Anatomy & Physiology of the Vestibulocerebellum

Clinician Quick-Reference • Australian Dizziness Clinics • 2026

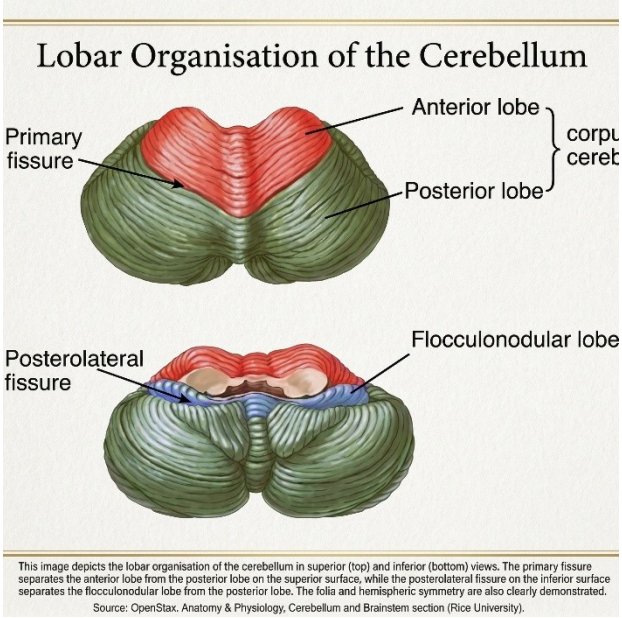
GROSS ANATOMY

Flocculonodular Lobe + Uvula (IX)

- **Flocculus** — inferior cerebellar surface; AICA territory; VOR gain + gaze holding
- **Paraflocculus (tonsil)** — adjacent to flocculus; optokinetic + smooth pursuit circuitry
- **Nodulus (X)** — roof of 4th ventricle; canal-dominant; velocity storage regulation
- **Uvula (IX)** — superior to nodulus; otolith-dominant; tilt/translation disambiguation

Vascular Territories

- **AICA** → flocculus, paraflocculus, anterior inferior cerebellum
- **PICA** → nodulus, uvula, posterior inferior cerebellum



This image depicts the lobar organisation of the cerebellum in superior (top) and inferior (bottom) views. The primary fissure separates the anterior lobe from the posterior lobe on the superior surface, while the posterolateral fissure on the inferior surface separates the flocculonodular lobe from the posterior lobe. The folia and hemispheric symmetry are also clearly demonstrated.

Source: OpenStax. Anatomy & Physiology, Cerebellum and Brainstem section (Rice University).

Deep Cerebellar Nuclei

- Fastigial — receives nodulus/uvula output, projects bilaterally to vestibular nuclei
- Interposed (globose + emboliform) — spinocerebellar motor coordination
- Dentate — receives cerebrocerebellum output; projects to thalamus via VL

CORTICAL HISTOLOGY

Three-Layer Architecture

- **Molecular layer** — Purkinje dendrites, parallel fibres, stellate + basket cells
- **Purkinje cell layer** — sole output of cerebellar cortex; inhibitory (GABA)
- **Granular layer** — granule cells (excitatory, glutamate); largest neuron population in CNS

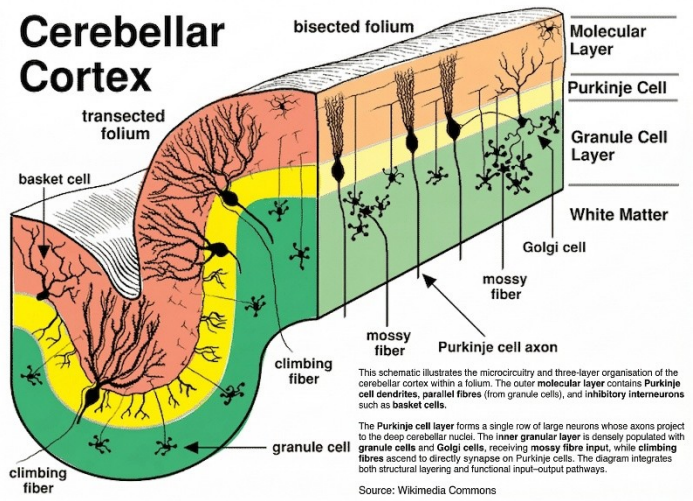
Two Afferent Systems

- **Mossy fibres** → granule cells → parallel fibres → Purkinje cell dendrites (divergent; modifiable)
- **Climbing fibres** (inferior olive) → powerful 1:1 synapse directly on Purkinje cell body (error signal)

Marr-Albus-Ito Learning Rule

- Simultaneous parallel fibre + climbing fibre activation → **LTD** at the parallel fibre–Purkinje synapse → reduced Purkinje inhibition of vestibular nuclei → VOR gain change

♥ *The inferior olive is the "teacher" — climbing fibre discharge encodes the error (retinal slip); LTD is the mechanism of VOR recalibration.*



CONNECTIONS — VESTIBULOCEREBELLUM

Structure	Key Afferents	Key Efferents	Clinical Function
Flocculus	Retinal slip (IFO dorsal cap); 2° vestibular (MVN/SVN); eye velocity (NPH)	MVN, SVN, NPH, Y-group	VOR gain adaptation; VOR cancellation; gaze holding
Nodulus (X)	Primary + 2° vestibular (canal-dominant); retinal slip via IFO VLO	MVN, SVN (inhibit velocity storage)	Velocity storage regulation; duration of post-rotatory nystagmus
Uvula (IX)	Primary + 2° vestibular (otolith-dominant); utricular + saccular signals	MVN, fastigial nucleus	Tilt/translation disambiguation; gravity–inertia resolution

PHYSIOLOGY SUMMARY

VOR Gain Adaptation — Flocculus

- Retinal slip (climbing fibres) + vestibular drive (parallel fibres) → LTD → Purkinje inhibition of MVN decreases → VOR gain recalibrated
- Floccular damage → gain stuck; cannot adapt to magnifying/minifying lenses
- Clinical test: **VOR suppression** (VORS) failure = floccular pathology

Velocity Storage — Nodulus

- Velocity storage prolongs VOR time constant beyond canal mechanical limit (~7 s)
- Nodulus actively **inhibits** velocity storage via MVN projections
- Nodular lesion → exaggerated velocity storage → prolonged post-rotatory nystagmus, PAN
- Clinical sign: **Periodic Alternating Nystagmus** (PAN) = nodular dysfunction

Tilt/Translation Disambiguation — Uvula

- Otoliths cannot distinguish gravity from linear acceleration (Einstein equivalence)
- Uvula integrates canal (fast) + otolith (slow) signals to disambiguate tilt from translation
- Uvular lesion → abnormal tilt perception; central positional nystagmus

Gaze Holding — Flocculus & Paraflocculus

- Neural integrator converts velocity commands to position signal for eccentric gaze
- Flocculus/paraflocculus provide leak-correction to prevent centripetal drift
- Failure → **gaze-evoked nystagmus** (GEN) beating away from centre
- Rebound nystagmus on return to centre = floccular sign

OCULAR MOTOR EXAMINATION

Test	Abnormal Finding	Localisation / Significance
Spontaneous nystagmus	Vertical (DBN/UBN); direction-changing; torsional	Central until proven otherwise — investigate
Gaze-evoked nystagmus	Nystagmus beating away from centre at eccentric gaze	Flocculus/paraflocculus or brainstem (neural integrator)
Rebound nystagmus	Nystagmus on return to centre, opposite to GEN direction	Floccular/cerebellar — highly specific central sign
Downbeat nystagmus	Fast phase downward; worse in lateral gaze + convergence	Flocculus/paraflocculus — see DBN section p.3
Smooth pursuit	Saccadic (cogwheel) pursuit	Paraflocculus, dorsolateral pons, or cortex
VOR suppression	Cannot suppress VOR on head-fixed target (thumb test)	Flocculus — key central sign; peripheral VOR intact
Saccades	Hypermetria (overshoot); macrosaccadic oscillations	Dorsal vermis / fastigial nucleus lesion
Head impulse test (vHIT)	Negative HIT in acute vertigo → suspect central	Exception: AICA infarction can give positive HIT + deafness
Skew deviation	Vertical misalignment on cover-uncover test	Disruption of otolith–ocular pathway; brainstem/cerebellum

GAIT, STANCE & COORDINATION

Gait & Stance

- **Tandem gait** — highly sensitive for vestibulocerebellar + spinocerebellar dysfunction
- **Romberg** — cerebellar patients may fall with **eyes open and closed** (vs vestibular/proprioceptive: eyes closed only)
- **Ipsipulsion** (lateral sway/falling toward lesion side) — fastigial nucleus pathology, Wallenberg syndrome
- **Broad-based ataxic gait** — spinocerebellar (anterior/posterior lobe)

Coordination Tests

- **Finger–nose–finger** — intention tremor, dysmetria; cerebellar hemisphere (dentate nucleus)
- **Heel–shin** — lower limb coordination; spinocerebellar ataxia
- **Rapid alternating movements** — dysdiadochokinesis; hemisphere sign
- **Rebound test** — failure of cerebellar braking after resistance release, hemisphere sign

♥ *Standard coordination tests are relatively insensitive to pure vestibulocerebellar pathology — the diagnosis lies in the ocular motor exam.*

ACUTE PRESENTATIONS — STROKE MIMICS

HINTS Examination

- **H**ead impulse + **I**nspection of nystagmus + **T**est of **S**kew
- Central pattern: **Normal HIT** + direction-changing or vertical nystagmus + **any skew deviation**
- Peripheral pattern: Positive HIT + unidirectional horizontal nystagmus + no skew (reassuring — not absolute)

PICA vs AICA Territory

- **PICA** — nodulus, uvula, posterior inferior cerebellum; negative HIT; acute vertigo + severe ataxia; risk of cerebellar oedema
- **AICA** — flocculus, anterior inferior cerebellum + brainstem; **positive HIT** (rare central exception); ipsilateral deafness + facial numbness; AICA infarction mimics vestibular neuritis closely

♥ *Normal HIT + nystagmus → CENTRAL until proven otherwise. MRI urgently if stroke is suspected — early DWI can be falsely negative in posterior fossa within 24h.*

Posterior Fossa Red Flags

- Inability to stand/walk (out of proportion to vertigo)
- Direction-changing nystagmus or vertical nystagmus
- Skew deviation (positive cover-uncover)
- Absent VOR suppression
- Ipsilateral limb ataxia
- Headache (especially occipital/nuchal)
- Dysphagia, hoarseness, Horner syndrome (Wallenberg)
- Risk factors: age >65, hypertension, atrial fibrillation, prior TIA/stroke

CHRONIC & SUBACUTE CEREBELLAR SYNDROMES

Syndrome	Key Features	Localisation	Treatment
Downbeat nystagmus (DBN)	DBN primary gaze; worse lateral/convergence; oscillopsia	Flocculus/paraflocculus Purkinje cell loss	**4-AP** (potassium channel blocker); gabapentin
Periodic alternating nystagmus (PAN)	Horizontal nystagmus reversing direction every 90–120 s	Nodulus — loss of velocity storage inhibition	**Baclofen** (GABA-B agonist) — pathognomonic response
Central positional nystagmus	DC nystagmus across Dix-Hallpike/roll; no latency; no fatigue	Nodulus/uvula lesion	Treat underlying cause
SCA6 (CACNA1A)	Late-onset ataxia; DBN; impaired VORS; misdiagnosed as BPPV	Flocculus/nodulus Purkinje cell degeneration	4-AP; genetic counselling
EA2 (CACNA1A)	Episodic ataxia (hours); interictal DBN; allelic with SCA6	Flocculus/nodulus — channel gain-of-function	**Acetazolamide** (first-line)
MSA-C	DBN; autonomic failure; poor levodopa response; rapid progression	Olivopontocerebellar degeneration	Symptomatic; fludrocortisone for OH
Chiari I	DBN; positional nystagmus; Valsalva-triggered vertigo; tonsillar herniation >5 mm	Flocculus/paraflocculus compression at foramen magnum	Surgical decompression if symptomatic
Paraneoplastic	Rapid-onset ataxia; anti-Yo (ovarian/breast), anti-Hu (lung)	Pan-cerebellar Purkinje cell loss	Treat primary tumour; immunotherapy

KEY CLINICAL PEARLS

♥ *A negative head impulse test in a patient with acute spontaneous vertigo does NOT mean peripheral disease — it increases probability of a central cause.*

♥ *PAN is baclofen-responsive by definition; if PAN resolves with baclofen, the localization is nodular until proven otherwise.*

♥ *DBN is the most common chronic central nystagmus in vestibular practice — always screen for SCA6 (genetic test) and Chiari I (MRI with cistern sequence).*