

# OCULAR MOVEMENT DISORDERS FROM THE PERSPECTIVE OF NEUROLOGY

Can Kocasarac

- Subclasses of eye movements
- Pathologic eye movements in stroke patients
- Pathologic eye movements in comatose patients
- Pathologic eye movements in vestibular disorders
- Cranial nerve palsies

## Gaze shifting

- Saccades
- Smooth pursuit
- Vergence

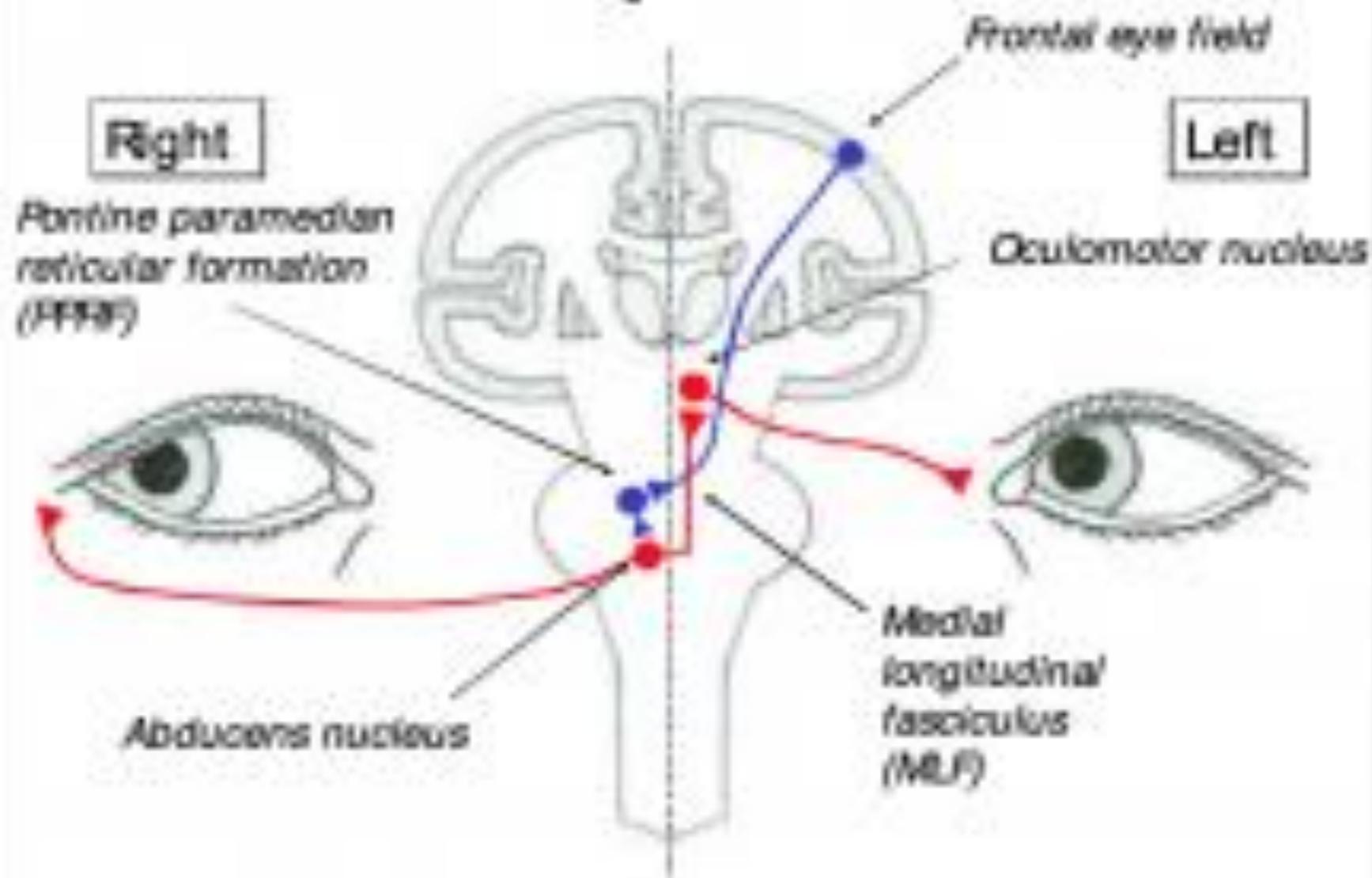
## Gaze holding

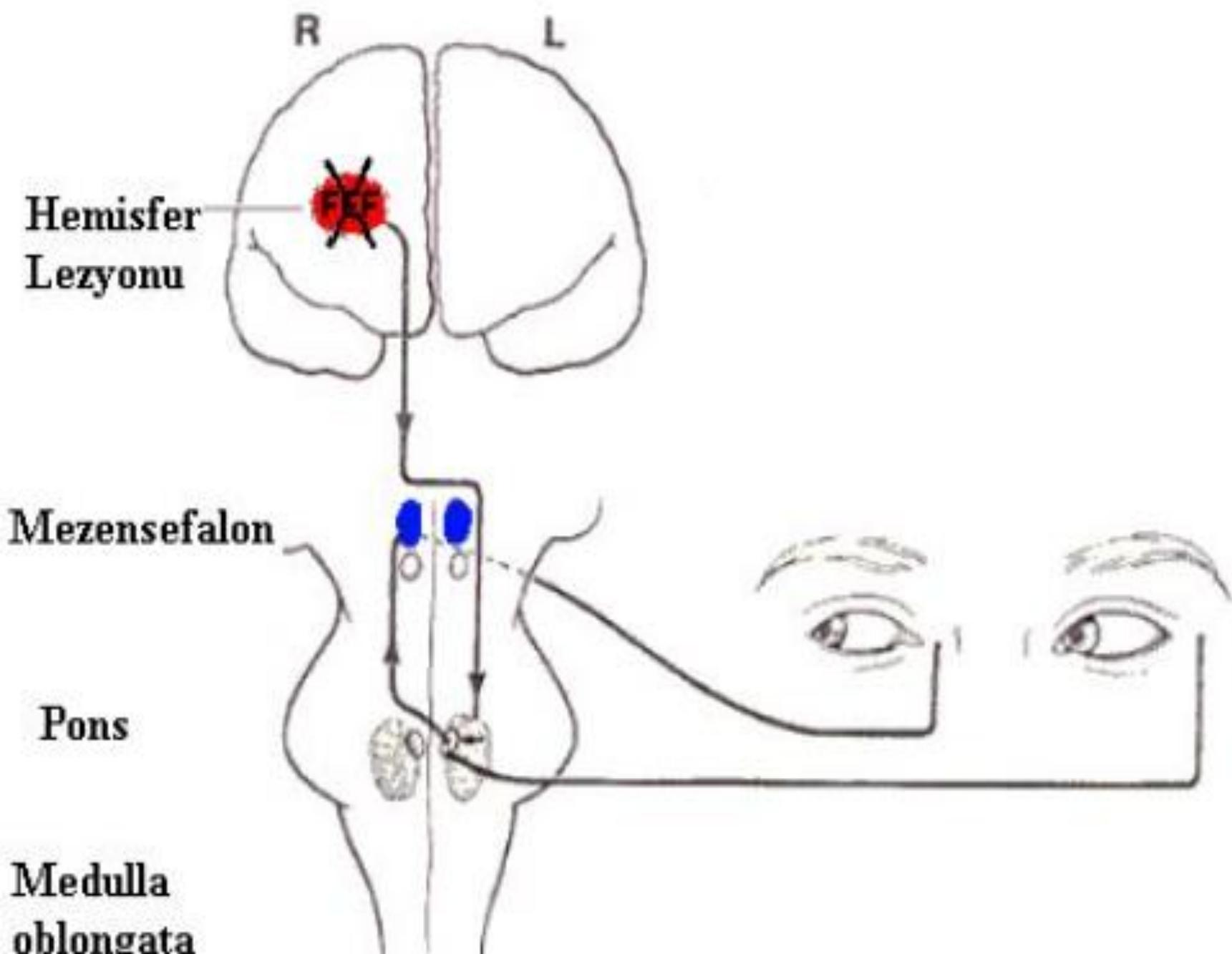
- Vestibular ocular reflex
- Optokinetic reflex

## Saccades:

- Rapid rotation of the eyes that bring image onto the fovea
- Occurs spontaneously in response to a sudden appearing object, or to scan a scene or to read
- Voluntary or reflexive
- Fastest eye movements (3 saccades a second)
- Generated in the frontal lobes and are under contralateral control; that is, right frontal lobe stimulation will result in a saccadic eye movement to the left

# Saccadic Eye Movements

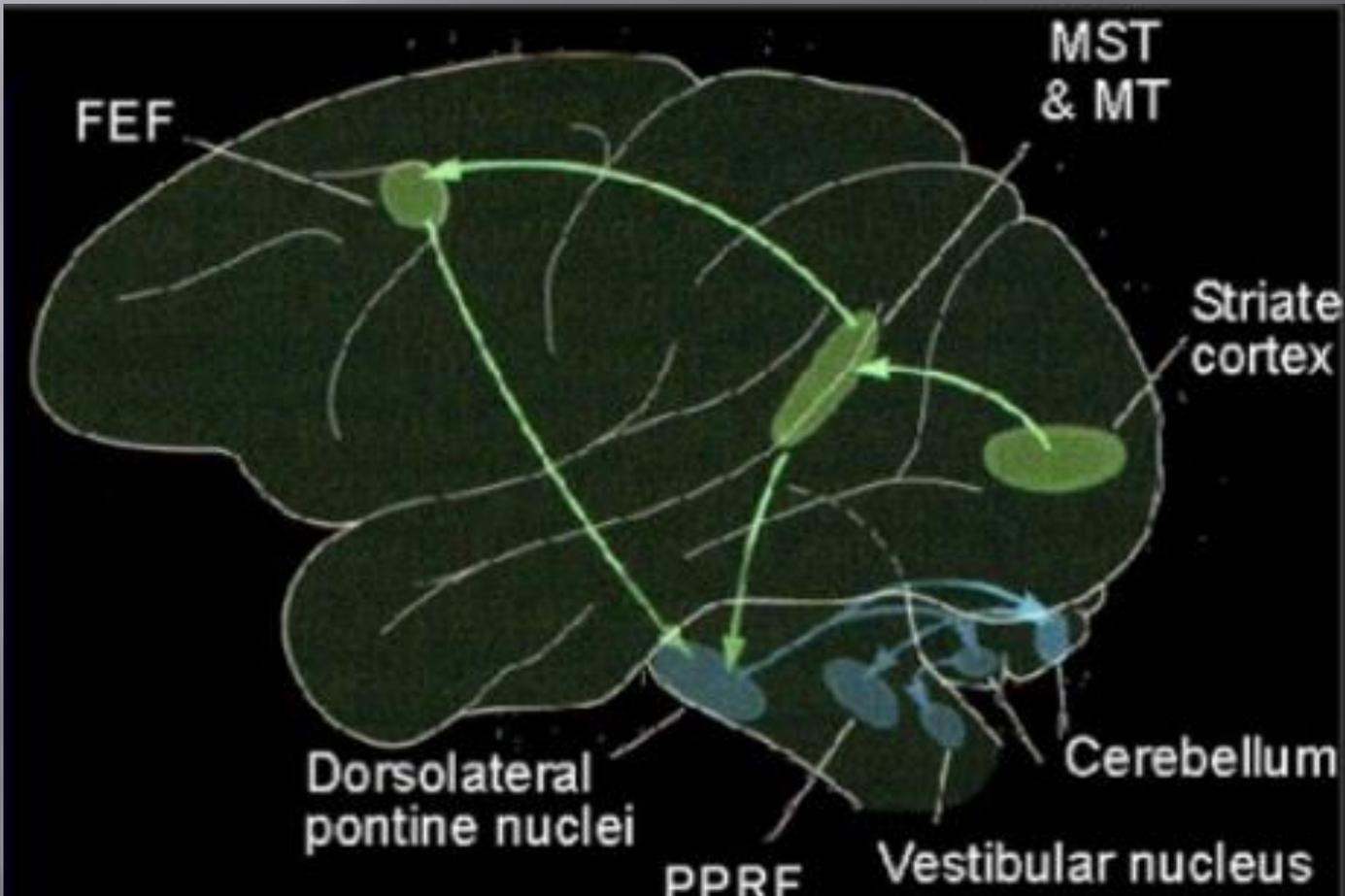




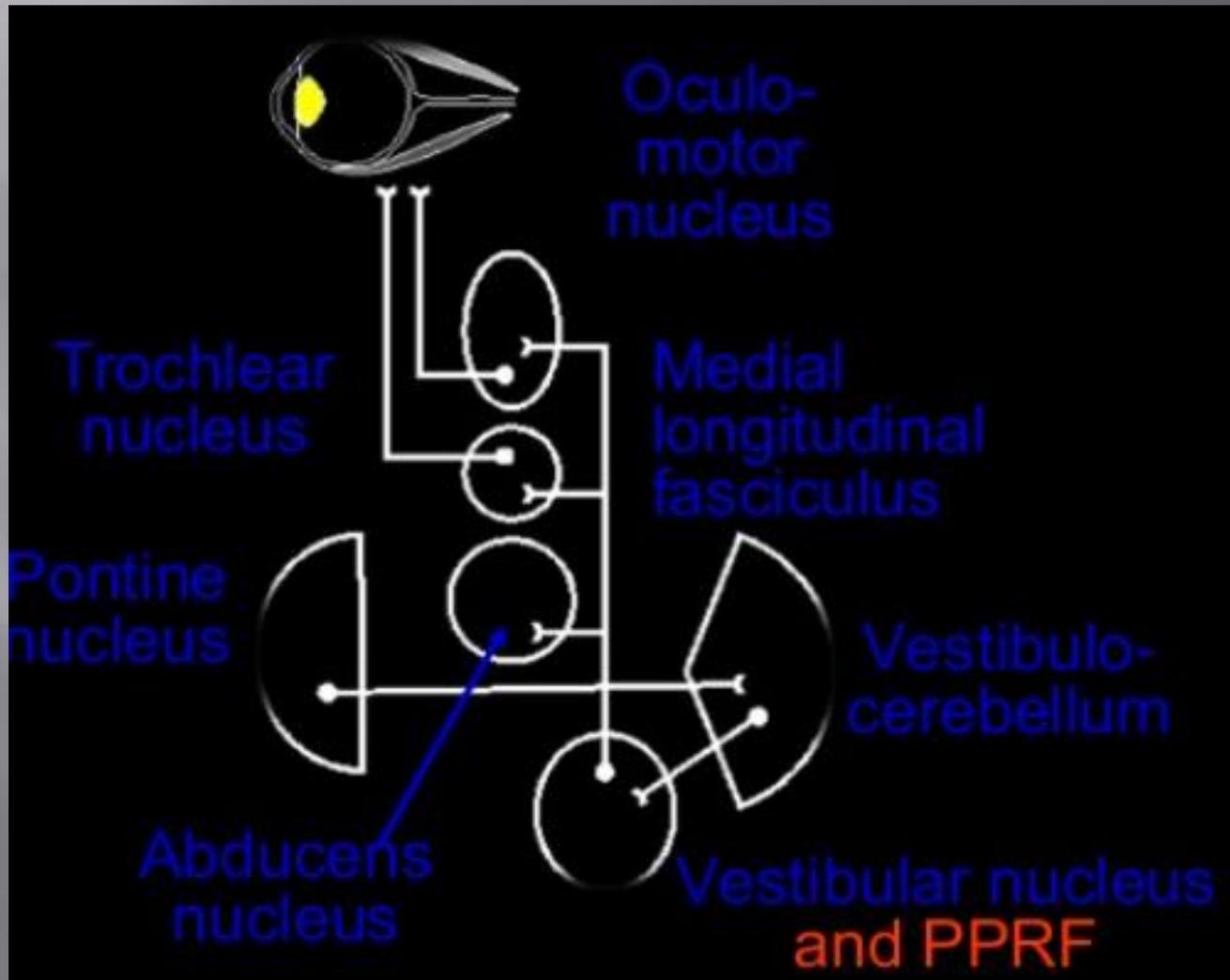
## Smooth pursuit

- Keeping a visible moving target on the fovea
- Isn't ballistic like saccades, instead moves smoothly

Smooth pursuit eye movements are generated in the occipital parietal temporal cortex



- Brainstem structures that are used to generate pursuit eye movement



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eye movement |

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## Vergence eye movements

-Aligns the fovea of each eye with target located at different distances from the observer.



## Vestibular ocular reflex (VOR)

- Compensating for head movements by rotating the eyes in opposite direction
- This reflex prevents visual images from slipping on the surface of the retina as head position varies
- Head movements are sensed by labyrinth of the inner ear

## Neuronal structures contributes to VOR

- Vestibular nucleus
- Abducens nucleus
- Oculomotor nucleus

# Eye movement disorders in stroke patients

-A recent Cochrane review of interventions for eye movement disorders in post-stroke patients concluded that “eye movement disorders may affect over 70% of stroke patients”

-Eye movement disorders may result in (a) “difficulty maintaining the normal ocular position” and (b) “difficulty moving the eyes appropriately” (ocular motility)

Interventions for disorders of eye movement in patients with stroke.

Pollock A1, Hazelton C, Henderson CA, Angilley J, Dhillon B, Langhorne P, Livingstone K, Munro FA, Orr H, Rowe FJ, Shahani U.

# Key medullary structures with ocular motor function and associated clinical findings

Structure	Main ocular motor function	Clinical findings in lesions
Vestibular nucleus (medial and caudal parts)	VOR Gaze-holding	Abnormal head impulse sign Skew deviation or ocular tilt reaction (OTR) Spontaneous nystagmus Gaze-evoked nystagmus
Perihypoglossal nuclei (NPH, Roller, and intercalatus)	Gaze-holding	Gaze-evoked nystagmus (mainly downbeat with lesions of NPH and upbeat with lesions of Roller and intercalatus nuclei)
Cell groups of paramedian tracts	Gaze-holding	Upbeat or downbeat nystagmus
Inferior olivary nuclei	Provide error signals to the cerebellum for adaption	Oculopalatal tremor
Inferior cerebellar peduncles (restiform bodies)	Convey sensory information to the cerebellum	Saccadic overshoot toward the lesion and undershoot away from the lesion (ipsipulsion)

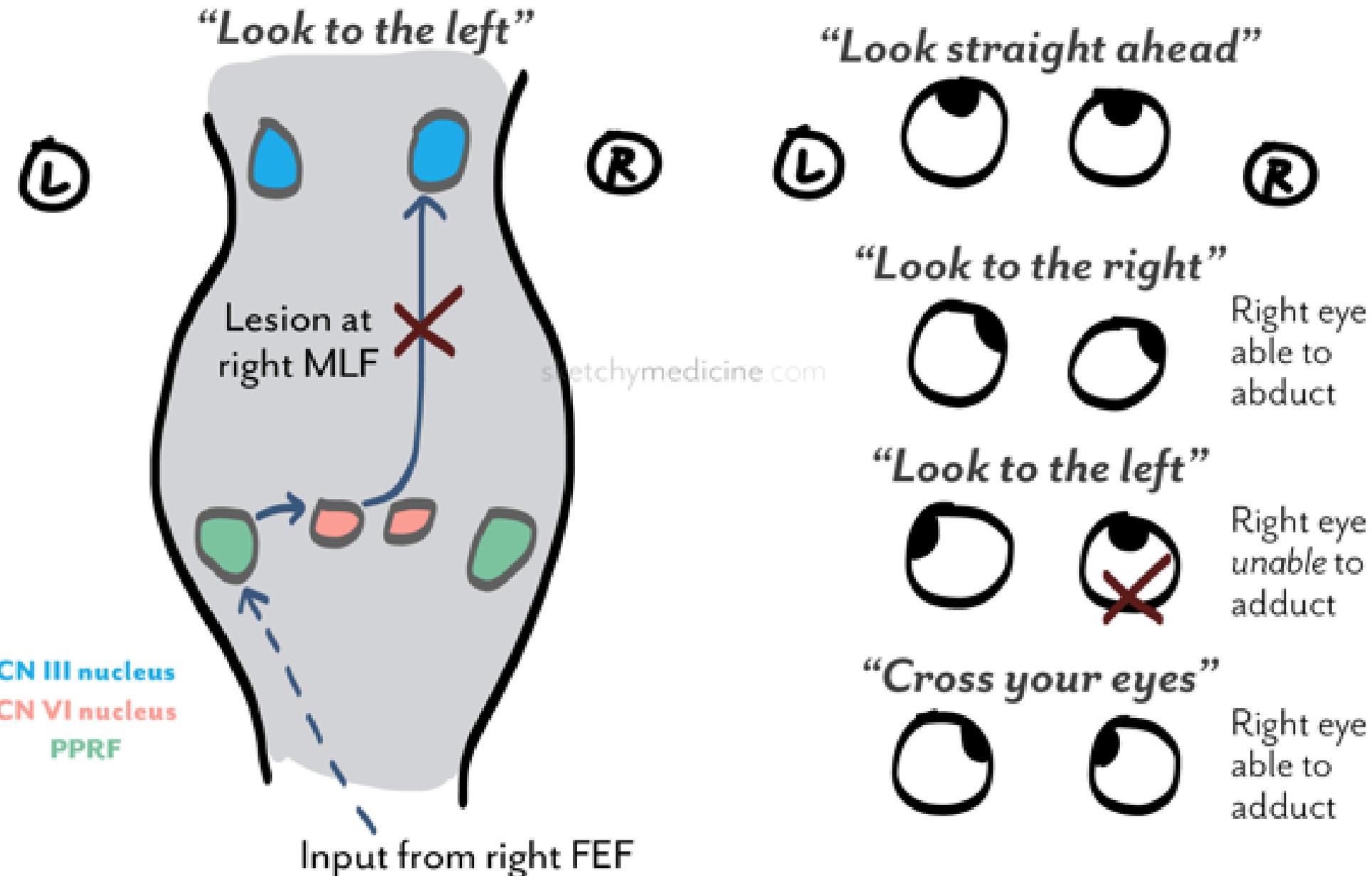
# Key pontine structures with ocular motor function and associated clinical findings

Structure	Main ocular motor function	Clinical findings in lesions
Abducens nucleus*	Conjugate horizontal gaze	Ipsilateral conjugate horizontal gaze palsy
PPRF*	Horizontal saccade generation	Selective horizontal saccadic palsy with sparing of vergence and pursuit
MLF	Conjugate gaze and VOR	Internuclear ophthalmoplegia (INO) Convergence can be spared Skew deviation or ocular tilt reaction (OTR) Asymmetric vertical VOR better with upward slow phases Dissociated vertical-torsional nystagmus
CTT	Conveys information from cerebellum to inferior olive	Oculopalatal tremor
Vestibular nucleus (rostral part)	VOR	Spontaneous nystagmus Abnormal head impulse sign Skew deviation

# INO

- Conjugate lateral gaze disorder
- Dysfunction of the Medial Longitudinal Fasciculus (MLF)
- MLF is a heavily myelinated tract which connects PPRF-abducens nucleus complex to oculomotor nucleus complex
- Bilateral INO= MS
- Older patient with an one sided INO, stroke

# Internuclear Ophthalmoplegia



# Key regions within the cerebellum with ocular motor function and associated clinical findings

Anatomic region

Ocular motor function

Clinical findings with lesions

Nodulus/uvula and flocculus/paraflocculus (vestibulocerebellum)

r-VOR amplitude and direction  
r-VOR duration (velocity storage)  
t-VOR generation  
Smooth pursuit  
Gaze-holding

Occasional abnormal head impulse sign  
Skew deviation, alternating on lateral gaze  
Downbeat nystagmus  
Gaze-evoked nystagmus  
Rebound nystagmus  
Periodic alternating nystagmus  
Head shaking-induced nystagmus  
Impaired smooth pursuit

Dorsal vermis (OMV)  
Fastigial nucleus (FOR)

Saccade initiation, acceleration accuracy and termination  
Smooth pursuit initiation and termination

## OMV

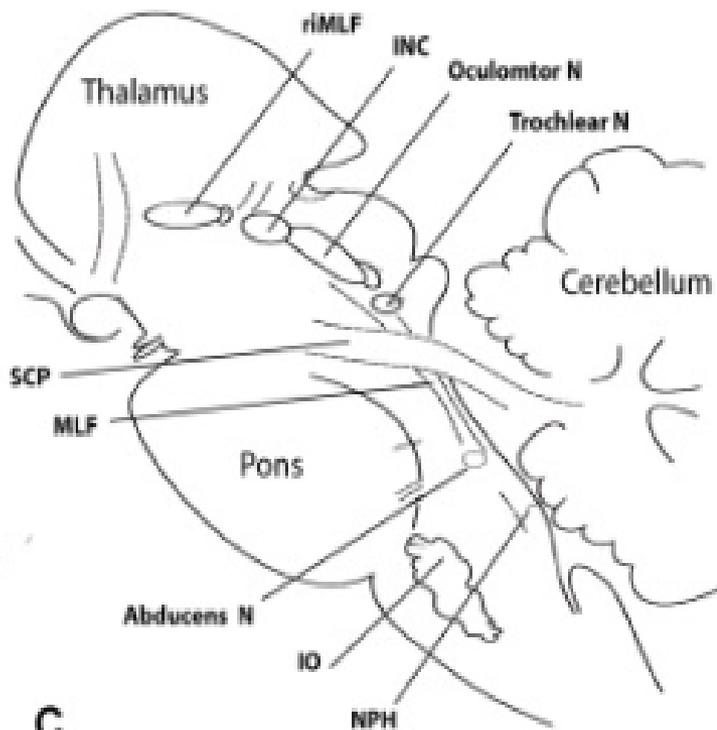
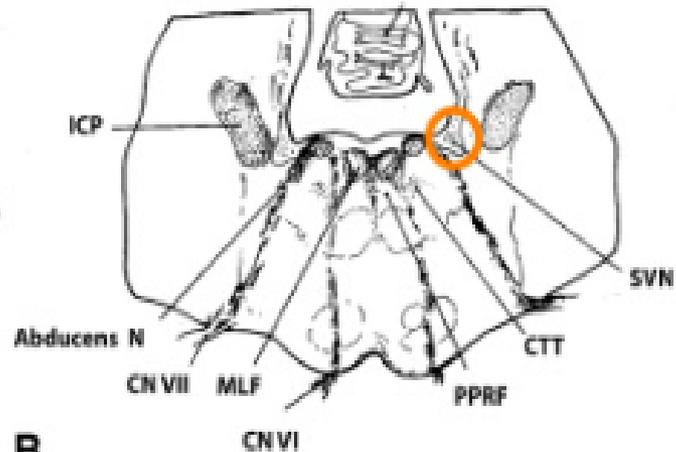
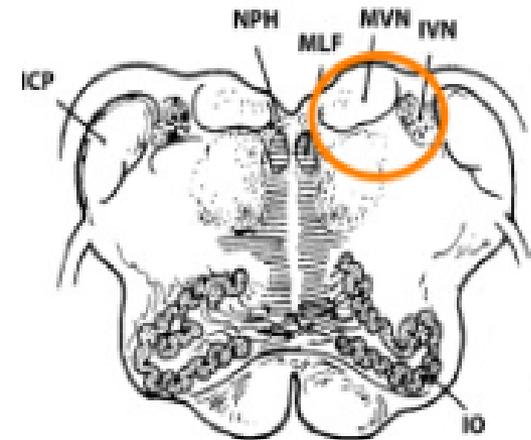
Ipsilateral saccade hypometria and contralateral hypermetria of saccades (contrapulsion)  
Bilateral lesions lead to hypometric saccades  
Impaired smooth pursuit toward the side of the lesion

## FOR\*

Hypermetric saccades

# Eye movement findings with lesions involving the midbrain

Structure	Main ocular motor function	Clinical findings with lesions
riMLF	Vertical saccade generation	<ul style="list-style-type: none"> <li>Impaired vertical saccades</li> <li>Tonic cyclodeviation of the eyes toward the side of the lesion</li> <li>Loss of quick phases of the nystagmus during the head roll toward the side of the lesion</li> <li>Torsional nystagmus, top pole beating away from the side of the lesion</li> </ul>
INC	Vertical gaze-holding	<ul style="list-style-type: none"> <li>Vertical gaze-evoked nystagmus</li> <li>Ocular tilt reaction (OTR)</li> <li>Torsional nystagmus, top pole beating toward the side of the lesion</li> </ul>
Posterior commissure*	Vertical gaze-holding	<ul style="list-style-type: none"> <li>Impairment of vertical eye movements (especially upward)</li> <li>Vergence abnormalities (convergence paralysis or spasm)</li> <li>Convergence-retraction nystagmus</li> <li>Pathologic lid retraction</li> <li>Light-near dissociation of pupillary reflex</li> </ul>
MLF	Conjugate gaze and VOR	<ul style="list-style-type: none"> <li>Internuclear ophthalmoplegia (INO)</li> <li>Skew deviation or OTR: higher eye on the side of the lesion, but head tilt and ocular counter-roll away from the side of the lesion</li> <li>Asymmetrical vertical VOR better with upward slow phases</li> <li>Dissociated vertical-torsional nystagmus</li> </ul>
Trochlear nucleus	Nucleus of trochlear (IV) cranial nerve	<ul style="list-style-type: none"> <li>Affected eye higher and extorted (as opposed to skew deviation in which higher eye is intorted)</li> </ul>
Oculomotor nucleus	Nucleus of oculomotor (III) cranial nerve	<ul style="list-style-type: none"> <li>Affected eye lower</li> <li>Limited adduction</li> <li>Ptosis</li> <li>Dilated pupil</li> </ul>



**A**

**B**

**C**

Key structures with ocular motor function within the medulla (**A**: axial section), pons (**B**: axial section), and midbrain (**C**: parasagittal section). MVN, medial vestibular nucleus; IVN, inferior vestibular nucleus; SVN, superior vestibular nucleus (orange circles); ICP, inferior cerebellar peduncle; NPH, nucleus prepositus hypoglossi; MLF, medial longitudinal fasciculus; IO, inferior olive; CN VI, cranial nerve VI; CN VII, cranial nerve VII; PPRF, paramedian pontine reticular formation; CTT, central tegmental tract; SCP, superior cerebellar peduncle; INC, interstitial nucleus of Cajal; rIMLF, rostral interstitial nucleus of medial longitudinal fasciculus.

(Modified from Leigh and Zee, 2015, with permission from Oxford University Press. **C** is based on a figure

(a) Posterior parietal cortex

-Unilateral spatial neglect (USN)

-Impaired eye movements to the contralesional hemifield

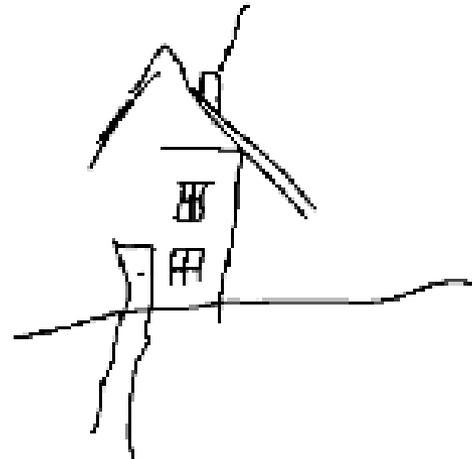
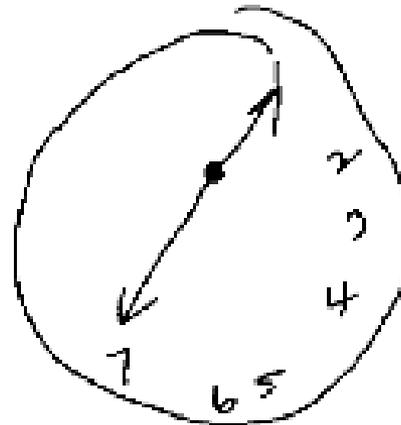
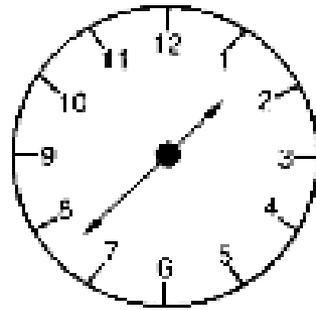
(b) Posterior occipito-temporal cortical areas

-Visual field defect; homonymous hemianopia

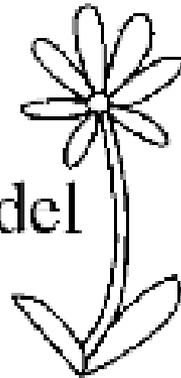
-Abnormal eye movement into the hemianopic visual field; USN),

(c) Frontal lobe

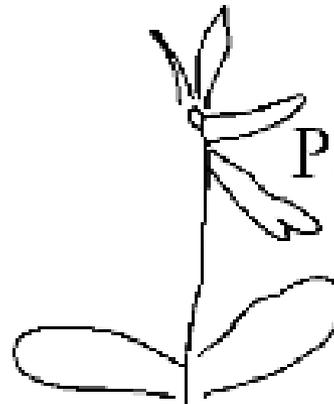
-Saccadic eye movement and smooth pursuit eye movement dysfunction



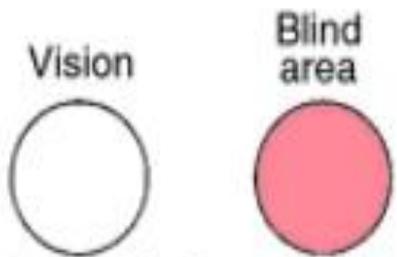
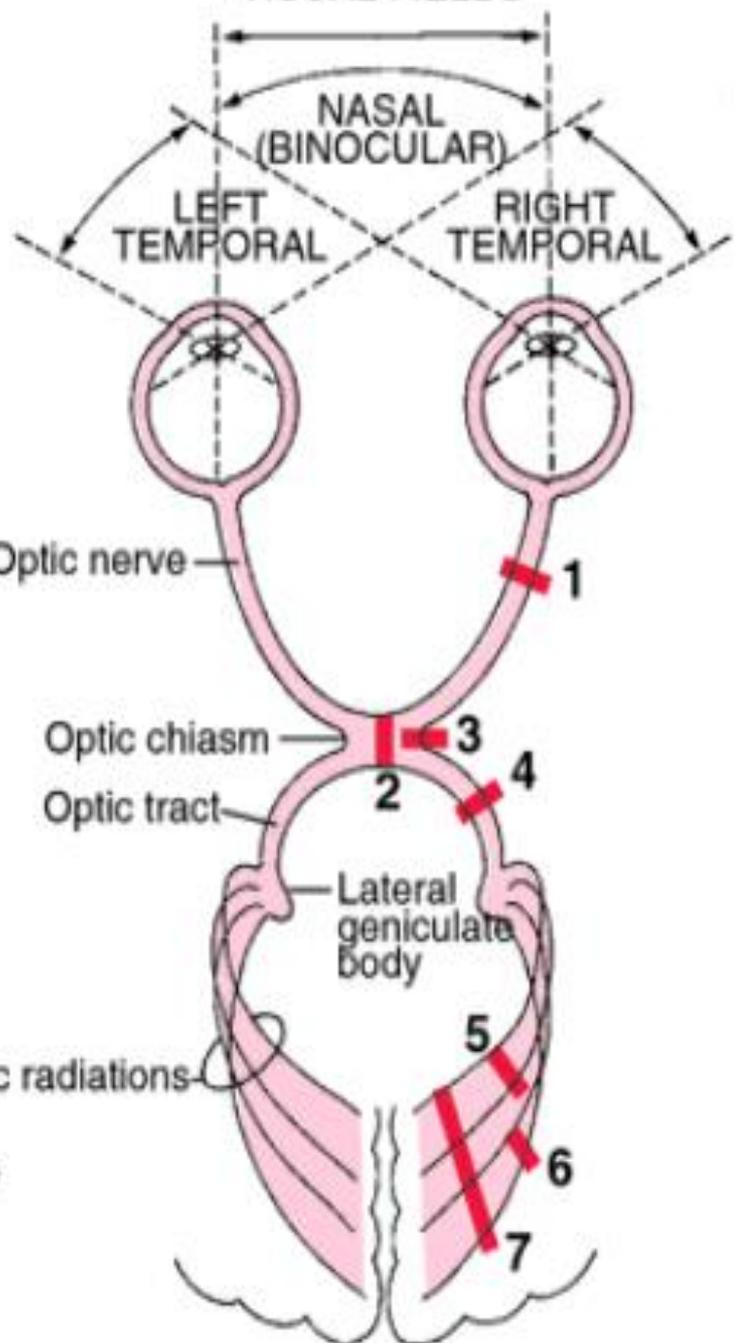
Model



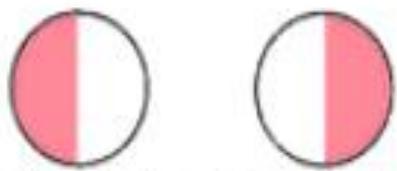
Patient's  
copy



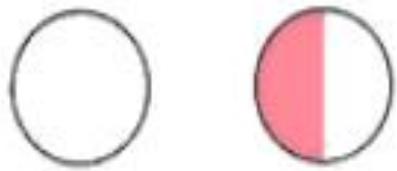
# VISUAL FIELDS



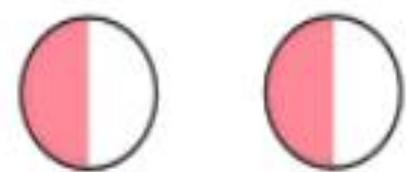
**1** Total blindness of right eye due to complete lesion of right optic nerve



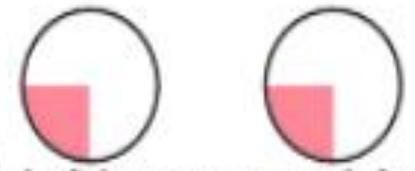
**2** Bipolar hemianopia due to midline chiasmatal lesion



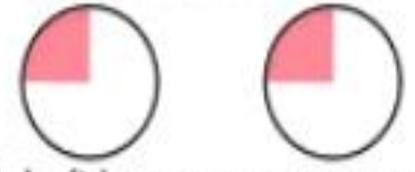
**3** Right nasal hemianopia due to lesion involving right perichiasmatal area



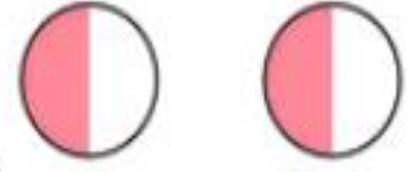
**4** Left homonymous hemianopia due to lesion or pressure on right optic tract



**5** Left homonymous inferior quadrantanopia due to involvement of lower right optic radiations



**6** Left homonymous superior quadrantanopia due to involvement of upper right optic radiations



**7** Left homonymous hemianopia due to lesion of right occipital lobe

#### (d) Basal ganglia

-Impaired saccadic- and smooth pursuit eye movements; impaired convergence; USN

#### (e) Thalamus

-Conjugate gaze deviation contralateral to the side of the lesion; abnormal subjective visual vertical; inaccurate saccadic eye movements; USN

#### (f) Brainstem

-Conjugate gaze abnormalities, nystagmus

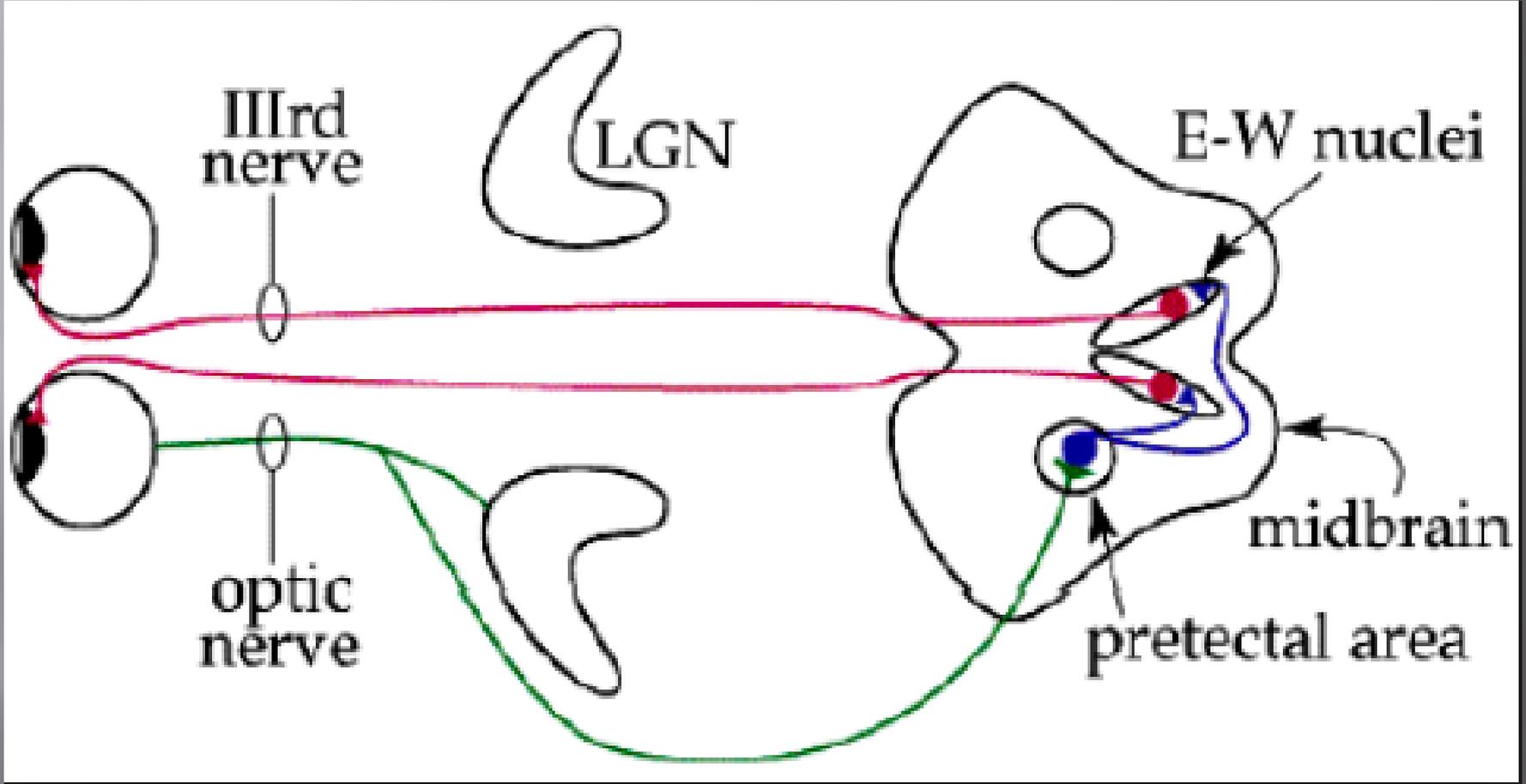
#### (g) Cerebellum

-Impaired smooth pursuit eye movements and VOR suppression; gaze-holding dysfunction;

# Clinical observations of patients with eye movement disorders following a stroke may include the following

- Head turn or head tilt during near (close-up) tasks
- Avoidance of near tasks
- Closing or covering one eye during conversations and/or activities due to blurred vision or double-vision
- Having difficulty maintaining eye contact
- Neglecting one side of the body or space during functional activities
- Difficulty with activities of daily living due to poor eye-hand coordination – knocking objects over or missing objects during reaching

- Decreased attention during conversations and/or activities
- Losing the place when reading
- Experiencing eye strain or headaches
- Not seeing people or objects approaching suddenly from one side
- Having difficulty concentrating on tasks
- Having difficulty “seeing” with or without glasses (cortical blindness)



# Eye movements in comatose patients

## (a) Roving eye movements

-Slow random predominantly horizontal conjugate eye movements

-Likely cause: metabolic encephalopathy (may be absent in deep coma), bilateral supranuclear lesions

## (b) Ocular bobbing

-Rapid, conjugate, downward movement; slow return to primary position

-Likely cause: Pontine strokes; other structural, metabolic, or toxic disorders

<https://www.youtube.com/watch?v=YAUT15d1YBU>

### (c) Ocular dipping

-Slow downward movement; rapid return to primary position

-Likely cause: Unreliable for localization; follows hypoxic-ischemic insult or metabolic disorder

### (d) Ping-pong gaze

-Horizontal conjugate deviation of the eyes, alternating every few seconds

-Likely cause: metabolic encephalopathy, bilateral cerebral hemispheric dysfunction; toxic ingestion

(e) Periodic alternating gaze deviation

-Horizontal conjugate deviation of the eyes,  
alternating every 2 minutes

-Likely cause: Hepatic encephalopathy; disorders  
causing periodic alternating nystagmus and  
unconsciousness or vegetative state

(f) Vertical myoclonus

-Vertical pendular oscillations

-Likely cause: Pontine strokes

## (g) Horizontal myoclonus

- Rapid horizontal pendular oscillations; the eyes appear to be shaking
- Serotonin toxicity

## (h) Monocular eye movements

- Small, intermittent, rapid monocular horizontal, vertical, or torsional movements
- Likely cause: Pontine or midbrain destructive lesions

# Pathologic eye movements in vestibular disorders

## Peripheral vestibular nystagmus

Mixed, horizontal torsional that beats away from the lesion

Jerk movements (fast phase and slow phase)

Strongly suppressed by visual fixation

Increase with gaze toward the fast phase (Alexander's law)

Does not change direction with change in gaze position

May be induced by convergence

## Central nystagmus

Mixed, pure torsional or pure vertical

Jerk or pendular movements

Usually weakly suppressed by visual fixation

May increase with gaze away from the fast phase (anti-Alexander's law)

May change direction (e.g., rebound, gaze-evoked, or periodic alternating nystagmus)

May be induced or change direction by convergence

# Third cranial nerve palsy

-Partial to complete weakness of the muscles innervated by the 3rd nerve

-Ptosis of the eyelid, outward drifting of the eye, mydriasis (strongly suggest oculomotor nerve compression).

-The affected eye is unable to look in towards the nose, up, or down



- Congenital, secondary to serious head trauma, vascular abnormality, tumor, infection, demyelination, inflammation, migraine
- Oculomotor paralysis in an increasingly unresponsive patient suggests transtentorial herniation
- If the pupil is spared, the cause is likely to be an ischemic process of the oculomotor nerve (DM)
- Neuroimaging, cerebral angiography

# Fourth Cranial Nerve Palsies

- Weakness of the muscle innervated by the 4th (trochlear) nerve (superior oblique muscle)
- These palsies are often difficult to detect because they affect vertical eye position predominantly when the eye is turn inward.
- The patient sees double images one above to the other.

-Many are idiopathic. Closed head trauma without skull fracture is a common cause  
Aneurysms, tumors, and multiple sclerosis are rare causes

-Neuroimaging, usually diagnosis is obvious from the history and physical examination

# Sixth Cranial Nerve Palsies

- Weakness of the muscles innervated by the abducens nerve.
- The eye is turned inward; it moves outward sluggishly, reaching the midline at most.
- Idiopathic, DM, trauma, aneurysm, MS





Thank You.