

# VESTIBULAR ANATOMY

Clinical Quick-Reference · Based on ADC Comprehensive Clinical Review

## TEMPORAL BONE & BONY LABYRINTH

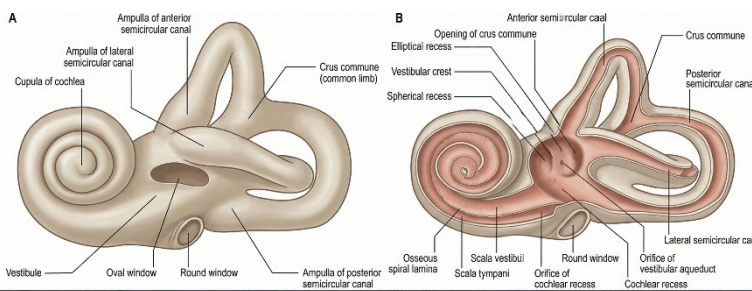
### Petrous Temporal Bone

- Hardest bone in the body; dense pyramidal structure projecting anteromedially at ~45° to sagittal plane
- Otic capsule: endochondral ossification complete before birth — virtually no Haversian remodelling throughout life
- Mastoid air cells → communicate with middle ear → coalescent mastoiditis can breach perilymphatic fissures → labyrinthitis

♥ **Otosclerosis:** persistent embryological remnants at *fissula ante fenestram* → focal bone resorption → stapes fixation. CT: perilymphatic radiolucency = *Schwartz sign* (flamingo-pink blush on otoscopy, reflecting increased vascularity of the cochlear promontory). Retrofenestral extension → sensorineural component.

### Bony Labyrinth — Key Dimensions

- **Vestibule:** 5mm AP × 3mm transverse; oval window 3.25 × 1.75mm
- **Bony canal diameter:** ~1.5mm; membranous duct only ~0.3-0.4mm (~¼ lumen)
- **SCC radii of curvature:** Horizontal & anterior ~3.2mm; posterior ~2.1mm
- Posterior canal: most dependent in upright head, inferiorly situated ampulla → otoconia migrate here → anatomical basis of posterior canal BPPV
- Lateral canal: inclined ~30° above horizontal in upright head
- **Vestibule posterior wall:** 5 openings: 3 ampullated + common crus (ant + post) + lat non-ampullated

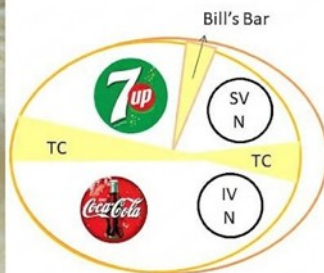
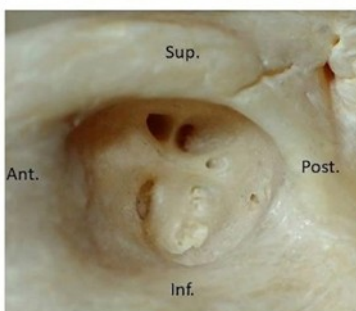


source: Drake RL, Vogt AW, Mitchell AWM. Gray's Anatomy for Students, 4th ed. Philadelphia, PA: Churchill Livingstone Elsevier; 2020.

### IAC & Aqueducts

- **IAC dimensions:** 8-10mm long, 4-5mm diameter
- **Bill's bar:** Vertical crest divides superior IAC into ant (facial nerve) and post (superior vestibular nerve) quadrants — key surgical landmark
- **Cochlear aqueduct:** Scala tympani ↔ subarachnoid space; meningogenic labyrinthitis route
- **Vestibular aqueduct:** Contains endolymphatic duct; >1.5mm at midpoint on CT → enlarged duct/sac → Pendred syndrome (SLC26A4)

## Right side - Fundus IAC



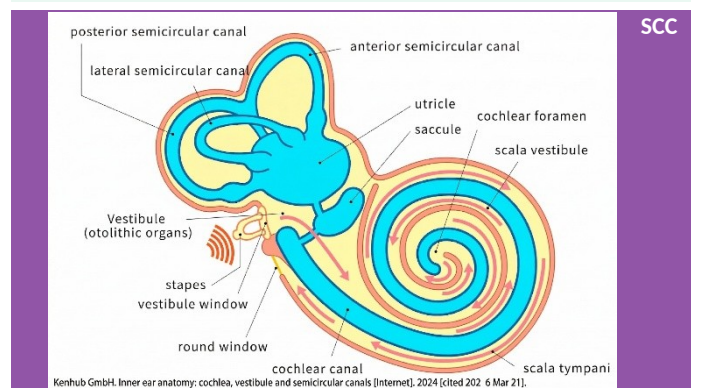
Right internal auditory canal fundus demonstrating the four-quadrant arrangement of cranial nerves VII and VIII. The falciform crest and Bill's bar divide the fundus into superior and inferior, anterior and posterior compartments. The mnemonic "7-Up, Coke down" illustrates the relative positions of the facial and cochlear nerves. Adapted from Radiopaedia and standard neuro-otology teaching sources.

## MEMBRANOUS LABYRINTH

- **SCDs:** 3 membranous ducts: ~0.3-0.4mm diameter, ~¼ of bony canal lumen
- **Utricle:** 3 × 2.5mm; elliptical recess, posterior superior vestibule; ~35,000 hair cells; macula tilted ~15-30° from horizontal → detects horizontal linear acceleration + tilt
- **Sacculle:** 3 × 1.5mm; spherical recess, anterior inferior vestibule; ~40,000 hair cells; macula in sagittal vertical plane → vertical linear acceleration + bone-conducted vibration
- **Utriculosaccular duct:** Connects utricle and sacculle → leads to endolymphatic duct
- **Ductus reuniens:** Sacculle ↔ scala media (cochlear duct); slow endolymph pressure equalisation

♥ **VEMP dissociation:** Superior vestibular neuritis → abnormal horizontal vHIT + absent oVEMP + intact cVEMP. Inferior vestibular neuritis → normal horizontal vHIT + pathological posterior vHIT + absent cVEMP + normal oVEMP. Allows precise nerve division localisation without imaging.

♥ **Tullio phenomenon (sound-induced vertigo):** SSCD creates a third window → sound energy shunts into vestibular labyrinth → activates sacculle + anterior canal. cVEMP threshold reduced (<80dBnHL), oVEMP amplitude increased. Confirmed by sub-mm CT temporal bones (Pöschl + Stenvers projections).



### GEOMETRY, EWALD'S LAWS & CALORIC TESTING

- **Coplanar pairs:** Lateral || Lateral | Anterior ↔ contralateral Posterior (three orthogonal planes)
- **Ewald's 1st Law:** Eye movement is in the plane of the stimulated canal
- **Ewald's 2nd Law:** Ampullopetal flow in lateral SCC excites (ampullofugal in vertical SCCs excites) — asymmetric response
- **Ewald's 3rd Law:** Excitatory stimulus > inhibitory stimulus — basis of canal paresis asymmetry

### Caloric Testing

- Lateral canal stimulated exclusively (near-horizontal, superficial position)
- Warm 44°C → endolymph less dense → rises → ampullopetal → nystagmus toward irrigated ear
- Cold 30°C → endolymph denser → sinks → ampullofugal → nystagmus away from irrigated ear
- COWS mnemonic: Cold Opposite, Warm Same
- **Jongkees CP formula:** CP = [(RW+RC)-(LW+LC)] / (RW+RC+LW+LC) × 100%; >25% = unilateral canal paresis
- Calorics probe ~0.003Hz; vHIT probes 5-15Hz — paradoxical: absent caloric + normal vHIT = incomplete neuritis or early Menière's, NOT complete lesion

**SENSORY END-ORGANS**

**Crista Ampullaris**

- **Size:** Saddle-shaped ridge ~2-3mm height; ~7,000 hair cells per crista
- **Cupula:** Glycoprotein-rich (otogelin); specific gravity ~1.001-1.004 = endolymph (~1.003) → gravity-neutral
- Neutral buoyancy: cupula responds to angular acceleration via endolymph inertia — NOT to gravity under normal conditions
- Cupulolithiasis (BPPV): otoconia adhere to cupula → heavier than endolymph → gravity-sensitive → persistent, non-fatigable nystagmus (no latency, no decay)
- Canalithiasis: free otoconia in duct → transient fatigable nystagmus with 1-5s latency
- Central crista (summit): Type I hair cells (calyx), irregular afferents, high gain — probed by vHIT (5-15Hz)
- Peripheral crista (slopes): Type II hair cells (bouton), regular afferents, lower gain — probed by calorics + rotational chair
- Horizontal crista polarisation: kinocilium faces utricle (ampullopetal = excitatory); vertical cristae: kinocilium away from utricle (ampullofugal = excitatory) → basis of Ewald's 2nd & 3rd Laws

**Maculae & Otolithic Membrane**

- **Otoconia:** Calcium carbonate crystals (calcite), hexagonal prisms 3-30µm; specific gravity ~2.71 vs endolymph ~1.003
- **Otoconin-90 (Oc90):** Regulates crystal nucleation; Otopetrin-1 (OTOP1) for seeding
- Age-related otoconial degeneration → fragmentation → BPPV
- **Striola:** 80-100µm wide; curved zone of polarisation reversal across each macula
- Utricular macula: hair cells polarised TOWARD striola on both sides → excites downside + inhibits upside during tilt
- Saccular macula: hair cells polarised AWAY from striola; striola runs ~35° from vertical → detects vertical + some horizontal vectors
- Striola properties: Type I cells predominate; phasic/high-dynamic; thinner gel layer; sparser otoconia

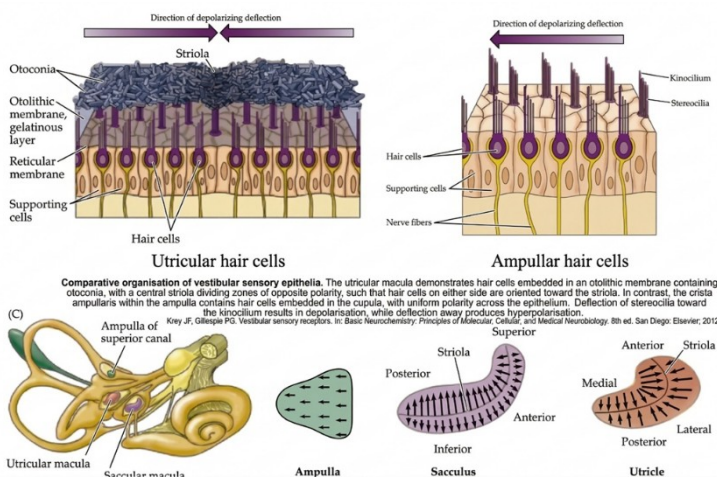
- Calyx synapse — dual transmission: (1) Quantal (vesicular glutamate, ribbon synapse, 0.5-1.0ms delay); (2) Non-quantal (K<sup>+</sup> accumulates in restricted calyx cleft → direct calyx depolarisation, microsecond range — enables >50Hz signals)
- Ribbon synapse: Cav1.3 L-type Ca<sup>2+</sup> channels cluster around ribbon → sustained glutamate release → maintains ~90 spikes/s tonic CN VIII discharge
- ♥ **Non-quantal transmission (NQT):** *otoferlin-knockout mice (no vesicular release) retain partial vestibular but no auditory function — calyx geometry is the structural prerequisite; cochlear inner hair cells cannot perform NQT as they lack the enclosed calyx.*

**Efferent Vestibular System**

- **Origin:** ~300-400 neurons/side; medial EVN + lateral EVN, adjacent to CN VI nucleus, pons
- **Course:** Travel with CN VIII; cross within brainstem → superior + inferior vestibular nerves
- **Targets:** Type II hair cells (bouton); outer surface of Type I calyx; afferent bouton endings
- **Neurotransmitter:** ACh (α9/α10 nicotinic) → Ca<sup>2+</sup> entry → SK channel activation → K<sup>+</sup> efflux → HYPERPOLARISATION; co-transmitters: CGRP, GABA, enkephalin
- Efference copy / active suppression: EVS activated slightly BEFORE voluntary head movement → suppresses expected reafference → reduces VOR gain for self-generated head turns (desirable)
- Protective: during high-intensity sustained stimulation → limits receptor potential → reduces excitotoxic afferent damage
- ♥ **Active vs passive:** *VOR gain to active head movement is slightly lower than to passive movement of same velocity. Normal passive HIT but symptoms only during active motion → consider efference copy failure or central EVS dysfunction.*

**VASCULAR ANATOMY**

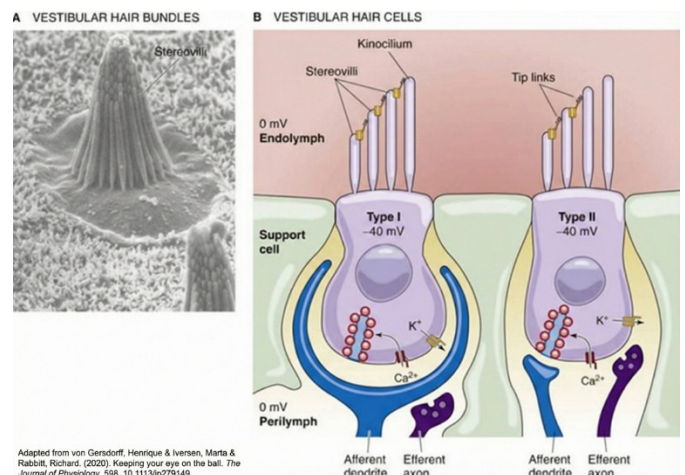
- Inner ear = single terminal vascular system with NO collateral circulation → immediate irreversible infarction
- **Labyrinthine artery origin:** AICA (~85%); direct basilar artery (~15%); travels with CN VII+VIII in IAC
- **AICA loop in IAC:** 10-15% of individuals; neurovascular compression → vestibular paroxysmia: brief stereotyped attacks, respond to carbamazepine/oxcarbazepine
- **Anterior Vestibular Artery (AVA):** Smaller branch → superior vestibular nerve, utricle, anterior + lateral canal ampullae. Virtually no anastomotic potential.
- **Common Cochlear Artery (CCA):** Larger branch → cochlea (spiral modiolar artery) + Posterior Vestibular Artery (PVA) → saccule, posterior canal ampulla, inferior utricle
- **AVA territory:** Matches superior division of Scarpa's ganglion exactly
- **Venous drainage:** Vein of Vestibular Aqueduct (VVA): drains SCCs + utricle → inferior petrosal sinus / jugular bulb. VVA adjacent to endolymphatic sac — obstruction → backpressure → secondary hydrops



**S7 · HAIR CELLS — ULTRASTRUCTURE**

Feature	Type I	Type II
Shape	Flask/goblet	Cylindrical
Innervation	Calyx afferent (enclosed)	Bouton afferents
Location	Centre/striola (summit)	Periphery/slopes
Afferent discharge	Irregular (high CV) — high gain	Regular (low CV) — tonic
Function	Dynamic phasic; vHIT	Sustained tonic; caloric range
Efferent synapse	On outer calyx surface	Direct on cell body

- Tip links: PCDH15 (lower) + CDH23 (upper) — gate MET channels; mutations → Usher syndrome type I



## SCARPA'S GANGLION & VESTIBULAR NERVE

- **Location:** Midpoint of IAC; bilobed; strict somatotopic organisation
- **Superior division:** Anterior canal ampulla + lateral canal ampulla + utricle → superior posterior IAC quadrant (behind Bill's bar)
- **Inferior division:** Posterior canal ampulla + saccule → inferior posterior IAC quadrant
- Fibres enter brainstem at pontomedullary junction → bifurcate: ascending → SVN + MVN; descending → MVN + LVN + DVN; small proportion → cerebellum (juxtarestiform body)
- **Irregular afferents:** High CV, high gain, phasic — encode dynamic head motion (vHIT frequency range); project to SVN/MVN for VOR
- **Regular afferents:** Low CV, low gain, tonic — encode sustained static tilt; project to LVN for postural control

♥ **Superior vestibular neuritis (SVN, ~90%):** superior CN VIII division preferentially affected (narrower bony canal, less vascular supply, longer intraosseous course). HSV-1 reactivation in superior Scarpa's ganglion. Signs: horizontal-torsional nystagmus fast phase away from lesion; pathological horizontal + anterior vHIT; absent oVEMP; INTACT cVEMP. Treatment: methylprednisolone 100mg tapering over 3 weeks within 3 days (Strupp et al., NEJM 2004). Antiviral therapy NOT recommended.

♥ **Inferior vestibular neuritis (IVN, ~10%):** posterior canal + saccule only. Normal horizontal vHIT; pathological posterior vHIT; absent cVEMP; normal oVEMP

## VESTIBULAR NUCLEAR COMPLEX (VNC)

- Occupies floor of 4th ventricle: lower pons to upper medulla. Only ~25% of VNC input is from vestibular nerve — remainder from cerebellum, contralateral VNC, reticular formation, spinal cord.

Nucleus	Eponym	Key Outputs
Superior	Bechterew	MLF → oculomotor; ipsilateral VOR arc
Medial	Schwalbe	MLF bilateral; MVST; horizontal + vertical VOR; commissural fibres
Lateral	Deiters'	LVST ipsilateral → limb extensors; postural control; giant Deiters' cells
Inferior	Spinal/Roller	Cerebellum ↔ nodulus/uvula; contralateral commissure; autonomic relay

### Functional Neuron Classes

- **PVP neurons:** Position-Vestibular-Pause: primary VOR premotor interneurons; fire ∝ head velocity; PAUSE during saccades (preventing VOR interference with voluntary gaze shifts)
- **VO neurons:** Vestibular-Only: encode head velocity; project bilaterally to cerebellum + other VNC; critical for velocity storage; do NOT pause during saccades
- **FTN neurons:** Floccular Target Neurons (SVN + MVN): receive direct floccular Purkinje cell inhibition → effector neurons of VOR gain adaptation
- **Type I neurons:** Excited by ipsilateral horizontal canal → project to contralateral CN VI via MLF (excitatory limb of VOR push-pull)
- **Type II neurons:** Inhibited by ipsilateral, excited by contralateral via commissure → inhibitory limb → sharpens directional tuning

### Accessory Groups

- **Group Y:** Dorsolateral to restiform body; interface between flocculus and CN III/IV → vertical VOR + floccular smooth pursuit
- **NPH (Nucleus Prepositus Hypoglossi):** Neural integrator for horizontal eye movements: converts VOR velocity command → sustained position-holding signal. NPH lesion → gaze-evoked nystagmus (eye drifts to primary position after each saccade)

### Commissural System

- Rich system of commissural fibres crosses brainstem midline — anatomical substrate for bilateral push-pull
- Inhibitory commissural fibres (GABA-ergic + glycinergic): dominant; ipsilateral VNC excitation → simultaneously suppresses contralateral VNC → sharpens directional tuning, doubles dynamic range
- Excitatory commissural fibres (glutamatergic): mediates velocity storage + bilateral synchronisation.

## VESTIBULOCEREBELLAR INTEGRATION

### Flocculus/Paraflocculus (VOR adaptation + smooth pursuit)

- **Mossy fibre inputs:** From VNC (MVN, SVN), NPH, pontine nuclei (visual signals)
- **Climbing fibre inputs:** From dorsal cap of inferior olive — error signal: fires when retinal slip detected (VOR gain inaccurate)
- **Purkinje cell output:** GABAergic inhibition onto FTNs in MVN/SVN — the pathway through which the flocculus adjusts VOR gain
- **LTD mechanism:** Climbing fibre (Ca<sup>2+</sup> rise) + parallel fibre (mGluR1/AMPA) co-activation → PKC → AMPA receptor internalisation → reduced Purkinje inhibition → increased VOR gain (or reverse)

### Nodulus/Uvula (velocity storage + gravity disambiguation)

- **Velocity storage:** Raw cupula signal time constant ~6s; nodulus/uvula extends to ~16–20s via VO neuron recurrent circuits; nodulus Purkinje cells → DVN/MVN → regulate via "tilt dumping" when otolith signal detected
- Nodulus lesion → pathologically prolonged velocity storage → Periodic Alternating Nystagmus (PAN) or prolonged post-rotatory nystagmus
- **Tilt/translation disambiguation:** Einstein's equivalence: otolith output cannot distinguish tilt from linear acceleration alone. Nodulus integrates canal signals (rotation but not tilt) with otolith signals to resolve ambiguity. Nodulus lesion → misperceive translation as tilt.

♥ **Cerebellar nystagmus signs:** Downbeat nystagmus (floccular/paramedian lesion) — causes: degeneration, Arnold-Chiari, B12 deficiency, lithium toxicity; treat with baclofen/4-aminopyridine. Upbeat nystagmus (anterior vermis/medulla/paramedian tract) — consider Wernicke's; give thiamine. PAN (nodulus) — baclofen (GABA-B agonist). Gaze-evoked nystagmus (NPH or diffuse cerebellar) — sedatives, anticonvulsants. Key distinction: cerebellar nystagmus WORSENS with fixation; peripheral nystagmus is SUPPRESSED by fixation.

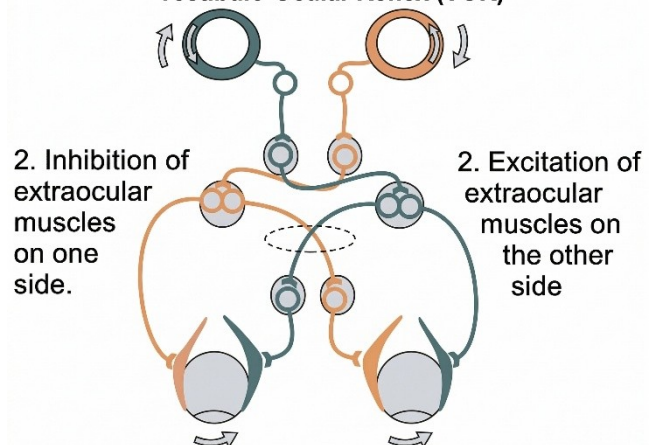
## VOR — THREE-NEURON ARC

- **Latency:** 7–15ms (far shorter than visual feedback ~100ms)
- **Gain:** 0.8–1.2 normal; <0.6 on vHIT = significant unilateral hypofunction
- **Bandwidth:** 0.05–>10Hz; vHIT = high-frequency end (5–15Hz); calorics = ~0.003Hz

### Horizontal Arc (rightward head rotation example)

- 1st neuron: R lateral SCC → irregular afferents → Scarpa's (R, superior division) → R CN VIII → R VNC (MVN, SVN)
- 2nd neuron (excitatory): R MVN → crosses midline via PPRF → L CN VI nucleus (abducens) + via L MLF → R CN III nucleus (medial rectus). Result: L lateral rectus + R medial rectus contract → eyes move left
- 2nd neuron (inhibitory): R MVN → inhibitory interneurons → R CN VI inhibited; commissural fibres → L MVN inhibited
- 3rd neuron: CN VI motor neurons → L lateral rectus; CN III motor neurons → R medial rectus
- **MLF:** Paired white matter tract in dorsal tegmentum pons/midbrain, adjacent to midline; carries: horizontal VOR internuclear signal, vertical VOR (SVN → III/IV), MVST (continues caudally to cervical cord)

### Schematic of the Three-Neuron Arc of the Horizontal Vestibulo-Ocular Reflex (VOR)



Detection of head rotation by the semicircular canals generates an excitatory signal to extraocular muscles on one side and a reciprocal inhibitory signal on the contralateral side, producing compensatory eye movement opposite to head rotation. This push-pull organisation underlies the three-neuron arc of the horizontal vestibulo-ocular reflex.

Adapted from: Vestibulo-ocular reflex. Wikipedia [Internet]. [cited 2026 Mar 21]. Available from: [https://en.wikipedia.org/wiki/Vestibulo-ocular\\_reflex](https://en.wikipedia.org/wiki/Vestibulo-ocular_reflex)

## VESTIBULOSPINAL TRACTS

- **LVST:** Origin: Deiters' nucleus (largest neurons in brainstem — giant Deiters' cells). Course: ipsilateral ventral funiculus, all spinal levels. Function: excites extensor (antigravity) motor neurons. Somatotopy: dorsal LVN → cervical; ventral LVN → lumbosacral. Primary input: UTRICLE (static head tilt)
- **MVST:** Origin: MVN + DVN. Course: bilateral within MLF → cervical + upper thoracic cord. Function: vestibulocollic reflex (VCR) — stabilises head in space after sudden displacement. Input: SCC cristae

♥ **Wallenberg syndrome (PICA infarction):** lateral medulla — damages VNC (lat + inf) + LVST/MVST origins + nucleus ambiguus (IX/X) + sympathetic fibres (Horner's) + spinal trigeminal (ipsilateral facial hemi-hypoalgesia) + spinothalamic (contralateral body hemi-hypoalgesia). Lateropulsion (fall toward lesion) = asymmetric LVST activation. Ocular Tilt Reaction: ipsilateral head tilt + ipsilateral hypotropia + ipsilateral torsion + ipsilateral-tilted SVV. HINTS: normal HIT (horizontal canal preserved), direction-changing/torsional nystagmus, POSITIVE skew deviation = central pattern → urgent neuroimaging.

## VESTIBULAR CORTEX

- No single primary vestibular cortex — distributed thalamo-cortical network around Sylvian fissure
- **Thalamic relays:** VPI + VPL (core relay → PIVC); VL nucleus (→ motor cortex, postural planning); Po (→ insula); Pulvinar (visual-vestibular integration → PPC)
- **PIVC:** Parieto-insular vestibular cortex: primary cortical hub (temporoparietal junction)
- **PIVC ↔ V5/MT:** Reciprocally inhibitory: PIVC activation suppresses visual motion cortex and vice versa — resolves sensory conflict during self-motion. Failure of this cortical suppression → visual sensitivity → PPPD mechanism
- **Right hemisphere dominance:** Greater caloric/galvanic activation in right hemisphere (right-handers)

Posterior Parietal Cortex (PPC, area 7 + VIP): body schema construction. PPC lesions: contralateral hemineglect (right PPC); pusher syndrome (actively pushes toward weak side — internal vertical reference shifted — right PPC or thalamic)

## VESTIBULO-AUTONOMIC INTEGRATION

- **RVLM:** Rostral ventrolateral medulla: master sympathetic vasomotor centre; receives DVN + MVN input → vestibulo-sympathetic reflex (VSympR)
- **NTS:** Nucleus of Solitary Tract: vagal sensory relay; receives MVN input → converges vestibular + visceral → Dorsal Motor Nucleus CN X + area postrema → nausea/vomiting of vestibular disturbance
- **Locus Coeruleus:** Noradrenergic; receives VNC + DVN input → arousal + anxiety. Neuroanatomically determined anxiety during acute vestibular loss — not purely psychological
- **Parabrachial nucleus:** Pontine visceral relay → amygdala → fear/anxiety; mediates motion sickness + vestibular-anxiety comorbidity

### Vestibulo-Sympathetic Reflex (VSympR)

- Feedforward CV control: utricular maculae detect head-up tilt → superior vestibular nerve → DVN/MVN → RVLM → IML of thoracic cord → sympathetic ganglia → peripheral vasoconstriction
- Unlike baroreceptor reflex (FEEDBACK — detects drop then corrects), VSympR PREDICTS haemodynamic consequence of orthostasis and pre-empts it
- Bilateral vestibular failure → impaired VSympR → orthostatic hypotension even without autonomic neuropathy

### Motion Sickness Circuit

- Sensory conflict computed at VNC (VO neurons, commissural system, cerebellar comparator)
- VNC → NTS → (1) Dorsal Motor Nucleus CN X → gastric hypomotility + bradycardia; (2) Area postrema (chemoreceptor trigger zone, outside BBB) → emesis.

## CLINICAL SYNDROME LOCALISATION (from ADC Comprehensive Clinical Review)

Syndrome	Anatomical Site (from ADC Review)	Nystagmus	Key Clinical Signs	Bedside Test
BPPV posterior (canalithiasis)	Posterior SCC duct — otoconial fragments from utricular macula	Torsional-vertical, geotropic, fatigable; latency 1–5s, <60s	Position-triggered; resolves spontaneously	Dix-Hallpike +ve
BPPV posterior (cupulolithiasis)	Posterior SCC cupula — otoconia adherent	Torsional-vertical, non-fatigable, NO latency, persistent	Gravity-sensitive cupula (heavier than endolymph)	Dix-Hallpike: no fatigue
Superior Vestibular Neuritis	Superior division CN VIII / superior Scarpa's ganglion; AVA territory	Horizontal-torsional, unidirectional, fast phase away from lesion	Pathological horiz + ant vHIT; absent oVEMP; intact cVEMP + post vHIT	vHIT + VEMP dissociation
Inferior Vestibular Neuritis	Inferior division CN VIII; PVA territory (sacculae + post canal)	Absent spontaneous nystagmus or subtle torsional	Normal horiz vHIT; pathological post vHIT; absent cVEMP; normal oVEMP	Posterior vHIT + cVEMP
Menière's Disease	Membranous labyrinth — endolymphatic hydrops; sacculae + cochlear duct	Horizontal (acute attack)	Episodic vertigo + fluctuant SNHL + tinnitus + aural fullness; sacculae balloons toward stapes footplate	cVEMP + ECochG + 3T MRI gadolinium
SSCD / Tullio Phenomenon	Anterior SCC roof dehiscence — third window effect	Vertical-torsional triggered by sound/pressure (Tullio)	Autophony; cVEMP threshold reduced; oVEMP amplitude increased	CT temporal bone (Pöschl + Stenvers)
Vestibular Paroxysmia	AICA loop compressing CN VIII in IAC (10–15% anatomical variant)	Brief, stereotyped attacks; seconds	Brief attacks; respond to carbamazepine/oxcarbazepine	MRI FIESTA showing neurovascular compression
Wallenberg (PICA)	Lateral medulla: VNC (lat+inf) + LVST + sympathetics + nucleus ambiguus + spinothalamic	Ipsipulsive; direction-changing or torsional; beats ipsilateral	Lateropulsion; Horner's; dysphagia; OTR (tilt + skew + torsion); SVV tilted ipsilaterally	HINTS: normal HIT + pos skew = CENTRAL
INO	MLF lesion between CN VI and III nuclei — same side as adduction failure	Abducting nystagmus in contralateral eye	Ipsilateral adduction failure; convergence preserved; younger = MS, older = lacunar	Cover test; horizontal pursuit
Thalamic Astasia	Posterolateral thalamus VPL/VPI lacunar infarct — graviceptive thalamus	None specific	Profound postural instability despite normal power/coordination/sensation; SVV tilted contralaterally	MRI DWI; bucket/SVV test
PAN (nodulus lesion)	Nodulus/uvula — failure of velocity storage regulation	Direction reverses every 90–120s	Truncal ataxia; no limb ataxia; tilt/translation misperception	Observe: spontaneous direction reversal

Source: Australian Dizziness Clinic Team. Anatomy of the Vestibular System: A Comprehensive Clinical Review. v1.0 April 2026.