

CHRONIC VISUAL LOSS

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Visual loss

Refractive errors

1.1 myopia

1.2 hyperopia

1.3 astigmatism

Cloudy of ocular media

2.1 acute/subacute

2.2 gradual/chronic

Sensory pathway abnormalities

3.1 acute/subacute

3.2 gradual/chronic

3.3 transient

Functional visual loss

Amblyopia
Malingering
Hysteria

Refractive errors

- Myopia
- Hyperopia
- Astigmatism

Cloudy of ocular media

- Acute/subacute

- AACG
- Corneal ulcer
- Acute anterior and posterior uveitis
- Ocular trauma
- Vitreous hemorrhage
- Infection

- Gradual/chronic

- **Cataract**
- Chronic anterior and posterior uveitis
- Corneal decompensation
- Dry eye

Sensory pathway abnormalities

1. Acute/subacute

- Retinal detachment
- Retinal vv. occlusion
- Retinitis (various causes)
- Optic neuritis
- ARMD

2. Gradual/chronic

- Diabetic retinopathy
- **POAG**
- **ARMD**
- Intoxication ; CQ

3. Transient

- Papilledema
- Migraine
- Amaurosis fugax
- Retina edema

Visual loss

Refractive errors

1.1 myopia

1.2 hyperopia

1.3 astigmatism

Cloudy of ocular media

2.1 acute/subacute

2.2 gradual/chronic

Sensory pathway abnormalities

3.1 acute/subacute

3.2 gradual/chronic

3.3 transient

Functional visual loss

Amblyopia
Malingering
Hysteria

Case I

24-year of age, Thai female

Problem : Difficult to see the far objects, but she has no problem to read the books

HOW DO YOU DO?

Hx. & P.E.

- No eye pain or redness
- No underlying diseases
- No ocular trauma
- VA : OD 20/70
OS 20/100
- VA c glasses
OD 20/40
OS 20/70

VA with correction

- VA c glasses c PH

OD : 20/30

OS : 20/30

What is the diagnosis?

How do you do next?

- Refraction*

	sphere	cylinder	axis	VA
OD	-3.00	+/-0	-	20/20
OS	-4.00	-1.50	90°	20/20

- Eye examination must be careful!!!

Case II

75-year of age, Thai female

Problem : Difficult to see the far and near objects in both eyes for 2 years

HOW DO YOU DO?

- History

- Slow progressive of blur vision
- Painless
- No underlying diseases

- Eye examination

VA c glasses :

OD : 20/200

OS : HM

- VA c glasses c PH

OD : 20/70

OS : NIPH

- **Eye examination**
- **RAPD** : not present
- **A/S** : normal
- **IOP** : 18/15 mmHg
- **Fundus** :
cupping 0.4 and normal
retina OD, abnormal red
reflex OU
- **Lens** : as the figured

- OD



- OS



- What is/are the diagnosis?
- How do you do?*_
- How is the prognosis in this case?

Case III

75-year of age, Thai female

Problem : Difficult to see the far and near objects in both eyes for 2 years

HOW DO YOU DO?

- History

- Slow progressive of blur vision
- Painless
- No underlying diseases

- Eye examination

VA c glasses :

OD : 20/200

OS : CF 2'

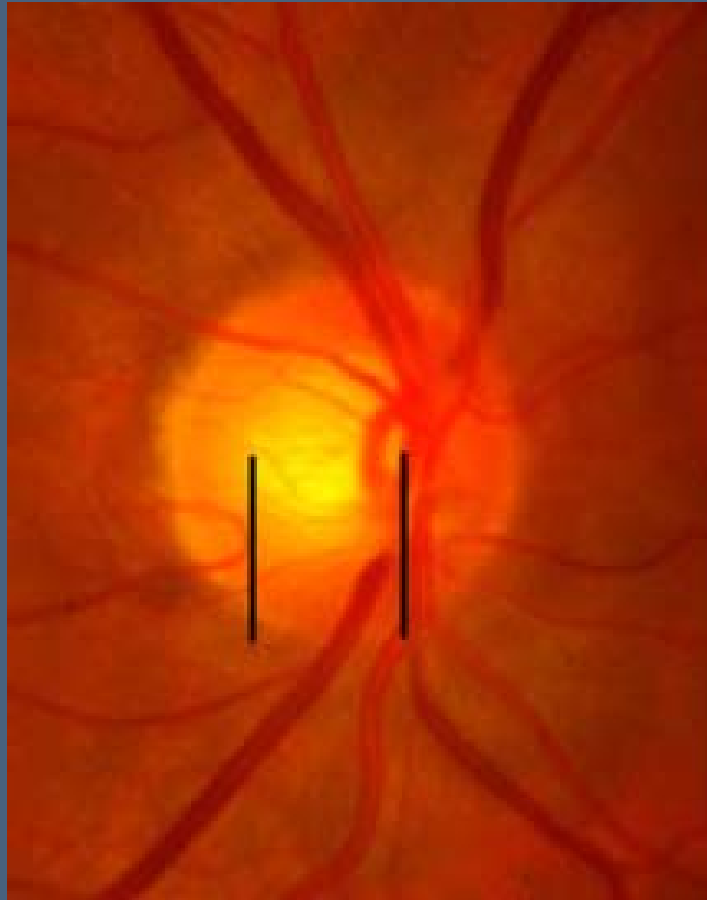
- VA c glasses c PH

OD : 20/70

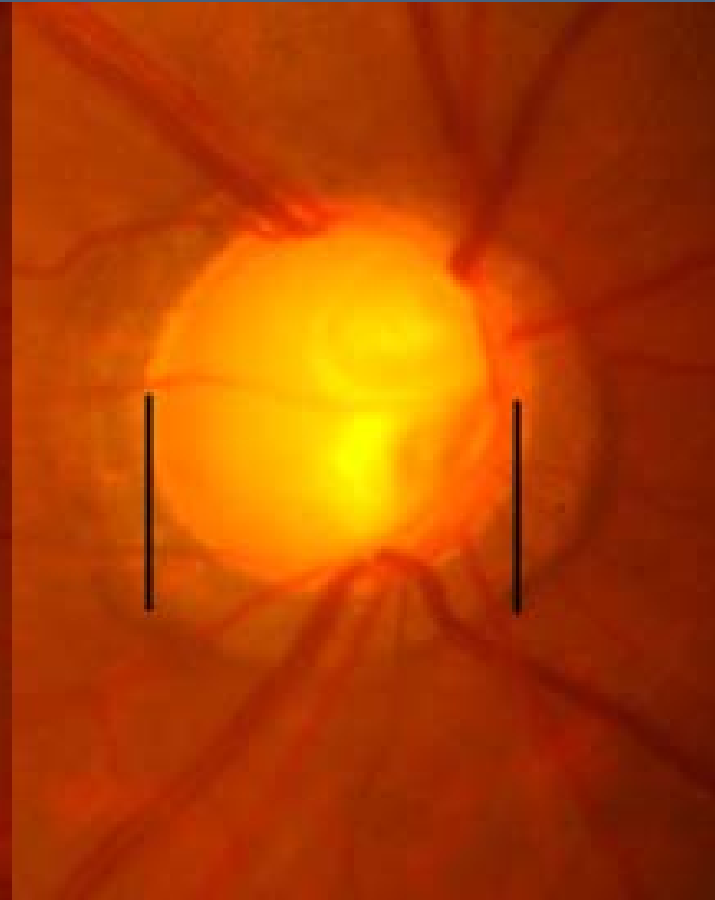
OS : 20/100

- **Eye examination**
- **RAPD** : not present
- **A/S** : normal
- **IOP** : 25/30 mmHg
- **Lens** : NS 2+
- **Fundus** : normal macula
,cupping as the figured

- OD



- OS



- What is/are the diagnosis?
- How do you do? ±

Case IV

80-year of age, Thai male

Problem : Difficult to see the far and near objects in both eyes for 1 year

HOW DO YOU DO?

- History

- Slow progressive, painless blur vision, especially central vision
- Farmer
- Smoking
- No underlying diseases

- Eye examination

VA c glasses :

OD : CF 2'

OS : 20/200

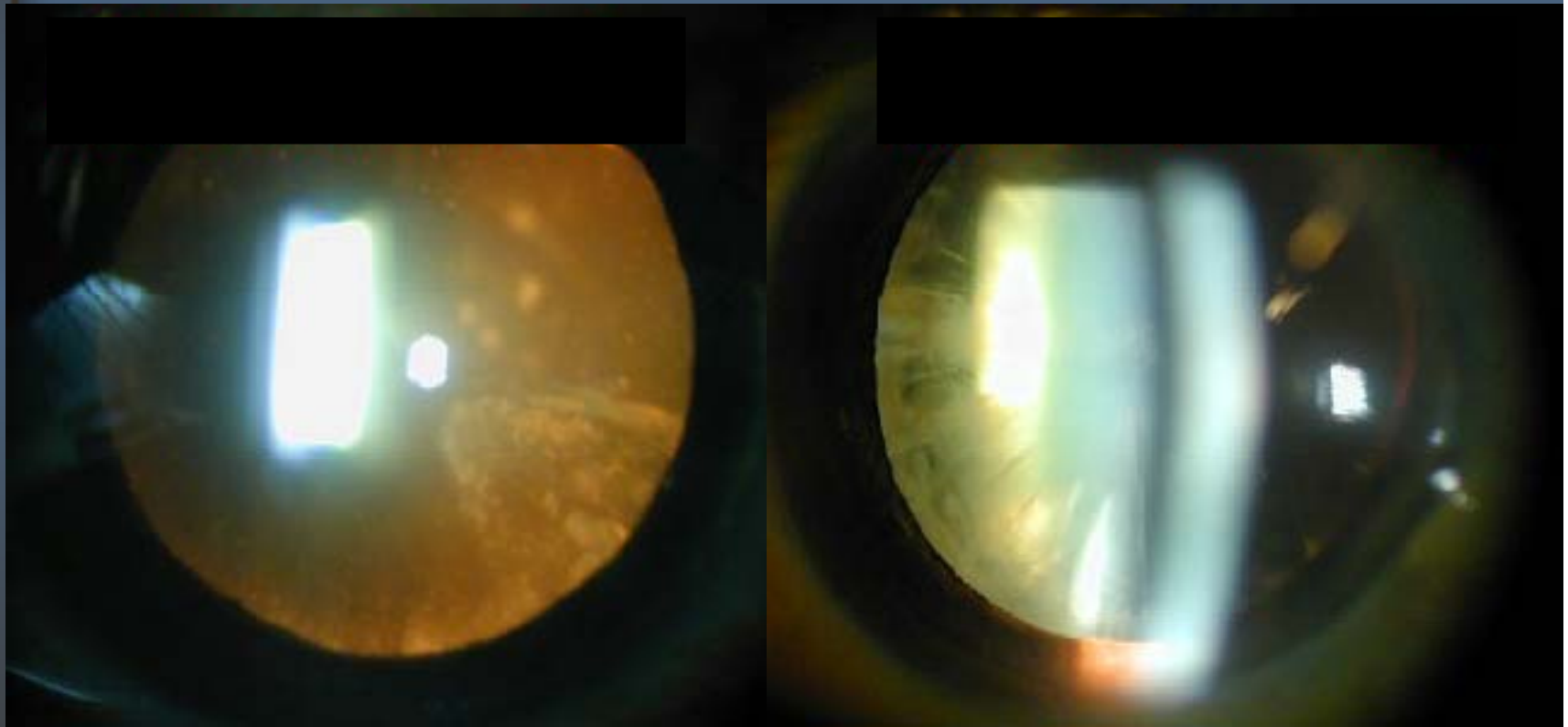
- VA c glasses c PH

OD : NIPH

OS : 20/200

- **Eye examination**
- **RAPD** : not present
- **A/S** : normal
- **IOP** : 22/23 mmHg
- **Lens** : as the figured
- **Fundus** : as the figured

Lenses

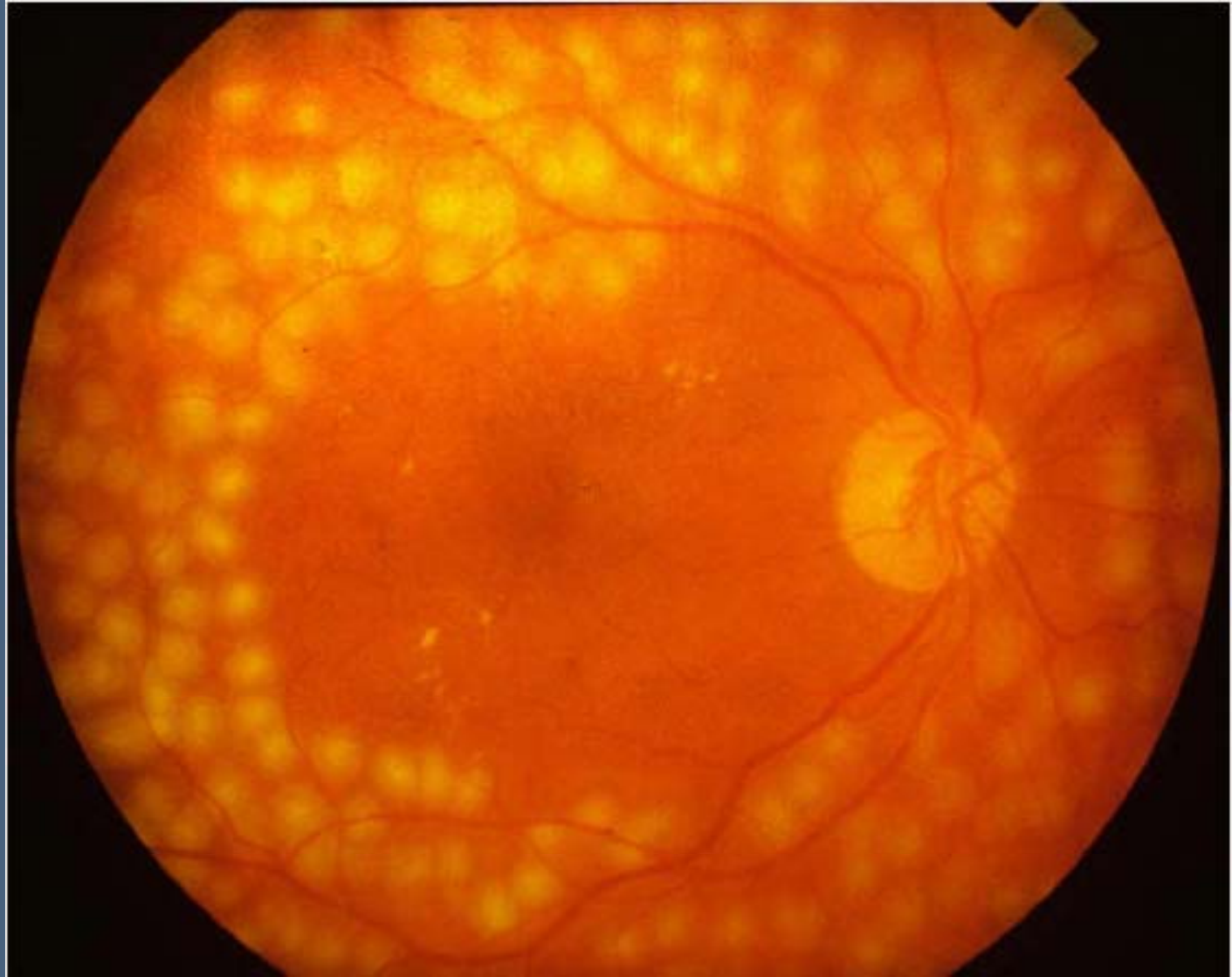


Fundus



- What is/are the diagnosis?*_
- How do you do?

What is this picture?



Objectives

- Characteristics of the optic disc, **determining normal or abnormal optic disc**
- **Recognize a cataract** and to determine its approximate potential effect on the patient's vision, determine whether a cataract is the only cause of a patient's visual decrease
- Examine the **macula** with the ophthalmoscope and recognize the **signs and symptoms of maculopathy**

Glaucoma

Relevance

- Significant cause of irreversible blindness, but the blindness can be prevented
- Most patients are asymptomatic, majority of patients lack of pain, ocular inflammation, or halo
- Peripheral vision can be lost before central vision
- Visual field defects are characterized by specific shaped scotoma and contraction of peripheral field

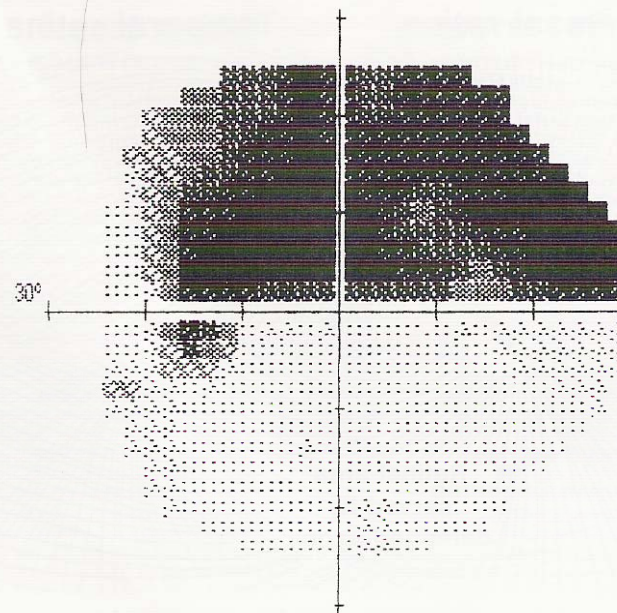
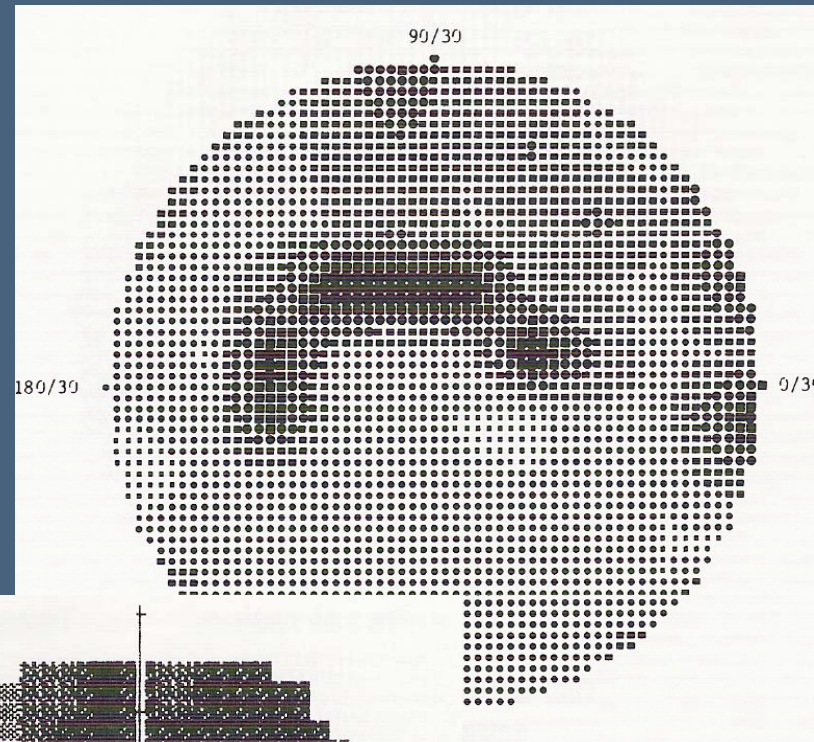
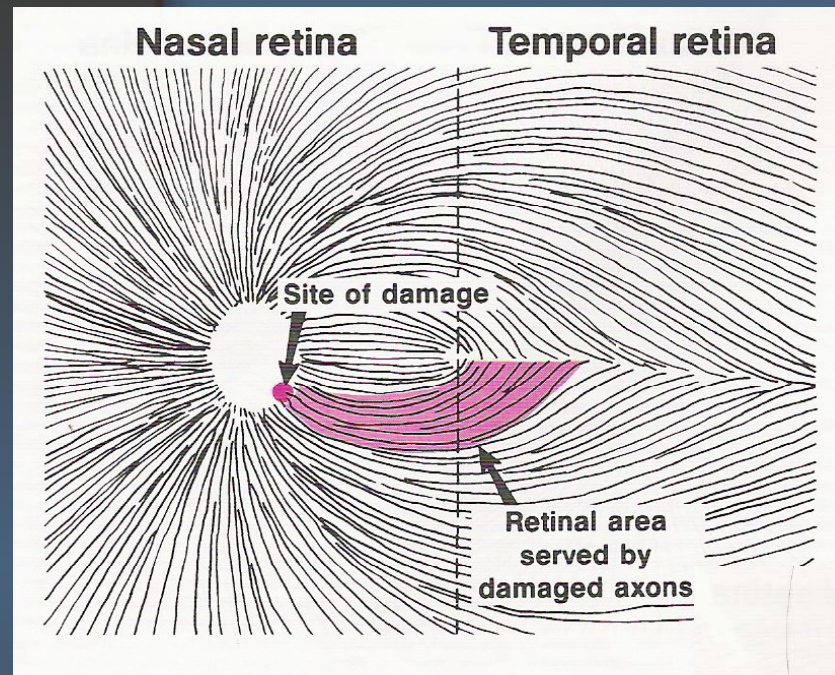


FIG III-21—Altitudinal defect, left eye.



Relevance

- **Early detection** of glaucoma is important
- Usually involves elevation of IOP above the statistically normal range
- Prolonged elevation of IOP can lead to optic nerve damage
- Routine IOP measurement is a valuable means of screening of glaucoma

Relevance

- In some cases, glaucomatous optic nerve changes **in normal IOP**
- Other disorders, such as brain tumor, can also cause changes in optic nerve

Basic information

Factors effect to IOP

1. **Aqueous production** : ciliary body epithelium (Non pigmented epithelium)
2. **Resistance to outflow** : conventional route (TM) , unconventional route (Uveoscleral)
3. **Episcleral venous pressure**

Basic information

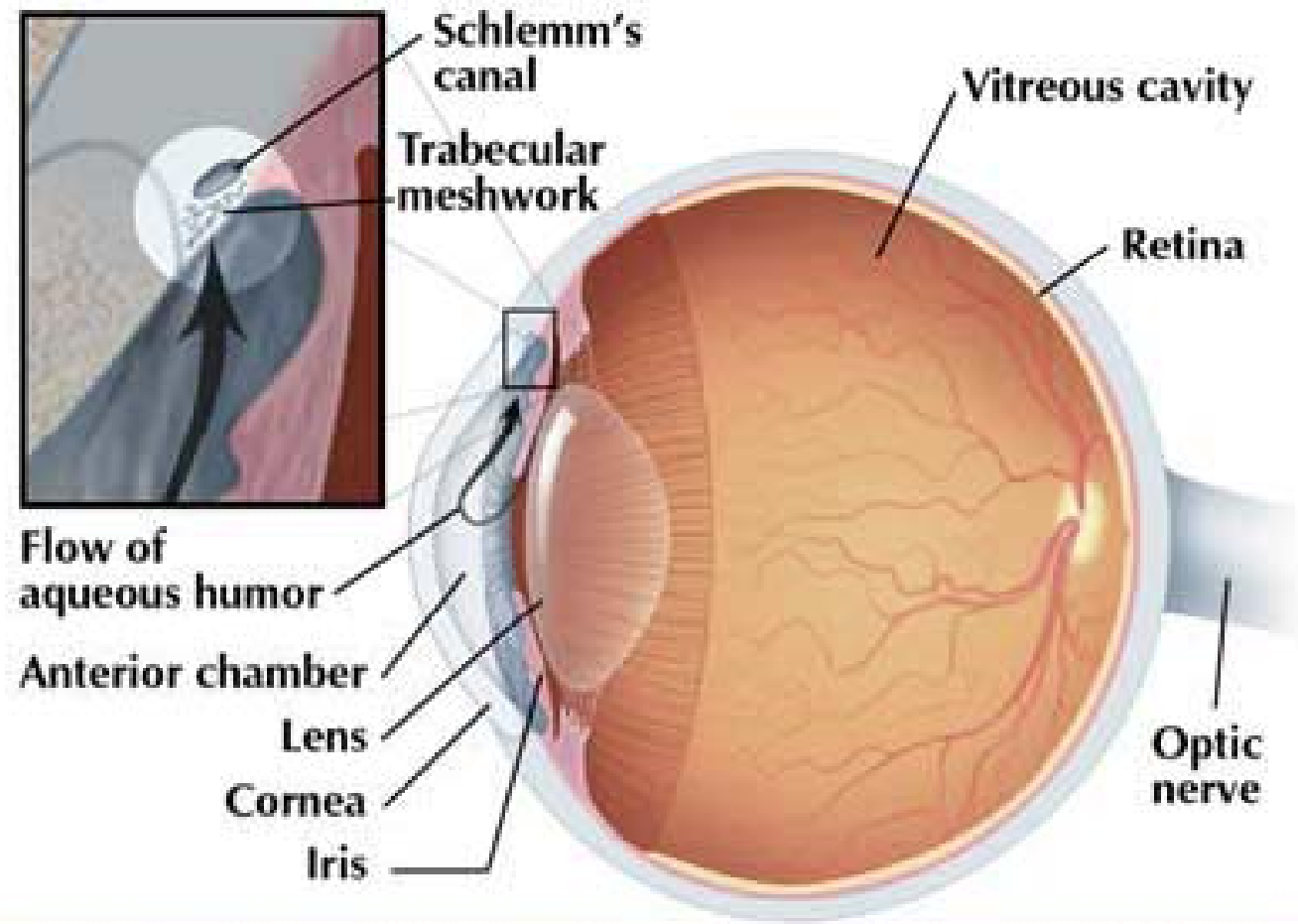
- **Conventional route**

: anterior chamber angle, trabecular meshwork, Schlemm's canal, collector channels, aqueous vein

: >80% of aqueous pass through this route

- **Unconventional route**

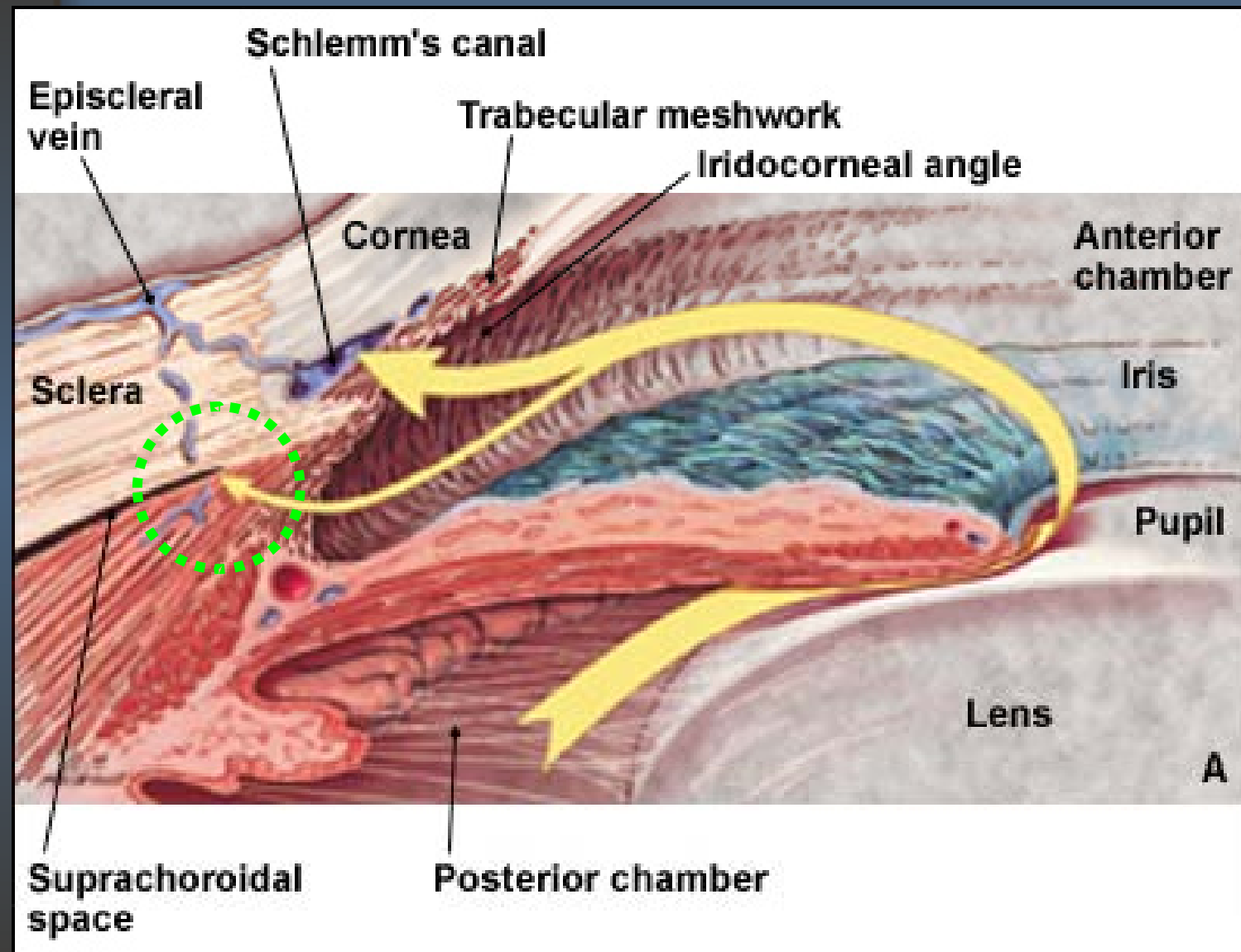
: anterior chamber, intermuscular space of ciliary muscle, suprachoroidal space



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Basic information

- **Intraocular pressure**
 - All eye have an internal pressure
 - IOP is largely dependent on the ease of flow to the trabeculum and Schlemm's canal
 - Greater the resistance to flow, the higher the IOP
 - Most normal eyes have an IOP of ≤ 21 mmHg.

Type of glaucoma

A. Primary glaucoma

1. Open-angle glaucoma : POAG
2. Angle-closure glaucoma : ACG
 - 2.1 acute ACG : ocular emergency
 - 2.2 subacute ACG
 - 2.3 chronic ACG : CACG

B. Congenital glaucoma

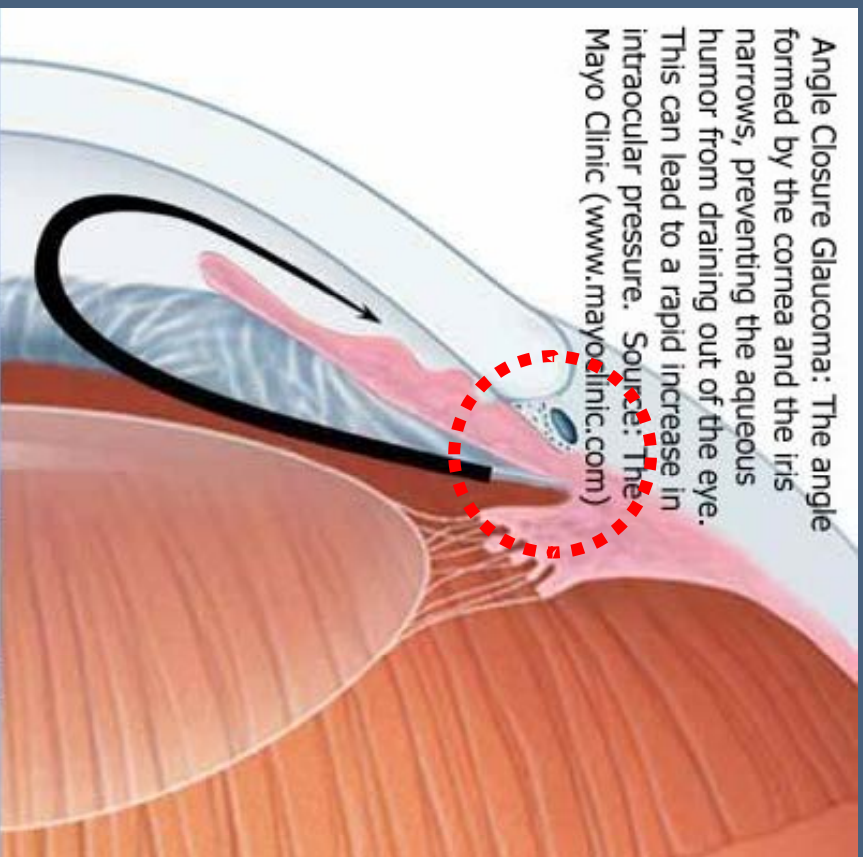
1. Primary congenital glaucoma
2. Glaucoma associated with congenital anomalies

Type of glaucoma

C. Secondary glaucoma (OAG, ACG or both)

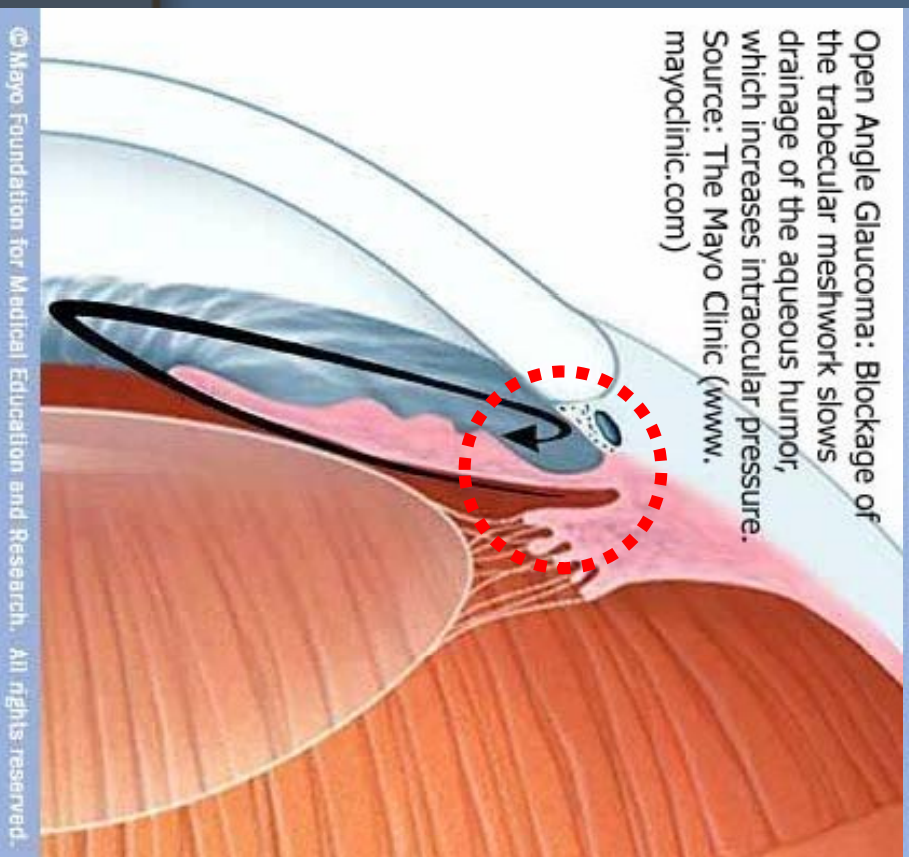
- pigmentary glaucoma
- pseudoexfoliation glaucoma
- due to change of the lens
- due to change of the uveal tract
- due to trauma
- neovascular glaucoma
- following surgical procedure
- steroid induced glaucoma
- etc.

- Angle-closure +



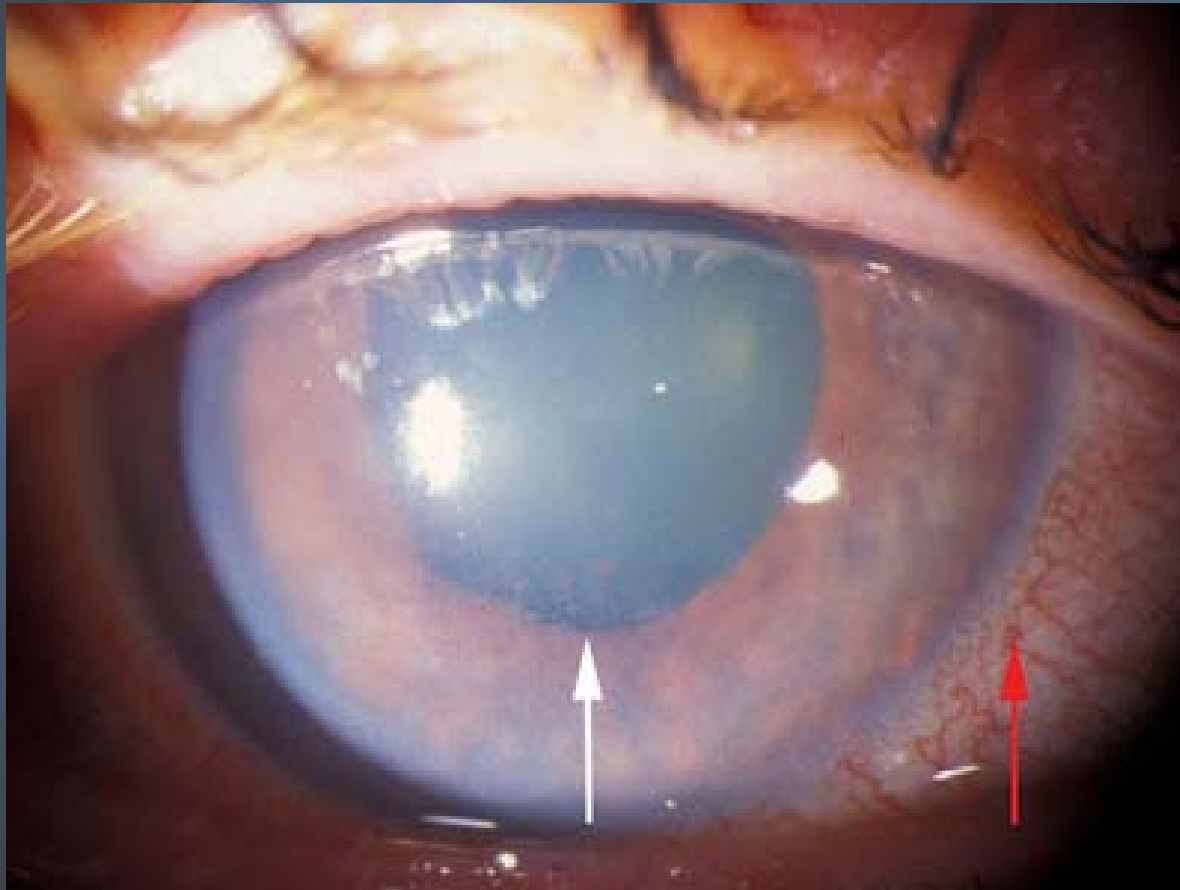
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- Open angle



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Acute angle closure glaucoma



Basic information

- **Optic nerve**
 - Composed of more than 1.2 million nerve fibers
 - Nerve fibers originate in the ganglion cells of the retina
 - At the point of origin, the nerve is called the optic disc, small depression in it called the cup of the optic disc

Optic nerve

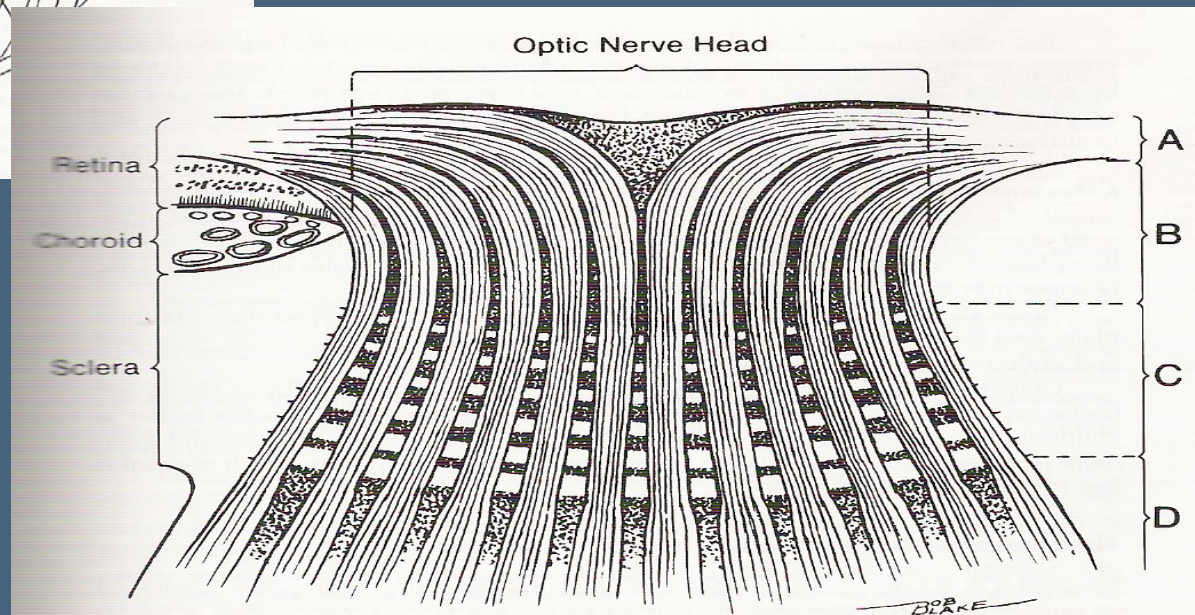
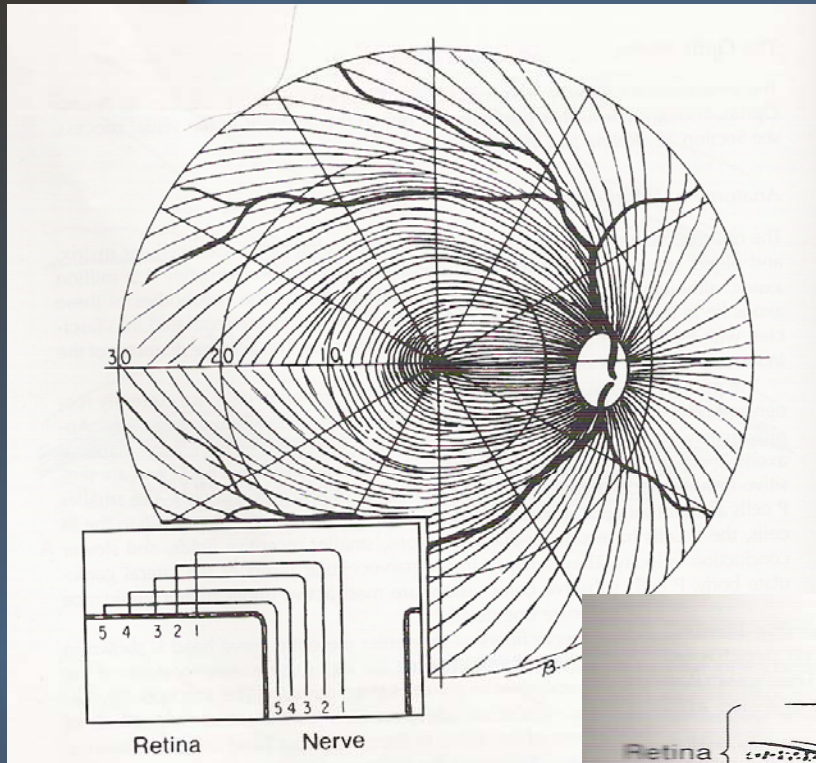
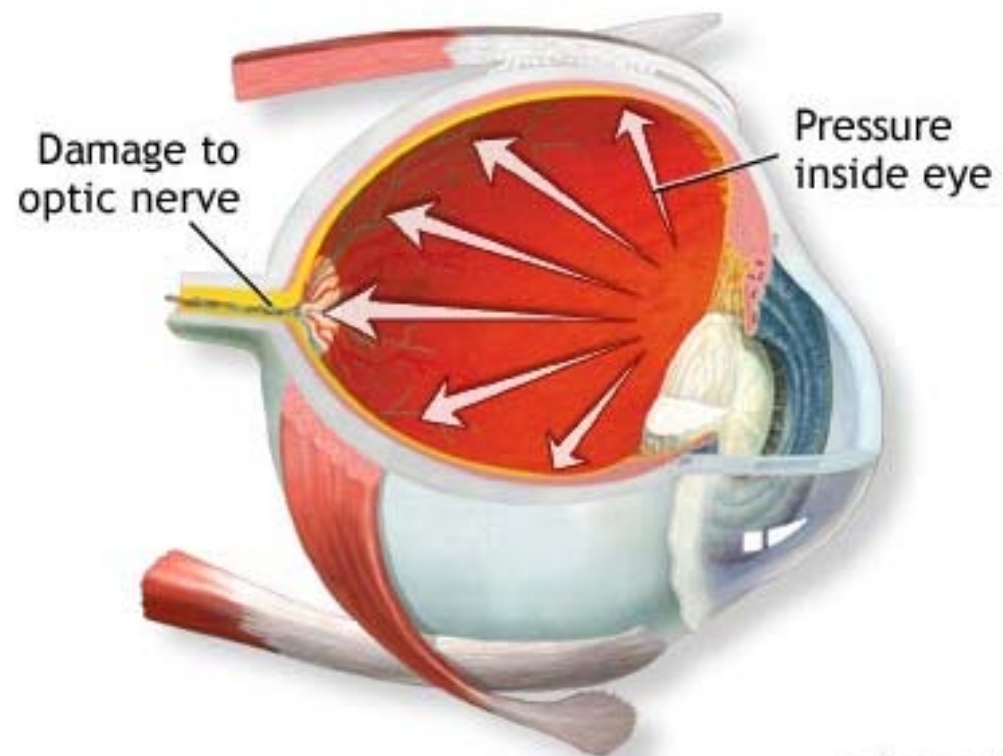


FIG III-10—Divisions of the optic nerve head. A, Surface nerve fiber layer. B, Prelaminar region. C, Lamina cribrosa region. D, Retrolaminar region. (Reproduced with permission from Shields MB. *Textbook of Glaucoma*. 3rd ed. Baltimore: Williams & Wilkins; 1992.)

Basic information

- **Relationship of IOP and Optic Nerve**
 - IOP exerted on walls of the eye, including the optic nerve and its blood vessels
 - Damage to the optic nerve results in visual field loss
 - Detection of glaucomatous visual loss is accomplished by visual field testing
 - **VA usually does not suffer initially**



When to Examine

Ophthalmoscopy

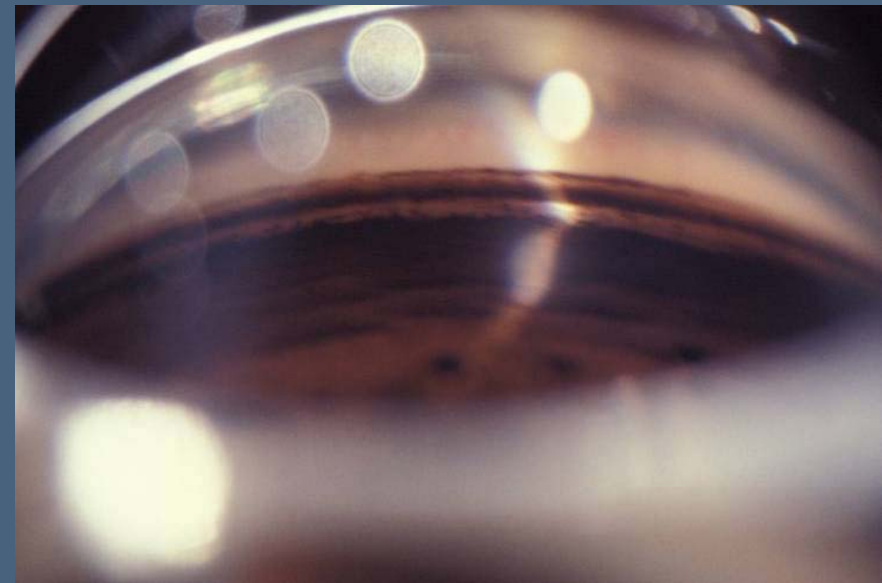
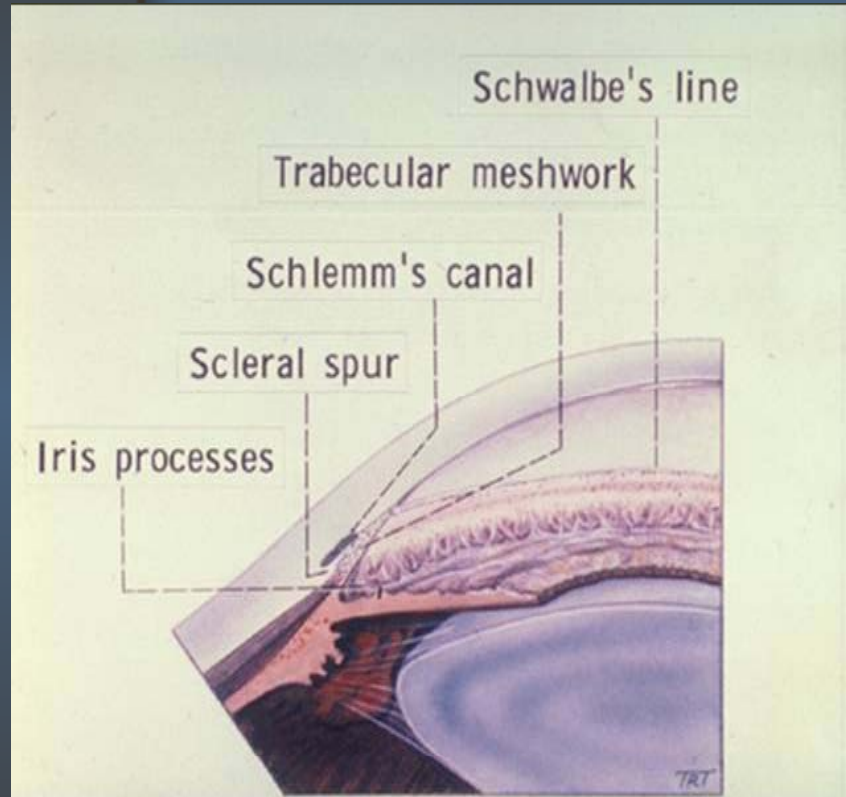
- AAO recommends a glaucoma screening **every 2 to 4 years past age 40**
- Incidence of the disease **increases with age, family history and race**
- African-Americans have an greater risk for development of glaucoma

How to Examine

- Palpation
- Tonometry
 - Indentation
 - : **Schiøtz**
 - Applanation
- Gonioscopy
- Perimetry
 - Goldmann
 - Automated



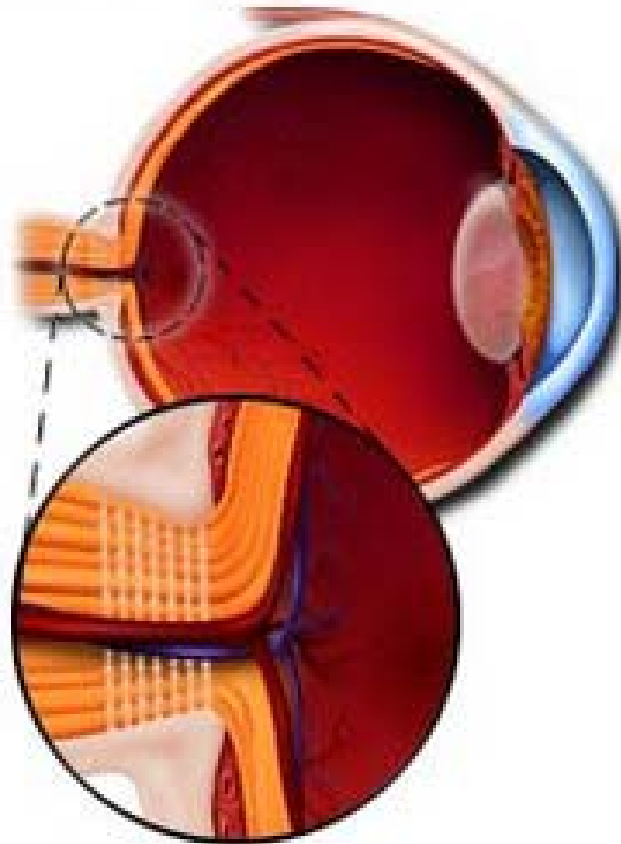
Anterior chamber angle



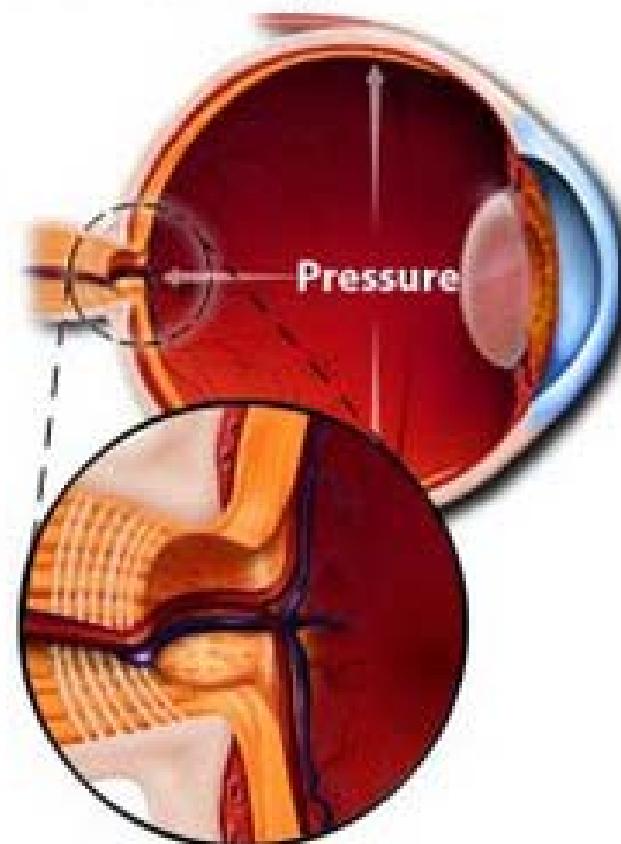
How to interpret the findings

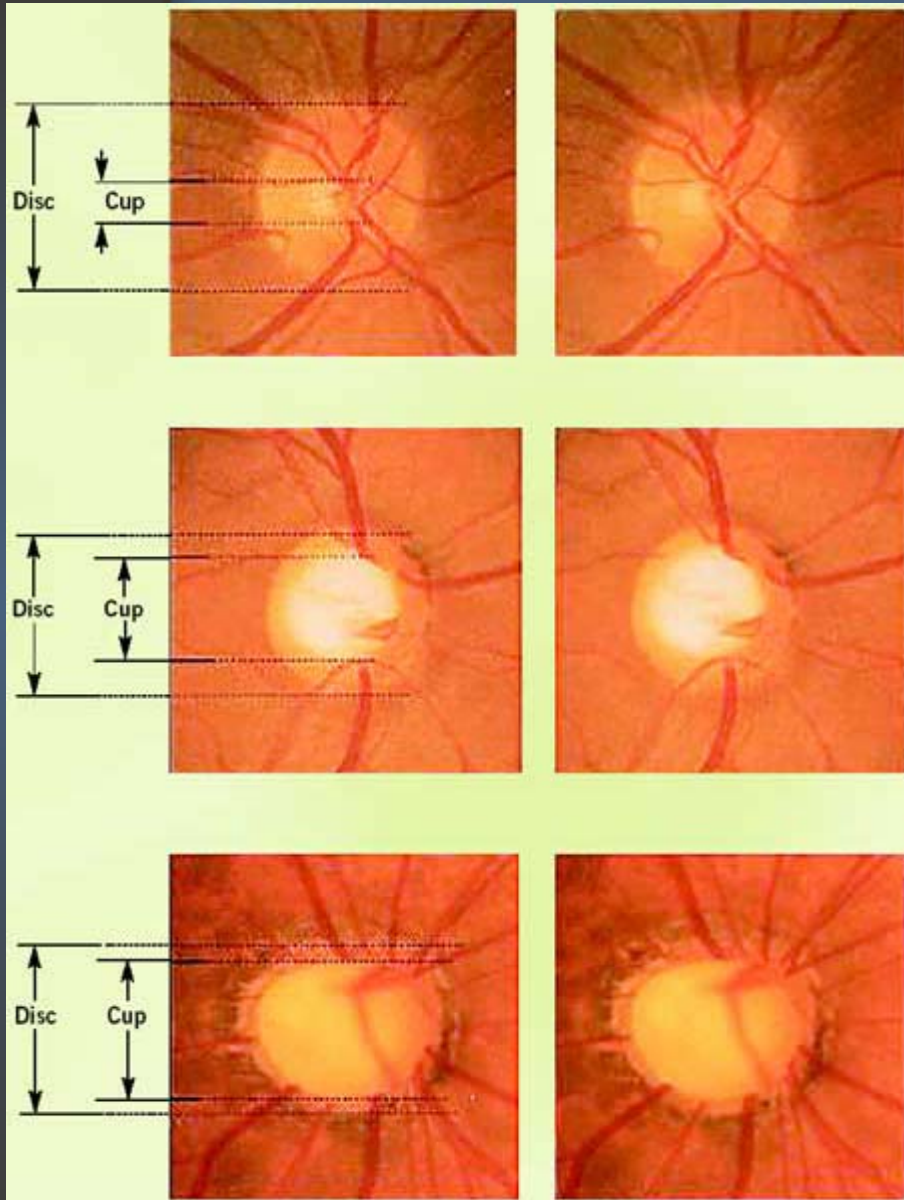
- **Appearance of the optic disc**
 - : Color
 - : Size of cup
 - : Vessels
- **The glaucomatous cupping**
 - : Increase in the size of the optic cup
(cup:disc ratio > 0.5 – raises suspicion of glaucoma)
 - : Vessel displacement
 - : Asymmetrical cupping (difference > 0.2)

Normal eye

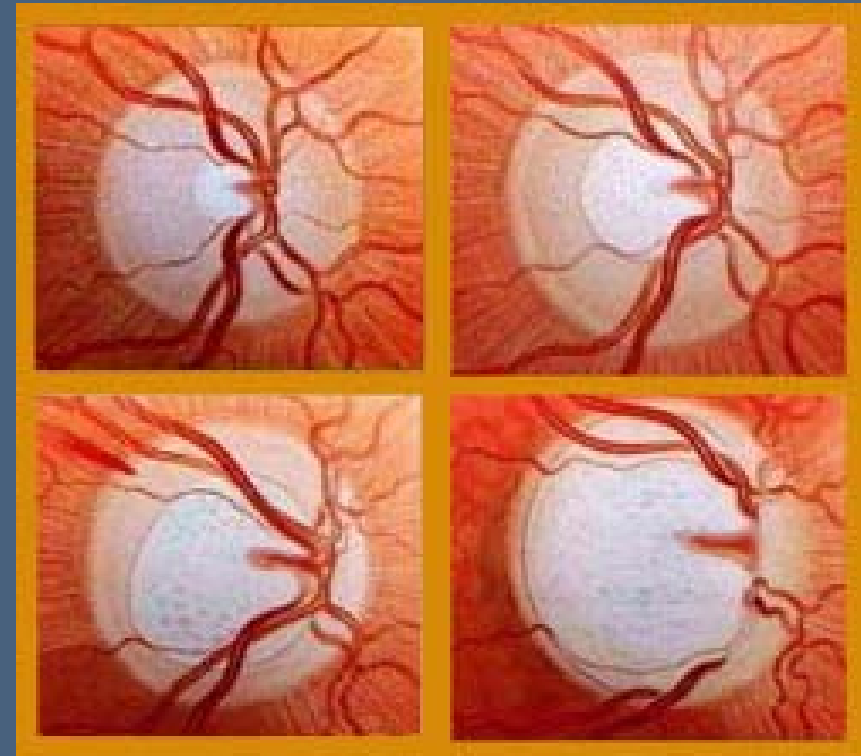


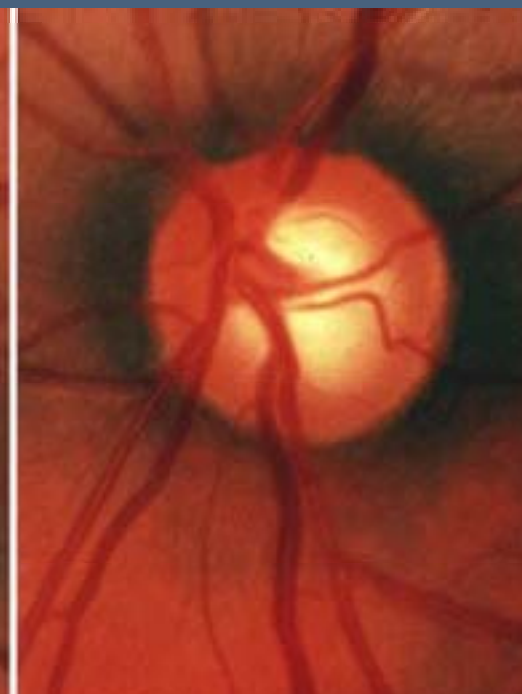
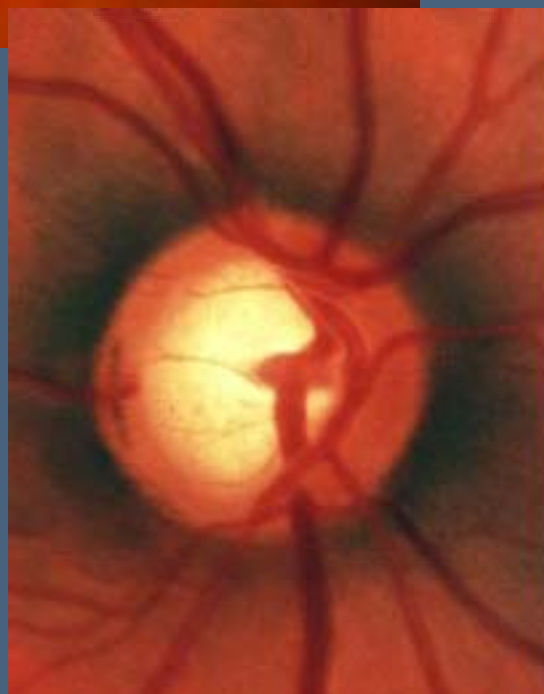
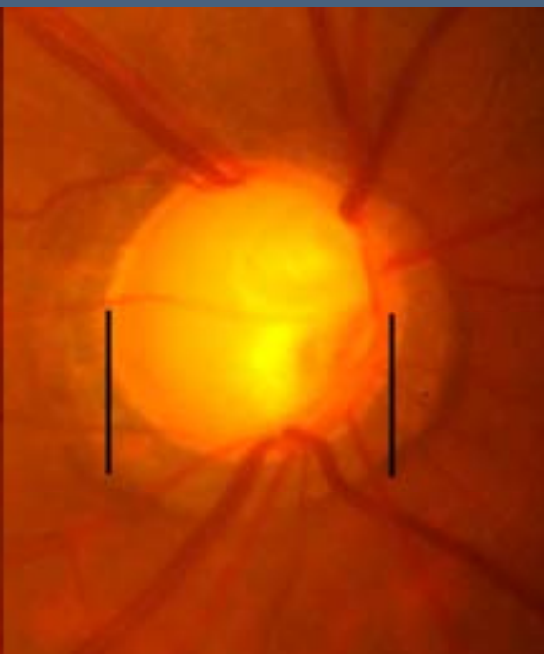
Glaucoma eye





+





Primary open angle glaucoma (POAG)

- “Rule of ten”

For every 1,000 persons age over 40 years.

- 100 are suspected of POAG by visual field, disc appearance, IOP findings or dense risk factors.
- 10 have POAG.
- 1 will be blind as a result of POAG.

IOP is the greatest risk of POAG

- Other risk factors

1. Old age

2. Family history of POAG

3. African heritage

4. Myopia

All of these risk factors are increase risk for presence and progression of POAG.

- Associated conditions : DM, thyroid, CVS dz.

Clinical characteristics of POAG

- Slow progression
- Most asymptomatic
- Usually bilateral, but may be asymmetry of severity
- Normal anterior chamber angle
- Not found other causes of glaucoma

Management or Referral

- ≥ 1 of the following conditions should be referred to an ophthalmologist :
 - IOP > 21 mmHg
 - IOP not elevated, but a difference ≥ 5 mmHg between the eyes
 - An optic cup diameter one half or more of the disc diameter
 - One cup significantly larger in one eye than in the other eye
 - Symptoms of acute glaucoma

Glaucoma Treatment

Goal : preserve normal loss of retinal ganglion cells with minimal complications

- Education
- Treatment options
 1. Medication
 2. Laser
 3. Surgery
 4. Cyclodestructive procedure

Anti-glaucoma drugs

1. β -blocker agents : timolol
2. Non-selective α -adrenergic agonists : dipivefrin
3. Selective α_2 -adrenergic agonists : brimonidine
4. Cholinergic drugs (miotics) : pilocarpine
5. Carbonic anhydrase inhibitors : acetazolamide
6. Prostaglandin derivatives : latanoprost, travoprost, bimatoprost
7. Hyperosmotic agents : glycerine, manitol

Anti-glaucoma drugs

- Mechanisms

1. Reduced aqueous production
2. Enhanced outflow : conventional
: unconventional
3. Combined 1.+2.
4. Decrease vitreous volume
5. Neuroprotective

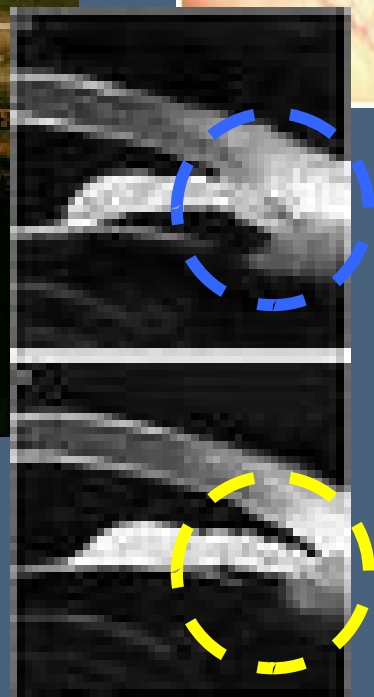
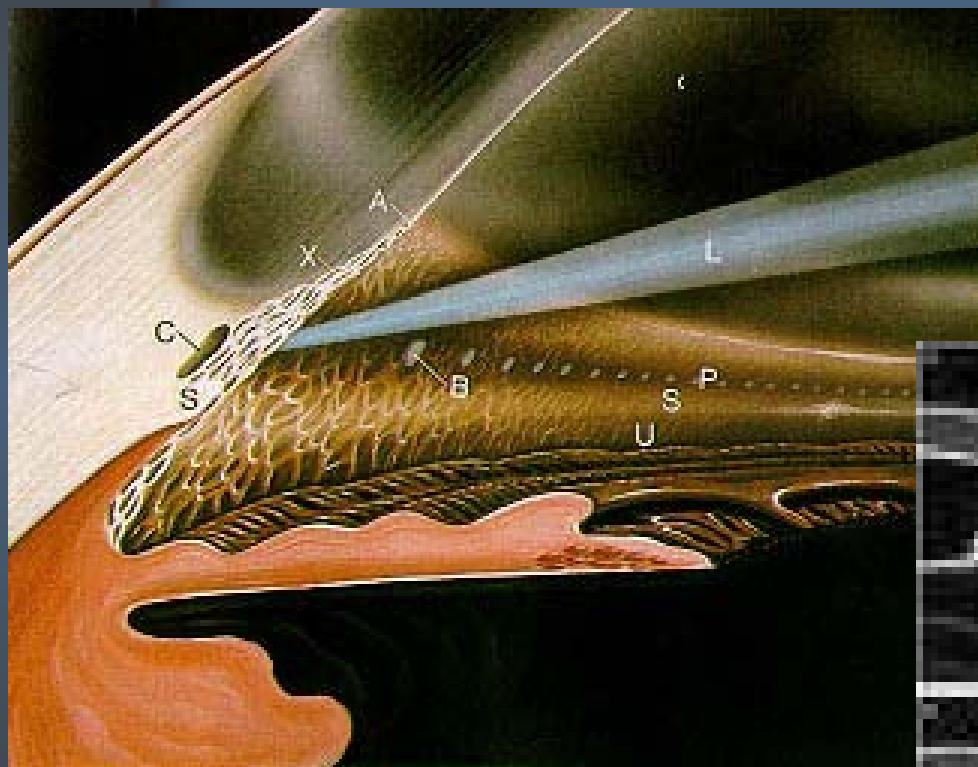
Anti-glaucoma drugs

Attention!!!

- Patient education
- Side effects
- Compliance
- Underlying disease : COPD, asthma, CVS dz., renal disease
- History or drug allergy : esp. **sulfa**

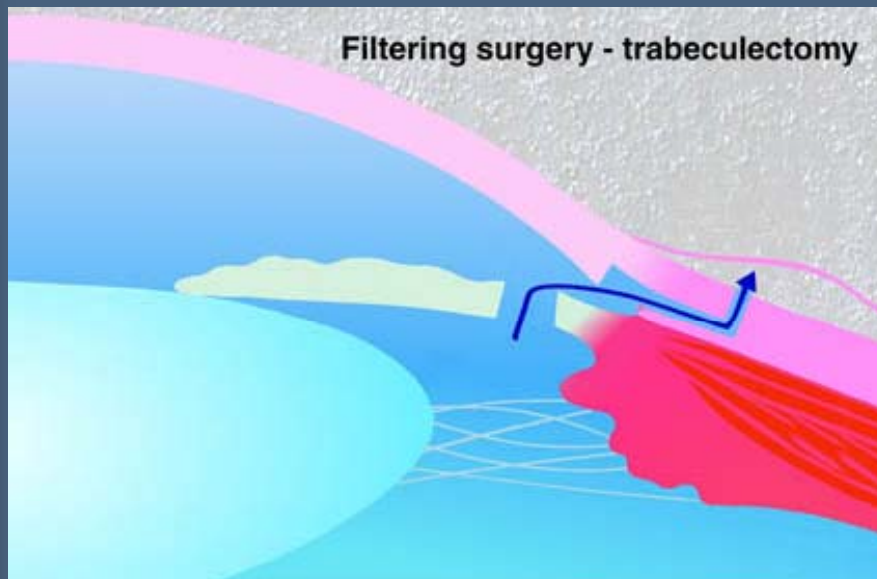
Laser treatment

- Argon laser trabeculoplasty (ALT)
- Selective laser trabeculoplasty (SLT)
- Laser peripheral iridotomy
- Laser iridoplasty

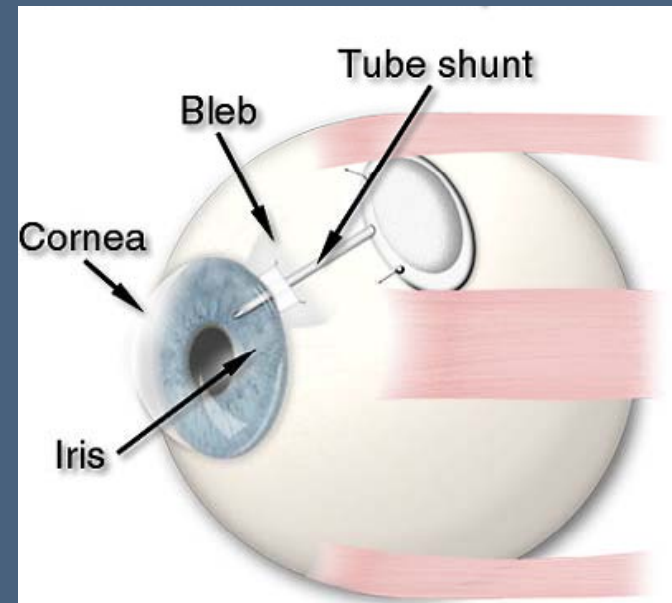


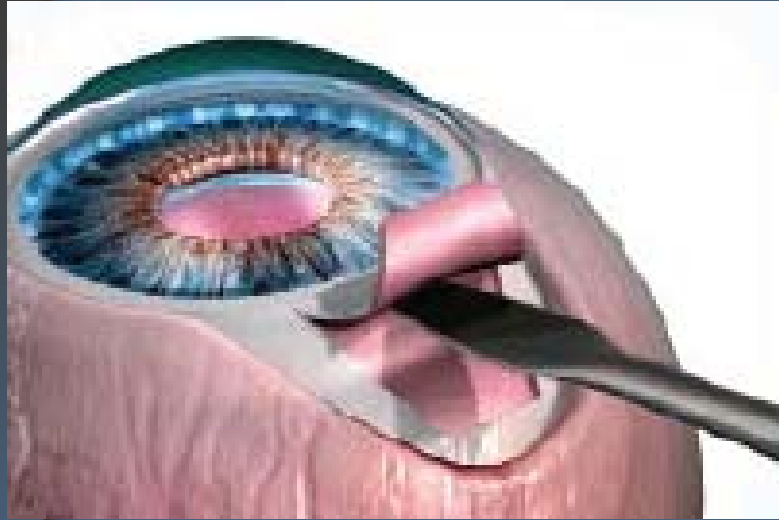
Surgery

- Filtering surgery
:Trabeculectomy +/-
mitomycin C or 5-FU

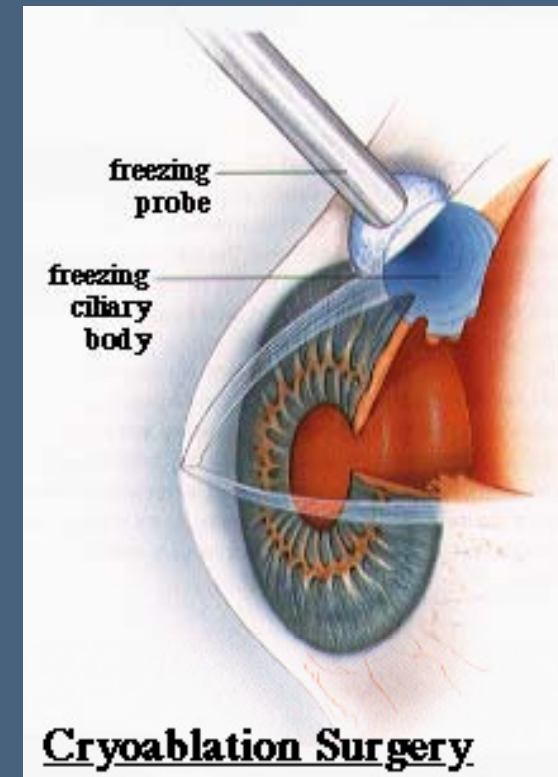
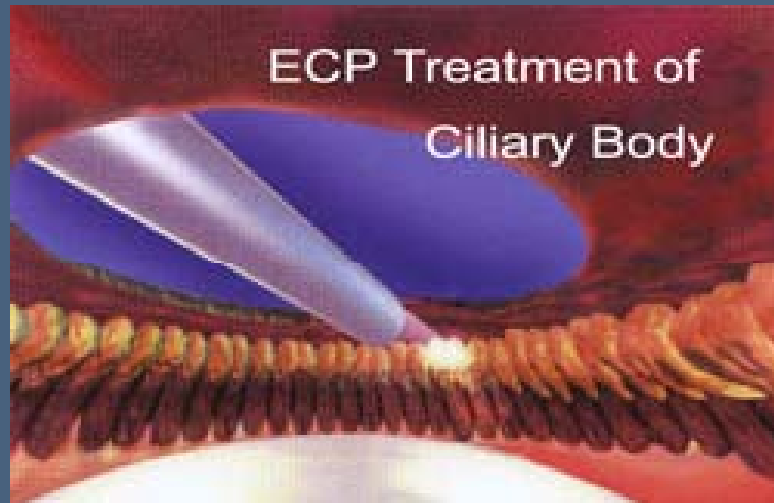


- Glaucoma drainage devices

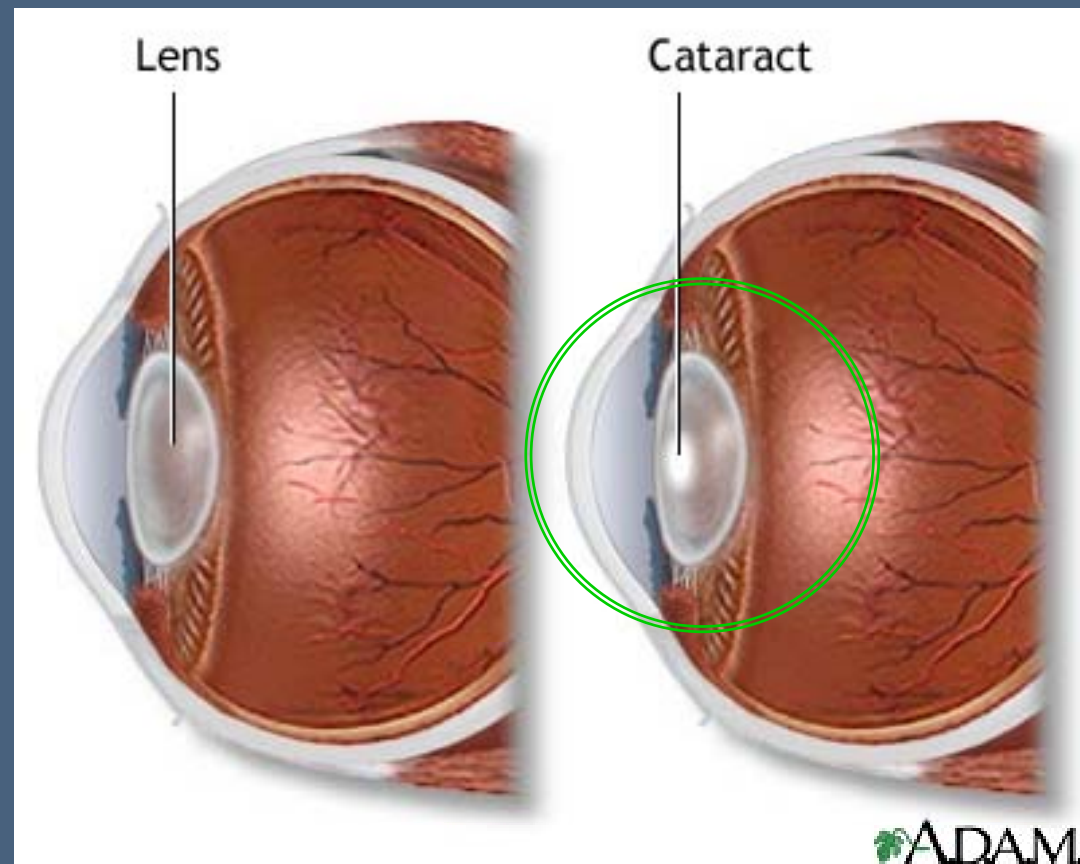




Cyclodestructive procedures_±



Cataract



Relevance

- Congenital, **genetic anomaly**, **various diseases**, or with increasing age (**most common cause**)
- Age-related cataract occurs in about 50% of people between ages 65 and 74
- One of the most successfully treated conditions in all of surgery
- Usually with intraocular lens implantation
- If an implant is not used, visual rehabilitation is still possible with a contact lens or aphakic glasses

Basic Information

- **Lens**

Function :

- refraction : refractive power **+20 D**
- accommodation
- protective function : U.V. , physical barrier

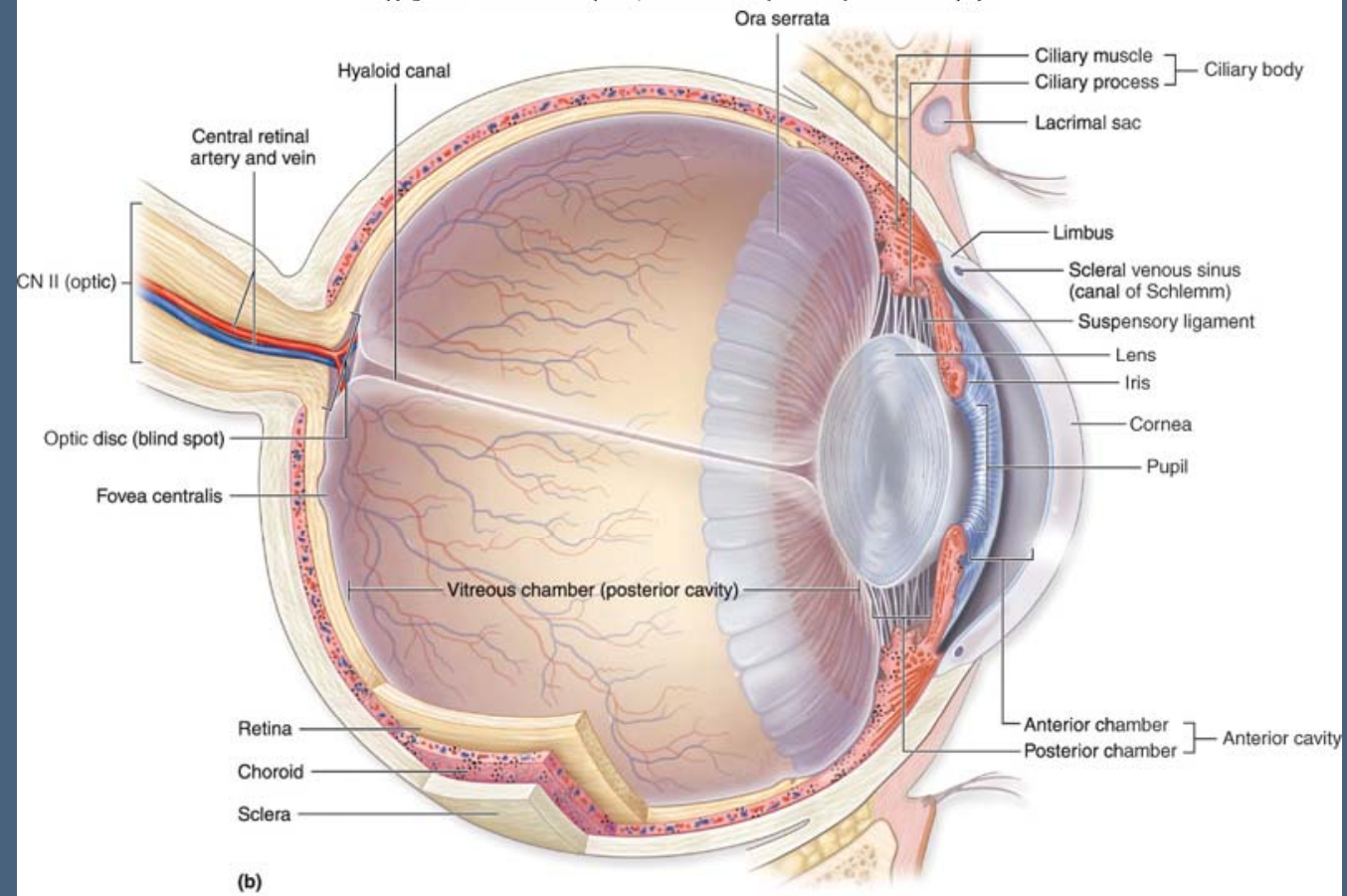
Anatomy :

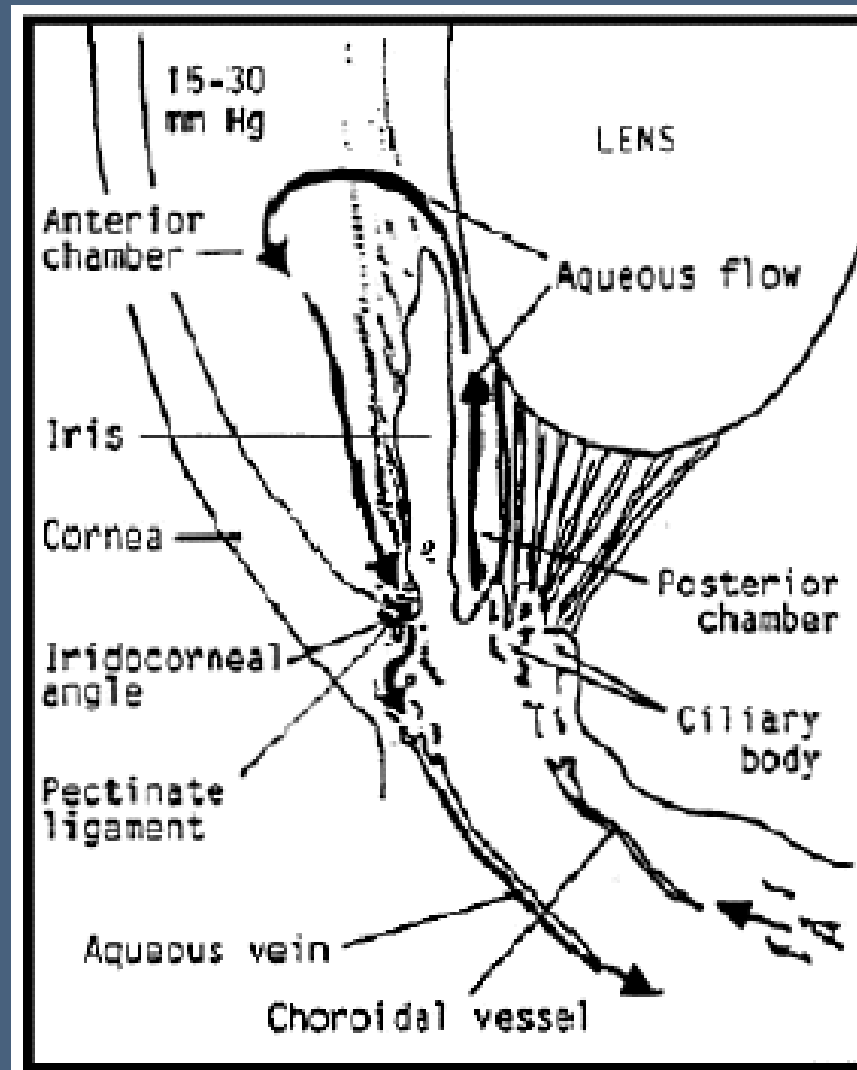
- transparent, biconvex shape
- thickness ~4 mm. , width ~ 9 mm.
- capsule, cortex, nucleus

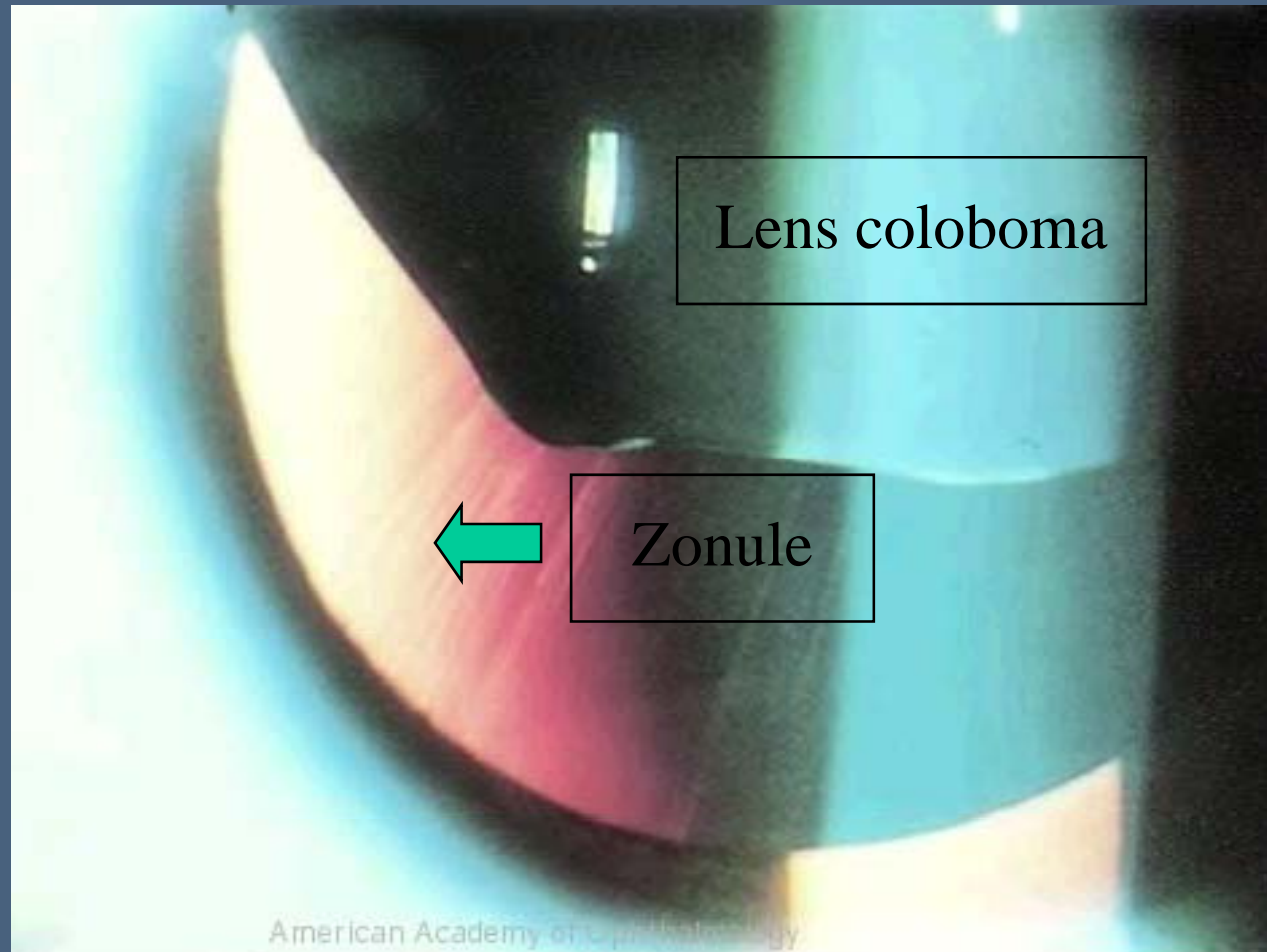
Basic Information

- **Lens**
 - Suspended by thin filamentous zonules (transparent collagen fibers) from the ciliary body
 - Contraction of the ciliary muscle permits focusing of the lens
 - The lens is enclosed in a capsule (elastic semipermeable basement membrane)

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Basic Information

- **Lens**

- : The capsule encloses the cortex and the nucleus of the lens as well as a single anterior layer of cuboidal epithelium

- : **No innervation or blood supply**

- : Nourishment comes from the aqueous fluid and the vitreous

Basic Information

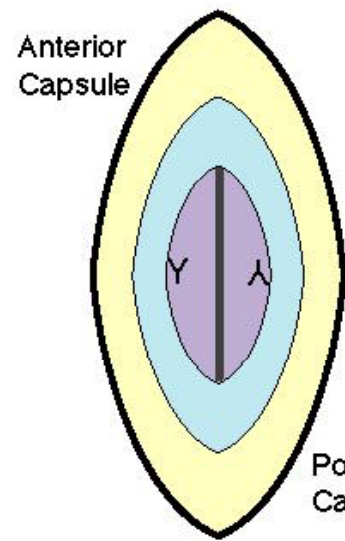
- Lens

- : Continues to grow throughout life

- : Epithelial cells continue to produce new cortical lens fibers

- : Consists of 35% protein, ~ 60% water by mass

- : Percentage of insoluble protein increases as the lens ages and as a cataract develops



Embryonic Nucleus

The dark central vertical band

Fetal Nucleus

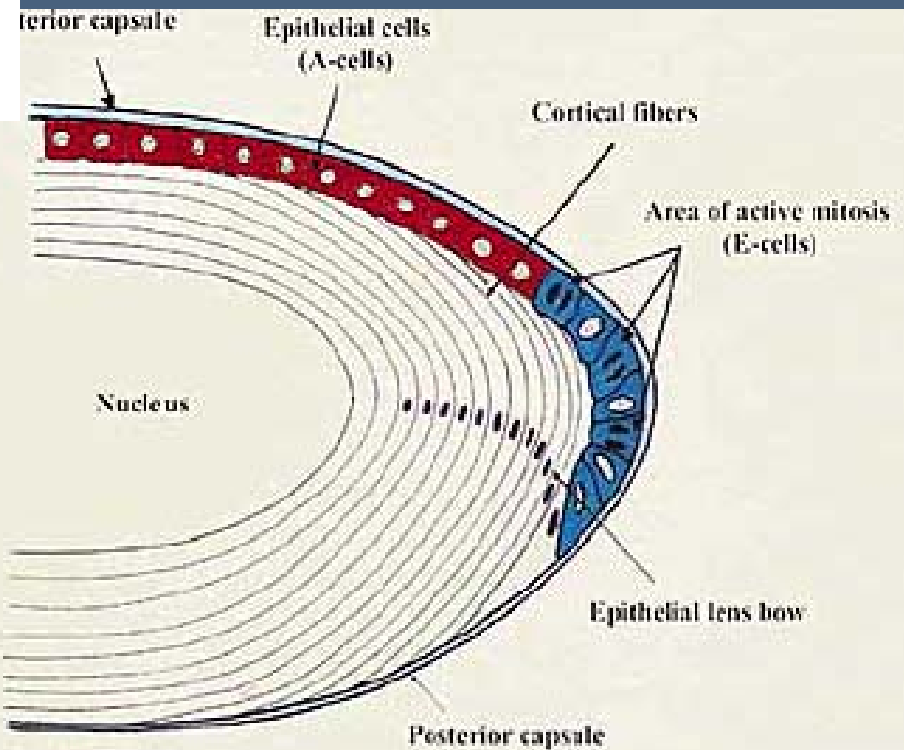
The fetal nucleus is the site of the Y-sutures

Adult Nucleus

The adult nucleus first appears at approximately 35-40 years of age and becomes more defined with increasing age.

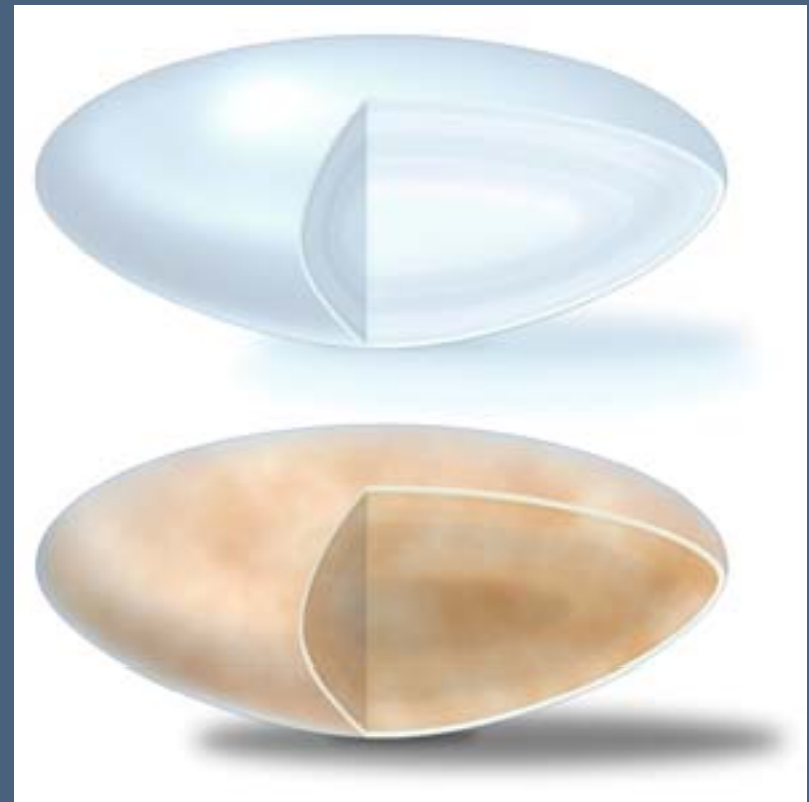
Cortex

The cortex consists of an anterior and posterior portion. It increases in size with age, and is the site of the first signs of some age-related cataracts.



Basic information

- **Cataract**
 - Any opacity or discoloration of the lens, whether a small, local opacity or the complete loss of transparency
 - Clinically : opacities that affect visual acuity



Basic information

- Cataract

- Opacification of the nucleus and cortex, there may be a yellow or amber color change to the lens
- May develop very slowly over the years or may progress rapidly, depending on the cause and type of cataract

Classification

- **Primary cataract**
 - congenital
 - juvenile
 - presenile
 - senile
- **Secondary cataract**
 - extraocular disorder
 - intraocular disorder

Primary cataract

- Congenital cataract

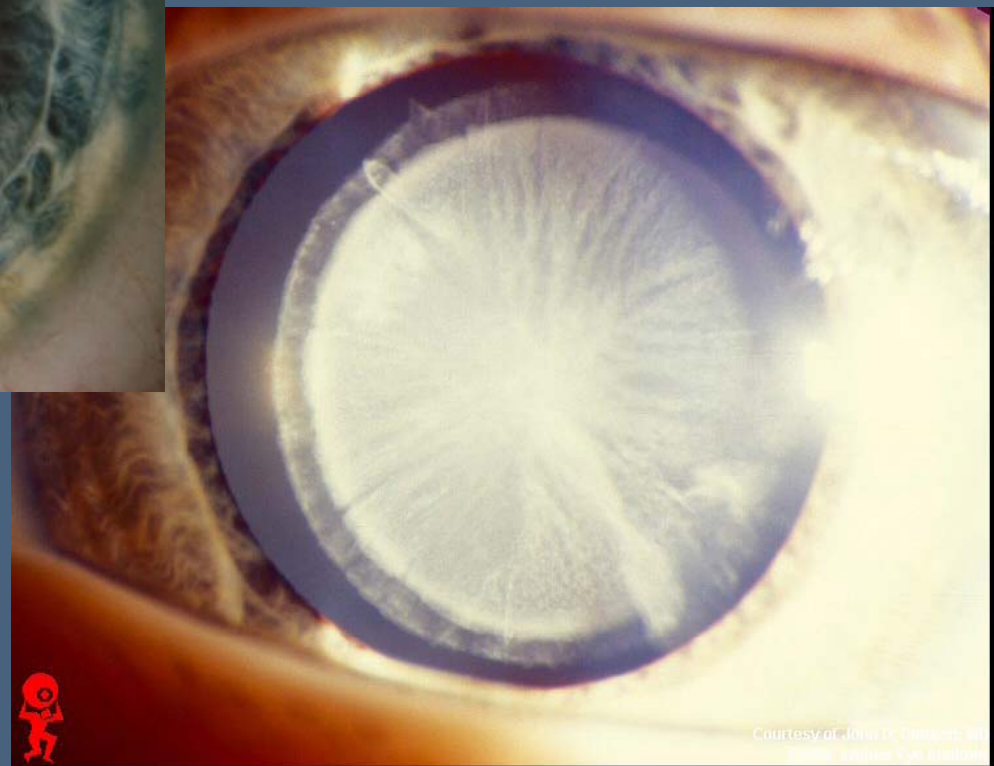
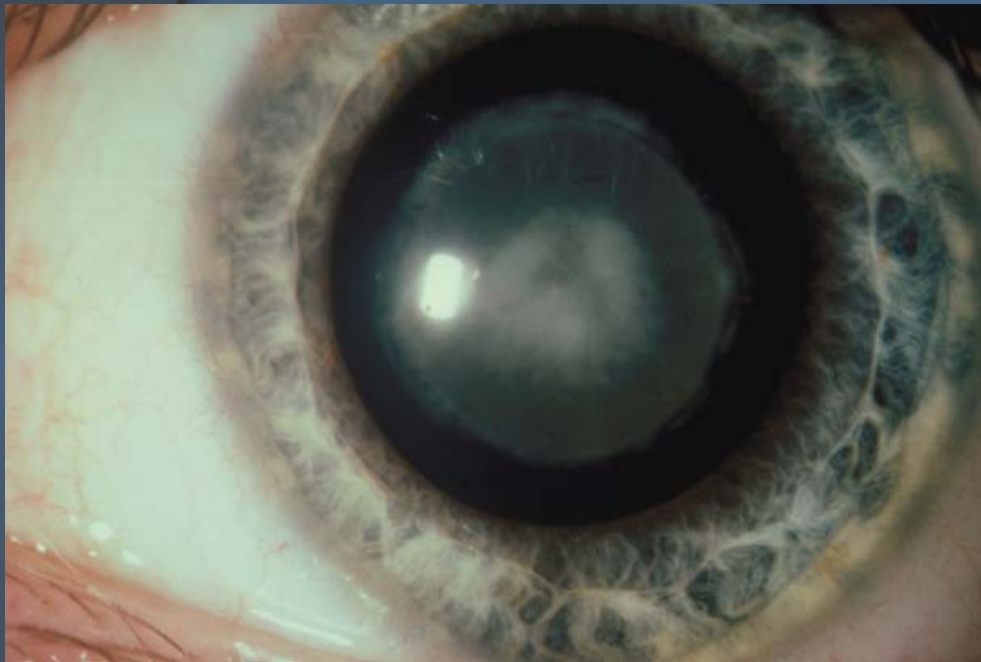
- : <3 mos.

- : usually unknown cause

- : may from rubella, steroid, maternal DM, radiation

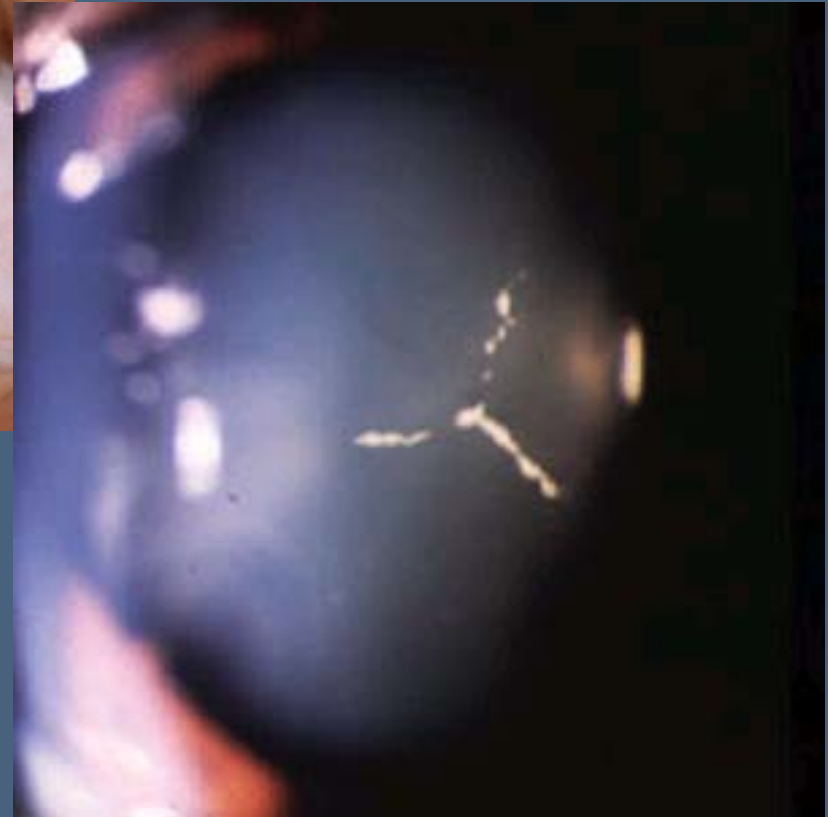
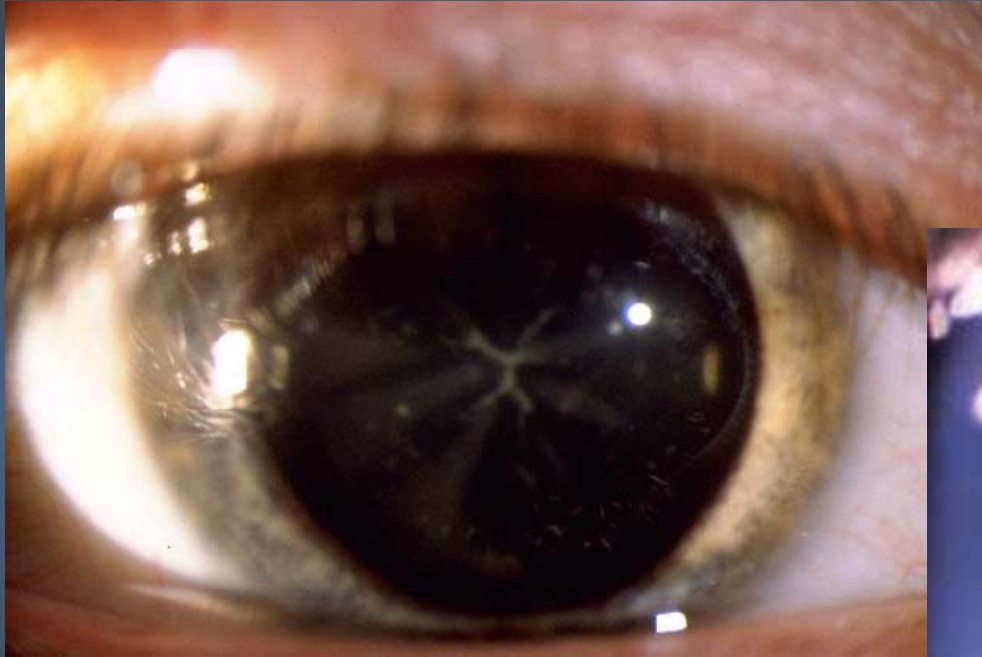
- : specific pattern of cataract – polar, suture, lamella cataract

Lamella cataract

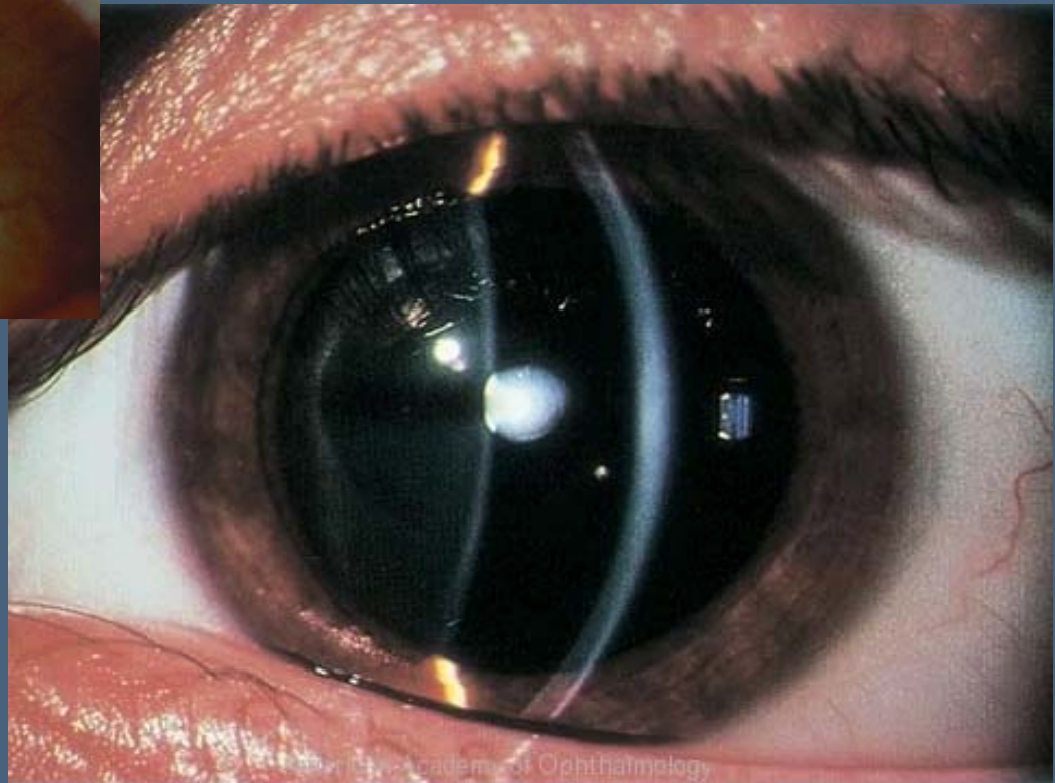
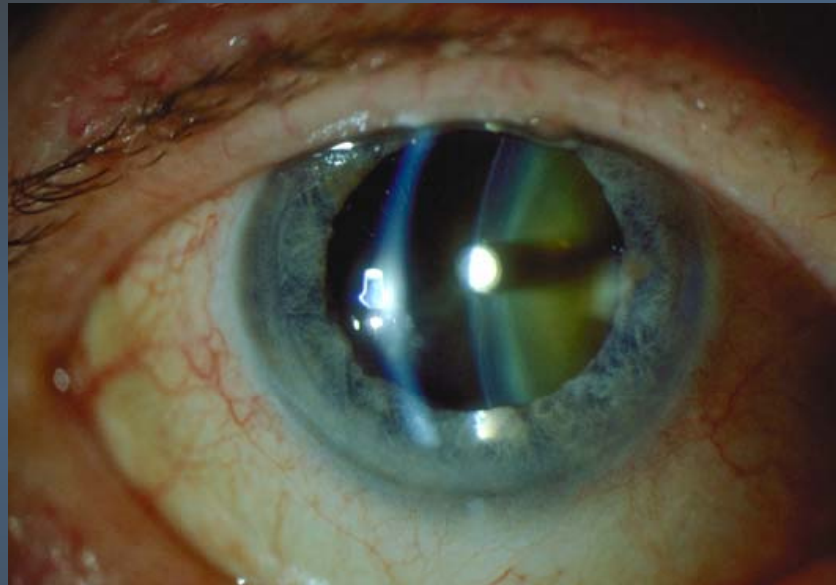


Courtesy of John D. Connor, MD
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Sutural cataract

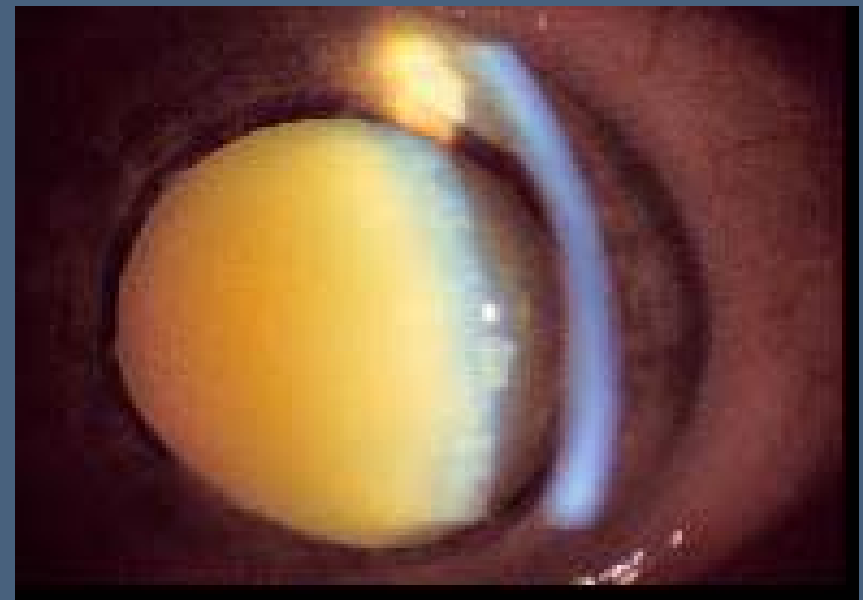
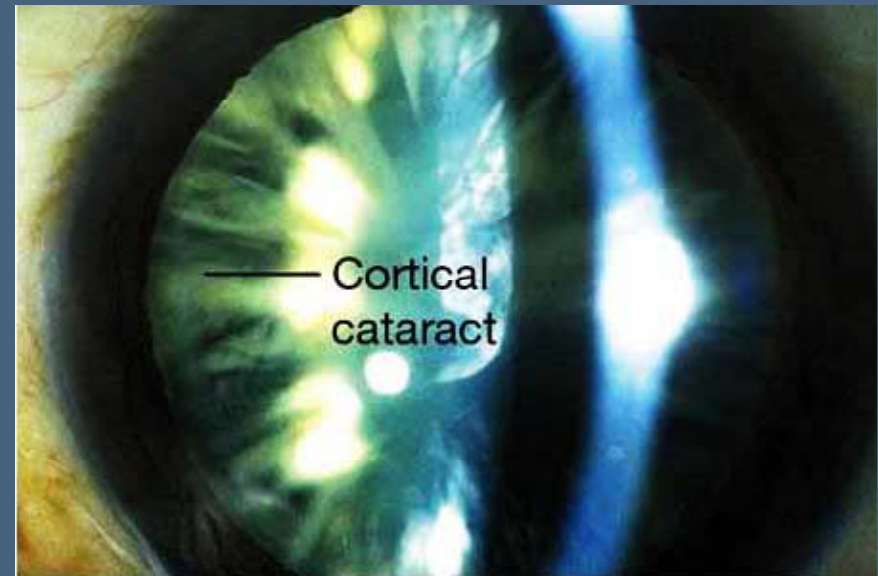


Anterior polar cataract

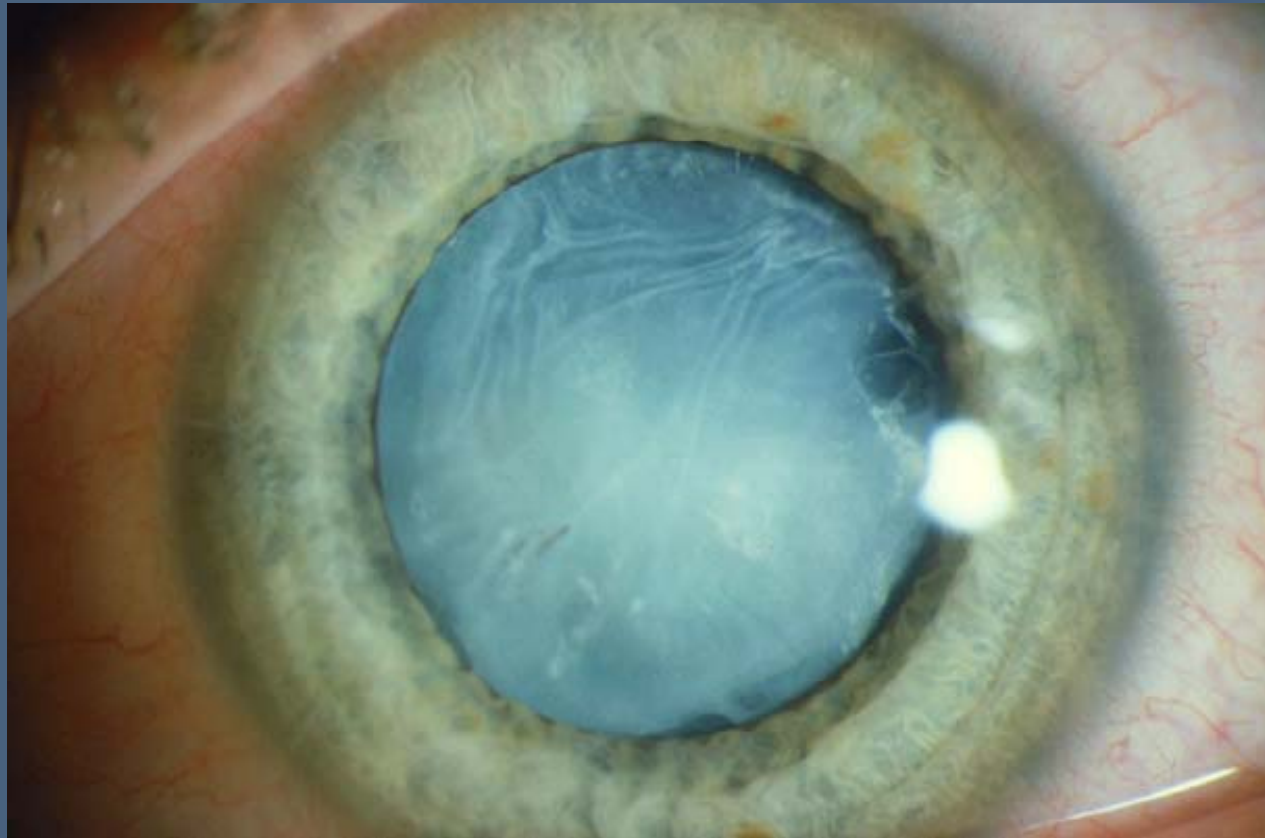


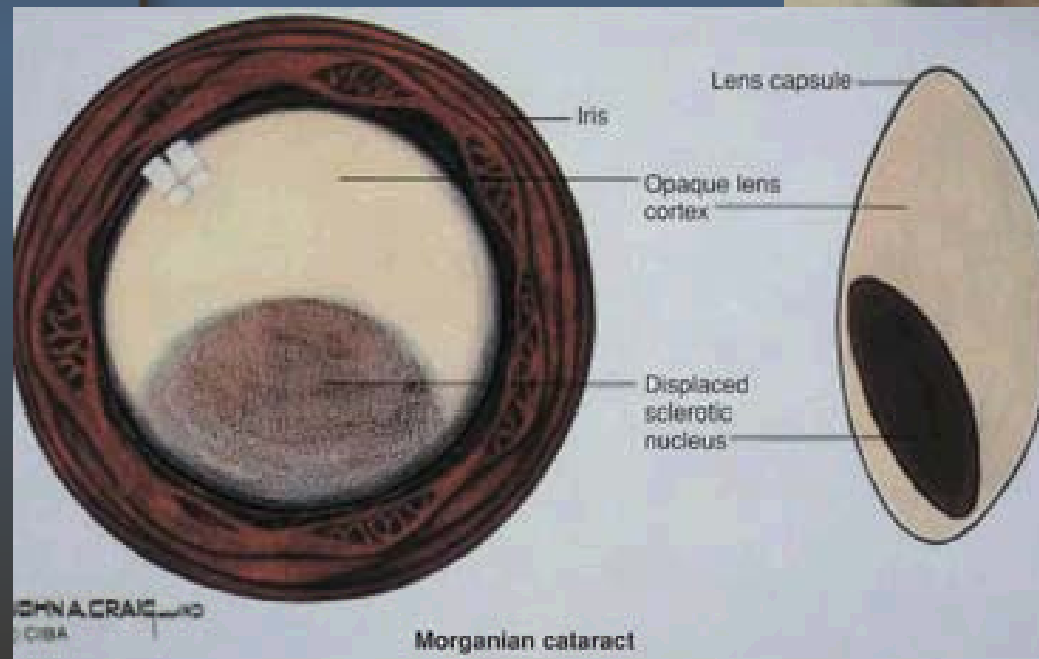
Primary cataract

- Juvenile cataract
- Presenile cataract : 35-40 years
- Senile cataract : aging process, > 40 years
 - Nuclear sclerosis
 - Cortical cataract ; immature, mature, intumescent, hypermature, morgagnian cataract
 - Subcapsular cataract ; anterior, posterior (may from secondary cause)



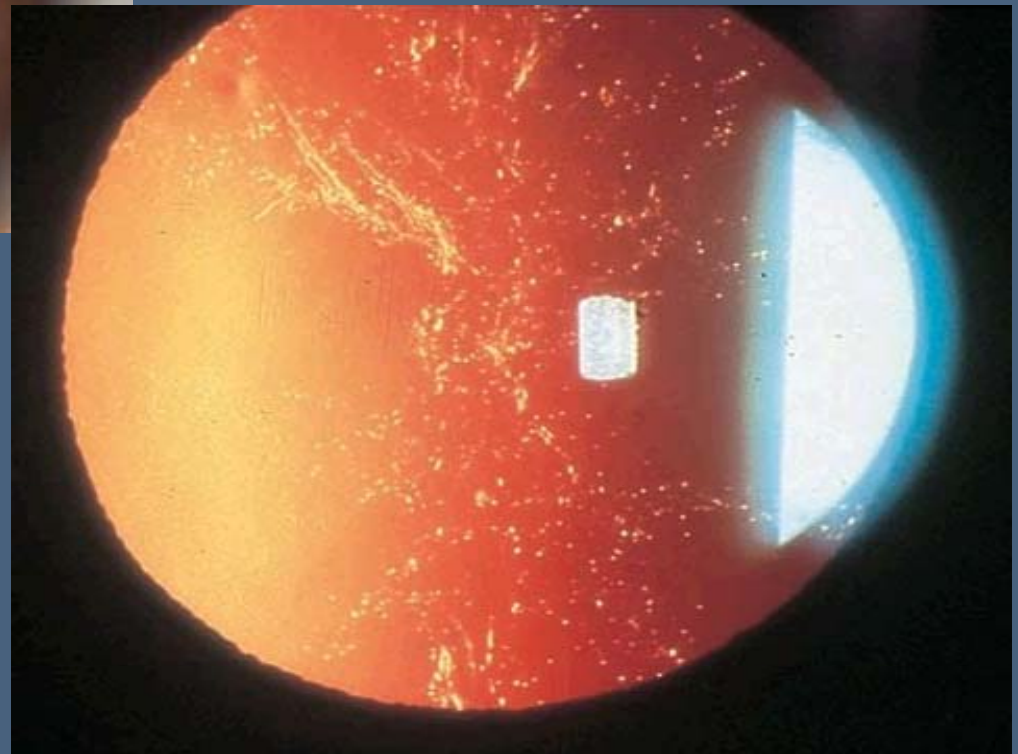
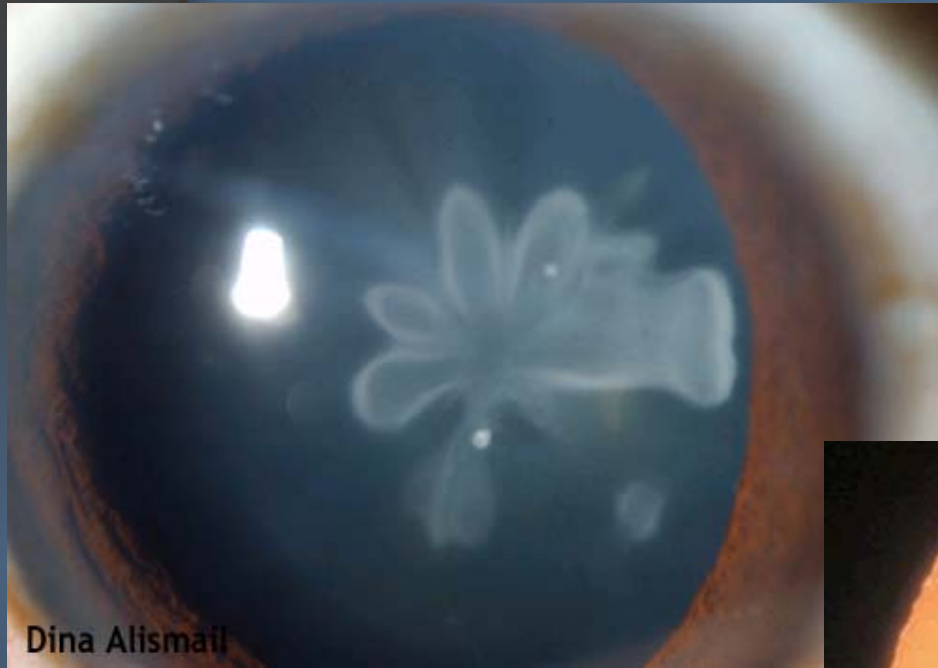
Hypermature cataract



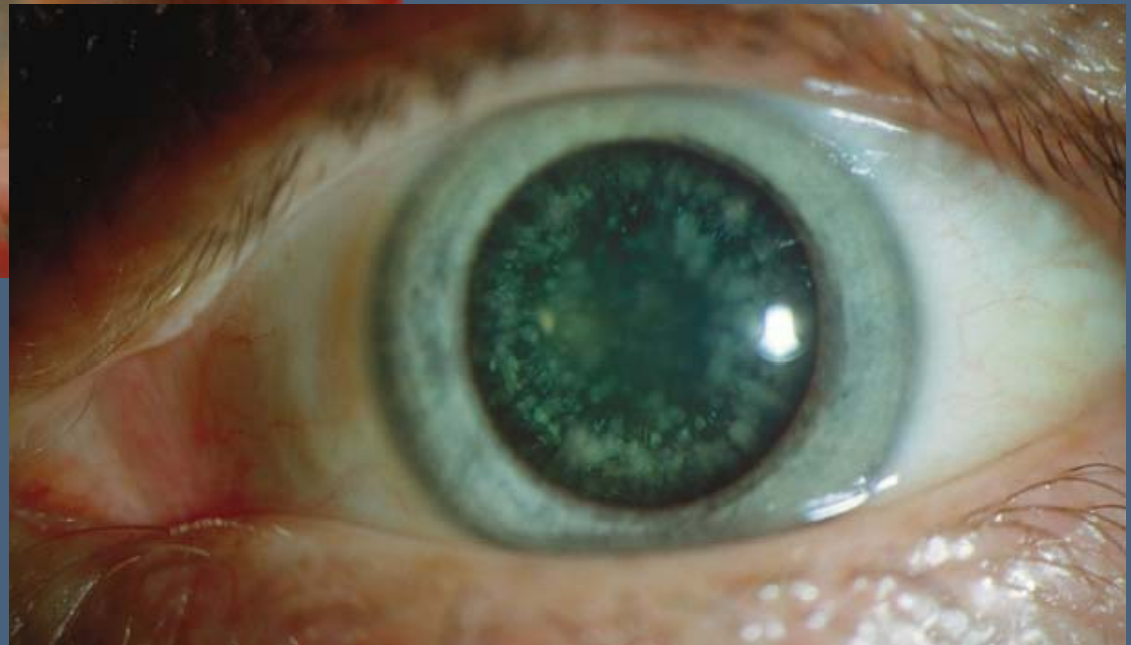
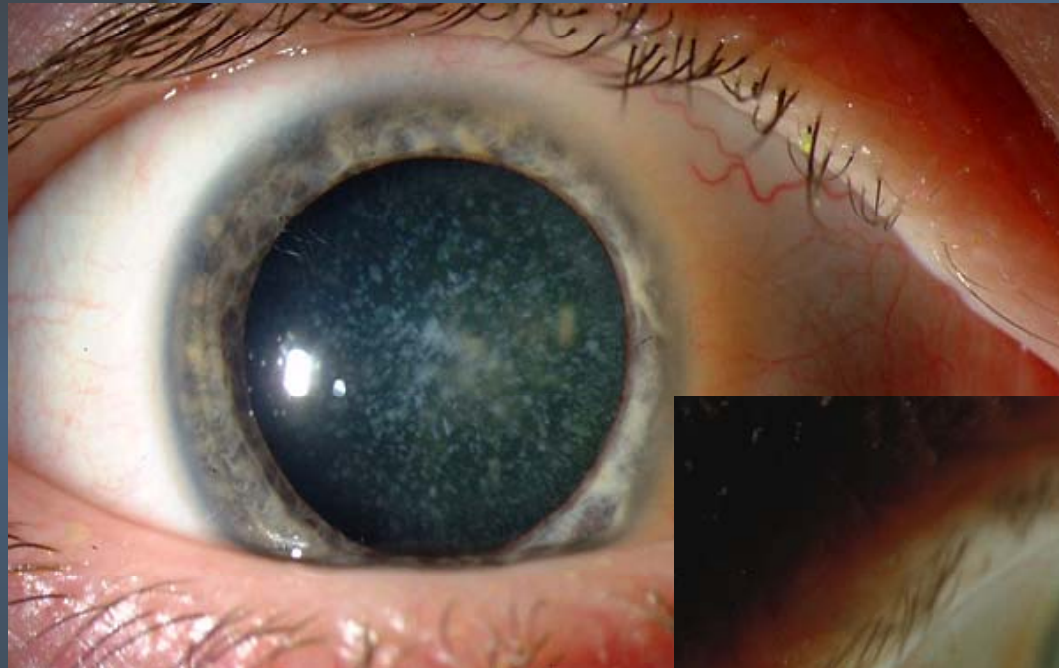


Secondary cataract

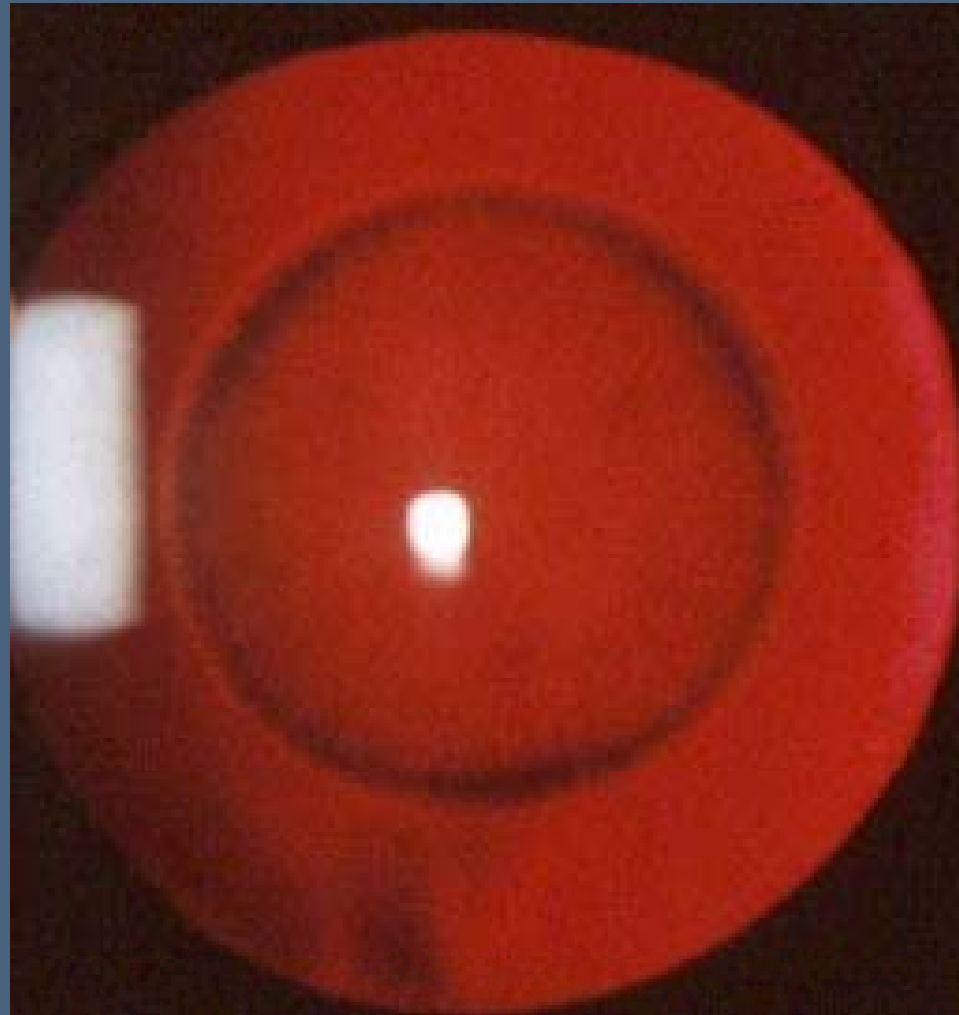
- **Extraocular disorder**
 - **Traumatic** : mechanical, physical
 - **Metabolic** : DM (fluctuation of vision, myopia), Wilson's disease (ASC)
 - **Toxic** : steroid, echothiophate iodide, phenothiazines
 - **Systemic disease** : hyperparathyroidism, myotonic dystrophy, galactosemia, Down's syndrome, trisomy 18, trisomy 13



Cerulean (blue-dot) cataract



Oil droplet cataract in Galactosemia



Polychromatic (Christmas tree) cataract



American Academy of Ophthalmology

Sunflower cataract



Snowflake cataract



Secondary cataract

- Intraocular disorder
 - uveitis esp. chronic uveitis
 - retinal detachment
 - retinitis pigmentosa
 - intraocular neoplasm

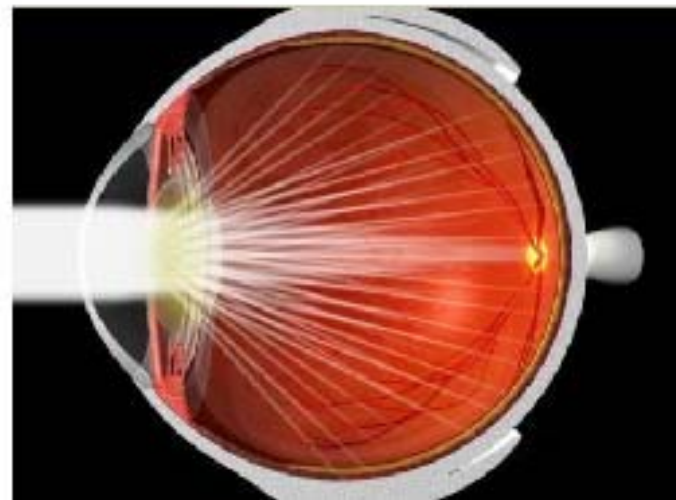
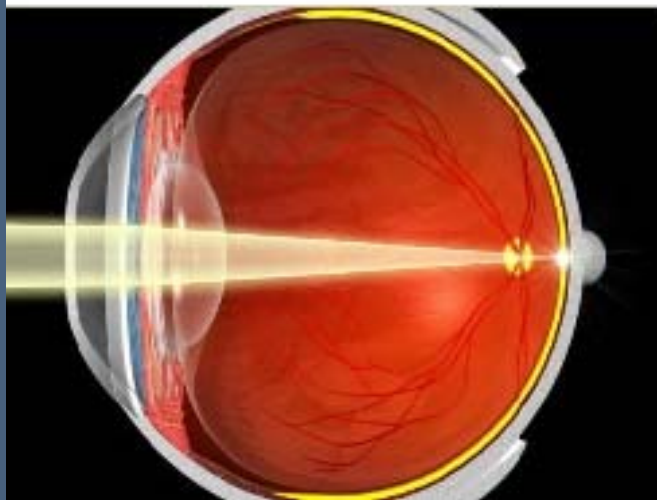
Basic information

- Symptoms of cataract
 - Image blur : depends on the size and location of opacity
 - : Axial opacities cause much more disabling visual loss than peripheral opacities
 - : Disturbance of vision, diminution, failure of vision
 - : Nuclear sclerosis may become progressively more myopic

Basic information

- Symptoms of cataract (cont.)
 - NS may develop a phenomenon called Second sight
 - Monocular double or multiple images, due to irregular refraction, prismatic effect within the lens
 - Posterior subcapsular cataract (PSC) may note a relatively **rapid decrease in vision** (esp. near vision), with glare as well as image blur and distortion
 - : PSC is frequently associated with metabolic causes : **DM, steroid use**





When to Examine

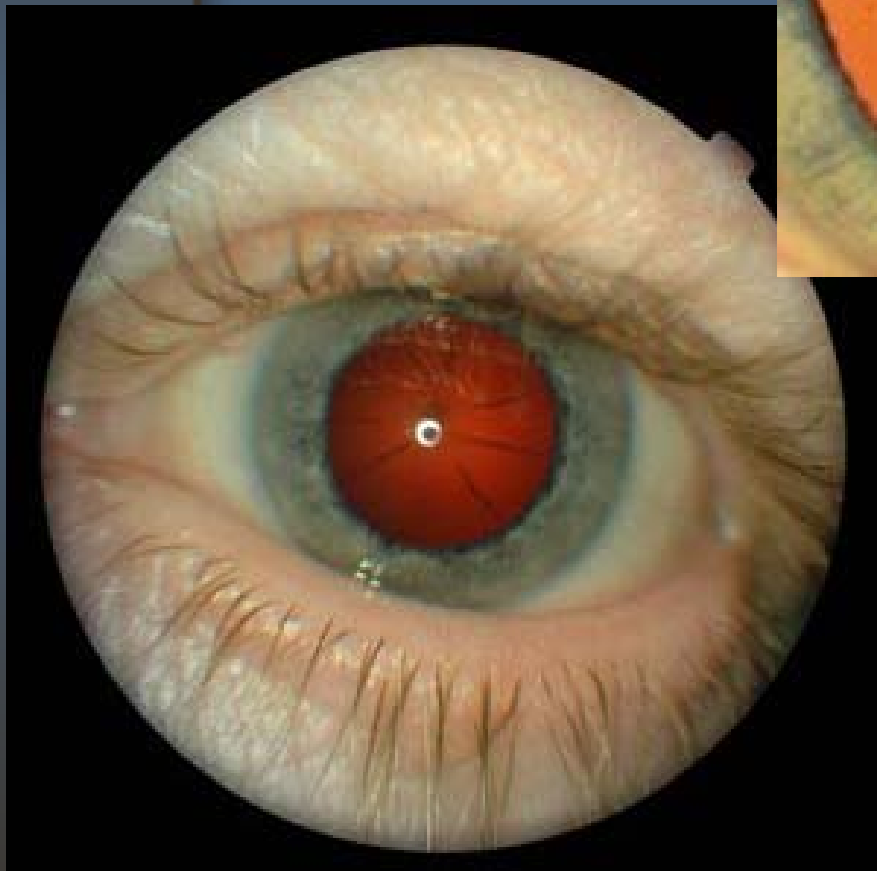
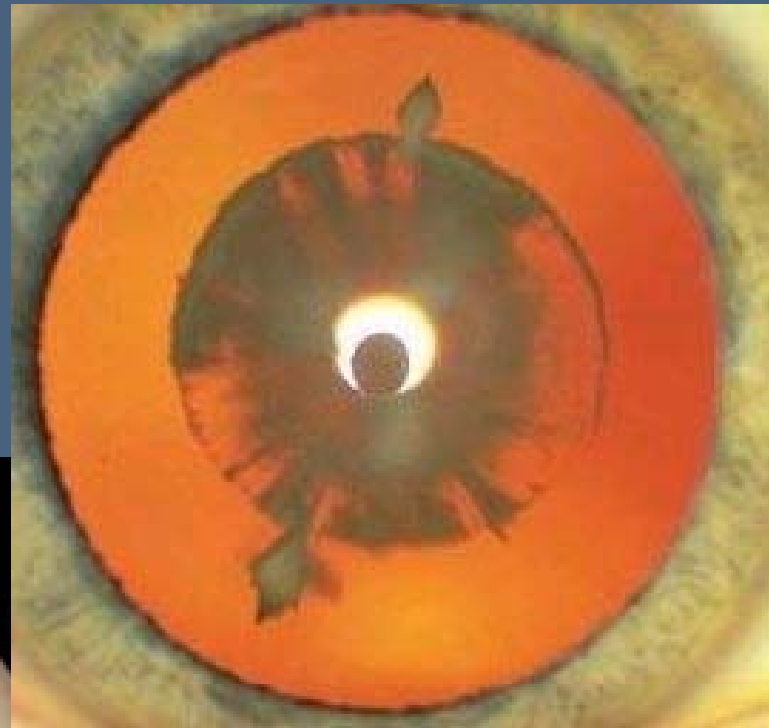
- A patient with decreasing vision
- Important to demonstrate that the retina and optic nerve are healthy
- If the lens is densely cataract
 - the risk of performing surgery for cataract without the assurance
 - RAPD, color test, 4-quadrants light projection

How to Examine

- Visual acuity
- Pupillary responses : advanced cataract would **not produce a RAPD**
- Anterior segment examination
- Ophthalmoscopy

How to Interpret the Findings

- Early cataract is not visible to the unaided eye
- Very dense cataract may appear as a white pupil, or leukocoria
- Ophthalmoscopy with plus-lens setting
 - partial cataract : black against the red reflex, poorer the red reflex



Complications of cataract

- Phacomorphic glaucoma
- Phacolytic glaucoma
- Lens-induced uveitis
- Phaco-anaphylactic endophthalmitis

Management or Referral

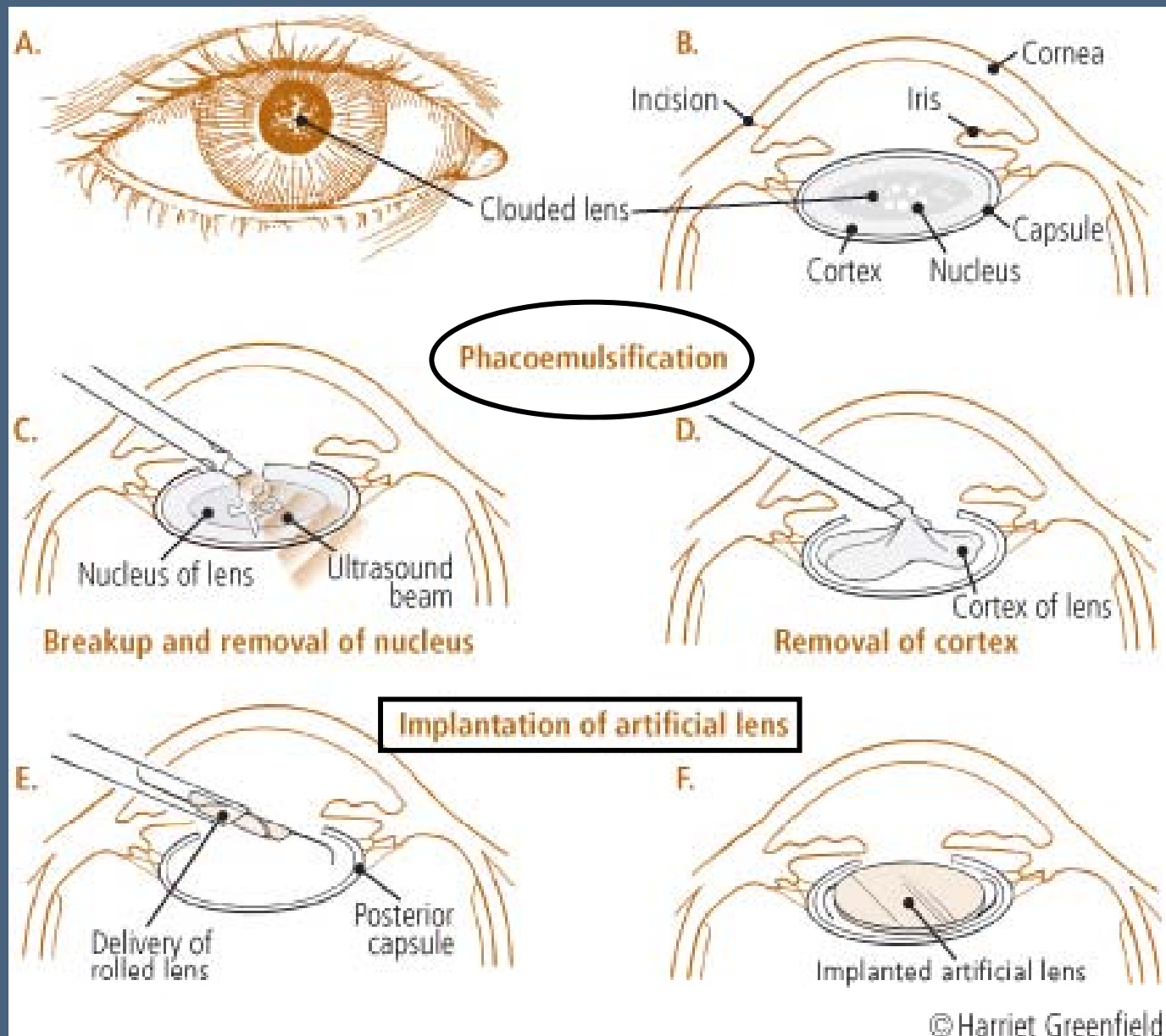
- Medical : ????
 - : Mydriatic drug
 - : Treatment of underlying cause or complications
- Indication for surgery
 1. Visual need
 2. Complications
 3. Posterior segment evaluation
 4. Cosmetic

Cataract surgery

- Preoperative evaluation
- Anesthetic techniques : TA, LA, GA
- Surgical techniques : ECCE, PE, PPL, ICCE
- Postoperative care and complications management
- Visual rehabilitation

Surgical techniques

- Extracapsular cataract extraction (ECCE)
- Phacoemulsification (PE)
- Intracapsular cataract extraction (ICCE)
- Pars plana lensectomy (PPL)
- Couching

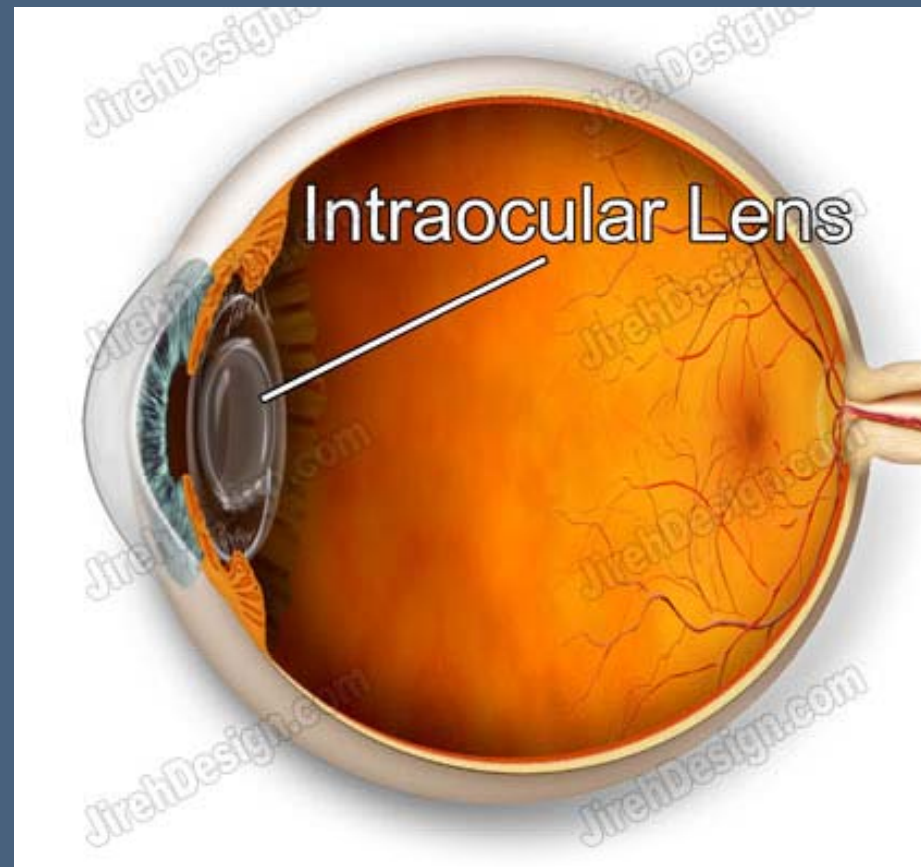


Couching



Visual rehabilitation

1. Aphakic glasses : +10 to +12 D
2. Contact lens : $\geq +10$ D
3. Intraocular lens : $\sim +20$ D
 - posterior chamber lens : in the bag, in the sulcus, scleral-fixated
 - anterior chamber lens : angle-fixated, iris-fixated lens



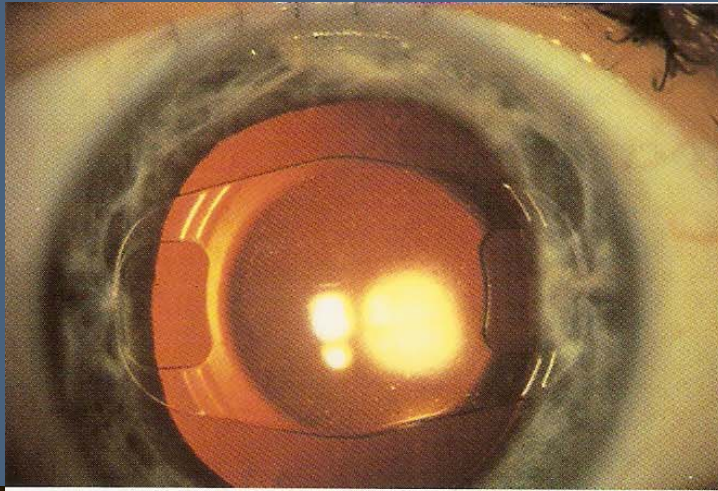


FIGURE 21.1. 5.0-mm diameter Artisan phakic IOL. Note that pupil dilation is not affected.

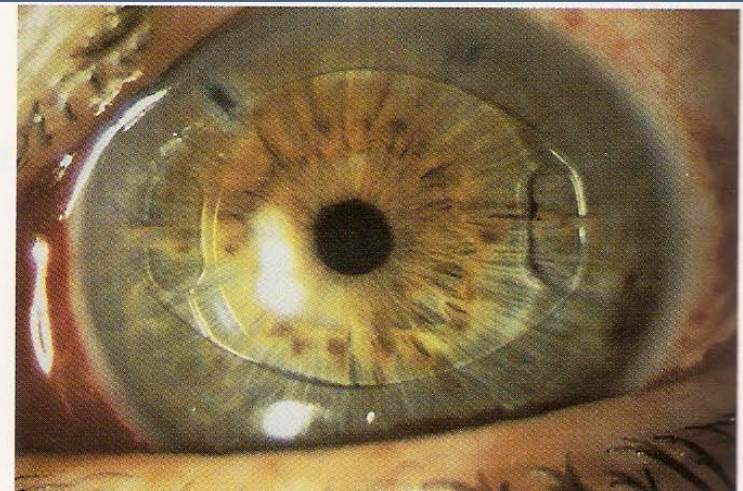
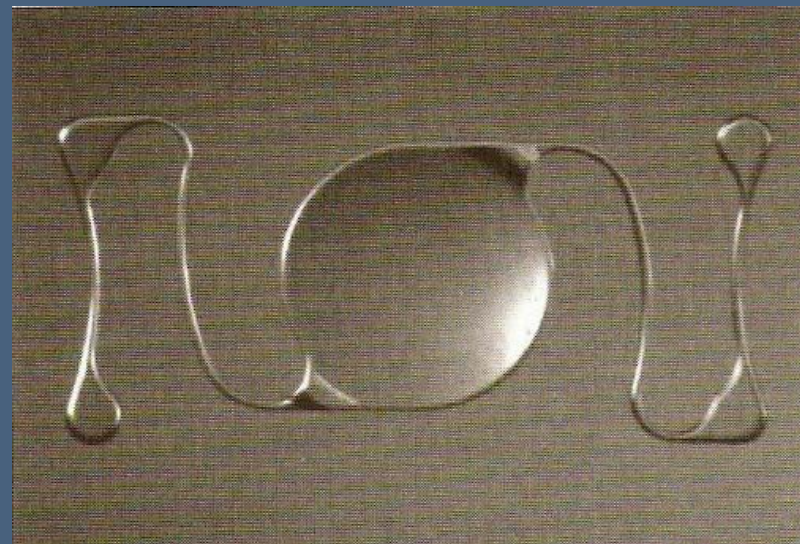


FIGURE 21.2. 6.0-mm diameter Artisan phakic IOL.



Complications of Cataract Surgery

- Intraoperative

Expulsive hemorrhage

Iris trauma

A/C hemorrhage

Ruptured PC

Corneal injury

Lens dislocation

- Postoperative

Early

: endophthalmitis, glaucoma,
uveitis, iris prolapse

Late

: posterior capsule opacity,
retinal detachment, cystoid
macular edema, corneal
decompensate,
astigmatism*

Macular Degeneration

Relevance

- Leading cause of **irreversible** **central visual loss** (20/200 or worse) in people over 50 years of age in U.S.A.
- *Certain types of AMD are treated effectively with laser*
- Important to recognize this entity and to refer for appropriate care

Basic Information

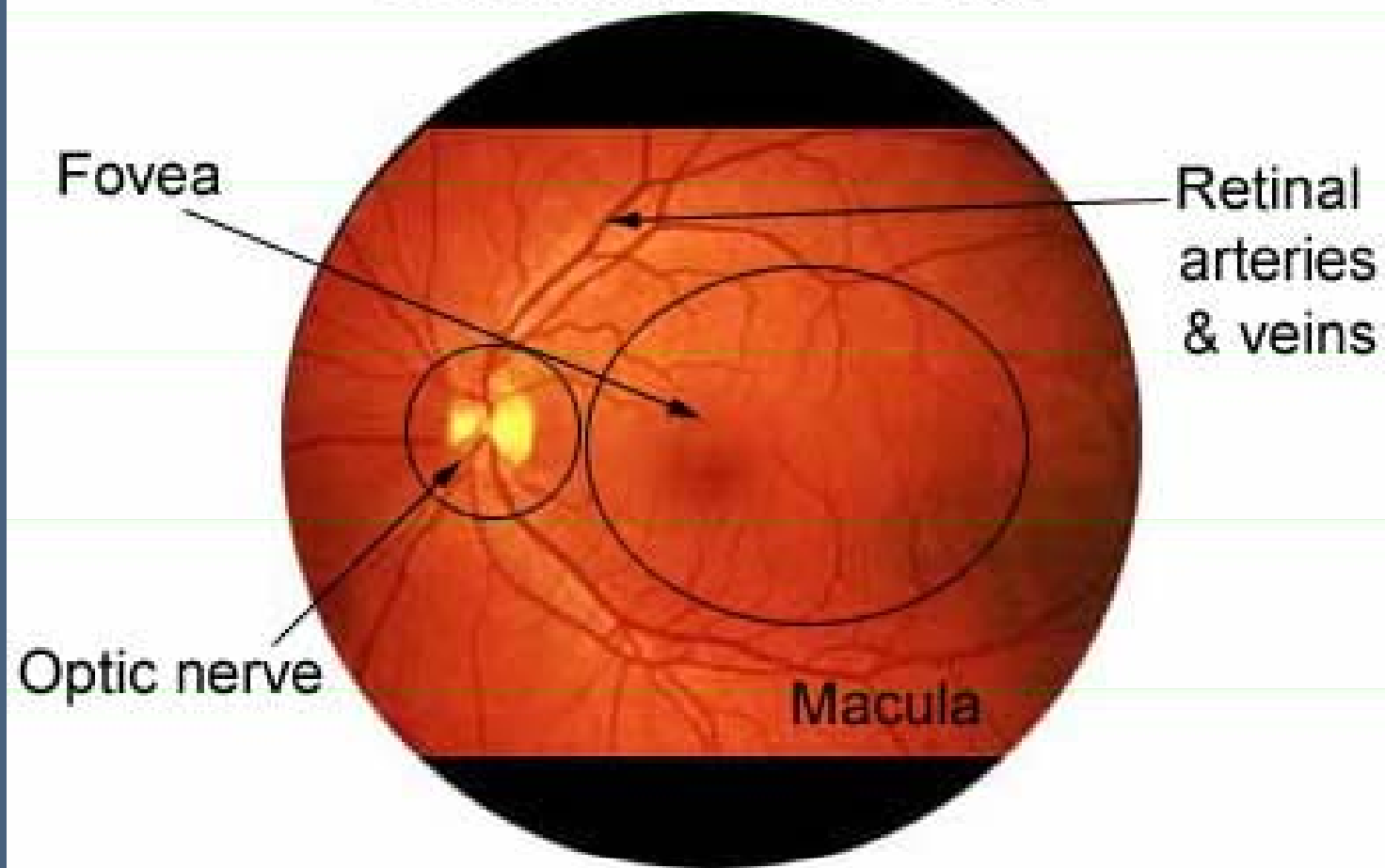
- **Macular Anatomy**
 - Oval area
 - 2 disc diameter temporal and slightly inferior to the optic disc
 - Composed of both rods and cones photoreceptor cell
 - Responsible for detailed, fine, central vision

Basic Information

- **Macular Anatomy**

- Central macular is **avascular** and appears darker than the surrounding retina
- Fovea is an oval depression in the center of macula, there is a **high density of cones but no rods are present**
- The central depression of the fovea may act like a concave mirror during ophthalmoscopy, producing a foveal reflex

Normal Macula



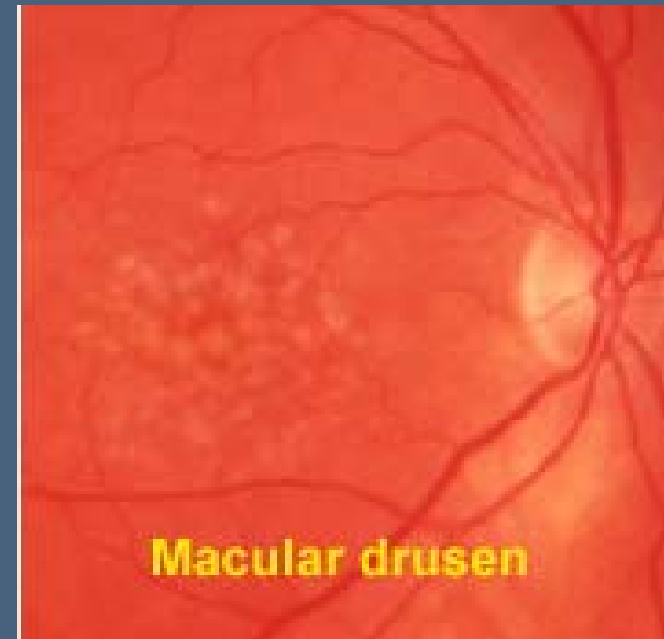
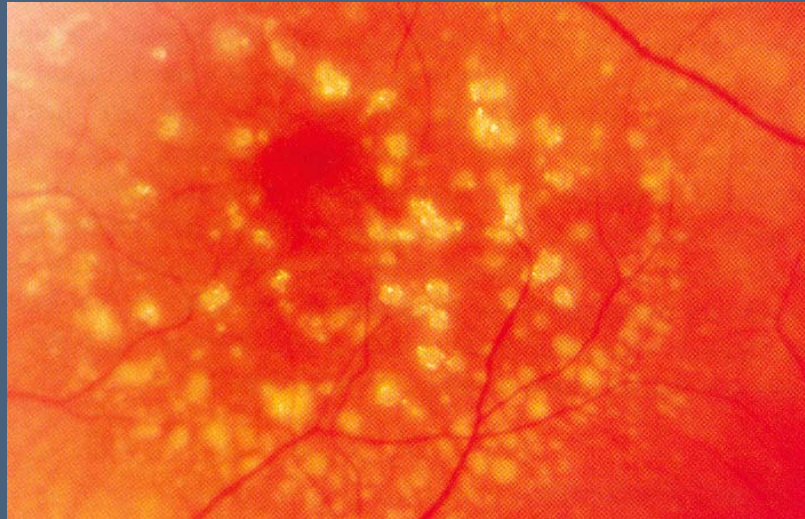
Basic Information

- Age-Related Macular changes
 1. Drusen
 2. Degenerative change in the retinal pigmented epithelium (RPE)
 3. Subretinal neovascular membranes (SRNM)

Drusen

- Hyaline nodule (or Colloid bodies) **deposited in Bruch's membrane**
- Small or large, discrete, irregular shapes, and indistinct edges
- Normal, near-normal VA with minimal metamorphopsia
- May be seen **increasing age, during retinal or choroidal degeneration** in disease states

Drusen



Drusen



Courtesy of J. Donald Gass, MD and Timothy D. Polk, MD
©1994 Bascom Palmer Eye Institute

Degenerative changes in RPE

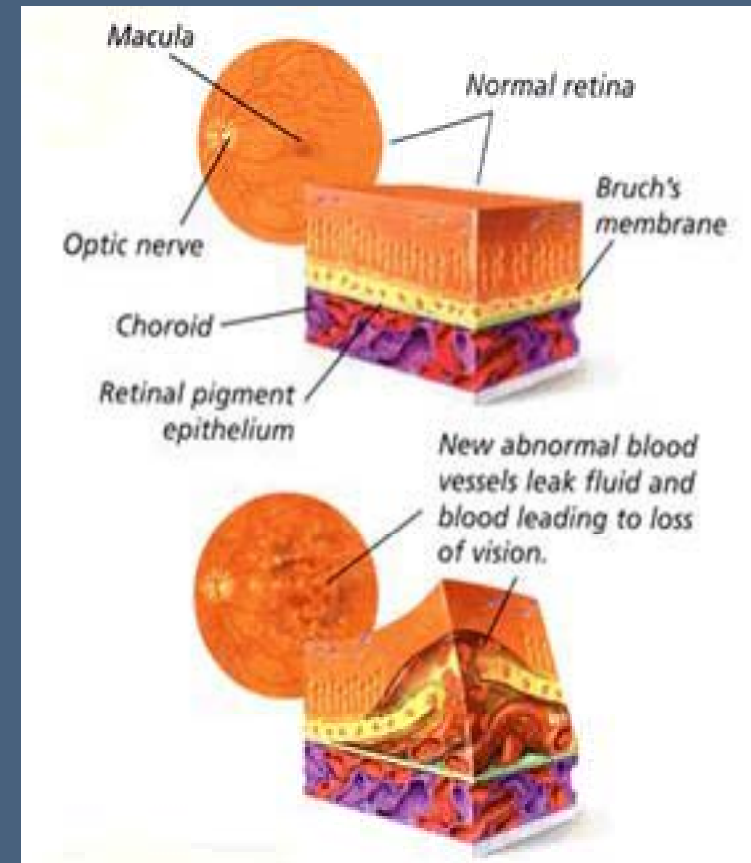
- May occur with or without drusen
- Manifested as clumps of hyperpigmentation or depigmented atrophic areas
- The effect on visual acuity is variable



Courtesy of J. P. Dunn, MD
©1991 Wilmer Eye Institute

Subretinal neovascular membranes

- 20% of eye with AMD
- The extension of vessels from the inner choroid layer into the subretinal space
(defect has developed in Bruch's membrane)



Subretinal neovascular membranes

- Associated with subretinal hemorrhage, fibrosis, RPE degeneration, photoreceptor atrophy
- Hemorrhage or subretinal fluid may result in acute visual loss
- Larger the membrane and the closer to the center of the fovea, the worse prognosis for good central vision



Courtesy of J. Donald Gass, MD and Timothy D. Polk, MD
©1993 Bascom Palmer Eye Institute

Subretinal neovascular membranes

Other causes of SRNM

1. High myopia
2. Angioid streaks
3. Presumed ocular histoplasmosis
4. Traumatic choroidal rupture

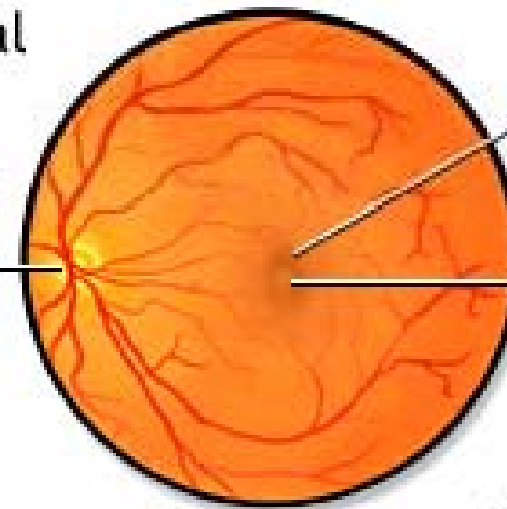
AMD classification

- Dry AMD (atrophic, nonvascular)
 - Majority of AMD
 - Suffer mild central visual loss
 - Drusen
 - Geographic atrophy (10% of severe visual loss)
- Wet AMD (vascular, exudative)
 - Subretinal NVM
 - Subretinal scar (disciform scar) 90% of AMD patients with severe visual loss



Normal
retina

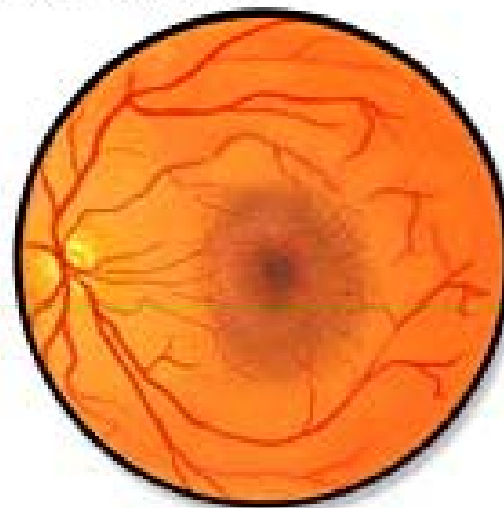
Optic
disc



Macula

Fovea
centralis

"Wet" macular
degeneration



"Dry" macular
degeneration



Dry AMD



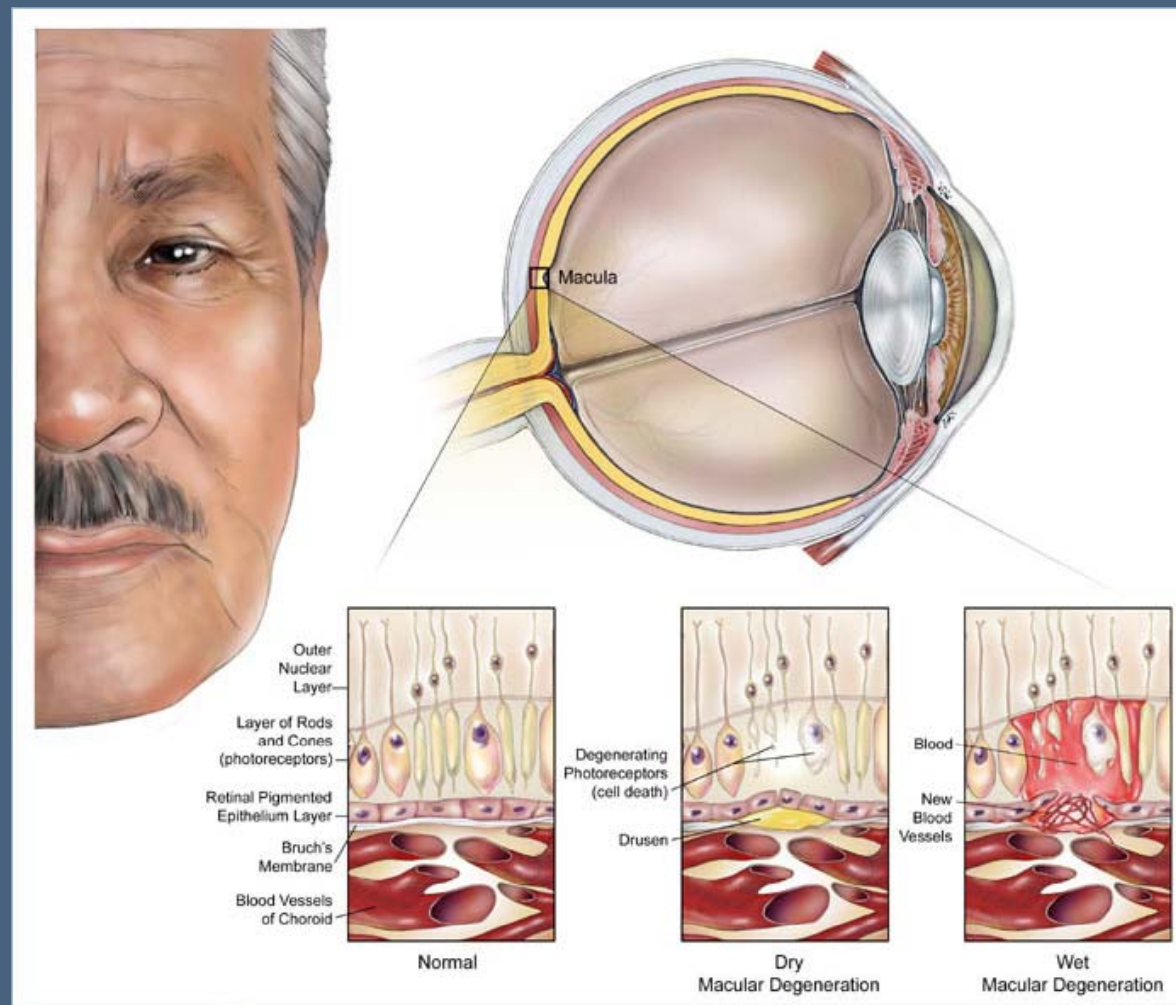
Courtesy of Mark Metzler and C. P. Wilkinson, MD
©1979 Greater Baltimore Medical Center

Dry AMD



Courtesy of Mark Metzler and C. P. Wilkinson, MD
©1978 Greater Baltimore Medical Center

Wet AMD





Courtesy of J. Donald Gass, MD and Timothy D. Polk, MD
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When to Examine

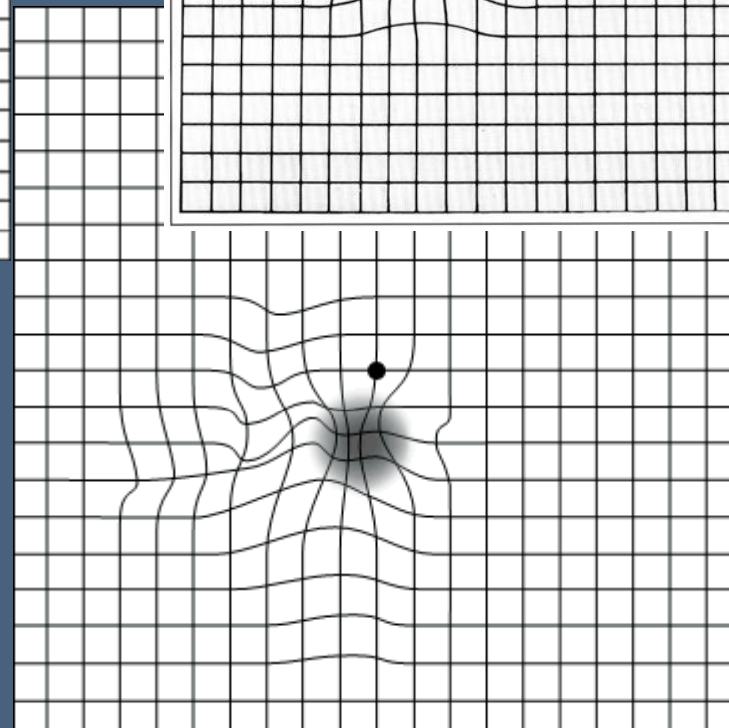
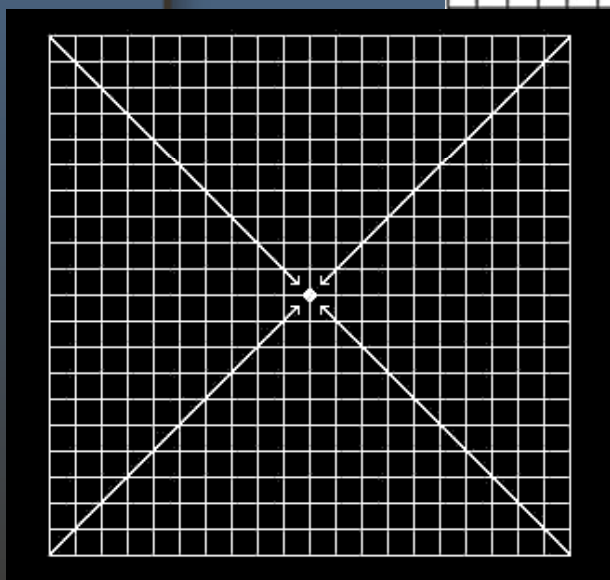
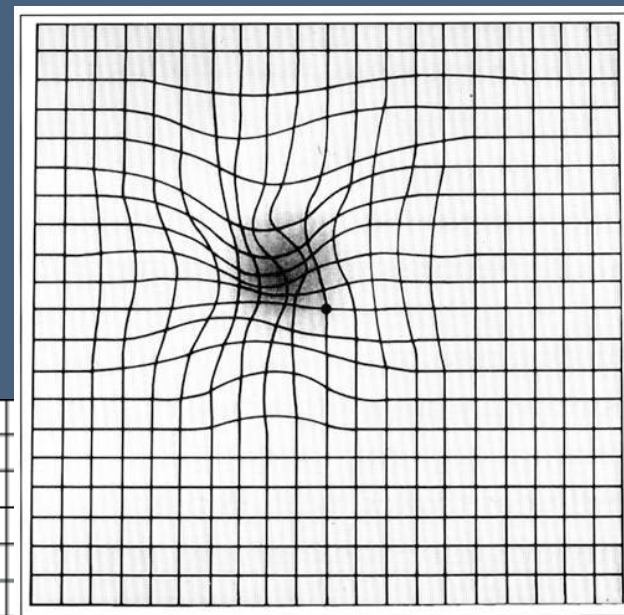
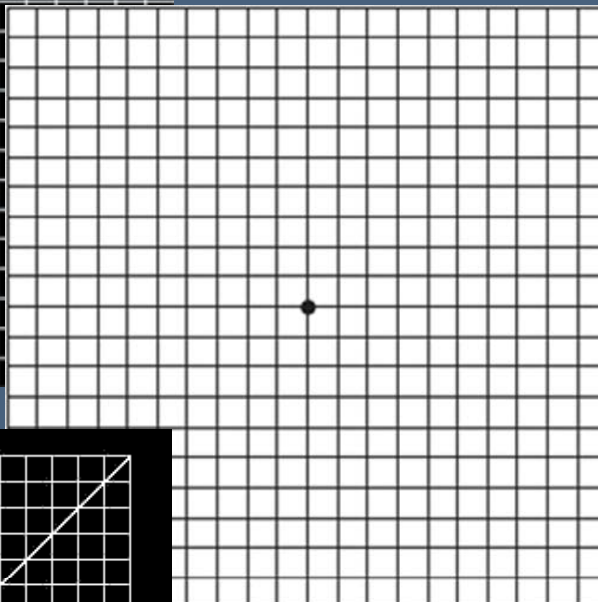
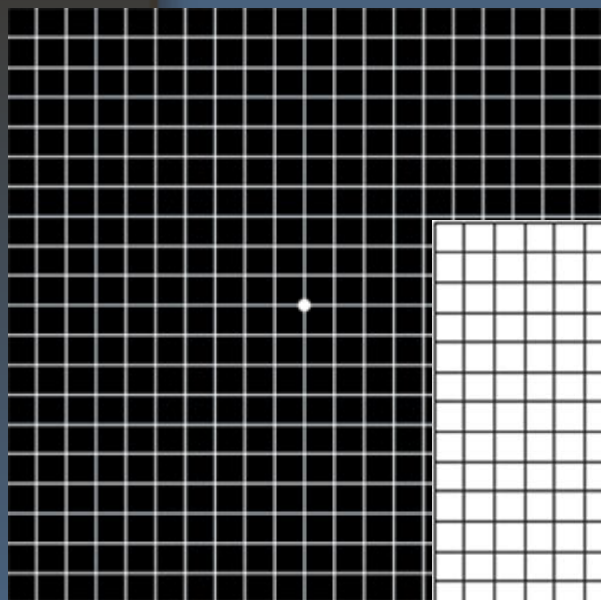
- Any patient with decreasing vision
- Patient with decreased or distorted central vision should be examine the macula



How to Examine

- Visual acuity
- Amsler grid testing
- Ophthalmoscopy : dilate pupil for adequate examination
- Additional studies : stereoscopic slit-lamp examination, fluorescein angiography

Amsler grid test



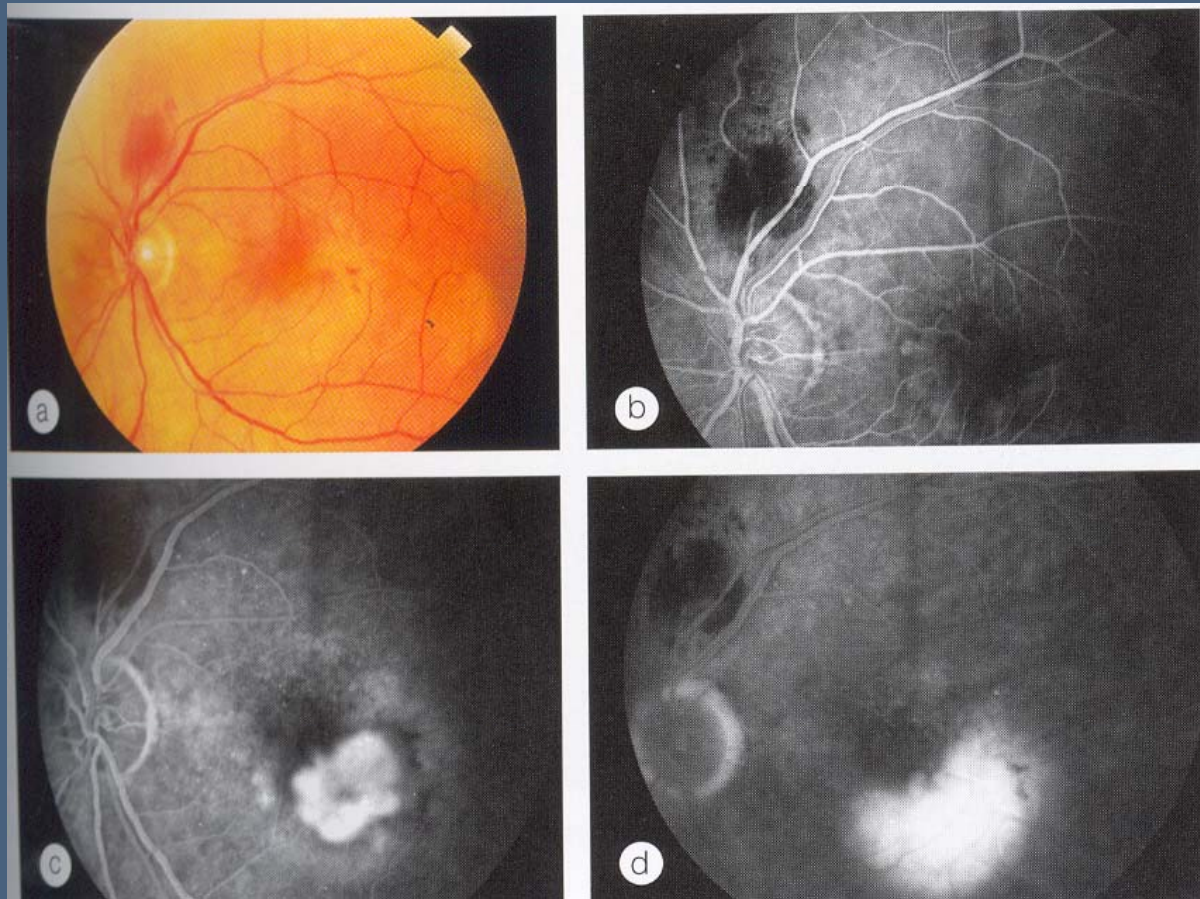
Amsler grid

- Irregularities in lines that are wavy, seem to bow or bend, appear gray or fuzzy, or absent in certain areas, **indicating a scotoma**
- Held at normal reading distance of **30 cm.** from the eye
- Measures **10 degree on each side of fixation**
- Allows for an evaluation of 5.36 mm in all directions from the center of the macula

Fluorescein angiography

- Necessary to identify neovascularization and is mandatory before considering laser surgery
- RPE acts as physical and optical barrier to fluorescein
- Identification of RPE defects
- Indocyanine green (ICG) is another dye used to demonstrate new vessels

Fluorescein angiography



How to Interpret the Findings

- Appearance of the macula often does not accurately predict the visual acuity
- Important signs to check for AMD
 - drusen
 - areas of increased or decreased pigmentation
 - subretinal exudate, hemorrhage, NV
- Absence of foveal reflex and a mottle appearance of the RPE are the early signs of macular disease

Referral

One or more of the following

- A recent onset of decreased VA
- A recent onset of metamorphopsia, or distortion of central vision
- A recent onset of a scotoma
- Any ophthalmoscopic abnormalities in the appearance of the macula

Management*

1. Drusen and abnormalities of RPE
 - micronutrient
2. Neovascular membrane complications
 - fluorescein angiogram
 - laser treatment
 - photodynamic therapy (PDT)
 - anti-VEGF
 - surgery

The Visually Impaired Patient

- The patient with AMD may have **very poor central vision**, but will tend to retain functional peripheral vision
- Visual aids, such as **high-plus magnifiers and telescopic devices**, may help the pts.



Diabetic Retinopathy

Classification

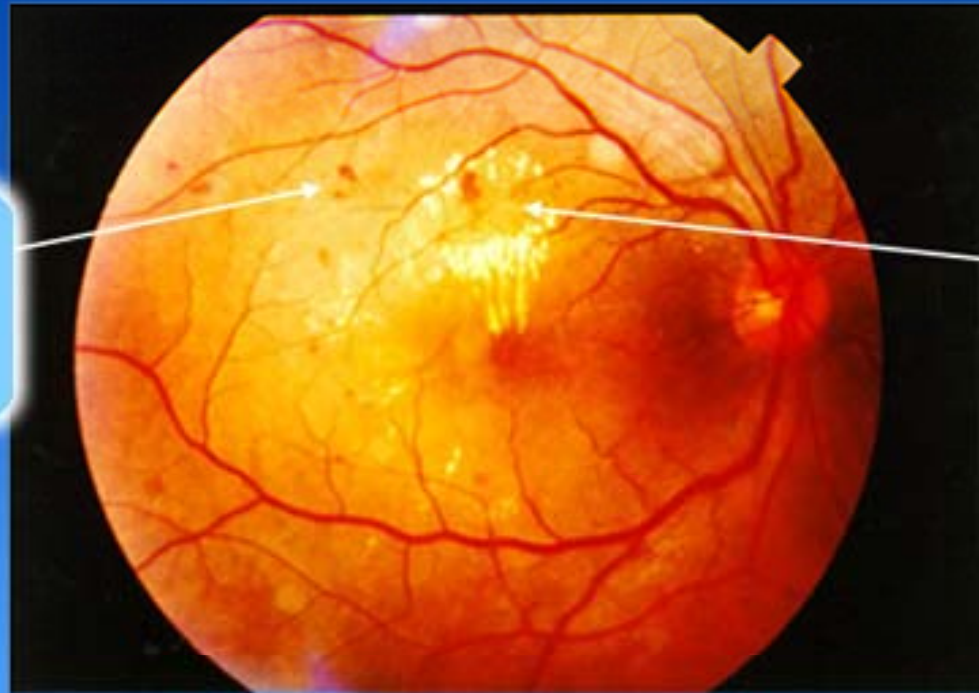
- Non proliferative DR (NPDR)
 - mild
 - moderate
 - severe
- Proliferative DR (PDR)

Signs & Symptoms

- NPDR
 - No symptoms
 - Vision loss
 - : lens edema
 - : macular edema
 - : CSME
 - : cataract
- PDR
 - No symptoms
 - Vision loss
 - : as NPDR
 - : VH
 - : TRD +/- RRD
 - : NVG
 - : macular ischemia

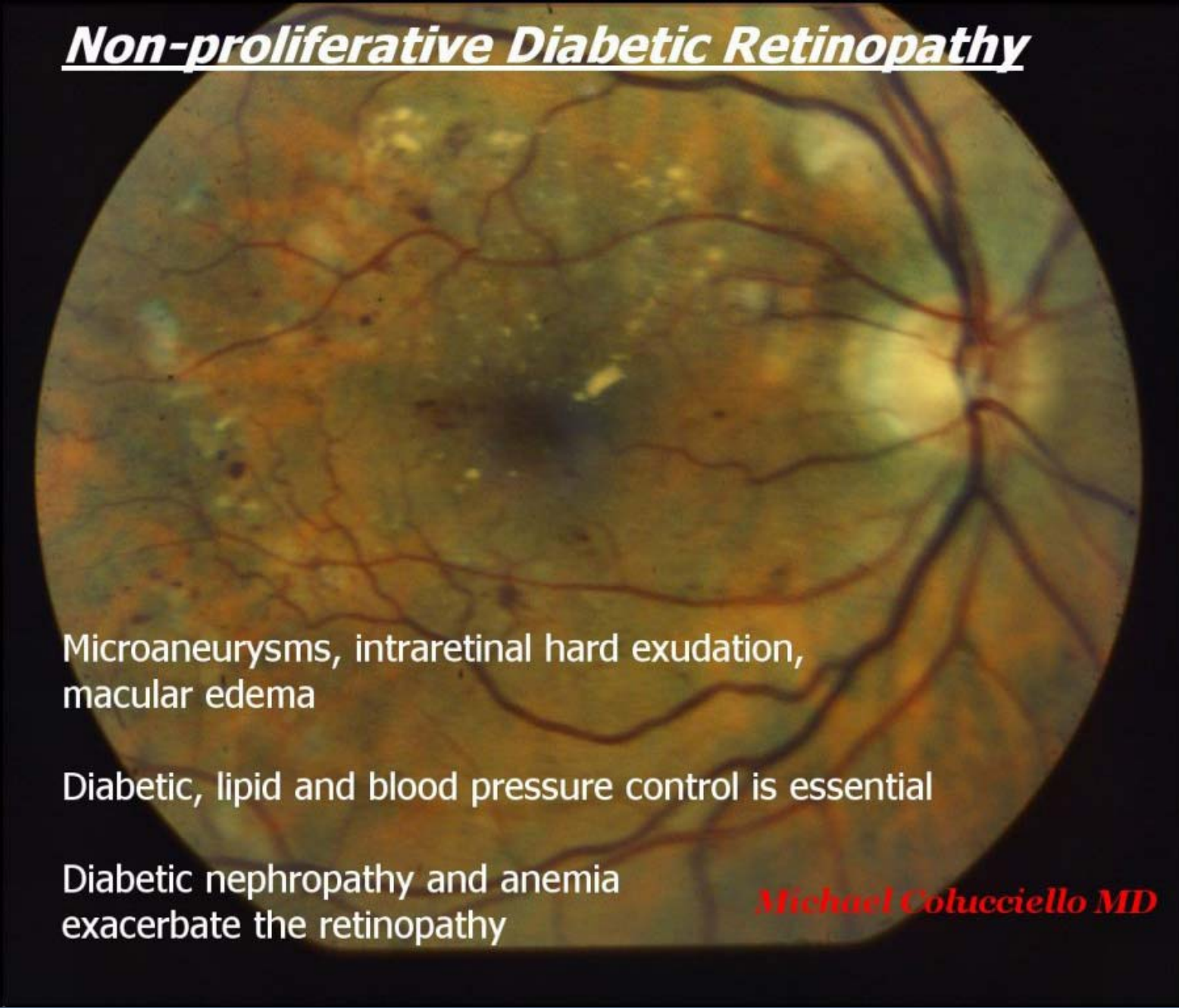
NPDR

Small
haemorrhages
(bleeds)



Leaking and
swelling from
blood vessels
damaged by
diabetes

Non-proliferative Diabetic Retinopathy

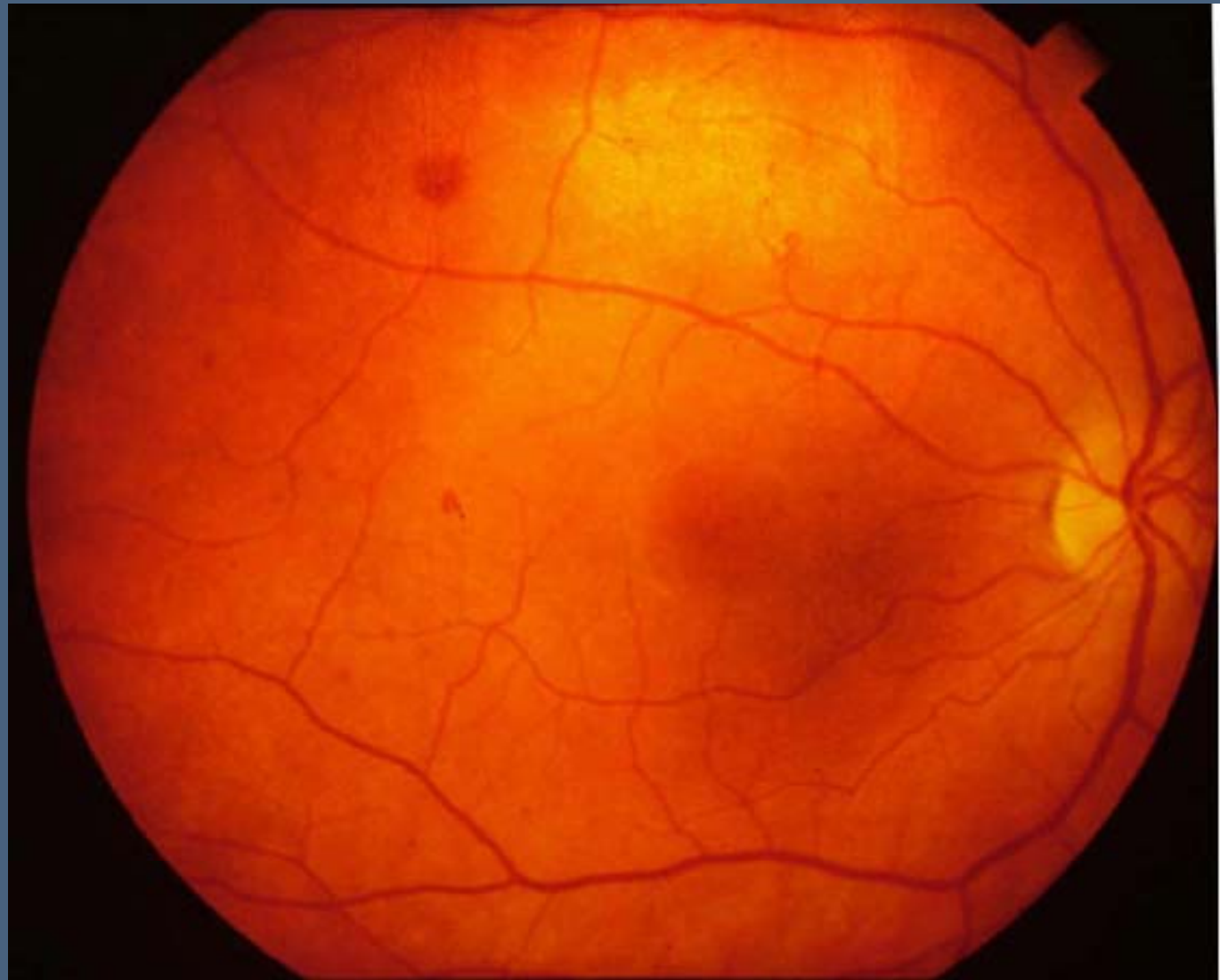


Microaneurysms, intraretinal hard exudation,
macular edema

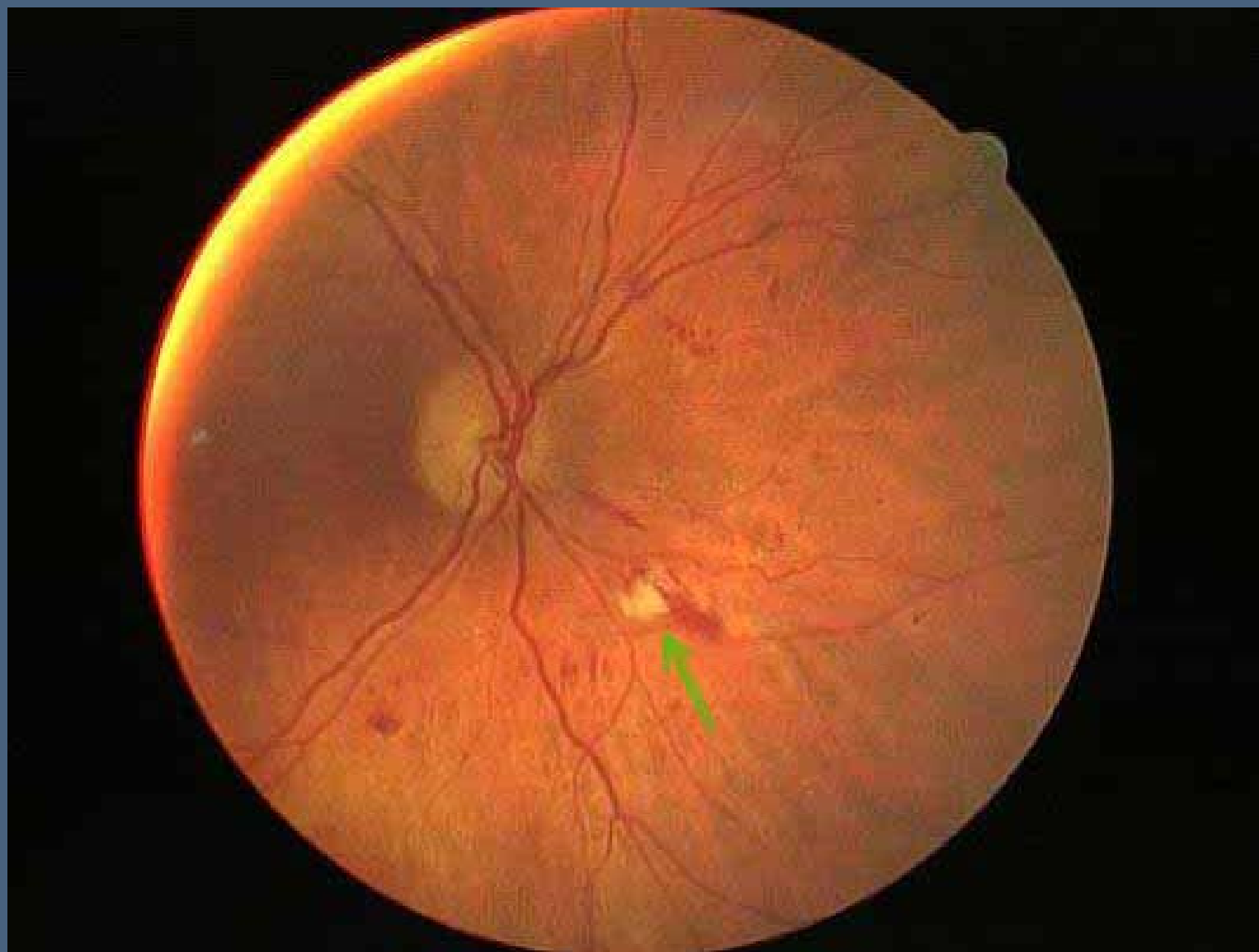
Diabetic, lipid and blood pressure control is essential

Diabetic nephropathy and anemia
exacerbate the retinopathy

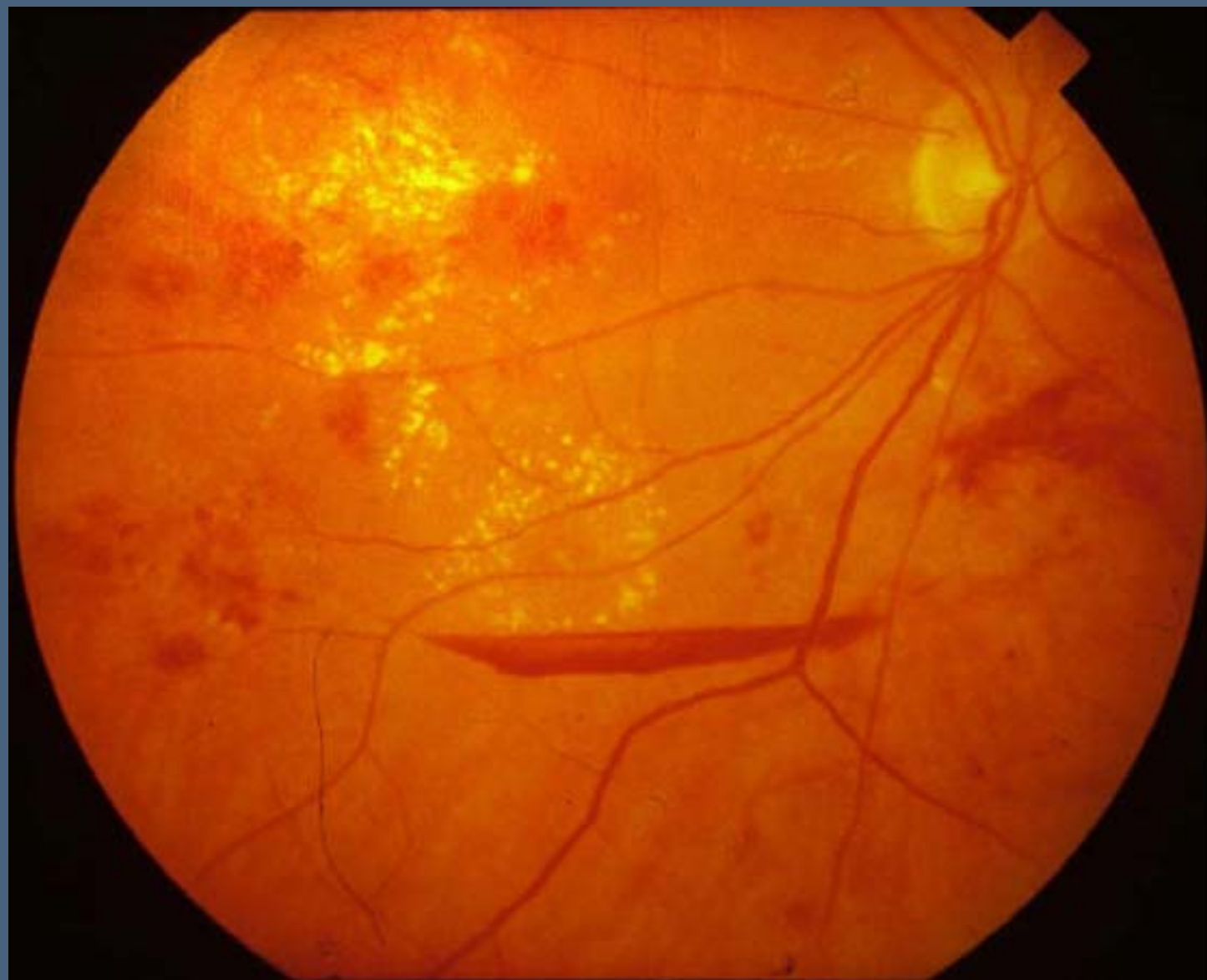
Michael Colucciello MD



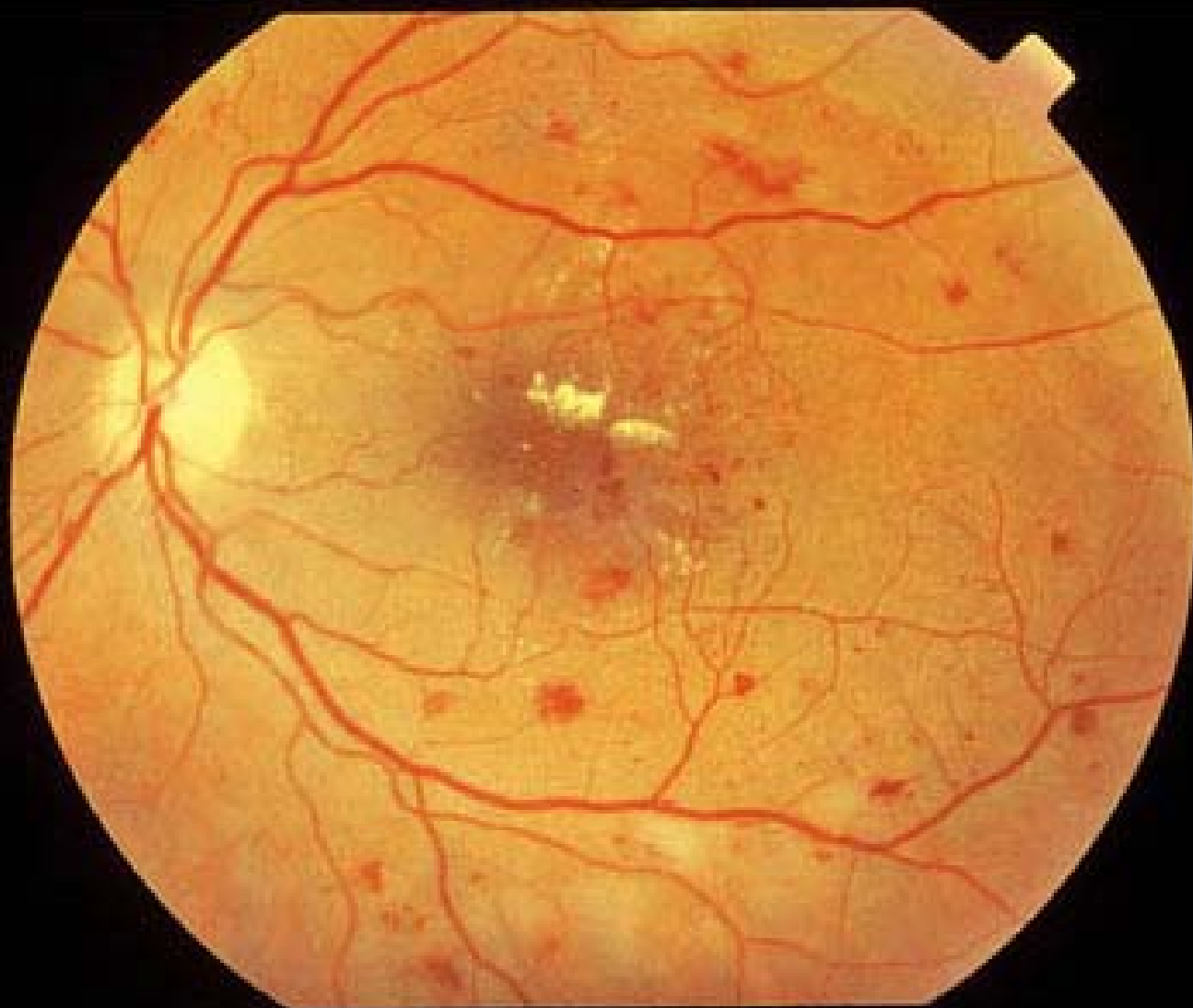


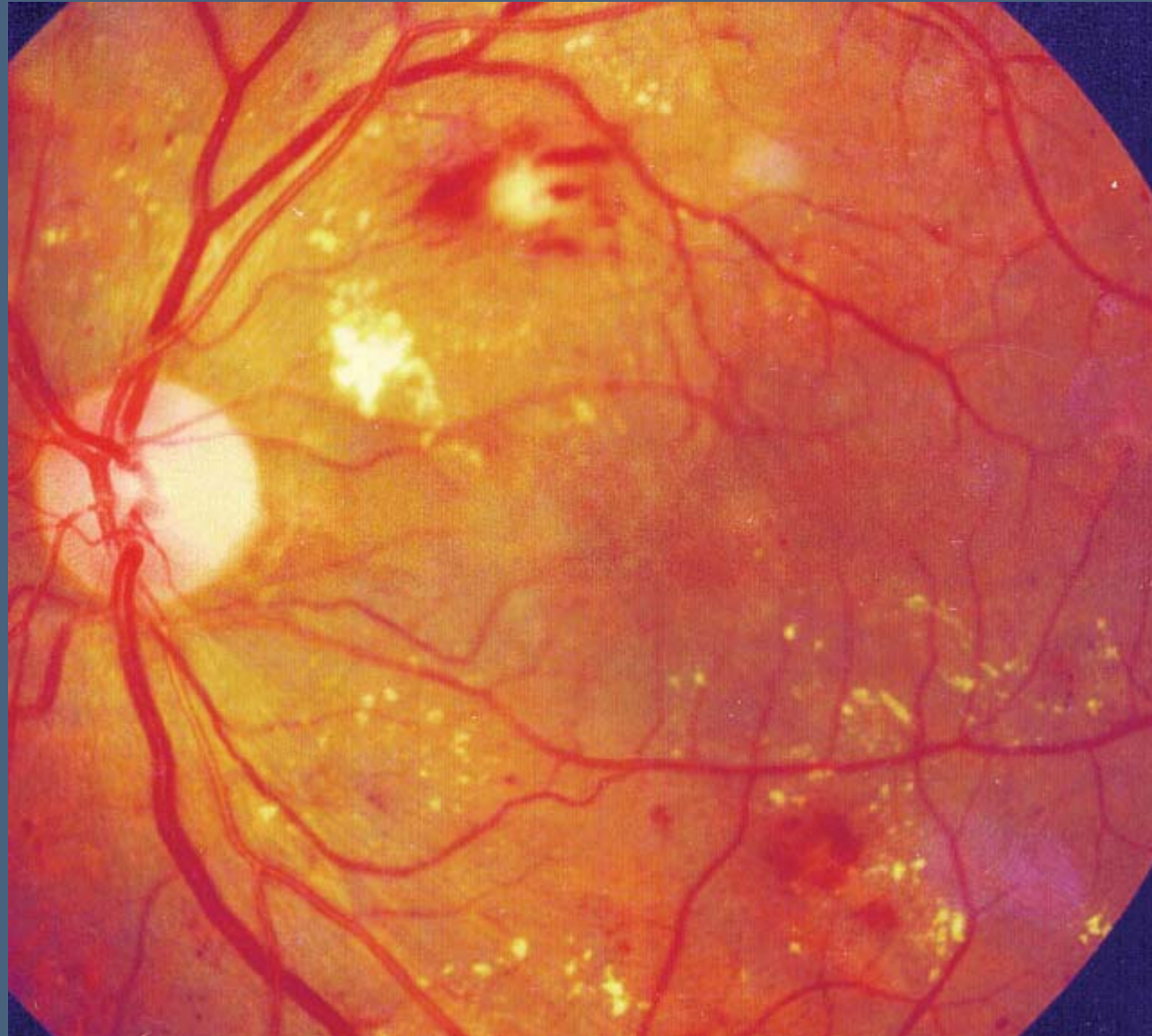




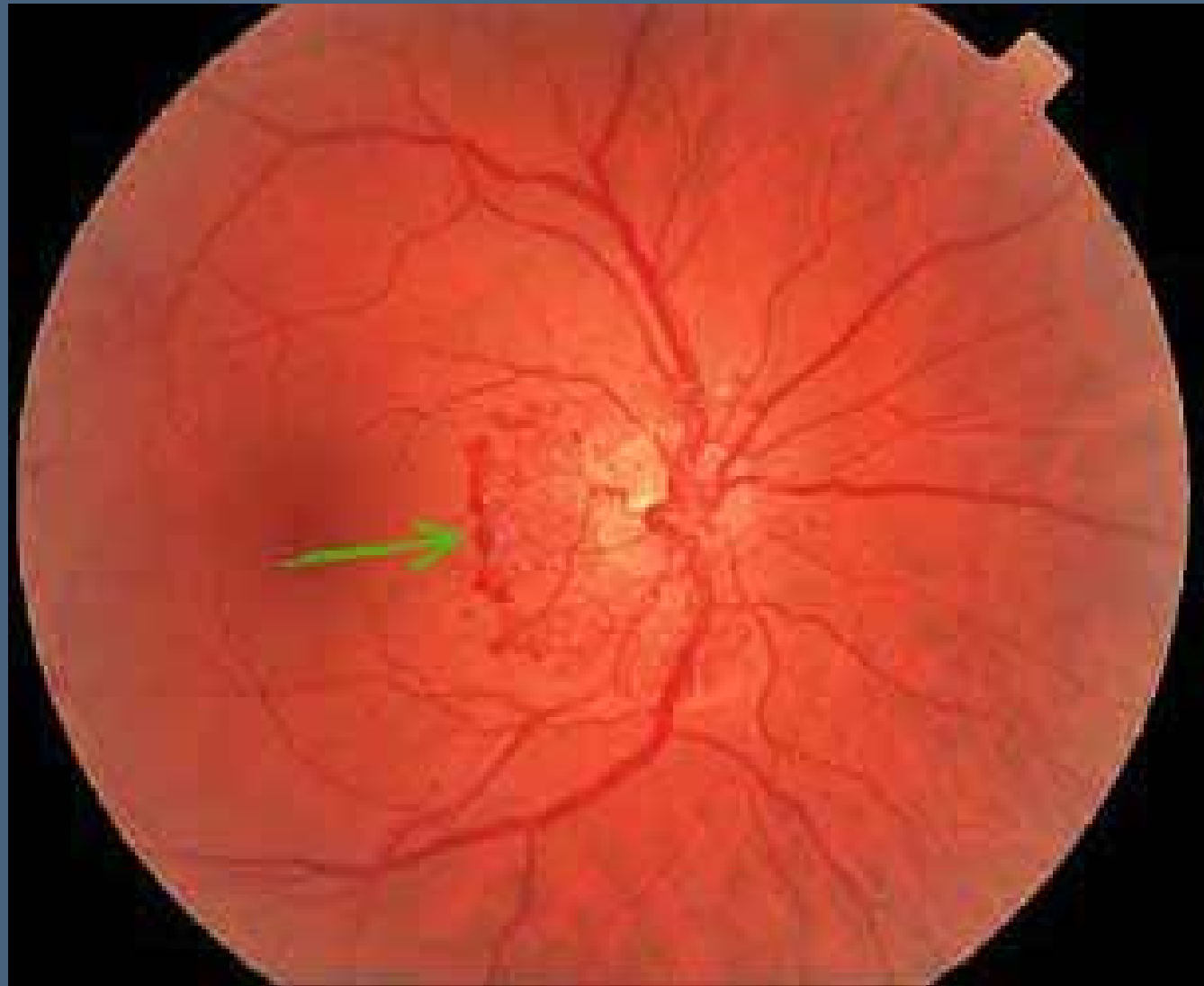


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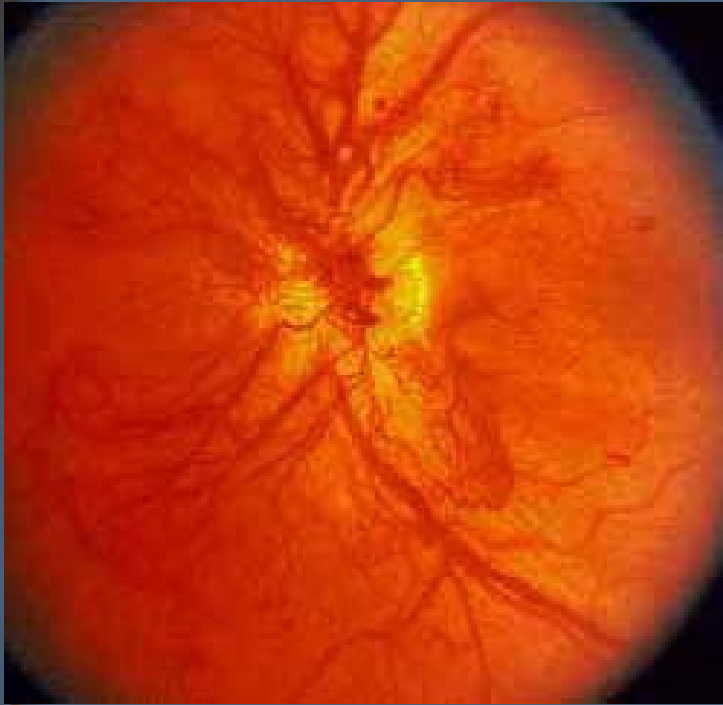


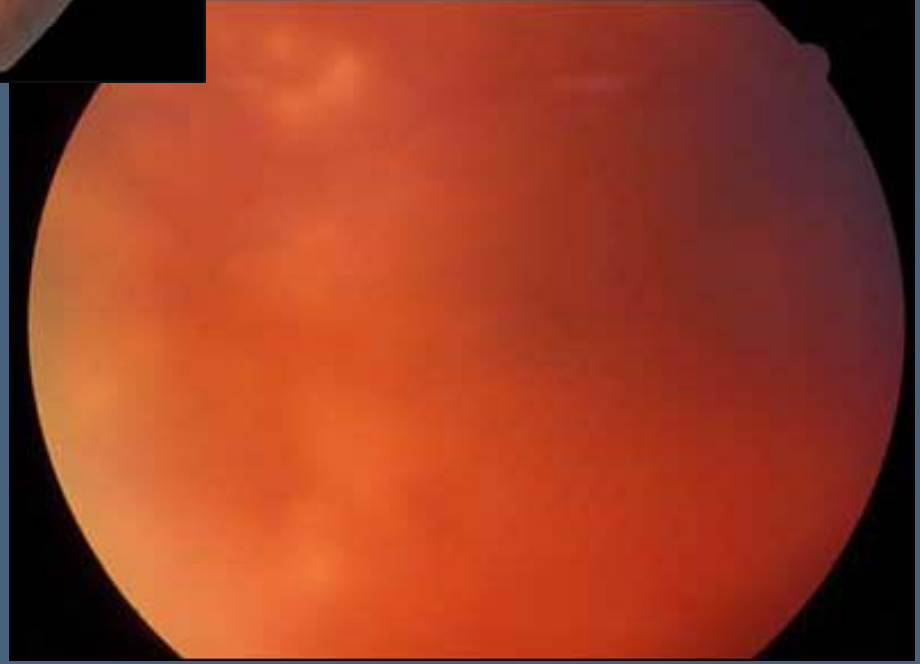


PDR



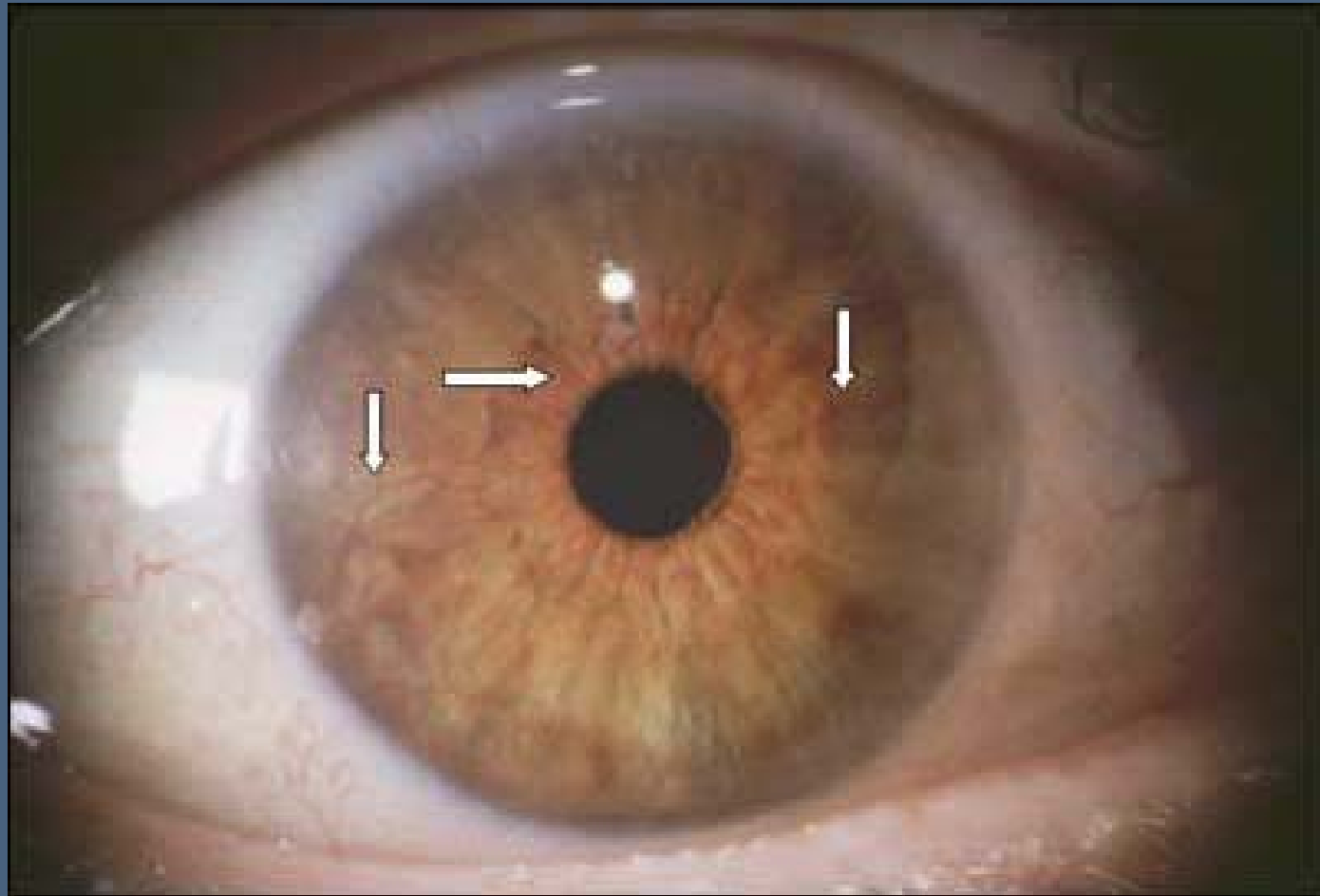








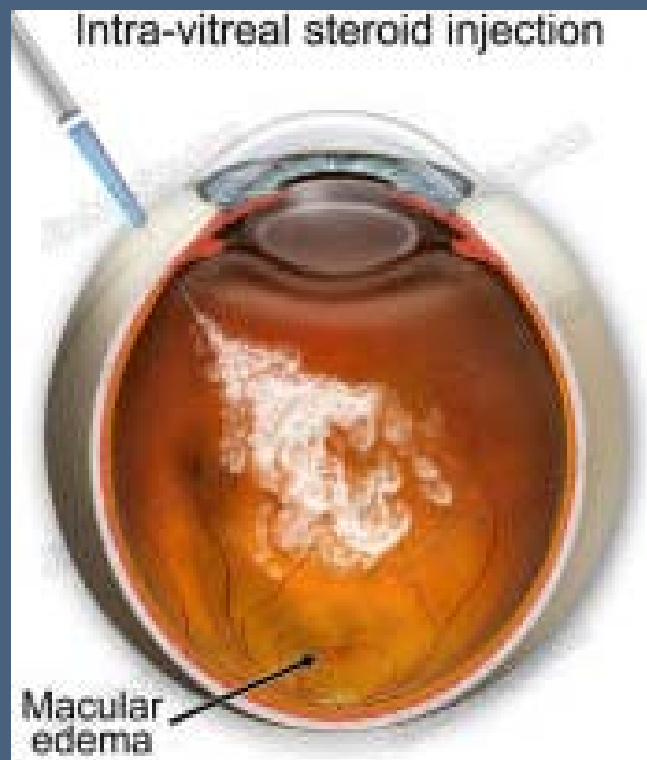
NVI



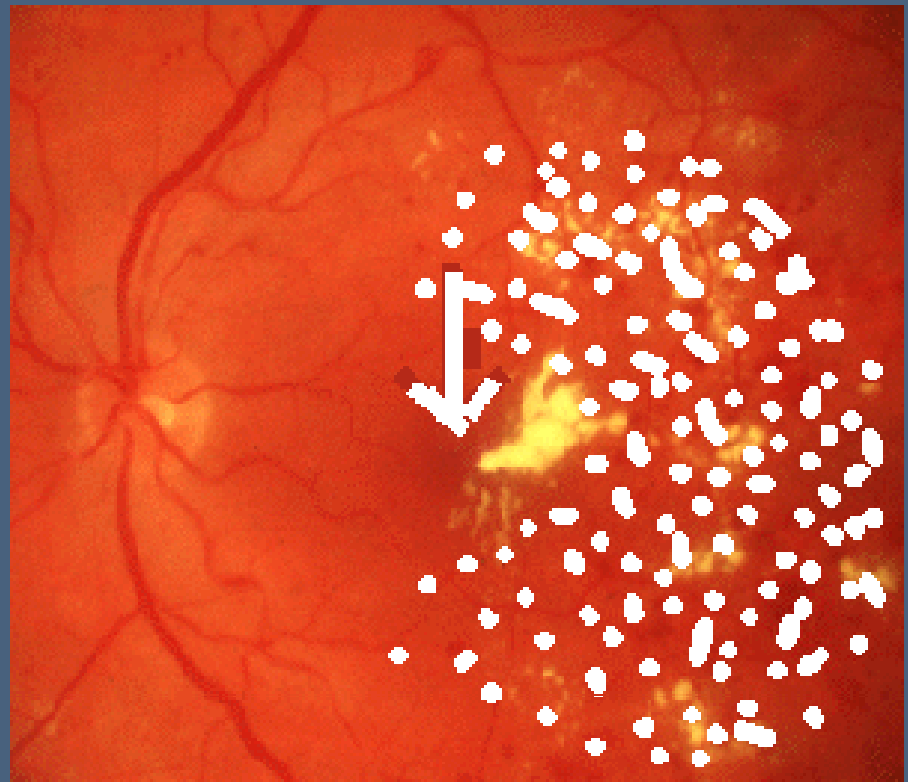
Treatment

- Control hyperglycemic status
- Control hypertension, anemia, hyperlipidemia
- F/U : duration????
- Laser photocoagulation
- Surgical intervention

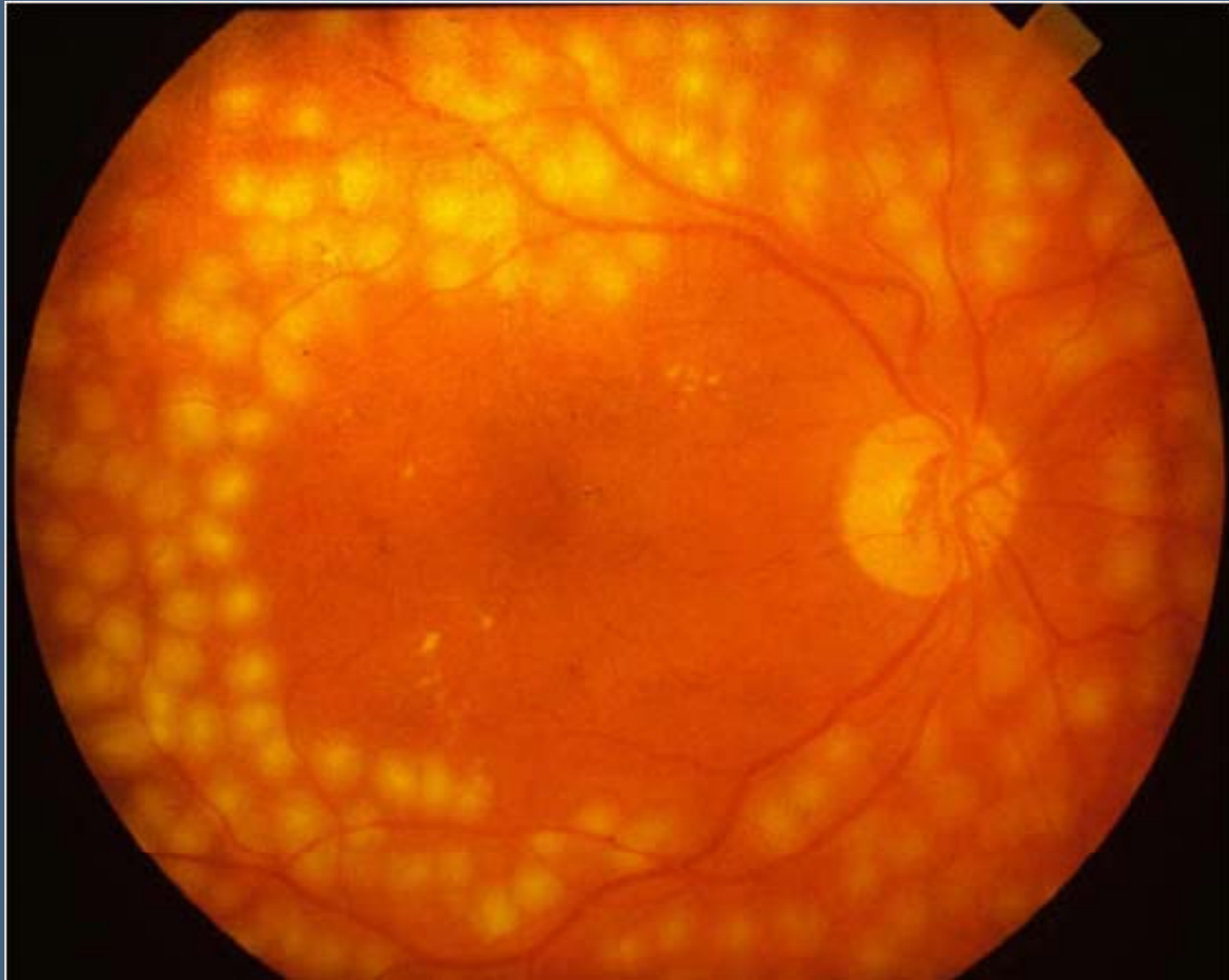
Intravitreal injection



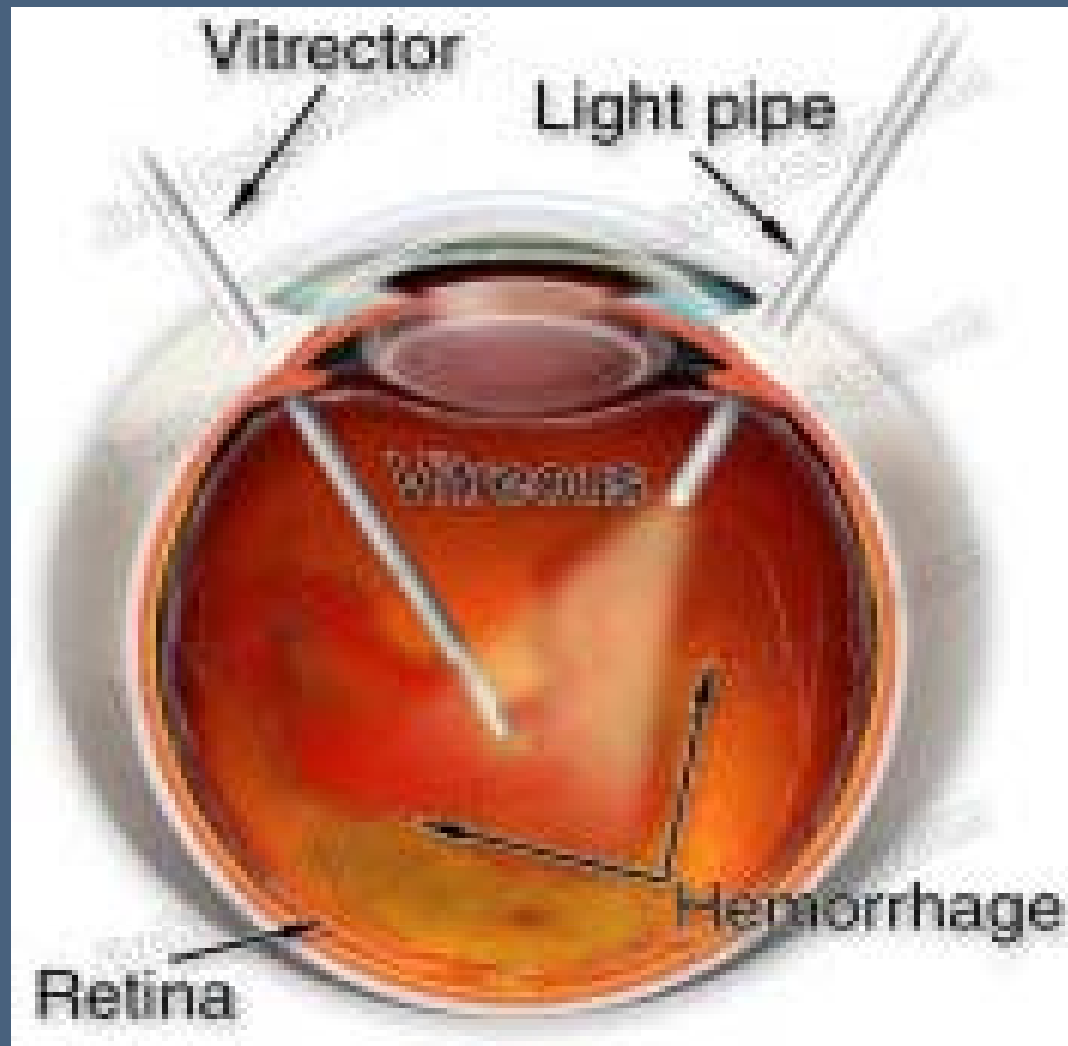
Focal laser photocoagulation



Pan-retinal laser photocoagulation



Pars plana vitrectomy



Refractive Errors & Presbyopia

Formation of Vision

- Light from object
- Refraction by optical element
- Image formation on retina
- Conversion into neural signals
- Perception by the brain

The Evolution of the Refractive State

- Hyperopia in newborn (2D)
- Gradual shift toward emmetropia
- Emmetropization
- More genetic than environmental
- Affected by ocular and systemic diseases, ocular surgery, etc

The **Focusing** element

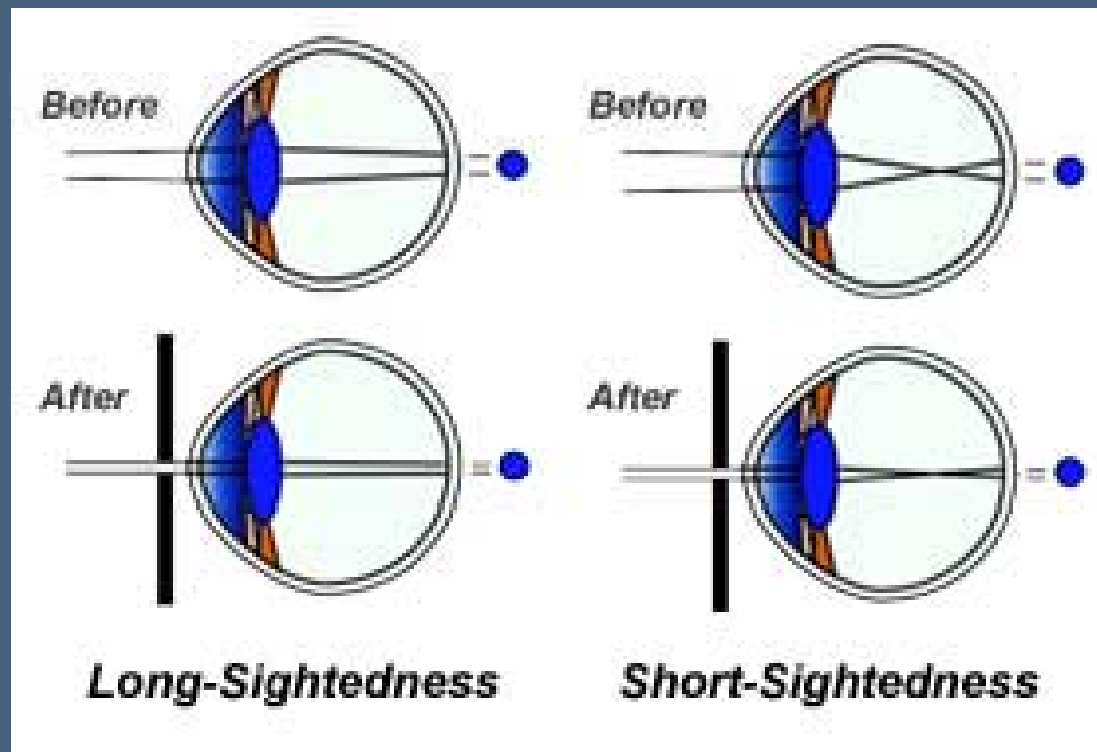
- Cornea
 - most powerful **+43 D**
- Crystalline lens : +20 D
- Accommodation

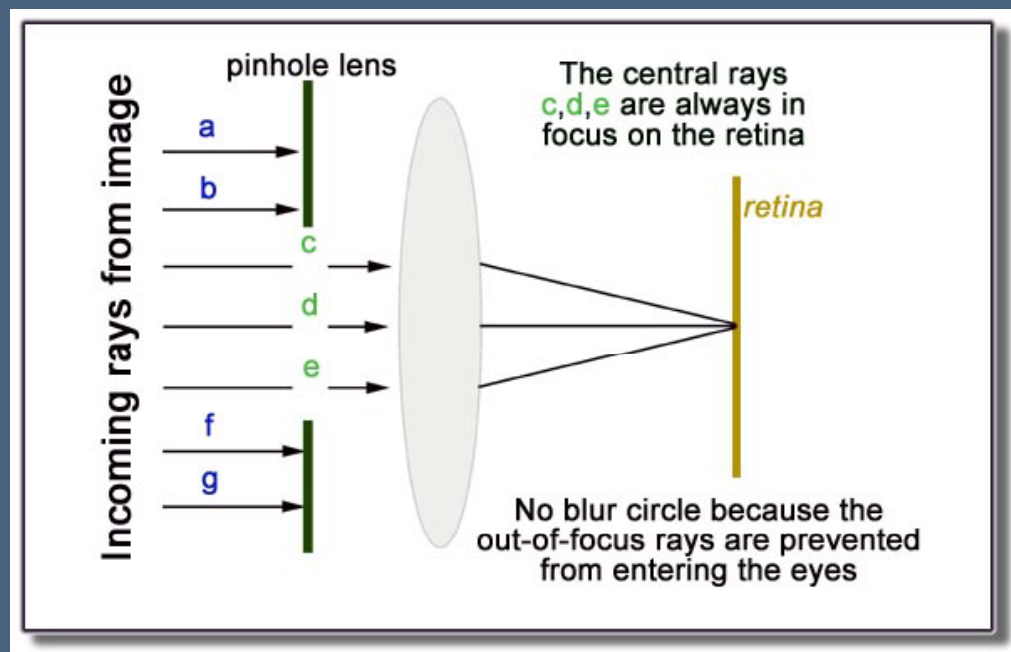
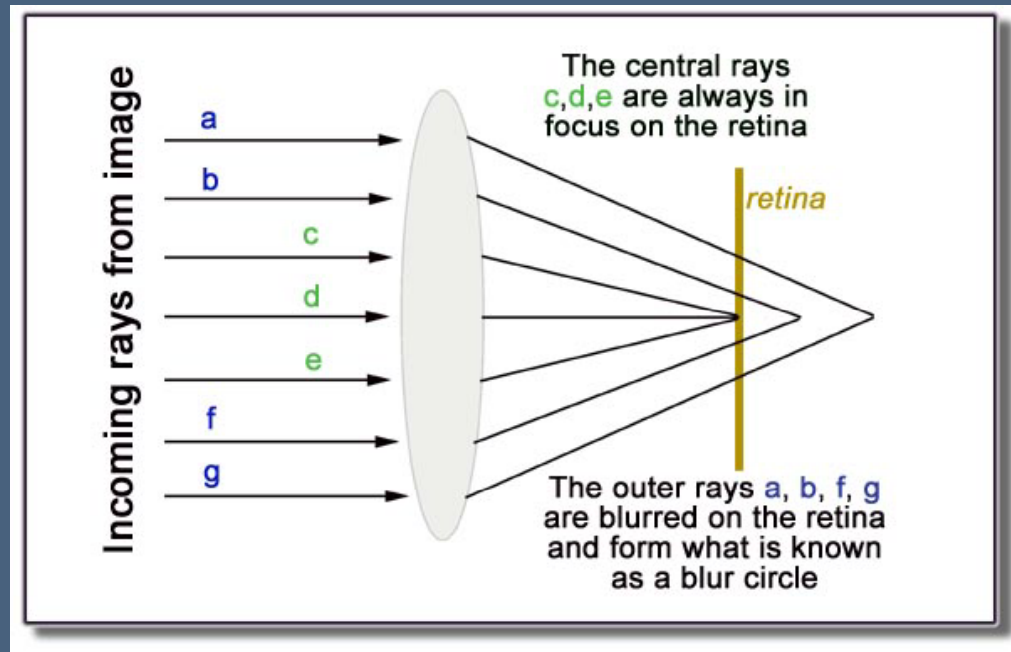
Refractive Errors (Ametropia)

= Defocusing

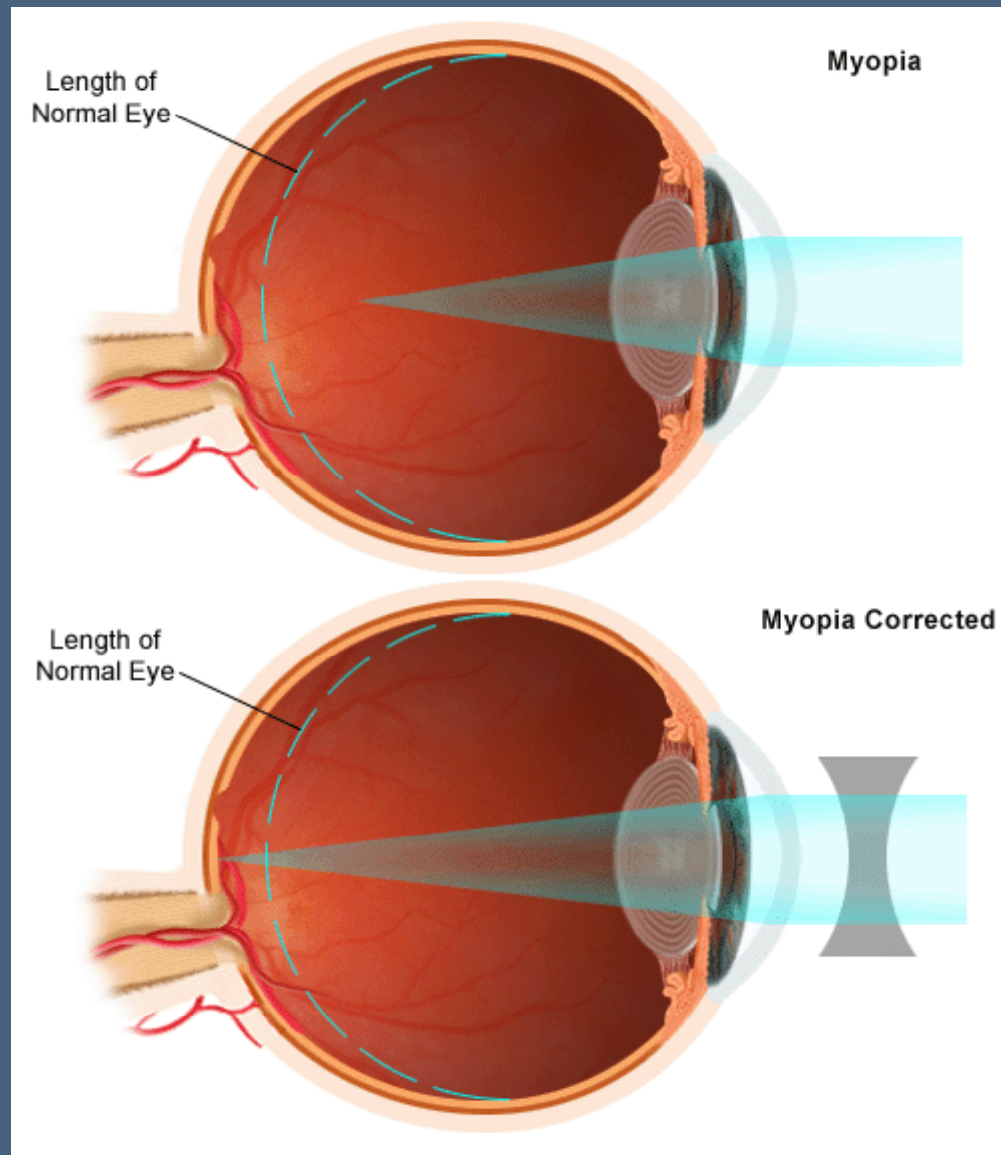


Defocusing and Aperture

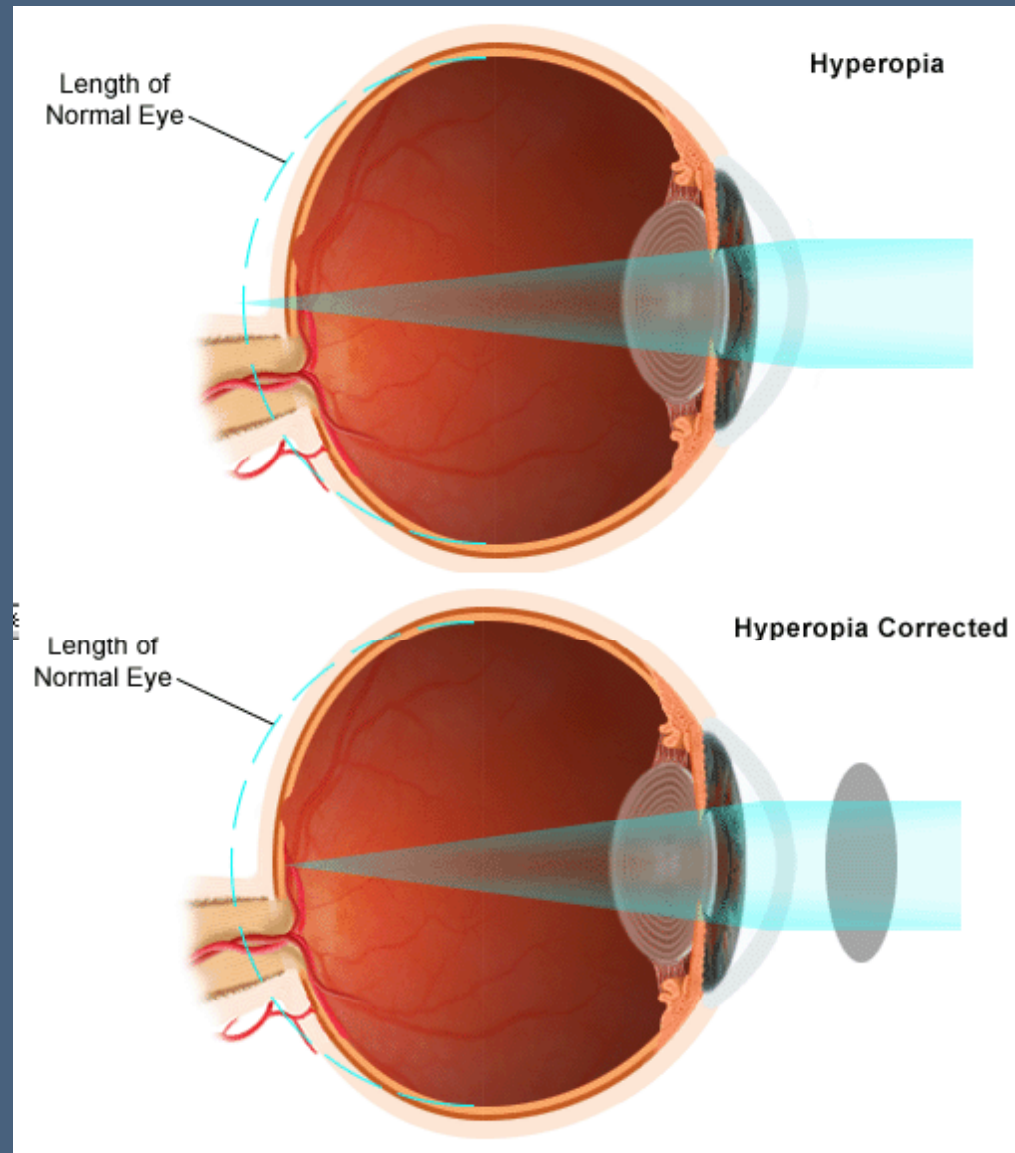




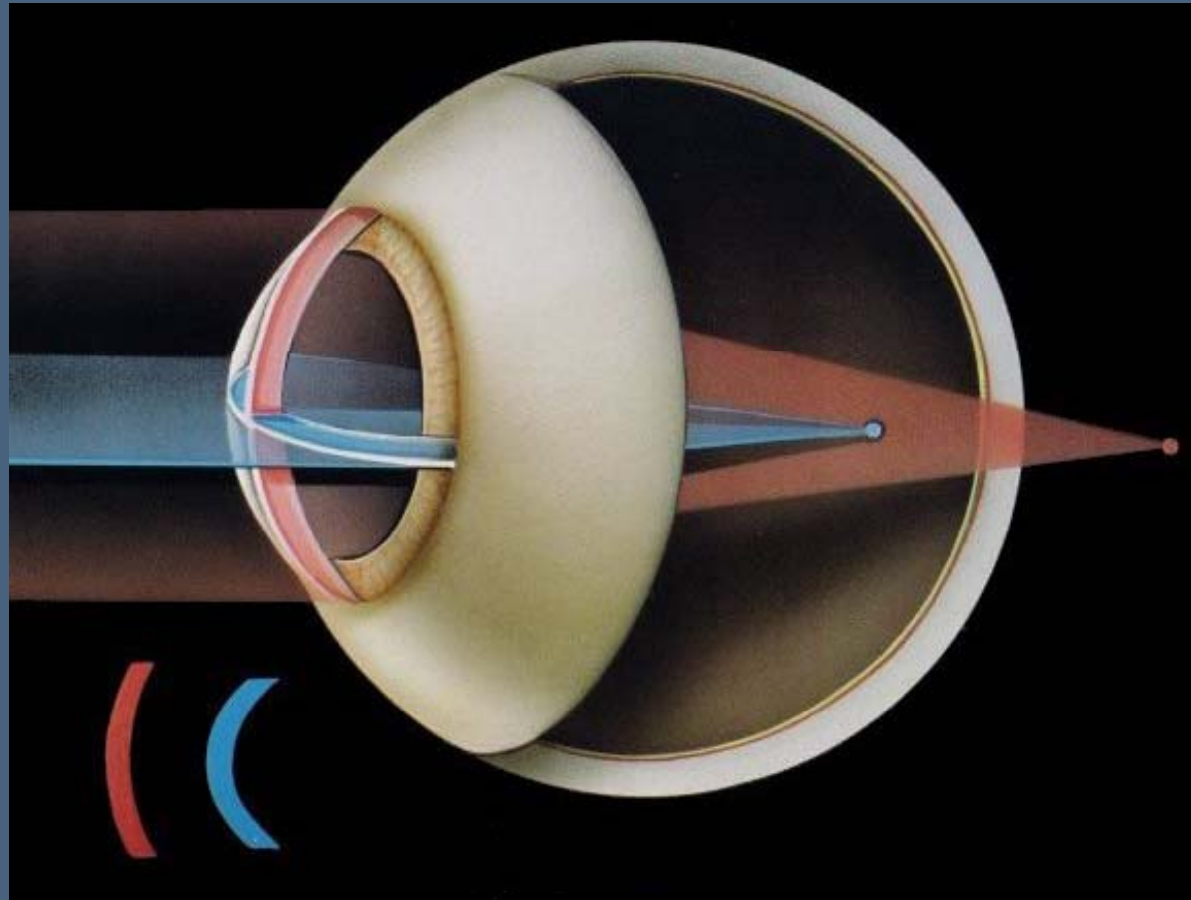
Myopia, Near Sightedness



Hyperopia, Far Sightedness



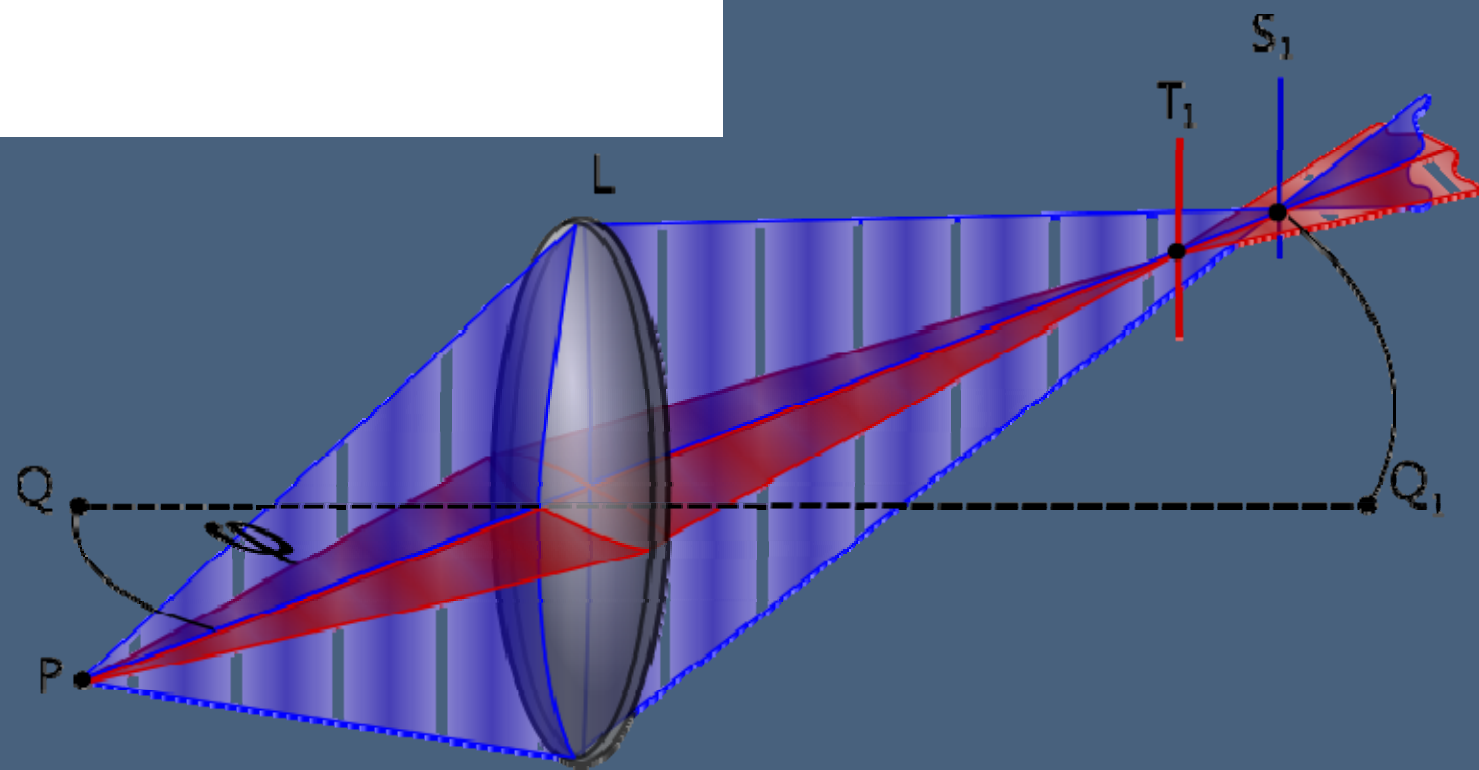
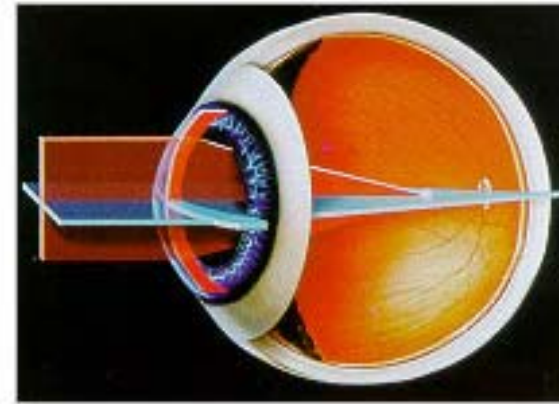
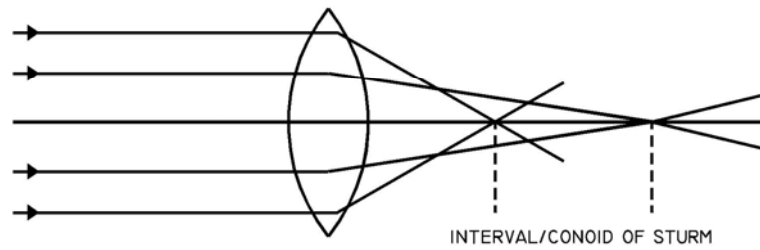
Astigmatism



Astigmatism



FIGURE 31

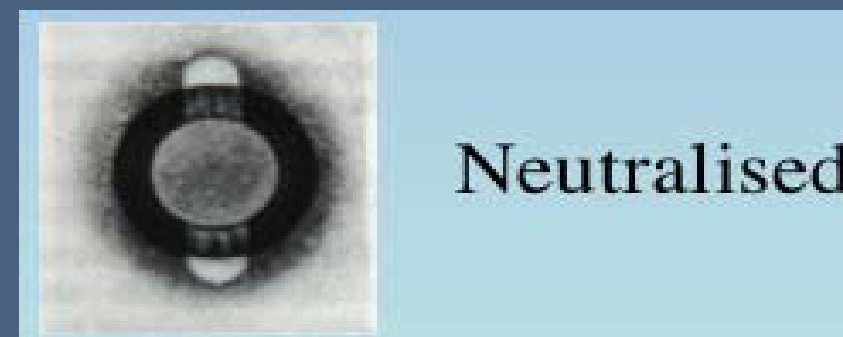
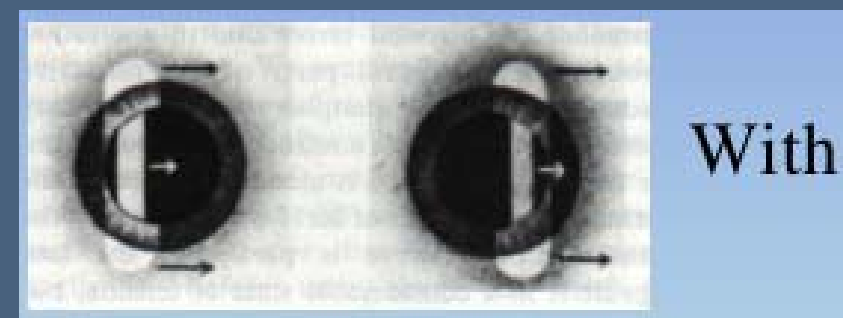
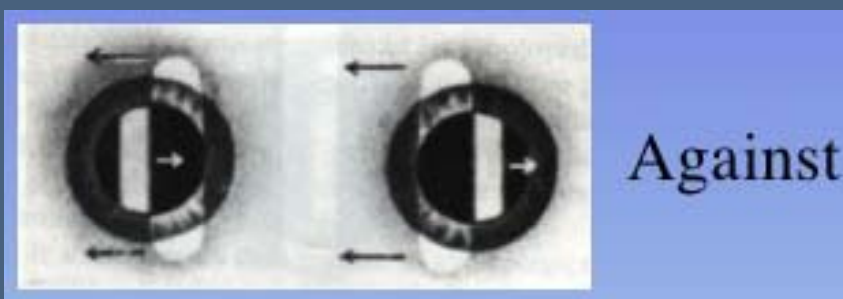


Presbyopia, Old Eyes

- Physiologic decrease of accommodation
- Not a true refractive error
- Over 40 years of age
- Need plus lenses to see up close

Refraction

- Measuring refractive errors
- Types : manifest., cycloplegic
- Methods
 1. Objective : retinoscopy, autorefractor
 2. Subjective : refinement



Correcting Refractive Errors

- Spectacles
- Contact lenses
- Surgery (refractive surgery)

● *
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