In the fovea region

• The fovea is the central region of the retina (in the macula region) that provides for the most clear vision (central vision). In the fovea, there are very few rods (with none at the foveola). There are mostly cones. The cones are also packed closer together here in the fovea than in the rest of the retina.

• The fovea is employed for accurate sharp central vision in the direction where it is pointed. It comprises less than 1% of retinal size but takes up over 50% of the visual cortex in the brain.

• The fovea sees only the central two degrees of the visual field, (approximately twice the width of your thumbnail at arm’s length). If an object is large and thus covers a large angle, the eyes must constantly shift their gaze to subsequently bring different portions of the image into the fovea (as in reading).

• The fovea also grossly corresponds to the retinal avascular zone (no blood vessels). This allows the light to be sensed without any dispersion or loss e.g. so light has a direct path to the photoreceptors.

• The fovea receives most of its oxygen from the vessels in the choroid, which is across the retinal pigment epithelium and Bruch's membrane.

• The high spatial density of cones along with the absence of blood vessels at the fovea accounts for the high visual acuity capability at the fovea.

• The centre of the fovea is the foveola (about 0.2 mm in diameter) - a central pit where only cone photoreceptors are present and there are virtually no rods.

• These cones are very densely packed

• In the fovea few cones target a bipolar cell.

• At the foveola there is a unique 1 cone: 1 bipolar cell : 1 ganglion cell connection. This results in a much smaller receptive field of ganglion cells (compared to peripheral region) => greater visual acuity (due to greater spatial frequency resolution)
PHYSIOLOGY OF THE RETINA

• The **lens of the eye** projects an **inverted image** onto the retina.

• The crystalline lens is a transparent, **biconvex (convergent lens with a positive power)** structure in the eye that, along with the cornea, helps to focus light to be focused on the retina.

• **The lens, by changing shape, functions to change the focal distance of the eye** so that it can focus on objects at various distances, thus allowing a sharp real image of the object of interest to be formed on the retina. **This adjustment of the lens is known as lens accommodation.** Accommodation is similar to the focusing of a photographic camera via movement of its lenses.

• The iris is a thin, circular structure in the eye, responsible for controlling the diameter and size of the pupil (aperture) and thus the amount of light reaching the retina. The color of the iris is often referred to as "eye color."

• In optical terms, the pupil is the eye’s aperture and the iris is the aperture stop.
Refraction

- A light ray is bent (refracted) when it strikes the surface of a medium of different density from the one in which it had been travelling (for example, moving from air into glass, or...
**convex lens (positive power e.g positive dioptres)**, which converges light rays before they reach the eye

- **Presbyopia = Age related longsightedness**: the effects of age on the lens (loses its natural elasticity) resulting in a progressively diminished ability to focus on near objects (age related longsightedness). Corrected with **convex lens (positive power e.g positive dioptres)**, which converges light rays before they reach the eye.

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**The loss of accommodation with age**

- The lens can alter its shape and hence the eye's effective focal length - a function called accommodation. This process allows the eye to focus on near objects.
- **Myopia** (short sightedness with a long eye) which is corrected with divergent concave (negative power) lens
- **Hypermetropia** (hyperopia or long sightedness with a short eye) which is corrected with a convergent convex (positive power) lens
- **Presbyopia** (age related longsightedness): With age the elastic properties of the lens deteriorate so that the amplitude of accommodation is reduced. This is presbyopia (age related long sightedness). Therefore, most adults need reading glasses (convergent convex lens with positive power) at age 40-45.

**Photoreceptors in the Retina**

- The retina contains a sheet of photoreceptors. The visual image (inverted) from the lens is focused on the retina with minimal distortion.
- Light is focused by the cornea and the lens and crosses the vitreous humor to reach the photoreceptors in the retina (deep layer of retina)
- Behind the photoreceptors is the pigment epithelium containing melanin (black). This functions to absorb light which prevents light not captured by the retina being reflected back.
- Light must travel through the layers of other cells in the retina before reaching the photoreceptors, degrading the visual image.
- To minimise light scattering and absorption, the cells in the proximal layers of the retina have unmyelinated axons so they are relatively transparent. In addition, the fovea region is avascular to enhance central visual acuity.
- To ensure that the ‘field of interest’ is projected onto the fovea we constantly move our eyes (and head) => gaze
- Optic nerve fibres leave the retina at the optic disc. There are no photoreceptors in this region therefore we have a blind spot in the visual field. **BLIND SPOT.**
• The structures entering through the optic canal are as follows:
  ➢ Optic nerve (CN II)
  ➢ Ophthalmic artery (central retinal artery is a branch of this artery)
  ➢ Central retinal vein

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The eye

• Conjunctiva: lines the inside of the eyelids and covers the sclera (white part of the eye). It is composed of non-keratinized, stratified columnar epithelium with goblet cells. The conjunctiva helps lubricate the eye by producing mucus and tears, although a smaller volume of tears than the lacrimal gland. It also contributes to immune surveillance and helps to prevent the entrance of microbes into the eye.

• Sclera: The sclera also known as the white of the eye, is the opaque, fibrous, protective, outer layer of the eye (surrounds whole eye) containing collagen and elastic fiber. The sclera is continuous with the cornea (which sits anterior to pupil and lens)
• Look at each eye individually when performing the “H” eye movement test.

Clinical testing of CN III, IV & VI

eyeball fully adducted to test superior and inferior oblique

does not matter whether you are testing the movements of the right or the left eye

eyeball fully abducted to test superior and inferior rectus
Pupillary light reflex

- Afferent = Retina + CN II
• Efferent = CN III (parasympathetic)

• Light shone in one eye elicits a pupillary light reflex that causes both the ipsilateral and the contralateral pupil to constrict (the direct and consensual reflex, respectively).

• Interestingly, this reflex does not depend on the presence of retinal rods or cones. Instead, a special subset of intrinsically photosensitive ganglion cells provide the afferent limb (CN II), with axons that distribute bilaterally in the optic chiasm and terminate in the pretectal area (midbrain).

• The pretectal area projects bilaterally to the Edinger-Westphal nuclei (the preganglionic parasympathetic component of the CN III oculomotor nucleus).

• Because light in either eye causes both the ipsilateral and the contralateral pupil to constrict (direct and consensual reflexes, respectively), both pupils ordinarily will be pretty much the same size under any given condition of illumination; if they are not (anisocoria), there likely is a problem with the autonomic innervation or with the iris itself.
The corneal reflex, also known as the blink reflex, is an involuntary blinking of the eyelids elicited by stimulation of the cornea (such as by touching or by a foreign body), or bright light, though could result from any peripheral stimulus.

Stimulation should elicit both a direct and consensual response (response of the opposite eye). The reflex consumes a rapid rate of 0.1 second. The evolutionary purpose of this reflex is to protect the eyes from foreign bodies and bright lights (the latter known as the optical reflex).

The reflex is mediated by:

- **Afferent**: ophthalmic branch (CN V1) of CN V (trigeminal nerve) sensing the stimulus on the cornea, lid, or conjunctiva (avoid touching the cornea!)

- **Efferent**: CN VII (Facial nerve) initiating the motor response (blinking) e.g. contraction of obicularis oculi

**Refractive Errors**

- **Emmetropia**: normal eyes with no refractive error

- **Hypermetropia or hyperopia** (long sightedness with short eye): defect of vision caused by an imperfection in the eye (often when the eyeball is too short or the lens cannot become convex enough), causing difficulty focusing on near objects and the image forming behind the retina. Corrected with convergent convex (positive dioptre lens).

- **Myopia** (shortsightedness - what I have with long eye): condition of the eye where the light that comes in does not directly focus on the retina but in front of it (often because eye is too long or lens is too convex). A defect of vision that causes the image that one sees when looking at a distant object to be out of focus, but in focus when looking at a close object. Corrected with divergent concave (negative dioptre lens).

- **Astigmatism**: optical defect in which vision is blurred due to the inability of the optics of the eye to focus a point object into a sharp focused image on the retina. This may be due to an irregular or toric curvature of the cornea or lens. **Corrected with toric lens.**

- **Presbyopia**: age related long sightedness (hypermetropia/hyperopia) - comes to us all. The ability to focus on near objects declines throughout life, from an accommodation of about 20 dioptres (ability to focus at 50 mm away) in a child, to 10 dioptres at age 25 (100 mm), and levels off at 0.5 to 1 dioptre at age 60 (ability to focus down to 1–2 meters only). Corrected with convex (positive dioptre lens) e.g. reading glasses.

**Taking an ophthalmic history**

**Visual symptoms**
• **Work superficial to deep:** red eye, discharge, crusting, pain, loss of vision, blurred vision, flashers and floaters, photophobia

• **Visual loss**
  - Site (localisation): central scotoma, peripheral scotoma, complete visual loss, bilateral?
  - Onset? Timing and duration? Evolution? Gradual or sudden? How long did it last for? Getting better or worse?
  - Character: Transient, improving?
  - Associated symptoms e.g. headache, N&V, ophthalmological systematic enquiry
  - Progressive or non-progressive?
  - Exacerbating or alleviating factors
  - Severity

• **Glare:** Glare is difficulty seeing in the presence of bright light such as direct or reflected sunlight or artificial light such as car headlamps at night. Eye diseases such as cataracts, macular degeneration and uveitis can cause glare.

• **Distortion (metamorphopsia):** Metamorphopsia is a type of distorted vision in which a grid of straight lines appears wavy and parts of the grid may appear blank. People with this condition often first notice this when looking at mini-blinds in their home. It is mainly associated with macular degeneration, particularly wet type age-related macular degeneration with choroidal neovascularisation.

• **Photophobia:** Symptom of abnormal intolerance to visual perception of light e.g. an experience of discomfort or pain to the eyes due to light exposure or by presence of actual physical sensitivity of the eye. Can occur as a result of several different medical conditions, related to the eye or the nervous system including migraine headaches, cataracts, mild traumatic brain injury (MTBI), meningitis, SAH, or severe ophthalmologic diseases such as uveitis or corneal abrasion. Often sinister finding.

• **Floaters:** Floaters are deposits of various size, shape, consistency, refractive index, and motility within the eye’s vitreous humour, which is normally transparent. Floaters are dark specks in the form of dots, circles, lines, that seem to move across your field of vision. At a young age, the vitreous is transparent, but as one ages, imperfections gradually develop. The common type of floater, which is present in most people’s eyes, is due to degenerative changes of the vitreous humour. Often associated with VH or RD.

• **Flashing lights:** Many causes including retinal diseases such as retinal tears and detachment (serious and uncommon), or, diseases affecting jelly-like fluid called the vitreous in front of the retina. **A more common cause of flashing lights is migraine.** Retinal detachemnts
Examination: equipment

- Acuity chart (Snellen chart) +/- Ishihara colour perception tests
- Pen torch
- Magnifying aid
- Ophthalmoscope
- Eye drops for examination

- **Fluorescein (orange liquid):** Fluorescein sodium eye drops is used extensively as a diagnostic tool in the field of ophthalmology and optometry. Fluorescein can only enter damaged cells of the eye. It will temporarily stain any cells it enters and therefore marks any damaged areas of the eye. Can be used for detecting damage on the surface of the eye, such as abrasions or ulcers, either on the conjunctiva (membrane that covers the white part of the eyeball and the inside of the eyelids) or on the cornea (transparent front part of the eye that covers the iris, pupil and anterior chamber). Remember that the cornea is continuous with the sclera. **STAINS DAMAGE GREEN under COBALT BLUE LIGHT.**

- **Anaesthetic drops** – particularly useful for removing foreign body

- **Mydriatic drops:** topical eye drops (*e.g.* tropicamide, cyclopentolate, atropine, phenylephrine) which cause mydriasis (dilation of the pupil). These drugs can be used for fundoscopic examination of the retina and other deep structures of the eye, and also to reduce painful ciliary muscle spasm. Purposely-induced mydriasis via mydriatics is also used as a diagnostic test for Horner's Syndrome. **Must be careful using mydriatic drops as can precipitate closed angle glaucoma (very low risk)**

- **NB:** Miosis = constriction of the pupil (as is seen in Horner's syndrome) - sympathetic mediated

- **NB:** Mydriasis = dilation of the pupil – CN III mediated (as is seen in CN III palsy)
Hyphema

- Hyphema is blood in the front (anterior) chamber of the eye. It may appear as a reddish tinge, or it may appear as a small pool of blood at the bottom of the iris or in the cornea.

- Hyphemas are frequently caused by injury, and may partially or completely block vision. The most common causes of hyphema are intraocular surgery, blunt trauma, and lacerating trauma.

Other complications of blunt trauma

- Tearing of intra-ocular structures

- Blow out fracture of orbit

- Dislocated lens (ectopia lentis)
CN VI nerve palsy

- Sixth nerve palsy (abducent nerve palsy) is a disorder associated with dysfunction of cranial nerve VI (the abducent nerve), which is responsible for contracting the lateral rectus muscle to abduct the eye.

- Results in lateral rectus palsy and **failure to abduct the affected eye**

- The condition is commonly unilateral but can also occur bilaterally

- Unilateral abducent nerve palsy is the most common of the isolated ocular motor nerve palsies

- The inability of an eye to turn outward (abduct) results in a **manifest convergent strabismus-squint (esotropia)** of which the primary symptom is **diplopia** in which the two images appear side-by-side (e.g. **horizontal diplopia**).

- Strabismus (squint) is a condition in which the eyes are not properly aligned with each other. It typically involves a lack of coordination between the extraocular muscles, which prevents bringing the gaze of each eye to the same point in space. It thus **hampers proper binocular vision**, and which may **adversely affect depth perception**, and **cause diplopia**

- **Esotropia** is a form of strabismus, or "squint", in which one or both eyes turns inward e.g. a **convergent squint** on distance fixation. On near fixation the affected individual may have only a latent deviation (esophoria) and be able to maintain binocularity or have an esotropia of a smaller size.

- The condition can be constantly present, or occur intermittently, and can give the affected individual a "**cross-eyed**" appearance

- The striabmus and esotropia can result in horizontal diplopia (double vision)

- Diplopia is typically experienced by adults with CN VI nerve palsies, but children with the condition may not experience diplopia due to suppression. The neuroplasticity present in
Clinical features

- Contralateral homonomous defects (right or left visual field affected, not both)
- **Contralateral homonomous hemianopia or quadrantanopia** (depending on location)
- Macula not spared
- Incongruous (not symmetric)

Occipital visual cortex (calcarine sulcus)

Aetiology:

- Vascular disease
- Demyelination

Clinical features:

- **Contralateral homonomous hemianopia defect**
- Often macular sparing
- Congruous (symmetric)
OCULAR PHARMACOLOGY

Routes of administration

Topical:

- Required to act on surface
- Require corneal penetration
- Drops vs Ointment

Corneal penetration

- **Good for LMW** (low molecular weight) drugs
- Hydrophilic drugs limited by epithelium (phospholipid bilayer)
- Hydrophobic drugs limited by stroma

Topical steroids

- **Need hydrophobic and hydrophilic capability**
- Chemical modification to steroid alters its characteristics
- Alcohol or acetate makes steroid more hydrophobic
- Phosphate makes it more hydrophilic

**Side effects:** raised IOP and cataracts

- Prednisolone acetate
  - hydrophobic
  - Good penetration in uninflamed cornea
  - Used post-operatively
- Prednisolone phosphate
  - Hydrophilic
  - Poor penetration in uninflamed cornea
• Used to deliver Anti-vascular endothelial growth factor (Anti-VEGF) e.g. Ranibizumab, a monoclonal antibody fragment Fab used for the treatment of age-related macular degeneration (wet type) and in some other conditions causing neovascularisation.

Endophthalmitis

• Endophthalmitis is an inflammation of the internal coats of the eye involving the vitreous and/or aqueous humors. It is a possible complication of all intraocular surgeries, particularly cataract surgery, with possible loss of vision and the eye itself.

• Infectious etiology is the most common and various bacteria and fungi have been isolated as the cause of the endophthalmitis. Other causes include penetrating trauma and retained intraocular foreign bodies.

• Sight threatening

Local Anaesthetic

Blocks sodium channels and impedes nerve conduction

Used extensively in ophthalmology for

• FB (foreign body) removal

• Tonometry (IOP measurement)
• Transient loss of vision
• Gradual loss of vision

**Causes of sudden visual loss**

Use the mnemonic ‘VARICOSE’:

• Vascular occlusion and vitreous haemorrhage
• Age related macular degeneration (wet type)
• Retinal detachment (RD)
• Ischaemic optic neuropathy – arteritic and non-arteritic
• Closed angle glaucoma (ACAG)
• Optic neuritis
• Stroke
• **Emergency referral**

**Central retinal artery occlusion (CRAO)**

• Central retinal artery occlusion (CRAO) is a disease of the eye where the flow of blood through the central retinal artery is blocked (occluded). There are several different causes or aetiologies of this occlusion, the most common is carotid artery atherosclerosis (therefore take a CV history, CV risk factory history +/- carotid Doppler).
• CRAO is an ocular analogue of cerebral stroke.

**Symptoms**

• **Sudden, profound visual loss** (e.g. can’t even count fingers, may detect light)
• **Painless** (in contrast to optic neuritis which is often painful on eye movements)

**Signs**

• **Relative afferent pupillary defect (RAPD):** A relative afferent pupillary defect (RAPD) is a medical sign observed during the swinging-flashlight test whereupon the patient’s pupil (of the affected eye) constricts less, therefore appearing to dilate, when a bright light is swung
• Leakage of proteins => soft and hard exudates

• Infarction of nerve fibres can also cause cotton wool spots

• Eventually retina ischaemic causes release of VEG F which causes neovascularisation

• Neovascularisation can cause VH, scarring, RD, and rubreosis iridis

**Symptoms of non proliferative diabetic retinopathy (NPDR)**

• Diabetic retinopathy often has no early warning signs. Even macular oedema, which may cause vision loss more rapidly, may not have any warning signs for some time. In general, however, a person with macular oedema is likely to have blurred vision, making it hard to do things like read or drive.

• In the first stage which is called non-proliferative diabetic retinopathy (NPDR) there are no symptoms, and patient may have 20/20 vision. However abnormalities can be detected by fundus photography (hence why DM patients have yearly fundoscopic screening).

• Microaneurysms: on fundi photography we can see microaneurysms (microscopic blood-filled bulges in the artery walls). If there is reduced vision, fluorescein angiography can be done to see the back of the eye. Narrowing or blocked retinal blood vessels can be seen clearly which result in retinal ischemia (lack of blood flow). **Microaneurysms can rupture and leak blood vessel contents into retina** => dot, blot or flame-shaped haemorrhages

• Macular oedema: may occur in which **blood vessels leak contents** into the macular region. This can happen at all stages of NPDR. The macular oedema symptoms are blurring, darkening or distorted images with not the same between two eyes. 10 percent of diabetic patients will get vision loss related with macular oedema. **If within one disc diameter from fovea => referable. Can be Tx with macular grid photocoagulation or anti-VEGF.**
Classification:

- Mild Nonproliferative Retinopathy. At this earliest stage, microaneurysms occur. They are small areas of balloon-like swelling in the retina's tiny blood vessels. At least one microaneurysm, and also dot, blot or flame-shaped haemorrhages in all four fundus quadrants.

- Moderate Nonproliferative Retinopathy: As the disease progresses, some blood vessels that nourish the retina are blocked. Intraretinal microaneurysms and dot and blot haemorrhages of greater severity, in one to three quadrants. Cotton wool spots, venous calibre changes including venous beading, and intraretinal microvascular abnormalities are present but mild.

- Severe Nonproliferative Retinopathy: Many more blood vessels are blocked, depriving several areas of the retina with their blood supply. At least one of the following should be present: a) ‘severe’ haemorrhages and microaneurysms in all four quadrants of the fundus, b) venous beading, which is more marked in at least two quadrants, and c) intraretinal microvascular abnormalities, which are more severe in at least one quadrant.

**Signs of non proliferative diabetic retinopathy (NPDR)**

- Microaneurysms
- Dot + blot haemorrhages
- Hard exudates (due to excavation of proteins from ruptured microanerysms)
- Cotton wool spots (caused by damage to nerve fibers and are a result of accumulation of axoplasmic material within the nerve fiber layer)
- Abnormalities of venous calibre e.g. venous beading
- Intra-retinal microvascular abnormalities (IRMA)
• The first time this happens, it may not be very severe. In most cases, it will leave just a few specks of blood, or spots, floating in a person's visual field (e.g. floaters), though the spots often go away after a few hours. It is often transient (in contrast to RD).

• These spots are often followed within a few days or weeks by a much greater leakage of blood, which blurs vision. In extreme cases, a person will only be able to tell light from dark in that eye. It may take the blood anywhere from a few days to months or even years to clear from the inside of the eye, and in some cases the blood will not clear. These types of large hemorrhages tend to happen more than once, often during sleep.

• Without timely treatment, these new blood vessels can bleed, cloud vision, and destroy the retina. Fibrovascular proliferation can also cause tractional retinal detachment.

• The new blood vessels can also grow into the angle of the anterior chamber of the eye and cause neovascular glaucoma.

**Signs of proliferative diabetic retinopathy (PDR)**

• Cotton wool spots

• Flame hemorrhages and dot-blot hemorrhages.

• **New blood vessels (neovascularization)** along the retina and in the clear, gel-like vitreous humour that fills the inside of the eye.

New vessels can:

• Grow on disc (new vessels on disc NVD)

• Grow in the periphery (new vessels elsewhere NVE)

• Grow on iris if ischaemia is severe (can result in neovascular glaucoma)
Associations with other causes of red eye

- Conjunctivitis
- Keratitis
- Episcleritis
- Note: can also be associated with dermatological conditions

Symptoms

- Redness of the eyelids
- Flaking of skin on the eyelids – “dandruff”
- Red eye
- Gritty eyes or foreign-body sensation
- Mild discharge

Signs of anterior blepharitis

Signs are present at lid margin

Staphylococcal:

- Lid margin red
- Lashes distorted, loss of lashes, ingrowing lashes (trichiasis)
• Foreign body sensation e.g. gritty eye

• Discharge: sticky eye especially if bacterial

• Watering (epiphoria) especially if viral

• Itch (especially if allergic)

**Conjunctivitis**

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**Signs**

• **Red eye**: note pattern of redness (forniceal injection) - more diffuse towards the fornices (greatest in conjunctival fornices under eye lids)

• Discharge: serous or mucopurulent (purulent discharge suggests bacterial infection)

• Papillae (vascular structures that appear to the naked eye as soft red swellings) – bacterial

• **Follicles**: suggests viral or chlamydial

• Subconjunctival haemorrhage (bleeding underneath the conjunctiva)

• Chemosis = oedema (swelling) of the conjunctiva

• Pre-auricular lymph nodes (suggestive of viral)
• **Serious disease**

• Scleritis is a serious inflammatory disease that affects the white outer coating of the eye, known as the **sclera**.

• The disease is often contracted through association with other diseases of the body, such as Wegener's granulomatosis or rheumatoid arthritis or connective tissue disease.

• **Episcleritis is inflammation of the episclera**, a less serious condition that seldom develops into scleritis.

• **Painful and injection (redness) of deep vascular plexus**

• **Widespread**

• **Very painful**

• **Phenylephrine test**: Scleritis may be differentiated from episcleritis by using phenylephrine eye drops, which causes blanching of the blood vessels in episcleritis, but not in scleritis

• Associated uveitis common

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*Episcleritis versus scleritis*
Topical antihypertensive drops e.g. topical beta blocker such as timolol

Topical steroids

Pilocarpine (once IOP < 50mmHg): Pilocarpine is a Parasympathomimetic (muscarinic agonist) drug which acts on a subtype of muscarinic receptor (M3) found on the iris sphincter muscle, causing the muscle to contract and engage in miosis (constriction of pupil). Pilocarpine (muscarinic agonist) also acts on the ciliary muscle and causes it to contract (lens accommodation). When the ciliary muscle contracts, it opens the trabecular meshwork. This action facilitates the rate that aqueous humor leaves the eye to decrease intraocular pressure (miosis also helps due to effects on iris muscles)

YAG Laser Peripheral Iridotomy (PI) creates alternative flow pathway for aqueous, and reduces risk of recurrence. The surgeon uses an Nd:YAG laser to create a small hole in the peripheral iris. This improves the circulation of fluid inside the eye and widens the anterior chamber angle. Fluid which is produced behind the iris has easier access to the eye’s internal drainage system. Sometimes this lowers the intraocular pressure, but that is not the primary goal of laser peripheral iridotomy. The primary goal of the procedure is to lessens the risk of acute angle-closure glaucoma. The other ("normal") eye should also receive prophylaxis treatment as at risk.

Subconjunctival haemorrhage

A subconjunctival hemorrhage is bleeding underneath the conjunctiva. The conjunctiva contains many small, fragile blood vessels that are easily ruptured or broken. When this happens, blood leaks into the space between the conjunctiva and sclera.

Such a hemorrhage may be caused by a sudden or severe sneeze or cough, or due to hypertension or as a side effect of blood thinners. It may also be caused by heavy lifting, vomiting, or even rubbing one’s eyes too roughly.

Can also be caused due to trauma

PAINLESS

Although its appearance may be alarming, in general a subconjunctival hemorrhage is a painless and harmless condition; however, it may be associated with high blood pressure, trauma to the eye, or a base of skull fracture if there is no posterior border of the haemorrhage visible.
• Conjunctivitis is inflammation of the conjunctiva (the outermost layer of the eye and the inner surface of the eyelids). It is commonly due to an infection (usually viral, but sometimes bacterial) or an allergic reaction.

Classification can be either by cause or by extent of the inflamed area.

By cause:

• Allergic conjunctivitis (atopic conjunctivitis)
• Bacterial conjunctivitis including chlamydial - pappiale, purulent
• Viral conjunctivitis – follicles, preauricular lymphadenopathy, epiphora
• Chemical conjunctivitis
• Neonatal conjunctivitis is often defined separately due to different organisms.

By extent of involvement:

• Blepharoconjunctivitis is the dual combination of conjunctivitis with blepharitis (inflammation of the eyelids).
• Keratoconjunctivitis is the combination of conjunctivitis and keratitis (corneal inflammation)
Conjunctivitis in contact lenses wearers

- Acanthamoeba (protozoa found in water)
- Pseudomonas aeruginosa (gram –ve bacterium also found in water) – resistant to chloramphenicol
- Treat with ofloxacin rather than chloramphenicol
- Be sure to rule out corneal abrasion keratitis, or ulcerative keratitis – fluroescien

Viral conjunctivitis

- Viral conjunctivitis is often associated with an infection of the upper respiratory tract, a common cold, and/or a sore throat.

Symptoms

- Viral conjunctivitis, commonly known as pink eye, shows a fine, diffuse pinkness of the conjunctiva, which is easily mistaken for the ciliary injection of iritis, but there are usually corroborative signs on microscopy, particularly numerous lymphoid follicles on the tarsal conjunctiva => MUST LOOK UNDER EYE LIDS
- Conjunctival redness (injection): conjunctival injection around the conjunctival fornices => forniceal injection
- Excessive watering (epithora) is characteristic of viral infections (in contrast to micropurulent discharge, grittiness and crusting which is characteristic of bacterial infections)
- Itching (pruritus)
- Pre auricular lymphadenopathy
- Follicles
• If treated with steroids can cause a corneal melt and perforation of the cornea (as steroids cause immunosupression) => DONT give steroids

• Tx with Aciclovir

• Dendritic ulcer (seen below) is very characteristic

![Dendritic ulcer](image)

**Adenoviral keratitis**

• Bilateral

• Usually follows an URTI e.g. pneumonia or common cold

• Contagious

• Can give topical AB to prevent secondary bacterial infection

• May affect vision

• Can require steroids to speed up recovery if becomes chronic e.g. development of subepithelial infiltrates

**Fungal keratitis with hypopyon**

• More indolent course (slowly progressive) than microbial keratitis

• Usually a history of **trauma from vegetation**

• Takes a long time to heal
Management:

- Intravitreal injection of amikacin and vancomycin (used to treat Staph epidermidis)
- Topical antibiotics
• Type II: cytotoxic: \textbf{IgG/IgM/complement mediated} + MAC (membrane attack complex which causes lysis) killing via complement

• Type III: immune complex: \textbf{IgG/IgM/complement} mediated but no activation of MAC

• Type IV: delayed type hypersensitivity reaction (DTHS) – cell mediated e.g. CD4, CD8, macrophages

Type 1 HS: Acute allergic conjunctivitis

• Chemosis (oedema/swelling)

• Redness (mainly due to vasodilatation e.g. hyperaemia)

• Epiphora (excessive tears)

• Pruritus (itching)

• \textbf{Often combined with rhinitis}

• \textbf{Most common cause is hayfever – ask about allergies}

Type II cytotoxic: Moorens ulcer

• Moorens ulcer is autoimmune condition (AI)

• Rapidly progressive, painful, ulcerative keratitis (cornea inflammation) which initially affects the peripheral cornea and may spread circumferentially

Type III immune complex: Idiopathic Iritis

• Uveitis is often immune complex mediated (type 3)

• Examplues include SLE
• Observe corneal reflections: hold a pen torch at 1/3 metre and see if the reflections are symmetrical. **The norm being slightly nasal.**

• **If asymmetrical, a manifest squint is suspected.** If the reflection is more nasal than normal this is suggestive of an esotropia. If the reflection is more temporal than normal this suggests an exotropia.

• Using an occluder cover the “straight” (fixing) eye and observe the movement of the uncovered eye.

**Esotropia (cross eyed)**

• **If the uncovered eye moves out (adducts) to take up fixation, it has been in a convergent position i.e. a manifest convergent squint or esotropia.**

• The uncovered eye moves out from its convergent position to take up fixation in an esotropia.

• Notice under the cover the “straight eye” is now convergent.

**Exotropia (wall eyed)**

• Cover the “normal” fixing eye. **If the uncovered eye moves in (adducts) to take up fixation,** it has been in a divergent position i.e a manifest divergent squint or exotropia.
• If the corneal reflections are symmetrical and no manifest deviation has been found, a latent deviation (phoria strabismus) is suspected.

• If latent strabismus is present it WILL BE IN BOTH EYES (binocular vision is NOT impaired)

• To detect a latent deviation: observe the movement of the eye which has been occluded as the occluder is taken away i.e. as the eye is uncovered. Repeat with the other eye. This is in contrast to the manifest squint COVER test, where we cover the “normal eye” and observe the movement of the other eye e.g. we observe only on covering NOT uncovering

• In a latent deviation, binocular single vision is present, and the occluder acts to dissociate the eyes and inhibit fusion. The movement that is seen as the occluder is removed is a recovery movement as fusion is regained (See figures 3 and 4 below)

• Therefore, in the cover-uncover test, we cover both eyes (separately) and observe each eye as it is uncovered.

• If the eye moves out as it is uncovered, it has been in a convergent position e.g. a latent convergence or esophoria.

• If the eye moves in as it is uncovered, it has been in a divergent position e.g. a latent divergence or exophoria.

• Both eyes are affected by latent squint e.g if an esophoria is found in the R, an esophoria will also be found in the L

Esophoria

• If the eye moves out as it is uncovered, it has been in a convergent position e.g. a latent convergence or esophoria.

• Each eye converges under cover in an esophoria. A recovery movement is seen as the cover is removed - the eye moves out from its convergent position to regain binocular vision.

Exophoria
Figure 5 – 9 positions of gaze

Muscles act in pairs in either eye and co-contract to make a movement.

Clinical testing of CN III, IV, & VI

- eyeball fully adducted to test superior and inferior oblique
- does not matter whether you are testing the movements of the right or the left eye
- eyeball fully abducted to test superior and inferior rectus
• Increased lens accommodation: the ciliary muscle contracts ⇒ causing zonules of Zinn to become slack ⇒ lens becomes more convex (convergent) ⇒ shortening of focal length (power is inversely proportional to focal length) ⇒ increase of the lens refractive power (positive dioptres). This is important for focussing on near objects.

• Convergence of the visual axes (eyes converge)

• Constriction of the pupils (the pupil constricts in order to prevent diverging light rays from hitting the periphery of the retina and resulting in a blurred image)

NB: The accommodation reflex is the opposite of the above for focussing on distant objects

Response to near

• Test with large target (e.g. fingertip) bringing it towards the patient

• Near response = pupil constriction and convergence (eye vergence)

• MUST ASK PT IF THEY ARE STILL ABLE TO FOCUS ON NEAR TARGET

A series of sites for lesions

These will be identified on diagrams and students will predict what effect such a lesion would have on the pupil reflexes of both eyes:

• Optic nerve (nervus opticus) ⇒ Complete afferent pupillary defect or RAPD (partial afferent defect)
  ➢ Marked defect ⇒ shining the light in the affected eye will result in no constriction of either pupil e.g. a severe defect in the left retina/CN II ⇒ shining the light in the left eye will result in no constriction of either pupil. Shining the light in the right eye will result in constriction of both pupils. This is due to damage to the left afferent pathway.
  ➢ Mild ⇒ shining the light in the affected eye will result in a slow constriction of both pupils, but then a gradual dilation of both pupils.
  ➢ In a normal swinging light test (i.e. there is no RAPD) the pupils of both eyes constrict equally regardless of which eye is stimulated by the light.
  ➢ In an abnormal swinging-light test (i.e. there is a RAPD) there is less pupil constriction in the eye with the retinal or optic nerve disease. A Marcus Gunn pupil (RAPD) may be seen in optic neuritis secondary to MS (as well as many other disorders). The affected pupil will appear to dilate on shining the light into it.

• Central (Argyll Robertson) ⇒ Argyll Robertson pupils (“AR pupils”) are bilateral small pupils that reduce in size when the patient focuses on a near object (e.g. the pupils accommodate),
but do not constrict when exposed to bright light. **They are a highly specific sign of neurosyphilis (can rarely occur in DM).** In general, pupils that “**accommodate but do not react**” are said to show light-near dissociation.

- Third nerve palsy => Pupil dilated (mydriasis – if compression lesion => surgical CN III palsy) and no response to light: direct or consensual from other eye (as parasympathetic innervation is dysfunctional => constrictor pupillae of iris is not functional). **If painful with mydriasis then suspect a posterior communicating aneurysm (emergency).** There will also be other signs such as loss of eye movements and ptosis (due to decreased innervations of levator superioris palpebrae muscle).

NB: Oculosympathetic palsy (Horner’s Syndrome) and causes of Horner’s Syndrome (congenital, carotid aneurysm, lesions of the neck, eg trauma or surgery, brain stem vascular disease or MS, Pancoast tumour of the lung, etc.) is characterised by ptosis (due to decreased innervations of superior tarsal muscle), miosis (small pupils), anhidrosis and other ANS effects. **PUPIL REFLEX UNAFFECTED IN HORNERS.**

**Distinguishing between Horner’s syndrome and CN III dysfunction**

**NORMAL**

**LEFT HORNER’S SYNDROME**
- Moderate ptosis
- Small pupil
- No problems with eye movements

**LEFT 3rd NERVE PALSY**
- Severe ptosis
- Large pupil
- Down/out gaze
Glaucoma Evaluation Tutorial

- Glaucoma is a group of conditions defined by a progressive optic neuropathy with accompanying visual field changes (e.g. Arcuate scotoms and loss of peripheral vision).
- Raised intra-ocular pressure (IOP) is classified as a risk factor but is not part of the definition.

Cup to disc ratio (CDR)

- CDR = the proportion of the disc that is occupied by the cup. This notation is commonly used in glaucoma clinics to classify discs and you will see in the casenotes CDR 0.4 or CDR 0.6. But opinions vary as to what CDR constitutes a glaucomatous disc some say 0.5 and some say 0.8.
- The CDR notation is also used to chart progression, an increase from 0.3 to 0.7 for example, being a sign of progression. However this technique of evaluating the optic disc has been shown to be inaccurate and highly variable in the way that it is recorded. Studies have shown that clinicians vary widely in the value they give a CDR when compared to each other and when retested themselves.

Glaucomatous changes

Optic disc appearance, even in a normal population, is not the same. Early glaucomatous changes are subtle and may appear similar to normal optic discs. What we rely on when evaluating a disc for evidence of glaucoma or progression in a glaucoma are specific patterns of change.

Even before glaucoma reveals its first symptoms, the ophthalmologist is able to determine the morphological changes of the optic disc (damaged optical nerve (which is called the “optic disc”) with a simple fundoscopy examination.

The ophthalmologist will assess for:

- Thinning of the nerve fiber layers (neuroretinal rim) e.g. optic atrophy (pallor of the disc) and increased CDR (enlargement of the cup)
- Increased normal cupping of the optic disc e.g. increased cup to disc ratio (due to pressure related damage of the nerve fibers of neuroretinal rim)
- Optic disc pallor (optic disc atrophy)

Optic nerve cupping

- The optic nerve carries impulses for vision from the retina in the eye to the brain. It is composed of millions of retinal nerve fibers that bundle together and exit to the brain.
• Visual acuity is recorded as the distance from the chart (numerator) over the number of the lowest line read (denominator)

• Record the lowest line the patient was able to read (e.g. 6/6 which is equivalent to 20/20 – patient can read at 6 metres what a healthy person could read at 6 metres)

• 6/6= normal vision; 6/9 => patient can read at 6 metres what a normal individual could read at 9 metres) => diminished acuity; 6/3 => patient can read at 6 metres what a normal person could only read at 3 metres => better than normal acuity

• If patient reads the 6/6 line, but gets 2 letters incorrect, you would record as 6/6 (-2)

• If patient gets more than 2 letters wrong, then the previous line should be recorded as their acuity

• You can have the patient read through a pinhole to see if this improves vision

• If vision is improved with a pinhole, it suggests there is a refractive element to their poor vision

• Repeat above with the other eye

• Can also use Ishihara test to assess for colour visual perception

2. Fine print reading

• Ask patient to cover one eye

• Ask patient to read a page of small print in a book/newspaper

• Repeat with the other eye

• If they normally wear glasses for reading, ensure these are worn for the assessment

Visual Fields

• Sit directly opposite the patient, at a distance of around 1 metre

• Ask patient to cover one eye with their hand

• If the patient covers their right eye, you should cover your left eye (just mirror the patient)

• Ask patient to focus on your face & not move their head or eyes during the assessment, you should do the same and focus your gaze on the patients face

• Ask the patient to tell you when they can see your fingertip wiggling

• Position your fingertip at the border of one of the quadrants of your visual field
To complete the examination

- Thank patient
- Wash hands
- Summarise findings
- Mention further investigations you would like to perform e.g. Ishihara colour testing plates, retinal photography, full cranial nerve examination, etc

ANATOMY REVISION
• Not uncommon for subconjunctival haemorrhage to occur

• Causes include eye trauma, coagulation disorder, head injury, whooping cough (or other extreme sneezing or coughing), child birth, and severe hypertension

• Although its appearance may be alarming, in general a subconjunctival hemorrhage is a painless and harmless condition; however, it may be associated with high blood pressure, trauma to the eye, or a base of skull fracture

• Looks alarming => but it is not => reassure patient (check patient blood pressure, as patient likes to see something been doing, however clinically this usually has no significance)

• Blood on surface of eye remains oxygenated as blood can pass through surface of conjunctiva => remains red for long time and we do not get yellowish/brownish changes which occur with bruises elsewhere

2. Patient presents with a suspected squint => what tests should we carry out? What is the type of squint?

• **Hirschberg test** is performed by shining a light in the person's eyes and observing where the light reflects off corneas (comparing right and left). In a person with normal ocular alignment the light reflex lies **slightly nasal from the center of the cornea**. When doing the test, the light reflexes of both eyes are compared, and will be **symmetrical in an individual with normal fixation**. For an abnormal result, based on where the light lands on the cornea, the examiner can detect if there is an exotropia (abnormal eye is turned out), esotropia (abnormal eye is turned in), hypertropia (abnormal eye higher than the normal one) or hypotropia (abnormal eye is lower than the normal one).

  ➢ In exotropia the light lands on the medial aspect of the cornea.

  ➢ In esotropia the light lands on the lateral aspect of the cornea.

  ➢ In hypertropia the light lands on the inferior aspect of the cornea. In hypotropia the light lands on the superior aspect of the cornea.

  ➢ A cover test can tell you the extent of the eso/exotropia

• The example above shows a manifest **right convergent (esotropia) squint**. It is a **manifest squint** as we can see it. We can say it convergent (esotropia) as the light reflex is on lateral aspect of cornea. **Could be due to right CN VI palsy.**
• The coloured clinical photograph above clearly shows hyphema

• A hyphema is a pooling or collection of blood inside the anterior chamber of the eye (the space between the cornea and the iris).

• Even a small hyphema can be a sign of major intraocular trauma with associated damage to vascular and other intraocular structures

• **Emergency => contact ophthalmologist immediately**

• Eye is closed system so anything that increases volume (e.g. bleed) => increase pressure => pain and damage can occur

7. When describing the optic disc use the 3Cs:
   • Colour
   • Contour
   • Cup
   • + shape and size
   • Venous pulsation
   • Drusen
   • Neovascularisation

Can also comment on blood vessels (always come out at nasal side)

8. What is the name of this lesion on the cornea?