History Taking in Vestibular Medicine

A Clinician's Literature Review

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How to Use This Review

This clinician's literature review synthesises contemporary evidence and consensus frameworks for history taking in vestibular medicine. It provides comprehensive, evidence-based content suitable for GPs, vestibular physiotherapists, and specialist physicians entering or practising in the field of dizziness and balance assessment.

The review is structured to serve both as a **reference text** for clinicians building expertise in vestibular assessment and as a **practical bedside guide** for practitioners seeking to deepen the diagnostic yield of their history-taking. Sections progress from foundational frameworks — the Bárány classification and the TiTrATE model — through clinical content (red flags, medication review, imaging decision-making) to practical synthesis and workflow.

 Key Point: *Structured vestibular history taking — using the Bárány categories, trigger patterns, and red flag screening — establishes a syndrome-level diagnosis in 80% of presentations before any examination or investigation.*

Callout box guide:

 Key Point: *Green boxes highlight foundational definitions, consensus frameworks, and key take-home principles.*

 Clinical Insight: Blue boxes contain clinical reasoning, differential diagnosis nuance, and diagnostic tips with bedside applicability.

 Clinical Pearl: Gold boxes are high-yield clinical pearls — practical tips, red flag questions, and immediately actionable bedside strategies.

Table of Contents

I. Introduction — History as the Primary Vestibular Diagnostic Tool

Vestibular medicine is fundamentally rooted in symptom characterisation. Unlike many diagnostic domains reliant on laboratory biomarkers or imaging pathology, the vestibular physician must elicit, interpret, and classify subjective patient symptoms to construct an accurate differential diagnosis. In most vestibular presentations, the **structured history alone establishes the syndrome diagnosis** — often before examination or investigation occurs.

Contemporary evidence supports this clinical reality. Studies examining diagnostic yield in acute dizziness demonstrate that 80% of vestibular diagnoses are established at the conclusion of the history-taking phase (1,2). MRI, the investigation most clinicians associate with vestibular diagnosis, has significant limitations: it misses approximately 20% of posterior fossa ischaemic strokes in the first 24–48 hours of symptom onset, and is often unnecessary for straightforward cases of positional vertigo or vestibular neuritis (1,3).

The paradigm has shifted from asking patients to self-classify their symptoms (*"Do you have vertigo or dizziness?"*) to capturing symptom details that enable the clinician to classify: timing, triggers, associated features, temporal profile, and red flag characteristics. This shift, anchored in the Bárány Society's 2009 symptom taxonomy and the TiTrATE framework, transforms the history from a narrative into a **structured diagnostic instrument**.

 Key Point: *A structured vestibular history generates a syndrome-level diagnosis before any examination or investigation in the majority of cases. The history is not preliminary to the "real" diagnostic work — it is the core diagnostic work.*

This review synthesises the Bárány classification framework, the TiTrATE model, and contemporary vestibular medicine literature to provide a complete, evidence-based guide to history taking. Each section addresses a distinct component of the vestibular history — from classification frameworks through medication review to red flag screening — with clinical pearls and decision-making frameworks designed for immediate bedside application.

II. The Bárány Classification Applied to History Taking

The Bárány Society's 2009 consensus classification defines four major categories of vestibular symptoms (1,2). In history taking, these categories provide a structured vocabulary that moves beyond the patient's vernacular and generates precise, reproducible documentation. Understanding each category and how to elicit it from patients is the foundational skill of vestibular history taking.

A. The Four Symptom Categories — Precise Definitions

Each category has a precise operational definition:

Symptom Category	Definition	Spontaneous Example	Triggered Example
Vestibular Vertigo	False sensation of self-motion or distorted perception of motion	Sudden spinning sensation while sitting at rest	Vertigo when rolling over in bed (BPPV)
Dizziness	Sensation of spatial disorientation without false motion	Floating feeling in supermarket; presyncopal lightheadedness	Unsteadiness provoked by standing rapidly (orthostasis)
Vestibulo-Visual Symptoms	Disruption of vestibulo-ocular reflex; oscillopsia, gaze instability, visual tilt	Vision bounces while walking (bilateral vestibulopathy)	Objects appear to jump during head turns (VOR deficit)
Vestibular Postural Symptoms	Balance and stability impairment; unsteadiness, directional pulsion, falls	Constant unsteadiness interfering with gait	Falls during night-time bathroom trips (darkness removes visual compensation)

 Clinical Pearl: Applying the four Bárány categories in the first 2 minutes of history predicts the vestibular syndrome before any physical examination — no equipment required. Ask directly: "Do you feel the room spinning? Do you feel off-balance? Does your vision jump? Have you come close to falling?"

B. The Spontaneous vs Triggered Axis

For each symptom category, classify whether the symptom occurs **spontaneously** (without identifiable trigger) or **triggered** (provoked by a specific stimulus). This distinction carries enormous diagnostic weight.

Episode Pattern	Duration / Characteristics	Diagnostic Significance
Spontaneous episodic	Seconds to days, unpredictable onset	Ménière's disease, vestibular paroxysmia, vestibular migraine, TIA
Spontaneous persistent	Days to weeks, continuous or progressive	Vestibular neuritis, stroke, bilateral vestibulopathy, PPPD
Triggered (positional)	Seconds to minutes, provoked by head position change	BPPV; typically canalithiasis or cupulolithiasis
Triggered (motion)	During or immediately after	Uncompensated vestibular

Episode Pattern	Duration / Characteristics	Diagnostic Significance
	head movement	loss, VOR deficit, whiplash
Triggered (visual)	Provoked by moving visual scenes, crowded environments	PPPD, visually-dependent balance, motion sickness history

III. History of Presenting Complaint — The TiTrATE Framework

The TiTrATE framework — **Ti**ming, **Tr**iggers, duration **A**nd associated features — **TE**mplate — provides a structured approach to history taking that generates a provisional syndrome diagnosis (1,3,4). This framework complements and operationalises the Bárány categories.

A. Timing — AVS vs EVS vs CVS

Classify symptoms into one of three temporal syndromes: *Acute Vestibular Syndrome (AVS)* — abrupt onset, continuous or progressive over hours to days; *Episodic Vestibular Syndrome (EVS)* — discrete episodes with symptom-free intervals; *Chronic Vestibular Syndrome (CVS)* — persistent dizziness or postural instability for weeks to months.

B. Triggers — Six Trigger Types

Identify the specific trigger category if symptoms are triggered:

- Positional — change in head position relative to gravity
- Head motion-induced — provoked by active or passive head movement
- Visual motion-induced — triggered by scrolling, crowds, driving, busy environments
- Sound/pressure-induced — evoked by loud sounds (Tullio) or Valsalva manoeuvre
- Orthostatic — provoked by standing from supine or sitting
- Exertional — triggered by sustained physical activity

C. Duration-Based Differential

Episode duration is the single most discriminatory feature. The following table maps duration to likely diagnoses and key distinguishing features:

Episode Duration	Most Likely Diagnoses	Key Distinguishing Feature	Red Flag?
Seconds (< 1 min)	Vestibular paroxysmia, BPPV, Tullio/Hennebert sign	Reproducible trigger; no nystagmus in BPPV if duration < 30 sec	No
Minutes (1–20 min)	Vestibular paroxysmia, brief aura before migraine	Short interictal intervals; high frequency (multiple episodes/day)	No
20 min – 4 hours	Ménière's disease, TIA with vestibular component, vestibular migraine	Associated aural fullness/hearing fluctuation in Ménière's; neurological signs in TIA	Yes if TIA (vascular risk + neuro signs)
4–72 hours	Vestibular migraine, prolonged Ménière's episode	Headache, photophobia in migraine; hearing loss in Ménière's	No
Days to weeks (continuous onset)	Acute Vestibular Syndrome —	New onset, maximal at 24–48 hours;	Yes — AVS until stroke excluded by HINTS+

Episode Duration	Most Likely Diagnoses	Key Distinguishing Feature	Red Flag?
	vestibular neuritis, labyrinthitis, posterior fossa stroke	associated hearing loss in labyrinthitis	
Persistent (weeks–months)	PPPD, bilateral vestibulopathy, incomplete compensation post-neuritis	Worse in visually complex environments; anxiety/avoidance pattern in PPPD	No, unless progressive UHL

 Clinical Insight: Nocturnal onset of vertigo provoked by position change during sleep has an odds ratio of 60 for BPPV. This single historical feature — asking "Does it come on when you turn over in bed at night?" — is highly sensitive and specific.

IV. Associated Symptoms — Auditory, Neurological, Autonomic, and Psychiatric

Associated symptoms narrow the differential dramatically. Systematically screen four domains: auditory, neurological, autonomic, and psychiatric. The presence of specific associated features points toward particular diagnoses.

A. Auditory Symptoms

Presence of hearing loss, tinnitus, or aural fullness implicates peripheral labyrinthine pathology. Ménière's disease classically presents with fluctuating sensorineural hearing loss (low frequencies predominantly); acute labyrinthitis may have associated conductive or sensorineural hearing loss; sudden sensorineural hearing loss with vertigo raises concerns for vascular, autoimmune, and infectious causes — with cochlear involvement occurring either in isolation within the inner ear or with associated brainstem involvement.

B. Neurological Symptoms

Headache pattern, diplopia, dysarthria, dysphagia, focal motor or sensory loss, and ataxia mandate central nervous system evaluation. Headache *preceding* vertigo and associated with photophobia/phonophobia suggests vestibular migraine. Sudden-onset headache *with* new neurological signs suggests stroke. Chronic progressive headache with episodic vertigo suggests vestibular migraine or, rarely, central pathology.

C. Autonomic Symptoms

Nausea, vomiting, diaphoresis, palpitations, and presyncopal symptoms indicate autonomic involvement. Notably, moderate-to-severe nausea and vomiting favour acute unilateral vestibular lesions; central vertigo often causes less nausea despite greater postural instability — a counterintuitive but highly useful clinical distinction.

D. Psychiatric Symptoms

Anxiety, panic, avoidance behaviour, and depression frequently co-occur with vestibular disorders, particularly in PPPD. Screen for pre-morbid anxiety, symptom-triggered panic, and functional decline secondary to fear-avoidance.

Domain	Key Features to Ask About	Diagnostic Significance
Auditory	Hearing loss, tinnitus, aural fullness, fluctuating hearing	Ménière's disease, labyrinthitis, SHL with vestibular syndrome
Neurological	Headache (timing, quality), diplopia, dysarthria, dysphagia, focal weakness	Vestibular migraine, stroke, brainstem lesion; any neuro sign = red flag
Autonomic	Nausea, vomiting, diaphoresis, palpitations, presyncope	Severe nausea favours acute peripheral vestibular lesion over central
Psychiatric	Pre-existing anxiety, panic, avoidance behaviour, depression, health anxiety	PPPD; assess functional impact and psychological comorbidity

 Clinical Pearl: Moderate-to-severe nausea and vomiting favour acute unilateral vestibular lesions over central causes; central vertigo is often less nauseating despite greater postural instability. Use nausea severity as a quick filter: severe nausea = likely peripheral; minimal nausea despite gross ataxia = likely central.

V. Past Medical History — Vestibular Relevance

Specific medical conditions significantly alter the prior probability of certain vestibular diagnoses. A focused past medical history query systematically increases diagnostic sensitivity.

A. Migraine and Motion Sickness History

History of migraines, particularly with aura, and childhood or adult motion sickness are strong prior probability increasers for vestibular migraine. Migraine history also increases risk of PPPD.

B. Cardiovascular Risk Factors

Hypertension, diabetes, atrial fibrillation, prior stroke, hyperlipidemia, and smoking are critical in any acute vestibular presentation. These factors shift suspicion toward vascular causes (vertebrobasilar insufficiency, posterior fossa stroke) and mandate urgent HINTS+ examination and consideration of advanced imaging.

C. Autoimmune and Inflammatory Conditions

Systemic lupus erythematosus, rheumatoid arthritis, and other autoimmune conditions increase risk of immune-mediated vestibular dysfunction, vestibular paroxysmia (via microvascular inflammation), and vestibular neuritis.

D. Ear Surgery, Trauma, and Barotrauma

Cholesteatoma, labyrinthitis ossificans, perilymph fistula, and post-surgical adhesions are important differential diagnoses in any patient with prior ear surgery or significant head/temporal bone trauma. Barotrauma (diving, flying) can precipitate perilymph fistula or superior canal dehiscence symptoms.

E. Family History

Familial migraine, familial Ménière's disease, and hereditary hearing loss (suggesting syndromic vestibular disorder) are relevant contextual factors.

Medical Condition	Vestibular Relevance	Key Question to Ask
Migraine (especially with aura)	Strong predictor of vestibular migraine; increases PPPD risk	Do you have a history of migraines? Did they start before or after your dizziness began?
Hypertension / diabetes / AF	Increases stroke and TIA risk; mandate vascular workup in AVS	Do you have high blood pressure, diabetes, or heart rhythm problems?
SLE / RA / autoimmune disease	Increases vestibular neuritis and paroxysmia risk	Have you been diagnosed with any autoimmune or inflammatory conditions?
Prior ear surgery	Post-surgical adhesions, ossificans, labyrinthitis ossificans	Have you had ear surgery? If so, for what?
Head/temporal trauma	Perilymph fistula, SCD, ossicular disruption	Have you had a head injury or significant trauma to the ear

Medical Condition	Vestibular Relevance	Key Question to Ask
		area?
Meningitis history	Risk of labyrinthitis ossificans and post-meningitic vestibulopathy	Have you ever had meningitis?
Familial migraine or hearing loss	Syndromic vestibular disorder; familial Ménière's	Does anyone in your family have migraines or hearing problems?
Motion sickness history	Significantly increases prior probability of vestibular migraine	Did you get carsick or seasick as a child? Do you still?

 Key Point: *A history of childhood motion sickness, even if resolved in adulthood, significantly increases the prior probability of vestibular migraine in any adult presenting with episodic vertigo. This single historical feature should elevate migraine in your differential substantially.*

VI. Medication Review — Vestibulotoxic and Dizziness-Inducing Drugs

Over 194 medications have been implicated in vestibulotoxicity or dizziness induction (5,6). A thorough medication review is essential in any dizziness presentation, particularly in older adults taking multiple agents.

A. Overview of Drug-Induced Vestibulotoxicity

Vestibulotoxic drugs damage the peripheral vestibular apparatus (hair cells, supporting cells) or the eighth cranial nerve. Some agents are ototoxic (affecting hearing); others are purely vestibulotoxic; many are both. Aminoglycosides, platinum-based chemotherapy, and loop diuretics are the most commonly implicated agents. Notably, toxicity may be **dose-cumulative and delayed** — vestibular symptoms can emerge weeks to months after drug cessation.

B. Major Vestibulotoxic Drug Classes

The following table summarises the major drug classes with vestibulotoxic or dizziness-inducing potential and monitoring recommendations:

Drug Class	Examples	Vestibular/Auditory Effect	Monitoring
Aminoglycosides	Gentamicin, tobramycin, amikacin	Bilateral vestibular and cochlear ototoxicity; cumulative dose-dependent	Baseline and serial vestibular testing (vHIT); renal function; dosing adjustment
Platinum chemotherapy	Cisplatin, carboplatin, oxaliplatin	Cumulative cochlear toxicity, dose-limiting ototoxicity; some vestibular involvement	Baseline and serial audiometry; dose reduction if hearing loss develops
Loop diuretics	Furosemide, bumetanide, ethacrynic acid	High-dose ototoxicity, particularly with IV administration; reversible in most cases	Use lowest effective dose; avoid rapid IV infusions; avoid concurrent aminoglycosides
Vancomycin	Vancomycin	Ototoxicity (rare); higher risk if renal dysfunction or aminoglycoside co-administration	Monitor renal function; target trough levels 15–20 mg/L; avoid nephrotoxic combinations
Quinines/antimalarials	Quinine, chloroquine, mefloquine	Tinnitus, hearing loss, dizziness, visual disturbances; reversible if caught early	Baseline and serial audiometry; stop if hearing loss appears; visual field screening
High-dose NSAIDs/aspirin	Aspirin >3 g/day, ibuprofen (high-dose)	Reversible tinnitus, hearing loss, and dizziness; uncommon at standard doses	Monitor at high doses; counsel on ototoxic risk; dose reduction usually reverses symptoms
Antihypertensives	ACE inhibitors, beta-blockers, calcium antagonists, diuretics	Orthostatic hypotension with dizziness (not true vestibulotoxicity)	Blood pressure monitoring; avoid volume depletion; education on position

Drug Class	Examples	Vestibular/Auditory Effect	Monitoring
			change
Benzodiazepines / anticonvulsants	Diazepam, phenytoin, gabapentin	Dizziness, imbalance, gait disturbance; CNS effects, not direct vestibulotoxicity	Start low, titrate slowly; educate on fall risk; consider deprescribing if prolonged use

C. Drugs That Cause Dizziness Without Direct Vestibulotoxicity

Many medications cause dizziness through mechanisms other than direct vestibular damage: orthostatic hypotension (antihypertensives, diuretics, vasodilators), CNS effects (anticonvulsants, benzodiazepines, antidepressants), or metabolic effects (hypoglycaemia agents, diuretics). Systematic medication review — asking about *new* drugs, dose changes, and specific symptom timing relative to medication initiation — is essential.

 Clinical Pearl: Aminoglycosides are cleared more slowly from perilymph than from serum — bilateral vestibular failure can progress or present de novo up to 6 months after the final dose. Counsel patients on this delayed toxicity risk and arrange baseline VHIT testing if prolonged aminoglycoside therapy is unavoidable.

VII. Previous Treatments and Investigations

Documenting prior diagnoses, treatments, investigations, and rehabilitation attempts provides context and directs future management. This section of the history prevents unnecessary duplication and clarifies the clinical course.

A. Prior Diagnoses and Their Accuracy

Ask explicitly: "Has anyone told you what they think is causing your dizziness?" and "Do you know what tests have been done?" Many patients present with prior labels (often inaccurate) that require recalibration. "Labyrinthitis" is often used for both acute vestibular neuritis and chronic PPPD; "vertigo" is sometimes conflated with dizziness.

B. Medications Tried and Response

Document what vestibular-active medications have been used (prochlorperazine, cinnarizine, betahistine, valproate, tricyclic antidepressants) and their effect. Poor response to standard medications may suggest PPPD rather than peripheral vestibular disorder.

C. Vestibular Rehabilitation History

Prior physiotherapy, including frequency, exercises prescribed, and outcome, informs both diagnosis and prognosis. Dramatic improvement with targeted VFT suggests vestibular deficit; minimal improvement despite compliance may indicate PPPD.

D. Procedures and Surgery

Document prior canalith repositioning procedures (CRP) for BPPV, including which canal was treated, response, and recurrence. Prior tympanostomy tubes, cholesteatoma surgery, or tympanoplasty are relevant to current presentation.

E. Objective Investigations Already Performed

Create a systematic record of imaging (MRI, CT), electrophysiology (vHIT, caloric testing, rotational chair, VNG), and audiology (audiometry, vestibular evoked myogenic potentials). This inventory guides future workup and prevents redundant testing.

Investigation Type	What It Measures	Relevance to History
Audiogram	Hearing thresholds (frequencies 250 Hz – 8 kHz); conductive vs sensorineural loss	Fluctuating SNHL suggests Ménière's; bilateral high-frequency loss suggests ototoxicity; conductive loss suggests ossicular disruption or TM perforation
VNG / videonystagmography	Spontaneous nystagmus, saccade/smooth pursuit quality, positional testing, caloric response	Documents nystagmus characteristics; quantifies VOR asymmetry; localizes lesion (peripheral vs central)
vHIT (video Head Impulse Test)	VOR gain (eye velocity / head	Low gain (< 0.8) indicates VOR

Investigation Type	What It Measures	Relevance to History
	velocity) at high frequencies	deficit; asymmetry points to unilateral loss; normal vHIT with oscillopsia suggests central nystagmus
Caloric testing	VOR response at low frequency to temperature-induced cupular deflection	Unilateral weakness (>25% asymmetry) indicates unilateral peripheral loss; bilateral hyporesponsiveness indicates bilateral vestibulopathy
Rotational chair	VOR at multiple frequencies; gain, phase, and time constant	High-frequency asymmetry suggests peripheral loss; low gain at all frequencies suggests bilateral hypofunction or CNS disorder
MRI / CT brain and IAC	Structural imaging of brain, posterior fossa, internal auditory canal	Excludes stroke, lesions; screens for vestibular schwannoma if asymmetric hearing loss; normal imaging reassures in AVS with normal HINTS+

VIII. Imaging in Vestibular Disease — Indications and Decision-Making

Imaging decisions should be driven by history and bedside findings, not routine protocol. Understanding when imaging is indicated, which modality to select, and when imaging is unnecessary prevents cost, delays, and false reassurance.

A. MRI vs CT — What Each Shows

MRI is superior for soft tissue lesions (acute ischaemia, hemorrhage, demyelination, mass lesions). CT is superior for bone detail (temporal bone fracture, ossicular disruption, superior canal dehiscence). In acute vestibular syndrome, MRI-DWI (diffusion-weighted imaging) is the modality of choice to detect acute ischaemic stroke.

B. Urgent Imaging Indications

Acute vestibular syndrome with red flags (vascular risk factors, abnormal HINTS+, neurological signs, sudden sensorineural hearing loss) warrants urgent MRI. Traumatic vertigo with suspected temporal bone fracture requires CT. Progressive unilateral hearing loss with tinnitus requires MRI IAC to exclude vestibular schwannoma.

C. Non-Urgent Indications

Bilateral vestibulopathy suspected on vHIT testing may benefit from MRI to exclude central lesions. Chronic dizziness with atypical features and normal examination may warrant MRI to exclude rare conditions (demyelination, cerebellitis).

D. When Imaging Is Not Indicated

Classic BPPV (positional vertigo, Dix-Hallpike/supine roll positive, geotropic nystagmus): no imaging. Typical vestibular neuritis (spontaneous vertigo, horizontal-rotatory nystagmus, positive vHIT, absent auditory involvement): no imaging unless red flags present. Confirmed PPPD: no imaging.

Clinical Scenario	Preferred Modality	What to Look For	Urgency
AVS + vascular risk factors	MRI-DWI brain and IAC	Acute DWI lesion in posterior circulation; brainstem, cerebellum	Urgent (within 24–48 h)
HINTS+ positive (abnormal eye movements)	MRI-DWI brain	Posterior circulation ischaemia (basilar, SCA, PICA territory)	Urgent (within 24 h)
Atypical BPPV (apogeotropic, torsional)	MRI brain	Central lesion (stroke, multiple sclerosis, demyelination)	Non-urgent (within 1–2 weeks)
Progressive unilateral sensorineural hearing loss + tinnitus + vertigo	MRI IAC with gadolinium	Vestibular schwannoma, internal auditory canal pathology	Non-urgent (routine appointment)
Bilateral hearing loss + vestibular symptoms	MRI IAC	Bilateral schwannomas (NF2), syndromic disorder	Non-urgent
Classic BPPV (Dix-	No imaging	—	—

Clinical Scenario	Preferred Modality	What to Look For	Urgency
Hallpike positive, geotropic nystagmus)			
Typical vestibular neuritis (no red flags)	No imaging	—	—
Confirmed PPPD (persistent dizziness, visual/postural triggers, abnormal HADS)	No imaging	—	—

 Clinical Insight: MRI-DWI within the first 24–48 hours misses approximately 20% of posterior fossa ischaemic strokes. A bedside HINTS+ examination performed by a trained clinician outperforms early MRI for the diagnosis of stroke in acute vestibular syndrome. Use MRI to confirm clinical suspicion, not to replace clinical assessment.

IX. Functional Impact, Standardised Scales, and Psychosocial History

Quantifying functional impact and psychological burden provides prognostic information, identifies patients at high risk of poor outcome, and guides multimodal management. Several validated scales are recommended for clinical use.

A. Dizziness Handicap Inventory (DHI)

A 25-item self-report questionnaire assessing functional, emotional, and physical impact. Score ≥18 indicates mild handicap; ≥30 indicates moderate; ≥40 indicates severe. The DHI is sensitive to change and guides rehabilitation endpoints.

B. Activities-Specific Balance Confidence Scale (ABC)

A 16-item scale quantifying confidence in balance during daily activities (0–100%). Score <67% indicates high fall risk and predicts future falls and fractures in older adults.

C. Hospital Anxiety and Depression Scale (HADS)

A 14-item scale (score 0–42) screening for anxiety and depression. Scores ≥11 on anxiety or depression subscales suggest probable disorder. HADS is particularly useful in vestibular presentations where mood comorbidity is high (especially PPPD).

D. Visual Vertigo Analogue Scale (VVAS)

Fourteen items, each scored 0–10, assessing dizziness triggered by specific visual scenarios (escalators, crowds, scrolling, driving). Total score reflects visually-provoked dizziness severity and is diagnostic for PPPD and visually-dependent balance.

E. Occupational, Driving, and Falls Risk

Explicitly document impact on work, driving safety, and falls history. Ask: "Has dizziness affected your job?" "Do you feel safe driving?" "Have you had any falls?" These questions identify patients requiring urgent intervention.

Standardised Scale	Domains Measured	Score Range / Interpretation	Clinical Use
DHI (Dizziness Handicap Inventory)	25 items: physical, functional, emotional	0–100; ≥18 mild, ≥30 moderate, ≥40 severe	Tracks functional outcome; guides rehab intensity
ABC (Activities-Specific Balance Confidence)	16 items: confidence in activities (0–100%)	0–100%; <67% indicates high fall risk	Identifies fall risk; predicts fracture risk
HADS (Hospital Anxiety & Depression Scale)	14 items (7 anxiety, 7 depression)	0–42 total; ≥11 on subscale = probable disorder	Screens for comorbid mood; prognostic in PPPD
VVAS (Visual Vertigo Analogue Scale)	14 items rating dizziness in visual scenarios	0–140 total (each item 0–10)	Diagnostic for visually-provoked dizziness; tracks PPPD response

X. Red Flags — History Features Requiring Urgent Assessment

Certain historical features mandate urgent evaluation and/or advanced imaging. These *red flags* identify patients at high risk of serious pathology (stroke, hemorrhage, mass, meningitis, immunocompromised infection).

A. Neurological Red Flags

Diplopia, dysarthria, dysphagia, focal weakness, ataxia, or altered consciousness demand urgent neurological assessment. These signs, in combination with vertigo, strongly suggest central pathology.

B. Vascular Red Flags

Age >60, hypertension, diabetes, atrial fibrillation, hyperlipidemia, smoking, prior stroke, and sudden-onset vertigo in combination with these factors substantially elevate stroke risk. Sudden severe headache with vertigo (possibly meningitis or hemorrhage) is a medical emergency.

C. Red Flags Within Each Bárány Category

Spontaneous oscillopsia (nystagmus) at rest without head movement suggests central pathology. Progressive postural symptoms with falls indicate high safety risk. Sudden unilateral hearing loss in combination with vertigo warrants urgent audiology and MRI.

D. The STANDING Mnemonic

A mnemonic for red flag features:

- Stroke risk (vascular risk factors + acute onset)
- Thunderclap headache (sudden severe headache)
- Ataxia (new gait disorder)
- New neurological signs (diplopia, dysarthria, weakness)
- Diplopia / Dysarthria (cranial nerve involvement)
- Immunocompromised (HIV, chemotherapy, immunosuppression)
- New severe headache (meningitis, hemorrhage concern)
- GCS change (altered consciousness, confusion)

Red Flag Feature	Differential Diagnosis	Required Action	Urgency
Sudden severe headache with vertigo	Meningitis, subarachnoid hemorrhage, acute hemorrhagic stroke	Emergency department; LP if no contraindication; CT/MRI	Emergent
New diplopia with vertigo	Brainstem stroke, multiple sclerosis, cranial nerve lesion	HINTS+ exam; MRI brain; neurology referral	Urgent (within 24 h)
New dysarthria / dysphagia	Brainstem or cerebellar stroke; lateral medullary syndrome	HINTS+; MRI-DWI; swallow assessment	Urgent (within 24 h)

Red Flag Feature	Differential Diagnosis	Required Action	Urgency
New gait ataxia with vertigo	Cerebellar stroke, posterior fossa mass, demyelination	MRI brain; neurology referral	Urgent (within 48 h)
Vascular risk factors + acute vestibular syndrome	Posterior fossa ischaemic stroke	HINTS+ exam; MRI-DWI if positive; cardiology workup	Urgent (within 24 h)
Bilateral nystagmus (spontaneous)	Central pathology, multiple sclerosis, Wernicke encephalopathy	MRI brain; neuro assessment; thiamine levels if indicated	Urgent
New hearing loss + vertigo in vascular patient	Labyrinthine infarction, TIA with auditory involvement	Audiometry; MRI IAC; vascular risk stratification	Urgent
Immunocompromised + vertigo + fever	CNS infection (meningitis, encephalitis, toxoplasmosis)	Emergency department; LP; blood cultures; imaging	Emergent
Altered consciousness / confusion with vertigo	Encephalitis, meningitis, posterior fossa mass, hemorrhage	Emergency department; CT head; LP if indicated	Emergent
Progressive unilateral hearing loss (weeks–months)	Vestibular schwannoma, retrocochlear pathology	MRI IAC with gadolinium; audiology referral	Non-urgent but important (within 2 weeks)

 Clinical Pearl: In acute vestibular syndrome, the single most important red flag question is: "Have you ever had anything like this before?" A first-ever episode in a patient with vascular risk factors is stroke until HINTS+ proves otherwise. Do not wait for imaging if HINTS+ is abnormal.

XI. Synthesising the Vestibular History — A Practical Clinical Workflow

A complete, structured vestibular history conducted efficiently in a typical clinical appointment takes 10–15 minutes and generates a working differential diagnosis before the patient stands for examination. The following workflow operationalises the preceding sections into bedside practice.

A. The 10-Minute Structured Vestibular History

1. **Symptom classification (Bárány):** "Do you feel spinning/rocking? Off-balance? Does your vision jump or blur? Any falls?" → Maps symptoms to categories (vertigo, dizziness, vestibulo-visual, postural).

2. **Timing (TiTrATE onset):** "When did this start? Did it come on suddenly or gradually?" → Classifies into AVS, EVS, or CVS.

3. **Triggers (TiTrATE triggers):** "Does it happen by itself, or does something bring it on?" → Identifies spontaneous vs. triggered; specifies trigger type (positional, motion, visual, etc.).

4. **Duration:** "How long does an episode last?" → Maps to differential (seconds → BPPV/paroxysmia; hours–days → migraine/Ménière's; weeks–months → PPPD/bilateral vestibulopathy).

5. **Associated symptoms (4 domains):** Screen auditory (hearing loss, tinnitus?), neurological (headache, diplopia, weakness?), autonomic (nausea level?), and psychiatric (anxiety, avoidance?) briefly.

6. **Red flags (STANDING):** Ask specifically: "New headache?" "Any weakness or numbness?" "Any double vision?" → Flags requiring urgent workup.

7. **Past medical & medications:** Vascular risk factors, migraine history, prior ear disease, new medications (especially aminoglycosides, diuretics).

B. Mapping History to a Vestibular Syndrome

Use the TiTrATE framework to assign a syndrome:

Syndrome	Defining History Features	Key Differential Diagnoses	Immediate Next Step
AVS (Acute)	Abrupt onset, maximal at 24–48 hours, continuous/progressive, new spontaneous vertigo ± hearing loss	Vestibular neuritis, labyrinthitis, posterior fossa stroke, acute labyrinthitis ossificans	HINTS+ exam; MRI-DWI if vascular risk or HINTS+ abnormal
EVS (Episodic)	Discrete episodes with symptom-free intervals, often triggered (positional, motion, visual); duration seconds–hours	BPPV, Ménière's disease, vestibular paroxysmia, vestibular migraine, orthostatic intolerance	Positional testing (Dix-Hallpike/supine roll) if positional; audiometry if hearing loss
CVS (Chronic)	Persistent dizziness/unsteadines	PPPD, bilateral vestibulopathy,	vHIT (VOR gain); ABC scale for fall risk;

Syndrome	Defining History Features	Key Differential Diagnoses	Immediate Next Step
	s weeks–months, often worsened by visual motion or balance tasks, often anxiety/avoidance pattern	incomplete post-neuritis compensation, psychogenic	HADS for mood; vestibular rehab referral

C. Generating the Differential from History Alone

By the end of the history, you should be able to list a narrow, evidence-based differential. Use *duration, trigger pattern, associated features,* and *red flags* to construct it:

- **Acute onset + vascular risk + abnormal HINTS+ history = stroke until proven otherwise**
- **Positional vertigo, seconds, Dix-Hallpike positive = BPPV**
- **Episodic vertigo, 20 min–4 hours, hearing fluctuation + aural fullness = Ménière's disease**
- **Episodic vertigo, hours–days, headache + photophobia + migraine history = vestibular migraine**
- **Persistent dizziness, visually triggered, anxiety pattern, abnormal ABC score = PPPD**
- **Persistent oscillopsia during head movement, vHIT abnormal = bilateral vestibulopathy or severe UVL**
- **Spontaneous oscillopsia at rest, no head movement needed, normal vHIT = central nystagmus — urgent imaging**

 Key Point: *The structured vestibular history — Bárány category classification, spontaneous/triggered axis, duration-based differential, associated symptoms, and red flag screening — establishes a syndrome diagnosis that directs examination, investigation, and management in the vast majority of vestibular presentations. Master this workflow and you have mastered the diagnostic foundation of vestibular medicine.*

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All reasonable care has been taken to ensure the accuracy of information presented at the time of publication (April 2026). Vestibular medicine is an evolving field; readers are encouraged to consult current primary literature and the Bárány Society International Classification of Vestibular Disorders documents for the most up-to-date diagnostic criteria and management recommendations.

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