VESTIBULAR MIGRAINE CHEAT SHEET

Vestibular Migraine — Cheat Sheet for Vestibular Physicians

Five episodes, five minutes to seventy-two hours, a migraine history, migraine features in half — and nothing else fits.

► Why vestibular migraine matters

The commonest cause of recurrent spontaneous vertigo in adults and the leading central cause of episodic vestibular symptoms in a dizziness clinic. Prevalence just under 1% (up to 2.7% in a US survey); onset mid-life; female 3:1–5:1; 7–11% of dizziness-clinic patients. Ménière's overlap 30–40%. Markedly under-diagnosed — most patients consult repeatedly before the diagnosis is made — yet highly treatable: the diagnostic standard separates a manageable trajectory from years of disability.

► Indications — when this pathway fits

► Recurrent vestibular symptoms 5 min – 72 h, moderate/severe, in a patient with a migraine history.
► Migraine features (migrainous headache, photophobia and phonophobia, or visual aura) in ≥50% of episodes — or stereotyped attacks WITHOUT headache (acephalgic).
► Preserved hearing between attacks; prominent visual-motion and head-motion sensitivity.
► Distinguish from Ménière's, BPPV, vestibular neuritis, posterior-circulation TIA/stroke, PPPD.

► Mechanism — why vestibular migraine happens

Layer	Mechanism	Clinical relevance
Trigeminovascular activation	CGRP and substance P at perivascular labyrinthine afferents; trigeminal–vestibulocochlear reflex → neurogenic inflammation.	Shared pain–vestibular pathway; CGRP blockade is therapeutically active in migraine and is being extended to VM.
Central vestibular network	Brainstem vestibular nuclei reciprocally linked to locus coeruleus, raphe magnus and periaqueductal grey.	Transient dysfunction gates vestibular signals abnormally — episodic vertigo without a fixed lesion.
Thalamo-cortical integration	Vestibulo-thalamo-cortical multisensory mismatch on functional imaging.	Explains visually-induced and head-motion-induced symptoms and central-type nystagmus in attacks.
Cortical spreading depression / genetics	Reversible cortical wave; familial clustering; calcium-channel signalling (e.g. CACNA1A).	Reversible, self-terminating attacks; polygenic susceptibility with a peripheral contribution in some.

Pearl — *Conceive VM as migraine expressed through vestibular circuits — the same biology that produces photophobia produces motion and visual sensitivity.*

► Diagnostic criteria — Bárány Society / IHS 2012 (reaffirmed 2022)

Tier	Required features
Definite VM	(1) ≥5 episodes of moderate/severe vestibular symptoms 5 min – 72 h. (2) Current or prior migraine (ICHD). (3) Migraine features in ≥50% of episodes. (4) Not better explained by another disorder.
Probable VM	≥5 vestibular episodes 5 min – 72 h, with only ONE of (migraine history) OR (migraine features) present. Responds to the same treatments.

Pearl — *There is no confirmatory laboratory or imaging test — the diagnosis is the pattern. Acephalgic attacks are the single greatest reason VM is missed; headache is not required.*

► Investigations — to exclude, not to confirm

Test	Purpose	When to order
Pure-tone audiometry	Usually normal; excludes Ménière's and other cochlear disease.	First presentation; repeat if auditory symptoms develop.
VNG / caloric, vHIT, VEMP	Often normal or mildly abnormal; exclude fixed vestibular loss.	Atypical features, unilateral signs, or pre-treatment baseline.
MRI brain ± IAM (gadolinium)	Exclude infarct, demyelination, cerebellopontine-angle tumour.	First presentation or any atypical/focal feature.
Bedside oculomotor (in attack)	Direction-changing or persistent positional nystagmus favours a central origin.	Whenever a patient can be examined during an acute attack.
Bloods (selective)	Exclude systemic contributors to dizziness.	Guided by the differential, not by VM itself.

Pearl — *Investigation excludes; it does not confirm. Migraine-related white-matter spots on MRI do not explain episodic vertigo and must not be over-interpreted.*

► Differential diagnosis — high-yield mimics

Mimic	Key distinguishing features

Ménière's disease	Fluctuating low-frequency SNHL, tinnitus, aural fullness; fixed progressive loss on audiogram. Genuine coexistence 30–40%.
BPPV	Brief (under 1 min) position-triggered vertigo; Dix–Hallpike nystagmus; no migraine features.
Vestibular neuritis	Single prolonged attack over days; positive head-impulse to the affected side; monophasic.
TIA / posterior-circulation stroke	Vascular risk; focal deficits; no migraine features; MRI may show infarct.
PPPD	Chronic daily dizziness over 3 months, worse upright and in busy visual environments; commonly overlaps VM.
Migraine with brainstem aura	Vertigo as an aura with other brainstem features preceding headache; largely shared management.

► **Red flags —** New progressive unilateral hearing loss · persistent down-beat nystagmus without torsion · any focal neurological sign · sudden severe (thunderclap) headache · first presentation with vascular risk in an older patient. Each warrants MRI and reconsideration of the diagnosis before further empirical therapy.

► Management — stepwise, step-up / step-down

Tier	Intervention	Practice principles
Lifestyle & triggers	Regular sleep, hydration, regular meals; moderate caffeine/alcohol; trigger diary; vestibular rehab for visual-motion sensitivity.	First-line for all; often sufficient alone. CBT and treating anxiety help.
Acute attack	Vestibular suppressants, antiemetics; NSAID or triptan when headache prominent.	SHORT-TERM only — daily suppressants impair central compensation.
Preventive (1st-line)	Propranolol, metoprolol, amitriptyline, venlafaxine, topiramate.	Match to comorbidity; trial 2–3 months at target; aim ≥50% reduction.
Preventive (2nd-line)	Flunarizine, valproate, alternative class or combination.	Treat comorbid anxiety, depression and PPPD in parallel.
Refractory	Off-label CGRP-pathway agents; vestibular physician referral; neuromodulation.	Re-examine the diagnosis before labelling refractory.

Pearl — *Evidence is low-certainty — PROVEMIG (metoprolol) was negative with a large placebo response, and propranolol equalled venlafaxine in an RCT. Maintain an effective preventive 6–12 months, then taper.*

► Preventive medications at a glance

Agent (class)	Typical dose	Key cautions / when to choose
Propranolol (β-blocker)	40–160 mg/day (divided)	Good with hypertension or anxiety; avoid in asthma/bradycardia; equalled venlafaxine in an RCT.
Amitriptyline (TCA)	10–50 mg nocte	Useful with insomnia or tension-type pain; anticholinergic effects; caution in older adults.
Venlafaxine (SNRI)	75–150 mg/day (XR)	First choice with anxiety, depression or PPPD overlap; monitor BP; taper to avoid withdrawal.
Topiramate (anticonvulsant)	50–100 mg/day	Titrate from 25 mg; paraesthesiae/cognitive effects; weight loss; avoid in pregnancy.
Flunarizine (Ca-channel blocker)	5–10 mg nocte	Reduced vertigo in an RCT; sedation, weight gain, low mood; availability varies by country.
Candesartan / CGRP mAb	8–16 mg/day; monthly injection	Reasonable alternatives; off-label CGRP-pathway agents reserved for refractory disease.

► Counselling and follow-up

► Chronic relapsing course — most still have attacks years later — but highly manageable; reassure it is not a stroke and does not damage hearing or brain.
► Anxiety, low mood and PPPD are common and treatable; raise them early and treat in parallel.
► Keep a trigger/episode diary; protect sleep, meals, hydration; build graded activity rather than avoidance.
► Driving: defer in the active phase; resume when episodes are controlled.
► Review to judge preventive response at 2–3 months; de-escalate after sustained control.

► Overlap, comorbidity and special situations

VM and Ménière's coexist in up to 30–40%; the tie-breaker is the audiometric trajectory — a fixed, progressive low-frequency loss favours Ménière's, while preserved hearing with central-type interictal nystagmus favours VM. For menstrual-related VM, short perimenstrual mini-prophylaxis can help. In pregnancy, avoid valproate and topiramate and lean on non-pharmacological measures. In older patients, minimise vestibular-suppressant and anticholinergic burden because of falls and cognitive risk, and actively seek coexisting BPPV or presbyvestibulopathy. Recognising coexisting PPPD changes management — add vestibular rehabilitation and a serotonergic agent.

Key references — Lempert T et al. J Vestib Res 2012;22:167–72 & 2022;32:1–6 · Furman JM et al. Lancet Neurol 2013;12:706–15 · Espinosa-Sánchez JM, López-Escámez JA. Front Neurol 2015;6:12 · Byun YJ et al. Laryngoscope 2021;131:186–94 · Webster KE et al. Cochrane 2023;CD015187 · Salviz M et al. Laryngoscope 2016;126:169–74 · Bayer O et al. (PROVEMIG) Trials 2019;20:813.