Vestibular Migraine:

A Vestibular Physician's Deep Review of Mechanism, Diagnosis, and Management

Vestibular Medicine for Vestibular Physicians

Central Vestibular Pathology — Module 2.5

Australian Dizziness Clinics | *www.AustralianDizzinessClinics.com*

Version 1.0 | May 2026

How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of central vestibular practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

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, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction, History and Epidemiology

Vestibular migraine (VM) is now recognised as the most common cause of recurrent spontaneous vertigo in adults and the leading central cause of episodic vestibular symptoms encountered in a dedicated dizziness clinic [1,7,12]. It sits at the intersection of neurology and otology — a migraine variant in which the brain's vestibular circuits, rather than (or in addition to) the trigeminovascular pain system, generate the dominant symptom. For the vestibular physician, VM is both the great mimic and the great under-diagnosis: its protean attacks overlap with Ménière's disease, BPPV, posterior-circulation ischaemia and functional dizziness, yet a confident clinical diagnosis can be reached at the bedside once the pattern is recognised and competing causes excluded [12,14,15].

The migraine–vertigo association is ancient. Vertigo accompanying headache was described by Aretaeus of Cappadocia, and modern characterisation began in the nineteenth and early twentieth centuries: Prosper Ménière's 1861 account of "apoplectiform cerebral congestion" is widely read as basilar migraine [41], and in 1917 Boenheim coined "hemicrania vestibularis" to describe episodic vertigo in familial migraineurs [40]. Slater's 1979 description of benign recurrent vertigo captured a migraine-related syndrome without consistent headache [23], and Dieterich and Brandt's 1999 series of episodic vertigo related to migraine popularised the term "vestibular migraine" [22]. Neuhauser and colleagues then operationalised migrainous vertigo and measured its prevalence, establishing VM as a discrete entity [6,7].

Consensus criteria followed. In 2012 the Bárány Society and the International Headache Society jointly published diagnostic criteria for vestibular migraine [2], which were incorporated into the appendix of the third edition of the International Classification of Headache Disorders (ICHD-3) [4] and aligned with the International Classification of Vestibular Disorders [5]. A 2022 update retained the original criteria unchanged while refreshing the supporting literature, reflecting a stable and widely adopted definition [3]. A syndrome once regarded as controversial is therefore now a defined diagnosis bridging migraine and vestibular science [1,3].

Epidemiologically, VM affects just under 1% of the general adult population, with a German population-based survey reporting a lifetime prevalence of 0.98% and a one-year prevalence of 0.89% [6,7]. A later United States population survey estimated a higher figure of approximately 2.7%, a difference attributed largely to improved recognition and case-finding [8]. A systematic review of prevalence and familial aggregation confirms substantial under-diagnosis and points to a meaningful hereditary component, with first-degree relatives at several-fold higher risk [9]. Onset is typically in mid-life — mean vestibular-symptom onset in the late thirties to early forties — usually in a patient with a pre-existing migraine history dating to young adulthood [7,8]. Women predominate: community samples show a modest female excess (~60–65%), whereas clinic series report ratios of 3:1 to 5:1, and many women report attacks clustering around menstruation [11,24].

Within specialist practice the burden is striking. VM accounts for roughly 7–11% of patients in dedicated dizziness clinics and is the single most common cause of recurrent vertigo in adults [1,10]. In headache and neurology clinics, approximately 10% of migraineurs meet VM criteria when systematically screened, with probable VM adding a further 2–3% [37]. The wide range across settings (up to 16–30% of dizzy patients in some neurotology series) reflects differing referral patterns and criteria application rather than true biological variability [10,11]. Table 1 summarises prevalence and demographic data.

Table 1. Prevalence of vestibular migraine by population and demographic.

Population / Demographic	Prevalence / Proportion
General adult population (global)	~1% (0.9–1.0%) [6,7]
General adult population (USA)	~2.7% [8]
Migraine sufferers (overall)	~10% meet VM criteria; +2–3% probable VM [37]
Patients in dizziness clinics	~7–11% [1,10]
Patients in headache / neurology clinics	~10% (definite) [37]
Female-to-male ratio (clinic series)	≈3:1, up to 5:1 [11,24]
Typical age of vestibular-symptom onset	Mid adulthood (mean ~38–41 years) [7,8]

Familial aggregation	Several-fold higher risk in first-degree relatives [9]
Ethnic / regional range	~1% (Asian) to ~2–3% (European/African descent) [9]

Functional impact is disproportionate to attack duration. Age-adjusted health-related quality-of-life scores are consistently lower in people with migrainous vertigo than in dizziness-free controls, and two-thirds of affected individuals consult a doctor yet only a minority receive the correct diagnosis [7]. Anticipatory anxiety, activity avoidance, and occupational disruption are common, and psychiatric comorbidity is high — themes returned to in Sections III and IX [25,26].

□ **Key Point:** Vestibular migraine is the commonest cause of recurrent spontaneous vertigo in adults, affects just under 1% of the population, and is markedly under-diagnosed — most patients consult repeatedly before the diagnosis is made [1,7,9].

II. Pathophysiology — Trigemino-Vestibular Mechanisms and Central Processing

The pathophysiology of VM is incompletely understood but is best framed as a migraine disorder expressed through vestibular circuits [12,13,15]. Migraine itself is increasingly conceived as a disorder of dysfunctional sensory gating and homeostatic control rather than a primary vascular event, with cortical spreading depression and trigeminovascular activation as central phenomena [16,20]. In susceptible individuals the same mechanisms perturb the vestibular system, producing episodic vertigo with or without headache. Figure 1 sets out the working cascade from genetic predisposition through to symptomatic attack.

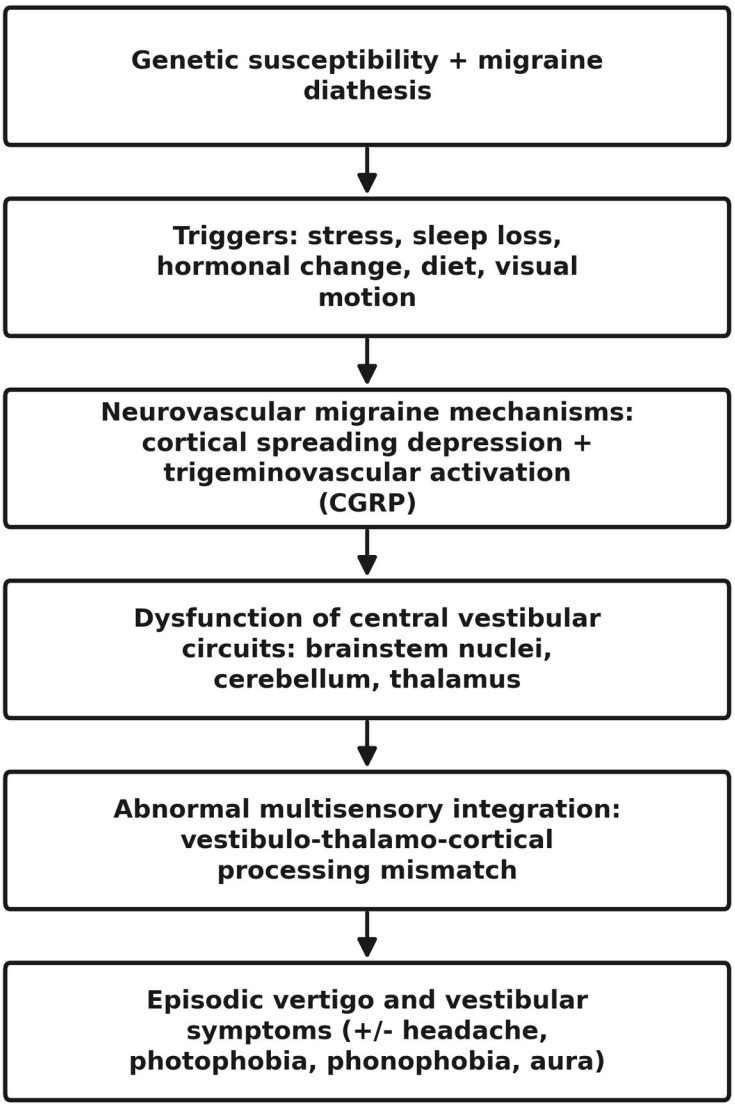

Figure 1. Proposed pathophysiological cascade of vestibular migraine.
Source: Adapted from Espinosa-Sanchez and Lopez-Escamez [13], Furman et al. [12], and Goadsby and Holland [16].

Trigeminovascular–vestibular interaction

The trigeminal innervation of labyrinthine vessels provides an anatomical substrate for cross-talk between the pain and balance systems. Vasoactive neuropeptides — calcitonin gene-related peptide (CGRP) and substance P — localise to perivascular trigeminal afferents in the inner ear, and activation of the trigeminal–vestibulocochlear reflex can produce neurogenic inflammation with plasma-protein extravasation and sensitisation of primary afferents [13,18]. CGRP infusion reproducibly triggers migraine in susceptible individuals and is implicated in photophobia and possibly dizziness, and CGRP-pathway blockade is therapeutically active in migraine — a mechanistic thread now being extended to VM [17,18,36]. Figure 2 depicts the reciprocal connections that underlie this interaction.

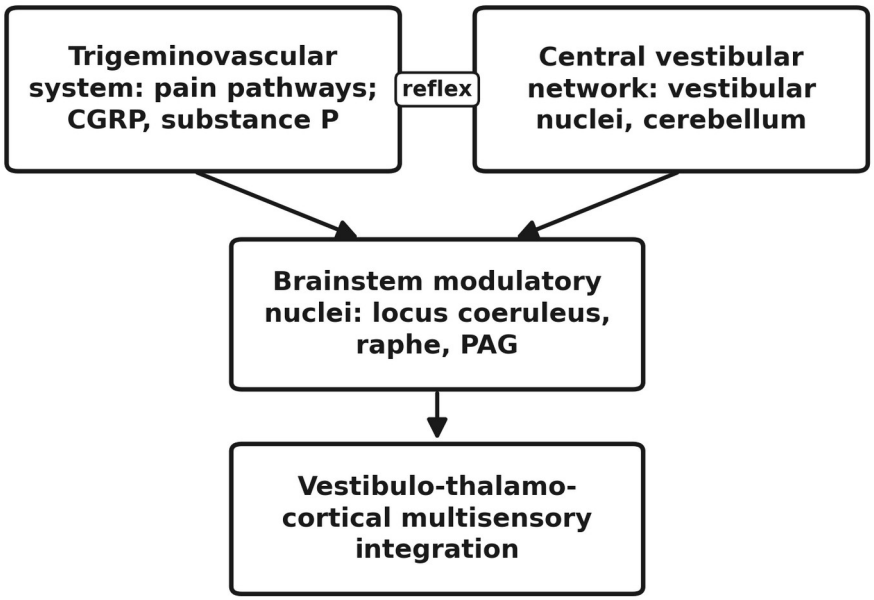

Figure 2. Reciprocal trigeminovascular and central-vestibular networks in vestibular migraine.
Source: Adapted from Espinosa-Sanchez and Lopez-Escamez [13] and Furman et al. [12].

Brainstem, cerebellar and thalamo-cortical dysfunction

Reciprocal connections between the brainstem vestibular nuclei and the structures that modulate trigeminal nociception — rostral ventromedial medulla, periaqueductal grey, locus coeruleus and nucleus raphe magnus — are pivotal to understanding VM, because transient dysfunction at these nodes can gate vestibular signals abnormally [12,13]. Functional imaging during and between attacks points to a vestibulo-thalamo-cortical disorder of multisensory integration, with altered activation across the insula, temporo-parietal junction and visual-motion processing areas and abnormal interplay between vestibular and nociceptive networks [13,15]. This central account explains two clinical hallmarks: the prominence of visually-induced and head-motion-induced symptoms, and the frequent observation of central-type nystagmus during acute attacks [19,21].

Genetic susceptibility and the peripheral contribution

Familial clustering is well described and candidate loci overlap with those implicated in migraine and episodic ataxia, including calcium-channel signalling pathways such as CACNA1A; no single causative gene has been confirmed, and a polygenic susceptibility model is most consistent with the data [9,13]. Although VM is fundamentally a central disorder, migraine mechanisms may secondarily affect the labyrinth — transient cochlear symptoms, mild fluctuating low-frequency thresholds, and the slow accrual of vestibulo-cochlear findings over years argue for a degree of peripheral involvement in a subset of patients [13,21]. This central-with-peripheral-overlap framing is also why VM and Ménière's disease can be so difficult to separate (Section VI).

Cortical spreading depression — the slowly propagating wave of neuronal and glial depolarisation that underlies the migraine aura — provides a unifying mechanism for the transient, fully reversible nature of VM attacks, and can plausibly affect cortical regions that process vestibular information [16,20]. The dominance of head-motion intolerance and visually-induced vertigo in VM is best explained at the level of abnormal sensory weighting: when the brain over-relies on visual and somatosensory cues, ordinary motion and complex visual scenes are misinterpreted as threatening, producing dizziness without any structural vestibular lesion [13,15]. This sensory-reweighting account links VM mechanistically to the visually-induced dizziness and motion sensitivity that so often accompany it and to the PPPD that frequently supervenes [15,27].

□ **Clinical Insight:** Conceptualise VM as migraine expressed through vestibular circuits: trigeminovascular activation and CGRP release acting on a brainstem–thalamo-cortical network that gates and integrates vestibular signals. This frame predicts both the central-type signs seen acutely and the responsiveness to migraine-directed therapy [13,16,18].

III. Clinical Features and the Phenotypic Spectrum

VM presents with recurrent episodes of vertigo or dizziness lasting from minutes to hours, and occasionally up to one to three days, but by definition not beyond 72 hours [2,3]. The vestibular symptom is heterogeneous both between patients and between attacks in the same patient: spontaneous internal or external spinning vertigo is common, but positional vertigo, head-motion-induced dizziness with nausea, and visually-induced vertigo are all recognised qualifying symptoms under the Bárány/ICHD framework [3,5]. One attack may be violent rotational vertigo and the next a subtle disequilibrium — a variability that itself is a diagnostic clue [12,19].

Migraine features and the acephalgic problem

By definition patients have a current or prior history of migraine, and migrainous features accompany the vestibular symptoms in at least half of episodes — migrainous headache, photophobia and phonophobia, or visual aura [2,3]. Crucially, headache is frequently absent during vestibular attacks: acephalgic episodes are common and are the single greatest reason VM is missed or mislabelled [15,37]. Vestibular symptoms and headache may be simultaneous, sequential, or entirely dissociated in time, and many patients report that the relationship has changed over the course of their disease [12,21].

Acute oculographic studies have clarified what is happening during an attack. In a prospective series, pathological nystagmus was recorded in around 70% of patients examined acutely, with the pattern pointing to central-vestibular dysfunction in roughly half, peripheral dysfunction in a minority, and indeterminate localisation in the remainder — confirming that VM can present as a central, a peripheral, or a mixed vestibular disorder [19]. This heterogeneity is itself the diagnostic signature: rather than a single stereotyped examination finding, VM produces a shifting picture across attacks, and it is the recurrence, the migraine association, and the absence of a fixed deficit — not any one positive sign — that secure the diagnosis [12,19].

Duration, frequency and triggers

Episodes most commonly peak in the 5-minute to several-hour range, with a substantial minority lasting longer [3,19]. Frequency is highly variable, with clusters of frequent attacks interspersed with long remissions. Attacks may be spontaneous or provoked by the classic migraine triggers — stress, sleep disruption, dehydration, hormonal change, dietary factors and intense or complex visual motion [14,15]. A lifelong history of motion sickness and childhood periodic syndromes is common and supports a migrainous diathesis [12,24].

Auditory symptoms and the examination

Unlike Ménière's disease, VM does not characteristically cause progressive hearing loss; transient aural fullness, pressure or mild tinnitus can occur during attacks but stable interictal audiometry is the expectation [12,14]. Persistent or progressive unilateral hearing loss should prompt reconsideration of a peripheral diagnosis. The interictal neuro-otological examination is often normal, but during acute attacks pathological nystagmus is observed in around 70% of patients — spontaneous, positional, or gaze-evoked — and frequently shows central features such as direction-changing or persistent down-beating patterns that do not fatigue [19,21]. Long-term follow-up shows that interictal ocular-motor abnormalities, particularly central-type positional nystagmus, accumulate over years and can help distinguish VM from peripheral disorders [21].

Comorbidity

Psychiatric comorbidity is a defining feature rather than an incidental association. Structured assessment shows markedly elevated rates of anxiety and depressive disorders in VM — on the order of 50–65%,

with odds ratios for anxiety disorders far above those of BPPV or vestibular neuritis [25,26]. Persistent postural-perceptual dizziness (PPPD) frequently coexists, producing a background of non-spinning dizziness between discrete attacks and demanding a combined treatment approach [27]. These comorbidities amplify symptom burden and must be screened for and treated directly (Section IX).

□ **Important:** Vestibular attacks without headache (acephalgic VM) are common and are the main reason the diagnosis is missed. A migraine history plus stereotyped recurrent vertigo with photophobia, phonophobia or visual aura — even without head pain — should raise VM strongly [3,15].

IV. Diagnostic Criteria and the Bárány Society Position

VM is a clinical diagnosis defined by the joint Bárány Society/IHS criteria, which specify two tiers — definite vestibular migraine and probable vestibular migraine — and were retained unchanged in the 2022 update [2,3]. Definite VM requires at least five episodes of moderate or severe vestibular symptoms lasting 5 minutes to 72 hours, a current or prior history of migraine, migrainous features during at least half of episodes, and exclusion of a better-fitting diagnosis [3,4]. Probable VM applies when only one of the two migraine-related criteria (history or concurrent features) is met, reflecting high clinical suspicion with one element missing [2,3]. Table 2 sets out both tiers, and Figure 3 renders the decision logic.

Table 2. Diagnostic criteria for vestibular migraine and probable vestibular migraine [2,3].

Criterion	Definite VM	Probable VM
A. Episodes	≥5 episodes of vestibular symptoms,* moderate/severe, lasting 5 min–72 h [3].	≥5 episodes (moderate/severe, 5 min–72 h) — same as definite [3].
B. Migraine history	Current or previous migraine (with or without aura) per ICHD [4].	Only one of B or C is fulfilled (not both) [3].
C. Migraine features during ≥50% of episodes	≥1 of: migrainous headache (≥2 of unilateral, pulsating, moderate/severe, aggravated by activity); photophobia and phonophobia; visual aura [3].	If migraine history absent, ≥1 migraine feature must occur during attacks to satisfy this single criterion [3].
D. Other causes excluded	Not better accounted for by another vestibular or neurological diagnosis [3].	Not better accounted for by another diagnosis — same as definite [3].
Notes	Vestibular symptoms = spontaneous, positional, visually-induced vertigo, or head-motion-induced dizziness with nausea, all of at least moderate severity [3,5].	Probable VM aids classification and research; many such patients respond to standard VM treatment [3,37].

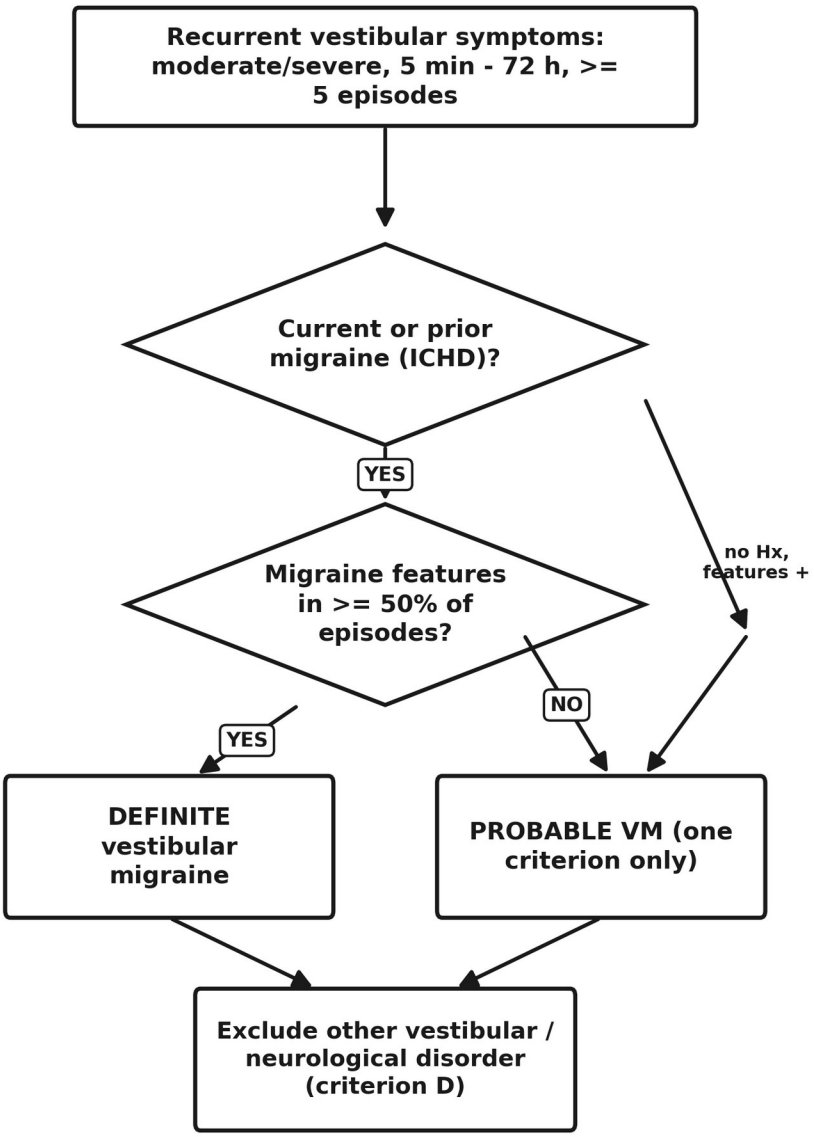

Figure 3. Diagnostic algorithm for vestibular migraine based on the Bárány Society/IHS criteria.
Source: Adapted from Lempert et al. [2,3] and the ICHD-3 classification [4].

In practice, a patient with unexplained recurrent vertigo, a clear migraine background and attacks that frequently carry migrainous features meets definite VM; where one element is missing the patient falls into the probable category, which nonetheless responds to the same treatments [3,37]. The criteria deliberately balance sensitivity against specificity by requiring multiple attacks and a migraine correlation [3]. There is no confirmatory laboratory or imaging test; the diagnosis rests on a history that fits the criteria once competing causes have been excluded [3,5].

□ **Clinical Pearl:** Five episodes, 5 minutes to 72 hours, migraine history, migraine features in half of attacks, nothing else fits — the five-part Bárány checklist is the most reliable route to a confident bedside diagnosis [2,3].

V. Investigations and the Role of Imaging

There is no pathognomonic test for VM; investigation serves to exclude alternative causes and to characterise vestibular function, particularly at first presentation or where features are atypical [3,12]. In a young or mid-aged patient with a classic history and a normal interictal examination, extensive testing

is low-yield, and over-investigation should be avoided [15]. A proportionate workup is summarised in Table 3.

Table 3. Key investigations in suspected vestibular migraine.

Investigation	Purpose and typical findings
Pure-tone audiometry	Usually normal in VM; helps exclude Ménière's disease and other cochlear disorders, and provides a baseline if auditory symptoms develop [12,14].
Vestibular function tests (VNG/caloric, vHIT, VEMP)	Often normal or only mildly abnormal interictally; mainly exclude fixed vestibular loss. A normal head-impulse test argues against neuritis; positional nystagmus without BPPV may support VM but is non-specific [3,19].
MRI brain (± internal auditory canals, gadolinium)	Excludes infarct, demyelination and cerebellopontine-angle tumour; typically normal aside from incidental migraine-related white-matter changes. Image internal auditory canals for unilateral auditory symptoms [12].
Bedside oculomotor exam (during attack)	Direction-changing, gaze-evoked or persistent positional nystagmus during an acute attack favours a central origin such as VM over a peripheral cause [19,21].
Blood tests (metabolic, thyroid, B12)	No specific abnormality in VM; used selectively to exclude systemic contributors to dizziness [12].
Vertigo / headache diary	Documents attack frequency, duration, triggers and treatment response; the principal tool for diagnosis refinement and monitoring [14].

Vestibular laboratory findings in VM are notably inconsistent: some patients show a unilateral caloric weakness or subtle ocular-motor abnormalities, but no pattern is specific, and findings vary between patients and between tests in the same patient [3,19]. The principal value of testing is therefore exclusionary — confirming the absence of unilateral vestibulopathy, a third-window phenomenon, or a central structural lesion. Migraineurs may show non-specific white-matter hyperintensities on MRI that do not explain episodic vertigo and should not be over-interpreted [12]. Because the criteria require that symptoms are "not better accounted for by another diagnosis," a deliberate search for alternatives is essential at first presentation [3].

□ **Important:** New or progressive unilateral hearing loss, persistent down-beat nystagmus without torsion, or any focal neurological sign mandates MRI and reconsideration of the diagnosis — these features are not attributable to vestibular migraine alone [12,19].

VI. Differential Diagnosis

Because VM has no confirmatory test, it remains substantially a diagnosis of exclusion, and the vestibular physician must hold a structured differential spanning peripheral and central causes of recurrent vertigo [3,12,14]. The most clinically demanding distinction is from Ménière's disease, with which VM both mimics and genuinely coexists; Figure 4 illustrates the overlap. Table 4 sets out the principal differentials and their distinguishing features.

Table 4. Differential diagnosis of vestibular migraine and key distinguishing features.

Differential diagnosis	Key distinguishing features
Ménière's disease	Vertigo 20 min–12 h with fluctuating low-frequency SNHL, tinnitus and aural fullness; progressive cochlear loss on audiometry — unlike VM, where hearing recovers between attacks. Genuine coexistence occurs in a substantial minority [14,21].
Benign paroxysmal positional vertigo (BPPV)	Brief (<1 min) position-triggered vertigo with characteristic Dix–Hallpike nystagmus and no migraine features; VM positional vertigo lasts longer and is less consistently position-locked. Migraineurs have higher BPPV risk [12,14].
Vestibular neuritis	Acute, monophasic vertigo lasting days, positive head-impulse to the

	affected side, no migraine features; imbalance resolves over weeks [12].
TIA / posterior-circulation stroke	Older patients with vascular risk; sudden vertigo with focal deficits, lacking migraine features; MRI may show posterior-circulation infarct [12,16].
Persistent postural-perceptual dizziness (PPPD)	Chronic daily dizziness >3 months, worse upright and in complex visual environments; commonly coexists with VM and needs vestibular rehabilitation and serotonergic therapy [27].
Panic / anxiety disorder	Short episodes with autonomic symptoms and light-headedness rather than true vertigo; no nystagmus or aura; frequently comorbid with VM and amplifies symptoms [25,26].
Migraine with brainstem aura	Vertigo as an aura with additional brainstem features preceding headache; major overlap with VM and largely shared management [4,12].
Episodic ataxia / central disorders (e.g. MS)	Vertigo with other neurological signs, characteristic MRI; channelopathies may share calcium-channel mechanisms with migraine phenotypes [9,12].

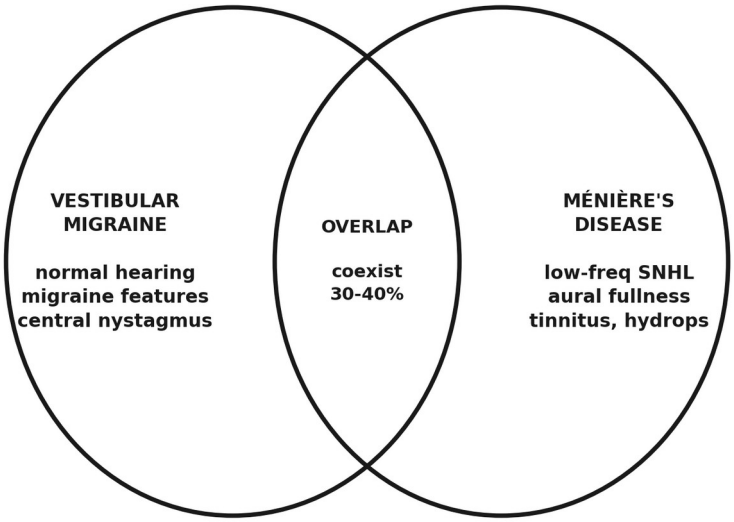

Figure 4. The vestibular migraine–Ménière's disease overlap.
Source: Adapted from Lempert and von Brevern [14] and Radtke et al. [21].

The VM–Ménière's relationship deserves particular attention. Migraine prevalence is higher among Ménière's patients than in the general population, transient cochlear symptoms occur in VM, and a subset of patients satisfy criteria for both conditions over time [14,21]. Longitudinal data show that some VM patients slowly develop mild bilateral low-frequency sensorineural hearing loss, further blurring the boundary [21]. In practice the distinction rests on the audiometric trajectory — fixed, progressive low-frequency loss favours Ménière's — and on the presence of central-type interictal nystagmus, which favours VM [19,21].

□ **Clinical Pearl:** When VM and Ménière's cannot be cleanly separated, treat the dominant, most disabling syndrome first and review the audiometric trajectory over time — a fixed progressive low-frequency loss is the single most useful tie-breaker toward Ménière's [14,21].

VII. Management — Lifestyle, Acute and Preventive Therapy

Management mirrors migraine practice adapted to the vestibular symptom, and follows a step-up/step-down structure: trigger control and education for all, acute treatment for attacks, and preventive medication for frequent or disabling disease, escalating to referral and novel therapy where refractory [12,14,28]. It is essential to be candid about the evidence base — randomised, placebo-controlled data in VM are sparse and of low certainty, so therapy is individualised to comorbidity and tolerability rather than driven by high-grade trials [28,29,31]. Figure 5 shows the escalation pathway.

Non-pharmacological measures

Patient education that VM is a real, manageable migraine variant is itself therapeutic. Trigger identification and lifestyle regularity — consistent sleep, hydration, regular meals, moderated caffeine and alcohol, and a vertigo/headache diary — are first-line for every patient [14]. Vestibular rehabilitation is valuable for motion sensitivity and PPPD overlap and, when introduced once attacks are reasonably controlled, improves both dizziness handicap and — in patients with coexisting headache — the headache burden [35]. Low-risk migraine adjuncts such as magnesium and riboflavin are commonly used; cognitive-behavioural therapy and graded exercise help particularly where anxiety or PPPD features dominate [14,27]. The Cochrane review of non-pharmacological prophylaxis found only low- or very-low-certainty evidence for probiotics, CBT and vestibular rehabilitation, underscoring that these measures are reasonable and low-risk rather than proven [30].

Acute (abortive) treatment

Acute treatment aims to relieve vertigo, nausea and headache, and is reserved for attacks rather than daily use to avoid tolerance and rebound [38]. Vestibular suppressants and antiemetics provide symptomatic relief; migraine-specific abortives such as triptans may help when headache is prominent, although VM-specific evidence is limited [12,38]. Table 5 summarises the acute options.

Table 5. Acute (abortive) treatment options in vestibular migraine.

Class / agent	Typical use	Cautions
Vestibular suppressants (e.g. antihistamines; short-course benzodiazepines)	Reduce acute vertigo and motion sensitivity during severe attacks	Sedation; avoid driving; restrict to attacks — daily use impairs central compensation and risks dependence [38].
Antiemetics (e.g. promethazine, prochlorperazine, ondansetron)	Control nausea and vomiting; some have adjunct vestibular benefit	Extrapyramidal effects with dopamine antagonists; QT considerations with ondansetron [12].
NSAIDs (e.g. ibuprofen, naproxen)	First-line analgesia when headache accompanies vertigo	Standard gastrointestinal and renal precautions [12].
Triptans (e.g. rizatriptan, sumatriptan)	Considered when migrainous headache is prominent	VM-specific evidence limited; avoid with vascular risk factors [12,28].

Preventive (prophylactic) treatment

Prophylaxis is indicated for frequent, prolonged or disabling attacks. No agent is VM-specific; selection follows migraine-prevention principles and is matched to comorbidity [12,28]. The trial evidence is instructive and sobering: the PROVEMIG randomised trial of metoprolol versus placebo was terminated for poor recruitment and showed no benefit over placebo, with a marked decline in attacks in both arms [34]; a randomised comparison found propranolol and venlafaxine equally effective at reducing attacks, with venlafaxine superior for depressive symptoms [32]; and a randomised trial of flunarizine reduced vertigo frequency and severity relative to non-specific treatment [33]. Two Cochrane reviews concluded that evidence for both beta-blockers and calcium-channel blockers is of low or very low certainty, and an earlier Cochrane review found no eligible placebo-controlled RCTs at all [29,31]. A systematic review and

meta-analysis of the broader literature reported improvement across all drug classes but could not establish a preferred agent owing to heterogeneity [28]. Table 6 lists the commonly used preventives.

Table 6. Common preventive medications for vestibular migraine.

Medication (class)	Typical dose range	Key considerations and cautions
Propranolol (β-blocker)	40–160 mg/day (divided)	Good with hypertension/anxiety; avoid in asthma/bradycardia; equal to venlafaxine in an RCT [32].
Metoprolol (β-blocker)	50–100 mg/day (ER)	Cardioselective; PROVEMIG showed no benefit over placebo but both arms improved [34].
Amitriptyline (TCA)	10–50 mg nocte	Helpful with insomnia/tension-type pain; anticholinergic effects; caution in older adults [38].
Venlafaxine (SNRI)	75–150 mg/day (XR)	Useful with anxiety/depression or PPPD overlap; monitor BP; taper to avoid withdrawal [27,32].
Topiramate (anticonvulsant)	50–100 mg/day	Titrate from 25 mg; hydrate; cognitive effects/paraesthesiae; avoid in pregnancy [28,38].
Valproate (anticonvulsant)	500–1000 mg/day	Reserve when others fail; teratogenic; monitor LFTs/FBC; weight gain/tremor [28,38].
Flunarizine (Ca-channel blocker)	5–10 mg nocte	Reduced vertigo in an RCT [33]; sedation, weight gain, depression; availability varies by country.
CGRP mAbs (e.g. erenumab)	Monthly injection	Off-label; observational cohorts report marked vertigo reduction [36]; reserve for refractory disease.

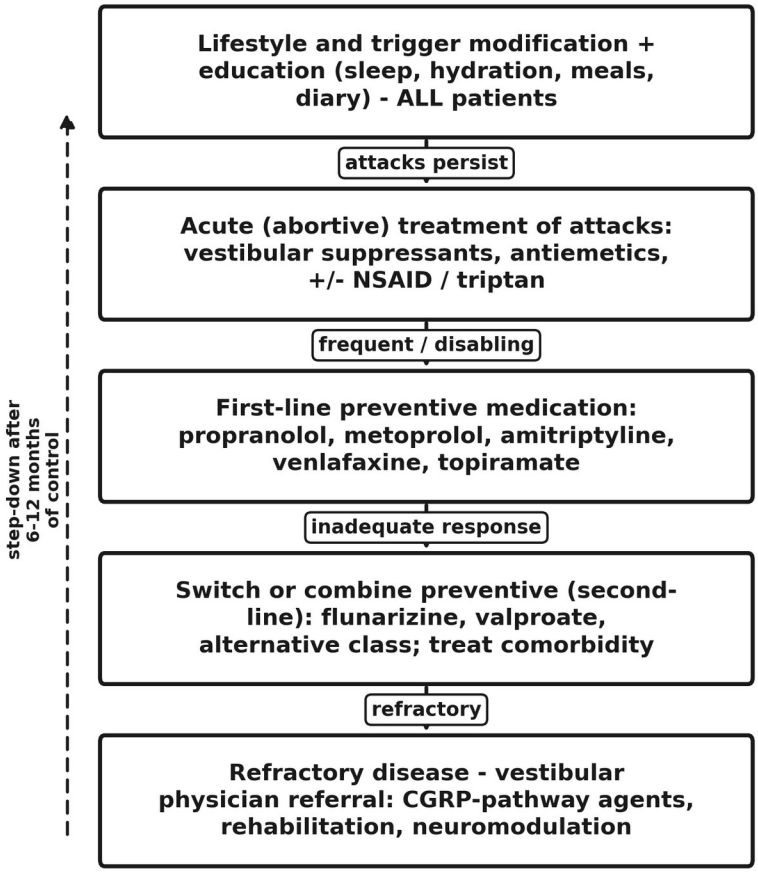

Figure 5. Step-up/step-down management algorithm for vestibular migraine.
Source: Adapted from Byun et al. [28], Webster et al. [29], and Lempert and von Brevern [14].

An adequate preventive trial is judged at roughly 2–3 months at target dose, with a ≥50% reduction in attack frequency or severity as the usual success benchmark; effective prophylaxis is generally maintained for 6–12 months of stable control before a cautious, single-agent-at-a-time taper [28,38]. Drug choice is best matched to the patient — propranolol for hypertension or anxiety, venlafaxine for anxiety, depression or PPPD overlap, topiramate for the overweight patient, amitriptyline for insomnia — and comorbidities should be treated in parallel [27,32,38].

Monitoring is structured around the vertigo/headache diary, which converts a fluctuating, subjective illness into trackable attack frequency, duration and severity and is the principal instrument for judging treatment response [14]. Where a single agent at an adequate dose fails, a measured switch to an alternative class is preferred before combining agents; rational combinations pair drugs with complementary mechanisms and comorbidity targets — for example a low-dose beta-blocker with a serotonergic agent where anxiety coexists — undertaken cautiously and one change at a time [28,32]. Throughout, the diagnosis should be periodically re-examined: a patient whose pattern drifts toward a fixed unilateral deficit, progressive hearing loss, or near-daily non-spinning dizziness has either declared a second diagnosis or developed a PPPD overlay, each of which redirects management [21,27].

□ **Clinical Insight:** The large placebo response and trial failures (notably PROVEMIG) mean lifestyle regularity and a confident explanation often do much of the therapeutic work. Match the preventive to

the comorbidity, give it a fair 2–3 month trial, and de-escalate after sustained control [28,32,34].

VIII. Refractory Disease, Novel Therapies and Special Populations

When attacks persist despite sequential adequate trials, the vestibular physician should re-examine the diagnosis, reinforce lifestyle measures, treat comorbid anxiety, depression and PPPD, and consider combination prophylaxis across drug classes [27,28]. CGRP-pathway agents — monoclonal antibodies and gepants — are increasingly used off-label by extrapolation from migraine, and a prospective observational cohort reported that around 90% of refractory VM patients achieved at least a 50% reduction in vertigo frequency on anti-CGRP antibodies [36]. Neuromodulation devices evaluated in migraine are of interest but remain unproven in VM, and acupuncture has supportive evidence for episodic migraine that is sometimes extended to VM care pathways despite sparse VM-specific data [39]. These options should be framed honestly as promising rather than established.

Special populations warrant tailored care. In women, menstrual clustering may justify short-term perimenstrual prophylaxis, and pregnancy contraindicates several standard agents (notably valproate and topiramate), shifting emphasis toward non-pharmacological measures [14,38]. In older patients, vestibular suppressant and anticholinergic burden must be minimised because of falls and cognitive risk, and coexisting presbyvestibulopathy or BPPV should be actively sought [38]. Across all groups, recognition of coexisting PPPD changes management substantially, adding vestibular rehabilitation and serotonergic agents to migraine-directed therapy [27].

□ **Clinical Pearl:** Before labelling VM refractory, look for an untreated comorbidity — anxiety, depression or PPPD — and an inadequate preventive trial. Anti-CGRP therapy is a rational next step in genuinely refractory disease but remains off-label [27,36].

IX. Prognosis, Natural History and Comorbidity

VM typically follows a relapsing–remitting course over many years, behaving as a chronic disorder akin to migraine rather than a self-limiting condition. In a nine-year follow-up cohort of definite VM, approximately 87% still reported recurrent vertigo, with frequency reduced in just over half, increased in around a third, and unchanged in the remainder [21]. Impact remained severe in a meaningful minority, and interictal ocular-motor abnormalities and mild bilateral low-frequency hearing loss accrued slowly in some patients [21].

With trigger management and appropriate prophylaxis, most patients achieve substantial, durable improvement and many reach a ≥50% reduction in attack burden, restoring near-normal function [28,32]. Spontaneous improvement can occur, sometimes after middle age or menopause, but relapse on stopping medication is recognised, supporting maintenance of effective prophylaxis for 6–12 months before a careful taper [14,38]. A subset evolve toward near-daily dizziness best understood as VM with superimposed PPPD, requiring multimodal therapy [27]. Importantly, VM is not life-threatening and does not cause neurodegeneration or progressive profound deafness; the principal hazards are secondary injury from falls or driving during attacks, and the considerable quality-of-life burden of unpredictable vertigo and its psychiatric comorbidity [7,25]. Screening for and treating anxiety and depression frequently improves the vestibular symptom itself and is integral to good outcomes [25,26].

□ **Key Point:** VM is usually chronic and relapsing — most patients still have attacks years later — but it is highly manageable, and treating comorbid anxiety, depression and PPPD often improves the vertigo as much as migraine-directed therapy does [21,25,27].

X. Guidelines, Controversies and Future Directions

The defining guidance for VM is the Bárány Society/IHS criteria, stable since 2012 and reaffirmed in 2022, alongside their incorporation into ICHD-3 and the ICVD framework [2,3,4,5,43]. Several genuine controversies remain. The first is the boundary with Ménière's disease: shared episodic vertigo, high migraine prevalence in Ménière's, and overlap phenotypes lead some to propose common mechanisms, yet the conditions remain separate diagnoses and the optimal approach to overlap cases is unsettled [14,21]. The second is the relationship between VM and migraine with brainstem aura, which ICHD treats as distinct despite substantial clinical overlap and largely shared management [4,12].

The third and most consequential controversy is therapeutic. No regimen is proven superior; practice varies geographically (flunarizine common in Europe, propranolol or topiramate in the United States), the PROVEMIG failure and the Cochrane reviews highlight how thin the placebo-controlled evidence is, and off-label CGRP-pathway agents are being adopted ahead of definitive VM-specific trials [29,31,34,36]. The absence of a validated biomarker invites both under- and over-diagnosis and motivates research into attack-phase vestibular and imaging signatures [13,15]. Practical priorities for the field are adequately-powered treatment trials with standardised outcomes, clarification of inner-ear versus central mechanisms, and multidisciplinary care pathways that bridge neurology and otology — the structural gap that left VM historically under-studied [12,15,28]. For the patient in clinic today, however, the message is pragmatic and optimistic: a confident criteria-based diagnosis, honest discussion of the evidence, comorbidity-matched prophylaxis and attention to anxiety, depression and PPPD deliver substantial improvement in the large majority [3,28,42].

□ **Clinical Insight:** The criteria are stable and the diagnosis is reliable; the open questions are mechanistic and therapeutic. Until better trials arrive, individualised, comorbidity-led management remains the standard of care [3,28,29].

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