

Retinal Detachment

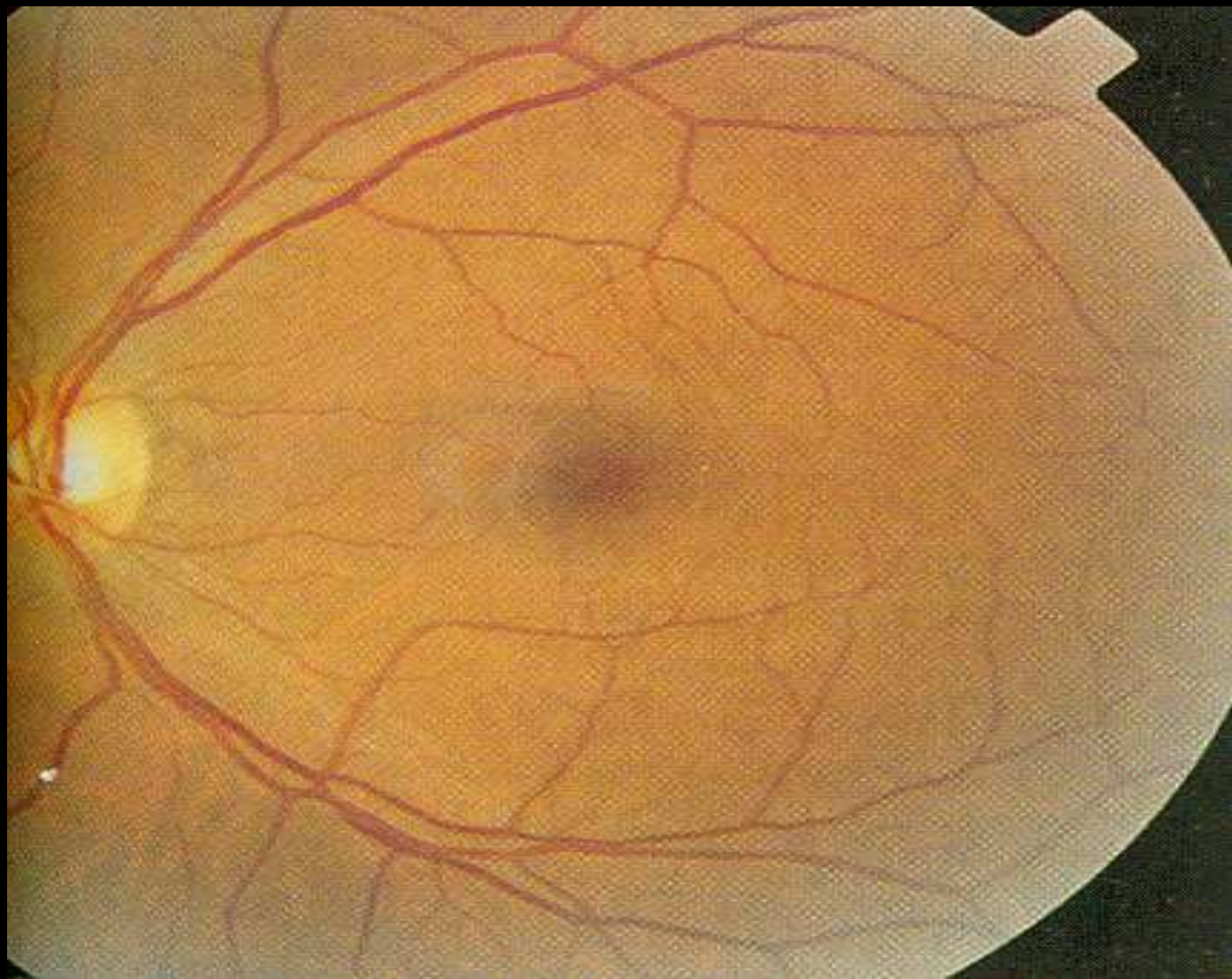
Dr.R.P.Gupta

Retina

- Inner most coat of Eye
- Extends from optic disc to ora serrata
- Highly sensitive part – Macula

Microscopic-

- Pigment Epithelium
- Neurosensory retina(10 Layers)
- Potential space between two layers



RETINAL DETACHMENT

- Separation of Neuro sensory retinal from pigment epithelium
- Two types – Primary (Rhegmatogenous)
 - Secondary
 - Tractional
 - Exudative

RETINAL DETACHMENT

Rhegmatogenous Retinal Detachment

Factors responsible

- Usually due to hole in the retina
- Vitreous Degeneration
- Vitreous Detachment

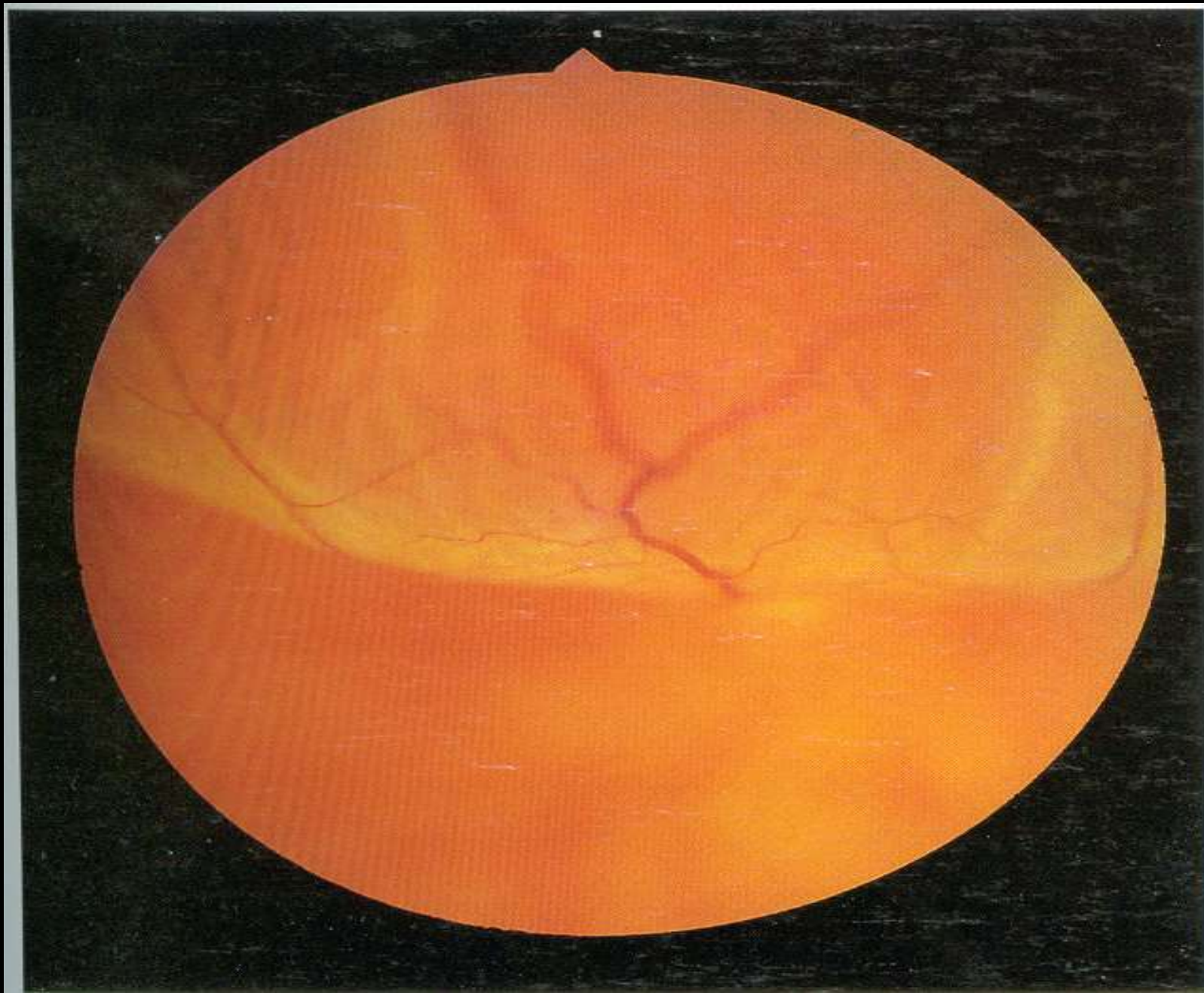
RETINAL DETACHMENT

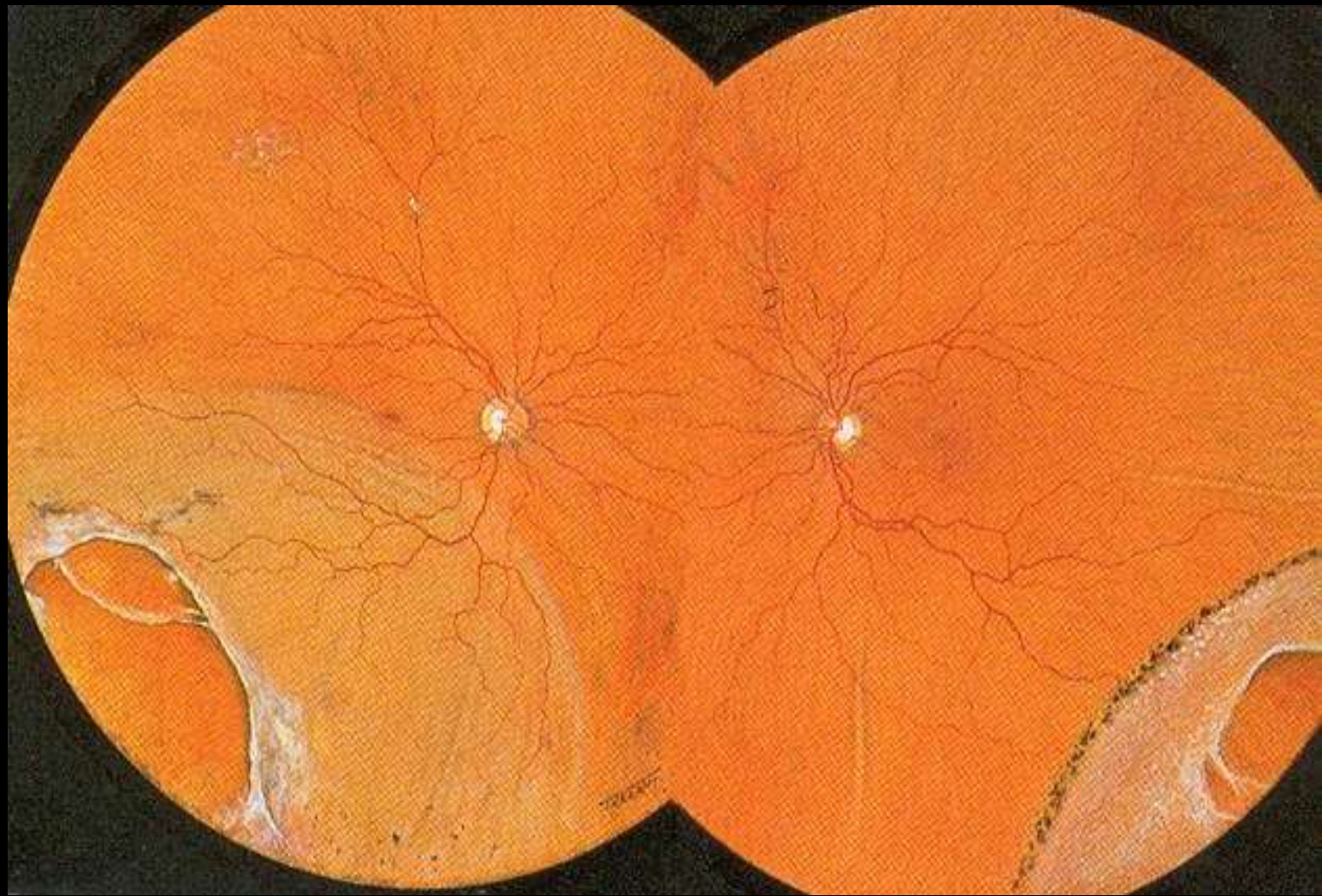
Symptoms

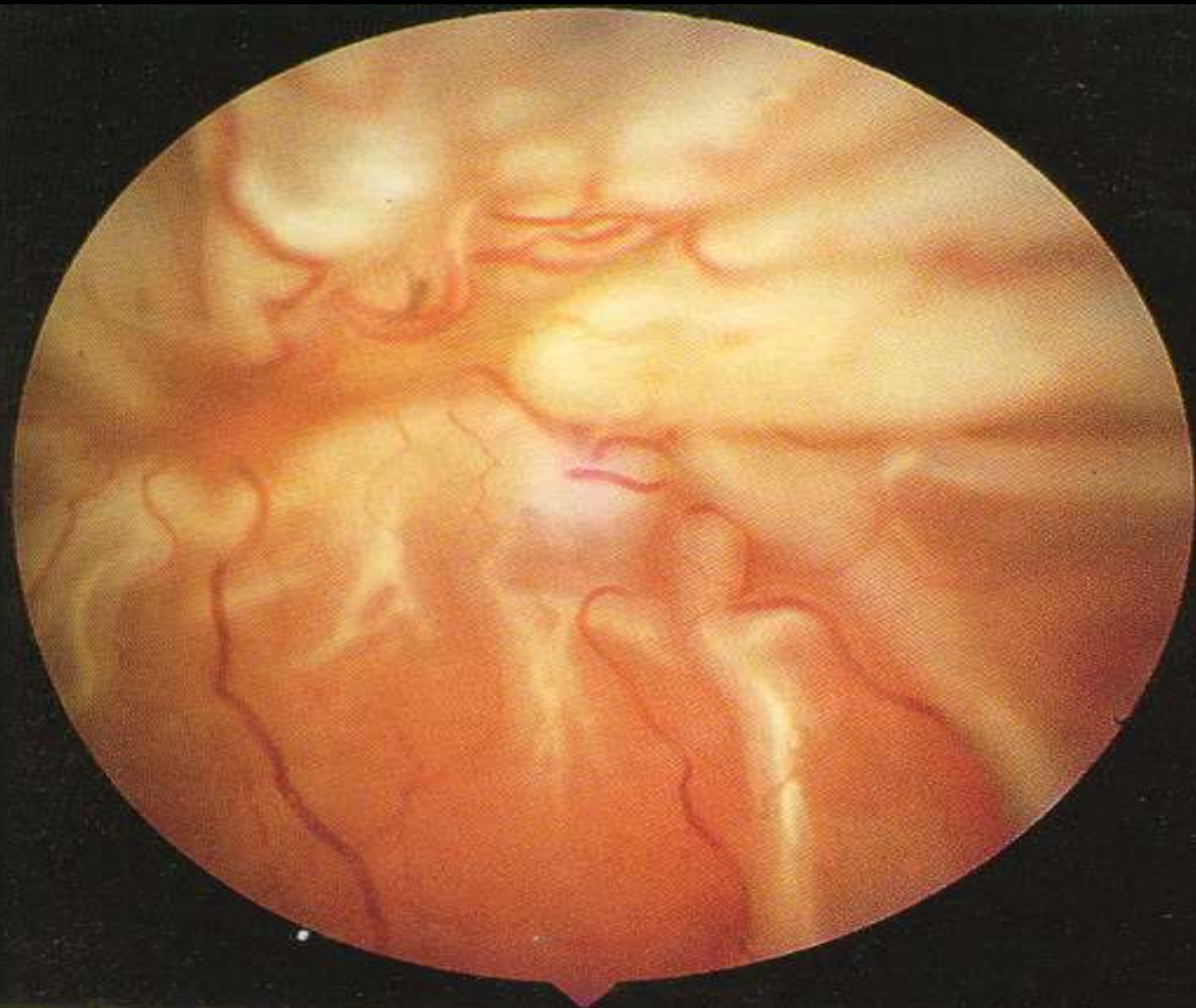
- Flashes of Light(Photopsia)
- Floaters
- Cloud / Veil in the field of vision
- Sudden painless diminution of vision

Retinal Detachment

- Vision grossly diminished
- Detached retina appears greyish white, ballooned(Convex) , shows undulation on movement
- Retinal vessels appears dull
- Retinal hole may be seen







Retinal Detachment

- Detailed examination of Fundus done with the help of Indirect Ophthalmoscope
- Routine investigation for surgery

Retinal Detachment

- Treatment is always surgical
- Treatment should be early
- Delay causes damage to macula and reduces visual recovery

RETINAL DETACHMENT

Treatment

- Cryopexy
- Sceral Buckling
- Sub Retinal Fluid Drainage

RETINAL DETACHMENT

Traction Retinal Detachment

- Detachment of Retina due to pull by fibro glial bands
 - Proliferative Diabetic Retinopathy
 - Retinal Vasculitis
- Detached retina is shallow, concave
And fixed
- Fibrovascular bands seen



Traction Retinal Detachment

Treatment

- Vitrectomy, cutting of bands
- Endolaser
- Internal Tamponade
- Silicon oil

Exudative Retinal Detachment

Aetiology

Choroiditis

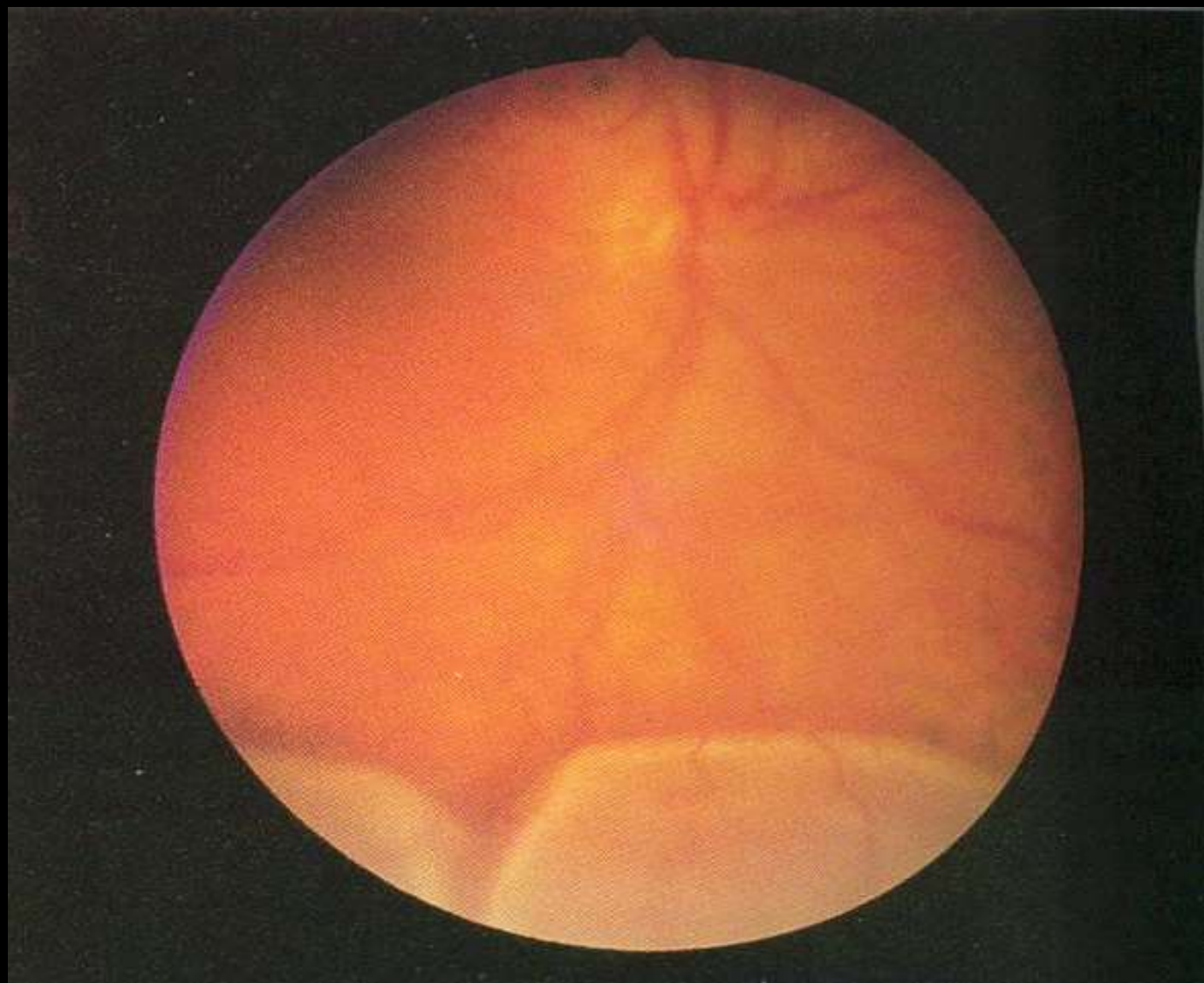
Tumours

Toxaemia of Pregnancy

Coats Disease/ Angiomatosis

- Detached retina is elevated, smooth surface with shifting fluid
- Local causes like tumours/ angiomatosis can be seen

Treatment – Treatment of cause



RETINITIS PIGMENTOSA



RETINITIS PIGMENTOSA

- ◆ Degenerative condition of Retina
- ◆ Hereditary condition
 - Autosomal Recessive- common
 - Autosomal Dominant
 - X Linked –least common
- ◆ Usually Bilateral
- ◆ Starts in childhood



RETINITIS PIGMENTOSA

- ◆ Primarily affects Rods
- ◆ Starts at equator
- ◆ Spreads anteriorly & posteriorly
- ◆ Macula affected last

Presenting Symptoms-

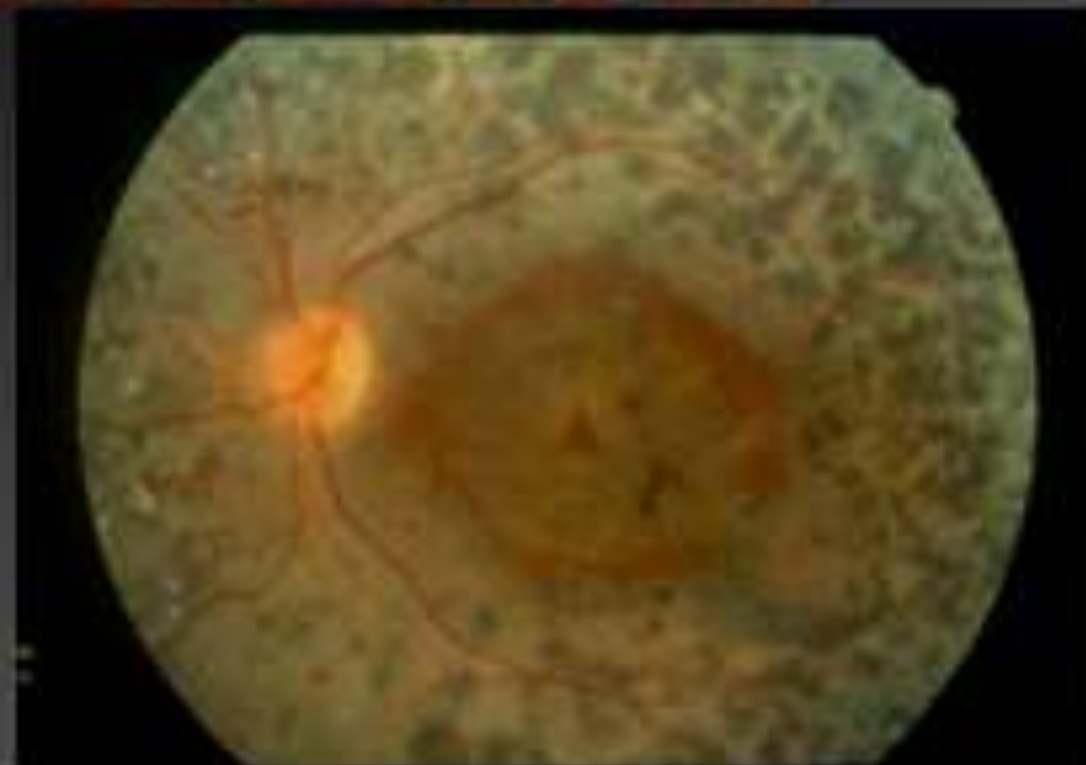
Night Blindness

RETINITIS PIGMENTOSA

Clinical Feature

- ◆ Anterior Segment – Normal
- ◆ Fundus Exam shows – Jet Black Bone
Corpuscular pigmentation in
equatorial region
- ◆ Arteriolar attenuation
- ◆ Waxy pallor of disc

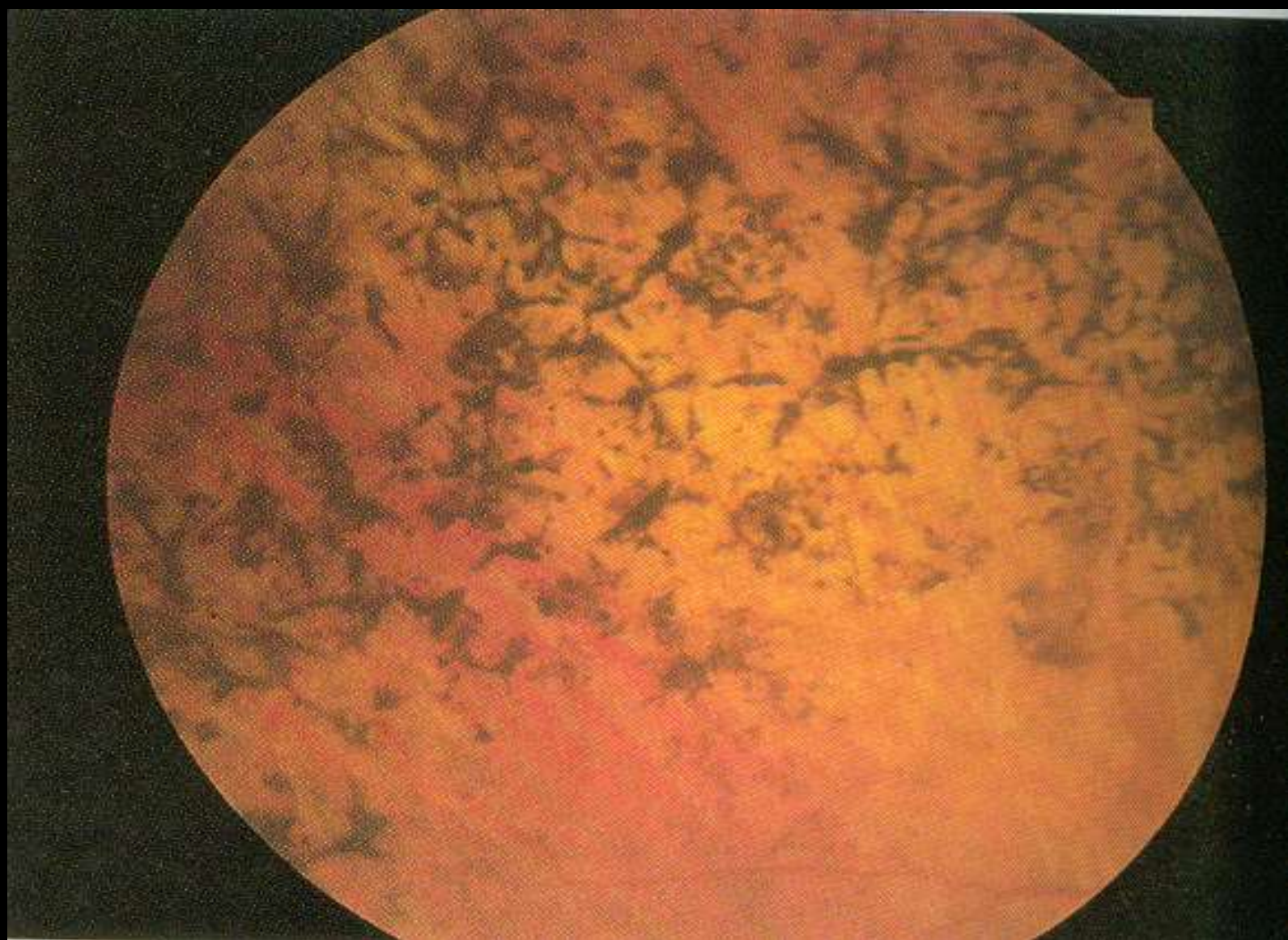
Retinitis Pigmentosa



Note the typical features of RP

- *Waxy pallor of disc
- *Attenuation of arterioles
- *Area of RPE hyperpigmentation that alternate with atrophic regions in the form of bone spicules





RETINITIS PIGMENTOSA

◆ Perimetry –

Ring Scotoma

Peripheral constriction of Fields

Tube vision

◆ Electroretinogram- Subnormal



RETINITIS PIGMENTOSA

◆ Complication-
Maculopathy
Cataract

Open angle Glaucoma- usually
associated

A silhouette of a runner in a starting block, positioned in the lower-left quadrant of the slide. The runner is in a crouched starting position, with their hands on the ground and feet in starting blocks. The background is a warm orange gradient with curved lines.

RETINITIS PIGMENTOSA

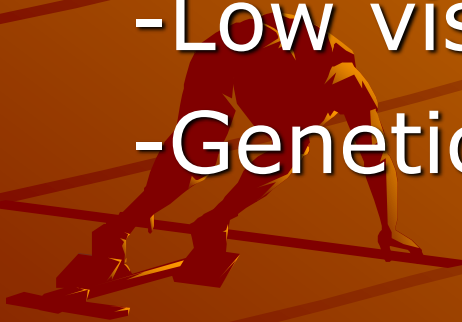
✦ Treatment

No specific treatment available

-Visual Rehabilitation

-Low vision aids

-Genetic Counseling



RETINAL VEIN OCCLUSION

- Most common vascular disorder

Aetiology

- Increased Blood Viscosity
 - Leukemia, Polycythemia
 - Macroglobulinaemia
 - Oral contraceptive pills



RETINAL VEIN OCCLUSION

■ Disease of Vessel wall-

Vasculitis

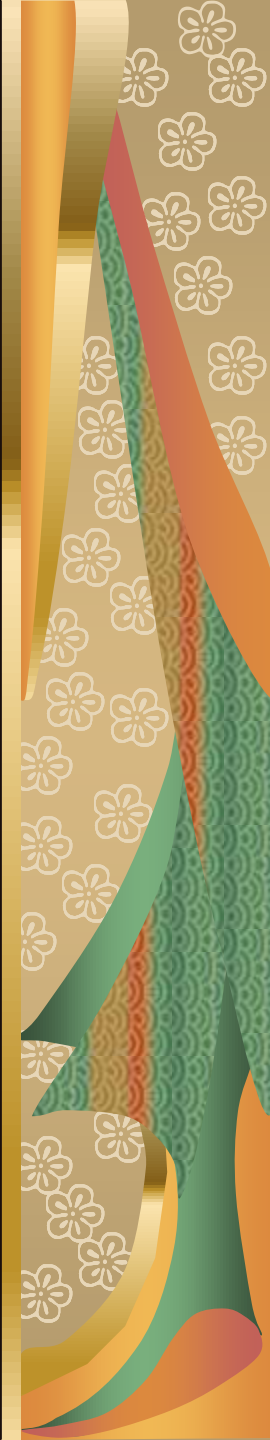
Eales Disease

■ Pressure over veins from outside

Arteriosclerosis

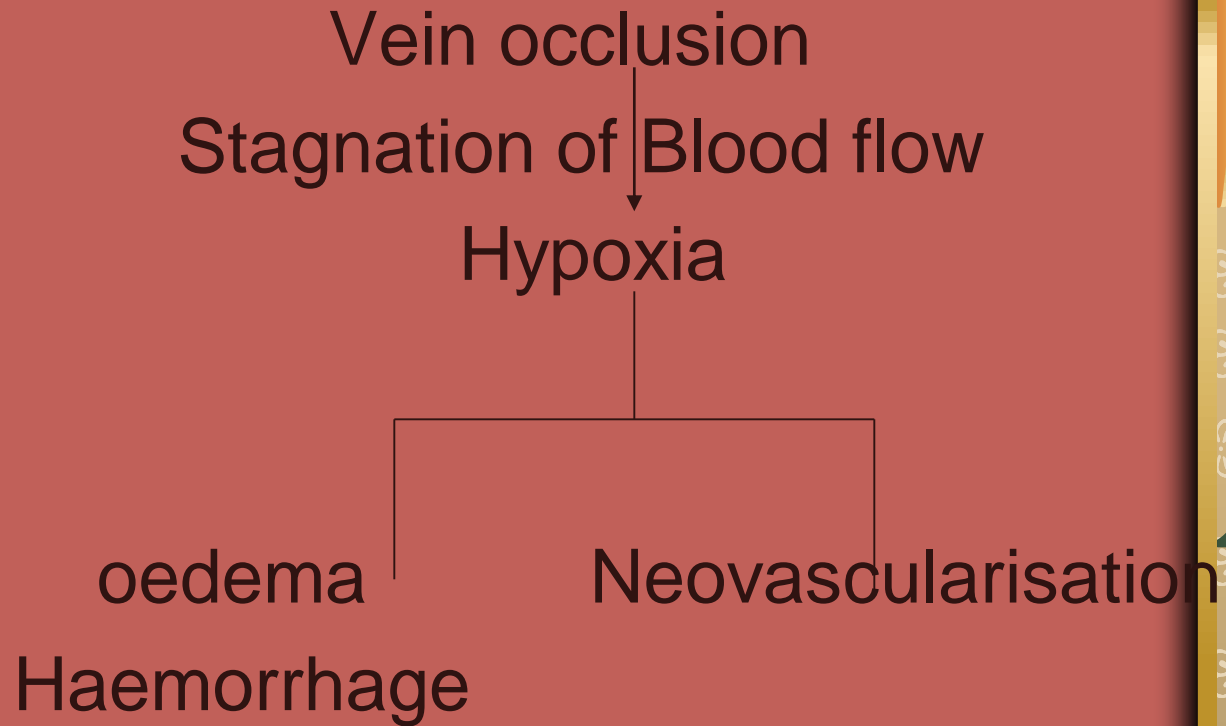
Hypertension

Open angle glaucoma



RETINAL VEIN OCCLUSION

■ Pathophysiology



RETINAL VEIN OCCLUSION

- Clinical Features
- Two Types-
- Central Retinal Vein Occlusion
- Branch Retinal Vein Occlusion



RETINAL VEIN OCCLUSION

Clinical Features

Sudden Gross Diminution of vision

RAPD may be present

Fundus Exam :-

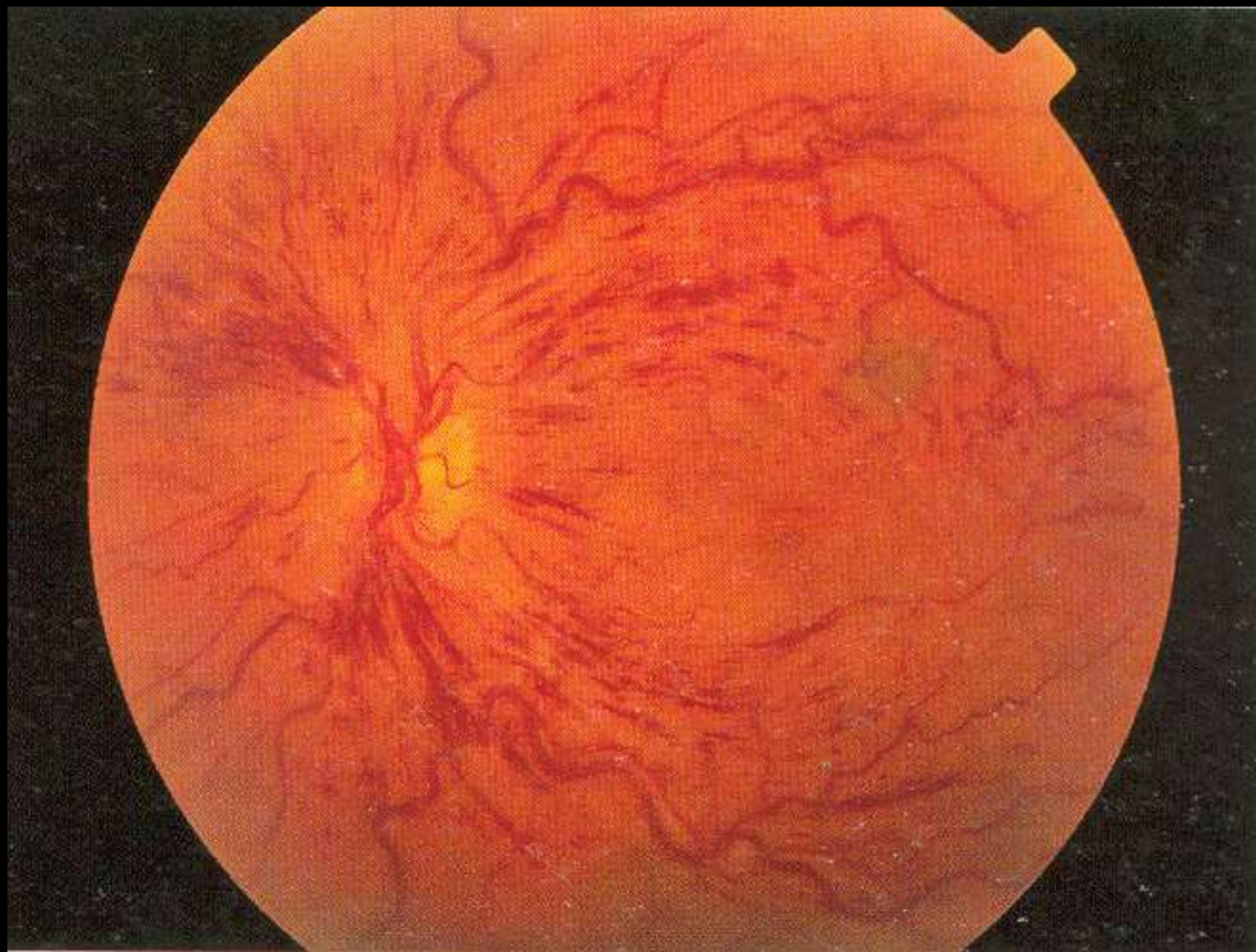
Optic Disc Congested, Margins Blurred

Veins engorged & Tortuous

Multiple Retinal Haemorrhages

cotton wool spots





RETINAL VEIN OCCLUSION

Clinical Features (BRVO)

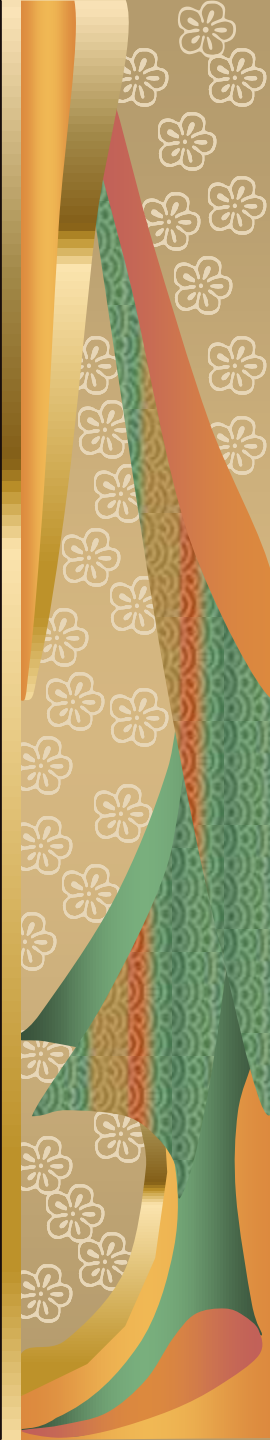
Changes Limited to area drained by the obstructed vein

Occlusion of Upper Temporal Branch is more common



RETINAL VEIN OCCLUSION

- Investigation
- Recording of Blood Pressure
- Recording of IOT
- Haemogram
- Blood Sugar estimation
- STS & Mantoux Test in young patients



RETINAL VEIN OCCLUSION

Complications

Neovascularisation of Retina

Rubeosis iridis

Neovascular Glaucoma

Vision affected due to

Macular oedema

Vitreous Haemorrhage



RETINAL VEIN OCCLUSION

Treatment:-

Treat the predisposing conditions

In young patient a course of steroids if vasculitis is suspected

Wait for 6 – 8 weeks

Pan Retinal Photocoagulation If NVE / NVD occurs

Macular Grid – Macular oedema



CENTRAL RETINAL ARTERY OCCLUSION

- Ocular Emergency
- Often leads to Blindness
- May be presenting symptom of systemic disease
- Central Retinal artery is an End artery
- Ganglion cells can withstand ischemia for only 5 min

CENTRAL RETINAL ARTERY OCCLUSION

Aetiology

Embolization – Heart/carotid artery

Vaso –obliteration

Giant Cell Arteritis

Takayasu disease

Polyarteritis Nodosa

Pressure from Outside

Increased IOT/ Pressure over
Globe

CENTRAL RETINAL ARTERY OCCLUSION

Clinical Features-

Sudden painless loss of vision in affected eye

No perception of light

Afferent Pupillary defect

CENTRAL RETINAL ARTERY OCCLUSION

Fundus Exam-

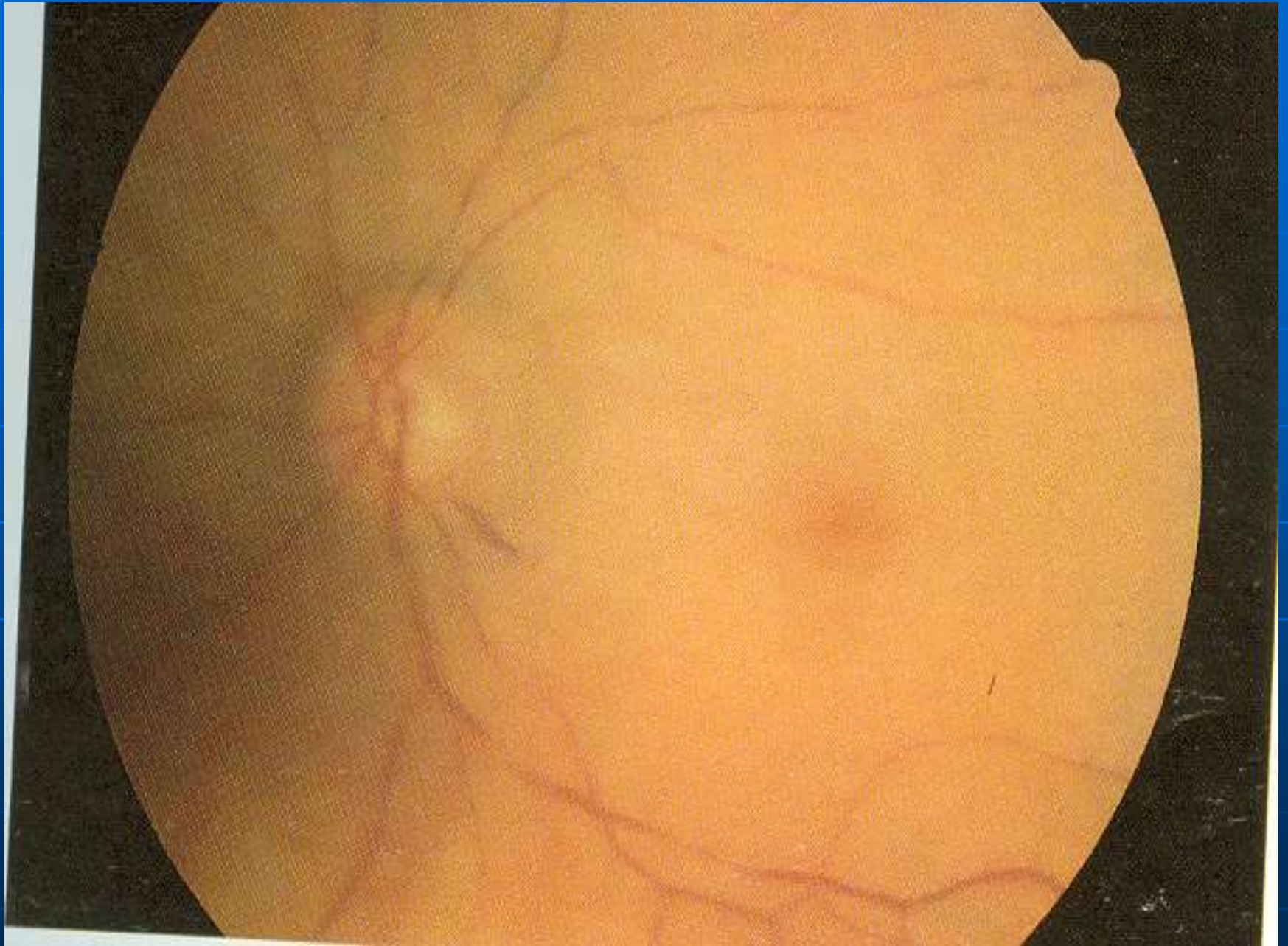
Retina oedematous, looks white

Cherry spot at Fovea

Marked narrowing of retinal arterioles

Segmentation of Blood column on pressure over globe

End result – Optic atrophy



CENTRAL RETINAL ARTERY OCCLUSION

Treatment

Ocular Massage

IV Acetazolamide 500mg

Inhalation of 95%O₂ + 5% CO₂

Paracentesis

CENTRAL RETINAL ARTERY OCCLUSION

- All patients with CRAO should be investigated fully
- Lipid Profile
- Carotid artery doppler
- Examination of Heart – Echo
- Temporal Artery biopsy
- ESR

DIBETIC RETINOPATHY

- Microangiopathy affecting retinal precapillary arterioles , capillaries and venules

DIBETIC RETINOPATHY

- Approximately 2% of all Diabetics become blind
- Incidence blindness is 20 times greater in Diabetics
- Incidence of Diabetic Retinopathy is related to duration Diabetes Usually occurs after 15 – 20 years of Diabetic age

DIBETIC RETINOPATHY

Pathogenesis

Microvascular Occlusion

Thickening of Capillary Basement Mn

Endothelial damage & proliferation

Changes in RBCs

Increased stickiness of Platelets

AV Shunts

Neovascularization

DIBETIC RETINOPATHY

Pathogenesis

Microvascular Leak

(Loss of Pericytes)



Retinal Haemorrhage

Retinal Oedema

Hard exudates

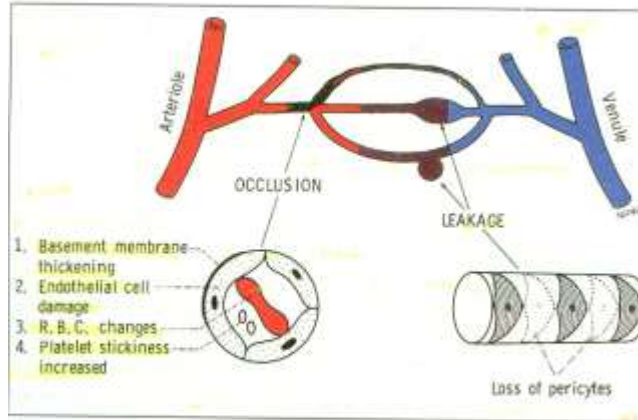


Figure 12.1 Pathogenesis of diabetic retinopathy

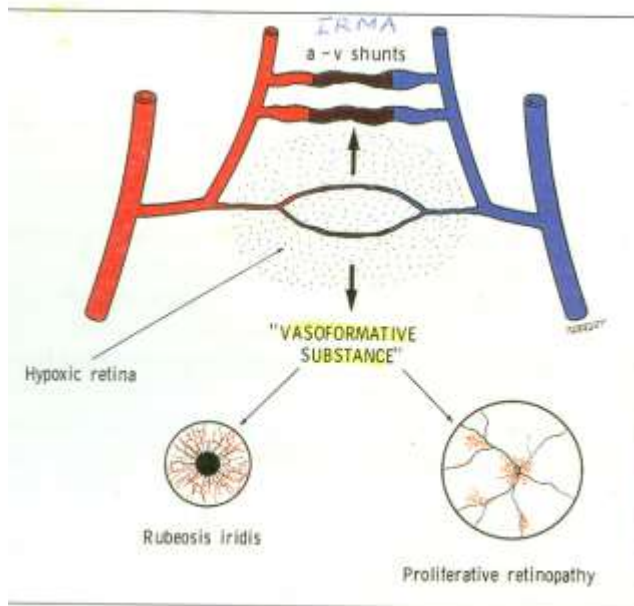
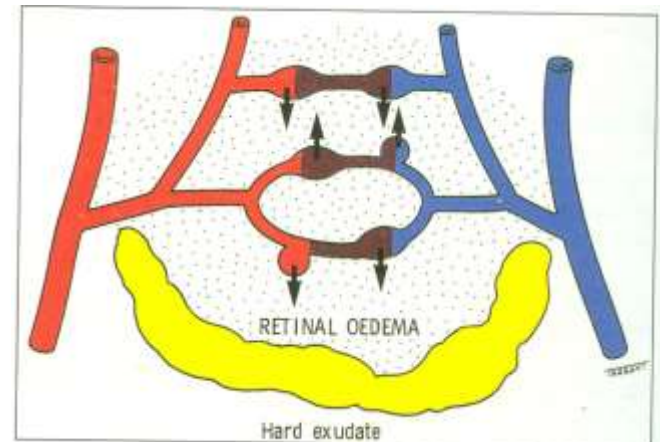


Figure 12.2 Consequences of retinal ischaemia in diabetic retinopathy



DIBETIC RETINOPATHY

Classification:-

- Non Proliferative Diabetic Retinopathy
(Mild, Moderate & Severe)
- Proliferative Diabetic Retinopathy
- Diabetic Maculopathy
- Advanced Diabetic Eye disease

Non Proliferative Diabetic Retinopathy

Clinical Features –

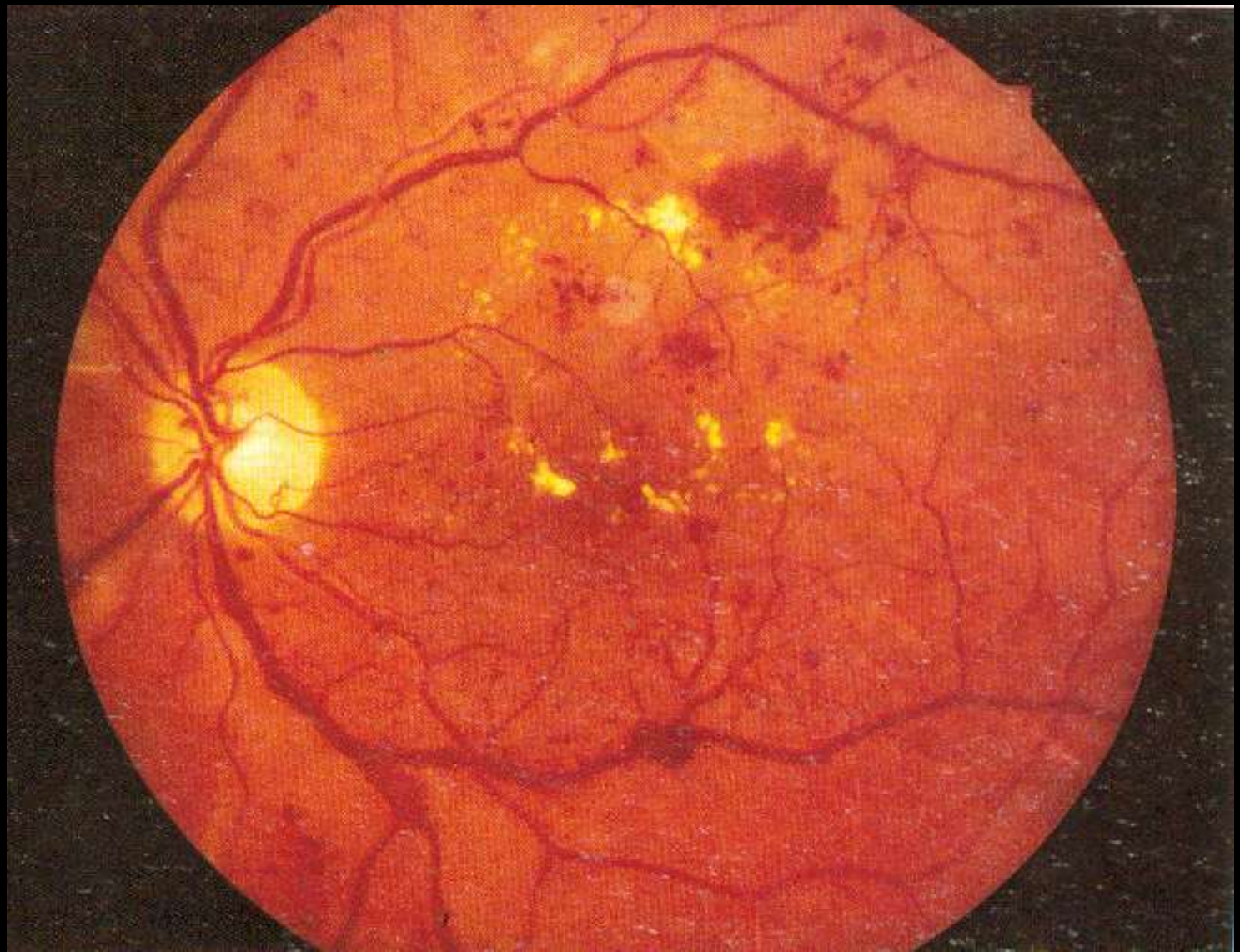
Microaneurysms

Dot & Blot Haemorrhages

Hard Exudates

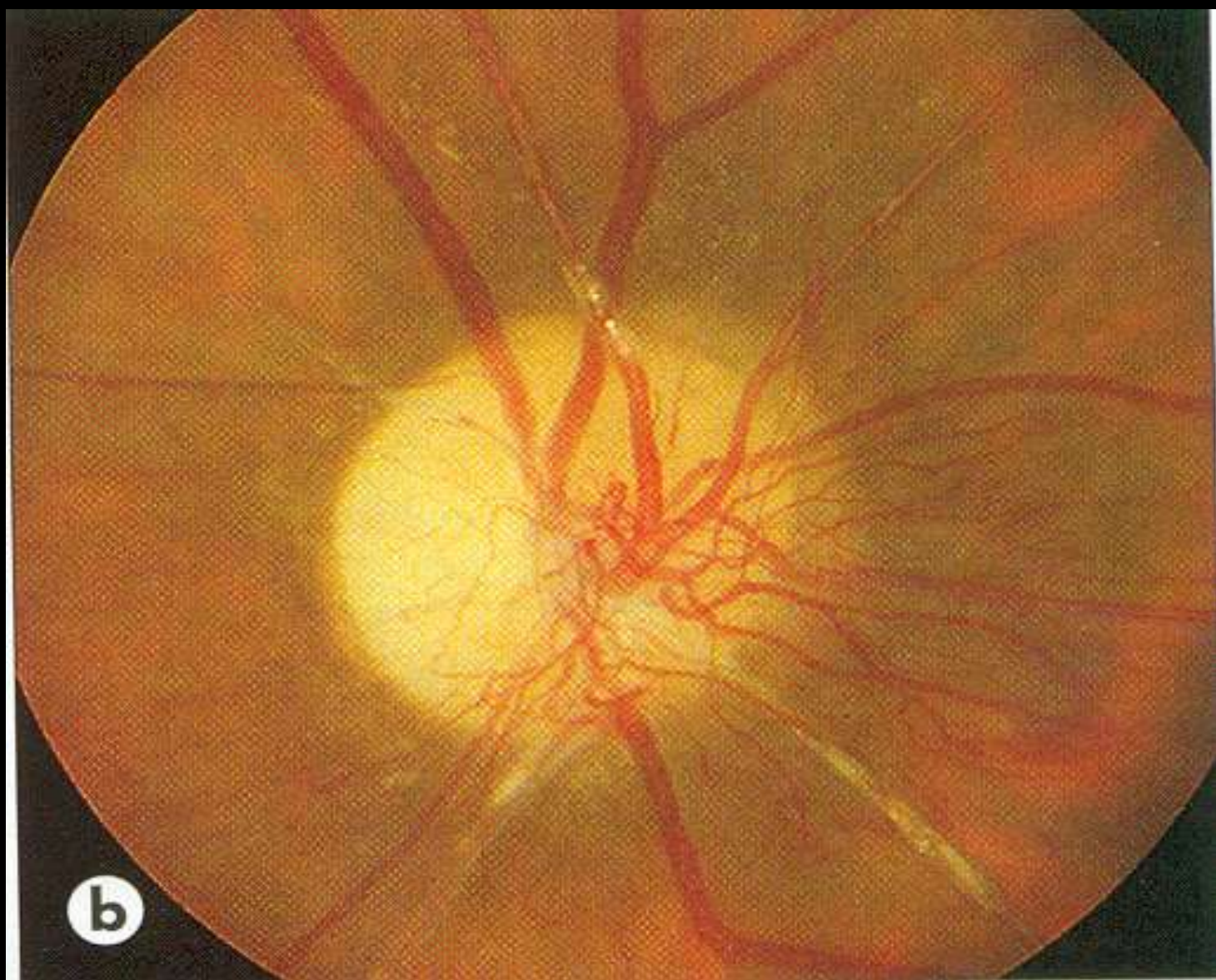
Flame Shaped Haemorrhages} In severe

Soft Exudates } cases



Proliferative Diabetic Retinopathy

- Characterized by Neovascularisation
- NVD / NVE



Diabetic Retinopathy

Complication:-

Vitreous Haemorrhage

Traction Retinal Detachment

Neovascular Glaucoma

Called as Advanced Diabetic Eye Disease

Diabetic Retinopathy

Treatment

NPDR

Good Diabetic control

Regular Fundus examination

Macular Grid photocoagulation for macular oedema

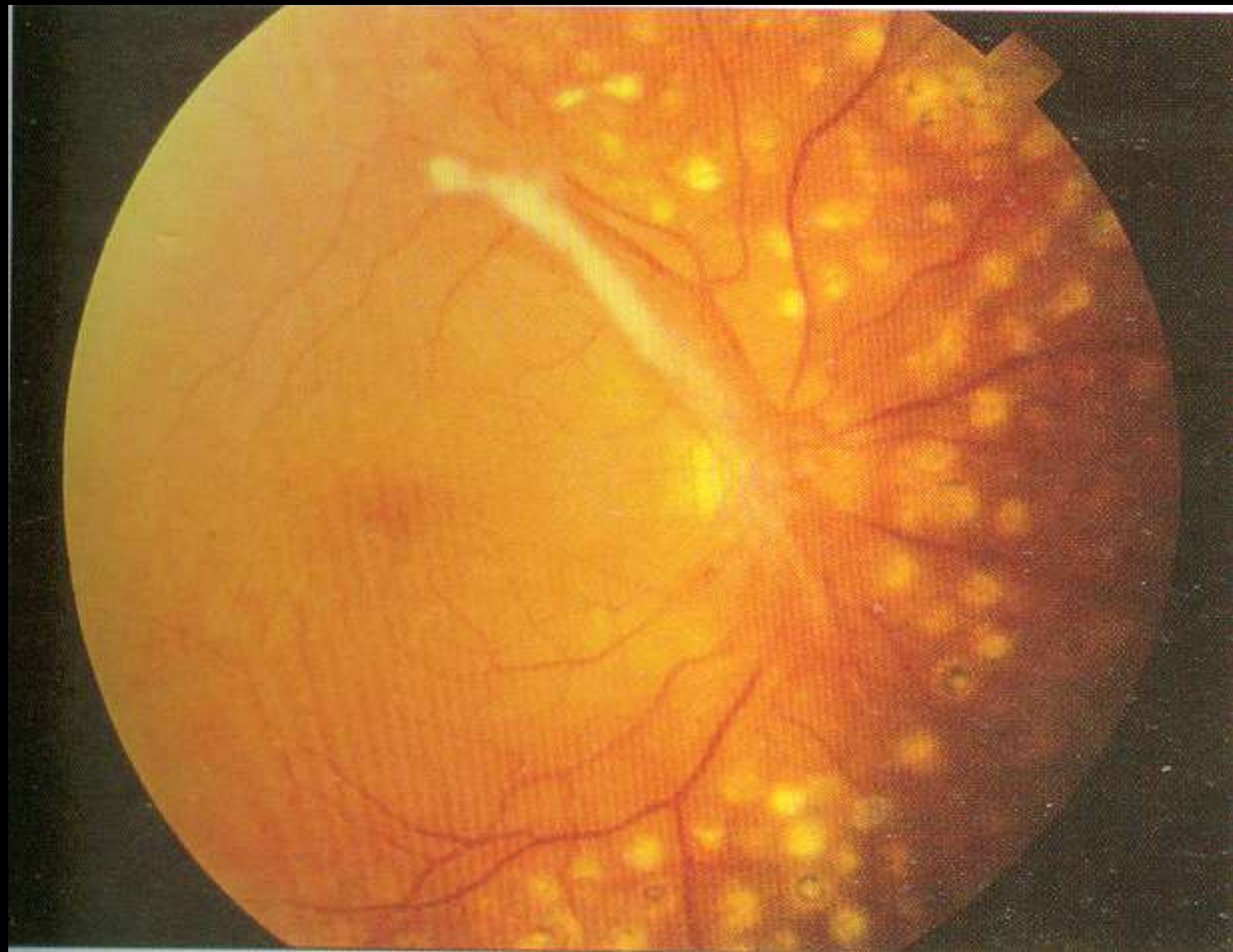
Diabetic Retinopathy

Treatment

Proliferative Diabetic Retinopathy

Pan Retinal Photocoagulation

Treatment of vitreous haemorrhage/
traction RD – Vitreo Retinal surgery



HYPERTENSIVE RETINOPATHY

- ◆ Central Retinal artery is a branch of ophthalmic artery which is a branch of Internal Carotid artery
- ◆ Mirrors the cerebral circulation
- ◆ Medium sized artery which can be visualized

HYPERTENSIVE RETINOPATHY

- ◆ Response of arteries to increased blood pressure
- ◆ In young individual , recent hypertension
- ◆ Spasm of arterioles
- ◆ Long standing Hypertension - Arteriosclerosis

HYPERTENSIVE RETINOPATHY

Fundus Picture

- ◆ Generalized narrowing & Focal Narrowing of arterioles
- ◆ Flame shaped haemorrhages
- ◆ Hard Exudates – Macular star
- ◆ Cotton wool spots
- ◆ Papilloedema – Malignant hypertension
- ◆ Ischemic Choroidal Infarct- Elschnigs spots



HYPERTENSIVE RETINOPATHY

Arteriosclerotic changes

- Broadening of light reflex
- Arterio venous crossing change
 - Concealment of vein-
 - deflection of vein- Salus sign
 - Banking of vein- Bonnet sign
 - Tapering of veins on either side- Gunns sign
- Copper wire arteries
- Silver wire arteries

HYPERTENSIVE RETINOPATHY

Classification

Keith Wagner Classification

- Grade- I Generalised narrowing of arterioles
Concealment of Veins
- Grade-II Focal narrowing of arterioles
Deflection of Veins- Salu's sign
- Grade-III Marked AV crossing changes
Copper / silver wire arteries
Retinal haemorrhages, Hard exudates
Cotton wool spots
- Grade – IV Papilloedema

HYPERTENSIVE RETINOPATHY

Classification (Scheie's Classification)

Hypertensive Retinopathy

Grade- I Generalised narrowing of arterioles

Grade-II Focal narrowing of arterioles

Grade-III Retinal haemorrhages, Hard exudates

Soft exudates (Cotton wool spots)

Grade – IV Papilloedema

HYPERTENSIVE RETINOPATHY

Arteriosclerotic changes

- | | |
|----------------------|--|
| Grade – I | Increased of light reflex
Concealment of vein |
| Grade – II
change | Marked AV crossing |
| Grade – III | Copper wire arteries |
| Grade - IV | Silver wire arteries |

HYPERTENSIVE RETINOPATHY

- ◆ Complications – BRVO/ CRVO
- ◆ Treatment – Good control of Hypertension

Retinopathy of Toxemia of Pregnancy

- ◆ Characteristic Hypertensive retinopathy found in cases of PIH
- ◆ Usually occurs at 6-9 months of Pregnancy
- ◆ May lead to blindness

Retinopathy of Toxemia of Pregnancy

Clinical Features

- ◆ Narrowing of Retinal arteries
- ◆ Retinal haemorrhages & Exudates
- ◆ Marked retinal oedema
- ◆ Exudative retinal Detachment

Retinopathy of Toxemia of Pregnancy

Treatment

- ◆ Control of hypertension
- ◆ Severe Retinopathy – Termination of Pregnancy

Albuminuritic Retinopathy

- ◆ Found in cases of Renal failure
- ◆ Characterized by more of soft exudates than hard exudates
- ◆ Macular star is a common feature

OPTIC NEURITIS

OPTIC NEURITIS

- Inflammation of Optic Nerve
- Clinically two types :-
 - Papillitis- Inflammation of Optic Nerve Head
 - Retobulbar Neeuritis- Inflammation of Optic Nerve Behind the globe

OPTIC NEURITIS

Aetiology :-

Multiple Sclerosis

Herpes Zoster Infection

Poliomyelitis

Encephalitis

Local – Uveitis

Meningitis'

Orbital Cellulitis

Sinusitis

OPTIC NEURITIS

Aetiology (contd)

- Endogenous – Acute Infectious Disease
(Measles , Mumps)
- Metabolic – Diabetes
Anaemia
Thiamine Deficiency

OPTIC NEURITIS

- Symptoms-

Sudden Onset

Rapid Deterioration of Vision

Pain on moving the Eye

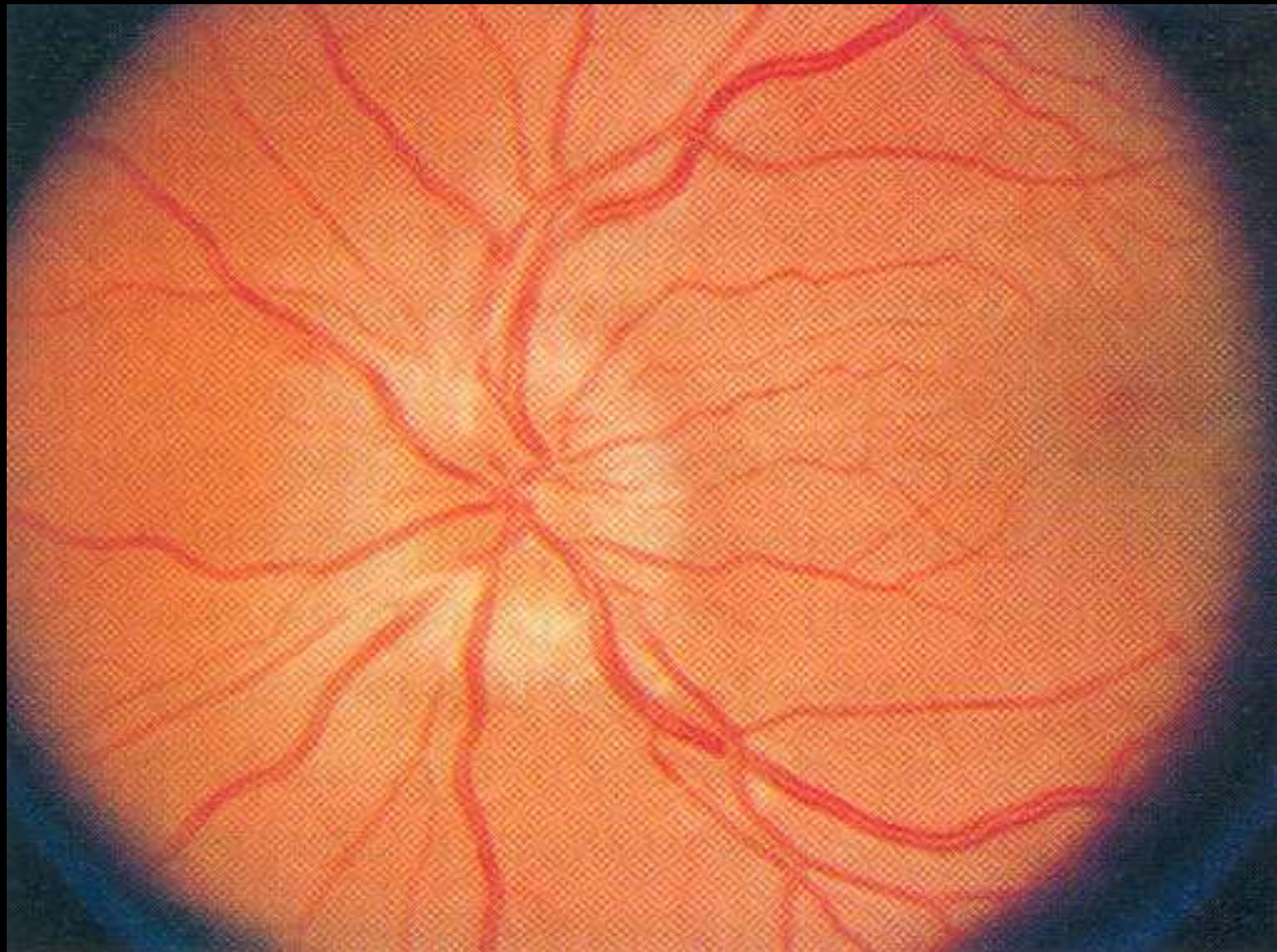
OPTIC NEURITIS

- Signs

- Vision grossly diminished
- Tenderness over insertion of superior Rectus Muscle
- Illusutained pupillary Reaction to Light
- Some cases Mracus Gunn pupil

OPTIC NEURITIS

- Signs (Contd)
- Fundus (In Papillitis)
 - Disc Hyperaemic
 - Margins Blurred
 - Oedema of surrounding Retina
 - Macular Fan
- ***Fundus Normal in cases of Retrobulbar Neuritis***



OPTIC NEURITIS

Investigation;-

- Perimetry-
 - Central Scotoma
 - Peripheral Constriction of Fields
- Visual Evoked Potential
 - Increased Latency

OPTIC NEURITIS

Treatment:-

- Pulse Steroid Therapy-

 - Methyl prednisolone

 - 250 mg IV x 6 hrly For 72 hrs

 - Followed by Oral Steroids for 2 weeks

- In Optic neuritis of Infective aetiology

 - Treatment of the cause

PAPILLOEDEMA

- Oedema of Optic Nerve Head (Optic Disc)
- Purely Hydrostatic , Non Inflammatory

Pathophysiology

- Axoplasmic Stasis
- Compression of Central Retinal vein

PAPILLOEDEMA

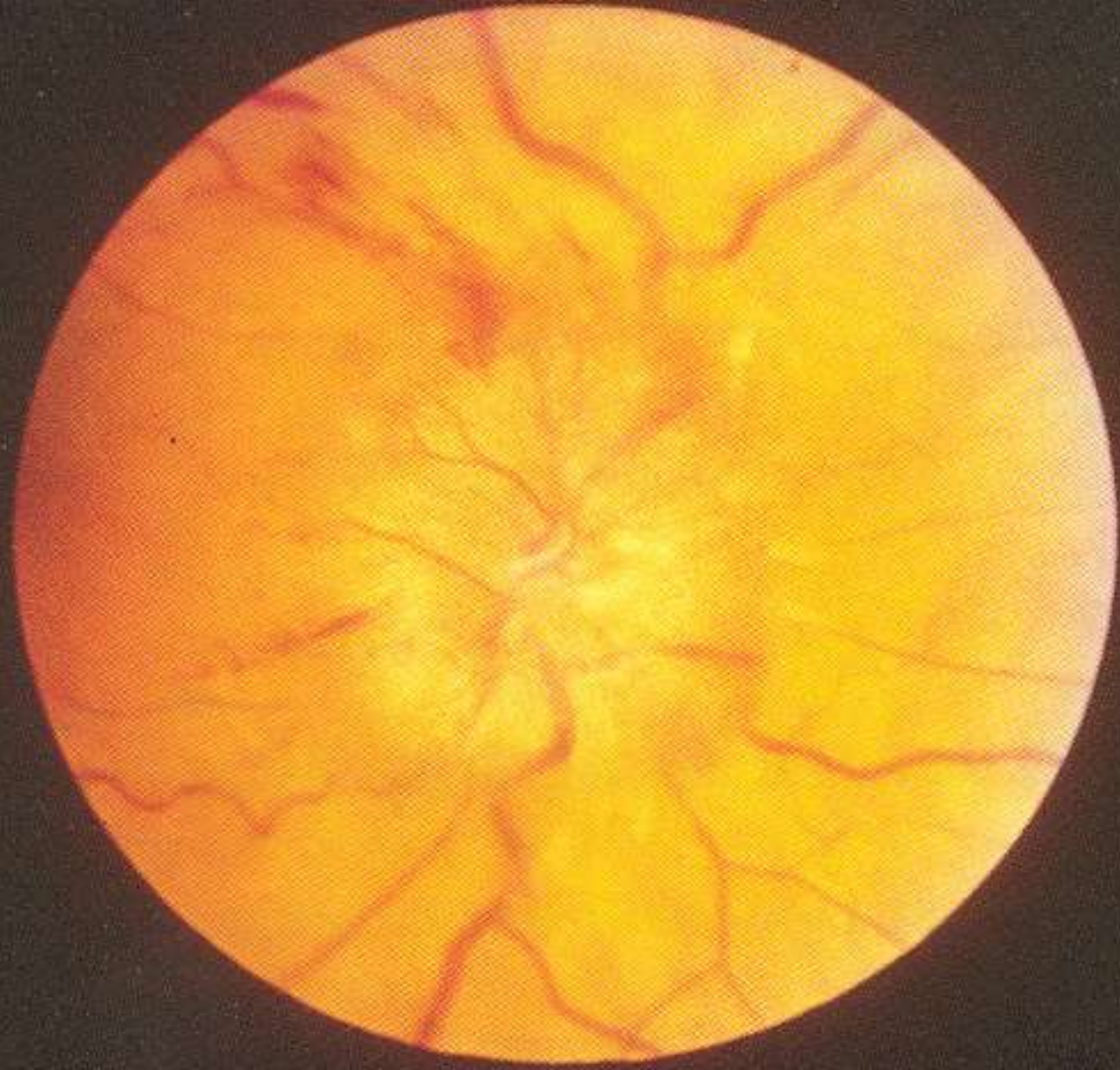
- Aetiology
 - Raised Intracranial Pressure
 - Intracranial Tumour
 - Cerebral Abscess
 - Cavernous Sinus Thrombosis
 - Subarachnoid Haemorrhage
 - Malignant Hypertension
 - Benign Intracranial Hypertension

PAPILLOEDEMA

- Clinical Picture
- Asymptomatic
- May complain of Transient Blurring of Vision
- Vision is Normal
- Anterior Segment Normal

PAPILLOEDMA

- Fundus- Blurring Of disc Margin
Congestion of Disc
Filling up of Physiological Cup
Retinal Veins Engorged
Venous pulsations absent
Haemorrhages over disc
Oedema of surrounding retina
Macular Fan



PAPILLOEDEMA

Differential Diagnosis

- Optic Neuritis
- Drusen of optic Disc
- Pseudoneuritis

PAPILLOEDEMA

Investigation

- Perimetry – Enlargement of Blind Spot
- CT Scan / MRI

PAPILLOEDEMA

Treatment

Treatment of Cause

TOXIC AMBLYOPIA

- Damage to the Optic Nerve due to exogenous toxins
- Tobacco Ethambutol
- Methyl Alcohol Lead
- Chloroquine Arsenic
- Quinine Oral Contraceptive

TOXIC AMBLYOPIA

Tobacco Amblyopia

- Common in heavy cigar smokers
- Symptoms – fogginess of Vision
- Vision diminished
- Fundus shows Temporal pallor
- Perimetry- Caeco Central Scotoma

TOXIC AMBLYOPIA

- Treatment
- Stop Smoking
- Inj B12 1000 μ g once in 5 days x 5 Inj

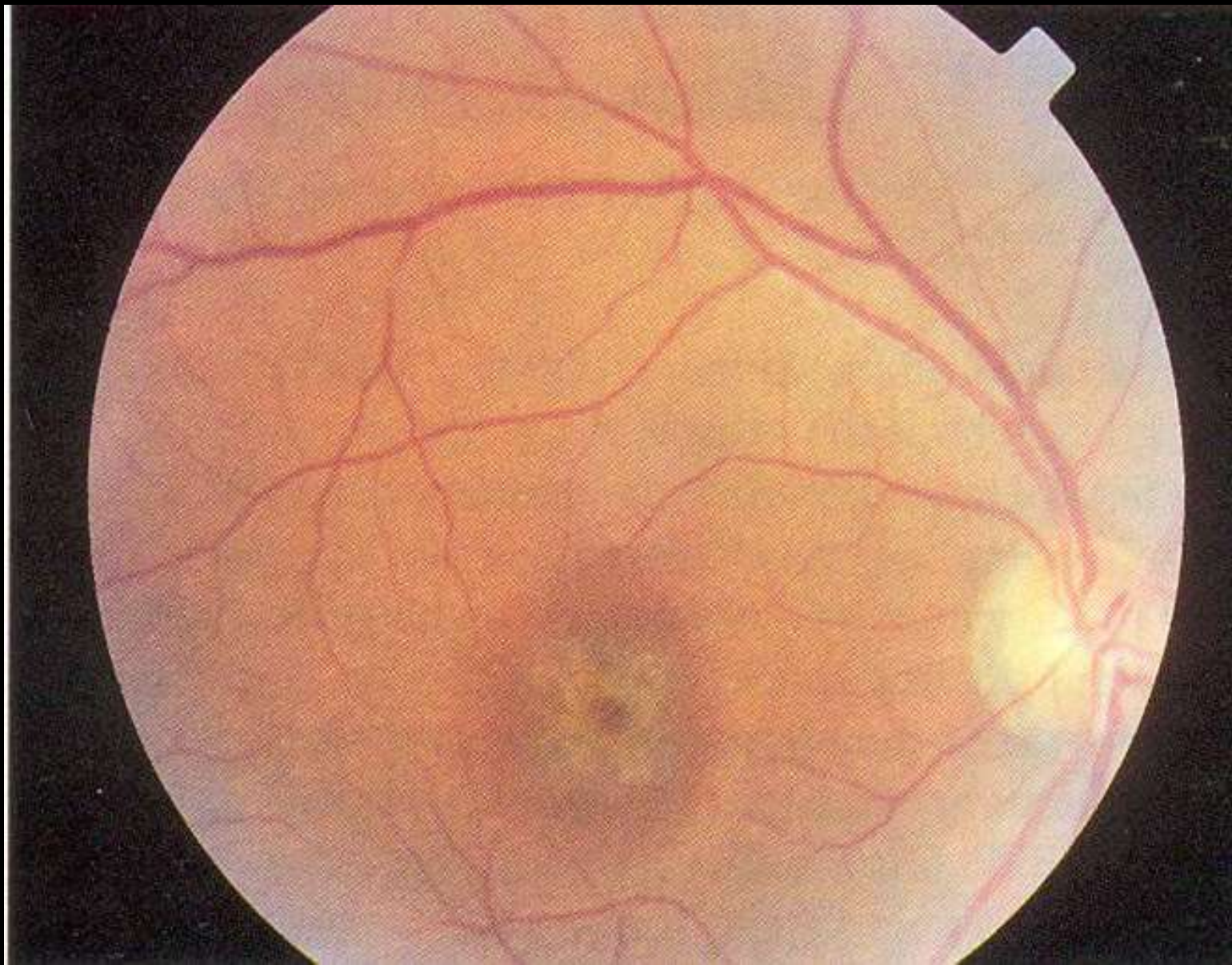
TOXIC AMBLYOPIA

- Methyl Alcohol
- Toxic to Ganglion cells
- Wide spread degeneration of ganglion cells
- Leads to Bilateral Optic Atrophy

TOXIC AMBLYOPIA

Chloroquine

- Seen in patients who are on long term Chloroquine therapy
- Initially pimentation at macula -Bulls Eye Maculopathy
- In advanced cases widespread Retinal degeneration similar to retinitis pigmentosa
- Optic Atrophy



TOXIC AMBLYOPIA

- Treatment
- Early cases recover if chloroquine therapy is stopped
- Advanced cases no effective therapy
- All cases who are on long term chloroquine therapy must undergo regular ocular check up
- Any sign of toxicity stop chloroquine
- Scotoma in central 5° field with red target

Optic Atrophy

- Degeneration of Optic Nerve

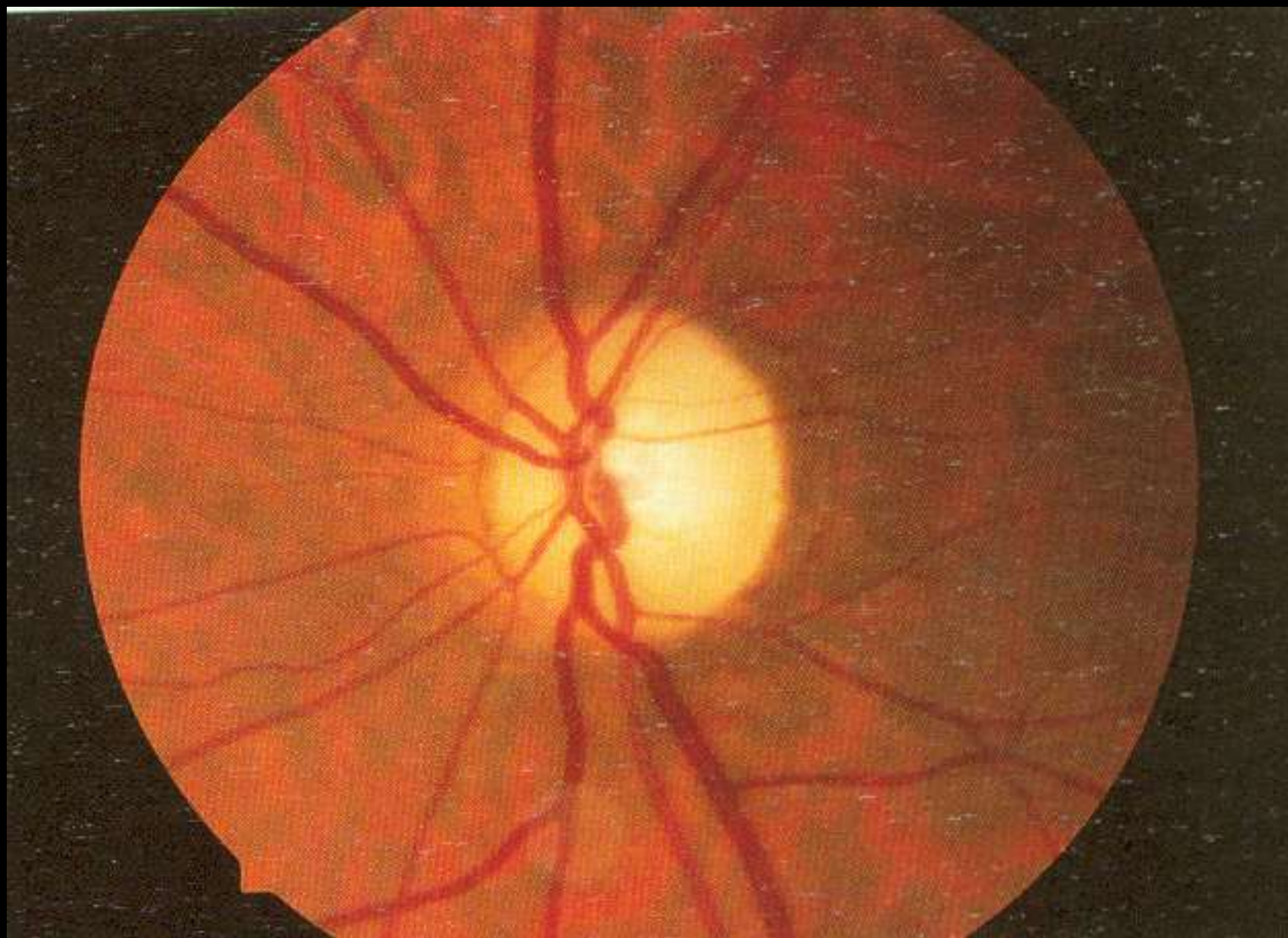
Classification

- Primary
- Secondary
- Consecutive
- Glaucomatous

Primary Optic Atrophy

Fundus picture

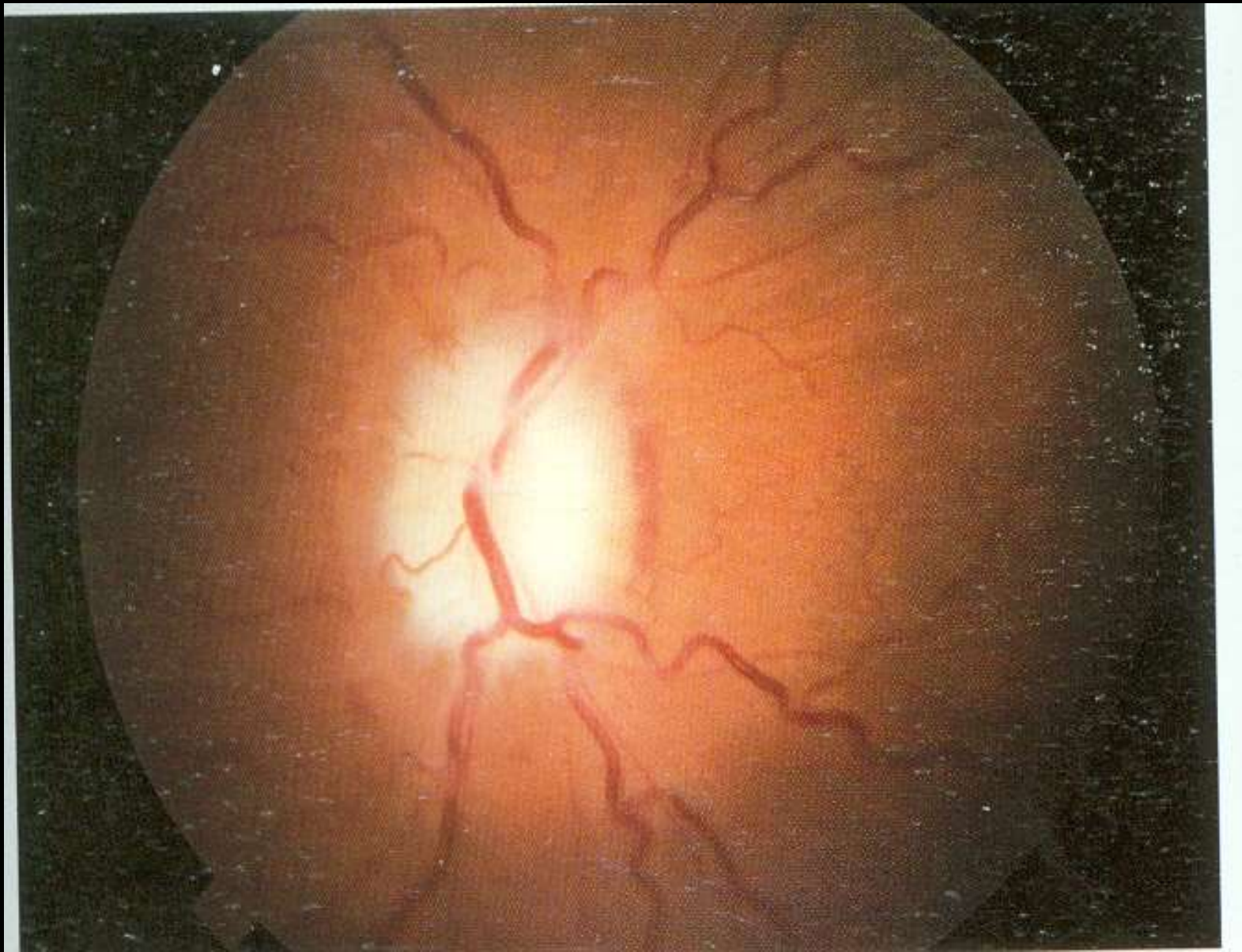
- Disc appears white
- Disc Margins well defined
- Physiological cup normal
- Blood vessels normal
- Aetiology
 - Pressure over Optic Nerve by Tumour
 - Meningitis
 - Retrobulbar Neuritis



Secondary Optic Atrophy

Fundus Picture

- Disc margin Blurred
- Disc appears dirty grey
- Physiological cup filled up
- Sheathing of vessels at disc
- Aetiology
- Optic Neuritis



Consecutive Optic Atrophy

- Optic Atrophy as a consequence of degeneration of retina
- **Fundus Picture**
- Waxy pallor of disc
- Attenuation of vessels
- Associated Retinal pathology can be seen

Consecutive Optic Atrophy

Aetiology

- Retinitis pigmentosa
- CRAO / CRVO
- Long Standing Retinal Detachment
- Wide spread choroidal atrophy
- Extensive Pan retinal Photocoagulation



Glaucomatous Optic Atrophy

- Long standing Glaucoma

Fundus Picture

- Disc pale with deep enlarged cup reaching upto disc margin
- Lamellar dot sign seen
- Also called as cavernous optic atrophy