Ophthalmics

Assessment

Usually based on history and examination, with additional tests rarely required.

Presentations:

- Red eye
- Loss of vision
- Painful eye
- Diplopia
- Squinting child
- Ocular trauma

Loss of vision

- Sudden
    - Painful, prolonged – optic neuritis/giant cell arteritis
    - Painless
        - Transient – amaurosis fugax/migraine/RICP (Papilloedema)
        - Prolonged – CRVO/BRVO/CRAO/BRAO/ARMD/AION/Vitreous haemorrhage/retinal detachment
- Gradual
    - Media cloudy – cataract/corneal opacity
    - Media clear
        - Retinal – ARMD/diabetic maculopathy/retinal dystrophy
        - Neuro-ophthalmic – glaucoma/optic neuropathy/visual pathway

Examination:

- Visual acuity
    - Normal resolving power ~1 minute of arc
    - Snellen chart, read at 6m
        - Test each eye separately, with glasses
        - VA corresponds to smallest line completed accurately
    - Then counting fingers (CF), hand movements (HM), perception of light (PL) and no perception of light (NPL)
    - Normal VA implies any refractive error corrected, cornea, lens & ocular media normal, fovea centralis, connections to visual cortex, and higher visual centres all intact
    - Pinhole limits rays to those nearly parallel to visual axis – reduces refractive error – used as test for refractive error
    - Alternative tests exist for:
        - Illiterate (Landolt broken ring, illiterate E test)
        - Children (Preferential looking test, Kays picture test)
- Visual fields
    - Peripheral extent of visual world
    - Necessary for independent existence
    - Confrontation tests (screening), computerised perimetry (mapping)
- Pupil reactions
    - Size (any anisocoria)
    - Light reflex (test optic nerve) – direct and consensual, any relative afferent papillary defect (RAPD)?

    - Near reflex (convergence, accommodation, constriction)
- Ocular movements
    - Position – any squint? Cover test/alternate cover test
    - Range of eye movement, any diplopia
- External examination of eye
    - Eyelids – ptosis, ectropion, blepharitis
    - Conjunctiva – injection, discharge
    - Cornea – clarity, fluorescein staining
    - Anterior chamber – hypopyon, hyphaema
    - Iris & pupil – abnormal pupil shape
- Ophthalmoscopy
    - Red reflex (opacities in cornea/lens/vitreous)
    - Fundi (disc, macula, retinal quadrants)
    - Pupil dilation facilitates examination
        - Tropicamide (short acting ~4hrs), cyclopentilate/phenylephrine for maximal dilatation

Refractive errors
- Most common cause of reduced VA
- Problem with refracting elements (cornea 2/3, lens ½ of refractive power) or axial length of globe
    - Myopia – focus in front of retina, globe usually too long
    - Hypermetropia – focus behind retina, globe usually too short
    - Astigmatism – cornea non-spherical, refractive power varies in different medians
- Accommodation – ciliary muscles contract, suspensory lens zonules relax, lens bulges and power increases
    - Presbyopia – age related loss of accommodation due to reduced elasticity of lens. Typical onset at 45

Cataracts

$35x10^6$ people in world blind (i.e. dependant on others), $110x10^6$ have impaired vision. Majority are preventable or treatable. Major causes are cataracts (~$18x10^6$), trachoma, refractive error, vitamin A deficiency, and diabetic retinopathy (in western world).

Cataract is an opacification of the lens. Common causes:
- Congenital (toxoplasma, VZV, etc, genetic, among others)
- Age related
- Drugs (inc. systemic steroids)
- Metabolic (diabetes, some inherited conditions)
- Trauma

Presentation:
- Visual loss
- Increased myopia/decreased hypermetropia
- Glare/multiple images
- Incidental finding

Examination
- Look for darker area on red reflex
- ‘Water clefts’ of protein polymerisation
- Loss of normal lamellar structure of lens
- ‘Intumescent’ advanced cataract, stretching capsule

Specific varieties:
- Severe eczema/atopy (cornea derived from ectoderm)
- Steroid use – posterior, subcapsular focal cataract. Vision usually worse in bright light as pupil constricts
- Chronic iritis causes inflammation and a dense cataract. Synechiae (adhesions) will give an irregular pupil outline
- Myotonic dystrophy is associated with a ‘Christmas tree’ cataract

Glaucoma

A progressive optic neuropathy featuring retinal ganglion cell death, characteristic optic disc changes, and associated visual field defects. Raised intraocular pressure is a risk factor for glauacoma, not its definition.

Classification:
- Open angle
    - Primary
        - 7.5 million known cases worldwide
        - Cause unknown, but raised intraocular pressure (IOP) most common association
        - Can be insidious onset
        - Risk factors: Older age, higher IOP, family history, Black race, hypertension, low diastolic/high IOP, ?diabetes
        - Refer if optic nerve damage, or if high IOP and young/high risk
    - Secondary
        - Usually physical blockage of trabecular meshwork
        - Causes:
            - Pigmentary
            - Lens related
            - Pseudoexfoliative
    - Glaucoma suspect
- Angle closure
    - Usually acute, painful onset
    - Primary
    - Secondary
        - With pupil block
        - Without pupil block

Assessment
- History
- Examination
    - RAPD

    - IOP
        - Applanation tonography gold standard
        - 'Air puff' can read high – confirm with applanation
    - Evidence of cause?
    - Gonioscopy
    - Accurate disc measurement
        - Vertical cup to disc ratio ↑ in glaucoma (<0.6 is normal)
        - Excavation, asymmetry, pallor, haemorrhages (sign of progression – not commonly seen)
- Investigations
    - Visual fields
    - IOP phasing
- Using an IOP cut-off level alone leads to underdiagnosis – consider all factors

Treatment:
- Progression is slow, so most patients will not go blind in their lifetime
- High prevalence, so lots of people affected
    - Treat aggressively when necessary, conservative management for most
- Open angle
    - Medical
        - Reduce aqueous production (β-blocker, α-agonist)
        - Increase outflow (pilocarpine, prostaglandin analogues)
    - Laser – reduce aqueous production or increase outflow
    - Surgery – increase outflow

Narrow angles
- Risk factor for angle closure glaucoma
- Risk increased by high hypermetropia, family history of glaucoma
- Full assessment of angles by goniometry, careful slit lamp exam useful for risk assessment
- Laser PI reduces risk of angle closure greatly
- Refer if any visual symptoms or signs of optic nerve damage, consider if 'at risk' and hypermetropic/FH

Diabetic Eye Disease

Prevalence Diabetic retinopathy increases with duration of diabetes – after 20 years almost 100% of type I, and 60% of type II. Most common (western) cause of blindness age 30-65.

Microvascular leakage
- Loss of capillary pericytes → reduced integrity of vessel wall → breakdown of internal blood-retinal barrier
- Development of microaneuryms
- Increased vascular permeability
- Usually gives combination of retinal haemorrhages, retinal oedema, and hard exudates (lipid – appear yellow with well defined margin)

Microvascular occlusion

- Endothelial cell damage and proliferation
- Basement membrane thickening
- Closure of capillary networks
- Development of AV shunts
- Development of retinal ischaemia
- Cotton wool spots (infarct – white with indistinct margin) and neovascularisation

Retinal laser treatment

- Argon, Krypton, Nd:YAG
- Absorbed by melanin in RPE layer, haemoglobin
    - Coagulative necrosis of adjacent tissue
    - 'Therapeutic retinal burn'
    - Control retinal vascular leakage, modulate ischaemia, destroy abnormal tissue, create chorioretinal adhesions

Classification

- Non-proliferative (background) DR
    - Microaneurysms
    - Retinal haemorrhages (dot haemorrhage, flame haemorrhage)
    - Retinal oedema
    - Hard exudates
    - Normal vision, macula unaffected by microvascular leakage
- Diabetic maculopathy
    - Develops from non-proliferative DR independently of more advanced DR
    - Microvascular leaks affect macula
        - Retinal haemorrhage
        - Macular oedema
        - Hard exudates
    - Sight threatening DR
        - Gradual loss of vision – oedema/exudates on fovea
        - Clinically Significant Macular Oedema (CSMO)
            - Oedema/exudates within 500μm of fovea (⅓ disc diameter)
            - Laser treatment for sites of leakage
            - Reduction in leakage, promotes reabsorption
            - 50% reduction in risk of severe visual loss
- Pre-proliferative DR
    - Cotton-wool spots
    - Dark blot haemorrhages
    - Retinal venous changes (dilation, beading, looping)
    - IRMA (Intraretinal Microvascular Abnormalities) – AV shunts
    - Normal vision, risk of progression
        - Patient requires close observation

- Laser treatment not required except in severe cases

- Proliferative DR
  - Neovascularisation
    - Optic disc (NVD) and/or retina (NVE)
    - Endothelial proliferations from retinal veins
    - NVD only if ~25% of retina non-perfused
    - Ischaemic peripheral retina releases vasoproliferative factors – diffuse within vitreous
  - Normal vision, but sight threatening DR
  - Pan-retinal photocoagulation indicated
    - Scattered burns in peripheral retina
    - Destruction of ischaemic areas reduces proliferative factors
    - Preserves central vision at expense of some peripheral vision and night vision
    - Reduces risk of severe loss by >60%
- Advanced proliferative DR
  - Sudden visual loss
    - Bleeding from new vessels – vitreous or subhyaloid haemorrhage
    - Fibrous proliferation with tractional retinal detachment
    - Rubeosis iridis (neovascular glaucoma)
  - Surgical treatment
    - Vitectomy, dissection of fibrovascular membranes from retina, repair of any detachment, endolaser panretinal photocoagulation
    - Variable outcome, but usually remain significantly impaired

Causes of blindness:
- Macular oedema
- Vitreous haemorrhage
- Tractional retinal detachment
- Type 1 diabetes – often proliferative DR
- Type 2 diabetes – often diabetic maculopathy

Risk factors for DR:
- Duration of diabetes
- Diabetic control
- Hypertension/hyperlipidaemia
- Smoking
  - Control of these reduces development of DR
- Pregnancy (may accelerate progress)

Screening
- Cost effective
- Laser therapy most effective before visual symptoms occur

- Type 1 diabetes – screening from age 12
- Type 2 diabetes – screening from diagnosis
- Annual or 6/12 interval
- Digital retinal photography best
- Non-proliferative DR – optimise risk factors, continue
- Sight threatening DR – refer, urgently if proliferative DR

Other eye problems in diabetes:

- Cataract
    - Accelerated age-related cataract (~20 years early)
    - Rarely – true 'diabetic' cataract
        - Snowflake appearance
        - Lens converts glucose to sorbitol, cataract from osmotic overhydration of lens
- Transient Refractive changes
    - Intermittent blurring of vision with poor control of diabetes
    - Hyperglycaemia causes osmotic swelling of lens
        - Increased refractive power → myopia
- Ocular motor nerve palsies
    - Microvascular nerve palsy with spontaneous recovery
    - III (pupil spared), VI, (rarely IV)

Strabismus and Amblyopia

Strabismus (squint) – misalignment of visual axes

- Lateral convergent (eso-) or divergent (exo-), vertical (hyper/hypo)
- Latent (phoria) or constant (tropia)
- Comitant (same angle for all positions of gaze), incomitant (angle varies with gaze, often restrictive or paralytic cause)
- Testing:
    - Symmetrical corneal light reflex (relate to limbus – skin margins can give pseudosquint)
    - Quality of red reflex – brighter in squinting eye
    - Cover/uncover and alternate cover – breaks down sensory fusion, uncovers latent squint
- Infantile esotropia
    - Age <1yr
    - Large angle
    - Cross fixation
    - No refractive error
- Accommodative esotropia
    - Age >1yr
    - Hypermetropic (constant accommodation → convergence)
    - Treating hypermetropia reduces squint
- Exotropia
    - Infantile
    - Intermittent exotropia
    - Sensory exotropia
    - Consecutive exotropia

- Vertical squints
    - SO laxity
    - IO overaction
    - IV and III nerve palsies

- Treatment
    - Treat refractive error
    - Treat amblyopia if present
    - Assess the child's sensory fusion
    - Functional strabismus surgery
    - Cosmetic strabismus surgery
- Surgery
    - Recess (or tenotomy or myectomy) to weaken muscle
    - Resect or tuck to strengthen
    - Treat both eyes
    - Muscles have secondary actions – not entirely simple
- Incomitant strabismus
    - III nerve palsy
    - IV
    - VI
    - Restrictive – TED, orbital floor fracture
    - Others
        - Congenital eg Duanes and Brown's
        - Acquired eg myasthenia
    - Investigate cause
    - Wait at least six months, for stable angle
    - Transpositions to improve field of binocular single vision

Laws of innervation:

- Sherington's law of reciprocal innervation (reciprocal action of medial and lateral rectus on each eye)
- Hering's law of yoke muscle innervation (equivalent action of muscles on each eye yoked)

Normal – sensory fusion of binocular images, normal innervation and anatomy, motor fusional capability
Sensory problems can lead to failure of motor fusion, motor problems can lead to loss of sensory fusion/amblyopia
Amblyopia – reduced VA persisting after correction of refractive or anatomical error of eye. Results from anatomical disturbances in lateral geniculate nucleus/occipital cortex. Usually reversible up to age 8.

Retinal Disorders

Macula – central retina. Fovea – Finest visual resolution. Foveola – central fovea.

Neurosensory retina – photoreceptors/neurones. RPE – single layer of cells between choroids and retina.
Blood supply: central retinal artery to inner 2/3 of retina, choroids (choriocapillaris) to outer 1/3. Blood-retinal barriers exist – retinal capillary endothelial cells (inner) and RPE cells (choroids).

Symptoms:
- Macular dysfunction
    - Blurred vision, scotoma, metamorphopsia (distorted vision), micro-/macropsia (decreased/increased image size)
- Peripheral retinal dysfunction
    - Absolute or relative peripheral visual field defect, night blindness if severe and generalised loss of rods

Evaluation:
- VA
- Visual fields
- Ophthalmoscopy
    - Direct ophthalmoscope or slit lamp
- Fluorescein angiography
    - IV injection of fluroscein, fundal photographs over 5-10 minutes – fluorescent image of retinal circulation
- Electrophysiology

Retinal disorders
- Acquired maculopathies
    - Age-related macular degeneration (AMD)
        - 10% of 65-75yrs, 30% of >75yrs, most common cause of blindness in >50yr
        - Atrophic ('dry') – 90% of AMD
            - RPE and photoreceptor degeneration
            - Discrete subretinal yellow lesions, accumulated photoreceptor 'waste products'
            - RPE hyperpigmentation or hypopigmentation (atrophy)
            - Slow, progressive loss of central vision over years. End stage is geographic atrophy
            - Untreatable
        - Neovascular ('wet') – 90% of AMD with severe visual loss
            - Choroidal neovascular membrane (CNVM)
            - New vessels from choriocapillaries through RPE to subretinal space → structural retinal damage
            - Subretinal fluid ± lipid deposits, subretinal haemorrhage
            - Rapid loss of central vision (days), may already have atrophic
            - End stage is subretinal fibrotic scar

            - Urgent ophthalmogy review and fluroscein angiography
            - Extrafoveal CNVM – laser photocoagulation
            - Subfoveal CNVM – photosesitising agent and selective destruction of CNVM
        - AMD affects both eyes (often asymmetrically)
        - Loss of central vision, periphery spared
        - Treatment rarely possible, and at best stabilises vision
        - Rehabilitation to support remaining vision

    - Central serous retinopathy
        - ♂ 20-45. Localised RPE abnormality with blood-retinal barrier breakdown and leakage of fluid to subretinal space, and localised serous retinal detachment
        - Rapid loss of central vision – blurred, relative scotoma, metamorphopsia ± micropsia
        - Dome-shaped retinal elevation
        - Spontaneous resolution <3mths, almost complete visual recovery. Laser photocoagulation of leakage point possible if no resolution
        - Recurrence in 30% of cases
    - Myopic maculopathy
        - High myopia (>-6D) leads to degenerative maculopathy
        - Macular atrophy, subretinal haemorrhage, choroidal neovascularisation
        - Progressive loss of central vision
    - Macular hole
    - Macular epiretinal membrane
- Retinal vascular disorders
    - Diabetic retinopathy
    - Retinal vein occlusion
        - Typically 50-70yrs, sudden loss of vision
        - Dilated, torturous retinal veins, scattered haemorrhages, cotton wool spots, macular oedema ± hard exudates
        - BRVO – one quadrant. CRVO – all quadrants + swollen disc
            - Intrinsic change in vein wall – compression at AV crossing, arteriosclerosis. Risk factors – diabetes, hypertension
            - Haematological abnormality (rare) – hyperviscosity/hypercoagualopathy
            - Raised intraocular pressure
        - Macular laser treatment for oedema in BRVO
        - Retinal ischaemia from neovascularisation or rebeosis iridis (CRVO)
    - Retinal artery occlusion
        - 50-70 yrs, sudden loss of vision
        - Retinal whitening (ischaemia), narrowed retinal arterioles, segmentation of blood column, ± visible embolus
        - BRAO – one quadrant, CRAO – 4 quadrants, cherry red spot at macula
        - Usually embolus – carotid artery disease, cardiac disease. Rare causes include giant cell arteritis, vasculitis (PAN, SLE, etc)
            - Ocular manoeuvres to dislodge embolus (rarely successful)

        - Cardiovascular assessment, antiplatelet therapy
        - >24hrs occlusion → severe visual loss
    - Hypertensive retinopathy
        - Retinal arteriolar narrowing
        - Microvascular leakage and/or occlusion
        - Malignant hypertension – swollen disc (may affect vision)

- Inherited retinal dystrophies
    - Retinitis pigmentosa
        - Mainly rods affected – nyctalopia, constricted visual fields
        - Classical triad of peripheral retinal pigmentation (bone spicule), attenuation of retinal arterioles, optic disc pallor
    - Macular dystrophies
        - Neurosensory – cone dystrophy
        - RPE – Stargardt macular dystrophy, Best vitelliform macular dystrophy
        - Choriocapillaris – Central areolar choroidal dystrophy
- Intraocular tumours
    - Choroidal naevus
        - Common (~10% population), asymptomatic
        - Slate-grey choroidal lesion, flat or minimally elevated, small (usually <3 disc diameters)
    - Choroidal melanoma
        - Age 40-70, asymptomatic or reduced VA/visual field defect
        - Pigmented, elevated choroidal mass, ± exudative retinal detachment
        - Treatment options include local excision, radiotherapy/proton beam radiotherapy, enucleation
        - Spread to liver or lung
    - Choroidal metastasis
        - Commonest 1°: ♀ Breast, ♂ Bronchus
        - Creamy-white elevated lesion, often multiple
        - Radiotherapy or chemotherapy
    - Retinoblastoma
- Retinal detachment

Retinal Detachment

Rate around 1 per 10 000 population per year.

Rhegmatogenous

- Presents as sudden visual loss or field loss
- Usually secondary to posterior vitreous detachment
    - 66% of population at some point. Peak incidence 45-65, earlier in myopes

    - May be partial or complete
    - Can be asymptomatic, but typically:
        - Floaters (protein debris), gradually sink and clear visual field
        - Flashing lights
            - Always single and in temporal field
            - Usually white or golden, dim, and provoked by eye movement
    - Test – patient to look downwards then ahead. Vitreous will settle, and hyaloid membrane will sink into view if detachment has occurred
    - 5% tear retina – highest risk in myopes, previous history of detachment, or family history of detachment

- Early (first few hours) laser or cryotherapy can prevent detachment
- Later – surgery to replace retina, gas bubble to splint, laser/cryo to cause adhesion
- Can be associated with vitreous haemorrhage from torn retinal vessels
    - Black/red rain in vision (individual erythrocytes seen)
    - Other causes include diabetes, sickle cell retinopathy, retinal ischaemia causing neovascularisation

Tractional

- Scar tissue in eye
    - Commonest cause is secondary to diabetes
    - Intraocular surgery
    - Infection
    - Others
- Treat surgically – excise scar tissue, retina will then return to place

Solid/Exudative

- Due to mass, usually in choroid
- Often tumour
    - Metastatic carcinoma most common (breast, lung)
        - Often multiple and affecting both eyes
    - Melanoma most common primary
    - Retinoblastoma very rare

Oculoplastics

Orbital cellulitis

- Pre-septal
    - Red and inflamed
- Post-septal
    - Much more serious
    - Decreased ocular movement, vision, visual fields, and colour vision

Ptosis

- Congenital

    - Risks amblyopia
    - Surgery indicated
        - Shorten levator
        - Brow suspension (if severe)
        - Risk overcorrection -> corneal problems

- Acquired
    - Neurogenic
        - III palsy
        - Horner's (subtle ptosis)
    - Myogenic
        - Myasthenia Gravis
        - Kearns-Sayer syndrome
    - Mechanical
        - Swelling/abscess/etc of eyelid
    - Involutional/senile
        - Levator dehisces from tarsal plate
    - Pseudoptosis
        - Small or artificial eye

Ectropion

- Mechanical
    - Infection/tumour
- Senile/involutional
    - Lateral and medial palpebral ligaments slacken
    - Inferior retractor weakens
    - Orbicularis stronger tends to increase problem
- Paralytic
    - Bells palsy (VII palsy)
- Cicatricial
    - Scar tissue causing retraction

Entropion

- Senile/involutional
- Cicatricial
    - Conjunctival scarring
    - Trachoma – chlamydial (tx single dose tetracycline)
    - Stevens-Johnson syndrome

Neuro-ophthalmology

Visual fields

- "Spatial awareness beyond point of regard" (outside region projected to macula)
- Needed for everyday functioning
- NB inferior fibres tend to remain inferior throughout the tract – pituitary tumour causes loss of superior temporal fields initially
- Testing:
    - Absolute – finger counting in all quadrants
    - Relative – perceived colour difference between identical objects (e.g. hands). Darker is abnormal, red target gives best discrimination
    - Lateral differences tend to be cortical, vertical tend to relate to eye or nerve

Optic disc

1. Colour – pale is atrophic, pink is papilloedema
2. Edge should be slightly blurred. Very sharp is atrophic, blurred is papilloedema
3. Cup – bigger indicates fewer nerve fibres, smaller may be due to swollen nerve
4. Vessels – normally only blood column seen – vessel walls should not be visible. Arteries are pinker and narrower than veins
5. 'Something else' – look for haemorrhage, swelling, cotton wool spots, etc

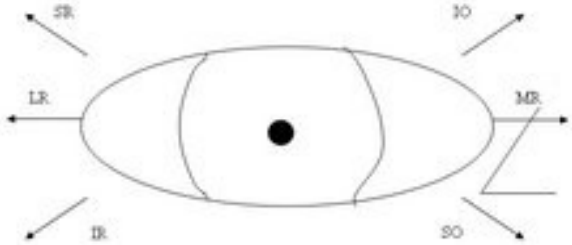
Papilloedema causes an increase in size of blind spot.

Pupil

- Dilated, unreactive pupil is a problem. Asymmetry is suspicious.
- Constricted
    - Opiates, pilocarpine, Horner's, (rarely) congenital
- Dilated – III n. palsy
    - Diabetes, tumour, haemorrhage (leading to increased pressure), posterior communicating artery aneurysm, muscarinic blocking or sympathomimetic drugs, (rarely) congenital

Eye movements

- III, IV, VI
- In palsy, lateral image is false. IV palsy – vertical diplopia, false image is rotated as superior oblique torts eye inwards



Red Eye

Vessels – additional O2 diffuses from surface of eyes, so even venous blood is fairly bright red. Deeper vessels are bluer.

'Safe' red eye – diffuse redness over bulbar conjunctiva
'Dangerous' red eye – limbus preferentially reddened. Focal redness also concerning.

- Keratitis
- Acute glaucoma (leading to ischaemia and dilatation of limbal vessels)
- Intra-ocular inflammation (uveitis, iritis)
- Scleritis
    - Pale ischaemic region in eye with surrounding vessels dilated
    - Acutely painful (necrotic tissue)
- Carotico-cavernous fistula
    - Torturous, dilated vessels on pale background (arterialised veins)
    - Painless
- Sub-conjunctival haemorrhage
    - Bright, even red colour, well demarcated by limbus. No vessels seen
    - Usually spontaneous, no treatment is required
    - If the posterior pole of the haemorrhage cannot be visualised in fornix, sign of basal skull fracture
- Hamartoma
    - Single dilated vessel with randomly branching end network
- Episcleritis
    - Branching network, lacks obvious feeding vessel
- Trachoma/viral surface infection
    - Tarsal conjunctiva inflamed, upper plate in particular.
    - Radial vessels and papillae seen
- Viral disease
    - 'Sausage shaped' follicles of lymphocyte aggregation, with red eye
- Molluscum contagiosum
    - Can cause follicular conjunctivitis
    - Skin lesions should be treated if near eye
- Bacterial conjunctivitis
    - Gritty, sticky eye, burning sensation
    - VA normal
- Viral conjunctivitis
    - Profusely watering eye, no sticky discharge
    - VA normal
- Allergic eye disease
    - Mucous produced
    - Itching
    - Associated with atopies
    - Topical steroid to settle, topical antihistamine to control
- Herpes zoster
    - Painful, sticky, red eye
    - Swollen, red eye lid with ptosis
    - ±vesicles on skin

- Lymphoma
    - Focal region of non-painful mass(es)
    - Red, well-demarcated, abuts normal tissue. Often in fornix.
    - Asymptomatic
    - Usually responds well to radiotherapy

Ocular Inflammation and Systemic Disease

Conjunctival
- Reiters
- Sarcoid
- Stevens-Johnson
- Cicatricial pemphigoid

Cornea/sclera
- Rheumatoid arthritis
- Wegener's granulomatosis
- Microvascular polyarteritis
- Polyarteritis nodosa
- SLE
- Relapsing polychondritis
- IgA nephropathy
- Inflammatory Bowel Disease

Ocular interior (uveitis/choroiditis/retinal vasculitis)
- HLA-B27 (ankylosing spondylitis, Reiters)
- IBD
- Sarcoid
- Behcet's
- Multiple Sclerosis
- SLE
- Autoimmune renal disease
- Infection (toxoplasma, TB, syphilis, borrelia, bartonella, candida, HSV, VZV, CMV)
- Lymphoma

Orbit
- Thyroid Eye Disease
- Orbital myositis
- Wegener's granulomatosis
- Orbital pseudotumour
- Orbital lymphoma
- (Temporal arteritis)

Thyroid Eye Disease
- Slow (1-3 year) cycle of inflammation of orbital muscles
- Autoimmune damage to orbital fibroblasts

    - Inflammation
    - Poor ocular motility, painful eye movement, vasodilatation
- Glycosamine deposition
    - Space occupation
    - Proptosis – corneal exposure, corneal ulceration
- Collagen secretion and fibrosis
    - Restriction
    - Optic nerve compression, orbital venous congestion, ocular hypertension

Ocular Trauma

Three main groupings – blunt, penetrating, and chemical.

Blunt

- Globe is protected on three sides, so damage usually from objects that 'fit' the socket, e.g. squash ball, fingers, etc
- Need to evert eyelids to check for retained matter
- Corneal abrasion
- Traumatic cataract
- Iris tear
- Hyphaema
    - Seen as blood fluid level in anterior chamber – may clot
    - Can block drainage, causing high pressure (late 2° glaucoma)
    - Usually resolves with time
- Blowout # of orbit
    - Usually through thin bone – floor (maxillary sinus) and/or medial wall (ethmoid sinus)
    - Eye recessed (enophthalmos), lids partially closed
    - Diplopia (e.g. damage to inferior oblique)
    - Surgical crepitus (emphysema)
    - Numb teeth/upper cheek – infraorbital nerve damage
- Traumatic retinal detachment (rare)
    - Coronal expansion of globe avulses retina
    - Fragments may be visualised in chamber
    - Need to stabilise remainder (e.g. laser)

Chemical

- Severity depends on nature and concentration of chemical, duration of contact
- Generally gases less noxious than solids/liquids

- Acids coagulate on surface, limiting penetration. Alkalis are lipid soluble so cause extensive, deep burns. Lime and mortar complicate this with retained particles. NaOH and liquid NH_3 cause limbal ischaemia.
- Ignore examination – irrigate with 2-3L of water/saline, evert lids or perform a blind sweep.

Penetrating

- Anterior segment (lens forward) has good prognosis with appropriate treatment, posterior segment is more variable
- Always assess VA
- Pupil asymmetry is a sign of penetrating trauma
- Foreign body usually needs removal
    - Retained ferrous body causes siderosis bulbi
    - Heterochromia, cataract, uveitis, mydriasis
    - Irreversible retinal toxicity
- High index of suspicion for any high velocity injury
    - X-ray

Misc

Conventionally report on right eye before left

Slit lamp can be used to give bright, even illumination, bright/dark field retro-illumination, or slit to give optical thin section of transparent object

Compression of III (surgical 3rd) – lose sympathetic
Ischaemia of III (medical 3rd) – normal pupil (central vessel occlusion → surface sympathetic fibres spared)

Early ocular problems affecting vision in infants tend to have an associated nystagmus