Physiology of the Vestibular System

A Comprehensive Clinical Review

Companion to: The Anatomy of the Vestibular System

Australian Dizziness Clinics | **www.AustralianDizzinessClinics.com**

Australian Dizziness Clinic Team

Version 1.0 | April 2026

How to Use This Document

This is the companion physiology document to the Anatomy of the Vestibular System review. Structure follows a peripheral-to-central organisation mirroring the anatomy document. Clinical Pearl boxes highlight physiology-to-bedside correlations. Key Facts tables at section ends provide rapid-review summaries for examination preparation.

Table of Contents

How to Use This Document....1
Learning objectives....3
Take-home points....3
1. Introduction....4
Clinical testing map (sensor and pathway focus)....4
2. Peripheral Biomechanics and Hydrodynamics....5
2.1 The Semicircular Canals: Angular Accelerometers....6
2.2 The Otolith Organs: Linear Acceleration and Gravity....9
3. 3. Molecular Physiology of Hair Cell Transduction....11
3.1 The Mechanotransduction Channel Complex: TMC1 and TMC2....12
3.2 Adaptation Mechanisms....12
3.3 Potassium Conductances: Type I versus Type II Hair Cells....13
3.4 Synaptic Transmission: The Calyx Paradox....14
3.6 The Efferent Vestibular System: Central Control of Peripheral Gain....16
4. Afferent Encoding and Frequency Tuning....16
5. Central Vestibular Nuclei Architecture....18
5.1 Functional Classification of Neurons....18
5.2 Intrinsic Membrane Properties and Plasticity....20
5.3 Neuropharmacology....20
6. Reflex Systems and Control Theory....22
6.1.1 Angular VOR (Canal-driven)....22
6.1.2 Otolith Contributions to Ocular Control (Linear VOR and Ocular Tilt Reaction)....23
6.1.3 Velocity Storage and Visual–Vestibular Integration....23
6.1.4 VOR Cancellation....25
6.1.5 The 3D coordinate transform problem....26
6.1.5 Bedside Interpretation: Fast Arc vs Velocity Storage....26
6.1.5.1 Probing the Short-Latency Arc: vHIT and the Head Impulse Test....26
6.1.5.2 Probing the Long Time Constant: Headshake and Post-Rotatory Testing....26

6.1.6 Alexander's Law and Gaze Dependence 27
6.3.4 Gravity Dependence and Storage Regulation 27
6.4 The vestibulo-collic reflex (VCR) 27
6.5 The vestibulo-spinal reflex (VSR) 29
6.6 The vestibulo-sympathetic reflex 30
7. Cerebellar Computation and Predictive Coding 32
7.1 Internal Models and Predictive Coding 32
7.2 Synaptic Plasticity and Learning 33
7.3 The Vestibular Cerebellum Is Not One Module: Flocculus versus Nodulus/Uvula 33
Functional organisation of the vestibular cerebellum 33
Floccular complex (flocculus + paraflocculus): gaze stabilisation, cancellation, pursuit, gaze holding, and downbeat nystagmus 34
Nodulus and Ventral Uvula: Gravito-inertial computation, tilt–translation resolution, and velocity-storage regulation 36
Summary of vestibular cerebellar functions 37
7.4 Clinical and Experimental Implications 37
8. Thalamocortical Pathways and Spatial Cognition 38
8.1 The Vestibular Thalamus 38
8.2 Cortical Vestibular Pathways 39
8.3 Spatial Navigation: Head Direction, Grid Coding, and Path Integration 40
9. Physiological basis of recovery and rehabilitation 41
9.1 Adaptation 41
9.2 Substitution 41
9.3 Habituation 41
Reference List 42

The Physiology of the Vestibular System: From Mechanotransduction to Spatial Cognition

Abstract: This comprehensive review summarizes vestibular physiology from the peripheral labyrinth (semicircular canals and otolith organs) to central brainstem and cerebellar processing, key reflex outputs (especially the VOR and vestibulospinal pathways), and higher-order spatial perception. The emphasis is on high-yield mechanisms that explain common clinical tests and bedside signs.

Learning objectives

- Describe how semicircular canals encode angular acceleration and why their output behaves like head velocity over physiological frequencies.
- Explain how otolith organs encode gravito-inertial acceleration and why tilt–translation ambiguity must be solved centrally.
- Link peripheral and central physiology to common clinical tests (vHIT, calorics/rotational chair, VEMP, subjective visual vertical).
- Differentiate velocity storage (brainstem–cerebellar persistence of rotation signals) from the gaze-holding neural integrator (eye position holding).
- Outline core principles of vestibular compensation and how rehabilitation leverages adaptation, substitution, and habituation.

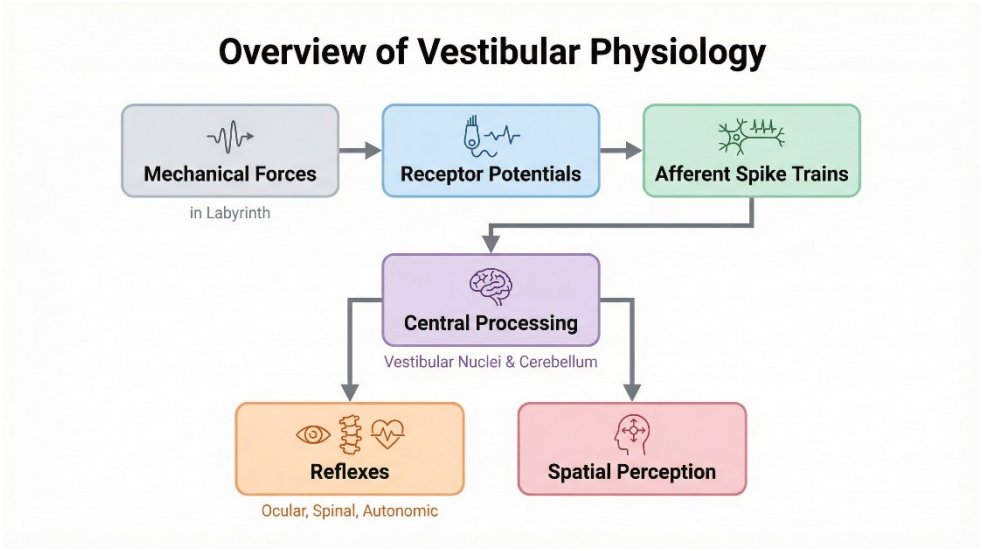
Take-home points

- Canals and otoliths are complementary inertial sensors: canals are rotation sensors; otoliths are gravity/translation sensors.
- Push–pull bilateral organisation improves dynamic range and explains spontaneous nystagmus after unilateral vestibular loss.
- Different tests probe different stimulus frequencies and pathways: vHIT emphasises high-frequency canal function; calorics and rotation emphasise low-to-mid frequencies; VEMPs probe otolith pathways.
- Cerebellum calibrates reflex gain and helps resolve sensory conflicts; failure can produce gaze-holding deficits, abnormal velocity storage, and visually induced dizziness phenotypes.
- Rehabilitation works by driving controlled error signals (retinal slip, postural error) while gradually reducing sensory conflict.

1. Introduction

The vestibular system is the inner-ear sensory system that detects head motion (rotation and translation) and head orientation relative to gravity. Its signals are essential for gaze stabilisation, postural control, and the perception of self-motion and upright. Because vestibular input is continuously combined with vision and proprioception, vestibular dysfunction can present with eye movement abnormalities, imbalance, oscillopsia, motion sensitivity, and disturbed perception of verticality.

Vestibular physiology is best understood as a sequence of transformations: mechanical forces in the labyrinth are converted into hair-cell receptor potentials, encoded as afferent spike trains, processed in vestibular nuclei and cerebellum, and then expressed as reflexes (ocular, spinal, autonomic) and as higher-order spatial perception. This review connects mechanisms to the clinical examination and to vestibular testing.



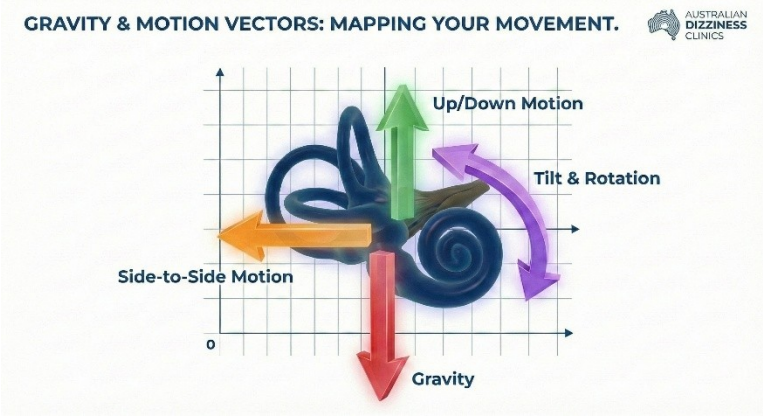
This review is organised from periphery to central pathways: Section 2 covers canal and otolith biomechanics; Section 3 summarises hair-cell mechanotransduction and synaptic output; Section 4 reviews vestibular afferent coding; Sections 5–6 cover vestibular nuclei, velocity storage, gaze holding, and major reflex pathways (VOR, vestibulospinal, vestibulo-collic, vestibulo-sympathetic); Sections 7–8 provide an account of cerebellar calibration and thalamocortical contributions to spatial perception; and Section 9 reviews compensation and rehabilitation principles.

Clinical testing map (sensor and pathway focus)

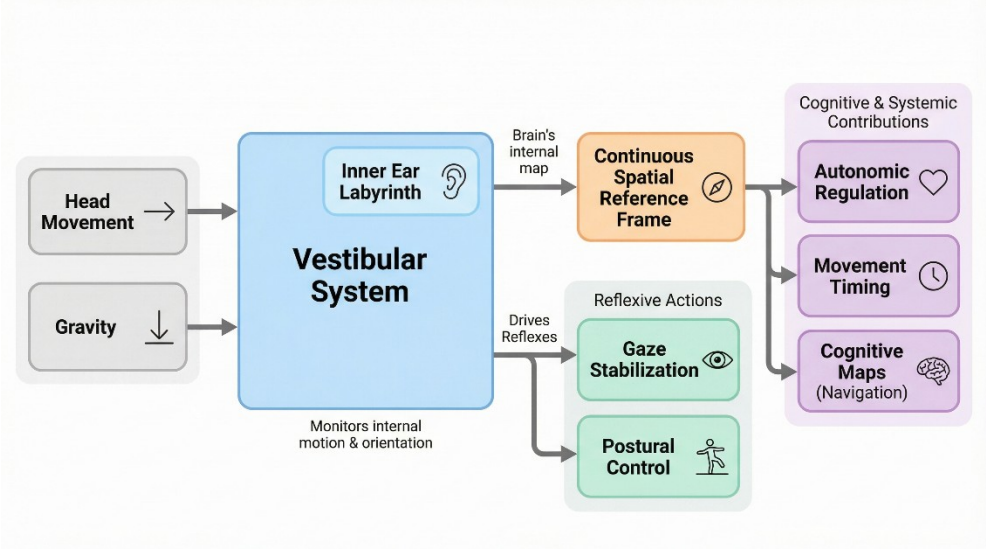
- **vHIT:** high-frequency semicircular canal function; identifies canal-specific hypofunction and catch-up saccades.
- **Calorics / rotational chair:** low-to-mid frequency canal function; helpful when vHIT is normal but symptoms persist.
- **cVEMP / oVEMP:** otolith pathways (saccule–inferior vestibular nerve; utricle–superior vestibular nerve) and their reflex projections.
- **Subjective visual vertical (SVV)** and **ocular tilt reaction signs:** graviceptive pathway integrity (mainly utricular/central).
- **Dynamic visual acuity and bedside head impulse:** functional correlates of VOR performance.

The vestibular system is the body's primary inertial guidance system. Phylogenetically, it is older than hearing and is present across vertebrate species as a fundamental mechanism for detecting motion. Unlike vision and hearing, which analyse stimuli originating outside the body, the vestibular system monitors internal motion and orientation. It senses head movement and position relative to gravity through specialised structures within the inner ear labyrinth.

Importantly, its function extends beyond simple reflexes. While it drives gaze stabilisation and postural control, it also provides a continuous reference frame that allows the brain to determine where the body is in space. This internal spatial reference contributes to autonomic regulation, movement timing, and the construction of cognitive maps used for navigation. For these reasons, the vestibular system is often described as a foundational sensory modality underlying both motor control and spatial perception. [1]



Building on this foundational role, modern neurophysiology no longer views the vestibular labyrinth as a simple reflex trigger. Instead, it is understood as part of a high-speed computational system. Within milliseconds, vestibular circuits differentiate head acceleration, integrate velocity signals over time, and transform information from canal and otolith coordinates into eye, head, and body motor commands. These operations resemble engineering control processes, but they are implemented by biological networks operating continuously in real time.



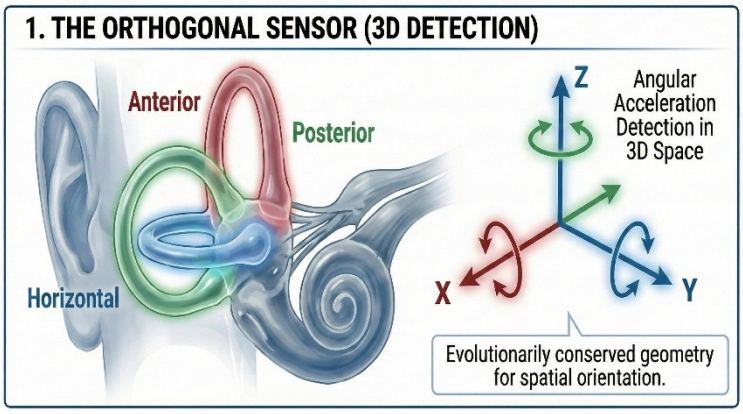
Accordingly, this review follows the vestibular signal across levels of organisation. We begin with the mechanical deflection of stereocilia at the nanometre scale, then examine how brainstem and cerebellar networks reshape these signals and finally consider how distributed cortical circuits generate conscious spatial awareness. By integrating molecular, systems, and clinical perspectives, the aim is to clarify how the central nervous system distinguishes self-generated from external motion, combines vestibular input with vision and proprioception, and preserves perceptual stability in a constantly changing environment. [1]

2. Peripheral Biomechanics and Hydrodynamics

Having outlined the system at a conceptual level, we now begin at the periphery, where mechanical forces are first converted into neural signals. The accuracy of vestibular perception depends critically on the mechanical properties of the peripheral end-organs within the membranous labyrinth. In practical terms, what the brain ultimately "knows" about motion is constrained by how these structures respond to acceleration across different frequencies.

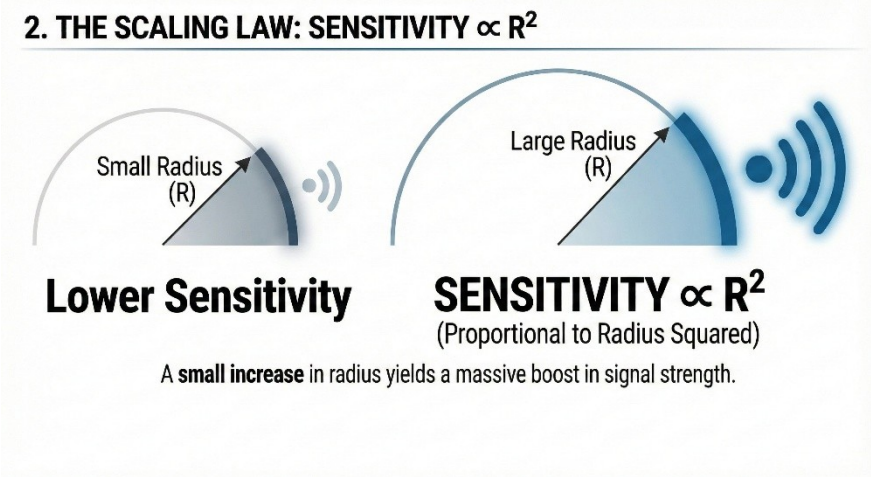
The three semicircular canals and the two otolith organs—the utricle and saccule—form this peripheral sensor array. Their role is to convert mechanical energy into bioelectrical activity. They do so through tightly regulated interactions between endolymph fluid and specialised gelatinous structures, which transmit force to hair cells. These fluid–structure dynamics determine the timing, magnitude, and frequency characteristics of the initial vestibular signal before it ever reaches the brainstem.

2.1 The Semicircular Canals: Angular Accelerometers

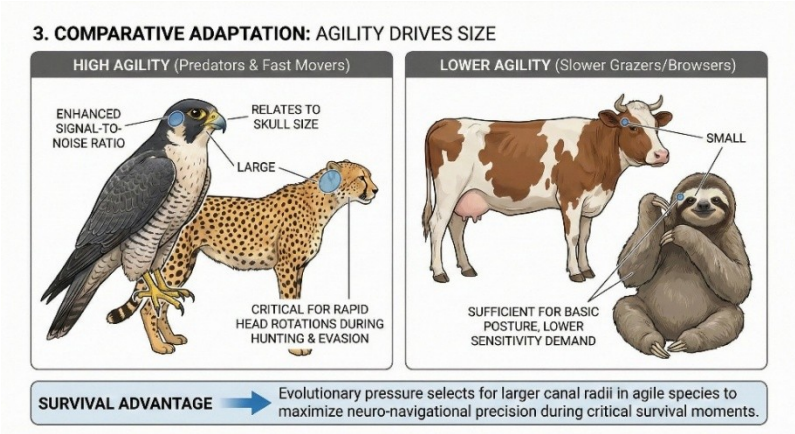


Having introduced the peripheral organs in general terms, we now focus on the semicircular canals, which are specialised for sensing rotation. The three canals—horizontal, anterior, and posterior—act as angular accelerometers. They are arranged in an approximately orthogonal configuration, so that rotation in any direction activates at least one canal pair strongly. Clinically, this corresponds to the three principal axes of head movement: yaw ("no"), pitch ("yes"), and roll (ear toward shoulder). This near-right-angle geometry is conserved across vertebrates because it provides an efficient solution for measuring rotation in three-dimensional space.

Although the overall arrangement is preserved, canal size varies between species in ways that reflect behavioural demands. A key determinant of sensitivity is the radius of curvature (R). A larger canal provides a longer path for the endolymph, which increases the effective inertial lag during head acceleration and produces a larger pressure gradient across the cupula. Experimental and comparative work suggests that canal sensitivity scales approximately with the square of the radius (Sensitivity ∝ R^2) [2,3].



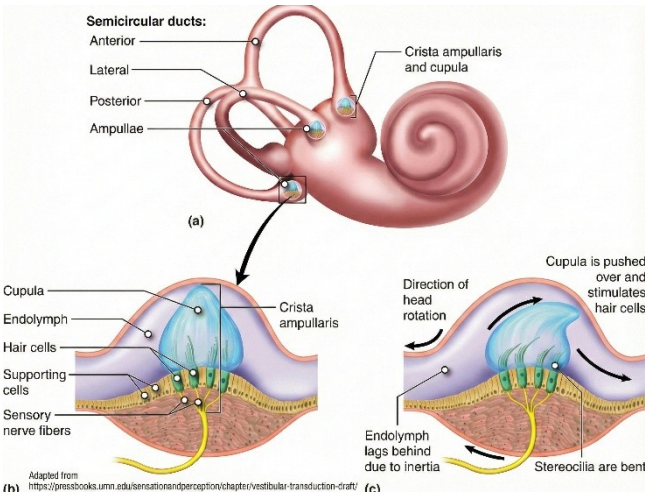
Species that rely on rapid and precise head movements—such as predatory birds and agile mammals—tend to have proportionally larger canals. Functionally, a larger radius improves signal-to-noise characteristics during fast rotations, enhancing gaze stabilisation and motion detection under demanding locomotor conditions.



Understanding canal geometry explains *where* rotation is detected; we now turn to *how* it is converted into a neural signal. Transduction occurs in the ampulla, the widened portion at the end of each canal. Within the ampulla lies the crista ampullaris, a ridge of sensory epithelium containing hair cells. The stereociliary bundles of these hair cells project into the cupula, a gelatinous structure that spans the lumen of the ampulla. When the cupula deflects, the hair bundles deflect with it, and this mechanical displacement alters vestibular afferent firing.

 Clinical Pearl: The Steinhausen model explains why the VOR persists seconds after rotation stops (T1 ≈ 6 s) but responds within milliseconds to onset (T2 ≈ 3 ms). vHIT probes the fast arc; calorics probe the slow time constant. This is why a patient with an abnormal caloric but normal vHIT has impaired low-frequency function with preserved high-frequency canal reserve.

The driving force for cupular deflection is a transient mismatch between skull motion and endolymph motion. At the onset of head rotation, the bony labyrinth moves immediately with the skull, but the endolymph initially lags because of inertia. This lag creates a pressure gradient across the cupula, functionally equivalent to a brief relative flow, which bends the cupula and modulates hair-cell activity. During sustained constant-velocity rotation, the endolymph gradually accelerates to match the canal wall, the pressure gradient dissipates, and the cupula returns toward its resting position. When rotation decelerates or stops, the same sequence occurs in reverse, producing an oppositely directed deflection. Thus, semicircular canals primarily encode changes in rotational velocity rather than steady constant rotation itself.

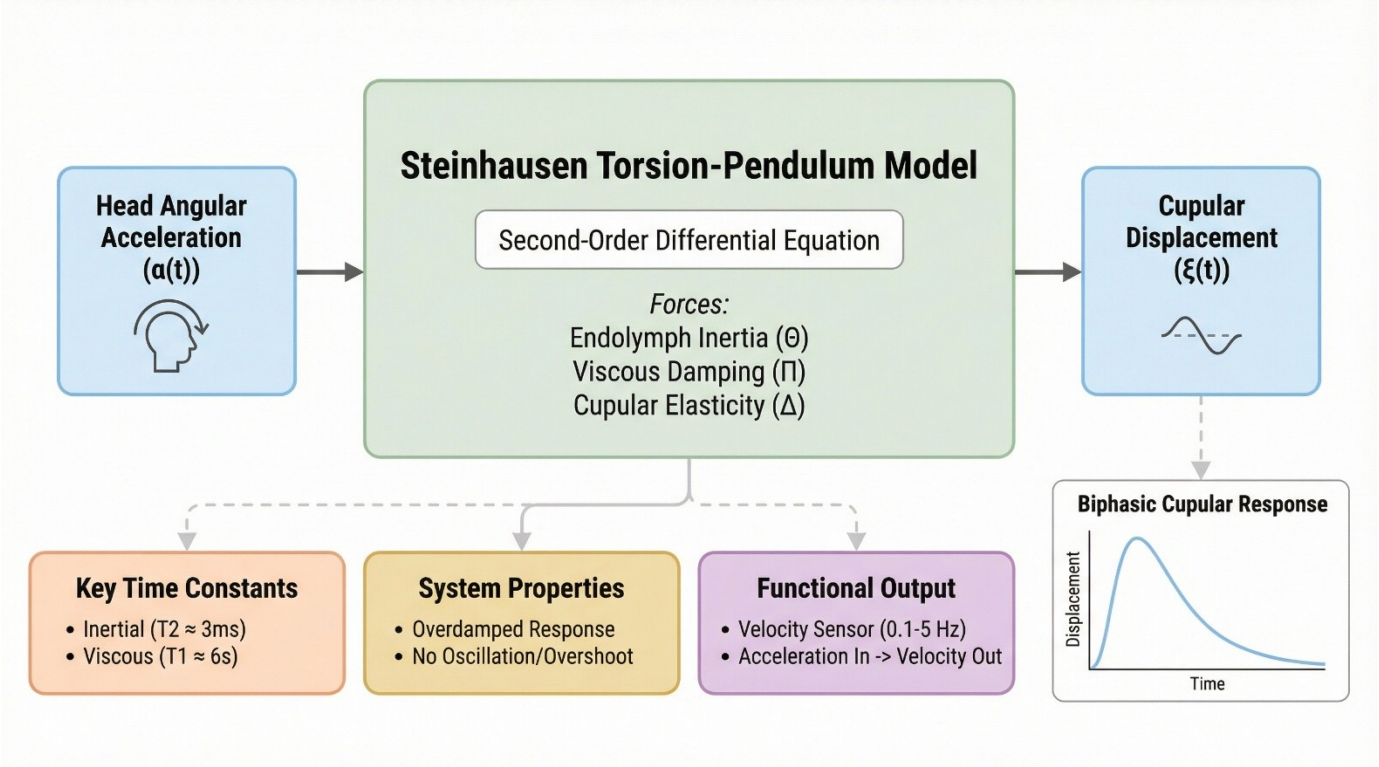


The behaviour described above can be formalised using the **Steinhausen torsion-pendulum model** [4,5]. While the underlying fluid mechanics could, in principle, be derived from the Navier–Stokes equations, the geometry and flow constraints of the semicircular canals allow a simplified description. In this model, cupular displacement ξ(t) follows a second-order differential equation:

$$\Theta\,\ddot{\xi} + \Pi\,\xi + \Delta\,\xi = \alpha(t)\,\Theta$$

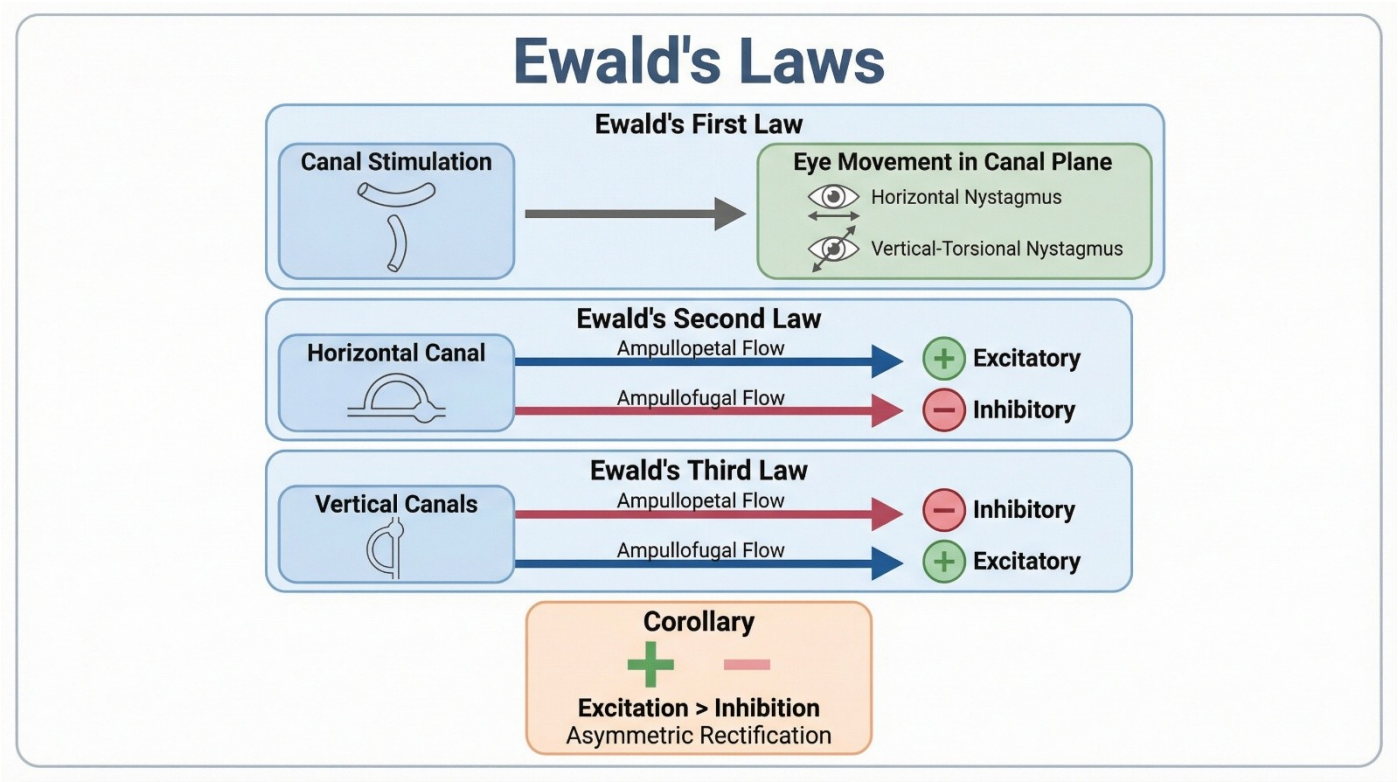
In this expression, Θ represents the effective inertia of the endolymph, Π represents viscous damping due to fluid resistance, Δ represents the elastic restoring force of the cupula, and α(t) is the angular acceleration of the head. Functionally, the equation captures four interacting forces: head acceleration drives the system; endolymph inertia resists rapid change; viscosity dissipates motion; and cupular elasticity restores the system toward equilibrium.

From this formulation arise two key time constants that define canal behaviour. When the head undergoes a step change in velocity, the cupular response is biphasic [6]. The first component is extremely rapid and reflects the immediate inertial lag of the endolymph at rotation onset. This inertial time constant ($T2 = \Theta/\Pi$) is typically on the order of a few milliseconds (≈ 3 ms). The second component is slower and reflects the gradual decay of relative motion as viscous forces and cupular elasticity restore equilibrium. This viscous time constant ($T1 = \Pi/\Delta$) is typically several seconds (≈ 6 s). Together, these two processes explain why the system responds briskly at motion onset but then adapts during sustained rotation.



Importantly, the **canal system is overdamped.** It does not oscillate or overshoot like a freely swinging pendulum. Instead, displacement rises and decays smoothly. This property prevents spurious oscillatory "ringing" that would otherwise distort motion encoding. Over the frequency range most relevant for natural head movements (approximately 0.1–5 Hz), these dynamics make the canals behave functionally like velocity sensors. Although the physical input is angular acceleration, the output within this range approximates head velocity. This explains a fundamental clinical observation: the onset and offset of rotation strongly drive vestibular reflexes and perception, whereas sustained constant-velocity rotation produces a signal that gradually decays over seconds.

The mechanical model explains how the canals generate a signal; **Ewald's Laws** explain how that signal appears at the bedside. They provide the clinical translation of canal physiology, linking the direction of endolymph flow and hair-cell polarisation to the direction and characteristics of nystagmus. In practice, the axis, direction, and intensity of nystagmus can be interpreted as a physiological readout of which canal pair is being biased. [7–8]



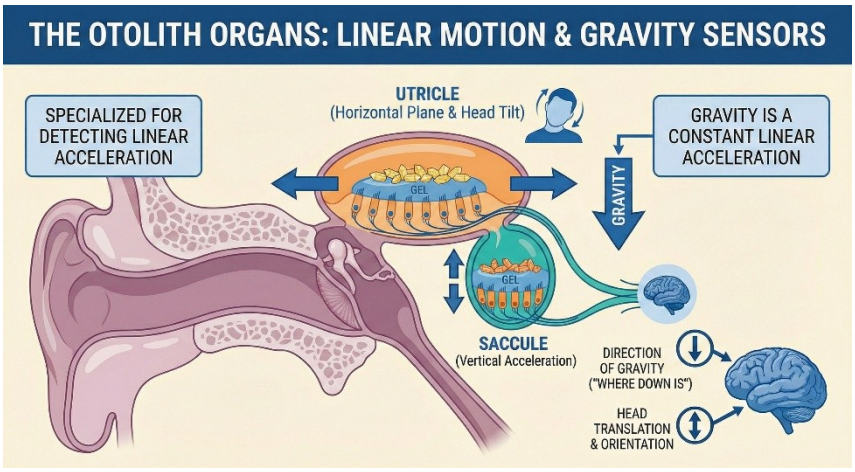
Ewald's First Law states that stimulation of a semicircular canal produces eye movements in the plane of that canal. Clinically, this is why horizontal canal activation produces predominantly horizontal nystagmus, while activation of the vertical canals produces nystagmus with vertical and torsional components aligned to that canal plane.

Ewald's Second Law states that in the horizontal canal, ampullopetal endolymph flow (toward the ampulla) is excitatory, whereas ampullofugal flow is inhibitory.

Ewald's Third Law states that in the vertical canals (anterior and posterior), the polarity is reversed: ampullofugal flow is excitatory and ampullopetal flow is inhibitory. [7–8]

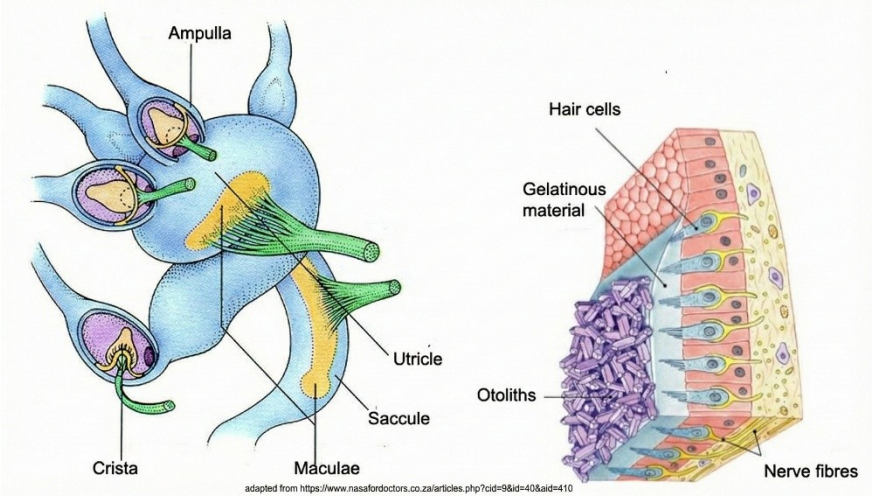
An important clinical corollary is that excitation produces a stronger neural drive than inhibition. Inhibitory deflection can only reduce afferent firing toward zero, whereas excitatory deflection can substantially increase firing above baseline. As a result, the push–pull system operates asymmetrically. This asymmetry explains why acute unilateral vestibular loss generates a strong spontaneous "paretic" nystagmus and why responses often appear more robust when stimulating the intact side compared with the lesioned side. [7–8]

2.2 The Otolith Organs: Linear Acceleration and Gravity



Having described how the semicircular canals encode rotation, we now turn to the complementary system that encodes linear motion and gravity. The otolith organs—the utricle and saccule—are specialised for detecting linear acceleration. Importantly, gravity is simply a constant linear acceleration, so these organs provide the brain with

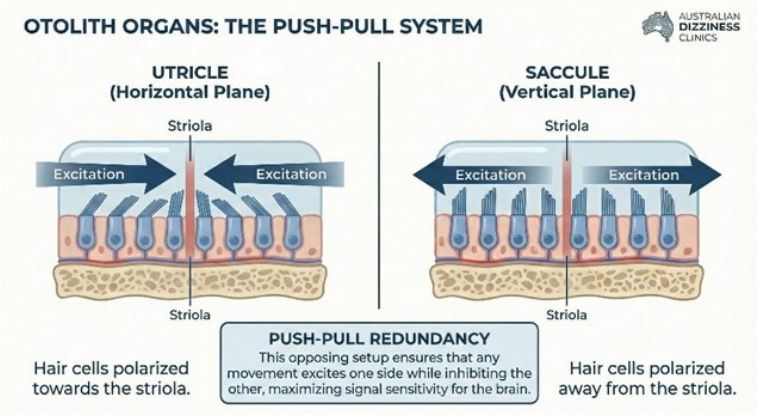
information about both head translation and head orientation relative to gravity. Functionally, the utricle is most sensitive to accelerations in the horizontal plane and to head tilt, whereas the saccule is oriented more toward vertical accelerations. Together, they continuously inform the brain about two essential variables: the direction of gravity (“where down is”) and whether the head is undergoing linear movement.



The otolith organs use a different mechanical strategy from the canals. Instead of fluid inertia within a curved duct, they rely on a mass-loaded membrane that can shear relative to the sensory epithelium. The sensory surface is called the macula. Above it lies the otolithic membrane, which contains dense calcium carbonate crystals known as otoconia. Because otoconia are much denser than the surrounding endolymph (specific gravity ≈ 2.71), they provide the inertial mass necessary to detect linear acceleration [9]. When the head accelerates, the macula moves with the skull, but the heavier otoconia lag slightly. This relative displacement produces shear within the otolithic membrane, bending the hair bundles embedded in it and thereby modulating vestibular afferent firing.

This mechanism can be visualised using a simple model: a weighted layer resting on a sensitive surface. During linear translation, the surface (macula) moves first, while the heavier otoconia lag behind. The resulting shear between the two bends the hair bundles and alters afferent firing. During sustained head tilt, gravity continuously pulls the otoconia sideways relative to the macula, producing a maintained deflection. This explains how the otoliths can **encode both transient linear acceleration and sustained orientation** relative to gravity.

 Clinical Pearl: The otolith organs cannot distinguish head tilt from linear translation — they only sense the resultant gravito-inertial force. The brain resolves this ambiguity using canal and visual cues. Clinically: loss of utricular function causes subjective visual vertical (SVV) tilt >2.5° and may produce an ocular tilt reaction (skew deviation + head tilt + ocular torsion). SVV is a bedside proxy for utricular pathway integrity.

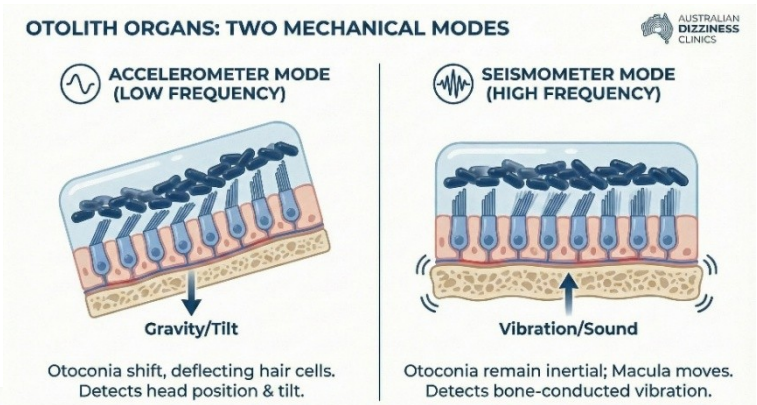


However, the macula is not mechanically or neurally uniform. Its organisation is centred on the striola, a specialised zone that divides the epithelium into regions with opposite hair-cell polarity. On either side of the striola, hair cells are oriented in opposing directions: in the utricle, kinocilia are oriented toward the striola, whereas in the saccule they are oriented away from it. This creates a **Line of Polarity Reversal (LPR)**, establishing an internal push–pull system within each organ. For a given acceleration vector, one group of hair cells increases firing while the opposing group decreases firing. This differential encoding enhances sensitivity, broadens dynamic range, and improves robustness against noise and baseline drift.

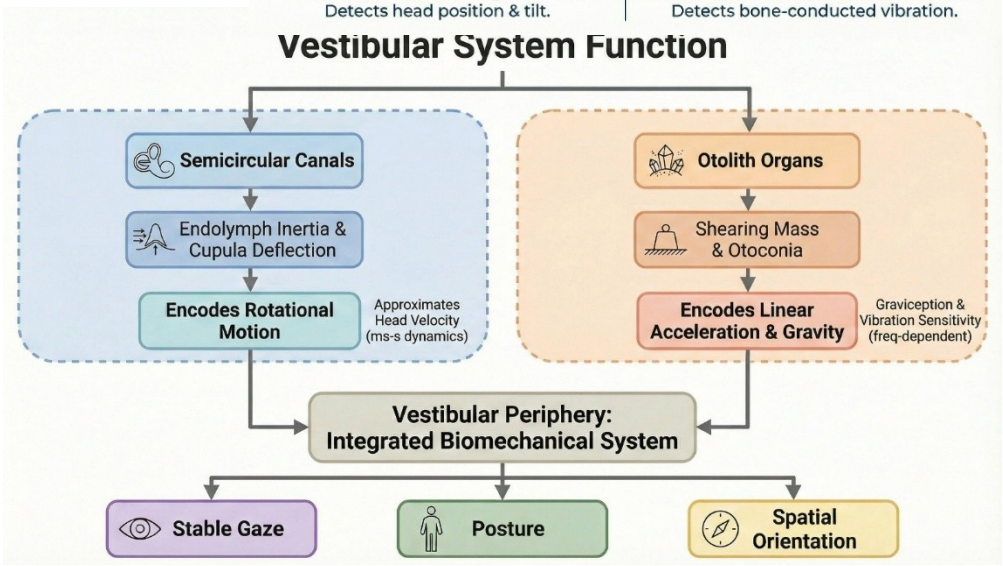
In addition to polarity organisation, recent biomechanical studies suggest that the three-dimensional curvature of the otolithic membrane affects how forces are distributed across the macula [10]. The macula is not a flat, uniformly moving surface. Variations in membrane curvature and material properties can lead to

region-specific differences in displacement amplitude for the same head movement. This implies local variations in mechanical gain and provides a structural basis for functional differences between striolar and extrastriolar regions.

These structural features give rise to frequency-dependent modes of otolith function [11]. At **low frequencies**—such as sustained tilt or slow translation—the otoconia shift relative to the macula, generating shear that is well suited for encoding head orientation relative to gravity and low-frequency linear acceleration. This behaviour is often described as an **"accelerometer mode,"** and it is strongly associated with extrastriolar regions.



At **higher frequencies**—above several tens of hertz, with responses extending into the range relevant for vibration and sound—the mechanics differ. Under these conditions, the otoconia act as a relatively inertial mass that does not fully track rapid skull micro-movements. The macula moves with the skull while the otoconia lag, producing relative motion that resembles a **"seismometer-like"** mode. This behaviour aligns with evidence that otolith organs can respond to bone-conducted vibration and sound, with striolar specialisation likely contributing to these high-frequency responses.



Taken together, the semicircular canals and otolith organs operate as complementary inertial sensors built on distinct physical principles. The canals use endolymph inertia within a curved duct and the elastic properties of the cupula to encode rotational motion, producing dynamics that span milliseconds to seconds and approximate head velocity over physiological frequencies. The otoliths use a dense, shearing mass to encode linear acceleration and gravity, incorporating internal polarity reversal and frequency-dependent mechanical behaviour to support both graviception and vibration sensitivity. Viewed in this way, the vestibular periphery functions as an integrated biomechanical system optimised for stable gaze, posture, and spatial orientation.

Key Facts: Peripheral Sensors

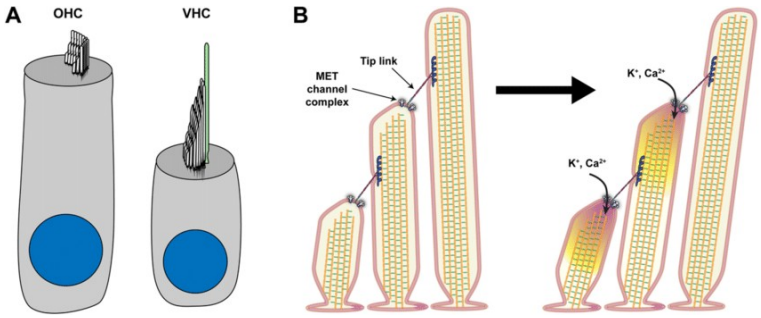
Feature	Semicircular Canals	Otolith Organs
Stimulus	Angular acceleration	Linear acceleration + gravity
Sensor structure	Cupula (gelatinous)	Otolithic membrane + otoconia
Frequency range	0.1–5 Hz (velocity sensor)	DC to ~100 Hz (freq-dependent)
Time constants	T1 ≈ 6 s; T2 ≈ 3 ms	Ongoing (sustained tilt) + transient
Clinical test	vHIT / calorics	VEMP / SVV

Feature	Semicircular Canals	Otolith Organs
Coding principle	Push–pull bilateral pairs	Internal LPR (striola polarity reversal)

3.3. Molecular Physiology of Hair Cell Transduction

Having described the mechanical behaviour of the canals and otoliths, we now move to the molecular interface where force becomes current. Vestibular hair cells convert mechanical displacement into electrical signals with extremely short latency. The **critical event occurs at the tips of the stereocilia**, where mechanically gated ion channels open within less than approximately 10 microseconds. This speed effectively rules out multi-step second-messenger pathways. Instead, channel gating is direct and mechanically coupled: displacement of the hair bundle transmits force directly to the channel complex, and channel opening follows almost immediately.

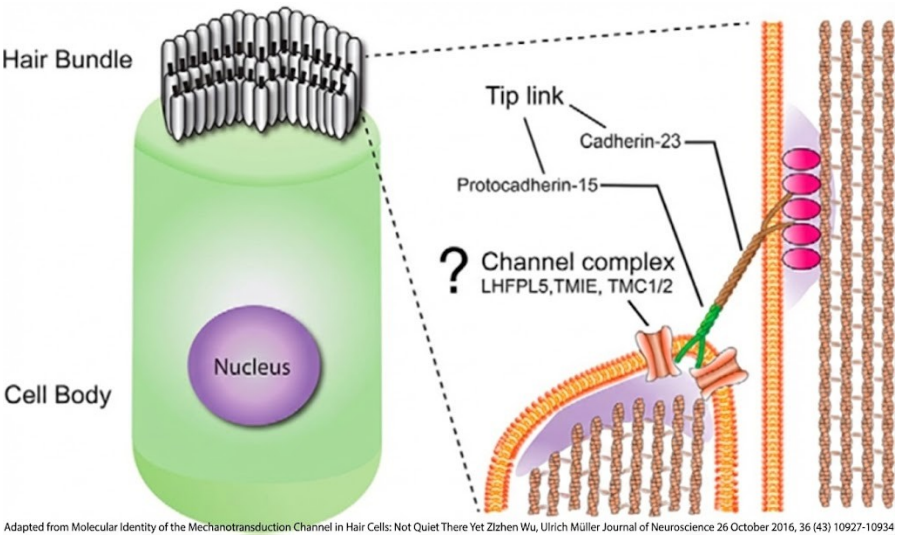
Structurally, the hair bundle resembles a staircase. Stereocilia are arranged in graded heights and are linked by fine extracellular filaments known as tip links. When the bundle deflects toward the tallest stereocilia, tension on the tip links increases, raising the probability that mechanotransduction (MET) channels open. When deflection occurs in the opposite direction, tension decreases, channels close, and current falls. This bidirectional "tension-in/tension-out" mechanism allows mechanical displacement to be translated into graded changes in ionic current, forming the core of vestibular transduction.



An important physiological context is the ionic environment in which this occurs. Vestibular hair cells are positioned between two distinct extracellular fluids: potassium-rich endolymph bathing the stereocilia and sodium-rich perilymph surrounding the basolateral membrane. When MET channels open, the resulting depolarising current is carried predominantly by K^+ (with a smaller Ca^{2+} component) entering the hair cell from the endolymph. The ionic composition of endolymph is maintained by specialised epithelial transport systems, including vestibular dark cells. Unlike the cochlea, the vestibular labyrinth does not generate a large positive endocochlear potential. Consequently, the driving force for the MET current depends primarily on the hair cell's resting membrane potential and the comparatively modest vestibular endolymph potential.

3.1 The Mechanotransduction Channel Complex: TMC1 and TMC2

Having established how mechanical force gates ion flow, we now examine the molecular identity of the channel responsible for that current. The mechanotransduction (MET) channel is not a single protein but a multi-protein complex. The pore-forming subunits are Transmembrane Channel-like proteins, primarily TMC1 and TMC2. Structural and functional data support the view that TMC subunits assemble as dimers within the complex [12]. Genetic evidence provides strong confirmation: deletion of both Tmc1 and Tmc2 abolishes mechanosensitivity in vestibular hair cells, meaning hair-bundle deflection no longer generates MET current. Additional support comes from point mutations that alter ion permeation and single-channel conductance. The Beethoven mutation, for example, changes channel properties in a manner consistent with TMC proteins forming the ion-conducting pore [13–14]A key distinction between vestibular and cochlear hair cells emerges at this level. In the cochlea, TMC2 expression is largely developmental and declines with maturation, leaving TMC1 as the dominant subunit in adult auditory hair cells.



Adapted from Molecular Identity of the Mechanotransduction Channel in Hair Cells: Not Quiet There Yet Zizhen Wu, Ulrich Müller Journal of Neuroscience 26 October 2016, 36 (43) 10927-10934

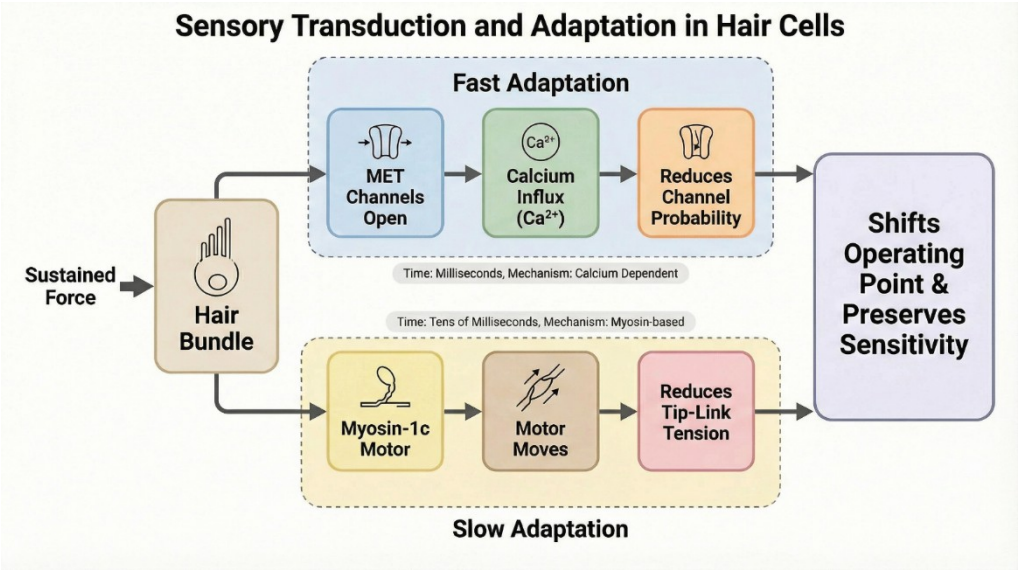
In contrast, vestibular hair cells maintain expression of both TMC1 and TMC2 into adulthood. This co-expression appears functionally meaningful rather than redundant. Channels containing TMC2 tend to exhibit higher single-channel conductance and greater calcium permeability, whereas TMC1-containing channels show lower conductance and are associated with structural stability and long-term hair-cell viability. The coexistence of both subunits likely broadens the dynamic operating range of vestibular transduction. The vestibular periphery must remain sensitive to subtle everyday head movements while also tolerating large accelerations during rapid locomotion or trauma. Co-expression of TMC1 and TMC2 provides a plausible molecular mechanism for balancing high sensitivity with resistance to saturation.

3.2 Adaptation Mechanisms

Having identified the MET channel, the next question is how hair cells remain sensitive in the presence of sustained forces. If MET channels simply opened in proportion to bundle displacement and stayed open during prolonged deflection, the system would quickly saturate. Gravity provides the clearest example: it is constant, yet the vestibular system must remain responsive to small, dynamic head movements superimposed on this steady load. Hair cells solve this problem through adaptation, which shifts the operating point of the hair bundle so that sensitivity to change is preserved even during static or prolonged stimuli.

Two forms of adaptation are commonly described, differing in time course and molecular mechanism [15,16]. **Fast adaptation** occurs within milliseconds and is **strongly calcium dependent.** When MET channels open, calcium enters the cell through the channel pore. This influx acts as a rapid feedback signal that reduces channel open probability, either by directly affecting the channel complex or associated membrane elements. In effect, channel opening promotes its own partial closure. This fast negative feedback loop enhances sensitivity to changes in force rather than to steady displacement.

Slow adaptation occurs over tens of milliseconds and is thought to involve a **myosin-based motor mechanism**, particularly Myosin-1c. The upper end of each tip link is anchored to the actin core of the stereocilium via this motor complex. During sustained deflection, the motor can move along the actin filament, reducing tip-link tension and resetting the likelihood of channel opening even while the bundle remains displaced. Functionally, this mechanism prevents prolonged forces from locking the system into saturation. It allows the hair cell to maintain responsiveness to small fluctuations superimposed on larger baseline forces, such as those encountered during upright posture under constant gravitational load.



3.3 Potassium Conductances: Type I versus Type II Hair Cells

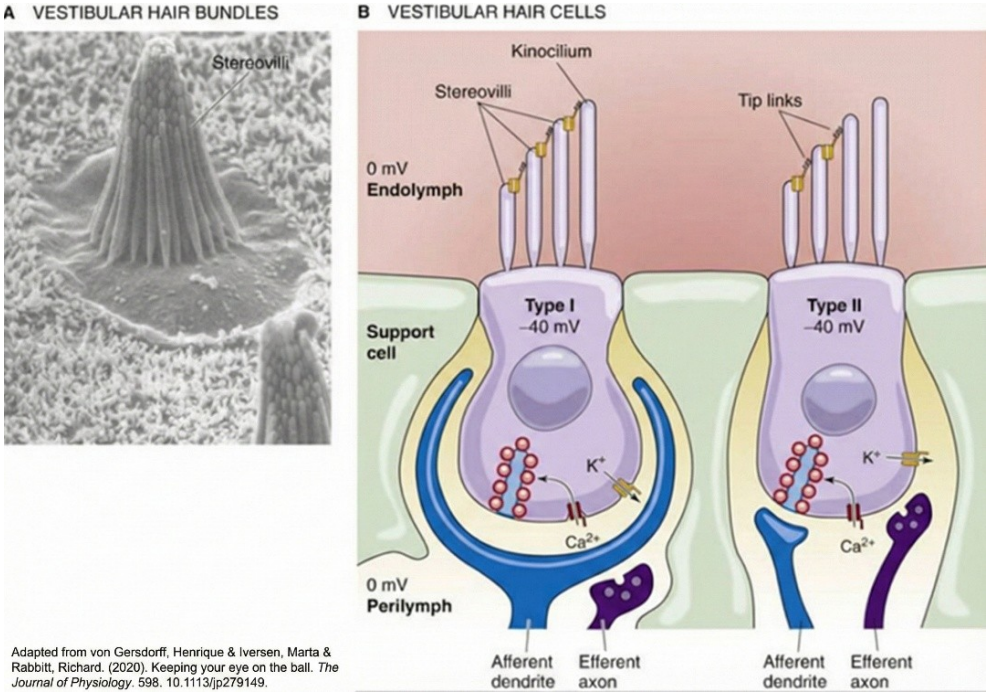
After mechanotransduction and adaptation shape the receptor potential, the next level of processing occurs at the basolateral membrane. Here, potassium conductances determine how the voltage signal is filtered, stabilised, and transmitted to afferent fibres. In amniotes (vertebrates whose embryos develop within an amniotic sac, beginning evolutionarily with reptiles and including birds and mammals), two distinct hair-cell phenotypes are present—Type I and Type II—distinguished by both their synaptic architecture and their complement of ion channels [17]. These phenotypes represent different electrical strategies for encoding motion rather than minor anatomical variants.

 Clinical Pearl: Type I hair cells with calyx endings drive irregular high-gain afferents — probed by vHIT (high frequency). Type II hair cells with bouton endings drive regular low-gain afferents — probed by calorics and rotation chair (low frequency). Selective loss of calyx afferents (e.g., early gentamicin toxicity, superior vestibular neuritis) can give abnormal vHIT with relatively preserved calorics.

Type II hair cells resemble the more ancestral configuration. They are contacted by multiple bouton-type afferent endings and express several well-characterised potassium currents. These include a delayed rectifier potassium current (IK,V) and a fast-inactivating A-type current (IA), both of which contribute to membrane repolarisation and regulate response timing during repetitive stimulation [18]. In addition, Type II cells express Kv7.4 (KCNQ4) channels that generate the M-current (IM), a non-inactivating potassium conductance that stabilises the resting membrane potential and modulates excitability [19,20]. Functionally, these conductances prevent excessive depolarisation, limit spike distortion, and support steady, graded signalling. In this sense, Type II hair cells behave as relatively conventional sensory receptors, prioritising stability and reliable transmission during ongoing stimulation.

In contrast to Type II cells, Type I hair cells represent a more specialised amniote adaptation. Each Type I hair cell is almost completely enveloped by a single large calyx afferent terminal, creating an unusually intimate pre- and postsynaptic arrangement. Their defining electrophysiological feature is a large low-voltage-activated potassium conductance, known as **IK,L**. This current activates at **very negative membrane potentials (as low as approximately −80 mV),** resulting in a markedly low input resistance (around 10 MΩ compared with roughly 1 GΩ in Type II cells) and a relatively hyperpolarised resting membrane potential [21]. Molecular studies identify Kv1.8 (encoded by Kcna10) as a key pore-forming subunit underlying IK,L [21]. When Kv1.8 is absent, IK,L is lost and the membrane reverts toward a higher-resistance, more Type II-like electrical profile.

The functional consequence of IK,L is a change in temporal filtering. A low input resistance shortens the membrane time constant, meaning the membrane potential can follow rapid synaptic or mechanical inputs with minimal temporal smearing. In contrast, a high-resistance membrane behaves like a larger capacitor, blurring fast fluctuations. Type I hair cells are therefore better suited to tracking rapid mechanical changes with high temporal precision. This electrical tuning aligns with the demands of high-speed vestibular reflexes, particularly the vestibulo-ocular reflex during locomotion, where even small delays can degrade gaze stability.



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

To consolidate the distinctions described above, the key structural and physiological differences between Type I and Type II vestibular hair cells are summarised below. This contrast is clinically useful because these phenotypes underpin differences in afferent firing patterns and temporal encoding.

Feature	Type I Hair Cells	Type II Hair Cells
Evolutionary context	Specialised amniote innovation	More ancestral hair-cell phenotype
Afferent contact	Single large calyx terminal	Multiple bouton-type afferent terminals
Synaptic architecture	Tight calyx enclosure; mixed chemical and non-quantal transmission possible	Conventional bouton synapses; primarily quantal chemical transmission
Dominant K^+ conductance	IK,L (low-voltage-activated K^+ current)	IK,V (delayed rectifier), IA (A-type), and IM (M-current via KCNQ4)
Molecular correlate	Kv1.8 (Kcna10) critical for IK,L	Kv7.4 (KCNQ4) contributes to IM
Activation range	Activates at very negative potentials (≈ −80 mV)	Activate closer to depolarised voltages
Input resistance	Low (~10 MΩ)	High (~1 GΩ)
Membrane time constant	Short (rapid voltage tracking)	Longer (greater temporal smoothing)
Resting membrane potential	More hyperpolarised	Less hyperpolarised
Temporal handling	Optimised for high-frequency, rapid signals	Optimised for stable, graded signalling
Functional emphasis	High-speed reflex encoding (e.g., VOR precision)	Stable, sustained encoding during ongoing stimulation

In summary, Type I hair cells are electrically tuned for speed and temporal precision, while Type II hair cells prioritise stability and graded responsiveness. Together, they provide complementary encoding strategies within the vestibular periphery.

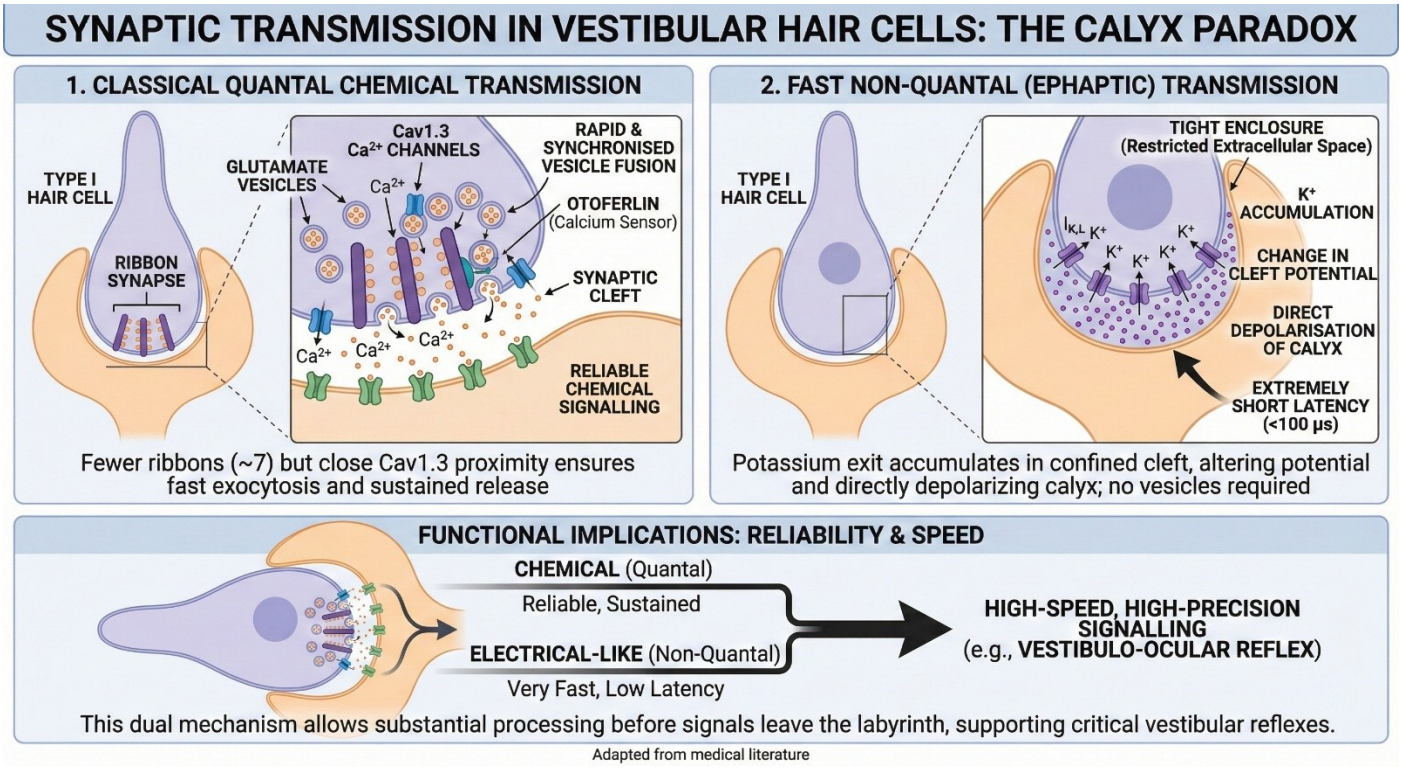
3.4 Synaptic Transmission: The Calyx Paradox

After understanding how Type I hair cells shape their electrical signal, the next step is how that signal reaches the nerve. The **Type I** hair cell–calyx synapse is unusual because it uses **two parallel transmission** mechanisms. One is conventional chemical transmission, and the other is a very fast, electrical-like

mechanism. This design allows the system to maintain both reliability and speed—an important requirement for reflexes such as the vestibulo-ocular reflex.

The **first mechanism is classical quantal transmission.** Type I hair cells release glutamate at ribbon synapses. Although they have fewer ribbons than cochlear inner hair cells (approximately 7 per cell compared with about 17 in cochlear cells), their Cav1.3 calcium channels are positioned very close to the release sites, allowing rapid and synchronised vesicle fusion [23]. Otoferlin acts as the calcium sensor that enables fast exocytosis and replenishment of vesicles, so transmitter release can continue during sustained stimulation [24,25]. This pathway provides reliable chemical signalling.

The **second mechanism is non-quantal transmission,** which depends on the tight enclosure formed by the calyx terminal. Because the calyx almost completely surrounds the Type I hair cell, the synaptic cleft becomes a restricted extracellular space rather than an open gap. When potassium exits the hair cell—particularly through IK,L—it can accumulate in this confined cleft. The rise in local potassium concentration changes the electrical potential of the cleft, and this directly depolarises the calyx terminal. This process does not require vesicle release or receptor activation, so it operates with extremely short latency, reported to be less than 100 microseconds [26,27]. In effect, the synapse behaves partly like a chemical synapse and partly like an electrical connection.



Adapted from medical literature

Overall, this arrangement shows that substantial processing occurs even before signals leave the labyrinth. Hair cells convert force to current rapidly, adjust sensitivity through adaptation, and shape voltage signals through specialised potassium conductances. The calyx then ensures that information is transmitted both reliably and with minimal delay. This combination supports the high-speed, high-precision signalling required for vestibular reflexes.

Key Ion Channels in the Vestibular Labyrinth

Channel / Current	Location	Primary Functional Role
TMC1 / TMC2	Hair Bundle Tips	Pore-forming subunits of the MET channel; convert force to voltage.
IK,L (Kv1.8)	Type I Hair Cells	Massive low-voltage activated K+ conductance; creates low input resistance.
IK,V (Delayed Rectifier)	Type II Hair Cells	Standard repolarization after depolarization; maintains excitability.
M-current (KCNQ2-4)	Type II & Afferents	Stabilizes resting potential; regulates discharge regularity

		in afferents.
Nav1.6 (Resurgent Na+)	Calyx Afferents / VN	Enables extremely high firing rates by reopening during repolarization.
Ih (HCN Channels)	Vestibular Nuclei	Mediates post-inhibitory rebound; amplifies "push-pull" signals.
ICAN (TRPC)	Neural Integrator	Calcium-activated cation current; supports persistent firing/memory.

3.6 The Efferent Vestibular System: Central Control of Peripheral Gain

So far, vestibular signalling has been described as a flow of information from the labyrinth to the brainstem. However, this pathway is not strictly one-way. The vestibular periphery is also subject to direct central modulation through the efferent vestibular system. **Cholinergic efferent fibres,** arising from the efferent vestibular nucleus, project to **Type II hair cells** and to afferent terminals. Their effects are modulatory rather than purely excitatory or inhibitory.

At the level of the hair cell, acetylcholine can produce hyperpolarisation. This typically occurs via α9/α10 nicotinic receptors that activate SK potassium channels, reducing the amplitude of the receptor potential and effectively lowering transduction gain. At the level of the afferent terminal, efferent activation can increase baseline firing rate—particularly in irregular afferents—while simultaneously reducing sensitivity to rotational input. This combination may seem counterintuitive, but it likely serves to optimise dynamic range and signal-to-noise characteristics rather than simply increasing or decreasing output [89,90].

Physiologically, the efferent system provides a mechanism for context-dependent control. During locomotion, heightened arousal, or active head movements accompanied by motor commands, central signals can adjust peripheral responsiveness before sensory input reaches the vestibular nuclei. This has clinical implications. Peripheral vestibular tests performed under passive, quiet conditions may not fully reflect vestibular performance during active movement, where efferent modulation may alter gain and responsiveness [90].

□ Key Facts: Hair Cell Mechanotransduction

Property	Detail
MET channel	TMC1/TMC2 heteromer; tip-link gated
Gating speed	Under 10 μs (mechanically direct)
Ionic current	K^+ (major) + Ca^{2+} (minor) via endolymph
Adaptation	Fast (under 1 ms) + slow (tens of ms) via motor proteins
Type I hair cell	Calyx synapse; irregular high-gain afferents; striolar
Type II hair cell	Bouton synapse; regular low-gain afferents; extrastriolar

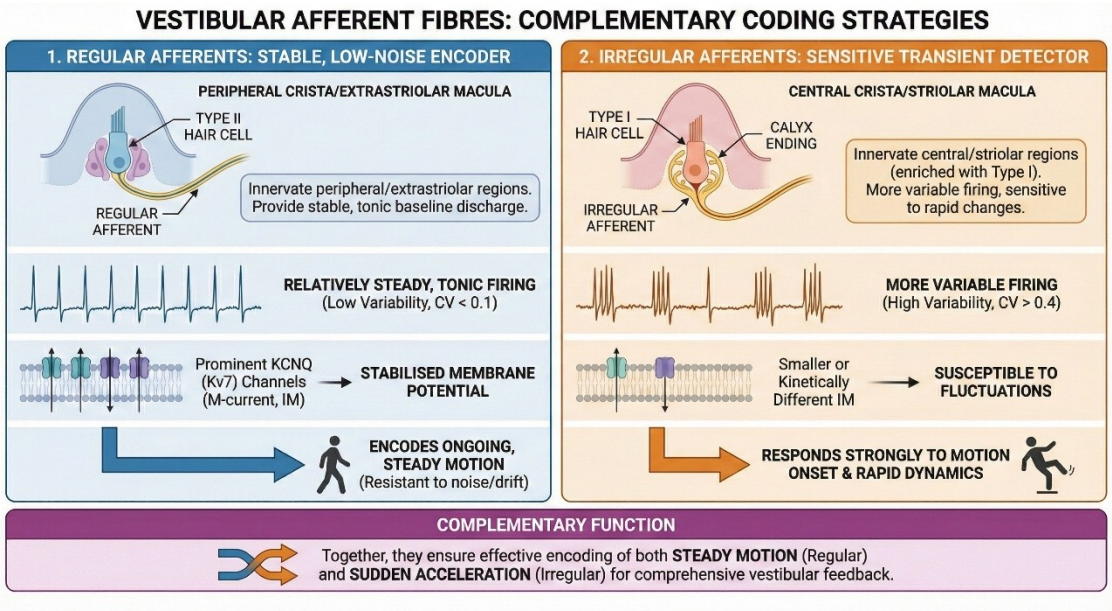
4. Afferent Encoding and Frequency Tuning

After synaptic transmission at the hair cell, vestibular information enters the nerve. Importantly, this output is not uniform. Afferent fibres arising from Scarpa's ganglion can be divided into **two major functional groups: regular and irregular afferents**. The distinction refers to the variability of their spontaneous firing, but it also reflects two complementary strategies for encoding head motion.

□ **Clinical Pearl:** vHIT and calorics can dissociate because they probe different afferent populations. A normal vHIT with abnormal calorics reflects preserved high-frequency irregular afferents but impaired low-frequency regular

afferents — typical of partial peripheral hypofunction. An abnormal vHIT with relatively normal calorics (rarer) suggests loss of high-frequency canal function with residual low-frequency response. Both findings are clinically meaningful; neither alone is sufficient.

Regular afferents primarily innervate the peripheral zones of the cristae and the extrastriolar regions of the maculae, areas more closely associated with Type II hair cells. They fire in a steady, tonic pattern, with low variability between spikes (coefficient of variation typically < 0.1). This regularity produces a stable baseline discharge that changes smoothly with stimulation. As a result, regular afferents are well suited to transmitting continuous, lower-frequency head-velocity information in a manner that is resistant to noise and drift.



Irregular afferents preferentially innervate the central crista and the striolar regions of the macula, where Type I hair cells and calyx endings predominate. Their firing pattern is more variable (coefficient of variation often > 0.4), and they respond more strongly to motion onset and rapid dynamic changes than to steady-state conditions. Intrinsic membrane conductances contribute significantly to this difference. In particular, KCNQ (Kv7) potassium channels generate the M-current (IM), a stabilising, non-inactivating potassium current. When IM is prominent, spike timing becomes more regular and stable. When IM is reduced or kinetically different, voltage fluctuations increase and firing becomes more irregular. Thus, membrane properties help determine whether an afferent behaves as a steady-state reporter or as a transient detector.

These differences also appear clearly when examined in the frequency domain. Regular afferents show relatively flat gain across physiological frequencies and maintain close phase alignment with head velocity. This makes them reliable encoders of slower and more predictable movements, supporting stable gaze and posture during everyday motion. Irregular afferents, in contrast, show greater gain at higher frequencies and may exhibit phase lead relative to head velocity. Functionally, this emphasises rapid changes in motion and provides early warning of fast, unpredictable head movements.

The practical implication is that vestibular afferents are frequency-tuned. Some channels preferentially encode steady, lower-frequency motion, while others emphasise rapid, higher-frequency changes. This helps explain why different clinical tests can produce different results in the same patient. Caloric testing and rotational chair primarily probe lower-frequency behaviour, whereas vHIT probes high-frequency, short-latency dynamics. Because these tests engage different regions of the vestibular frequency spectrum—and therefore weight different afferent populations—they may not always agree.

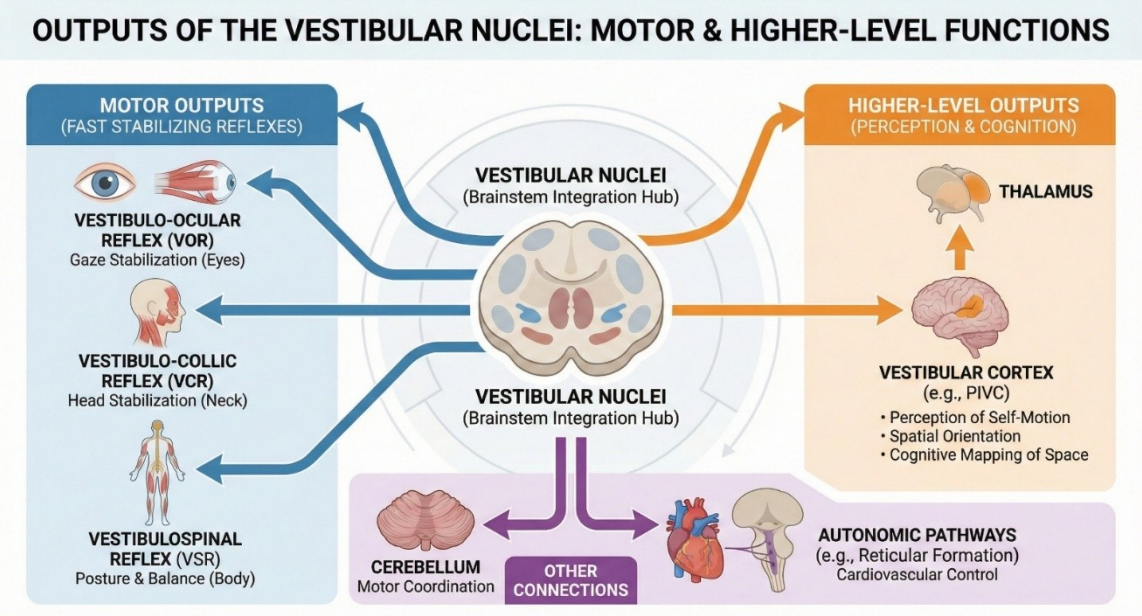
□ Key Facts: Afferent Fibre Classes

Property	Regular Afferents	Irregular Afferents
Coefficient of variation	Below 0.1	Above 0.4

Property	Regular Afferents	Irregular Afferents
Zone innervated	Peripheral crista / extrastriolar	Central crista / striolar
Hair cell type	Predominantly Type II	Predominantly Type I (calyx)
Frequency preference	Low (DC–1 Hz)	High (1–10+ Hz)
Gain dynamics	Flat; phase-aligned with velocity	Increasing with frequency; phase lead
Clinical probe	Calorics / rotation chair	vHIT

5. Central Vestibular Nuclei Architecture

After afferent encoding in the nerve, the signal reaches the vestibular nuclei (VN) in the dorsal brainstem. This is the point at which vestibular information stops being a relatively raw sensory readout and becomes organised into functional commands. Input from **Scarpa's ganglion carries head-motion information encoded in regular and irregular firing patterns.** The VN acts as the first major processing hub that transforms this input into multiple output streams serving different purposes.



Some of these **outputs** are **motor**, driving fast stabilising reflexes of the eyes (vestibulo-ocular reflex), head and neck (vestibulo-collic reflex), and body (vestibulospinal reflex). Others influence **autonomic pathways,** including cardiovascular control.

Additional projections ascend toward the **thalamus and cortex,** contributing to perception of self-motion, spatial orientation, and cognitive mapping of space. Importantly, the VN is also the **first site of strong multisensory integration.** Neurons here receive convergent input from visual pathways, somatosensory systems (especially neck proprioception), and the cerebellum. This arrangement allows the brainstem to compare vestibular signals with visual and proprioceptive information in real time and to adjust output gain when signals are concordant or conflicting [35].

5.1 Functional Classification of Neurons

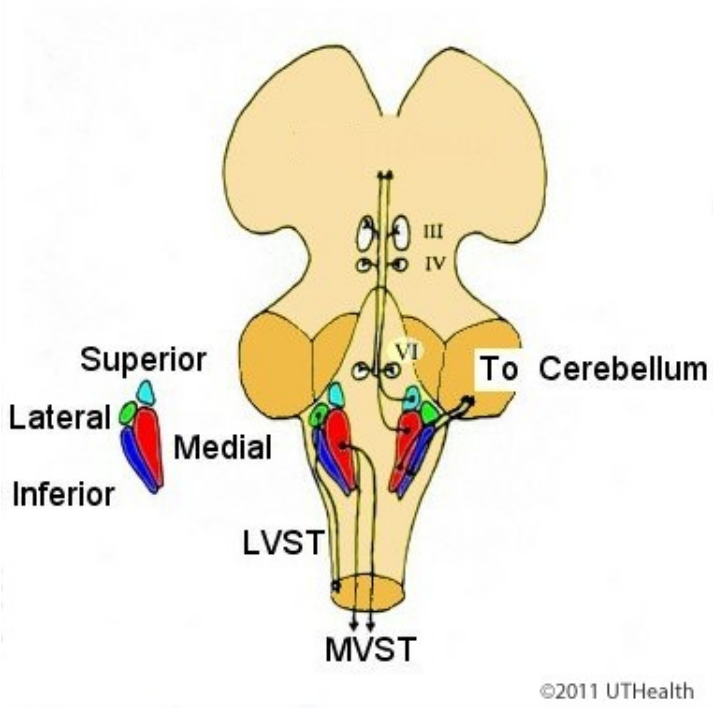
Although the vestibular nuclei are anatomically divided into **superior, medial, lateral, and descending** subdivisions, clinical understanding improves when neurons are classified functionally—by what they encode and where they project—rather than purely by anatomical location. From a systems perspective, what matters is the computation a neuron performs and the circuit it influences.

 Clinical Pearl: Gaze-evoked nystagmus (GEN) — drift of the eyes from eccentric gaze back to centre, followed

by a corrective saccade — is the bedside sign of gaze-holding integrator failure. It is physiologically distinct from velocity storage dysfunction (post-rotatory nystagmus, direction-changing nystagmus). GEN localises to the vestibular nucleus/cerebellum circuit and is exacerbated by anticonvulsants and alcohol.

One particularly important distinction is between **velocity integration and position integration.** These terms are often confused, yet they refer to different physiological processes. **Velocity storage** is a brainstem–cerebellar mechanism that prolongs canal-driven velocity signals beyond the short mechanical time constant of the labyrinth. It effectively keeps an estimate of rotation active for longer than peripheral mechanics alone would allow. In contrast, the gaze-holding neural integrator is part of the oculomotor system. It integrates eye velocity commands into sustained eye position commands, allowing the eyes to maintain eccentric gaze against the elastic restoring forces of the orbit [7,36–37].

If the **gaze-holding integrator** fails, the eye behaves like a leaky spring: after a saccade or vestibular slow phase, eye position drifts back toward centre, producing gaze-evoked nystagmus and difficulty maintaining fixation. This phenomenon is physiologically distinct from abnormalities of velocity storage. A patient may have an intact three-neuron VOR arc but impaired gaze holding due to a defective position integrator. Conversely, a patient may show abnormal velocity storage dynamics with relatively preserved gaze holding. Recognising this distinction is essential for accurate clinical localisation [7,36–37].

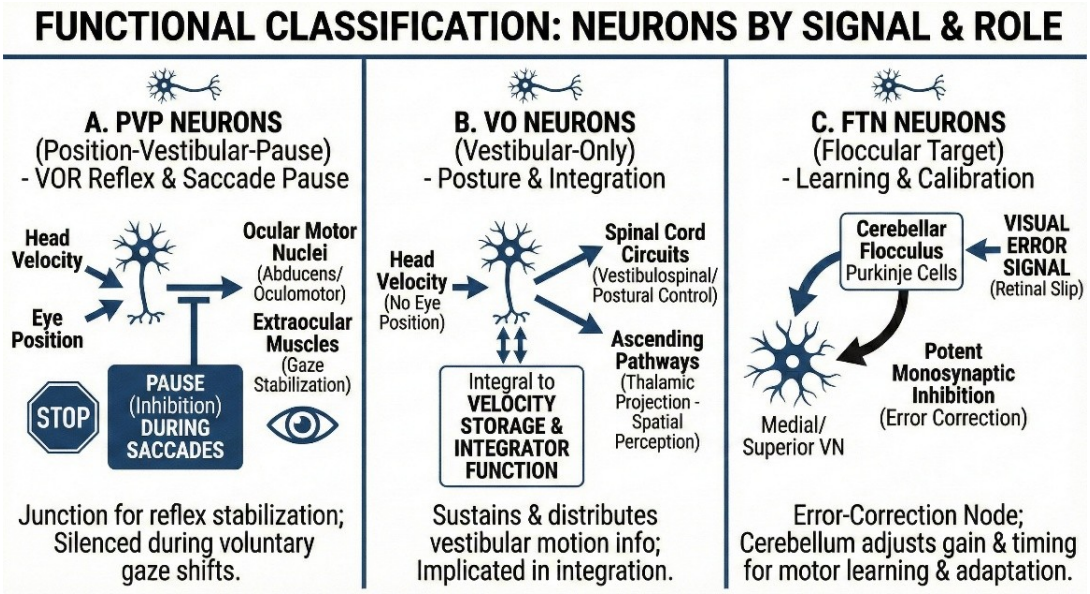


Building on the distinction between velocity and position integration, specific neuron classes within the vestibular nuclei can now be understood in terms of their circuit roles.

Position-Vestibular-Pause (PVP) neurons are the principal premotor interneurons of the vestibulo-ocular reflex (VOR). They encode head-velocity signals that are transformed into compensatory eye movement commands. These neurons **project directly to ocular motor nuclei,** including the abducens and oculomotor nuclei, which in turn activate the extraocular muscles to stabilise gaze during head motion [39]. The “pause” component of their name is physiologically important. During saccades—voluntary rapid eye movements—the stabilising reflex must be temporarily suppressed so that the eye can move freely to a new target. PVP neurons are actively inhibited during saccades, creating a pause in vestibular-driven output. This prevents the VOR from opposing the intended eye movement. Functionally, PVP neurons sit at the interface between reflex stabilisation and voluntary gaze control, permitting stabilisation during head motion but silencing it during intentional gaze shifts.

Vestibular-Only (VO) neurons also encode head velocity, but they do not carry eye position signals and do not pause during saccades. This suggests a different functional role. Rather than directly commanding extraocular motor neurons, VO neurons **participate in maintaining and distributing vestibular motion information across central networks.** They are strongly implicated in the **velocity storage mechanism**, whereby the central vestibular system extends the effective duration of canal signals beyond peripheral mechanical limits. Their projections are consistent with this broader role: they influence vestibulospinal pathways for postural control and ascend toward thalamic and cortical circuits that contribute to motion perception and spatial orientation. Unlike PVP neurons, they are not primarily dedicated to immediate eye movement execution.

Floccular Target Neurons (FTNs) highlight a further layer of organisation within the vestibular nuclei: **learning and calibration.** Located particularly in the medial and superior vestibular nuclei, FTNs receive powerful monosynaptic inhibition from **Purkinje cells of the cerebellar flocculus**. This circuitry provides a mechanism for error-driven adaptation. When retinal slip occurs—indicating imperfect gaze stabilisation—the cerebellum interprets this as an error signal and modifies vestibular output accordingly. Through floccular influence on FTNs, the gain and timing of the VOR can be recalibrated so that future head movements generate more accurate compensatory eye movements. In this sense, FTNs function as an error-correction interface, linking cerebellar learning signals to brainstem vestibular reflex circuits and supporting long-term adaptation of vestibular gain [40].



5.2 Intrinsic Membrane Properties and Plasticity

Vestibular nucleus (VN) neurons are not passive relays. They are capable of sustained, high-frequency firing and are electrically specialised to operate continuously with high temporal precision. Many VN neurons fire tonically at rates exceeding 100 Hz, which is essential for maintaining stable gaze and posture. This capability depends on intrinsic membrane properties that allow rapid recovery between spikes and prevent firing from collapsing during sustained input.

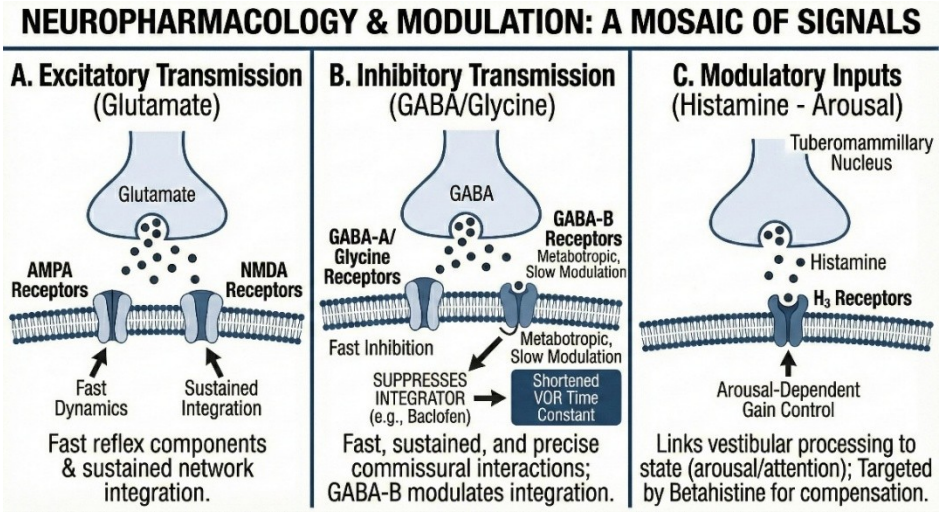
In addition to supporting fast discharge, VN neurons exhibit intrinsic plasticity. A key example is post-inhibitory rebound (PIR), in which a neuron increases firing immediately after a period of inhibition. Because head rotation excites one labyrinth and inhibits the other, this rebound mechanism enhances contrast in the push–pull system. In practical terms, the release from inhibition becomes an active signal, sharpening bilateral asymmetry and supporting rapid vestibular reflexes. These intrinsic properties help stabilise vestibular output and contribute to compensation after unilateral lesions.

5.3 Neuropharmacology

In addition to its intrinsic electrical properties, the vestibular nuclei (VN) are defined by a rich neurochemical environment. Rather than functioning as a uniform relay, the VN operates as a mosaic of excitatory, inhibitory, and modulatory influences. Primary vestibular afferents use glutamate as their neurotransmitter. Postsynaptically, glutamate acts mainly through AMPA receptors, which mediate fast synaptic transmission suited for rapid reflex responses, and NMDA receptors, which have slower kinetics and voltage-dependent properties that support sustained activity and integrative functions within the network [45]. This division aligns with functional demands: rapid components support immediate reflex adjustments, while slower components contribute to ongoing integration and velocity storage–related behaviour.

 Clinical Pearl: Vestibular suppressants (antihistamines, phenothiazines, benzodiazepines) blunt acute symptoms by reducing vestibular nucleus excitability — but they also impair central compensation. Use sparingly in acute vestibular loss; wean early to allow adaptation. Betahistine (H3 inverse agonist / H1 agonist) is used adjunctively: it enhances cochlear microcirculation and modulates histaminergic input to vestibular nuclei, though

clinical trial evidence for vertigo suppression remains mixed.



Inhibitory transmission within the VN is mediated by both GABA and glycine. These systems provide fast and precisely timed inhibition, particularly in commissural pathways that shape bilateral push–pull processing. Of special relevance clinically are GABA-B receptors, which are metabotropic and slower acting. GABA-B signalling plays a key role in velocity storage. Pharmacological activation of GABA-B receptors—for example with baclofen—reduces integrator activity and shortens the VOR time constant [46]. This provides a clear example of how receptor-level pharmacology can produce measurable changes in reflex dynamics.

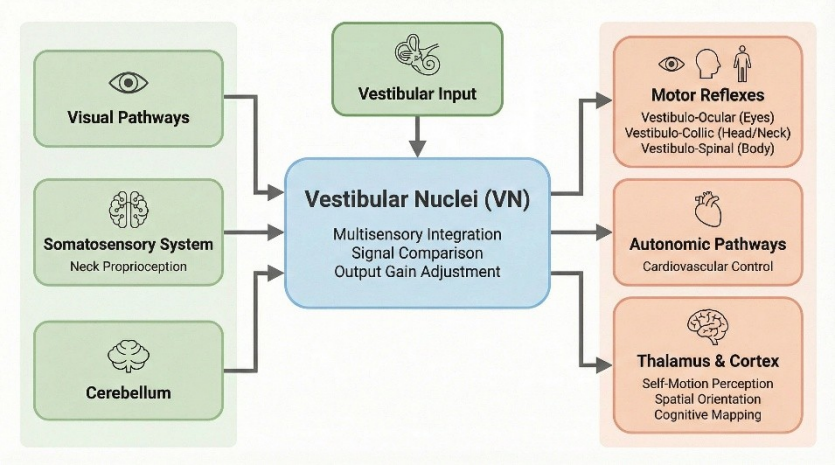
The VN also receives modulatory input linked to behavioural state. Histaminergic projections from the tuberomammillary nucleus influence VN activity, particularly via H3 receptors, adjusting vestibular gain according to arousal and attentional state [47]. This pathway has clinical relevance because it is targeted by betahistine. Modulating histaminergic signalling can alter network excitability and plasticity, potentially facilitating central compensation after vestibular injury.

Key Facts: Vestibular Nucleus Neuron Types

Neuron Type	Function	Clinical relevance
PVP (Position-Vestibular-Pause)	VOR premotor relay; pauses during saccades	VOR arc integrity; VOR gain
VO (Vestibular-Only)	Velocity storage; ascends to thalamus/cortex	Storage time constant; spatial orientation
FTN (Floccular Target)	Cerebellar error-correction interface	VOR adaptation; vestibular compensation
Gaze-holding integrator	Converts eye velocity to eye position	Gaze-evoked nystagmus if impaired

6. Reflex Systems and Control Theory

Having examined how vestibular signals are processed within the brainstem, we now consider how they are used to drive action. The vestibular nuclei do not simply represent motion; they generate outputs that feed directly into reflex pathways.

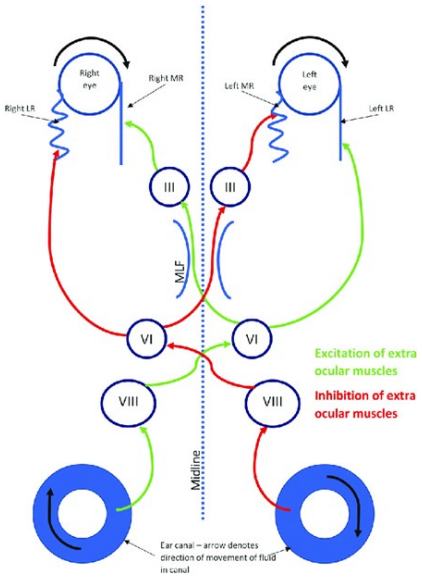


These reflex loops control eye movements, head and neck stability, whole-body posture, and even sympathetic cardiovascular responses. Because these systems must operate with very low latency, remain stable (without oscillation), and function across a wide range of movement frequencies, they can be usefully understood using principles from control systems theory.

In this framework, the brain acts as a controller that regulates different mechanical systems, or "plants." The eye plant, head–neck plant, whole-body postural plant, and cardiovascular plant each have distinct physical properties—different inertia, elasticity, and time delays. As a result, the vestibular controller must be tuned differently for each target. This perspective helps explain why vestibular disorders can present with diverse clinical patterns. A disturbance affecting eye stabilisation may look very different from one affecting posture or blood pressure regulation, even if the underlying sensory signal is similar.

6.1 The Vestibulo-Ocular Reflex (VOR)

6.1.1 Angular VOR (Canal-driven)



Among the vestibular reflexes, the vestibulo-ocular reflex (VOR) is the most clinically accessible and conceptually clear. Its function is straightforward: when the head rotates, the eyes rotate by an equal amount in the opposite direction so that the visual image remains stable on the retina. When the VOR is deficient, patients experience oscillopsia, reduced dynamic visual acuity, and often avoid head movement because vision becomes unstable. The VOR is central to vestibular physiology because it represents a sensory–motor loop in which the relationship between input (head motion) and output (eye motion) can be analysed with relative precision [48].

A defining feature of the **VOR is its extremely short latency.** Vestibularly driven eye movements begin within a few milliseconds of head rotation onset, typically under 10 ms, with many measurements clustering between 5–8 ms in healthy individuals [7]. This is substantially faster than visually mediated eye movements, which generally require more than 100 ms. As a result, the early VOR response is effectively open loop: it is generated before visual feedback can influence performance. The initial response therefore reflects the integrity of the peripheral labyrinth and brainstem circuitry rather than visual correction.

 Clinical Pearl: The video head impulse test (vHIT) directly probes the three-neuron VOR arc. A reduced VOR gain (below 0.8) with corrective saccades (covert or overt) = ipsilateral canal hypofunction. Key point: vHIT is canal-specific — horizontal canal vHIT tests the horizontal VOR; LARP/RALP positions test the vertical canals. A normal vHIT does NOT exclude peripheral vestibular pathology at low frequencies.

The classic VOR circuit is described as a **three-neuron arc**. Semicircular canal afferents project to vestibular nuclei neurons, which then project to extraocular motor nuclei (abducens, oculomotor, and for vertical and torsional components, trochlear). These nuclei activate the extraocular muscles to produce compensatory eye rotation. A key computational step is the transformation of head-centred velocity signals into eye-centred motor commands with the correct three-dimensional orientation and opposite direction. The push–pull organisation of paired canals is critical here: rotation excites one side and inhibits the other, creating a differential signal that enhances speed and signal-to-noise ratio.

6.1.2 Otolith Contributions to Ocular Control (Linear VOR and Ocular Tilt Reaction)

So far, the discussion of the VOR has focused mainly on the semicircular canals and rotational eye stabilisation (angular VOR). The otolith organs contribute to ocular control in two related but distinct ways.

 Clinical Pearl: Skew deviation is a vertical ocular misalignment from utricular pathway imbalance, not from CN IV palsy. Clinical rule: hypertropia on the lesion side suggests peripheral or lower-brainstem localisation; same-side hypertropia suggests pontine/midbrain. SVV deviation >3° confirms graviceptive pathway dysfunction. Skew deviation without other brainstem signs can be the presenting feature of a posterior fossa stroke — always consider neuroimaging.

First, there is the **linear VOR (lVOR)**. This is a dynamic reflex that stabilises gaze during head translation rather than rotation. For example, when the head moves forward while fixating a near target, the eyes must rotate in the opposite direction to maintain fixation. This reflex depends primarily on otolith input and is geometry-dependent, because the required eye movement varies with viewing distance. The lVOR is therefore the translational counterpart of the angular VOR.

Second, there are **gravity-dependent alignment pathways**, which are not primarily about stabilising vision during movement but about maintaining proper alignment of the eyes relative to gravity. These pathways use utricular input to maintain vertical ocular alignment and perception of upright. When these otolith–brainstem circuits are imbalanced, the result is the **ocular tilt reaction (OTR)**.

The ocular tilt reaction consists of a triad:

- Skew deviation (vertical misalignment of the eyes),
- Ocular torsion,
- Head tilt,
  often accompanied by deviation of the subjective visual vertical [7,9,49].

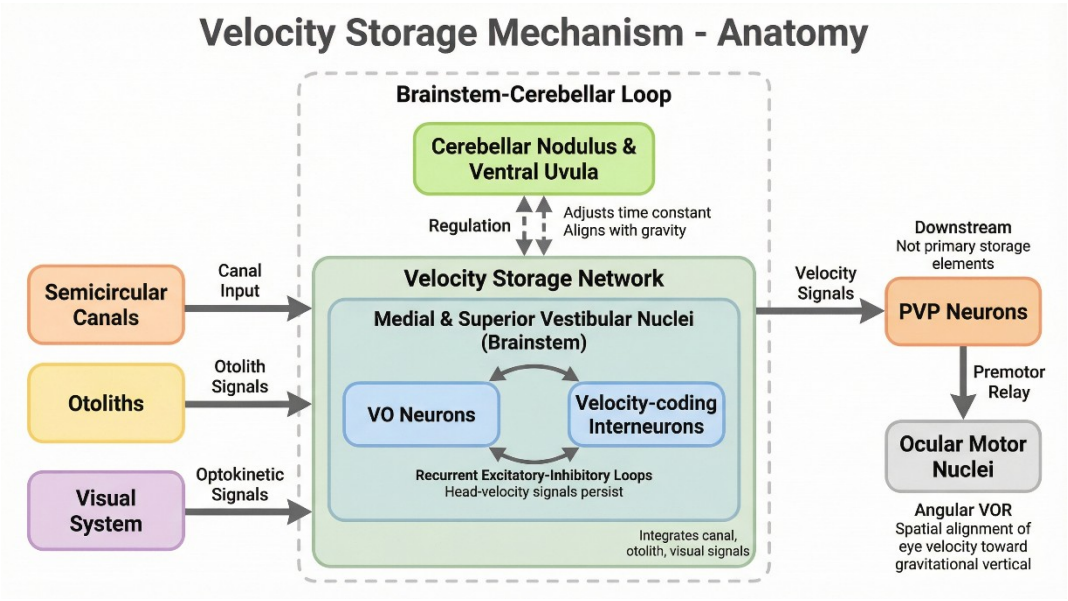
OTR reflects a biased estimate of gravity rather than a failure of rotational stabilisation. In other words, it is not primarily a problem of dynamic VOR performance, but a disorder of graviceptive alignment. Skew deviation can arise anywhere along the utriculo-ocular pathway, from peripheral otolith afferents through vestibular nuclei and ascending projections. It may be accompanied by torsional nystagmus and abnormal ocular counterroll.

It is important to distinguish this from **convergence-retraction nystagmus**, which is not an otolith-mediated phenomenon. Convergence-retraction nystagmus is a dorsal midbrain sign (classically associated with Parinaud syndrome) and is relevant mainly for anatomical localisation when vertical gaze abnormalities coexist [7,49].

6.1.3 Velocity Storage and Visual–Vestibular Integration

If the VOR depended only on semicircular canal mechanics, eye velocity would decay within a few seconds during sustained rotation. Instead, VOR responses typically persist much longer, often with a dominant time constant of approximately 15–20 seconds in humans. This prolongation reflects **velocity storage**, a central brainstem–cerebellar mechanism that extends canal-driven velocity signals beyond peripheral mechanical limits [46,50–52].

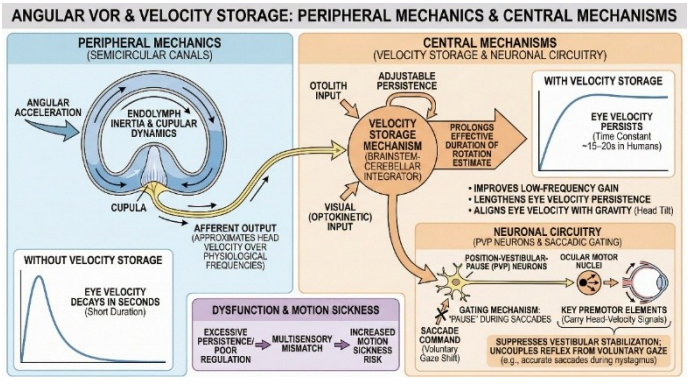
 Clinical Pearl: Velocity storage is the physiological substrate for visually-induced dizziness (VID). When the storage network receives optokinetic input (e.g., scrolling screens, moving traffic) that conflicts with vestibular stillness, it generates a 'real motion' estimate — experienced as dizziness, nausea, or postural instability. This mechanism is amplified in Persistent Postural-Perceptual Dizziness (PPPD). Treatments targeting visual reliance (vestibular-first rehabilitation, graded visual desensitisation) work partly by recalibrating this storage-optokinetic coupling.



Anatomically, velocity storage is centred within the **medial and superior vestibular nuclei. Vestibular-only (VO) neurons and related velocity-coding interneurons** form recurrent circuits that allow head-velocity signals to persist after peripheral decay. Position-vestibular-pause (PVP) neurons transmit velocity signals to ocular motor nuclei but are not considered the primary storage elements; they function mainly as premotor relay neurons for execution of the angular VOR.

Crucially, velocity storage is not a purely vestibular process. Optokinetic pathways, driven by full-field visual motion, converge on the same vestibular nuclear circuitry. Canal input during rotation in darkness charges the storage network and produces post-rotatory nystagmus that decays with the storage time constant. Similarly, sustained visual motion can charge the same system, producing **optokinetic nystagmus (OKN),** and when the visual motion stops, **optokinetic after-nystagmus (OKAN)** reflects discharge of the same storage mechanism. In this sense, velocity storage behaves as a shared internal “state variable” that integrates vestibular and visual motion signals.

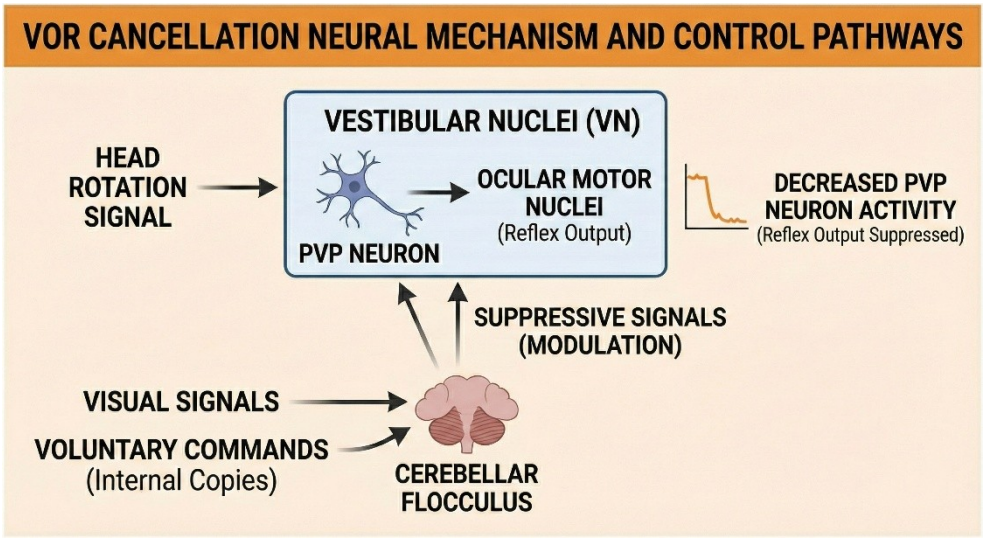
The storage network is **regulated by the cerebellar nodulus and ventral uvula.** These structures adjust the time constant of storage and ensure that stored velocity remains aligned with gravity. During prolonged off-vertical rotation, the axis of nystagmus gradually reorients toward the gravitational vertical, reflecting cerebellar supervision of spatial alignment [53]. Lesions of the nodulus–uvula shorten or destabilise velocity storage, and stimulation can rapidly discharge the stored signal.



Clinically, abnormal velocity storage–optokinetic coupling helps explain prolonged motion aftereffects, visually induced dizziness, and motion sickness susceptibility. Excessively persistent or poorly regulated storage increases multisensory conflict, whereas reduced storage can weaken low-frequency gaze stabilisation. Different clinical tests probe different components of this system: head impulse testing assesses the short-latency canal arc, prolonged rotation in darkness assesses the storage time constant, and OKN/OKAN testing assesses visual–vestibular convergence.

6.1.4 VOR Cancellation

The VOR is not an inflexible reflex that always operates at full gain. In many behavioural contexts, it must be actively suppressed. For example, when a person fixates on a target that moves with the head—such as their own thumb during head rotation—the appropriate eye movement is to remain aligned with the target rather than counter-rotate. In this situation, the VOR must be cancelled.



VOR cancellation depends on cortical and cerebellar control, particularly the **cerebellar flocculus.** Visual signals and internal copies of voluntary head-movement commands inform the cerebellum that compensatory eye movements are not required, allowing the VOR to be suppressed. The flocculus modifies activity within vestibular nuclei circuits, reducing vestibular-driven eye velocity commands. At the neuronal level, **PVP neurons** decrease their activity during cancellation, reflecting suppression of reflex output.

 Clinical Pearl: Test VOR cancellation at the bedside: ask the patient to fixate their own outstretched thumb while you rotate their whole body (or they rotate their head onto a fixed trunk). Persistent nystagmus during fixation = impaired suppression = cerebellar floccular dysfunction. This is distinct from a normal head impulse test — a patient can have an intact three-neuron arc (normal vHIT) but deficient floccular modulation (impaired VOR suppression).

Clinically, **impaired VOR cancellation is a classic sign of cerebellar dysfunction, especially floccular pathology.** It can be tested by asking the patient to fixate on a head-fixed target during passive head rotation. If corrective eye movements persist instead of being suppressed, cerebellar modulation is defective.

VOR cancellation illustrates a central principle of vestibular physiology: reflex output is state-dependent. The same head-velocity signal can either drive compensatory eye movement or be suppressed, depending on behavioural goals and predictive context.

A **key clinical parameter of the VOR is gain,** defined as the ratio of eye velocity to head velocity (with opposite sign). Ideally, gain is close to 1.0, meaning the eyes rotate equally and oppositely to the head. If gain is reduced, retinal slip occurs, leading to oscillopsia and catch-up saccades. In addition to gain, **phase** is important: eye velocity must be appropriately timed relative to head velocity. Errors in either gain or phase result in dynamic visual blur.

The **VOR is also plastic**. Its performance can be recalibrated when persistent retinal slip signals an error. The **cerebellar flocculus and ventral paraflocculus are central** to this adaptation process, adjusting reflex gain and timing. Clinically, VOR abnormalities broadly reflect either reduced peripheral drive (e.g., canal hypofunction) or impaired central calibration (e.g., cerebellar dysfunction or abnormal velocity storage). These mechanisms may coexist but often produce different patterns across tests.

It is useful to distinguish **angular VOR (aVOR)** from **linear VOR (lVOR)**. The aVOR, driven by semicircular canals, stabilises gaze during head rotation. The lVOR, driven primarily by otolith organs, stabilises gaze during translation and depends on viewing distance. Because translational demands are geometry-

dependent and often integrate visual input, lVOR testing is less straightforward than rotational testing in clinical settings.

Finally, the VOR is context-dependent rather than rigid. It can be suppressed when stabilisation would be inappropriate, such as during voluntary head movement while tracking a head-fixed target. This cancellation reflects cerebellar modulation of brainstem circuits. Together, these features emphasise that the VOR is a dynamically regulated control system rather than a fixed reflex.

6.1.5 The 3D coordinate transform problem

Head movements are typically oblique combinations of yaw, pitch, and roll. Because semicircular canals are oriented in oblique planes, the brain must transform canal signals into the correct three-dimensional eye movement commands. The VOR therefore computes eye rotation about the same instantaneous axis as head rotation, but in the opposite direction.

However, eye movements are constrained by **Listing's law.** The system does not always achieve perfect geometric axis alignment; instead, it prioritises mechanical efficiency of the eye plant. Mild torsional underperformance during oblique rotations reflects this physiological compromise rather than reflex failure.

6.1.5 Bedside Interpretation: Fast Arc vs Velocity Storage

Understanding the VOR at the bedside requires separating two components:

1. The **short-latency three-neuron arc** (peripheral canal-driven response)
2. The **longer time-constant velocity storage network** (central persistence)

These components are stressed by different tests and may fail independently.

6.1.5.1 Probing the Short-Latency Arc: vHIT and the Head Impulse Test

The bedside head impulse test and video head impulse testing (vHIT) assess the high-frequency, short-latency component of the VOR. Because the head impulse occurs over milliseconds, it primarily tests the direct canal → vestibular nucleus → ocular motor pathway and largely bypasses velocity storage.

A peripheral lesion produces:

- Reduced VOR gain
- Overt or covert corrective saccades

In contrast, patients with central disorders affecting velocity storage or visual–vestibular integration (e.g., visually induced dizziness, mal de Débarquement–like syndromes) may have normal vHIT because the peripheral canal function is intact.

Thus, vHIT isolates the fast canal-driven arc.

6.1.5.2 Probing the Long Time Constant: Headshake and Post-Rotatory Testing

To assess velocity storage, sustained or repetitive stimulation is required.

Head-shake nystagmus (HSN) is the most practical bedside method. After rapid horizontal head oscillations:

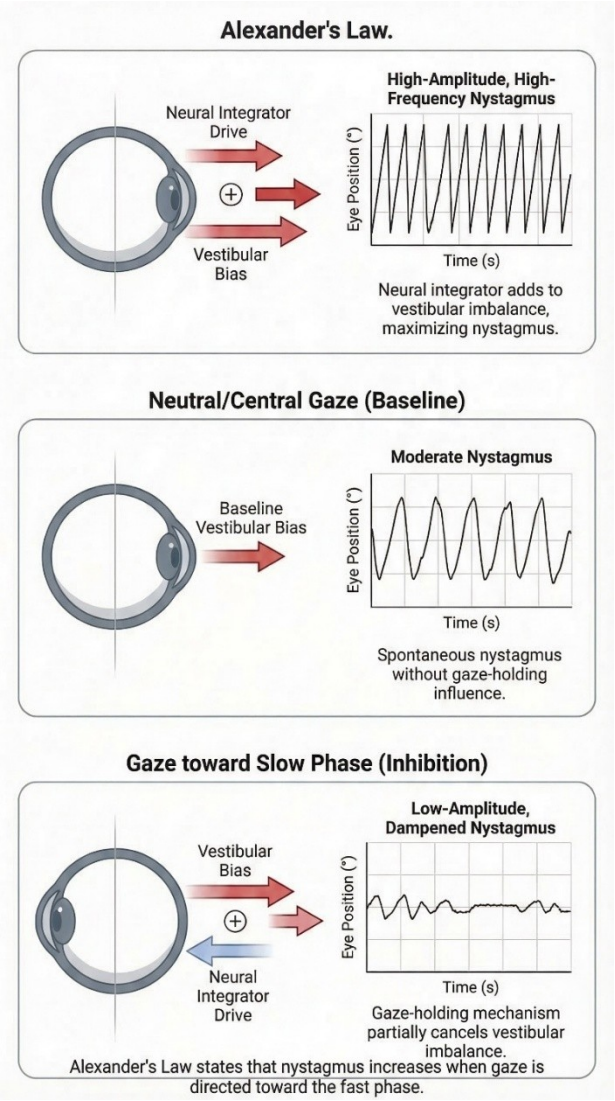
- Brief, low-amplitude nystagmus often reflects peripheral asymmetry.
- Prolonged or high-intensity nystagmus suggests altered central storage dynamics.

Post-rotatory testing in darkness also probes storage time constant. Canal input charges the storage network during rotation; when rotation stops, the persistence of nystagmus reflects the strength and damping of storage.

These tests assess the slower integrative component of the VOR rather than the fast arc.

6.1.6 Alexander's Law and Gaze Dependence

A classic bedside observation in acute vestibular imbalance is **Alexander's law**: spontaneous **vestibular nystagmus increases when the patient looks in the direction of the fast phase and decreases when looking toward the slow phase.** This occurs because a persistent vestibular tone imbalance (typically from unilateral hypofunction) interacts with the gaze-holding neural integrator. When the eyes are held in the direction of the fast phase, the neural integrator adds position-related drive to the existing vestibular bias, making the nystagmus appear stronger. When gaze is directed toward the slow phase, this added drive partially counteracts the imbalance, reducing its intensity. Clinically, Alexander's law highlights that spontaneous nystagmus reflects both peripheral asymmetry and central gaze-holding mechanisms, and its presence supports an acute peripheral vestibular lesion while also demonstrating intact brainstem integrator function.



6.3.4 Gravity Dependence and Storage Regulation

Velocity storage is normally regulated in a gravity-referenced frame by the cerebellar nodulus and uvula. In some patients, particularly with cerebellar involvement, storage may be poorly damped or improperly aligned with gravity. This can produce exaggerated motion aftereffects or visually induced dizziness.

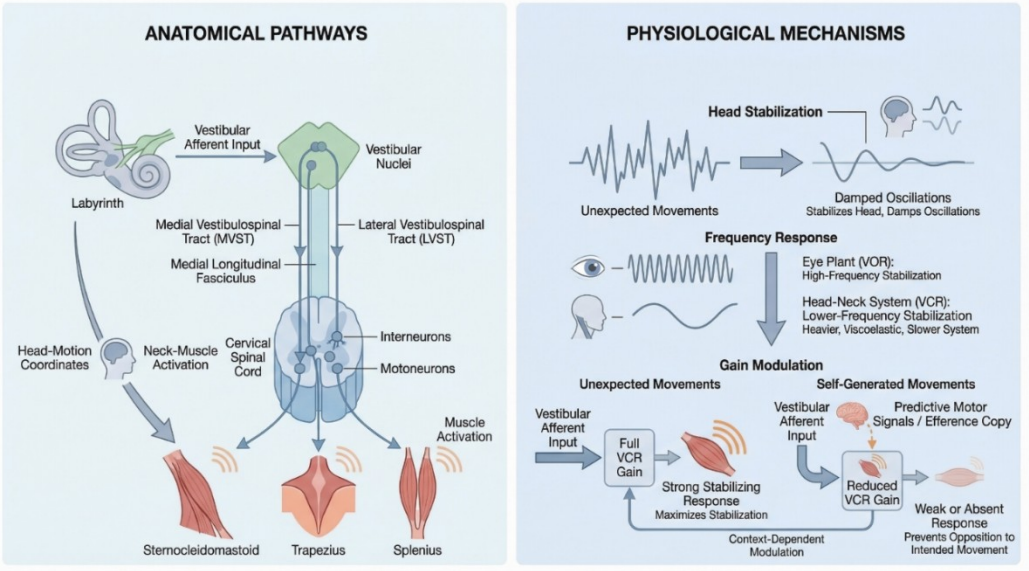
While specialised "tilt-dump" testing can probe this mechanism, in routine clinical practice it is more important to recognise patterns of abnormal persistence and spatial disorientation rather than perform formal gravity discharge tests.

6.4 The vestibulo-collic reflex (VCR)

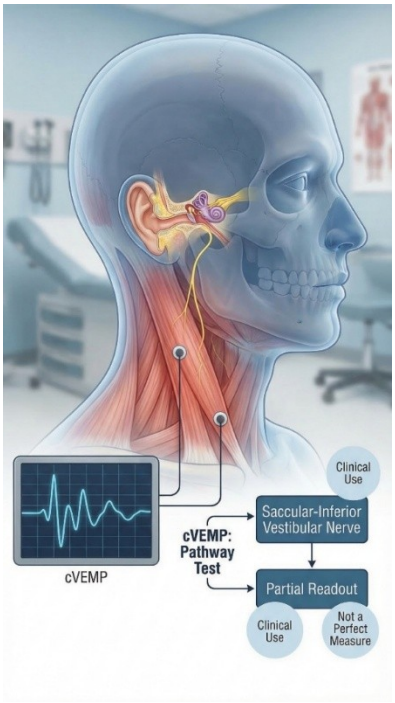
The vestibulo-collic reflex (VCR) is the neck-muscle counterpart of the vestibulo-ocular reflex. Whereas the VOR stabilises the visual image by moving the eyes, the VCR contributes to stabilising the head in space by driving cervical musculature. This broader perspective is important in vestibular physiology, because gaze stability in daily life does not depend on the eyes alone; it depends on coordinated control of the head, neck, and eyes as an integrated sensorimotor system. For that reason, the VCR should be understood not as an isolated reflex, but as one of the major motor outputs of the vestibular system, alongside the VOR and vestibulospinal pathways [91,92].

Anatomically, the VCR begins with vestibular afferent input from the labyrinth to the vestibular nuclei, and from there descends to the cervical spinal cord through vestibulospinal pathways. The medial vestibulospinal tract (MVST) descends within the medial longitudinal fasciculus and projects mainly to cervical levels, making it especially relevant for head and neck control. The lateral vestibulospinal tract (LVST), although classically associated with posture and extensor control, also has cervical-projecting components that contribute to neck

and shoulder stabilisation. Experimental work has shown that these descending pathways influence interneurons and motoneurons supplying muscles such as sternocleidomastoid, trapezius, and splenius. In physiological terms, the vestibular signal therefore has to be transformed from head-motion coordinates into coordinated patterns of neck-muscle activation rather than into a simple one-sensor/one-muscle mapping [93].



Physiologically, the VCR helps to stabilise the head during unexpected body movements and may also help damp oscillations of the head–neck system. This reflex operates under very different mechanical conditions from the VOR: the head and neck are heavier, more viscoelastic, and mechanically slower than the eye plant, so the VCR is relatively more useful for lower-frequency stabilisation than for the very fast bandwidth achieved by the VOR. Another important point is that the VCR is not simply switched on at full gain during all movements. Review work suggests that vestibulo-collic responses are reduced during self-generated head movements, probably because the central nervous system uses predictive motor signals, including efference copy, to avoid reflexively opposing intended movement. This mirrors a broader theme in vestibular physiology: vestibular reflexes are continuously modulated according to behavioural context rather than functioning as rigid output loops [92]. In clinical practice, the most useful window into vestibulo-collic function is the cervical vestibular-evoked myogenic potential (cVEMP), recorded from a pre-contracted sternocleidomastoid muscle. Under standard clinical stimulation conditions, **cVEMP** is widely interpreted as reflecting a **predominantly saccular–inferior vestibular nerve driven otolith-cervical pathway**, and animal and human lesion/stimulation studies strongly support this interpretation.



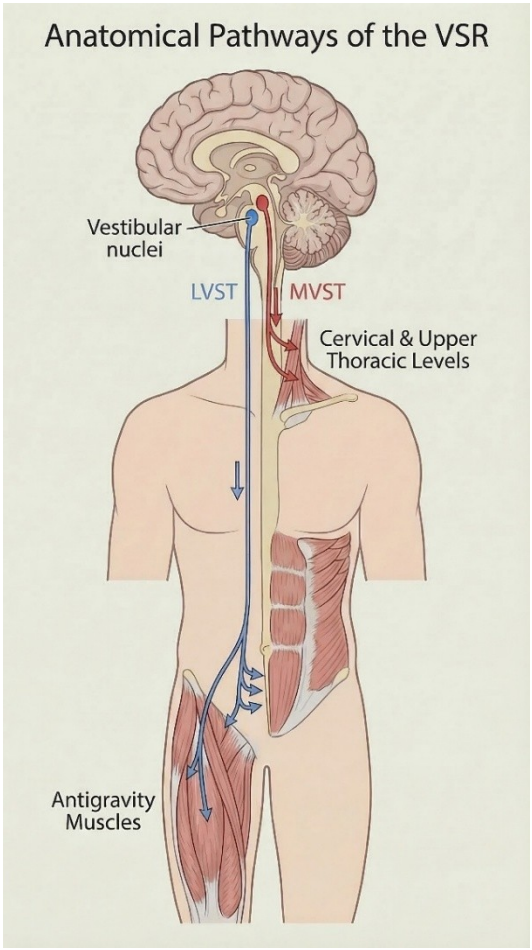
At the same time, more recent critical reviews have emphasised that cVEMP should be interpreted as a pathway test rather than as a perfect one-to-one measure of total saccular function, because normal cVEMP responses do not necessarily prove intact function of the entire sustained otolith system and contributions from other vestibular end organs cannot be completely excluded. In practical terms, cVEMP is therefore best viewed as a clinically valuable but physiologically partial readout of vestibulo-collic circuitry [94,95].

This is why cVEMP complements, rather than duplicates, VOR testing. The VOR mainly assesses canal-driven ocular stabilisation through tests such as vHIT, calorics, and rotational chair testing, whereas cVEMP provides access to otolith-driven cervical pathways. Taken together, these tests offer a broader functional map of the vestibular system: canal-ocular, otolith-cervical, and, when combined with other measures such as oVEMP and balance testing, increasingly precise lesion localisation. For vestibular clinician, the key clinical message is that VOR and VCR are parallel expressions of the same vestibular physiology, and both are needed to appreciate how the vestibular system stabilises vision, head position, and orientation in space [92,94,95].

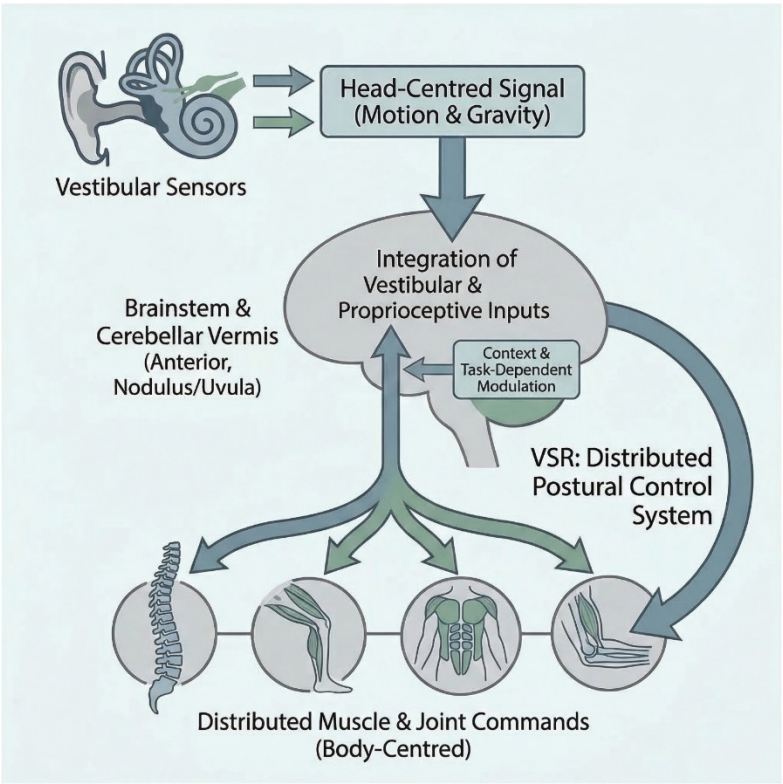
6.5 The vestibulo-spinal reflex (VSR)

The vestibulo-spinal reflex (VSR) is the major descending vestibular system responsible for stabilising posture. Whereas the VOR stabilises the visual image and the VCR helps stabilise the head, the VSR helps stabilise the body as a whole by converting vestibular estimates of head motion and orientation into spinal motor commands for the trunk and limbs. In functional terms, it is the **vestibular system's principal postural output**, allowing the body to resist sway, maintain upright stance, and respond rapidly to unexpected perturbations. This role is especially important because postural control depends on continuous integration of vestibular, visual, and somatosensory information, with vestibular input becoming particularly important when the other cues are unreliable or conflicting [96,97].

Anatomically, the VSR arises from vestibular nuclei projections to the spinal cord through two principal pathways: **the lateral vestibulospinal tract (LVST) and the medial vestibulospinal tract (MVST).** The **LVST** originates mainly from the **lateral vestibular nucleus and descends predominantly ipsilaterally** through the spinal cord, where it has major functional links to antigravity musculature, extensor tone, and stance stability. The **MVST** arises from rostral portions of the **descending vestibular nucleus and adjacent medial and lateral vestibular nuclei, descends in the medial longitudinal fasciculus, and projects mainly to cervical and upper thoracic levels,** where it contributes to head, neck, and upper trunk control [97,98]. Thus, although the VSR is often discussed as a single reflex, its anatomy already shows a division of labour: the LVST is more strongly linked to whole-body postural support, whereas the MVST is more closely linked to segmental control of the upper body and natural interaction with vestibulo-collic function [97,98].



Physiologically, the VSR must solve a more complex motor problem than the VOR. Vestibular sensors encode head motion relative to gravity, but corrective outputs must be distributed across many muscles and joints to generate an appropriate whole-body response. In other words, the vestibular signal has to be transformed from a head-centred estimate of self-motion into body-centred motor commands. This is why vestibular influences on posture are strongly task-, muscle-, and context-dependent rather than fixed one-to-one responses. Modern work on the cerebellar vermis has further clarified this point: the anterior vermis and nodulus/uvula integrate vestibular and proprioceptive inputs to transform head-centred vestibular signals into body-centred postural commands, while also modulating reflex gain according to

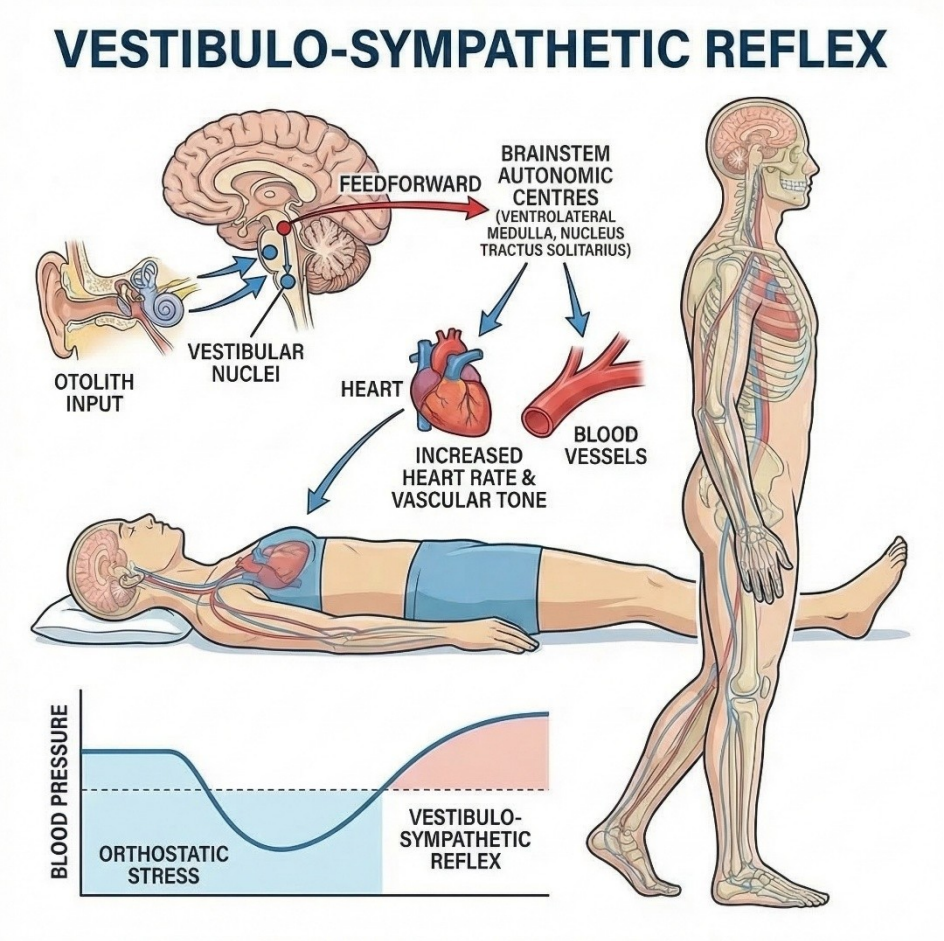


behavioural context [99]. This makes the VSR not just a spinal reflex, but a distributed brainstem–cerebellar postural control system [97,99].

From a practical clinical perspective, VSR dysfunction is recognised less by a single “pure” bedside reflex than by its behavioural consequences: postural instability, gait disequilibrium, increased sway, and dependence on visual and somatosensory cues. Unlike the VOR, which can be observed directly in eye movements, the VSR is inferred from stance and gait testing, posturography, vestibular-evoked balance responses, and galvanic vestibular stimulation paradigms. Experimental studies show that vestibulospinal responses can be recorded from neck, trunk, and leg muscles, although the magnitude and polarity of these responses vary with head position and body configuration, emphasising that vestibular postural output is highly state-dependent [100,101]. Clinically, this is why postural manifestations of vestibular disease are often context-sensitive: a patient may cope reasonably well in a stable visual environment but deteriorate markedly in darkness, on uneven ground, or during challenging balance tasks. For residents, the key point is that the VSR complements the VOR and VCR: together, these three outputs explain how vestibular physiology stabilises the image, the head, and the body in space [97,100,101].

6.6 The vestibulo-sympathetic reflex

The vestibulo-sympathetic reflex is the **vestibular contribution to cardiovascular stability during movement and postural transitions**. Whereas the VOR stabilises the visual image, the VCR stabilises the head, and the VSR stabilises posture, the vestibulo-sympathetic reflex helps preserve cerebral and systemic perfusion when body position changes relative to gravity. In this broader physiological framework, the vestibular system is not simply a balance organ; it is also an anticipatory regulator of autonomic function. This is especially important during standing, head-up tilt, and other orthostatic challenges, when blood can rapidly pool in the lower body and reduce venous return before slower compensatory systems fully respond [102-104].



Anatomically, the vestibulo-sympathetic reflex begins with vestibular afferent input, particularly from the **otolith organs,** which are well suited to detect head tilt and linear acceleration associated with postural change. These signals reach the vestibular nuclei and are then relayed to autonomic regulatory centres in the brainstem, including **the rostral ventrolateral medulla (RVLM), caudal ventrolateral medulla (CVLM), and nucleus tractus solitarius (NTS)** [102,103,105,106]. The RVLM is especially important because it provides a major descending drive to sympathetic preganglionic neurons in the intermediolateral cell column of the spinal cord. In this way, vestibular signals can influence vascular tone, heart rate, and regional blood distribution through direct and indirect brainstem autonomic pathways [102,103,105].

Physiologically, the key feature of the vestibulo-sympathetic reflex is that it acts as a feedforward mechanism. The baroreflex is the classic feedback system for blood pressure control: it detects a fall in arterial pressure after it has occurred and then initiates compensatory sympathetic responses. By contrast, vestibular input can respond earlier, because the otolith organs detect body tilt and movement as soon as posture changes begin. This allows the vestibular system to bias sympathetic outflow in anticipation of the haemodynamic consequences of standing, thereby reducing the magnitude of blood pressure fall before the baroreflex has fully engaged [102-104]. In this sense, vestibular-autonomic pathways complement rather than replace baroreflex control.

This anticipatory role has been supported by both animal and human studies. Experimental work has shown that vestibular nucleus neurons project to key autonomic centres and that lesions of the labyrinth or brainstem autonomic relay sites can diminish vestibulo-sympathetic responses [102,103,105]. Human studies using otolith stimulation, such as head-down rotation, have also demonstrated changes in muscle sympathetic nerve activity consistent with vestibular modulation of sympathetic outflow [104]. These findings support the general principle that the vestibular system contributes to defending against orthostatic stress and that loss of vestibular input may leave the body more dependent on slower cardiovascular feedback mechanisms.

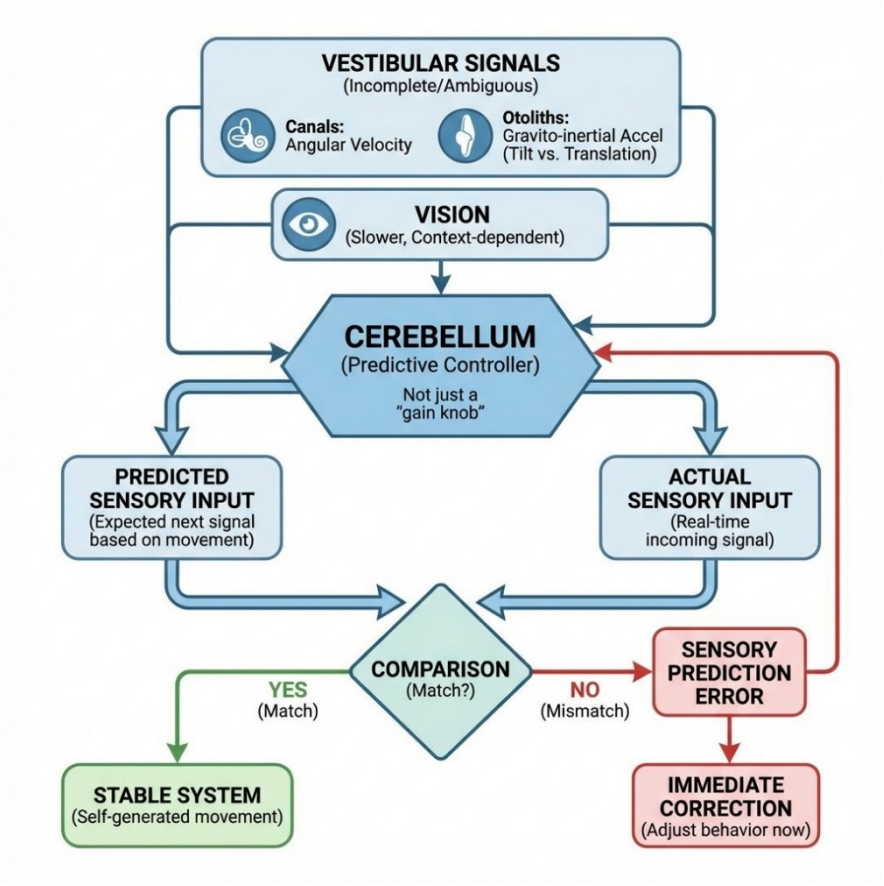
Clinically, the vestibulo-sympathetic reflex is most relevant in disorders characterised by orthostatic dizziness, presyncope, motion sensitivity, and abnormal autonomic responses to position change. Its dysfunction does not usually produce a single pathognomonic bedside sign in the way that abnormal head impulse testing reflects VOR dysfunction. Instead, it is inferred from the clinical context: symptoms provoked by standing, abnormal autonomic testing, or failure to tolerate orthostatic stress despite apparently intact cardiovascular structure. This is also why vestibular physiology has relevance beyond eye movements and balance alone. In a comprehensive understanding of vestibular function, the vestibulo-sympathetic reflex demonstrates that vestibular inputs are used not only to stabilise gaze and posture, but also to maintain internal physiological stability in a gravitational environment [102,103,107].

Key Facts: Vestibular Reflexes

Reflex	Stimulus	Output	Key Test
Angular VOR	Canal (angular accel)	Compensatory eye rotation	vHIT; calorics
Linear VOR	Otolith (linear accel)	Translational eye movement	Head translation testing
Ocular Tilt Reaction	Utricle (graviceptive)	Skew + torsion + head tilt	SVV; fundus photography
VCR	Canals + otoliths	Head / neck stabilisation	Clinical head-on-trunk exam
LVST (lateral VSR)	Utricle; facilitates extensors	Postural extensor tone	Platform posturography
MVST (medial VSR)	Canals; inhibits extensors	Neck/axial reflexes	Clinical exam
Vestibulo-sympathetic	VN to brainstem	BP regulation; orthostasis	Tilt-table; orthostatic HR/BP

7. Cerebellar Computation and Predictive Coding

Having described the brainstem reflex circuits, the next question is how vestibular behaviour remains accurate, stable, and adaptable across different contexts. The cerebellum is central to that answer. It is not simply a "gain knob" that turns reflexes up or down. Rather, it acts as a predictive controller that continuously asks a practical question: *if the body has just moved in a certain way, what sensory input should arrive next?* When actual sensory input matches that prediction, the movement can be treated as self-generated and the system remains stable. When the incoming signal does not match what was expected, the brain detects a **sensory prediction error**. That error can be used immediately to correct behaviour and, over time, to recalibrate the system for future movements.



This predictive role is especially important in vestibular physiology because the raw signals themselves are incomplete and sometimes ambiguous. The semicircular canals encode angular velocity, not position. The otolith organs encode gravito-inertial acceleration, which means they cannot by themselves distinguish head tilt from linear translation. Vision helps, but it is slower and heavily context dependent. The cerebellum helps combine these signals into a coherent estimate of head motion and orientation in space, while keeping vestibular reflexes appropriately tuned across different speeds, tasks, and environments [60-62].

7.1 Internal Models and Predictive Coding

A major challenge in vestibular control is distinguishing **exafference** (motion imposed on the body from outside) from **reafference** (sensory input generated by one's own movement). A widely used framework is that the vestibular cerebellum implements a **forward internal model**. In this model, the brain sends a motor command to move the head or body and, at the same time, sends an internal copy of that command to the cerebellum. The cerebellum uses that copy to predict the sensory consequences of the movement and then compares the prediction with the actual vestibular input returning from the labyrinth [60-63]. When prediction and reality match, the input is interpreted as expected self-motion. When they do not match, the resulting prediction error signals that something unexpected has happened—for example, the body has been pushed, the surface has slipped, or the movement did not occur as intended.

Researchers often describe this process using a **Kalman filter–like** framework. The essential idea is simple: the brain combines a prediction with incoming sensory measurements and weights them according to how reliable each one is in the current context. In darkness, visual information becomes less reliable, so vestibular and predictive signals carry more weight. During sudden unexpected perturbations, prediction becomes less reliable, so real sensory input must dominate. This reliability-weighted estimation provides a useful way of understanding how the brain maintains stable orientation in the face of noisy or incomplete sensory input [61,64-65].

Physiological studies support this view. Many vestibular nucleus neurons respond strongly to passive head motion but much less to comparable active head motion. This does not mean the canals are silent during self-generated movement; rather, it suggests that expected sensory input is centrally cancelled or attenuated. More recent work has shown that cerebellar pathways can explicitly encode sensory prediction errors during self-motion, closely matching the logic of **internal model theories** [66-68]. In practical terms, when you move your own head in a planned way, the brain tries not to overreact to that expected vestibular signal. When the movement is unexpected, the mismatch is highlighted and demands attention.

7.2 Synaptic Plasticity and Learning

Prediction is only useful if the system can learn from its mistakes. The clearest experimental example of cerebellar learning in vestibular physiology is **VOR/OKR adaptation**. If retinal slip persists—that is, if the visual image continues to move on the retina during head motion—the system treats this as an error and gradually adjusts reflex gain and timing to reduce that error on future movements. The cerebellar flocculus and its downstream vestibular nucleus targets are central to this process [69-70].

 Clinical Pearl: VOR adaptation is the cellular basis of gaze stabilisation exercises in vestibular rehabilitation. Retinal slip during head movement = error signal → climbing fibre activation → LTD at parallel fibre–Purkinje cell synapse → recalibrated VOR gain. This is why exercises that generate controlled retinal slip (moving the head while fixing on a target) drive adaptation faster than static balance training. The flocculus must be intact for this to work — cerebellar pathology blunts rehabilitation response.

The classic teaching signal for this learning is thought to arrive via **climbing fibres** from the inferior olive, which signal that an error has occurred, commonly operationalised as retinal slip. **Parallel fibres** provide the context in which that error occurred, such as the pattern of head movement or visual motion. When contextual input and error input coincide, synaptic strength changes. In this way, the cerebellum does not simply react to sensory input; it uses error signals to improve future performance.

Traditionally, the best-known cellular mechanism here has been **long-term depression (LTD) at the parallel fibre–Purkinje cell synapse**. In simple terms, LTD weakens those synaptic inputs that were active when the error occurred, thereby altering Purkinje cell output and retuning downstream vestibular circuits [69,71]. However, this is no longer viewed as the whole story. Modern evidence shows that **long-term potentiation (LTP)** at parallel fibre synapses and plasticity within the vestibular nuclei themselves also contribute, and that different phases of learning may rely on different combinations of these mechanisms [70,72]. The key takeaway is that **cerebellar learning is distributed:** it involves **both cerebellar cortex and brainstem circuits,** allowing adaptation to be both rapid and durable.

At the molecular level, LTD at the parallel fibre–Purkinje cell synapse depends on a rise in postsynaptic calcium and activation of intracellular signalling pathways including PKC and CaMKII. These cascades alter AMPA receptor trafficking, particularly involving GluR2-containing receptors, and thereby reduce synaptic strength [71,73-74]. The details matter less than the principle: millisecond-scale sensory errors can be converted into long-term changes in circuit behaviour.

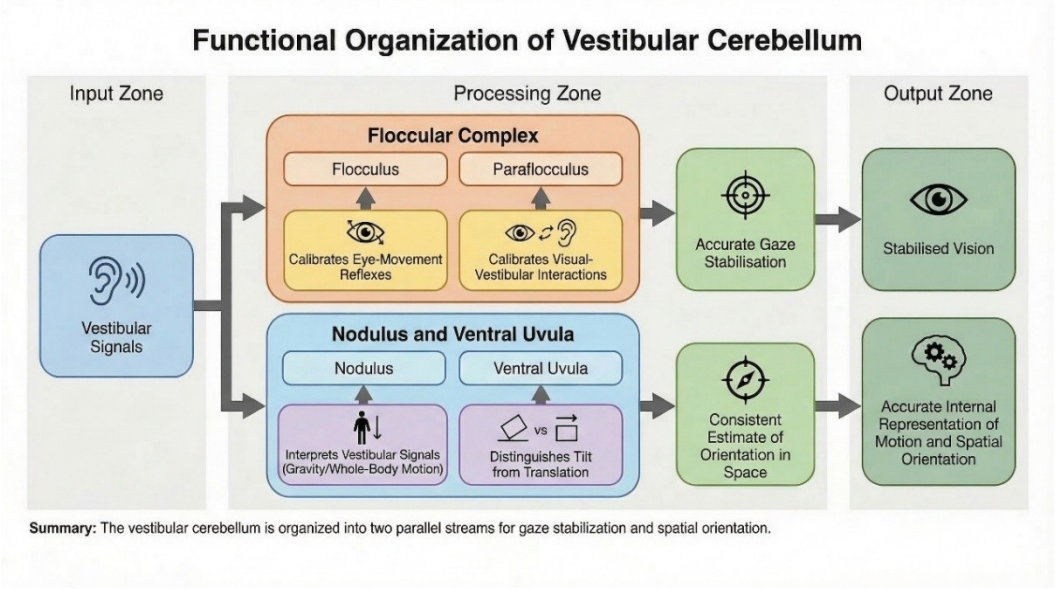
7.3 The Vestibular Cerebellum Is Not One Module: Flocculus versus Nodulus/Uvula

Functional organisation of the vestibular cerebellum

The vestibular cerebellum is often described as operating through two complementary functional modules. The **floccular complex (flocculus and paraflocculus)** primarily calibrates eye-movement reflexes and visual–vestibular interactions, ensuring that gaze stabilisation remains accurate during head motion and changing visual conditions. The **nodulus and ventral uvula**, by contrast, focus on interpreting vestibular signals in relation to gravity and whole-body motion, helping the brain distinguish tilt from translation and maintaining a physically consistent estimate of orientation in space. Together, these two cerebellar regions

allow the vestibular system not only to stabilise vision but also to maintain an accurate internal representation of motion and spatial orientation.

Floccular complex (flocculus + paraflocculus): gaze stabilisation, cancellation, pursuit, gaze holding, and downbeat nystagmus

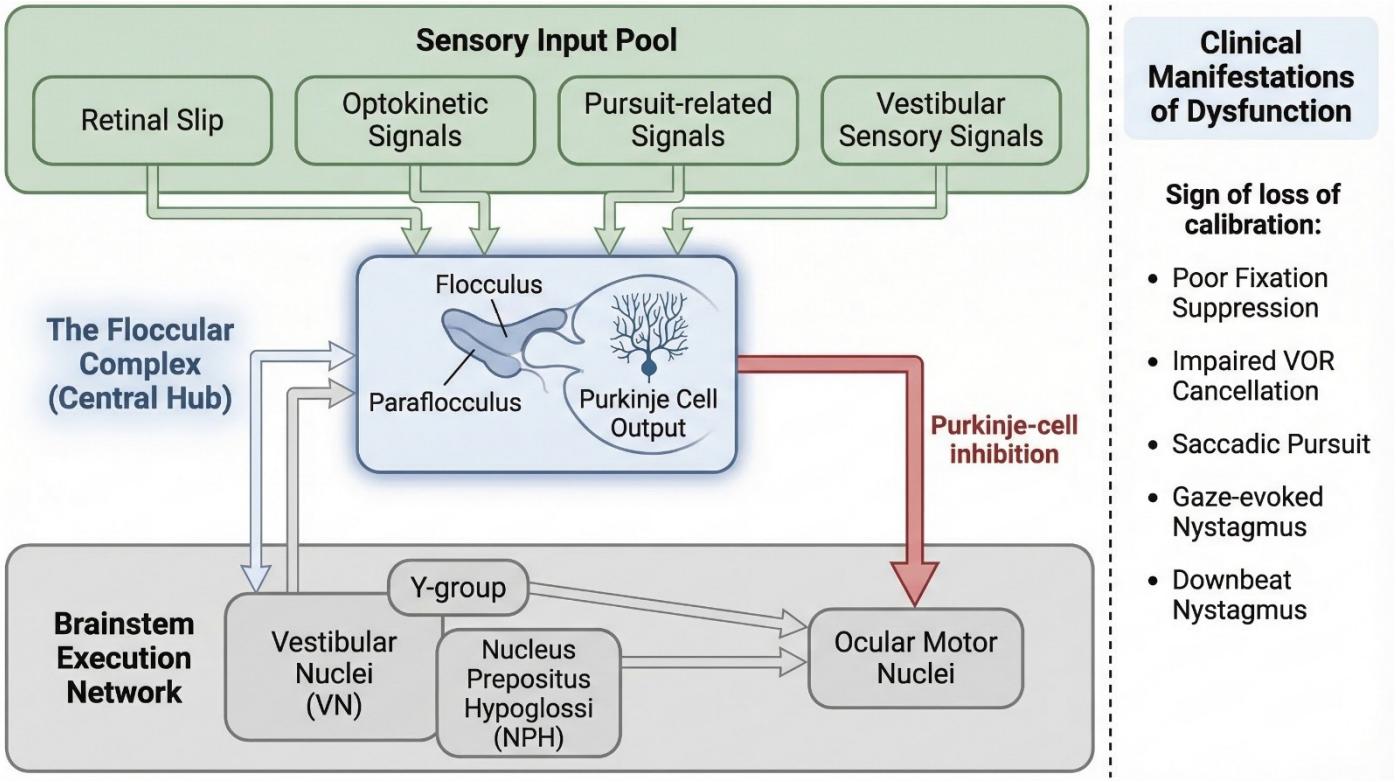


The floccular complex sits at the visual–vestibular interface of the cerebellum. It receives retinal slip, optokinetic, pursuit-related, and vestibular signals, and sends Purkinje-cell output back to vestibular premotor circuits—neurons mainly within the vestibular nuclei (including position–vestibular–pause and vestibular-only neurons) that transform vestibular sensory signals into eye-velocity motor commands before they reach the ocular motor nuclei—that generate eye movements. For horizontal eye movements, this output interacts mainly with vestibular nuclei and related gaze-holding networks such as the nucleus prepositus hypoglossi (NPH) and adjacent medial vestibular nucleus circuits for horizontal gaze holding; for vertical and torsional eye movements, it also interfaces with relay structures such as the superior vestibular nucleus and the Y-group. The Y-group is a small cluster of neurons located in the dorsolateral vestibular nuclear complex near the superior vestibular nucleus that participates in vertical and torsional vestibulo-ocular pathways, particularly in transforming vestibular signals into appropriate vertical eye-movement commands. In physiological terms, the flocculus does not replace the brainstem ocular motor network; rather, it continuously calibrates that network so that the VOR, smooth pursuit, fixation, and gaze holding remain accurate across different visual and mechanical conditions [108-111,117-119].

Poor fixation suppression and impaired VOR cancellation occur because the floccular complex is a major route through which visual information can modify vestibular reflex output. These two tasks are related but not identical. In fixation suppression, the target is earth-fixed, and the normal response is to damp vestibular nystagmus when a stable visual reference is available. In VOR cancellation, the target moves with the head, so the normal canal-driven counter-rotation of the eyes must be actively suppressed. Classic lesion studies showed that unilateral floccular lesions abolish or markedly reduce visual suppression of caloric or vestibular nystagmus on the side of the lesion, while bilateral lesions cause bilateral loss of suppression [110,111]. The physiology reflects a loss of cerebellar modulation of vestibular premotor circuits. The labyrinth continues to generate vestibular slow-phase eye movements through the vestibular nuclei, but without normal floccular Purkinje-cell inhibition these signals are no longer appropriately calibrated by visual input, so the brainstem circuitry cannot impose the visual correction that normally suppresses or dampens the nystagmus. Lesion studies of the floccular complex, particularly when the ventral paraflocculus is involved, also show linked deficits of smooth pursuit, VOR cancellation, and VOR adaptation [112,113]. These findings illustrate a fundamental principle of vestibulocerebellar physiology: the floccular complex continuously calibrates slow eye-movement systems that rely on accurate velocity signals. When this calibration is lost, several related abnormalities appear together. Visual fixation can no longer suppress vestibular slow phases (poor fixation suppression), vestibular counter-rotation cannot be appropriately gated during head-fixed target tracking (impaired VOR cancellation), visually guided slow eye velocity becomes inaccurate (abnormal smooth pursuit), and the neural integrator becomes unstable, producing gaze-evoked drift (gaze-holding abnormalities). In other words, the flocculus normally ensures that slow eye-movement commands generated in vestibular premotor circuits match behavioural goals and visual context; when floccular output is impaired,

these systems become uncoupled and produce the characteristic cluster of ocular motor findings seen in vestibulocerebellar disease [110-113].

Abnormal pursuit and gaze-holding abnormalities also follow naturally from floccular dysfunction. Purkinje cells in the flocculus are active during fixation, smooth pursuit, optokinetic stimulation, and VOR suppression, so this region contributes directly to generating accurate slow eye velocity [108,109]. When the floccular complex is damaged, pursuit gain falls and the eyes can no longer maintain a smooth slow phase; the patient then uses catch-up saccades, producing saccadic pursuit. Gaze-holding abnormalities arise because the floccular complex also helps tune the common neural integrator. If that cerebellar calibration is lost, the



position command becomes "leaky": after the eyes move eccentrically, the hold signal decays and the eyes drift back toward centre, producing gaze-evoked nystagmus. Clinically, these abnormalities often cluster together. In patient studies, substantial gaze-evoked nystagmus is usually accompanied by pursuit impairment, and human lesion work supports the flocculus as one of the key cerebellar components of the gaze-holding network [114,115].

Consistent with these broader gaze-stabilisation roles, downbeat nystagmus is the signature ocular-motor syndrome of floccular/parafloccular dysfunction. Its slow phase is an upward drift of the eyes, followed by corrective downward quick phases. Two non-exclusive physiological explanations are most useful. The first is a vertical pursuit asymmetry model: most vertical gaze-velocity Purkinje cells in the floccular lobe show a preference for downward pursuit, so floccular damage disproportionately weakens the downward eye-velocity system, leaving a relative upward drift [117,120-122]. Human studies of downbeat nystagmus support this idea: patients show selectively impaired downward pursuit, reduced floccular activation during downward pursuit, and normal subjects show greater floccular activation for downward than upward pursuit [120-122]. The second is a vertical vestibular disinhibition model: floccular output normally modulates vertical vestibular relay pathways, including circuits involving the superior vestibular nucleus and Y-group; when this cerebellar control is lost, upward slow-phase pathways become relatively overactive, again producing an upward drift with corrective downward beats [116-119]. In practice, these mechanisms likely interact, which is why downbeat nystagmus is so often accompanied by impaired pursuit, poor fixation suppression, and gaze-holding failure in floccular disease [116-122].

For vestibular clinicians, the practical localisation rule is this: when poor fixation suppression, impaired VOR cancellation, saccadic pursuit, gaze-evoked nystagmus, and downbeat nystagmus occur together, the vestibulocerebellum—especially the floccular complex—should move high on the differential. That cluster is

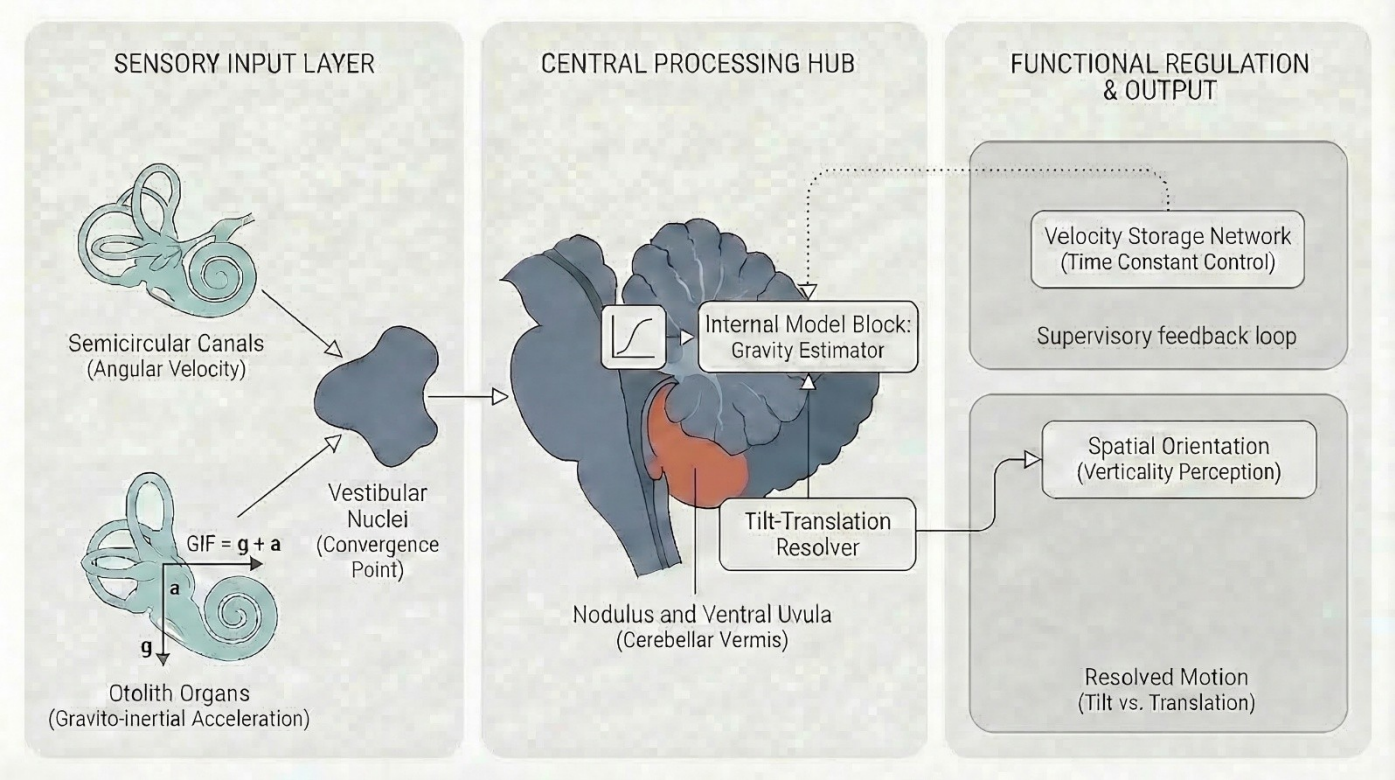
physiologically coherent, not a random bundle of ocular motor signs [110-122]. This floccular role contrasts with that of the nodulus and ventral uvula, which are less concerned with calibrating eye movements and more with ensuring that vestibular signals are interpreted within a gravity-referenced framework and that tilt and translation are correctly distinguished.

Nodulus and Ventral Uvula: Gravito-inertial computation, tilt–translation resolution, and velocity-storage regulation

While the floccular complex is primarily involved in gaze stabilisation and calibration of eye-movement reflexes, the nodulus and ventral uvula of the cerebellar vermis play a complementary role in interpreting vestibular signals within the context of gravity. These structures form a key component of the vestibular cerebellum and receive dense input from both the semicircular canals and the otolith organs via the vestibular nuclei. Their fundamental task is to ensure that the brain's estimate of motion obeys the physical constraints of the real world—particularly the constant presence of gravity. In practical terms, they help the nervous system determine whether a change in otolith signal represents head tilt relative to gravity or actual linear translation through space.

This problem arises because the otolith organs measure **gravito-inertial acceleration**, which combines gravitational acceleration and translational acceleration into a single signal. For example, tilting the head backward and accelerating forward can produce nearly identical otolith activation patterns. Resolving this ambiguity requires combining otolith signals with information from the semicircular canals that report angular velocity. The nodulus and uvula participate in this computation by integrating canal-derived rotation estimates over time to determine the orientation of the head relative to gravity. Once gravity is estimated, it can be mathematically separated from the otolith signal to reveal the translational component of motion. This process is a classic example of an internal model operating within the vestibular cerebellum [62,75].

Physiological and lesion studies show that disruption of the nodulus/uvula alters how the brain interprets motion relative to gravity. Animals with nodular lesions exhibit impaired tilt–translation discrimination, abnormal spatial orientation, and disordered processing of gravito-inertial cues. In humans, dysfunction of these regions is associated with perceptual distortions of verticality, abnormalities of velocity storage, and unusual positional nystagmus patterns. This is because the nodulus and uvula help ensure that stored velocity signals remain aligned with the gravitational vertical. Without this regulation, stored vestibular signals



may persist in an incorrect spatial frame, leading to inappropriate eye movements or altered perception of self-motion.

The nodulus and uvula also interact closely with the **velocity storage network** discussed earlier. Velocity storage extends the duration of canal-derived rotation signals within the vestibular nuclei, improving low-frequency performance of the VOR. However, this persistence must be carefully controlled; otherwise the stored signal can become excessive or spatially misaligned. The nodulus and ventral uvula act as supervisory regulators of this network, adjusting the time constant of storage and ensuring that the stored rotation estimate remains consistent with gravity. Experimental stimulation or lesions of these regions can abruptly terminate velocity storage or alter the axis of nystagmus during prolonged rotation [62,75,76].

Clinically, disorders affecting the nodulus and uvula may present with abnormalities of motion perception rather than purely ocular-motor findings. Patients may report spatial disorientation, unusual motion sensitivity, or exaggerated responses to visual motion. They may also show positional or gravity-dependent nystagmus patterns that do not follow the typical canal-based patterns seen in peripheral vestibular disorders. In this way, nodulus/uvula dysfunction highlights an important theme in vestibular physiology: symptoms may arise not only from faulty sensors but also from incorrect interpretation of otherwise normal sensory signals.

Summary of vestibular cerebellar functions

In simplified terms, the vestibular cerebellum can be viewed as coordinating three related computations: the floccular complex maintains accurate eye stabilisation by calibrating VOR, pursuit, and gaze-holding mechanisms; the nodulus and ventral uvula ensure that vestibular signals are interpreted in a gravity-consistent reference frame and help resolve tilt–translation ambiguity; and the velocity storage network, regulated by these cerebellar regions, extends the persistence of rotational signals so that the brain maintains a coherent estimate of self-motion over time. Together, these mechanisms allow the vestibular system to stabilise vision, correctly interpret motion, and maintain spatial orientation in a gravitational environment.

 Clinical Pearl: Clinically, floccular vs nodular syndromes are distinguishable: floccular lesions cause GEN, poor fixation suppression, saccadic pursuit, and impaired VOR cancellation. Nodular/uvular lesions cause direction-changing positional nystagmus (DCPN) and prolonged post-rotatory nystagmus. DCPN mimics BPPV but is non-geotropic (or geotropic without position preference) and lacks fatigability — always consider MRI if positional nystagmus does not behave as expected for canalithiasis.

7.4 Clinical and Experimental Implications

If the cerebellum is responsible for prediction and error-based recalibration, then cerebellar lesions should produce two broad classes of problems. The first is **impaired prediction or cancellation**. In this situation, patients may become overly sensitive to self-generated motion, may have difficulty suppressing reflexes when context demands it, and may feel particularly unstable in situations that require predictive control—walking while turning, moving through visually busy environments, or making rapid combined eye–head movements. The second is **impaired learning from error**. In this case, VOR gain may fail to recalibrate appropriately, compensation after vestibular injury may be slower or incomplete, and persistent retinal slip may not drive the normal adaptive response [60,68-70].

These general principles help explain several common clinical findings. Floccular dysfunction may produce poor fixation suppression, impaired VOR cancellation, abnormal pursuit, and gaze-evoked nystagmus. Nodulus/uvula dysfunction may produce disordered gravity perception, abnormal velocity storage, positional or motion-induced symptoms that feel disproportionate, and difficulty resolving tilt from translation. This is why cerebellar dizziness should not be reduced to “ataxia plus nystagmus.” In many patients, the sensory organs themselves are still working reasonably well. The problem is that the brain’s internal estimate of what those signals mean has become unreliable.

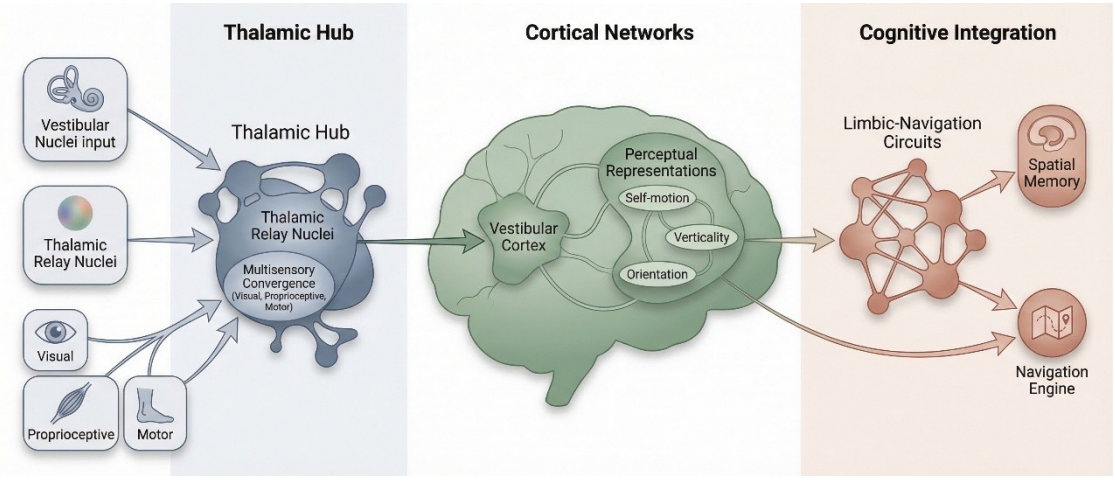
That idea is worth holding onto, because it captures the cerebellum's place in vestibular physiology. The cerebellum is not an accessory added onto the vestibular system after the important work is done. It is one of the main reasons the vestibular system works at all in the real world—where movements are self-generated, sensors are noisy, gravity is constant, and the brain must continually decide what is expected, what is unexpected, and what needs to be corrected next.

Key Facts: Vestibular Cerebellar Modules

Region	Primary Function	Lesion Sign
Flocculus / paraflocculus	VOR gain, cancellation, pursuit, gaze holding	Poor fixation suppression; saccadic pursuit; GEN; impaired VOR cancellation
Nodulus / ventral uvula	Velocity storage; tilt–translation disambiguation	Direction-changing positional nystagmus; prolonged post-rotatory nystagmus

8. Thalamocortical Pathways and Spatial Cognition

Vestibular information does not end at the brainstem reflex circuits. Beyond stabilising the eyes, head, posture, and autonomic responses, vestibular signals also contribute to **perception of self-motion, upright orientation, body-in-space awareness, and navigation.** These higher functions depend on ascending pathways that link the vestibular nuclei with the thalamus, distributed cortical networks, and ultimately limbic–navigation circuits. Unlike the three-neuron reflex arcs, this ascending system is not a single labelled "vestibular tract." Instead, it is a distributed vestibulo–thalamo–cortical network in which vestibular signals are progressively integrated with visual, proprioceptive, and motor-related information. In practical terms, this network helps the brain solve a fundamental problem: whether motion reflects movement of the head in space, movement of the body under the head, or movement of the external visual world [77-79].

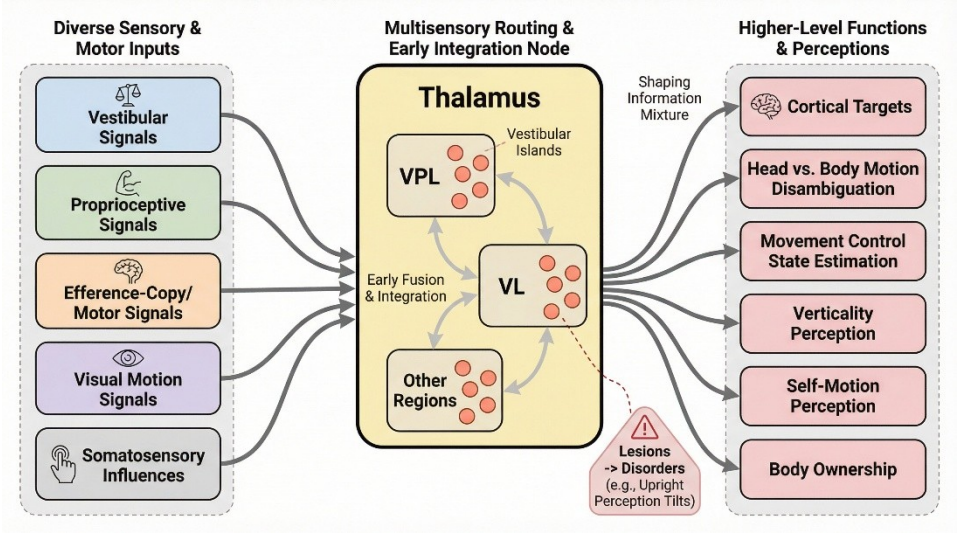


A useful way to organise this material is to begin with the thalamus and then move to the cortex. The thalamus is the principal diencephalic relay through which vestibular brainstem signals are routed toward higher centres, but in vestibular physiology it does more than simply relay them. It acts as an early multisensory integration hub. The cortex, in turn, transforms these already-integrated signals into perceptual representations of self-motion, verticality, and body orientation, and these cortical representations are then used by still higher cognitive networks for navigation and spatial memory.

8.1 The Vestibular Thalamus

The thalamus is the major relay nucleus between subcortical sensory systems and the cerebral cortex. In vestibular physiology, however, it is better understood as a distributed integration and relay station rather than as a single dedicated "vestibular thalamic nucleus." Vestibular afferent information reaches the thalamus primarily from the vestibular nuclei through ascending vestibulothalamic projections—especially from the superior and medial vestibular nuclei, travelling largely in the ipsilateral vestibulothalamic tract within the

brainstem tegmentum/medial lemniscus—and is distributed across several thalamic territories, including parts of the ventral posterior complex, particularly ventroposterior inferior and neighbouring ventral posterolateral regions, as well as ventrolateral and more posterior thalamic territories [78,80,84]. Rather than forming one compact vestibular nucleus, vestibular-responsive neurons appear in clusters embedded within thalamic regions better known for somatosensory and motor functions.

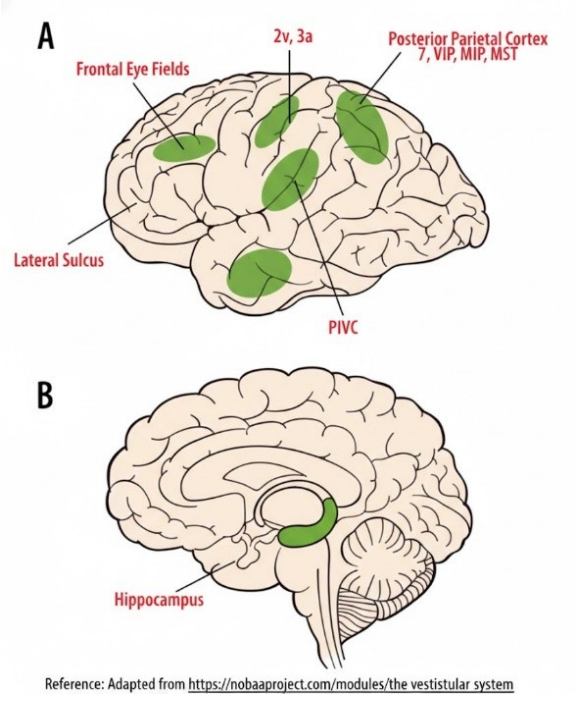


This anatomical arrangement is physiologically important. By placing vestibular signals alongside proprioceptive and motor-related information, the thalamus is positioned to begin the process of distinguishing head motion from whole-body motion and self-motion from externally imposed motion. Thalamic vestibular neurons commonly show convergence of vestibular, visual, somatosensory, and motor-related signals. As a result, the thalamus shapes what kind of vestibular information reaches cortex. It is not merely passing on a "sense of motion," but weighting and combining vestibular input with other signals relevant to orientation, verticality, and body schema [78,84].

Clinically, this means that thalamic vestibular dysfunction can produce symptoms that are clearly vestibular in flavour but do not resemble peripheral labyrinthine syndromes. Lesions involving thalamic vestibular territories may produce tilt of subjective visual vertical, postural lateropulsion, altered perception of upright, and spatial disorientation that cannot be neatly explained by a single canal or otolith lesion [78,79]. The practical teaching point is that the thalamus represents the first major stage at which vestibular disorders may become disorders of interpretation rather than disorders of sensing.

8.2 Cortical Vestibular Pathways

At the cortical level, vestibular processing is best understood as a network rather than a single "vestibular cortex." The best-established core hub is the parieto-insular vestibular cortex (PIVC), located in the posterior insular–parietal opercular region. In humans, closely related neighbouring regions such as the posterior insular cortex and parietal operculum are also heavily involved, and more broadly the vestibular cortical network extends into the temporo-parietal junction and posterior parietal cortex [81,82]. These areas receive vestibular information through thalamic relays and combine it with visual motion, somatosensory, and proprioceptive signals.

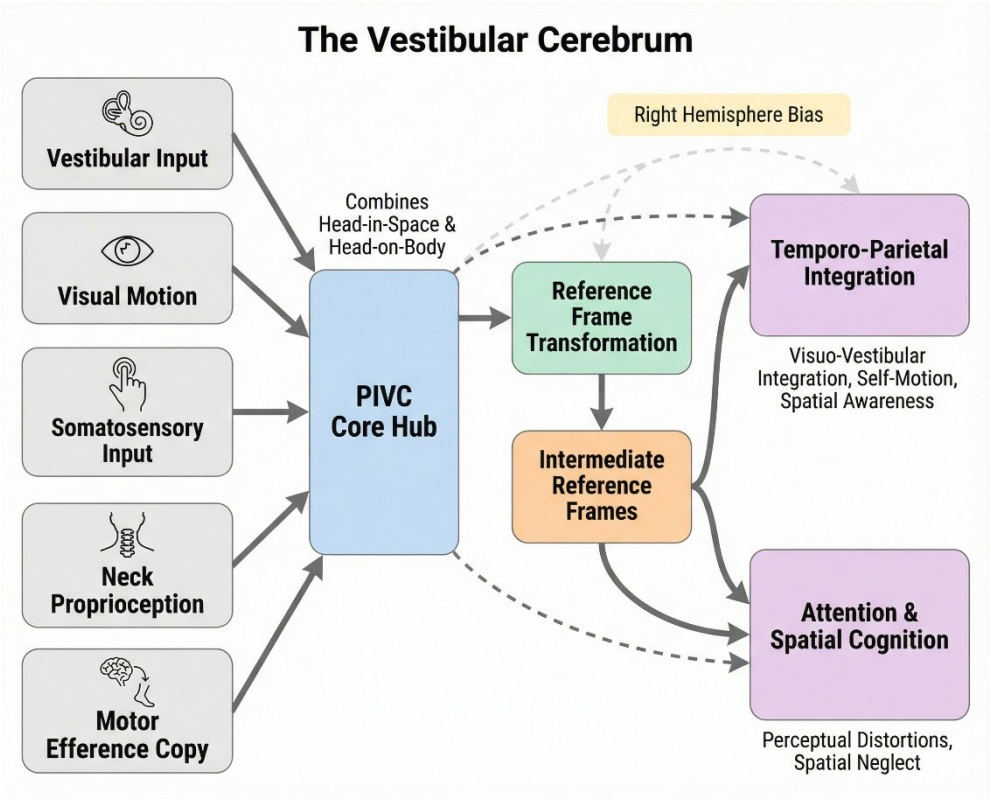


The physiology of this cortical system is fundamentally multisensory and reference-frame dependent. Cortical vestibular neurons do not simply respond to "vestibular on" or "vestibular off." Instead, they combine head-in-space vestibular information with neck proprioception, body-centred somatosensory input, and visual motion to generate a more useful estimate of body motion in space. In other words, the cortex performs reference-frame transformations: it converts head-centred vestibular signals into perceptual representations that are useful for orientation, self-motion

perception, and interaction with the environment [81,82]. This helps explain why cortical vestibular processing is so important for judging verticality, interpreting motion in visually complex settings, and distinguishing self-motion from motion of the external world.

 Clinical Pearl: SVV tilt from cortical lesions occurs because PIVC and PIC integrate utricular and visual vertical signals to build a gravitational reference frame. Right hemisphere dominance in vestibular cortex means right-sided parieto-insular strokes disproportionately disrupt SVV and spatial orientation. Pusher syndrome (lateral postural instability after stroke) involves posterior parietal cortex disruption of body-verticality estimation.

Clinically, cortical vestibular dysfunction may produce symptoms that look vestibular but are more perceptual than reflexive. Patients may describe visual motion intolerance, disturbed verticality, altered body-in-space perception, or difficulty deciding whether they are moving or whether the environment is moving, particularly in visually busy scenes. Importantly, short-latency peripheral tests such as vHIT or calorics may be normal, because the disturbance lies upstream of the labyrinth and brainstem reflex arcs. Human lesion and imaging studies also suggest hemispheric asymmetry, with right temporo-parietal networks particularly relevant to spatial awareness. This helps explain why cortical vestibular syndromes can include neglect-like features, disturbed sense of upright, or impaired spatial attention rather than only vertigo [77,79,81-83].



8.3 Spatial Navigation: Head Direction, Grid Coding, and Path Integration

Vestibular signals contribute to spatial navigation by providing an internal estimate of self-motion, which is especially important when visual landmarks are sparse or unreliable. This is seen first in the head-direction system, which acts as an internal compass and depends on vestibular angular-velocity input to keep directional coding stable through circuits involving the dorsal tegmental nucleus, lateral mammillary nucleus, and anterior thalamus [83-85]. Further upstream, grid cells in the medial entorhinal cortex use this self-motion information, together with proprioceptive and visual cues, to support path integration and internal mapping of position [86,87]. Clinically, vestibular dysfunction can therefore impair navigation even when reflex testing is relatively preserved, leading to difficulty orienting in darkness, uncertainty in unfamiliar environments, and reduced confidence in spatial mapping when visual cues are removed [83-87].

Key Facts: Vestibular Cortical Pathway

Region	Function	Lesion Sign
Vestibular thalamus (VPLc/VPI)	Vestibular relay to cortex	Mild SVV tilt; spatial disorientation
PIVC / PIC	Graviceptive reference frame	SVV tilt; poor righting

Region	Function	Lesion Sign
Posterior parietal cortex	Multisensory spatial integration	Pusher syndrome; spatial neglect
Hippocampus (via entorhinal)	Cognitive mapping; path integration	Poor navigation; hippocampal atrophy in chronic VH

9. Physiological basis of recovery and rehabilitation

Vestibular symptoms after injury are not simply the passive consequence of a damaged sensor; they evolve because the CNS re-optimises its internal estimates and motor outputs under new constraints. The physiology of recovery therefore sits at the intersection of vestibular compensation, sensory reweighting, and motor learning. Vestibular rehabilitation therapy (VRT) operationalises these principles by using structured exposure and task-driven error to reshape reflex gain, improve multisensory integration, and reduce symptom amplification over time. [8,88]

9.1 Adaptation

Adaptation refers to genuine recalibration of vestibular reflexes, most familiarly the ability of the VOR to change gain when the relationship between head motion and retinal slip is altered. At a mechanistic level, this is cerebellar learning applied to a control problem: retinal slip (and related error signals) drive changes in the transformation from vestibular input to motor output so that gaze stabilisation becomes accurate again. Clinically, gaze-stabilisation exercises (for example, VOR x1 and VOR x2 paradigms) are designed to create controlled retinal-slip error during active head movement, thereby engaging the same adaptation machinery that calibrates the VOR throughout life. [8,88]

9.2 Substitution

Substitution describes the strategy of achieving functional stability by recruiting non-vestibular signals or alternative ocular motor strategies when vestibular information is unreliable or absent. At the systems level, this is sensory reweighting: vision, proprioception, and efference copy can be upweighted to support posture and navigation, while predictive, feedforward eye-movement strategies (for example, pre-programmed saccades or increased reliance on smooth pursuit/optokinetic cues) can partially replace a deficient canal-driven response. The same computational theme that appears in central vestibular physiology—combining cues to form a best estimate of self-motion—reappears here as the brain learns which cues are still trustworthy in different contexts. [1,8,88]

9.3 Habituation

Habituation refers to a reduction in symptom response (dizziness, motion sensitivity, visually induced discomfort) with repeated exposure to provocative stimuli. Physiologically, habituation is not “getting used to it” in a vague sense; it reflects central recalibration of stimulus salience and prediction error, so that repeated, non-threatening sensory conflicts generate progressively smaller autonomic and perceptual responses. In practice, graded exposure to motion, visual complexity, and positional triggers is used to drive this down-regulation while avoiding avoidance learning, which otherwise reinforces symptom sensitivity. [8,88]

 Clinical Pearl: Match the rehabilitation strategy to the mechanism: ADAPTATION (gaze stabilisation exercises) for patients with peripheral hypofunction and an active VOR error signal; SUBSTITUTION (somatosensory/visual reliance training) when adaptation is impossible (bilateral loss, cerebellar disease); HABITUATION (Brandt-Daroff, positional manoeuvres) for motion-triggered symptoms. Forcing adaptation training in a fully compensated patient without residual VOR asymmetry achieves little. Re-assess VOR gain before designing exercises.

Conclusion The physiology of the vestibular system is defined by its speed, precision, and extensive integration. From the molecular gating of TMC channels to the infinite-memory line attractors of the neural integrator, and the Bayesian computations of the cortex, the system operates across multiple levels of complexity. Decoding these algorithms offers the key to treating balance disorders and provides a window into the fundamental computational principles of the brain.

Reference List

1. Angelaki DE, Cullen KE. The vestibular system: multimodal integration and encoding of self-motion for motor control. Annu Rev Neurosci. 2008;31:125-50.

2. Jones GM, Spells KE. A theoretical and comparative study of the functional dependence of the semicircular canal upon its physical dimensions. Proc R Soc Lond B Biol Sci. 1963;157(968):403-19.

3. Muller M. Semicircular canal geometry, afferent sensitivity and animal behavior. Lab Anim (NY). 2002;31(2):32-6.

4. Steinhausen W. Uber die Beobachtung der Cupula in den Bogengangsampullen des Labyrinths des lebenden Hechts. Pflugers Arch Gesamte Physiol Menschen Tiere. 1933;232:500-12.

5. Lim DJ. Derivation of the Torsion-Pendulum Model. In: The Semicircular Canals. Oxford: Oxford University Press; 2019.

6. Kim J, Curthoys IS. Models for response dynamics of vestibular end organs: a review. J Vestib Res. 2004;14(1):1-12.

7. Leigh RJ, Zee DS. The Neurology of Eye Movements. 5th ed. Oxford University Press; 2015.

8. Herdman SJ, Clendaniel RA, editors. Vestibular Rehabilitation. 4th ed. Philadelphia: F.A. Davis; 2014.

9. Uchino Y, Kushiro K. The Anatomical and Physiological Basis of Clinical Tests of Otolith Function. A Tribute to Yoshio Uchino. Front Neurol. 2011;2:43.

10. Kondrachuk AV. Otolith responses to dynamical stimuli: Results of a numerical investigation. J Vestib Res. 2001;11(5):317-29.

11. Curthoys IS, Grant JW. A review of mechanical and synaptic processes in otolith transduction of sound and vibration for clinical VEMP testing. J Neurophysiol. 2015;114(6):3010-31.

12. Pan B, Geleoc GS, Asai Y, et al. TMC1 and TMC2 are components of the mechanotransduction channel in the auditory and vestibular hair cells of mice. Neuron. 2013;79(3):504-15.

13. Jia S, He DZ. Regulation of membrane homeostasis by TMC1 mechanoelectrical transduction channels is essential for hearing. bioRxiv [Preprint]. 2023.

14. Wu Z, Müller U. Molecular identity of the mechanotransduction channel in hair cells: not quiet there yet. J Neurosci. 2016 Oct 26;36(43):10927-10934. doi:10.1523/JNEUROSCI.1149-16.2016.

15. Eatock RA, Corey DP, Sperling MA. Two mechanisms for transducer adaptation in vertebrate hair cells. Proc Natl Acad Sci U S A. 1987;84(24):8991-5.

16. Holt JR, Gillespie PG, Eatock RA, Corey DP. Mechanotransduction in Mammalian Sensory Hair Cells. In: The Senses: A Comprehensive Reference. Elsevier; 2008.

17. Ricci AJ, Wu YC, Fettiplace R. Variations in the ensemble of potassium currents underlying resonance in turtle hair cells. J Physiol. 2000;524(2):423-42.

18. Spaiardi P, Toselli M, Masetto S. Currents in Vestibular Type I and Type II Hair Cells of the Embryo. J Physiol. 2017;595(21):6725-43.

19. Masetto S, Correia MJ. Studies of ionic currents in the isolated vestibular hair cell of the chick. J Neurophysiol. 1997;78(3):1413-24.

20. Spitzmaul G, Lagostena L, Knirsch M, et al. KCNQ4 potassium channels are crucial for the distinctive outwardly rectifying conductances of type I and II vestibular hair cells. bioRxiv [Preprint]. 2023.

21. Dierich M, Altoè A, Kros CJ, et al. The potassium channel subunit KV1.8 (Kcna10) is essential for the distinctive outwardly rectifying conductances of type I and II vestibular hair cells. PMC. 2020.

22. Caprara G, Peng A. Mechanotransduction in mammalian sensory hair cells. Mol Cell Neurosci. 2022;120:103706. doi:10.1016/j.mcn.2022.103706.

23. Vincent PF, Bouleau Y, Safieddine S, et al. Exocytotic Machineries of Vestibular Type I and Cochlear Ribbon Synapses Display Similar Intrinsic Otoferlin-Dependent Ca2+ Sensitivity But a Different Coupling to Ca2+ Channels. Front Cell Neurosci. 2014;8:257.

24. Roux I, Safieddine S, Nouvian R, et al. Otoferlin acts as a Ca2+ sensor for vesicle fusion and vesicle pool replenishment at auditory hair cell ribbon synapses. Cell. 2006;127(2):277-89.

25. Michalski N, Goutman JD, Auclair SM, et al. Otoferlin as a multirole Ca2+ signaling protein: from inner ear synapses to cancer pathways. Front Cell Neurosci. 2017;11:37.

26. Goldberg JM. Signal transmission in mature mammalian vestibular hair cells. Front Cell Neurosci. 2012;6:18.

27. Songer JE, Eatock RA. Simultaneous recordings from vestibular Type I hair cells and their calyx afferents. Front Cell Neurosci. 2013;7:143.

28. Sadeghi SG, Minor LB, Cullen KE. Models of vestibular semicircular canal afferent neuron firing activity. J Vestib Res. 2007;17(4):169-81.

29. Meredith FL, Rennie KJ. KCNQ2/3 regulates efferent mediated slow excitation of vestibular afferents in mammals. bioRxiv [Preprint]. 2020.

30. Hullar TE, Minor LB. Determinants of Spatial and Temporal Coding by Semicircular Canal Afferents. J Neurophysiol. 1999;82(6):3310-21.

31. Goldberg JM, Fernández C. Variation in response dynamics of regular and irregular vestibular-nerve afferents during sinusoidal head rotations and currents in the chinchilla. J Neurophysiol. 1971;34(4):676-84.

32. Kim J, Curthoys IS. The Neural Basis for Biased Behavioral Responses Evoked by Galvanic Vestibular Stimulation in Primates. J Neurosci. 2004;24(1):123-30.

33. Hullar TE, Della Santina CC, Hirvonen T, et al. Responses of Irregularly Discharging Chinchilla Semicircular Canal Vestibular-Nerve Afferents During High-Frequency Head Rotations. J Neurophysiol. 2005;93(5):2777-86.

34. Sadeghi SG, Chacron MJ, Taylor MC, Cullen KE. Neural Variability, Detection Thresholds, and Information Transmission in the Vestibular System. J Neurosci. 2007;27(4):771-81.

35. Cullen KE. The vestibular system: multimodal integration and encoding of self-motion for motor control. PMC. 2012.

36. Zee DS. Role of neural integrators in oculomotor systems: a systematic narrative literature review. J Med Life. 2015;8(4):423-9.

37. Robinson DA. The oculomotor system as an example of an integrator. In: The Visual System: Neurophysiology and Psychophysics. 1989.

38. Crawford JD, Cadera W, Vilis T. Generation of torsional and vertical eye position signals by the interstitial nucleus of Cajal. Science. 1991;252(5012):1551-1553. doi:10.1126/science.2047862.

39. Scudder CA, Fuchs AF. Properties of superior vestibular nucleus flocculus target neurons in the squirrel monkey. I. General properties in comparison with flocculus projecting neurons. J Neurophysiol. 1992;68(2):431-51.

40. Zhang X, de Waele C, Baird RA, et al. Role of the flocculus in mediating vestibular nucleus neuron plasticity during vestibular compensation in the rat. J Physiol. 2005;566(1):257-71.

41. Browne L, Smith KE, Jagger DJ. Persistent and resurgent Na+ currents in vestibular calyx afferents. J Gen Physiol. 2017;149(11):1021-39.

42. Patel RR, Barbosa C, Brustovetsky T, et al. Human Nav1.6 Channels Generate Larger Resurgent Currents than Human Nav1.1 Channels. PLOS One. 2015;10(3):e0119463.

43. Beraneck M, Idoux E. Intrinsic Firing Dynamics of Vestibular Nucleus Neurons. Front Neurol. 2012;3:131.

44. Kodama T, Gittis AH, Shin M, et al. Transgenic Mouse Lines Subdivide Medial Vestibular Nucleus Neurons into Discrete, Neurochemically Distinct Populations. J Neurosci. 2012;32(41):14142-53.

45. Smith PF, Darlington CL. Neuropharmacology of Vestibular System Disorders. Front Neurol. 2013;4:159.

46. Yates BJ, Miller AD, Lucot JB. The neural basis of motion sickness. J Neurophysiol. 1998;79(2):453-62.

47. Lacour M, Sterkers O. Histamine and betahistine in the treatment of vertigo: elucidation of mechanisms of action. CNS Drugs. 2001;15(11):853-70.

48. von Gersdorff H, Iversen M, Rabbitt R. Keeping your eye on the ball. J Physiol. 2020;598. doi:10.1113/JP279149.

49. Wong AMF. Understanding skew deviation and a new clinical test to differentiate it from trochlear nerve palsy. J AAPOS. 2010;14(1):61-67. doi:10.1016/j.jaapos.2009.11.019.

50. Laurens J, Angelaki DE. Coding of Velocity Storage in the Vestibular Nuclei. Front Neurol. 2018;9:351.

51. Raphan T, Cohen B, Precht W. Mechanism of vOR velocity storage in the cat. J Neurophysiol. 1979;42(4):1020–1034.

52. Marsella P, Gupta A, Palla A, et al. On labyrinthine function loss, motion sickness immunity, and velocity storage. Front Neurol. 2024;15:1426213.

53. Waespe W, Cohen B, Raphan T. Stimulation of the nodulus and uvula discharges velocity storage in the vestibulo-ocular reflex. Exp Brain Res. 1985;59(3):444-62.

54. Bockisch CJ, Hegemann SC. Alexander's law and the oculomotor neural integrator: three-dimensional eye velocity in patients with an acute vestibular asymmetry. J Neurophysiol. 2008;100(6):3105-3116. doi:10.1152/jn.90381.2008.

55. Yates BJ. Descending Influences on Vestibulospinal and Vestibulosympathetic Reflexes. Front Neurol. 2014;5:224.

56. ScienceDirect Topic. Lateral Vestibulospinal Tract. [Reference to definition and anatomy of LVST].

57. ScienceDirect Topic. Medial Vestibulospinal Tract. [Reference to MVST anatomy].

58. Ray CA. Vestibulo-sympathetic reflex during orthostatic challenge in aging humans. J Appl Physiol. 2002;92(3):1160-4.

59. Balaban CD. Vestibular autonomic regulation (including motion sickness and mechanism of vomiting). Curr Opin Neurol. 1999;12(1):29–33.

60. Cullen KE. Internal models of self-motion: neural computations by the vestibular system. Nat Rev Neurosci. 2023.

61. Laurens J, Droulez J. A unified internal model theory to resolve the paradox of active versus passive self-motion sensation. eLife. 2017;6:e28074.

62. Angelaki DE, Yakusheva TA. How vestibular neurons solve the tilt/translation ambiguity. Ann N Y Acad Sci. 2009.

63. Green AM, Angelaki DE. Internal models and neural computation in the vestibular system. Exp Brain Res. 2010.

64. MacNeilage PR, Turner AH, Angelaki DE. Computational approaches to spatial orientation. J Neurophysiol. 2008.

65. Laurens J. The otolith vermis: a systems neuroscience theory of nodulus and ventral uvula function. Front Syst Neurosci. 2022.

66. Roy JE, Cullen KE. Dissociating self-generated from passively applied head motion: neural mechanisms in the vestibular nuclei. J Neurosci. 2004;24(9):2102–2111.

67. Brooks JX, Cullen KE. Early vestibular processing does not discriminate active from passive head movement if there is a discrepancy between predicted and actual sensory consequences. J Neurophysiol. 2014.

68. Cullen KE. Neural correlates of sensory prediction errors in monkeys. Curr Opin Neurobiol. 2015.

69. Inoshita T, Hirano T. Occurrence of long-term depression in the cerebellar flocculus during oculomotor learning. eLife. 2018;7:e36209.

70. Schonewille M, et al. Reevaluating the role of LTD in cerebellar motor learning. Neuron. 2011.

71. Hirano T. Long-term depression and other synaptic plasticity in the cerebellum. Proc Jpn Acad Ser B. 2013;89:183–195.

72. Jörntell H, Hansel C. Synaptic memories upside down: bidirectional plasticity at cerebellar parallel fiber–Purkinje cell synapses. Neuron. 2006.

73. Ito M. Cerebellar long-term depression: characterization, signal transduction, and functional roles. Physiol Rev. 2001;81(3):1143–1195.

74. Rondi-Reig L, et al. How the cerebellum may monitor sensory information for spatial representation. Front Syst Neurosci. 2014.

75. Yakusheva TA, et al. Spatiotemporal properties of optic flow and vestibular translation responsiveness in nodulus/uvula. J Neurosci. 2013.

76. Mackrous I, et al. Cerebellar prediction of the dynamic sensory consequences of gravity during self-motion. Curr Biol. 2019.

77. Zwergal A, Grabova D, Schöberl F. Vestibular contribution to spatial orientation and navigation. Curr Opin Neurol. 2024;37(1):52-58.

78. Wijesinghe R, Protti DA, Camp AJ. Vestibular interactions in the thalamus. Front Neural Circuits. 2015;9:79.

79. Dieterich M, Kirsch V, Brandt T. Perception of verticality and vestibular disorders of balance and falls. Front Neurol. 2019;10:172.

80. Deecke L, Schwarz DW, Fredrickson JM. Nucleus ventroposterior inferior (VPI) as the vestibular thalamic relay in the rhesus monkey. Exp Brain Res. 1974;20(1):88-100.

81. Chen A, DeAngelis GC, Angelaki DE. Macaque parieto-insular vestibular cortex: responses to self-motion and optic flow. J Neurosci. 2010;30(8):3022-3042.

82. Frank SM, Greenlee MW. The parieto-insular vestibular cortex in humans: more than a single area? J Neurophysiol. 2018;120(3):1438-1450.

83. Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. Proc Natl Acad Sci U S A. 2000;97(25):13931-13936.

84. Meng H, May PJ, Dickman JD, Angelaki DE. Vestibular signals in primate thalamus: properties and origins. J Neurosci. 2007;27(50):13590-13602.

85. Blair HT, Cho J, Sharp PE. The anterior thalamic head-direction signal is abolished by bilateral lesions of the lateral mammillary nucleus. J Neurosci. 1999;19(15):6698-6711.

86. Jacob PY, Casali G, Spieser L, Page H, Overington D, Jeffery K. An independent, landmark-dominated head-direction signal in dysgranular retrosplenial cortex. Nat Neurosci. 2017;20(2):173-175.

87. Chen G, Manson D, Cacucci F, Wills TJ. Absence of visual input results in the disruption of grid cell firing in the mouse. Curr Biol. 2016;26(17):2335-2342.

88. Hall CD, Herdman SJ, Whitney SL, et al. Vestibular rehabilitation for peripheral vestibular hypofunction: an updated clinical practice guideline from the Academy of Neurologic Physical Therapy of the American Physical Therapy Association. J Neurol Phys Ther. 2022;46(2):118-177. doi:10.1097/NPT.0000000000000382.

89. Yu Z, McIntosh JM, Sadeghi SG, Glowatzki E. Efferent synaptic transmission at the vestibular type II hair cell synapse. J Neurophysiol. 2020;124(2):360-374. doi:10.1152/jn.00143.2020.

90. Mathews MA, Camp AJ, Murray AJ. Reviewing the role of the efferent vestibular system in motor and vestibular circuits. Front Physiol. 2017;8:552. doi:10.3389/fphys.2017.00552.

91. Wilson VJ, Boyle R, Fukushima K, Rose PK, Shinoda Y, Sugiuchi Y, et al. The vestibulocollic reflex. J Vestib Res. 1995;5(3):147-70.

92. Goldberg JM, Cullen KE. Vestibular control of the head: possible functions of the vestibulocollic reflex. Exp Brain Res. 2011;210(3-4):331-45.

93. Boyle R. Medial and lateral vestibulospinal projections to the cervical spinal cord of the squirrel monkey. Front Neurol. 2025;15:1513132.

94. Curthoys IS, Vulovic V, Manzari L, Burgess AM. The neural basis of vestibular evoked myogenic potentials. The cVEMP is a specific indicator of saccular function. Front Neurol. 2025;16:1644120.

95. Kjærsgaard JB, Hougaard DD, Kingma H. Thirty years with cervical vestibular myogenic potentials: a critical review on its origin. Front Neurol. 2025;15:1502093.

96. Markham CH. Vestibular control of muscular tone and posture. Can J Neurol Sci. 1987;14(3 Suppl):493-6.

97. McCall AA, Miller DM, Yates BJ. Descending influences on vestibulospinal and vestibulosympathetic reflexes. Front Neurol. 2017;8:112.

98. Boyle R. Medial and lateral vestibulospinal projections to the cervical spinal cord of the squirrel monkey. Front Neurol. 2025;15:1513132.

99. Mildren RL, Cullen KE. Sensorimotor transformations for postural control in the vermis of the cerebellum. J Neurosci. 2025;45(21):e0249252025.

100. Jecko V, Garcia L, Doat E, Leconte V, Liguoro D, Cazalets JR, et al. Vestibulospinal reflexes elicited with a tone burst method are dependent on spatial orientation. PeerJ. 2024;12:e17056.

101. Marchand S, McLaren R, Dakin CJ. A wide-ranging review of galvanic vestibular stimulation: from its genesis to basic science and clinical applications. J Neurophysiol. 2025.

102. Yates BJ, Bolton PS, Macefield VG. Vestibulo-sympathetic responses. Compr Physiol. 2014;4(2):851-87.

103. McCall AA, Miller DM, Yates BJ. Descending influences on vestibulospinal and vestibulosympathetic reflexes. Front Neurol. 2017;8:112.

104. Sauder CL, Ray CA. Greater sensitivity of the vestibulosympathetic reflex in the upright posture in humans. J Appl Physiol (1985). 2008;105(1):65-69.

105. Holstein GR, Friedrich VL Jr, Martinelli GP. Projection neurons of the vestibulo-sympathetic reflex pathway. J Comp Neurol. 2014;522(9):2053-74.

106. Sugiyama Y, Suzuki T, Yates BJ. Role of the rostral ventrolateral medulla in the vestibulosympathetic reflex in rats. Neurosci Lett. 2011;487(3):300-304.

107. Bogle JM, Benarroch E, Sandroni P. Vestibular-autonomic interactions: beyond orthostatic dizziness. Curr Opin Neurol. 2022;35(1):126-134.

108. Noda H, Suzuki DA. The role of the flocculus of the monkey in fixation and smooth pursuit eye movements. J Physiol. 1979;294:335-348.

109. Büttner U, Waespe W. Purkinje cell activity in the primate flocculus during optokinetic stimulation, smooth pursuit eye movements and VOR-suppression. Exp Brain Res. 1984;55(1):97-104.

110. Takemori S, Cohen B. Loss of visual suppression of vestibular nystagmus after flocculus lesions. Brain Res. 1974;72(2):213-224.

111. Takemori S. Visual suppression of vestibular nystagmus after cerebellar lesions. Ann Otol Rhinol Laryngol. 1975;84(3 Pt 1):318-326.

112. Cullen KE, Belton T, McCrea RA. A non-visual mechanism for voluntary cancellation of the vestibulo-ocular reflex. Exp Brain Res. 1991;83(2):237-252.

113. Rambold H, Churchland A, Selig Y, Jasmin L, Lisberger SG. Partial ablations of the flocculus and ventral paraflocculus in monkeys cause linked deficits in smooth pursuit eye movements and adaptive modification of the VOR. J Neurophysiol. 2002;87(2):912-924.

114. Büttner U, Grundei T. Gaze-evoked nystagmus and smooth pursuit deficits: their relationship studied in 52 patients. J Neurol. 1995;242(6):384-389.

115. Baier B, Dieterich M. Incidence and anatomy of gaze-evoked nystagmus in patients with cerebellar lesions. Neurology. 2011;76(4):361-365.

116. Zee DS, Yamazaki A, Butler PH, Gücer G. Effects of ablation of flocculus and paraflocculus on eye movements in primate. J Neurophysiol. 1981;46(4):878-899.

117. Fukushima K, Fukushima J, Kaneko CRS, Fuchs AF. Vertical Purkinje cells of the monkey floccular lobe: simple-spike activity during pursuit and passive whole body rotation. J Neurophysiol. 1999;82(2):787-803.

118. Sato Y, Kawasaki T. Operational unit responsible for plane-specific control of eye movement by cerebellar flocculus in cat. J Neurophysiol. 1990;64(2):551-564.

119. Blazquez P, Partsalis AM, Gerrits NM, Highstein SM. Input of anterior and posterior semicircular canal interneurons encoding head-velocity to the dorsal Y group of the vestibular nuclei. J Neurophysiol. 2000;83(5):2891-2904.

120. Glasauer S, Hoshi M, Büttner U. Smooth pursuit in patients with downbeat nystagmus. Ann N Y Acad Sci. 2005;1039:532-535.

121. Kalla R, Deutschlander A, Hüfner K, Stephan T, Jahn K, Glasauer S, Brandt T, Strupp M. Detection of floccular hypometabolism in downbeat nystagmus by fMRI. Neurology. 2006;66(2):281-283.

122. Glasauer S, Stephan T, Kalla R, Marti S, Straumann D. Up-down asymmetry of cerebellar activation during vertical pursuit eye movements. Cerebellum. 2009;8:385-388.

Disclaimer

This document is intended for educational purposes only and is directed at qualified health professionals, including general practitioners, physiotherapists, and clinicians with an interest in vestibular medicine. It is not intended for use by patients or members of the general public, and does not constitute medical advice, clinical guidelines, or a substitute for professional clinical judgement.

The physiological descriptions, clinical frameworks, and reflex summaries presented here are based on published peer-reviewed literature, as cited in the Reference List. They reflect current understanding at the time of writing and may not incorporate the most recent developments in vestibular medicine. Clinicians should exercise independent professional judgement and consult current guidelines and primary literature when making clinical decisions.

Australian Dizziness Clinics accepts no liability for any clinical decisions made on the basis of information contained in this document.

Copyright