

**VESTIBULAR
PAROXYSMIA
CHEAT SHEET**

Vestibular Paroxysmia — Cheat Sheet for Vestibular Physicians

Think VP whenever vertigo is brief (under a minute), stereotyped and frequent — a treatable neurovascular cross-compression of CN VIII that responds dramatically to low-dose sodium-channel blockers.

► **Definition — what vestibular paroxysmia is**

- Concept: Recurrent, brief attacks of vertigo caused by neurovascular cross-compression of the vestibulocochlear (CN VIII) nerve — the vestibular analogue of trigeminal neuralgia and hemifacial spasm.
- Mechanism in one line: A pulsatile arterial loop chronically contacts CN VIII at its root entry zone → focal demyelination → ephaptic “short-circuit” firing → sudden, stereotyped vertigo.
- Why it matters: Uncommon but highly treatable — recognising it spares patients years of mislabelling and delivers a drug response that often abolishes attacks.

► **Epidemiology — who and how common**

Feature	Figure	Note
Prevalence	Below 0.05% of general population	Rare overall; commoner where vestibular expertise concentrates
Referral share	~3% of tertiary dizziness referrals	~1 in 30 in a large neuro-otology cohort
Age of onset	Mean ~47-51 years	Proposed bimodal peak; small youth peak (congenital vessel anomaly)
Sex	No strong bias (women ~55%)	Roughly balanced across large series
Offending vessel	AICA in ~75%	PICA, vertebrobasilar branch or vein account for the rest

► **Pathophysiology — cross-compression to ephaptic firing**

- Susceptible anatomy: Long cisternal CN VIII segment (~14-19 mm) and a central-peripheral myelin transition at the root entry zone make the nerve vulnerable to a looping vessel.
- Compression: Chronic pulsatile arterial contact (usually AICA) produces focal demyelination at the contact point.
- Ephaptic transmission: Demyelinated axons allow electrical “cross-talk” between adjacent fibres → paroxysmal hyperexcitability, triggered by arterial pulsation and head movement.
- Supporting evidence: Attacks abolished by microvascular decompression; nystagmus can invert mid-attack (excitation → transient block); a minority show mild interictal vestibular deficit from the same demyelination.

► **Clinical features — attack phenomenology**

Feature	Typical pattern
Duration	Seconds to under 1 minute — brief and stereotyped
Frequency	Many attacks per day during active phases; cluster over days-weeks
Triggers	~2/3 spontaneous; ~1/4 provoked by head position/movement (not gravity-dependent like BPPV)
Hyperventilation	~1/3 develop nystagmus/vertigo after 30-60 s of hyperventilation — useful bedside sign
Nystagmus (if seen)	Horizontal or horizontal-torsional; may transiently reverse direction
Hearing	Usually normal; ~20-30% brief unilateral tinnitus/hyperacusis; mild stable HF loss in ~10-15%
Not features	LOC, diplopia, dysarthria, limb signs → think central cause

► **Diagnostic criteria — Bárány Society 2016**

Criterion	Definite VP	Probable VP
Number of attacks	≥ 10 stereotyped attacks	≥ 5 attacks
Duration	Each under 1 minute	Up to 5 minutes
Provocation	Spontaneous or head-movement facilitated	Spontaneous or movement-triggered
Phenomenology	Stereotyped within the patient	Stereotyped
Treatment response	Responds to a Na-channel blocker (e.g. carbamazepine/oxcarbazepine)	Not required
Exclusion	Not better accounted for by another diagnosis	Not better explained by another disorder

***Pearl** — “Probable” VP is a working diagnosis; a successful treatment trial reclassifies it as “definite.” MRI is deliberately excluded from the criteria — a vascular loop is supportive, never confirmatory, because contact is common in normals. Subclassify as classical (arterial), secondary (tumour/cyst/canal narrowing) or idiopathic.*

► **Investigations — exclude mimics, support the diagnosis**

Test	Finding / role
MRI (3D-CISS / FIESTA + MRA)	Mandatory — excludes schwannoma, meningioma, stroke, MS; shows vascular loop in most VP, but contact seen in up to half of normals (supportive only)
Pathogenic-contact clues	Nerve indentation, angulation/displacement at contact, unilaterality concordant with symptoms
Audiometry	Normal or mild high-frequency loss; mainly to exclude Ménière's (low-frequency loss)
Caloric testing	Mildly reduced on affected side in ~1/3
ABR	May show prolonged I-III interpeak latency on affected side (slowed CN VIII conduction)
Therapeutic trial	Low-dose oxcarbazepine/carbamazepine → dramatic fall in attacks within days-2 weeks strongly supports VP

► Differential diagnosis — high-yield mimics

Mimic	Distinguishing feature	First-line treatment
BPPV	Positional, latency + fatigable nystagmus on Dix-Hallpike	Repositioning manoeuvres
Vestibular migraine	Minutes-hours, migrainous features	Migraine prophylaxis
Ménière's disease	20 min-hours, low-freq fluctuating hearing loss, fullness	Lifestyle, diuretic, IT therapy
Superior canal dehiscence	Sound/pressure-evoked vertigo, autophony; CT + VEMP	Conservative or surgical repair
MS brainstem paroxysms	Also carbamazepine-responsive — brainstem MRI lesions are the key	Treat MS + symptomatic
Vertebrobasilar TIA	Usually minutes with brainstem features; vascular risk	Vascular risk management

► **Red flags** — *Fluctuating/progressive hearing loss, LOC, diplopia, dysarthria or limb signs are NOT VP → reconsider central or structural cause. A VP-like history with progressive cochlear signs warrants contrast-enhanced MRI to exclude schwannoma; a favourable drug response alone does not confirm VP (MS shares it).*

► Management — medical first, surgery for the refractory

Tier	Agent / action	Dose & notes
First-line	Carbamazepine	100 mg BD → 200 mg BD; relief often at 300-400 mg/day. Watch hyponatraemia, drowsiness, diplopia, enzyme induction
First-line	Oxcarbazepine	150-300 mg start → 600-900 mg/day; fewer interactions; VESPA RCT confirmed efficacy. Check serum Na
Second-line	Lacosamide	50 mg BD, titrate; no hyponatraemia, little cognitive impact; caution PR prolongation
Refractory	Microvascular decompression	Retrosigmoid, Teflon interposition; 70-80% relief; reserve for refractory, certain diagnosis + clear loop
Supportive	Education + precautions	Reassure (benign, treatable); driving/fall precautions until controlled; VR only for residual deficit

Pearl — *Choose one first-line agent, titrate over 1-2 weeks against a daily attack diary, and judge response over 4-6 weeks. Most patients achieve a 90%+ reduction. Intolerant of one drug? Switch to the other. Serum sodium at 2-4 weeks in older patients or those on diuretics. Brief attacks mean vestibular suppressants add little.*

► Prognosis

- Treatment response: ~90% reduction in attacks on carbamazepine/oxcarbazepine, maintained without tachyphylaxis.
- Long-term course: Cohorts diverge — some report persistent attacks (~72% over ~3.4 y), others ~75% attack-free at ~4.8 y with many off medication; truth likely between.
- Strategy: Treat 1-2 years then taper cautiously; ~1/4 relapse on withdrawal and simply resume. Hearing prognosis reassuring — VP does not drive progressive cochlear decline.
- Special groups: Children usually treated medically with possible spontaneous remission (rarely operated); older patients with vascular tortuosity managed conservatively given operative risk.

► Clinical pearls

- Keep VP on the differential for brief, frequent, stereotyped vertigo — the single highest-yield move.
- Hyperventilation for 30-60 s is a quick, useful bedside provocation.
- A vascular loop on MRI is supportive, not diagnostic — look for nerve indentation/angulation and symptom-concordant side.
- Carbamazepine responsiveness is shared by MS brainstem paroxysms — always pair the drug trial with brainstem imaging.
- Any change in attack character (prolongation, new hearing loss) warrants fresh assessment, not dose escalation.

Key references — Strupp M et al. Vestibular paroxysmia: diagnostic criteria. *J Vestib Res* 2016;26:409-15 · Brandt T, Dieterich M. Vestibular paroxysmia. In: *Vertigo and Dizziness*, 2nd ed. Springer 2017 · Hüfner K et al. Vestibular paroxysmia: diagnostic features and medical treatment. *Neurology* 2008;71:1006-14 · Bayer O et al. Results of the VESPA randomised, placebo-controlled trial of oxcarbazepine. *J Neurol* 2018;265(Suppl 1):291-8 · Møller MB et al. Microvascular decompression of the eighth nerve in disabling positional vertigo. *Am J Otol* 1993;14:512-6 · Dieterich M, Brandt T. Vestibular paroxysmia: an update. *J Neurol* 2025.