VOR Testing & HINTS Examination:
A Comprehensive Clinical Review

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Section 3B — VOR & Canal Assessment | Vestibular Function Testing Series

How to Use This Review

This document is the companion clinical literature review to the vestibulo-ocular reflex testing and the HINTS bedside battery video series on the ADC education hub at www.australiandizzinessclinics.com. It is designed for vestibular physicians, audiologists, and neurologists building expertise in laboratory vestibular function testing.

The review follows clinical testing sequence: from theoretical foundations and neural substrates through methodology, normative values, interpretation frameworks, and clinical application. Callout boxes throughout identify clinically high-yield points and evidence-based pearls.

Callout box guide:

 Clinical Insight: *Clinically relevant observations derived directly from the basic science — the bridge between laboratory findings and patient management.*

 Clinical Pearl: *High-yield, memorable clinical points — the key facts that separate a competent clinician from an expert in vestibular function testing.*

 Key Point: *Foundational concepts and summary statements that anchor the clinical framework. Master these to interpret the full testing battery.*

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Vestibulo-Ocular Reflex Function in Vestibular Diagnosis

Physiology, Examination, and Localisation

Purpose statement: The vestibulo-ocular reflex (VOR) is the core gaze-stabilizing reflex that allows clear vision during head motion and provides one of the highest-yield localisation windows in vestibular medicine. A careful understanding of VOR physiology, its central modulators, and its frequency-dependent testing (bedside, vHIT, caloric, and rotational paradigms) allows the vestibular physician and neuro-otologist to (i) distinguish peripheral from central causes of acute vestibular syndrome, (ii) localise lesions to end-organs, nerve divisions, nuclei, or cerebellar modules, and (iii) connect abnormal VOR behaviour to patients' symptoms such as vertigo, oscillopsia, and imbalance. This document complements a separate review focused on gaze-holding and spontaneous nystagmus mechanisms, which are tightly coupled to VOR circuitry in clinical practice.

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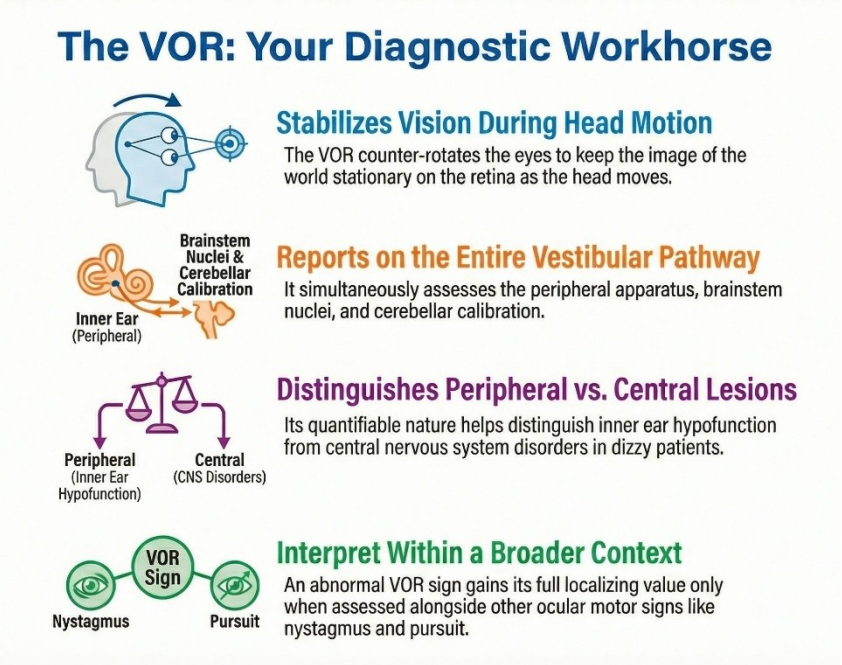


1. Introduction

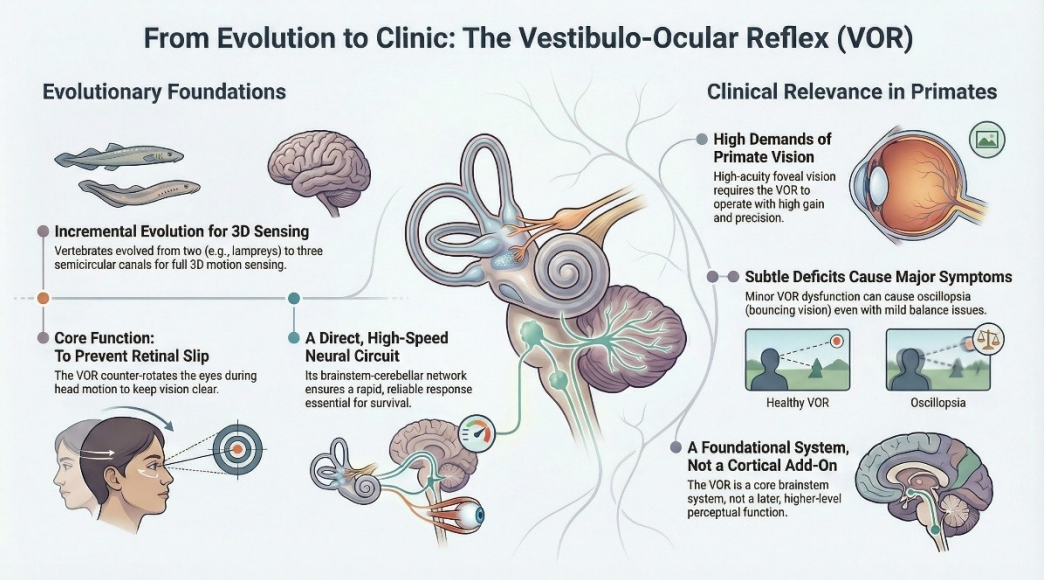
1.1 Clinical relevance: why VOR is the diagnostic workhorse

The VOR is the reflex that counter-rotates the eyes during head motion so that the image of the world remains near-stationary on the retina.

In a dizzy patient, this deceptively simple function becomes a localization lever: VOR behaviour simultaneously reports on peripheral transduction (hair cells and afferents), brainstem premotor transformation (vestibular nuclei and ocular motor nuclei), and cerebellar calibration (vestibulocerebellum). Because it is fast, stereotyped, and quantifiable, the VOR is among the most reliable bedside and laboratory "signals" for distinguishing peripheral vestibular hypofunction from central ocular motor disorders, particularly in acute vestibular syndrome and in chronic oscillopsia syndromes [1,2]. In clinic, the VOR is not assessed in isolation. Spontaneous nystagmus, gaze-holding, fixation suppression, and smooth pursuit all share anatomy and interact computationally with vestibular premotor networks. The practical outcome is that a single abnormal bedside sign (for example, an abnormal head impulse) only gains its full localisation meaning when interpreted alongside the broader ocular motor context.

1.2 Phylogenetic origin: why an ancient reflex still explains modern symptoms

The VOR is an evolutionarily ancient solution to a physical constraint: if the head moves and the eyes do not counter-rotate, retinal slip degrades vision. Among vertebrates, the semicircular canals and otolith organs evolved as highly conserved inertial sensors, and reflexive eye movements driven by vestibular inputs are present across vertebrate classes from fish to mammals [3]. This deep conservation is clinically relevant because it explains why VOR circuitry is organized as a relatively direct premotor network: survival advantage favoured a short-latency, high-reliability transformation from head motion to compensatory eye motion [1,3].



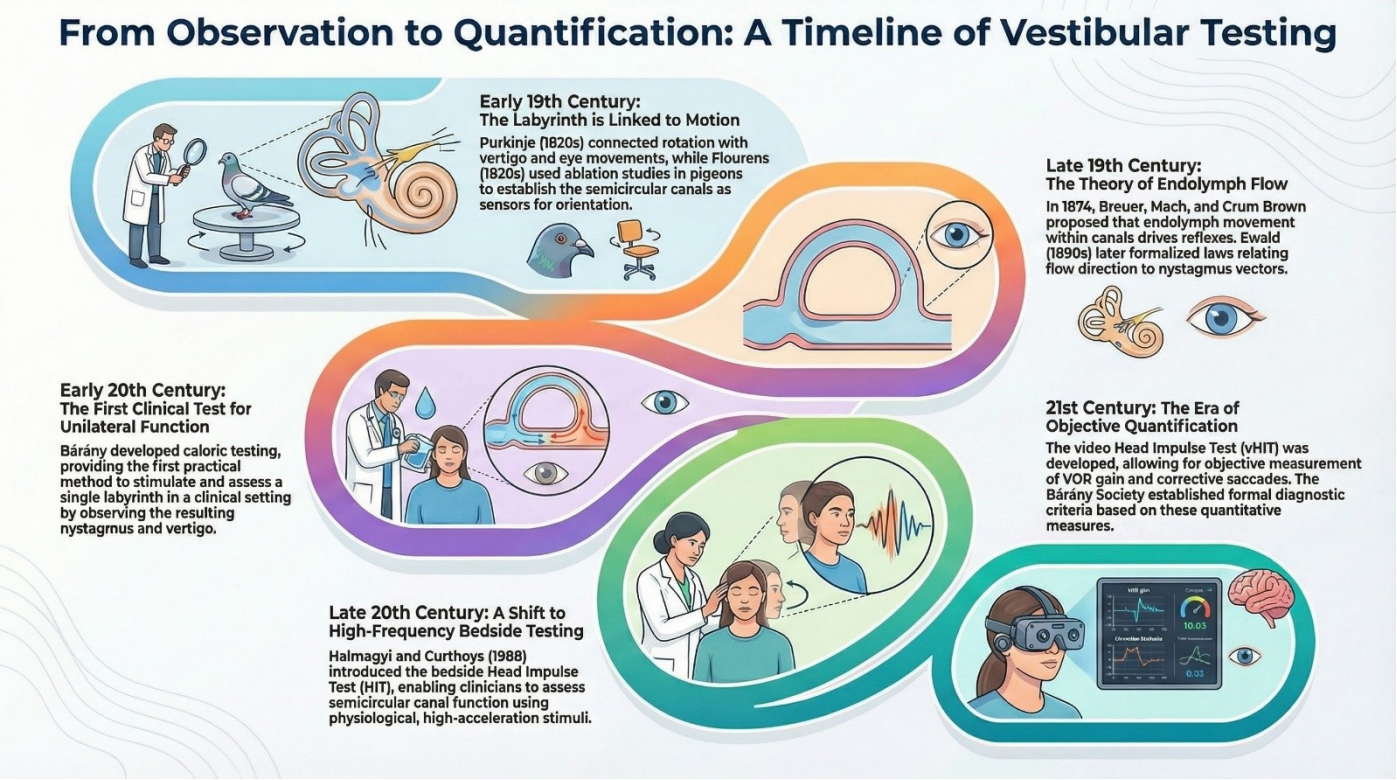
Comparative vestibular biology also clarifies how "three canals" became a standard. Early vertebrate lineages show incremental acquisition of canal structures; for example, lampreys possess two semicircular canals rather than the triad typical of gnathostomes, illustrating an evolutionary progression toward full three-dimensional angular sensing and correspondingly richer vestibulo-ocular control [4]. The vestibular apparatus and its central projections are therefore not a late cortical add-on but a foundational brainstem–cerebellar system upon which higher perceptual functions were later layered [3,4].

In primates, the VOR operates in the context of high-acuity foveal vision and frequent head-on-body movements during locomotion and exploration. This combination pushes the VOR toward high gain, broad bandwidth, and tight integration with saccades and pursuit. The consequence is that subtle deficits can be symptomatically obvious (oscillopsia) even when "balance" complaints are mild, because visual stability requirements are stringent [1,3].

1.3 Historical milestones in VOR understanding and testing

Modern vestibular testing is built on a sequence of conceptual breakthroughs that progressively linked labyrinthine anatomy to eye movements and then to quantitative clinical tests.

Early 19th century: observation and experimental lesioning. Purkinje systematically studied post-rotational phenomena and eye movements during and after rotation (early 1820s), linking vertigo and eye motion as coupled outputs of rotation [5]. Experimental ablation studies by Flourens in pigeons (1820s) demonstrated that damaging semicircular canals produced characteristic disordered movements and turning behaviour, helping establish the labyrinth as a sensor for orientation and motion rather than an anatomical curiosity [6].



Late 19th century: semicircular canal theory and vector laws. In 1874, Breuer, Mach, and Crum Brown independently advanced a theory (now broadly correct) that endolymph movement within semicircular canals drives reflexive responses, including nystagmus, during rotation [7]. Ewald's experiments in the 1890s formalized "laws" relating endolymph flow direction to response magnitude and canal-plane specificity, providing principles still used clinically to interpret canal stimulation and nystagmus vectors [8].

Early 20th century: caloric testing and clinical vestibular function. Bárány's work on the caloric reaction provided the first practical method for unilateral labyrinth testing in humans and established an enduring link between controlled vestibular stimulation and observable nystagmus/vertigo [7].

Late 20th century: bedside impulse testing and modern frequency thinking. Halmagyi and Curthoys (1988) described the bedside head impulse test as a clinical sign of semicircular canal paresis, shifting bedside vestibular diagnosis toward high-acceleration, physiological stimuli rather than low-frequency surrogates [9].

21st century: quantification with video technology and refined diagnostic criteria. vHIT extended the head impulse paradigm to objective measurements of canal-specific VOR gain and corrective saccades, while consensus criteria from the Bárány Society formalized test thresholds for syndromes such as bilateral vestibulopathy and acute unilateral vestibulopathy [10,11].

2. Neurophysiology and anatomical substrates

2.1 Peripheral vestibular sensors: semicircular canals and otolith organs

The vestibular end organs provide the “input space” for the VOR. The semicircular canals (horizontal/lateral, anterior/superior, posterior) encode angular acceleration of the head. Hair cells within the crista ampullaris transduce cupular deflection caused by endolymph motion into changes in afferent firing. This encoding is directionally organized: each canal is paired with a contralateral coplanar partner in a push–pull arrangement, so head rotation excites one canal while inhibiting its paired partner. This push–pull architecture improves sensitivity and extends dynamic range by allowing differential coding of motion direction [1,2].

The otolith organs (utricle and saccule) encode linear acceleration and gravity-relative head orientation. They contribute to the translational vestibulo-ocular reflex (tVOR), particularly important for near viewing where translational head movement produces large retinal slip if uncompensated. The tVOR is inherently distance-dependent: the eye rotation required to stabilize a near target during translation is greater than for a far target, and otolith-driven pathways are structured to incorporate such target distance information in their effective output [12].

A clinically crucial concept is that vestibular tests are not interchangeable “measures of vestibular function.” They interrogate different stimulus domains. Caloric stimulation approximates a very low-frequency canal response; rotational chair testing probes mid-frequencies; head impulse paradigms probe high-frequency, high-acceleration function. The peripheral organs and central networks respond differently across these domains, so discordant test results can reflect true physiological frequency dependence rather than error [2].

2.2 Vestibular afferents and nerve divisions

Primary vestibular afferents have cell bodies in Scarpa’s ganglion and project centrally via the vestibular nerve, which is functionally divided into superior and inferior divisions. The superior division predominantly carries signals from the horizontal and anterior canals and the utricle; the inferior division carries signals from the posterior canal and saccule [2]. This division matters clinically because selective involvement (for example, superior division vestibular neuritis) yields a predictable canal pattern on vHIT (horizontal and anterior canal deficits with relative posterior canal sparing) and corresponding characteristic nystagmus vectors [1,2].

The peripheral-to-central transformation begins at the vestibular nuclei, but vestibular primary afferents also project directly to vestibulocerebellar structures as mossy fibres, providing a pathway for rapid cerebellar modulation of reflex gain and spatial orientation processing [13].

2.3 Vestibular nuclei as premotor hubs

A distinctive feature of vestibular processing is that the first central synapse sits in a premotor

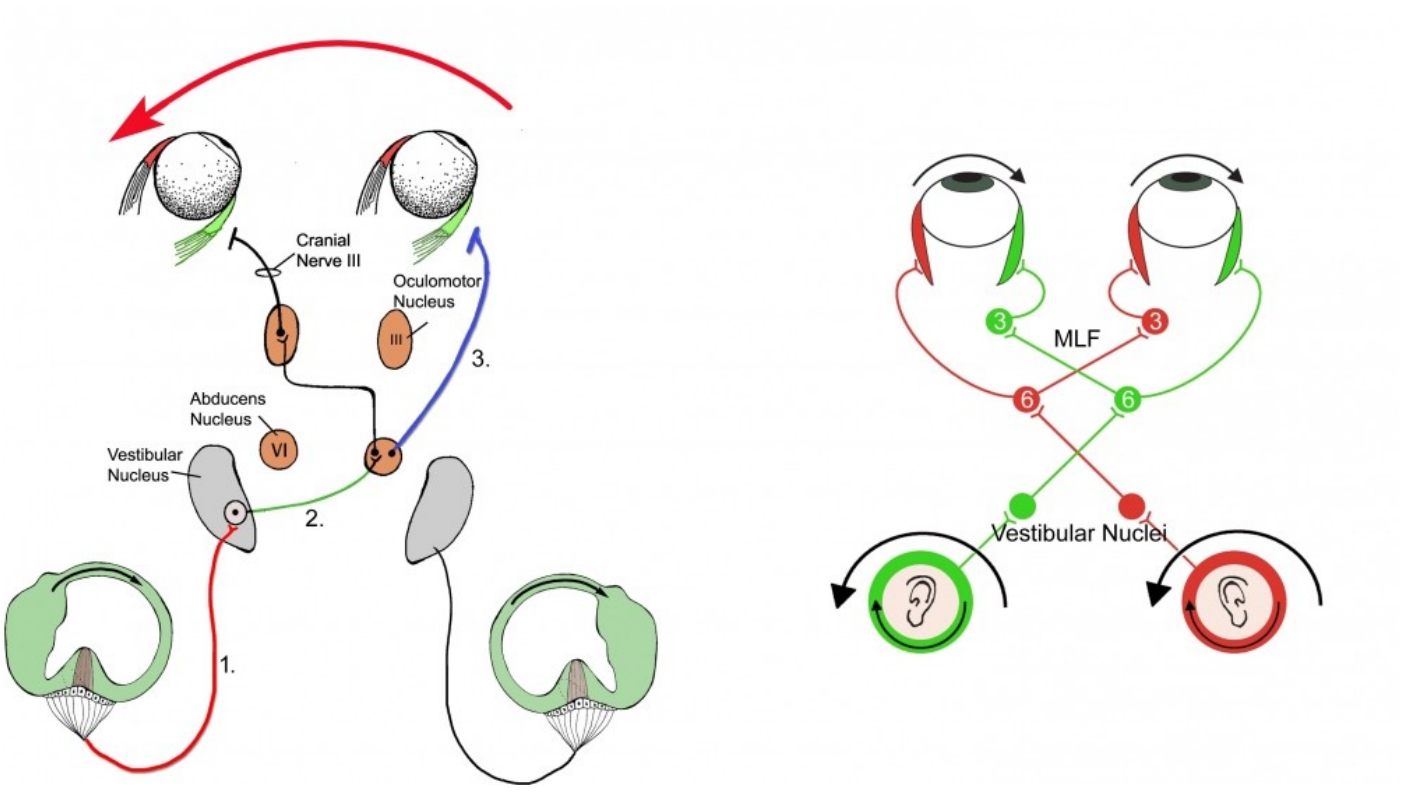
system: vestibular nucleus neurons are simultaneously sensory recipients and direct contributors to motor outputs. The vestibular nuclei complex (classically superior, medial, lateral/Deiters, and inferior/descending nuclei) integrates vestibular afferents with visual, proprioceptive, and efference-copy signals and sends outputs to ocular motor nuclei, spinal cord pathways, cerebellum, and thalamocortical circuits [14].

In the context of the VOR, the medial and superior vestibular nuclei are especially important for ascending vestibulo-ocular pathways. The vestibular nuclei also contain commissural inhibitory connections linking left and right sides, which are important for push–pull dynamics, compensation after unilateral loss, and velocity storage behaviour [1,2].

2.4 The three-neuron arc and plane-specific VOR pathways

The canonical angular VOR is often described as a three-neuron arc: hair cell/primary afferent, vestibular nucleus neuron, ocular motor neuron. This architecture underpins the VOR's short latency and robustness [1,2].

Horizontal canal pathway. Rightward head rotation excites the right horizontal canal and inhibits the left. Excitatory vestibular nucleus neurons project to the contralateral abducens nucleus. Abducens motor neurons drive the ipsilateral lateral rectus, while abducens internuclear neurons cross via the medial longitudinal fasciculus (MLF) to the oculomotor nucleus to drive the contralateral medial rectus. The net result is conjugate leftward eye rotation to compensate for rightward head rotation [1,2]. Parallel inhibitory pathways suppress antagonists. This produces approximately equal and opposite eye velocity relative to head velocity when gain is near unity.



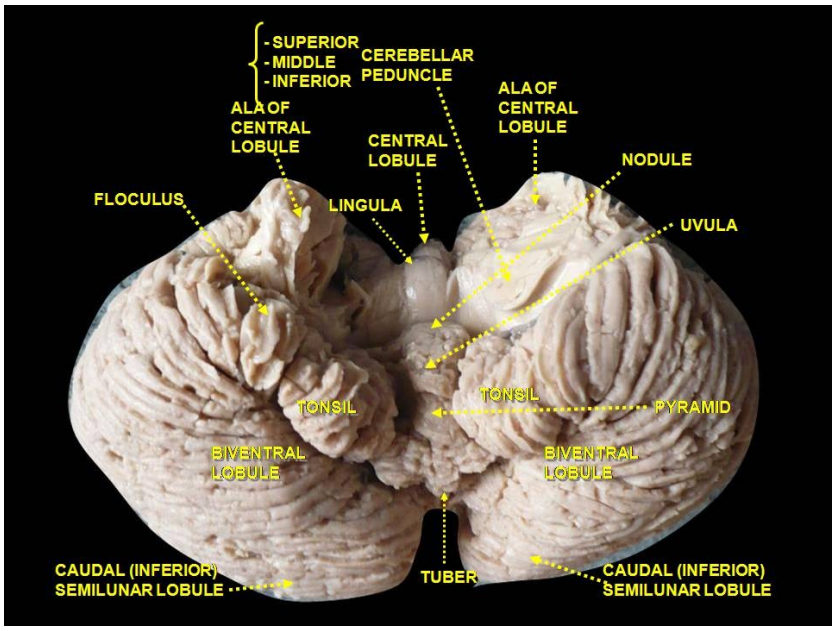
Vertical and torsional canal pathways. Anterior and posterior canal signals project through vestibular nuclei to the oculomotor and trochlear nuclei to drive vertical and torsional components appropriate to the canal plane. Clinically, these pathways are assessed by aligning head

impulses with the LARP (left anterior–right posterior) and RALP (right anterior–left posterior) planes during vHIT or skilled bedside vertical HIT, enabling canal-specific localization [2].

Translational VOR pathways. Otolith-driven ocular reflexes generate compensatory eye movements during head translation. Unlike aVOR, tVOR requires integration of head translation, target distance, and often vergence state; thus, it relies on more complex computations and tends to show greater variability in clinical testing. In many routine vestibular clinics, otolith contributions are often inferred indirectly (for example, skew deviation in acute vestibular presentations) or assessed with VEMP paradigms rather than direct tVOR quantification [1,2].

2.5 Cerebellar modules: calibration, adaptation, and velocity storage

The vestibulocerebellum is not merely an accessory; it is an essential adaptive controller that keeps the VOR accurate over the lifespan while allowing flexible suppression when appropriate.



Flocculus/paraflocculus: gain calibration and cancellation. The flocculus and ventral paraflocculus modulate VOR gain and contribute to the ability to suppress or cancel the VOR during combined head–target motion (for example, tracking a target moving with the head). Experimental work demonstrates that floccular region integrity is necessary for normal voluntary VOR cancellation behaviours [15].

Nodulus/uvula: spatial orientation and velocity storage control. The nodulus and uvula play key roles in controlling the velocity storage mechanism and aligning eye velocity with gravito-inertial acceleration, including effects that produce cross-coupled eye movements when head orientation changes relative to gravity [16].

Cerebellar learning and VOR adaptation. VOR gain is plastic. Wearing magnifying or minifying lenses induces VOR gain adaptation over hours to days, and cerebellar mechanisms (including Purkinje cell learning rules) are essential for this adaptive calibration. Classic reviews describe the cerebellum as a key site for motor learning in the VOR [17,18].

Clinical relevance of cerebellar modules. In practice, the vestibular physician sees these modules “leak” as specific patterns: **impaired cancellation (flocculus), abnormal velocity storage signatures such as periodic alternating nystagmus (nodulus/uvula), and gaze-evoked drift due to ocular motor integrator dysfunction**. The companion review on gaze-holding mechanisms develops these patterns and their neuroanatomical substrate in detail.

2.6 Visual–vestibular integration and cancellation

The VOR operates synergistically with visually driven reflexes, especially optokinetic responses, which help stabilize gaze during sustained low-frequency motion and contribute to after-

nystagmus phenomena. Visual tracking (smooth pursuit) can also be used to suppress vestibular-driven eye movements when the target is head-fixed. Mechanistically, VOR cancellation likely uses a combination of reducing vestibular reflex gain and adding pursuit-driven eye velocity in the opposite direction; models and experimental evidence support multiple complementary mechanisms rather than a single switch [19].

This integration is clinically exploited: if a patient cannot suppress the VOR during head-and-body rotation while attempting to fixate a head-fixed target, the deficit is typically central, often pointing toward cerebellar dysfunction (especially floccular region involvement) [1,2,15].

2.7 Functional anatomy summary

The VOR can be conceptualized as a layered system. The peripheral sensors (canals and otoliths) transduce head motion into afferent firing; vestibular nuclei transform these signals into premotor commands to ocular motor nuclei via stereotyped plane-specific pathways; commissural networks and integrator-like elements shape temporal dynamics; and cerebellar modules (flocculus, nodulus/uvula) adapt, calibrate, and spatially align the output while coordinating suppression and integration with vision. Clinical VOR patterns emerge from how and where in this hierarchy the system is damaged or dysregulated [1,2].

3. Mechanisms and classification

3.1 Core definitions: gain, phase, time constants, and frequency response

The clinically useful descriptors of VOR performance are gain, phase, symmetry, and time constant.

Gain is the ratio of eye velocity to head velocity (for aVOR) with sign convention such that ideal compensation yields gain near 1.0 with opposite direction to head motion. Phase describes timing: an ideal VOR produces eye velocity nearly 180° out of phase with head velocity for sinusoidal stimuli (i.e., equal and opposite), minimizing retinal slip. Asymmetry captures side-to-side differences, often reflecting unilateral vestibular hypofunction. Time constants describe how quickly vestibular-driven nystagmus decays after a step change in rotation; these depend on both peripheral canal biomechanics and central processing (notably velocity storage) [2].

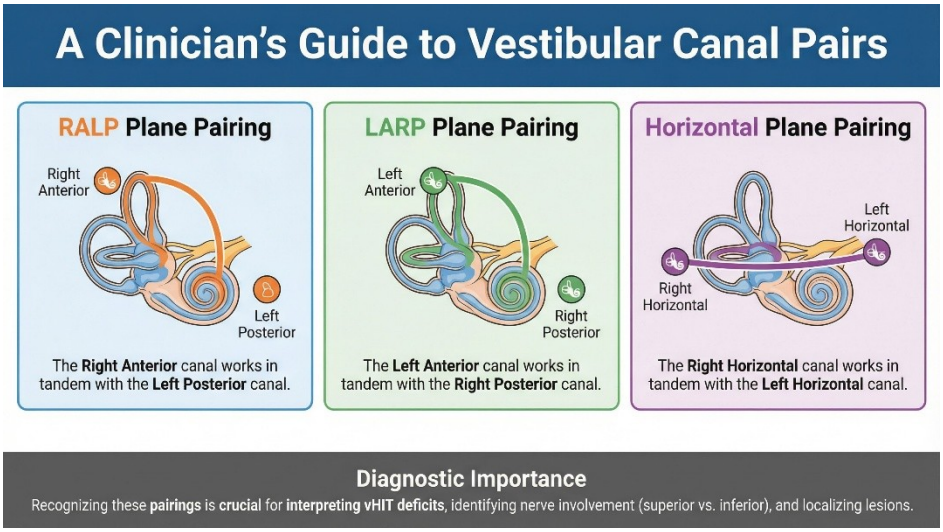
VOR bandwidth is clinically essential. The aVOR is designed to be effective at high frequencies and accelerations typical of natural head movements. Visual feedback loops are slower and cannot fully stabilize vision during rapid head motion, which is why loss of high-frequency aVOR function produces movement-induced blur and oscillopsia even when smooth pursuit is intact [1,2].

3.2 Classification by stimulus type: angular vs translational VOR

Angular VOR (aVOR) is semicircular canal-mediated and is the primary focus of bedside HIT, vHIT, caloric, and rotational chair testing. Translational VOR (tVOR) is otolith-mediated and becomes especially relevant for near targets during linear head motion; it is less directly assessed in routine vestibular test batteries but is implicated in certain symptom profiles and in complex ocular motor presentations [12].

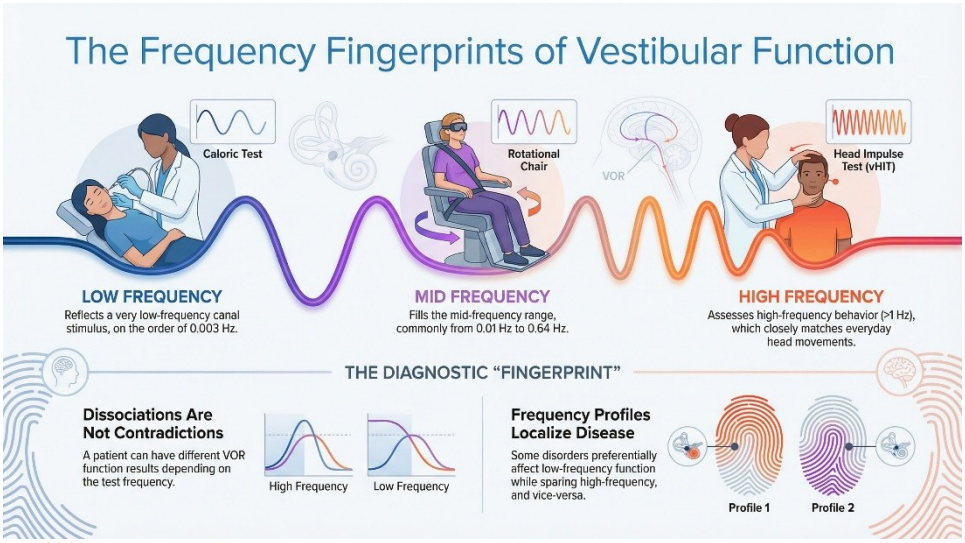
3.3 Classification by plane and canal pair

Clinically, VOR is best understood in canal planes rather than "horizontal versus vertical" simplifications. Each semicircular canal drives eye movements aligned with its plane, and canal pairs act as functional units. For example, the right anterior canal pairs with the left posterior canal (RALP plane), and the left anterior pairs with the right posterior (LARP plane). Recognizing these pairings allows clinicians to interpret vertical-torsional vHIT deficits and to connect them to nerve division involvement (superior vs inferior) and to localize peripheral end-organ lesions [2].



3.4 Classification by test frequency band (caloric, rotational, impulse)

The same patient can have different "VOR function" depending on frequency. A clinically useful approximation is that **caloric responses reflect a very low-frequency canal stimulus** on the order of 0.003 Hz, **rotational chair testing fills the mid-frequency range** (commonly around 0.01–0.64 Hz in sinusoidal paradigms), and head impulse/vHIT assesses high-frequency behaviour (>1 Hz, often several Hz) that more closely matches everyday head movements [20,21].

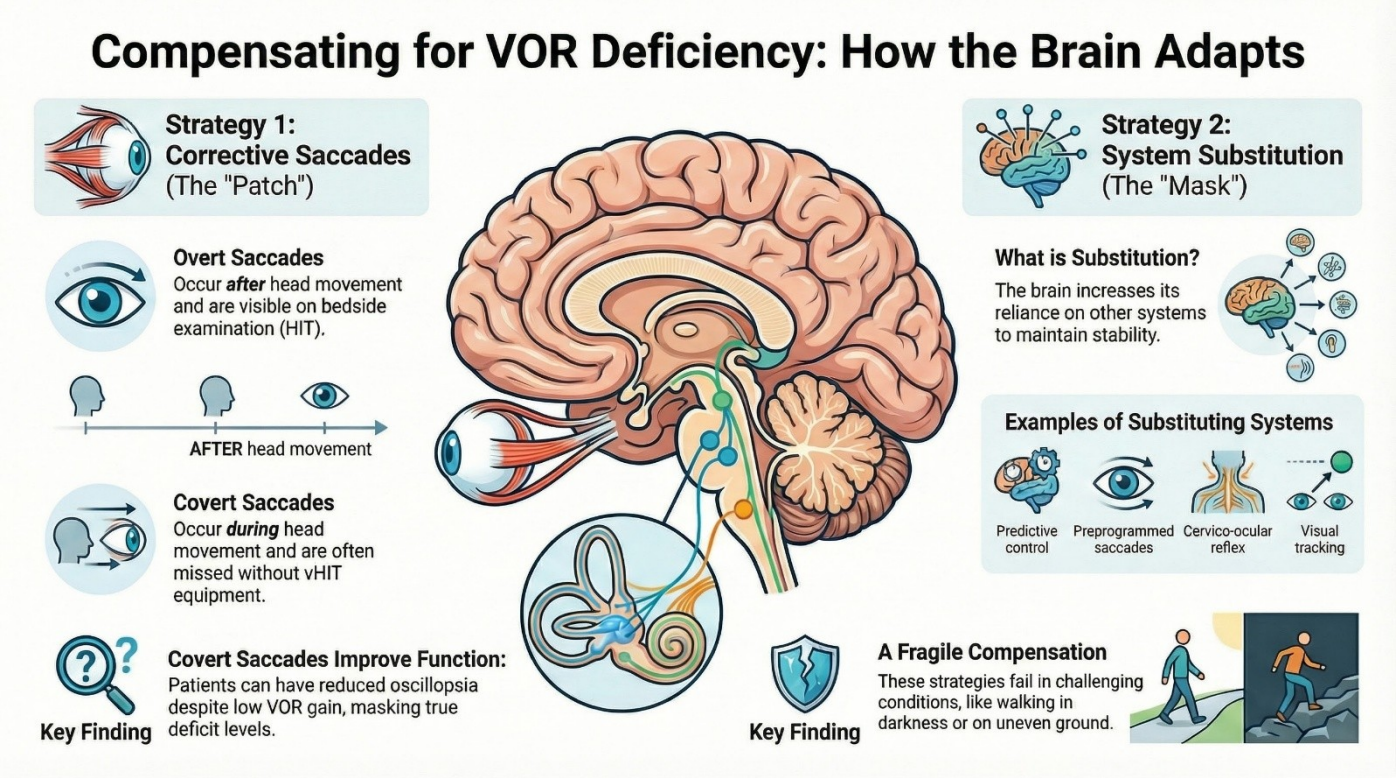


This frequency stratification is not a technical detail; it is one of the main reasons VOR testing localises disease. Some disorders preferentially affect low-frequency function (or its test surrogates) while sparing high-frequency performance, and vice versa. When interpreted correctly, such dissociations are not contradictions but fingerprints.

3.5 Compensation and plasticity: how the system fails (and how it "cheats")

When the VOR is deficient, the nervous system uses compensatory strategies. In unilateral vestibular loss, spontaneous nystagmus reflects baseline asymmetry in vestibular tone. With time, central compensation reduces this asymmetry and suppresses spontaneous nystagmus, but dynamic deficits may

persist.



During rapid head motion, the brain can “patch” gaze stability with corrective saccades. Overt saccades occur after the head movement and are visible on bedside HIT. Covert saccades occur during the head movement and may be missed at bedside but appear on vHIT traces. Clinically, covert saccades can improve functional gaze stability and reduce oscillopsia despite persistently low VOR gain, which is why symptom severity does not always scale linearly with gain metrics [2].

A separate axis of compensation is substitution: increased reliance on predictive control, preprogrammed saccades, cervico-ocular reflex contributions, and visual tracking. These strategies can mask deficits on some tasks while leaving patients impaired in others (for example, walking in darkness or on uneven ground).

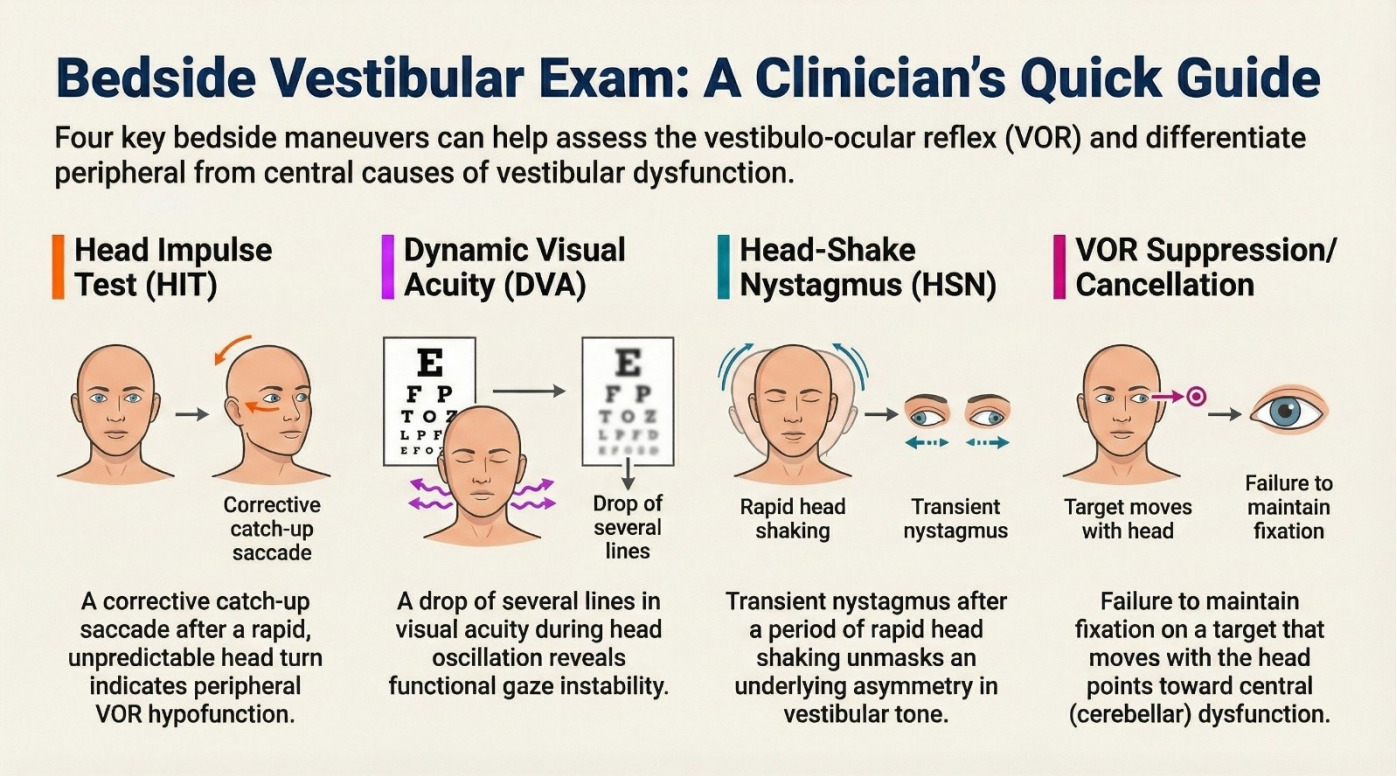
4. Clinical examination methodology

4.1 Bedside head impulse test (HIT): protocol and technical pitfalls

The bedside horizontal HIT is the most direct clinical test of high-frequency aVOR integrity. The method is conceptually simple: deliver brief, small-amplitude, unpredictable head impulses while the patient fixates a stationary target and observe whether the eyes remain on target. A corrective catch-up saccade indicates insufficient VOR output for that impulse direction. The clinical sign was formalized by Halmagyi and Curthoys in 1988 [9].

Technique matters. The head impulse should be small amplitude (often about 10–20°), high acceleration, and unpredictable in timing and direction. The patient should be instructed to keep eyes on the target and not “help.” The examiner should avoid pre-rotations or slow drifts that allow predictive strategies. The clinician should also ensure the neck is safe to move and consider contraindications such as cervical instability or severe pain.

Vertical canal bedside HIT is more challenging and less reliable without video-oculography. When attempted, impulses should be delivered in the LARP and RALP planes with appropriate head pitch and gaze alignment, but clinicians should generally confirm vertical canal deficits with vHIT because torsional components and subtle vertical saccades are easily missed [2].



4.2 Dynamic visual acuity and gaze-stability bedside proxies

Dynamic visual acuity (DVA) testing measures the functional consequence of VOR failure: loss of visual acuity during imposed head motion. Classic work demonstrates that DVA during head motion provides a clinically feasible measure related to oscillopsia and functionally significant VOR abnormalities [22].

At bedside, simplified DVA can be performed by measuring static acuity and then repeating acuity testing while the clinician oscillates the patient's head at a modest frequency and amplitude. A drop of several lines suggests impaired gaze stabilization, though the test is influenced by visual factors, attention, and technique. In a vestibular clinic, DVA is often used as a functional adjunct rather than a localisation tool.

4.3 Head-shake and vibration-evoked responses as asymmetry probes

Head-shake nystagmus (HSN) is a bedside method to unmask asymmetry in vestibular tone and dynamic responses. The head is rapidly oscillated for a brief period (commonly ~20 cycles) and then stopped; the clinician observes for transient nystagmus. A sustained post-headshake nystagmus suggests peripheral asymmetry but is less specific than HIT, as central disorders of

velocity storage and cerebellar modulation can also shape HSN behaviour [2]. Head vibration (mastoid vibration) can similarly evoke nystagmus in unilateral hypofunction.

These tests are best interpreted as “asymmetry detectors” rather than “canal-specific measures.” They gain localisation value when combined with HIT/vHIT patterns and spontaneous nystagmus direction.

4.4 VOR suppression/cancellation: cerebellar bedside window

VOR cancellation is tested by asking the patient to fixate a target that moves with the head, such as the patient’s thumb held at arm’s length while the clinician rotates the patient’s head-and-body together. In this condition, an intact system should suppress vestibular-driven compensatory eye movements and instead maintain fixation on the head-fixed target. Failure to cancel the VOR produces corrective saccades and/or nystagmus-like oscillations and points toward central dysfunction, particularly cerebellar (flocculus/paraflocculus) involvement [15,19].

In practice, this is one of the most clinically useful bedside differentiators between peripheral vestibular hypofunction (which typically preserves cancellation) and cerebellar disease (which often impairs cancellation even when basic aVOR pathways are intact).

4.5 Documentation standards for clinical notes

Because VOR findings are vectorial and frequency-dependent, documentation should be explicit. A minimal standard for the vestibular physician is to document: the plane tested (horizontal, LARP/RALP if applicable), laterality (rightward vs leftward impulses), qualitative result (normal/abnormal), whether corrective saccades were overt (visible) or suspected covert (not visible but possible), and any constraints (neck pain, poor fixation, visual limitations). For DVA, record static acuity, dynamic condition (approximate head oscillation frequency), and line loss. For cancellation, record whether the patient could maintain fixation during head-and-body rotation and whether saccades were observed.

5. Interpretation framework

5.1 Normal versus abnormal: what “VOR failure” looks like

A normal VOR keeps the eyes on target during head motion with minimal retinal slip. In a healthy human, initial VOR responses begin within roughly 7–10 ms after onset of head rotation, consistent with a short-latency reflex arc [12].

Clinically, abnormal VOR manifests in two complementary ways: deficient slow-phase compensation (low gain) and compensatory corrective saccades. Bedside HIT detects overt catch-up saccades. vHIT provides quantitative gain and can detect covert saccades that occur during the impulse, which may be symptomatically relevant because covert saccades can reduce oscillopsia by “rescuing” gaze stability within the movement.

The VOR can also be abnormal in less intuitive ways. Cerebellar disorders can produce inappropriate gains (too high or too low), impaired adaptation, abnormal time constants, or impaired suppression/cancellation rather than simple unilateral hypofunction.

5.2 Red flags for central pathology

The diagnostic power of VOR assessment peaks in acute vestibular syndrome (AVS), where central causes (especially posterior circulation stroke) may mimic peripheral vestibular loss. The HINTS examination (Head-Impulse, Nystagmus, Test-of-Skew) leverages VOR physiology: a normal horizontal head impulse in a patient with continuous vertigo and spontaneous nystagmus is a major red flag for central pathology, whereas an abnormal head impulse supports a peripheral lesion but is not fully excluding. The original HINTS study and subsequent work demonstrate high sensitivity for stroke when performed by trained clinicians, with HINTS outperforming early diffusion-weighted MRI in some settings [23,24].

The reason is mechanistic. Many ischemic lesions that cause AVS spare the peripheral end organ and the three-neuron arc, leaving the high-frequency aVOR intact; instead, they disrupt central integration, gaze holding, or vestibular nucleus/cerebellar circuits, producing direction-changing nystagmus, skew deviation, and impaired cancellation. Conversely, some strokes (notably AICA territory) can affect both central and peripheral structures, producing abnormal head impulse tests and thus potentially misleading “peripheral-appearing” patterns. This is not a paradox; it reflects vascular supply realities. Contemporary analyses emphasize these pitfalls and the need for full ocular motor context rather than reliance on a single sign [24].

5.3 Modifiers and confounders (age, drugs, ocular motor comorbidity)

Interpreting VOR findings requires attention to modifiers that can change either the reflex itself or the measurement.

Age and presbyvestibular changes can reduce vestibular function and increase reliance on compensatory strategies; however, many older adults maintain near-normal vHIT gains, so age alone should not be used to “explain away” gross abnormalities. Sedatives, alcohol, anticonvulsants, and other centrally acting drugs can alter nystagmus behaviour and pursuit/cancellation, potentially mimicking cerebellar dysfunction.

Ocular motor comorbidities are common confounders. Internuclear ophthalmoplegia, gaze palsies, or ocular motor nerve paresis can produce corrective saccades or gaze instability that may be mistaken for VOR deficits. Visual acuity limitations can also degrade fixation and generate apparent catch-up saccades.

Finally, mechanical factors matter: limited neck mobility, pain, or examiner hesitancy can reduce head impulse acceleration, potentially yielding a false “normal” bedside HIT even in peripheral hypofunction.

5.4 Integrating bedside and laboratory results

The central logic of integrating VOR tests is frequency coherence. Bedside HIT and vHIT probe high-frequency function. Caloric testing probes very low-frequency canal responses and is sensitive to mild unilateral hypofunction. Rotational chair fills mid-frequency gaps and is valuable for bilateral loss and for evaluating time constants/velocity storage behaviour.

Therefore, a coherent interpretation expects internal consistency within each frequency band and explains cross-band dissociations physiologically. For example, a patient with bilateral

vestibulopathy should show reduced aVOR across modalities, whereas a patient with Ménière's disease may show low-frequency deficits (abnormal calorics) with relatively preserved high-frequency gains on vHIT, reflecting disease-specific pathophysiology rather than measurement error [25].

5.5 Inter-test dissociations and how to reason about them

The most clinically important dissociations include **caloric–vHIT dissociation, canal-specific patterns (superior vs inferior division lesions), and cancellation abnormalities** with otherwise preserved gains.

Caloric–vHIT dissociation. Caloric tests and vHIT probe different frequency domains; caloric responses approximate extremely low-frequency stimulation (~0.003 Hz), whereas vHIT probes high-frequency impulses. Dissociation (abnormal caloric with normal vHIT) is a recognized pattern in disorders such as Ménière's disease and endolymphatic hydrops, and multiple mechanisms have been proposed including hydropic changes altering thermal responses or canal mechanics [20,25].

Canal-division patterns. Selective deficits on vHIT can localise pathology to superior versus inferior vestibular nerve divisions. Recent work highlights that vestibular neuritis can involve both divisions more often than assumed; nevertheless, canal-specific vHIT and otolith tests (VEMPs) can refine localisation across the spectrum of acute vestibular neuropathy [26].

Cancellation abnormalities. Impaired VOR cancellation with relatively preserved basic gains strongly implicates cerebellar involvement, particularly floccular regions and their interaction with pursuit systems [15,19].

6. Central versus peripheral differentiation

6.1 Structured comparison framework

Peripheral vestibular hypofunction typically produces a consistent syndrome of unilateral VOR deficiency (abnormal head impulse toward the lesion), unidirectional horizontal-torsional spontaneous nystagmus that follows Alexander's law and suppresses with fixation, and preserved VOR cancellation (unless patient cooperation or vision is limiting). Central disorders are more likely to show direction-changing gaze-evoked nystagmus, vertical nystagmus, impaired gaze holding, skew deviation, and impaired VOR cancellation even when head impulse is normal [1,2].

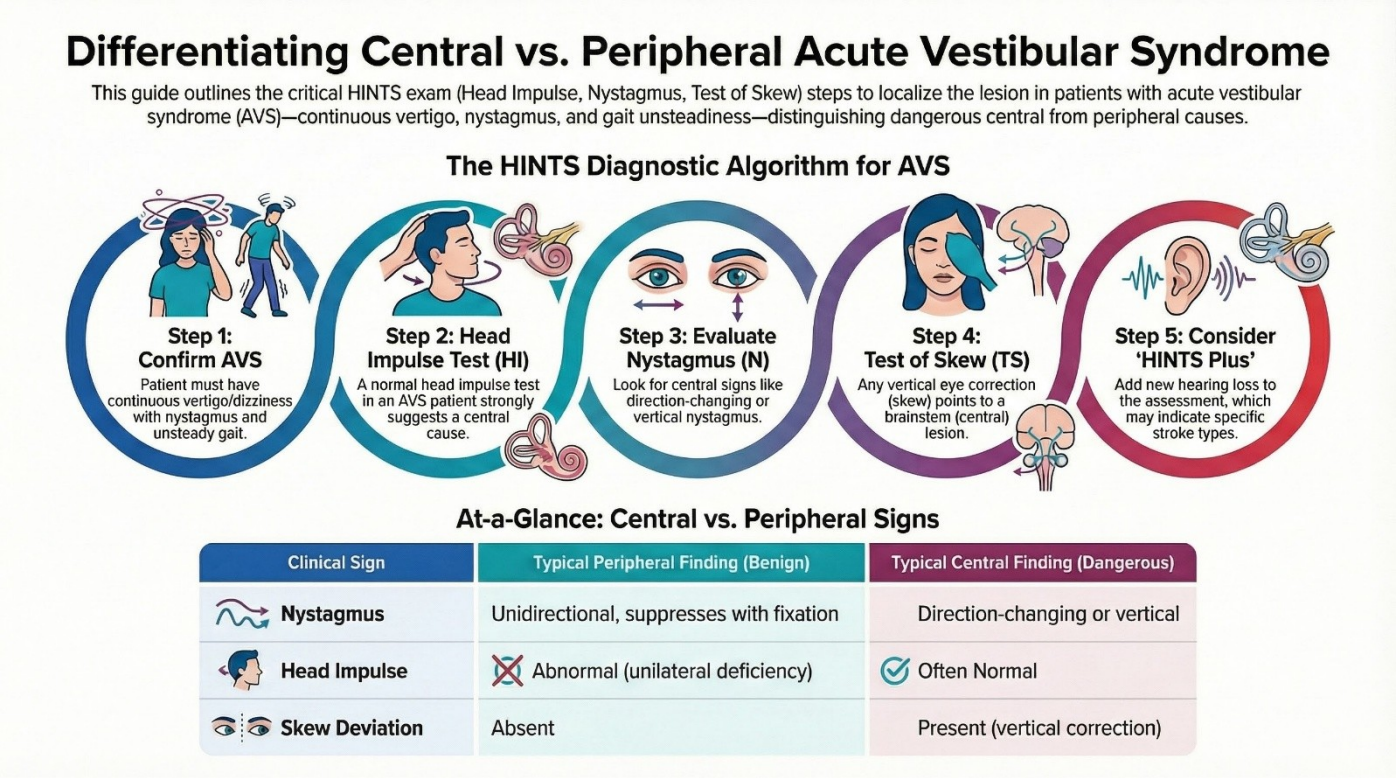
6.2 Exam-focused diagnostic algorithm for acute vestibular presentations

In the acute vestibular syndrome (continuous vertigo/dizziness with spontaneous nystagmus and gait unsteadiness), the goal is localisation and urgency rather than etiologic cataloguing.

Step 1: Establish that the patient truly has AVS. Symptoms should be continuous for hours with spontaneous nystagmus or marked gait disturbance. Episodic positional vertigo patterns require a different pathway.

Step 2: Perform horizontal head impulse testing. If the head impulse is normal bilaterally in a patient with AVS, the probability of central pathology rises substantially, and further central signs should be sought aggressively (nystagmus direction changes, skew deviation, severe truncal ataxia) [23,24].

Step 3: Evaluate spontaneous nystagmus pattern with and without fixation if possible. Unidirectional horizontal-torsional nystagmus that suppresses with fixation supports a peripheral lesion; direction-changing gaze-evoked or vertical nystagmus supports central localisation [1,2].



Step 4: Perform test-of-skew (cover–uncover or alternate cover test). A vertical corrective movement (skew) supports brainstem involvement in AVS and can identify stroke even when an abnormal head impulse suggests a peripheral lesion [23].

Step 5: Consider "HINTS plus" elements when clinically appropriate (new hearing loss, severe imbalance). New hearing loss can indicate labyrinthine or AICA territory involvement, and vHIT can be misleading in some AICA infarcts; therefore, the full syndrome and risk context must drive localisation urgency [24].

7. Instrumented and laboratory assessment (overview only)

This section is intentionally an overview; each modality merits a separate deep dive.

7.1 Video head impulse testing (vHIT)

vHIT quantifies aVOR gain during brief, high-acceleration head impulses and detects overt and covert corrective saccades. It can assess all six semicircular canals by delivering impulses in horizontal and vertical canal planes. Compared with bedside HIT, vHIT provides objective gain

metrics and makes covert saccades visible on recordings, improving diagnostic precision, especially for subtle deficits and for vertical canal evaluation [2].

Key outputs include canal-specific gain, saccade presence and timing, and asymmetry. Gain calculation methods vary across devices and analysis windows, and this can affect thresholds; therefore, clinicians should interpret gain values in the context of device-specific norms and quality metrics [29].

7.2 Caloric testing

Caloric testing stimulates the horizontal canal system using thermal irrigation (water or air), provoking nystagmus through mechanisms related to temperature-dependent fluid dynamics and/or neural effects. Clinically, the caloric test is most useful as a low-frequency probe and remains valuable for detecting mild unilateral hypofunction and for characterizing some dissociation patterns (for example, hydrops-related caloric weakness with preserved vHIT). The classic output is peak slow-phase velocity and derived indices such as unilateral weakness and directional preponderance (often computed with Jongkees-type formulas) [2].

A practical interpretive anchor is frequency: caloric responses are often conceptualized as approximating a stimulus frequency on the order of 0.003 Hz, far below the head impulse domain [20].

7.3 Rotational chair testing

Rotational chair testing provides controlled angular motion stimuli and quantifies the aVOR across mid-frequency ranges and step responses. Sinusoidal harmonic acceleration paradigms commonly use frequencies roughly between 0.01 and 0.64 Hz, quantifying gain, phase, and symmetry across that range [21]. Velocity step tests assess time constants of nystagmus decay, providing a window into velocity storage behaviour and compensation dynamics [2].

Rotational chair testing is particularly valuable in bilateral vestibulopathy because it can quantify residual function when calorics are absent and can evaluate mid-frequency dynamics that vHIT may not capture.

7.4 Ancillary quantitative tools (VOG/VNG, DVA platforms, imaging)

Video-oculography (VOG) and video nystagmography (VNG) provide objective recording of eye movements, improving detection of subtle nystagmus, fixation suppression, and saccadic intrusions. Instrumented DVA platforms quantify functional gaze stability. Imaging is not a “vestibular function test” but is essential when central localisation is suspected, particularly in acute settings or when ocular motor patterns point to brainstem/cerebellar pathology. These tools augment localisation rather than replace bedside examination.

8. Advanced insights, controversies, and evolving concepts

8.1 Velocity storage: physiology, clinical signatures, and why it matters

Velocity storage is a central integrative mechanism that prolongs and shapes vestibular

responses beyond the mechanical time constant of the semicircular canals. It contributes to sustained nystagmus during prolonged rotation and influences the spatial orientation of eye velocity relative to gravity. The nodulus and uvula exert key cerebellar control over velocity storage time constants and spatial alignment, as reviewed in work on vestibulocerebellar control of VOR orientation [16].

Clinically, velocity storage becomes visible when it is abnormal: excessive or misaligned velocity storage can contribute to periodic alternating nystagmus and complex motion intolerance phenotypes, whereas reduced time constants may appear in bilateral vestibular loss and may influence rotational chair results.

8.2 VOR plasticity as a diagnostic clue, not a nuisance

VOR adaptation is often framed as a rehabilitation concept, but diagnostically it explains why the same lesion can present differently across time. Early after unilateral loss, spontaneous nystagmus and overt saccades dominate; later, covert saccades and improved static stability may emerge despite persistent dynamic gain deficits. Cerebellar learning mechanisms are integral to this adaptation, and cerebellar disease can be inferred when expected compensation fails or when VOR gain becomes unstable across contexts [17,18].

8.3 Central lesions that mimic peripheral VOR patterns (and vice versa)

Two clinically important mimicry directions exist.

Central mimicking peripheral. Some **brainstem lesions involving vestibular nuclei** or their projections can produce abnormal head impulse tests and caloric weaknesses, creating a peripheral-appearing VOR deficit. Conversely, **cerebellar lesions** can produce unidirectional nystagmus and nausea, mimicking vestibular neuritis, particularly early.

Peripheral mimicking central. **AICA territory ischemia can** involve the labyrinth and create abnormal head impulse findings and **hearing loss** yet is a stroke syndrome; therefore, reliance on “abnormal HIT equals benign peripheral” is unsafe in acute contexts [24,27].

These mimicries underline a general rule: the VOR is a localisation tool when interpreted as part of a network, not as a standalone “diagnosis machine.”

8.4 What phylogeny predicts about human VOR constraints

Phylogeny predicts both strengths and limitations. The VOR’s ancient origin and brainstem implementation explain its speed and reliability, but also its rigidity: it is designed to stabilize images reflexively, and higher voluntary behaviours must either suppress it or work around it (for example, VOR cancellation during head-fixed target tracking). Moreover, because the VOR’s core pathways are conserved and “hardwired,” many disease phenotypes are stereotyped, enabling bedside localisation. At the same time, uniquely human demands—high-acuity vision, complex environments, frequent head-on-body movement—make even mild deficits symptomatic and amplify the clinical importance of compensatory strategies such as covert saccades [1,3].

9. Summary of clinical utility

The VOR is the vestibular physician's most direct window into peripheral vestibular function and into brainstem–cerebellar ocular motor integration. Understanding the VOR as a frequency-dependent, plane-specific reflex implemented by a short-latency premotor arc and calibrated by cerebellar modules allows precise clinical localisation. Bedside HIT and VOR cancellation testing provide rapid, high-yield differentiation between peripheral hypofunction and central ocular motor pathology, especially in acute vestibular syndrome when time-critical decisions depend on recognising stroke mimics. Laboratory tools—vHIT, calorics, and rotational chair testing—extend this framework by quantifying canal-specific function, probing distinct frequency bands, and revealing compensation and velocity storage dynamics. **Ultimately, VOR abnormalities map to symptoms through two primary mechanisms: dynamic gaze instability (oscillopsia) when gain is insufficient, and vestibular tone imbalance (vertigo/nystagmus) when asymmetry is acute or poorly compensated**. The clinician's task is to interpret these outputs in anatomical and computational context rather than treating "vestibular function" as a single scalar property [1,2].

Tables

Table 1. Definitions and classification of VOR-related phenomena

Term	Definition	Clinical relevance
Vestibulo-ocular reflex (VOR)	Reflexive eye rotation driven by vestibular inputs to stabilize retinal images during head motion	Core localisation tool; deficits cause oscillopsia and abnormal HIT/vHIT
Angular VOR (aVOR)	Canal-mediated VOR responding to angular head motion	Primary target of HIT, vHIT, caloric, rotational chair
Translational VOR (tVOR)	Otolith-mediated ocular responses to linear head motion, distance-dependent	Near-target stabilization; implicated in some complex symptom profiles
Gain	Eye velocity / head velocity (ideal near 1.0 for aVOR)	Low gain indicates hypofunction or central dyscalibration
Phase	Timing relationship between eye and head motion	Abnormal phase/lead in chair testing can indicate central dynamics or compensation
Overt corrective saccade	Catch-up saccade after head impulse	Visible on bedside HIT; indicates deficient aVOR
Covert corrective saccade	Catch-up saccade during head impulse	Detected on vHIT; compensatory strategy that may reduce oscillopsia
Velocity storage	Central mechanism prolonging and shaping vestibular-driven eye velocity beyond canal mechanics	Influences rotational chair time constant; abnormal patterns contribute to central nystagmus phenotypes

VOR cancellation/suppression	Ability to suppress vestibular-driven eye movements when tracking a head-fixed target	Impairment suggests cerebellar dysfunction (flocculus/paraflocculus)
Unilateral vestibular hypofunction	Reduced function of one labyrinth/nerve division	Abnormal head impulse toward lesion; spontaneous nystagmus; imbalance
Bilateral vestibulopathy	Reduced function of both labyrinths	Oscillopsia, gait unsteadiness, worse in dark; reduced gains bilaterally [11]

Table 2. Peripheral versus central distinguishing features (VOR-focused)

Feature	Peripheral vestibular hypofunction (typical)	Central (brainstem/cerebellum) pattern
Bedside horizontal HIT	Abnormal toward affected side	Often normal in AVS strokes; can be abnormal in some brainstem lesions or AICA strokes
vHIT	Canal-specific low gain with overt/covert saccades; often matches nerve division patterns	May be normal despite severe symptoms; or show bilateral/complex patterns depending on lesion
Nystagmus	Often unidirectional horizontal-torsional; fixation suppressible; Alexander's law	Direction-changing gaze-evoked, vertical, or purely torsional; poor fixation suppression
VOR cancellation	Usually preserved	Often impaired (especially cerebellar flocculus/paraflocculus)
Skew deviation	Usually absent or small	More likely present in brainstem involvement in AVS
Frequency coherence	Often coherent across tests, but may show mild frequency differences	Dissociations common due to central integration/cancellation deficits
Symptom emphasis	Vertigo early, then imbalance; oscillopsia if uncompensated	Gait/truncal ataxia, ocular motor signs, impaired cancellation; symptoms may exceed peripheral findings

Table 3. Lesion localisation patterns (finding → anatomy → common causes)

Key finding pattern	Likely anatomy/localisation	Common clinical contexts
Abnormal horizontal HIT/vHIT to one side with spontaneous unidirectional nystagmus	Ipsilateral horizontal canal/superior vestibular nerve division	Acute unilateral vestibulopathy/vestibular neuritis; labyrinthitis; vestibular schwannoma
Horizontal + anterior canal vHIT deficits with posterior canal sparing	Superior vestibular nerve division predominance	"Superior VN" pattern; some acute vestibular neuropathy spectra

Isolated posterior canal vHIT deficit with otolith involvement	Inferior vestibular nerve division/posterior canal	Inferior vestibular neuritis (rarer); selective end-organ pathology
Bilaterally reduced horizontal vHIT gain (<0.6) and/or absent calorics and reduced chair gain	Bilateral labyrinth/afferent loss	Aminoglycoside ototoxicity; bilateral vestibular loss; advanced bilateral vestibulopathy [11]
Normal head impulse in AVS with direction-changing or vertical nystagmus and/or skew	Central vestibular pathways (brainstem/cerebellum)	Posterior circulation stroke; cerebellitis; demyelination
Markedly impaired VOR cancellation with gaze-evoked nystagmus	Flocculus/paraflocculus and ocular motor integrator network	Degenerative cerebellar disease (e.g., SCA), cerebellar infarct
Caloric weakness with preserved vHIT gains	Low-frequency canal response abnormality with preserved high-frequency function	Ménière's disease/endolymphatic hydrops; test dissociation patterns [25]
Short rotational chair time constant with low gains	Reduced velocity storage and/or bilateral vestibular loss	Bilateral vestibulopathy; severe peripheral loss

Table 4. Common pitfalls, confounders, and interpretation errors

Domain	Pitfall/confounder	How it misleads	Mitigation
Bedside HIT	Predictable impulses, low acceleration, patient anticipates	False-normal HIT	Make impulses unpredictable; ensure adequate acceleration; stabilize trunk
Bedside HIT	Ocular motor palsy/INO	Apparent corrective saccades not due to vestibular loss	Assess ocular alignment and saccades; interpret in full ocular motor context
vHIT	Goggle slippage, calibration errors, eyelid artifacts	Artificially low or high gains; spurious saccades	Verify fit, repeat calibrations, inspect traces, correlate with clinical findings
vHIT	Strong spontaneous nystagmus	Distorts baseline and gain computation	Use appropriate analysis settings; re-test as nystagmus reduces; integrate with other modalities
Calorics	Ear canal obstruction, perforation, poor irrigation technique	False weakness/asymmetry	Otoscopy first; standardized irrigation; repeat if artifact suspected
Rotational chair	Patient alertness fluctuations	Alters nystagmus measures and time constants	Keep patient alert; monitor attention; interpret with

			context
Cancellation testing	Poor vision or inability to track	Appears "impaired cancellation"	Ensure target visibility; assess pursuit separately
Global interpretation	Over-reliance on single test	Misses central mimics or frequency dissociation syndromes	Integrate frequency bands and ocular motor signs; apply syndrome-based reasoning

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Accuracy and Currency

While every effort has been made to ensure the accuracy and completeness of the content, vestibular medicine is a rapidly evolving field. Clinicians are encouraged to verify specific protocols, normative values, and therapeutic recommendations against current published guidelines and primary literature.

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