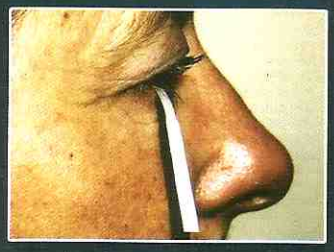
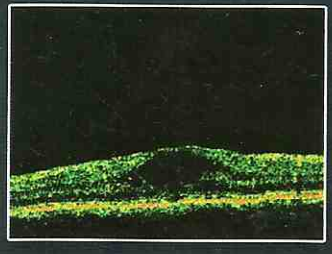
Abd El- Haleem

Summary Of OPHTHALMOLOGY





by

Dr. M. Abd El-Haleem

PART II

INDEX
Part II

شكر خاص للأستاذ الدكتور/ عمرو الشبراوي جامعة الزقازيق
شكر خاص للأستاذ الدكتور/ أحمد الصواف FRCS

GLAUCOMA

written

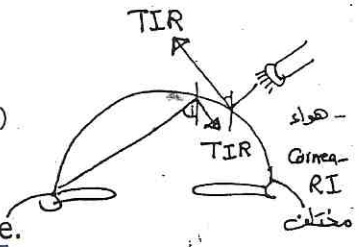
THE ANGLE OF THE ANTERIOR CHAMBER

● Definition:

- It is a recess which lies between the periphery of the cornea & root of the iris.

Give reason bec.

- The angle of the anterior chamber cannot be examined **Except by Gonioscopy + slit lamp as:** See atlas page (133)



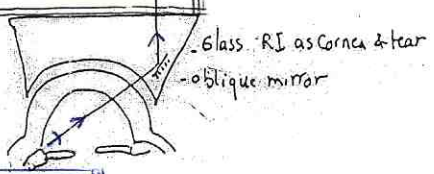
- The rays coming from the angle, undergo **total internal reflection** at the cornea-air interface.

- To overcome this → a lens is placed on the cornea

(Goniolens = CL made of glass has the same RI as cornea & tear film & has an oblique mirror.) to change cornea-air interface into cornea-glass interface.

So → No total internal reflection.

e.g Goldmann 3-mirror gonioscopy



NB. Uses of e.g Goldmann 3-mirror gonioscopy مهم :

1- Examination of the angle
2- Examination of the fundus.
3- Laser ALT
4- Laser to the funsus.

NB. Types of Goniolenses: مهم

1) *Indirect* :

Allowing viewing of the angle by mirror, used in conjunction with slit lamp, e.g.:

- Gold man one , two or three mirrors : need local anesthesia & methyl cellulose .

- Ziess four mirror: need local anesthesia only

Indirect mirror slit lamp Goldman & Ziess

direct Koeppe → surgery

2) *Direct*: See atlas page (133)

Allowing viewing of the angle direct without mirrors,
don't require slit lamp

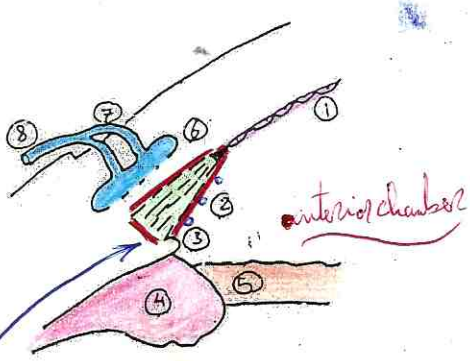
- **Koeppe :** used for surgery

onioscopic appearance: See atlas page (133,134)

) Schwalb's line: termination of Descemet's membrane (thickened).

2) Trabecular meshwork:

- It is composed of parallel laminae.
- Sponge like with holes (spaces of Fontana),
  These holes decrease in size as we go towards the Schlemm's canal. مهم



- In cross section, it appears triangular with:
  1- Apex: attached to Schwalb's line.
  2- Base: attached to scleral spur.
  3- Outer surface: in contact with the inner wall of Schlemm's canal.
  4- Inner surface: in contact with the anterior chamber.

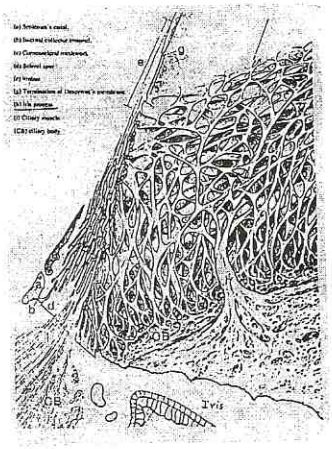
The Anterior part of TM is non pigmented , non functioning
& the posterior part if pigmented & functioning. مهم

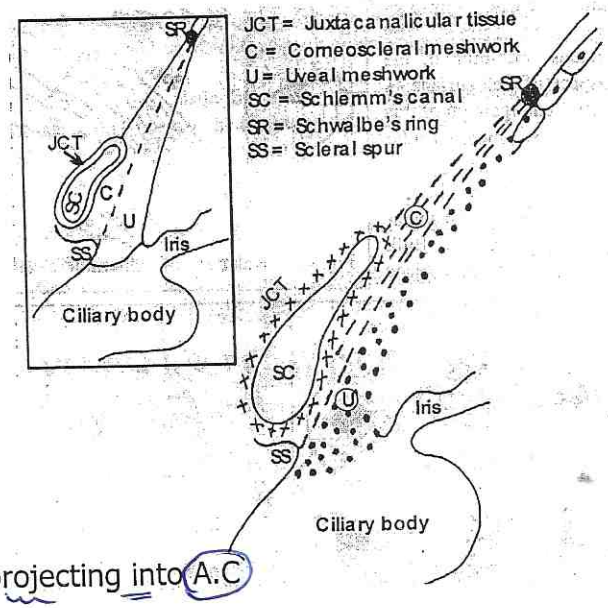
NB. TM can be differentiated into:

1) **Anterior part:** Non pigmented, non functioning .
2) **Posterior part:** : pigmented, functioning .

NB. As we go towards the canal of Schlemm's as the spaced of TM becomes smaller.

Glaucoma





(3) Scleral spur:

- A small ridge of sclera, projecting into A.C
- Gives origin to the longitudinal ciliary ms.→ its contraction opens the spaces of Fontana.

(4) Ciliary band: Small part of C.B base , its width depend on the insertion of the position of iris insertion.

(5) Root of iris.

(6) Iris processes: Small extensions from the iris attached to T.M. across the angle recess.

(7) Blood vessels :

Q: How you can differentiae between normal & abnormal BVS at the angle?

The following structures are not seen on gonioscopy:

(1) ***Schlemm's canal:*** Circular canal inside the limbus (Endothelial lines) , which is connected to trabecular meshwork by internal collector channels, It appears oval in cut section.

> **NB.** Canal of Schlemm is invisible when it is empty of blood, but when it contains blood it forms a well marked line seen through the T.M.

(2) ***Aqueous veins:*** Drain the aqueous from schelmm's canal to the episcleral veins→ Ant. Ciliary veins → Venous circulation.

- **SAFFER GRADING of the angle :** See atlas page (122)
  - GRADE 0: iridocorneal contact
  - GRADE 1: Schwalbs line is see
  - GRADE 2: TM is seen
  - GRADE 3: SS is see
  - GRADE 4: CB is see
  - Open angle Grade 4,3
  - Closed angle Grade 1,0

PHYSIOLOGY OF THE AQUEOUS

- **Aqueous humour:** It is a transparent intra-ocular fluid that fills the anterior & posterior chamber, with very low ptn content than the plasma.
- **Volume:** 1.25 ml
- **Function:**
  - Maintainence of the normal IOP.
  - Nutrition to avascular structure (cornea & lens).
  - One of the refractive media (RI = 1.33).
- **Formation:** By non-pigmented epith. of the ciliary body processes (pars plicata) 2μL/ min through:

(1) Active secretion: By carbonic anhydrase enzyme & Na/K ATPase pump. (60% of aqueous formation).

$$H_2O + CO_2 \xrightarrow{CA} H_2CO_3$$ ((مهمه المعادله دي))

SO CAI
- ↓ aqoues formation
- ↑ CO_2 وده يفيدني في ايه ؟؟

(2) Ultra filtration: Due to difference in the hydrostatic pressure, Pressure inside the BVs > outside.

(3) Passive diffusion (through BVs walls): Osmotic pressure in chambers & CB stroma > BVs.

2 & 3 → passive mechanisms (40% of aqueous formation)

Glaucoma

- **Aqueous circulation :** See atlas page (123)

  From ciliary body (non pigmented epithelial) → post. Chamber → Pupil → anterior chamber
  → Angle of the ant. Chamber → space of Fontana → schlemm's canal
  → Aqueous veins → Episcleral veins → Ant. Ciliary veins
  → venous circulation

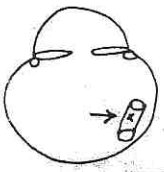
NB. How can aqueous pass through the endothelium of the angle ?

- **Aqueous drainage:**

(1) Trabecular route (85-90%): Through the angle .

(2) Uveo-scleral out flow (10-15%): Through the iris crypts & face of C.B. → between ciliray ms → suprachoroidal space → choroid → venous circularion

INTRAOCULAR PRESSURE (IOP)



- **Normal IOP:** It is the pressure under which the eye functions normally (with opened BVs).
- **Average IOP:** - Ranges between 10-21 mm Hg (± 2-3 diurnal variation)
  - With normal difference between both eyes < 4 mm Hg.

Diurnal variation:

It is highest in the morning & minimum in the evening (± 2-3 mmHg) d.t:

(1) Ocular massage during the day → ↑ drainage (ف الضغط يقل علي بالليل)

(2) Venous stagnation during sleeping → ↓ aq. Outflow (ف الضغط يزيد علي الصبح)

(3) Circadian rhythm of steroids: steroids ↑ at the morning & ↓ at night.
(Steroids have secretory action , ↑ aqueous formation)

Measurement of IOP:

1. Digital palpatation
2. Tonometry

Angle closure glaucoma

(1) Digital palpation: See atlas page (132)

- Rough method , used only in case of ACG >> stony hard IOP.
- While the pt. is looking down without closing his eye (to avoid bell's phenomenon) , pressing on the sclera by index finger & recieve the impulse by the other index finger(like indentation of a cyst)
- Expressed as : Tn T+ T-

(2) Tonometry: Measuring IOP by tonometers:

1) Indentation (Schiotz) tonometer: See atlas page (131)

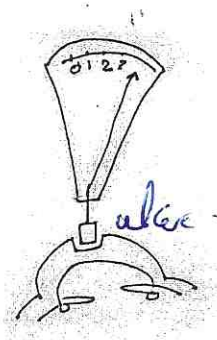
- While the patient lying on his back + surface anaesthesia.
- The tonometer is placed perpendicular to the cornea, which measures the degree of corneal indentation produced by a known weight on the cornea by the movement of a lever on a scale
- The reading of indentation is converted into mm Hg on a special table.
- **Advantages:** simple, used for follow up & used in bed ridden pt.
- **Disadvantages:**

1- Affected by the scleral rigidity(thickness) , so not accurate in myopes or hypermetropes.

2 - Done while the pt. is supine → venous return → ↑ IOP → cardiac problems .

3 - It is a contact tonometer:

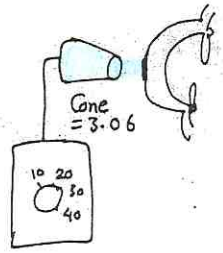
- C. ulcer
- Carries infections → HIV, hepatitis B&C, Adenovirus so sterilization with alcohol or hydrogen peroxide is very important.



2) Applantation tonometer (Goldman tonometry):

See atlas page (131,132)

- While the patient sitting on slit lamp using cobalt blue filter + surface anaesthesia + flourescine stain,
- A cone (3.06 mm) with a double prisms inside is used to applanates equal area of the cornea.
- When it touches the cornea 2 yellow semi-circles will be seen.
- The dial of the tonometer is rotated till the inner edges of the semi-circles will just touch, now the cornea has been flattened.

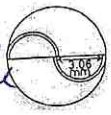


Glaucoma

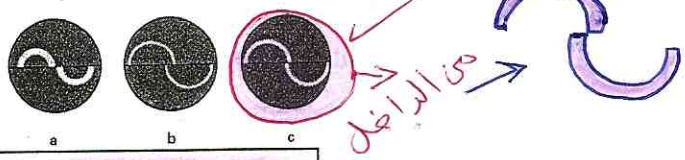
- The reading on the dial is multiplied by ten, equal the IOP
- It measures the IOP by recording the force needed to flatten a certain area of the cornea (3.06 mm).

NB. Why 3.06 mm ?? لو شاطر

scleral rigidity & tear film S.T. will cancel each other at 3.06mm



Correct applanation area



C is the correct position

- **Advantages:**
  1- More accurate (less affected by scleral rigidity).
  2- Done while the pt is sitting.
- **Disadvantages:** It is a contact tonometer (C. ulcer, carries infection....).

3) Perkins (hand held) tonometer: See atlas page (132)

- used in bed bound pt.

4) Air puff tonometer: See atlas page (132)

It flattens the cornea by jet of air, the time required to flatten the cornea relates directly to the level of IOP.

- **Advantages:** Non contact so no risk of infection transmission or corneal ulcer, no surface anaesthesia + patient is sitting.
- **Disadvantages:** noise startle the pt.

5) Tonopen. See atlas page (132)

- Can measure IOP in cases of distorted or edematous cornea.

GLAUCOMA

Definition: It is pathological elevation of IOP that leads to:

Optic nerve damage & visual field defect.

	↑ IOP	Cupping	Field changes	Family history	Risk factors
POAG (pathologic open angle glaucoma)	+ve	+ ve	+ ve	+ ve	+ ve
Ocular hypertension	+ ve	- ve	- ve	- ve	- ve
Normal (low) tension glaucoma	- ve	+ ve	+ ve	+ ve	+ ve
Glaucoma suspect	+ ve	+ ve	- ve	+ ve	+ ve

NB. Ocular hypertension = risk factor may become OAG

NB. In normal tension glaucoma the outer coat & BVs can't withstand the normal IOP. Or the cornea is thin.

NB. In ocular hypertension: the outer coat & BVs can withstand the high IOP. Or the cornea is thick.

Classification

- Congenital
- Acquired
  - Primary
    - Closed angle CAG
    - Open angle OAG
  - Secondary
    - Closed angle.
    - Open angle.

* angle closed بـ Iris = Closed angle glaucoma
* " " " else = open angle glaucoma

Glaucoma

Congenital Glaucoma (Buphthalmos) Ox-eye جاموسة

See atlas page (123,124)

◊ **Definition**: It is ↑ IOP leading to optic n. damage due to "congenital anomalies" closing the angle of the anterior chamber, Symptoms & signs appears between birth & 1st year.

◊ **Etiology:**

(1) 1ry Buphthalmos:

"Trabecular dysgenesis"

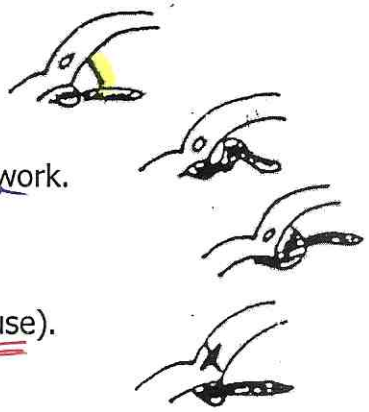
1- Abnormal mesodermal membrane close angle of A.C. "Barkan's memb".

2- Forward insertion of iris & C.B. to the trabecular meshwork.

3- Failure of complete separation of iris from the cornea.

4- Absence of schlemm's canal (a result rather than a cause).

((1,2,3,4 called trabecular dysgenesis)).



(2) 2ry Buphthalmos:

Due to associated anomalies or syndromes as:

1) Microphthalmos.

2) Ectopia lentis :

1) Marfan's syndrome: See atlas page (113)

1- Lens subluxation, glaucoma. Cataract,RD & blue sclera

2- High arched palate.

3- Cardiac anomalies (Die before 20 yrs)

4- Long digital bones.

2) Homocystinurea .

3) phakomatosis: (Tumours)

- Struge weber syndrome: See atlas page (124)

Ipsilat. hemangioma in skin along distribution of 5th n.(port wine stain) , meninges & choroid.

- Neurofiromatosis type I مهم

Glaucoma

4) **Retinoblastoma, ROP.**

5) **Birth trauma** → Neonatal iridocyclitis & hyphema. → Bleeding in A.C.

6) **Congenital Rubella/Lowe syndrome**(error in metabolism of amino a.) oral

Both lead to congenital cataract & congenital glaucoma شفوي

7) **Iridocorneal dysgenesis:**

- **Axenfeld anomaly**
- **Reiger anomaly**
- **Aniridia:** Absence of the iris with rudimentary stump that occludes the angle

this pt. should have abdominal US at regular interval. ليه؟؟

What Caus Cong. Cataract & glaucoma?

◊ Incidence: 3B

Before 6 month
Boys mostly
Bilateral

1) Age: 80% of cases are presented **b**efore the age of 6 months.
2) Sex: More in **b**oys 65%(m : f= 2:1)
3) Side: commonly **b**ilateral (80% of cases).

◊ Clinical picture:

- ***Symptoms:*** given by the mother.

1) **Early:** Lacrimation, photophobia , blepharospasm (due to irritation of the corneal nerves by the oedema & stretch).

2) **Late:** Large eye (bull's eye or ox eye عين الثور) due to elasticity of outer coat.

& hazy cornea > defective vision. See atlas page (124)

- ***Signs:*** The eye will distend with ↑ IOP as the outer coat is still elastic.

(1) Cornea: - Diameter: ↑12-16 mm (normal 10.5 mm).

Haab stria - Curvature: ↓(more flat).

- Transparency :↓ (Hazy & oedematous).

Glaucoma

- May also show: Haab's stria : See atlas page (124)
  transverse Linear opacities(healed breaks in Descement memb.) usually in the lower part of cornea due to tears of descement's memb. which is very thin in children).

- Widening of the area of the limbus into large bluish zone.

* **(2) Sclera:** blue (being thin showing the choroid).

(3) AC: - Deep as the lens becomes flat & displaced backwards.
& - large .

(4) Pupil: Large & sluggish reaction due to corneal oedema.

(5) Iris: Early: Tremulous as the lens is post. displaced.
Late: Atrophic patches.

(6) Lens: flattened & displaced backward .
(d.t stretching of zonules).

(7) IOP: High but less than real (underestimation) d.t.
1) Distinsability of the globe.
2) High scleral rigidity.

(8) Fundus: Glaucomatous cupping & atrophy of the optic disc occur
Late (d.t scleral distensibility).

NB. Fundus examination is very important To exclude retinoblastoma.

(9) Gonioscopy: Abnormalities of the angle.

(10) Refraction: Axial myopia d.t. enlargement of the globe , But ~~less~~
Less than expected from the axial length: d.t.
1- Flat cornea. 2- Lens flat & backward displaced.

◊ Diagnosis:

1) Measurement of corneal diameter. See atlas page (123)
2) Gonioscopy.
3) Measurement of IOP (under ketamine anesthesia)?? Not affect the IOP.

Glaucoma

◊ Differential diagnosis: from other causes of:

(1) Corneal enlargement in:

Megalocornea & Congenital myopia: No signs of buphthalmos.

(2) Corneal clouding: As

- keratitis from intrauterine infectiom (Rubella Keratitis).
- Traumatic corneal oedema (Birth trauma with Forceps of delivery).
- Metabolic disorders : mucopolysacharidosis.
- Sclero-cornea
- Corneal dystrophies e.g CHED [Cong. Hereditary endothelial dystrophy]

(3) Blue sclera. Sclera اكتبها من P.146

Buphthalmos, High myopia, Staphyloma, Osteogenesis imperfecta, Marfan syndrom

(4) Watering of the eye in child:

① - Buphthalmos. ② - Congenital NLDO Naso Lacrimal duct obstruction

③ Viral conjunctivitis (ophthalmia neonatorum)

④ - Corneal abrasion .

◊ Complications:

(1) Loss of vision d.t. optic atrophy & corneal opacities

(2) Impairment of vision: due to :

① Complicated cataract (↓ nutrition of the lens due to stagnation of aqueous)

② Subluxation of lens , ③ corneal opacity → ⑤ ⑥

④ Nystagmus (Bilateral cases). & Amblyopia & squint (Unilateral cases)

Bilat. Nystagmus — Unilat. Amblyopic squint

لازم ألحقه قبل 6 سنين

(3) Rupture globe from minor trauma.(عيش ندل) (fragile eye)

NB. Fragile eye (avoid contact sports) مهم شفوي

1- Buphthalmos 2- High myopia 3- Blep 4- Amblyopic eye

5- Post LASIK (أحمد السقا) 6- Angiod streaks

◊ Treatment: Mainly operative

[the early interference the better the prognosis].

Glaucoma

(1) Medical: to ↓ IOP as preoperative preparation & after operation.

1- Systemic CAI e.g. Acetazoliamide(Diamox).

2- Topical CAI e.g. Dorzolamide

3- Beta blocker (Timolol 0.25% twice daily).

NB. Miotics are non-effective ,used only preoperative to stretch the iris.

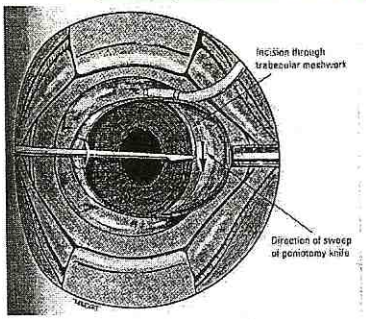
(2) Surgical: → early → goniotomy / Trabeculotomy
→ Late → external fistulization = Trabeculectomy + Molteno or Ahmed Valve

(1) Early cases: {Corneal diameter < 13 mm }

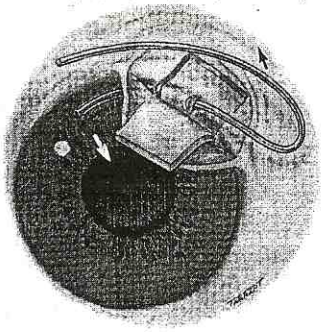
{Canal of Schlemm is present}

i) *Clear cornea* → **Goniotomy.** See atlas page (124)

ii) *Hazy cornea* → **Trabeculotomy.** See atlas page (124,136)



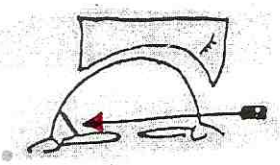
Goniotomy



Trabeculotomy

Goniotomy: (Cut in the angle)

- Under visualization with a gonioscopy.
- A goniotomy knife is introduced from the limbus across anterior chamber to cut the mesodermal membrane (Barkan memb.) in the opposite side.
  (1/3 of the angle circumference is opened).
- Complications: 1) Intra-ocular hge "hyphema"
  2) Iridodialysis.
  3) Lens injury.



Glaucoma

Trabeculotomy

INDICATIONS:

- Failure of repeated Goniotomy.
- Hazy cornea. (not clear cornea) e.g. Haab's striae

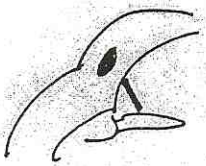
STEPS

1. Limbal incision.
2. A fine metal probe (Trabeculotome) is passed into the canal of Schlemm.
3. The probe is rotated towards the A.C.

PRINCIPLE: to expose the schlemm's canal directly to the aqueous in the AC.

STRUCTRUE TO BE CUT:

- Inner side of Canal of Schlemm
- TM (Trabecular meshwork)
- Mesodermal membrane.



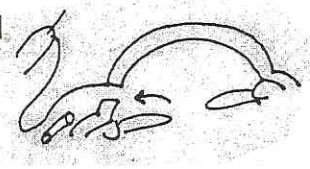
(2) Late cases: {corneal diameter ≥ 13 mm}

{Schlemm's canal absent or fibrosed}

Do → **External fistulzing operation** e.g. trabeculectomy (ectomy not otomy)

Either i) Alone

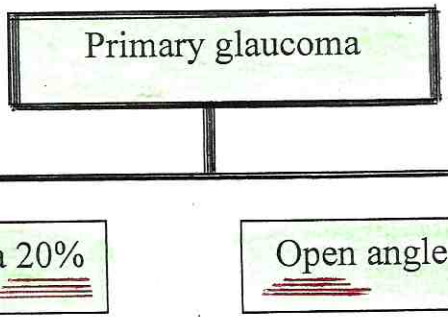
or with ii) Adjuvant use of Mitomycin C or 5 fluro-uracil → Antimitotics



iii) With use of glaucoma shunting devices:

e.g. Ahmed valve or Molteno tube. See atlas page (130)

Valve is to Tube open at certain pressure e.g. 13 mm Hg



Glaucoma

A) Closed angle glaucoma (20%)

= Acute congestive glaucoma

★ **Definition:** It is acute increase of IOP due to sudden closure of a narrow angle by the iris, occurs in attacks.

★ **Incidence:**

- Bilateral (one eye before the other).
- More common in females. ♀
- > 40 years old.

★ **Aetiology:**

(1) Predisposing factors:

1) Local: narrow angle (commonly seen with shallow A.C.) as in :

1- Hypermetropes (small eyes).

2- Old age (due to progressive ↑ in lens thickness → which push the iris forward).

3- Menopausal females: due to hormonal changes → congestion of CB → which push the iris forward).

2) General:

★ Nervous individuals → (Imbalance between sympathetic & parasympathetic) → high sympathetic tone → ↑HR → C.B. congestion → pushing iris forward.

(2) Precipitating factors:

Pupillary mid-dilatation due to → mydriatics. د.ابوليه

→ Excitement. فيلم رعب

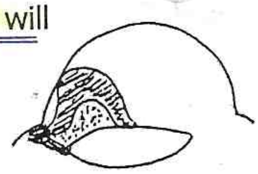
→ Prolonged stay in dark. سينما

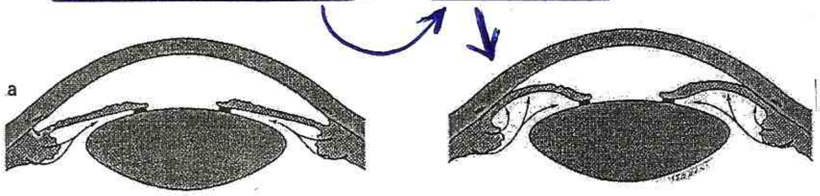
Lead to angle closure through 2 mechanisms:

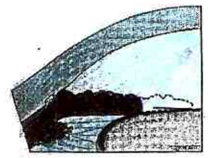
Glaucoma

(a) Relative pupillary block with iris bombé (most common mechanism):

- In eyes with narrow angle & shallow AC, there is tight apposition of iris to lens → relative pupillary block → aqueous retention in posterior chamber.
- Dilatation of pupil → relaxation of iris → the trapped aqueous will push the iris root forward → angle closure. See atlas page (76)



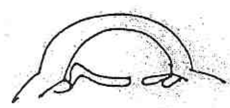




(b) Iris crowdening (Plateau iris) (less common):

Mydriasis → increase the thickness of the iris = iris crowdening.

Root of dilated iris is crowded in the angle occluding it.



> **N.B**: The best proof that 1st mechanism انها الصح is the curative effect of peripheral iridectomy or laser iridotomy.

★ Clinical picture (Stages) :

(1) Latent (asymptomatic) stage:

- By slit lamp: Shallow AC & the periphery of the iris is close to the cornea.
- Gonioscopy : grade 1 or 2
- TTT: Prophylactic laser peripheral iridectomy to decrease the risk of ACG.

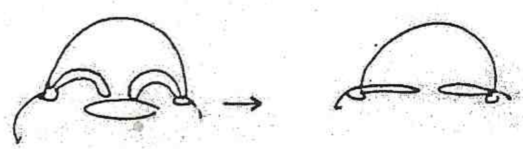
(2) (Intermittent= Subacute) Prodromal stage: انزار

* Characterized by transient mild attacks of ↑ IOP →

* Transient attacks of:

- Headache.(pressure on the outer coat).
- Blurred vision (hazy cornea).
- Colored haloes around light .

(diffraction of light by corneal edema).



(Relieved by sleep or exposure to bright light→ d.t. miosis).

Glaucoma

* **Signs:** 1) No or mild congestion.
2) Pupil: Slightly dilated, sluggish reaction.
3) Tension: Moderately elevated (30 mmHg)

* **Diagnosis:**

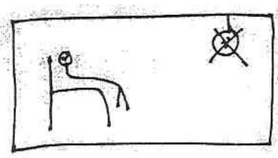
Normal tension (inbetween attack) doesn't mean that the patient is free & diagnosis depends on: لا تعتمد علي قياس ضغط العين

1) History: transient attacks after prolonged stay in the dark or excitement.
2) Gonioscopy: Revealed narrow angle.
3) Provocative tests :

*Idea: depends on pupillary dilatation. نضع الشخص في نفس الظروف

1- Dark room test:

- IOP is measured.
- Patient stays in a dark room for one hour (awake as sleeping → miosis).
- IOP is remeausred.



* **Increase IOP by 8 mmHg or more is diagnostic.**

2- Dark room prone test :

As dark room test but the pt is in prone position.

3- Mydriatic test:

- IOP is measured.
- Weak mydriatic is instilled e.g. phenylephrine 2.5 - 5% (not atropine).
- IOP is remeausred after 1 hour.

* **Increase IOP by 8 mmHg or more is diagnostic**

(3) Acute (congestive) stage:

(The attack not relieved 30 mmHg → 50 →70 ....)

Glaucoma

* Symptoms:

1) Headache & Ocular pain:

- Sudden severe bursting, due to stretch of corneal nerves.

عيني هتطلع مني= تاني اقوي وجع ف الدنيا

- referred to temple & teeth along 5th nerve branches .

ممكن يروح لدكتور السنان غلط

2) Diminution of vision (Rapid & marked) HM or PL:

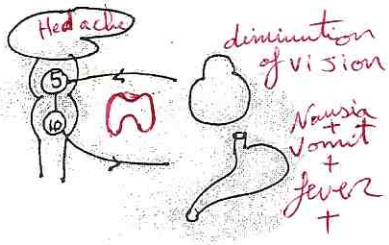
Due to a) Corneal & lens oedema.

b) Optic n. ischaemia by mechanical pressure.

3) Lacrimation, photophobia, blepharospasm & coloured haloes around light d.t. corneal oedema.

4) Nausea & vomiting: due to reflex vagal stimulation as there is central connection between the Trig. n. & Vagus n. nuclei in the brain stem.

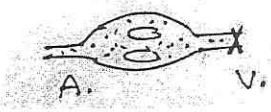
Also pain → vagal stimulation



5) Fever: as the vomiting → electrolyte disturbance → disturbance of HRC

ممكن يروح لدكتور باطنه غلط 4,5

* Signs:



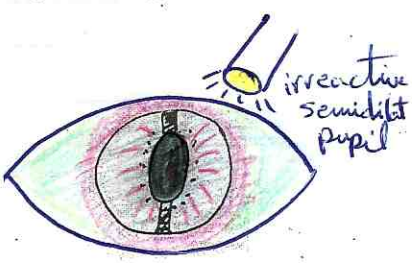
1- Lid: oedema.

2- Conj.: Ciliary injection ((**congestion** as the dilatation is mainly venous))

3- Cornea: Hazy (oedema), hypothesia.

4- AC: very shallow.

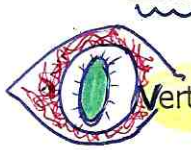
5- Iris: Congestion, iris bombe.



6- Pupil: See atlas page (125)

Semidilated , irreactive (dead position) due to paresis of both muscles

د.حمدي الكومي له تفسير اخر



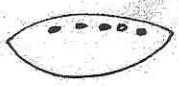
Vertically oval (as vertical fibers of the dilator ms is more strong than horizontal = segmental ↑ of the tone of the vertical fibers).

Color of the pupil :greenish blue due to corneal & lens edema

Glaucoma = Sclera Blue, Pupil Blue green

7- Lens: shows minute ant. subcapsular opacities (glaucoma-flecken = cataracta acuta).

هيدخل بكميات كبيرة للعدسة Aqueous



8- Fundus: Not seen (corneal oedema) so put drop of glycerine (hypertonic) → hyperemic & edematous op. disc. & CRA may found pulsating impending CRAO مهم

9- Gonioscoy : grade 0. irido corneal contact

10-IOP: Digitally: stony hard. (Maximum IOP is 70 mmhg (اشمعنا؟) → bec. will close B.V. of C.B.

Aviod the use of tonometry. مهم

11- Vision: markedly ↓ may reach H.M or P.L.so, visual field can't be examined.

12- Field of vision : not examined due to marked ↓ of vision.

Fate: the attack may end in:

1- Resolution → spontaneous or with ttt (20 mmHg).

2- Improves not completely → subacute or chronic stage (50 mmHg).

3- Remains → absolute glaucoma (100 mmHg)

Blind , painful eye

DD: 1) From other causes of red eye الجدول المشهور

2) From other causes of Colored haloes: a) Acute conjunctivitis.

b) Incipient cataract.

3) From other causes of rapid rise of IOP:

- Glaucomatocyclitic crisis. → open angle & deep A.C.
- phacomorphic & phacolytic glaucoma.

(4) Chronic stage: اتعالج صح بس متأخر

- In this stage: long contact between iris & cornea → PAS formation

- Miotics (ttt) will improve the acute stage but:

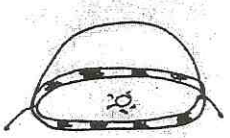
  * IOP: ↓ but not normal (due to PAS). ≃50 mmHg

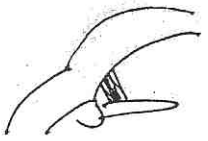
  * Optic nerve: shows glaucomatous cupping.

  * Field: glaucomatous field defects.

  * Cornea: oedema.

  * Pupil: dilated ,irreactive.





* Conj. : congestion.
* Gonioscopy: PAS.
* AC : shallow.
* Lens : complicated cataract.

(5) Absolute glaucoma: علاج خطأ أو متأخر أوي

The end stage of "any uncontrolled glaucoma"

* **Symptoms**: Blind (no. P.L), Painful eye due to Complete closure of angle.

* **Signs**:

1- **Conj. :** Ciliary congestion.

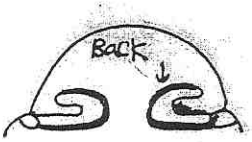
2- **Cornea:** Bollous keratopathy → ↑ size → its rupture →
ulcer (stiching pain).
→ Degenerative pannus.

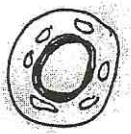
(Corneal oedema→ loss of compactness → vascularization)

3- **Sclera:** staphyloma & blue sclera
(due to scleral atrophy, showing the underlying choroid)

4- **A.C:** Shallow.

5- **Iris:** a- White atrophic patches. See atlas page (93)
b- Ectropion uvea See atlas page (125,126)
(rolling of the edge of pupil toward the AC
due to fibrosis → dark ring around pupil).
c- Rubeosis iridis.





6- **Fundus:** Optic atrophy & cupping.متوقعة

7- **Lens:** Complicated cataract (post. Cortical غالباً).

8- **Tension:** very high.

9- **Pupil:** Semidilated , irreactive but not vertically oval (vertical fibers of dilator ms becomes affected)

(No PL vision , so → → → no field).

(6) Atrophic stage:

Due to atrophy of ciliary body epith. → ↓ Aqueous formation → ↓ IOP
(Atrophia bulbi) .

Glaucoma

Treatment:

Mainly surgical

- No PAS → Laser iridotomy
  or Surgical peripheral iridectomy if the cornea is hazy.
  See atlas page (93,94)
- PAS → External fistulizing operation. Trabeculectomy + Aimed valve

(1) Prodromal stage:

AS there is no PAS → peripheral iridectomy is curative

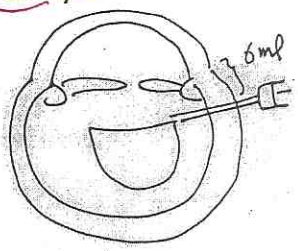
- By allowing a way for aqueous from PC → AC.
- Prophylaxis against future angle closure.
- Do peripheral iridectomy as a prophylaxis in the other eye several days after the 1st operation (as glaucoma is bilateral disease)

(2) Acute stage (emergency) :

- Hospitalization.
- ttt is essentially surgical, however Medical ttt is given for 24 hours :
  * to ↓ IOP to avoid sudden drop of IOP → expulsive Hge.

1- Medical ttt succeed in ↓ IOP: (Pain↓, vision improves , pupil constrict & tension ↓) then gonioscopy is done:

- No or minimal PAS <180° → Peripheral iridectomy.
- PAS >180° →External fistulizing operation



2- Medical ttt fails to ↓ IOP:

do posterior sclerotomy (vitreous tap)to↓ IOP surgically, then do → External fistulizing operation.

Q: gonioscope من غير PAS ازاى تعرف ان فيه

Use miotics : - miosis → no PAS

- no miosis → PAS

Q: How you can differentiate between PAS & iris bombé (between apositional & synechial angle closure)

الاجابة → Do indentation gonioscopy . د. سعيد شلبي

Angle

(3) Chronic stage: PAS are present, so do external fistulizing operation.

(4) Absolute glaucoma: this is blind, painful eye, so do:

- Enucleation: after written consent from the pt.
- Retrobulbar alcohol.
- Defunctioning op. (Cyclodestructive operation): See atlas page (134)
  - Partial damage to CB non pigmented epithelium (180° 6-8 spots)
  - → ↓ Aqueous formation → ↓ IOP → ↓ pain.

As cyclocryotherapy (painful): 8 cryo applications are done through intact bulbar conjunctiva.

Or cyclodiathermy (fibrosis).

Or cyclophotocoagulation (diode laser).

Retrobulbar injection of alcohol:

1 cc of novocaine 4% is injected followed by injection of 1 cc of alcohol 70%

Aim: to relieve pain by destroying the ciliary ganglion (demylination).

Complications:

1- Retrobulbar haematoma → proptosis.

2- Nerve lesion : →ptosis (3rd), Squint(3,4,6 th nerves), Neuroparalytic keratitis (5th n).

3- Recurrence: Due to remylination after 6-4 months.

NB. Site of ciliary Ganglion:

- 1 cm infront of optic foramen.
- Between optic n. & LR ms.

Medical treatment: pre operative for 24 hours — local, general

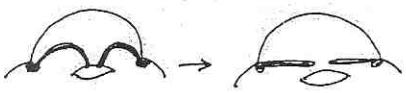
Closed Angle glaucoma

Glaucoma

(1) Local ttt:

1) Miotics:

- **Examples:** pilocarpine nitrate 2-4% drops Or eserine (obsolete).
- **Administration:** Every 5 min, till the pupil constricts, then ~~every~~ every 2-3hours for 24 hour until the operation.
- **Disadvantages:** not act if the IOP > 40 mmHg (due to temporary paralysis of the sphincter ms). علشان كده مش بنبتدي بيه
- **Action:** 1- Contraction of sphincter pupillae ms leads to:
  - Pulls the iris away from the angle .



  - Widens the iris crypts → ↑drainage.

2- Contraction of ciliary ms leads to:

- (Longitudinal part) Pulls on the scleral spur → widens spaces of Fontana.
- (Circular part)Compress the ciliary arteries → ↓ blood flow to CB → ↓aqueous formation.

NB. Although eserine is stronger but it is not used : oral وزراعة
1- Unstable (oxidized في الضوء) → Rubroeserine : weak & irritant.
2- Sensitivity → Follicular conjunctivitis.
3- Produce headache (spasm of ciliary ms).

NB. Pilocarpine 2-4 % اشمعنا؟
< 2% → Weak
> 4% → relative pupillary block

NB. Don't strat with pilocarpine → ليه
If the IOP > 40 mmhg Pilocarpine will not act

NB. Start with mannitol ? ليه → as it ↓ corneal edema?? د سعيد شلبي.
↳ ↑ penetration of other drug

Glaucoma

2) Other topical drugs:

1- Topical B-blockers: e.g. timolol maleate 0.5%.

2- Topical CAIs e.g. drozolamide. = local , systemic = Acetazolamide

fibrosis ← 3- Topical steroids : ↓ PAS & iritis.

(2) General : Analg. + Antiemetic, CAI, Dehydrating agent

1) Analgesics & Antiemetics:

e.g. morphine (10-20 mg I.V, SC & IM) strong pain killer. → only not miotic effect

(Pin point pupil → toxic dose غلط لان المورفين عاشان يجيب نتيجه محتاج)

2) Carbonic Anhydrase inhibitors (CAI)

Complications

- **Example**: Acetazolamide (diamox or Cidamex tab., IV amp).
- **Administration**: 1 Tab (250 mg) / 6 hours (IV 500 mg if vomiting).
- **Action**: 1- ↓aqueous formation (by inhibiting active secretion 60%).

  2- Improve optic nerve circulation .ازاي؟؟

Q: glaucoma قولي استخدام اخر لل systemic CAI غير في علاج ال → Cystoid macular oedema

oral

3) Dehydrating agents (hyperosmotic agents)

* **Examples:**

1- I.V: Mannitol (20-25%) : Dose: 1 gm / kg body weight , (250cc) ↓ Corneal edema

→ Rapid, dilution — Rate: 60 drops /min - 1 dp / sec.

2- Oral :

(a) Glycerine 50% :

- Dose: 1 gm/ kg/ Bw/ (150 cc) (3/4 كبايه) ½ - ¾
- Drawbacks: i) sweet → nauseating, so given with lemon juice.

  ii) It is metabolized into glucose, so given with caution to diabetics.

(b) Isosorbide 50%:

- Adv. : - Not nauseating .

  - Not metabolized to glucose so suitable in diabetics.

3- Local : - Saline 4% - Glycerin → painful. – Honey

Glaucoma

* **Action:** ↑ blood osmolarity:

(i) ↓ aqueous formation (passive mechanism 40%).

(ii) Draw H_2O out of eye → ↓ vitreous volume.

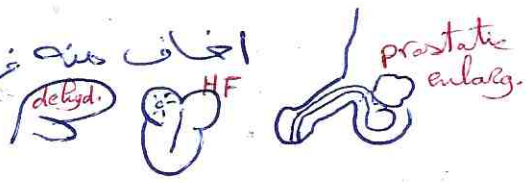
(iii) ↓ Corneal edema.

* **Administration:** Once for fear of:

1- Brain cell dehydration .

2- Circulatory overload → HF & RF.

3- In old patients with prostatic enlargement → urine retention.



Surgical treatment:

① peripheral iridect. [surgery, Laser [YAG, Argon]

② external fistulizing

③ Dejunctioning operation

(1) Peripheral iridectomy:

- **Principle:** Communication bet. post. & ant. Chambers, so prevent future closure of angle.

- **Indications:** (i) Prodromal stage.

(ii) Acute stage with no PAS. (Peripheral Ant. synechia)

(iii) Prophylactic ttt in other eye.

- **Methods:** either by (i) surgery. See atlas page (93)

(ii) Laser iridotomy (iridectomy مش):

See atlas page (94)

- Argon → heat coagulating laser.
- YAG → cutting laser.

- **Site:** upper part, over any crypt.؟؟د. حمدي الكومي له, Miotic preoperative.

- Advantages of laser surgery:

1- Out-patient procedure: (no hospitalization, no general anesthesia)

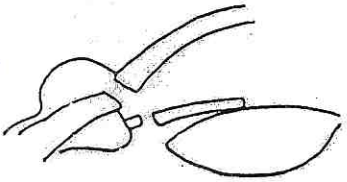
2- No risk of endophthalmitis or wound leak or I.O. hge.

- Disadvantages of laser surgery:

1- Cannot be done with hazy cornea. or oedema

2- Multiple settings may be necessary.

3- The opening will be small so can be closed.



(2) External fistulizing operation: e.g: trabeculectomy

- **Indications:** (i) Acute stage occurs on top of chronic as PAS are formed during a previous attack.
  (ii) Chronic stage.

(3) Defunctioning op. (Cyclodestructive operation) :

Partial damage to CB epithelium (180 °) → ↓ Aqueous formation → ↓ IOP→ ↓ pain.

As cyclocryotherapy (painful) :

Or cyclodiathermy (fibrosis).

Or cyclophotocagulation (diode laser).

-Indications: 1- Absolute glaucoma.
2- Failure of many fistulizing op.
3- Neovascular glaucoma.

Open angle glaucoma

Most Common 90%

2nd Cause of Blindness in egypt

Chronic simple glaucoma

(Glaucoma simplex)

◊ **Definition:** Bilateral, Asymmetrical, non-congestive ↑ of IOP in absence of angle closure Leading to optic. n. damage & visual field defect.

◊ **Incidence:**

- **Age:** above 50 yrs (maximum in 7th decade).
- **Sex:** Equal. (Closed = ♀)
- **Bilateral:** But one eye precedes the other.

What is Aetiology?
→ with age T.M. sclerosis

N.B.

* OAG is the commonest type of glaucoma :
  - Affecting 90% of gl. cases.
  - 2% of population > 50yrs.
* 2nd cause of blindness in Egypt.(Now it becomes the 1st cause after PC& trachoma).

Glaucoma

◊ Risk factors

- Positive family history.
- Race: More common in black.
- Ocular diseases: myopia & CRVO.
- Systemic diseases: DM, vasospastic disorders: migraine.
- Ocular hypertension (↑ IOP with no manifestations of glaucoma).

◊ Etiology:

With age →

1) Sclerosis of T.M → narrowing of spaces of Fontana → ↓aqueous outflow.
2) Sclerosis of Schlemm's canal & exit channels.

→ decrease of aqueous outflow facility.

~~3) Proliferation of endothelia lining of the canal of schlemm.~~

◊ Clinical picture:

Symptoms:

1. **Asymptomatic .**
2. **Mild (few) symptoms as:**
   a) Headache : chronic , simple.
   b) Premature (early= pathological)presbyopia:
      d.t. weakness of ciliary ms. with failure of accommodation d.t pressure on ciliary ns. & vs.
   c) Delayed dark adaptation even night blindness
      d.t. peripheral field defects.
   d) Field defect: that progresses gradual & painless
      → Suddenly discovered complete loss of vision in one eye
      (when the macula is affected).

العيان مش هيشتكي الا في الاخر يكون نظره راح

- No pain: tension 35 mmHg.
- No vision affection: as the macula is the last part to be affected in the retina.

Glaucoma

Signs: 3 Cardinal ↑IOP, Cupping of disc, Field changes

① ↑ Tension ② Gl. Cupping ③ Filed defects ④ Gonioscopy → OPEN ANGLE

+ Corneal thickness (pachymetry) مهم جدا:

- Thin cornea → Underestimation e.g post lasik مهم جدا
- Thick cornea → Overestimation.

Tension:

↑ IOP above 21 mmHg in presence of open angle is suspicious.

But if Tn → not exclude OAG (may be normal tension glaucoma)

Do the following:

(I) Tension in both eyes:

- Normally the difference doesn't exceed 2-3 mmHg.
- Difference more than 8 mmHg is suggested.

(II) Diurnal variation:

- Normally it doesn't exceed 2-3 mmHg.
- In hospital the IOP is measured every 4 hours for 24 hours.
- Variation is 8 mmHg or more is suggestive.

(III) Measure the corneal thickness (pachymetry):

- Thin cornea → Underestimation.
- Thick cornea → Overestimation.

So pt with ocular hypertension usually have thicker cornea

Pt with low tension glaucoma usually have thinner cornea.

(IV) Provocative tests:

* **Idea:** ↑ aqueous formation (as drainage is not normal → IOP will ↑)

(1) Water drinking test:

1. Patient comes fasting & IOP is measured. Then,
2. Patient drinks a liter of H_2O (Over 5-15 min to avoid reflex diuresis) → ↓ osmolarity of blood → ↑aqueous formation.

Glaucoma

3. Tension is remeasured after one hour, ↑ IOP by 8 mmHg or more is diagnostic.

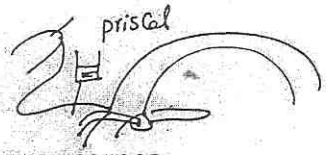
(2) Priscol test:

Vasodilator

1) 10 mg priscol (V.D) is injected subconjunctivally→ ↑ blood flow to CB →↑ formatiom of aqueous .

2) Tension is measured before & after one hour of injection.

3) ↑ IOP by more 8 mmHg is diagnostic.



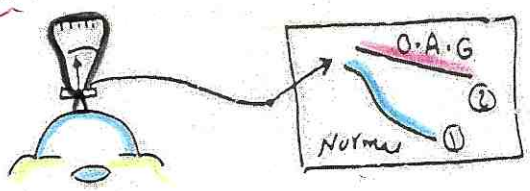
(3)Tonography:

- An electronic Schiotz tonometer is continuously placed over cornea for 4 min.
- Tension is measured every 1/4 minute (16 readings).
- A curve is drawn, from curve the facility of aq. outflow is calculated.

(give an idea about the aqueous outflow :

② - Sloping curve → normal.

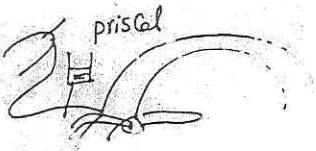
② - Nearly flat curve → OAG.)



X(4) Jugular vein compression : مهم المحاضره

↑ IOP by more 8 mmHg is diagnostic.

سؤال. هنعمل Jugular vein compression باستخدام ايه؟؟؟

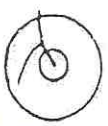


Glaucomatous cupping:

Late

early

* Normal cup/ disc ratio (C/D) is 0.3 or less
* OAG cup is characterized by : C/D > 0.4 & Assymetric bet. 2 eyes) in early stages



* LATE CHANGES: See atlas page (126,138,139)

1- Large cup/ disc ratio 0.6 – 0.9.

2- Deep (can be measured by ophthalmoscope every 1 ml depth = 3D approx).

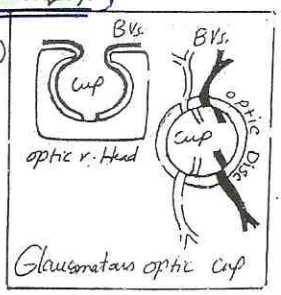
3- Nasal shifting of the BVs come from the cup See atlas page (139)

Apparent bec. Temporal first necrosis (affected)

4- With overhanging (undermined) edge يعني لازم

(flask shaped) & the BV appears as if interrupted

لكن not interrupted نقطة شكل كده مش صحيح



Glaucoma

5- Arterial pulsation in sever cases.

6- Post glaucomatous optic atrophy

(late: cupping & Pallor) .

✷ EARLY CHANGES:

C/D Ratio > 0.4

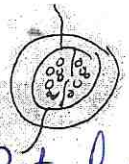
7- Asymmetry of cups of both eyes(≥ 0.2). See atlas page (139)

Splinter Hge

8- Splinter haemorrhage : See atlas page (139)

are of red infarction last for 2-6 months

9- Vertical notching (vertical elongation of cup). See atlas page (138)



10- ↑ visibility of pores of lamina criprosa.

11- Prominent α & ß Zones around the disc. (peripapillary chorio Retinal Atrophy)

12- RNFL defect (BVs at the area of defect appear darker& prominent).

Retinal nerve fibre layer

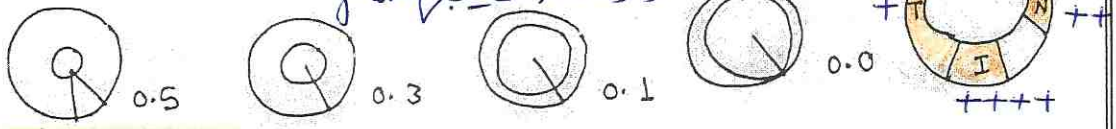
To see NFL use red free filter during examination.

in glaucoma
* ↑ Cupping C/D
* ↓ neural rim R/D

NB. 1- Recently we use R/D (rim/disc) ratio instead of C/D ration

Normal R/D ratio = 0.5 (this number ↓ as glaucoma advances = thining of rim).

2- Normally the neural rim follow ISNT rule.



Neural retinal rim : is the area between the cup & the outer edge of the disc, it contains the nerve fibers.

3- OCT is done to detect defect or thinning of the nerve fiber layer.

What is OCT ? See atlas page (148)

optical Coherence Tomography

- Optical Coherence Tomography
- This technique use a polarized light to make an optical sections of the retina & ONH.

-Value:

1- Study the thickness of the nerve fiber layer (early affected in glaucoma).

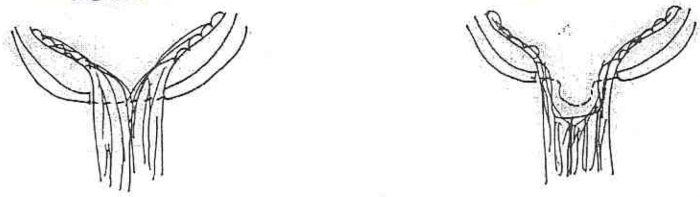
2- Study the macular lesions (oedema , hole or vitromacular traction,epiretial membranes).

Glaucoma

N.B. The cause of glaucomatous cupping may be:

(1) Mechanical theory: ↑IOP → Backward bowing of lamina cribrosa, as it's the weakest part of sclera + Compression & degeneration of optic nerve fibers.

Cupping in Normal tension glaucoma where no ↑IOP But +ve cupping & +ve nerve atrophy



(2) Ischaemic (Vascular) theory: Sclerosis of vessels that supply disc → optic nerve fibers atrophy.

(This explains cupping with low tension glaucoma).

(3) Apoptosis: Auto digestion of the ganglion cells & their Axons.

(4) Genetic.

(5) Autoimmune free radicals.

(6) Glutamine toxicity → ca++ ions influx into the cells → cell death.

NB. HOW u can evaluate the optic n. head?

1- Direct ophthalmoscope
2- In direct ophthalmoscope
3- Slit lamp biomicroscopy with auxiliary lenses
4- HRT (Heildelberg retinal tomography = Scanning laser ophthalmoscope): Objective method for evaluate the optic n. head, it will give you 3 dimensional map of the disc

3D map of disc +

5- OCT → optical Coherence Topography

Glaucoma

Field changes: → Central → Peripheral → Complete

early diagnostic — Late prognostic

40% of O.N. fibers Lost

Cause: due to optic nerve fibers damage caused by ischemia & mechanical Pressure (field defect appears if 40 % of optic n. fibers are lost).

Rules: 1- The most crowded fibers affected first.

Arcuate(Temporal) f.→ Nasal f. → macula)

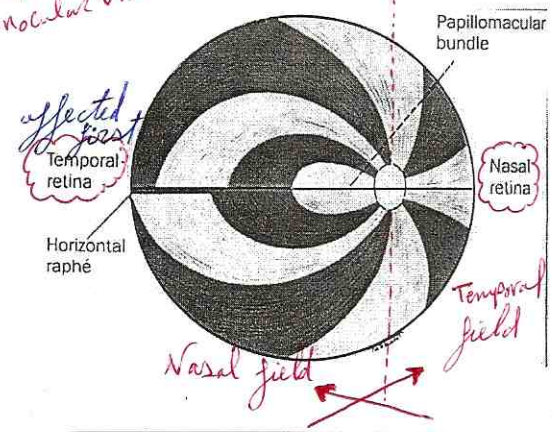
2- The most central receptors are affected first .

3- Macula is the last to be affected (good vascularity & least crowded)

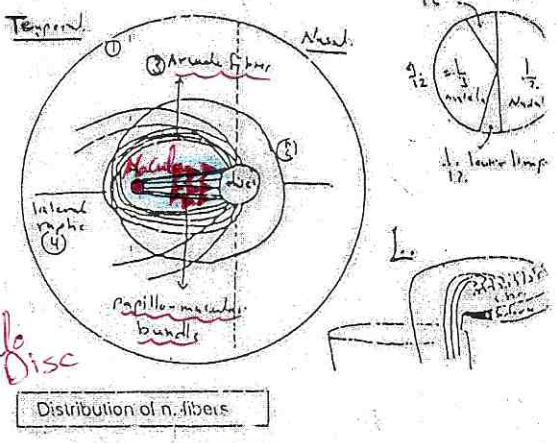
N.B.: Scotoma: means an island of blindness surrounded by sea of vision.

Blind spot of Mariotte : corresponds to the optic disc, present in the central field between 10 & 20 degree on the temporal side of fixation, it is the best example for absolute negative scotoma.

visually not sensed bec. ① physiological myostegmus ② not represented in brain ③ Binocular vision



affected first



Papille = Disc

Distribution of n. fibers

(I) Changes in the central field:

Early Diagnostic

- The changes are early & diagnostic.

Normally - Central field : is the central 30° of the visual field around the point of fixation (corresponds to fovea).

- Testing (Campimetry = Bjerrum screen): See atlas page (129)

1×1 or 2×2 meters at the center of which there is a white fixation spot.

& pt. sits at a distance of 1 meter.

Glaucoma

Central field changes

- The changes include:

(a) **Baring of the blind spot:** Exclusion of blind spot from the central field (due to contraction of the central field).

(b) **Isolated paracentral scotoma:** Later enlarged & connected with blind spot → Seidle scotoma.

(c) **Seidle scotoma:** Extension of blind spot above or below in a sickle shape . شكل منجلي

(d) **Bjerrum (Arcuate) scotoma:** which is continuous with blind spot &arch above or below the fixation point to meet the horizontal raphe.

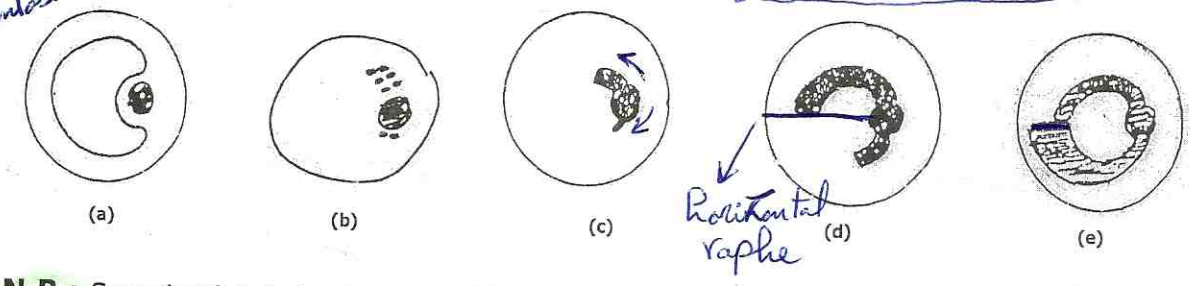
(e) **Annular (Ring= Double arcuate) scotoma:**

Due to fusion of the 2 arcuate scotoma upper & lower, its continuous with the blind spot (central)

NB . There may be Ronne step where the 2 arcute scotoma meets

With open Central angle glaucoma

Retinitis pigmentosa

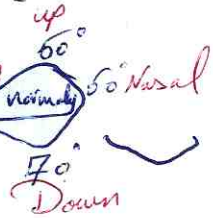


N.B.: Superior & inferior temporal fibers of optic disc are most vulnerable to glaucomatous damage, fibers of papillomacular fibers are most resistant.

(II) Changes in the peripheral field:

Late prognostic

* The changes are late & prognostic.
* The peripheral field limits - Up & nasal→ 60° - Down→ 70° - Temporal→ 90°



*** Testing with perimetry :**

① **♦ Kinetic:** يستعمل وتابت الضوء والحجم.

It involves presentation of a moving stimulus of fixed size & Intensity from non-seeing area until its perceived .

e.g.Goldmann's perimetry. See atlas page (129)

② **♦ Static :** متغير القوه والحجم وثابت

It involves presentation of non-moving stimuli of varying Intensity in the same position .

e.g. Automated perimetry (Humphery & Octopus):

See atlas page (127,128)

- More accurate & rapid
- It facilitates screening.
- Goldman & automated perimetires test both central & peripheral fields
- Displays: مهم جدا الروند
  - * Reliability indices : more than 33% Unreliable
    1- Fixation losses
    2- False positive
    3- False negative
  - * Geary scale , numerical scale, Total Deviation & patten deviation
  - * Global indices: e.g MD - 2 : - 6 → mild
    - 6 : -12→ mod.
    > -12 → severe.

③ **♦ SWAP (short wave automated perimetry):** المحاضرة

This test detect early glaucoma as blue cones (M cells) are scarce in the retina & affected early, نادر

here we use blue target on a yellow background .

④ **♦ MFERG (Multi focal REG) :**

it is objective method for testing the filed المحاضرة

→ Topographic map of retinal function.

*** These changes include:**

(a) Nasal contraction:(as temporal fibers more dense & so suffer more)

Glaucoma

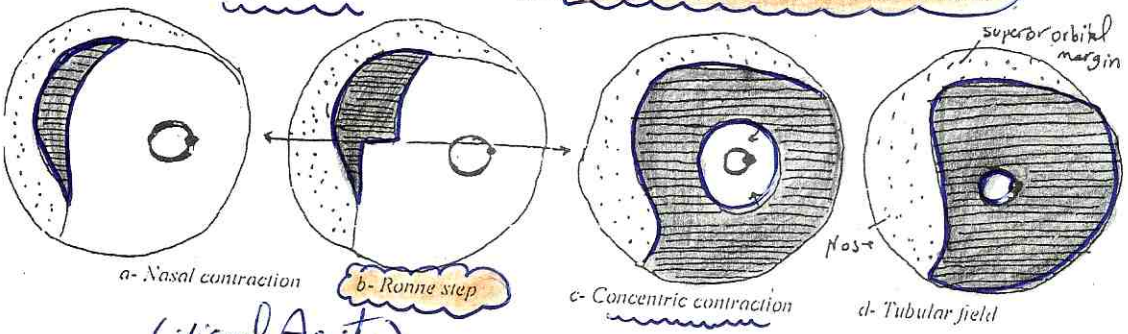
Peripheral changes تابع

(b) Roenne step : nasal field defect with sharp horizontal border at the site of the lateral raphe (asymmetrical shape of upper & lower field defects that meet at the horizontal meridian).

(c) Concentric contraction: More on the nasal side. See atlas page (127)

(d) Tubular field: central & peripheral fields meet & papillomacular fibers still normal. See atlas page (127) only macula normal

(III) Complete loss of vision: is the end stage with fusion of central & peripheral field changes with affection of macular fibers.



(Visual Acuity)

N.B.: V.A. is not a measure for severity of OAG because tubular 6/6 vision remains till the end stage of disease .

Q: What is ICE water hand immersion perimetry? من المحاضرات

It is to do perimetry before & after ICE water hand immersion for 1/3 hours for detection of early cases of glaucoma

(VASCULAR STERSS TEST)

D.D.:

(1) Causes of diminution of vision especially: -

a) cataract b) Optic atrophy c) retinal detachment d) chronic iridocyclitis.

(2) Causes of optic cupping; physiological & atrophic.

(3) Other glaucoma: primary closed angle glaucoma: gonioscopy→ narrow angle

(4) Causes of tubular field.

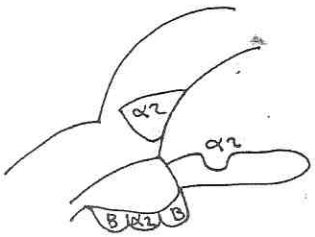
(5) from ocular hypertension & low tension glaucoma الجدول

Glaucoma

Treatment: TTT of OAG is essentially **medical & for life**

- **Medical**
- **Laser trabeculoplasty**
- **Surgical Ext. Fist. op. Trabeculectomy**

(A) Medical TTT:



(1) Local TTT:

1) B-Blockers : دايماً بنبتدي بيها العلاج

- **Examples:** * Timolol maleate 0.25 – 0.5 %
  * Betaxolol HCl (Betoptic) :
    - cardioselective B- blocker(B1)→ not induce bronchospasm.
    - Neuroprotective effect.
    - Weak action in ↓ of IOP.
- **Administration:** Every 12 hours.
- **Action:** ↓ aqueous formation (Block B receptors on ciliary body epith.)
- **Side effects:**
  - *** LOCAL:** - Dry eye(Block B receptors on epith. of lacrimal gland)
    - Punctate keratitis
  - *** GENERAL:** - Bronchospasm(use selective BB) Contra Indic. in Bronchial Asthma
    - Bradychardia & hypotension even heart block (قيس النبض الأول)
    - Depression & impotence as it cross BBB.

NB> BB not given at bed time ليه

2) Prostaglandin Analogues:

- Examples: as * Latanoprost (Xalatn 0.005%).
  * Travoprost (Travatan).
  * Bimatoprost (Lumigan).

Glaucoma

2) Prostaglandins Action: ↑ uveo-scleral out flow. ازاي؟؟

- Side effects: 1- Change iris colour (so not used in unilateral cases , (يلاحظها الاوروبيين فقط)

2- Redness.

2ry glauc. 2ry to iritis

3- Iritis (so it is contraindicated in glaucoma 2ry to iritis)

4- Overgrowth of lashes.

5- CMO Cystoid Macular edema

- Administration: once at night (so redness will come during sleep)

3) Topical CAIS: Carbonic Anhydrase Inhibitors

- Example: Dorzolamide (Trusopt 2%). → systemic Acetazolamide
- Administration: 2 times / day
- Side effects: burning pain, irritative conjunctivitis , belpharitis &high cost.

4) Alpha agonist:

1- Epinephrine1-2% (α & ß stimulant)**:**

- **Example:** adrenaline 1-2% (obsolete).
- **Action:** *↑ Aqueous out flow (stimulation of α2 receptors in the TM, but doesn't reach B receptors deep in the CB) also it↑ uveoscleral out flow .

* ↓ aqoues formation : stimulation of α2 receptors in the ciliary epith which are inhibitory receptors.

- **Side effects:**
  - GENERAL: ↑ HR & hypertension.
  - LOCAL: conj. Pigmentation & mydriasis
- **Contraindication:** CAG as mydriasis will precipitate the attack. Closed angle glaucoma

2- Dipivalyl epinephrine(Dipivefrine) : → يتحول Adrenaline

prodrug→ adrenaline inside the eye so no systemic side effects. ← Local only داخل العين

3- α2 Agonists :

- **Brimonidine 0.2%** (**Alphagan**): - ↑ outflow. in ciliary body

- ↓ aqueous formation.
- neuroprotective effect.

- **Aprachlonidine :** loss the effect after 2 months(tachyphylaxis).

5) Miotics : زمان كانت أول اختيار دلوقتي فيه حاجات أحسن

- **Examples:** Pilocarpine 1-4% drops.
- **New forms of pilocarpine (long acting):**
  1- Ocusert : sandwich releasing pilocarpine at a steady rate for one week.
  2- Gel.
- **Action:** contract the longitudinal ciliary ms that pull on the scleral spur → widen the spaces of Fontana.
- **Administration:** 3-4 times dialy /for life.
  (so frequency & concentration must be the least that control IOP).
- **Side effects:**
  1- Ciliary spasm :→ Headache & fluctuating myopia تحصل مع القطرة (1-2D)
  2- Miosis: → ↓ night vision, contraction of peripheral field & marked diminution of vision in nuclear cataract.
  3- Ocular congestion (VD).
  4- In a high myopic pt. it may induce RD.
  5- Pupillary block.

6) Fixed combinations :

- e.g. 1) Dorzolamide with Timolol (Cosopt).
  2) Xalatan with Timolol (Xalacom).
- Advantages: fewer daily drops.

(2) General TTT:

1) Carbonic Anhydrase inhibitors: (Acetazlomaide tab or amp).

* **Example, administration & action:** as before.

* **Uses**: before operation, not for long term use, d.t its side effects

*** Side effects:** ↓K^+

(1) Hypokalemia → parasthesia of fingers & toes (give K+ tab),
So it is contraindicated in Diabetic pt as there is hypokalemia with DM

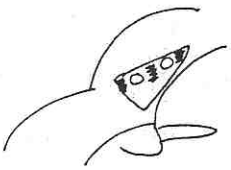
(2) Leucopenia: due to bone marrow depression.

(3) Renal stones. (4)Vomiting & diarrhea (GIT disturbance),

2) Neuroprotectives & neuroregeneratives : (Antioxidants e.g. Vit A,C,E) prevent the toxic effect of free radicals liberated from dead cells

N.B: Follow up: at regular intervals for : tension, disc & field is essentail

(B) Laser Trabeculoplasty:



- **Principle:** 1- By inducing minimal laser burn at trabecular meshwork (50 burn/180°), using gonioscopic lens, That contract
  → widening of spaces of Fontana.
  2- To induce inflammation →marcrophages→ will engulf the debris inside the TM. مهم
  3- Laser induce opening in the TM
- **Laser:** Argon (ALT), recently Diode laser is used.
- **Advantages:**
  1) Out patient procedure without anaesthesia.
  2) Fewer complication than the surgery as the eye is not opened.
- **Disadvantages:**
  - Decreased success rate.
  - If the laser burns comes posterior to the scleral spur→ post laser rise of IOP & PAS formation.
- **Indications:** 1- With medical ttt .(to avoid poly pharmacy)
  2- Especially with pigmentary or Pseudoexfoliation glaucoma (as the pigments absorb laser)

Glaucoma

RECENTLY SLT (selective laser trabeculoplasty): مهم

- Select pigmented, with no thermal effect so it's safer

(C) Surgical treatment:

(1) External fistulizing operations :

(Sub-scleral trabeculectomy = Filtering op.):

* **Indications** : if

(1) Medical TTT fails to control IOP (target ↓ 30%).

(2) Significant side effects of medical ttt.

(3) Deterioration of field (inspite of IOP control).

(4) The patient is poor, negligent or cannot be followed up هو ده العيان المصري

(5) Young pt (increased life expectancy)

* **Principle** : A fistula between A.C & subtenon's (sub conjunctival)space where aqueous is absorbed by sub conj. BVs.

* **Steps of trabeculectomy:** See atlas page (135)

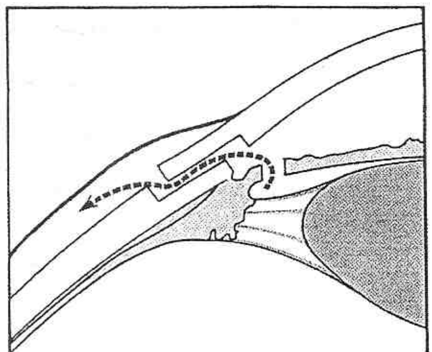
1- Lid retraction .

2- Sup. Rectus suture.

3- Conj. Flap. (limbal based or fornix based)

4- Scleral lamellar flap is raised:

- Rectangular 3×4 ml.
- Lamellar 1/2 thickness of sclera.
- Attached to the limbus.



Trabeculectomy operation

NB. Function of the scleral flap :

- Limits the flow of aqueous.
- Prevents extension of infection inside the eye, especially if Button hole occurs in the overlying conjunctival flap.

5- Paracentesis before opening the globe.

6- A block of TM is excised (Trabeculectomy): through deep sclerectomy مهم including the TM to enter the AC.

7- Peripheral iridectomy.

to prevent closure of Angle (fistula) by Iris

8- Closure of sclera by interrupted 10/0 nylon sutures at the posterior corners .

9- Closure of the conj. To make a bleb. See atlas page (130)

NB.: To keep the fistula opened :

1- Setons (implants).

2- Mitomycin C or 5 fouro-uracil.

3- Iris inclusion operation.

NB. Recently : Non penetrating glaucoma surgery :

Principle: exposure of ant. TM & DM after removal of the roof of Schlemm's canal through deep sclerotomy

(2) Drainage(Seton = Shunting) implant: See atlas page (130)

The same principle as external fistulizing operation + using:

- Motleno implant (non valved),

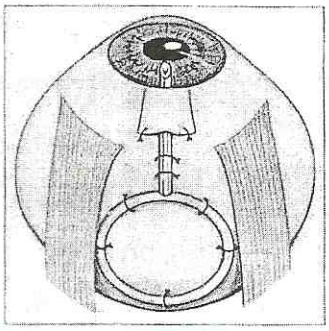
or - Ahmed implant :

(valved → pressure controlled د. ساميه (أحسن لأنه

Indications: 1- Recurrent glaucoma operation.

2- NVG (neovascular glaucoma.)

3- Secondary glaucoma



(3) Defunctioning op.: Cyclodiathermy, Cyclocryotherapy & Cyclophotocoagulation on C.B to ↓aq. Formation.

Indication: in absolute gl.

N.B: Internal fistulizing operations (Cyclodialysis):

- **Principle**: to communicate (fistula) between A.C. & suprachoroidal space where aqueous is absorb by choroidal blood vessels.

- **Indication**: in aphakic glaucoma (as vitreous may herniates& block the ext. fistula).

RECENTLY :aphakic glaucoma is treated with:

External fist. operation + anterior vitrectomy.

Glaucoma

	Closed angle Gl.	Open angle GL.
(1) Incidence	Less common 20%	More common 80%
(2) Age	Above 40 ys	Above 50 ys
(3) Sex	More in female	Equal
(4) Patient	Nervous pt.	Any
(5) Eye	Hypermetrope	Myope
(6) Cause	Angle closure	Trabecular sclerosis
(7) Prodroma	Present	Absent
(8)Symptoms	Marked	Few
(9)TTT	Essentially surgical	Medical

Hypotony

- **Definition**: IOP below 8 mm Hg.
- **Aetiology**:

(1) General causes: dehydration due to:

(i) Diabetic coma (ii) uraemia

(iii) Hyper osmotic agents.

(2) Local causes:

1- ↓ Aq. Formation: * Post inflammatory (cyclitis)

* Post traumatic (C.B shock)

2- ↑ Aq. Drainrage: **cornel fistula , wound gap &**

{**R.D.** + liquefied vitreous that passes through the tear to be absorbed by the BVs under the retina e.g. high myopia }

- **Complications**: IOP↓ → V.D → transudation →

retinal & disc oedema (papilloedema) & RD.

SECONDARY GLAUCOMA

سؤال تحريري

- **Definition:** it is ↑ of IOP secondary to local or systemic disease.

- **Aetiolgy: (causes)**

(A) SYSTEMIC DISEASES :

- it results from rise of pressure in the episcleral viens → ↓ of aqueous drainage

e.g. Dysthyriod ophthalmopathy ,superior vena caval obstruction

CS thrombosis . CC fistula.

(B) LOCAL DISEASES

(1) Corneal:

1. Corneal ulcer: due to iritis → plasmoid aqueous (OA)
2. Leucoma adherent & ant. staphyloma: due to PAS (CA)
3. Corneal fistula: (after closure)due to PAS. (CA)

(2) Ant. Chamber:

1) Plasmoid aqueous, hypopyon & hyphaema (OA).

2) Epithelial cyst (epithelia invasion of AC)

due to - Wound leak.

- Corneal fistula (not treated) → pupillary block (CA).

3) Ant. Dislocation (glaucoma inversus) → pupillary block (CA).

Any glaucoma due to pupillary block = glaucoma inversus

(3) Uveal tract:

1) Acute iritis:

- Early : the IOP decrease as the CB is inflammed.
- Late : the IOP increases d.t. plasmoid aq., hypoypon(OA).

& oedema of the TM especially when the CB regains its activity.

or steroid used during ttt

TTT: ttt of glaucoma & inflammation (miotics CI) → Contraindicated @ ↑ Congestion & VD @ Posterior Synechiae

2) Chronic iritis: d.t. PAS or ring synechia (CA) or steroid used during ttt

3) Rubosis iridis: d.t. closure of angle by new vessels→NVG.

See atlas page (100,101)

(4) Lens causes (Phacogenic causes of glaucoma) مهمه جدا:

A. OPEN ANGLE

1. Phacolytic glaucoma (hypermature cataract): See atlas page (108)

* Due to leakage of irritant degenerated lens matter through microscopic opening in the lens capsule, which will be engulfed by macrophages that swell & trapped in TM.

(NB. The capsule is intact)

* ttt : lens extraction+ BB+ steroids

2. Traumatic cataract (Phacoanaphylactic or phacoantigenic) :

Follows trauma or ECCE, with rupture of lens capsule & liberation of lens matter into ant. chamber→ initiates auto immune reaction (iritis). the products of this reaction are deposited in the TM.

(NB. The capsule is opened).

3. Subluxation or post. Dislocation: due to iritis by the herniated vitreous (OA)

4. Exfoliation glaucoma : See atlas page (129,130)

* Due to exposure to infra-red rays→ exfoliation of lens capsule → deposited at angle → **True exf. Glaucoma (glaucoma capsulair).**

NB. Also infra-red rays may lead to glass blowers cataract.

* Excessive production dandruff-like material from ocular epith. → deposited at angle

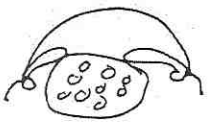
→ **Pseudo exfoliation Glaucoma**

NB سؤال د عبدالباسط. Miotic- mydriatic action of pupil → giving rise to 3 zones → Central disc & peripheral band with a clear zone inbetween.

* ttt : - Medical ttt.

- Lens extraction - Laser trabeculoplasty .

B. CLOSED ANGLE



1) Phacomorphic glaucoma: d.t. intumescent cataract

See atlas page (108) → pupillary block.

2) Anterior dislocation: due to pupillary block

See atlas page (113) (glaucoma inversus).

3) Posterior dislocation or Subluxation:

it leads to pupillary block by vitreous.

4) Micro-spherophakia: See atlas page (117)

Small, rounded lens with long relaxed zonules

→ pupillary block.

TTT: Removal of the lens:

C. Aphakic glaucoma:

It is increased IOP: following cataract extraction due to:

(1) Postoperative iritis (manipulation or retained lens matter).

(2) Postoperative hyphaema.

(3) Pupillary block by vitreous , air (after cataract op. to reform AC) & AC IOL.

(4) Steroid induced glaucoma.

TTT : it's a refractory glucomma so

1) Filtering op. + Mitomycin C or iomplnat

2) Correction of aphakia (scleral fixatig IOL if no post. Capsule)

3) Ant. vitrectomy

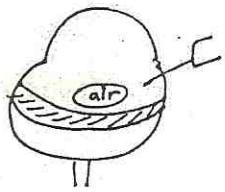
(5) Retinal causes:

(a) Diabetic retinopathy & CRVO : due to retinal ischaemia → release of vasogenic factor → rubeosis iridis

→ Hgic glaucoma(due to hyphaema).

→ NVG (neovascular gl. =100 day glaucoma) as it occurs 3 months after CRVO.

(b) Retinal detachment surgery : due to



1- Tight scleral buckle.

2- Intraocualr injection of gases or silicon oil.

3- Steroid ttt.

(6) Vitreous hge.

(7) Intraocular tumours:

Mechanism : 1- Space occupying lesion.

2- Pushing the iris-lens diaphragm forward→ angle block.

3- I.O Hge. → Ghoast cell glucoma

4- iridocyclitis : Hypopyon or hyphaema.

5- Pushing the iris-lens diaphragm forward→ angle block.

6- Malignant cells invade & block the angle

(seedling in retinoblastoma).

7- Robiosis iridis

8- infiltration of the vortex veins.

(7) Trauma:

Due to (1) Hyphaema.

(2) Corneal ulcer & traumatic iritis.

(3) Angle recession glaucoma :مهمه

Trauma → separation between longitudinal & circular fibers of the ciliary muscle → Angle recession ,

Glaucoma developed years after the trauma due to development of hyaline membrane on the recessed area extending to the TM.

ttt : - Medical ttt

- Filtering op.

(4) Siderosis bulbi d.t. I.O iron FB → damage of the T.M by ferrous oxide.الصدأ

(6) Corneal laceration with iris prolapse into the wound.

(7) Lens dislocation.

(8) Iridocyclitis by lens matter.

(9) Chemical burn: due to - Trabecualr damage.

- & scarring of the episcleral viens.

(8) Drugs:

(1) long term Steroids:

1- Deposition of hyalouronic acid crystals
d.t. inhibition of hyalourindase enzyme.

2- ↑Aqueous formation d.t. salt & H2O retention → ↑ passive formation of aqueous (if steroid is taken systemic) .

3- Deposition of TIGR protein & MPS in the TM.

(2) Mydriatics: in eyes with narrow angle→ iris bombé → CAG.

(9) Miscellaneous:

*** Pigmentry glaucoma:**

1) **Due to PDS (pigment dispersion sendrome) :** See atlas page (134)
↑ pressure in AC > PC → bowing of the iris to rub against lens & zonules → releasing of pigment from the post. surface of the iris ⇒ deposited in the TM → pigmentry glaucoma
⇒ Deposited on the back of the cornea
→ Kruken burge spindle. See atlas page (76)

2) **High myopia** 3) **DM** (thickened iris)

*** Ghost cell glaucoma:**

due to : 1) Vitreous hge + aphakia or pseudophakia.
or 2) hyphaema.
→ degenerated RBCs (RBCs which lose its hemoglobin = ghost cells) will close the angle.

*** Malignant glaucoma:** في الزيادات

Glaucoma زيادات

♦ What is the DD of 1ry OAG?

(1) Causes of gradual painless diminution of vision (see ocular symptoms).

(2) Causes of optic cupping.

(3) Other glaucomas.

♦ What is the DD of optic cupping?

Characters	(1) physiological CUP	(2) Atrophic CUP	(3) Glaucomatous CUP
(1) Cup:			
1. Size:	Small.	Moderate.	Large.
2. Depth:	Usually shallow.	Very shallow.	Deep.
3. Edge:	Well defined.	Ill-defined	Overhanging.
(2) Disc:			
1.Colour:	Pale pink.	Pale.	Pale.
2. Vessels at its edge:	1- Pass normally. 2- No pulsations.	1- Pass normally 2- No pulsations.	1- Kink or interruption. 2- Abnormal **arterial** pulsations.
3. Peripapillary halo:	None.	None.	Present.
(3) Other signs:	None.	Of the cause.	Of glaucoma.
(4) Fluorescein angiography:	Fluorescence.	No fluorescence.	No fluorescence.
(5) Vision:	Not affected	Diminished.	Diminished.

Glaucoma زيادات

♦What is the DD of glaucomas?

Character	(1) Congenital glucoma	(2) CAG	(3) OAG
(1) Cause:	Congenital angle anomalies	Angle closure.	Trabecular sclerosis.
(2) Incidence: 1- Age: 2- Sex: 3- Glaucoma: 4- Refraction: 5- Personality:	 Early childhood More in boys Less common Axial myopia Any personality	 Above 40 yrs. More in females Less common Hypermetropia Nervous	 Above 45 yrs. Equal in both sexes Common Any refraction Any personality
(3) Symptoms: 1) Onset: 2) Early symptoms	 Rapid Photophobia, lacrimation, large globe	 Sudden Haloes, pain, ↓ of vision up to HM	 Gradual No symptoms early, pre-mature presbyopia
(4) Signs: 1- AC: 2- Angle: 3- Tension: 4- Fundus: 5- Field: 6- Other signs:	 Deep Closed by membrane. High Late cupping Early changes Large globe, tremulous iris, axial myopia .....etc	 Shallow Closed by iris crowding or pupillary block. Stony hard Late cupping Late changes Dilated oval irreactive pupil, ciliary congestion	 Normal Open. High Early cupping Early changes Sluggish pupil late.
(5) TTT: 1) Medical: 2) Surgical:	 Of little value before the operatio. Essential	 Essential before the operation. Essential	 Ideal if it controls the case IF medical ttt failed
(6) prognosis:	Bad if not treated early	Good	Bad if undiagnosed early

♦ **Why tonometry is not used as a routine for measuring the tension in acute congestive glaucoma?**

Because it should be used cautiously if needed to avoid rupture of vesicles in corneal epithelium due to corneal oedema.

♦ **What is Cup / Disc ratio in glaucoma?**

0.5-0.9 (less than 0.3 normally).

♦ **What are the diagnostic methods for glaucoma?**

(1) Provocative tests: to detect rise in IOP after stress condition:

1) In CAG: depends on mydriasis for one hour by:

1. Dark room test.
2. Mydriatic test (5% phenylephrine).

2) In OAG: depends on increasing aqueous formation for one hour by:

1. Water drinking test (one litre of water).
2. Priscoline test (10 mg pricscoline in 1 cc water subconjunctivally).

(2) Tonography: to calculate rate of aqueous outflow which is slower in glaucoma.

(3) Gonioscopy: for narrow angle or PAS.

(4) Diagnostic triad:

1) Tonometry.
2) Ophthalmoscopy.
3) Perimetry.

♦ **What are the main causes of marked diminution of vision in acute congestive glaucoma?**

(1) Corneal oedema.

(2) Retinal ischaemia.

♦ **What is to be done if both eyes are affected together with acute attacks of CAG?**

Both eyes should be operated at the same sitting.

♦ **Why enucleation is preferred in blind painful eye with high tension as absolute glaucoma?**

Because this eye may get a malignant tumour.

♦ **What are the causes of gradual painless diminution of vision in 1ry OAG?**

(1) Hypermetropia: Due to weakness of ciliary muscle from pressure on it and on its vessels and nerves.
(2) Retinal and optic atrophy: Due to mechanical pressure and ischaemia.
(3) Premature presbyopia: due to weakness of accommodation.
(4) Field defects: late.

♦ **Glaucoma inversus = any pupillary block glaucoma:**

1. Anterior dislocation of lens.
2. Iris bombé & ring synechia.
3. papillary block after operation:
    - air bubble
    - vitreous
    - Ring synechia.
    - IOL blocking pupil if iridectomy not done.
4. Intumescent cataract.

- Why called inversus ? as miotics will worsen the condition.
- TTT : pilocarpine + removal of the lens.

♦ **What is malignant glaucoma ?**

- It follow intra-ocular surgery particularly cataract & glaucoma
- Mechanism : misdirection of aqoues posteriorly → push the vitreous & lens >> pupillary block >>push iris forward >> angle block.

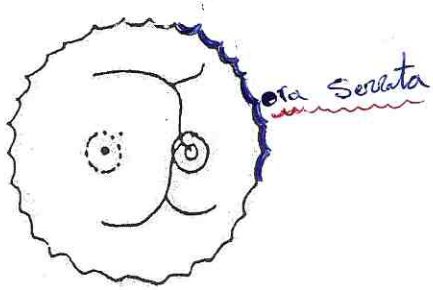
Retina

Gross Anatomy

It is the innermost layer of the eyeball. (مكانها)

- It starts at ora serrata & ends at optic disc. (حدودها)
- It is very thin & transparent showing the red colour of choroid. (خصائصها)
- It is sensitive only to light (translates photons to electrical impulses that reach the visual cortex and transformed into a visual sensation). (وظيفتها)

* **Anatomically**, the retina is divided into:



1- Central Retina: (Macula Lutea = 5.5 mm) the center of the macula is an avascular depression called "fovea centralis" (1.5 mm).

Function: (Mainly cones). Responsible for visual acuity, Color &day vision .

NB. *The macula is the area hugged by the temporal arches.*

2- Peripheral Retina: ends at the ora serrata.

Function: (Mainly rods) responsible for night vision and peripheral field.

so any defect in periphery of Retina → night blind

Microscopic anatomy

The retina is composed of 10 layers :

See atlas page (137)

→ makes Retina semi transparent

Cornea = 5 Layers

(1) Pigment epithelium (RPE):

- It is the outermost layer (in contact with Bruch's membrane of choroid).
- It is made of one layer of pigmented cubical cells.

(2) Photo-receptors (rods & cones): [Body]

The light sensitive layer (contains visual pigments).

→ Rhodopsin → Rods
→ Iodopsin → Cones

د.سعيد الدسوقي ليه ال Photoreceptors باصين لتحت (مقلوبين) ؟

عشان يمتص الضوء اكتر

(3) Outer limiting membrane: It is made of the outer ends of Muller's fibers.

(4) Outer Nuclear layer: it's made of nuclei of rods & cones.

(5) Outer plexiform layer [Henel layer in the macula arranged radially جدا مهم]:

Made up of the synapses between the outer & inner nuclear layers.

(synapses between photoreceptors & bipolar cells axons & processes of horizontal cells).

(6) Inner Nuclear layer:

It is made up of bipolar cells & cells of Muller's fibers

+ Amacrine & horizontal cells

(7) Inner pelxiform layer: the synapses between bipolar cells , Ganglion cells & Possesses of amacrine cells .

(8) Ganglion cell layer.

(9) Nerve fiber layer: it is made up of the axons of ganglion cells which form the optic n. (it relays in the LGB مسافة طويلة)

(10) Inner limiting membrane:

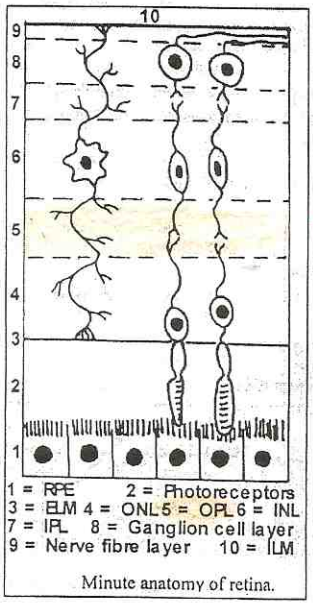
it's made of the inner ends of Muller's fibers & in contact with the vitreous.

N.B. Horizontal cells :

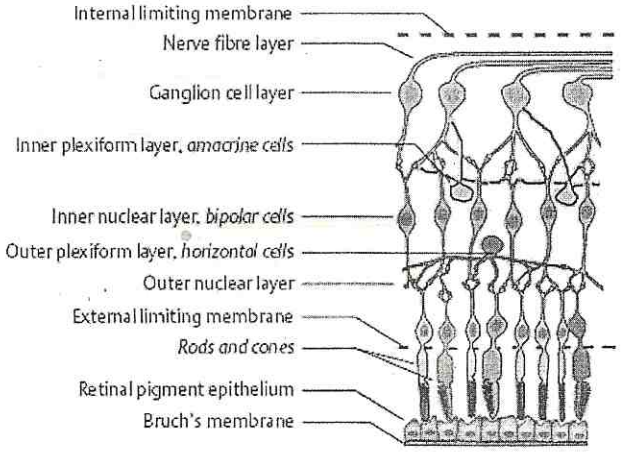
- Connect the photoreceptors with each other.
- Synapse with outer plexiform layer.
- 2 types : Type A → synapse with cones.
  Type B → synapse with rods.

N.B. Amacrine cells :

- Large with pyriform body.
- Connect the ganglion cells with each other.
- Synapse with plexiform layer.



Minute anatomy of retina.



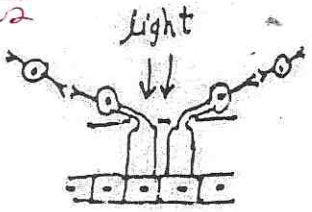
N.B: The layers 2-10 are considered the sensory retina

- the RPE is derived from the outer layer of the optic cup & the sensory retina is drived from the inner layer with a potential space between the sensory retina & the RPE (their separation causes RD.

((the adhesion between RPE & bruch's membrane is strong but the adhesion between the RPE & the sensory retina is weak → potential space))

The fovea: - Avascular depression in the center of the macula.

- It is composed of 3 layers only.
- Gives the best V.A. because:



(i) Very thin (3 layers), so light falls directly on cones

1- RPE: densly pigmented

2- photoreceptors: cones only

3- Outer limiting membrane

(the nerve cells & fibers of the inner layers displaced peripherally leaving only the photo receptors in the center).

(ii) Avascular (nutrition by diffusion from choroids).

(iii) High concentration of receptors (cones only . 100 000 cones at the fovea & foveola).

FUNCTION OF THE RPE:

(1) Nutrients & metabolites from & into the choroid

(Outer blood retinal barrier = Zonula occludence between RPE cells)

NB. Inner Blood retinal barrier = Zonula occludence between endothelium of BVs of the retina.

(2) Essential for Rhodopsin (Vit. A) & Iodopsin formation.

(3) Absorption of scattered light "perfect optical image".

(4) Phagocytosis of there is any tissue damage.

(5) Adhesiveness of the underlying sensory retina (prevents RD).

PHOTORECEPTORS:

Rods	Cones
1- Thin (easily affected in some diseases)	1- Thick
2- Contain Rhodopsin.	2- Contains Iodopsin.
3- Responsible for night vision. (Scotopic vision)	3- Responsible for day & colour vision. (Photopic vision)
4- Maximum concentration in perifoveal region160,000/mm2. **In the periphery rods only**	4- Maximum concentration in the Center (foveola 147,000/mm2). **In the foveola cones only**
5- Total number 120 milions.	5- Total number 6.5 milions.

Blood supply:

(1) Arteries:

- Inner 5 layers: from central retinal artery (from the ophthalmic artery).
- Outer 5 layers: avascular (nutrition by diffusion from the choroids capillaries).

The fovea: outer 5 layers (it's avascular nutrition by diffusion from the Choroidal capillaries).

The CRA : (End Artery with no collaterals):

- **Origin:** branch of ophthalmic artery arises form it close to optic foramen.
- **Course:**

1- Runs forwards below the optic n.

2- Pierces the optic nerve meninges at the inferomedial aspect of the optic n. (12 ml behind the globe)

3- Accompanied by the vein run in the center of the nerve and passes through the lamina cribrosa to enter the eye through the physiologic cup.

4- To appear inside the eye dividing into upper & lower branches.

N.B ***(In 20% of normal people: a separate artery originating from one of the posterior ciliary arteries(or Circle of Zinn) supplies the macula called cilio-retinal artery = Separate blood supply).***

Circle of Zinn:

- Circular anastomosis between Short ciliary arteries.
- Lies in the sclera close to optic n.

(2) Veins:

Central retinal vein CRV drains the inner 5 layers.

(to superior ophthalmic vein or directly to the cavernous sinus).

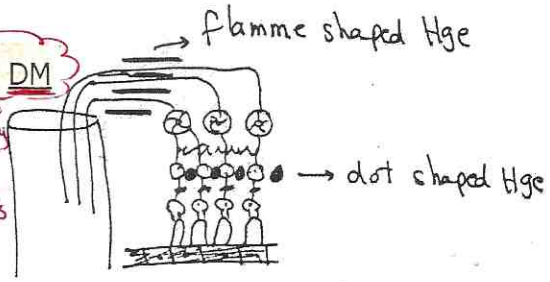
Retinal Capillaries:

superficial
deep 9th Layer

- **Superficial plexus** (in nerve fiber layer): Hge in this plexus takes the distribution of the nerve fiber layer → flame shaped hges.
  - Formed of large BVs which are affected by: hypertension – atherosclerosis- toxaemia of pregnancy.
- **Deep plexus** (in inner nuclear layer & outer plexiform): Hges. from this plexus → rounded (dot) hge.
  - Formed of small BVs which are affected by: DM

6th Layer 5th Layer

micro angiopathy
DM only effect small BVs



The Fundus Oculi

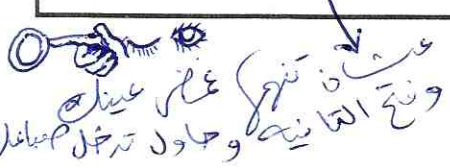
Includes: قاع العين

1- The retina
2- The optic Disc (the visible part of the optic nerve).
3- The choroids
4- The vitreous.

Examination of the fundus : By

1 direct ophthalmoscope
2 indirect "
3 slit Lamp + fundus lens
4 fluorescin Angiography

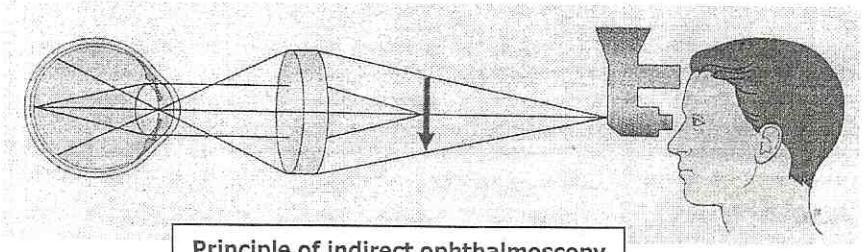
(1) Direct ophthalmoscope See atlas page (160)	(2) Indirect ophthalmoscope with a condensing lens +20 , +13 or +30 See atlas page (161)
Erect image.	Vertically & laterally inverted image.
Magnification: 15 X	Magnification : 3 X
Field smaller (details of lesion).	Field larger(general look& to see the periphery)
Uniocular. عين واحده	Binocular. عينين
No stereoposis	Stereopsis البعد الثالث: to see elevated lesions.



NB. Indirect ophthalmoscope with scleral indentation :مهم

See atlas page (160)

To enhance visualization of the peripheral retina anterior to the equator.



Principle of indirect ophthalmoscopy

(3) Slit lamp bio-microscopy: With a fundus lens :

* **Non contact : Volk 90 or 78 D** See atlas page (161)

* **Contact : Goldmann 3-mirror Contact lens.** See atlas page (133)

- It need methyl cellulose to be inserted as the curvatture of the lens in steeper than that of the cornea.

- **Advantages:** High magnification, and stereopsis.
- **Disadvantages:** Inverted image.

(4) Fluorescein Angiography: See atlas page (142) [FA]

- It is a useful investigation to evaluate the retinal & choroidal vasculature.

- Technique: 1- Pupil of the patient is dilated.

2- 5 cc of 10 % of Na Fl. Is injected IV.

3- The passage of the dye is visualized and photographed using a fundus camera.

4- Normal FA can be divided into 5 phases: (مهم جدا من المحاضره)

1) Choroidal phase (prearterial)

2) Arterial phase 3) Arterio-venous phase (capillary).

4) Venous phase 5) Late phase (elimination)

Areas of vascular occlusions → black= hypoflourescence

& new BVs → leaks → white = hyperflourescence

NB. Dark appearance of the fovea مهم:

1) FAZ

2) Xanthophylls pigment & dense RPE at the fovea block the back ground choroidal fluorescence

- Indications:

(1) Fundus angiography : as in

- Diabetic retinopathy
- Macular diseases - Papilloedema
- Retinal vein occlusion.

(2) Iris angiography: for iris neovascularization as in diabetes.

- Side effects : Nausea & vomiting , flushing, itching, death due to bronchospasm & anaphylactic shock.

NB. Why choroidal circulation details is not seen in ordinary FA ?

1- Rapid leakage of flourescein from choriocapillaries .

2- Melanin in RPE block the choroidal circulation

NB. How u can study the choroidal circulation? مهم الروند

Using indocyanine green (ICG) angiography.

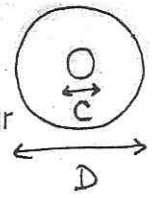
Appearance of Normal Fundus:

See atlas page (138)

سؤال لوحده

(1) Optic Disc:

- **Site**: Nasal to the macula
- **Shape**: Rounded or vertically oval with physiological cup in its center



(The cup from which the central vessels emerge is white in colour due to fewer BVs & here the lamina cribrosa is less obscured)

- Normally C/D = 0.3 or less.
- **Size**: 1.5 mm in diameter & can be used as the unit of measurement.
- **Colour**: Pale pink

- Pale due to nerve fibers . – Pink due to numerous capillaries.

- **Edge**: Sharply defined & slightly raised.

(2) Macula lutea:

It's the area hugged by the temporal arches.

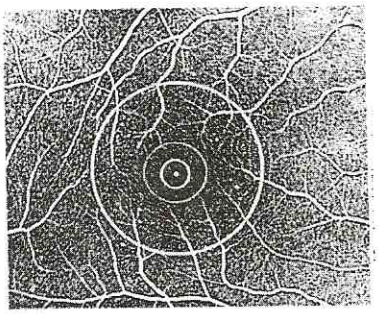
- Diameter: about 5.5 mm.
- Shape: circular or oval & ill-defined.
- Site: its center is 4.75 ml temporal & slightly (1mm) below the center of disc.

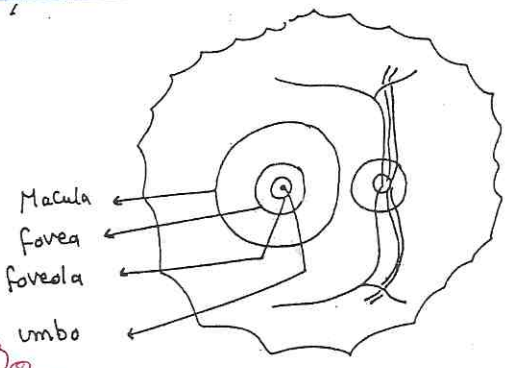
(or nearly 2 DD from the edge of the disc to the center of macula)

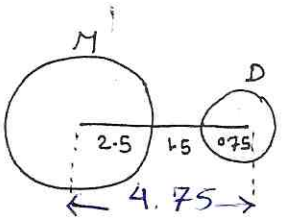
- Colour: Yellow-red due to high content of xanthophil (caroteniod) pigment.

NB. Hislologically the macula has more than one layer of ganglion cells مهم

- **Fovea centralis:**
  - is a small dark red depression in the center of macula (1 DD= 1.5 ml).
  - With oval light reflex due to hump around it (it act as concave mirror)
  - Causes of dark red colour of the fovea:
    1- Blood supply under it is very rich.
    2- Densly pigmented RPE
  - inside the fovea → foveola (0.35 ml in diameter): no rods at all
  - inside the foveola → Umbo = yellow foveolar reflex due to high concentration of cones .
  - Foveal avascular zone (FAZ) is 500 um in the center devoid of capillaries.







(3) Retinal vessels:

- The CRA emerges from cup & then divides into upper & lower branches. These branches subdivided into nasal & temporal branches.
- The veins follow the arteries and show Arterio-venous crossing (without constriction of the vein below the artery).
  - Arteries are smaller than veins (A/V = 3/4).
  - The vascular reflex : white line along the axis of Bvs, The vascular reflex of the artery is brighter (as the artery is more convex than the vein)
  - The artery is lighter in colour.
- They are transparent tubes, showing only the colour of blood through them.
- Pulsations: - Arterial pulsations → pathological
  - Venous pulsations → normal. مهم د. البرادعي

(4) General background:

The retina being nearly transparent (REP & BVs علشان)

It shows: Tigroid (Tesselated) fundus: خلفية حمراء منقطه بأسود → Tigroid Tiger جلد النمر

- Choroidal vessles (red areas)
- Choroidal pigment (dark areas)

(5) ora Serrata. **(6) Vitreous :** clear.

Diseases of Retina

Central Retinal Artery Occlusion (CRAO)

↳ Common on Left side

لو اتقفل end artery = no anastomosis فالـ Retina تموت

◊ Etiology:

1- Thrombosis: In : arteriosclerosis (Most Common Cause)

(in old age , DM ,hypertension & hyperlipidemia).

2- Embolism:

a- Cardiac: e.g. vegetations on the valves as in sub-acute B. Endocarditis, detached thrombus due to myocardial infarction .

b- Carotid embolus مهم :

1- Cholesterol : rarely causes significant obstruction.

2- Calcific: leads to permanent damage.

3- Fibrin-platelet : leads to amaurosis fugax.

3- Spasm: as in
- Hypertensive Encephalopathy in young age.
- Quinine poisoning
- Raynaud's disease & Migraine.

4- Arteritis: as in Giant cell arteritis (GCA), SLE , Poly arteritis nodosa & Behcet syndrome .

(GCA) → Anterior Ischemic optic neuropathy → CRAO } يعني GCA Causes 2 diseases

5- Very high IOP:

As - during RD surgery (large gas bubble = pneumatic retinopexy)

- ACG.

↳ Acute Congestive glucoma

◊ CL. Picture:

History: of recurrent attacks of Amaurosis fugax. مهم

Symptoms: sudden painless loss of vision "unilateral"

(CRA is an end artery; its occlusion → coagulative necrosis of the inner 5 layers of retina in minutes → complete loss of vision).

- Sometimes , if the macula is supplied from the cilio-retinal artery. (in 20% of population) → Central (tubular) 6/6 vision is preserved.

  See atlas page (143)

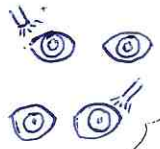
- Branch occlusion → sectorial field defect. See atlas page (142)

Signs:

(1) Vision: HM → NO p.L (20% → 6/6 tubular vision احياناً).

(2) Pupil: Afferent pupillary defect: APD

- Direct reaction → absent. ×
- Consensual → preserved. ✓



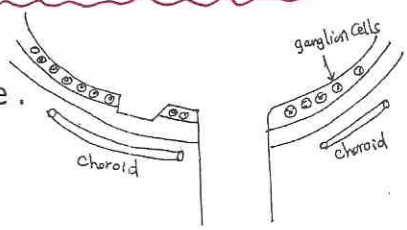
(3) Fundus: See atlas page (141)

(i) Early (within minutes):

- Arteries: attenuated (thread-like).
- Veins: little attenuation & stagnant blood column may be segmented (Cattle truck), as the musclosa is weak cant squeeze the blood inside the vein

  See atlas page (141)

- Retina: - White (cloudy) especially at the post pole .
  - Center (fovea)→ shows cherry red spot.
  - Emboli may be see



- **FA:** delayed retinal circulation_.

N.B: Cherry RS: appears 2-3 hours form the onset of occlusion & remains for 2-3 weeks

(i) Milky white: due to coagulative necrosis in ganglion cell layer.

(ii) Fovea contains no gang. Cells & takes nutrition from the choroids → remains red (showing choroids).

(II) Late (several weeks later):

* **Arteries:** sheathing
* **Retina:** the cherry red spot disappears (after 2-3 weeks) .
  (As the macrophages engulf the dead ganglion cells)
* **Discs:** shows consecutive optic atrophy (plae)

NB. Some cases of CRAO develop neovascularization of the iris, angle & retina & need PRP (Pan Retinal photocoagulation)

◊ D.D:

- **Other causes of cherry red spot**
  - Commotio retinae .
  - Amaurotic family idiocy.
  - Quinine poisoning → spasm of CRA.
  - Macular hole surrounded by R.D. - CRAO
- **Other causes of sudden loss of vision:**
  - Hysteria & malingerers - Trauma → Commotio retinea.
  - Amaurosis fugax (epilepsy of vision): it's intermittent spasm of CRA → momentary loss of vision.
- **Other causes of Consecutive optic atrophy:**
  - Retinitis pigmentosa
  - Amaurotic family idiocy.

◊ Treatment:

1- Emergency ttt

♥ Usually useless (if not immediate , after 30-90 min → irreversible damage to retina).

♥ Aim is to induce vasodilatation:

If spasm: relief the spasm.

If embolus : dislodge the embolus to a smaller peripheral branch أقل ف الخطورة

By ♦ **GENERAL :**

1- Inhalation or sublingual nitroglycerin.
2- Inhalation of carbogen(5% O_2 + 95% Co_2).يتنفس في كيس ← vasodilatation
3- I.V. Acetazolamide 500 mg (Diamox) to lower the IOP.
(hypotony)

4- I.V. mannitol 20%. → hypotony

5- I.V. Streptokinase 750 000 units (fibrinolytic).

♦ **LOCAL** 1- Paracentesis (leading to hypotony --- V.D.).

2- Ocular massage to lower the IOP.

3- Retrobulbar vasodilators (Acetyl choline – Priscol).

2- Systemic work up

After the emergency ttt u have to do systemic work up to search for the cause :

- Referral to cardiologist :

1- Cardio-vascular examination 2- Echo-cardiogram

3- Colour Doppler on the carotid.

- Screech for GCA (old age)

- Vasculitis (young age)

Central Retinal Vein Occlusion (Thrombosis) CRVO

♦ Etiology:

1- Inside the vein: due to increased blood viscosity as in:

- Blood diseases: polycythemia, leukaemia.
- Dehydration & contraceptive pills.

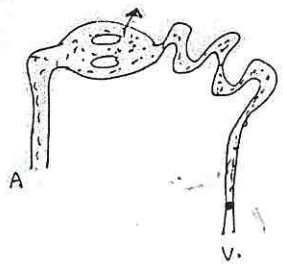
2- In the vein wall:

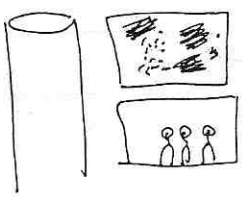
- Phlebitis: Behcet syndrome & Sarcoidosis.
- Rough intima: as in atherosclerosis (old) , DM , hypertension & hyperlpidemia

Most common cause

3- Outside the vein: due to pressure on vein by

- Orbital tumour
- increased IOP.
- Sclerosed artery. ⇒ Branch CRVO
- Orbital cellulitis





♦ Clinical picture:

Symptoms:

Rapid painless diminution of vision (6/60 → HM)

not sudden

due to hge & oedema & ischaemia of the inner 5 layers. (usually observed at the morning as the thrombus forms at night (due to venous stasis at night).

NB. branch vein occlusion → sectorial field defect. See atlas page (141)

Signs:

- **Pupil:** Dilated & Sluggish direct reaction (due to retinal oedema and hge).
  - Afferent pupillary defect in ischaemic type. مهم
- **Fundus:** shows **stormy sunset appearance.** الغروب العاصف See atlas page (140)

1- Veins: engorged & tortuous.

2- Opto-ciliary shunt: مهم

3- Retina: Show extensive Hge. (dots , blots & flame shaped), edema, cotton wool exudates +micro aneurysms.

4- Macula: shows oedema .

5- Disc: Hyperaemic, oedematous (papilloedema).

- **Flourescine angiography(FA): its done after 6 weeks** ليه؟
  - Delayed arterio-venous filling time.
  - Leakage of Fl. into the retina.
  - Areas of retinal ischaemia in ischemic type . to know if ischamic or not

N.B: S & S are 1- Mild in Non-ischaemic type 2- Marked in ischaemic type.

- **Investigations** :

(1) General : BP , ECG,CBC, Blood glucose level , lipid profile. & Chest x- ray.

(2) Local : OCT to detect macular edema: See atlas page (148)

- Hyporeflective spaces within the macula
- ↑ in macular thickening with loss of foveal depression.

- **Complications:**

1- Macular oedema (up to cystoid macular oedema) See atlas page (140)

→ Macular scar with severe affection of vision.

→ Coalescence of microcycstic spaces → Lamellar macular hole.

Q: Cause of macular hole: مهم

1) **Age related:** abnormal vitrofoveal attachement with tangentional traction.

TTT: vitrectomy

2) **High myopia**

3) **Blunt trauma** → commotion retinea

4) **CMO** → lamellar macular hole

2- Neo-vessels formation In ischaemic type:

Due to retinal ischaemia → release of vasogenic material

- **Site** : * Retina (NVD & NVEs). See atlas page (147)

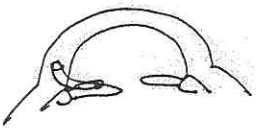
*Iris (Rubeosis Iridis = NVI). See atlas page (100,101)

- **These vessels may rupture**: leading to - hyphaema - vitreous Hge.

- ↑ in retinal hge .

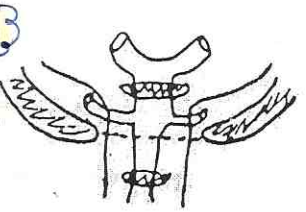
3- Secondary Glaucoma : due to :

- **Organized blood in angle** (haemorrhagic glaucoma).
- **Neo-vessels in angle** (neovascular glaucoma =NVG):
  - Occur in 20% of patients (that suffer from ischemic type)
  - Takes about 3 months to develope (100 day glaucoma)
  - Of bad prognosis (due to accompanying retinal ischeemia).
  - ttt : ext. fist. Op. + valve.
  - Other causes of NVG : DR (لسه جاي).



♦ Classification:

(I) Ischaemic. (2) Non-Ischaemic.



	Non-ischemic	Ischaemic = Cotton wool spots
Site of obstruction	Posterior to L. cirbrosa (good collaterals) e.g. Orbital tumours	Anterior to L. cirbrosa (no collaterals) e.g. Glaucoma.
Incidence	More common 80%	Less common 20%
Symptoms	Blurring of vision >6/60	Marked drop of vision<6/60
Hge, exudates	Mild. Hard.	Extensive. Soft (retinal infarction).
Pupil	Normal	APD اهم واحده

Retina

	Non - Ischemic	Ischaemic
ERG electro retenography	normal.	reduced.
Complications	Rare.	Common.
Prognosis	Good.	Poor.

♦ Fate:

- Resolution (in non-ischemic type) due to: good collaterals.
- Complication & permanent loss of vision (in ischemic type).

♦ Treatment:

(1) TTT of the cause : control hypertension & DM. مهم جدا

(2) Follow up till new vascularization or macular edema occur

(2) Laser Photo-coagulation (argon) PRP: See atlas page (148,149)

- Indication: NEW Vascularization
- Aim :
  - To destroy the peipheral hypoxia areas (→ anoxic) → not the macula
    no vasogenic material release.
  - Regression of the neo-vessels.
  - Improvement of circulation of the central area.
- Copmlications of PRP: - macular burn – constricted field – occlusuion of major BV

(3) Intravitreal مهم جدا: **1- Steroids** (Trimcinolone acetenoide) : (Kenacort)

2- Avastin : (anti VEGF)

- Indication: Macular edema.

(4) TTT Of Neovascular glaucoma:

- **Seeing eye**
  - Medical: Timolol + Diamox + Steroid locally (↓ inflammation caused by vasogenic material)
  - Laser ttt (PRP): to avoid regrowth of new BVs.
  - External fistulizing + Implant = seton (Ahmed valve or Molteno tube)
  - If failed do cyclocryotherapy.
- **Non – seeing (Blind Painful eye):**
  - Retrobulbar injection of alcohol.
  - Cyclocryotherapy
  - Enucleation.

NB. Pilocarpine (miotics) is contraindicated : →↑ congestion of the new BVs which are fragile→↑ hge .

Vascular sclerosis

The arteriosclerosis changes in the CRA & its branches :

• Pathology:

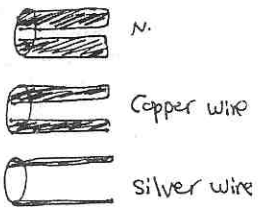
1- Atheromatous plaques (intimal hyalinization) deposited on the inner side of the arterial wall → detachement → Ulcer → fibrosis of the wall.

2- Endothelial hyperplasia.

3- Medial hypertrophy

(1) Changes in vascular light reflex :

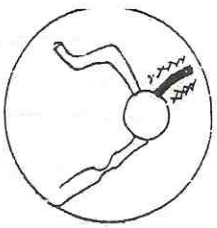
becomes wide due to thickening of the vessel wall → ↑ reflection of light.



- Copper wire .سلك النحاس
- Silver wire.؟؟؟؟؟ سلك الفضه

(2) Sheathing of vessels:

Fibrosis → white line along the sides of vessel wall .



See atlas page (150)

(3) Change in :

- **Course :** ↑ tortousity
- **Diameter :** irregular or narrowing (generalized or localized). → attenuation

(4) Arterio - Venous (A/V) crossing changes:

• **Concealment:** Obscuration or hidening of a part of the vein below the arterial wall (that becomes not transparent due to fibrosis)

• **Deflection (Salus sign):** deflection of course of vein by rigid artery (being enclosed in one sheath),

They become perpendicular to each other to minimize the area of contact:

- Vertical (U shaped) See atlas page (150)
- Lateral (S or Z- shaped).

- **Banking** (Bonnet sign): See atlas page (150)

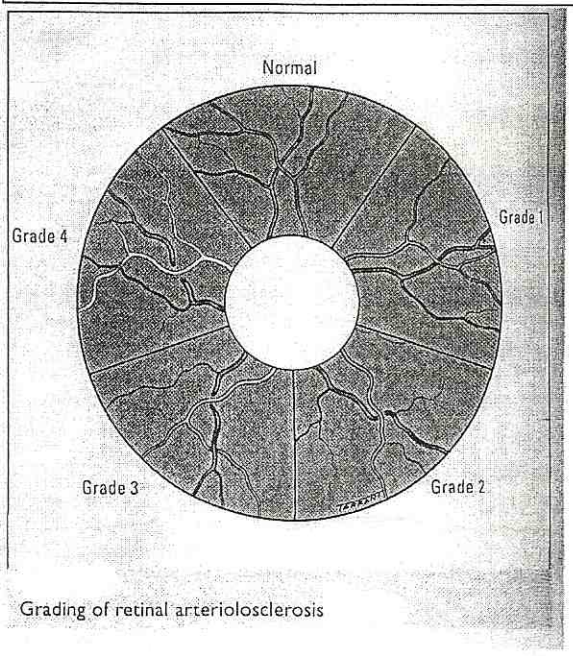
  Distension of vein distal to crossing

- **Gunn's sign (Nicking قطع):** Constriction & tapering of vein on either sides of Crossing.

◊ **Clinically :** Risk of CRAO , CRVO لو عاوز تكبر الاجابه اتكلم عليهم.

*** Grading of arteriosclerosis:** مهم د. البرادعي

- **Grade 1 :** Subtle broadening of the arteriolar light reflex, mild generalized arteriolar attenuation & vien concealment.
- **Grade 2 :** Obvious broadening of the arteriolar light reflex, with deflection of veins at the A/V crossing
- **Grade 3 :** Copper wiring of arterioles , Banking & tapering of viens on the sides of tha A/V crossing Gunn sign.
- **Grade 4 :** Silver wiring of arterioles associated with grade 3 changes



Grading of retinal arteriolosclerosis

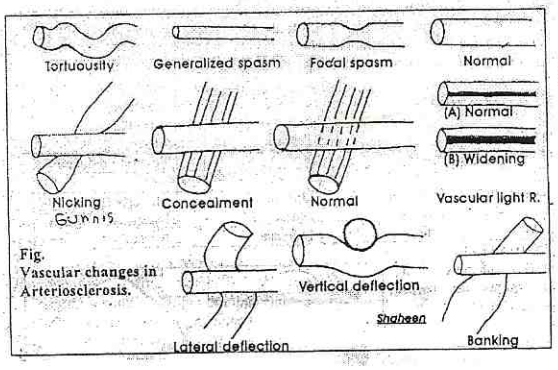


Fig.
Vascular changes in Arteriosclerosis.

Vascular Retinopathy

Definition:

It is bilateral retinal affection 2ry to systemic vascular Diseases:

Severe : - Renal (malignant) hypertension.
- Toxaemia of pregnancy . (eclampsia)

Mild : * DM.
* Benign hypertension & Atherosclerosis.

Clinical Picture:

Fundus examination shows ophthalmoscopic signs :

- **Changes in the Blood Vessels:** تعتمد علي سن العيان

- Vasoconstriction (sclerosis or spasm) :

1- Atherosclerotic changes → in old age → Atherosclerosis.

2- Spasm → in young age→ IN SEVERE CASES (malignant hypertension or Toxaemia of pregnancy).

3- micro aneurysm (only diabetic)

- **Retinal Hge:**

- **Superficial:** Flame shaped (in the nerve fiber layer). → [كله ماعدا DM]

- **Deep:** Dots (in the inner nuclear layer& outer plexiform)→DM as it affects the small BVs.

- **Vitroues , Subhyaloid, subretinal, hge under the RPE**

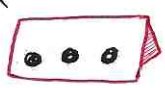
- **Retinal exudate:**

- Soft (Cotton wool) exudate: → IN SEVERE ISCHEMIC CASES

White, large, ill-defined , cloud like ,superficial patches

(Retinal infarction in the nerve fiber layer due to micro-vascular occlusion >> accumulation of axoplasm)

- Hard exudate: → IN CASE WITH LEAKAGE as Diabetic R.: (not only reason)



Yellowish white, small ,rounded ,deep spots with well-defined edges

(its due to affection of inner blood retinal barrier→ ↑permiability ↑permeability→lipoprotiens collects in the inner nuclear layer & lipid filled macrophages).

- **Macular oedema & macular scar & papilloedema:** → IN SEVERE CASES.

oedema on disc

Symptoms:

1 - Mild cases : Asymptomatic.

distortion of object due to distortion of Retina

2- Severe cases : - blurring of vision & Metamorphopsia (macular edema)

- Rapid↓ of vision (due to exudative RD).

Signs: Fundus picture:

Mild → Severe

	1- Benign hypertension & Athersclerosis	2- Malignant & Renal Hypertension	3- Toxaemia of pregnancy (eclampsia)
Vessels	Sclerotic changes	Sclerotic or spasm	Spasm → ischaemia
Hge	Flame-shaped	Flame-shaped	Flame-shaped → because only dot in D.M.
Exudates	Hard(rounded)	Soft	Soft
Macular Star (fan): As exudates takes the distribution of n. fibers.	Absent	Present	Present
Retinal oedema	Absent or mild	Present (in the retina & in the disc = Papilloedema)	Present (retina & disc) & up to bilateral exudative RD.
Significance	Pt. is liable to cardiac & cerebral accidents, Medical care is in need	Pt rarely lived more than 2ys. death is usually due to renal failure.	Termination of pregnancy must be done to save life & vision of mother

Hypertensive retinopathy

See atlas page (149,150)

Systemic hypertension is diagnosed on several consecutive measurements of 140/90 or more.

* **Pathogenesis:**

- Chronic hypertension leads to damage of vessel wall with breakdown of blood retinal barrier & vascular leakage (ماء +دم+ دهن)
- Chronic hypertension leads to Arteriosclerotic changes.
- Elevated acute BP leads to vasoconstriction with generalized & focal narrowing of arterioles.((the degree of narrowing depends on the pre-existing

involutional sclerosis , in older pt. , rigidity of arterioles due to involutional sclerosis prevent the same degree of narrowing seen in young pt.))

- VC → leads to closure of retinal capillaries→ cotton wool spots & ischaemia
- Malignant hypertension (diastole more than 140), I may also lead to papilloedema & exudative RD.

* **Clinical picture:**

♦ **Symptoms :**

- May be asymptomatic.
- Blurred vision.
- Episodes of temporary visual loss.

♦ **Fundus picture :**

- **Arteriolar narrowing :**
  1- Generalized & Focal .
  2- Severe hypertension may lead to obstruction of pre-capillary arterioles & development of cotton wool spots.
- **Vascualr leakage:**
  1- Flame-shaped hge
  2- Retinal oedema
  3- Hard exudates. (if in the macula → macular star) See atlas page (149,150)
  4- Papilloedema & exudative RD in case of malignant(accelerated) hypertension.
- **Arteriosclerosis :** in chronic hyper tension for many years. اكتب

* **Grades of Hypertensive Retinopathy:** See atlas page (149)

Grade 1: Generalized arteriolar narrowing.

Grade 2: Generalized and focal arteriolar narrowing

Grade 3: As grade 2+ Retinal flamme hge + retinal exudates + Cotton wool spots. + all sclerotic changes.

Grade 4: Severe grade 3 + papilloedema + macular star + Exudative RD

NB. Arteriosclerosis & hypertensive changes are commonly associated , pure form of hypertensive retinopathy is commonly seen in young pt. e.g Toxaemia of pregnancy , malignant hypertension in renal failure

*** Ocular associations & complications of hypertension:**

- Retinal vein occlusion.
- Retinal artery occlusion .
- Retinal artery macro-aneurysm
- Ocular motor nerve palsy.

***Treatment:** - Control hypertension
- Weight control . – Exercise. - Sodium control.

Retinopathy of prematurity(ROP) د حموده غرابه

♦ **Pathogenesis:**

ROP is a proliferative retinopathy affects pre-term infants <30 weaks or low birth weight < 1500gm who exposed to high O2 concentration leads to damage of incompletely vascularized retina = temporal periphery → produce VEGF.

(Retinal Bvs reach the nasal periphery at the 8th month of gestation & reach the temporal periphery one month after delivery).

♦ **Zones :**

zone 1 ,2 & 3

Zone 1 is more central & more dangerous.

♦ **Staging :**

- Stage 1 : Demarcation line separate vascular from avascualr retina.
- Stage 2 : Elevated ridge .
- Stage 3 : Ridge + Extra retinal fibrovascular proliferation extend from the ridge into the vitreous → retinal & vitreous hge.
- Stage 4 : Subtotal RD.
- Stage 5 : Total RD .

♦ **Extent :** number of clock hour involved.

♦ **Other considerations :**

Plus disease : - Gross vascular engorgement of the iris & failure of the pupil to dilate
- Engorged veins & tortuous arteries in the post pole.
- Vitreous haze.
- Increase of the vitreous hge.

when these signs are present a plus sign is added to the stage number & indicates tendency to progression of the diseases.

Threshould disease : eye that have 5 contiguous clock hours or 8 non-contagious clock hours of extra-retinal neovascularization (stage 3) in zone 1 or 2 associated with plus disease

Q: Threshould disease؟ لو وجدته تعمل ايه

♦ **Fate :** - 80% regress spontaneous

- 20% go to cicatricial diseases :

→ dragging of the macula & disc

→ retrolental fibroplasia → leuckocoria , RD & ACG.

♦ **Management :**

- Screening : Who to screen ??

babies born ≤ 31 weeks or weighting ≤1500 gm

Should be screened for ROP.

How to screen ??

فحص كل أسبوعين من أول الاسبوع السادس بعد الولادة,

Until Complete retinal vascularization الحمد لله

- **TTT:**
  - Stage 1,2 → no ttt.
  - Stage 3 → laser photocoagulation or cyclocryotherapy for the avascular retina if Threshould disease present.
  - Stage 4,5 → vitrectomy for tractional RD.

Diabetic retinopathy (DR)

◊ Definition:

It's a micro-angiopathy affecting the retinal arterioles , capillaries & venules. Always bilateral but asymmetrical.

◊ Etiology:

Unknown (DM more than 10 yrs , so DR is related to duration of DM not related to severity of DM , so DR is commom in insulin dependent = Juvenile DM = type 1 DM).

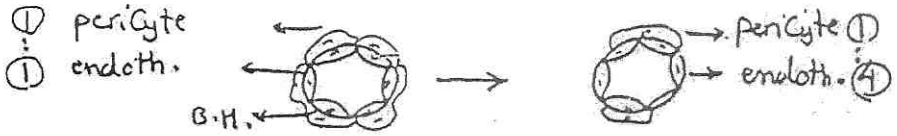
NB. DR is the 3rd cause of blindness in Egypt.

◊ Risk factors: - Young age & long duration.
- Poor control of DM.
- Coexisting hypertension.
- Obesity & Hyperlipidemia.
- nephropathy & Pregnancy.

◊ Pathogenesis:

- ***Micro - vascular leakage:***

1- Due to : loss of pericytes due to sorbitol → disturbance of blood - retinal barrier.



2- Resulting in : - Edema (↑ capillary permeability)
- Hge (rupture capillaries)
- exudate
- Micro –aneurysm (localized capillary distension)

ميه+دم +دهن

⇒ **SIMPLE DR**

NB. Micro-aneurysms present in : - DR → central .
& - CRVO → peripheral.

- ***Micro - vascular occlusion***

A) Due to

1- Thickening of basement memb. due to glycogen deposition→ ↓ diffusion → retinal ischemia

2- Aggregation & stickiness of platelets → microthrombus.

3- Endothelial cell damage & proliferation.(by diabetic metabolites).

((1,2,3 Organic occlusion)).

4- RBCs changes - Glycosylated Hemoglobin → ↓ O_2 transport

- Lack of deformability (elasticity) of RBCs.

((Functional occlusion)).

5- ILM thickening → ↓ diffusion of O_2 from vitreous to retina .

B) Resulting in:

Retinal ischemia which release chemical (vasogenic) factor which stimulate new vessels formation. ⇒ PROLIFERATIVE DR

◊ Clinical picture:

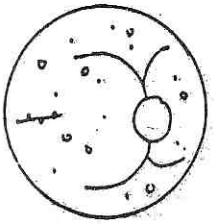
[Symptoms] - Asymptomatic.

- Gradual diminution of vision.
- Floaters. - Acute loss of vision.

[Signs = Fundus picture]

1- Non -Proliferative (Simple or Background) DR:

See atlas page (145,146)



ميه+دم +دهن

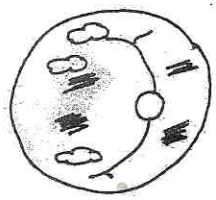
- Retinal Hge: dot hge (Deep ,rounded hge), due to affection of deep plexus.
- Retinal exudate: Deep (hard) exudates arranged in clumps or rings called circinate surround leaking microaneurym.
- Retinal edema.
- Retinal vessels: Micro – aneurysms, (seen by Fl. Angiography as hyperflourescence spots)

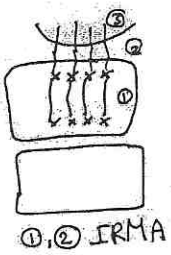
Mild NPDR : ميه+دم +دهن + 1 Micro – aneurysms in < 4 quadrants.

Modeate NPDR : ميه+دم +دهن in the 4 quadrants.

Severe NPDR = Pre-proliferative DR

2- Pre-proliferative DR:





- Retinal Hge.: blot hge = hgic retinal infarction
- Retinal exudates: Soft (cotton wool spots)
- Retinal vessels:

IRMA (intra retinal micro-vascular abnormalities) لسه لم تدخل الvitrous

+ Venous beading + looping. ايه سببهم؟؟ See atlas page (146)

3- Proliferative DR:

See atlas page (146,147)

Pre-proliferative + new vessels at :

1- The Disc (or within one DD around the disc)→ NVD See atlas page (147)
((dangerous ؟؟ليه)).

2- The Retina → NVE (elsewhere). See atlas page (147)

4-Advanced Diabetic Eye

(Complications of DR) :

- Vitreous hge & sub-hyaloid = pre-retinal: bleeding from the neo-vessels
  → may organized → vitreous fibrosis & epi-retinal membranes.

- Tractional retinal detachment: Due to pull by the vitreous fibrosis (جار السوء)
  & contraction of the epi-retinla membranes. See atlas page (147)

NB. Formation of epi-retinalmembranes? Breaks in the internal limiting membrane → proliferation of glial tissue & formation of membrane between the retina & vitroues. - This membrane can be detected by OCT
- Complications: CME
- TTT: vitrectomy + removal of the membrane (e.g. delamination)

- Rubeosis Iridis that may lead to neo-vascular glaucoma. See atlas page (100)

N.B. Regular fundus examination of diabetic pts is essential every 6 months.

NB. What are the types of glaucoma occurs with DR ? خمسه اكتبهم معايا

5- Diabetic Maculopathy: تأتي مع اي مرحله

Either:

A) Focal edema: from microaneurysm.

B) Diffuse edema (لما تزيد>>CME):

FA → flower petals. See atlas page (140)

due to accumulations of the dye within the microcystic spaces in the radially arranged henel layer of the macula.

Also can be detected using OCT: هتلاقي ايه See atlas page (148)

CME may lead to → foveal cyst → rupture →lamellar macular hole.

NB. Clinically significant macular edema:

1- Retinal edema within 500 micron of the center of the fovea.

2- Hard exudates within 500 micron of the center of the fovea + Retinal thickening

2- Retinal edema 1 DD (1500 μ) or larger any part of which is within one DD of the center .

- This is the most common cause of diminution of vision in diabetic pts.

NB. If there is CSME → Do OCT→Vitromacular traction → PPV

No Vitromacular traction → Do FA → LASER

C) Ischemic maculopathy : due to occlusion of macular BVs.(NO TTT)

FA → Large irregular FAZ

◊ **Investigation:** * **FA (Flourescine angiography):**

- CSME: focal or diffuse or ischemic
- To detect leaking micro-aneurysms and ischemia.
- To differentiate IRMA from new BVs (IRMA no leaking)

* **OCT** : detect foveal thickening , sensory detachment . vitro-macular traction

* **Systemic :** Glycosylated HB , Renal function , lipid priofile

◊ **DD:** From other causes of retinal he & exudates

1- **macular drusen**
2- **Hypertensive retinopathy**
3- **Old CRVO**
4- **Radiational retinopathy**
5- **Retinal artery macro-aneurysm**

◊ Treatment:

1- Systemic work up

- Control DM (يؤجل المشاكل ولا يلغيها) & hypertension .
- Controle other risk factors like hyperlipidemia & nephropathy.

2- Back ground DR : follow up.

3- Argon Laser ttt:

- **Indications :**

- In Pre. and proliferative DR & robeiosis iridis → do **PRP** See atlas page (148,149)

- In diabetic macular edema do - **Focal ttt** (in focal edema).
  - **Grid ttt** (in diffuse edema).

- **Aim :** as CRVO.
- **complications:** 1- Field defect , decrease night vision (يفقد رخصه السواقه)
  2- Rupture of large BV >> vitreous hge
  3- Iris burn 4- Foveal burn 5- induce CNV.

4- Intravitreal Triamcinolone acetonide injection مهم:

- Now it replace grid laser espeacilly if the thickness of macula is more than 400 micron by OCT (when the thickness decrease again do laser to prevent recurrence)

5- Vitrectomy + laser endophotocoagulation :

- Indications:
  1- Persistent vitreous hge for 1 month
  2- Tractional RD if threaten the macula or associated with tear.
  3- Persistent macular edema unexplained or from Taut post. Hyaloid
  4- Premacular hge : if dense & persistent
  may lead to macular detachement.

◊ Prognosis of vision:

1- Background: GOOD except if the macula is affected.

2- Proliferative : POOR

◊ Causes of blindness in Egypt :

1- PC 2- OAG 3- DR

Ocular manifestations of DM

كل كتاب الرمد صح

(1) Lid: Blepharitis, recurrent styes & Xanthelasma .

(2) Conjunctiva: Conjunctivitis.

(3) Cornea: Keratitis (ulcer). **(4) AC:** Hyphaema (From rubeosis iridis)

(5) Iris: Rubeosis Iridis. **(6) Lens:** Complicated Cataract(types ?).

(7) Rapid changes in refraction: شفوي

a) Hyperglycemia → Index Myopia.

b) Hypoglycemia → Index Hypermetropia.

(8) Vitreous: Vitreous hemorrhage (From new vessels in DR)

(9) Retina: - DR.

- CRVO (→ Retinal hge).
- Lipaemia retinalis: due to hyper-lipidaemia (showing Pale fundus & milky vessels).

See atlas page (155)

(10) IOP : May : - ↑ as in Neovascular , hemorrhagic glaucoma , ghost cell Glaucoma due to vitroues hge , pigmentry glaucoma & OAG مهم جداً

- ↓ "Hypotony " in diabetic coma due to dehydration.

(11) Optic nerve : Optic neuritis.

(12) Orbit: Orbital cellulitis.

(13) Extra-ocular muscles: Paralytic squint (especially the Lat. Rectus).

(14) Post-operative complications: Hge, infection, delayed wound healing.

Retinal detachment (RD)

★ Definition:

It's a condition in which the retina is separated into 2 layers:

1- Retinal pigment epith. (R.P.E.).

2- Sensory retina (as there is an embryological potential space between these 2 layers)

So, the term retinal separation is more accurate (as the retina is separated not detached).

NB. Factors lead to fixation of the retina: د حموده غرابه

1- Apposition force : anatomy of the retina suites the inner surface of the globe.
(الشبكية ملائمه لمكانها)

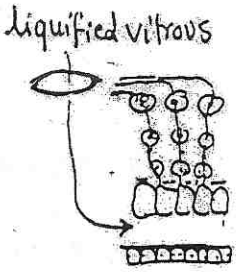
2- Adsorptive force: adhesive material between photoreceptors & RPE.

3- Absorptive force: pump function of the RPE.

★ Etiology of RD:

(A)Iry (Simple - Rhegmatogenous)

See atlas page (151)



* **Def.:** It's due to formation of a retinal tear, which allow the liquefied vitreous to enter between the retinal layers causing retinal separation.

* **What detaches the retina:** 1- Retina break: may be
  - Vitrogenic → due to PVD
  - Retinogenic → due to atrophy of the retina

2- Traction on the break.

3- Moving fluid(shaking) مهم

* **Risk Factors:** of Rhegmatogenous RD :

(a) Chorio-retinal degeneration as in high myopia.

(b) Blunt trauma.

(c) Aphakia.

(d) Family history of RD or history of RD in the fellow eye.

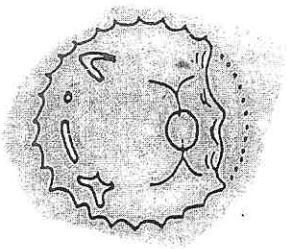
(e) PVD . الزبادي في الثلاجة

(f) Chorio-retinitis: → toxins → necrosis & tear.
→ liquefaction of vitreous
→ Vitreo-retinal adhesions.

* **Incidence**
  - Patient: > 40 why ?
  - Sex : > ♂ why ?
  - Refraction : > Myopia
  - Bilateral in > 10% of cases

• N.B.

Shape of retinal tears:



(1) Horse-shoe tear (the most common). See atlas page (151)

(2) Retinal hole. See atlas page (151)

(3) Arrow head tear.

(4) Operculated tear : the flap is completely torn away from the retina.

(5) Irregular or linear.

(6) Dis-insertion (retinal dialysis): Separation of the retina from its root at the ora serrata.

(7) Giant tear: 90 degree or more of globe circumference (more than 3 o'clock).

Site: - Oral. - Post-oral. - Equatorial. - Post-equatorial - Macular

(B) 2ry Retinal detachment

Def.: It is pushing or pulling of the retina by intraocular disease with no retinal tear.

1- Tractional RD:

* In which retina is pulled by vitreous fibrosis.
* As in:
  - Cyclitic membrane, organized vitreous hge,
  - Proliferative diabetic retinopathy (PDR).
  - Retinopathy of prematurity (ROP).
  - Penetrating post. segment trauma: with vitreous loss & vitreous hge.

DD from Rheg. & Tractional RD:

1- No photopsia & Floatera as the traction occurs gradual.
2- No retinal tear.
3- Restricted mobility of the retina.
4- No shift of sub-retinal fluid.

* Complications : traction may leads to retinal tear (combined Tractional & Rheg.)

2- Exudative RD:

In which the retina is pushed by fluid derived from the choroid which gain access to the subr-etinal space through damaged RPE, as in:

- Choroiditis as in "Harada disease".
- Posterior scleritis .
- Neoplasm (M.M. of choroids or secondries).
- Coat's disease.
- Sub-retinal neovascularization & Hge as in ARMD .
- Systemic causes : toxaemia of pregnancy , malignant hypertension.

D.D. from Rheg. & exudative RD:

1- Photopsia is absent as there is no vitreous traction but floaters are present due to accompanying vitritis.

2- No tear.

3- The detached retina is very mobile with characteristic shifting of SRF to the most dependent position by gravity.

TTT of 2 ry RD:

Is the ttt of the cause

1- Tractional: Vitrectomy.

2- Exudative:

- Inflammatory (choroiditis & post.scleritis)→ give cortisone.
- Malignant → Enucleation.

★ **Clinical Picture:** of rhegmatogenous RD

* **<u>Symptoms:</u> May be asymptomatic for long time**

When ??

1- Early: The retina still has some nutrition.

- Flashes of light (photopsia): Due to mechanical irritation of rods & cones by vitreous traction .

- ***Floaters: (Musca volitans):*** Due to Vitreous Degeneration → opacities , minute hge from the tear into the vitreous & P.E. migration
- ***Metamorphopsia , micropsia, macropsia*** (Distortion of objects).

2- late: Death of photoreceptors (choroid الـ عن بعدت لانها)

- ***Field defect (black curtain):ستارة سوداء***

Its site is useful in predicting the site of the retinal break (in the opposite quadrant).

- **Failure of vision(HM or PL vision):** (painless & rapid) due to foveal involvement (foveal detachment).

*** Signs:**

1- R.R: Grey. (as the retina is thick, wavy , opaque , grayish) .

2- Tonometry: Soft due to : - absorption of the SRF by the choroidal vessels.
- extension of inflammation to CB.

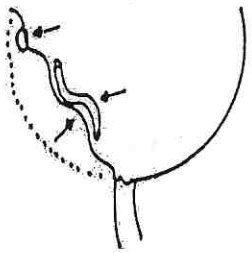
3- Pupil : Afferent pupillary defect in total RD.

4- Slit lamp: may show:

i- Flare (AC & vitreous) if Iridocyclitis occurs.
(as the detached retina releases lactic acid → iritis)

ii- Tobacco dust (pigment cells in the retro-lental space & ant .vitreous , released from the detached RPE)

5- Ophthalmoscope : *(full dilated pupil is necessary)* Fundus examination shows :



- Retina→ Thick, wavy , grayish, convex & undulates freely with eye movements, in fresh RD (tremulous retina)
- Retinal vessels→ wavy (follow the retina) & darker ?? لو شاطر قول لبه
- Retinal tear:appears red as - it show the choroid.
- contrast with gary area of RD)
- Retinal hge → may be seen (from the tear).

6- Ultrasound **:** detached retina are attached to the optic disc See atlas page (154)

7- OCT : also can detect RD See atlas page (148)

*** Longstanding (Old) RD:** Shows

- Retinal thinning due to atrophy.
- Intra-retinal cysts.

- High water marks: demarcation lines caused by proliferation of RPE at the junction of attached and detached retina.
- Robisis iridis

* **Proliferative Vitreo-retinopathy (PVR): due to** proliferation of membranes:
  - on the inner retinal surface (epi-retinal membrane).
  - on the outer retinal surface (sub-retinal membrane).

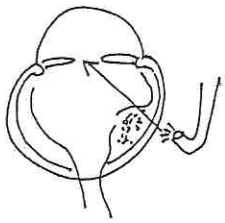
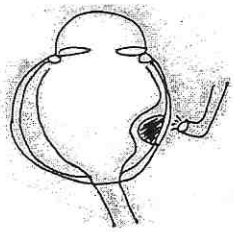
★ Complications:

1- Iridocyclitis → Complicated cataract.
2- Total RD due to spread of tear.
3- Retinal degeneration → permanent visual loss.
4- Rubeosis iridis.
5- PVR proliferative vitreoretinopathy : RPE يغزو الشبكيه
6- Atrophia bulbi: in neglected cases due to :
   - Absorption of the SRF by the choroidal vessels.
   - Extension of inflammation to CB.

★ DD:

1- Simple from malignant RD:

	Simple (1ry)	**Malignant RD (due to choroidal malignant melanoma)**
Incidence:	More common	Rare
Age:	Any	Above 40
History :	Trauma (may be)	–ve
Refraction:	High Myopia (usually)	Any refraction
IOP:	Soft	May be ↑ (2ry glaucoma)
Fundus: ▪ Tear: ▪ Retina: ▪ B.V:	 ▪ Present ▪ Wavy, tremulous ▪ Wavy	 ▪ Absent ▪ Stretched (tent like),not moving. ▪ Abnormal vascularisation Of tumor.
Investigations:	Wavy	Abnormal Vascularization

▪ Ultra-sonography: ▪ Trans-illumination: ▪ Radioactive P32 uptake	▪ No mass (detached retina attached to disc). ▪ Translucent. ▪ Normal uptake. 	▪ Mass is present ▪ Transopaque. ▪ ↑ Uptake. 

2- RD from other causes of rapid drop of vision:

1- Corneal ulcer. 2- Retinopathy.

3- Iritis 4- Optic neuritis. 5- RD

NB. What is retinoschisis:

It's is splitting of the neuro-sensory layer due to affection of Muller cells.

- Senile
- X-linked

★ Treatment:

• Prophylaxis:

Retinal tear → Sealing لحام مانع للتسرب See atlas page (151,152)

- Indications:

a) Patient: - Myopic - Aphakic (vitreous herniation → tear يوسع).

- R.D in the others eye.

b) Break: - Large - Superior → spread rapidly by gravity.

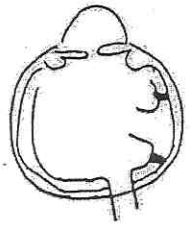
- Technique of sealing:

Most breaks are adequately treated by:

* Argon laser:(trans-pupillary):

especially if the media is clear & the tear is central .

* Cryotherapy: (trans- scleral) especially if the media is Hazy or peripheral tear).



- **Curative:**

a) The pt. is atropinized and examined:

- To determine extent of RD.
- To detect number & site of breaks .

(Ultrasonography may help in opaque media e.g. vitreous hge).

b) The pt. rest in bed (to avoid shaking movement) with tear in the most dependent part .

c) Technique:

I- Sealing of the break:

by - Cryotherapy. - Laser. x

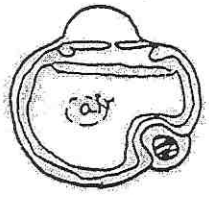
which will induce sterile chorio-retinitis → that heals by chorio-retinal scar → adhesion between retina & choroid that prevents leakage of fluid under the retina → prevent spread.

II: Approximation of the retina & choroid (reposition of the retina):

- **Evacuate the Subretinal fluid (SRF)**: needle is inserted in the most dependent part.
- **Scleral buckling: تحزيم العين** See atlas page (154)

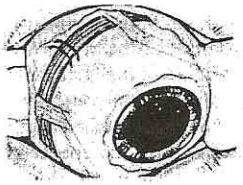
pushing the sclera by:

- Silicone implant : it is sutured on the sclera over the tear (to do invagination ← implant ال حجم من أكبر الغرز)



or - Silicon bandage: that encircles the eye ball.

both help in approximation of retina & choroid and Reduction of vitreo- retinal traction (External indentation of Sclera).



Encirciling band.

* Complications of Scleral buckling :

- ↑ Axial length of the globe → more myopic.
- Scleral necrosis.
- Glaucoma - Metamorphopsia.
- Tight blukle → signs of ischemia (aqoues flare مهم)

- **Intra-vitreal injection of air or expandable gases like sulpher hexaflouride** يتمدد مرتين.**(Pneumatic retinopexey)**

*Complications of silicon oil : - Inverted hypopyon in AC. See atlas page (64,94)
- Cataractogenic .
- Close the angle → secondary glaucoma.
- Uveitis.

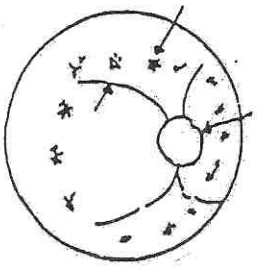
III: Pars Plana Vitrectomy+ Intra-vitreal injection of silicon oil (to relif retina from vitreo-retinal traction): + endo laser :in the following cases:

1- RD associated with PVR (vitreous traction).
2- To clear vitreous Opacities e.g. Hge, to visualize retinal breaks.
3- Posterior retinal breaks.
4- Giant retinal breaks.

NB. Look to the follow eye why ?مهم جدا المحاضره

Degeneration of the Retina

(I) Retinitis Pigmentosa



• Definition:

It is heredo-familial, bilateral, progressive , pigmentry retinal degeneration of unknown etiology.

• Etiology:

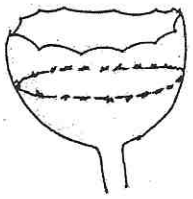
Unknown, may be:

1- Abiotrophy (نقص تغذية): ischemia & Vit A ↓.
2- Phototoxicity.
3- Hereditary: mode of inheritance may be ,
- Autosomal dominant - Autosomal recessive. - X-linked **(so common in boys)**

• Pathology:

- Degeneration affects the photoreceptors (particularly the rods) & RPE.

Retina



- It starts at equatorial region (less blood supply) & progress centrally & peripherally leading to complete blindness at middle age (40 سنة).
- RPE proliferate & migrate inwards towards the inner retinal layers working as

  macrophages to engulf the dead rods → (bone corpuscles appearance).

• Clinical Picture:

◊ **Symptoms:**

- Night blindness (defective dark adaptation): due to affection of rods.
- Progressive visual field contraction.
- Finally, Complete loss of vision.

◊ ***Signs:***

1- Fundus Picture:

- Retina: Spider(Bone corpuscle) like pigmented spots at equator, then spread ant. & post. See atlas page (152)
- Vessels: Markedly attenuated.
- Disc: Consecutive optic atrophy (Waxy or pale disc).

2- Field changes:

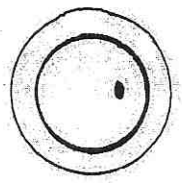
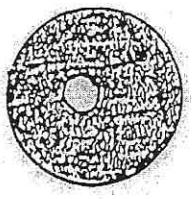
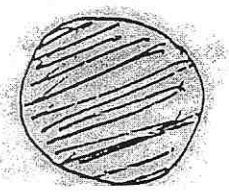
- Early: ring (annular) scotoma : due to equatorial pigmentry degeneration.

  DD from ring Scotoma of OAG مهمة جداً :

  1- Peripheral not continuous with the blind spot ,

  2- Has no nasal step.

- Late: - tubular field - Complete loss of vision.

3- Investigations

- ERG: is markedly diminished especially ***Scotopic ERG*** ,it's affected early.

  (Photopic REG is affected later)
- EOG : subnormal.

N.B

Common associations (rather than complications) with **R. Pigmentosa:**

1- Local : i) OAG ii) Myopia iii) Keratoconus

vi) PVD v) Posterior sub-capsular complicated cataract

K O M P P

2- General: Syndromes as :

* Bardet- Biedel syndrome: - Obesity & Hypogonadism (due to hypopituitarism)
  - Mental Retardation - Polydactyly+ RP
* Refsum syndrome: - Deafness - Ataxia - polyneuropathy - Cardiomyopathy.

NB. Atypical Retinitis pigmentosa:

1- RP sin pigmento (without pigment spots).

2- Retinitis punctata albescence (Show white spots). See atlas page (153)

3- Unilateral RP.

4- Sectorial RP. See atlas page (152)

5- Pericentric RP. See atlas page (153)

• Prognosis:

Loss of vision may be due to:

1- Complicated cataract.

2- Optic atrophy (consecutive).

3- Macular affection

4- Myopia 5- OAG.

• D.D - From other causes of

1- Night blindness.

2- Ring scotoma & tubular field.

3- Optic atrophy. الجدول

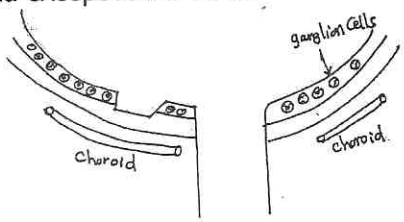
• TTT:

* Useless: 1- Vitamin A. 2- Vasodilators. 3- placental extract.
* Low vision aid.
* Genetic counseling .

(II) Amaurotic family Idiocy

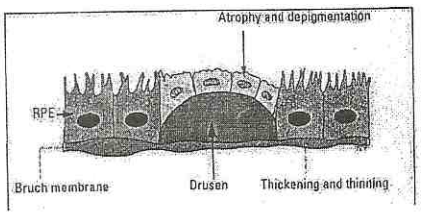
TAY-SACK'S DISEASES

- It is lipoid degeneration of the ganglion cells of the brain (idiocy & general paralysis) and retina (blind & white retina).
- It is common in Jewish children (+ve consanguinity).
- **ONSET** : Appears in the first year of life and death usually occurs within 1-2 years.
- **Fundus** : 1 - Cherry red spot (degeneration of all the retina except fovea as no ganglion cells in the fovea).
  2 - Consecutive optic atrophy.



(III) AGE REALTED MACULAR DEGENERATION (ARMD) مهم جداً

* **Definition :** It's a macular disease leads to severe affection of the central vision in old age above 50 yrs.
* **Laterality :** bilateral & symmetrical.لازم



* **C/P :**

(1) Age related maculopathy = drusen :

Small, discrete, yellow white, slightly elevated bilateral symmetrical spots

- Pathogenesis: failure to clear the depris dischrged into this region → accumulation of the depris between the RPE & the Burch's membrane → thickening of Burch's membrane .
& also lead to RPE atrophy → windo defect

- FA: hyperflurescene due to window defect.

- DD from hard exudated : - Age - Laterality
- Exudated arranged in clumps or ring + microanerysms + hges

? AMD هي drusen هل كل

NB. Risk factors of development of ADM :

large soft confluent drusen = high risk drusen

- smoking- hypertension – cataract

- *TTT of high risk drusen:*

(stop smoking,treat hypertension, follow up by daily Amsler gird.)+ multivitamins & antioxidants prevent development of AMD

(2) AMD with 2 main types:

1- Dry(atrophic) type :

- There is slowly progressive geographic atrophy of photoreceptors ,RPE & chorio capillaries.
- C/P: gradual impairment of vision
- FA → Window defect مهم جدا
- **TTT:** no effective ttt

(stop smoking ,treat hypertension, follow up by daily Amsler gird) low vision aid may be helpful.

2- Wet type : due to abnormal neovascularization from the choroids (CNV) under retina grow through adefect in the Bruchs membrane. (appears as gray-green lesion) → leakage

* C/P: impairment of central vision with metamorphopsia .
* *Complications*: - Serous PED
  - CMO
  - Sub RPE hge (Hgic PED) → then sub retinal hge (sensory detachement) → Vitreous hge(hge at multiple levels)
  - RPE tear
  - Massive exudation
  - Sub-retinal disciform scarring → permanent visual loss.
* *FA* → leakage (lacy pattern)

In occult CNV better to use ICG angiography for diagnosis.

** TTT:*

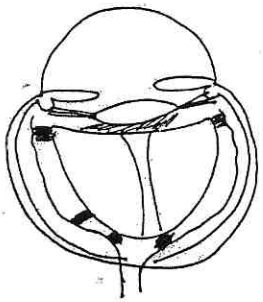
stop smoking ,treat hypertension & follow up by daily Amsler gird +

1) Argon laser photocoagulation: for well-defined extrafoveal CNV.
   the aim it to destroy the CNV without damage to the fovea.

2) PTD (photodynamic therapy) : for subfoveal CNV.
   - it is low energy laser(illumination) applications after IV injection of a special photosensitive dye (Vertoprofine)→ thermal damage & occlusion of the new BVs without harmful effect on normal retina.
   - Complication :
     sensitivity to bight light for 48 hours: العيان يجي متنكر

3) Intravitreal injection of Avastin (Anti VEGF) : for all types of CNV.

The Vitreous



Anatomy:

* ***Site:*** Posterior cavity between the lens & the retina.

* ***Transparent & clear*** (avascular & contains no nerves).

* ***Surfaces:***

1- Outer: Adherent to the retina at the ora serrata (vitreous Base =كيس شفاف) & around optic disc.

2- Anterior: adherent to the lens by hyalo-capsular ligament "ring like attachment" with retro-lental space inbetween.

* ***Hyaloid " Cloquet" canal:***

A narrow canal passing from the optic disc, through the central part of the vitreous substance, to the central part of the post. Lens capsule (Canal of Hyaloid artery → before birth only).

Nutrition:

From aqueous, Choroidal & Retinal vessels, as it's avascvular.

NB. The vitreous contains no BVs or nerve fibers.

Composition:

- 99% water.
- The rest is composed of collagen fibrils making a network & hyaluronic acid (MPS) → viscosity of the vitreous.

Function:

1- Optical function: A refractive medium.

2- Maintains the form of the eye.

3- Support for lens & retina.

Vitreous Hemorrhage

★ **Def. :** hge derived from retinal BVs (as the vitreous has no Bvs).

★ **Causes:**

* **Local causes:**

1- Trauma (+++).

2- Retinal tear (± R.D) . (U/S)

3- Diabetic retinopathy (from new BVs).

4- CRV thrombosis (from new BVs) .

5- Intra - ocular tumors : e.g. malignant melanoma eroding BVs. (U/s)

6- Eales disease : Idiopathic occlusive peripheral periphlebitis retinae of unknown cause may be **T.B. hypersensitivity** مهمه

→ neovascularization→ recurrent virtuous hge.& tractiona RD

TTT: PRP

* **General causes:**

Blood diseases : as leukemia , purpura , DM , hypertension , haemophilia.

★ **Clinical causes :**

* ***Symptoms:*** rapid ↓ of V.A.

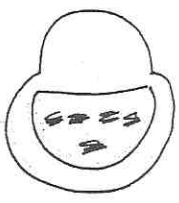
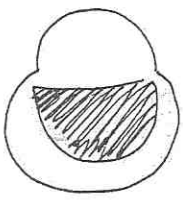
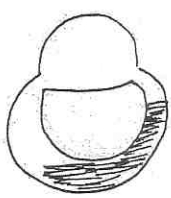
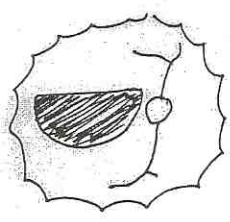
* ***Signs:***

- **Fundus exam.** → shows the Hge
- **Red reflex** → black هام

Intra vitreal : - red patches.
- Massive Hge.

Between the retina & vitreous (Subhyaloid hge.) See atlas page (158)

→ half spherical (boat shaped = level) by gravity.

★ **Fate:**

1- Absorption: slow as the vitreous is avascular.

2- Ghost cell glucoma

3- Organization (fibrosis): that may pull on the retina → tractional RD.

★ Treatment:

**** ttt of the cause.***

**** Medical ttt:*** 1- Vitamin C (to prevent recurrence).

2- Alpha - chymotrypsin (to enhance absorption).

* ***Vitrectomy:*** **If organized (for fear of R.D.)**

Musca Volitans (Vitreous Floaters)

Definition:

It is the appearance of moving opacities in front of the eye due to the presence of vitreous Opacities that cast a shadow on the retina.

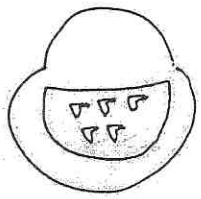
Causes:

* ***Congenital:*** due to remnants of the hyaloid artery. See atlas page (104)

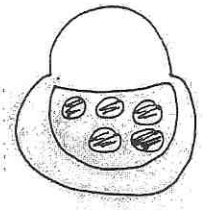
* ***Acquired:***

i) Endogenous : due to changes in the vitreous itself.

- Protein coagula : when the vitreous becomes liquefied & degenerated as in:
  1. High myopia.
  2. Following trauma.
  3. Old age (syneresis).
- Synchesis scintillans: Cholesterol crystals in the vitreous
  → Flat angular golden opacities freely mobile in a liquefied vitreous.



- Astroid bodies: calcium soaps in the vitreous.
  → large white bodies like snow balls not freely mobile as the vitreous is normal.



ii) Exogenous : due to changes in the surrounding structures

- Inflammatory cells : as in cyclitis , uveitis, papillitis.
- Blood cells : as in vitreous Hge.قول أسبابه
- Tumor cells : as in Retinoblastoma.

C/P: * **Symptoms :** - Moving opacities

- ↓ of vision.

* **Signs : by ophthalmoscope :**

- Dust like تراب
- Threads

+ Black RR

DD: Dark spot infront of eye :

- Fixed musca (corneal or lens opacity).
- V. Floaters (vitreous opacity).

Treatment:

- ttt of the cause
- Vitrectomy : for massive vitreous opacification or hge.

Indications of vitrectomy

(Diagnostic & Therapeutic)

1- Removal of pathological vitreous:

- Old standing vitreous Hge.
- Infected vitreous (Endophthalmitis).
- Tractional RD (vitreous fibrosis).

2- Removal of I. 0. F.B. : in the posterior segment of the eye.

♦Retinal hemorrhage?

As in vitreous hemorrhage + retina كل ال

♦What it the highest incidence of 1ry RD?

1ry RD is usually unilateral, in middle or old age males with high myopia.

♦What are the uses of cyrosurgery (cryotherapy) in ophthalmology?

(1) Cryopexy: to seal tear in RD.

(2) Cryoextraction:

- ICCE.
- subluxated lens.

(3) Cyclocryotheapy:

1. Absolute glaucoma.
2. Recurrent glaucoma.
3. Neovascular glaucoma.
4. Aphakic glaucoma.

(4) Cryocautery for:

1. Dendritic ulcer.
2. Rubbing lashes.
3. Spring catarrh (resistant cases).

♦What are the Causes of Robiosis iridis بنها وطنطا ?

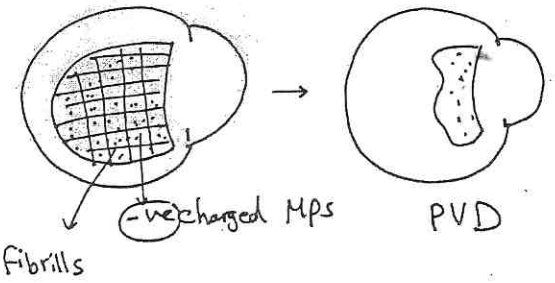
- Ischemic CRVO
- Some cases of CRAO
- DR
- Long standing Iridocyclitis
- Long standing RD
- IOS : carotid .

Vitreous detachment (VD)

(1) Posterior vitreous detachment (PVD): (الزبادي في الثلاجة)

- **Definition:** separation of the periphery of the vitreous from its attachment around the optic disc.

NB. The vitreous consists of reticulum of collagen fibrils, inside this reticulum, there are –ve charged MPS & hyalouronic acid molecules thus rebelling each other.



- **Etiology:**
  1) Senility with synersis in old age > 40 yrs→ loss of –ve charge → contraction of the vitreous gel → separation of its liquid from solid components → multiple fluid cavities → large central cavity).
  2) High myopia. 3) Inflammations e.g. chronic uveitis.
  4) Trauma

- **Clinical picture and complications:**

  1) Symptoms: 1- Musca volitants (moving black spots).
  2- Photopsia (seeing flashes of light).

  2) Signs:

  1. PVD with collapse of the vitreous gel as a result of syneresis: With associated vitreous organization and abnormal vitreo-retinal adhesions → pull on the retina and virteous → vitreous hemorrhage , retinal tear & retinal detachement.

  2. PVD without collapse of the vitreous gel: without synersis.

- **Treatment:** treat the cause.

(2) Anterior vitreous detachment (AVD):

* Post-traumatic usually and is accompanied with vitreous hemorrhage.
* Vitreous is detached from the lens and zonule.

differ in water & ptn Content
exudate ↑ptn | transudate ↑ H_2O.

Diseases of the Optic Nerve

Papilledema

Congestion not inflamation

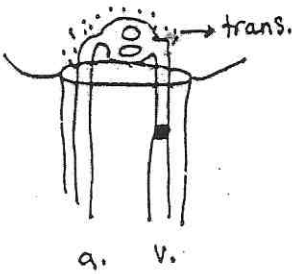
◈ Definition:

bilateral

It is passive (non - inflammatory) edema of the optic Disc.

(Due to congestion not inflammation & the fluid is mainly transudate).

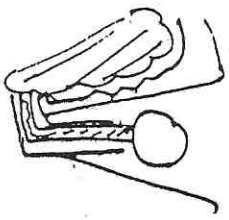
◈ Etiology:



① * **Intra - cranial** (most common):

Due to increased intra-cranial pressure e.g. in :

- Neoplasms (70%).
- Inflammation (as brain abscess, meningitis)
- Vascular: subarachnoid Hge ,
  cavernous sinus thrombosis, Aneurysms.
- Pseudo-tumor cerebri (benign increased ICT with no IC mass).
  - It is more common in young obese females.
  - Cause: Idiopathic, contraceptive pills or excessive vit. A & D
- ↓ in the cranial capacity : microcephaly.



unilateral

② * **Orbital** :

Due to e.g. : ♦ Neoplasms.

♦ Inflammation (orbital cellulitis) .

③ * **Ocular**:

Due to e.g. • CRVO.

• Hypotony e.g. corneal fistula or glaucoma surgery (V.D. → transudation).

④ * **Systemic**:

Due to e.g.:

• Malignant hypertension, Eclampsia (Toxemia of pregnancy)..

تسمم الحمل

- Polycythemia →↑ blood viscosity → CRVO.
- Anemia .??

N.B:

- ***The term Papilledema*** **is** usually used to describe disc edema due to increased ICT, while other causes are usually termed disc edema .

- ***Side: 1- Bilat. Papilledema*** ; in intra-cranial & Systemic causes.
  2- Unilat. Papilledema: in Ocular & orbital causes ,previous unilateral optic atrophy & Foster kennedy syndrome.

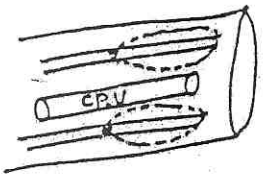
- Mechanism of Papilledema with ↑ ICT:

1- Mechanical theory: intra-cranial subarachnoid space is communicating with subarachnoid space around the optic n., so any ↑ in ICT will be transmitted to optic n. pressing on CRV → Transudation.

((Optic n. محوط بنفس ال **meninges of brain** زي ال جاكت))

2- Recently:

Increased ICT in the subarachnoid space around the op. n. leads to block of the axoplasmic flow ,later the swollen axons compress the veins → congestion → transudation.



NB. Axoplasmic transpots : is the movement of cytoplasmic organelles within a neuron between the call body & the terminal synapse.

◈ Clinical picture:

- Symptoms:

* ***Symptoms of increased ICT***: Headache – Blurring of vision projectile vomiting – Diplopia- ↓ consciousness

* ***Visual symptoms***:

(i) Early → Asymptomatic.

→ Sometimes, amaurosis Fugax: transient loss of vision

→ Diplopia: 6th nerve affection.

(ii) Late →gradual progressive Painless diminution of vision ,due to :

Post papilledemic optic atrophy (due to pressure & Ischemia).

Optic n.

- Signs:

1. Pupil: a) Early: RRR (round regular reactive).

b) Late: sluggish or irreactive to direct reflex,
due to optic nerve damage,
but the indirect reaction is preserved (APD).

2. Fundus (CRVO زي ال): See atlas page (143,144)

1 - Disc:

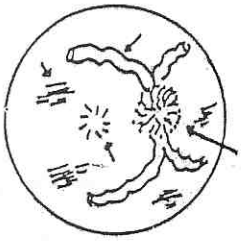
- **EARLY** - ill-defined (blurred) edges.
  - Hyperemic (dark red محتقن).
  - Loss of previous spontaneous venous pulsation

(but this is of little significance as 20% of individuals has no venous pulsation
but the presence this pulsation is strongly against the diagnosis of papilledema)

- **LATE (Established) cases:**
  - Filling or obliteration of the cup.
  - Elevated (up to 9 D= 3 mm):

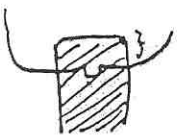
((elevation استخدمت عدسة قوتها كام Dعلشان تشوف ال))

((every 1 ml elevation need → 3 D))



2 - Retina: Flame Hge ,edema & cotton wool exudates.

3- Macula : Exudates (macular fan)



4 - Veins : Engorged & Tortuous.

- **LONG STANDING CASES:**
  - Marked elevation of the disc with a " Champange cork appearance".
  - Cotton wool spots & hges are absent.
  - Optociliary shunts.

3. Field:

1- Enlargement of the blind spot: due to ↑ the size of disc because of the disc edema & peripapillary exudative RD

See atlas page (143)

2- Central scotoma for blue (relative).

Cones

Blue	Red & green
Affected by pressure &	Affected by inflammation

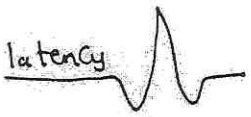
◊ Investigations :

1- Field of vision.

2- Color vision : Central scotoma for blue.

3- Radiology : a- X- ray.

b- CT scan.



*visual evoc potential

4- VEP : - Early : normal.

- Late : affected (long latency + low amplitude)

◊Complications:

Post - papilledemic (2ry) Optic atrophy يشرح بالتفصيل

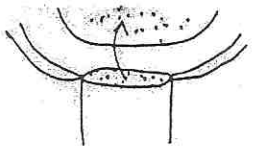
◊ **D.D.:** from other causes of ill-defined edge of the disc .

1)

	Papilledema	**Papillitis**	**Pseudo papillitis**
↑ ICT	Present	Absent	Absent
Vision	Normal (early)	Marked & rapid drop due to optic n. affection.	Hypermetrope.
Side	Commonly Bilate	Commonly Unilat.	Unilat. or Bilat.
Field	Central scotoma for blue & enlarged blind spot	Central scotoma for red & green.	Normal
Fundus	Disc is elevated Up to + 9 D = 3mm (transudate الماء يخرج كتير بسهوله) - Disc hyperemic +	Less elevation (about + 3D = 1 mm) due to high content of Ptns in exudates. - Disc hyperemic	Less elevation (about +2D).
Vitreous	Clear	Turbid (inflame. Cells & Ptns)	Clear

Optic n.

Pupil	Reactive (early)	Reactive (unsustained) due to bad conduction along the nerve fibers due to inflammation . upto APD	Reactive



Q: What is Hippus pupil ? د. سعيد شلبي

***2) Optic dicc drusen*:** مهم

- **Types :** 1- buried 2- exposed.
  its hyaline calcific material within the substance of the optic nerve
- **Clinically :** disc elevated bur the cup is preserved.
- **FA:** autofluorescence
- **US & CT:** detect the calicifications.

3) Anterior ischemic optic neuropathy AION : مهم جدا

- ***Def**: Sowllen optic disc due to local anoxia of the anterior part of the optic neve*
- ***Cause :** occlusion of the short posterior ciliary arteries leads to defective vision*
  → swollen disc with cotton wool exudates & splinter hge .

-Types: *1) Arteritic : due to giant cell (temporal) arteritis:*

- *Tenderness & loss of pulsation of temporal arteries.*
- *systemic manifestations :*
  - *1- Jaw claudications.*
  - *2- Scalp tenderness لما يسرح شعره & headache, even sclap gangrene.*
- *Investigation: - ESR & CRP raised*
  - *temporal artery biobsy؟؟ بتتعمل ازاي*
- *TTT : Intensive course of systemic steroids.*

2) Non- arteritic :

-PPFs : hypertension, DM & cataract surgery

- investigation: blood glucose & lipid profile.

- Typical altitudinal field defect ليه؟؟؟

- (no effective ttt).

◈ Treatment:

1- ttt of the cause:

e.g. Brain tumour .

2- Optic nerve decompression operation:

- Dehydrating measures : ↓ CSF pressure.
- Making an opening (fenestration) in the nerve sheath.
- Shunting op.(lumboperitoneal)

NB. Pseudo papilledema (Bilateral disc swelling):

1- Malignant hypertension - Toxemia of pregnancy- Anaemia.
2- Bilateral papillitis.
3- Bilateral Anterior ischemic optic neuropathy.
4- Bilateral CRVO.
5- High hypermetropia.

Optic neuritis

Definition:

It is inflammation of the op. nerve.

Classification:

1- Papillitis : Acute Inflammation of the disc.

2- Retrobulbar neuritis: Inflammation of nerve behind globe (orbital part).

It may be : - Acute.
- Chronic (toxic amblyopia).

3- Neuro-retinitis.

Papillitis

Etiology:

1- Demyelinating diseases: especially disseminated sclerosis "DS".

2- Antigen -Antibody reaction to a septic focus (toxin) or systemic immune diseases.

3- Metabolic diseases : e.g. DM, vitamin B_1 deficiency.

4- Infection:

- Iry : - Viral (polio – Herpes-Mumps) .
  - Bacterial (TB & $).
  - Cat scratch fever .
  - Lyme disease : spirochaetal infection follows tick bite (Borelia).
- 2ry : Extension of infection from:
  - a) Brain (meningitis).
  - b) Orbit (cellulitis).
  - c) Ocular (Uveitis & Retinitis) .
  - d) Sinuses: Sinusitis (especially sphenoidal).

Clinical picture:

◈ **Symptoms:** **rapid & marked** drop of vision down to PL+ Severe headache.

◈ **Signs:**

* **Fundus** : i- Disc ii- Vitreous (See table).
  iii- Retinal veins: less congested.
* **Field :** See table.
* **Pupil :** - unsustained reaction.
  - Late cases : APD.
* **VEP:** long latency & diminished amplitude.

NB. MRI in case of DS with show white plaques مهمه جدا

D.D:

see the table.

Fate:

(i) Recovery (in most cases) But recurrence is common.

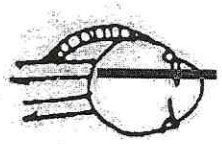
(ii) Post-papillitic (2ry) Optic atrophy.

Treatment:

(1) ttt of cause +

(2) Systemic: IV مش oral Cortisone + vitamin B complex + vasodilators.

Retrobulbar Neuritis



(I) Acute:

* **Etiology** : as papillitis.

* **Clinical Picture:**

  • *Symptoms*: as papillitis **+ Painful eye movements especially up & in**
  (as SR & MR muscles takeadditional origin from op. nerve meninges).

  • *Signs* :1) as papillitis, but the **fundus is Normal**
  (Disc & virtuous normal)
  → Pt say nothing & doctor say nothing

  2) Tenderness over the SR.

  3) Pupil: - Unsustained reaction.
  - Late cases : APD (afferent pupillary defect).

  4) Field : central scotoma.

  5) VEP: long latency & diminished amplitude.

* **Fate :** - Regression with complete recovery
  - If left untreated → 1ry optic atrophy.

* **ttt:** as papillitis.

NB. Pt say nothing & doctor say nothing:

1- Retrobulbar neuritis.
2- Hysterical blindness.
3- Occipital cortex lesion.
4- Pituitary apoplexy (hge.)

(II) Chronic:

Toxic Amblyopia

Definition:

It is bilateral optic n. damage due to "Exogenous toxins "

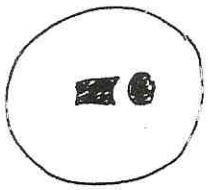
Toxins → chronic inflammation.

(1)Tobacco amblyopia :

* It is due to degeneration of the ganglion cells of the Papillo-macular bundle by the decomposition products of nicotine → Cyanide , either by :
  1- Chewing tobacco for many years.
  2- Smoking: Cigar & pipes (may be) cigarettes (rare).
  3- Exposure to tobacco dust in tobacco factories .

NB. Normally Vit-B_{12} neutralize the effect of cyanide , so pernicious anemia & malnourised pt. is in risk.

* ***Clinical Picture:***

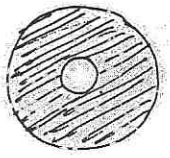


1- ↓ V/A .
2- Fundus : Normal (pt say nothing &doctor say nothing)
3- Field : Bilateral centro - Cecal scotoma especially for red & green (1st relative then the scotoma becomes absolute) (central to blind spot = between blind spot & point of fixation).

* **TTT :** stop tobaccc + VD + Vit. B complex tab . هبطل السجاير

(2) Quinine amblyopia :

* It is due to idiosyncrasy حساسية to the drug → Vasospasm.
* Clinical Picture :
  1- Fundus: Cherry red spot & attenuated BVs .
  (due to arterial spasm).
  2- Field: Bilateral total blindness bur in less severe cases –→Tubular field (leading to night blindness).



* **TTT :** stop Quinine + VD.

(3) Methyl alcohol (Methanol) amblyopia: منقوع البراطيش

Pathogenesis:

i- Breakdown of methyl Alcohol in liver → formaldehyde+ formic acid.

ii- formic acid → acidosis → Anoxia → Degeneration of the ganglion cells of the reina & brain .

(also formaldehyde → Degeneration of the ganglion cells).

Clinical Picture:

1- Acute stage: Nausea, Vomiting, headache, Coma & death.

2- If the patient survives, vision is usually lost due to Optic atrophy.

(4) Other drugs:

-Ethambutol - Isoniazide - Digitalis - Chloroquine

there is central field defect & it's a reversible toxicity.

Treatment of Toxic amblyopia:

* Stop the drug * Vasodilators * Vitamin B complex.

* In methyl alcohol Amblyopia:

Acute stage : 1- Stomach lavage with Ethyl Alcohol is required .

(competitive inhibition to Methyl Alcohol , it binds to receptors & displaces methyl الكبد هيستنضف).

2- Oral& IV sodium bicarbonate (for acidosis) :

may be Vision & life saving.

Optic Atrophy

Definition:

It is a term applied to the condition of the disc when→ op. nerve fibers are degenerated due to interruption of the nerve fibers at any point between the ganglion cell layer in the retina & the Lateral geniculate body (2nd order neuron)

NB. 3rd order neuron lesion → fundus لا يظهر في ال

Etiology:

Optic n.

(1) Iry Optic Atrophy: [Causes behind the eye**]**

1- Idiopathic.

2- CNS diseases:

* D.S. (++++) * Tabes dorsalis (neuro $ زمان).

3- Optic nerve diseases:

* Tumors : pressing on optic nerve or chisma r the tract

e.g. Pituitary gland & frontal lobe tumors.

* Trauma (fracture base of skull): pressing on op. nerve → injury .
* Ischemia : as in Severe blood loss (as the blood gush to heart & brain).
* inflammation : retro bulbar neuritis.

(2) 2ry Optic Atrophy : [Cause at the optic disc itself**]**

(1) Post - papilledemic optic atrophy .

(2) Post-Papillitic op. atrophy (post neuritic).

(3) Consecutive Optic Atrophy: [The cause is inside the eye =Following a Sever retinal disease**]**

-CRAO, ischemic CRVO

- R. Pigmentosa.
- Degenerative Myopia.
- Am. Family Idiocy (Tay –Sack's Disease).
- Chorio-retinitis : as Behcet disease

(4) Glaucomatous Optic Atrophy.

Clinical picture:

- Symptoms: - Gradual painless diminution of vision (down to no PL).

- Field defect : concentric contraction & scotoma.

- Signs:

1) Pupil: 1) *Unilateral cases*: afferent pupillary defect APD (paradoxical pupil = Marcus Gunn pupil) :

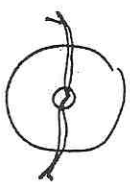
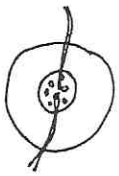
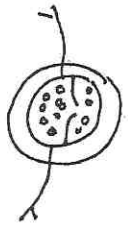
- ♦ Direct reaction : absent or affected .
- ♦ Indirect rection : preserved .

(due crossing of fibers at optic chiasma).

- This can be tested by **Swinging reflex**.

2) *Bilateral cases:* Bilat. Dilated fixed pupil .

2) Fundus: See atlas page (144,145)

	1ry	2ry	Consecutive	Glaucomatous
(I) Disc				
(1) Color	- Milky white	Dirty grey white **Due to fibrosis**	Yellow (waxy) **Due to gliosis** to engulf dead cells	Pale white
(2) Edge	- Well defined	- Irregular.	- Irregular.	- Defined & overhanging.
(3) Cup	- Moderately Enlarged but shallow	-Obliterated.	Mild obliterated.	- Deep & large.
(4) lamina C.	- Seen.	- Not seen	Not seen.	- Well seen(+++)
(5) Vessels	- Normal or mild attenuation.	- Attenuated & Sheathed	- Marked attenuation.	- interrupted with abnormal arterial pulsation.
(II) Rest of Retina:	- Normal	- Normal + pigmentations around disc	- Shows the cause	Tigroid & nerve fiber bundle defect.
					

DD of optic Atrophy :

1- DD of other causes of optic atrophy الجدول.

2- DD of other causes of gradual painless ↓ of vision.

3- DD of types of optic cupping : - Physiological.

- Atrophic .

- Glaucomatous. لهم جدول في زيادات ال جلوكوما

Treatment: **Hopeless**

1- ttt of cause.

2- Vasodilators + vitamin B complex. But visual Prognosis: Poor.

NB. Causes of APD:

1- Optic neuropathy : - Unilateral optic atrophy, Papillitis, Retrobulbal neuritis

2- Extensive retinal disease : CRAO , RD, Ischemic CRVO.

Normal disc :

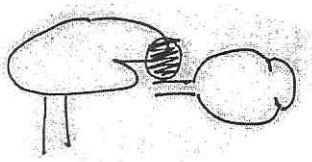
- Edge: Well defined .
- Color : Pale pink.
- Center: Physiological cup showing openings of lamina C.

Foster- Kennedy Syndrome

See atlas page (145)

- It's ipsi-lateral optic atrophy + contra-lateral papilledema (↑ICT).
- It is commonly seen with frontal lobe tumors.

NB. The atrophic optic n. not capable of being edematous.



Errors of Refraction

INTRODUCTION

Emmetropia = Normal person

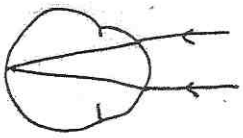
A condition of refraction in which with "*Accommodation is Completely relaxed*"

To differentiate between emmetrope & hypermetrope.

1- Parallel rays come to focus on the retina. on one point

2- Rays coming out from the retina leave the eye parallel (i.e. they meet at infinity) ∞

3- The retina &, infinity are called (**Conjugate Foci**).



~~Ammetropia~~ Normal = غير طبيعي

A condition of refraction in which "*with Accommodation completely relaxed*"

- parallel rays do "Not" come to focus on the retina.
- It includes:
  - Myopia.
  - Hypermetropia.
  - Astigmatism.
  - Aphakia and Anisometropia.

Factors affecting the eye as an optical system:

Myope ← زود أي حاجه

Hypermetrope ← قلل أي حاجه

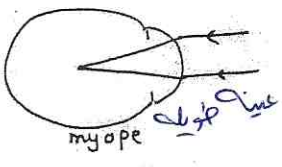
طول العين ***1- Axial length:*** It is the antero-posterior axis normally ~ 24 mm.

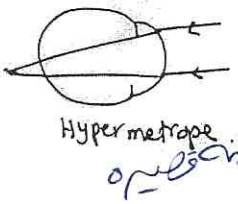
* Every 1 mm increase or decrease in the axial length of the eye results in 3 diopters change of the refraction.

قوة العين ***2- Refractive power:*** depends on

* Curvature of - Cornea &- Lens
* Refractive index of lens (RI).

3- Pathological : Some diseases lead to change in refraction.





Accommodation

Definition:

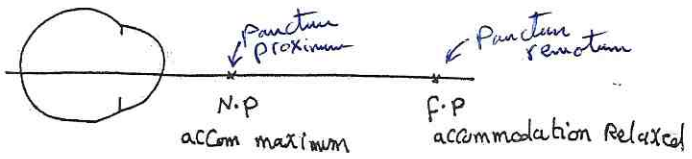
It is the ability of lens to change its refractive power so that it can focus object at different distances from the eye.

Stimulus: blurred image. د/حموده غرابه

Mechanism:

Contraction of ciliary ms. → smaller ciliary ring → relaxation of zonules → increase curvature of lens (being elastic) → increase its power.

NB. The posterior curvature not changed as it's supported by vitreous, the change occurs at the ant. surface,



1- Near point (Punctum proximum):

It is the nearest point from eye where objects appear clear (Here: accommodation is maximum).

Amount of accommodation مكانها علي حسب ال

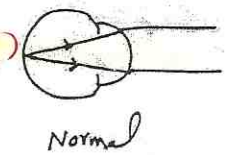
وده يعتمد علي سن العيان

- 5 years → 15 D
- 35 years → 9 D
- 40 years → 5 D
- 50 years → 2 D
- 60 years → 0 D

2- Far point (Punctum remotum):

It is the most distant point from eye where objects appear clear.

(Here: accommodation is completely relaxed).



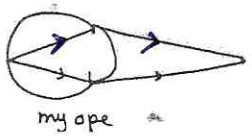
* For: - Emmetropia → at infinity. (infinity > 6 meters).

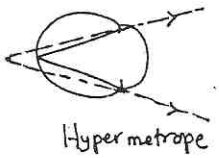
So in emmetrope the infinity & retina are conjugate foci

- Myopia → near than infinity (infront of the eye)

- Hypermetropia → Virtual point behind the eye.

((Conjugate foci of the retina = far point مكان ال))





3- Range of accommodation:

It is the distance between near point and far point.

4- Amplitude of accommodation:

It is the difference between accommodation for near and accommodation for far

{ = refractive power at near - refractive power for far}

* The Diopter

It is the unit of lens power.

It is defined as : the power of a lens which brings parallel rays falling on it to a focus at a distance of one meter (100 cm).

Power	Focal length
1D	100cm
2D	50cm
4D	25 cm

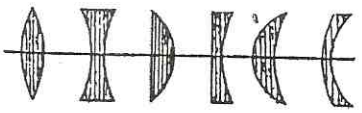
$P = 1/F$ بالمتر

$P = 100/F$ بالسنتيمتر

Focal length

قوة العدسة تتناسب عكسي مع البعد البؤري

* Types of lenses



(a) (b) (c) (d) (e) (f)

Fig. 6.1: Basic forms of spherical lenses: *(a) Biconvex; (b) Biconcave; (c) Plano-convex; (d) Plano-concave; (e) Convex meniscus; (f) Concave meniscus.*

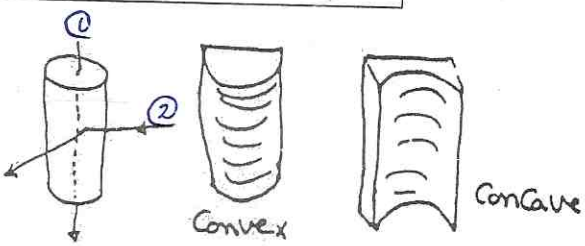
1- Spherical lenses:

- Are segments of spheres
  (has the same power in all meridians)
  bring light rays → point of focus. نقطه
- 2 types : concave and convex.

Convex (Plus +) 	**Concave (Minus -)** 
1- Thicker at the cente.r	1-Thicker at the periphery.
2- Objects looked at appear larger.	2- Objects looked at appear smaller.
3- Objects looked at more in opposite direction to the movement of the lens .	3- Move in the same direction.
4- Used for ttt of hypermetropia.	4- Used for ttt of myopia.

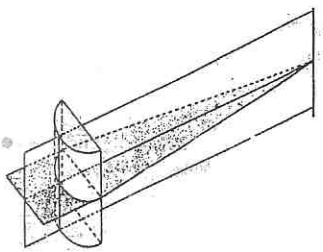
2- Cylindrical lenses

- Are segments of a cylinder
  (convex & concave)
  have no power in the meridian of the axis.



① - Light rays passing in a plane of axis of the cylinder → undergo no refraction.
(as it meets flat surface)

② - Light rays passing through the cylinder perpendicular to its axis → undergo maximum refraction. (as it meets curved surface).
bring light rays → line. خط
- for ttt of astigmatism.



3- Spherocylindrical (toric) lenses:

Spherical lens with cylindrical lens superimposed upon it.

Identification of lenses (spectacle)

1- Detection of lens type: by studying the image formed when 2 lines crossed at 90 ° are viewed through the lens:

a) Spherical lens: causes no distortion of the cross, however when the lens is moved from side to side & up down the cross also appears to move:

* Convex lens: the cross moves in the opposite direction to the movement of the lens.

* Concave lens: : the cross moves in the same direction to the movement of the lens.

b) Astigmatic lens: rotation of the lens causes "scissors" movement of the cross.

c) Prism : displaces one line of the cross

2- Estimation of lens power

1- By neutralization with lenses of opposite kind and known power : are superimposed upon the unknown lens until this combination make no movement of the underlying objects.

2- Phacometer (lensemeter) = focimeter. See atlas page (164)

3- Geneva lens meter = lens clock.

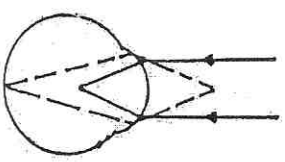
Myopia = Short sight

قصر النظر

Definition:

It is a condition of refraction in which *"with accommodation at rest"*

- Incident parallel rays come to focus in a "Point" **infront of retina.**
- Rays emerging from a point on the retina leave the eye convergent and meet at a point infront of the eye (punctum remotum = Far point).



Etiology:

1- Axial myopia (commonest type):

Due to increase ↑ in axial length of the eye (1 mm → -3D)

It may be (Types):

	1- Simple	2- Degenerative = High (Malignant) myopia	3- Congenital
1. Age of onset.	Around 14ys ثانوي	Around 7 ys ابتدائي	Since birth
2. Progress till.	About 25ys	Continue after 25 ys .	Stationary
3. Degree	Less than -6D	May reach -6 : -25D	About -10D
4. Degenerative changes of retina	Absent	Present	Less

HIGH MYOPIA : **Refraction > -6 D**

Axial length of the globe > 26 ml

2- Refractive myopia:

Due to increased↑ refractive power of eye. It includes:

i) Curvature myopia: as in

a- Increase curvature of cornea :

(keratoconus & keratoglobus).

b- Increase curvature of lens ((lenticonus –

Spasm of accommodation (irits or miotics) -

-ant. dislocation – Subluxated lens))

ii) Index myopia:

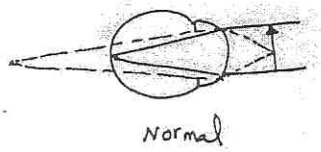
a- ↑ **RI Nucleus:** Nuclear cataract :

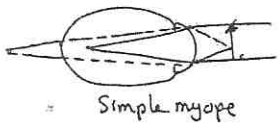
b- ↓ **RI cortex** : Uncontrolled D.M. (hyperglycemia)

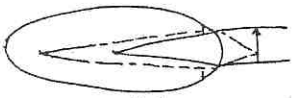
iii) Pathological: KC, lenticonus , DM , senile sclerosis, iritis,

Ant. Dislocation of lens.

Clinical picture:


Normal


Simple myope


High myope

* Symptoms

1- Simple: →

- Indistinct far vision.
- Frowning (عبوس) :

  Screw the lids to make the palpebral fissure narrow to cut the peripheral rays like a pin-hole & prevent the aberrations (this will lead to blepharitis , styes , headache& redness) .
- **NB.** The near work is comfortable due to minimal use of accommodation.

2- Malignant:

i) Indistinct far vision .

ii) Discomfort after near work (muscular athenopia)

→ Uni ocular & close near work:

Due to disproportion between accommodation & convergence need (عدم التوازن في الاحتياجات) .

iii) Musca volitans: due to vitreous degeneration.

iv) Photopsia: due to stimulation of rods & cones by V-R traction .

v) Night blindness: due to degeneration of retinal periphery.

(due to Occlusion of the peripheral retinal BVs , it's smaller than the central).

vi) Field defect.

* Signs

1- Simple → Normal Eye.

2- Malignant:

i) *Oblique illumination:* - Large eye: (- Large cornea - Deep A.C.)
- Large pupil.
- Pseudoproptosis - Blue sclera (thin sclera)

ii) *Fundus examination:*

Exaggerated Tigroid (نمري الأثر) fundus : See atlas page (155,156)

- It's red areas (Choroidal BVs) alternating with dark areas (choroidal pigment).
- Atrophy of RPE so the retina becomes more transparent.

iii) *Retinoscopy :* the movement is against.

iv) *Tonometry:* Glaucoma (OAG or Pigmentry).

v) *Perimerty :* Peripheral field defects.

Complications:

1- Complicated cataract: Due to decrease the blood supply to the eye.

↓ ACC. → ↓ Convergence → Weak MR ms

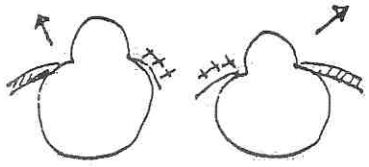
2- Squint: * **Mild error:** - Latent divergent squint (Exophoria) حول كامن المخ يعدله

* **High error:** - Manifest divergent squint (Exotropia)

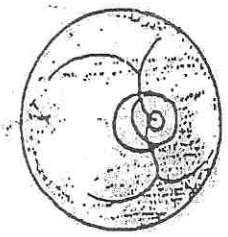
حول ظاهر المخ مش قادر يعدله

- Apparent convergent squint (due to -ve angle alpha)

حول ظاهري غير حقيقي



3- Retinal changes :[fundus picture of high myopia]:

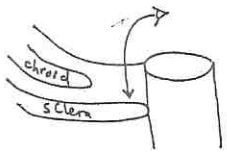


i- Temporal crescent: - It is a white crescent .

See atlas page (156) - Site: temporal to disc.

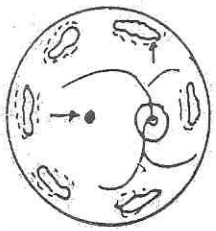
- Cause: traction on the choroid showing the sclera.

- Later : it will be annular. See atlas page (156)



ii- Chorio-retinal degeneration, scars & atrophy See atlas page (155)

- It is white areas surrounded by pigmentations (macrophges)
- Site: peripheral .
- Cause : choroidal & retinal atrophy showing the sclera .
- It may lead to → tear → **RheagmatgenousR.D.**



iii- fuch's spot: See atlas page (155)

- It is a dark spot.
- Site: at macula.
- Cause: Unknown: * Fibro-vascular invasion of the fovea due to rupture of Bruch's membrane →sub-foveal hge
  → when resolve it will leave heamosidren
  * May be proliferated RPE at the macula.
  → loss of central vision.

iv- lacquer cracks: due to breaks in Bruch's membrane
→ **CNV** مهم → **subretinal hge**

v- Lattice degeneration: arborizing net work of white line, due to discontinuity of internal limiting membrane & atrophy of sensory retina → hole.

4- Vitreous degeneration:

Vitreous becomes - Liquefied - Contains opacities (Musca volitans) - PVD

5- Posterior staphyloma:

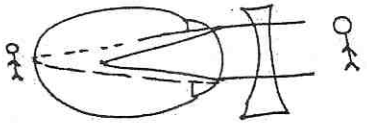
- It is ectasia at the posterior segment .
- Site: temporal to disc.
- Seen: by fundus examination or U/S

6- Macula : - Hge . - Degeneration. – Hole.

7- Glaucoma :

- OAG (association).
- Pigmentry glaucoma (due to release of uveal pigment from stretch → angle)
- Steroid responsivness

8- Consecutive optic atrophy : due to advanced chorio-retinal degenrations.



Treatment:

I- Optical treatment:

i- Glasses

(concave or minus lenses with its focal point coincided with the far point مهم , so incident parallel rays will diverge before entering the eye as they are coming from the far point → focused by the optical system of the eye on the rerina):

- **Simple myopia:** give full correction (least power → 6/6),
  to avoid use of accommodation for far → athenopia

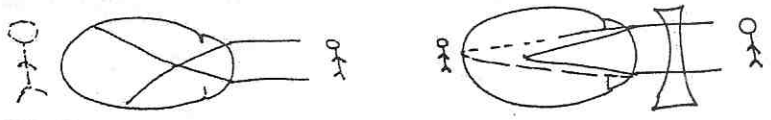
- **High myopia:**

1) Children: give full correction (to allow normal mental development)

2) Adult (1st time)جاى لأول مرة: give him under-corrected tinted glass
(why?)

Because the patient is not accustomed to the small, sharp and bright retinal image given by the high minus lens → ocular discomfort.

(So, for a myope -20.0 D give him e.g. - 18.0D).



ii- Contact lenses: in high error

Advantages: 1- Cosmetically better.
2- Bigger field.
3- No significant decrease in retinal image size.

((CL بيكون مقاسها اقل))

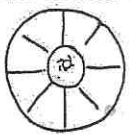
(+ General ttt of high myopia : - Vitamins & ca++ (ايه دورها)
- Instructions: (near work with good illumination & good position).

II- Surgical treatment (Refractive surgery) تصحيح النظر جراحيا

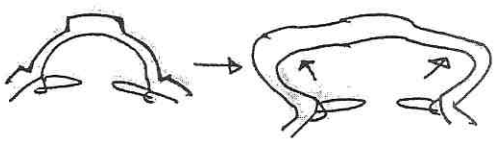
A- CORNEAL:

1) Radial keratotomy (rarely used nowadays): See atlas page (159)

* 4-8-16 incisions (deep reach the D.M. & radial) are made from the edge of the optical zone(central 2.5 ml) → limbus



* **Idea:** the periphery of the cornea will bulge (by the IOP) &the center will flatten



*** Limits:** - Degree: not less than -2D→over correction.
& not more than -7D→ under correction.
- Age: not before the age of 20 yrs. (unstable myopia).

*** Complications:**

1) Operative: perforation

2) Post-Operative: - Infection - Scars → colored haloes.
- Intrastromal inclusion cyst.

2) Excimer laser:

to flatten=applanate=evaporate the central cornea

Contraindications: Thin cornea, ocular disease , dry eye. Collagen diseases

I- PRK(photorefractive-keratectomy):

• Steps:

- Remove the epith. Using alcohol or brusher.
- Using excimer laser, do applanation to Bowman's membrane & ant. Stroma at the optical zone

• Indications: 1- Myopia -6D → -12D .
2- Hypermetropia +2D → +5D.(ازاي؟)

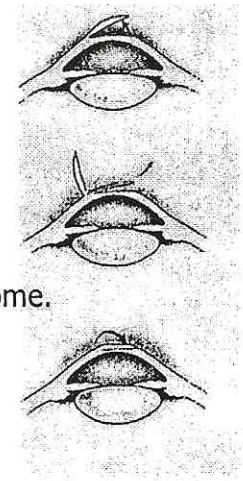
• Complications:

1- Post-operative bulging
2- Post-operative haze due. to affection of the Bowman's membrane
3- Under correction or over-correction.
4- Post operative severe pain due to corneal ulcer →ttt T CL

• Advantage: Preserve thickness.

II- LASIK (laser intrastromal keratomileusis)

اعادة تشكيل القرنيه من الداخل باستخدام الليزر



◊ Steps:

1- Make a corneal flap 100-160 micron
(Epith. + Bowman's membrane + ant. stroma) using microkeratome.
2- Using excimer laser, do applanation in the stromal bed.
NB. Residual stromal bed should be not less than 250-300 micron
3- Reposition of the flap.

◊ **Indications:** as PRK.

◊ **Complications:**

1- Bulging (not preserve the thickness).

2- Wrinkling of the flap.

3- Incomplete cutting of the flap.

4- Complete cutting of the falp

5- Button holing

6- Interface opacity.

◊ **Advantages:**

1) No post-operative haze (not affect **B**owman's membrane).

2) No post-operative pain.

3) More accurate specially with the use of Wave front technique (to remove low& high order aberrations→ Customized LASIK ليزر تفصيلي).

III- LASEK (laser sub-epithelial keratomileusis):

◊ **Steps:** As PRK but the epith. is removed 1st using alcohol then reposited after using the excimer laser.

◊ **Advantages:**

- No post-operative pain.
- Preserve the thickness.

◊ **Disadvantages**: toxic effect of alcohol on cornea

◊ **Indication:** Thin cornea.

IV- EPILASIK:

As lasik but the flap is thinner (epith.) using **epi-keratome**

V- FEMTO LASIK (INTRA LASE) = Bladeless lasik:

As lasik but the flap is removed using **femto second laser** not microkeratome.

NB. Before any excimer laser operation do *pachymetry & corneal Topography* to evaluate the corneal thickness(if < 500 micron it's not suitable for this surgery) & presence of KC See atlas page (67,76)

NB. Not done before the age of 21 yrs (stable myopia).

3) Epikeratophakia: e.g aphakia.

4) keratoplasty: e.g keratoconus.

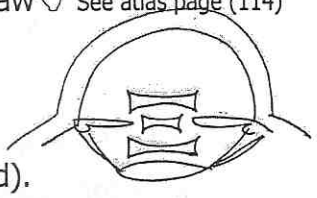
5) Intra corneal rings: Intacs, Ferrara rings or Kerrings for ttt of keratoconus. See atlas page (68)

6) Intra corneal lens(Corneal inlay) : corrects myopia up to - 3 D .

B- NON-CORNEAL:

1) Phakic IOL:

- Site: * AC * PC (infront of crystalline lens) * Iris Claw See atlas page (114)
- Advantages: 1- Correct high error (up to -20 D).
  2- Not affect the cornea or the retina.
  3- Reversible (can be secondary implanted).
- Disadvantages: - Iritis → Glaucoma.
  - Cataract.



2) Clear lens extraction:

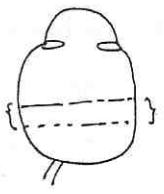
- Advantages: correct high error more than -20 D(theoretically -25 D).
- Disadvantages: - vitreous herniation → RD.
  - Loss of accommodation.

3) Scleral resection:

If there is high myopia with post. Staphyloma.

NB. In late cases with optic atrophy→ *low vision aids*:

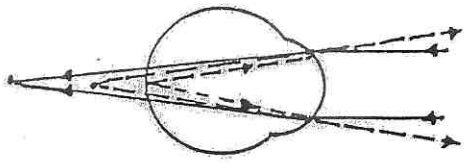
See atlas page (164)



- Magnifying lens : for far - Telescopes : for near

Hypermetropia (hyperopia)

★ Definition



It is a condition of refraction in which

" *with accommodation at rest* "

i- Incident parallel rays come to focus in a point behind the retina,

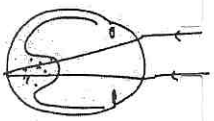
ii- Rays emerging from a point on the retina leave the eye divergent and appear as if coming from a virtual point behind the retina (p. remotum).

★Etiology

1-Axial hypermetropia: ((every 1mm → 3 D))

Due to small antero-posterior axis (small eyes) seen in children (< 7ys) as the eyes are small. As the eyes grow, they become less hypermetropic:

- If the eyes reach the proper length (24 mm) → emmetrope.
- If the eyes don't reach the proper length → remain hyper.
- If the eyes exceed the proper length → become myopic.



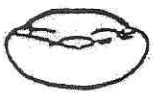
(It may be acquired, if the retina is pushed forward as in choroidal hge or tumors ,exudates or Central serous retinopathy (CSR)

NB. What is CSR?

- Idiopathic focal RPE defect (disturbance of outer blood retinal barrier) → fluid leak from choroids to the subretina → sensory macular detachement
- C/P: blurred vision + micropsia
- FA: smoke-stack or ink-blot ((pooling = hyperfluerescence ↑ size & intensity))
- OCT : diagnostic
- TTT: - most cases not require ttt as it is self limiting.
  - Argon laser to the RPE leak may speed the recovery.
  - PDT: for subfoveal leak.

2- Refractive hypermetropia:

Due to decrease refractive power of the eye:



i- Curvature H.: due to flattening of the cornea (cornea plana).

ii- Index H.: due to ↑RI of the cortex → ↓ RI of lens :

- Immature Cortical cat.
- Hypoglycemia

3- Pathological : Aphakia , anterior lens dislocation , microphthalmos, Cornea planna, RD, IO tumours.

★ Components of hypermetropia:

- In a hypermetropic patient, the tone of the ciliary ms. corrects a portion of hypermetropia (latent H.) and is about 1 D. The remaining portion (Manifest H.) may be corrected by accommodation (facultative H.)
- If the patient cannot correct all his manifest H, a part will remain uncorrected (absolute H.) and leads to decrease of vision.

Total hypermetropia(+10)

- Latent (+1)
- Manifest (+9)
  - Facultative(+5)
  - Absolute(+4)

- Total H.:
it's the amount of H. measured under the effect of atropine (no accmmmodation , no tone)
(= the power of convex lens → 6/6 (with atropine).

- Latent H.: Amount of H. corrected by the tone of ciliary ms.
(= Total - Manifest).

- Manifest H.: Amount of H. not corrected by the tone of ciliary ms.
(= **highest** power of convex lens → 6/6 (without atropine). ★

- Facultative H.: the part of manifest H. corrected with accommodation.
(= Manifest - Absolute).

- Absolute H.: The remaining part of manifest H. not corrected by accommodation.
(= **least** power of convex lens → 6/6 (without atropine). ★

NB. - In infants all manifest is → Facultative (very high accommodation).
- In old age all manifest is → Absolute (no accommodation).
((with age the absolute hypermetropia increase)).

★Clinical Picture

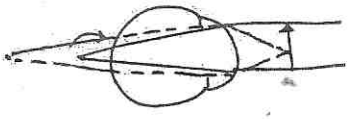
- Symptoms:

طفل a-Young age: no symptoms (accommodation is strong and correct all H.).

شاب b- With advance of age: the pt. finds difficulty especially in near vision:

- Early Presbyopia: (as accommodation is consumed to correct the error).
- Accommodative athenopia : due to over use of accom. →

1- Pain in & around the eye.
2- Redness
3- Lacrimation & recurrent stye.



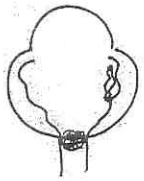
عجوز c- Old age: difficulty in far & near vision.

- Signs:

1- Mild degree: as normal eye.

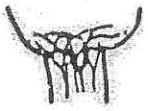
2- High degree:

- Oblique illumination: small eye (Small cornea- Shallow AC- Small pupil).
- Fundus exam.: - Bright reflex.
  - Tortuous retinal vessels.
  - Pseudo-papillitis.



- Retinoscopy : the movement is with .

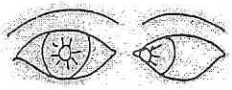
* Pseudo-papillitis: ill-defined slightly elevated optic disc with obliteration of the cup due to crowdening of retinal nerve fibers in a small lamina cribrosa.زحمة يا دنيا زحمة



★Complications

↑ ACC. → ↑ Convergence → Strong MR ms.

1- Squint: - Mild degree: Latent convergent squint (esophoria).



- High degree: - Manifest convergent squint (Accom. esotropia).
  - Apparent divergent squint (due to large +ve angle alpha).

2- Angle closure glaucoma: due to small eye with narrow angle.

★ Treatment:

*** Mild degree:** No symptoms → so, no ttt.

*** High degree:**

1- Optical correction - Glasses (convex lens) , or

- Contact lens.

طفل i- Children: give full correction (highest plus lens) : to avoid use of accommodation for far, which may lead to accommodative squint.

بالغ ii- Adult

*1st give the highest "tolerated" convex lens (as the patient cannot tolerate full correction because he cannot relax the ciliary ms. at one time due to its spasm.

* After 6 months → give full correction (highest plus lens) for far & for near he can use the accommodatiom.

عجوز iii- Old: - For far → give full correction (the Accommodation is weak)

- For near → give full correction + 3 D *(persbyopic correction)*

نظارة القراءة

2-Surgical ttt: 1- PRK & LASIK.

2- Holmium Laser thermo-Keratoplasty:

6-18 laser burns are placed in 2 rings in the periphery of cornea ,this produces stromal shrinkage + ↑ central curvature.

3- Phakic IOL.

4- Epikeratophakia.

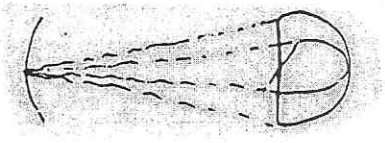
Astigmatism

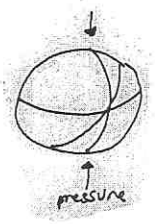
A → No

Stigma → Point.

◈ Definition:

It is a condition of refraction in which the incident parallel rays don't come to a point focus on the retina but form a line (as the eye doesn't have the same power in all meridia)





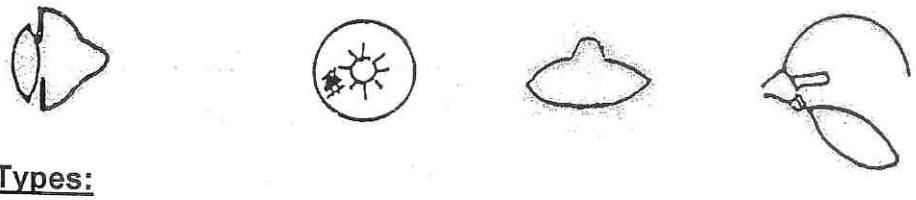
Errors of ref.

◊ Etiology:

Due to irregularities in the curvature of cornea or lens.

1- Corneal astigmatism : as in Keratoconus , Opacities , large chalazion or post-opertaivce scar or congenital in 95% of cases.

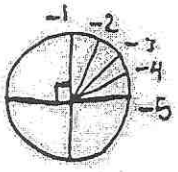
2- Lenticular astigmatism: as in Lenticonus or Subluxation or immature cat.

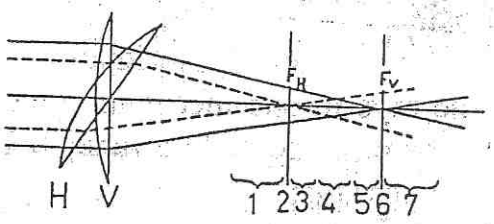


◊Types:

I- Regular

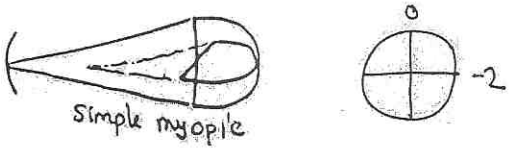
- Meridians of the highest and least powers (principle meridians) are perpendicular to each other.
- Transition from the highest meridian to the least is gradual.
- Usually congenital.
- The image formed as Sturm of Conoid جدا مهم ← **5 possibilities.**





1- Simple astigmatism: in which * one meridian → emmetrope
& * the other → ammetrope.

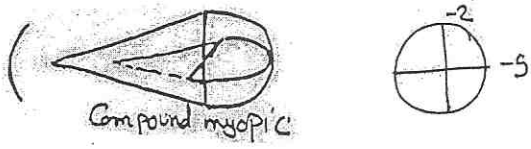
so, we have: i- Simple myopic astigmatism.
ii- Simple hypermetropic Astigmatism.



2- Compound Astigmatism:

in which → both meridians are ammetrope but of the same type,

so, we have i- Compound myopic astigmatism &
ii- Compound Hypermetropic astigmatism.



3- Mixed Astigmatism:

in which * one meridian is myopic

* and the other is hypermetropic.

NB. - *Straight Astig (vertico-horizontal) :* the principle meridians at the 90 & 180 degree

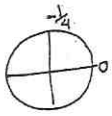
- *Oblique Astig*: the principle meridians are perpendicular but not at the 90 & 180 degree.

II- Irregular

- Meridians of highest and least powers are "not" perpendicular to each other.
- Transition from the highest meridian to the least is "not" gradual.
- e.g. after corneal scarring, keratoconus & lenticonus.
- It can't be corrected by glasses .

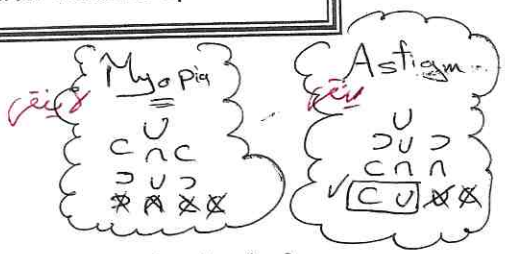
Rule of astigmatism

In Emmetropia: the vertical meridian (due to pressure by the lids on the cornea) is more curved than the horizontal → more powerful→ more myopic (1/4 -1/2 D =Physiological astigmatism).



In Astigmatism:

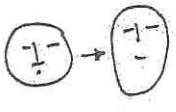
- If the vertical meridian is more myopic → astigmatism. **"With the rule".**
- If the horizontal meridian is more myopic → astigmatism. **"against the rule".** e.g. after cararact op.



◈ Clinical picture:

*** Symptoms:**

- Indistinct vision (near & far)
- Accommodative athenopia (headache) : Due to subsequent contraction& relaxation of Ciliary ms if there is hypermetropic element .
- Distorted objects. e.g. rounded objects appear oval or irregular due to difference in the curvature → difference in the magnification.
- Uniocular diplopia in high degrees of astigmatism.
- see better in some directions
- Squeezing of lids >> chalzion (viscous circle).



Errors of ref.

*** Signs:**

1- Gross signs: e.g.: keratoconus, corneal opacities, and subluxation.

2- Landolt's chart: See atlas page (117)

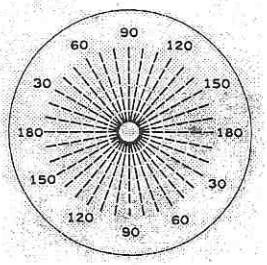
"some" openings in **"the same line"** are not seen.

(مثلاً شايف ال Vertical فقط)

3- Placcido's disc: shows irregular circles.

4- Astigmatic fan :

Some lines appear sharp black & others are blurred or grayish. See atlas page (163)



5- Ophthalmoscopy: the optic disc appears oval.

(في حاجه كمان هتظهر Oval)

6- Retinoscopy: جهاز عمل النظارة القديم

7- Auto-refractometer: جهاز عمل النظارة بالكمبيوتر See atlas page (163)

8- Keratometry: the curvature (and the power) of each meridian of the cornea can be measured. See atlas page (75)

9- Corneal Topography: See atlas page (67)

(خريطة القرنية بالكمبيوتر)The most accurate method but expensive.

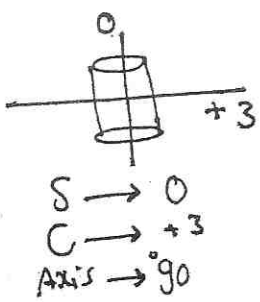
◈ Treatment:

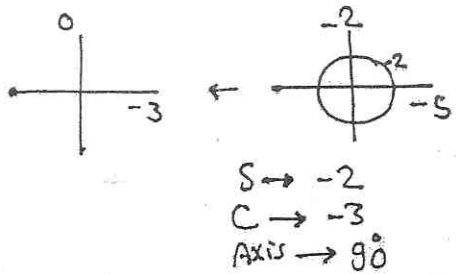
A- Regular astigmatism; corrected by:

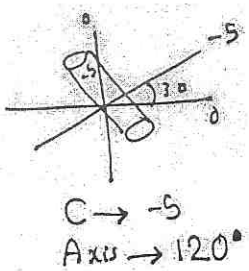
*** Optical ttt:**

- Glasses: 1- Simple astig. : Give **cylindrical** lens with its axis perpendicular to the Ammetropic meridian.

2- Compound & mixed astig.: give **sphero-cylindrical lens**







- Contact lenses : 1-2 C → Soft CL. (spherical equivalent)

2-7 C → Torric CL.

NB. Simple transposition مهم جدا??تعرف تعمل

Indications :

- To keep the axis of the cylinder in both eyes nearly in the same direction
- To keep the lens as light as possible . اديني مثال

> 7 C → Hard(rigid gas permeable بدلها)CL.

*** Surgical TTT**

* Lasik: in low degrees (up to 5 C).

B- Irregular astigmatism:

(1) corneal:

1- Give RGP C.L (hard بدل ال).

2- PK : in deep corneal opacity.

3- lamellar keratoplasty : in superficial corneal opacity.

(2) lenticular : lens extraction.

Aphakia

- A → No. **- Phakos → lens.**

* **Definition:** It means absence of lens.

* **Etiology:**

1- Congenital: very rare.

2- Acquired: i- Surgical removal (most common):

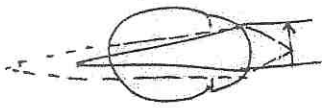
- Cataract. - ttt of myopia. - Dislocation& subluxation.

(??clear lens امتي نشيل)

ii- Trauma to the lens:

- In children (with opened capsule→ absorption of lens matter "soft").
- In adult → Posterior dislocation of lens (due to weak zonules).

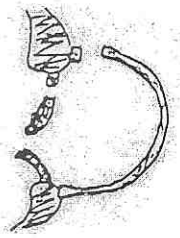
*** Clinical picture:**



- Symptoms:

- Defective vision especially for near (no accommodation).
- Blue or violet colour: may be seen by the pt as the aphakic eye

is sensitive to UV rays which are normally absorbed by the normal lens if present.



- Signs:

1- +ve history of operation.

2- Scar of previous operation (at the upper limbus غالباً).

3- A.C → deep.

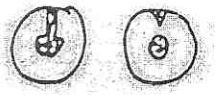
4- Iris: - Iridectomy. (It's done with ICCE to prevent iris prolapse in the wound & pupillary block glaucoma by the virteous or AC IOL) .

- Tremulous=iridodonesis (lack of support by the lens).

5- Pupil: - Jet black. (as there is no reflection of light to the eye of the examiner زي الاطفال).

- May be rounded or irregular (with key-hole iridectomy).

- After cataract >> grayish . See atlas page (111)

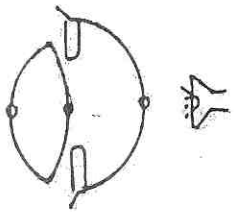


6- Absence of 2 out of 3 Purkinje- Sanson images :

* 1 image only ***"Diagnostic"*** .

* الا اذا pseudophakia (IOL implantation) with intact post capsule

→ 2 images present.



7- Refraction: shift "toward hypermetropia".

8- fundus examination : lens is not present in the vitreous .

<u>Optical condition after lens extraction</u>:

1- Loss of accommodation.

2- Astigmatism against the rule 1- 3 D; (due to flattening of vertical meridian by scar of operation at upper limbus).

NB. This astigmatism may be with the rule ???? امتي لو شاطر

NB. This astigmatism is minimal with phaco due to small scar.

3- <u>Loss of a refractive surface:</u> shift of refraction towards hypermetropia:

a- A previously emmetropic patient → hypermetropia + 10.0 D.

b- A previously hypermetropic patient → hypermetropia > + 10.0 D.

c- A previously myopic patient:

- Simple myopia → Hypermetropia < + 10.0 D
- High Myopia. → Much less myopia.
- Theoretically, a myope -25.0 D → Emmetropia.

4- <u>Anisometropia in unlilateral aphakia:</u> اكتبها

NB. Discuss pseudophakia: It is the presence of IOL.

- Same C/P of aphakia except : 1- Purkinge images → 2.
  2- Refraction → normal (no shift).
- Types of IOl :.....

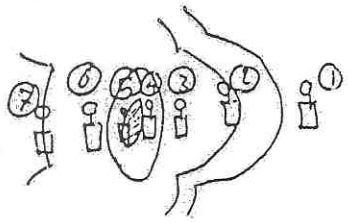
*** DD.: from post. Dislocation of the lens :**

1- History : - No history of operation. - There is a history of trauma.

2- Scar : No limbal scar.

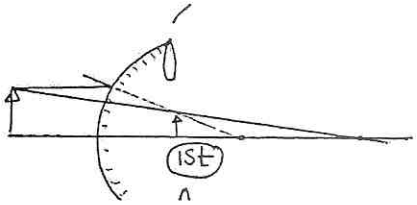
3- No iridectomy.

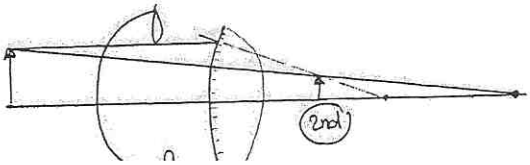
4- Fundus ex. : dislocated lens in the vitreous.

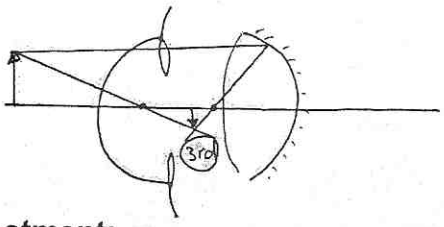


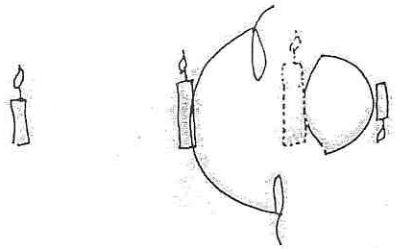
Purkinge images : using light source with oblique angle 35° with cornea:

	I	II	III
1) formation:	By ant. Corneal surface (convex mirror).	By ant. Lens surface (convex mirror).	By post. Lens surface (concave mirror).
2)size:	Large	Largest (ant. Lens surface is the least convex)	Small
:3)brightness	Bright.	Faint(inside the vitrous).	Bright.
4)nature:	Virtual	Virtual	Real
5)position:	Erect	Erect	Inverted
6)movement:	With	With	Against
7) with accommodation:	No change in the size	Smaller in size (due to ↑ curvature of lens)	Smaller in size (due to ↑ curvature of lens)









***Treatment:**

1- Glasses: See atlas page (116)

2 pairs : one for far & one for near (or bifocal or trifocal lenses).

* Disadvantages: 1- Large image (25-30%) .

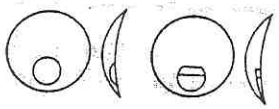
So can not be used in unilateral aphakia → Anisokonia

2- Prismatic effect → Limited field & Aberrations

2- Contact lenses:

Rarely tolerable due to - Loss of accommodation&

- Difficult dexterity in old age.



Bifocal & trifocal lenses

3- IOL Implantation: either Iry or 2ry أفضل طريقة

>> no magnification . better field .

if the pt. was emmetrope :

- Glasses : +10 Sphere / +2 Cylinder axis 180°
- CL : + 14D
- AC IOL : + 19 D
- PC IOL : + 21 D

So. Bilateral aphakia can be corrected by:

- Glasses. - Contact lenses. - IOL.

Unilateral aphakia can be corrected by:

- Contact lenses. - IOL. - But not glasses (glasses → Anisokonia→ diplopia)

Anisometropia

Definition:

It is a significant difference in refractive power between the 2 eyes (rarely tolerable when the difference → 4 Diopters).

Etiology: 1- Congenital. 2- Acquired e.g, unilateral aphakia.

Types: **(1) Simple anisometropia :** one eye is emmetropic & the other is ammetropic.

(2) Compound anisometropia: both eyea are hypermetropic or myopic.

(3) Mixed anisometropia : one eye myopic & the other hypermetropic.

Problem: Cannot be corrected by glasses (why?) → anisokonia → Binocular diplopia.

Vision: **1- Uniocular:** → anisometropic ambylopia & squint.

2- Alternating : - Myopic eye → for reading.

- Hypermetrpic eye → for far vision.

3- Binocular : → Athenopia & Squint .

Treatment:

1- Glasses:

- Not used .
- Can be used after under-correcting the eye with a higher error (at the expense of good vision)

2- Contact lenses: produce retinal image difference 6-10%.

3- secondry IOL: reduce the retinal image to less than 1%.

4- Refractive corneal surgery: e.g. Epikeratophakia.

5- Aniseikonic lenses مهمه:

- Glass plate which causea magnification & has no refractive power.
- Not used as it leads to only 5% magnification.

N.B.:

Aniseikonia: it is significant difference between retinal image sizes of the 2 eyes.

Presbyopia

♦ **Definition:**

It is physiological recession تراجع of near point (punctum proximum) due to decrease in accommodation power with age making near work uncomfortable.

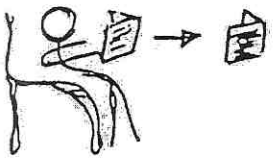
♦ **Etiology:**

Errors of ref.

1- Lens sclerosis in old age →↓ physical accommodation.
(Related to change in the curvature of the lens).

2- Weakness of the ciliary ms →↓ physiological accommodation.
(Related to contractile power of Ciliary ms.).

♦ Clinical picture:



1- Difficulty in near work: The pt. holds the book at a greater distance.

2- Accommodative athenopia → headache.

N.B the onset of presbyopia varies with the refractive state of eye

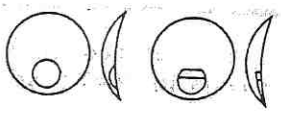
- Emmetrope : around age of 45 ys.
- Hypermetrope : before that age.
- Myope : after 45 ys.

N.B The accommodation power ↓ with age :

-35 yrs → 9 D.
- 40 yrs → 5 D.
- 45 yrs → 3 D.
- 60 yrs → 1 D.

♦ Treatment:

1- Reading glasses:



Bifocal & trifocal lenses

i- Unifocal lenses: for reading only .

ii- Bifocal lenses: for far and near vision.

iii- Multifocal lenses: for all distances.

2- Bifocal & Multifocal Contact lenses : tried with limited success.

3- Multifocal IOL: used after cataract extraction.

4- Surgery: incisions and implants for presbyopia (under research).

5- Excimer laser: - Lasik surgery for far & glasses for near.
- Laski correction one eye for near & other eye for far.
- Prepyopic lasik : for far & near in the same eye.

NB. Reading glasses = (3D + error)

1- Correct error for far (if present e.g. + 10D).

2- Measure near point .

3- Estimate the amplitude of accommodation.

4- Keep 1/3 of accommodation in reserve & 2/3 in use.
(Not to use all the accom. & also not to relax all the accommodation).
5- Add plus lenses to 2/3 the accommodation to make the sum 3D.
(3 اشمعنا → because the standard working distance is 33 cm).
6- Add this plus lens to the far glasses of the pt.

Contact lenses

Very thin lenses applied directly to the cornea.

◊ Principle:

Abolishes the cornea as a refractive surface and replaces it by "contact lens-fluid lens system" between the lens & the cornea.

◊ Indications:

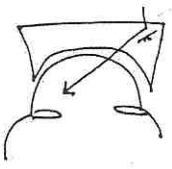
1- Optical : to correct errors that can be hardly corrected by glasses as:
- High errors.
- Unilateral aphakia.
- Irregular astigmatism.→ hard CL.

2- Therapeutic : for
- Resistant ulcers
- Neuroparalytic keratitis.
- Keratitis metaherpetica.
- Trichiasis - Lagophthalmos.

3- Cosmetic : colored CL for - Corneal opacities &-Albinism.

4- Occupational. الرياضين

5- Diagnostics : e.g. Gonioscopy to see the angle of the eye.



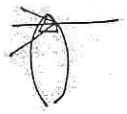
◊ Types:

1- Hard (PMMA) : for keratoconus.
2- Rigid gas permeable (silicone/Acrylate =S/A) : for astigmatism .
3- Soft (HEMA): for spherical errors.

◊ Advantages:

1- Larger field: No frame + move with the eye
2- No Aniseikonia → No binocular diplopia.

3- Less prismatic effect by the lens periphery.



4- Cosmetically better.

5- Avoid aberrations which result from prismatic effect of lens periphery.

◊ **Disadvantages**:

1- Intolerance by some (>with hard) &

Allergy by others esp. with soft C.L.→ Giant papillary conjunctivitis

2- Corneal abrasions or keratitis.

3- CL deposits (tear film من ال) if not properly cleaned.

→ FB sensation & nucleus for infection.

4- Corneal edema & vascularization : due to ↓ O_2 supply to cornea

(now there is gas permeable CL).

5- Special care for cleanliness and storage.

Retinoscopy (skiascopy)

See atlas page (160) عمل النظارة

***Definition:** it is a method for estimation of refraction of eye.

***Instruments:** 1- Light source and plane mirror فيها خرم or electric retinoscope.

2- Trial frame.

3- Lenses of different powers.

***Technique:**

1- Cycloplegia: if the age less than 40 yrs.

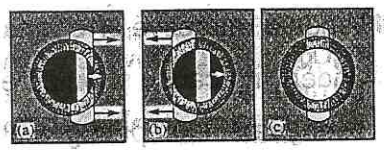
- Children: Atropine ointment 1% tds/3 days (strong ciliary ms. needs atropine).
- Above 10 ys: homatropine 1% or Mydriacyl 1 %.

2- The patient sits in a dark room at a distance of one meter from the examiner.

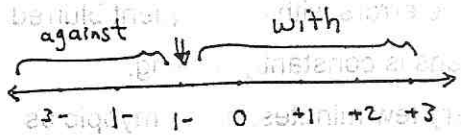
3- The observer reflects the light into the patient pupil to see the red reflex. Then, the mirror is moved and the movement of the red reflex is noted.

4- The movement of the R.R. may be one out of three:

- Against (to the opposite direction to that of mirror) → **myopia > -1 D.**
- No movement → **myopia - 1 D.**
- With movement (to the same direction as the mirror): may
  - **Myopia < -1 Diopter.**
  - **Emmetrope.**
  - **Hypermetropia.**



Movement of the red reflex(shadow) with the movement of the streak retinoscope(streak of light in the pupil) in the vertical meridian:
(a) With movement (H, E or M of less than -1D).
(b) Against movement (M of more than -1D).
(c) No movement (M of -1D).



5- If R.R. moves with add plus lenses, and if moves against add minus lenses

in ascending fashion till the movement of RR stops → **neutral point.**

6- Astigmatism is diagnosed when one meridian is corrected while the other is still moving. The power of the lens which corrects the first meridian is noted, (and more lenses are used till the second is neutralized).

7- **Prescription is given to the patient:**

Add -1 D : for the distance of one meter (if the distance is 50 cm add -2)

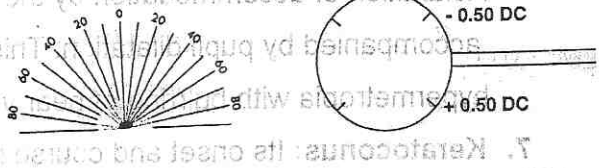
-1 D : if a cycloplegic is used. (for the tone of ciliary ms)

neutral point + (-1) for 1 meter **+ (-1)** tone of ciliary ms

8- **Post mydriatic test:**

After few days give him :

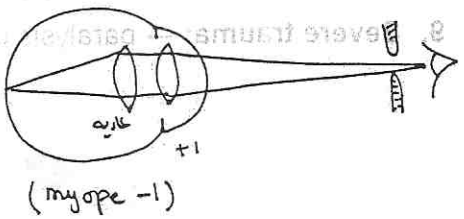
- for myopia the least minus lens that give the best vision
- for hypermetropia the highest plus lens that give the best vision
- check the axis of the cylinder using :
  - Cross cylinder or
  - Astigmatic fan



Astigmatic fan. Cross cylinder.

NB.

If use phenyl-ephrine ,it is mydriatic only not cycloplegic so not add -1 of the tone



Rapid changes in refractive errors

When a refractive error changes over a short period e.g: days or weeks, then an underlying cause should be always be sought. Refractive errors change only slowly over years, except in growing children, so a rapid change should always alert the doctor or optometrist.

Causes:

1. **SUbluxation of the lens:**
   This induces a rapid change in the refractive errors with consequent blurred vision. In a simple subluxation, when the lens is constantly moving, a change in refractive error may occur every few minutes, being myopic as the lens moves backwards and hypermetropics as the lens moves forward.

N.B: Tumors of the iris or ciliary body may cause displacement of the lens, giving similar changes.

2. **Ocular swelling:** e.g., meibomian cyst or neoplasm or chalazion in the eyelid or in the orbit, will induce astigmatism because of distortion of the cornea.
3. **Senile nuclear cataract:** May lead to progressive myopia due to rapid change (increase) of refractive index of the lens.
4. **Diabetes mellitus:**
   An uncontrolled diabetic may becomes rapidly high myopic over a few weeks because of increased blood sugar level which leads to change in the aqueous sugar ,treatment will cause hypermetropia.
5. **Miotic eye drops:** the spasm of accommodation produced by miotics e.g.: pilocarpine, induce an artificial myopia.
6. **mydriatic, cycloplegic eye drops:**
   Relaxation of accommodation by the use of cycloplegic eye drops will be accompanied by pupil dilatation. This will induce a state of increased hypermetropia with bulrring of near vision.
7. **Keratoconus:** Its onset and course may be rapid ----- increase in myopia ,and astigmatism over short period occurs.
8. **Mild trauma:** Will irritate the ciliary ms→ Myopia.
9. **Severe trauma:** → paralysis of ciliary ms → presbyopia.

Binocular vision

لا تولد بل
كيف تتعلمها مع الوقت

* **Definition:** it is the ability to see single image of an object using both eyes.

* **Grades:**

1- Simultaneous perception (3-6months):

it means stimulation of corresponding points on the retina.

2- Fusion (after 6months):

the ability to fuse 2 images with control i.e., fusing 2 similar objects with missing details in each → one complete image.

3- Depth perception (stereopsis) (6 yrs).

* **Values:**

1- Stereopsis (depth perception).

2- Binocular visual field is larger than uniocular.

3- Optical defects and field defects (scotoma) in one eye are masked by the other overlapping field.

***Assessment:**

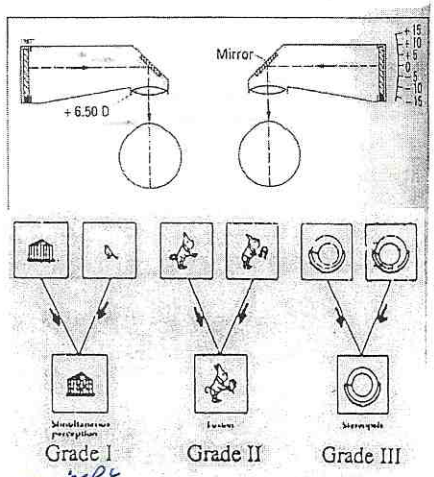
1- Synoptophore(major amblyscope)

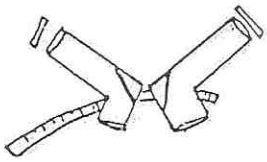
See atlas page (169)

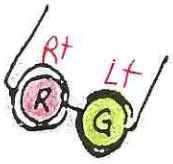
Grade I: If the 2 test objects are superimposed,

Grade II: If 2 incomplete test objects → one complete image.

Grade III: It is the ability to perceive the depth when viewing 2 slightly dissimilar objects.





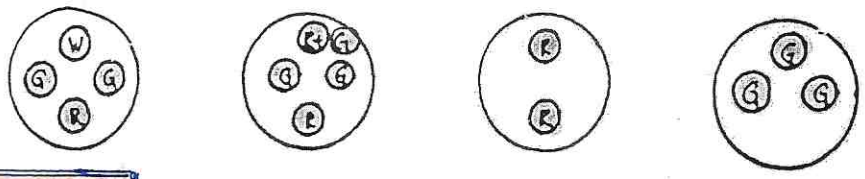


2- Worth's 4 dots test: See atlas page (170)

- Ask the patient to wear red-green goggles (red infront of right eye) & sits at distance 6 meters .
- Then, ask him to look to the 4 colored illuminated dots (one red, one white & 2 green).

Squint

- If the patient sees (possibilities ?):
  - 4 dots → Good binocular vision.
  - 5 dots → Diplopia.
  - 2 red dots → left eye suppression .
  - 3 green dots → right eye suppression.
  - 4 dots & the eye is squinting → ARC. !!



◊ Projection : it's to see opposite the stimulated retina

1+1 = 1 → normal.

1+1 = 2 → Diplopia (due to stimulation of uncorresponding points)

ايه الفرق ؟؟ شفوى

- **Light perception** → test the center of the retina (V/A).
- **Light projection** → test the periphery of the retina (field).

◊ Eye movements:

Monocular eye movement:

- Duction : one eye moves in (adduction) or out (abduction).

Binocular eye movement:

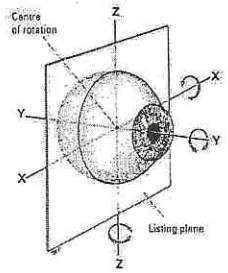
- Version : Movements of the 2 eyes to the right (dextro-version) .
  Movements of the 2 eyes to the left (levo-version)
- Vergence: movement of the 2 eyes in (convergence).
  movement of the 2 eyes out (divergence).

The extra-ocular muscles

There are 6 EOMs (4 recti and 2 obliques) which rotate the eye around:
3 axes of Fick →

4+ 2 = 6

Squint



(i) **Vertical axis** -> adduction and abduction.

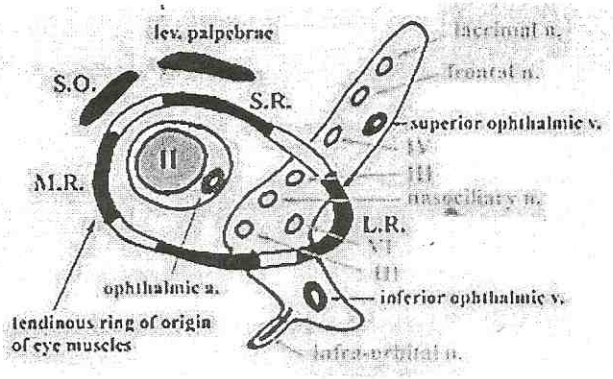
(ii) **Horizontal axis** -> elevation and depression.

(iii) **Antero-posterior axis** -> intorsion and extorsion.

NB. The center of rotation is 12-13 mm behind the cornea or 10 mm infront of retina.

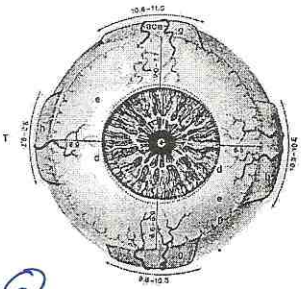
Recti muscles

attached to sclera
Anterior to equator



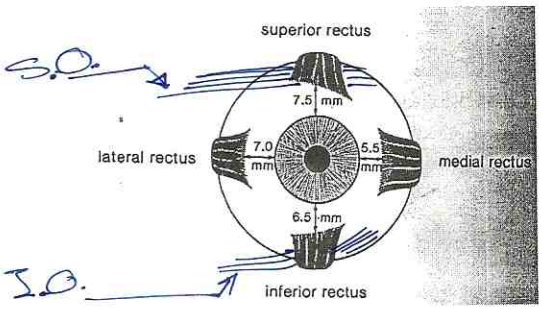
- **Origin:** arise from a fibrous ring placed around the op. Foramen → annulus of Zinn.

(SR &MR takes additional origin from meninges of the optic nerve, So painful eye movement on looking "up & in" & tenderness on pressure on SR in acute retrobulbar neuritis).



- **Insertion** to the sclera anterior to the equator at a variable distances from the limbus by a flat tendon 10 mm in dreadth:

(MR 5.5, IR 6.5, LR 7 , SR 7.7 mm from the limbus respectively).



Oblique muscles

Origin:

- **Superior oblique:** from the orbital apex above and medial to the annulus of Zinn. (Body of Spheniod), this is the anatomical origin, but the functional origin is trochlea.

- **Inferior oblique:** from shallow depression or roughness at the orb. floor lateral to the opening of NLD. (it's the only ms that originate anteriorly infront of eye ball & it's attached at its origin also to the fascia of the lacrimal sac.).

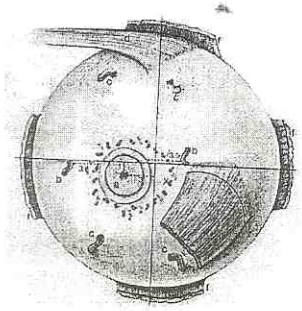
Squint

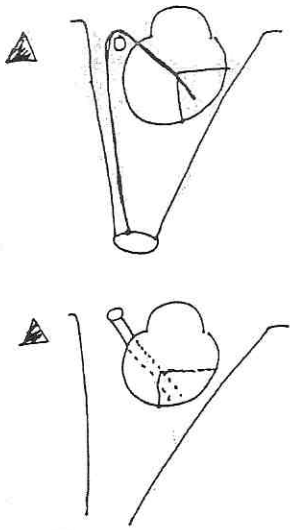
Insertion:

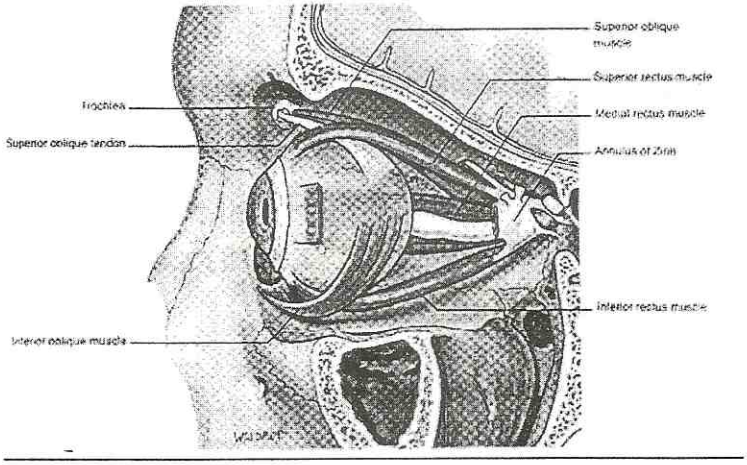
S.O: the ms passes forwards and medially then become tendinous and pass through U-shaped fibrous ring (cartilage) at the upper antero-medial part of the orbital roof ,4 mm from the orbital margin "trochlea" ,
to be reflected backwards and laterally under the SR ms. to be inserted in the sclera in the **upper postero-lateral quadrant.**

NB. Trochlear angle = 55°

I.O.: the ms passes backwards and laterally below the globe & below IR to be inserted in the sclera in the **lower postero-lateral quadrant,**
(5 mm from op. nerve almost over the macula).
(nasal end of the line of insertion is 2.2 mm from the macula)







- **Nerve supply:**
  - 3rt n. (Occulomotor): - Upper division → SR (+ levator) ms.
    - Lower division→ MR , IR , Inf. oblique
    - IOMS : sphincter pupillae ms & ciliary ms.
  - 4th n. (Trochlear): superior oblique. (SO4).
  - 6th n. (Abducent): lateral rectus. (LR6).

- **Blood supply:**
  Muscular branches of ophthalmic artery (2 for each ms. except the LR 1 branch, so the LR is more affected in DM).

Squint

• **Action** : when the eye in the 1ry position

MR: • Adduction

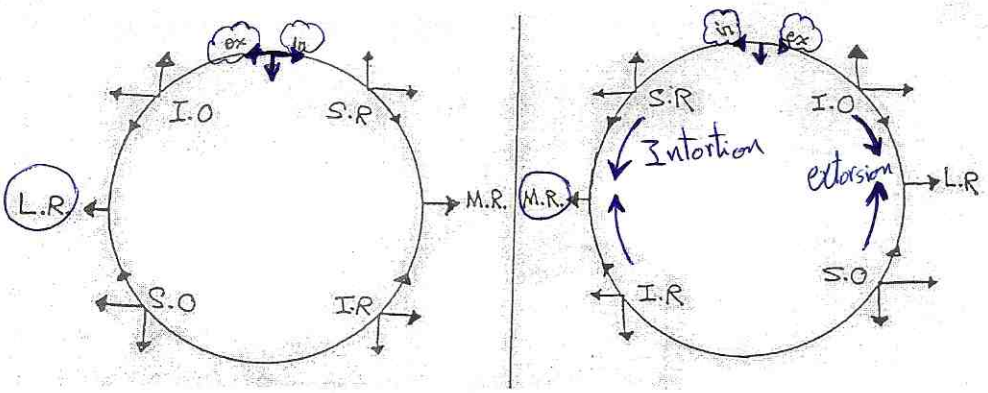
LR: • Abduction

SR: - Elevation (primary action)
- Adduction & intorsion (subsidiary action)

IR: * Depression (primary action)
* Adduction & extorsion (subsidiary action)

SO: ○ Intorsion (primary action)
○ Depression & abduction (subsidiary action)

IO: - Extorsion(primary action)
- Elevation & abduction(subsidiary action)



superior = intorsion
Inferior = extorsion

* Superior muscles are intorters while inferior muscles are extorters.
* Recti are adductors while obliques are abductors.
* Elevators : SR & IO.
* Depressors : IR & SO.
* **Sherrington's Law of reciprocal innervation (for the same eye):**
Contraction of ms is accompanied by relaxation of the antagonistic ms
e.g. LR & MR (of the same eye).
* **Herring's law of equal innervation:**
There is equal & simultaneous innervation of synergestic (Yoke) Muscles e.g Rt LR & Lt MR,
(voluntary movement only يطبق في ال Herring's law)

Squint

The muscle axis:

It is the line between the origin and insertion

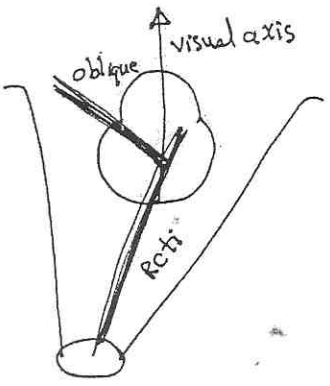
* **The muscle action is:**

→ Maximum: if ms axis is parallel to the visual axis.

→ Nil: if ms axis is perpendicular to the visual axis.

So, in extreme abducted position the only elevators & depressors are the recti,

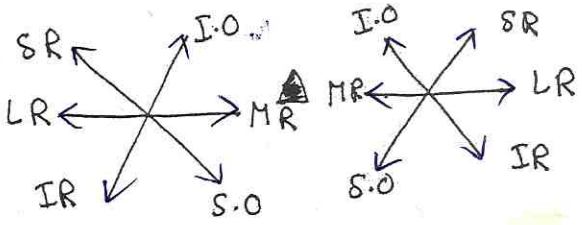
Because in this position the 2 obliques are' perpendicular to the visual axis & vice versa.



6th cardinal directions :

Each ms. is tested for its main action in one of 6 cardinal positions by asking the pt to follow the examiner finger in that positions:

1- Out → LR
2- In → MR
3- Out & up→ SR
4 - Out & down→ IR
5- In & up → IO
6- In & down→ SO



NB. some add 3 positions to be 9 cardinal directions - pure elevation.
- Pure depression.
- 1ry position.

So they are 6 → uniocular &
9 → Binocular.

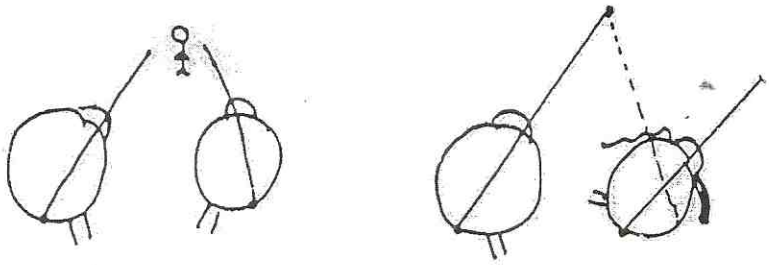
↓Not Cardinal because more than one muscle work at this movement

Squint

at min 26 Revision

Definition:

A condition in which the visual axes of the 2 eyes are "not" directed to the same Object = Deviation of the visual axis .انحراف



Types:

Apparent / True → Manifest / Latent → Incomitant / Concomitant → Paralytic / restrictive fibrosis → forced duction test

1- Apparent =false = Pseudo squint ظاهري غير حقيقي

2- True squint:

- **Latent squint (Heterophoria)** كامن.
- **Manifest squint(Heterotropia)** ظاهر:

i- Incomitant : (paralytic or restrictive):

to differentiate do forced duction test

e.g. Convergent squint may be - paralysis of LR (تتحرك ص ع) ← ①

or - fibrosis of MR (تتحرك ص ع) ← ②

ii- Concomitant (non paralytic)

- Accommodative (يتصلح كلياً بالنظاره) See atlas page (166)
- Non accommodative (لا يتحسن بالنظاره محتاج جراحة)
  - → Unilateral
  - → Alternating
- Partially accommodative

(يتصلح جزئياً بالنظاره والزاويه الباقية محتاجه جراحة)

iii- Kinetic strabismus.

N.B.

Nodal point: It is a point just ant. to the posterior pole of the lens through which light rays undergo no refraction & must reach the fovea.

((it is the optical center of the dioptric system of the eye))

((optic & visual center intersect at the nodal point))

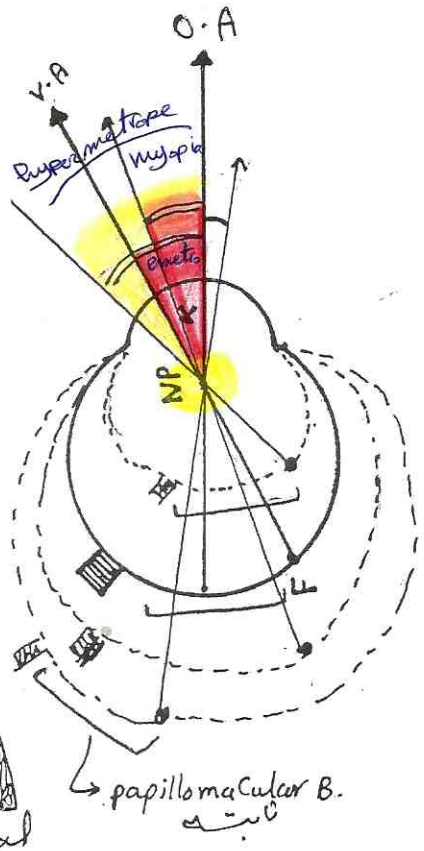
Visual axis: It is the line connecting the macula and object of regard ,passing through the nodal point.

Optic axis: المنصف It is the line connecting the center of the cornea, lens, retina.

Angle Alpha (Kappa) :

It is the angle between the visual axis and optic axis.

- **In emmetropes** → 5° and + ve (visual axis cut the cornea nasal to optic axis)
- **In Hypermetropes** → It is large +ve. → Apparent divergent squint (exo)
- **In high myopia** → It is small or even -ve. → Apparent convergent squint. (eso)



Apparent (false) squint

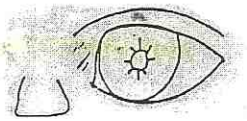
حول ظاهري غير حقيقي

★ Definition:

A condition in which the person appears to have squint although the 2 visual axes are directed to the same object.

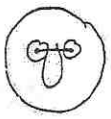
★ Etiology:

1- Apparent convergent squint (Pseudo-esotropia):



a- Epicanthus: a skin fold over the medial canthus

(congenital, racial دول حنوب شرق اسيا) See atlas page (171)



b- High myopia: due to -ve angle alpha.

c- Small IPD : orbits are close together (normal IPD : 55-70 mm).

interpupilary distance

2- Apparent divergent squint (Pseudo-exotropia):

a- Lateral ankyloblepharon: adhesion between the lid margins,

See atlas page (27)

b- High hypermetropia: due to large +ve angle alpha .

c- Large IPD : hypertelorism العملقه or Negroes →

(wide distance between the 2 orbits).

See atlas page (171)

3- Apparent vertical squint(Pseudo-hyper or hypotropia):

a- Ptosis → Pseudo-hypertropia.

b- Lid retraction→ Pseudo-hypotropia.

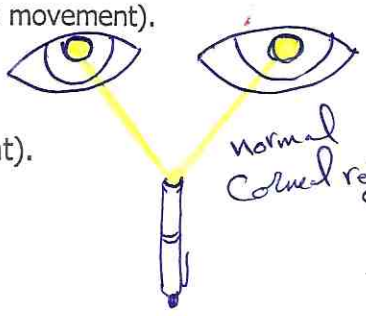
★ Diagnosis:

Normal زي

1. Presence of a cause.
2. Cover test → no movement as the visual axes are normal.

(we cover one eye & ask pt to fix the figer by the other eye → no movement).

3. Corneal light reflex: normally centered. See atlas page (171)



★ Treatment: Treatment of the cause only (as there is no squint).

Latent squint (heterophoria)

الحول الكامن

◊ Definition:

A condition in which the eye has a tendency to deviate due to EOM imbalance, "but" this tendency is checked by the brain **subconsciously** to maintain "binocular vision" till the patient is fatigued or the brain loses interest in binocular vision→ eye will deviates again.

◊ PPF: - General fatigue. - Mental fatigue. - Ocular fatigue.

◊ Etiology:

EOM imbalance due to:

1- Uncorrected Errors of refraction:

a- In Hypermetropia: the patient uses excessive accommodation to see clearly, leading to excessive convergence and increased MR ms strength →latent convergent squint (Esophoria).

b- In Myopia: : the patient relaxes his accommodation, leading to lack of convergence and decreased MR ms strength →Exophoria.

2- Mild weakness of one of EOM: not sufficient to produce paralytic squint. (Weakness → مش paralysis).

◊ Types:

1- Exophoria :the eye tends to deviate outwards (LR>MR).

2- Esophoria :the eye tends to deviate inwards (MR >LR).

3- Hyper phoria :the eye tends to deviate upwards (SR> IR).

4- Hypophoria: the eye tends to deviate downwards (IR>SR).

5- Excyclophoria : the eye tends to roll out (IO > SR).

6- Incyclophoria : the eye tends to roll in (SR> IO).

Squint

◊ Symptoms:

1- Compensated cases: show no symptoms (most cases).

2- Decompensated cases: may show:

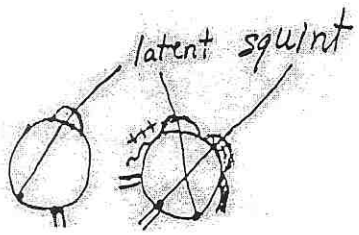
a- Symptoms due to trial to maintain binocular vision → muscular athenopia (eye strain) due to spasm of EOMs After prolonged close work:

- Red eye - Ocular pain. - Headache. - Lacrimation.
- Recurrent styes & blepharitis.

b- Symptoms due to failure to maintain Binocular vision :

General , mental or ocular fatigue. or the brain loses interest in binocular vision يسرح → eye will deviates again →

* Running letters :
  - الكلام في نفس السطر دخل في بعضه → Horizontal deviation.
  - السطور بتركب علي بعض → Vertical deviation .
* Latent → manifest.
* Intermittent diplopia.



◊ Diagnosis:

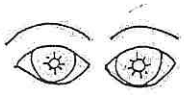
Cover-uncover test
Maddox rod
Maddox wing

Principle: depends on abolishing the B.V. by:

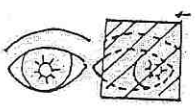
- covering one eye,
- or presenting different images to each eye, so, the brain will lose interest of B.V. and the latent squint will become → manifest squint.

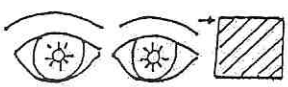
"Phoria → Tropia".

1- Cover-uncover test :



- Ask the patient to fix an object (tourch).
- Cover then uncover one eye.
- If this eye deviates under the cover (علي راحتها تحت الغطاء) then correct its position with uncovering to get B.V. again → latent squint. (هتشوفها وهي بترجع مكانها)





A- in esophria : the covered eye deviates in, under the cover

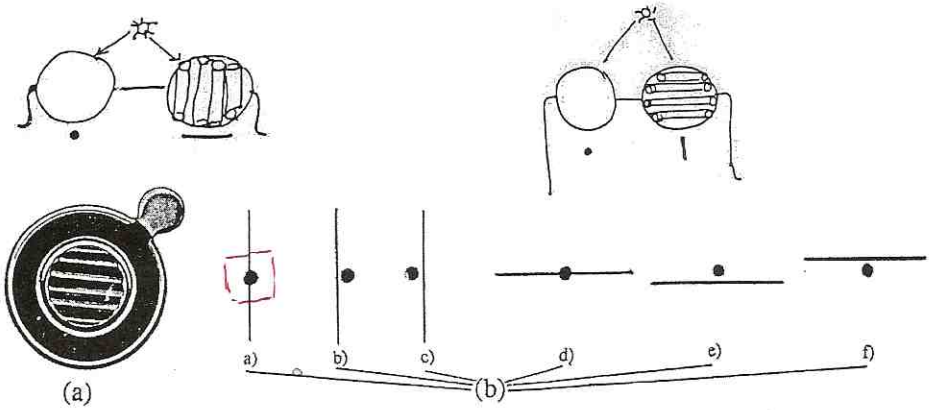
B- in exophoria : the covered eye deviates out, under the cover

((**NB. Alternate cover test:** measure the phoria + tropia))

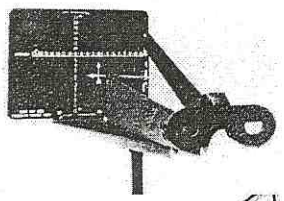
Squint

2- Maddox Rod (for far) See atlas page (169,170)

- Maddox rod is a group of red cylinders (4-5) arranged parallel to each other.
- They produce a red line image from a point source of light (This red line is perpendicular to the axis of the cylinders).
- The rod is placed in the trial frame infront of one eye and the other uncovered,
- patient fixates a source of light at a distance of 6 meters in a dark room.
- An orthophoric (normal) sees the point of light coinciding with the middle of the red line.
- If the red line is seen on either side of the light spot = Heterophoria.



Maddox rod test: (a) Maddox rod.
(b)Patient's view with the Maddox rod before the right eye: *a)Horizontal orthophoria; b)Exophoria;c)Esophoria; d) Vertical orthophoria; e)Right hyperphoria; f)Right hypophoria.*



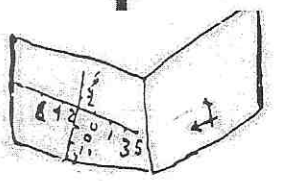
3- Maddox wing (for near) See atlas page (169)

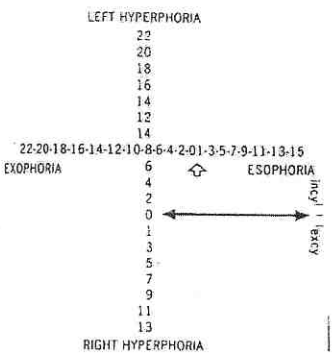
- The Rt. Eye sees 2 arrows (vertical & Horizontal).
- The Lt. Eye sees 2 scales (V. & H.).

An orthrophoric sees the arrows point to the → zero.

In Heterophoria the patient can read directly his angle of squint :

* If he reads odd فردي number → Convergent squint.
* If he reads even زوجي number → Divergent squint.





◊ Treatment:

1- Compensated cases: no symptoms → No. ttt.

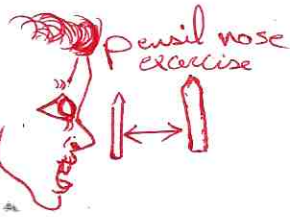
2- Decompensated cases:

i- Correct any error of refraction.

ii- Strengthen the weak ms. by:

a- Orthoptic exercises: esp. in Exophoria (convergence insufficiency):

Using - Synoptophore. - Pencil nose exercise .

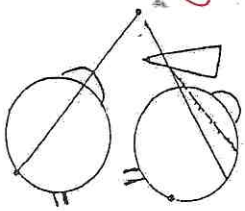


b- Exercising prisms:

increase muscular effort→ strength the ms

Disadvantages : headache so used in young pt.

(Base towards the Deviation)



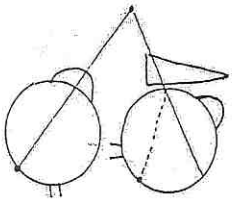
c- Surgery: if all the above failed (in large degrees) :

Strengthen the weak ms or weaken the overacting ms.

iii- Relieving prisms: * Disadvantages : latent → manifest.

* Used in old age & in vertical phoria.

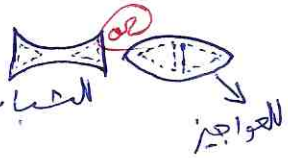
(Base of the prism is against the deviation.)



Decentration : ممكن نضيف Δ للنظاره بهذه الطريقه

P = D X H → amount of decentration

↓ prism



Paralytic squint

Manifest → paralytic / non paralytic

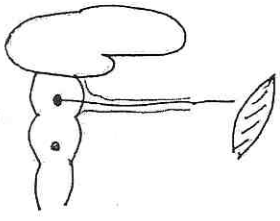
◊ Definition:

It is manifest squint due to paralysis of one or more of EOM. in which **the angle of squint is variable in different directions of gaze.**

◊ Etiology:

It is due to lesion in the muscle or its nerve supply

(Nuclear, nerve, or ms lesion = lower motor neuron lesion):



1- Nuclear lesions:

- Congenital: e.g. absence of the nucleus.
- Inflammatory: e.g. encephalitis.
- Vascular: e.g. thrombosis, embolism, cerebral hge

- Neoplastic : tumors of brain stem .
- Other ("toxic): as in diphtheria → toxic neuritis and with alcohol, lead.

2- Nerve lesions:

- Congenital: aplasia.
- Inflammatory: meningitis, cavernous sinus thrombosis والعصب ماشي فيها ينضرب, orbital cellulitis, **DM**, herpes zoster.
- Vascular: Hge (subdural, subarachnoid) .
- Neoplastic : Brain tumor (increased ICT),orbital tumor.
- Trauma : e.g, fracture base of the skull.

3- Muscle lesion:

- Congenital: e.g. maldevelopment of the ms.
- Trauma: Fracture orbital bones → contusion, hge in the ms. sheath.
- Neuro-muscular: Myopathy & Myasthenia gravis.
- Thyrotoxic Exophthalmos.
- Neoplastic : orbital tumors.
- Inflammatory: myositis.

NB. Supra-nuclear (cortical lesion): doesn't cause paralysis of the ms , but will lead to paralysis of conjugate movement (2 eye doesn't move at the same direction).

N.B.

* **Ophthalmoplegia** = paralysis or eye muscles:

1- External Ophthalmoplegia → paralysis of EOM.

2- Internal Ophthalmoplegia → paralysis of intraocular muscles.

2- Total Ophthalmoplegia → paralysis of EOM and intra OM.

*** Superior orbital fissure syndrome:** life give to see No insult at all

it includes affection of 3rd, 4th, 6th cr. Nerves →Total Ophthalmoplegia and 1st division of the 5th cranial nerve (lacrimal, frontal,nasociliary) → loss of corneal sensation

- **Orbital apex syndrome.** It includes SOF syndrome
- + Op.n affection (papilledema , neuritis or atrophy)

Squint

◊ Clinical picture

- Manifest squint
- Limitation of ocular movement
- diplopia
- false projection
- headache + nausea + vomiting

CHP = ocular torticollis

*All signs & symptoms occur in **same** the direction of action of the paralyzed ms, except ocular deviation (squint itself) which occurs on the **opposite** side:*

1- Manifest squint: to the "opposite" direction of the action of the paralyzed ms,

(the paralyzed ms. Loses its tone → the antagonistic draws the eye toward it).

e.g. in LR paralysis → the eye will deviate inwards by MR.

N.B.: The angle of squint: in paralytic squint (= angle between the deviated VA & the normal VA)

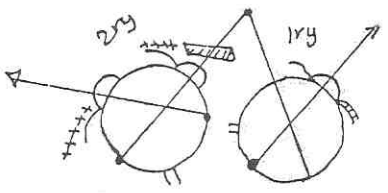
مهم جداً د. أحمد لطفي

The 2ry angle of deviation	>	the 1ry angle of deviation
(زاوية انحراف العين السليمة)		(زاوية انحراف العين البايظة)
(When the paralyzed eye is fixing)		(When the normal eye is fixing)

In Paralytic Squint But in non Paralytic the 2 angles are Same

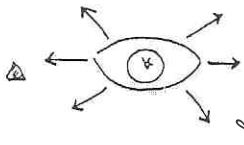
Explanation: (Herring Law)

- Normally, the impulses reaching the 2 eyes in order to look to any direction are equally distributed between the 2 eyes (Herring law).
- When the paralyzed eye is fixing (the normal eye is covered) → more impulses reach the paralyzed ms. to force it to contract. But, at the same time ,in the normal eye the synergistic ms. will receive equal amount of impulses (Herring law of equal innervation) Thus, the normal eye will move much more (under the cover) → larger 2ry angle.



2- Limitation of ocular movement:

- In the "same" direction of action of the paralyzed ms (not reach mid-line).
- Can be detected by doing the 6th cardinal directions.
- Do forced duction test to differentiate between paralytic or restrictive lesions (which occur due to fibrosis of the direct antagonistic ms).



* Paralytic Squint تابع *

Squint

3- Diplopia:

Cause: the image falls on non-corresponding retinal points.

- The image seen by the normal eye is clear (falls on the fovea) → **true image.**
- The image seen by the squinting eye is blurred (falls outside the fovea)→ **False image.**

Characters:

1- Binocular: disappears if one eye is covered.

2- More in the "same" direction of action of the paralyzed ms

3- It may be crossed or uncrossed.

4- diplopia doesn't present before the age of 6 years as the binocular vision is not fully developed so child can do suppression.

The patient fights diplopia by:

1- Suppression (child). — before age of 6

2- Covering one eye.

3- CHP.!!

* Compensatory head position

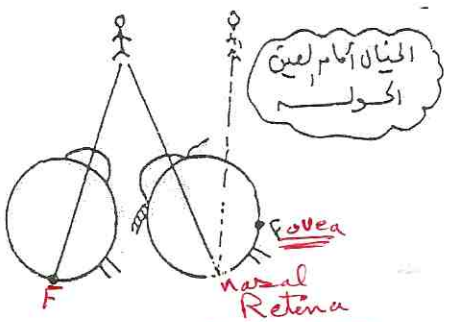
N.B

1- Uncrossed (Homonymous) diplopia: when the false image falls on the "same" side of the paralyzed eye e.g. in lat. R. paralysis.

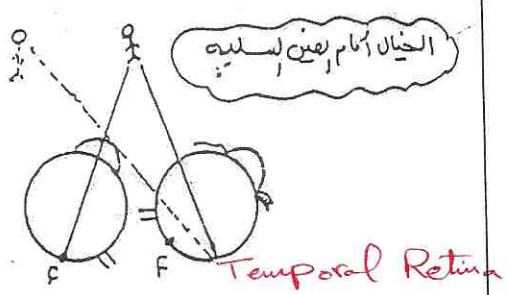
- Abductor paralysis (esotropia) → Uncrossed diplopia.

2- Crossed (Heteronymous) diplopia: when the false image falls on the "opposite" side of the paralyzed eye e.g. in Med. R. paralysis.

- Adductor paralysis (exotropia) → crossed diplopia.



* LR paralysis→ uncrossed d.
As the image fall on the nasal retina → Projected temporally.



* MR paralysis → crossed d.
as the image fall on the temporal retina → Projected nasally.

Squint

4- False projection (past pointing) ماسك الهوا بأديه:

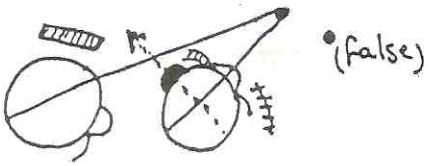
- It is false orientation of objects situated in the "same" direction of action of the paralyzed ms i.e. wrong estimation of the site (with the normal eye covered).

- Cause: the brain sends excessive impulses to the paralyzed ms. to force it to contract → the pt. points away in the same direction of action of paralyzed ms.

NB. projection occurs through :

1- Site of the image on the retina.

2- Effort produced to fix object (impulses كمية ال)

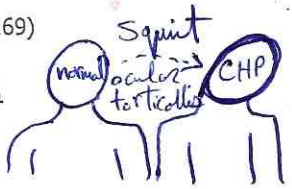


5- Headache. Nausea. Vertigo:

- Cause: the diplopia and the false projection.

Due to connection between 5th & vagus cr.nerves.

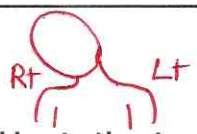
6- Compensatory head posture CHP (ocular torticollis): See atlas page (169)



- It is abnormal head posture adapted by the patient to minimize diplopia.
- It occurs in the "same direction of action of the paralyzed ms.

e.g, * In horizontal deviation → simple (face turn):

e.g. in right LR paralysis → face turn to the right.



(to send the diplopia behind him or to avoid looking to the rt side as diplopia is maximum at this site = mover the head instead of the eye)

* In vertical deviation → compound:
  - Face turn.
  - Chin elev. or depression.
  - Head tilt. See atlas page (169)

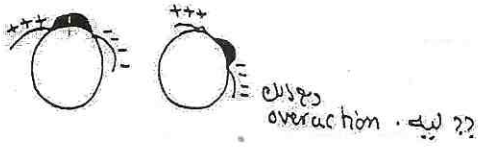
◊ Complications: → suppression = strabismic amblyopia
→ Ms. changes

1- Suppression: of the false blurred image (to avoid diplopia) .

في الأطفال → strabismic amblyopia (functional ↓ of vision) in long standing cases من عدم الاستخدام

Squint

2- Ms. Changes:



- Direct antagonist ms.
  - → Overaction > as its unopposed >>
  - → Contracture (on prolonged palsy). Inhibitory impulses رغم
    (Do forced duction test).
- Contralateral synergistic ms. → overaction due to stimulatory impulses .
- Contralateral antagonist ms. → underaction due to inhibitory impulses[2ry palsy].

NB. Dwain retraction syndrome : it's due to contraction of the 2 ms MR & LR
>> enophthalmos & narrowing of palbebral fissure.

◊ Diagnosis

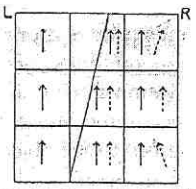
1- Neurological examination CT & MRI:

to diagnose where & what is the lesion.

2- Testing of ocular motility: ask the patient to fix your finger in the 6 cardinal directions to detect the paralyzed ms.

3- Diplopia chart:

- Aim: to determine the paralyzed ms.
- Method: 1) A red glass infront of the RT eye & a green glass infront of the LT eye of the pt. (to differentiate between the 2 images) in a dark room.
  2) A tourch with a stenopaeic slit is used to project a linear light.
  3) The pt. is asked about data to investigate diplopia:
     - Area & type of diplopia (crossed or uncrossed)
     - Relative position of the image
     - Distance between the 2 images.



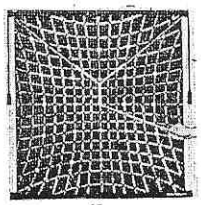
Diplopia chart for Rt. LR paralysis.

4- Hess screen: See atlas page (171)

* Aim : 1- To determine the degree of paralyzed ms.
2- To determine the secondary changes affecting the other muscles.

* Principle :

Special screen records the degree of false projection in different directions of gaze by dissociating the images of the 2 eyes with colored goggles:
(red & green glasses)


Hess screen.

Treatment

1- Treatment of the cause (by neurologist):

- Treat DM , hypertension .
- Give Vit B &vasodilators.

This may lead to complete recovery (spontaneous nerve regeneration).

- During that → cover the squinting eye (to avoid diplopia).

If you cover the normal eye this will lead to false projecton.

2- Surgical treatment:

Required if no recovery occurs after 6 months of ttt.

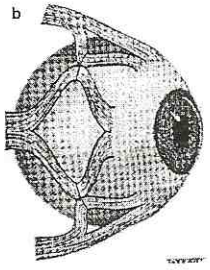
a- If the ms. is weak (the squinting eye crosses the mid-line i.e. ms. paresis)

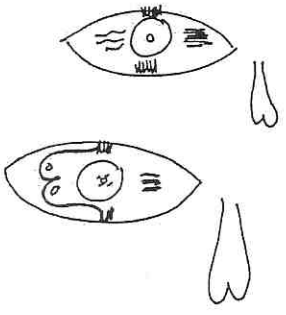
الحل→ strengthen the paretic ms by resection & weaken its direct antagonist by recession.

b- If the ms is completely paralyzed (cannot move to mid-line):

الحل⇨ **Ms transposition (Jensen operation) :**

- Slings from Sup. & inf. Recti are sutured with the LR.
- Used in 6th n. palsy only (LR paralysis), not used in 3rd n. palsy.





⇨ **Relieving prisms :** the base against the deviation (if the above measures failed)

3rd nerve palsy See atlas page (6,168,169)

1- Ptosis (due to levator paralysis).

2- Paralysis of EOM → deviation out (lat. R.). د احمد لطفي

(Down (SO) × الصح غلط → No depression by S.O. as the S.O. cannot depress abducted eye)

3- Limitation of elevation , depression & adduction.

4- Paralysis of accommodation (paralysis of ciliary ms).

5- No diplopia & no CHP(due to Ptosis), if the ptosed lid elevated → Crossed diplopia

6- Dilated fixed pupil (paralysis of constrictor ms) with loss of direct & indirect reaction.

7- Proptosis (mild) due to loss of tone of the paralyzed ms→ retrobulbar fat will push the eye forwards.

NB. 3rd nerve palsy may be: مهم جدا

- **Medical = Ischemic (DM)** → pupil normal (as the parasympathetic fibers of the pupil will not affected due to good blood supply from the pial plexus).
- **Surgical (mechanical) (tumor)** → pupil will be affected.
  (as the parasympathetic fibers of the pupil is superficial so easily affected).

4th nerve palsy: See atlas page (167):

1- Paralytic squint: the eye deviates up (hypertropia)
(IO → extorsion ,so SR → intorsion& elevation).

2- Limitation of depression on adduction.

3- Diplopia: uncorssed & increase on looking "down & in" with reading & eating (diplopia اسوأ)
S.O. is the ms of reading → depression on adduction
وهي العضلة بتاعت السلم (مهم شفوي)

4- Chin depression & tilting of head toward the opposite shoulder (ocular torticolis).

5- Bielschwoski sign: مهمه if the head is forcibly tilted to the side of the paralyzed eye, the affected eye will move upwards more.(increase hypertropia)

6- Nausea & vomiting ,on looking→ down &in

7- False projection: downwards.

6th nerve palsy: if the right side is affected See atlas page (167):

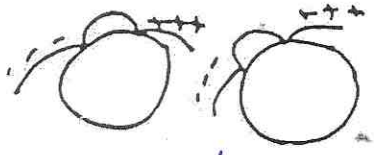
1- Rt. Convergent Squint.

2- Limitation of ocular Movement to the rt.(limitation of abduction)

3- Diplopia: uncrossed and increase on looking to the rt.

4 -False projection on looking to the rt.

5-Face turn to the rt.



6- Muscle changes:

- LT MR→overaction .
- RT MR →overaction due to contracture.=
- LT LR → underaction.

3rd nerve palsy → no diplopia → no CHP

Concomitant squint → No Paralysis

✷ Definition:

It is a manifest squint, not due to paralysis of EOMs, in which the angle of squint is the same in all directions of gaze(1ry angle = 2ry angle) so the movements of the covered & uncovered eyes are equal .

✷ Incidence:

- Age : 2-5 yrs.
- Type of deviation : usually convergent (stronger MR in children)
- Hereditary : present in 10% of cases.

✷ Etiology:

Binocular vision is fully developed between the ages of **6 months** and **6 years**. Any obstacle to binocular vision during this critical period would result in

→ ocular deviation ((EOMS imbalance with no binocular vision memnory)) .

Such obstacles might be:

1- Refractive: (Uncorrected error of Refraction)

- In Hypermetropia (more than 3D), the child accommodates to see clearly
  Accommodation is associated with convergence → Esotropia
  (concomitant convergent squint).
- In Myopia, relaxation of accommodation & lack of convergence → Exotropia
  (concomitant divergent squint).

2- Non-refractive:

I- Congenital: esotropia is more common than exotropia

- Peripheral (motor) obstacle: any anomaly of EOM e.g. LR hypertrophy.
  (العين محوله من ساعة الولاده)
- Central obstacle (defective development of presumed المفترض وجوده fusion center or visual pathway).

II- Sensory obstacle: due to monocular impaired vision. If the visual acuity in one eye is weak ,the brain will suppress it → unilateral squint,
e.g. unilateral congenital cataract, corneal opacity ,congenital ptosis, macular affection , anisometropia, retinoblastoma.
(العين مش شايفه من ساعة الولادة)

✳ Types:

1) Unilateral: the deviation always in one eye.
2) Alternating: the deviation is by either eye at different times.

Sequlae of concomitant squint:

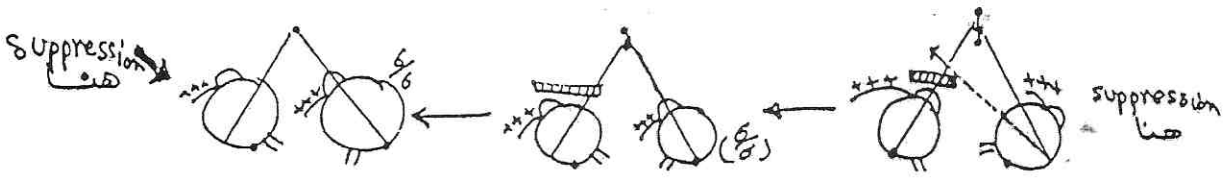
With the onset of squint, the images of the object will fall on non-corresponding points of the retina and binocular diplopia must occur. But, as the age of the patient is young, the brain finds a solution for this and diplopia doesn't persist. These solutions are:

1-Suppression:

- It is active neglect by the brain to the image seen by the squinting eye.
- It occurs in alternating aquint.

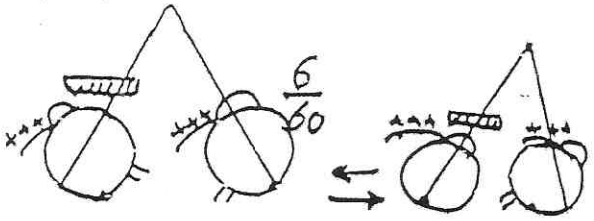
- On covering the fixing eye → the squinting eye will fix and with good V.A. (6/6) wih no suppression,
  → & the eye under the cover will deviate (by same angle)
- On removal of the cover → the new postion will remain,
  & suppression will be transmitted to the other eye.

So the suppression is <u>temporary</u> & <u>alternating</u> .



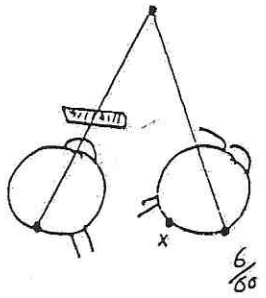
2- <u>Amblyopia ex-anopsia</u> خارج الاستعمال:

- It is decrease of V.A. in the squinting eye due to prolonged suppression.
- It occurs in unilateral squint.
- On covering the fixing eye → the squinting eye will fix and with bad V.A. (6/60)
- On removal of the cover → the original position will return.



3- <u>Eccentric fixation (ARC)</u>

- It occurs in some cases of amblyopia when the patient develops the ability to fix objects by a part of the retina other than the macula <u>(false macula).</u>
- It is due to dense suppression of the original macula.
- On covering the fixing eye → the squinting eye will not move {as it fix the object in the deviating position }
  Vision in this eye usually bad V.A. 6/60



Eccentric fixation & ARC ؟؟؟ اية الفرق

✳C/P:

1- Ocular deviation (with constant angle).
2- Defective vision in the squinting eye but:
   - No diplopia (as there is no binocular vision).
   - No limitation of movement.

Squint

✳Investigations (diagnosis):

A) History : Age of onset, duration, birth history .

B) Ocular examination:

- Pupillary reactions.
- Examination of the ocular media.
- Retinoscopy.
- Fundus examination.
- Visual acuity.

C) Examination of the EOMs:

1- Test the ocular motility:

In the 6 cardinal directions → to exclude paralytic squint.

2- Measure the visual acuity:

- If equal in both eyes → alternating squint, (pt. can fix by both eyes).
- Poor in the squinting eye → unilateral squint (Rt. Or Lt).

3- Cover test: See atlas page (172)

- Ask the patient to fix an object at a distance 50cm .
- Cover the fixing eye: - The squinting eye will fix.
  & - The fixing eye will deviate.
- Remove the cover: - If the new position remains → alternating Squint.
  - If the original position retuned → unilateral Squint.

NB. No movement on cover test:

1) Eccentric fixation.
2) Apparent (pseudo) squint.
3) Paralytic squint → test 6 cardinal direction. (ممكن تتحرك العين لحد الMid line)
4) Blind eye e.g. optic atrophy.
5) Uncooperative pt.
6) Orthophoria. 7) Marked ambylopia (V.A. less than 50 cm).

Squint

4- Measurement of the angle of squint:

1) Corneal light reflex (Hirschberg test):

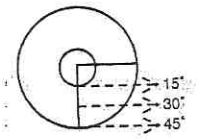
- Hold a tourch infront of the pt's nose (50 cm).
- Observe the light reflex on the cornea of the squinting eye.
- Normally, the light reflex is centered or slightly nasal in both pupils.
- It's Situated on the temporal side in convergent squint & on the nasal side in divergent squint.
- Its position indicates the angle of squint as follows :

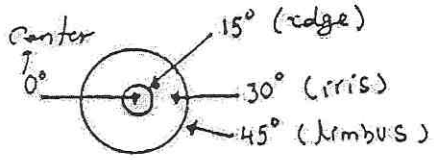
a) Angle 15° : if at the pupillary margin See atlas page (166,170).

b) Angle 30° : if mid way between pupillary margin & limbus (on the iris) See atlas page (166)

c) Angle 45° : if at the limbus See atlas page (168,170)

d) After this Add 7° for every 1 mm on the sclera (7 or 14 or 21)+ 45.



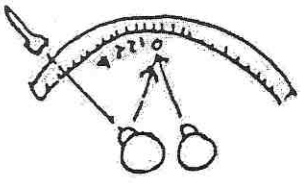


2) Synoptophore (major amblyoscope):

- The synoptophore is an instrument, composed of 2 tubes which can be moved on a graded scale.
- Ask the pt. to look through the tubes.
- Then, ask him to rotate the tubes until the 2 test objects (at the tube end) are superimposed (e.g. the bird inside the cage).
- The angle between the 2 tubes = angle of squint.

3) The perimeter:

A torch is moved on the arc until the light reflex is centered on the squinting eye.

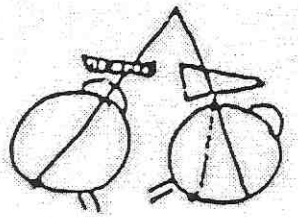


4) Maddox rod with tangent scale or with prism.

Squint

5) Prism - cover test:

- Prisms of increasing power are placed
- ★**infront of the squinting eye** with base against deviation , while covering the other (fixing eye).
- The angle = the prism power that abolish the movement of the squinting eye on cover test.
- The angle in degrees = 1/2 Δ (prism diopter) مثال **60Δ = 30°**



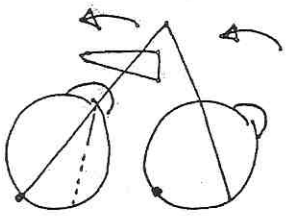
6) Prism test(Krimsky test): See atlas page (170)

{deeply amblyopic يستخدم لو العين الحوله}

- Prisms of increasing power are placed
- ★**infront of the fixing eye** with base against deviation.
- Until the squinting eye becomes straight & the corneal reflex is centered.

((نضعه أمام العين السليمة ف العين الحوله هتتحرك غصب عنها الي أن تتعدل))

- Angle of squint in degrees = 1/2 power of this prism.



prism test (krimsky test)

5- Assessment of binocular vision:

1- Synoptophore.

2- Worth's 4 dot test.

N.B. Concomitant convergent squint :

(1) accommodative : (late onset due to late development of accomm.)

A) Refractive :

* Due to uncorrected high hypermetrpia → excessive accommodation → excessive convergence (more than the capacity of fusional divergence amplitude)
* The angle equal in near & far.
* ttt : correct the hypermertropia with glasses .

B) Non refractive : AC/A = N- D

* Due to high AC/A ration : { - Convergence excess . - hypo accommodation.
* Angle is greater at near : because of additional accommodation required to maintain a clear image at near.

* TTT : bifocal glasses : ↓ accommodation at near so convergence will ↓

See atlas page (166).

(2) Partially accommodative :

The angle of squint is not fully corrected with glasses so surgery is needed to correct the residual angle.

(3) Non- accommodative: (early onset)

Cannot be corrected with glasses & surgery is necessary.

✳Treatment

Aim:

- To build and restore binocular vision.
- To improve and preserve visual acuity of squinting eye.
- To improve the cosmetic appearance.

Time: as early as possible (before age of 7 ys.) :

1- To avoid amblyopia.

2 - To allow binocular vision to develop.

Cases treated after age of 7 years are treated for cosmetic appearance only.

Lines of ttt:

1- Glasses: correction of error of refraction, giving full cycloplegic refraction to relax accommodation .

- Children below 3 yrs can't wear glasses , so if accommodative squint exists give: **1) Atropine oint.** → no accom. → no convergence → no squint (but interferes with vision).

2) Miotics (Eserine or pilocarpine) → peripheral accommodation with no convergence,this will correct the hypermetropia → no squint.

2- Suppression: Orthoptic ttt :

* Training of B.V. by synoptophore ساعتين ف اليوم, this need cooperative child> 4yrs & equal VA,

* The angle of squint will decrease gradual → Complete recovery, or

Residual angle→ surgery.

3- Amblyopia therapy: See atlas page (171)

by covering the sound eye part time occlusion

(one weak/year) to improve the vision of the amblyopic eye.

لو عندة 6 سنين اغطي عنيه 6أسابيع و ولازم الطفل يلعب في الفتره اللي عنيه متغطيه

د. سيد عرفه الولد كل شويه يشيل الغطاء بأديه تعمل ايه؟

4- Eccentric fixation:

- Occlusion of the squinting eye 3-4 weaks (to suppress eccentric point). then pleoptic ttt (foveal stimulation).

1- After image method :

it's to shield the fovea & temporary blind the parafoveal areas by strong light (using - Euthoscope or - Projectoscope) then pt looks. at white screen to get a negative after image.

(هيشوف الستارة كلها سوده ماعدا منطقة الfovea)

2- Direct foveal stimulation : the projectoscope is used to stimulate the fovea with strong light.

5- Surgical ttt :

Indications: i - Squint with no error (motor المشكلة) .

e.g. non-accommodative.

ii - Residual angle more than 10°

(after glasses and orthoptic training).

iii - Age > 7 years (for cosmetic appearance).

Vi – Most cases of exotropia.

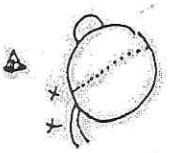
Technique:

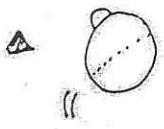
i- Weakening procedures:

1- Recession تراجع : carrying the insertion backward

2- Tenotomy (obsolete) now partial myotomy : Z-shaped.

3- Faden operation أحدث حاجة.



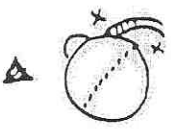


ii- Strengthening procedures:

1- Resection: shorten the ms. → increase its power.

2- Advancement:

carrying the insertion ant.→ stretch the ms

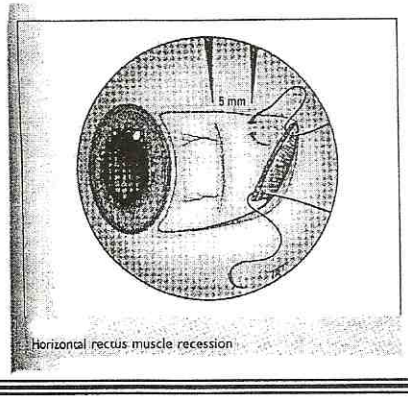


but cosmetically bad as the ms will appear in the palb. Fissure,
so use it in vertical squint only as the ms. will be covered by the lids.

3- Tucking: تني العضلة بدل ما نقصها

NB. If you need fine results: مهمه

Adjustable sutures are performed : it allow the muscle to move forward or backward during the immediate post-operative period (need cooperative pt).



Horizontal rectus muscle recession

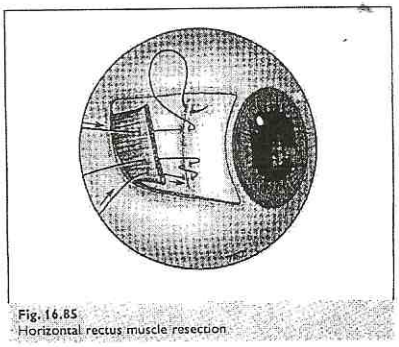


Fig. 16.85
Horizontal rectus muscle resection

*** Rules:**

- Every 1mm (recession or resection) of M.R corrects 3°.
  د. سيد عرفة الجمله دي ناقصه كلمة ??
- Every 1mm (recession or resection) of L.R corrects 1.5°.
- If angle of squint is large → operate on several ms :
  - 15° : oprerate on one ms.
  - 15°-30° : oprerate on 2 ms.
  - > 30° : oprerate on 3-4 ms.
- Maximum MR recession is 6 mm (to allow good near work) & LR resection is 10 mm.
- In alternating squint, symmetrical surgery is required.
- More the 3 ms >> Ant. segment ischemia.

*** Types of Operation :**

1) Unilateral strabismus : 1- Esotropia : a) MR recession.
b) LR resection.
2- Exotropia : a) MR resection.
b) LR recession.

2) Alternating Strabismus: 1- *Esotropia* : Bilateral MR recession.
2- *Exotropia* : Bilateral LR recession.

Squint

	Concomitant	paralytic
Oc. motility	free	limited
Angle of squint	1ry = 2ry	2ry> 1ry
Diplopia	Absent due to suppression	present
False projection, vomiting, CHP	Absent	present

Kinetic squint

في الزيادات

د. سيد عرفه الاختصارات دي تساوي ايه؟

ET = ??

ÉT = ??

XT = ??

XT = ??

Cover (screen) test:

◊ **Aim:** 1) To differentiate between apparent, latent and manifest strabismus.

2) To differentiate between paralytic and concomitant strabismus.

3) To differentiate between unilateral and alternating concomitant strabismus.

4) TO differentiate between convergent and divergent concomitant strabismus.

◊ **Methods:**

1. The patient looks to a fixation object with both eyes open.
2. The screen is shifted from one eye to the other and movements of the covered and uncovered eyes are observed .

◊ **Results:**

1) In concomitant strabiamus:

1. One eye is fixing (straight) and the other eye is squinting (deviated) to certain direction (in , out , up or down).
2. Covering the fixing eye results in movement of the squinting eye to take up fixation.
3. Remove the cover then observe:
   a) *Unilateral strabismus (with centric fixation):* the squinting eye deviates again and the fixing eye takes up fixation.
   b) *Alternative strabismus:* the squinting eye maintains fixation and the fixing eye remains deviated.
   c) *Esotropia (convergent strabismus):* the squinting eye moves outwards to take up fixation.
   d) *Exotropia (divergent strabismus):* the squinting eye move inwards to take up fixation.
4. Movement of the covered and uncovered eyes are equal, as the primary and secondary angles of deviation are equal.

N.B: On covering the fixing eye in unilateral strabismus with eccentric fixation, the deviating eye fixes the object without movement from its position (i.e., remains deviating and so the corneal reflex is not at the centre of the pupil).

2) In paralytic strabismus:

When the normal eye is coverd, it will move under the cover to a greater extent than the movement of the paralyzed eye (i.e., the secondary angle of deviation is much larger than the primary angle of deviation).

3) In latent strabismus: Only the eye under the cover will move and on removing the cover it will return to its original position.

4) In pseudo strabismus: Neither eye moves on shifting the cover from one eye to the other (negative cover test).

NB1: Causes of negative cover test (no movement). discussed

NB2: Value of the cover test:

(1) Diagnosis of strabismus and DD between its types.

(2) Diagnosis of eccentric fixation.

(3) DD between: 1) Refractive and muscular asthenopia.
2) Uniocular and binocular diplopia .

◊ How can you diagnose ambylopia?

(1) Visual acuity: poor vision in one eye.
- the acuity will be decreased when the symbols are presented close to each other rather than single or widely separated, (due to crowding phenomenon),

(2) Natural density filter: it decreases the VA of the normal eye markedly, where amblyopic eye will remain unchanged or improve slightly .

(3) Eye glasses or pin hole: vision will not improve.

(4) Worth 4-Dot test: 2 or 3 images .

(5) Synoptophore : only one image is seen.

(6) VEP : in children.

◊ **How can you diagnose eccentric fixation?**

(1) Cover test : the squinting eye will not move on covering the normal eye.

(2) Visuoscope: it is an ophthalmoscope having a small star interposed in the light beam and the patient is asked to fixate upon the star while exam. his fundus:

1-In centric (fovel) fixation: the examiner will observe the star on the fovea.

2-In eccentric (extrafoveal) fixation: the star is observed on point other than the fovea.

◊ **What are the uses of the synoptphore?**

(1) Diagnostic:

1. Measures angle of squint.
2. Determines grades of binocular vision.
3. Detects suppression.
4. Measures angle alpha.

(2) Orthoptic treatment: training exercises to restore binocular vision.

Kinetic squint

*** Definition:**

It is an apparent form of strabismus due to irregular action of EOMs. (not paralysis), caused by unequal stimulation of the nerves.

*** Aetiology :**

1) Irritative intracranial lesions : - Meningitis. - Brain tumors.

2) Spasmodic contracture : in the antagonist of a paretic ms.

*** C/P :** of the cause.

*** Treatment :**

1) Irritative intracranial lesions : treat the cause.

2) Spasmodic contracture : 1- Weakening op.
2- Inject Botulinum toxin in the affected ms.

Eye injuries

The protective mechanisms of the eye:

(1) Eye lids & lashes: as the hair follicles are supplied by nerve plexus with a very low threshold of excitation & also the lashes prevent FB from entrance into the eye.

(2) Tear fluid (Mechanical & bactericidal) Lysosymes

(3) Corneal sensation.

(4) Bony orbit.

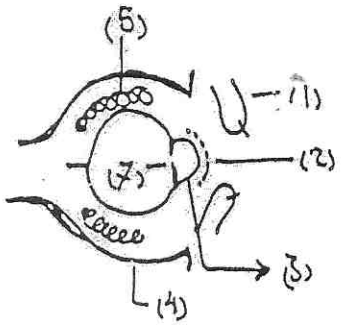
(5) The cushioning effect (وسادة) of the retrobulbar fat (Shock absorbing) .

(6) Bell's phenomenon.

(7) Constriction of the pupil on exposure to a strong light.

(8) Neck withdrawal reflex.

Anatomy ج

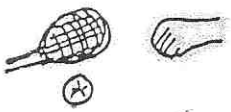


Types of ocular injuries:

1- Blunt 2- Perforating 3- F.B. 4- Radiation 5- Chemical 6- thermal

(I) Blunt Injuries

Cause: trauma by a small blunt object e.g. Fist or Tennis ball.



Mechanism of damage:

1- *Coup & countercoup:*

- Coup refers to local damage at the site of impact e.g. corneal abrasion
- While counter-coup refers to distant damage caused by shock waves that traverses the eye to the posterior pole e.g. commotio retinae.

Also, rebound of waves from the back of eye leads to more damage.

2- *Antero - posterior compression & Horizontal expansion:*

This leads to damage e.g. rupture globe & iridodialysis.

انفجار العين = separation of iris & ciliary body

Effect & ttt

(1) Orbit:

1- Traumatic proptosis:

Cause :

* Retro-bulbar hematoma.
* Peri-bulbar air (surgical emphysema): due to communication between the orbit and peri-orbital sinuses (especially Ethmoid).

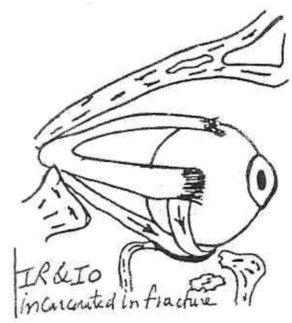
CLINICALLY lids are swollen & crepitations are felt by palpation, it dramatically increases with coughing & blowing the nose.

* Carotid-cavernous fistula (late)

2- Traumatic enophthalmos & hypophtalmos:

Cause:

The rise of intra-orbital pressure will lead to fracture of the orbital floor or medial wall **(blow out fracture)** See atlas page (176) with escape of the orbital fat & extra-ocular ms into the maxillary sinus. This may be associated with:



1- Limitation of up & down ward gaze due to IR & IO entrapment → Vertical Diplopia (Hess screen)
2- Loss of sensation of lower lid : due to affection of infra-orbital n.
3- Surgical emphysema of lids.
4- Enophthaloms & hypophthalmos.
5- DEATH ????

TTT: surgical repair with bone ships or metallic prostheses after the edema subsides.

3- Ophthalmoplegia.

N.B. RETROBULBAR HGE.:

- **Means** bleeding into the orbital space behind the globe & orbital septum.
- **Causes :** 1- Blunt or penetrating trauma.
  2- during retrobulbar anaesthesia.
  3- Spontaneous : with valsalva.
- **C/P:** 1- Pain , nausea , vomiting.
  2- Proptosis with subconjunctival hges.
  3- Restricted ocular motility & diplopia.
  4- Raised IOP.
  5- Reduced VA.
- **TTT:**
  * CONSERVATIVE : if no optic nerve compression or elevation of IOP:
    - Ice bag - head elevation - avoid anticoagulant & straining
  * URGENT INTERFERENCE .

(2) Lids:

1- Ecchymosis: Subcutaneous hematoma (Black eye):

Haematoma collects in the sub-cutaneous tissue as the skin of lid is loosly adherent to the underlying tissue.

TTT : - 1st 24 hours → Cold compresses (VC).
- After 24 hours → Hot compresses (help the absorption).
- Surgical evacuation of large ones to reduce the risk of skin necrosis or secondary infection. See atlas page (47,176)

2- Surgical emphysema : due to ethmoid or maxillary fracture.

3- Ptosis: See atlas page (7)

- Causes:
  (i) Mechanical: due to increased weight of the lid (hematoma).
  (ii) Paralytic : due to injury to the levator - 3 rd n.
- TTT: - Management of haematoma.
  - Wait for 6 months after the 1ry trauma to asses the function of levator & any residual damage is dealt with surgery.

4- Wounds: See atlas page (176,177)

*(i) **Vertical***: Gapping → heals by excessive scarring

→ cicatricial ectropion , lagophthalomos & epiphora.

*(ii) **Horizontal:*** Do not gape (as it is along the fibers of the orbicularis), and heals by less scarring.

TTT :

- Wash with saline or AB solution.
- Search for FB.
- Debridement of any necrotic tissue.
- Identification of land marks (lid margin – corners) should be sutured first.
- Suture in layers (1st suture at the gray line)

(3) Conjunctiva:

1- Wounds:

- If small (< 1 cm) → leave it (AB) .
- If large → Suture it.

2- Subconjunctival Hge: See atlas page (47)

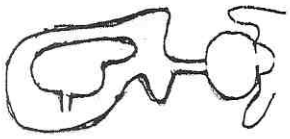
may be due to

i) Direct trauma to the eye → rupture of conj. BVs.

or ii) Trauma to the head (fracture base)

→ rupture of orbital or cerebral Bvs

(extra-dural hge) خطير.

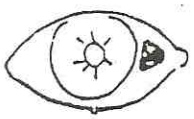
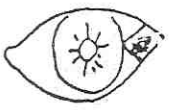


NB. No colour changes .

TTT : as black eye (ecchymosis).

Causes of subconjunctival hge:

1- Whooping cough
2- Blood diseases
3- Spontaneous
4- Bacterial or viral conjunctivitis due to toxic capillaritis .
5- Hypertension
6- Trauma .

	Ocular trauma	Head trauma
(1) Onset	Immediate	Delayed (24 h.)
(2) Conscious state	Not affected	Lost + vomiting
(3) Color	Bright red till it absorbed within 2-3wks due to its oxygenation from air.	Dark red (retained in the skull).
(4) Shape	Triangular with the base towards cornea.	Triangular (apex → cornea) or irregular.
(5) posterior limit	Seen (defined).	Not seen.
(6) Site	Usually temporal .	Usually in the lower fornix.
(7) Proptosis	Absent .	Present .
		

3- Chemosis : edema of the conj.

(4) Cornea

1- Abrasion (ulcer) : due to damage to the corneal epithelium.

TTT : قوله انت See atlas page (59)

2- Endothelial injury→ stromal edema.

***3- Wounds* = Rupture globe ± iris prolapse** see atlas page (92,175,176)

less common than scleral wounds (cornea is stronger).

& it's is common in eye with history or pervious surgery (keratoplasty, LASIK).

TTT: - Reposition of the prolapsed iris (if after 24 h → excision).

- Suture of the wound See atlas page (73)

(if small wound treat as perforated ulcer).

4- Blood staining of the cornea See atlas page (174)

- Cause: Hyphema + increased IOP that lead to endothelial damage.

- ***Clinical picture:***
  - The color of the cornea is first → reddish brown → then greenish yellow → then Grey → Clear.
  - The condition usually clears from the periphery → to the center by phagocytic action (over 2 years or more as the cornea is avascular)

- **ttt:**
  - Prevention: Control of IOP in cases of hyphema + ttt of hyphema.

واستني سنتين

  - Curative: PKP if Blood staining becomes Permanent.

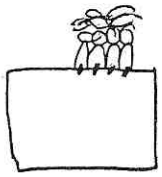
N.B. In infant PK علطول for fear of ambylopia.

5- Corneal Edema: due to endothelial & descemet membrane damage.

6- Recurrent corneal erosion:

* ***Cause***: erosion is caused by corneal scratches with e.g. finger nails.

While recurrence occurs with any slight trauma due to imperfect healing of basement membrane due to weak desmosomes.



(5) Sclera:

Laceration (Rupture Globe).

- **Rupture Globe :**

Site:

1- Cornea : less common as the cornea is stronger than the sclera.

2- Limbal: weak due to the presence of canal of Schlemm.

3- Sclera : Commonly up & in (about 3 mm form the limbus) Why?:
- Trauma: is common from down & out (less protected by the nose).
- Eye: is pushed against the trochlea of the SO ms

4- Site of insertion of EOMs.

Clinical Picture:

1- Vision : diminished.

2- Conjunctiva: wounds, chemosis or sub-conjunctival hge.

3- AC : Flat +/- hyphema.

4- Uveal tissue : Prolapse.

5- Lens: Traumatic cataract, subluxation, dislocation or even extrusion.

6- Vitreous :hge. 7-Retina : tears & RD . 8- IOP: hypotony.

NB. مهمه Posterior (occult rupture) : History of trauma – AC deep- hypotony

Complications:

- Infection (endophthalmitis).
- Intra - ocular Hge.
- Prolapse of intra - ocular contents → Iris, vitreous.
- Sympathetic ophthalmitis التهاب العين التعاطفي.
- Atrophia bulbi.

Treatment:

1- Immediate Patching & avoid direct pressure .

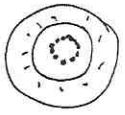
2- Medical ttt: - Systemic Antibiotic. - Anti-tetanic toxoid - Anti-emetic.

3- X ray is done to exclude the presence IOFB .

4- Surgery:

- Hopeless كباب حله (no PL vision) → Enucleation to avoid sympathetic ophth.
- Hopeful → Reposition or excision(after 24 h if its contaminated or necrotic) of the prolapsed tissues

(6) Lens:



(1) Subluxation.

(2) Subluxation or dislocation.

(3) Traumatic Cataract (+ Vossius ring). See atlas page (110)

(Rosette shaped: due to disruption of lens architecture at the cortical suture).

(VII) Anterior chamber:

(1) Ant. Dislocation→ Pupillary block glaucoma.

(2) Hyphema: See atlas page (174)

- **Source:** From lacerated iris & CB.
- **Fate:**
  - **Absorption through :** the iris crypts & the angle of the AC within 7 days
  - **Complications:** (i) 2ry glaucoma (angle closure).
    (ii) Blood staining of the cornea.
    (iii) Iridocylitis. (iv) rebleeding.

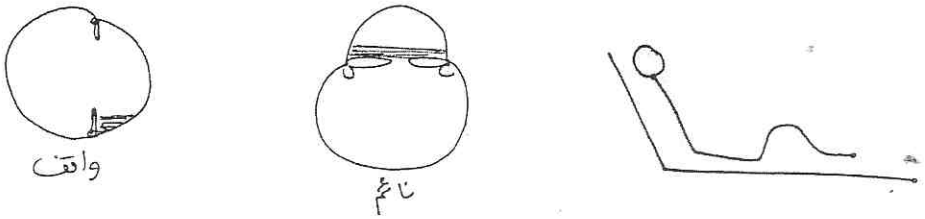
• **ttt:**

(i) Rest in bed (in a semi-sitting position = Folwer's position)

→↑ Absorption from the angle& iris crypts.

(ii) Bandage of both eyes حتي لا يتحرك كتير & to induce complete eye rest.

(iii) Daily monitoring of the IOP.ليه؟



(iv) Medical ttt:

- Local: Steroid (for iritis) &. B- blocker & diamox (control IOP).
  Streptokinase 50 000 unit intracameral
- General: - Aminocaproic acid (anti-fibrinolytic) and avoid NSAI drugs.
  - Vit C & Ambezym tab. or alphachymotrypsin IM.
  - Sedatives & laxatives : to avoid straining .

(iv) Surgery : for high IOP not responding to medical ttt.
(50 mmhg for 2 days or 35 mmhg for 7 days)
or clotted hyphema:
→ Evacuation of hyphema: Paracentesis or I/A.

NB. - No miotics → pupillary block & Synechia.
- No mydriatics → ↓ absorptive surface (angle & iris crypts) & may lead to angle closure.
- Miotics & mydriatics → iris movements→ loosen any formed clot → rebleeding

- Other causes of hyphema: 1- Trauma.
2- Inflammation (HSV-HZV- TB- Gonococci - DM).
3- Vascular (CRVO – DR).
4- Neoplastic: intraocular tumors.
5- Sytemic: bleeding tendency e.g. leukemia

(8) Pupil:

(1) Traumatic Miosis:

- It is a transient event in all contusions.
- It is due to mild trauma → irritation of 3 rd nerve fibers → spasm of both ms (the sphincter اقوي & dilator).
- Associated with spasm of Accommodation (transient myopia).

(2) Traumatic Mydriasis:

- it's preceded by miosis & may be transient or permanent.
- It is due to Severe trauma → damage of motor nerves (3rd nerve) → paralysis of both ms.
- Associated with paralysis of Accommodation (blurred near vision).

(3) Adie Pupil

(9) The iris

(1) Traumatic Irido – cyclitis.

(2) Pupillary (sphincteric) Laceration :

(very strong trauma → sudden mydriasis)

- V- shaped tear at the pupillary border See atlas page (175).

(Mydriatics are contra-indicated → may enlarge the tear)

NB. Tear can be sutured .

- It may be accompanied with:

(1) Anti -flexion

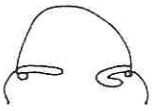
Torn part is rolled anterior &. pigment epith. Faces the ant. chamber.

(2) Retro - flexion

Torn part- is rolled backward between lens equator & ciliary body,

DD from iridectomy.

(3) Irido - donesis (Tremulous Iris):

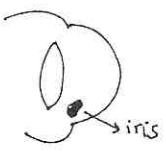


Occurs with → Subluxation - Dislocation of lens.

(4) Traumatic aniridia:

Here, complete avulsion of the iris at its root & the iris falls in the bottom of A.C. by gravity (as a black ball).

TTT: Colored CL. (recently iris prostheses)



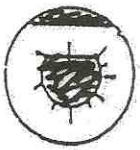
(5) Irido – dialysis: هام جداً See atlas page (95)

- **Definition:**

Blunt trauma to iris→ separation of iris root from C.B., This results from : 1- pressure in A.C. pushing iris backwards, the iris periphery being not supported by lens may separate.

2- Antero-posterior compression with horizontal expansion.

- **Symptoms: Uniocular diplopia except if:**
  - Up (covered by the lid) .
  - Very small.
  - Associated with cataract.
- **Signs:**
  - Pupil → D-shaped See atlas page (95)
  - Red reflex → double.
  - Dark area at the periphery.
  - Usually associated with Hyphema.



- **D.D.:** - from malignant melanoma of the iris (dark mass)
  → pupil distoted & RR is single See atlas page (100)
  - Peripheral iridectomy .
- **Complication:** traumatic flexion iris (retro or anti)
- **ttt:**
  - If no diplopia → atropine + cortisone (iridocyclitis).
  - If diplopia exists:
    1- Cover the defect (colored C.L.)
    2- Close the defect → suture the dialyzed iris to limbus.
    3- Iridectomy(to make one opening but may lead to photophobia).
    + TTT of iritis.

(10) Ciliary Body

(1) Hypotony: due to CB shock or Cyclo- dialysis .

(2) Glaucoma: due to CB laceration:

- Hyphema
- Healing by fibrosis closing the angle (angle recession glaucoma).

(3) Spasm of accommodation With temporary myopia.

(4) **Paralysis of accommodation** with blurring of near vision.

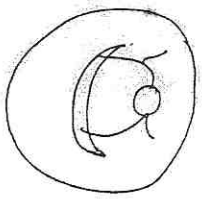
(5) **Intra-Ocular Hge :** Hyphema, vitreous Hge.

(11) Choroid :

(1) Choroidal Effusion or Hge→→RD.

(2) Choroidal Rupture:

- **Site:** Temporal to the disc.
- **Shape:** Crescentic with its concavity towards the disc.
- **Color:** early: red (hge).
  Late : White (showing the sclera).
- **Crossed** by the retinal vessels ,
  (Retina may be intact).
- **Fate:** Good if away from the fovea.
- **ttt :** Rest + Vit C (no specific ttt).



(3) Traumatic choroiditis.

(4) Choroidal detachement from hypotony.

(12) Vitreous

(1) Vitreous Hge.

(2) Vitreous Opacification: Musca volitans,

- Due to: (i) Hge (ii) Coagulated protein.

(3) Vitreous Loss : through a ruptured globe with retinal traction.

(13) Retina:

(1) Retinal Tears (Dialysis or giant tear) → RD.

(2) Traumatic macular hole: due to vitreous traction at its firm attachement to the macula

(3) Retinal hge: Intra-retinal, sub-hyaloid.

(4) Retinal edema (Commotio retinae = Berlin's edema).

(5) Retinal Detachment: may be:

i- Rhegmatogenous : due to retinal tear.

ii- Exudative : due to severe hypotony.

iii- Tractional : due to vitreous loss & incarceration in scleral wound

Commotio Retinae(Berlin's edema)

Definition : it is retinal edema following blunt trauma .

Cause : A countercoup to the posterior pole of the eye → compression of the posterior pole vessels & decompression → Ret. Edema (within few hours) (most marked in the central part).

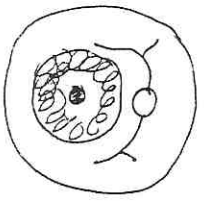
Onset : minutes to hours after the trauma

Clinical picture:

(i) Symptoms: Rapid drop of vision if the macula is involved (HM).

(ii) Signs:

- Pupil: slugish
- Fundus exam. → shows **Pseudo-cherry red spot** See atlas page (141) (± retinal hge).
- White color (due to edema collected mainly in the ganglion cell layer).
- The fovea (no gang. cells) → still show the color of the choroid.



Fate: (I) Spontaneous Resolution in few days if small amount
(2) Macular edema (cyst) which may rupture → macular hole.

D.D: From (i) Other causes of cherry red spot.
(ii) Other causes of rapid drop of vision.

TTT: Rest (+ systemic steroids).

د.سعيد شلبي What is the difference between the milky white color of CRAO & Commotio retinea?

(14)Optic Nerve:

(1) Avulsion of the optic nerve : due to extreme torsion or ant. displacement of the intracanlicualr part which is fixed to the canal unlike the intraorbital part.

(2) Optic nerve hge.

(3) Edema with hypotony.

(4) Op. atrophy (1ry): damage with fracture base.

(15) Extra-ocular muscles: Ophthalmoplegia.

(16) lacrimal system:

- laceration of canaliculus or the sac → epiphora.
- lacrimal gland displacement.

(17) IOP:

(1) Traumatic glaucoma : due to - Corneal ulcer - Iritis - Hyphema
- Angle recession glaucoma
- Ant. dislocation of lens
- Lens particle glaucoma (due capsule tear)

(2) Traumatic hypotonoy: due to CB shock , iridocyclitis or rupture globe.

(18) Refraction:

1- Myopia : due to spasm of ciliary ms.
2- Loss of accommodation : due to paralysis of ciliary ms.
3- Astigmatism : due to lens subluxation
4- Aphakia : due to post. dislocation.

(II) Perforating trauma

Causes: Trauma by sharp instruments (knife, scissors)

Effects

I- Mechanical effects:

1) Cut wounds in lid, cornea, sclera, lens capsule.
± Uveal prolapse or vitreous loss.
2) Traumatic cataract.

2- Infection: usually ends in Endo. Or even panophthalmitis.
Onset: 24-48 h (bacterial) or weeks (fungal) .

3- Sympathetic ophthalmitis.

Sympathetic Ophthalmitis

التهاب العين التعاطفي

♦ **Definition:**

It is Bilateral inflammation of the uveal tract following trauma to One eye in which part of the uveal tract is involved leading to marked diminution of vision.

- The traumatized eye is called **the exciting eye.**
- The other eye is called **the sympathizing eye.**

♦ **Incidence** - Bilateral
- Children.
- Rare with suppression (destructed uveal pigment).

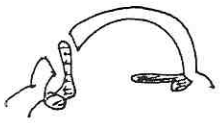
♦ **Etiology:**

* **Predisposing Factors:** **The incidence is increased with**

(i) Injury to the CB (dangerous zone) due to ↑↑↑ B.V.s & pigments.

(ii) Retained IOFB: as the F.B. will sensitize the immune system → exaggerated response.

(iii) Incarcerated uveal tissue in the wound.



* **Theories:**

(1) Allergic theory:

-The uveal pigments are normally isolated from the immune system.

- An ocular injury will liberate the uveal pigment (Ag) to the circulation.

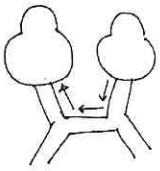
-The Antibodies formed against the uveal pigment will attack both eyes.

- This theory is the most accepted as:

i- The latent period is about 4-8 weeks which the average time needed by the immune system to synthesize antibodies.

ii- The patients have cutaneous hypersensitivity to the uveal pigments.

iii- Sym. Ophthalmitis is rare in endophthalmitis (pigment destroyed by pus).طب الحمد لله



(2) *Infective theory* غلط:

In which the organism (may be virus?? Change the code of uveal pigment → antigenic) reaches the exciting eye with the trauma & reach the other eye via Op. n. → chiasma → other op. n.

- This theory is not accepted as: no organism was isolated.

♦ Clinical picture:

• **Onset:** 4-8 weeks after the trauma (may be up to years)

• Prodromal picture: انذار

***Symptoms:**

- Pain, lacrimation, Photophobia & defective vision.
- Loss of accommodation (Indistinct near object).

***Signs:**

1- Signs of trauma in the exciting eye.

2- Signs of bilateral iridocyclitis with variable degrees of severity.

NB. Symptoms &S. are bilateral but start & more marked in the exciting eye, followed (days- weeks) by the sympathizing eye.

& better seen in the sympathizing eye (as it's quite eye not traumatized)

NB. Photophobia is the earliest symptom .

• Full picture:

The condition progresses into:

- Severe bilateral, pan-uveitis (corneal edema, muddy iris ,mutton fat KPs & Plastic pupillary membrane).
- Dalen Fuch's nodules.
- 2ry Glaucoma, Complicated cataract, RD due to cyclitic membrane or exudative RD
- Finally → Atrophia bulbi (bilateral).

♦ Investigations: CBC → eosinophilia .

♦ Treatment:

(1) Prophylactic ttt:

(i) If the injured eye is hopeless → do Enucleation

(ii) If the injured eye is hopeful → the followings are done:

- Excise the prolapsed tissues.
- Remove any IOFB.
- Proper suturing of the wound +Follow up.أهم حاجه

if : *1- the injuried eye resists to be quite down inspite of ttt.*

2- If the Prodromal symptoms & signs appear in the normal eye.

shift to curative ttt

(2) Curative ttt:

1- Give:

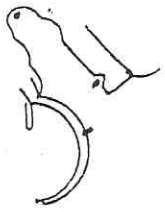
- Topical: Atropine & cortisone ,may be subconjunctival (iritis).
- General: Cortisone + NSAI + Diamox. Even Immunosuppressive drugs e.g. Methotrexate.

2- Enucleate the traumatized eye (target of immune system),if there is no response to medical ttt .(help to ameliorate the condition).

(III) Injuries by F.B.

Cause The foreign body may be: 1- Metallic: Iron, copper, lead.
2- Non-metallic: piece of glass, etc.

(I) Extra-ocular F.B.

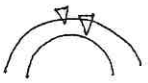


Commonest sites: *Cornea See atlas page (58) * Fornix
*Sulcus subtarsalis See atlas page (49)
* Lacrimal punctum

Complications: 1- Corneal ulcers.
2- Corneal opacities (If Bowman Membrane is damaged).

Treatment: ***Removal (under surface anesthesia)***:

- Sweep the corneal surface with a glass rod, over its tip is wrapped with a piece of cotton.
- If embedded → use a needle (or F.B. spud عتلة).



- **Iron FB with rust ring :** should be removed with scrubbing the surrounding epith., also the rust can be removed using Deferoxamine 5% drpos .

(II) Intra-Ocular F.B.

Effects:

(1) Mechanical effects: This depends on:

a) Route of entry (cornea or sclera).

b) Size (large F.B → more damage) .

c) Shape (ragged F.B → more-damage) . d) Velocity.

(2) Infection:

Not common → as many IOFB are sterile by the heat generated during their emission. زي طلقه الرصاصة

Sterile inflammation is common especially with pure Copper.

(3) Sympathetic Ophthalmitis. قولها

(4) Chemical effects: delayed and depend on the chemical nature:

- If the F.B is chemically inert (Glass) → it will be surrounded by fibrosis.وخلاص
- If chemically active (Iron) → Siderosis . - Copper → Chalcosis.

Siderosis Bulbi

Definition: It is the toxic effect of iron on the eye.

Etiology: (1) Iron IOFB .

(2) Intra - ocular Hge : due to breakdown of the hemoglobin with release of iron into the ocular cavity.

Mechanism:

Iron is oxidized (rust → ferrous أخطر & ferric oxide) & the rust separates from the F.B & dissolves in the tissue fluids & circulates inside the eye leading to:

1- Staining of ocular tissues with a rusty (brown - red) color.

2-Then, enters cells (especially the epithelial cells) producing toxic effect on the cellular protein & inactivates intracellular oxidative enzymes.

Clinical Picture:

Symptoms: - Impairment of vision. - Night blindness.

Signs:

- **Cornea:** krukenburg spindle (iron in endothelium).
- **Iris & C.B:** Atrophic changes (Mydriasis) & heterochromia (darker iris).
- **Pupil :** mydriasis (degeneration of sphincter pupillae ms.).
- **Deposited in the angle:** Rusty appearance → scarring of the TM → 2 ry glaucoma.
- **Lens:** Cataract (not true cataract but rusty discoloration).
- **Retina (rods):** Pseudo-retinitis Pigmentosa (night blindness).
- **Optic N.:** Consecutive op atrophy (→ blindness).

Localization and treatment: see later

Chalcosis Bulbi (Wilson's disease)
Hepato-lenticular degeneration

Definition: it is the toxic effect of copper on the eye (not pure copper<85%).

N.B. Pure copper: produces severe inflammation that simulates sterile endophthalmitis.

Mechanism:

Copper is oxidized into Copper oxide which separates from the F.B → dissolves in tissue fluids & circulate inside the eye leading to → staining of ocular tissues (especially collagen& basement membranes as DM & lens capsule) with a yellow – green color, with no atrophic changes.

Clinical Picture:

- **Symptoms:** impairment of vision (better than iron FB, less degenerative changes) .
- **Signs:** - Lens: Sun - flower cataract (Golden green & radiating like petals of flowers)
  - Cornea: Kayser - Flischer ring (golden or green brown ring at the corneal periphery) See atlas page (174)

لاحظ يصبغ الحاجات الشفافة

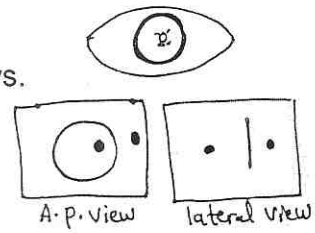
Localization and treatment: see later.

Diagnosis & Localization:

- **History.**
- **Slit lamp:** to detect (i) Route of entry. (ii) F.B in the AC or lens.
- **Gonioscopy:** to detect F.B hidden in the angle.
- **Ophthalmoscopy:** to detect F.B in the posterior segment (Retina, vitreous).
- **If the media is opaque:** Due to hyphema, Cataract, vitreous hge, use:

  (i) Plain X-ray.

  (ii) X-ray using metallic limbal ring (12 mm diameter) which is sutured to the limbus & used as indicator for localization :
  X- ray are taken in antero-posterior & lateral views.



  (iii) Ultrasonography:
  (provided that the globe is intact or after its repair)

  (iv) C.T scan.

  ((MRI not used in metallic FB مهمة جدا or metallic heart valves or peace makers)).

  NB. What are the contraindications of CT? د اشرف الدسوقي

Treatment:

1) Removal of the F.B. (Via the nearest way to minimize the damage):
- F.B in A.C or on the iris: Remove it through a limbal incision using forceps.
- F.B entangled in the iris → Do iridectomy including the FB.
- F.B in the lens → Do lens extraction.
- F.B in the posterior segment: Removed through pars plana incision using:
  1- Giant Magnet or
  2- Vitrectomy + Forceps extraction.
  3- + ttt of any associated damage e.g. retinal tears.

2) Local : Atropine & AB.

3) Systemic : AB & Anti-tetanic serum (IM).

4) Enucleation : in hopeless eye (NO PL) :to guard against sympathetic ophth.

(IV)Radiation (Physical) Injuries

(I) **Infra- red rays** :

* **Effect:** 1- Glass blower cataract : in glass workers.
2- Iron melter's cataract: in iron worker.
3- Eclipse blindness : macular burn due to exposure to sun-eclipse.
4- True exfoliation of the lens capsule .

(2) **X - ray:**

* **Effect** → Madarosis, cataract, retinopathy, op. neuropathy.

(3) **Ultra - violet rays**:

• **Effect** → photophthalmia See atlas page (74)

It is Kerato-conjunctivitis 2ry to exposure to short wave lengths,As in:
- Skiing (snow blindness) - Welding arcs.بتوع اللحام

• **C/P:** Pain, lacrimation, severe photophobia (SPK) & also may lead to iritis (occurs after a latent period of 6-8 hrs).

• **ttt:** 1- Prevention: (protective goggles).
2- Curative : Bandage for 24 hours + antibiotic drops(2ry bacterial inf.).
3- Local anaesthesia → ↓ healing في ناس بتقول ممنوع
4- Short acting cycloplegic (iritis).

(4) **Excessive light Effect:**

* **Effect**

• Transient blindness: → due to saturation of visual receptors.
(sun light→ visual pigment يكسر كل ال ,
regeneration و بعد شوية يحصل),

• Permanent damage: looking directly to the sun (solar ray in sun eclipse).
This is due to absorption of light by RPE → heat → macular burn:
Early (1-2 day after exposure) → aspeptic patch of yellow dots resemble chorioretinitis .
Late : atrophic patch with pigmentation or lamellar hole.

(5) **Electrical burn**: - Complicated cataract - Anterior uveitis.

(V)Chemical Injuries

◊ Cause:

May be due to:

- Strong acids :e.g. - Sulphuric acid as in Toilet cleaner & Battery fluid مية النار
  - Sodium or Ca++ hypocloride 705 as in poll cleaner.
- Strong alkalies - e.g. - Caustic potash (K OH), caustic soda (Na OH).البطاس
  - Sodium & potassium hydroxide in drain cleaner.
- Corrosives :e.g. - Lime (anhydrous Ca O) الجير الحي : it is the most dangerous as it react with water from the tissues & forming Ca OH with production of heat.
  - Aniline dye (aniline pencils) اقلام الكوبية :
    → Lid edema & massive discharge .DD from purulent conj.
- Chemicals: Iodine صبغة اليود **and** war gases .

◊Effect:

Depends on:

- **Concentration.**
- **Duration:** interval between chemical trauma & 1st aid.
- **Pathogenesis:**

(1) ***Alkalies*** : - Cell disruption .

- Dissolve MPS & corneal stroma & penetrate rapidly into the eye ball.
- Necrosis of the conjunctival BVs leading to PORCELAIN WHITE appearance . See atlas page (174)

(2) ***Acids***: localized damage :

As it precipitates ptns at the epithelial level leading to: physical barrier & buffer effect .

- **Penetration:**

- Acids are less serious than Alkalies because they coagulate & precipitate protein (Coagulative necrosis), which acts as a barrier that prevents further penetration (superficial burns).

◊Clinical Picture

- **Symptoms:** Pain, lacrimation, blepharospasm, photophobia and drop of vision.

- Signs:

	Immediate effect:	Delayed effects
1- Lid	Edema, dermatitis, ulcer.	Cicatricial Entropion, ectropion.
2- Conj.	Ulcer, Injection and chemosis.	Symblepharon, xerosis, pseudopterygium.
3- Cornea	Edema, ulcer (up to melting).	Perforation (endoph.) , vascularizsed opacities, xerosis.
4- Uvea	Anterior uveitis (mild-severe).	Iridocyclitis, atrophia bulbi (due to damage to CB).
5- IOP	May be increased (iritis).	Increased (conj. fibrosis including aq.vein).

◊Treatment: د/أحمد يوسف

*** First Aid ttt:**

- Immediate copious irrigation of the eye with water or physiologic saline for 20 minute using 2 liters , Then:
- **If the nature of the chemical substance is known:**

wash the eye with the proper antidote: as

(i) Strong acids → wash with weak alkali (e.g. Na HCO_3 3%)كربوناتو.

(ii) Strong alkalies → wash with weak acid (e.g. Boric acid lotion 4%) خل.

(iii) Iodine → wash with starch solution or milk.لبن- نشا

(v) Aniline → wash with: Alcohol 10% (Then 10% glycerin).

(iv) Lime: a) Pick the particles with forceps.

b) Wash with: - EDTA 0.1%.

- Neutral ammonium tartarate 10%.

- Saturated Sugar solution → neutral lime saccharate.

محلول سكر مركز

- NB. Water for all except lime (→ Heat & CaOH)

- EDTA is the universal antidote

*** If the nature of the chemical substance is unknown or not available →**

wash the eye with either:

ماء (i) Tap water → to dilute the chemical substance.

لبن (ii) Milk: - Dilution.

- Buffers acids & alkalies.
- Forms a superficial film which protects the underlying tissues.

NB. **If the nature of the chemical substance is unknown we can use litmus paper (PH paper to determine if it's alkalie or acid).**

*** Local ttt:**

1- Antibiotic: against 2ry infection .

2- Atropine: for corneal ulceration & iritis.

3- Steroid: to decrease inflammation, Corneal vascularization, edema, scarring (but should not used early).

4- Soft CL : if the epithelium is not progressing.

5- if the medial aspect is affected : Daily punctual dilatation or silicon intubation .

**** General ttt:***

(i) Antibiotic. (ii) Vitamins (A&C). (iii) Steroids.

(iv) Analgesics as pethidine.

**** ttt of complications:***

1- 2ry glaucoma: CAI tab. And B.B. if failed do TRABECULECTOMY.

2- Small corneal perforation: Cyanoacrelate tissue glue.

3- Impending perforation, corneal opacities : Keratoplasty.

4- Prevention of symblepharon :

- Glass rod coated with antibiotic ointment passed in the fornices 3-4 times daily)
- Cortisone oint.: (not used if there is corneal ulcer)
- Contact shell (cornea scleral →) كبيره عن ال CL العادية
- Soft CL.
- Limbal stem cell transplantation :help epithelialization
- AMG: suppress fibrosis.

5- Lagophthalmos: tarsorraphy.

6- Corneal vascularization : Periotomy + B-irradiation

7- TTT of Xerosis.

8- Surgical ttt of trichiasis & lid entropion.

9- Keratoplasty for corneal scarring.

10- Glaucoma due to scarring of the exit channels of aqueous : by Medical ttt of Filering operation .

(VI) Thermal injuries

- **Types** (1) Splashes of molten metal, hot ashes or exploding powder.
  (2) Hot water, hot liquids and steams.
  (3) Dry burns.
- **Clinical picture:** As in chemical burns.
- **Treatment:** (1) Local: medications and prevention of symblepharon as in chemical burns.
  (2) General :
  1) As in chemical burns.
  2) Transfusion of plasma or its substitutes (to replace the fluid loss).

◈ Why thermal injuries of the eye are less serious than chemical injuries?

Because of rapid closure of lids with corneal protection.

◈ Why FB in upper sulcus subtrasalis is more serious than FB in lower sulcus subtarsalis?

Because of the movement of upper lid which may lead to corneal ulcer.

◈What are the indications of cold compresses?

(1) Recent ocular injury:
1. Black eye (lid ecchymosis).
2. Subconjunctival haemorhage.

(2) Allegic conditions:
1. Spring catarrh.
2. Angioneruortic oedema.

(3) PC: If lid oedema and chemosis are marked.

The orbit

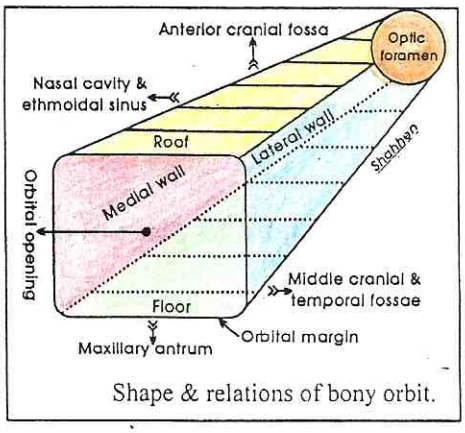
Page 82

Anatomy:

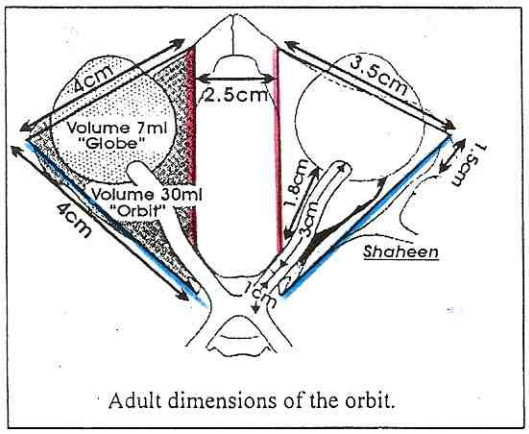
- The orbit is the bony socket of the eye.
- It is 4 sided wall pyramidal with base anterior and the apex posteriorly.
- The 2 adult lateral orbital walls make with each other angle 90°
  & with the sagittal midline angle 45° .
- The 2 medial walls are parallel & makes angle 45° with the lateral walls
- The 2 medial walls → 2.5 cm apart .

- Orbital rim:

is the anterior opening of the orbit & has a rectangular configuration with a horizontal dimension 40-45mm & vertical 30-35 mm.



Shape & relations of bony orbit.



Adult dimensions of the orbit.

- Volume = 30 ml.

NB. Eye volume = 7ml

• Orbital fissures and foramina:

1- Optic foramen (canal): connects the orbit with middle cranial fossa .

It transmits: ① - Optic nerve with its meningeal covering.
② - Ophthalmic artery (below then lateral to optic n.). MCQ
③ - Sympathetic twigs around the artery .

2- Superior orbital fissure:

- **LIVE** = Lacrimal n.
- **FREE** = Frontal n.
- **TO** = Trochlear n.

+ Superior ophthalmic vein → **Above the ring**

- **SEE** = Superior division of 3rd n.
- **NO** = Nasociliary n.
- **INSULT** = Inferior division of 3rd n.
- **AT ALL** = Abducent n.

+ Inferior Ophthalmic Vein → **inside the ring**

+ Sympathetic fibers to the ciliary ganglion .

NB. Nothing below the ring but rarely → inferior ophth. Vein.

NB. No lymphatics inside the orbit.

NB. There is a close relation between the orbit & the brain :

- Optic foramen connects the orbit to → middle cranial fossa
- orbital roof separates the orbit from ant. cranial fossa.

3- Inferior orb. Fissure:

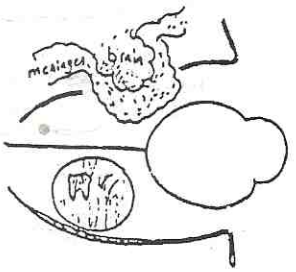
It transmits
- Maxillary division of Trigeminal nerve (Zygomatic n.).
- Infra-orbital artery & n.
- Some times inf. ophthalmic vein.

Diseases of the Orbit

Proptosis

Definition: It is a Passive protrusion of the eyeball outside the orbital rim.

* (Passive = it's pushed by intra orbital space occupying lesion like mass or cyst).
* (Active = Exophthalmos = endocrinal cause: here protrusion of the eye is due to increase the size of normal orbital structure like fat & ms).



Orbit

Etiology:

I- Congenital:

e.g. ① - Dermoid cyst.

② - Meningo-encephalocele : Herniation of a part of meninges & brian through a defect in the orbital roof.

See atlas page (87)

here proptosis is pulsating & increase on crying.

Dermoid cysts:

- Are painless , slowly growing ,benign cyctic lesions that composed of tissues not usually found at the involved site,
- Most common site : anterolateral & superomedial orbit → called superficial dermoids (children).
- Deep orbital lesions occur in adults.
- TTT : surgical removal through lateral orbitotomy approach.

NB. Demoid cyst is one of the **choristoma.** → normal tissue in abnormal place

II- Acquired:

→ Traumatic
→ Inflamatory
→ Neoplastic
} Tin

1- Traumatic e.g.

① Retrobulbar Hematoma دم.

② Surgical emphysema هواء

due to fracture of medial wall of orbit with escape of air from eithmoidal sinus to the orbit & subcutaneous tissue of lid → sense of crepitus .

③ Carotid-cavernous fistula (A-V shunt). → Varices

④ Fracture base of the skull : with escape of CSF into the orbit

2- Inflammatory e.g.

- ***Acute:*** * Orb. Cellulites , phlebitis, periostitis → inflamation of periosteum .

Cavernous Sinus * CS thrombosis→ Varices .

* Panophthalmitis. pus inside + outside
* Tenonitis
* Sinusitis → mucocele formation & also transmitted edema to the orbit both, lead to proptosis.

Orbit

- ***Chronic:*** - **Specific :** T.B. → granuloma ,& Syphilis → Gumma.

- **Non-specific:** orbital pseudo- tumor

(Idiopathic orbital inflammatory disease).

Non specific , non neoplastic inflammatory mass of unknown etiology , responding dramatically to oral steroids within the 48 to 72 hours of the initial dosage .

NB. malignant transformation → lymphoma

TTT : 1- oral steroids.
2- Radiotherapy.
3- Immunosupressives :
Cyclophosphamide

3- Neoplastic: (benign or malignant).

1- Primary:

i- From orbital structures:

- Lacrimal gland Tumors (e.g. Adenoma, Adenocarcinoma, mixed tumor).
- Optic nerve tumors
  (e.g., glioma, meningioma → Sammoma bodies (فيها calcification).
- Rhabdomyosarcoma, lymphomas, Hemangioma, Osteoma.

ii- Extension from intra-ocular tumors (e.g., retinoblastoma).

iii- Extension from nose and sinus tumors : e.g. cancer maxilla.

iv- Extension from intra-cranial tumors.

Teratoma is* only germ cell tumor in Testes & ovary

Capillary hemangioma:

- This lesion is hamartoma, composed of tissue normally found at the involved site.
- Usually presents in the 1st 6 months of life.
- Site: * Superficial →subcutaneous , anterior orbital, or conjunctival.
  * Deep in the orbit.
- TTT: intralesional injection of steroids .
  (when heamangioma threaten to cause ambylopia).

NB. Capillary hemangioma is one of the **hamartomas**

2- Secondary:

i- From the breast (in female), bronchi and prostate (in male).

ii- Secondaries from leukemia.

Lacrimal gland tumors: د.أشرف الدسوقي

→ epithelial → proptosis + diplopia

→ Lymphoid (65%) non epithelial → no proptosis

(1) Epithelial 35%:

- Not respect surrounding structures :
  - → proptosis & displacement of the globe & flattening of globe(diplopia)
  - → Globular pattern of enlargement.
- Benign 50% e.g. BMT (Pleomrphic adenoma)
  - → malignant transformation.
  - ttt: local excision without prior biopsy to avoid recurrence.
- Malignant 50% e.g. Adenoid cystic carcinoma:
  - → Histological ex. →Swiss cheese pattern.

(2) Non-epithelial (Lymphoid) tumors 65%:

- Respect surrounding structures :
  - → No proptosis & No displacement of the globe.
  - →Diffuse pattern of enlargement.

Optic nerve tumors:

→ glioma

→ Meningioma

- GLIOMA:

1- Before the age of 10 yrs. (but in adult onset it will be very malignant).

2- ↓ of vision due to optic n. atrophy + proptosis + squint (but no diplpia)

3- Slowly progressive tumors.

4- Fusiform appearance (onion shape) → enlargement of optic foramen.

See atlas page (86)

5- Associated with type I neurofibromatosis.

6- Minimal or no calcifications.

- MENINGIOMA:

1- Occurs in old age 70-80 yrs (but in childhood onset it will be very malignant).

2- Presses on the optic nerve → CRVO → Opto-cilliary shunt.

3- Calcifications (Sammoma bodies) See atlas page (86).

4- Other causes:

a- Vascular: Aneurysm of ophthalmic a., tumors, A.V. shunts and varices.

b- Cyst: Dermoid cyst, hydatid cyst. → echinococcus granulosus (Casoni test)

c- Endocrinal: Dysthyroid Ophthalmopathy في الزيادات.

d- Paralysis of extra-ocular ms. (3rd n palsy): Orbital fat will push the eye due to loss of tone of the EOMs.

N.B. *Pseudo-Proptosis:*

1- Ipsilateral causes: i- Large eye (high myopia- Buphthalmos).
ii- Shallow orbit .
iii- Lid retraction e.g. in thyrotoxicosis.

2- Contralateral causes: Contra-lateral enophthalmos or atrophic eye.

⬢Diagnosis:

I- History:

- Onset and course:
  - Acute: inflammation (orbital cellulites), trauma , Malignancy (Rahbdomyosarcoma), Vascular or Orbital Pseudo-tumor
  - Intermittent: *Orbital varices (on leaning forward or Valsalva).
    See atlas page (86)
    * Recurrent hge. (Hemophilia)
  - Slowly progressive: Neoplastic (Benign)
  - Regressive : Inflammations .

- History of trauma.
- Pain :indicates **inflammations** :
  (orb. Cellulites – Csthrombosis- panophthalmitis – acute dacyoadenitis)
  Or **malignancy**.
- Diminution of vision داشرف الدسوقي : - Compression of optic nerve
  - Exposure keratopathy
  - Pressure on globe → cordial folds

NB. What are the causes of pain in the orbit ? د.اشرف الدسوقي

1- Inflammation involving periosteum
2- Bone destruction
3- Rapid ↑ of intraorbital pressure

II- Examination:

A) General

أنف و أذن 1- ENT examination : tumor or sinusitis

باطنه 2- Medical ex: Thyroid Look for primary

, Enlarged lymph nodes (Leukemia).

B) Local

1- Inspection:

i- Unilat. or bilat.: 1- Bilateral: - Reticulosis (leukemia, lymphoma)

- Endocrinal
- Late cases of CST Cavernous sinus thrombosis

2- Unilateral: other causes (e.g. inflamm., trauma, tumors)

ii- Direction:

- Directly forward :e.g. optic. n. tumors See atlas page (85),
  thyrotoxicosis See atlas page (85).
- Forward, down and in: e.g. Lacrimal gland tumors. See atlas page (55)
- Forward, down and out e.g. Frontal or ethmoidal mucocele.or meningioencephalocele as it arise from nasal angle of the orbit. (أي حاجة في Sinus)
- Upward: Cancer maxilla .

الخلاصة * Axial in conical lesions.

* Displaced in extraconical lesions.

iii- Pulsations ; best seen on profile view:

e.g. - Oph. artery. aneurysms.

- Vascular tumors (sarcoma).
- Carotid-cav. fistula .
- Meningo-encephalocele (CSF pressure ↑ with systole).

iv- Signs of inflammation:

- Lid: edema. - Conjunctiva: Chemosis, hyperemia.

v- Scleral rim .

vi- Ocular motility affection: restrictive myopathy, orbital fracture, neurological lesion.

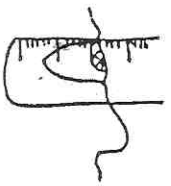
2- Palpation: - Consistency: hard → malignancy

- Fixation of the lesion: Fixed → malignancy.
- Pulsation. - Tender or not.
- Thril in carotid-cavernous fistula.
- Erosion of orbital bones by orbital tumour.

3- Auscultation: in A-V fistula → machinary murmer (Bruit) = Helicopter Sound.

4- Measurement of Proptosis:

1- The distance between : Lateral orb. Margin & Apex of the cornea



Normally is 20 mm

(difference between the 2 eye is 2 ml)

The measurement is done by:

i- Simple transparent ruler. See atlas page (87)

ii- Hertels exophthalmometer. See atlas page (87)

2- Stand behind the pt. & tilt his head backwards & notice the 2 eyes .

See atlas page (88)

5- Visual acuity مهم: decreased in exposure keratitis, optic nerve compression, choroidal folds.

6- Pupil مهم: APD in case of optic nerve compression

7- Fundus مهم: O malignancy , papliedema ,optic atrophy & choroidal folds.

III- Investigations:

A) ***Laboratory:*** - CBC e.g., for leukemia→ Leucocytosis (↑↑ WBCs)

- Tuberculin test. - Wather Man reaction.
- T3 , T4 and TSH levels.
- Casoni test : for hydatid cyst.

B) ***Radiological*** :

1- Plain x-ray skull : - Wide op. foramen (e.g. glioma of op n.).

- Calcification = Sammoma bodies (Meningioma).

2- Arteriography and venography (C.C. fistula).

3- Ultrasonography.

4- C.T. scan.

5- MRI (not used if there is intra-orbital metallic FB).

C) ***Surgical Biopsy***: - Orbitotmy & Excisional biopsy.
- Needle biopsy : through the skin of the lid.

●Complication: Exposure keratitis→ pain, lacrimation .....

●Treatment:

I- Treatment of the cause (Leukemia , inflammation).

2- Orbitotomy : See atlas page (88)

Surgical removal of the cause through orbital approach:

i- Anterior Orbitotomy (through skin or conj.) :
for lesions in the ant. 1/2 of orbit (e.g. lacrimal gl.).

ii- Lateral Orbitotomy (through lateral orbital wall) : For deep lesions.

iii- Trans-frontal Orbitotomy (through anterior cranial fossa) :
For orbital apex lesions or tumors invade the brain ,its done by Neurosurgeon.

Orbital infections

1) Presebtal cellulites: acute suppurative inflammation ant. to the orbital septum.

2) Sub-periosteal abscess: collection of bus from inflammed sinus under the perioestum.

مهم جدا من المحاضره 1 & 2

3) Orbital cellulites

4) Cavernous sinus thrombosis.

Presebtal cellulites

See atlas page (83)

- Causes: 1- Skin trauma
2- Spread of infection from dacyocystitis.

- Symptoms : FAHM + pain

- Signs: Lid redness & edema فقط **+ quite eye**

(NO proptosis, chemosis, VA affection, pupillary reaction affection or affection of ocular motility)

- **CT:** Opacification ant. to orbital septum. See atlas page (84)
- **Complications:** → Orbital cellulitis & Cavernous sinus thrombosis.
- **TTT:** AB

Sub-periosteal abscess

- **Symptoms : FAHM + pain**
- **Signs:** - Lid edema.
  - Limitation of ocular motility in the same direction of the abscess.
  - Eye is pushed to the opposite direction of the abscess.
- **CT:** opacification related to the affectes sinus that also appears opaque.
- **Complications:** → orbital cellulitis & Cavernous sinus thrombosis.
- **TTT:** drain the pus either by nasal endoscoe or surgical .

Orbital cellulitis

See atlas page (83)

• **Definition:** It is acute suppurative inflammation of the orbital fibro-fatty tissue, behind the orbital septum (retrobulbar).

• **Incidence :** more common in children. شاهين

• **Etiology**:

(I) Routes Of Infection:

1- **Exogenous infection:** by - Penetrating wounds.
- Following septic orbital surgery (squint, R.D.).

2- **Endogenous infection:**

i- Spread of infection: from
- Sinuses (ethmoiditis due to thin wall of ethmoidal bone): This is the most common cause 60% of cases.
- Teeth.
- Globe (Endophthalmitis).
- Otitis media -Stye - Acute dacryocystitis

ii- blood borne: as in septicemia, SABE.

(II) Organisms:

1- Adults : Staph, Strept, pneumo.& rarely fungi in immuno-compromised pt. (orbital mucormycosis).

2- Children: Hemophillus influenza.

• Clinical picture :

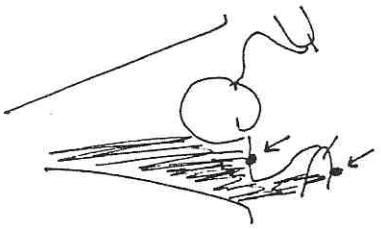
* **Symptoms:** - **General:** Fever, headache, malaise.(FAHM)

- **Local:**
  - * **Pain** → increased by eye movements
    → Dull then it becomes throbbing pain.
  - * **Vision**
    - Early: Good.
    - Late: Decreases due to :
      - Op. neuritis.
      - orbital abscess formation → compression

مهم

  - * **Diplopia.**

* **Signs:**



1- Lids : Oedema , hyperemia & tenderness.

2- conj.: Chemosis & ciliary injection.

3- Proptosis (Axial).

4- Limitation of ocular Motility (myositis , neuritis & pain with movement).

5- An abscess may forms & points through :

- Lower fornix by gravity.
- Skin near lower orb. Margin.

(lower & lateral because of the orbital floor direction)

6- Pupil: APD or dilated (when abscess if formed → compression)

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• Complications:

1- Extension of infection: into
- Cranium → (brain abscess, cav. sinus thrombosis , meningitis).
- Op. nerve → Op. Neuritis .
- Globe → endophthalmitis.
- General spread → Septicemia & Pyemia.

2- Increased intra-orbital pressure : - CRV thrombosis → Papilloedema.
- CRAO.
- Optic atrphy.

3- Proptosis : leads to corneal ulcer (exposure) .

4- Corneal anaesthesia.

5- Post-inflammatory fibrosis : Enophthalmos & restricted ocular motility (frozen orbit).

• **D.D:** 1- Cavernous sinus thrombosis (See the table).

2- Acute suppurative dacryoadenitis.

•Treatment:

(i) Hospitalization.

(ii) Culture from conj , nose & blood

(iii) Medical ttt:

1- *General* : - Antibiotics (IV anti Gm +ve , Gm –ve & anti anaerobics)
- Asprin : ليه؟
- IV fluids : ليه؟

2- *Local* : Hot fomentations, antibiotic drops and oint. to protect the cornea.

Then do CT & systemic work up

- Follow up 48 hours (vital signs, conciseness ,pain , ocular motility,VA, proptosis)

If no improvement do CT again (it may be fungal , retained FB, orbital pseudo-tumor or rahbdomyosarcoma)

(iv) Surgical drainage: if orbital or subperiosteal abscess .

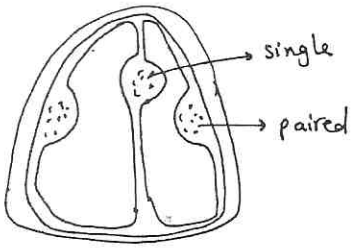
Cavernous sinus thrombosis

★Definition: It is thrombo-phlebitis of the Cavernous sinus (inflammation & thrombosis of the cavernous sinus).

Orbit

★Etiology:

Anatomy of the cavernous sinus



It is one of the paired dural sinuses.

Relations: **- Medially:** Sella tursica (pit. gland)& sphenoidal sinus

- Laterally: Temporal lobe.

Contents: 1- The cavity contains:

- Internal carotid artery (surrounded by sympathetic plexus)
- Abducent nerve.

2- The lateral wall contains (from above downwards):

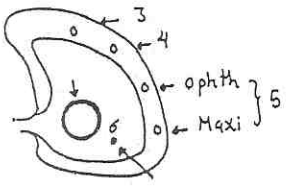
- 3rd n.
- 4th n.
- Ophthalmic n.
- Maxillary n.

} **In the post part**

- 4th n.
- 3rd n.
- Ophthalmic n.
- Maxillary n.

} **In the ant. part**

Communications:



1- Anteriorly: I) Superior & inferior ophthalmic veins.

II) Central retinal vein.

(sometimes CRV→ Sup. ophthalmic V. → Cavernous sinus).

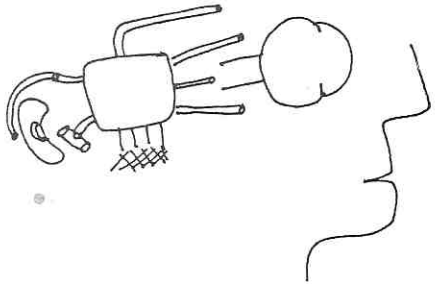
- Thus communicating with: a- Eye & orbit, b- Face as ophthalmic veins communicate with angular vein.

2- Posteriorly: Superior petrosal sinus(SPS) & inferior petrosal sinus(IPS),

- Thus communicating with :

a- Subcutaneous layer behind the ear:- via the SPS & mastoid emissary veins .

b- Middle ear : via IPS & internal jugular vein.



3- Superiorly: Middle cerebral vein.

4- Inferiorly: Emissary veins → Pterygoid plexus,

- Thus communicating with the mouth, pharynx & nasal sinuses.

5- **Medially:** Inter-cavernous sinuses to the other cavernous Sinus.

So The infection may come from:

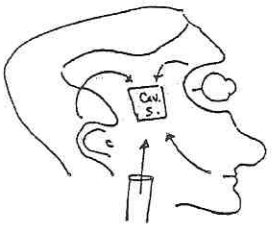
a- Face and orbit (Dangerous ∆) via ophthalmic vein.

b- Middle ear (inf. Petrosal sinus).

c- Mastoiditis: via SPS.

d- Mouth and pharynx (pterygoid plexus).

e- Blood borne (metastatic).



* **Organisms:** Staph and Strept.

★Clinical picture:

* **Symptoms:** - **General:** fever, headache, malaise FAHM (marked) + cerebral symptoms = drowsy = ↓ of conscious state

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- **Local:** - Severe orbital pain :due to irritation of sensory nerves.

- Vision: - *Early*: Good.

- *Late*: decrease due to op. neuritis (spread to optic n. along meninges)

- Diplopia :due to paralysis of 3rd nerve.

* **Signs:**

1- **Lids:** edema & hyperemia.

2- **conj.:** edema & ciliary **congestion**.

3- **Axial proptosis :** (in late cases, Extension to the other sinus → bilat. Proptosis).

4- **Limitation of ocular motility (Total ophthalmoplegia):** due to neuritis.

5- **Fundus:** engorged veins and disc edema (papilloedema).

6- **Oedema of mastoid region :** due to thrombosis of mastoid emissary veins

7- **Pupil :**dilated fixed (3rd n.) .

N.B. The earliest sign in the other eye → convergent squint {6th n. palsy}.

NB. MRI → shadow of sup. opthamic vein مهم المحاضره.

★ Complications: **death** usually occurs due to:

1- Meningitis and brain abscess.

2- Pulmonary complications as pulmonary embolism or infection.

★D.D.: see the table.

★Treatment:

The condition is very serious & could be fatal if not well treated.

- **Hospitalization.**
- **General ttt:** 1- Massive antibiotics IV .

  2- Anticoagulant: heparin 5000 i.u. every 6 hours.

  (to prevent spread of thrombus).
- **Local ttt:** Corneal protection from exposure keratitis.
- **Surgical ttt:** Open & extract the thrombus (by neuro-surgeon).

	Endophthalmitis See atlas page (84)	**Panophthalmitis** See atlas page (84)	**Orbital cellulitis**	**Cav. Sinus thrombosis**
Definition:	It is suppurative inflamm. Primarily in uveal tract (sclera is free).	It is severe sup. Inflamm., primarily of uveal tract and involve the outer coat+ orbital tissue	It is suppurative inflamm. of orbital cellular tissue.	It is thrombo-phlebitis of the cavernous sinus.
Aetiology:	As infective iridocylitis (see before).	As infective iridocyclitis (see before).	See the orbit.	See the orbit .
Symptoms: **1- General (FAHM)** **2- LOCAL:** **- Pain** **- Vision**	(+) Severe decrease to no PL	More(++) Severe no PL	(++) Severe good (early)	Severe (++++) Severe good (early)
Signs: **1- lid:** **2- Conj.** **3- Cornea** **4- Proptosis** **5- OC. Motility**	Almost unilateral oedema chemosis + cil inj. Hazy+ Kps absent normal	Almost unilateral oedema chemosis + cil inj. Hazy+ Ring Abscess Present limitted See atlas page (84)	Almost unilateral oedema chemosis+ cil inj. Clear Present limited	Almost unilateral oedema chemosis + cil cong Clear Present limited

6- Red reflex	yellow	yellow	normal	normal
Treatment: **Antibiotic+**	- Seeing eye → Intra-vitreal AB or Vitrectomy - Non seeing eye → Evisceration	Usually non seeing→ Evisceration. **((enucleation not done ؟؟؟ليه))**	AB+ Orbital abscess drainage.	Anticoagulant +AB+ neurosurgical ttt.

N.B. Meningio-encephalocele:

- Herniation of the meninges and part of the brain through orbital roof defect.
- It pulsates with the heart.

Surgical emphysema

- Air passes from the nasal sinuses into the orbit and subcutaneous tissue of the lids through a fracture of the ethmoidal bone.
- It shows: - Proptosis: increase with blowing the nose and -crepitations (are felt in lid).
- Treatment: 1- Pressure bandage. 2- Antibiotics 3- Not to blow the nose.

Carotid-Cavernous fistula:

Causes: it is due to rupture of internal carotid artery as it passes in the cavernous sinus following severe trauma.

- It pulsates with the heart.

Dysthyroid Ophthamopathy

See atlas page (85)

Definition:

- It is ocular & orbital changes resulting from thyroid gland dysfunction
- It is the commonest cause for unilateral & bilateral proptosis.
- It may occurs with hyperthyroidism

→ **Thyrotoxic exophthalmos (Grave's disease)**

hypo-, or euthyriod patient → **Thyrotropic exophthalmos**.

Pathogenesis:

- Immunogenic deposition of MPS & collagen & lymphocytic infiltration in the orbit & EOMs →↑ volume of orbital content & ms enlargemet 8 times → **exophthalmos.**

- Subsequent degeneration of muscles → fibrosis → **restrictive myopathy.**
- Sympathetic over stimulation of muller's ms → Spasm
  → **upper & lower lid retraction.**

Incidence: **(1) Age :** average 35 years.
(2) Sex: Mainly in women.
(3) Laterality: Usually bilateral.

Clinical Picture:

(1) General manifestations of thyrotoxicosis:

Raised basal metabolic rate with loss of weight, heat intolerance palpitation,sweating , tremors (of out-stretched hands) & nervousness.

(2) *Ocular manifestations:*

1- Proptosis (Exophthalmos) : usually axial , occurs gradual.

2- Lid :

1- Lid retraction(commonest sign):fibrosis of SR & levator.
2- Lid twitches.
3- Edema .
4- Lagophthalmos.
5- Dalrymple's sign: lid retraction →Scleral rim(show)
6- Stellwag's sign: infrequent blinking →Starring look.
7- Von Graefe's sign (lid lag): upper lid does not follow the eye on looking down.
8- Joffer's sign: Lack of forehead wrinkling on looking up.
9- Mobius Sign : Weakness of convergence.

3- Conjunctiva:

Chemosis and hyperemia especially over the horizontal recti.

4- Cornea: may be ulcerated (exposure).

5- Restricted extra-ocular ms: - Early due to swelling of the muscles .

& - Later due to fibrosis→ diplopia.

(IR→ MR→ SR → LR)

6- Compressive optic neuropathy: leading to

i- Visual acuity : slowly progressive drop of VA.

ii- Color vision : Affected (red and green).

iii- Visual field : Central or Paracentral scotoma.

iv- Pupil :Afferent pupillary defect

v- Fundus: edema. and finally atrophy.

✖ Investigations:

1- Laboratory : - high T_3, T_4, TSH.

- Thyroid state may be normal or hypo :

now the proptosis is due to - EPS from the ant. Pituitary.

- Auto Abs against eye only.

2- Radiological: Sonar & CT scan shows thick extra-ocular ms.

(except the tendon) See atlas page (85)

✖ Treatment:

(1) Medical TTT of thyrotoxicosis

(2) Protect the cornea : Lubricant - dark glasses – Taping of lids during sleep

(3) ↓ orbital infiltration : Systemic Steroids- cytotoxic drugs -

Radiotherapy to the orbit if steroids are contraindicated.

(4) Surgical ttt:

i- Severe Proptosis : orbital decompression (fat – bone)

ii- Diplopia: ms surgery (recession of the restricted ms).

iii) Lagophthalmos :Tarsorraphy – disinsertion of Muller's ms.

Enophthalmos : See atlas page (87,88)

1- Atrophy of orbital fat:

- Senil - Cachexia & dehydration.
- Post radiotherapy - eye boking in blind infant

2- Fibrosis of the orbital fat:

- Chronic orb. cellulitis
- Schirrus carcinoma : secondaries from the breast

3- Traumatic ☹most common cause .

- Blow out fracture(fracture of orbital f

4- Post-operative :after removal of a large orbital mass.

5- Tumours of the lid or the palpebral part of the lacrimal gland.

6 - Pseudo-enophthalmos:

- Microphthalmos , Pthisis bulbi
- Ptosis e.g Horner → apparent enophthalmos.

Operations of the orbit:

1- Orbitotomy.

2- Enucleation استئصال:

- Principle: the eye ball is excised, while Conj., Op.n., EOMs →are left then suture upper & lower conj.(once the wound heal after about one weak usually an artificial eye is inserted in place to avoid contracted socket).

- Indications: ((Written concent from the pt.)) العيان ممكن يوديك السجن

1- To stop pain e.g. in - absolute glaucoma .زمان

- Staphyloma.

2- To save life e.g. in intraocular malignancy .

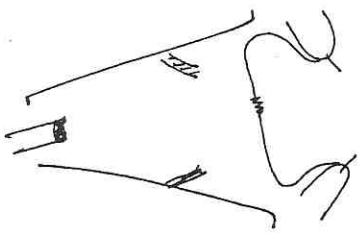
(here the optic nerve is cut as far as possible)

3- To save other eye e.g. in Sympathetic ophthalmitis.

4- To improve appearance e.g, in total ant. Staphyloma.

- Steps:

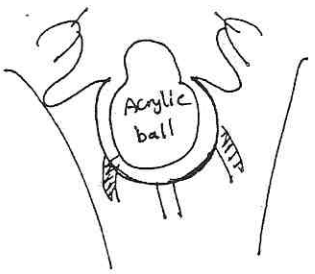
1) Eye speculum is applied.
2) Bulbar conj. is opened around the cornea.
3) The 4 recti are cut close to the globe.
4) Optic nerve is cut using a curved scissor .
5) Removal of the eye ball.
6) Orbital cavity is packed to stop hge.
7) Conjunctiva is closed.
8) Post-operative : - AB.



- Artificial eye after healing of the wound See atlas page (89)

3- Evisceration تفريغ العين See atlas page (89)

- Principle: cornea is excised and all "contents" of the eye-ball are evacuated, while the sclera is left.
- Indications: endophthalmitis and panophthalmitis. (if the eye is non seeing)



N.B. Enucleation cannot be done for fear of extension of infection along the sheath of op. n. → brain.

- Adv. : Cosmetically better (العين تتحرك).

4- Orbital exentration: See atlas page (89)

Principle: It is operation where all the contents of the orbit are removed inside the orbital periosteum. The lids & conj. may be also removed → all in one mass.

Indications: 1- Malignant orbital tumors.
2- Malignant tumors of: lid, conj., globe, (invading the orbit).

Provided that i- Periosteum is free.
ii- No metastasis.

Exentration = remove GLOBE + ORBITAL TISSUE
- Total → (+) PERIORBITA.
- Subtotal → (-) PERIORBITA.
- Radical → (+)PERIORBITA + BONE.

Contracted Socket

* Definition:

Fialure of the conjunctival sac (socket) to hold artificial eye.

* Causes :

1- Neglected wear of arificial eye after enucleation.
2- wearing of loose or tight artificail eye.
3- Excision of conjunctiva at enucleation.
4- Cicatrizing inflammation of the conj.

* ttt : 1) Socket reconstruction using mucous membrane graft.
2) Tarsorraphy:leaving it empty.اقفله وخلاص

Ocular Pharmacology

I) Mydriatics

♥ include:

1- Parasympatholytics	2- Sympathomimetics
1. Atropine sulfate 1%.	1- Phenyl-ephrine 2.5-10%: We use 10% in Egypt (drak races) due to strong dilator ms.
2. Homatropine hydrobromide 1%.	2-Adrenaline (1/1000)"
3. Hyoscine hydrobromide 1/4 %.	- Subconjunctival injection.
4. Cyclopentolate hydrochloride 1%.	- Intra-cameral.
5. Tropicamide (mydriacyl) 1%.	(as Adrenaline is destructed by tear film alkalinity)

♥Action: 1- Pupillary dilatation →↓ Synechia formation & break any new synechia.
2-↓ Pain.
3- ↓ permeability of iris BVs.

♥ Indications:

1-Treatment of: - Corneal ulcers.
- Uveitis.
- Accommodative squint in children < 3 years.

2- Diagnostic: - Before fundus examination.
- Before retinoscopy in children < 10 yrs as the ciliary ms is strong.
- Provocative test in AC glaucoma.
- Post-mydriatic vision test: before visual iridectomy.

3- Operations: * **Pre-operative :** as in cataract & RD surgery.
* **Post-operative:** as in cataract, glaucoma & RD surgery.

♥ Side effects:

1- *Atropine toxicity:*

* Due to systemic absorption.

* Cl. Picture:

Fever. **F**lushing of face.

Fits. **F**ast heart (tachycardia) .

Dry mouth & skin.

* TTT : 1- cold compresses + Antipyretics (fever).

2- Pilocarpine 10 mg IM. or Eserine 1mg S.C. (lipid soluble → reach CNS)

((Parasympathomimetics)).

2- *Atropine sensitivity:*

- Occurs with drops or ointment.
- Clinical picture: allergic dermatitis – follicular conjucntivits.
- TTT: stop atropine – use cyclopentolate – topical steroids.

3- *Dangers:*

i- Angle closure glaucoma in eyes with narrow angle.

ii- prevent monitoring of the pupil in comatosed patients.

iii- Prevent monitoring of the pupil during anesthesia.

iv- Behavioral disturbances (hallucination) especially with cyclopentolate.

NB. The only antidote to the mydriatic action of atropine is ACH intracameral

II) Miotics

◊ Include:

a- Direct parasympathomimetics	**b- Indirect parasympathomimtics (Anti-cholinesterase)**
1- Pilocarpine nitrate 1-4%.	1- Eserine salicylate 1/4 – 1/2%
2- Acetyl Choline (intracameral) لانه بيتكسر بسرعة	2- DFP 0.1% :Obsolete (organophosphorous).

c- Double action: (direct & indirect): Carbacol .

◊ **Indications:**

1- ttt of : -1 ry glaucoma (CAG & OAG).

- Some cases of Accommodative squint.

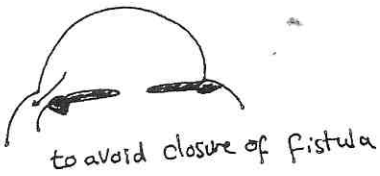
2- Before some operations: - Goniotomy (to see the mesodermal memb.)

- Filtering surgery I
- Keratoplasty (to protect the lens)I
- Laser iridotomyI

3- After some operations:

* Goniotomy (to avoid adhesions بعد العملية)I
* Cyclodialysis (internal fistulising op.):

to prevent closure of the fistula.



4- Counteract mild mydriatic drugs.

5- Pilocarpine used in diagnosis of Adie's pupil.

◊ **Side effects:**

- Systemic: Headache, GIT disturbance, Bradycardia .
- Local: see glaucoma .

NB. Although Eserine is stronger, pilocarpine is preferable ؟؟؟ فاكر ليه (see glaucoma)

III)Corticosteroids

Corticosteroids very important group of drugs that have many indications, with serious side effects on prolonged use.

◊ **Action:** 1- Anti-inflammatory. 2- Anti-Allergic,

3- Decrease fibroblastic activity.

◊ **Indications:**

1- Lids: Allergic dermatitis, insect bites , Allergic blepharitis, To avoid symblepharon.

2- Conjunctiva: Allergic conjunctivitis, (Phlyctenular conj., spring catarrh)

3- Anterior segment:

- Keratitis (interstitial ,deep).
- Episcleritis & scleritis.
- Iridocyclitis.
- Acute phase (1st 10 days) of chemical injuries and bruns.

In all of the above corticosteroids are used topically, or by local injections.

4- Posterior segment:

In these conditions topical steroids are not effective and either systemic or retrobulbar injections are used:

1- Posterior uveitis.

2- Anterior ischemic optic neuropathy: where mega doses of steroids may be used. The patient is hospitalized and doses as high as one gram are given intravenously under the supervision of an internist.

3- Sympathetic ophthalmitis.

4- Optic neuritis.

5- Post-operative: to decrease post operative inflammation (cataract). and also to prevent graft rejection in cases of keratoplasty.

◊ Dose of systemic steroids:

The usual dose of systemic steroids ranges from 1-2 mg/ kg/day i.e., 60-120 mg for adults. In certain conditions up to 1000 mg can be infused daily (mega dose). (Hospitalization & internist).

◊ Dangers of local steroids:

1- Steroid induced glaucoma : due to
   - Unknown.
   - Salt & water retention.
   - Deposition of hyalo-uronic acid crystals in TM.
   - TIGR protein.

2- Steroid induced cataract (post. subcapsular) due to metabolic & hormonal disturbance.

3- Increasing susceptibility to infections.

4- Reactivation of dormant organisms e.g.herpes virus. (↓ immunity).

5- Delaying wound healing.

◊ Dangers of systemic steroids (prolonged use):

1- Peptic ulceration.

2- Steroid induced diabetes.

3- Hypertension due to salt and water retention.

4- Steroid induced cataract.

5- Muscle wasting & osteoporosis.

6- Reactivation of dormant infection (T.B).

7- Cushingoid state.

8- Psychic disturbances.

9- Sudden stoppage after prolonged use may leads to acute adrenal insufficiency (Addisonian crisis) so r gradual withdrawal **is important.**

Packaging standards

- Mydriatics & cyclo → red.
- Miotics → green .
- B – blockers → yellow or blue.
- NSAID→ grey.
- Anti – infectives → brown.
- CAI → orange.

♦Mention the eye lotions, their uses and actions?

(1) Boric acid lotion 4%:

1) Conjunctivitis as MPC and spring catarrh.

2) Alkali burns.

(2) Sodium bicarbonate 3%:

1) Squamous and ulcerative belpharitis.

2) Acid burns.

(3) Sodium chloride 0.9% : Unknown chemical burns.

(4) Starch: Iodine burn.

Action of eye lotions:

(1) Mechanical removal.

(2) Dilute toxins.

(3) Mild antiseptic.

Ocular tumors

(i) Extra ocular tumors:

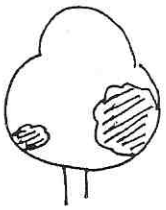
- Tumors may arise from extra ocular structures such as the lacrimal gland, optic n., lids and conjunctiva.
- The most common extra ocular tumors include mixed lacrimal gland tumors,

basal cell carcinoma of the eye lids: - Has rolled edeges.
- Upper lid is protected by eye brow from the sun.

squamous cell carcinoma of the conjunctiva .

Glioma & meningioma of the optic nerve.

(ii) Intra ocular tumors:



RETINOBLASTOMA (GLIOMA RETNAE) مأساه

- **Definition:** it's malignant tumor of the retina
- **Origin :** from primitive retinoblasts due to inactivation of retinoblastoma gene.
- **Age:** 2-5 years.
- **Hereditary:** plays a role(retinoblastoma gene located on chromosomes13 &14).

"افحص العيله "

- **Laterality:** bilateral in 25% of cases & multicentric. "افحص العين التانيه"

NB. Primitive cells disappear with in the 1st few years of life so tumor is seen only at 2-5 years.

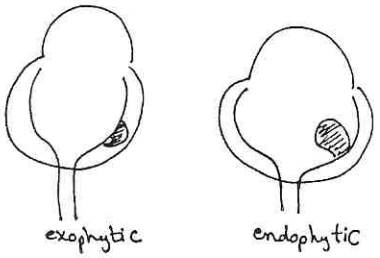
Clinical picture:

1) White pupil = Luco-coria = Amaurotic cat's eye عين القطة العمياء See atlas page (157)
2) Squint: (so fundus examination is mandatory in all cases of childhood strabismus).
3) Visual affection: if the macula is affected.
4) Secondary glaucoma (Buphthalmos): leads to → enlargement of the eye.
5) Proptosis
6) Anterior segment invasion (iridocyclitis). :
   - Hyphema - Malignant hypopyon .
   - Painful red eye
   - Iris nodules
7) Orbital inflammation : like orbital cellulitis (exact mechanism is unknown).

Stages:

(1) Quiscent stage: العين كويسة

- Tumor is dormant inside the eye.
- Ealy cases : flat ,rounded white lesion.
- Endophytic tumors : project from the retina into the vitreous as grayish white mass projecting into the vitreous with fine new-vascularizations & few patches of hge upon the tumor , calcifications & vitreous seeding may be present. See atlas page (156)
- Exophytic type: tumor grows outwards as subretinal white mass with an overlying RD.



(2) Glaucoma stage (secondary buphthalmos): why?

- Space occupying lesion
- Pushing the iris lens diaphragm forwards
- Toxic iridocyclitis→ Hyphema or hypopyon.
- Malignant cells block the angle (psuedohypopyon)
- Intra ocular hge
- Closure of vortex veins → IO congestion
- Rubeosis iridis

(3) Stage of extra-ocular extension:

- Along the optic n.: → to the brain (death) most common spread ***MCQ.***
  See atlas page (156,166)
- Through sclera : → to the orbit (proptosis). See atlas page (157)

(4) Stage of distant meatastasis:

- Along B.V. : → to L B L B. (Very rare as the tumor not highly vascular like MM)
- Along lymphatics: → to regional L.N. (rare).

Investigations:

1- Indirect ophthalmoscope examination : with full mydriasis & scleral indentation by strabismus hook under general anesthesia, (without indentation pre equatorial tumors may be missed).

2- X-ray skull: calcifications & wide optic canal.

3- Ultrasound & C.T. : calcifications See atlas page (157)

4- MRI : cannot detect calcifications ,but superior to CT in detection of optic nerve involvement.

DD:

From other causes of white pupil = Amaurotic cat's eye (blind eye Showing yellow reflex). مهم

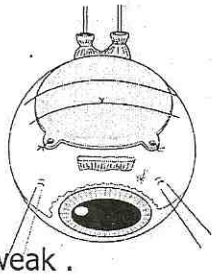
TTT:

(1) Small tumor (not more than 4 mm in diameter without vitreous seeding or optic nerve affection) :

1- Trans pupillary thermotherapy(TTT):

- using argon laser .
- Recently diode laser is used, not absorbed by RPE, so not affect the retina.

2- Trans-scleral Cryotherapy: for tumors ant. to the equator which cannot be treated with laser.



(2) Medium sized tumors (not more than 12 mm diameter):

1- *Brachytherapy*: radio-active plate مشحونة fixed to the eye for 1 weak .

2- *Chemotherapy*: followed by thermotherapy or cryotherapy.

3- *External beam irradiation* : high risk of complications as retinopathy & cataract & may induce a second malignancy like osteosarcoma.

(3) Large tumors:

1- Chemotherapy : is used to shrink the tumor (chemoreduction), so local ttt can be delivered to a smaller tumour (thereby avoiding enucleation)

2- Enucleation: with long stump of optic nerve & without perforation of the enucleated globe : if chemoreduction fails & in cases of diffuse tumors because of poor visual prognosis & increase the risk of recurrence

See atlas page (156)

(4) Extra-ocular extension :

is treated with→ Exenteration افرغ+ radiotherapy to the orbit + systemic chemotherapy.

(5) Metastatic tumor : palliative ttt (high doses of chemotherapy).

N.B. Observation of the other eye is V. important→ as it's bilateral.

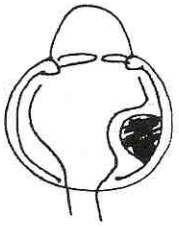
Prognosis:

1- Optic nerve involvement beyond the surgical cut : is associated with high mortality rate,
2- Tumor size & location : small posterior tumors do best.
3- Cellular differentiation: mortality rate is high with undifferentiated tumors.
4- Invasion of the choroid or vortex veins or sclera : of poor prognosis.
5- Over all mortality rate is 2-5% .

MALIGNANT MELANOMA

See atlas page (157,158)

- **Definition:** it's malignant tumor of the choroid.
- **Origin :**from melanocytes.
- **Age:** adult above 40 yrs (it's the most common primary intra-ocular tumor in adults).
- **Hereditary:** has no role.
- **Laterality:** unilateral & unicentric (one lesion).
- **Race:** rare in negroes



Clinical picture :(symptoms)

1 - Asymptomatic : detected by chance.
2- Diminution of vision: due to
- Malignant RD . - Involvement of the macula. - IO Hge
- Corneal oedema due to 2ry glaucoma. - Cataract.

3- Field defect
4- pain: due to -2ry glaucoma -iridocyclitis
5- Photopsia

Stages: (signs)

(1) Quiscent (asymptomatic) stage:

- Tumor is dormant inside the eye.
- Dome shaped dark (brown) mass with pigmentations & hge over it → mashroom shaped (collar stud)after it passes through the Bruch's membrane.

See atlas page (158)

- It may be amelanotic (non-pigmented)
- Orange pigment (Lipofuscin) on the surface of the tumor is common.

- Malignant RD with no retinal tear.

(2) Glaucoma stage: why?

As before

(3) Stage of extra-ocular extension:

- Along the optic n.: very rare?? ليه due to marginal tissue of ELsching (Malignant melanoma never go to optic nerve).
- Through sclera : - post.→To the orbit (proptosis).
  - Ant. → dark mass under bulbar conj.

(4) Stage of distant metastasis:

- Along B.V. : → to L B L B (Common as the tumor is highly vascular).
- Along lymphatics: → to regional L.N. (rare).

Investigations:

1- *Indirect ophthalmoscopy.*

2- *Ultrasound & MRI*: if the media is opaque.

3- *FA*: dual circulation

4- *ICG*: provide more information about the extent of the tumour.

5- *Transillumination test*: black pupil (mass is opaque).

* method : strong beam of light from transilluminator is put in contact with the sclera & red color of the pupil is noted:
  → Black pupil.
  → Red pupil.

6- *Radio-active P32 uptake*: increased.

7- *General medical examination* : to exclude other metastasis & the site of primary tumor (bronchi & breast).

DD: Malignant RD from 1ry RD جدول اكتبه

TTT:

1) Brachytherapy:

- 1st line as it's effective.
- Indication: Tumor less than 10mm in elevation & less than 20 mm in diameter
- We can add Transpupillary thermotherapy (TTT) to enhance the result.
- Regression starts after 1-2 months.

- Complications : Retinopathy , papillopathy , cataract & recurrence of tumour.

2) Charged particle irradiation (External radiotherapy):

- Advantages over bachytherapy : beam can be slightly focused.
- Dose is divided over 4 days , each dose is 30 seconds period.
- Indications : - Large tumors.
  - Posterior tumors located 4 mm from disc or fovea.
- Complications : neovascular glaucoma & exudative RD.

3) Transpupillary thermotherapy (TTT) :

- Using Diode laser that causes hyperthermia not coagulation.
- Indication : pigmented small near fovea lesion.

4) Transcleral local resection:

- Indication : tumors that too thick for radiotherapy & less than 16 mm diameter.
- Complications: Hges & RD.

5) Enucleation : See atlas page (158)

- In very large tumours with loss of all useful vision.
- It should be carefully done to avoid blood dissemination of malignant cells.

6) Exenteration : in cases with extra-ocular spread.

7) palliative ttt : with chemotherapy in pt. with secondaries.

Modified Callender classification of uveal melanoma :

1- Spindle cell melanoma 45% : of good prognosis.
2- Pure epithelioid cells 5%: of poor prognosis.
3- Mixed cell melanoma 45% : spindle & epithelioid cells.
4- Necrotic melanoma 5% : the predominant cell type is unrecognized .

Prognostic factors :

1- Tumors of epithelioid cells have the worst prognosis.
2- Large tumors & extra-ocular extension have poor prognosis.
3- Peripheral tumors have poor prognosis (as they are diagnosed later).
4- Age : pt. above 65 years usually have poor prognosis.

N.B. observation of the other eye is not necessary.

Luco-coria = white pupil = Amaurotic cat's eye= yellow reflex

Causes:

1) Glioma Retinae(Retinoblastoma).
2) Pseudo-Glioma:
   1- Congenital cataract.
   2- Cyclitic membrane.
   3- Endophthalmitis.
   4- Total RD.
   5 -Retrolental fibroblasia (ROP) retinopathy of prematurity نتيجة الحضانة
   6- PHPV(persistent hyperplastic primary vitreous
      = persistent fetal vascularure):
   7- Coloboma of the retina & choroid → Showing the sclera. See atlas page (153)
   8- **Coat's disase:**
      - idiopathic (مش وراثه) telangectasia , abnormal vascularization of the retina → extensive sub-retinal exudates & exudative RD.
      - age : boys in the 1st decade
      - TTT: laser photocoagulation to obliterate telangectazia
   9- Post. pole toxocariasis: due to ingestion of intestinal worm of dogs (toxocara- canis) → granuloma.
   10- Retinal astrocytoma.
   11- Retinal dysplasia : failure of the retina to develop during embryonic life → white retrolental membrane in a microphthalmos eye.
   12- Vitro-retinal dysplasia: → white retrolental mass.

Retrolental fibroblasia(ROP) retinopathy of prematurity:

Premature infant exposed to hyperbaric O_2 in incubators will develop vaso-spasm of the peripheral retinal B.V. →ischemia with release of vasogenic factor → new vessels which will invade the vitreous → vitreous hge → organisation& fibrosis →retrolental fibroblasia & tractional RD.

The Pupil

Function:

1- Regulates amount of light entering the eye.

2- Decreases aberrations (chromatic &spherical) by cutting peripheral rays.

3- Increases depth of focus. يعني ايه؟

The light reflex

→ In Mid brain EWN + pretectal

When light falls on eye: - Ipsilateral pupil constricts (direct reflex).
- Also other pupil (consensual reflex).

1- Stimulus: light .

2- Receptors: rods and cones.

T = Temporal = Same side
N = Nasal = opposite

3- Afferent:

1- Optic nerve.

2- Optic tract (temporal fibers) and optic chiasma (nasal fibers).

3- Mid brain (pretectal nucleus) & from pretectal nucleus, new neurones (2 intercalated neurones) deliver impulses to Edinger Westphal (parasympathetic) nucleus on both sides.

4- Center: Edinger Westphal nucleus (part of 3rd n. nucleus).

5- Efferent : 3rd n → ciliary ganglion (through nerve to IO) مهمه
→ short ciliary nerves
→ constrictor pupillae ms. of both eyes = Consensual reflex

N.B. The consensual reflex:

is due to crossing of fibers at:

1- Chiasma (nasal fibers).

2- Mid brain (intercalated neurones to 2 Edinger W. nuclei)

The near reaction

→ no need to pretectal nucleus
→ only need Edinger W. nucleus

When a near object is viewed 3 related (synkinesis) reflexes occur:

1- **Accommodation:** due to contraction of ciliary ms.

2- **Convergence:** due to contraction of 2 medial Recti muscles.

3- **Miosis.** → Constrictor pupil

Pathways:

1- Stimulus: blurring of image.

2- Receptors: rods and cones.

3- Afferent: Optic Nerve → Optic chiasma. → Optic Tract. → Lat. Geniculate body (LGB) → Op. Radiation → Occipital cortex → Frontal cortex → Internal capsule →

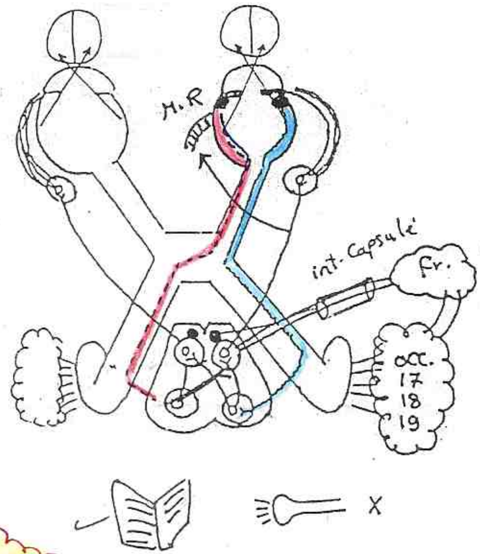
1) Edinger w. nucleus. → parasympathetic fibers to ciliary & constrictor ms.

2) Motor nucleus of convergence → MR.

1) & 2) called oculomotor nuclear complex.

4- Center: Edinger W. nucleus.

5- Efferent: as the light reflex. (3rd n)



Light near dissociation

See atlas page (101)

1- Argyl Robertson pupil:

***Characters:**

1- It is small pupil : as the lesion irritates EWN.
(usually bilateral but assymmetrical).

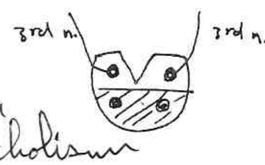
2- That does not react to light but reacts to accommodation(near).

3- It dilates poorly in dark (atropine parasympatholytic → dilatation).

*** Site of lesion:** pretectal nucleus (or intercalated neurone).

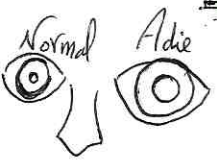
*** Cause:** - Commonest: neurosyphilis.

others = D.M., encephalitis, trauma & chronic alcholism



- Other causes: D.M., encephalitis, trauma,
Chronic alcoholism.

2- Adie (tonic Pupil):



Clinical Picture : mostly affect females between second and fourth decades.

i- Large and regular pupil

(except in long standing old cases → miosis & irregular pupil).

ii- Anisocoria especially in intense light.

iii- Absent light reflex: with vermiform movement

iv- Tonic and segmental near reflex.

(the pupil responds slowly to near & redilatation is slow).

v- Accommodation is diminished.

NB. If assimilated with absent knee jerk >> Adies Holmes syndrome

Etiology:

Damage to the ciliary ganglion or short posterior ciliary nerves

(post-ganglionic parasympathetic fibers = efferent). Usually viral.

Diagnosis: Give 1/8 % (0.125 %) pilocarpine :

- Normal eye → not react.
- Adie's pupil → constrict due to denervation hypersensitivity.

NB. In the ciliary ganglion → 1 fiber for light reflex.

→ 30 fibers for near reflex.

Hutchinson's pupil
(Sign of lateralization)

In subdural Hge → if Hge is increasing, coma will deepen and pupils show the following changes

	Ipsilateral pupil	Contralateral pupil
Early stage	Constricted	RRR
Advanced	Dilated and irreactive	Constricted.
More advanced	Dilated irreactive	Dilated and irreactive

- Constriction → Dilatation in the ipsilateral pupil , followed by Constriction → Dilataeion in the contralateral pupil.
- A dilated pupil + increased coma →is an indication for cerebral decompression surgery (افضي الدم ده)

Horner Syndrome: (Oculo-sympathetic palsy): See atlas page (7)

- **pathway: ??**
- **Cause:** lesions of sympathetic innervation of the eye, due to
  - *Central :* vascular or demylinating diseases.
  - *Pregangloinc* :Bronchogenic carcinoma ,neck injury or surgery .
  - *post ganglionic:* Caverous sinus thrombosis.
- **Characters:** 3(osis) ∈ ptosis, Miosis, Anhydrosis
  1- Mild Ptosis (due to weakness of Muller ms.).
  2- Miosis (unopposed action of sphincter pupillae).
  3- Anhydrosis (reduced sweating): if the lesion below the superior cervical ganglion (preganglionic)
  4- Apparent enophthalmos. due to ptosis
  5- Heterochromia iridis .
- **Pharmacology:** كهن- بن

Afferent pupillary defect APD = Paradoxical pupil

♦ **Cause:** Unilateral optic atrophy- papillitis – retrobulbar neuritis or extensive retinal damage (CRAO – Total RD)l

♦ **Characters:**

- Consensual reflex: normall
- Direct reflex: • affected → Relative APD ((marcus-Gunn pupil))
  • absent completely → Absolute APD ((Amurotic pupil))
- No anisocoria (both pupils are equal in size) .

♦ **Diagnosed by: *Swinging flash light test* :**

When a light is swinged from normal side to diseased eye → both pupils dilate instead of constricting [dilatation is due to withdrawing light from normal eye that outweighs يغلب constriction produced by stimulating the normal eye].

Causes of Miosis and Mydriasis:

Normal pupil Size: 3-4 mm

	Miosis (pupil <2 mm)	Mydriasis (pupil> 7mm)
1- Physiological	i- Light and Near reflexes. ii- During sleep (parasymp.). iii- 3rd stage of anesthesia. iv- Old age (Sclerosis of constrictor) & newly born (dilator developed at 10 months). v-Corneo-pupillary reflex.	i- Withdrawal of light . ii- Emotion (sympathetic). iii- 2nd stage of anesthesia. v- Cilio-spinal reflex : due to pinching the skin on one side of the neck → mydriasis.
2- Drugs	- Miotics ..... - Morphine poisoning & addiction	- Mydriatics ..... - Datura poisoning (atropine source).
3- Local diseases	- Trauma (mild). - Iridocyclitis due to irritation. - Hypermetropia. - puncture of AC(paracentesis)? due to - Hypotony → engorgement of iris BVs. = ↓IOP	Trauma (severe). Acute congestive glaucoma. Myopia, buphthalmos . Blind eye: CRAO, OP. Atrophy ماشي ف الضلمه
4- Neurological	i- Horner's syndrome. ii- Argyl Robertson pupil. iii- Pontine hge, meningitis, C.S. thrombosis ii,iii → irritation of EWN. iv- Irritative (early)stage of cerebral compression (Hut.)	i- 3rd n. palys. ii- Adies pupil iii- Coma except in : 1- morphine poisoning. 2- Pontine Hge. ((1,2 → coma+ miosis)) iv- paralytic (late) stage of Hut.Pupil. iiv- 4th stage of anesthesia.

Neuro-ophthalmology

Anatomy:

1- Photoreceptors: Rods & cones
which synapses with bipolar cells → ganglion cells .

2- Axons of ganglion cells run in the nerve fiber layer of retina & converge to form the **Optic nerve.**

3- At the Optic chiasma: the nasal fibers decussate to reach the **Optic tract** on the opposite side, while the temporal fibers pass uncrossed to the ipsilateral tract. Finally the fibers reach the LGB (except the afferent pupillary fibers which terminate in the pretectal nucleus), where they synapse so, the op. tract carries ipsilateral temporal and contralateral nasal fibers.

4- From **the Lateral Geniculate body** (LGB), new axons spread out over broad area of Parietal (from upper retina) & temporal lobes (from lower retina) to form the optic radiation.

5- Fibers in **Optic radiation** end in the visual cortex, which includes:

- Area 17 (1ry visual area - striate cortex).
- Areas 18 & 19 (visual association areas).

Physiology:

The visual pathway has three order of neurons excluding the Photoreceptors:

1- 1st order neurone → the bipolar cells.

2- 2nd order neurons → the ganglion cells & their axons in the nerve fiber layer & the optic nerve till the LGB.

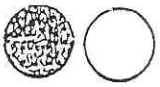
3- 3rd order neurone → extends from the LGB to the visual cortex along the optic radiation .

N.B. each occipital cortex receive temporal fibres from same side & Nasal from opposite side

N.B. right occipital cortex responsible for Left field

I- Visual pathway lesions

See atlas page (177)



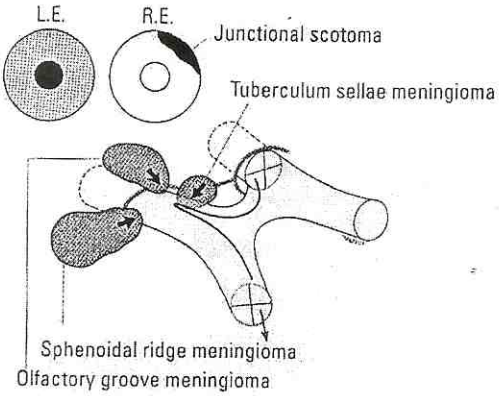
1- Op. nerve Lesions: Ipsilateral blindness + ADP.

NB. Lesion at the junction of the optic nerve with the chiasma leads to :

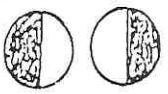
- Ipsilateral cental scotma : due to otic nerve compression

&- Contralateral upper temporal defect (junctional scotoma) :

due to damage of the anterior knee of wilbrand ؟؟؟؟؟ سهله مش كده

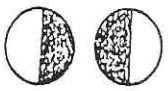


2- Op. chiasma lesions:



* **Bitemporal hemianopia** (nasal fibers damage).

e.g. Pit. Gland tumor.



* **Binasal hemianopia** (temporal fibers damage).

2 lesions must exist:

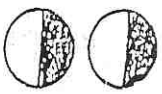
1- Distension of 3rd ventricle e.g. Hydrocephalus (↑ CSF).

2- Bilateral cavernous sinus thrombosis.

3- Aneurysm of the 2 internal carotid arteries.

NB. Pupil in chiasmal lesions → APD (Wernicke hemianopic pupil)

3- Op. tract lesions: Contralateral. Homo. Hemianopia CHH



(+ Loss of light reflex غلط).

- Ant. 2/3 → Wernicke hemianopic pupil

affected on one side only (APD).

& on the other side the reaction is normal

- Post. 1/3 → normal light reflex.

AS the pupillary fibers leaves the ant. 2/3 of optic tract to pretectal nucleus.

NB مهمه. CHH caused by optic tract lesion is incongruous

NB مهمه. The more posterior the lesion the higher the congruity.

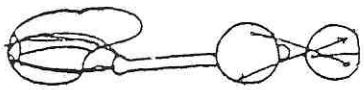
NB مهمه. Optic tract lesion cause optic atrophy

NB. CHH:

Means loss of temporal field (1/2) in one eye & the nasal in the other, i.e. Pt cannot see on one side

4- Lat. Gen. body: Contralateral Homonymous hemianopia CHH
(+ preserved light reflex).

5- Op. radiation:



- Upper fibers (parietal lobe) → Contralateral
Homo.inferior quadrantic defect
(pie in the floor شاهين)
(+ preserved light reflex).

- Lower fibers = **Meyer loop** (temporal lobe)
→ Cont. Homo.superior quadrantic defect
(pie in the sky شاهين)
(+ preserved light reflex).

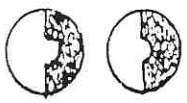
(لو ال2 يبقي العيان مات)

NB. Lesions of the optic radiations don't produce optic atrophy, because these fibers are the third order neuron that originate from the LGB

6- Occipital cortex:

→ Contralateral Homo. hemianopia with macular sparing,
Due to:

i- Large area of representation in the occipital cortex.
ii- Bilateral representation of the macular fibers.
iii- Double blood supply :
(From the middle and posterior cerebral arteries).



NB مهمه. Damage to the tip of occipital cortex(supplied by middle cerebral artery)→ homonymous macular defect

II- Ocular manifestations of Brain Tumors

A) General signs of increase ICT:

1- Triad of: headache, projectile vomiting, blurring of vision (due to papilledema).

2- False localizing signs: e.g. 6th n. palsy (squint & diplopia) being compressed against apex of petroués pone

B) Focal signs:

Which help in localization:

1- Frontal lobe: → Foster-Kennedy syndrome.

2- Parietal lobe: → Contralat. Inferior quadrantic defect+ hemiplegia+ acalculia , agraphia & left right disorientation.

3- Temporal lobe: - Contralateral superior quadrantic defect.
- Hemiparesis + visual hallucination (Agnosia).

4- Occipital lobe: - Contralat. Homo. Hemianopia with macular Sparing.
- Visual hallucination.

5- Cerebellum: - Nystagmus (increased on looking to the side of lesion).
+ Ataxia

6- Pituitary: - Bitemporal hemianopia + endocrinal changes (hypo or hyper pituitary.)

7- Desaturation of colors. (Optic n. atrophy مرحلة قبل ال)

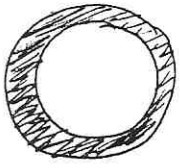
8- APD (afferent pupillary defect).

9- Ocular motor affection : 3rd , 4th & 6 th nerves.

10- Transient attack of loss of vision.

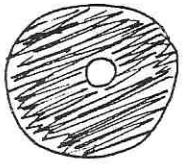
III- Abnormalities of the field of vision

1) Concentric contraction:



1- Retinitis pigmentosa
2- OAG .
3- Optic atrophy.
4- Quinine amblyopia.
5- High myopia
6- Miotics
7- Incipient cataract.
8- Colored CL

2) Tubular field:



1- Advanced retinitis pigmentosa.
2- Advanced POAG.
3- CRAO (with preserved cilio-retinal artery).

Scotoma:

Definition:

Field defect surrounded by normal field (island of blindness).

Classifications:

1- Absolute and relative:

a- Absolute: no light sensitivity in the affected area.

b- Relative: defective field for certain colors or low light intensity.

e.g. * Red & green in optic neuritis.

* Blue in papilloedema.

2- Positive or negative:

a- Positive: patient is aware about his defect.

b- Negative : patient is not aware about his defect.

(blind spot is the best example of absolute negative scotoma).

3-According to the site:

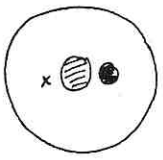
a) **Central scotoma:** involving the fixation point (macula):

i- Papilledema (relative scotoma for blue).

ii- Optic neuritis & toxic ambylopia (relative scotoma for red & green).

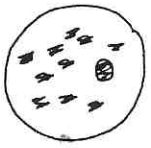
iii- macular lesions : hole, Hge, cyst.

iv- Central chorio-retinitis.



b) Centro-caecal scotoma:

between the blind spot& point of fixation (macula) as in tobacco amblyopia due to destruction of papillo-macular bundle by cyanide.

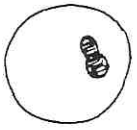


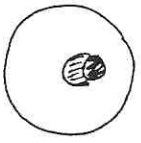
c) Disseminated Scotoma:

i- Disseminated choroiditis (+ve then –ve) due to accommodation by brain .

ii- Disseminated chorio-retinal degeneration in high myopia (-ve from the start as it gradually occurs) .

d) Enlargement of the blind spot:





i- Papilledema.

ii- Siedle scotoma in OAG .
(the enlargement is vertical)

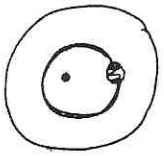
iii-Temporal crescent in myopia.

iv- Perpapillary Myelinated n. fibers

v- Juxta-papillary choroiditis.

4- According to the shape:

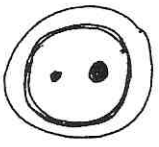
i- Arcuate scotoma : in OAG



ii- Ring (annular) scotoma: in.

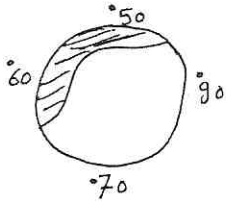
a) OAG (in the central field , continuous with the blind spot, with nasal step)

b) Ret. Pigmentosa:(in the peripheral field , without nasal step)



NB.

1)Limits of the normal visual field
- Up → 50° (eye brow)
- Down → 70° (maxilla)
- In → 60° (nose)
- Out → 90°



هل هي هي مع العمر؟؟

With age the field becomes smaller

2) Blind spot of Mariotte : corresponds to the optic disc, present between 10 & 20 degree on the temporal side of fixation, it is the best example for absolute negative scotoma.

Wernicke hemianopic reaction:

Where stimulation of the blind part of the retina → no reaction.

But : stimulation of the seeing part of the retina → reaction.

Causes :

1- Lesion in the ant. 2/3 of optic tract.

2- Chiasmal lesion.

Hemianopia (نصف العمي):

- Definition: defect involving half of the visual field.
- Types:

(1) Heteronymous H.: loss of non-corresponding halves of both fields i.e. two nasal or two temporal:

- Bitemporal H.(قول أسبابها).
- Binasal H. (قول أسبابها).

(2) Homonymous H.: loss of temporal half of one field & nasal half of the other field i.e. the pt. cannot see on the Rt side ot Lt side.

Causes: lesions of : 1- Optic tract.
2- LGB.
3- Optic radiation.
4- Occipital cortex lesion.

* تعرف تفرق ما بينهم؟

Medical ophthalmology

Endocrinal :

1- DM. 2. Thyrotoxicosis.

Vitamin deficiency:

1. Vit. A: i. Xerosis ii. Corneal Ulcer iii. Keratomalacia
iv. Madarsosis v. Night blindness

2. Vit. B:

B1 → Op. neuritis, ocular ms. palsy, nystagmus.

B2 → Corneal vascualrization

B6 → Op. neuritis, ocular ms palsy, cataract.

B12 → Op. neuritis, ocular ms play, cataract.

3. Vit. C: Scurvy +

1- Cataract. 2- Decrease wound healing. 3. Hge (lid.& conj.) .

4- Vit. D: i. Cataract ii. Myopia.

5. Vit. K → Bleeding tendencies (conj. ,retina, orbit, during op.).

Infective diseases:

I- Bacteria:

- Diphtheria → membranous conj., Paralytic squint.
- Pneuomina → hypopyon ulcer.

II- Viral:

- **H. Simplex:** 1ry blepharo-conjunctivits, dendretic ulcer, iritis, ARN, optic neuritis.
- **H. Zoster:** HZO.
- **Rubella:** total cataract.
- **Influenza** : Dendretic ulcer, iritis, Op. neuritis , conjunctivitis , keratitis.

Demyelinating diseases:

1. Multiple sclerosis:

- Papillitis, retrobulbar N.
- 1ry op. atrophy.
- Paralytic squint due to EOMs paralysis.
- supranuclear gaze palsy.
- Field defect (involving chiasma & op. tract).
- Nystagmus due to cerebellar affection.

2. Neuromyelitis optica (Devic's disease):

- Bilateral optic neuritis.
- Paraplegia due to spinal cord affection.

Important symptoms

Diminution of vision:

(I.) Sudden loss of vision: 1- CRAO. 2. Amaurosis fugax.
3- Hysteria.
4. Trauma to the eye : → - Rupture globe .
- Massive vit. Hge.
- Avulsion of optic n.

(II) Rapid decrease of vision:

a. In few hours: 1. CRVO. 2. Acute congestive glaucoma.
3. Commotio R.
4. RD, choroiditis. 5. Vitreous hge .

b. In few days: 1. Keratitis, iritis 2. RD, Choroditis.
3. Retinopathy. 4. Optic neuritis.

(III) Gradual paniless diminution of vision: (Within months or years):

1) Cornea: keratoconus.
2) IOP: Primary open angle glaucoma.
3) Uveal tract: Chronic iridocyclitis.
4) Lens: Senile cataract (world wide).
5) Vitreous: vitreous opacities.
6) Retina: Retinopathies, Retinitis pig., age-related macular degeneration .
7) Optic nerve: Chronic optic neuritis, 1ry optic atrophy.
8) Amblyopia : In unilateral squint.
9) Degenerative myopia.

Character	Senile cataract	OAG	1ry optic atrophy
Age	Above 50 yrs.	Above 50 yrs	Above 35 yrs.
Sex	Equal	Equal	More in males
Other symptoms	Fixed black spots	Field changes	Of the cause
Pupil color	Grayish	Normal	Normal
Direct light reflex	Present	present	Absent (dilated pupil)
Red reflex	Abnormal	Normal	Normal
Fundus	Normal (if seen)	Cupping	White disc-shallow cup
Field	Normal(if tested)	Field changes	Peripheral contraction
Tension	Normal	Raised	Normal
Tonography	Normal outflow	Impaired outflow	Normal outflow

- **What are the main causes of diminution of vision in infants?**
  (1) Congenital glaucoma.
  (2) Congenital cataract.
  (3) Progressive myopia.
- **What are the main causes of painful ↓ of vision?**
  1- keratitis 2- iritis 3- ACG 4- Endophthalmitis & panophthalmitis.
  5- OIS (ocular angina) مهمه

The opsias:

i. Metamorphopsia: Objects appear distorted.

ii. Macropsia: objects appear large.

iii. Micropsia: objects appear smalle.

* Causes: distortion of the macular rods: and cones as in:

- Retinitis & Choroiditis. - RD - Commotio R.

iv- Photopsia: seeing flashes of light.

* Causes: stimulation of rods and cones mechanically or by inflam. exudates as in : Retinitis, Choroiditis, RD, High myopia.

v- Chromatopsia: colored vision:

1. Erythropsia (red vision): occurs after cataract extraction.
2. Xanthopsia (yellow vision): occurs in jaundice.
3. Cyanopsia (blue vision): occurs in hysteria.

★ Amblyopia = lazy eye:

- Definition: Amblyopia is diminution of vision with no organic defect to explain it.
e.g. pt sees 1/60 & his eye is free.

- Causes:

1. Strabismus amblyopia: In order to avoid diplopia, the patient will suppress the deviating eye which over time will result in amblyopia.
2. Anisometropic amblyopia: is the result of marked difference in the refractive error between the two eyes. The patient suppresses the blurred image of one eye causing amblyopia.
3. Deprivation amblyopia(exanopsia):
media opacities will degrade the image from the affected eye leading to amblyopia.
e.g. defective vision in early life as in congenital cataract , complete ptosis.

- Diagnosis:

(1) No improvement of vision with pin-hole test or eye glasses.
(2) Worth 4-dot test.
(3) Cover test.

(4) Visuoscope.

- Treatment: as early as possible before the age of 9 yrs.

(1) Occlusion of the eye: to improve vision in amblyopic eye: See atlas page (171)

1) *Of fixing eye*: in ambylopia with central fixation.

2) *Of amblyopic (Squinting) eye*: in amblyopia with eccentric fixation.

(2) Pleoptics: to develop full vision in amblyopic eye with eccentric fixation by foveal stimulation:

1. After image method(visuoscope → ينظر علي ستارة بيضاء).
2. Direct foveal stimulation method.

(3) Orthoptics: to develop binocular vision .

(after occlusion and pleoptic treatment).

Amaurosis:

- Definition: complete rapid loss of vision in one or both eyes without organic lesion in eye → occurs within hours and recovers within hours.

- Causes:

(1) Uraemia : toxic effect on the occipital cortex (pupillary reaction is normal).

(2) Meningitis.

(3) Hystrical.

(4) Amaurosis fugax .

What is amaurosis fugax?

*** Definition:** Momentary sudden loss of vision in eye → occurs within minutes and recovers within minutes.

*** Causes:**

1- Central retinal artery spasm.

2- Papilloedema.

3- Sudden rise from sitting position → postural hypotension.

4- Migraine

5- Raynaud's disease .

Night blindness:

It means difficulty to see in dim illumination.

Causes : 1- General: -Vit. A deficiency .

- Liver diseases & alcoholics→ disturb Vit. A metabolism.

2- Local:

- Hysterical.
- Peripheral lens opacities (incipient cataract).
- High myopia. - OAG.
- R. Pigmentosa. - Siderosis bulbi → pseudo-retinitis pigmentosa.

3- Congenital: most common type (MCQ).

Day blindness:

it is difficulty to see in bright illumination.

Causes: 1- Central corneal opacity.

2- Central lens opacity (nuclear).

3- Central retinal lesion (macular degeneration).

Color blindness:

- **Causes:** 1- Congenital: (most common type), common in females.
  2- Acquired: as in papilledema and papillitis & macuar lesions .
- **Types :**

1- Anomalous trichomacy : Weakness of one of 3 cones

* Prot-anomaly.
* Duter-anomaly.
* Trita-anomaly.

2- Dichromatic vision: Absence of one of the 3 cones

* Prot-anopia.
* Duter-anopia.
* Trita-anopia.

3- Monochromatic vision : Absence of 2 of the 3 cones (there is only one cone).

4- Achromatic vision: complete failure of color discrimination.

- **Tests (Colour vision tests give an idea about macular function):**

(1) **Ishihara test(Isochromatic):** plates with coloured figures (used to test colour blindness). See atlas page (162)

(2) **Coloured matching test:** Colored beads خرز & wool صوف (child)

(3) **Coloured glass discs:** red , green & blue discs (in mature cataract).

See atlas page (162)

Diplopia (Double vision):

Types : 1- Binocular diplopia: each image is seen by one eye (i.e., diplopia disappears if either eye is covered e.g. in:

1) Physiological.
2) Paralytic sq.
3) Anisometropia: if corrected with glasses.
4) Mysthenia gravis. 5) Proptosis.
6) Restrictive eye movement :
   - symblepharon, orbital tumors , after RD , Pterygium op. Or blow out fracture.

2- Uniocular diplopia:

Presents even if the normal eye is covered e.g. in :

i. Incipient cat.
ii. Subluxation.
iii. Iridodialysis.
iv. Irregular astigmatism.

Q. what are the causes of diplopia after trauma ?

1- Paralytic sq. 2- Incipient cat. 3- Subluxation 4- Iridodialysis.

Ocular pain, discomfort, headache:

1. **Discomfort:** in conjunctivitis.
2. **Severe pain:** in corneal ulcer (stitching), iritis (Neuralgic), acute gl. (Bursting).
3. **Eye strain:** asthenopia.
4. **Itching pain :** 1- Allergic conj. (especially vernal).
   2- Blepharitis (especially angular) .
5. **Headache:** referred with hypertension, increase ICT, sinusitis, Refractive errors, Latent squint, Inflammation of eye of adnexa & glaucoma.
6. **Orbital pain:** - Retrobulbar neuritis - Eye strain. - Orbital trauma - Orbital cellulitis - Myositis. - Orbital periostitis.

♦ **What are the types and causes of ocular pain?**

(1) Discomfort: asthenopia.
(2) Burning: conjunctivitis.
(3) Itching: blepharitis and spring catarrh.
(4) Sandy (gritty) sensation: trachoma (PTDs).
(5) Pricking (Stitching): corneal ulcer or foreign body.
(6) Throbbing: stye and endophthalmitis.
(7) Bursting: acute glaucoma.
(8) Aching (eye-ache): Errors of refraction.
(9) Neuralgic: acute iridocyclitis.
(10) Referred: To temple: Acute glaucoma.

♦ **What are the causes of coloured haloes around light?**

(1) Acute glaucoma (corneal oedema).
(2) Incipient cataract (fluid droplets in lens).
(3) Acute conjunctivitis (mucous beads on cornea).

Important signs

I- Abnormal depth AC:

- **AC normal depth** = 4.2 ml (from the back vertex of the cornea → vertex of the lens).
- **Method :** pachymetry or A-scan .

i. Shallow:

1- Intumescent cat. 2. Congestive gl.
3- Small eye (hypermetropia, microphthalmos).
4- Iris bombé (ACG).

ii- Absent AC: - After intra-ocular operation with wound leak.
- Corneal fistula , rupture corneal ulcer.

ii- Deep:

1- Hypermature cat. 2- Buphthalmos. 3- High myopia.
4- Aphakia. 5- Post. Dislocation.
6- Keratoconus. 7- Keratoglobus.

iii- Iregular:

1. L. Adherent 2- Keratectasia 3. Ant. Staphyloma.
4. Subluxation. 5. Synechia.

- **Abnormal contents of the AC ?**

- Flare (cells). - hypopyon . – hyphema.
- lens (anterior dislocation or IOL). – FB.

II- Iris signs:

i. Color changes:

1- Muddy (iridocyclitis). 2- Grey (atrophic patches) .
3- Pink (albino). 4- Heterochromia (different color).

ii- Tremulous iris: (irido-donesis) = Ant. Post. shacking movement.

1) Large eye, as in high myopia and buphthalmos.
2) loss of support of iris by the lens as in:
   - Aphakia- subluxation- post. Dislocation.
   - Hypermature cataract (lens shrink).

III- Signs in the pupil:

i- Irregular pupil:

1. Oval: acute glaucoma.
2. pear shape: (a) Anterior synechia. (b) Leucoma adherent.
3. D-shape: Iridodialysis.
4. Key-hole: Key- hole iridectomy.
5. Festooned on dilatation: posterior synechia.
6. Coloboma of iris. 7. Iridectomy 8. partial ant. Staphyloma.
9. Trauma : - Pupillary laceration.
   - Iridodialysis

ii- Colour:

1. White: (a) mature cataract. (b) Intumescent cataract.
2. Grey: (a) immature cataract. (b) Nuclear sclerosis.
3. Yellow: (a) retinoblastoma. (b) Nuclear sclerosis.
4. Brown: brown (black) cataract.
5. Green or blue: Acute glaucoma.

6. Red: albinism.

IV- Red reflex:

- **How u can test the red reflex?**

Reflected light (of light source behind left side of patient) from a perforated plane mirror held by examiner at one metre → RR is seen by examiner.

- **What is the principle of the RR?**

RR is mainly due to colour of circulating blood in choroid.

- **What is the importance of the RR?**

(1) Opacities in ocular media:

1. Fixed balck defects: corneal or lens opacities.
2. Floating (moving) black defects: Aqueous or vitreous opacities.

(2) Intraouclar diseases:

1- Yellow (endo. , panophthalmitis & retinoblastoma)

2. Grey (R.D.)

3- Dark Grey (cat)

4. Black: Vit. Hge & cataracta nigra.

V- Causes of lens pigmentation ?

1- Siderosis bulbi.

2- Chalcosis bulbi.

3- Iridocyclitis.

4- Vossious ring.

5- Glaucoma.

6- Nuclear cataract.

- **What is slit – lamp?**

Instrument used for examination of eye:

1- Sli lamp alone: to examine anterior segment of eye (cornea, AC, iris, lens and anterior part of vitreous) and its adenxa (lids, conjunctiva and lacrimal puncti).

2- Slitlamp + gonioscopic lens: As Goldmann 3- mirror contact lens in; gonioscopy.

3- Slit- lamp + applanation tonometer: In tonometry.

4- Slit –lamp + fundus lens: As Goldmann 3- Mirror contact lens for fundus Examination & Volk lens

OPHTHALMIC LASER

- **Definition:** LASER is **l**ight **A**mmplification by **S**timulated Emission of **R**adiation.
- **Principles:**

(1) lasing material (laser generators): 1- Argon 2- Diode 3- YAG 4- Excimer.

(2) Energy: 1- Strong light. 2- Electric current.

(3) Laser beam: the laser material is stimulated by energy to emit the laser beam which consists of very strong waves of monochromatic light (of only one wave length).

(4) Types of laser:

1- Laser photocoagulation(heat coagulating laser): monochromatic light of the laser beam is absorbed by a pigmented target(as pigment epithelium of the retina) which transform that light into heat:

- Argon laser - Diode laser - YAG laser photocoagulation.

2- Surgical laser (Cutting laser)

- YAG laser photodisruption - Excimer laser (Ablative decomposition laser)

- Uses of LASER in Ophthalmology:

a) Lid :

i- Removal of warts and papillomas --> Co_2 laser is used.

ii- Rubbing lashes.

b) Cornea:

i- Photo-therapeutic keratectomy (PTK) : for superficial opacities.

ii- Removal of corneal sutures.

c) Errors of refraction:

i- Excimer laser photo-ablation (LASIK & PRK) .

ii- Holmium laser thermoplasty : for hyperopia.

d) Cataract:

i) Anterior capsulotomy in cataract extraction (before ECCE).

ii) YAG laser capsulotomy : to treat PCO (following ECCE).

iii) Phacolaser :Erbium-YAG laser: to remove the cataractous lens.

e) Glaucoma:

i) Laser Iridotomy using YAG laser: in ACG, Drawn up pupil.

ii) Argon or diode laser Trabeculoplasty in some cases of OAG.

iii) Cyclo-photocoagulation in cases of neovascular glaucoma.

iv) Scanning laser Ophthalmoscopy to visualize the optic disc in OAG.

f) **Retinal :** using Argon or diode laser in:

i) Sealing retinal tears.

ii) Treatment of diabetic retinopathy & CRVO.

iii) Areas of peripheral retinal degenerations in cases of high myopia.

iv) Photodynamic therapy (PDT) : in CNV.

iv) Trans-pupillary thermotherapy (TTT) in retinoblastoma & M.M.

- **Complications of laser:**

1- Opacification of the cornea , lens, vitreous.

2- Hemorrhage : hyphema or retinal hge.

3- Foveal damage : in non-cooperative pt.

4- Increased tension : usually transient.

♦ What are the ocular manifestations of hysteria?

Hysteria is a type of psychoneurosis and is due to unresolved problem:

(1) Visual disturbances:

- Hysterical blindness or amblyopia.
- Night blindness.

(2) Hysterical ptosis.

(3) Hysterical asthenopia.

(4) Hysterical photophobia and blepharospasm.

♦ How can you diagnose malingering?

A person who claims to have a pathological conditions as visual defect can be diagnosed by:

(1) No organic disease with abnormal personality.

(2) High convex lens (+ 10 DS) before good eye → if the pt can see the distant types on the chart , he is malingering .

(3) Catford drum (Optokinetic nystagmus).

(4) Prism : the eye will deviate to regain fixation.

♦ What are the types and causes of ocular discharge?

1. Watery: Acute follicular conjunctivitis.
2. Mucoid: catarrhal conjunctivitis.
3. Mucopurulent: mucopurulent conjunctivitis.
4. Purulent: purulent conjunctivitis.
5. Blood stained: Diphtheritic conjunctivitis.
6. White ropy (thready and sticky) : spring catarrh.

♦ What are the main causes of disfigurement of the eye?

- Ptosis. - pterygium - corneal opacities. - squint. - staphyloma. - proptosis.

♦ Calcification in ophthalmomogy

Ocular:	Orbital:
1- Retinoblastoma 2- Band shaped keratopathy. 3- Phthisis bulbi. 4- Haemangioma of the retina or the choroid. 5- Optic nerve drusens.	1- Lacrimal gland epithelial tumors. 2- Haemangiom.a 3- Meningioma. 4- Phelpolith.

NB. Calcifications is Completely absent in:

Lymphoma ,Secondaries ,Glioma of optic nerve.

♦ What is asthenopia (eye strain)?

- ***Definition***: discomfort with ocular symptoms usually after near work.

- ***Types and causes:***

(1) Accommodative: (Due to excessive use of ciliary ms.)

as in hypermetropia and presbyopia, Astigmatism , Anisometropia.

(2)Muscular : (Due to spasm of EOMs)

As in latent squint .

- ***Clinical picture:***

- **Symptoms:**

1) pain (eye-ache) 2) lacrimation 3) Redness 4) Photophobia

5) Frequent blinking 6) Dizziness & vertigo(rare)

- **Signs :**

1- Lid & conj.: Blepharo-conjunctival hyperaemia.

2- Cover test: Muscular asthenopia disappears if one eye is covered (unlike other types).

◆ **What is photophobia?**

Inability to open eyes in light & occurs in:

1- Acute conjunctivitis

2- Corneal ulcer 3- Photophthalmia

4- Acute iridocyclitis 5- Congenital glaucoma

6- ACG 7- FB (corneal or conjunctival)

◆ **What are the ocular emergencies?**

(1) Ocular injuries: as rupture globe, chemical burn, retained FB.

(2) Acute congestive glaucoma: As loss of vision may occurs within 24 hours due to optic atrophy.

(3) progressive corneal ulcer: As it may perforates.

(4) Severe infection: as endo and panopthalmitis, Cav. Sinus thrombosis, Orbital cellulitis.

(5) Central retinal artery occlusion: as it leads to loss of vision within 30 minutes.

(6) Spreading retinal detachment: As macula may be affected.

(7) Ant. dislocation of the lens.

◆ **How can you test for corneal irregularity?**

1. Placido disc: irregular circles as in window reflex.
2. Retinoscopy: spinning red reflex.
3. keratometer: Accurate.

◆ **Why visual acuity is recorded at 6 metres?**

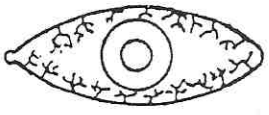
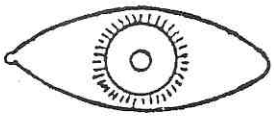
Because at 6 meters:

- Light rays reach parallel.
- Accommodation of patient is relaxed.

◆ **What is 6/18 vision:**

It is the vision of eye which can see at 6 meters what normal eye can see at 18 meters distance.

Table DD of conjunctival and ciliary injection See atlas page (49)

Character	Conjunctival injection	ciliary injection
(1) blood vessels	1. Bright red in color	1. Dark red.
	2. tortuous and dilated	2. thin
	3. Moves freely with movement of conjunctiva	3. Cannot move, but conjunctiva moves over them.
	4. Empty, and slowly fill on release of pressure applied on the lower lid	4. fill at once.
	5. More marked at the fornix	5. More marked at the limbus
	6. Vessels are seen (not blurred).	6. Vessels are not constricted by adrenaline
	7. Vessles are constricted by adrenaline.	7. Vessels are not constricted by adrenaline.
(2) origin:	Posterior conjunctival vessels	Anterior ciliary vessels
(3) Cause:	Conjunctivitis	Keratitis, iridocyclitis ,acute glaucoma
		

♦ **What is the difference between ciliary injection and congestion?**

Both are usually called ciliary injection but scientifically:

1- Ciliary injection: arterial dilatation (hyperaemia) in inflammatory conditions as acute iridocyclitis and corneal ulcer.

2- Ciliary congestion: venous dilatation (engorgement) in ACG.

♦ **What is the blind spot of mariotte?**

Blind part of fundus which corresponds to optic disc and is:

1. Temporal to fixation point.
2. Below horizontal meridian.
3. Absolute (no light perception in affected area).
4. Negative (patient is unaware of it).

♦ **What is X-ray examination for the eye?**

1) Plane X-ray:

1- Orbital fractures and diseases.

2- Localization of intraocular foreign bodies with aid of metallic limbal ring.

2) Dacryocystography: X-ray with radio-opaque dye (lipidol) to detect obstruction of lacrimal passage.

♦ **What is computerized tomography (CT)?**

Method which localizes ocular and orbital lesions.

♦ **What is magnetic resonance imaging (MRI)?**

- **Indications:** complementary to computured tomography in orbital lesions (but more important in cranial lesions as it outlines soft tissues from bones).
- **Advantage:** No harmful effect of ionizing radiation.
- **Contraindication:** magnetic intraocular or intraorbital foreign bodies .

♦ **What are the uses of fluorescine in ophthalmology? د.عمرو عواره**

1- Fluorescine test for epiphora.

2- fluorescine test for corneal ulcer or abrasion.

3- applanation tonometry.

4- fluorescine angiography.

5- Fitting of hard contact lenses.

6- Siedle test : for corneal fistula or perforation

7- Tear film beakup time test.

- It may be contaminated with pyocyaneus bacilli (with rapid growth) and so fluorescine solution must be freshly autoclaved or use strips or disposable units.

♦ **What are the uses of Cryotherapy in ophthalmology?**

1- Lid : Congenital trichiasis.

2- Conj.: Papillae of spring catarrh.

3- Cornea: Cryo-cautery.

4- Lens: Cryo-extraction of the lens.

5- **Glaucoma:** Cyclo-cryotherapy.

6- **Retina:** RD for sealing of retinal tears

7- Errors: - Epikeratophakia - keratomilleusis

8- **Intra-ocular tumors:** in small tumors & seeing eye.

- **What are the ocular manifestation due to UV rays exposure بنها؟**

  1) conj: pingeucula – spring catarrh- ptergyium

  2) Corena: activation of HSV→dendretic , photophthalmia

  3) Lens: senile cataract

- **What is the blood supply of the eye ball?**

  1) Ciliary circulation: see uveal tract.
  2) Retinal circulation: see retina.

- **What are the avascular parts of the eyeball and their nutrition?**

  1) Cornea diffusion from:
     1- Limbal capillaries.
     2- Aqueous
     3- Tear film.
  2) Lens: Diffusion form aqueous.
  3) Vitreous: diffusion from:
     Choroid, ciliary body & retina.
  4) Fovea: Diffusion form choriocapillaries.
  5) Outer layers of retina: As fovea.

THE EYE AND SYSTEMIC DISEASES

Many systemic diseases, as well as drugs used to treat them, have significant ocular manifestations. The most common are:

INFECTIOUS DISEASES

1. ***Kerato-Conjunctivitis:*** may be
   a- Acute: in many viral infections as measles, chicken pox and rubella.
   b- Chronic granulomatous conjunctivitis, as TB and syphilis.
2. ***Sub-conjunctival hemorrhage:*** may occur in hemorrhagic fevers as in rift valley fever, Ebola virus, and spirochaetal diseases.

3. ***Uveitis:*** may occurs in:
   a- Many viral infections (non-specific).
   b- Metastatic purulent uveitis (endophthalmitis) can occur with IV drug abusers.
   C- Granulomatous uveitis occurs with TB and syphilis.

4. ***Retinitis:*** may occusr in:
   a- Cytomegalovirus infection in AIDS patient .
   b- Acute retinal necrosis can occur with herpes simplex retinitis.
   c- Congenital rubella can produce a retinitis pigmentosa-like disease in the newly-born (salt and pepper fundus).
   d- Cysticercosis may give rise to sub-retinal parasitic cysts.
   e- Toxocara species can produce a retinal granuloma in children.

5. ***Optic neuritis*** and subsequently optic atrophy can occur with encephalitis and meningitis of viral or bacterial origin. Optic atrophy may be a complication of therapy as with the use of ethambutol (anti-tuberculous drug).
6. **Orbital parasitic cysts** : may be seen in hydatid disease (Echinococcus granulosus) and myositis of the extra-ocular muscles with Trichenella spiralis infestation.

HEMATOLOGICAL DISEASES

1. ***Coagulation disorders*** :as hemophilia, thrombocytopenia and anti-coagulant therapy can produce hemorrhage anywhere in the eye, the most significant of which are vitreous and retinal hemorrhages.

2. ***Severe anemia:*** can produce pallor of the conjunctiva, retinal venous tortuosity ,retinal hemorrhages and optic disc edema.
   - Sickle-cell anemia : produce retinal arteriolar occlusions and retinal neovascularization.
   - pernicious anemia :optic atrophy.

3. ***Hematological malignancies: as leukemias and lymphomas :***
   (1) Anterior segment :
   - iritis from iris infiltration→ pseudo-hypopyon
   - Dry eye from lacrimal gland involvement.
   - Sub-conjunctival hemorrhage.
   - Spontaneous hyphema

   (2) Posterior segment :
   - Cotton woll spots retinal hges:due to vascular occlusion by leukemic cells
   - Roth spots: retinal hges with white centers (leukemic cells)
   - Peripheral neovascularization
   - Optic nerve infiltration.
   - Serous RD from choroidal infiltration.

   (3) Neuro: - Papillitis - cranial nerve palsies.

   (4) Protosis due orbital involvement .

4. Polycythemia : Thrombosis of CRA &CRV.

COLLAGEN DISEASES

They include rheumatoid arthritis, systemic lupus erythematosus, polyarteritis nodosa, scleroderma, polymyositis and Sjogren syndrome.

1. keratoconjunctivitis sicca: due to atrophy of the main and accessory lacrimal glands.
2. Keratitis, keratolysis, spontaneous sterile corneal perforations and scleritis are common manifestations of rheumatoid arthritis.

3. Iridocyclitis in pauciarticular juvenile rheumatoid arthritis.
4. Systemic lupus and polyarteritis produce retinal and choroidal vascular occlusions with the most common manifestation being cotton-wool spots of the retina.

ENDOCRINAL DISORDERS

1. Proptosis is seen in dysthyroid eye disease .
2. Retinal changes in diabetes .
3. Bitemporal hemianopia : as in pituitary chromophobe adenomas.

METABOLIC DISEASES

1. Metabolic cataract: as in DM, Cushing's syndrome , hypoparathyroidism, galactosemia, Lowe's syndrome and Wilson's disease (sun-flower cataract).
2. Subluxated lens: is seen in Marfan's syndrome and homocystinuria.
3. Corneal rings : are seen in Wilson's disease and hypercholesterolemia.
4. Corneal infiltrates and edema: is a common manifestation of mucopolysacharidosis and corneal crystals are seen in cystinosis.
5. Lid nodules (xanthomas) : common feature of hyperlipidemia.
6. Cherry-red spot of the fovea: is a common finding in sphingolipidosis and mucohlipidosis as Tay-Sachs disease and Niemanin-Pick disease.
7. Optic atrophy is common in many end-stage metabolic disorders.

NUTRITIONAL DEFICIENCIES

1. Vitamin A deficiency: causes xerosis of the conjunctiva, keratomalacia in severe cases and night blindness.
2. Severe vitamin B deficiency as in Ben-Ben and tobacco-alcohol amblyopia can produce ophthalmoplegia, various scotomas and finally optic atrophy.
3. Deficiency of vitamin C and other anti-oxidants may predispose to cataract and age-related macular degeneration.

Cardiovascular system & the eye

- **Systemic hypertension :**
  1- Hypertensive retinopathy
  2- CRAO & CRVO
  3- Ocular motor nerve palsy
  4- Ischemic optic meuropathy
- **Infective endocarditis :**
  1) Retinal occlusion
  2) Endophthalmitis from infected emboli
- **Giant cell arteritis :**
  1- Ischemic optic neuropathy resulting from post. Ciliary artery
  2- CRAO
  3- Anterior segment ischemia
  4- Cortical blindness

AIDS

1- Retinal micropathy.
2- Opportunistic infections : CMV retinitis – Cryptococcus choroiditis.
3- Kaposi sarcoma.
4- Neurological lesions :associated with intra-cranial infections & tumors.

- **What are the ocular manifestations of TB?**
  (1) Lids: Lupus vulgaris.
  (2) Lacirmal apparatus:
  1- Dacryoadenitis. 2- dacryocystitis.
  (3) Conjunctiva:
  1. Granulomatous conjunctivitis.
  2. phlyctenular conjunctivitis (allergic).
  (4) Cornea:
  1. Interstitial keratitis (allergic).
  2. phlyctenular keratitis (allergic).
  (5) Sclera: 1.Episcleritis (allergic) 2. scleritis.

(6) Uveal tract:

1. Granulomatous uveitis. 2. Exudative uveitis (allergic).

(7) retina: 1.Exudative retinitis (allergic)

2.periphlebitis retinae (Eale disease).

(8) Optic nerve: Optic neuritis (allergic).

(9) EOMs: Ophthalmolplegia (from basal meningitis).

(10) Orbit: periostitis.

♦ **What are the ocular manifestations of leprosy?**

(1) Lids and eye brows:

1) Lepromatous skin nodules.

2) Loss of hair of outer part of eyebrows and madarosis.

3) Ectropion and lagophthalmos.

(2) Conjunctiva: Granulomatous conjunctivitis.

(3) Cornea:

1) Superficial keratitis (Pannus). 2) Interstitial keratitis.

(4) Iris: Granulomatous iritis.

♦ **What are the ocular manifestations of $?**

(1) Congenital $:

1. Interstitial Keratitis. 2. Iridocyclitis. 3. Choroido-retinitis.

(2) Acquired $:

1) Lids:

1. Chancre (primary sore). 2. Gumma (in tarsus).

2) Lacrimal apparatus:

1. Chronic dacryoadenitis. 2. Chronic dacryocystitis.

3) Conjunctiva:

1. Chancre. 2. Gumma.

4) Cornea:

1. Interstitial keratitis (allergic). 2. Neuroparalytic keratitis.

5) Uveal tract:

1. Grnaulomatous uveitis. 2. Exudative uveitis (allergic).

6) Pupil: Argyl robertson's pupil.

7) Retina: Choroid-retinitis.

8) Optic nerve:

1. Optic neuritis 2. Primary optic atrophy.

9) EMOs: Ophthalmoplegia.

10) Orbit: periostitis.

- **What are the ocular manifestations of pregnancy?**

1. Toxaemia of pregnancy: due to eclapsia.
2. chiasmal syndrome: Due to pituitary hyperplasia.
3. Optic neuritis: In hyperemesis gravidarum.
4. retinal haemorrhages and exudates (Vascular retionopathy): in hyperemesis gravidarum.
5. Diabetic retinopathy
6. Pseudotumour cerebri
7. Changes in refration

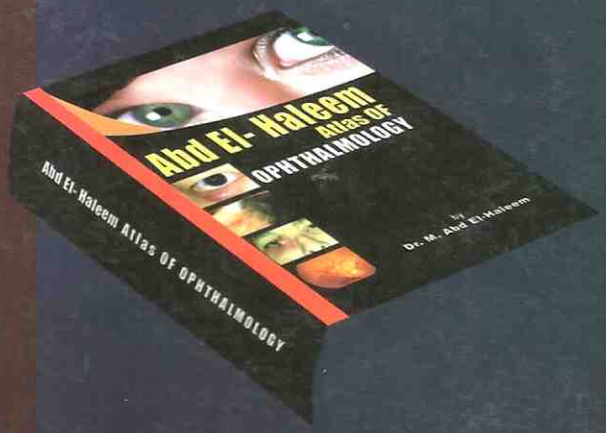
- **What are the ocular manifestations of renal disease?**

1. Edema of lids. 2. Blepharo-conjunctivitis.
3. Renal retinopathy. 4. Uraemic ammaurosis.

- **What is gout and its relation to ocular disease?**

Gout is in born error of metabolism with excess uric acid in blood and attacks of arthritis + ocular inflammations as conjunctivitis, episcleritis, keratitis and iridocylitis.

- Ophthalmologic Summary..... Mohammad Abd El Haleem gives some splendid Topics and examples...His range of reference is impressively wide. He conveys large amounts of detail with a pleasant urgency.
- A comprehensive, up-to-date viewpoint on diverse cases and issues related to ophthalmology.
- Highly recommended for students, faculty and professional doctors.
- Provocative and diverse, this Summary explores the anxious relationships between different similar and dissimilar medical cases.
- Useful friendly format for examinations.



LOTUS VISION