

Bedside Examination of the Dizzy Patient: A Structured, Evidence-Based Approach for General Clinicians

Vestibular Medicine for General Clinicians

Topic 2 of 14

Australian Dizziness Clinics | www.AustralianDizzinessClinics.com

Version 1.0 | April 2026

How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, emergency clinicians, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with dizziness.

The review is designed to be read in a single 20–30 minute sitting, or used as a desktop reference. It is supported by an A4 one-page cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

Callout Box Guide

- **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.
- **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.
- **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.
- **Important:** Red flags, emergencies, and critical safety points requiring immediate action.

Table of Contents

- I. Why Bedside Examination Still Matters**
- II. General Inspection and Gait**
- III. The HINTS Examination**
- IV. HINTS Plus and Audiological Screening**
- V. The Dix-Hallpike Manoeuvre**
- VI. Supine Roll Test for Lateral Canal BPPV**
- VII. Orthostatic Blood Pressure and Autonomic Assessment**
- VIII. Focused Neurological Examination**
- IX. Putting It Together — Examination Algorithm**
- X. Documentation, Pitfalls, and When to Refer**
- References**
- Disclaimer and Copyright**

I. Why Bedside Examination Still Matters

The bedside examination of the dizzy patient is one of the highest-yield clinical skills in acute medicine. In an era of advanced imaging, it remains the case that a correctly performed bedside examination outperforms early MRI for detecting posterior circulation stroke in the first 24–48 hours of symptom onset [1,2]. The Kattah HINTS paper, now cited in every major emergency medicine and neurology guideline, demonstrated that a three-component oculomotor examination had a sensitivity of 100% and specificity of 96% for central causes of acute vestibular syndrome — compared with 80% sensitivity for early diffusion-weighted MRI [1].

Despite this, bedside examination for vertigo is often omitted, truncated, or performed incorrectly in general practice and emergency settings. Surveys of emergency clinicians have found that HINTS is performed in fewer than 10% of eligible patients, and Dix-Hallpike in fewer than 25% of patients with positional symptoms [3,4]. The commonest cause is not lack of knowledge but lack of confidence — the examination looks complex and patients feel unwell.

□ Key Point:

In the dizzy patient, a structured bedside examination will change or confirm the diagnosis in more than 80% of cases — more than any single investigation you can order. Do the examination before you order the imaging, not after.

The examination is not a single protocol. It is a set of targeted tests, selected by the diagnostic category identified in the history (AVS, s-EVS, t-EVS, or chronic). This review covers the four examinations every general clinician should be competent to perform at the bedside: HINTS for acute vestibular syndrome, Dix-Hallpike and supine roll for positional vertigo, orthostatic vitals for pre-syncope and autonomic causes, and a focused neurological examination for central red flags.

II. General Inspection and Gait

Before laying a hand on the patient, observe. Much of the discriminating information is available in the first 60 seconds of the encounter.

Observation at Rest

Patients with an acute peripheral vestibular lesion (vestibular neuritis, labyrinthitis) typically lie still, eyes closed, nauseated, and visibly distressed. They will usually prefer to lie on the side of the affected ear down because head movement exacerbates symptoms. Patients with a cerebellar or brainstem stroke may look less distressed than the severity of their examination findings would suggest — a key clue that should not be overlooked.

Patients with vestibular migraine between attacks and patients with PPPD typically appear entirely well at rest. Patients with orthostatic dizziness appear well supine but may become pale, diaphoretic, or yawning on standing.

Gait Assessment

Gait is the single most sensitive bedside test for cerebellar disease and the most neglected part of the dizziness examination. Every dizzy patient who can stand should have their gait assessed. Ask the patient to walk a short distance, turn, and walk back. Then perform tandem gait (heel-to-toe walking) for at least five steps.

- **Normal gait:** consistent with peripheral vestibular lesion, orthostatic causes, or functional disorder.
- **Unilateral veering:** non-specific, may occur in unilateral peripheral vestibular lesions; veers toward the affected side.
- **Wide-based, ataxic gait:** cerebellar lesion until proven otherwise.
- **Inability to tandem walk:** highly suspicious for a central lesion in a patient with AVS.
- **Inability to stand unaided:** a red flag. Patients with isolated vestibular neuritis can almost always stand, even if they veer; inability to stand with eyes open suggests a central cause.

❑ **Important:**

A patient who cannot stand with acute continuous vertigo has a cerebellar stroke until proven otherwise. This finding alone should prompt urgent neuroimaging (CT followed by MRI), regardless of other examination findings [5].

❑ **Clinical Pearl:**

The "grade 3 truncal ataxia" sign — cannot sit unsupported — has 95% specificity for cerebellar stroke in the AVS population. It is the single most powerful clinical sign you can elicit in 30 seconds without equipment [5,6].

III. The HINTS Examination

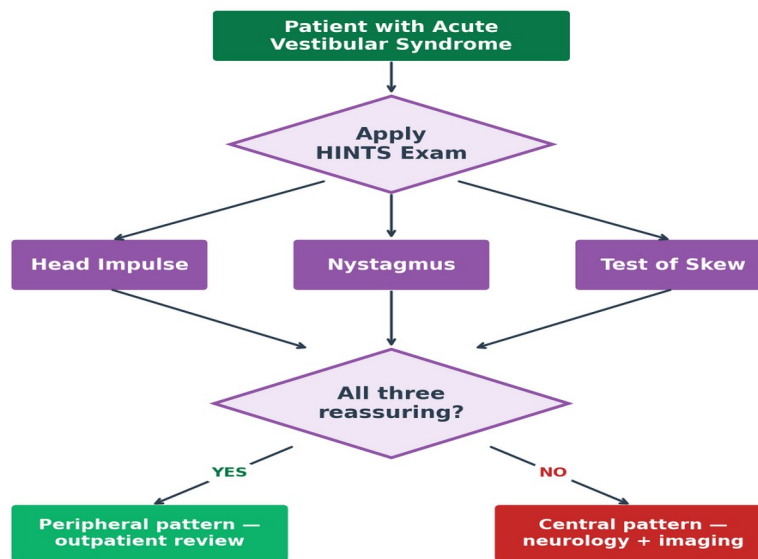


Figure 1. HINTS examination. All three components reassuring = peripheral pattern. Any component abnormal = central pattern requiring neurology input and imaging.

Source: Australian Dizziness Clinics — clinical flowchart.

HINTS (Head Impulse, Nystagmus, Test of Skew) is a three-component oculomotor examination designed to distinguish peripheral from central causes of acute vestibular syndrome (AVS — continuous vertigo lasting more than 24 hours with nystagmus, nausea, and gait unsteadiness) [1].

❑ **Important:**

HINTS is only valid in patients with acute vestibular syndrome — continuous vertigo and spontaneous nystagmus at the time of examination. It must not be applied to patients with episodic vertigo, positional vertigo, or those whose symptoms have resolved. Applying HINTS outside AVS generates false reassurance [7].

Head Impulse Test (h-HIT)

The head impulse test assesses the vestibulo-ocular reflex. The examiner holds the patient's head, asks them to fix their gaze on the examiner's nose, and performs a small (10–20 degree), rapid, unpredictable head rotation in the horizontal plane. In a normal VOR, the eyes remain locked on the target. In a unilateral peripheral vestibular lesion, the eyes move with the head and then make a corrective saccade back to the target — a positive (abnormal) head impulse, which is the reassuring finding in AVS.

- **Abnormal (corrective saccade present) = peripheral lesion.** Reassuring in the context of AVS.
- **Normal (no corrective saccade) in a patient with continuous vertigo and nystagmus = central lesion.** This is the counter-intuitive but critical finding.

Nystagmus Characterisation

Examine for spontaneous nystagmus with the eyes in primary gaze, then in gaze to the right and left. Do not test in extreme lateral gaze, which induces physiological end-point nystagmus in normal subjects.

- **Unidirectional horizontal nystagmus that obeys Alexander's Law** (beats in one direction, stronger on gaze toward the fast phase) = peripheral.
- **Direction-changing gaze-evoked nystagmus** (beats right on right gaze, beats left on left gaze) = central.
- **Pure vertical or pure torsional nystagmus** = central until proven otherwise.

Test of Skew

Perform an alternate cover test. Ask the patient to fix on the examiner's nose. Cover one eye, then rapidly uncover and cover the other. Any vertical corrective movement of the previously covered eye is a skew deviation, indicating a central (brainstem) lesion.

Table 1 — Interpreting the HINTS Examination in AVS

Component	Peripheral pattern	Central pattern (any one = central)
Head Impulse	Abnormal (corrective saccade)	Normal
Nystagmus	Unidirectional, horizontal	Direction-changing, vertical, or torsional
Test of Skew	Absent	Present (vertical corrective movement)
Overall read	All three peripheral = reassuring	Any one central = central lesion until excluded

The mnemonic **INFARCT** (*Impulse Normal, Fast-phase Alternating, Refixation on Cover Test*) captures the central pattern [1].

□ Clinical Insight:

HINTS-plus is more sensitive than early MRI for posterior circulation stroke in AVS (100% vs 80% in the original Kattah cohort). A peripheral HINTS-plus pattern in a patient with AVS is more reassuring than a normal MRI performed within 48 hours of symptom onset [1,2].

IV. HINTS Plus and Audiological Screening

HINTS was extended to HINTS-plus with the addition of a bedside hearing test, which increases sensitivity for anterior inferior cerebellar artery (AICA) territory stroke. AICA infarction can mimic labyrinthitis because the AICA supplies both the labyrinth and the lateral brainstem [8].

Bedside Hearing Assessment

A simple finger-rub test, performed 15–30 cm from each ear with the contralateral ear occluded, detects asymmetric hearing loss with reasonable sensitivity. Any patient who cannot clearly hear the finger rub in one ear, or reports a change in hearing during the current illness, has a positive hearing component.

- **New unilateral hearing loss with AVS:** consider AICA stroke (central) or labyrinthitis (peripheral). HINTS alone does not distinguish these — imaging is required.
- **Normal hearing with AVS:** HINTS read stands as above.

□ Key Point:

HINTS-plus: any central HINTS finding OR new unilateral hearing loss in a patient with AVS mandates urgent MRI with diffusion-weighted imaging, ideally with MR angiography of the posterior circulation [8,9].

The "Normal Exam in an Actively Vertiginous Patient" — A Central Red Flag

One of the most important and under-appreciated principles in vestibular medicine is that a normal bedside examination in a patient who is actively and severely vertiginous is itself a red flag for a central cause. Patients with vestibular neuritis have floridly abnormal findings — horizontal unidirectional nystagmus, a positive head impulse on the affected side, severe gait unsteadiness. If the examination looks normal but the patient is prostrate with vertigo, reconsider the diagnosis.

□ **Important:**

Isolated vertigo with a "normal" bedside examination is not automatically reassuring. In acute cerebellar stroke — particularly in the medial branch of the posterior inferior cerebellar artery (PICA) — the examination may show only subtle findings (mild truncal ataxia, direction-changing nystagmus on lateral gaze, inability to tandem walk). The absence of HINTS peripheral findings in an acutely vertiginous patient is central until proven otherwise [5,10].

□ **Clinical Pearl:**

The "HINTS reassurance" pattern requires all three peripheral findings AND flurid peripheral-type nystagmus AND an obviously abnormal head impulse. If the findings are equivocal, the examination is not diagnostic — escalate to imaging.

V. The Dix-Hallpike Manoeuvre

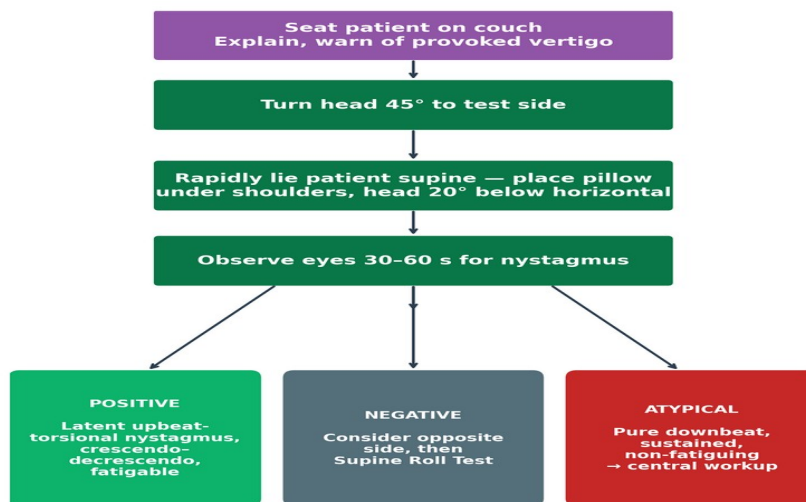


Figure 2. Dix-Hallpike test — procedure and interpretation. Positive (latent upbeat-torsional nystagmus, fatigable) = posterior canal BPPV. Atypical patterns (pure downbeat, persistent, non-fatiguing) warrant central workup.

Source: Australian Dizziness Clinics — clinical flowchart.

The Dix-Hallpike is the diagnostic test for posterior semicircular canal BPPV, which accounts for 80–90% of all BPPV [11]. It is safe, rapid, and can be performed at any bedside.

Indication

Perform the Dix-Hallpike in any patient with triggered episodic vertigo lasting seconds, particularly brought on by lying down, rolling over in bed, looking up, or bending forward. It can be performed in patients with neck stiffness, osteoporosis, or limited cervical range, with appropriate modification.

Technique

- Seat the patient on the examination couch with the head rotated 45 degrees to the side being tested.
- Support the head firmly and quickly lie the patient supine, so that the head extends about 20° below horizontal by placing a pillow underneath the shoulders.
- Maintain the head in this position for at least 30 seconds, observing the eyes for nystagmus.
- Return the patient to the sitting position slowly and observe for reversal nystagmus.
- Repeat on the opposite side after a brief rest.

Interpretation

A positive Dix-Hallpike for posterior canal BPPV produces a characteristic nystagmus with the following features: latency of 1–5 seconds before onset, upbeat and torsional (top poles of the eyes beat toward

the downward ear), lasts less than 60 seconds, fatigues on repeat testing, and is accompanied by vertigo [11,12].

Table 2 — Nystagmus Patterns on Dix-Hallpike

Pattern	Suggests
Upbeat-torsional, latent, <60 seconds, fatigable	Posterior canal BPPV (ipsilateral)
Downbeat, no latency, persistent	Central positional nystagmus — consider cerebellar lesion
Horizontal geotropic or apogeotropic	Lateral canal BPPV — confirm with supine roll test
Purely torsional, persistent	Anterior canal BPPV (rare) or central mimic
No nystagmus but vertigo reported	Equivocal — retest or consider alternative positional cause

Features that should prompt concern for a central mimic: no latency, persistent nystagmus beyond 60 seconds, pure downbeat or vertical pattern, lack of fatigability.

□ Clinical Insight:

A positive Dix-Hallpike is both diagnostic and therapeutic — it tells you this is BPPV, and the affected side, and positions you to perform the Epley manoeuvre in the same encounter. Most general clinicians should aim to become competent in both the diagnostic and the therapeutic manoeuvre.

Pitfalls

The commonest reason for a false-negative Dix-Hallpike is premature termination — clinicians lift the patient back up when they complain of dizziness. The nystagmus may take 5–10 seconds to emerge and is the diagnostic finding, not the subjective vertigo. Maintain the position for at least 30 seconds, and ideally 60 seconds, with the eyes observed throughout.

VI. Supine Roll Test for Lateral Canal BPPV

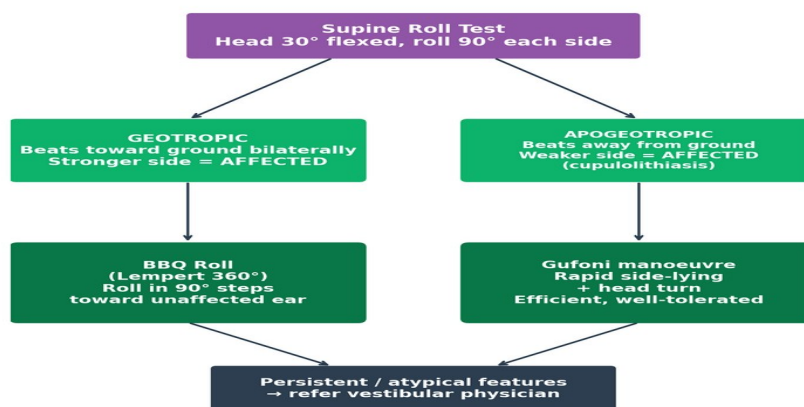


Figure 3. Horizontal (lateral) canal BPPV — diagnosis and treatment. Supine roll differentiates geotropic from apogeotropic patterns and identifies the affected side, guiding BBQ roll or Gufoni manoeuvre selection.

Source: Australian Dizziness Clinics — clinical flowchart.

Lateral (horizontal) canal BPPV accounts for 10–15% of all BPPV and is often missed because the Dix-Hallpike is insensitive for it [13]. Suspect lateral canal BPPV when a patient has typical BPPV-type history (brief positional vertigo) but a negative or equivocal Dix-Hallpike.

Technique

- Lie the patient supine with the head neutral and elevated about 20 degrees.
- Rapidly turn the head 90 degrees to one side and observe for nystagmus for at least 30 seconds.

- Return the head to neutral, allow symptoms to settle, then rotate 90 degrees to the opposite side and again observe for 30 seconds.

Interpretation

- **Geotropic nystagmus** (beats toward the ground-ward ear on each side), more intense on one side: canalithiasis of the lateral canal; the more intense side is the affected side.
- **Apogeotropic nystagmus** (beats away from the ground-ward ear): cupulolithiasis of the lateral canal or canalithiasis in the anterior arm; the less intense side is the affected side.
- **Persistent, direction-changing horizontal nystagmus without positional dependence** = consider central cause.

□ Key Point:

If positional vertigo persists after apparently adequate Epley treatment, perform a supine roll test. Untreated lateral canal BPPV is one of the commonest causes of treatment failure in primary care.

□ Clinical Pearl:

The affected side in lateral canal BPPV can be difficult to determine. Ewald's second law (geotropic pattern: affected side is the ear with stronger nystagmus; apogeotropic: weaker side) is the practical rule at the bedside [13].

VII. Orthostatic Blood Pressure and Autonomic Assessment

Orthostatic dizziness is a common and under-diagnosed cause of presentation in older adults, polypharmacy patients, and those with diabetes or Parkinson's disease. Measurement of lying-standing blood pressure takes three minutes and has a diagnostic yield of 20–30% in patients presenting with "lightheadedness" [14].

Technique

- Ask the patient to lie supine for 5 minutes. Measure BP and heart rate.
- Ask the patient to stand. Measure BP and heart rate at 1 minute and at 3 minutes.
- Continue until 5 minutes if symptoms emerge later (delayed orthostatic hypotension).
- Record any reproduction of the patient's symptoms.

Interpretation

- **Orthostatic hypotension:** drop of systolic BP ≥ 20 mmHg or diastolic ≥ 10 mmHg within 3 minutes of standing.
- **Postural tachycardia syndrome (POTS):** sustained heart rate rise of ≥ 30 bpm (≥ 40 bpm in adolescents) within 10 minutes of standing, without significant BP drop, with orthostatic symptoms for >3 months.
- **Neurogenic orthostatic hypotension:** BP drop without compensatory tachycardia — consider autonomic failure, Parkinson's, multi-system atrophy, diabetic autonomic neuropathy.
- **Delayed orthostatic hypotension:** BP drop occurring after 3 minutes; missed if testing stops too early.

□ Important:

In older patients with "dizziness" and a polypharmacy profile (antihypertensives, diuretics, alpha-blockers, tricyclic antidepressants, antipsychotics), orthostatic hypotension is more likely than vestibular disease. Perform the lying-standing BP before ordering an MRI.

□ Clinical Insight:

A negative single measurement does not exclude orthostatic hypotension. Repeat measurements on different days, ideally first thing in the morning and after meals (post-prandial hypotension), increase diagnostic yield substantially [14].

VIII. Focused Neurological Examination

A focused neurological examination in the dizzy patient is a two-minute examination designed to detect posterior circulation stroke and other central causes. It does not replicate a full neurological examination — it targets the structures most likely to be involved in dizziness-relevant pathology.

Components

- **Visual fields:** confrontation testing for homonymous hemianopia (posterior cerebral artery).
- **Eye movements:** smooth pursuit, saccades, gaze-evoked nystagmus, skew, INO.
- **Facial sensation and strength:** crossed findings (facial sensory loss with contralateral limb weakness) suggest brainstem.
- **Dysarthria and dysphagia screen:** ask the patient to speak and swallow water if safe.
- **Limb power, tone, and coordination:** finger-nose and heel-shin; incoordination out of proportion to vertigo is central.
- **Reflexes and plantars:** asymmetry or an upgoing plantar is central.
- **Gait and tandem gait:** the single most sensitive cerebellar test.

Table 3 — Focal Neurological Signs Suggesting Central Cause

Sign	Localisation
Crossed sensory findings (face one side, limbs other)	Brainstem (lateral medullary, lateral pontine)
Horner's syndrome with vertigo	Lateral medullary (PICA territory)
Dysarthria, dysphagia, or hoarseness with vertigo	Brainstem
Limb ataxia out of proportion to vertigo	Cerebellar hemisphere
Truncal ataxia (inability to sit unsupported)	Cerebellar vermis
New unilateral hearing loss with vertigo	AICA territory until proven otherwise
Direction-changing gaze-evoked nystagmus	Central (brainstem or cerebellum)
Skew deviation	Brainstem

Any single finding in this table in a patient with acute vertigo warrants urgent imaging.

Clinical Pearl:

The rule of parsimony is unreliable in posterior circulation stroke. A single subtle central sign — for example, direction-changing nystagmus with otherwise normal examination — is sufficient evidence of central pathology. Do not wait for multiple confirmatory signs.

IX. Putting It Together — Examination Algorithm

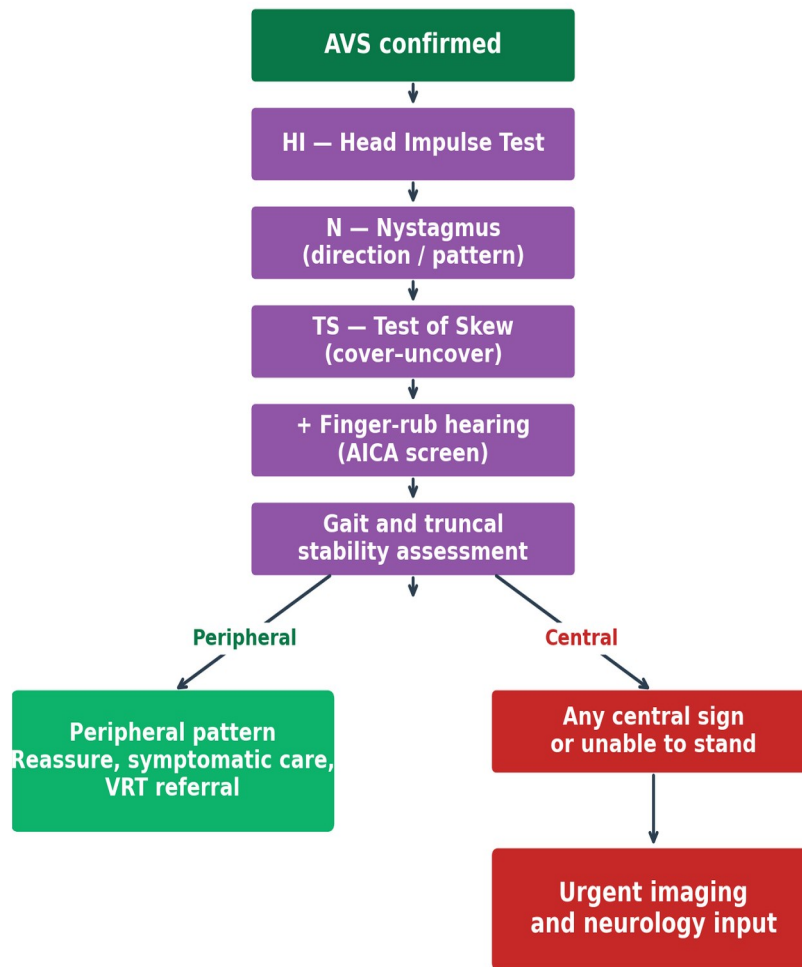


Figure 4. AVS bedside examination algorithm — HINTS+ combined with gait and truncal stability assessment. Peripheral pattern: symptomatic care, VRT referral. Any central sign or inability to stand unaided: urgent imaging and neurology input.

Source: Australian Dizziness Clinics — clinical algorithm.

The bedside examination should be driven by the diagnostic category identified in the history. Performing every test on every patient wastes time and introduces false-positive findings. The algorithm below maps the four TiTrATE categories to the appropriate bedside tests.

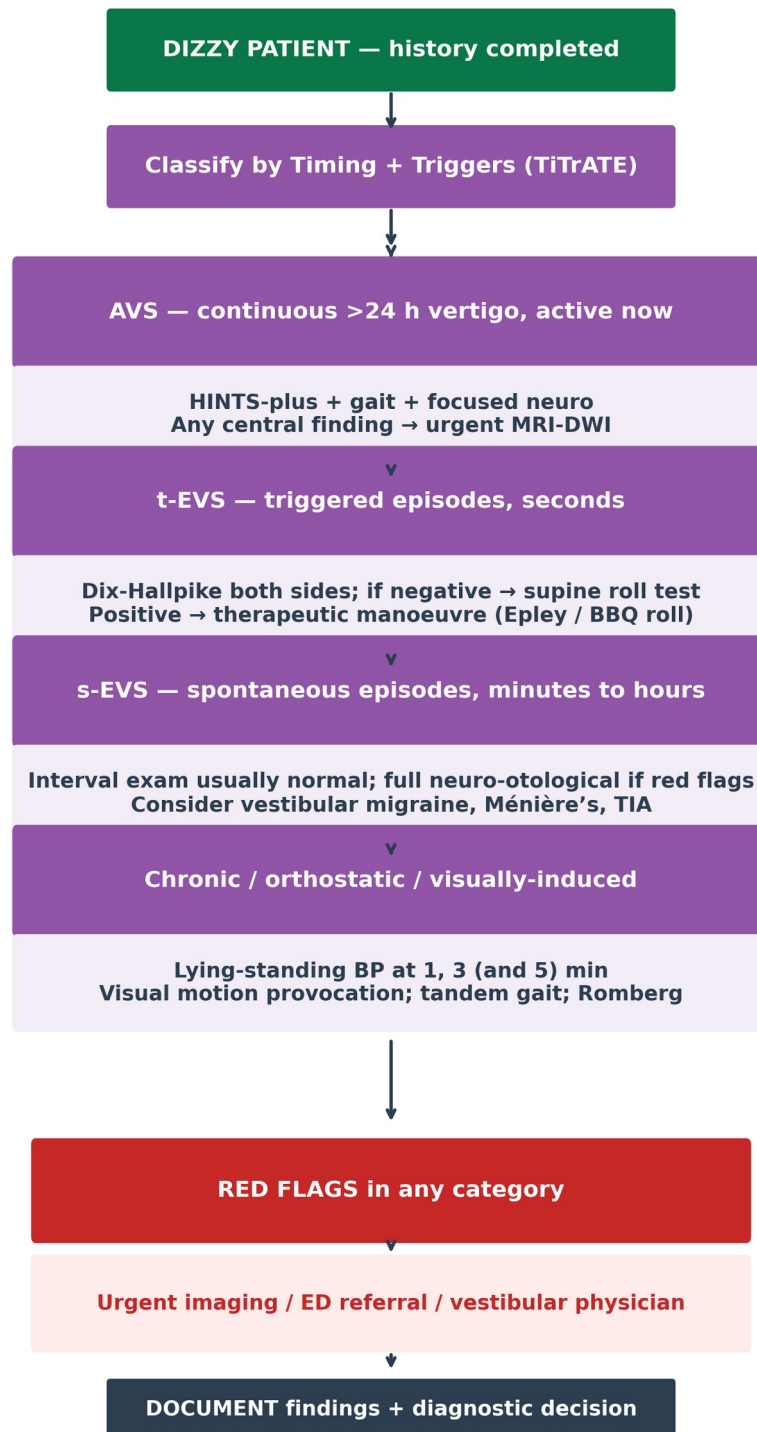


Figure 5. Bedside examination algorithm by diagnostic category — TiTrATE-driven examination selection from history through to documentation.

Source: Australian Dizziness Clinics — clinical algorithm.

The examination you perform is selected by the category the history has placed the patient in. Not every dizzy patient needs HINTS, and not every dizzy patient needs Dix-Hallpike.

□ **Key Point:**

Targeted examination — chosen by category — is faster, more accurate, and more defensible than a non-specific "neurological examination" performed on every dizzy patient.

X. Documentation, Pitfalls, and When to Refer

Documentation of bedside findings is essential for medico-legal clarity and handover. At minimum, record the presence or absence of spontaneous nystagmus (direction, character), the head impulse result and the side tested, the test of skew result, the Dix-Hallpike or supine roll findings (when performed), the orthostatic vitals, the gait and tandem gait, and the key focal neurological findings, as appropriate for the syndrome identified by your history taking.

Common Pitfalls

- Performing HINTS on patients who are not in active AVS — invalid and misleading.
- Terminating the Dix-Hallpike at 10 seconds — too early for the nystagmus to emerge.
- Interpreting end-point nystagmus on extreme lateral gaze as pathological.
- Mistaking an abnormal head impulse as "central" — an abnormal (corrective saccade) head impulse is reassuring.
- Missing lateral canal BPPV because only Dix-Hallpike was performed.
- Stopping orthostatic BP testing at one minute and missing delayed orthostatic hypotension.
- Reassurance based on "no focal neurological signs" in a patient with truncal ataxia and direction-changing nystagmus.

When to Refer to a Vestibular Physician or Dedicated Service

- Any patient with AVS and any central HINTS finding, new hearing loss, truncal ataxia, or focal neurological sign — urgent ED referral for MRI-DWI.
- Persistent BPPV failing two correctly performed Epley manoeuvres on different days — lateral canal, cupulolithiasis, or alternative diagnosis.
- Recurrent or severe BPPV with atypical features (downbeat, purely torsional, persistent nystagmus).
- Any dizzy patient with progressive unilateral hearing loss — exclude vestibular schwannoma.
- Dizziness that persists more than 3 months despite peripheral diagnosis and adequate treatment — consider PPPD, vestibular migraine, bilateral vestibular loss.
- Any diagnostic uncertainty after a structured history and targeted examination.

Clinical Insight:

A confident negative examination in the right clinical category is a powerful diagnostic statement. A confident positive examination shortens the path to treatment. Equivocal findings — not negative, not clearly positive — are the indication for onward referral, not for reassurance [15].

References

- [1] Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke*. 2009;40(11):3504–3510.
- [2] Tarnutzer AA, Berkowitz AL, Robinson KA, Hsieh YH, Newman-Toker DE. Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. *CMAJ*. 2011;183(9):E571–E592.
- [3] Kerber KA, Morgenstern LB, Meurer WJ, et al. Nystagmus assessments documented by emergency physicians in acute dizziness presentations. *Acad Emerg Med*. 2011;18(6):619–626.
- [4] Edlow JA, Carpenter C, Akhter M, et al. Guidelines for Reasonable and Appropriate Care in the Emergency Department (GRACE-3): Acute Dizziness and Vertigo. *Acad Emerg Med*. 2023;30(5):442–486.
- [5] Lee H, Sohn SI, Cho YW, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular topographical patterns. *Neurology*. 2006;67(7):1178–1183.
- [6] Moulin T, Sablot D, Vidry E, et al. Impact of emergency room neurologists on patient management and outcome. *Eur Neurol*. 2003;50(4):207–214.
- [7] Ohle R, Montpellier RA, Marchadier V, et al. Can emergency physicians accurately rule out a central cause of vertigo using the HINTS examination? A systematic review and meta-analysis. *Acad Emerg Med*. 2020;27(9):887–896.

- [8] Newman-Toker DE, Kerber KA, Hsieh YH, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. *Acad Emerg Med*. 2013;20(10):986–996.
- [9] Saber Tehrani AS, Kattah JC, Mantokoudis G, et al. Small strokes causing severe vertigo: frequency of false-negative MRIs and nonlacunar mechanisms. *Neurology*. 2014;83(2):169–173.
- [10] Choi KD, Lee H, Kim JS. Vertigo in brainstem and cerebellar strokes. *Curr Opin Neurol*. 2013;26(1):90–95.
- [11] Bhattacharyya N, Gubbels SP, Schwartz SR, et al. Clinical practice guideline: benign paroxysmal positional vertigo (update). *Otolaryngol Head Neck Surg*. 2017;156(3 Suppl):S1–S47.
- [12] von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: diagnostic criteria. *J Vestib Res*. 2015;25(3–4):105–117.
- [13] Nuti D, Masini M, Mandalà M. Benign paroxysmal positional vertigo and its variants. *Handb Clin Neurol*. 2016;137:241–256.
- [14] Freeman R, Wieling W, Axelrod FB, et al. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. *Clin Auton Res*. 2011;21(2):69–72.
- [15] Strupp M, Brandt T. Peripheral vestibular disorders. *Curr Opin Neurol*. 2013;26(1):81–89.

Disclaimer and Copyright

© Copyright Notice

Copyright © 2026 Australian Dizziness Clinics. All rights reserved. This document and its contents are the intellectual property of Australian Dizziness Clinics. No part of this publication may be reproduced, distributed, transmitted, or stored in any retrieval system in any form or by any means without the prior written permission of Australian Dizziness Clinics.

Educational Use Only

This review is produced solely for the continuing professional development of healthcare clinicians. It is not intended for lay distribution and does not constitute individualised medical advice. Clinical decisions must always be made in the context of each treating clinician's professional judgement and the specific circumstances of each patient.

Accuracy and Currency

Whilst every effort has been made to ensure accuracy at the time of publication, vestibular medicine is a rapidly evolving field. Australian Dizziness Clinics makes no warranties, express or implied, regarding the accuracy, completeness, or fitness for purpose of the content.

Australian Dizziness Clinics
www.AustralianDizzinessClinics.com