Classification of Vestibular Symptoms:

A Comprehensive Clinical Review

Part of the Approach to the Dizzy Patient Series

Australian Dizziness Clinics | **www.AustralianDizzinessClinics.com**

Australian Dizziness Clinics Team | Version 1.0 | April 2026

How to Use This Review

This document forms **Section 2** of the *Approach to the Dizzy Patient* clinician series produced by Australian Dizziness Clinics. It provides a comprehensive, evidence-based review of the classification of vestibular symptoms — the foundational language of vestibular medicine — synthesising seminal international consensus documents, contemporary epidemiological data, and modern clinical frameworks.

The review is structured to serve both as a **reference text** for clinicians entering vestibular medicine and as a **quick-access summary** for practitioners who already work in the field. Sections build logically: from the rationale for classification, through the landmark Bárány Society symptom taxonomy, into epidemiology, and concluding with the clinical frameworks — TiTrATE and vestibular syndrome categories — that translate symptom language into bedside diagnosis.

 Key Point: *Consistent symptom language is the single most important quality control tool in vestibular medicine. Mastering the Bárány Society definitions transforms your history-taking and enables accurate cross-disciplinary communication.*

Callout box guide:

 Key Point: *Green boxes highlight foundational definitions and key take-home points.*

 Clinical Insight: Blue boxes contain clinical context, diagnostic reasoning, and nuance relevant to daily practice.

 Clinical Pearl: Gold boxes are bedside pearls — high-yield clinical tips with immediate applicability.

Table of Contents

I. Introduction: Why Symptom Classification Matters

Vestibular medicine is fundamentally a symptom-driven discipline. Unlike specialties that rely primarily on objective biomarkers or imaging, the vestibular physician must elicit, interpret, and classify subjective patient-reported experiences to generate an accurate differential diagnosis. This dependence on symptom language makes standardised terminology not merely convenient but clinically essential.

For decades, the vocabulary used to describe vestibular complaints varied widely across neurology, otolaryngology, and general practice. Terms such as *"dizziness"*, *"vertigo"*, and *"light-headedness"* were used interchangeably or inconsistently, leading to diagnostic confusion and barriers to meaningful research collaboration (1). Patients were often asked to classify their own symptoms into investigator-defined categories — a practice shown to have poor reliability and reproducibility (2).

The absence of formal criteria for vestibular symptoms stood in stark contrast to parallel specialties such as headache medicine, where the International Headache Society had long published precise, operationalised definitions that transformed both clinical practice and research. The Bárány Society recognised this gap and, through its Committee for the Classification of Vestibular Disorders, embarked on a systematic effort to establish internationally agreed definitions of vestibular symptoms as the foundation for a broader International Classification of Vestibular Disorders (ICVD) (1).

 Clinical Insight: The shift from asking patients "what type of dizziness do you have?" to asking about timing, triggers, and associated features — guided by standardised symptom definitions — is one of the most important advances in vestibular medicine practice.

The landmark 2009 Bárány Society paper by Bisdorff, von Brevern, Lempert, and Newman-Toker established the first formal consensus definitions of vestibular symptoms (1). This document, and the ICVD framework it anchored, has since underpinned the diagnostic criteria for all major vestibular disorders — from Ménière's disease and BPPV to Persistent Postural-Perceptual Dizziness (PPPD) and vestibular migraine (3,4,5).

This review synthesises the 2009 classification with subsequent ICVD developments, epidemiological context, and the clinically practical TiTrATE framework — providing a complete reference for the vestibular physician approaching a dizzy patient.

 Key Point: *The Bárány Society's 2009 symptom classification is the foundation of all subsequent ICVD diagnostic criteria. Every vestibular diagnosis rests on precise symptom definitions.*

II. The Bárány Society 2009 Classification Framework

The 2009 consensus document by Bisdorff et al. defined four major categories of vestibular symptoms, each with precise operational definitions: **vertigo**, **non-vertiginous dizziness**, **vestibulo-visual symptoms**, and **postural symptoms**. This taxonomy was explicitly designed to be *symptom-based rather than aetiological*, enabling classification independent of — and prior to — the establishment of a specific diagnosis (1).

Vertigo

Vertigo is defined as a *"false sensation of self-motion or distorted perception of self-motion"* in the absence of actual movement, or an exaggerated and distorted perception during actual movement (1). It is further subdivided into:

Subtype	Definition
Internal Vertigo	False sensation of self-motion — the patient perceives that they themselves are moving or spinning
External Vertigo	False sensation that the visual surround (environment) is moving or spinning; synonymous with oscillopsia when the percept is one of back-and-forth oscillation
Positional Vertigo	Vertigo triggered by a change in head position relative to gravity
Head Motion-Induced Vertigo	Vertigo provoked by head movement, independent of position change

Clinically, vertigo points to dysfunction in the peripheral vestibular apparatus or its central connections. It does not, however, distinguish between peripheral and central causes — both BPPV and posterior fossa stroke can present with vertigo. The *quality* and *temporal profile* of vertigo, rather than its mere presence, guide the differential (2,6).

 Clinical Pearl: Do not anchor on "spinning" as the defining feature of vertigo. Patients describe vestibular vertigo in many ways — rocking, tilting, swaying, bouncing. The core diagnostic criterion is falsely perceived motion, not a specific quality of that motion.

Non-Vertiginous Dizziness

Non-vertiginous dizziness is defined as a *"sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion"* (1). It encompasses the common patient descriptions of light-headedness, floating sensation, or feeling "off" — where spatial disorientation is present but no illusion of movement occurs.

This category is clinically distinct from vertigo in its implications. While vertigo strongly suggests vestibular pathology, non-vertiginous dizziness has a broader differential that includes:

- Persistent Postural-Perceptual Dizziness (PPPD) — the most common cause of chronic non-vertiginous dizziness in tertiary vestibular clinics (3)
- Orthostatic hypotension and haemodynamic causes
- Anaemia, metabolic, and pharmacological causes
- Anxiety and somatoform disorders
- Chronic uncompensated vestibular loss

 Key Point: *Non-vertiginous dizziness is the dominant symptom in PPPD, which affects up to 15–20% of patients referred to specialist vestibular services. Distinguishing PPPD from acute vestibular disorders early prevents unnecessary investigation and directs patients toward appropriate multimodal management.*

 Clinical Insight: Patients with PPPD typically describe their non-vertiginous dizziness as worsened by visual motion stimuli (crowded environments, scrolling screens, busy patterns) and by active engagement with balance tasks. This pattern of visually-mediated exacerbation distinguishes it from most peripheral vestibular conditions.

Vestibulo-Visual Symptoms

Vestibulo-visual symptoms arise from disruption of the vestibulo-ocular reflex (VOR) and its interaction with the visual system. The 2009 classification defines five distinct vestibulo-visual symptoms (1,7):

Symptom	Definition and Clinical Significance
Oscillopsia	Illusory oscillation or movement of the visual environment. Most commonly gaze-evoked during head movement (dynamic oscillopsia). Bilateral vestibular hypofunction is the classic cause; also occurs in acquired nystagmus.
External Vertigo	Perceived spinning or movement of the stationary visual environment. Distinguished from oscillopsia by the rotatory or directional nature of the percept.
Visual Lag	A delay in the visual scene catching up with head movement. Reflects impaired VOR gain — the eye does not move sufficiently fast to keep the image stable on the fovea.
Visual Tilt	Perceived tilt of the visual environment (objects appear leaning). Indicates otolith dysfunction or central disruption of the subjective visual vertical, seen in labyrinthine pathology and lateral medullary strokes.
Movement-Induced Blur	Blurring of vision during head movement in the absence of frank oscillopsia. Represents mild VOR gain asymmetry or incomplete compensation.

 Clinical Pearl: Oscillopsia during head movement (Dizziness Handicap Inventory question: "Do you have difficulty reading?") is highly sensitive for bilateral vestibular hypofunction. The head impulse test — VHIT in the clinic — quantifies the VOR deficit responsible.

Vestibulo-visual symptoms are highly relevant to functional assessment and rehabilitation planning. Patients with bilateral vestibular loss require gaze stabilisation exercises targeting the VOR; those with PPPD-type visually-induced dizziness require a graduated visual desensitisation programme (7,8).

 Key Point: *Vestibulo-visual symptoms implicate the VOR or its integration with central visual processing. They narrow the differential significantly — from the peripheral hair cell to the flocculus — and guide targeted VFT selection.*

Postural Symptoms

Postural symptoms represent the balance and stability impairment domain of vestibular dysfunction. The 2009 classification defines four postural symptoms (1,8):

Symptom	Definition
Unsteadiness	Feeling of being unstable (tendency to fall or deviate) while sitting, standing, or walking, without a directional preference. The most common postural vestibular symptom.
Directional Pulsion	Tendency to deviate or fall in a specific direction. Peripheral causes include acute unilateral vestibular loss; central causes include lateral medullary and cerebellar lesions.
Near-Falls	Episodes of almost falling that require a protective movement or grasping to prevent a fall. Indicates significant balance compromise.
Falls	Actual loss of balance resulting in hitting the floor or a supporting surface. The most clinically urgent postural symptom; requires fall risk assessment and mitigation.

The distinction between unsteadiness (undirected) and directional pulsion (directed) is diagnostically valuable. Directional pulsion — particularly ipsilateral falling — in the acute setting points to acute

unilateral vestibular deafferentation (vestibular neuritis, labyrinthitis) or a central lesion affecting the vestibulocerebellar pathways (9).

 Clinical Pearl: Falls in elderly patients with vestibular dysfunction carry a mortality risk equivalent to hip fracture. Screening for postural symptoms and proactive falls risk management — including home hazard assessment, vestibular rehabilitation, and medication review — is a core vestibular physician responsibility.

III. Spontaneous and Triggered Vestibular Symptoms

A dimension of the Bisdorff 2009 classification that deserves explicit attention is the **spontaneous versus triggered** axis (1). The 2009 document applies this distinction across all four symptom categories — vertigo, non-vertiginous dizziness, vestibulo-visual symptoms, and postural symptoms — though the diagnostic implications are not equal across categories. Understanding where the distinction is most powerful, and where it is more nuanced, is essential for applying the classification framework correctly at the bedside.

 Key Point: *The spontaneous/triggered axis applies to all four Bárány Society symptom categories. However, it carries the most diagnostic precision in vertigo and dizziness, has critical but underappreciated implications in vestibulo-visual symptoms, and is clinically useful but less binary in postural symptoms.*

Application to Vertigo and Non-Vertiginous Dizziness

This is where the spontaneous/triggered distinction is most fully developed in the 2009 paper and where it has the clearest validated diagnostic pathways.

Spontaneous vestibular symptoms occur **without an identifiable precipitating trigger**. Spontaneous episodic vertigo — discrete attacks with symptom-free intervals — is characterised by duration as the primary discriminator:

Duration of Spontaneous Episode	Key Differential Diagnoses
Seconds	Vestibular paroxysmia (neurovascular compression of CN VIII)
Minutes to hours (20 min – 12 h)	Ménière's disease; TIA (<1 hour, with neurological features)
Hours to days (up to 72 h)	Vestibular migraine; prolonged Ménière's attack
Days to weeks (continuous onset)	Acute Vestibular Syndrome — vestibular neuritis, labyrinthitis, posterior fossa stroke

Triggered vestibular symptoms are **reproducibly provoked by a specific stimulus**. The 2009 classification identifies five major trigger categories (1):

Trigger Type	Clinical Example
Positional	Change in head position relative to gravity → BPPV (canal repositioning diagnostic and therapeutic)
Head Motion-Induced	Active or passive head movement triggers symptoms → uncompensated unilateral or bilateral vestibular loss
Visual Motion-Induced	Moving visual stimuli (scrolling, crowds, driving) → PPPD; also visually-

Trigger Type	Clinical Example
	induced dizziness in bilateral vestibular hypofunction
Sound/Pressure-Induced	Loud sounds (Tullio) or Valsalva (Hennebert) trigger vertigo → Superior Canal Dehiscence, perilymph fistula
Orthostatic	Standing from sitting/lying → orthostatic hypotension, POTS, vasovagal syncope

 Clinical Pearl: The single most useful question in the vestibular history: "Does the dizziness come on by itself, or does something bring it on?" This one question stratifies the entire differential. Spontaneous episodic symptoms with clear interictal baseline point to Ménière's disease, vestibular migraine, or vestibular paroxysmia. Triggered symptoms demand identification of the specific trigger to reach a diagnosis.

Application to Vestibulo-Visual Symptoms

The spontaneous/triggered axis applies equally to vestibulo-visual symptoms, but its clinical significance is **underappreciated in practice**. The 2009 paper explicitly describes both spontaneous and triggered forms of oscillopsia and other vestibulo-visual symptoms, and the distinction is diagnostically important (1,7).

The most critical distinction is in **oscillopsia**. Oscillopsia arising *spontaneously at rest* — without any head movement or positional change — almost invariably indicates **acquired nystagmus from a central neurological cause**: cerebellar disease, brainstem lesions, or acquired pendular nystagmus. It is a red flag symptom that warrants urgent neurological investigation (7).

By contrast, oscillopsia that is *triggered by head movement* (dynamic oscillopsia) indicates **impaired VOR gain** — the eye does not rotate sufficiently to stabilise the image during head motion. This is the hallmark of bilateral vestibular hypofunction and severe unilateral vestibular loss. It is peripheral in origin and managed with gaze stabilisation vestibular rehabilitation (7,8).

Oscillopsia Type	Trigger Pattern
Spontaneous oscillopsia	Present at rest, without head movement
Dynamic oscillopsia	Triggered by head movement only
Postural oscillopsia	Triggered by walking or running

 Clinical Insight: The same symptom word — "things bounce when I walk" — represents two completely different pathologies depending on trigger pattern. Oscillopsia appearing only during locomotion is a VOR gain deficit; oscillopsia present while sitting still looking at a stationary target is nystagmus requiring neuroimaging. Failure to distinguish these has significant consequences for investigation and management.

Visual tilt — the perception that the visual environment is tilted — also has a spontaneous/triggered dimension. *Spontaneous* visual tilt (present at rest) indicates otolith dysfunction or central disruption of the subjective visual vertical, as seen in lateral medullary infarction (Wallenberg syndrome) or acute labyrinthine pathology. *Triggered* visual tilt (occurring during or after head movements) is less specific but may indicate dynamic otolith asymmetry or incomplete central compensation.

 Key Point: *Always ask about the trigger pattern of vestibulo-visual symptoms explicitly. Spontaneous oscillopsia at rest = urgent neurological cause until proven otherwise. Head-motion triggered oscillopsia = VOR deficit requiring VHIT quantification and rehabilitation planning.*

Application to Postural Symptoms

The spontaneous/triggered distinction also applies to postural symptoms, though with somewhat less binary diagnostic precision than in the preceding categories. Nonetheless, the 2009 classification's application of this axis to postural symptoms carries meaningful clinical information (1,8).

Unsteadiness that is *constant and present in all environments* points to a fixed vestibular deficit — bilateral vestibular hypofunction, severely uncompensated unilateral loss, or cerebellar ataxia. Unsteadiness that is *triggered by head movement* suggests ongoing dynamic vestibular asymmetry or incomplete compensation following acute vestibular neuritis. Unsteadiness that is predominantly *triggered by visual motion stimuli or complex visual environments* (supermarkets, escalators, busy streets) is a key diagnostic feature of PPPD, reflecting the over-reliance on visual input in the face of a dysregulated postural threat-response system (3).

Postural Symptom Pattern	Trigger
Constant unsteadiness	Spontaneous — no specific trigger
Motion-triggered unsteadiness	Head movement
Environment-triggered unsteadiness	Visual motion, busy spaces
Orthostatic unsteadiness	Standing
Directional pulsion	Spontaneous (acute onset)

Falls and near-falls also have a spontaneous/triggered dimension with practical safety implications. Falls occurring in the dark or on uneven surfaces (removing visual and proprioceptive compensation) indicate vestibular-dominant balance control with poor multisensory redundancy — characteristic of bilateral vestibular hypofunction. Falls occurring during head turns or rapid positional changes suggest dynamic VOR or otolith dysfunction.

 Clinical Pearl: Ask falls patients specifically: "Do you fall more in the dark or in the shower?" A yes points to vestibular-dependent balance with insufficient somatosensory and visual backup — a hallmark of bilateral vestibulopathy. This single question informs both the likely diagnosis and the immediate safety intervention (nightlight, shower chair).

Clinical Significance of the Spontaneous/Triggered Distinction Across All Domains

Applying the spontaneous/triggered axis systematically across all four symptom categories transforms the clinical history from a narrative into a structured diagnostic tool. The following principles consolidate the clinical application:

- **Examination targeting:** Triggered positional symptoms → positional testing (Dix-Hallpike, supine roll). Spontaneous AVS → HINTS+. Head-motion triggered oscillopsia → VHIT. Spontaneous oscillopsia at rest → neuroimaging.
- **Investigation selection:** Sound/pressure-triggered symptoms → CT temporal bone. Spontaneous episodic vertigo with hearing fluctuation → audiometry, electrocochleography. Spontaneous oscillopsia → MRI brain.
- **Management pathway:** Triggered BPPV → canalith repositioning. Visual motion-triggered dizziness in PPPD → graded visual desensitisation. Spontaneous Ménière's → endolymph management. Head-motion triggered dynamic oscillopsia → VOR gain rehabilitation.

 Key Point: *Document the trigger pattern explicitly for every vestibular symptom — not just vertigo. The provocation mode is not supplementary history; it is diagnostic data. Spontaneous at rest vs.*

triggered by movement applies differently to each symptom category but always adds clinically actionable information.

IV. The International Classification of Vestibular Disorders (ICVD)

Building on the 2009 symptom definitions, the Bárány Society's Classification Committee (CCBS, later renamed COSC-BS) embarked on a comprehensive programme to produce evidence-based, consensus-driven diagnostic criteria for all major vestibular disorders. This project — the International Classification of Vestibular Disorders (ICVD) — has now produced over 20 consensus documents, all published open-access in the *Journal of Vestibular Research* (12).

Von Brevern et al. (2015) provided an overview of the ICVD structure, describing a three-tier hierarchy:

Level	Description
Symptoms	The foundational layer — standardised definitions of individual vestibular symptoms (Bisdorff 2009)
Syndromes	Symptom constellations that define clinical presentations (e.g., Acute Vestibular Syndrome, Episodic Vestibular Syndrome)
Diseases / Disorders	Specific diagnostic entities with operationalised criteria (e.g., Ménière's disease, BPPV, PPPD, vestibular migraine)

Key ICVD disease criteria documents include diagnostic criteria for Ménière's disease (2015), BPPV (2015), Vestibular Paroxysmia (2016), PPPD (2017), Bilateral Vestibulopathy (2017), Vestibular Migraine (2022), and Presbyvestibulopathy (2019), among many others (12,13).

 Key Point: *Every ICVD diagnostic document explicitly references the 2009 Bisdorff symptom taxonomy. The symptom layer is not historical context — it is the living foundation on which all vestibular diagnoses are constructed.*

A 2025 review by Kaski et al. examined the achievements, challenges, and future directions of the ICVD project, noting that ICVD criteria have now been widely adopted in clinical trials and epidemiological research, significantly improving the comparability of data across centres and countries (14). The review also identified future priorities including criteria for vestibular symptoms in children, motion sickness, and mal de débarquement syndrome.

 Clinical Insight: Using ICVD diagnostic criteria in clinical documentation — even in private practice — aligns your practice with international research standards. When your patients eventually present at tertiary centres or participate in research, symptom and diagnosis coding to ICVD standards ensures data continuity.

V. The TiTrATE Framework: Syndrome-Based Clinical Classification

While the Bárány Society taxonomy defines *individual* symptoms, clinical practice requires organising those symptoms into *syndromes* that guide the examination. Newman-Toker et al. (2015) proposed the **TiTrATE framework** — **Ti**ming, **Tr**iggers, **A**nd **T**argeted **E**xaminations — as a systematic, evidence-based approach to diagnosing acute dizziness and vertigo (9).

The TiTrATE approach rests on the observation that the *type* of dizziness (vertigo vs. non-vertigo vs. presyncope) has poor diagnostic discriminatory value, whereas the *timing and triggers* of symptoms

reliably identify clinically coherent vestibular syndromes. The framework organises all dizzy patients into three primary syndromes:

Acute Vestibular Syndrome (AVS)

AVS is defined by the **rapid onset of sustained dizziness/vertigo lasting days to weeks**, typically accompanied by nausea, vomiting, gait unsteadiness, nystagmus, and head-motion intolerance. The cardinal diagnostic challenge in AVS is distinguishing peripheral causes (vestibular neuritis) from potentially life-threatening central causes (posterior circulation stroke or TIA) (9,15).

The HINTS+ examination (Head Impulse test, Nystagmus pattern, Test of Skew, plus hearing) performs superiorly to MRI DWI in the first 24–48 hours for ruling out posterior circulation stroke in AVS, with sensitivity and specificity exceeding 95% when performed by a trained clinician (15).

 Clinical Pearl: In AVS: a NORMAL head impulse test (no corrective saccade) in the context of vestibular symptoms is a RED FLAG for central pathology. The peripheral vestibular system is implicated by an ABNORMAL (positive) head impulse test — this is the reverse of what many clinicians intuit.

Episodic Vestibular Syndrome (EVS)

EVS is characterised by **discrete, recurrent episodes of vestibular symptoms with return to a symptom-free baseline between episodes**. Episodes may be *spontaneous* (no identifiable trigger, as in vestibular migraine and Ménière's disease) or *triggered* (predictably provoked, as in BPPV — triggered by head position change — or superior canal dehiscence — triggered by Valsalva or loud sounds) (9).

EVS Type	Key Examples
Spontaneous EVS (s-EVS)	Vestibular migraine (seconds to days), Ménière's disease (minutes to hours), TIA (minutes)
Triggered EVS (t-EVS)	BPPV (head-position triggered, seconds–minutes), SCD (Valsalva/sound triggered), orthostatic hypotension (standing triggered)

 Clinical Insight: Duration of spontaneous EVS episodes is diagnostically powerful: TIA episodes last minutes; Ménière's disease attacks last 20 minutes to 12 hours; vestibular migraine episodes last minutes to 72 hours. Episode duration should be documented precisely in the clinical record.

Chronic Vestibular Syndrome (CVS)

CVS describes **ongoing, persistent vestibular symptoms lasting months to years**. This category encompasses bilateral vestibular hypofunction (continuous oscillopsia and unsteadiness), PPPD (predominantly non-vertiginous dizziness with postural and visual triggers), and incompletely compensated unilateral vestibular loss (9).

PPPD has emerged as the most common diagnosis in CVS, accounting for 15–20% of all referrals to specialist dizziness clinics. Its Bárány Society diagnostic criteria require persistent dizziness/unsteadiness or non-spinning vertigo on most days for ≥3 months, provoked or exacerbated by upright posture, active or passive motion, and/or visual stimuli (3).

 Key Point: *The three-syndrome framework (AVS / EVS / CVS) maps directly onto distinct examination priorities: HINTS+ for AVS, Dix-Hallpike/positional testing for t-EVS, and VOR gain measurement with VHIT for CVS. Knowing the syndrome before reaching for the frenzel lens is the hallmark of the trained vestibular physician.*

VI. Clinical Implications for the Vestibular Physician

The classification of vestibular symptoms is not an academic exercise — it has direct, practical implications for every clinical encounter. The following principles summarise the translation of symptom taxonomy into clinical practice.

Precision in History-Taking

The Bárány Society definitions provide a structured vocabulary for history-taking that moves beyond the patient's vernacular description. The physician's task is to map the patient's language onto the classification: *"Spinning" = vertigo (internal); "the room is spinning" = vertigo (external/oscillopsia); "off balance" = postural symptom (unsteadiness); "things bounce when I walk" = vestibulo-visual symptom (dynamic oscillopsia)*. This mapping process is the core diagnostic skill of vestibular medicine (1,2).

 Clinical Pearl: Ask three questions to capture all four symptom domains in every new vestibular patient: (1) "Do you have any spinning or rocking sensation?" (vertigo); (2) "Does your vision blur or bounce during movement?" (vestibulo-visual); (3) "Do you feel unsteady or have you had any falls?" (postural). Non-vertiginous dizziness will emerge spontaneously from the history if the above are negative.

Documentation and Communication

Using standardised ICVD symptom terminology in clinical documentation — patient records, referral letters, and discharge summaries — markedly improves the quality of specialist communication. A referral letter that documents "spontaneous episodic vertigo, duration 20 minutes to 4 hours, with ipsilateral aural fullness and sensorineural hearing loss" is far more actionable than one describing "attacks of severe dizziness" (12).

For the GP-vestibular physician interface, shared use of ICVD-based symptom language also reduces the rate of unnecessary investigations. A patient documented as presenting with "triggered episodic vertigo provoked by rolling in bed, lasting 30–60 seconds, Dix-Hallpike positive for geotropic nystagmus" requires no brain MRI — the diagnosis is made at the bedside (4).

 Clinical Insight: ICVD-based documentation is increasingly expected in medicolegal contexts. Precise symptom classification and the clinical reasoning chain from symptoms to syndrome to diagnosis provides a defensible record that protects both the clinician and the patient.

Rehabilitation and Management Planning

Symptom classification drives rehabilitation selection. Vestibulo-visual symptoms (oscillopsia, gaze instability) indicate gaze stabilisation exercises targeting VOR gain. Postural symptoms (unsteadiness, near-falls) require dynamic balance training and falls risk mitigation. Non-vertiginous dizziness in the CVS context typically indicates PPPD-directed therapy: vestibular rehabilitation combined with cognitive-behavioural strategies and, where appropriate, serotonin-norepinephrine reuptake inhibitors (3,8).

 Key Point: *The vestibular physician's diagnostic formulation should explicitly map each identified symptom domain to a management target: vestibular deficit → rehabilitation; PPPD → multimodal care; falls risk → safety interventions. Symptom classification is the prerequisite for this structured approach.*

References

1. Bisdorff A, Von Brevern M, Lempert T, Newman-Toker DE. Classification of vestibular symptoms: towards an international classification of vestibular disorders. J Vestib Res. 2009;19(1-2):1–13. doi:10.3233/VES-2009-0343
2. Strupp M, Dieterich M, Brandt T. The treatment and natural course of peripheral and central vertigo. Dtsch Arztebl Int. 2011;108(29-30):505–515. doi:10.3238/arztebl.2011.0505
3. Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): Consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society. J Vestib Res. 2017;27(4):191–208. doi:10.3233/VES-170622
4. von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: Diagnostic criteria. J Vestib Res. 2015;25(3-4):105–117. doi:10.3233/VES-150553
5. Lopez-Escamez JA, Carey J, Chung WH, et al. Diagnostic criteria for Ménière's disease. J Vestib Res. 2015;25(1):1–7. doi:10.3233/VES-150549
6. 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. doi:10.1503/cmaj.100174
7. Bronstein AM. Vision and vertigo: some visual aspects of vestibular disorders. J Neurol. 2004;251(4):381–387. doi:10.1007/s00415-004-0410-7
8. Salmito MC, Simoceli L, Manso A, et al. Falls in patients with vestibular disorders. Braz J Otorhinolaryngol. 2020;86(2):139–148. doi:10.1016/j.bjorl.2018.10.009
9. Newman-Toker DE, Edlow JA. TiTrATE: A novel, evidence-based approach to diagnosing acute dizziness and vertigo. Neurol Clin. 2015;33(3):577–599. doi:10.1016/j.ncl.2015.04.011
10. Strupp M, Dieterich M, Brandt T. The treatment and natural course of peripheral and central vertigo. Dtsch Arztebl Int. 2011;108(29-30):505–515.
11. von Brevern M, Bertholon P, Brandt T, et al. Overview of the International Classification of Vestibular Disorders. J Vestib Res. 2015;25(5-6):211–221.
12. Kim HA, Bisdorff A, Bronstein AM, et al. Hemodynamic orthostatic dizziness/vertigo: Diagnostic criteria — Consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society. J Vestib Res. 2019;29(2-3):45–56.
13. Kaski D, Tarnutzer AA, Agrawal Y, et al. The International Classification of Vestibular Disorders: Achievements, challenges, and future directions. J Vestib Res. 2025. doi:10.1177/09574271251313803
14. 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.

Disclaimer & Copyright

This document has been produced by the Australian Dizziness Clinics Team for educational purposes only. It is intended for use by healthcare professionals with appropriate training and clinical experience. The content is synthesised from peer-reviewed literature and international consensus documents; it does not constitute individual clinical advice and should not be used as a substitute for professional clinical judgment.

All reasonable care has been taken to ensure the accuracy of information presented at the time of publication. Vestibular medicine is an evolving field; readers are encouraged to consult current primary literature and the Bárány Society ICVD documents for the most up-to-date diagnostic criteria.

Part of the Approach to the Dizzy Patient Clinician Series | www.australiandizzinessclinics.com