VISION LOSS AFTER STROKE

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Nothing to disclose

FINANCIAL DISCLOSURE

- None

AIM

- Identify common post stroke vision changes.
- Explain the anticipated effects of vision changes after stroke.

INTRODUCTION

- Cerebrovascular accidents (CVAs) occur from decreased cerebral perfusion leading to neuronal death.
- 3 types of strokes:

A Ischemic strokes : 85% CVAs;

Thrombotic, with blood clots forming locally in the artery

Embolic, where blood clots formed elsewhere, (heart, aorta, carotid arteries, or in the venous system with arterial crossover (paradoxical strokes)

Cerebral hypoperfusion from systemic shock

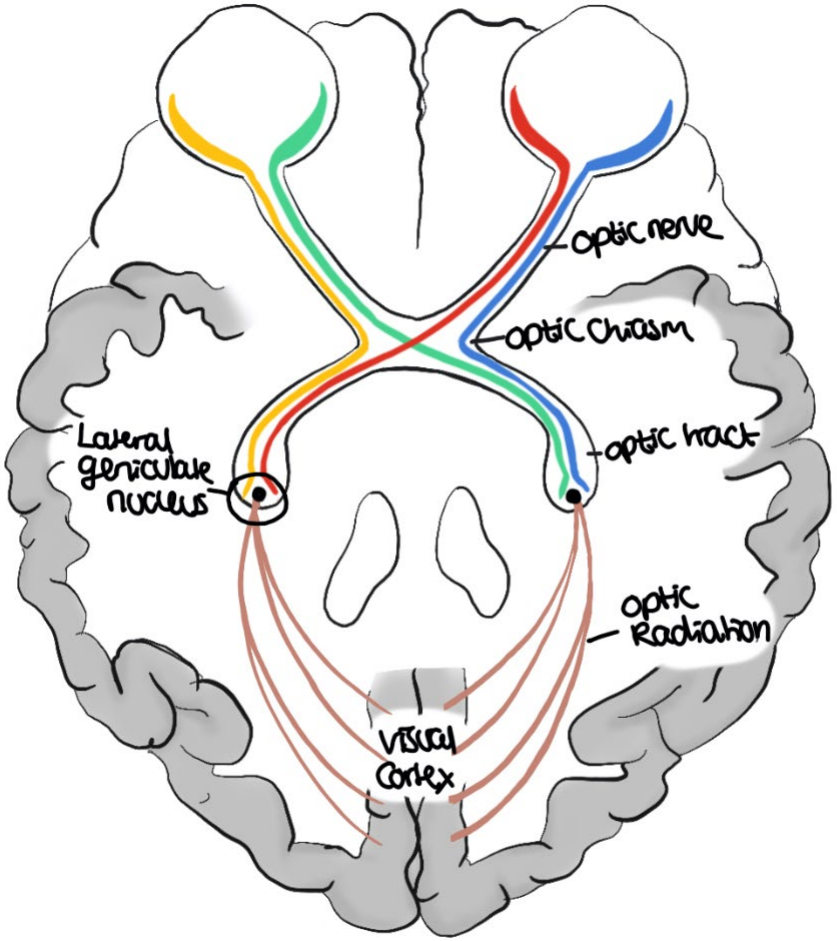
B. Hemorrhagic stroke : intraparenchymal or subarachnoid hemorrhage.

C Transient ischemic attacks (TIAs) : transient episodes of neurologic dysfunction caused by focal brain ischemia without acute infarction.

INTRODUCTION

- A large portion of CNS is dedicated to visual pathways.
- Strokes have a high likelihood of involving vision.
- 8 to 25% stroke patients develop visual field loss.
- Post stroke visual loss impairs quality of life, affects independence and causes depression.
- Recovery of visual function is variable.
- No standard therapeutic interventions exist.

VISUAL PATHWAY



The efferent visual pathways are the motor nerve fibers that carry signals *away* from the central nervous system(brain stem and higher) to control eye and eyelid movements and pupillary responses.

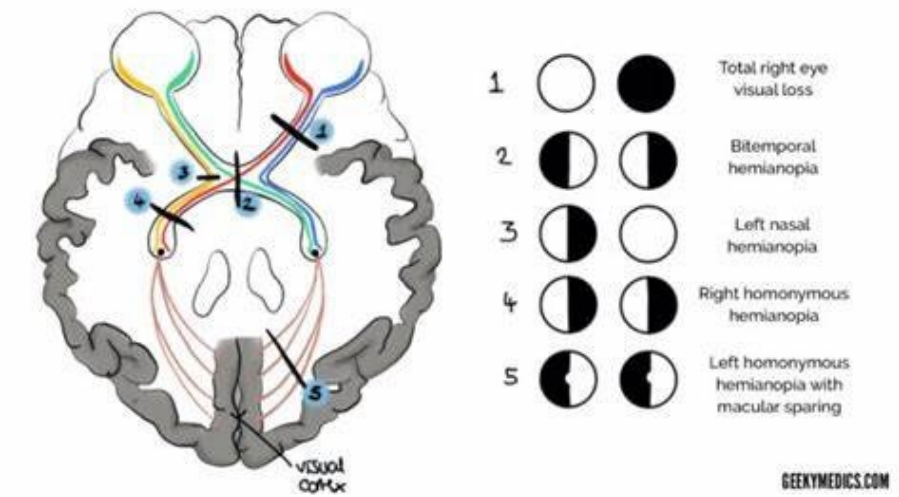
TYPES OF VISUAL IMPAIRMENT

- The afferent or efferent pathway may be affected.

Afferent visual pathway impairment produces

A. transient or permanent visual loss

B. Visual field loss



EFFERENT VISUAL PATHWAY INVOLVEMENT

Symptom : diplopia

- Ptosis
- Internuclear ophthalmoplegia (INO)
- One-and-a-half syndrome
- Gaze palsies
- Saccadic intrusions
- Impaired smooth pursuits
- Nystagmus

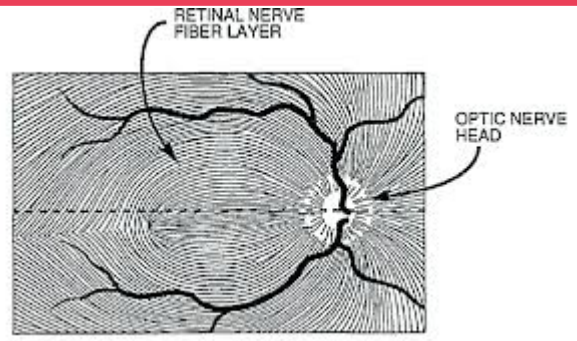
BLOOD SUPPLY

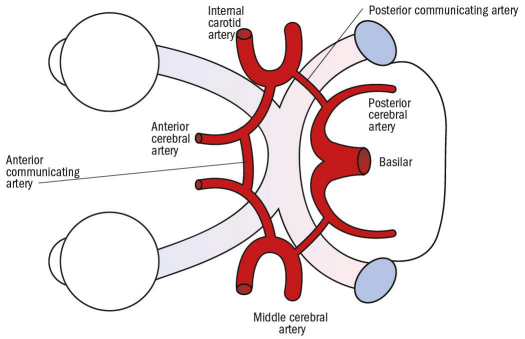
- Prechiasmal pathway- axons of nerve fiber layer of retina forming optic nerves till chiasm

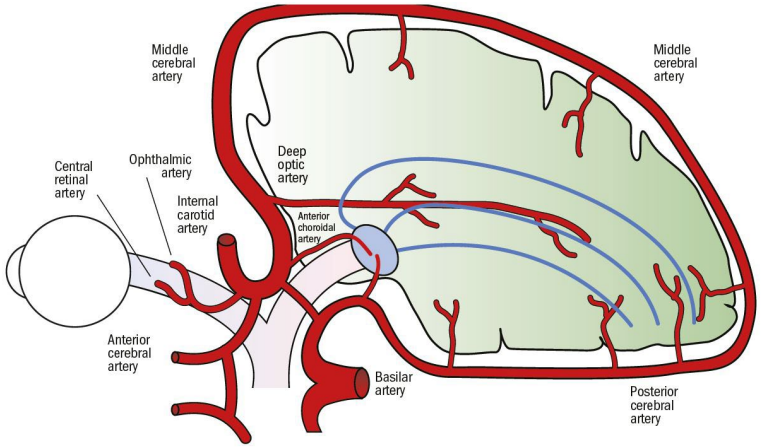
➢Central retinal artery supplies retina

➢Ophthalmic artery and pial vessels of ICA supply prechiasmatic optic nerve

- Circle of Willis : optic chiasm





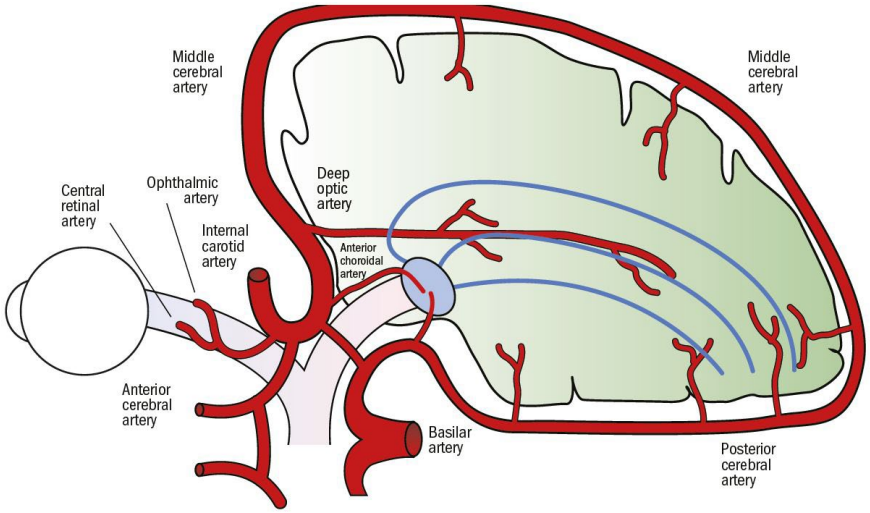


HealthCare NORTON HEALTHCARE

STROKE CARE NETWORK

BLOOD SUPPLY

- Retrochiasmal pathway
- ➢ Optic tract to LGB: anterior choroidal artery (branch of ICA)
- ➢ Lat geniculate body: Ant choroidal artery and Lat post choroidal artery (branch of PCA) – terminal anastomosis, vulnerable to ischemia
- ➢ Optic radiations : posterior/ middle cerebral artery and ant choroidal artery
- ➢ Occipital cortex : largely by PCA(terminal branches of basilar artery)



VISUAL SYMPTOMS

- In a review [Sand KM et al]
  - ➢75% had normal vision
  - ➢8.9% had difficulty watching TV or reading
  - ➢0.6% were almost blind
- Large prospective UK study found reading problems in 19.3% patients, only a minority had decreased acuity(Rowe F et al. Int J Stroke 2011; 6:404–411).

REDUCED VISUAL ACUITY

70% will develop acutely decreased visual acuity after a stroke, multifactorial cause.

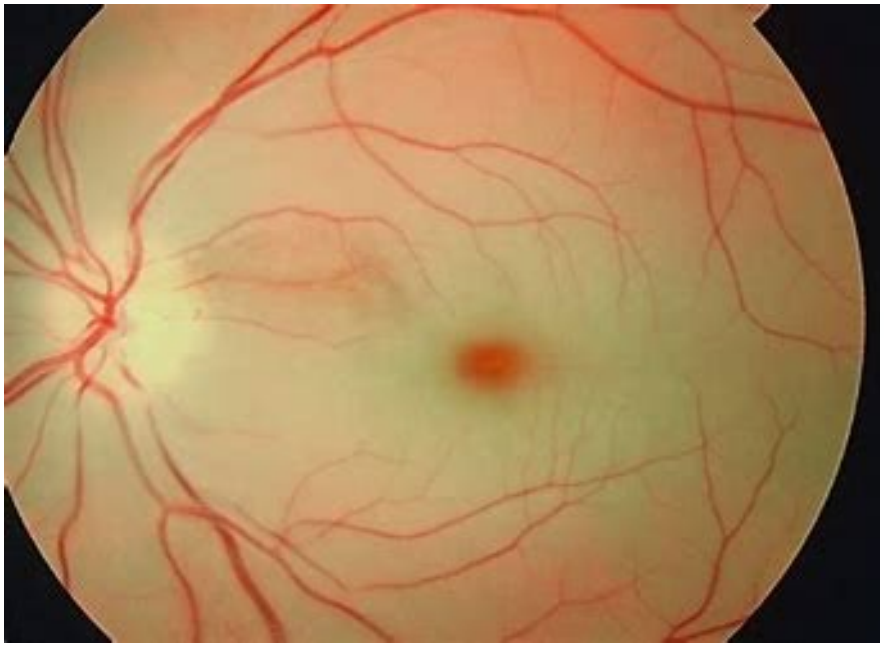
- reduced contrast sensitivity (60% strokes affecting parietal, temporal and occipital cortices)
- perception defects
- convergence insufficiency
- visual field loss
- reduced depth perception
- dry eye from poor blinking or facial nerve palsies
- cortical blindness: bilateral occipital lobe infarcts (rare)
- Vision loss from dirty, lost, or broken glasses

(Rowe F; Strabismus 2013; 21:150–154)

MONOCULAR VISION LOSS

- Pre-chiasmal.
- Transient retinal ischemia – amaurosis fugax.
- Secondary to occlusion of ophthalmic artery or its branches .
- Retinal ischemia is included in the definition of TIA, according to American Heart Association/American Stroke Association guidelines.
- Transient monocular vision loss can precede stroke.
- 93% TIA present with TMVL, ischemic.

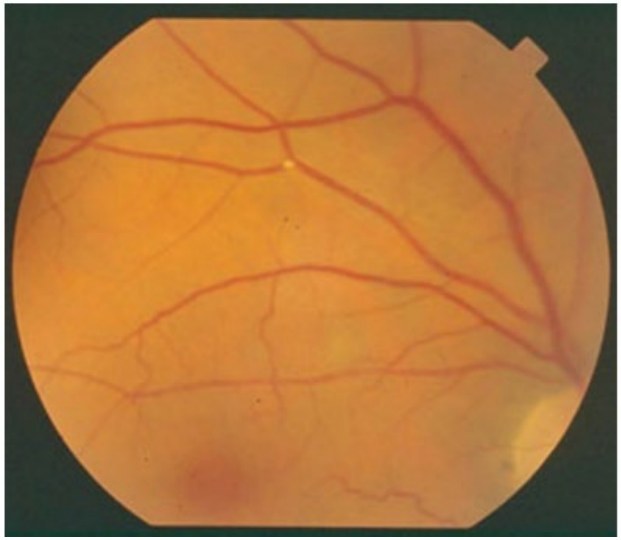
PERMANENT RETINAL ISCHEMIA



- Branch retinal artery occlusion, Central retinal artery occlusion or ophthalmic artery occlusion(rare).
- Permanent visual loss, loss of visual acuity and visual field defects.
- Acute stroke occurred in 23% of 213 patients presenting with CRAO or BRAO
- Vision loss was isolated presenting symptom in 90% (Lauda et al).

HOLLENHORST PLAQUE

- A Hollenhorst plaque -cholesterol plaque appearing yellow or white in the lumen of a retinal artery.
- Denotes a thromboembolic predisposition.
- Visual field defect or may be asymptomatic.
- Beaver Dam Eye Study (BDES), among 4926 patients showed 5-year incidence of retinal emboli is 0.9% and prevalence is 1.3.
- Asymptomatic patients with retinal emboli experienced an increased risk of stroke-related death (Hazard Ratio (HR)=2.61, 95% Confidence Interval (CI) 1.12 to 6.08) compared to those without retinal emboli.
- Work up recommended for vascular disease, increased future risk of stroke.

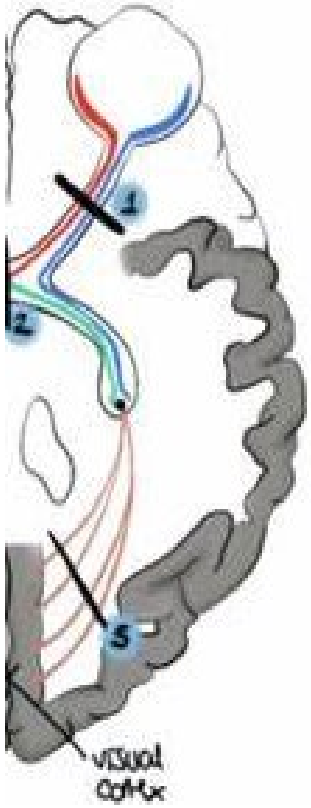


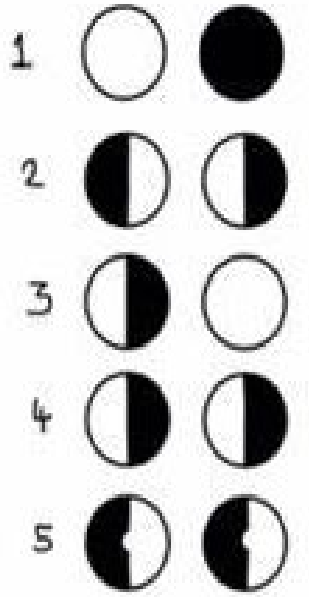
TRANSIENT BINOCULAR VISUAL LOSS

- Transient binocular visual loss (TBVL) – not prechiasmal, but vertebro-basilar ischemia.
- TBVL produces homonymous visual field loss and a warning sign of stroke.

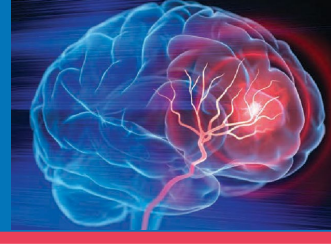
APPROACH TO VISUAL FIELD DEFECTS

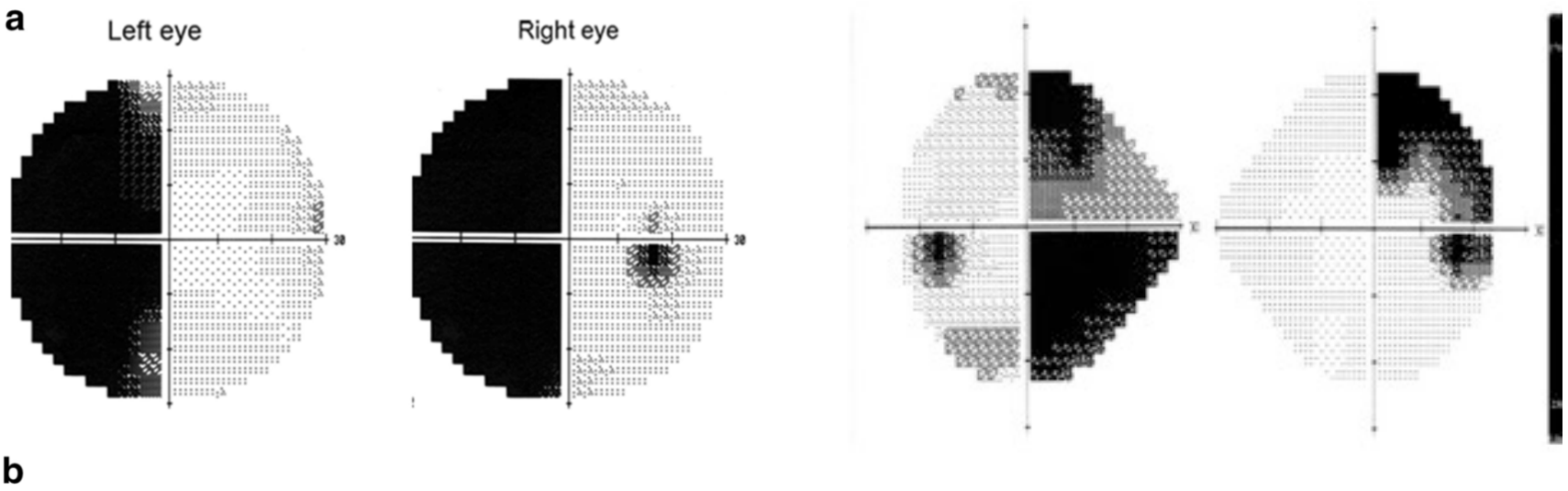
- Does the field defect involve one eye or two?
- Does it respect the vertical meridian?
- If both eyes are involved and it respects the vertical meridian, are the defects on the same side of the vertical meridian in each eye (homonymous) or on opposite sides (heteronymous).





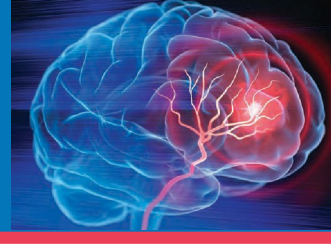
APPROACH TO VISUAL FIELD DEFECTS

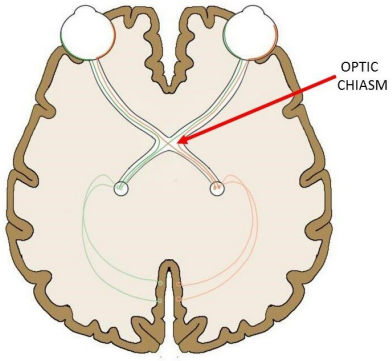




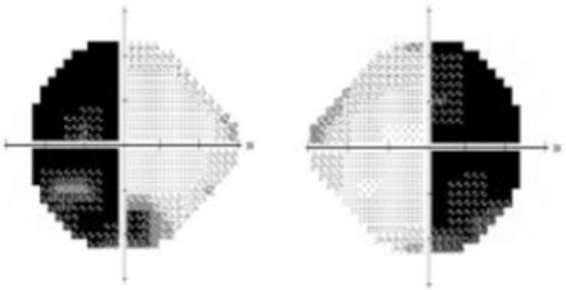
- If homonymous, is it congruous (similar/equal) or incongruous (unequal).
- Incongruous : more anterior: optic tract , LGB.
- Congruous: more posterior: occipital lobes 54%, optic radiations 35%.

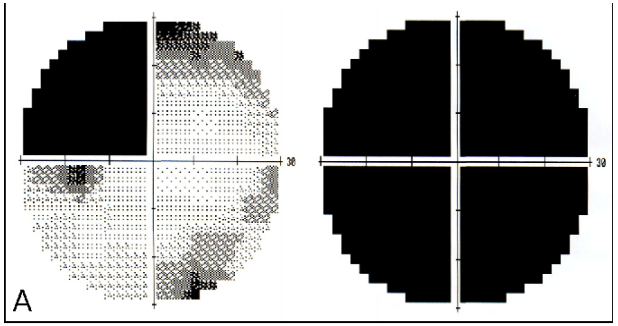
CHIASMAL ISCHEMIA





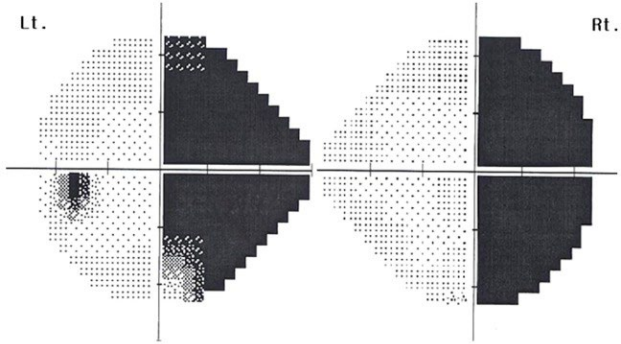
Humphrey visual field showing bitemporal hemianopia.





- Rare, due to rich collateral provided by the Circle of Willis(Fabian et al)
- Chiasmal strokes : acute bitemporal hemianopia.
- Atypical presentations : sup temporal defect in one eye with contralateral complete monocular vision loss, the so-called junctional scotoma- an anterior chiasmal infarction.

POST CHIASMAL STROKE



- Ischemia in the LGB, optic radiations, or occipital lobe
- 50% from occipital stroke, 30% from parietal stroke, 25% from temporal stroke 5% from optic tract and/or lateral geniculate nucleus [Pambakian and Kennard).
- Type of visual field loss was

✓Complete HH 57% , Partial HH 19.5%

✓HH either congruous or incongruous

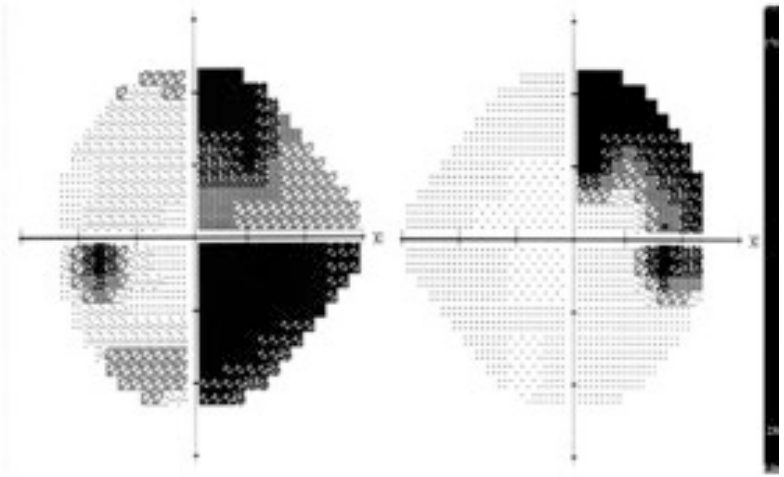
✓Also, can have sectoranopias, quadrantanopias, visual field constriction, scotomas

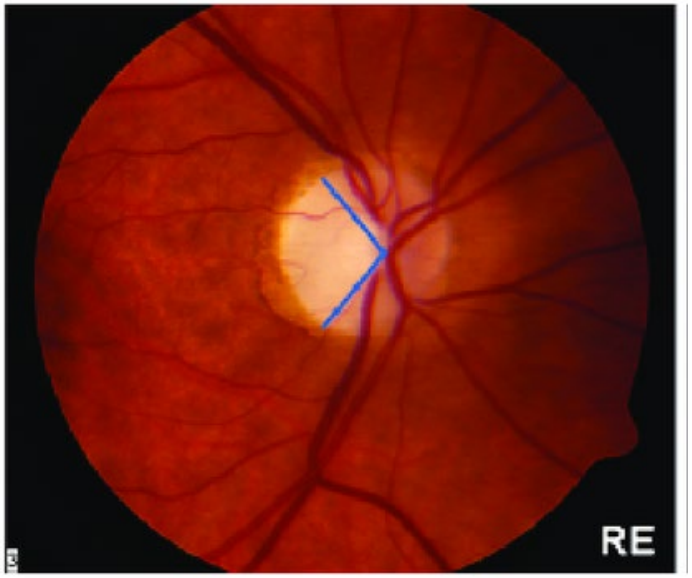
✓Bilateral Homonymous hemianopia/cortical blindness rare (Rowe et al).

✓Stroke is the most common cause for homonymous hemianopia.

OPTIC TRACT STROKE

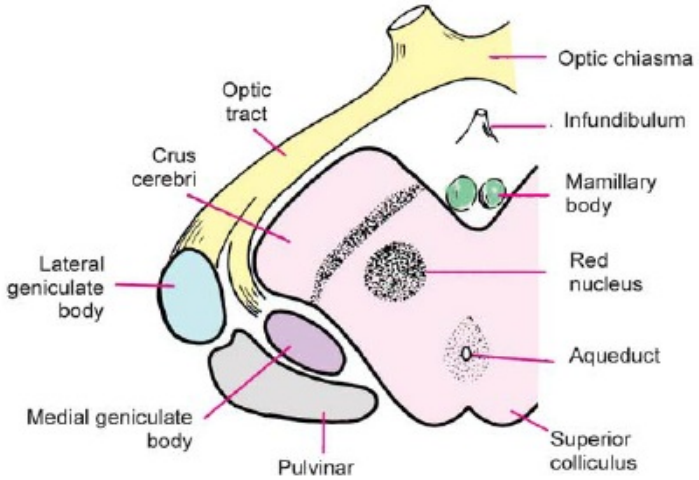
- Incomplete injury to the optic tract and the spatial spread of visual fibers in the optic tract can lead to incongruous contralateral homonymous hemianopia.
- Sectoral optic atrophy with ipsilateral temporal disc pallor & contralateral wedge-shaped pallor can occur.

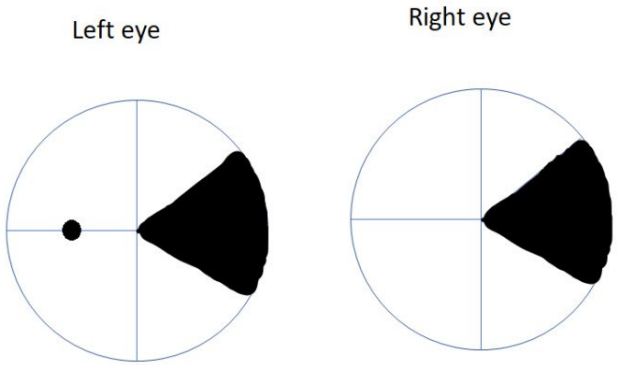




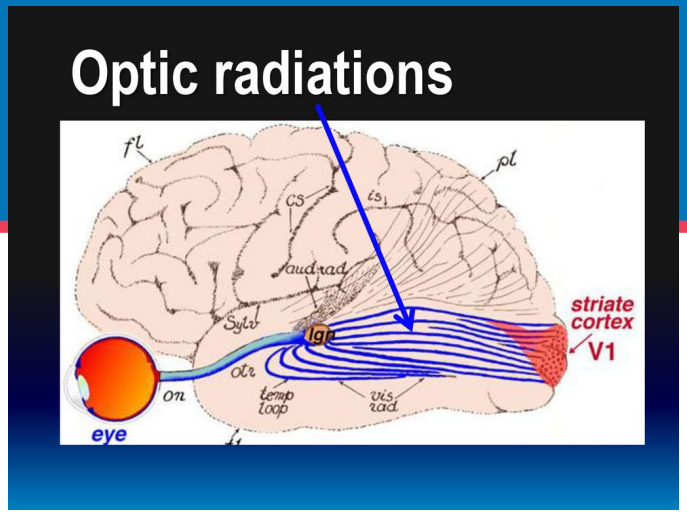
LAT GENICULATE BODY

- LGB is in a watershed area, prone to ischemia during hypoperfusion.
- Extensive LGB injury manifests as a complete HH.
- Medial LGB infarction presents as a wedge-shaped HH.
- Bilateral LGB infarction is rare, usually from hypoperfusion or hemorrhage.
- Incongruous Bil HH (bitemporal and binasal) with acute bilateral vision loss and dyschromatopsia.

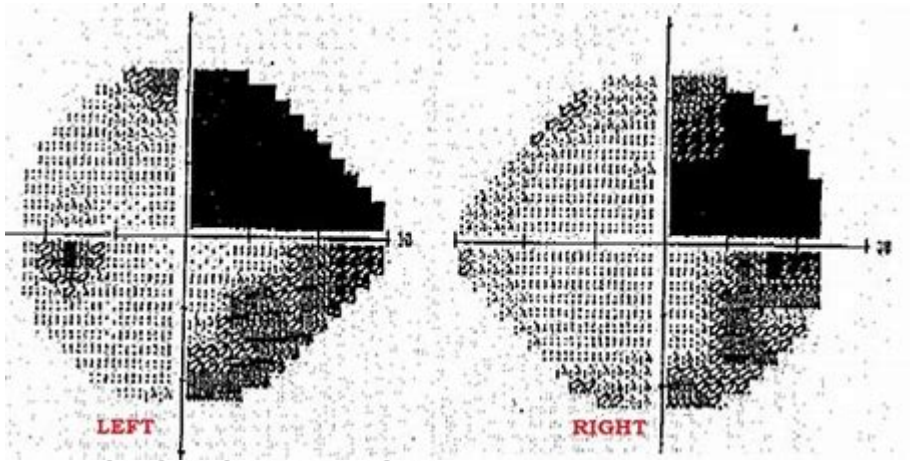




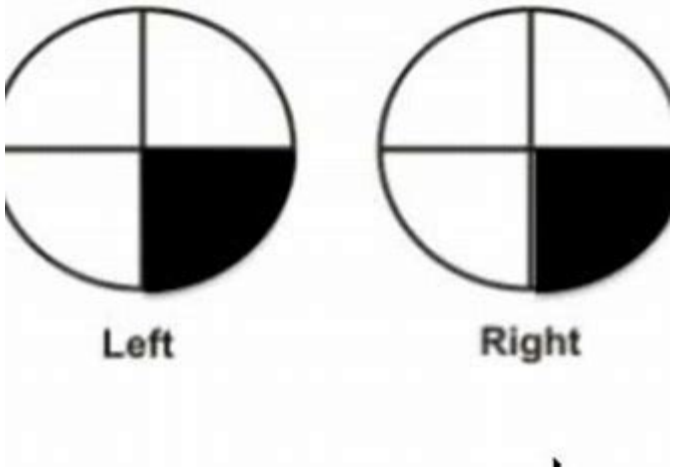
OPTIC RADIATION INFARCTION



Contralateral homonymous superior quadrantanopia, **'pie-in-the-sky'** defects, occurs with temporal lobe infarction involving Meyer's Loop with possible associated symptoms of seizure, memory loss and receptive aphasia.



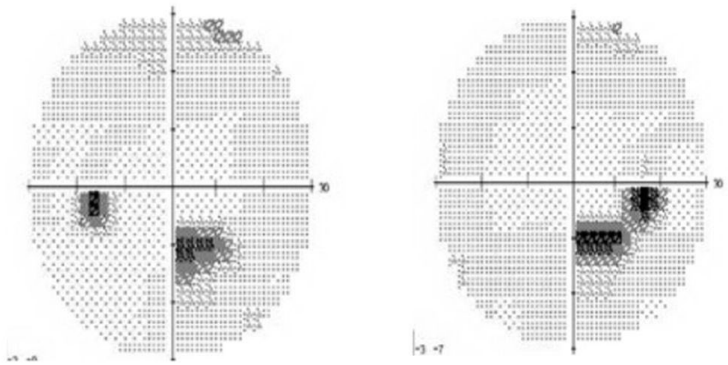
Contralateral homonymous inferior quadrantanopia **(pie-on-the-floor)** occurs with parietal lobe infarction, involving the parietal optic radiations with associated hemineglect.

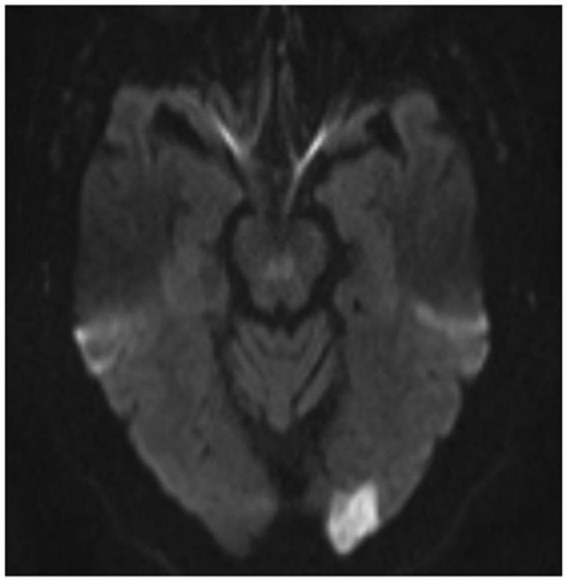


OCCIPITAL LOBE INFARCT

- PCA ischemia can produce congruous HH.
- Congruity occurs more posterior lesions in the visual pathway due to convergence in the occipital lobe.
- Congruous HH is predominantly caused by occipital lobe lesions (54%), followed by the optic radiations (33%).

OCCIPITAL LOBE INFARCT





- Macular sparing, where the center of the visual field remains unaffected.
- The dual blood supply from the PCA and the superior temporo-occipital sylvian artery causes macular sparing.

BILATERAL OCCIPITAL LOBE INFARCTS

- Cortical blindness : bilateral occipital lobe ischemia.
- Associated agnosia of the cortical blindness is Anton syndrome.
- Intact pupillary light reflex and normal appearing fundi.

VISUAL FIELD LOSS RECOGNITION CHALLENGES

- VF loss prevalence underestimated because many MCA strokes will have decreased levels of consciousness and are unable to report vision loss.
- Can be missed if confrontational visual field testing alone is used (Townsend BS et al).
- In a prospective study 62% failed to recognize their right- or left-sided visual field defect (Celesia et al).
- In the VIS Study, only 45% with visual field loss reported symptoms (Rowe et al).

RECOVERY

- 43% stroke patients had partial improvement in reading abilities while 11% had complete recovery(Rowe etal).
- Several studies have 57% will have persistent visual field defects, with contralateral homonymous hemianopsias and quadrantonopia being most common.
- Complete recovery occurred in 42.6 and 45 % by 30 and 90 days, respectively.

EFFERENT VISUAL PATHWAY

- Prevalence of ocular motility dysfunction after stroke: 22 to 70% [Pambakian A et al].
- 16.5% of poststroke patients had ocular misalignment with diplopia(Rowe et al).
- Diplopia occurs from horizontal or vertical ocular misalignment.

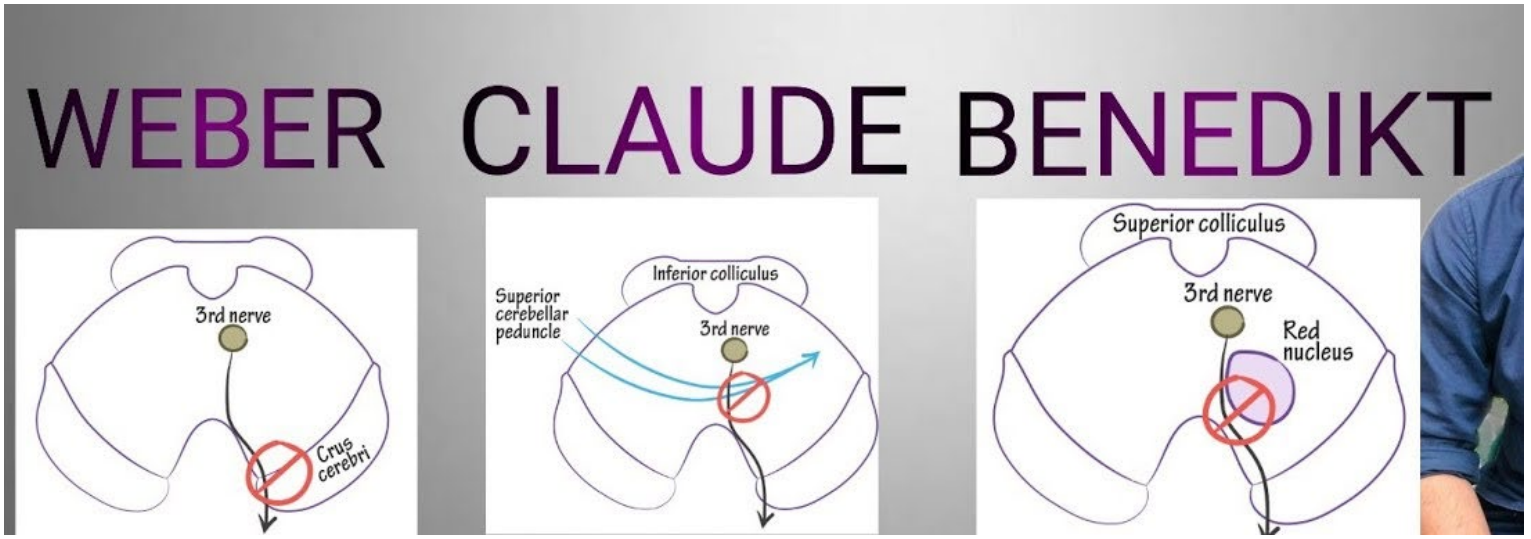
STRABISMUS

- Strabismus mainly seen with cortical strokes (69%)(Rowe et al)
- Most common strabismus was exotropia(74%).
- Strabismus from cranial nerve III palsy had the highest rate of occurrence.
- 6th and 3rd nerve palsies are more common in stroke than 4th nerve palsy.
- In 3rd N palsy, the medial rectus subnucleus may be affected in isolation.

THIRD/OCULOMOTOR NERVE PALSY

- Pure midbrain infarctions from PCA represent a small number of strokes (0.6%–2.3%).
- In midbrain stroke oculomotor palsy often will have other neurological findings such as hemiparesis or cerebellar dysfunction.

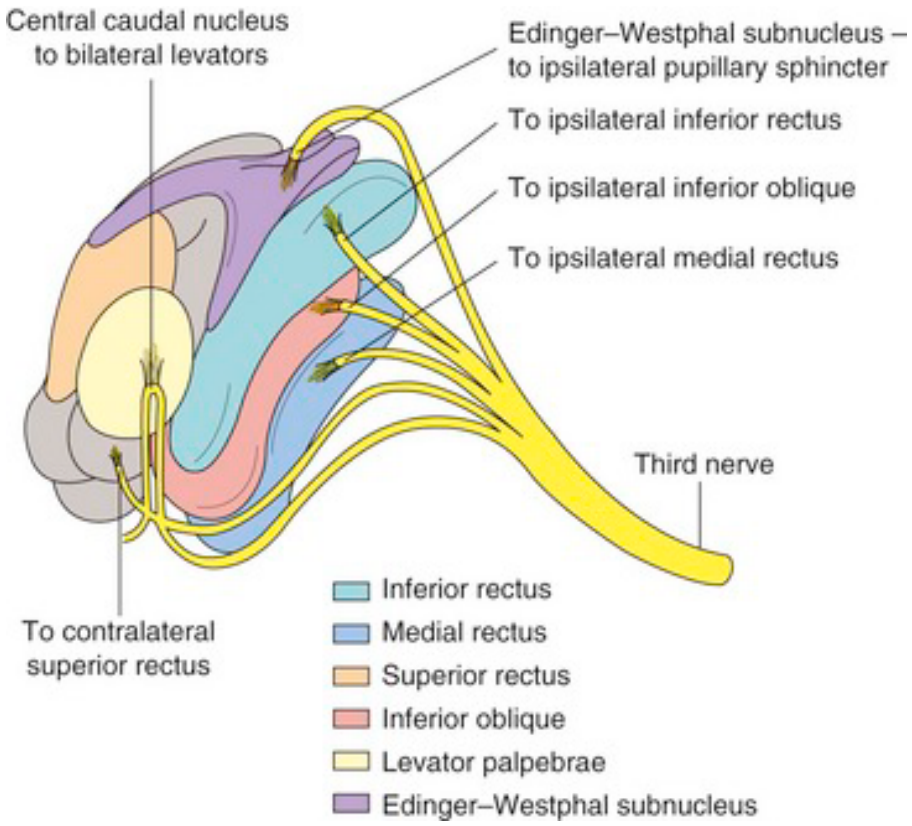
PARAMEDIAN MIDBRAIN INFARCTS



- Weber's syndrome -oculomotor nerve & contralateral hemiparesis
- Claude syndrome - oculomotor nerve and red nucleus
- Benedikt syndrome -oculomotor nerve + corticospinal tract + cerebellar ataxia

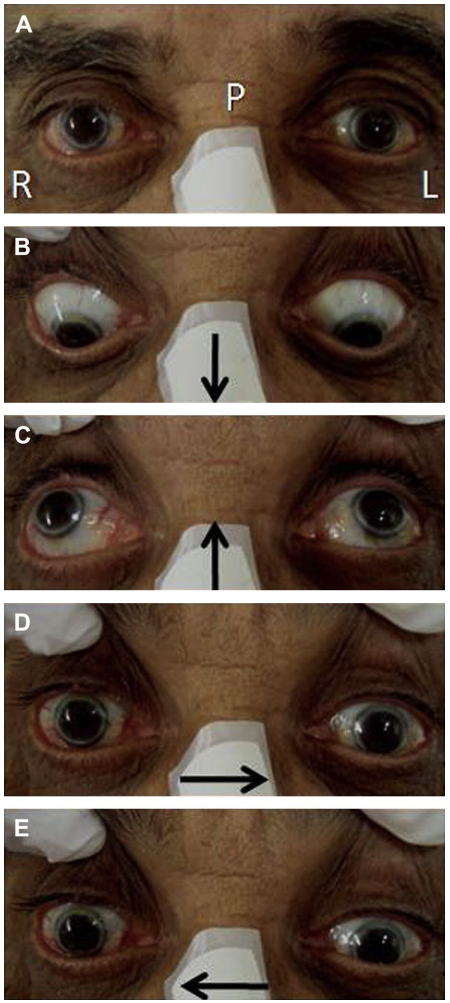
PTOSIS

- Paramedian midbrain infarctions can manifest as ipsilateral ptosis without ophthalmoplegia (Sugawara et al).
- Acute bilateral ptosis implies injury to the unpaired midline central caudal nucleus, a subnucleus of the oculomotor nucleus in the midbrain.
- Oculomotor fascicular infarction can also produce isolated unilateral ptosis without ophthalmoplegia or pupillary involvement.

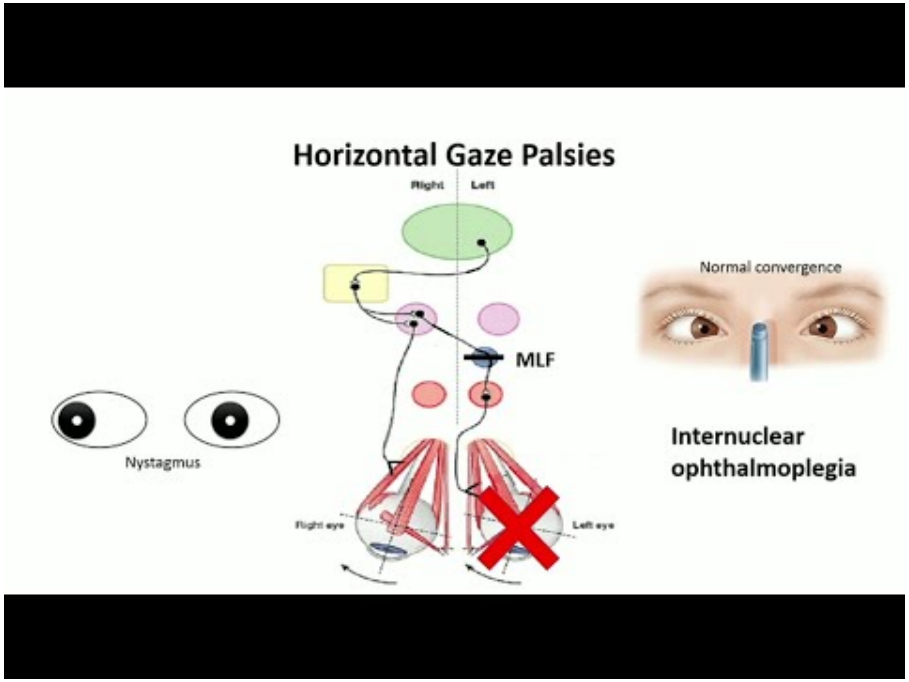


GAZE PALSY

- Gaze palsy is the most common ocular motility disturbance after stroke, reported in 18–44% CVA [Clisby, Rowe, Tao].
- Isolated or associated with other motility problems.
- Horizontal gaze palsies are more prevalent than vertical, and complete palsies are more common than partial.
- Horizontal gaze paresis indicates pontine sixth nerve nucleus injury involving the horizontal gaze center, the parapontine reticular formation (PPRF).
- Bilateral horizontal gaze palsy is rare and characteristic of tegmental pontine infarctions.



INTERNUCLEAR OPHTHALMOPLEGIA

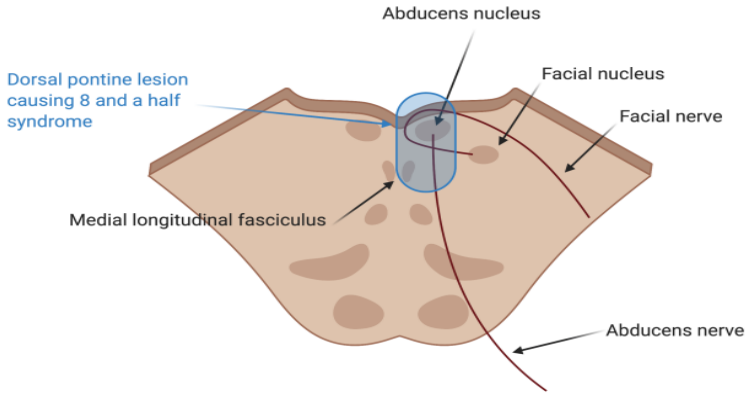


- INO predominantly unilateral.
- A stroke to the MLF is the most common cause of INO in those in 6th decade.
- Characterized by impaired horizontal conjugate gaze with ipsilateral adduction impairment and concomitant contralateral horizontal gaze-evoked abducting nystagmus.
- Convergence is spared in INO; (discerning INO from a third nerve palsy).

ONE AND HALF SYNDROME

- One-and-a-half syndrome : ipsilateral horizontal gaze palsy and Ipsilateral INO(contralateral adduction deficit.)

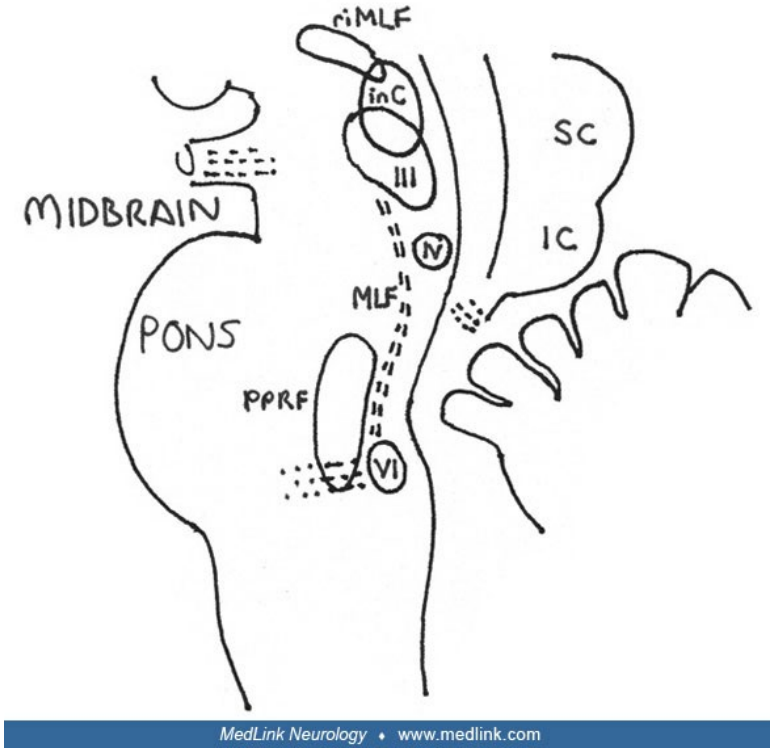
EIGHT AND HALF SYNDROME



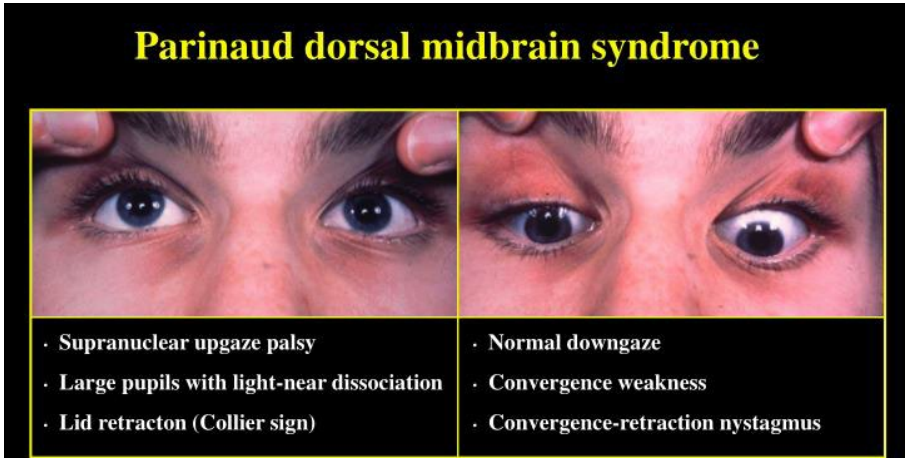
- Eight and-a-half syndrome, described in 1998 by Eggenberger, characterized by one-and-a-half syndrome & ipsilateral facial palsy.
- Lesions in the dorsal tegmentum in the caudal pons involving PPRF, MLF (one-and-a-half syndrome) and facial nerve nucleus and fasciculus (ipsilateral facial palsy).

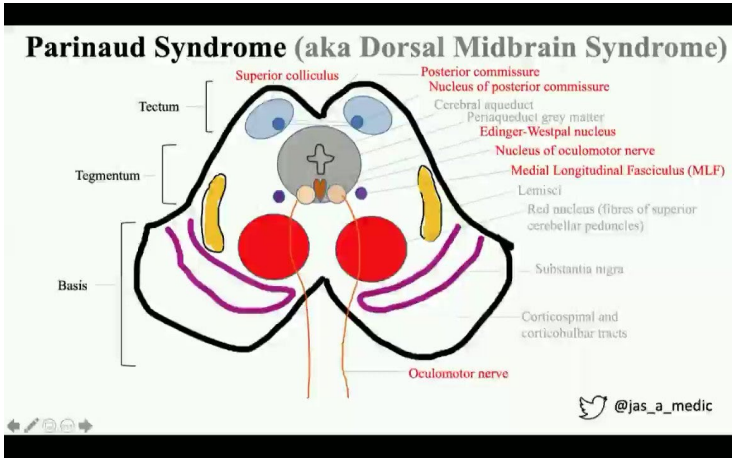
VERTICAL GAZE DEFECTS

- Vertical gaze (facilitated by 3rd and 4th nerves) impaired in midbrain and thalamic infarcts.
- Impaired gaze holding can result from injury to the **interstitial nucleus of Cajal, nucleus prepositus hypoglossi or medial vestibular nuclei.**
- Complete vertical gaze palsy can result from dorsal caudal pontine or bilateral paramedian midbrain–thalamic infarcts.
- Complete vertical, upgaze and downgaze palsies result from bilateral rostral interstitial MLF (riMLF) lesions.
- Isolated downgaze palsy : bilateral mediocaudal riMLF lesions.
- Isolated upgaze palsy: unilateral mesenphalic reticular formation lesions.



DORSAL MIDBRAIN SYNDROME



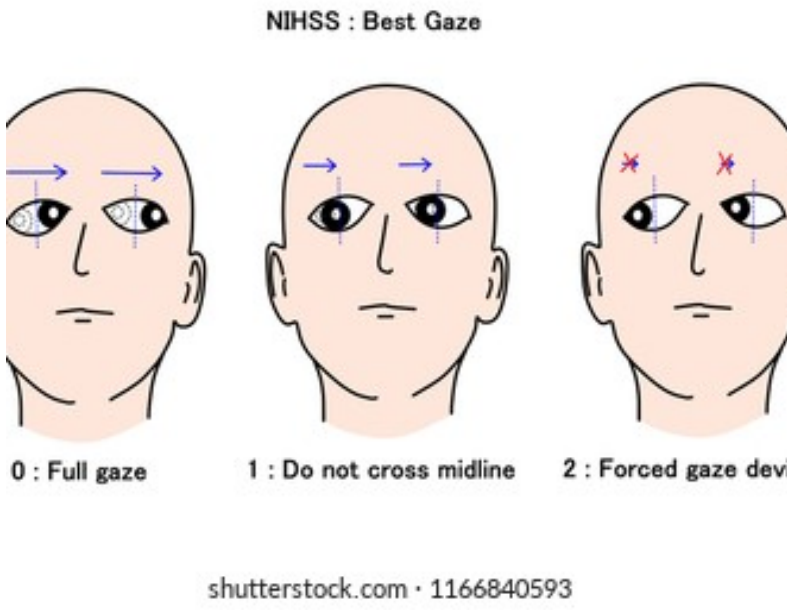


Parinaud's syndrome can arise from dorsal midbrain strokes and is characterized by

- impairment of upward saccades and pursuit
- light-near dissociation
- convergence-retraction nystagmus
- Lid retraction, known here as Collier's sign

SACCADIC/SMOOTH PURSUIT DEFICITS

- Both cortical and brainstem infarcts can lead to saccadic defects.
- Left frontal lobe lesion results in transient right gaze palsy and left gaze preference/deviation, which resolves when the PPRF assumes control for saccades.
- Hypermetric saccades towards the affected side and concomitant hypometric saccades towards the contralateral side occurs with brainstem and cerebellar infarcts.
- Impaired smooth pursuit occurs in ipsilateral occipitoparietal lesions.
- Cerebellar lesions also affect smooth pursuit.



NYSTAGMUS

- Prevalence of nystagmus after stroke is likely underestimated [Siddique Man etal].
- One study reported 24% with posterior circulation stroke had nystagmus [Searls de et al].
- Cerebellar stroke has the strongest association with acute nystagmus [Baier B et al].
- Bidirectional horizontal nystagmus can occur in posteroinferior cerebellar artery strokes with greater amplitude toward the ipsilateral side of the lesion.
- Upbeat nystagmus can occur in cerebellar strokes involving the superior vermis.

VERGENCE

- Convergence insufficiency (CI) is quite common after stroke(55% per Cilisby et al)
- Rowe noted that CVA patients had reading difficulties due to impaired near point of convergence.

CONCLUSION

- Wide variety of visual dysfunction occur in stroke.
- Not as widely recognized as motor or speech defects as a sign of stroke.
- Post stroke vision problems may be overlooked.
- Treatment remains limited.
- Continued education and recognition remains important.