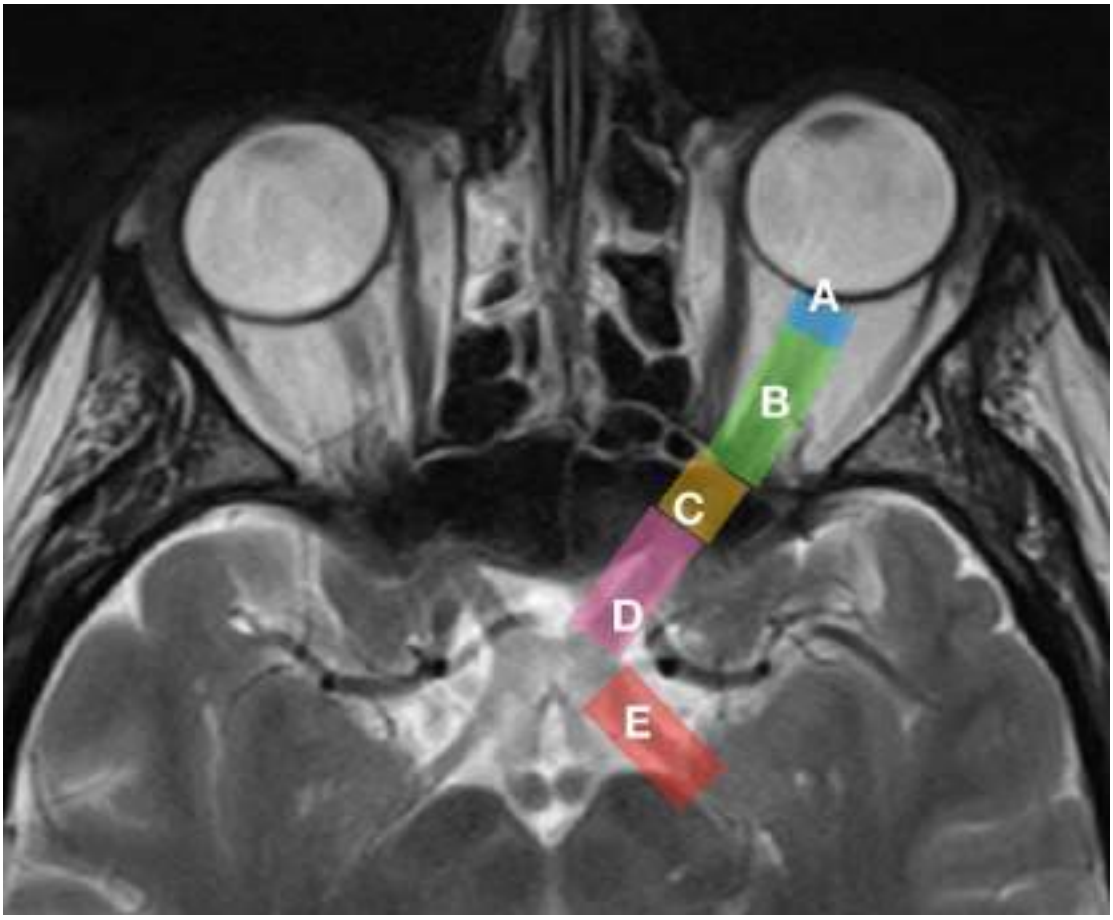
THE OPTIC NERVE
DISORDERS OF OPTIC NERVE

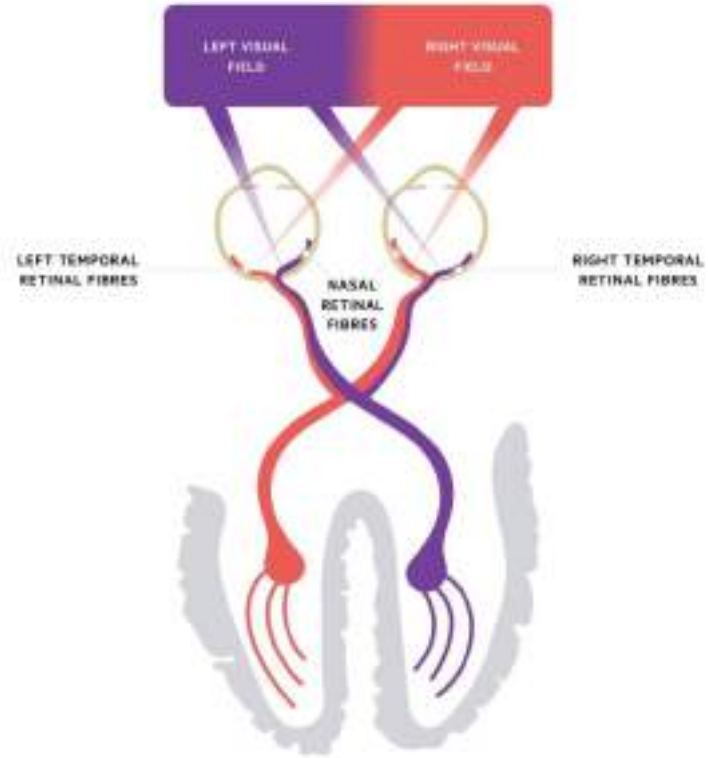
The optic nerve is a **crucial part** of the **visual system**, transmitting **visual information** from the **eye** to the **brain**. It consists of **over a million tiny nerve fibres** that **converge** at the **back of each eye**, **forming** the **optic nerve**. Its primary function is to **carry visual signals, encoded as electrical impulses, from the retina to the brain's visual processing centres,** specifically the **thalamus** and the **occipital lobe**. These signals are interpreted in the brain, allowing us to perceive and visual stimuli, such as **shapes, colours, and motion.** The optic nerve **varies** in **length** from person to person-and even **differs** between the **two eyes** of a **single** individual-but generally measures **35-55 mm.** The most efficient way to describe the structure of the nerve is by dividing it into **four main sections**: **(A) optic nerve head (or intraocular part), (B) intraorbital part, (C) intracanalicular part, and (D) intracranial part.**



VISUAL FIELD DEFECT: is a loss of part of the usual field of vision.

The visual field describes the **area that can be seen by an individual with their eyes fixed on a single point**. Our **normal field of vision is typically 135º vertically and 180º horizontally (160º for monocular vision).** The visual fields can be divided into **two** components:

- **Left visual field**: detected by the **right temporal retinal fibres (outer) and left nasal retinal fibres (inner)**
- **Right visual field**: detected by the **left temporal retinal fibres (outer) and right nasal retinal fibres (inner)**



The optic nerve is the **second cranial nerve (II**) that transmits visual information from the **retina** to the **visual cortex**. The optic nerves are paired structures that carry sensory information from **ganglion cells located within the retina** of each eye The optic nerve is made up of **>1 million myelinated axons of the retinal ganglion cells** that can sensory information from the retina to the primary visual cortex in the **occipital lobe.**

The optic nerve is divided into four components:

- **Optic nerve head**: most anterior component located in the eyeball. Corresponds to the blind spot in the visual field
- **Intraorbital part:** passes from the posterior eyeball to the start of the optic canal
- **Intracanalicular part:** passes through the optic canal within the lesser wing of the sphenoid
- **Intracranial part:** joins with the contralateral optic nerve to form the optic chiasm

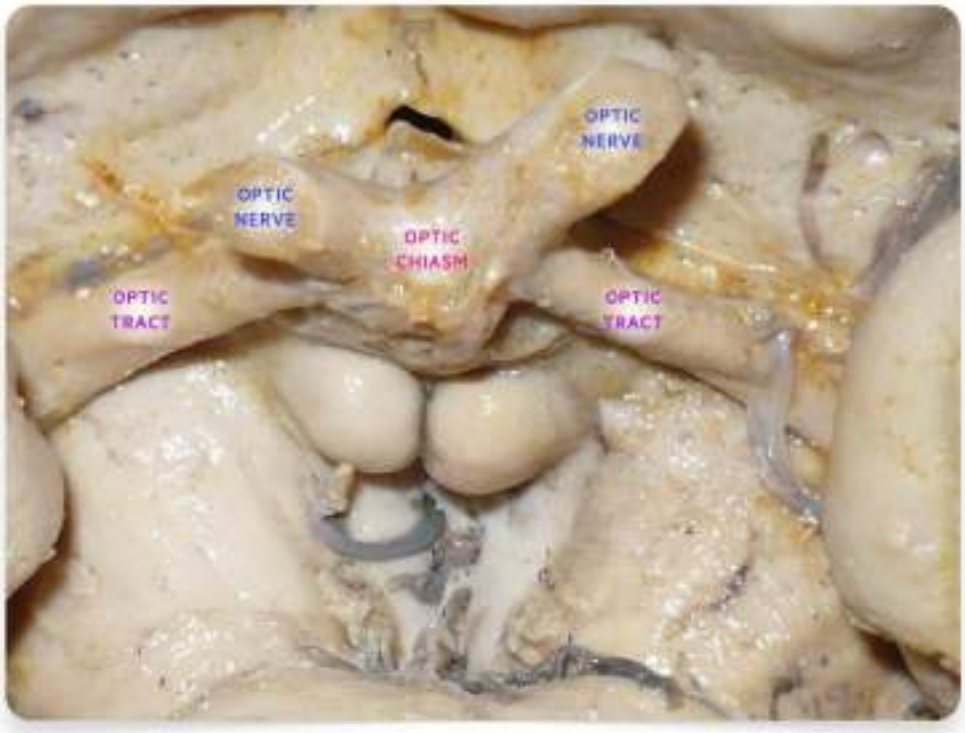
OPTIC CHIASM

The optic chiasm is an important location where retinal fibres of the optic nerve cross over. The optic chiasm has an '**X' shaped** appearance and is located inferior to the hypothalamus and superior to the sella turcica that is the boney structure that contains the pituitary gland. Therefore, it can become compressed by pituitary tumours.

The optic chiasm is an **important** location **due to the crossing over of optic nerve fibres.**

- **Nasal retinal fibres**: **cross** at the optic chiasm to join the **temporal fibres** on the other side
- **Temporal retinal fibres**: **do not cross** at the optic chiasm and joined by the **nasal fibres** from the other side

Beyond the chiasm, the nasal and temporal fibres continue **as two distinct optic tracts (right and left).** Crossing ensures information from both eyes concerning the same part of the visual field passes to the same area of the visual cortex. The **right optic tract** corresponds to the **left visual field** and the **left optic tract** corresponds to the **right visual field**.

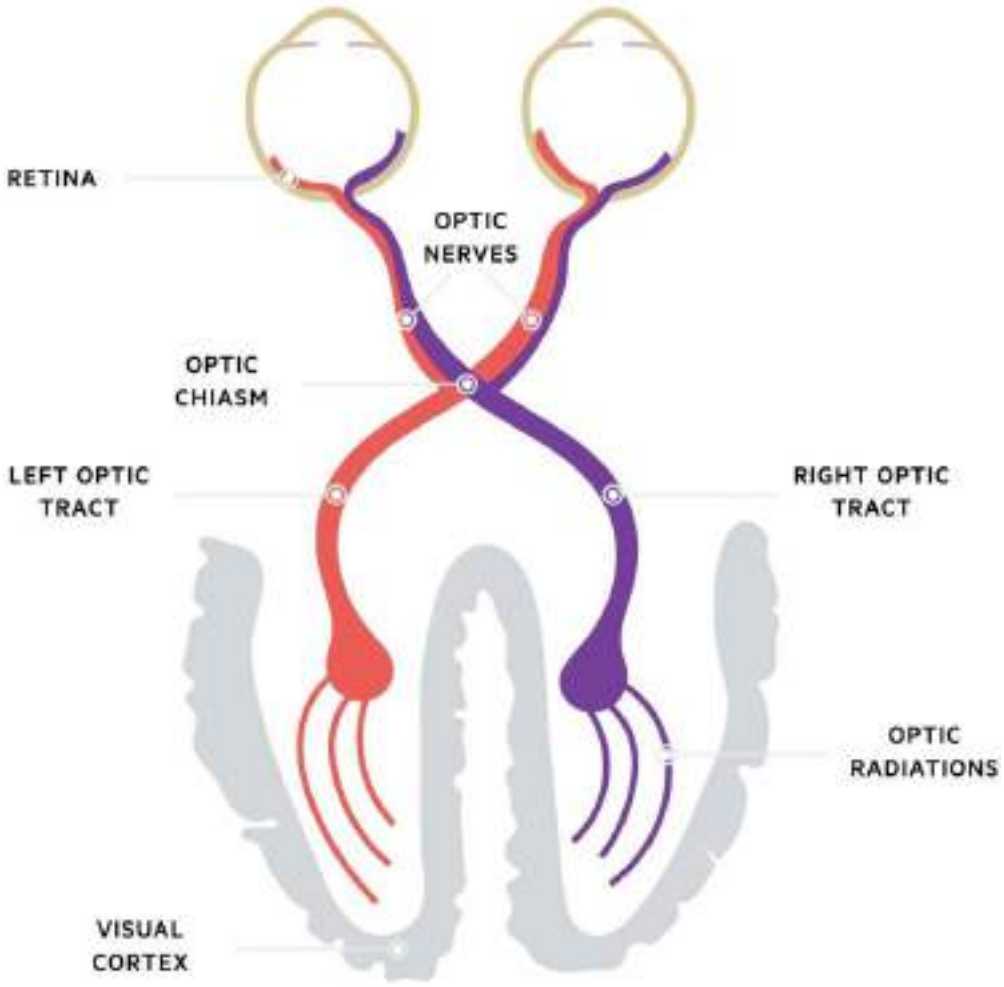


VISUAL PATHWAY

The visual pathway refers to the **structures that are responsible for perceiving, relaying, and subsequently processing visual information.**

The visual pathway describes the path of visual information from the **retina to the visual cortex in the occipital lobe.** It is divided into several components:

- **Retina**
- **Optic nerve**
- **Optic chiasm**
- **Optic tract**
- **Optic radiations**
- **Visual cortex**



RETINA

Specialized photoreceptors, known as **rods and cones**, are located within the **retina**. They are **stimulated** by light entering the eye and **transmit** information to **retinal bipolar cells**, which are a type of **interneuron that connect photoreceptors to retinal ganglion cells**. Consequently, bipolar cells transmit signals to these ganglion cells that **converge** at the optic disc. The axons of the retinal ganglion cells form the **optic nerve.**

OPTIC NERVE

Sensory information is passed along the optic nerve, through the **optic canal**, to the **optic chiasm** to join the **contralateral optic nerve**.

OPTIC CHIASM

The optic chiasm is formed by the **joining of the paired optic nerves**. **Nasal** retinal fibres **cross at the optic chiasm** to join the contralateral retinal fibres.

OPTIC TRACT

Posterior to the optic chiasm the visual pathway continues as the **right and left optic tracts.** These tracts pass **posteriorly** with the **lateral geniculate body** within the **midbrain**. This structure is considered an **extension of the thalamus.**

The **right optic tract** corresponds to the **left visual field** and the **left optic tract** corresponds to the **right visual field**.

OPTIC RADIATIONS

Neurons from **the lateral geniculate body pass dorsally** towards the **medical aspect** of the **occipital lobe**. These fibres are collectively known as the **optic radiations** or, and can be divided into **two loops:**

- **Upper (Baum's)**: runs through the **parietal lobe**. Corresponds to **inferior visual field**
- **Lower (Meyer's)**: runs through **temporal lobe**. Corresponds to **superior visual field**

VISUAL CORTEX

Fibres from the optic radiations terminate within visual nuclei in the occipital lobe that makes up the **primary visual cortex**. From the visual cortex, **neural signals** are sent to **other parts** of the brain (e.g. frontal lobe, parietal lobe, temporal lobe), to help **explain** the **visual information received.**

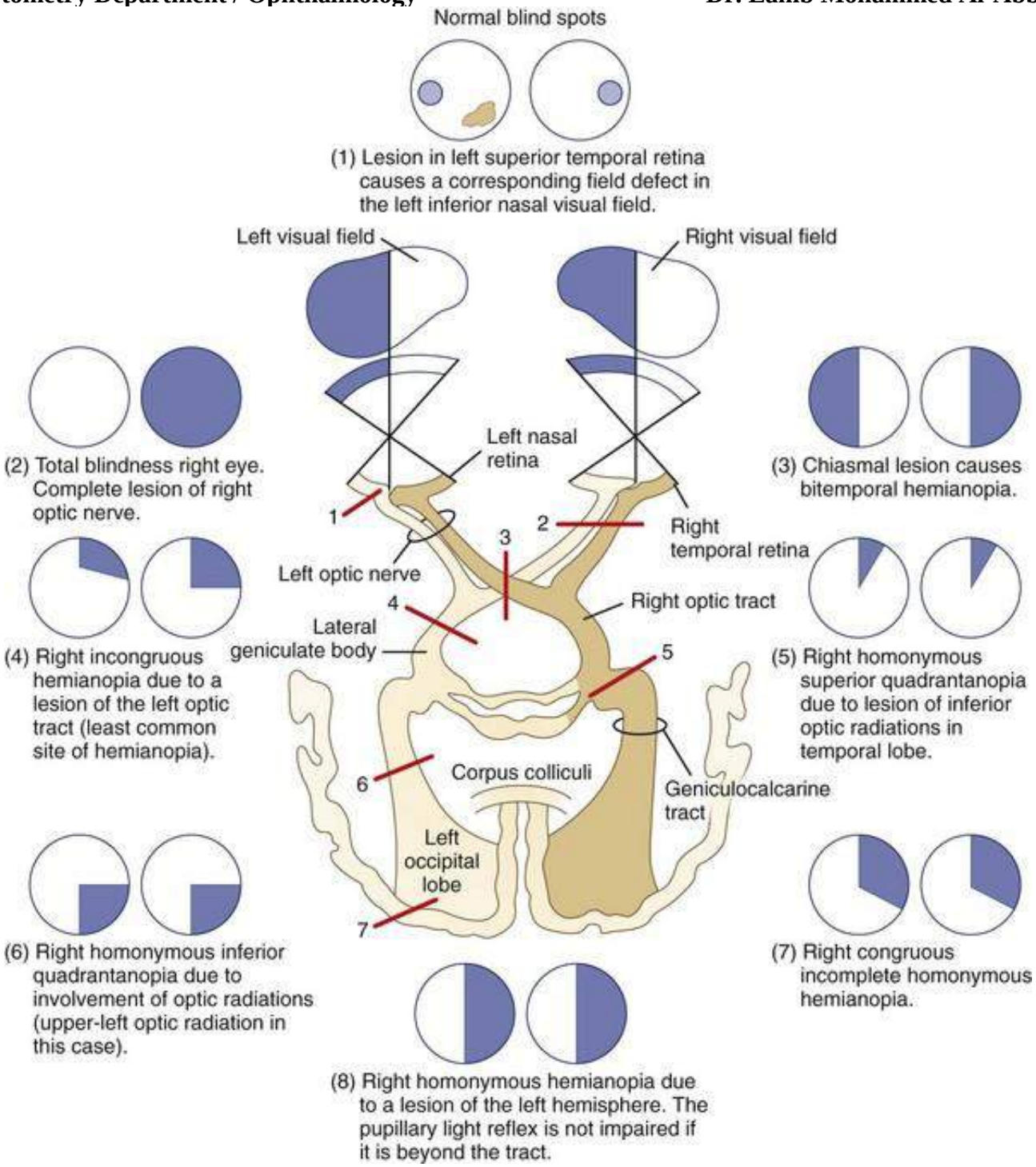

Left Homonymous Hemianopia


Left Inferior
Quadrant Hemianopia


Right Superior
Quadrant Hemianopia


Left Homonymous Hemianopia


Left Homonymous Hemianopia
with Macular Sparing



Both **diseases** and **pathological processes** of the optic nerve can induce **optic nerve damage**, **injury** and **alteration**, each of which is characterized by **structural abnormalities** typical of and particular to the **origin of the damage**, and can result in the **partial** or **complete loss** of optic nerve **function** and, ultimately, to **vision loss** . The **severity** of vision loss generally corresponds **directly** to the extent of **structural damage.**

During the **acute stage**, both **structural** changes of the optic nerve and **vision loss** can be **reversed**, however, if no symptomatic treatment is applied during this **period**, then optic neuropathy enters the **chronic stage** when structural and functional alterations **can no longer return to normal conditions.**

The origins (aetiology) of optic nerve damage may be divided into **two groups**, depending on the **location** of the given pathological process: **within or in close** proximity to the

eyeball (ocular), or **remote** from the eye, **nearer to the brain (cerebral).** The management of both medical conditions tends to be different.

Ocular medical conditions include **blood supply failures**, **local infections and local events** such as **optic nerve compression**.

Several **hereditary diseases and developmental anomalies** affect optic nerve function at the level of the eyeball.

Cerebral causes are the most remote from the eye itself but damage the **optic nerve along its pathway from the eye to the subcortical brain's visual center** (located in the thalamus). In these cases, typical causes of **damage** include **elevated intracranial pressure**, **local pathological processes along optic pathways**, common pathological factors such as **inflammation**, **autoimmune processes** and **various intoxications**. Most of these cause **secondary damage** of the optic nerve.

1. **Optic nerve tumors**

   The most commonly known tumors are **optic nerve gliomas**, optic nerve tumors can be divided into **primary tumors** which are tumors of the **nerve** and tumors **arising from the sheath.**

2. **Glaucoma**

   Glaucoma a leading cause of **blindness** is a **group of eye diseases** that **progressively** and, in many cases, **silently damages the optic nerve** causing **gradual and permanent vision loss. Generally**, glaucoma is **associated** with **increased fluid pressure within the eye**, or **intraocular pressure (IOP)**. At present, further damage to the optic nerve or **glaucomatous optic neuropathy** is **avoided by lowering IOP**, and the effectiveness of glaucoma treatment and management is measured by how well IOP is controlled. **Reducing eye-fluid pressure** in the eye through **medications (eye drops) or surgery** is a standard approach for treating glaucoma. But even with IOP **lowered** and **stabilized**, **vision loss is not definitively prevented.**

3. **Anterior ischemic optic neuropathy leading to ischemia/infarction of the nerve tissue.**

   Depending on the segment of the optic nerve affected, ischemic optic neuropathies (IONs) are divided into anterior and posterior categories. Anterior IONs (AION) are subdivided into nonarteritic and arteritic etiologies. Arteritic anterior ischemic optic neuropathy (AAION) is caused by inflammation of arteries and requires immediate therapy to prevent blindness. The most common disorder associated with arteritic AION is giant cell arteritis (GCA). Non-arteritic ischemic optic neuropathy (NAION) is more common than arteritic AION, accounting for up to 95% of ischemic optic neuropathies. This condition is the result of the occlusion (or blockage) of small blood vessels supplying blood to the optic nerve head.

4. **Optic nerve damage due to ocular (eye) trauma.**

   Traumatic optic neuropathy are subdivided into **direct** and **indirect** forms.

   Direct injury is the result of **penetrating eye trauma**, **seen frequently with orbital fractures**. Several varieties of direct optic nerve injury may be revealed as optic nerve avulsion, transection, optic nerve sheath haemorrhage, orbital haemorrhage and orbital emphysema.

5. **Hereditary degeneration of the optic nerve**

   Hereditary optic neuropathies comprise a **group of disorders which includes Leber Hereditary Optic Neuropathy (LHON)** and **Autosomal Dominant Optic Atrophy (DOA),** also known as **Kjer disease.** Major lesions can be seen in **papillomacular bundles which lead to the loss of central vision**. This results in **massive retinal ganglion cells**

(RGC) loss, seen clearly in the central region of the retina. More devastating, however, is that in most cases hereditary optic neuropathies are **progressive**. The **pattern of transmission of the genetic deficit** is **employed** for the classification of inherited optic neuropathies.

6. Inflammation of the optic nerve (Optic Neuritis, Neuromyelitis Optica and Neuromyelitis Optica Spectrum Disorders (NMOSD)

Optic neuritis is an **inflammatory condition affecting the optic nerve**. It is **idiopathic** (or of an **unknown cause**) in most cases, but carries a **strong association** with **multiple sclerosis (MS**), where the **demyelinating process** is the **main cause** for developed **optic nerve damage**. In **rare** cases, other etiologies like **infectious, inflammatory, and other pathological immunological responses** play a role in this medical condition, typically occurred at **age 20-50**, of which approximately **75% are female.**

Neuromyelitis Optica (NMO) must be distinguished from multiple sclerosis as it is a completely different type of **autoimmune damage** leading to the **appearance of specific antibodies against the astrocytic water channel aquaporin-4 (AQP4).**

7. Developmental anomalies (optic nerve hypoplasia, colobomas, retarded myelination, congenital swelling of the optic disc (pseudo papilloedema).

Optic nerve hypoplasia (ONH) is characterised by an **abnormally small** optic nerve head frequently seen together with head cupping. **Loss of vision** occurs because the **number of axons** in the optic nerve are **limited** due to **apoptosis** during the **development stages** of the **visual system.** ONH is a **unilateral or bilateral non-progressive underdevelopment of the optic nerve, and is considered to be a non-local syndrome rather than a more diffuse condition.** It can be divided into three clinical subtypes: **a) Optic Nerve Hypoplasia Simplex**; **b) Septo-optic dysplasia** (de Morsier's syndrome); and **c) Septo-Optic-Pituitary Dysplasia.** Septo-optic dysplasia is considered to be a combination of ONH, pituitary gland hypoplasia and midline brain abnormalities.

8. Autoimmune optic neuropathies

including **single isolated optic neuritis (SION), relapsing isolated optic neuritis (RION), chronic relapsing inflammatory optic neuropathy (CRION),** the **neuromyelitis optica spectrum disorder (NMO), multiple sclerosis associated optic neuritis (MSON) and unclassified forms (UCON).**

9. Cerebral vasular pathology

Cerebral vasular pathology is **associated with diabetes, hypertension, and hypercholesterolaemia.**

10. Toxic or Metabolic

nutritional optic neuropathy, **toxic amblyopia, tobacco, methyl alcohol.**

11. Bacterial and viral infections

Tuberculosis, Syphilis, Lyme disease, meningitis. Viral infections (e.g., encephalitis, measles, mumps, rubella. chickenpox, herpes zoster, mononudeosis). **Respiratory infections (e.g., mycoplasma pneumonia and other common upper respiratory tract infections**

RETINAL DETACHMENT

Retinal Detachment

Definition

Retinal detachment refers to the **separation of the neurosensory retina from the underlying retinal pigment epithelium**. This can be classified into **four types**:

❖ **Rhegmatogenous** retinal detachment results from a **tear**, i.e., a break in the retina.

❖ **Tractional** retinal detachment results from **traction**, i.e., from vitreous strands that exert tensile forces on the retina .

❖ **Exudative** retinal detachment is caused by **fluid**. Blood, lipids, or serous fluid accumulates **between** the neurosensory retina and the retinal pigment epithelium. **Coats' disease** is a typical example.

❖ **Tumour-related** retinal detachment.

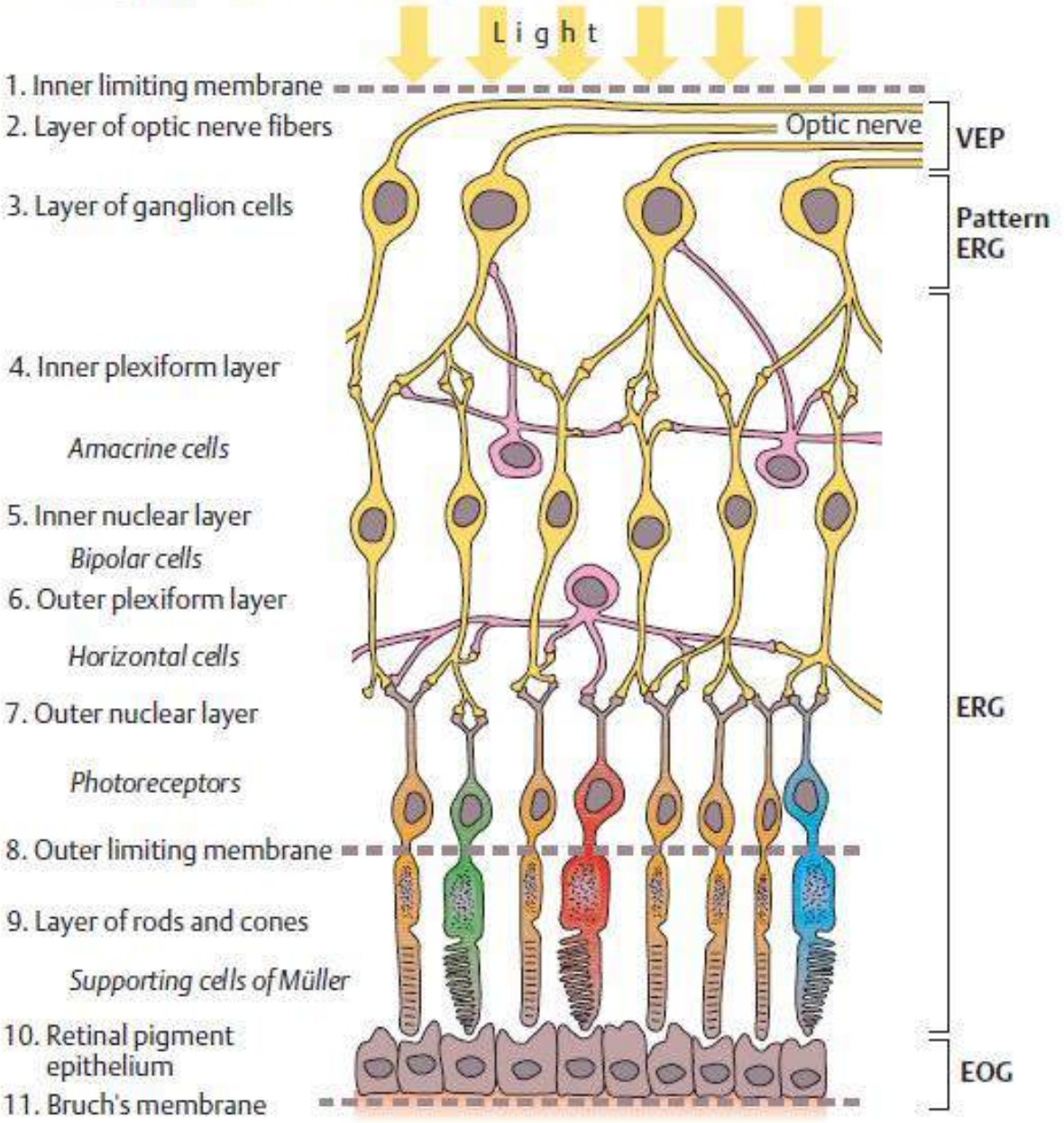
Primary retinal detachment usually results from a **tear**. In **rare** cases, **secondary retinal detachment** may also result from a tear due to other **disorders or injuries**. **Combinations** of both are also **possible but rare**.

What are the symptoms of a retinal detachment?

The following symptoms can be the first signs of a retinal detachment:

- floaters
- flashing lights
- a dark shadow in your vision blurred vision.

Histology and function of the layers of the retina.



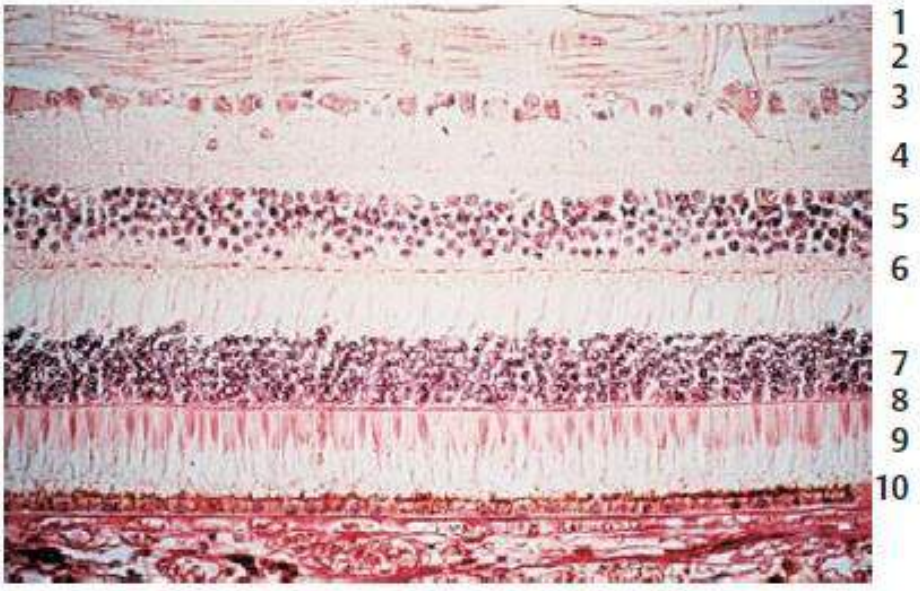


Fig. 12.2

Rhegmatogenous retinal detachment is caused by **breaks, holes or tears** in the **retina** which **allow** the **fluid** to **pass** from **vitreous cavity** to **subretinal area** between the retina and retinal pigment epithelial causing the sensory retina to be separated from the underlying retinal pigment epithelial layer. **Rhegmatogenous retinal detachment** (most frequent form): Approximately **7%** of all adults have retinal breaks. The incidence of this finding increases with **advanced age**. The peak incidence is between the **fifth and seventh decades of life**.

Exudative, tractional, and tumour-related retinal detachments are encountered far **less frequently.**

Rhegmatogenous retinal detachment : This disorder develops from an **existing break in the retina.** Usually this break is in the **peripheral** retina, **rarely** in the **macula**. Two types of breaks are distinguished:

- **Round breaks:** A portion of the retina has been completely torn out due to a **posterior vitreous detachment.**
- **Horseshoe tears**: The retina is **only slightly torn**.

Not every retinal break leads to retinal detachment. This will occur only where the **liquified vitreous body separates**, and **vitreous humor penetrates beneath the retina through the tear.**

The retinal detachment **occurs** when the **forces of adhesion can no longer with stand this process**. **Tractional forces** (tensile forces) of the **vitreous body** (usually vitreous strands) can also cause retinal detachment. In this and every other type of retinal detachment, there is a dynamic interplay of tractional and adhesive forces. Whether the **retina will detach** depends on which of **these forces is stronger**.

Risk factors for Rhegmatogenous Retinal Detachment

1- Posterior Vitreous Detachment

Separation of vitreous from the retinal occur normally in **older patients** and there are factors that causing the vitreous to **detach in younger age** like in **myopic patient, trauma, intraocular inflammation and diabetic retinopathy.** Sometimes with posterior vitreous detachment there will be traction on the retina with tear formation causing retinal detachment.

2- High Myopia

Myopic patient will have high incidence of retinal detachment because of **high incidence of lattice degeneration, earlier posterior vitreous detachment** and **more peripheral retinal lesions.**

3- Intraocular Surgeries

Intraocular surgeries can increase the risk of retinal detachment especially when associated with vitreous loss. Surgeries that can cause detachment are cataract extraction, keratoplasty or corneal graft surgery and also can occur after YAG laser capsulotomy.

4- Peripheral Retinal Lesions

There are specific lesions on the **peripherally** of the retinal which might **increase** the risk of retinal detachment.

5- Trauma

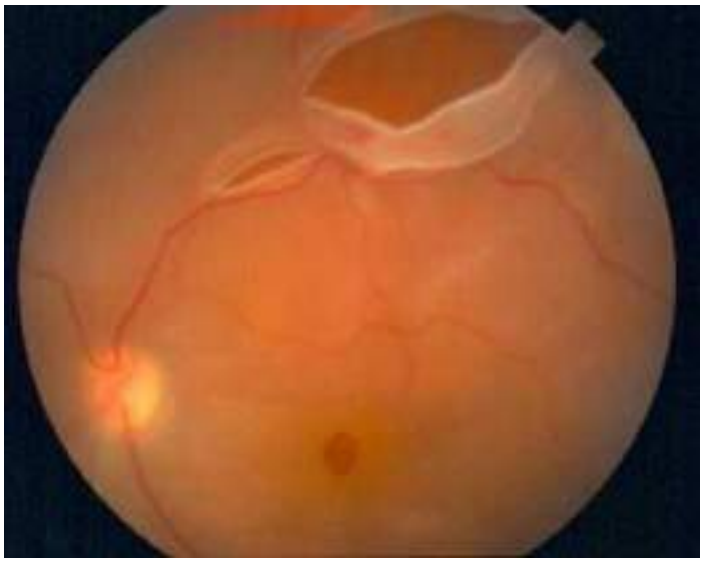
Trauma can cause detachment by creating **peripheral tears or breaks after** blunt trauma to the eyes.

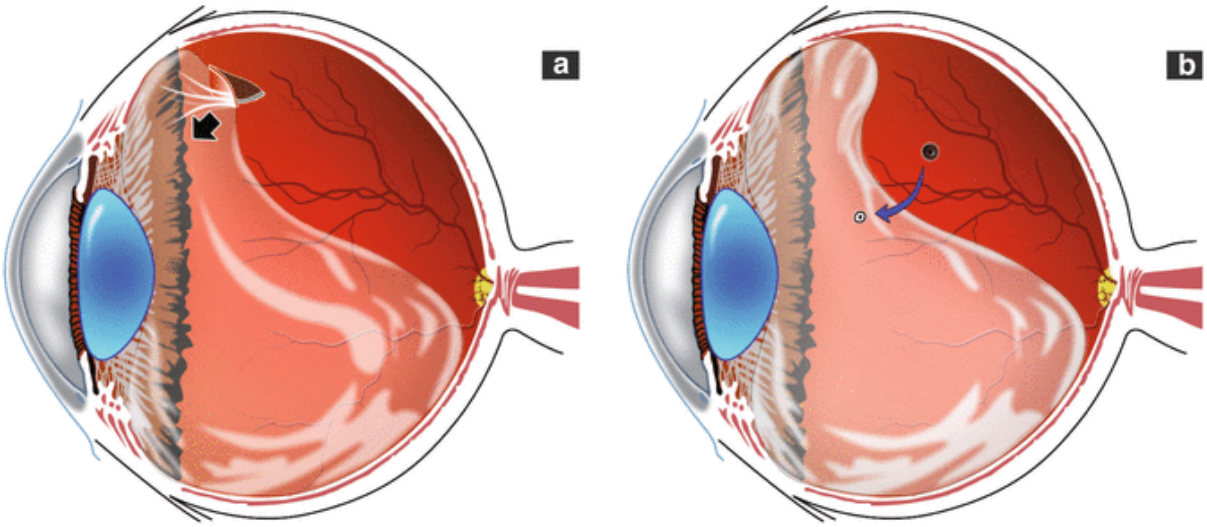
6- Intraocular Inflammation and Infection

Intraocular inflammation or infection can cause retinal detachment. This inflammation will cause **necrosis and death of cells in the retina which will become thin.**

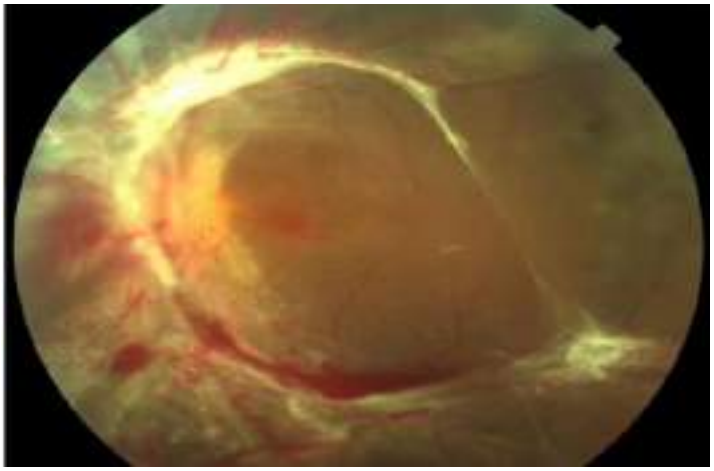
7- Collagen Diseases

Collagen diseases like Marfan's syndrome and Ehlers-Danlos syndrome can predispose to retinal detachment because collagen is an important component of the retina and in these syndromes.

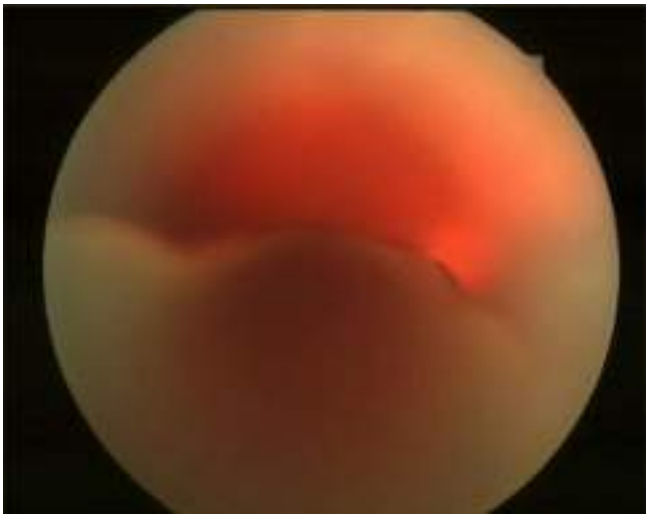




Tractional retinal detachment : This develops from the tensile forces exerted on the retina by pre retinal fibro vascular strands, especially in proliferative retinal diseases such as diabetic retinopathy.



Exudative retinal detachment : The primary cause of this type is the break-down of the inner or outer blood – retina barrier, usually as a result of a vascular disorder such as Coats' disease. Sub retinal fluid with or without hard exudate accumulates between the neurosensory retina and the retinal pigment epithelium.



Tumour-related retinal detachment : Either the transudate from the tumour vasculature or the mass of the tumour separates the retina from its underlying tissue.

Diagnostic considerations:

The lesion is diagnosed by stereoscopic examination of the fundus with the pupil dilated. The detached retina will be white and will lose its transparency. Ophthalmoscopy will reveal a bullous retinal detachment; in **rhegmatogenous retinal detachment**, a bright red retinal break will also be visible. The tears in rhegmatogenous retinal detachment usually occur in the superior half of the retina in a region of equatorial degeneration. In **tractional retinal detachment**, the bullous detachment will be accompanied by pre retinal gray strands. In **exudative retinal detachment**, one will observe the typical picture of serous detachment; the exudative retinal detachment will generally be accompanied by massive fatty deposits and often by intra retinal bleeding. The **tumor-related retinal detachment** (as can occur with a malignant melanoma) either leads to secondary retinal detachment over the tumor or at some distance from the tumor in the inferior peripheral retina. Ultrasound studies can help confirm the diagnosis where retinal findings are equivocal or a tumor is suspected.

TREATMENT FOR RETINAL DETACHMENT

Treatment for retinal detachment depends on a few factors including how much retina is detached and where in the eye the detached is located. The treatment options are **laser surgery**, cryotheray, or eye surgery to fix any tears or breaks in the retina. The eye surgeon (ophthalmologist) will discuss the kind of surgery they recommend, its risks, and its benefits with you.

Sometimes, the recommened treatment involves using more than one of these treatments at the same time.

1. Laser surgery (photocoagulation)

In laser surgery treatment, a medical laser is focused on the retina. This laser makes small burns around the retina hole or tear and small surrounding retinal detachment. Small scars are created because of the burns and this helps to stick the retina down and prevent the detachment from becoming larger.

The procedure involves putting anaesthetic eye drops and focusing the laser through the pupil of the eye. A flashing light may be visible to the patient when the laser energy is delivered.

2. Cryopexy (freezing)

Cryopexy is another way of treating retinal hole or tear. In this procedure, a freezing probe is applied to the outer surface of your eye directly over the tear. Similar to photocoagulation, the freezing causes a scar that helps in holding the retina to the eye wall.

3. Surgery

If your retina is significantly detached, eye surgery in a hospital may be required to situate the retina back to its proper place

For a short time, you may need to stay in the hospital after the surgery. It can be a few weeks' time before your vision starts to improve after the surgery.

There are 3 main types of surgery to fix a detached retina:

Pneumatic retinopexy:

A small bubble is injected into the eye. The purpose of the bubble is to push your retina back into place so that laser or cryopexy can be effectively applied to repair any holes or tears in the retina. The bubble (if positioned correctly) can push the area of the retina with the retinal hole against the eyewall. This stops more fluid from flowing into the area behind the retina and making the detachment larger. This procedure may be possible in the ophthalmologist clinic. The patient may be required to hold their head in a certain position for several days to keep the bubble's position in the right place. During a pneumatic retinopexy, the eye is numb using anaesthetic drops. A fine needle is inserted into the vitreous cavity to remove a small amount of fluid, followed by injection of an air bubble. Once the retina is flat, the surgeon will apply laser or freezing treatment to repair holes or tears in the retina. The air bubble will be visible in your peripheral vision after the surgery for some time, and will disappear on its own.

Scleral buckle:

The white of the eye is known as the sclera. During scleral buckle surgery, a tiny, flexible silicone band is inserted onto the sclera. The function of the band is to help your retina reattach by gently intending the sclera toward the detached retina. The buckle is usually left in place but may need to be removed later on. Scleral buckling surgery is performed in a hospital and the eye is anaesthetised fully. The surgeon will also apply laser or freezing treatment to repair holes or tears in the retina.

Vitrectomy:

Vitrectomy surgery involves careful removal of vitreous from the eye. The surgeon will also apply laser or freezing treatment to repair holes or tears in the retina. A gas or replacement fluid like silicone oil may be injected in place of the vitreous. If silicone oil is used, it may need to be removed a few months later.

THE OCULAR INJURIES

Ocular injuries are an important workplace hazard that can lead to vision loss, decreased functioning.

CLASSIFICATION OF OCULAR INJURIES BY MECHANISM OF INJURY

❖ **Mechanical injuries:**

– Eyelid injuries.

Eyelid injuries can occur in practically every facial injury. The following types warrant special mention:

❖ **Eyelid lacerations** with involvement of the **eyelid margin**.

❖ **Avulsions** of the **eyelid** in the **medial canthus** with avulsion of the **lacrimal canaliculus.**

Treatment:

Surgical repair of eyelid injuries, especially lacerations with involvement of the eyelid margin, should be performed with care.

Laceration of the upper and lower eyelids with avulsion of the lacrimal system.

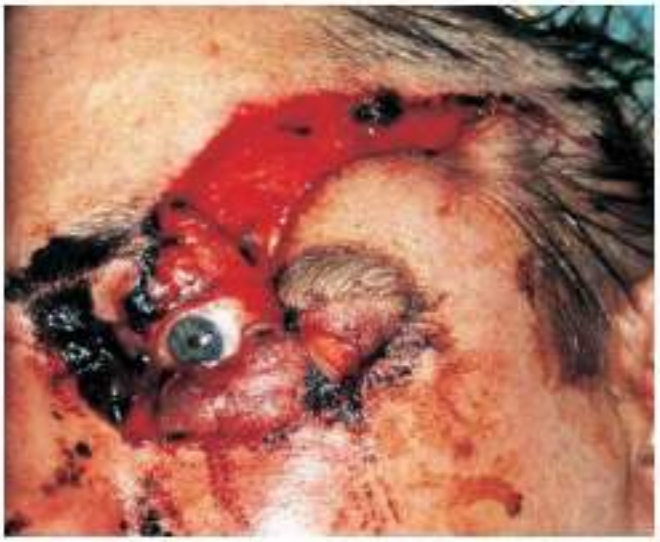
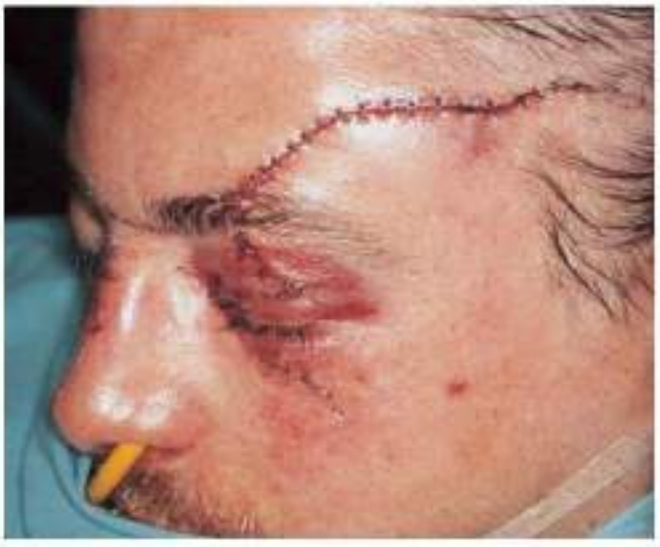


Fig. 18.1 **a** The injury has exposed the cornea. The patient is unable to close the eye, and the cornea and conjunctiva can no longer be moistened.



b Postoperative findings.

– Injuries to the lacrimal system.

Lacerations and tears in the **medial canthus** (such as dog bites or glass splinters) can **divide** the lacrimal duct. Obliteration of the **punctum** and **lacrimal canaliculus** is usually the result of a **burn or chemical** injury. Injury to the **lacrimal sac or lacrimal gland** usually occurs in conjunction with severe **trauma** (such as a kick from a horse or a traffic accident).

Surgical treatment of avulsion of the eyelid with avulsion of the lacrimal system (bicanicular ring intubation).

Treatment:

Lacrimal system injuries are repaired under an operating microscope. A ring-shaped silicone stent is advanced into the canaliculus using a special sound. The silicone stent remains in situ for three to four months and is then removed.

Surgical repair of eyelid and lacrimal system injuries must be performed by an ophthalmologist.

– Conjunctival laceration.

Due to its **exposed position**, **thinness**, and **mobility**, the conjunctiva is susceptible to lacerations, which are usually associated with sub-conjunctival haemorrhage. Conjunctival lacerations most commonly occur as a result of penetrating wounds (such as from bending over a spiked-leaf palm tree or from a branch that snaps back on to the eye).

The patient experiences a foreign body sensation. Usually this will be rather mild. Examination will reveal circumscribed conjunctival reddening or sub-conjunctival hemorrhage in the injured area.

Treatment:

Minor conjunctival injuries do not require treatment as the conjunctiva heals quickly. **Larger** lacerations with mobile edges are approximated with absorbable sutures.

– Foreign body in the cornea and conjunctiva.

Foreign bodies on the **cornea and conjunctiva** are the **commonest** ocular emergency encountered by general practitioners and ophthalmologists. **Airborne foreign bodies** and **metal splinters** from grinding or cutting disks in particular often become **lodged** in the **conjunctiva** or **cornea** or burn their way into the **tissue**.

– Corneal Erosion

This disorder follows initial **trauma** to the surface cornea, such as the fingernail of a child, a spiked-leaf palm tree, or a branch that snaps back on to the eye. Properly treated, this epithelial **defect usually heals within a short time, i.e., 24 to 48 hours** depending on the **size** of the defect.

Blunt Ocular Trauma (Ocular Contusion)

Ocular contusions resulting from blunt trauma such as a fist, ball, champagne cork, stone, falling on the eye, or a cow's horn are very common. Significant deformation of the globe can result where the diameter of the blunt object is less than that of the bony structures of the orbit.

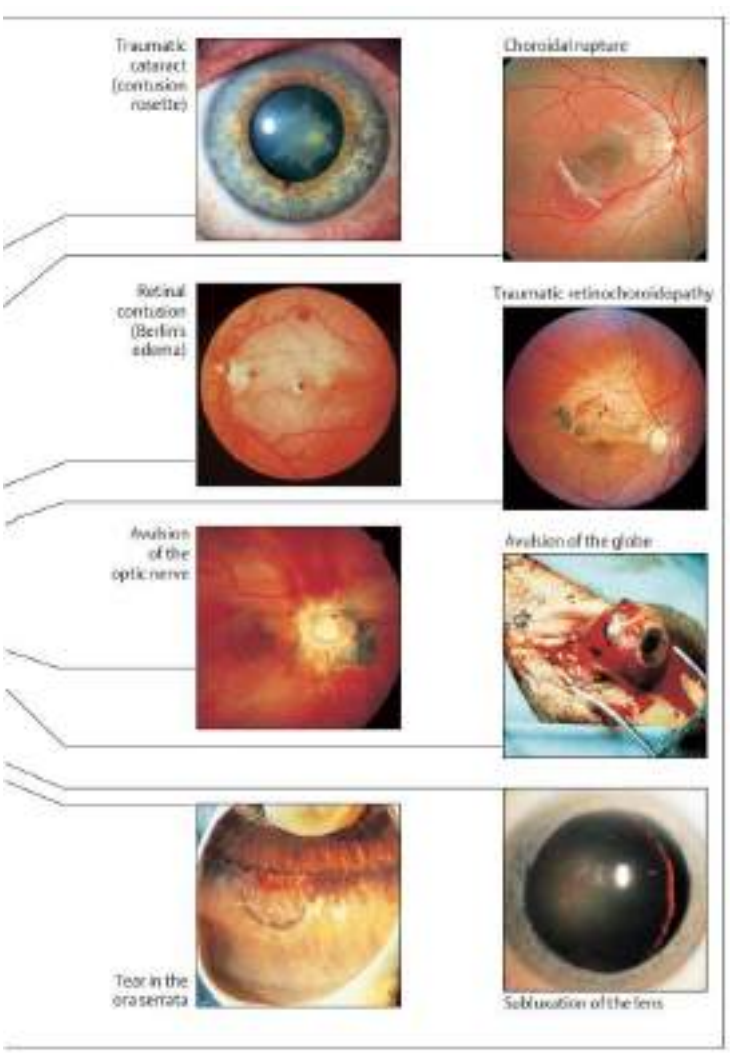
Deformation exerts significant traction on intraocular structures and can cause them to tear. Often there will be blood in the anterior chamber, which will initially prevent the examiner from evaluating the more posterior intraocular structures.

Do not administer medications that act on the pupil as there is a risk of irreversible mydriasis from a sphincter tear, and pupillary movements increase the risk of subsequent bleeding. The posterior intraocular structures should only be thoroughly examined in mydriasis to deter-mine the extent of injury after a week to ten days.

Treatment:

This involves immobilizing the eye initially, to allow intra ocular blood to settle.

Subsequent bleeding three or four days after the injury is common.



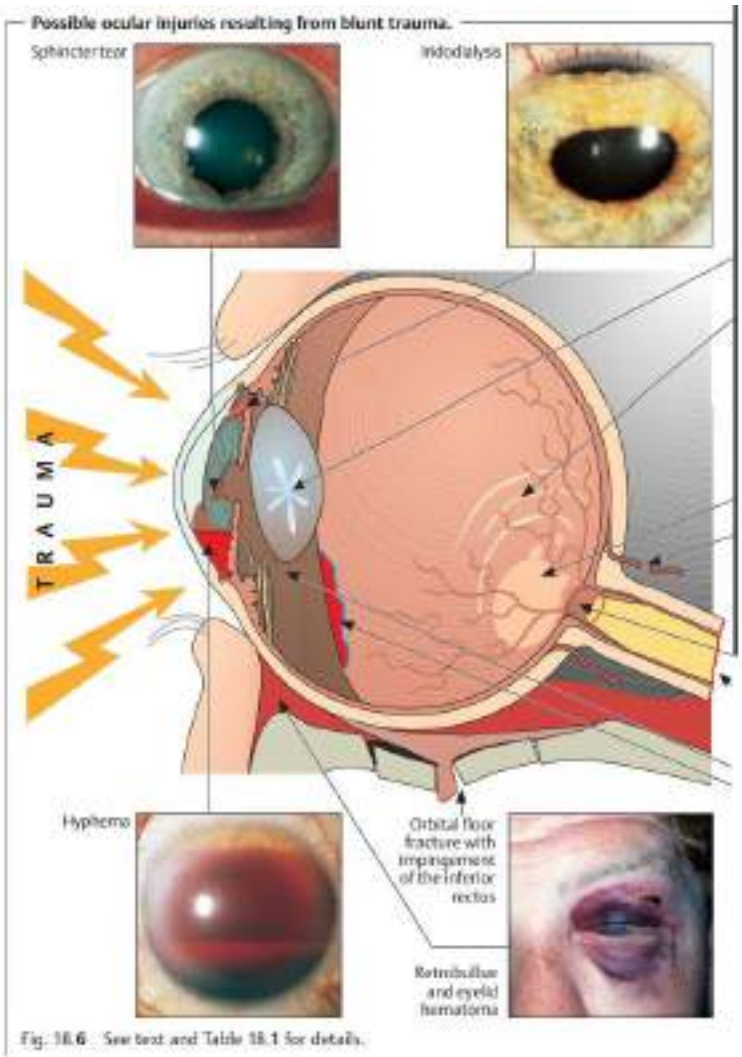


Fig. 18.6 See text and Table 18.1 for details.

– Blow-out fractures of the orbit result from blunt trauma. Blunt objects of small diameter, such as a fist, tennis ball, or baseball, can compress the contents of the orbit so severely that orbital wall fractures. This fracture usually occurs where the bone is thinnest, along the paper-thin floor of the orbit over the maxillary sinus. The ring-shaped bony orbital rim usually remains intact. The fracture can result in protrusion and impingement of orbital fat and the inferior rectus and its sheaths in the frature gap. Where the medial ethmoid wall fractures instead of the orbital floor, emphysema in the eyelids will result. The more severe the contusion, the more severe the intraocular injuries and resulting visual impairment will be. Impingement of the inferior rectus can result in **diplopia**, especially in upward gaze. Initially, the diplopia may go unnoticed when the eye is still swollen shut. A large bone defect may result in displacement of larger portions of the contents of the orbital cavity. The eye may recede into the orbit (**enophthalmos**) and the **palpebral fissure may narrow**. Injury to the infraor-bital nerve, which courses along the floor of the orbit, may result. This can cause **hypesthesia of the facial skin**.

Crepitus upon palpation during examination of the eyelid swelling is a sign of emphysema due to collapse of the ethmoidal air cells. The crepitus is caused by air entering the orbit from the paranasal sinuses. The patient should refrain from blowing his or her nose for the next four or five days to avoid forcing air or germs into the orbit. Radiographs should be obtained and an ear, nose, and throat specialist consulted to help determine the **exact location of the fracture.** CT studies are more precise and may be indicated to evaluate difficult cases.

Tissue displaced into the maxillary sinus will resemble a hanging drop of water in the CT image.

Treatment:

Surgery to restore normal anatomy and the integrity of the orbit should be performed within ten days. This minimizes the risk of irreversible damage from scarring of the impinged inferior rectus. Where treatment is prompt, the prognosis is good.

Tetanus prophylaxis and treatment with antibiotics are crucial.

– Open-Globe Injuries

The **most devastating forms** of ocular **trauma**. They are caused by **sharp** objects that **penetrate** the **cornea and sclera**. A distinction is made between penetration with and without an intraocular **foreign body.**

Depending on the severity of the injury, the following **diagnostic signs** will be present in an open-globe injury:

- The **anterior chamber** will be **shallow or absent**.
- The **pupil** will be displaced **toward** the penetration site.
- **Swelling** of the **lens** will be present (traumatic cataract).
- There will be **bleeding** in the anterior chamber and vitreous body.
- **Hypotonia** will be present.
- **Radiographs** in two planes to determine whether there is a **foreign bod**y in the eye.
- **CT studies**, that permit precise **localization of the foreign body**.

Treatment:

First aid.

Where penetrating is suspected, a **sterile bandage should** be applied and the patient referred to an eye clinic for treatment.

Surgery.

Surgical treatment of penetrating injuries must include suturing the globe and reconstructing the anterior chamber. Any **extruded** intraocular tissue (such as the iris) **must be removed**. Intraocular foreign bodies should be removed when the wound is repaired.

Penetrating injury.

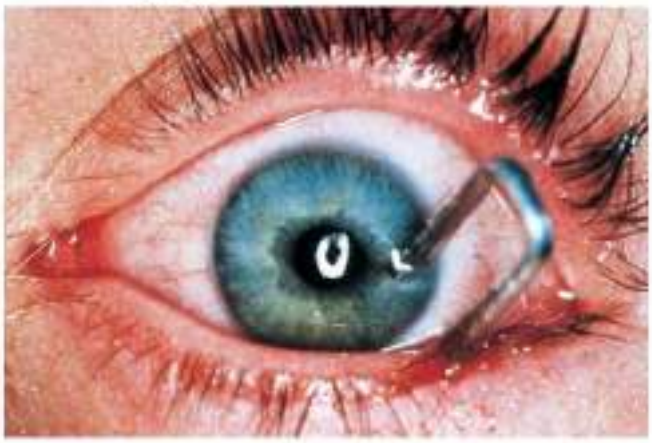


Fig. 18.7 Open-globe injury from a staple involving the cornea, iris, lens, sclera, and retina.

Impalement Injuries of the Orbit

Impalement injuries occur most frequently in situations such as these:

- ❖ Children may fall on pencils held in their hands.
- ❖ Injuries may result from the actions of other persons (such as arrows or darts).
- ❖ A knife may slip while a butcher is removing a bone from a cut of meat.

Impalement injury of the right orbit.

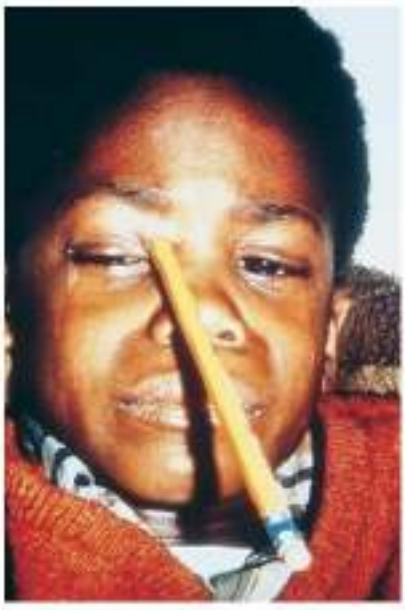


Fig. 18.9 Orbital injury without injury to the globe following a fall on a pencil held in the patient's hand. (Photograph courtesy of Prof. W. D. Green, M.D., Baltimore, Maryland.)

❖ Chemical injuries

Chemical injuries can be caused by a **variety of substances such as acids, alkalis, detergents, solvents, adhesives, and irritants like tear gas. Severity** may **range from slight irritation of the eye to total blindness.**

Chemical injuries are among the **most dangerous ocular injuries**. First aid at the site of the accident is **crucial** to **minimize** the risk of **severe such as blindness**.

As a general rule, acid burns are **less** dangerous than alkali burns.

Acids differ from alkalis in that they **cause immediate coagulation necrosis** in the **superficial** tissue. This has the effect of **preventing** the acid from penetrating **deeper** so that the burn is effectively a **self-limiting** process. **However**, some acids penetrate **deeply** like alkalis and cause similarly **severe injuries**. **Concentrated sulfuric acid** (such as from an exploding car battery) draws water out of tissue and simultaneously develops **intense heat** that affects every layer of the eye. **Hydrofluoric acid and nitric acid** have a **similar** penetrating effect.

Alkalis differ from most acids in that they can **penetrate** by **hydrolysing structural proteins** and **dissolving cells**. This is referred to as liquefy active **necrosis**. They then cause severe intraocular damage by **alkalizing the aqueous humor**.

Treatment:

First aid rendered at the scene of the accident often decides the fate of the eye. The first few seconds and minutes and resolute action by per-sons at the scene are crucial. Immediate copious irrigation of the eye may be performed with any watery solution of neutral pH, such as tap water, mineral water, soft drinks, coffee, tea, or similar liquids. Milk should be avoided as it the increases penetration of the burn by opening the epithelial barrier. A second person must rigorously restrain the severe blepharospasm to allow effective irrigation. A topical anesthetic to relieve the blepharospasm will rarely be available at the scene of the accident. Coarse particles (such as lime particles in a lime injury) should be flushed and removed from the eye. Only after these actions have been taken should the patient be brought to an ophthalmologist or eye clinic.

Chronology of treatment of chemical injuries:

❖ First aid at the scene of the accident

– Restrain blepharospasm by rigorously holding the eyelids open.

– Irrigate the eye within seconds of the injury using tap water, mineral water, soft drinks, coffee, tea, or similar liquids. Carefully remove coarse particles from the conjunctival sac.

– Notify the rescue squad at the same time.

– Transport the patient to the nearest ophthalmologist or eye clinic.

❖ Treatment by the ophthalmologist or at the eye clinic:

– Administer topical anesthesia to relieve pain and neutralize blepharospasm.

– With the upper and lower eyelids fully everted, carefully remove small particles such as residual lime from the superior and inferior conjuncti-val fornices under a microscope using a moist cotton swab.

– Flush the eye with a buffer solution. Long-term irrigation using an irri-gating contact lens may be indicated (the lens is connected to a cannula to irrigate the eye with a constant stream of liquid).

– Initiate systemic pain therapy if indicated.

❖ **Additional treatment on the ward in an eye clinic:**

The following therapeutic measures for severe chemical injuries are usu-ally performed on the ward:

– Continue irrigation.

– Initiate topical cortisone therapy (dexamethasone 0.1% eyedrops and prednisolone 1% eyedrops).

– Administer subconjunctival steroids.

– Immobilize the pupil with atropine 1% eyedrops or scopolamine 0.25% eyedrops twice daily.

– Administer anti-inflammatory agents (two oral doses of 100 mg indomethacin or diclofenac) or 50 – 200 mg systemic prednisolone.

– Administer oral and topical vitamin C to neutralize cytotoxic radicals.

– Administer 500 mg of oral acetazolamide (Diamox) to reduce intraocu-lar pressure as prophylaxis against secondary glaucoma.

– Administer hyaluronic acid for corneal care to promote re-epithelial-ization and stabilize the physiologic barrier.

– Administer topical antibiotic eyedrops.

– Debride necrotic conjunctival and corneal tissue and make radial inci-sions in the conjunctiva (Passow's method) to drain the subconjunctival edema.

❖ **Additional surgical treatment in the presence of impaired wound healing following extremely severe chemical injuries:**

– A *conjunctival and limbal transplantation* (stem cell transfer) can replace lost stem cells that are important for corneal healing. This will allow re-epithelialization.

– Where the cornea does not heal, cyanoacrylate glue can be used to attach a *hard contact lens* (artificial epithelium) to promote healing.

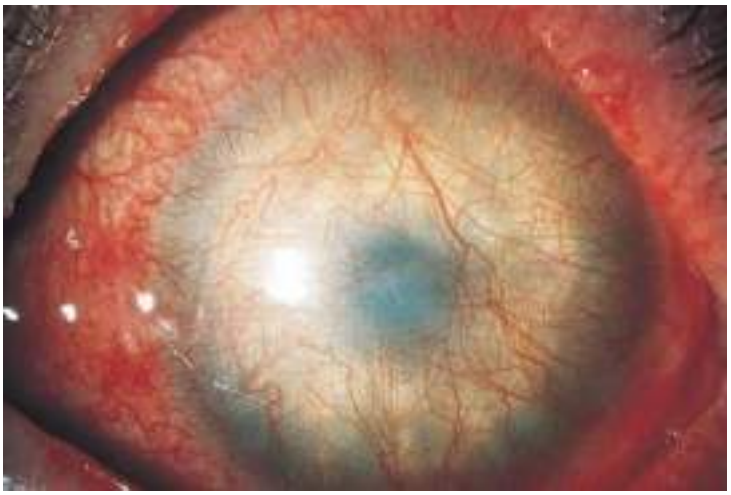
– A Tenon's capsule plasty (mobilization and advancement of a flap of subconjunctival tissue of Tenon's capsule to cover defects) can help to eliminate conjunctival and scleral defects.

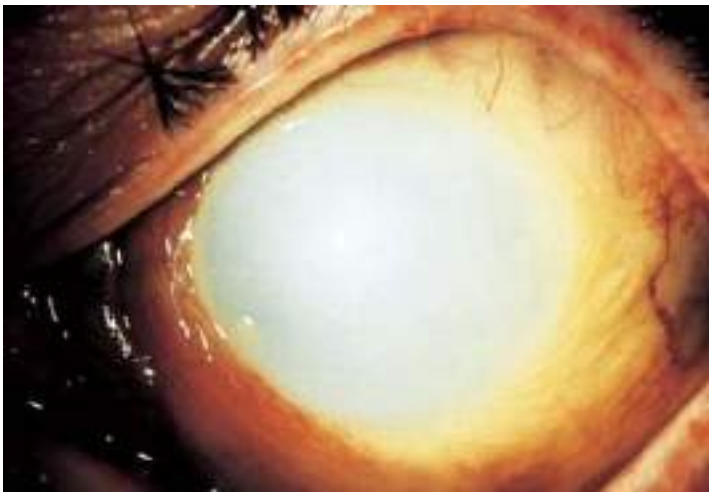
❖ **Late surgical treatment after the eye has stabilized:**

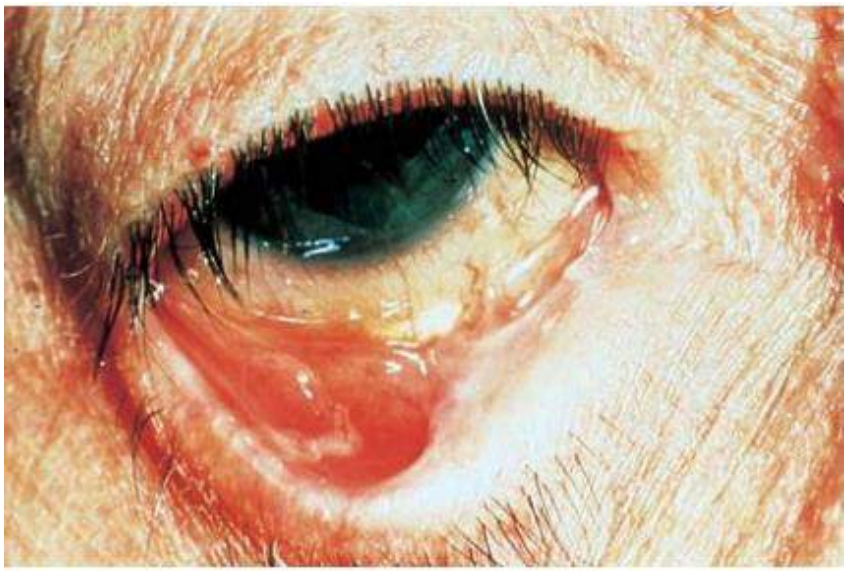
– Lysis of symblepharon (symblepharon refers to adhesions between the palpebral and bulbar conjunctiva; see also prognosis and complica-tions) to improve the motility of the globe and eyelids.

– Plastic surgery of the eyelids to the release the globe. This should be only performed 12 to 18 months after the injury).

– Where there is total loss of the goblet cells, transplantation of nasal mucosa usually relieves pain (the lack of mucus is substituted by goblet cells from the nasal mucosa).







Injuries Due to Physical Agents

Ultraviolet Keratoconjunctivitis

Injury from ultraviolet radiation can occur from **welding without proper eye protection**, exposure to **high-altitude sunlight** with the eyes open without proper eye protection, or due to **sunlight reflected** off snow when **skiing** at high altitudes on a **sunny** day. **Intense** ultraviolet light can lead to **ultraviolet keratoconjunctivitis** within a **short time** (for example just a **few minutes** of welding without proper eye protection). Ultraviolet radiation penetrates **only slightly** and therefore causes only **superficial necrosis** in the corneal epithelium. The exposed areas of the cornea and conjunctiva in the palpebral fissure become **edematous**, **disintegrate**, and are finally **cast off**. Ultraviolet keratoconjunctivitis is one of the **most common** ocular injuries.

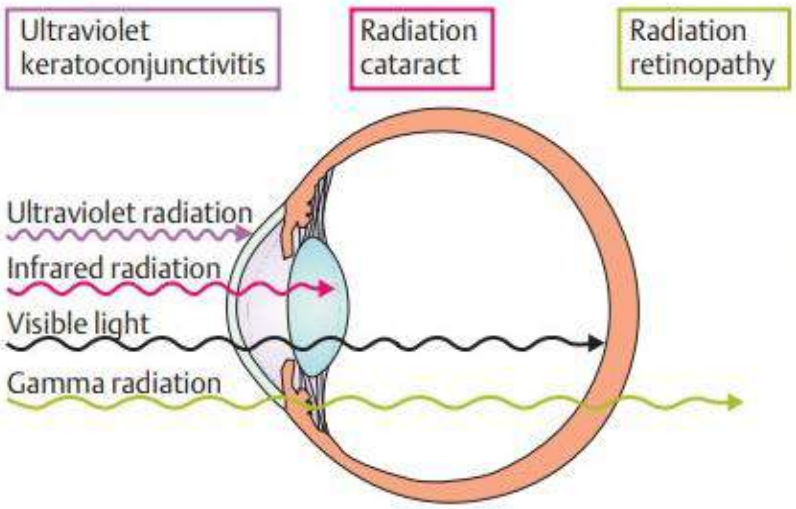
Burns

Flaring such as from a **cigarette lighter**, **hot vapors**, **boiling water**, and **splatters of hot grease** or **hot meta**l cause **thermal coagulation** of the **corneal and conjunctival surface.** Because of the **eye closing reflex**, the **eyelids** often will be **affected** as well.

Injuries due to explosion or burns from a starter’s gun also include particles of burned powder (powder burns). Injuries from a gas pistol will also involve a chemical injury.

Radiation Injuries (Ionizing Radiation)

Ionizing radiation (**neutron, or gamma/x-ray radiation**) have high **energy** that can cause **ionization and formation of radicals in cellular tissue**. Penetration depth in the eye **varies** with the **type of radiation**, i.e., the wavelength, resulting in characteristic **types of tissue damage.** This tissue damage always manifests itself after a latency period, often only after a period of years. Common sites include the lens (**radiation cataract**) and retina (**radiation retinopathy**).



Indirect Ocular Trauma: Purtscher's Retinopathy

Arterial and **venous circulatory disruption** in the **retina** characterized by a **sudden increase** in intravascular **pressure** may occur following **severe chest injuries** (compression trauma) or **fractures** of **long bones** (presumably due to fat embolisms or vascular spasms).

Symptoms and diagnostic considerations:

Acute retinal ischemia with impaired vision and loss of visual acuity will occur either immediately or within three to four days of the injury. Examination of the fundus will reveal cotton-wool spots and intra retinal bleeding indicative of focal retinal ischemia. Lines of bleeding will also be observed.

Treatment:

Fundus symptoms will usually disappear spontaneously within four to six weeks. Reduced visual acuity and visual field defects may occasionally persist. Occasionally treatment with high doses of systemic steroids and prostaglandin inhibitors is attempted.

OCULAR SYSTEMIC DISEASES WITH EYE

Many diseases can directly or indirectly damage the eyes and vision, while other diseases possess associated ocular signs symptoms and visual impairment.

SYSTEMIC DISEASES WITH OCULAR MANIFESTATIONS.

Main systemic disorders which may affect our eyes include endocrine and /or metabolic diseases, inflammatory and immune response processes, infections, hematological, cardiovascular, and cerebrovascular disorders, cancer, skin illnesses, and congenital/hereditary conditions. A summary of the processes that can affect the eyes and vision is reflected as follows. Systemic diseases with eye involvement include the following:

- Hematologic and lymphatic diseases.
- Cardiovascular/cerebrovascular diseases.
- Gastrointestinal/nutritional disorders.
- Metabolic/endocrine disorders.
- Musculoskeletal pathologies.
- Pulmonary diseases.
- Renal disorders.
- Systemic viral infections.
- Systemic bacterial infections.
- Systemic protozoal infections.
- Systemic fungal infections.
- Systemic cestode and nematode infections.
- Dermatologic pathologies.
- Phacomatoses.
- Collagen diseases.
- Multi systemic autoimmune diseases.
- Granulomatous diseases.
- Immunosuppressive agents used in management of eye disease.
- Ocular complications of certain systemically administered drugs.
- Neoplastic diseases with ocular metastases.
- Vitamins and eye diseases.
- Miscellaneous systemic diseases with ocular manifestations.
- Heritable connective tissue diseases.
- Hereditary metabolic disorders.
- Genetic syndromes.

MAJOR PATHOLOGIES WITH OCULAR INVOLVEMENT.

Some particular diseases are openly acknowledged to disturb the visual system, to a degree that the ocular manifestation may be used to accurately confirm the most complete diagnosis, as well as monitoring the appropriate therapy, such as incases of diabetes mellitus (DM), hypertension blood pressure(HBP), hyperthyroidism, sarcoidosis, tuberculosis, arthritis, psoriasis, scleroderma, or systemic infections.

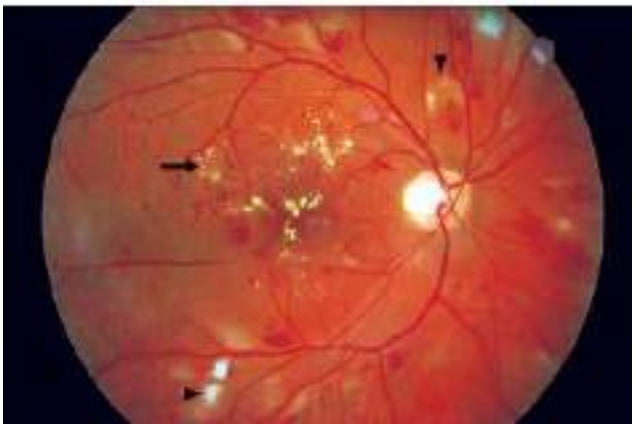
Ocular Consequences of Systemic Disease:

DIABETIC RETINOPATHY

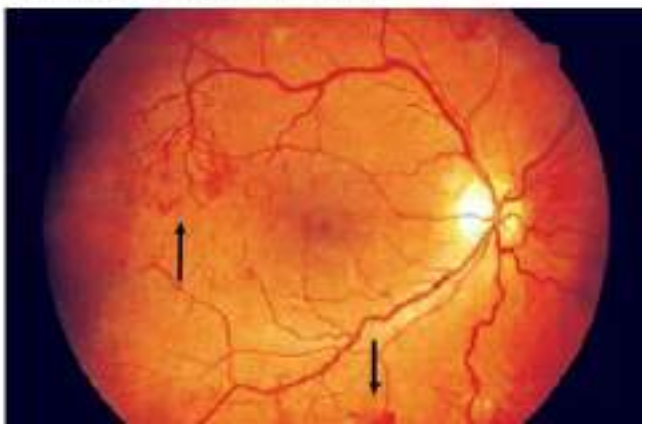
Diabetes mellitus can lead to **changes** in almost every ocular tissue. These include symptoms of keratoconjunctivitis sicca, xanthelasma, mycotic orbital infections, transitory refractory changes, cataract, glaucoma, neuropathy of the optic nerve, oculomotor palsy. However, 90% of all visual impairments in diabetic patients are caused by diabetic retinopathy.

Diabetic retinopathy remains **asymptomatic** for a long time. Only in the late stages with macular involvement or vitreous hemorrhage will the patient notice visual impairment or suddenly go blind, is characterized by tiny aneurysms (**baloon like swellings**) of the capillaries (tiny blood vessels) in the retina. New abnormal blood vessels which are fragile and bleed readily grow on the retinal surface. Haemorrhage into the vitreous humour may occur, fibrous tissues can also grow forward into vitreous humour. Treatment by laser surgery can often halt a progress of the condition. The risk of blindness due to diabetic retinopathy can be reduced by optimum control of blood glucose, regular ophthalmologic examination, and timely therapy, but it cannot be completely eliminated.

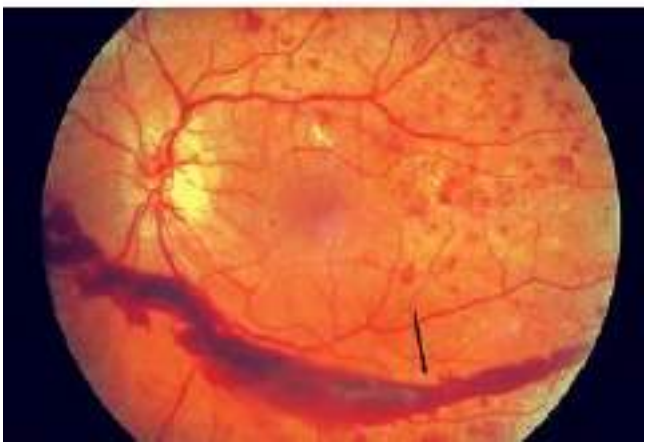
- Moderate nonproliferative diabetic retinopathy.



- Proliferative diabetic retinopathy.



- High-risk proliferative diabetic retinopathy.



HYPERTENSION-RELATED EYE CHANGES

Hypertensive retinopathy is characterized by narrowing of the retinal arteries. Areas of the retina may be destroyed and causes haemorrhage and white deposits may also occur in the retina. It may even lead to retinal detachment. Remedy is laser treatment.

Hypertension, known as the silent killer, can shorten the life span by as many as 20 years. End-organ damage affects the heart, brain, kidney, and eye. Hypertension may be manifested in one of two forms: chronic or acute. This differentiation is determined by the rapidity in rise of the blood pressure as well as the degree of elevation. The retinal changes observed with each form are different and have different consequences for the eye.

Chronic hypertension and atherosclerosis go hand in hand, and the associated retinal changes are evidenced by the development of retinal arteriolar changes, such as narrowing, and a change in light reflex. Funduscopic examination reveals a copper or silver coloration of the arterioles and venous compression (arterio venous nicking) at the arteriolar and venous crossings. Intraretinal hemorrhages from hypertension appear flame shaped because they occur in the nerve fiber layer of the retina.

Acute hypertension can result from pheochromocytoma, acute renal failure, pregnancy-induced hypertension, and malignant essential hypertension. The retinopathy associated with these states is extensive, and the manifestations include cotton-wool spots, retinal hemorrhages, retinal edema, and retinal exudates, often clustered around the macula.

The choroid is also affected by the profound and abrupt rise in blood pressure and resulting vasoconstriction, and ischemia may result in serous retinal detachments and infarction of the retinal pigment epithelium (RPE). Ischemic optic neuropathy and papilledema (ie, swelling of the optic disc due to increased IOP) may also result.

CYTOMEGALOVIRUS RETINITIS

Many ophthalmic complications are associated with AIDS. On autopsy, up to 90% of patients have ocular lesions directly related to AIDS. Cytomegalovirus (CMV) is the most common cause of retinal inflammation in patients with AIDS. About 40% of patients who have CMV retinitis lose their central vision in both eyes by the time of death.

Early symptoms of CMV retinitis vary from patient to patient. Some patients complain of floaters or a decrease in peripheral vision. Some patients have a paracentral or central scotoma, whereas others have a fluctuation in vision from macular edema. The retina often becomes thin and atrophic and susceptible to retinal tears and breaks.

CMV retinitis generally takes one of three forms: hemorrhagic, brushfire, or granular. In the hemorrhagic type, large areas of white, necrotic retina may be associated retinal hemorrhage. The brushfire form appears to have a yellow-white margin, which begins at the edge of burned-out atrophic retina. This retinitis expands and, if untreated, involves the entire retina. The granular form of CMV retinitis consists of white, granular lesions in the periphery of the retina that gradually expand. The white, feathery infiltration of the retina destroys sensory retina and leads to necrosis, optic atrophy, and retinal detachment.

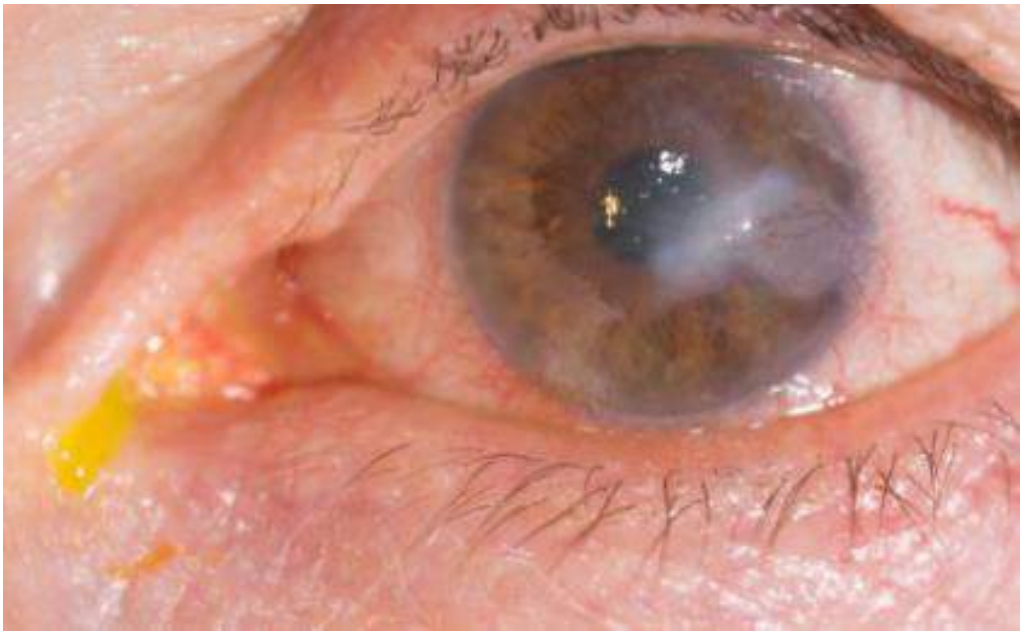
OCULAR SYSTEMIC DISEASES WITH EYE - 2

RHEUMATOID ARTHRITIS

It is estimated that approximately **25%** of patients with rheumatoid arthritis (RA) present with ocular manifestations. The **most common** external manifestation is **keratoconjunctivitis sicca**, which can be diagnosed using **Schirmer's test**. The test evaluates the **functionality** of the **lacrimal gland** by measuring the amount of tear production through the placement of a Schirmer strip into the lower conjunctival culde sac.

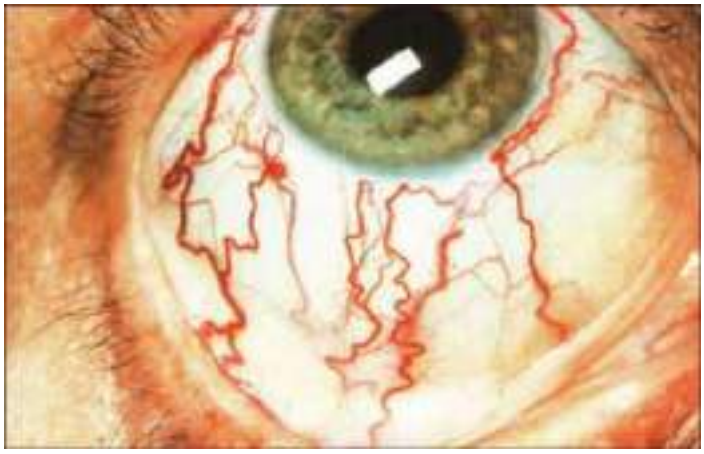
Episcleritis (**superficial** inflammation of the sclera, an inflammatory condition affecting the tissue between the conjunctiva (the membrane that lines the inside of the eyelid) and the sclera (the white part of the eye). The red appearance of episcleritis looks similar to pink eye (conjunctivitis), and **scleritis** (**deeper** inflammation of the sclera, is a severe ocular inflammatory condition affecting the sclera, the outer covering of the eye. It can be categorized as anterior with diffuse, nodular, or necrotizing subtypes and posterior with diffuse or nodular subtypes) are common ocular manifestations of RA, occurring in up to **25% and 10%** of RA patients, respectively.

Corneal disease is commonly associated with **dry eye** or with a form of anterior scleritis; it can include the presence of **keratitis (**also known as a corneal ulcer, is an inflammation or irritation of the cornea, although treatable, this condition is the most common cause of corneal blindness through an infection**), sclerosing keratitis (**a progressive opacification of the cornea which typically begins in an inferior arc from nasal to temporal aspects and extends to include all the lower cornea**).**



A **combination** of steroids, immunosuppressive therapy, and surgery often is necessary to prevent perforation of the **cornea and permanent vision loss**.

Scleritis and episcleritis are distinguished based on **anatomy and appearance**. While the symptoms may be similar, **pain is typically more severe in patients with scleritis**. Application of **phenylephrine** 10% will help distinguish scleritis from episcleritis: **Engorged vessels** in episcleritis will blanch, while those in scleritis will not. In RA, episcleritis **is more common** than scleritis; however, scleritis tends to be a more **destructive process**, with **necrotizing** scleritis with **inflammation** being one presentation. It is important to differentiate scleritis from episcleritis, as studies have documented a **higher mortality rate** and wider spread of systemic disease in RA patients with scleritis.



SYSTEMIC LUPUS ERYTHEMATOSUS

Systemic lupus erythematosus (SLE) is a **chronic, autoimmune, multisystem disease that affects the eyes in up to a third of patients,** in which the immune system attacks its own tissues, causing widespread inflammation and tissue damage in the affected organs. It can affect the joints, skin, brain, lungs, kidneys, and blood vessels

Ocular manifestations can be potentially sight-threatening and may be the presenting symptom of the disease. Ocular manifestations of SLE include **anterior** segment complications (usually associated with **pain and redness**) and **posterior** segment complications.

Common **anterior eye** complications in SLE include **keratoconjunctivitis and discoid lupus erythematosus.** Keratoconjunctivitis is the **most** common symptom **associated with SLE**, occurring in up to **30%** of patients. Symptoms, clinical assessment, and treatment of dry eye are similar to those described in patients with RA. **Cyclosporine 0.05%** is a commonly prescribed and effective treatment for dry eye syndrome.

Eyelid disease presents with a discoid lupus-type **rash** over the **eyelids** that consists of **raised, scaly lesions**.

Note : **Discoid lupus erythematosus (DLE)** is a chronic skin condition of sores with inflammation and scarring favoring the face, ears, and scalp and at times on other body areas.

Posterior segment complications consist mainly of **retinal disease.** Retinopathy is present in **10%** of patients with SLE. Some forms of retinopathy in SLE are similar to **hypertensive retinopathy** and **DR**, making management and monitoring difficult when these diseases occur at the same time. Aggressive, severe retinopathy, in extreme cases, may lead to **exudative retinal detachment**, and treatment with systemic **immunosuppression** is necessary to control the disease.