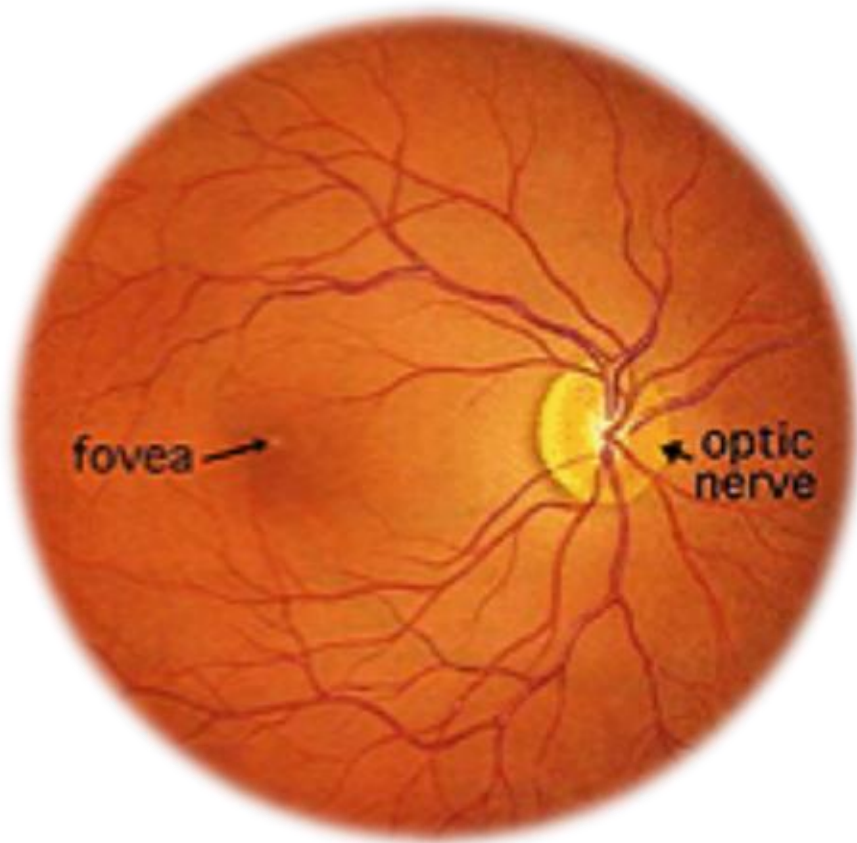


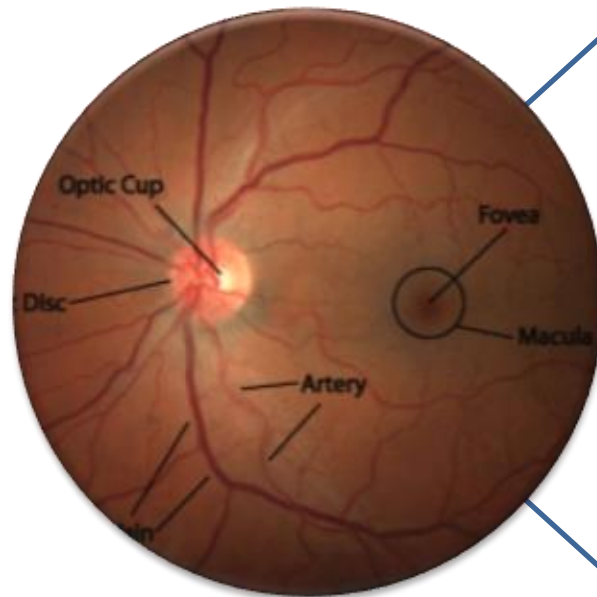
Retinal Diseases

Dr. Abidi



Normal Fundus

Anatomy



Retina extends from the optic disc to the ora serrata.

- It is divided into two distinct regions:
- Posterior pole
- Peripheral retina

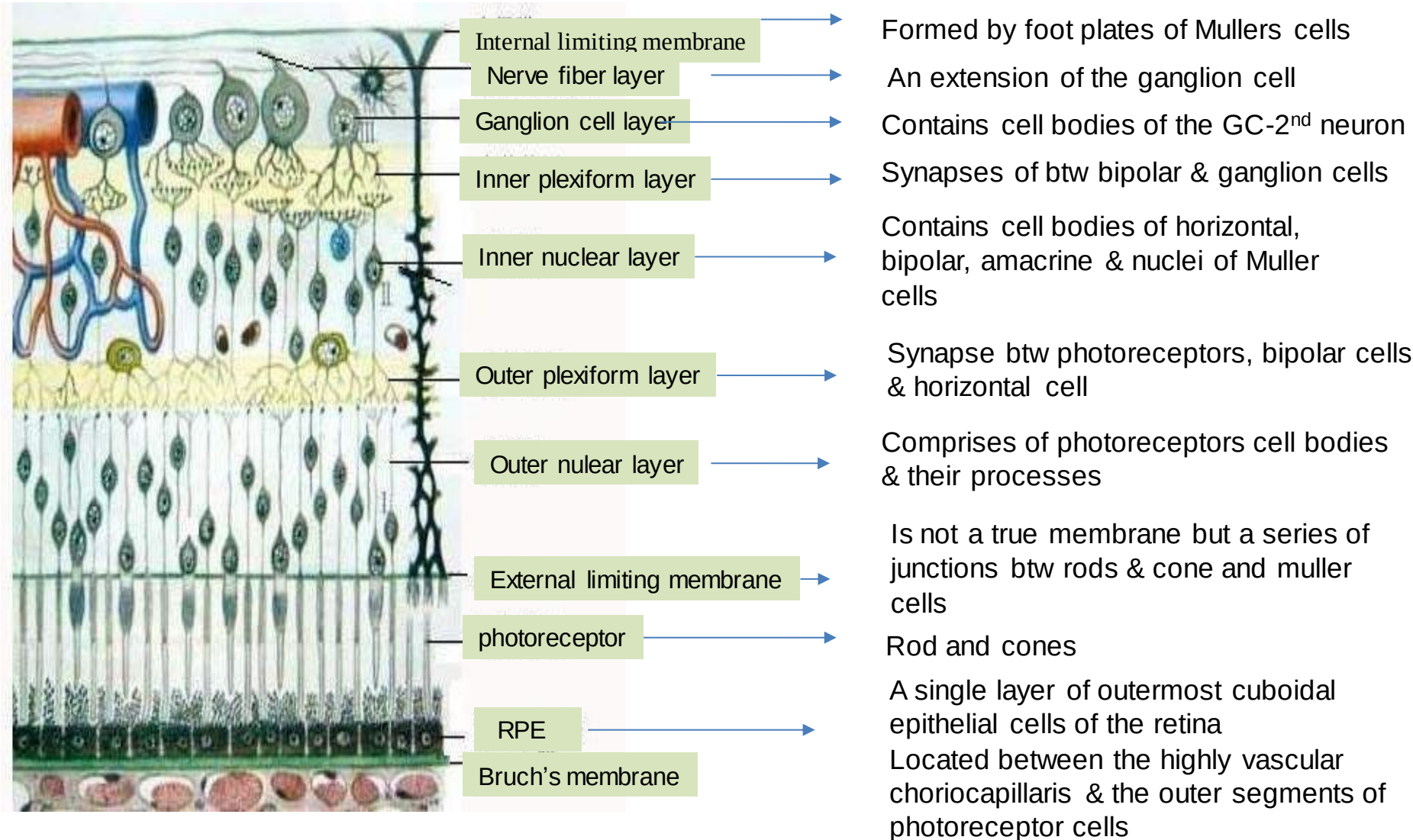
Size of Retina

- Retina is 0.56 mm thick near optic disc
- 0.1 mm at ora serrata
- Thinnest at the center of fovea

Macula lutea

- Is also called the yellow spot
- Situated at the posterior pole temporal to the optic disc.

Histology of retina



Gross Anatomy

The retina proper is a thin and delicate layer of nervous tissue- covers $\frac{3}{4}$ of inner eye wall

Grossly retina is divided into two parts;

Central retina

1. Foveola
2. Fovea
3. Para fovea
4. Perifovea
5. Macula

Rich in cones, has more ganglion cells per area than elsewhere, and is a relatively small portion of the entire retina.

Peripheral retina

1. Near periphery
2. Mid periphery
3. Far periphery
4. Ora serrata

Makes up most of the retina, and rods dominate

Clinical function

- Designed for fine visual acuity,
- Photopic vision
- Stereopsis
- Color vision

Clinical function

- Designed for gross vision
- Scotopic/night vision
- Sensitive to motion and stimulates turning of eye/head

Macula lutea

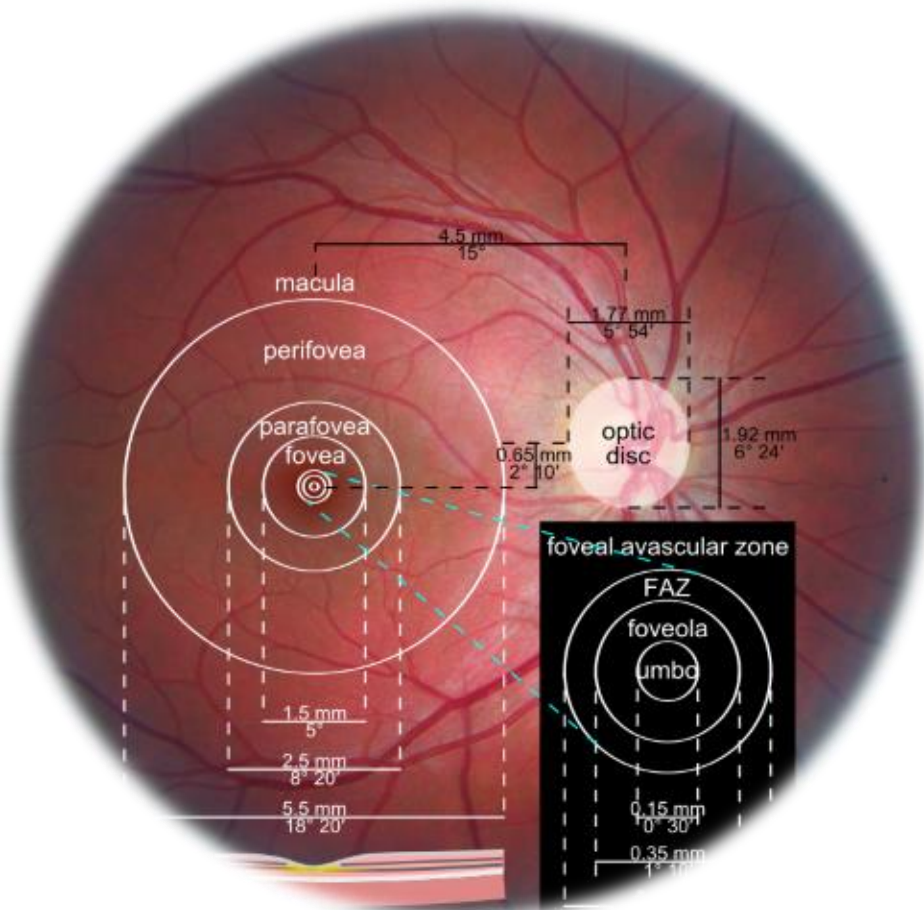
Foveola

↓
Umbo

Fovea

Parafoveal

Perifoveal



Note;

- *Thin foveal layers and compact cone photoreceptor helps to form sharpest vision and stereopsis*
- Loss of foveal reflex implies disruption of neural layers

Fovea

- ✓ Specially differentiated central 1.5 mm of macula in width
- ✓ 0.25 mm in thickness
- ✓ Most sensitive part of macula
- ✓ Corresponds to 5 degree of visual field
- ✓ Densest concentration of cones

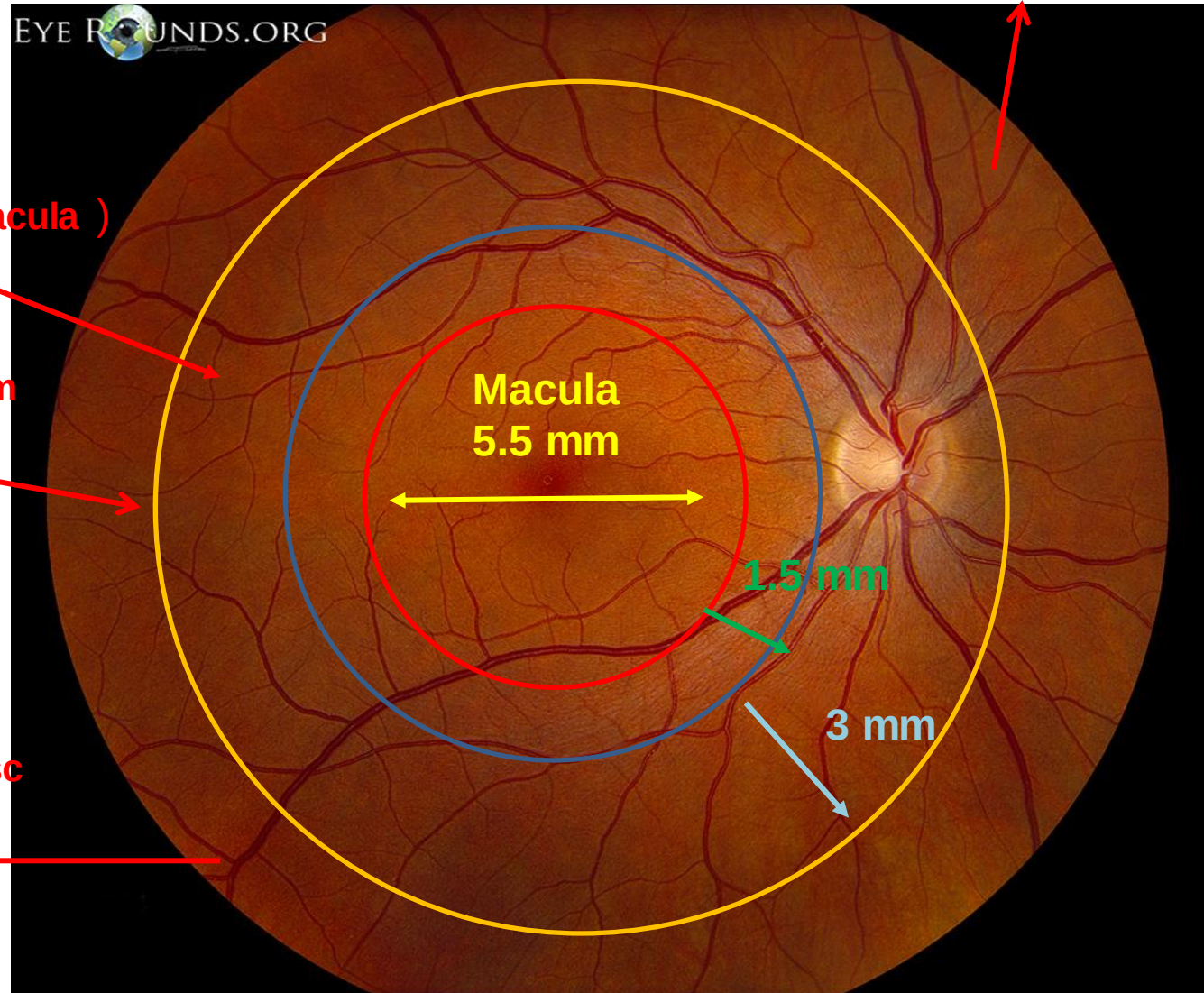
Foveola

- ✓ 0.35 mm wide depression or pit in the central floor of fovea.
- ✓ It is 2 DD (3mm) away from the temporal edge of optic disc & 1mm below the horizontal meridian.
- ✓ Thinnest part of retina
- ✓ Devoid of ganglion cells
- ✓ Has only cones & muller cells
- ✓ Vision is most acute at this area.

✓ Umbo

- ✓ Tiny depression in the centre of foveola.
- ✓ Corresponds to the ophthalmoscopically visible foveal reflex.
- ✓ **Foveal reflex:** intensely bright spot of light visible ophthalmoscopically visible due to reflexion of light from the wall of foveal depression.
- ✓ Loss of FR may be an early sign of damage.

Far Periphery 16 mm on
the nasal side in the
horizontal meridian



1. Near
Periphery
(1.5mm around the macula)

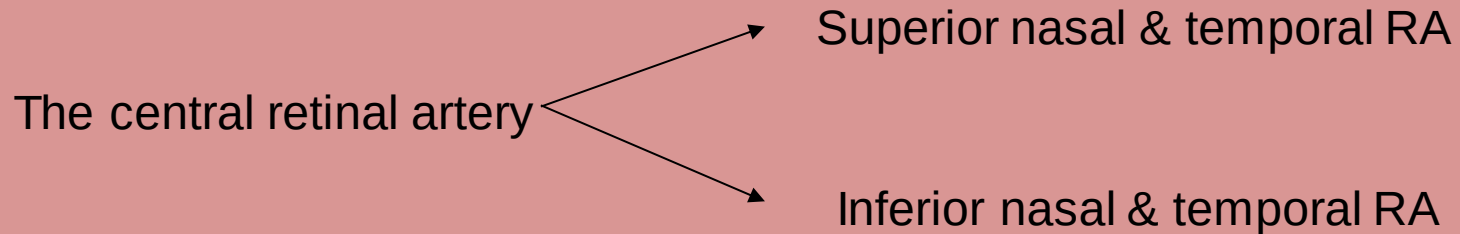
2. Mid -Periphery 3 mm
around near periphery

3. Far Periphery 9-10
mm from the Optic disc
on the temporal side

4. Ora Serrata

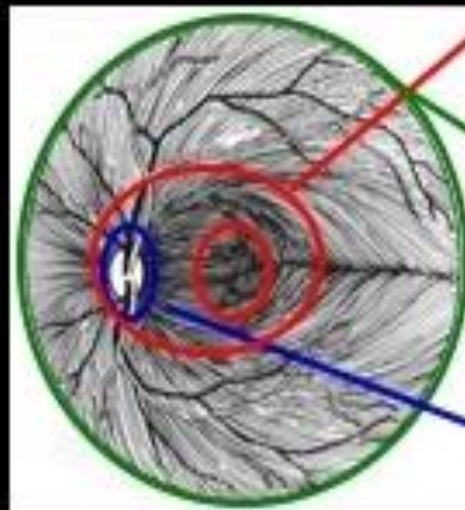
Equator is 24 mm from the optic disc.

Blood supply of retina



Occasionally a cilioretinal artery branching from the ciliary circulation, Will supply blood to the portion of inner retina btw optic nerve & macular

Outer 4 layers of retina are supplied by Choriocapillaris
Inner 6 layers by branches of the Central Retinal Artery



Area of retina responsible for central visual field (30 deg) includes macular area – detailed vision

60-70% of fibres optic nerve are from central 30 deg

All other areas of the retina contribute to peripheral field

EXCEPT
Optic nerve head – non 'seeing' part of the retina (takes signals from retina to higher areas)

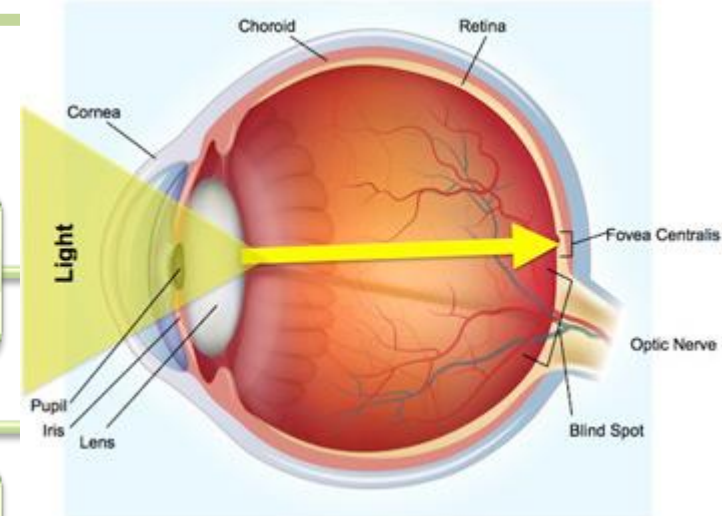
Functions of the Retina

To absorb photons of light

To translate light into a biochemical message

Translate biochemical message into electrical impulse

Transmit electrical impulse to the brain via ganglion cells



Symptoms of Retinal Diseases

Retinal diseases vary widely, but most share some common symptoms.
Most of the symptoms are visual

Seeing floating
specks or cobwebs



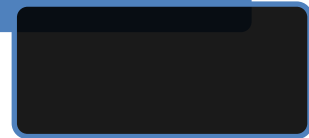
Blurred or distorted
vision



Defects in the field
of vision



Loss of vision



Flashes of light



Questions

1. Name the branches of the retinal artery
2. Name the layers of the retina
3. What are the symptoms of retinal disease
4. What is metamorphopsia

Classification of retinal diseases

- Vascular diseases
- Macular diseases
- Retinal detachment
- Retinal degeneration
- Retinal tumor
- Ocular manifestation of general diseases

Retinal vascular disease

Retinal artery occlusion

Retinal venous occlusion

Diabetic retinopathy

Vasculitis

Coats disease

Central retinal artery occlusion, CRAO

Etiology

Common causes:

- Embolism is the most common cause of CRAO,
 - The major source of this being carotid artery disease, usually due to atherosclerotic plaques.
 - Carotid stenosis and the heart are other important sources of emboli
- It is equally probable that an occlusive thrombus at the level immediately posterior to the lamina cribrosa also causes CRAO

Risk Factors

The risk factors and demographics of retinal artery occlusion are similar to ischemic stroke and include several modifiable risk factors:

- Older age
- Male gender
- Smoking
- Hypertension
- Obesity
- Diabetes
- Hyperlipidaemia
- Cardiovascular disease
- Coagulopathy

Symptoms



☐ Sudden painless vision

☐ Unilateral

☐ Persistent



Signs



☐ A relative afferent pupillary defect (RAPD)

☐ Retinal opacity in the posterior pole (58%),

☐ Cherry Red Spot 90%

☐ Arterial attenuation 32% cattle tracking 19%

☐ Optic disk oedema & pallor

Work up

- Investigate the patient for systemic cardiac disease:
 - Carotid Doppler
 - Echocardiography
 - ESR & C-reactive protein
 - Prothrombin time
 - Serum Homocysteine levels (in young patients especially)
- Management of systemic disease is always after the initial emergency treatment
- Look for ocular complications-may develop neovascular glaucoma

Treatment

- The management of CRAO should be divided into:
 - *Acute*: Attempt to restore ocular perfusion to the CRA.
 - *Subacute*: Preventing secondary neovascular complications to the eye.
 - *Long term*: Preventing other vascular ischaemic events to the eye or other end organ.

Treatment

Use of sublingual isosorbide dinitrate

Systemic pentoxifylline

Inhalation of a carbogen, hyperbaric oxygen

To increase blood oxygen content and dilate retinal arteries

Ocular massage to attempt to dislodge emboli

Intravenous acetazolamide and mannitol

Anterior chamber paracentesis

To increase retinal artery perfusion pressure by reducing intraocular pressure

Laser embolectomy

Lysis of the emboli can be achieved with Nd:YAG laser. ^[28, 29, 30] Retinal perfusion with gain of visual function can be achieved. However, complications such as creation of false aneurysms and vitreous hemorrhage may arise.

Pars plana vitrectomy with direct central retinal artery massage

A probe was designed to apply direct gentle pressure/massage over the CRA and the optic nerve head. Of 10 patients, circulation was restored in only 4. ^[31]

Intra-arterial fibrinolysis

CRAO is one of the few ophthalmology emergencies in which management depends on time of onset. Intraarterial thrombolysis has been used anywhere from 1 hour to up to 24 hours, although this procedure is very controversial. ^[32]

Thrombolytic agents that have been studied for CRAO treatment include intra-arterial tissue plasminogen activator (tPA) and urokinase.

Digital subtraction angiography (DSA)–guided superselective ophthalmic artery or selective carotid thrombolysis remains the preferred treatment method for CRAO. More recently, branch retrograde thrombolytic intervention of the ophthalmic artery (urokinase and papaverine) was shown to be effective for CRAO. ^[33] Mercier et al reported that fibrinolysis was more effective than conservative management in 16 patients with CRAO. ^[34] In 11 patients, Nedelmann et al found clinically relevant visual improvement with thrombolysis in patients with CRAO only in the absence of a “spot sign,” an ultrasonographic sign they hypothesize may indicate calcified intra-arterial emboli due to atherosclerotic plaques. ^[35]

Questions

1. What are other causes of sudden painless loss of vision
2. Why do we see cherry red spot
3. Why is macula called the yellow spot
4. How soon should a case of CRAO be attended
5. How long does retinal tissue remain viable after first reporting by the patient

Classification

Central retinal vein occlusion (CRVO)

- Non-ischemic CRVO All 4 quadrant involved
- Ischemic CRVO

Branch retinal vein occlusion

- Major BRVO Segmental area involved
- Macular BRVO

• Hemicentral retinal vein occlusion

- Non-ischemic HCRVO 2 quadrant involved
- Ischemic HCRVO vertical

Retinal Vein Occlusion

Location of Occlusion

CRVO at or behind the Lamina Cribrosa

BRVO at retinal a/v crossing

HRVO at or behind the Lamina Cribrosa

Pathophysiology

That most forms of RVO share a multifactorial mechanism:

Thrombosis of the central retinal vein at and posterior to the level of the lamina cribrosa.

In some cases, a thickened central retinal artery may impinge on the central retinal vein  **turbulence**  **Endothelial damage**  **thrombosis**

Risk Factors-Age

```
graph LR; A[Risk Factors-Age] --- B[Systemic-hypertension]; A --- C[Diabetes mellitus]; A --- D[Cardiovascular disease]; A --- E[Hyperlipidemia]; A --- F[Open-angle glaucoma]; A --- G[Oral contraceptive pill];
```

Systemic-hypertension

Diabetes mellitus

Cardiovascular disease

Hyperlipidemia

Open-angle glaucoma

Oral contraceptive pill

Symptoms of CRVO

1. 50+ years of age
2. The patient may be asymptomatic, but often will complain of sudden painless unilateral loss of vision and/or visual field
3. May complain of a sudden onset of floating spots or flashing lights.
4. Acuity may range anywhere from 6/6 to finger counting.
5. If vision loss is severe, there may be an RAPD.

Ischemic

Severe

Usually presents with severe visual loss

Dot-Blot & splinter retinal hemorrhages

Cotton-wool spots

Macular edema

Marked Venous tortuosity & dilatation

RAPD +

Poor perfusion to retina.



Non-ischemic

Milder.

It may present with
good vision.

Mild V-field defect

Dot-Blot retinal hemorrhages

Cotton wool spots

Mild macular edema

Mild optic disc swelling

Mild venous dilatation

RAPD absent

Good perfusion to the
retina.



Ischemic vs. Non-ischemic

Primary difference involves presence of retinal hypoxia because of ischemia

Ischemic characterized by at least 10DD of retinal capillary non-perfusion

Determined by fluorescein angiogram.

Complications

The hypoxic retinal tissue in CRVO may release vascular endothelial growth factor (VEGF) and inflammatory mediators

Macular
Oedema

Vitreous
Haemorrhage

Neovascular
Glaucoma

Management

Find underlying cause

Complete Ophthalmic check up. Rule out Glaucoma

FFA, OCT

Lab Tests: CBC, RBS, Lipid profile

Complete cardiac check up

Management

Current treatment is directed at secondary complications of CRVO that affect vision, including:

Macular edema

NVG

Retinal neovascularization

R.D

Macular ischemia (CNP) another cause of visual loss in CRVO haven't effective treatment.

Treatment also involves management of predisposing risk factors, such as diabetes, hypertension and hyper-lipidemia

Medical Care

No known effective medical treatment is available for either prevention or the treatment of CRVO

Possibilities include:

- Aspirin
- Systemic anticoagulation
- Fibrinolytic agents
- Systemic corticosteroids
- Anti-inflammatory agents
- Isovolumic haemodilution
- Plasmapheresis.

Surgical Care

Recommended to wait for the development of early iris neovascularization and then apply pan retinal photocoagulation (PRP)

Many procedures are under study

Prognosis

Non-Ischemic CRVO Prognosis

Complete recovery with good visual recovery occurs only in about 10% of cases.

Fifty percent of patients will have 6/60 or worse vision.

About one third of patients convert to ischemic CRVO within 3 years; 15% convert within the first 4 months.

Prognosis

Ischemic CRVO Prognosis

More than 90% of patients will have 6/60 or worse vision.

About 60% of patients develop ocular neovascularization with associated complications.

About 10% of patients can develop CRVO or other type of vein occlusions either within the same eye or fellow eye within 2 years

Central Vein Occlusion Study (CVOS) (Summary)

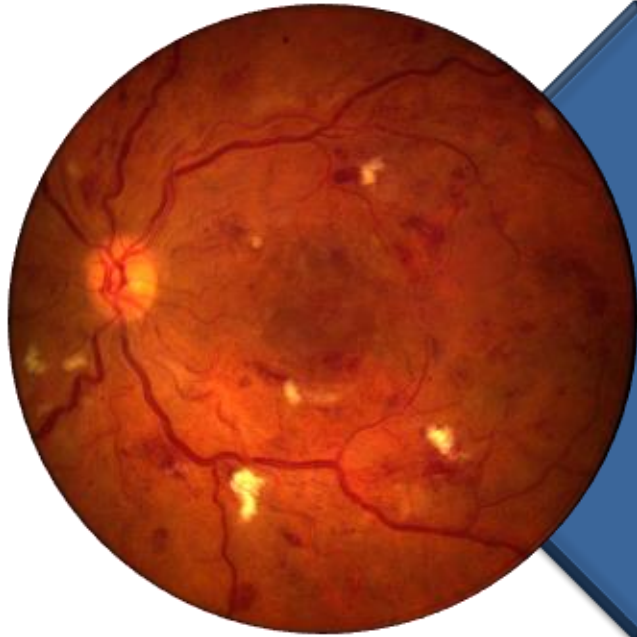
To determine whether photocoagulation therapy can help prevent iris neovascularization in eyes with CVO and evidence of ischemic retina.

To assess whether grid-pattern photocoagulation therapy will reduce loss of central visual acuity due to macular edema secondary to CVO.

To develop new data describing the course and prognosis for eyes with CVO.

Questions

1. How many types of retinal venous obstructions are there?
2. Name them
3. Which has a better prognosis
4. What are risk factors for CRVO
5. What is the site of occlusion in CRVO



Diabetic Retinopathy

Diabetic Retinopathy

Diabetes is a disease that affects over 65 million persons in India



This figure is estimated to rise to 79.4 million by 2030



Prevalence of DR increases with increasing duration of diabetes

Overall prevalence of retinopathy in Indians was about 15% in 2016

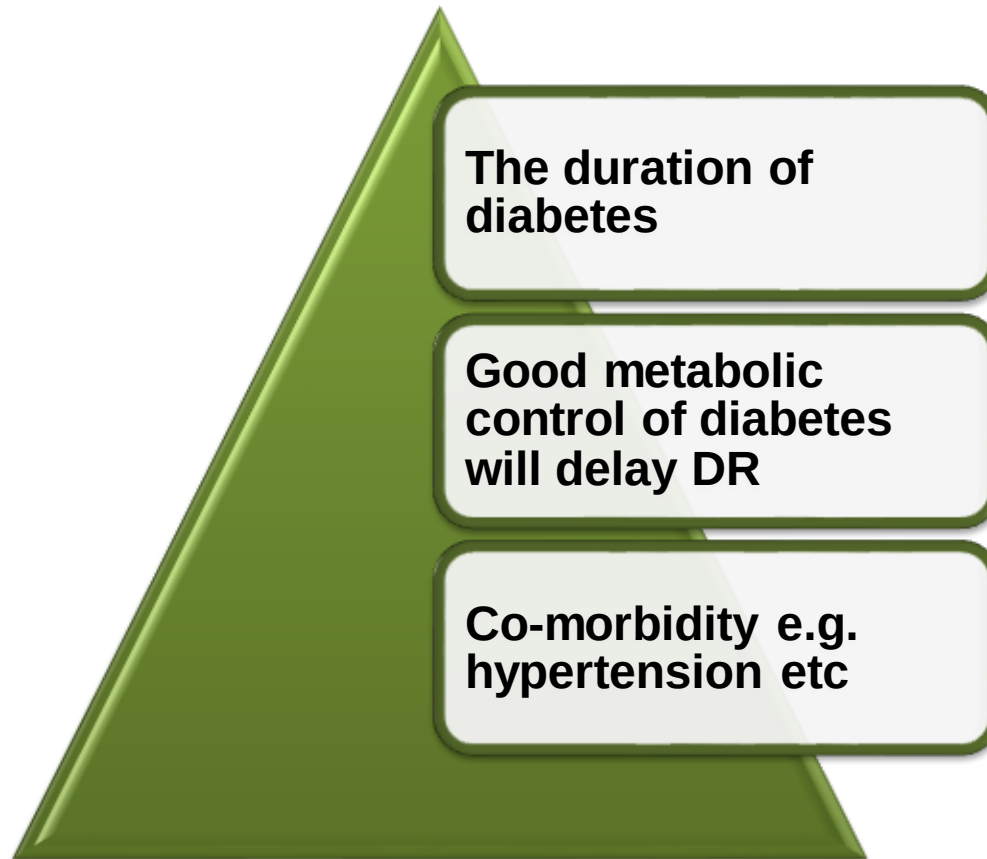


Retinopathy affects nearly one out of every ten persons with diabetes



DR is duration-dependent, good care of co-morbidities, in an aging diabetic population, the burden of retinopathy is likely to increase

Risk Factors



Diabetic Retinopathy

Classification

Non-proliferative

Mild

Moderate

Severe

Proliferative

Neovascularization

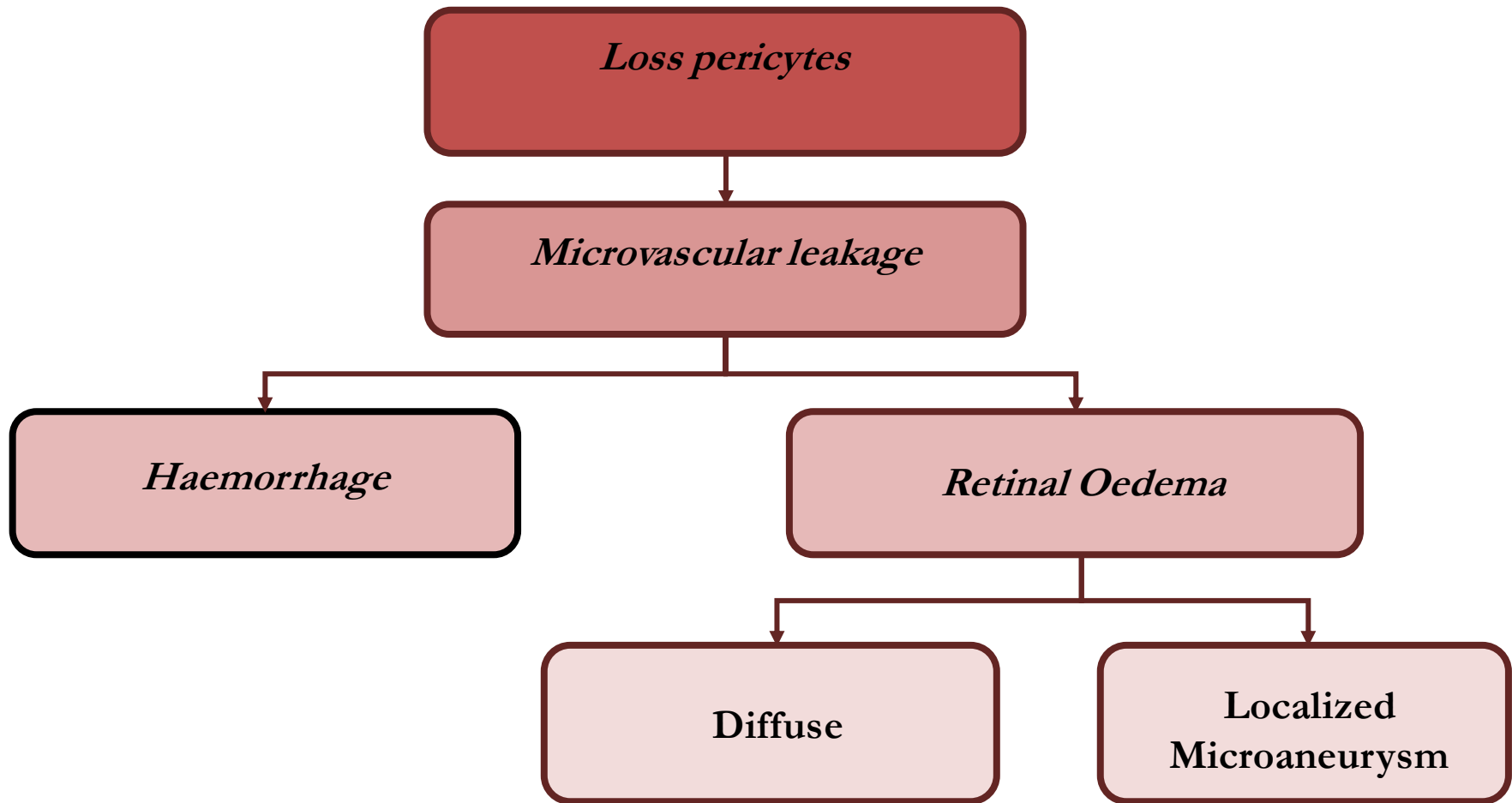
Vitreous/
preretinal
haemorrhage

Clinically significant
macular oedema

PATHOGENESIS

- Diabetic retinopathy is a microangiopathy affecting the retinal precapillary arterioles, capillaries and venules .
- Retinopathy has features of both:
 - microvascular leakage. (mild- mod NPDR)
 - microvascular occlusion .(sever NPDR-PDR)

PATHOGENESIS



PATHOGENESIS

Microvascular occlusion



Retinal Ischaemia



Retinal Hypoxia



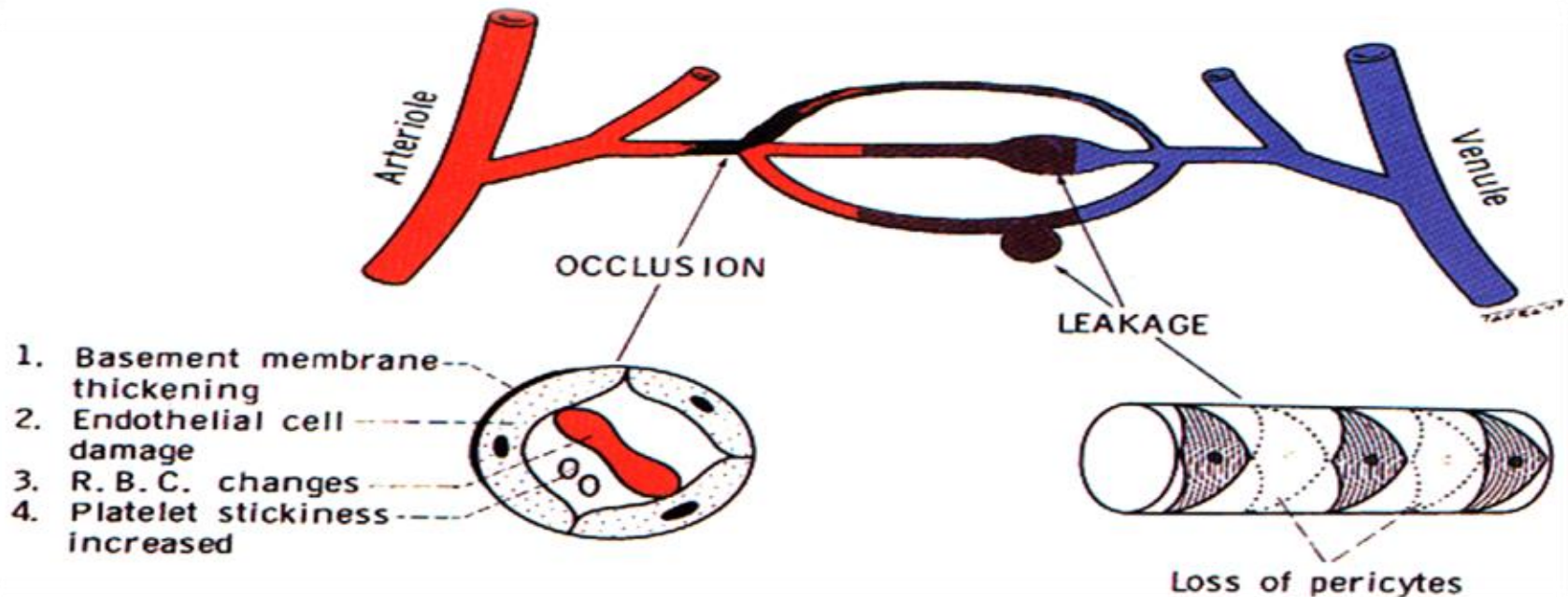
**Arteriovenous shunts
(IRMA)**

**Fibrous glial cell
proliferation**

Neovascularization

PATHOGENESIS

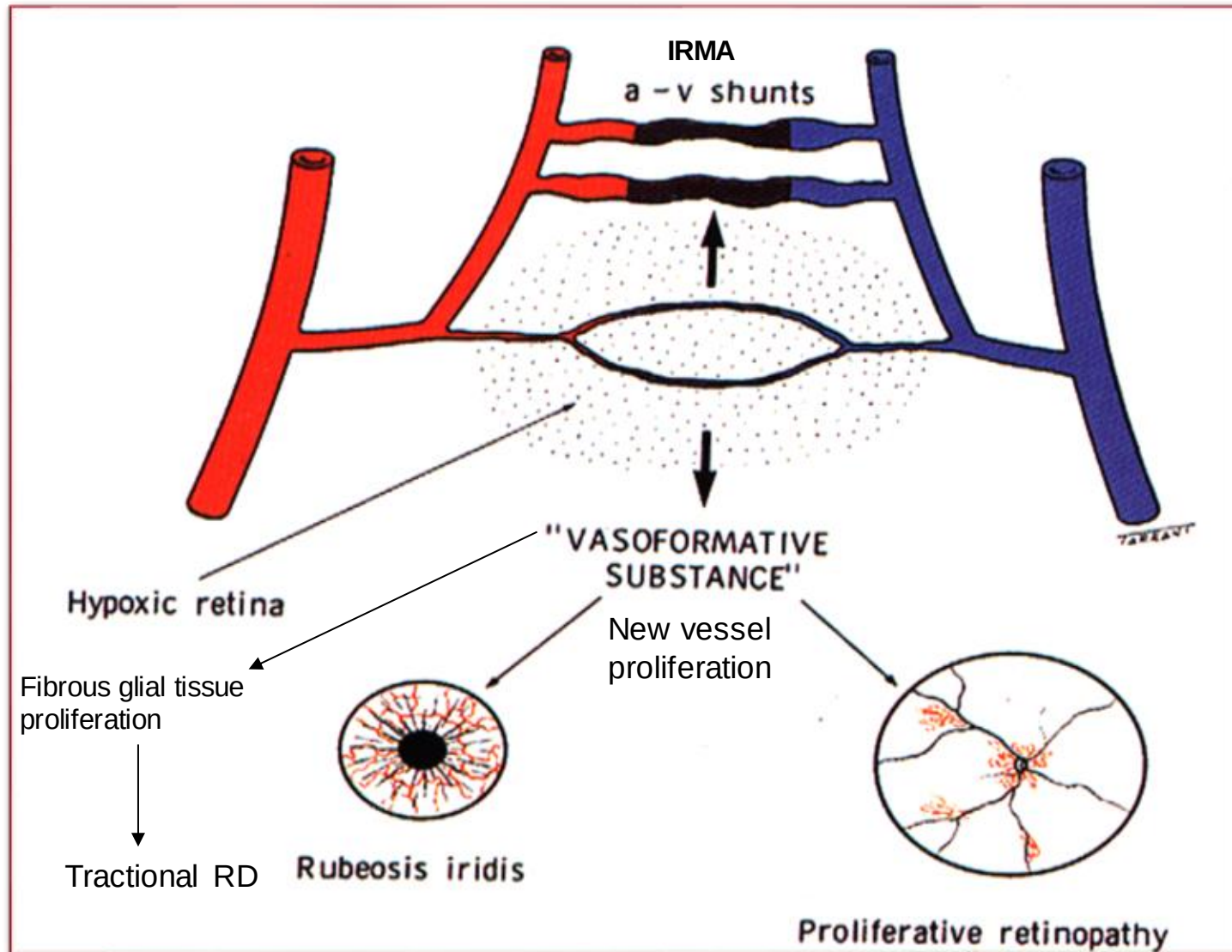
- **Microangiopathy** which has features of both microvascular **leakage** and **occlusion**
- Affecting the retinal precapillary arterioles, capillaries and venules
- Larger vessels may also be involved



PATHOGENESIS

- **Microvascular leakage**
- Loss of pericytes results in distention of weak capillary wall producing *microaneurysms which leak.*
- Blood-retinal barrier breaks down causing plasma constituents to leak into the retina – *retinal oedema, hard exudates*

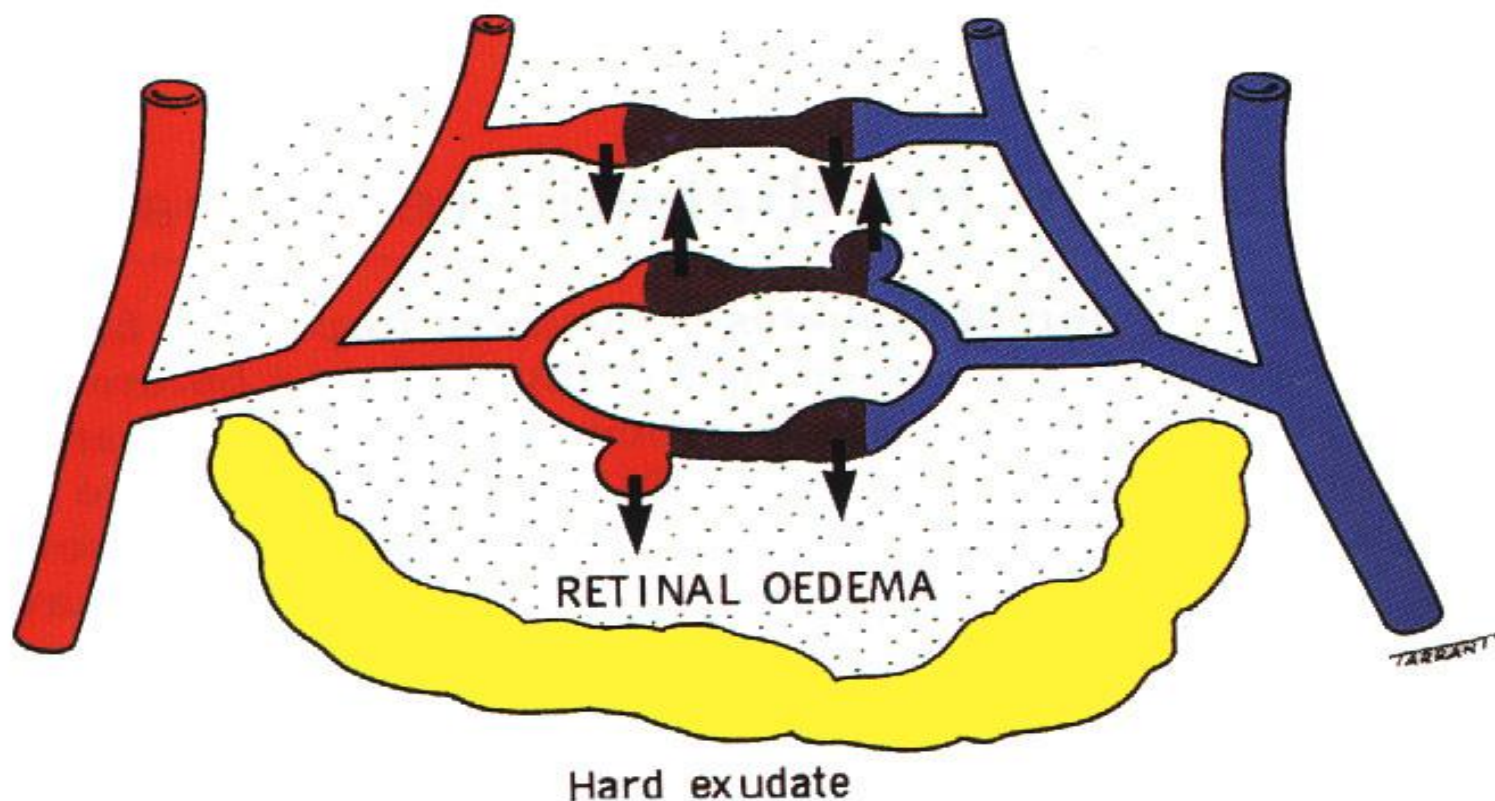
PATHOGENESIS



Consequences of retinal ischaemia

PATHOGENESIS

Consequences of chronic leakage



*Yellow Waxy **Hard Exudates**.* — They are composed of lipoprotein and lipid-laden macrophages. The exudates are located between the inner plexiform and inner nuclear layers of the retina. They appear as yellow-white refractile deposits, particularly in the region of the macula.

PATHOGENESIS

- **Microvascular leakage:**
 - Due to reduction in the number of pericytes .
 - The pericytes are wrapped around the capillaries and are thought to be responsible for the structural integrity of the vessel wall.
 - Development of retinal edema requires accumulation of fluid which occurs if :

Leakage :

- Microaneurysms.
- Incompetent capillaries



Absorption :

- Uptake from adjacent capillaries
- Healthy RPE cells

Neovascularisation

Caused by capillary non perfusion leading to retinal hypoxia.

New vessel growth is thought to be caused by an imbalance between angiogenic and anti-angiogenic factors in an attempt to re-vascularise the hypoxic retina

Angiogenic factors	Anti angiogenic factors
VGEF	Endostatin
PDGF	Angiostatin
HGF	PEDF
FGF	
Growth Hormones	

IRMA (intraretinal microvascular abnormalities) are shunts that run between the retina from arterioles to venules

Microvascular Occlusion



Ischemia



Increased VEGF



Neovascularisation



Fibrovascular bands



Tractional Retinal Detachment



Infraction



Cotton Wool spots



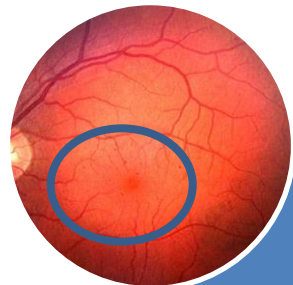
Neo-Vascular Glaucoma



Vitreous Haemorrhage

International Clinical Diabetic Retinopathy Disease Severity Scale	
Proposed Disease Severity Level	Findings Observable Upon Dilated Ophthalmoscopy
No apparent retinopathy	No abnormalities
Mild NPDR	Microaneurysms only
Moderate NPDR	More than just microaneurysms but less severe NPDR
Severe NPDR	US Definition Any of the following (4-2-1 rule) and no signs of proliferative retinopathy: - Severe intraretinal hemorrhages and microaneurysms in each of four quadrants - Definite venous beading in 2 or more quadrants - Prominent IRMA in 1 or more quadrants
PDR	One or both of the following: - Neovascularization - Vitreous/preretinal hemorrhage

Non-Proliferative Diabetic Retinopathy



Very Mild NPDR



Mild-NPDR



Moderate-NPDR

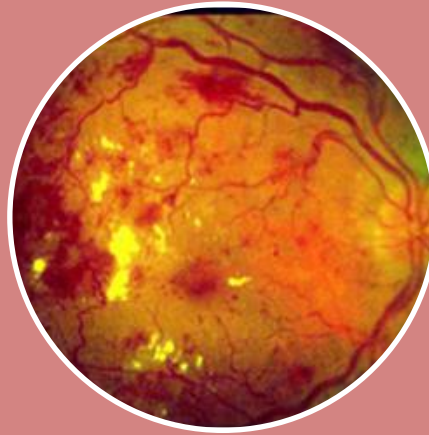


Severe-NPDR

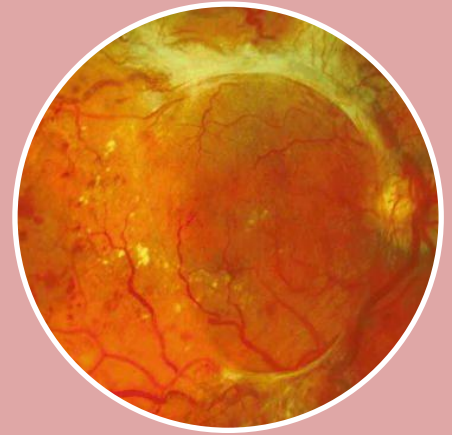
Proliferative Diabetic Retinopathy



New vessels on the disc (NVD) < 1/3 disc area



NVD > 1/3 disc area
Any NVD with vitreous or preretinal H'age
NVE > 1/2 disc area with vitreous or preretinal H'age



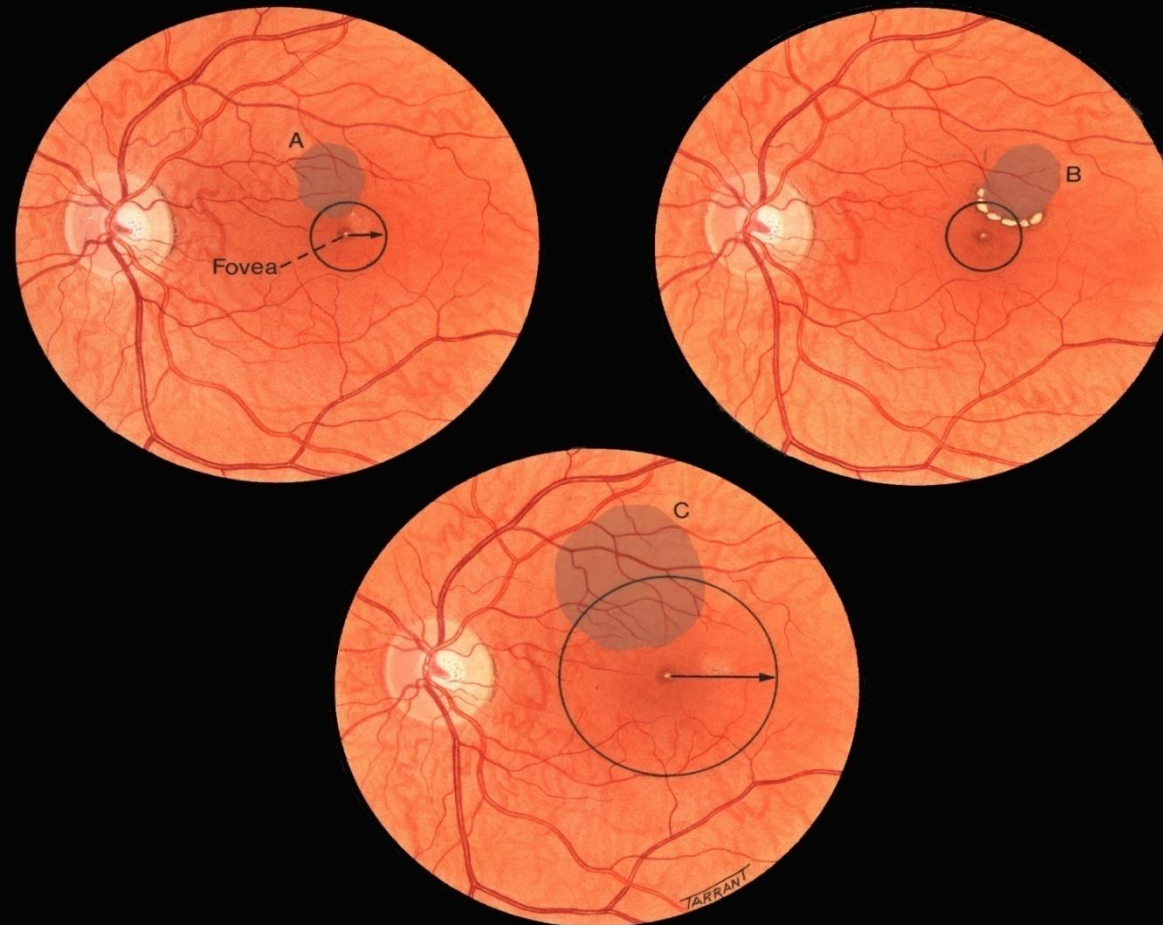
Preretinal &/or vitreous H'age
Tractional RD
Tractional Retinoschisis
Rubeosis Iridis

Mild PDR

Moderate PDR

Severe PDR

CSME defined as the presence of one or more of the following features



A. Retinal oedema within **500 μm** of the centre of the fovea

B. Hard exudates within **500 μm** of the fovea, if associated with adjacent retinal thickening (which may be outside the 500 μm limit) .

C. Retinal oedema that is **one disc area** (1500 μm) or larger, any part of which is **within one disc diameter** of the centre of the fovea.

Clinically Significant Macular Oedema



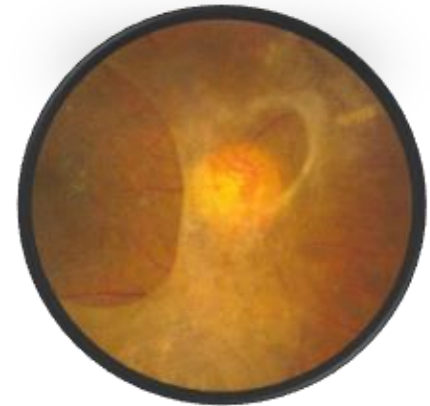
CSME – Hard exudates
close to fovea and
associated retinal thickening

Complications of Diabetic retinopathy

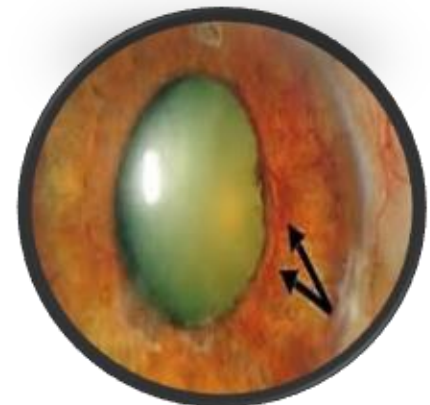
Vitreous
Haemorrhage



Tractional Retinal
Detachment



Rubeosis Iridis



Neovascular
Glaucoma

Blindness

Brief Early Treatment Diabetic retinopathy study (EDTRS) Classification

Category

Management

Non-Proliferative Diabetic Retinopathy (NPDR)

No DR	Review after 12 months
Very Mild Microaneurysms	Review most patient in 12 months
Mild Any or all of microaneurysms, retinal H'age hard exudates, cotton wool spots	Review range 6-12 months depending on severity of signs
Moderate Severe Ret. H'age in 1-3 quadrants or mild IRMA Significant venous beading in more than 1 quadrant Cotton wool spots	Review in approximately 6 months, (PDR in up to 26%, high-risk PDR in up to 8% within a year)
Severe The 4-2-1 rule Severe ret. H'age in 4 quadrants Significant venous beading in ≥ 2 quadrants Moderate IRMA in ≥ 1 quadrants	Review in 4 months (PDR in up to 50%, high-risk PDR in up to 15% within a year)
Very Severe ≥ 2 of the criteria for severe	Review in 2-3 months High-risk PDR in up to 45% within a year)

PDR requires the presence of newly formed blood vessels or fibrous tissue, or both, arising from the retina or optic disc and extending along the inner surface of retina or optic disc or into the vitreous cavity

Category

Management

Proliferative Diabetic Retinopathy (PDR)

Mild-Moderate

New vessels on the disc (NVD) < 1/3 disc area

Treatment considered according to severity of signs, stability, systemic factors, & patient personal circumstances
If not treated, review in 2 months or less

High-Risk

NVD > 1/3 disc area
Any NVD with vitreous or preretinal H'age
NVE > 1/2 disc area with vitreous or preretinal H'age vitreous

Laser photocoagulation
Intravitreal anti-VGEF
Intravitreal Triamcinolone
Pars plana vitrectomy
Lipid lowering drugs

Advanced Diabetic Eye Disease

Preretinal (retrohyloid) &/or vitreous H'age
Tractional RD
Tractional Retinoschisis
Rubeosis Iridis

Pars plana vitrectomy

What is the full form of NPDR?

Presence of DR depends on what factors?

What is meant by photocoagulation?

What is 4-2-1 rule in Severe PDR?

What are hard exudates



Hypertensive Retinopathy (HTR)

Incidence of HT

**Many causes
mainly essential
HT**

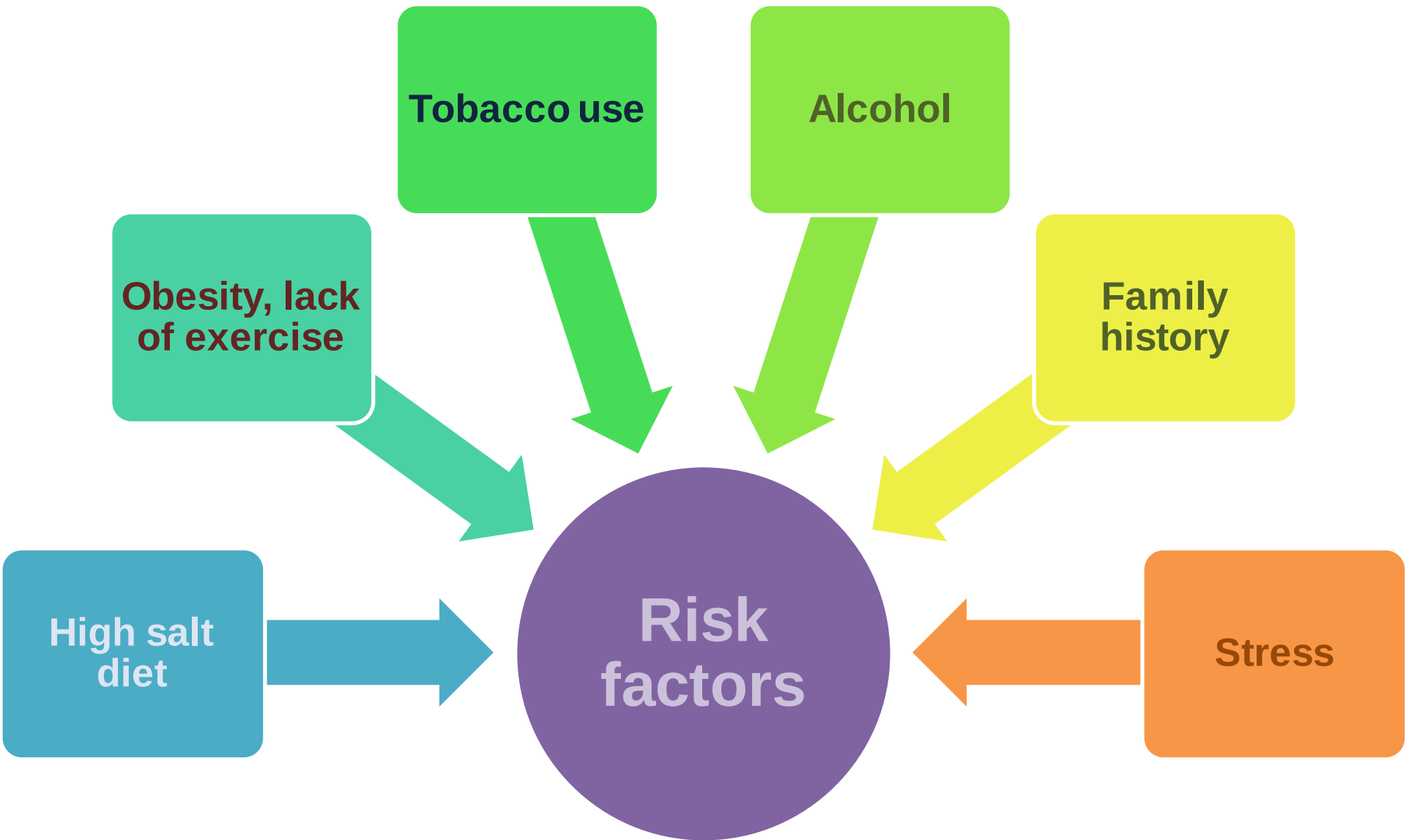
**Overall
prevalence for
HT in India is
29.8%**

**Malignant HT
(240/140 mm hg)
0.5-0.75%**

HTR 4-10%

Aetiology of HTR

The arteriosclerotic changes of hypertensive retinopathy are caused by chronically elevated blood pressure, defined as systolic greater than 140 mmHg and diastolic greater than 90 mmHg



Hypertensive retinopathy includes two disease processes

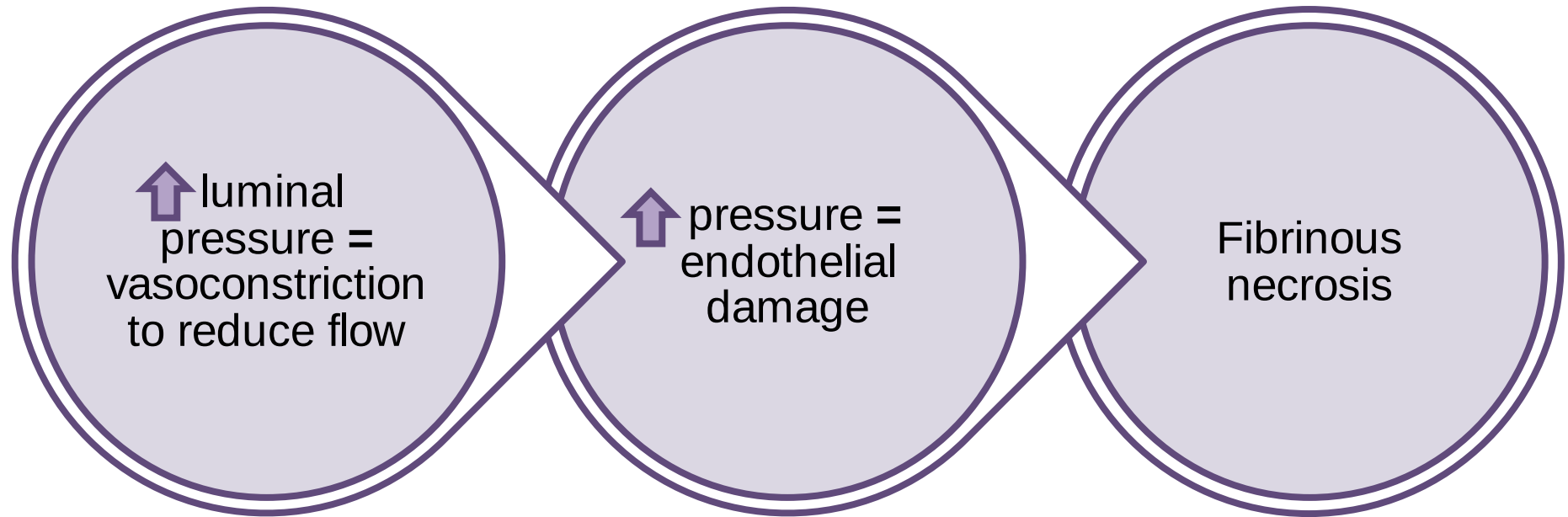
The Acute effect:

of systemic arterial hypertension are a result of vasospasm to autoregulate perfusion

The Chronic effect:

of hypertension are caused by arteriosclerosis and predispose patients to visual loss from vascular occlusions or macro aneurysms

General Pathology



Pathophysiological Changes in Hypertensive Ocular Disease

1. HYPERTENSIVE CHOROIDOPATHY

2. HYPERTENSIVE RETINOPATHY

- Vasoconstrictive phase
- Arteriosclerotic changes
- Increased vascular permeability

3. HYPERTENSIVE OPTIC NEUROPATHY

- Optic disc edema
- Optic atrophy
- Ischemic optic neuropathy

Hypertensive Retinopathy

Grading of Arteriolo sclerosis

GRADE 0

NORMAL

GRADE 1

**INCREASED LIGHT REFLEX, WITH
MILD ARTERIOVENOUS CROSSING
DEFECTS**

GRADE 2

COPPER WIRE APPEARANCE

GRADE 3

**SILVER WIRE APPEARANCE,
WITH MARKED ARTERIO
VENOUS CROSSING DEFECTS**

GRADE 4

FIBROUS CORD

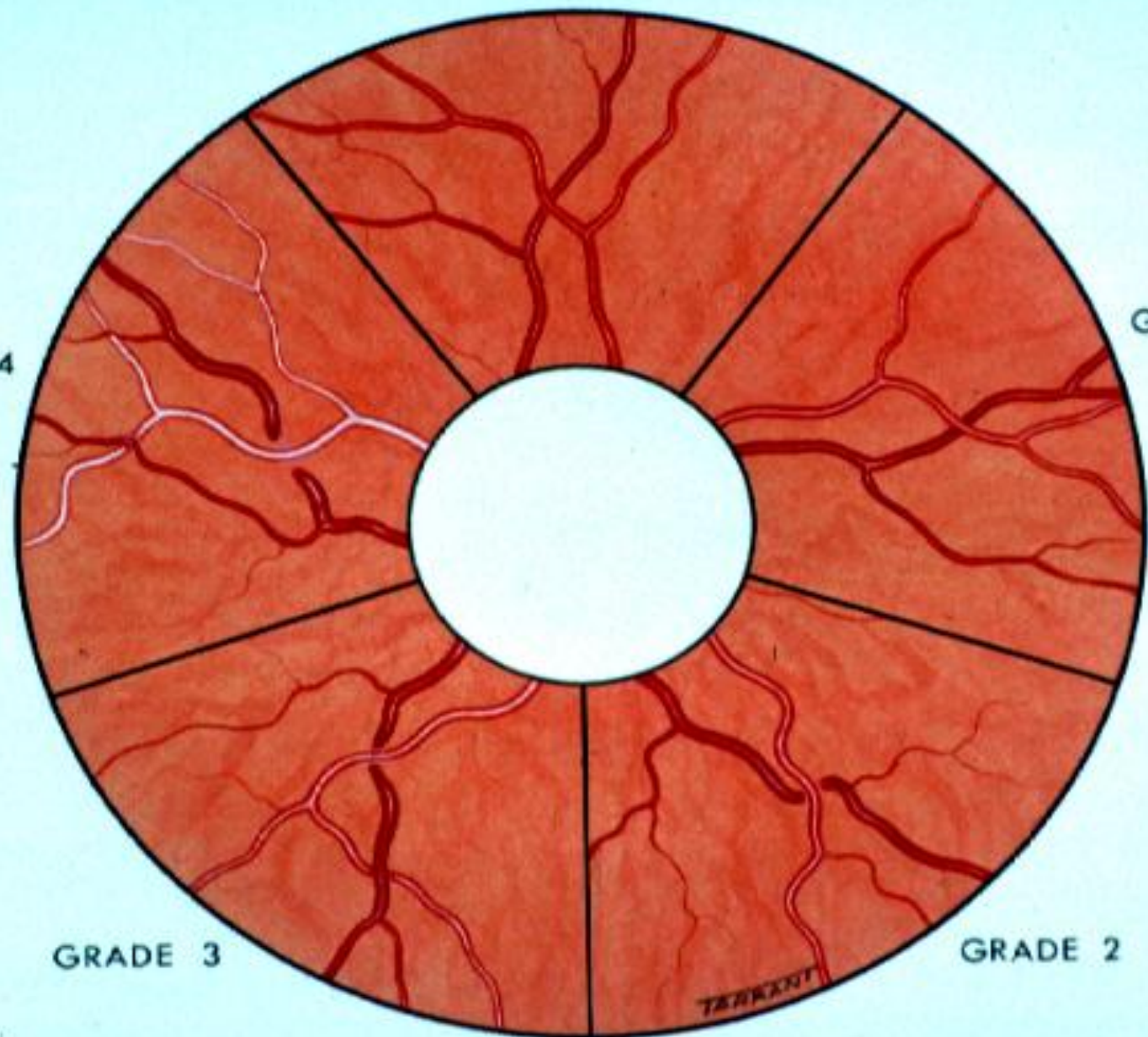
NORMAL

GRADE 1

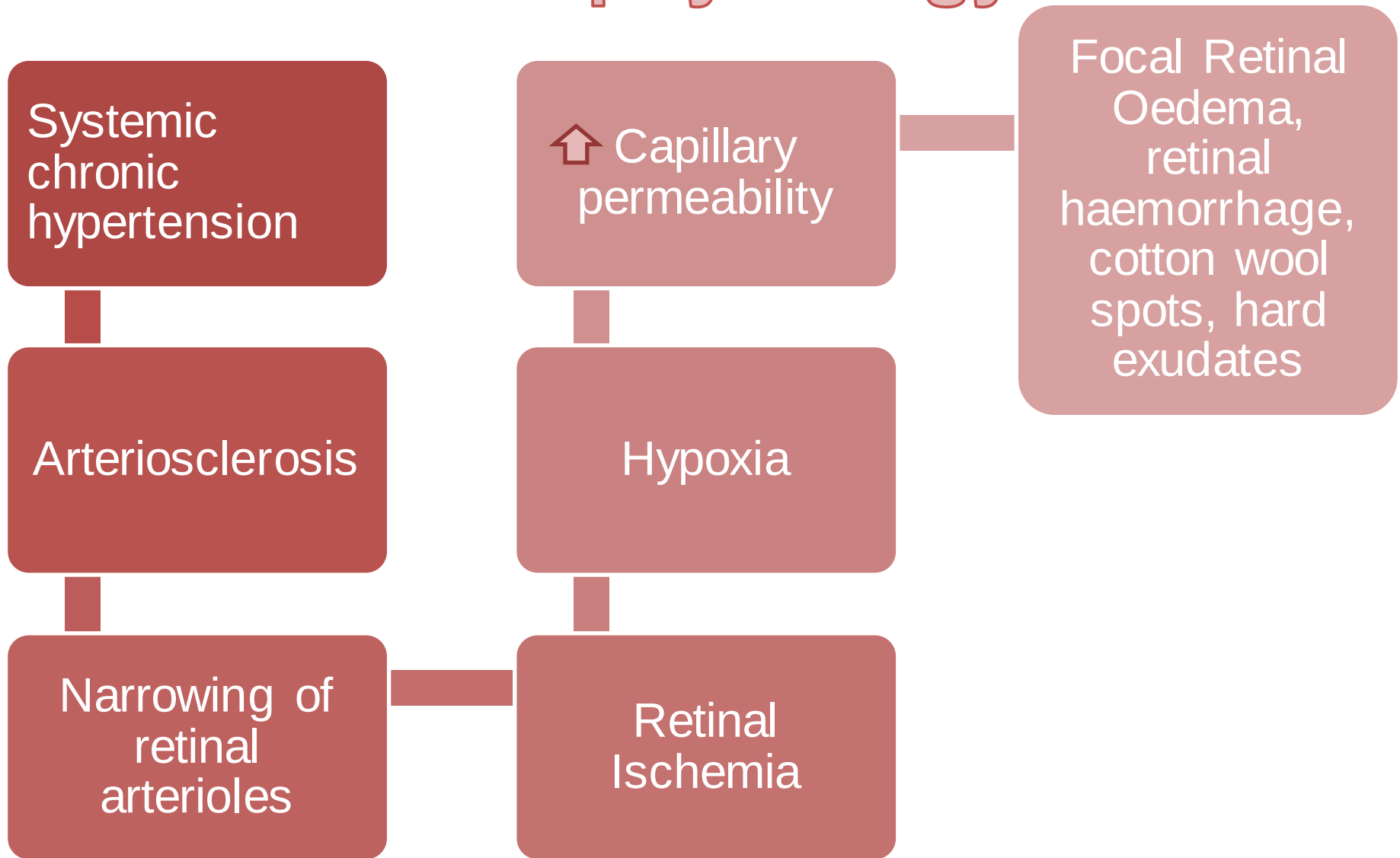
GRADE 4

GRADE 3

GRADE 2



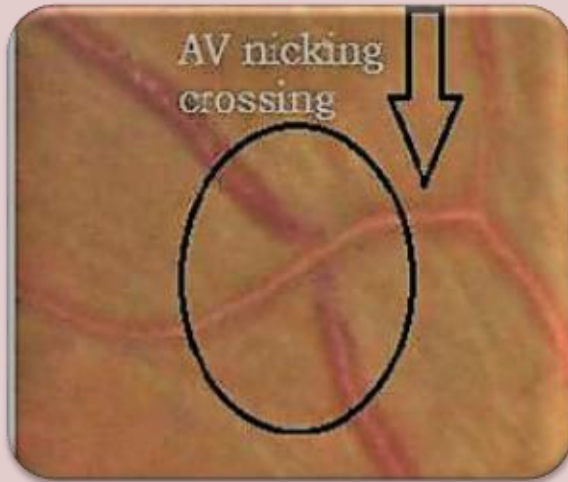
Pathophysiology



Pathophysiology of hypertensive retinopathy

	<i>Pathophysiological mechanism</i>	<i>Signs</i>
Acute hypertension	Vasospasm Increased vascular tone	Generalized arteriolar narrowing
Chronic hypertension	Intimal thickening, media wall hyperplasia, hyaline degeneration of arterioles Compression of venules at their common adventitial crossings	Diffuse and focal narrowing ('copper-wiring') and opacification ('silver-wiring') of arteriolar walls 'Arteriosclerotic stage': arteriovenous nicking
Severe hypertension	Inner blood-retinal barrier breakdown Necrosis of vascular smooth muscle and endothelial cells Persistent damage to retinal microvasculature	'Exudative stage': exudation of blood (retinal hemorrhages) and lipids (hard exudates) Nerve fiber layer ischemia (cotton wool spots)
Accelerated hypertension	Intracranial pressure elevation Fibrinoid necrosis of choroidal arterioles	Hypertensive optic neuropathy/'malignant retinopathy' Optic nerve ischemia Optic disc swelling (papilloedema) Hypertensive choroidopathy Infarction of segment of choriocapillaris RPE infarcts: Elsching's spots Linear RPE hyperplasia over infarcted choroidal arterioles (Siegrist's streaks) Localized bullous neurosensory/RPE detachments

Abbreviation: RPE, retinal pigment epithelium.



Salu's sign –
deflection of
veins at A-V
crossing

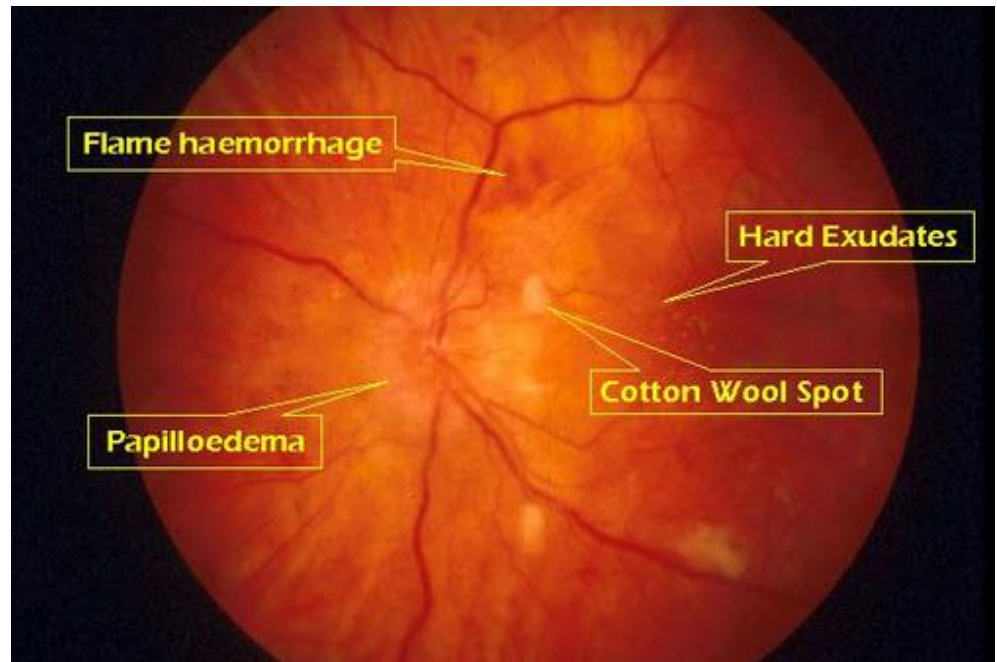
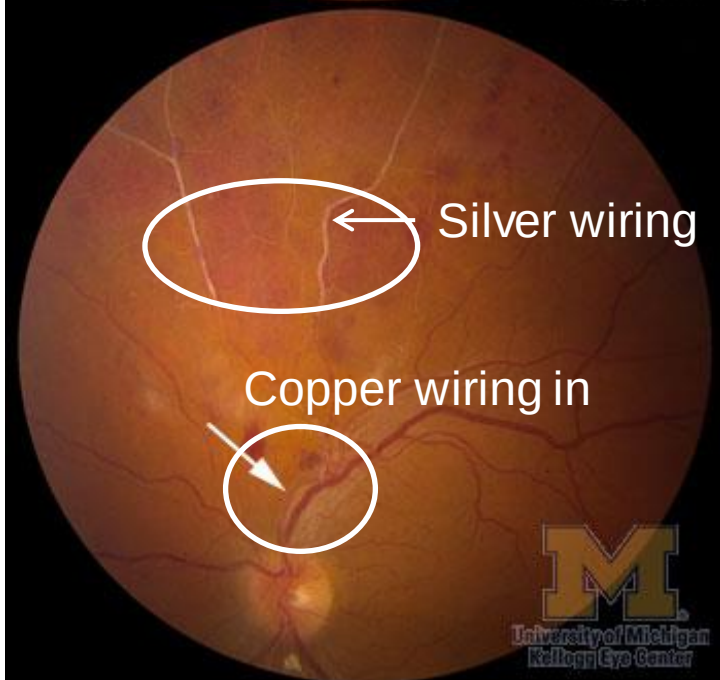
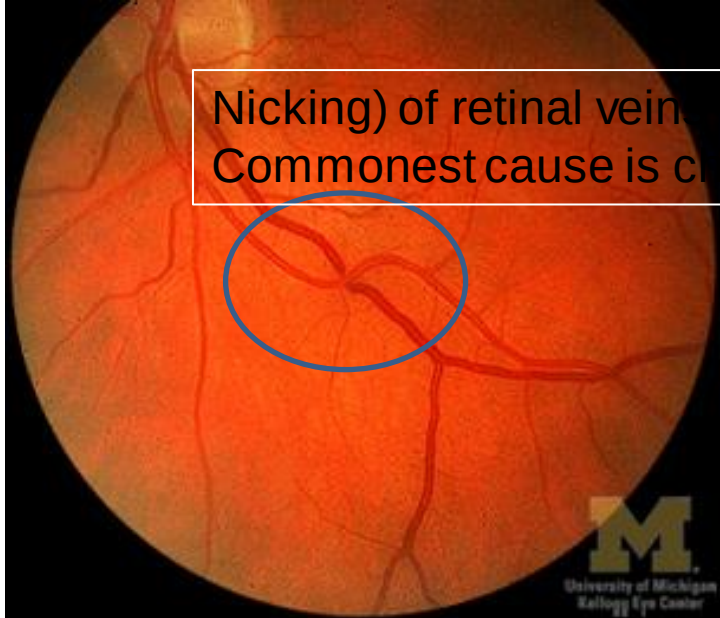


Bonnet sign
– banking of
veins distal
to A-V
crossing



Gunn sign –
tapering of
veins on
either side of
crossing

Nicking) of retinal veins by stiff (arteriosclerotic) retinal arteries
Commonest cause is chronic hypertension



Malignant hypertension

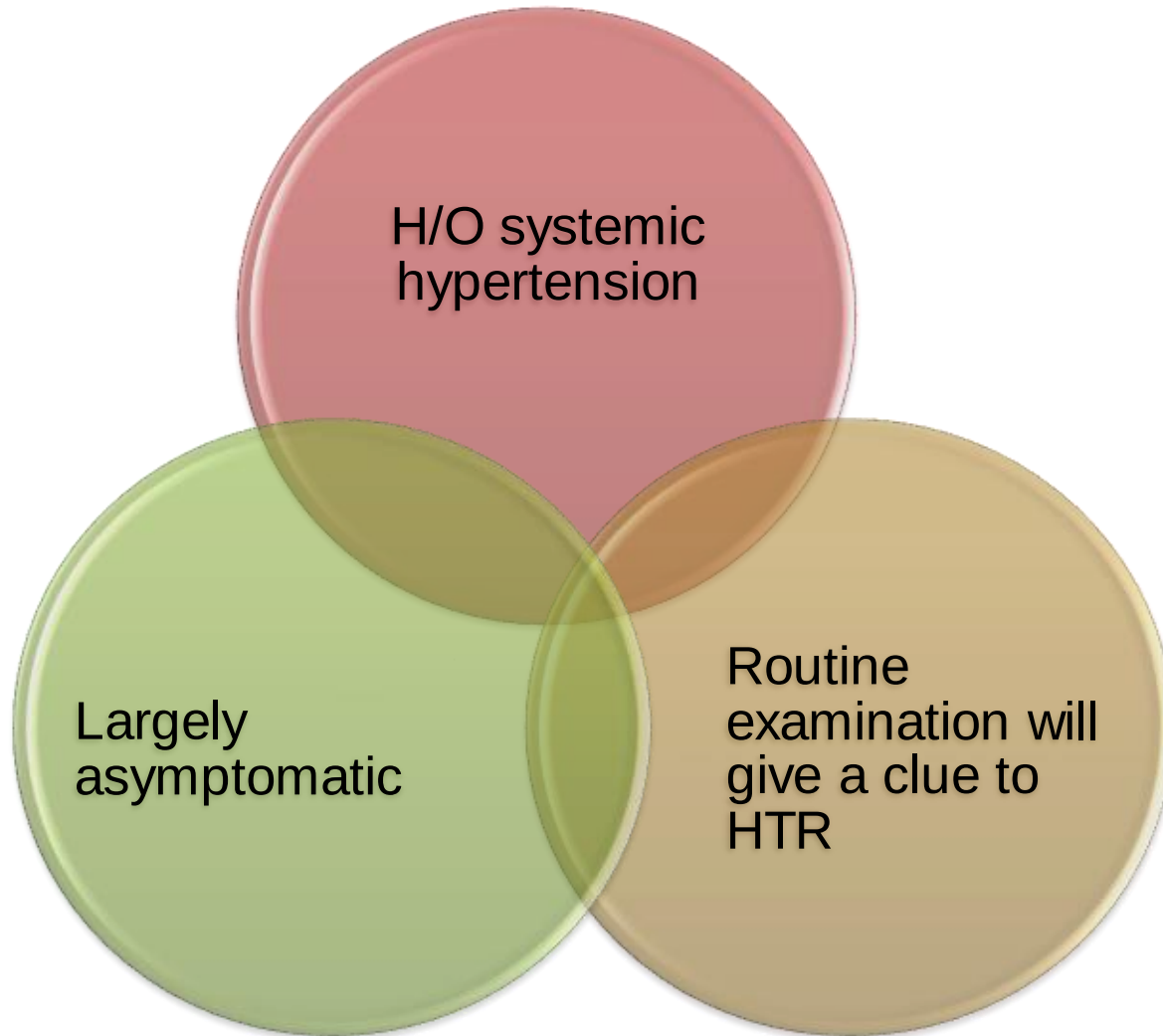
Keith, Wagener, and Barker Classification

Grade	Classification
Grade 1	Mild generalised retinal arteriolar narrowing or sclerosis
Grade 2	Definite focal narrowing and a/v crossing changes Moderate to marked sclerosis of the retinal arterioles Exaggerated arterial light reflex
Grade 3	Retinal h'ages, exudates and cotton wool spots Sclerosis and spastic lesions of retinal arterioles
Grade 4	Grade 3 plus papilledema

Modified Scheie Classification

Grade 0	No Changes
Grade 1	Barely detectable arterial narrowing
Grade 2	Obvious arterial narrowing with focal irregularities a/v crossing changes (<u>salus sign</u>)
Grade 3	Copper wiring of arterioles. Venous banking distal to A-V crossing (<u>bonnet's sign</u>) Venous tapering on either side of crossing (<u>gunn's sign</u>) Right angle deflection of veins. Flame shaped hemorrhages cotton wool spots, hard exudates.
Grade 4	Grade 3 plus optic disc swelling

History & Symptoms



RETINOPATHY KEY FEATURES

Narrowing and irregularities of retinal arteries

AV nicking

Blot retinal haemorrhages

Cotton wool spots

Management

Mild HT

BP control only

Moderate HT

BP control
+
Assess cholesterol levels & if
indicated cholesterol
lowering agents

Accelerated HT

Urgent anti hypertensive
management by stepwise
control of BP over a few hours

What is malignant HT?

What are cotton wool spots?

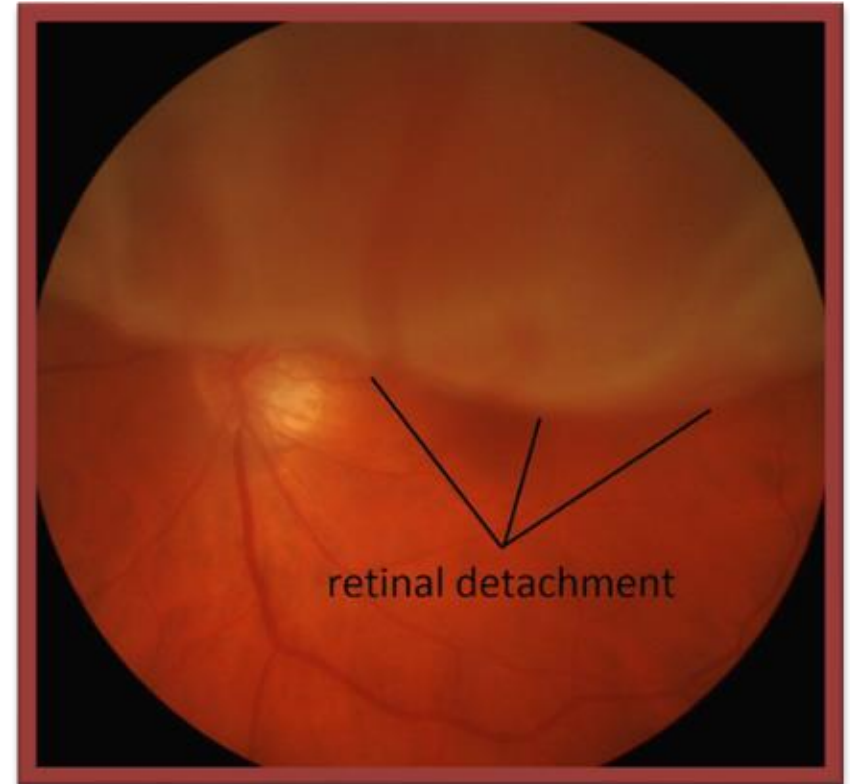
What is the normal AV ratio

What is papilledema?

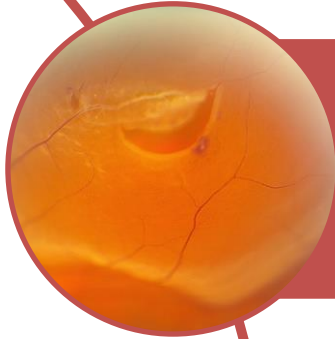
What is silver wire appearance

Retinal Detachment

Rhegmatogenous Retinal Detachment is defined as the **separation** of the **neurosensory** retina from the **retinal pigment epithelium**, which is caused by a retinal break with subsequent shift of intra-ocular fluid and liquified vitreous into the subretinal space.



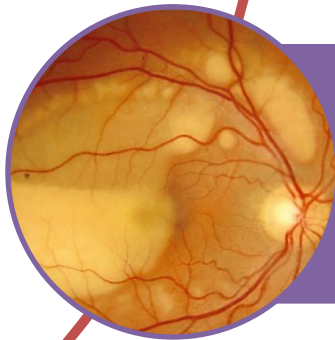
Classification



Rhegmatogenous



Tractional



Exudative

Pathophysiology of RD

Rhegmatogenous Retinal Detachments - are caused by a tear or break in the retina associated with posterior vitreous detachment or trauma.

Tractional retinal detachment occurs when proliferative membranes on the surface of the retina or vitreous. These membranes can pull on the neurosensory retina causing a physical separation between the neurosensory retina and retinal pigment epithelium

Exudative detachment, is due to accumulation of subretinal fluid d/t inflammatory mediators or exudation of fluid from a mass lesion. It could be d/t idiopathic, inflammatory, neoplasm or metastatic and infectious causes. Sometime systemic causes e.g. Toxemia of pregnancy

Risk Factors for Rhegmatogenous RD

Pathologic
Myopia

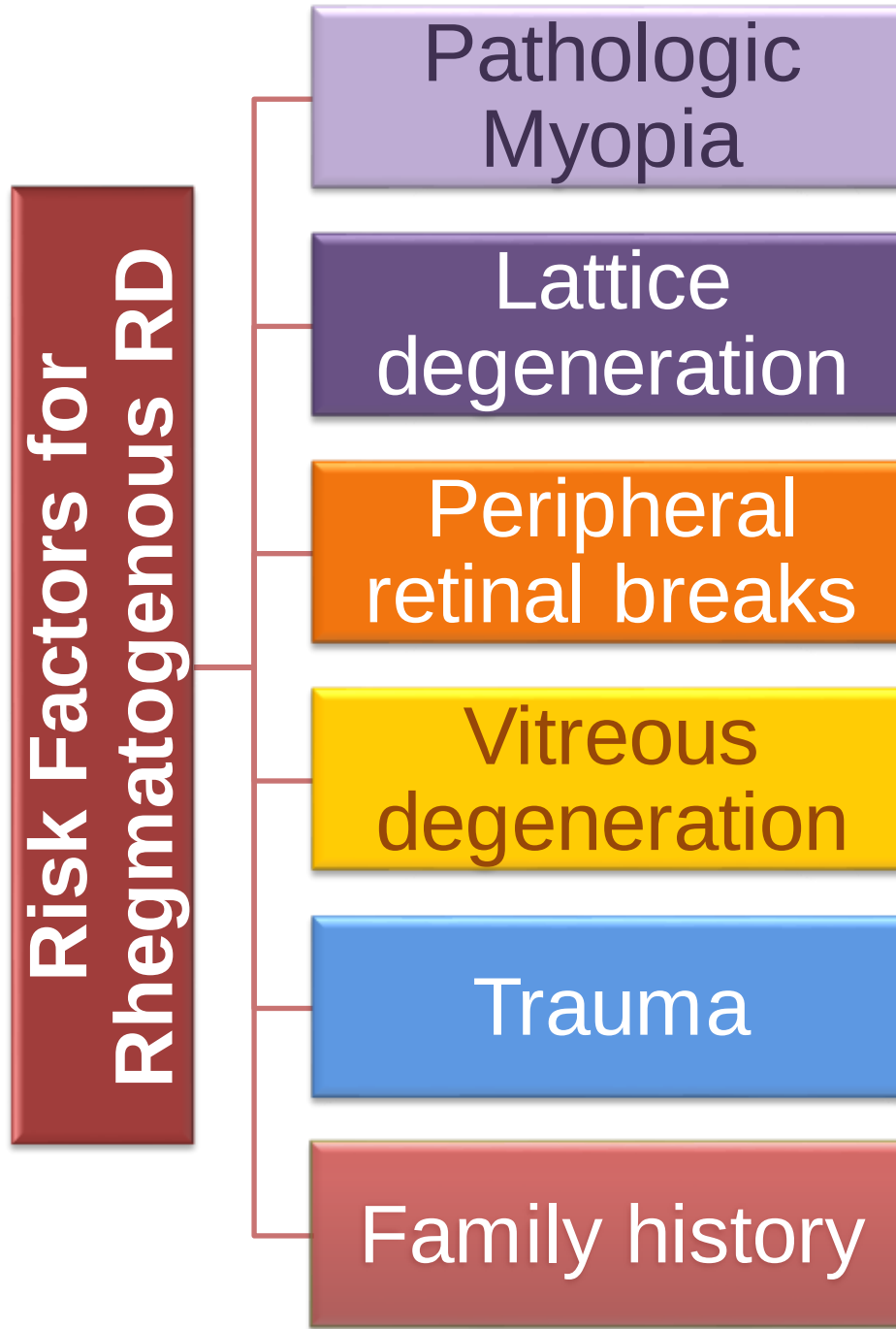
Lattice
degeneration

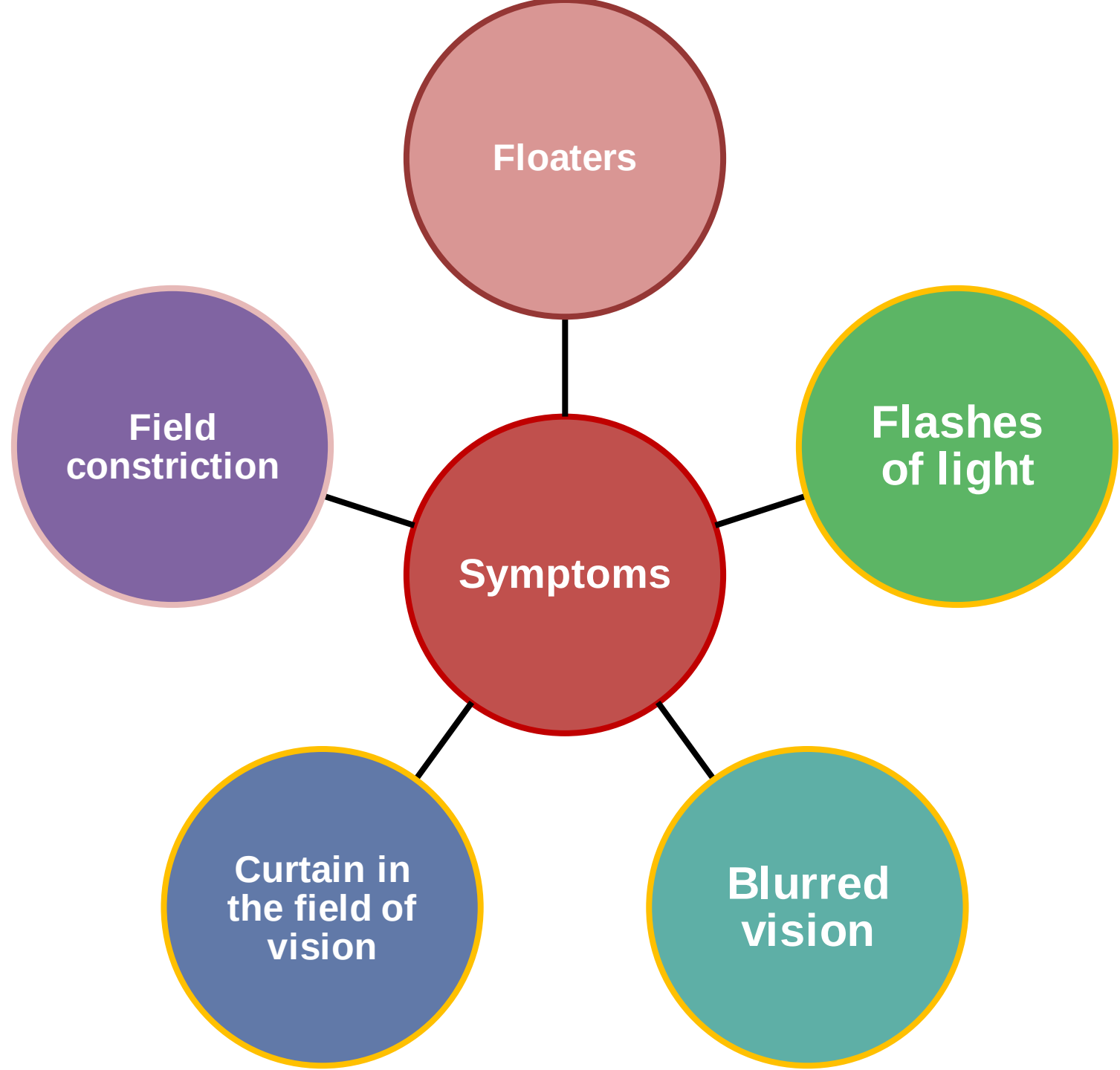
Peripheral
retinal breaks

Vitreous
degeneration

Trauma

Family history





Low IOP
&
RAPD

Mild iritis

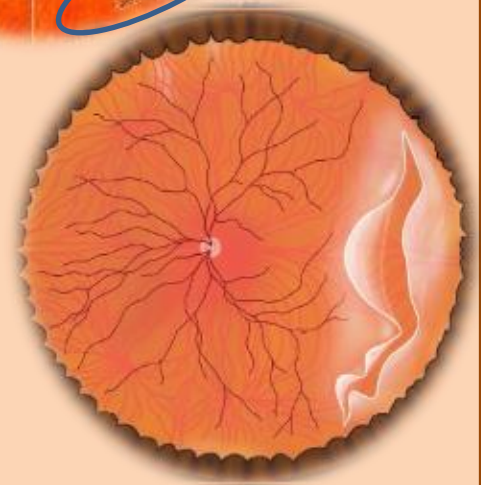
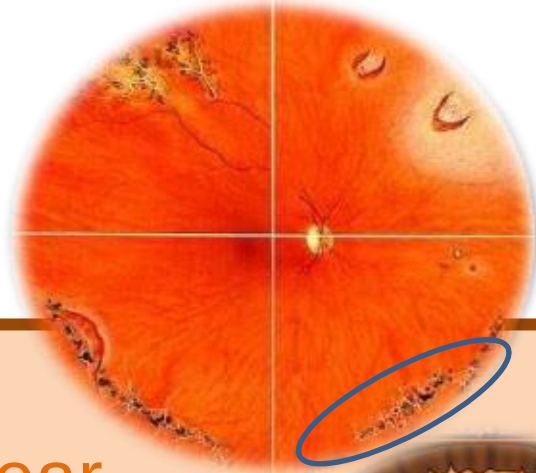
Signs

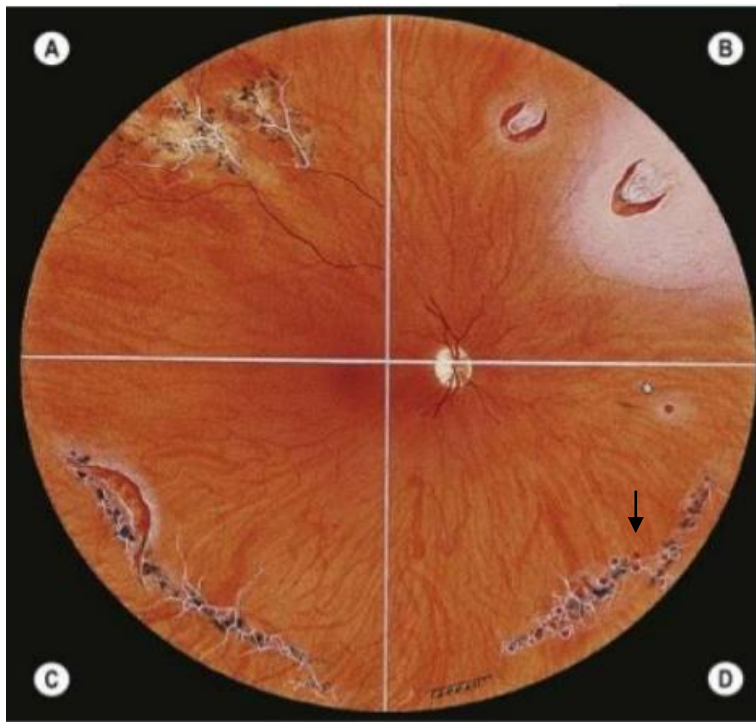
Grey detached
retina & Retinal tear

'Tobacco
dust'[shafer's sign]

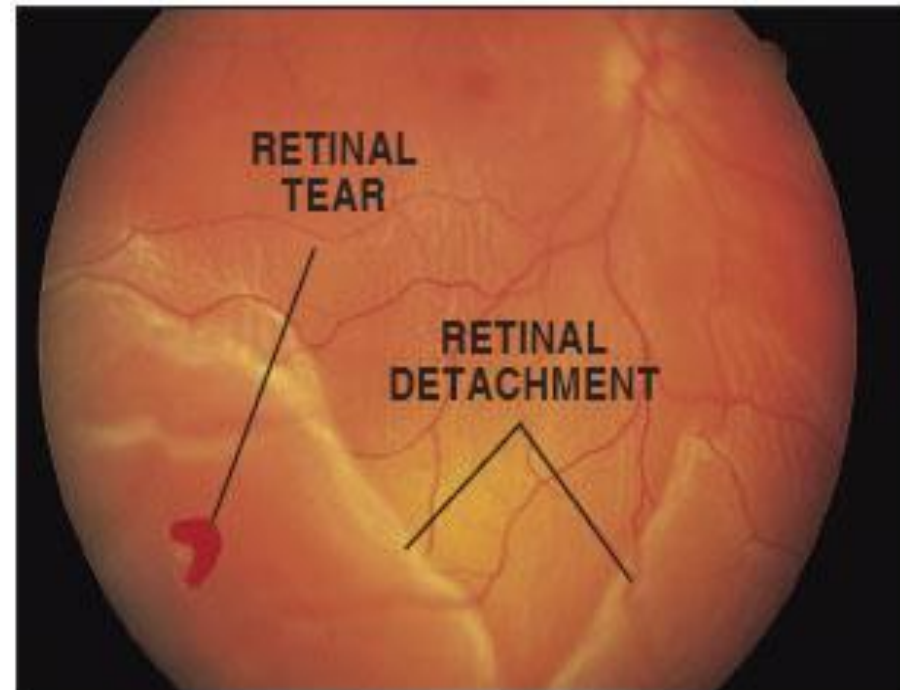
Types of Break

1. Horseshoe or U-shaped tear
2. Atrophic hole
3. Operculated
4. Dialysis
 - a) Involves splitting of vitreous base
 - b) Usually inferotemporal
 - c) Can be spontaneous or traumatic
5. Macular hole
 - a) Idiopathic; myopia; Commotio retinae





- A. Atypical radial lattice without breaks
- B. B. U-tears
- C. Linear tears
- D. Multiple holes within islands of lattice



Diagnosis

Visual Acuity



Fundoscopy both Direct and Indirect



Refraction, IOP



Slit-lamp Ex and use 90D lens for fundus Ex



Fundus photography, USG B Scan

Principles of Treatment

- Localisation & closure of breaks
- Relief of vitreoretinal traction
 - Buckling or vitrectomy
- Neuroretinal-RPE adhesion
 - Photocoagulation or cryotherapy
- Internal tamponade
 - Air; longer acting gases [SF₆, C₃F₈];
 - Silicone oil

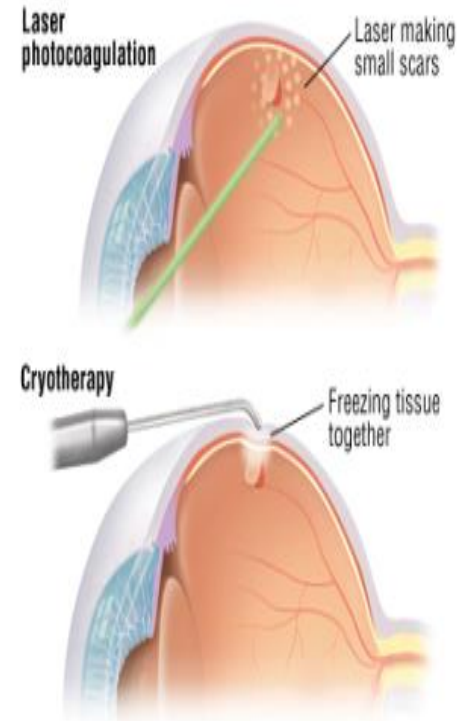
Treatment for RD
requires a surgical
intervention.

**Surgical
reattachment**
involves
indentation of the
sclera to relieve
pressure on the
retina, allowing it
to re-attach.

- Laser photocoagulation
- Cryopexy
- Scleral Buckling
- Vitrectomy
- Pneumatic retinopexy

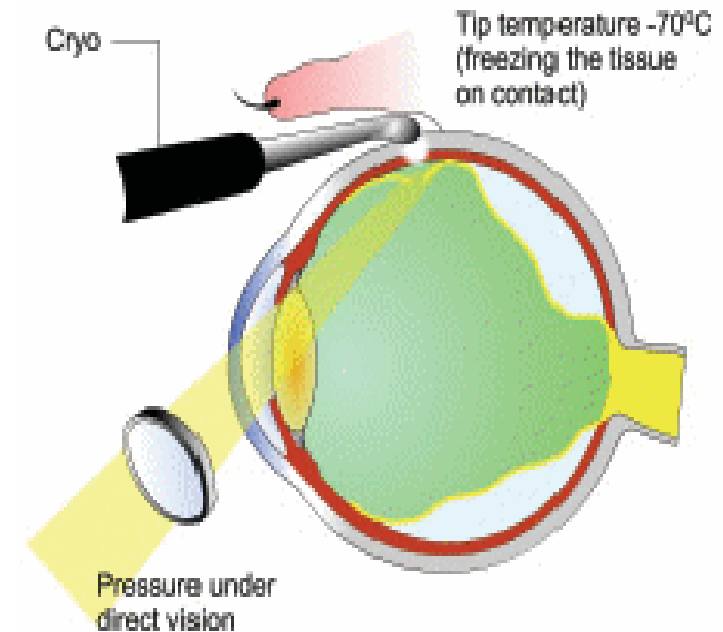
Laser (Photocoagulation)

1. Used to seal the tears or holes in the retina before retinal detachment
2. The laser burns around the tear site, and the resulting scarring affixes the retina to the back of the eye



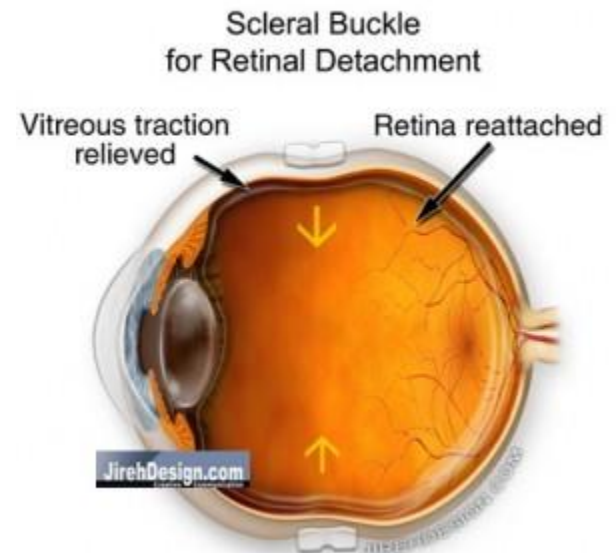
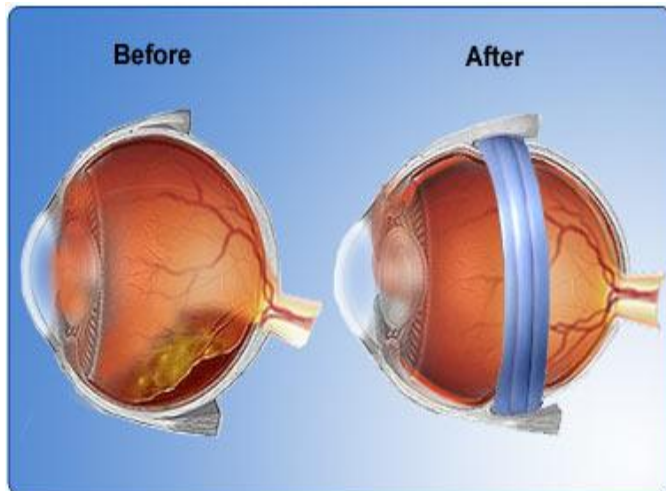
Cryopexy

1. Is the application of intense cold with an ice probe leading to the formation of scar that holds the retina to the underlying layer.
2. This technique is used in the combination with the injection of a gas bubble and the maintenance of a specific head positions to prevent the accumulation of fluid behind the retina.



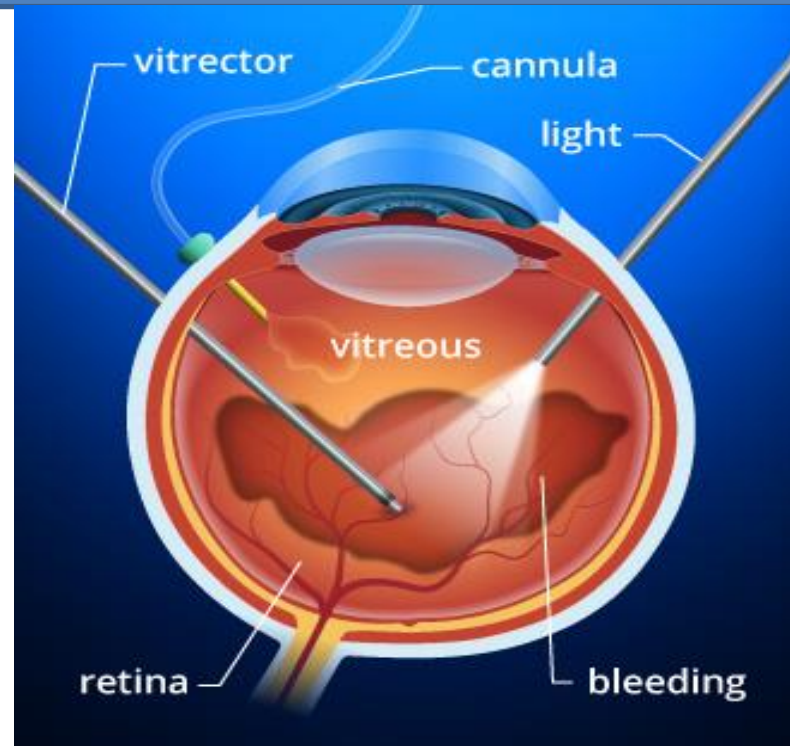
Scleral buckling

1. Is a procedure where the causative retinal tear are located and treated.
2. A flexible piece of silastic is then sewn to the sclera to support the area of the tears and detachments. Fluid may be drained from under the detached retina.



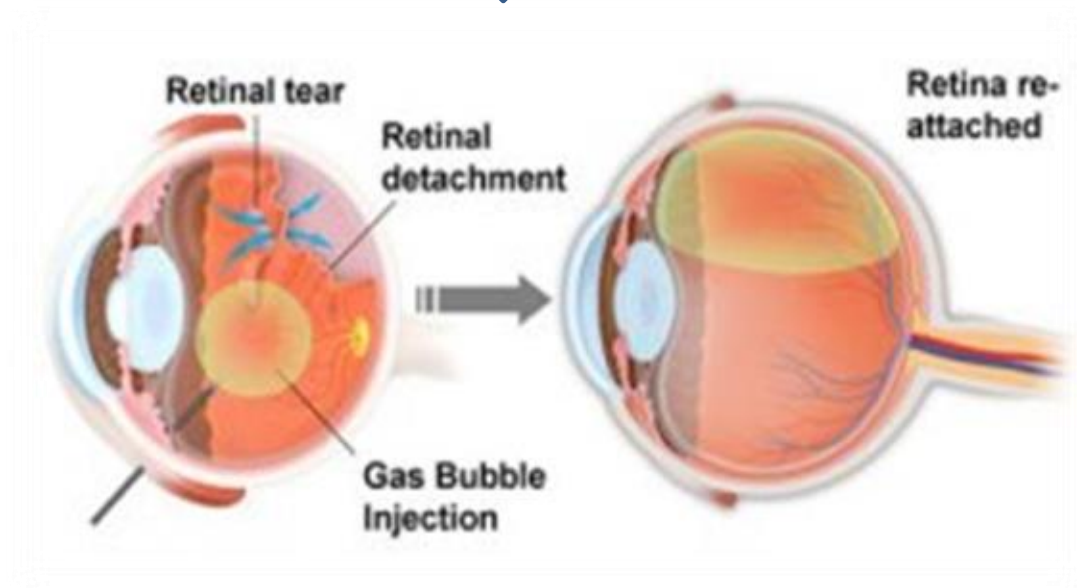
Vitrectomy

1. Is used for detachment with unusual or difficult features, such as very large tears, scar tissue on the retina, excessive blood in the vitreous, or detachments that failed by other methods.
2. The surgeon removes the vitreous along with any tissue that is tugging on the retina. Air, gas or silicone oil is then injected into the vitreous space to help flatten the retina.



Pneumatic retinopexy

1. This method is not suitable for all types of detachment.
2. The causative tear is identified and treated.
3. A bubble of a special gas is then injected into the eye.
4. The gas is used to push against the area of the retinal tear.



What is retinal Detachment?

Types of RD?

Can you recall some symptoms?

How are retinal tears treated

What is retinal pneumopexy



Retinitis Pigmentosa

Aetiopathogenesis

- There are no known aetiology for RP other than genetic predisposition. RP may occur as an isolated sporadic disorder, or be inherited as autosomal dominant, autosomal recessive or X-linked
- Predominant damage to photoreceptor cell death by apoptosis (rods followed by cones)
- Rods, which provide side (peripheral) and night vision are affected most

Classification

- **According to fundus appearance:**
- **Retinitis pigmentosa sine pigmento** - RP without signs of intraretinal pigmentosa. In almost all cases these represent early RP
- **Retinitis punctata albescens** - in early stage of RP fine, whitish, punctate lesions in the mid & far periphery

Symptoms

- By age of 30 year over 70% patients are symptomatic
- The first complaint is difficulty in night vision
- Prolonged time to adjust to dim light
- Peripheral contraction of vision as if they are in a tunnel (thus the term tunnel vision)
- Inability to differentiate colors

Signs

- Decreased vision may vary from 6/6 to HM
- Pupil may be normal or RAPD may be present
- Adult patients may develop post. subcapsular cataract (PSC)
- Fundus changes

What It's Like



This is how a street scene looks with normal vision.

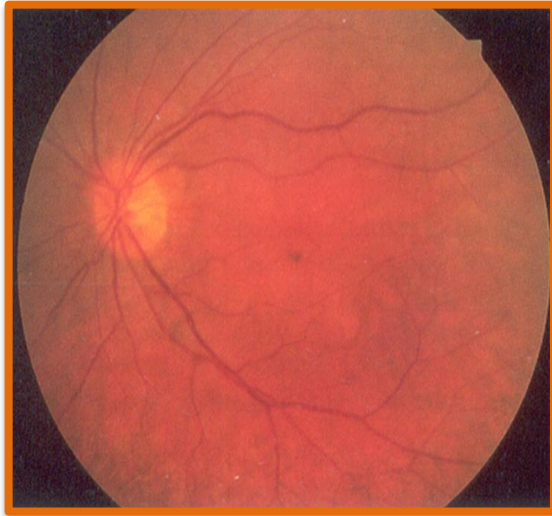


Example of Retinitis Pigmentosa

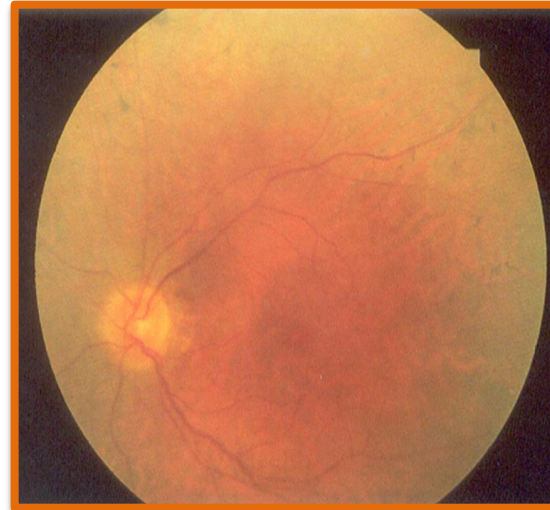
Fundus Changes

- **Arteriolar attenuation** - arteriolar narrowing, fine dust like intraretinal pigmentation & loss of pigment from RPE
- **Bone spicule pigmentation** - initially seen in mid retinal periphery
- **Waxy disc pallor** - least reliable sign of the retinitis pigmentosa triad. progressive contraction of visual field give rise to tiny island of central vision at this stage
- **Advanced features** - unmasking of choroidal vessels giving the fundus tessellated appearance, severe arteriolar attenuation,& marked optic disc pallor

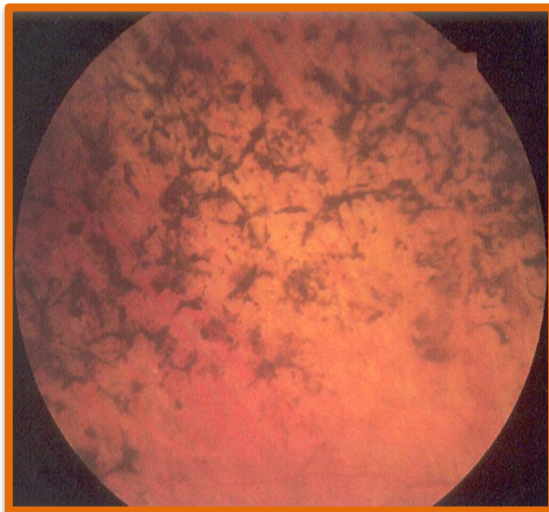
Very early RP



Early bony pigmentation



Dense pigmentary changes



Advanced RP



Treatment

Specific treatment:

- There is no known treatment for retinitis pigmentosa.

Supportive treatment:

- Aiding the patient by supplying useful information & support.
- Careful refraction.
- Cataract extraction when indicated.
- Treatment of macular edema.
- Referral for low vision aids (LVA).
- Periodic visual field examination.



Retinoblastoma

Retinoblastoma

Retinoblastoma is a proliferation of neural cells which have failed to evolve normally. **It is a primitive neuroectodermal intraocular malignancy that affects young children.**

The tumour is confined to infants and very young children and is frequently congenital. It presents from infancy to 5 years of age. The disease is rare.

Presentation

Leukocoria (Amaurotic cat's eye)

Strabismus (convergent)

Raised IOP

Pseudo-hypopyon, hyphema

Tumor seedings on Iris,

Iris neovascularisation

Orbital inflammation, Orbital invasion with proptosis

Bony involvement, metastasis in regional lymph nodes and brain

If left untreated retinoblastoma runs through the stages:

1. The quiescent stage, lasting from 6 months to a year
2. The glaucomatous stage
3. The stage of extraocular extension and
4. The stage of metastasis.

Early diagnosis and treatment of the tumor is essential for child survival and salvaging the eye to give useful vision

Treatment is by various modalities and is tailor made for each patient

.

