Anatomy of Vestibular System:
A Comprehensive Clinical Review

Companion to: The Physiology of the Vestibular System

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How to Use This Document

This anatomy review is the companion to the Physiology of the Vestibular System document. Structure follows peripheral to central organisation. Where physiology concepts arise — mechanotransduction, reflex arcs, adaptation — the text cross-references the companion document rather than repeating detailed physiological derivations. Cross-reference boxes appear throughout:

> *Companion reference: see Physiology document, Section X — for the physiological basis of the concept described here.*

Clinical Pearl boxes highlight anatomy-to-bedside correlations. Key Facts tables at section ends provide rapid-review summaries. Self-assessment questions at the close of the document are clinically framed and test integrative anatomy-physiology understanding.

Learning Objectives

On completing this review, the reader should be able to:

- Describe the gross and microanatomy of the bony and membranous labyrinths, including the dimensions, orientation, and fluid compartments of each component.
- Explain the functional geometry of the semicircular canals, including Ewald's Laws and the basis of caloric testing, with reference to ampullary anatomy.
- Detail the structure of the crista ampullaris and macula and describe the striola's role in encoding linear acceleration direction.
- Differentiate Type I and Type II hair cells structurally, ultrastructurally, and functionally, and explain the significance of the calyx synapse.
- Trace the complete arterial supply of the inner ear from the basilar artery to end-organ level and predict the precise clinical syndrome resulting from occlusion at each branch point.
- Describe the internal auditory canal compartments (Bill's bar, four-quadrant anatomy), the topographic organisation of Scarpa's ganglion, and the clinical basis of superior versus inferior vestibular neuritis.
- Name the four major vestibular nuclei with their eponyms, locations, primary inputs, projection tracts, and lesion syndromes.
- Describe the efferent vestibular system — its origin, target, neurotransmitter, and functional significance.
- Trace the three-neuron arc of the horizontal VOR from hair cell to extraocular muscle, identify the MLF as its anatomical backbone, and localise lesions along this pathway.
- Describe the lateral and medial vestibulospinal tracts: origins, courses, targets, and reflex functions.
- Explain the anatomy of the vestibulocerebellum (flocculus versus nodulus/uvula) and its distinct roles in VOR adaptation versus velocity storage and tilt/translation disambiguation.
- Localise central vestibular lesions based on clinical syndrome — Wallenberg, AICA infarction, INO, skew deviation, thalamic astasia — using anatomical reasoning.

Table of Contents

1. Introduction

The vestibular apparatus is one of the most phylogenetically ancient sensory systems in vertebrate biology. Its fundamental architecture — a set of fluid-filled canals and otolith organs enclosed within a dense osseous housing — has been conserved across 500 million years of evolution, from jawless fish to humans [1]. This conservation reflects the immutability of its task: the detection of gravitational force and inertial acceleration, physical constants to which every organism must respond.

Clinically, the vestibular system presents a unique challenge: it operates almost entirely below the level of consciousness, yet its failure produces some of the most disabling and frightening symptoms in medicine. Vertigo, oscillopsia, imbalance, and the cascade of autonomic symptoms that accompany acute vestibular loss collectively account for approximately 5% of all primary care consultations and a disproportionate burden of emergency presentations [2]. Despite this, the vestibular system remains one of the least formally taught areas in medical education.

The anatomy of the vestibular system is inseparable from its function. Understanding why the posterior semicircular canal is the most common site of BPPV, why AICA infarction causes hearing loss and Wallenberg does not, why superior vestibular neuritis spares the saccule, or why a thalamic lacune can produce an inability to stand — all of these require a precise structural map. This document provides that map.

This review is explicitly a companion to *The Physiology of the Vestibular System* (companion document). Biomechanical derivations, mechanotransduction kinetics, and reflex gain equations are covered there. Here the focus is structure: dimensions, orientations, vascular territories, nuclear boundaries, and projection pathways, with physiological concepts invoked only where they illuminate anatomy.

 Companion reference: Physiology document, Section 1 (Introduction) — system overview and signal transformation sequence.

2. The Temporal Bone and Bony Labyrinth

2.1 The Petrous Temporal Bone

The vestibular apparatus is housed within the petrous temporal bone (pars petrosa), the hardest bone in the human body — a dense pyramidal structure projecting anteromedially from the skull base at approximately 45° to the sagittal plane [3]. Its extreme density reflects the unique biology of the otic capsule: endochondral ossification completed before birth, with minimal subsequent remodelling. This confers robust mechanical protection for the delicate membranous labyrinth.

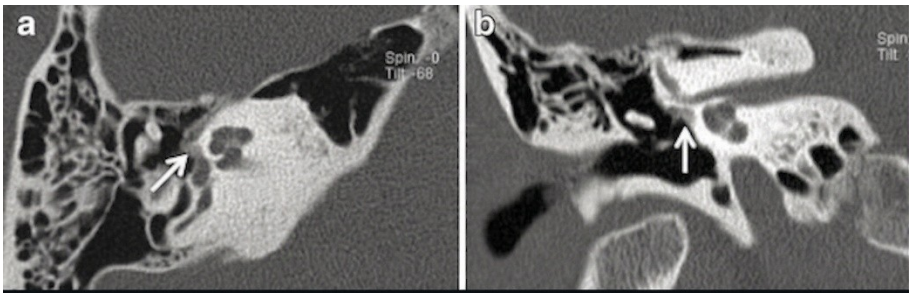
The otic capsule itself is a shell of endochondral bone with three important clinical properties:

- **Minimal remodelling throughout life:** Unlike virtually all other skeletal bone, the otic capsule undergoes no physiological Haversian remodelling after foetal ossification. This confers resistance to the infiltrative and metabolic bone diseases that remodel other skeletal sites throughout life but paradoxically renders the capsule vulnerable to otosclerosis — see Clinical Pearl below.
- **Pneumatisation:** The temporal bone surrounding the otic capsule is extensively pneumatised — riddled with air-filled spaces called mastoid air cells, which are lined by respiratory-type epithelium and communicate with the middle ear via the aditus ad antrum. The otic capsule itself is not pneumatised and remains a solid endochondral shell. However, the mastoid air cells lie in close anatomical proximity to the capsule, separated only by thin bony partitions and perilabyrinthine fissures. When mastoid infection becomes coalescent — meaning the partitions between air cells are destroyed by spreading osteitis — the infection can breach these fissures and reach the labyrinth directly, causing labyrinthitis. This is the anatomical basis of labyrinthine complications in acute mastoiditis: not invasion of the capsule itself, but bacterial or inflammatory access through the fissures at its periphery.

♥ CLINICAL PEARL: Otosclerosis — otic capsule anatomy and clinical correlates

The same absence of Haversian remodelling that protects the otic capsule from most bone diseases makes it uniquely vulnerable to otosclerosis. Persistent embryological remnants — particularly at the fissula ante fenestram, a small fibrous-cartilaginous cleft anterior to the oval window — harbour foci of latent osteoclast activity. When aberrantly activated, these drive focal bone resorption and disorganised vascular woven bone deposition entirely unopposed by any normal remodelling response. The result is a self-propagating otospongiotic lesion that progressively fixes the stapes footplate, and in retrofenestral disease, disrupts the cochlear endosteum and spiral ligament vascularity, producing the mixed conductive-sensorineural hearing loss of advanced otosclerosis.

On high-resolution CT, the active phase produces a characteristic **perilabyrinthine radiolucency** — a hypodense halo most conspicuous at the fissula ante fenestram,



Axial HRCT images demonstrating hypodense demineralised plaques at the fissula ante fenestram consistent with fenestral otosclerosis.
source- "Purohit B, Hermans R, Op de Beeck K. Imaging in otosclerosis: a pictorial review. Insights Imaging. 2014;5(2)245–252. doi:10.1007/s13244-014-0313-9"

reflecting focal demineralisation replaced by vascular spongy woven bone. This is the radiological correlate of the **Schwartze sign:** the flamingo-pink blush visible through the tympanic membrane on otoscopy, caused by increased vascularity shining through the cochlear promontory. Active perilabyrinthine lucency suggests ongoing disease amenable to sodium fluoride therapy; its absence implies the quiescent sclerotic phase. Retrofenestral extension — involvement of the cochlear endosteum — signals a sensorineural component and portends a less favourable outcome from stapes surgery.

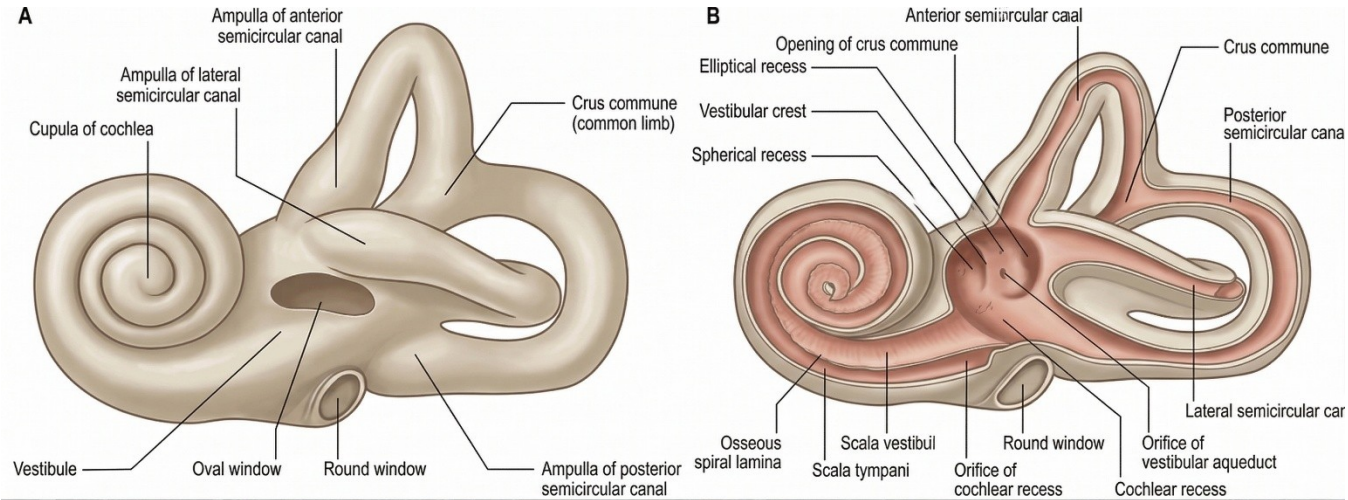
2.2 The Bony Labyrinth — Architecture

The bony labyrinth is a series of interconnected perilymph-filled cavities within the otic capsule. It comprises three components: the vestibule, the semicircular canals, and the cochlea.

2.2.1 The Vestibule

The vestibule is the central ovoid chamber of the bony labyrinth, approximately 5 mm in the anteroposterior dimension and 3 mm transversely. It occupies the space between the cochlea anteriorly and the semicircular canals posteriorly. Several anatomically critical features:

- **Lateral wall:** Bears the oval window (fenestra vestibuli), approximately 3.25 × 1.75 mm, closed by the stapes footplate and annular ligament. Otosclerosis classically fixes this footplate, causing conductive hearing loss. See Clinical Pearl: Otosclerosis, Section 2.1.
- **Medial wall:** Corresponds to the fundus of the internal auditory canal (IAC). Contains the elliptical recess (housing the utricle) superiorly and the spherical recess (housing the saccule) inferiorly, separated by the vestibular crest. Perforated by branches of CN VIII.
- **Inferior wall:** The round window niche (fenestra cochleae), closed by the secondary tympanic membrane, opens to the scala tympani of the cochlea. Allows pressure equalisation across the perilymphatic space. The round window membrane also serves as a route of entry for pathogens causing labyrinthitis, and as a pathway for intratympanic drug delivery.
- **Posterior wall:** Five openings from the three semicircular canals: each canal contributes one ampullated end (three total), while the non-ampullated ends of the anterior and posterior canals merge to form the common crus, reducing their two non-ampullated openings to one. The lateral canal contributes its own non-ampullated opening separately, giving five openings in total.

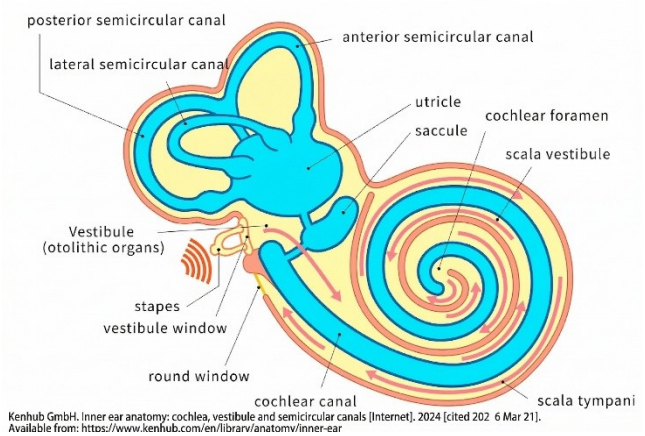


source- "Drake RL, Vogl AW, Mitchell AWM. Gray's Anatomy for Students. 4th ed. Philadelphia, PA: Churchill Livingstone Elsevier; 2020."

2.2.2 The Semicircular Canals — Bony Architecture

The bony semicircular canals are the C-shaped osseous tubes that house the membranous semicircular ducts. Key dimensions and relationships:

- **Dimensions:** Each bony canal is approximately 1.5 mm in internal diameter. The membranous duct occupies only ~1/4 to 1/3 of this lumen cross-section (~0.3–0.4 mm diameter), suspended within the perilymph-filled space.
- **Radii of curvature:** Horizontal canal ~3.2 mm; anterior canal ~3.2 mm; posterior canal ~2.1 mm. Larger radii confer greater sensitivity to angular acceleration (see companion Physiology document).
- **Anterior canal:** The highest-positioned canal, arches superiorly. Its non-ampullated arm joins the posterior canal's non-ampullated arm to form the common crus (crus commune) before entering the posterior vestibule. Its arch is occasionally visible through the tegmen — the thin bone forming the roof of the middle ear.
- **Posterior canal:** The most dependent canal in the upright head. Its ampullated end opens inferiorly into the vestibule, near the round window niche. **This dependent position and inferiorly situated ampulla explain why otoconia preferentially migrate here — the anatomical basis of posterior canal BPPV.**
- **Lateral (horizontal) canal:** Inclined approximately 30° above horizontal in the upright head. Its posterior non-ampullated end opens into the posterior vestibule; its anterior ampullated end opens into the vestibule just below the oval window.



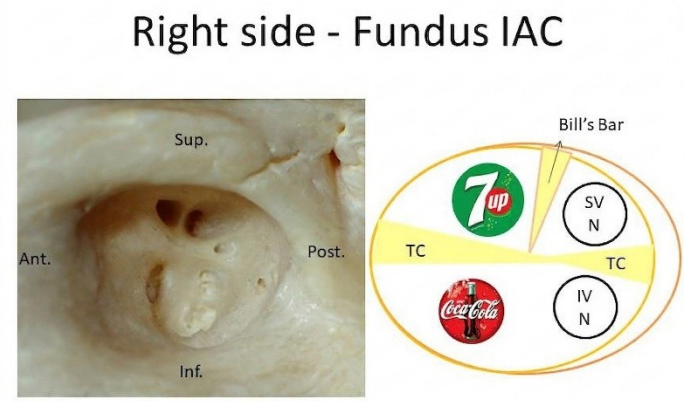
Kenhub GmbH. Inner ear anatomy: cochlea, vestibule and semicircular canals [Internet]. 2024 [cited 202 6 Mar 21]. Available from: https://www.kenhub.com/en/library/anatomy/inner-ear

Companion reference: Physiology document, Section 2.1 — canal biomechanics, torsion pendulum model, and time constants.

2.2.3 The Internal Auditory Canal (IAC)

The IAC is the bony channel through which CN VII (facial nerve) and CN VIII (vestibulocochlear nerve) pass from the brainstem to the inner ear. It averages 8–10 mm in length and 4–5 mm in diameter [4]. The fundus (lateral end) is the critical anatomical landmark for understanding vestibular nerve anatomy:

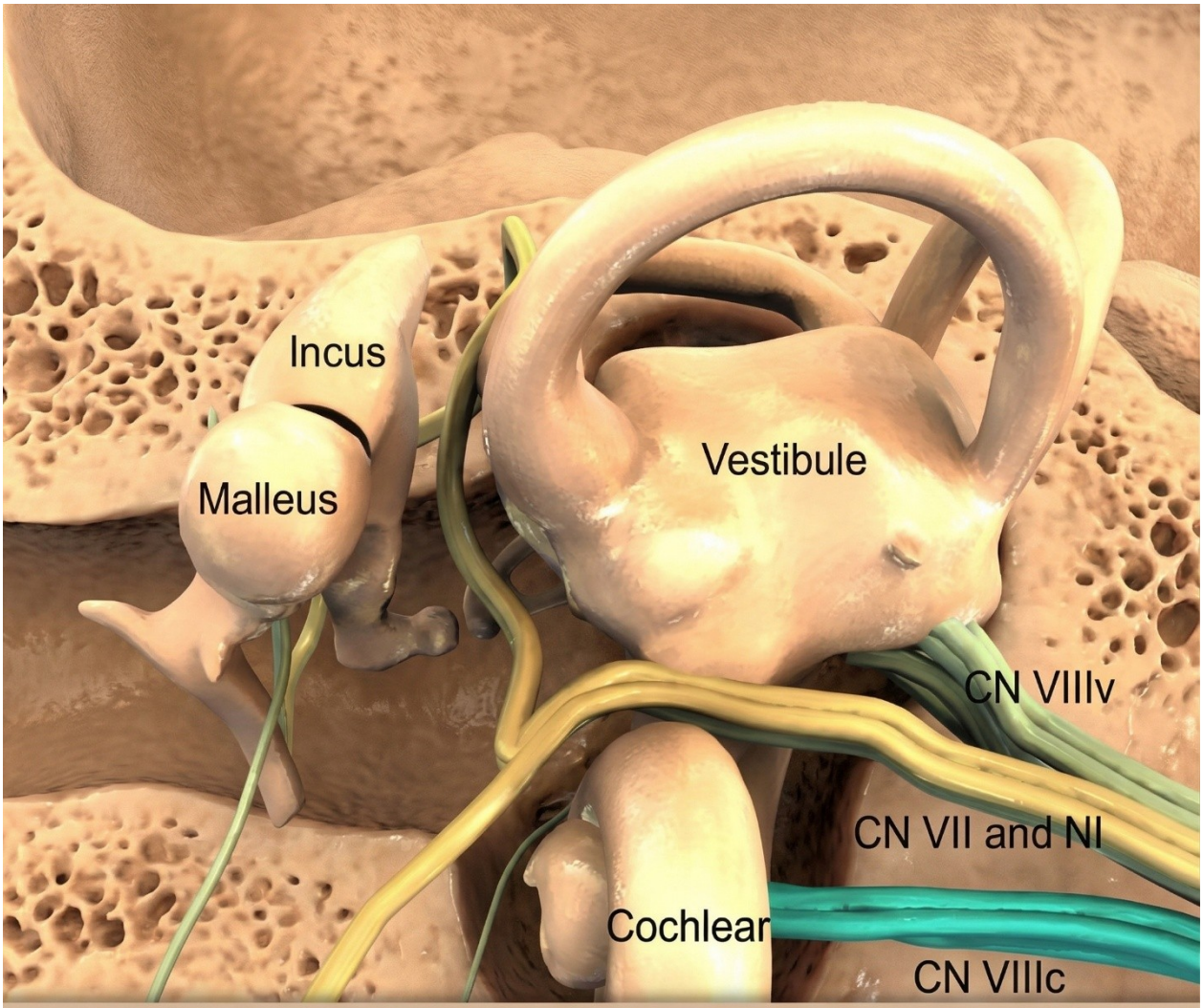
- **Falciform crest (crista falciformis):** A horizontal bony shelf dividing the IAC fundus into superior and inferior halves.
- **Vertical crest (Bill's bar):** A vertical ridge in the superior half, first described by William House, dividing it into anterior and posterior quadrants. It is the key surgical landmark used to identify and protect the facial nerve during vestibular neurectomy via the posterior cranial fossa approach



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.

The four-quadrant arrangement of the IAC fundus:

Quadrant	Contents	Clinical Note
Superior Anterior	Facial nerve (CN VII)	Preserved in selective vestibular neurectomy via posterior cranial fossa approach

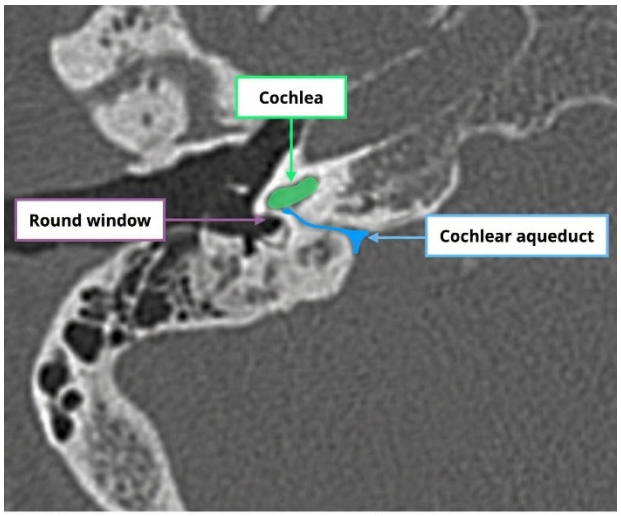


Three-dimensional representation of the middle and inner ear demonstrating the ossicular chain (malleus, incus), vestibule, cochlea, and cranial nerves VII and VIII within the temporal bone. Adapted from the University of Dundee 3D Ear model.

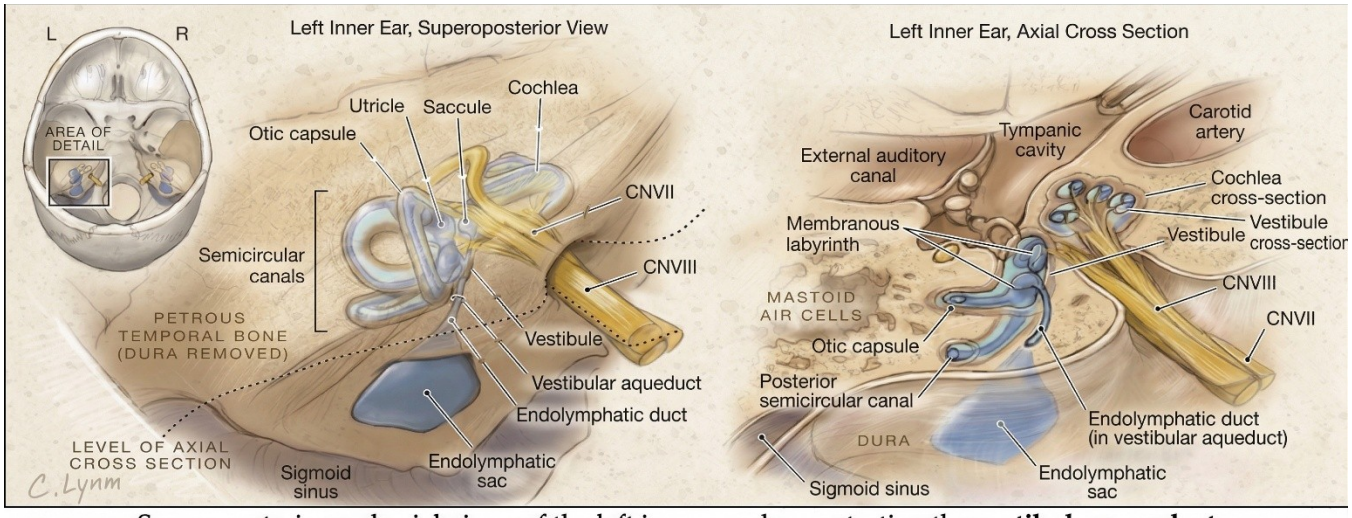
Quadrant	Contents	Clinical Note
Superior Posterior	Superior vestibular nerve (SVN)	Innervates utricle, anterior + lateral canal ampullae; affected in superior vestibular neuritis
Inferior Anterior	Cochlear nerve	Loss = sensorineural deafness; spared in selective SVN lesions
Inferior Posterior	Inferior vestibular nerve (IVN)	Innervates saccule + posterior canal ampulla; affected in inferior vestibular

2.2.4 The Cochlear Aqueduct and Vestibular Aqueduct

- **Cochlear aqueduct:** A small bony canal connecting the scala tympani (perilymph) to the subarachnoid space, near the round window. Allows bidirectional pressure equalisation. Clinically relevant in meningogenic labyrinthitis, where infection tracks inward from the subarachnoid space, and in sudden sensorineural hearing loss associated with raised or lowered CSF pressure.
- **Vestibular aqueduct:** A bony canal running from the medial wall of the vestibule, through the petrous bone, to the posterior surface of the petrous pyramid. Contains the endolymphatic duct. On CT, a wide vestibular aqueduct (>1.5 mm at midpoint) is associated with enlarged endolymphatic duct/sac — a radiological correlate of endolymphatic hydrops and a finding in Pendred syndrome (SLC26A4 mutations).



Axial high-resolution CT of the temporal bone demonstrating the cochlea, round window niche, and cochlear aqueduct (arrow), which arises from the scala tympani near the round window and extends medially toward the posterior cranial fossa. Adapted from Radiopaedia.



Superoposterior and axial views of the left inner ear demonstrating the **vestibular aqueduct,** endolymphatic duct and sac, and their relationship to the otic capsule and posterior petrous surface.
Adapted from educational anatomical illustration (C. Lynn)

3. Fluid Compartments: Perilymph and Endolymph

3.1 Perilymph

Perilymph fills the space between the bony labyrinth and the membranous labyrinth. Its ionic composition is similar to extracellular fluid: high Na+ (~140 mM), low K+ (~5 mM) [5]. It is thought to be derived partly from plasma ultrafiltrate via the vascular labyrinth and partly from CSF via the cochlear aqueduct, though the relative contributions remain debated.

Perilymph bathes the basolateral surfaces of the hair cells and the peripheral processes of CN VIII fibres.

3.2 Endolymph — Composition and Production

Endolymph is a unique extracellular fluid with an ionic composition resembling **intracellular fluid: high K+ (~150 mM), low Na+ (~1–15 mM), very low Ca2+ (~20 µM)** [5]. This composition is not maintained passively — it requires continuous active ion transport and represents a substantial metabolic investment, predominantly by the stria vascularis in the cochlea and the vestibular dark cells in the labyrinth. This energetic dependence explains why even brief ischemia abolishes mechano-electrical transduction almost immediately.

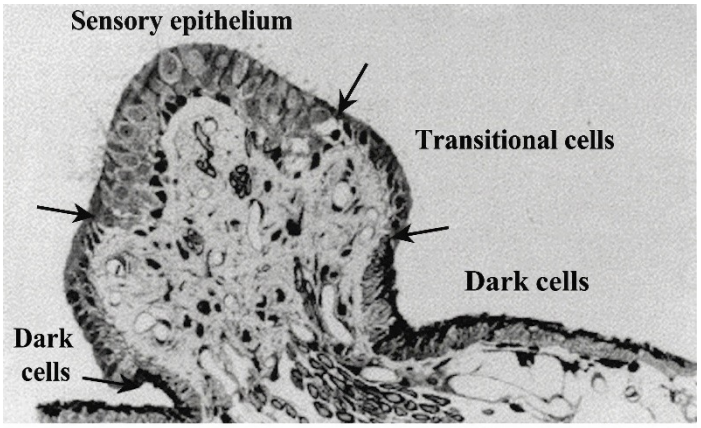
The high K+ concentration, combined with a positive endolymphatic potential (+80–100 mV in the cochlea; somewhat lower in the vestibular labyrinth), establishes a steep electrochemical gradient at the hair cell apex. When MET channels open, K+ flows down this combined concentration and electrical gradient from endolymph into the hair cell, depolarising the cell.

Companion reference: Physiology document, Section 3 (Molecular Physiology) — electrochemical basis of MET, K+ cycling.

3.3 Endolymph Homeostasis

Endolymph production and reabsorption are spatially segregated:

- **Production — Vestibular dark cells:** Located in the transitional zone at the base of the cristae and in the utricular wall. Packed with mitochondria and Na+/K+-ATPase, dark cells actively secrete K+ into the endolymph and reabsorb K+ from perilymph after it has exited hair cells, completing the K+ recycling loop. The melanin pigment in dark cells may serve as a free-radical scavenger or drug sink, including sequestration of aminoglycosides — relevant to their selective vestibulotoxicity.



Histological section of the vestibular epithelium demonstrating sensory epithelium, transitional cells, and vestibular dark cells located at the base of the crista. Dark cells form a specialised ion-transport epithelium responsible for potassium secretion into endolymph.
Adapted from classic vestibular histology studies, particularly the seminal work by Robert S. Kimura titled "Distribution, structure, and function of dark cells in the vestibular labyrinth"

- **Production — Stria vascularis (cochlea):** The cochlear equivalent of vestibular dark cells, and the primary generator of the endocochlear potential. Comprises three cell layers (marginal, intermediate, and basal). Disruption of any layer impairs K+ secretion and collapses the endocochlear potential, resulting in profound sensorineural deafness.
- **Reabsorption — Endolymphatic sac:** The terminal portion of the endolymphatic duct system, partially intraosseous and partially extraosseous on the posterior surface of the petrous bone. Lined by epithelial cells with absorptive function, characterised by abundant lysosomes and phagocytic capacity. Beyond reabsorption, the endolymphatic sac is the only site of immune

surveillance within the inner ear, housing T-lymphocytes and macrophages — a function that may be relevant to the inflammatory hypothesis of endolymphatic hydrops.

3.4 The Endolymphatic Duct and Sac

The endolymphatic duct arises from the utriculosaccular duct (at the junction of the utricle and saccule), passes through the bony vestibular aqueduct, and terminates in the endolymphatic sac — a blind-ended, partially intraosseous, partially extraosseous structure on the posterior surface of the petrous bone, within the posterior fossa dura [6].

The endolymphatic sac measures approximately 5–9 mm in length. Its proximity to the posterior fossa dura allows surgical access via endolymphatic sac decompression or shunting, though the evidence base for these procedures in Menière's disease remains contested [7].

♥ CLINICAL PEARL: Menière's Disease — Endolymphatic Hydrops Anatomy

Pathological basis: excess endolymph volume distends the membranous labyrinth (endolymphatic hydrops). The most accepted hypothesis implicates endolymphatic sac dysfunction — reduced reabsorptive capacity leading to endolymph accumulation.

Anatomical consequences: the distended cochlear duct causes Reissner's membrane to bulge into the scala vestibuli; the saccule balloons toward the stapes footplate — a position that renders it vulnerable to mechanical trauma and is the proposed anatomical basis of pressure-induced vertigo and the Tullio phenomenon in hydrops; the utricle and canals distend in advanced disease.

Rupture hypothesis: acute attacks may result from rupture of the membranes separating endo- and perilymph, causing K+ flooding of the perilymph space, depolarisation block of CN VIII afferents, and sudden vertigo with hearing drop. Healing by re-sealing of the ruptured membrane explains attack resolution.

Imaging: High-resolution 3T MRI with delayed gadolinium enhancement (4 hours post-IV gadolinium) can directly visualise hydrops as non-enhancing spaces within the gadolinium-enhanced perilymph. This is currently considered the gold standard for hydrops confirmation in vivo.

Betahistine mechanism: betahistine acts primarily as an H3 receptor antagonist, increasing endogenous histamine release. This produces vasodilation of the stria vascularis and vestibular dark cells, improving endolymph reabsorption and reducing production. Modulation of histaminergic tone in the vestibular nuclei may provide an additional mechanism of symptom relief.

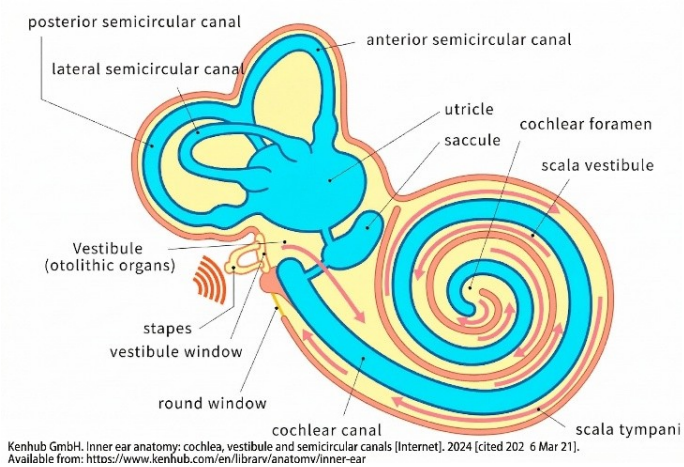
KEY FACTS: Fluid Compartments

Feature	Perilymph	Endolymph
Location	Bony labyrinth (extracellular space)	Membranous labyrinth (closed compartment)
Na^+ concentration	~140 mM (extracellular-like)	~1–15 mM (intracellular-like)

Feature	Perilymph	Endolymph
K^+ concentration	~5 mM	~150 mM
Electrical potential	~0 mV	+80–100 mV (cochlea); +5 mV (vestibule)
Produced by	Plasma ultrafiltrate / CSF via cochlear aqueduct	Dark cells (vestibule); stria vascularis (cochlea)
Reabsorbed by	Fibrocytes of spiral ligament	Endolymphatic sac epithelium
Clinical relevance	Meningogenic labyrinthitis; sudden loss with CSF pressure change	Endolymphatic hydrops (Meniere's disease); aminoglycoside toxicity

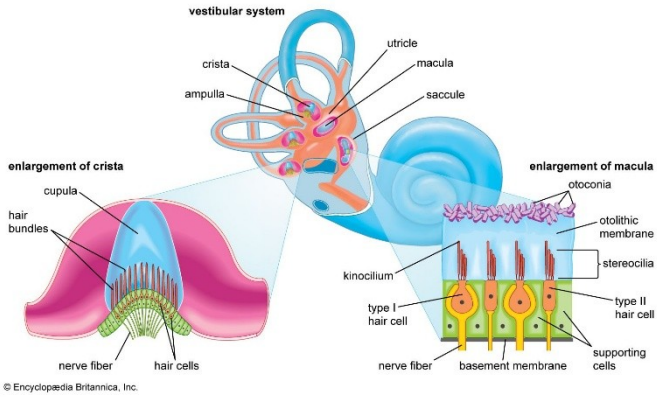
4. The Membranous Labyrinth

The membranous labyrinth is a closed system of endolymph-filled ducts and sacs suspended within the perilymph of the bony labyrinth. It comprises five sensory organs (three cristae ampullares and two maculae), two non-sensory connecting structures (the ductus reuniens and the utriculosaccular duct), and the endolymphatic duct and sac.



Kenhub GmbH. Inner ear anatomy: cochlea, vestibule and semicircular canals [Internet]. 2024 [cited 202 6 Mar 21]. Available from: https://www.kenhub.com/en/library/anatomy/inner-ear

4.1 The Semicircular Ducts



The three membranous ducts — anterior, posterior, and lateral — correspond to the three bony canals but occupy only approximately 1/4 to 1/3 of the bony canal lumen, leaving a large perilymphatic space. Each duct is ~0.3–0.4 mm in internal diameter. The duct wall comprises a thin connective tissue layer (tunica propria), an outer epithelial layer facing the perilymph, and a single-layered epithelium facing the endolymph.

Each duct has an ampullated end, where the lumen expands to ~1.5–2.0 mm to accommodate the crista ampullaris. The non-ampullated ends of the anterior and posterior ducts converge to form the common crus (crus commune) before entering the posterior vestibule.

4.2 The Utricle

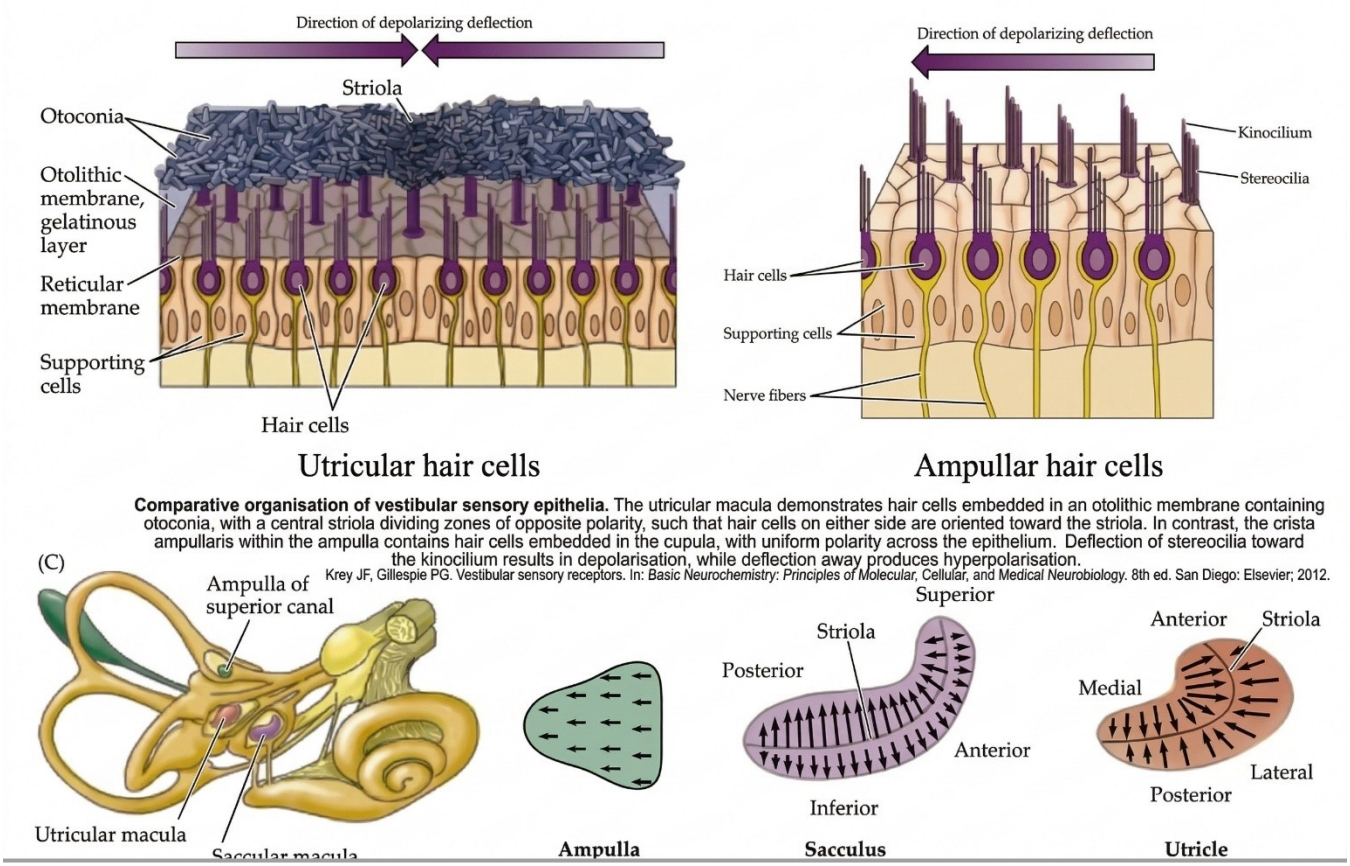
The utricle is an elliptical membranous sac, approximately 3 × 2.5 mm, located in the elliptical recess of the posterior superior vestibule [8]. In the upright head position, the utricular macula lies in a roughly horizontal plane (tilted ~15–30° anteriorly and medially from true horizontal), making it optimally oriented to detect horizontal linear acceleration and static tilt from the vertical.

- **Connections:** Receives the five canal openings (three ampullated + common crus + lateral canal non-ampullated). Drains via the utriculosaccular duct into the endolymphatic duct.
- **Sensory epithelium:** The utricular macula (~3 × 2.5 mm, ~35,000 hair cells) occupies the floor of the utricle. The striola — a curved zone of polarisation reversal — runs approximately in the midline; hair cells on either side are polarised towards the striola.
- **VEMP correlation:** The utricle is the primary end-organ generating the ocular VEMP (oVEMP) in response to sound or vibration, via the superior vestibular nerve. The oVEMP response is recorded from the contralateral eye, reflecting the crossed otolith-ocular pathway. oVEMP tests utricular and superior vestibular nerve pathway integrity.

4.3 The Saccule

The saccule is a smaller, roughly spherical membranous sac (~3 × 1.5 mm) located in the spherical recess of the anterior inferior vestibule [8]. In the upright head, the saccular macula is oriented approximately in the sagittal vertical plane, making it optimally sensitive to vertical linear acceleration (e.g., elevator motion, footfall during gait) and bone-conducted vibration.

- **Connections:** Communicates with the utricle via the utriculosaccular duct; with the cochlear duct via the ductus reuniens (Hensen's duct); with the endolymphatic sac via the endolymphatic duct.
- **Ductus reuniens:** A narrow connecting channel between the saccule and the scala media (cochlear duct). Allows slow equilibration of endolymph pressure between the vestibular and cochlear compartments.
- **Sensory epithelium:** The saccular macula (~3 × 1.5 mm, ~40,000 hair cells). Hair cells are polarised away from the striola. The striola runs approximately 35° from vertical, giving sensitivity to both vertical and some horizontal linear vectors in the sagittal plane.
- **VEMP correlation:** The saccule is the primary end-organ generating the cervical VEMP (cVEMP) in response to high-intensity air-conducted sound or bone-conducted vibration. Activation travels via the inferior vestibular nerve to the lower brainstem, where it drives ipsilateral sternocleidomastoid inhibition via the accessory nucleus. cVEMP tests saccular and inferior vestibular nerve pathway integrity.



Comparative organisation of vestibular sensory epithelia. The utricular macula demonstrates hair cells embedded in an otolithic membrane containing otoconia, with a central striola dividing zones of opposite polarity, such that hair cells on either side are oriented toward the striola. In contrast, the crista ampullaris within the ampulla contains hair cells embedded in the cupula, with uniform polarity across the epithelium. Deflection of stereocilia toward the kinocilium results in depolarisation, while deflection away produces hyperpolarisation.
Krey JF, Gillespie PG. Vestibular sensory receptors. In: *Basic Neurochemistry: Principles of Molecular, Cellular, and Medical Neurobiology*. 8th ed. San Diego: Elsevier; 2012.

♥ CLINICAL PEARL: Tullio Phenomenon and Saccular Anatomy

Tullio phenomenon refers to sound-induced vertigo or oscillopsia — pathological activation of the saccule (and sometimes utricle) by acoustic energy that does not normally reach the vestibular labyrinth.

Under normal anatomy, the oval window transmits sound vibration primarily to the fluid of the scala vestibuli, and the vestibular labyrinth is effectively shielded. In Superior Canal Dehiscence (SCD), an absence of bone overlying the anterior semicircular canal creates a third window — sound energy is shunted into the vestibular labyrinth, directly activating the saccule and anterior canal ampulla.

Tullio phenomenon is strongly suggestive of a third-window lesion, of which SCD is the most common cause, though it has also been reported in perilymph fistula and other dehiscence variants.

VEMP findings in SCD reflect the augmented sound transmission produced by the third window: cVEMP thresholds are paradoxically reduced (a response is elicited at lower stimulus intensities than normal) and oVEMP amplitudes are increased. Diagnosis is confirmed with high-resolution CT of the temporal bones using sub-millimetre slices, assessed in both Pöschl and Stenvers projections to visualise the anterior canal roof in orthogonal planes.

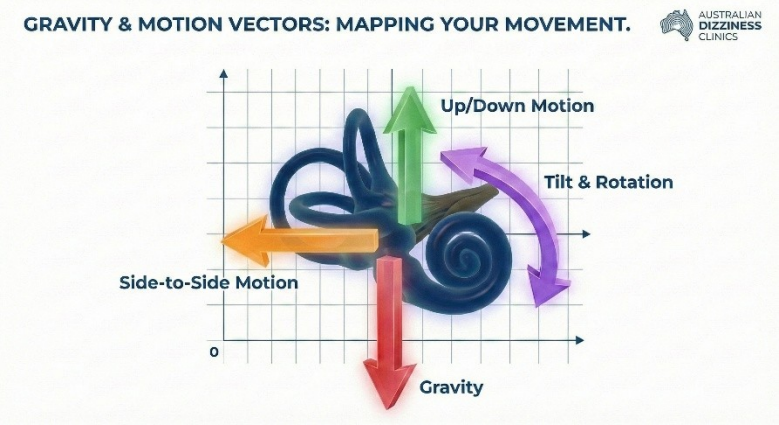
5. Semicircular Canal Geometry, Functional Pairing, and Ewald's Laws

5.1 Spatial Orientation

The three canals are oriented approximately 90° to one another in three planes, providing complete angular motion detection:

Canal	Plane	Orientation in Upright Head	Functional Pair
Anterior (superior)	Vertical	45° to sagittal, perpendicular to posterior canal of same ear	Right anterior + Left posterior (RALP)
Posterior	Vertical	45° to sagittal, perpendicular to anterior canal of same ear	Left anterior + Right posterior (LARP)
Lateral (horizontal)	Roughly horizontal	30° above true horizontal in erect posture	Right lateral + Left lateral

The push-pull organisation of coplanar pairs means that when one canal is excited (ampullopetal or ampullofugal flow, depending on canal polarity), its coplanar partner on the contralateral side is inhibited [9]. This bilateral symmetry doubles the dynamic range of the system and is the basis for spontaneous nystagmus after unilateral vestibular failure: the intact side generates unopposed tonic firing, driving nystagmus fast phase toward the healthy side.



Companion reference: Physiology document, Section 2.1 — push-pull bilateral organisation, spontaneous nystagmus physiology.

5.2 Ewald’s Laws — Anatomical Basis and Clinical Application

Ewald's Laws (1892) were derived from semicircular canal stimulation experiments in pigeons but remain the cornerstone of clinical semicircular canal physiology [10]. They translate canal anatomy into predictable nystagmus direction:

Law	Statement	Anatomical Basis	Clinical Application
1st	Eye movements (and nystagmus fast phase) are in the plane of the stimulated canal	Canal geometry — each canal is sensitive to rotation in its own plane; eye muscles are biomechanically aligned with canal planes	BPPV nystagmus plane identifies the culprit canal; HIT catch-up saccade direction identifies the hypofunction side
2nd	Horizontal canal: ampullopetal endolymph flow is EXCITATORY (stronger response) vs ampullofugal (inhibitory)	Lateral crista hair cells polarised toward utricle → kinocilium faces utricle → ampullopetal = excitatory	Explains caloric asymmetry: cold irrigation of right ear = ampullofugal inhibition; warm = ampullopetal excitation. COWS mnemonic.
3rd	Vertical canals (anterior + posterior): ampullofugal is EXCITATORY	Vertical crista hair cells polarised away from utricle → ampullofugal = excitatory	Explains why posterior canal BPPV — where free-floating canalith debris moves ampullofugally, deflecting the cupula — produces upbeat-torsional nystagmus

5.3 Caloric Testing — Anatomical and Physical Basis

Caloric irrigation stimulates exclusively the lateral semicircular canal because of its near-horizontal orientation and superficial position relative to the ear canal. Cold or warm water creates a temperature gradient across the horizontal duct, altering endolymph density and generating a convection current [11]:

- **Warm irrigation (44°C):** Endolymph near the lateral canal wall is warmed, becomes less dense, and rises in the upright head. This generates ampullopetal flow, exciting the ipsilateral lateral canal and driving the nystagmus fast phase towards the irrigated ear.
- **Cold irrigation (30°C):** Endolymph cools, increases in density, and sinks. This generates ampullofugal flow, inhibiting the ipsilateral lateral canal and driving the nystagmus fast phase away from the irrigated ear.
- **Mnemonic — COWS:** Cold Opposite, Warm Same, referring to the direction of the nystagmus fast phase relative to the irrigated ear.
- **Significance of Ewald's 2nd Law:** Because ampullopetal excitation produces a stronger response than ampullofugal inhibition, warm irrigation generates a larger nystagmus than cold irrigation of the same ear. The Jongkees canal paresis formula accounts for this: $CP = [(RW+RC) - (LW+LC)]/(RW+RC+LW+LC) \times 100\%$; asymmetry exceeding 25% indicates unilateral canal paresis.

♥ CLINICAL PEARL: Caloric Testing — When the Canal Cannot Convect

Caloric testing relies on convection currents and therefore requires the lateral canal to be approximately horizontal, achieved by positioning the patient supine with the head elevated 30°. This positioning requirement limits its use in certain contexts: in horizontal canal BPPV,

repositioning manoeuvres should precede caloric testing to avoid confounding results; during MRI, caloric testing is not feasible and rotational or vHIT-based assessment is required instead.

Ice water caloric (Fitzgerald-Hallpike): 20 mL of ice water is instilled to test for any residual labyrinthine function. Absence of nystagmus indicates absent labyrinthine function on that side and is useful in confirming labyrinthine infarction or a dead labyrinth.

Rotational chair testing (sinusoidal harmonic acceleration, 0.01–1.0 Hz): Unlike calorics, rotational chair testing stimulates both labyrinths simultaneously and is unaffected by middle ear disease. It probes mid-to-low frequency canal function, complementary to vHIT which probes the high-frequency end of the same reflex arc.

Unilateral absence of caloric response with normal vHIT: this apparently paradoxical finding reflects the frequency-specific nature of the two tests. Calorics probe extremely low-frequency canal function (~0.003 Hz), while vHIT probes high-frequency responses (5–15 Hz head impulses). A canal that fails at low frequencies but remains functional at high frequencies produces precisely this pattern and should prompt consideration of incomplete vestibular neuritis or early Menière's disease rather than a complete labyrinthine lesion.

6. Sensory End-Organs of the Vestibular Labyrinth

6.1 The Crista Ampullaris — Structure and Mechanics

The crista ampullaris is a saddle-shaped ridge of neuroepithelium, approximately 2–3 mm in height, positioned transversely across each duct ampulla [12]. It is the sensory organ for angular acceleration. Each crista contains approximately 7,000 hair cells in humans.

6.1.1 The Cupula

The cupula is a gelatinous, glycoprotein-rich structure (principally OTOG — otogelin — and other proteoglycans) that extends from the surface of the crista to the roof of the ampulla, forming a fluid-tight diaphragm [13]. Its precise specific gravity (~1.001–1.004) is essentially equal to that of endolymph (~1.003). This neutral buoyancy is physiologically critical:

- **Consequence of neutral buoyancy:** The cupula responds to angular acceleration via endolymph inertia, NOT to gravitational force. A tilt of the head does not deflect the cupula (unlike the otolith organs). This is why semicircular canal-mediated nystagmus is not gravity-dependent under normal circumstances.
- **Exception — Cupulolithiasis:** When otoconia adhere to the cupula, as in one form of BPPV, the cupula acquires a specific gravity heavier than endolymph, rendering it gravity-sensitive. The result is persistent positional nystagmus characterised by no latency, no fatigue, and no direction reversal with repeated positioning — distinguishing it from canalithiasis BPPV, where nystagmus is transient and fatigable.
- **Dimensions:** The cupula exactly fills the ampullary lumen, forming a hermetic seal. This prevents endolymph from flowing past the crista — all fluid displacement must deflect the cupula.

Companion reference: Physiology document, Section 2.1 — torsion pendulum model; time constants Tc (~6s) and Tp (~16–20s); velocity storage extension.

6.1.2 Hair Cell Polarisation on the Crista

The crista is not structurally uniform. It has a central zone (summit) and peripheral zone (slopes), with distinct hair cell populations:

- **Central zone:** Predominantly Type I hair cells with calyx afferents. These afferents are irregular in their discharge pattern (high coefficient of variation of interspike intervals) and have high gain, encoding high-frequency, transient angular motion — the stimulus range probed by the vHIT.
- **Peripheral zone (slopes):** Predominantly Type II hair cells with bouton afferents. These afferents are regular in discharge (low coefficient of variation) and lower in gain, encoding sustained, lower-frequency rotations — the stimulus range probed by rotational chair and caloric testing.

Beyond this zonal organisation, all hair cells across a given crista share a uniform polarisation axis: for **the horizontal crista, the kinocilium faces toward the utricle; for the vertical cristae (anterior and posterior), the kinocilium faces away from the utricle**. This directional uniformity means the entire crista responds as a unit to deflection in one direction, and this asymmetry between horizontal and vertical crista polarisation is the anatomical basis of Ewald's Second and Third Laws (see Section 5.2) [14].

6.2 The Maculae — Structure, Polarisation, and Striola

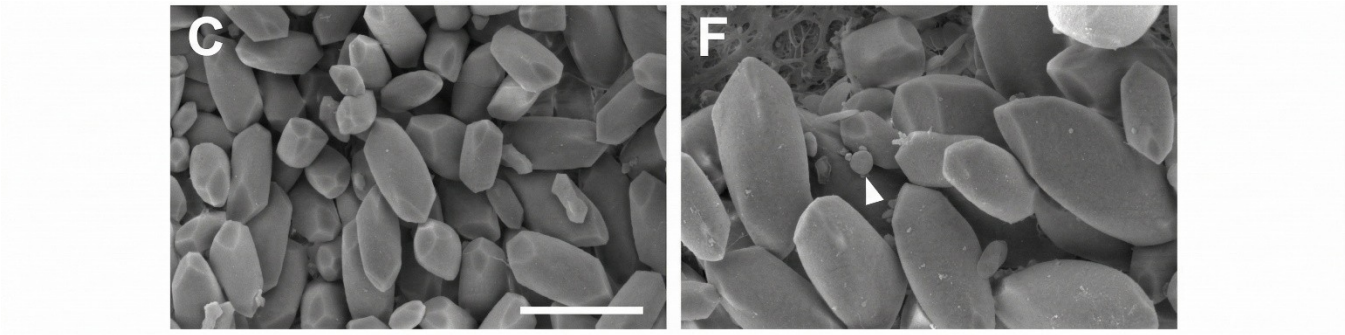
The maculae of the utricle and saccule transduce linear acceleration and static head tilt. Each macula is a flat sheet of neuroepithelium covered by the otolithic membrane — a gelatinous layer supporting the otoconia.

6.2.1 The Otolithic Membrane

The otolithic membrane has two mechanically distinct layers:

- **Gel layer:** A gelatinous matrix directly overlying the hair cell stereocilia, formed primarily of otogelin, otogelin-like proteins, and associated glycoproteins.
- **Otoconial layer:** The gelatinous layer is studded with otoconia — calcium carbonate crystals in the calcite polymorph, shaped as hexagonal prisms, 3–30 µm in diameter. Their specific gravity (~2.71) greatly exceeds that of endolymph (~1.003). This density contrast provides the inertial mass that allows the otoconial layer to lag behind the macula during acceleration, shearing the stereocilia.

Otoconial biology: Otoconia are not inert stones but biocrystals containing Otoconin-90 (Oc90), a glycoprotein that regulates crystal nucleation, and Otopetrin-1 (OTOP1), essential for initial seeding [15]. Turnover is slow, occurring over weeks, and decreases further with age. Age-related degeneration of the protein matrix leads to otoconial fragmentation, detachment from the macula, and migration into the semicircular canals — the pathophysiological basis of BPPV.



Scanning electron micrographs of utricular otic otoconia demonstrating their characteristic elongated, prismatic morphology with smooth calcite variability in size and clusterid clustering reflects normal structural organisation of the otoconial layer. Otoconia are calcium carbonate (calcite) bioc- that provide inertial mass to the otolithic membrane, enabling detection of linear acceleration and head tilt.

- Adapted from-

Boyle R, Varelas J. Otoconia structure after short- and long-duration exposure to altered gravity. *J Assoc Res Otolaryngol*. 2021;22(3):281–299.

6.2.2 The Striola — The Functional Watershed

The striola is a specialised zone of neuroepithelium approximately 80–100 µm wide, curving across the centre of each macula [16]. It represents the boundary between two regions of opposing hair cell polarity:

- **Utricular macula:** Hair cells on both sides of the striola are polarised towards it, with the kinocilium facing the striola. Any lateral tilt therefore excites hair cells on the downside while inhibiting those on the upside, encoding both the magnitude and direction of tilt simultaneously.
- **Saccular macula:** Hair cells are polarised away from the striola. The striola runs approximately 35° from vertical, providing sensitivity to both vertical and some horizontal linear vectors in the sagittal plane.

The striola region has distinct anatomical properties compared to the extrastriola: Type I hair cells predominate; afferents are calyx-only or dimorphic; otoconia are more widely spaced; and the gel layer is thinner. These properties make the striola a phasic, high-dynamic detector of rapid linear transients, while the extrastriola provides tonic DC encoding of sustained tilt [16].

♥ CLINICAL PEARL: Otolith Organ Testing — VEMP Anatomy

Cervical VEMP (cVEMP): High-intensity air-conducted sound (95–100 dB nHL) or bone-conducted vibration activates the saccule, which projects via the inferior vestibular nerve to the lower brainstem. The pathway crosses within the brainstem to reach the ipsilateral accessory nucleus, driving inhibition of the ipsilateral sternocleidomastoid — which is why the cVEMP is recorded ipsilaterally despite the brainstem crossing. cVEMP tests saccular and inferior vestibular nerve integrity.

Ocular VEMP (oVEMP): Sound or vibration activates the utricle, which projects via the superior vestibular nerve through CN VIII to the brainstem, where a crossed pathway activates the contralateral inferior oblique motor neurons. The response (N10) is therefore recorded beneath the contralateral eye. oVEMP tests utricular and superior vestibular nerve integrity.

Clinical dissociation: In inferior vestibular neuritis, horizontal vHIT and oVEMP are normal but the ipsilateral cVEMP is absent or reduced. In superior vestibular neuritis, horizontal vHIT and oVEMP are abnormal but cVEMP is intact. This dissociation pattern is highly characteristic of divisional vestibular nerve involvement and allows precise localisation of the affected nerve division without imaging.

Superior Canal Dehiscence: cVEMP thresholds are reduced (below 80 dB nHL) and amplitudes increased, reflecting the third window effect that augments sound transmission to the saccule.

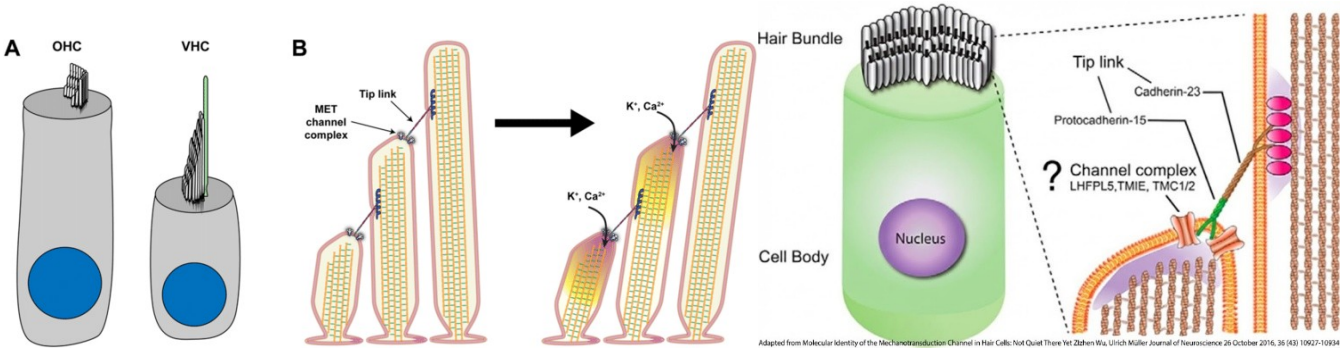
7. Vestibular Hair Cells — Ultrastructure and Transduction

7.1 The Hair Bundle

Each vestibular hair cell bears a hair bundle on its apical surface: a stereociliary staircase of 50–100 actin-filled stereocilia arrayed in graded heights, with a single kinocilium (a true cilium with a 9+2 microtubule axoneme) at the tallest pole [17]. This structural polarity determines the cell's directional sensitivity.

The stereocilia are linked by multiple extracellular filament types:

- **Tip links:** Fine oblique filaments connecting the tip of a shorter stereocilium to the side of the taller neighbour. Composed of Protocadherin-15 (PCDH15) at the lower end and Cadherin-23 (CDH23) at the upper end. These links directly gate the MET channels. Mutations in PCDH15 or CDH23 cause Usher syndrome type I (congenital deaf blindness with vestibular areflexia).
- **Ankle links:** Connect stereocilia near their base; important for bundle cohesion and maintaining staircase geometry.
- **Horizontal top connectors:** Connect stereocilia of similar height; provide lateral stability.



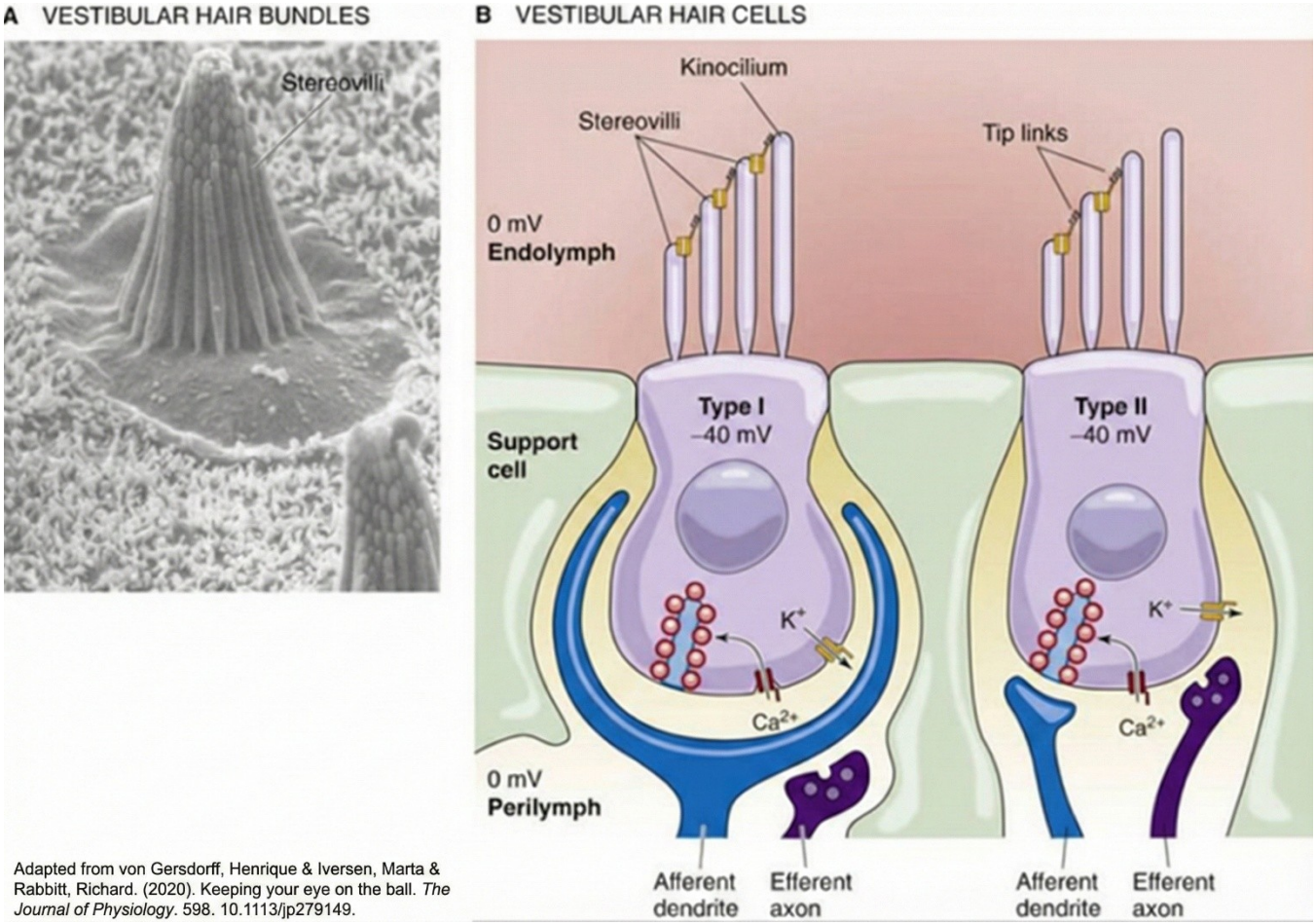
Companion reference: Physiology document, Section 3 — tip link mechanics, MET channel identity (TMC1/TMC2), adaptation mechanisms, K+ cycling.

7.2 Type I versus Type II Hair Cells

All amniote (reptile, bird, mammal) vestibular neuroepithelia contain two morphologically and functionally distinct hair cell types:

Feature	Type I Hair Cell	Type II Hair Cell
Body shape	Flask-shaped; narrow neck	Cylindrical; uniform width
Afferent innervation	Enclosed by a single giant calyx terminal	Multiple small bouton endings
Location	Central crista; striola of maculae	Peripheral crista; extrastriola of maculae
Basolateral K+ conductance	IK,L (low voltage activated): very low input resistance; fast time constant	IK (delayed rectifier) + IKCa: higher input resistance; slower time constant

Feature	Type I Hair Cell	Type II Hair Cell
Afferent firing pattern	Irregular (high coefficient of variation)	Regular (low CV)
Dynamic range	High gain; phasic — encodes rapid transients	Lower gain: tonic — encodes sustained stimuli
Clinical test best probed	vHIT (high-frequency canal function)	Caloric, rotational chair (low-mid frequency)
Phylogeny	Unique to amniotes (evolved ~310 Ma)	Present in all vertebrates (ancestral type)



Adapted from von Gersdorff, Henrique & Iversen, Marta & Rabbitt, Richard. (2020). Keeping your eye on the ball. *The Journal of Physiology*. 598. 10.1113/jp279149.

7.3 The Calyx Synapse — Quantal and Non-Quantal Transmission

The Type I hair cell is almost entirely enclosed by the calyx terminal, creating a highly restricted synaptic cleft. This specialised geometry enables two parallel transmission modes [18]:

- **Quantal (vesicular) transmission:** Ca2+-triggered glutamate release from ribbon synapses (approximately 5–10 ribbons per Type I cell, fewer than cochlear inner hair cells). Relatively slow (0.5–1.0 ms synaptic delay). The primary mode of Type II cell transmission.
- **Non-quantal transmission (NQT):** K+ efflux from the hair cell accumulates within the restricted calyx cleft. This rise in extracellular [K+] directly depolarises the calyx terminal without vesicle

fusion, eliminating synaptic delay. NQT operates in the microsecond range — enabling transmission of high-frequency (>50 Hz) head motion signals that vesicular transmission alone cannot follow.

NQT has direct functional importance: in otoferlin-knockout mice, which lack vesicular release entirely, vestibular function is partially preserved precisely because NQT remains intact, while auditory function is abolished. Cochlear inner hair cells rely exclusively on quantal transmission because they lack the enclosed calyx geometry that enables K+ accumulation — the structural prerequisite for NQT [18].

Companion reference: Physiology document, Section 3.4 (Calyx Paradox) — full mechanistic derivation of NQT.

7.4 The Ribbon Synapse

Both Type I and Type II hair cells transmit via ribbon synapses — specialised presynaptic structures consisting of a dense body ("ribbon") anchored in the cytoplasm, which tethers a halo of glutamate-filled vesicles close to the presynaptic membrane [19]. Cav1.3 L-type voltage-gated Ca2+ channels cluster around the ribbon. The ribbon acts as a vesicle conveyor belt, ensuring rapid, sustained glutamate release without fatigue — essential for maintaining tonic CN VIII discharge of more than 50 spikes per second at rest.

8. The Efferent Vestibular System

8.1 Anatomy

The efferent vestibular system (EVS) consists of a group of neurons in the brainstem whose axons project **centrifugally** — from the brainstem to the vestibular periphery. The EVS provides the only direct central mechanism for modulating peripheral vestibular gain at the hair cell level [20].

- **Origin:** Two bilateral nuclei adjacent to CN VI (abducens nucleus) in the pons: the **medial EVN** and the **lateral EVN** (the latter near the superior olive). Approximately 300–400 neurons per side in humans.
- **Course:** Efferent fibres travel with CN VIII, cross within the brainstem, and reach the labyrinth via the superior and inferior vestibular nerves.
- **Targets:** Efferent fibres synapse on three target populations: Type II hair cells via bouton endings directly on the cell body; the outer surface of the Type I calyx terminal; and afferent bouton endings adjacent to Type II cells.

8.2 Neurotransmitters and Postsynaptic Mechanisms

- **Primary neurotransmitter:** Acetylcholine (ACh), acting on α9/α10 nicotinic receptors on Type II hair cells. In hair cells, nicotinic receptor activation paradoxically causes hyperpolarisation: nicotinic receptors are Ca2+-permeable → Ca2+ entry activates adjacent SK (small-conductance K+) channels → K+ efflux → membrane hyperpolarisation → reduced MET current → attenuated receptor potential.
- **Co-transmitters:** CGRP (calcitonin gene-related peptide), GABA, enkephalin — modulatory functions not fully resolved.

8.3 Functional Role

The EVS provides gain control and context-dependent modulation of vestibular afferent output [21]:

- **Efference copy / active motion suppression:** During self-generated head movements, motor commands generate an efference copy that activates the EVS slightly before movement onset, pre-suppressing the vestibular response to expected reafference. This appropriately reduces the VOR gain during voluntary head turns — gaze compensation for a self-initiated movement would be counterproductive to the intended motor act.
- **Protective function:** During high-intensity sustained stimulation, EVS activation limits hair cell receptor potential amplitude, reducing the risk of excitotoxic damage to afferents.
- **Clinical correlate:** VOR gain to active head movement is slightly lower than to passive head movement of the same velocity. If a patient shows a normal VOR to passive head impulse but symptoms only during self-generated motion, EVS dysfunction or central efference copy failure should be considered.

Companion reference: Physiology document, Section 3.6 — efferent vestibular system, ACh/SK channel mechanism, locomotion-correlated efferent activation.

9. Vascular Anatomy of the Inner Ear

9.1 Overview — The End-Artery Problem

The entire membranous labyrinth is supplied by a single terminal vascular system with essentially no collateral circulation [22]. This makes the inner ear **uniquely vulnerable to ischemia:** occlusion of even a small labyrinthine vessel causes immediate, irreversible infarction. Unlike the brain, where collateral circulation may allow penumbral salvage, there is no collateral rescue in inner ear ischemia.

9.2 The Labyrinthine Artery (Internal Auditory Artery)

The labyrinthine artery is the sole arterial supply of the membranous labyrinth. Origin:

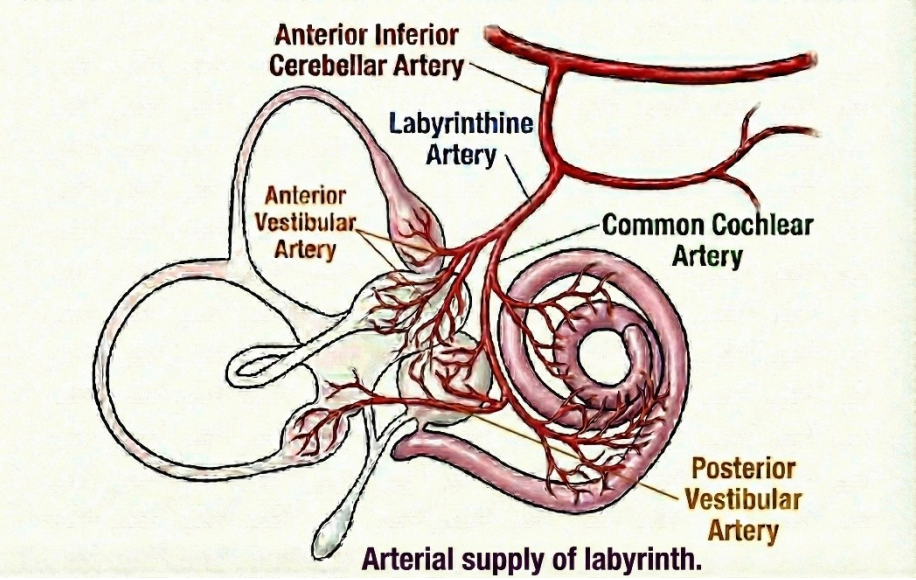
- **Most common (~85%):** Branch of the Anterior Inferior Cerebellar Artery (AICA).
- **Less common (~15%):** Arises directly from the basilar artery.

The artery enters the IAC at the porus acusticus and travels with CN VII and CN VIII to the fundus. In approximately 10–15% of individuals, a loop of the AICA enters the IAC, sometimes compressing CN VIII against the osseous wall — a **neurovascular compression** variant sometimes termed vascular loop syndrome. This anatomical variant is the proposed pathophysiological basis of **vestibular paroxysmia:** brief, stereotyped attacks of vertigo that respond to sodium channel blockers such as carbamazepine or oxcarbazepine [23].

9.3 Branching Pattern and End-Organ Territories

At the fundus of the IAC, the labyrinthine artery divides into two main branches:

- **Anterior Vestibular Artery (AVA):** Supplies the vestibular nerve (superior division), utricle, and the ampullae of the anterior and lateral semicircular canals. Its territory exactly mirrors the superior division of Scarpa's ganglion. It is the smaller of the two terminal branches and has virtually no anastomotic potential.
- **Common Cochlear Artery (CCA):** The larger branch; supplies the cochlea via the spiral modiolar artery. Additionally gives off the **Posterior Vestibular Artery (PVA)** — which supplies the saccule, the posterior canal ampulla, and a small inferior portion of the utricle — matching the inferior vestibular nerve territory.



Arterial supply of labyrinth.

♥ CLINICAL PEARL: Ischaemic Vestibular Syndromes — Know Your Territories

AVA occlusion (isolated anterior vestibular infarction): Acute vertigo with pathological vHIT to the lesion side (horizontal and anterior canal), utricular dysfunction (contralateral ocular tilt, reduced oVEMP), and preserved hearing (cochlea supplied by CCA is intact). Posterior canal vHIT is normal and cVEMP is intact, as the saccule is supplied by the PVA. The presentation closely resembles superior vestibular neuritis but is distinguished by its abrupt onset without subsequent recovery and the absence of the temporal variation characteristic of inflammatory neuritis.

Labyrinthine artery occlusion (total labyrinthine infarction): Simultaneous sudden unilateral sensorineural hearing loss and complete ipsilateral vestibular loss, without brainstem signs. MRI within 24 hours is typically negative — even dedicated diffusion-weighted sequences of the labyrinth have limited sensitivity in the first 24–48 hours, as the volume of infarcted tissue is too small for standard resolution. A negative early MRI does not exclude labyrinthine infarction.

AICA infarction: Labyrinthine infarction combined with lateral pontine and cerebellar signs — ipsilateral facial paresis, ipsilateral facial numbness, ipsilateral Horner's syndrome, ipsilateral limb ataxia, and sensorineural hearing loss from cochlear infarction. The presence of hearing loss distinguishes AICA from PICA infarction, as the cochlear supply arises from AICA in both cases.

PICA infarction (Wallenberg syndrome): Lateral medullary syndrome comprising ipsilateral facial hemihypoalgesia, contralateral body hemihypoalgesia, Horner's syndrome, dysphagia, hoarseness, ataxia, nystagmus, and lateropulsion. Hearing is preserved, because the labyrinthine artery — and therefore the cochlear supply — arises from AICA, not PICA.

Key diagnostic principle: In any patient presenting with acute vertigo and sudden hearing loss, AICA stroke must be actively excluded. A normal MRI within 24 hours does not exclude infarction. HINTS examination (Head Impulse test, Nystagmus direction, Test of Skew) combined with urgent audiometry should be performed in all such presentations.

9.4 Venous Drainage

The venous drainage of the inner ear follows a different topography from the arterial supply [24]:

- **Vein of the Vestibular Aqueduct (VVA):** The dominant vestibular drainage vessel. Drains the semicircular canals and utricle. Traverses the bony paravestibular canaliculus adjacent to the vestibular aqueduct. Drains into the inferior petrosal sinus or the jugular bulb.
- **Vein of the Cochlear Aqueduct:** Drains the saccule and cochlea. Enters the posterior fossa via the cochlear aqueduct.
- **Labyrinthine vein:** A variable vessel that may drain portions of both compartments into the inferior petrosal sinus.

The VVA runs in close anatomical proximity to the endolymphatic sac. Pathological obstruction or fibrosis of the VVA has been postulated as a mechanism for backpressure on the endolymphatic system — a contributing factor in secondary endolymphatic hydrops [24]. High-resolution MRI is now capable of visualising the VVA in some cases.

KEY FACTS: Vascular Anatomy

Vessel	Origin	Territory	Occlusion Syndrome
Labyrinthine a.	AICA (~85%)	Entire membranous labyrinth	Sudden unilateral deafness + total vestibular loss; no brainstem signs
Anterior Vestibular a.	Labyrinthine a.	Vestibular nerve, utricle, ant. + lat. canal ampullae	Vertigo; horiz. + ant. canal HIT+; utricular loss; HEARING SPARED; saccule intact
Posterior Vestibular a.	Common Cochlear a.	Saccule, posterior canal ampulla	Vertigo; absent cVEMP; normal hearing
AICA	Basilar a.	Labyrinth + lateral pons/cerebellum	Labyrinthine infarct + hearing loss + facial palsy + ataxia + Horner's
PICA	Vertebral a.	Lateral medulla + post. inf. cerebellum	Wallenberg syndrome; HEARING SPARED
Basilar a.	Junction of vertebral aa.	Pons, midbrain, cerebellum, labyrinth (via AICA)	'Top of basilar' syndrome; bilateral involvement; locked-in state

10. Scarpa's Ganglion and the Vestibular Nerve

10.1 Scarpa's Ganglion — Anatomy and Topography

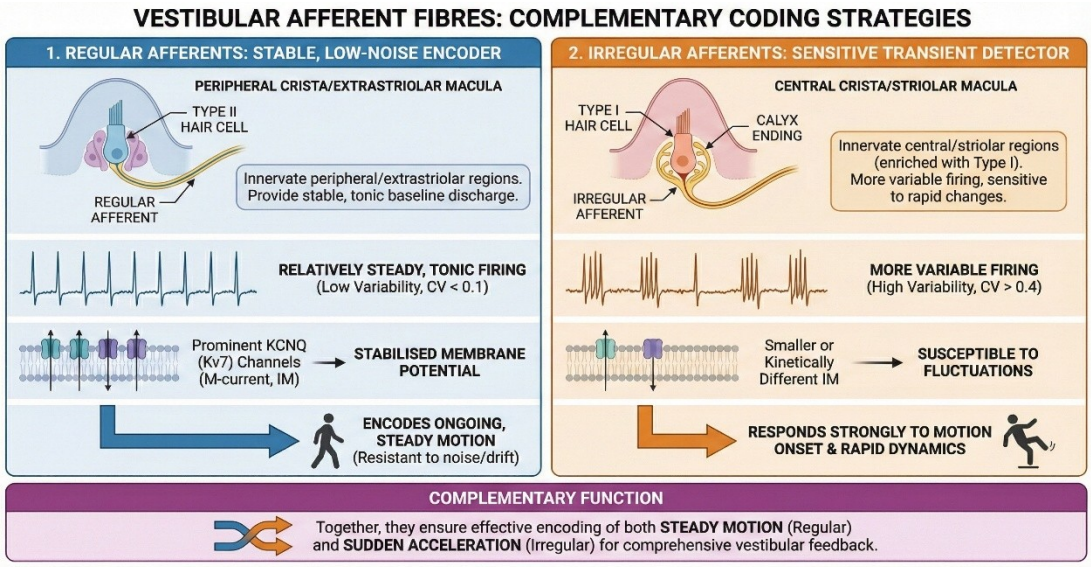
Scarpa's ganglion (vestibular ganglion) contains the cell bodies of the primary vestibular afferents. It is located within the IAC, at its midpoint, and is a bilobed structure with strict somatotopic organisation that mirrors the four-quadrant IAC anatomy [25]:

- **Superior division:** Innervates the anterior canal ampulla, lateral canal ampulla, and utricle (including most of the utricular macula). Axons form the superior vestibular nerve, running in the superior posterior quadrant of the IAC (Bill's bar between it and the facial nerve anteriorly).
- **Inferior division:** Innervates the posterior canal ampulla and saccule. Axons form the inferior vestibular nerve, running in the inferior posterior quadrant of the IAC.

The vestibular nerve fibres enter the brainstem at the pontomedullary junction, lateral to the facial nerve, and immediately bifurcate **ascending fibres** project to the superior and medial vestibular nuclei; **descending fibres** project to the medial, lateral, and descending vestibular nuclei. A small proportion project directly to the cerebellum (the **vestibulocerebellar fibres**) via the juxtarestiform body [26].

10.2 Afferent Fibre Classes

Primary vestibular afferents are classified by discharge regularity (coefficient of variation, CV, of interspike intervals) [27]:



Feature	Regular (Low CV)	Irregular (High CV)
Terminal type	Bouton (on Type II cells)	Calyx and dimorphic (on Type I cells)
Location on crista/macula	Peripheral zones	Central / striolar zones
Resting discharge	Regular, ~50–100 sp/s	Irregular, ~50–100 sp/s (high variance)
Gain	Lower gain, flat frequency response	High gain, especially at high frequencies
Phase	Near-in-phase with head velocity	Phase-leads head velocity
Best for encoding	Sustained tilt, slow drift	Rapid head turns, transients (vHIT)

Feature	Regular (Low CV)	Irregular (High CV)
Channel contribution	Tonic 'state' channel	Phasic 'event' channel

Companion reference: Physiology document, Section 4 — afferent encoding and frequency tuning; canal vs otolith afferent distinctions.

♥ CLINICAL PEARL: Superior vs. Inferior Vestibular Neuritis — Anatomical Basis

Superior vestibular neuritis (SVN, ~90% of vestibular neuritis cases): Preferential involvement of the superior division of CN VIII. The superior vestibular nerve is anatomically predisposed to injury: it passes through a narrower bony canal with less vascular supply than the inferior branch, and its longer intraosseous course may render it more susceptible to viral-mediated demyelination. Herpes simplex virus type 1 reactivation within the superior Scarpa's ganglion is the leading aetiological hypothesis.

Clinical signs: horizontal-torsional spontaneous nystagmus with the fast phase away from the lesion side; pathological horizontal vHIT with reduced VOR gain and catch-up saccades; pathological anterior canal vHIT; normal cVEMP (saccule and inferior vestibular nerve intact); normal posterior canal vHIT; and reduced or absent ipsilateral oVEMP.

Inferior vestibular neuritis (IVN, ~10% of cases): Affects the posterior canal ampulla and saccule via the inferior vestibular nerve. Normal horizontal vHIT; pathological posterior canal vHIT; absent ipsilateral cVEMP; normal oVEMP.

Treatment: Methylprednisolone (100 mg initially, tapering over three weeks) significantly improves long-term recovery of canal function when started within the first three days of symptom onset [Strupp et al., NEJM 2004], although new studies have now questioned this findings. Valaciclovir, evaluated in the same trial, added no benefit over corticosteroids alone, and antiviral therapy is not currently recommended. Vestibular rehabilitation accelerates central compensation and should be initiated early.

11. The Vestibular Nuclear Complex (VNC)

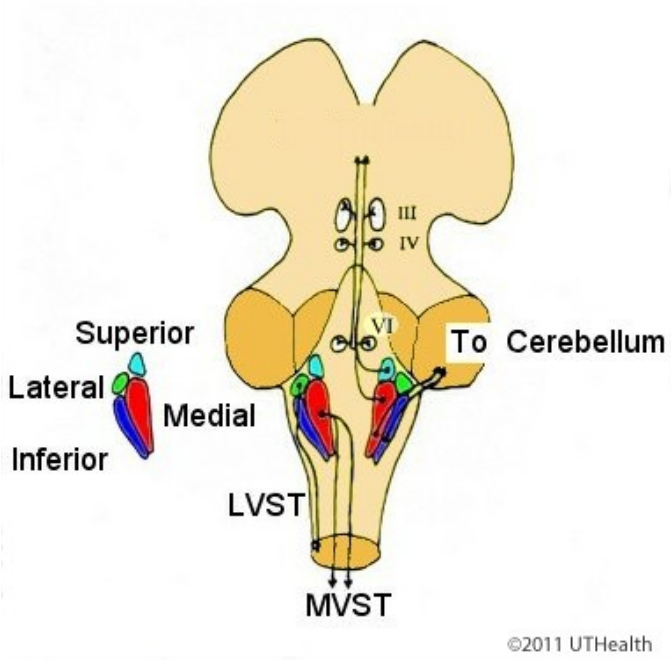
11.1 Overview

The vestibular nuclear complex (VNC) occupies the floor of the fourth ventricle from the lower pons to the upper medulla (area vestibularis). It is **not a relay station** — it is a sophisticated multimodal integration hub that combines vestibular, visual, proprioceptive, cerebellar, and motor efference copy signals to generate appropriate motor outputs [28]. Notably, only approximately 25% of VNC input arises from the vestibular nerve itself; the remainder arrives from the cerebellum, contralateral VNC, reticular formation, and spinal cord — reflecting the VNC's role as a convergence site for multisensory and motor signals rather than a dedicated vestibular processor.

11.2 The Four Major Nuclei

The VNC comprises four major nuclei and several accessory groups:

Nucleus	Eponym	Location	Inputs	Outputs	Key Function
Superior (SVN)	Bechterew's	Dorsolateral pons	anterior and horizontal cristae; flocculus	MLF → CN III/IV bilaterally	Vertical + torsional VOR
Medial (MVN)	Schwalbe's	Largest; medulla to pons	All cristae; commissural fibres; flocculus; spinal cord	MLF → CN III/IV/VI; MVST bilaterally; NTS	Horizontal VOR; VCR; vestibulo-autonomic
Lateral (LVN)	Deiters'	Lateral medulla/pons; giant Deiters' cells	Utricle; cerebellar vermis (direct)	LVST ipsilaterally to all spinal levels	Vestibulospinal reflex; antigravity tone
Descending (DVN)	Roller's (inferior vestibular nucleus)	Caudal medulla	Saccule + utricle; nodulus/uvula; spinal cord	Reticular formation; contralateral VNC; cerebellum	Otolith-cerebellar integration; verticality perception



11.3 Functional Neuron Classes Within the VNC

Beyond simple anatomical divisions, the VNC is better understood through functionally defined neuron classes [29]:

- **Position-Vestibular-Pause (PVP) neurons:** The primary premotor interneurons of the **VOR.** During passive head rotation they fire in proportion to head velocity, projecting via the MLF to extraocular motor nuclei as the direct pathway of the three-neuron VOR arc. Their defining feature is that they pause during saccades — a suppression that prevents the VOR from interfering with voluntary gaze shifts — and their firing is also reduced during active VOR cancellation, distinguishing them from higher-order vestibular interneurons.
- **Vestibular-Only (VO) neurons:** Encode head velocity but carry no eye position signal and do not pause during saccades. Located primarily in the MVN and DVN, they project bilaterally to the cerebellum and to other VNC neurons. They are critical for **velocity storage** (see Section 12.3) and for transmitting vestibular signals to non-oculomotor targets including the spinal cord and autonomic centres.
- **Floccular Target Neurons (FTNs):** Located in the SVN and MVN, FTNs receive direct inhibitory input from floccular Purkinje cells. They are the effector neurons through **which cerebellar learning modifies VOR gain**: when the flocculus detects a gain error via climbing fibre signals, it adjusts its inhibitory output onto FTNs, directly changing their contribution to the VOR arc and producing the observed gain adaptation.
- **Type I neurons:** Excited by ipsilateral horizontal canal stimulation. Project to the contralateral abducens nucleus via the MLF, forming the excitatory limb of the horizontal VOR push-pull network.
- **Type II neurons:** Inhibited by ipsilateral canal stimulation and excited by contralateral input via commissural fibres from contralateral Type I neurons. They form the inhibitory limb of the bilateral push-pull network, sharpening directional tuning and extending the effective dynamic range of the system.

Companion reference: Physiology document, Section 5.1 — PVP, VO, and FTN neuron physiology; velocity storage vs. gaze-holding integration distinction.

11.4 Accessory Vestibular Groups

- **Group Y (nucleus Y):** Located dorsolateral to the inferior cerebellar peduncle (restiform body). Acts as a critical interface between the cerebellum (flocculus) and the oculomotor system, relaying upward eye position and velocity signals from the vertical canals and saccule to CN III/IV. Essential for **vertical VOR and floccular smooth pursuit control.**

- **Nucleus Prepositus Hypoglossi (NPH):** Located medial to the MVN, adjacent to the hypoglossal nucleus. The NPH is the **neural integrator for horizontal eye movements** — it mathematically integrates the eye velocity command from the VOR arc into a sustained position-holding signal, converting a transient velocity input into the tonic firing required to hold the eye at an eccentric gaze angle. A lesion of the NPH produces gaze-evoked nystagmus: following each saccade, the eye drifts back toward the primary position because the position-holding signal decays, and a corrective saccade is repeatedly required to reacquire the target.

11.5 The Commissural System

The two VNCs are interconnected by a rich system of commissural fibres that cross the brainstem midline [30]. This commissural system is the anatomical substrate for the push-pull bilateral organisation of the vestibular system:

- **Inhibitory commissural fibres (GABA-ergic and glycinergic):** The dominant pathway. When ipsilateral labyrinth excites the ipsilateral VNC, commissural inhibitory fibres simultaneously suppress the contralateral VNC. This sharpens directional tuning and doubles the effective dynamic range.
- **Excitatory commissural fibres (glutamatergic):** A smaller subpopulation. May mediate velocity storage and bilateral synchronisation during symmetric bilateral stimulation.

The commissural system is also critical for vestibular compensation after unilateral vestibular loss. In the acute phase, the intact side generates unopposed tonic input, producing spontaneous nystagmus and postural tilt toward the lesioned side. Over days to weeks, the commissural inhibition from the intact side onto the lesioned VNC decreases, and the lesioned VNC upregulates its intrinsic firing rate via post-inhibitory rebound plasticity, gradually restoring bilateral symmetry [31].

11.6 Neuropharmacology of the VNC

Transmitter	Source / Pathway	Receptor Types	Clinical Pharmacology
Glutamate	Hair cell afferents → VNC; ascending VOR pathways	AMPA, NMDA, mGluR	Primary excitatory transmitter. Vestibular suppressants reduce glutamate drive indirectly — benzodiazepines via GABAergic potentiation, antihistamines via histaminergic modulation — rather than through direct glutamate receptor antagonism
GABA	Commissural fibres; floccular Purkinje cells → VNC; reticular formation	GABAA, GABAB	GABA-B agonists (baclofen) are useful in downbeat nystagmus and for reducing pathologically prolonged velocity storage
Glycine	Some commissural fibres; inhibitory interneurons	Glycine receptors	Glycinergic inhibition of VNC contributes to bilateral push-pull
Histamine	Tuberomammillary nucleus (hypothalamus) → VNC	H1, H2, H3	Betahistine acts as an H3 receptor antagonist, increasing endogenous histamine release, producing vasodilation of the

Transmitter	Source / Pathway	Receptor Types	Clinical Pharmacology
			stria vascularis and dark cells, and modulating MVN excitability. H1 antagonists (antihistamines, promethazine) suppress vestibular responses and produce sedation via central H1 blockade
Acetylcholine	Efferent vestibular system; cholinergic pontine nuclei	Nicotinic, muscarinic	Scopolamine (muscarinic antagonist) is used for motion sickness prophylaxis, acting on the vestibular nuclear complex and nucleus of the solitary tract (NTS)
Dopamine	Substantia nigra and ventral tegmental area (VTA) projections	D1, D2	Dopaminergic projections modulate VNC gain. Antiemetics including metoclopramide and prochlorperazine act via D2 blockade in the area postrema

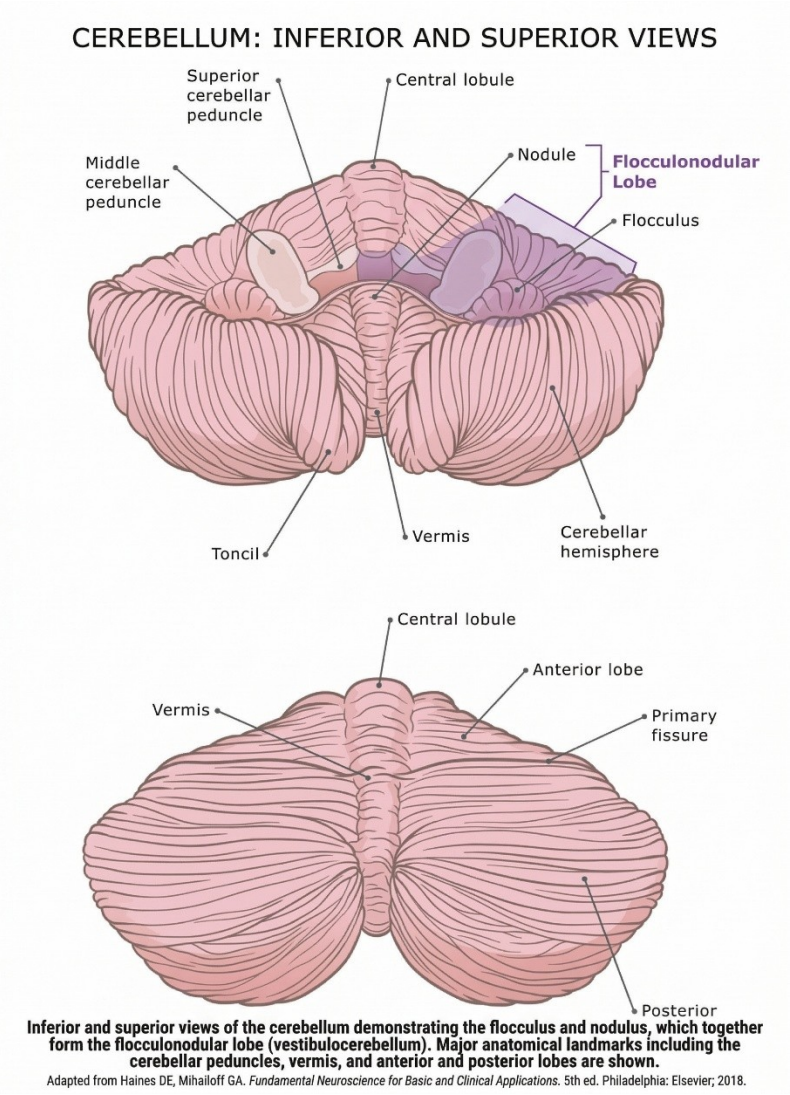
Companion reference: Physiology document, Section 5.3 — neuropharmacology of the vestibular nuclei; intrinsic membrane properties and plasticity.

12. Vestibulocerebellar Integration

12.1 Overview

The cerebellum is not simply a modulator of vestibular reflexes — it is an obligatory computational element in every vestibular circuit. No vestibular reflex operates normally without intact cerebellar input. Cerebellar vestibular function is understood through two conceptual frameworks: **adaptive calibration** (keeping reflexes accurate over a lifetime) and **predictive coding** (distinguishing expected from unexpected motion) [32].

The vestibulocerebellum (flocculonodular lobe = lobule X and adjacent lobules) is the cerebellar region most directly connected to the vestibular system. It comprises two anatomically and functionally distinct modules.



Inferior and superior views of the cerebellum demonstrating the flocculus and nodulus, which together form the flocculonodular lobe (vestibulocerebellum). Major anatomical landmarks including the cerebellar peduncles, vermis, and anterior and posterior lobes are shown.

Adapted from Haines DE, Mihailoff GA. *Fundamental Neuroscience for Basic and Clinical Applications*. 5th ed. Philadelphia: Elsevier; 2018.

12.2 The Flocculus and Paraflocculus

The flocculus (lobule X lateral) and paraflocculus (lobule IX lateral) are the primary sites of VOR gain adaptation and smooth pursuit control [33].

12.2.1 Connections

- **Mossy fibre inputs:** From the VNC (MVN, SVN), nucleus prepositus hypoglossi (NPH), and pontine nuclei carrying visual signals via the optic tract and pretectum.
- **Climbing fibre inputs:** From the dorsal cap of the inferior olive — the error signal pathway. Climbing fibres fire when retinal slip is detected, that is, when the image moves on the retina during head movement, indicating that VOR gain is inaccurate. This firing signals to the Purkinje cells that gain adjustment is required.
- **Purkinje cell output:** Inhibitory (GABAergic). Projects directly to the Floccular Target Neurons (FTNs) in the MVN and SVN — the efferent pathway by which the flocculus reduces VOR gain when gain is too high, or releases inhibition to allow gain to increase when gain is too low.

12.2.2 VOR Gain Adaptation — Cellular Mechanism

When VOR gain is inaccurate (e.g., wearing magnifying or minifying lenses), retinal slip triggers climbing fibre firing. This coincides with parallel fibre (granule cell) activity in the flocculus. The co-activation of climbing fibres + parallel fibres triggers **Long-Term Depression (LTD)** at the parallel fibre-Purkinje cell synapse [34]:

- **LTD mechanism:** Climbing fibre activation raises intracellular Ca2+ in the Purkinje cell. Simultaneous parallel fibre input activates mGluR1 and AMPA receptors. The coincidence of Ca2+ together with mGluR1/PKC signalling triggers AMPA receptor internalisation at the parallel fibre synapse, reducing Purkinje cell excitability, reducing inhibition of FTNs, and increasing VOR gain. The reverse sequence applies when gain is too high.
- **Timescale:** Early adaptation within minutes-hours (LTD at parallel fibre synapses). Consolidation and long-term storage may involve downstream VNC plasticity and even cortical changes.

Clinically, patients with floccular damage cannot adapt their VOR. They are unable to use spectacle corrections effectively and develop oscillopsia with prismatic lenses. Smooth pursuit is also impaired, as the flocculus carries smooth pursuit commands in addition to VOR calibration signals [33].

Companion reference: Physiology document, Section 7.2 — LTD, climbing fibre error signals, VOR/OKR adaptation.

12.3 The Nodulus and Uvula (Lobules IX–X, Medial)

The nodulus (lobule X medial) and ventral uvula (lobule IX medial) are concerned with gravity and velocity storage — fundamentally different functions from the flocculus [35].

12.3.1 Velocity Storage — Anatomy

The raw semicircular canal signal (cupula displacement) has a time constant of approximately 6 seconds, decaying as the cupula returns to equilibrium even during sustained rotation. The nodulus and uvula extend this to approximately 16–20 seconds via the velocity storage mechanism: VO neurons in the VNC form recurrent circuits that hold the vestibular signal in a charged state beyond the mechanical time constant of the cupula [36].

The nodulus inhibits velocity storage via Purkinje cell projections to the DVN and MVN. When the head tilts, the resulting otolith signal causes the nodulus to accelerate the decay of stored velocity, preventing spatial disorientation — a mechanism termed tilt dumping. Nodulus lesions impair this regulation, producing pathologically prolonged velocity storage and resulting in Periodic Alternating Nystagmus (PAN) or prolonged post-rotatory nystagmus [36].

12.3.2 Tilt/Translation Disambiguation

The otolith organs face an inherent ambiguity known as Einstein's Equivalence Principle: a tilt of the head in gravity is physically indistinguishable from a linear acceleration based on otolith output alone [37]. The nodulus resolves this by integrating canal signals, which encode rotation but not tilt, with otolith signals. If canal rotation signals accompany otolith activation, the system infers tilt; if the otolith activates without canal input, the system infers translation. Nodulus lesions impair this disambiguation, causing patients to misperceive translation as tilt and vice versa.

♥ CLINICAL PEARL: Cerebellar Nystagmus — Central Vestibular Signs

Downbeat nystagmus (fast phase downward): produced by floccular or paramedian cerebellar lesions. The eyes drift upward because the gaze-stabilising system fails in the upward direction. Causes include cerebellar degeneration, Arnold-Chiari malformation, vitamin B12 deficiency, and lithium toxicity. Treatment options include baclofen and 4-aminopyridine, a potassium channel blocker that increases Purkinje cell excitability.

Upbeat nystagmus: associated with anterior cerebellar vermis, medullary, or paramedian tract lesions. Wernicke's encephalopathy should be considered and thiamine administered before glucose in any at-risk patient.

Periodic Alternating Nystagmus (PAN): spontaneous nystagmus that reverses direction approximately every 90–120 seconds, reflecting nodulus dysfunction with failure to regulate velocity storage. May also be congenital. Treatment: baclofen, acting as a GABA-B agonist to suppress velocity storage.
Gaze-evoked nystagmus: reflects dysfunction of the neural integrator (NPH or MVN). Following each saccade, the eyes drift back toward the primary position because the position-holding signal decays — the integrator is insufficiently stable. Common causes include sedatives, anticonvulsants, and diffuse cerebellar disease. Horizontal and vertical components must be assessed separately, as they may reflect different lesion sites.

13. The Vestibulo-Ocular Reflex — Anatomy of the Three-Neuron Arc

13.1 Function and Performance Characteristics

The VOR generates compensatory eye movements equal and opposite to head rotation, maintaining stable gaze during head movement. Its key performance characteristics [38]:

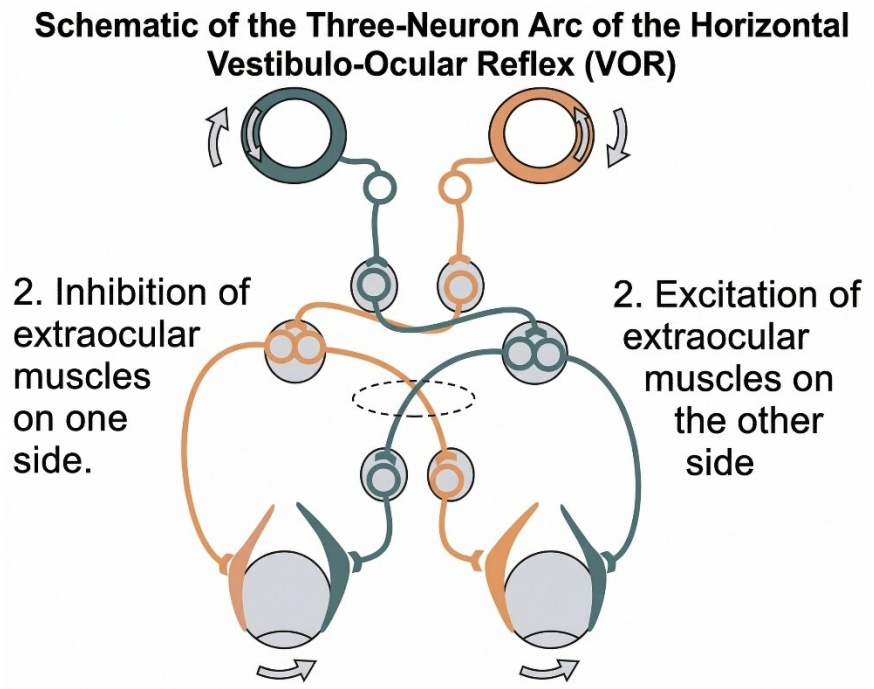
- **Latency:** 7–15 ms (often cited as less than 10 ms) — far shorter than the visual feedback latency of approximately 100 ms, and achievable only because of the pre-programmed three-neuron arc architecture.
- **Gain:** Ideally 1.0, where eye velocity equals head velocity with opposite sign. The normal range is 0.8–1.2. On vHIT assessment, a gain below 0.6 indicates significant unilateral vestibular hypofunction on that side.
- **Bandwidth:** Functional from 0.05 Hz to more than 10 Hz. The vHIT tests the high-frequency end (5–15 Hz head impulses); caloric testing probes the extremely low-frequency end (~0.003 Hz).

Companion reference: Physiology document, Section 6.1 — VOR physiology, gain/phase, velocity storage, VOR cancellation, gain adaptation.

13.2 The Horizontal Three-Neuron Arc — Complete Pathway

13.2 The Horizontal Three-Neuron Arc — Complete Pathway

The classical VOR circuit for the horizontal canal, using rightward head rotation as the example:



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

- **1st neuron:** Right lateral semicircular canal (SCC) hair cells (ampullopetal deflection) → Type I irregular afferents → Scarpa's ganglion (right, superior division) → right CN VIII → pontomedullary junction → right vestibular nuclei (MVN, SVN).
- **2nd neuron (excitatory):** Right MVN → crosses midline via the paramedian pontine reticular formation (PPRF) → left CN VI nucleus (abducens). Simultaneously, right MVN → via the left MLF → right CN III nucleus (activating medial rectus). Result: left lateral rectus and right medial rectus contract, turning both eyes left and compensating rightward head rotation.
- **2nd neuron (inhibitory):** Right MVN → via inhibitory interneurons → right CN VI nucleus is inhibited. Right MVN → via commissural fibres → left MVN is inhibited. This bilateral inhibition ensures crisp, rapid VOR responses without co-contraction.

- **3rd neuron:** CN VI motor neurons → left lateral rectus; CN III motor neurons → right medial rectus. Final common pathway to the extraocular muscles.

13.3 The Medial Longitudinal Fasciculus (MLF)

The MLF is the anatomical backbone of the VOR and vertical gaze coordination. It is a paired white matter tract running in the dorsal tegmentum of the pons and midbrain, adjacent to the midline, from the mesencephalon down to the cervical spinal cord [39]. It carries:

- **Horizontal VOR signal:** From contralateral CN VI nucleus → via MLF → CN III nucleus (medial rectus activation). This is the internuclear pathway.
- **Vertical VOR signal:** From SVN → via MLF → CN III and CN IV nuclei.
- **Vestibulocollic signal:** Continues caudally as the MVST to the cervical spinal cord.

♥ CLINICAL PEARL: INO — Internuclear Ophthalmoplegia — MLF Anatomy
Anatomy: The MLF carries the internuclear signal from the abducens interneurons in the CN VI nucleus to the contralateral CN III nucleus (medial rectus subdivision). A lesion of the MLF between these two nuclei produces an internuclear ophthalmoplegia (INO).
Signs: Ipsilateral adduction failure on horizontal gaze, with contralateral abducting nystagmus. The abducting nystagmus arises because the full conjugate gaze drive reaches the abducens nucleus but cannot be transmitted to the contralateral medial rectus, leaving lateral rectus activation without its paired adduction — the resulting imbalance produces nystagmus in the abducting eye. Convergence is typically preserved, as converging input reaches CN III via a pathway independent of the MLF.
Localisation: The lesion is on the same side as the adduction failure, as the MLF carries the signal destined for the ipsilateral medial rectus.
Causes: In younger patients, INO most commonly reflects demyelination from multiple sclerosis; in older patients, a small vessel lacunar infarct is the more likely cause. MS may produce bilateral INO — bilateral adduction failure in a young woman is MS until proven otherwise.
One-and-a-half syndrome: A unilateral pontine lesion involving both the MLF and the ipsilateral PPRF produces an ipsilateral conjugate gaze palsy combined with an internuclear ophthalmoplegia in the remaining direction of gaze. The only preserved horizontal eye movement is abduction of the contralateral eye — the 'half' that remains.

13.4 Vertical VOR Anatomy

The vertical VOR is anatomically more complex than the horizontal VOR because it crosses the anatomical mid-sagittal plane [40]:

- **Upward VOR (anterior canal driven):** Right anterior canal excited → right SVN → MLF → bilateral CN III (contralateral superior rectus + ipsilateral inferior oblique). Simultaneously inhibits bilateral CN IV (inferior oblique antagonists).
- **Downward VOR (posterior canal driven):** Right posterior canal excited → right MVN → contralateral CN IV nucleus (superior oblique) + ipsilateral CN III inferior rectus subdivision.

The crossing nature of the vertical VOR pathway is clinically significant: anterior canal signals from the right VNC cross via the MLF to activate the left superior rectus at the contralateral CN III nucleus. This decussation explains why skew deviation — produced by disruption of the otolith-ocular pathway in the brainstem — results in a contralateral hypertropia relative to the lesion side. The asymmetric otolith input is misinterpreted by the brain as a head tilt, triggering a corrective ocular tilt reaction: the full triad of head tilt, skew deviation, and ocular torsion reflects this misdirected compensatory response [41].

14. Vestibulospinal Pathways

14.1 Overview

The vestibulospinal tracts are the motor output limb of the vestibulospinal reflex (VSR) — the mechanism by which the vestibular system drives postural muscle activity to counteract gravity and prevent falls. Two anatomically and functionally distinct tracts operate in parallel [42]:

Feature	Lateral VST (LVST)	Medial VST (MVST)
Origin	Lateral Vestibular Nucleus (Deiters')	Medial Vestibular Nucleus
Laterality	Ipsilateral (uncrossed); runs in ventral funiculus	Bilateral; runs within the MLF
Spinal levels	All levels (cervical → lumbosacral)	Cervical + upper thoracic cord only
Primary input	Utricle (gravity/linear acceleration); cerebellar vermis	Semicircular cristae (rotation signals)
Motor neuron target	α and γ motor neurons of extensor (antigravity) muscles	Neck and upper trunk motor neurons
Somatotopy (LVST)	Dorsal LVN → cervical cord; ventral LVN → lumbar cord	Not somatotopically organised
Reflex mediated	Vestibulospinal reflex (VSR) — prevents falls	Vestibulocollic reflex (VCR) — stabilises head
Lesion effect	Asymmetric LVST damage → lateropulsion, falls toward lesion	Impaired head-righting; abnormal VCR

14.2 The Lateral Vestibulospinal Tract

The LVST originates from the Lateral Vestibular Nucleus (Deiters' nucleus), which contains the largest neurons in the brainstem (giant Deiters' cells). It descends ipsilaterally in the ventral funiculus of the spinal cord [43], exerting excitatory drive on extensor (antigravity) motor neurons at all spinal levels. The LVN has a strict somatotopic organisation: dorsal regions project to cervical cord (upper limb/neck); ventral regions project to lumbosacral cord (lower limb). The primary input driving the LVN is the **utricle** — static head tilt away from vertical activates the LVN on the downward side, reflexly extending the downward limbs to restore upright posture.

14.3 The Medial Vestibulospinal Tract

The MVST descends bilaterally within the MLF (caudally) to the cervical and upper thoracic cord. Its primary inputs are the semicircular canal cristae (via the MVN). It drives the Vestibulocollic reflex (VCR): when the body is suddenly displaced, rapid angular motion stimulates the cristae → MVST activates neck muscles to stabilise the head in space [44]. The VCR and VOR are functionally complementary: the VOR stabilises gaze by moving the eyes; the VCR stabilises the platform on which the eyes sit (the head).

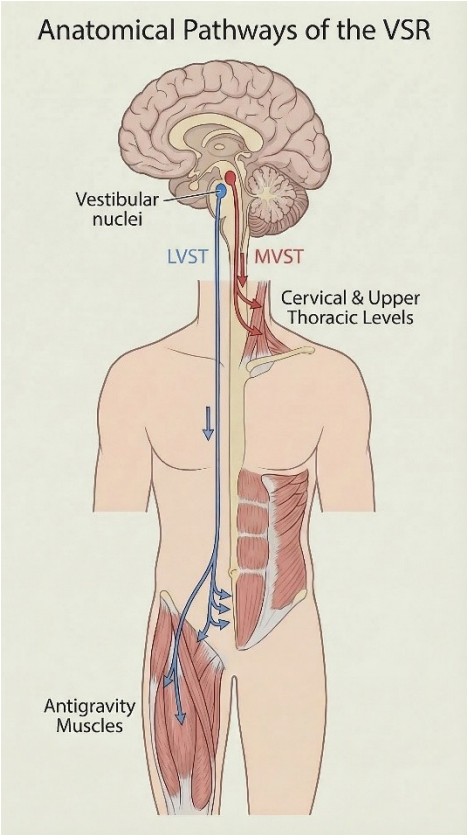
♥ CLINICAL PEARL: Lateropulsion in Wallenberg Syndrome — LVST Anatomy

PICA territory infarction involves the ipsilateral lateral medulla, producing a constellation of deficits: damage to the vestibular nuclei (lateral and inferior predominantly) and portions of the LVST and MVST origins; ipsilateral nucleus ambiguus (CN IX and X) causing dysphagia and hoarseness; ipsilateral sympathetic fibres causing Horner's syndrome; ipsilateral spinal trigeminal nucleus causing ipsilateral facial hemihypoalgesia; and the spinothalamic tract causing contralateral body hemihypoalgesia.

Lateropulsion — the tendency to fall or lean toward the side of the lesion — arises from asymmetric otolith-tilt signalling from the damaged vestibular nuclei, driving the LVST asymmetrically. Ocular lateropulsion (ipsilateral conjugate eye deviation at rest) and ipsipulsion of saccades accompany the postural findings.

The Ocular Tilt Reaction (OTR) — comprising ipsilateral head tilt, ipsilateral hypotropia, ipsilateral ocular torsion, and ipsilateral tilting of the subjective visual vertical — is a hallmark of **Wallenberg syndrome**, reflecting asymmetric otolith-ocular pathway function from lateral medullary involvement.

The HINTS pattern in Wallenberg syndrome is that of a central vestibular lesion: a normal or near-normal head impulse test (the horizontal canal is often relatively preserved), direction-changing or torsional nystagmus, and a positive test of skew. This pattern should always prompt urgent neuroimaging.



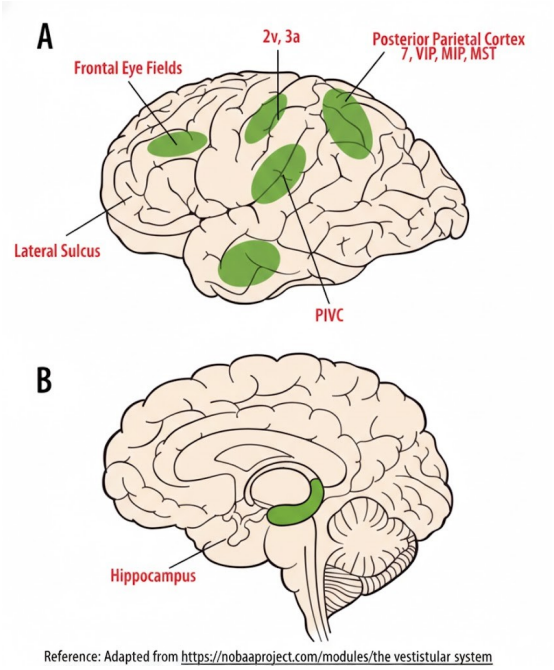
Companion reference: Physiology document, Section 6.5 — VSR physiology, clinical assessment of postural instability.

15. Ascending Vestibular Pathways and the Vestibular Cortex

15.1 The Vestibulo-Thalamic Projection

Vestibular signals ascend from the VNC primarily via the vestibulo-thalamic tract to multiple thalamic nuclei [45]. Unlike the visual and somatosensory systems, which have a single, topographically organised primary cortex, the vestibular system projects to a distributed thalamo-cortical network:

- **Ventral Posterior Inferior (VPI) and Ventral Posterior Lateral (VPL):** The 'core' vestibular relay. Project to the parietal operculum and somatosensory cortex. In humans, the VPI region is the main thalamic nucleus relaying to the PIVC (see below).
- **Ventral Lateral (VL) nucleus:** Projects to motor cortex (Area 4/6). Involved in postural planning and integrating vestibular signals into voluntary motor control.
- **Posterior Nuclear Group (Po):** Projects to the insular cortex and retroinsular cortex.
- **Pulvinar:** Integrates visual and vestibular motion signals; projects to posterior parietal cortex.



Reference: Adapted from https://nobaaproject.com/modules/the vestistular system

The thalamic relay also conveys vestibular signals to limbic areas via the **mediodorsal thalamus** and its projections to the prefrontal cortex. Separately, direct projections from the VNC to the locus coeruleus provide a noradrenergic link between vestibular disturbance and arousal and anxiety responses — a connection explored further in Section 16 [46].

15.2 The Vestibular Cortex — PIVC and PIC

There is no single "primary vestibular cortex" in humans [47]. Instead, vestibular information is distributed across a network of multimodal areas in and around the Sylvian fissure. Two hubs have been consistently identified by fMRI and lesion studies:

Area	Location	Activation	Key Function
PIVC (Parieto-Insular Vestibular Cortex)	Parietal operculum, Sylvian fissure (Area OP2 / Brodmann's Area 43)	Vestibular stimulation: activated. Visual motion: inhibited	Primary vestibular area. Conscious sense of self-rotation. Visual-vestibular suppression to prevent motion sickness.
PIC (Posterior Insular Cortex)	Retroinsular cortex, posterior to PIVC	Vestibular and visual motion: both activate it	Visual-vestibular integration. Estimates head motion from combined optic flow and vestibular signals. Key for self-motion perception.

A critically important anatomical principle: PIVC and visual motion cortex (area V5/MT) have a reciprocally inhibitory relationship [48]. When visual motion cortex is activated during optokinetic stimulation, it inhibits PIVC, and vice versa. This mutual suppression is thought to resolve sensory conflict during normal self-motion, preventing dizziness from simultaneous vestibular and visual inputs. Failure of this cortical suppression — whether from cerebellar lesions that remove adaptive gain control, central sensitisation, or psychological factors — may underlie visually induced dizziness and PPPD (Persistent Postural-Perceptual Dizziness). This represents a cortical-level mechanism distinct from peripheral or brainstem explanations of visual sensitivity and is relevant to understanding why visually induced dizziness persists in the absence of any ongoing peripheral vestibular deficit.

15.3 Right Hemisphere Dominance

Caloric and galvanic vestibular stimulation studies consistently demonstrate greater activation of the right hemisphere compared to the left in right-handed individuals [47]. This right-hemisphere dominance for vestibular cortical processing may explain why:

- **Right parietal lesions:** More likely to cause spatial disorientation and severe subjective visual vertical deviation than equivalent left-sided lesions.
- **Thalamic astasia:** Left-sided thalamic lesions may cause more prominent postural instability because the left thalamus projects predominantly to the right hemisphere — the vestibularly dominant side — meaning that a left thalamic lesion disproportionately disrupts the dominant vestibular thalamo-cortical relay.

♥ CLINICAL PEARL: Thalamic Astasia — Anatomy

Lesion: Posterolateral thalamus, specifically the VPL/VPI region (the graviceptive thalamus). Typically, a small lacunar infarct.

Signs: Profound postural instability — the patient cannot sit or stand unsupported and falls to the contralateral side — despite normal limb power, coordination, and sensation on standard neurological testing.

Mechanism: The thalamic VPL/VPI is the relay for vestibular signals to the motor and somatosensory cortices. Disruption eliminates central upright-orientation signals, effectively removing the patient's internal gravity reference.

The disproportionate postural instability in the context of a near-normal neurological examination is the clinical hallmark. Awareness of this syndrome is essential to avoid misattribution to a functional disorder.

Distinguishing features from functional disorder: Acute onset; CT or MRI demonstrating a lacunar infarct in the posterolateral thalamus; absence of give-way weakness; Romberg strongly positive in all conditions including those that would be expected to improve performance such as visual input or firm surface; and objective tilting of the subjective visual vertical (SVV) contralaterally — a measurable finding absent in functional postural disorders.

15.4 Posterior Parietal Cortex

Beyond the PIVC/PIC network, vestibular signals reach the posterior parietal cortex (PPC), including area 7 and the ventral intraparietal sulcus (VIP) [49]. The PPC integrates **vestibular, visual, and proprioceptive signals** to construct the body schema — the internal model of where the body is in space. PPC lesions produce:

- **Contralateral hemineglect:** Most commonly from right PPC damage; patient ignores contralateral space. Vestibular stimulation (cold caloric) temporarily reduces neglect — the right hemisphere is transiently activated, temporarily restoring spatial representation.
- **Pusher syndrome:** Patient actively pushes toward the weak (contralateral) side despite able to stand. Seen with right PPC or thalamic lesions. The patient perceives their own pushing as upright — their internal vertical reference is shifted. Responds to visual feedback strategies in rehabilitation.

16. Vestibulo-Autonomic Integration

16.1 Anatomical Pathways

The vestibular nuclei (principally MVN and DVN) project to multiple autonomic centres, providing the anatomical substrate for the systemic symptoms of vestibular disease [50]:

- **Rostral Ventrolateral Medulla (RVLM):** The master sympathetic vasomotor centre. Receives direct projections from the DVN and MVN. RVLM activation increases vascular resistance and cardiac output and is the origin of the otolith-driven vestibulo-sympathetic reflex (VSympR).
- **Nucleus of the Solitary Tract (NTS):** The vagal sensory relay (CN IX and X inputs). Receives direct MVN input. Convergence of vestibular and visceral signals at the NTS mediates the nausea and vomiting of vestibular disturbance via projections to the Dorsal Motor Nucleus of CN X and the area postrema.
- **Locus Coeruleus (LC):** Noradrenergic nucleus in the dorsal pons. Receives input from the VNC and DVN. LC activation increases arousal, alertness, and anxiety. This connection explains the significant anxiety and panic that accompany acute vestibular loss — a neuroanatomically determined response rather than a purely psychological reaction.
- **Parabrachial Nucleus:** Pontine relay for visceral sensations that projects to the amygdala, contributing to fear and anxiety responses. Part of the circuit mediating motion sickness and the well-recognised vestibular-anxiety comorbidity.

16.2 The Vestibulo-Sympathetic Reflex

The vestibulo-sympathetic reflex (VSympR) is a feedforward cardiovascular control mechanism driven by the otolith organs [51]. The stimulus is head-up tilt detected by the utricular maculae as linear acceleration away from the gravity baseline. Utricular hair cells project via the superior vestibular nerve to the DVN and MVN, which in turn activate the RVLM. The RVLM drives the intermediolateral cell column (IML) of the thoracic spinal cord, activating sympathetic ganglia and producing vasoconstriction of the peripheral vasculature.

The functional significance of this feedforward architecture is that anticipatory vasoconstriction occurs before any blood pressure drop on standing. Unlike the baroreceptor reflex — a feedback mechanism that detects a pressure drop and then corrects it — the VSympR predicts the haemodynamic consequence of orthostasis and pre-empts it.

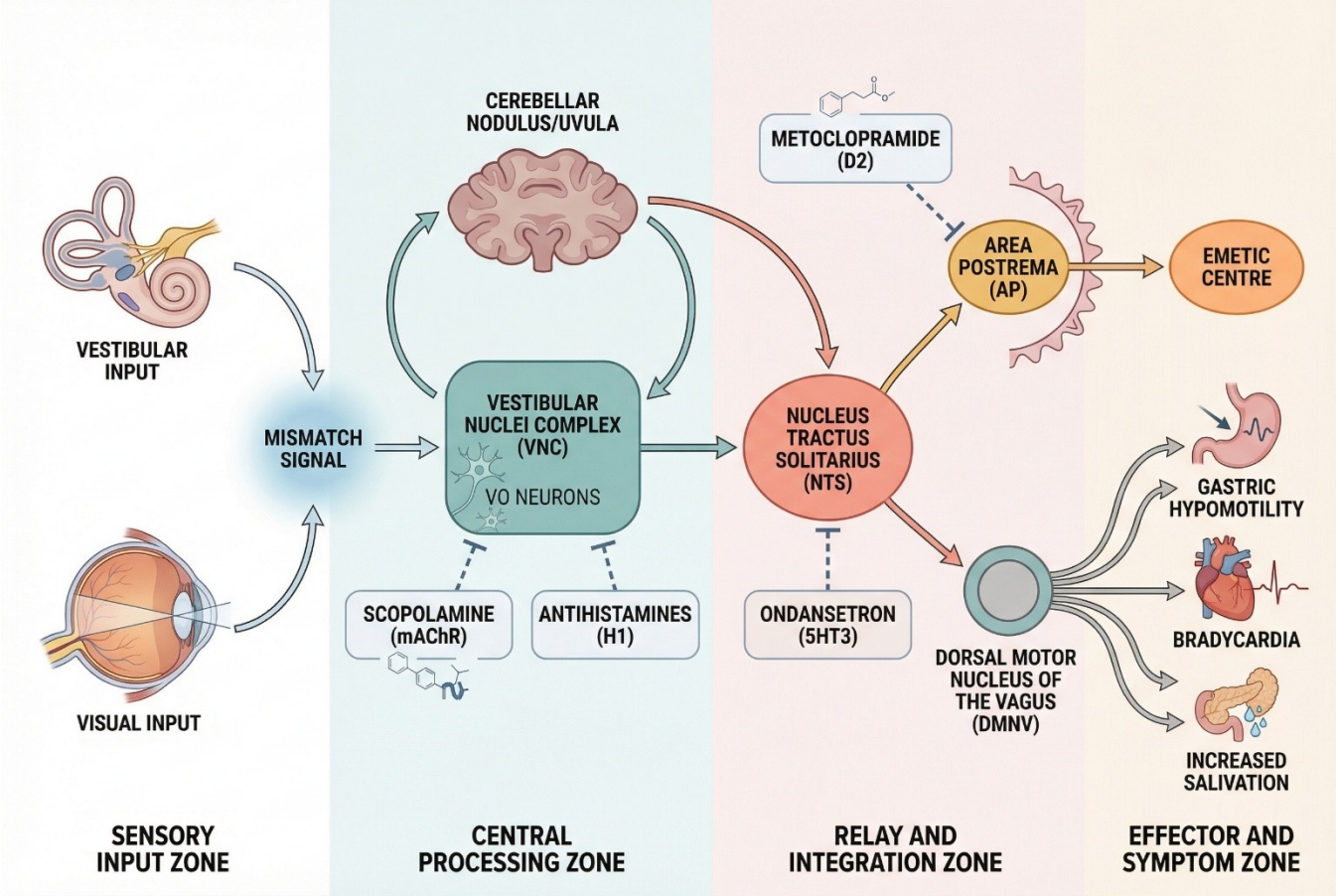
Patients with bilateral vestibular failure have impaired VSympR due to bilateral loss of otolith function, rendering them susceptible to orthostatic hypotension even in the absence of autonomic neuropathy [52]. This is an underappreciated clinical finding: dizziness on standing in a patient with known bilateral vestibular hypofunction may represent impaired VSympR rather than primary cardiac or autonomic disease.

16.3 Motion Sickness — Anatomical Circuit

Motion sickness results from sensory conflict: a mismatch between expected and actual vestibular and visual inputs [53]. The anatomical circuit proceeds as follows:

The mismatch signal is computed at the level of the VNC, particularly by VO neurons and the commissural system, possibly with contributions from cerebellar comparator mechanisms. The VNC projects directly to the NTS and also via the nodulus and uvula pathway. From the NTS, two parallel outputs drive the symptoms of motion sickness: projections to the Dorsal Motor Nucleus of the Vagus produce vagal efferent activation, causing gastric hypomotility, hypersalivation, and bradycardia; and projections to the area postrema (the chemoreceptor trigger zone, situated outside the blood-brain barrier) activate the emetic centre, producing nausea and vomiting.

Pharmacological targets reflect this circuit: scopolamine acts as a muscarinic ACh antagonist at the NTS and area postrema level; antihistamines (H1 antagonists) act at the VNC and NTS; metoclopramide acts via D2 blockade at the area postrema; and ondansetron acts via 5HT3 antagonism at the same site — the latter two sharing a common anatomical target but through distinct receptor mechanisms.



♥ CLINICAL PEARL: Vestibular Migraine and Autonomic Connections

Vestibular migraine (VM) is currently recognised as the most common cause of episodic vertigo in the outpatient setting, exceeding BPPV in frequency in vestibular specialist clinics.

The anatomical basis for vestibular symptoms in migraine lies in the trigemino-vascular system: the trigeminal nerve projects to dural vessels and has direct connections to the MVN and cerebellum via the trigeminal nucleus caudalis. Trigeminovascular activation during a migraine attack can directly drive VNC excitability changes, producing vestibular symptoms in the absence of any labyrinthine pathology.

The dorsal raphe nucleus projects serotonergic fibres to the VNC, cochlea, and vestibular labyrinth. Migraine-associated serotonin flux modulates peripheral vestibular function, providing a plausible anatomical explanation for the clinical overlap between vestibular migraine and Menière's disease.

PPPD (Persistent Postural-Perceptual Dizziness): Although distinct from vestibular migraine, PPPD shares relevant anatomical substrates. It is characterised by chronic dizziness, unsteadiness, and visual sensitivity, and the proposed mechanism involves maladaptive re-weighting of vestibular, visual, and proprioceptive inputs following an acute vestibular event. This re-weighting is thought to be driven by anxiety-augmented locus coeruleus activation — the connections between the locus coeruleus and the vestibular nuclei providing the anatomical substrate through which heightened arousal and anxiety perpetuate and amplify vestibular symptoms.

Companion reference: Physiology document, Section 6.6 — vestibulo-sympathetic reflex physiology.

17. Clinical Syndrome Localisation — Integrated Summary

The following table integrates vestibular anatomy with clinical localisation. Each syndrome reflects a specific disruption of the anatomical pathways described in this document:

Syndrome	Lesion Site	Vestibular Signs	Distinguishing Feature
Sup. Vestibular Neuritis	SVN / superior division Scarpa's ganglion	Horizontal-torsional nystagmus (fast phase away from lesion); abnormal horizontal vHIT; abnormal anterior canal vHIT; reduced oVEMP; normal cVEMP	Preserved hearing and saccule; most common cause of acute peripheral vertigo
Inf. Vestibular Neuritis	IVN / inferior division CN VIII	Normal horizontal vHIT; abnormal posterior canal vHIT; absent ipsilateral cVEMP; normal oVEMP	Rare; saccular loss without horizontal canal involvement
Posterior Canal BPPV	Otoconial debris in PSCC lumen	Upbeat-torsional nystagmus on Dix-Hallpike; latency 5–10s; fatigable	Positive Dix-Hallpike; Epley manoeuvre curative
Meniere's Disease	Endolymphatic hydrops (cochlea + saccule/utricle)	Episodic vertigo; fluctuating low-freq SNHL; aural fullness; tinnitus; abnormal oVEMP in late disease	MRI hydrops on 4h delayed gadolinium; electrocochleography
AVA Occlusion	Anterior vestibular artery	Acute severe vertigo; abnormal horizontal and anterior canal vHIT; reduced oVEMP; hearing preserved; intact cVEMP	Distinguished from SVN neuritis by absence of recovery
Labyrinthine Infarction	Labyrinthine artery (total)	Simultaneous sudden SNHL and complete unilateral vestibular loss	Hearing loss distinguishes from vestibular neuritis
AICA Infarction	AICA (labyrinth and lateral pons/cerebellum)	Total audiovestibular loss ipsilaterally + facial palsy, Horner's, hemiataxia, face numbness	Hearing loss combined with brainstem signs indicates AICA stroke
Wallenberg Syndrome	PICA (lateral medulla)	Vertigo; torsional nystagmus; ipsipulsion; OTR; Horner's; facial analgesia ipsilateral; body hemihypoalgesia contralateral	Hearing spared — PICA territory does not include the labyrinthine artery
INO	MLF (pons)	Ipsilateral adduction failure on lateral gaze + contralateral abducting nystagmus; convergence intact	Young patient: demyelination (MS); older patient: vascular. Lesion side = side of adduction failure.

Syndrome	Lesion Site	Vestibular Signs	Distinguishing Feature
Skew Deviation	Brainstem/cerebellar otolith pathway	Vertical ocular misalignment; full OTR triad (head tilt, ocular torsion, skew deviation)	Central sign, Bielschowsky head tilt test differentiates skew from 4th nerve palsy — vertical misalignment increases with ipsilateral head tilt in 4th nerve palsy but not in skew.
Thalamic Astasia	Posterolateral thalamus (VPL/VPI)	Profound postural instability with contralateral falls; normal motor and sensory exam	Lacunar infarct on CT/MRI; the near-normal routine neurological examination belies the severity of postural instability.
Vestibular Paroxysmia	AICA vascular loop compression of CN VIII	Brief stereotyped attacks of vertigo; sometimes auditory symptoms; hyperventilation may trigger	MRI AICA loop in IAC; responds to carbamazepine/oxcarbazepine
Superior Canal Dehiscence	Dehiscence of bone over anterior SCC	Tullio phenomenon; Hennebert sign; pulsatile tinnitus; reduced cVEMP threshold	CT temporal bones (Pöschl view); third window signs on VEMP

References

References are numbered in order of first citation in the text.

[1] Straka H, Baker R, Gilland E. The frog as a unique vertebrate model for studying the maturation of vestibular function. Brain Res Bull. 2001;55(4):509-516.

[2] Neuhauser HK. The epidemiology of dizziness and vertigo. Handb Clin Neurol. 2016;137:67-82.

[3] Gulya AJ. Anatomy of the temporal bone and ear. In: Gulya AJ, Minor LB, Poe DS, eds. Glasscock-Shambaugh's Surgery of the Ear. 6th ed. PMPH-USA; 2010.

[4] Lo WW, Solti-Bohman LG. The internal auditory canal. In: Valvassori GE, ed. Imaging of the Head and Neck. Thieme; 1995.

[5] Salt AN, Plontke SK. Endolymph composition: A spatial gradient of ionic concentrations along the length of the cochlea. Hear Res. 2018;362:1-10.

[6] Friberg U, Rask-Andersen H. Vascular occlusion in the endolymphatic sac in Meniere's disease. Ann Otol Rhinol Laryngol. 2002;111(3):237-245.

[7] Pullens B, van Benthem PP. Intratympanic gentamicin for Meniere's disease or syndrome. Cochrane Database Syst Rev. 2011;(3):CD008234.

[8] Fernandez C, Goldberg JM. Physiology of peripheral neurons innervating the otolith organs. J Neurophysiol. 1976;39:970-1008.

[9] Highstein SM, Goldberg JM, Moschovakis AK, et al. Inputs from regularly and irregularly discharging vestibular nerve afferents to secondary neurons in the vestibular nuclei. J Neurophysiol. 1987;58:479-512.

[10] Ewald JR. Physiologische Untersuchungen über das Endorgan des Nervus Octavus. Bergmann; 1892.

[11] Colebatch JG, Halmagyi GM. Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. Neurology. 1992;42(8):1635-1636.

[12] Boyle R, Highstein SM. Resting discharge and response dynamics of horizontal semicircular canal afferents in the toadfish, Opsanus tau. J Neurosci. 1990;10:1557-1569.

[13] Silver RB, Reeves AP, Steinacker A, Bhalla US. Examination of the cupula and its associated structures. J Neurosci. 1998;18:3023-3037.

[14] Shotwell SL, Jacobs R, Hudspeth AJ. Directional sensitivity of individual vertebrate hair cells to controlled deflection of their hair bundles. Ann N Y Acad Sci. 1981;374:1-10.

[15] Zhao X, Yang H, Yamoah EN, Lundberg YW. Otoconin-90 deletion leads to imbalance but normal hearing. Neuroscience. 2008;153(1):289-299.

[16] Lindeman HH. Regional differences in sensitivity of the vestibular sensory epithelia to ototoxic antibiotics. Acta Otolaryngol. 1969;67(2-6):177-189.

[17] Hudspeth AJ. How the ear's works work. Nature. 1989;341(6241):397-404.

[18] Contini D, Price SD, Art JJ. Nonquantal transmission at the vestibular hair cell–calyx synapse: KLV currents modulate fast electrical and slow K^+ potentials. PNAS. 2017;114(29):E6905-E6914.

[19] Moser T, Predoehl F, Starr A. Review of hair cell ribbon synapse dysfunction, pathology, and physiology. Otol Neurotol. 2013;34(7):1175-1185.

[20] Holt JC, Lysakowski A, Goldberg JM. Mechanisms of efferent-mediated responses in the turtle posterior crista. J Neurosci. 2006;26(51):13180-13193.

[21] Cullen KE. The vestibular system: Multimodal integration and encoding of self-motion for motor control. Trends Neurosci. 2012;35(3):185-196.

[22] Kim JS, Lee H. Inner ear dysfunction due to vertebrobasilar ischemic stroke. Semin Neurol. 2009;29(5):534-540.

[23] Hüfner K, Barresi D, Glaser M, et al. Vestibular paroxysmia: Diagnostic features and medical treatment. Neurology. 2008;71(13):1006-1014.

[24] Friberg U, Rask-Andersen H. Vascular occlusion in the endolymphatic sac in Meniere's disease. Ann Otol Rhinol Laryngol. 2002;111:237-245.

[25] Scarpa A. Anatomicae Disquisitiones de Auditu et Olfactu. Ticino; 1789.

[26] Brodal A. The vestibular nuclei in the cat. J Comp Neurol. 1952;98:55-133.

[27] Goldberg JM. Afferent diversity and the organisation of central vestibular pathways. Exp Brain Res. 2000;130(3):277-297.

[28] Highstein SM, Holstein GR. The anatomy of the vestibular nuclei. Prog Brain Res. 2006;151:157-203.

[29] Cullen KE, McCrea RA. Firing behaviour of brain stem neurons during voluntary cancellation of the horizontal vestibuloocular reflex. J Neurophysiol. 1993;70(2):828-843.
[30] Dieringer N, Precht W. Mechanisms of compensation for vestibular deficits in the frog. Exp Brain Res. 1977;30(2-3):209-226.
[31] Curthoys IS, Halmagyi GM. Vestibular compensation: A review of the oculomotor, neural, and clinical consequences of unilateral vestibular loss. J Vestib Res. 1995;5(2):67-107.
[32] Angelaki DE, Cullen KE. Vestibular system: The many facets of a multimodal sense. Annu Rev Neurosci. 2008;31:125-150.
[33] Zee DS, Yamazaki A, Butler PH, Gucer G. Effects of ablation of flocculus and paraflocculus on eye movements in primate. J Neurophysiol. 1981;46(4):878-899.
[34] Ito M. Long-term depression. Annu Rev Neurosci. 1989;12:85-102.
[35] Angelaki DE, Hess BJ. Inertial representation of angular motion in the vestibular system of rhesus monkeys. J Neurophysiol. 1994;71(3):1222-1249.
[36] Raphan T, Matsuo V, Cohen B. Velocity storage in the vestibulo-ocular reflex arc (VOR). Exp Brain Res. 1979;35(2):229-248.
[37] Mayne R. The functional limitations of the semicircular canal in relation to angular velocity and acceleration. In: Fischgold H, ed. First International Congress of Neurological Sciences; 1957.
[38] Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. Arch Neurol. 1988;45(7):737-739.
[39] Buttner-Ennever JA. A review of otolith pathways to brainstem and cerebellum. Ann N Y Acad Sci. 1999;871:51-64.
[40] Highstein SM. The central nervous system efferent control of the organs of balance and equilibrium. Neurosci Res. 1991;12(1):13-30.
[41] Brandt T, Dieterich M. Skew deviation with ocular torsion: A vestibular brainstem sign of topographic diagnostic value. Ann Neurol. 1993;33(5):528-534.
[42] Pompeiano O. Vestibulospinal relations: Vestibular influences on gamma motoneurons and primary afferents. Prog Brain Res. 1972;37:197-232.
[43] Wilson VJ, Maeda M. Connections between semicircular canals and neck motoneurons in the cat. J Neurophysiol. 1974;37(2):346-357.
[44] Bilotto G, Goldberg J, Peterson BW, Wilson VJ. Dynamic properties of vestibular reflexes in the decerebrate cat. Exp Brain Res. 1982;47(3):343-352.
[45] Dieterich M, Bense S, Stephan T, et al. Dominance for vestibular cortical function in the non-dominant hemisphere. Cereb Cortex. 2003;13(9):994-1007.
[46] Balaban CD, Thayer JF. Neurological bases for balance-anxiety links. J Anxiety Disord. 2001;15(1-2):53-79.
[47] Lopez C, Blanke O. The parieto-insular vestibular cortex in humans: More than a single area? J Neurophysiol. 2011;104(6):2946-2958.
[48] Brandt T, Dieterich M, Danek A. Vestibular cortex lesions affect the perception and neurophysiology of the visual-vestibular interaction. Ann Neurol. 1994;35(3):403-412.
[49] Bisley JW, Goldberg ME. Neuronal activity in the lateral intraparietal area and spatial attention. Science. 2003;299(5603):81-86.
[50] Holstein GR. Glutamate and GABA in vestibulo-sympathetic pathway neurons. Front Neuroanat. 2012;6:1-12.
[51] Yates BJ, Miller AD. Physiological evidence that the vestibular system participates in autonomic and respiratory control. J Vestib Res. 1998;8(1):17-25.
[52] Yates BJ, Jian BJ, Cotter LA, Cass SP. Responses of vestibular nucleus neurons to tilt following chronic bilateral removal of vestibular inputs. Exp Brain Res. 2000;130(2):151-158.
[53] Yates BJ, Miller AD, Lucot JB. Physiological basis and pharmacology of motion sickness: An update. Brain Res Bull. 1998;47(5):395-406.

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