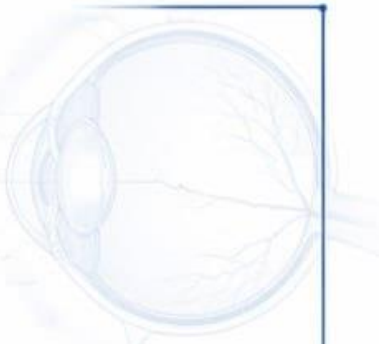
OPHTHALMOLOGY



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Preface

This summary was created in response to a recurring problem: the lack of a single, reliable, and comprehensive resource for ophthalmology. Existing summaries are often scattered, inconsistent in format, and vary widely in comprehensiveness. As a result, students spend unnecessary time navigating between sources instead of focusing on understanding.

This work attempts to address that gap.

It is based primarily on the most recent available slides (last sub-surgery group: 2021) and aims to be as complete as possible—covering more than 95% of the material, based on my judgment. The structure follows a slide-based approach, but with improved organization and formatting to make the content easier to follow and review.

Who this summary is for

- Students who prefer **thorough, structured material** over brief notes
- Those who find the original slides **poorly organized or difficult to follow**
- Anyone willing to dedicate **3–4 days for a proper first read of ophthalmology**
- Students considering skipping lectures (not recommended—all lectures are relatively manageable)
- Those looking for a **solid resource for revision**

Who this summary is *not* for

- Students looking for **high-yield, ultra-concise notes** for a first read
- Anyone planning to study ophthalmology **one day before the exam (or on the exam day)**

While concise, past-paper-based summaries can be sufficient for achieving high marks, this document is intentionally not designed in that way.

Expectations and Disclaimer

This summary was **not written based on past papers**, in order to avoid bias in content selection and ensure broader conceptual coverage.

However, solving past papers remains essential. I strongly recommend using Shahid Atiyat past papers, and the topics in this summary have been arranged to align with their general structure.

Be aware that:

- Some exam questions are **not directly covered in the recent slides** available online
- Certain topics—such as **keratoconus** and **retinal detachment**—are emphasized heavily in past papers, sometimes beyond the depth provided in lectures

For this reason, relying on this summary alone is not sufficient. It should be used alongside active practice of past exam questions.

Special thanks to ChatGPT

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Optics and Refraction

Introduction

The human eye functions according to the principle of **Refraction**, which refers to the bending of light as it passes between different media. Visual clarity is achieved when the refractive components of the eye (primarily the **cornea** and the **lens**) focus incoming light precisely onto the retina.

Refractive Components of The Eye

1. **Cornea**
   - Provides approximately **two-thirds** of the eye's refractive power
   - Has **fixed** refractive power
2. **Lens**
   - Provides approximately **one-third** of the eye's refractive power
   - Has **dynamic** refractive power, adjustable through **Accommodation**

Measurement of Refractive Power

Refractive power is measured in **Diopters (D)** → **Diopter = 1 / focal length** (in meters)

Near Reflex Triad

1. **Miosis**
2. **Convergence**
3. **Accommodation**
   - Accommodation is the process by which the eye focuses on near objects. Near light rays diverge and therefore require greater refractive power.
   - Ciliary muscle contracts → Zonules relax → Lens becomes thicker → ↑ **Refractive power**

Emmetropia (Normal Vision)

- Parallel rays from distant objects focus exactly on the retina while the eye is in a resting state with no accommodation

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Classification of Refractive Errors

1. Hypermetropia (Hyperopia / Farsightedness) (طول النظر)

- Light focuses **behind the retina**
- Near objects are blurry; distant objects may also be affected

Causes:

- Short eyeball
- Abnormal corneal or lens shape
- Physiological (normal development stage in children)

Clinical Features:

- Often present **at birth**
- Young individuals compensate via accommodation (no need for glasses)
- Adults over 50 experience blurred distance vision (loss of accommodation → they need correction)

Correction:

- Convex (converging, positive) lenses

Complications:

1. **Angle-closure glaucoma**
2. **Strabismus** (especially accommodative esotropia) **(الحول)**
3. **Amblyopia (الكسل البصري)**

2. Myopia (Nearsightedness) (قصر النظر)

- Light focuses **in front of the retina**
- Distant vision is blurry; near vision remains normal

Epidemiology:

- Begins between ages 6–14
- Progresses until early adulthood (rarely after age of 25 → suspect **DM** or **Cataract**)

Causes:

- Long eyeball
- May be associated with **keratoconus (القرنية المخروطية)**

Correction:

- Concave (diverging, negative) lenses
- Refractive surgery

Complications (rare):

1. Retinal detachment
2. Retinal tears
3. Macular holes
4. **Open-angle glaucoma**

3. Astigmatism (اللابؤرية)

- Non-uniform refraction across different meridians
- Caused by irregular corneal or lens curvature

Characteristics:

- Distorted vision at both near and far distances

Correction:

- Cylindrical lenses
- Refractive surgery

4. Presbyopia (طول النظر الشيخوخي)

- Age-related condition (>45 years)
- Loss of accommodation

Effect:

- Light focuses **behind the retina**
- Difficulty with near vision tasks such as reading

Terminologies

Aniseikonia	Difference in perceived image size between eyes
Anisometropia	Refractive difference greater than 2 diopters between eyes
Aphakia	Absence of the natural lens
Pseudophakia	Presence of an artificial intraocular lens
Ectopia lentis	Displacement of the lens

Common Symptoms of Refractive Errors

- Blurry, hazy, or double vision
- Halos or glare around lights
- Squinting (حول)
- Eye strain
- Headaches, especially during reading or computer use

Management and Corrective Strategies

1. Non-Surgical Options

A. Glasses

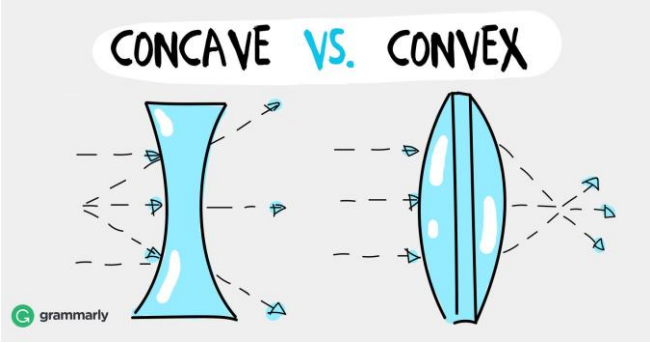
- Myopia → concave (negative) lenses
- Hyperopia → convex (positive) lenses
- Astigmatism → cylindrical lenses

B. Contact Lenses

- Available as soft or rigid lenses

Complications:

- Corneal hypoxia
- Neovascularization
- Corneal abrasions
- Giant papillary conjunctivitis
- Corneal ulcers
- Keratitis



2. Surgical Interventions

A. Corneal Refractive Surgery

1. **PRK** (Photorefractive Keratectomy)
2. **LASIK** (Laser-Assisted In Situ Keratomileusis)

B. Lens Refractive Surgery

LASIK Procedure

Steps

1. Application of numbing drops
2. Creation of corneal flap
3. Laser reshaping (e.g., flattening for myopia)
4. Repositioning of the flap

Prerequisites

- Suitable age
- Stable refractive error

Contraindications

- Pregnancy / lactation
- Collagen tissue diseases
- **Keratoconus**
- Active corneal disease
- **Severe dry eye**

Potential Complications

- Flap-related issues
- Corneal opacification or perforation
- Under- or over-correction
- Corneal ectasia
- Epithelial ingrowth
- Recurrance

Anatomy & Physiology of Eye Movements

Extraocular Muscles (6 muscles)

1. Lateral rectus (LR)
2. Medial rectus (MR)
3. Superior rectus (SR)
4. Inferior rectus (IR)
5. Superior oblique (SO)
6. Inferior oblique (IO)

Nerve Supply

- **3rd cranial nerve (Oculomotor)** → MR, IR, SR, IO
- **4th cranial nerve (Trochlear)** → **SO**
- **6th cranial nerve (Abducens)** → **LR**

Yoke Muscles

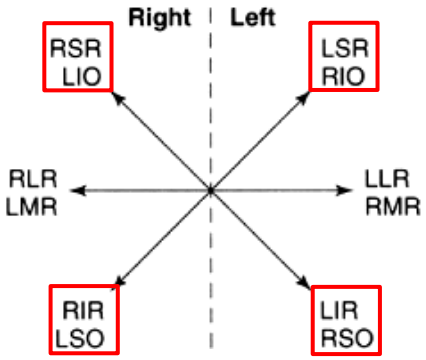
- Pairs of muscles (one in each eye) that move both eyes in the same direction.

Positions of Gaze

- 6 Cardinal positions of gaze
- 9 Total positions of gaze

6 Cardinal positions & responsible extraocular muscles

Eyes up and right	RSR and LIO
Eyes up and left	LSR and RIO
Eyes right	RLR and LMR
Eyes left	LLR and RMR
Eyes down and right	RIR and LSO
Eyes down and left	LIR and RSO



*Note: while **yoke muscles** are the primary pair responsible for directing gaze into a specific position, complex eye movements (e.g., looking up-right) involve coordinated action of multiple extraocular muscles in both eyes.*

Binocular Vision & Stereopsis

Binocular Single Vision

Both eyes focus on the same object → image of that object created in each retina → images fuse in the brain → single vision perception & **Stereopsis** (رؤية مجسمة) **Importance**

of Stereopsis

- Increased field of vision
- Elimination of blind spot
- Better visual acuity
- Depth perception
- Distance estimation

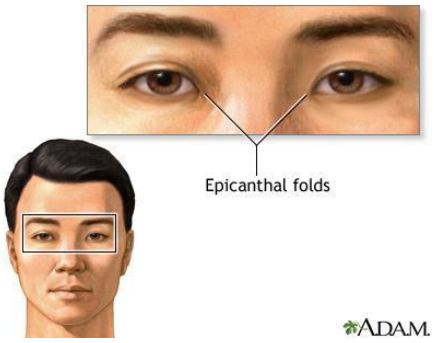
Strabismus (Squint)

Introduction

Strabismus (squint) is a **misalignment of the two eyes**, where both eyes are not directed toward the same point simultaneously.

It is a common ophthalmologic condition, especially in children, and may indicate underlying neurological, muscular, or refractive abnormalities.

Clinical Evaluation



A. History

- Frequency (constant vs intermittent)
- Onset (congenital vs acquired)
- Family history
- Associated systemic or neurological disease
- Past medical/surgical history

B. Examination

- **Binocular eye movement evaluation**
- **Epicanthal folds**
  - *Can mimic squint (Pseudo-strabismus) but does not exclude it*
- Facial asymmetry
- Ocular alignment tests:
  - **Cover–uncover test** → detects **Tropia** **(Manifest Squint, الحول الظاهر)**
  - **Alternate cover test** → detects **Phoria** **(Latent Squint, الحول الخفي)**
- **Visual acuity** (important for **amblyopia** detection)
- **Cycloplegic refraction** (atropine/cyclopentolate) to assess refractive error accurately

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Classification of Strabismus

A. By Comitancy (Eye Movement Pattern)

1. Non-Paralytic (Concomitant) Squint

Features

- Full ocular movements
- **Constant** angle of deviation in all gazes
- Usually occurs in children
- No diplopia (brain suppresses image)
- Risk of **amblyopia (before age of 10)**
- Often:
  - **Congenital** or **Associated with hypermetropia**

Pathophysiology

- Sensory adaptation → suppression → amblyopia

Treatment

- Optical correction (glasses)
- Amblyopia therapy (**patching better eye**)
- Surgery (muscle correction)

2. Paralytic (Incomitant) Squint

Features

- Limitation in ≥1 extraocular muscle due to nerve palsy or extraocular mechanical restriction
- Angle **varies** with gaze direction
- **Diplopia** present
- Usually **acquired**
  1. Isolated Cranial Nerve Palsies
  2. Extraocular Muscle Disorders

A. Isolated Cranial Nerve Palsies

1. 6th Nerve (Abducens)

1. Failure of abduction → Eye deviates medially (**Esotropia**)
2. **Horizontal diplopia**
3. **Compensatory head tilt**
    - Patients tilts head **toward** the affected side to compensate diplopia

2. 4th Nerve (Trochlear)

1. Defective depression in adduction (**Hypertropia**)
2. **Vertical diplopia**
3. **Compensatory head tilt**
    - Patient tilts head **away** from the affected side to compensate diplopia

3. 3rd Nerve (Oculomotor)

1. Loss of eye adduction, elevation & depression
2. Unopposed action of LR + SO → **Eye positioned "down and out"**
3. **Multidirectional diplopia**

Etiology of palsy

1. Orbital causes
    - Tumors
2. Vascular
    - **DM** (**Pupil-sparing CN III palsy**)
    - HTN
    - **Posterior Communicating Artery (PCOM) Aneurysm**
        - **Emergency**
        - Presentation:
            1. **3rd nerve palsy**
                - Strabismus
                - Ptosis
                - Mydriasis + Loss of accommodation
            2. **Sudden severe headache** if ruptured
    - **Stroke**
3. Trauma
4. Neoplasia
    - Meningioma
    - Glioma
    - Acoustic neuroma
5. Raised ICP (3rd/6th nerve palsy)
6. Inflammation
    - Sarcoidosis
    - Giant cell arteritis
    - Guillain-Barré Syndrome
7. **Cavernous sinus thrombosis**

B. Extraocular Muscle Disorders

1. Thyroid (Dysthyroid) Eye Disease

- Lymphocytic infiltration & GAG deposition
- Leads to:
    - **Proptosis**
    - Exposure keratopathy
    - Restricted movements
- Seen in hyper/hypothyroidism

2. Myasthenia Gravis

- Autoimmune (ACh receptor antibodies)
- More common in females (15–50 years)
- **40%** → only extraocular involvement

Features

- Variable diplopia
- Fatigable ptosis

Diagnosis

- Edrophonium test

Treatment

- Anticholinesterases (**neostigmine**)
- Thymectomy

3. Ocular myositis

4. Ocular myopathy

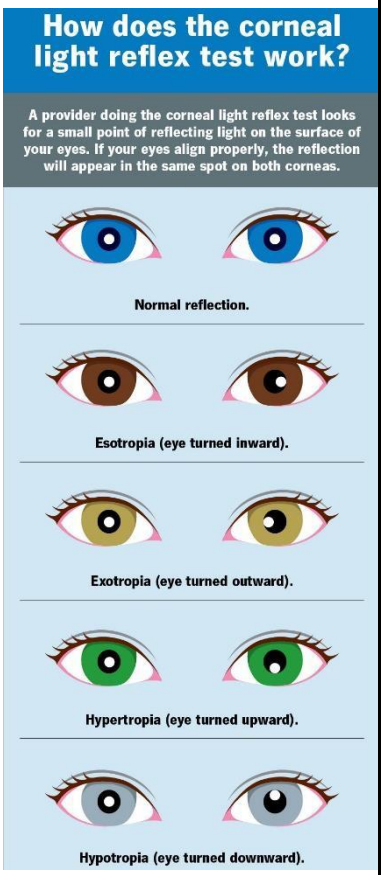
5. Brown syndrome

6. Duane syndrome

B. By direction of deviation

1. Esotropia
2. Exotropia
3. Hypertropia
4. Hypotropia

→ How it detected? Eyeball direction & Corneal Light Reflex



→ Phoria vs Tropia (Cover Tests)

1. Cover–Uncover Test → Detects Tropia (Manifest Squint)

- Used to detect Strabismus (tropia) = visible deviation
- Method:
  - Cover one eye while the patient fixates on a target
  - Observe the uncovered eye
- Interpretation:
  - If the uncovered eye moves to fixate → tropia present
- Meaning:
  - The eye was already deviated even when both eyes were open

2. Alternate Cover Test → Detects Phoria (Latent Squint)

- Used to detect phoria (latent deviation)
- Method:
  - Rapidly alternate covering each eye (no time for fusion)
- Interpretation:
  - Each eye refixates when uncovered → phoria present
- Meaning:
  - Eyes are aligned normally with both eyes open
  - Deviation appears only when fusion is disrupted

A. Esotropia

General Types

- Right / Left / Alternating
- Concomitant or Incomitant
- Primary / Secondary / Consecutive (Overcorrection)

1. Congenital (Infantile) Esotropia → Concomitant esotropia

- Onset: < 6 months
- Large angle deviation
- No association with hypermetropia
- Child otherwise neurologically normal

Treatment

- Bilateral medial rectus recession (Surgery)

2. Accommodative Esotropia

Mechanism

- **Hypermetropia** → increased accommodation
- **Accommodation linked to convergence (Synkinesis)**
  - Near reflex triad:
    - Accommodation
    - Convergence
    - Miosis
- Excess convergence → esotropia

Key Point

- Risk of **amblyopia (before age of 10)**
- Treatment:
  - **Hypermetropia correction**
  - Bifocals
  - **Surgery**
    - Glasses are still required postoperatively

3. Non-Accommodative Esotropia

Causes:

- Emotional/physical stress
- **Sensory deprivation**:
  - Congenital cataract
  - Optic atrophy
- **Retinoblastoma**

B. Exotropia

Types

1. Intermittent
2. Constant

1. Intermittent Exotropia

- Onset < 5 years

Triggered by:

- Fatigue
- Illness
- Stress
- Inattention

Special Feature:

- Bright light → reflex eye closure in one eye (**photophobia–squint response**)
  - Common, characteristic sign of intermittent exotropia

2. **Non-Accommodative Exotropia**

Example: Crouzon Syndrome

- FGFR2 mutation
- Autosomal dominant (chromosome 10)

Features:

- **Shallow orbits**
- Craniosynostosis
- **Hypertelorism**
  - An abnormally increased distance between the eyes
- Cardiac anomalies (PDA, coarctation)

Management of Strabismus

- Early detection
- Optical correction (glasses)
- Surgery:
  - Recession (weakening muscle)
  - Resection (strengthening muscle)

Glasses and surgery are **not interchangeable**

Amblyopia (الكسل البصري / الغمش)

Definition

- **Amblyopia** is a **permanent decrease** in **visual acuity** in one or both eyes due to **abnormal visual development** during childhood, without any detectable structural abnormality of the eye.

Pathophysiology

- The brain receives unequal or abnormal visual input from the two eyes.
- To avoid confusion or diplopia, it suppresses the poorer-quality image.
- Over time, this leads to:
  - Poor development of visual cortex pathways
  - Permanent reduction in visual acuity if untreated (before age of 10)

Types

1. Strabismic Amblyopia

- Due to long-standing squint
- Brain suppresses deviated eye
- Common in children

2. Anisometropic Amblyopia

- Unequal refractive error
- One eye dominates → other suppressed

3. Form Deprivation Amblyopia

- Due to obstruction:
  - Retinoblastoma
  - Cataract
  - Corneal opacity/scarring (e.g., birth trauma)
  - Ptosis
  - ~~Corneal~~ Congenital Hemangioma on eyelids
  - …
- Leads to poor visual development

Cornea

Overview

Anatomy of the Cornea

- A **transparent, avascular structure** forming the anterior part of the eye. It plays a crucial role in **vision and protection**.

Dimensions of the Cornea

- Thickness: ~0.5 mm
- Diameter: 11–12 mm

Layers of the Cornea (5 Layers)

1. **Epithelium**
   - Stratified squamous
   - Rapid regeneration from **limbal stem cells**
2. **Bowman's Membrane**
   - Acellular protective layer
3. **Stroma**
   - ~90% of corneal thickness
   - Composed of **parallel collagen fibrils**
4. **Descemet's Membrane**
   - Strong and elastic
5. **Endothelium**
   - Single layer, **non-regenerating**
   - Pumps fluid out of stroma → maintains transparency
   - Damage → edema → corneal clouding

Physiology of the Cornea

Nutrition of the Cornea

- From **aqueous humor** (posterior layers)
- From **tear film & ambient air** (anterior layers)

Key Features

- Avascular
- Rich sensory innervation (trigeminal nerve – nasociliary branch)

Functions of the Cornea

- **Protection** of intraocular structures
- **Refraction** (major role in focusing light)

Refractive Power of the Cornea

- Cornea: ~⅔ (fixed)
- Lens: ~⅓ (adjustable via accommodation)

Defense Mechanisms of the Cornea

- Blinking
- Tear flow (washing debris)
- Mucus trapping particles
- Antimicrobial tear components
- Intact epithelium barrier

  Disruption → increased infection risk

I. Infective Corneal Diseases (Keratitis)

A. Herpes Simplex & Herpes Zoster Keratitis

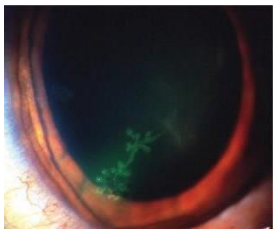
Etiology

- HSV-1 (most common), HSV-2, VZV

Pathogenesis

- Virus remains latent in **trigeminal ganglion**
- Reactivation → **recurrent disease** (usually acquired in early life & what we see in clinic is reactivation)

HSV-1/2 Clinical Features



- **Severe pain**, photophobia
- **Watery discharge**
- Eyelid swelling
- **Dendritic ulcers (pathognomonic)** → →
  - Usually **heal without scar**

VZV Clinical Features

- Skin vesicles (ophthalmic trigeminal distribution)
- **Hutchinson's sign** → nasociliary involvement
  - A vesicular rash on the tip of the nose in herpes zoster ophthalmicus
  - It is associated with a 6× higher likelihood of **ocular problems**

Complications

- Stromal involvement → edema → **scarring** → **corneal graft** needed

Treatment

- **Topical acyclovir** → **dendritic ulcer (HSV-1)**
- **Oral** antivirals → **skin involvement (VZV)**
- **Avoid steroids in dendritic ulcers**

B. Bacterial Keratitis

Epidemiology

- 90% of corneal infections

Common Organisms (unable to penetrate intact epithelium**)**

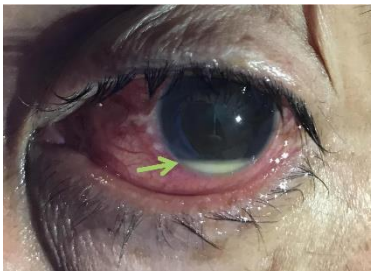
- Staphylococcus
- Streptococcus

Special Case

- ***Neisseria gonorrhoeae*** the only organism penetrates intact epithelium

Risk Factors

- **Epithelial defect**
- **Dry eye**
- Contact lens use
- Steroid use



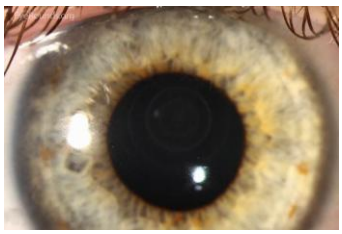
Symptoms

- Pain
- **Purulent discharge**
- Visual loss
- **Hypopyon** → → →

Treatment

- Broad-spectrum topical **antibiotics** **(covers G+ve & G-ve)**
  - **Fluoroquinolones** (monotherapy option)

C. Acanthamoeba Keratitis



Cause

- Protozoa

Association

- Contact lens users

Features

- Severe pain
- **Ring-shaped corneal infiltrate → → →**

Treatment

- **Chlorhexidine** + **Propamidine**
- Prolonged therapy
- May require **corneal graft**

D. Fungal Keratitis

Risk Factors

- Warm climates
- Trauma with plant material
- Steroid use
- Non-response to antibiotics

Features

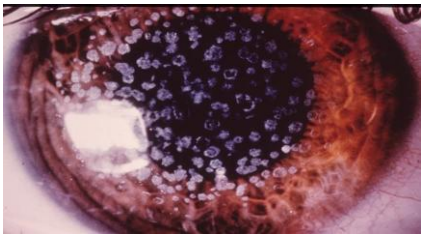
- **Fluffy corneal infiltrates → → → →**

Treatment

- Topical + systemic **antifungals**

II. Non-Infective Corneal Disorders

A. Corneal Dystrophies



Characteristics

- Inherited disorders (rare but relatively common in Jordan)
- Affect corneal transparency

Types

1. Anterior → recurrent corneal erosions
2. Stromal → visual loss
3. Posterior → gradual visual loss

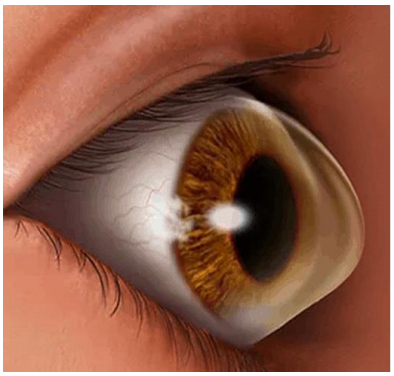
B. Keratoconus (KC)

Definition

- Progressive thinning of central cornea → cone shaped cornea

Effects

- **Myopia**
- **Astigmatism**
- Visual impairment without pain
- May be subclinical



Diagnosis

- **Corneal topography**

Treatment Options

- Glasses
- Contact lenses
- **Corneal cross-linking** (stops progression)
- **Corneal graft** (advanced cases)

Corneal Surgery

A. Corneal Grafting (Keratoplasty)

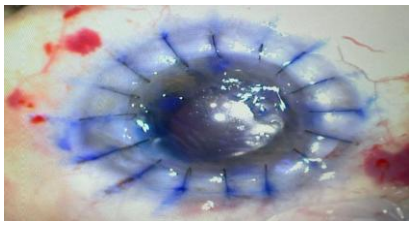
Definition

Corneal grafting is the transplantation of donor corneal tissue to:

- Restore corneal clarity
- Repair corneal perforations

Key Features

- The cornea is **avascular**, providing an **immune-privileged environment**
- High success rate (~80%)
- **No HLA matching required**
- Donor tissue:
  - Extracted within **24 hours after death**
  - Can be used within **2 weeks**



Postoperative Care

- **Topical steroid eye drops** to prevent graft rejection
- **Non-absorbable sutures** may remain for ~1 year

B. Refractive Surgery of the Cornea

Types

1. **PRK** (Photorefractive Keratectomy)
2. **LASIK** (Laser-Assisted In Situ Keratomileusis)
3. **LASEK** (Laser-Assisted Subepithelial Keratectomy)
4. **Trans-PRK** (Transepithelial Photorefractive Keratectomy)
5. **Corneal cross-linking, rings, grafts**

Overview

- 90–95% of cases are treated using **laser-based procedures**
- Success rate approaches ~99%

Excimer Laser Mechanism

Characteristics

- Excited Dimer
- UV laser (193 nm)
- A cool laser beam → **minimal thermal damage**
- Causes **photoablative decomposition** of corneal stroma

Effects on Vision

- **Myopia** → flattens cornea
- **Hypermetropia** → steepens cornea
- **Astigmatism** → flattens steepest meridian

Major Procedures

A. PRK

- Epithelium removed
- Laser applied directly to corneal surface
- Epithelium regenerates later

B. LASIK

- Creation of a **corneal flap**
- Laser reshapes underlying stroma
- Faster recovery compared to PRK

Preoperative Assessment

Before laser surgery:

- **Must do** → **Corneal topography**
  - Corneal thickness measurement (Pachymetry)

Contraindications to Refractive Surgery

- Age < 19 years
- Unstable refractive error
- Corneal diseases (e.g., **keratoconus**)
- Severe **dry eye** or inflammation
- Keratitis
- Poor vision from other causes

Sclera

Overview

Structure

- White outer coat of the eye
- Composed of **interwoven collagen fibrils**
- Thickness:
  - ~1 mm near optic nerve
  - ~0.3 mm posterior to muscle insertions

Function

- Provides structural integrity
- Attachment site for **six extraocular muscles**

Diseases of the Sclera

A. Episcleritis

Features

- Inflammation of superficial scleral layer
- Mild discomfort
- **Segmental redness**

Characteristics

- Usually **self-limiting**
- Rarely associated with systemic disease

Management

- May use **topical anti-inflammatory drug**

B. Scleritis

Features

- **Severe ocular pain**
- **Diffuse redness**
- Scleral swelling

Associations

- Strongly linked to **collagen vascular diseases**
  - Most commonly **Rheumatoid Arthritis**

Complications

- Scleral thinning (**Scleromalacia**)
- Keratitis
- Uveitis
- Cataract
- Glaucoma

Management

- Mild cases:
  - **Topical Steroids**
  - **Oral NSAIDs**
- Moderate–severe cases:
  - Systemic steroids
  - Cytotoxic drugs
- **Requires evaluation for underlying systemic disease**

Lens

Anatomy

Location and Structure

- The lens is a **transparent, biconvex structure** located **behind the iris and in front of the vitreous body**
- It is enclosed by a capsule and suspended by **zonular fibers** attached to the ciliary body

Key Features

- **Avascular** (no blood supply)
- Derived from **surface ectoderm**
- Highly **elastic** (elasticity decreases with age → **Presbyopia**)
- Primary function: **focus light on the retina**

Histology

Components

1. **Lens Capsule (LC)**
    - Thick basement membrane
    - Composed of **collagen type IV and proteoglycans**
    - Functions:
        - Protection
        - Attachment for zonules
2. **Lens Epithelium (LE)**
    - Single layer of **cuboidal cells**
    - Present only on the **anterior surface**
    - Responsible for **cell proliferation**
3. **Lens Fibers**
    - Formed from epithelial cells near the equator
    - Fibers are parallel to the epithelium
    - Two types:
        1. **Differentiating fibers** → elongated, nucleated, rich in **crystallins**
        2. **Mature fibers** → lose nuclei, densely packed → maintain transparency

Functional Significance

- The unique parallel arrangement and absence of organelles in mature fibers allow **optical clarity**

Cataract

Definition

- Opacification of the lens, resulting in loss of transparency

Epidemiology

- Most common cause of treatable blindness worldwide

Pathophysiology of Cataract

Normal Vision

- Clear lens → **focused light** → **sharp retinal image**

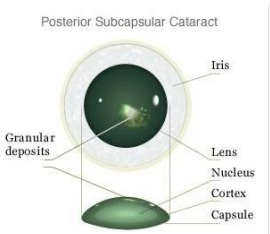
Cataract Vision

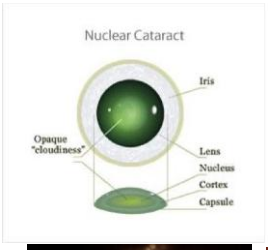
- Opaque lens → **light scattering** → **blurred, hazy image**

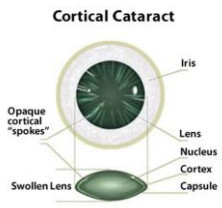
Clinical Effects

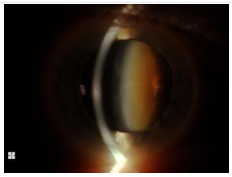
A. Symptoms

- **Progressive painless vision loss**
- **Blurred vision**
- **Change in refractive error**
- **Color desaturation**
- **Glare & Halos around lights** (especially at night)
- **Amblyopia**









B. Signs

- Reduced visual acuity
- Lens opacity on slit-lamp examination
- **Diminished or irregular red reflex**
- **Leukocoria** (white pupillary reflex)

Classifications of Cataract

A. By Anatomy

1. Nuclear Cataract

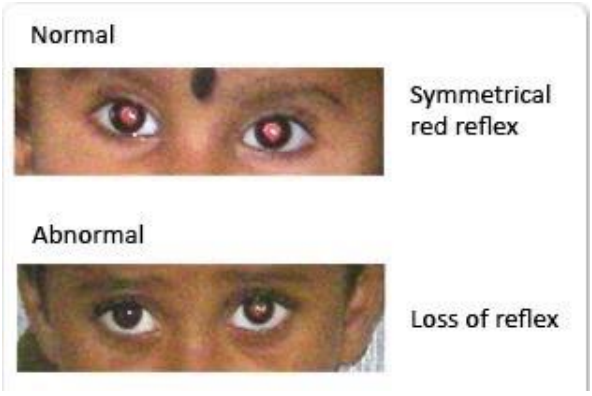
- Most common type
- Involves **central (nuclear) part of the lens**
- Pathogenesis:
  - condensation of the lens & deposition of brown pigment within lens → **Nuclear Sclerosis**
  - Nuclear Sclerosis → ↑ in Refractive Index → light focus in front of the retina (Myopia)
- Clinical features:
  - **Myopic shift**
    - **Second Sight** → Temporary improvement in near vision due to myopic shift
    - Progressive **distance vision deterioration**
  - **Central cloudiness** on slit lamp

2. Cortical Cataract

- Affects the **lens cortex (outer layer)**
- Pathogenesis:
  - **↑ in water content** in periphery of the lens → separation of fibers → fissures & vacuole formation
- Clinical features:
  - **Glare** (especially at night)
  - **Radial cortical "spoke-like" opacities** on slit lamp

3. Posterior Subcapsular Cataract (PSC)

- Located at the **posterior pole beneath the capsule**
- Important feature:
  - Causes **disproportionately severe symptoms** relative to size
    - Mechanism → Light becomes more focused (converges) toward the back of the lens
- Clinical features:
  - Early visual impairment
  - Difficulty with reading and bright light
  - **Granular opacities** on slit lamp



B. By Maturity

1. Immature cataract

- Partial opacification
- Some lens fibers remain clear

2. Mature cataract

- Entire lens becomes opaque

C. By Age of Onset

1. Congenital Cataracts

- Present at birth or early life. May be detected in adults
- Morphological Subtypes include:
  - Lamellar
  - Polar
  - Sutural
- Important clinical features → **Leukocoria**

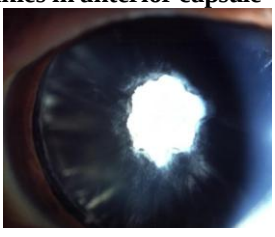
2. **Developmental Cataract**
3. **Age-related (Senile Cataract)**
    - Most common cause
    - Results from cumulative exposure to:
        - Ultraviolet (UV-B) radiation
        - Smoking
        - Oxidative stress
        - Elevated blood glucose levels

D. By Etiology (Causes of Cataract)

1. **Ocular Causes**
    - Trauma
    - Uveitis
    - High myopia
    - Prolonged use of topical steroids
    - Intraocular tumors
2. **Systemic Causes**
    - **Metabolic diseases**:
        - Diabetes mellitus
        - Galactosemia
        - Hypocalcemia
        - Fabry disease
    - **Drugs**:
        - Steroids → Systemic & Topical
        - Chlorpromazine
    - **Infections**:
        - Congenital **Rubella**
    - **Genetic/Systemic conditions**:
        - Myotonic dystrophy
        - **Down syndrome**
        - Lowe syndrome
    - **Others**:
        - Atopic dermatitis
        - External radiation

Special Associations

- **Lowe syndrome**: a rare **X-linked** AR disorder with **congenital cataracts**, hypotonia & areflexia, mental retardation, RTA Type 2, and **glaucoma** (~50%).
- **Fabry Disease**: a rare **X-linked** lysosomal storage disease
- **Atopic dermatitis cataract**:
    - Occurs in ~10% (age 15–30)
    - Often bilateral
    - Frequently mature cataract
    - Characteristic **anterior subcapsular (shield) cataract + wrinkles in anterior capsule**



Management

General Principles

- No proven method of prevention
- Definitive treatment is **surgical removal of the lens**
- Indication: **functional visual impairment affecting quality of life**
- No need to wait for "maturation" of cataract

Cataract Surgery

- Usually a **day-case procedure**
- Can be done under:
  - Topical anesthesia
  - Local anesthesia
  - General anesthesia
- Procedure involves:
  - Removal of most of lens fibers and epithelium
  - Implantation of appropriate **intraocular lens (IOL)**

Types of Cataract Surgery

1. Phacoemulsification (Most Common)

Steps:

1. Local/topical anesthesia
2. Corneal incisions (2 cuts for instruments)
3. **Capsulorhexis** (opening of lens capsule)
4. Ultrasonic emulsification of lens (**Phacoemulsification**)
5. Aspiration of lens material
6. Insertion of foldable plastic intraocular lens ± Antibiotics
7. Wound hydration and sealing

2. Extracapsular Cataract Extraction (ECCE)

- Lens removed manually
- Capsule largely preserved
- Requires **larger incision** (10–12 mm)
- Sutures needed
- Useful for **hard cataracts**
- Variant: **Manual Small Incision Cataract Surgery (MSICS)**
  - No need for sutures (self-sealing scleral tunnel wound)

3. Intracapsular Cataract Extraction (ICCE)

- Entire lens + capsule removed in one piece
- Requires large incision
- Rarely used due to high complication rate

Postoperative Care

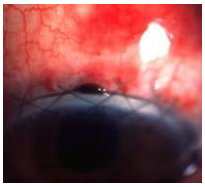
- Topical **antibiotics and steroids**
- Correction for near vision (since **patient loss accommodation after surgery**):
  - Glasses
  - Multifocal intraocular lenses → Glares is most common complaint

Complications of Cataract Surgery

A. Early Complications

1. Vitreous loss (Intra-op)

- Risk: glaucoma, retinal traction
- Management: vitrectomy at operation time, delay of IOL



2. Iris prolapse (Immediately post-op)

- Causes:
  - Inadequate suturing of incision
  - Inappropriate management of vitreous loss
- Management: surgical repair

3. Endophthalmitis (Emergency)

- Symptoms few days after surgery:
  - Painful red eye
  - Reduced vision
  - **Hypopyon**
- Treatment:
  - **Intravitreal broad-spectrum antibiotics**
- Common organisms:
  - Staphylococcus epidermidis
  - Staphylococcus aureus
  - Pseudomonas

B. Late Complications

1. Posterior Capsule Opacification (PCO)

- Most common late complication (20%)
- Due to proliferation of residual lens epithelial cells
- Causes gradual decrease in vision after initial improvement

Management:

- Outpatient → Nd:YAG laser capsulotomy

2. Cystoid Macular Edema (CME)

- Accumulation of fluid in the macula
- Leads to decreased central vision

Features:

- Painless reduction in vision weeks after surgery

Management:

- Topical NSAIDs ± steroids

3. Postoperative Astigmatism

- Caused by tight or uneven sutures
- Leads to refractive error

4. Retinal Detachment

Diabetic Retinopathy

Introduction

Diabetic eye disease refers to a spectrum of ocular complications arising from diabetes mellitus. These include:

- Corneal abnormalities
- Iris and angle neovascularization
- Neovascular glaucoma
- Cataracts (including "**snowflake cataracts**" in young patients)
- Ocular neuropathies
- **Diabetic retinopathy (DR)**

Definition

Diabetic retinopathy is a **progressive microangiopathy** affecting retinal blood vessels due to chronic hyperglycemia.

Epidemiology

It is **the leading cause of moderate to severe vision loss** in working-age adults (25–74 years).

Anatomy of the Retina

Layers:

- **Inner neurosensory retina (NSR):** contains rods and cones
- **Outer retinal pigment epithelium (RPE)**

Blood supply:

- Central retinal artery
- Choroidal circulation

Risk Factors

Major risk factors for diabetic retinopathy include:

- Long duration of diabetes
- **Poor glycemic control**
- **Nephropathy**
- Hypertension
- Hyperlipidemia
- Obesity
- Smoking
- Pregnancy

Pathogenesis

The fundamental mechanism is **microangiopathy**, characterized by:

1. Microvascular Leakage

- Loss of pericytes
- Formation of microaneurysms
- Breakdown of blood-retinal barrier
- Leads to:
  - **Retinal edema**
  - **Lipid exudates**
  - **Hemorrhages**

2. Microvascular Occlusion

- Basement membrane thickening
- Endothelial damage
- RBC deformities
- Platelet aggregation
- Leads to → **retinal ischemia**

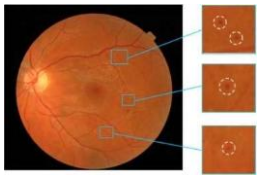
3. VEGF Production

- Hypoxia stimulates VEGF release
- Leads to → **neovascularization**

Types of Diabetic Retinopathy

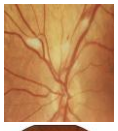
1. **Non-Proliferative Diabetic Retinopathy (NPDR)**

Early stage characterized by (on **Fundus Exam**):

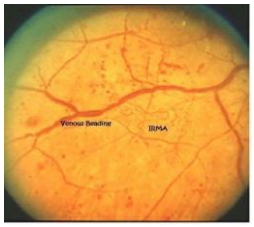


1. **Microaneurysms (earliest sign)**
   - Small red dots in superficial retina
   - Can rupture → hemorrhages
2. **Hemorrhages**
   - Dot and blot hemorrhages (deep retinal layers)
     - Similar to microaneurysms on fundus exam → **Fluorescein Angiography**
   - Flame-shaped hemorrhages (superficial retinal layers)

3. **Cotton-wool spots**
   - Nerve fiber layer infarction
   - Indicate ischemia → no capillary perfusion on fluorescein angiography



4. **Hard exudates**
   - Lipid deposits from vascular leakage
5. **Retinal edema** (Discussed below)
6. **Venous abnormalities** (beading, looping)
7. **Intraretinal microvascular abnormalities (IRMAs)**
   - Shunt vessels near non-perfused areas
   - Sign of **severe NPDR**



Circinate exudates

Diabetic Macular Edema (DME)

A major cause of vision loss in DR.

Definition

Retinal thickening and/or **hard exudates** in the macula.

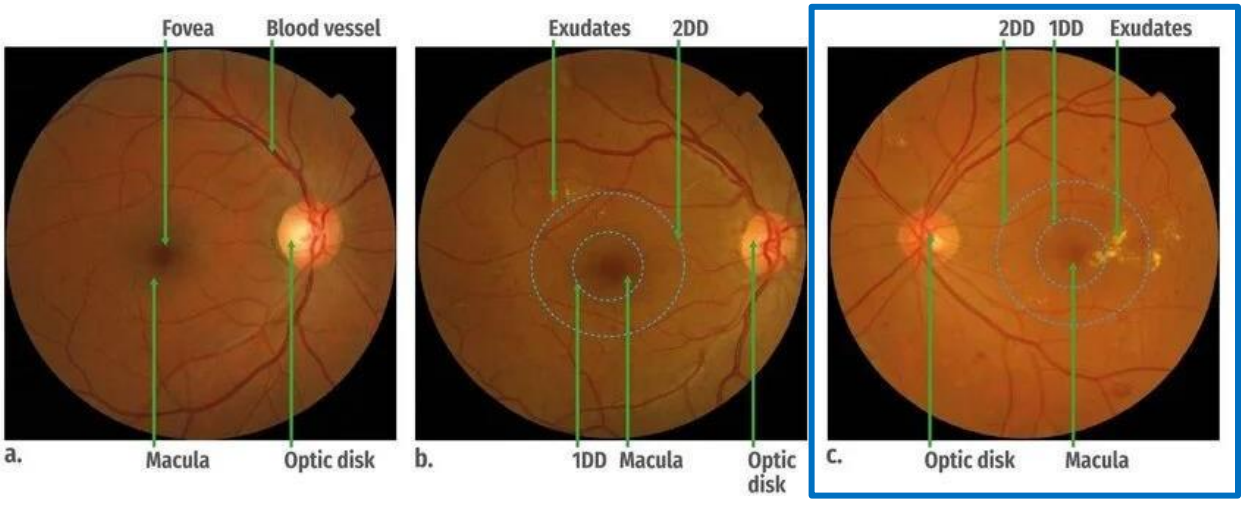
Severity Classification

- **Mild:** distant from macular center
- **Moderate:** approaching center
- **Severe:** involving the center

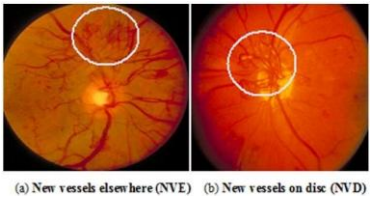
Clinically Significant Macular Edema (CSME)

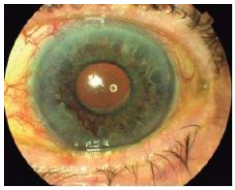
Defined by any ONE of the following (clinical diagnosis):

- Retinal thickening **within 500 µm of the center of the fovea**
- Hard exudates within 500 µm of the center of the fovea, if associated with adjacent retinal thickening
- **Retinal thickening ≥1 disc area with any part of it within 1500 µm (~1 disc diameter) of the center of the fovea**



2. Proliferative Diabetic Retinopathy (PDR)





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Late stage characterized by (on **Fundus Exam**):

1. **Neovascularization**
   - New vessels on disc (NVD)
   - New vessels elsewhere (NVE)
   - New vessels on iris (NVI / rubeosis iridis)
   - The new vessels break easily → hemorrhages
2. **Vitreous hemorrhage**
3. **Pre-retinal hemorrhage**
4. **Traction retinal detachment**
5. **Neovascular glaucoma**

Severity classification (AAO)

1. No DR

- No abnormalities

2. Mild NPDR

- **Microaneurysms** only

3. Moderate NPDR

- More than mild but less than severe
- Includes:
  - Hemorrhages/microaneurysms
  - Cotton-wool spots
  - Venous beading (in 1 quadrant)

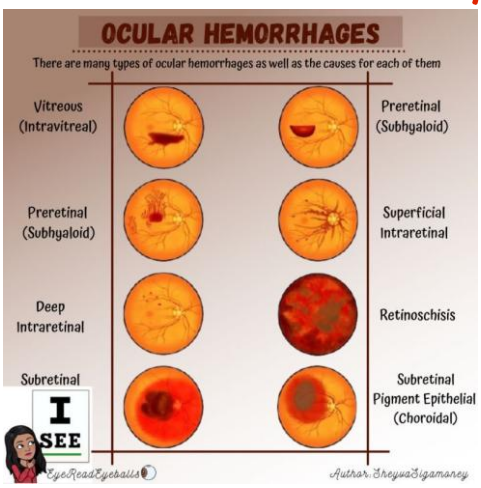
4. Severe NPDR (4-2-1 Rule)

Any one of (with no neovascularization):

1. ≥20 hemorrhages/microaneurysms in 4 quadrants
2. Venous beading in ≥2 quadrants
3. **IRMA** in ≥1 quadrant

5. Proliferative DR (PDR)

- Neovascularization
- Vitreous or pre-retinal hemorrhage



Follow-Up Recommendations

Severity	Follow-up
No DR	12 months
Mild NPDR	9 months
Moderate NPDR	6 months
Severe NPDR	4 months
PDR	Immediate referral
Clinically Significant Macular Edema (CSME)	Immediate referral

Management

A. General Measures

- Strict glycemic control (HbA1c)
- Control risk factors
- Regular ophthalmic follow-up

B. Treatment by Stage

1. Mild–Moderate NPDR

- No specific ocular treatment
- Focus on systemic control

2. Severe NPDR

- Close monitoring by ophthalmologist (every 4 months)

3. Diabetic Macular Edema (DME)

Treatment:

1st line →

- **Intravitreal anti-VEGF injections** (e.g., Avastin)
- **Focal/grid laser photocoagulation**
  - Laser targets leaking microaneurysms
  - Reduces edema and exudates over time (months)

4. Proliferative DR

Treatment:

- **Pan-retinal photocoagulation (PRP)**
  - Scattered laser burns to ischemic retina
  - Reduces VEGF production
- **Anti-VEGF therapy**
  - Causes regression of neovascularization

Symptoms

Early stages are typically **asymptomatic**.

Late symptoms include:

- Blurred vision
- Floaters
- **Poor color vision**
- **Poor night vision**
- Visual field defects
- **Sudden & total vision loss**

Investigations

1. Laboratory

- HbA1c
- Blood glucose

2. Imaging

- **OCT (Optical Coherence Tomography)**
  - Detects macular edema and thickness
- **Fluorescein angiography**
  - Identifies leakage and ischemia

Complications

- Vitreous hemorrhage
- Tractional retinal detachment
- Rubeosis iridis
- Neovascular glaucoma (complication of rubeosis iridis)
- Blindness

Causes of Blindness in DR

- Non-clearing vitreous hemorrhage
- Traction retinal detachment
- Macular ischemia
- Neovascular glaucoma

Prevention of Complications

- Early detection (screening)
- Annual fundus examination
- Tight glycemic control
- Early treatment of retinopathy
- Timely referral to ophthalmologist

Glaucoma

Introduction

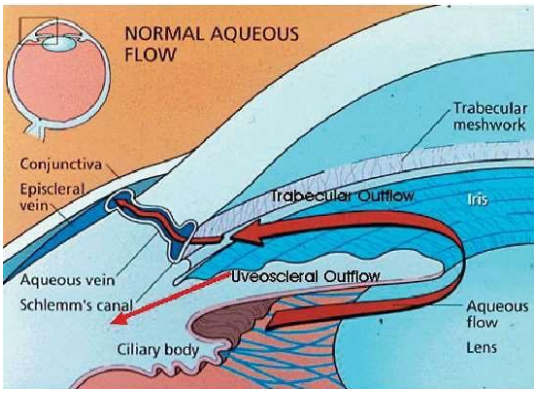
Glaucoma is a progressive **optic neuropathy** characterized by structural damage to the optic nerve head and corresponding visual field defects (from nerve fiber bundle loss). It is a leading cause of **irreversible blindness** worldwide. The disease often progresses silently, with peripheral vision loss preceding central vision impairment.

Diagnosis primarily depends on:

1. Optic nerve head appearance (**Cupping**)
2. Visual field assessment (**Visual field defects**)
3. **↑ IOP is not mandatory for diagnosis**

Prevalence

- **~6%** when defined by either optic nerve or field changes
- **~2.5%** when both criteria are present



Aqueous Humor Dynamics

Production

- Produced by non-pigmented ciliary epithelium
- Rate: ~2.5 µL/min
- Total volume: ~250 µL
- Turnover time: ~100 minutes
- ¼ is in PC & ¾ is in AC

Flow Pathway

Posterior chamber → through pupil → Anterior chamber

Drainage Pathways

- **Conventional (85%)**
  - Trabecular meshwork → **Canal of Schlemm** → Aqueous & Episcleral veins
- **Uveoscleral (15%)**
  - Through ciliary body & iris → Suprachoroidal space

Pathophysiology

- Elevated IOP usually results from **reduced outflow**, not overproduction

---

Risk Factors

Major Risk Factors

- Increasing age
- Ethnicity
- Family history
- Elevated intraocular pressure (**↑ IOP**) *(most important)*
- Trauma
- Previous eye surgery
- Medications (e.g., steroids)
- Refractive errors
- Diabetes

Non-IOP dependent Risk Factors

- Systemic vascular dysregulation (e.g., migraine, Raynaud's phenomenon, Prinzmetal angina)
- Nocturnal hypotension
- Sleep apnea

Classification of Glaucoma

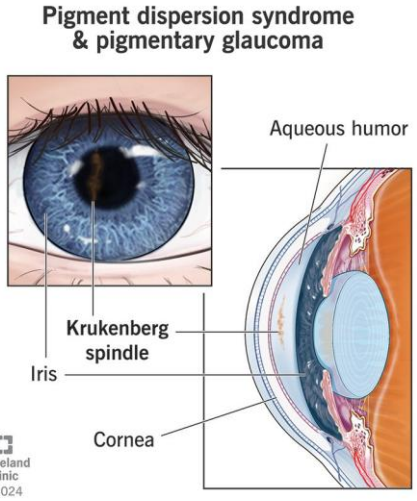
Based on Anatomy of Anterior Chamber Angle

1. Open-angle glaucoma (1° OR 2°)
2. Angle-closure glaucoma (with pupillary block OR without pupillary block) → (1° OR 2°)
3. Developmental glaucoma (1° OR 2°)
4. Mixed mechanism glaucoma

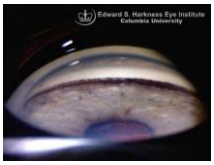
A. Open-Angle Glaucoma

1. Primary Open-Angle Glaucoma (POAG)

- Most common type
- Idiopathic increased outflow resistance
- Equal gender distribution
- More common in **myopes**
- Asymptomatic until late
- IOP: **typically ↑** (20–40 mmHg)



2. Secondary Open-Angle Glaucoma (SOAG)



Causes:

1. Trabecular Meshwork Obstruction

- Hyphema (RBCs)
- Uveitis (WBCs)
- Pigment Dispersion Syndrome (Pigment)
- Melanoma (Pigment)
- Pseudo-exfoliation Syndrome (Proteins)

2. Increased Episcleral Venous Pressure

- Carotid-cavernous fistula
- **Sturge-Weber syndrome**
- SVC obstruction

3. Structural Damage (Scarring)

- Angle recession (trauma)

4. Drug-induced

- Steroids

B. Angle-Closure Glaucoma

Predisposing Factors

- Shallow anterior chamber (**hyperopia**, short eye)
- Age > 60 years
- **Female** sex
- Asian/Inuit ethnicity
- Drugs (antihistamines, TCAs, MAOIs, sulfonamides)
- Mydriasis (mydriatics, darkness, stress/fear response)

Pathophysiology of Angle Closure

- Blocked trabecular meshwork → ↓ **aqueous drainage** → ↑ IOP

Types

1. Primary Angle-Closure Glaucoma:

- The chamber angle is narrowed due to peripheral iris obstructs trabecular meshwork

2. Secondary Angle-Closure Glaucoma:

Causes:

- Adhesions
  - Posterior Synechiae
  - Peripheral Anterior Synechiae

- Lens-related causes
  - Lens luxation
  - Large cataract
- **Rubeosis iridis (Neovascular Glaucoma)**

  Chronic Angle-Closure Glaucoma (مش بالسلايدات↓)
  - **Proliferative diabetic retinopathy** (PDR) (most common)
  - Central retinal vein occlusion (CRVO)
  - Central retinal artery occlusion (CRAO)
  - Ocular ischemic syndrome (carotid artery disease)
  - Other Causes
    - Retinal detachment
    - **Chronic uveitis**
    - Intraocular tumors
    - Post-surgical or traumatic ischemia

Diagnosis

A. History

1. Symptoms of acute angle-closure glaucoma

- Sudden onset
- Severe ocular pain
- Red eye
- Photophobia
- **Excessive tearing (injection)**
- Headache
- Nausea and vomiting
- Blurred vision
- Halos around lights

2. Symptoms of open-angle glaucoma

- Often **asymptomatic early**
- Gradual peripheral vision loss (tunnel vision)
- Blurred vision
- Halos around lights

3. Ocular History

- Trauma
- Previous laser treatment
- Ocular surgery
- Other eye diseases

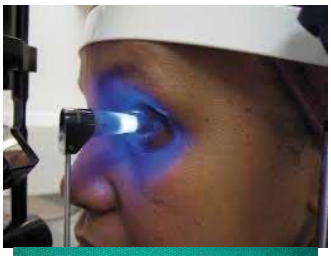
4. Medical History

- Diabetes
- Hypertension
- Asthma
- Drug allergies

B. Clinical Examination

1. Visual Assessment

- Best corrected visual acuity (BCVA)
  - Reduced visual acuity
- Relative afferent pupillary defect (RAPD)
  - Non-reactive, fixed, **mid-dilated** oval pupil
- Refractive status (myopia vs hyperopia)



2. Measurement of Intraocular Pressure (IOP)

- Methods
  - Gold standard → **Goldmann Applanation Tonometry (GAT)**
  - Alternative → **Tonopen**
- Normal range: **10–21 mmHg**. Diurnal variation present
- Elevated IOP is **not mandatory** for diagnosis
- Elevated IOP is a major modifiable risk factor, but:
  - **Normal tension glaucoma** → damage without high IOP
  - **Ocular hypertension** → high IOP without damage
- Higher IOP → higher risk
- IOP **>40 mmHg** in acute attacks

3. Pachymetry

- Measures central corneal thickness (CCT)
- Important for interpreting IOP
- Corneal thickness affects IOP readings:
  - Thin cornea → falsely low IOP
  - Thick cornea → falsely high IOP
- Not yet standard of care

4. Slit Lamp Examination → **Classify glaucoma based on anterior chamber angle**

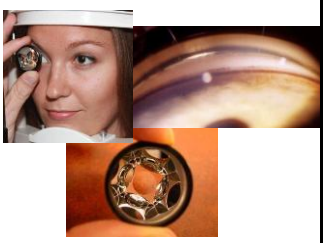
Purpose:

1. Classify glaucoma based on anterior chamber angle
2. Look for 2° glaucoma causes

Key findings for glaucoma:

- Cloudy (edematous) cornea
- Shallow anterior chamber

5. Gonioscopy → **Classify glaucoma based on anterior chamber angle**



- Essential for all suspected cases.

Purpose:

1. Classify glaucoma based on anterior chamber angle
2. Look for 2° glaucoma causes

6. Optic Nerve Head Evaluation→ **Staging glaucoma based on ONH damage**

- Method: **Fundus Exam**

Key findings:

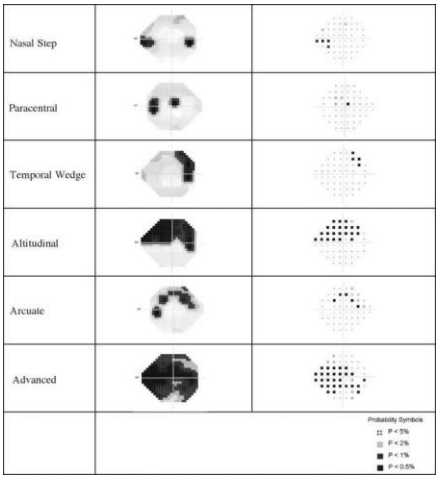
1. Increased **cup-to-disc ratio** (>0.6 suspicious → **Cupping**)
2. **Asymmetry** between eyes (>0.2 difference)
3. **Notching**
4. **Neuroretinal rim thinning** (ISNT rule violation)
   - In a healthy eye, the neuro-retinal rim (NRR) thickness decreases in the order of:
     Inferior > Superior > Nasal > Temporal
5. **Optic disc hemorrhages** (Drance hemorrhage)

7. Visual Field Testing → **Staging glaucoma based on VF damage**

- Detects functional loss before symptoms.

Purpose:

1. Stage disease based on VF damage



Typical defects:

- Nasal step
- Paracentral scotoma
- Arcuate scotoma
- Altitudinal defect
- Tunnel vision in advanced stages

8. **Optic Nerve Head Imaging**
   A. Heidelberg Retinal Tomography (HRT)
   B. GDx (polarimetry)
   C. **Optical Coherence Tomography (OCT)**
      - Measures **retinal nerve fiber layer (RNFL) thickness**
      - Findings:
        - RNFL thinning (early structural damage)
        - Ganglion cell loss

Management

Principles of Treatment

- Optic nerve damage is **irreversible**
- Goal: **Preserve remaining vision**
- Strategy: **Lower IOP**

Treatment Modalities

A. Medical Treatment

1. **Prostaglandin Analogues**
   - First-line
   - Increase uveoscleral outflow
   - Once daily

   Side effects:
   - Conjunctival Hyperemia
   - Iris pigmentation (irreversible)
   - Eyelash growth
   - Periorbital fat loss → deepening of upper lid sulcus
   - Hyperpigmentation of periocular skin

2. **Beta Blockers**
   - Decrease aqueous production
   - Twice daily

   Contraindications:
   - Asthma
   - Bradycardia
   - Heart block

3. **Alpha-2 Agonists**
   - Decrease aqueous production
   - Neuroprotective

   Side effects:
   - Allergic conjunctivitis
   - Contact dermatitis

4. **Carbonic Anhydrase Inhibitors**
   - Topical or systemic (Acetazolamide)
   - Decrease aqueous production
   - Sulfa allergy risk

5. **Cholinergic Agents (Pilocarpine)**
   - Increase trabecular outflow
   - Cause miosis

   Side effects:
   - Myopia
   - Risk of retinal detachment

6. IV Mannitol

- ↑ plasma osmolarity → draws water from vitreous → ↓ vitreous volume → ↓ IOP

Side effects:

- Pulmonary edema (volume overload)
- Electrolyte imbalance (Na^+, K^+)
- Dehydration
- Headache, confusion
- AKI

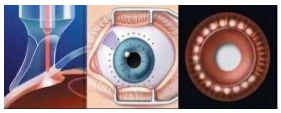
B. Laser Therapy

1. Increase Outflow:

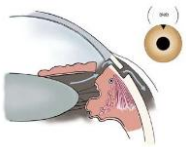
- Trabeculoplasty
- **Laser Peripheral Iridotomy (YAG laser)**

2. Decrease Production:

- Cyclodiode laser



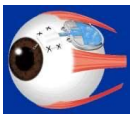
C. Surgical Treatment



1. Trabeculectomy

- Creates new drainage pathway
- Used in advanced/uncontrolled cases

2. Glaucoma Drainage Devices



D. Preventive Measures

- Examine the fellow eye
- Prophylactic treatment if necessary

E. Screening Recommendations (high-risk individuals)

- Age > 40
- Family history
- Diabetes, hypertension
- High myopia
- African or Asian ancestry

Complications of Glaucoma

- **Irreversible optic nerve damage**

Key Clinical Principles

1. Every patient has glaucoma until proven otherwise.
2. IOP is not sufficient nor necessary for diagnosis of Glaucoma.
   However, it is the most important modifiable risk factor.
3. There are over 100 subtypes of glaucoma → Classify type before treatment.

Uveitis

Introduction

- Uveitis refers to **inflammation of the uvea**, which is the **middle vascular layer of the eye**.
- The term *uvea* comes from Latin, meaning **"grape"**, reflecting its anatomical appearance.

Anatomy of the Uvea

The uvea consists of three main structures:

- **Iris**
- **Ciliary body**
- **Choroid**

These structures are highly vascular and play roles in:

- Aqueous humor production
- Light regulation
- Nutritional support of the retina

Classification of Uveitis

A. Anterior Uveitis

- Primary site: **Anterior chamber**
- Includes:
  - Iritis
  - Anterior cyclitis
  - Iridocyclitis

B. Intermediate Uveitis

- Primary site: **Vitreous**
- Includes:
  - Pars planitis

C. Posterior Uveitis

- Primary site: **Choroid**
- Includes:
  - Choroiditis
  - Retinitis
  - Chorioretinitis

D. Panuveitis

- Involves all parts of the uvea

Etiology (Causes)

Main categories:

1. Infectious causes
2. Systemic immune-mediated diseases
3. **Ocular Syndromes (Eye-limited Uveitis)**
4. Idiopathic

Epidemiology

Distribution:

- Anterior: **52%**
- Intermediate: **13%**
- Posterior: **19%**
- Panuveitis: **16%**

Common Associations

Anterior Uveitis Causes

- **Idiopathic** (most common)
- **Seronegative spondyloarthropathies**
- **Sarcoidosis**
- **Juvenile idiopathic arthritis** (silent uveitis)
- SLE
- **Behçet disease**
- AIDS

Posterior Uveitis Causes

- **Toxoplasmosis** (most common)
- Idiopathic
- **CMV**
- SLE
- **Sarcoidosis**

Panuveitis Uveitis Causes

- **Sarcoidosis**
- **Behçet disease** (most dangerous uveitis → severe, recurrent & often bilateral)
- VKH syndrome

Clinical Features

A. History

1. Symptoms

A. Acute Anterior Uveitis

- **Eye pain**
- **Redness**
- **Photophobia**
- **Excessive tearing (Epiphora)**
- Decreased vision

B. Chronic Anterior Uveitis

- **Blurred vision**
- Mild redness
- Minimal pain or photophobia

C. Intermediate Uveitis

- **Floaters**
- **Painless vision loss**

D. Posterior Uveitis

- **Blurred vision**
- **Floaters** (عوائم العين)
- Less pain

E. Intermediate Uveitis

- May present with any or all of these symptoms

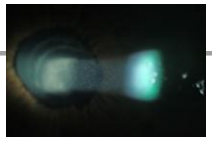
2. Medical Hx

- Important because **~50% have systemic disease.**
- Key systems to evaluate:
  - Respiratory → TB, Sarcoidosis …
  - Skin → Behçet disease, Sarcoidosis …
  - Joints → Ankylosing spondylitis, JIA …
  - GI → IBD …
  - Infectious → STDs, HIV, Herpes, fungal infections, Syphilis …

B. Physical Examination

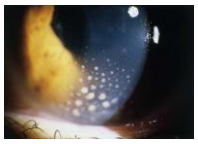
I. General Sign of Uveitis

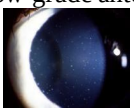
- Reduced visual acuity



II. Signs of Anterior Uveitis

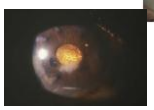
- Slit Lamp Examination → **Aqueous cells & flare**
  - Indicates inflammatory process (exudate) in anterior chamber
- **Ciliary injection** (redness near limbus)
- Pupillary exam → **Miosis** (**common**) & **Photophobia** (direct and consensual)
- **Hypopyon**
- **Keratic precipitates (KPs)**
  - Clumps of inflammatory cells on corneal endothelium
  - Usually inferiorly distributed
  - Large keratic precipitates (KPs) → **Sarcoidosis**
  - **Fuchs heterochromic iridocyclitis**
    - A form of chronic, low-grade anterior uveitis that is typically unilateral and often asymptomatic.
    - Fine, stellate KPs
    - Diffuse distribution



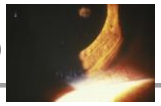


- **Posterior synechiae (التصاق القزحية)**
  - Iris–lens adhesion
  - Indicates chronic or recurrent inflammation, but may occur in severe acute cases
  - Risk of 2nd ry angle-closure **Glaucoma (Pupillary block glaucoma)**

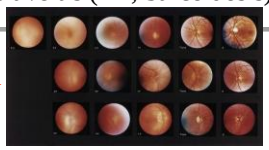
- **Peripheral anterior synechiae**
  - Iris–cornea adhesion or Iris-trabecular meshwork adhesion
  - Risk of 2nd ry angle-closure **Glaucoma (Drainage angle glaucoma)**



- **Iris Nodules**
  - Indicates → Granulomatous uveitis (TB, Sarcoidosis)



III. Intermediate & Posterior Uveitis Signs



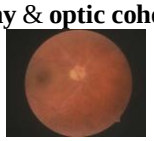
Main:

- **Vitreous cells**
- **Retinal/choroidal foci of inflammation**
  - Yellow-white lesions in retina or choroid on fundoscopy
- **Cystoid macular edema**

  - Central vision loss
  - Detected by **fluorescein angiography** & **optic coherence tomography**

Other:

- **Retinal vascular sheathing**
  - Seen in **idiopathic retinal vasculitis**

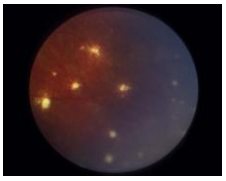


- **Exudative retinal detachment**
  - Seen in **Vogt–Koyanagi–Harada syndrome (VKH)**
  - Typically seen inferior to macula

- **Fibrotic band (tractional change)**
  - Dense fibrous tissue extending from disc to arcade

- **Choroidal granuloma (Sarcoidosis)**
  - Large yellow elevated lesion near optic disc

- **Dalen–Fuchs nodules**
  - Small, discrete yellow-white lesions (often peripheral)
  - Located between Retinal Pigment Membrane and Bruch's membrane
  - Composed of inflammatory cell collections
  - Seen in conditions like **VKH** and **sympathetic ophthalmia**



C. Investigations (When to test)

- **No lab work needed** in mild, unilateral, non-granulomatous uveitis if:
  - Trauma present
  - Known systemic disease
  - No systemic features on history/exam
- **Do investigations if uveitis is:**
  - Bilateral
  - Granulomatous (iris nodules)
  - Recurrent

Management

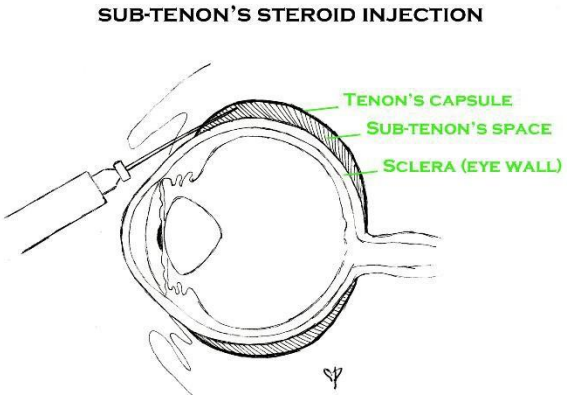
Treatment Goals

- Relieve pain and inflammation
- Prevent damage (especially to macula & optic nerve)

Main Treatment

A. First-line

- **Cycloplegics (e.g., atropine, cyclopentolate)**
  - Relieve ciliary spasm pain
  - Prevent posterior synechiae
- **Steroid Therapy (mainstay of treatment)**
  - Anterior uveitis → Steroid eye drops
  - Posterior uveitis → Systemic steroids OR **Sub-tenon's steroid injection**



B. Synechiae management

- **Intensive mydriatics drops** (cyclopentolate + phenylephrine)
  - Subconjunctival injection if resistant
  - Otherwise, synechiae will interfere with normal dilatation of the pupil

C. Additional treatment

- Antibiotics/antivirals if infectious cause
- Immunosuppressants (e.g., azathioprine, cyclosporine) in severe cases like Behçet's disease

Complications

- **Cataract** (from steroids or inflammation)
- **Glaucoma** (especially from synechiae)
- **Permanent vision loss** (**posterior uveitis**)
  - Macular edema
  - Retinal destruction (e.g., toxoplasma, CMV)

Etiology (Causes)

Main categories:

1. Infectious causes
2. Systemic immune-mediated diseases
3. **Ocular Syndromes (Eye-limited Uveitis)**
4. Idiopathic

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1. Systemic immune-mediated causes

A. Seronegative Spondyloarthritis (SpA) → **30%** of SpA patients have uveitis

- Spondyloarthritis accounts **~7%** of all uveitis cases

Types:

1. Ankylosing spondylitis
2. Psoriatic arthritis
3. Reactive arthritis (Triggered by GI or GU infections)
4. IBD-associated arthritis (20% of IBD patients)

Major Clinical Features:

- Inflammatory back pain
  - Insidious onset
  - Pain at night
  - Age <40
  - Improves with exercise
  - No improvement with rest
- Peripheral arthritis
  - Asymmetric
  - Oligoarticular
  - Large joints
  - Lower limbs > upper
- Dactylitis
- Enthesitis
- **Acute unilateral anterior uveitis** (most common extra-articular manifestation)
  - Recurrence rate: **~50%**
  - **~50% of acute anterior uveitis** cases are **HLA-B27+**

B. IBD

Types:

1. Crohn disease → **4.5%** of CD patients have uveitis non-anterior types
2. Ulcerative colitis → **2%** of UC patients have uveitis

Notes:

- Many IBD + uveitis patients fall under IBD-associated SpA

C. Sarcoidosis → **35%** of sarcoidosis patients have uveitis

- Any ocular involvement in sarcoidosis **~50%**. Uveitis is most common ocular manifestation
- Sarcoidosis accounts for **~7%** of all uveitis cases

Type of uveitis in sarcoidosis

- Usually: **Chronic bilateral granulomatous uveitis**
- Can involve **any part of the eye**:
  - Anterior (Anterior > Posterior)
  - Intermediate
  - Posterior
  - Panuveitis

D. Behçet Disease → **60%** of Behçet disease patients have uveitis

- Behçet Disease accounts for **~7%** of all uveitis cases
  - Higher in endemic regions (e.g., Silk Road countries)
- Ocular involvement in these patients: **Severe Uveitis** + **Retinal Vasculitis**
- Most dangerous uveitis

Type of uveitis in Behçet Disease

- Classically: **Severe recurrent bilateral uveitis**
- Distribution:
  - **Panuveitis** (most characteristic)
  - Posterior uveitis (common)

Hallmark features

- **Retinal vasculitis** (often occlusive)
- Explosive attacks with rapid vision loss
- Hypopyon may occur (classically shifting)

E. Juvenile Chronic Arthritis (JIA) → **~15%** of JIA patients have uveitis

General Features:

- Occurs in children → **~30%** of all uveitis cases in pediatrics
- Seronegative (**RF -ve** & **ANA** may be **+ve**)

Types:

- Systemic
- **Pauciarticular** → **~25%** of Oligoarticular JIA patients have uveitis (Highest)
- Polyarticular

Type of uveitis

- **Chronic bilateral anterior uveitis**
- Usually:
  - **Asymptomatic (silent)** → **Screening (Regular Slit-Lamp Examination based on risk)**

Risk factors for uveitis in JIA patients

- ANA positive
- Early onset (<6 years)
- Female sex

Complications

- Cataract
- Glaucoma
- Band keratopathy

2. Ocular Syndromes

A. Fuchs' Heterochromic Uveitis

- Fuchs' Heterochromic Uveitis accounts for **~4%** of all uveitis cases

General:

- Rare
- Affects young adults
- **Mild chronic unilateral anterior uveitis**
- No systemic association

Symptoms:

- **Heterochromia**
- Blurred vision
- Floaters

Signs:

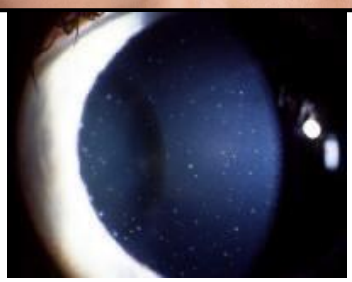
- Signs of anterior uveitis
- **Diffuse stellate keratic precipitates**

Complications:

- Cataract (~70%)
- Glaucoma

Treatment:

- Steroids ineffective
- Cataract surgery effective
- Treat glaucoma conventionally



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3. Infectious Causes

A. Toxoplasmosis→ **~2%** of toxoplasmosis patients have uveitis

- Most common **infectious cause**
- Most common cause of **posterior uveitis** globally (**~35%**)

Clinical Features of Toxoplasmosis:

- Usually **asymptomatic**
- Infectious mononucleosis (glandular fever)–like presentation:
  - Fever
  - Fatigue
  - Cervical lymphadenopathy

Clinical Features of Ocular Toxoplasmosis:

- Hazy vision
- Floaters
- Red painful eye
- Photophobia

Type of uveitis

- **Posterior Uveitis (Retinochoroiditis)**

Types:

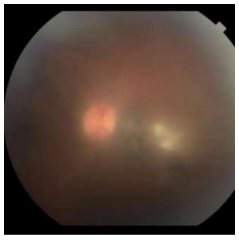
1. Congenital toxoplasmosis

- Laterality: typically **bilateral**
- Onset: lesions may be present at birth or appear later (childhood/adolescence)
- **Lesion pattern:**
  - Recurrent inflammation at **old pigmented scar** margins (often macular)
- Severity: generally **more severe**
- Associations: **CNS involvement** (classic triad)
- Visual outcome: often **worse visual outcome** due to macular involvement

Fig. 9.3 The appearance of an inactive toxoplasma retinitis.

2. Acquired toxoplasmosis

- Laterality: usually **unilateral**
- Onset: acute symptomatic episode (young adults)
- Lesion pattern:
  - **Focal necrotizing retinochoroiditis**
    - Appear as creamy focus of inflammatory cells at the margins of old chorioretinal scar (such scar usually atrophic, with a pigmented edge)
  - Overlying **vitritis** → classic **"Headlight in the fog" appearance**
    - Appear as bright retinal lesion + hazy vitreous



- Severity: usually **less severe** initially
- Associations: typically no systemic/CNS disease
- Visual outcome: **better visual outcome** unless macula involved

Complications

- **Recurrent uveitis** (hallmark)
- Retinal scarring → permanent vision loss
- Secondary complications:
  - Glaucoma
  - Cataract
  - **Retinal detachment** (less common)

Diagnosis

- Primarily **clinical (fundoscopy)**
- Supported by:
  - Serology for Toxoplasma gondii
  - Ocular fluid PCR

Treatment (when vision-threatening)

- Anti-parasitic → Pyrimethamine + Sulfadiazine + Leucovorin
- Add **corticosteroids** (after antiparasitic therapy started)
- Alternatives: TMP-SMX

B. HIV/AIDS-related Ocular Conditions

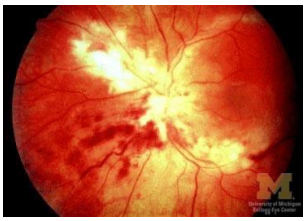
- Ocular disease is a common manifestation of AIDS
- Patients develop a variety of ocular conditions:
  1. Microvascular occlusion → **retinal hemorrhages and cotton wool spots** **(**Infarcted areas of the nerve fiber layer of the retina)
  2. Corneal endothelial deposits
  3. Neoplasms of the eye and orbit
  4. Neuro-ophthalmic disorders including oculomotor palsies
  5. **Opportunistic infections** of which the most common is **CMV retinitis**

History

- The patient may complain of:
  - **Blurred vision**
  - **Floaters**
- A diagnosis of HIV disease has usually already been made, often **with other AIDS-defining features**

Opportunistic infections

- **CMV**, toxoplasmosis, HSV & VZV
- **CD4+ <50/ml**
- Occurs in **more than 1/3 of AIDS patients**
- Reduced after HAART



Fundus appearance

- Mixture of:
  - Cotton wool spots
  - Infiltrates
  - Hemorrhages
- This combination spells **death for the retina**
- The virus gets into the vascular endothelium, closes off blood vessels, and spreads through tissue rapidly
- The entire retina can be destroyed **within weeks**
- Earliest sign may be a **cotton wool spot**
  - In early stages there is diagnostic problem. Cotton wool spot also seen in **non-infectious** microvascular occlusion
  - In severely immunocompromised patients → **presume early CMV retinitis** and monitor carefully

Treatment

- Parenteral **Ganciclovir**, **Foscarnet** or **Cidofovir**
- Oral **Valganciclovir**

Prognosis

- **Prolonged treatment is required** to prevent recurrence

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4. Idiopathic Uveitis

- Diagnosis after exclusion
- Large proportion of cases

Eye Injuries

Introduction

Eye injuries are common clinical conditions that may range from minor superficial damage to severe vision-threatening emergencies. Although the eyes are anatomically protected, they remain vulnerable due to their exposed position.

General Approach to Eye Injuries

1. Assess ABCs first
2. Identify life-threatening conditions
3. Evaluate visual acuity
4. Perform focused ocular examination
5. Identify **vision-threatening** injuries
6. Initiate urgent management where needed

Common Eye Injuries

1. Eyelid Lacerations

Eyelid injuries are frequently associated with deeper ocular damage, occurring in up to **two-thirds of cases**.

Types:


- Full-thickness lacerations
- Lacerations with orbital fat prolapse
- Lid margin lacerations
- Injuries involving the lacrimal drainage system
- Poorly aligned or avulsion injuries

These injuries require careful evaluation due to cosmetic and functional implications.

2. Corneal Abrasions

A corneal abrasion is defined as **loss of the corneal epithelial layer**.

Clinical features:

- **Severe eye pain**
- **Photophobia** (difficulty opening the eye)
- Foreign body sensation
- Typically normal visual acuity
- Normal pupillary response
- **Fluorescein staining defect**

3. Corneal Foreign Bodies

Foreign bodies embedded in the cornea may cause:

- Irritation
- Pain
- Risk of infection

Prompt removal is necessary to prevent complications such as corneal ulceration.

4. Conjunctival Injuries

These include:

- **Subconjunctival hemorrhage**
- Lacerations

Usually **less severe** but may mask deeper injuries.

5. Orbital Fractures

Fractures of the orbital bones may result in:

- Diplopia
- Enophthalmos
- Entrapment of extraocular muscles

Vision-Threatening Conditions

These require **immediate recognition and intervention**

1. Ocular Chemical Burns

Cause

Exposure to:

- Acids
- Alkalis (more severe damage)

Clinical Presentation

- Decreased vision
- Moderate to severe pain
- **Blepharospasm** (inability to open eyelids)
- Conjunctival redness
- Photophobia
- **Symblepharon** (adhesion of eyelid to globe)

In severe alkali injuries:

- Eye may appear **white** due to ischemia

Management

- Immediate **continuous irrigation** with water or saline
- Aim for **neutral pH (30–60 minutes)**
- Use of **Morgan lens** if available
- Avoid forceful irrigation if globe rupture suspected

2. Orbital Compartment Syndrome (OCS)

Definition

A **true ophthalmologic emergency** caused by increased intraorbital pressure leading to ischemia of the optic nerve.

It is clinical diagnosis.

Pathophysiology

- Trauma → hemorrhage → increased orbital pressure
- Compression of optic nerve → vision loss

Clinical Features

- Sudden decreased vision
- Diplopia
- Eye pain
- **Proptosis**
- Periorbital swelling
- **Subconjunctival hemorrhage**
- **Chemosis**
- Ophthalmoplegia
- **Afferent pupillary defect**
- Tight eyelids
- Resistance to globe retropulsion

Management

Immediate decompression is critical:

- Lateral canthotomy
- Inferior cantholysis

Additional management:

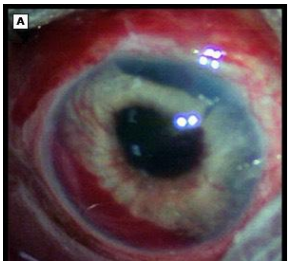
- Hospital admission
- **Elevate head (≥45°)**
- Control intraocular pressure
- Pain management
- Prevent pressure spikes (antiemetics, stool softeners)
- Correct coagulopathy
- Stop anticoagulants if necessary
- Imaging (CT/MRI; avoid MRI if metallic foreign body suspected)

3. Open Globe Injuries

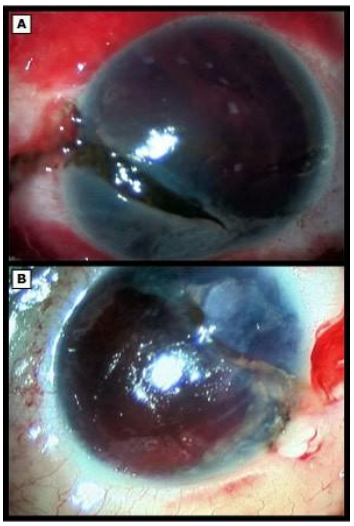
Definition

Open globe injuries involve **full-thickness disruption of the eye wall** and are classified into:

- **Rupture** → due to blunt trauma
- **Laceration** → due to sharp object



Rupture



Lac.

Types of lacerations:

- Penetrating (entry only)
- Perforating (entry + exit)
- Corneal
- Corneoscleral
- Scleral

Clinical Features

- Markedly decreased vision
- Relative afferent pupillary defect (RAPD)
- **Teardrop pupil** or **Irregular pupil**
- Vitreous extrusion
- Prolapse of uveal tissue (iris/choroid)
- Low intraocular pressure
- **Positive Seidel sign**

Associated **Rupture** findings may include:

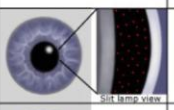
- Subconjunctival hemorrhage
- **Hyphema**
- Iridodialysis (separation of iris)

Management

- **Emergency ophthalmology referral**
- Keep patient **NPO**
- **Broad-spectrum antibiotics**
- **Tetanus prophylaxis**
- **Primary surgical closure within 24 hours**

Early closure improves visual outcome significantly

Traumatic hyphema: Grading and prognosis

Grade	Anterior chamber filling	Diagram	Best prognosis for 20/50 vision or better
Microhyphema	Circulating red blood cells by slit lamp exam only		90 percent
I	<33 percent		90 percent
II	33-50 percent		70 percent
III	>50 percent		50 percent
IV	100 percent		50 percent

4. Traumatic Hyphema

Definition

Blood in the **anterior chamber**:

- Macroscopic (visible layering)
- Microhyphema (seen only on slit lamp)

Causes

- Blunt trauma → tearing of iris or ciliary body vessels

Clinical Features

- Visible blood level
- Photophobia
- Decreased vision
- **Anisocoria**
- Increased intraocular pressure

Management

Goal: **prevent complications** (**rebleeding**, **glaucoma**)

Conservative:

- Eye shield
- Bed rest / activity restriction
- Monitor IOP
- **Cycloplegics** (pain control)
- **Steroid eye drops**

Surgical indications:

- Large hyphema **≥ grade III (50%) >10 days**
- Corneal blood staining
- **IOP ≥ 50 mmHg >5 days** despite treatment

5. Vitreous Hemorrhage

Definition

Blood within the vitreous cavity.

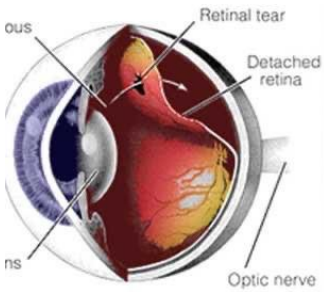
Clinical Importance

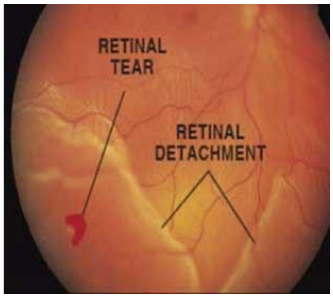
- Suggests **retinal tear or retinal detachment**
- May be associated with:
  - Head trauma
  - Subarachnoid/subdural hemorrhage
  - Abusive injury in children

6. Retinal Trauma

Effects

- Partial or complete vision loss





A. Retinal Tear

B. Retinal Detachment

Symptoms:

- Flashes
- Floaters
- Visual field defects

Management:

- Bed rest
- **Urgent surgical repair**
- **Immediate surgery if macula threatened**

C. Commotio Retinae (ارتجاج الشبكية)

- Retinal edema after blunt trauma
- May be asymptomatic or ↓ vision
- Fundoscopy: **whitish retina with normal vessels**
- Usually resolves spontaneously
- Must exclude retinal tear/detachment

7. Optic Nerve Injury (Traumatic Optic Neuropathy)

Clinical Features

- Decreased vision
- **Reduced color vision** (red desaturation)
- **Afferent pupillary defect**

Mechanisms

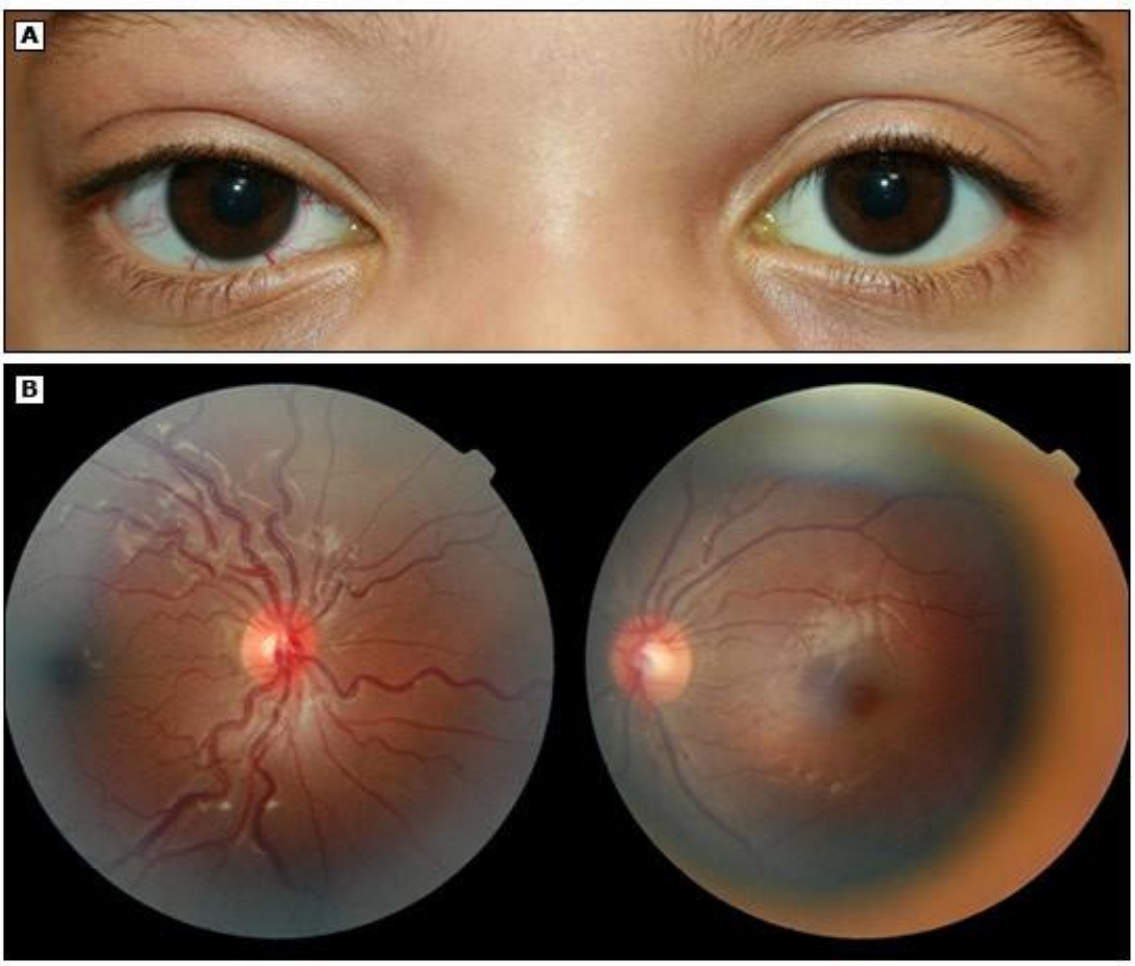
- **Direct injury** → rare (penetrating trauma)
- **Indirect injury** → common (blunt trauma to brow/temple)

8. Periocular Vision-Threatening Conditions

Example: **Carotid–cavernous fistula**

Features

- **Tortuous "corkscrew" conjunctival vessels**
- Chemosis
- Proptosis
- Dilated retinal vessels
- Increased IOP



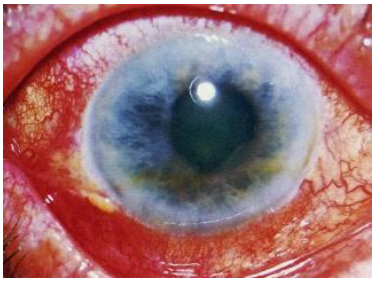
Red Eye

Benign Causes

- Stye (hordeolum)
- Chalazion
- Blepharitis
- Subconjunctival hemorrhage
- Conjunctivitis:
    - Bacterial
    - Viral
    - Allergic
- Dry eye
- Urgent follow-up needed
    - Corneal abrasion
    - Corneal foreign body
    - Contact lens overwear

Serious Causes (Require Urgent/Emergency Care)

- Emergency Care
    - Angle-closure glaucoma
    - Hyphema
    - Hypopyon
    - Infectious keratitis (Bacterial)
- Urgent Care
    - Iritis
    - Scleritis
    - Infectious keratitis (Viral)



Loss of Vision

Definition of Vision Impairment

According to **(ICD-10, 2018)**:

- **Distance vision impairment** → Visual acuity worse than **6/6** using a Snellen chart
- **Near vision impairment** → Visual acuity worse than **N6 or M.08** despite correction

Severity Classification (Distance Vision)

Category	Visual Acuity
Mild	≤ 6/18 (0.3)
Moderate	< 6/18 to ≥ 6/60 (0.3-0.1)
Severe	< 6/60 to ≥ 3/60 (0.1-0.05)
Blindness	< 3/60 to No light perception (0.05-0)

Classification of Vision Impairment

A. By Onset

1. Sudden Vision Loss

Occurs over seconds to days.

a. Transient (< 24 hours) = STVL = Amaurosis Fugax

1. Vascular → Carotid disease, Emboli, Giant Cell Arteritis (GCA), Vasospasm
2. Neurogenic → Retinal Migraine
3. Ophthalmic → Papilledema, Subacute Angle-closure Glaucoma, Optic Disc Drusen

b. Persistent (> 24 hours) = SPVL

1. Acute Angle-closure glaucoma
2. Microbial keratitis
3. Acute Anterior Uveitis
4. Endophthalmitis
5. Hyphema
6. Vitreous Hemorrhage
7. Retinal vascular occlusions → CRAO, BRAO, CRVO, BRVO
8. Anterior ischemic optic neuropathy
9. **Rhegmatogenous Retinal detachment**
10. Optic neuritis

2. Gradual Vision Loss

- Progressive over weeks to years
- Usually painless
- Often bilateral

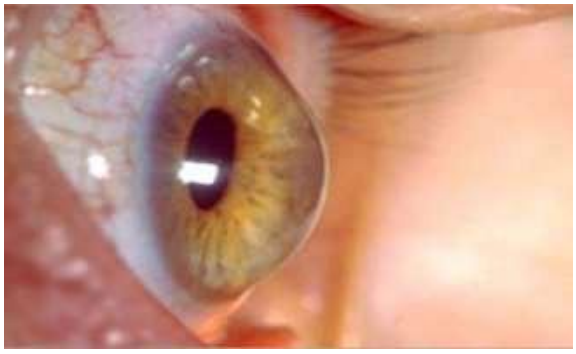
B. By Etiology

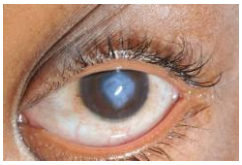
1. Uncorrected Refractive Errors (Ametropia)

Failure of parallel light rays to focus on the retina with eyes at rest.

Most Common Types:

1. **Myopia** (most common ametropia)
2. Hyperopia
3. Astigmatism
   - **Keratoconus → Myopia + Astigmatism**
4. Presbyopia



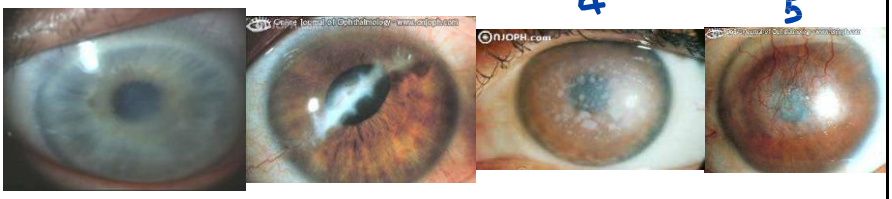

Hydrops Keratoconus

2. Media Causes

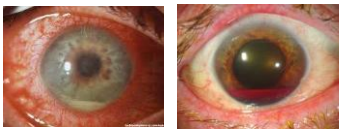
A. Precorneal Tear Film Causes

B. Corneal Causes

1. Corneal edema
2. Corneal Scar
3. Keratitis
4. Corneal Dystrophies
5. Corneal Degeneration



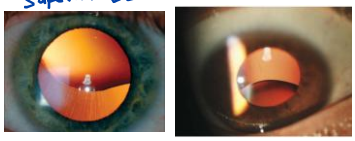
C. Anterior Chamber Disorders



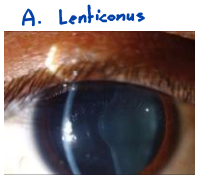
1. Anterior uveitis
2. Hypopyon
3. Hyphema, causes:
   - **Trauma** (most common cause)
   - Non-traumatic
     - **Rubeosis Iridis** (most common non-traumatic)
     - Anterior Uveitis
     - Tumors
     - Blood disorders (SCD)
     - Vascular abnormalities
     - Medications

D. Lens Disorders

1. **Cataract** (most common reversible cause of vision loss)
2. **Ectopia lentis** (lens displacement), causes:
   - Trauma
   - CTD → Marfan Syndrome
   - Metabolic → Homocystinuria
   - Congenital

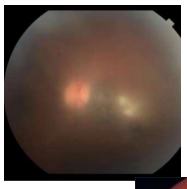


3. Shape abnormalities (**Lenticonus**)
   - Types: Anterior **Lenticonus & Posterior Lenticonus**
   - **Cause → ↑ blood sugar (reversible within days-weeks after normalization)**

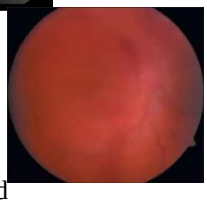


E. Vitreous Causes

1. Vitritis
   - Infection → Toxoplasmosis / Endophthalmitis
   - Autoimmune → Behçet Disease / Sarcoidosis



2. Vitreous hemorrhage, causes:
   - Trauma
   - Non-traumatic
     - Complicated PDR (NVDs,NVEs)
     - Wet AMD (Choroidal Neovascularization)
   - Notes:
     - Vision loss severity depends on the amount of blood
     - ± ↓ Red Reflex / Retina may be invisible by fundus exam



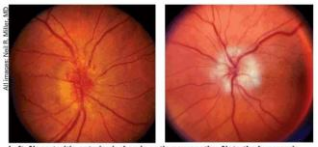
3. Retinal Causes

1. Diabetic Retinopathy (DR)
2. Retinal Vascular Occlusions → CRAO, BRAO, CRVO, BRVO
3. Age-related Macular Degeneration (AMD)
   - Dry AMD (Atrophic)
   - Wet AMD

4. Macular diseases
    - Macular hole
    - Epiretinal membrane
5. Posterior Uveitis
6. Retinal Dystrophies
    - Retinitis Pigmentosa
7. Macular Dystrophies
    - Stargradt's Disease
8. **Retinal Detachment**

4. Optic Nerve Disorders (OND)

1. **Optic neuritis**
    - Most common cause of OND in young adults
    - Associated with **MS**
    - Presenting complaint in **15%** of MS patients, affects **50%** of MS patients at some point
    - Symptoms (sudden unilateral):
        - **Pain** on eye movement
        - ↓ Visual acuity
        - ↓ Color vision
    - Signs:
        - **RAPD is present**
        - **Optic disc is normal**
2. **Ischemic optic neuropathy**
    - Most common cause of OND in elderly
    - Types → Anterior (affecting optic disc) & Posterior (retrobulbar)
      Arteritic & Non-arteritic
    - Symptoms (sudden unilateral):
        - **Painless**
        - ↓ Visual acuity
        - ↓ Color vision
    - Signs:
        - **RAPD is present**
        - **Optic disc swelling**



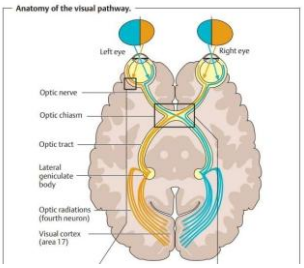
Left: Nonarteritic anterior ischemic optic neuropathy. Note the hyperemic swelling of the optic disc associated with the flame-shaped peripapillary hemorrhage. Right: Arteritic anterior ischemic optic neuropathy. Note the pallid swelling of the optic disc and a peripapillary cotton-wool spot.

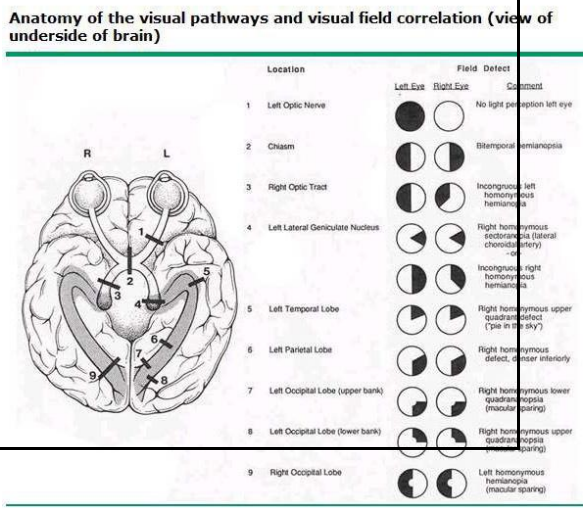
3. **Papilledema**
    - Symptoms:
        - Transient **visual obscurations** (**graying out**) by activities ↑ ICP (coughing, sneezing, bending down, straining)

          OR
        - Mild persistent blurred vision
    - Signs:
        - **Bilateral optic disc swelling** (due to ↑ ICP)
        - **RAPD is absent**

5. Visual Pathway Lesions

6. Nonorganic (NOVL) or Functional (FVL)





Retinal Detachment

Definition

Retinal detachment refers to the separation of the neurosensory retina from the underlying retinal pigment epithelium (RPE). This disrupts photoreceptor function and, if untreated, leads to permanent visual loss.

Classification

1. **Rhegmatogenous Retinal Detachment (RRD)**
    - Most common type
    - Caused by a retinal break allowing liquefied vitreous to enter the subretinal space
    - Associated with posterior vitreous detachment (PVD)
2. **Tractional Retinal Detachment (TRD)**
    - No retinal break
    - Caused by fibrovascular membranes exerting traction
    - Common in:
        - Proliferative diabetic retinopathy (PDR)
        - Retinopathy of prematurity (ROP)
3. **Exudative (Serous) Retinal Detachment**
    - No break or traction
    - Due to fluid accumulation from:
        - Inflammation (e.g., uveitis)
        - Tumors (e.g., choroidal melanoma)
        - Vascular disorders

Epidemiology

- Incidence: ~10–20 per 100,000/year
- Higher risk in:
    - Males
    - Myopes
    - Elderly population

Risk Factors

- High myopia
- Trauma
- Cataract surgery (especially complicated cases)
- Lattice degeneration
- Family history
- Posterior vitreous detachment
- Diabetic retinopathy (for TRD)

Pathophysiology

A. Rhegmatogenous RD

- Vitreous liquefaction (syneresis) → Posterior vitreous detachment → Retinal tear formation
- Retinal Tear → Fluid enters subretinal space → Retinal Detachment

B. Tractional RD

- Fibrovascular proliferation contracts → mechanical elevation of retina

C. Exudative RD

- Breakdown of blood-retinal barrier → fluid accumulation

Clinical Presentation

Symptoms

- Flashes (**photopsia**)
- Floaters / Black dots
- Persistent monocular visual field defect ("curtain" or "shadow")
- Sudden painless vision loss (if macula involved)

Signs

- Reduced visual acuity
- ± Relative afferent pupillary defect (RAPD)
- Elevated, mobile retina (RRD)
- Taut, concave retina (TRD)

Examination

Fundoscopy

- **RRD** → Corrugated, undulating retina; retinal breaks may be visible
- **TRD** → Concave, immobile detachment with traction bands
- **Exudative RD** → Smooth, dome-shaped elevation

Additional Tests

- OCT
- B-scan ultrasonography (if media opacity present)
- Visual field testing

Macula Status

- **Macula-on RD** → Surgical **Emergency** (better prognosis)
- **Macula-off RD** → Surgical **Urgency** (but visual recovery limited)

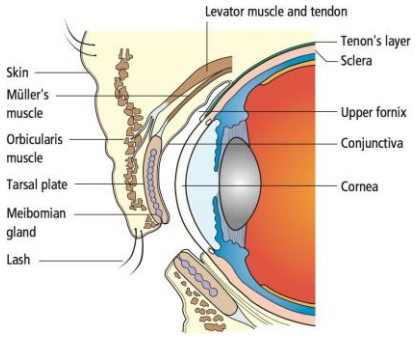
Eyelids and Lacrimal System

Anatomy of the Eyelid

Layers of the Eyelid

The eyelid is composed of several anatomical layers:

1. Skin (thin and elastic)
2. Orbital septum (fibrous barrier)
3. Orbital fat (cushioning)
4. Muscles:
    a. **Orbicularis oculi** (closure)
    b. **Levator palpebrae superioris** (main elevator)
    c. **Müller's muscle** (sympathetic accessory elevator)
5. Tarsal plates (structural support) & **Meibomian glands** (lipid secretion, **prevent evaporation**)
6. Conjunctiva (inner lining)
7. Eyelashes (protection)



Eyelid Muscles and Innervation

- **Levator palpebrae superioris** → CN III (oculomotor)
- **Müller's muscle** → sympathetic supply
- **Orbicularis oculi** → CN VII (facial nerve)

Functions of Eyelids

- Protection from trauma and foreign bodies
- Tear production and drainage
- Maintenance of tear film stability

Eyelid Pathologies

1. Abnormal Lid Positions

A. Ptosis

Definition: Drooping of the upper eyelid

Normal: Upper lid covers 1–2 mm of cornea

Causes:

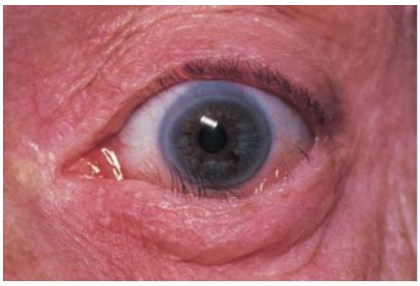
- Mechanical (tumors, edema, scarring)
- **Aponeurotic** (aging; most common acquired)
    - Caused by disinsertion or stretching of the levator aponeurosis
- Neurological:
    - **CN III palsy**
    - **Horner's syndrome**
- Myogenic:
    - Congenital → MC is **malformation of the levator palpebrae superioris muscle**
    - **Myasthenia gravis**
    - Muscular dystrophy
    - Botulism

Signs:

- **Reduced palpebral fissure**
- **Abnormal or absent lid crease**
- Associated neurological or myogenic signs

Management:

- **Treat underlying cause**
- Surgery if persistent
- Important in children
    - ↑ risk of amblyopia (كسل العين)



B. Entropion

Definition: Inward turning of eyelid (usually lower lid)

Features:

- Lashes rub against cornea
- Redness, irritation, tearing

Types:

- Involutional (aging)
  - Due to age-related weakening of the lid retractors, causing the orbicularis muscle to override the tarsal plate.
- Spastic (tightly closing the eye)
- Cicatricial (scarring)

Management:

- **Taping** of the lower lid & **Lubrication**
- Botulinum toxin
- Surgery

C. Ectropion

Definition: Outward turning of eyelid

Causes:

- Aging (lid laxity)
- **Facial nerve palsy**
- Skin scarring

Symptoms:

- **Dryness**
- **Excess tearing** (poor drainage)

Management

- Surgery

---

2. Blepharitis (Eyelid Inflammation)

General Features

- Chronic inflammation of lid margins
- Symptoms: itching, redness, soreness (**worse in morning**)
- Associated with **dermatological conditions** (Atopic eczema, Seborrheic dermatitis, Rosacea)
- Requires a long-term treatment

A. Anterior Blepharitis

Lower lid with inflammation with characteristic scales on the eyelashes.

- Affects eyelash base
- Caused by **Staphylococcus**

Signs:

- Scaling
- Redness
- **Collarettes**
- **Lash loss**

Complication

- Blepharokeratitis
- Marginal keratitis

Management:

- **Lid hygiene** (baby shampoo)
- Topical **antibiotics** (systemic in case of long-standing infection)
- Topical **Steroids**

B. Posterior Blepharitis

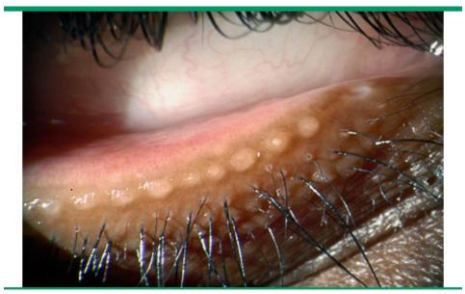
- Due to **Meibomian gland dysfunction (MGD)**

Features:

- Thick, cloudy secretions
- Lid margin inflammation

Management:

- **Warm compresses**
- Oral **antibiotics** (doxycycline, azithromycin)
- **Artificial tears**



Lower eyelid with characteristic posterior lid inflammation and oily white plugs visible at the meibomian gland openings.

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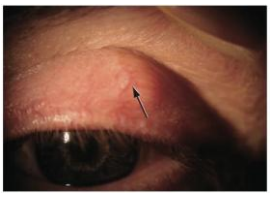
3. Benign Eyelid Lesions

1. Chalazion

- Meibomian gland obstruction → lipogranuloma
- **Painless**
- Resolves spontaneously (within 6 months)

Treatment: Incision and curettage if persistent



2. Hordeolum (Stye)

- **Painful** abscess

Types:

1. Internal (Meibomian gland)
2. External (eyelash follicle)

Management:

- Warm compress
- Antibiotics
- Drainage

3. Molluscum Contagiosum

- Viral (poxvirus) lesion
- Causes follicular conjunctivitis
- Most common in **childhood** & early adolescence

Transmission:

- Direct skin contact & **Fomites**
- Autoinoculation

Risk Factors:

- **Immunosuppression**
- Active atopic dermatitis
- Hot & humid climates
- Crowded living conditions with poor hygiene

Treatment: Excision

4. Cysts

1. Sebaceous cyst
2. **Cyst of Moll** (sweat gland, **translucent**)
3. **Cyst of Zeis** (sebaceous gland, **opaque**, associated with eyelashes)

5. Squamous Cell Papilloma

- Benign "skin tag"
- Caused by HPV

Treatment: Excision (cosmetic)

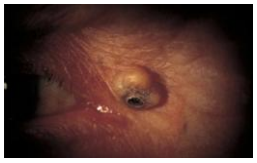
6. **Xanthelasma**

- Yellow plaques
- Associated with hypercholesterolemia

Management:

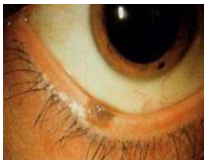
- Lipid profile check
- Cosmetic excision

7. **Keratoacanthoma**



- Rapidly growing lesion with keratin-filled central crater
- May mimic malignancy (**grow over 3-6 weeks** in contrast to malignancy)

Complication: Ugly scar if left untreated



Management: Excision and histology

8. **Nevus**

- No treatment is necessary

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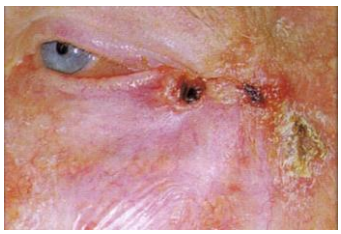
4. **Malignant Eyelid Tumors**

1. **Basal Cell Carcinoma (BCC)**
   - **Most common eyelid malignancy (≈90%)**
   - Accounts for ~10% of all BCC in the body
   - **Characteristics**:
     - Slow-growing
     - **Locally invasive**
     - Rarely metastasizes
     - **Painless** lesion
     - Can be nodular, ulcerative "rodent ulcer", or scaly

   - **Risk Factors**:
     - Fair skin
     - Blue eyes
     - Chronic sun exposure
   - **Management**:
     - Excision biopsy with safety margins
     - **Mohs micrographic surgery** (preferred)
     - Alternatives: cryotherapy, radiotherapy
   - **Prognosis**: Good
2. **Squamous Cell Carcinoma (SCC)**
   - **Less common (~5%) but more aggressive**
   - **Characteristics:**
     - Faster growth
     - Metastasizes to regional lymph nodes
     - Appears as hard nodule or scaly patch



   - **Origin:**
     - May arise de novo
     - Or from actinic keratosis
   - **Management:**
     - Excisional biopsy with safety margins

5. Eyelash Abnormalities

1. Trichiasis

- **Definition:** Misdirected eyelashes toward the globe (normal lid position) → VS. Entropion
- **Symptoms:**
  - Corneal irritation
  - Tearing
  - Abrasion
- **Association:**
  - **Trachoma** (**Chlamydia trachomatis**) in developing world
- **Management:**
  - **Epilation** (temporary)
  - Electrolysis / cryotherapy (recurrent)
  - Surgical correction

Lacrimal System

Tear Production

- Lacrimal glands produce **~1.5 µL/min**
- Tears:
  - Partly evaporate
  - Remainder drains into the nose
- Tear film is renewed with each blink

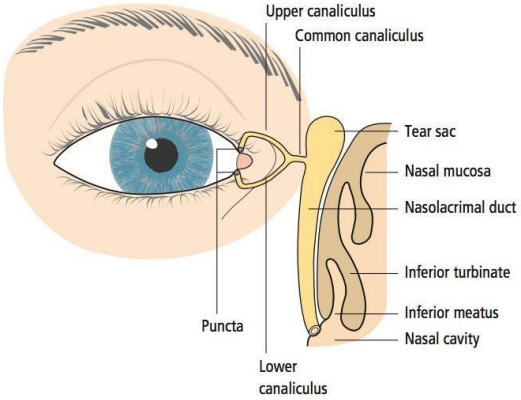
Tear Drainage Pathway

Upper & Lower Puncta → Upper & Lower Canaliculi → Common Canaliculus → Lacrimal Sac → Nasolacrimal Duct → Nasal Cavity

Tear Film

Structure (3 layers)

1. **Mucin layer (inner)**
   - Produced by goblet cells
   - Adheres tears to ocular surface
2. **Aqueous layer (middle)**
   - From lacrimal gland
   - Provides nutrients & oxygen
3. **Lipid layer (outer)**
   - From Meibomian glands
   - Prevents evaporation



Functions

- Lubrication and hydration
- Smooth refractive surface
- Oxygen supply to cornea
- Debris removal
- Antibacterial action (IgA, lysozyme, lactoferrin)

6. Lacrimal System Disorders

1. Dry Eye (Keratoconjunctivitis Sicca)

Pathophysiology

- **↓ tear production OR ↑ evaporation**
- Leads to:
  - Ocular surface hyperosmolarity & damage → Inflammation → discomfort & vision loss

A. Aqueous-Deficient Dry Eye

Causes

- Lacrimal gland dysfunction
- **Most common:** Sjögren's syndrome

Types

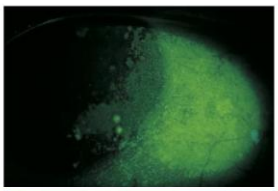
- **Primary Sjögren's**
  - Autoimmune destruction of glands
  - Associated with **dry mouth**
- **Secondary Sjögren's**
  - Associated with RA, SLE, scleroderma

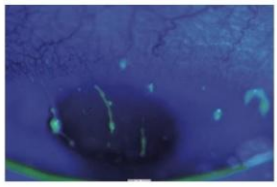
Symptoms (worse in evening)

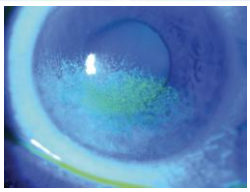
- Burning
- Grittiness (sand sensation)
- Photophobia
- Fatigue
- ↓ vision (corneal involvement)

Diagnosis

- ↓ Salivary & lacrimal functions
- **Fluorescein stain**
  - **Early tear film breakup (<5 sec)**
  - **Punctate Epithelial Erosions** in interpalpebral zone
  - **Severe** → confluent staining, possible filamentary erosions
- **Biopsy** → T-cell infiltrates in minor salivary gland
- **Anti-Rho** & **Anti-La** titers







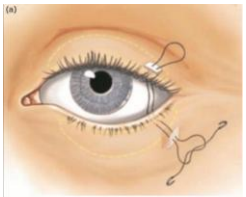
Management

- **Artificial tears**
- **Protective eyewear**
- **Puncta plugs**
- **Topical anti-inflammatory** (cyclosporine, steroids)

B. Evaporative Dry Eye

Causes

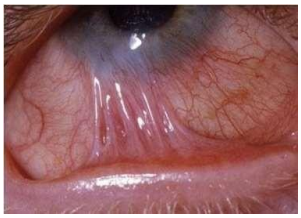
- **Meibomian gland dysfunction (MGD)**
- Lid abnormalities:
  - **Ectropion**
  - **Lagophthalmos** (incomplete lid closure, 7th nerve palsy)
  - **Proptosis**
- Reduced blinking (e.g., **Parkinson's**)



Management

- Treat underlying cause
- Artificial tears
- Warm compresses
- Botulinum toxin (to induce ptosis)
- **Lateral tarsorrhaphy**

C. Cicatricial Conjunctival Disorders



Features

- Loss of goblet cells
- Conjunctival scarring
- **Symblepharon formation**

Loss of goblet cells seen in

- Stevens–Johnson syndrome
- Chemical burns
- Trachoma
- Vitamin A deficiency

Treatment

- **Artificial tears**

2. Tear Drainage Disorders (Epiphora = Excessive tearing)

A. Congenital Nasolacrimal Duct Obstruction

Cause

- Failure of canalization at distal duct

Clinical Features

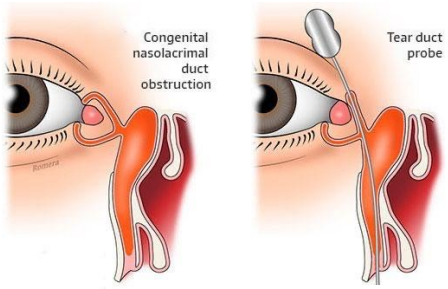
- Watering eye
- **No conjunctival inflammation**
- Sticky discharge on pressure over lacrimal sac

Complications

- **Dacryocystitis & Mucocele**

Management

- Usually **resolves spontaneously** (first year)
- If persistent → **probing**



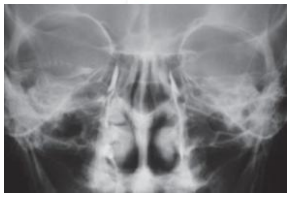
B. Adult Nasolacrimal Duct Obstruction

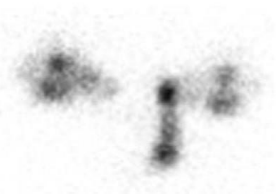
Causes

- Infection
- Trauma \ Fracture
- Topically applied drugs

Symptoms

- Tearing
- Sticky discharge
- **Worse in cold/wind weather**





Diagnosis

A. Slit lamp → stenosed punctum

B. Patency of nasolacrimal duct assessment

- Syringing test
- Fluorescein dye disappearance test (<5min)
- Dacryocystography (X-ray)
- Dacryoscintigraphy (Nuclear image)

Management

- Treat underlying cause
- **Dacryocystorhinostomy (DCR)** (definitive surgery)

C. Lacrimal System Infections

7.1 Dacryocystitis

- Infection of lacrimal sac (usually due to obstruction)

Features

- **Painful** swelling at medial canthus
- Redness
- Tenderness

Organisms

- Staphylococcus
- Streptococcus

Treatment

- Systemic **antibiotics**
- **DCR** after resolution

7.2 Mucocele

- Mucus accumulation in blocked sac
- **Painless** swelling

Treatment

- **DCR**

The Orbit

Introduction

The orbit is a compact, complex anatomical structure that houses and protects the eye and its associated components. Despite its small volume (**~30 cc**), it contains critical structures including the globe, optic nerve, extraocular muscles, blood vessels, and lacrimal apparatus.

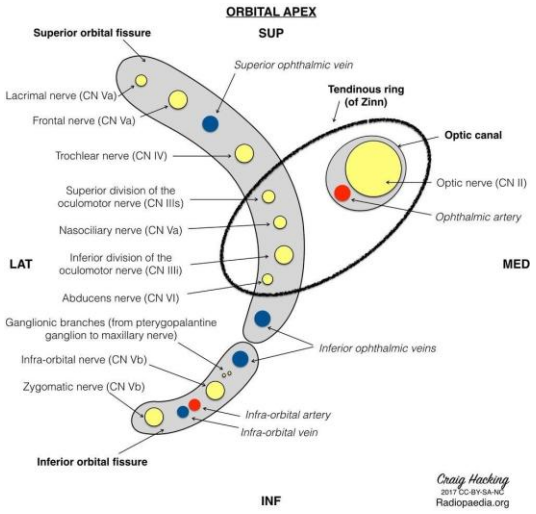
Because of its confined space and proximity to the brain and paranasal sinuses, even minor pathological changes can produce significant clinical manifestations.

Anatomy of the Orbit

Structure

The orbit is:

- A **bony socket** shaped like a **four-sided pyramid**
- Designed to **protect the eye** and allow movement



Orbital Bones

The orbit is formed by **seven bones**:

- Frontal
- Ethmoid
- Lacrimal
- Sphenoid
- Maxillary
- Palatine
- Zygomatic

Openings and Contents

Key openings include:

Optic canal

- Located at the orbital apex
- Transmits:
  - Optic nerve
  - Ophthalmic artery
  - Central retinal vein

Superior orbital fissure

- Transmits:
  - Cranial nerves III, IV, VI
  - Branches of trigeminal nerve (V1) (Lacrimal, Frontal, Nasociliary)
  - Superior ophthalmic vein
  - Orbital branch of MMA
  - Recurrent branch of lacrimal artery
  - Superior orbital vein

Inferior orbital fissure

- Transmits:
  - Infraorbital and zygomatic nerves
    - Infraorbital vessels
    - Inferior ophthalmic vein
    - Parasympathetic fibers

Lacrimal System

- Lacrimal gland: superolateral orbit
- Lacrimal sac: medial wall

Functions

- Protection of the globe
- Support and movement via extraocular muscles
- Conduit for nerves and blood vessels

Proptosis (Exophthalmos)

Definition

Proptosis is the forward displacement of the globe due to a space-occupying lesion.

It may be:

- **Bilateral** → Graves' Disease
- **Unilateral** → Tumors, Infections

Causes of Proptosis

1. **Infectious**
   - **Orbital cellulitis** (most common cause of unilateral proptosis in children)
   - **Mucormycosis**
   - Rapid, painful onset
2. **Inflammatory**
   - **Thyroid eye disease**
   - **Orbital inflammatory syndrome** (Diagnosis of Exclusion)
3. **Neoplastic**
   - Lacrimal gland tumors
   - Lymphoma, leukemia
   - Meningioma (intra-conal → axial proptosis)
   - Optic nerve glioma
   - Metastases (breast, lung, prostate, GI, kidney)
   - Ossifying fibroma
   - Rhabdomyosarcoma (children, rapidly developing proptosis)
4. **Vasculitis**
   - Wegener granulomatosis (GPA)
   - Churg–Strauss syndrome (EGPA)
5. **Vascular**
   - Orbital varix (orbital venous malformation)
     - Transient proptosis, induced with ↑ cephalic venous pressure
   - AV malformations
   - Carotid–cavernous fistula
6. **Trauma**
   - Orbital hemorrhage
   - Orbital/facial fractures
7. **Pseudoproptosis**
   - Buphthalmos
   - Contralateral enophthalmos
   - Contralateral blepharoptosis
   - Lid retraction
   - High myopia

Measurement

Using **Hertel Exophthalmometer**:

Measurement Category	Hertel Exophthalmometer Reading
Normal	10 – 21 mm
Mild Proptosis	21 – 23 mm
Moderate Proptosis	24 – 27 mm
Severe Proptosis	≥ 28 mm
Significant Asymmetry	**≥ 2 mm difference between eyes**

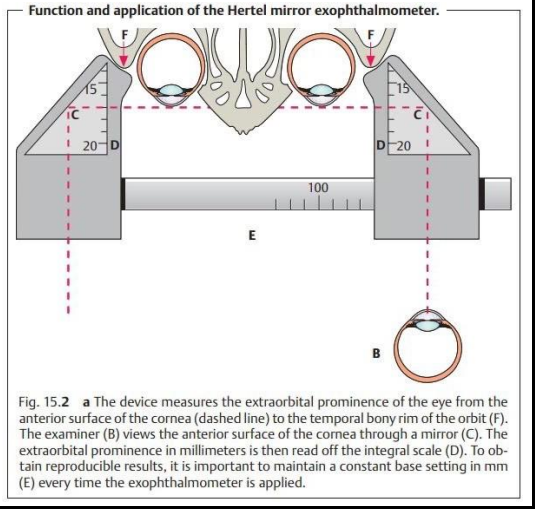


Fig. 15.2 **a** The device measures the extraorbital prominence of the eye from the anterior surface of the cornea (dashed line) to the temporal bony rim of the orbit (F). The examiner (B) views the anterior surface of the cornea through a mirror (C). The extraorbital prominence in millimeters is then read off the integral scale (D). To obtain reproducible results, it is important to maintain a constant base setting in mm (E) every time the exophthalmometer is applied.

Enophthalmos

Backward displacement of the globe seen in:

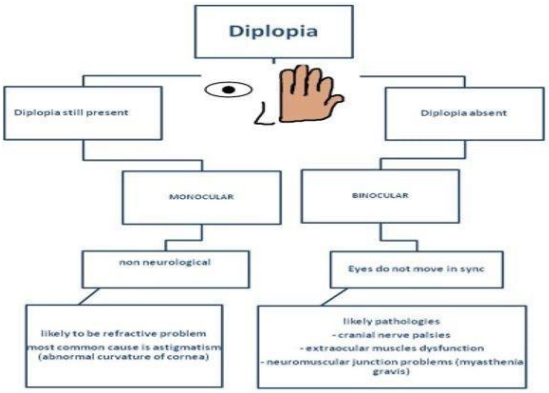
- Orbital fractures
- Horner syndrome
- Orbital fat atrophy
- Congenital conditions

Diplopia (Double Vision)

Definition

Diplopia is the perception of **two images** of a single object, which may be:

- Horizontal
- Vertical
- Oblique



Causes

1. Extraocular muscle disease
2. Cranial nerve palsies
3. NMJ disorders (e.g., myasthenia gravis)

Types of Diplopia → Cover-Uncover Test

1. Monocular Diplopia

- Present when one eye is open
- Usually due to refractive errors (e.g., astigmatism)

2. Binocular Diplopia

- Present only when both eyes are open
- Due to misalignment of eyes (muscle or nerve problem)

Clinical Evaluation

Important History Points

1. Pain

- May indicate serious causes (e.g., **PComA aneurysm** - emergency)

2. Onset

- Acute → vascular
- Gradual → inflammatory or compressive

3. Image alignment

- Horizontal → **CN VI** or lateral/medial rectus muscles disease
- Vertical → **CN IV** or TED or orbital floor fractures

4. Timing

- Worse at end of day → myasthenia gravis

5. Gaze direction

- Helps localize affected muscle

6. Trauma

- Important contributing factor

7. Past history & systemic disease

- Diabetes, thyroid disease, neurological disorders

Thyroid Eye Disease (TED)

Overview

- **Most common cause of unilateral & bilateral proptosis in adult**
- **Most common cause of bilateral proptosis in children**

Pathophysiology

- **Autoimmune** activation of T-cells
- Target: **TSH receptors** in orbital fibroblasts
- Increased GAG → fluid accumulation
- **Muscle swelling** → increased orbital pressure → proptosis

Most Affected Muscle

- **Inferior rectus muscle**

Clinical Features

Symptoms

- Red, painful eye
- Periorbital edema
- **Diplopia** (double vision)
- Proptosis
- Reduced vision
- Lid retraction
- Inflammatory conjunctivitis

Signs on Examination

- Asymmetrical proptosis
- Chemosis (conjunctival edema)
- Lid retraction → "staring appearance"
- Lid lag (delayed eyelid movement)
- Restricted eye movements

Complications

- Exposure keratopathy
- Compressive optic neuropathy → blindness

Management

- Lubrication
- Prisms for diplopia
- **Steroids** (acute cases)
- Orbital decompression surgery

Severity Assessment

Severity is graded using the **"NO SPECS" classification**: **0 → 6**

- N – No signs
- O – Only signs (lid retraction)
- S – Soft tissue involvement
- P – Proptosis
- E – Extraocular muscle involvement
- C – Corneal involvement
- S – Sight loss

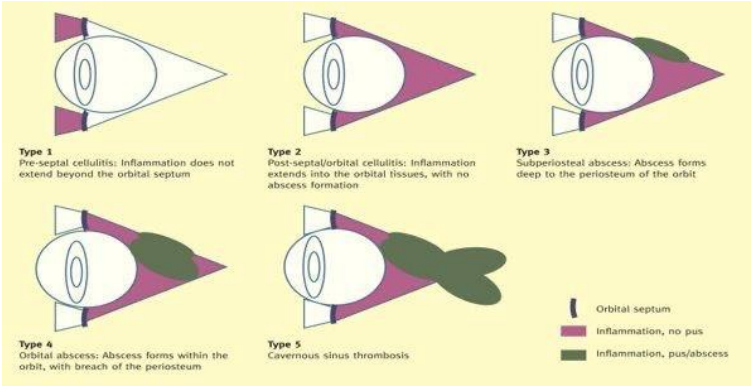
Differential Diagnosis of Orbital Diseases

A. **Trauma**
B. **Infection**
C. **Vascular**
D. **Tumors**

A. Orbital Infection

Classification (Chandler's Classification)

1. Preseptal cellulitis
2. Orbital cellulitis
3. Subperiosteal abscess
4. Orbital abscess
5. Cavernous sinus thrombosis



1. Preseptal Cellulitis

Definition

Infection of tissues anterior to the orbital septum (eyelids and superficial structures).

Etiology

- Trauma
- Skin infections

Clinical Features

- Swollen, tender, red eyelids
- Mild fever
- **Normal vision and eye movements**
- **No proptosis**

Key Point

- Can progress to orbital cellulitis if untreated

Treatment

- Topical **antibiotics**
- Severe cases / child <1 year of age → manage as orbital cellulitis

2. Orbital Cellulitis

Definition

A **medical emergency** involving infection posterior to the orbital septum affecting orbital fat and muscles.

Common in children, elderly & immunocompromised

Etiology

- Spread from sinus (especially **ethmoid**) infections
- Dental infections
- Trauma

Common organisms → Staphylococcus & Streptococcus

Clinical Features

- Red eye
- Periorbital swelling and inflammation
- Pain (especially with eye movement)
- Fever and headache
- Chemosis (conjunctival edema)
- **Proptosis**
- **Restricted eye movements (ophthalmoplegia)**
- **Reduced vision ± RAPD**

Diagnosis

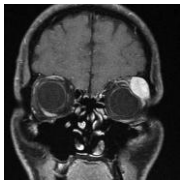
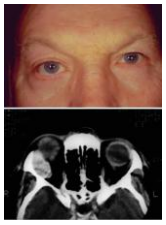
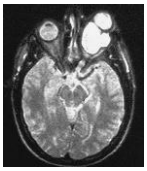
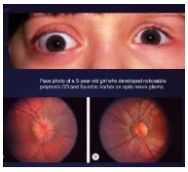
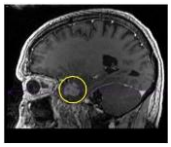
- CT / MRI

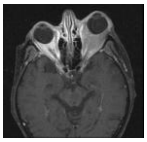
Treatment

1. Hospital admission
2. **IV antibiotics** (ceftriaxone + vancomycin)
3. Surgical drainage if abscess forms
4. Optic nerve decompression if needed (Endoscopic OND)

Complications

- Optic nerve damage
- **Cavernous sinus thrombosis**
- Meningitis / brain abscess
- Vision loss
- Death (severe cases)




B. Orbital Tumors

1. Lacrimal Gland Tumors

- May be benign or malignant
- Malignant tumors → poor prognosis
- Benign tumors require **removal** to prevent malignant transformation

2. Optic Nerve Glioma

- Often associated with **neurofibromatosis (NF)**
- Usually slow-growing
- May **not require treatment** in some cases

3. Optic Nerve Sheath Meningioma

- Rare and difficult to remove
- May be managed with **radiotherapy**
- Can spread from cranial cavity via optic canal

4. Orbital Lymphoma

- **Requires systemic evaluation**
- Treatment:
  - **Chemotherapy** (systemic disease)
  - **Radiotherapy** (localized disease)

5. Rhabdomyosarcoma

- **Most common malignant orbital tumor in children (6–9 years)**
- **Most common benign orbital tumor in children** → **Capillary Hemangioma**
- Rapidly growing tumor of skeletal muscle origin
- Responds to **chemotherapy** if localized

6. Metastatic Tumors

In Children

- **Neuroblastoma** (most common in children)
- Ewing sarcoma
- Wilms tumor
- Leukemia

In Adults

- **Breast** (most common in women), **Lung** (most common in men), Prostate, GI cancers

Characteristics

- **Spread via bloodstream** (orbit has no lymphatics)
- May cause **proptosis or enophthalmos**
- Neuroblastoma may cause **periocular hemorrhage**

Prognosis

- Variable
- Adults with metastasis → poor life expectancy
- Neuroblastoma in children <11 months → relatively good prognosis
- Some respond to radiotherapy or chemotherapy

7. Dermoid Cysts

- Congenital lesions from **ectodermal** tissue
- Common in superior orbit (medial/lateral)
- Usually removed for:
  - Cosmetic reasons
  - Prevent rupture and scarring
- CT scan may be needed before surgery (may have deeper connection)