Visual pathway and Optic nerve

Dr. Suresh Kumar
Associate Prof.
Department of Ophthalmology

Anatomy and Physiology of Visual Pathway

Optic nerve

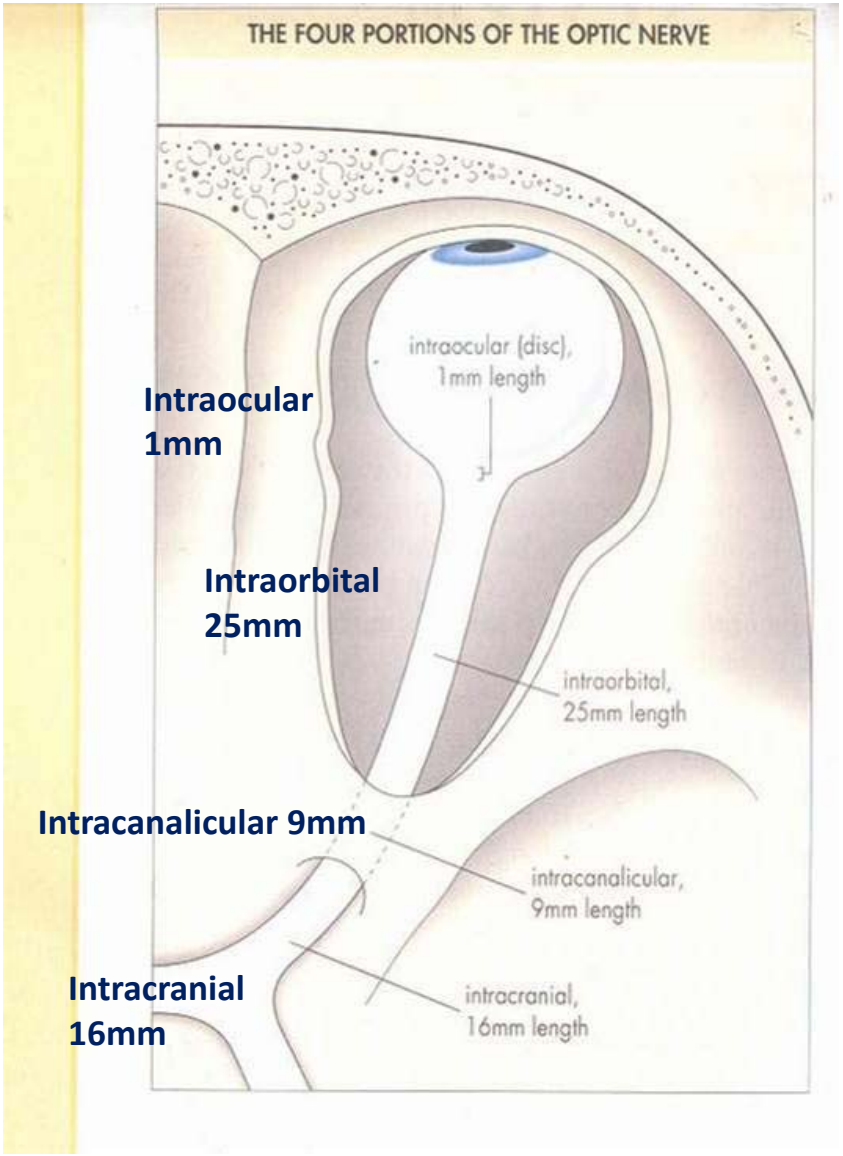
- Second cranial nerve
- Each starts from optic disc and extends upto optic chiasma
- Backward continuation of nerve fibre layer of retina which consist of axons originating from ganglion cells
- Contains afferent fibres of pupillary light reflex

- Unlike peripheral nerves – not covered by neurilemma
- Does not regenerate when cut
- Myelinated by oligodendrocytes
- Not by Schwann cells
- Fibres of optic nerve (approx. 1 million) – diameter 2-10 micron as compared to 20 micron of sensory nerves

Parts of optic nerve

- 47-50 mm in length
- Divided into 4 parts
  - Intraocular – 1 mm
  - Intraorbital – 30 mm (slightly sinuous to allow for eye movements, near optic foramina surrounded by annulus of Zinn, some fibres of superior rectus adherent to its sheath)
  - Intracanalicular – 6-9 mm (ophthalmic artery lies inferolateral to it, sphenoid and posterior ethmoid sinuses lie medial)
  - Intracranial – 10 mm (lies above cavernous sinus and converges with its fellow to form chiasma)

Parts of optic nerve



Optic chiasma

- It is a flattened structure measuring about 12mm horizontally and 8mm anteroposteriorly
- It is ensheathed by the pia and surrounded

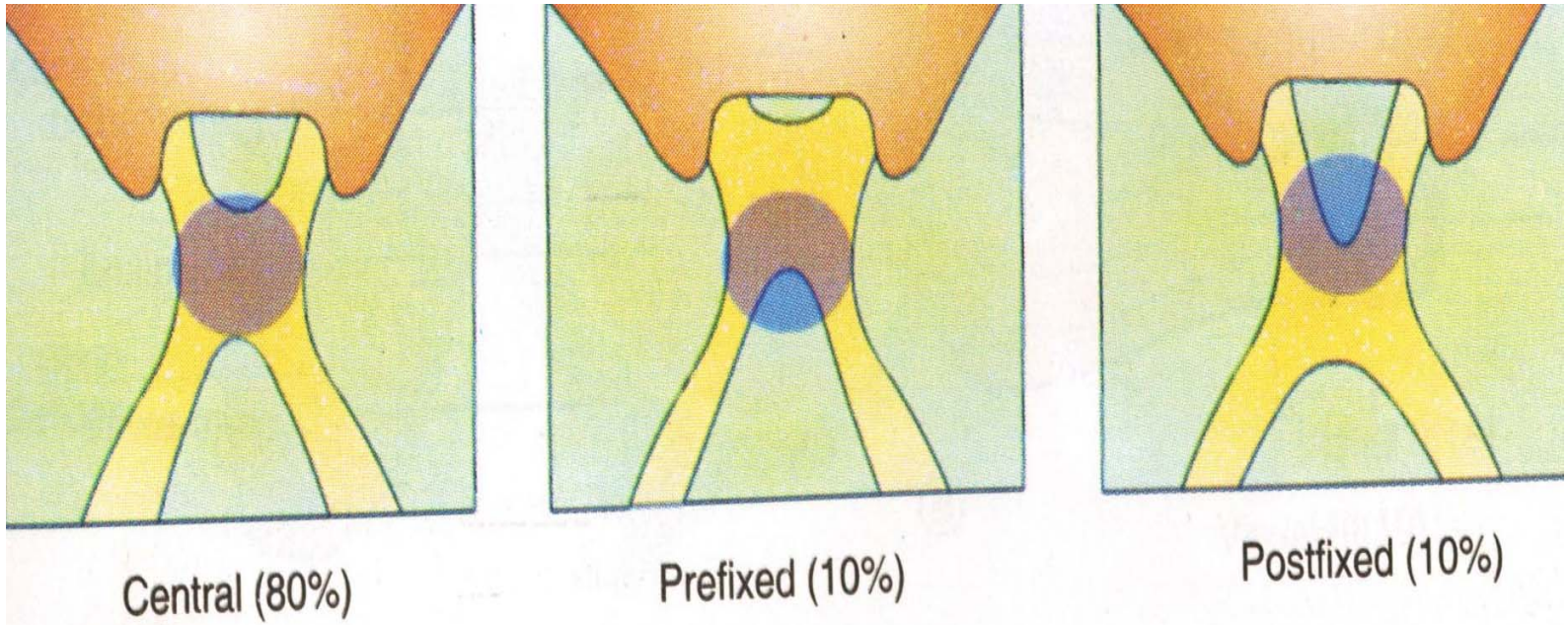
by CSF.

Variations in the location of chiasma

central chiasma

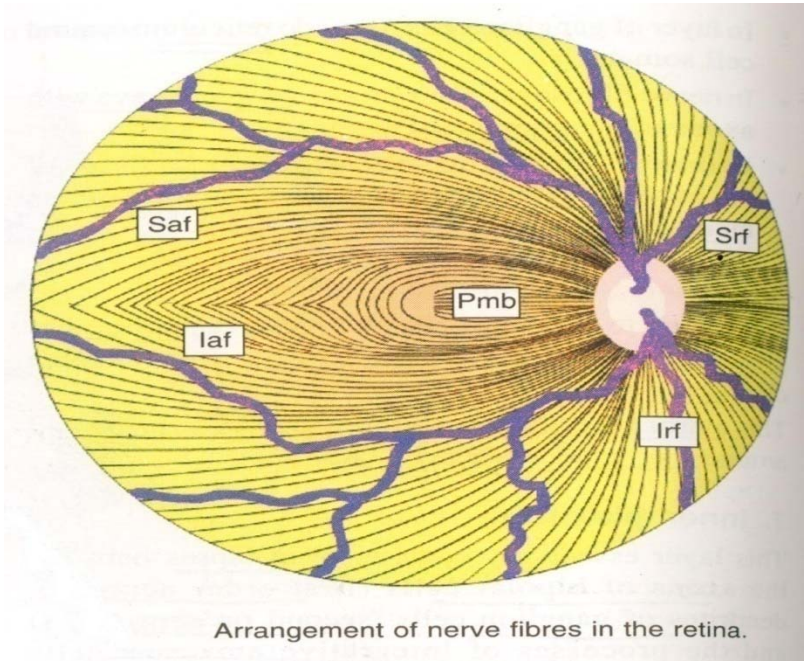
prefixed chiasma

post fixed chiasma



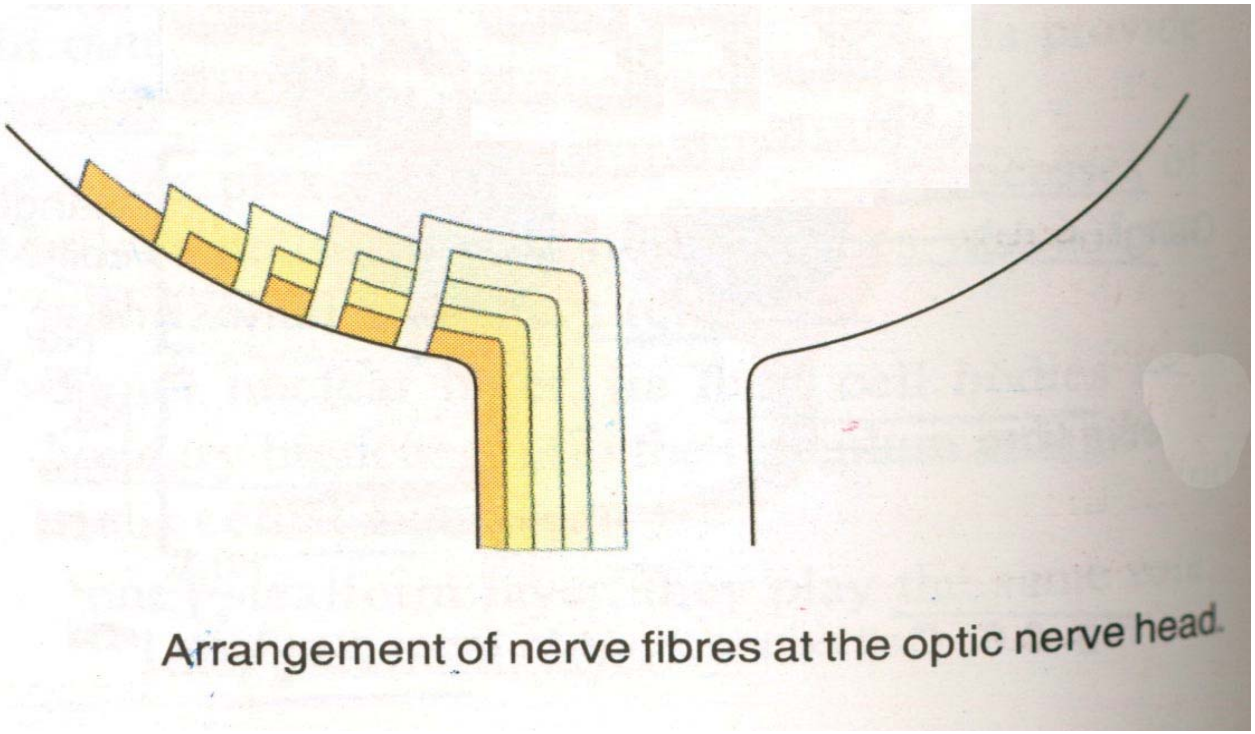
Anatomical variations in the position of normal optic chiasma.

Arrangement Of Nerve Fibers In Different Parts Of The Visual Pathway – Retina



Arrangement of nerve fibres in the retina.

In The Optic Nerve



Arrangement of nerve fibres at the optic nerve head.

In The Chiasma

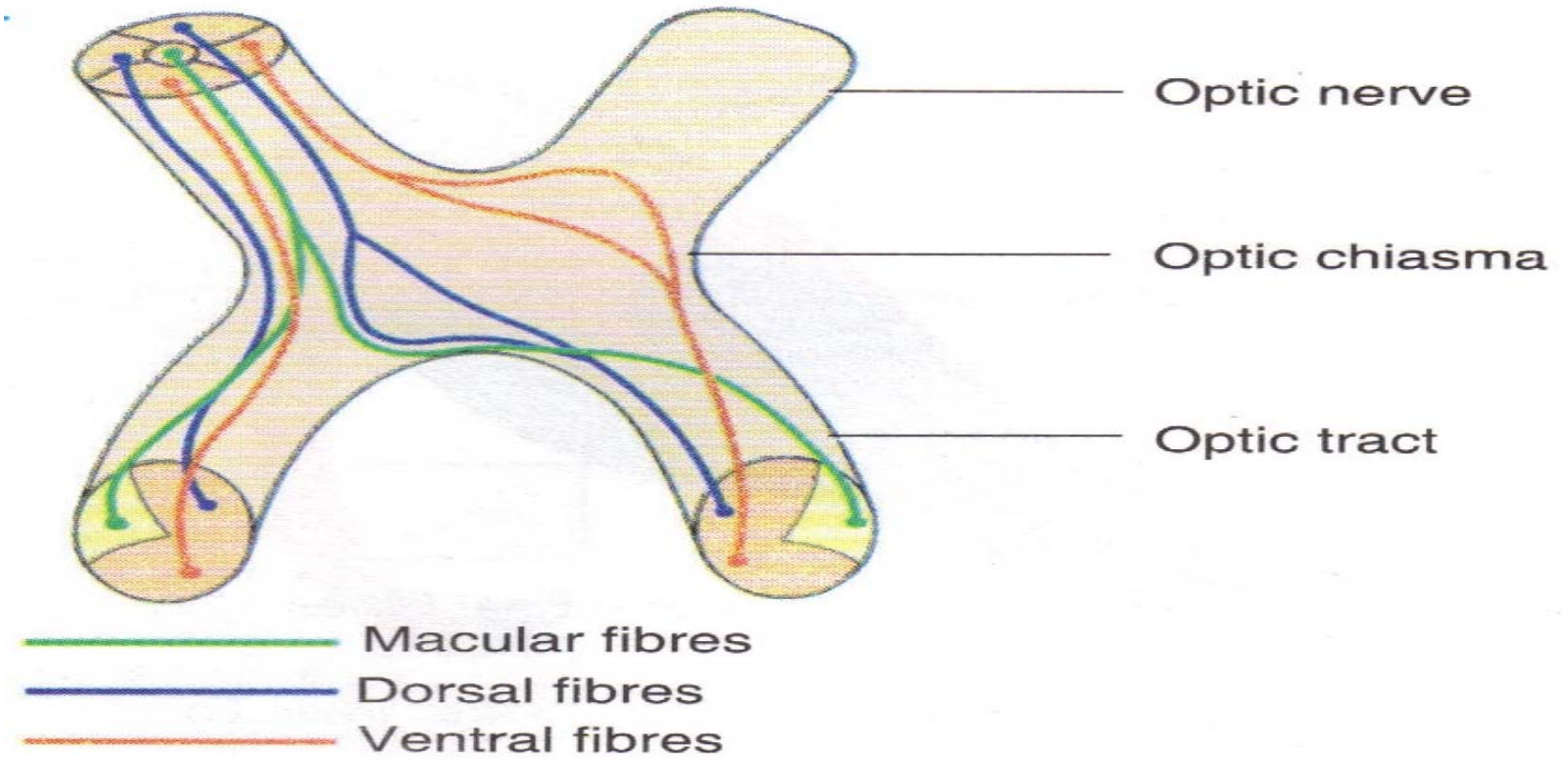


Fig. 6.23. Decussation of fibres in the chiasma.

Blood supply of optic nerve head

- Surface layer of optic disc – retinal arterioles
- Prelaminar region- centripetal branches of peripapillary choroid and vessels of lamina cribrosa
- Lamina cribrosa – posterior ciliary arteries and arterial circle of Zinn
- Retrolaminar part – branches from central retinal arteries and pial plexus

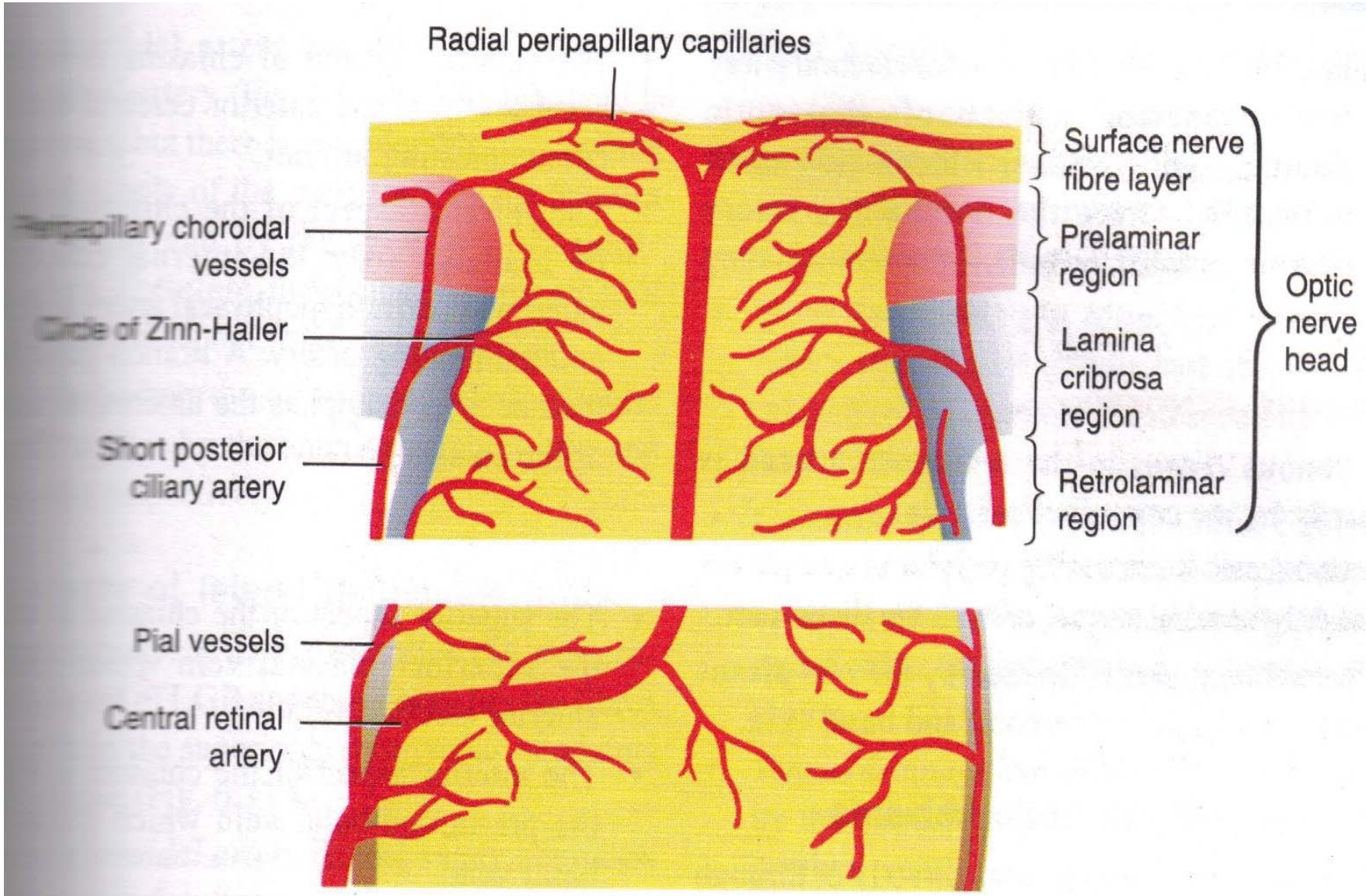
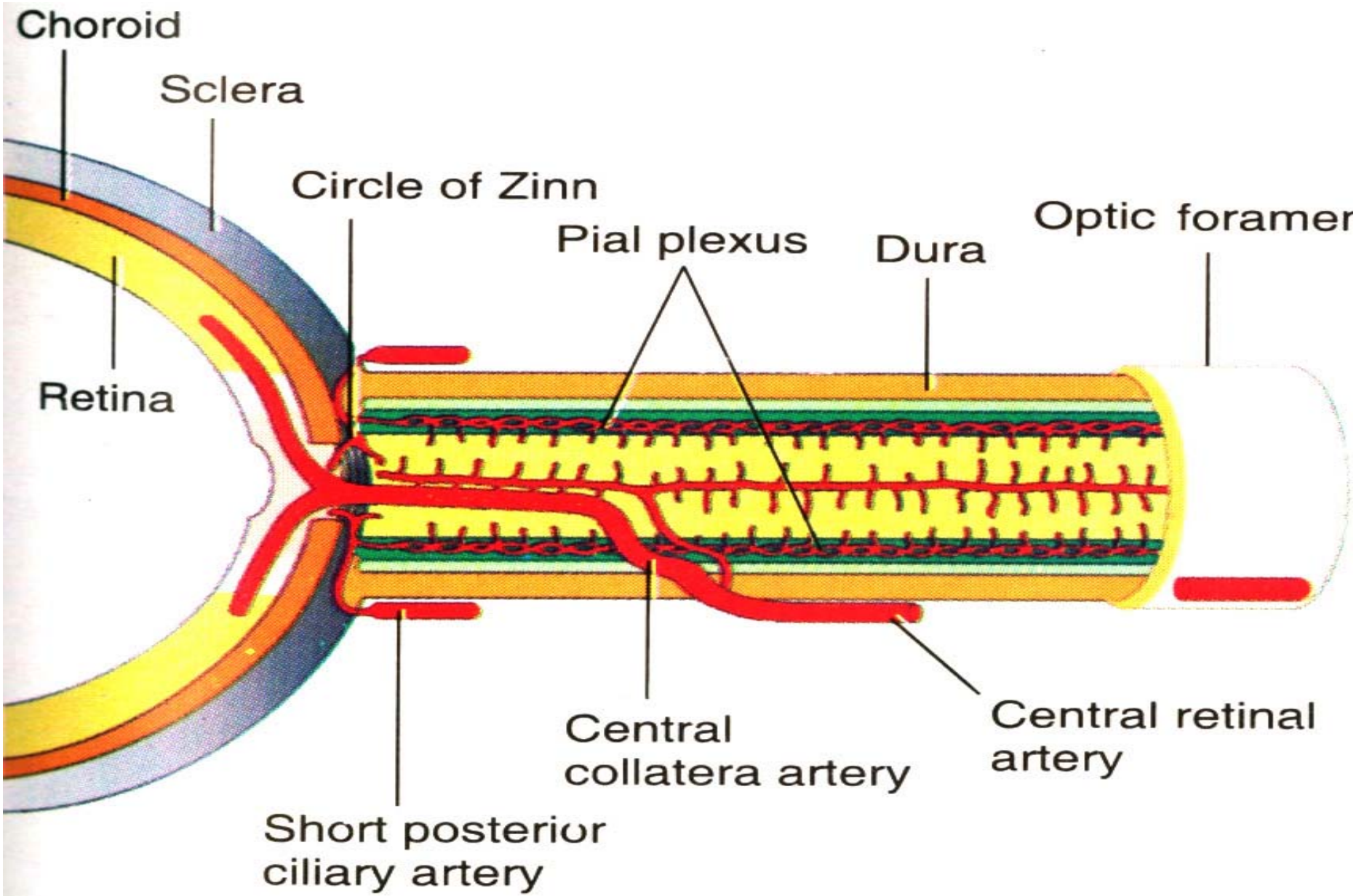
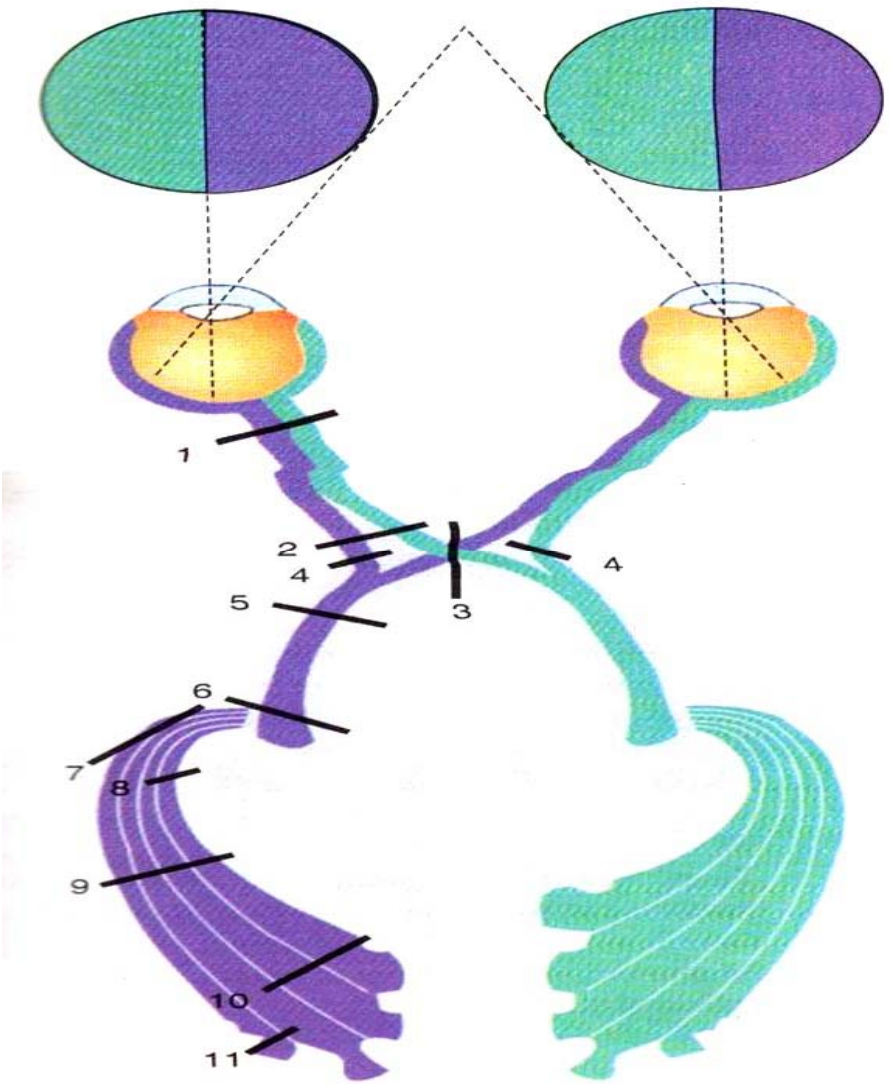


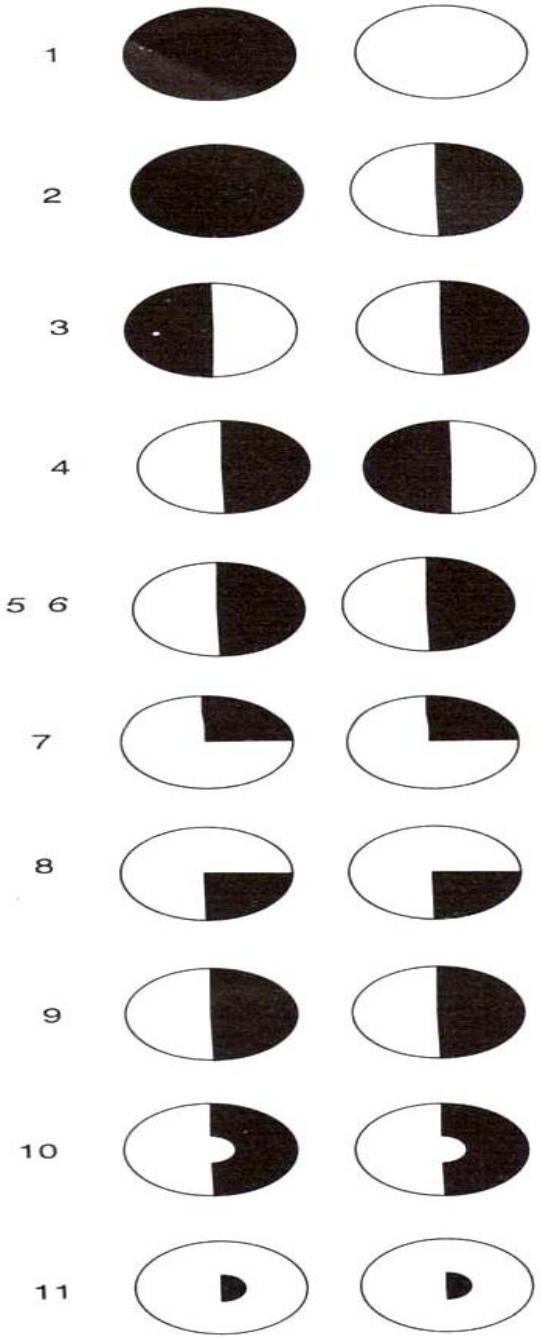
Fig. 6.29. Blood supply of the optic nerve head.



Blood supply of the optic nerve.



Normal field

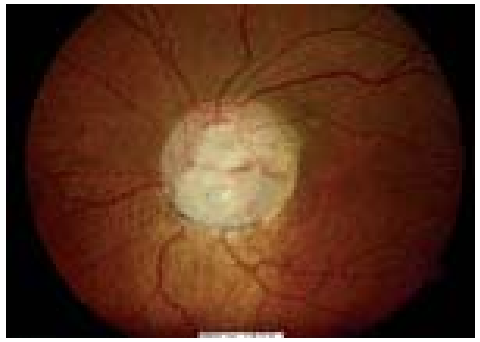


Diseases of optic nerve

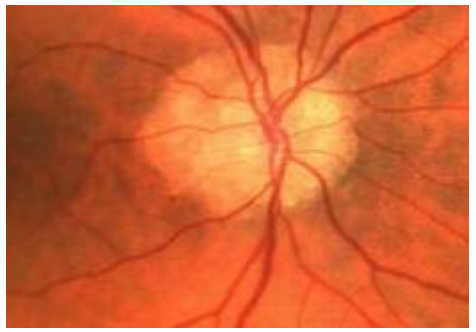
- Congenital anomalies
- Optic neuritis
- Anterior ischemic optic neuropathy
- Papilloedema
- Tumours

Congenital anomalies

- Coloboma of optic disc



- Drusen of optic disc



- Hypoplasia of optic disc

Optic neuritis

- Diagnosis of ON is basically a clinical one
- Nettleship (1884) first described a syndrome characterised by failure of sight ,often accompanied by pain in moving the eye .
- Subsequently parinaud (1884),Uththoff (1890) Buzzard(1893),Gunn (1897) described similar patients.

- Optic neuritis is -inflammation of the optic nerve
- When associated with a swollen disc it is called papillitis
- When the optic disc appears normal- retrobulbar neuritis
- Neuroretinitis – inflammatory involvementof ON & peripapillary retina
- Optic perineuritis – Inflammation of ON sheath

Ophthalmoscopic classification :

- Retrobulbar neuritis : in which optic disc appearance is normal

  It is the most frequent type seen in adults & is frequently associated with edema
- Papillitis : in which the pathological process affects the optic nerve head . It is characterised by variable disc hyperemia & It is the most common type of optic neuritis in children.
- Neuroretinitis :is characterised by papillitis in association with a macular star shaped pattern of hard exudates.

Aetiological Classification

- Demyelinating – the most common cause
- Parainfectious – which may folllow a viral infection or immunization.
- Infectious – which may be sinus –related or associated with a cat scratch fever , syphillis ,lyme disease &

  cryptococcal meningitis in pts with AIDS

Walsh & Hoyt's clinical neuro ophthalmology

- Idiopathic & primary demyelinating optic neuritis
- Acute idipathic demyelinating optic neurtitis
- Chronic demyelinating optic neuritis
- Subclinical optic neuritis
- Neuromyelitis optica (Devic's disease)
- Optic neuritis in myelinoclastic diffuse sclerosis (Schilder's disease)

- Optic neuritis from viral & bacterial diseases
- Optic neuritis after vaccination
- Optic neuritis in Syphillis
  - Sarcoidosis
  - SLE & other vasculitis
  - HIV patients
  - Lyme disease
  - Sinus disease

- BE optic neuritis in children
- Neuroretinitis
- Optic perineuritis
- Optic neuropathies that mimic acute neuritis

- Most common cause of optic neuritis
- Isolated or associated with MS

  ACUTE
  CHRONIC
  SUBCLINICAL
- Much information regarding optic neuritis has been obtained from ONTT

- Age : 20-50 yrs, Avg 32± 7 yrs (ONTT)
- Sex : Females > males 77 % female (ONTT)
- Race : Caucasian 85% (ONTT)
- Incidence 5.1 per 100,000 person - years

  prevalence – 115/100,000

Neurology 1995

- Loss of central visual acuity
  - over 90%(optic neuritis study group)
  - usually abrupt
  - monocular in most cases
  - Degree of visual loss varies widely
- Ocular or orbital pain
  - > 90%
  - usually mild
  - may precede or occur concurrently with visual loss

exacerbated by eye movement { helpful in differentiating from AION

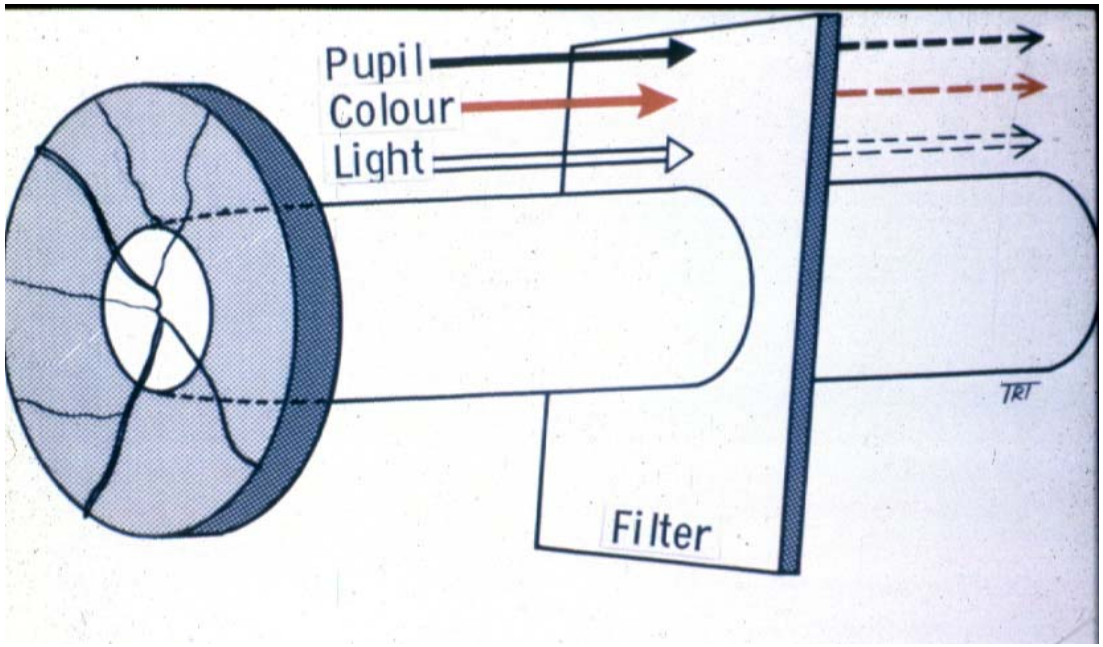
Generally lasts only for a few days

ONTT – 92 % pts

Pain is initiated by inflammation of the optic nerve in the apex of the orbit, where the extraocular muscles are firmly attched to the sheaths of the nerve *J Neuro Ophthal 1995*

- Positive visual phenomenon (photopsias)

  30% of pts in ONTT



- **Reduced visual acuity**
- **Afferent pupillary conduction defect**
- **Dyschromatopsia**
- **Diminished light brightness sensitivity**

- **Visual acuity** : mild reduction to no light perception

(ONTT 1991)

20/20-11%
20/5-20/40 – 25%
20/50-20/190 – 29%
20/200-20/800-20%
CF-4%
HM-6%
LP-3%
No PL-3%

- **Colour vision : Almost always abnormal in ON**

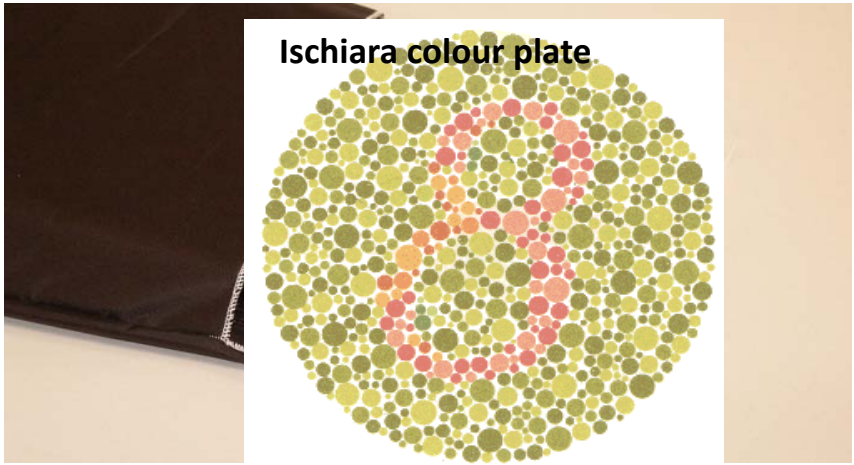
  Usually more severely affected than visual acuity

  Ischiara colour plates – abnormal in 88%

  Farnsworth Munsell 100 hue test – 94%

  (ONTT Gp)

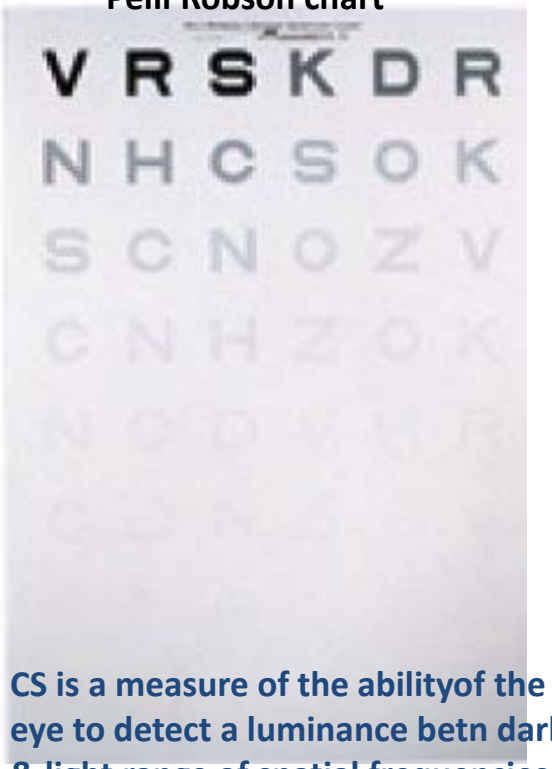
More sensitive – recommended for detection of various optic neuropathies



farnsworth Munsell 100 hue

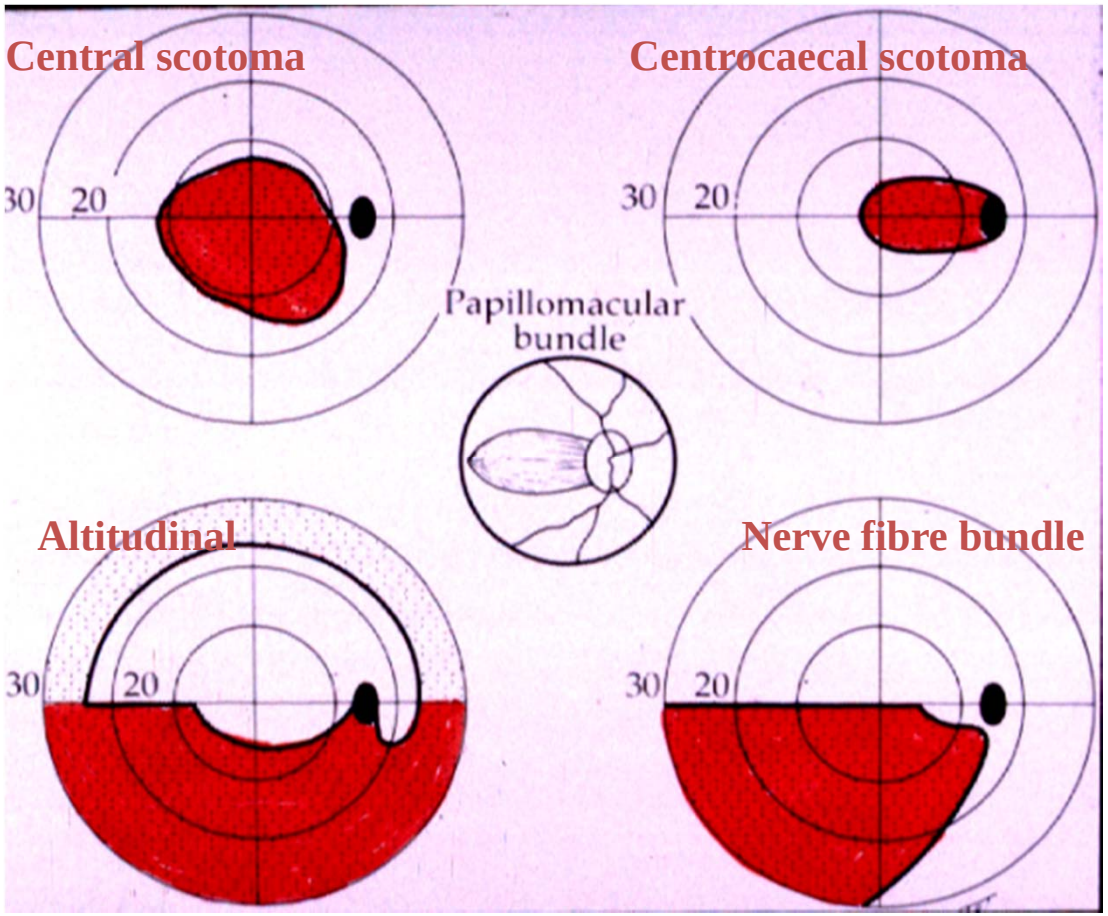
- Reduction in contrast sensitivity often parallels the reduction in visual acuity ***Neuro Ophthalmol 1984***
- Abnormal in 93 % in acute phase & 78 % in resolved phase. Even when VA improved 67% still showed CS abnormality – (***BJO 1884*** ***'CS measurements in acute & resolved Optic neuritis')***
- ONTT – 98% abnormal CS

Pelli Robson chart



CS is a measure of the abilityof the eye to detect a luminance betn dark & light range of spatial frequencies

Arch Opththalmol 1993,1994

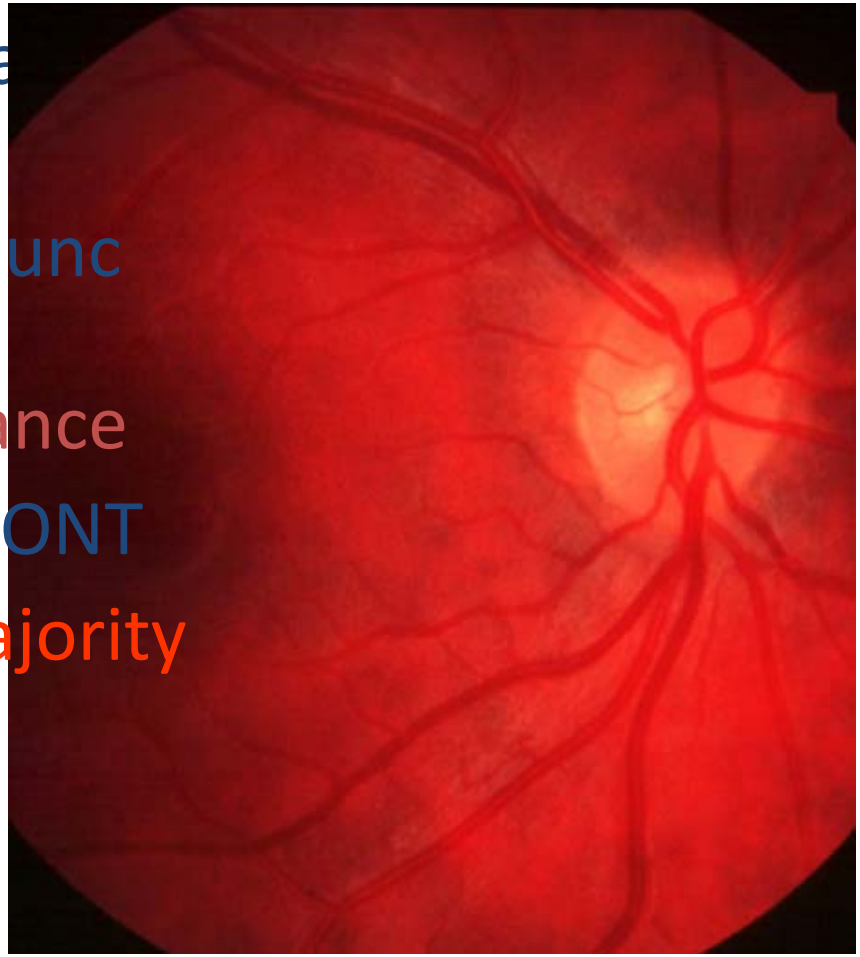


Visual Field : typical VF defect Central Virtually any type of field defect cn occur in an eye with ON including an arcuate,centrocaecal,altitudinal,paracentral,hemianopic.

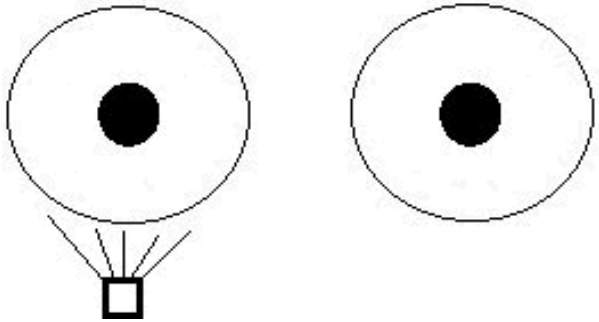
In ONTT focal 52 %& diffuse in48%arcuate,altitudinal,nasal were +nt more frequently than Central,centrocaecal-8%

- Pupillary reaction : RAPD a
  unilateral cases
- Neutral density filter may unc

- Ophthalmoscopic appearance
  - optic disc swelling 35% (ONT
  - Normal looking disc – majority
  - CHRONIC cases
    - Diffuse pallor
    - Temporal pallor



“Swinging flashlight test".



In a person with two normal eyes, if we shine the light on one eye, the pupil of that eye constricts immediately; then if we swing the light to the other eye, that pupil also constricts immediately. However, if one eye has retinal or optic nerve disorders, then if we first shine the light on the normal eye that pupil constricts, and then shine the light on the bad eye, instead of constricting the pupil immediately dilates in that eye

- Diagnostic studies : CT scan

  MRI preferred

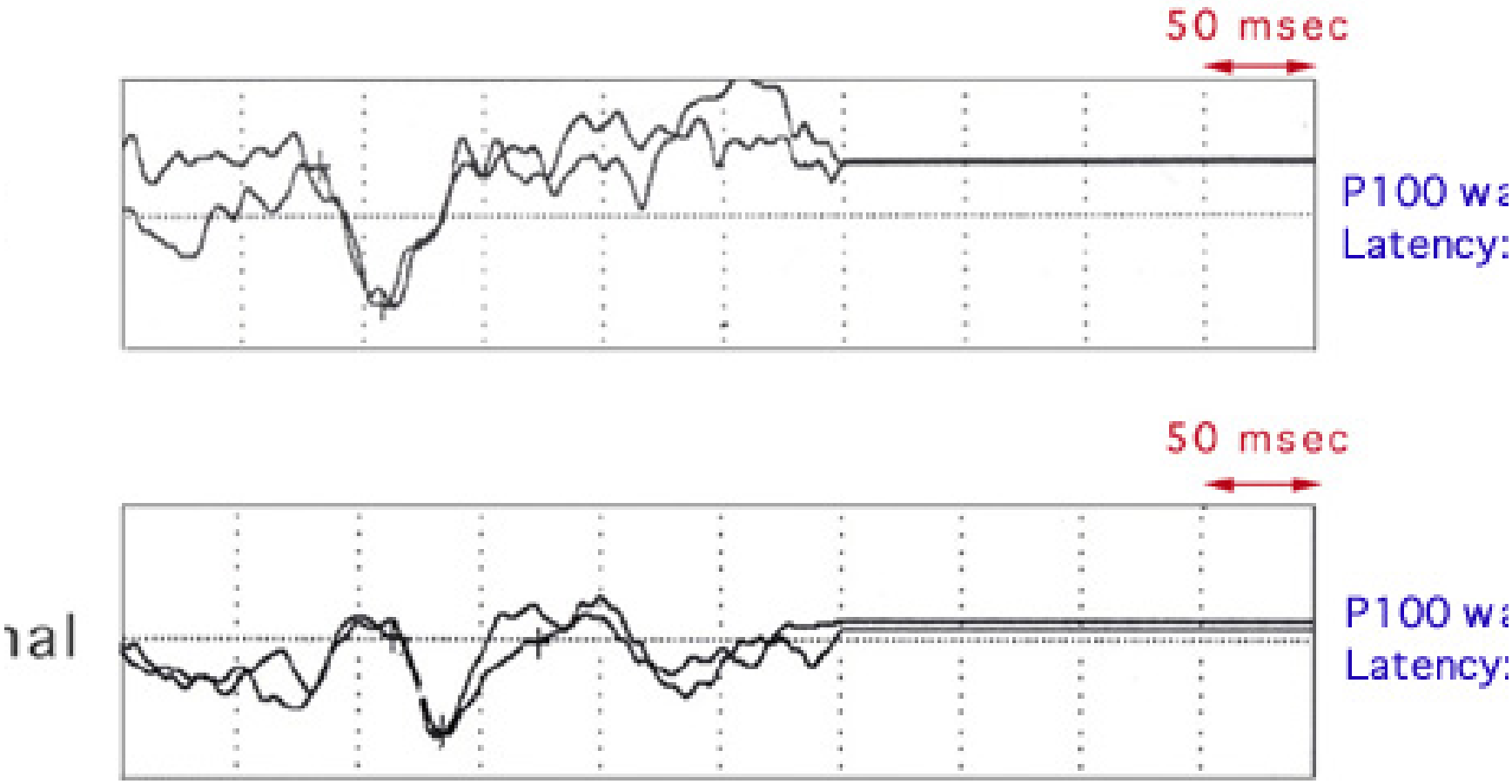
  unwarranted in pts with a typical history & findings suggestive of ON

- Etiological studies –

  H/O sarcoidosis , syphillis, SLE , Lymes disease

  (ANA, FTA AbS,CXR)

al Evoked Potentials

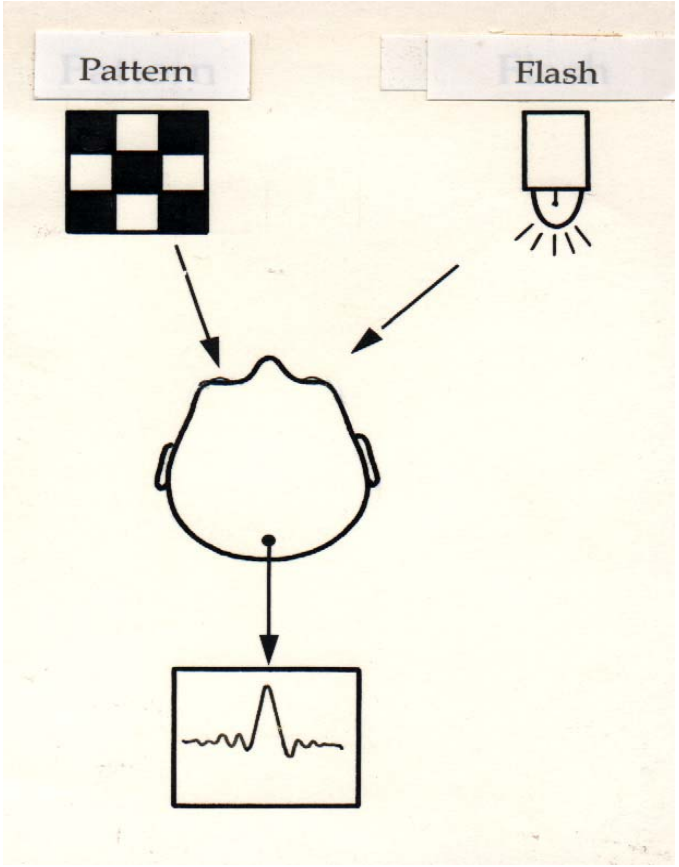


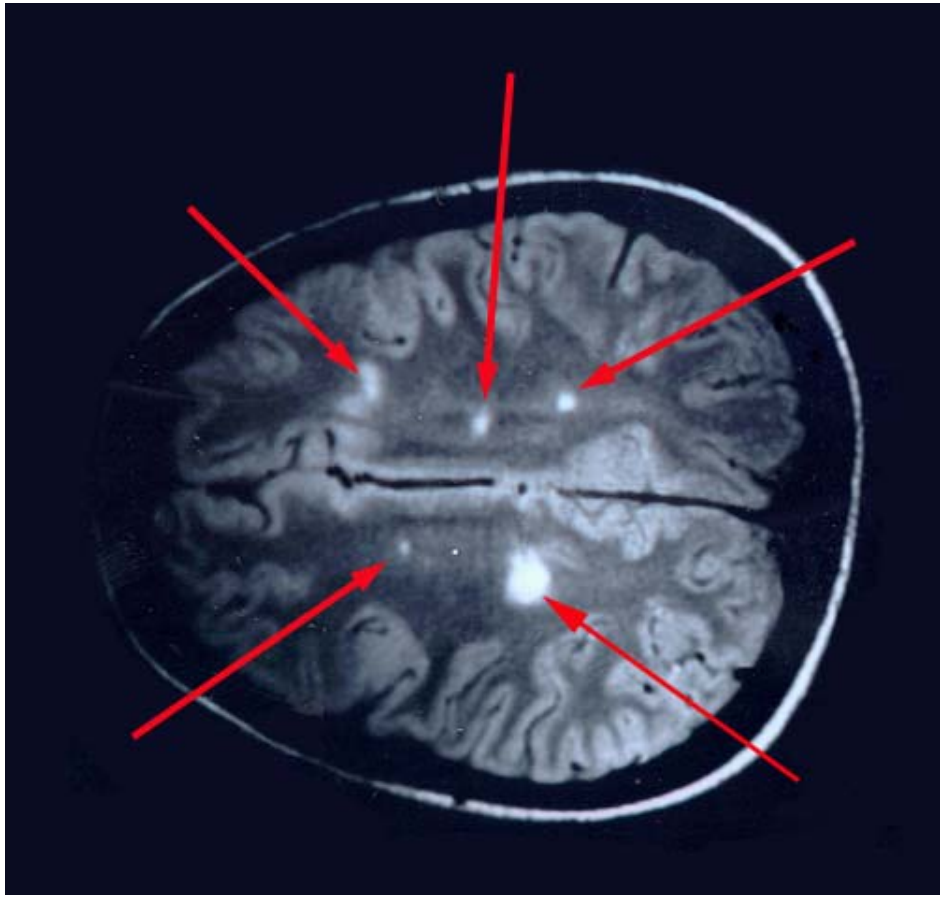
- Pattern ERG
- Pattern VEP

Assessment of electrical activity of visual cortex created by retinal stimulation.

Delayed VEP latency

Near normal ERG amplitude in optic neuropathy

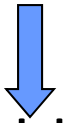




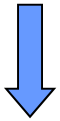
Brain MRI abnormality – strong predictor of CDMS
MRI – multiple lesions :
periventricular

other white matter

Demyelination of optic nerve



Complete conduction block OR Slowing of conduction



Failure to transmit transmit rapid train of impulse.

{ demyelination of white matter}

ONTT

Beck et al N Eng J Med 1992

Three Groups

i. Oral prednisolone 1mg/kg/day - 14days

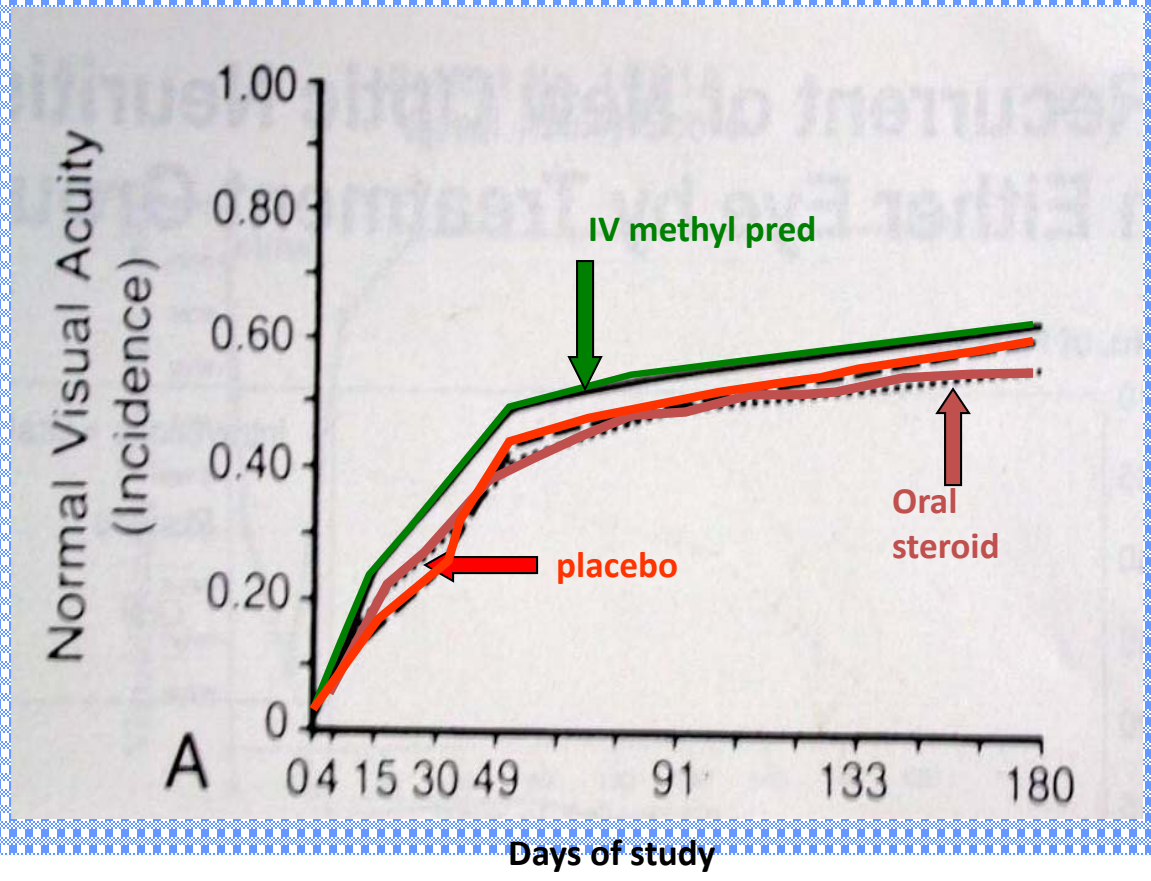
ii. IV methyl Pred 250 mg qid + 1mg/kg/day 11days oral prednisolone

iii. Placebo - 14 days

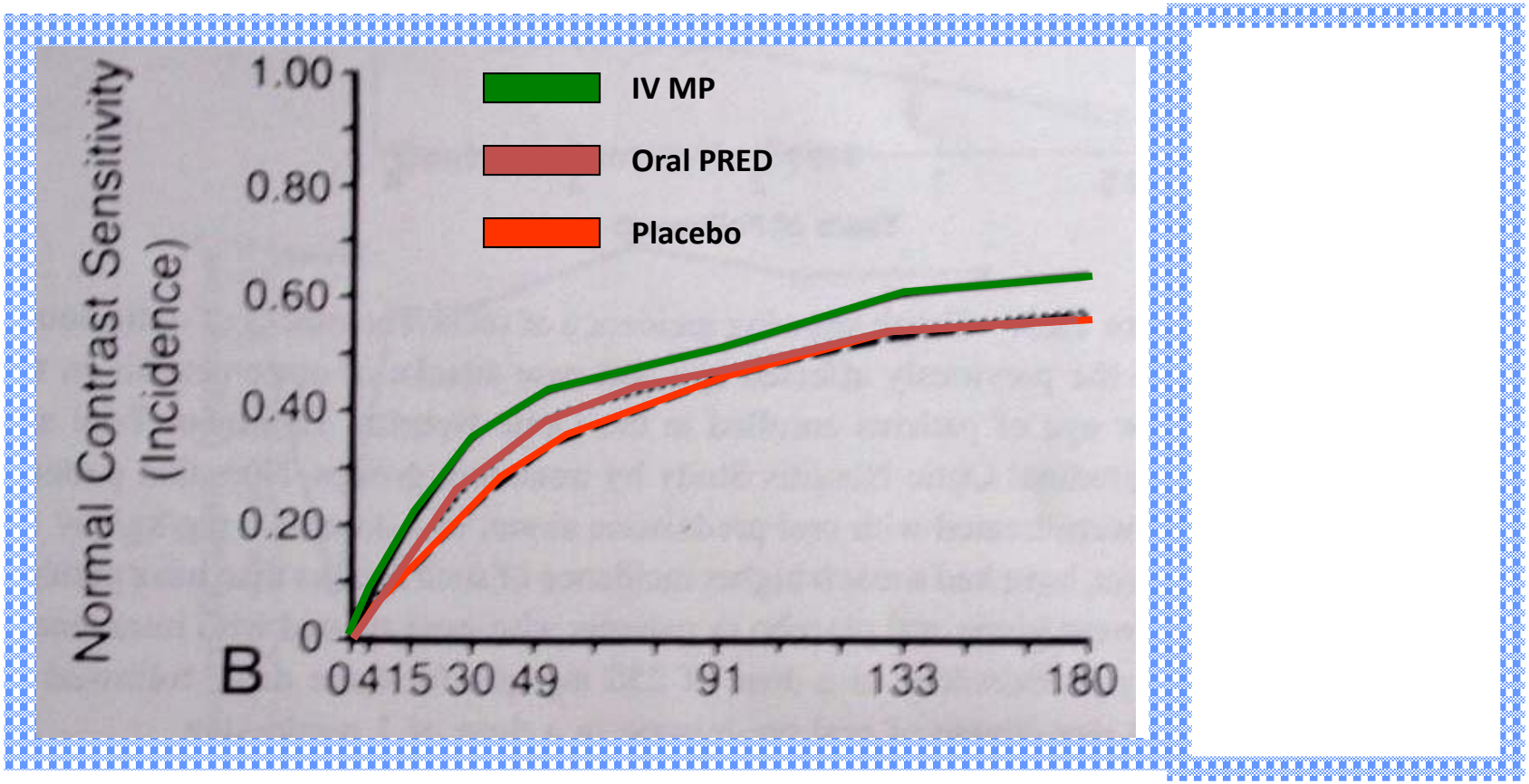
Arch Ophthal 1997

- **Most retained good visual outcome**
- **87% affected eye > 20/25 or better in a 5 yr study**
- **(After 5 yrs)**

Visual function test	% of abnormal eyes
Visual acuity	37 %
Contrast sensitivity	59 %
Colour vision	33 %

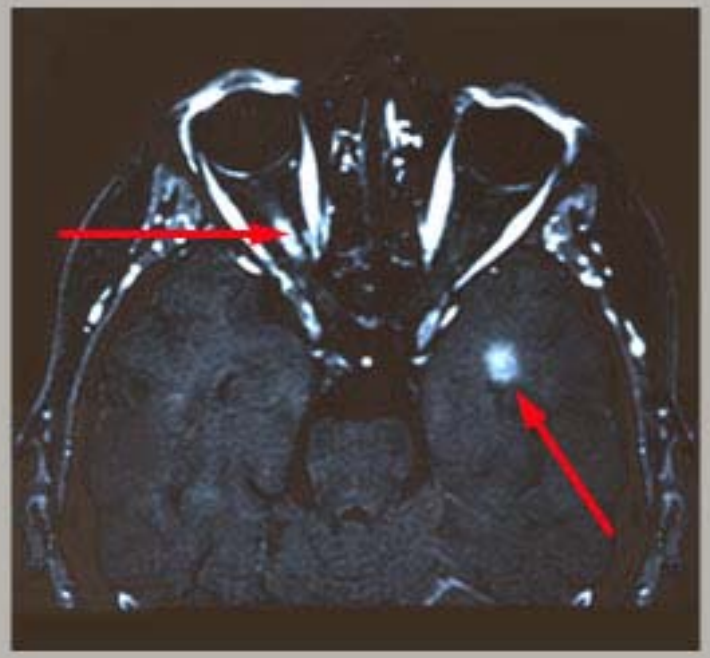


After 6 months , median visual acuity -20/16 & less than 10 % of pts in each group had a VA of 20/50 or worse.


Normal Contrast Sensitivity (Incidence)
1.00
0.80
0.60
0.40
0.20
0
B
0 4 15 30 49 91 133 180
IV MP
Oral PRED
Placebo

- There is no Rx for optic neuritis that can improve ultimate visual prognosis
- Intravenous therapy - Increase in the speed of recovery of vision by 2-3 wks
- Oral steroids alone does not improve visual outcome or speed recovery but is associated with a significantly higher incidence of recurrent attacks of optic neuritis

- ON occurs in 50 % (*Survey Ophtalmol 1991)* of pts with MS & in 20 % is the presenting sign



This MRI scan from a patient with acute opticneuritis. This MRI scan shows enhancement of involved area in optic nerve (left top arrow).

A second area of contrast enhancement is seen in the contralateral lobe (right lower arrow).

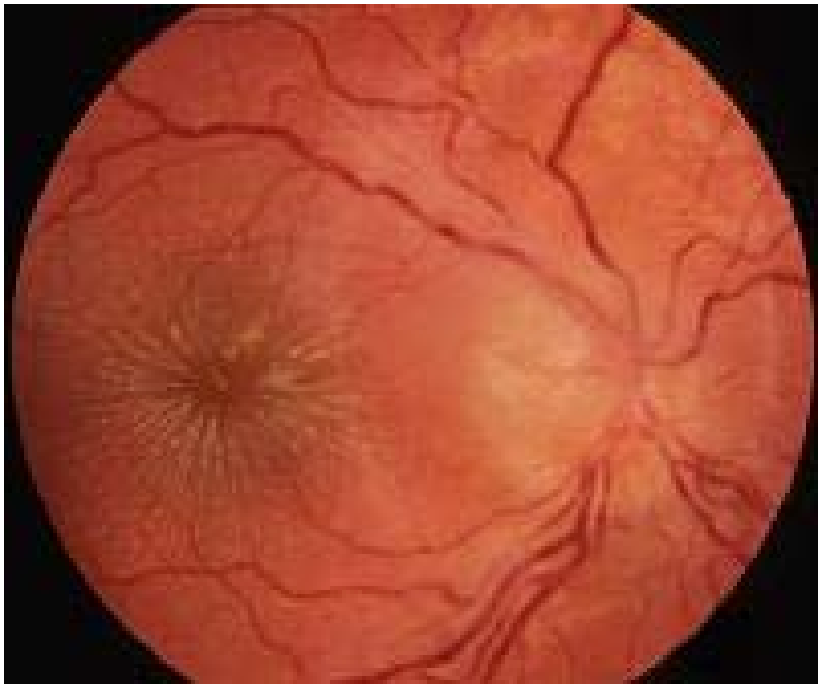
- Close relationship betn ON & MS more important than its visual prognosis
- MRI is a single more important predictor of future CDMS
- What is CDMS?

  CDMS is diagnosed when a pt develops new neurological symptoms attributable to demyelination in one or more regions of the CNS, other than Optic neuritis occuring atleast 4 wks after ON & lasting more than 24 hrs with abnormalities on neurological examn

More commonly associated with HIV

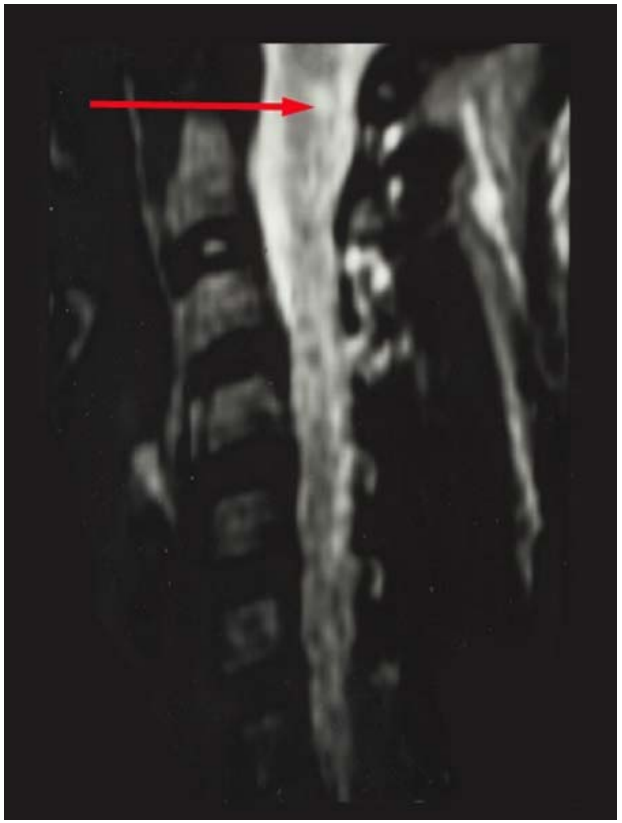
Cellular reaction in vitreous ,

Neuroretinitis more common.

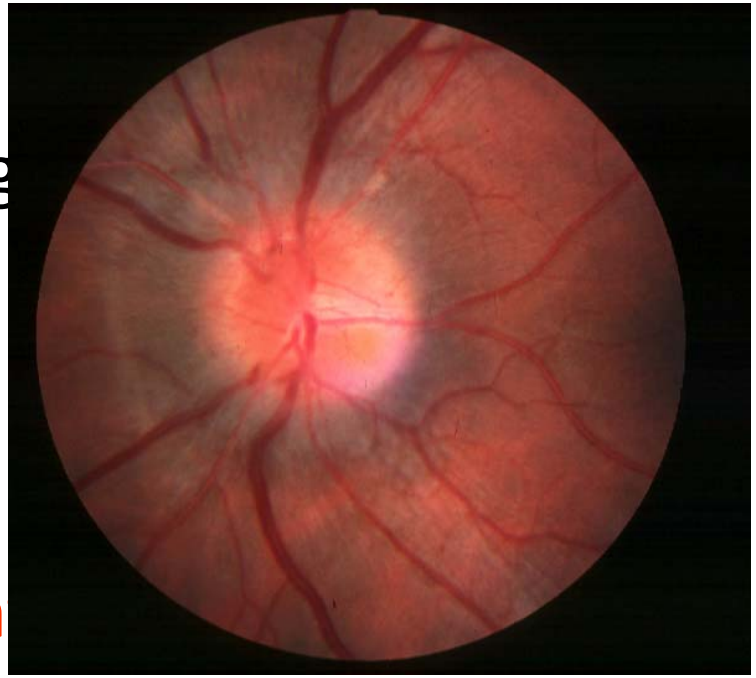


Neuromelitis optica

- Children & young adults
- Visual loss rapid, bilateral
- Spinal cord demyelination
- Paraplegia / bladder involvement



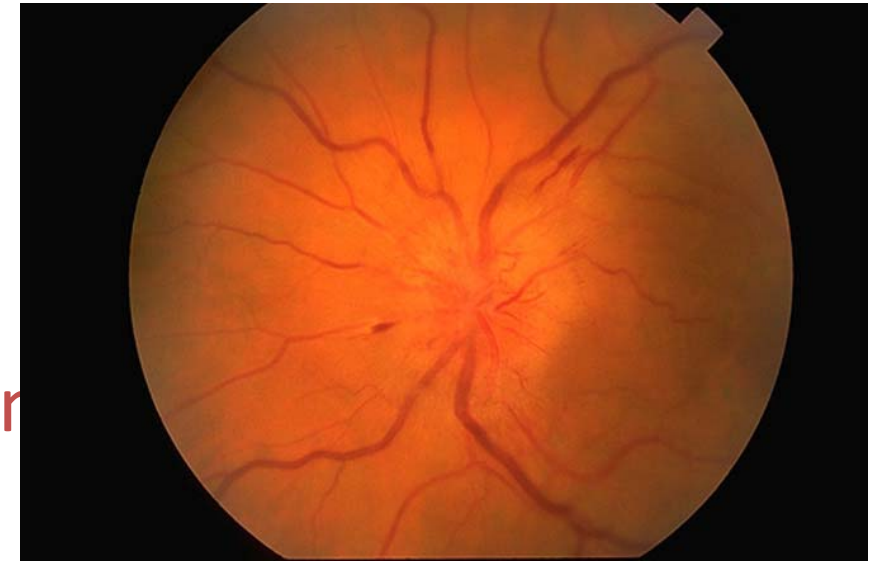
- More common in children than adult
- One to three wks following viral infection
- Usually bilateral
- Optic disc may be normal or swollen
- Visual recovery is excellen with or without treatment



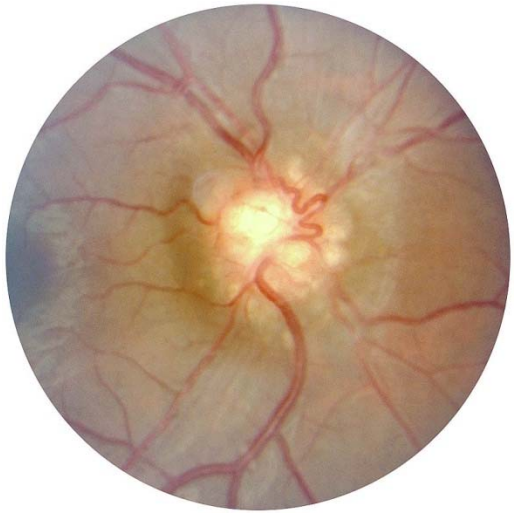
BCG,HBV,Rabies vaccine.

Usually 1-3 wks following vaccination

Ant variety disc oedema



- Granulomatous inflmn of ON producing ant or retrobulbar optic neuritis
- Optic disc characteristically lumpy white appearance
- Other ocular signs of sarcoidosis
- Rapid response to steroid therapy & subsequent worsening when steroids are tapered



Typical sarcoid nodules of the right optic disc in a 21-year-old Black man with biopsy-proven sarcoidosis.

- Optic neuritis both anterior & retrobulbar
- Probably caused by infection of the ON by HIV virus itself
- Opportunistic infection
- Cryptococcal meningitis
- Cytomegalovirus infection
- Herpes virus infection
- TB meningitis

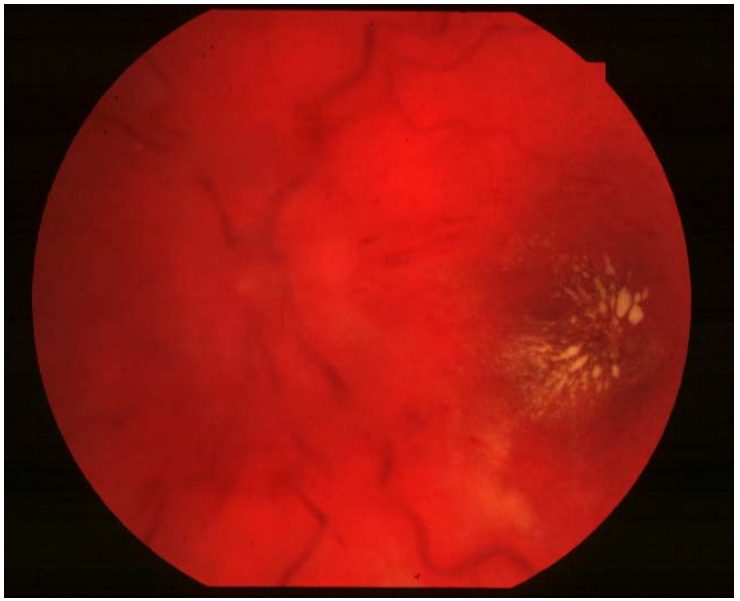
- SLE , PAN , other vasculitis
- Pathology : vasculitis → ischaemia

↓

demyelination

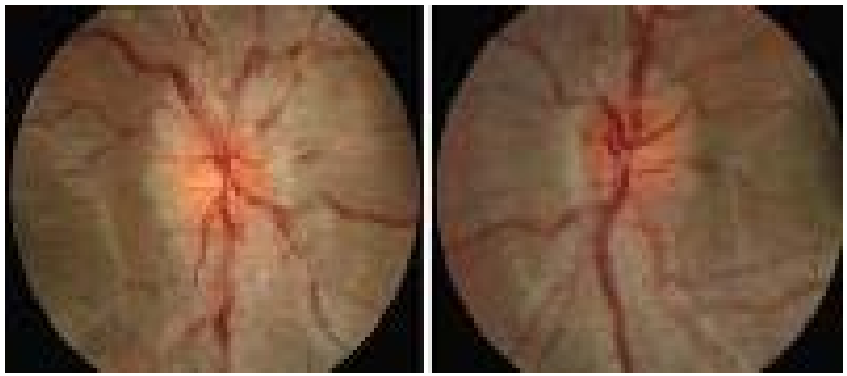
Diagnosis : systemic signs & symptoms , ANF ,ANCA

- Skin lesions
- Neurological signs
- Anterior , retobulbar optic neuritis
- Also cause neuroretinitis



- Pre antibiotic era
- Spread from paranasal sinus → optic nerve
  (aspergillosis / other fungal infections)

- More often anterior (disc swelling)
- Occur within 1-2 wks of presumed viral infection
- Bilateral simulatneous
- It is less often associated with MS
- Steroid sensitive & steroid dependent



- Inflammation of ON sheath
- Exudative
  Purulent

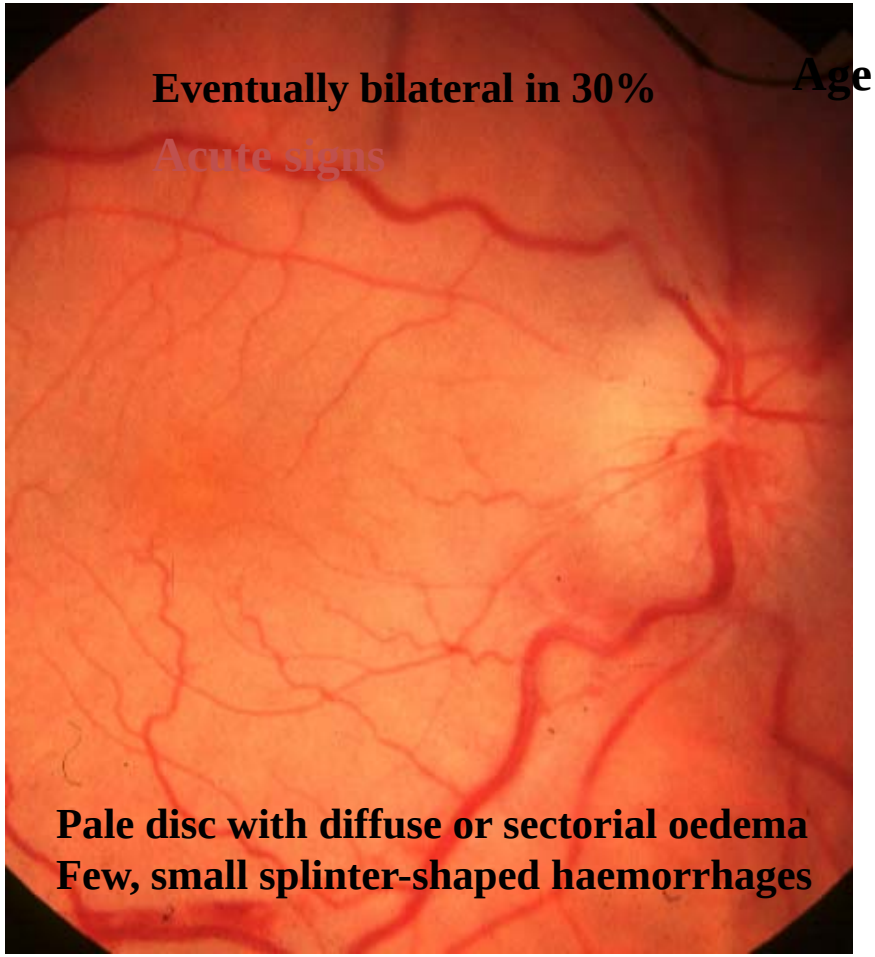
- Optic disc swelling without visual symptoms
- Enlargement of Blind spot

- Age usually above 60 yrs
- Rapid visual loss
- NOT associated with ocular pain
- Typical altitudinal field defect
- Pale disc oedema

  segmental oedema

  Flamed shaped hamerrohage
- FFA - AAION Delayed disc & choroidal filling

  NAION : Delayed disc filling



Eventually bilateral in 30%

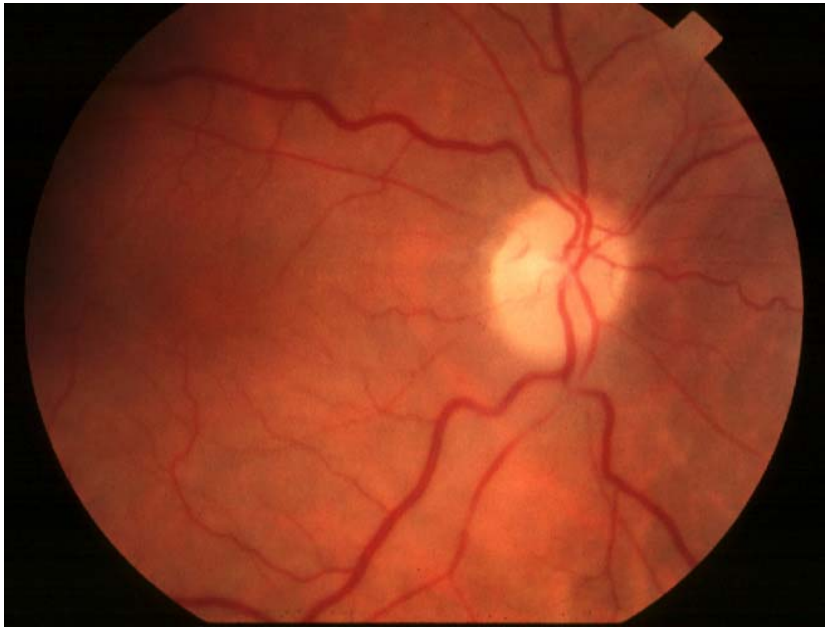
Acute signs

Pale disc with diffuse or sectorial oedema
Few, small splinter-shaped haemorrhages

Age - 45-65 years

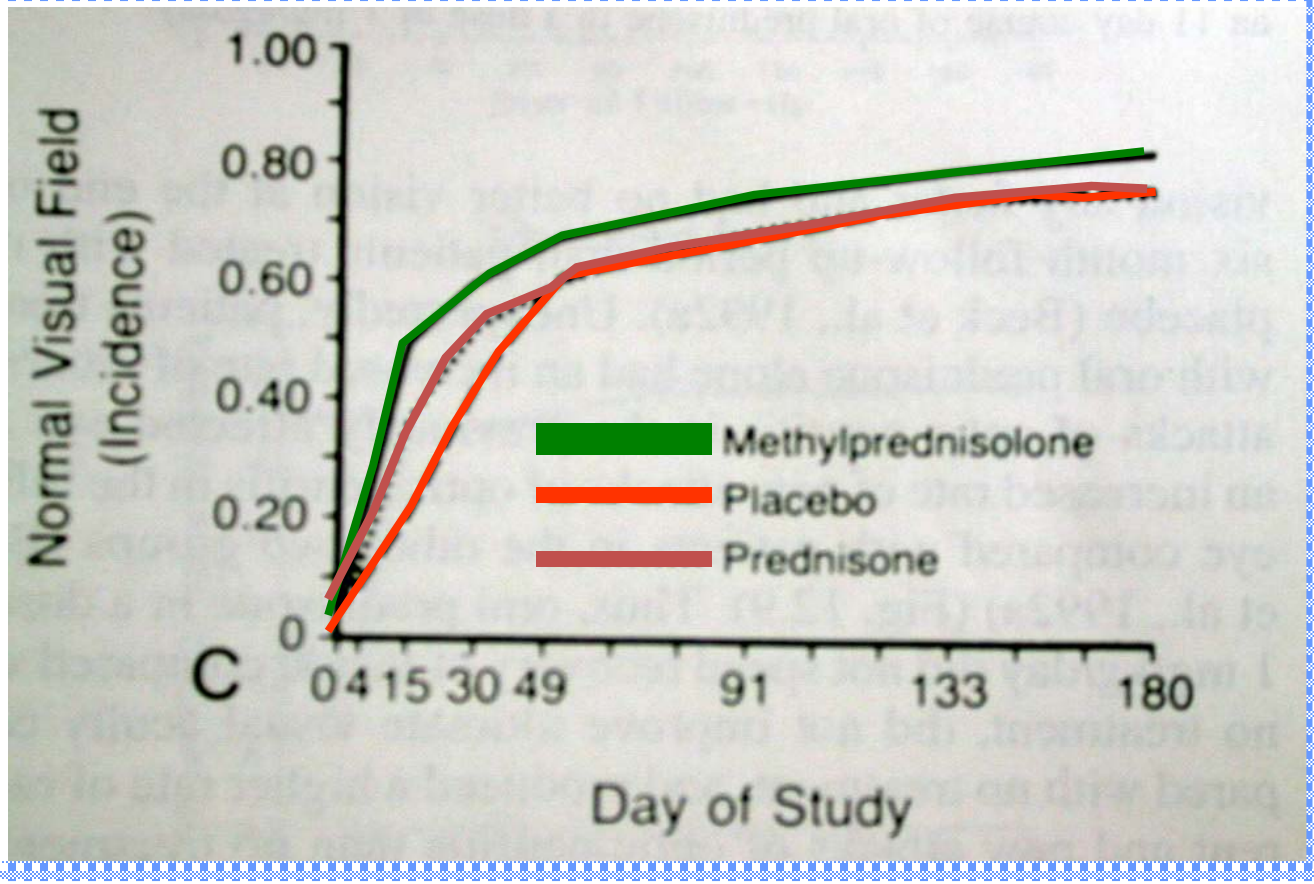
Altitudinal field defect

Late signs



Resolution of oedema and haemorrhages

·Optic atrophy and variable visual loss


Normal Visual Field
(Incidence)
1.00
0.80
0.60
0.40
0.20
0
C
0 4 15 30 49
91
133
180
Day of Study
Methylprednisolone
Placebo
Prednisone

Visual Acuity :Most pts recover to normal or near normal VA

ONTT after 12 mths VA > 20/20 in 69%, 20/200 in only 3%

Colour Vision : Persistant disturbances of CV +nt in a high% with otherwise resolved ON.In ONTT CV normal in 60 %

Visual Field : Residual visual field defects are usually +nt in eyes after resolution of acute ON even when VA has returned to 20/20 . **ONTT at 6mths 32% abnormal**

Contrast Sensitivity : remains abnormal in most eyes In ONTT at 6mths CS measured was abnormal in 56%

Stereopsis : worse than predicted by the level of VA

Pupillary reaction : Many pts have a persistent RAPD .

ONTT after 6mths 54%

Optic disc appearance: optic disc pallor is almost always +nt.In ONTT 63 % had disc pallor at 6 mths

VEP : most pts have a prolonged latency

Uhthoff's phenomenon : Foll. An episode of ON patients may complain of vision loss exacerbated by heat or exercise or emotional stress (ONTT after 6 mths 10 % reported symptoms)

- **Most of the pts have good visual recovery with or without treatment**
- **No Rx – Visual recovery starts within 2 wks**

maximum 1-2 months

continue upto 1 yr

Arch Ophthal 1997

19% for affected eye

17% for the fellow eye

30% for either eye

- Two fold more risk in pts who had or developed CDMS
- Two fold more frequent in pts Rx with oral prednisolone

90% pts had better vision after 2nd attack

- On MRI 50-70 % pts with ON have clinically silent MS like lesions
- MS like LESION (MRI) = situated in the white matter , high intensity , at least 3mm in size
- Risk of CDMS in 5yrs (ONTT exp) *neurology 1997*

  3 or more MRI lesions – 51%

  no MRI lesions - 16%

- Lack of pain
- Presence of OD swelling
- Mild visual loss

Acta Scan 2001

- Beneficial in MS by
  - reducing relapse
  - delaying progression of disability
  - decrease MRI evidence of disease

IFβ is NOT a CURE

Survey Ophthal 1991

Why tell the patient ?

Informing the patient allows him/her to make some important decisions regarding future & life style

- Thorough neurophthal examination
- Inv. - MRI brain

- If –ve → CSF study for oligoclonal bands

(Acta Ophtha Scan 1998)

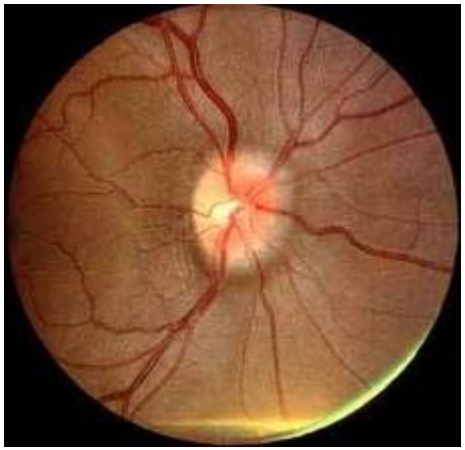
- One should point out the risk factors
- Stress that in MS - Spectrum of disability ranges from mild to severe disability
- In young women - Risk of exacerbation pregnancy & postpartum
- INF β reduces relapse rate and disability

Lebers disease

- Type of hereditary optic neuritis
- Young males around 20 yrs of age
- Transmitted by female carriers
- Progressive visual failure
- Disc hyperemia with telangiectic microangiopathy
- Eventually primary optic atrophy

Toxic amblyopias

- Tobacco
- Ethyl alcohol
- Methyl alcohol
- Ethambutol
- Quinine



Papilledema

Definition

- Papilledema is an optic disc swelling that is secondary to elevated intracranial pressure
- Vision usually is well preserved with acute papilledema
- Bilateral phenomenon and may develop over hours to weeks.

Pathophysiology

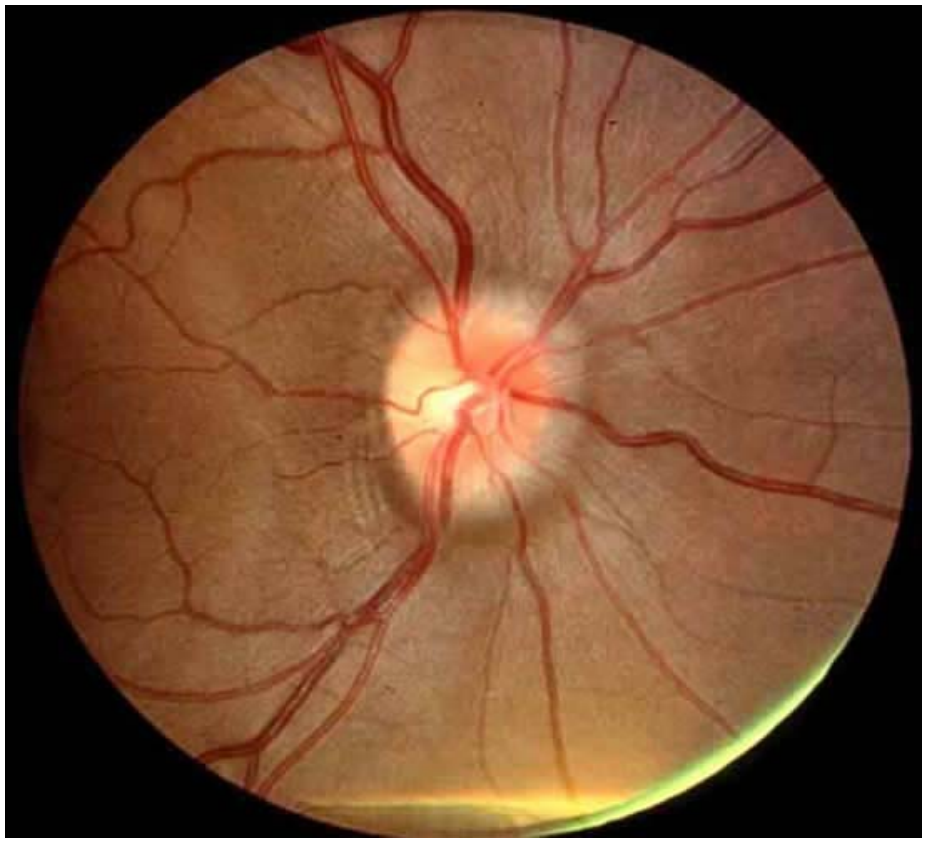
- The disc swelling in papilledema is the result of axoplasmic flow stasis with intra-axonal edema in the area of the optic disc.
- The subarachnoid space of the brain is continuous with the optic nerve sheath.
- Hence, as the cerebrospinal fluid (CSF) pressure increases, the pressure is transmitted to the optic nerve, and the optic nerve sheath acts as a tourniquet to impede axoplasmic transport.
- This leads to a buildup of material at the level of the lamina cribrosa, resulting in the characteristic swelling of the nerve head.
- Papilledema may be absent in cases of prior optic atrophy. In these cases, the absence of papilledema is most likely secondary to a decrease in the number of physiologically active nerve fibers.

Symptoms

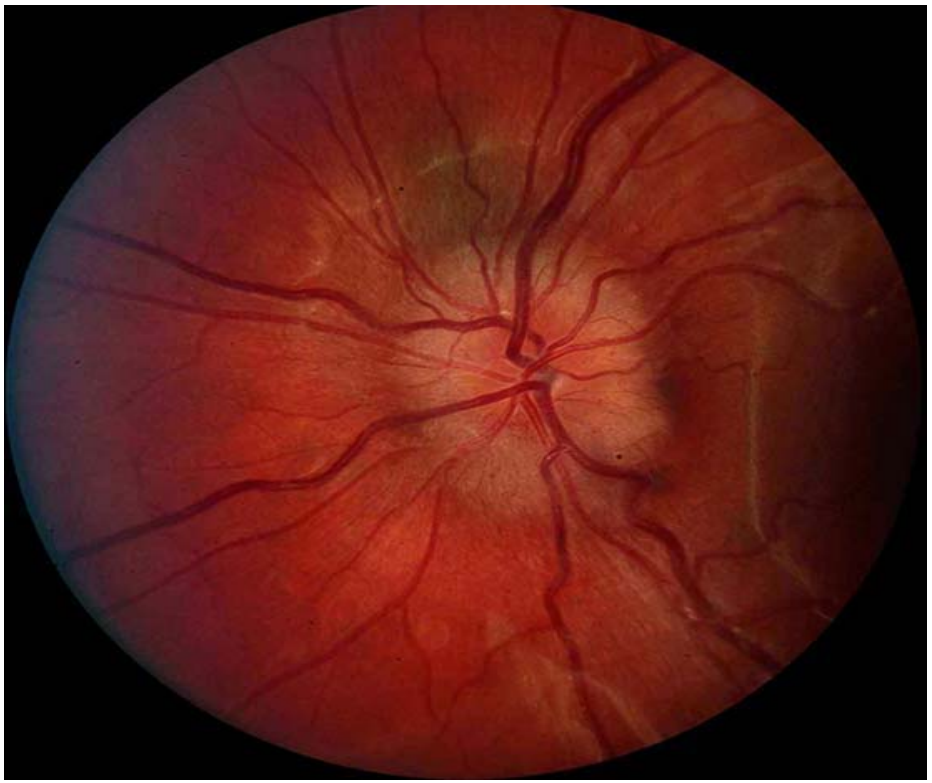
- Most symptoms in a patient with papilledema are secondary to the underlying elevation in intracranial pressure
  - Headache
  - Nausea and vomiting
  - Pulsatile tinnitus
  - Transient visual obscurations
  - Blurring of vision, constriction of the visual field, and decreased color perception may occur.
  - Diplopia may be seen occasionally if a sixth nerve palsy is associated.
  - Visual acuity may be well-preserved, except in very advanced disease.

Grades of papilledema

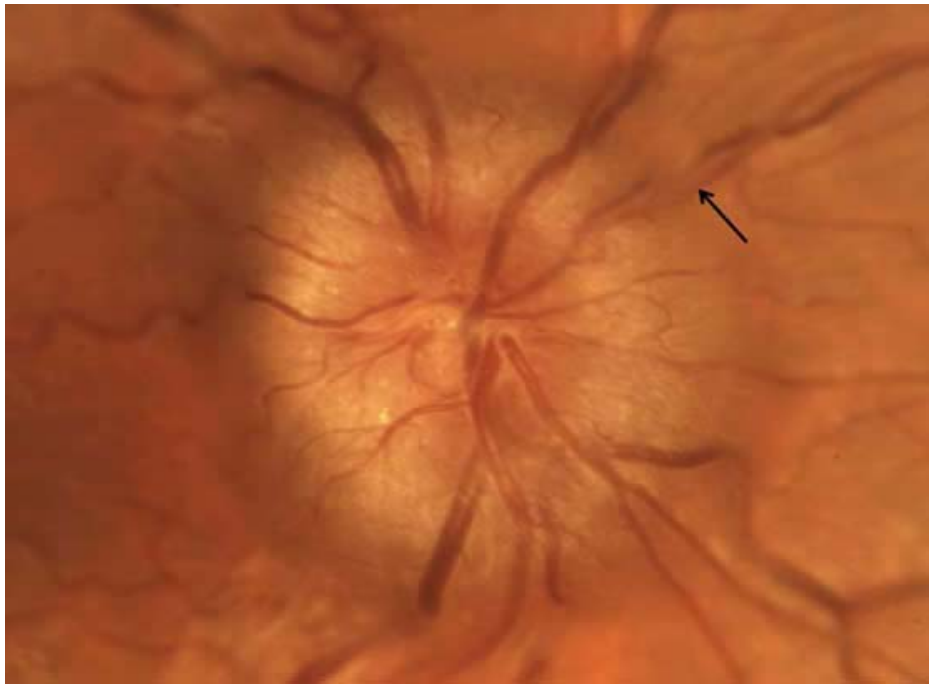
- Grade I papilledema is characterized by a C-shaped halo with a temporal gap



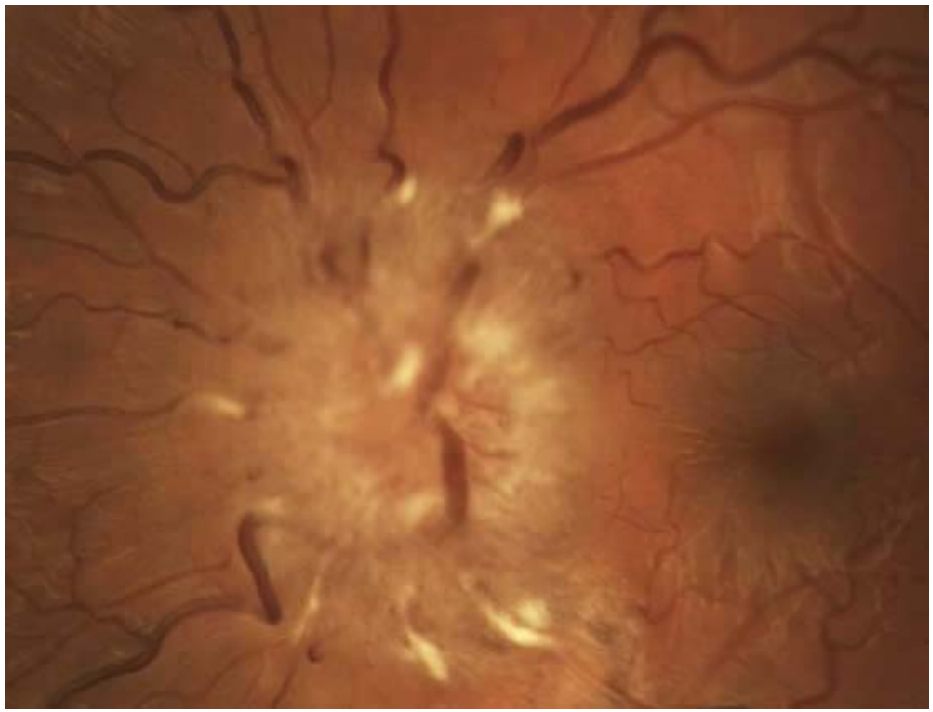
- In Grade II papilledema, the halo becomes circumferential



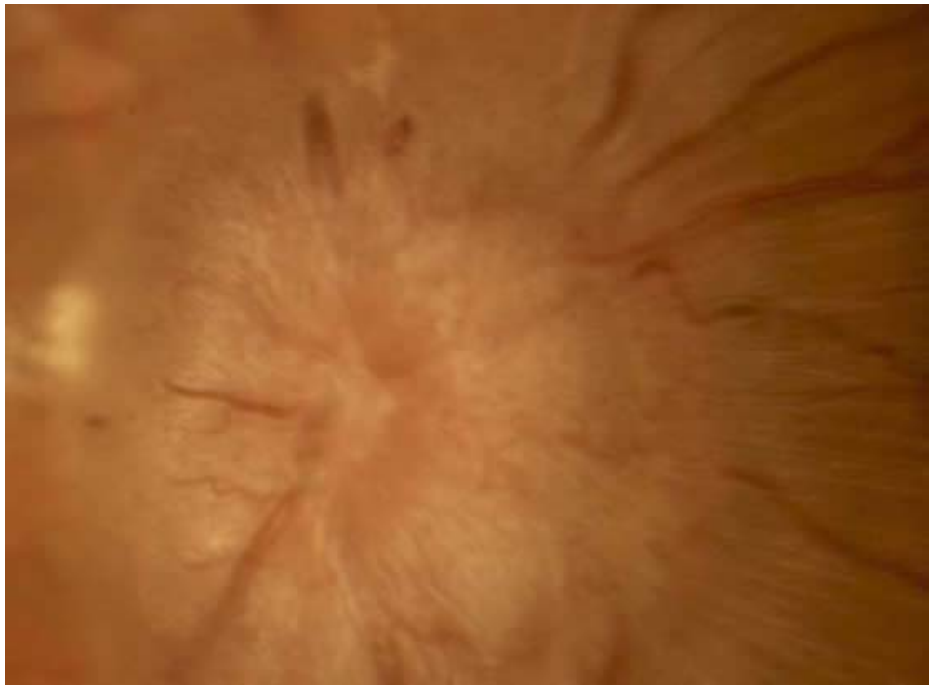
- Grade III papilledema is characterized by loss of major vessels AS THEY LEAVE the disc (arrow)



- Grade IV papilledema is characterized by loss of major vessels ON THE DISC.



- Grade V papilledema has the criteria of grade IV plus partial or total obscuration of all vessels of the disc.



Causes

- Any tumors or space-occupying lesions of the CNS
- Idiopathic intracranial hypertension (also known as pseudotumor cerebri
- Decreased CSF resorption (eg, venous sinus thrombosis, inflammatory processes, meningitis, subarachnoid hemorrhage)
- Increased CSF production (tumors)
- Obstruction of the ventricular system
- Cerebral edema/encephalitis
- Craniosynostosis
- Medications, for example, tetracycline, minocycline, lithium, Accutane, nalidixic acid, and corticosteroids (both use and withdrawal)

Medical treatment

- Diuretics: The carbonic anhydrase inhibitor, acetazolamide (Diamox), may be useful in selected cases, especially cases of idiopathic intracranial hypertension.
- Weight reduction is recommended in cases of idiopathic intracranial hypertension and can be curative.
- Bariatric surgery may be considered in cases refractory to conventional methods of weight loss.
- Corticosteroids may be effective in cases associated with inflammatory disorders (eg, sarcoidosis).
- Consider withdrawing causative medications, as weighed against other medical necessities and alternatives

Surgical treatment

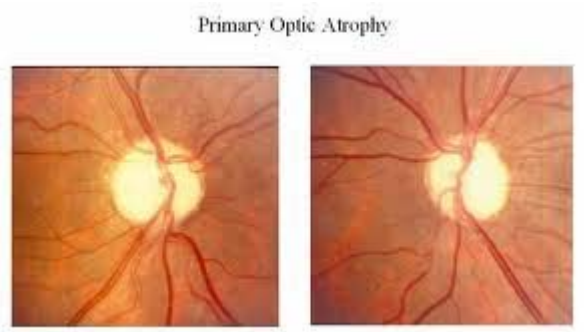
- The underlying mass lesion, if present, should be removed.
- Lumboperitoneal shunt or ventriculoperitoneal shunt can be used to bypass CSF.
- Optic nerve sheath decompression can be used to relieve worsening ocular symptoms in cases of medically uncontrolled idiopathic intracranial hypertension.

Differential diagnosis of papilledema

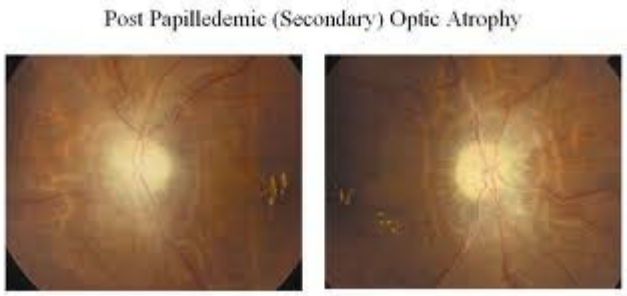
Features	Papilledema	Papillitis	Pseudopapilledema
Laterality	b/l	u/l or b/l	May be u/l
Visual acuity	Transient decrease	Marked loss	Defective based on ref. error
Pain	Absent	May be present with EOM	Absent
Media	Clear	Vitreous haze	Clear
Disc colour	Red juicy	Marked hyperemia	Reddish
Margins	Blurred	Blurred	Not well defined
Swelling	2-6 D	Not more than 3 D	Depends on hypermetropia

Peripapillary edema	Present	Present	Absent
Venous engorgement	More marked	Less marked	Not present
Retinal h’ges	Marked	Not present	Not present
Retinal exudates	More marked	Less marked	Absent
Macula	Macular star	Macular fan	Absent
Fields	Enlarged blind spot	Central scotoma	No defect
FFA	Pool of dye due to leakage	Minimal leakage	No leakage

Optic atrop



- Primary optic atrophy
- Secondary optic atrophy- following any pathological process which produces optic neuritis or papilledema



- Ascending optic atrophy
- Descending optic atrophy

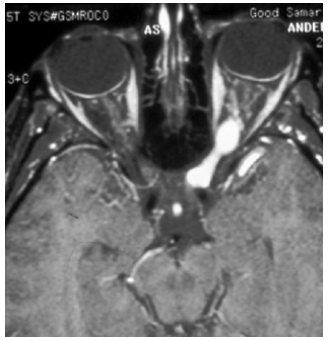
Ophthalmoscopic classification

- Primary/simple optic atrophy
- Consecutive optic atrophy
- Post-neuritic optic atrophy
- Glaucomatous optic atrophy
- Ischemic optic atrophy

Optic nerve tumours

Optic nerve gliomas

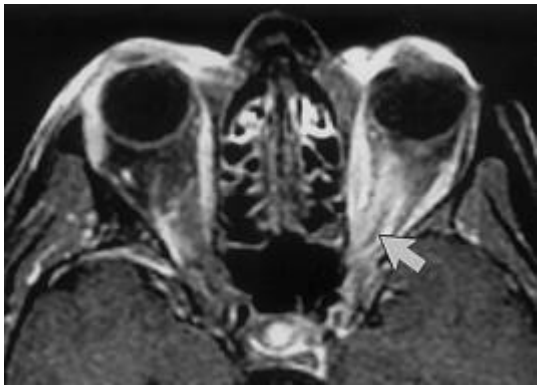
- Optic nerve glioma (also known as optic pathway glioma) is the most common primary neoplasm of the optic nerve.
- Along with reducing visual acuity, it produces unilateral axial proptosis
- Seen in first decade
- Benign tumour of astrocytes



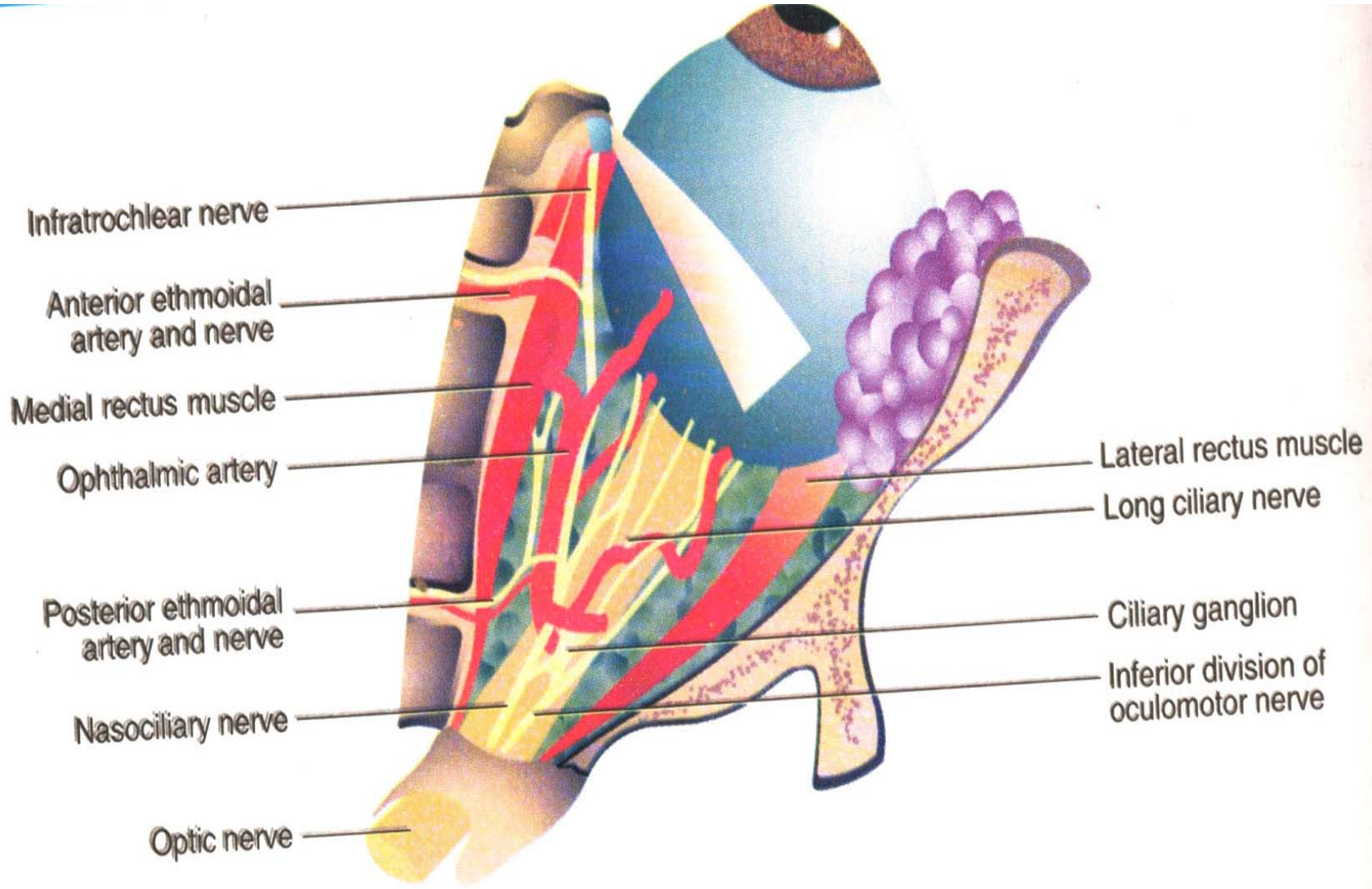
- Fundus shows optic atrophy or papilledema
- Fusiform enlargement of optic nerve on MRI
- Treatment- excision/radiotherapy

Optic nerve sheath meningiomas

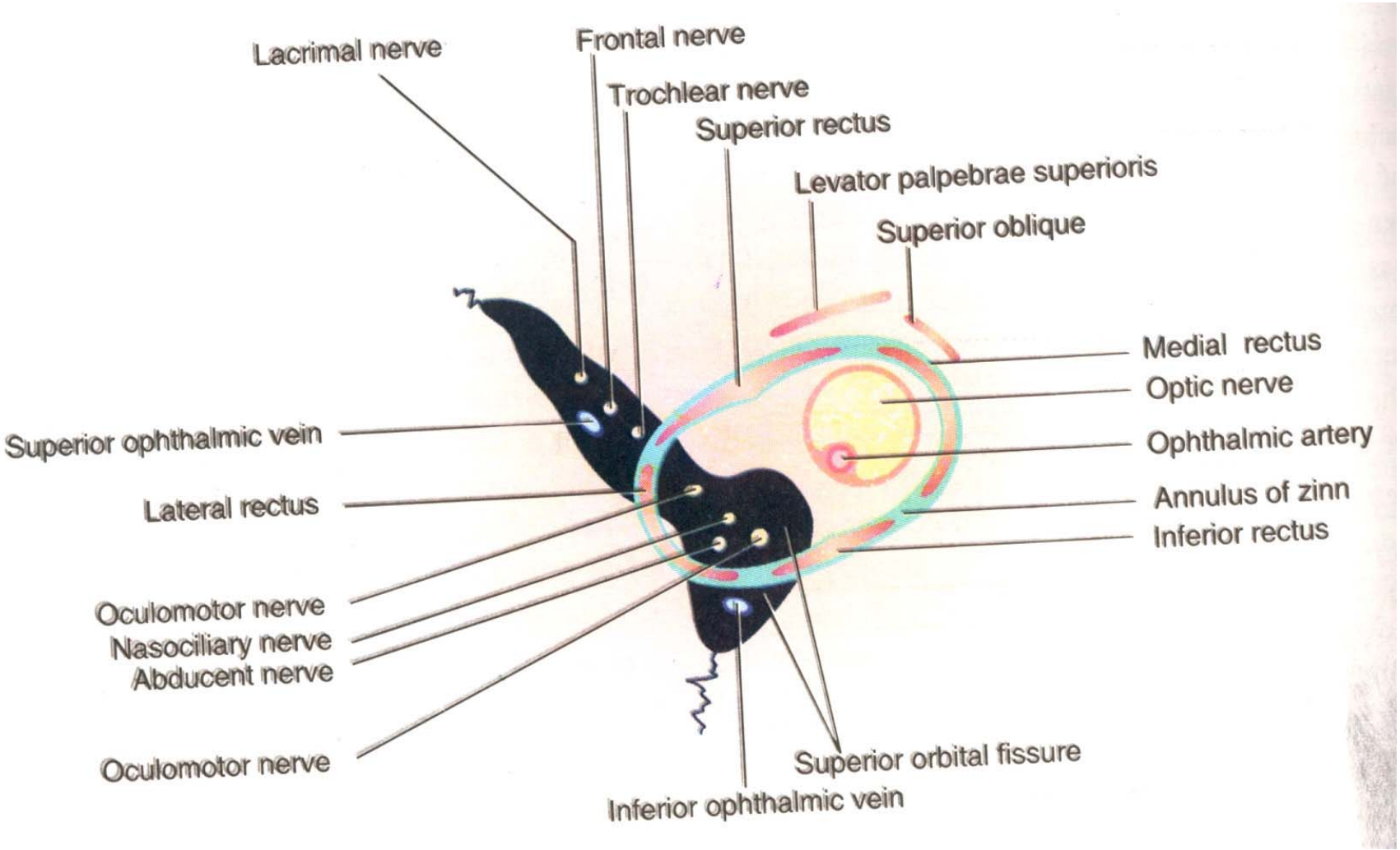
- Visual loss and slowly progressive proptosis
- Fundus shows optic atrophy or papilledema and optociliary shunt vessels




Thank you



Relations of intraorbital part of optic nerve.



Relations of Intracanalicular part of optic nerve

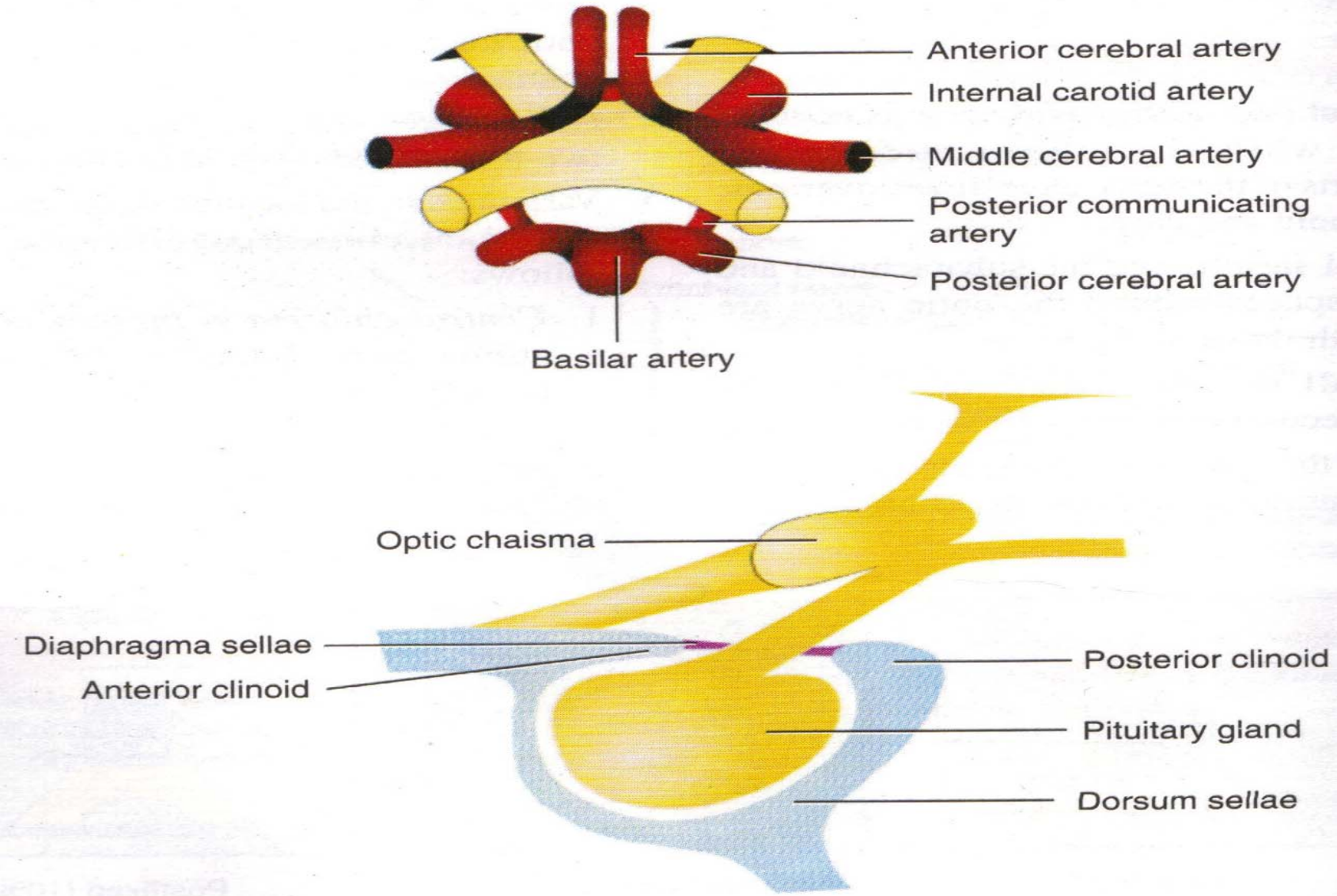


Fig. 6.17. Showing relations of optic chiasma.

Optic Tracts

- These are cylindrical bundles of nerve fibers running outwards and backwards from the poster lateral aspect of optic chiasma.
- Each optic tract consists of fibers from the temporal half of retina of the same eye and nasal half of opposite eye.

Lateral Geniculate Body (Lgb)

These are oval structures situated at the termination of optic tracts.

Visualcortex

It is located on the medial aspect of occipital lobe in and near the calcarine fissure. It may extend on the lateral aspect of the occipital lobe, but limited by a semi lunar sulcus, the sulcus lumatus. The visual cortex is sub divided into visual sensory area (striate area 17) that receives the fibers of the optic radiations and the surrounding visuopsychic area (peristriate area 18 and parastriate area 19)

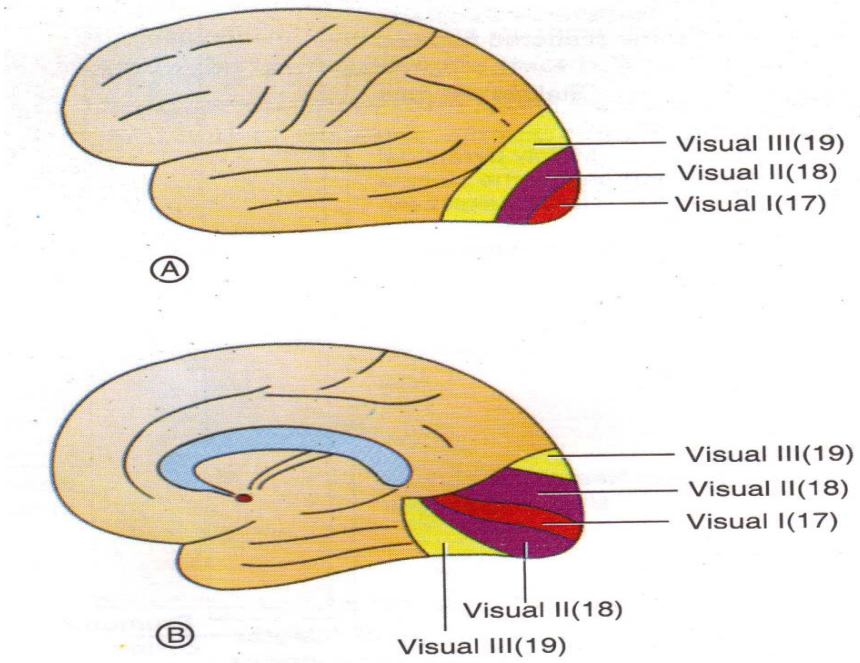


Fig. 6.19. Location of visual cortex on superolateral: A, and medial; B, surfaces of the cerebral hemisphere.

In The Optic Tract

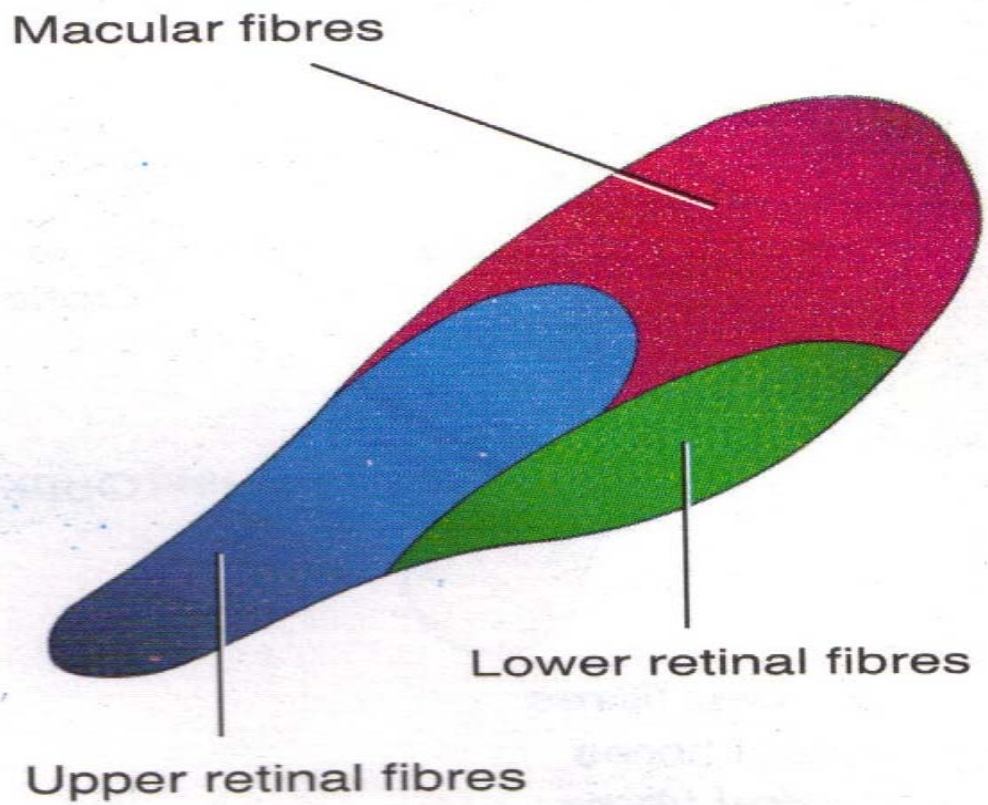
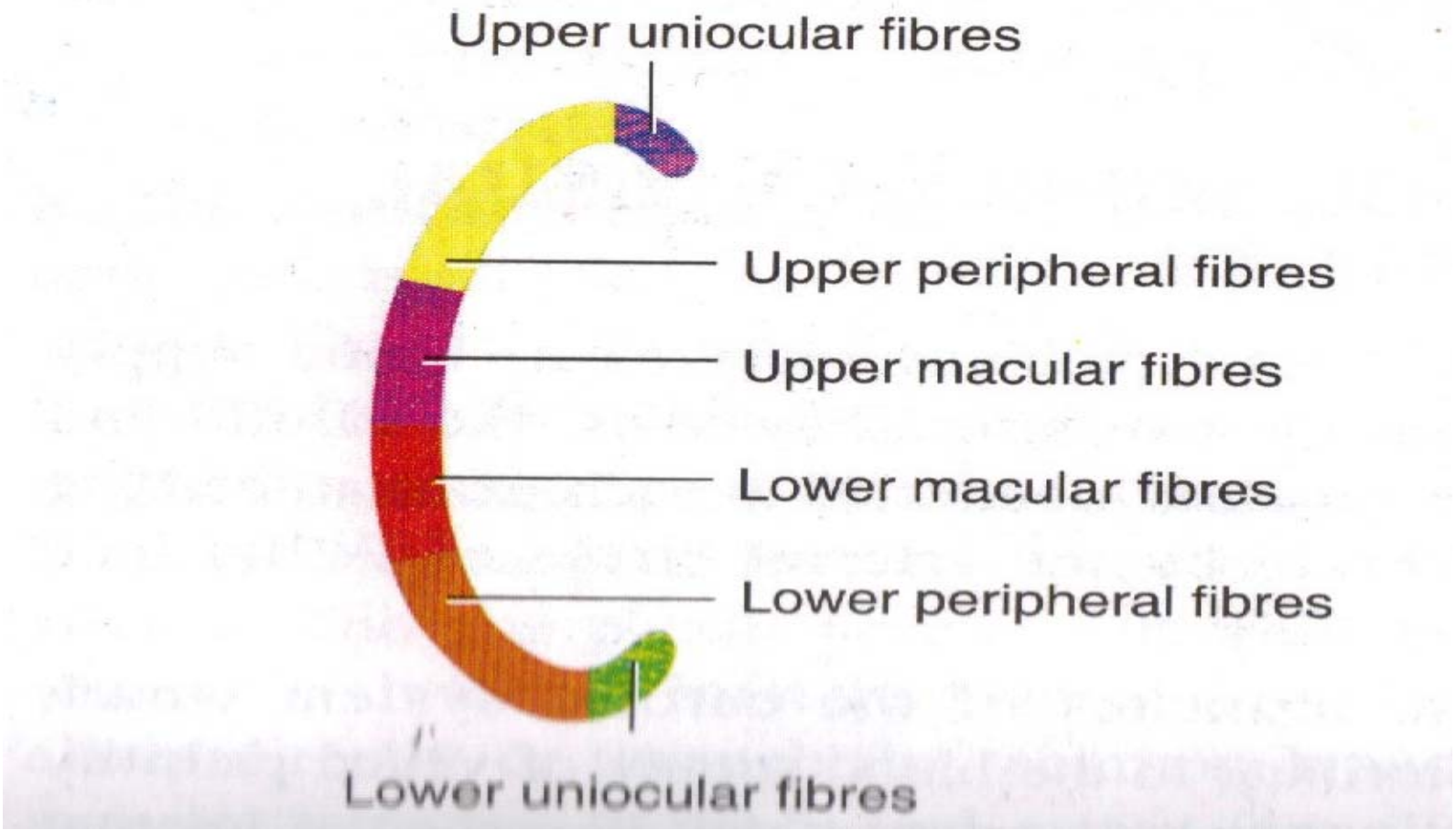


Fig. 6.24. Arrangement of fibres in the optic tract.

In The Optic Radiations



In The Visual Cortex

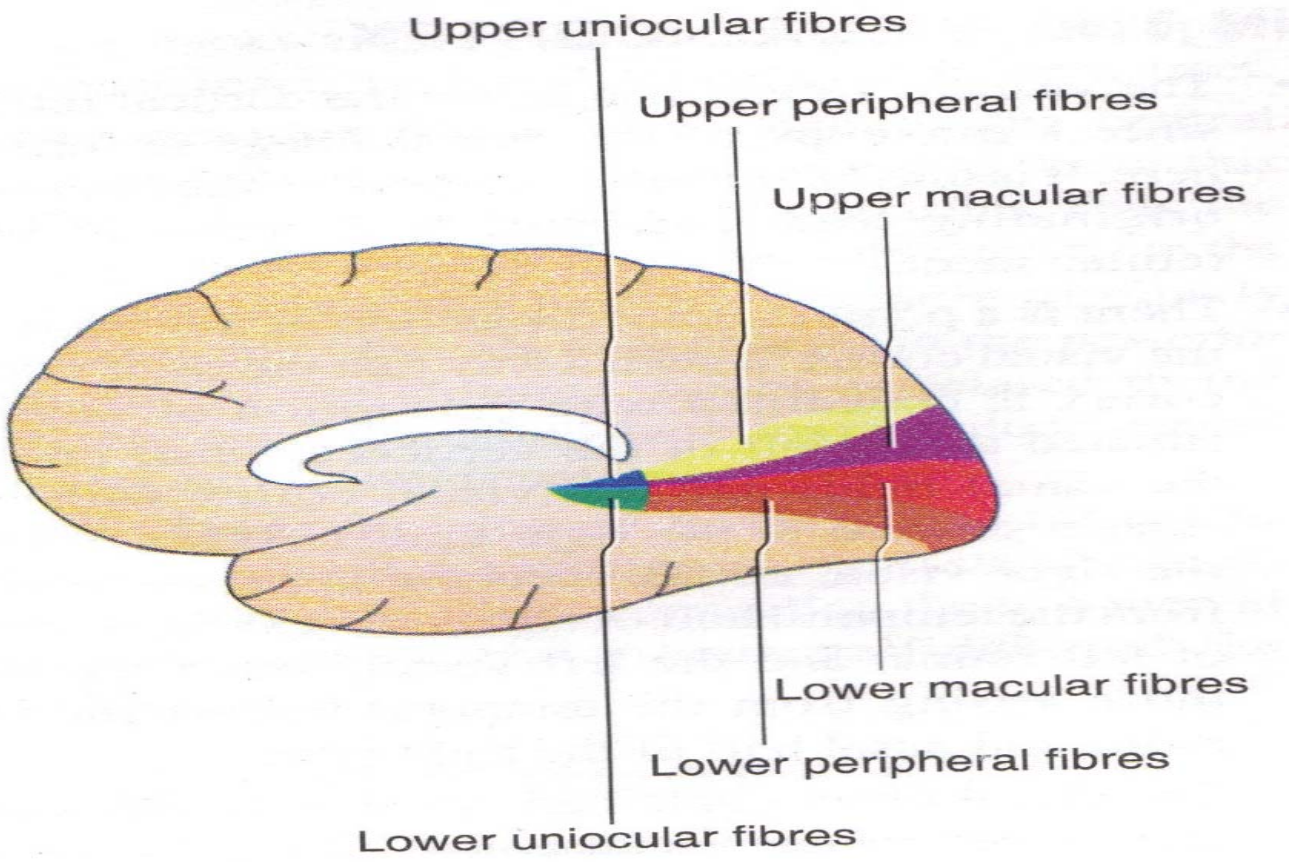
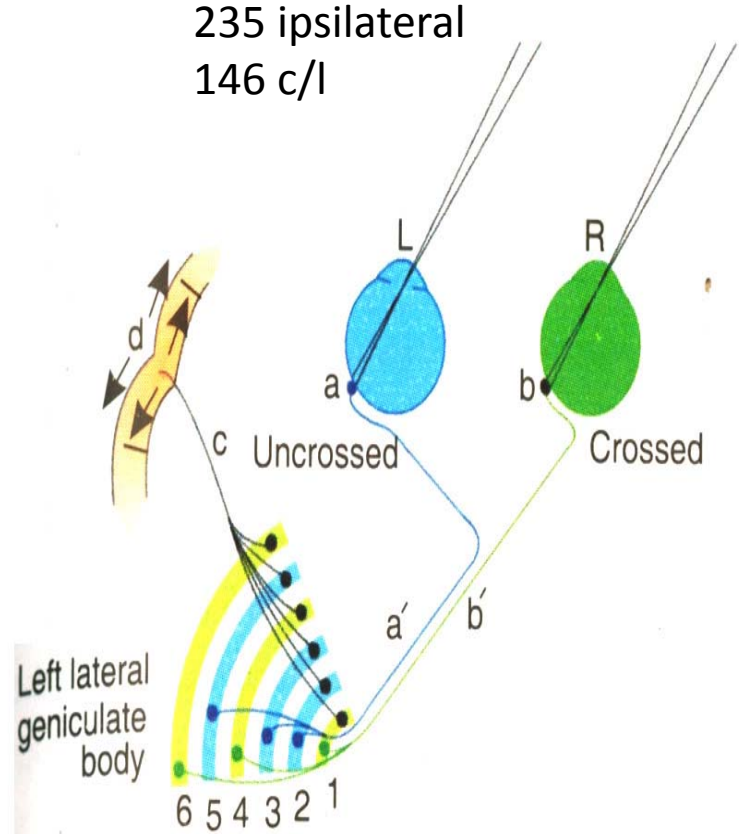


Fig. 6.28. Arrangement of fibres in visual cortex.

In The Lateral Geniculate Body

235 ipsilateral
146 c/l



Arrangement of termination of axons of ganglion cells (second order neurons of vision) of the two eyes in the lateral geniculate body. For explanation see text.

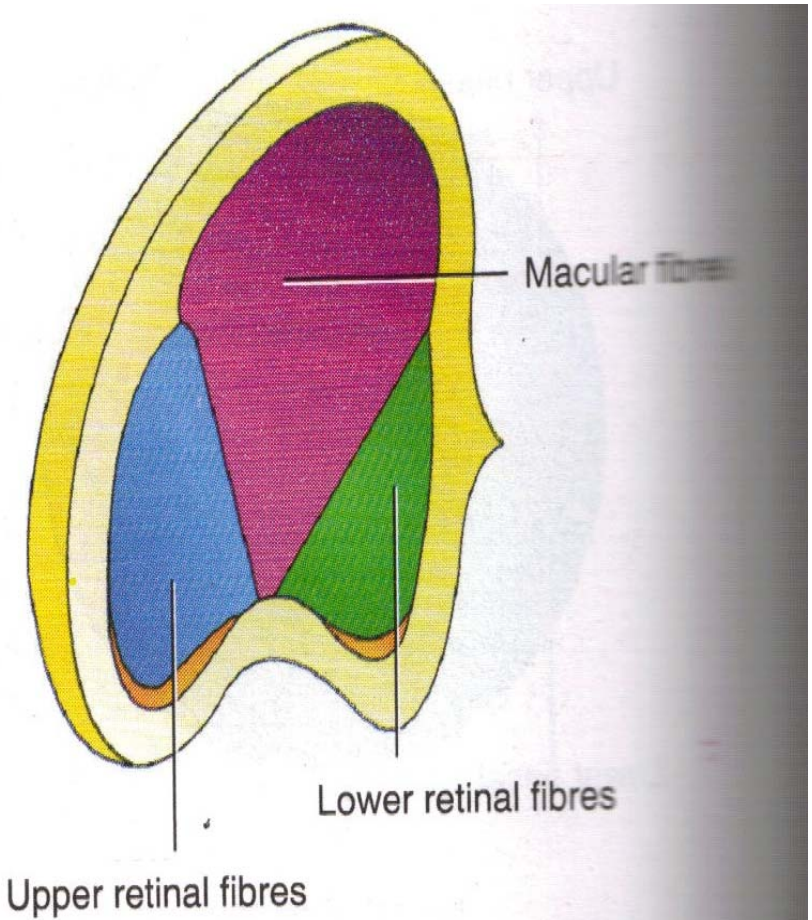
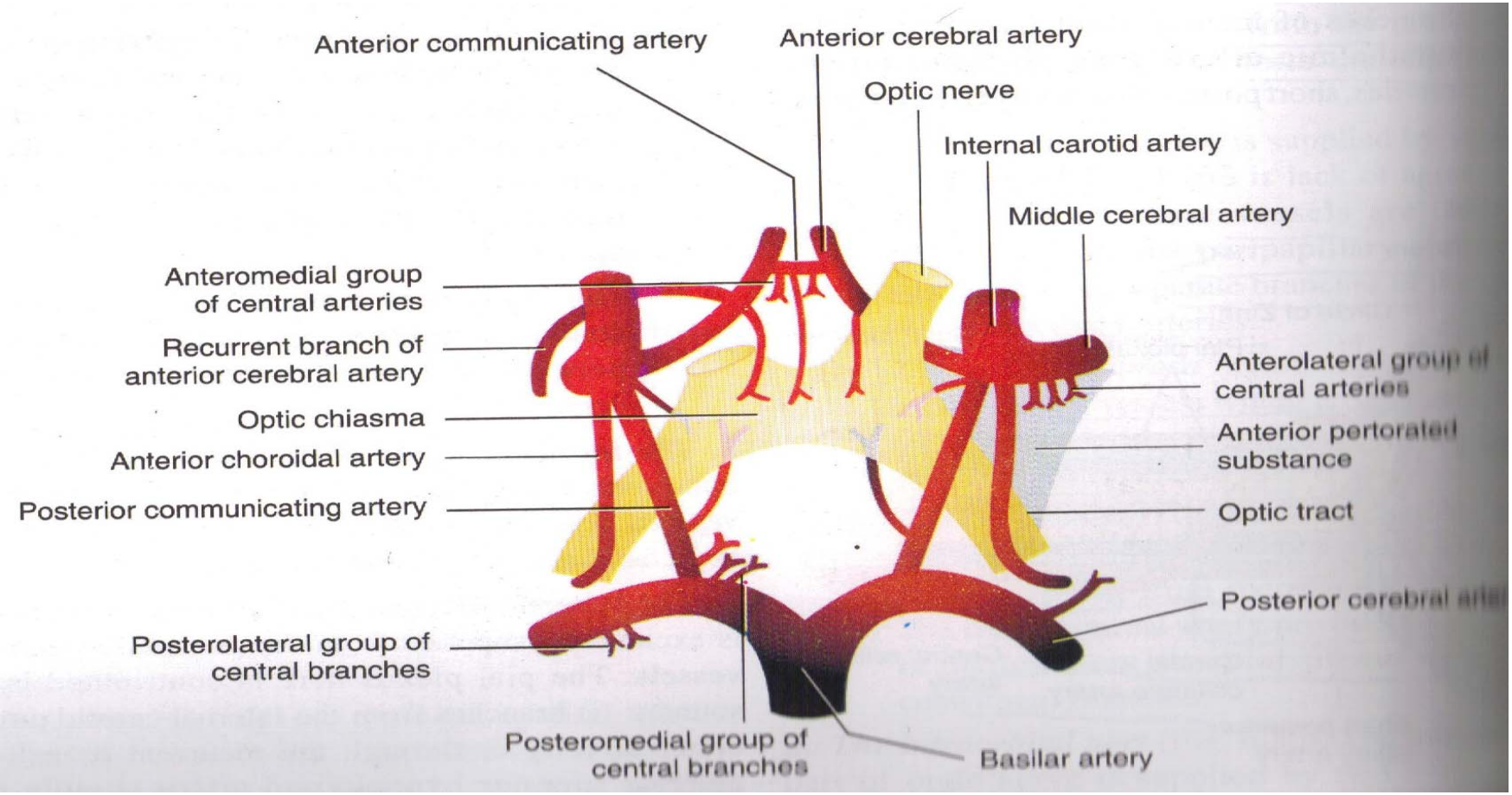


Fig. 6.25. Arrangement of fibres in the lateral geniculate body.

Blood Supply Of Optic Chiasma



Blood Supply Of Optic Tract

Blood Supply Of Lateral Geniculate Body

Blood Supply Of Optic Radiations

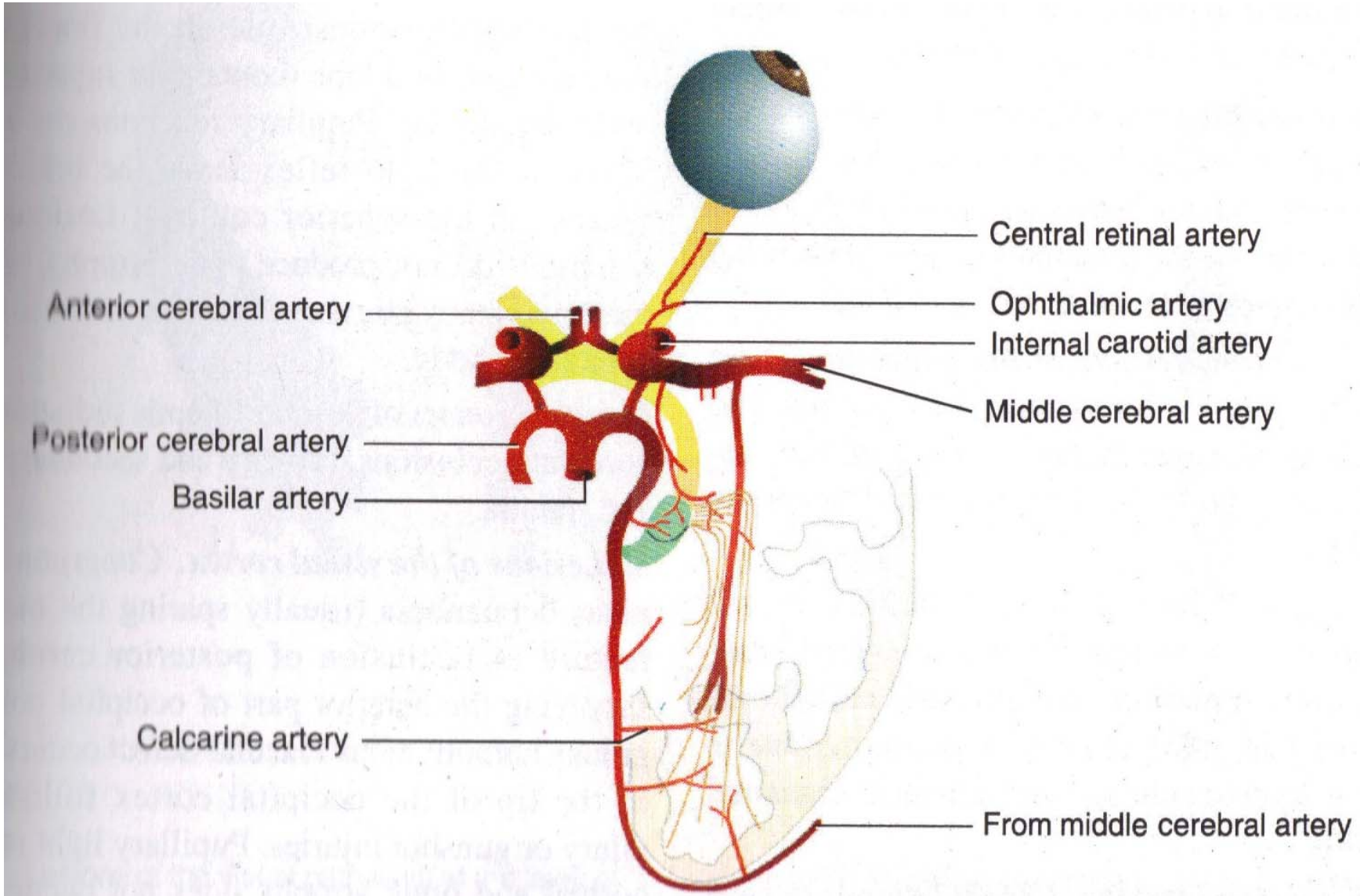
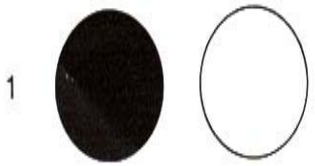


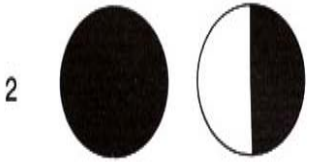
Fig. 6.32A. Blood supply of posterior visual pathway.

1. **LESIONS OF THE OPTIC NERVE**



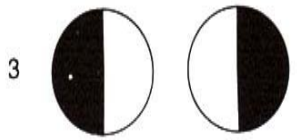
These are characterized by marked loss of vision or complete blindness on the affected side associated with abolition of the direct light reflex on the ipsilateral side and consensual on the contralateral side. Near (accommodation) reflex is present.
Common Causes of optic nerve lesions are : optic atrophy, traumatic avulsion of the optic nerve, indirect optic neuropathy and acute optic neuritis.

2. LESIONS THROUGH PROXIMAL PART OF THE OPTIC NERVE



Salient features of such lesions are : Ipsilateral blindness, contralateral hemianopia and abolition of direct light reflex on the affected side and consensual on the contralateral side. Near reflex is intact.

3. SAGITTAL (CENTRAL) LESIONS OF THE CHIASMA

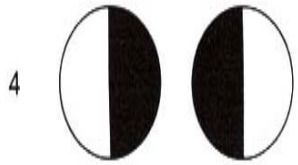


These are characterised by bitemporal hemianopia and bitemporal hemianopia and bitemporal hemianopic paralysis of pupillary reflexes. These usually lead to partial descending optic atrophy.

COMMON CAUSES of central chiasmal lesions are : suprasellar aneurysms, tumours of pituitary gland, craniopharyngioma, suprasellar meningioma and glioma of the third ventricle; third ventricular dilatation due to obstructive hydrocephalus and chronic chiasmal arachnoiditis.

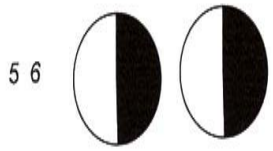
4. LATERAL CHIASMAL LESIONS. Salient features of such lesions are binasal hemianopia , associated with binasal hemianopic paralysis of pupillary reflexes. These usually lead to partial descending optic atrophy.

Common Causes of such lesions are distension of third ventricle causing pressure on each side of the chiasma and atheroma of the carotids or posterior communicating arteries.



5. LESIONS OF THE OPTIC TRACT

These are characterized by incongruous homonymous hemianopia associated with contralateral hemianopic pupillary reaction (WERNICKE'S REACTION) . These lesions usually lead partial descending optic atrophy and may be associated with contralateral third nerve paralysis and ipsilateral hemiplegia.
COMMON CAUSES of optic tract lesions are syphilitic meningitis or gumma, tuberculosis and tumours of optic thalamus and aneurysms of superior cerebellar or posterior cerebral arteries.

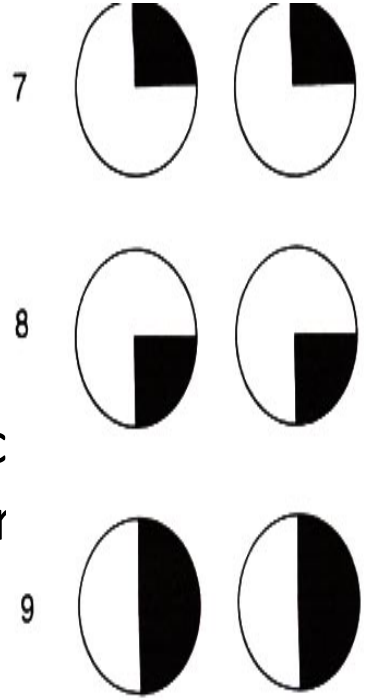


6. LESIONS OF LATERAL GENICULATE BODIES.

These produce homonymous hemianopia with sparing of pupillary reflexes, and may end in partial optic atrophy.

LESIONS OF OPTIC RADIATION

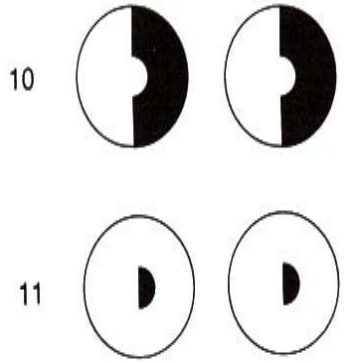
Their features vary depending on the site of lesion. Involvement of total optic radiations produces complete homonymous hemianopia (sometimes sparing the macula). Inferior quandratic hemianopia (pie on the floor) occurs in lesions of parietal lobe (containing superior fibers of optic radiations). Pupillary reactions are normal as fibers of the light reflex leave the optic tracts to synapse in the superior colliculi. Lesions of optic radiations do not produce optic atrophy, as the first order neurons (optic nerve fibers) synapse in the lateral geniculate body.

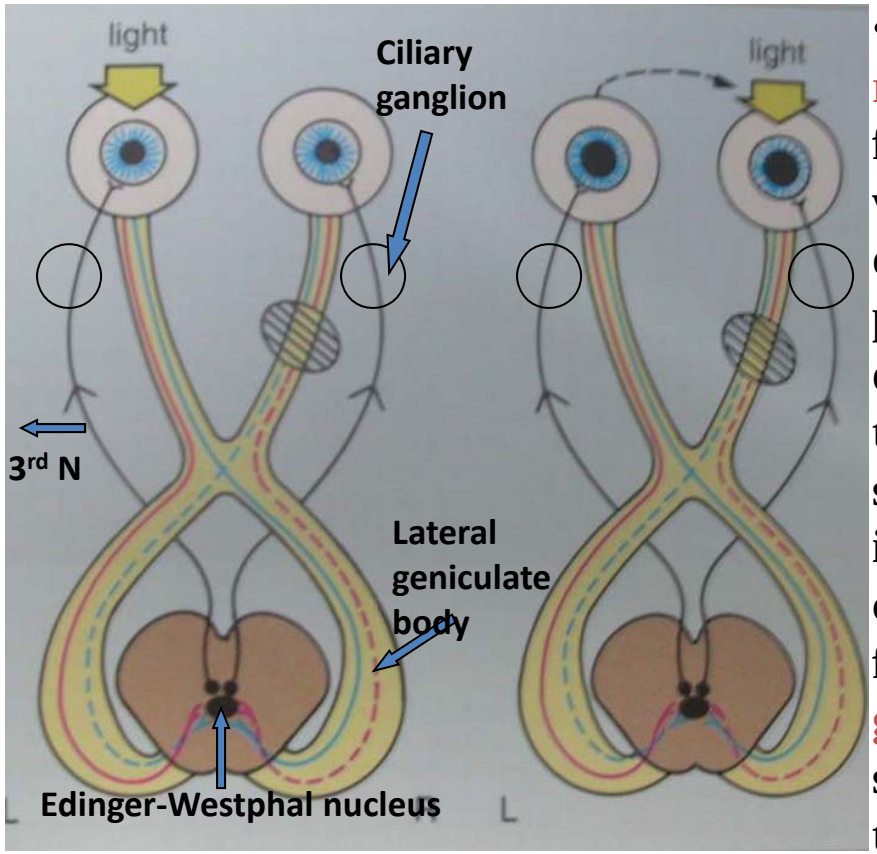


Common causes of lesions of optic radiations include vascular occulusions, primary and secondary tumours and trauma.

LESIONS OF THE VISUAL CORTEX

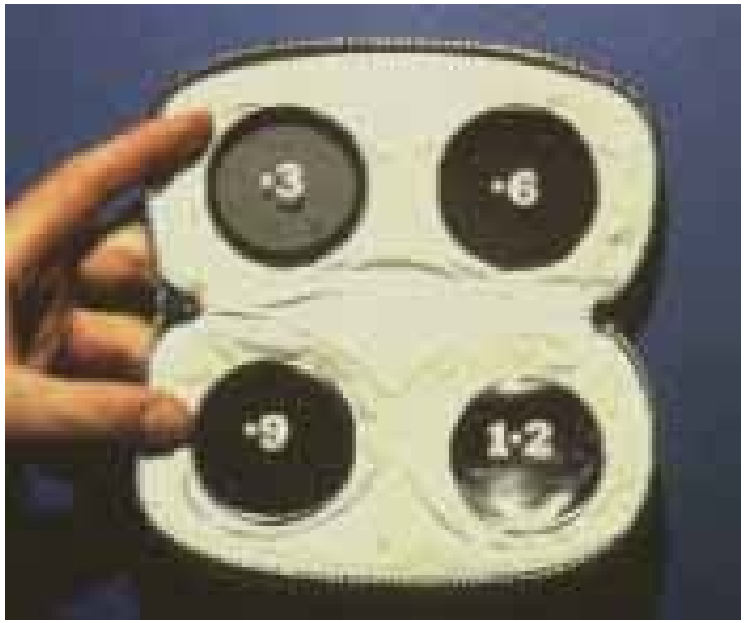
Congruous homonymous hemianpia (usually sparing the macula) is a feature of occlusion of posterior cerebral artery supplying the anterior part of occipital cortex. Congruous homonymous macular defect occurs in lesions of the tip of the occipital cortex following head injury or gun shot injuries. Pupillary light reflexes are normal and optic atrophy does not occur following visual cortex lesions.





•**The afferent pupillary light reflexes are mediated thro' axons from ganglion cells in the retina which pass back in the ON & decussate in the chiasm .The pupillary fibres pass thro' the optic tract to the EW nucleus ,here they synapse to produce a simultaneous & bilateral response in each 3rd N thro interneuronal connections.Efferent PS axons run forward & pass into the ciliary ganglion where they synapse to supply the constrictor pupillae by the short ciliary nerves**

They come in 0.3, 0.6, 0.9 and 1.2 log units of transmission density, each 0.3 reducing the light by half.



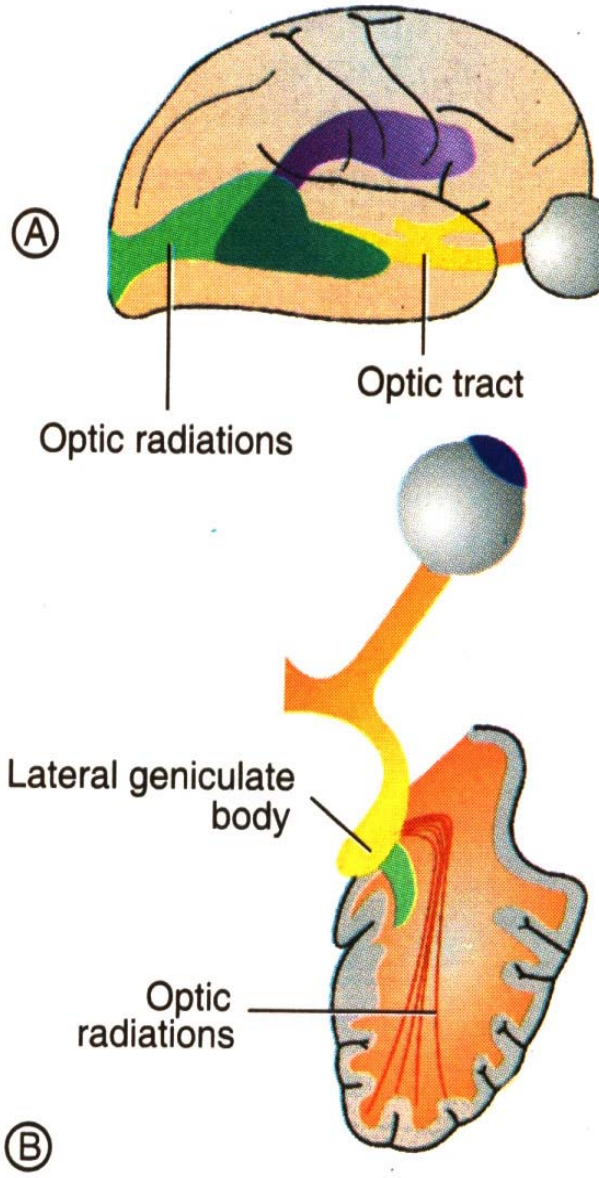
This filter is placed in front of the normal eye, *not the bad eye*. We start with 0.3 log unit filter in front of the normal eye. If on doing the test, the pupil in the bad eye still dilates, then we go to the next filter, 0.6 log unit. If it still dilates we go to the next filter, 0.9 log unit, and after that to the 1.2 log unit filter. (In fact, we can combine these filters to get higher log units.) So we keep doing that until the pupil in the bad eye starts to constrict instead of immediately dilating. That gives us the degree of the relative afferent pupillary defect

Optic Radiations

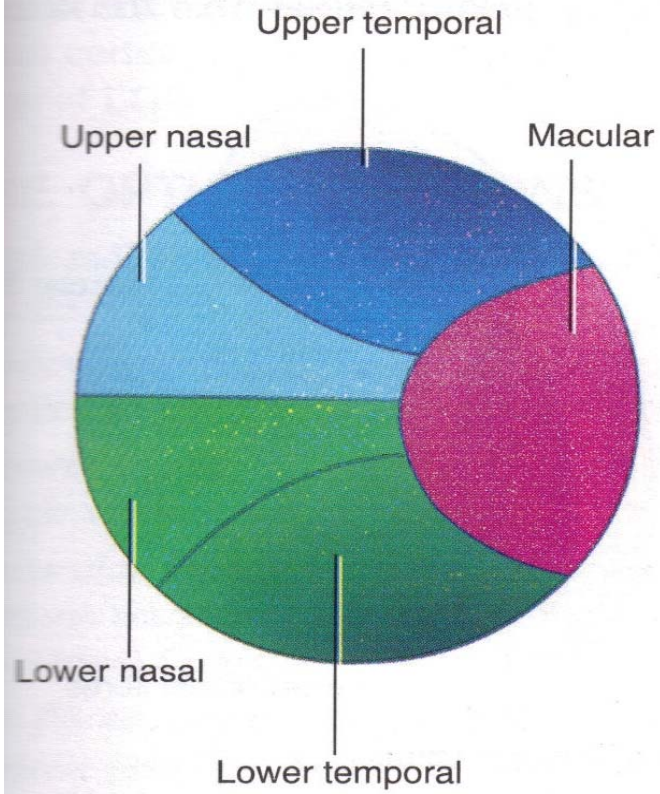
The Optic radiations or geniculocalcarine pathway extend from the LGV two visual cortex. They pass forwards and than laterally through there area of Wernicke as optic peduncles, anterior to lateral ventricle and traversing the retrolenticular part of internal capsule, behind the sensory fibers and medial to auditory tract. The fibers of optic radiations then spread out fanwise to form a medullary optic lamina. This is at first vertical but becomes horizontal near the visual cortex.

The inferior fibers of the optic radiations which sub serve the upper visual field, first sweep antero inferiorly in MEYER'S LOOP around the anterior tip of the temporal horn of the lateral ventricle, and into the temporal lobe.

The superior fibers of the radiations which sub serve the inferior visual fields, proceed directly posteriorly through the parietal lobe to the visual cortex.



Optic radiations: A, lateral view; B, transverse section.



. Arrangement of fibres in the distal region (behind the eyeball) of optic nerve.

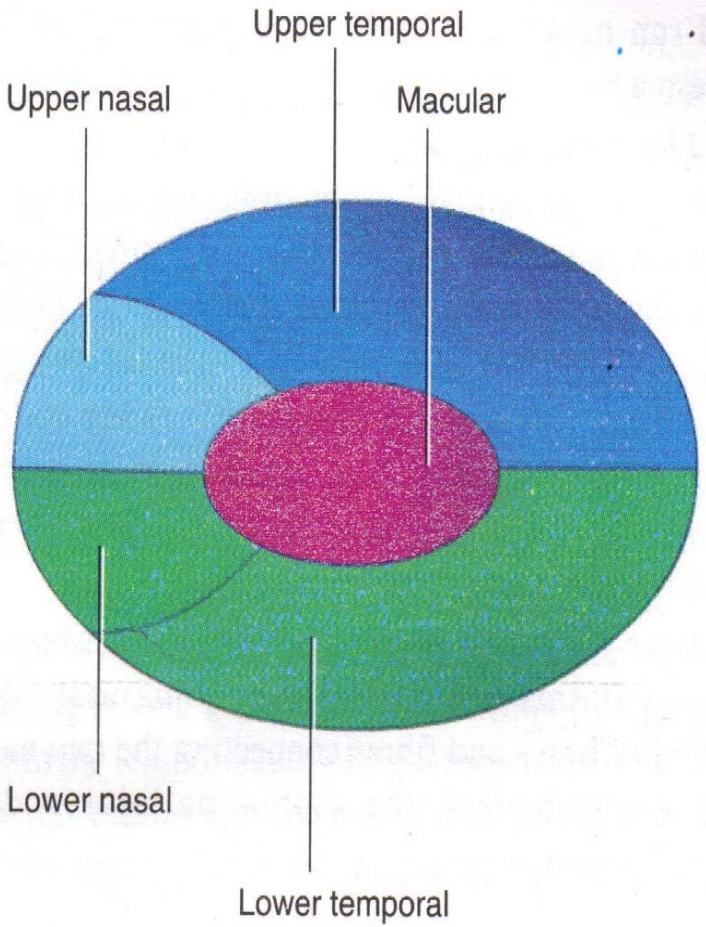


Fig. 6.22. Arrangement of fibres in the proximal region of optic nerve. Note central position of papillomacular bundle.

Blood Supply Of Visual Pathway

carotid and the vertebral

carotid system

vertebral systems

pial network

except the orbital part of optic nerve which is also supplied by an axial system derived from the central retinal artery.