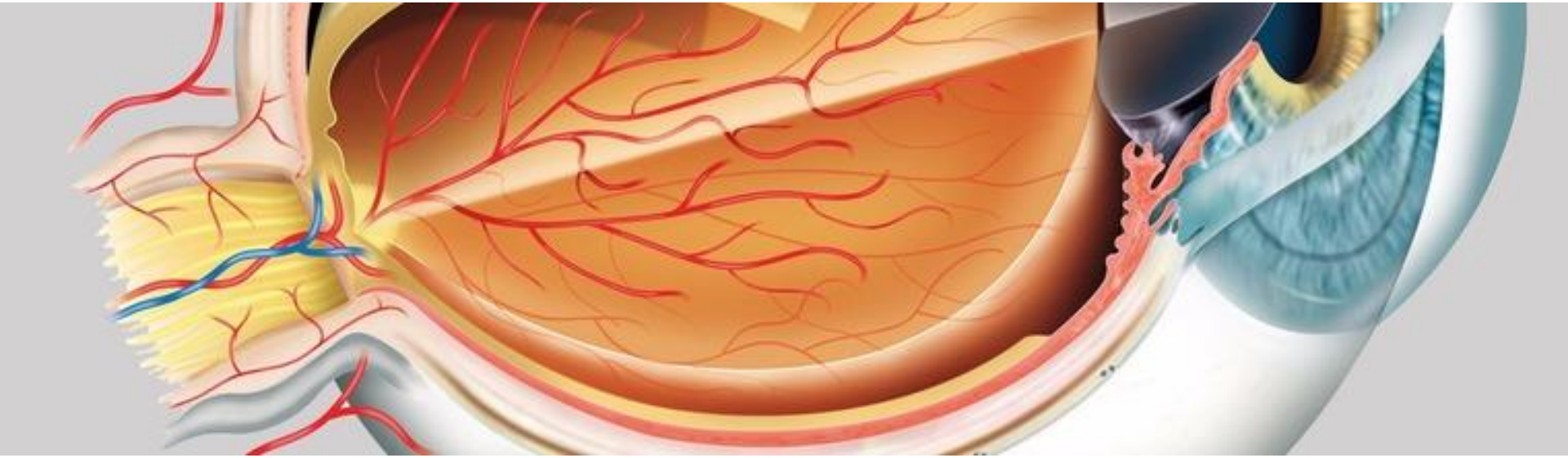
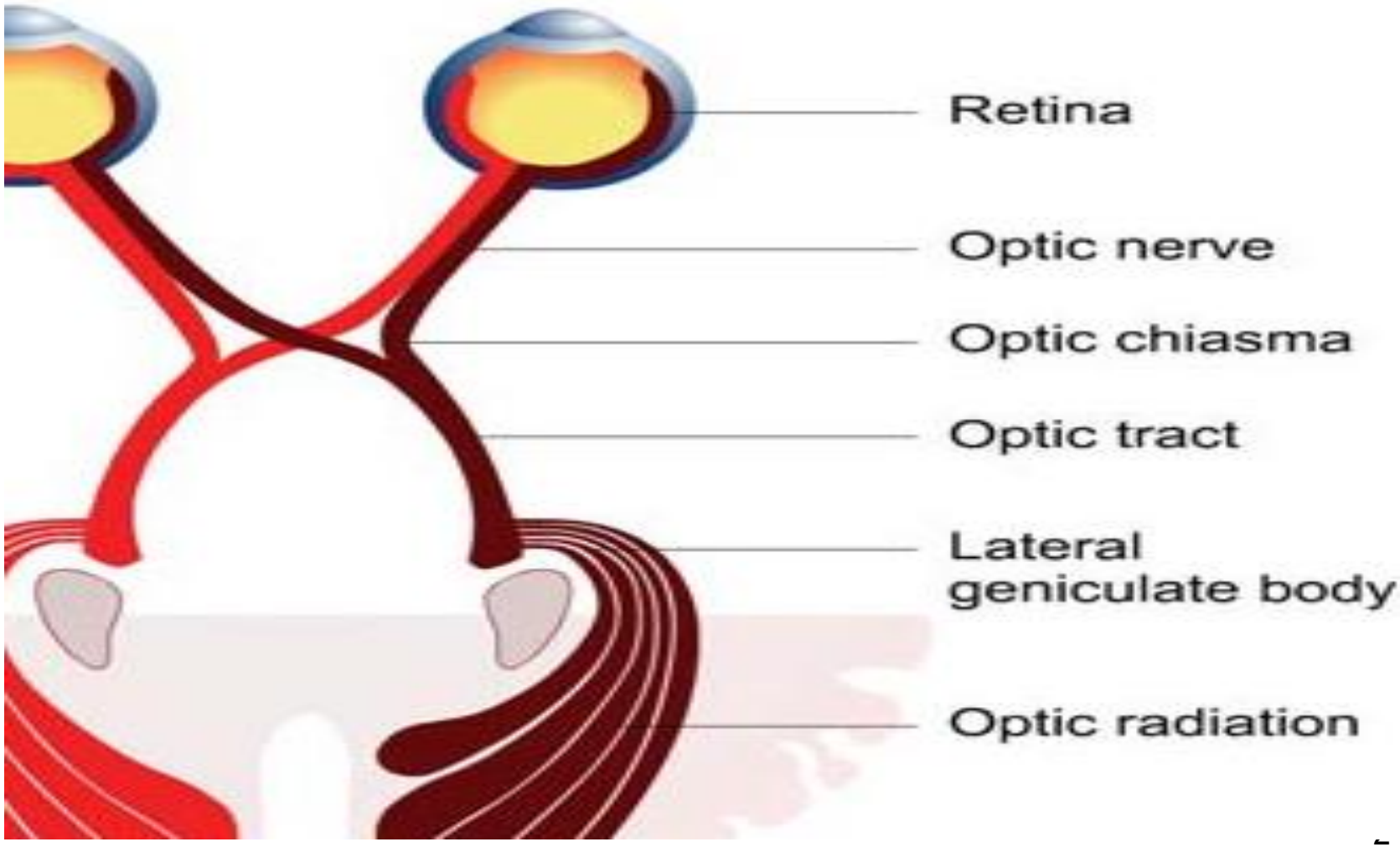
# *Optic Nerve Head (Optic Disc)*



Dr Engy Mohamed Mostafa

MD, PhD

# Anatomy


Retina
Optic nerve
Optic chiasma
Optic tract
Lateral geniculate body
Optic radiation

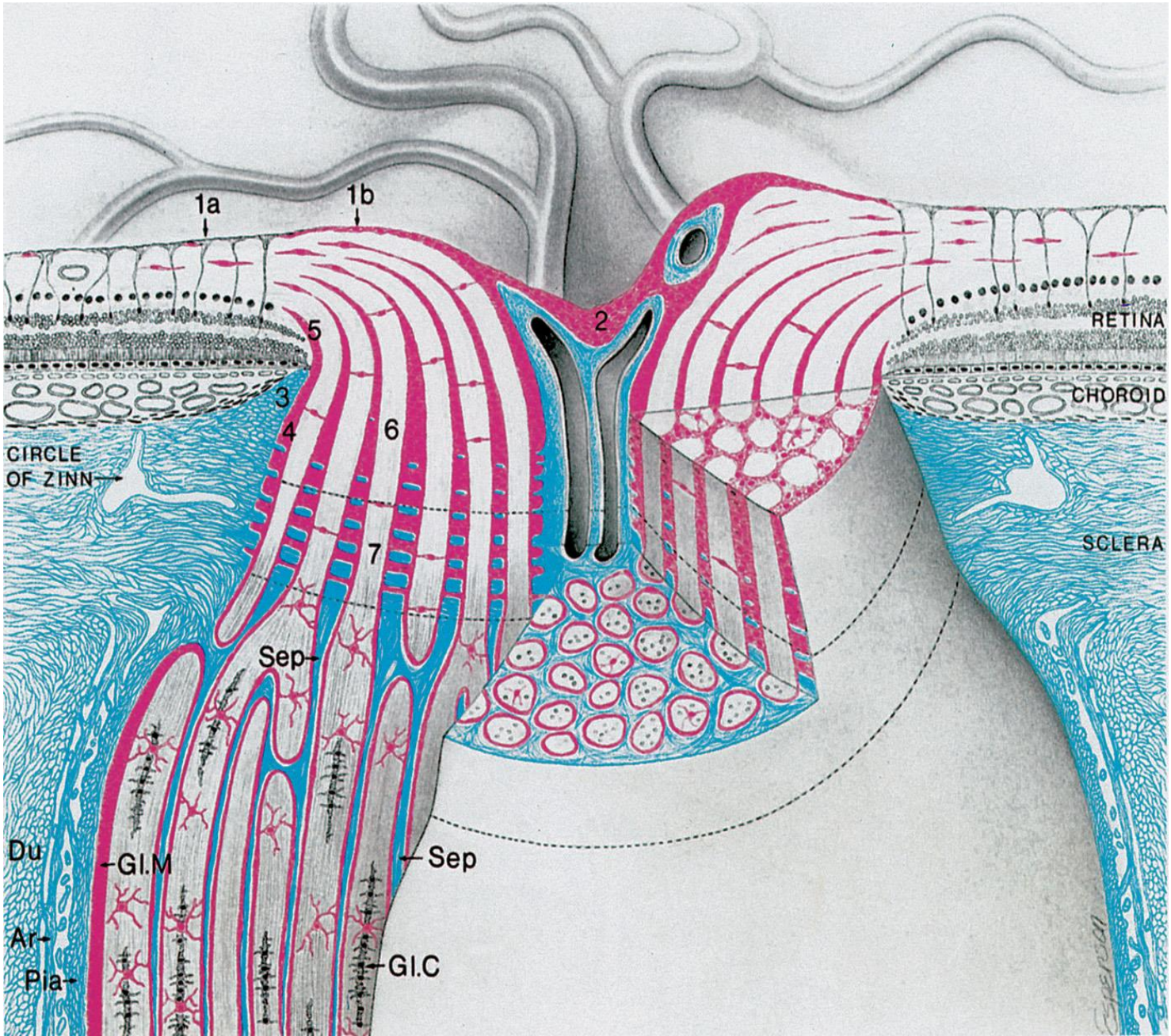
# Parts of optic nerve

The optic nerve is about 47-50 mm in length, and can be divided into 4 parts:

- intraocular (1 mm)
- intraorbital (30 mm)
- intracanalicular (6-9 mm)
- intracranial (10 mm).

# Intraocular part

Passes through sclera (converting it into a sieve-like structure—the lamina cribrosa), choroid and finally appears inside the eye as optic disc.


1a
1b
2
5
3
4
6
7
RETINA
CHOROID
SCLERA
CIRCLE
OF ZINN
Sep
Sep
Du
Gl.M
Ar
Pia
Gl.C

# Intraorbital part

extends from back of the eyeball to the optic foramina. This part is slightly sinuous to give play for the eye movements.

Posteriorly, near the optic foramina, it is closely surrounded by the annulus of Zinn and the origin of the four rectus muscles.

Some fibres of superior rectus muscle are adherent to its sheath here, and accounts for the painful ocular movements seen in retrobulbar neuritis.

# Intracanalicular part

- Closely related to the ophthalmic artery which lies inferolateral to it and crosses obliquely over it, as it enters the orbit, to lie on its medial side.
- Sphenoid and posterior ethmoidal sinuses lie medial to it and are separated by a thin bony lamina. This relation accounts for retrobulbar neuritis following infection of the sinuses.

# Intracranial part

lies above the cavernous sinus and converges with its fellow (over the diaphragma sellae) to form the optic chiasma.

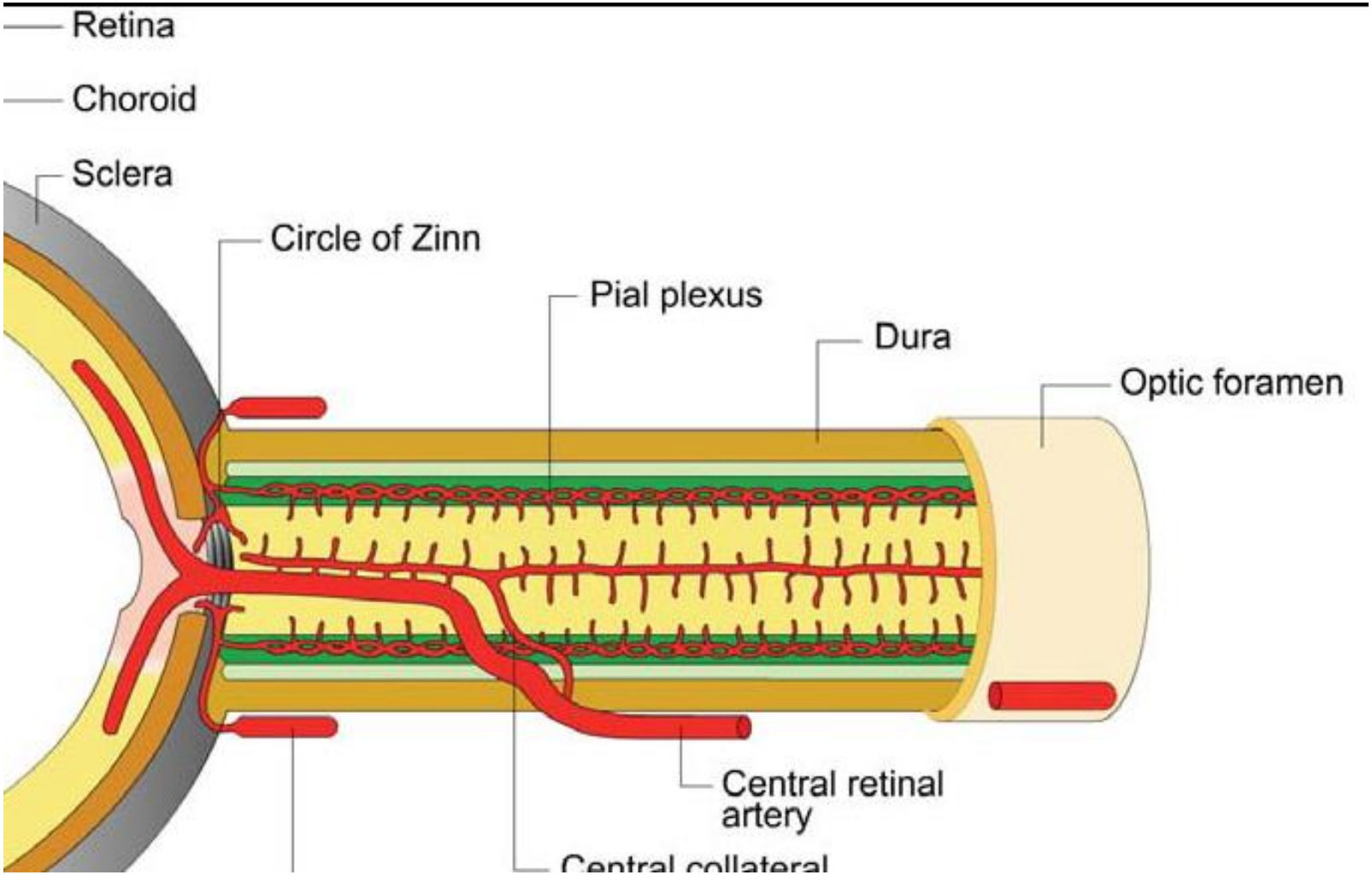
# *Blood supply of the optic nerve head*

The *surface layer of the optic disc is supplied by* capillaries derived from the retinal arterioles.

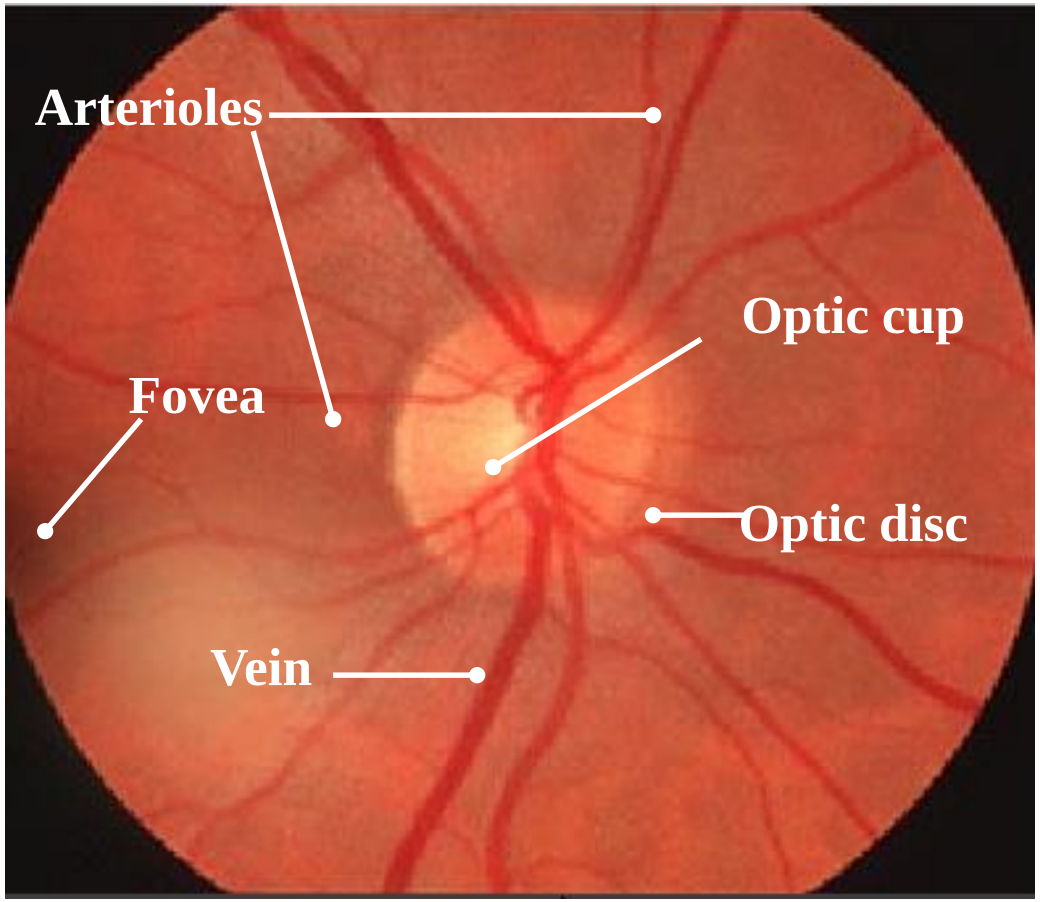
*The prelaminar region is mainly supplied by* centripetal branches of the peripapillary choroid with some contribution from the vessels of lamina cribrosa.

The *lamina cribrosa is supplied by branches* from the posterior ciliary arteries and arterial circle of Zinn.

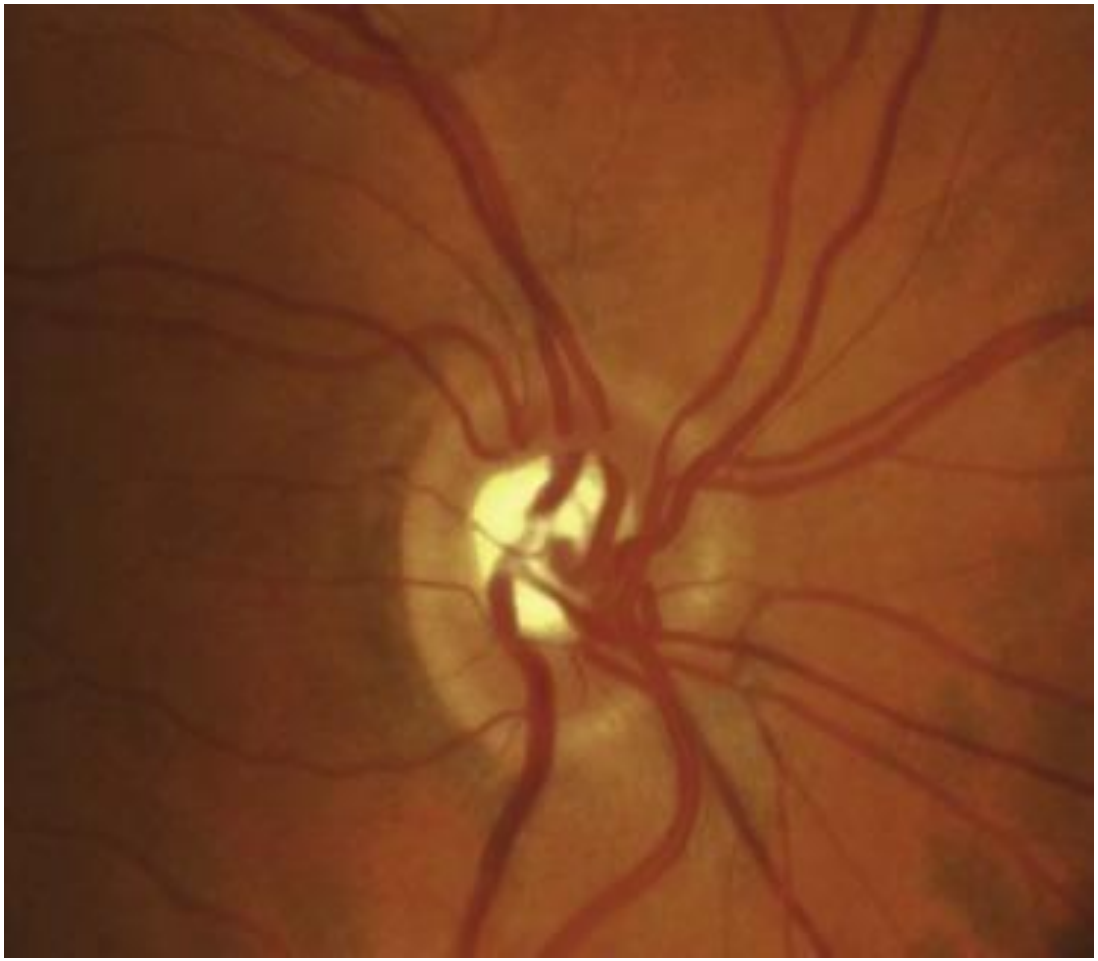
The *retrolaminar part of the optic nerve is* supplied by centrifugal branches from central retinal artery and centripetal branches from pial plexus formed by branches from the choroidal arteries, circle of Zinn, central retinal artery and ophthalmic artery.


Retina
Choroid
Sclera
Circle of Zinn
Pial plexus
Dura
Optic foramen
Central retinal artery
Central collateral

# Normal Ocular Fundus

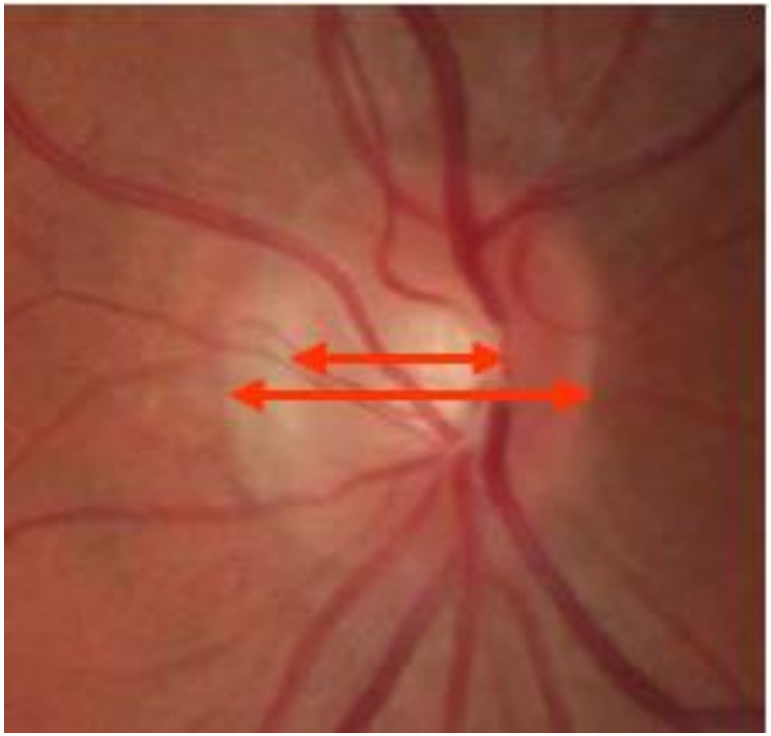


# Normal optic disc



- ✓Colour
- ✓Edges
- ✓Optic cup
- ✓Nasal blurring,
- ✓temporal pallor

# Fundoscopic Examination



C/D = 0.6

- Cup to Disk Ratio
  - Diameter of the cupped region of the optic nerve head

    divided by the diameter of the optic nerve head.

- Normal is ~0.3-0.5.
- Abnormal values are higher and are associated with

  glaucoma

# Fundoscopic Examination

- Prerequisites-
  - Good ophthalmoscope
  - A large pupil
  - A still field

- Diminish illumination in the room( to overcome light reflex)
- Instruct the pt to look at a distant point, which is clearly defined( to overcome accomodation and keeping the eye still)
- Rt eye for examining rt fundus, lt eye for left fundus

# Cup: Size, Shape, location in relation to the disc size

Optic Cup= Excavation in the optic nerve head

Stereoscopic evaluation

In normal eyes= Areas of optic disc & Optic cup are corelated

Large optic discs=Large cup

Small optic disc =Small cup or no cup

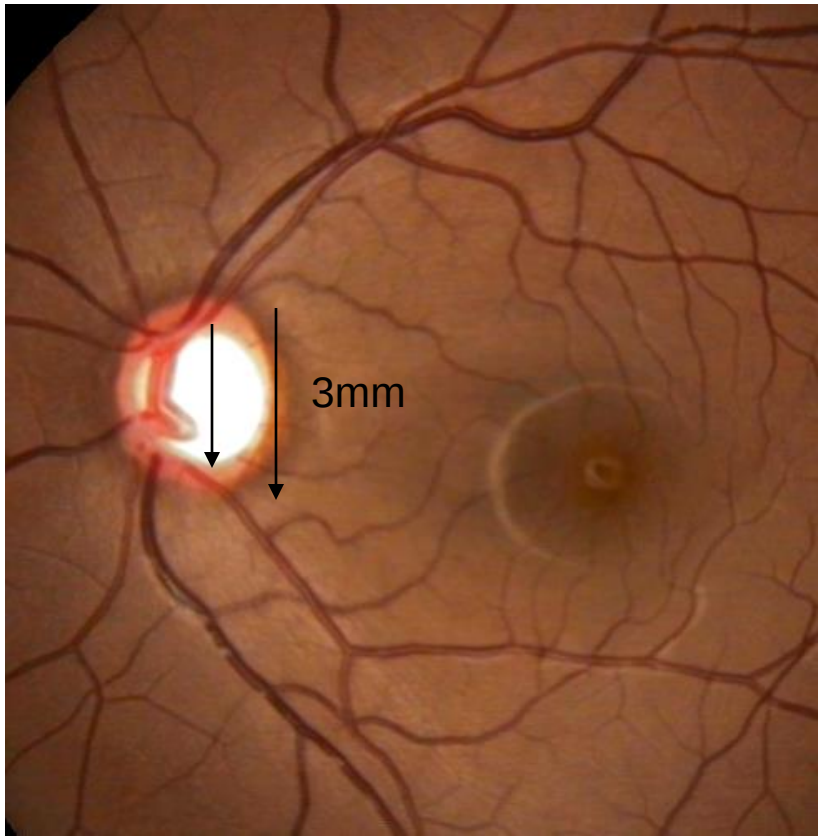
# Optic Disc: Size & Shape

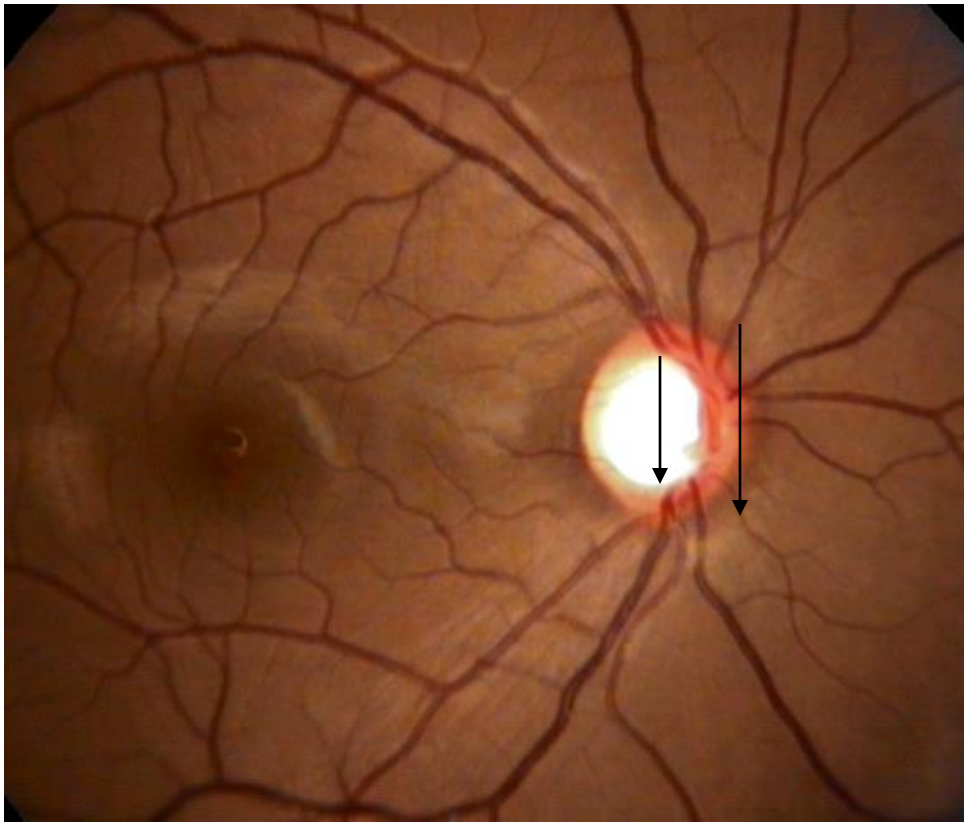
Measurement of Vertical Disc diameter :

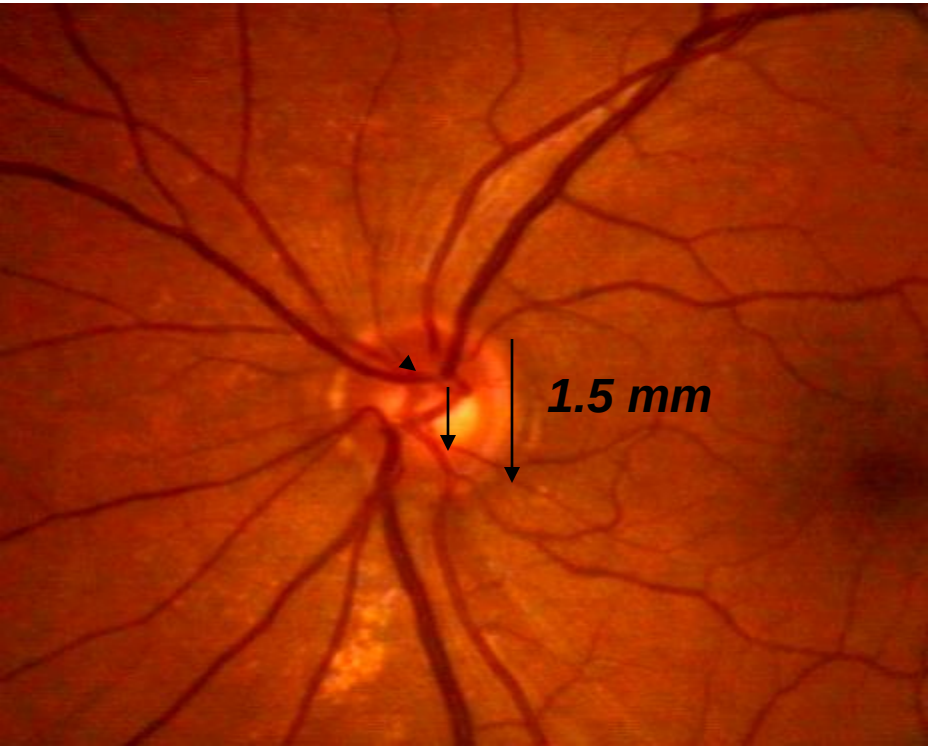
Length of the vertical beam of slit lamp light

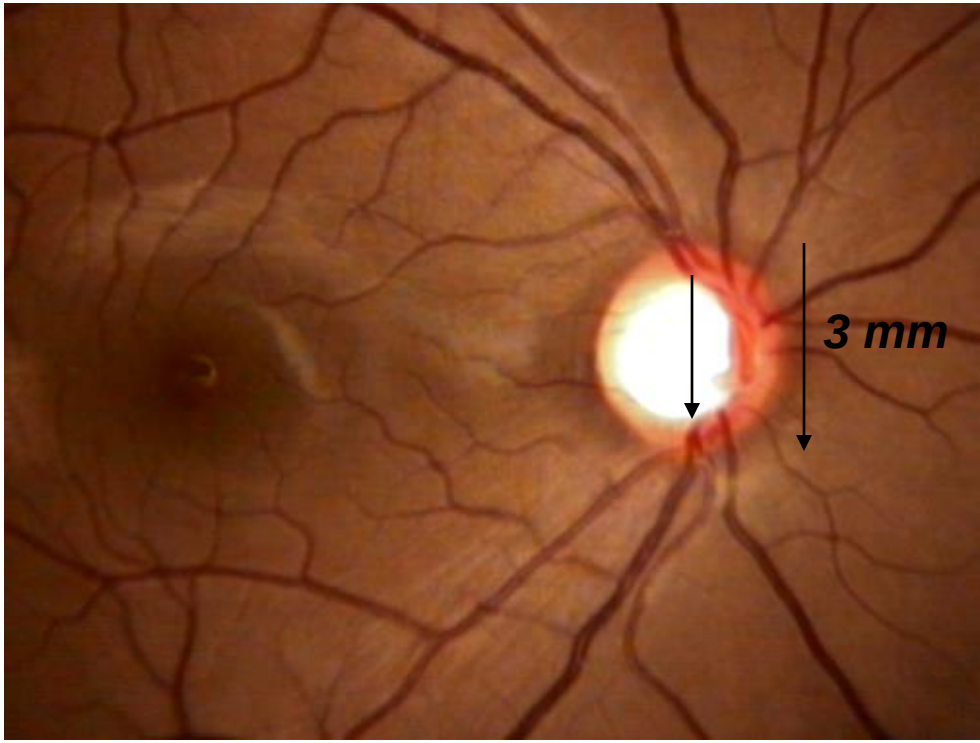
Multiplied by correction factor of the condensing lens

# Large Disc=Large Cup

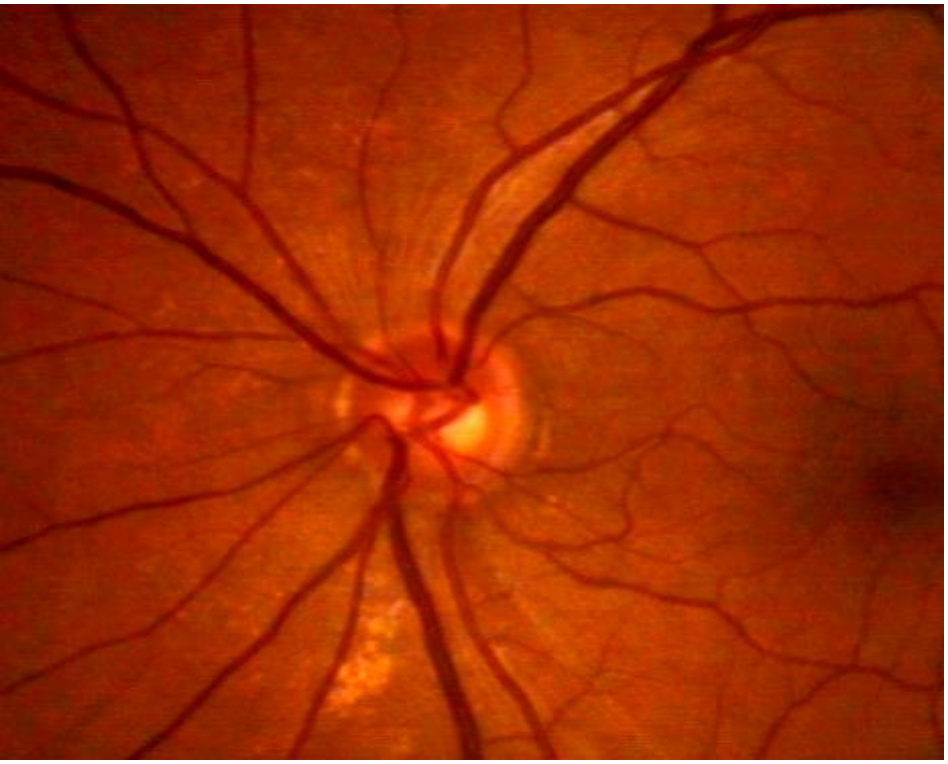


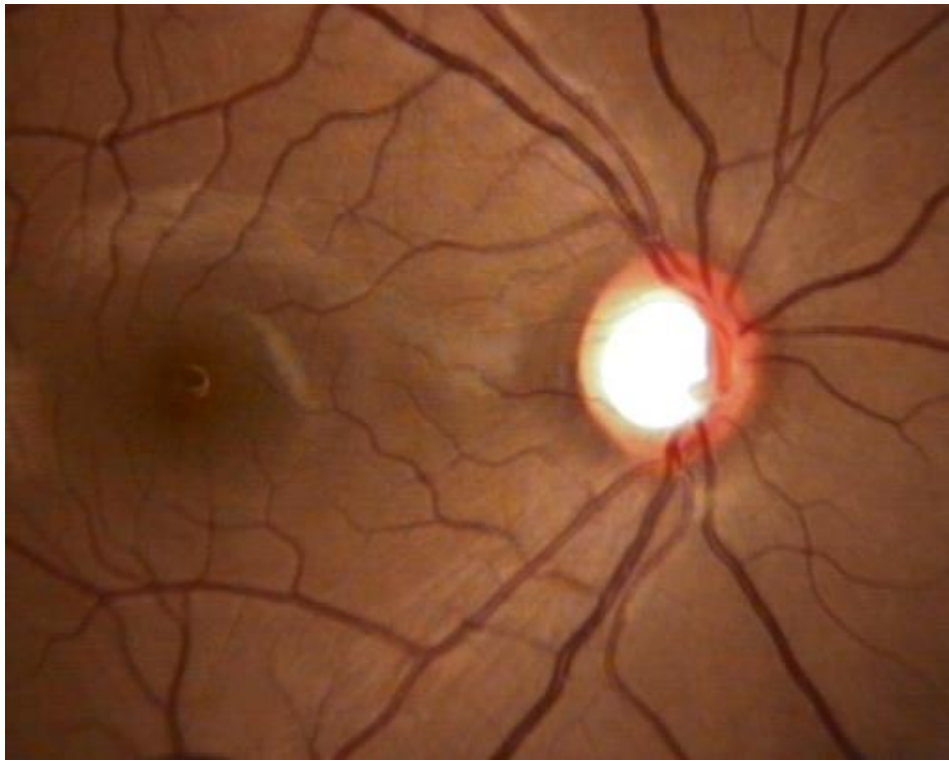



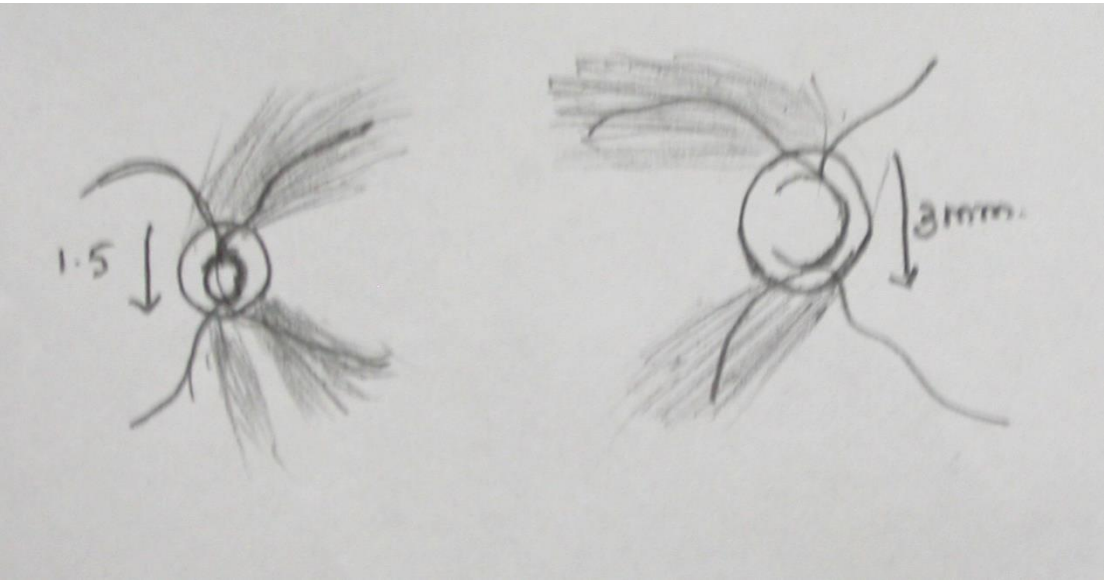

1.5 mm

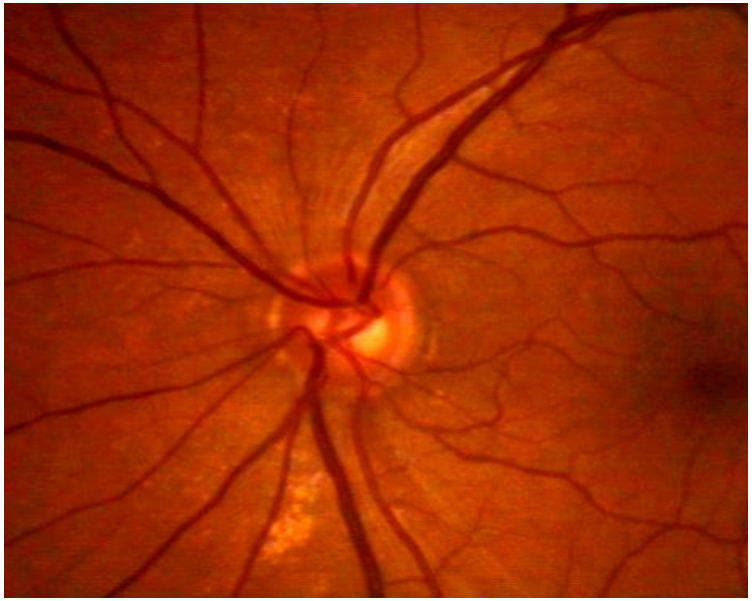

3 mm

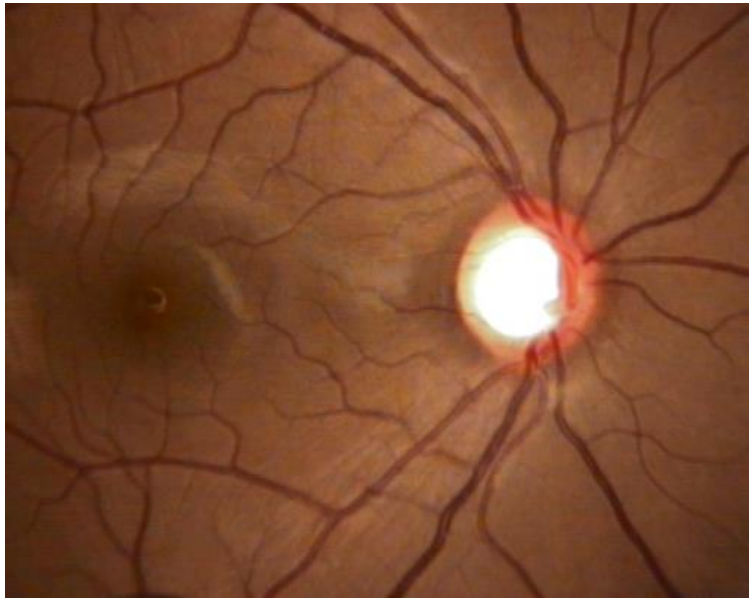
Early & moderate glaucomatous damage in small disc may be missed because of the erroneously low cup disc ratios






1·5
3mm.





# Vertical Cup Disc Ratio

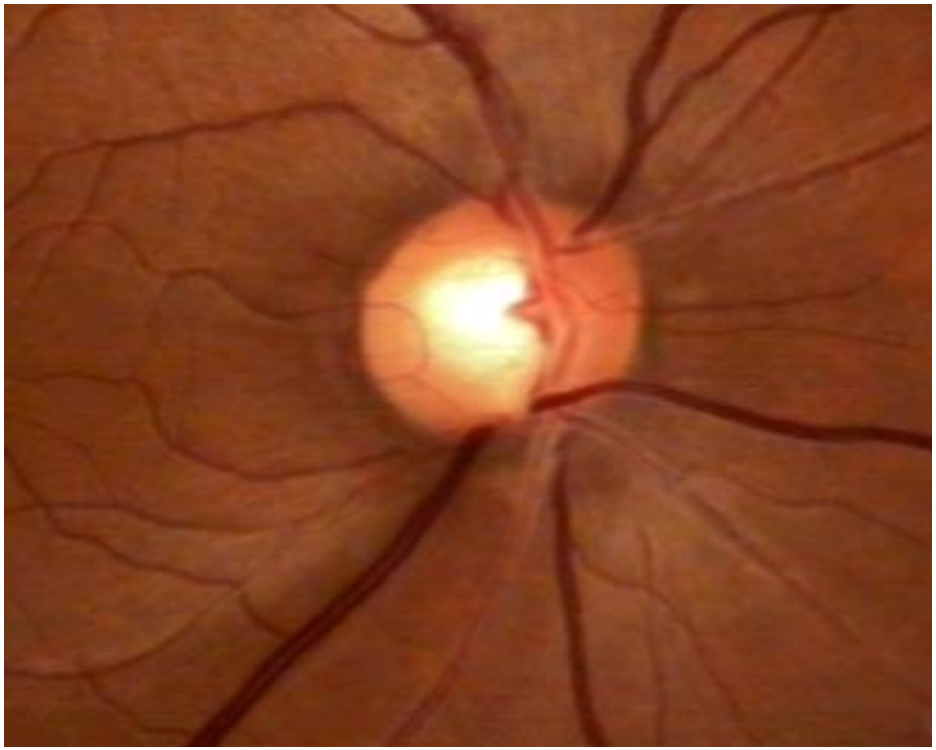
Vertically oval optic disc

Horizontally oval optic cup

In normal eyes: Horizontal CD ratio **>** than vertical CD ratio

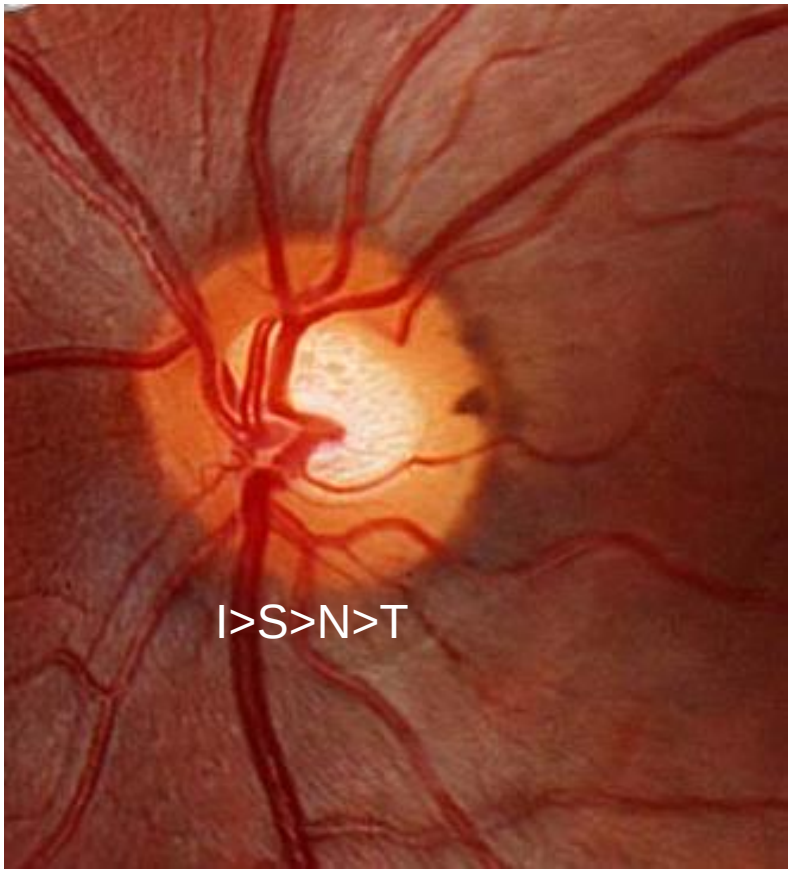
In Glaucomatous eyes: Vertical CD ratio **>** than the horizontal CD ratio

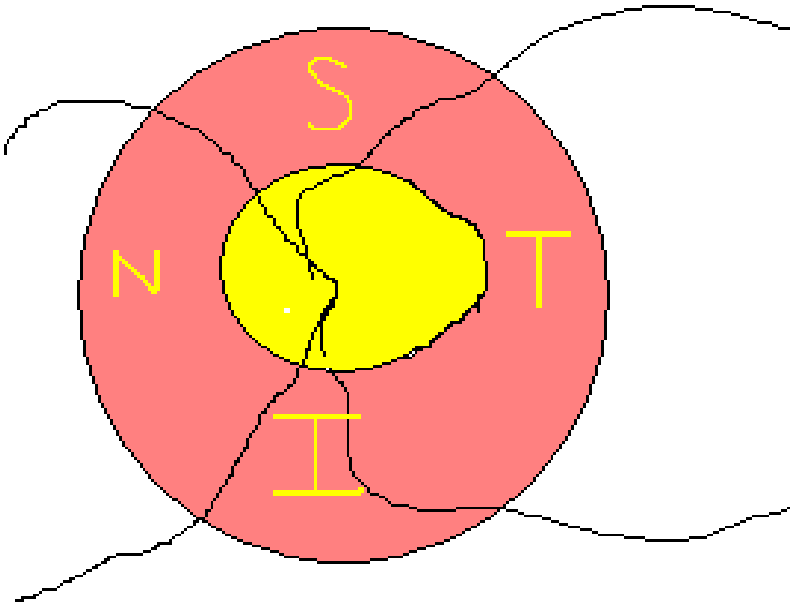
# Vertical CD ratio



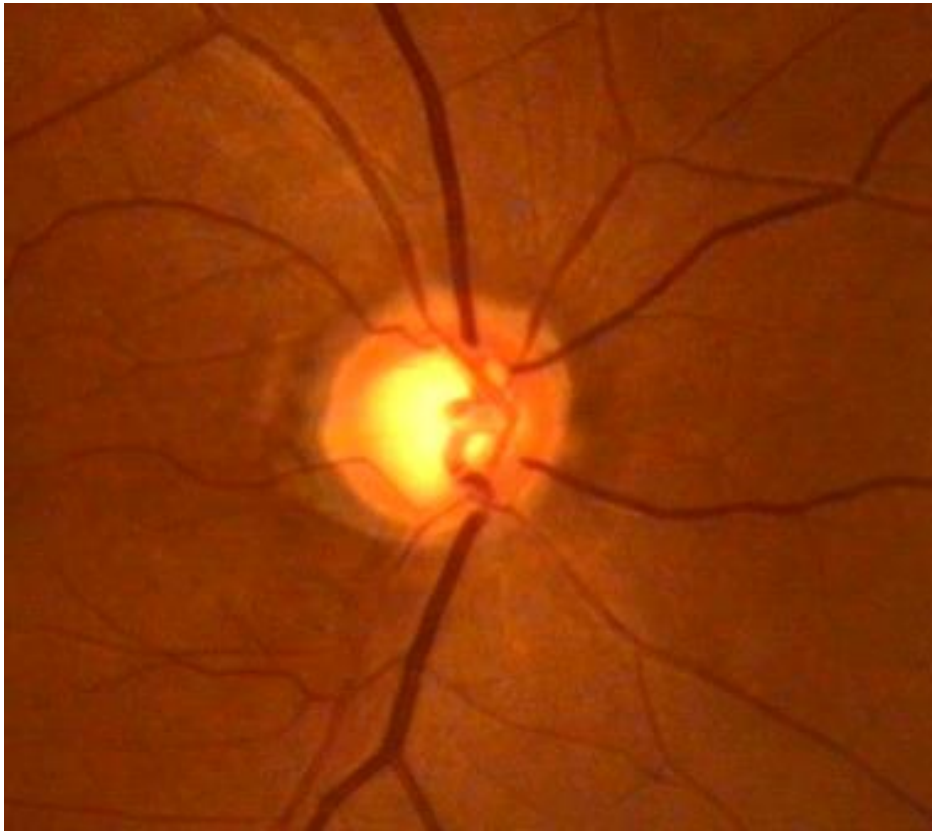
# The Neuroretinal Rim

- Size, Shape, Pallor.
- The ISNT rule:



- Thinning of the NRR
- Pallor of NRR
- Notching:
  - A notch is a localized defect in the Neuroretinal rim on the cup side of the rim



# The Neurretinal rim loss in Glaucoma

Usual sequence of NRR loss in Glaucoma:

Inferotemporal

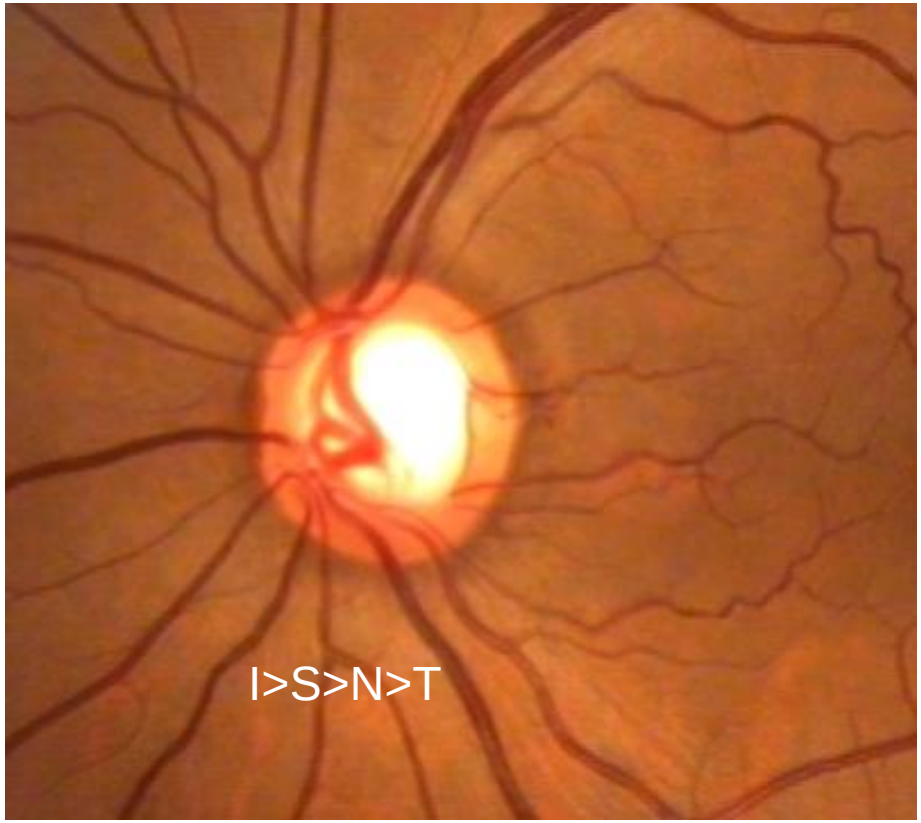
Superotemporal

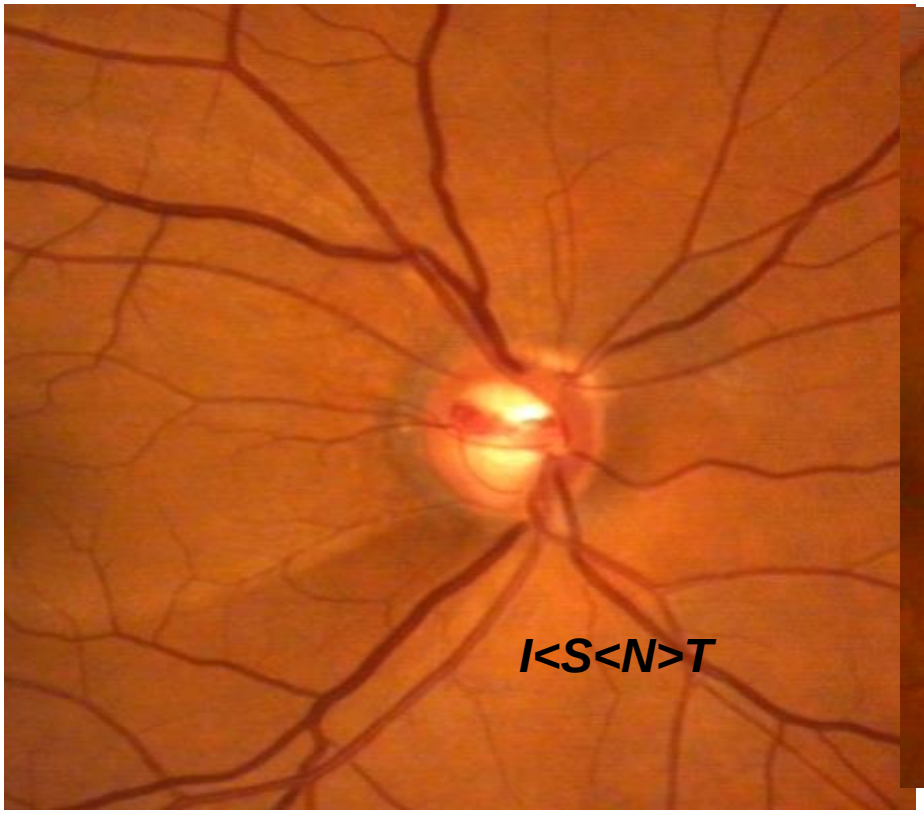
Horizontal temporal

Inferonasal

Superonasal

# NRR , the “ISNT Rule”

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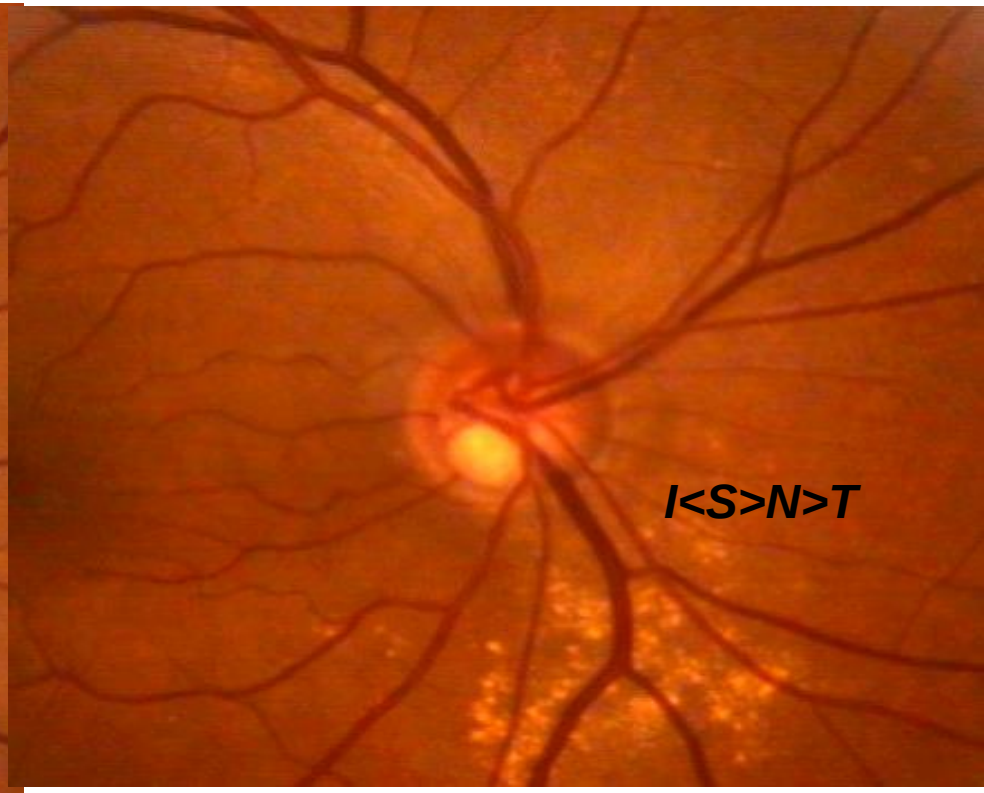
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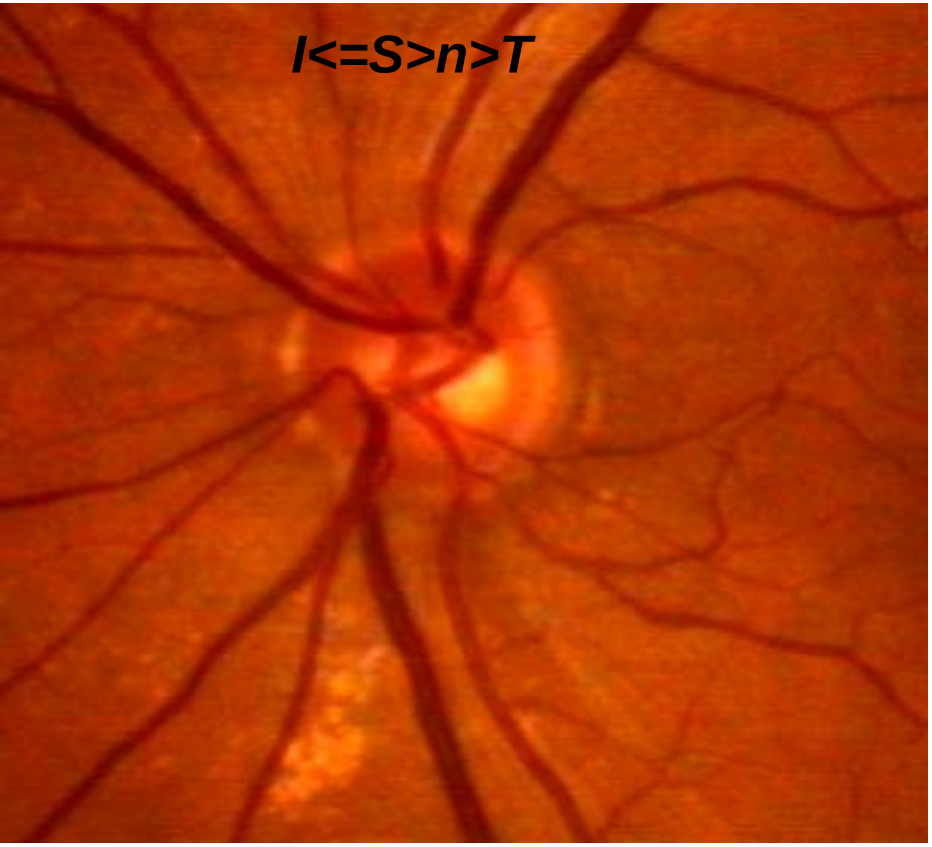
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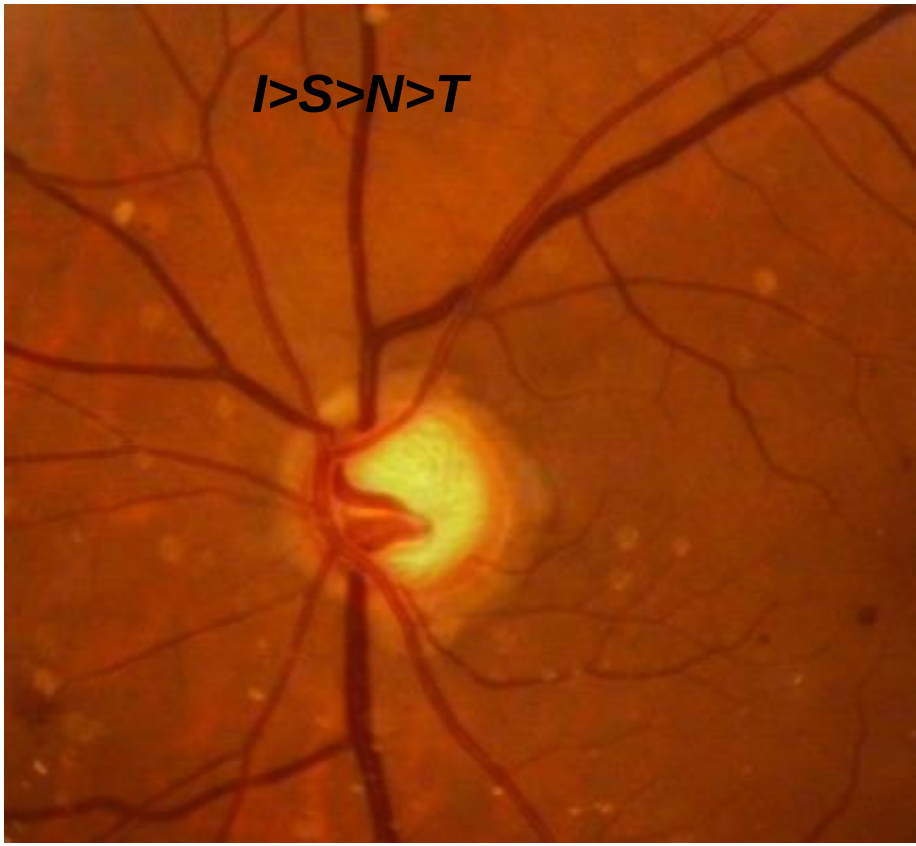
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# Optic disc oedema

# D/D: Causes of 'disc oedema'

- Papilloedema
- Papillitis, neuroretinitis
- Anterior Ischemic Optic Neuropathy
- Optic Nerve glioma, meningioma
- Central Retinal Venous Occlusion

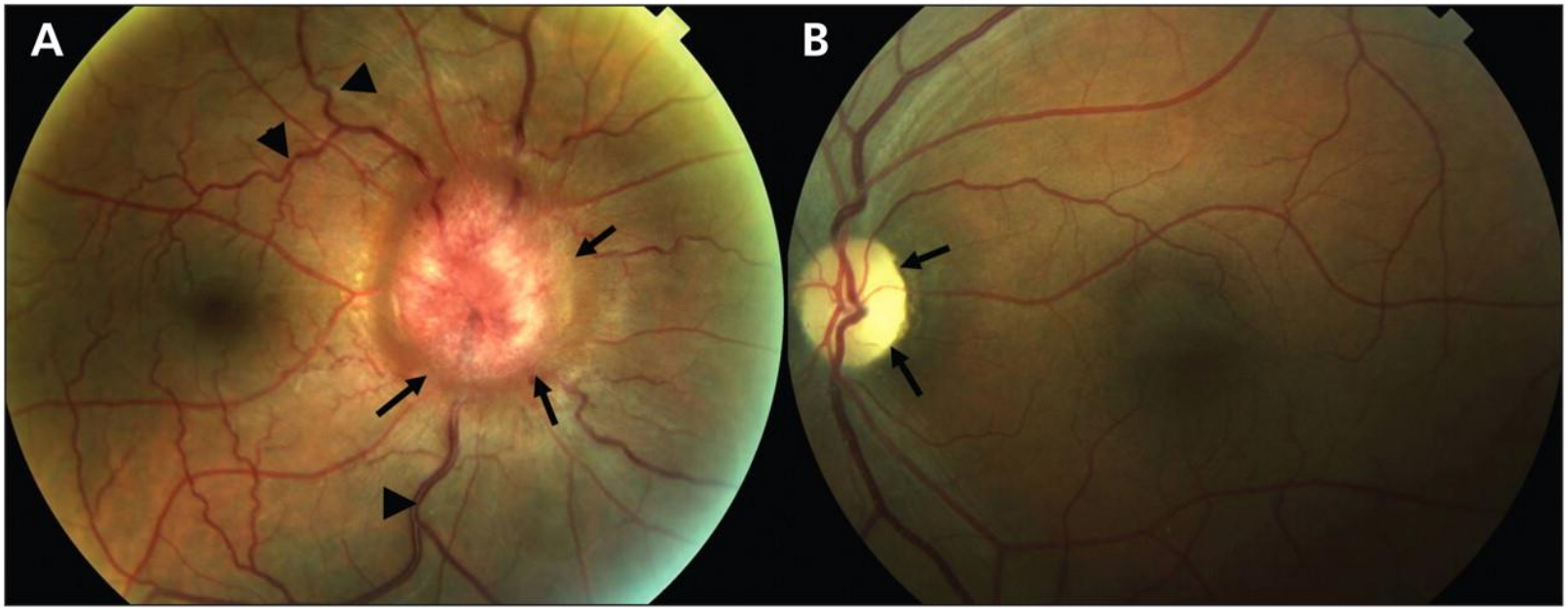
# *Papilloedema*

Bilateral, non-inflammatory passive swelling of optic disc due to raised Intracranial pressure.

Usually bilateral, although it may be asymmetrical

***Foster-Kennedy syndrome :*** contralateral papilloedema with ipsilateral pressure atrophy of optic nerve

Due to - frontal lobe tumour, olfactory meningioma



# Causes of papilledema (Raised ICP)

Primary causes

- Idiopathic pseudotumor cerebri syndrome (idiopathic intracranial hypertension) with papilledema or without papilledema

Secondary causes

- Hydrocephalus
- Shunt failure in patient with hydrocephalus (ventriculomegaly may be absent)
- Mass lesions—tumor, hemorrhage, large infarction, abscess
- Meningitis/encephalitis
- Subarachnoid hemorrhage
- Trauma
- Arteriovenous malformations with high blood flow overloading venous return
- Intracranial or extracranial venous obstruction
- Secondary pseudotumor cerebri syndrome due to certain systemic diseases, drugs, or pregnancy

# General Symptoms

- Headache, made worse by coughing or straining
- Vomiting
- Focal neurological deficit with/without changes in level of consciousness

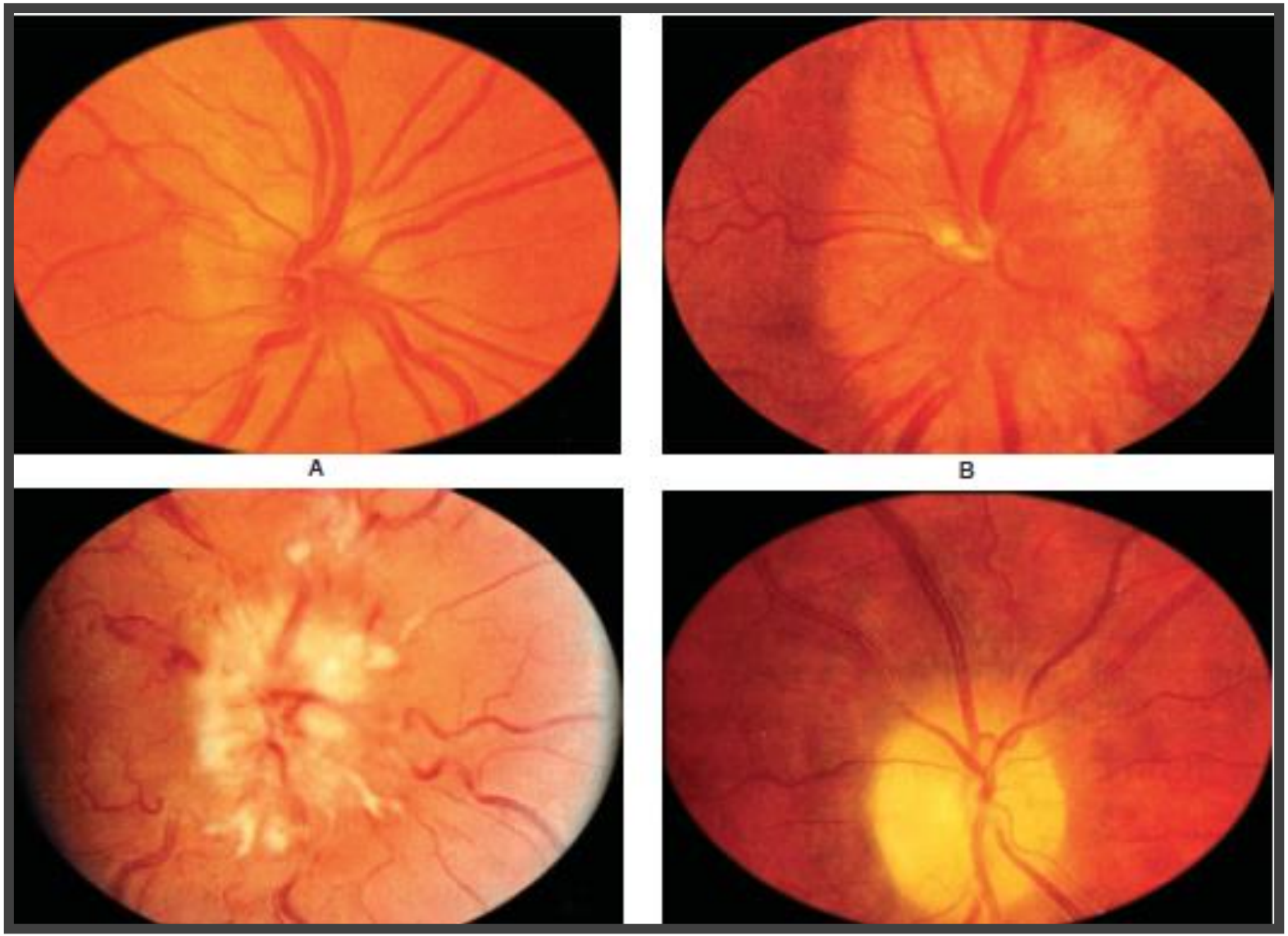
# Ocular symptoms

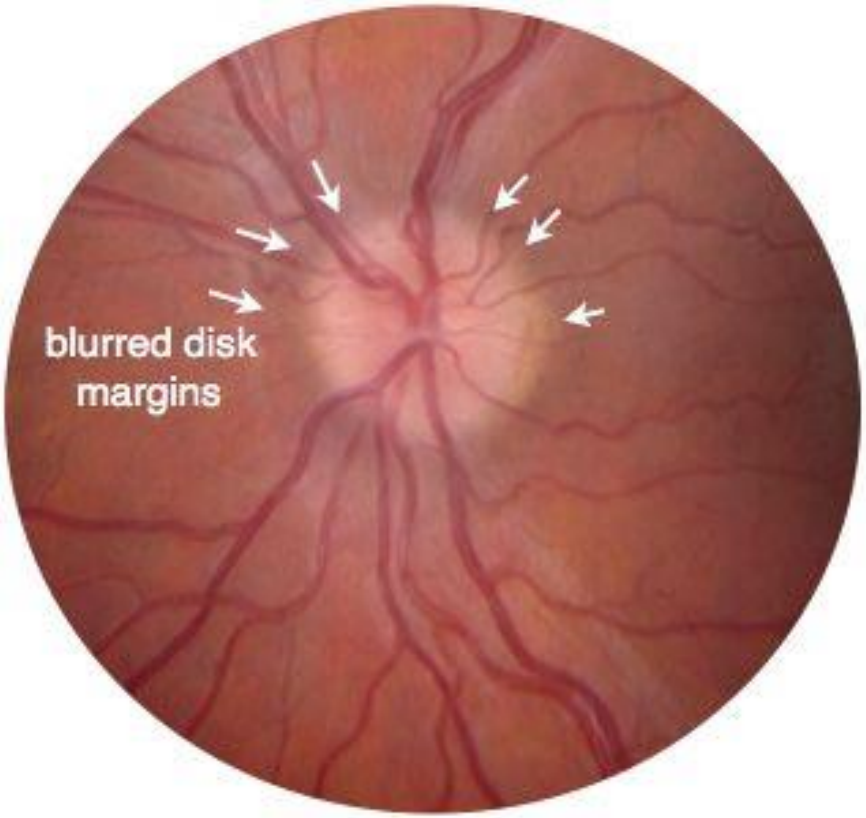
- VA may be normal until late stages
- Amaurosis fugax in some
- In 25% patients, visual symptoms occur only in severe, advanced papilloedema

# Clinical Features of Papilledema

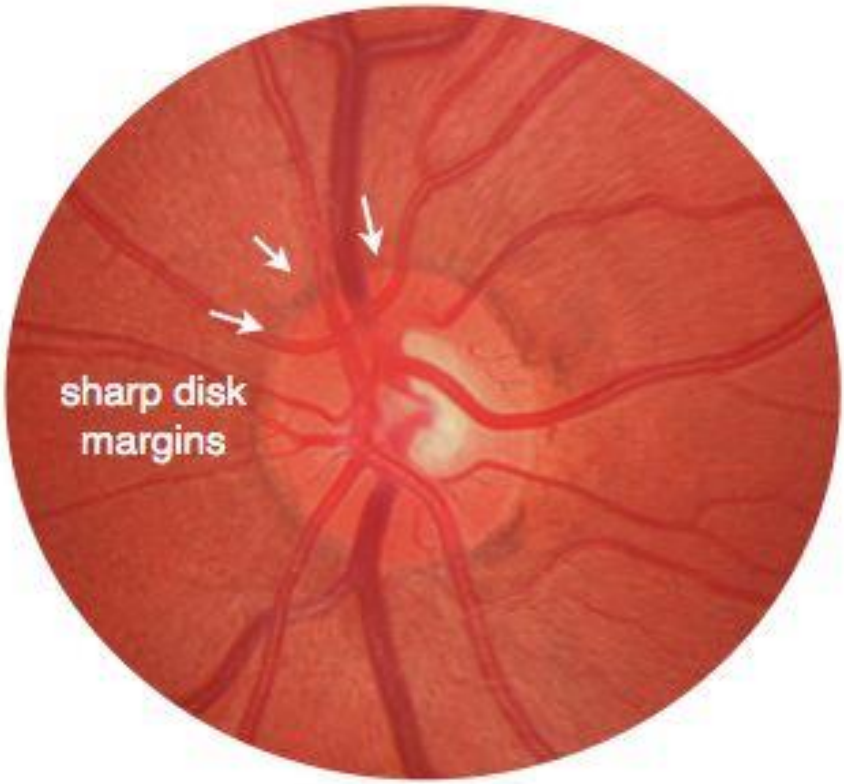
- Usually bilateral but may be unilateral or asymmetric
- Usually preserved visual acuity and color vision early
- May have transient visual loss lasting seconds (obscurations of vision)
- Visual field defects
- Enlarged blind spot
- Generalized constriction
- No afferent pupillary defect

# *Papilledema showing blurred disc margins and dilated tortuous vessels*






**Papilledema**




**Normal Optic Disk**

# Early papilledema

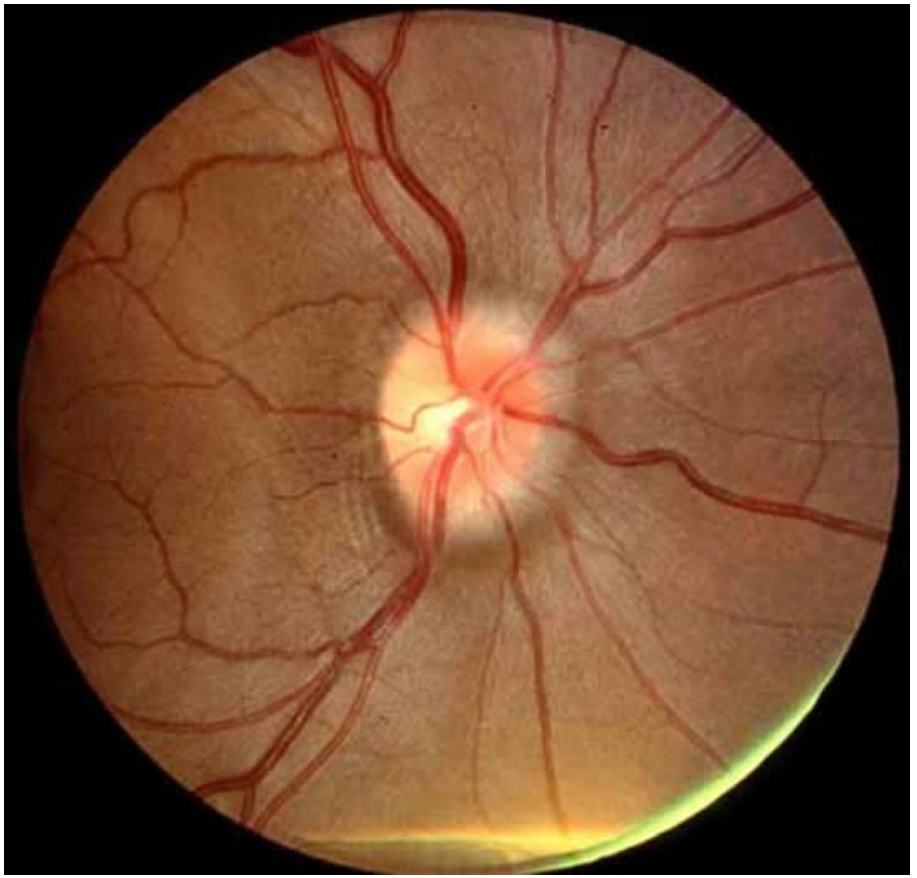
- Minimal disc hyperemia with capillary dilation
- Early opacification of nerve fiber layer (peripapillary retina loses its superficial linear and curvilinear light reflex and appears red without luster)
- Early swelling of disc
- Absence of venous pulsations
- Peripapillary retinal nerve fiber layer hemorrhage

# Fully developed papilledema

- Engorged and tortuous retinal veins
- May have splinter hemorrhages at or adjacent to the disc margin
- Disc surface grossly elevated
- Surface vessels become obscured by now
- May have cotton wool spots
- Hemorrhage and exudates

# Frisen Papilledema Grading System – Stage 1

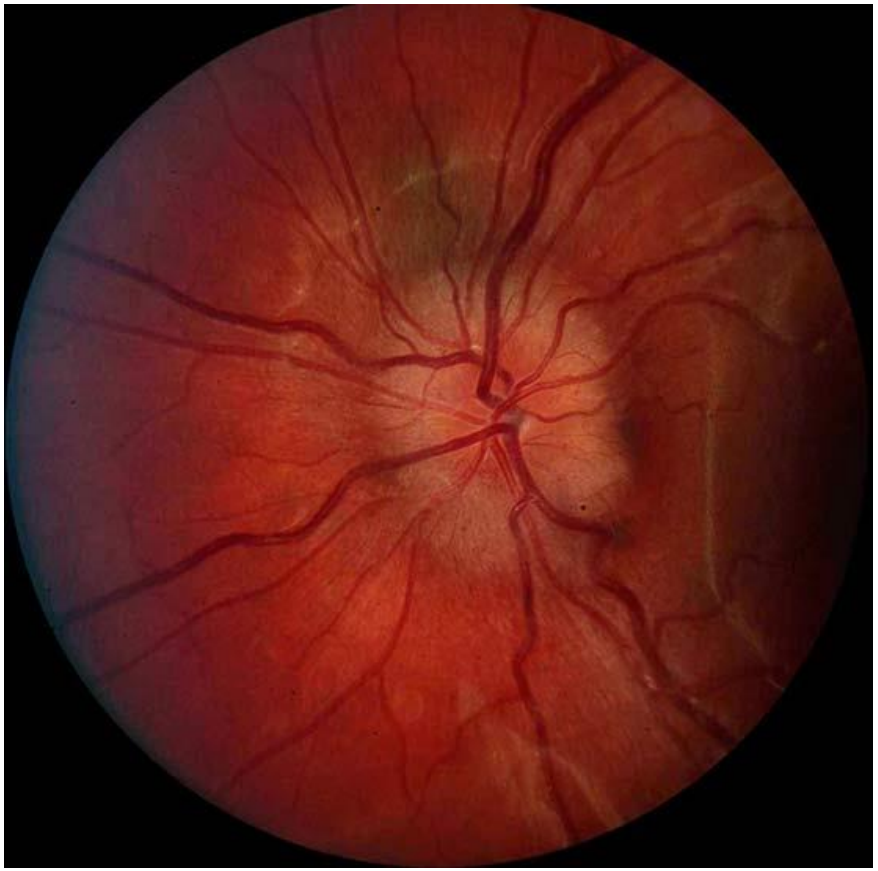
- Obscuration of the nasal border of the disc by opaque nerve fiber layer
- No elevation of the disc borders
- Disruption of the normal radial nerve fiber layer (NFL) arrangement with grayish opacity accentuating nerve fiber bundles
- Normal temporal disc margin
- Subtle grayish halo with temporal gap



C-shaped halo with a temporal gap

# Frisen Papilledema Grading System – Stage 2

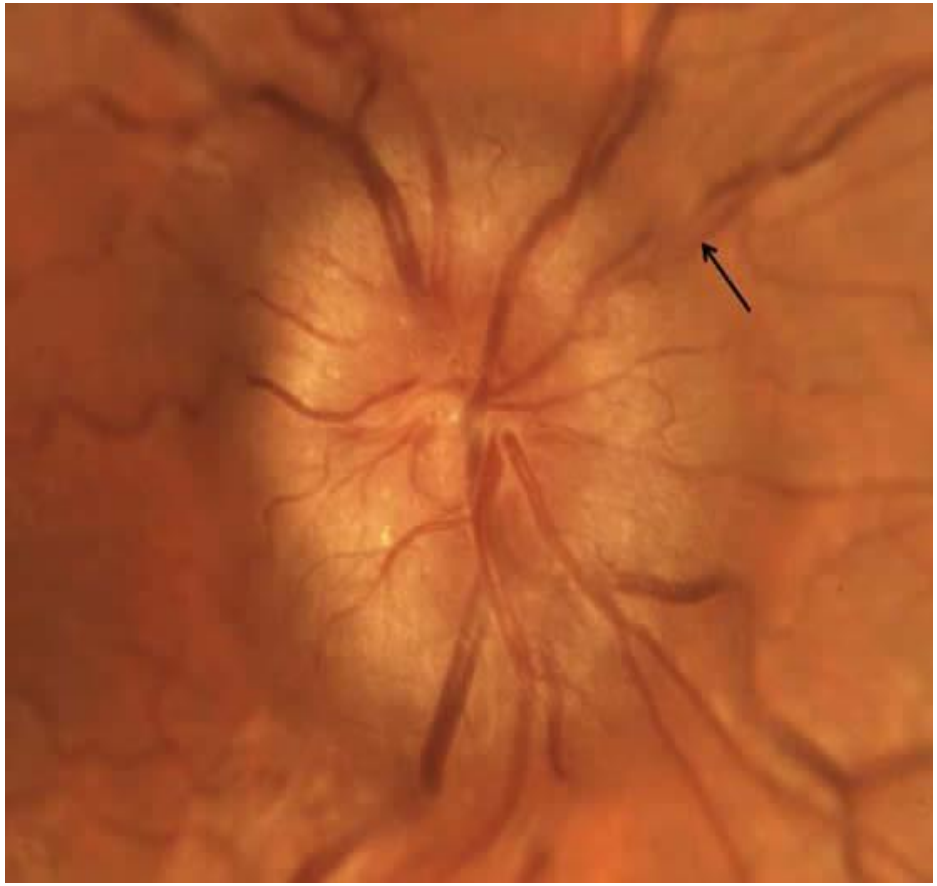
- Obscuration of all borders
- Elevation of nasal border
- Complete peripapillary halo



Halo becomes circumferential

# Frisen Papilledema Grading System – Stage 3

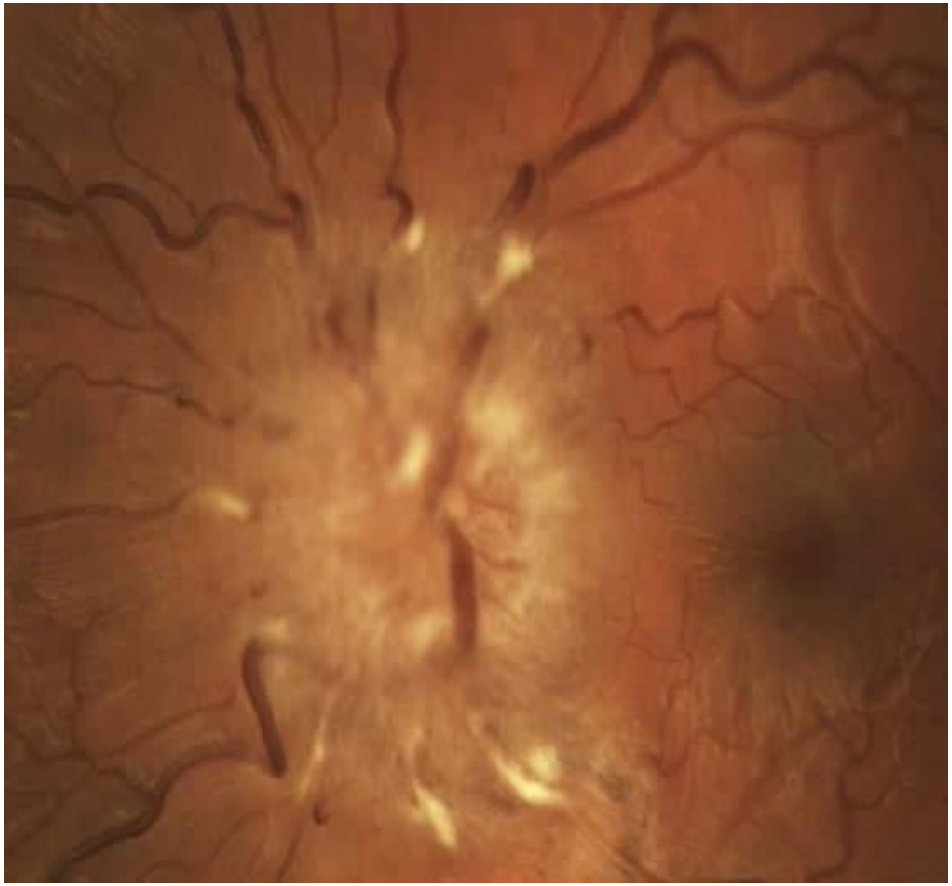
- Obscuration of all borders
- Elevation of all borders
- Increased diameter of the optic nerve head
- Obscuration of one or more segments of major blood vessels leaving the disc
- Peripapillary halo—irregular outer fringe with finger-like extensions



Loss of major vessels as they leave the disc (arrow)

# Frisen Papilledema Grading System – Stage 4

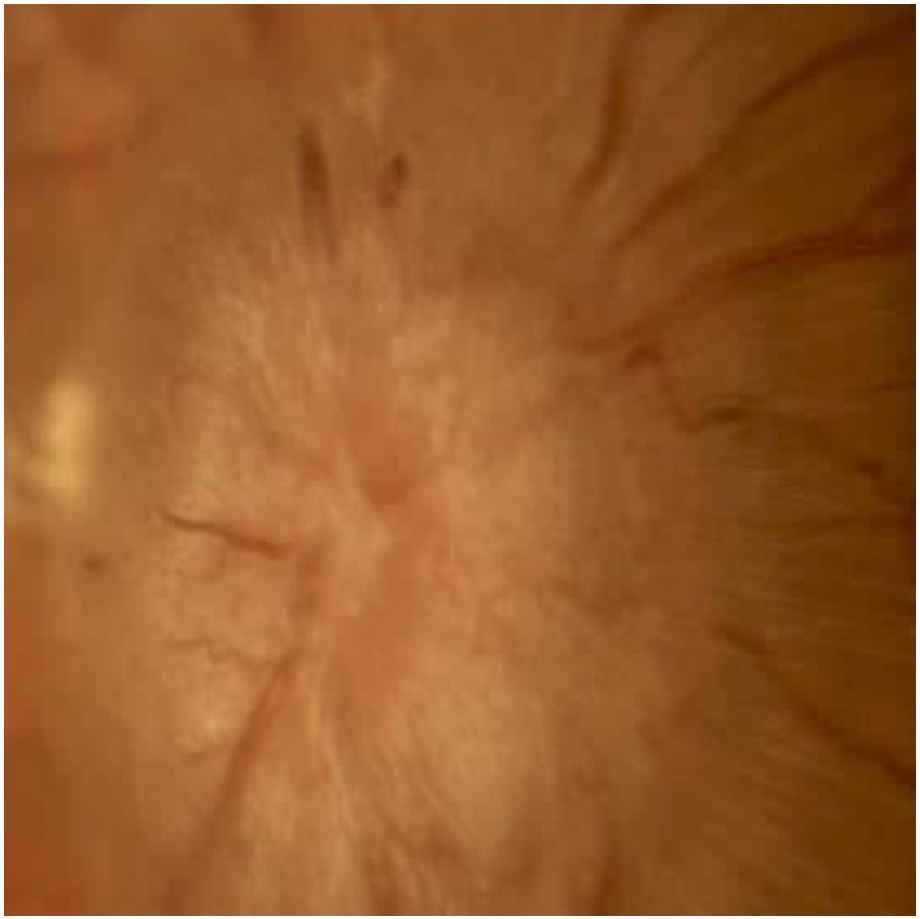
- Elevation of entire nerve head
- Obscuration of all borders
- Peripapillary halo
- Total obscuration on the disc of a segment of a major blood vessel
- Paton’s lines (circumferential retinal folds) or choroidal folds



loss of major vessels ON THE DISC

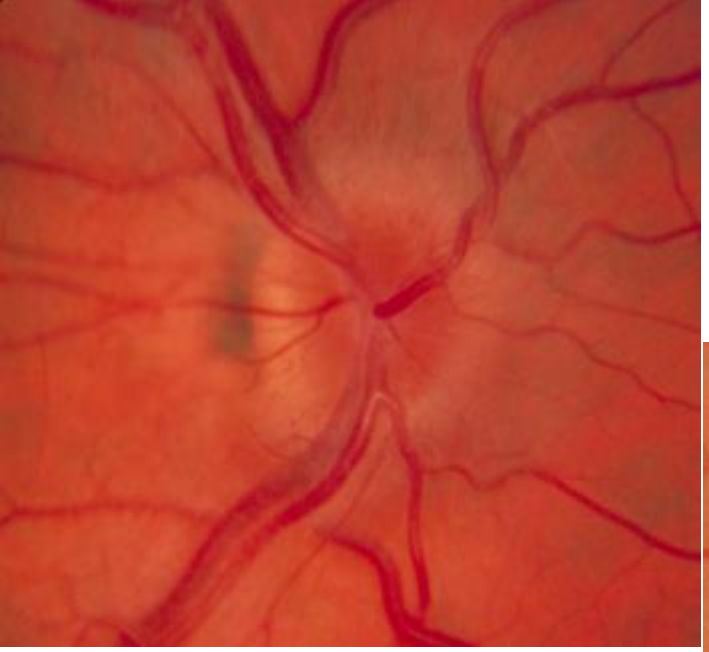
# Frisen Papilledema Grading System – Stage 5

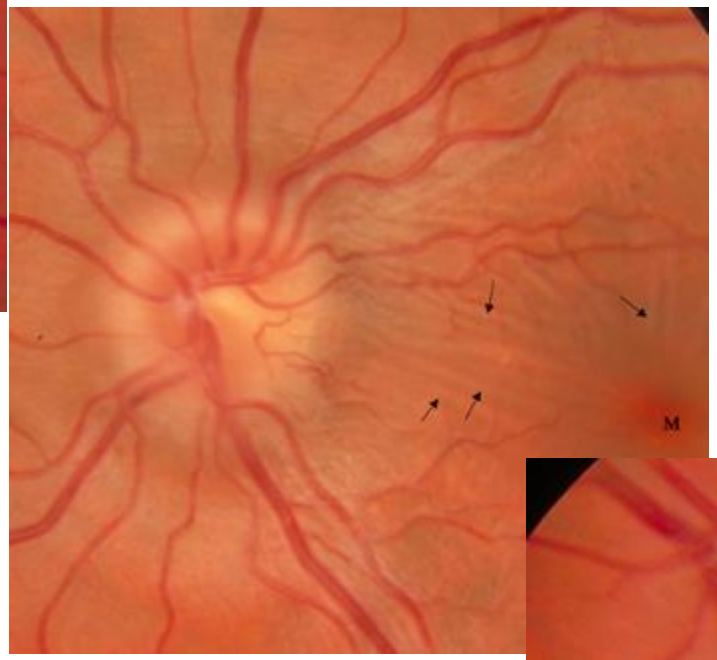
- Dome-shaped protrusions representing anterior expansion of the optic nerve head
- Peripapillary halo is narrow and smoothly demarcated
- Total obscuration of a segment of a major blood vessel may or may be present
- Obliteration of the optic cup

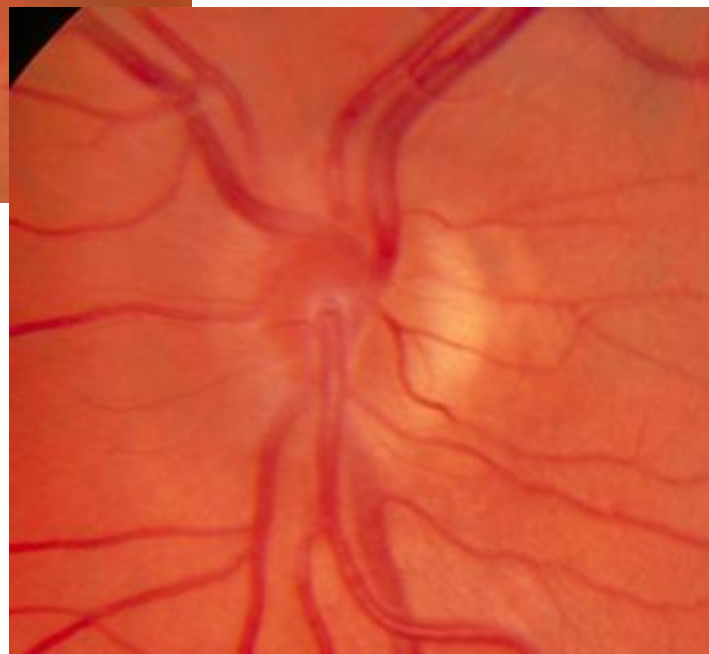


Grade IV plus partial or
total obscuration of all vessels of the disc

# Stage 1

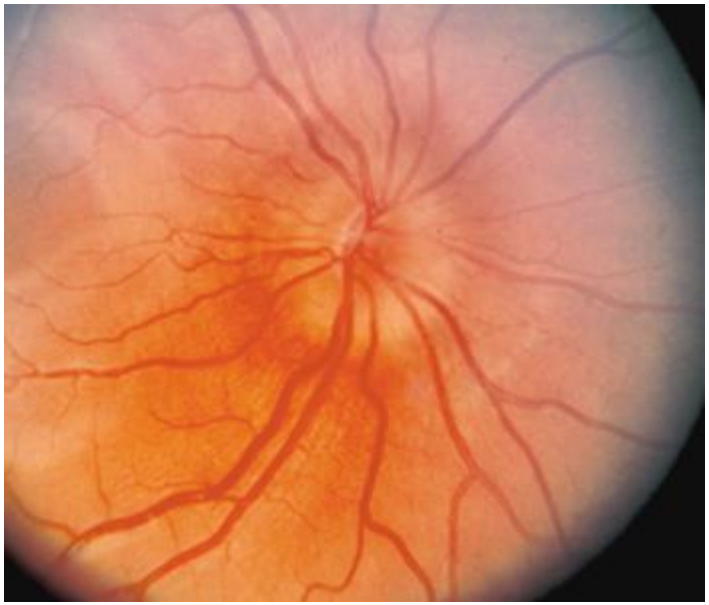






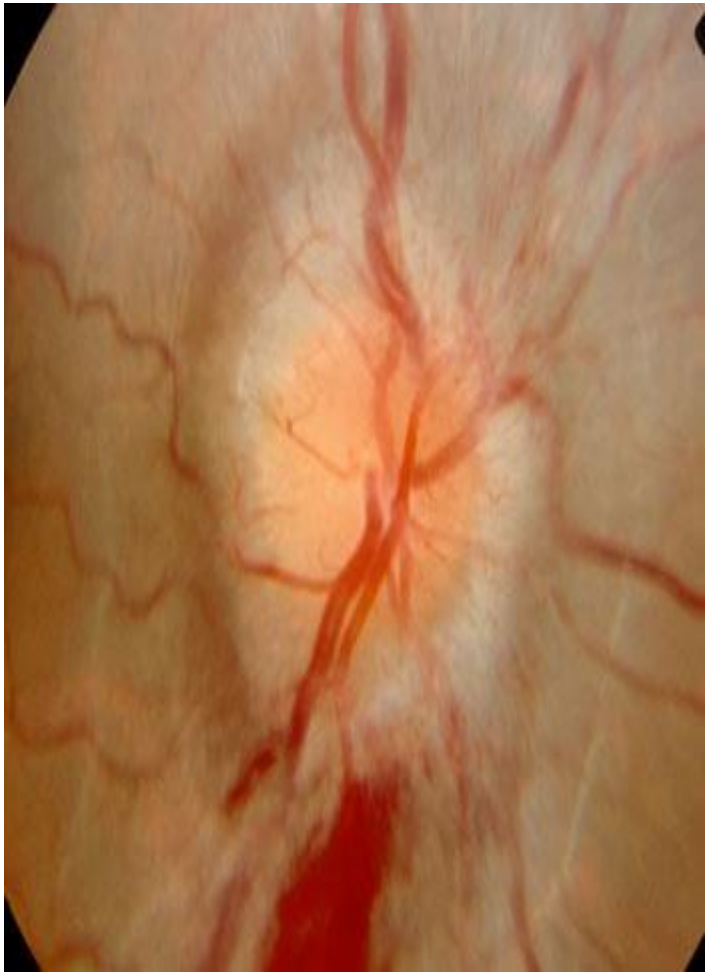
# Stage 2

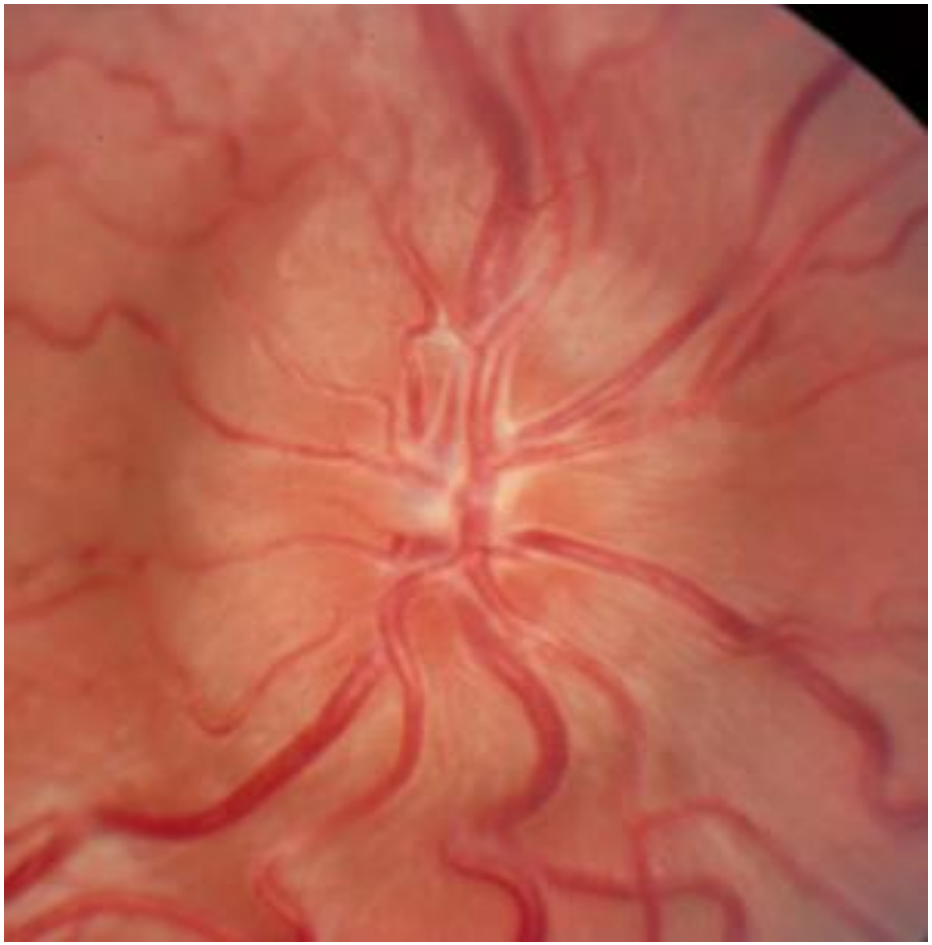
Elevation of the disc margin 360 degrees. Since the blood vessels at the disc margin are not swollen or obscured, this disc could be mistaken for pseudo-papilledema.

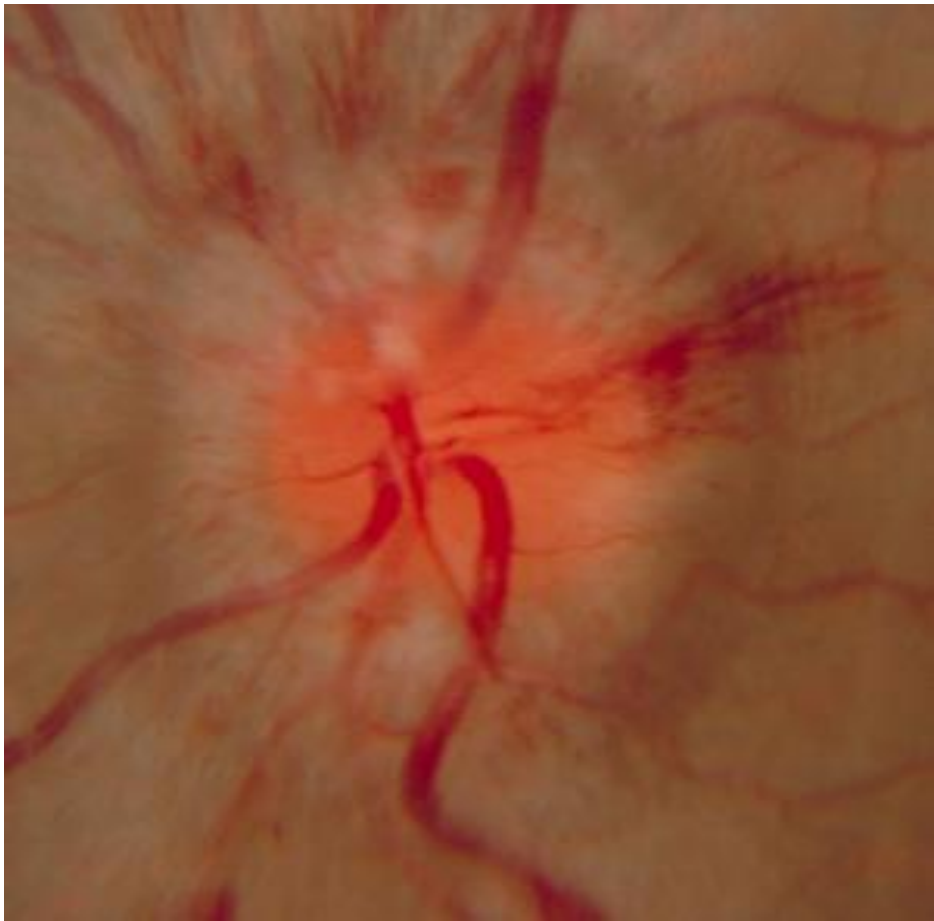


# Stage 3

Elevation of the entire disc with partial obscuration of the retinal vessels at the disc margin.

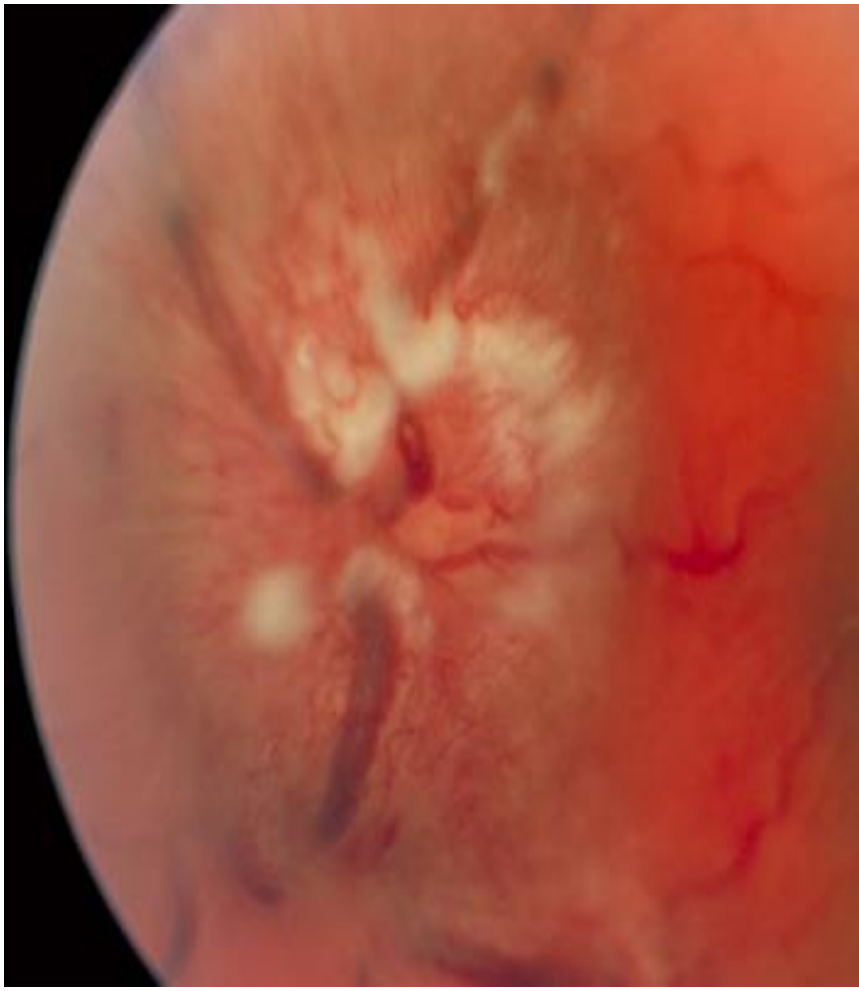


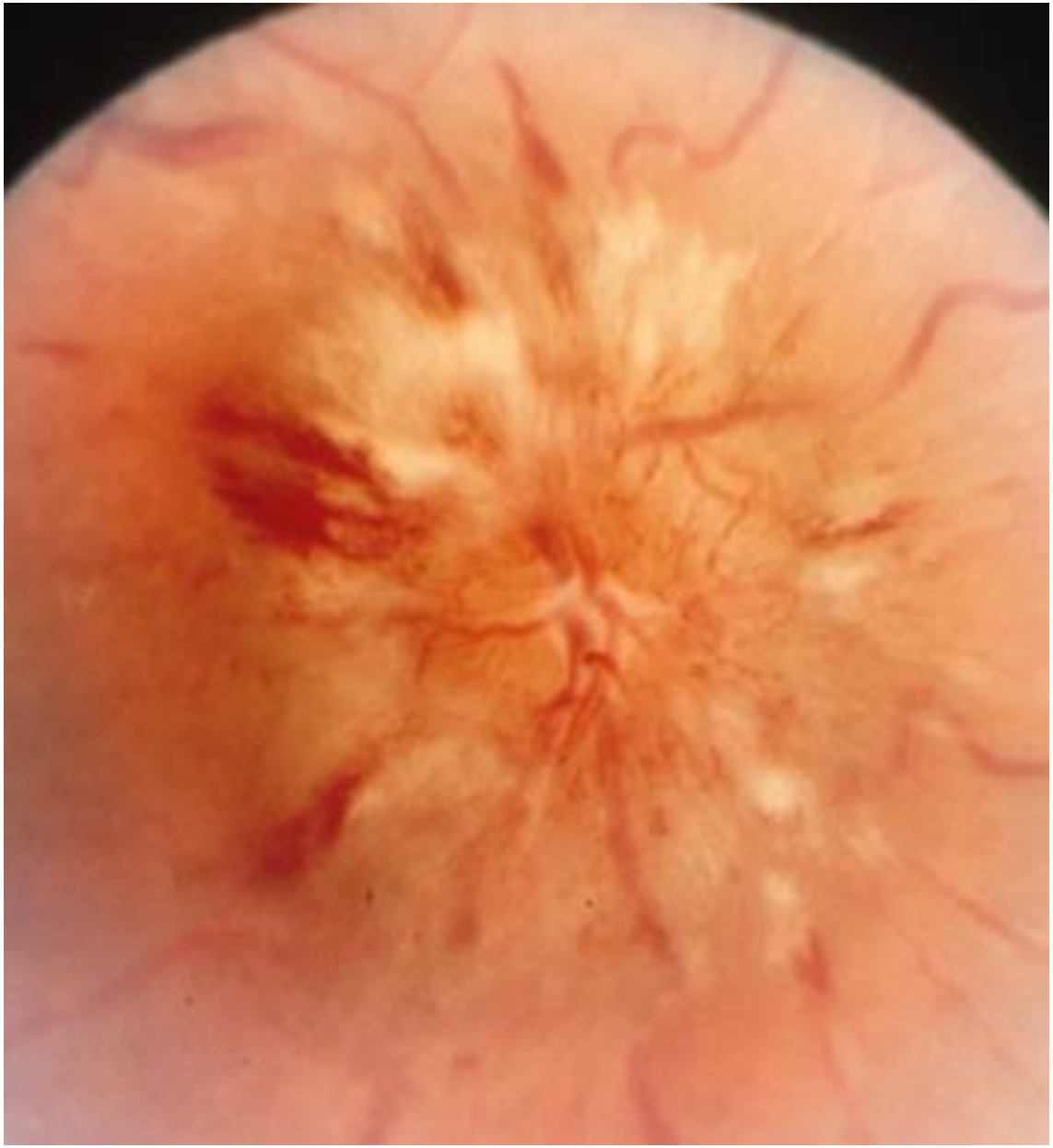




# Stage 4

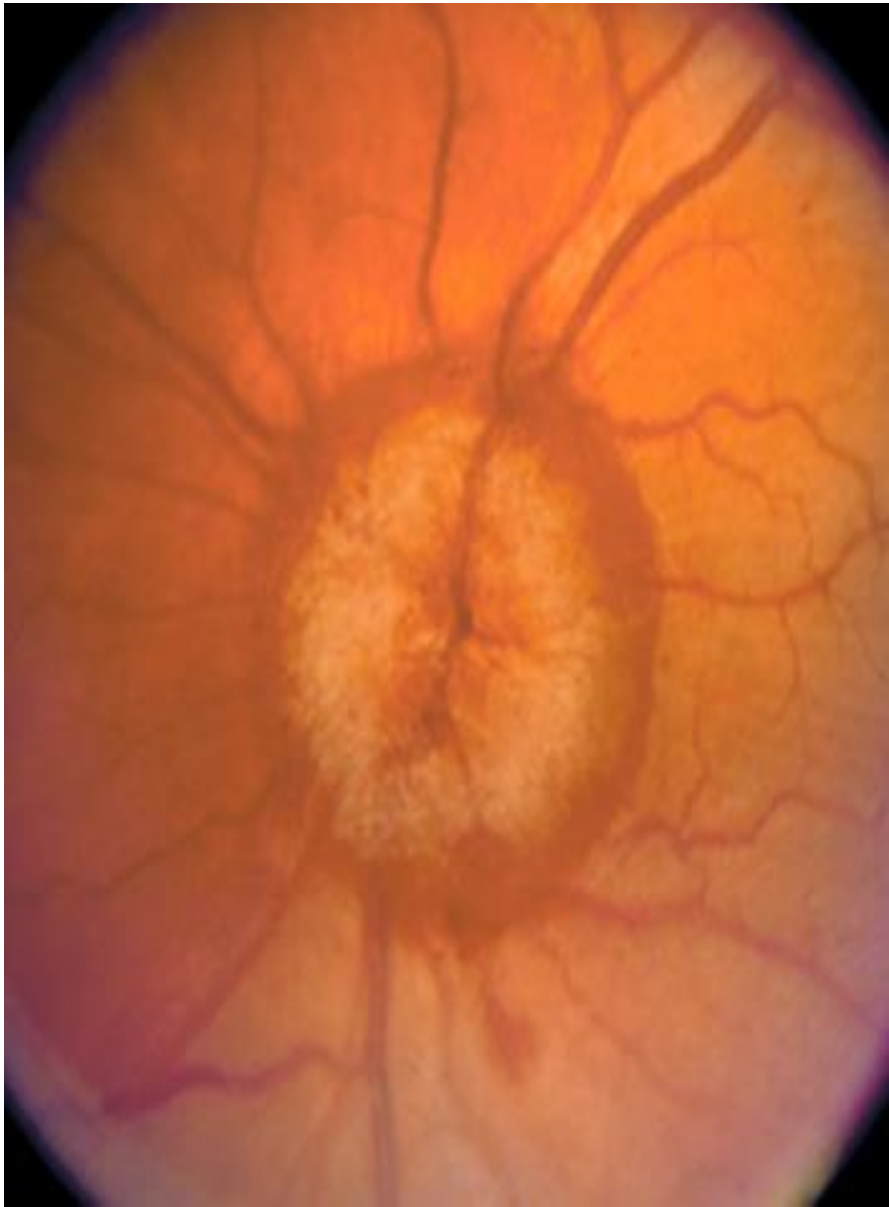
Complete obliteration of the cup and complete obscuration of at least some vessels on the surface of the disc. There may be small dilated capillaries on the disc that resemble telangiectasia. It is not the NFL infarcts or hemorrhages but the obscuration of the vessels themselves that makes this disc stage 4.

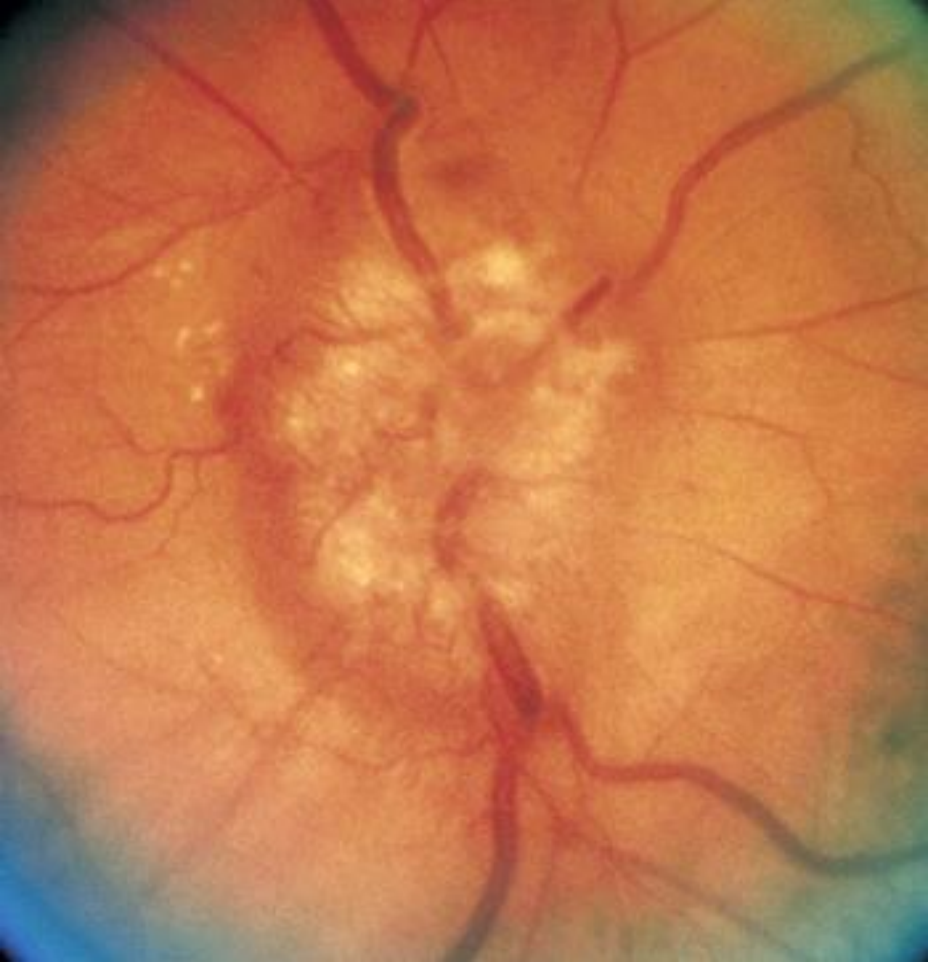


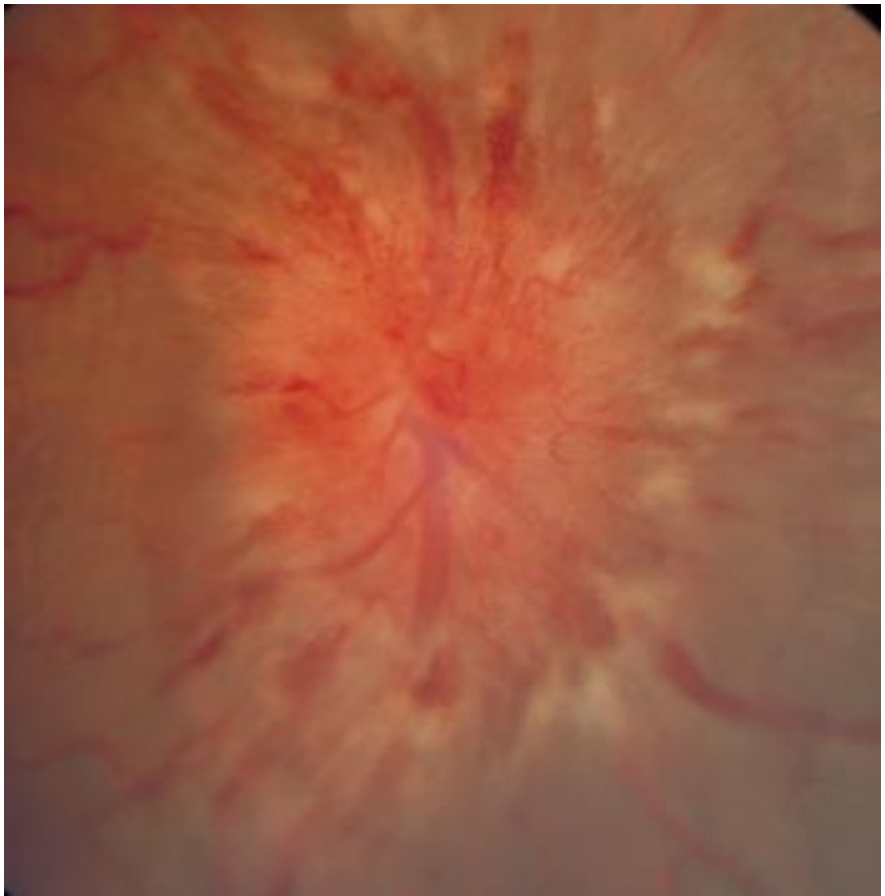


# Stage 5

Dome-shaped appearance with all vessels being obscured. (Sometimes called "champagne cork" swelling﷽because of its dome shape.)





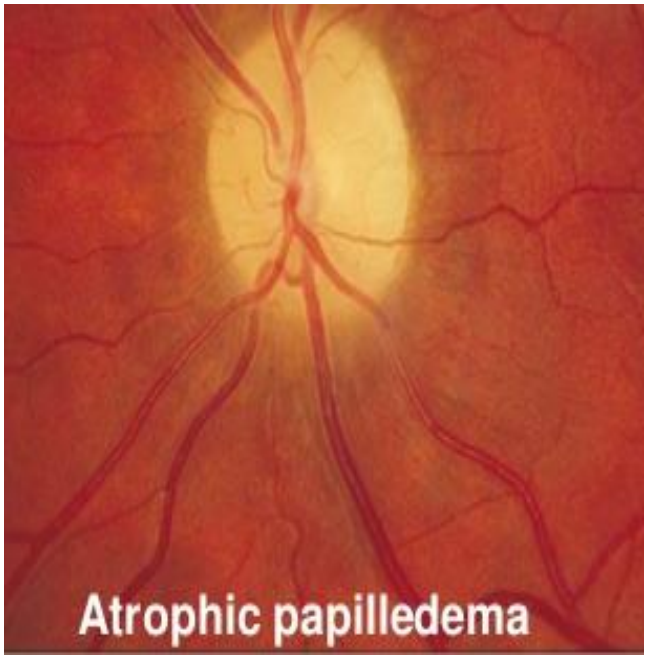


# Atrophic papilloedema

Retinal vessels attenuated with perivascular sheathing

Dirty white colour due to reactive gliosis

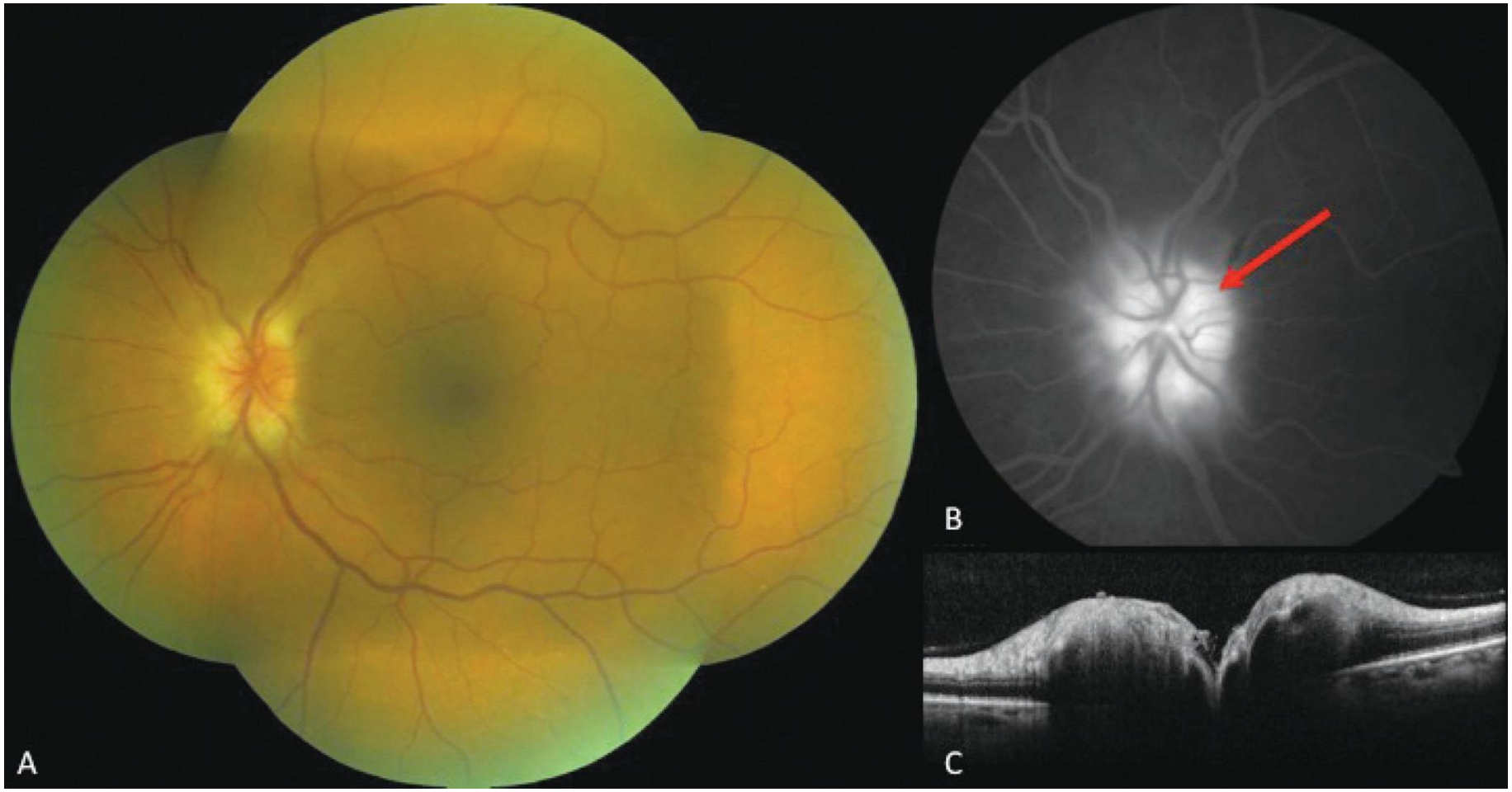
Leads to secondary optic atrophy



# Visual fields

- Early-no changes
- Established stage- enlarged blind spot
- Chronic- peripheral constriction of field with nerve fibre bundle defects
- Finally- total loss of visual field

# Fundus photo, FFA , OCT



# Idiopathic intracranial hypertension (benign intracranial hypertension or pseudotumor cerebri)

. It is a diagnosis of exclusion made in the presence of normal neuroimaging and CSF analysis, but with an elevated CSF opening pressure.

The prevalence is around 0.9/100,000 in the general population but up to 19/100,000 in obese young women.

# Risk factors

| | |
|---|---|
| Drugs | Tetracycline<br>Corticosteroids<br>OCP<br>Vitamin A derivatives<br>Nalidixic acid |
| Endocrine | Hypoparathyroidism<br>Adrenal adenomas |
| Habitus | Obesity<br>Obstructive sleep apnea syndrome |
| Hematological | Cerebral venous thrombosis |

# Clinical features

- Visual obscurations (transient dVA, few seconds duration, uni- or bilateral, up to 30x/day, may be precipitated by posture, straining, etc.); diplopia; field defects (usually enlarged blind spot); sustained dVA may be early in aggressive disease (usually an indication for shunting).

Headache (in 94% of cases; often worse lying down or straining), retrobulbar pain, pulsatile tinnitus.

- Disc swelling

# Investigation

- MRI with gadolinium enhancement and MRV: aim to rule out all other causes of ICP.
- LP: check opening pressure, glucose, protein, protein electrophoresis, microscopy, and culture. Normal opening pressure in adults is usually <20 cm H2O, or <25 cm H2O in the obese; in children, lower levels are normal.

# Treatment

Titrate treatment against symptoms and risk of visual loss (monitor VA, color vision, fi elds, discs). The evidence base for treatment is weak.

Treatment may include the following:

- Weight loss.
- Medical: acetazolamide (up to 500 mg 4x/day), or consider furosemide.

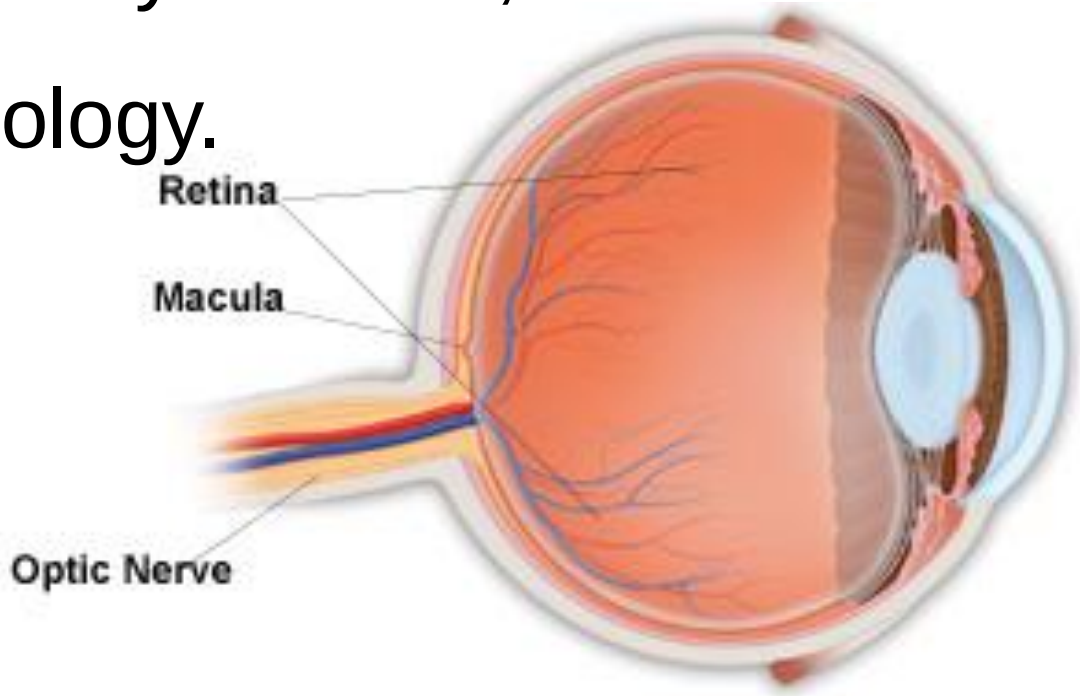
# Pseudopapilledema

- Optic nerve drusen
- Medullated nerve fiber
- Hypermetropic disc
- Congenital anomalous elevation

# *Optic Neuritis*

Definition: Inflammation of the optic nerve, impairing nerve conduction.

Secondary to demyelination, infection or autoimmune pathology.

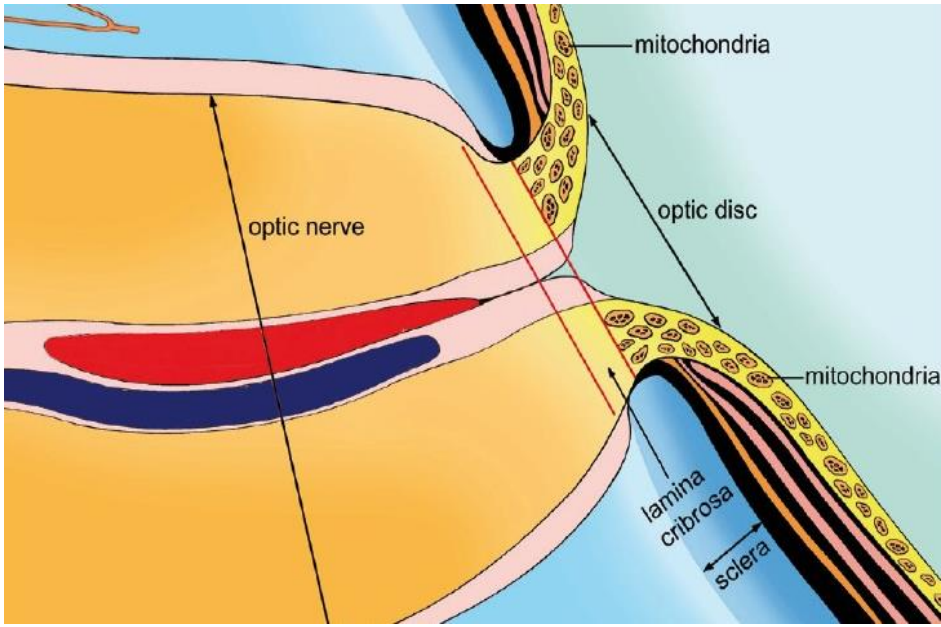


# Anatomical Classification

A. Papillitis

B. Retrobulbar neuritis

C. Neuroretinitis



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***Neuroretinitis*** *refers to combined involvement of* optic disc and surrounding retina in the macular area.

***Retrobulbar neuritis*** *is characterized by* involvement of optic nerve behind the eyeball. Clinical features of acute retrobulbar neuritis are essentially similar to that of acute papillitis except for the fundus changes and ocular changes described below.

# Aetiology

Idiopathic

Demyelinating disorders

Multiple Sclerosis

Presenting feature in 25% patients

70% cases occur in established disease

Recurs in same/ opposite eye in 25% patients

# Aetiology

Neuromyelitis optica (of Devic): acute, bilateral optic neuritis in young patient with paraplegia

Post-viral: mumps, measles, chicken pox, whooping cough

Metabolic/Nutritional deficiency:

B1, B6, B12, B2, Folic acid deficiency

Thyroid dysfunction, diabetes

Hereditary optic neuritis (Leber's disease)

# Aetiology

Toxic amblyopia:

Chloroquine, Ethambutol

Tobacco, Ethyl alcohol, methyl alcohol

Lead, Arsenic.

Ischaemic: Giant cell arteritis, Takayasu's disease,

PAN, SLE

Granulomatous inflammation:

Sarcoidosis, tuberculosis, syphilis

# Symptoms

- Idiopathic/demyelinating : 20-40 years of age
- Viral: children
- Uniocular sudden/rapid diminution of vision
- Visual loss, usually maximum by end of second week, improves by 1-4 weeks
- Discomfort/pain behind eyeball especially when moved superiorly

- *Visual loss. Sudden, progressive and profound*

visual loss is the hallmark of acute optic neuritis.

- *Dark adaptation may be lowered.*
- *Visual obscuration in bright light is a typical*

symptom of acute optic neuritis.

- *Impairment of colour vision is always present in*

optic neuritis. Typically the patients observe reduced vividness of saturated colours.

- *Movement phosphenes and sound induced phosphenes may be percieved by patients with* optic neuritis. Phosphenes refer to glowing sensations produced by nonphotic or the so called inadequate stimuli.
- *Episodic transient obscuration of vision on* exertion and on exposure to heat, which recovers on resting or moving away from the heat (Uhthoff's symptom) occurs in patient with isolated optic neuritis.

# Signs

***Visual Acuity:*** Usually 6/60 or less

***Local tenderness***

***Pupillary reaction:*** Sluggish or RAPD

***Impaired coloured vision:*** hue, brightness

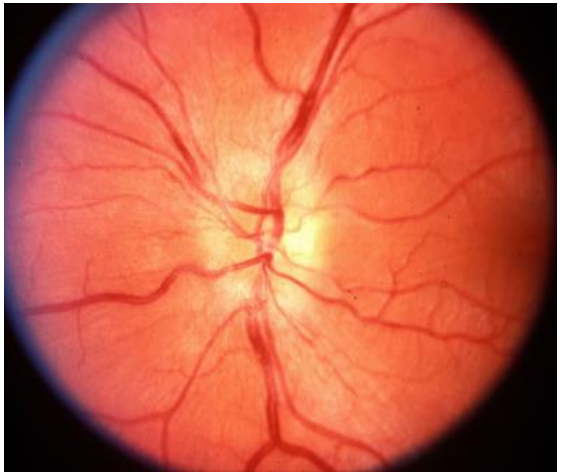
Impaired contrast sensitivity

Delayed dark adaptation

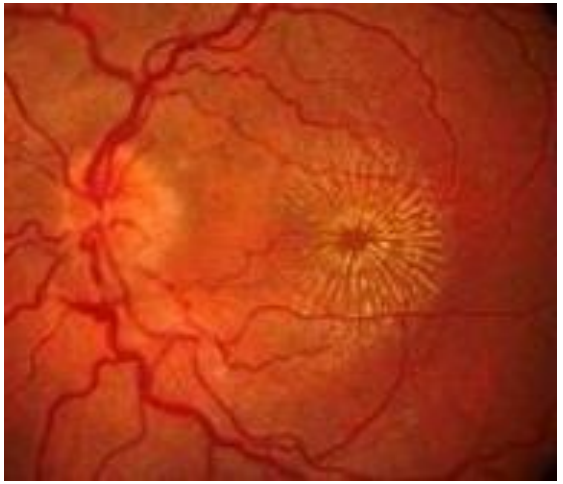
***Visual Field:*** central, centrocaecal or paracentral scotoma, more pronounced for coloured fields

# Ophthalmoscopic findings

***Optic neuritis:*** MC in children, engorged, oedematous optic disc with obliteration of optic cup, small haemorrhages on disc



***Retrobulbar neuritis:*** MC in adults



***Neuroretinitis:***

Optic neuritis+ macular star

# Differential diagnosis

## Papilloedema

## Pseudopapillitis

High hypermetropia,

Myelinated nerve fibres,

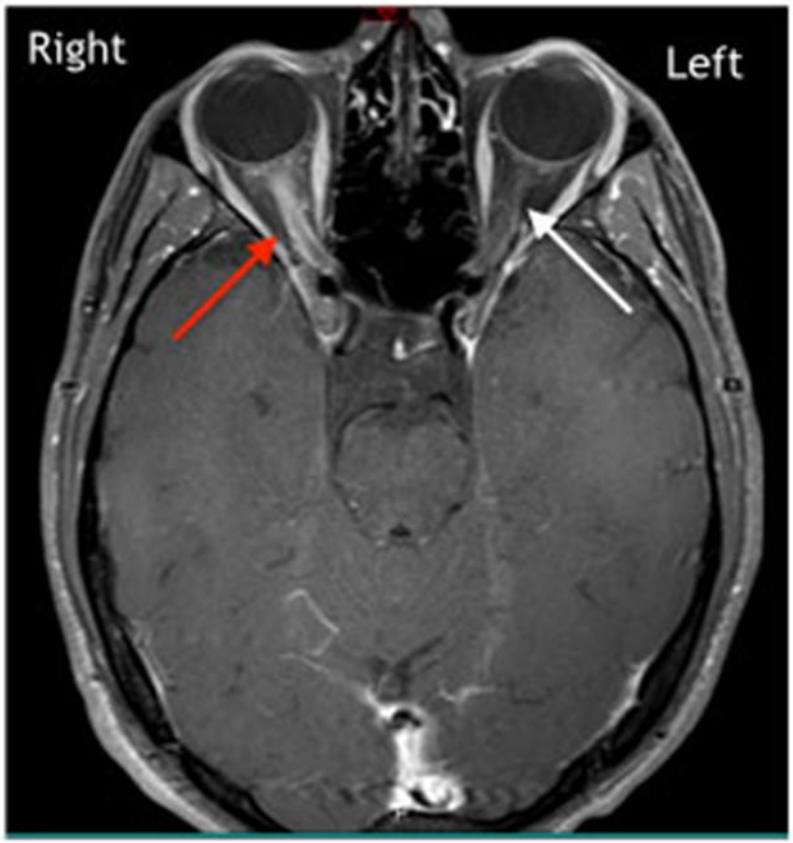
Optic nerve head drusen

(blurred margin, disc not significantly elevated, no vascular changes, stationary)

# Investigations

MRI: demyelinating lesions, SOL

VEP: reduced amplitude and delayed transmission time




Right optic neuritis

# Course and prognosis

Recovery takes 4-6 weeks

90% recover normal VA, but colour vision defects may persist

No correlation between initial visual loss and final visual outcome

10% secondary or post-neuritic optic atrophy

Better outcome in young, unilateral cases

# Treatment

- Of cause e.g. anti-infective therapy
- Intravenous methyl prednisolone 20 mg/kg/day(250 mg QID) for 3 consecutive days followed by oral prednisolone 1-1.5 mg/kg
- Dexamethasone 200 mg OD pulse for 3-5 days is a cheaper alternative
- Supportive therapy

| | *Papilloedema* | *Optic neuritis* |
|---|---|---|
| History | Headache, vomiting | Rapid DV preceded by fever/respiratory infection |
| Laterality | usually bilateral | usually unilateral |
| VA | normal till late stage | severely reduced ≤6/60 |
| Pain/tenderness of eyeball | absent | may be present |
| Pupil reaction | normal | RAPD (Marcus-Gunn's pupil) |
| Disc swelling | >+3 D in established | +2D to +3D |
| Haemorrhage, exudates | More, in established | relatively less |
| Visual fields | Enlarged blind spot, later gradual constriction | Central or centrocaecal scotoma |
| Colour vision | No effect | Affected |
| CT/MRI | SOL | Demyelinating disorder |
| Recovery of vision | May not be complete even after treatment | Usually complete after adequate treatment |

# ANTERIOR ISCHAEMIC OPTIC NEUROPATHY (AION)

It refers to the segmental or generalised infarction of anterior part of the optic nerve.

# AION

- is a significant cause of acute visual loss in the elderly population,
- affecting up to 10/100,000/year of those over 50 years of age.

# Arteritic AION

- In arteritic AION, short posterior ciliary artery vasculitis leads to ischemic necrosis of the optic nerve head.
- Constitutes 5–10% of cases of AION
- Giant cell arteritis (GCA) is an ophthalmic emergency.

# Clinical features

- Sudden decreased VA (<20/200 in 76%); headache, scalp tenderness, jaw claudication, weight loss, night sweats, myalgia (association with polymyalgia rheumatica); may have a warning episode of transient dVA.

RAPD, swollen disc (typically pale; rarely segmental), ± peripapillary hemorrhages and cotton wool spots, abnormal temporal arteries (thickened, tender, nonpulsatile).

- Associations: CRAO, BRAO, cilioretinal artery occlusion, CN III, IV, VIpalsy.

# Investigations

Immediate ESR, CRP, CBC

Consider urgent temporal artery biopsy (aim to perform it within a few days, although positive results may be obtained up to 7 days after corticosteroid treatment).

# Treatment

immediate adequate steroid treatment (e.g., 1 g methylprednisoloneIV 1x/day for 1–3 days) followed by oral prednisolone 1–2 mg/kg 1x/day).

Treatment may last several years so osteoporosis prophylaxis is important.

# Prognosis

The risk of second eye involvement ranges from 10% (if treated) to 95% (untreated). Other complications of GCA include TIA, stroke, neuropathies, thoracic artery aneurysms, and death.

| | Arteritic AION | Nonarteritic AION |
|---|---|---|
| Incidence | 1/100,000/year | 10/100,000/year |
| Cause and possible associations | Giant cell arteritis | *Major*: diabetes mellitus, hypertension, optic disc morphology<br>*Minor*: smoking, hyperlipidemia, hypotension, anemia, hypermetropia, obstructive sleep apnea |
| Age (mean) | 70 years | 60 years |
| VA + field | Sudden ↓<br>Usually <20/200 | Sudden ↓<br>Usually >20/200<br>Often altitudinal field loss |
| Associated symptoms | Scalp tenderness, jaw claudication, headache | Usually none |
| Disc | Swollen<br>Commonly pale | Swollen (often sectoral)<br>Commonly hyperemic<br>Predisposed (small + crowded) |
| ESR | ↑↑ (mean = 70 mmHg) | Normal |
| CRP | ↑↑ | Normal |
| Plt | ↑ | Normal |
| Risk to fellow eye | 10% (if treated) to ≤95% (untreated) | 19% over 5 years |
| Prognosis | Up to 15% improve | 40% improve (by ≥2 Snellen lines) |

Nonarteritic AION comprises 90–95% of AION cases (see Table 16.5).

It is proposed that an insuffi cient circulation to a crowded optic nerve head may lead to local edema, causing further vascular compromise and subsequent infarction. Identifi ed vascular

# Risk factors

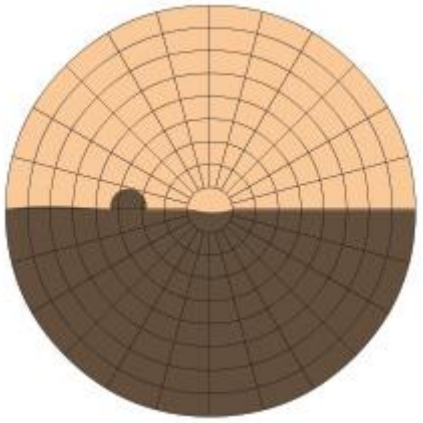
- Diabetes, hypertension
- Optic disc morphology (“disc at risk”—crowded disc with a small cup).
- smoking, hyperlipidemia, hypotension, anemia, hypermetropia, and obstructive sleep apnea.

# Clinical features

- dVA (usually sudden but can be progressive;
- RAPD, fi eld loss (45% inferior altitudinal; 15% superior altitudinal),

swollen optic disc (typically hyperemic, ± segmental, telangiectasia).

Visual fields show typical altitudinal hemianopia involving the inferior (commonly) or superior half



# Investigations

- First rule out GCA
- If nonarteritic, then obtain BP, glucose, lipids, CBC. If patient is <50 years of age, then consider also vasculitis screen.

# Treatment

There is no proven benefit for any treatment (including steroids, optic nerve sheath fenestration, hyperbaric oxygen, dopamine, and aspirin);

Refer to the physician for vascular assessment and treatment.

Treatment. Immediate treatment with heavy doses of corticosteroids (80 mg prednisolone daily) should be started and tapered by 10 mg weekly. Steroids in small doses (5 mg prednisolone) may have to be continued for a long time (3 months to one year).

# Prognosis

The risk of second eye involvement is around 19% over 5 years, with an increased risk after cataract surgery. Additionally, cardiovascular and cerebrovascular diseases are more common, possibly with increased mortality.

| | *Feature* | *Papilloedema* | *Papillitis* | *Pseudopapillitis* |
|---|---|---|---|---|
| 1. | Laterality | Usually bilateral | Usually unilateral or bilateral | May be unilateral |
| 2. | Symptoms | | | |
| | (i) Visual acuity | Transient attacks of blurred vision Later vision decreases due to optic atrophy | Marked loss of vision of sudden onset refractive error | Defective vision depending upon the degree of |
| | (ii) Pain and tenderness | Absent | May be present with ocular movements | Absent |
| 3. | Fundus examination | | | |
| | (i) Media | Clear | Posterior vitreous haze is common | Clear |
| | (ii) Disc colour | Red and juicy appearance | Marked hyperaemia | Reddish |
| | Disc margins | Blurred | Blurred | Not well defined |
| | Disc swelling | 2-6 dioptres | Usually not more than 3 dioptres | Depending upon the degree of hypermetropia |
| | (iii) Peripapillary oedema | Present | Present | Absent |
| | (iv) Venous engorgement | More marked | Less marked | Not present |
| | (v) Retinal haemorrhages | Marked | Usually not present | Not present |
| | (vi) Retinal exudates | More marked | Less marked | Absent |
| | (vii) Macula | Macular star may be present | Macular fan may be present | Absent |
| 4. | Fields | Enlarged blind spot | Central scotoma more for colours | No defect |
| 5. | Fluorescein angiography | Vertical oval pool of dye due to leakage | Minimal leakage of dye | No leakage of dye |

# Optic atrophy - Definition

Optic nerve shrinkage from any process that produce degeneration of axons

# ***Optic Atrophy***

Definition: Degeneration of optic nerve fibres in the ant.visual (Retinogeniculate) pathway with loss of their myelin sheaths characterised by pallor of the optic disc due to loss of vascularity owing to obliteration of disc capillaries

# CLASSIFICATION OF OPTIC ATROPHY

## PRIMARY-

## SECONDARY –

Post- papilloedemic optic atrophy

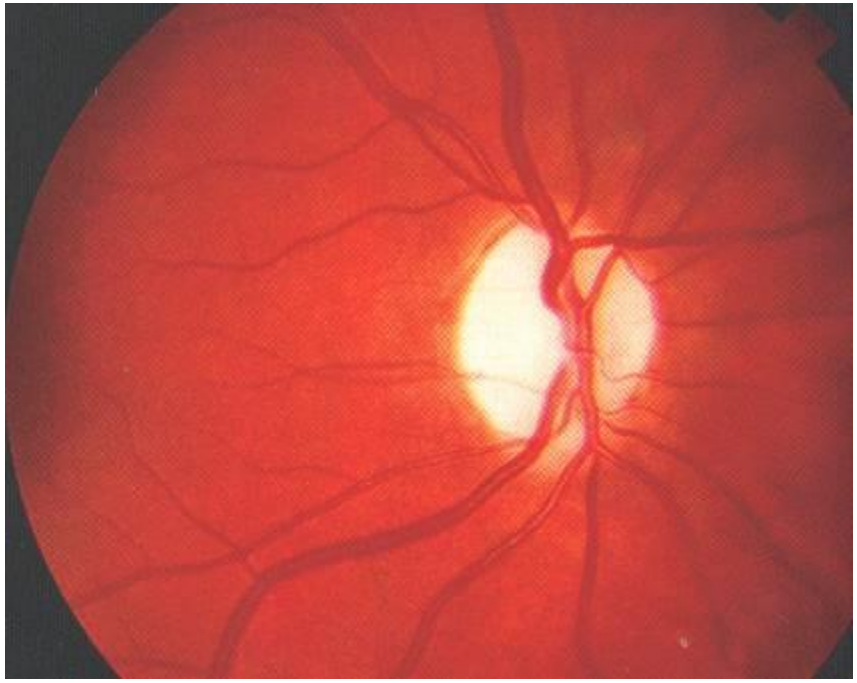
Post-Neuritic optic atrophy

Glaucomatous optic atrophy

Consecutive optic atrophy

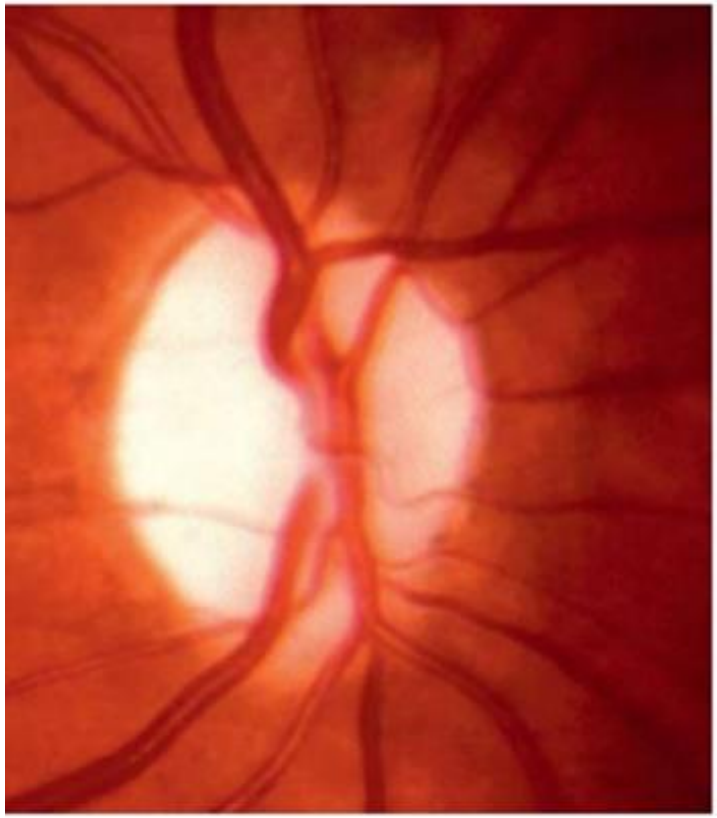
# PRIMARY OPTIC ATROPHY

- Pale disc
- Chalky white(full moon against a dark red sky)
- Clear margin of disc/sharply demarcated
- Normal cup
- Well seen lamina cribrosa
- Normal retinal vessels



# Classification-Aetiological
# Primary Optic Atrophy

No local disturbance, associated with CNS disease or no discoverable cause



Commonest cause

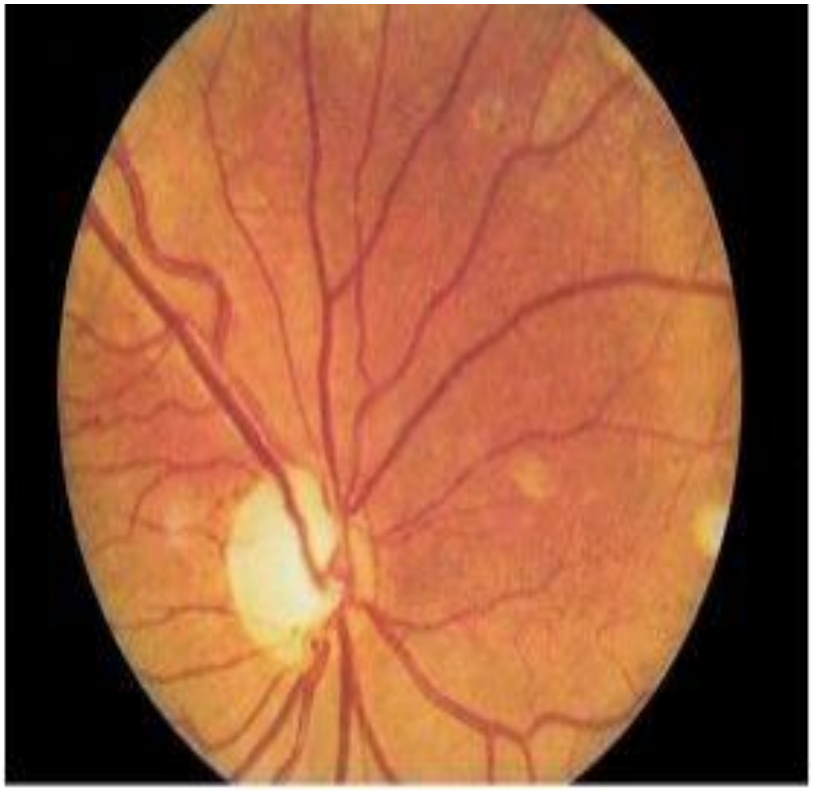
- Multiple Sclerosis
- Leber's optic atrophy
- Nerve compression:
- Tumour, hydrocephalus
- Injury to retrobulbar optic nerve

# Primary OA ophthalmoscopy

The Kestenbaum count is the number of capillaries observed on the optic disc.

The normal count is approximately 10.

In optic atrophy, the number of these capillaries reduces to < 6; in a hyperemic disc, the count is > 12



Primary Optic Atrophy

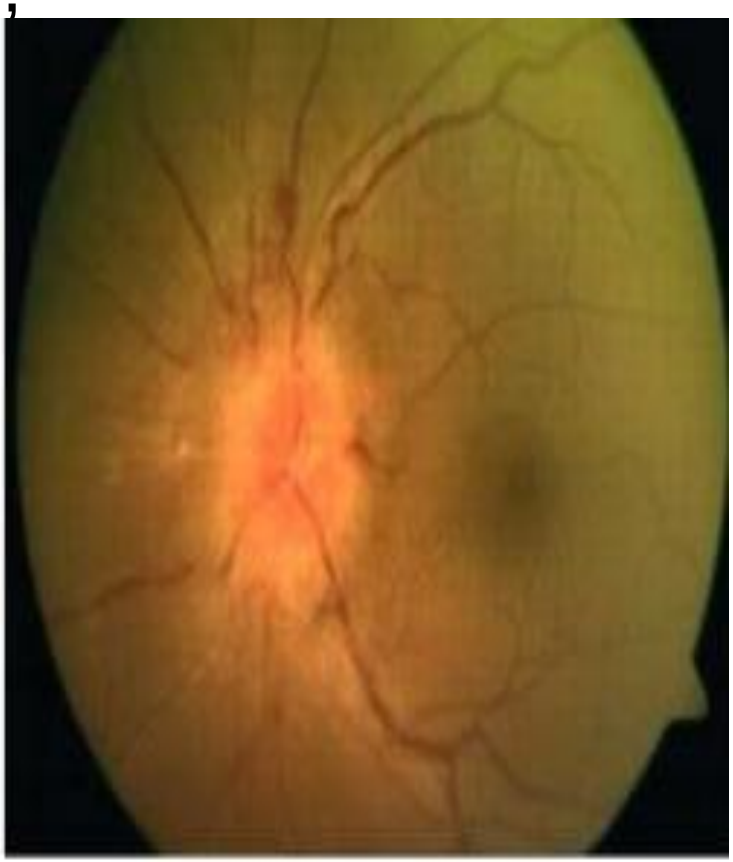
# Secondary OA ophthalmoscopy

Pale disc with dirty-grey colour, blurred margins

Physiological cup is full, lamina cribrosa obscured

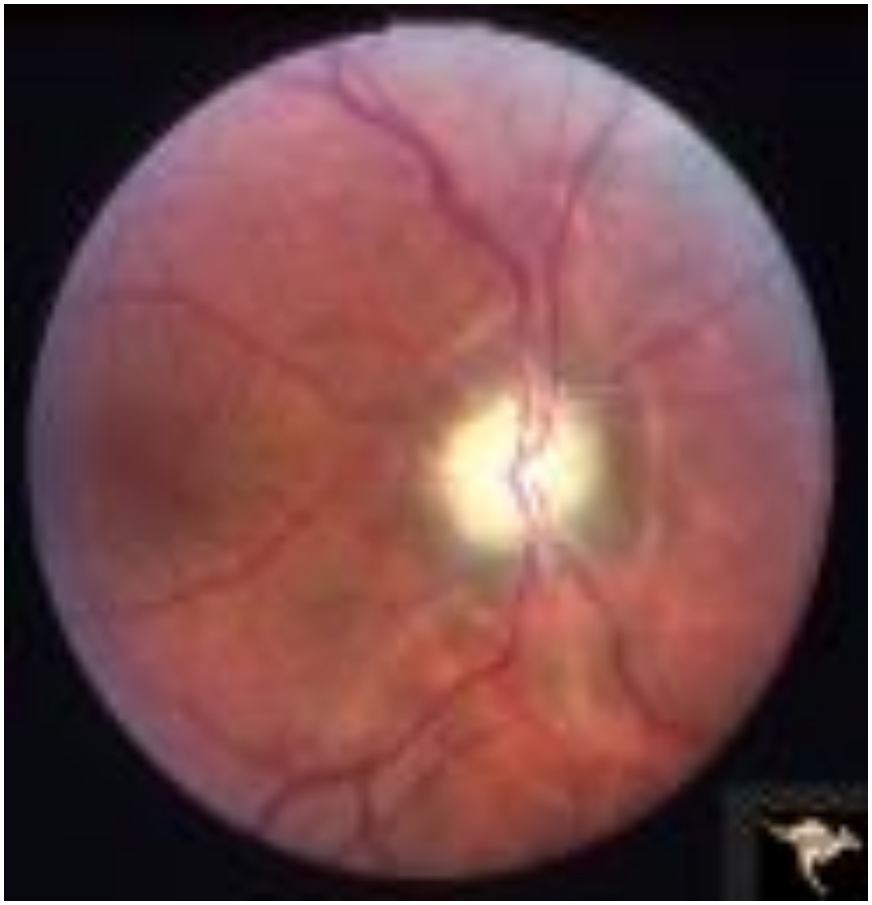
Narrowing of blood vessels with sheathing

Gliosis over disc surface extending towards peripapillary retina



Secondary Optic Atrophy

# Secondary optic atrophy



# Aetiological classification

Secondary optic atrophy: preceded by swelling of optic disc- papilledema, optic neuritis, neuroretinitis

Consecutive optic atrophy: follows extensive disease of the retina - Retinitis pigmentosa

Long-standing retinal detachment

# Symptoms

Gradual/rapid loss of central/peripheral vision

Impairment of colour vision

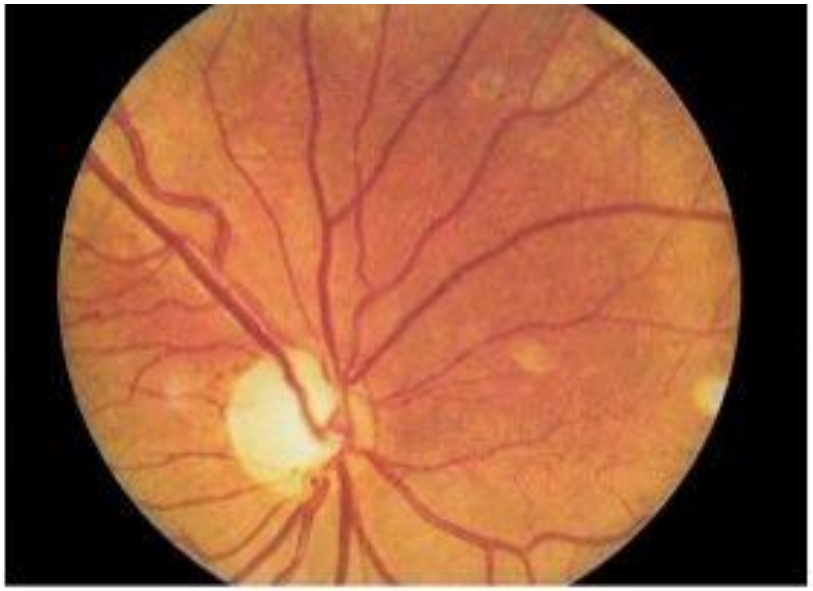
# Signs

Visual Acuity impaired in proportion to death of optic nerve fibres

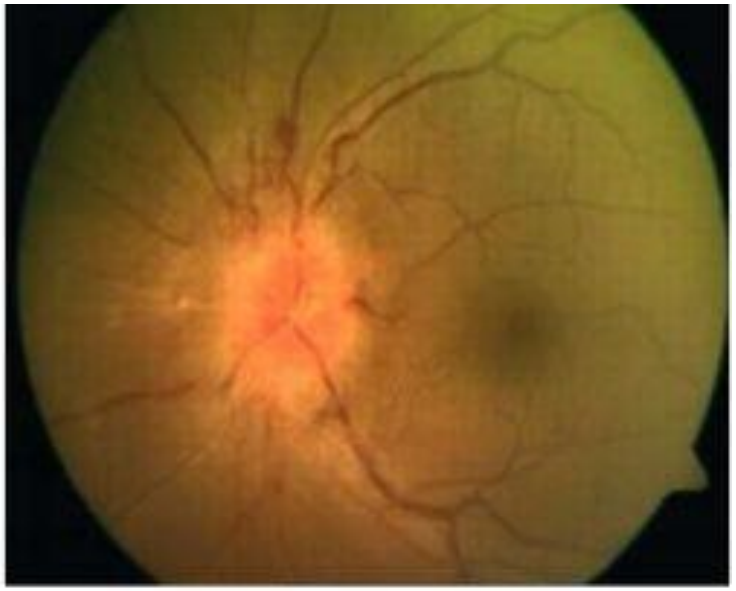
RAPD in unilateral Optic Atrophy

Ultimately pupil dilated and immobile

| | *Primary OA* | *Secondary OA* |
|---|---|---|
| Appearance | chalky white | dirty grey |
| Margins | sharply defined | blurred |
| Cup | deep | obliterated |
| Laminar dots | visible | not visible |
| Glial proliferation | absent | marked |
| Vessels | no sheathing | sheathing |
| Previous disc oedema | absent | present |



Primary Optic Atrophy



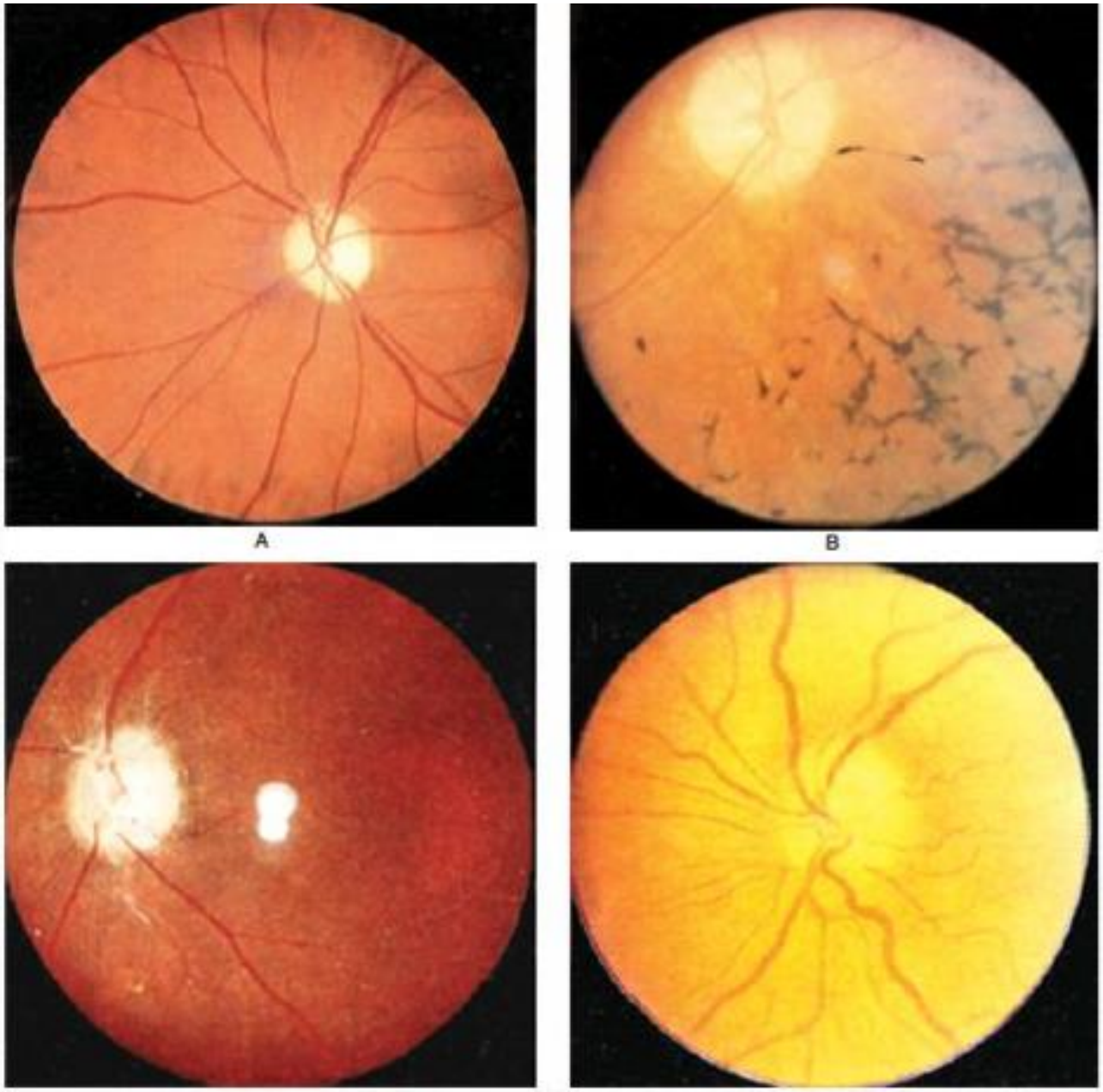
Secondary Optic Atrophy

# Consecutive optic atrophy

Yellowish-waxy pallor of the disc

Margins less sharply defined

Marked narrowing, even obliteration of retinal blood vessels




a) Primary, b) Consecutive, c) Postneuritic, d) Ischemic

# Investigations

Visual field: In partial OA, central vision is depressed with concentric contraction of the visual field

FFA of Optic nerve head

VEP especially in children

Neurological evaluation

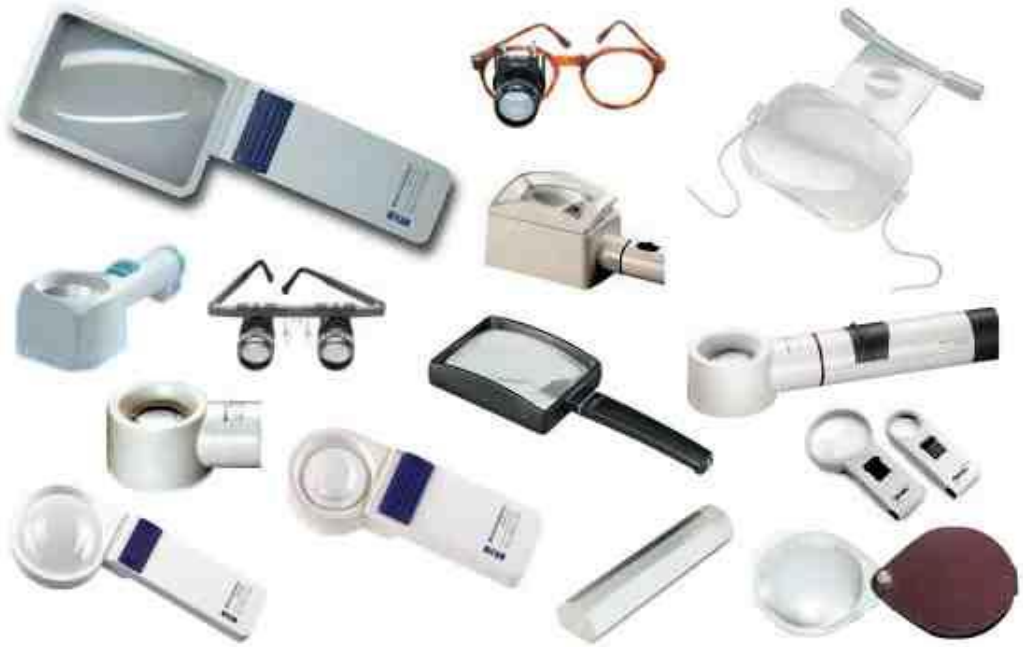
# Treatment

Treat the cause

Gene therapy emerging

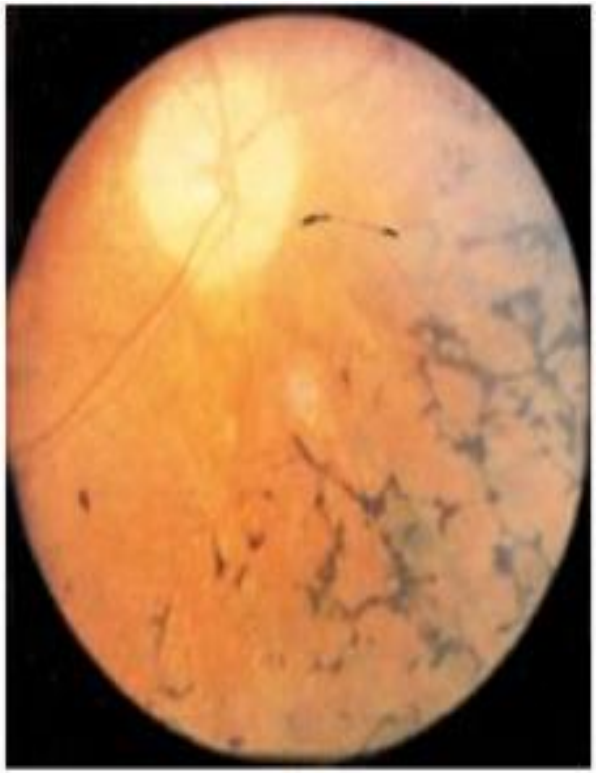
Community bas rehabilitation

bilateral cases as prognosis is poor



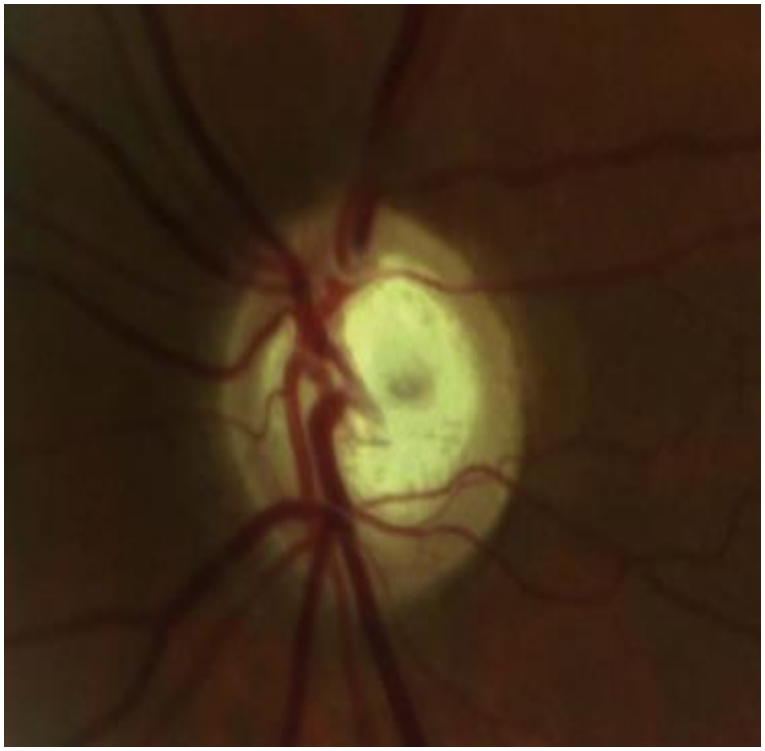
Low-vision aids

# Question



- The photograph belongs to a 12 year old boy who has difficulty in seeing at night.
- Can you identify the disc abnormality in the photograph?
- What condition does he suffer from?

# Question



- The disc in question is of a 60 year old myope who is instilling timolol eye drops since the past 5 years.
- Is this a normal optic disc?


Thank you