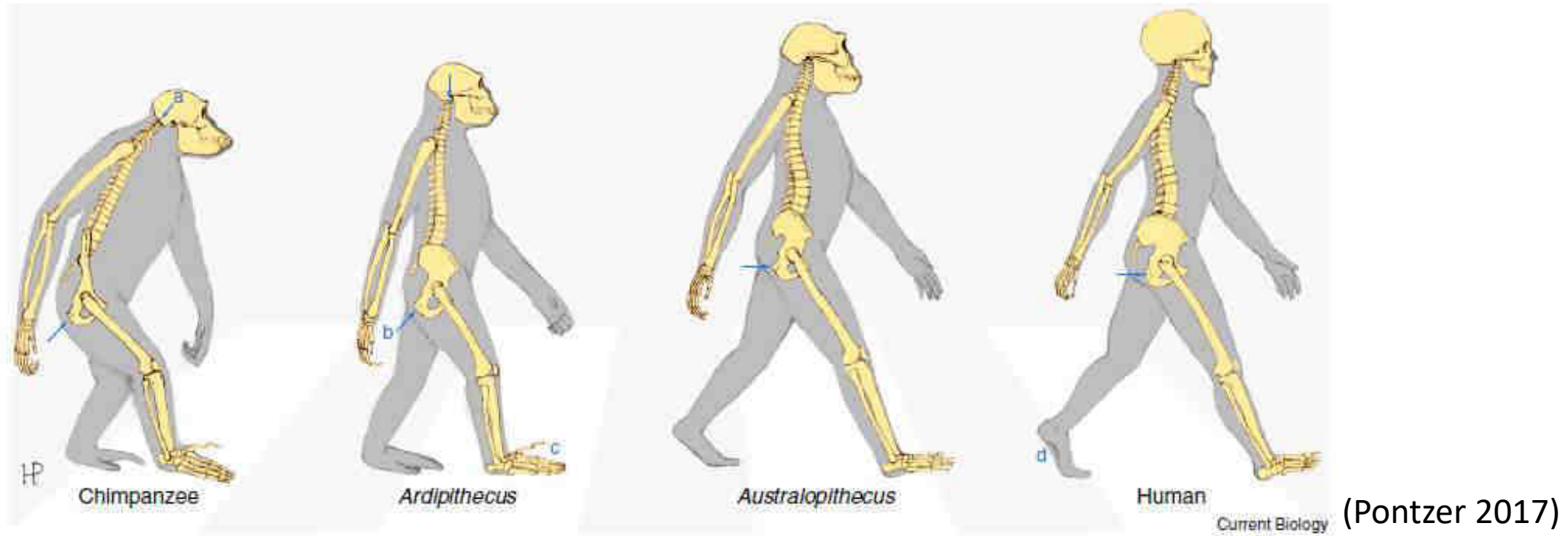




Maintaining Balance

Dr Abhijit Das MD, DM
AMRI Mukundapur, Kolkata



Human Bipedalism – a unique evolutionary feet

- Economy of energy expenditure
- Freeing of upper limbs
- Transient unstable erect positions (dance, sports etc)

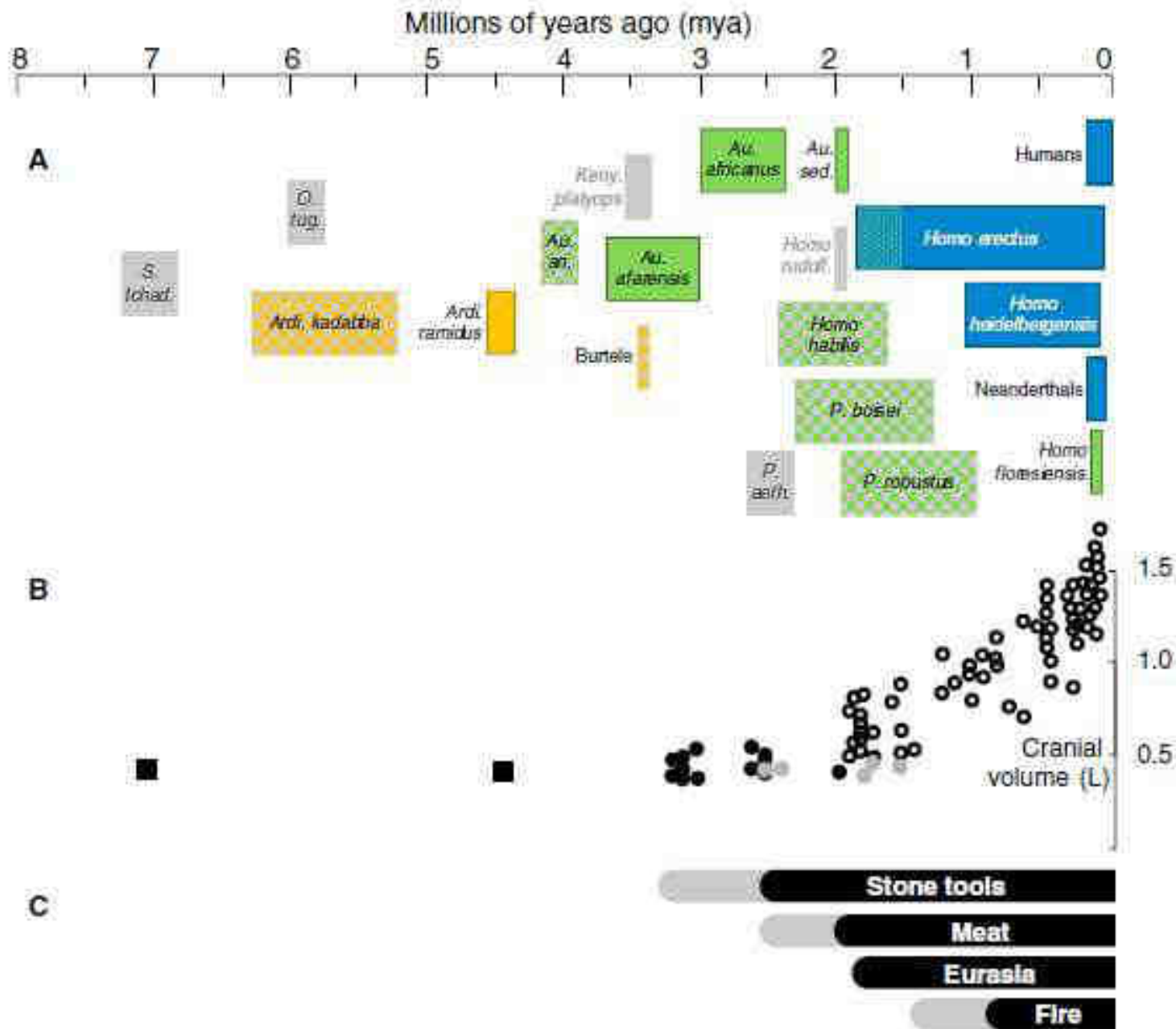


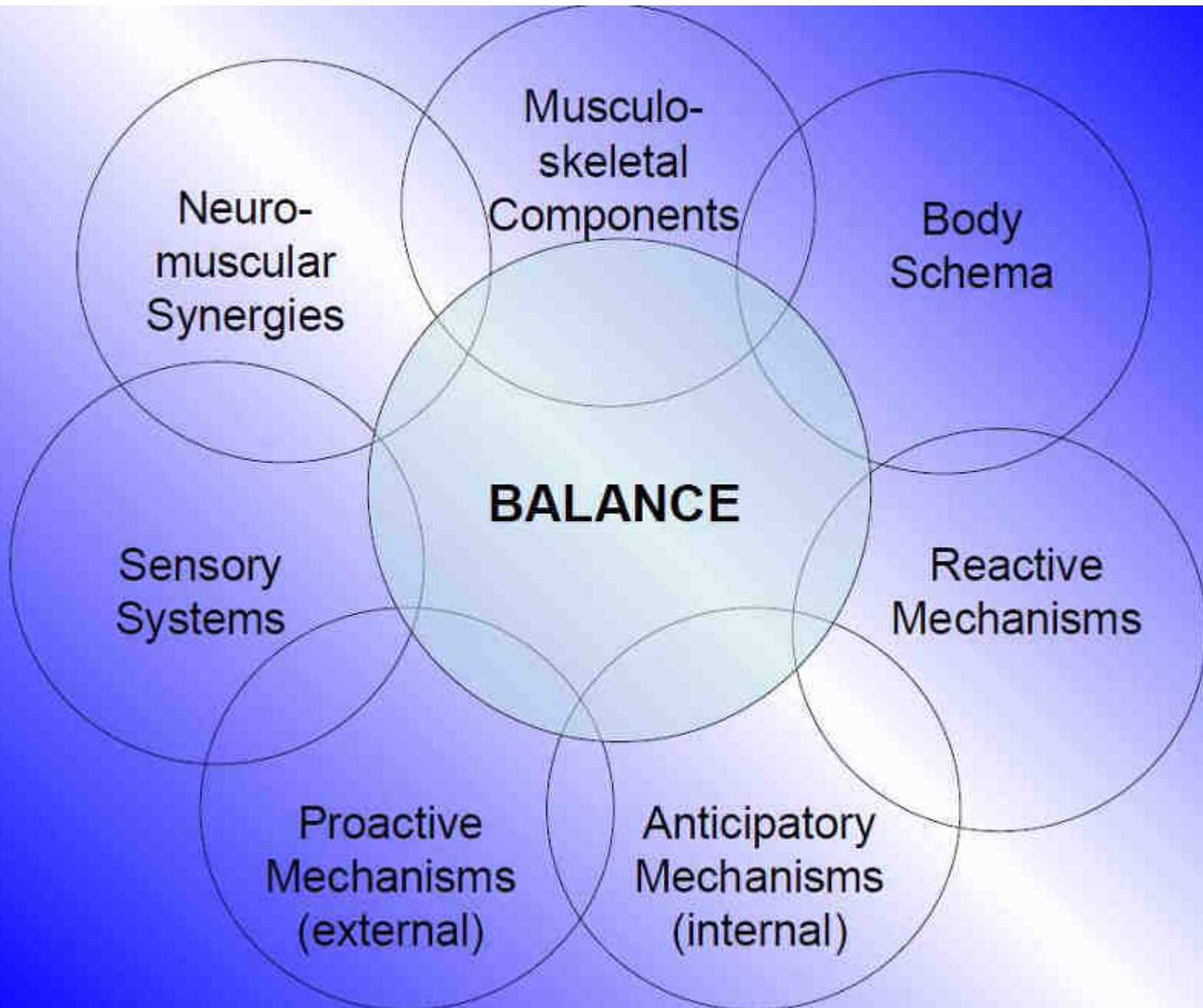
Figure 3. Hominin evolution overview.

(A) Hominin species age ranges [41] and their inferred locomotor capabilities. Yellow: poor economy and low endurance. Green: good economy but limited endurance. Blue: human-like economy and endurance. Hatched: economy and endurance inferred from limited evidence. Gray: insufficient fossil evidence to determine economy or endurance. See text. (B) Brain sizes (cranial volumes) increase beginning ~2 mya. Open circles: *Homo*; closed circles: *Australopithecus*; gray circles: *Paranthropus*; squares: other. Redrawn from [12,44,88]. (C) Earliest proposed appearance (gray) and widespread evidence (black) for stone tool use [98], butchery of game animals (meat) [86,87], expansion into Eurasia [51], and the control and use of fire [99].

A strong co-evolution

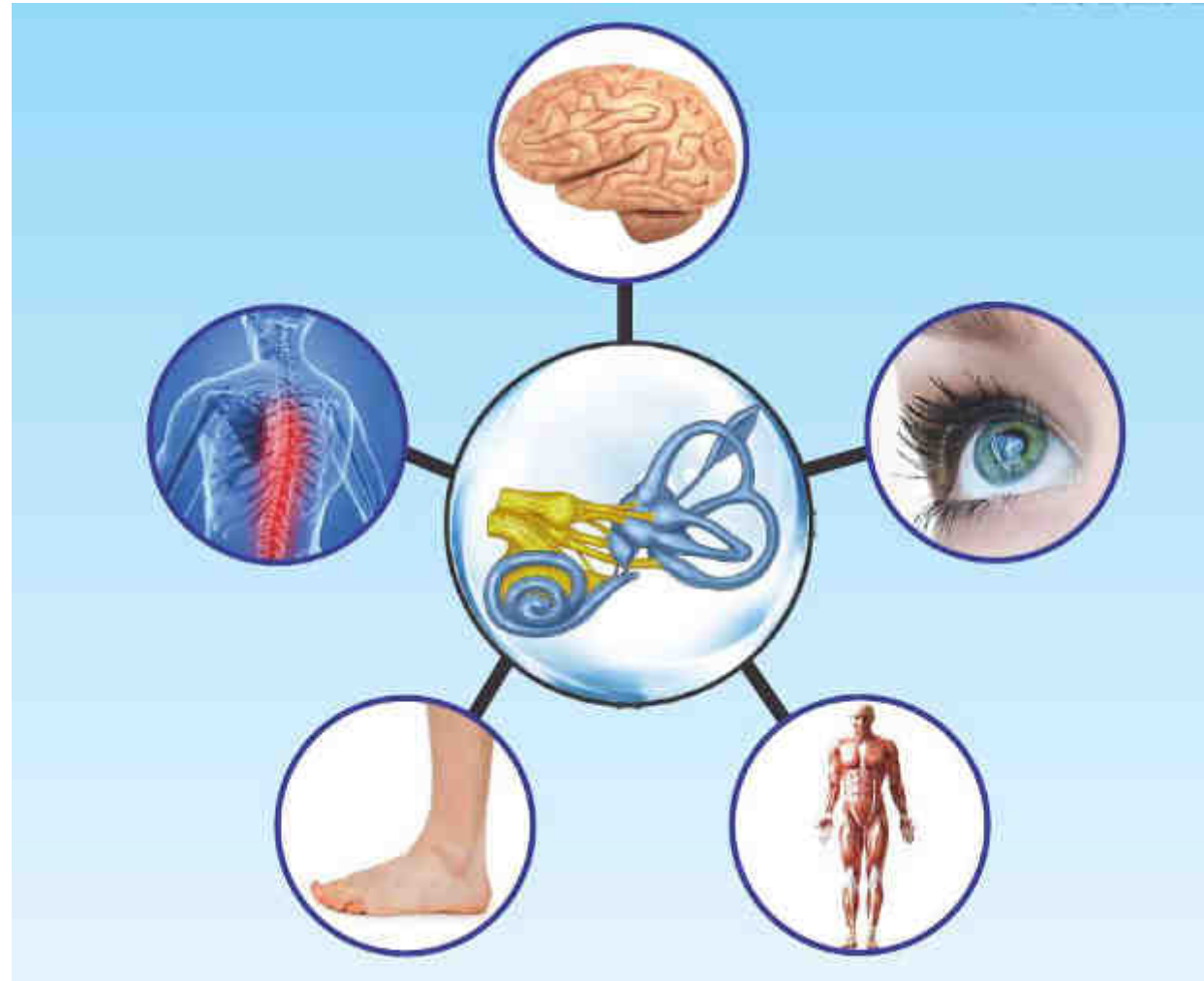
- Balance
- Cognition
- Emotion
- Upper limb function





Maintaining balance
is a major
Physiological function

The Human Balance System

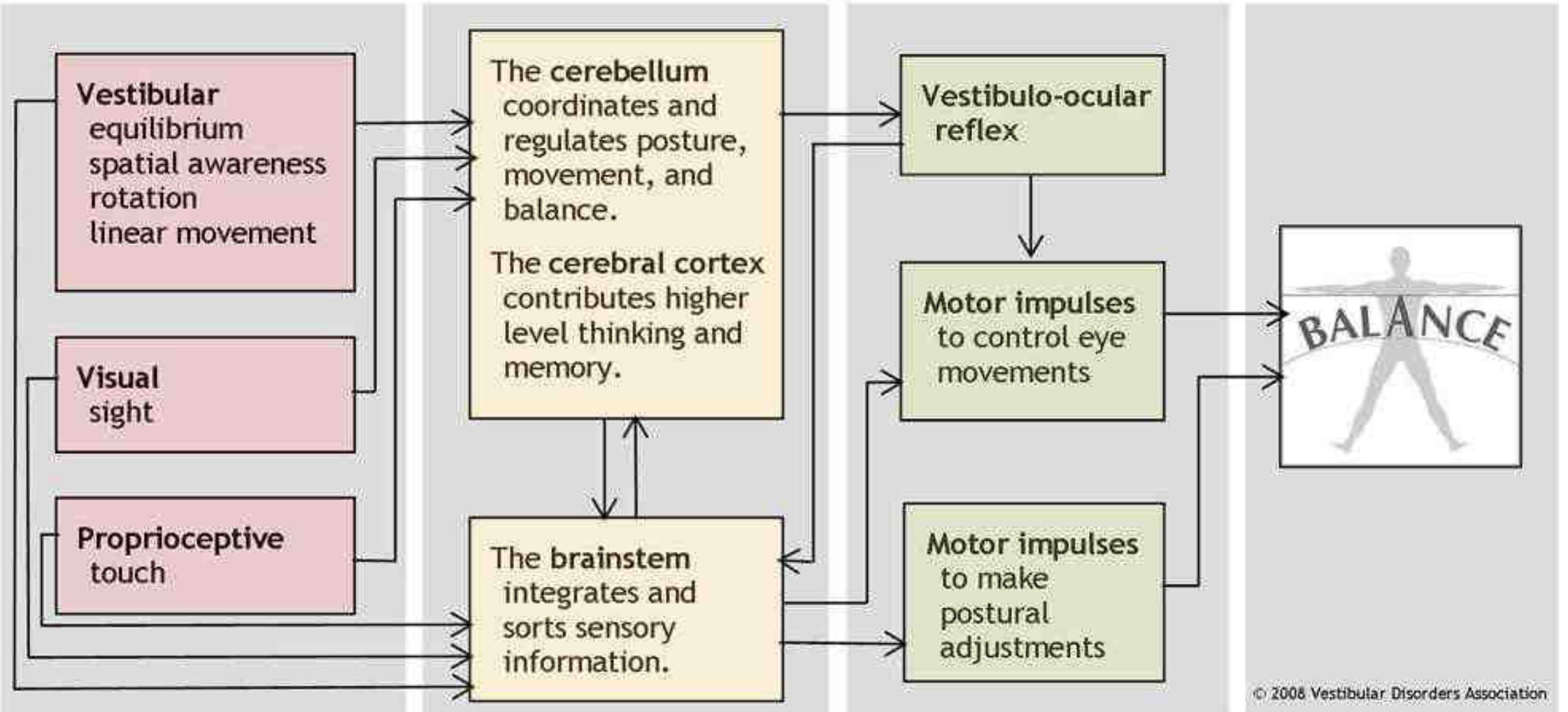


SENSORY INPUT

INTEGRATION OF INPUT

MOTOR OUTPUT

BALANCE



Maintenance of Balance

CNS collects information about static/ dynamic position of the body in relation to the ground and the surroundings from certain sensors in different parts of the body



Information from different sensors integrated in the brain and compared with previously stored experiences



A very precise, coordinated and accurately timed *motor output generated reflexly* which contracts some specified muscles and maintains or restores balance

Physiology of Balance

Afferent **SENSORY** system
(input to the brain from):

Vestibular Labyrinth



Eyes



Proprioreceptors



Efferent **MOTOR** system (output
generated by the brain to):

Muscles of **LIMBS/TRUNK/NECK** through
VESTIBULO-SPINAL reflex
system.



Muscles of the **EYES**
through
VESTIBULO-OCULAR
reflex system.

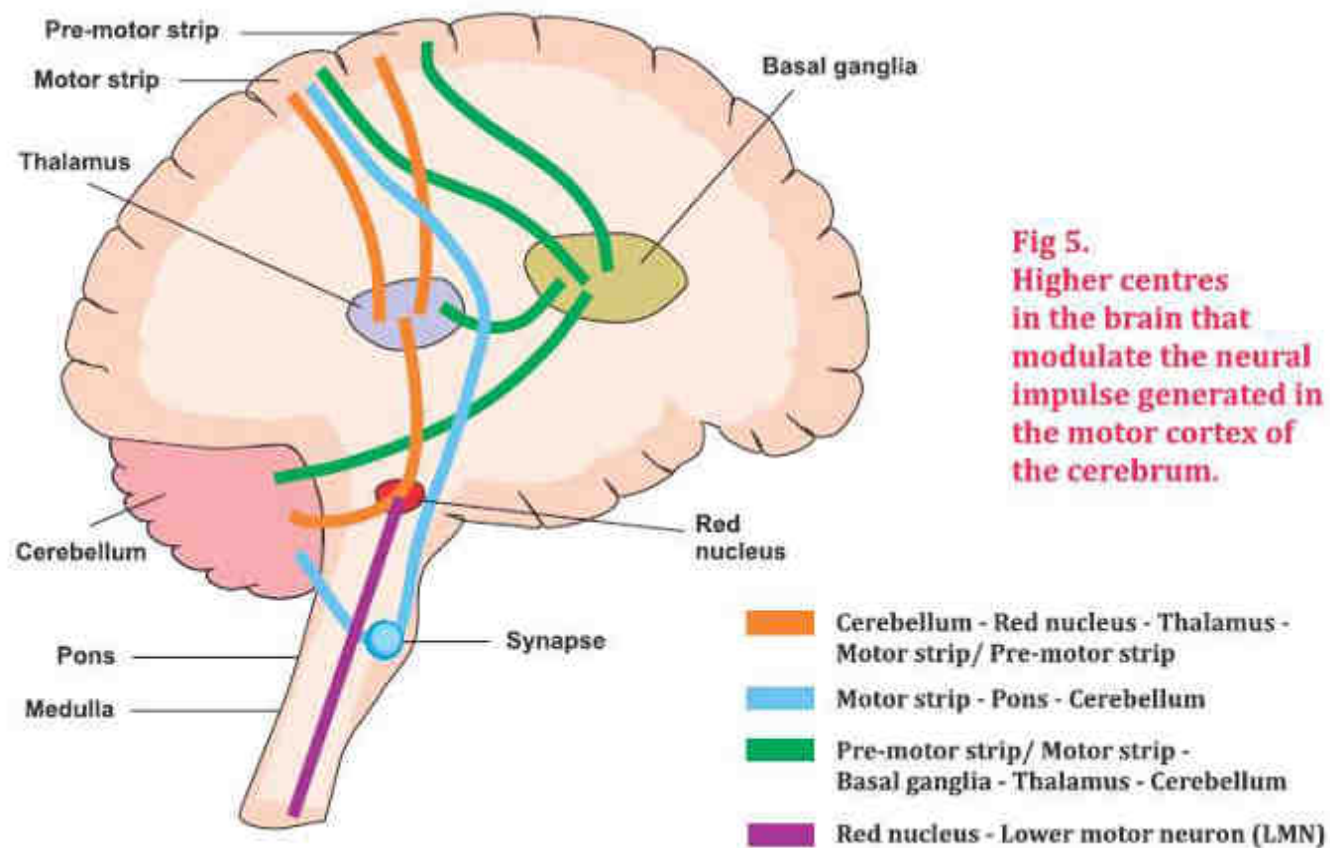


- *Cerebellum fine tunes the motor output*
- *Cognitive system determines the nature of the response*
- *Higher centers in the brain modulate the motor response*

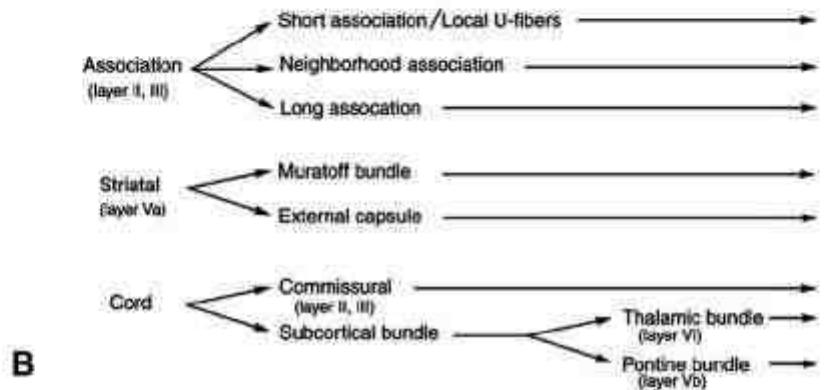
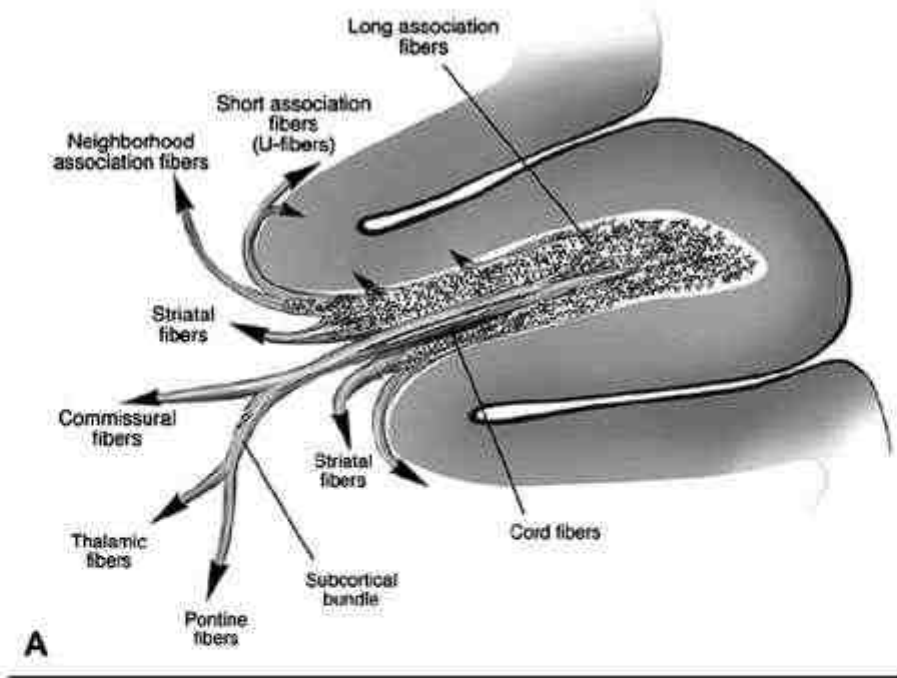
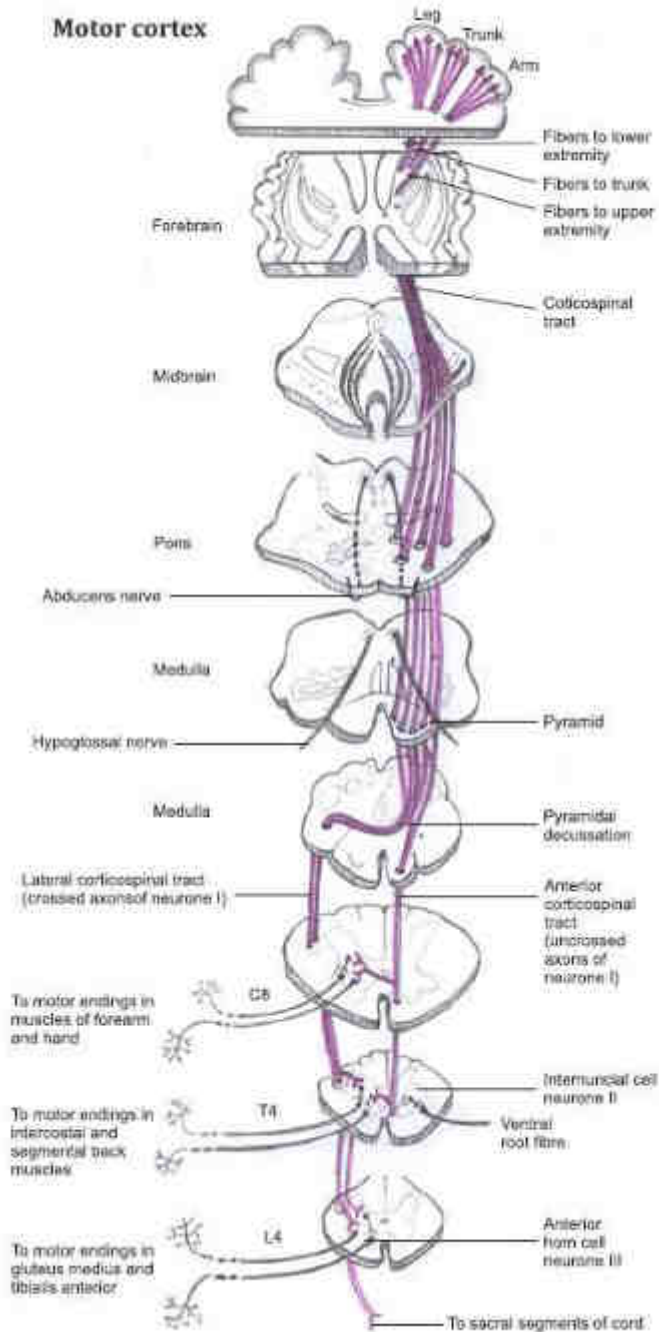
VESTIBULAR CONNECTIONS AND PATHWAYS

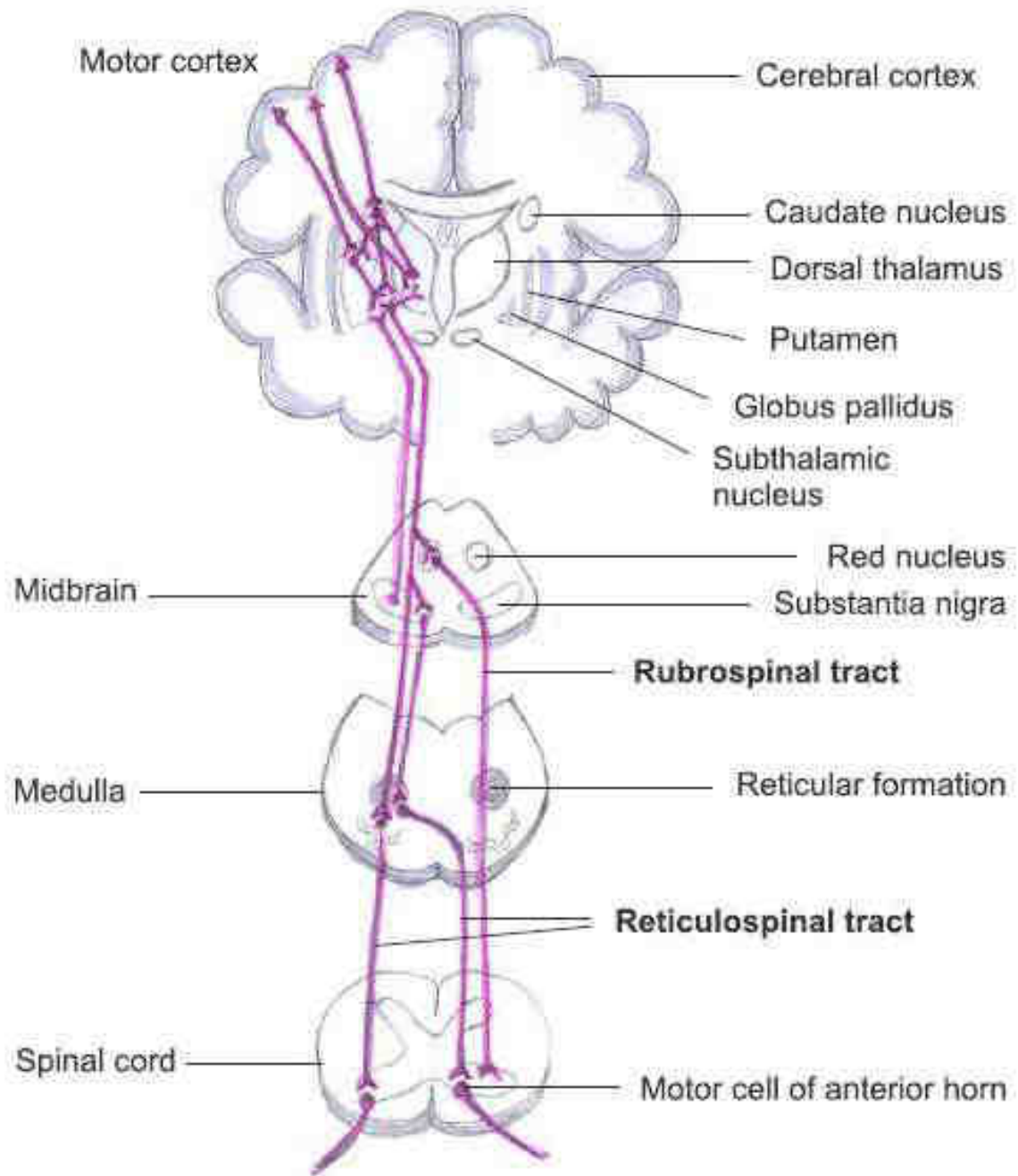
Vestibulo-spinal Pathways	Vestibulo-cerebellar Pathways	Vestibulo-cerebral Pathway	Vestibulo-autonomic Pathway
<p>Medial vestibulospinal pathway: Efferent medial vestibular nucleus fibers → Descend in MLF → Become medial vestibulospinal tract → Cervical and upper thoracic motor nuclei</p> <p>Function: Stabilize Head and neck posture.</p> <p>Lateral vestibulospinal pathway: Efferent lateral vestibular nucleus fibers → Descend on anterior horn of spinal cord as lateral vestibulospinal tract up to cervical region only.</p> <p>Function: Maintain the tone of the antigravity muscles of the fore-limb and thereby maintain the forelimb anti-gravity posture.</p>	<p>Involved part of cerebellum: Midline cerebellum (Archicerebellum)</p> <p>Direct vestibulo-cerebellar tract: Vestibular labyrinth → Fibers directly to vermis in the midline cerebellum</p> <p>Indirect vestibulo-cerebellar tract: Vestibular labyrinth → Inferior vestibular nucleus → Ipsilateral inferior cerebellar peduncle → Uvula and flocculonodular lobe in midline cerebellum</p> <p>Midline cerebellum → Efferent fibers → Bilateral vestibular nucleus complex</p>	<p>Efferent vestibular projections to bilateral Ventral Posterior group of thalamus.</p> <p>Cortical regions of the brain known to be involved with vestibular processing:</p> <ol style="list-style-type: none"> 1. Frontal eye fields: Control eye movements and receive vestibular motion information 2. Primary somatosensory cortex (Areas 2v and 3a): Map body location and movement signals 3. PIVC (Parieto-Insular Vestibular Cortex): Responds to body and head motion information 4. Posterior parietal cortex: Motion perception and responds to both visual and vestibular motion cues 5. Hippocampus and parahippocampul regions: spatial orientation and navigation functions 	<p>Some vestibular efferent projections to reticular formation, dorsal pontine nuclei, and nucleus of solitary tract.</p> <p>Function: Stabilize respiration and blood pressure during body motion and changes relative to gravity. The saccule senses up-down movement i.e., movement away from and towards gravity e.g., when we suddenly stand up from the sitting posture and this information has to be fed to the baroreceptors to control blood-pressure accordingly. They also have a role to play in motion sickness.</p>

The Efferent System



Pyramidal System





Extrapyramidal System

The Connectors

The Long Association Fibers

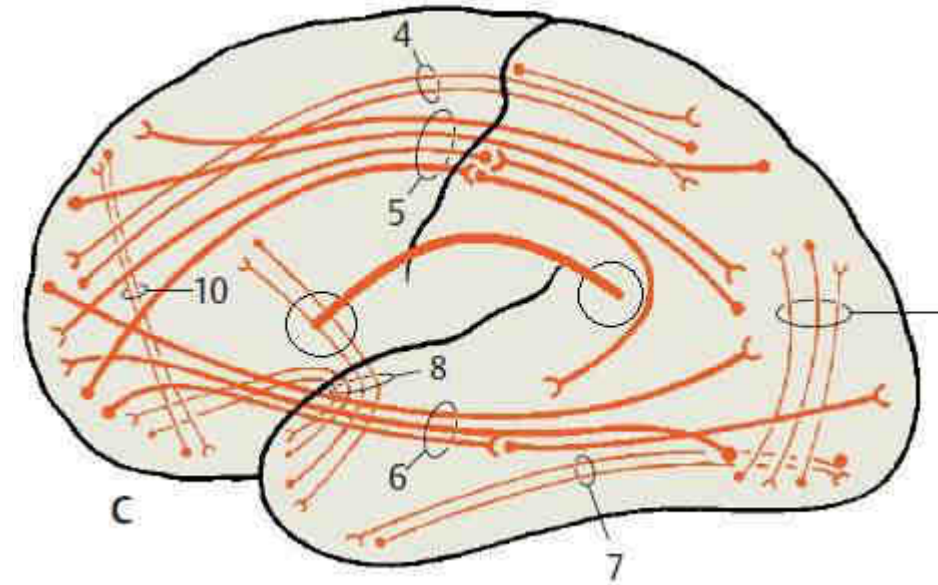


Table 1 - Identity and location of long association fibers of the cerebral hemisphere

Long association fiber pathway	Location in cerebral white matter
Superior longitudinal fasciculus I	White matter of superior parietal lobule and superior frontal gyrus
Superior longitudinal fasciculus II	Centrum semiovale, lateral to and crossing through the corona radiata, above Sylvian fissure
Superior longitudinal fasciculus III	White matter of the parietal and frontal opercula
Arcuate fasciculus	White matter of superior temporal gyrus, and deep to upper shoulder of the Sylvian fissure, ventrally adjacent to SLF II
Middle longitudinal fasciculus	White matter of caudal inferior parietal lobule extending into white matter of the superior temporal gyrus
Extreme capsule	Between claustrum and insula caudally, and between claustrum and orbital frontal cortex rostrally
Inferior longitudinal fasciculus	Vertical limb between sagittal stratum medially and parieto-occipital and temporal cortices laterally. Horizontal component in the temporal lobe
Fronto-occipital fasciculus	Above body and head of the caudate nucleus and subcallosal fascicle of Muratoff, lateral to corpus callosum, medial to corona radiata
Uncinate fasciculus	White matter of rostral temporal lobe, limen insula, white matter of orbital and medial prefrontal cortex
Cingulum bundle	Dorsal component in white matter of the cingulate gyrus. Ventral contingent in white matter of the caudal part of parahippocampal gyrus

The Afferent System

Beyond Vestibular Nucleus

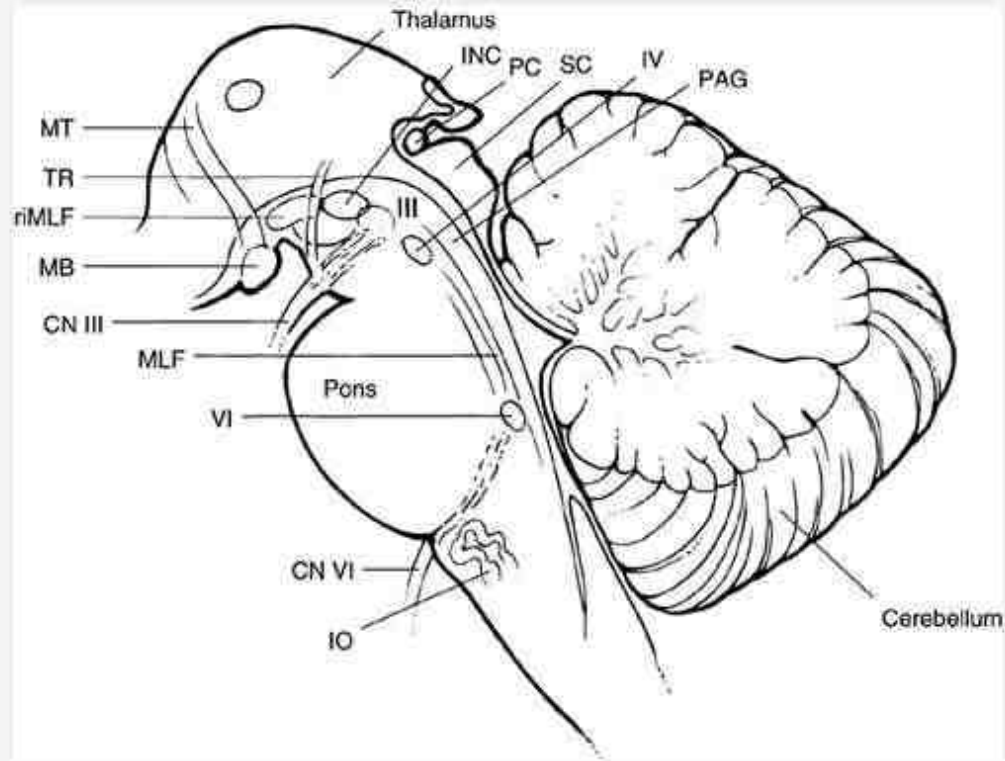


FIGURE 8-3 Sagittal section through the brainstem showing structures that play an important role in the motor control of eye movements. *III* = nucleus of cranial nerve III; *IV* = nucleus of cranial nerve IV; *VI* = nucleus of cranial nerve VI; *PAG* = periaqueductal gray; *SC* = superior colliculus; *PC* = posterior commissure; *INC* = interstitial nucleus of Cajal; *TR* = tractus retroflexus; *riMLF* = rostral interstitial nucleus of the medial longitudinal fasciculus; *MB* = mammillary bodies; *CN III* = cranial nerve III; *CN VI* = cranial nerve VI; *MLF* = medial longitudinal fasciculus; *IO* = inferior olive 6; *MT* = mammillothalamic fibers.

Supranuclear OCULOMOTOR centers in the brain

Important Supranuclear OCULOMOTOR centers in the brain are:

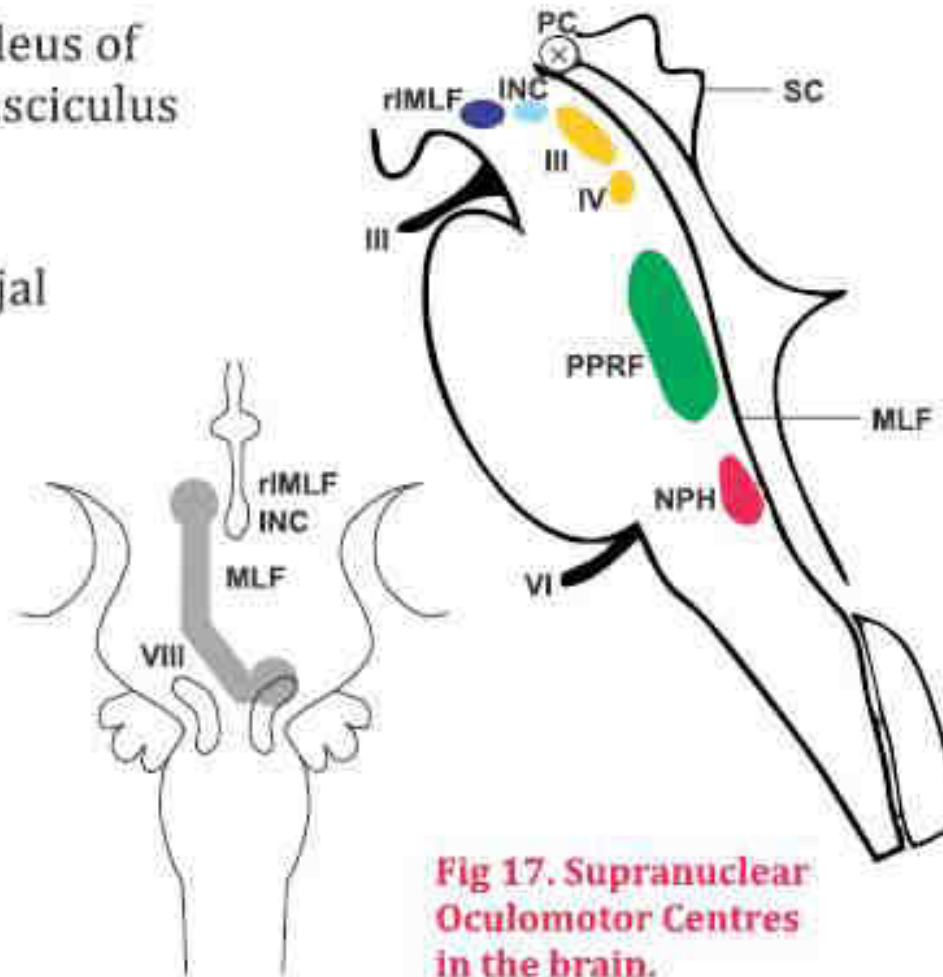
rMLF: Rostral interstitial nucleus of
medial longitudinal fasciculus
(upper midbrain)

INC: Interstitial nucleus of Cajal
(upper midbrain)

PPRF: Paramedian Pontine
Reticular Formation

NPH: Nucleus Praepositus
Hypoglossi

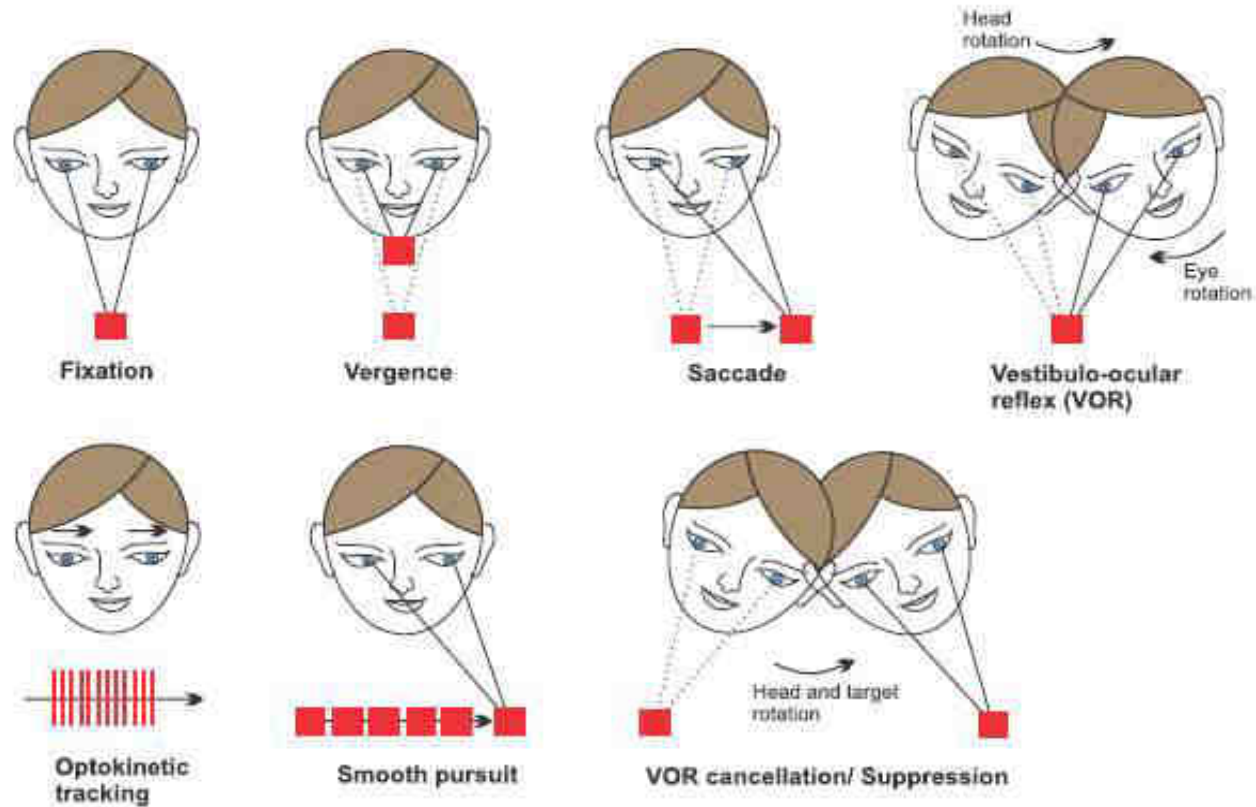
PC: Posterior Commissure
(connects the
rMLF of 2 sides)



**Fig 17. Supranuclear
Oculomotor Centres
in the brain.**

Supranuclear Oculomotor Mechanisms

- 1) *Smooth pursuit system*
- 2) *Saccadic system*
- 3) *Convergence system*
- 4) *Visual fixation with gaze holding system*
- 5) *VOR and suppression of VOR by visual fixation*
- 6) *Optokinetic system*



Vestibulo-Ocular Reflex

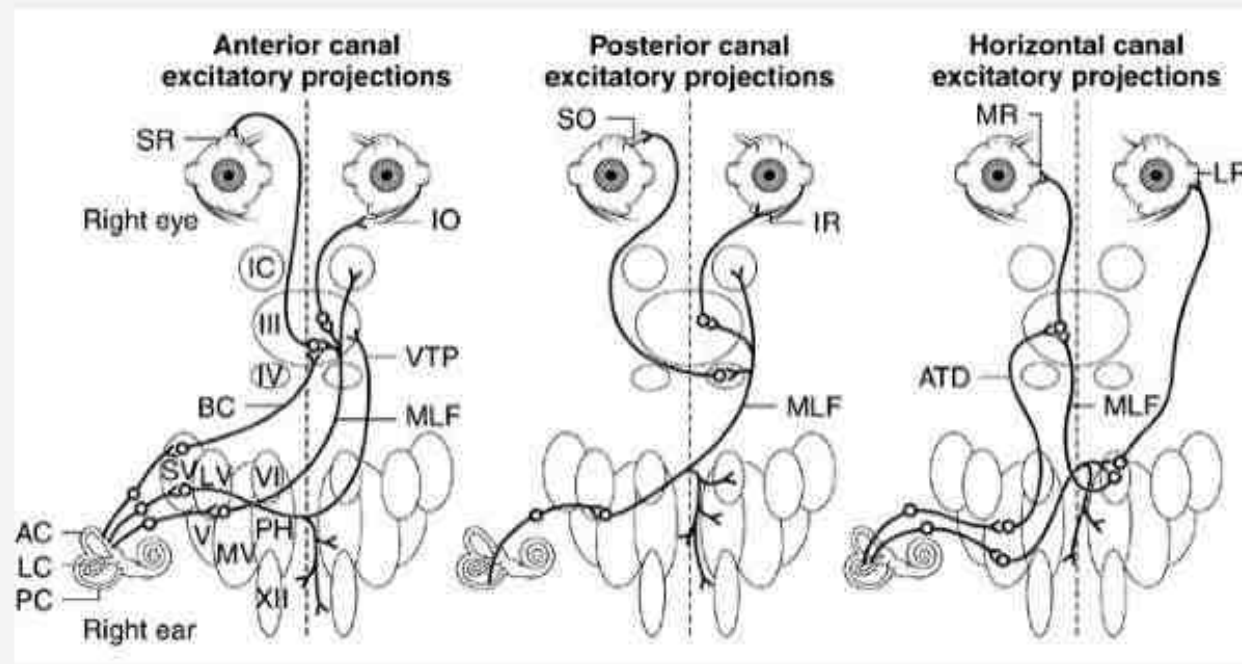
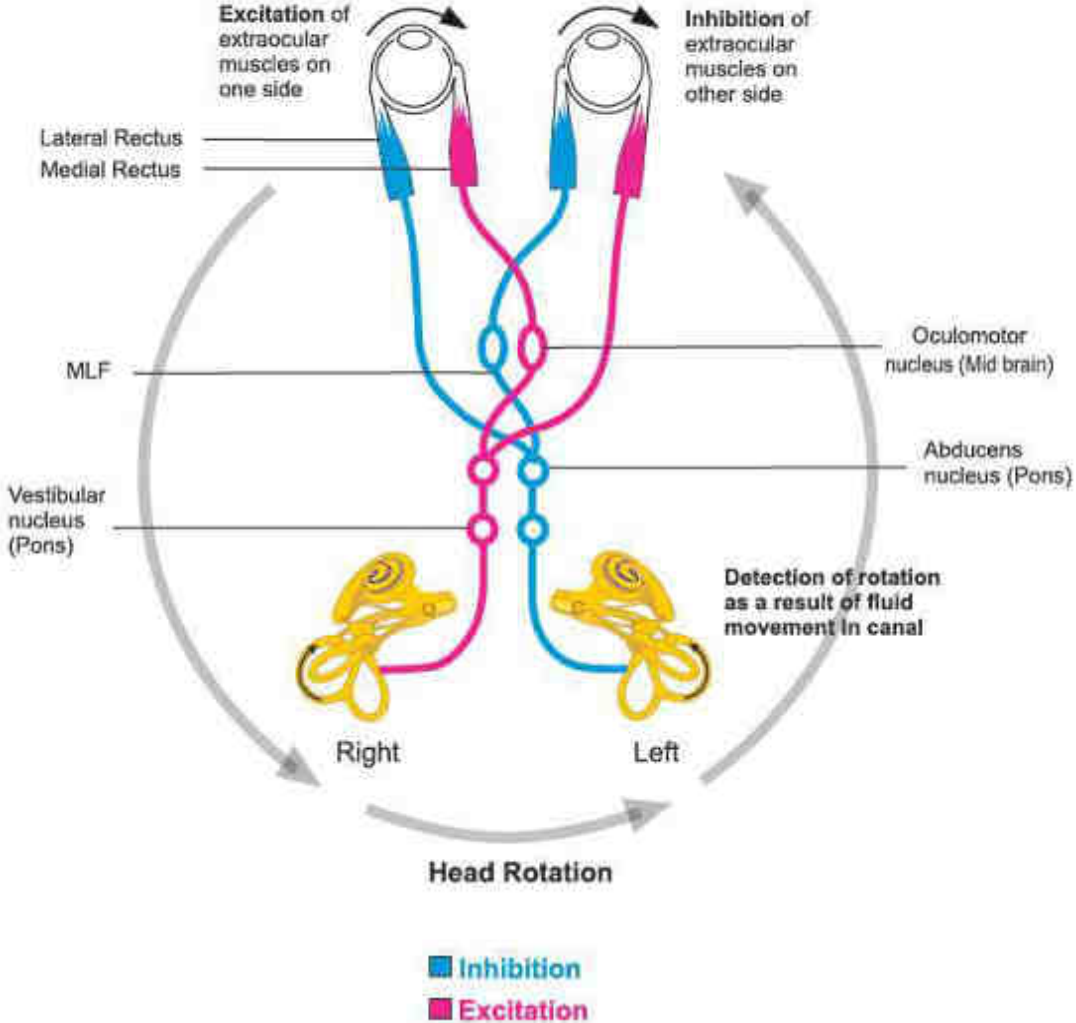


FIGURE 8-12 Vestibulo-ocular pathways. **A:** Anterior canal excitatory pathways; **B:** Posterior canal excitatory pathways; **C:** Horizontal canal excitatory pathways.

1. horizontal vestibulo-ocular impulse originating in the horizontal canal is relayed from the ipsilateral MVN to the contralateral abducens and the ipsilateral MR subnuclei neurons, resulting in deviation of the eyes to the contralateral side
2. Stimulation of the anterior canal (e.g., by downward head acceleration) excites the ipsilateral SR muscle and the contralateral IO muscle, whereas stimulation of the posterior canal (e.g., by upward head acceleration) excites the ipsilateral SO muscle and the contralateral IR muscle

COMPENSATING EYE MOVEMENT



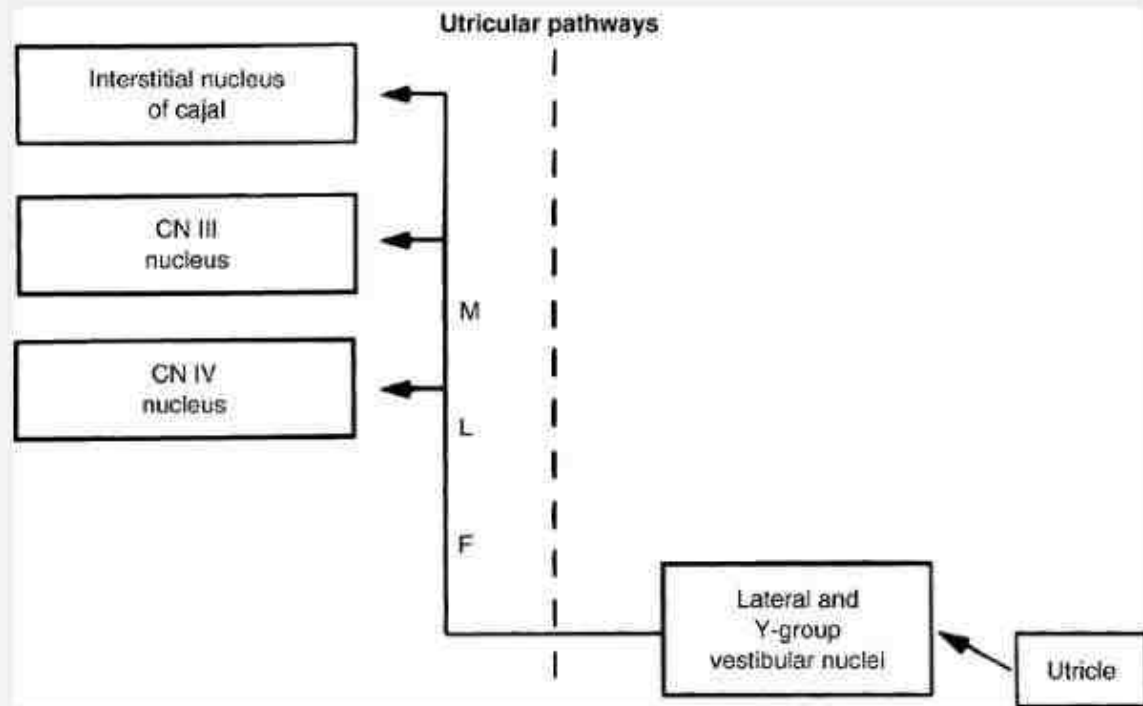


FIGURE 8-13 Diagram showing utricular pathways. *CN* = cranial nerve; *MLF* = medial longitudinal fasciculus. (From Brazis PW. Ocular motor abnormalities in Wallenberg's lateral medullary syndrome. *Mayo Clin Proc* 1992;67:365, Reprinted with permission.)

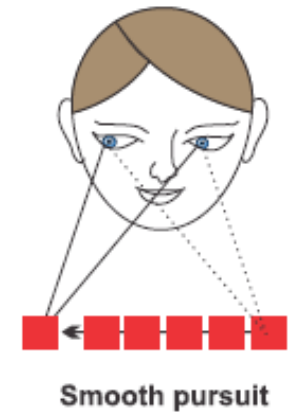
Brain site/clinical syndrome	Artery
<i>Medulla oblongata</i>	
Wallenberg's syndrome (DVD) with OTR and its features (head tilt, vertical divergence of the eyes, ocular torsion, deviation of the SVV) ipsiversive: lesion of the medial vestibular nuclei	Branches of the vertebral artery or PICA Rare: posterior spinal artery
"Vestibular pseudo-neuritis" (DVD) OTR ipsiversive: lesion of the superior vestibular nuclei	Branches of the vertebral artery or PICA Branches of the AICA
<i>Pons and midbrain</i>	
OTR or its components toward the opposite side: lesion of the MLF	Paramedian arteries of the basilar artery
UBN in combination with INO: lesion of the superior vestibular nuclei and the CVTT	Paramedian arteries from the basilar artery
SVV tilt ipsiversive: lesion of the medial lemniscus (IVTT)	Paramedian arteries from the basilar artery
<i>Rostral midbrain</i>	
OTR or its components contraversive: lesion of the INC and riMLF	Paramedian midbrain arteries from the basilar artery
<i>Paramedian thalamus</i>	
OTR contraversive to the lesion, only if rostral midbrain is affected (INC lesion)	50 % of the paramedian midbrain arteries originate with the paramedian thalamus arteries from the basilar artery
<i>Posterolateral thalamus</i>	
Tendency to fall to the side, SVV deviation, perhaps also astasia ipsiversive or contraversive	Thalamogeniculate arteries or perhaps branches of the posterior cerebral artery
<i>Temporoparietal cortex</i>	
Tendency to fall to the side, SVV deviation mainly contraversive, perhaps pusher syndrome	Branches of the middle cerebral artery
<i>Vestibulocerebellum</i>	
OTR with its components contraversive (ca. 60 %) or ipsiversive (ca. 25 %): lesions of the uvula/nodulus/dentate nucleus or parts of the cerebellar hemispheres	Branches of the PICA and AICA

OTR ocular tilt reaction, MLF medial longitudinal fascicle, riMLF rostral interstitial nucleus of the MLF, INC interstitial nucleus of Cajal, CVTT central ventral tegmental tract, IVTT ipsilateral vestibulothalamic tract, SVV subjective visual vertical, AICA anterior inferior cerebellar artery, PICA posterior inferior cerebellar artery

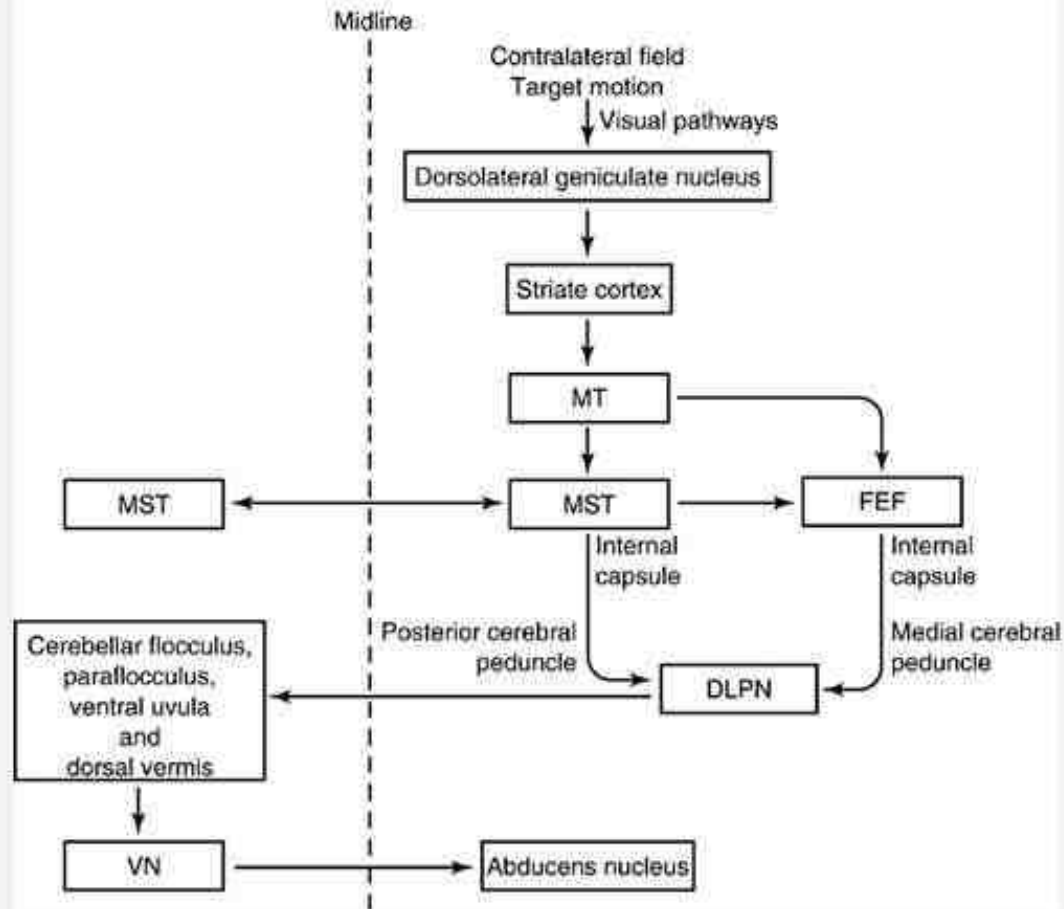
Smooth Pursuit



The smooth tracking system functions only when the eyes track a moving object that is traversing a predictable trajectory and is moving at a speed of less than 1.2Hz. The smooth pursuit system works best under this condition only and fails if the speed is more than 1.2Hz or the trajectory is uneven i.e., if the moving object abruptly changes speed or follows an unpredictable and changing trajectory.



Hypothetical scheme for horizontal smooth pursuit



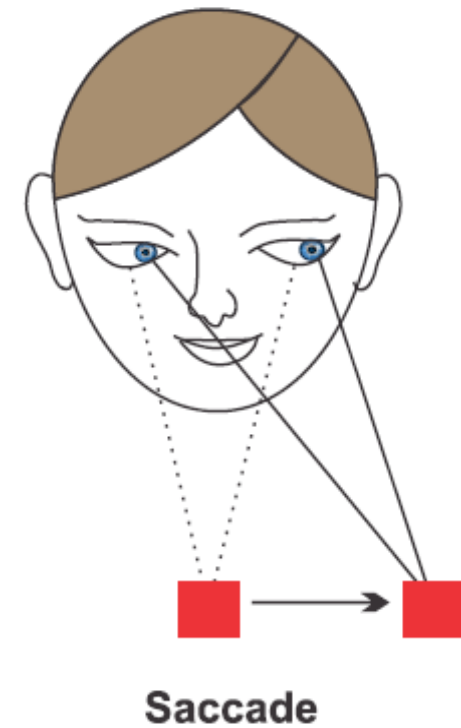
PURSUIT SYSTEM:

Control of smooth pursuit eye movements is a complex process. The stimulus for pursuit movement is movement of an image across the fovea at velocities greater than 3 to 5 degrees per second. Visual (striate and peristriate) cortex projects to parieto-temporo-occipital junction (PTO) as well as to Frontal eye field (FEF). The PTO projects via the internal sagittal striatum and the posterior limb of internal capsule to ipsilateral dorsolateral and lateral pontine nuclei (DLPN). Pursuit pathways control ipsilateral tracking hence undergo double decussation.

FIGURE 8-14 Schematic diagram illustrating major pathways involved in smooth pursuit eye movements. MT = middle temporal area; MST = medial superior temporal area; FEF = frontal eye field; DLPN = dorsolateral pontine nucleus; VN = vestibular nucleus.

Saccades

The saccade system is for stabilizing image of a visual target at the end of the visual field in the fovea by a rapid single eye movement. It facilitates visual tracking when SPS fails and when speeds are more than 1 Hz (rather 1.2Hz). The primary functional goal of the saccadic movements is to reposition a visual target of interest onto the fovea with a single rapid eye motion.



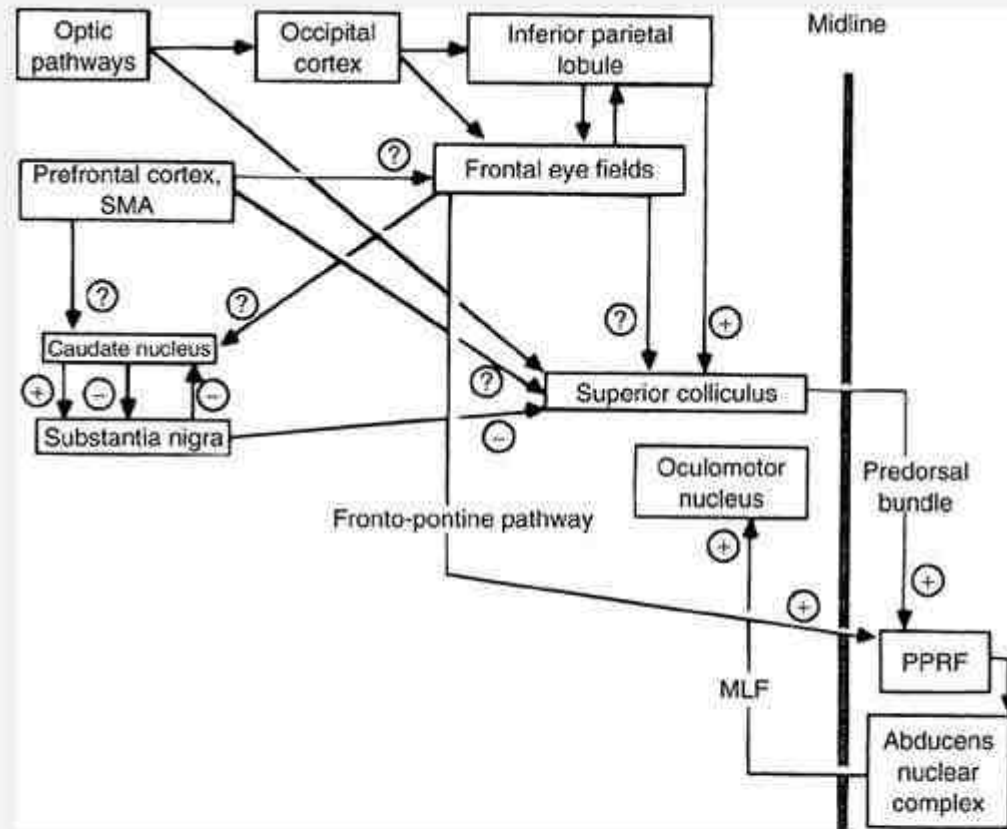
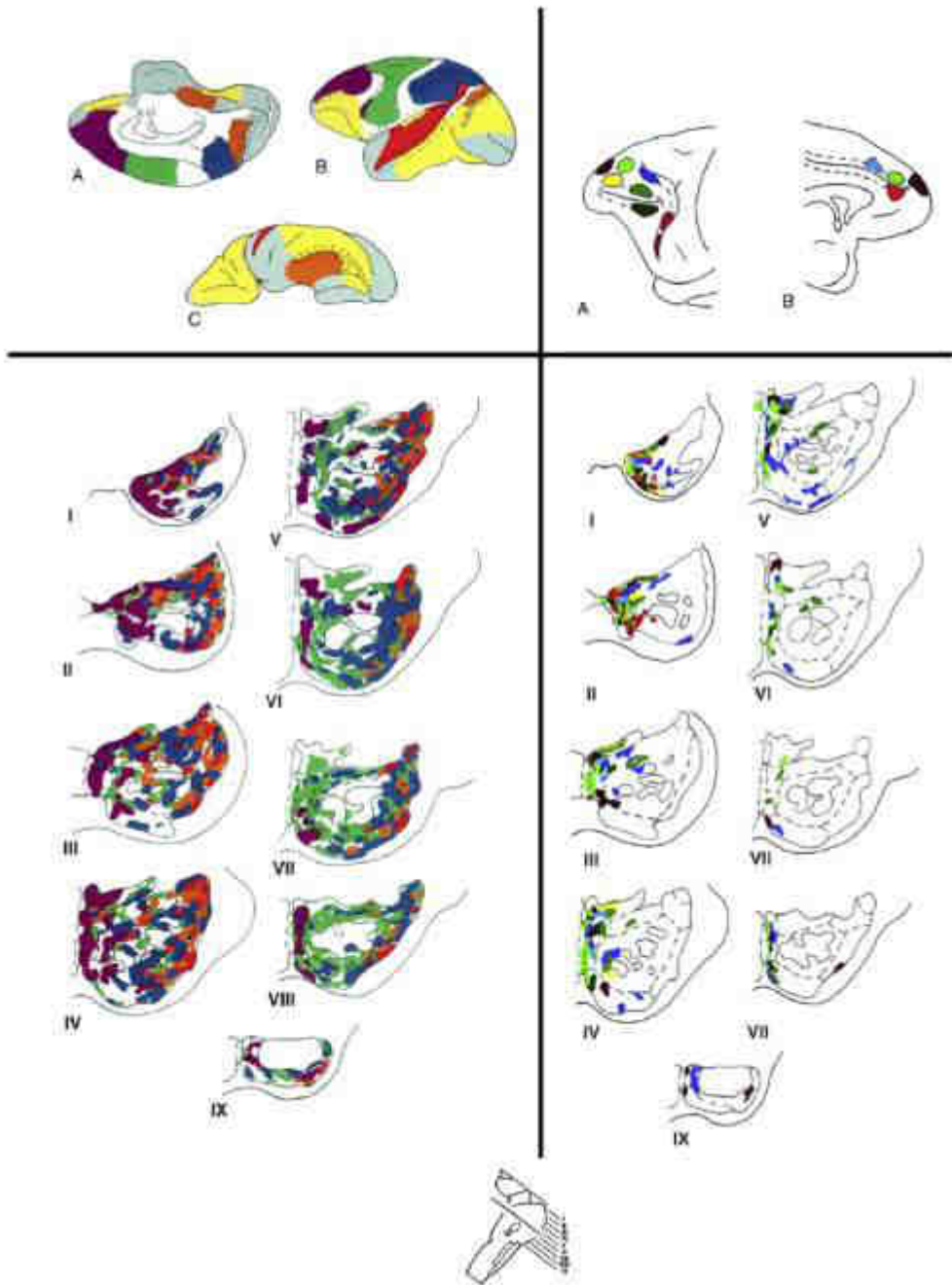


FIGURE 8-15 Schematic diagram illustrating the supranuclear pathways for lateral visually guided saccades. + = excitatory; - = inhibitory; ? = unknown effect (Adapted from Pierrot-Deseilligny C, Rivaud S, Penet C, et al. Latencies of visually guided saccades in unilateral hemispheric cerebral lesions. *Ann Neurol* 1987;21:138; Pierrot-Deseilligny C, Rivaud S, Fournier E, et al. Lateral visually guided saccades in progressive supranuclear palsy. *Brain* 1989;122:471.)

Saccadic System Disorders

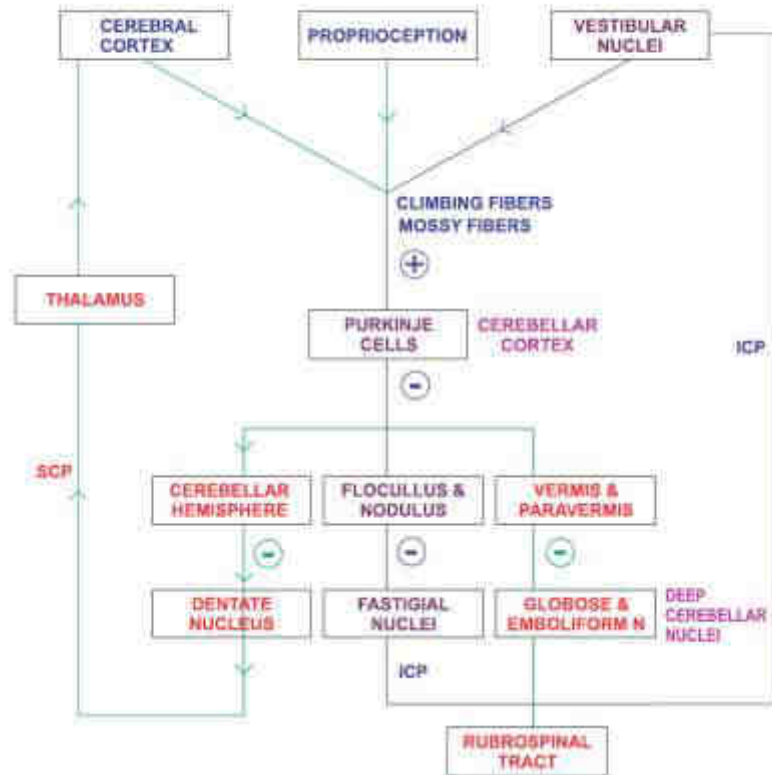
EXAMINATION FINDING	INFERENCE
Slowing of saccades/ hypometria	<ul style="list-style-type: none">• Intoxication• Neurodegenerative disorders
Slowing of horizontal saccades	<ul style="list-style-type: none">• Suggests brain lesions usually in ipsi PPRF (Paramedian pontine reticular formation)
Slowing of vertical saccades	<ul style="list-style-type: none">• Suggests brain lesions usually in riMLF (rostral interstitial medial longitudinal fasciculus) like Progressive supranuclear palsy
Slowing of adducting saccades	<ul style="list-style-type: none">• Suggests inter nuclear ophthalmoplagia
Hypermetric saccades	<ul style="list-style-type: none">• Suggests cerebellum (vermis) lesions or lesions in the cerebellar pathway, e.g. Wallenberg's syndrome due to damage to the inferior cerebellar peduncle

Cerebellum



Cerebellar Connections

Cerebellum: a key structure in maintenance of posture



Cerebellum has to and fro communications with three key systems of the body. They are cerebral cortex, vestibular system and proprioceptors. All of them provide valuable inputs related to body postures, eye and head positions of an individual. Cerebellum is connected by means of three peduncles (superior, middle and inferior) with the Midbrain, Pons and Medulla respectively. Superior cerebellar peduncle (SCP) is the main efferent pathway while inferior cerebellar peduncle (ICP) is the main afferent pathway which is in comprehensive connection with vestibulocerebellum. All afferent fibers (climbing, mossy) reach purkinje cells in the cerebellar cortex. This connection is excitatory. Various areas of cerebellar cortex then project to deep cerebellar nuclei. This connection is inhibitory in nature. Flocculonodular system in particular project to fastigial nucleus which in turn has feedback pathway with vestibular nuclei via inferior cerebellar peduncle (ICP). Cerebellum and cerebrum form a continuous feedback loop via dentate nucleus and thalamus. These dedicated circuits help in constant modulation of motor activity.

CEREBELLAR CONTROL OF OCULAR MOVEMENTS

The cerebellum coordinates the ocular motor system to drive the eyes smoothly and accurately and is richly supplied by afferent fibers conveying ocular information from vestibular system, afferent visual system, PPRF.

1) **Dorsal vermis and fastigial nuclei** determine accuracy of saccades by modulating saccadic amplitude also, they adjust innervations to each eye selectively to ensure precise conjugate movements. Lesions of these structures result in saccadic dysmetria (often overshoot dysmetria that is greater centripetally), **Macrosaccadic oscillations** and disorders of vergence.

2) **Flocculus** - it is a part of vestibulo-cerebellum which is responsible for matching saccadic pulse and step appropriately and for stabilizing images on fovea. Lesions of the flocculus result in gaze holding deficits, such as **gaze evoked, rebound and downbeat nystagmus**. Floccular lesions impair smooth pursuit, cancellation of VOR by pursuit system during combined head and eye tracking.

3) **Nodulus** - lesions of cerebellar nodulus cause loss of GABA mediated inhibition from the purkinje cells to vestibular nuclei leading to instability in velocity storage mechanism. Post rotational response is excessively prolonged leading to **periodic alternating nystagmus**. Some authorities believe that lesions in the nodulus may also cause central positioning nystagmus.

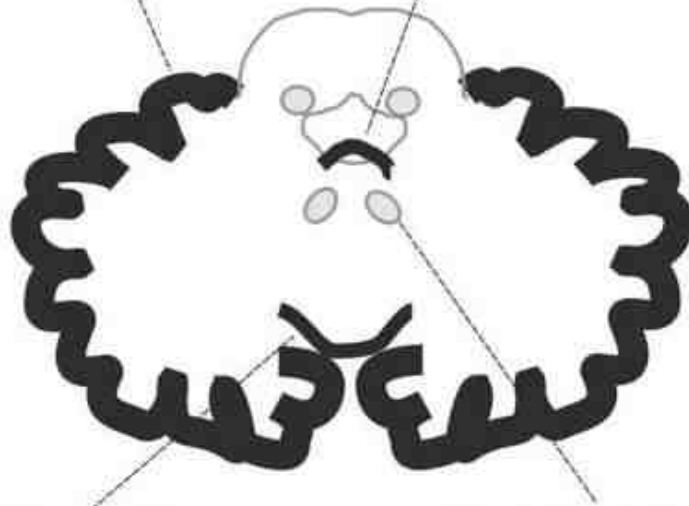
4) Deep cerebellar fastigial nuclei dysfunction leads to **pendular nystagmus**.

FLOCCULUS/PARAFLOCCULUS

Downbeat nystagmus
Gaze-evoked nystagmus and rebound
Impaired smooth pursuit (catch-up saccades, induced torsional nystagmus)
Impaired VOR visual suppression
Impaired head impulse VOR gain and direction

NODULUS/UVULA

Periodic alternating nystagmus
Positional downbeat nystagmus
Positional apogeotropic horizontal nystagmus
Head shaking nystagmus
Alternating skew deviation



OCULOMOTOR VERMIS

Saccadic hypometria
Impaired smooth pursuit
Esophoria

FASTIGIAL NUCLEUS

Saccadic hypermetria
Impaired smooth pursuit
Macrosaccadic oscillations
High frequency saccadic oscillations
Square wave jerks

Proprioception

Mechanoreceptors for Proprioception

Muscle spindles: length

Group IA:

Velocity + direction

Group II:

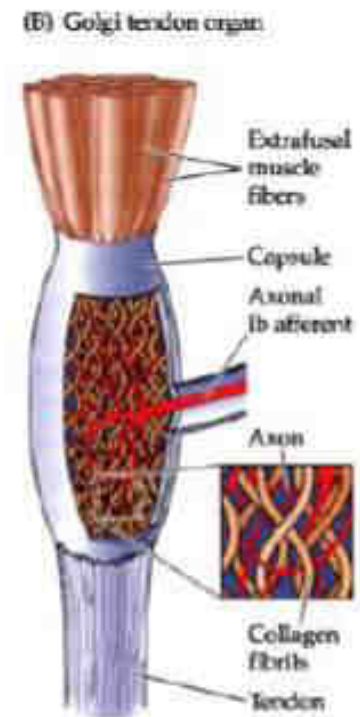
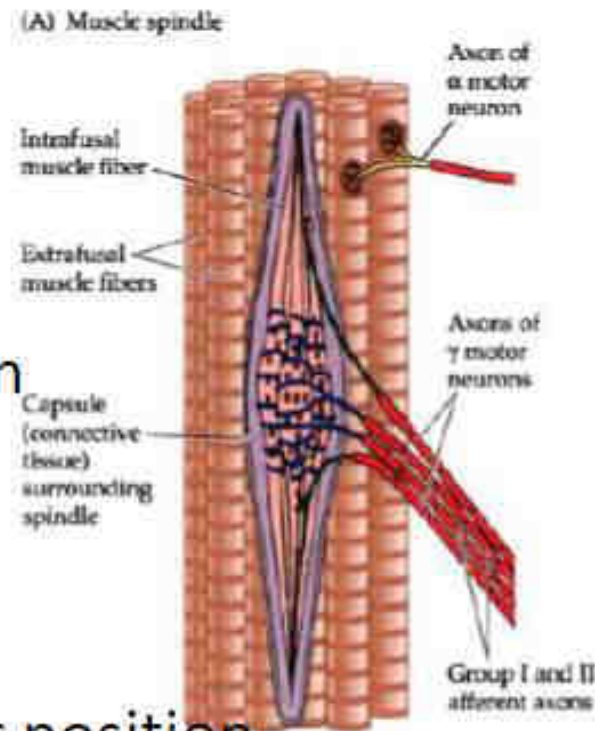
Sustained, static position

Golgi tendons: tension

Group Ib:

Branched in collagen fibers to form tendons

Joint receptors: finger position



Central Pathways

First order neurons in dorsal root and cranial nerve ganglia

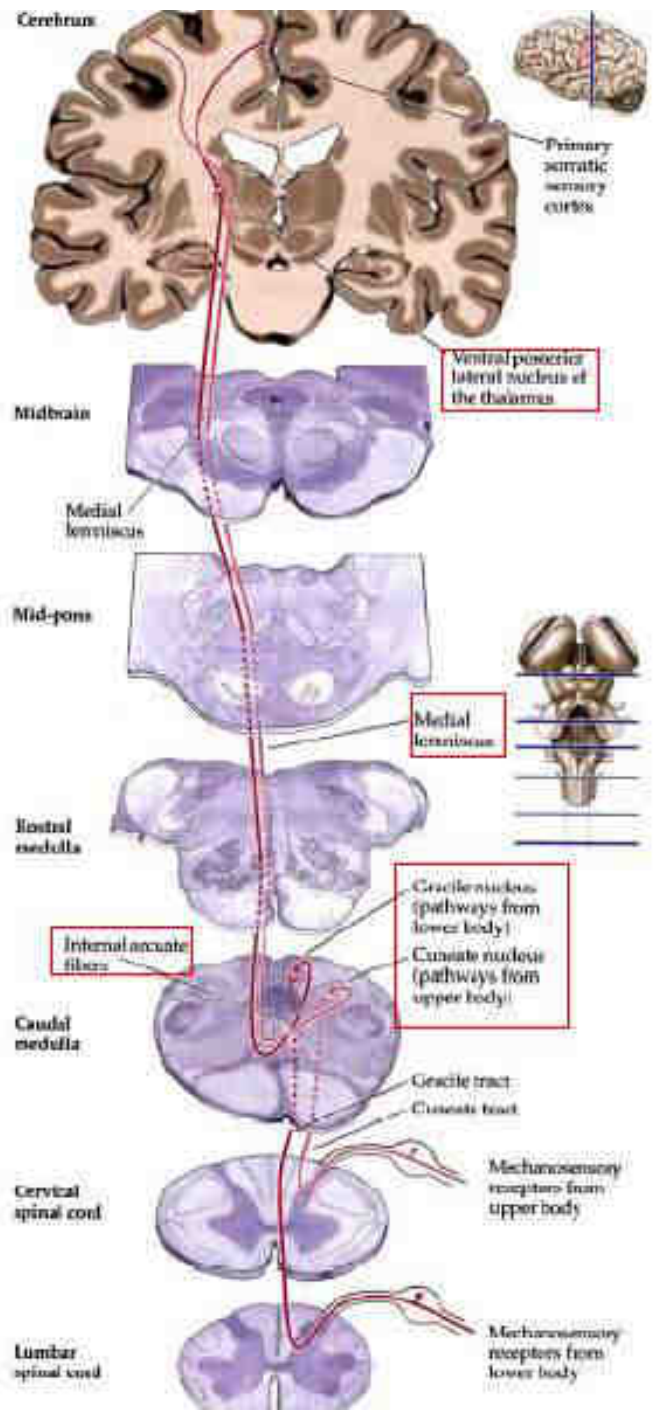
```
graph TD; A[First order neurons in dorsal root and cranial nerve ganglia] --> B[Second-order neurons in brainstem nuclei]; B --> C[Third order neurons in thalamus]; C --> D[Cerebral cortex];
```

Second-order neurons in brainstem nuclei

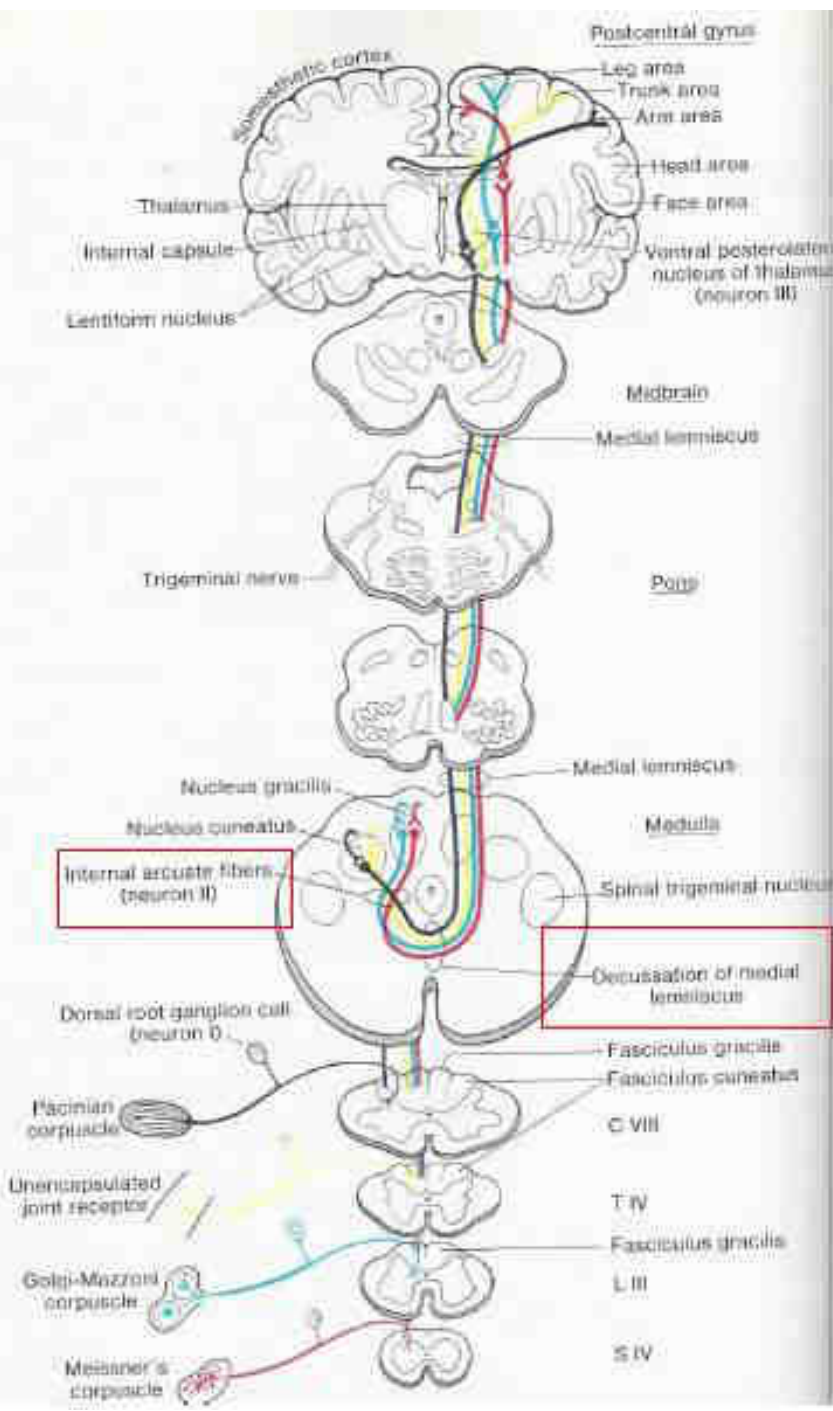
Third order neurons in thalamus

Cerebral cortex

Tactile Information from the Body



Dorsal Column-Medial Lemniscal System



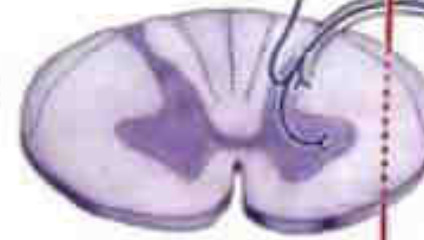
Proprioceptive Pathways for Body

- Upper: dorsal columns
 → medulla → nuclei
 → cross the midline
 → medial lemniscus → VPL

- Lower: medulla
 → outside gracilis
 → decussate and join
 medial lemniscus → VPL



Cervical spinal cord



To cerebellum and dorsal column nuclei

Muscle spindle afferents, upper body

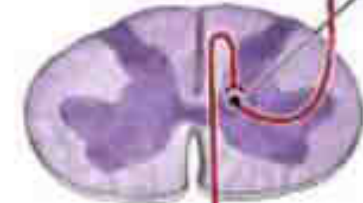


Thoracic spinal cord



Dorsal spinocerebellar tract

Lumbar spinal cord



Clark's nucleus

Muscle spindle afferents, lower body

Sacral spinal cord



Trigeminothalamic System

Trigeminal nerve → trigeminal brainstem complex:

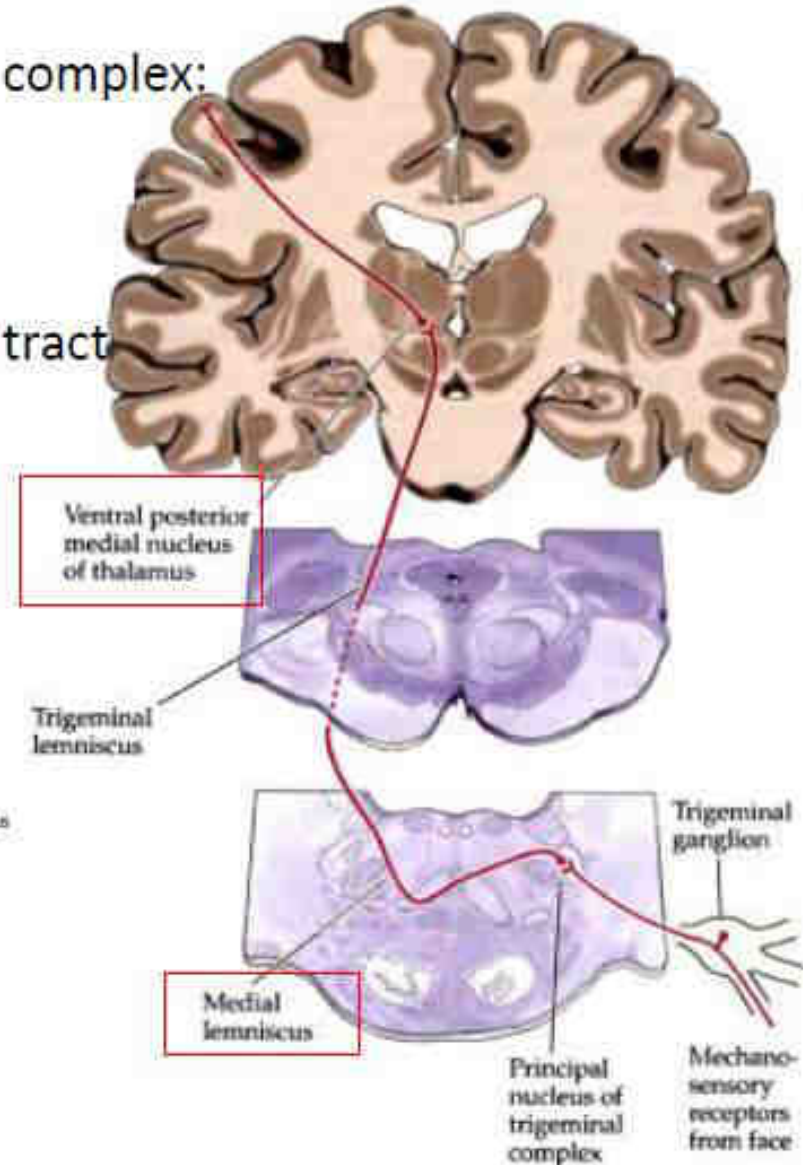
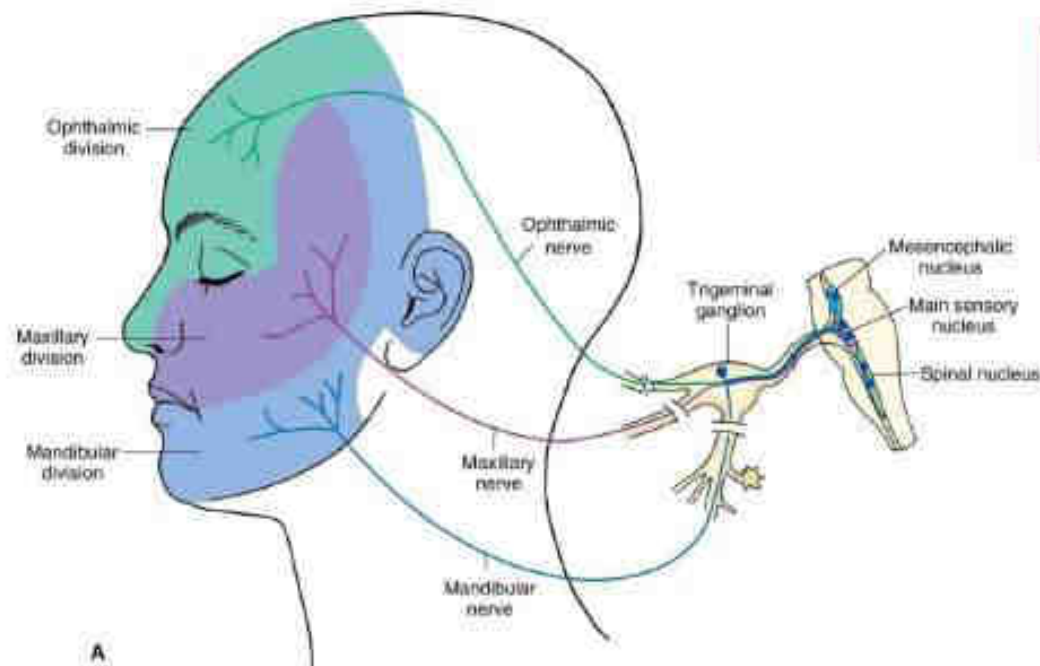
i) **principal**, ii) spinal, iii) mesencephalic

Low threshold mechanoreceptors →

principle nucleus → cross midline →

trigeminal lemniscus /trigeminothalamic tract

→ VPM → SI and SII

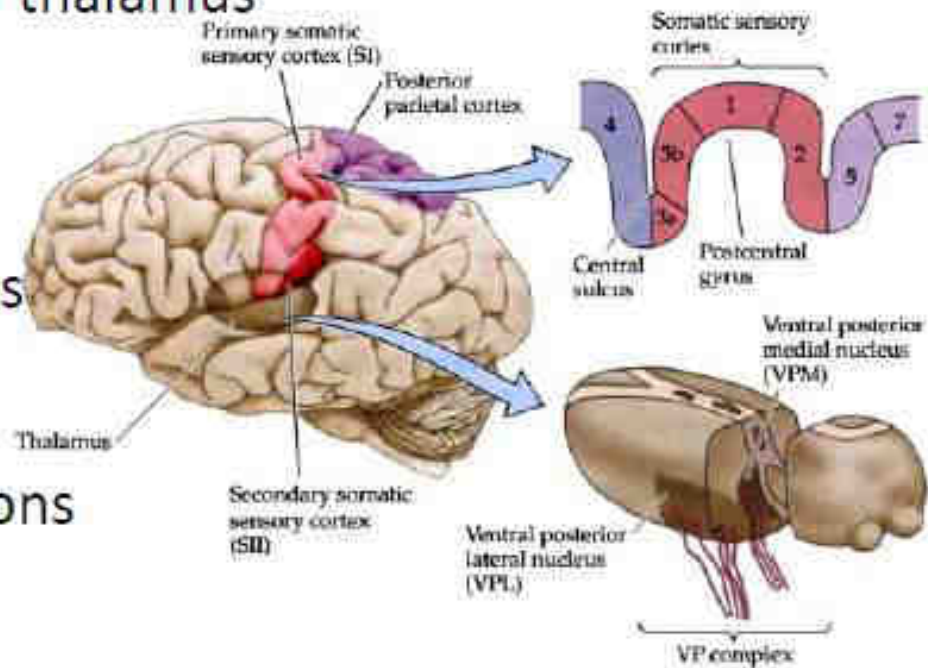


Somatic Sensory Portion of Thalamus

- Ascending: spinal cord and brain stem →
Ventral Posterior Complex of thalamus

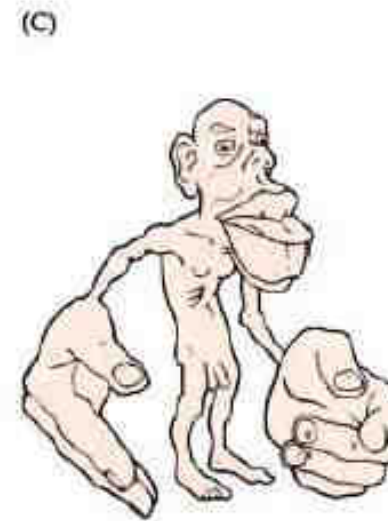
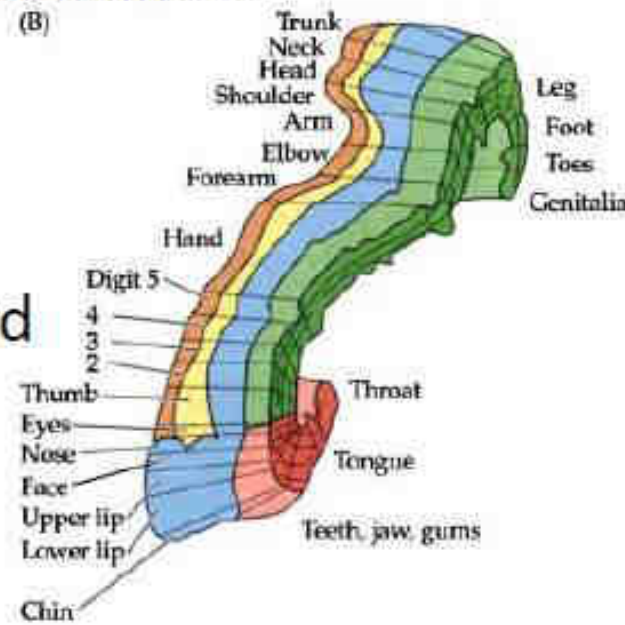
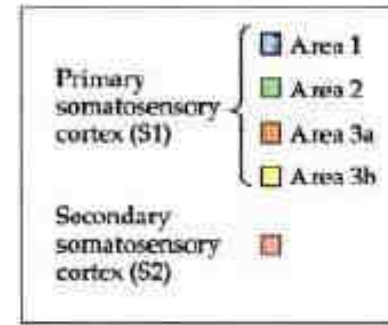
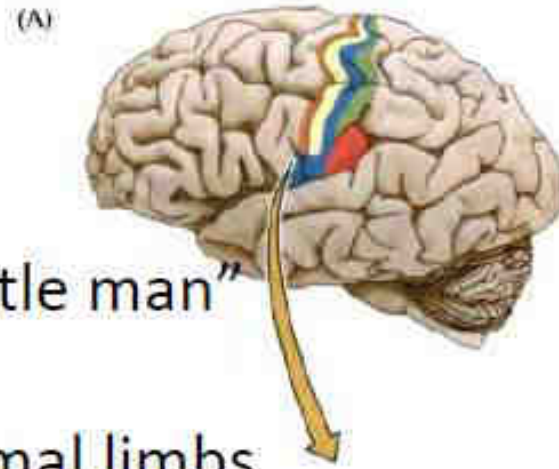
- VPL ← medial lemniscus
from posterior head + body
- VPM ← trigeminal lemniscus
from face

- Muscle spindle/ Golgi tendons

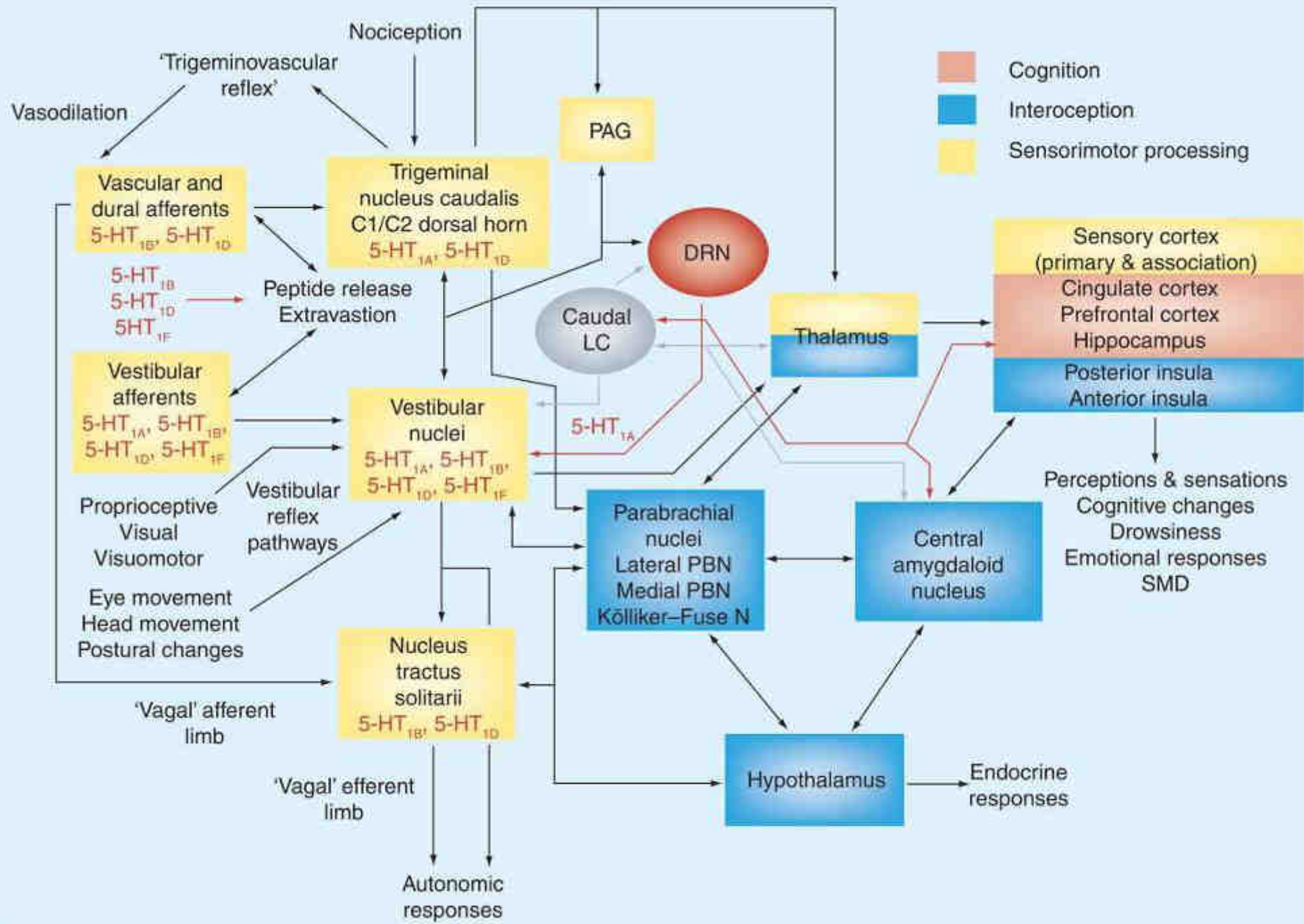


Somatotopic Map

- Homunculus - “Little man”
- Face and hands
 > torso and proximal limbs
- Manipulation,
 facial expression
 and speech
- Cervical spinal cord
- Receptor density



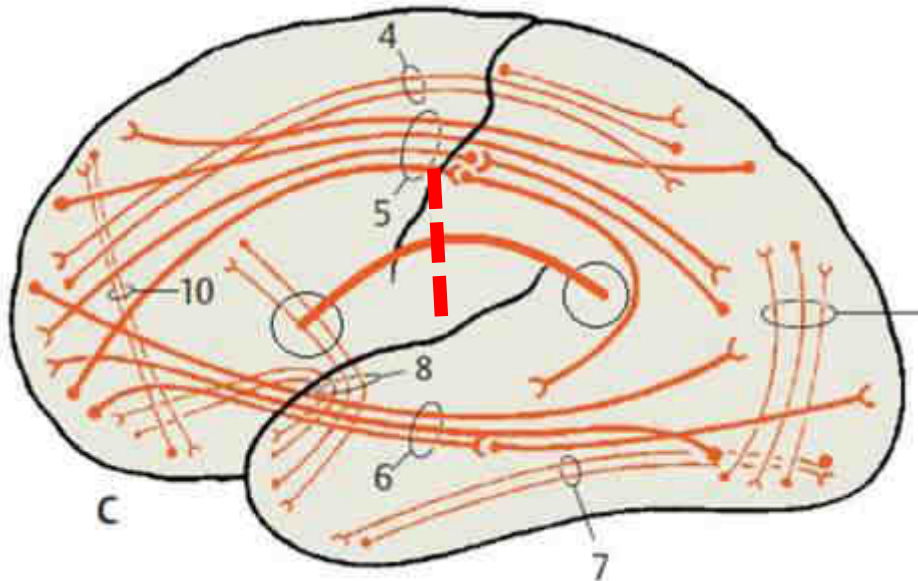
Putting them all together



Vertigo: A Disconnection Syndrome

Disconnection Syndromes

- Generic term for a number of neurological symptoms caused by damage to the white matter axons of communication pathways—via lesions to association fibers or commissural fibers—in the cerebrum, independent of any lesions to the cortex



Conduction Aphasia:

Can Read/Understand,
Can Speak,
Cannot Repeat



Short communication

Are white matter abnormalities associated with “unexplained dizziness”?



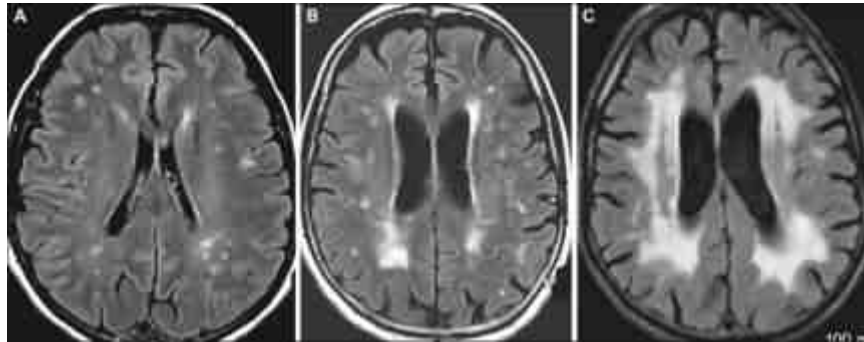
Hena Ahmad ^{a,1}, Niccolò Cerchiai ^{b,1}, Michelangelo Mancuso ^c, Augusto P. Casani ^b, Adolfo M. Bronstein ^{a,*}

^a Academic Department of Neuro-otology, Division of Brain Sciences, Imperial College London, Charing Cross Hospital, London, United Kingdom

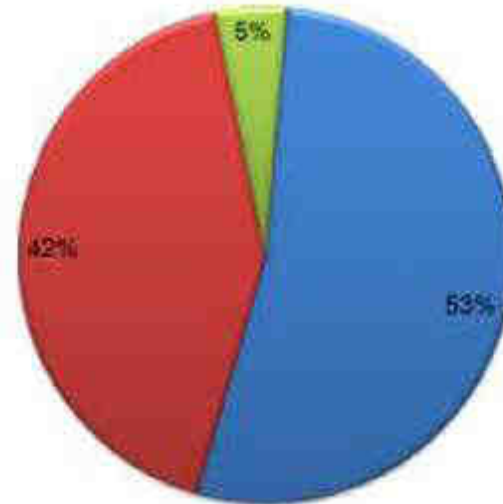
^b Department of Medical and Surgical Pathology, Otorhinolaryngology Unit, Pisa University Hospital, Pisa, Italy

^c Neurological Clinic, University of Pisa, Pisa, Italy

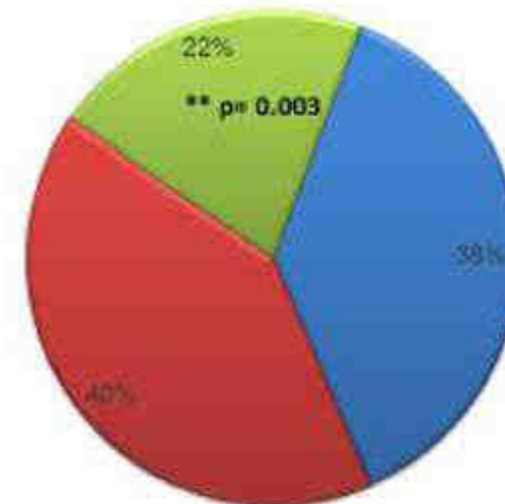
H. Ahmad et al. / Journal of the Neurological Sciences 358 (2015) 428–431



Explained dizziness



Unexplained dizziness



■ Fazekas 1 ■ Fazekas 2 ■ Fazekas 3

Fig. 1. Severity of white matter disease on MRI (Fazekas scores), expressed as percentage of patients with “explained” and “unexplained” causes of dizziness.



Nancy Chiaravalloti,
Director, NNL and TBI Labs,
Kessler Foundation, USA



Shubhajit Roy Chowdhury,
Assistant Professor,
IIT Mandi



Glenn Wylie,
Asst Director, NNL
and Neuroimaging,
Kessler Foundation, USA



Assia Jaillard,
Professor, CHU, Grenoble, France



Uttama Lahiri,
Assistant Professor
IIT - Ahmedabad



Anirban Dutta
Associate Researcher,
INRIA, France





Thank you