Herpes Simplex Keratitis Masquerading as a Refractory Corneal Abrasion

Description
- The HSV is a DNA virus that usually presents as a coarse, punctate, linear, stellate, branching lesion with club-shaped terminal end bulbs but may begin as a diffuse SPK that subsequently coalesces into branching patterns.
- Edges of lesions are heaped up and stain with rose bengal; center of ulcer stains with fluorescein (NaFl); corneal abrasions do not stain with rose bengal.

Differential Diagnosis
- Herpes zoster (skin lesions along dermatome, corneal lesions do not stain well with NaFl, and corneal lesions do not have terminal end bulbs)
- Recurrent corneal erosion (healing lesions may resemble dendrites but do not have terminal end bulbs)
- CL-related SPK (SPK may coalesce but does not have terminal end bulbs)

Symptoms
- Pain, redness, photophobia, lacrimation, decreased vision
- With repeated episodes, corneal sensitivity decreases, and pain is not as prominent.

Epithelial Disease
- Branching pattern with club-shaped terminal end bulbs
- Enlargement of the dendritic ulcer may lead to an amoeboid-shaped geographic ulcer with a dendritic edge

Neurotrophic Ulcer (Metaherpetic Ulcer, Trophic Ulcer, Indolent Ulcer)
- A sterile, oval epithelial defect with raised, gray borders located in the lower ½ of the cornea; has no dendritic edge
- Often has underlying stromal edema
- Defect not caused by active disease
- May occur months after initial HSV keratitis
- Etiology is thought to be a defect in basement membrane/adhesion complex due to decreased sensory input (sensory nerves are compromised from the initial infection).
Stromal Disease (Disciform Edema and Necrotizing Interstitial Keratitis)

Disciform Edema
- Disciform edema is a disc-shaped stromal edema with an intact epithelium.
- Edema localized to the central 2/3 of the cornea
- No stromal necrosis or neovascularization

- A stromal immune ring of infiltrates (Wessley ring) may be present.
- The limbal tissue may be thickened and inflamed (limbitis).
- May be accompanied by a mild granulomatous anterior uveitis and elevated IOPs
- Edema thought to be due to a defect in the endothelial pump as a result of a viral antigen hypersensitivity reaction; elevated IOPs are due to a trabeculitis.

Necrotizing Interstitial Keratitis
- Diffuse white/gray stromal infiltrates
- May be accompanied deep stromal neovascularization and stromal thinning
- Severe anterior uveitis and hypopyon may be present.
- Ultimately, collagen breaks down and necrosis and scarring occurs.
- Etiology is thought to be active invasion of the virus into the stroma resulting in an antigen-antibody-complement response.

Treatment of HSV Epithelial Disease
- Treat epithelial disease with trifluridine 1% (Viroptic) 9x/day; cycloplegia, i.e. 5% Homatropine or .25% Scopolamine BID
- If there is no improvement in 3 days, debride necrotic epithelium with wet cotton swab or blunt instrument and continue antivirals.
- If there is no stain with rose bengal after 10 days, discontinue antivirals.
- If epithelial defects persist after 14 days there may be antiviral toxicity or neurotrophic ulcer; discontinue antivirals.
- Oral acyclovir 400mg 5X/day may be used to reduce the frequency of topical antivirals; oral acyclovir 400 mg 2Xday for one year reduces recurrence.

Treatment of Neurotrophic Ulcers
- Do not use antivirals.
- Use non-preserved artificial tears Q2H and ointment HS.
- If there is no improvement use erythromycin ointment and pressure patch for 24 hours. Then use erythromycin ung QID and artificial tears Q2H for 5 days. If the is no improvement use a bandage CL and topical antibiosis, amnionic membrane cover, or refer for tarsorrhaphy.
Treatment for Stromal Disease
- If disciform edema is present but VA is not affected, use cycloplegia and/or observe.
- If anterior uveitis or limbitis is present or VA is affected, use cycloplegia and steroid e.g., loteprednol .2% (Alrex) BID, and cover with antiviral drops 5X/day to prevent recurrence of epithelial disease; oral acyclovir shortens course of the anterior uveitis
- If limbitis is present, use topical steroids with an antiviral cover.
- With nerotizing stromal keratitis - cycloplegia, mild steroid and antiviral cover.

Herpetic Eye Disease Study
- Adding oral acyclovir (400 mg 5x/day) to Viroptic did not prevent stromal keratitis and did not shorten course of epithelial disease.
- Utilizing topical steroid with Viroptic did reduce the progression and shortened the course of stromal keratitis.
- Adding oral acyclovir (400 mg 5x/day) to topical steroid did not reduce the progression or shorten the course of stromal keratitis.
- Adding oral acyclovir 400 mg 5x/day did reduce the progression and shorten the course of HSV iridocyclitis.
- Utilizing oral acyclovir 400 mg BID for one year reduced the rate of progression for all types of HSV keratitis

Carotid Vascular Disease Masquerading as Non-Proliferative Diabetic Retinopathy

Description of Carotid Insufficiency/Ocular Ischemic Syndrome
- A usually unilateral, generalized ischemia to the anterior and posterior ocular segments occurring in patients over the age of 60
- Anterior segment: orbital pain, dilated, tortuous, episcleral arteries, corneal edema, mild flare and cells, poorly responsive pupil, neovascularization of the iris and/or angle often leading to neovascular glaucoma
- Posterior segment: reduced IOP, dilated, non-tortuous retinal veins, neovascularization of the optic disk and/or other parts of the retina, mid-peripheral blot and dot hemorrhages, arteriole plaques, artery occlusions (CRA or BRA)

Etiology of Ocular Ischemic Syndrome
- Ipsilateral atherosclerotic carotid stenosis that is greater than 80%
- Associated with diabetes mellitus, hypertension, ischemic heart disease, and cerebrovascular disease
Workup of Carotid Insufficiency/Ocular Ischemic Syndrome

- History
  1. Consider the risk factors of hypertension, smoking, hypercholesterolemia, diabetes mellitus, obesity and sedentary lifestyle, cardiac dysfunction, vasculitis, and hyperviscosity syndromes.
  2. TIAs?
  3. Light-induced amaurosis?
- Slit lamp, ophthalmoscopy to evaluate anterior and posterior segments
- Auscultation of the carotid vasculature
- Duplex Doppler scanning; MRA
- CBC
- Palpate the facial pulses.
- Determine the blood pressure in each arm.
- Perform visual fields.
- Obtain CBC and blood chemistry data; rule out coagulopathies.
- Obtain ESR and C-reactive protein data.
- Obtain Doppler and/or MRA studies.

Treatment
- Reduce risk factors; antiplatelet therapy; Coumadin
- PRP and/or antiVEGF therapy
- Carotid endarterectomy if the stenosis is greater than 70% and the patient is symptomatic

 Conjunctival Intraepithelial Neoplasm (CIN) Masquerading as a Pterygium

Description of CIN
- An elevated, fleshy gelatinous, lobulated white-gray vascular lesion at the limbus; feeder vessels are often prominent
- Composed of dysplastic malignant cells confined to the conjunctival and corneal epithelium
- If dysplastic cells penetrate to the stroma, squamous cell carcinoma is diagnosed.

Epidemiology
- Usually occurs in males over age 60 who have had long-term exposure to the sun and who smoke
- Can occur in young patients with HIV

Differential Diagnosis
- Pterygium – A transparent fold of fibrovascular, conjunctival tissue advancing onto the limbus relacing the epithelium and Bowman’s membrane
- Papilloma – Pedunculated or flat based; pink color, dilated tortuous vessels are seen below surface
Treatments
- Excisional biopsy followed by cryotherapy
- Topical Mitomycin C
- Topical Mitomycin C and Cyclosporine
- Topical cidofovir
- 5-Flourouracil
- Topical Interferon alpha 2B

Choroidal Nevus Masquerading as a Choroidal Melanoma

Description
- A benign tumor of the choroid composed of an increased number of melanocytes
- Usually flat or minimally elevated (less than 2 mm) with a slate, gray-brown or amelanotic (pale yellow) color and with ill-defined margins
- Usually 1-5 mm in diameter
- More common in whites than blacks
- May have overlying drusen
- Usually, does not cause a break in Bruch’s membrane, and does not cause a RPE detachment, serous RD, or choroidal neovascularization.
- A nevus is most apt to convert to a malignant melanoma if it is greater than 2 mm in thickness, is close to the disc, has overlying orange pigment, has significant subretinal fluid, has associated visual symptoms

Differential Diagnosis
1. Congenital hypertrophy of the retinal pigment epithelium (CHRPE)
   - Flat, well-demarcated borders, solid black or may have depigmented spots within the lesion (lacunae)
   - Often ringed with a shallow border of hypopigmentation
2. Subretinal hemorrhage
3. Choroidal metastasis
4. Choroidal melanoma

Choroidal Melanoma
- Most common primary intraocular malignancy occurring mostly in fair-skinned elderly whites who have been exposed to UV light
- Arises from the spindle or epithelioid dendritic melanocytes of the choroid
- Tends to metastasize to the liver
- Presents as a mushroom-shaped, elevated lesion which is greater than 2 mm in height and greater than 10 mm in diameter
- May be brown, gray, or amelanotic with ill-defined borders and large clumps of overlying orange pigment (lipofuscin)
- Often has large areas of subretinal fluid surrounding the base with a concomitant serous retinal detachment; flashes and floaters may occur
- Lesion may be very close or actually abut the optic nerve head
- May lead to a vitreous hemorrhage or liberate pigment into vitreous or AC

**Workup**
- Ophthalmoscopic evaluation for clinical features and diameter
- Ultrasonography for thickness and morphology (low internal reflectivity)
- Fluorescein angiography - early hyperfluorescence with progressive staining in the late phases

**Prognosis and Management - Collaborative Ocular Melanoma Study**
- Epithelioid melanomas are most apt to metastasize and carry the greatest risk.
- Observation is appropriate for small tumors with little evidence of growth.
- Transpupillary thermotherapy, plaque brachytherapy, or local proton-beam radiation is reserved for smaller lesions.
- Enucleation best for large melanomas, i.e. greater than 10 mm in height and 12 mm in diameter.

**Optociliary Shunt Vessels Masquerading as an Old Central Retinal Vein Occlusion**

**Description**
- Optociliary shunt vessels consist of enlarged pre-existing capillaries on the optic nerve head that shunt blood from the central venous circulation to the peripapillary choroidal circulation.
- Occurs when there is an obstruction of the normal venous drainage behind the lamina
- The shunted blood bypasses the retinal venous circulation and passes into the choroidal venous system; subsequently, the blood passes through the vortex veins and into the superior and inferior ophthalmic veins.

**Etiology**

**Central Retinal Vein Occlusion**
- Usually occurs as a result of compression of the central retinal vein by the central retinal artery in the common adventitial sheath; may also occur as a result of impaired inflow due to carotid insufficiency, a central retinal vein thrombus from autoimmune disease, excessive coagulation of the blood, or compression of the lamina from glaucoma
- About 20% of CRVOs are ischemic with marked optic nerve head edema, extensive four quadrant flame-shaped and dark, full-thickness hemorrhages, multiple cotton wool spots, marked venous dilation and tortuosity, extensive retinal edema masking the choroidal detail, markedly reduced vision, and an RAPD.
- About 80% of CRVOs are non-ischemic with mild optic nerve head edema, scattered blot, dot, and flame-shaped hemorrhages, few cotton wool spots, mild venous dilation and tortuosity, mild retinal edema which does not mask the
choroidal detail, mildly reduced vision which often recovers to 20/40, and no RAPD.
- Non-ischemic CRVOs do not develop retinal or iris neovascularization.

**Optic Nerve Sheath Meningioma**
- Neoplasm arises from arachnoid and affects women over 40
- Involves optic nerve within the orbit causing compression and occlusion of the veins of the optic nerve
- Causes slow, progressive vision loss
- Optociliary shunt vessels develop to allow venous blood to exit the optic nerve.

**Anterior Chiasmal Syndrome Masquerading as Normotensive Glaucoma**

**Anatomic Considerations – Optic Nerve**
- The optic nerve consists of axons from retinal ganglion cells, i.e. the nerve fiber layer (NFL).
- NFL is divided into 3 bundles: papillomacular bundle, arcuate bundles, nasal radial bundle
  1. Papillomacular bundle
     - Fibers run a straight course from the macula to the temporal half of the optic disc.
     - Lesions produce **temporal pallor** and central or cecocentral scotomas.
     - Lesions cause VA reduction, APD, color vision deficit, light sensitivity deficit
     - Glaucoma preferentially **spares** the papillomacular bundle until very late in the disease process.
     - **Temporal pallor is not a normal manifestation of glaucoma.**
  2. Arcuate bundles
     - Fibers originate temporal to the macula, arch above and below the papillomacular bundle, and enter disc at the superior and inferior poles.
     - Fibers are segregated into upper and lower compartments and do not cross the horizontal raphe.
     - **Glaucoma preferentially affects the arcuate bundles.**
     - Lesions affecting arcuate fibers near the fovea, produce paracentral scotomas.
     - Lesions affecting arcuate fibers near the optic disc, produce partial scotomas which do not cross the nasal horizontal meridian.
     - Lesions affecting fibers remote from the disc, produce nasal steps.
  3. Nasal radial fibers
     - Fibers originate nasal to the optic disc.
     - Fibers fan into the optic disc in a radial fashion.
     - Fibers do not segregate into upper and lower bundles and do cross the horizontal raphe.
- Lesions produce wedge-shaped defects which do not respect the horizontal meridian and do not produce a step-like depression.

Anatomic Considerations - Chiasm
- Within the chiasm the nasal retinal fibers cross; the temporal retinal fibers do not cross.
- The inferior nasal fibers from each optic nerve sweep into the posterior aspect of the contralateral optic nerve where the contralateral optic nerve joins the anterior aspect of the chiasm.
- Chiasmal lesions are often compressive and emanate from the underlying pituitary gland.
- The configuration of visual field defects from chiasmal lesions, depends upon the position of the chiasm relative to the pituitary gland.
- If the body of the chiasm is directly over the pituitary gland, compression of the chiasm results in a bitemporal hemianopsia; the visual field defects do not cross the vertical midline.
- If the body of the chiasm is in front of the pituitary gland, i.e. prefixed, pituitary lesions impact the crossing nasal macular fibers producing a bitemporal hemianopsia within the central 10 degrees, or impact the optic tract producing a noncongruent hemianopsia.
- If the body of the chiasm is behind the pituitary gland, i.e. postfixed, pituitary lesions impact the crossing inferior nasal fibers producing bitemporal superior quadrantanopsias or impact the inferior nasal fibers at the junction of the optic nerve and the chiasm producing an ipsilateral optic nerve defect and a contralateral superior temporal defect, i.e. anterior chiasmal syndrome

Retinoschisis Masquerading as a Retinal Detachment

Description
- A splitting of the sensory retina due to an accumulation of fluid within the sensory retina

Congenital Retinoschisis
- A rare genetic disorder affecting males
- Cystoid spaces develop in the fovea; appears as a bicycle wheel with radiating spokes
- A retinal splitting occurs separating the nerve fiber layer (NFL) from the rest of the retina.
- Central VA is reduced.
- Vitreous hemorrhage often occurs as the inner blood vessels tear.

Degenerative Retinoschisis
- A retinal splitting occurring in 3-10% of population.
- Usually bilateral; usually occurring in the inferotemporal fundus
- Split occurs in the outer plexiform layer between the outer nuclear layer and the inner nuclear layer.
- Neural elements are lysed and an absolute scotoma results.
- Bulging inner wall is smooth and transparent or has a bronze-beaten metal appearance.
- Retina is immobile and does not undulate.
- Retinal vessels are often sheathed.
- White dots (snow flakes) are on the underside of inner wall.
- No pigment cells or hemorrhage in the vitreous.
- A rhegmatogenous retinal detachment if the outer wall of the schisis cavity develops a hole.

**Differential Diagnosis**

**Rhegmatogenous Retinal Detachment**
- A usually unilateral separation of the sensory retina from the RPE as a result of fluid percolating through a break in the sensory retina.
- Retina looks corrugated and undulates.
- Flashes, floaters, or a curtain may be seen.
- Pigment cells and hemorrhage may be in the vitreous.

**Exudative Retinal Detachment**
- Bruch’s membrane/RPE barrier is compromised allowing fluid from the choroid to separate the RPE from the sensory retina thus creating an RD.
- Subretinal fluid shifts according to the position of the head.
- Reduced VA occurs only when the macula is involved.
- Etiology includes underlying inflammation, e.g. scleritis, orbital inflammation, toxoplasmosis, Lyme disease; autoimmune disease; vascular disease, e.g. Coat’s disease, acute systemic hypertension, eclampsia; neoplasm, e.g. choroidal melanoma, choroidal metastasis.

**Talc Retinopathy Masquerading as Drusen**

**Description**
- Intraretinal, yellow, refractile particles in patients found in patients who repeatedly inject drugs intended for oral use (Ritalin, Methadone) intravenously.
- The oral drugs contain inert fillers such as talc.
- The number of talc particles in the retina is dependent upon the duration of drug abuse and the number of oral medications that have been injected.

**Pathophysiology**
- Oral drugs with talc fillers are injected IV.
- Talc passes through the right auricle and right ventricle and enters the lung.
- Some of the talc particles become entrapped in the pulmonary capillaries.
- Granulomas develop causing further occlusion of larger arteries; diminished lung capacity and reduced pulmonary function result.
- Pulmonary hypertension results causing enlargement of the right ventricle (cor pulmonale); chest pain, syncope, and sudden death may result.
- As pulmonary hypertension increases, collateral blood vessels develop in the lung.
- The collaterals are large enough to allow the talc particles to gain access to the systemic circulation as blood passes through the left auricle and left ventricle.
- The talc embolizes to the eye, skin, liver, kidney, and bone marrow.
- The talc can be scattered throughout the fundus but concentrates in the small arterioles, precapillaries, and capillaries in the macular area. (There is a denser capillary net and greater blood flow in that area.
- Capillary occlusion may result in venous engorgement, blot hemorrhages, cotton wool spots, and peripheral neovascularization.

**Differential Diagnosis**

- Cholesterol deposits (Hollenhorst plaques)
- Tamoxifen
- Canthaxanthine (oral tanning agent)
- Calcific drusen

**Drusen**

- RPE phagocytizes the tips of the outer segments of the rods and cones.
- The engulfed particles are degraded and recycled or are voided into the choriocapillaris.
- With increasing age, Bruch’s membrane thickens and prevents the deposition of the breakdown products into the choriocapillaris; the particles become stored in Bruch’s membrane as drusen.

**Isolated Discrete Drusen**

- Small, isolated, discrete, yellowish bodies with little evidence of RPE atrophy; VA relatively good

**Confluent Large Soft Drusen**

- Indicate significant thickening of Bruch’s membrane and RPE atrophy
- Have the highest risk of developing into wet AMD

**Hard Calcific Drusen**

- Occur in long standing, indolent AMD; VA usually good

**Workup of Talc Retinopathy**

- Fluorescein angiography if neovascularization is suspect
- Lung x-ray and lung function tests
Recurrent Phlyctenulosis Masquerading as a Nodular Episcleritis

**Description**
- A pinkish-white nodule at the limbal conjunctiva; may not involve the cornea
- Nodule is centered in an area of dilated blood vessels with a local leash of blood vessels running away from the lesion
- Usually occurs in children or young adults

**Conjunctival phlyctenulosis**
- Mild symptoms of discomfort
- After several days, lesion becomes necrotic and sloughs off leaving no scar

**Corneal phlyctenulosis**
- Lesion has a triangular shape and sits astride limbus
- Significant symptoms of pain, photophobia, and lacrimation
- Lesion may be stationary or may migrate onto cornea
- If corneal migration occurs, neovascularization and scarring may occur.

**Etiology**
- Results from a Type 4 delayed hypersensitivity reaction to a systemic antigen
- Etiology is usually staphylococcus; tuberculosis is the second most common etiology.
- Other etiologies include intestinal parasites, chlamydia, HSV, gonococcus, and as a forme fruste of cutaneous rosacea.

**Differential diagnosis**
- Inflamed pinguecula
- Nodular episcleritis
- Vernal limbal keratoconjunctivitis
- HSV - Corneal neovascularization running into a stromal infiltrate
- Ocular rosacea

**Treatment**
- If staphylococcal blepharitis is present – lid hygiene and antibiotic ointment along with topical steroids, e.g. FML, Lotemax
- If rosacea is present – systemic tetracycline or erythromycin along with topical steroid
- If TB, intestinal parasites, chlamydia, or gonococcus are present coordinate with the PCP, and continue the topical steroid.
Idiopathic Orbital Inflammation Masquerading as Orbital Cellulitis

Description of Idiopathic Orbital Inflammation
- An acute, rapidly-developing idiopathic inflammation affecting any tissue within the orbit
- Unilateral in the adult; may be bilateral in children
- No constitutional symptoms in the adult; children may have mild fever and leukocytosis

Pathophysiology of Idiopathic Orbital Inflammation
- Soft orbital tissues become infused with lymphocytes, neutrophils, and monocytes
- Any or all of the following structures may be infiltrated: lacrimal gland, extraocular muscles (belly and tendon), Tenon’s capsule, posterior sclera, orbital fat, optic nerve sheath (producing optic neuritis).

Signs and Symptoms of Idiopathic Orbital Inflammation
- Proptosis, restricted ocular motility, reduced vision (if optic nerve sheath becomes involved) conjunctival redness and chemosis, baggy eyelid edema and erythema, pain, diplopia
- Less commonly – uveitis, elevated IOP, hyperopic shift

Workup of Idiopathic Orbital Inflammation
- CT, MRI, and B-scan ultrasonography
- Measure temperature to rule out cellulitis
- CBC, ESR, ANA, ANCA,
- Orbital biopsy if patient has a history of cancer

Treatment of Idiopathic Orbital Inflammation
- Systemic steroids, e.g. prednisone 80-100 mg/day and taper
- Radiation if patient not responsive to steroids

Description of Orbital Cellulitis
- A slowly-evolving bacterial inflammation behind the orbital septum; mostly in children
- Patient has a high fever, proptosis, warm, taut eyelid edema, restricted ocular motility with pain on attempted eye movement, reduced periorbital sensation optic neuropathy, paranasal sinuses are inflamed

Pathophysiology of Orbital Cellulitis
- Direct extension of acute internal hordeolum, preseptal cellulitis, paranasal sinus infections, infectious dacryoadenitis or dacryocystitis, dental abscess, orbital trauma, insect bites, facial cellulitis
Workup and Treatment of Orbital Cellulitis
- Rule out preseptal cellulitis, i.e. diffusely swollen and reddened lid, no fever, no motility deficit
- CT scan of orbit and sinuses
- CBC with differentials
- Hospitalization and broad-spectrum IV antibiotics

Bilateral Episcleritis Masquerading as Conjunctivitis

Description
- A benign, usually unilateral, but often bilateral, inflammation of the episclera
- Presents acutely, usually in young females
- Superficial episcleral vascular plexus is engorged, and eye is bright red
- Superficial episcleral plexus will blanch with sympathomimetic amines
- Usually sectoral but may be nodular or diffuse
- VA not affected; no photophobia; no discharge
- No conjunctival morphological changes
- Mild pain may occur.

Differential Diagnosis
- Conjunctivitis – No pain, has discharge, papillae or follicles
- Scleritis – Older patients; severe pain; VA reduced; A/C reaction; deep episcleral vascular plexus is engorged, has a violet color, and will not blanch

Etiology
- Usually idiopathic
- Autoimmune disease if episcleritis is recalcitrant or recurrent

Treatment
- If non-painful, use lubrication
- If painful, use topical steroids, e.g. Lotemax, FML
- If recalcitrant, test for autoimmune disease and use systemic NSAIDS

Optic Neuropathy Masquerading as Glaucoma

Anatomic Considerations – Optic Nerve
- The optic nerve consists of axons from retinal ganglion cells, i.e. the nerve fiber layer (NFL).
- NFL is divided into 3 bundles: papillomacular bundle, arcuate bundles, nasal radial bundle
  1. Papillomacular bundle fibers run from the macula to the temporal half of the optic disc. Lesions produce temporal pallor and central or cecocentral scotomas and result in reduced VA, APD, color vision deficit, light brightness deficit
- Glaucoma preferentially **spares** the papillomacular bundle until very late in the disease process.
- **Temporal pallor is not a normal manifestation of glaucoma.**

2. Arcuate bundles
- Fibers originate temporal to the macula, arch above and below the papillomacular bundle, and enter disc at the superior and inferior poles.
- Fibers are segregated into upper and lower compartments and do not cross the horizontal raphe.
- **Glaucoma preferentially affects the arcuate bundles.**
- Lesions affecting arcuate fibers near the fovea, produce paracentral scotomas.
- Lesions affecting arcuate fibers near the optic disc, produce partial scotomas which do not cross the nasal horizontal meridian.
- Lesions affecting fibers remote from the disc, produce nasal steps.

3. Nasal radial fibers
- Fibers originate nasal to the optic disc.
- Fibers fan into the optic disc in a radial fashion.
- Fibers do not segregate into upper and lower bundles and do cross the horizontal raphe.
- Lesions produce wedge-shaped defects which do not respect the horizontal meridian and do not produce a step-like depression.

**Optic Neuritis Masquerading as Papilledema**

**Description**
- Optic neuritis is an acute, rapidly progressing, but short-lasting inflammation of the optic nerve.
- Optic neuritis usually presents unilaterally and affects patients between the ages of 18-45; it may occur in children and present bilaterally.
- There is usually some ocular pain that is exacerbated by eye movement.
- Decreased VA, decreased color vision, decreased contrast sensitivity, decreased light brightness sensitivity, and an afferent pupillary deficit are present to some degree.

**Ophthalmoscopic Presentation**
- Papillitis – the intraocular form of optic neuritis in which there is pink, disc edema with an occasional flame-shaped hemorrhage and a few vitreous cells in front of the disc; it is often bilateral and occurs more frequently in children and young adults
- Retrobulbar optic neuritis – the optic nerve, fundus, and vitreous appear unchanged because the inflammation is behind the lamina; it is usually unilateral.
Etiology
- Idiopathic
- Multiple sclerosis (MS)
- Childhood viral diseases e.g., measles, mumps
- Mononucleosis
- Herpes zoster
- Granulomatous infiltrations e.g., syphilis, sarcoidosis, TB
- Intraocular inflammations e.g., scleritis, uveitis, retinitis
- Inflammations of the orbit and sinuses

MS
- A chronic, inflammatory, demyelinating disease of the CNS most commonly affecting females between ages 20-40
- Non-ocular symptoms include paresthesias, clumsiness, ataxia, incontinence
- Ocular manifestations include: optic neuritis, ocular motor disturbances e.g., diplopia, nystagmus, gaze palsy, impaired pursuits and saccadics, internuclear ophthalmoplegia (lesion is in the medial longitudinal fasciculus - on attempted lateral gaze there is weakness or absence of adduction in the eye ipsilateral to the lesion and nystagmus in the contralateral abducting eye.
- Diagnosis of clinically-definite-MS requires at least 2 neurologic events that have separate anatomic etiology in the CNS and that occur at different times. (separated in both space and time)
- Early symptoms in MS result from axonal demyelination in the CNS.
- Neuronal conduction is subsequently reduced.
- Relapsing-remitting MS occurs in 80% of cases. (Symptoms occur; patient stabilizes; patient improves; and then patient relapses.)
- Primary progressive MS occurs in 20% of cases. Symptoms are progressive and non-remitting, and the patient continues to deteriorate.

Diagnosing MS
- MRI with gadolinium dye reveals multifocal, demyelinating white plaques and scarring around the ventricles (the hallmark of chronic MS); MRI may also show demyelinating plaques within the optic nerve, brainstem, cerebellum, and spinal cord.
- Spinal fluid analysis - elevated IgG; oligoclonal bands
- VER of the optic nerve and spinal cord reveals a delay in conduction.

Optic Neuritis Treatment Trial
- After optic neuritis, 50% of patients reveal MRI evidence of demyelination; many of these patients are initially asymptomatic.
- Within 5-10 years of the initial optic neuritis episode, approximately 50% of patients with 2 or more plaques that are larger than 3mm in size will manifest clinically significant MS.
- 13%-16% of patients who demonstrate no white matter plaques after optic neuritis will be diagnosed with MS in 5 years.
- If patients revealed white matter plaques after optic neuritis, utilizing IV methylprednisolone for 3 days followed by oral prednisone for 11 days accelerated the recovery of vision and delayed the onset of MS.

**Current Management approaches to MS**
- If patients with optic neuritis have white plaques on MRI, treatment with IV methylprednisolone for 3 days followed by oral prednisone for 11 days delays the onset of new clinical manifestations of MS.
- Systemic interferon Beta-1a (Avonex), interferon Beta-1b (Betaseron), or glatiramer acetate (Copaxone), reduce exacerbations and slows progression.

**Papilledema**
- **Description**
- Bilateral optic disc swelling caused by increased intracranial pressure

**Symptoms**
- Headaches, worse upon awakening and worse with valsalva maneuver and change in head position
- Transient obscurations in vision lasting 1-5 seconds
- Nausea and vomiting
- Unilateral or bilateral horizontal diplopia
- Tinnitus
- Normal visual acuity, pupils, and color vision

**Signs**
- Bilateral disc edema with indistinct disc margins
- Opacification of the nerve fiber layer producing obscuration of the vessels as they cross the disc margin
- Hyperemia of the disc
- Loss of venous pulse (many normals have no venous pulse)
- Flame-shaped disc or peripapillary hemorrhages
- Enlarged blind spot