THE VESTIBULAR SYSTEM

VOLUME I

Functional Anatomy and Physiology

A first-principles guide for otolaryngology residents

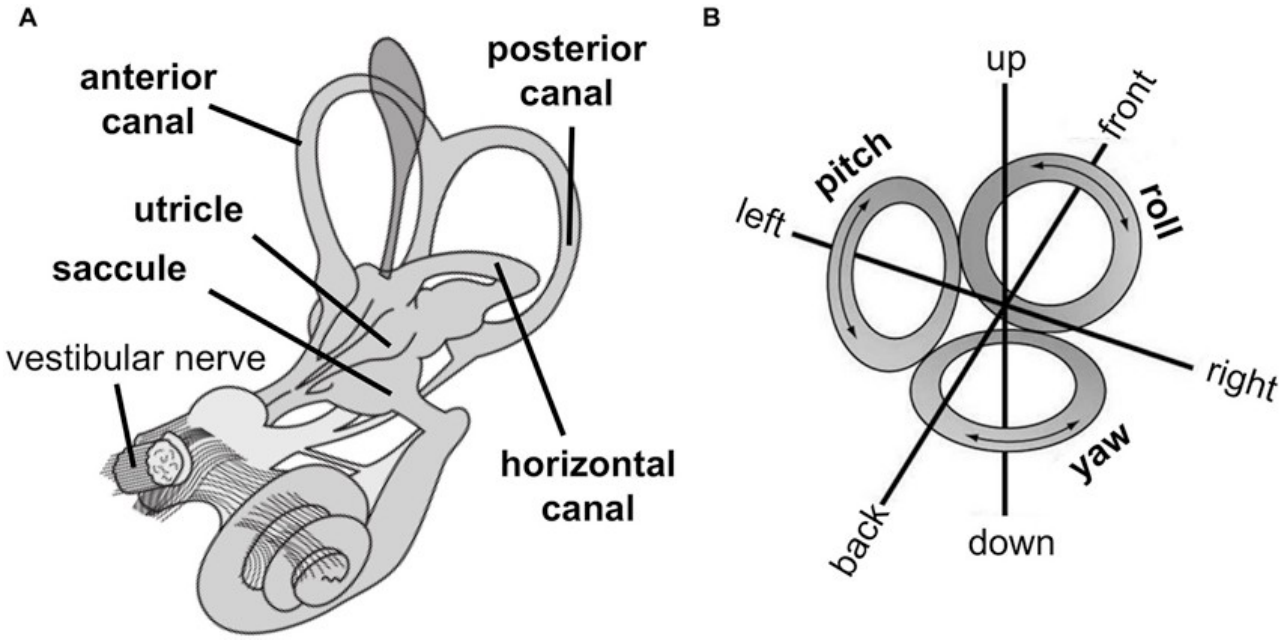

Figure 1. Peripheral vestibular organs and their relationship to the three axes of head rotation. Pfeiffer, Serino, and Blanke; CC BY 3.0.

Resident study guide • July 2026

START WITH A MODEL, NOT A LIST

ORIENTATION. How to use this volume

Vestibular physiology becomes difficult when it is taught as a collection of disconnected rules: ampullopetal versus ampullofugal flow, utricle versus saccule, fast phase versus slow phase, or flocculus versus nodulus. Those rules become much easier to retrieve when they are attached to a single causal story. This volume therefore follows a head movement from the physical world, through the labyrinth and vestibular nerve, into central computations, and finally out to the eyes, neck, body, and conscious perception.

The aim is not to turn a resident into a vestibular neuroscientist. It is to build enough mechanistic understanding that common findings become predictable. By the end, an abnormal head impulse, a direction of nystagmus, a mismatch between calorics and vHIT, or a patient's oscillopsia should feel like consequences of a system rather than isolated facts.

THE CENTRAL QUESTION

At every stage ask: **What physical variable is being represented, in which coordinate frame, and what comparison allows the brain to infer motion?**

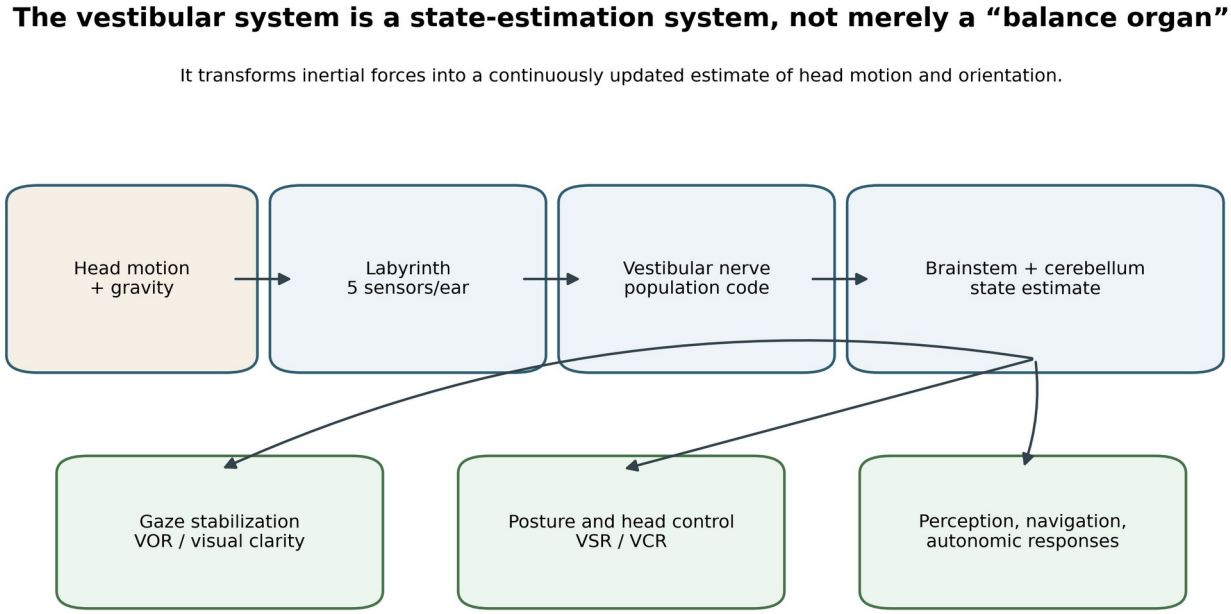

Figure 2. Original synthesis: the vestibular system converts inertial forces into an estimate of head motion and orientation, then distributes that estimate to reflex, postural, perceptual, and autonomic networks.

What this volume includes

- Why the vestibular system is best understood as an inertial state-estimation network.

- Functional anatomy of the five sensory organs in each labyrinth and the superior and inferior vestibular nerve divisions.
- Hair-cell transduction, tonic resting discharge, and regular versus irregular afferent coding.
- Semicircular-canal biomechanics, push-pull organization, and the physiologic basis of Ewald's laws.
- Otolith physiology, gravito-inertial acceleration, and the tilt-translation ambiguity.
- Vestibular nuclei, vestibulocerebellum, velocity storage, multisensory integration, and internal models.
- The vestibulo-ocular, vestibulospinal, and vestibulocollic reflexes.
- Adaptation, habituation, substitution, and compensation after vestibular loss.

What is intentionally deferred

Volume II will use this physiology to localize dizziness at the bedside and interpret vestibular tests. Volume III will apply the same framework to BPPV, acute unilateral vestibulopathy, stroke, Ménière disease, vestibular migraine, bilateral vestibulopathy, third-window syndromes, PPPD, and other clinical entities. Disease names appear here only when they clarify a physiologic principle.

Contents

Table 1. Narrative structure of Volume I.

Chapter	Core question
1. The problem the system solves	What must the brain know to keep vision, posture, and orientation stable?
2. Peripheral hardware	How do five sensors in each ear sample six degrees of freedom?
3. Hair-cell transduction	How does micrometer-scale bundle deflection become a bidirectional neural signal?
4. Semicircular canals	How do fluid mechanics turn rotation into a push-pull code?
5. Otolith organs	How do the maculae encode gravity and linear acceleration - and why is that ambiguous?
6. Primary afferents	Why do regular and irregular fibers carry complementary information?
7. Central processing	How are vestibular, visual, proprioceptive, and predictive signals combined?
8. Reflex outputs	How is a head-motion estimate transformed into eye, neck, and body responses?
9. Plasticity and compensation	Why do symptoms improve even when the peripheral lesion remains?

FIRST PRINCIPLES

1. The problem the vestibular system solves

A person can walk in darkness, turn the head while reading a street sign, land after a jump, and know which way is upright even when visual information is poor. These abilities require a continuously updated estimate of how the head is moving and how it is oriented relative to gravity. The peripheral labyrinth supplies the inertial evidence, but the useful product is a central estimate assembled from vestibular, visual, proprioceptive, and motor-prediction signals.[1,2]

The vestibular system measures forces, not symptoms

The peripheral organs do not detect “dizziness,” “balance,” or even self-motion in the everyday sense. Semicircular canals respond to angular motion. Otolith organs respond to gravito-inertial acceleration, which includes both linear acceleration and the gravitational vector. Hair cells transform those mechanical forces into changes in afferent firing. Everything beyond that - whether the person feels rotation, keeps the eyes on a target, activates an antigravity muscle, or becomes nauseated - is a consequence of central interpretation and output.

This distinction explains why vestibular disease is so multisystemic. A single asymmetric signal can alter eye movements, posture, perception, autonomic tone, and spatial confidence. It also explains why a patient can have severe symptoms with a relatively small peripheral perturbation: the brain is not simply reading a damaged sensor. It is trying to reconcile conflicting estimates of reality.

Six degrees of freedom

A rigid body moving in three-dimensional space has six degrees of freedom. It may rotate around three axes - yaw, pitch, and roll - and translate along three axes - fore-aft, left-right, and up-down. Each labyrinth contains three semicircular canals that sample angular motion and two otolith organs that sample linear acceleration and gravity. The sensors are not perfectly aligned with textbook axes, so natural movement is represented by a distributed pattern across multiple receptors rather than by one isolated organ.

Table 2. The peripheral organs sample physical motion; the brain constructs behaviorally useful variables.

Motion class	Examples	Dominant peripheral sensors	Immediate useful output
Angular	Turning the head, nodding, cartwheeling	Three semicircular canals	Angular velocity estimate for VOR, head control, and perception
Linear	Starting in a car, elevator acceleration, landing	Utricle and saccule	Translation estimate for ocular, postural, and perceptual responses
Static orientation	Head tilt relative to gravity	Utricle and saccule	Estimate of vertical and head

Motion class	Examples	Dominant peripheral sensors	Immediate useful output
			orientation
Combined natural motion	Walking, running, reaching, turning	All five organs plus other senses	Multisensory estimate of self-motion and orientation

Three jobs that must happen quickly

The vestibular estimate supports three broad functions. First, it stabilizes gaze. During a brief head movement, the vestibulo-ocular reflex can move the eyes with a latency of roughly several milliseconds, much faster than visually driven tracking. Second, it stabilizes the head and body through vestibulocollic and vestibulospinal pathways. Third, it contributes to spatial orientation, self-motion perception, navigation, and autonomic regulation. These functions share sensory evidence but apply it in different coordinate frames and at different time scales.[1,8]

CLINICAL BRIDGE

A patient with bilateral vestibular hypofunction may have little spontaneous vertigo because there is no large left-right imbalance, yet experience severe **oscillopsia and gait instability** because the dynamic estimate is weak on both sides. Symptoms follow the failed computation, not simply the amount of tissue loss.

Reference frames: the hidden difficulty

A canal reports motion in a head-fixed coordinate frame. The extraocular muscles act in orbital coordinates. Postural responses must be organized in body- and gravity-centered coordinates. Navigation eventually requires world-centered and memory-based maps. The central vestibular system therefore performs a series of transformations: it converts sensor-specific vectors into estimates that can be used by eyes, neck, trunk, limbs, and cortex.[1,2]

This is one reason central vestibular signs can be complex. A lesion may spare the peripheral measurement yet disrupt the transformation from head-centered input to an earth-vertical estimate, or from a canal signal to a conjugate ocular command. Skew deviation, ocular tilt reaction, impaired subjective visual vertical, and central positional nystagmus are not random findings; they reflect failures of particular transformations.

The brain relies on differences and predictions

Vestibular neurons fire at rest. The brain therefore reads motion mainly from changes in activity and from differences between paired sensors. This allows rapid bidirectional coding, but it creates a vulnerability: loss of tonic input from one side mimics a sustained motion signal even when the head is still. Central prediction adds another layer. During active movement, an efference copy of the motor command and neck proprioception help the brain predict the expected vestibular

consequences. Unexpected passive motion is processed differently from self-generated motion, even when the physical head trajectory is similar.[2]

RESIDENT TAKEAWAY

The unifying pathophysiology of an acute unilateral peripheral lesion is **false motion produced by an asymmetric tonic code**. The unifying pathophysiology of bilateral loss is **an inadequate dynamic code**.

CHAPTER SYNTHESIS

- The labyrinth supplies inertial evidence; the brain constructs self-motion, verticality, and orientation.
- Six degrees of freedom are represented by a population of canals and otolith receptors rather than one sensor per everyday movement.
- Fast reflexes, posture, perception, and autonomic responses share vestibular information but transform it into different coordinate frames.
- Tonic discharge makes bidirectional coding possible and makes unilateral loss look like motion.

ANATOMY THAT PREDICTS PHYSIOLOGY

2. Peripheral hardware: the labyrinth as a sensor array

The bony labyrinth protects a delicate membranous labyrinth filled with endolymph. Within the membranous labyrinth, three cristae and two maculae contain the hair cells that detect head motion. The gross anatomy is not merely descriptive: canal shape determines fluid mechanics, the cupula determines angular sensitivity, the otoconial membrane determines sensitivity to gravity and translation, and nerve divisions predict patterns of selective dysfunction.

Bony and membranous labyrinths

The bony labyrinth contains perilymph and includes the vestibule, semicircular canals, and cochlea. Suspended within it is the membranous labyrinth, a continuous epithelial system filled with endolymph. The sensory portions of the vestibular membranous labyrinth are the three semicircular ducts, the utricle, and the saccule. Each semicircular duct expands near one end into an ampulla. The ampulla houses the crista ampullaris and its gelatinous cupula. The utricle and saccule contain maculae covered by an otoconial membrane.

The vestibular organs share the inner ear's unusual ionic arrangement. Endolymph is potassium-rich and maintains a positive electrical potential relative to the hair-cell interior, while perilymph resembles extracellular fluid and is sodium-rich. Apical hair bundles are exposed to endolymph; the basolateral surface is exposed to perilymph. This separation creates a strong electrochemical drive for potassium to enter when mechanically gated channels open. Dark cells and other specialized epithelia help maintain endolymph composition.[4,5]

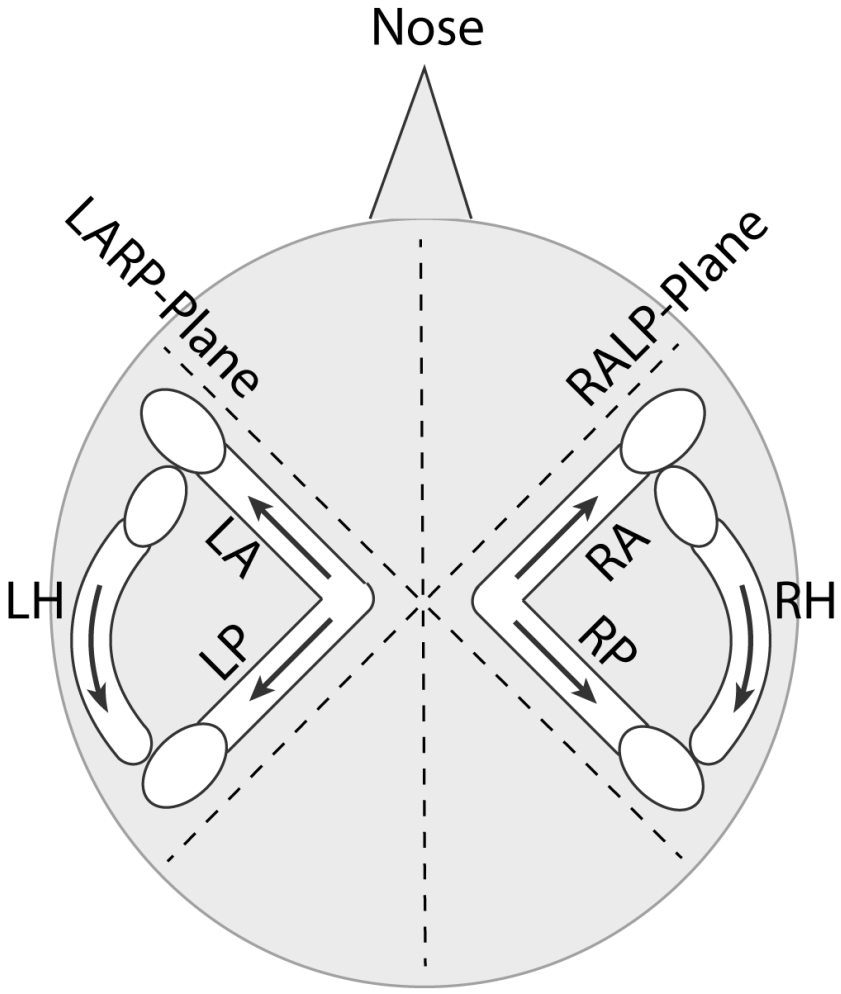

Figure 3. The three canals occupy approximately orthogonal planes. Across the two ears, the horizontal canals form one functional pair; the left anterior pairs with the right posterior, and the right anterior pairs with the left posterior. NASA; public domain.

The five sensors in each ear

Table 3. Boards-relevant organization of the peripheral vestibular end organs.

End organ	Primary mechanical stimulus	Approximate orientation / role	Vestibular nerve division
Horizontal canal	Angular acceleration/velocity in its plane	Predominantly yaw; ~20-30° above true horizontal in upright head	Superior
Anterior canal	Angular motion in vertical canal plane	Combined pitch and roll components; paired with contralateral posterior canal	Superior
Posterior canal	Angular motion in vertical canal plane	Combined pitch and roll components; paired with contralateral anterior canal	Inferior
Utricle	Gravito-inertial acceleration	Macula roughly horizontal when upright; broad	Superior

End organ	Primary mechanical stimulus	Approximate orientation / role	Vestibular nerve division
		sensitivity to horizontal translation and tilt	
Saccule	Gravito-inertial acceleration	Macula roughly vertical when upright; broad sensitivity to vertical acceleration and gravity	Inferior

The table is useful, but the words “horizontal” and “vertical” should not be treated as strict one-dimensional labels. Hair-cell polarization varies across each macula, and the canal planes are oblique to the standard cranial axes. The brain infers a three-dimensional vector from the pattern across many afferents.

The crista and cupula

Within each ampulla, hair cells sit on the crista ampullaris. Their bundles project into the cupula, a gelatinous structure that spans the lumen of the ampulla. Because the cupula has nearly the same density as endolymph, gravity alone does not significantly deflect it in a normal labyrinth. This is why a healthy semicircular canal reports rotation rather than static head position.

During angular acceleration, inertia creates relative endolymph movement and a pressure difference across the cupula. The cupula bends, the hair bundles bend with it, and afferent firing changes. The cupula also provides elastic restoring force. The resulting system behaves like a damped torsional element: it responds strongly during the onset and offset of rotation and then gradually returns toward baseline during prolonged constant velocity.[3]

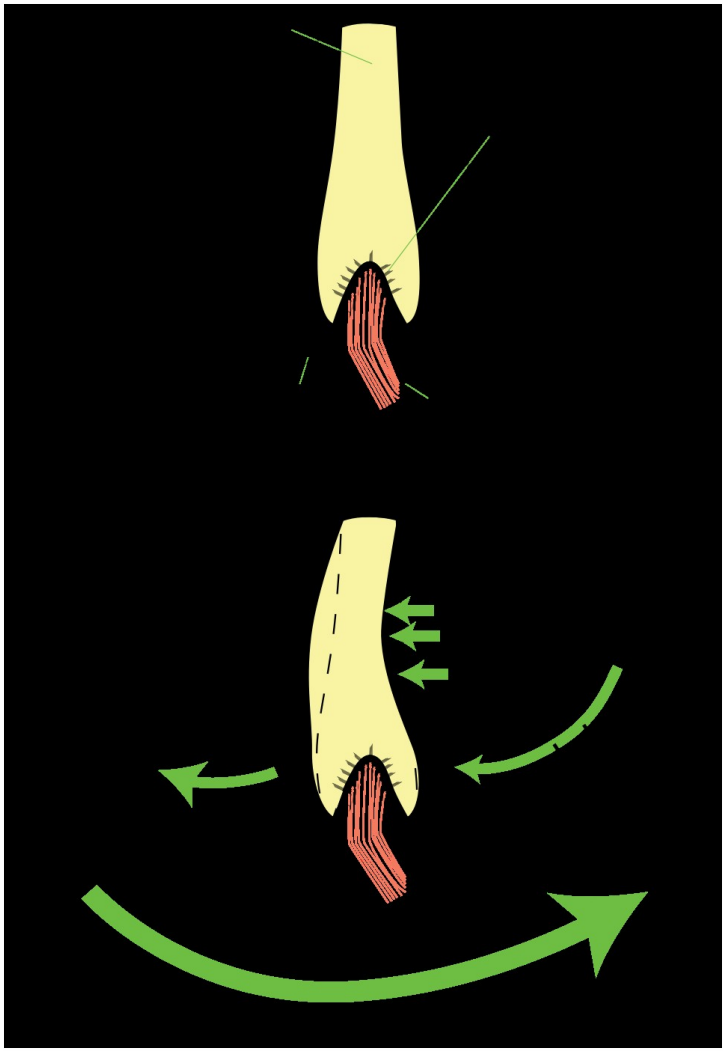

Figure 4. Crista, cupula, and endolymph mechanics within an ampulla. Relative endolymph flow deflects the cupula and bends the embedded hair bundles. Thomas Haslwanter; CC BY-SA 3.0.

The macula and otoconial membrane

The utricular and saccular sensory epithelia are maculae. Their hair bundles project into a gelatinous otolithic membrane loaded with calcium carbonate crystals called otoconia. The added mass and density of this layer make it responsive to gravity and linear acceleration. When the head tilts or translates, inertia and gravity create shear between the otoconial membrane and the sensory epithelium. This bends hair bundles and changes afferent discharge.

A curved landmark called the striola divides each macula. Hair-cell polarity reverses across it. In the utricle, kinocilia are generally oriented toward the striola; in the saccule, they are generally oriented away from it. This arrangement creates a two-dimensional map of preferred directions. Any natural acceleration excites some hair cells, inhibits others, and leaves others relatively unchanged.

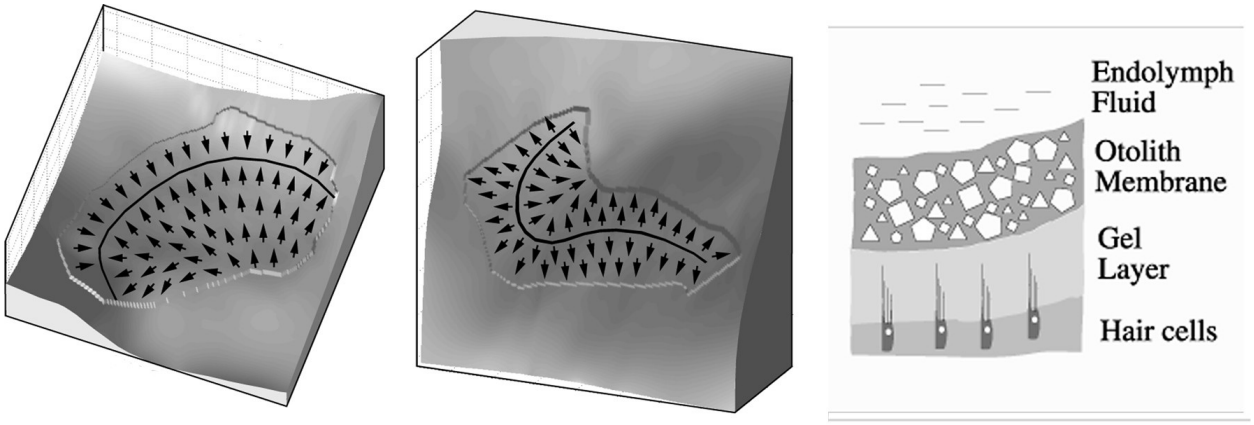

Figure 5. Utricle, saccule, local hair-cell preferred directions, striolae, and a cross-section through the otoconial membrane. Thomas Haslwanter; CC BY-SA 3.0.

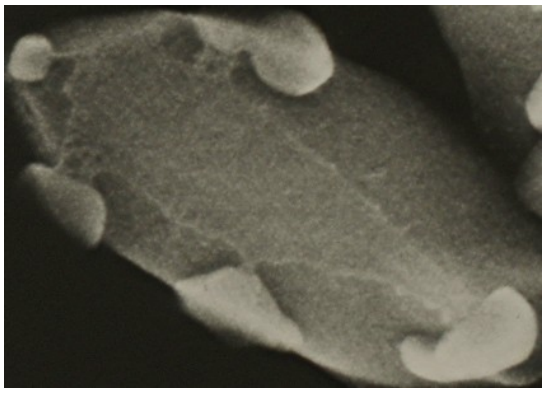

Figure 6. Scanning electron micrograph of a vestibular otoconium. Otoconia add inertial mass to the otolithic membrane. Cesar D. Fermin; CC BY-SA 3.0.

Scarpa ganglion and nerve divisions

Primary vestibular neuron cell bodies reside in Scarpa's ganglion. Their peripheral processes contact vestibular hair cells; their central processes enter the brainstem with cranial nerve VIII. The superior vestibular nerve carries input predominantly from the horizontal and anterior canals and the utricle. The inferior division carries input from the posterior canal and saccule. A small anastomotic branch, Voit's nerve, creates some variability, so clinical patterns are not always perfectly compartmentalized.

This division matters clinically. A selective superior vestibular neuritis pattern can impair horizontal and anterior canal function and utricular pathways while sparing the posterior canal and saccular pathways. Conversely, posterior canal and cVEMP abnormalities suggest inferior-division involvement. Volume II will connect these anatomic predictions to vHIT and VEMP interpretation.

ANATOMIC TRAP

The canals are commonly called “horizontal,” “anterior,” and “posterior,” but bedside testing should be aligned with their **actual planes**, not with an imagined orthogonal skull grid. The horizontal canal is brought closer to the earth-horizontal plane by flexing the head about 20-30°.

CHAPTER SYNTHESIS
- Each labyrinth contains three cristae for angular motion and two maculae for gravito-inertial acceleration.
- Cupular density approximates endolymph, minimizing gravitational sensitivity of healthy canals.
- Otoconia add mass to the otolithic membrane, creating sensitivity to gravity and translation.
- Superior versus inferior vestibular nerve anatomy predicts selective patterns on canal and VEMP testing.

CELLULAR PHYSIOLOGY

3. Hair-cell transduction: from bundle deflection to afferent firing

All five vestibular organs use the same fundamental receptor: the mechanosensory hair cell. The organ-specific mechanics determine what bends the bundle; the hair cell converts that bend into a graded receptor potential and neurotransmitter release. The key principle is bidirectionality. A vestibular hair cell is not simply off at rest and on during movement. It sits near the middle of its operating range, allowing deflection in one direction to increase firing and deflection in the other to decrease it.

The polarized hair bundle

A vestibular hair bundle contains rows of actin-rich stereocilia arranged in increasing height and a single kinocilium adjacent to the tallest row. The bundle therefore has a morphological axis. Deflection toward the kinocilium is excitatory; deflection away from it is inhibitory. Deflection perpendicular to this axis produces much less response. This preferred direction is the microscopic basis of the vector maps across the cristae and maculae.

Tip links connect adjacent stereocilia and transmit tension to mechanically gated ion channels. When the bundle bends toward the kinocilium, tip-link tension increases and more transduction channels open. Potassium - and some calcium - enters from potassium-rich endolymph, depolarizing the cell. Voltage-gated calcium channels at the basolateral membrane open, increasing glutamate release at ribbon synapses. Deflection away relaxes the tip links, closes channels, hyperpolarizes the cell, and reduces transmitter release.[4,5]

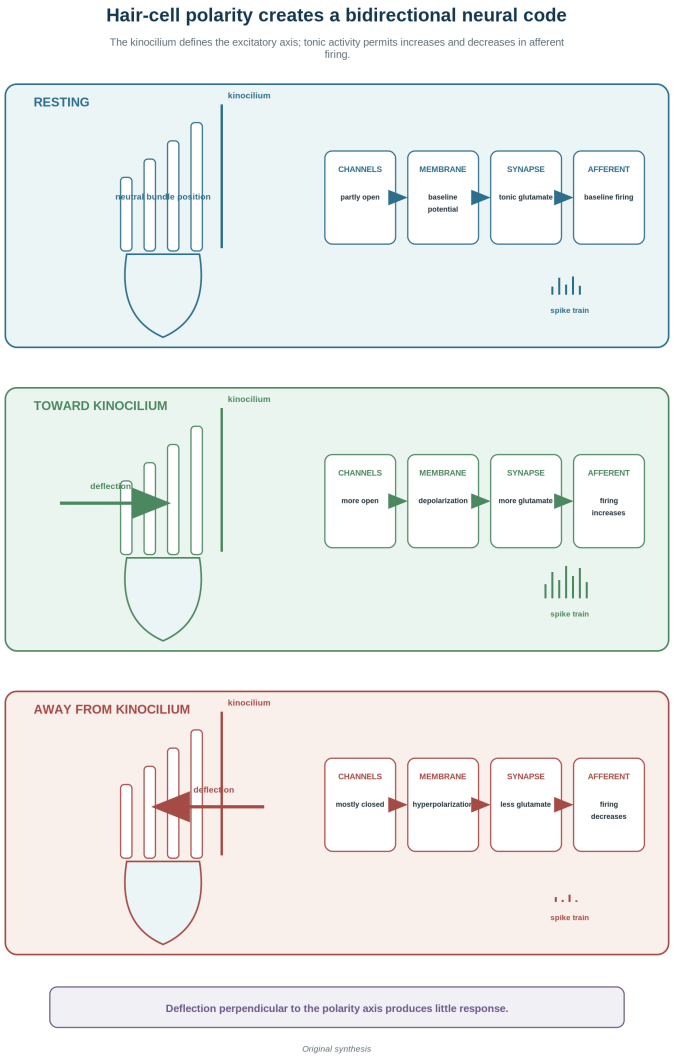

Figure 7. Original synthesis. Hair-cell polarity creates a bidirectional code: deflection toward the kinocilium increases channel opening and afferent firing; deflection away decreases both.

Why potassium enters rather than leaves

The hair-cell interior is electrically negative relative to endolymph, and endolymph has a high potassium concentration. Opening apical channels therefore drives potassium into the cell. This is opposite the intuition learned from most neurons, where opening potassium channels generally causes potassium efflux and hyperpolarization. The unusual extracellular environment of the inner ear turns potassium into the principal depolarizing ion at the apical mechanotransduction channel.

BOARDS PEARL

In a vestibular hair cell, **K+ influx from endolymph depolarizes the cell**. Basolateral K+ efflux into perilymph then helps repolarize it.

Graded receptor potentials and tonic discharge

Hair cells do not encode ordinary head motion with all-or-none action potentials. Their receptor potential is graded, and transmitter release varies continuously with bundle deflection. The primary

afferent generates action potentials. Because transduction channels have a nonzero open probability at rest and ribbon synapses release transmitter tonically, vestibular afferents maintain spontaneous firing even when the head is still.

This resting discharge provides room for both increases and decreases. Excitatory bundle deflection increases spike rate. Inhibitory deflection decreases it, potentially toward zero during strong stimuli. The system's ability to encode inhibition is therefore limited by an inhibitory cutoff: firing cannot fall below zero. Excitatory responses have greater dynamic range, one reason strong excitatory responses often dominate clinical nystagmus and underlie parts of Ewald's second and third laws.

Type I and type II hair cells

Mammalian vestibular organs contain two hair-cell morphologies. Type I cells are flask-shaped and are surrounded by calyx afferent endings. Type II cells are more cylindrical and receive bouton afferent contacts. Many primary afferents are dimorphic, contacting both type I and type II cells. Morphology, epithelial zone, synaptic arrangement, membrane properties, and afferent regularity are interrelated but not perfectly interchangeable categories.[6,7]

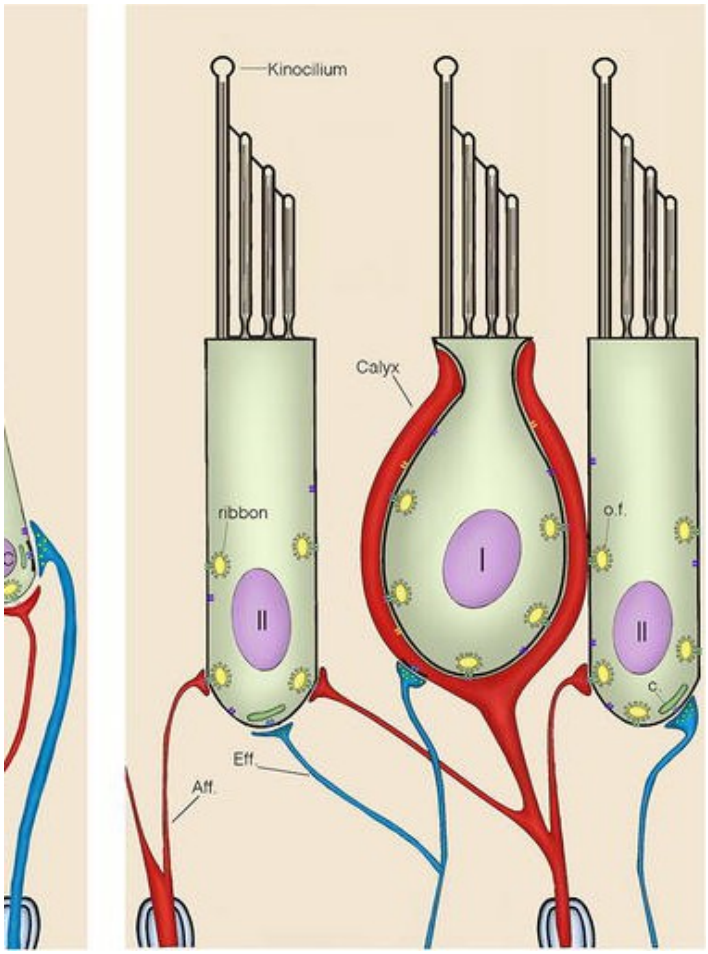

Figure 8. Vestibular hair-cell and afferent morphologies: type I cells with calyx endings, type II cells with bouton endings, and dimorphic afferents. Cropped to the vestibular panel from Masetto, Spaiardi, and Johnson; CC BY 3.0.

Table 4. A useful but deliberately non-absolute comparison of vestibular hair-cell classes.

Feature	Type I hair cell	Type II hair cell	Interpretive caution
Shape	Flask-shaped	Cylindrical	Morphology does not by itself define the entire afferent response.
Afferent ending	Calyx; often part of calyx-only or dimorphic afferent	Bouton; often part of bouton-only or dimorphic afferent	Most vestibular afferents integrate multiple contacts.
Distribution	Enriched in central/striolar zones but present broadly	Present throughout, with regional differences	Human and animal distributions vary.
Functional association	Often linked to irregular, transient, high-frequency-sensitive signaling	Often linked to more regular, sustained signaling	Regularity is a property of the afferent spike train, not simply the hair-cell type.

Adaptation at the receptor level

If a bundle remains deflected, the transduction apparatus partially adapts. Hair-bundle mechanics and channel gating reset over multiple time scales, preserving sensitivity to further changes. This local adaptation is distinct from the much larger central phenomena called VOR adaptation or vestibular compensation, although all serve the broad purpose of keeping the system responsive within a useful operating range.[4]

Efferent vestibular modulation

The vestibular periphery also receives a bilateral cholinergic efferent innervation from the brainstem. Efferent activation can influence type II hair cells, calyces, boutons, and afferent firing. Its exact behavioral role in humans remains less clear than the olivocochlear system's role in audition. Proposed functions include adjusting peripheral sensitivity during movement, increasing signal reliability, and modulating responses across behavioral states.[18]

CLINICAL BRIDGE

Aminoglycoside toxicity, age-related loss, genetic disease, and inner-ear inflammation may affect hair-cell populations and afferent classes unevenly. A normal response at one frequency or in one end organ therefore does not prove that the entire labyrinth is normal.

CHAPTER SYNTHESIS

- Hair-bundle polarity makes each receptor direction-selective.
- Deflection toward the kinocilium opens mechanotransduction channels, producing K^+ influx, depolarization, glutamate release, and increased afferent firing.
- Tonic discharge allows motion to be encoded by both excitation and inhibition but creates an inhibitory cutoff.
- Type I/type II hair cells and calyx/bouton/dimorphic afferents contribute to complementary sustained and transient channels.

ROTATION

4. Semicircular canals: fluid mechanics and push-pull coding

The semicircular canals are often described as angular-acceleration detectors. That is mechanically true at the instant rotation begins, but physiologically incomplete. Over the frequencies of ordinary head movement, cupular dynamics and central processing allow canal afferents to represent angular head velocity remarkably well. Understanding the onset, steady-state, and stopping phases of rotation makes nystagmus, the head impulse test, calorics, and rotary-chair responses easier to predict. [3]

A loop of fluid with an elastic gate

When the head begins to rotate, the bony canal and membranous duct move with the skull. Endolymph initially lags because of inertia. The resulting relative fluid motion creates a pressure difference across the cupula. Cupular deflection bends the crista's hair bundles. As the fluid catches up and the cupula's elasticity restores it toward center, the peripheral response decays even if the head continues at constant angular velocity.

When rotation stops, the skull and canal stop first but endolymph continues briefly. The cupula deflects in the opposite direction, creating an after-response. This is why prolonged chair rotation produces nystagmus at the beginning and an oppositely directed post-rotatory nystagmus after stopping.

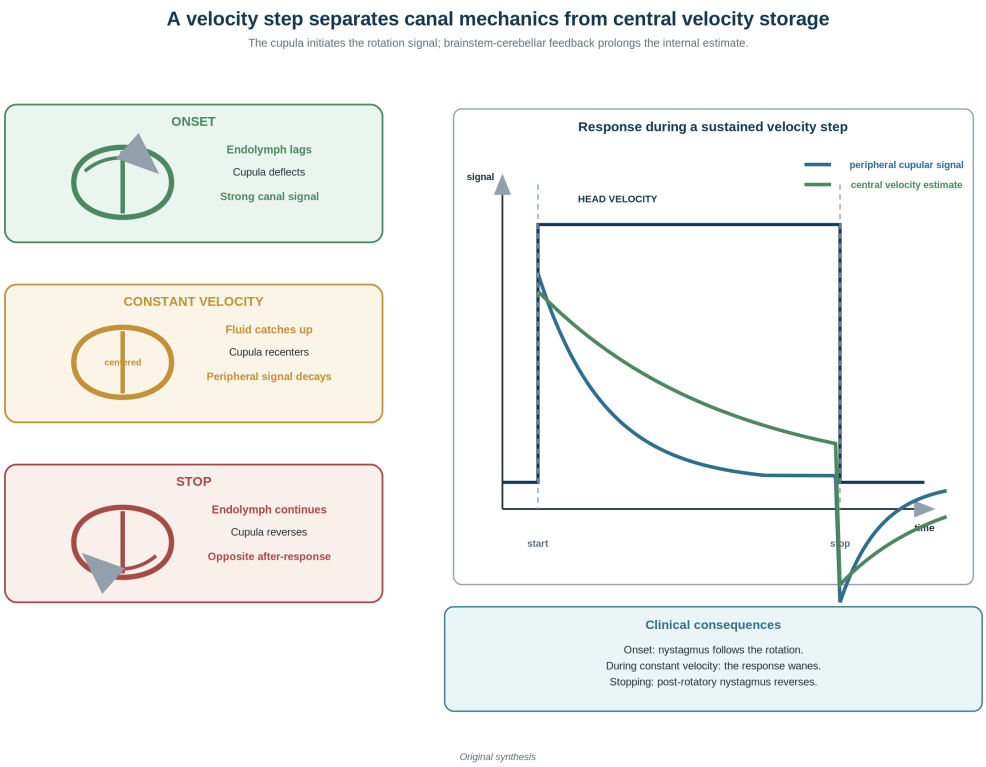

Figure 9. Original synthesis. During a velocity step, the peripheral cupular signal decays while central velocity storage prolongs the rotation estimate and produces an opposite after-response at stopping.

A mechanical high-pass filter that behaves like a velocity sensor

The canal-cupula system can be modeled with a short inertial time constant and a longer elastic time constant. At very low frequencies, the cupula returns toward center and the canal poorly represents sustained slow rotation. Across much of the range of natural head movements, however, cupular displacement is approximately proportional to angular velocity. At very high frequencies, additional biomechanical and neural factors shape the response. The practical lesson is that different tests sample different portions of this frequency response.[3]

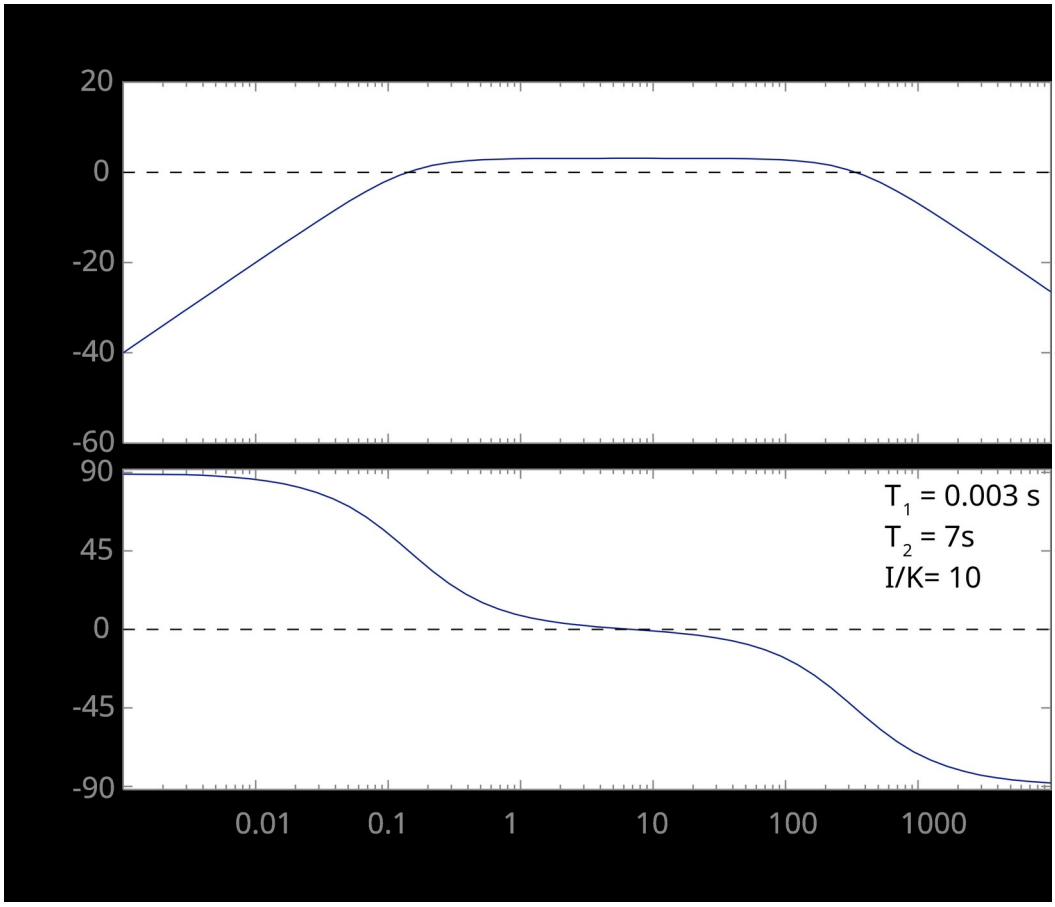

Figure 10. Frequency response of a human semicircular-canal model. The mid-frequency region approximates a velocity-sensitive plateau; performance falls at very low frequencies. Thomas Haslwanter; CC BY-SA 3.0.

CLINICAL BRIDGE

Calorics create an extremely low-frequency equivalent stimulus, rotary chair samples low-to-mid frequencies, and vHIT samples high-frequency, high-acceleration head motion. These tests are complementary, not redundant.

Canal planes and vector projection

Each canal is most sensitive to angular velocity about an axis perpendicular to its plane. A pure yaw rotation strongly drives the horizontal pair. A rotation in the left-anterior/right-posterior plane drives the LARP pair. A rotation in the right-anterior/left-posterior plane drives the RALP pair. Most natural movements project onto more than one canal plane, so the brain reconstructs a three-dimensional angular-velocity vector from the combined afferent pattern.

Push-pull organization

Coplanar canals across the two ears operate as functional pairs. During a leftward yaw rotation, endolymph mechanics excite the left horizontal canal and inhibit the right horizontal canal. The central nervous system compares the two sides. The difference increases the signal-to-noise ratio, expands directional coding, and allows common-mode fluctuations to be rejected.

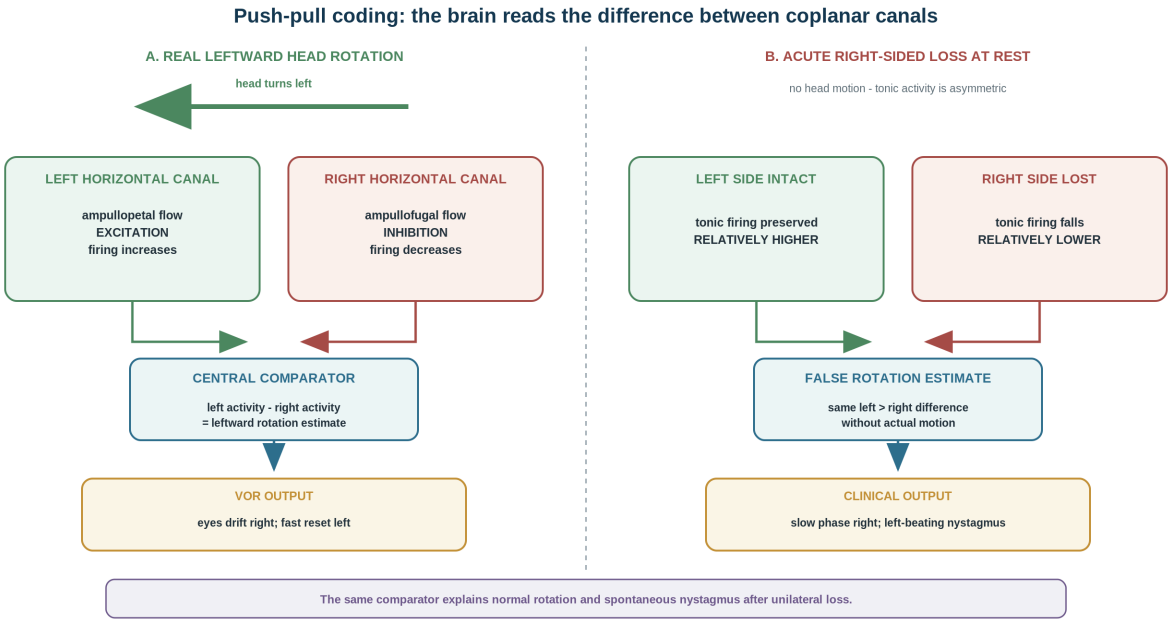

Figure 11. Original synthesis. The same left-greater-than-right activity difference represents real leftward rotation and the false rotation signal produced by acute right unilateral loss.

Push-pull organization also explains acute unilateral vestibular loss. If right-sided tonic input suddenly falls, the left side is relatively more active. The brain initially interprets that difference as leftward rotation. The eyes drift slowly toward the weaker right side through the VOR and reset rapidly to the left, producing left-beating nystagmus. The fast phase names the nystagmus; the slow phase reflects the vestibular drive.

Ampullopetal and ampullofugal flow

The relationship between fluid direction and excitation differs between horizontal and vertical canals because hair-cell polarity is arranged differently. In the horizontal canal, ampullopetal flow - toward the ampulla - is excitatory. In the anterior and posterior canals, ampullofugal flow - away from the ampulla - is excitatory. Memorization is easier when tied to the clinically useful Ewald framework.

Table 5. Ewald's laws as consequences of canal plane and excitatory-inhibitory asymmetry.

Ewald law	Meaning	Clinical use
First	Stimulation of a canal produces eye movement in the plane of that canal.	The axis of the slow-phase eye movement identifies the canal plane being driven.
Second	In the horizontal canal, ampullopetal flow produces a stronger response than ampullofugal flow.	Geotropic horizontal-canal BPPV: the stronger response generally occurs with the affected ear down.
Third	In the vertical canals, ampullofugal flow produces a stronger response than ampullopetal flow.	Helps predict vertical-canal excitation and positional nystagmus patterns.

Why excitation often dominates inhibition

An afferent can increase its firing far above baseline, but inhibition is bounded at zero. During strong stimulation, the excited side therefore contributes more dynamic range than the inhibited side. This excitatory dominance is especially relevant during high-acceleration head impulses and intense positional responses. It is not an excuse to ignore the inhibited partner; the central code remains fundamentally bilateral.

The head impulse as a physiologic stress test

A small, rapid, unpredictable head impulse demands a high-frequency VOR. If the canal-nerve pathway on the side of the impulse cannot generate adequate eye velocity, the eyes move with the head and leave the target. A corrective saccade returns fixation. The test is powerful because the stimulus is too fast for smooth pursuit and predictive visual tracking to fully substitute. Covert saccades during the impulse can make bedside observation falsely reassuring, which is one reason vHIT can add value.

REASON IT THROUGH

A rightward head impulse normally excites the right horizontal canal. If right horizontal canal output is weak, leftward compensatory eye velocity is insufficient. The eyes are carried rightward with the head, then make a **leftward refixation saccade** back to the target.

CHAPTER SYNTHESIS

- Canal mechanics convert angular acceleration into a cupular signal that approximates head velocity over behaviorally important frequencies.
- Prolonged constant velocity produces response decay; stopping produces an opposite after-response.
- Coplanar canals form push-pull pairs, and unilateral tonic loss mimics rotation toward the intact side.
- Ewald's laws link canal plane, flow direction, and excitatory dominance to eye-movement patterns.

LINEAR ACCELERATION AND VERTICALITY

5. Otolith organs: gravity, translation, and ambiguity

The utricle and saccule are sometimes reduced to "horizontal" and "vertical" accelerometers. That shorthand is useful at the beginning, but the deeper concept is that both organs encode gravito-inertial acceleration. Their output alone cannot determine whether the head tilted relative to gravity or translated through space. The brain must solve that ambiguity by integrating canal, visual, proprioceptive, and predictive information.[9]

Shear across a weighted membrane

Otoconia make the otolithic membrane denser than the surrounding endolymph. During translation, the sensory epithelium moves with the skull while the weighted membrane lags, producing shear. During static tilt, gravity pulls the membrane relative to the macula. Both mechanisms bend hair bundles. Unlike the cupula, the otoconial layer remains sensitive to a sustained gravitational vector, so the otoliths can report static head orientation.

The utricle and saccule are vector maps

The utricular macula lies roughly in the horizontal plane in an upright person and is particularly useful for interaural and fore-aft acceleration. The saccular macula lies roughly vertical and is particularly useful for vertical acceleration. Yet each contains hair cells with many preferred directions, and both contribute to natural three-dimensional motion. The polarity reversal at the striola means a given acceleration increases firing in one population and decreases it in another.

Table 6. Functional comparison of the otolith organs.

Feature	Utricle	Saccule
Approximate macular orientation upright	Predominantly horizontal	Predominantly vertical
Common functional shorthand	Horizontal translation and head tilt	Vertical translation and gravity
Hair-cell polarity relative to striola	Kinocilia generally toward striola	Kinocilia generally away from striola
Dominant nerve division	Superior vestibular nerve	Inferior vestibular nerve
Common clinical assay	oVEMP, subjective visual vertical, ocular tilt pathways	cVEMP; sound/vibration-sensitive saccular pathways
Important caution	Not a one-axis sensor	Not exclusively a vertical sensor

The gravito-inertial acceleration vector

At the receptor, the relevant physical quantity is the vector sum of gravity and linear acceleration. This creates the tilt-translation ambiguity. A backward linear acceleration while upright can produce the same otolith shear as a backward head tilt in a stationary person. The peripheral signal is physically identical. The distinction must emerge centrally.

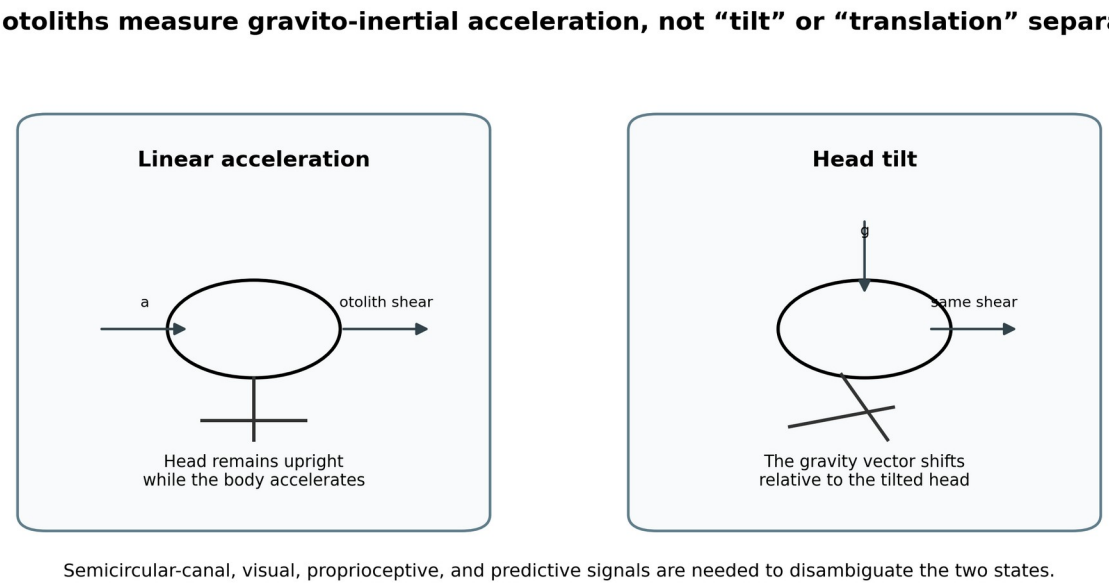

Figure 12. Original synthesis: linear acceleration and head tilt can create the same otolith shear, so extra-otolith cues are required.

This is a biological expression of the equivalence between gravity and linear acceleration. The brain estimates tilt by integrating angular-velocity information from the semicircular canals over time and combining it with the otolith vector. Visual vertical, somatosensory cues, and prior expectations further constrain the estimate. At low frequencies, canal signals decay and the computation becomes less accurate, which helps explain somatogravic illusions and other forms of spatial disorientation.[9]

AVIATION ANALOGY

During sustained forward acceleration without reliable visual cues, the otolith signal can be interpreted as a backward tilt. A pilot may feel that the nose is pitching up even when the aircraft attitude is unchanged. The receptor is not malfunctioning; the ambiguous signal is being resolved incorrectly.

Otolith-ocular responses

Otolith signals influence the eyes in two broad ways. Static head tilt evokes ocular counter-roll and contributes to the ocular tilt reaction. Linear translation evokes translational VOR responses that depend strongly on viewing distance: a nearby target requires more eye rotation than a distant target for the same head translation. This geometric dependence distinguishes translational VOR from the angular VOR, whose ideal gain is near one regardless of target distance.

Otolith-spinal and perceptual responses

Otolith pathways contribute to antigravity tone, postural alignment, perception of vertical, and autonomic adjustments to posture. Because these functions depend on integration across both ears and across multiple senses, isolated unilateral otolith loss may be less obvious than a canal deficit. Patients may report tilt, lateropulsion, vague disequilibrium, or visual vertical distortion rather than a classic spinning sensation.

Sound and vibration sensitivity

Irregular afferents, particularly in striolar regions, can respond to high-frequency bone-conducted vibration and sufficiently intense acoustic stimuli. This property enables vestibular evoked myogenic potentials. cVEMP predominantly assays a saccular-inferior vestibular nerve pathway to the ipsilateral sternocleidomastoid, whereas oVEMP predominantly reflects a utricular-superior vestibular nerve pathway expressed beneath the contralateral eye. These are pathway-biased tests, not perfectly isolated organ assays.[6]

CLINICAL BRIDGE

A low-threshold, high-amplitude VEMP is not simply "excellent otolith function." In the appropriate clinical setting it can reflect abnormally efficient sound/pressure transmission through a third mobile window.

CHAPTER SYNTHESIS

- Otoliths encode gravito-inertial acceleration through shear of a weighted otoconial membrane.
- Utricle and saccule are multidirectional vector maps, not single-axis switches.
- Tilt and translation are indistinguishable at the peripheral receptor and must be separated centrally.
- Otolith signals contribute to verticality, ocular counter-roll, translational VOR, posture, and VEMP responses.

THE VESTIBULAR NERVE

6. Primary vestibular afferents: complementary neural channels

The vestibular nerve does not transmit one uniform copy of hair-cell activity. Primary afferents vary in resting regularity, sensitivity, dynamics, morphology, epithelial location, and frequency preference. This diversity allows the same end organ to represent both sustained low-frequency motion and brief high-frequency transients.

Resting discharge and population coding

Individual afferents fire spontaneously, but their rates and regularity differ. A head movement changes the distribution of firing across thousands of fibers. Central neurons can therefore estimate motion from both the mean change and the pattern of activity. A population code is robust: noise or loss in a few fibers does not necessarily abolish the estimate, but selective loss can distort particular frequencies or stimulus classes.

Regular and irregular afferents

Regular afferents have relatively uniform interspike intervals and often show more sustained, linear responses. Irregular afferents have more variable resting intervals, higher sensitivity to transient acceleration, greater phase lead, and strong responses to high-frequency vibration or sound. Calyx-only and dimorphic afferents in central or striolar zones tend to be irregular; bouton afferents in peripheral or extrastriolar zones tend to be more regular, but the mapping is not absolute.[6]

Table 7. Complementary afferent channels. These are tendencies rather than rigid classes.

Property	More regular / sustained channel	More irregular / transient channel
Spike timing	Relatively uniform interspike intervals	Variable interspike intervals
Typical morphology/zone	Bouton and some dimorphic; peripheral or extrastriolar	Calyx-only and some dimorphic; central or striolar
Dynamic emphasis	Steady and lower-frequency components	Rapid changes, high acceleration, vibration, sound
Input-output behavior	Often more linear over a broad range	Often higher gain and more nonlinear
Conceptual role	Reliable estimate of ongoing motion	Sensitive detection of unexpected transients

Sustained and transient systems

A useful conceptual model divides vestibular signaling into sustained and transient systems. The sustained channel provides a stable representation of ongoing head motion and orientation. The transient channel emphasizes abrupt, high-frequency events. Natural behavior requires both. Walking and standing require reliable low-frequency orientation signals, while a slip, stumble, or rapid head turn demands immediate detection of a transient.

This framework also helps explain why tests can dissociate. A patient may have preserved responses to slow rotation but impaired high-acceleration impulses, or the reverse. The lesion may involve different receptor zones, afferent classes, frequencies, or central compensatory mechanisms.

Superior and inferior divisions revisited

The superior division carries the horizontal and anterior canal branches and most utricular afferents. The inferior division carries the posterior canal and saccular branches. Because the central processes converge in the vestibular nuclei and because peripheral anastomoses occur, this map predicts rather than guarantees test patterns. Nonetheless, it is clinically valuable: horizontal/anterior vHIT and oVEMP abnormalities cluster with superior-division dysfunction, while posterior vHIT and cVEMP abnormalities cluster with inferior-division dysfunction.

Active versus passive head motion

Primary afferents mainly encode the physical motion of the head and respond similarly during active and passive movements. The distinction between self-generated and externally imposed motion emerges strongly at the first central stages, where vestibular signals are compared with motor efference copy and proprioceptive feedback. During expected active head motion, central neurons can suppress the sensory consequences that do not require a corrective response; unexpected passive perturbations remain salient.[2]

CLINICAL BRIDGE

The head impulse test is deliberately **passive and unpredictable**. Predictable active motion gives the brain opportunities to use motor commands, preprogrammed eye movements, and substitution strategies.

CHAPTER SYNTHESIS

- Primary afferents form a diverse population code rather than a homogeneous cable.
- Regular and irregular fibers emphasize complementary sustained and transient motion information.
- Superior and inferior nerve divisions provide a useful framework for interpreting selective canal and VEMP abnormalities.
- Discrimination of active from passive motion is largely a central computation.

BRAINSTEM, CEREBELLUM, AND CORTEX

7. Central vestibular processing: comparison, integration, and internal models

Vestibular information becomes multisensory almost immediately after entering the brainstem. The vestibular nuclei receive labyrinthine input, but also visual motion, eye-position, neck proprioceptive, cerebellar, commissural, and motor-prediction signals. The central system is therefore not a relay station. It estimates which component of sensory activity represents actual head motion, transforms that estimate into useful coordinates, and calibrates reflexes to current behavior.[1,2]

The vestibular nuclear complex

Four major vestibular nuclei - superior, medial, lateral, and inferior - extend through the rostral medulla and caudal pons, with additional smaller cell groups. Their borders and functions overlap. Broadly, superior and medial nuclei are heavily involved in vestibulo-ocular pathways; the lateral nucleus is a major source of the lateral vestibulospinal tract; medial and inferior regions integrate canal, otolith, cerebellar, reticular, spinal, and commissural information.

Primary afferents project ipsilaterally to the nuclei and directly to the cerebellum. Vestibular nuclear neurons project to ocular motor nuclei through the medial longitudinal fasciculus, to the spinal cord, to reticular and autonomic centers, to the cerebellum, and through thalamic relays to a distributed cortical network. The system has no single primary vestibular cortex analogous to primary visual cortex.

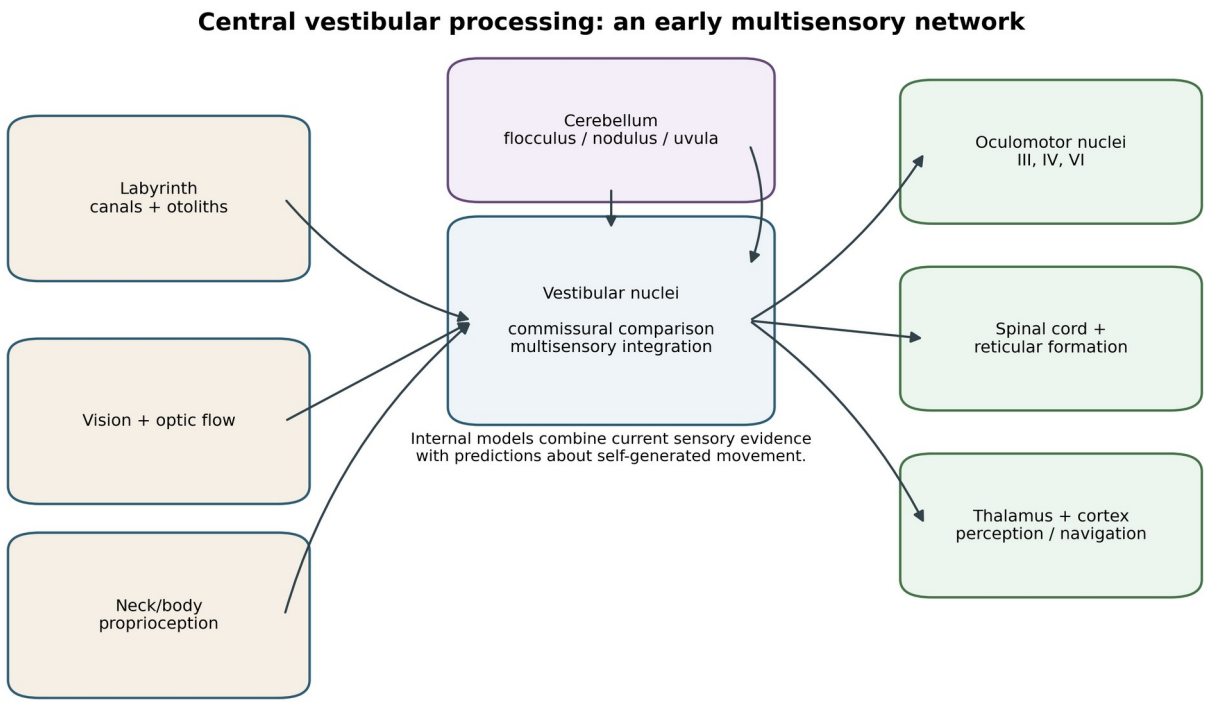

Figure 13. Original synthesis: vestibular nuclei combine labyrinthine, visual, proprioceptive, and predictive information, with cerebellar calibration and widespread motor and perceptual outputs.

Commissural comparison

The two vestibular nuclear complexes are linked by strong excitatory and inhibitory commissural pathways. These circuits sharpen the push-pull comparison and contribute to the profound imbalance after unilateral loss. Immediately after a unilateral lesion, activity in ipsilesional vestibular nuclei falls both because peripheral input is lost and because the intact side exerts commissural inhibition. Recovery of balanced resting activity requires changes on both sides.

The vestibulocerebellum

The flocculus and paraflocculus calibrate the VOR, support smooth pursuit and VOR cancellation, and drive motor learning when retinal slip indicates that eye velocity is too high or too low. The nodulus and ventral uvula process otolith-canal interactions, orient velocity storage to gravity, and help distinguish tilt from translation. The fastigial nucleus and vermal regions contribute to postural, locomotor, and spatial transformations.

The cerebellum does not merely inhibit vestibular responses. It tunes gain, timing, axis, and context. A normal VOR must be strong during an unexpected head perturbation, reduced when the eyes and head intentionally move together, and recalibrated when lenses alter visual consequences. Cerebellar lesions can therefore produce gaze-evoked nystagmus, impaired VOR suppression, abnormal duration or orientation of vestibular responses, and poor adaptation.[8,12]

Velocity storage

Peripheral cupular responses decay with a time constant of only several seconds, yet vestibular nystagmus and perception during rotation can persist longer. A distributed brainstem-cerebellar network called velocity storage extends the effective time constant of angular-velocity signals,

especially for low-frequency horizontal rotation. It also helps transform rotation signals according to gravity and contributes to optokinetic after-nystagmus.[8]

Velocity storage is useful because it preserves a motion estimate after the peripheral signal begins to decay. It can also create symptoms when poorly calibrated. Motion sickness susceptibility, prolonged post-rotatory responses, and some forms of central positional nystagmus involve abnormal sensory conflict or velocity-storage dynamics.

THINK OF IT AS MEMORY

Velocity storage is a short-lived neural memory of rotation. The canals initiate the estimate; central feedback prevents it from disappearing as quickly as the cupula returns toward center.

Internal models and sensory prediction

The brain continually predicts the sensory consequences of movement. A motor command to turn the head is accompanied by an efference copy. Neck proprioception reports whether the head actually moved relative to the trunk. Vision reports optic flow. The central nervous system compares prediction with incoming vestibular evidence. If they match, reflex responses to expected self-generated motion can be selectively shaped. If they do not, the discrepancy signals an external perturbation or an error in the current model.[2]

Internal models are also necessary because vestibular sensors do not directly provide every variable the organism needs. The canals measure rotational dynamics, while the otoliths measure gravito-inertial acceleration. To estimate earth-vertical orientation and translation, the brain mathematically combines them over time. The solution is effective but not perfect, particularly at low frequencies or when vision and proprioception disagree.[9]

Cortical vestibular processing

Vestibular perception is distributed across parietal opercular, insular, temporoparietal, somatosensory, visual-motion, hippocampal, and frontal networks. These regions contribute to self-motion perception, body ownership, verticality, spatial attention, navigation, and memory. Vestibular stimulation can modulate spatial neglect and body representation, illustrating that the system reaches far beyond reflex balance.[1]

Autonomic integration

Vestibular and visceral networks interact in the brainstem, cerebellum, hypothalamus, and cortex. Conflicting motion estimates can produce nausea, pallor, diaphoresis, and vomiting. The sensory-conflict concept of motion sickness is intuitive: the brain receives a pattern of vestibular, visual, and proprioceptive evidence that does not fit its learned internal model of movement.

CENTRAL LOCALIZATION PRINCIPLE

Peripheral lesions distort the **input**. Central lesions may distort comparison, time constants, coordinate transformations, gravity alignment, prediction, or the mapping from vestibular signals to eye and body responses. That is why central vestibular findings are often less stereotyped.

CHAPTER SYNTHESIS
- The vestibular nuclei are early multisensory processors, not passive relays.
- Commissural pathways sharpen bilateral comparison and contribute to acute unilateral imbalance.
- Floccular circuits calibrate VOR gain; nodulus/uvula circuits shape otolith-canal integration and velocity storage.
- Internal models combine sensory evidence with motor prediction to distinguish self-generated motion, external perturbation, tilt, and translation.
- Vestibular cortex is distributed and contributes to perception, attention, body representation, and navigation.

MOTOR CONSEQUENCES

8. Reflex outputs: keeping vision, head, and body stable

A vestibular signal becomes clinically visible when it moves the eyes, head, or body. The vestibulo-ocular reflex is the most precisely characterized output, but vestibulocollic and vestibulospinal pathways are equally important to function. All three must transform a head-centered motion estimate into commands appropriate for the mechanics and coordinate frames of their target muscles.

The purpose of the angular VOR

When the head turns, a stationary target would sweep across the retina unless the eyes rotate in the opposite direction. Retinal image motion is called retinal slip. The angular VOR minimizes slip by producing eye velocity that is approximately equal in magnitude and opposite in direction to head velocity. The reflex works in darkness and begins too quickly to depend on visual feedback, although vision later refines and calibrates it.

VOR gain is conventionally defined as eye velocity divided by head velocity, with sign conventions handled separately. An ideal compensatory horizontal VOR has a magnitude near 1.0 and approximately opposite phase. Gain below one leaves residual retinal slip in the direction of head movement. Excessive gain moves the image in the opposite direction. The appropriate gain can vary with frequency, viewing conditions, and behavior.

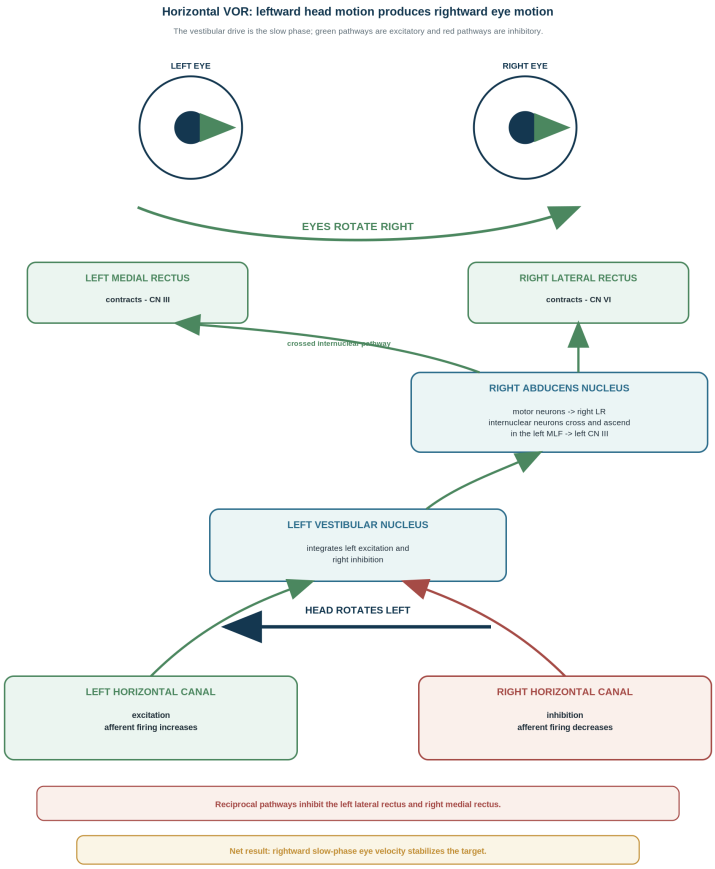

Figure 14. Original synthesis. The horizontal three-neuron VOR arc converts leftward head rotation into rightward slow-phase eye movement through the left vestibular nucleus, right abducens nucleus, and left MLF.

The horizontal three-neuron arc

Consider a leftward head turn. The left horizontal canal is excited and the right is inhibited. Left vestibular nuclear neurons excite the right abducens nucleus. Right abducens motor neurons contract the right lateral rectus. Abducens internuclear neurons cross and ascend in the left medial longitudinal fasciculus to the left oculomotor nucleus, activating the left medial rectus. Reciprocal inhibitory pathways relax the antagonists. The eyes move rightward while the head moves leftward.

1. Left horizontal canal afferent firing increases.
2. Left vestibular nuclear activity increases relative to the right.
3. Right abducens nucleus activates right lateral rectus and sends internuclear fibers to left oculomotor nucleus.
4. Left medial rectus contracts; antagonists are inhibited.
5. Both eyes rotate rightward to preserve fixation.

LOCALIZING WITH THE SLOW PHASE

Vestibular drive produces the **slow phase** of nystagmus. The fast phase is a central reset saccade. Nystagmus is named for the fast phase because it is easier to see, but physiology is understood from the slow phase.

Vertical and torsional canal pathways

Vertical canals activate combinations of vertical and torsional extraocular muscles aligned with the canal plane. Excitation of an anterior canal tends to drive the eyes upward with torsion; excitation of a posterior canal tends to drive the eyes downward with torsion. Because both eyes contribute differently and the canal planes are oblique, bedside vertical-canal nystagmus is typically mixed vertical-torsional rather than purely vertical. Remember that these are slow-phase eye movements generated by vestibular drive; the named fast phase points in the opposite direction.

Table 8. Conceptual canal-to-eye mapping. Positional nystagmus naming follows the fast phase and depends on viewing direction and canal geometry.

Excited canal	Dominant slow-phase eye movement	Fast-phase nystagmus direction
Left horizontal	Rightward	Left-beating
Right horizontal	Leftward	Right-beating
Left anterior	Upward with torsion away from the left ear	Downbeat with upper poles rotating toward the left ear
Right anterior	Upward with torsion away from the right ear	Downbeat with upper poles rotating toward the right ear
Left posterior	Downward with torsion away from the left ear	Upbeat with upper poles rotating toward the left ear
Right posterior	Downward with torsion away from the right ear	Upbeat with upper poles rotating toward the right ear

Visual-vestibular cooperation

The VOR dominates rapid head movements. Smooth pursuit and optokinetic responses contribute more at lower frequencies and when a visual scene moves. Together they maintain gaze across a broad range of behaviors. In light, visual feedback corrects residual retinal slip. In darkness, the VOR reveals the quality of the vestibular estimate more directly.

VOR cancellation is required when the eyes and head should move together, such as tracking a thumb held in front of the face while turning the trunk. The cerebellum suppresses the otherwise automatic counter-rotation. Impaired cancellation suggests central, often cerebellar, dysfunction, although attention and pursuit limitations can confound bedside testing.

The translational VOR

During lateral translation, the eyes rotate to maintain fixation on a target. The required response depends on target distance. A nearby target shifts more on the retina and requires greater compensatory eye movement than a far target. The brain therefore combines otolith signals with vergence and visual depth information. Translational VOR is particularly important during walking, when the head undergoes repeated linear movement.

Vestibulocollic reflexes

Vestibulocollic pathways activate neck muscles to stabilize the head in space and damp unexpected oscillation. The medial vestibulospinal tract descends bilaterally, mainly to cervical and upper thoracic levels, coordinating head and neck responses. These reflexes interact with cervicocollic reflexes driven by neck proprioception and with voluntary motor commands. The head is not simply held rigid; stabilization is task-dependent.[5]

Vestibulospinal reflexes

The lateral vestibulospinal tract arises largely from the lateral vestibular nucleus and descends ipsilaterally throughout the spinal cord, facilitating extensor and antigravity activity. The medial vestibulospinal tract descends bilaterally through the MLF and is especially important for head-neck coordination. Reticulospinal, cerebellar, proprioceptive, and visual pathways contribute heavily, so posture cannot be assigned to one tract.

Acute unilateral vestibular imbalance biases postural tone and can cause falling or veering toward the weaker side. This lateropulsion is the body-level counterpart of the ocular slow phase. As central tone rebalances, static lateropulsion improves, but gait in darkness or on uneven surfaces may remain impaired if dynamic vestibular information is inadequate.

Table 9. Major vestibular motor outputs and their clinical expression.

Output	Primary goal	Key pathways	Bedside consequence of failure
Angular VOR	Stabilize retinal image during rotation	Canal → vestibular nuclei → MLF → III/IV/VI	Corrective saccades, reduced dynamic visual acuity, oscillopsia
Translational VOR	Stabilize near targets during translation	Otolith + vergence/depth signals → ocular motor pathways	Visual blur during walking/translation
Vestibulocollic	Stabilize head relative to space/body	Medial vestibulospinal and cervical networks	Poor head stabilization, neck-dependent imbalance
Vestibulospinal	Maintain posture and antigravity tone	Lateral/medial vestibulospinal plus reticulospinal/cerebellar networks	Lateropulsion, imbalance, worse gait without visual support

OSCILLOPSIA FROM FIRST PRINCIPLES

If head velocity is 150°/s but the eyes counter-rotate at only 75°/s, VOR gain is 0.5. The image still moves across the retina at roughly 75°/s. The patient experiences the world as bouncing or blurring during movement.

CHAPTER SYNTHESIS

- The angular VOR minimizes retinal slip with an eye movement equal and opposite to head motion.
- The slow phase reflects vestibular drive; the fast phase resets the eyes and names the nystagmus.
- Translational VOR depends on viewing distance, whereas angular VOR gain is approximately distance-independent.
- Vestibulocollic and vestibulospinal pathways stabilize the head and body and explain lateropulsion and gait dysfunction.

PLASTICITY

9. Adaptation, substitution, habituation, and compensation

Vestibular symptoms can improve dramatically even when a damaged labyrinth does not recover. That improvement is not one mechanism. Static tone rebalances, reflex gains adapt, repeated provocative stimuli become less symptomatic, and visual or proprioceptive strategies substitute for missing information. Distinguishing these processes clarifies why early mobilization and vestibular rehabilitation help, why chronic suppressants can hinder recovery, and why some dynamic deficits persist. [13,14]

Static versus dynamic deficits

After sudden unilateral loss, static asymmetry produces spontaneous nystagmus, ocular tilt, severe vertigo, autonomic symptoms, and postural bias. Over days, resting activity in the vestibular nuclei rebalances through changes in commissural inhibition, intrinsic excitability, cerebellar input, and multisensory weighting. The spontaneous nystagmus and severe vertigo usually improve even if the peripheral asymmetry remains.

Dynamic deficits are harder. During a rapid head turn toward the weak side, the missing peripheral drive cannot be fully recreated by simply equalizing resting tone. VOR gain may remain low, and corrective saccades persist. Function improves through partial gain adaptation, predictive and compensatory saccades, visual substitution, and behavioral strategies, but recovery may be incomplete.

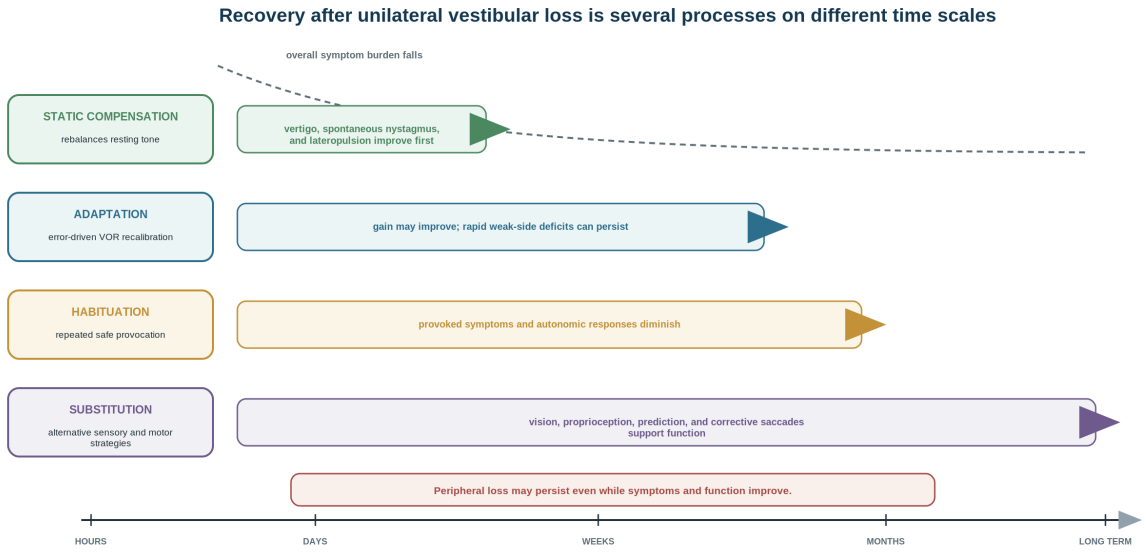

Figure 15. Original synthesis. Static compensation, adaptation, habituation, and substitution operate on different time scales; peripheral loss may persist despite functional recovery.

Four words that should not be used interchangeably

Table 10. Distinct components of functional recovery.

Process	Definition	Example
Compensation	Umbrella term for central recovery after vestibular injury	Rebalancing of vestibular nuclear resting activity after unilateral loss
Adaptation	Error-driven recalibration of a reflex or sensorimotor mapping	Increasing VOR gain when repeated retinal slip shows the eyes are moving too little
Habituation	Reduced response to repeated, non-harmful provocative stimulation	Less dizziness with repeated position changes
Substitution	Use of alternative sensory or motor strategies	Greater visual/proprioceptive reliance or planned corrective saccades

VOR adaptation and retinal slip

The cerebellum treats consistent retinal slip during head motion as an error signal. If the image repeatedly moves in the direction of the head, eye velocity is insufficient and VOR gain should increase. If the image moves opposite the head, gain may be excessive and should decrease. Magnifying and minifying lenses demonstrate this experimentally: the VOR recalibrates to the altered relationship between head movement and retinal motion.[12]

Gaze-stability exercises use the same principle. The patient fixes a target while turning the head fast enough to challenge the VOR but not so fast that the target becomes persistently unreadable. Repeated, direction-specific error drives adaptation and promotes compensatory saccades. Progression changes speed, target size, background complexity, stance, and walking conditions.

Habituation

Some movements provoke symptoms because they create sensory conflict or expose a residual asymmetry. Repeating a safe provoking movement can reduce the symptomatic and autonomic response. Habituation is not simply “pushing through.” The stimulus must be tolerable, repeatable, and sufficiently specific to induce learning without causing prolonged decompensation.

Substitution and sensory reweighting

When vestibular information is unreliable, the brain increases reliance on vision, proprioception, and prediction. This can improve function but create new vulnerabilities. A visually dependent patient may feel worse in grocery aisles, crowds, scrolling environments, or moving traffic. A proprioceptively dependent patient may deteriorate on foam, uneven ground, or in neuropathy. Rehabilitation aims for flexible weighting rather than rigid dependence on one substitute.

Why movement is therapeutic

Central recalibration requires error signals. If the head is kept still and vestibular suppressants blunt neural activity, the brain receives fewer opportunities to compare predictions with actual consequences. Once severe nausea and vomiting are controlled and dangerous central causes have been addressed, progressive mobilization generally supports compensation. Prolonged routine use of vestibular suppressants can slow adaptation and increase sedation and fall risk.

IMPORTANT NUANCE

Short-term suppressants may be appropriate for severe acute symptoms. The physiologic concern is **continued use after the acute phase**, when the therapeutic goal shifts from symptom suppression to exposure, recalibration, and movement.

Why compensation fails or remains incomplete

Recovery can be limited by bilateral loss, fluctuating disease, migraine, visual or somatosensory impairment, cerebellar dysfunction, anxiety and threat monitoring, deconditioning, poor sleep, sedating medication, and avoidance of head movement. The central system learns from the evidence it receives. If the signal is inconsistent or the patient avoids the situations needed for recalibration, the internal model may remain uncertain.

Compensation can also mask lesion severity. A chronically compensated patient may feel well at rest and have no spontaneous nystagmus, yet retain a marked unilateral vHIT deficit. Conversely, symptom severity does not map directly onto measured peripheral loss. Perception, visual dependence, migraine biology, autonomic responses, and psychological threat all shape the lived experience.

The bridge to bedside localization

Volume I has followed the signal from motion to recovery. Volume II will reverse that pathway. Starting with a patient's timing, triggers, eye movements, hearing, gait, and examination, it will ask which component of this system is failing. The aim is to move from a finding back to its mechanism: abnormal slow-phase vector to canal plane, corrective saccade to VOR deficit, skew to otolith-ocular imbalance, and test dissociation to stimulus frequency or end-organ selectivity.

FINAL FIRST-PRINCIPLES SUMMARY

Motion bends a mechanical element. Hair cells convert the bend into a bidirectional change in tonic firing. Paired sensors and commissural circuits compare the two sides. Central networks integrate canals, otoliths, vision, proprioception, and prediction. Reflexes stabilize the eyes, head, and body. Plasticity recalibrates the model when the sensors or environment change.

CHAPTER SYNTHESIS

- Static tone can rebalance even when peripheral loss persists; dynamic VOR deficits are more likely to remain.
- Adaptation, habituation, substitution, and compensation describe different mechanisms.
- Retinal slip drives VOR recalibration through cerebellar learning.
- Flexible sensory reweighting is helpful; rigid visual or proprioceptive dependence creates context-specific symptoms.
- Progressive motion provides the error signals required for recovery.

RAPID SYNTHESIS

APPENDIX A. High-yield physiology map

This appendix compresses the mechanistic story into a retrieval framework. It is not a substitute for the chapters; it is the final layer of condensation after the physiology has been understood.

Table 11. Mechanism-first interpretation of common findings.

If you observe…	The physiologic inference is…
Spontaneous unidirectional horizontal-torsional nystagmus	A sustained left-right vestibular tone imbalance is driving the slow phase; fast phase resets away from the slow phase.
Corrective saccade after a rapid head impulse	High-frequency VOR eye velocity was insufficient for the direction of the impulse.
Normal calorics but abnormal vHIT	Low-frequency horizontal canal response is preserved while high-frequency/high-acceleration response is impaired, or there is a technical/contextual issue.
Abnormal calorics but relatively normal vHIT	Low-frequency function is impaired but high-frequency canal function is preserved or centrally compensated.
Oscillopsia while walking	Residual retinal slip from inadequate dynamic gaze stabilization, often bilateral or severe unilateral loss.
Skew or ocular tilt reaction	Imbalance in graviceptive otolith-ocular pathways, often central when prominent in acute vestibular syndrome.
Worse balance in darkness or on foam	Visual or somatosensory substitution is being removed, exposing weak vestibular information.
Symptoms improve despite persistent vHIT deficit	Central compensation and substitution have improved function without restoring the peripheral sensor.

The full story in twelve steps

1. Head rotation or translation creates inertial force relative to the skull.
2. Canal endolymph or the otoconial membrane moves relative to the sensory epithelium.
3. The cupula or otolithic membrane bends polarized hair bundles.
4. Deflection toward the kinocilium increases mechanotransduction-channel opening.
5. K^+ influx depolarizes the hair cell and increases glutamate release.
6. Primary afferent firing rises or falls around a tonic baseline.
7. Paired canals and bilateral otolith populations create a differential population code.
8. Vestibular nuclei combine that code with visual, proprioceptive, cerebellar, and predictive signals.

9. Internal models estimate angular velocity, translation, tilt, and earth vertical.

10. Ocular motor pathways generate compensatory slow-phase eye movements.

11. Vestibulocollic and vestibulospinal pathways stabilize the head and body.

12. Cerebellar and distributed central plasticity recalibrate the system after error or injury.

Minimal equations worth remembering

VOR GAIN

Gain = eye velocity / head velocity. For an ideal compensatory angular VOR, the **magnitude** is near 1 and eye motion is opposite head motion.

GRAVITO-INERTIAL ACCELERATION

Otolith output reflects the vector combination of gravity and linear acceleration. The receptor alone cannot uniquely separate tilt from translation.

CANAL PAIR ESTIMATE

For a coplanar pair, the central rotation estimate is proportional to the difference between left- and right-sided activity. A unilateral resting loss therefore creates a false motion signal.

TERMS

APPENDIX B. Glossary

Vestibular language is compact and can hide assumptions. These definitions are phrased to preserve the physiology behind the term.

Table 12. Core physiology vocabulary.

Term	Mechanistic definition
Ampulla	Expanded end of a semicircular duct containing the crista and cupula.
Ampullofugal	Relative endolymph flow away from the ampulla.
Ampullopetal	Relative endolymph flow toward the ampulla.
Canal paresis	Caloric asymmetry suggesting reduced low-frequency horizontal canal pathway responsiveness on one side.
Compensation	Central recovery processes following vestibular injury.
Crista ampullaris	Sensory epithelium within a semicircular canal ampulla.
Cupula	Gelatinous structure spanning the ampulla and embedding

Term	Mechanistic definition
	crista hair bundles.
Efference copy	Internal copy of a motor command used to predict sensory consequences.
Gravito-inertial acceleration	Vector experienced by the otoliths that combines gravity and linear acceleration.
Kinocilium	Single cilium adjacent to the tallest stereocilia; defines the excitatory direction of a vestibular hair bundle.
Macula	Sensory epithelium of the utricle or saccule.
Ocular counter-roll	Partial torsional eye rotation opposite static head roll.
Otoconia	Calcium carbonate crystals that add mass to the otolithic membrane.
Retinal slip	Movement of an image across the retina.
Scarpa ganglion	Cell bodies of primary vestibular afferent neurons.
Striola	Curved region of polarity reversal within an otolith macula.
Velocity storage	Central network that prolongs and gravity-aligns rotational vestibular and optokinetic responses.
VOR cancellation	Suppression of the VOR when the eyes and head intentionally move together.
VOR gain	Ratio of eye velocity to head velocity, usually discussed by magnitude with direction considered separately.

EVIDENCE BASE

REFERENCES. Selected references

The guide prioritizes mechanistic reviews and foundational physiology. Numbering corresponds to bracketed citations in the text.

1. Angelaki DE, Cullen KE. Vestibular system: the many facets of a multimodal sense. Annu Rev Neurosci. 2008;31:125-150. doi:10.1146/annurev.neuro.31.060407.125555.

2. Cullen KE. The vestibular system: multimodal integration and encoding of self-motion for motor control. Trends Neurosci. 2012;35(3):185-196. doi:10.1016/j.tins.2011.12.001.

3. Rabbitt RD. Semicircular canal biomechanics in health and disease. J Neurophysiol. 2019;121(3):732-755. doi:10.1152/jn.00708.2018.

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

5. Vollrath MA, Kwan KY, Corey DP. The micromachinery of mechanotransduction in hair cells. Annu Rev Neurosci. 2007;30:339-365. doi:10.1146/annurev.neuro.29.051605.112917.

6. Curthoys IS, MacDougall HG, Vidal PP, de Waele C. Sustained and transient vestibular systems: a physiological basis for interpreting vestibular function. Front Neurol. 2017;8:117. doi:10.3389/fneur.2017.00117.

7. McPherson DR. Sensory hair cells: an introduction to structure and physiology. Integr Comp Biol. 2018;58(2):282-300. doi:10.1093/icb/icy064.

8. Bronstein AM, Patel M, Arshad Q. A brief review of the clinical anatomy of the vestibular-ocular connections - how much do we know? Eye. 2015;29:163-170. doi:10.1038/eye.2014.262.

9. Angelaki DE, Yakusheva TA. How vestibular neurons solve the tilt/translation ambiguity: comparison of brainstem, cerebellum, and thalamus. Ann N Y Acad Sci. 2009;1164:19-28. doi:10.1111/j.1749-6632.2009.03939.x.

10. Carriot J, Jamali M, Cullen KE. Rapid adaptation of multisensory integration in vestibular pathways. Front Syst Neurosci. 2015;9:59. doi:10.3389/fnsys.2015.00059.

11. Carriot J, Jamali M, Chacron MJ, Cullen KE. Statistics of the vestibular input experienced during natural self-motion: implications for neural processing. J Neurosci. 2014;34(24):8347-8357. doi:10.1523/JNEUROSCI.0692-14.2014.

12. Raymond JL, Lisberger SG, Mauk MD. The cerebellum: a neuronal learning machine? Science. 1996;272(5265):1126-1131. doi:10.1126/science.272.5265.1126.

13. Peusner KD, Shao M, Reddaway R, Hirsch JC. Basic concepts in understanding recovery of function in vestibular reflex networks during vestibular compensation. Front Neurol. 2012;3:17. doi:10.3389/fneur.2012.00017.

14. Lacour M, Helmchen C, Vidal PP. Vestibular compensation: the neuro-otologist's best friend. J Neurol. 2016;263(Suppl 1):S54-S64. doi:10.1007/s00415-015-7903-4.

15. Sadeghi SG, Minor LB, Cullen KE. Neural correlates of motor learning in the vestibulo-ocular reflex: dynamic regulation of multimodal integration in the macaque vestibular system. J Neurosci. 2010;30(30):10158-10168. doi:10.1523/JNEUROSCI.1368-10.2010.

16. Highstein SM, Holstein GR. The anatomy of the vestibular nuclei. Prog Brain Res. 2006;151:157-203. doi:10.1016/S0079-6123(05)51006-9.

17. OpenStax College. Anatomy & Physiology. Rice University; 2013. Vestibular system and vestibulo-ocular reflex illustrations licensed CC BY 3.0.

18. Poppi LA, Holt JC, Lim R, Brichta AM. A review of efferent cholinergic synaptic transmission in the vestibular periphery and its functional implications. J Neurophysiol. 2020;123(2):608-629. doi:10.1152/jn.00053.2019.

19. Pfeiffer C, Serino A, Blanke O. The vestibular system: a spatial reference for bodily self-consciousness. Front Integr Neurosci. 2014;8:31. doi:10.3389/fnint.2014.00031.

20. Goldberg JM, Wilson VJ, Cullen KE, et al. The Vestibular System: A Sixth Sense. Oxford University Press; 2012.

Figure and diagram credits

Table 13. Open-license web images and original diagrams used in Volume I.

Figure	Credit / license / source
Figure 1 and cover	Pfeiffer C, Serino A, Blanke O. "Peripheral vestibular organs in the inner ear." CC BY 3.0. Wikimedia Commons file page: https://commons.wikimedia.org/wiki/File:Peripheral_vestibular_organs_in_the_inner_ear.jpg
Figure 2	Original synthesis created for this guide.
Figure 3	NASA. "Roll, pitch, and yaw - the plane of motion of each

Figure	Credit / license / source
	semicircular canal." Public domain. https://commons.wikimedia.org/wiki/File:Roll,_pitch,_and_yaw-_the_plane_of_motion_of_each_semicircular_canal.jpg
Figure 4	Thomas Haslwanter. "Ampulla of SemicircularCanal." CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:Ampulla_of_SemicircularCanal.svg
Figure 5	Thomas Haslwanter. "Otoliths w CrossSection." CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:Otoliths_w_CrossSection.jpg
Figure 6	Cesar D. Fermin. "6OTO-SEM." CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:6OTO-SEM.jpg
Figure 7	Original synthesis created for this guide.
Figure 8	Masetto S, Spaiardi P, Johnson JS. "Auditory and vestibular hair cells and their innervation in adult mammals." CC BY 3.0; cropped to vestibular panel. https://commons.wikimedia.org/wiki/File:Auditory_and_vestibular_hair_cells_and_their_innervation_in_adult_mammals.png
Figure 9	Original synthesis created for this guide.
Figure 10	Thomas Haslwanter. "SemicircularCanals BodePlot." CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:SemicircularCanals_BodePlot.svg
Figures 11-13	Original syntheses created for this guide.
Figure 14	Original synthesis created for this guide.
Figure 15	Original synthesis created for this guide.