Anatomy of the Eye

The eyeball is located in the orbit along with extra-ocular muscles, fascia, fat, blood vessels, nerves and lacrimal gland. The orbit is a bony four sided pyramid, with optic canal at apex and sup and inf orbital fissures.

Coats of eye:
Outer fibrous layer – cornea, sclera, lamina cribosa.
Middle vascular layer – iris, ciliary body (pars plicata and pars plana), choroid.
Inner nervous layer – pigment epithelium, retinal photoreceptors, retinal neurones.

Compartments of the eye:
Anterior chamber – between cornea and iris.
Posterior chamber – between iris, lens and zonule and ciliary body.
Vitreous chamber – behind lens and zonule.

Intraocular fluids:
Fluids are similar composition and pressure to CSF.
Aqueous humour – watery, similar to tissue fluid but low protein content, nourish lens and cornea, formed by active secretion and ultrafiltration from ciliary processes. Flows from posterior to anterior chamber through pupil, drains through trabecular meshwork and canal of Schlemm.
Vitreous humour – transparent gel with collagen fibres, hyaluronic acid and water. With age becomes more liquid, bits break off forming floaters and detaches from retina, especially in myopes.
Blood – mostly in choroid, for nutrition, heat exchange, maintain intraocular pressure.

Cornea, conjunctiva and sclera:
Cornea is in anterior one sixth, junction with sclera is called limbus, containing stem cells for epithelium. Epithelium of cornea is continuous with conjunctiva - thin loose mucous membrane. Endothelium is non-regenerating.
Cornea is needed for refraction – transparent, smooth curved surface.
Conjunctiva covers anterior sclera and is reflected to inner surface of eyelids.
Sclera is irregular collagen fibres, continuous posteriorly with dural sheath of optic nerve.
Sclera is thickest posteriorly and thinnest at insertion of muscles.
Inner part of sclera is lamina cribosa – fenestrated layer of collagen which nerve fibres pass through from retina.
Medial canthus has caruncle and upper and lower punctums.

Uvea:
Pigmented and vascular.
Anterior part is iris. Contraction of sphincter constricts pupil, contraction of dilator pupillae dilates pupil.
Ciliary body – attached to iris, scleral spur and choroid. Inner part has pars plicata (anterior with ciliary processes) and pars plana (posterior, flattened).
Posterior part is choroid. Bruch’s membrane on external surface of RPE, choriocapillaries, larger vessels, pigmented cells. Lens is avascular. Thick elastic capsule prevents movement of molecules. Grows throughout life. Suspended from ciliary body by zonule.
Neurosensoric layer:
Outer part – retinal pigment epithelium, stops light scattering and regenerates photopigments in dark-light adaptation. Loosely attached to retina except at periphery.
Inner sensory part (moving inwards) – rods, cones, bipolar cells, ganglion cells.
10 layers.
Fovea has cones giving acute colour vision, avascular.
Nerve fibres radiate to optic nerve without crossing horizontal line in arcs. Papillo-macular bundle is therefore at about 9 o clock on disc.

Eyelids:
Muscles – levator palpebrae superior, sympathetic levator (Mullers muscle), orbicularis oculi.
Tarsal plate – tough collagen layer.
Eyelashes.
Meiboniam glands – oil of tear film.
Needed to protect eye, maintain tear film.

Lacrimal glands:
Sup temporal part of orbit.
Ducts open to palpebral conjunctiva.
Tears collect at medial part and pass through puncta to lacrimal sac to nasolacrimal duct.
At rest only produced by accessory glands. Lacrimal glands by reflex stimulation.

Tear film:
Mucoid layer – next to epithelium, produced by goblet cells.
Aqueous layer – produced by lacrimal glands, contains proteins, electrolytes, lysozyme, immunoglobulins, glucose and oxygen.
Oily layer – superficial layer, produced by meiboniam glands of eyelid, prevent evaporation.
Forms air tear interface to prevent distortion with refraction, provides oxygen, removes debris, antibacterial.
Drain by puncta, canaliculi, lacrimal sac, naso-lacrimal duct.

Blood supply:
Internal carotid gives of ophthalmic artery which branches to central retinal, anterior ciliary and posterior ciliary arteries.
Central retinal – in optic nerve to interior of eye, supplies inner half of retina.
Anterior ciliary arteries – arterial circle in ciliary body.
Posterior ciliary arteries – supply choroid and link with ciliary body (long posterior).
Veins – central retinal vein, 4 vertex veins leaving each of posterior quadrants.

Nerves:
Optic nerve goes to posterior nasal part of globe.
Forms optic disc on fundoscopy.
Long and short ciliary nerves carry autonomic and sensory fibres to iris muscles and ciliary body.
Sympathetic dilates, from hypothalamus, T1, superior cervical ganglion, along external carotid, ciliary nerves.
Parasympathetic constricts, travels with CN III.
Notes on Ophthalmology. Author: Liz Tatman

Visual pathway:
Retina (rods and cones), bipolar cells, ganglion cells.
Optic nerve, optic chiasm, optic tract.
Lateral geniculate nucleus (cortical relay) and superior colliculus (eye movements).
Superior fibres pass through parietal lobe, inferior fibres through temporal lobe (Meyer’s lobe).
Visual cortex (area 17), occipital lobe.

Light reflex pathway:
Optic tract.
Decussation from pretectal nucleus to Edinger Westphal to give consensual reflex.
Efferent pathway along 3rd nerve to constrict pupil.

Accommodation:
Prestriate cortex.
Edinger-Westphal nucleus.
3rd nerve.

History
Eye symptoms:
- Pain
- Disturbances of vision – distortion, diplopia, flashing, floaters, haloes, field defect.
- Reduction of vision – duration, one or both eyes, transient/permanent.
- Red eye – watery, sticky, painful, visual loss, duration.
- Change in appearance – discolouration, swelling, ptosis, squint

Other - watering, eye movements, dry eye

History should include:
- Time of onset, eye affected, associated symptoms.
- Past ocular history.
- Past medical history – especially hypertension, diabetes, thyroid, systemic inflammatory disease.
- Drug history, family history, allergies.
Examination

Orbit
Lesions tend to cause proptosis – can measure with exophthalmometer, difference of more than 3mm is significant. Eye can be displaced:
- Forward – intraconal lesion e.g. optic nerve sheath meningioma.
- To one side – extraconal lesion e.g. tumour of lacrimal gland.
- Transient with increased cephalic venous pressure – orbital varices.
- Orbital cellulitis – preseptal (normal eye movements) or postseptal (more serious). Generally from ethmoid sinus, H inf.
- Carotic-cavernous fistula (proptosis and dilated veins, leads to raised intraocular pressure).
- Thyroid disease – most common cause of proptosis.
- Orbital tumours – lacrimal gland, optic nerve gliomas, meningiomas, lymphomas, rhabdomyosarcomas, metastases.
- Investigate with CT or MRI.

Optthalmoscopy
To examine retina.

Dilate pupil, generally with tropicamide.
Patient looks straight ahead. Use opposite eye, 30cm away at 15degree angle.
Red reflex – cataracts.
Optic disc –
Contour – should be distinct, swollen with poorly defined edges and haemorrhages in papilloedema (must be bilateral, increased ICP), optic neuritis, malignant hypertension, anterior ischaemic optic neuropathy.
Colour – should be pink, pale with compression of optic nerve, ischaemic optic neuropathy, glaucoma, retinal vein or artery occlusion, optic neuritis.
Cup – increased ratio (above 0.4) in glaucoma.
Macula – foveal reflex (bright pinpoint of light), haemorrhages, exudates, cotton wool spots.
Arteries and veins – AV nipping, emboli, new vessels.

Anterior eye
Generally use slit lamp.
Conjunctiva – injected, discharge, haemorrhage. Papillae (upper tarsal conjunctiva, chronic inflammation, giant in allergic eye disease) and follicles (lower tarsal conjunctiva, viral and chlamydial infections).
Cornea and lens – clear, reflections, red reflex.
Hypopyon, hyphaema (blood in ant chamber, shows over iris).
Fluorescein – absorbs blue light and emits green. Use to show corneal abrasion and penetrating corneal injury.
Evert upper lid.

Normally lower lid should be at junction of iris and cornea and upper lid just crossing iris. Look for retraction of lids (most common cause is Graves’ disease) and proptosis.
Visual acuity
Generally Snellen – tests resolving power. Record as reading distance/row number (distance at which should be able to read it).
Children – follow objects, Cardiff acuity test (preferential for complex target), matching letters or pictures.

Visual field
Represented by contours or isoptres of ability to resolve size or brightness. Eye has better resolution towards centre (fovea) giving a hill of vision. Blind spot on temporal side due to optic nerve head. Remember all reversed so nasal fibres = temporal field and superior fibres = inferior field etc. Always check acuity first.

Confrontation tests – compare to examiner, bring target (usually white to start) in from periphery, compare object on each side. Look at examiner’s face first and count fingers in each quadrant for obvious scotomas.
Red objects are often used as most sensitive to optic nerve lesions (red desaturation). Perimeters – more accurate plotting. Kinetic field (when light moving from periphery is first seen) and static field (when stationary light of increasing brightness is first seen). Fovea is centre of grid. Check intra-ocular pressure with Goldmann tonometer.

Visual field defects:
Arcuate scotoma if papillomacular bundle affected. Lesions before optic chiasm maintain a well-demarcated horizontal meridian as nerves in retina don’t cross horizontal line.
Lesions after the chiasm have a vertical meridian as fibres have split.
Lesions in the optic nerve affect just one eye and cause decreased vision, RAPD and red desaturation, maybe fundoscopy signs.
Lesions at optic chiasm cause bitemporal hemianopia.
Lesions in optic tract or optic radiation cause homonymous hemianopia. This is more incongruous (unequal both sides) if closer to the chiasm as less organisation of fibres.
Lesions of the occipital cortex can be macular sparing as this locates to the tip of the lobe and has a different blood supply (middle cerebral a instead of posterior).
Lesions of the temporal lobe affect superior quadrant.
Lesions of the parietal lobe affect inferior quadrant.

Pupils
Miosis – constricted, mydriasis – dilated. Assess in uniform light for asymmetry (anisocoria). If present:
If worse in dim light, constricted pupil is pathological.
If worse in bright light, dilated pupil is pathological.
Generally, constricted pupil is due to damage to SNS and dilated pupil due to PNS.
Look for posterior synachiae – inflammation of anterior eye causes iris to stick to lens giving irregular pupil.

Pupillary responses – consensual and direct.
Patient must focus on distant object to prevent accommodation. Dimly lit room so can see pupils but not constricted.
Afferent pupillary defect – due to problem with optic nerve or tract or retinal ganglion cells. No direct or consensual response for affected side.
Efferent defect – problem with 3rd nerve. Affected side has no direct or consensual response. Obvious anisocoria in bright light.

Swinging flashlight test for relative afferent pupillary defect. Both eyes constrict to light but stimulus is less from one, so when light is moved from normal eye to affected eye, pupils appear to dilate.

Near reflex – pupils constrict when looking at near object (stimulated by accommodation and convergence). This just uses efferent limb of pupil reflex so can locate problem.
Get light-near dissociation if no light reflex but intact accommodation – due to midbrain or ciliary ganglion lesion.

Horner's syndrome:
Problem with sympathetic pathway.
Affect pupil is smaller, especially in dark.
Partial ptosis and apparent enophthalmos.
Lack of sweating if lesion proximal to base of skull.
Common causes – surgery, neck injury, lung apex tumours, syringomyelia. Various further tests to determine level.

Argyll-Robertson pupil:
Neuro-syphilis.
Small irregular pupils.
Sluggish light response, leads to blindness from optic atrophy.

Adie pupil:
Enlarged pupil.
Often young patients, especially female.
Tonic pupil – absent light reflex and slow near response (light-near dissociation), efferent pupillary defect.
Often also abnormal tendon reflexes.
Ciliary ganglionitis denervates iris and ciliary body.

Ocular motility
Position of eyes at rest – squint.
Range of movement – check from primary position, ask about diplopia, look for nystagmus.
Type of movement – nystagmus, test pursuit (following) and saccadic (switch targets).

CN3 supplies:
Medial rectus – look in.
Inferior rectus – look down.
Superior rectus – look up.
Inferior oblique – in and up.
Pupil sphincter and lid.

CN4 supplies superior oblique – in and down.
CN 6 supplies lateral rectus – look out.
Palsies can be:
Neurogenic – vascular, inflammation, trauma, space occupying.
Mechanical – blow out fracture (muscle caught in fracture) thyroid eye disease (inflammation and fibrosis of muscle).
Myogenic – MG, MS, Parkinsons.
Cranial nerve palsies:
CN3 – eyes down and out, ptosis, pupil may be dilated (generally not if vascular). Common causes – vasa nervosum obstruction, posterior communicating artery aneurysm.
CN4 – eyes deviated up, head tilted with chin down, vertical diplopia. Common causes – congenital, vasa nervosa obstruction, closed head injury (false localising sign), intra-cranial disease.
CN6 – eyes deviated in, horizontal diplopia, head turned to side. Common causes – vasa nervosum infarction, head injury, raised ICP, infection spreading to petrous temporal bone (Gradenigo’s syndrome), cavernous sinus disease.
CN7 – exposure due to no orbicularis oculi. Test corneal sensation and Bell’s phenomenon (eye rolls up when closed).

Eyelids

Abnormal lid position
Inflammation
Lumps
Abnormal lashes

Ptosis – mechanical factors (oedema, scarring etc.), neurological factors (3rd nerve palsy, Horner’s, Marcus Gunn), myogenic factors (MG, muscular dystrophies).
Entropion – inturning of lid, typically in elderly people with weak orbicularis oculi or conjunctival scarring. Lashes irritate eye and abrade cornea.
Ectropion – eversion of lid, usually orbicularis laxity, scarring, facial nerve palsy. Prevents tear drainage causing epiphora and exposes conjunctiva.
Blepharitis – common, chronic eyelid inflammation. Sore tired eyes especially in morning, crusting, debris around eyelash, fewer eyelashes, obstruction of meibomian ducts, injection of lid margin, cloudy meibomian secretion. Manage with lid toilet to remove debris, lid massage, antibiotics if staphylococcal disease, oral tetracycline to improve meibomian gland function.
Drugs

**Mydriatics:**
Usually also cause cycloplegia.
Usually parasympatholytic.
May precipitate acute angle closure glaucoma.
Uses – diagnostic, treatment in iritis, break up or prevent posterior synachiae.
E.g. atropine (long acting), cyclopentolate, tropicamide (short acting).

**Miotics:**
Also cause cyclotonia, reduction in IOP as parasympathomimetics.
E.g. pilocarpine, choline ester, anticholinesterases.
Used to treat glaucoma.

Antibiotics – usually use chloramphenicol e.g. for conjunctivitis, trauma. Also gentamicin or cefuroxime.

Local anaesthetics.

Steroids – oral and topical (for anterior segment e.g. blepharitis, iritis), may cause cataracts or glaucoma. Serious risk is inducing progression of dendritic ulcers (corneal ulcers due to HSV, so need prior slit lamp inspection).

Antiallergic agents.

Tear substitutes.
Glaucoma

Raised ocular pressure causes damage to the optic nerve head by mechanical damage and ischaemia. Axon loss leads to visual field defects and a reduction in acuity. Pressure depends mainly on aqueous humour production and removal. Produced by ciliary processes in posterior chamber, passes through pupil and leaves by trabecular meshwork, Schlemm’s canal and episcleral veins (conventional pathway), and a little bit through ciliary body (uveoscleral pathway). Pressure is normally 16mmHg. Can rise to 70 in acute angle closure.

Prevalence is about 2%. Higher in Afro-Caribbeans. Glaucoma can be:
Congenital – often related to large eyes (bupthalamos = increased corneal diameter), rubella, aniridia.
Primary – acute or chronic, closed or open angle.
Secondary – trauma, surgery, uveitis, caroticocavernous sinus fistula, drugs (mydriatics, steroids, anticholinergics).
Normal tension – pressure is not raised but similar damage to optic disc with field loss.

Open angle:
Angle between iris and cornea is clear, trabecular meshwork is visible. Resistance to outflow of aqueous due to thickening of meshwork, generally with age, leading to inefficient drainage. Generally chronic. Common in older people, especially with family history. Chronic glaucoma is generally asymptomatic until vision is lost with end stage disease. Screening is needed.

Closed angle:
Lens approaches iris (peripheral anterior synachiae, thickens with age) and aqueous accumulates in posterior chamber. Iris bulges forward and occludes trabecular meshwork. Typically in hypermetropic eyes. Usually acute as pressure rises. Eye is very painful and photophobic. Other symptoms – headache, fixed semi-dilated pupil, red eye, cloudy cornea.

Examination findings:
Increased pressure (normal is 16mmHg, raised is over 21, higher in angle closure). Optic disc cupping – normally 0.4, sometimes notches. Perimetry field testing – usually get arcuate scotomata, lose nasal superior fields first, tunnel vision with advanced disease. Look at iridocorneal angle. Look for secondary cause.

Treatment:
Medical treatment – beta blockers (decrease secretion), prostaglandin analogues (increase outflow), carbonic anhydrase inhibitors (topical and systemic), alpha 2 agonist (decrease production and increase outflow), parasympathomimetics (e.g. pilocarpine, cause meiosis but also accommodation spasm). Mostly topical. Laser trabeculoplasty, iridotomy. Trabeculectomy – fistula between anterior chamber and subconjunctival space. With acute angle closure glaucoma, have to bring pressure down a bit first with systemic drugs so can give topical drops.
Cataracts

A cataract is any opacity on or within the lens. Found in 75% of over 65s. Cause blurred vision, change in spectacle lenses and dazzling.

Aetiology:
Senile – subcapsular (anterior or posterior), nuclear sclerotic (myopic shift), cortical.
Traumatic – penetrating, blunt (iris pigment), infrared radiation (glass blower’s – anterior lens capsule).
Metabolic – diabetes (earlier senile, osmotic overhydration), galactosaemia, hypocalcaemic syndromes etc.
Toxic – corticosteroids (post subcapsular), chemotherapy.
Secondary – anterior uveitis, high myopia.
Maternal infection or drug ingestion – rubella, toxoplasmosis, CMV.
Hereditary – dystrophia myotonica, atopic dermatitis.
Syndromes – Down’s, Alport’s etc.

Develops from immature (still get red reflex), to mature (cortex opaque), to hypermature (lens gets smaller and capsule wrinkles).

Management:
Assess patient and disability.
Correct other visual problems.
Take into account general health, exclude diabetes.
Treat by surgery – don’t need to wait until matures.
Use biometry – calculate required intraocular lens power.
If congenital need to treat rapidly to prevent amblyopia.

Cataract surgery:
Intracapsular cataract extraction – rarely performed, large wound.
Extracapsular cataract extraction – becoming rarer.
Phacoemulsification – normal method. Peel anterior lens capsule, fragment and aspirate lens with high frequency ultrasound probe, insert intraocular lens. This is better as smaller incision, faster recovery, less astigmatism, quick operation.
Complications – posterior capsule opacification, vitreous loss, retinal detachment, endophthalmitis, astigmatism.
Uveitis

Inflammation of iris (anterior – can see white cells and protein as flare in aqueous humour), ciliary body (intermediate) or choroid (posterior – inflammatory cells in vitreous).

50% associated with systemic disease – sarcoid, TB, Behcets, psoriasis, ankylosing spondylitis, IBD, syphilis, shingles.

Symptoms – pain (generally not with posterior uveitis), photophobia, blurring, redness, associated systemic symptoms.

Signs – ciliary injection (around limbus), keratitic precipitates (inflammatory cells on endothelium of cornea), hypopyon, posterior synechiae.

Treatment – steroids as eye drops (anterior) or systemic (posterior), dilate pupil, address cause.

Acute Visual Loss

Retinal vein occlusion:
Hypertension, diabetes, glaucoma, blood dyscrasias, hyperlipidaemia. Usually due to atheroma in artery occluding vein where it crosses or thrombotic.
Get haemorrhages all over retina or in distribution of drainage if branch vein. Tortuous and swollen veins.
Onset typically less acute than arterial occlusion.
Loss of vision, rubeotic glaucoma, macular oedema.

Retinal artery occlusion:
Can be embolic (fibrin-platelet, cholesterol, calcific) or vaso-obliterative. In young people may be migraine.
Rapid onset profound visual loss, which may clear (amaurosis fugax).
See cherry red spot – fovea is supplied by choroidal vessels so looks pink against pale retina.
Treatment – aim to get embolus to pass distally - ocular massage, paracentesis, breating carbon dioxide.

Ischaemic optic neuropathy:
Mono-ocular visual loss of blood supply to optic nerve is compromised.
Generally due to atherosclerosis or giant cell arteritis.

Optic neuritis:
Progressive visual loss with RAPD and red desaturation.
Eye movements may hurt.
Usually recovers but about 50% are related to MS.

Vitreous haemorrhage – neovascularisation, retinal detachment (especially myopes), injury.
Retinal detachment.
Chronic Visual Loss

- Cataracts
- Age related macular degeneration
- Diabetic retinopathy
- Glaucoma
- Choroiditis e.g. toxoplasmosis, TB, sarcoid.
- Malignant melanoma of choroid.

Trauma

History – type of injury, first aid given, past ocular history, tetanus.
If chemical injury wash out first – alkali is worse as can penetrate.

Full examination including visual acuity in both eyes. Give local anaesthetic if needed. Work systematically through parts of eye – orbit, eyelids, lacrimal apparatus, conjunctiva, cornea etc. examine pupils, eye position and movement, fields and sensation on cheek (as nerves go through orbit – V2). Investigations – pH, imaging for orbital injury or foreign body.

Blunt injury – corneal abrasion (very painful, but heal quickly), hyphaema (blood in anterior chamber, increased IOP), iris damage, lens dislocation, retinal oedema, retinal detachment, blow out fracture (periocular bruising, surgical emphysema, infra-orbital anaesthesia, diplopia, enopthalmus), vitreous haemorrhage.
Perforating injury – corneal lacerations, intraocular foreign body, iris prolapse. A hole in iris will give red reflex. Risk of infection, disturbance of ocular contents, sympathetic ophthalmitis.
Foreign bodies – subconjunctival haemorrhage, irregular pupil, iris prolapse, hyphaema, vitreous haemorrhage, retinal tears. Evert eyelids to check, give antibiotic drops.
Corneal foreign body – abrasion, rust ring, anterior uveitis, severe inflammatory reaction (if copper). Remove by topical anaesthetics, give antibiotics, dilate pupil to check, check under eyelids.
Chemical injury – acids coagulate, alkalis saponify. Need immediate irrigation. Topical antibiotics, steroids, vitamin C and dilating pupil also help.
Red Eye

Man be due to:

Haemorrhage
Subconjunctival - see posterior edge, small, localised, not serious, no pain.
Retrobulbar - proptosis, restricted movements, can't see back edge, sight threatening. Pressure on optic
nerve causes RAPD. Typically due to head injury or anaesthesia for eye surgery.
Commonly due to trauma, bleeding disorders, iatrogenic.
Congestion and vasodilatation – conjunctivitis, anterior uveitis, keratitis (corneal inflammation), acute
glaucoma, episcleritis.

Conjunctival redness – superficial vessels, especially in fornices, which blanch with topical
vasoconstrictors. Usually conjunctivitis.
Circumcorneal redness is most severe and needs referral. Deeper vessels (dusky red), most marked
around limbus. Can be due to keratitis, acute glaucoma or iridocyclitis.

Conjunctivitis:
Normal vision, clean cornea and anterior chamber, normal pupil.
Viral – sore, bilateral, stress related, watery discharge, follicles and glands involved, self limiting but very
contagious. Adenoviruses.
Bacterial – very sore, bilateral or unilateral, muco-purulent discharge, lid swelling, crusting and matting
lashes, needs treatment with antibiotics. Staph, strep, pneumococcus, haemophilus.
Usually self limiting but can give antibiotic eyedrops.

Keratitis – inflammation of cornea. Rapid progressive vision loss, severe pain, lid swelling, cornea
involved, hypopyon, constricted pupil, needs antibiotics. Immune, HSV (dendritic ulcers) or bacteria.

Iridocyclitis – blurred vision, ache, no lid swelling, keratic precipitates on cornea, constricted pupil,
usually immune mediated, dilate and give steroids.

Trachoma – commonest cause of blindness worldwide, subconjunctival fibrosis due to repeated
infections, corneal scarring and trichiasis (eye lashes directed backwards). Chlamydia is carried by
housefly.

Episcleritis – inflammation of superficial layer, less severe, self-limiting. Tender raised red area. Can be
caused by collagen diseases. Treat with steroids. Similar to hyctenular conjunctivitis (little bit of white in
middle of nodule).

Scleritis – more severe, associated with RA, pain, inflammation and ischaemia.
Optic disc swelling

On fundoscopy, disc has poorly defined edges, dilated vessels and haemorrhages.

Optic neuritis:
Generally swelling of disc, but not if retrobulbar.
Pain on movement, RAPD, red desaturation, central scotoma.
Acute loss of vision.
Often other transient neurological symptoms.
At risk of developing MS.

Papilloedema:
Bilateral disc swelling due to increased ICP.
Transient visual obscurations, progressive field loss, splinter haemorrhages, exudates, cotton wool spots, headache.
Normal visual field but large blind spot. No RAPD. May have obscurations (fleeting visual loss on change of posture).
Headache, nausea, diplopia.
Leads to optic atrophy.

Malignant hypertension:
Reduced vision, haemorrhagic disc swelling, retinal haemorrhages, exudates and cotton wool spots.
Raised blood pressure.

Anterior ischaemic optic neuropathy:
Either arteritic (due to inflammation of arteries) or non-arteritic.
If non-arteritic – ESR normal and no systemic symptoms. Generally atherosclerosis secondary to hypertension.

Optic atrophy – pale disc due to loss of nerve fibres. Usually reduced vision.
Diabetic Retinopathy

Common cause of visual impairment in under 50s.
Risk factors - duration of diabetes, control of diabetes, age, smoking, hypertension, hyperlipidaemia, pregnancy.

Due to vascular leakage or occlusion (leading to neovascularisation due to VEGF production).
Classified as non-proliferative (background - assymptomatic) or proliferative (sight threatened, cottong wool spots, new vessels, may have reduced vision). Either of these can lead to maculopathy.

Signs on fundoscopy:
Microaneurysms
Exudates – leaked lipoproteins
Haemorrhages – dot, blot or flame-shaped (nerve fibre layer).
Cotton wool spots – micro-infarction of nerve fibre layer.
Venous beading.
New vessels if proliferative – irregular, extending into vitreous, fragile and likely to bleed.
Retinal fibrosis, causing traction and retinal detachment.

Treatment:
Decrease risk factors.
Laser photocoagulation – either of new vessels or pan-laser-photocoagulation if in fovea (less ischaemic retina, so less stimulus for new vessel formation).

Other diabetic eye disease:
Iris neovascularisation – glaucoma.
Increased infections and cataracts.
Nerve palsies.
Decreased healing of corneal abrasions.
Macular Degeneration

Age related macular degeneration is the commonest cause of blindness in the UK. Leads to progressive loss of central vision. This is usually bilateral but one eye is presents initially. See drusen on fundoscopy – these are yellow spots due to lipids causing thickening of Bruch’s membrane. It is deeper, more refractile and less yellow than hard exudate. Normally, RPE is between the choroid and photoreceptors. Thickening of Bruch’s membrane disturbs RPE and causes atrophy of photoreceptors.

Dry (atrophic) AMD:
More common, less severe.
Gradual reduction of central vision.
Small drusen, atrophy of RPE, changes in pigmentation.
No treatment. Use low vision aids.

Wet (neovascular) AMD:
Relatively rapid, severe central visual loss.
As dry but also proliferation of fragile choroidal vessels in subretinal space.
Can cause retinal or vitreous haemorrhage.
Treat by laser photocoagulation or photodynamic therapy (infuse blood vessels with photosensitising agent, so laser is more selective). Depends on site – extrafoveal (laser), juxtafoveal or subfoveal (PDT).

Risk factors – age, smoking, CV disease, hypertension, hyperlipidaemia, low anti-oxidant levels.
Symptoms – blurring, distortion, loss of vision.
Investigations – visual acuity, reading speed, contrast sensitivity, fundoscopy, fluroscein angiography, optical coherent tomography.
Management – laser photocoagulation, PDT, pharmacological agents (anti-angiogenic, permeability and inflammatory agents), blind registration, low vision aids.
Retinal Disease

Retinal detachment:
Rhegmatogenous – most common, a hole in retina allows fluid to get under retina and pushes it up. In elderly people the vitreous degenerates and detaches from the retina giving floaters. This can cause a retinal hole (flashing lights). This may break vessels (vitreous haemorrhage) or cause detachment around the hole (progressive field loss).
Traction – e.g. fibrous vitreous diabetes.
Secondary – melanoma, after cataract surgery or trauma.
More common in myopes, people with retinal holes e.g. due to trauma, Marfan’s syndrome.
Symptoms – floaters, flashes (retina is abnormally stimulated – localise by shape, spots if retinal problem, zigzag lines if occipital cortex), field loss, falling acuity.
Treat urgently by closing hole, scleral silicone implants, cryotherapy, pneumatic retinoplexy, laser coagulation.


Central serous retinopathy – fluid between retina and RPE, distorting photoreceptor layer. Typically affecting young males. Due to localised breakdown in RPE structure. Gives micropsia as retina is stretched.

Retinal vascular disease:
Leakage – haemorrhages, oedema, exudates.
Occlusion – cotton wool spots, new vessels, ischaemia.
New vessel formation – occult (under RPE) or classic (between RPE and retina).

Retinitis pigmentosa – inherited, lose predominantly rods. Get tunnel vision and reduced night vision.

Macular pucker – macropsia. Fibrous tissue pulls in retina.

**Visual Rehabilitation**

**Causes of visual impairment:**
Children – congenital cataracts, optic atrophy, albinism, congenital glaucoma, myopia, retinopathy of prematurity.
Adults – diabetic retinopathy, myopia, uveitis, corneal dystrophies, macula degeneration, retinitis pigmentosa.
Elderly – age related macular degeneration, glaucoma, cataract, diabetic retinopathies.

**Services:**
Vision assessment
Magnification devices – with or without light, hand held or stand.
Other devices – CCTV, software, voice activated, screen readers, Braille keyboards, talking books or watches, typoscopes (reduce glare), tactile stickers, kitchen aids.
Liaison with others

**Blind registration:**
Visual impairment – VA <6/60, or up to 6/24 with field contraction, or severe field loss.
Severe visual impairment – VA <3/60, or less than 6/60 with field constriction.

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**Optometry**

Emmetropic – no refractive error.
Ametropic – refractive error, image is not focussed on retina. Correct with lenses.
Refractive – myopia, hypermetropia.
Axial ametropia – astigmatism.

Contact lenses can be used for – refractive errors, aphakia, irregular corneas, occupational, cosmetic or psychological reasons. Complications include – giant papillary conjunctivitis, corneal abrasions, infective keratitis, corneal ulcers, neovascularisation, corneal hypoxia.

Accommodation – refractive power of lens can be changes by ciliary muscle. Amplitude of accommodation starts to decrease at about 40 years due to thickening of lens making near vision difficult (presbyopia).
Orthoptics

Children are born with poor vision and no ability to integrate the input from each eye. Normally, they develop binocular vision by about 4 months. This involves:
- Simultaneous perceptions – overlapping image from each eye.
- Fusion – moving eyes together.
- Stereopsis – 3D vision.

A squint (strabismus) occurs when one eye has an abnormal position so both eyes cannot focus on a target at the same time. This means that input to one eye is reduced and may lead to poor acuity in that eye and no binocular vision. This must be treated by patching before 8 years of age, whilst the brain retains elasticity, so vision develops.

There are 2 types of squint:

**Manifest squint:**
- Heterotropia. Exo = divergent, eso = convergent, hyper = looking up, hypo = looking down.
- There is deviation of one eye when fixing on a target so no binocular vision.
- Either get double vision (adult) or suppression of one eye (children).

**Latent squint:**
- Heterophoria.
- Underlying tendency for eyes to deviate a small degree when covered (return to rest position).
- Present in 80% population.
- Rarely causes problems.

Squints are also divided into:
- **Concomitant (non-paralytic)** – both eyes move normally, angle of squint is constant, due to supranuclear abnormalities.
- **Inconcomitant (paralytic)** – one eye moves abnormally so angle of squint depends on direction of gaze, due to problems with eye muscles or nerves.

Investigate squints by cover tests.

**Cover – uncover:**
- To look for manifest squint.
- Look at a light (so don’t accommodate). Can also do with near target (more likely to converge) and with glasses (hypermetropic prescriptions relax accommodation and reduce convergent squints, myopic prescriptions worsen convergent squints).
- Look at other eye moving to take up focus. The eye that moves is the squinting eye.

**Alternate cover test:**
- For latent squint.
- Flick cover between eyes so can’t use together.
- Look at eye as cover is removed moving from rest position to take up focus.

Management of squints:
- Correct refractive error. Accommodative amblyopia is when uncorrected hypermetropia means child must accommodate to see clearly. Accommodation is linked to convergence so child must resist this. If they do not a squint develops which is worse when looking at close things or if the child is tired.
Treat amblyopia (weakness in vision in one eye without pathological cause – due to uncorrected refractive error - ametropic, uneven refractive error - anisometric, lack of sensory input, squints - strabismic), generally by patching good eye.

Surgery – for cosmetic appearance.

Diplopia:
Only get this in adults with squint due to muscle problem as can’t adapt.
Type of diplopia can indicate problem.
May have abnormal head position to counteract this e.g. turning head with CN6 palsy, dip chin with CN4.
Prisms can be used to measure and correct double vision.
Otherwise occlusion and often recovers or surgery.
Paediatric Ophthalmology

Vision develops after birth – mainly as a result of brain development. This requires stimulus. New born babies usually have astigmatism, infants are typically hypermetropic.

**Congenital abnormalities**
- Sclerocornea
- Coloboma – iris doesn't fuse properly.
- Cataracts – either part of syndrome, inherited, due to infection e.g. rubella or cryptogenic.
- Capillary hemangioma – only a problem if on eyelid as causes stimulus deprivation.
- Glaucoma – causes extreme photophobia, eye watering, globe enlargement.
- Aniridia – related to Wilm’s tumour.
- Optic nerve hypoplasia – not genetic, due to pre-natal insult.

**Retinopathy of prematurity**
Retina does not grow out to the peripheries then starts to grow too fast so vessels grow into the vitreous. The vitreous becomes organised and contracts so the retina is avulsed. This is treated by laser. Associated with prematurity, low birth weight, supplemental oxygen.

**Opthalmitis neonatorum**
This is a notifiable condition. It is commonly caused by gonococcus, Chlamydia, S. aureus, and E. coli. Gonococcus is the most serious as pus builds up and puts pressure on the eyes and damages the cornea.

**Orbital cellulitis**
Typically due to ethmoid sinus infection. Can be pre or post septal. Post-septal is very serious as can lead to cavernous sinus thrombosis.

**Retinoblastoma**
Causes leukocoria. Can be sporadic or inherited. Also: Retinal haemorrhage, Viral conjunctivitis
Sticky eye – due to poor drainage, usually resolves.

**Important Note**
These notes were written by Liz Tatman, as a fourth year medical student in 2006. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources. Disclaimer: no responsibility can be taken by either the author or publisher for any loss, damage or injury occasioned to any person acting or refraining from action as a result of this information.

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