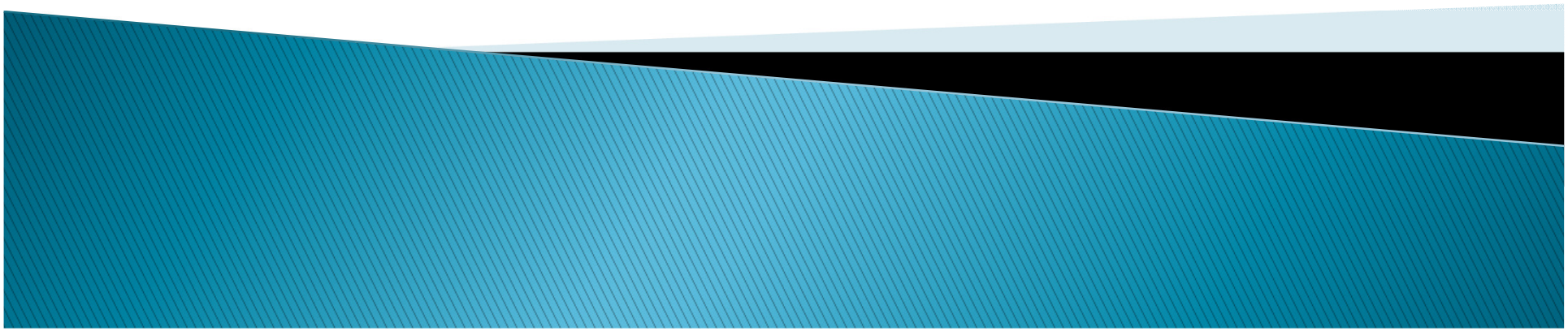
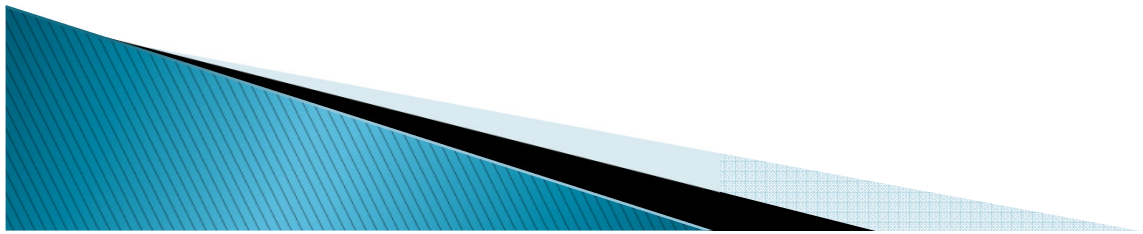
Eye Emergencies

August 2012



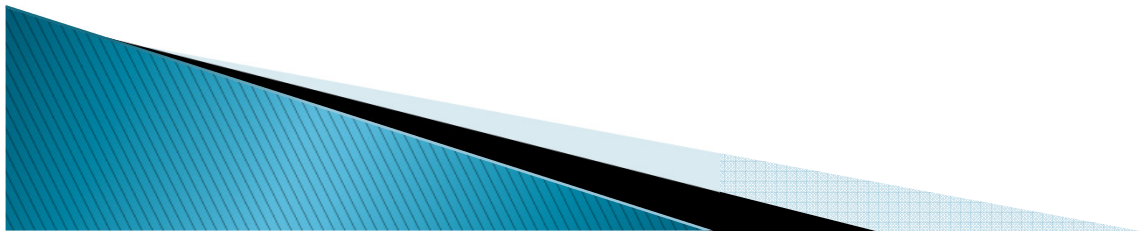
The voice at the other end…

- Regardless of the clinical scenario, patient management requires some form of triaging.
- I would not expect a referring doctor or optometrist to triage the case, however would hope that:
  - If sufficiently concerned or
  - Well-versed in what constituted an emergent case,
- Then, <u>not</u> to hesitate and contact the appropriate service



Triaging

- As you are aware, the more information is available to make an assessment of the situation
- then the more accurate a working diagnosis can be
- hence more appropriate triaging and management can occur

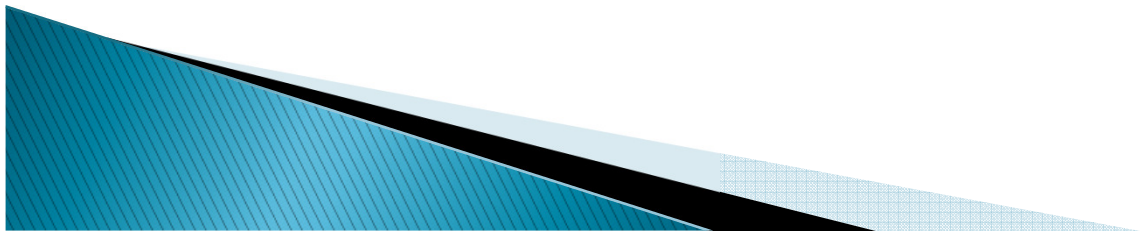


Eye Emergencies

Generally speaking, emergent situations in ophthalmology can be divided into (potentially):

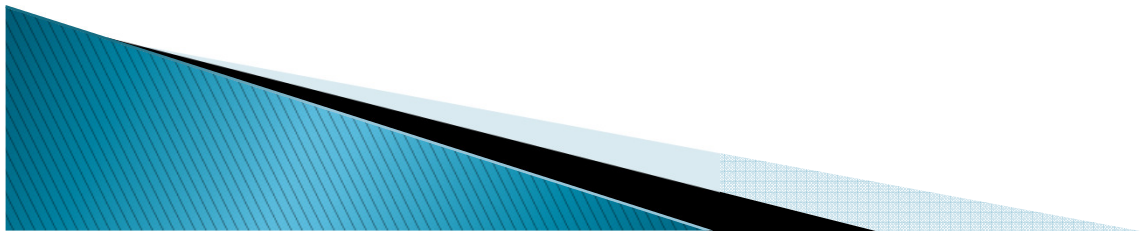
- Vision-threatening conditions or
- Life-threatening conditions.

The other relevant factor is that something can be done (or attempted) to improve the prognosis



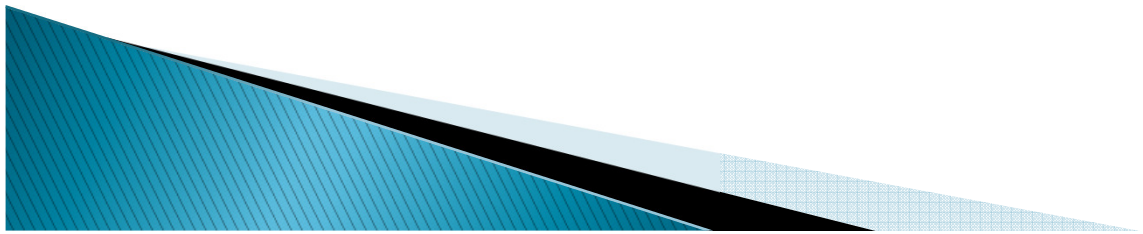
Trauma

- Classified into closed globe injuries and open globe injuries, both of which may have severe consequences.
- Generally, an open globe injury tends to be the more emergent of the 2 situations;
  - A “depressurised” eye is at risk of expulsive haemorrhage
  - An open wound increases the risk of infection (endophthalmitis)

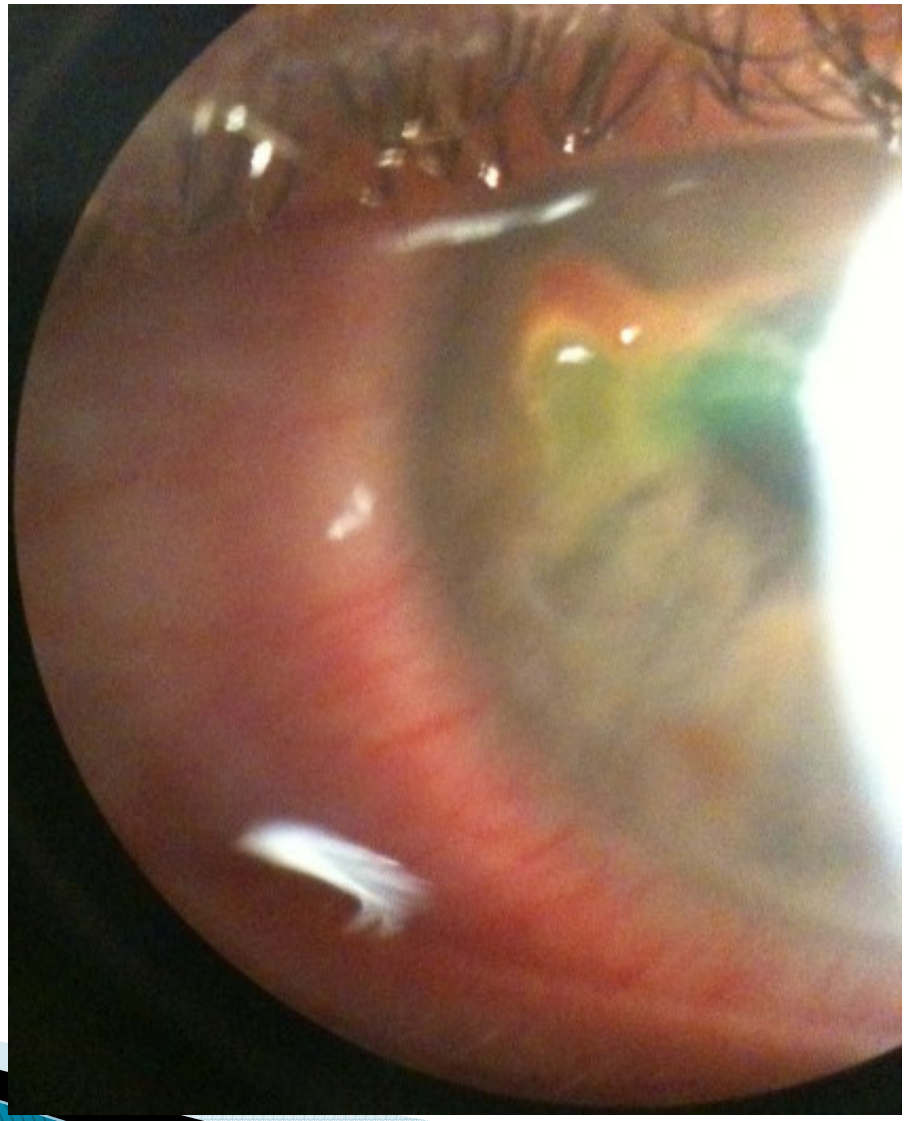


Open globe injuries

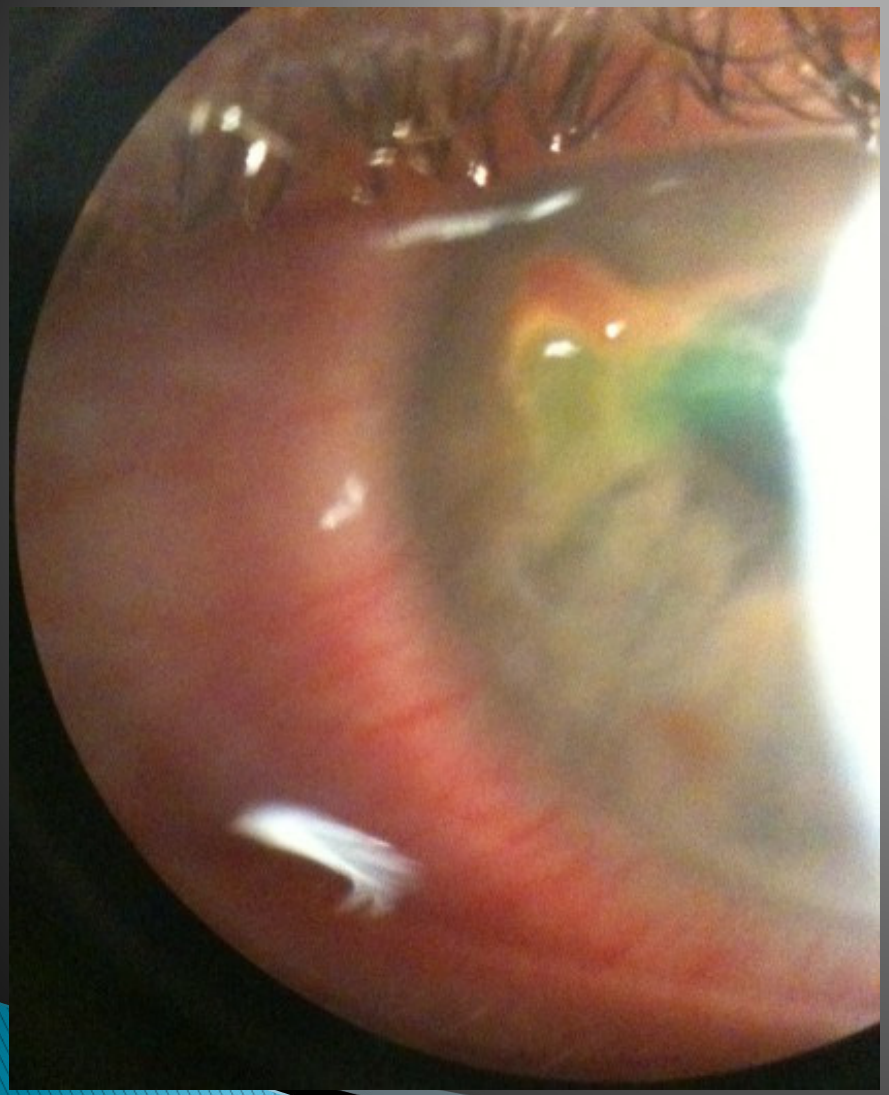
- Can be subdivided into penetrating and perforating eye injuries and globe rupture .
- A penetrating eye injury is defined as having one entry/exit, compared to a perforating injury that has an entry and exit wound.
- Globe ruptures are usually due to a massive blunt eye injury, and there are various points of weakness of the eye that are rupture points.



Recent PEI case

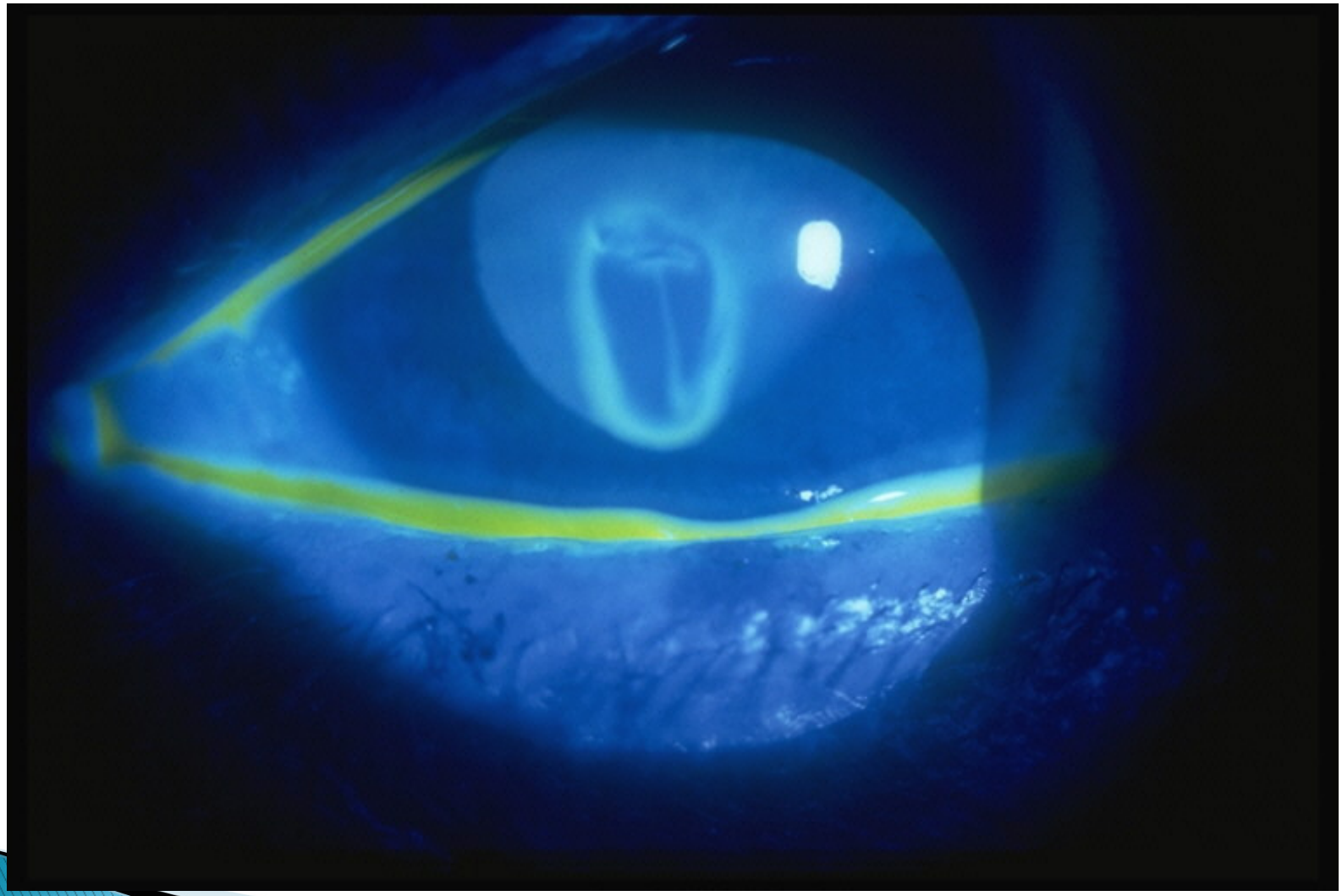


Critical features



- Variable decreased visual acuity
- Pupil peaking (in this case iris plugging wound)
- Shallow anterior chamber
- Seidel's test +ve

Positive Seidel's test



Immediate management

If possible, elicit mechanism of injury (to determine if there is likelihood of an intraocular foreign body)

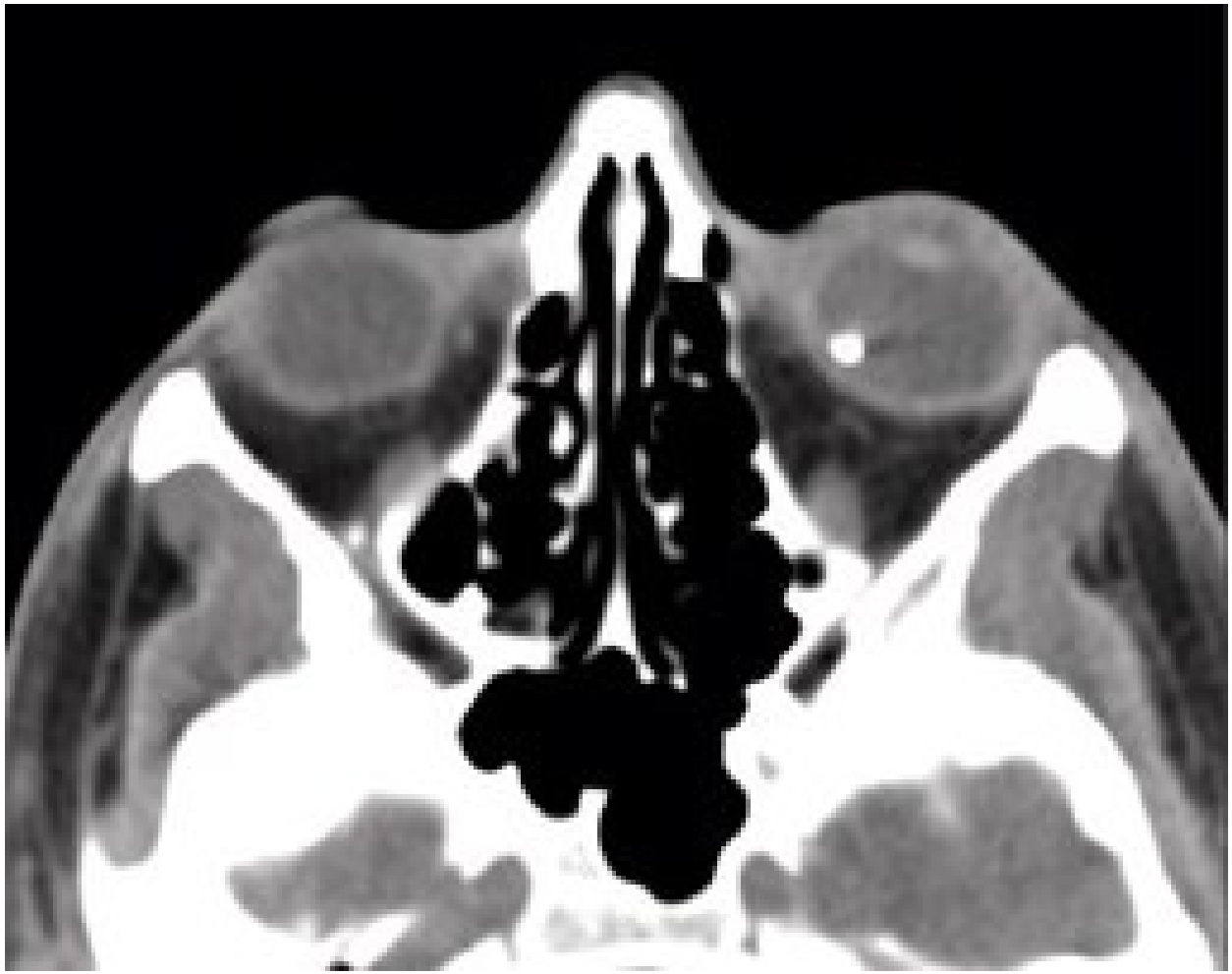
Protect the eye hence...

- Shield the eye: plastic shields available or make-your-own with card or Styrofoam cup

Refer for urgent repair so...

- Patient to be nil by mouth (specifically told and emphasised)
- Ophthalmologist informed

Intraocular foreign body (IOFB)



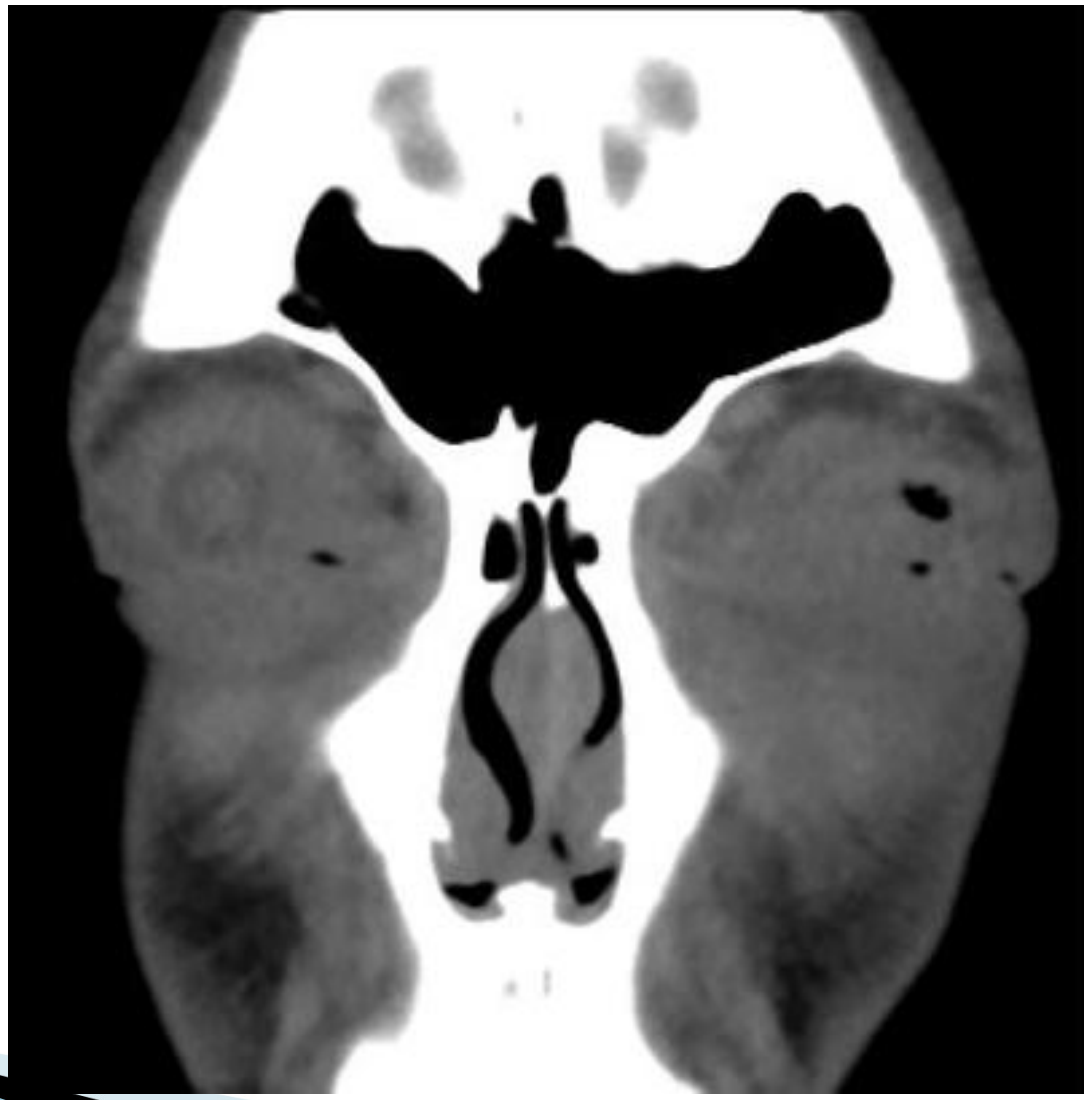
Globe rupture

Critical features:

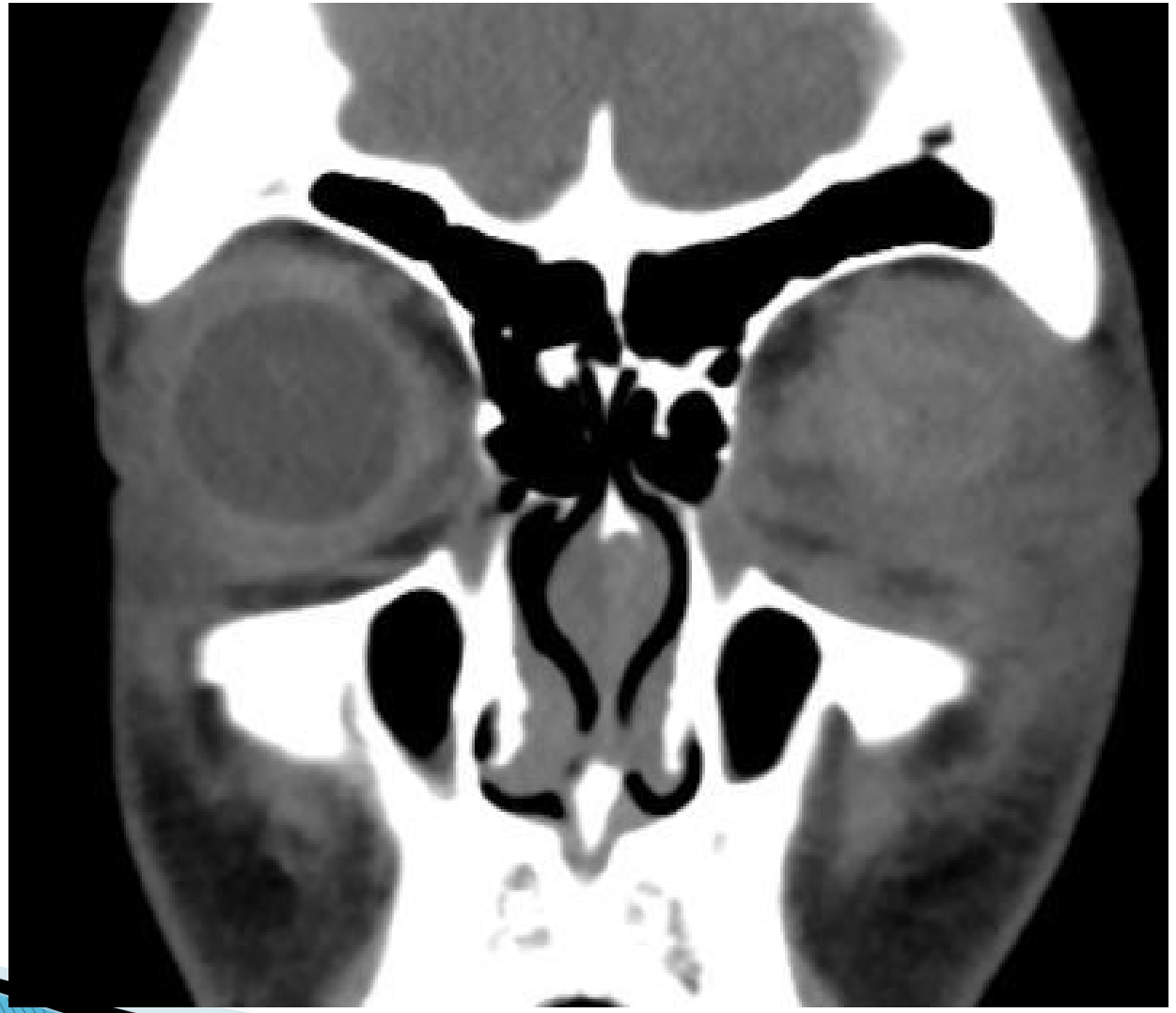
- History of (usually) extreme blunt force trauma
- Decreased visual acuity
- Hyphaema
- (Likely) Hypotony

- May be very difficult to determine especially when there is extensive soft tissue swelling/bruising (requiring imaging)

Recent globe rupture



Recent globe rupture

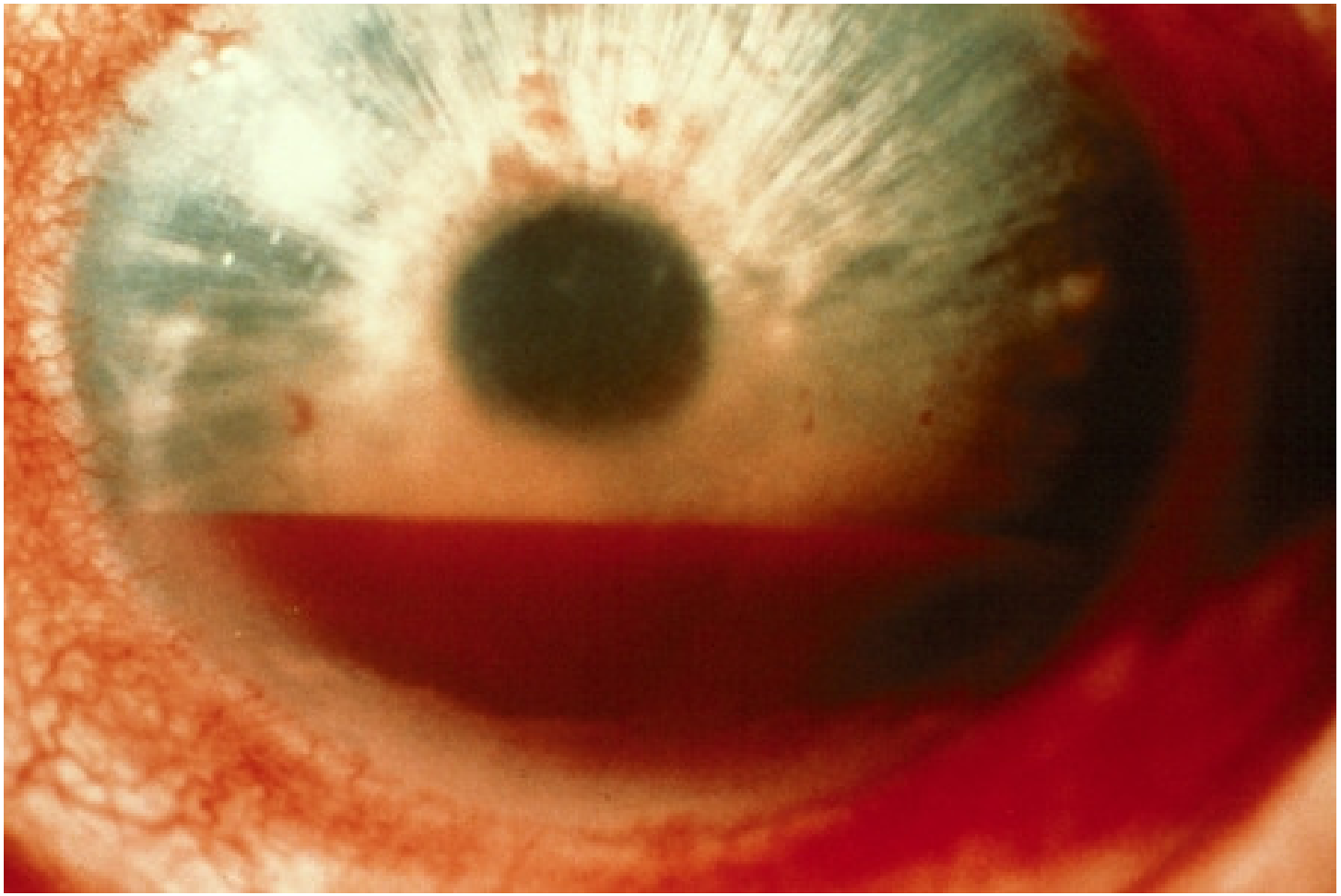


Closed globe injuries

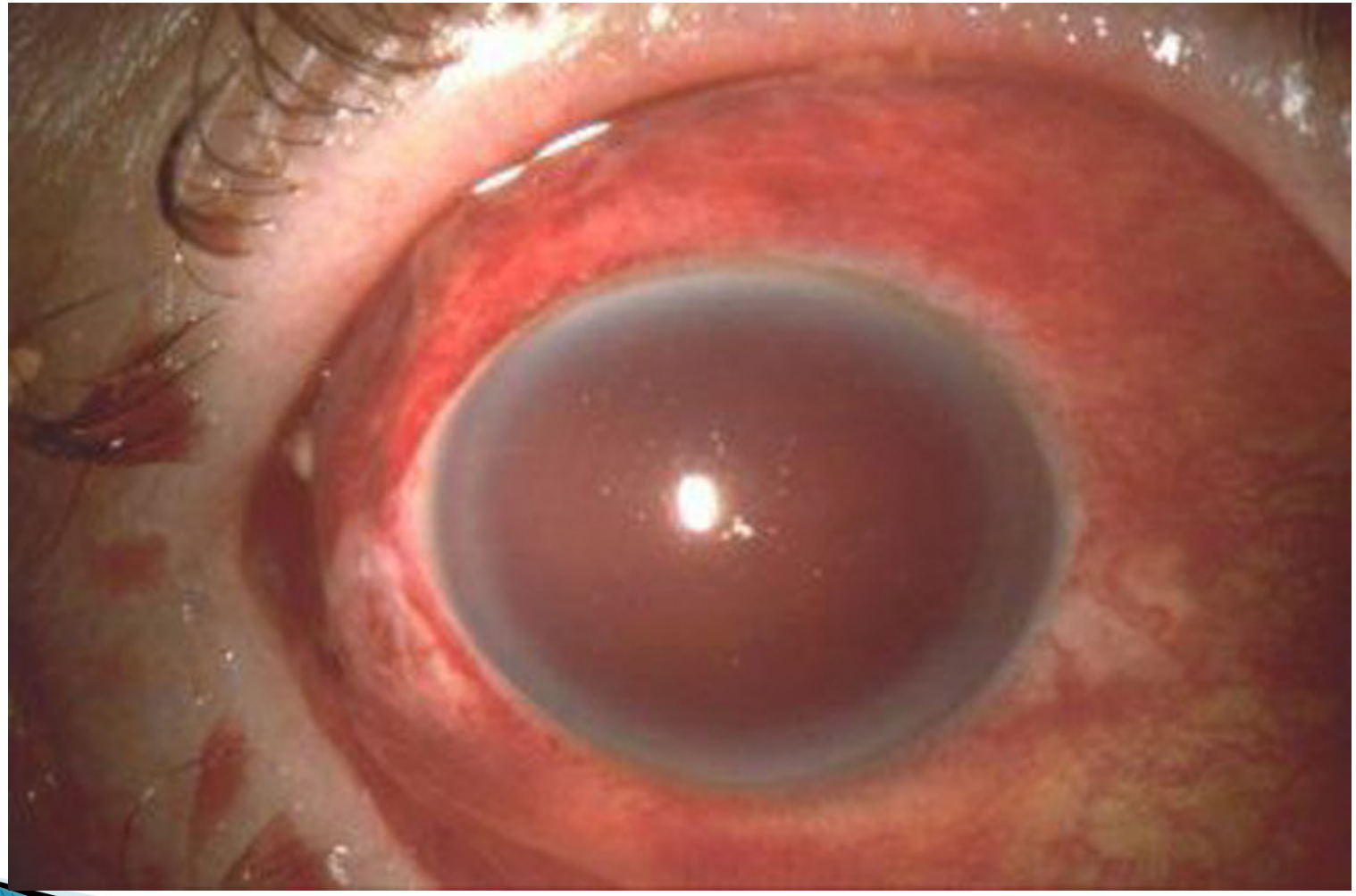
- Occurs following blunt force trauma;
  - Can range from mild to severe
- Critical features:
  - Variable decreased visual acuity (normal to light perception only)
  - Hyphaema; microhyphaema to “eight-ball”
  - Irregular pupil/Iridodialysis

- May have acute rise in IOP (RBCs block trabecular meshwork) and develop corneal blood-staining

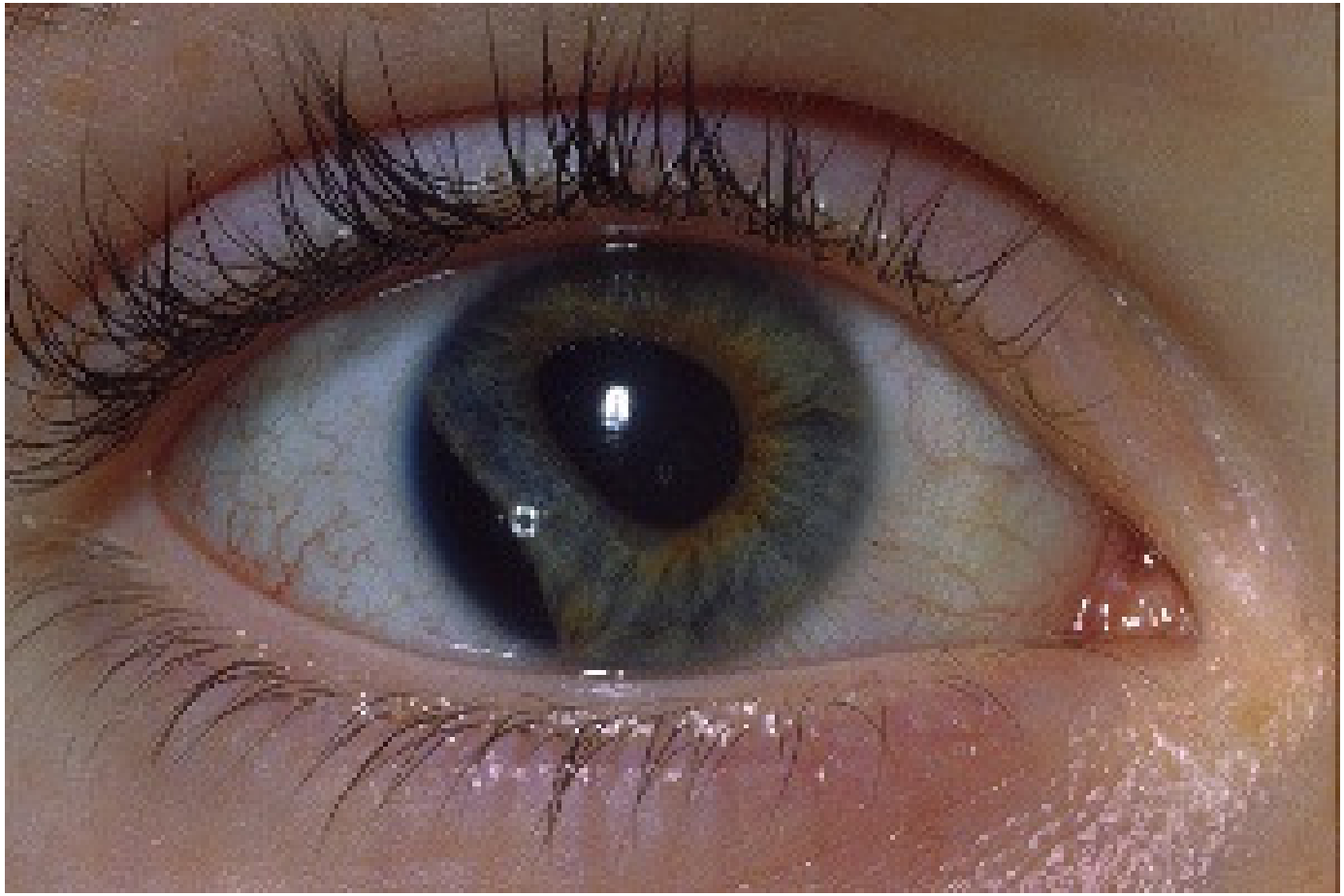
Hyphaema



Eight Ball Hyphaema



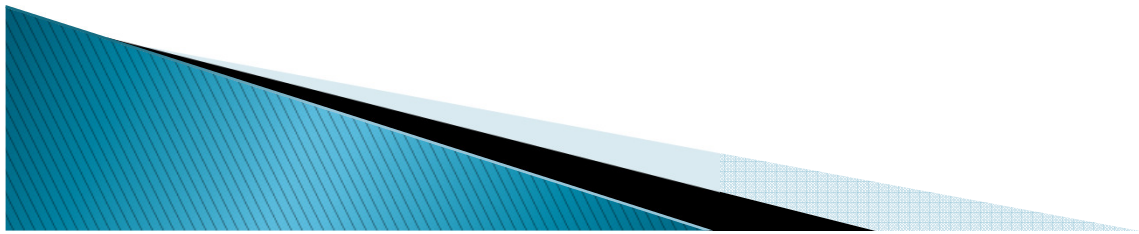
Iridodialysis and Irregular pupil



Chemical injuries

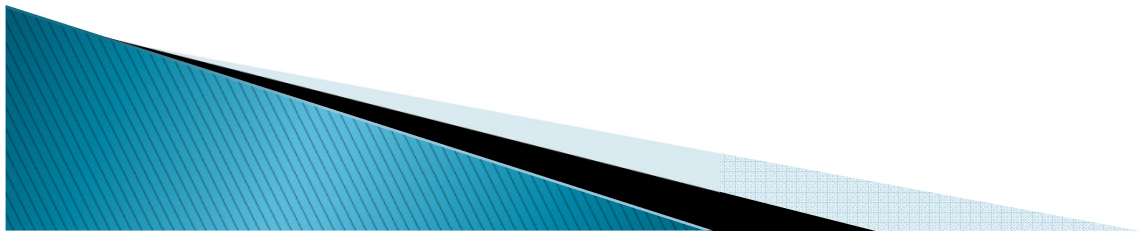
- Typically work-related and accidental
- Can range from mild to severe

- It is very important to try to determine the offending agent because...



Chemical injuries (cont.)

- Alkali injuries are worse than those caused by acidic solutions
- Acids denature proteins resulting in precipitation and coagulation, preventing further penetration (except for hydrofluoric acid)
- Alkalis saponify cell membranes resulting in deeper penetration and more extensive ocular injury

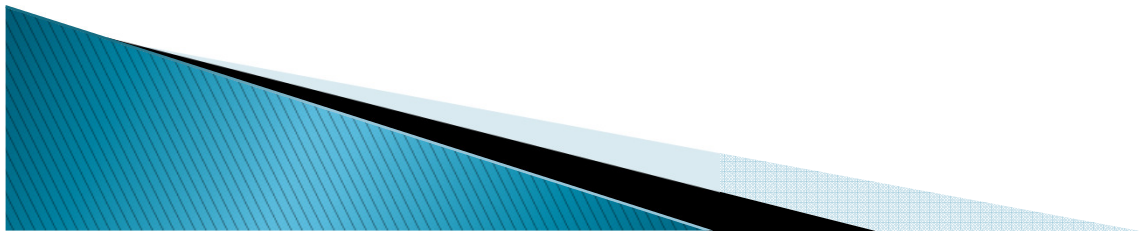


Critical features

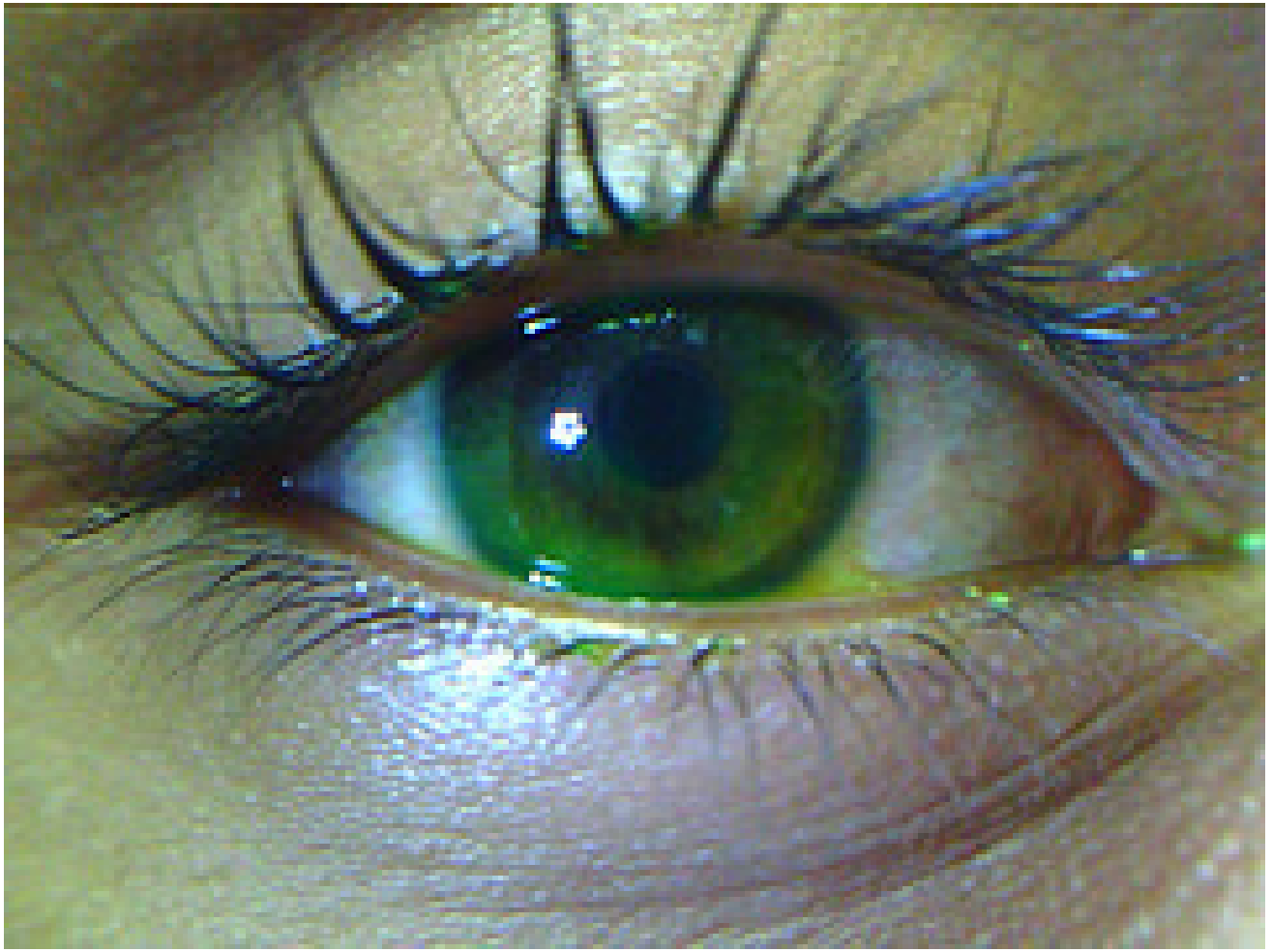
Variable visual acuity depending upon...

- Variable corneal opacification (usually more severe in alkali injuries)
- Corneal epithelial defect, can be extensive, usually inferior (due to Bell's phenomenon)

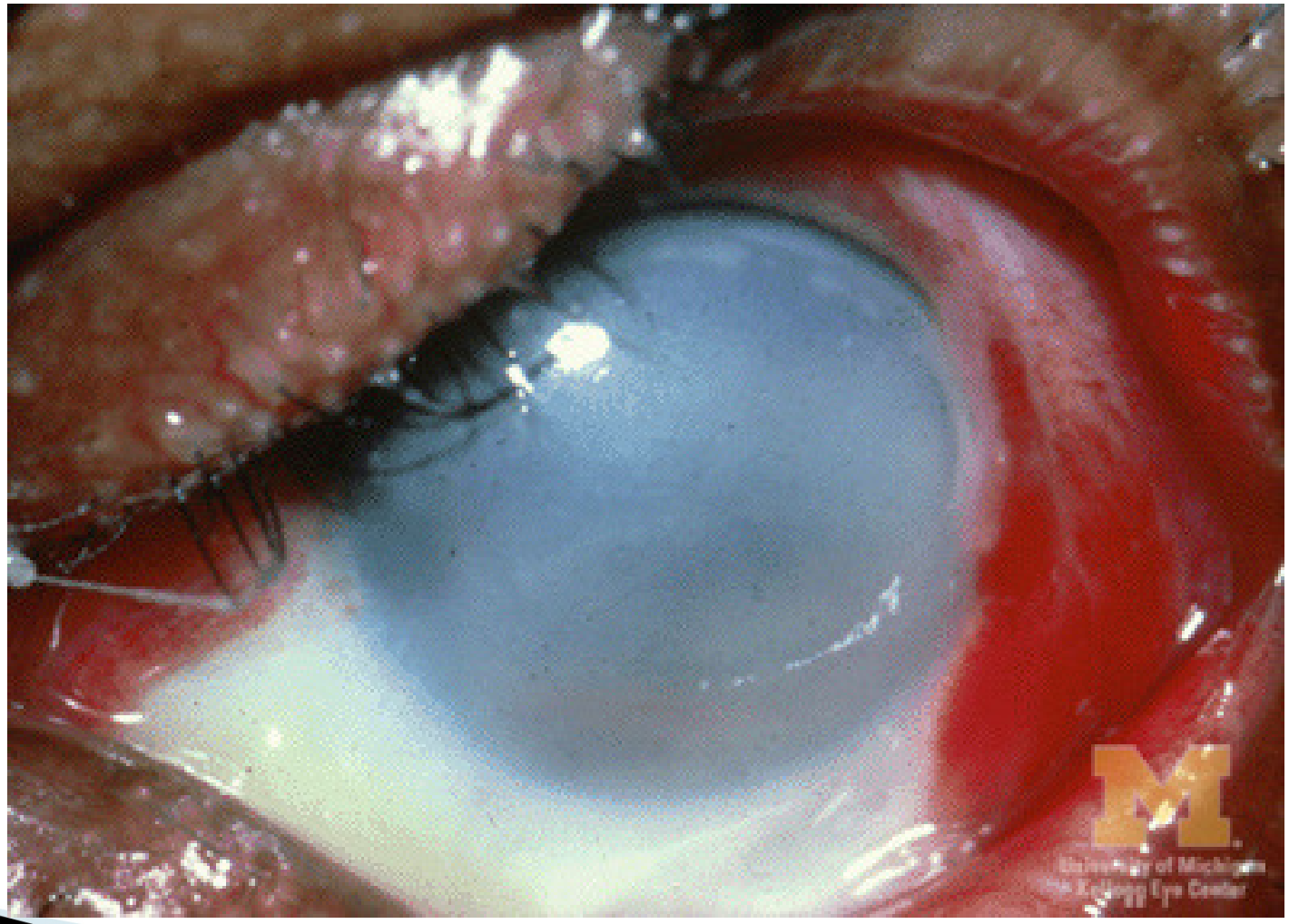
- A cloudy cornea and a (mostly) white eye is a worrying sign



Mild chemical injury



Severe alkali injury



Management

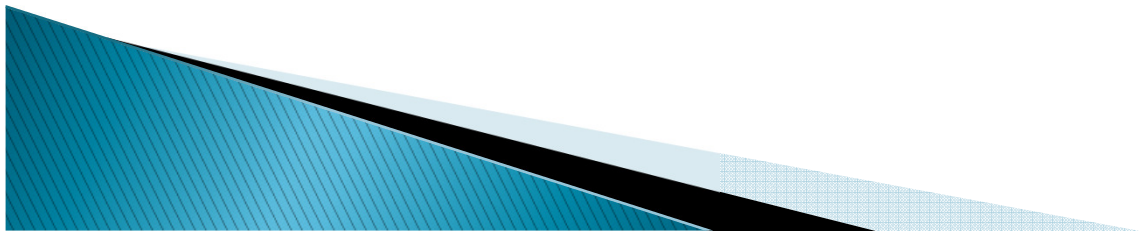
- Immediate (or as soon as possible) irrigation is the key:
  - Buffered solutions, sterile saline/water, clean water, ?other irrigating fluids
  - Continue until pH (if able to be measured) is equal to uninjured eye or normal (1981 study determined that the range is between 6.5 – 7.6 [mean 7.0]).
- Ophthalmic treatment consists of topical antibiotics/steroids/ sodium ascorbate and sodium citrate, oral doxycycline and vitamin C, managing IOP

Abelson MB, Udell IJ, Weston JH. Normal human tear pH by direct measurement. *Arch Ophthalmol*. 1981 Feb;99(2):301.

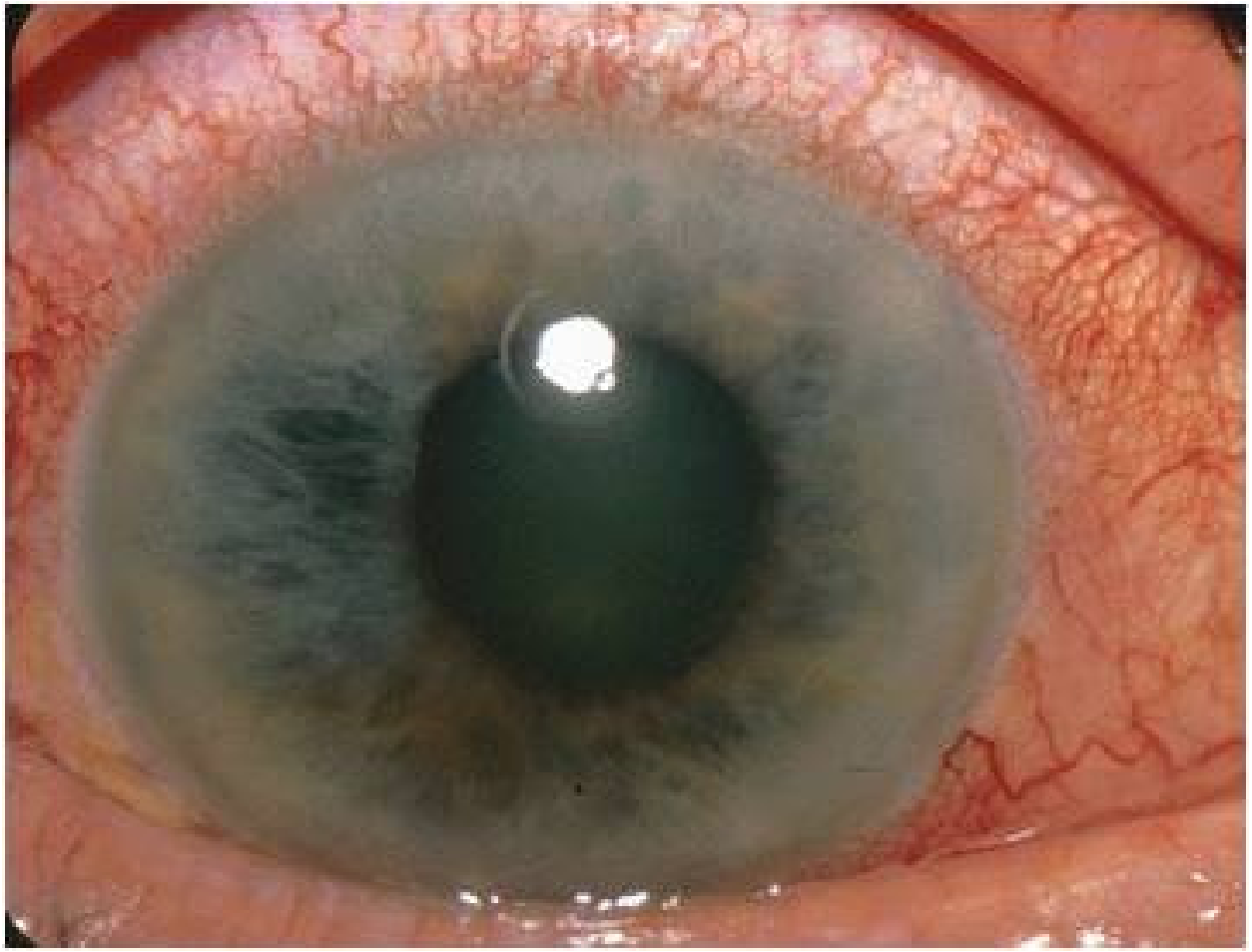
Acute angle closure glaucoma

Critical features:

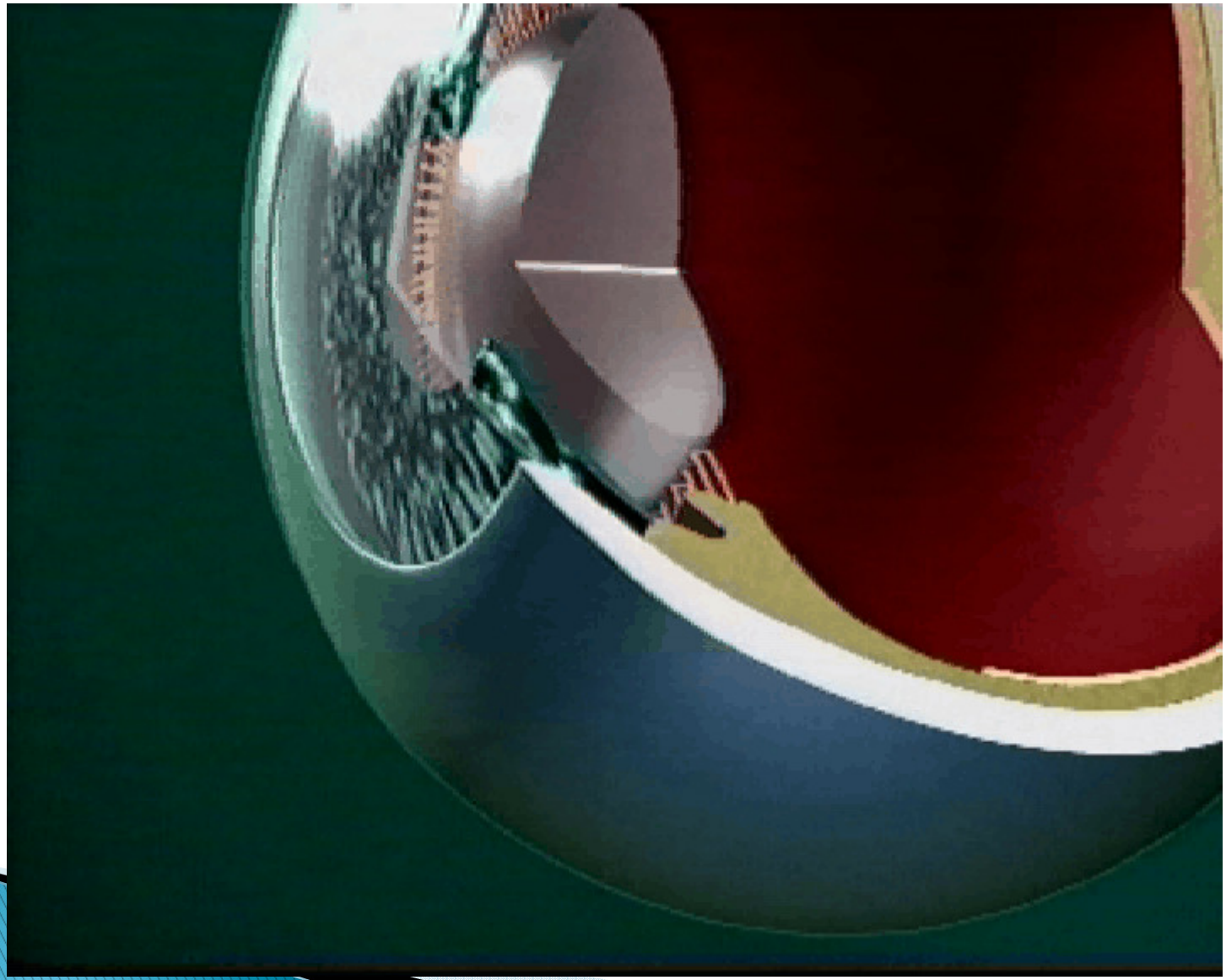
- Painful, red eye/s
- “Headache”, nausea, vomiting
- Cloudy cornea (oedema)
- Mid-dilated, unresponsive pupil
- RAPD (if unilateral)
- Shallow anterior chamber



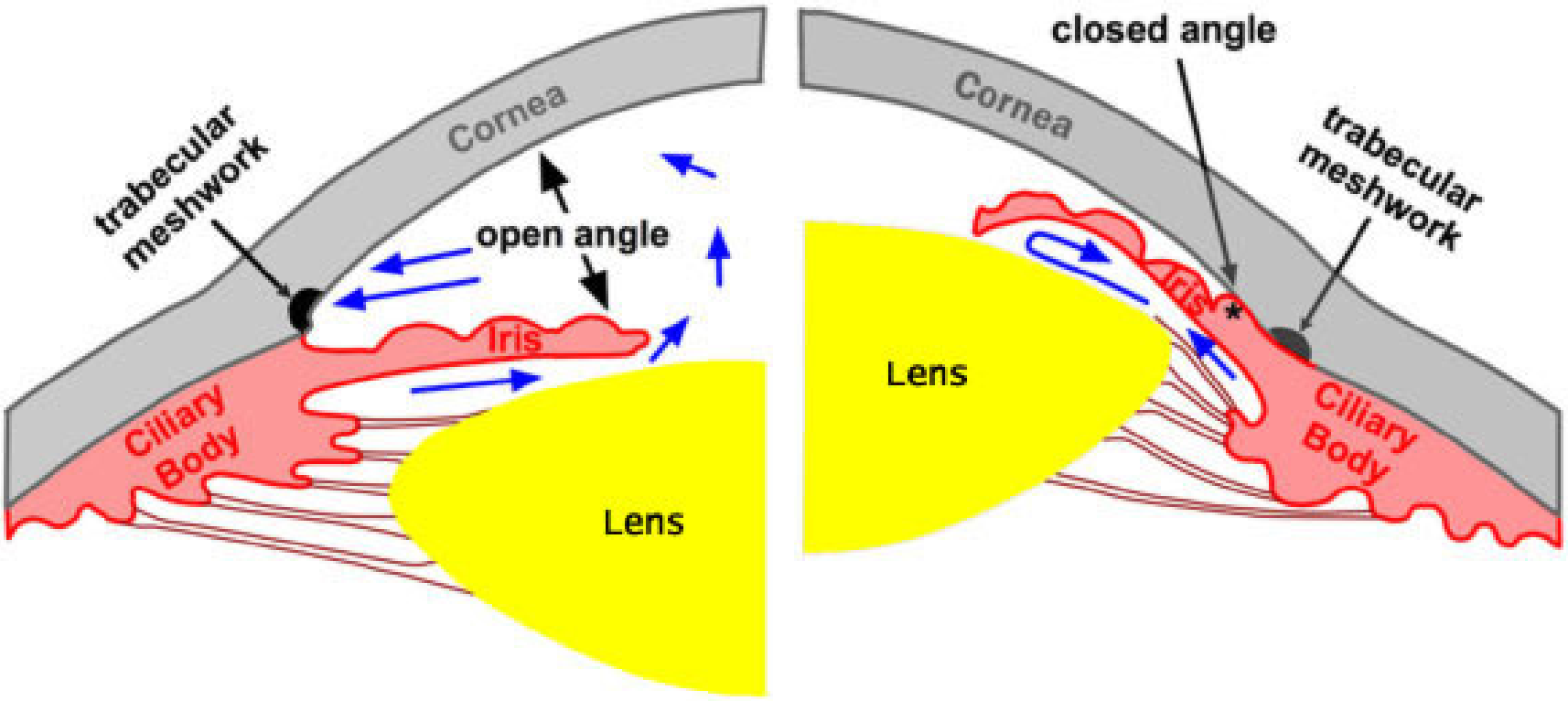
AACG



Pathophysiology



Pathophysiology (cont.)

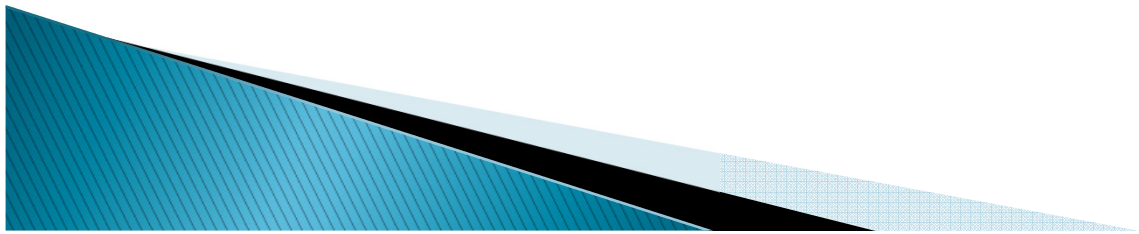


Management

Decrease the IOP:

- PO/IV Diamox (Acetazolamide) 250-500mg
- Topical Timolol/Iopidine/Prostaglandin analogue
- Topical Steroid
- Pilocarpine 2%

- Referral to ophthalmology (but hopefully well on the way to breaking the attack)

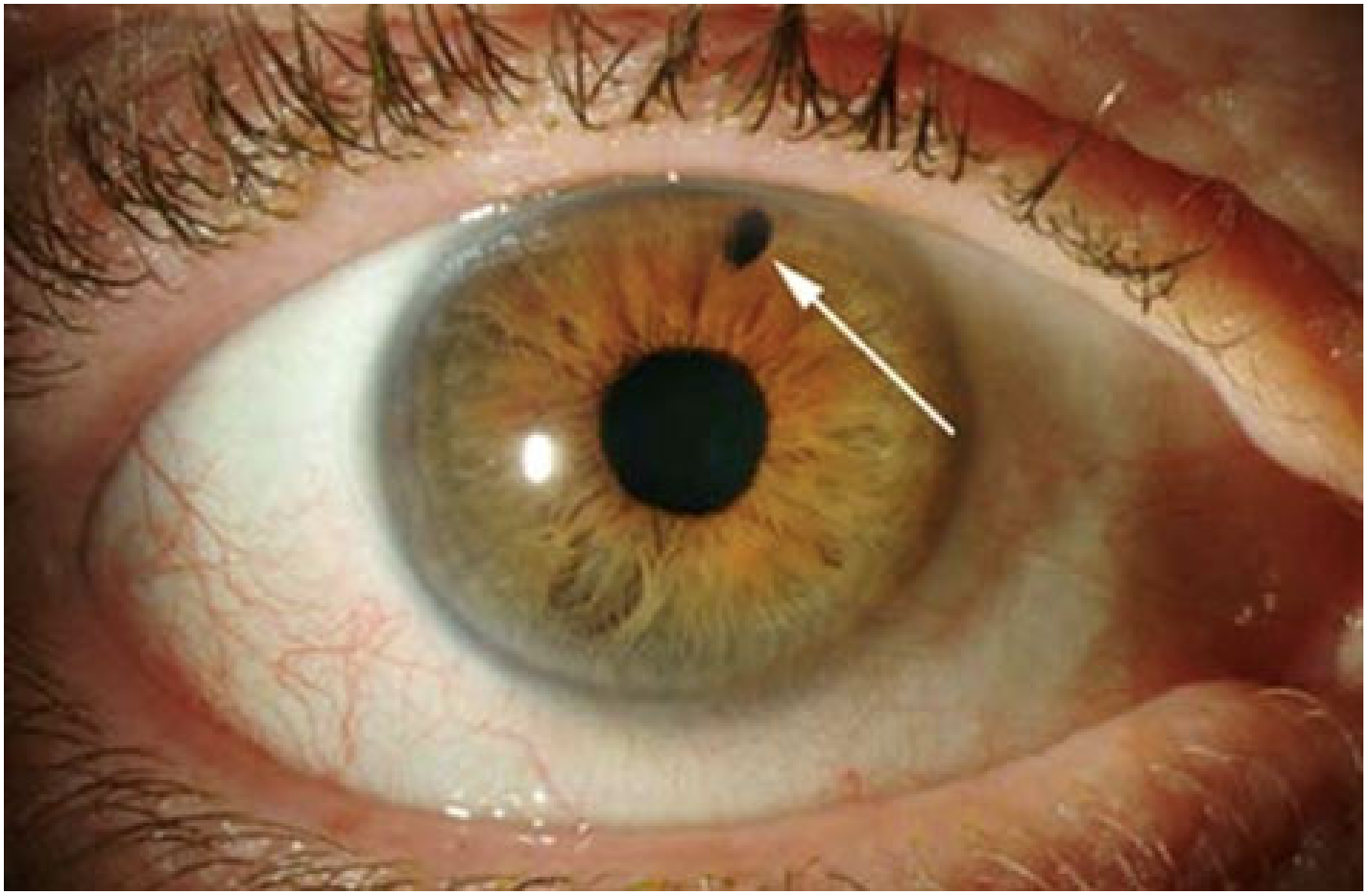


Management (cont.)

- Sometimes the previous treatment is insufficient and may require IV Mannitol (1-2g/kg infusion over 45 minutes)

- Definitive treatment attempts to reopen the angle, usually with (laser) iridotomy, which relieves iris bombe by establishing a new/shorter pathway of aqueous flow

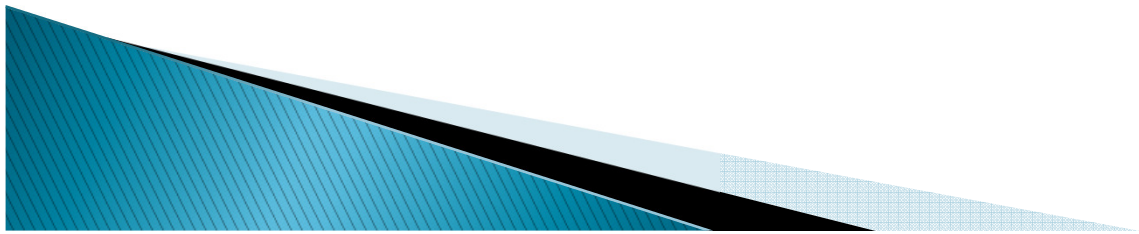
YAG Laser peripheral iridotomy



Should you dilate a patient?

A full ophthalmic examination should include a dilated fundus examination.

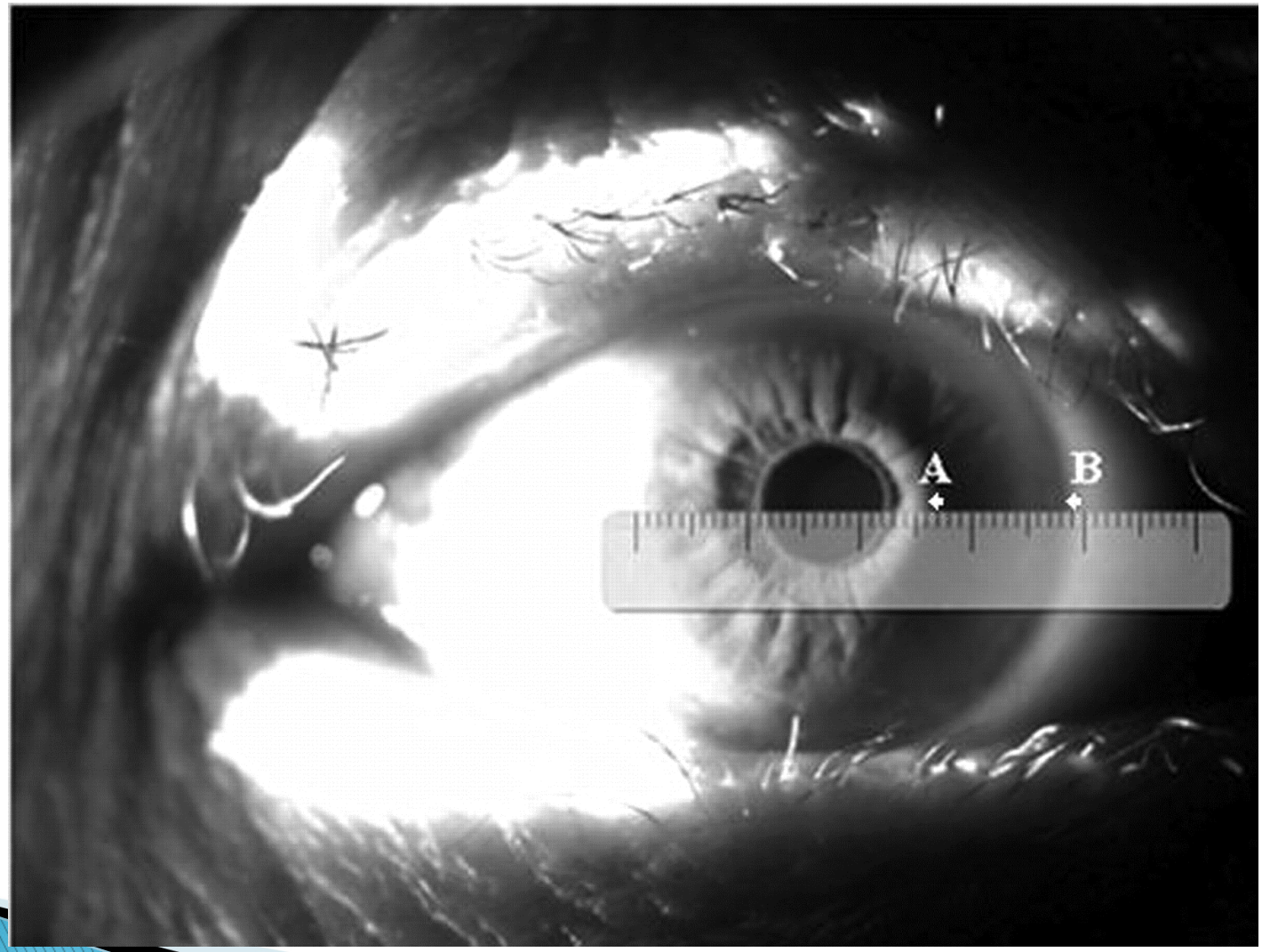
In general, I think, that causing acute angle closure when medical treatment (i.e. your care) is available is a better situation than a patient waiting 6 hours to 1 week+ before seeking attention for their acute angle closure.



Clues to possible narrow angles

- History:
  - ± Hypermetropia (long-sightedness)
  - Intermittent angle closure (episodes of blurred vision with haloes around lights)
- Examination:
  - Shallow anterior chamber with slit-lamp exam
  - “Eclipse” sign: light directed along the iris plane may demonstrate the forward displacement of the iris/pupil margin, throwing a shadow/“eclipse” on the opposite side

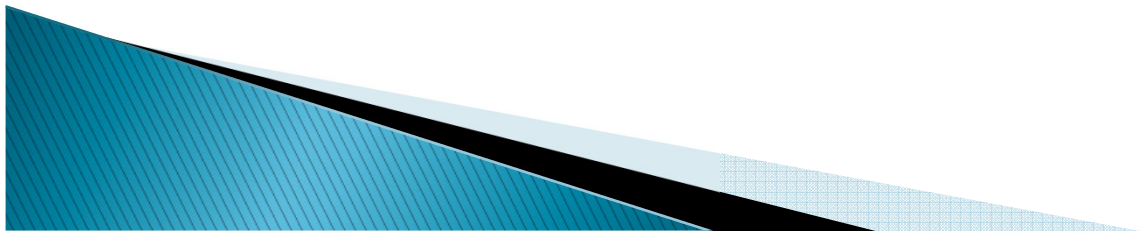
Eclipse sign



Flashes and Floaters

What do these symptoms represent?

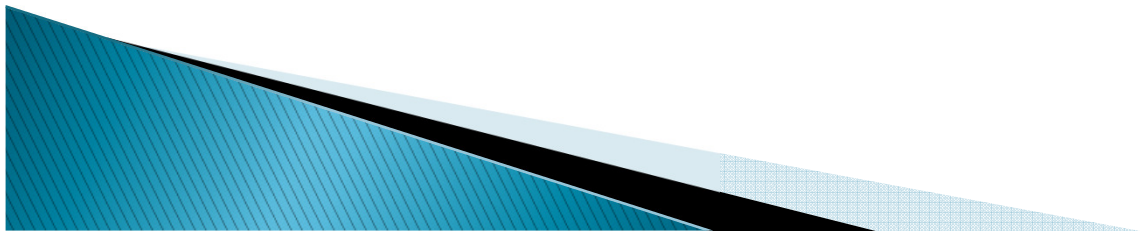
- To the patient;
  - something ranging from annoying to worrying
- To you;
  - something ranging from annoying to worrying (tending to the worrying side)
- To ophthalmologists:
  - something ranging from annoying to worrying (depending upon the history and your clinical findings)



What I have been asked to tell you by the Eye Registrars

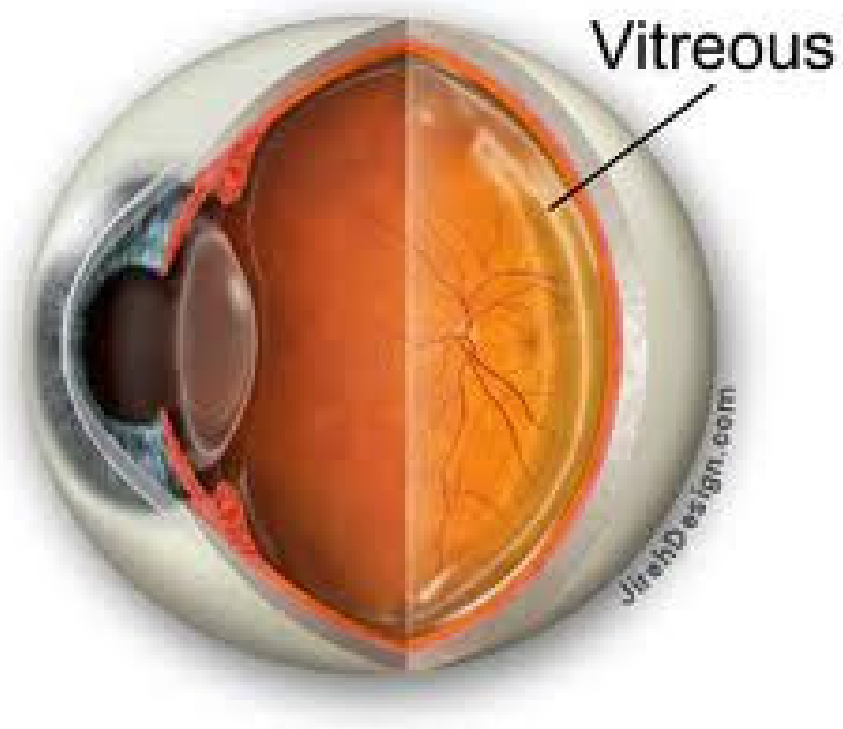
- Not all flashes and floaters = a retinal detachment

- What could flashes and floaters signify?
  - Retinal detachment
  - Vitreoretinal traction
  - Posterior vitreous detachment
  - Posterior uveitis/White dot syndromes



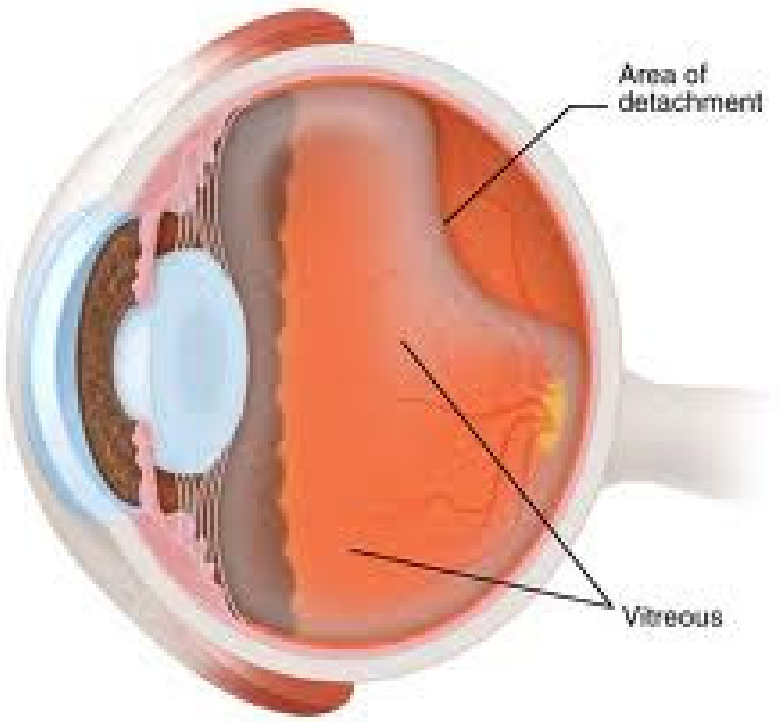
Aetiology of flashes and floaters

- The posterior segment is filled with vitreous; a jelly composed of ≈98% water, some collagen fibrils and some cells (hyalocytes, others)



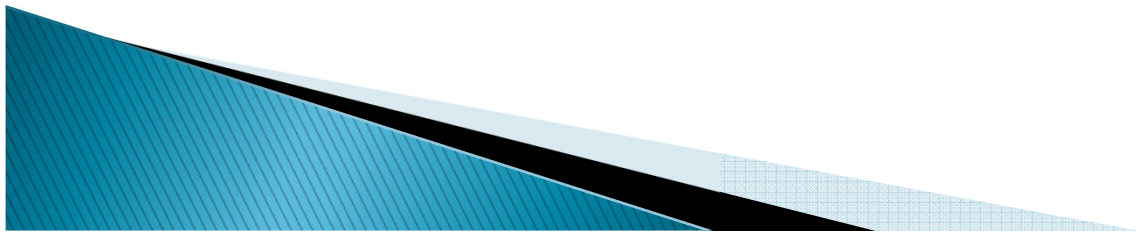
Aetiology of flashes and floaters (cont.)

- As we get older, the collagen framework degenerates, and the vitreous separates into water and collagen



Aetiology of flashes and floaters (cont.)

- The collapsed collagen network and any areas where the framework is slightly more solid (e.g. at the optic disc and macula) can be seen as floaters.
- Some people already have floaters (without being old); more common in myopes perhaps

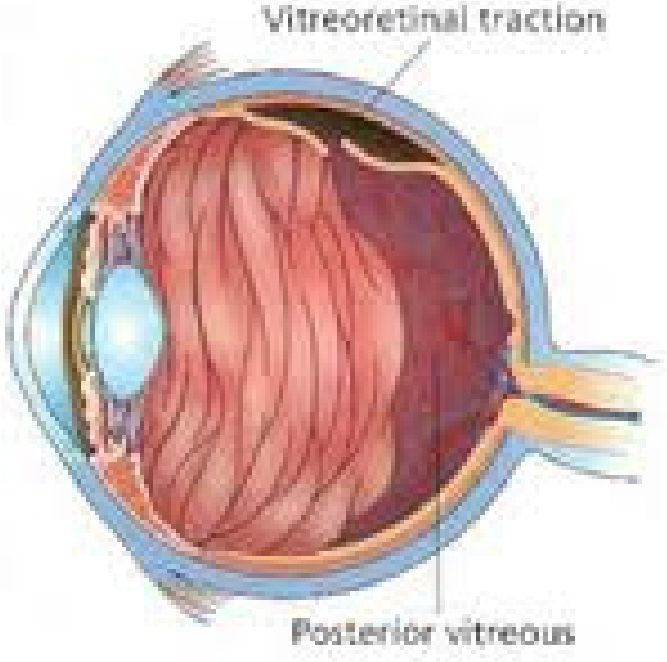


Aetiology of flashes and floaters (cont.)

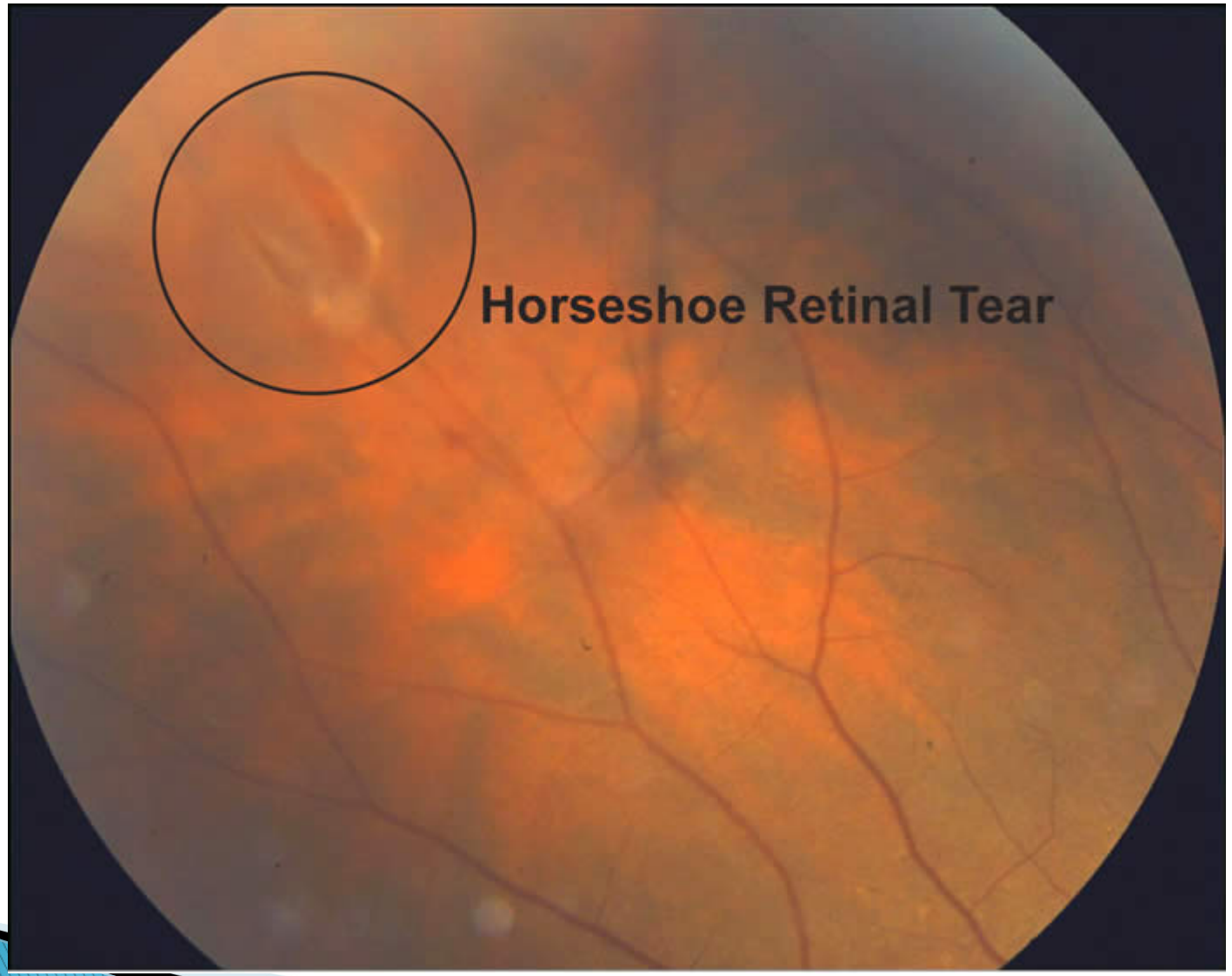
- Flashes occur due to traction of the collapsing/ed vitreous on the retina

Aetiology of flashes and floaters (cont.)

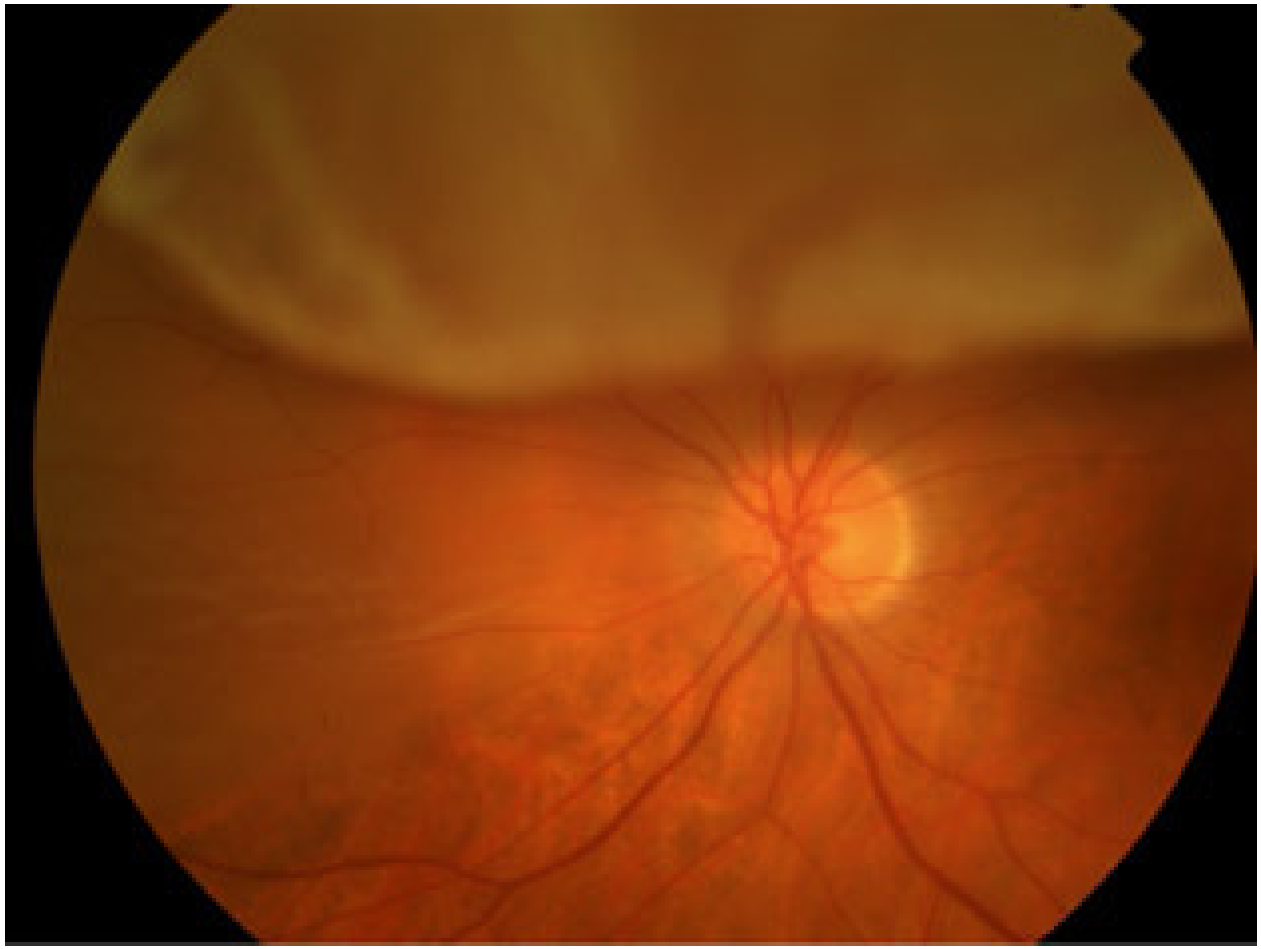
- The traction may tear the retina, which then may have vitreous fluid track underneath the retina...



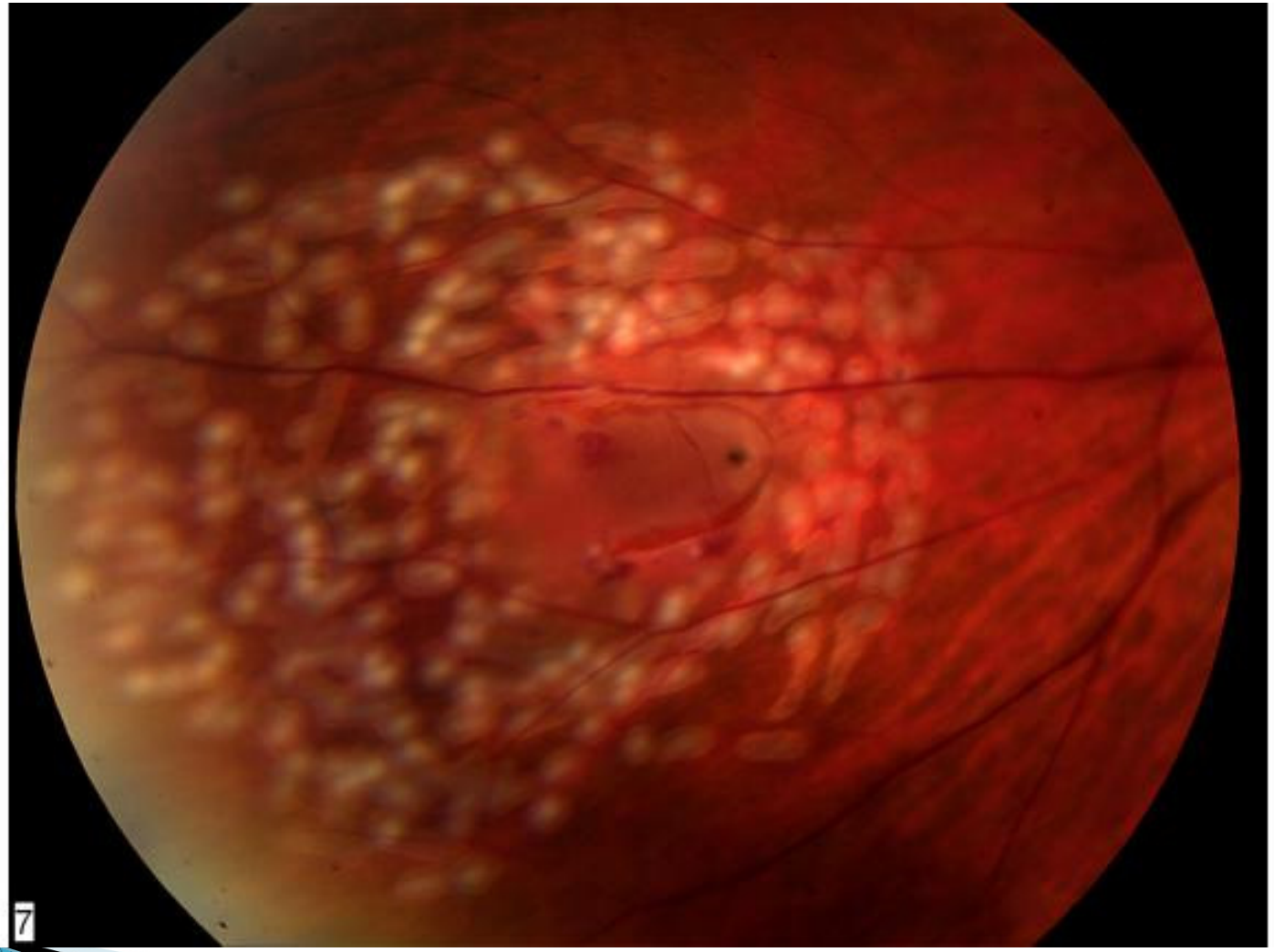
Retinal Tear



Retinal detachment



Unless we get to it first



Critical features

- Visual acuity may be normal to markedly ↓ed.
- Symptoms are key
- Duration of symptoms determines urgency

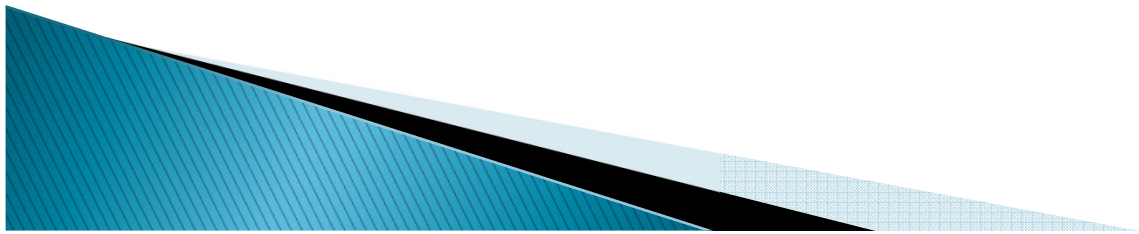
- A relative afferent pupillary defect and a visual field defect is more likely to suggest retinal detachment

- Be aware that once the macula is detached, the urgency is greatly reduced

Infective corneal ulcers

- Tend not to be an “emergency” although rapid assessment/treatment is important...
- Unless, the cornea has perforated (a definite emergency)

- Infective agents include:
  1. Viral (HSV)
  2. Bacterial
  3. Fungal
  4. Acanthamoeba

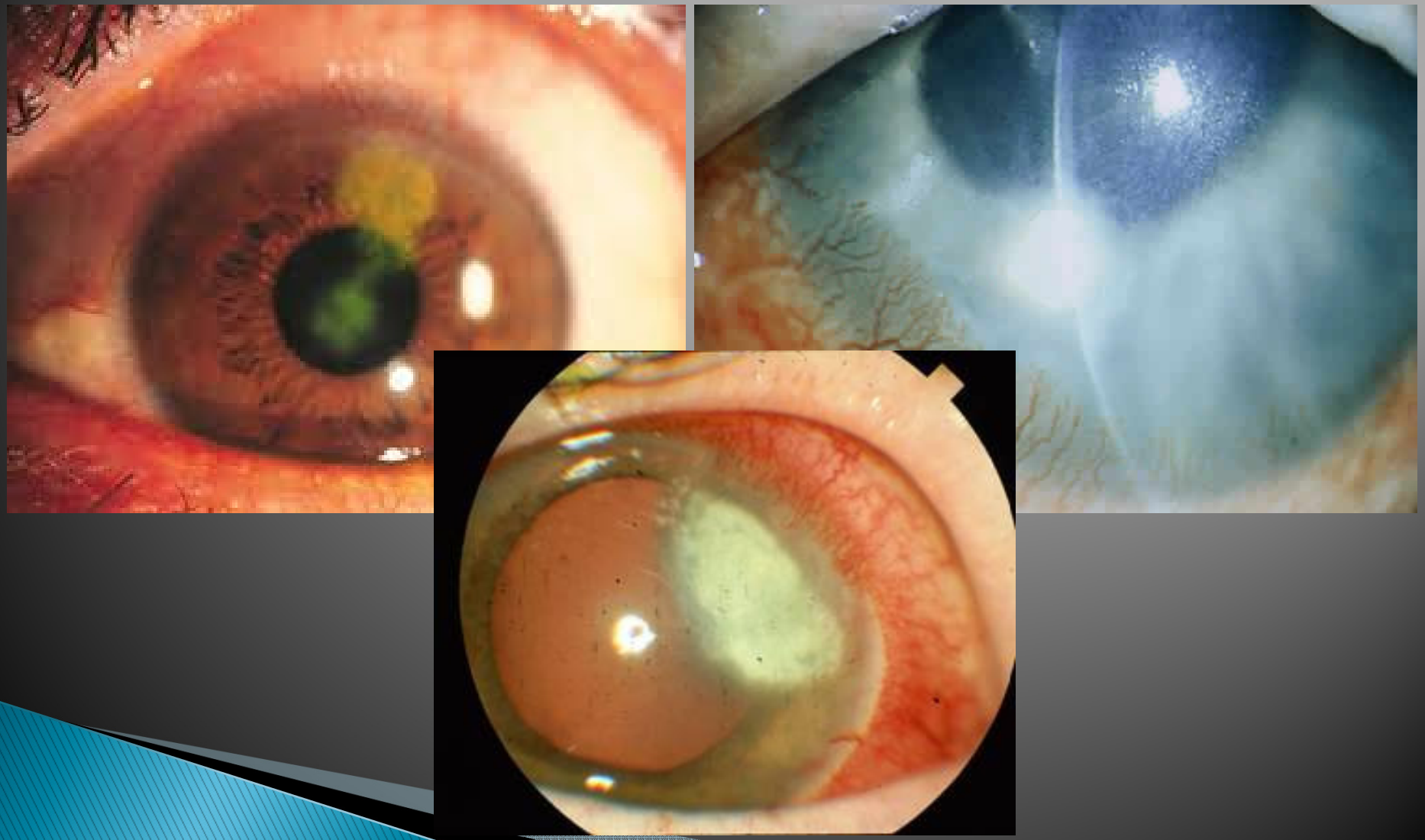


Critical features

- Visual acuity dependant upon location of ulcer and degree of inflammation
- Painful, red eye usually with epithelial defect ± stromal opacity
- Hypopyon indicates severe inflammatory response

- Predisposing factors:
  - Trauma
  - Contact lenses
  - Topical steroids
  - Dry eye/Ocular surface disease
  - Corneal anaesthesia

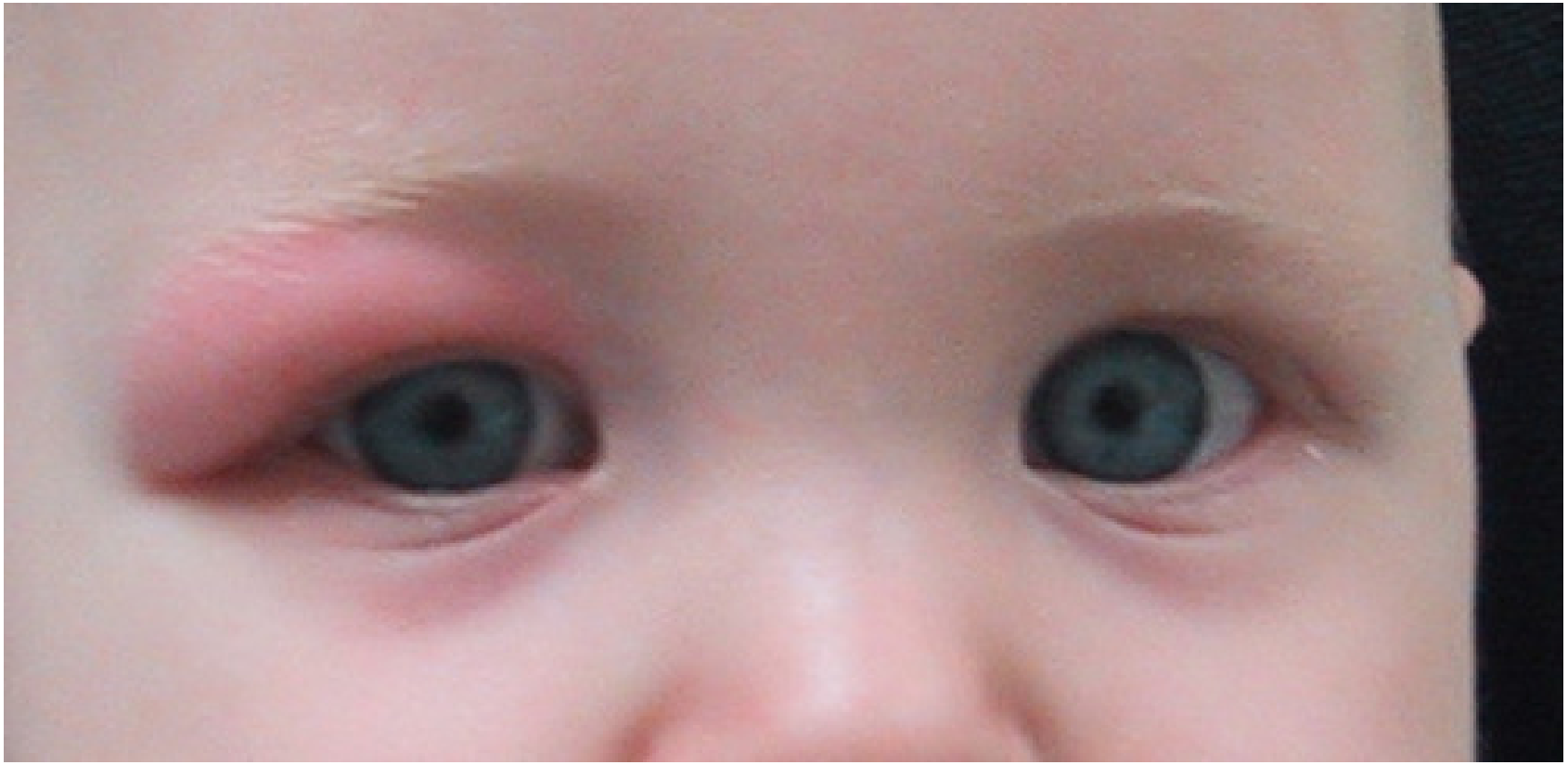
Infective corneal ulcers



Orbital Cellulitis

- Divided into preseptal and orbital cellulitis
- Preseptal cellulitis is defined as an infection of the subcutaneous tissues anterior to the orbital septum
- Critical features:
  - Unilateral, tender, red periorbital and lid oedema
  - May have antecedent cause, e.g. Laceration
  - Eye is not involved, i.e. Conjunctiva should be white, full range of movements

Preseptal cellulitis

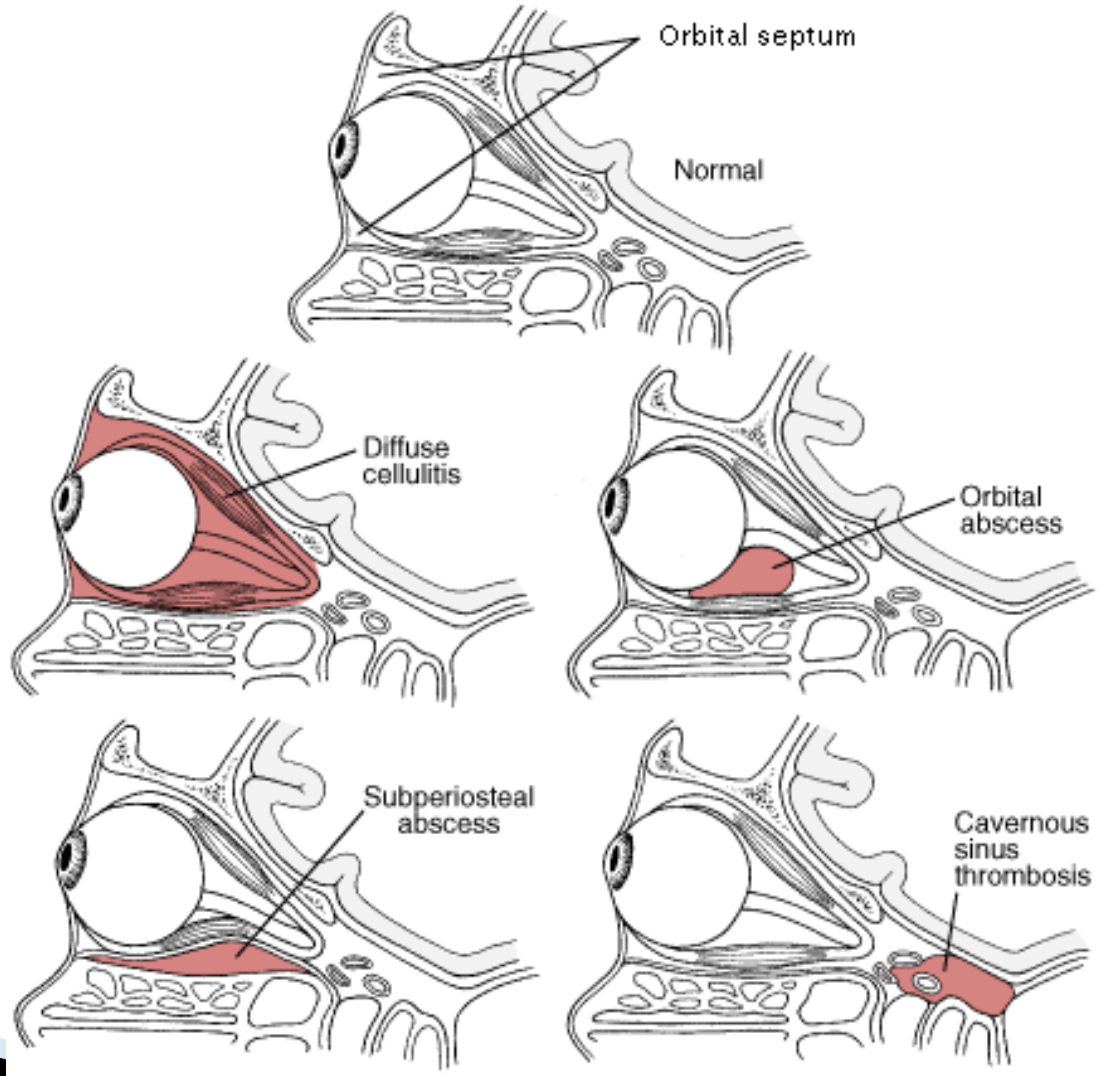


Orbital cellulitis

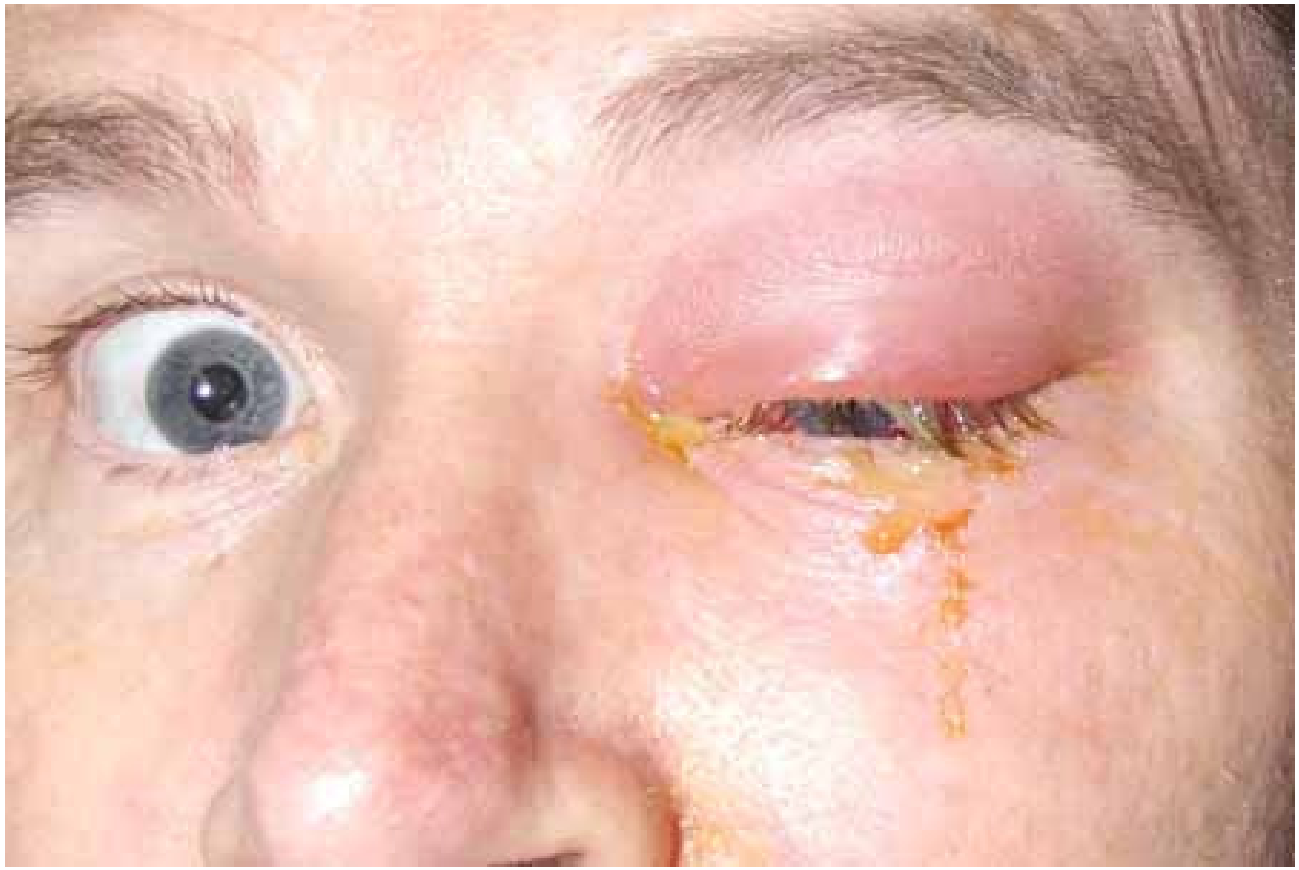
- Potentially life-threatening infection of the tissues behind the orbital septum
- More common in children but any age may be affected

- Critical features:
  - Unilateral, tender, red periorbital and lid oedema
  - Proptosis
  - Painful ophthalmoplegia
  - Optic nerve dysfunction (RAPD, decreased vision)

Orbital cellulitis



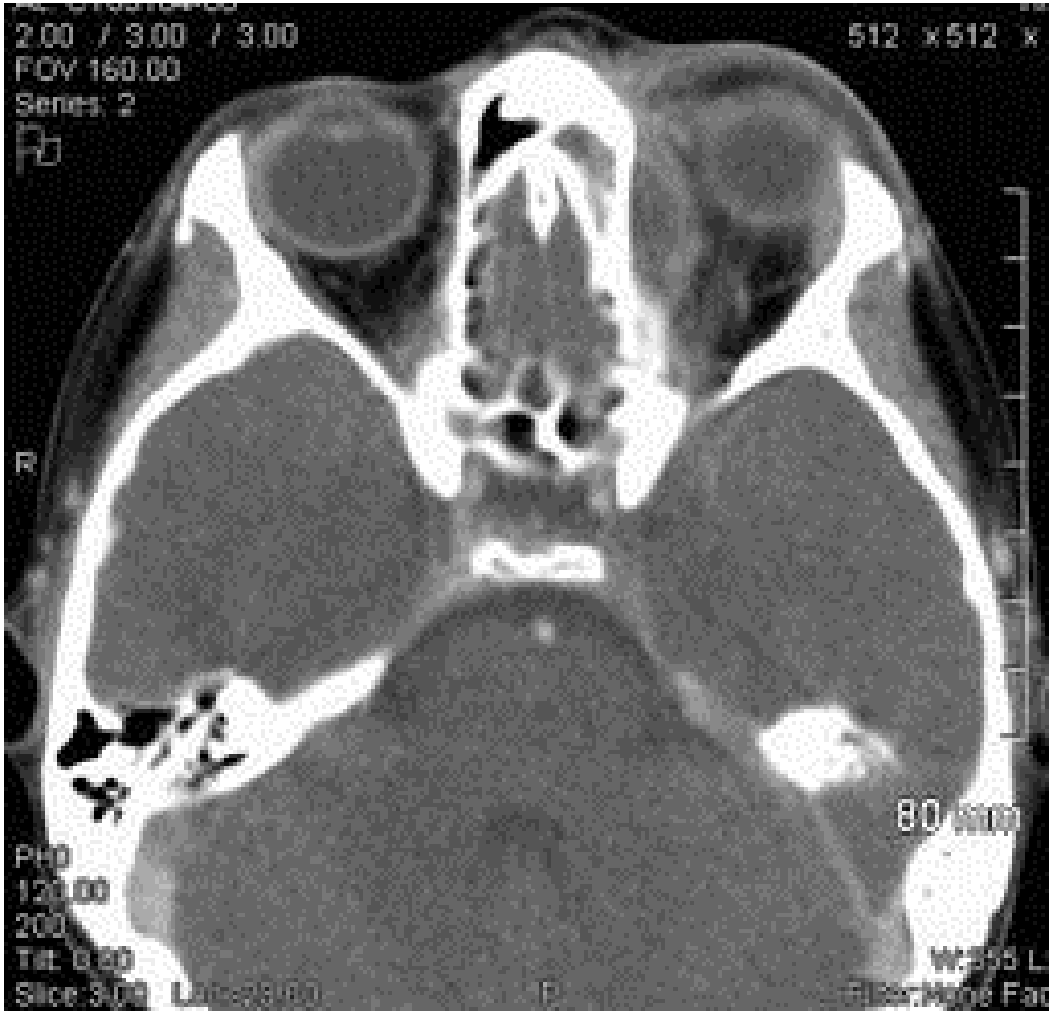
Orbital cellulitis



Orbital cellulitis

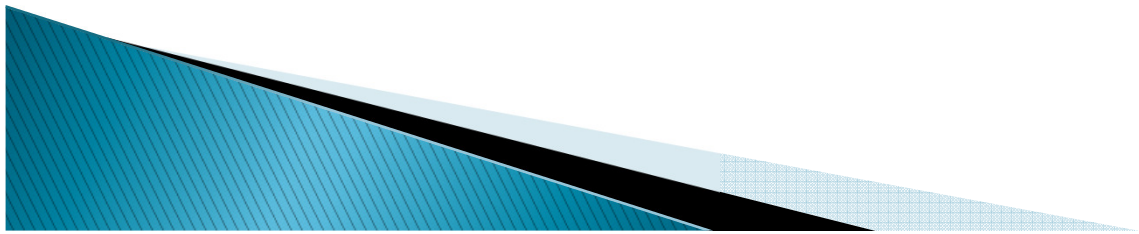
- Typically associated with sinus disease however may be due to extension of preseptal cellulitis, local spread (dacryocystitis, dental abscess), haematogenous or post-traumatic/surgical
- Complications:
  - Ocular: endophthalmitis, CRA/V occlusion, optic neuropathy
  - Intracranial (rare): meningitis, abscess, cavernous sinus thrombosis
  - Subperiosteal/Orbital abscess

Orbital cellulitis



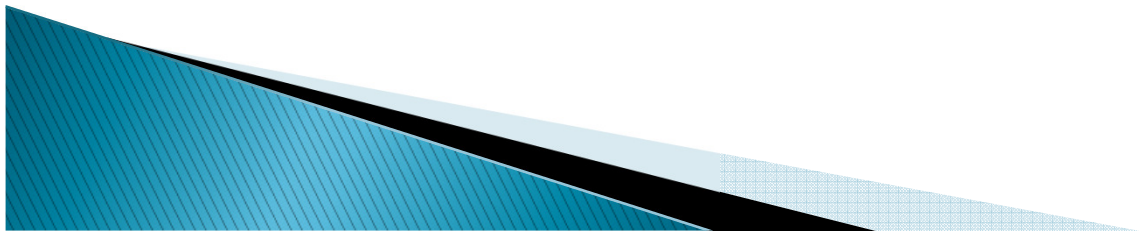
Ophthalmology and Headaches

- A very broad topic as “headache” can commonly be associated with ophthalmic symptoms;
  - Ranges from the “harmless” such as aesthenopia (or “eye strain”),
  - To very uncomfortable such as Migraine with visual aura,
  - To vision-threatening such as Acute angle closure (previously mentioned),
  - To possibly life-threatening…

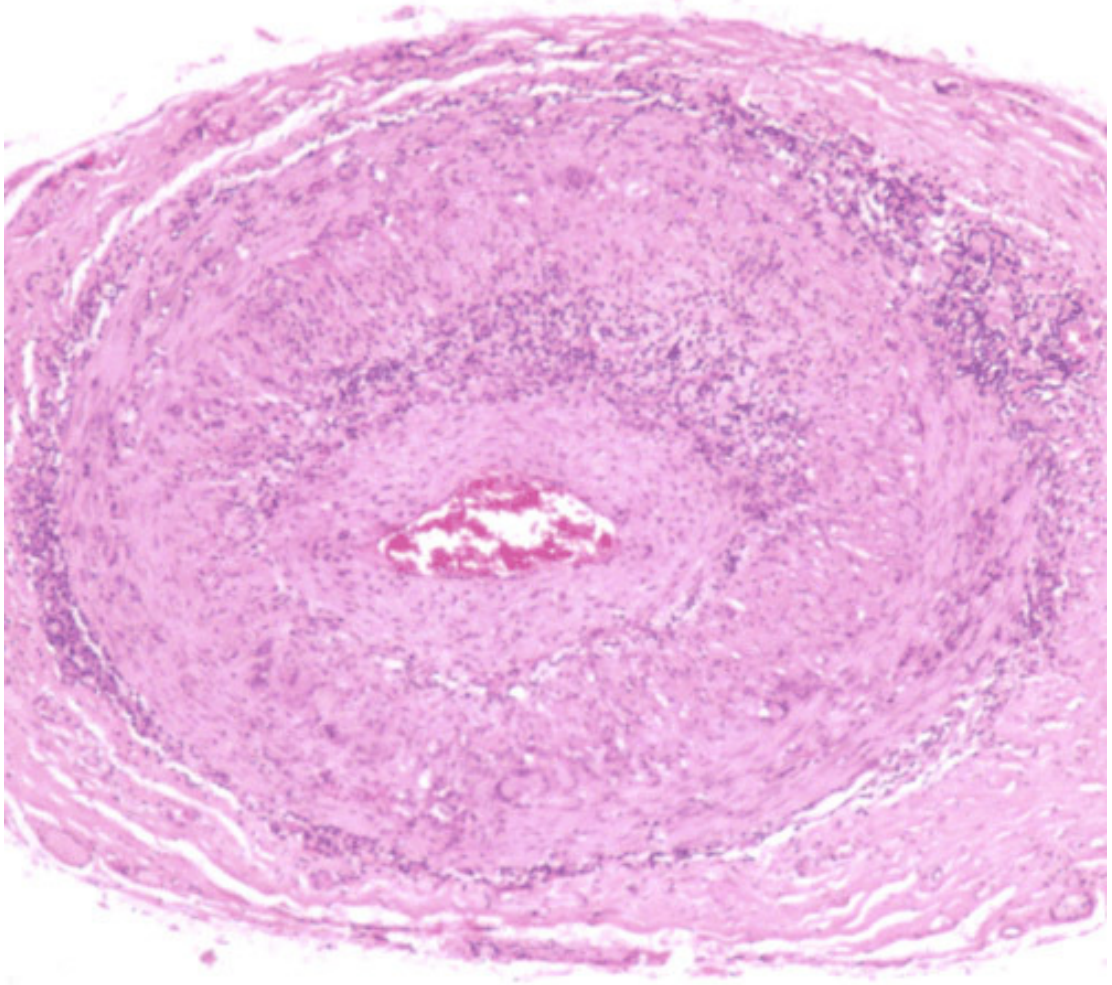


Giant cell/Temporal arteritis

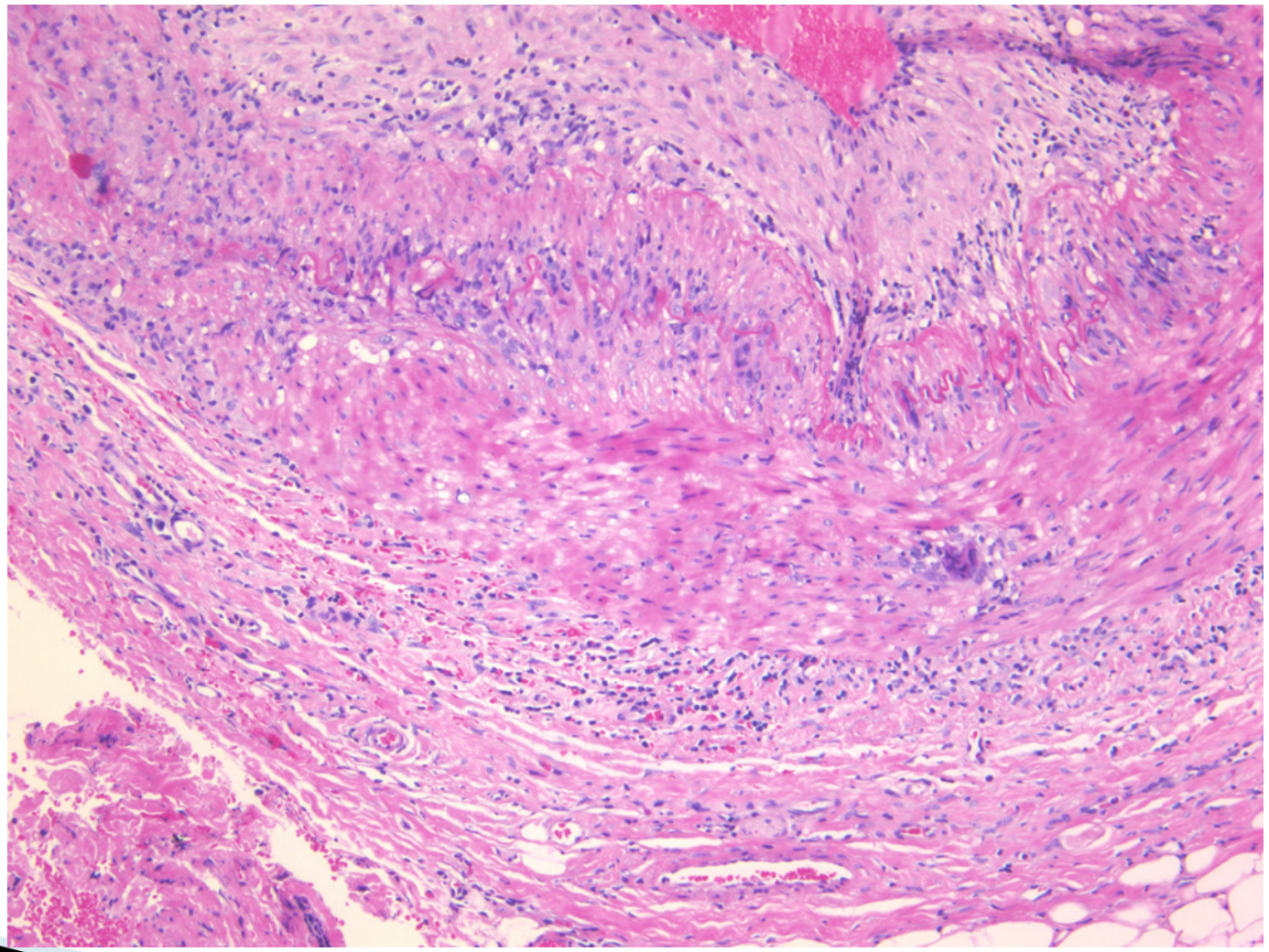
- Granulomatous inflammatory disease of medium to large arteries
- Ophthalmic emergency as it can cause profound and (generally) permanent ischaemic optic neuropathy
- Medical emergency as patients also at risk of MI, brain stem stroke, dissecting aneurysms and renal failure due to involvement of extracranial arteries



Arteritis



Giant cell



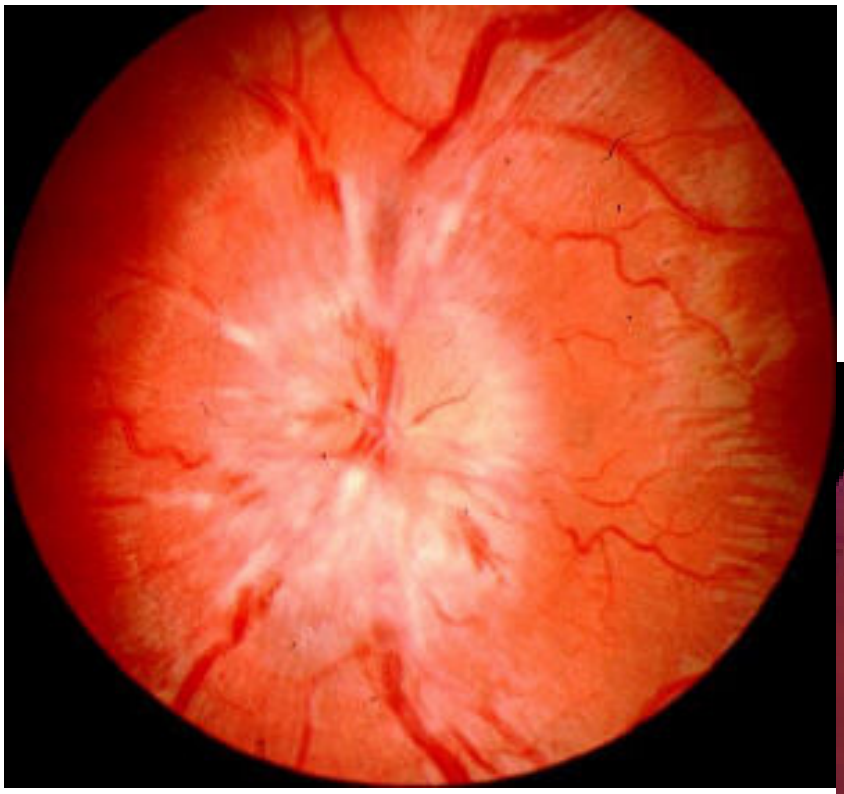
GCA (cont.)

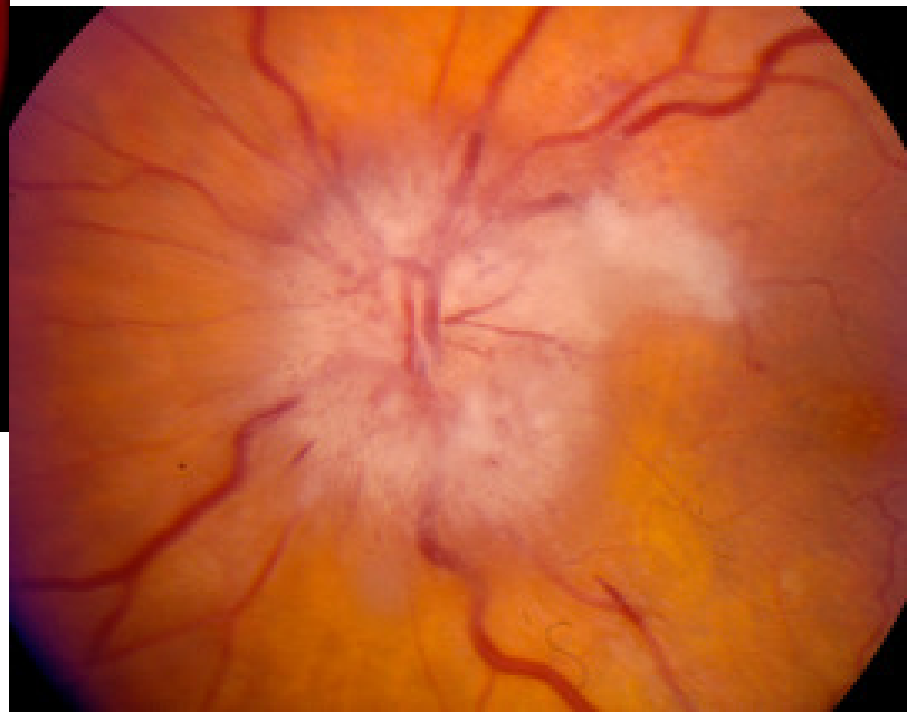
Critical features:

- New-onset headache, jaw claudication, scalp tenderness
- Non-specific symptoms include malaise, night sweats, fever, malaise
- May present with profound loss of vision, RAPD if unilateral optic neuropathy, optic disc pale and swollen

- High ESR/CRP/platelet count

GCA (cont.)





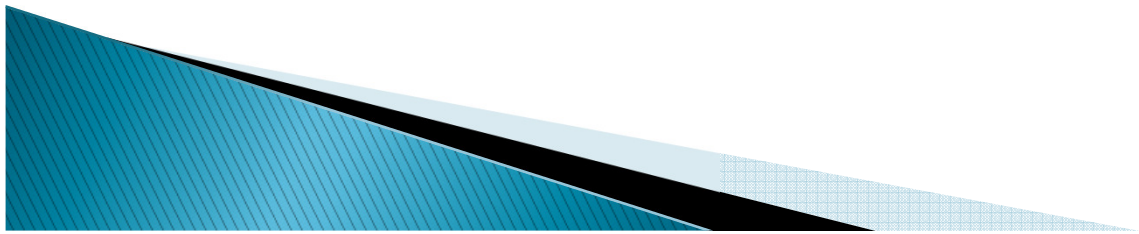
GCA (cont.)

- Estimated that about half the patients with GCA have underlying polymyalgia rheumatica
- About 15% of patients with PMR may develop GCA

- Peak age of incidence is between 60-80 years of age, and generally not considered a diagnosis to be made in patients less than 50 years of age

GCA (cont.)

- As ophthalmology SHOs and registrars, we are taught that if one writes “?GCA”, then the next sentence has to be “Prednisone 60mg” or “Admit for IV Methylprednisolone” (if AAION)
- If there is a high index of suspicion, then steroids should be started and a temporal artery biopsy organised with the appropriate service (either ophthalmology or general/vascular surgical team)



GCA (cont.)

Criteria set by the American College of Rheumatology (presence of ≥3 yields diagnostic sensitivity of 93.5% and specificity of 91.2%)

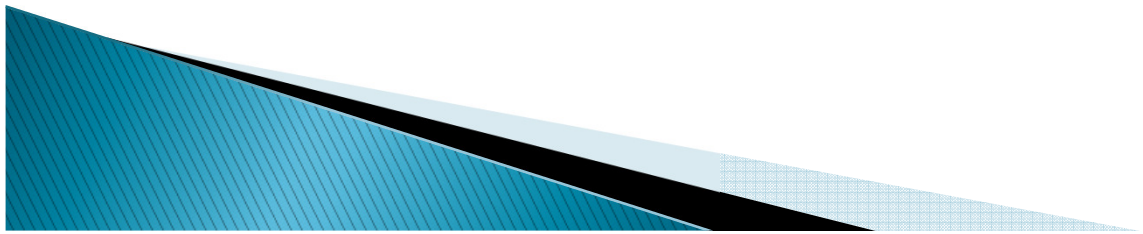
1. Age ≥ 50 years of age
2. New-onset localised headache
3. Temporal artery tenderness or decreased pulsation
4. ESR at least 50mm/h
5. Abnormal artery biopsy specimen

Hunder GG, Bloch DA, Michel BA, Stevens MB, Arend WP, Calabrese LH, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum*. Aug 1990;33(8):1122-8.

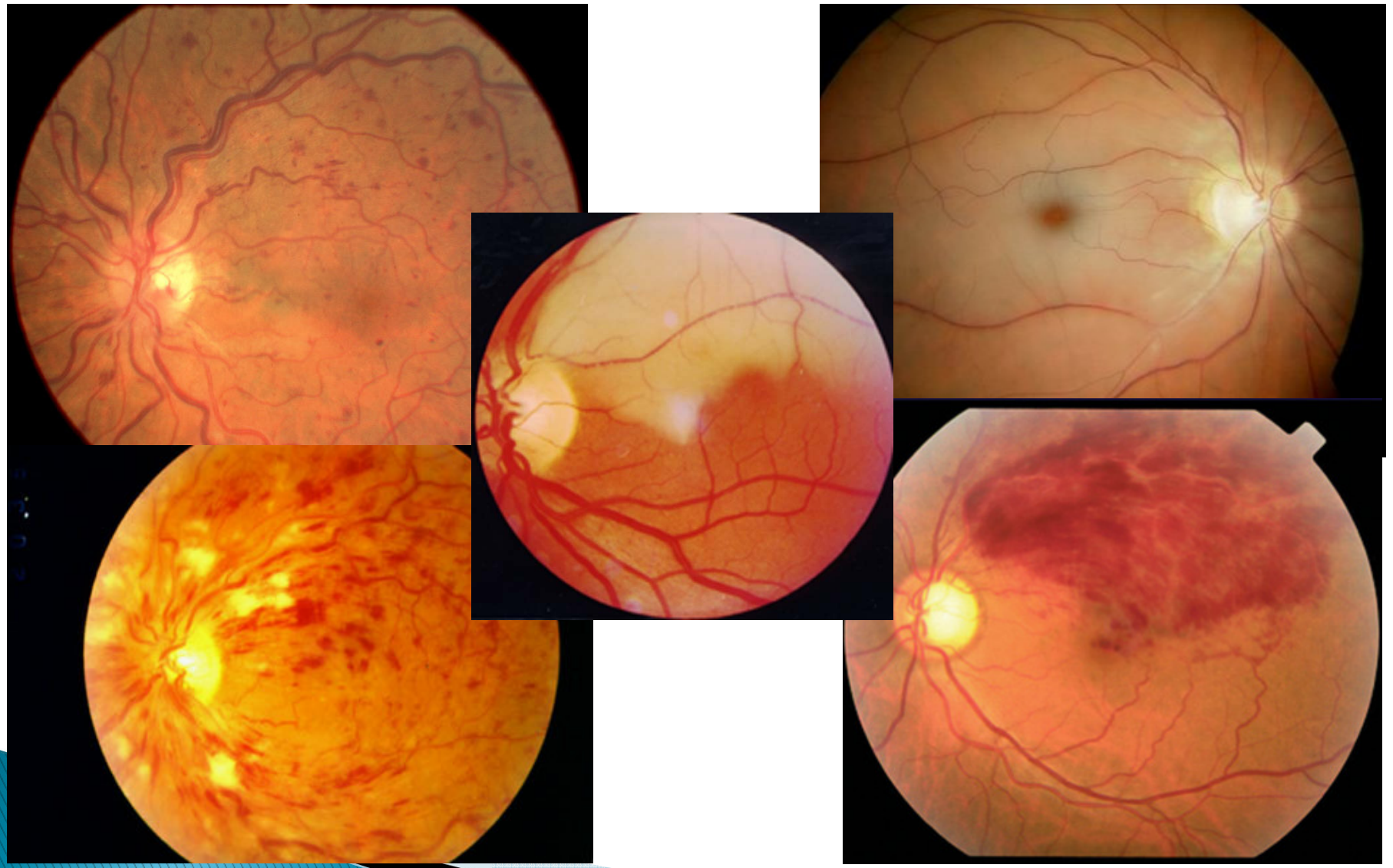
GCA (cont.)

- Interesting to note that a positive temporal artery biopsy (TAB) may not be sufficient to establish a diagnosis of GCA, and
- that you may not need a TAB to make the diagnosis of GCA

- However, a positive TAB does make a very strong case for continuing steroids especially when side-effects become an issue



Vascular occlusions

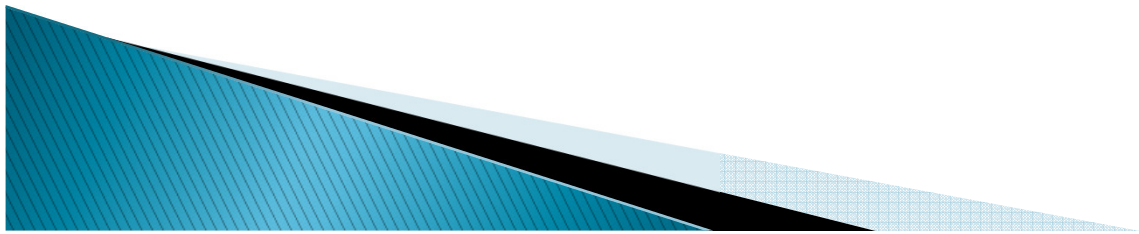


Venous occlusions

Generally I would not triage as an emergent situation, unless there are:

1. Signs of intraocular inflammation (suggestive of a systemic condition that could have significant morbidity or even mortality) or
2. There is a history of thrombotic events (and then it would require anticoagulation)

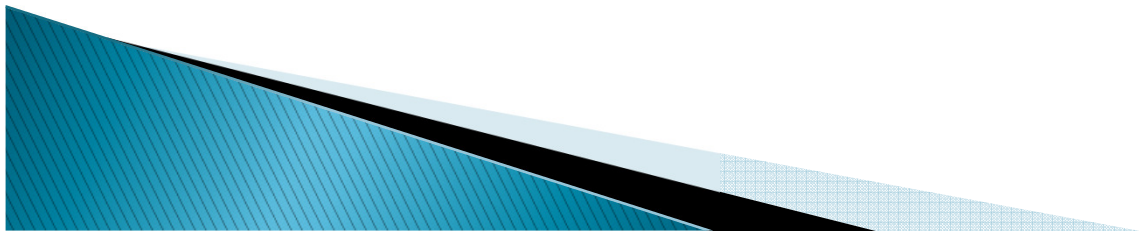
- More common risk factors include age, hypertension, ocular hypertension/ glaucoma, diabetes, smoking and obesity



Arterial occlusions

Retinal arterial occlusions are, in their barest terms, forms of cerebrovascular events

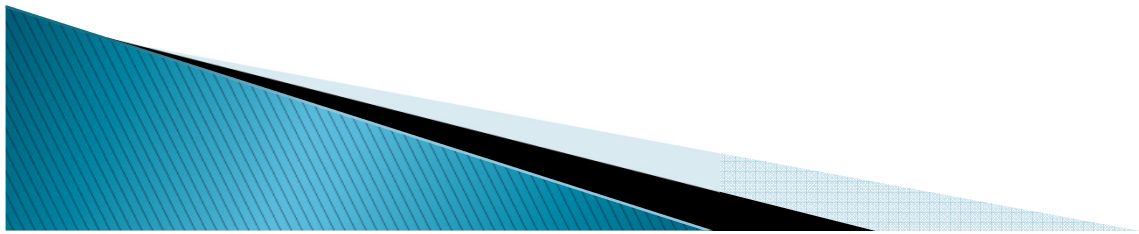
- As such, they can be considered as signs of a potentially life-threatening process
- Usually due to embolic phenomena and thus a thorough cardiovascular assessment (rather than an ophthalmic assessment) is crucial



Arterial occlusions (cont.)

- From an ophthalmic perspective, retinal arterial occlusions tend to present to the eye clinic greater than 6 hours after the event
- By this time, there is (likely) permanent damage to the retinal ganglion cells

- If noticed and identified early, we do try ocular massage (to dislodge the embolus), lowering the IOP (by paracentesis/Diamox), possibly YAG laser, maybe hyperbaric oxygen

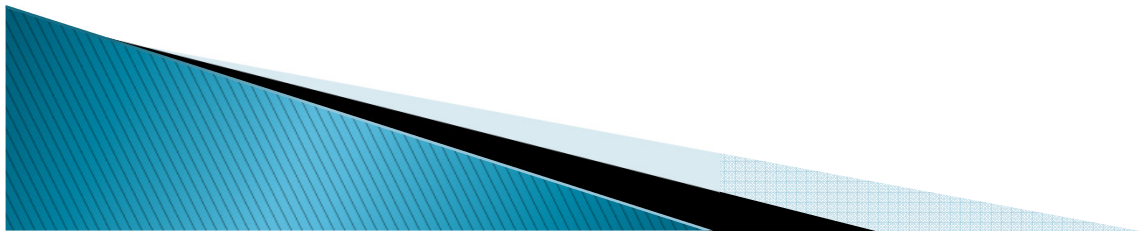


Neuro-ophthalmic emergencies

The following conditions tend to be treated as eye emergencies although the treatment of them are best managed by other specialities, both surgical and medical...

- In other words, the ophthalmologist (and the referrer) tend to be the gateway to further management

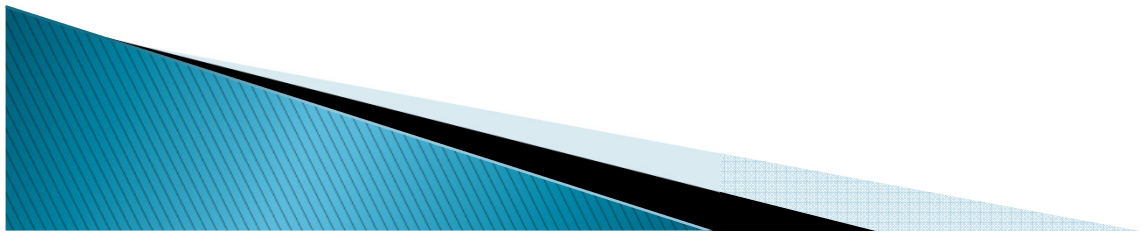
- These conditions also make us (well, me, at least) feel like true, holistic physicians, when diagnosed and referred correctly



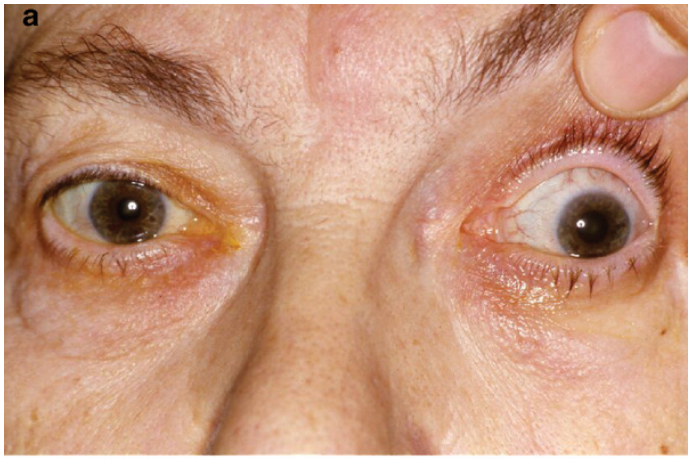
3rd nerve palsy

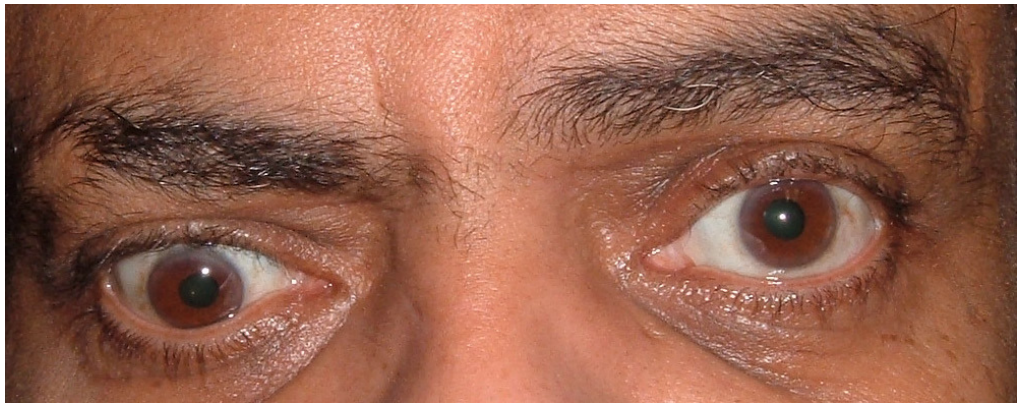
The Oculomotor nerve supplies 4 of the 6 extraocular muscles (the superior, medial and inferior recti, the inferior oblique), the *levator palpebrae superioris*, and the parasympathetic supply to the *constrictor pupillae* (and the sympathetic supply "hitchhikes")

So, varying degrees of "down-and-out" diplopia, ptosis and pupil involvement

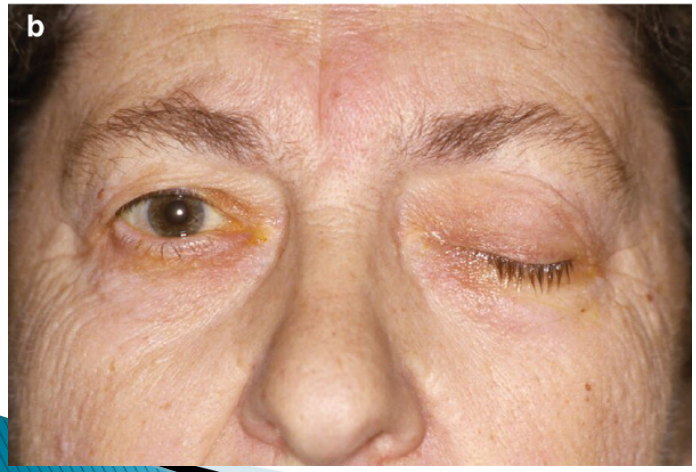


3rd nerve palsy (cont.)





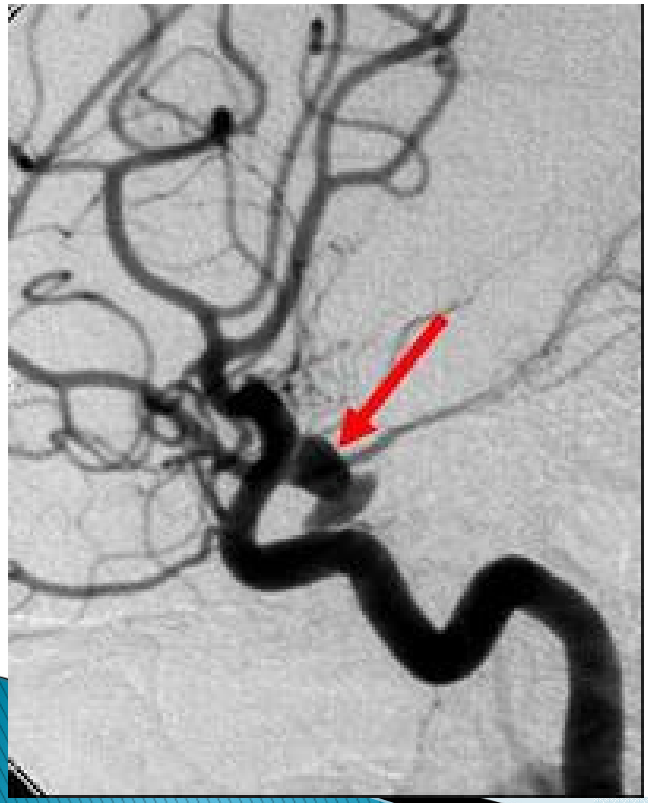
Partial 3rd nerve palsy

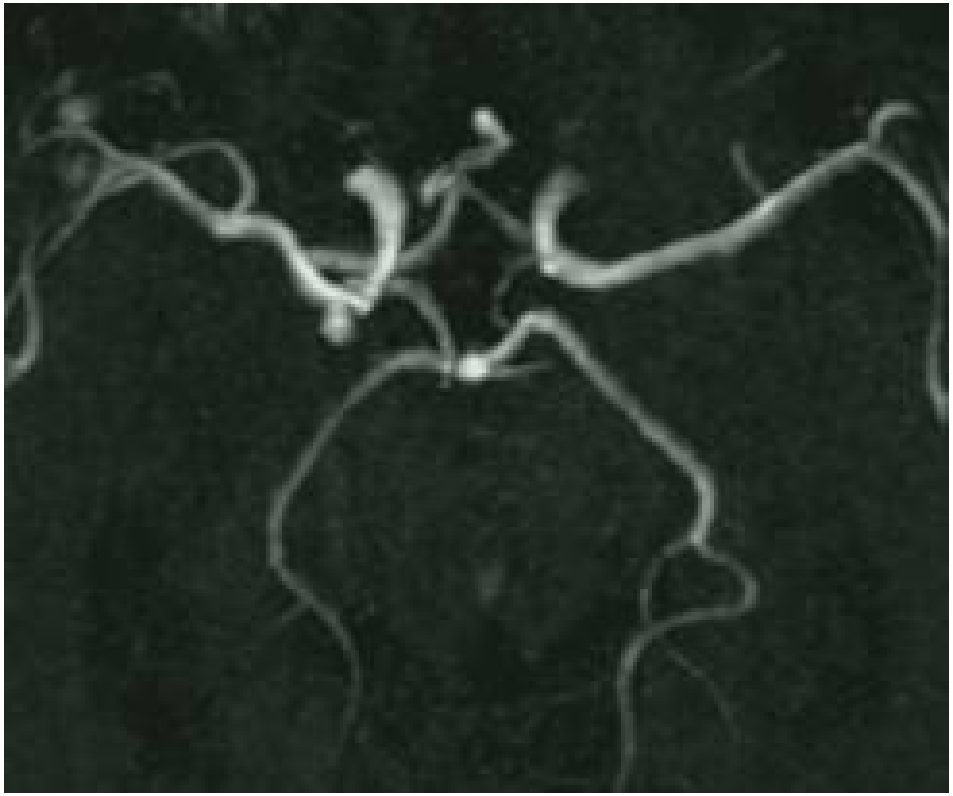


Complete 3rd nerve palsy

3rd nerve palsy (cont.)

The greatest concern is that a 3rd nerve palsy is due to a posterior communicating artery aneurysm





3rd nerve palsy (cont.)

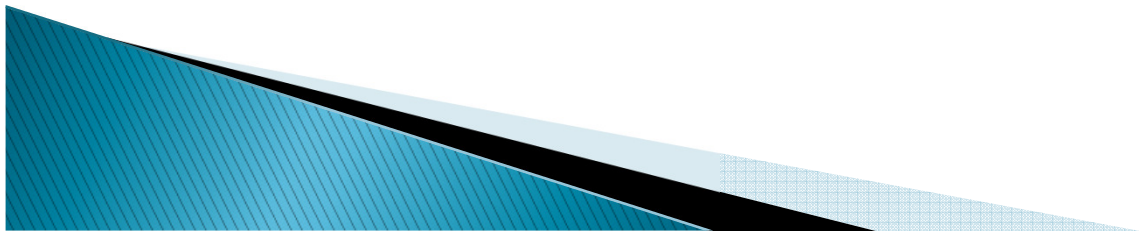
The pupillomotor fibres occupy a superficial position on the Oculomotor nerve, hence susceptible to compression

Critical feature: **Pupil-involvement** where the anisocoria is more evident in bright light

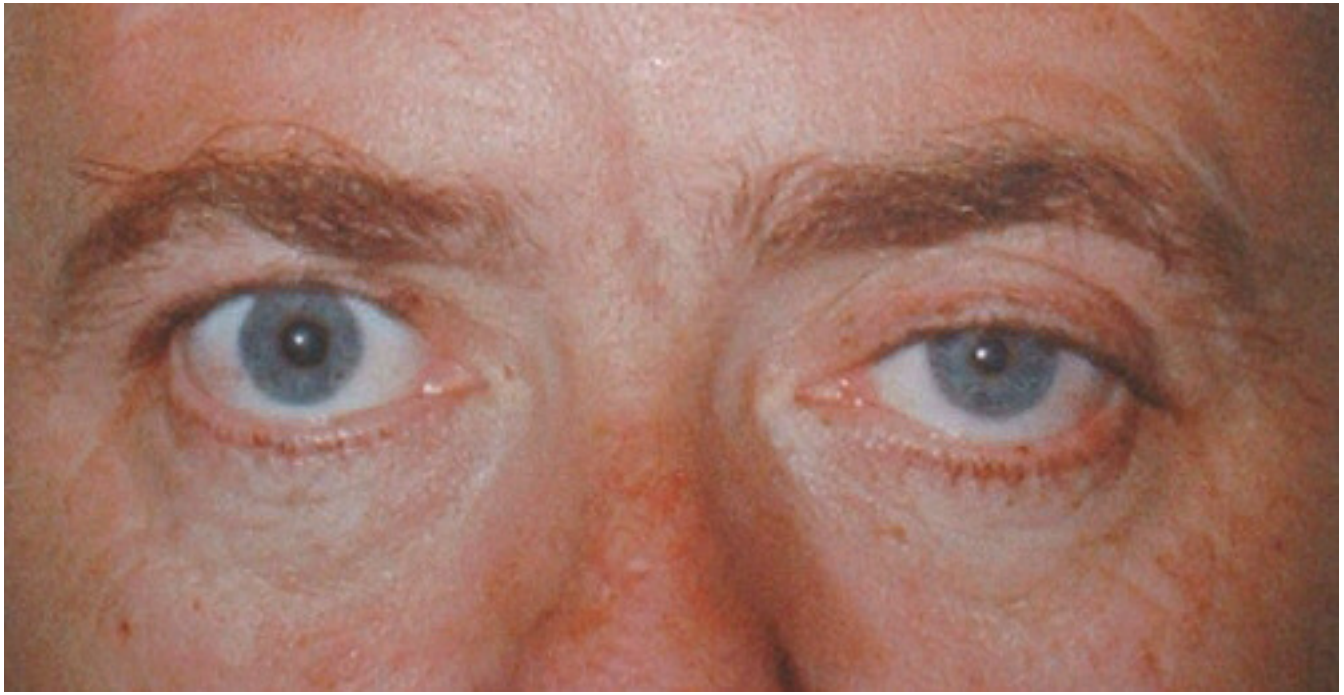
- A 3rd nerve palsy with fixed dilated pupil is PCom aneurysm until proven otherwise
- A complete 3rd nerve palsy that spares the pupil is highly unlikely to be a PCom aneurysm
- A partial 3rd nerve palsy with no apparent pupil involvement is tricky

Horner's syndrome

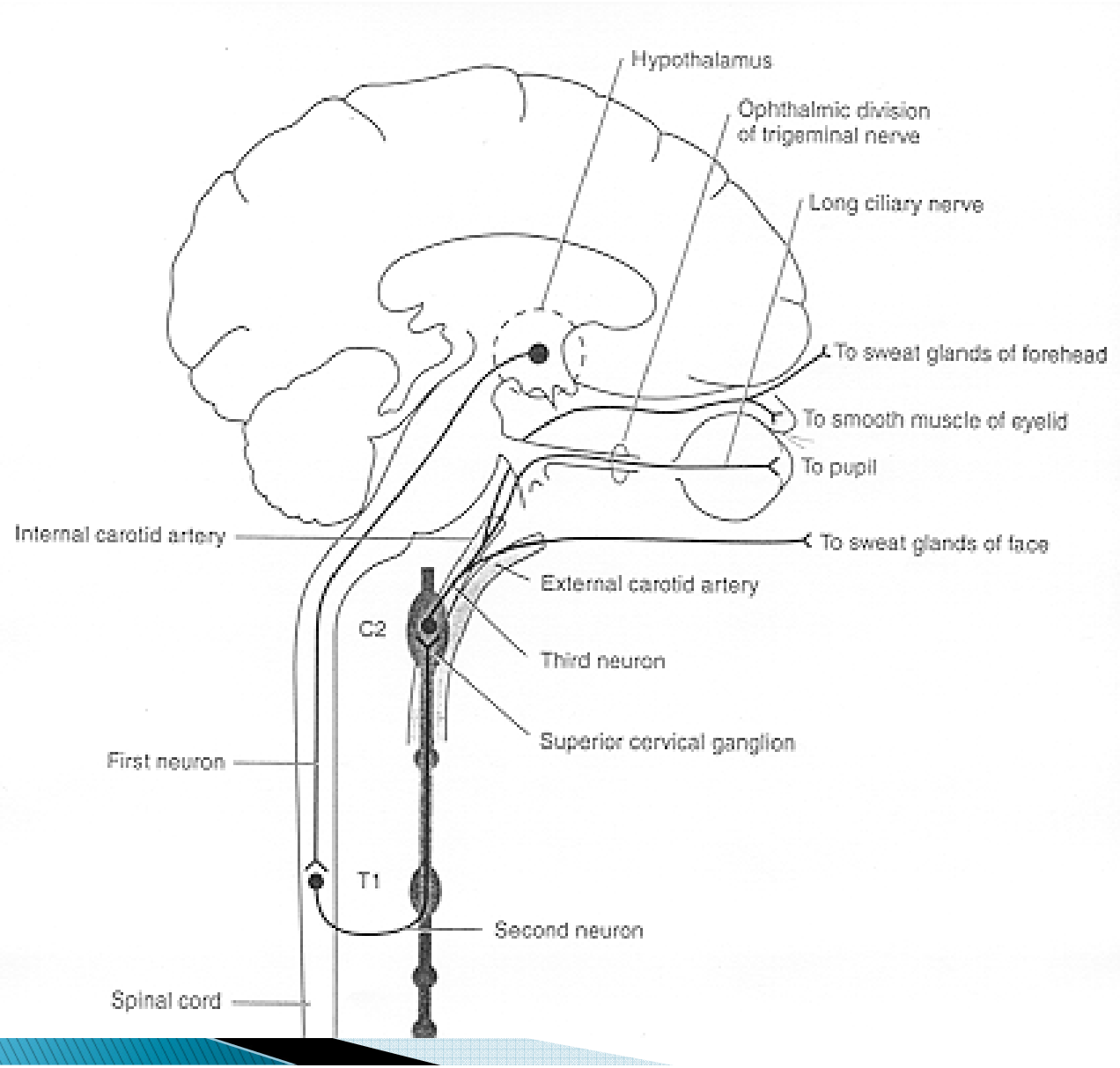
- Occurs due to disruption of the sympathetic supply to the eye
- Critical features:
  - **Anisocoria** where it is more evident in dim light, and the abnormal pupil is the smaller one
  - **Ptosis** on the affected side
  - ± anhidrosis (loss of sweating, depending on location of lesion)
  - ± iris heterochromia (more lightly coloured) in congenital cases of Horner's



Horner's syndrome (cont.)



Horner's syndrome (cont.)



Horner's syndrome (cont.)

- In previous times, it was vital to determine at which level or which order of neuron was affected
  - Pharmacologic testing:
    1. Cocaine 10%: confirms Horner's if smaller pupil remains small (cocaine is a noradrenaline reuptake inhibitor, hence, if no NA is released at the synapse, then no accumulation occurs to cause the pupil to dilate)
    2. Hydroxyamphetamine 1%: causes NA release, hence a 3rd order lesion would hve no response, whereas a 1st or 2nd order neuron would respond with pupil dilation

Horner's syndrome (cont.)

- However, with modern neuro-imaging techniques, trying to localise the lesion is less necessary.
- Furthermore, Cocaine can be quite difficult to obtain

- Recently, to confirm Horner's syndrome, Iopidine has been used more widely, as there is a denervation hypersensitivity to the weak α-adrenergic effects of Iopidine

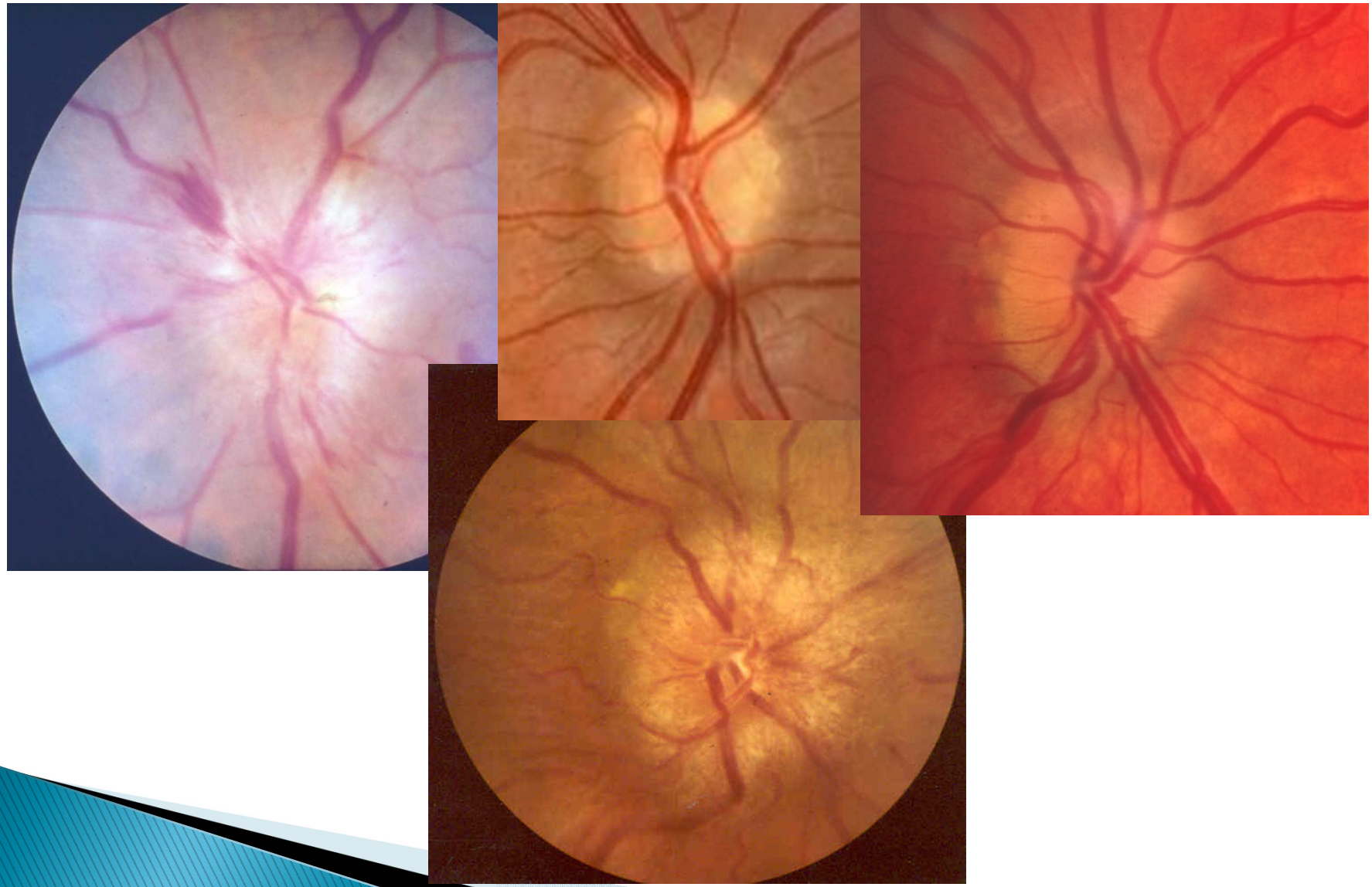
Papilloedema/Swollen discs

The word papilloedema is generally reserved for optic disc swelling that is due to raised intracranial pressure

Critical features:

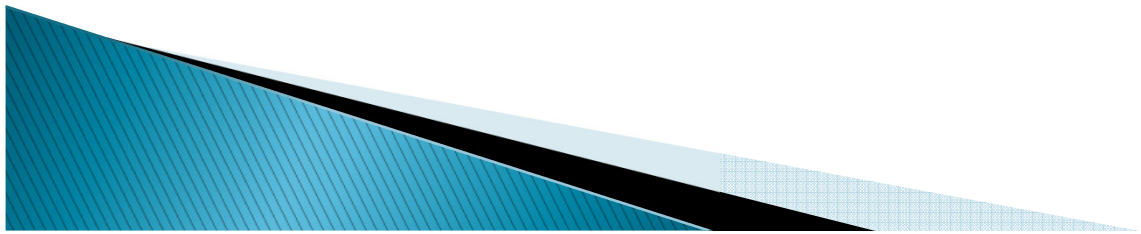
- Symptoms: headache (possibly postural effects), transient visual obscurations (bilateral vision loss lasting seconds), diplopia
- Signs: normal visual acuity (usually), enlarged blind spot, CN VI/LR palsies, bilateral (may be asymmetric) optic disc swelling

But it can be difficult…



Papilloedema (cont.)

- An emergent situation as the causes include:
  - Primary and metastatic intracranial tumours
  - Sub/Epidural haematomas
  - Subarachnoid haemorrhage
  - AV malformations
  - Meningitis/Cerebral abscesses
  - Intracranial venous sinus thrombosis



Thanks for listening