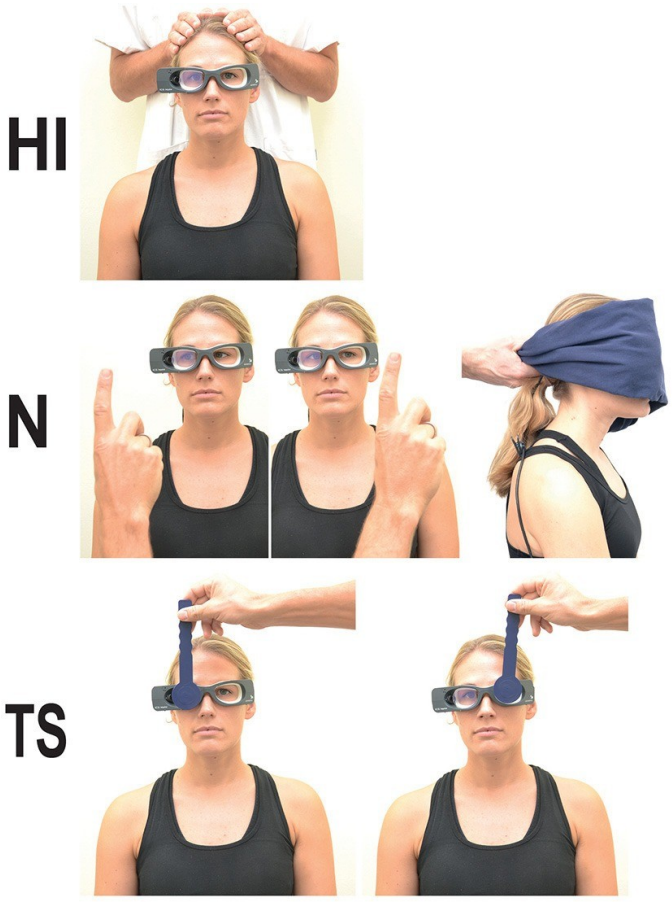
THE VESTIBULAR SYSTEM

VOLUME II

Bedside Localization and Vestibular Testing

From the dizzy complaint to a defensible anatomic hypothesis



Cover image. The HINTS examination components: head impulse, nystagmus assessment, and test of skew. von Martial et al., via Wikimedia Commons, CC BY 4.0.

Resident study guide • July 2026

READ THE SYNDROME BEFORE THE TEST

ORIENTATION. How to use this volume

Volume I explained how head motion becomes a neural signal. Volume II begins one step later: a patient arrives with dizziness, imbalance, oscillopsia, or positional vertigo, and the resident must decide what the symptom pattern means, which bedside observations are trustworthy, and which test can resolve the uncertainty that remains.

Vestibular diagnosis is often made unnecessarily difficult by starting with disease names or by treating the word “vertigo” as if it were a localization. The more reliable sequence is to classify the syndrome by timing and triggers, examine the eyes and stance while the patient is in that syndrome, and then use laboratory or imaging tests only to answer a defined question. This volume follows that sequence. [1-3]

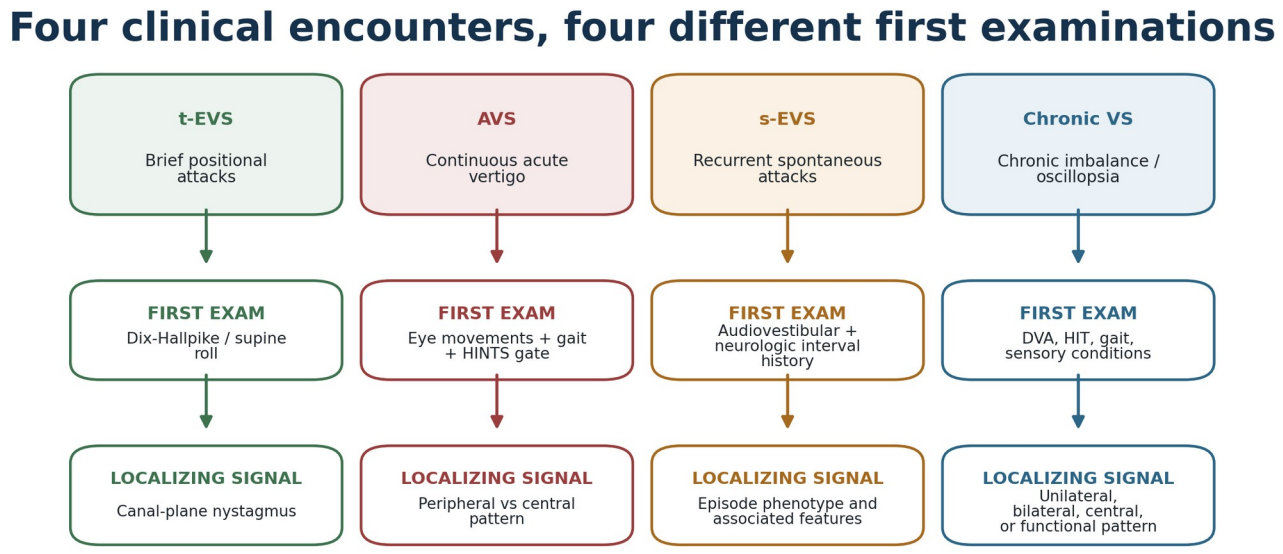


Figure 1. The same symptom vocabulary leads to different first examinations depending on the vestibular syndrome. Original synthesis.

What this volume includes

- A timing-and-triggers approach to acute, episodic, positional, and chronic dizziness.
- A systematic ocular motor examination that treats eye movements as vectors and waveforms rather than labels.
- Practical performance and interpretation of the bedside head impulse test, test of skew, HINTS, and HINTS+.
- Canal-specific positional testing, including Dix-Hallpike and supine roll testing and recognition of central positional mimics.
- Bedside assessment of hearing, stance, gait, dynamic visual acuity, and perceived vertical.

- Interpretation of VNG/ENG, calorics, vHIT, rotary chair, cVEMP, oVEMP, and posturography.
- Imaging choices framed by the residual diagnostic question rather than by dizziness alone.
- Four integrated clinical encounters that demonstrate how the pieces fit together.

What is intentionally deferred

Volume III will focus on named disorders, diagnostic criteria, and management. This volume necessarily uses disorders as examples, but its central task is localization: deciding whether the dominant signal is peripheral or central, unilateral or bilateral, canal or otolith, acute or compensated, and whether the testing result actually explains the patient's syndrome.

> **The central question**
>
> Every examination maneuver and every laboratory test should answer a sentence that begins, "I need to know whether…" If the question cannot be stated, the test is unlikely to clarify the case.

Contents

Table 1. Narrative structure of Volume II.

Chapter	Central question
1. From dizziness to syndrome	What happened in time, and what truly triggered it?
2. The ocular motor localization battery	What do alignment, gaze holding, saccades, and pursuit reveal before vestibular maneuvers begin?
3. Nystagmus as a vector	What is the slow phase doing, and does the pattern obey peripheral physiology?
4. Head impulse, skew, and HINTS+	Is HINTS applicable, and do the components form a coherent peripheral or central pattern?
5. Positional testing	Which canal geometry is being engaged, and is the response compatible with displaced otoconia?
6. Hearing, stance, gait, and functional vision	What non-nystagmus clues alter localization or urgency?
7. The vestibular laboratory	Which end organ, pathway, and frequency range does each test actually interrogate?
8. Imaging and test selection	What question remains after bedside localization, and which modality can answer it?
9. Four integrated encounters	How does the framework change the first five minutes of care?

BEGIN WITH TIME

1. From dizziness to a vestibular syndrome

The patient's chosen noun is usually less informative than the temporal architecture of the illness. "Vertigo," "lightheadedness," "wooziness," and "imbalance" overlap heavily across vestibular, neurologic, cardiovascular, metabolic, and functional disorders. The

useful questions are: When did it begin? Is the patient symptomatic continuously now? How long does an untreated episode last? What reliably initiates an episode, rather than merely worsening an existing one? [1-3]

Why symptom quality underperforms

Patients often change descriptors when the same experience is presented in a different way. A patient may call the initial sensation “spinning,” the residual state “floating,” and the gait consequence “lightheadedness,” although all three belong to one acute vestibular event. Conversely, two patients who both say “vertigo” may have posterior canal BPPV and cerebellar infarction. Symptom quality still matters - oscillopsia, diplopia, hearing change, presyncope, and autophony are useful clues - but it should refine a syndrome rather than define one.

Trigger versus exacerbation

A trigger causes an attack that was not already present. An exacerbating factor intensifies symptoms that are already ongoing. Rolling to the right in bed can trigger a 20-second attack of posterior canal BPPV. Turning the head while walking can worsen nausea in acute unilateral vestibulopathy, cerebellar stroke, vestibular migraine, or bilateral vestibular loss. The second observation is nonspecific because head motion challenges every unstable gaze and balance system.

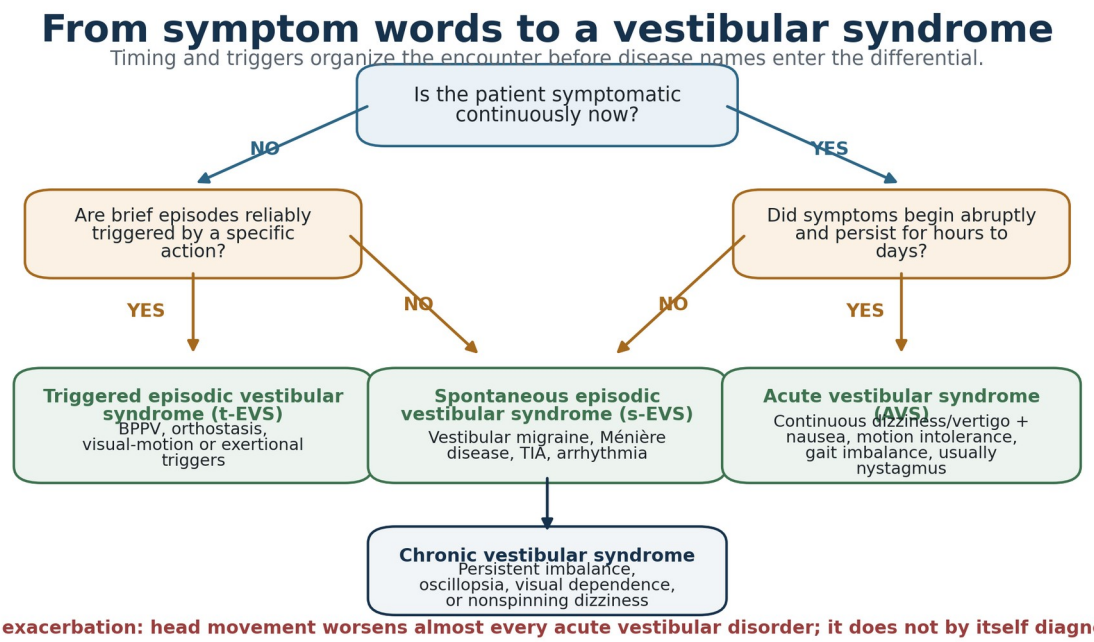


Figure 2. Timing and triggers divide the dizzy presentation into syndromes that demand different examinations. Original synthesis.

The four working syndromes

Table 2. Timing-based vestibular syndromes and their first diagnostic tasks.

Syndrome	Defining temporal pattern	First bedside priority	Representative differential
Triggered episodic vestibular syndrome (t-EVS)	Brief attacks that occur only after a reproducible trigger; the patient is usually near baseline between attacks.	Reproduce the event with the correct positional or physiologic challenge while observing objective signs.	BPPV, orthostatic hypotension, visually induced dizziness, exertional cardiopulmonary causes.
Spontaneous episodic vestibular syndrome (s-EVS)	Discrete attacks without an obligatory immediate trigger; duration may be minutes to hours.	Characterize the complete episode phenotype and search for auditory, migrainous, focal neurologic, or systemic accompaniments.	Vestibular migraine, Ménière disease, TIA, panic/autonomic episodes, arrhythmia.
Acute vestibular syndrome (AVS)	Acute onset of continuous dizziness or vertigo lasting days, with motion intolerance, nausea/vomiting, gait disturbance, and often spontaneous nystagmus.	Ocular motor localization, gait/truncal assessment, hearing screen, and stroke risk appraisal.	Acute unilateral vestibulopathy, labyrinthitis, posterior circulation stroke, demyelination, toxic/metabolic causes.
Chronic vestibular syndrome	Persistent imbalance, oscillopsia, visual dependence, or nonspinning dizziness over weeks to months.	Identify dynamic bilateral or unilateral loss, central signs, sensory dependence, and functional overlay.	Bilateral vestibulopathy, incomplete compensation, cerebellar disease, neuropathy, PPPD, medication effects.

The syndrome must be present when the examination is interpreted

A normal head impulse test performed after a 15-minute episode has resolved does not have the same meaning as a normal head impulse test during continuous AVS with spontaneous nystagmus. Likewise, HINTS cannot retrospectively classify an attack that ended yesterday. Examination findings are state-dependent. Before interpreting a negative or normal test, ask whether the physiologic state that the test was designed to interrogate is still present.

A resident-level opening history

1. Anchor the onset: exact day and approximate time, abrupt versus progressive, and what the patient was doing.
2. Determine whether symptoms are continuous now or occur in discrete attacks.
3. Measure episode duration without treatment: seconds, minutes, hours, or days.
4. Separate triggers from exacerbations. Ask what causes an attack from baseline.
5. Identify associated hearing change, tinnitus, aural pressure, headache, photophobia, diplopia, dysarthria, limb symptoms, syncope, chest symptoms, and neck pain.
6. Define baseline between attacks and the trajectory since onset.
7. Review vascular risk, recent infection, migraine history, otologic surgery/trauma, ototoxic exposure, and medications that alter eye movements or balance.

> **Boards and call-room pearl**
>
> "Worse with head movement" is not a BPPV diagnosis. BPPV is a triggered episodic syndrome in which a specific change in head position relative to gravity initiates a brief attack and a canal-specific positional nystagmus pattern.

Clinical bridge: the patient who says "I am lightheaded"

Suppose a 58-year-old patient says "lightheaded" but reports 20-second attacks each time she rolls toward the left in bed and feels normal while sitting still. The symptom noun sounds nonvestibular; the timing is strongly vestibular. Conversely, a patient who says "the room spins" continuously for twelve hours with inability to walk has AVS and requires central-versus-peripheral localization, not a Dix-Hallpike maneuver as the first test. The framework protects the clinician from both vocabulary traps.

LOOK BEFORE PROVOKING

2. The ocular motor localization battery

Before moving the head or positioning the patient, observe the eyes in primary gaze. Alignment, spontaneous drift, gaze holding, saccades, and pursuit often reveal whether the problem is confined to the labyrinth and eighth nerve or involves the brainstem, cerebellum, or supranuclear ocular motor network. [4,5]

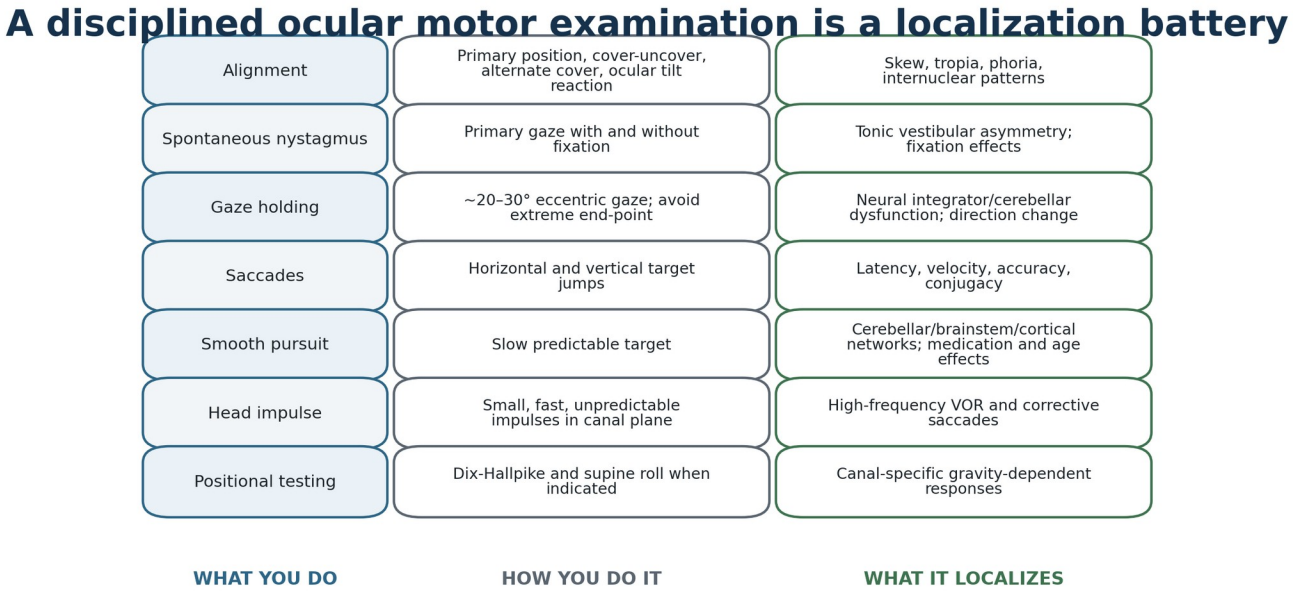


Figure 3. A disciplined ocular motor examination progresses from alignment and spontaneous eye movements to dynamic vestibular and positional challenges. Original synthesis.

Set up the examination

Seat or position the patient so the eyes are well illuminated and the examiner can see both pupils. Correct refractive error when possible. Ask about baseline strabismus, amblyopia, ocular surgery, visual loss, and congenital nystagmus. Sedatives, antiseizure medications, alcohol, severe fatigue, and visual inattention can degrade pursuit, saccade accuracy, and fixation. These confounders should be documented rather than ignored.

Alignment and cover testing

Begin with ocular alignment in primary position. Look for gross vertical separation, cyclotorsion, ptosis, internuclear ophthalmoplegia, gaze palsy, or a cranial neuropathy. The cover-uncover test detects a manifest deviation; the alternate cover test dissociates both eyes and can reveal a latent or small vertical misalignment. In AVS, a clear vertical corrective movement when the cover alternates is a skew deviation until proven otherwise. Skew reflects imbalance in graviceptive pathways extending from the vestibular nerve and nuclei through the medial longitudinal fasciculus, brainstem, cerebellum, and thalamus. Large or changing skew strongly favors a central lesion, although small skews can occur with peripheral vestibular injury.

Primary gaze and fixation conditions

Observe for spontaneous nystagmus with the patient looking straight ahead. Then reduce visual fixation if equipment and safety permit. Frenzel lenses, video goggles, or an ophthalmoscope can uncover peripheral nystagmus that is suppressed by fixation. Fixation suppression is not an absolute peripheral-central separator, but a marked increase when fixation is removed supports a peripheral vestibular generator. Failure of fixation suppression can accompany cerebellar dysfunction, medications, poor visual acuity, or inadequate fixation.

Gaze holding

Ask the patient to look approximately 20 to 30 degrees right, left, up, and down while keeping the head still. Avoid forcing the eyes to extreme eccentric positions, where physiologic end-point nystagmus becomes common. Direction-changing horizontal gaze-evoked nystagmus - right-beating in right gaze and left-beating in left gaze - suggests impaired gaze holding in the neural integrator, often from cerebellar or brainstem dysfunction, medication effect, or intoxication. A unidirectional peripheral vestibular nystagmus may increase when the patient looks in the direction of its fast phase, but it should not reverse simply because gaze crosses midline.

Saccades

Use targets separated horizontally and vertically. Ask the patient to look back and forth without moving the head. Assess initiation, peak velocity, accuracy, range, and conjugacy. Markedly slow saccades suggest dysfunction of premotor burst neurons or neurodegenerative systems; dysmetric saccades implicate cerebellar calibration; an adducting delay with abducting nystagmus suggests internuclear ophthalmoplegia. Mild hypometria is common and nonspecific. Saccades should be interpreted as part of a pattern, not as a solitary “central” checkbox.

Smooth pursuit and optokinetic responses

Move a target slowly and predictably through the horizontal and vertical planes. Normal pursuit is smooth; saccadic pursuit reflects repeated catch-up movements. Pursuit depends on vision, attention, age, medication state, cerebral cortex, pontine pathways, and cerebellum, so it is sensitive but not specific. Asymmetric pursuit or a cluster of central ocular motor abnormalities is more meaningful than mildly broken pursuit in an elderly, nauseated, medicated patient. Optokinetic stimulation can provide a related assessment of slow-phase tracking and fast-phase resetting, but bedside asymmetries must be interpreted cautiously.

Table 3. High-yield ocular motor findings before dynamic vestibular testing.

Finding	Most useful implication	Important caveat
Direction-changing gaze-evoked nystagmus	Central gaze-holding dysfunction or drug effect.	Physiologic end-point nystagmus occurs at extreme gaze; use modest eccentricity.
Pure vertical spontaneous nystagmus	Central localization is strongly favored.	Confirm it is spontaneous rather than positional and exclude congenital patterns.
Internuclear ophthalmoplegia	MLF lesion, commonly demyelinating or ischemic.	Subtle adduction slowing can be difficult to distinguish from poor effort.
Marked saccadic dysmetria	Cerebellar calibration abnormality.	Small hypometria is common and nonspecific.
Impaired fixation suppression	Cerebellar/central dysfunction is possible.	Visual acuity, attention, medication, and technique affect the result.
Skew deviation	Central otolith-ocular pathway imbalance, especially if large.	Small skew can occur in acute peripheral vestibulopathy.

A practical sequence

Primary gaze → fixation removed if available → right/left/up/down gaze → alignment and alternate cover → saccades → pursuit → head impulse when indicated → positional testing when indicated. This order captures baseline signs before provocative maneuvers alter them.

THE EYE MOVEMENT IS THE DATA

3. Nystagmus as a vector and waveform

Nystagmus is not merely present or absent. It has a waveform, axis, direction, intensity, gaze dependence, fixation dependence, temporal profile, and positional context. The disciplined examiner describes those properties before assigning a lesion. [4,5]

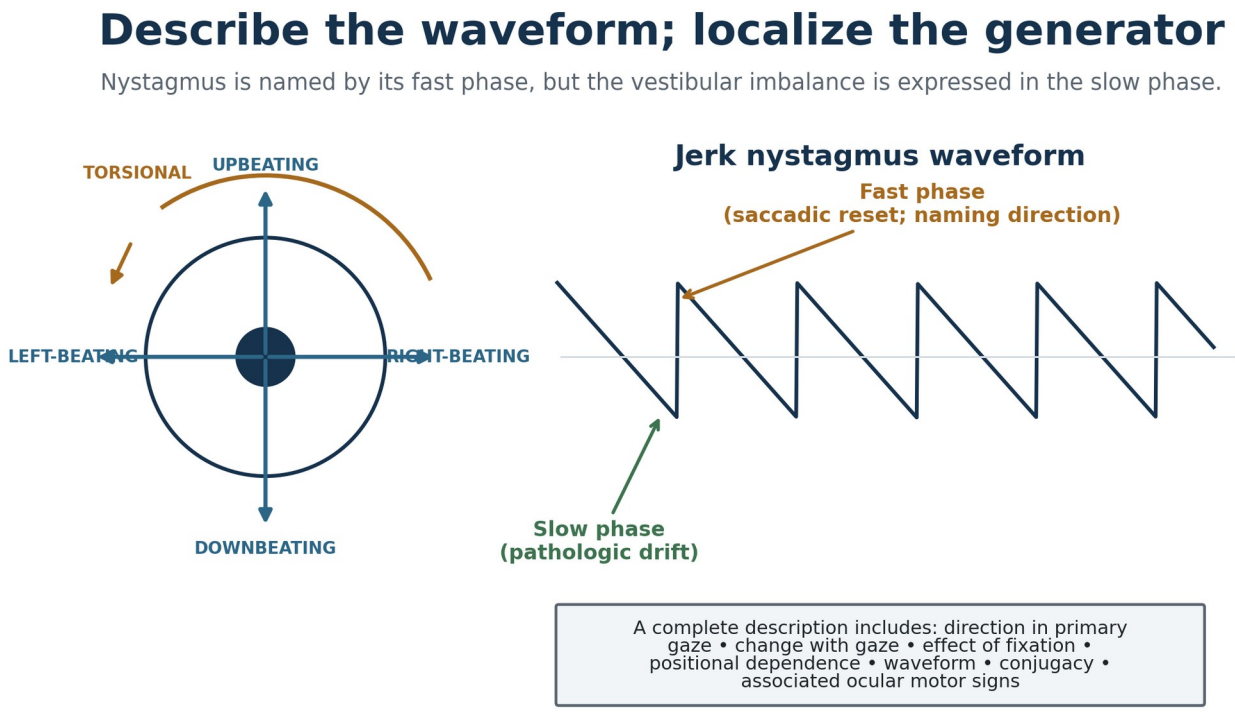


Figure 4. Jerk nystagmus is named by the fast phase, but the pathologic vestibular signal is represented by the slow drift. Original synthesis.

Name by the fast phase; reason from the slow phase

In jerk nystagmus, the eyes drift slowly because of a tonic vestibular or gaze-holding imbalance and then reset with a fast saccade. Clinical convention names the nystagmus by the fast phase. A left-beating nystagmus therefore has a slow drift to the right and a corrective fast phase to the left. In acute right unilateral peripheral loss, tonic activity from the intact left labyrinth dominates, creating a slow drift toward the weaker right side and a fast phase toward the intact left side. The nystagmus is left-beating, while the lesion is usually right-sided.

Describe the axis

Horizontal, vertical, and torsional components can occur together. Peripheral canal signals generate eye movements in the plane of the stimulated canal, so acute unilateral vestibular loss commonly produces a mixed horizontal-torsional pattern. Purely vertical or purely torsional spontaneous nystagmus is difficult to reconcile with a single peripheral canal imbalance and strongly favors central disease. Positional nystagmus is different: its vector should be interpreted in the canal plane engaged by gravity and the maneuver.

Alexander's law and gaze dependence

A typical peripheral vestibular nystagmus often becomes more intense when the patient looks toward the fast phase and weaker when looking toward the slow phase. This is Alexander's law and reflects interaction between vestibular tone and gaze-holding mechanisms. The key is that the nystagmus remains unidirectional. If the fast phase changes direction with horizontal gaze, the pattern is gaze-evoked and central-worrisome rather than a simple peripheral application of Alexander's law.

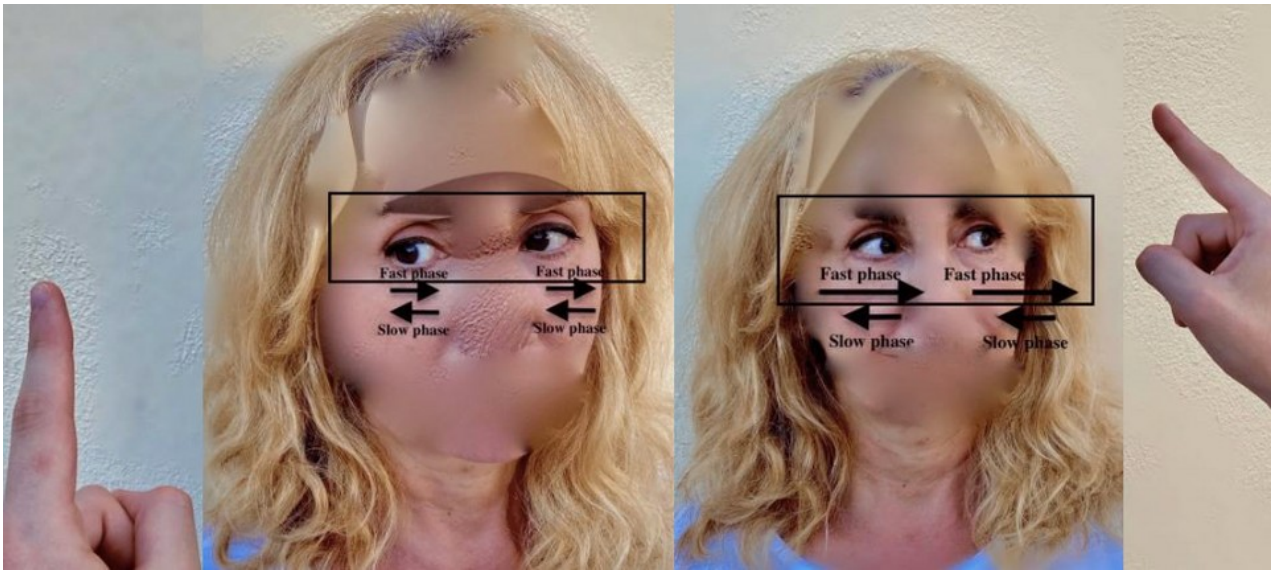


Figure 5. Illustration of unidirectional versus direction-changing gaze-evoked nystagmus during a HINTS examination. Koukoulithras et al., via Wikimedia Commons, CC BY 4.0.

Fixation and the latent peripheral signal

Visual fixation suppresses many peripheral vestibular nystagmus patterns. A patient may have little visible nystagmus in a brightly lit room yet develop a clear horizontal-torsional nystagmus under Frenzel lenses or video goggles. Conversely, prominent nystagmus despite fixation does not by itself prove a central lesion; severe acute peripheral asymmetry can overcome fixation. The useful observation is the direction and magnitude of change under controlled conditions.

Central patterns worth recognizing

Downbeat nystagmus suggests dysfunction near the vestibulocerebellum or cervicomedullary junction and has a broad differential including structural, degenerative, toxic, and metabolic causes. Upbeat nystagmus often localizes to brainstem or cerebellar pathways. Seesaw, periodic alternating,

convergence-retraction, and pendular nystagmus point toward specific central networks. Bruns nystagmus combines coarse, low-frequency nystagmus when looking toward the side of a cerebellopontine angle lesion with finer, faster nystagmus in the opposite gaze, reflecting both gaze-holding failure and vestibular imbalance.

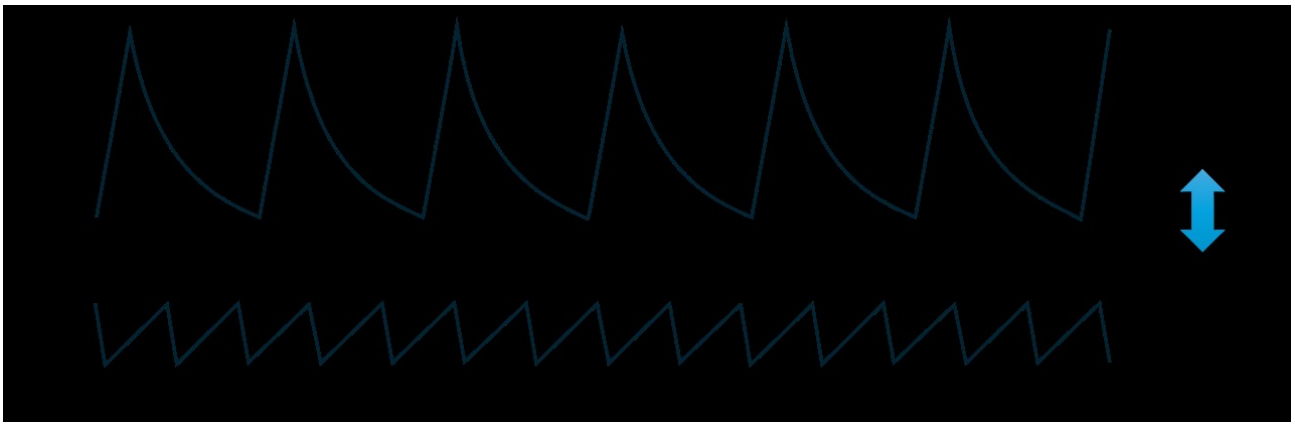


Figure 6. Oculographic recording of Bruns nystagmus, illustrating gaze-dependent changes in amplitude and frequency. Deepika Karnan, via Wikimedia Commons, CC BY 4.0.

Head-shaking nystagmus

With the head pitched forward approximately 30 degrees, the examiner oscillates the head horizontally for about 15 to 20 seconds and then observes the eyes after stopping. A transient horizontal nystagmus can reveal a dynamic asymmetry that was not visible at rest, often beating toward the stronger side. Vertical or markedly cross-coupled responses raise central concern. The test is supportive rather than definitive and depends on adequate head velocity, suppression of fixation, and the state of compensation.

Table 4. Pattern recognition in nystagmus localization.

Feature	Peripheral-compatible pattern	Central-worrisome pattern
Primary-gaze axis	Horizontal-torsional mixed jerk nystagmus.	Pure vertical, pure torsional, or complex disconjugate pattern.
Effect of horizontal gaze	Unidirectional; intensity follows Alexander's law.	Direction changes with gaze or there is rebound nystagmus.
Fixation	Often suppressed or reduced.	May persist or fixation suppression may fail.
Positional logic	Vector matches a canal plane and the expected gravity-dependent response.	No canal-plane logic, persistent atypical vertical response, or multiple inconsistent vectors.
Associated eye signs	Usually no major saccadic, pursuit, or alignment abnormality.	Skew, ophthalmoplegia, saccadic dysmetria, gaze palsy, or marked pursuit abnormality.

Do not localize from one adjective

"Horizontal" does not equal peripheral, and "torsional" does not equal central. The complete pattern - spontaneous versus positional, unidirectional versus gaze-changing, fixation behavior, canal-plane logic, and associated ocular motor signs - carries the localization.

USE THE GATE BEFORE THE MNEMONIC

4. Head impulse, skew, and HINTS+

HINTS is powerful because it integrates three physiologic observations in the acute vestibular syndrome. It is dangerous when used as a generic dizziness checklist, when performed after the syndrome has resolved, or when the examiner has not mastered the individual components. [3,6-9]

The bedside head impulse test

Ask the patient to fixate a small target, usually the examiner's nose. Hold the head securely, pitch it slightly forward for horizontal canal testing, and deliver small-amplitude, high-acceleration, unpredictable impulses to each side. The patient should not be able to preprogram the direction. During a normal impulse, the VOR keeps the eyes on target. If the VOR is deficient for the direction of the impulse, the eyes move with the head and then make a corrective saccade back to the target.

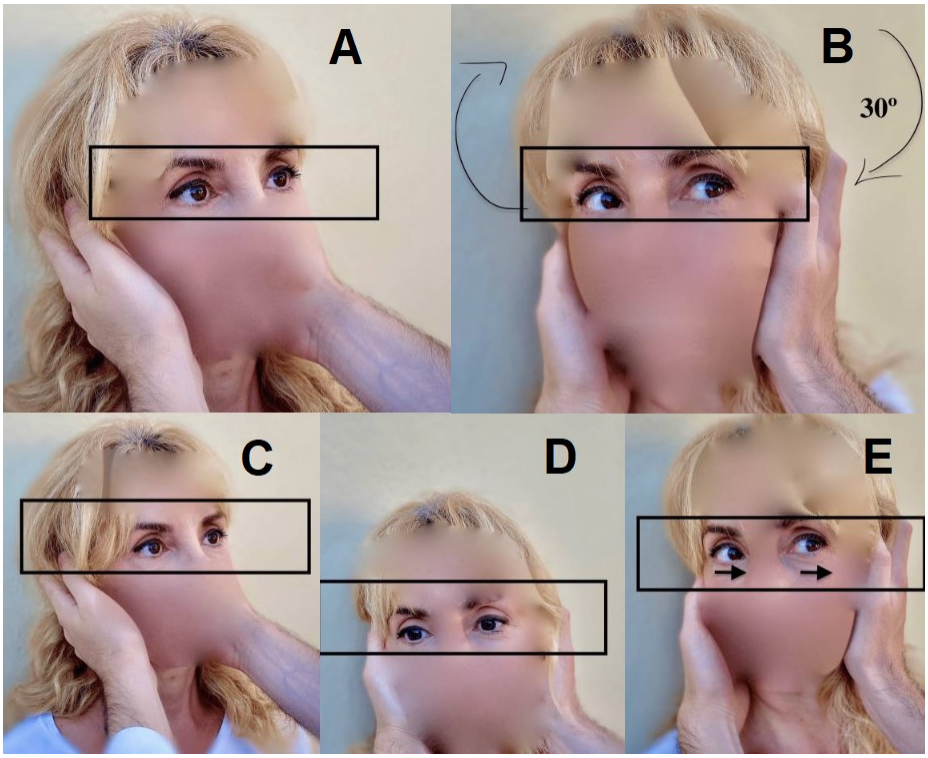


Figure 7. Bedside head impulse testing and the corrective saccade that follows an impaired VOR. Koukoulithras et al., via Wikimedia Commons, CC BY 4.0.

What an abnormal impulse means

A corrective saccade after an impulse to the right supports deficient rightward high-frequency VOR function, usually implicating the right horizontal canal pathway. In AVS, an abnormal impulse toward the side opposite the nystagmus fast phase creates a coherent acute peripheral pattern. However, central lesions involving the vestibular nucleus, root entry zone, or floccular pathways can

occasionally produce abnormal impulses. Likewise, mild peripheral loss can be missed at the bedside, especially if corrective saccades occur covertly during the impulse.

Technique errors that create false results

- Impulses that are too slow or too large test prediction and pursuit rather than the high-frequency VOR.
- Predictable alternating impulses allow preprogrammed saccades.
- Moving the target or allowing the patient to blink obscures the corrective saccade.
- Testing through painful or restricted neck motion degrades both safety and interpretation.
- A patient with poor vision, ocular motor palsy, or inability to attend may not provide an interpretable result.

The test of skew

Perform an alternate cover test while the patient fixates a target. Move the occluder briskly from one eye to the other and watch the newly uncovered eye for a vertical refixation. Repeated vertical correction indicates a vertical ocular misalignment. A prominent skew in AVS suggests central involvement of otolith-ocular pathways and is often accompanied by ocular torsion and head tilt as part of the ocular tilt reaction. Small skews can occur with peripheral vestibular lesions, so magnitude and the rest of the examination matter.

The HINTS applicability gate

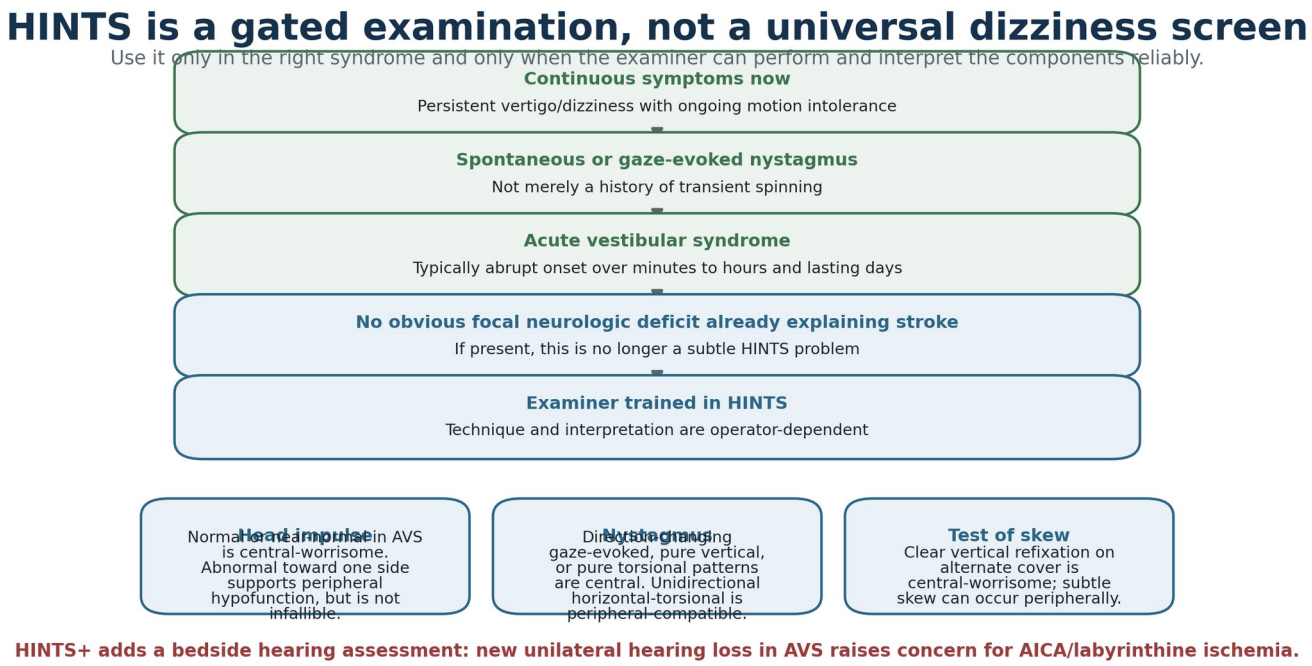


Figure 8. HINTS should be entered only after confirming an acute vestibular syndrome with ongoing nystagmus and a trained examiner. Original synthesis.

The classic HINTS population has continuous symptoms, spontaneous or gaze-evoked nystagmus, and acute gait or motion intolerance. HINTS is not validated as a rule-out test for patients who are asymptomatic, for isolated episodic dizziness, for straightforward BPPV, or for nonspecific chronic imbalance. If a patient already has a focal neurologic deficit, a severe new headache with concerning

features, or another obvious stroke syndrome, the clinician should pursue urgent central evaluation rather than seek reassurance from HINTS.

Peripheral versus central HINTS patterns

Table 5. Interpreting the components of HINTS+ in the correct clinical population.

Component	Peripheral-compatible AVS	Central-worrisome AVS
Head impulse	Clearly abnormal toward one side, usually the side of peripheral hypofunction.	Normal or near-normal bilaterally despite ongoing AVS, or a pattern inconsistent with the rest of the examination.
Nystagmus	Unidirectional horizontal-torsional nystagmus, often stronger in gaze toward the fast phase.	Direction-changing gaze-evoked nystagmus, pure vertical/torsional nystagmus, or other central ocular motor pattern.
Skew	Absent or very small.	Clear vertical refixation on alternate cover.
Hearing in HINTS+	No new unilateral hearing loss.	New unilateral hearing loss raises concern for labyrinthine/AICA ischemia, although peripheral cochleolabyrinthine disease remains possible.

HINTS+ and hearing

The “plus” adds a bedside screen for new unilateral hearing loss. The labyrinthine artery usually arises from AICA, and ischemia can injure both cochlear and vestibular structures before other obvious brainstem signs appear. Bedside finger rub or whispered voice is a screening maneuver, not a replacement for audiometry. Cerumen, chronic asymmetry, conductive disease, patient attention, and ambient noise can mislead. The relevant finding is a new asymmetry temporally linked to the acute syndrome.

Why the pattern outperforms any one component

A normal head impulse in AVS is worrisome but not sufficient to diagnose stroke; an abnormal impulse is supportive of peripheral loss but not perfectly exclusive. The strength of HINTS is the coherent combination. A patient with a right corrective saccade, left-beating unidirectional nystagmus, no skew, and no new hearing loss has a physiologically coherent right peripheral pattern. A patient with normal impulses, direction-changing gaze-evoked nystagmus, or skew has a central-worrisome pattern that requires stroke-pathway evaluation. In trained hands, this bedside strategy can be highly sensitive, but evidence and guidelines emphasize examiner training and appropriate patient selection. [3,7-9]

> **Safety rule**
>
> Do not document “HINTS negative.” Document each component, whether the patient met the AVS eligibility gate, the direction of nystagmus, the side and quality of any corrective saccade, the presence or absence of skew, and the hearing comparison.

LET GRAVITY SELECT THE CANAL

5. Positional testing and canal localization

Positional vertigo is diagnosed by reproducing a characteristic eye movement, not by reproducing dizziness alone. The maneuver changes the orientation of a canal relative to gravity; the resulting nystagmus should make geometric sense. [10,11]

Dix-Hallpike testing

For the standard right Dix-Hallpike maneuver, begin with the patient sitting and turn the head 45 degrees to the right. Bring the patient briskly to a supine head-hanging position with approximately 20 degrees of neck extension while maintaining the head turn. Observe the eyes during the transition, throughout the dependent position, and while returning to sit. The head turn brings the right posterior canal closer to the sagittal plane, allowing gravity to move free otoconia through the canal.

Modify the maneuver when cervical extension is unsafe or impractical. A side-lying test can engage the posterior canal without head hanging: from sitting, turn the head away from the test side and rapidly bring the patient onto the test-side shoulder. The diagnostic principle is unchanged; only the body geometry differs.

Posterior canal BPPV

Posterior canal canalithiasis typically produces a brief latency, followed by a crescendo-decrescendo upbeat torsional nystagmus. The upper poles of the eyes beat toward the dependent, affected ear. The response usually lasts less than one minute and may reverse when the patient sits up. Repetition may reduce the response, but lack of fatigability does not by itself exclude BPPV. The nystagmus vector, time course, and maneuver context are more important than a single classic adjective.

Supine roll testing

If the history is positionally triggered but Dix-Hallpike testing is negative or produces horizontal nystagmus, perform a supine roll test. Position the patient supine with the head elevated about 30 degrees to align the horizontal canals closer to the earth-horizontal plane. Rotate the head approximately 90 degrees to each side and compare the direction and intensity of nystagmus.

Geotropic horizontal nystagmus

When nystagmus beats toward the ground in both lateral positions, horizontal canal canalithiasis is likely. The stronger response usually occurs with the affected ear down because excitatory ampullopetal flow in the horizontal canal produces the larger response. The side comparison is useful only if head angles and observation conditions are comparable.

Apogeotropic horizontal nystagmus

When nystagmus beats away from the ground in both positions, cupulolithiasis or short-arm canalithiasis is considered. The affected side is often the side with the weaker apogeotropic response, but lateralization can be difficult. Bow-and-lean testing, null points, conversion to a geotropic pattern,

and treatment response can provide additional evidence. Avoid overclaiming the side from a subtle intensity difference.

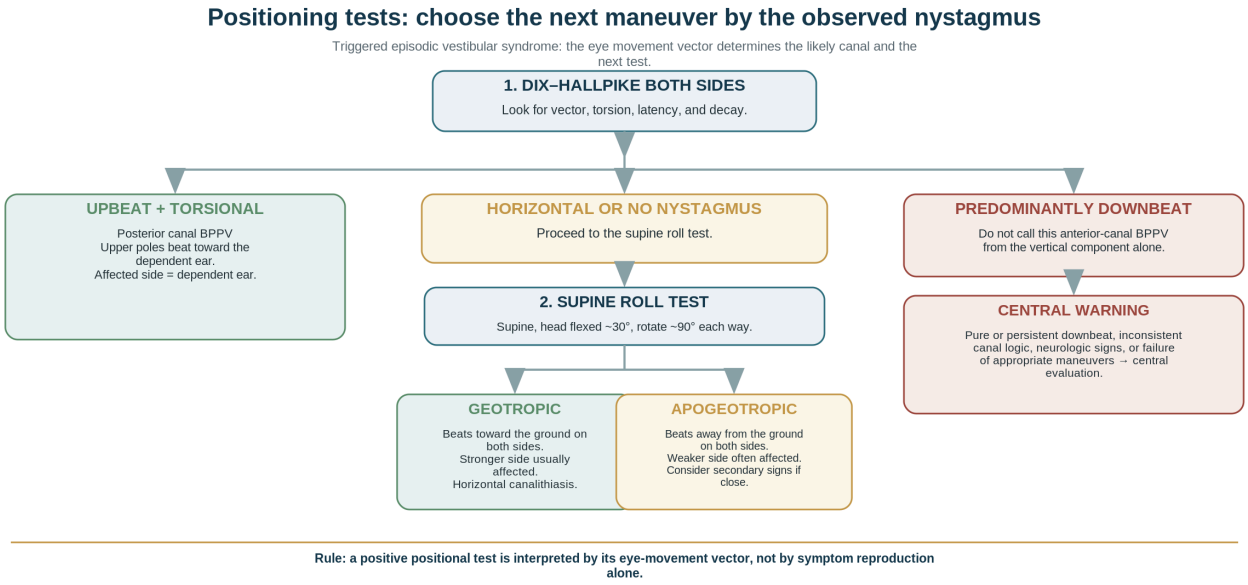


Figure 9. A practical map from the suspected canal to the appropriate positioning test and expected pattern. Original synthesis.

Anterior canal and central positional downbeat nystagmus

Anterior canal BPPV is uncommon and may produce downbeat nystagmus with a torsional component during head-hanging maneuvers. The torsional component may be small, making lateralization unreliable. Because central positional downbeat nystagmus is more consequential and can appear similar, persistent downbeat responses, absent canal-plane logic, neurologic signs, atypical time course, or failure of appropriate maneuvers should lower the threshold for central evaluation.

Canalithiasis versus cupulolithiasis

Table 6. Mechanistic differences between free-moving and cupula-associated positional debris.

Feature	Canalithiasis	Cupulolithiasis / attached debris
Mechanism	Free particles move through the canal after a position change.	Particles adhere to the cupula or alter its gravity sensitivity.
Latency	Often a short latency before onset.	Often little or no latency.
Duration	Usually transient as particles settle and cupular deflection decays.	May persist while the provocative position is maintained.
Response to repetition	Often attenuates, although not reliably.	May remain sustained.
Interpretive caution	Atypical timing can occur with high particle load or technique variation.	Persistent positional nystagmus can also be central; geometry still matters.

Central positional red flags

- Pure downbeat, pure upbeat, or pure torsional nystagmus without a convincing canal-plane explanation.
- Persistent nystagmus without the expected decay or with a pattern that changes inconsistently across repetitions.
- Little or no vertigo despite striking eye movements, although symptom intensity alone is not decisive.
- Associated gaze-evoked nystagmus, skew, ophthalmoplegia, saccadic dysmetria, severe truncal ataxia, or other neurologic signs.
- Repeated failure of correctly selected and correctly performed repositioning maneuvers.
- A new headache, focal neurologic symptoms, or a syndrome that is not truly triggered episodic.

> **Boards pearl**
>
> A negative Dix-Hallpike does not rule out BPPV. It may indicate horizontal canal disease, an inactive interval, an inadequately positioned canal, or a non-BPPV diagnosis. The next step is determined by the history and observed vector, not by the word “negative.”

THE EYES ARE CENTRAL, BUT NOT SUFFICIENT

6. Hearing, stance, gait, and functional vision

Vestibular localization becomes more reliable when eye findings are integrated with hearing, postural control, and gaze stability during head movement. These observations often determine urgency even before laboratory testing is available.

Bedside hearing

Ask specifically about new unilateral hearing change, tinnitus, fullness, autophony, sound- or pressure-induced symptoms, and chronic asymmetry. Inspect the canals and tympanic membranes. A finger rub, whispered voice, or tuning fork can screen for asymmetry, but formal audiometry is more reproducible and distinguishes conductive from sensorineural loss. In AVS, new unilateral sensorineural hearing loss broadens the differential from isolated vestibular neuritis toward labyrinthitis, inner ear disease, or labyrinthine/AICA ischemia.

Audiometry as a localization tool

Pure-tone thresholds define the configuration and degree of hearing loss; speech recognition and rollover can add retrocochlear clues; tympanometry and acoustic reflexes help identify conductive or middle-ear confounders. A fluctuating low-frequency sensorineural pattern supports a cochlear hydrops phenotype but is not pathognomonic. Asymmetric sensorineural loss may prompt MRI of the internal auditory canals depending on degree, progression, speech findings, and clinical context. Hearing results should be interpreted alongside the vestibular syndrome, not as a separate chart.

Stance and gait

Observe the patient sitting unsupported, standing with a broad and then narrow base when safe, turning, and walking. Severe inability to sit or stand without support is strongly central-worrisome, although a profoundly nauseated patient with acute peripheral loss may need assistance walking. The key distinctions are truncal control, lateropulsion, limb coordination, and whether the gait deficit is proportionate to the ocular motor findings.

Table 7. What bedside balance observations can and cannot establish.

Observation	Interpretive value	Limitations
Falling or veering toward one side	May occur toward the side of acute peripheral hypofunction; can also reflect central lateropulsion.	Direction is not sufficiently specific to stand alone.
Unable to sit upright without support	Strong central red flag, especially with modest vertigo or eye findings.	Severe illness, sedation, weakness, and pain can confound.
Tandem gait impairment	Sensitive to vestibular, cerebellar, proprioceptive, and age-related dysfunction.	Poor specificity; normal performance does not exclude stroke.
Romberg worsens with eyes closed	Demonstrates dependence on vision when proprioceptive or vestibular input is impaired.	Does not distinguish vestibular from proprioceptive loss.
Fukuda/Unterberger rotation	May suggest asymmetry in some patients.	Insufficiently reliable for lesion side or diagnosis; do not overinterpret.

Dynamic visual acuity

Dynamic visual acuity tests whether the patient can preserve visual detail while the head is moving. Measure static acuity, then repeat while the head oscillates horizontally at a standardized frequency and amplitude. A loss of several lines or at least 0.2 logMAR in an appropriate protocol supports impaired gaze stability, particularly bilateral vestibular hypofunction. The result also depends on baseline acuity, neck mobility, oscillation speed, predictive strategies, and the patient's ability to read the chart.

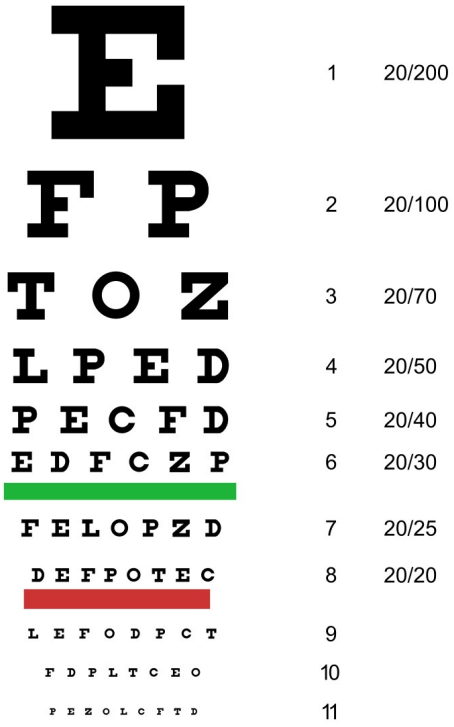


Figure 10. Snellen-style visual acuity chart used to illustrate the principle of comparing static and dynamic visual acuity. Jeff Dahl, via Wikimedia Commons, CC BY-SA 3.0.

Subjective visual vertical

The patient aligns a luminous line to perceived gravity in darkness, or performs a bedside bucket test. Acute unilateral peripheral vestibular loss often tilts perceived vertical toward the affected side, reflecting otolith tone imbalance. Brainstem, cerebellar, thalamic, and cortical lesions can produce larger or differently directed tilts. The test is useful as evidence of graviceptive imbalance but rarely identifies a disease by itself.

Oscillopsia and bilateral loss

Ask whether the environment bounces or blurs during walking, driving over uneven roads, or quick head movement. Oscillopsia during head motion is a functional expression of VOR failure and is characteristic of bilateral vestibular hypofunction, though severe unilateral loss can cause it acutely. Patients with bilateral loss often report little spinning vertigo; instead they describe visual instability and imbalance that worsens in darkness or on uneven ground. This symptom pattern should direct testing toward bilateral high- and mid-frequency VOR function. [16]

> **Clinical bridge**
>
> A patient who walks reasonably in bright hallways but becomes markedly unstable in darkness and reports bouncing vision while walking has already supplied a physiologic hypothesis: reduced vestibular contribution with visual and somatosensory substitution. The laboratory should test that hypothesis across frequencies.

THE LABORATORY SAMPLES PHYSIOLOGY

7. The vestibular laboratory: tests, frequencies, and pathways

No single vestibular test measures “the vestibular system.” Each test applies a particular stimulus, samples a frequency range, emphasizes specific end organs and reflex pathways, and is vulnerable to specific artifacts. Interpretation begins by naming those constraints. [12-18]

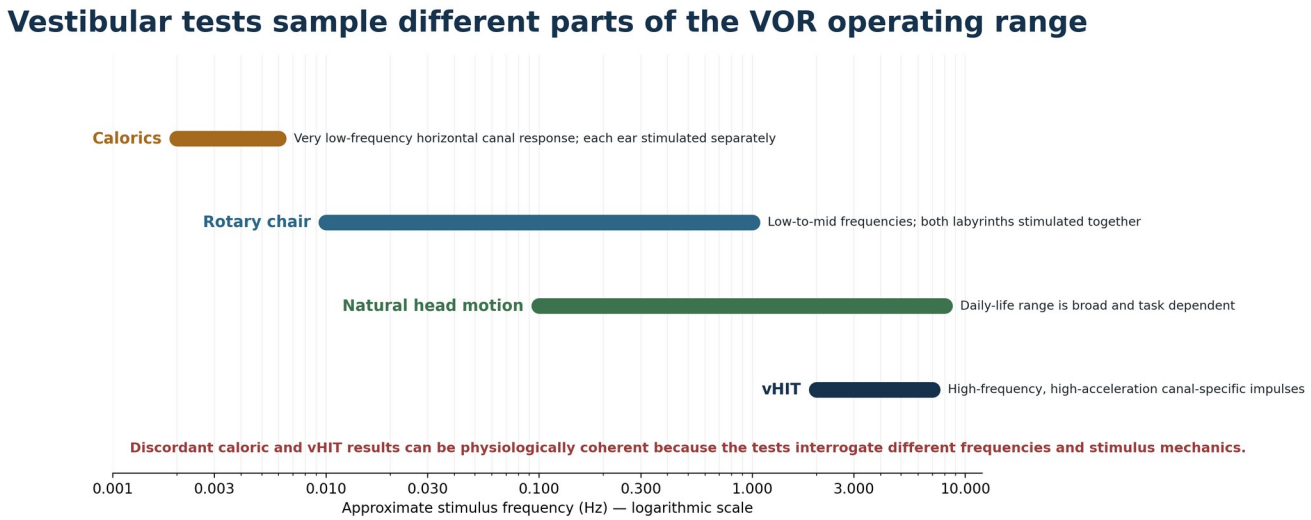


Figure 11. Calorics, rotary chair, and vHIT interrogate different stimulus frequencies and therefore may disagree without either test being wrong. Original synthesis.

VNG and ENG: the recording platform

Videonystagmography records eye position with infrared cameras and can capture horizontal, vertical, and sometimes torsional components. Electronystagmography uses the corneoretinal potential and is most robust for horizontal movements but cannot directly characterize torsion. A standard battery may include spontaneous and gaze testing, saccades, pursuit, optokinetic responses, positional testing, and calorics. The central ocular motor subtests are sensitive to many factors, so abnormality should be interpreted in clinical context rather than converted automatically into “central vestibulopathy.”

Video head impulse testing

vHIT quantitatively records head velocity and eye velocity during rapid, small-amplitude impulses. It estimates VOR gain and detects corrective saccades that may be covert during the impulse or overt after the head stops. With appropriate technique, all six canals can be tested: horizontal impulses for lateral canals and diagonal LARP and RALP planes for vertical canal pairs. [12,13]

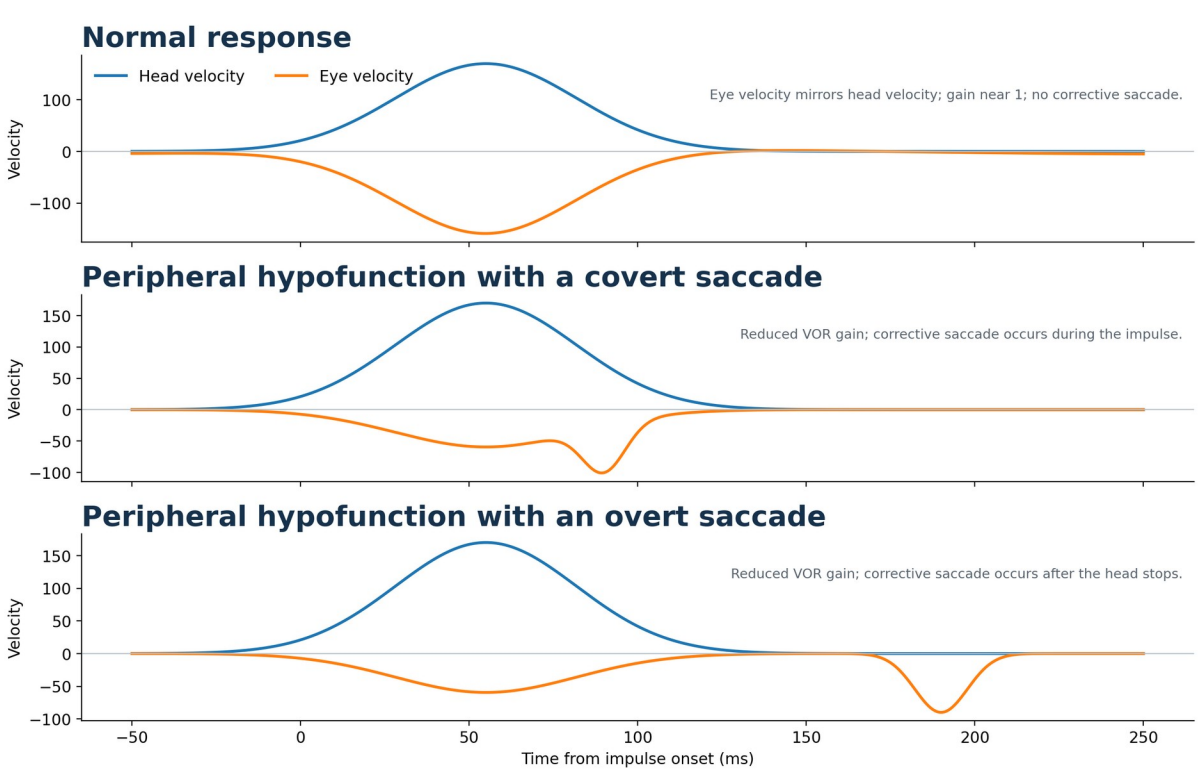


Figure 12. Schematic vHIT traces showing normal VOR, reduced gain with a covert saccade, and reduced gain with an overt saccade. Original synthesis.

Interpret gain and saccades together

A low gain with reproducible, appropriately directed corrective saccades is stronger evidence of canal hypofunction than either finding alone. Device algorithms, age, target distance, goggle fit, calibration, impulse velocity, and canal plane influence normative thresholds. Horizontal gain below roughly 0.8 and vertical gain below roughly 0.7 are often treated as abnormal in clinical systems, but laboratory-specific norms are essential. The Bárány criteria for bilateral vestibulopathy use a more severe bilateral horizontal vHIT threshold below 0.6. [16]

Common vHIT artifacts

Table 8. High-yield vHIT artifacts and how to recognize them.

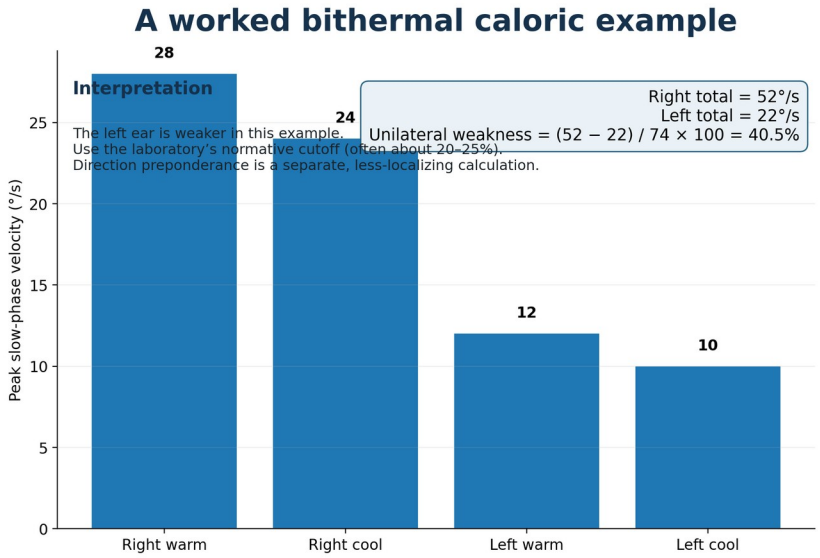
Artifact	Appearance	Prevention / interpretation
Goggle slippage	Spurious high or low gain, often with oscillation or nonphysiologic eye trace.	Tight fit, stable facial contact, smaller impulses, inspect raw traces.
Poor calibration	Systematic gain error across impulses.	Recalibrate with clear targets and adequate vision.
Predictable impulses	Preprogrammed saccades that can mimic or mask covert corrections.	Randomize timing and direction.
Blink / eyelid artifact	Abrupt eye-velocity spikes not coupled to head movement.	Repeat impulse and inspect video/raw data.
Wrong canal plane	Apparent vertical canal asymmetry or cross-coupling.	Align impulses precisely in LARP or RALP planes and account for gaze position.

Artifact	Appearance	Prevention / interpretation
Too slow / too large	Under-challenges the VOR and introduces pursuit or neck motion.	Use small, brisk, high-acceleration impulses within comfort and safety limits.

Caloric testing

Bithermal caloric testing stimulates each horizontal canal separately at an extremely low effective frequency. With the head elevated about 30 degrees, warm or cool irrigation creates a temperature gradient in the lateral canal and changes cupular drive. In an awake patient, warm irrigation produces fast phases toward the irrigated ear and cool irrigation produces fast phases away - the familiar COWS mnemonic describes the fast phase, not the slow vestibular drift.

The major strength of calorics is ear-specific low-frequency testing. The major limitation is that the stimulus is nonphysiologic, depends on external and middle-ear heat transfer, and interrogates only a narrow portion of horizontal canal function. Cerumen, tympanic membrane status, prior mastoid surgery, air versus water technique, baseline nystagmus, medications, alertness, and fixation all influence the result.



Do not call this "left vestibular neuritis" from calorics alone. The test identifies low-frequency horizontal canal pathway asymmetry, not etiology or chronicity.

Figure 13. Worked example of unilateral weakness calculated from peak slow-phase velocities. Original synthesis.

Jongkees calculations

Unilateral weakness compares the summed warm and cool responses of one ear with the summed responses of the other ear, divided by the total response. The sign convention varies; clinically, report the weaker ear and the percentage. Many laboratories use an abnormal cutoff near 20 to 25 percent. Direction preponderance compares right-beating with left-beating responses. It may reflect baseline spontaneous nystagmus or central bias and is generally less localizing than unilateral weakness. Always use the laboratory's validated norms.

Caloric-vHIT dissociation

A patient can have abnormal calorics and normal vHIT because low-frequency horizontal canal function is impaired while high-frequency function is preserved. This pattern is common in some hydropic disorders. Conversely, severe high-frequency deficits may be more obvious on vHIT. Discordance should prompt physiologic interpretation, not dismissal of one test.

Rotary chair testing

Rotary chair stimulates both labyrinths simultaneously and samples low-to-mid frequencies between calorics and vHIT. Sinusoidal harmonic acceleration measures gain, phase, and symmetry across frequencies. Step-velocity testing assesses the time constant and velocity storage. Visual-VOR and VOR-suppression paradigms assess visual-vestibular interaction and cerebellar control. Rotary chair is particularly useful when bilateral hypofunction is suspected, when caloric responses are absent or technically limited, and when the degree of compensation must be characterized. It is less effective than calorics for identifying the side of a compensated unilateral lesion because both ears are driven together.

Figure 14. Rotary chair testing illustrates controlled whole-body angular stimulation of the VOR. Victor Zelentsov/NASA, public domain.

Vestibular evoked myogenic potentials

VEMPs use sound or vibration to evoke short-latency muscle responses through vestibular pathways. They are not direct electrical recordings from the saccule or utricle. The stimulus must reach the labyrinth, activate a receptor population, traverse the vestibular nerve and central reflex arc, and

produce a recordable muscle response. A missing waveform can therefore arise anywhere along that chain.

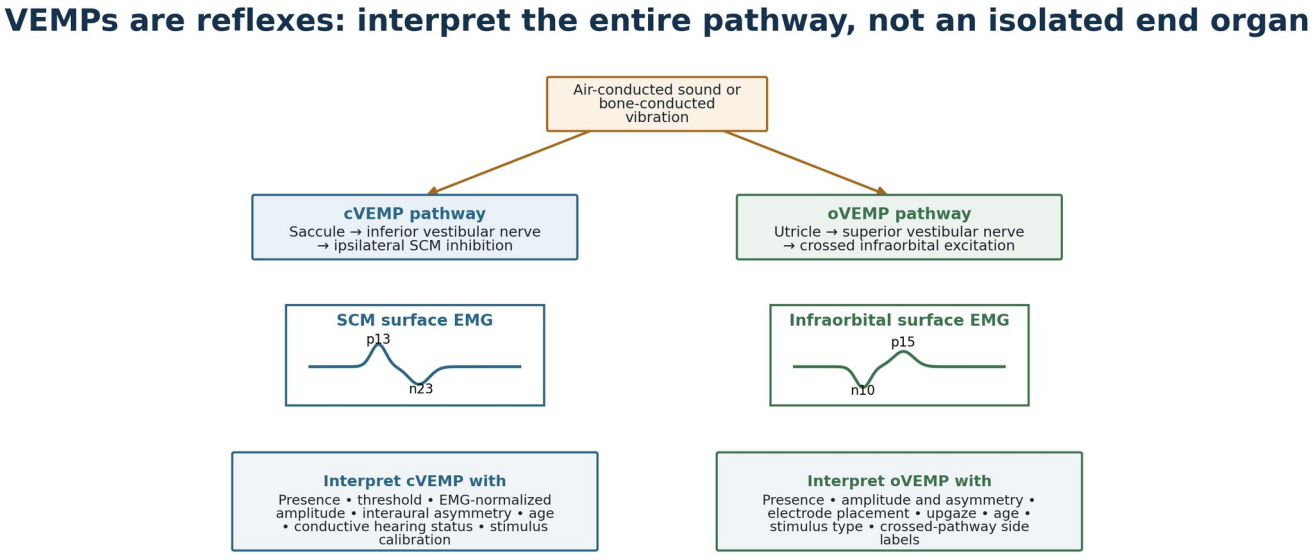


Figure 15. The dominant cVEMP and oVEMP pathways and the variables that must be considered during interpretation. Original synthesis.

cVEMP

The air-conducted cVEMP predominantly reflects saccular and inferior vestibular nerve function through an ipsilateral inhibitory vestibulocollic reflex recorded over the contracted sternocleidomastoid. Amplitude depends strongly on tonic SCM activation, so raw interaural amplitude differences are uninterpretable without monitoring or normalization. Conductive hearing loss can attenuate or abolish air-conducted responses despite intact vestibular function. Threshold, normalized amplitude, waveform reproducibility, and age-specific norms are all relevant. [17,18]

oVEMP

The oVEMP predominantly reflects utricular and superior vestibular nerve function through a crossed excitatory pathway recorded beneath the eye contralateral to the stimulated ear. Upgaze activates the inferior oblique region and increases the response. Amplitudes decline with age and are sensitive to electrode placement, stimulus type, and facial anatomy. Because the pathway crosses, side labels must be stated explicitly: the response under the left eye after right-ear stimulation is attributed primarily to the right utricular-superior nerve pathway.

Third-window physiology

A third mobile window can make vestibular organs unusually sensitive to sound or vibration, producing lower VEMP thresholds and/or larger amplitudes. These findings support but do not independently diagnose a third-window syndrome. Symptoms, audiometry, middle-ear measures, and high-resolution temporal bone CT reconstructed in the relevant canal plane must be concordant. An anatomic dehiscence on CT without the appropriate physiologic syndrome may be incidental or overcalled.

Posturography and sensory organization

Computerized dynamic posturography measures sway under conditions that alter visual and somatosensory reliability. It can demonstrate sensory dependence, quantify functional balance impairment, and guide rehabilitation or fall-risk assessment. It is generally weak for anatomic localization because many peripheral, central, musculoskeletal, visual, neuropathic, cognitive, and functional disorders can produce overlapping patterns. Treat it as a performance test rather than an imaging study of the labyrinth.

A test-to-question matrix

Table 9. Choose the vestibular test by the unresolved physiologic question.

Question	Most useful test	What a positive result establishes	What it does not establish
Is one horizontal canal pathway weaker at very low frequency?	Bithermal calorics	Ear-specific low-frequency asymmetry.	Etiology, chronicity, or function of vertical canals.
Do individual canals generate an adequate high-frequency VOR?	Six-canal vHIT	Canal-specific gain and corrective saccades.	Low-frequency function or otolith function.
Is bilateral VOR function reduced across low-to-mid frequencies?	Rotary chair	Reduced gain/abnormal phase compatible with bilateral hypofunction.	Precise lesion side in a compensated unilateral process.
Is the saccular-inferior nerve reflex pathway recordable?	cVEMP	A reproducible cervical reflex under controlled conditions.	A pure isolated measurement of the saccule.
Is the utricular-superior nerve reflex pathway recordable?	oVEMP	A reproducible crossed ocular reflex.	A pure isolated measurement of the utricle.
Is gaze stability functionally impaired during head motion?	Dynamic visual acuity / functional HIT	Loss of acuity or target recognition during movement.	The exact end organ or lesion site.
How does the patient weight vision, surface, and vestibular cues?	Dynamic posturography	A sensory-performance pattern and fall-related functional limitation.	Specific peripheral versus central localization.

IMAGE THE HYPOTHESIS, NOT THE SYMPTOM

8. Imaging and choosing the next test

Dizziness is not an imaging indication by itself. Imaging becomes useful when the bedside and audiovestibular evaluation leaves a specific structural, vascular, retrocochlear, or bony question. [3,10,19,20]

Noncontrast CT

Noncontrast head CT is appropriate when the question is intracranial hemorrhage, major trauma, mass effect, or another CT-visible emergency. It is insensitive for small posterior fossa ischemic strokes and should not be used to reassure a patient with central-worrisome AVS. Beam-hardening in the posterior fossa further limits detection. A normal CT does not neutralize a central ocular motor pattern.

MRI with diffusion-weighted imaging

MRI with DWI is the preferred parenchymal study for posterior fossa ischemia, demyelination, tumor, and many central vestibular syndromes. Early DWI can be false-negative, particularly for small brainstem or cerebellar infarcts. When bedside findings and clinical trajectory remain strongly concerning, neurology/stroke evaluation, vascular imaging, observation, and repeat MRI may be warranted despite an initially negative study. Imaging timing and local protocols should be discussed with the treating stroke and radiology teams.

CTA and MRA

Vascular imaging answers a vascular question: suspected dissection, basilar or vertebral occlusion, high-risk TIA, or posterior circulation ischemia requiring vessel assessment. It should not be ordered reflexively for every dizzy patient. New neck pain, severe occipital headache, focal neurologic findings, recurrent brief brainstem symptoms, and high-risk AVS may raise the pretest probability.

MRI of the internal auditory canals

Dedicated IAC imaging is used for asymmetric sensorineural hearing loss, unilateral tinnitus in selected contexts, retrocochlear signs, facial or trigeminal involvement, or concern for a cerebellopontine angle process. The indication arises from the audiologic and neurologic pattern, not from chronic dizziness alone.

High-resolution temporal bone CT

Temporal bone CT is most useful for bony anatomy: suspected superior canal dehiscence or another third-window lesion, temporal bone trauma, labyrinthine fistula, otic capsule disease, or surgical planning. Thin slices and multiplanar reconstructions aligned with the canal are important. Because apparent dehiscence can be overestimated by partial-volume artifact, CT findings should agree with symptoms and physiologic testing.

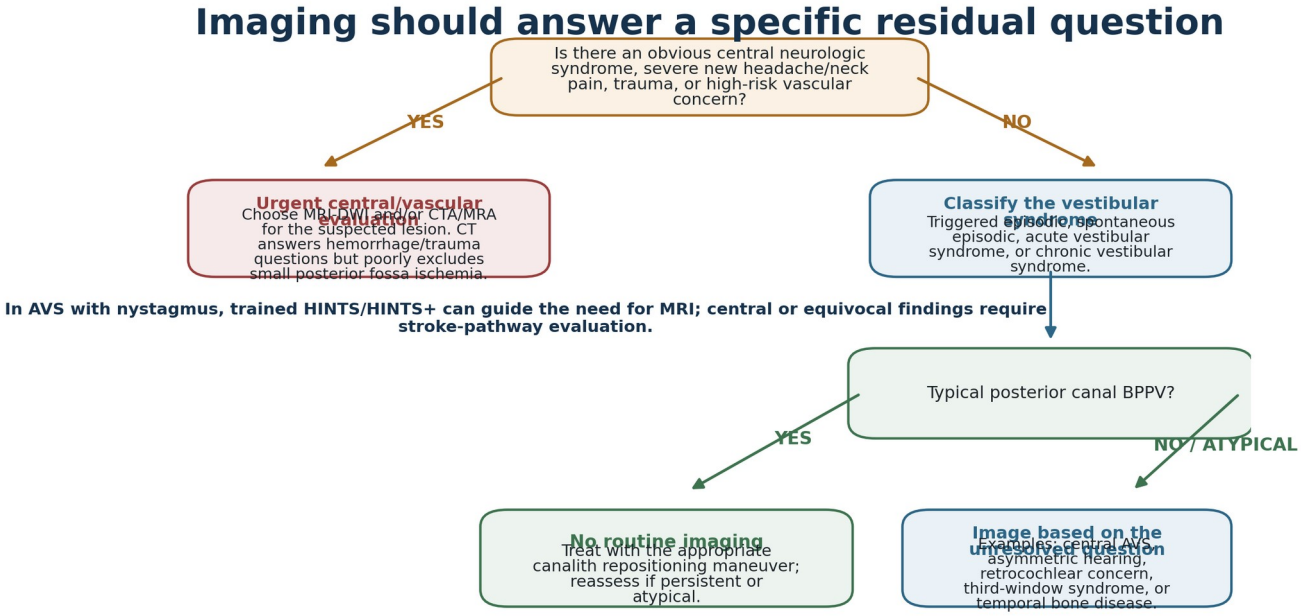


Figure 16. Imaging is selected after the syndrome and residual question are defined. Original synthesis.

When not to image

Patients who meet clinical criteria for typical posterior canal BPPV generally do not need routine imaging. The AAO-HNS guideline emphasizes positional diagnosis and appropriate repositioning rather than low-yield radiographic testing. Imaging becomes appropriate when the response is atypical, central signs are present, the syndrome is not truly triggered episodic, or the patient fails a well-selected and correctly performed management pathway. [10]

The “next test” hierarchy

1. Restate the syndrome and the current localization hypothesis.
2. Identify the single uncertainty that would change management.
3. Choose the bedside maneuver, audiogram, vestibular test, or imaging study that samples that uncertainty.
4. Predict the result expected under the leading hypothesis before ordering the test.
5. After the result returns, ask whether it is technically valid and whether it explains the clinical syndrome.
6. If results conflict, reconcile differences in stimulus frequency, end-organ coverage, timing, compensation, and artifacts before inventing a second disease.

> **A useful documentation sentence**
>
> “Because the patient has continuous AVS with direction-changing gaze-evoked nystagmus and a normal bedside head impulse, I am concerned for a central lesion and recommend stroke-pathway MRI-DWI and vascular evaluation.” The sentence connects syndrome, sign, localization, and test.

PUT THE SEQUENCE INTO PRACTICE

9. Four integrated clinical encounters

The following cases are deliberately organized by the first five minutes of care. The diagnosis is not pulled from one symptom or one test; it emerges from a sequence of syndrome classification, objective examination, and selective testing.

Encounter 1: brief vertigo when rolling in bed

A 46-year-old patient reports intense spinning for 15 to 30 seconds when rolling to the right or looking upward. She is asymptomatic while sitting still. There is no hearing change, headache, diplopia, or gait difficulty between attacks.

Step 1 - classify the syndrome

This is triggered episodic vestibular syndrome. The position change causes a discrete attack from baseline; it is not simply worsening continuous dizziness.

Step 2 - choose the examination

Perform ocular motor screening in primary gaze, then right and left Dix-Hallpike maneuvers while observing the eyes. A right Dix-Hallpike produces a short-latency, transient upbeat torsional nystagmus with upper poles beating toward the right ear. The vector and time course identify right posterior canal canalithiasis.

Step 3 - decide whether more testing is needed

No routine MRI, CT, calorics, or vHIT is needed when the history and nystagmus are typical and there are no central signs. Treat with a canal-specific repositioning maneuver and reassess. If the response were horizontal, the next test would be a supine roll test, not imaging by reflex.

> **What changed management**
>
> The observed canal-plane nystagmus converted a symptom report into a mechanical diagnosis.

Encounter 2: continuous vertigo and vomiting since morning

A 67-year-old patient develops abrupt continuous vertigo, vomiting, and inability to walk without support. Examination shows spontaneous left-beating nystagmus. There is no obvious limb weakness or aphasia.

Step 1 - classify the syndrome

This is acute vestibular syndrome. The first task is not to ask whether the patient “feels peripheral”; it is to distinguish acute peripheral vestibular loss from posterior circulation stroke.

Step 2 - confirm the HINTS gate

The patient is continuously symptomatic and has spontaneous nystagmus. The examiner is trained in HINTS. Perform small, unpredictable head impulses, assess nystagmus in primary and eccentric gaze, perform alternate cover testing, compare bedside hearing, and grade gait/truncal control.

Peripheral-coherent branch

A clear corrective saccade after impulses to the right, unidirectional left-beating horizontal-torsional nystagmus, no skew, no new hearing loss, and a gait deficit proportionate to vertigo form a coherent right peripheral pattern. The bedside pattern supports acute right unilateral vestibular hypofunction, while clinical context still determines need for imaging or consultation.

Central-worrisome branch

Normal impulses, direction-changing gaze-evoked nystagmus, skew, new unilateral hearing loss, severe truncal instability, or other central ocular motor signs require stroke-pathway evaluation. A negative noncontrast CT would not resolve the concern. MRI-DWI and vascular assessment are selected because the residual question is posterior circulation ischemia.

> **What changed management**
>
> HINTS was useful because the patient met the syndrome gate and each component was documented as a physiologic observation.

Encounter 3: recurrent spontaneous attacks with auditory symptoms

A 39-year-old patient has three episodes of spontaneous vertigo lasting one to three hours. During attacks, the left ear feels full and hearing seems muffled. Between attacks, the examination is normal except for mild asymmetric low-frequency sensorineural hearing loss on audiometry.

Step 1 - classify the syndrome

This is spontaneous episodic vestibular syndrome. HINTS is not appropriate between attacks, and a negative Dix-Hallpike would not address the main problem.

Step 2 - use auditory and interval data

The key evidence is the recurrent episode duration and the temporally linked unilateral auditory phenotype. Audiometry documents a cochlear asymmetry. Vestibular testing may characterize baseline function, but a normal vHIT does not exclude a hydropic disorder, and caloric-vHIT dissociation may occur because the tests sample different frequencies.

Step 3 - exclude competing patterns

Ask about migrainous features, neurologic symptoms, vascular risk, and attack-to-attack consistency. MRI IAC may be appropriate for asymmetric sensorineural loss depending on local criteria, while acute stroke imaging is not triggered solely by a resolved recurrent pattern without central features.

> **What changed management**
>
> The episode phenotype and audiogram determined the pathway; the absence of findings between attacks did not invalidate the syndrome.

Encounter 4: imbalance in darkness and bouncing vision

A 72-year-old patient reports progressive unsteadiness over months, especially in darkness and on uneven ground. He denies spinning attacks but says signs and faces bounce while he walks. Bedside head impulses show bilateral corrective saccades, and dynamic visual acuity declines by four lines.

Step 1 - classify the syndrome

This is a chronic vestibular syndrome with oscillopsia and visual dependence, strongly suggesting bilateral vestibular hypofunction.

Step 2 - test across frequencies

Quantify horizontal and vertical canal function with vHIT and assess low-to-mid frequency bilateral VOR function with rotary chair. Calorics may be included but can be absent for technical or severe bilateral reasons and must be interpreted with the broader battery. Review ototoxic exposure, neuropathy, cerebellar signs, hearing status, and medication history.

Step 3 - connect laboratory data to function

Bilateral low vHIT gain with grouped corrective saccades, reduced rotary chair gain, and impaired dynamic visual acuity create a coherent physiologic and functional picture. Posturography may quantify sensory dependence and assist rehabilitation planning but is not needed to establish the lesion by itself.

> **What changed management**
>
> The complaint of “no vertigo” did not argue against vestibular disease. Bilateral loss often presents as oscillopsia and imbalance rather than spinning.

A final resident algorithm

1. Classify timing and triggers before naming diseases.
2. Observe primary gaze, alignment, gaze holding, saccades, pursuit, stance, and gait before provoking symptoms.
3. Describe nystagmus by waveform, axis, direction, gaze dependence, fixation dependence, and positional context.
4. Use HINTS/HINTS+ only for eligible AVS with ongoing nystagmus and a trained examiner.
5. Use positional maneuvers for triggered episodic syndromes and interpret the eye-movement vector in canal geometry.
6. Select vestibular tests by end organ, reflex pathway, and frequency range.
7. Use imaging to answer a residual structural or vascular question.
8. Reconcile discordant results through physiology, timing, compensation, and artifacts.

RAPID RETRIEVAL

APPENDIX A. High-yield localization maps

Test-to-structure and frequency matrix

Table 10. One-page vestibular test matrix.

Test	Dominant organ/pathway	Frequency / stimulus	Best use	Major trap
Bedside HIT	Canal-VOR, mainly horizontal at bedside	High acceleration, high frequency	Detect overt unilateral or bilateral hypofunction in real time.	Covert saccades and poor technique cause false negatives.
vHIT	All six canal VOR pathways	High frequency/high acceleration	Canal-specific gain and saccades.	Goggle slip, calibration, canal-plane error.
Calorics	Horizontal canal-superior nerve pathway, each ear separately	Very low effective frequency; thermal	Quantify unilateral low-frequency asymmetry.	External/middle ear anatomy and alertness influence response.
Rotary chair	Bilateral horizontal canal VOR	Low-to-mid frequency angular motion	Confirm/quantify bilateral loss; compensation and velocity storage.	Poor side localization for compensated unilateral lesions.
cVEMP	Predominantly saccule-inferior vestibular nerve to ipsilateral SCM	Sound or vibration reflex	Inferior division and third-window physiology.	SCM contraction and conductive loss dominate amplitude/presence.
oVEMP	Predominantly utricle-superior vestibular nerve to crossed extraocular response	Sound or vibration reflex	Superior division and third-window physiology.	Age, upgaze, electrode placement, crossed side labels.
Dynamic visual acuity	Functional VOR/gaze stability	Active or passive head oscillation	Functional bilateral or severe unilateral gaze instability.	Baseline vision and predictive strategies.
Posturography	Integrated sensory weighting and postural performance	Altered visual/surface conditions	Quantify balance strategy and guide rehabilitation.	Weak anatomic localization.

Nystagmus localization card

Table 11. Rapid nystagmus pattern-to-action map.

Observed pattern	Most likely implication	Immediate next step
Unidirectional horizontal-torsional spontaneous nystagmus	Peripheral-compatible tonic vestibular asymmetry.	Look for abnormal HIT toward the slow-phase/lesion side, fixation suppression, no skew, and coherent gait pattern.

Observed pattern	Most likely implication	Immediate next step
Direction-changing horizontal gaze-evoked nystagmus	Central gaze-holding dysfunction.	Complete ocular motor examination and pursue central evaluation in the appropriate syndrome.
Pure downbeat or upbeat spontaneous nystagmus	Central localization strongly favored.	Assess cerebellar/brainstem signs, medications/metabolic causes, and obtain appropriate imaging.
Upbeat torsional nystagmus on Dix-Hallpike	Posterior canal BPPV if canal-plane appropriate and transient.	Treat with canal-specific repositioning; no routine imaging if typical.
Geotropic horizontal positional nystagmus	Horizontal canal canalithiasis; stronger side usually affected.	Use side comparison and horizontal canal repositioning strategy.
Apogeotropic horizontal positional nystagmus	Cupulolithiasis or short-arm debris; weaker side often affected.	Use additional lateralizing signs and avoid overconfidence.
Persistent positional downbeat without convincing torsion	Central positional nystagmus must be considered.	Search for central ocular motor/neurologic signs and image when appropriate.

Formula card

Table 12. Common calculations and the caveat attached to each one.

Measure	Formula / convention	Interpretive note
VOR gain	Eye velocity ÷ head velocity, with sign handled by device convention.	Interpret with saccades and raw traces; use device/lab norms.
Caloric unilateral weakness	(Stronger ear total − weaker ear total) ÷ total of all four irrigations × 100.	Report weaker ear and percentage; common cutoff approximately 20–25%, lab specific.
Caloric direction preponderance	Difference between total right-beating and left-beating responses ÷ total × 100.	Can reflect spontaneous nystagmus; less localizing than unilateral weakness.
VEMP interaural asymmetry	|Right normalized amplitude − Left normalized amplitude| ÷ sum × 100.	Requires appropriate EMG normalization and laboratory norms.
Dynamic visual acuity	Dynamic acuity minus static acuity in lines or logMAR.	Several-line or ≥0.2 logMAR loss can support bilateral hypofunction, protocol dependent.

SHARED VOCABULARY

APPENDIX B. Glossary

Acute vestibular syndrome (AVS). Acute onset of continuous dizziness or vertigo lasting days, generally with nausea, motion intolerance, gait disturbance, and spontaneous nystagmus.

Alexander's law. Increase in intensity of a unidirectional vestibular nystagmus when gaze is directed toward its fast phase.

Canalithiasis. Free otoconia moving within a semicircular canal and transiently deflecting the cupula after position change.

Covert saccade. Corrective refixation saccade occurring during a head impulse and often invisible to the unaided examiner.

Cupulolithiasis. Debris attached to the cupula, altering its sensitivity to gravity and often producing more persistent positional nystagmus.

Direction preponderance. Caloric asymmetry between right-beating and left-beating nystagmus, not necessarily a lesion-side measure.

Dynamic visual acuity. Visual acuity measured during head motion and compared with static acuity as a functional test of gaze stability.

Gaze-evoked nystagmus. Nystagmus generated or reversed by eccentric gaze because of impaired gaze holding or related central mechanisms.

HINTS. Head-Impulse, Nystagmus, Test-of-Skew examination for appropriately selected patients with AVS and ongoing nystagmus.

HINTS+. HINTS with a bedside assessment for new unilateral hearing loss.

Overt saccade. Corrective refixation saccade occurring after the head impulse has ended.

Skew deviation. Vertical ocular misalignment caused by imbalance in graviceptive/otolith-ocular pathways.

Triggered episodic vestibular syndrome. Brief attacks initiated by a reproducible action or position, with relative baseline between attacks.

Unilateral weakness. Caloric comparison of summed responses from the two ears, identifying low-frequency horizontal canal pathway asymmetry.

VOR gain. Ratio of compensatory eye velocity to head velocity; ideal magnitude approaches 1 during appropriate fixation conditions.

Velocity storage. Central mechanism that prolongs and shapes vestibular responses beyond the mechanical time constant of the canals.

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> **Educational use**
>
> This guide is intended for resident education. Bedside examination quality, local stroke pathways, laboratory normative data, patient comorbidities, and attending-level clinical judgment determine real-world management.