

Pragmatic Clinical Approach to “Dizziness”

“Dizziness” is an imprecise symptom covering **presyncope/cardiovascular hypoperfusion, true vertigo, disequilibrium and nonspecific light-headedness**. Do not rely solely on the patient’s descriptive label; diagnostic accuracy improves by establishing the **timing, duration and triggers**, then applying a syndrome-specific examination.

Dr Paul Tervit · July 2026

First identify immediate danger	Assess ABCDE, mental state, blood glucose, temperature, oxygen saturation, pulse and blood pressure. Urgent escalation is required for: • Ongoing chest pain, severe dyspnoea, shock or significant arrhythmia. • Syncope during exertion or while supine. • Persistent bradycardia or tachycardia. • New focal neurological deficit, diplopia, dysarthria or dysphagia. • New severe occipital headache or neck pain. • Inability to sit, stand or walk unsupported. • Sudden unilateral hearing loss with continuous vertigo. • Recent neck trauma/manipulation or suspected vertebral artery dissection. Sudden dizziness associated with focal neurological signs, severe gait ataxia or central ocular findings should be treated as possible posterior-circulation stroke.	
Establish what occurred	Presyncope/cardiogenic phenotype	Ask whether the patient experienced: • A sensation of impending blackout rather than environmental movement. • Visual dimming, “grey-out,” muffled hearing or generalized weakness. • Loss of consciousness or postural tone. • Palpitations immediately before the event. • Chest pain, dyspnoea or exertional onset. • Sudden onset and recovery without prolonged nausea. • Occurrence while exercising or lying supine. • Known structural heart disease, heart failure, previous myocardial infarction or arrhythmia. • Family history of sudden unexplained death. • Recent bleeding, diarrhoea, vomiting, dehydration or medication changes. A warm sensation, sweating, nausea and a prolonged-standing, emotional or painful trigger favour reflex/vasovagal presyncope. Symptoms appearing after standing favour orthostatic hypotension, although arrhythmia can also present with nonspecific light-headedness.
	Vertiginous phenotype	Clarify whether there is an illusion of: • Spinning or rotation. • Tilting, rocking or environmental movement. • Being pulled to one side. • Movement-induced nausea or oscillopsia. Associated hearing loss, tinnitus or aural fullness suggests a peripheral audiovestibular process, but new unilateral hearing loss during an acute vestibular syndrome can also occur with an anterior inferior cerebellar artery stroke.

Classify by timing and triggers	Acute vestibular syndrome	Continuous dizziness/vertigo lasting hours to days, with nausea, spontaneous nystagmus, head-motion intolerance and gait unsteadiness. Principal differential: • Vestibular neuritis. • Labyrinthitis. • Cerebellar or brainstem stroke. • Less commonly toxic, metabolic or inflammatory disease.
	Triggered episodic vestibular syndrome	Brief discrete attacks, usually seconds to less than 1 minute, consistently triggered by: • Rolling in bed. • Looking upward. • Bending forward. • Sitting up or lying down. This pattern strongly suggests benign paroxysmal positional vertigo, but the positional manoeuvre must reproduce the characteristic nystagmus. Distinguish a trigger from an exacerbating factor: almost all ongoing vestibular disorders feel worse when the head moves; this does not make them BPPV.
	Spontaneous episodic vestibular syndrome	Recurrent attacks without a reliable positional trigger, lasting minutes to hours. Consider: • Vestibular migraine. • Ménière disease. • Transient ischaemic attack. • Arrhythmia or reflex presyncope. • Panic or hyperventilation. • Hypoglycaemia.
	Chronic persistent dizziness	Symptoms lasting weeks to months suggest bilateral vestibulopathy, medication toxicity, sensory disequilibrium, persistent postural-perceptual dizziness or a neurodegenerative/cerebellar disorder rather than an acute cardiogenic event.

Focused bedside examination	Cardiovascular and general examination	Vital signs Record: • Pulse rate and regularity. • Blood pressure in both arms when dissection or vascular disease is suspected. • Oxygen saturation. • Temperature. • Capillary glucose. Orthostatic measurements After resting supine for approximately 5 minutes: 1. Record supine blood pressure and pulse. 2. Stand the patient safely. 3. Repeat at 1 and 3 minutes, while documenting reproduced symptoms. Orthostatic hypotension is conventionally a fall in systolic pressure of ≥20 mmHg or diastolic pressure of ≥10 mmHg within 3 minutes of standing. Interpret results in context: dehydration, medication effects and autonomic dysfunction are common, and an orthostatic fall does not automatically exclude another cause. Cardiovascular examination Look for: • Irregular pulse suggesting atrial fibrillation or ectopy. • Marked bradycardia or tachycardia. • Aortic stenosis murmur. • Signs of heart failure. • Pulse deficits or unequal arm pressures. • Evidence of anaemia, bleeding or volume depletion. A normal examination between episodes does not exclude intermittent arrhythmia.
	Neurological examination	Perform and document: • Conscious level and cognition. • Speech and language. • Pupils, visual fields and ocular alignment. • Cranial nerves, including facial sensation and movement. • Palatal movement, voice and swallowing. • Limb tone, power, sensation and reflexes. • Finger–nose and heel–shin testing. • Rapid alternating movements. • Truncal stability. • Standing and gait, including tandem gait when safe.

		Particularly concerning findings • Diplopia, dysarthria, dysphagia or facial numbness. • Limb weakness or sensory loss. • Dysmetria or asymmetric limb ataxia. • Direction-changing or vertical nystagmus. • Severe truncal ataxia. • Inability to walk independently. • New Horner syndrome. • New headache or neck pain. Posterior-circulation stroke may present without obvious hemiparesis or aphasia, so ocular motor and gait findings are especially important.																				
	Eye and vestibular examination	Examine the eyes in primary position and during horizontal and vertical gaze. Remove visual fixation with Frenzel lenses if available. Nystagmus <table><tr><th>Finding</th><th>More suggestive of peripheral disease</th><th>More suggestive of central disease</th></tr><tr><td>Direction</td><td>Unidirectional horizontal–torsional fast phase</td><td>Direction-changing with gaze</td></tr><tr><td>Vertical component</td><td>Minor torsional component possible</td><td>Pure vertical nystagmus</td></tr><tr><td>Torsional pattern</td><td>Typical positional torsional nystagmus</td><td>Pure torsional or atypical pattern</td></tr><tr><td>Visual fixation</td><td>Often suppresses nystagmus</td><td>Little suppression</td></tr><tr><td>Other neurological signs</td><td>Usually absent</td><td>May be present</td></tr><tr><td>Gait</td><td>Usually ambulant, although unsteady</td><td>Severe truncal or gait ataxia possible</td></tr></table> These are tendencies, not absolute rules.	Finding	More suggestive of peripheral disease	More suggestive of central disease	Direction	Unidirectional horizontal–torsional fast phase	Direction-changing with gaze	Vertical component	Minor torsional component possible	Pure vertical nystagmus	Torsional pattern	Typical positional torsional nystagmus	Pure torsional or atypical pattern	Visual fixation	Often suppresses nystagmus	Little suppression	Other neurological signs	Usually absent	May be present	Gait	Usually ambulant, although unsteady
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Syndrome-specific bedside testing	Continuous acute vestibular syndrome: HINTS+	Use HINTS only when all of the following apply: • The patient has continuous acute vestibular symptoms. • Spontaneous nystagmus is present. • The patient remains symptomatic during examination. • The examiner is appropriately trained. It is not a screening test for all dizzy patients and should not be applied to brief episodic symptoms or typical positional BPPV. GRACE-3 recommends trained bedside eye-movement testing in acute vestibular syndrome rather than routine indiscriminate imaging. 1. Head impulse test Ask the patient to fixate on the examiner’s nose and deliver small, rapid, unpredictable head rotations. • Corrective catch-up saccade: supports peripheral vestibular hypofunction. • Normal head impulse despite continuous vertigo and nystagmus: concerning for central disease. A positive head impulse does not completely exclude stroke, particularly an AICA-territory event. 2. Nystagmus • Unidirectional horizontal–torsional nystagmus supports a peripheral lesion. • Direction-changing gaze-evoked, vertical or pure torsional nystagmus suggests a central lesion. 3. Test of skew Alternately cover each eye while the patient fixates on a target. • Vertical corrective refixation suggests skew deviation and a central lesion. 4. Hearing: HINTS+ Test each ear separately with finger rub or another bedside hearing assessment. • New unilateral hearing loss with acute vestibular syndrome should increase concern for labyrinthine or AICA ischaemia. Interpretation A central HINTS pattern is any of: • Normal head impulse. • Direction-changing or vertical nystagmus. • Skew deviation. • New unilateral hearing loss in a concerning context. Such patients require urgent stroke evaluation even if limb examination is normal.
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	Triggered positional attacks	Dix–Hallpike manoeuvre Use for suspected posterior-canal BPPV. A typical positive result is: • Brief latency. • Transient vertigo. • Upbeating torsional nystagmus. • Fatigability on repetition. Atypical, persistent, purely vertical or non-fatigable positional nystagmus raises concern for a central positional disorder. Supine roll test Use when positional symptoms are typical but Dix–Hallpike is negative, particularly when horizontal-canal BPPV is suspected. Do not perform provocative positional testing when there is significant cervical instability, recent neck trauma, suspected vascular dissection or another contraindication to rapid neck movement.
	Episodic presyncope or possible arrhythmia	Perform: • Twelve-lead ECG. • Orthostatic observations. • Cardiovascular examination. • Medication review. • Capillary glucose. • Haemoglobin, electrolytes and other blood tests only when clinically indicated. ECG features increasing concern include: • High-grade atrioventricular block. • Significant sinus bradycardia or pauses. • Ventricular tachycardia. • Pre-excitation. • Long or short QT interval. • Brugada pattern. • New ischaemic change. • Bifascicular block. • Evidence of hypertrophic cardiomyopathy. History, physical examination—including orthostatic measurements—and ECG form the core initial assessment for suspected syncope or cardiovascular presyncope.

Targeted investigations

Clinical situation	Appropriate investigations
Possible arrhythmia	ECG; telemetry if high-risk or symptomatic; ambulatory monitor chosen according to event frequency
Exertional symptoms or structural heart disease	Echocardiography; cardiology assessment; exercise testing when appropriate
Orthostatic symptoms	Serial lying/standing blood pressure and pulse; medication and volume assessment; autonomic testing selectively
Suspected blood loss/metabolic cause	Full blood count, electrolytes, renal function, glucose and other tests guided by history
Central acute vestibular syndrome	Urgent stroke pathway; MRI brain with diffusion-weighted imaging where available
Suspected dissection or vascular occlusion	CT or MR angiography of head and neck
Typical posterior-canal BPPV	Usually no laboratory testing or neuroimaging
New unilateral hearing loss	Formal audiometry and urgent ENT/stroke-directed assessment according to presentation

Routine non-contrast CT is relatively insensitive for small acute posterior-fossa infarction. A normal early MRI can also occasionally miss posterior-circulation stroke; persistent central bedside findings should therefore not be dismissed solely because initial imaging is negative.

Practical differentiating features

Feature	Cardiogenic/presyncope	Peripheral vertigo	Central vertigo
Dominant sensation	Impending blackout, visual dimming	Spinning or movement	Spinning, imbalance or vague dizziness
Typical duration	Seconds–minutes; arrhythmia variable	Seconds in BPPV; hours–days in neuritis	Minutes–days
Trigger	Standing, exertion, arrhythmia; sometimes none	Specific head position or acute vestibular injury	Often spontaneous; sometimes positional
Palpitations/chest symptoms	May be prominent	Unusual	Unusual
Hearing symptoms	Not typical	May occur	New hearing loss possible in AICA stroke
Nystagmus	Usually absent	Unidirectional horizontal–torsional	Direction-changing, vertical or pure torsional
Head impulse	Usually irrelevant	Often abnormal in vestibular neuritis	Often normal
Skew deviation	Absent	Usually absent	May be present
Focal neurological signs	Absent unless hypoperfusion is severe	Absent	May be present
Walking	Weak/light-headed but neurologically coordinated	Usually able to walk with assistance	Disproportionate truncal/gait ataxia
ECG/orthostatics	May be abnormal	Usually unrelated	Usually unrelated

Diagnostic pearls

- Ask “**When does it occur, how long does it last, and what triggers it?**” before asking the patient to choose between “spinning” and “light-headed.”
- Head movement that merely **worsens continuous dizziness** is not equivalent to positionally triggered BPPV.
- Do not use HINTS in an asymptomatic patient, in brief episodic dizziness, or without spontaneous nystagmus.
- A patient with vestibular neuritis should generally have no focal deficit and should usually remain capable of walking, albeit unsteadily.
- Severe truncal ataxia or inability to walk is a central warning sign.
- A normal ECG does not exclude intermittent arrhythmia; match monitoring duration to symptom frequency.
- Palpitations occurring **before** dizziness are more concerning for arrhythmia than palpitations occurring after anxiety and nausea begin.
- Typical BPPV is diagnosed by a characteristic positional nystagmus, not by positional symptoms alone.
- Never allow an apparent peripheral feature to override clear focal neurological findings.

Stepwise algorithm

- Stabilize:** ABCDE, glucose, vital signs and immediate ECG if cardiovascular symptoms or presyncope are possible.
- Screen for emergencies:** cardiac instability, focal neurology, severe gait ataxia, new headache/neck pain or acute hearing loss.
- Define timing:** continuous, triggered episodic, spontaneous episodic or chronic.
- Assess cardiogenic probability:** exertional/supine onset, palpitations, structural heart disease, family history, medications and volume loss.
- Examine:** orthostatic observations, cardiovascular system, complete focused neurological examination, eye movements and gait.
- Continuous syndrome with nystagmus:** trained HINTS+ examination; central or equivocal findings → urgent stroke pathway.
- Brief position-triggered syndrome:** Dix–Hallpike, followed by supine roll testing when indicated.
- Presyncope phenotype:** ECG and targeted laboratory testing; telemetry, ambulatory monitoring or echocardiography according to risk.
- Image selectively:** MRI/vascular imaging for suspected central disease; avoid routine imaging for characteristic BPPV.
- Reassess:** if the findings do not form a coherent peripheral or cardiovascular syndrome, assume a potentially central or systemic cause until adequately excluded.



