Identifying True Ocular Emergencies of the Posterior Pole

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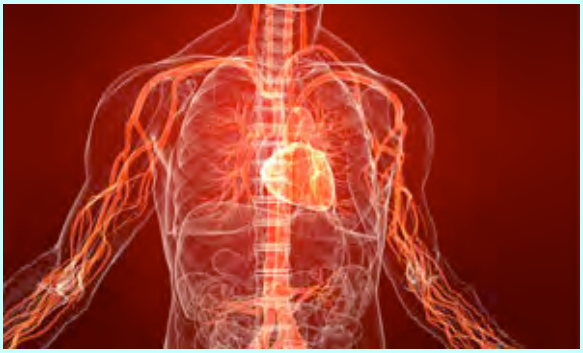
QUESTIONS

AND ANSWERS



Identifying True Ocular Emergencies of the Posterior Pole

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https://www.istockphoto.com/photos/center-city-philadelphia

7

Emergencies vs. Urgencies

- **Differentiate "Emergency" vs. "Urgency"**
- Proper Triage necessary (Front desk, Doctor away, After hours)
- Understand the " 10 A Club "

- PapillaedemA
- Giant Cell Arteritis
- Aneurysm
- Pituitary Apoplexy
- Carotid Artery Dissection
- Central Retinal Artery Occlusion
- PerforAted Globe
- Acute Angle Closure Glaucoma
- Acid / Alkaline Chemical Burn
- HyphemA

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CAUSES OF UNEXPLAINED VISION LOSS

RETINA

- **Foveal ischemia**
- **Macular Edema**
  - **CME**
  - **CSME**
- **Macular Hole**
- **Epiretinal membranes**
- **Central serous retinopathy**
- **Degenerative Myopia**
- **Macular Degeneration**

NEURO

- Functional Vision Loss
- Ocular Ischemic Syndrome
- Optic Neuropathy
  - Optic Neuritis
  - Ischemic Optic Neuropathy
- Visual Pathway Damage
  - Stroke
  - Tumor

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Emergency Triage

Mosby's Medical Dictionary defines triage as "a process in which a group of patients is sorted according to their need for care"

Triage, initially developed by military surgeons to deal with the large number of war casualties, is designed to ensure that patients with conditions requiring urgent treatment are seen first.

- Triage often begins with the telephone operator who is the first person to interact with patients needing care
- There are certain "red flags" that indicate a patient needs to be advised to present to the emergency service immediately for immediate triage and/care by a licensed practitioner:
  - Loss of vision within the previous 24-48 hours
  - Chemical injuries
  - Recent ocular (or head) trauma
  - Acute onset of ocular pain (with or without a red eye)
  - Foreign object in the eye

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Visual Disturbance

- It is very important to interview patients with visual disturbance very thoroughly, even in triage circumstances

  Temporal arteritis and CRA occlusion must be detected and managed ASAP to have a chance to retain vision

- The initial assessment should indicate the possible anatomic level of dysfunction

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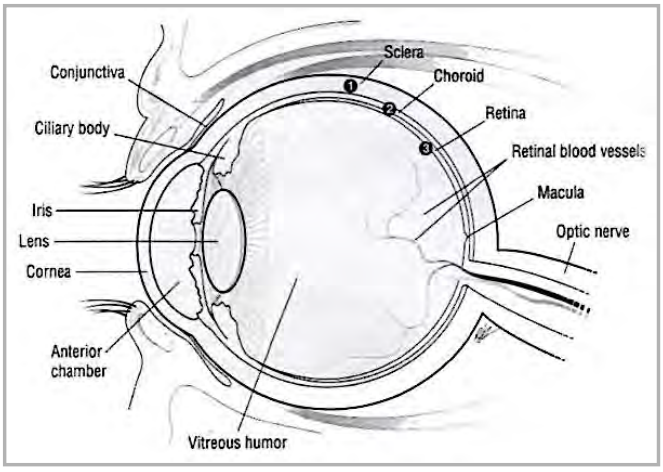
Emergencies – Anterior to Posterior



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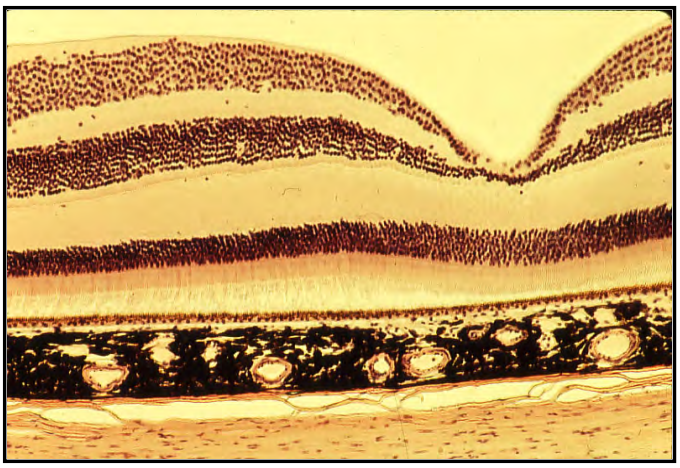
Retinal anatomy



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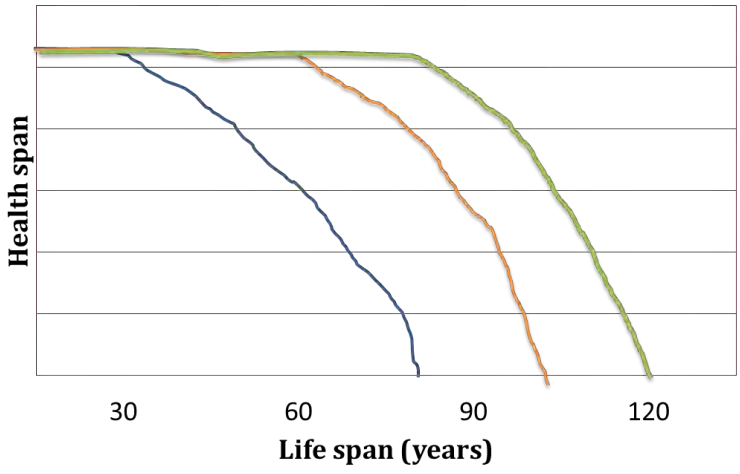
Longevity



Disease shifts the curve to the left

The goals for longevity:
Reduce chronic inflammation
and when inflammation/disease begin

Health span:

- Cognitive ability – Short term memory, processing speed, executive function
- Physical ability – muscle mass, bone density, freedom from pain and inflammation, sexual function
- Emotional decline (sense of purpose, relationships, social network support system)

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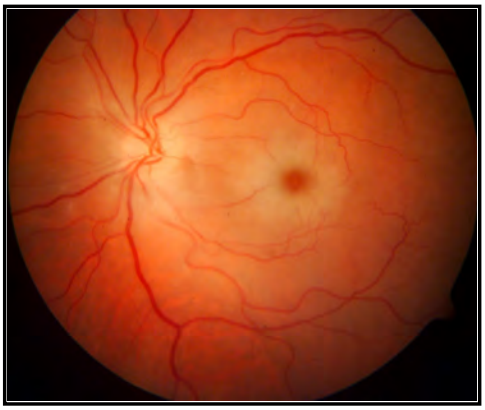
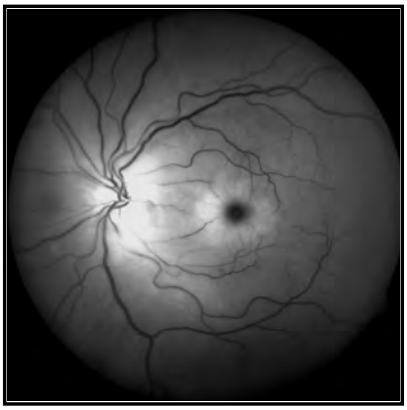
Central Retinal Artery Occlusion





- The initial visual acuity is count vision to hand motion in approximately 70 % of patients
- Central scotoma in the 30 degree visual field
- Visual acuity improvement occurs within the first 7 days and varies with the type of CRAO
- Site of occlusion in CRAO is where the artery pierces the nerve approximately 10 mm posterior to the lamina cribrosa

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CRAO consists of 4 categories

Non-arteritic CRAO

Transient non-arteritic CRAO

Transient impaction of an embolus
Fall in perfusion pressure below the critical point in the retinal vascular bed
Fall in the mean arterial pressure such as nocturnal hypotension
Increase in the intraocular pressure
Vasospasm of the central retinal artery

Non-arteritic CRAO w cilioretinal artery sparing

Arteritic CRAO with giant cell arteritis

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Central retinal artery occlusion facts

CRAO is found in 1/10,000 outpatient visits. Of these patients, 1-2% present with bilateral involvement.

Mortality/Morbidity

Life expectancy of patients with CRAO is 5.5 years compared to 15.4 years for an age-matched population without CRAO.

Sex
Men are affected slightly more frequently than women.

Age
The mean age of presentation is in the early 60s, although a few cases have been reported in patients younger than 30 years. The etiology of occlusion changes depending on the age of presentation.

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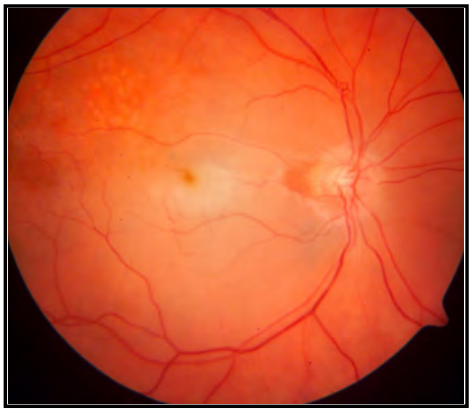
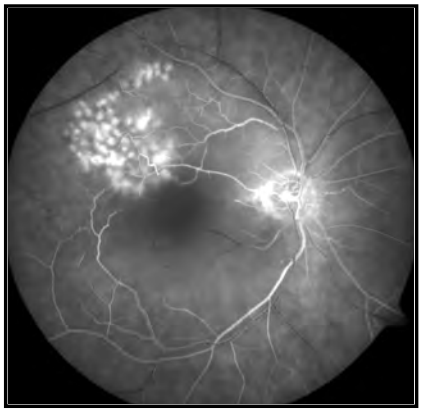
Central Retinal Artery Occlusion





- Embolism is the most common cause of CRAO and BRAO
- Plaque in the carotid artery is the most common source
- Aortic artery or the mitral valve are less common sources

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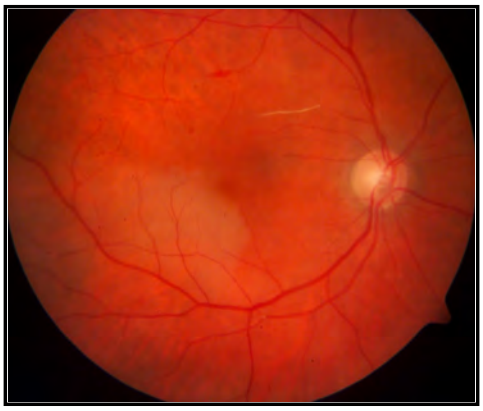
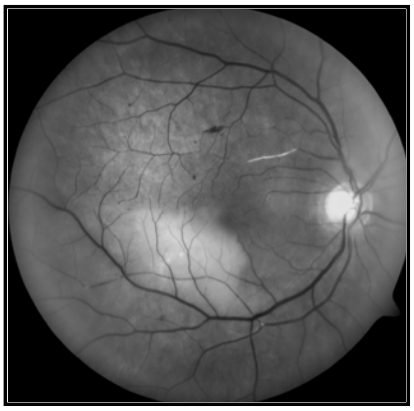
Branch Artery Occlusion





- Retinal arteries are in fact arterioles after the first branching in the retina.
- Their diameter near the optic nerve is about 100 um (the typical diameter of an arteriole)
- They do not have an internal elastic lamina nor a continuous muscular coat
- GCA affects medium and large arteries therefore GCA can not be a cause of BRAO

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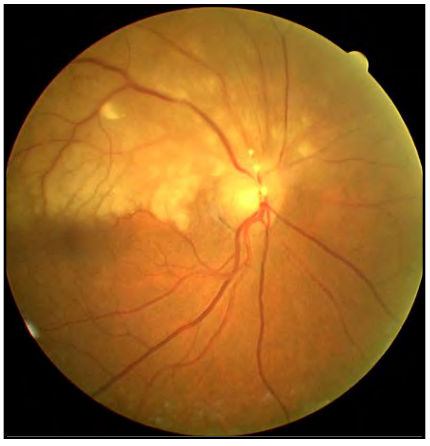
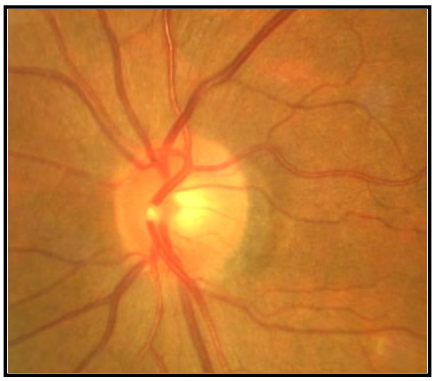
Branch Artery Occlusion





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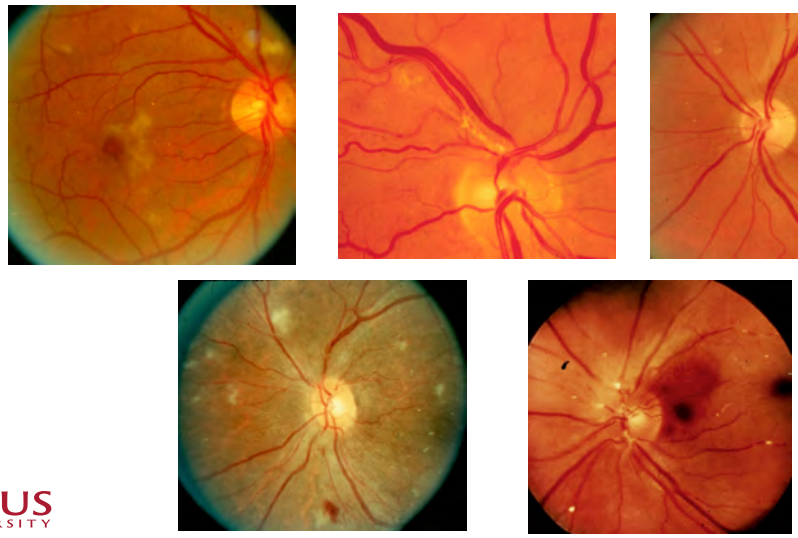
Branch Artery Occlusion



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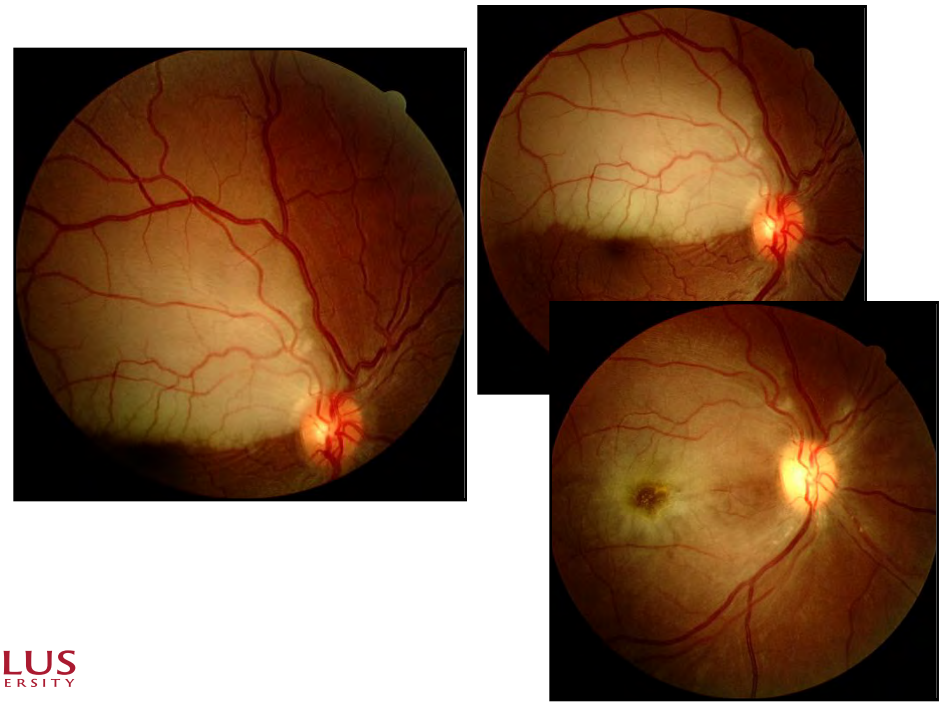

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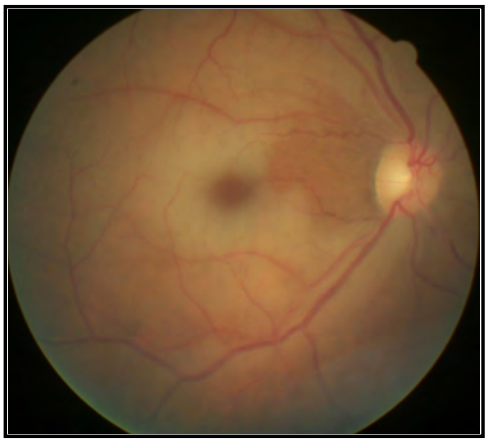
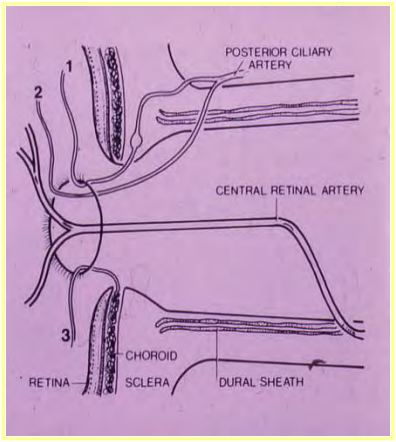
Blood supply to the retina





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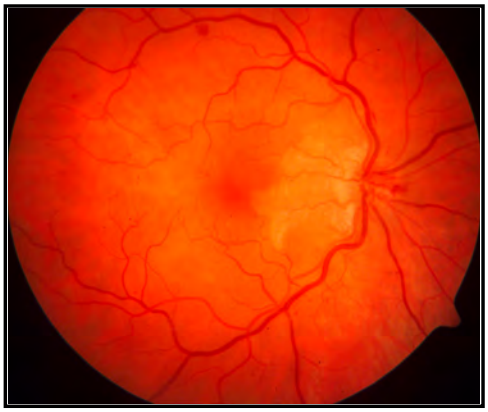
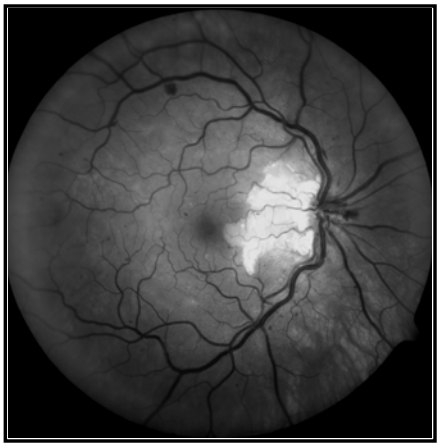
Cilio-retinal artery occlusion





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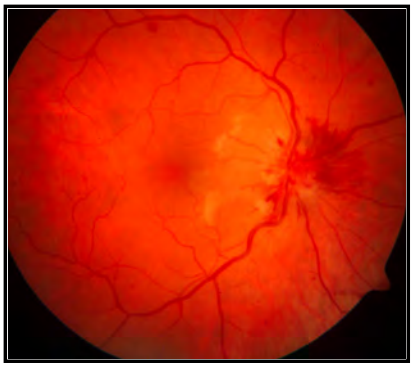
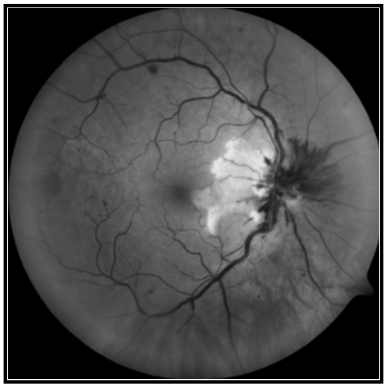
Cilio-retinal artery occlusion – think GCA

Cilio-retinal artery occlusion found in GCA has erroneously been diagnosed as BRAO.

Branch retinal arteries are in fact arterioles and therefore GCA can not cause BRAO since the process attacks medium and large arteries





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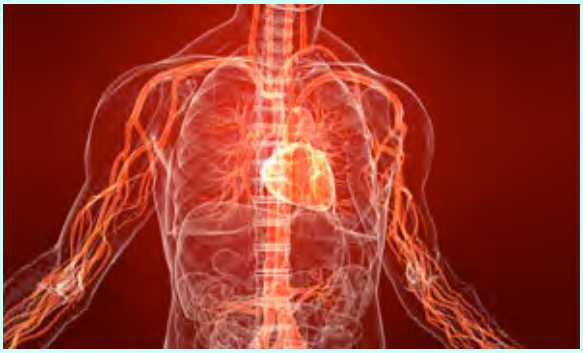
25

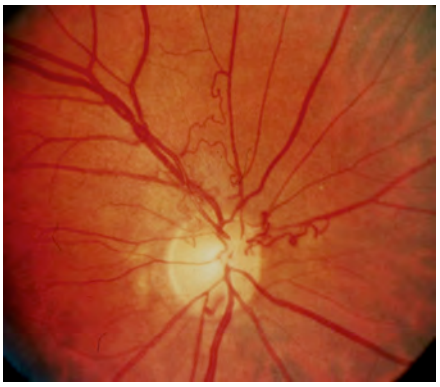
QUESTIONS

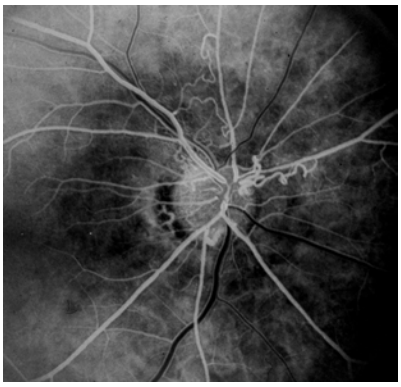
AND ANSWERS



Optic Nerve Arterial Collateral Vessels after CRAO

- Rare in chronic or slowly progressive CRAO
- May spare visual acuity and visual field





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Presentation

- Superficial retinal whitening with a "cherry red spot" in the fovea
- Retinal whitening within 1hour (97 minutes) from ischemic necrosis
- CRAO lasting more than 4 hours leads to massive irreversible retinal infarction
- Narrowed retinal arterioles with segmentation
- Cilio-retinal artery may spare the fovea
- Relative Afferent Pupillary Defect
- Optic nerve pallor weeks to months later

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Etiology

- Arterial emboli (cholesterol, fibro-platelet, calcific) seen in 20% of cases
- Thrombus formation secondary to arteriosclerosis
- Arteritis – Giant cell arteritis, Lupus, Polyarteritis nodosa
- Blood dyscrasias (hypercoagulation disorder), sickle cell
- Migraine
- Optic nerve drusen
- Atrial fibrillation, homocysteine, heart valves, oral birth control pills

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Treatment

- Immediate ocular massage (digital pressure) – 5 sec on / 5 sec off x 1 to 2 minutes
- High dose aspirin if possible (325 mg)
- Lowering of intraocular pressure(IOP) …. beta blocker, diamox, etc.
- Anterior chamber paracentesis
- Sublingual Nitroglycerin / Intra-Arterial fibrinolysis with (tPA)
  - 75% cholesterol emboli – fibrinolytics can not dissolve
  - 10% calcific material emboli – fibrinolytics can not dissolve
  - 15% fibrino-platelet emboli

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Follow up

- RTC at **1 month** to check for neovascularization of disc/iris
- RTC at **3 months** to check for neovascularization of disc/iris
- Neo of **iris** = 20 % of patients at about 4 weeks
- Neo of **disc** = 3 % of patients
- Extremely important to perform a complete **medical work-up** to stop the progression of the disease along with any systemic sequelae

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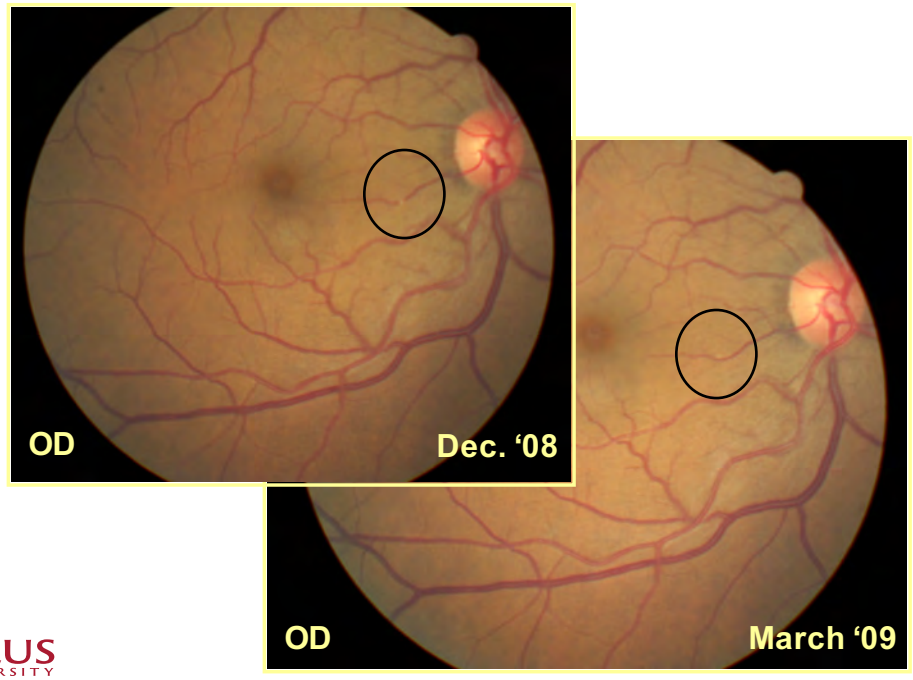

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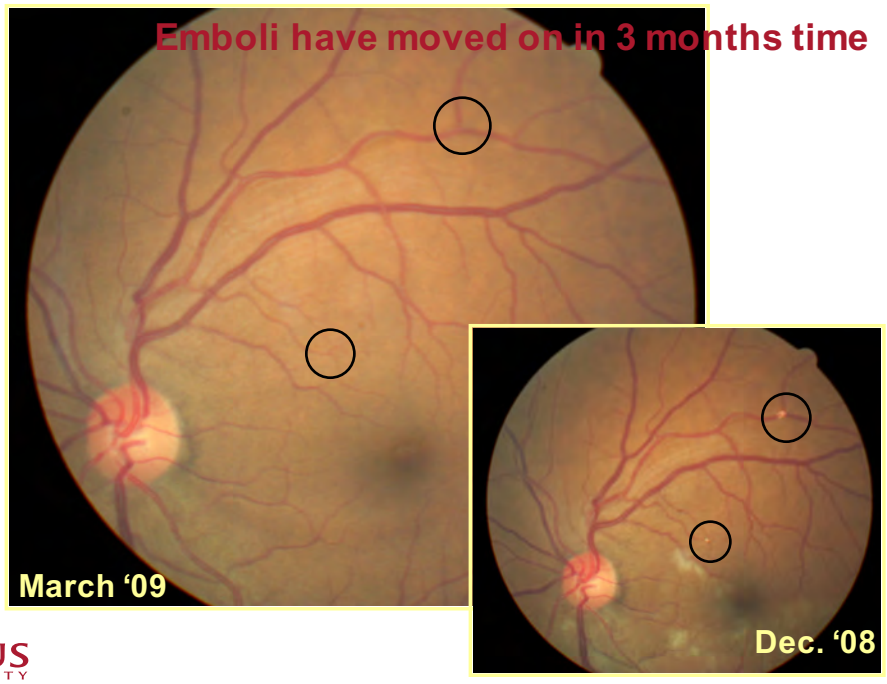

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Systemic Testing for CRAO

- Immediate Westergren Sed. Rate if patient is > 55 years old; R/O GCA
- Carotid artery evaluation, imaging of cervical and intracranial vessels (CTA)
  - The presence or absence of plaque is usually of much greater importance than the degree of stenosis
- Blood pressure, lipid profile, blood sugar
- CBC with platelets and coagulation studies
- Echocardiogram / EKG – cardiac evaluation
  - Transthoracic cardiac echography may show no abnormality - transesophageal may show better results
  - Transesophageal type of echocardiography is superior to the transthoracic type for cardiac abnormalities
  - Mitral valve, aortic valve, both valves, patent foramen ovale and a left atrial myxoma
- DWI-MRI within 24 to 48 hours

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What to do next ? Any TIA or Retinal Ischemia/Emboli treated the same!

Co-occurrence of Acute Retinal Artery Occlusion and Acute Ischemic Stroke: Diffusion-Weighted Magnetic Resonance Imaging Study

JUNWON LEE*, SEUNG WOO KIM*, SUNG CHUL LEE, OH WOONG KWON, YOUNG DAE KIM, AND SUK HO BYEON

Am J Ophthalmol 2014; 157: 1231-1238

¼ of patients with acute retinal ischemia (even if transient) had an acute brain infarction on brain DWI-MRI

10-15% of patients will have a disabling stroke within 3 months after a TIA, with half occurring within 48 hours after resolution of TIA.

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What needs to be done?

Do NOT send these patients to their PCP, cardiologist, neurologist, neuro-ophthalmologist, or retinal specialist.

Do NOT try to obtain the work-up yourself.

Send the patient to an ED (emergency department) with an Acute Stroke Care Center!

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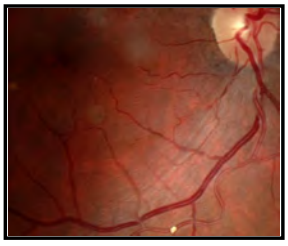
Causes of embolism

I Cardiac Disease

- Arrhythmias
- Valvular Disease
- Endocarditis
- Ischemic lesions
- Tumors

II Carotid Disease



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Stroke and central retinal artery occlusion

The risk factors for CRAO are the same atherosclerotic risk factors as for stroke and heart disease.

Individuals with CRAO may be at risk of ischemic end organ damage such as a cerebral stroke.

The management of CRAO is not only to restore vision, but at the same time to manage risk factors that may lead to other vascular conditions.

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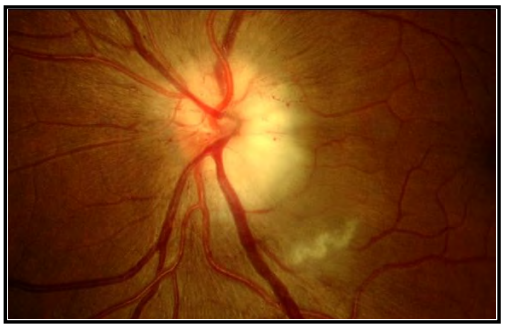
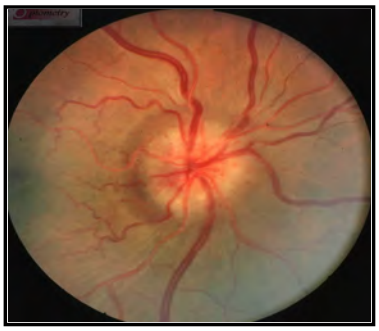
Arteritic Anterior Ischemic Optic Neuropathy

Pale swelling of the optic nerve





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Etiology and Ocular Presentation

Occlusion of the "short posterior ciliary" arteries with giant white blood cells

- Acute painless vision loss (vision loss is usually permanent) CF to NLP in 65% of patients
- Pale swelling of the optic nerve head with flame shaped hemes
- Central retinal artery occlusion may occur
- Cranial nerve palsy (CN 3,4,6) may also be present, cotton wool spots

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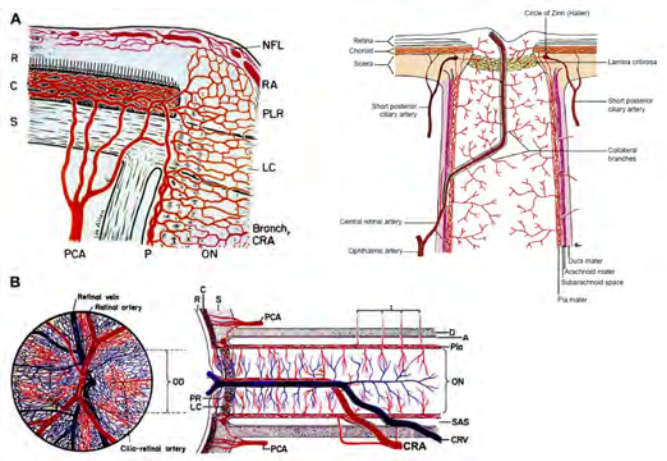
Optic Nerve Blood Supply



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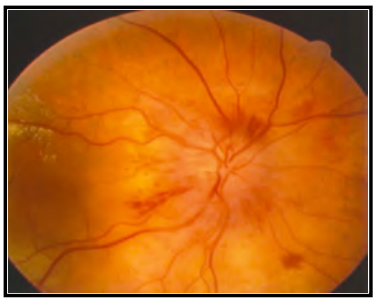
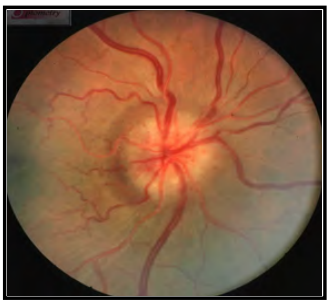
Arteritic Anterior Ischemic Optic Neuropathy

Must be differentiated from "Non-Arteritic" AION

Sudden painless loss of vision – amaurosis fugax is an early sign of future vision loss
Females > Males (2:1 ratio) – one study showed 70% females and 30% males
Patients usually > 60 years of age





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Systemic Presentation and Labs -AAION

- Headache
- Scalp tenderness
- Jaw claudication
- Night sweats
- Weight loss
- Fever
- Polymyalgia rheumatica
- Depression

Immediate Erythrocyte Sedimentation Rate (Westergren ESR)

Immediate C –reactive protein (Acute Phase Reactant **) >2.45 mg/dl**

Platelet count (Thrombocytosis) = risk for permanent visual loss

CBC with differential = anemia of chronic inflammation

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Giant Cell Arteritis

GCA ranges from 10 to 30 cases per 100,000 in patients older than 50.

Scandinavian and Northern European descent have the highest incidence

Occlusive inflammatory process (middle and large size arteries)

Temporal or Occipital headache (80% of patients)

- The headache has no distinctive characteristics
- Unique headache
- New headache in a patient > 50 years of age, GCA ?

Pain and tenderness of the scalp, face or oral mucosa, jaw claudication

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Giant Cell Arteritis

White Blood Cells:

T-cells infiltrate arteries
Cytokines IL,TNF,IFN

- Temporal Arteritis
- Cranial Arteritis
- Granulomatous Arteritis

Attacks **medium** and **large** sized arteries
- Superficial Temporal Artery
- Coronary Artery
- Subclavian Artery
- Facial Artery

Possible association with **"Polymyalgia Rheumatica"** (PMR)
- Stiffness in the neck, shoulder, and hip
- **50%** of Giant Cell patients have PMR
- Is there a link between GCA and PMR ???

Cerebral Vascular Accident increased and recurrence of GCA can occur

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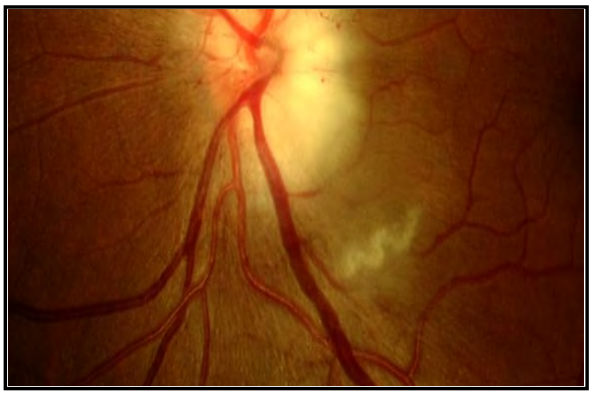
Arteritic Anterior Ischemic Optic Neuropathy



A normal ESR does not R/O GCA ; Normal in ~ **13%** of GCA

20% of GCA patients do not have systemic symptoms

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Arteritic Anterior Ischemic Optic Neuropathy

Temporal Arteritis
Cranial Arteritis
Granulomatous Arteritis

White Blood Cells
- T-cells infiltrate arteries
- Cytokines IL, TNF, IFN

Attacks **medium** and **large** sized arteries

- Superficial Temporal Artery
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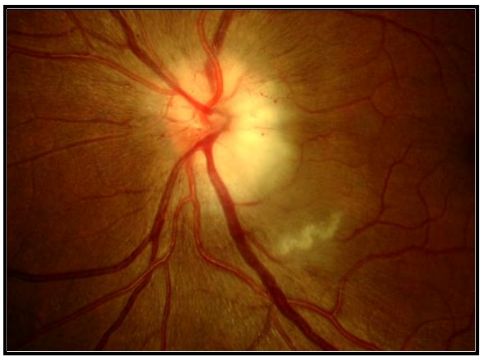
- Subclavian Artery
- Facial Artery



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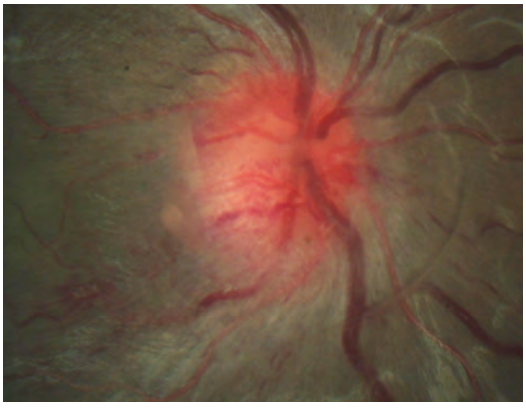
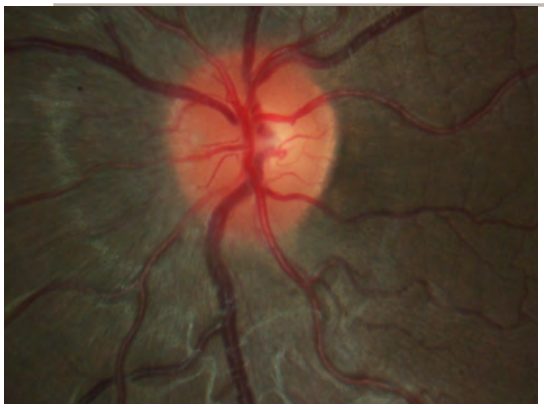
47

Hyperemic swelling of the optic nerve





Neuro-retinitis = catch scratch disease bartonella hensalae and clarridgeiae bacteria infection
The domestic cat is the primary host and the cat flea is the transmitting vector
Ciprofloxacin 500 mg bid and doxycycline 100 mg bid

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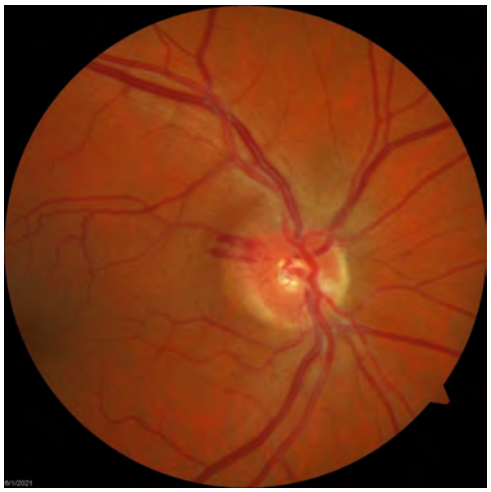
Hyperemic swelling of the optic nerve

The patient has an elevated white blood cell count and is in need of a bone marrow biopsy

Rule out NAION vs. infiltrative leukemic optic neuropathy

Visual acuity is 20/20 in each eye



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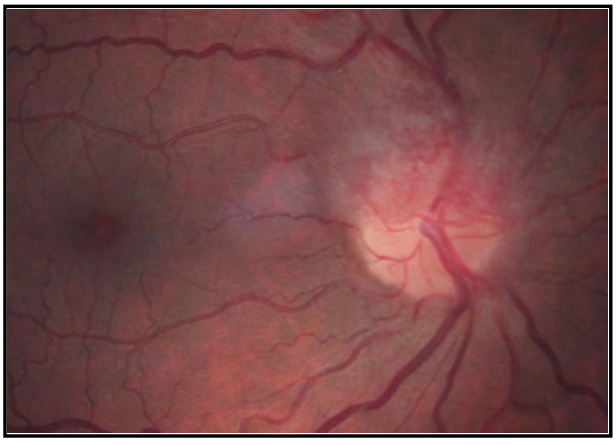
Hyperemic swelling of the optic nerve



Non arteritic AION

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Biopsy of Arteritic AION

- Possible temporal artery biopsy
- Done within a week of starting steroids
- Specimen is 2.5 cm long
- If biopsy is negative but suspicion high, then biopsy the opposite side
- ~ 13 % of cases will be positive on the opposite side

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Treatment of AAION

Signs and symptoms of GCA	**Treatment plan for AAION**
#1. Evidence of new-onset ischemia (CRAO, AAION, stroke, AF)	One or no clinical finding present – look at other causes
#2. Any new-onset headache, neck pain or scalp tenderness	Two finding present – start oral prednisone (1 mg/kg) and plan for temporal artery biopsy
#3. Abnormal laboratory results (ESR, CRP or platelet count)	Three or more findings present – start oral prednisone or high dose IV methylprednisolone (1 gram per day) and plan for temporal artery biopsy
#4. Jaw claudiction	
#5. Abnormal superficial temporal artery (beading, nodularity, absecne of pulse local tenderness	

Immediate IV steroid therapy – IVMP x 3 days / Oral Prednisone x 24 months

Methotrexate and/or Prednisone – for life

Always suspect recurrence

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AAION rule

The "One Third Rule" in Giant Cell Arteritis

- 1/3 of optic nerves in the fellow eye will become infarcted within 48 hours in untreated patients.
- 1/3 of optic nerves in the fellow eye will become infarcted within 1 month in untreated patients.
- Second eye infarctions are rare after more than 1 month

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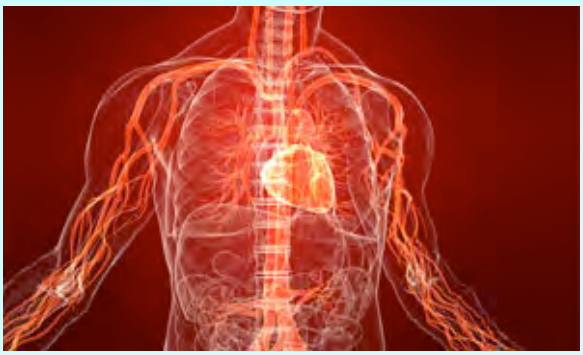
QUESTIONS

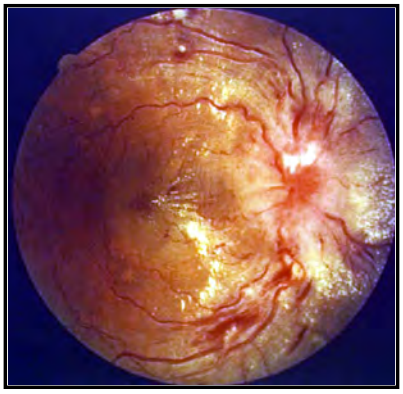
AND ANSWERS

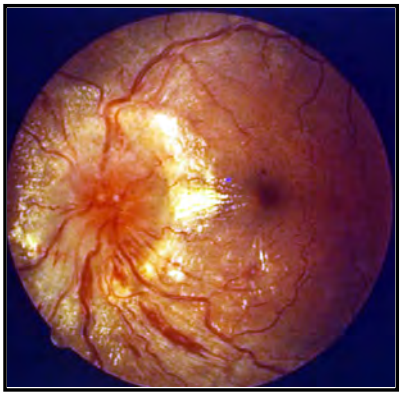


Malignant Hypertension

Immediate attention also if chest pain, cognitive impairment, hematuria, sensory or motor problems

"Malignant hypertension" defined as blood pressure > 180/120





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The definition of hypertension

	Systolic BP		Diastolic BP
Normal	<120 mmHg	AND	<80 mmHg
Elevated Blood Pressure	120-129 mmHg	AND	< 80 mmHg
Stage 1 Hypertension	130-139 mmHg	OR	80-89 mmHg
Stage 2 Hypertentions	>140 mmHg	OR	>90 mmHg
Hypertensive Crisis	≥180 mmHg	AND/ OR	≥120 mmHg
Hypertensive Emergency	Hypertensive Crisis Systolic and Diastolic #s + end organ damage URGENCY!!!		

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Types of hypertension

Essential Hypertension 90-95%

Secondary hypertension 5-10%

Sleep apnea
Medications (i.e., steroids, decongestants, stimulants, birth control)
Endocrine disorder (i.e., Cushing's disease, hypothyroidism, hyperthyroidism)
Adrenal gland (pheochromocytoma)
Renal disease (renal artery stenosis)
Illegal drug use (i.e., cocaine, narcotics, methamphetamine)

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Malignant Hypertension

Very high pressure with retinal hemorrhages, exudates, CWS or optic nerve swelling

Usually diastolic blood pressure is above 120 mm Hg

Hypertensive Urgency

Severe BP elevation >120 without retinopathy or CNS changes

Hypertensive Emergency

Severe BP elevation >120 with retinopathy or CNS changes

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Grading of hypertensive retinopathy

Stage	Description	Ocular symptoms	Systemic symptoms
1	Mild to moderate narrowing or sclerosis of arterioles	(-)	(-)
2	Moderate to marked narrowing of arterioles Focal or localized narrowing of arterioles Exaggeration of the light reflex Arteriovenous crossing changes	(-)	(-)
3	Retinal arteriolar narrowing and focal constriction Retinal edema Cotton wool spots Retinal hemorrhages Retinal exudates	(+)/(-)	Cardiac, renal or cerebral dysfunction evident
4	Stage 3 (+) optic nerve swelling Elschnig spots possible	(+)	Severe Cardiac, renal or cerebral dysfunction

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Grading of Hypertensive Retinopathy

Grade 1	Retinal vessels narrowed	> 90 and < 110 Diastolic BP
Grade 2	Nicking of retinal vessels	> 90 and <110 Diastolic BP
Grade 3	CWS, Hemes, Lipid exudates	> 110 – 115 Diastolic BP
Grade 4	Grade 3 + Optic disc swelling	> 120 Diastolic BP

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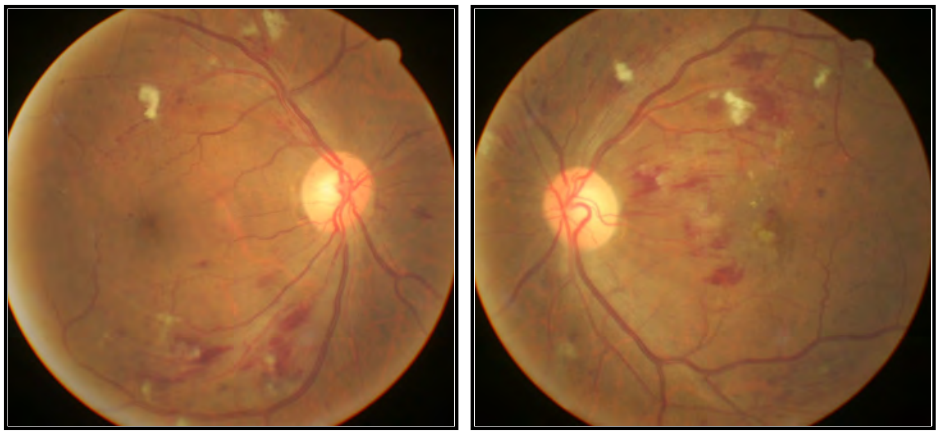
Hypertensive Retinopathy Grade 3



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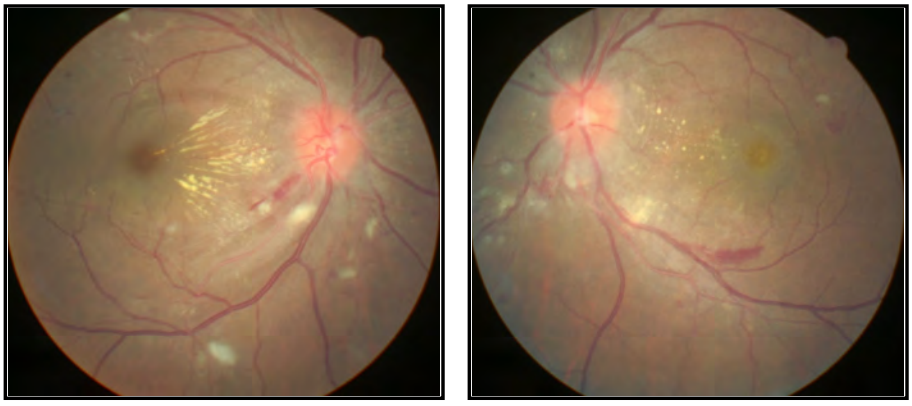
Malignant Hypertension



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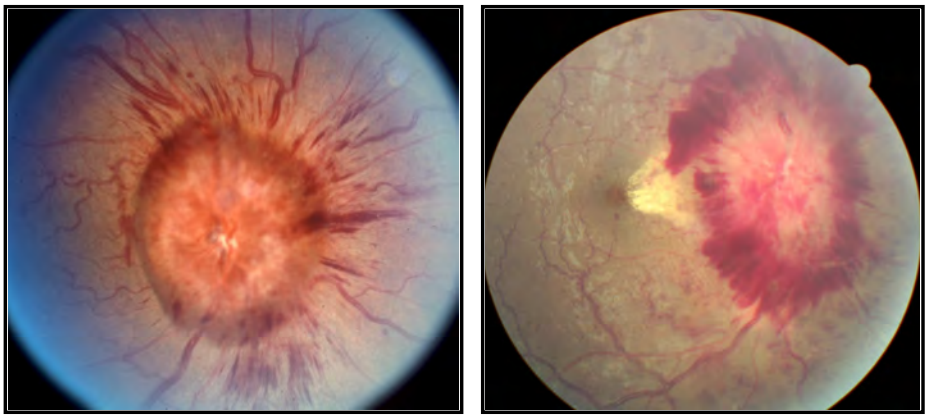
Malignant Hypertension



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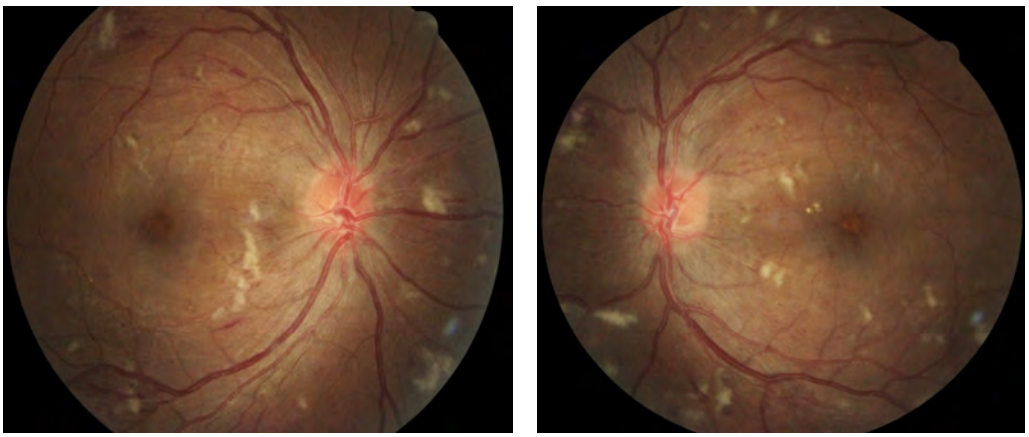
Malignant Hypertension



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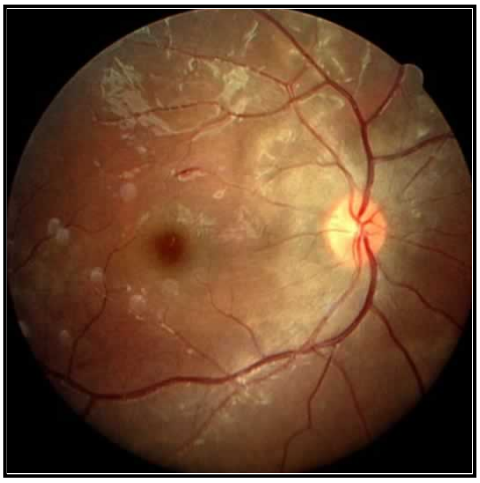
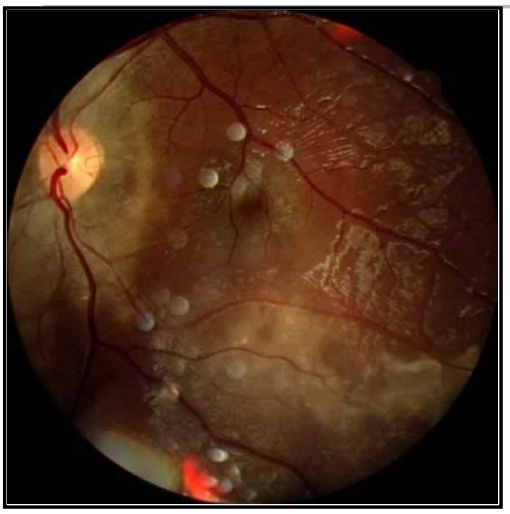
Hypertensive choroidopathy





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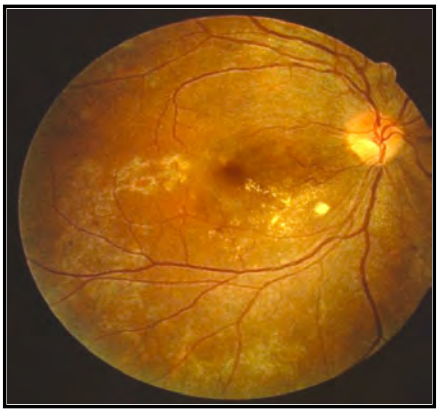
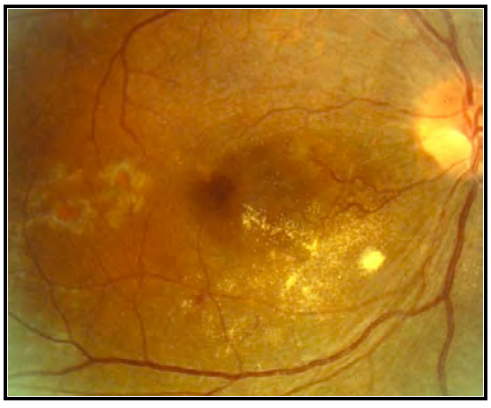
Hypertensive Choroidopathy

Elschnig spots



Siegrist streaks



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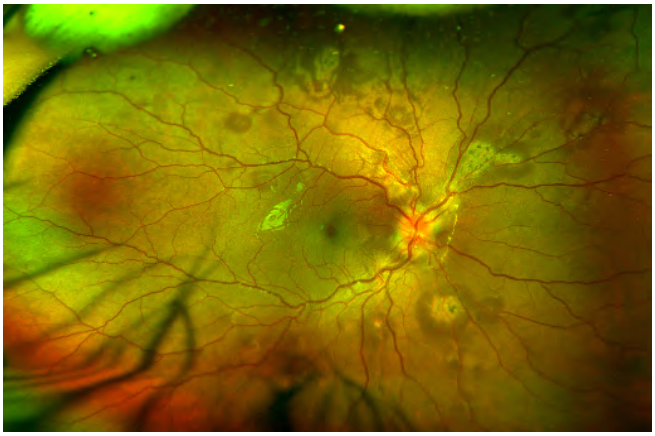
Hypertensive Choroidopathy

Elschnig spots



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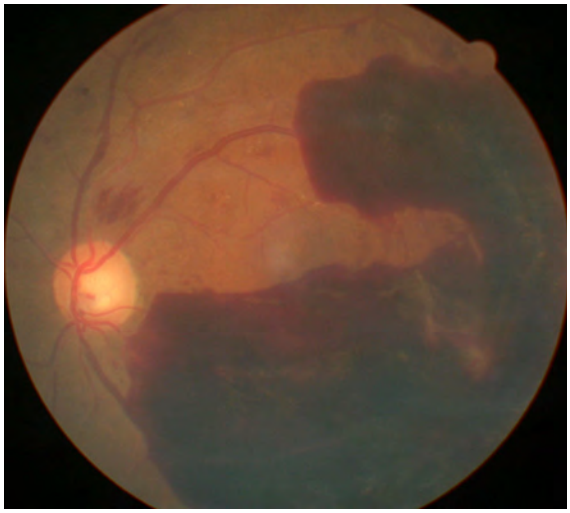
Retinal Arterial Macroaneurysm
Grade 3 hypertensive retinopathy



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Causes of Malignant Hypertension

- Collagen vascular disease, such as scleroderma (**pulmonary hypertension**)
- Kidney disease (**renal artery stenosis**)
- Spinal cord injuries
- Tumor of the adrenal gland (**pheochromocytoma**)
- Use of certain medications, including birth control pills
- Preeclampsia/eclampsia
- Coarctation of the aorta
- Use of illegal drugs, such as **cocaine**

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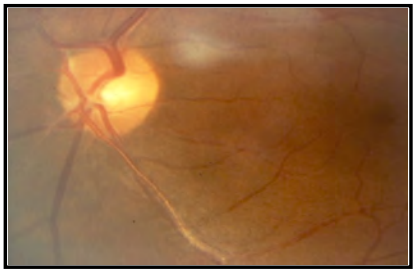
Malignant Hypertension

Hypertensive hemorrhages typically occur in areas where arteriolosclerosis is most severe:

Basal ganglia (60%)
Thalamus (10%)
Pons (10%)
Cerebellum (10%)



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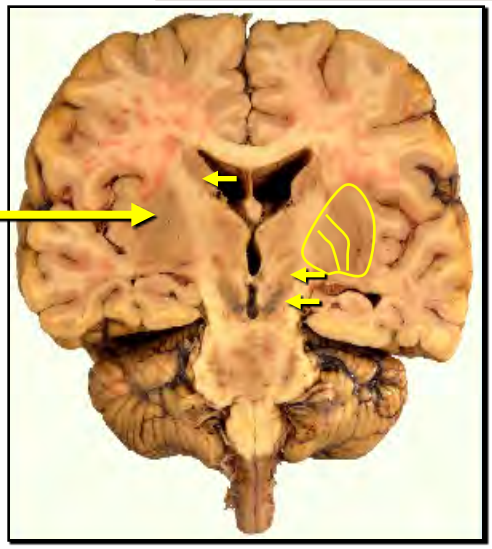
The Basal Ganglia

Hypertensive hemorrhages typically occur in areas where arteriolosclerosis is most severe:

Basal ganglia embedded in white matter



Much (not all) of basal ganglia are lateral to internal capsule

- Caudate
- Putamen
- Globus Pallidus

- Substantia nigra
- Subthalamic nucleus

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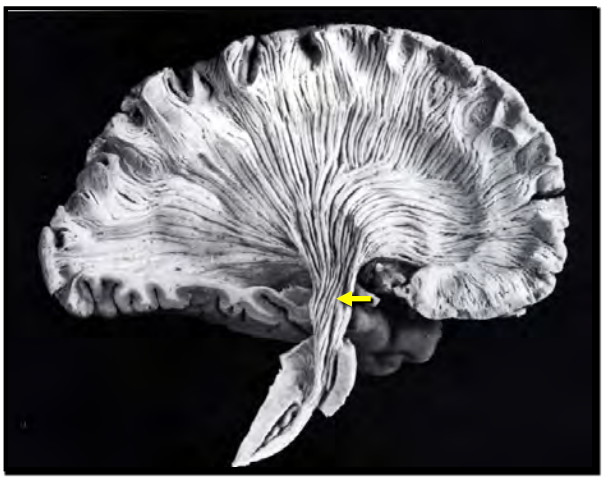
Internal capsule – could be effected in HBP

Internal Capsule (white matter)

Corticobulbars / corticospinals in crus cerebri of midbrain pons and medulla



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Treatment of Malignant Hypertension

Blood pressure measurement

Immediate referral to emergency room or PCP for **slow lowering** of the blood pressure

Severe BP elevation >120 without retinopathy or CNS changes **– Hypertensive Urgency**

Severe BP elevation >120 with retinopathy or CNS changes. **Hypertensive Emergency**

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Questions for malignant hypertension

Hypertensive encephalopathy

- Syncope
- Seizures
- Focal weakness
- Speech problems

Hypertensive renal problems

- Change in renal volume
- Hematuria, abdominal pain

Hypertensive cardiac involvement

- Chest pain
- Palpatations
- Cough
- Dyspnea

Organ dysfunction uncommon if DBP is less than 120 mmHg

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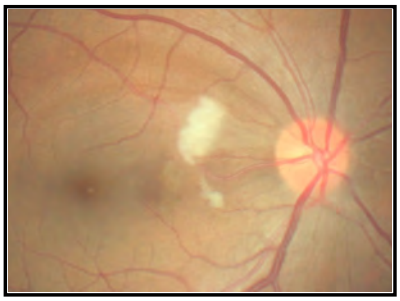
Treatment Medications

Vasodilators in the ER

- Clonidine
- Labetolol
- Nitroglycerine
- Esmolol
- Nitroprusside



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Complications of Malignant Hypertension

- Aortic dissection
- Coma
- Pulmonary edema
- Heart attack
- Heart failure
- Stroke
- Sudden kidney failure

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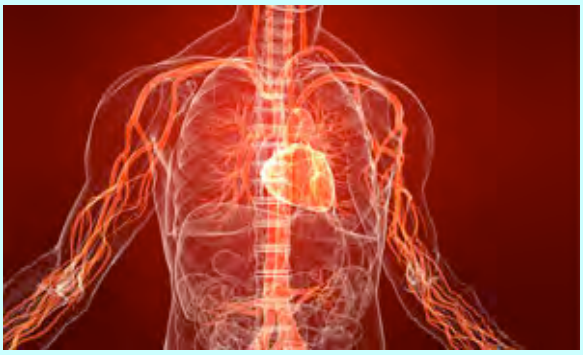
QUESTIONS

AND ANSWERS



Acute or Chronic Meningitis

Intense headache is followed by a stiffness of the neck that prevents passive flexion of the head on the chest

The headache is slightly less acute than subarachnoid hemorrhage. The headache develops gradually over hours

The headache at times is not the main feature. Look a the patients overall health presentation. The patient may **look ill**, has a **fever** and **nuchal rigidity**

The patient may also present loss of consciousness at times and **neurological deficits**

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Types of Meningitis

Bacterial
Viral
Fungal
Parasitic

Eye Signs and Symptoms:

Photophobia
Disc Edema

Diagnosis and Treatment:

MRI / CT followed by a lumbar puncture

Lumbar Puncture = identify the infectious organism

Treatment is with systemic antibiotics if bacterial

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Meningitis

Will cause pleocytosis = increase in white blood cells in the cerebral spinal fluid

Acute = Hours to days

Chronic = 4 weeks or more

Aseptic meningitis = No CSF bacteria found
(example: enterovirus)

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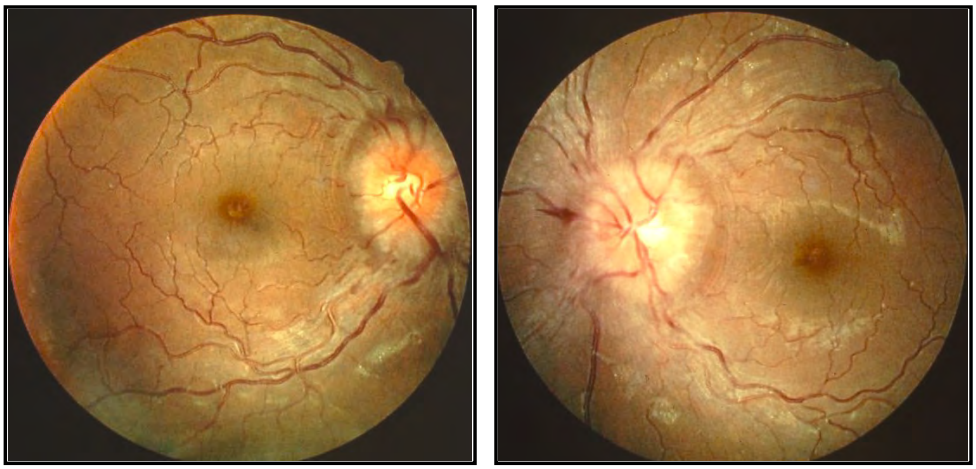
Meningitis



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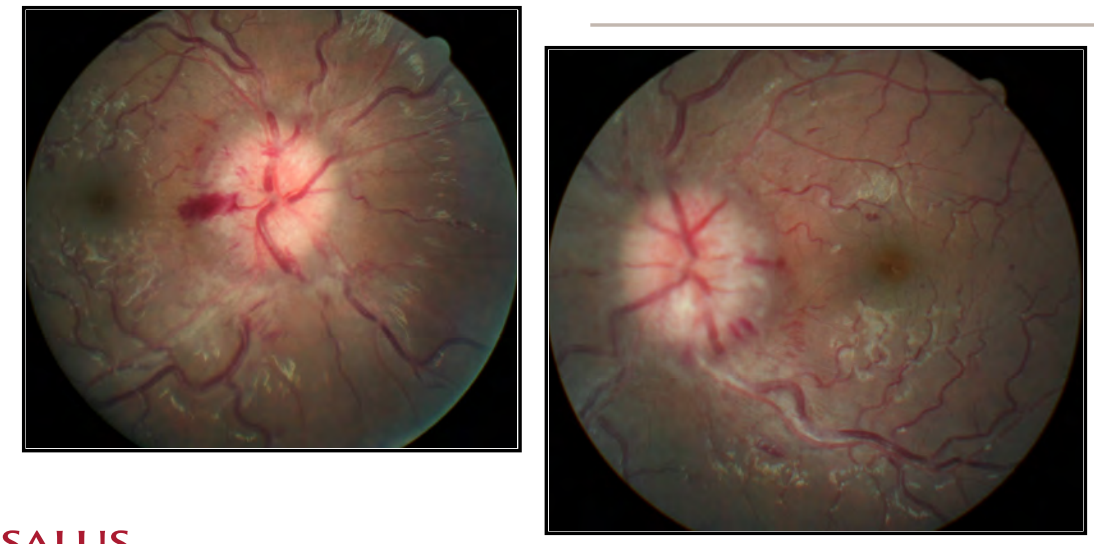
Meningitis



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Other causes of Meningitis

Lyme

Syphilis

TB

Listeria

Amoeba (swimming)

West Nile Virus

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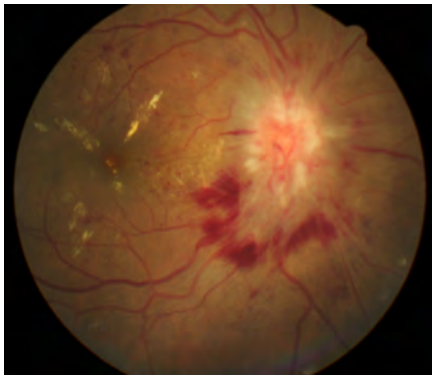
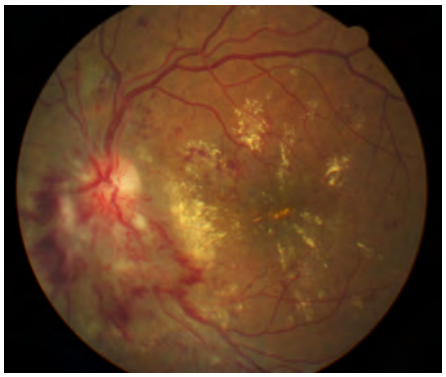
Psuedotumor Cerebri and Diabetic Papillopathy
Systemic History:

- DM x 14 years (known diabetic retinopathy)
- Hypercholesterolemia
- Iron deficiency anemia
- Proteinuria
- s/p MI





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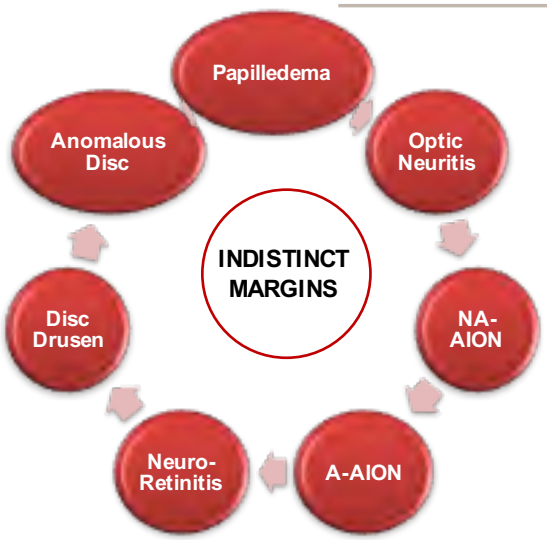
CAUSES OF INDISTINCT OPTIC DISC MARGINS



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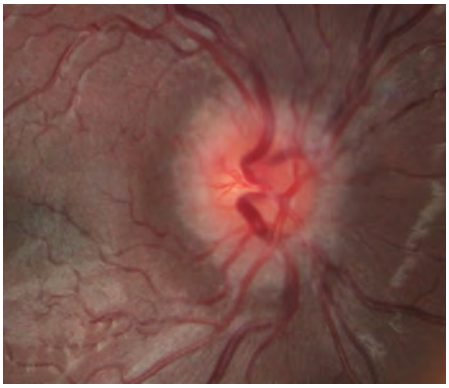
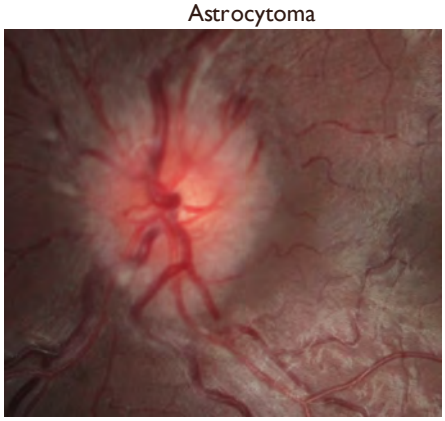
Bilateral optic nerve swelling



16 year old male

Left Temporal Lobe

Astrocytoma



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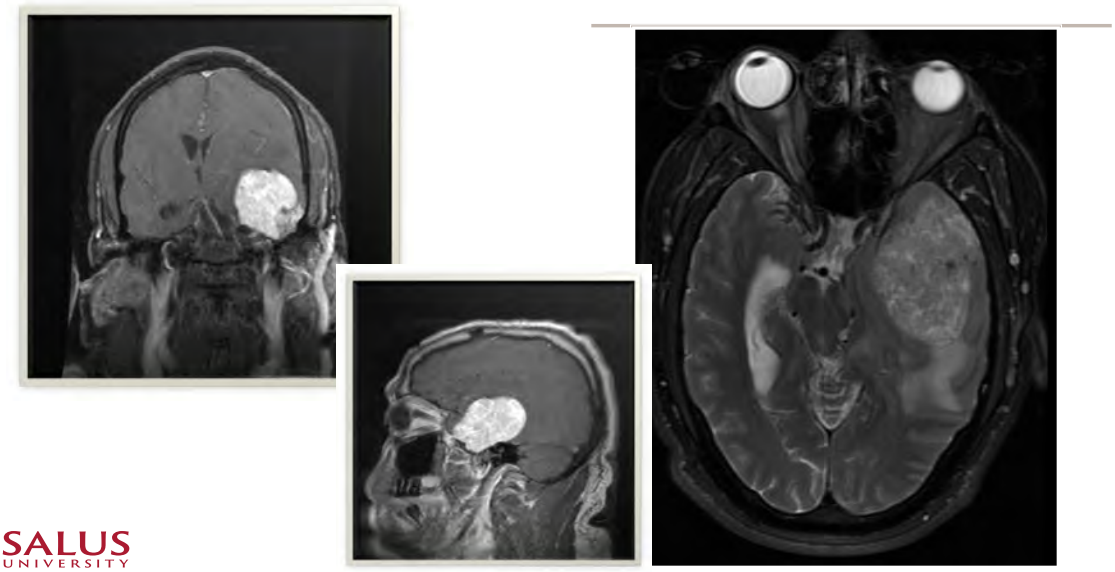
Swelling of the optic nerves



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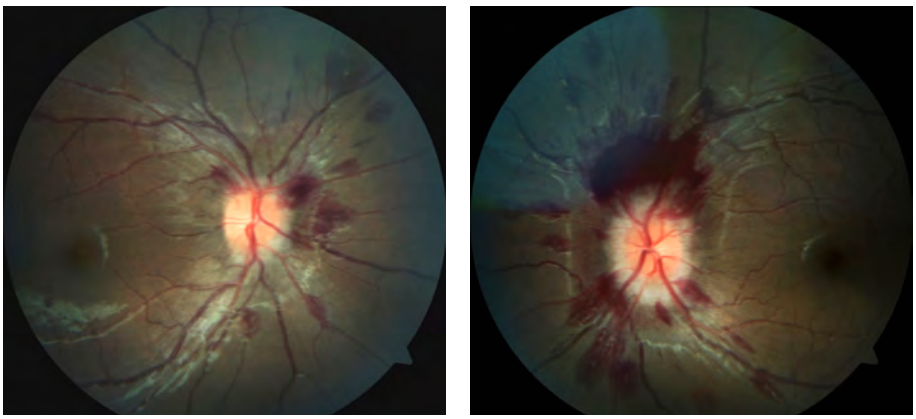
PTC in a 16 year old - (317 lbs. BMI 52)



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Thanks to Dr. Lindsey Perno

88

Causes of Papilledema

Increased intracranial pressure

- **Mass or space-occupying lesion**
- Primary idiopathic intracranial hypertension (IIH)
- Secondary Intracranial Hypertension
  - Subdural venous thrombosis
  - Sagittal sinus thrombosis
  - Chiari malformation
  - Arteriovenous malformation
  - Meningitis/encephalitis
- Medication
- What causes the disc to swell?
  - Axoplasmic stasis and extrusion at the level of lamina cribrosa

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Adult brain tumors

The most common primary central nervous system neoplasm in adults:

Astrocytomas = comprise **80**% of all CNS tumors

1. Pilocytic Astrocytomas – childhood and cerebellar
2. Fibrillary Astrocytomas – (25 years old)
3. Anaplastic Astrocytoma
4. Glioblastoma Multiforme – most aggressive

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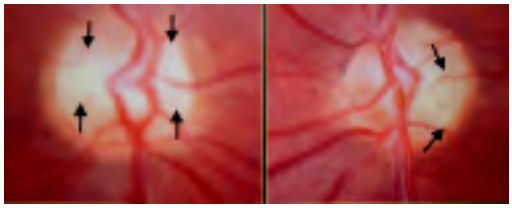
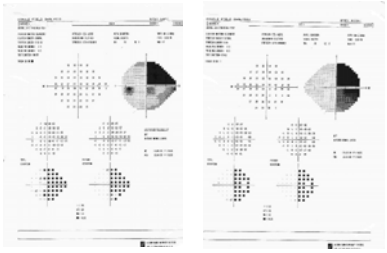
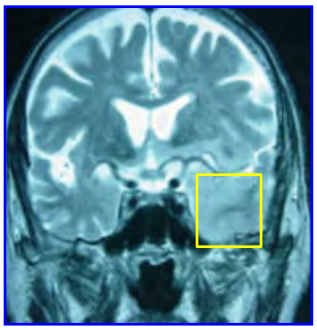
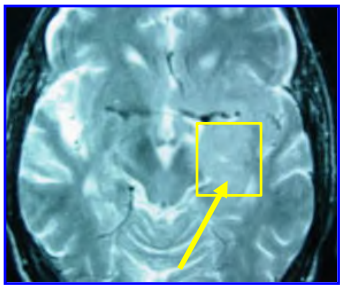
Glioblastoma Multiforme
Note the specific type of pallor in each optic nerve









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Brain tumor suspicion

The greatest concern for most chronic headache patients is that they have a brain tumor

Patients with primary or metastatic brain tumors have a headache at the time of diagnosis (~30%)

Brain tumor headache have pain worse in the morning, nausea and vomiting = seen in **20**% of patients

Most often the headaches are intermittent, dull ache, unilateral and mild initially but usually occur daily

Headache occurs in **70**% of brain tumor patients

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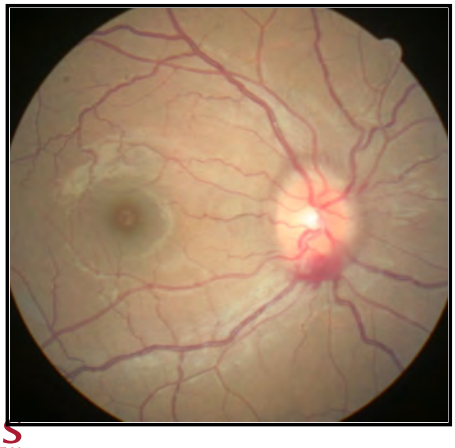
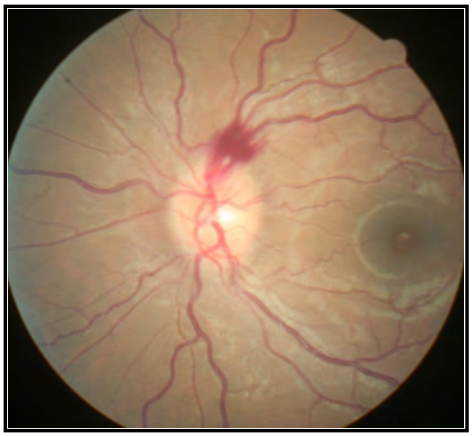
Typical appearance and symptoms of PTC

Headache
Tinnitus





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Things that must be considered with ONH swelling

- Immediately order a **CT** or **MRI** – to rule out mass lesion
- If no lesion is found – **lumbar puncture** will be performed to rule out infection and to measure ICP
- **MRV** to rule out "venous sinus thrombosis"
- Get **visual fields** to make sure no progressive field loss
- Always, Always, Always …. check **blood pressure**

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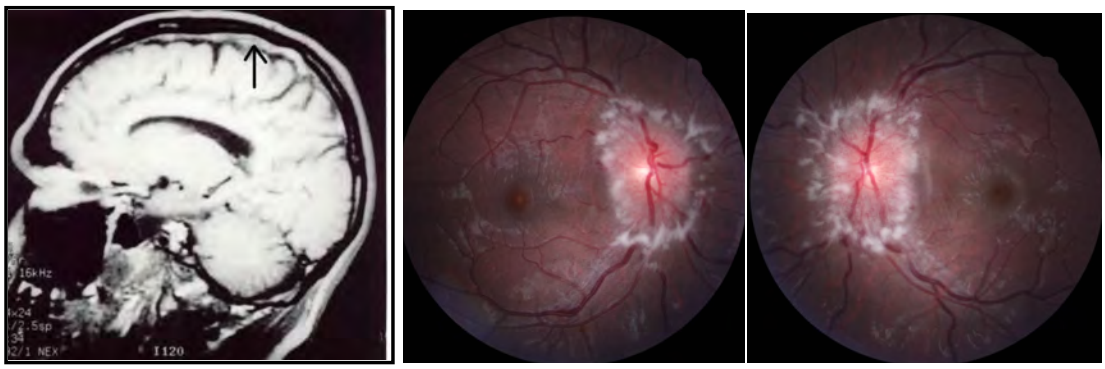
Rule out venous sinus thrombosis



MRV – performed to image dural venous sinuses

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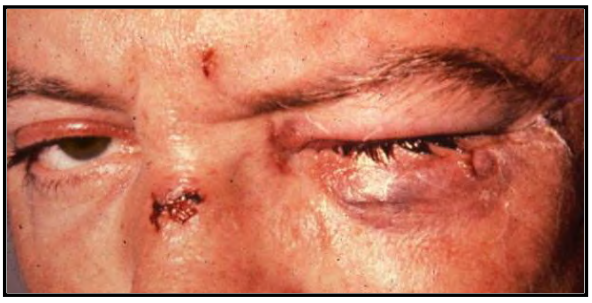
Open Globe Injuries

Types:

- Rupture
- Penetrating
- Perforating
- Intraocular FB
- Mixed



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Pattern of Injury

- **Bimodal distribution** of injury with the young and the elderly most affected - young men in their teens and 20's bear the burden of eye injury
- Men 3 – 5 times as frequently than women = vision threatening eye injuries
- Ocular trauma is a significant cause of visual loss, especially in lower socioeconomic strata and countries
- Ocular trauma is a recurrent disease

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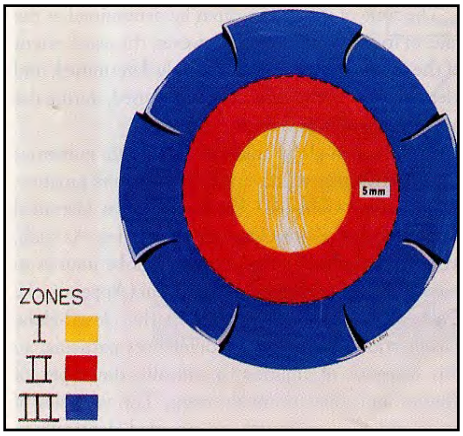
Open-globe injury - zone of injury



- **Zone I: opening of globe is limited to cornea or corneoscleral limbus**
- **Zone II: those that involve the anterior 5mm of the sclera (not more posterior than the pars plana)**
- **Zone III: those that extend the full thickness into the sclera more than 5mm posterior to the limbus**

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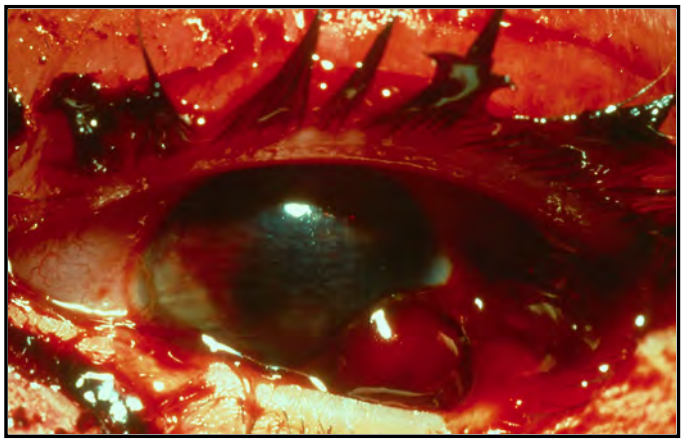
Ocular Trauma



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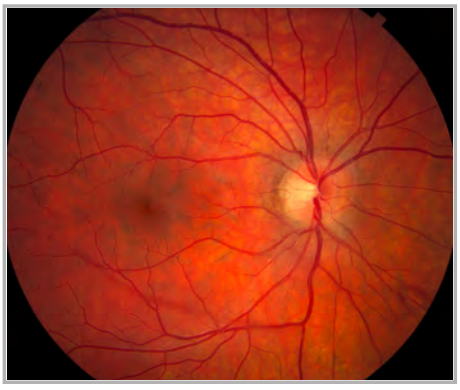
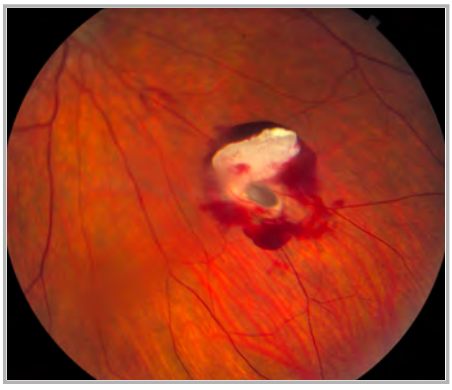
99

Open Globe Injuries

Blood in the vitreous



Open globe injury with penetration



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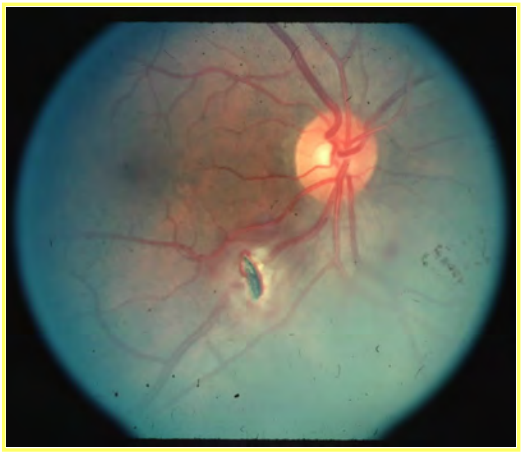
Open Globe Injury with metal



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Endophthalmitis following metallic foreign body

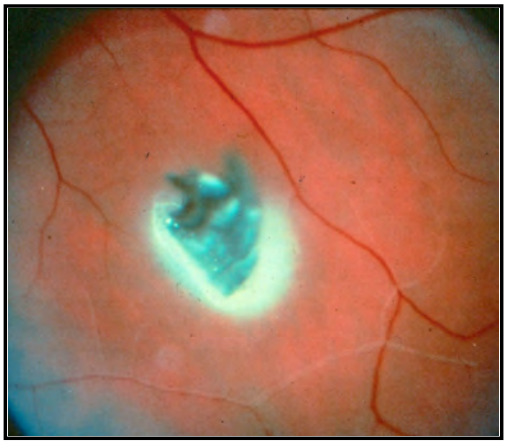
The incidence of **endophthalmitis** following penetrating injuries is between 5% to 14%.

The USEIR incidence is 2.6% and more common in males.

The incidence is more common in rural settings (30%) or involves an IOFB (15%).

Infections with more than one organism are common and occur approximately 50% of the time

Bacillus and staph are **most prevalent**.



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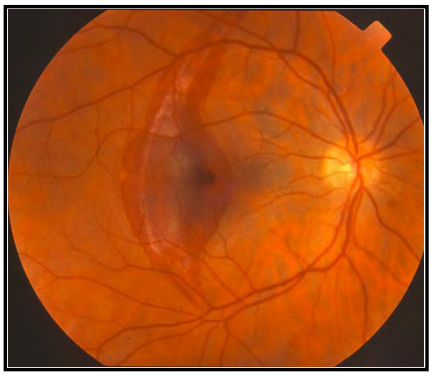
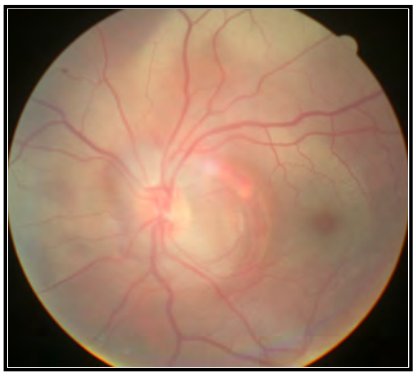
Closed globe injuries

Choroidal Rupture



Choroidal Rupture



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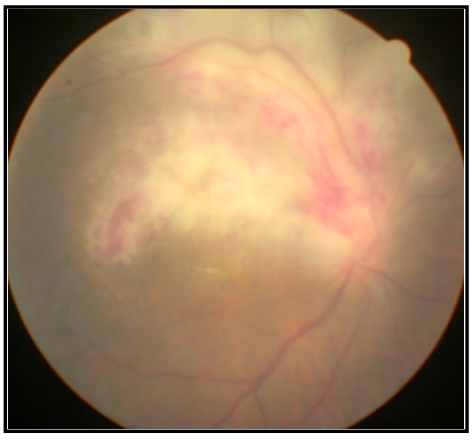
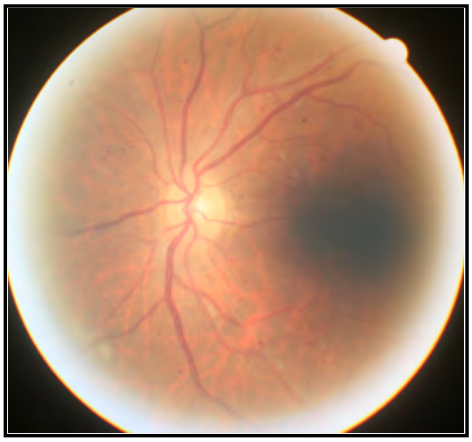
103

Cytomegalovirus retinitis



HIV encephalopathy



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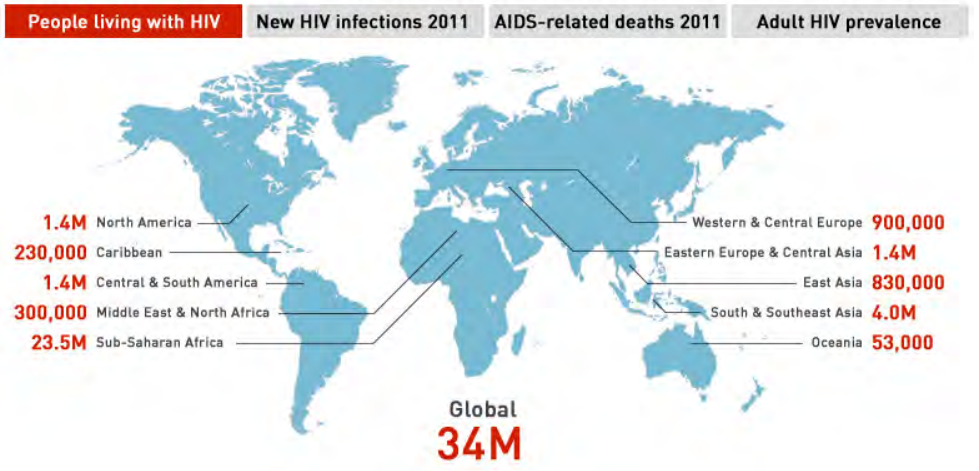
HIV statistics



http://www.cbc.ca

Acquired Immune deficiency syndrome

Course of the Disease

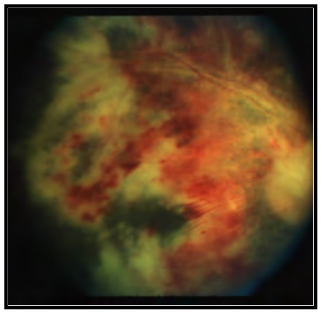
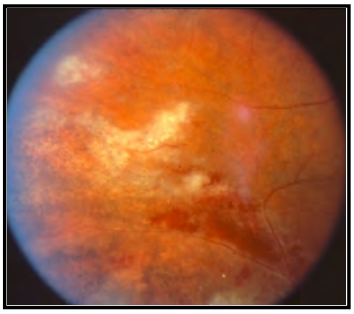
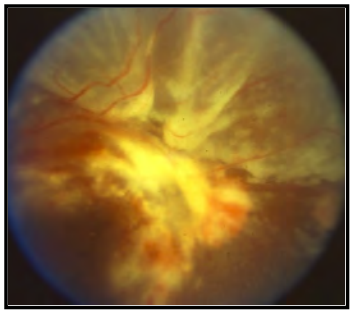
- **Initial Stage** – Influenza like illness ~ 4-12 weeks after infected
- **Chronic Stage** – Latent period ~ 10 years - minor immune dysfunct
- **Final (Crisis) Stage** – Weight loss, fever, skin rashes, opportunistic infections and neoplasms. The virus is replicating within the lymph nodes

Cytomegalovirus retinitis

Cytomegalovirus – DNA virus belonging to the herpes virus family

Fulminant form – necrotic and hemorrhagic fundus

Indolent form – granular retinitis with less edema and hemorrhage

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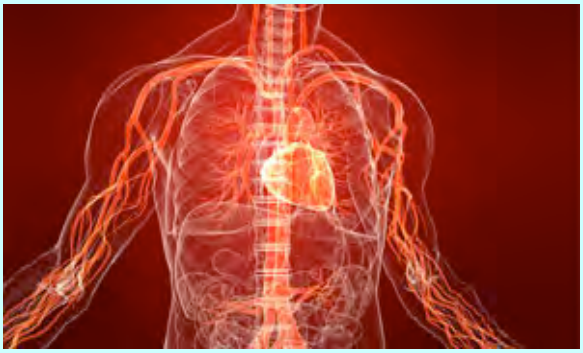
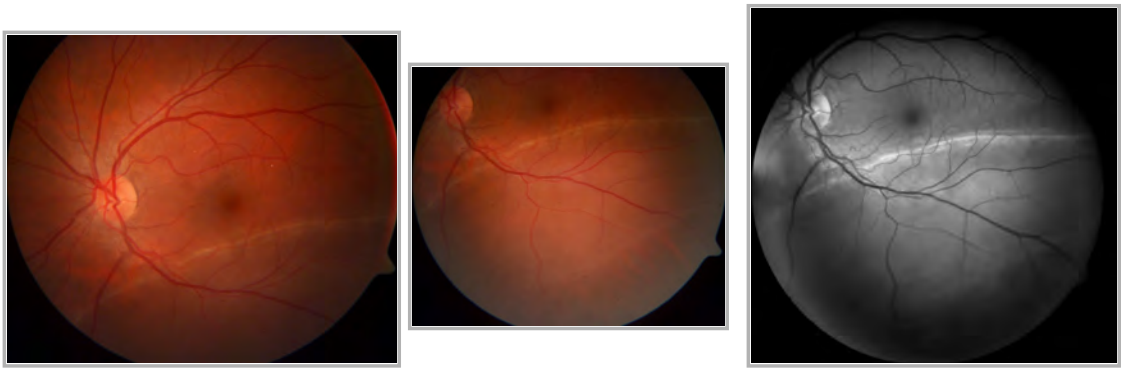
QUESTIONS

AND ANSWERS



Retinal detachment threatening the fovea



The retinal detachment is in the macula however not in the fovea

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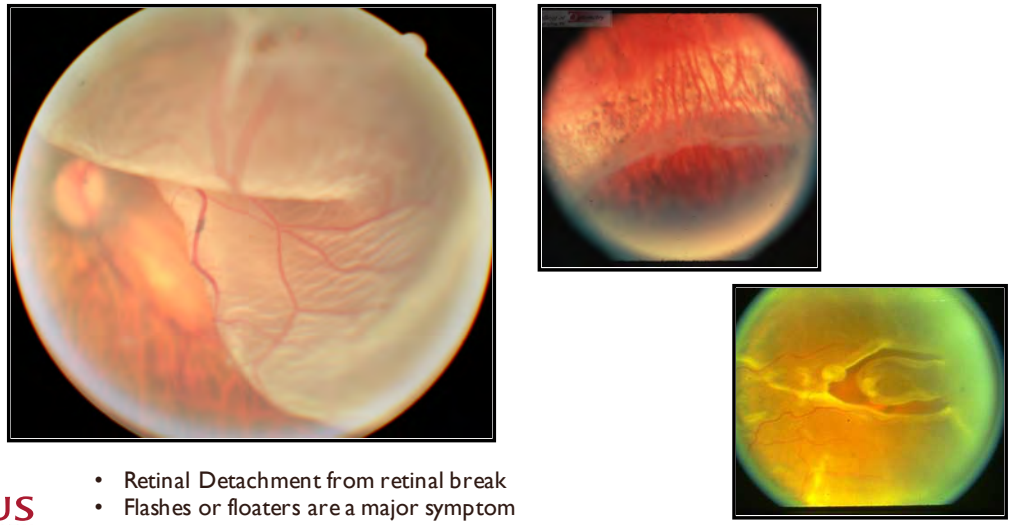
Rhegmatogenous retinal detachment



- Retinal Detachment from retinal break
- Flashes or floaters are a major symptom

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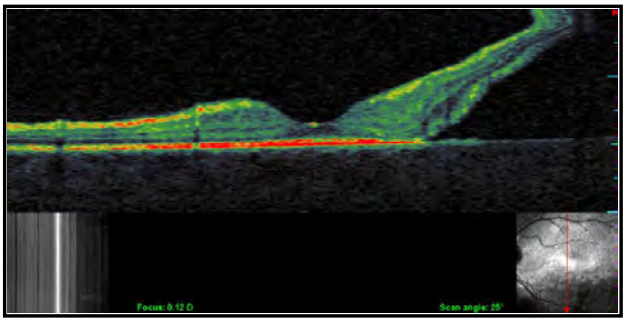
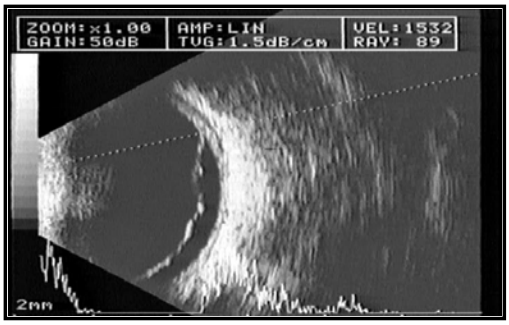
Other Testing for Retinal Detachment





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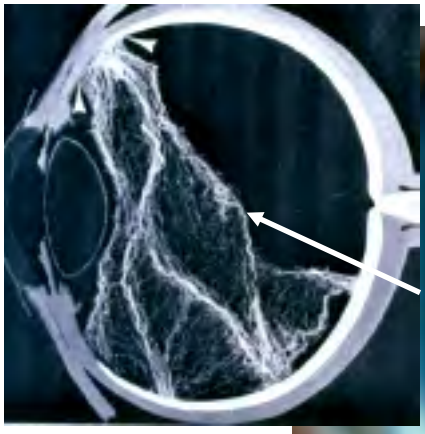
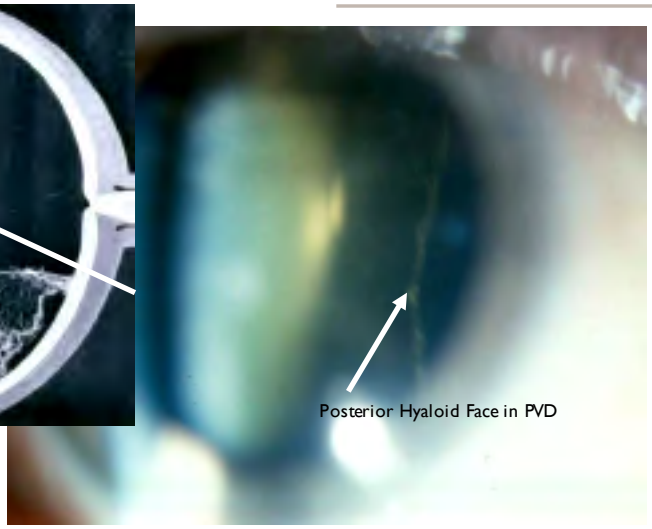
111

Clinical Appearance of PVD





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Treatment of retinal break and detachment

- Referral to a retinal specialist for repair immediately
- Laser retinopexy
- Scleral buckle
- Cryotherapy
- C3F8 or SF6 "gas bubble" as a tamponade

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Treatment Options for Retinal Detachment

Vitrectomy with "Gas Bubble"
Air (78% nitrogen and 21% oxygen) – lasts 2-3 days
SF6 (Sulfur hexafluride) – lasts 10-14 days
C3 F8 (Perfluoropropane) – lasts 4-6 weeks

Vitrectomy with Silicone Oil - due to "PVR" proliferative vitreo-retinopathy
Used for recurrent or chronic retinal detachment

Pneumatic Retinopexy – gas without vitrectomy and uses laser or cryo to seal the break

Vitrectomy c Scleral Buckle – band of silicone sewn onto the sclera

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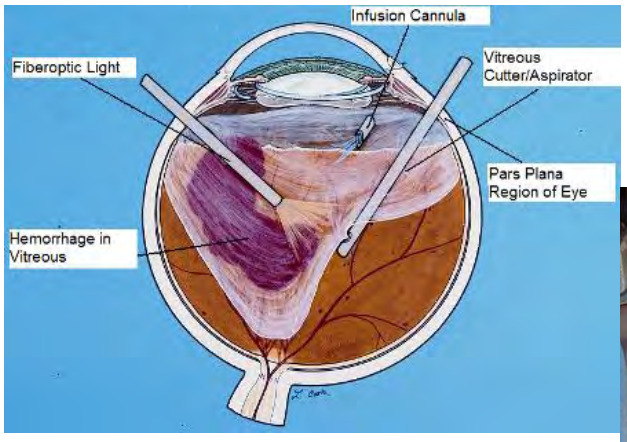
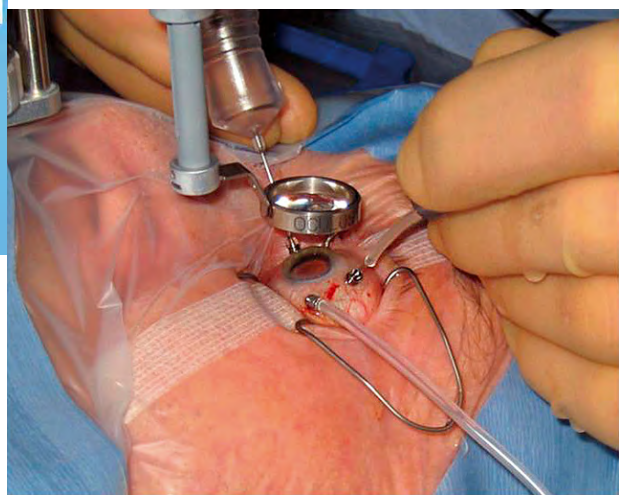


Pars Plana Vitrectomy



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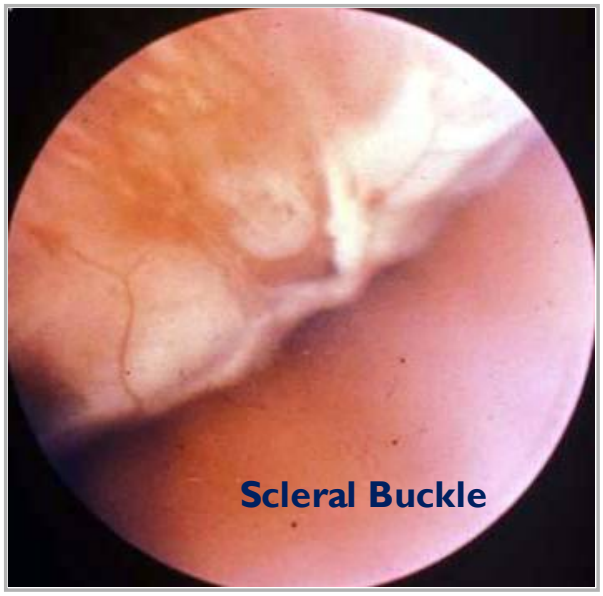

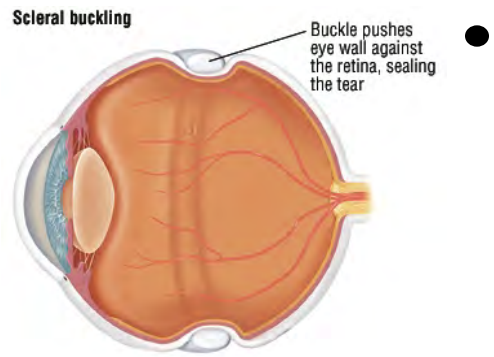

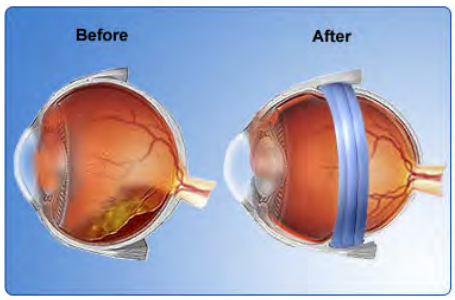

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Conclusions

- Optometrists and their office staff should be aware of what constitutes an ocular/medical emergency involving the posterior pole.
- Stroke protocols should be followed in new CRAO/BRAO.
- Any neuro-ophthalmic sign in an older patient may be GCA!

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Thank you !

Carlo and Joe

Questions and Answers

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