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 glaucoma, 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 microaneurysms
- 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
    - Vitrectomy, 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
  o Accelerated age-related cataract (~20 years early)
  o Rarely – true ‘diabetic’ cataract
    ▪ Snowflake appearance
    ▪ Lens converts glucose to sorbitol, cataract from osmotic overhydration of lens
• Transient Refractive changes
  o Intermittent blurring of vision with poor control of diabetes
  o Hyperglycaemia causes osmotic swelling of lens
    ▪ Increased refractive power → myopia
• Ocular motor nerve palsies
  o Microvascular nerve palsy with spontaneous recovery
  o 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:
  o Symmetrical corneal light reflex (relate to limbus – skin margins can give pseudosquint)
  o Quality of red reflex – brighter in squinting eye
  o Cover/uncover and alternate cover – breaks down sensory fusion, uncovers latent squint
• Infantile esotropia
  o Age <1yr
  o Large angle
  o Cross fixation
  o No refractive error
• Accommodative esotropia
  o Age >1yr
  o Hypermetropic (constant accommodation → convergence)
    ▪ Treating hypermetropia reduces squint
• Exotropia
  o Infantile
  o Intermittent exotropia
  o Sensory exotropia
  o 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**

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 ophthalmology review and fluorescein angiography
- Extrafoveal CNVM – laser photocoagulation
- Subfoveal CNVM – photosensitising 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, tortuous 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/hypercoagulopathy
      - 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
    o Hypertensive retinopathy
      ▪ Retinal arteriolar narrowing
      ▪ Microvascular leakage and/or occlusion
      ▪ Malignant hypertension – swollen disc (may affect vision)

• Inherited retinal dystrophies
  o Retinitis pigmentosa
    ▪ Mainly rods affected – nyctalopia, constricted visual fields
    ▪ Classical triad of peripheral retinal pigmentation (bone spicule), attenuation of retinal arterioles, optic disc pallor
  o Macular dystrophies
    ▪ Neurosensory – cone dystrophy
    ▪ RPE – Stargardt macular dystrophy, Best vitelliform macular dystrophy
    ▪ Choriocapillaris – Central areolar choroidal dystrophy

• Intraocular tumours
  o Choroidal naevus
    ▪ Common (~10% population), asymptomatic
    ▪ Slate-grey choroidal lesion, flat or minimally elevated, small (usually <3 disc diameters)
  o 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
  o Choroidal metastasis
    ▪ Commonest 1°: ♀ Breast, ♂ Bronchus
    ▪ Creamy-white elevated lesion, often multiple
    ▪ Radiotherapy or chemotherapy
  o 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
    o 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
  o Neurogenic
    • III palsy
    • Horner’s (subtle ptosis)
  o Myogenic
    • Myasthenia Gravis
    • Kearns-Sayer syndrome
  o Mechanical
    • Swelling/abscess/etc of eyelid
  o Involutional/senile
    • Levator dehisces from tarsal plate
  o Pseudoptosis
    • Small or artificial eye

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

Entropion
• Senile/involutional
• Cicatricial
  o Conjunctival scarring
  o Trachoma – chlamydial (tx single dose tetracycline)
  o 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
  o Focal region of non-painful mass(es)
  o Red, well-demarcated, abuts normal tissue. Often in fornix.
  o Asymptomatic
  o 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
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

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