ACUTE VISUAL LOSS

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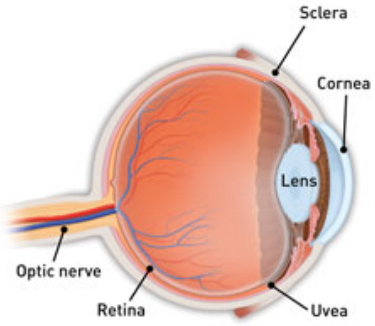
- **Resources**:
  - Lecture notes on ophthalmology book
  - The Lecture slides
  - Wikipedia definitions
  - Pubmed health website

CAUSES OF ACUTE VISUAL LOSS

- **Painful:**
  - Keratitis (inflammation of the Cornea)
  - Acute Angle Closure Glaucoma
  - Uveitis (swelling and irritation of the Uvea)



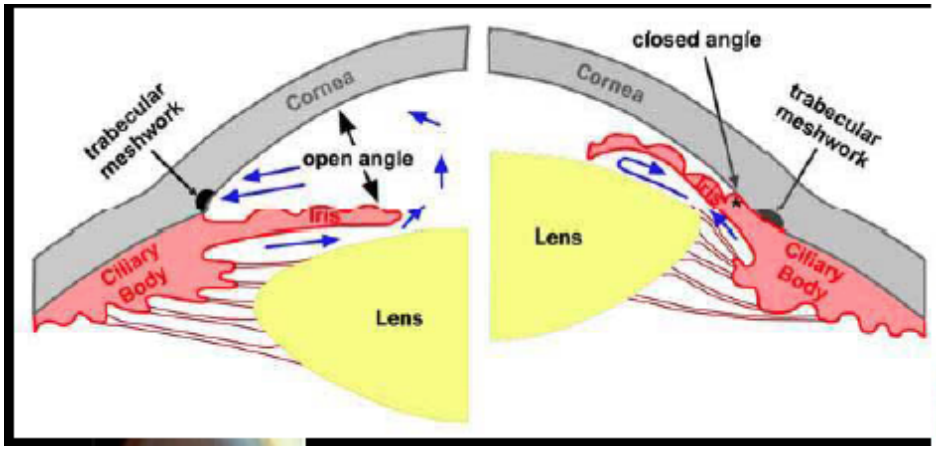
- **Painless:**
  - Vitreous Hemorrhage (*It is the extravasation, or leakage, of blood into the areas in and around the vitreous humor of the eye*)
  - Retinal Detachment *(is a disorder of the eye in which the retina peels away from its underlying layer of support tissue).*
  - Optic Neuritis *(inflammation of the optic nerve)*
  - Ischemic Optic Neuropathy *(is loss of vision due to damage to the optic nerve from insufficient blood supply).*
  - Cerebrovascular Accidents (CVA)
  - Functional Visual Loss *(is a decrease in visual acuity and/or visual field not caused by any organic lesion – also called non-organic visual loss)*

ACUTE GLAUCOMA (CLOSED ANGLE)

Glaucoma is a condition of optic disc changes with/without raised IOP (intraocular pressure). It occurs whenever the aqueous fluid is prevented from its drainage, hence, building up the pressure in the eyes and causing its complications.

- **Types of Glaucoma:**
  - Chronic Open angle glaucoma
  - Acute and Chronic Angle Closure glaucoma
  - Congenital/Developmental Glaucoma

- Secondary Glaucoma (steroid induced, ocular trauma or surgery, hypertension – raised epi-sclera venous pressure)



1) CAUSES

- Occurs in small eyes (i.e. often hypermetropic) with shallow anterior chambers.
- Bowing of the iris when the pupil is dilated may cause adhesions in the aqueous fluid normal drainage and so stops it, hence; raised IOP
- Ocular hypertension is one of the most important complications

2) CLINICAL MANIFESTATIONS

- It is an emergency; with the following clinical manifestations:
  - Sudden ocular pain
  - Seeing halos around lights
  - Red eye
  - Very high intraocular pressure (>30 mmHg)
  - Nausea and vomiting
  - Suddenly decreased vision
  - A fixed, mid-dilated pupil.
  - It is also associated with an oval pupil in some cases
- Diagnosis is through a ***Tonometer***; to measure IOP, ***Gonioscope***; to examine anterior chamber angle and an ***Ophthalmoscope;*** to check and confirm optic disc changes

3) MANAGEMENT

- Goals of Glaucoma Management:
  - Break attacks: void further damage by lowering IOP
  - Prevent further attacks
- Emergent Management (to break the attack and lower IOP):

- **Acetazolamide**, first through IV then orally (reduce aqueous secretion and the pressure across the iris).
- **Beta-Blockers**, topically (reduce aqueous secretion and the pressure across the iris).
- **Pilocarbine,** topically (constricts the pupil and draws the peripheral iris out of the angle).

- Long-term Management (to prevent further attacks):
  - A small hole (iridotomy or iridectomy), through YAG laser or surgically, and is made in the peripheral iris to prevent subsequent attacks of angle closure
  - If the pressure has been raised for some days the iris becomes adherent to the peripheral cornea (peripheral anterior synechiae or PAS). The iridocorneal angle is damaged and additional medical or surgical measures may be required to lower the ocular pressure.

RETINAL ARTERY AND VEIN OCCLUSION

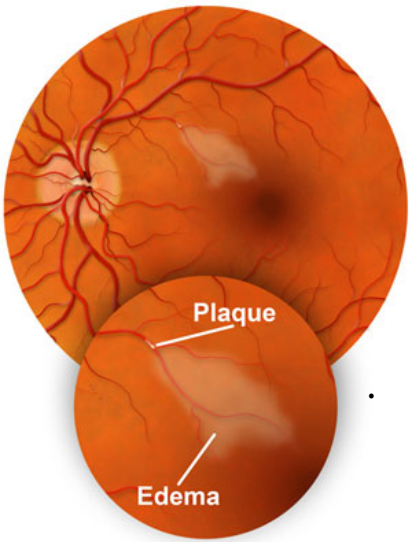
1) CAUSES

- Central and branch retinal artery occlusions are usually embolic in origin. Three types of emboli are recognized:
  - **Fibrin-platelet emboli** commonly from diseased carotid arteries
  - **Cholesterol emboli** commonly from diseased carotid arteries
  - **Calcific emboli** from diseased heart valves.
  - **Vasculitis;** such as giant cell arteritis
- Central retinal vein occlusion (CRVO) may result from:
  - Abnormality of the blood itself (the hyperviscosity syndromes and abnormalities in coagulation)
  - An abnormality of the venous wall (inflammation)
  - An increased ocular pressure.

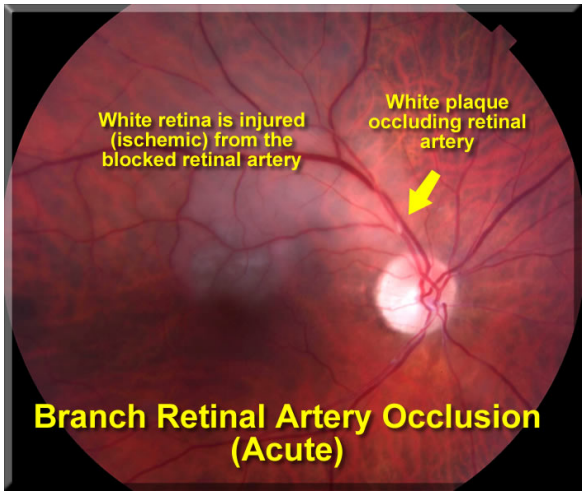
2) CLINICAL MANIFESTATIONS

- Arterial occlusion manifestations:
  - Sudden painless loss of all or part of the vision.
  - Acute Signs on a retinoscope:
    - > Occasionally, a series of white platelet emboli can be seen passing rapidly through a vessel
    - > More often a bright yellow, reflective cholesterol embolus is noted occluding an arterial branch point.

Branch Retinal Artery Occlusion

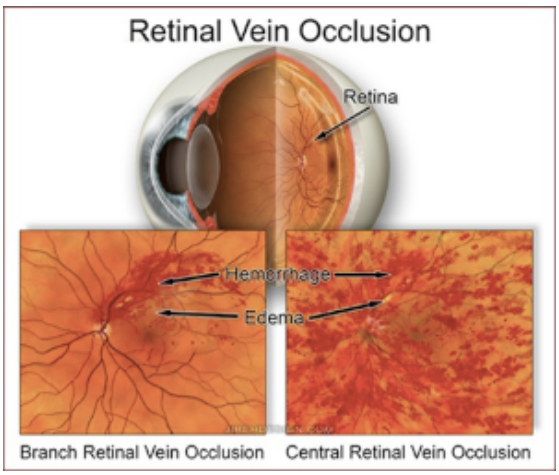


> The acutely affected retina is swollen and white (oedematous),
> The fovea is red (cherry red spot) as it has no supply from the retinal circulation and is not swollen
> The normal choroid can be seen through the fovea.



- After several weeks the disc becomes pale (atrophic) and the arterioles attenuated.

- **Venous Occlusion Manifestations:**
  - Sudden partial or complete loss of vision although onset may be less acute than that of arterial occlusion.



· Acute Signs on Retinoscope:

> There is marked haemorrhage and great tortuosity and swelling of the veins.
> The optic disc appears swollen.
> Branch retinal vein occlusion may originate at the crossing point of an arteriole and a vein where the arteriole has been affected by arteriosclerosis associated with hypertension (a/v nipping).

· Subsequent changes:

> Abnormal new vessels may grow on the retina and optic disc, causing vitreous haemorrhage (This happens if the retina has become ischaemic as a result of the vein occlusion - an ischaemic retinal vein occlusion).
> In ischaemic retinal vein occlusion abnormal new vessels may grow on the iris causing rubeotic glaucoma.

3) MANAGEMENT

- **Arterial occlusion management:**
  - Lowering the intraocular pressure with intravenous acetazolamide
  - Ocular massage

- Paracentesis (a needle is inserted into the anterior chamber to release aqueous and lower the intraocular pressure rapidly)
- Getting the patient to rebreath into a paper bag firmly applied around the mouth and nose to use the vasodilatatory effect of raised carbon dioxide levels.

- **Venous occlusion management:**

  Retinal laser treatment is given if the retina is ischaemic to prevent the development of retinal and iris new vessels. Laser treatment may improve vision in some patients with a branch retinal vein occlusion by reducing macular oedema.

RETINAL DETACHMENT

- It is a disorder of the eye in which the retina peels away from its underlying layer of support tissue.
- Initial detachment may be localized, but without rapid treatment the entire retina may detach, leading to vision loss and blindness.
- It is a medical emergency

1) CLASSIFICATION

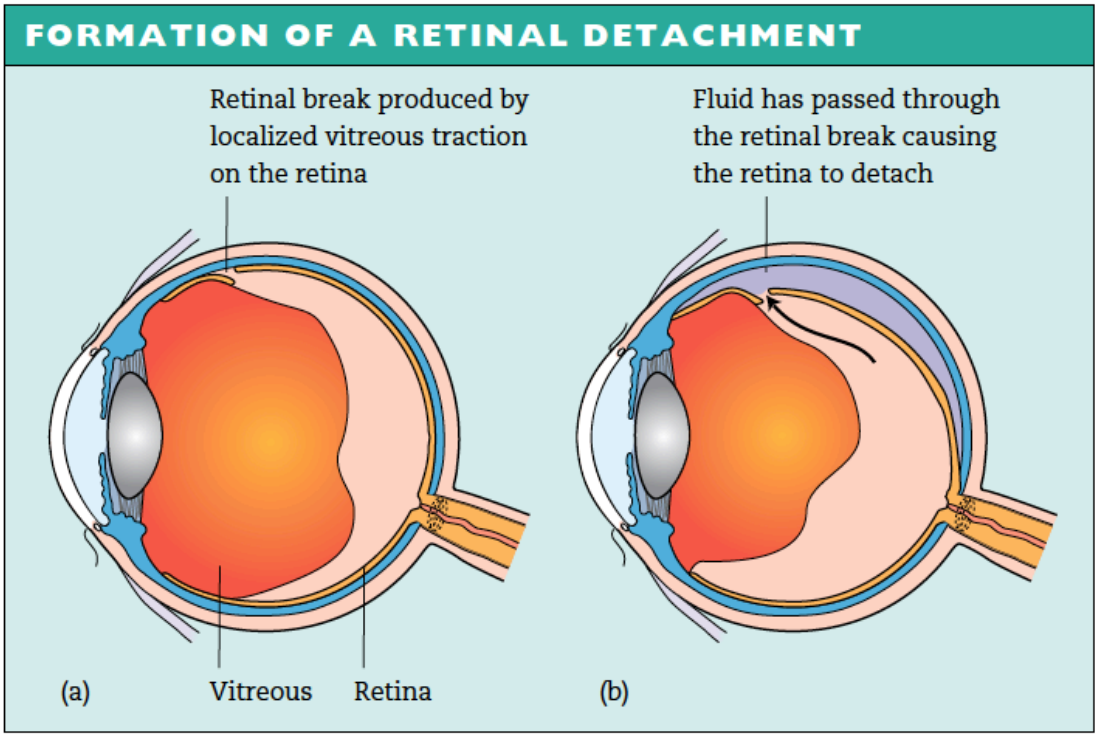


Fig. 11.8 The formation of a rhegmatogenous retinal detachment. (a) The detaching vitreous has torn the retina. The vitreous continues to pull on the retina surrounding the break (vitreous traction). (b) Fluid from the vitreous cavity passes through the break, detaching the retina from the underlying retinal pigment epithelium.

RHEGMATOGENOUS RETINAL DETACHMENT

- It occurs due to a retinal tear or break that allows fluid to pass from the vitreous space into the subretinal space between the sensory retina and the retinal pigment epithelium.
- Retinal breaks are divided into three types:
    - **Holes**; Form due to retinal atrophy especially within an area of lattice degeneration.
    - **Tears**; are due to vitreoretinal traction).
    - **Dialyses**, which are very peripheral and circumferential; may be either tractional or atrophic (the atrophic form most often occurring as idiopathic dialysis of the young).

EXUDATIVE, SEROUS, OR SECONDARY RETINAL DETACHMENT CAUSES

- An exudative retinal detachment occurs due to inflammation, injury or vascular abnormalities that results in fluid accumulating underneath the retina without the presence of a hole, tear, or break.
- In evaluation of retinal detachment, it is critical to exclude exudative detachment as surgery will make the situation worse, not better.
- Although rare, exudative retinal detachment can be caused by the growth of a tumor on the layers of tissue beneath the retina, namely the choroid. This cancer is called a choroidal melanoma.

TRACTIONAL RETINAL DETACHMENT

- The retina is pulled away from the pigment epithelium by contracting fibrous tissue which has grown on the retinal surface.
- This may be seen in proliferative diabetic retinopathy or may occur as a result of proliferative vitreoretinopathy.
- Vitreoretinal surgery is required to repair these detachments.

2) CAUSES

1. Tear in the retina
2. Uncontrolled Diabetes
3. Chronic inflammation
4. Cancer (choroidal melanoma)

3) CLINICAL MANIFESTATIONS

- Bright flashes of light, especially in peripheral vision

- Blurred vision
- Floaters in the eye
- Shadow or blindness in a part of the visual field of one eye

4) MANAGEMENT

- There are two major surgical techniques for repairing a retinal detachment:
  1. External (conventional approach).
  2. Internal (vitreoretinal surgery).

UVEITIS

1) CAUSES

- Autoimmune disorders (such as sarcoidosis, rheumatoid arthritis, systemic lupus erythematosus, Behcet's disease, and ankylosing spondylitis)
- infections (such as syphilis and toxoplasmosis)
- Some are “idiopathic.

CAUSES OF UVEITIS

Infectious	Associated with systemic disease	Ocular disease
Toxoplasmosis	Ankylosing spondylosis	Advanced cataract
Postoperative infection	Sarcoidosis	Sympathetic ophthalmitis
Fungal	Reiter’s disease	Retinal detachment
CMV	Behçet’s disease	Angle closure glaucoma
Herpetic	Psoriatic arthritis	Intraocular tumours
Tuberculosis	Juvenile chronic arthritis	
Syphilis	Inflammatory bowel disease	
Metastatic infection		
Toxocara		

Table 9.1 Table showing some causes of uveitis (this is not an exclusive list).

2) CLINICAL MANIFESTATIONS

- Ocular pain (less frequent with posterior uveitis or choroiditis, as posterior uveitis may not be painful).
- Photophobia;
- Blurring of vision;
- Redness of the eye.

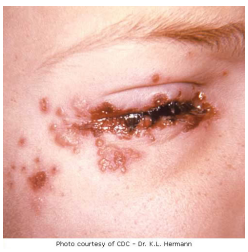
3) MANAGEMENT

- Steroids to stop the inflammation, given as eye drops, injection in or around the eye, orally (by mouth), or intravenously, depending on the extent and severity of the inflammation.
- In certain situations, alternatives to steroids (such as indomethacin, methotrexate, and others) may be used.
- The duration of the treatment may be as short as a week or several months or even years, depending on the cause.
- Treating the underlying cause; if the cause is infectious, an anti-infective medication will also be used (antibiotic, antiviral, or antifungal) to combat the underlying infectious agent.

KERATITIS

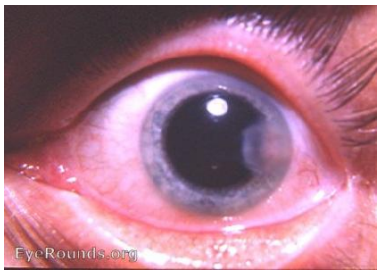
1) CAUSES

HERPES SIMPLEX KERATITIS



- Usually type 1 HSV. The cornea may not be involved although punctate epithelial damage may be seen.
- Recurrent infection results from activation of the virus lying latent in the trigeminal ganglion of the fifth cranial nerve.
- It is characterized by the appearance of dendritic ulcers on the cornea that usually heal without a scar

CLINICAL MANIFESTATIONS:



- Fever;
- Vesicular lid lesions;
- Follicular conjunctivitis;
- Pre-auricular lymphadenopathy;
- Most are asymptomatic.

MANAGEMENT:

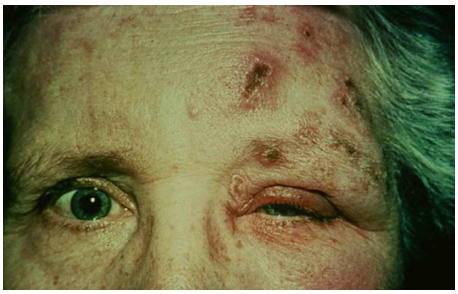
Usually antiviral. Topical steroids must not be given to patients with a dendritic ulcer since they may cause extensive corneal ulceration. In patients with stromal involvement (keratitis) steroids are used under ophthalmic supervision and with antiviral cover.

ANTIVIRAL AGENTS
Vidarabine
Trifluorothymidine
Aciclovir
Ganciclovir

HERPES ZOSTER OPHTHALMICUS (OPHTHALMIC SHINGLES)

- This is caused by the varicella-zoster virus which is responsible for chickenpox.
- The ophthalmic division of the trigeminal nerve is affected.
- Unlike herpes simplex infection, there is usually a prodromal period with the patient systemically unwell.

CLINICAL MANIFESTATION:



- Lid swelling (which may be bilateral);
- Keratitis;
- Iritis;
- Secondary glaucoma.

MANAGEMENT:

- Oral antiviral treatment (e.g. aciclovir and famciclovir) is effective in reducing post-infective neuralgia (a severe chronic pain in the area of the rash) if given within 3 days of the skin vesicles erupting.
- Ocular disease may require treatment with topical antivirals and steroids.

BACTERIAL KERATITIS

CLINICAL MANIFESTATIONS:

- **Pain**, usually severe unless the cornea is anaesthetic;
- **Purulent discharge;**
- **Ciliary injection;**
- **Visual impairment** (severe if the visual axis is involved);
- **Hypopyon** sometimes (a mass of white cells collected in the anterior chamber
- **White corneal opacity** which can often be seen with the naked eye

BACTERIA CAUSING CORNEAL INFECTION

- *Staphylococcus epidermidis*
- *Staphylococcus aureus*
- *Streptococcus pneumoniae*
- Coliforms
- *Pseudomonas*
- *Haemophilus*

MANAGEMENT:

- Scrapes are taken from the base of the ulcer for Gram staining and culture.
- The patient is then treated with intensive topical antibiotics often with dual therapy (e.g. cefuroxime against Gram +ve bacteria and gentamicin
- for Gram -ve bacteria) to cover most organisms.

- The use of fluoroquinolones (e.g. Ciprofloxacin, Ofloxacin) as a monotherapy is gaining popularity.

OTHER CAUSES:

- ACANTHAMOEBA KERATITIS; a freshwater amoeba that is responsible for infective keratitis. The infection is becoming more common due to the increasing use of soft contact lenses
- FUNGAL KERATITIS; It should be considered in:
  - Lack of response to antibacterial therapy in corneal ulceration;
  - Cases of trauma with vegetable matter;
  - Cases associated with the prolonged use of steroids.
- INTERSTITIAL KERATITIS; This term is used for any keratitis that affects the corneal stroma without epithelial involvement. Classically the most common cause was syphillis, leaving a mid stromal scar with the outline ('ghost') of blood vessels seen.

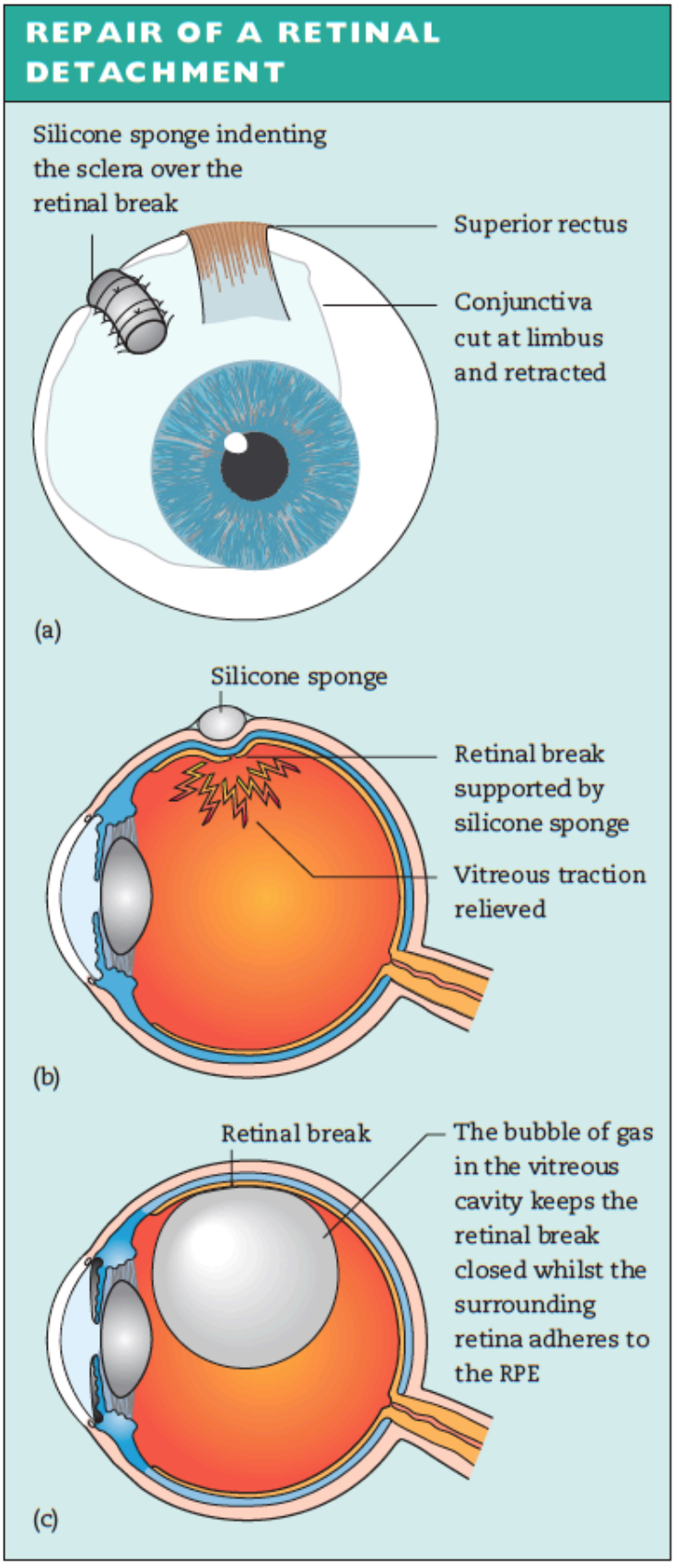


Fig. 11.10 The repair of a retinal detachment: (a) external approach, a silicone sponge has been sutured to the globe to indent the sclera over the retinal break following drainage of the sub-retinal fluid and application of cryotherapy; (b) sagittal section of the eye showing the indent formed by the silicone sponge, the retina is now reattached and traction on the retinal break by the vitreous is relieved; (c) internal approach, following removal of the vitreous gel and drainage of sub-retinal fluid an inert fluorocarbon gas has been injected into the vitreous cavity.

OTHER LECTURE-ONLY CONTENT

i.e. the following points where mentioned by the dr. during the lecture, are included in 428 handouts but are NOT mentioned at all in the given **objectives**.

DIABETIC RETINOPATHY (Refer to "Chronic Visual Loss" lecture)

EPIDEMIOLOGY

- One of the main causes of acquired blindness
- Approximately 90% of diabetic patients will have retinopathy after 20 years.

PATHOGENESIS

- DM leads to changes in almost every ocular tissue, but diabetic retinopathy is 90% of it.
- Duration of the disease is one of the most important factors affecting the incidence of diabetic retinopathy.
- Co-existent hypertension is one of the adverse effects to diabetic retinopathy
- Visual symptoms may be:
  - Gradual; with slow encroachment of the macula with a ring of exudates or microaneurysms causing chronic visual loss
  - Rapid; with vitreous hemorrhage accuring, causing acute visual loss
- Microaneurysms are tiny outpouchings of vessel walls, vascular tortuousity, venous, "dot and blot" hemorrhages
- Exudates and new blood vessels suggest the possibility of diabetic retinopathy which may be divided into the following subsections:
  - **Background diabetic retinopathy**
    benign stage characterized by microaneurysms, dot and blot hemorrhages, a few hard exudates, venular dilatation and with tortuousity (with relative absence of these changes in the macular area)
  - **Maculopathy**
    exudation at the macular resulting from microvascular leakage and lipid deposits. These lipid diffuse out from an edematous area in rings of increasing diameter when these lipids encroach on the macula, they distort the cones and cause visual distortion and loss of acuity
  - **Pre-proliferative diabetic retinopathy**
    fundal changes characterized with cotton-wool spots, deep and superficial hemorrhages, intra-retinal microvascular abnormalities, venous dilatation, looping and irregularities (by definition: no new vessels are present)
  - **Proliferative diabetic retinopathy**
    retinal ischemia in diabetics → neovascularitzation (identified by: their abnormal position, small caliber, tendency to grow forward into the vitreous cavity). Treatment is pan-retinal photocoagulation with argon laser. They may also grow in the iris (anterior segment assessment is important to note presence of rubeosis iridis). These new vessels may bleed causing vitreous and subhyaloid hemorrhages (hyaloid membrane is the vitreous membrane that separate the vitreous humor from the rest of the eye) and fibrosis may lead to development of → tractional retinal detachment and vitreous hemorrhage.
    Even quite dense vitreal hemorrhages may absorb spontaneously with good visual recover. However, if blood persisted in the vitreous, a vitrectomy is indicated and is successful in restoring reasonable clarity.

MEDIA OPACITIES

Any significant irregularity or opacity in the clear refractive media of the eye (cornea, anterior chamber, lens and vitreous) will cause blurred vision or a reduction of visual acuity. Acute visual loss may result from conditions that cause rapid changes in the transparency of these tissues. Examples:

CORNEAL EDEMA

One cause of sudden opacification of the cornea, which is commonly seen in increased IOP (angle closure glaucoma) and in chronic damage to the corneal epithelium by dystrophies or after cataract surgery (especially photoemulsification).

CORNEAL INFECTION

Any acute infection or inflammation of the cornea (e.g.herpes simplex keratitis) may mimic corneal edema. Here we have slight edema, but it doesn't cause visual loss as the problem is the infection itself.

ANTERIOR CHAMBER

If it is not transparent, like in bleeding (hyphemia) due too trauma or neovasuclariziation

LENS

Sublaxation or traumatic cataract

VITREOUS HEMORRHAGE

The symptoms are sudden, partial or complete visual loss in one eye which may be preceded by the appearance of showers of black spots in front of this eye. The fundus view is usually poor and there may be complete loss of the red reflex when the cornea and lens appear clear on examination.

Causes of vitreous hemorrhage:

Various ischemic conditions of the retina can cause new vessel formation → ramify on posterior surface of the detached vitreous → the new vessels are delicate → liable to recurrent hemorrhage. This happens in:

- diabetic retinopathy of the proliferative type
- retinal Vasculitis
- sickle cell disease
- central retinal vein occlusions
- retinal detachment
- trauma
- hypertension
- sometimes; it is accompanied with subarachnoid hemorrhage

Management:

Cause is sought and treated where possible. Ultrasound should be performed to determine whether a retinal detachment or other lesions are present (when retinal view is poor) to determine the treatment.

VISUAL PATHWAY LESIONS

OPTIC NEURITIS

- Commonest lesion
- Inflammation of the optic nerve
- Usually idiopathic but may be associated with multiple sclerosis
- Usually affect young females
- Regular features: APD + reduced visual acuity
- The optic nerve appears hyperemic and swollen\certain patients of optic neuritis may benefit from high-dose IV corticosteroids
- Recovery is usually complete

CORTICAL LESIONS

Rare condition where the patient has normal fundal examination (no optic disc atrophy in lesions behind the later geniculate nucleus) and normal pupillary reaction

- If parietal radiation is affected → lower field defect
- If temporal radiation is affected → upper field defect
- Lesion in the chiazma or retrochiazma → bilateral fields defect

FUNCTIONAL DISORDERS

The adjective “functional” is used in preference to ***hysterical*** or ***malingering*** to describe visual loss without organic basis. Diagnosis is apparent when examination results are incompatible with organic blindness.

CLINICAL ASSESSMENT OF ACUTE VISUAL LOSS

HISTORY TAKING

Important patient history questions to ask in the event of sudden loss:

- Did the visual loss ovvur ***abruptly*** or did it develop over ***hours, days or weeks***?? (onset)
- Is the visual loss ***momocular*** or ***binocular***?? (site)
- Is the visual loss ***Transient*** or ***persistent***?? (timing)
- What's the patient's age and medical condition?
- Did the patient have documented normal vision in the past?

CLINICAL EXAMINATION

How to examine:

- **Visual Acuity testing**
  The vital sign of the eye; the FIRST thing to be done
- **Confrontation visual field testing**
  As normal acuity does not assure that significant vision has not been lost, because the entire visual field – including peripheral vision – must be considered. For instance; person with homonymous hemianopia (has lost all peripheral vision on one side of both eyes) has normal visual acuity.
- **Pupillary Reactions**
  It is useful in evaluation of monocular visual loss. The absence or presence of an afferent pupillary defect (APD) is a marker for monocular visual loss. (normal pupillary reflex and fundus exam → the cause of visual loss is either cortical or functional)
- **Ophthalmoscopy**
  most important in examination
- **Pen-light Examination**
  to detect corneal disease causing acute visual loss.
- **Tonometry**
  to measure IOP (helps confirming the presence of angle-closure glaucoma).