SUPRANUCLEAR GAZE CONTROL AND LESIONS

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SUPRANUCLEAR GAZE CONTROL

There are six ocular motor subsystems (OMS) that exist for supranuclear control of eye movements. These enable the fovea to find and fixate a target and maintain binocular foveation during head or target movement or both.

THE SIX OCULAR MOTOR SUBSYSTEMS

- **Fixation** – holds eye still during intent gaze
- **Saccadic** – points fovea to objects of interest
- **Smooth pursuit** – keeps moving targets on the fovea
- **Vergence** – aligns eyes to look at different depths
- **Vestibulo-ocular reflex (VOR)** – holds images still on the retina during brief head movements. It is driven by the vestibular system.
- **Optokinetic (OKN)** – holds images still during sustained head rotation. It is driven by the visual system.

SACCADIC MOVEMENTS

Saccadic movements enable the eyes to look in any desired direction until an object of interest is found. It allows for little flick movement of the eyes and moves the eyes rapidly (up to 800º/s). It supplies the brain with still pictures.

HORIZONTAL SACCADIC MOVEMENTS

- For leftward gaze, the left lateral rectus muscle and the right medial rectus muscle receive a pulse (burst of innervation). The right lateral rectus muscle and the left medial rectus muscle are reciprocally inhibited.
- The pathway for horizontal saccades originates in the premotor cortex, the frontal eye fields. The fibers then pass to the contralateral horizontal gaze center in the paramedian pontine reticular formation (PPRF). Saccades are therefore initiated by the contralateral frontal lobe.
- Pause cells lie in the nucleus raphe interpositus.
- Tonic neurons lie in the neural integrator – Nucleus prepositus hypoglossi.
- During a saccade, the burst neurons are activated, the pause neurons are switched off and the neural integrator allows for maintenance of gaze away from the midline.

PARAMEDIAN PONTINE RETICULAR FORMATION (PPRF)

The PPRF is located ventral and lateral to the medial longitudinal fasciculus (MLF). It innervates the ipsilateral abducens nucleus, rostral medulla and midbrain reticular formation. It coordinates horizontal and vertical eye movements.

The PPRF receives input from the:

- Medial Vestibular Nucleus
- Contralateral Frontal Eye Field
- Ipsilateral Posterior Parietal Cortex
- Superior Colliculus
- Cerebellum

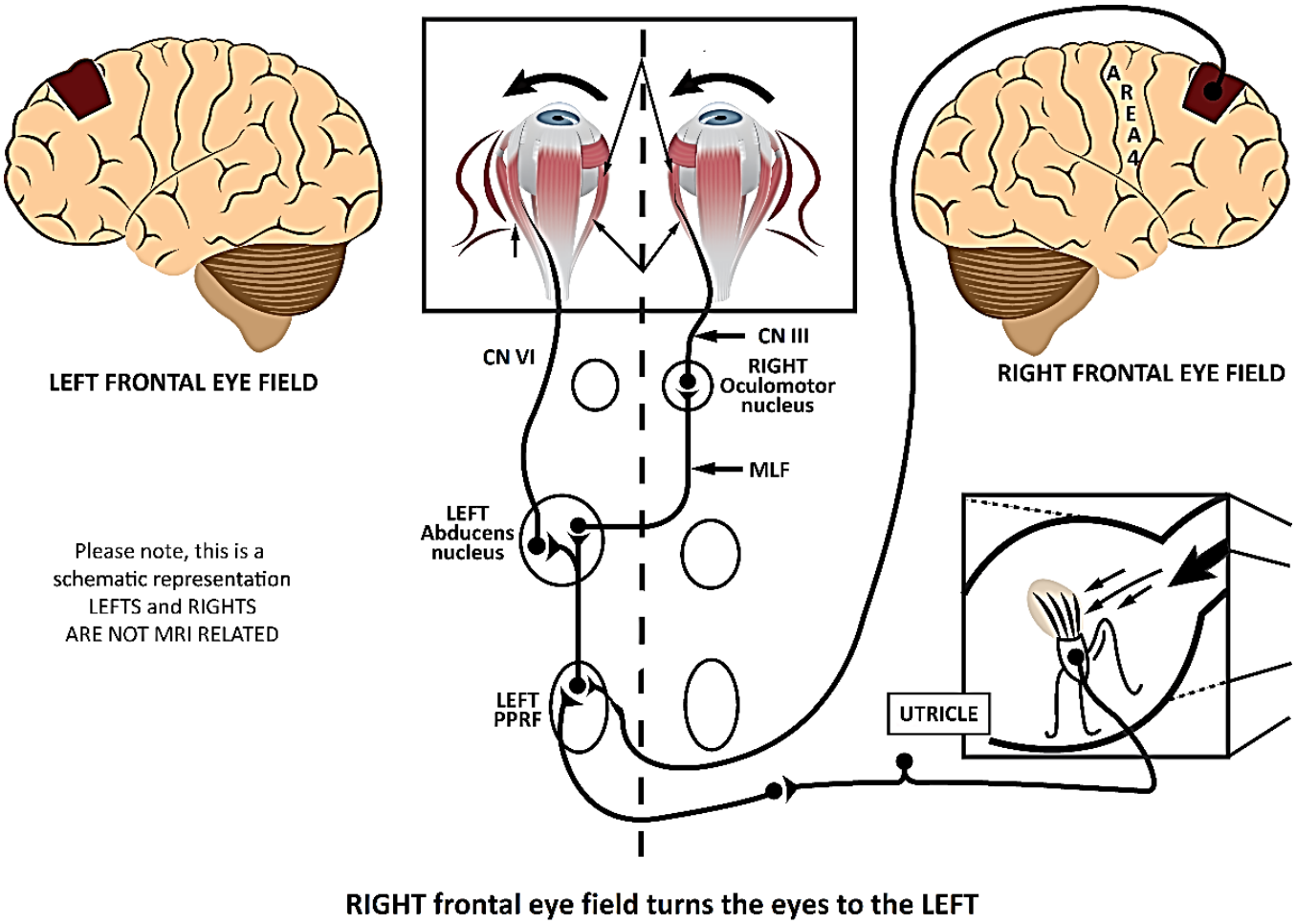


***Figure 4.1**: Horizontal gaze control*

VOLUNTARY GAZE MECHANISM

Cortical areas controlling voluntary gaze lie in the premotor strip of the frontal lobe (AREA 8). The descending pathways pass in the corona radiata through the anterior limb of the internal capsule. It descends to the level of the 6th N nucleus into the paramedian pontine reticular formation (PPRF). The 6th N nucleus is activated via this area. Via the medial longitudinal fasciculus, the opposite 3rd N nucleus is activated. Hence simultaneously, the ipsilateral 3rd nerve and contralateral 6th nerve result in conjugate gaze opposite to the activated frontal gaze center.

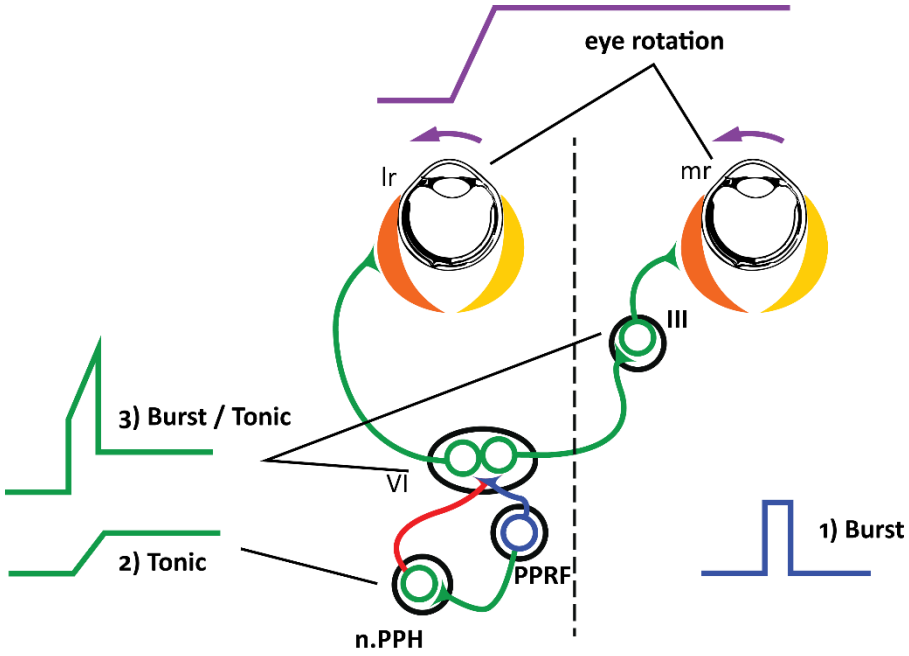


Figure 4.2: *Voluntary gaze mechanism*

PURSUIT MOVEMENTS

Pursuit is the ability to hold the object on the same point of the retina in spite of movement of the object. It consists of smooth movements to avoid jerky vision. This is controlled by the parieto-occipital cortex. It enables the eyes to track slowly moving targets (up to 70º/s) and is limited by object acceleration. A fast moving target moves the image off the fovea which produces a retinal error signal. If the signal is < 70º/s, it triggers the parieto-occipital cortex and if it is greater than 70º/s, it provokes the saccadic system to make a catch-up saccade.

PURSUIT EYE MOVEMENTS

The object is followed by voluntary effort. The eyes 'lock in' on the object and move in response to movement of the object, which is kept focused on the macula. The pathway originates in the peristriate cortex of the occipital lobe. Direct relay from the visual cortex to AREA 19 (Parietal), controls the movement. Area 19 projects via cortico-tectal and cortico-tegmental fibers into the midbrain and pons. Each occipital lobe controls pursuits to the ipsilateral side. There is also an input from the vestibular nuclei and cerebellum modifying pursuit movements. There is a large projection to the frontal eye field from area 19 (Figure 4.29).

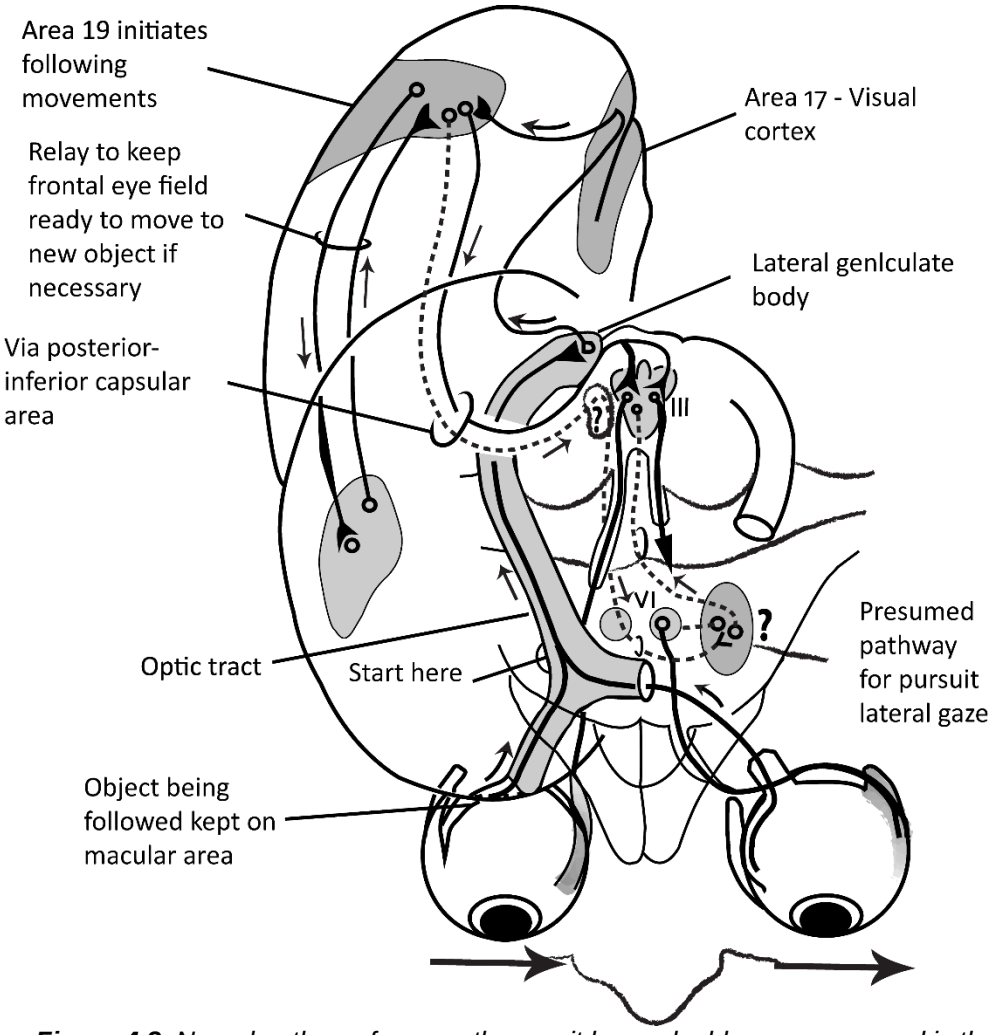


***Figure 4.3**: Neural pathway for smooth pursuit has a double cross over and is therefore responsible for ipsilateral pursuit*

VESTIBULAR OCULAR REFLEX

The vestibular ocular (VOR) reflex allows for the maintenance of the straight-ahead position of the eyes. It is responsible for continual adjustment of the eye position in relation to the head position and is mediated by vestibular activity and proprioceptive information integrated at brainstem and cerebellar levels. The semi-circular canals respond to rotational acceleration of the head, this drives the VOR. Otoliths (saccule and utricle) are gravity receptors that respond to linear acceleration and static head tilt. The otoliths are responsible for ocular counter rolling (Figure 4.30).

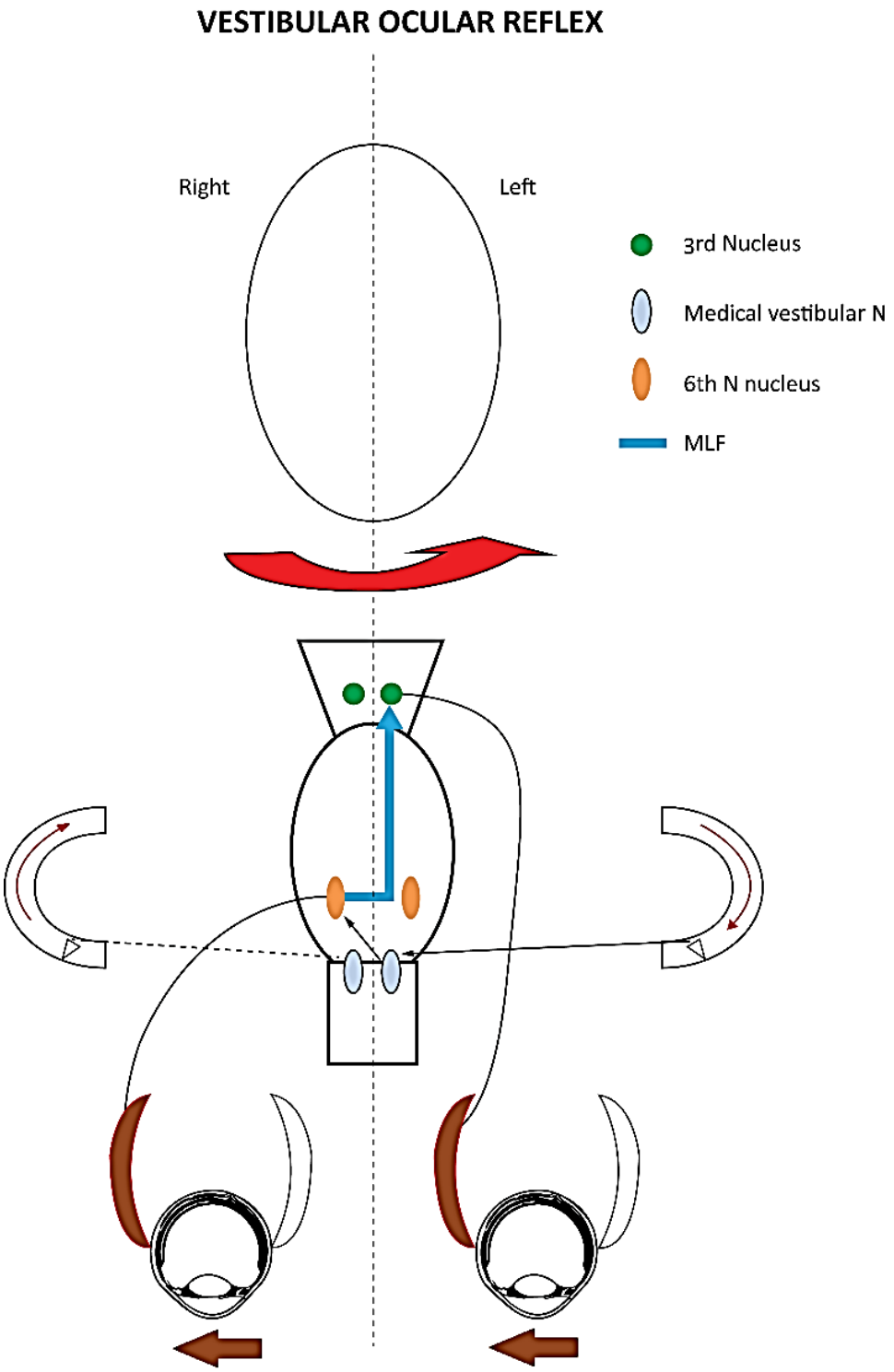


Figure 4.4: *Vestibular ocular reflex*

VESTIBULAR INFLUENCES ON EYE MOVEMENTS

With sharp head turning to the left, the eyes will continue to look straight ahead if they deviate to the same degree to the right. This is achieved by the forward shift of endolymph in the left lateral semi-circular canal (SCC) induced by head rotation to the left. The cupola in the left lateral SCC is stimulated, which in turn stimulates the vestibular nerve and then the medial vestibular nucleus (MVN). The MVN stimulates the opposite 6th nerve nucleus and via the medial longitudinal fasciculus (MLF), the ipsilateral 3rd nerve nucleus. The result is a deviation of both eyes to the right.

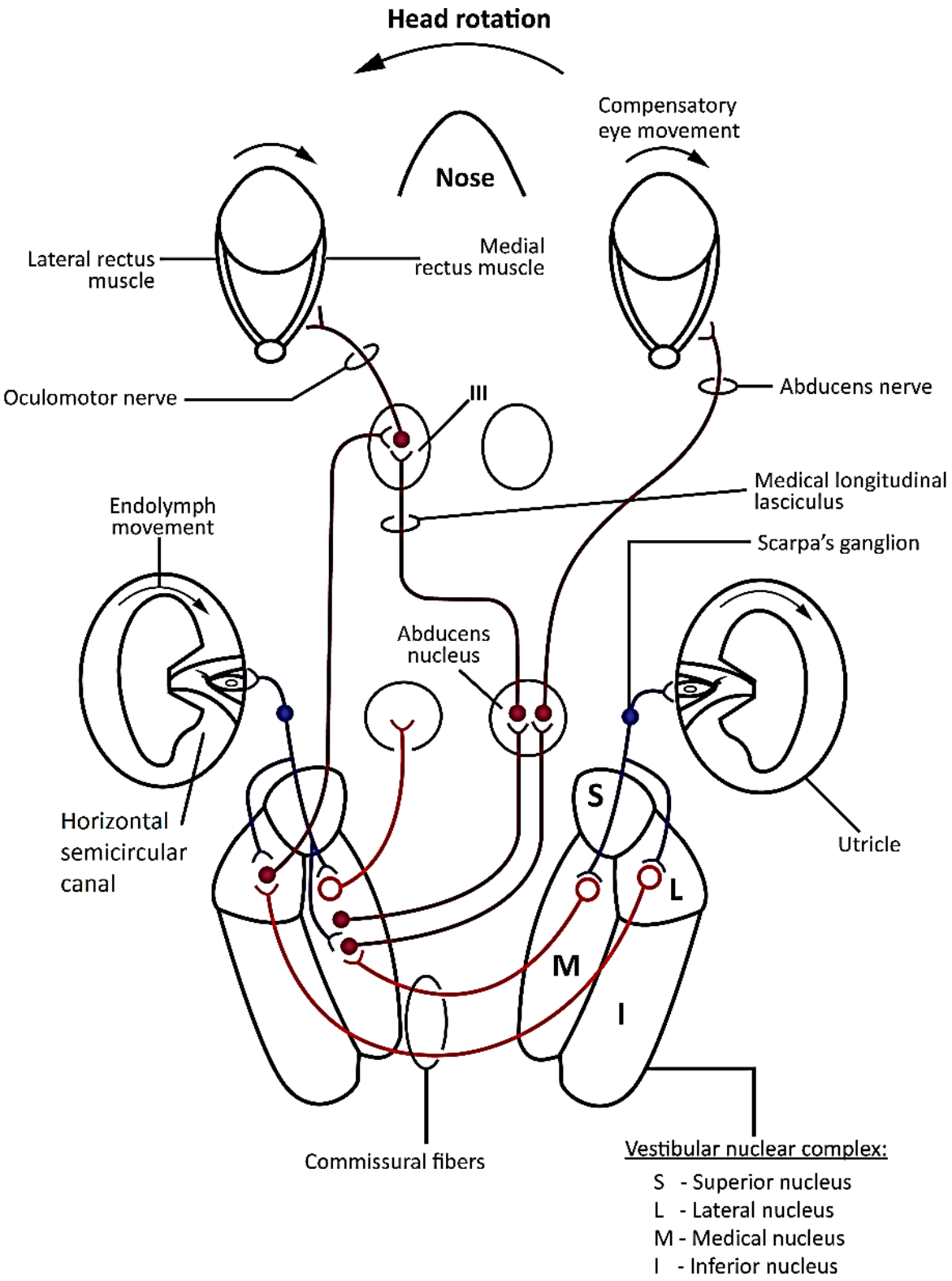


Figure 4.5: *Left head rotation results in equal amount of movement of the eyes to the right*

OPTOKINETIC SYSTEM

The optokinetic system (OKN) uses visual reference points in the environment to maintain orientation. It complements the VOR which becomes less responsive during slow or sustained head movements, such as in spinning. When the eyes reach their limit in the orbits, a reflex saccade allows refixation to a point in the direction of head rotation.

VERGENCE SYSTEM

The vergence system enables the disconjugate movement of the eyes (convergence or divergence) in the horizontal plane. It maintains binocular fixation on a target moving toward or away from the subject. This is essential for binocular single vision and stereoscopic depth perception.

Binocular fusion is necessary to maintain ocular alignment on an approaching or retreating object. Electromyography shows that divergence is an active movement but not as dynamic or under voluntary control as convergence. The driving stimulus for vergence movements is retinal blur (unfocused) and fusional disparity (diplopia).
Vergence is relayed via the occipital cortex. The precise location of the convergence and divergence centers is unknown. Two areas, namely the midbrain pretectum and nucleus reticularis tegmenti pontis (NRTP) are important. Lesions of the pretectum cause vergence abnormalities. The NRTP functions as a Vergence Integrator. Lesions of the NRTP in monkeys cause sustained convergence or pendular convergence-divergence oscillations. The occipito-mesencephalic pathway runs more ventrally than does the light reflex pathway. It is less susceptible to extrinsic compression.

VISUAL FIXATION

The rostral portion of the superior colliculus has a representation of the fovea referred to as the 'fixation zone.'
Neurons inhibit movement-related neurons in the caudal parts of the superior colliculus and project to the nucleus of the dorsal raphe to inhibit saccades.
With lesions within the superior colliculus, the patient is more likely to make saccades to distracting stimuli.

CEREBELLAR INFLUENCES ON EYE MOVEMENTS

The cerebellum coordinates the ocular motor system to drive the eyes smoothly and accurately. The flocculo-nodular lobe forms a direct connection with the eye muscle nuclei and connects to the opposite 3rd nerve nucleus and to ipsilateral 6th nerve nucleus via the MLF and therefore moves the eyes to the same side. Lesions in this area prevent smooth tracking and cause horizontal nystagmus with a downward component. The dorsal vermis and fastigial nuclei determine accuracy of the saccadic amplitude. Lesions result in saccadic dysmetria.

CEREBELLAR INFLUENCE

The flocculus matches the saccadic pulse and step. It stabilizes images on the fovea. Lesions of the flocculus cause gaze-evoked nystagmus, rebound and downbeat nystagmus, impair smooth pursuit and impair suppression of VOR during fixation. The nodulus influences vestibular eye movements. Lesions produce periodic alternating nystagmus.

VERTICAL EYE MOVEMENTS

Premotor substrate for vertical gaze lies in the Midbrain Reticular formation. Some vertical saccades are programmed in the PPRF and an alternate MLF pathway. The rostral interstitial nucleus of the MLF (riMLF) contains excitatory burst neurons (EBN) for both upgaze and downgaze.
EBN's for upward saccades project dorsally and laterally from riMLF. They decussate in the posterior commissure and innervate both ipsilateral and contralateral 3rd and 4th nerve nuclei.

Downward saccades: EBNs project dorsomedially and caudally to innervate the 3rd and 4th nerve nuclei bilaterally.
Vertical saccades require bilateral supranuclear innervation. The neural integrator for vertical and torsional movements is located in the interstitial nucleus of Cajal (INC). Burst-tonic and tonic neurons lie in INC.
Retinal slip is the sensory stimulus for vertical pursuit. The information is encoded by the dorsolateral pontine nuclei and relayed to the flocculus and posterior vermis, thereafter converges on the INC on the midbrain and finally onto the relevant ocular motor nuclei.

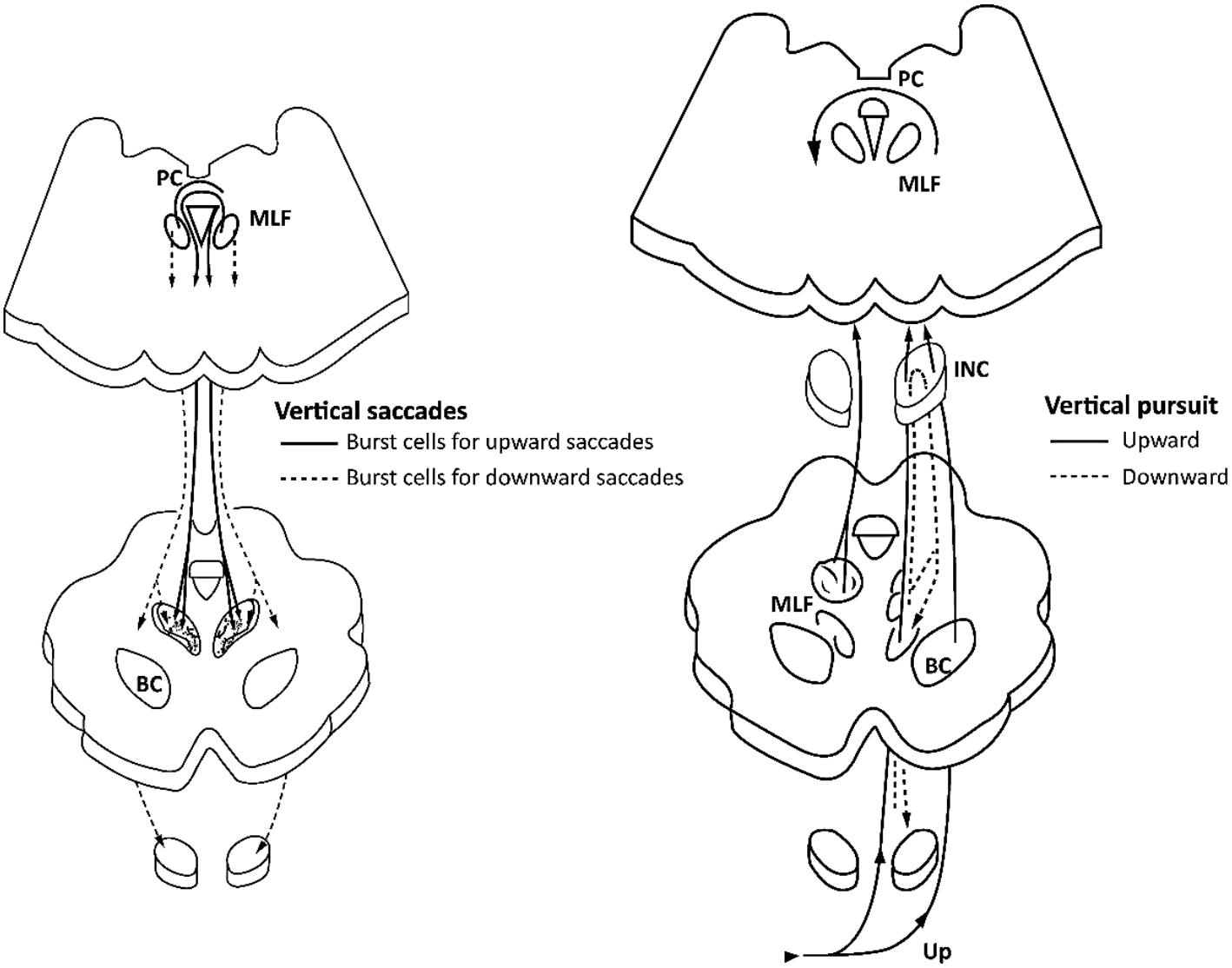


***Figure 4.6**: Bilateral innervation to the ocular motor nuclei for vertical control of eye movements*

Pearl:

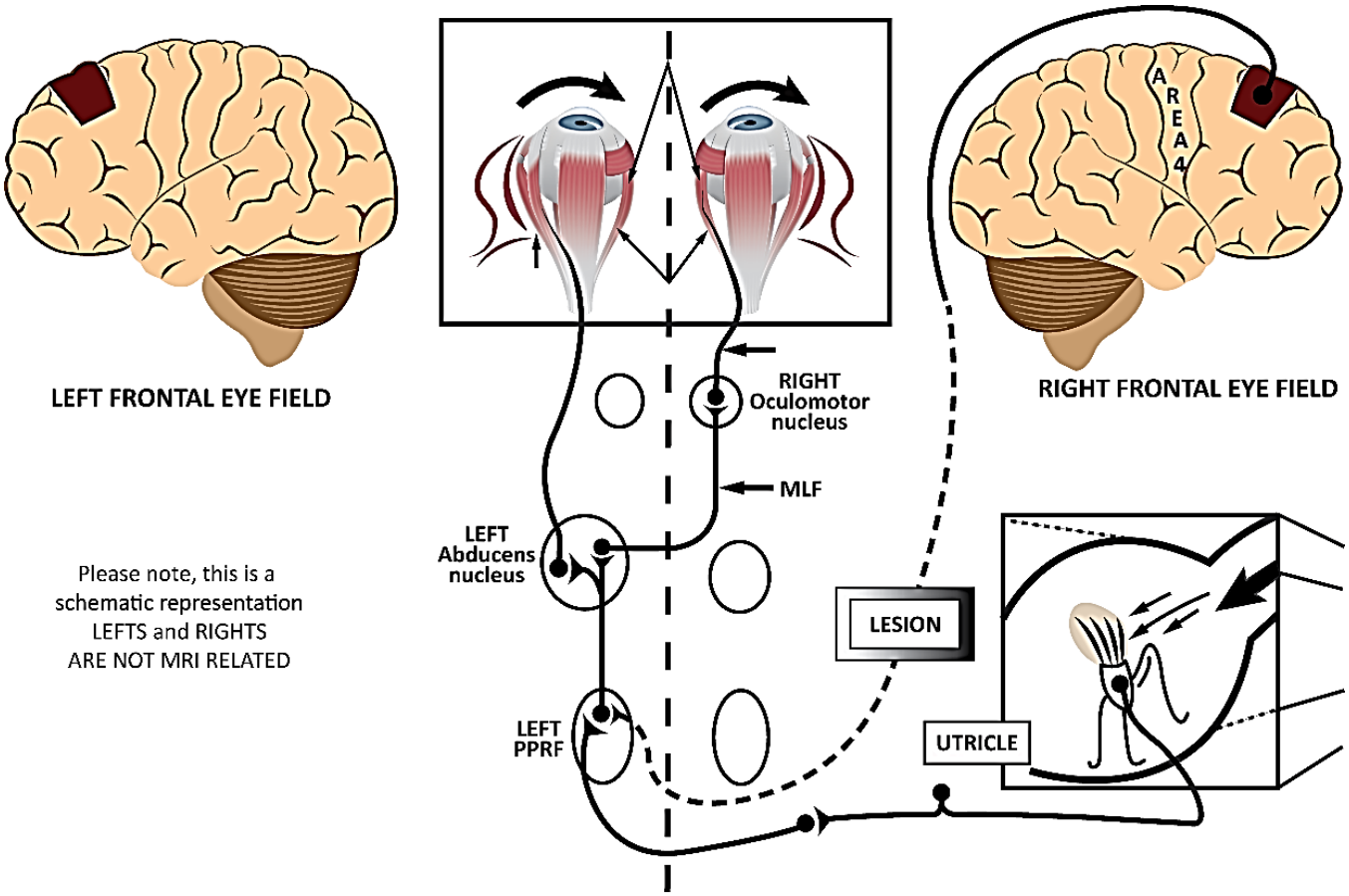
Tools for the hunt: The lion can use the various eye movements to catch its prey:

- Saccadic movement – target the prey
- Pursuit movement - follow the prey
- Vergence system - depth perception
- VOR – fixate on the prey despite rapid head movement
- OKN – slow or sustained head rotation – which allows refixation of target

DISORDERS

LESIONS IN FRONTAL CORTEX (FRONTAL EYE FIELD) AREA 8

- **Overactivity** results in seizures. There is deviation of the eyes to the opposite side.
- **Underactivity** results in gaze paresis to the opposite side



Lesion is the RIGHT frontal eye field - tonic deviation of the eyes to the RIGHT (LEFT side "driving"), No atrophy or diplopia

Figure 4.7: *Right frontal lobe lesion*

LESIONS IN THE PARIETO-OCCIPITAL CORTEX

- **Overactivity** results in seizures. There is a gaze deviation to the opposite side.
- **Underactivity** results in the loss of optokinetic nystagmus

BASAL GANGLIA

- **Overactivity** results in oculogyric crisis
- **Underactivity** results in an impaired vertical gaze

COLLICULAR AREA LESIONS OF THE SUPERIOR COLLICULUS

Parinaud's Syndrome:

Signs include:

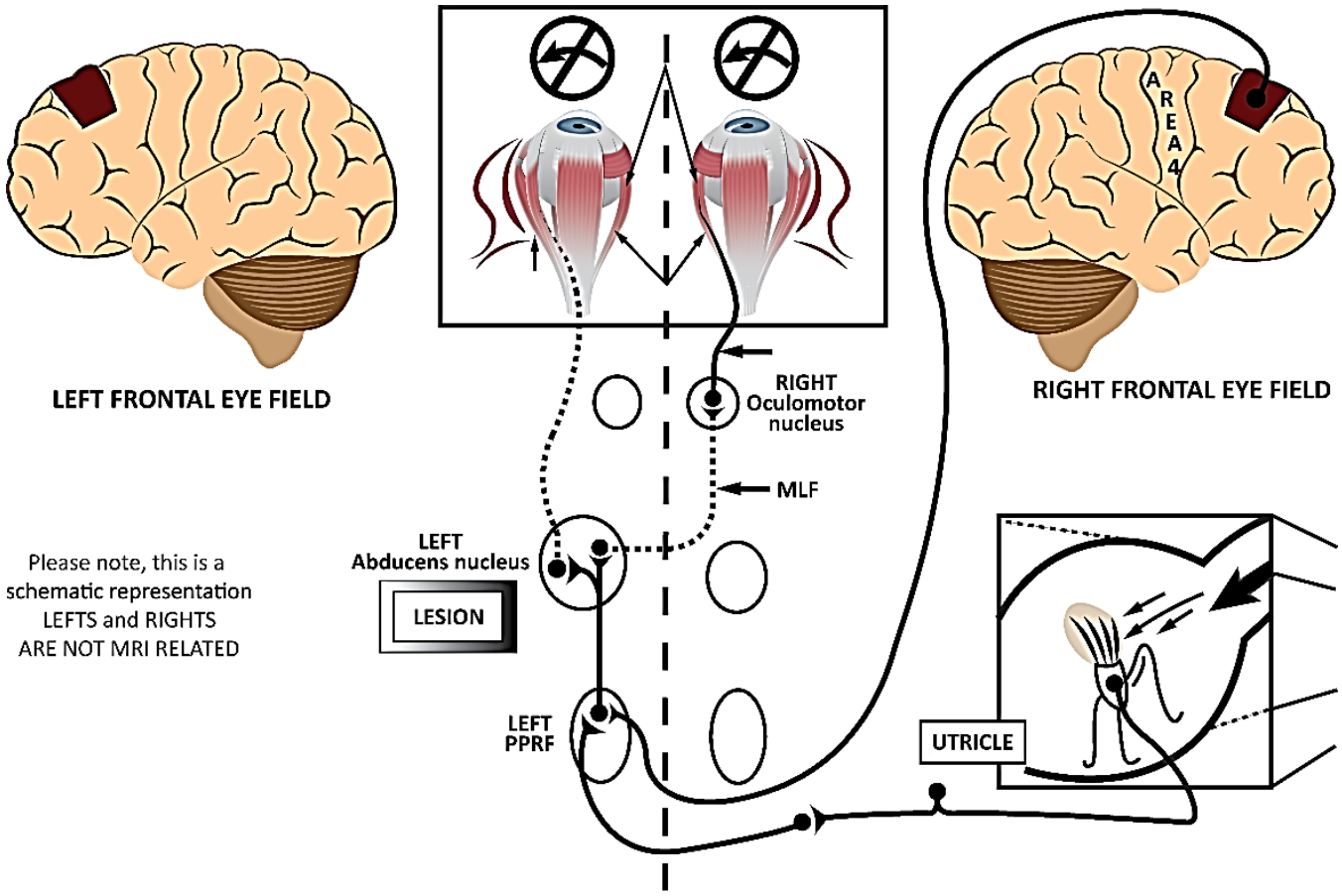
- Pupillary Dilatation
- Loss of accommodation reflex
- Lid retraction
- Lid Lag
- Light/Near Dissociation
- Retraction – convergence Nystagmus
- Skew deviation
- Normal downgaze and eyes straight in the primary position

LESIONS OF THE INFERIOR COLLICULUS:

- Loss of downward gaze
- Loss of Convergence, (Normal light reflex)
- Loss of accommodation of pupil
- Skew deviation

LESIONS OF THE PONTINE AREA

- A lesion in the PPRF results in inability to look to the side of the lesion
- There is a horizontal gaze palsy
- The eyes are deviated towards the hemiplegic side



Lesion in the LEFT abducens nucleus = inability to voluntarily move both eyes past the midline to the LEFT (atrophy of the LEFT lateral rectus but not the RIGHT medial rectus), Diplopia

***Figure 4.9**: Left pontine lesion*

INTERNUCLEAR OPHTHALMOPLEGIA

Internuclear ophthalmoplegia (INO) occurs due to a lesion in the medial longitudinal fasciculus (MLF), pons, midbrain or cerebellum. Clinical signs of an INO include:

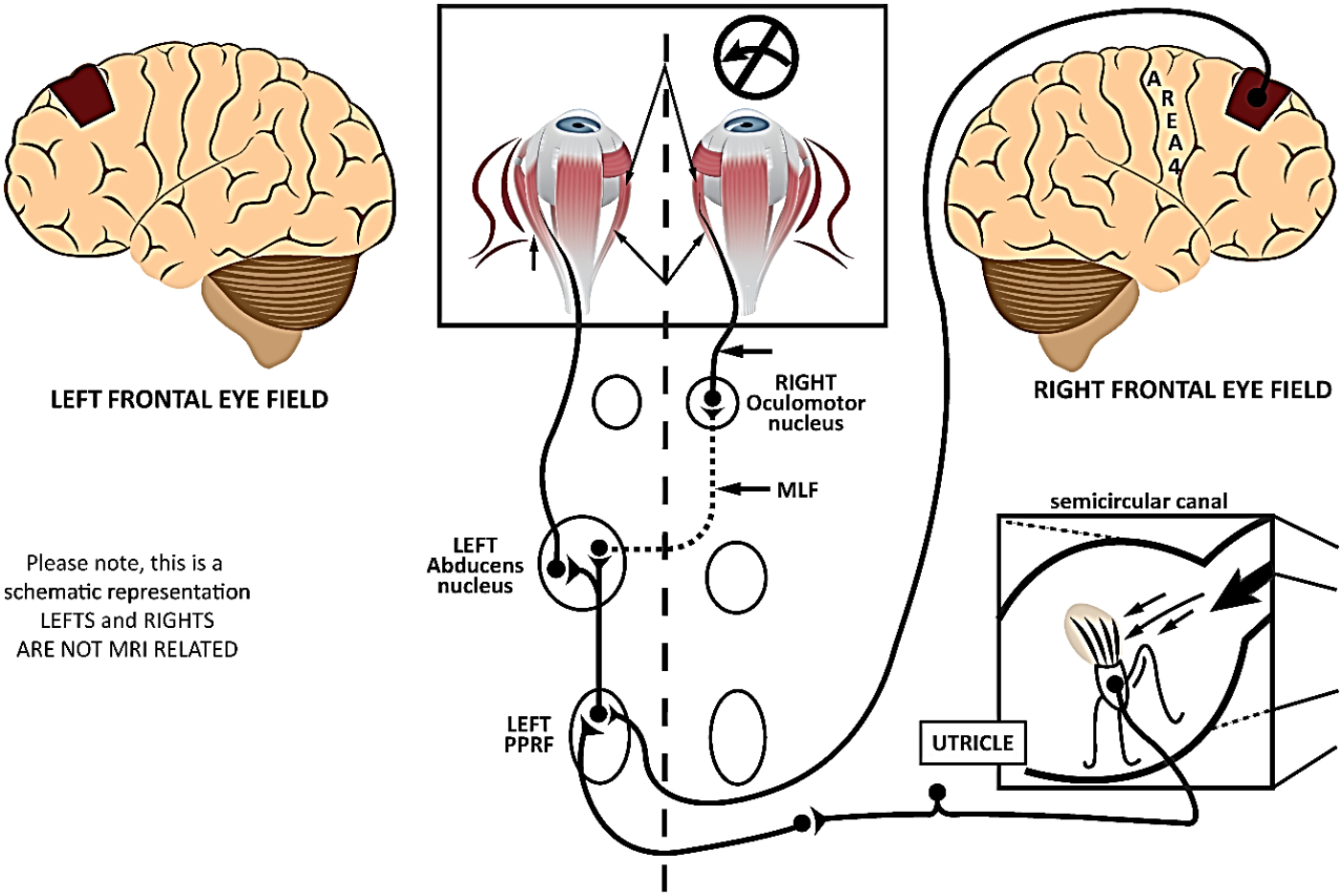
- Medial rectus paresis
- Exo-deviation
- Skew deviation
- Failure of adduction – ipsilaterally
- Nystagmus of abducting eye – contralaterally

INO occurs unilaterally in younger patients and in patients with MS. In older patients and patients with systemic vascular disease, it may occur bilaterally.

Causes of INO may also include:

- Demyelination
- Vascular disease
- Tumors of the brain stem and fourth ventricle
- Remote effects of carcinoma
- Drug-induced

- Encephalitis
- Hydrocephalus
- Trauma
- Progressive supranuclear palsy



Lesion of the Right MLF = inability to turn the RIGHT eye past the midline to the LEFT = INTERNUCLEAR OPTHALMOPLEGIA (INO), No atrophy of the RIGHT medial rectus, Diplopia when attempting to look LEFT of the midline, also there is nystagmus of the LEFT eye

***Figure 4.10**: Right MLF lesion*