Bedside Clinical Examination in Vestibular Medicine

A Clinician's Evidence-Based Reference

Prepared by Australian Dizziness Clinics
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How to Use This Review

This literature review is designed as a comprehensive clinical reference for vestibular physicians and clinicians with a specialist interest in vestibular medicine. It covers the full spectrum of bedside vestibular examination — from nystagmus assessment and the HINTS protocol to positional testing, dynamic visual acuity, and cardiovascular evaluation.

Each section synthesises current evidence and clinical practice, with diagnostic accuracy data, technique descriptions, and interpretation principles. Callout boxes throughout the document highlight key points, clinical insights, and clinical pearls:

Key Point
Fundamental principles and must-know facts that anchor clinical reasoning.

Clinical Insight
Evidence-based nuances, caveats, and diagnostic considerations from the literature.

Clinical Pearl
High-yield practical tips for immediate bedside application.

Table of Contents

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Version History

Version	Date	Notes
v1.0	2026	Initial publication — full systematic bedside clinical examination review

I. Overview and Principles of Bedside Vestibular Examination

The bedside vestibular examination is the clinical cornerstone of vestibular medicine — an irreplaceable tool that no technology has yet displaced. It provides real-time, physiological assessment of VOR function, vestibulospinal reflexes, and brainstem-cerebellar integration, allowing the clinician to localise pathology, gauge severity, and direct urgent management decisions at the point of care.

Unlike formal vestibular function testing, which measures frequency-specific thresholds in a controlled laboratory environment, the bedside examination reveals how the vestibular system performs under naturalistic, high-acceleration conditions. The bedside head impulse test, for instance, mimics the rapid head movements of everyday life and exposes compensatory saccades invisible to video goggles during slow ramp-and-hold stimuli.

> **Key Point**
> *The HINTS examination (Head Impulse–Nystagmus–Test of Skew) has been shown to outperform early diffusion-weighted MRI in detecting posterior fossa stroke in acute vestibular syndrome, with a sensitivity of 100% and specificity of 96% in expert hands (Kattah et al., 2009).*

The Systematic Examination Sequence

A structured approach prevents omissions and ensures reproducibility. The recommended sequence is:

- **1. Inspection at rest:** Observe for spontaneous nystagmus in primary gaze with and without fixation
- **2. Ocular motor assessment:** Smooth pursuit, saccades, and gaze-evoked nystagmus
- **3. Fixation suppression test:** Does visual fixation dampen spontaneous nystagmus?
- **4. Bedside Head Impulse Test (bHIT):** Horizontal canals; extend to vertical planes if available
- **5. HINTS composite (in AVS):** HIT + nystagmus direction + Test of Skew — simultaneously
- **6. Head Shaking Nystagmus (HSN):** After 20 cycles at 2 Hz; look for contralateral fast phase or perverted nystagmus
- **7. Positional testing:** Dix-Hallpike bilaterally; Supine Roll Test if lateral canal BPPV suspected
- **8. Dynamic Visual Acuity:** Snellen chart — static vs. active head oscillation
- **9. Vibration-Induced Nystagmus (VIN):** 100 Hz over mastoid — confirms asymmetry, lateralises
- **10. Otoscopy and tuning forks:** Tympanic membrane, Weber, Rinne — correlate with auditory symptoms
- **11. Cardiovascular screen:** Supine and standing BP/HR — orthostatic hypotension and POTS
- **12. Cranial nerve screen:** CN V (facial sensation), CN VII (facial movement — peripheral vs. central pattern), CN IX/X (palate, dysarthria, dysphagia), CN XI, CN XII — essential in any AVS or suspected posterior fossa lesion
- **13. Cervical spine assessment:** Range of motion, reproduction of dizziness with neck movement, smooth pursuit neck torsion test, joint position error — for suspected cervicogenic dizziness

This sequence moves from least to most provocative, minimising patient discomfort and the risk of carry-over nystagmus contaminating subsequent tests.

> **Clinical Insight**
> *In acute presentations, prioritise the HINTS triad before positional testing — a normal HIT with direction-changing nystagmus in AVS is a stroke until proven otherwise and should trigger urgent*

neuroimaging rather than Dix-Hallpike manoeuvre.

□ Clinical Insight
A targeted cranial nerve screen takes under two minutes and should not be omitted in any acute vestibular presentation. The combination of ipsilateral Horner syndrome, ipsilateral facial hypoalgesia (CN V), ipsilateral palatal palsy (CN X), and contralateral limb pain and temperature loss in a patient with acute dizziness and imbalance is Wallenberg syndrome until proven otherwise — and will be missed without a structured cranial nerve assessment.

What the Bedside Examination Adds Beyond History and VFT

The structured history establishes temporal profile and symptom quality. Vestibular function tests quantify threshold impairment across frequencies. The bedside examination uniquely provides:

- **Dynamic localisation:** Real-time lateralisation and peripheral-vs-central discrimination
- **Functional severity:** Degree of VOR failure, compensatory mechanism activation
- **Triage utility:** Identifies the "must-not-miss" stroke in AVS — immediately actionable
- **Positional diagnosis:** Confirms and classifies BPPV — curative manoeuvre follows immediately
- **Serial monitoring:** Tracks central compensation or decompensation over follow-up visits

II. Nystagmus — Examination Technique and Diagnostic Significance

Nystagmus is involuntary rhythmic oscillation of the eyes. In vestibular medicine, it is the most diagnostically rich physical sign — reflecting the spatial distribution of vestibular tone, its laterality, the time course of a lesion, and whether pathology is peripheral or central. Precise characterisation of nystagmus is non-negotiable.

II.A — Examination Method: Room Light, Frenzel Goggles, and Video Goggles

Room-light examination allows only the most prominent nystagmus to be detected, as visual fixation actively suppresses peripheral vestibular nystagmus through the flocculonodular lobe. Studies suggest that up to 60–70% of spontaneous peripheral vestibular nystagmus is missed under standard room illumination, because the intact visual pathway inhibits the slow-phase drift.

Frenzel goggles (+20 dioptre lenses with internal illumination) eliminate fixation while magnifying the eyes, revealing nystagmus invisible in room light. They remain the pragmatic gold standard for clinic use — inexpensive, portable, and highly sensitive.

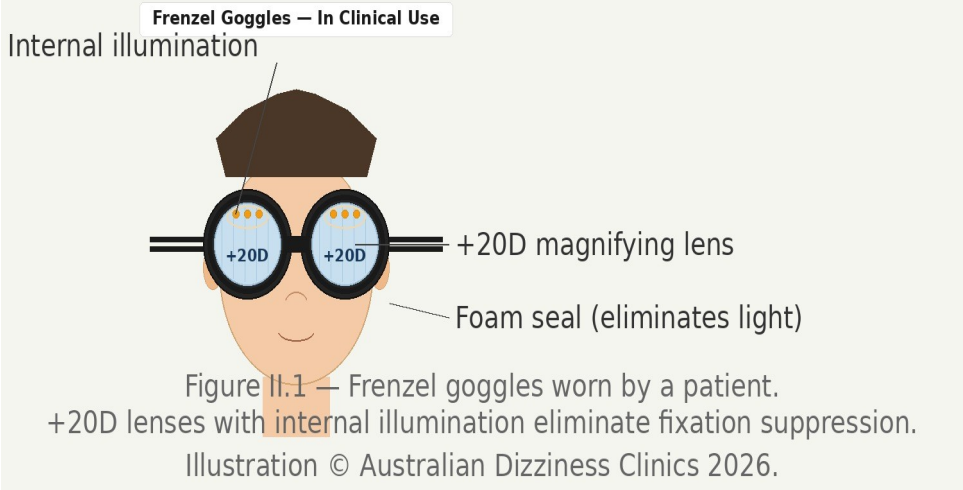

Figure II.1 — Frenzel goggles (Frenzelbrille): thick-framed spectacles fitted with +20 dioptre magnifying lenses and internal LED illumination. Illustration © Australian Dizziness Clinics 2026. Reference photograph: Polarlys, CC BY-SA 3.0, Wikimedia Commons.

Infrared video goggles (e.g., Interacoustics EyeSeeCam, ICS Impulse) offer quantitative, recorded nystagmus assessment with slow-velocity measurements. They are the preferred tool when available, particularly for detecting covert saccades during the head impulse test, subtle positional nystagmus, or vertical and torsional components that are easily missed by naked-eye observation.

> **□ Clinical Pearl**
> *Always examine nystagmus in darkness or with Frenzel goggles first. Then re-examine in room light to assess fixation suppression. The contrast between the two is diagnostic — not just the nystagmus itself.*

II.B — Alexander's Law and Nystagmus Grading

Alexander's Law describes the relationship between gaze direction and nystagmus intensity in peripheral vestibular lesions: nystagmus is greatest when gaze is directed in the direction of the fast phase, and diminishes (or reverses) when gaze is directed opposite to the fast phase. This reflects the additive or subtractive interaction between the vestibular imbalance signal and the gaze-holding neural integrator.

This allows bedside grading into three degrees:

- **First-degree:** Nystagmus present only on gaze toward the fast phase direction
- **Second-degree:** Nystagmus present in primary gaze and increases toward the fast phase
- **Third-degree:** Nystagmus present in all gaze positions, including gaze toward the slow phase

Third-degree nystagmus indicates a severe, acute, or incompletely compensated lesion. The degree of Alexander's Law compliance correlates inversely with the duration of the lesion — chronic compensation attenuates nystagmus intensity, often rendering it detectable only in Frenzel goggles.

II.C — Direction-Fixed vs. Direction-Changing Nystagmus

Direction-fixed nystagmus (fast phase consistently beats in the same direction regardless of gaze position) is the hallmark of peripheral vestibular lesions. The horizontal-torsional character reflects the unopposed activity of the contralateral vestibular nucleus following ipsilateral deafferentation.

Direction-changing nystagmus (also called gaze-evoked nystagmus) — in which the nystagmus changes direction depending on gaze — is a reliable indicator of central pathology, typically involving the cerebellar flocculus, gaze-holding neural integrator, or medullary vestibular nuclei. It reflects the failure of the neural integrator to maintain eccentric gaze against elastic restoring forces.

> **Clinical Insight**
> *Direction-changing positional nystagmus can occur peripherally (e.g., cupulolithiasis lateral canal BPPV with apogeotropic nystagmus), and should not automatically be labelled central. The critical distinguishing feature is the clinical context, associated signs, and the full HINTS evaluation. Pure vertical nystagmus or torsional nystagmus without a horizontal component strongly implicates central pathology.*

II.D — Fixation Suppression: Peripheral vs. Central Significance

In healthy individuals and in peripheral vestibular lesions, the vestibulocerebellum (specifically the flocculus and paraflocculus) mediates active suppression of the VOR during smooth pursuit — this same pathway suppresses spontaneous vestibular nystagmus under visual fixation. Peripheral vestibular nystagmus is therefore characteristically suppressed when the patient fixes on a stationary target.

In central lesions involving the flocculus, brainstem, or cerebellar pathways, this suppression mechanism is disrupted. Nystagmus persists or worsens despite attempted fixation — the fixation suppression failure sign. This is one of the highest-yield discriminators of central from peripheral nystagmus at the bedside.

- **Fixation suppresses nystagmus:** Strongly favours peripheral vestibular aetiology
- **Fixation fails to suppress or worsens nystagmus:** Red flag for central pathology — requires neuroimaging

II.E — Nystagmus Planes and the Clinical Significance of Vertical Nystagmus

The plane of nystagmus reflects which vestibular pathways are active or imbalanced:

Nystagmus Plane	Peripheral Interpretation	Central Interpretation
Horizontal-torsional	HSCC or superior vestibular nerve lesion — expected	Rarely — lateral medullary infarct
Pure horizontal	May occur with chronic compensation	Pontine gaze pathways
Pure vertical (upbeat/downbeat)	Not a peripheral sign — almost always central	Brainstem, cerebellar flocculus, or craniocervical junction
Pure torsional	Not expected peripherally	Midbrain, interstitial nucleus of

Nystagmus Plane	Peripheral Interpretation	Central Interpretation
		Cajal
Periodic alternating	Not a peripheral sign	Nodulus, uvula of cerebellum

Table II.1 — Nystagmus plane as a localising sign. Pure vertical or torsional nystagmus mandates central localisation.

Clinical Pearl
Downbeat nystagmus (fast phase downward) in primary gaze or on lateral gaze is never a peripheral sign. It localises to the vestibulocerebellum (flocculus/nodulus), the craniocervical junction, or medullary pathways, and requires MRI with posterior fossa sequences — not Dix-Hallpike.

III. Bedside Head Impulse Test (bHIT)

The head impulse test (HIT), originally described by Halmagyi and Curthoys in 1988, is the single most important bedside test of semicircular canal function. It directly assesses the gain of the vestibulo-ocular reflex (VOR) by measuring whether the eyes remain stationary in space during a rapid, unpredictable head rotation.

III.A — Physiological Basis and Technique

When the head rotates rapidly, the ipsilateral semicircular canal afferents fire a burst of action potentials, driving contralateral eye movement to maintain gaze on the target. An intact VOR produces eye velocity approximately equal and opposite to head velocity — a gain of ~1.0. In unilateral semicircular canal hypofunction, afferent firing is reduced on the affected side, and the VOR fails to drive the eye adequately. The eyes lag behind the target, and a corrective catch-up saccade is generated to refixate.

Technique:

- **Patient position:** Seated, examiner holds the head gently but firmly with both hands
- **Target:** Patient fixates the examiner's nose or a target on the wall 1–2 metres away
- **Head movement:** Small amplitude (~10–20°), very high velocity (>150°/s) horizontal impulses delivered unpredictably to left and right
- **Observation:** Does the eye remain on target throughout, or does it slip off target followed by a corrective saccade?

A positive bHIT (abnormal) — in which a catch-up saccade is visible — indicates reduced VOR gain on the side toward which the head was impulsed.

> **Clinical Pearl**
> *In acute vestibular neuritis affecting the right side, the bHIT is abnormal (corrective saccade visible) when the head is impulsed toward the right (toward the affected ear), and normal when impulsed toward the left. This corresponds to right HSCC hypofunction.*

III.B — Overt vs. Covert Saccades

The original bedside HIT detects only overt (visible) corrective saccades — those that occur after the head has stopped moving. Newer video-HIT technology has revealed that covert saccades — rapid compensatory eye movements occurring during the head impulse itself — are equally pathognomonic of VOR hypofunction but are invisible to the naked eye.

Covert saccades occur so quickly (within the first 100–150 ms of head rotation) that they are complete before the head movement ends. In patients who have developed covert saccades, the bHIT may appear normal (no visible overt saccade) despite significant HSCC hypofunction detected on vHIT. This is the primary limitation of the clinical bedside test.

- **Overt saccade:** Visible corrective movement after head stops — reliably detected clinically
- **Covert saccade:** Occurs during head movement — invisible clinically, detected only by vHIT
- **Mixed:** Both components may coexist, particularly in subacute or partially compensated lesions

III.C — Sensitivity, Specificity, and Limitations vs. vHIT

The bedside HIT has moderate sensitivity for unilateral HSCC hypofunction. Published data suggest sensitivity of approximately 35–71% for peripheral vestibular hypofunction when performed clinically, compared to vHIT as the reference standard (Jorns-Häderli et al., 2007; Weber et al., 2008). Specificity is high (~95–98%) — a positive bHIT reliably indicates peripheral hypofunction in the appropriate clinical context.

The sensitivity gap is explained by covert saccades: patients who develop covert saccades early in their recovery (within days of onset) will test negatively on bHIT despite persistent vHIT abnormality. This has important implications for acute assessment — a "negative" bHIT in the right clinical context should not be interpreted as a normal VOR.

Feature	Bedside HIT (bHIT)	Video HIT (vHIT)
Equipment	None	Infrared goggles + software
Detects overt saccades	Yes — reliable	Yes — recorded
Detects covert saccades	No — major limitation	Yes — primary advantage
VOR gain measurement	No	Yes — quantitative
Sensitivity (UVH)	~35–71%	~80–96%
Specificity	~95–98%	~90–96%
Vertical canals tested	No (clinical)	Yes (LARP/RALP)
Portability	Fully portable	Clinic-based

Table III.1 — Comparison of bedside HIT and video HIT (vHIT). UVH = unilateral vestibular hypofunction.

Key Point
A positive bHIT (corrective saccade) in a patient with acute vertigo and horizontal-torsional nystagmus strongly favours peripheral vestibulopathy. A negative bHIT in the same context does not exclude it — covert saccades may have already developed. In acute vestibular syndrome, the full HINTS triad must be assessed together.

IV. The HINTS Examination in Acute Vestibular Syndrome

The HINTS examination — Head Impulse, Nystagmus, Test of Skew — is the most evidence-based bedside protocol for evaluating the acute vestibular syndrome (AVS). Developed and validated by Kattah, Newman-Toker, and colleagues at Johns Hopkins, it exploits the specific oculomotor signatures of posterior fossa stroke versus acute peripheral vestibulopathy to achieve stroke detection accuracy that surpasses early diffusion-weighted MRI.

IV.A — Defining the Acute Vestibular Syndrome

The AVS is the clinical syndrome of acute onset, continuous (not episodic) vertigo or dizziness lasting more than 24 hours, with nausea/vomiting, gait instability, and nystagmus. The two most common diagnoses are:

- **Acute peripheral vestibulopathy (APV):** Formerly called "vestibular neuritis" — viral or post-viral inflammation of the vestibular nerve. Accounts for ~75–80% of AVS.
- **Posterior circulation stroke:** Infarction of the cerebellum or lateral medulla (PICA, AICA, or SCA territory). Accounts for ~5–10% of AVS — but carries high morbidity and risk of missed diagnosis.

The clinical challenge is that posterior fossa stroke can present with symptoms and signs indistinguishable from peripheral vestibulopathy — isolated vertigo, nystagmus, and gait instability without limb ataxia, Babinski signs, or other "hard" neurological findings. A normal CT is present in the majority of posterior fossa strokes in the first 24–48 hours. Early DWI-MRI has a false-negative rate of 12–17% for posterior fossa infarcts within the first 24–48 hours. The HINTS examination, in expert hands, outperforms all of these.

IV.B — HINTS Components and the INFARCT Mnemonic

The HINTS triad examines three specific oculomotor signs. Each component has a "safe" (peripheral) pattern and a "stroke" (central) pattern:

Component	Peripheral (Safe) Pattern	Central (Stroke) Pattern
H — Head Impulse Test	Positive bHIT — corrective catch-up saccade (reduced VOR gain on affected side)	Negative bHIT — no corrective saccade (normal-appearing VOR despite AVS)
N — Nystagmus	Direction-fixed, horizontal-torsional, suppressed by fixation	Direction-changing (alternates with gaze direction) OR vertical nystagmus OR nystagmus not suppressed by fixation
T — Test of Skew	No vertical misalignment of eyes on cover-uncover test	Skew deviation — vertical misalignment of eyes (one higher than the other) persisting on alternate cover test

Table IV.1 — HINTS components. A "safe" result across all three favours peripheral lesion. A single "stroke" sign mandates neuroimaging.

The INFARCT mnemonic operationalises the interpretation:

INFARCT: *Impulse Normal, Fast-phase Alternating, Refixation on Cover Test*

- **I** — Impulse Normal (no corrective saccade on bHIT): STROKE signal
- **N** — Nystagmus direction-changing or vertical: STROKE signal
- **F** — Fast phase Alternating (direction-changing nystagmus on lateral gaze): STROKE signal

- **R** — Refixation on Cover Test (vertical skew — one eye covers and then refixates vertically): STROKE signal

Any single INFARCT-positive finding in a patient with AVS mandates urgent MRI with DWI and posterior fossa sequences.

> **Key Point**
> *The HINTS exam is only valid in the acute vestibular syndrome — continuous vertigo lasting hours or more with nystagmus at rest. It cannot be applied to episodic vertigo (where nystagmus is typically absent between episodes) or to resolved symptoms. Applying HINTS outside AVS leads to false reassurance.*

IV.C — Diagnostic Accuracy and Comparison with Early DWI-MRI

The landmark study by Kattah et al. (2009, Stroke) evaluated HINTS prospectively in 101 patients with AVS. Sensitivity for stroke was 100% (no strokes missed), and specificity was 96% (few false positives). Critically, early DWI-MRI in the same cohort had a false-negative rate of 12% — HINTS outperformed neuroimaging in the first 24–48 hours.

A subsequent meta-analysis (Newman-Toker et al., 2013) confirmed that HINTS performed by trained clinicians achieves sensitivity of 96.5–100% for posterior fossa stroke in AVS, with specificity of 84–96%. However, training and experience are critical — sensitivity drops significantly with inexperienced examiners.

> **Clinical Insight**
> *In a patient presenting with AVS and a completely negative HINTS exam (positive bHIT, direction-fixed nystagmus, no skew), the pre-test probability of stroke drops dramatically. However, "negative HINTS" does not mean no further workup — MRI should follow within 24–48 hours in any admitted patient with AVS, as subacute DWI is more sensitive than hyperacute imaging.*

IV.D — HINTS-Plus: Hearing as the Fourth Element

Newman-Toker and colleagues proposed extending the HINTS triad to "HINTS-Plus" by incorporating acute hearing loss as a fourth element. This is based on the observation that sudden unilateral hearing loss accompanying AVS is almost always caused by labyrinthine infarction (occlusion of the labyrinthine artery, a terminal branch of the AICA) — a posterior circulation stroke pattern — rather than peripheral neuritis.

In HINTS-Plus, the presence of acute unilateral hearing loss in AVS shifts the result toward "central (stroke)" even if the HINTS triad itself is "peripheral-appearing." This modification increases sensitivity slightly at a modest cost to specificity, and is particularly valuable when bHIT is positive (which would otherwise reassure).

> **Clinical Pearl**
> *Acute hearing loss + acute vertigo + positive bHIT ≠ simple vestibular neuritis. The combination raises the possibility of AICA territory infarct affecting both the anterior inferior cerebellar artery branches to the cochlea and vestibular nerve. Obtain urgent MRI with DWI and posterior fossa sequences.*

V. Positional Testing — Dix-Hallpike and Supine Roll Test

Positional testing is the definitive diagnostic manoeuvre for benign paroxysmal positional vertigo (BPPV) — the most common peripheral vestibular disorder, accounting for 20–30% of all dizzy patients in specialised clinics. Accurate identification of the affected canal is essential, as each canal subtype requires a specific repositioning manoeuvre.

V.A — Dix-Hallpike: Technique and Posterior Canal BPPV

The Dix-Hallpike manoeuvre, described by Dix and Hallpike in 1952 and refined since, tests for posterior semicircular canal (PSC) BPPV — the most common subtype (~85–90% of cases).

Technique:

- **Step 1:** Patient sits upright on the examination table, head turned 45° toward the ear being tested. This aligns the posterior canal with the plane of head rotation.
- **Step 2:** Patient is quickly lowered to supine with the head hanging approximately 20–30° below horizontal, maintaining the 45° turn. The examiner supports the head throughout.
- **Step 3:** Observe the eyes for nystagmus for at least 30 seconds. Look for latency, direction, and fatigability.
- **Step 4:** Return patient to sitting and observe for reversal of nystagmus (brief nystagmus in the opposite direction on rising is confirmatory).

Positive test for posterior canal BPPV:

- Latency of 3–30 seconds before nystagmus onset (canalith settling time)
- Geotropic nystagmus: upbeat with a torsional component, fast phase toward the ground (toward the affected — downward — ear)
- Duration typically 10–45 seconds, then resolves
- Reversal nystagmus on sitting up (brief, reversed direction)
- Fatigability with repeated testing

> **Clinical Pearl**
> *If the nystagmus in the Dix-Hallpike position is direction-changing, purely vertical (downbeat or upbeat without torsion), lacks latency, is non-fatigable, or lasts more than one minute — consider central positional nystagmus and pursue neuroimaging before presuming BPPV. Central positional nystagmus can mimic BPPV convincingly but requires MRI for exclusion.*

V.B — Supine Roll Test: Lateral Canal BPPV

Lateral semicircular canal (LSC) BPPV is the second most common subtype (~10–15% of BPPV). It is identified by the Supine Roll Test (also called the Roll Test or Bárány Roll Test):

- **Position:** Patient supine, head elevated ~30° (flexed slightly to bring lateral canal into horizontal plane — its plane of maximal sensitivity)
- **Manoeuvre:** Head is rotated briskly 90° to the right (keeping patient supine), observed for 60 seconds, then returned to centre, then rotated 90° to the left and observed again

Two patterns of lateral canal BPPV:

- **Geotropic (canalolithiasis):** Horizontal nystagmus beating toward the ground (toward the ear facing down) on both sides. Nystagmus is stronger on the affected side. Typically brisk, short-lived (<60 seconds). The side with the stronger nystagmus is the affected ear.

- **Apogeotropic (cupulolithiasis):** Horizontal nystagmus beating away from the ground on both sides. The side with the weaker nystagmus is typically the affected ear (inverse rule). Nystagmus is longer-lasting, more persistent, and less likely to fatigue.

> **Clinical Insight**
> *Lateral canal BPPV can be more disabling than posterior canal BPPV due to the large amplitude of nystagmus and the horizontal plane of symptom provocation (head turning in bed). Geotropic lateral canal BPPV is treated with the Barbecue Roll (Lempert) manoeuvre; apogeotropic requires the Gufoni manoeuvre or forced prolonged positioning. Understanding which subtype is present determines treatment.*

V.C — Modified Dix-Hallpike and Straight Head-Hanging

When standard Dix-Hallpike is negative or borderline, two variations may improve diagnostic yield:

- **Modified Dix-Hallpike (Kim & colleagues):** Performed with the head turned 45° but with a smaller angle of hanging — useful in patients with limited neck extension or who cannot tolerate full hyperextension.
- **Straight head-hanging test:** Head is lowered to hanging position without lateral rotation. This tests the anterior (superior) semicircular canals, which lie nearly in the sagittal plane. Positive result = upbeat-torsional nystagmus. Anterior canal BPPV is rare (<5% of BPPV), and the same nystagmus can arise from central pathology — interpret with caution.

V.D — Differentiating Peripheral from Central Positional Nystagmus

Feature	Peripheral (BPPV)	Central Positional Nystagmus
Latency	3–30 seconds (typical)	Absent or < 3 seconds
Duration	< 60 seconds (usually < 45s)	> 60 seconds or continuous
Direction	Direction-fixed, geotropic torsional (PSC)	Direction-changing or pure vertical
Fatigability	Yes — diminishes with repeated testing	No — persists or increases
Torsional component	Present in PSC BPPV	Often absent (pure upbeat/downbeat)
Associated signs	None — isolated to the manoeuvre	May have smooth pursuit, saccade, or VOR abnormalities

Table V.1 — Differentiating peripheral from central positional nystagmus. Any feature suggesting centrality warrants MRI.

VI. Head Shaking Nystagmus and Vibration-Induced Nystagmus

Head Shaking Nystagmus (HSN)

Head Shaking Nystagmus is a provocation test for asymmetric vestibular function. It exploits the velocity storage mechanism — the neural integrator that prolongs the VOR time constant beyond the mechanical time constant of the semicircular canal cupula — to reveal latent vestibular tone asymmetry that may not produce spontaneous nystagmus at rest.

Technique:

- Patient wears Frenzel goggles (or video goggles) to eliminate fixation suppression
- Examiner oscillates the patient's head in the horizontal plane at 2 Hz for 20 complete cycles, through ~30° of arc (±15°)
- Immediately after stopping, observe the eyes in primary gaze for nystagmus
- Record direction of fast phase, intensity, and duration

Interpretation:

- **Contralateral fast phase (normal):** HSN beats away from the side of unilateral hypofunction. After shaking, the velocity storage asymmetry discharges as a nystagmus whose fast phase is toward the intact side. Confirms peripheral unilateral vestibular hypofunction.
- **No nystagmus:** Either symmetric function or full central compensation — a negative HSN does not exclude unilateral hypofunction, particularly in chronic lesions.
- **Perverted HSN (vertical nystagmus after horizontal shaking):** A red flag for central pathology. Horizontal shaking should produce horizontal nystagmus if anything — vertical HSN indicates abnormal cross-axis vestibular projections, typically in cerebellar or brainstem disease (floccular lesions, Chiari malformation).

> **Clinical Insight**
> *HSN is most sensitive in the subacute phase (days to weeks) of unilateral vestibular hypofunction, before central compensation eliminates the velocity storage asymmetry. In the first 48 hours, spontaneous nystagmus typically dominates; in the chronic phase (months), even HSN may be negative despite persistent vHIT asymmetry. Use HSN alongside bHIT and VIN for a comprehensive asymmetry assessment.*

Vibration-Induced Nystagmus (VIN)

Vibration-Induced Nystagmus is an elegant provocation test that exploits the differential sensitivity of the vestibular otolithic organs (utricle and saccule) to bone-conducted vibration. It can lateralise unilateral vestibular hypofunction and confirm the vestibular origin of asymmetry even when spontaneous and head-shaking nystagmus have resolved.

Technique:

- A 100 Hz vibrator (or 100 Hz tuning fork applied perpendicular to the skin) is applied to the mastoid process on each side sequentially
- Application duration: 10–15 seconds per side, with patient in primary gaze
- Use Frenzel or video goggles to observe nystagmus direction and velocity

Interpretation:

- **Nystagmus beats toward the intact side:** Indicates unilateral hypofunction on the opposite (ipsilateral to vibrator placement) side. Both mastoids typically produce nystagmus in the same

direction — toward the intact side — because the asymmetric otolithic input predominates regardless of which side is vibrated.

- **No nystagmus:** Symmetric function or full compensation — common in compensated unilateral hypofunction
- **Different directions from each side:** Atypical — consider bilateral hypofunction or central pathology

VIN has the clinical advantage of detecting asymmetric vestibular function long after spontaneous nystagmus has resolved. It may be positive months to years after acute vestibular neuritis, confirming the chronic unilateral hypofunction and providing objective lateralisation at any follow-up visit.

> **Key Point**
> *VIN is particularly valuable in the assessment of chronic dizziness where spontaneous nystagmus is absent: a positive VIN (nystagmus consistently toward the same side from both mastoids) confirms ongoing vestibular asymmetry and points to the affected ear — guiding vestibular rehabilitation direction and monitoring recovery.*

VII. Ocular Motor Examination

Systematic examination of ocular motor function is essential in any vestibular assessment — particularly when central pathology is suspected. The ocular motor examination reveals the integrity of the brainstem, cerebellum, and their connecting pathways, and abnormalities here are among the most reliable localising signs in all of neurology.

VII.A — Smooth Pursuit and Saccades

Smooth Pursuit: The smooth pursuit system tracks slowly moving targets, maintaining foveal fixation. It is mediated by the cortex (parieto-occipital cortex, frontal eye fields), pontine nuclei, and cerebellum (flocculus). Bedside assessment involves asking the patient to follow a slowly moving finger or target at approximately 20–30°/s.

Normal smooth pursuit is smooth and continuous. Abnormalities include:

- **Saccadic pursuit (catch-up saccades):** The eye cannot maintain velocity matching, falling behind and correcting with small saccades. May be age-related, a drug effect (anticonvulsants, benzodiazepines), or indicate cerebellar or brainstem pathology.
- **Asymmetric pursuit (ipsilateral pursuit worse):** Impaired pursuit toward the side of a cortical or cerebellar lesion. Lateralising sign.

Saccades: Rapid ballistic eye movements that redirect gaze to a new target. Assessed by asking the patient to look back and forth between two fixed targets.

Parameters to assess:

- **Latency:** Time from target appearance to saccade initiation (normal ~200 ms). Prolonged in brainstem disease, basal ganglia disorders, or cerebellar degeneration
- **Accuracy:** Does the eye land on target (normoaccurate), overshoot (hypermetric), or undershoot (hypometric)?
- **Velocity:** Normal saccades are very fast (peak velocity >400°/s for 20° saccades). Slow saccades indicate pontine disease (PPRF) or neuromuscular junction disorders

> **Clinical Insight**
> *Bilateral hypermetric saccades (consistent overshooting in both directions) are a reliable sign of cerebellar dysfunction, particularly involving the posterior vermis and fastigial nucleus. This finding in a dizzy patient shifts localisation firmly to the cerebellum and should prompt MRI with attention to posterior fossa structures.*

VII.B — Skew Deviation and the Ocular Tilt Reaction

Skew deviation is a vertical misalignment of the eyes — one eye is higher than the other — caused by asymmetric otolithic input to the vertical eye motor pathways. It is the "Test of Skew" component of the HINTS examination and is assessed by the alternate cover test.

Alternate Cover Test technique:

- Patient fixates a distant target in primary gaze
- Examiner alternately covers each eye while observing the uncovered eye for refixation movements
- **Positive skew:** Vertical refixation movement of the uncovered eye — one eye was resting higher, the other lower, and both refixate vertically when each is uncovered

Skew deviation indicates asymmetric graviceptive otolithic input, typically from:

- Brainstem lesions (lateral medullary, pontine, or midbrain)
- Cerebellar lesions (nodulus, uvula)

- It can rarely occur in peripheral vestibular disease but is almost always transient and of small magnitude

The Ocular Tilt Reaction (OTR) is the full syndrome of skew deviation + head tilt + cyclotorsion of the eyes (counter-rolling). It represents the output of an asymmetric graviceptive pathway and localises to the utriculo-ocular pathway. It can be ipsiversive (toward the lesion — lateral medullary infarct) or contraversive (away from the lesion — pontine or midbrain). The OTR is a highly specific central sign.

> **Clinical Pearl**
> *Any skew deviation detectable on alternate cover test in the setting of acute dizziness is a HINTS stroke signal and mandates urgent MRI. Do not dismiss a small but reproducible vertical misalignment — even 1–2 mm of vertical refixation constitutes a positive skew if it is consistent and reproducible.*

VII.C — VOR Suppression and Gaze-Holding

VOR suppression testing assesses the ability of smooth pursuit to cancel the VOR during head movement while tracking a target moving with the head — a function critical for reading while walking. The patient holds both arms extended with thumbs raised, and is asked to focus on their thumb while they rotate both their head and thumb together at 1–2 Hz.

Normally, the eyes remain fixed on the target (VOR is suppressed by pursuit). Failed suppression — eyes drifting off target with corrective saccades visible — indicates cerebellar (floccular) pathology.

Gaze-holding: The neural integrator maintains eccentric gaze position against elastic restoring forces. Failure produces gaze-evoked nystagmus — the eye drifts back toward primary position, generating a centripetal slow phase and a corrective fast phase in the direction of gaze. Cerebellar gaze-evoked nystagmus is typically bidirectional (beats right on right gaze, left on left gaze), while brainstem gaze-evoked nystagmus may be unidirectional. Failure of gaze-holding is always central.

VIII. Cerebellar Examination at the Bedside

Cerebellar testing is not an optional add-on in the vestibular examination — it is a mandatory component of every assessment. The cerebellum is anatomically and functionally inseparable from the vestibular system: the vestibulocerebellum (flocculonodular lobe) directly modulates VOR gain, smooth pursuit, gaze-holding, and vestibulospinal reflexes. Posterior fossa pathology — including cerebellar infarct, haemorrhage, demyelination, and degeneration — can present with dizziness and imbalance that is clinically indistinguishable from peripheral vestibulopathy unless the cerebellar examination is performed systematically.

> **Key Point**
> *Cerebellar testing should be performed routinely in every dizzy patient — not reserved for those already suspected of central pathology.*

VIII.A — Indications: Routine vs. Targeted Examination

Routine (all dizzy patients):

- Finger-nose-finger test and rapid alternating movements (dysdiadochokinesia)
- Heel-shin test
- Gaze-evoked nystagmus in all positions
- Tandem gait (assessed in Section XII — Gait and Balance)

Enhanced cerebellar screen (targeted — any of the following triggers):

- Direction-changing or purely vertical nystagmus on bedside examination
- Abnormal smooth pursuit or hypermetric saccades
- Gait severely disproportionate to VOR findings
- Acute vestibular syndrome with negative bHIT
- Associated headache, diplopia, dysarthria, dysphagia, or facial numbness
- Prominent imbalance in the absence of significant dizziness
- Older patient (>50) with new dizziness and vascular risk factors
- Suspected demyelinating disease, hereditary ataxia, or paraneoplastic syndrome

VIII.B — Oculomotor Cerebellar Signs

The most sensitive cerebellar signs are oculomotor and are assessed during the standard vestibular examination. These are covered in detail in Section VII but are listed here as a clinical reminder:

- **Gaze-evoked nystagmus:** Direction-changing horizontal or omnidirectional — cerebellar gaze-holding failure
- **Downbeat nystagmus:** Fast phase downward in primary gaze or on lateral gaze — flocculus, nodulus, or craniocervical junction
- **Rebound nystagmus:** Nystagmus in primary gaze after sustained eccentric gaze, beating in the direction of the return — specific for cerebellar disease
- **Hypermetric saccades:** Overshooting the target bilaterally — posterior vermis and fastigial nucleus
- **Saccadic (cogwheel) smooth pursuit:** Loss of smooth tracking — cerebellar or cortical, age-independent when severe and bilateral
- **Impaired VOR suppression:** Eyes cannot hold fixation on target moving with head — floccular dysfunction

Clinical Insight
Rebound nystagmus is highly specific for cerebellar pathology and is elicited by asking the patient to hold eccentric gaze (e.g., 30° right) for 20 seconds, then return to primary gaze. If a brief burst of nystagmus appears in primary gaze beating in the opposite direction (e.g., left-beating after rightward gaze), this is rebound nystagmus — a cerebellar sign not seen in peripheral vestibulopathy.

VIII.C — Limb Coordination Tests

Finger-Nose-Finger (FNF) Test:

The patient alternately touches the examiner's outstretched finger and their own nose, with the examiner periodically moving their finger to a new position. Assess for:

- **Dysmetria:** Overshooting or undershooting the target — action tremor increasing near the target (intention tremor)
- **Past-pointing:** Consistently missing to one side — lateralising sign toward the ipsilateral cerebellar hemisphere
- **Intention tremor:** Tremor amplitude increasing as the target is approached — contrasted with resting tremor of basal ganglia disease which diminishes on movement

Heel-Shin Test:

Patient supine, places their heel on the opposite knee and slides the heel smoothly down the shin to the ankle, then returns. Assess for:

- **Ataxia:** Irregular, jerky, or oscillating heel movement along the shin — cerebellar coordination failure
- **Decomposition of movement:** The movement is broken into sequential segments rather than smooth arcs

Dysdiadochokinesia — Rapid Alternating Movements:

Patient rapidly alternates between pronation and supination of the forearm (patting the knee alternately with palm and dorsum). Assess for:

- **Adiadochokinesia / dysdiadochokinesia:** Irregular rate, rhythm, and amplitude of alternating movements — cerebellar hemisphere dysfunction ipsilateral to the affected limb

Rebound Test (Stewart-Holmes Sign):

The patient holds their arm flexed at the elbow against the examiner's resistance. The examiner suddenly releases resistance. Normally, a rapid check (antagonist braking) prevents excessive rebound. In cerebellar disease, the arm rebounds excessively — overshooting due to failure of the check reflex. Perform with adequate clearance to avoid patient self-injury.

VIII.D — Speech

Cerebellar dysarthria is characterised by scanning speech — monotonous, irregular cadence, with syllables of equal stress separated by irregular pauses. It reflects failure of cerebellar coordination of the laryngeal, palatal, and articulatory muscles. Assess by asking the patient to say "British constitution" or "baby hippopotamus" and observing for explosive, slurred, or scanning quality.

Clinical Pearl
Unilateral limb ataxia (ipsilateral finger-nose dysmetria, ipsilateral past-pointing) combined with contralateral limb weakness, ipsilateral Horner syndrome, and ipsilateral facial numbness constitutes the lateral medullary syndrome (Wallenberg syndrome) — the most frequently missed posterior fossa stroke presenting as dizziness. The combination of nausea, severe imbalance, ipsilateral facial and contralateral limb sensory loss, dysphagia, and hoarseness should always prompt emergency MRI.

Cerebellar Sign	Test	Localisation
Gaze-evoked nystagmus	9-position gaze exam	Flocculus / gaze-holding integrator
Downbeat nystagmus	Primary gaze + lateral gaze	Flocculus, nodulus, craniocervical junction
Rebound nystagmus	Eccentric gaze then return	Cerebellar disease — specific
Hypermetric saccades	Horizontal saccade test	Posterior vermis, fastigial nucleus
Saccadic pursuit	Smooth pursuit tracking	Cerebellar / cortical (non-specific)
Finger-nose dysmetria	FNF test	Ipsilateral cerebellar hemisphere
Dysdiadochokinesia	Rapid alternating movements	Ipsilateral cerebellar hemisphere
Rebound test positive	Stewart-Holmes	Cerebellar (non-lateralising)
Scanning dysarthria	Speech assessment	Cerebellar vermis / bilateral
Truncal ataxia	Unsupported sitting / standing	Vermis, vestibulocerebellum

Table VIII.1 — Cerebellar signs, bedside tests, and anatomical localisation. All bold-text signs are routine in every dizzy patient.

IX. Dynamic Visual Acuity (DVA)

Dynamic Visual Acuity tests the ability of the VOR to maintain stable retinal images during head movement. It provides a functional measure of VOR performance directly relevant to a patient's lived experience — their ability to read signs, recognise faces, or navigate safely during locomotion.

Technique

The standard bedside DVA test uses a Snellen acuity chart:

- **Step 1 — Static acuity:** Record best corrected visual acuity with the head stationary. This is the baseline.
- **Step 2 — Dynamic acuity:** Patient oscillates the head horizontally at 1–2 Hz (or the examiner passively oscillates at a similar rate) while reading the smallest line possible on the Snellen chart.
- **Result:** Record the number of lines lost compared to static acuity.

Normal: Loss of ≤1 line of acuity during head movement. The intact VOR generates eye velocity closely matching head velocity, maintaining retinal image stability.

Abnormal: Loss of ≥2 lines indicates clinically significant VOR failure, implying bilateral vestibular hypofunction or, less commonly, severe unilateral hypofunction without adequate compensation.

> **Key Point**
> *DVA loss of ≥3 lines is highly suggestive of bilateral vestibular hypofunction (BVH). DVA should be performed in all patients complaining of oscillopsia (visual instability during head movement) or imbalance worsening in the dark, as these symptoms are the functional signature of bilateral VOR failure.*

Clinical Significance

DVA abnormality has direct implications for:

- **Bilateral vestibular hypofunction (BVH):** The single most consistent bedside finding — bilateral VOR failure causes severe DVA loss, oscillopsia, and characteristic Romberg deterioration in darkness
- **Monitoring vestibular compensation:** DVA normalises as central compensation restores effective VOR gain — useful as a serial outcome measure
- **Driving and occupation:** Abnormal DVA predicts difficulty with tasks requiring stable vision during head movement — medico-legal relevance
- **Vestibular rehabilitation efficacy:** Gaze stabilisation exercises specifically target DVA improvement by promoting VOR adaptation and substitution strategies

A computerised DVA test (e.g., Bertec Balance Advantage, NeuroCom) provides quantitative measurement with standardised head velocity input, improving sensitivity and reproducibility compared to bedside Snellen-based methods. However, the bedside test remains clinically useful for screening and follow-up in settings without access to formal VFT.

> **Clinical Pearl**
> *In patients with complaints of visual blurring when turning the head, or instability exclusively in the dark or on uneven ground, test DVA before ordering vHIT. A dramatically abnormal DVA with bilateral positive bHIT virtually confirms BVH and should prompt referral for formal vHIT, caloric testing, and assessment for the underlying cause (ototoxicity, meningitis, autoimmune, genetic).*

X. Otoscopic, Otologic, and Audiometric Assessment

The ear examination is an essential component of the vestibular assessment. The cochlear and vestibular systems share the same labyrinth, blood supply, and inner ear fluid compartment — otoscopic and audiometric findings frequently provide critical diagnostic information that completes or refines the vestibular picture.

Otoscopic Examination

Every patient presenting with vestibular symptoms should have otoscopic examination of both ears. Key findings to seek:

- **Tympanic membrane appearance:** Assess landmarks (cone of light, handle of malleus, pars tensa and pars flaccida), colour, and translucency. A normal tympanic membrane is translucent, grey, with a light reflex at the antero-inferior quadrant.
- **Cholesteatoma:** A white, pearly keratinous mass, typically in the posterosuperior quadrant or pars flaccida. Often associated with retraction pockets, chronic discharge, and conductive hearing loss. Critical finding: cholesteatoma eroding into the labyrinth causes a labyrinthine fistula, presenting with pressure-induced vertigo (Hennebert sign) and potential acute vertigo. Mandates urgent ENT/otological referral and CT imaging.
- **Haemotympanum:** Blood behind an intact tympanum (blue-grey discolouration). In the context of head trauma with vertigo, indicates temporal bone fracture or inner ear barotrauma. Requires CT temporal bones.
- **Tympanic membrane perforation:** May indicate past or current otitis media with potential labyrinthine involvement, or post-traumatic injury with associated perilymphatic fistula.
- **Middle ear effusion:** Reduced light reflex, amber or yellow colour, air-fluid level, or bubbles behind the tympanum. Associated with conductive hearing loss and may affect tympanometric findings — relevant when interpreting auditory tests.

Fistula Test

When a labyrinthine fistula is suspected (cholesteatoma, perilymphatic fistula from trauma or iatrogenic injury), a fistula test (Hennebert test) should be performed:

- Apply gentle positive then negative pressure to the external canal using a pneumatic otoscope or by pressing the tragus inward
- Observe for vertigo, nausea, or nystagmus provoked by the pressure change
- **Positive fistula test:** Vertigo and/or nystagmus provoked — indicates an abnormal communication between the middle ear and inner ear. Mandates further investigation and ENT referral.

> **Clinical Pearl**
> *Tullio phenomenon (vertigo or nystagmus provoked by loud sounds, Valsalva, or pressure) is a related finding suggesting either a labyrinthine fistula or Superior Semicircular Canal Dehiscence (SSCD). In SSCD, the third window effect produces pressure sensitivity. Always ask about sound-induced dizziness and consider high-resolution CT temporal bones if suspected.*

Tuning Fork Tests: Weber and Rinne in Vestibular Context

Tuning fork tests rapidly screen for hearing asymmetry and help direct audiometric evaluation:

Weber test (512 Hz placed on vertex or mid-forehead):

- **Normal:** Sound perceived in midline or equally in both ears
- **Lateralises to the affected ear:** Unilateral conductive hearing loss — the conductive block reduces ambient air-conducted masking noise reaching the affected ear, making bone-conducted sound appear louder on that side
- **Lateralises to the better (unaffected) ear:** Unilateral sensorineural hearing loss — the damaged cochlea cannot process the bone-conducted signal, so the intact cochlea perceives it as louder

Rinne test (512 Hz):

- **AC > BC (Rinne positive):** Normal, or sensorineural hearing loss pattern
- **BC > AC (Rinne negative):** Conductive hearing loss on the tested side

Vestibular-audiological correlation:

Audiological Pattern	Vestibular Correlation
Sudden unilateral SNHL + acute vertigo	Labyrinthine infarct (AICA), labyrinthitis, perilymphatic fistula, autoimmune
Fluctuating low-frequency SNHL + episodic vertigo	Ménière's disease — endolymphatic hydrops
Progressive bilateral SNHL + imbalance	Bilateral vestibular hypofunction — ototoxicity, autoimmune, genetic, bilateral Ménière's
Asymmetric SNHL (high-frequency) + chronic imbalance	Acoustic neuroma (vestibular schwannoma) — urgent MRI IAMs
Conductive HL + vertigo + chronic discharge	Cholesteatoma with labyrinthine erosion — urgent ENT referral

Table IX.1 — Audiological patterns and their vestibular correlates. Weber and Rinne guide initial lateralisation; formal audiometry is required for characterisation.

□ Key Point
When Weber lateralises, audiometry is mandatory — tuning forks screen but cannot quantify the pattern or degree of loss. A purely low-frequency SNHL asymmetry (characteristic of Ménière's) will be missed by a 512 Hz Weber test, as it tests predominantly mid-frequency hearing. Always arrange audiometry when there is any suspicion of asymmetric sensorineural loss.

XI. Cardiovascular and Autonomic Assessment

Cardiovascular and autonomic disorders are a frequently underdiagnosed cause of dizziness, accounting for 15–25% of dizzy patients in general practice. Orthostatic hypotension (OH), postural tachycardia syndrome (POTS), vasovagal syncope, and cardiac arrhythmias can all produce dizziness that superficially resembles vestibular disorder — particularly the "presyncope" quality (lightheadedness, near-faintness, "swimming head" sensation).

The systematic cardiovascular examination distinguishes these disorders from true vestibular pathology and is particularly important because their management is entirely different from vestibular rehabilitation.

Orthostatic Hypotension (OH) — Measurement Protocol

OH is defined as a sustained drop in systolic blood pressure of ≥20 mmHg or diastolic ≥10 mmHg within three minutes of standing (or head-up tilt to 60°), in association with symptoms. Measurement technique is critical — an abbreviated or incorrectly timed protocol produces false negatives.

Active Standing Test — Step-by-Step Protocol:

- **Step 1 — Rest:** Patient lies supine for a minimum of 5 minutes (10 minutes preferred in patients with suspected autonomic failure). Ensure room is quiet and patient is comfortable.
- **Step 2 — Supine baseline:** Measure BP and HR with an automated cuff on the non-dominant arm. Record two readings 1 minute apart; use the lower value as baseline.
- **Step 3 — Stand:** Patient stands actively and immediately (not gradual). Start a timer at the moment of standing. Avoid patient gripping or supporting themselves on arms — this activates the muscle pump and masks OH.
- **Step 4 — Measure at 1 minute:** BP and HR. Note whether symptoms are reproduced at this point.
- **Step 5 — Measure at 3 minutes:** BP and HR. This is the primary diagnostic timepoint per consensus definition (Freeman et al., 2011).
- **Step 6 — Measure at 5 minutes (recommended):** Detects delayed OH — a BP drop occurring after 3 minutes, which represents mild autonomic dysfunction often missed by the standard protocol. Particularly important in patients with prolonged postural symptoms.
- **Step 7 — Record symptoms:** Document presence and timing of dizziness, presyncope, visual changes, or headache at each timepoint. Asymptomatic BP drops are less clinically significant than symptomatic ones.

Timepoint	Measure	Diagnostic Threshold	Interpretation
Supine (baseline)	BP + HR	—	Record reference values
1 min standing	BP + HR	SBP drop ≥ 20 or DBP drop ≥ 10 mmHg	Classical OH if symptomatic; Initial OH transient < 30s
3 min standing	BP + HR	SBP drop ≥ 20 or DBP drop ≥ 10 mmHg	Classical OH — primary diagnostic timepoint
5 min standing	BP + HR	SBP drop ≥ 20 or DBP drop ≥ 10 mmHg	Delayed OH — early autonomic dysfunction
HR change (all)	**HR increase**	**Rise ≥ 30 bpm (adult); ≥ 40 bpm (age 12–19)**	POTS if without OH and with symptoms

Table XI.1 — Active Standing Test protocol. SBP = systolic BP; DBP = diastolic BP. HR change assessed across the full 5-minute period.

Clinical Pearl
Document the exact BP figures at each timepoint — not just "normal" or "abnormal." A drop of 18/9 mmHg with clear presyncope is clinically significant even if it misses the numerical threshold. Equally, a drop of 25 mmHg without any symptoms in an elderly patient may not require intervention. Symptoms determine clinical significance.

Subtypes of OH:

- **Classical OH:** BP drop within 3 minutes — autonomic failure pattern (Parkinson's disease, MSA, pure autonomic failure, diabetic autonomic neuropathy, medications)
- **Delayed OH:** BP drop after 3 minutes — milder autonomic dysfunction or early failure; often missed without 5-minute measurement
- **Initial OH:** Transient BP drop within 15 seconds of standing, recovering within 30 seconds — requires beat-to-beat monitoring (sphygmomanometer protocol misses this); suspect in young patients with brief presyncope on rapid rising

Clinical Insight
Medications are the most common reversible cause of OH in older patients — antihypertensives, diuretics, alpha-blockers, tricyclics, and dopamine agonists are frequent culprits. A comprehensive medication review is mandatory before attributing postural dizziness to primary autonomic dysfunction.

Postural Tachycardia Syndrome (POTS)

POTS is defined as a heart rate increase of ≥30 bpm (or ≥40 bpm in adolescents aged 12–19) within 10 minutes of standing, in the absence of OH, with associated orthostatic symptoms.

POTS frequently presents to vestibular clinics because patients describe dizziness, lightheadedness, cognitive slowing ("brain fog"), and palpitations — symptoms that overlap with vestibular dysfunction. The key discriminating feature is the relationship to posture: symptoms reproduce consistently on standing and resolve with sitting or lying.

Clinical clues favouring POTS over primary vestibular disorder:

- Symptoms consistently worse on standing, relieved by lying flat
- Associated palpitations or awareness of heartbeat
- Cognitive "fogginess," fatigue, or exercise intolerance
- Often affects young women aged 15–45
- Absence of nystagmus and normal HINTS examination
- Normal vestibular function testing

Key Point
The simple 10-minute Active Standing Test costs nothing and takes 10 minutes. It identifies OH and POTS — two common, treatable causes of dizziness — that are frequently missed when the history is interpreted as "vestibular." Measure BP and HR at 1, 3, and 5 minutes in every dizzy patient with postural or exertional symptoms.

Cardiac Causes and Vascular Assessment

Arrhythmia-related dizziness (typically paroxysmal, reproducible, associated with palpitations or syncope) requires 12-lead ECG in the initial assessment and Holter monitoring or event recording if the ECG is unremarkable. Key ECG findings relevant to vestibular medicine:

- **Long QTc syndrome:** Risk of torsades de pointes — episodic presyncope or syncope often mistaken for vestibular attacks
- **Sick sinus syndrome / second-degree AV block:** Paroxysmal bradycardia producing presyncope
- **Paroxysmal atrial fibrillation:** May produce embolic vestibular events (AICA territory TIA) in addition to haemodynamic presyncope

Carotid sinus hypersensitivity — exaggerated response to carotid pressure causing bradycardia and/or hypotension — can be identified by carotid sinus massage in a controlled clinical environment and is a consideration in older patients with dizziness provoked by head turning or neck pressure.

> **Clinical Pearl**
> *Vertebrobasilar insufficiency (VBI) is over-diagnosed and rare. True VBI dizziness almost always accompanies other brainstem symptoms (diplopia, dysarthria, dysphagia, Horner syndrome, facial numbness) and lasts minutes, not seconds. Brief dizziness provoked by head turning in isolation is far more likely to be BPPV, orthostatic hypotension, or anxiety than VBI. Reserve vascular imaging for those with additional neurological features.*

XII. Gait and Balance Assessment — An Overview

Gait and balance testing completes the bedside vestibular examination, providing a global functional measure of how the patient's vestibular, cerebellar, proprioceptive, and visual systems integrate in real-world locomotion. Abnormalities here may be the only objective finding in patients with early bilateral vestibulopathy, early cerebellar degeneration, or functional dizziness.

> **Key Point**
> *A detailed, evidence-based review of gait and balance assessment — including computerised dynamic posturography, dynamic gait index, the Fukuda stepping test, and quantitative Romberg variants — is provided in the next chapter: Examination of Balance and Gait. This section provides a focused bedside overview for clinical integration.*

XII.A — Romberg Test and Modified Romberg

The Romberg test assesses postural stability by systematically removing visual input, revealing reliance on vestibular and proprioceptive information:

- **Standard Romberg:** Patient stands with feet together, arms crossed, eyes open then closed. Time in each condition (30-second target). Normal: stable with eyes open and closed.
- **Positive Romberg:** Patient stable with eyes open but sways or falls with eyes closed — indicates the patient is over-relying on vision to maintain balance, consistent with vestibular or proprioceptive dysfunction.
- **Romberg failure with eyes open:** Suggests cerebellar ataxia — visual substitution is itself insufficient. This is not a standard Romberg positive; it indicates more severe coordination failure.
- **Foam Romberg (Romberg on foam surface):** Patient stands on compliant foam with eyes open then closed. Removes proprioceptive contribution — isolates vestibular input. Bilateral vestibulopathy produces significant instability on foam with eyes closed that exceeds what would be expected for age.

XII.B — Fukuda Stepping Test

The patient marches on the spot with eyes closed, arms outstretched, for 50 steps. Normal: rotation and drift of less than 30°. In unilateral vestibular hypofunction, patients rotate toward the affected side — the intact vestibular system drives the contralateral stepping pattern. This test is useful for confirming lateralisation but has modest sensitivity and specificity when used in isolation.

XII.C — Tandem Gait

Patient walks heel-to-toe along a line for 10 steps. Assess for sway, stepping off the line, or requiring arm extension for balance. Tandem gait is highly sensitive to posterior vermis dysfunction and bilateral vestibulopathy. Inability to tandem walk in a patient who is otherwise stable at rest suggests disproportionate dynamic postural impairment — a red flag for central pathology.

XII.D — Observation of Spontaneous Gait

Clinical observation of natural gait should precede any formal test. Vestibular gait is characteristically wide-based, with lateral deviation or veering toward the affected side. Cerebellar gait is ataxic — irregular, broad-based, and trunk-swaying, often worsening on tandem walking and turning. Propulsive or festinating gait suggests basal ganglia disease. High-stepping gait (foot drop) indicates peripheral neuropathy affecting the lower limb.

Ask the patient to turn rapidly through 180° — inability to turn smoothly in fewer than four steps (turn test) is a sensitive indicator of postural instability.

Clinical Insight
In patients with bilateral vestibulopathy, spontaneous gait in adequate lighting may appear almost normal — the visual and proprioceptive systems largely compensate in bright, even-surface environments. The deficit emerges most clearly in the dark, on uneven surfaces, or on foam with eyes closed. Always test under reduced-input conditions before concluding gait is intact.

Test	Technique	Key Finding	Interpretation
Romberg	Feet together, eyes open then closed	Sway or fall on eyes closed	Vestibular or proprioceptive loss
Foam Romberg	Romberg on compliant foam	Instability with eyes closed > expectation for age	Bilateral vestibulopathy
Fukuda stepping	50 steps eyes closed; measure rotation	Rotation > 30° toward affected side	Unilateral vestibular hypofunction
Tandem gait	Heel-to-toe, 10 steps	Falls off line, requires arm support	Posterior vermis, bilateral vestibulopathy
Spontaneous gait	Natural walking observed	Wide base, veering, ataxic, festinating	Pattern suggests aetiology
Turn test	Turn 180° — count steps	> 4 steps or unsteady	Postural instability

Table XII.1 — Bedside gait and balance assessment. Detailed analysis including posturography and dynamic gait index is covered in the next chapter.

Clinical Pearl
Never skip gait observation because a patient describes their main problem as "dizziness" rather than "imbalance." Patients with significant vestibular or cerebellar dysfunction frequently underestimate or misattribute their gait impairment. Objective gait assessment may reveal a risk of falls that has medicolegal and patient safety implications requiring immediate attention.

XIII. Cervical Spine Assessment in the Dizzy Patient

Cervicogenic dizziness (CGD) is a diagnosis of exclusion — dizziness attributed to disrupted proprioceptive input from cervical mechanoreceptors (primarily C1–C3 facet joint afferents), with features that cannot be explained by a peripheral or central vestibular lesion. It remains one of the more contentious diagnoses in vestibular medicine, but it is clinically real when diagnostic criteria are met and other causes have been excluded. A systematic cervical spine assessment should be incorporated into the bedside examination of any patient whose dizziness is provoked or modulated by neck movement or position.

> **Key Point**
> *CGD is always a diagnosis of exclusion — never a first-line explanation for dizziness. The assessment sequence is: (1) exclude peripheral vestibular pathology, (2) exclude central pathology, (3) if both negative and dizziness is clearly neck-movement-related, then pursue cervical assessment. Attributing dizziness to the cervical spine prematurely is one of the most common diagnostic errors in vestibular medicine.*

XIII.A — Clinical Features Suggesting Cervicogenic Dizziness

The following clinical pattern, when present together, supports CGD as a contributing diagnosis:

- Dizziness is consistently provoked or reproduced by neck movement, sustained neck positions, or palpation of the upper cervical facet joints
- Neck pain or stiffness is present — often preceding or accompanying the dizziness
- **No auditory symptoms** — absence of hearing loss, tinnitus, or aural fullness distinguishes CGD from Ménière's and labyrinthine disorders
- **No nystagmus** at rest and no nystagmus provoked during standard Dix-Hallpike or positional testing
- Normal or near-normal vestibular function testing (vHIT, calorics)
- Normal HINTS examination — no bHIT abnormality, direction-fixed nystagmus absent, no skew deviation
- Relevant cervical history: prior trauma (whiplash), cervical spondylosis, upper cervical surgery, rheumatoid arthritis, or hypermobility syndrome

XIII.B — Cervical Range of Motion

Assess active range of motion in six planes: flexion, extension, left and right lateral flexion, left and right rotation. For each movement, record:

- **Range:** Approximate degrees of movement (normal: flexion ~45°, extension ~45°, lateral flexion ~45°, rotation ~70° bilaterally)
- **Symptom reproduction:** Does any movement reproduce the patient's dizziness? If so, which direction, and at what point in the range?
- **Pain provocation:** Neck pain or occipital headache provoked by specific movements — localises involved segments
- **End-feel and resistance:** Hard or soft restriction; protective muscle guarding

Asymmetric restriction of rotation (commonly C1–C2 in CGD) with ipsilateral dizziness provocation is a useful pointer. However, many patients with cervical spondylosis have restricted range of motion without CGD — range limitation alone is insufficient for diagnosis.

XIII.C — Smooth Pursuit Neck Torsion Test (SPNT)

The Smooth Pursuit Neck Torsion test assesses whether smooth pursuit gain changes when the trunk is rotated relative to a fixed head position — isolating the effect of cervical proprioceptive input on gaze control.

Technique:

- Patient seated; examiner assesses smooth pursuit gain (normal tracking of a target at ~20°/s) in the standard position (head aligned with trunk)
- The trunk is then rotated 45° to the right relative to the fixed head (head remains forward-facing, but neck is now in left rotation relative to the body) and smooth pursuit is re-assessed
- Repeat with trunk rotated 45° to the left

Positive SPNT: A deterioration of smooth pursuit gain (more saccadic pursuit, reduced smoothness) when the neck is in torsion compared with the neutral position — indicating that abnormal cervical proprioceptive input is interfering with oculomotor control. This is considered a positive sign for cervicogenic contribution to the dizziness.

> **Clinical Insight**
> *The SPNT was validated by Tjell and Rosenhall (1998) and further studied by Reid and colleagues. It has been shown to be positive in patients with whiplash-associated CGD and negative in vestibular and central disorders. Its specificity is improved when used in combination with joint position error testing and cervical range of motion assessment — no single test is sufficient in isolation.*

XIII.D — Joint Position Error (JPE) Testing

The Joint Position Error test quantifies cervical proprioceptive accuracy — the ability to return the head to a neutral position after active rotation, with eyes closed.

Technique:

- A small laser pointer is mounted on a headband and projected onto a target board at 90 cm distance
- Patient actively centres the laser on the target with eyes open — this marks the neutral head position
- Patient closes eyes, actively rotates the head fully left (or fully right), then attempts to return to neutral
- Distance between the neutral target and the head's actual return position is the joint position error (in centimetres or degrees)

Normal threshold: < 4.5° error in healthy adults. An error > 4.5° indicates impaired cervical proprioception and is associated with chronic neck pain, whiplash injury, and CGD. Errors > 8° are considered clinically significant.

XIII.E — Upper Cervical Palpation and Facet Joint Assessment

Palpation of the upper cervical facet joints (C0–C1, C1–C2, C2–C3) assesses for tenderness, restriction, and symptom reproduction:

- **C0–C1 (atlanto-occipital joint):** Palpate just posterior to the mandibular ramus below the mastoid. Tenderness here with occipital headache and dizziness suggests atlanto-occipital involvement.
- **C1–C2 (atlanto-axial joint):** Palpable as a soft tissue mass just below the occiput. The most common cervical segment implicated in CGD. Restricted rotation and local tenderness are key findings.

- **C2–C3:** Palpate the spinous process of C2 (first palpable bony prominence below the skull) and the adjacent facet joints bilaterally.

Reproduction of the patient's dizziness (not merely local tenderness) on sustained C1–C2 palpation or segmental pressure is the most specific provocative sign for CGD.

XIII.F — Vertebrobasilar Insufficiency Screening

Before any sustained cervical manipulation or provocative testing, consider the risk of vertebrobasilar insufficiency (VBI) — a rare but serious cause of dizziness arising from dynamic compromise of the vertebral or basilar artery during cervical movement.

Clinical red flags for VBI (the 5 D's + 3 N's):

- **Diplopia** — brainstem ocular motor involvement
- **Dysarthria** — corticobulbar or cerebellar pathway involvement
- **Dysphagia** — bulbar involvement
- **Dizziness** — posterior fossa or labyrinthine ischaemia
- **Drop attacks** — sudden loss of tone without loss of consciousness — vertebrobasilar TIA pattern
- **Nausea** (severe, acute onset)
- **Numbness** — ipsilateral face / contralateral limbs
- **Nystagmus** — new onset horizontal-torsional or vertical

True VBI dizziness almost always accompanies at least one other brainstem symptom from the list above. Isolated, brief dizziness with head turning in the absence of any other brainstem features is rarely VBI and is far more likely to be BPPV, orthostatic hypotension, or cervicogenic in origin.

> **Clinical Pearl**
> *The De Kleyn test (head rotation and extension to assess vertebral artery compression) has been largely abandoned in evidence-based practice due to insufficient sensitivity and a risk of provoking vertebral artery dissection in susceptible individuals. If genuine VBI is suspected from the history, the appropriate investigation is MRI/MRA of the posterior circulation — not provocative cervical positioning at the bedside.*

XIII.G — When to Image the Cervical Spine

Cervical spine imaging (CT or MRI) is indicated when:

- **Trauma:** Any acute dizziness following cervical trauma, particularly whiplash, falls, or head injury — exclude cervical fracture and ligamentous instability before examination
- **Craniocervical instability:** Suspected in patients with Down syndrome, rheumatoid arthritis, Ehlers-Danlos syndrome (hypermobility type), or prior cervical surgery — MRI with flexion-extension sequences
- **Myelopathy signs:** Spasticity, upper motor neuron signs, or bladder/bowel dysfunction with neck pain and dizziness — MRI cervical cord
- **Cervical artery dissection:** Acute neck pain + headache + dizziness + Horner syndrome — urgent MRI/MRA; do not delay for plain films
- **Progressive symptoms:** Worsening neurological deficit despite conservative management — exclude compressive pathology

Test	Technique	Positive Finding	Clinical Significance
Cervical ROM	Active 6-plane assessment	Dizziness reproduced by specific movement	Supports cervicogenic contribution

Test	Technique	Positive Finding	Clinical Significance
SPNT	Smooth pursuit with trunk rotation	Pursuit gain drops in torsion vs. neutral	Abnormal cervical proprioceptive input
Joint Position Error	Laser return-to-neutral, eyes closed	Error > 4.5°	Impaired cervical proprioception
C1–C2 palpation	Suboccipital facet pressure	Dizziness (not just pain) reproduced	Specific sign for C1–C2 CGD
VBI red flags screen	History — 5 D's + 3 N's	Any brainstem symptom accompanying dizziness	MRI/MRA — do not proceed with provocation

Table XIII.1 — Cervical spine bedside tests for the dizzy patient. CGD = cervicogenic dizziness; ROM = range of motion; SPNT = Smooth Pursuit Neck Torsion test.

XIV. The Structured Bedside Examination — Clinical Synthesis

The bedside vestibular examination is most powerful when its components are integrated into a coherent diagnostic narrative. Individual signs have limited sensitivity and specificity; patterns of signs provide reliable localisation and guide definitive investigation.

Clinical Patterns and Diagnostic Algorithms

Clinical Presentation	Key Bedside Findings	Likely Diagnosis	Next Step
Acute continuous vertigo + nystagmus	HINTS: +HIT, direction-fixed, no skew	Acute peripheral vestibulopathy	Supportive care, vestibular rehab, MRI at 48h if atypical
Acute continuous vertigo + nystagmus	HINTS: -HIT, direction-changing OR skew	Posterior fossa stroke	Urgent MRI DWI, stroke team
Brief positional vertigo, Dix-Hallpike +	Latency, geotropic torsional, fatigable, <1 min	Posterior canal BPPV	Epley manoeuvre immediately
Brief positional vertigo, Roll Test +	Horizontal nystagmus, geotropic or apogeotropic	Lateral canal BPPV	Barbecue roll or Gufoni manoeuvre
Oscillopsia + DVA loss ≥3 lines	Bilateral positive bHIT, reduced DVA, Romberg-	Bilateral vestibular hypofunction	vHIT, calorics, ototoxicity review, genetic
Postural dizziness on standing	BP drop ≥20 mmHg systolic on standing	Orthostatic hypotension	Medication review, autonomic evaluation
Recurrent episodic vertigo + low-freq SNHL	Weber lateralises, HSN positive, VIN positive	Ménière's disease	Audiometry, electrocochleography, MRI endolymph
Pressure-induced vertigo (Valsalva/sound)	Positive fistula test, Tullio phenomenon	Labyrinthine fistula or SSCD	CT temporal bones

Table XI.1 — Integrated bedside examination patterns and corresponding diagnostic pathways.

> **□ Key Point**
> *The bedside examination is not a screening tool — it is a diagnostic instrument. Each component should be performed systematically, with findings documented and integrated. A complete and accurately documented bedside examination provides the foundation for all subsequent investigation, prevents unnecessary neuroimaging in low-risk cases, and ensures that high-risk stroke cases are identified and escalated urgently.*

Documentation Standards

Each bedside examination should document:

- Nystagmus: direction, plane, degree of Alexander's Law, fixation suppression result
- bHIT: normal or abnormal bilaterally — which side, overt or covert saccade

- HINTS result (if AVS): HIT / nystagmus / skew — peripheral or central pattern
- Positional testing: Dix-Hallpike bilateral, Supine Roll — result with latency, direction, duration, fatigability
- Head Shaking Nystagmus: direction, duration — or negative
- VIN: direction from each mastoid — or negative
- Ocular motor: pursuit quality, saccade accuracy, gaze-holding, skew
- Cerebellar: FNF, heel-shin, dysdiadochokinesia, rebound test, gaze-evoked nystagmus, dysarthria — each recorded normal or abnormal
- DVA: static acuity and number of lines lost during head movement
- Otoscopy: both ears — normal or abnormal (describe findings)
- Weber, Rinne: lateralisation and result
- Blood pressure: supine, 1-min standing, 3-min standing, 5-min standing; HR change — OH or POTS criteria met or not
- Gait: observation, Romberg (eyes open/closed), tandem gait result, Fukuda if indicated
- Cervical spine (if indicated): range of motion, SPNT result, JPE error, upper cervical palpation findings, VBI red flags present or absent

□ Clinical Insight
In the era of electronic records and medico-legal accountability, structured bedside examination documentation is essential. A positive HINTS result that is undocumented and followed by a missed stroke is a preventable clinical and legal failure. Document "HINTS performed: HIT positive right/left, nystagmus direction-fixed/changing/vertical, skew absent/present" as a structured entry in every AVS assessment.

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