Anatomy and Physiology of the Eye

Important eye anatomy, external to internal:

1. External structures:
   a. Eyelids – protection, tear film production/distribution, tear flow/drainage
   b. Tears – lubrication, $O_2$ transmission, visual clarity, immunological protection
   c. Conjunctiva – $O_2$ transmission, tear production, tear film adherent → defence, protection and integrity against micro-organisms and infection
   d. Sclera – protection, ocular rigidity, attachment of extraocular muscles

2. Anterior chamber:
   a. Cornea – refraction (2/3 total power), clarity, protection against micro-organisms, $O_2$ transmission, ocular rigidity, heavily innervated → easily irritated
      i. Epithelium, Bowman’s membrane, stroma, Descemet’s layer, endothelium
      ii. Endothelium pumps $H_2O$ out, keeping it relatively dehydrated (→ transparent)
   b. Lens – refraction (1/3 total, variable power), accommodative mechanism, smooth interface with aqueous humour
      i. Epithelium, fibrous cells → proliferate → pushed centrally → acellular
      ii. Gets larger and stiffer with age, can opacify (cataract)
   c. Iris and pupil – restriction of light (variable aperture) → reduction of light scatter
   d. Ciliary body/epithelium – suspend lens, accommodation, aqueous production
      i. Circular muscle suspends lens via zonules – contraction → higher power
      ii. Aqueous humour flow:
         1. Ciliary body → pupil → anterior chamber
         2. Trabecula → canal of Schlemm → collecting channels

3. Posterior chamber:
   a. Vitreous chamber – vitreous humour maintains shapes, protective in trauma
   b. Choroid – provides oxygen and nutritious to outer retina
   c. Retina – visual sensation, supported by retinal pigment epithelium (photopigment formation, photoreceptor renewal, reduced damage from scatter light and nutrition)
      i. Three layers of cell bodies, two layers of synapses:
         1. Cells – rods/cones, bipolar cells, ganglion cells
         2. Synapses – photoreceptor-bipolar synapse, bipolar-ganglion synapse
      ii. Rod and cone distribution varies by site e.g. fovea (cones), peripheral (rods)
   d. Optic nerve – transmits electrical signals via the optic chiasm, optic radiation, lateral geniculate nucleus and occipital cortex for further processing of the visual image

Retinal physiology:

1. Humans are able to detect light of a wavelength between 400-700nm
2. When a photon collides with the retina it is transmitted through an inverted design (moronic engineering, really) to the photoreceptors where photochemical transduction occurs
3. Rods and cones differ in terms of spectral sensitivity, light and dark adaptation, minimum detectable number of photons and spatial/temporal summation (can you spell “buzz-word”?)
4. The functioning of rods and cones vary under scotopic (low light) and photopic (daylight) light levels, location within the visual field, retinal location as well as stimulus size and position
   a. Rods are more suited to processing visual images seen at low light levels (twilight, scotopic) while cones process images at high light levels (daytime, photopic)
   b. Cones are responsible for detection of colour and fine details (acuity) and are concentrated centrally, while rods are responsible for detection of peripheral motion

Visual performance:

1. Form/spatial vision – acuity (varies with contrast, brightness, eccentricity and procedure)
2. Colour vision – hue, saturation, brightness, interactions (saturation testing, Ishihara plates)
3. Binocular vision – needs clear images in both eyes, coordination and intact cortical fusion

The Squinting Child

A squint (strabismus) is a misalignment of the visual axes due to dysfunction of the 12 extraocular muscles (6 for each eye) with far-ranging consequences particularly in younger children.

1. Epidemiology
   a. Squint affects 3-4% of children, and amblyopia (‘lazy eye’) affects 5% of children
   b. 30-50% develop secondary visual loss of untreated, and early recognition is crucial
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2. Pathophysiology
   a. Orthotropia – central fixation, smooth pursuit at 2-3/12; stable alignment by 4/12
   b. Misalignment leads to confusion and double vision → suppression of conflicting input → strabismic amblyopia (decreased visual acuity, potentially reversible early in life)
      i. Unilateral → preference for undeviated eye, loss of vision for deviated eye
      ii. Alternating bilateral → normal vision development as both eyes used in turn
   c. The critical period of visual development (tightly linked to alignment) is between birth and 8 years of age, though visual acuity increases exponentially in the first few months
      i. Interval between onset of constant strabismus and treatment in a child older than 3 years is a key determinant of visual outcome
      ii. Similarly, strabismus of duration <5% a day is associated with better outcome
   d. Note that any strabismus may be associated with a neurological or visual pathway abnormality, or may be an isolated phenomenon
      i. Paralytic strabismus – nerve palsies (e.g. CN III → oculomotor palsy)
      ii. Vision impairment – refractive errors, media opacity, retinal disease (retinoblastoma, retinopathy), optic nerve disease (coloboma, hypoplasia)

3. Nomenclature:
   a. Prefixes indicate direction – eso- in, exo- out, hyper- up, hypo- down
   b. Suffixes: -tropia – misalignment is always manifest; -phoria – latent misalignment
   c. Comitant – angle of deviation between eyes remains constant in different directions

Clinical features:
1. History:
   a. Age of onset, variability (worse when child tired or ill), intermittent or constant
   b. One eye or both (same time or alternating), head tilting/turning, family history
   c. Double vision suggests recent onset, paresis points to nerve palsies and CNS
2. Examination
   a. General – interaction, visual behaviour, abnormal head postures, dysmorphology
   b. Fixation behaviour – central, steady, maintained
   c. Visual acuity – Snellen chart, illiterate E chart, Sheridan-Gardner single letter matching, Kay picture cards, pattern matching
   d. Ocular alignment:
      i. Hirschberg’s test (corneal light reflex) – light falls on different parts of cornea
      ii. Brückner test (red reflex, both at once) – dimmer in one eye → misalignment
      iii. Cover test – movement of uncovered eye indicates misalignment
      iv. Alternate cover test – deviating eye moves rapidly to compensate
   e. Ocular mobility in 9 cardinal directions (determines comitant or incomitant)
3. Clinical syndromes:
   a. Pseudostrabismus – flat broad nasal bridge, prominent epicanthal folds or narrow intercanthal distance; corneal reflex and cover tests differentiate from true squint
   b. Non-paralytic strabismus:
      i. Infantile esotropia (<6/12) – patching undeviated eye allows normal visual development in the other; glasses for far-sightedness, surgery before 2yrs
      ii. Accommodative esotropia (6/12-7yrs) – most common, eye accommodates to correct a hyperopic/blurred image → esotropia (intermittent → constant)
         1. Glasses for hyperopic refractive error (may need bifocals)
         2. Surgery for significant residual deviation
      iii. Intermittent exotropia (6/12-4yrs) – outward deviation when distance focusing, worse with fatigue/illness; surgery for severe/frequent exotropia
      iv. Constant exotropia – associated with neurologic disease or abnormalities of the bony orbit; eye-patching, surgery for cosmetic appearance only
   c. Paralytic strabismus:
      i. CN III – usually congenital; esotropia with downward deviation and ptosis, pupillary dilation may be present; surgical correction, botulinum toxin
      ii. CN IV – congenital > acquired (trauma); weak SO muscle → hypertropia, head tilts to opposite side to minimise double vision; surgery corrects both
      iii. CN VI – congenital (Duane retraction syndrome (odd innervation → ocular dysmotility) or acquired (infection, ICP); crossed eyes with limited lateral movement, head turns to same side to maintain binocular vision; surgery
Vision screening in childhood is recommended to detect vision impairment, squint or serious ocular pathology:

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Tester</th>
<th>Tests</th>
<th>Condition</th>
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</thead>
<tbody>
<tr>
<td>0-6 weeks</td>
<td>GP</td>
<td>External eye exam</td>
<td>Congenital abnormalities</td>
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<tr>
<td></td>
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<td>Red reflex</td>
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<td>Infant (3-15 months)</td>
<td>Plunket</td>
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<td></td>
<td></td>
<td>Squint</td>
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<tr>
<td>18-36 months</td>
<td>Vision tester, Plunket</td>
<td>Photorefraction</td>
<td>Squint, Refractive errors</td>
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<tr>
<td>Preschool (3.5-5yrs)</td>
<td>Vision tester, Plunket</td>
<td>Visual acuity (4m letter matching)</td>
<td>Amblyopia, Refractive errors</td>
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<tr>
<td>New entrant (5-6 years)</td>
<td>Hearing/Vision tester</td>
<td>Visual acuity (4m LMT or Snellen)</td>
<td>Amblyopia, Refractive errors</td>
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<tr>
<td>Form I and IV (10-15 years)</td>
<td>Hearing/Vision tester</td>
<td>Visual acuity (Snellen)</td>
<td>Refractive errors</td>
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Bottom line: refer all children with strabismus to an ophthalmologist, particularly if a paralytic strabismus is suspected or there is loss of the reflex (refer urgently).

- **Symptoms and Signs of Eye Disease**

  Clinical history should ascertain the nature and characteristics of the patient’s complaint; particularly the duration of and the circumstances surrounding the onset of symptoms, whether onset was gradual/sudden, unilateral/bilateral; whether the problem is intermittent/persistent/progressive; and if there are any alleviating or aggravating factors. Past and family ocular history are also useful.

  **Key symptoms** (order doesn’t really make sense, but more sense than the handout)
  1. **Visual loss** – unilateral (ocular) or bilateral (CNS), sudden (CVA) or gradual (cataract), partial (retinal detachment, macular degeneration) or complete, transient/constant/progressive
     a. **Photopsia** – flashes of light with new floaters \(\rightarrow\) vitreous degeneration/detachment
     b. **Diplopia** – monocular (cataract) or binocular (strabismus), vertical or horizontal
     c. **Photophobia** – due to glare (cataract), inflammation (uveitis, keratitis)
  2. **Pain** – sharp (foreign body, corneal abrasion) or dull, intermittent/constant, deep/superficial, alleviating/aggravating factors (e.g. reading or accommodation – iritis)
     a. **Redness** – unilateral (iritis/glaucoma) or bilateral (conjunctivitis), distribution (diffuse – conjunctivitis, sectorial – episcleritis/scleritis, circumcorneal – keratitis/anterior uveitis)
     b. **Discharge** – purulent (infective) or mucous/watery (allergic)
  3. **Epiphora and lacrimation** – 2” to irritation or inflammation, increased tearing or poor drainage
     a. **Itchiness** – history of hay fever or allergic eye disease, blepharitis
     b. **Grittiness/irritation** – ocular surface abnormality (dry eye, small erosion, foreign body)

**Examination:**
  1. **Vision** – near and distance vision (pinhole may improve refractive error), colour vision (optic neuritis \(\rightarrow\) red desaturation, Ishihara plates for congenital and acquired colour defects)
  2. **Visual fields** – look at face (missing parts \(\rightarrow\) hemianopia, centra scotomas), finger counting centrally, white pin or peripheries and red pin to plot blind spot
  3. **Eye movements** – corneal reflexes, cover-uncover tests, 9 cardinal directions
  4. **Pupils** – mobility, shape, equality; direct and consensual response (lack of constriction or relative dilatation in one pupil suggests optic nerve or severe retinal disease), accommodation
  5. **External eye** (may use fluorescin) – eyelids, conjunctiva, cornea, anterior chamber, iris
  6. **Posterior segment** – red reflex, fundoscopy – optic nerve \(\rightarrow\) vessels \(\rightarrow\) retina \(\rightarrow\) macula

- **Vision Loss**

  Vision loss can be subdivided anatomically into 3 groups – lesions anterior to the chiasm (unilateral visual loss), chiasmal lesions (bitemporal visual loss) and retrochiasmal lesions (hemianopic field defects). Aspects of history that should be considered in assessment:
  1. Speed of onset – acute (emergency) or gradual
  2. Extent of visual loss – total/central/partial/hemifield, unilateral/bilateral

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3. Other ocular symptoms – photopsia, metamorphopsia, ocular pain (acute red eye differential)
4. Systemic symptoms – headache, jaw claudication (giant cell arteritis)
5. Systemic disease – hypertension, diabetes, cardiovascular disease

Acute loss of vision (all ophthalmic emergencies)
1. Ischaemic optic neuropathy – arteritic (giant cell) or non-arteritic
   a. Aetiology – DM, SLE, migraine, severe haemorrhage, hypertension (>40), GCA (>60)
   b. History – age >50 (usually 70s-80s), M=F, amaurosis fugax, symptoms of GCA (headache, jaw claudication, scalp pain, constitutional symptoms, PMR)
   c. Examination – afferent papillary defect, disc swelling, flame-shaped haemorrhage
   d. Investigations – ESR, temporal artery biopsy (note skip lesions)
   e. Majority have profound vision loss, 20% with antecedent transient loss
      i. 1/3 lose vision in other eye within 2 days, 1/3 in one week, 1/3 in one month
      ii. Thrombocytosis with acute visual loss is a risk factor for permanent blindness
      iii. Glucocorticoids may not be sufficient – consider adding anti-platelet drugs
2. Central retinal artery occlusion:
   a. Aetiology – hypertension, DM, carotid atherosclerosis (Hollenhorst plaque), cardiac valve disease (calific plaque), vasculitis, vasospasm, trauma, cholesterol emboli
   b. History – sudden painless visual loss (central scotoma, peripheral field)
   c. Examination – afferent pupillary defect, milky white retina in posterior pole (segmental blood flow), optic disc becomes pale over a month
   d. Irreversible damage to retina occurs within 90 minutes of occlusion, although 10% of patients retain central vision due to patent cilioretinal artery perfusing fovea
   e. Treatment – digital globe pressure, ↓ IO pressure, 95% CO2/5% O2, Ca+2 blocker
3. Retinal vein occlusion – central retinal vein or branch retinal vein
   a. Aetiology – HT, glaucoma, DM, hyperopia, mitral valve or collagen vascular disease
   b. Presentation >60yrs, extensive haemorrhage of retinal layers, cotton-wool spots, tortuous veins, macular oedema (non-ischaemic, may → ischaemic – 10% at 6/12)
   c. Photocoagulation no longer recommended prophylactically, macular grid laser not effective for macular oedema, IPA studies ongoing (streptokinase → haemorrhage)
4. Vitreous haemorrhage
   a. Aetiology – diabetic retinopathy, retinal tear, retinal vein occlusion with proliferative retinopathy, posterior vitreous detachment
   b. Bleeding may be from normal vessels, diseased retinal vessels or from new vessels
   c. Complications – haemosiderosis bulbi, glaucoma (ghost cell, haemolytic and haemosiderotic types), persistent blood cell debris
   d. Treatment – pars plana vitrectomy
5. Retinal detachment – separation of retina from retinal pigment epithelium
   a. Rhegmatogenous (liquid vitreous tracks subretinally), exudative (fluid accumulation from leaky vessels or abnormal RPE), tractional (proliferative retinopathy, iatrogenic)
   b. Symptoms – floaters, photopsia, retinal tear, visual field loss ± macular detachment
   c. Examination – undulated/folded retina, shifting fluid, may be smooth/immobile
   d. Treatment – laser photoocoagulation, cryotherapy, scleral buckling, vitrectomy
6. Retrobulbar/optic neuritis:
   a. Pain on moving eyes, F>M, ages 15-45, associated with multiple sclerosis, SLE (1%)
   b. Central scotoma, afferent pupillary defect (unless bilateral), optic disc swelling/pallor, Uhthoff's transient visual obscuration (with exertion or temperature)
   c. Methyldprednisone 250mg iv q6h for 3 days, then prednisone 1mg/kg po for 11 days
   d. 20% progress to MS within 2 years, 45-80% within 15 years (delayed by interferon-β)
7. Cerebral infarction - involving optic tract, radiation or occipital cortex (see neurology notes)

Gradual loss of vision:
1. Age-related macular degeneration
   a. Non-neovascular (dry) – drusen (pale yellow-white deposits of vesicles and abnormal collagen between RPE basement membrane and Bruch's membrane), RPE atrophy
      i. Treatment – monitor activity with Amsler grid, low vision aids
      ii. Balanced diet with fruits, leafy vegetables, limited evidence for anything else
   b. Neovascular (wet) – choroidal neovascularization (breaks in Bruch's membrane → detachment of RPE, retina, haemorrhage, lipid deposits), disciform scarring
      i. Treatment – photoocoagulation, photodynamic therapy, ?thalidomide, INF-α
2. **Chronic open angle glaucoma**
a. Affects 1% of persons between 60-70yrs, 3% of the population >75yrs, family history
b. Aetiology – myopia, hypertension, DM, smoking, age, family history, elevated IOC
c. Characterised by open anterior chamber angle, chronically raised intraocular pressure, cupping of the optic disc, progressive loss of visual fields (nasal, temporal)
d. Treatment – lower IOP by lowering aqueous humour production or increasing outflow
   i. β-blockers, prostaglandins, α₂-blockers, carbonic anhydrase inhibitor
   ii. Argon laser trabeculoplasty, surgical trabeculectomy

3. **Senile cataract** – see cataracts lecture
4. **Diabetic retinopathy** – see endocrinology lectures
   a. Non-proliferative:
      i. Mild – microaneurysms, intraretinal haemorrhage (dots, blots, flames), hard exudates, macula oedema
      ii. Moderate/severe – four quadrants of haemorrhages, two quadrants of venous bleeding, one quadrant of intraretinal microvascular abnormalities
   b. Proliferative – neovascularisation (disc, peripheral, iris), haemorrhage
5. **Extreme myopia** – greater than -8.0 to 10.0 dioptres → myopic macular degeneration, chronic open angle glaucoma, cataract, retinal detachment
6. **Others** – nutritional amblyopia (tobacco/alcohol), toxic optic neuropathies, retinitis pigmentosa

### Lasers in Ophthalmology

#### Principles of laser therapy:
1. **Thermal effects** → photocoagulation
2. **Mechanical effects** → photodisruption
3. **Evaporation** → photoablation

#### Different laser uses:
1. **Argon (514nm)** – retinal laser photocoagulation, proliferative diabetic retinopathy, retinal vein occlusions, retinopathy of prematurity, retinal breaks, macular oedema/degeneration
2. **Nd:YAG (1064nm)** – laser capsulotomy (cataract surgery), peripheral iridectomy (glaucoma)
3. **Infrared laser (810nm)** – cyclophotoablation (glaucoma), trans-pupillary thermotherapy (melanin tumours, age-related macular degeneration)
4. **Excimer laser (152nm)** – photorefractive surgery

#### Laser safety:
1. **Class 1** – incapable of causing tissue damage
2. **Class 2** – injury with chronic exposure, safety afforded by aversion
3. **Class 3** – instantaneous injury
4. **Class 4** – scattered light poses hazard

### The Acute Painful Red Eye

#### General principles:
1. **History** – onset, duration and nature of symptoms; systemic symptoms, past ocular history
2. **Examination** – visual acuity, pen torch, fluorescein, local anaesthetic drops, slit lamp
   a. Note eyelid inflammation, distribution of redness, corneal epithelial defects (e.g. dendritic ulcer suggests HSV), corneal clarity (ground-glass suggests *Pseudomonas*)
3. **Management** – treat the cause, symptomatic relief

#### Common causes:
1. **Conjunctivitis**
   a. Eye and upper eyelid redness and discharge, conjunctival injection, scleral injection
      i. Follicles (aggregations of lymphoid tissue), papillae (chronic irritation)
      ii. Discharge – purulent (bacteria), mucopurulent (*Chlamydia*), mucoid/watery (allergic), watery (viral)
   b. Aetiology – infective (*Neisseria, Chlamydia*, viral), post-infective (shingles affecting V₁), allergic (atopy, irritants), sicca syndromes (e.g. Sjogren’s syndrome, vitamin A deficiency, ocular cicatricial pemphigoid, Stevens Johnson syndrome, drug-induced
   c. Treatment varies by aetiology, but in general swab → culture/sensitivity → treat
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1. **Bacterial** – purulent discharge + lymphadenopathy ➔ urgent IV ceftriaxone, topical antibiotics; otherwise antibiotic drops for 7-10 days (chloramphenicol)
   i. Chloramphenicol – may need topical and systemic tetracycline for 2-3 weeks
   ii. **Viral** – broad-spectrum antibiotic drops may speed recovery, antiviral agents for herpesvirus infections (DO NOT USE STEROIDS)
      iv. **Allergic** – eliminate irritant, antihistamines, topical NSAID, local steroids
      v. **Sicca syndromes** – treat cause, artificial tears, punctal blockage, tarsorrhaphy

2. **Keratitis**
   a. Aetiology – neurotrophic keratopathy, dry eyes, rheumatoid arthritis, corneal infection
   b. Reduced vision with pain, corneal epithelial erosion, hyperaemia, hypolacrimation
   c. IGF-1 may help heal cornea, substance P is synergistic for neurotrophic keratopathy

3. **Uveitis/iritis**
   a. **Anterior (iritis)** – ocular pain, redness, miosis, photophobia, decreased vision
      i. Associated with HLA-B27 spondyloarthropathies, ulcerative colitis, trauma, sarcoid, JCA, syphilis, herpesvirus, Kawasaki disease, Behcet’s syndrome
      ii. Topical corticosteroids, cyclosporine/methotrexate/azathioprine if refractory
   b. **Posterior (chorioretinitis)** – blurred vision, scotoma, floaters
      i. Associated with toxoplasmosis, retinal vasculitis, relapsing polychondritis, other infection, acute retinal necrosis (herpesvirus), retinochoroidopathies
      ii. Usually needs systemic steroids or depot triamcinolone injection
   c. **Panuveitis** – combination of the above two:
      i. Associated with herpesviruses, HIV, bacteria (TB, MAI, brucella), parasites (Toxoplasma, Pneumocystis), Lyme disease, syphilis, ocular histoplasmosis
      ii. Weird causes to scare your classmates with – pars planitis, Fuchs’s heterochromic cyclitis, sympathetic ophthalmia, acute posterior multifocal placoid pigment epitheliopathy, Vogt-Koyanagi-Harada syndrome
   d. Visual loss is 2° to cataract, glaucoma, macular oedema, band keratopathy (Ca**2+** deposition in cornea), vitreous opacification

4. **Acute closed angle glaucoma** – iris blocks the trabecular meshwork in the angle of the eye
   a. Aetiology – closure of ciliary body/iris angle, inability of fluid to escape from posterior chamber, pressure in posterior chamber, creping angle closure (anterior synechiae)
   b. History – F>M (1/1000 in >40yrs), sudden onset pain, red eye with blurred vision and haloes around lights, headache, nausea and vomiting
   c. Examination – fixed, mid-dilated pupil, foggy iris (difficult to visualise), increased IOP
   d. Treatment – β-blockers, carbonic anhydrase inhibitors, paracentesis, laser iridectomy (gold standard, treat other eye), surgical iridectomy – can get chronic angle closure

5. **Scleritis**
   a. Aetiology – idiopathic, HSV, collagen vascular diseases (RA + ulcers bad prognosis)
   b. Violaceous hue, globe tender to touch with fixed blood vessels (focal scleral injection, do not blanch with phenylephrine), referred severe/piercing pain to jaw and forehead
   c. Therapy – NSAIDs, systemic glucocorticoids and immunosuppressives

6. **Episcleritis**
   a. Aetiology – usually idiopathic, may be associated with collagen vascular disease
   b. More acute, milder pain, with mild focal injection (vessels blanch with phenylephrine)
   c. Treatment – generally self-limited, use lubricating drops ± topical NSAIDs

7. **Subconjunctival haemorrhage**
   a. Aetiology – trauma, Valsalva, bleeding disorders, hypertension, Kaposi sarcoma
   b. Resolves spontaneously over several weeks

8. **Ocular trauma** – see ocular trauma lecture

9. **Herpes zoster ophthalmicus**
   a. Corneal dendrites, stromal keratitis, anterior uveitis, raised IOP, oculomotor nerve palsies, acute retinal necrosis, postherpetic scarring and neuralgia
   b. Hutchinson’s sign – vesicles forming on the tip of the nose ➔ V1 nerve involvement
   c. Treatment – antiviral agents, cycloplegics, steroids if severe to prevent scarring

• **Introduction to Ocular Pharmacology**

Anatomical and physiological issues:

1. **Blood-eye barrier:**
   a. Tight capillary junctions at the retina and iris
   b. Tight junctions at the ciliary epithelium and retinal pigment epithelium
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c. Epithelium lipophilic, stroma hydrophilic and endothelium lipophilic – so drugs that pass through the cornea must be both ionised and deionised

2. Compartments – cul-de-sac (fornix), anterior camber, vitreous cavity, retro/periorbital space
   a. Cul-de-sac normally 7-10μL, can expand transiently to 30μL
   b. Topical drops usually 40-70μL (i.e. far more than necessary to compensate for loss)
   c. A second drop (i.e. another drug) can lead to a washout effect – reduction to 45% at 30 seconds, 17% at 120 seconds and <5% at 5 minutes

3. Tear film turnover – 15% per minute, 30% after an eye drop
   a. Blinking → 15% of drug remains at 5 minutes
   b. Pouch method with eye closure → 53% remains at 5 minutes

4. Elimination is by initial overflow, aqueous humour outflow, diffusion into blood vessels and inactivation via metabolism

Routes of administration – note efficacy is also affected by solubility, pH, particle size, suspension
1. Topical (prolonged action, non-stinging) – drugs (G.), ointment (Oc.)
2. Periocular injection – subconjunctival (higher levels, long-acting), subtenial, peri/retrobulbar
3. Intraocular – intravitreal injection, devices
4. Oral – antibiotics, antivirals, ocular antihypertensives
5. Intravenous – limited use in ophthalmology

Most medications are applied by topical drop due to rapidity of intra-ocular penetration through the cornea – on the other hand, ointments provide a more prolonged action at the expense of visual blurring. A combination (daytime drops, night-time ointment) gives maximal effect in diseases such as severe conjunctivitis or acute uveitis. Periocular injection is infrequent, usually in a surgical setting.

Commonly used ophthalmic agents – most in order of preference
1. Anaesthetics (toxic to epithelium) - G. benoxinate, amethocaine, lignocaine (can be injected)
2. Antibiotics – G. chloramphenicol, fluoroquinolones, cephalosporins/aminoglycosides
   a. Note that chloramphenicol drops can → bone marrow suppression (mainly in kids)
3. Antivirals – Oc. acyclovir, G. idoxuridine (rarely)
4. Corticosteroids – G. prednisone, fluorometholone, dexamethasone
   a. Indications – post-op inflammation, inflammatory disease (uveitis), atopy, transplant
   b. Adverse effects – ocular hypertension, posterior subcapsular cataract, HSV activation
   c. Contraindicated in the acute red eye without ophthalmologist review
5. Glaucoma drugs:
   a. Reduce production
      i. β blockers – G. timolol, betaxolol, levobenolol
      ii. Carbonic anhydrase inhibitors – G. dorzolamide, po acetazolamide
      iii. α agonists – brimonidine
   b. Increase drainage
      i. Sympathomimetics – adrenaline, dipivefrine
      ii. Parasympathetics – pilocarpine (myopic shift, can → retinal detachment)
      iii. Prostaglandin analogues – latonoprost (causes pigment melanogenesis)
6. Mydriatics:
   a. Parasympathetics – G. tropicamide 1% (3-4hrs), cyclopentolate 1%, atropine
      i. Atropine has a ridiculously silly half-life (~2 weeks)
      ii. Fatal adult dose for atropine is 100mg, for a 4kg baby this is 10mg (20 drops)
   b. Sympathomimetics – phentolamine 2.5% or 10%
7. Tear supplements – Lacri-Lube, liquifilm tears, viscotears, hyromellose
8. Others – G. Na cromoglycate (mast cell stabiliser), vital stains (G. fluorescein, rose Bengal)

Ocular Trauma

General principles of assessment:
1. History – determine the nature of the injury, first-aid treatment given and past medical history
   a. Low velocity – unlikely to have caused a penetrating injury
   b. High velocity – must be treated with a high degree of suspicion for penetration
   c. Thermal injuries – may cause severe ocular damage, usually needs referral
   d. Chemical injuries – potentially extremely dangerous, irritate and refer urgently
2. Examination – “I can’t examine the eye because the patient won’t let me” is not an excuse
   a. Pain relief via topical anaesthetic drops e.g. benoxinate, lignocaine or amethocaine
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b. Use bright light and magnification or slit lamp if available – note that foreign bodies are often small but cause intense pain or irritation
c. Visual acuity, conjunctiva (including subtarsal), cornea (fluorescein, corneal reflex), pupil shape/reaction, hyphaema (anterior chamber), red reflex, ophthalmoscopy

3. Referral guidelines:
   a. Immediate – chemical burn, corneal laceration, globe perforation
   b. 24 hours – blunt trauma, corneal abrasion, foreign body
   c. Late – extracted foreign body, minimal trauma, late presentation

Classification of ocular trauma:

1. Combined injuries:
   a. Lid/orbit and eyeball injury
   b. Lid/orbit and eyeball injury with non-ophthalmic injury (may be life-threatening)

2. Injuries to eyelids and orbit – note that the eyes may show many signs of other head trauma
   a. Blunt eyelid trauma – ‘black eye’ → massive bruising, lid swelling, blood tracking across the nose; swelling may mask ocular or other orbital trauma
   b. Eyelid lacerations – partial thickness (suturing, gluing), margin involvement (reconstruction in layers), involvement of canaliculae and tarsal ligaments
   c. Retrobulbar haemorrhage – blunt or penetrating trauma causes bleeding from orbital vessels (note that the globe is a fixed pyramidal space), may cause compression and damage to the orbital contents; proptosis, altered pupils, decreased visual acuity
   d. Fractures:
      i. Blow-out fracture – sudden ↑IOP → ‘blow-out’ of inferior and medial orbital walls → lid bruising, diplopia, enophthalmos, anaesthesia along infraorbital nerve; avoid blowing nose, prophylactic antibiotics, surgery if severe
      ii. Facial fractures – trauma to facial bones can cause damage to orbital walls and hence impact on orbital contents and ocular function
         1. Le Fort I – transverse maxillary fracture above dental apices traversing pterygoid plate; nasal complex stable
         2. Le Fort II – fracture through frontal process of maxilla, through orbital floor and pterygoid plate; midface is mobile
         3. Le Fort III – complete craniofacial separation; differs from Le Fort II by extending through the nasofrontal and frontozygomatic sutures

3. Injuries to the eyeball
   a. Blunt ocular trauma → hyphaema, usually partial with formed fluid level of blood; may mask other ocular damage (iris damage, angle recession, lens subluxation, retinal damage, globe rupture). Treated by rest (reduces re-bleeding risk), return if painful
   b. Penetrating trauma:
      i. Scleral lacerations (partial/full thickness) – may be masked by haemorrhage; if full thickness dark red choroid may prolapse
      ii. Corneal wounds → flattened anterior chamber, pupil distortion, iris prolapse
   c. Corneal abrasion → intense pain, photophobia, blepharospasms; usually only seen as bright green/yellow areas when stained with fluorescein and illuminated by blue light; treatment by topical antibiotic ointment ± padding (resolves in 1-3 days)
   d. Subconjunctival haemorrhage – bright red blood beneath transparent conjunctiva; visually striking but usually insignificant (though can mask more severe pathology)
   e. Foreign material:
      i. Intraocular foreign bodies – B-scan/2D echography, high resolution CT (MRI absolutely contraindicated if magnetic); treatment is surgical, infection risk
      ii. Chemical burns – most require early irrigation, if potent substance may need copious irrigation until pH normal; refer all cases particularly with foreign body

Special considerations:

1. Subtarsal foreign bodies – material may trap in the subtarsal sulcus (2mm from lid margin)
   a. Clues – history of foreign body entry with sensation but no foreign body visible
   b. Action – instill fluorescein, look for linear abrasions of superior cornea, evert eyelid

2. Penetrating injury – severe and sight-threatening
   a. Clues – history of high velocity or suspected intraocular foreign body, distorted pupil, hyphaema or haemorrhaphalmos
   b. Action – protect the eye, refer to an ophthalmologist
3. **Children** – may be difficult to examine and should be referred (even then may need EUA)

4. **Type of foreign body material:**
   a. Organic – e.g. agricultural → fungal infection
   b. Metallic – iron → corneal rust ring, siderosis/degeneration, copper → endophthalmitis
   c. Glass or plastic – usually inert, may not need extraction

- **Cataracts**
  
  **Cataracts** are complete or partial opacifications of the crystalline lens that prevent transmission of a clear image. They affect 50% of the population above 70yrs and 100% of those over 90yrs, and are the most common cause of preventable blindness worldwide.

  1. **Classification**
     a. Subcapsular – directly under the outer lens capsule
        i. Anterior – may not affect vision considerably
        ii. Posterior (‘frost on a cold mug’) – interferes with vision (glare, axial position)
     b. Cortical (peripheral white spokes) – within cortex between capsule and nucleus
     c. Nuclear – within the nuclear material
  
  2. **Aetiology:**
     a. Congenital – 1/3 autosomal dominant, 1/3 due to other syndromes, 1/3 idiopathic
     b. Acquired:
        i. Prolonged intraocular inflammation - uveitis
        ii. Metabolic disturbances – diabetes, galactosaemia, Wilson's disease
        iii. Degeneration – lens sclerosis (compaction of fibres) → myopia, yellowing
        iv. Drugs – glucocorticoids (systemic/inhaled – posterior), radiation/UV exposure
        v. Trauma

  **Clinical features:**
  
  1. **History** – begins with normal vision with changes at variable rates, symptoms include:
     a. Glare with halo effect around lights
     b. Generalised blurriness with loss of best corrected visual acuity
     c. Induced myopic shift leading to ‘second sight’ with improved near vision
     d. May have monocular diplopia (as opposed to diplopia from motility disturbance)
     e. End stage is total blindness
  
  2. **Signs:**
     a. Reduced Snellen visual acuity, uncorrected refractive error
     b. Normal pupillary response (no relative afferent pupillary defect)
     c. Attenuated red reflex
     d. Slit-lamp examination shows lens opacification

  3. **Management:**
     a. Do nothing (visual acuity better than 6/12 if not symptomatic)
     b. Surgery – requires vision correction with contact lenses, glasses or intraocular lenses
        i. Intracapsular extraction – complete removal of lens, rarely done in 1st world
        ii. Extracapsular extraction – removal of small section of the anterior capsule, cortex and nucleus, leaving the posterior capsule in (allows lens implant)

  4. **Timing of surgery** – NZ prioritisation questionnaire ranks patients based on:
     a. Clinical modifiers (age-related macular degeneration, DM retinopathy): -10 to +20
     b. Work and independence: 0 to +10
     c. Non-vision physical disabilities: 0 to +10
     d. ADL questionnaire: 0 to +13
     e. Driving: 0 to +7

- **Introduction to Ophthalmic Surgery**

  **Cataract surgery:**
  
  1. **Phacoemulsification** is used for 98% of all cataract extractions in NZ:
     a. 3-4 mm shelving incision in cornea or sclera, viscoelastic maintains anterior chamber
     b. 4-6 mm hole in anterior capsule allows passage of a phaco-probe to emulsify/remove
        the nucleus, with any remaining strands of cortex removed with an aspiration probe
     c. Remaining intact capsular bag is filled with viscoelastic, then acrylic or silicone
        intraocular lens folded and inserted into the capsular bag
     d. Wound is usually self-sealing and does not require sutures

  2. **Extracapsular cataract extraction** used if phacoemulsification is contraindicated (dense lens)
Ophthalmology

a. 8-10 mm incision made in cornea or limbus and a hole made in the anterior capsule
b. Intact nucleus is extracted, with a rigid intraocular lens placed in the capsular bag
c. Nylon sutures used to close wound

3. Intracapsular cataract extraction – rare, only for unstable cataracts with extensive zonules
   a. Involves large incision and removal of entire lens including capsule
   b. Anterior chamber lens placed in front of lens (as there is no capsular bag left)

4. Weird and wacky third world techniques – Ow!

Retinal detachment:
   1. Cryopexy – used to ‘weld’ the retina back to the choroid and sclera
   2. Scleral buckling – synthetic band sutured to eye pushes sclera and choroid against the retina
   3. Vitrectomy – vitreous removed and gas or silicone oil inserted to push the retina back

Squint surgery:
   1. Muscles are shortened or repositioned on the sclera to move the eye into the correct position
   2. For example to correct an esotropia:
      a. Medial rectus recessed (moving it back towards equator, effectively making it weaker)
      b. Lateral rectus resected (shortened, effectively making its action more pronounced)

Glaucoma surgery:
   1. Performed when glaucoma drops are no longer controlling the intraocular pressure
   2. For example trabeculectomy:
      a. Conjunctiva pulled back and a partial thickness trapdoor/flap made in sclera
      b. Full thickness hole into the anterior chamber made under the scleral flap
      c. Scleral flap and conjunctiva sutured back into place
      d. Effectively allowing aqueous to escape the anterior chamber → conjunctival bleb