

Brainstem Clinical Neuroanatomy Made Flabbergastingly Simple

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May 1, 2025

Introduction

Skills to Achieve

- Locate lesions (***)
- Predict functional deficits (in addition to the ones mentioned in the vignette) (**)
- Identify arteries involved (*) (less important since most of the time these textbook syndromes occur due to tumours or other lesions, and rarely due to such well-targeted strokes)

Why?

To motivate the forthcoming study plan and the previously stated target skills, we'll take a look at a table differentiating between common brainstem stroke syndromes.

Key Brainstem Syndromes Distilled

Syndrome	Region & Artery	Cranial Nerves	Key Features
Claude	Midbrain (red nucleus), PCA	CN III	i3 + contralateral ataxia
Weber	Midbrain (cerebral peduncle), Basilar (paramedian branches)	CN III	i3c7u + contralateral hemiparesis
Parinaud	Midbrain (tectum), SCA & posterior choroidal (PCA branch)	—	Vertical gaze palsy, light-near dissociation
Millard-Gubler	Ventral pons, Basilar artery	CN VI, CN VII	i6, i7 + contralateral hemiplegia
Wallenberg	Lateral medulla, PICA	CN V (spinal nucleus), CN IX, CN X	Ipsilateral face pain/temp loss, contralateral body pain/temp loss, dysphagia, vertigo
Dejerine	Medial medulla, Anterior spinal artery	CN XII	i12 + contralateral hemiplegia, proprioception loss

Skills-Oriented Study Plan

We have to master the following topics in order to hammer down the aforementioned skills:

- Know the **basic sensory and motor tracts** including their pathways and functions
 - Tracts:
 - * Corticospinal
 - * Spinothalamic
 - * Dorsal column-medial lemniscal system (DCMLS)
 - * Corticobulbar
 - Pathways: must know where each of these tracts
 - * Decussate (if it does)
 - * is located in
 - Cerebral cortex
 - Int. capsule
 - Midbrain
 - Pons
 - Medulla
 - Spinal cord
- Know the **locations of the cranial nerve nuclei**
- Know the **clinically important reflex pathways**
- Know the clinically relevant **subdivisions** in **cross sections** of the different areas of the **brainstem**
- Know the **arterial supply** of the different parts

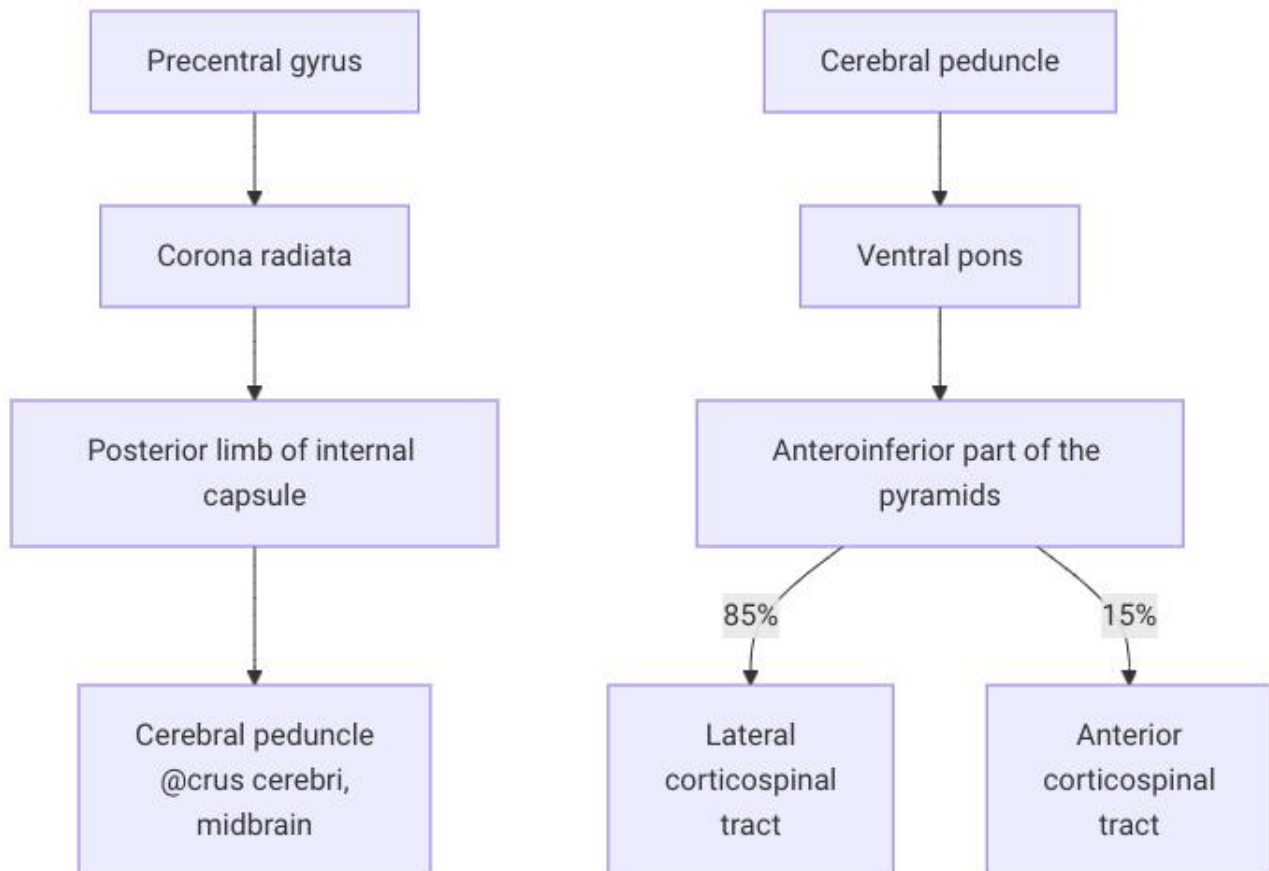
Tracts & Fasciculi

Corticospinal Tract

Functions

- Convey all motor signals to voluntary muscles

Pathway - Simplified



Pathway - Detailed

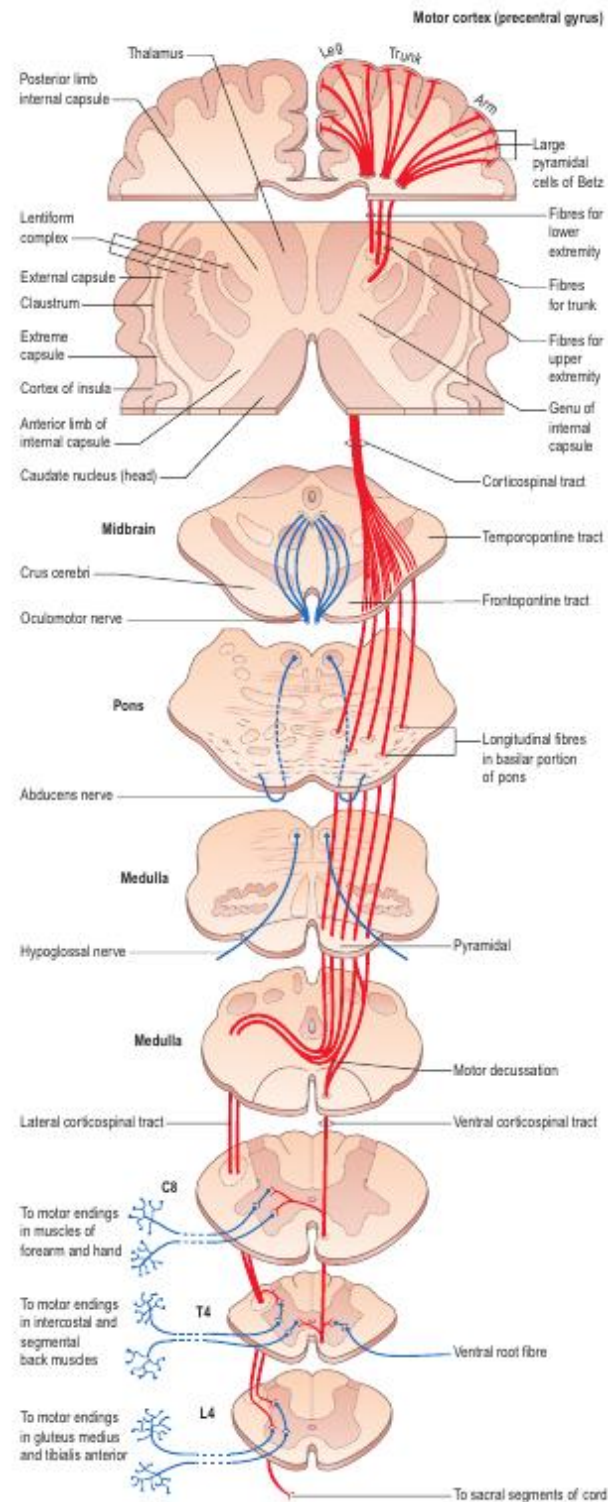


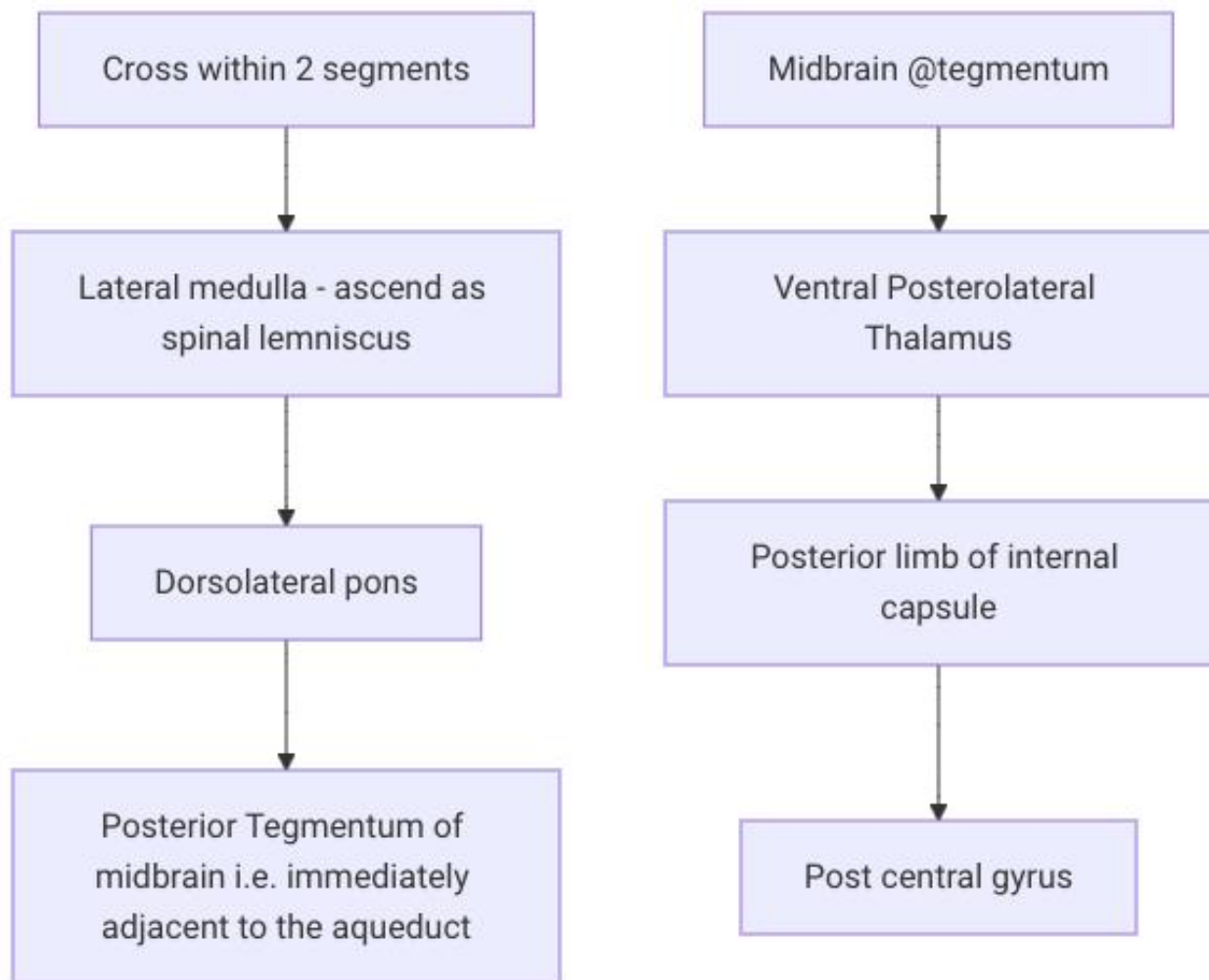
Fig. 20.16 The corticospinal tracts. (Redrawn with permission from Carpenter MB 1991 Core Text of Neuroanatomy, 4th edn. Baltimore: Williams and Wilkins.)

Spinothalamic tract

Functions

- **Anterior:**
 - Crude touch
 - Pressure
- **Lateral:**
 - Pain
 - Temperature

Pathway - Simplified



Pathway - Detailed

Ventral Spinothalamic tract

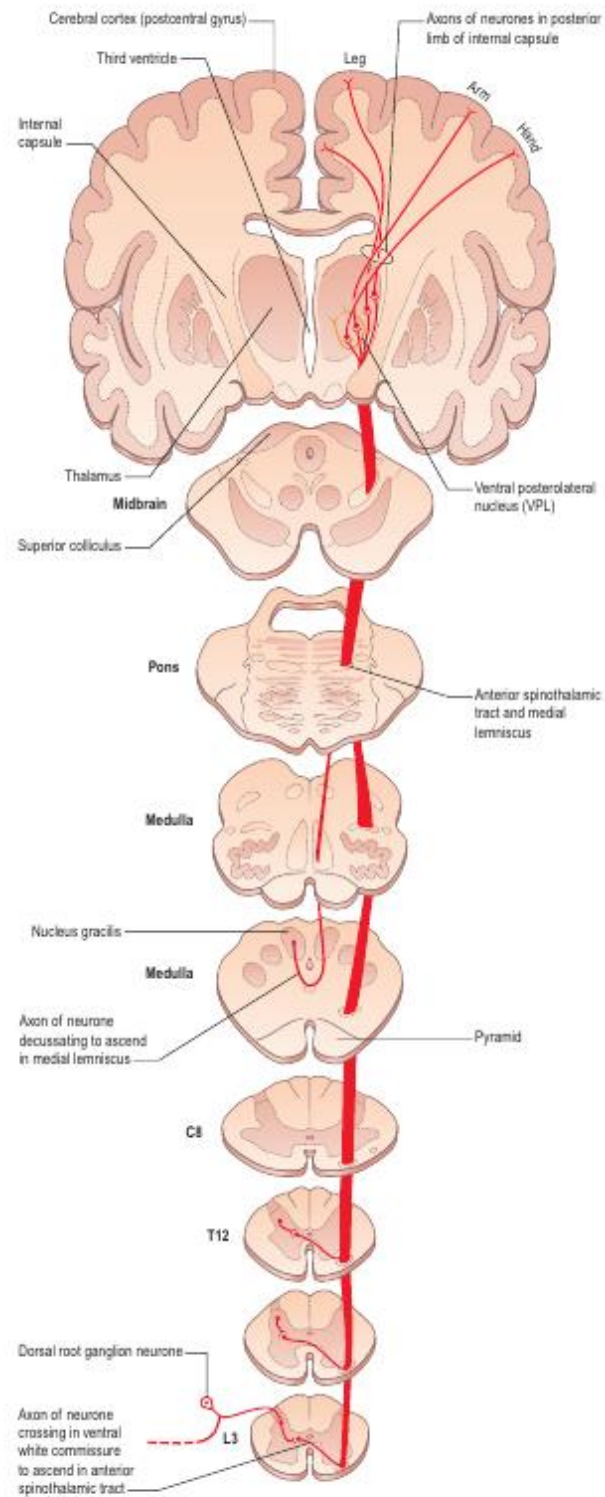


Fig. 20.13 The ventral (anterior) spinothalamic tract. (Redrawn with permission from Carpenter MB 1991 Core Text of Neuroanatomy, 4th edn. Baltimore: Williams and Wilkins.)

Lateral Spinothalamic Tract

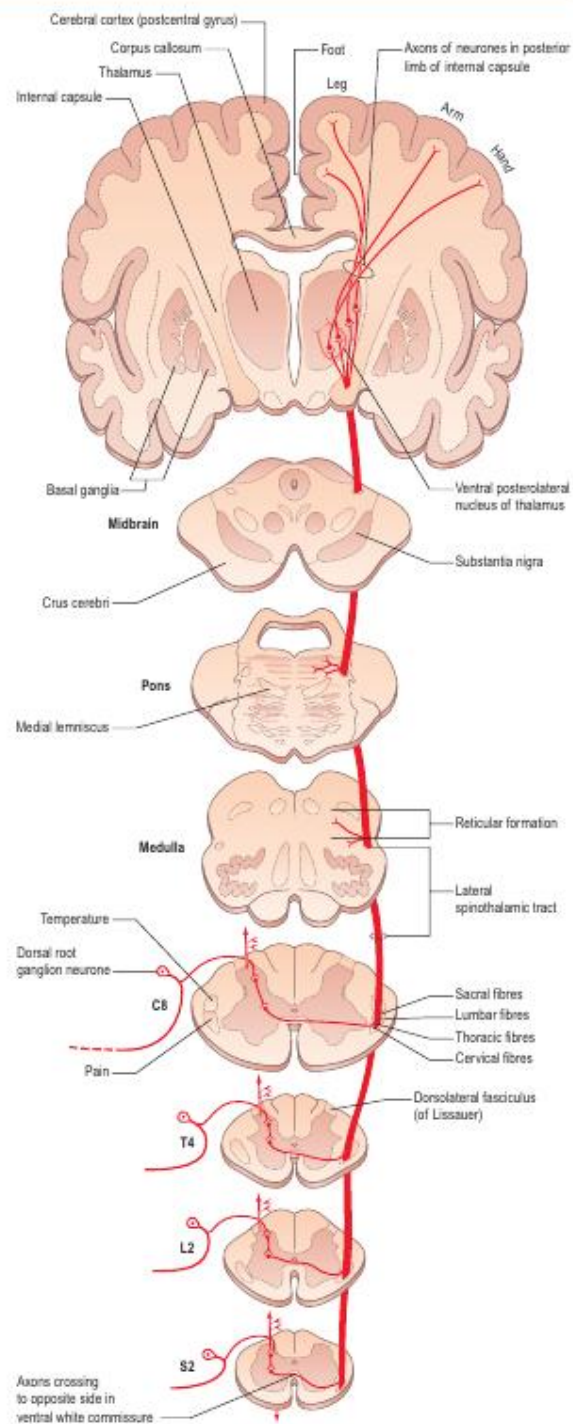


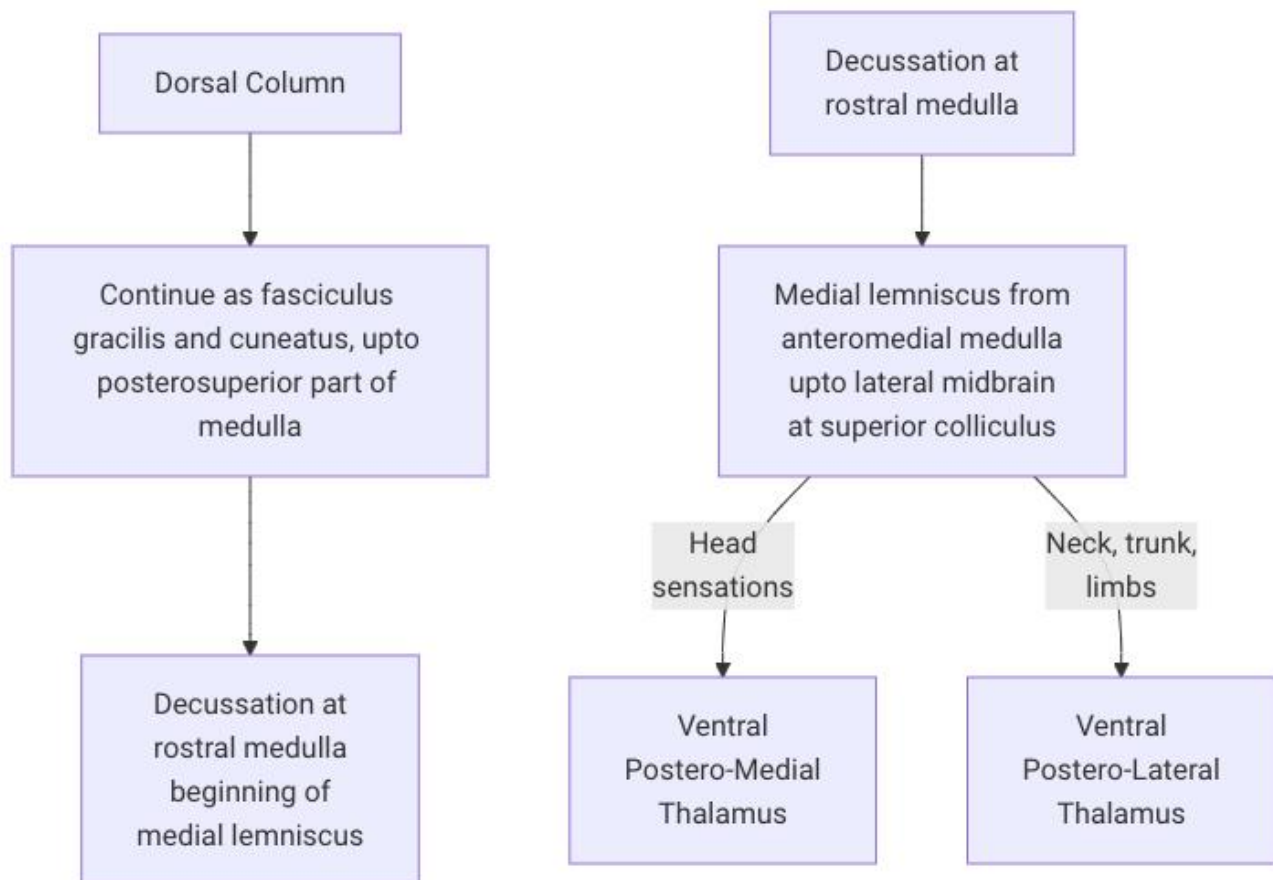
Fig. 20.12 The lateral spinothalamic tract. (Redrawn with permission from Carpenter MB 1991 Core Text of Neuroanatomy, 4th edn. Baltimore: Williams and Wilkins.)

Dorsal Column-Medial Lemniscus System (DCMLS)

Functions

- Fine touch
- Vibration
- Proprioception

Pathway - Simplified



Pathway - Detailed

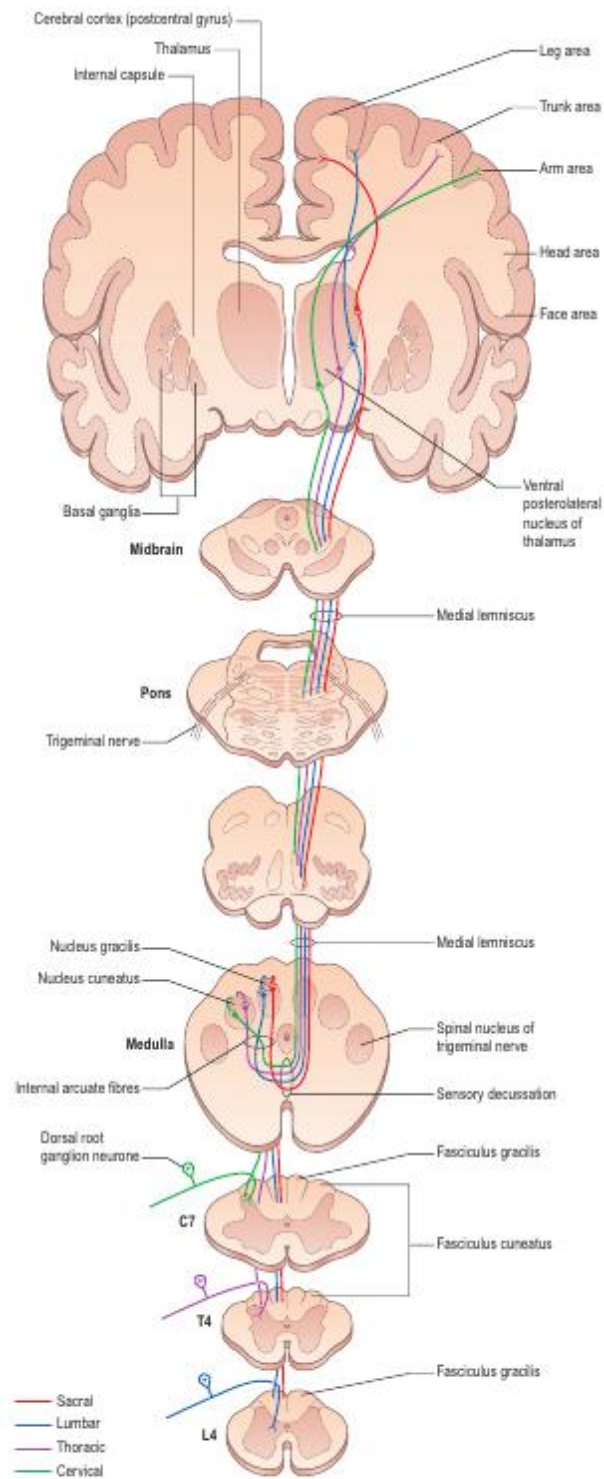


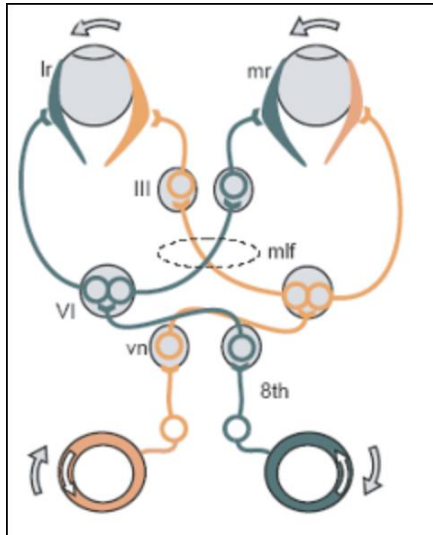
Fig. 20.10 The dorsal columns. Primary afferent fibres from different levels and their associated second- and third-order neurones are depicted in different colours.

Medial Longitudinal Fasciculus

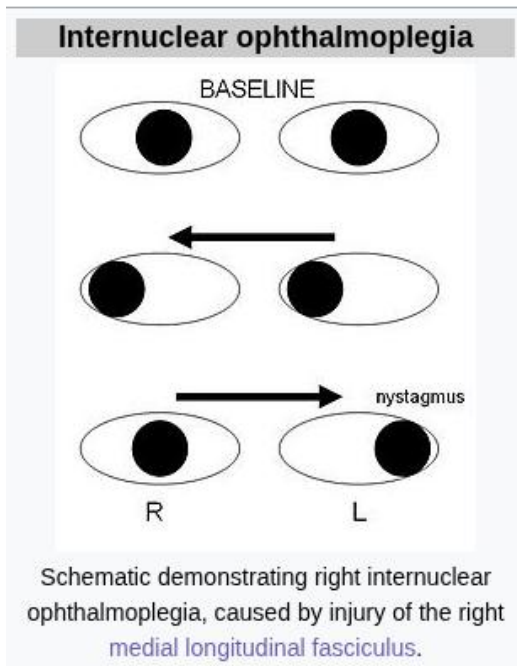
Functions

- Control conjugate eye movements by coordinating the actions of the nuclei of CN3, 4, and 6

Pathway



Effect of lesion - INO (internuclear ophthalmoplegia)



Midbrain

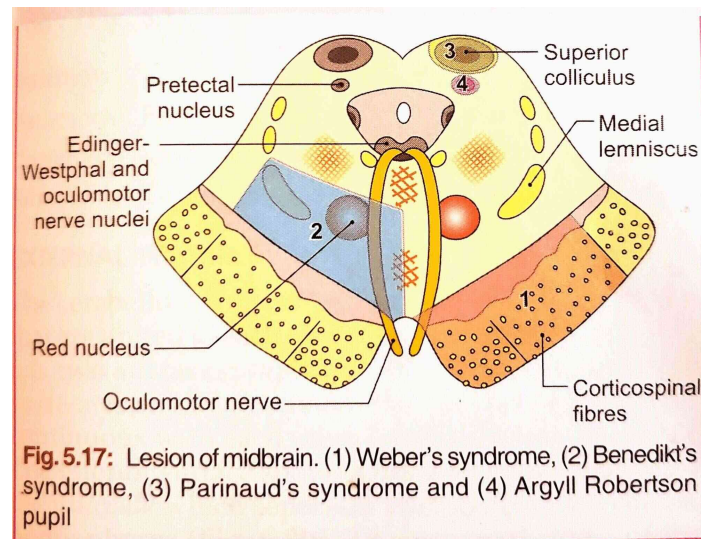


Figure 1: Midbrain

Region 1: Crus cerebri (Weber's Syndrome)

(cerebral peduncle = crus anteriorly and tegmentum posteriorly)

- *Artery:* Ventral paramedian branches of PCA
- *Lesion:* **Weber's**
- Clinically relevant *structures*:
 - Corticospinal and corticobulbar fibres in the cerebral peduncles
 - Oculomotor nerve fibres exiting from interpeduncular fossa
- Clinical *syndrome*: **i3 + c7u + CHP**
 - Ipsilateral: Oculomotor palsy ("down and out")
 - Contralateral: UMN facial, hemiplegia

Region 2: Tegmentum (Claude's Syndrome)

- *Artery:* Dorsal paramedian branches of PCA
- *Lesion:* **Claude's**
- Clinically relevant *structures*:
 - Red nucleus containing fibres from contralateral dentate nucleus of cerebellum (part of dentato-rubro-thalamic pathway)
 - Oculomotor nerve fibres in the central tegmental region
- Clinical *syndrome*: **i3 + CCA_t**
 - Ipsilateral: Oculomotor palsy (CN3)
 - Contralateral: Cerebellar ataxia

Region 3: Tectum (Parinaud's Syndrome)

- *Artery*: Posterior choroidal artery (br. of PCA) at sup. colliculus, superior cerebellar artery (SCA) at inf. colliculus
- *Lesion*: **Parinaud's**
- Clinically relevant *structures*:
 - Interstitial nucleus of Cajal at superior colliculus aka rostral interstitial nucleus of the MLF (riMLF) which is the vertical gaze centre
 - Pretectal nucleus: relays light reflex input arm signals to Edinger-Westphal nucleus, which then relays it to oculomotor nucleus
- Clinical *syndrome*:
 - Vertical gaze palsy (due to riMLF lesion)
 - Pupillary disorders (e.g. light-near dissociation) (due to pretectal nucleus lesion)

Pons

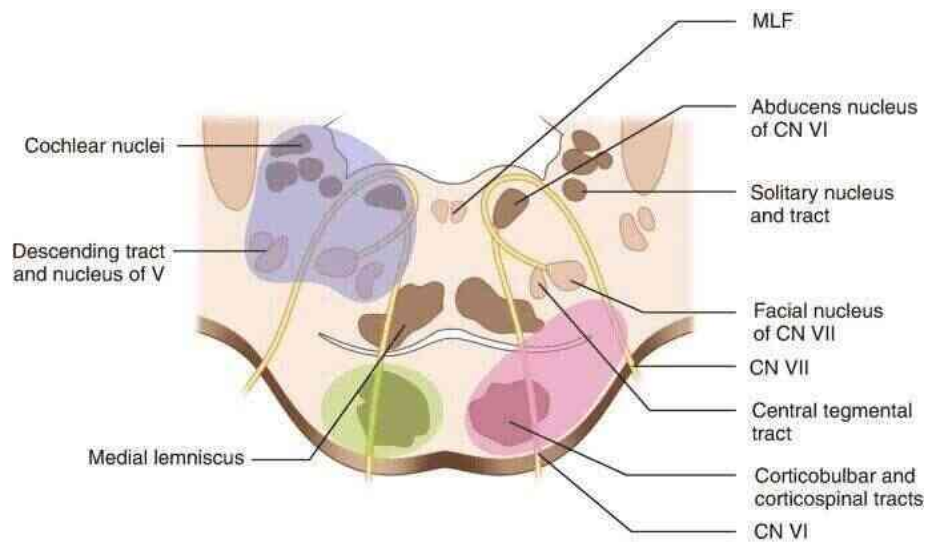


Figure 2: Pons

Ventral (Basilar) Pons (Millard-Gubler Syndrome)

- *Artery*: Basilar artery (lodges in the median sulcus between the two sides of pons)
- *Lesion*: **Millard-Gubler's**
- Clinically relevant *structures*:
 - Corticospinal tract in the paramedian area
 - Axons of CN6 and CN7
- Clinical *syndrome*: **i6, i7 + CHP**
 - Ipsilateral lateral rectus (CN6) and LMN facial palsy (CN7)
 - Contralateral hemiplegia

Medulla

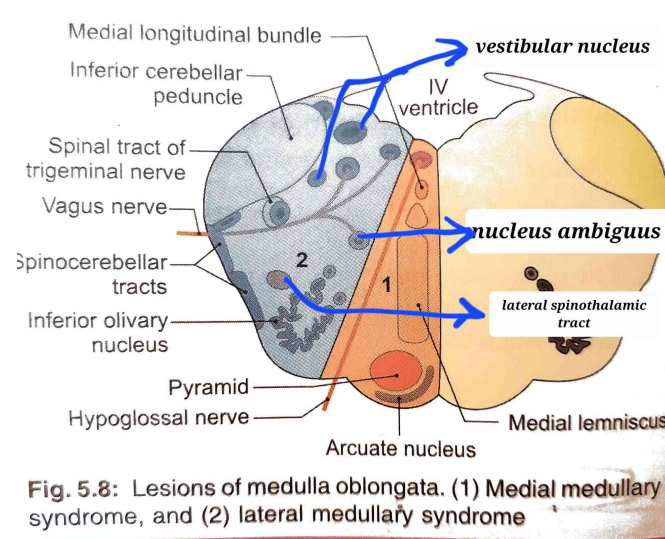


Figure 3: Medulla

Posterolateral Medulla (Wallenberg / lateral medullary syndrome)

- **Artery:** PICA (posteroinferior cerebellar artery, br. of vertebral)
- **Lesion:** **Wallenberg / lateral medullary syndrome**
- Clinically relevant *structures*:
 - Nucleus ambiguus: motor nucleus of CN 9, 10, 11 - ipsilateral soft palate, pharynx, larynx
 - Spinal trigeminal nucleus and tract: ipsilateral face - pain and temperature senses
 - Lateral spinothalamic tract: contralateral body - pain and temperature senses
 - Vestibular nucleus
 - Spinocerebellar tracts, inferior cerebellar peduncles - coordinate ipsilateral limb movements
 - Descending sympathetic fibres from hypothalamus - ipsilateral Horner
- Clinical syndrome: **i5, 9-11 + CPT**
 - Ipsilateral - 5 (pain and temperature lost at ipsilateral face), [9, 10, 11] (bulbar palsy) lesion; dysequilibrium (vestibular nucleus); ataxia (spinocerebellar tract); Horner's (ptosis, miosis, anhidrosis, enophthalmos)
 - Contralateral - pain and temperature lost (lateral spinothalamic) at contralateral body

Anteromedial Medulla (Dejerine syndrome)

- **Artery:** Anterior spinal artery
- **Lesion:** **Dejerine syndrome**
- Clinically relevant *structures*:
 - Pyramidal tract
 - Medial lemniscus
 - Hypoglossal nucleus and nerve
- Clinical syndrome: **i12, dc + CHP**
 - Ipsilateral: fine touch, vibration (DCMLS); tongue paralysis (CN12)
 - Contralateral: hemiplegia (pyramidal)

High-Yield Summary

Region	Artery	Syndrome	Key Features
Midbrain (crus)	PCA (ventral paramedian)	Weber's	i3 + contralateral hemiplegia + c7 UMN
Midbrain (tegmentum)	PCA (dorsal paramedian)	Claude's	i3 + contralateral ataxia
Midbrain (tectum)	Post. choroidal, SCA	Parinaud's	Vertical gaze palsy + pupillary light-near dissociation
Pons (ventral)	Basilar	Millard-Gubler	i6, i7 + contralateral hemiplegia
Medulla (anteromedial)	Anterior spinal	Dejerine's	i12 + contralateral hemiplegia + DCMLS loss
Medulla (posterolateral)	PICA	Wallenberg	i5, i9–11, Horner + contralateral pain/temp loss

Reflexes

Light and Accommodation Reflexes

Simplified Pathways

- Light enters through the retina and passes through the optic nerves to the primary visual cortex, V1, generating the visual experience
- V1 signals are transmitted to V2, V3 etc for higher-order processing
- Another part of the signals from CN2 go to the pretectal nucleus, which then forwards it to Edinger-Westphal nuclei *of both sides*, thereby triggering, through CN3 efferents, the **light reflex** in both eyes
- In case of accommodation, signals from V2, V3 etc higher order visual centres of both sides directly travel to EWN of both sides, thereby triggering the **accommodation reflex**.

Clinico-anatomical correlations

- Since the *accommodation reflex bypasses pretectal nucleus*, lesions of the pretectal nucleus will cause **light-near dissociation**
- Also, if the retina/CN2/optic chiasm happens on one side, then the pretectal nucleus of that side won't be stimulated when light is shone, and so light reflex doesn't get triggered. However since the accommodation reflex depends on afferents from higher-order visual processing which is inherently bilateral where both V1's share information through the corpus callosum, during lesions of retina/CN2 etc of one side, the other side will still contribute to the V2 and higher, and that means that V2 on the diseased side will trigger accommodation - i.e. there's also light-near dissociation in this case.

Aetiology of LND

- **Argyll-Robertson pupil:** Pretectal nucleus lesion due to syphilis, which causes LND by the aforementioned mechanism.
- **Holmes-Adie's Tonic Pupil:** Results from damage to the ciliary ganglion or postganglionic parasympathetic fibers (e.g., due to viral infection, diabetes, or autoimmune conditions like Sjögren's syndrome).

The pupil is initially dilated, with slow or absent light response. Over time, aberrant regeneration of fibers to the ciliary muscle (for accommodation) restores the near response, but the light response remains poor.

RAPD (Marcus-Gunn Pupil) Mechanism

- RAPD results from **asymmetric afferent pathway damage** (retina or optic nerve) reducing light signal transmission from the affected eye to the pretectal nucleus.
- During the swinging flashlight test, weaker afferent input from the affected eye leads to reduced EWN activation, causing less pupillary constriction or paradoxical dilation in both eyes when the affected eye is stimulated.
- The bilateral projection from the pretectal nucleus to both EWNs ensures that the defect affects both pupils' responses, making RAPD a sensitive indicator of unilateral or asymmetric afferent pathology.
- The near reflex is spared because it relies on cortical pathways, not the pretectal nucleus, explaining *light-near dissociation in RAPD* cases.