg; most if > 40 mm Hg (or more) will develop glaucoma
- normal, average IOP is 16 +/- 2 mm Hg (diurnal variation, higher in a.m.)
- pressures > 21 more likely to be associated with glaucoma
- normal C/D (cup/disc) ratio < 0.4
- suspect if glaucoma if C/D ratio > 0.6, difference between eyes > 0.2 or cup approaches disc margin
- loss of peripheral vision most commonly precedes central loss
- sequence of events: gradual pressure rise, followed by increased in C/D ratio, followed by visual field loss as cup approaches disc margin (usually asymptomatic)
- screening tests should include
 - medical and family history
 - visual acuity testing
 - slit lamp exam: to assess anterior chamber depth
 - ophthalmoscopy: to assess the disc features
 - tonometry by applanation or indentation: to measure the IOP
 - Humphrey visual field testing

PRIMARY OPEN ANGLE GLAUCOMA

- most common form, > 55% of all glaucoma cases
- due to obstruction to aqueous drainage within the trabecular meshwork and its drainage into the canal of Schlemm
- screening is critical for early detection

Risk Factors

- elevated intraocular pressure (> 21 mm Hg)
- age > 40
- higher incidence in blacks
- myopes
- familial, polygenic (10x increased risk)
- diabetes
- chronic topical steroid use on eyes in steroid responders
- previous ocular trauma
- anemia/hemodynamic crisis (ask about blood transfusions in past)
- hypertension

Symptoms and Signs (see Colour Atlas OP26)

- asymptomatic initially
- insidious, painless, gradual rise in IOP due to restriction of aqueous outflow
- bilateral, but usually asymmetric
- visual field loss
 - slow, progressive, irreversible loss of peripheral vision
 - arcuate scotoma and nasal step are characteristic
- late loss of central vision if untreated
- earliest signs are optic disc changes
 - increased cupping of disc with a cup/disc ratio > 0.4 (the cup is usually enlarged asymmetrically (i.e. eye more affected initially) and cup approaches disc margin)
 - large vessels become nasally displaced
- may have hemorrhage at disc margin
- safe to dilate pupil

Management

- principles: increase the drainage of aqueous and/or decrease the production of aqueous
- medical treatment: see Ocular Medications section
 - increases aqueous outflow
 - topical cholinergics
 - topical adrenergics
 - topical prostaglandin agonist
 - decreases aqueous outflow
 - topical beta-blockers
 - topical and oral carbonic anhydrase inhibitor
 - topical adrenergics
- surgical treatment
 - laser: trabeculoplasty, selective destruction of ciliary body
 - microsurgery: trabeculectomy, tube shunt placement
- visual field testing to monitor course of disease

PRIMARY ANGLE CLOSURE GLAUCOMA

- 12% of all glaucoma cases
- peripheral iris bows forward in an already susceptible eye with a shallow anterior chamber obstructing aqueous access to the trabecular meshwork

Risk Factors

- hyperopia: small eye, big lens - large lens crowds the angle
- age > 70, female
- family history, more common in Asians and Inuit
- mature cataracts
- shallow anterior chamber
- pupil dilation (topical and systemic anticholinergics, stress, darkness)

Symptoms

- unilateral, but other eye predisposed
- RED FLAG: red, painful eye
- decreased visual acuity, vision acutely blurred from corneal edema
- halo around lights
- nausea and vomiting
- abdominal pain

Signs

- fixed mid-dilated pupil
- corneal edema with conjunctival injection
- marked increase in IOP even to palpation (> 40 mm Hg)
- shallow anterior chamber, +/- cells in anterior chamber

Complications

- irreversible loss of vision if untreated, within hours to days
- permanent peripheral anterior synechiae

Management

- immediate treatment important to
 - preserve vision
 - prevent adhesions of peripheral iris to trabecular meshwork (peripheral anterior synechiae) resulting in permanent closure of angle
- medical treatment: see Ocular Medications section
 - miotic drops (pilocarpine)
 - topical beta-blockers
 - systemic carbonic anhydrase inhibitors
 - systemic hyperosmotic agents (oral glycerine; IV hypertonic mannitol)
 - topical steroids (not in primary care)
- surgical treatment (for acute angle closure glaucoma)
 - laser iridotomy or surgical iridectomy

SECONDARY OPEN ANGLE GLAUCOMA

- increased IOP secondary to ocular/systemic disorders which clog the trabecular meshwork
- steroid-induced glaucoma
 - due to topical/systemic corticosteroid use
 - develop in 25% (higher in extended use) of general population (responders) after 4 weeks (or less) of OID topical steroid use
 - 5% of population are super-responders
- traumatic glaucoma
 - hyphema-induced increase in IOP
 - angle recession glaucoma occurs with blunt, non-penetrating trauma to globe and orbit, causing tears in trabecular meshwork and ciliary body with secondary scarring
- pigmentary dispersion syndrome
 - iris pigment clogs trabecular meshwork
 - typically seen in younger myopes
- pseudoexfoliation syndrome
 - abnormal basement membrane-like material clogs trabecular meshwork
 - seen mostly in the elderly
- neovascular glaucoma
 - abnormal blood vessels develop on surface of iris (rubeosis iridis)
 - due to retinal ischemia associated with proliferative diabetic retinopathy and CRVO
 - treatment with laser therapy to retina, to reduce neovascular stimulus to iris vessels

SECONDARY ANGLE CLOSURE GLAUCOMA

- uveitis
 - inflamed iris adheres to lens (posterior synechiae)

NORMAL PRESSURE GLAUCOMA

- IOP in normal range but cupping and field loss typical of glaucoma are present
- often found in women > 60 but may occur earlier
- may see splinter hemorrhages of disc margin
- damage to optic nerve may be due to vascular insufficiency

Management

- treat any underlying medical condition and lower the IOP still further

CONGENITAL GLAUCOMA

- due to inadequate development of the filtering mechanism of the anterior chamber angle

Symptoms and Signs

- cloudy cornea, increased IOP
- photophobia, tearing
- buphthalmos (large eye), blepharospasm

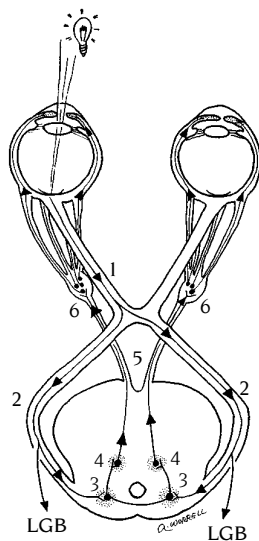
Management

- filtration surgery is required soon after birth to prevent blindness

PUPILS

PUPILLARY LIGHT REFLEX (see Figure 4)

- light shone directly into eye travels along optic nerve (1) to optic tracts (2) to both sides of midbrain
- impulses enter both sides of midbrain via pretectal area (3) and Edinger-Westphal nuclei (4)
- nerve impulses then travel down both CNs III (5) to reach the ciliary ganglia (6), and finally to the iris sphincter muscle, which results in direct and consensual light reflex



1. optic nerve
2. optic tracts
3. pretectal area
4. Edinger-Westphal nuclei
5. cranial nerve III
6. ciliary ganglia

LGB = lateral geniculate body

Figure 4. Pathway of direct light reflex from left eye and consensual light reflex from right eye

Illustration by Aimée Worrell

DILATED PUPIL (MYDRIASIS) DIFFERENTIAL DIAGNOSIS**Physiologic Anisocoria (unequal pupil size)**

- occurs in 20% of population
- difference < 1-2 mm, most pronounced in dark
- normal light reactivity, may vary from day to day

Sympathetic Stimulation

- fight or flight response
- drugs: epinephrine, dipivefrin (Propine), phenylephrine

Parasympathetic Understimulation

- cycloplegics/mydriatics: atropine, tropicamide, cyclopentolate (parasympatholytic)
- CN III palsy
 - eye deviated down and out with ptosis present
 - etiology includes cerebrovascular accident (CVA), neoplasm, aneurysm, diabetes mellitus (DM) (may spare pupil)
- dilated pupil from medication does not respond to constricting drugs (e.g. pilocarpine), unlike a CN III palsy

Acute Angle Closure Glaucoma

- fixed, mid-dilated pupil

Adie's Tonic Pupil

- 80% unilateral, females > males
- pupil reacts poorly to light (both direct and consensual) constricts with accommodation
- hyporeflexic (Adie's syndrome)
- defect at ciliary ganglion: results in denervation hypersensitivity of constrictor muscle
 - 0.125% solution of pilocarpine will constrict tonic pupil
 - no effect on normal pupil
- pupil eventually gets smaller than the unaffected eye

Trauma

- damage to iris sphincter from blunt or penetrating trauma
- iris transillumination defects may be apparent using ophthalmoscope or slit lamp
- pupil may be dilated (traumatic mydriasis) or irregularly shaped

CONSTRICTED PUPIL (MIOSIS) DIFFERENTIAL DIAGNOSIS**Physiologic Anisocoria (unequal pupil size)**

- as for Dilated Pupil section above

Senile Miosis

- decreased sympathetic stimulation with age

Parasympathetic Stimulation

- local or systemic medications
- cholinergic agents: pilocarpine, carbachol
- cholinesterase inhibitor: phospholine iodide
- opiates, barbiturates

Horner's Syndrome

- lesion in sympathetic pathway
- difference in pupil size greater in dim light, due to decreased innervation of adrenergics to iris dilator muscle
- associated with anhidrosis, ptosis of ipsilateral face/neck
- application of cocaine to eye does not result in pupil dilation (vs. normal pupil)
- causes: brainstem infarct, demyelinating disease, cervical or mediastinal tumour, aneurysm of carotid or subclavians, goiter, cervical lymphadenopathy, surgical sympathectomy

Iritis

- miotic initially
- later, may be irregularly shaped pupil due to posterior synechiae
- does not react to light in later stages

Argyll Robertson Pupil

- pupils irregular and < 3 mm diameter, may have ptosis
- does not respond to light stimulation
- does respond to accommodation
- suggestive of CNS syphilis or other conditions (DM, encephalitis, MS)

PUPILS ... CONT.

RELATIVE AFFERENT PUPILLARY DEFECT (RAPD) (see Neurology Chapter)

- ❑ defect in visual afferent pathway anterior to optic chiasm
- ❑ differential diagnosis: optic nerve compression, optic neuritis, large retinal detachment, CRAO, CRVO, advanced glaucoma
- ❑ does not occur with media opacity e.g. corneal edema, cataracts
- ❑ test: swinging flashlight
 - if light is shone in the affected eye, direct and consensual response to light is decreased
 - if light is shone in the unaffected eye, direct and consensual response to light is normal
 - if the light is moved quickly from the unaffected eye to the affected eye, "paradoxical" dilation of both pupils occurs
 - use ophthalmoscope with "+4" setting, using red reflex especially in patients with dark irides

Clinical Pearl

- ❑ **Even dense cataracts do not produce a relative afferent pupillary defect.**

NEURO-OPHTHALMOLOGY

VISUAL FIELD DEFECTS (see Neurology Chapter)

- ❑ lesions in the visual system have characteristic pattern losses
- ❑ several tests used: confrontation (screening), tangent screen, Humphrey fields, Goldman perimetry

BITEMPORAL HEMIANOPSIA

- ❑ a chiasmal lesion

Etiology

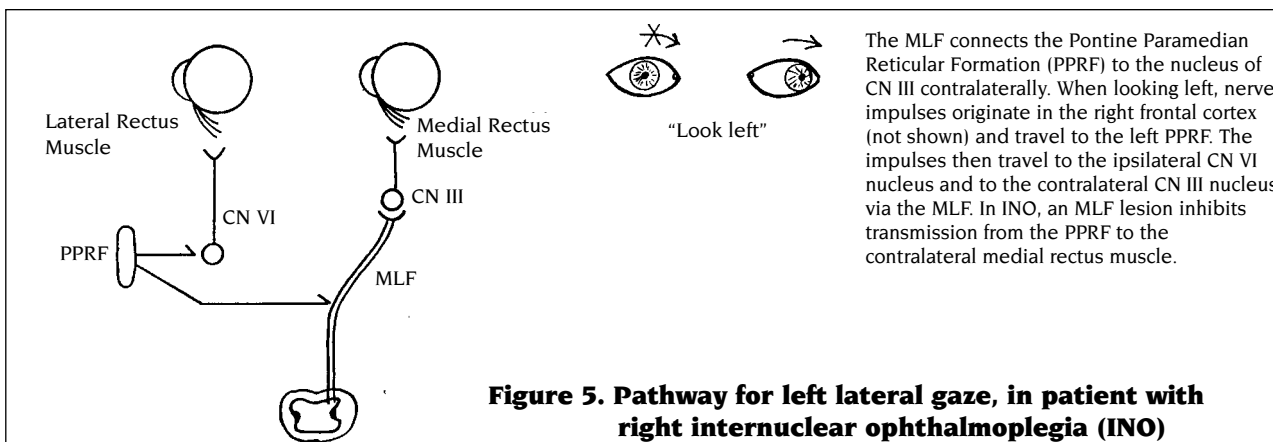
- ❑ In children: craniopharyngioma
- ❑ In middle aged: pituitary mass
- ❑ In elderly: meningioma

HOMONYMOUS HEMIANOPSIA

- ❑ a retrochiasmal lesion
- ❑ the more congruent, the more posterior the lesion
- ❑ check all hemiplegic patients for ipsilateral homonymous hemianopsia
- ❑ e.g. left hemisphere → right visual field (VF) defect in both eyes

INTERNUCLEAR OPHTHALMOPLEGIA

- ❑ commonly seen in multiple sclerosis (MS)
- ❑ lesion of medial longitudinal fasciculus (MLF) (see Figure 5)
- ❑ delayed movement in ipsilateral adducting eye
- ❑ monocular nystagmus in contralateral abducting eye



NYSTAGMUS (see Otolaryngology Chapter)

- ❑ definition: rapid, involuntary, small amplitude movements of the eyes that are rhythmic in nature
- ❑ direction of nystagmus is defined by the rapid eye component of motion
- ❑ can be categorized by movement type (pendular, jerking, rotatory, coarse) or as normal vs. pathological

Pendular Nystagmus

- ❑ due to poor macular function or cerebellar lesion
- ❑ eye oscillates equally about a fixation point
- ❑ may be present in people who become blind early in life

NEURO-OPHTHALMOLOGY ... CONT.

Jerking Nystagmus

- due to brainstem, labyrinthine, cerebellar disease
- rapid movement in one direction, slow movement in opposite direction

Coarse Nystagmus

- due to weakness of ocular muscles or lower motor neuron disease
- coarse jerking in the direction of the weakened muscle

Normal Nystagmus

- endpoint
 - at extremes of gaze, subsides after a few beats
 - also seen in patients on anticonvulsant therapy
- evoked
 - optokinetic: occurs when patient viewing a moving object
 - vestibular: labyrinthine stimulation from thermal or rotatory movement
 - unlike central lesions, vestibular nystagmus is suppressed by fixation

Abnormal Nystagmus

- any nystagmus which occurs in the field of binocular vision persistently
- vertical nystagmus
 - always abnormal if persistent
 - up-beating: usually due to acquired disease or lesion in medulla
 - down-beating: usually due to lesion in lower brainstem or upper cervical cord
- congenital nystagmus
 - pendular, may cause amblyopia from poor fixation
- gaze-evoked nystagmus
 - in certain fields of gaze
 - with a brainstem/cerebellar lesion, acoustic neuroma phenytoin, ethanol, barbiturates, demyelinating diseases
- spasmus nutans
 - asymmetrical fine nystagmus of unknown pathogenesis
 - in children 4-12 months of age, usually recover by 2 years
 - head nodding to offset pathological eye movements
- ataxic nystagmus
 - one eye has more marked nystagmus than the other
 - associated with internuclear ophthalmoplegias

INTRAOCULAR MALIGNANCIES

- uncommon site for primary malignancies
- eye usually affected secondarily by cancer or cancer treatments

MALIGNANT MELANOMA

- most common primary intraocular malignancy
- more prevalent in Caucasians
- arise from uveal tract
- hepatic metastases predominate

Management

- choice is dependent on the size of the tumour
- radiotherapy, enucleation, limited surgery

RETINOBLASTOMA

- incidence: 1/1000; sporadic or genetic transmission
- unilateral or bilateral (in 1/3 of cases)
- malignant - direct or hematogenous spread
- diagnosis
 - may be detected by leukocoria (white pupil) in infant
 - CT scan: dense radiopaque appearance (contains calcium)

Management

- radiotherapy, enucleation (removal of globe from eye socket), or both

METASTASES

- most common intraocular malignancy in adults
- most common from breast and lung
- usually infiltrate the choroid, but may also affect the optic nerve or extraocular muscles
- may present with decreased or distorted vision, irregularly shaped pupil, iritis, hyphema

Management

- local radiation, chemotherapy
- enucleation if blind painful eye

OCULAR MANIFESTATIONS OF SYSTEMIC DISEASE

HIV

- up to 75% of patients with AIDS have ocular manifestations
- ocular findings include: opportunistic infections, ocular vascular manifestations, neoplasms,

Lids/Conjunctiva

- Kaposi's sarcoma
 - affects conjunctiva of lid or globe
 - numerous vascular skin malignancies
 - DDX: subconjunctival hemorrhage (non-clearing), hemangioma
- molluscum contagiosum
- herpes zoster

Cornea

- herpes simplex keratitis
- herpes zoster

Retina

- HIV retinopathy
 - most common ocular manifestation of HIV
 - cotton wool spots
 - capillary abnormalities
 - intraretinal hemorrhage
- cytomegalovirus (CMV) retinitis (**see Colour Atlas OP25**)
 - most common ocular opportunistic infection in HIV patients
 - most common cause of visual loss in HIV patients, occurring in > 25% of patients
 - develops in late stages of HIV when severely immunocompromised (CD4 count \leq 50)
 - a necrotizing retinitis, with retinal hemorrhage and vasculitis, brushfire or pizza pie appearance
 - untreated infection will progress to other eye in 4-6 weeks
 - symptoms and signs: scotomas related to macular involvement and retinal detachment, blurred vision, and floaters
 - treat with virostatic agents: gancyclovir IV or intravitreal injection, foscarnet IV
- necrotizing retinitis
 - from herpes simplex virus, herpes zoster, *Pneumocystis carinii*, toxoplasmosis
- disseminated choroiditis
 - *Pneumocystis carinii*, *Mycobacterium avium intracellulare*, *Candida*

OTHER SYSTEMIC INFECTIONS

- most common are herpes zoster and candidiasis
- herpes zoster (see Cornea section)
- Candida
 - fluffy, white-yellow, superficial retinal infiltrate that may eventually result in vitritis
 - may see inflammation of the anterior chamber
 - treatment: amphotericin B

DIABETES MELLITUS (DM)

- most common cause of blindness in young people in North America
- blurring of distance vision with rise of blood sugar
- consider DM if unexplained retinopathy, cataract, EOM palsy, optic neuropathy, sudden change in refractive error
- loss of vision due to
 - progressive microangiopathy, leading to macular edema
 - progressive diabetic retinopathy \rightarrow neovascularization \rightarrow vitreous hemorrhage \rightarrow traction \rightarrow retinal detachment
 - Rubeosis Iridis (neovascularization of the iris) leading to neovascular glaucoma (poor prognosis)

Retina

- background
 - altered vascular permeability
 - retinal vessel closure
- non-proliferative: increased vascular permeability and retinal ischemia
 - dot and blot hemorrhages
 - microaneurysms
 - retinal edema
 - hard exudates (lipid deposits)
- advanced non-proliferative (or pre-proliferative):
 - non-proliferative findings plus
 - venous sausageing (in 2 of 4 retinal quadrants)
 - intraretinal microvascular anomalies-IRMA (in 1 of 4 retinal quadrants)
 - IRMA: dilated, leaky vessels within the retina
 - macular edema
 - cotton wool spots (nerve fibre layer infarcts)

- proliferative (**see Colour Atlas OP23**)
 - 5% of patients with diabetes will reach this stage)
 - neovascularization: iris, disc, retina to vitreous
 - neovascular glaucoma (secondary to rubeosis iridis)
 - vitreous hemorrhage, fibrous scarring, retinal detachment
 - increased risk of severe visual loss

Screening Guidelines for Diabetic Retinopathy

- Type I DM
 - screen for retinopathy beginning annually 5 years after disease onset
 - screening not indicated before the onset of puberty
- TYPE II DM
 - initial examination shortly after diagnosis, then repeat annually
 - if initial exam negative, repeat in 4 years, then annual exams
- pregnancy
 - ocular exam in 1st trimester, close follow-up throughout
 - gestational diabetics not at risk for retinopathy

Management

- Diabetic Control and Complications Trial (DCCT) (see Endocrinology Chapter)
 - tight control of blood sugar decreases frequency and severity of microvascular complications
- blood pressure control
- focal laser for macular edema
- panretinal laser photocoagulation, reduces neovascularization by decreasing retinal metabolic demand
 - reduces risk of blindness
- vitrectomy for vitreous hemorrhage and retinal detachment

Lens

- earlier onset of senile nuclear sclerosis and cortical cataract
- may get hyperglycemic cataract, due to sorbitol accumulation (rare)
- sudden changes in refraction of lens: changes in blood glucose levels (poor control)
 - may cause refractive changes by 3-4 diopters

Extra Ocular Movement (EOM) Palsy

- usually secondary to CN III infarct
- pupil usually spared in diabetic CN III palsy, but get ptosis
- may involve CN IV and VI
- usually recover within one year

Optic Neuropathy

- visual acuity loss due to infarction of optic disc/nerve

MULTIPLE SCLEROSIS (see Neurology Chapter)

- relapsing or progressive CNS disease characterized by disseminated patches of demyelination in the brain and spinal cord resulting in varied symptoms and signs
- many ocular manifestations

Symptoms

- blurred vision secondary to optic neuritis
- decreased colour vision secondary to optic neuritis
- central scotoma, since papillomacular bundle of retinal nerve fibres tends to be affected
- diplopia secondary to internuclear ophthalmoplegia (see Figure 5)

Signs

- RAPD, ptosis, nystagmus, uveitis
- optic atrophy (**see Colour Atlas OP22**), optic neuritis
- internuclear ophthalmoplegia (INO)

Management

- with optic neuritis, treatment with oral steroids leads to greater likelihood of getting MS later on than treatment with IV steroids

HYPERTENSION

- Keith-Wagener Grading
 - I: arteriosclerosis (slight narrowing of arterioles)
 - II: AV nicking, copper wire arterioles, cotton wool spots, hard exudates
 - III: flame hemorrhages (superficial), retinal edema, macular star
 - IV: papilledema (choked disc) (**see Colour Atlas OP21**)

AMAUROSIS FUGAX

- sudden, transient blindness from intermittent vascular compromise
- typically monocular, lasting < 5-10 minutes
- may be associated with paresthesia/weakness in contralateral limbs
- Hollenhorst plaques (glistening microemboli seen at branch points of retinal arterioles)

OCULAR MANIFESTATIONS OF SYSTEMIC DISEASE ... CONT.

HYPERTHYROIDISM/GRAVES' DISEASE

- mnemonic - **NO SPECS**
 - Grade 0: **N**o signs or symptoms
 - Grade 1: **O**nly signs are proptosis < 22 mm (measure by Hertel exophthalmometer) +/- lid lag; +/- lid retraction
 - Grade 2: **S**oft tissue involvement - conjunctival + lid miosis
 - Grade 3: **P**roptosis > 22 mm
 - Grade 4: **E**xtraocular muscle involvement
 - Grade 5: **C**orneal involvement - exposure keratitis
 - Grade 6: **S**ight loss due to optic neuropathy from compression
- ocular changes are often permanent

Management

- treat the hyperthyroidism
- proptosis can cause exposure of the cornea
 - mild stage: use artificial tears
 - more severe: steroids, surgery (lateral tarsorrhaphy, orbital decompression), radiation

CONNECTIVE TISSUE DISORDERS

- most common ocular manifestation: dry eyes (keratoconjunctivitis sicca)
- RA, JRA, SLE, Sjogren's syndrome
- ankylosing spondylitis, polyarteritis nodosa (PAN), giant cell arteritis

GIANT CELL (TEMPORAL) ARTERITIS

- abrupt monocular loss of vision
- ischemic optic atrophy
- 50% lose vision in other eye if untreated
- see Blurred Disc Margin section

SARCOIDOSIS

- granulomatous uveitis with large "mutton fat" keratic precipitates and posterior synechiae
- neurosarcoidosis: optic neuropathy, oculomotor abnormalities, visual field loss

Management

- steroids and mydriatics

STRABISMUS

- non-alignment of eyes, found in 3% of children
- object not visualized simultaneously by fovea of each eye
- complications: amblyopia, cosmetic

TROPIA

- a manifest (apparent) deviation
- deviation not corrected by the fusion mechanism

Types

- exo- (lateral deviation), eso- (medial deviation)
- hyper- (upward deviation), hypo- (downward deviation)
- esotropia = "crossed-eyes"; exotropia = "wall-eyed"
- pseudoesotropia: epicanthal folds give appearance of esotropia but Hirschberg test is normal, more common in Asians

Tests

- Hirschberg: positive if the light reflex in the cornea of the two eyes is asymmetrical
 - light reflex lateral to central cornea indicates esodeviation; medial to central cornea indicates exodeviation
- cover test (see Figure 6)
 - ask patient to fixate on target
 - cover the non-deviated eye, the deviated eye will then move to fixate on the target
 - the covered eye will move in the same direction under the cover
 - if deviated eye moves inward on covering the fixating other eye = exotropia
 - if deviated eye moves outward on covering the fixating other eye = esotropia
- the deviation can be quantified using prisms

PHORIA

- a latent deviation
- a deviation corrected in the binocular state by the fusion mechanism (therefore deviation not seen when patient is using both eyes)
- the Hirschberg test will be normal (light reflexes symmetrical)
- very common - majority are asymptomatic
- may be associated with asthenopia (eye strain)

STRABISMUS ... CONT.

Tests

- cover-uncover test (see Figure 6)
 - placing a cover over an eye with a phoria causes a breakdown of fixation of that eye, which allows it to move to a misaligned position
 - then, uncovering the covered eye will allow it to return to a normal central position
 - covered eye moves inward on removing cover = exophoria
 - covered eye moves outward on removing cover = esophoria
- alternating cover test
 - alternating the cover back and forth from eye to eye detects both tropia and phoria
 - maintain cover over one eye for 2-3 seconds before rapidly shifting to other eye

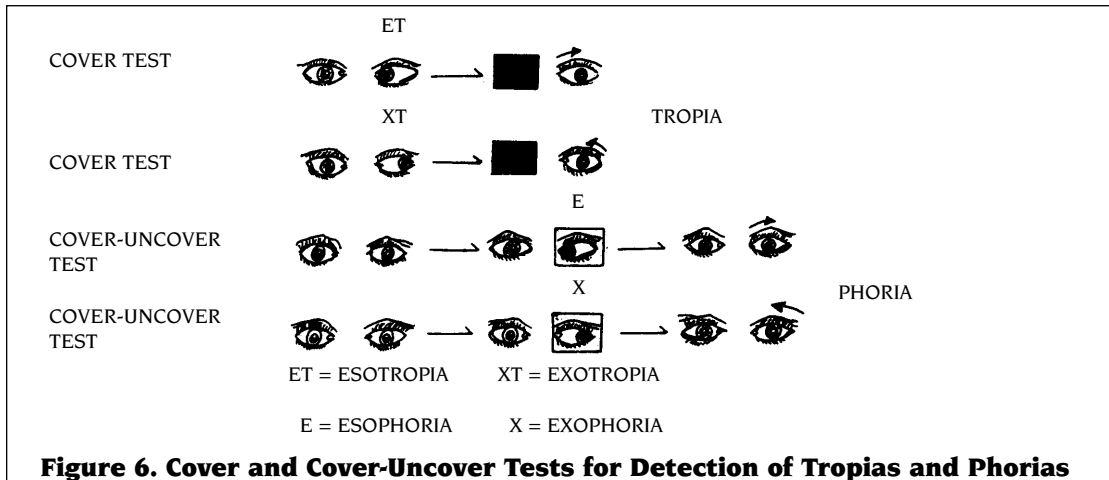


Figure 6. Cover and Cover-Uncover Tests for Detection of Tropias and Phorias

PARALYTIC STRABISMUS

- non-comitant strabismus
- deviation varies in different positions of gaze

Etiology

- neural: arteriosclerotic (CVA), DM, MS, brain tumour
- muscular: myasthenia gravis, Grave's disease
- structural: orbital fracture due to trauma

Features

- mostly in adults, acquired
- diplopia (since adults do not get amblyopia)
- greatest deviation in field of action of the weakened muscle
- vision is usually unaffected in either eye, unless CN II is involved

NON-PARALYTIC STRABISMUS

- comitant strabismus
- deviation equal in all directions of gaze

Features

- usually begins in infancy
- no diplopia (child suppresses the image from the misaligned eye)
- deviated eyes may become amblyopic (see Amblyopia section) if not treated when the child is young i.e. 3-4 years old
- amblyopia treatment rarely successful after age 8
- amblyopia will not develop if child has alternating strabismus or intermittency - allow neural pathways for both eyes to develop

Clinical Pearl

- All children with strabismus should be promptly referred to Ophthalmology.**
- All children with defective vision should be promptly referred to detect and treat non-strabismic amblyopia (e.g. from anisometropia: difference in refraction between two eyes).**

Accommodative Esotropia

- normal response to approaching object is near reflex: convergence, accommodation, and miosis
- hyperopes must constantly accommodate - excessive accommodation can lead to esotropia
- usually reversible with correction of refractive error

Nonaccommodative Esotropia

- accounts for 50% of childhood strabismus
- most are idiopathic
- may be due to ocular pathology (e.g. anisometropia, cataract, retinoblastoma)

PEDIATRIC OPHTHALMOLOGY

AMBLYOPIA

Definition

- decrease in visual acuity due to inappropriate visual stimulation during development
- not due to ocular pathology and not correctable by refraction
note: pathology in visual cortex and lateral geniculate body

Detection

- "Holler Test": younger child upset if good eye covered
- quantitative visual acuity by age 3-4 years using picture charts and/or matching game (Sheridan-Gardiner), testing each eye separately
- not commonly treatable after age 8-9 years since the neural pathways for vision are now formed
- prognosis: 90% will have good vision restored and maintained if treated < 4 years old

Etiology and Management

- strabismus
 - correct with glasses for accommodative esotropia (50% of children experience relief of their esotropia with glasses and will not require surgery)
 - surgery: recession (weakening) = moving muscle insertion further back on the globe; or resection (strengthening) = shortening the muscle
 - botulinum toxin for single muscle weakening
 - cycloplegics or miotics (not commonly used)
 - even after ocular alignment is restored (glasses, surgery, botulinum toxin), patching is frequently necessary until approximately age 8
- refractive errors
 - anisometropia (amblyopia usually in the more hyperopic eye)
 - causes the less hyperopic eye to receive a clear image while the more hyperopic eye receives a blurred image so that its optic pathway does not develop normally
 - astigmatism
 - treat with glasses to correct refractive errors
 - patching is required if amblyopia persists > 4-8 weeks
- other
 - occlusion due to ptosis, cataract, retinoblastoma, corneal opacity
 - occlusion amblyopia: prolonged patching of good eye may cause it to become amblyopic

General Treatment

- correct the cause
- patching of good eye (duration is individually determined)
- monitor vision until age 8-9 years

LEUKOCORIA

- white pupil (red reflex is absent)

Differential Diagnosis

- retinoblastoma, retinal coloboma
- retinopathy of prematurity (ROP) (retrolental fibroplasia): associated with supplemental oxygen use in premature infants
- persistent hyperplastic primary vitreous (PHPV)
- total retinal detachment
- congenital cataract
- corneal scar

NASOLACRIMAL SYSTEM DEFECTS

- delay in duct formation for a few weeks after birth may be normal
- increased tearing, crusting and discharge (sometimes mucopurulent)
- treatment: massage over lacrimal sac
- usually resolve within 8 months
- consider referral for lacrimal probing if persistent at 9-12 months

OPHTHALMIA NEONATORUM

- newborn conjunctivitis
- Chlamydia* is most common cause
- other causes include: chemical (i.e. silver nitrate), bacterial (N. *gonorrhoea*, *S. aureus*, *Pseudomonas*), HSV
- gonococcal infection is the most serious threat to sight
- topical prophylaxis most commonly with erythromycin is required by law, less commonly with silver nitrate or providone-iodine

RUBELLA

- infection in the mother in first trimester
- the infant may suffer from any or all of the following:
congenital cataract, heart disease, deafness, microcephaly, microphthalmos, mental deficiency

OCULAR TRAUMA

Clinical Pearl

- ❑ **Always test visual acuity (VA) first! – medicolegal protection.**

BLUNT TRAUMA

- ❑ e.g. fist, squash ball
- ❑ history: injury, ocular history, drug allergy, tetanus status
- ❑ exam: VA first, pupil size and reaction, EOM (diplopia), external and slit lamp exam, ophthalmoscopy
- ❑ if VA normal or slightly reduced, globe less likely to be perforated
- ❑ if VA reduced, may be perforated globe, corneal abrasion, lens dislocation, retinal tear
- ❑ bone fractures
 - blow out fracture: restricted upgaze, diplopia
 - ethmoid fracture: subcutaneous emphysema of lid
- ❑ lids (swelling, laceration, emphysema)
- ❑ conjunctiva (subconjunctival hemorrhage)
- ❑ cornea (abrasions - detect with fluorescein and cobalt blue)
- ❑ anterior chamber (assess depth, hyphema, hypopyon)
- ❑ iris (prolapse, iritis)
- ❑ lens (cataract, dislocation)
- ❑ refer if you observe any of these signs of ocular trauma: decreased VA, shallow anterior chamber, hyphema, abnormal pupil, ocular misalignment or retinal damage

PENETRATING TRAUMA

- ❑ include ruptured globe +/- prolapsed iris, intraocular foreign body
- ❑ be suspicious if history of "metal striking metal"
- ❑ initial management: refer immediately
 - ABCs
 - don't press on eyeball!
 - check vision, diplopia
 - apply rigid eye shield to minimize further trauma
 - keep head elevated 30-45 degrees to keep IOP down

CHEMICAL BURNS

- ❑ alkali burns have a worse prognosis vs. acid burns because acids coagulate tissue and inhibit further corneal penetration
- ❑ poor prognosis if cornea opaque, likely irreversible stromal damage
- ❑ even with a clear cornea initially, alkali burns can progress for weeks (thus, very guarded prognosis)

Management

- ❑ IRRIGATE at site of accident immediately, with water or buffered solution
 - IV drip for at least 20-30 minutes with eyelids retracted in emergency department
- ❑ do not attempt to neutralize because the heat produced by the reaction will damage the cornea
- ❑ cycloplegic drops to decrease iris spasm (pain) and prevent secondary glaucoma (due to posterior synechiae formation)
- ❑ topical antibiotics and patching
- ❑ topical steroids (not in primary care) to decrease inflammation, use for less than two weeks (in the case of a persistent epithelial defect)

HYPHEMA

- ❑ bleed into anterior chamber, often due to damage to root of the iris
- ❑ may occur with blunt trauma

Management

- ❑ refer to Ophthalmology
 - patch, shield and bedrest x 5 days
- ❑ may need surgical drainage if hyphema persists or if re-bleed occurs

Complications

- ❑ risk of rebleed highest on days 2-5, resulting in secondary glaucoma, corneal staining, and iris necrosis
- ❑ never prescribe aspirin as it will increase the risk of a rebleed

OCULAR TRAUMA ... CONT.

BLOW OUT FRACTURES (see Plastic Surgery Chapter)

- blunt trauma causing fracture of orbital floor and orbital contents to herniate into maxillary sinus
- orbital rim remains intact
- inferior rectus and/or inferior oblique muscles may be incarcerated at fracture site
- infraorbital nerve may be damaged

Symptoms and Signs

- pain and nausea at time of injury
- diplopia, restriction of upgaze
- infraorbital and upper lip paresthesia (CN V2)
- enophthalmos, periorbital ecchymoses

Diagnosis

- plain films: Waters view and lateral
- CT: anteroposterior and coronal view of orbits

Management

- refrain from coughing, blowing nose
- systemic antibiotics may be indicated
- surgery if fracture > 50% orbital floor, diplopia not improving, or enophthalmos > 2 mm
- may delay surgery if the diplopia improves

SYMPATHETIC OPHTHALMIA

- severe bilateral granulomatous uveitis
- occurs after ocular trauma (usually penetrating and involving uveal tissue) or eye surgery, 10 days to years later
- possibly due to a hypersensitivity reaction to uveal pigment
- the injured eye becomes inflamed first and the other eye (sympathizing) second

Symptoms and Signs

- photophobia
- blurred vision
- red eye

Management

- if vision not salvageable in affected eye, enucleate to prevent sympathizing reaction
- if inflammation in sympathizing eye is advanced, treat with local steroids and atropine → cyclosporin

OCULAR EMERGENCIES

- these require urgent consultation to an ophthalmologist for management
- trauma, especially intraocular foreign bodies, lacerations
- corneal ulcer
- gonococcal conjunctivitis
- orbital cellulitis
- chemical burns
- acute iritis
- acute angle closure glaucoma
- central retinal artery occlusion (CRAO)
- retinal detachment
- endophthalmitis
- giant cell arteritis

OCULAR MEDICATIONS

TOPICAL OCULAR DIAGNOSTIC DRUGS

Fluorescein Dye

- water soluble orange-yellow dye
- green under cobalt blue light - ophthalmoscope or slit lamp
- stains damaged corneal and conjunctival and mucus epithelium and contact lenses

Anesthetics

- e.g. proparacaine HCl 0.5%, tetracaine 0.5%
- indications: removal of foreign body and sutures, tonometry, examination of painful cornea
- toxic to corneal epithelium and can lead to corneal ulceration and scarring with prolonged use, therefore NEVER prescribe

Mydriatics

- dilate pupils
- cycloplegic
- indications: refraction, ophthalmoscopy, therapy for iritis
- cholinergic blocking
 - paralyze iris sphincter i.e. dilation and cycloplegia

Table 6. Mydriatic Drugs and Duration of Action

| Drugs | Duration of action |
|-----------------------------|--------------------|
| Tropicamide 0.5%, 1% | 4-5 hours |
| Cyclopentolate HCL 0.5%, 1% | 3-6 hours |
| Homatropine HBr 1%, 2% | 3-7 days |
| Atropine sulfate 0.5%, 1% | 1-2 weeks |
| Scopolamine HBr 0.25%, 5% | 1-2 weeks |

- adrenergic stimulating
 - stimulate pupillary dilator muscles, no effect on accommodation
 - e.g. phenylephrine HCl 2.5% (duration: 30-40 minutes)
 - usually used with tropicamide for additive effects
 - side effects: hypertension, tachycardia, arrhythmias

GLAUCOMA MEDICATIONS

Beta-Adrenergic Blockers

- decrease aqueous humour formation
- nonselective beta-blockers (e.g. Timolol, Levobunolol, Metapranolol)
- systemic side effects: bronchospasm, bradycardia, heart block, hypotension, impotence, depression, exacerbation of congestive heart failure

Cholinergic Stimulating

- increases aqueous outflow
- e.g. pilocarpine, carbachol
- side effects: miosis, decreased night vision, headache, increased GI motility, decreased heart rate

Adrenergic Stimulating

- decrease aqueous production and increase flow
- e.g. epinephrine HCl, dipivifrin (Propine), brimonidine (Alphagan)
- side effects: contact allergy, hypotension in children

Carbonic Anhydrase Inhibitor

- decrease aqueous production
- e.g. oral acetazolamide (Diamox), topical dorzolamide
- side effects: renal calculi, nausea, vomiting, diarrhea, weight loss, aplastic anemia, bone marrow suppression, systemic acidosis
- side effects generally absent with topical preparations

Prostaglandin Agonists

- improves uveoscleral outflow
- e.g. latanoprost (Xalatan)
- side effects: iris colour change, lash growth, trichiasis

TOPICAL OCULAR THERAPEUTIC DRUGS

Decongestants

- weak adrenergic stimulating drugs (vasoconstrictor)
- e.g. naphazoline HCl 0.012%, phenylephrine HCl 0.12%, tetrahydrazaline 0.05%
- rebound vasodilation with over use can exacerbate angle closure glaucoma

OCULAR MEDICATIONS ... CONT.

Corticosteroids

- never prescribed by primary care physician unless emergency indications
- complications
 - potentiates herpes simplex keratitis and fungal keratitis as well as masking symptoms (within days)
 - posterior subcapsular cataract (within months)
 - increased IOP, more rapidly in steroid responders (within weeks)

Antibiotics

- indications: bacterial conjunctivitis, keratitis, or blepharitis
- commonly as topical drops or ointments, may give systemically
- e.g. sulfonamide (sodium sulfacetamide, sulfisoxazole), gentamicin, erythromycin, tetracycline, bacitracin, polymyxin

OCULAR DRUG TOXICITY

Table 7. Drugs with Ocular Toxicity

| | |
|-------------------------------|--|
| Amiodarone | Corneal microdeposits and superficial keratopathy |
| Chloroquine | Bull's eye lesion at macula Secondary keratopathy |
| Chlorpromazine | Anterior subcapsular cataract |
| Contraceptive Pills | Decreased tolerance to contact lenses Migraine Optic neuritis Central vein occlusion |
| Digitalis | Yellow vision Blurred vision |
| Ethambutol | Optic neuropathy |
| Haloperidol (Haldol) | Oculogyric crises Blurred vision |
| Indomethacin | Superficial keratopathy |
| Isoniazid | Optic neuropathy |
| Nalidixic Acid | Papilloedema |
| Steroids | Posterior subcapsular cataracts Glaucoma Papilloedema (systemic steroids) Increased severity of HSV infections (geographic ulcers) Predisposition to fungal infections |
| Tetracycline | Papilloedema |
| Thioridazine | Pigmentary degeneration of retina |
| Vitamin A Intoxication | Papilloedema |
| Vitamin D Intoxication | Band keratopathy |

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