Vestibular Paroxysmia:

A Vestibular Physician's Deep Review of Neurovascular Cross-Compression, Diagnosis, and Management

Vestibular Medicine for Vestibular Physicians

Peripheral Vestibular Pathology — Module 2.6

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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 peripheral 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 paroxysmia (VP) is a syndrome of recurrent, brief attacks of vertigo attributed to neurovascular cross-compression of the vestibulocochlear nerve. Its conceptual roots trace to 1975, when Jannetta described “hyperactive dysfunction symptoms of the eighth cranial nerve” and linked brief episodic vertigo to vascular compression of CN VIII [1]. The same group later applied the label “disabling positional vertigo” to such cases, first in a 1984 clinical report [2] and then in a surgical series describing diagnosis and microvascular treatment [3]. Definitions in that era were broad and overlapped with other vertigo syndromes.

The modern concept was crystallised in 1994 by Brandt and Dieterich, who proposed the term “vestibular paroxysmia” and a working set of features: short attacks of seconds to minutes, frequent recurrence, possible unilateral tinnitus or hyperacusis during spells, subtle vestibulocochlear deficits between attacks, and — critically — a clear response to carbamazepine [4,5]. This emphasis on sodium-channel-blocker responsiveness foreshadowed the current diagnostic approach. Recognition accelerated after the Bárány Society published consensus diagnostic criteria in 2016 [6], and the entity is now consolidated in dedicated reviews of its natural history and treatment [7,8,9].

Historically VP was frequently mislabelled. Its subtle interictal signs and the requirement for a therapeutic trial meant that many cases described in earlier decades as atypical Menière's, benign recurrent vertigo, or simply unexplained dizziness were almost certainly VP before the entity was formally characterised [4,7]. The 1980s term disabling positional vertigo captured the same neurovascular concept but with broader, less specific boundaries [2,3]. Ongoing debate about how genuinely new the syndrome is reflects this long pre-history of under-recognition rather than any doubt about the underlying mechanism [9].

VP is uncommon. Population prevalence is difficult to establish, but a frequently cited estimate is below 0.05% of the general population [6,7]. In specialised settings it is a measurable and regularly encountered subset: a large German tertiary dizziness clinic reported VP in roughly 3% of more than 45,000 vertigo patients seen over 26 years — about one in thirty referrals to a neuro-otology service [7,10]. Epidemiological surveys of vertigo more broadly underscore how a rare but highly treatable entity can be systematically missed when brief spells are reflexively attributed to commoner diagnoses [12].

The mean age of onset lies in mid-life, approximately 47–51 years [7,10]. A second, smaller peak is proposed in youth, possibly related to congenital vascular anomalies, generating a bimodal distribution: young adults with developmental vessel anomalies and a larger middle-aged group with acquired arterial elongation and tortuosity [6,7]. Larger compilations show no strong sex bias, with women representing around 55% of cases [10]. VP is very rare in children, described only in isolated cases and small series, although paediatric disease is likely under-recognised [22]. No clear regional or ethnic predilection has been reported; the condition is simply diagnosed more often where vestibular expertise concentrates [9,11].

Incidence figures are even less secure than prevalence, constrained by diagnostic difficulty and the absence of population-based studies. Given the typical age of onset, new diagnoses presumably rise through the fourth to sixth decades in parallel with age-related arterial elongation, consistent with the acquired-tortuosity arm of the proposed bimodal model [6,7].

□ **Key Point:** Vestibular paroxysmia is the eighth-nerve analogue of trigeminal neuralgia: brief, frequent, stereotyped vertigo from pulsatile neurovascular compression of CN VIII that characteristically responds to low-dose sodium-channel blockers. It is rare in the population but accounts for roughly 3% of tertiary dizziness referrals.

Table 1. Epidemiology and clinical signature of vestibular paroxysmia at a glance [6,7,10].

Measure	Typical value	Notes
Population prevalence	Below 0.05%	Rare; precise figures limited by under-recognition
Tertiary clinic frequency	~3% of vertigo referrals	About 1 in 30 at a neuro-otology centre

Mean age of onset	47–51 years	Proposed second peak in youth (congenital vessels)
Sex distribution	~55% female	No strong sex bias in larger series
Attack duration	Seconds to under 1 minute	Up to 5 minutes permitted for “probable” VP
Offending vessel	AICA in ~75%	PICA, vertebrobasilar branches or a vein less often

II. Pathophysiology — Neurovascular Cross-Compression and Ephaptic Transmission

VP is fundamentally a neurovascular compression syndrome affecting the vestibular division of the eighth nerve, directly analogous to trigeminal neuralgia and hemifacial spasm [17,48]. The pathogenesis centres on a pulsatile vessel — usually an arterial loop — chronically contacting the nerve at or near its root entry zone (REZ), the transition between central (oligodendrocyte) and peripheral (Schwann-cell) myelin [13,14]. This transitional zone is intrinsically vulnerable to compression injury. The cisternal segment of the vestibular nerve is long (~14–19 mm), giving a looping vessel ample opportunity for contact compared with the much shorter central-myelin segments of other cranial nerves (around 4 mm for the trigeminal) [13,15].

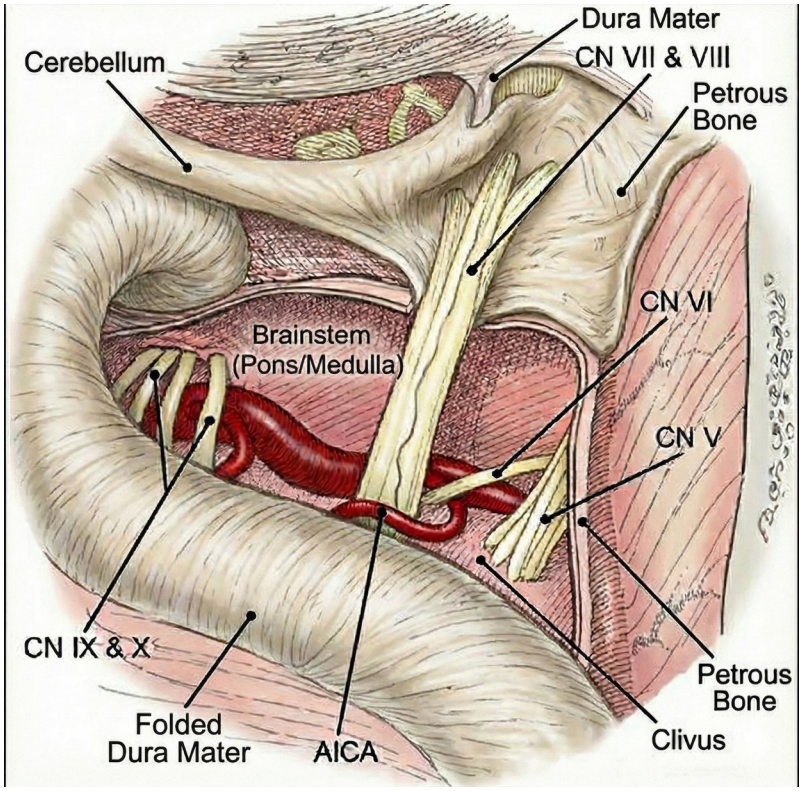

Figure 1. Microsurgical anatomy of the cerebellopontine angle, showing CN VII–VIII and the anterior inferior cerebellar artery (AICA) coursing in close relation to the nerve complex.

Source: cerebellopontine angle microsurgical anatomy, reproduced for educational use.

Chronic pulsatile compression produces focal demyelination. Demyelinated axons support ephaptic transmission, in which electrical activity crosses between adjacent fibres without normal insulation [6,18]. The result is paroxysmal hyperexcitability — brief bursts of spontaneous or mechanically triggered impulses perceived as sudden vertigo, with arterial pulsation and head movement acting as precipitants [6,16]. The mechanism mirrors trigeminal neuralgia (CN V) and hemifacial spasm (CN VII), and experimental work on the REZ confirms that the central-myelin transitional zone is the critical site of vulnerability [14,17].

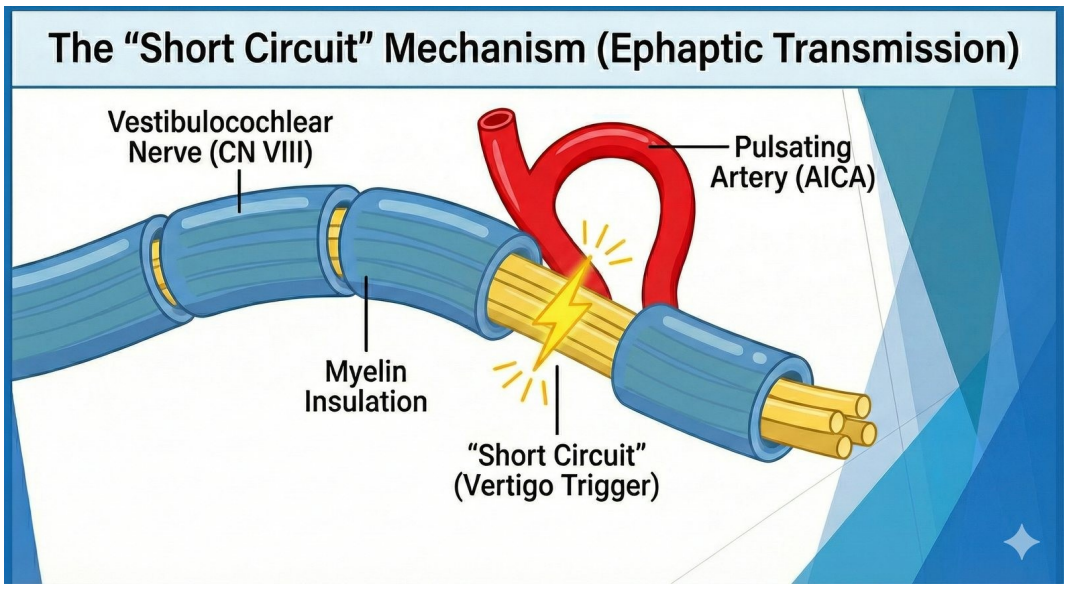

Figure 2. The “short-circuit” mechanism: pulsatile contact from AICA focally demyelinates CN VIII, allowing ephaptic cross-talk between axons that is perceived as a brief vertigo attack.

Source: Australian Dizziness Clinics educational schematic.

Several independent lines of evidence support this model. First, many patients become attack-free after microvascular decompression that physically separates vessel from nerve [3,39]. Second, the direction of nystagmus can abruptly invert within a single attack, consistent with an initial excitatory burst giving way to transient conduction block as ephaptic firing exhausts [16,18]. Third, between attacks a subset shows coexisting signs of mild vestibular deficit and irritation, in keeping with focal demyelination causing intermittent dysfunction [13,18]. Histopathological studies of analogous compression syndromes demonstrate focal demyelination precisely at the site of arterial contact [17].

The anterior inferior cerebellar artery (AICA) is by far the most common offender, identified in approximately 75% of CN VIII neurovascular conflicts; the posterior inferior cerebellar artery, a vertebrobasilar branch, or an aberrant vein account for most of the remainder [16,19]. The contacting segment is typically a tortuous loop lying close to the nerve in the cerebellopontine angle, and developmental anomalies such as an arterial fenestration may explain some paediatric and young-adult cases [7,20,38]. MRI-based anatomical classifications of the AICA–cochleovestibular nerve relationship help characterise these contacts [19].

Over time, chronic compression can extend the demyelination and produce a degree of vestibular hypofunction superimposed on the paroxysmal symptoms, explaining why a minority of patients show mild interictal imbalance alongside transient bursts of vertigo from surviving hyperexcitable fibres [6,13]. High-resolution MRI has also identified mild endolymphatic hydrops on the affected side in a subset, plausibly an epiphenomenon of nerve dysfunction rather than a primary process; unlike Menière's disease it is mild and non-progressive [6]. In summary, VP requires a susceptible anatomy, chronic pulsatile compression causing focal demyelination, and resultant ephaptic firing of vestibular fibres [6,13,15].

Intraoperative observations during decompression reinforce the model: separating vessel from nerve can abolish an irritative electrophysiological response in real time, and electron-microscopic studies of analogous syndromes demonstrate focal demyelination precisely at the contact point [16,17]. The same transitional-zone vulnerability explains why trigeminal neuralgia, hemifacial spasm and glossopharyngeal neuralgia share both mechanism and pharmacological responsiveness, situating VP within a single family of cranial-nerve hyperactivity syndromes [13,48].

□ **Clinical Insight:** The trigeminal-neuralgia analogy is the most useful teaching anchor for VP: the same transitional-zone demyelination, the same ephaptic mechanism, and the same response to sodium-channel blockers. If you can explain trigeminal neuralgia, you can explain vestibular paroxysmia in a sentence.

III. Clinical Features and Attack Phenomenology

The defining feature of VP is frequent, brief, stereotyped vertigo. Attacks typically last seconds to under a minute and are described as sudden spinning, swaying, or tilt, often recurring many times — sometimes dozens of times — per day during active phases [6,8]. The phenomenology is remarkably consistent within an individual, a feature that itself supports the diagnosis [6,7].

Triggers and provoking factors

In about two-thirds of patients attacks are spontaneous; roughly a quarter notice provocation by particular head movements or positions [6,8]. Unlike benign positional vertigo, these triggers are not strictly gravity-dependent and do not evoke the classic fatiguing positional nystagmus of BPPV; they more likely reflect mechanical modulation of the vascular contact [6,8]. A distinctive minority — around one-third — develop nystagmus or vertigo after 30–60 seconds of hyperventilation, a useful bedside provocation [21].

The clustering of attacks is itself characteristic: patients commonly report runs of spells over days to weeks interspersed with quieter periods, and the brevity of each event means that capturing one in clinic is the exception rather than the rule [7,8]. Where an attack is witnessed, the accompanying nystagmus is typically horizontal or horizontal-torsional and may transiently reverse, a behaviour not seen in BPPV or Menière's [16,18].

Auditory and neurological features

Most attacks lack prominent aural fullness or hearing change, with roughly 70–80% of patients denying cochlear symptoms during spells [6,8]. A significant minority (around 20–30%) report brief unilateral tinnitus or hyperacusis during attacks, and mild, stable high-frequency hearing reduction occurs in perhaps 10–15% — far milder than Menière's and often unnoticed by the patient [6,8,20]. Fluctuating or progressive hearing loss is not typical of VP and should prompt reconsideration. Attacks are otherwise isolated vertigo; loss of consciousness, diplopia, dysarthria, or limb symptoms are not features and point towards central causes [6,7].

Paediatric presentation

VP is rare in children but reported cases resemble adult disease, with auditory symptoms seldom reported and spells hard for children to articulate [22]. A notable paediatric scenario is a congenitally narrow internal auditory canal with a hypoplastic vestibulocochlear nerve producing VP-like attacks, often triggered by rapid head movement or exertion, responding well to low-dose carbamazepine and sometimes resolving as skull–vessel relations change with growth [22].

□ **Important:** Isolated vertigo is the rule in VP. Any attack accompanied by diplopia, dysarthria, limb weakness, numbness or loss of consciousness, or any fluctuating or progressive hearing loss, points away from VP and toward a central or alternative cause requiring imaging.

□ **Clinical Insight:** Ultra-short duration is the single most discriminating historical feature. If a patient reports stereotyped vertigo lasting seconds, recurring many times a day, with normal hearing between spells, VP should be high on the list — especially when previous repositioning manoeuvres and vestibular suppressants have failed.

IV. Diagnostic Criteria and the Bárány Society Position

Current diagnosis rests on the 2016 Bárány Society consensus criteria, which define “definite” and “probable” VP [6]. Definite VP requires at least ten stereotyped attacks of spinning or non-spinning vertigo, each lasting under one minute, occurring spontaneously or facilitated by certain head movements, with stereotyped phenomenology, no better alternative diagnosis, and a response to a sodium-channel blocker such as carbamazepine or oxcarbazepine [6,7]. Probable VP requires at least five attacks lasting up to

five minutes, spontaneous or movement-triggered, stereotyped, and not better explained by another disorder [6]. These criteria sit within the Bárány Society's international classification of vestibular disorders, which standardises symptom definitions across episodic vestibular syndromes [23].

In practice, “probable” VP functions as a working diagnosis until a treatment trial confirms it, at which point it is reclassified as “definite” [6,7]. Neuroimaging is deliberately excluded from the formal criteria: MRI may show a vascular loop contacting CN VIII and is supportive, but it is neither necessary nor sufficient because neurovascular contact is common in asymptomatic people [13,24]. After diagnosis, VP is subclassified as classical (arterial cross-compression), secondary (compression by a tumour, arachnoid cyst, or extreme canal narrowing), or idiopathic (typical picture without demonstrable compression), with idiopathic cases managed as classical if they respond to treatment [6,9].

Table 2. Bárány Society 2016 diagnostic criteria — definite versus probable vestibular paroxysmia [6].

Feature	Definite VP	Probable VP
Number of attacks	At least 10	At least 5
Attack duration	Under 1 minute	Up to 5 minutes
Onset	Spontaneous or head-movement facilitated	Spontaneous or head-movement triggered
Phenomenology	Stereotyped for the patient	Stereotyped for the patient
Treatment response	Response to sodium-channel blocker required	Not required for diagnosis
Exclusion	Not better accounted for by another diagnosis	Not better accounted for by another diagnosis

□ **Clinical Pearl:** Because the “definite” label hinges on drug response, an empirical low-dose carbamazepine or oxcarbazepine trial with a daily attack diary is both treatment and confirmatory test — no other vertigo syndrome responds so consistently to a sodium-channel blocker.

V. Investigations: Imaging, Audiovestibular Testing and the Diagnostic Trial

Investigation in VP serves two purposes: to document subtle eighth-nerve involvement and, more importantly, to exclude mimics. Between attacks the neuro-otological examination is often normal or only subtly abnormal; about 20% show a mild unilateral vestibular deficit such as a positive head-impulse catch-up saccade or head-shaking nystagmus, and audiometry may reveal slight unilateral high-frequency loss — far milder than in Menière's and often subclinical [6,8,20]. If an attack is captured, a spontaneous horizontal or horizontal-torsional nystagmus is seen, occasionally reversing direction mid-attack, a finding highly specific for VP [16,18].

Provocative manoeuvres

Two bedside manoeuvres are useful. Hyperventilation for 30–60 seconds frequently induces transient nystagmus or vertigo in VP, attributed to altered conduction in the demyelinated segment triggering ectopic discharge [21]. Vigorous head-shaking may unmask an asymmetry-induced nystagmus indicating a vestibular paresis component [21]. These signs are supportive when present but their absence does not exclude VP.

Audiovestibular testing

Audiometry is typically normal or shows mild high-frequency loss, primarily serving to exclude Menière's, which produces low-frequency loss [6]. Caloric testing is mildly reduced on the affected side in about a third of patients [21]. Auditory brainstem response may show a prolonged I–III interpeak latency on the affected side, an objective — though non-specific — sign of slowed eighth-nerve conduction consistent with demyelination [21]. Normal vestibular testing is common and does not argue against the diagnosis.

Imaging

High-resolution posterior-fossa MRI is mandatory in suspected VP — not to confirm VP, but to exclude vestibular schwannoma, meningioma, brainstem stroke, or demyelination, any of which would change management [6,24]. Heavily T2-weighted sequences (3D-CISS or FIESTA) with MR angiography demonstrate a vascular loop contacting CN VIII in the large majority of VP patients [24,25]. Crucially, however, similar contacts are seen in up to half of asymptomatic individuals, so a loop is supportive only in context [24,26]. Features that raise confidence that a contact is pathogenic include nerve indentation, angulation or displacement at the contact point, and unilaterality concordant with symptoms [24,25]. Systematic reviews of vascular loops causing otological symptoms reach similarly cautious conclusions about the limited specificity of contact alone [27,28]. In paediatric or congenital cases, temporal-bone CT is added to measure the internal auditory canal and identify a narrow canal or hypoplastic nerve [22,24].

Several refinements increase confidence that a contact is pathogenic. Nerve deformity, indentation or angulation at the contact point is rarely seen in asymptomatic controls and argues for a causal relationship, whereas bilateral loops — common in normal individuals — make symptom attribution to one side harder [24,25]. Advanced techniques add further weight: diffusion tensor imaging may show reduced fractional anisotropy in the affected nerve, and high-field 7-Tesla imaging confirms contacts while still failing to resolve microscopic demyelination, a reminder that even the best imaging remains supportive rather than diagnostic [21,26].

A trial of treatment is, in effect, part of the work-up. Starting low-dose oxcarbazepine or carbamazepine and logging attack frequency typically produces a dramatic fall — more than 50% or complete cessation — within days to a couple of weeks, strongly supporting the diagnosis; failure at maximum tolerated dose after 4–6 weeks should prompt reconsideration [6,8]. Laboratory testing is non-diagnostic and is used only to exclude metabolic or thyroid contributors to dizziness [6].

□ **Key Point:** MRI in VP is for exclusion, not confirmation. A vascular loop contacting CN VIII supports the diagnosis but is seen in up to half of asymptomatic people; the diagnosis is clinical and is secured by the response to a sodium-channel blocker.

Table 3. Key investigations in suspected vestibular paroxysmia and their role [6,21,24].

Investigation	Purpose	Typical yield
High-resolution MRI / MRA (CISS/FIESTA)	Exclude tumour, MS, stroke; show neurovascular contact	Loop visible in the majority; contact also common in normals
Audiometry	Exclude Menière's; document baseline	Normal or mild high-frequency loss
Caloric testing	Detect unilateral paresis	Mildly reduced in ~1 in 3
Auditory brainstem response	Objective sign of slowed conduction	Prolonged I–III latency in some; non-specific
Hyperventilation / head-shaking	Provoke ephaptic or asymmetry nystagmus	Positive in a minority; supportive when present
Temporal-bone CT (paediatric)	Measure IAC; narrow canal / hypoplastic nerve	Reserved for congenital or paediatric cases
Therapeutic trial (CBZ/OXC)	Confirm diagnosis via response	Marked reduction within days to weeks

VI. Differential Diagnosis

Because VP is brief, episodic, and leaves few persistent signs, several disorders mimic it and must be excluded. Duration, trigger pattern, accompanying symptoms, and treatment response are the most useful discriminators [6,7]. BPPV produces positionally triggered vertigo with characteristic latency, crescendo–decrescendo and fatigability on Dix–Hallpike, and responds to repositioning rather than sodium-channel blockers [29]. Vestibular migraine attacks usually last minutes to hours with migrainous features and do not consistently respond to carbamazepine [30]. Menière's disease produces 20-minute-

to-hours attacks with fluctuating low-frequency hearing loss, roaring tinnitus and aural fullness [31]. Superior canal dehiscence causes sound- or pressure-evoked brief vertigo with autophony, confirmed on CT and VEMP [32].

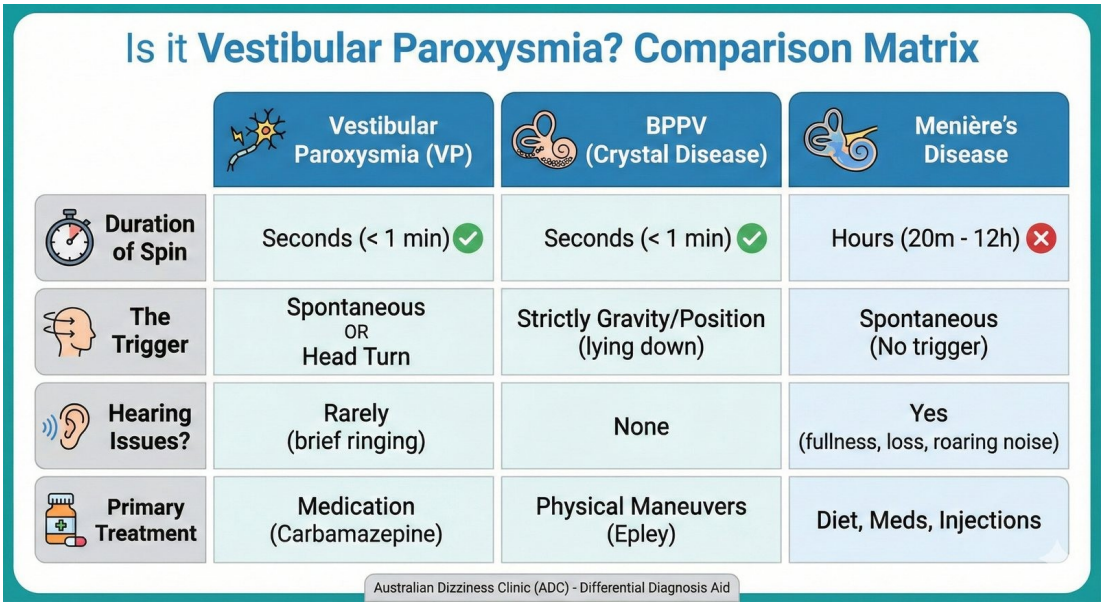

Figure 3. Rapid comparison matrix contrasting vestibular paroxysmia with BPPV and Menière's disease across duration, trigger, hearing involvement and primary treatment.

Source: Australian Dizziness Clinics educational schematic.

Central and vascular mimics deserve particular care. Paroxysmal brainstem attacks in multiple sclerosis can be brief, positional, and — because the mechanism is also ephaptic — carbamazepine-responsive, so accompanying signs and brainstem MRI lesions are the key discriminators [30,33]. Vertebrobasilar TIAs cause abrupt vertigo but usually last minutes with additional brainstem features, and the Bárány vascular-vertigo criteria help frame the work-up [33,34]. Rarer mimics include vestibular epilepsy, rotational vertebral-artery (bow-hunter) compression, orthostatic hypotension, and panic or persistent postural-perceptual dizziness; each is distinguished by pattern and associated features [6,7]. MRI excludes MS and central causes, vestibular testing separates BPPV and Menière's, and careful history clarifies the rest [6,24].

Two practical pitfalls deserve emphasis. First, multiple sclerosis brainstem paroxysms share VP's carbamazepine responsiveness, so a favourable drug response does not by itself confirm VP and must be interpreted alongside dedicated brainstem imaging [30,33]. Second, an intracanalicular vestibular schwannoma can itself generate paroxysmal vertigo through intermittent nerve irritation, so a VP-like history with progressive cochlear signs should lower the threshold for contrast-enhanced imaging rather than an empirical drug trial [24,31].

Several rarer entities round out the differential. Vestibular (epileptic) vertigo from temporal or parieto-insular seizures can produce seconds-long spells, distinguished by altered awareness, experiential auras or EEG changes [6]. Rotational vertebral-artery (bow-hunter) compression provokes vertigo only at end-range head rotation and is confirmed on dynamic angiography [24]. Orthostatic hypotension causes brief light-headedness on standing rather than true spinning, and persistent postural-perceptual dizziness produces continuous, situationally exacerbated unsteadiness unlike the discrete spells of VP [7]. Each may coexist with VP and is separated by pattern and associated features rather than by any single test [6,7].

Table 4. Differential diagnosis of vestibular paroxysmia — key distinguishing features.

Differential	Distinguishing features versus VP
BPPV [29]	Strictly positional; latency, crescendo and fatigability; responds to repositioning
Vestibular migraine [30]	Minutes to hours; migrainous features; inconsistent carbamazepine response

Menière's disease [31]	20 min to hours; fluctuating low-frequency loss, roaring tinnitus, fullness
Superior canal dehiscence [32]	Sound/pressure-evoked; autophony; CT dehiscence and low VEMP thresholds
MS brainstem paroxysms [33]	Other brainstem signs; MRI plaques; may also respond to carbamazepine
Vertebrobasilar TIA [34]	Minutes; vascular risk factors; additional brainstem symptoms
Vestibular epilepsy [6]	Altered awareness, aura, post-ictal features; EEG changes
Orthostatic / PPPD [7]	Postural light-headedness or persistent daily dizziness, not seconds-long spins

VII. Medical Management — Pharmacotherapy and Supportive Care

Management combines symptomatic pharmacotherapy, supportive measures, and patient education, with surgery reserved for refractory disease. Because the mechanism parallels trigeminal neuralgia, sodium-channel blockers are the treatment of choice, stabilising the inactivated state of voltage-gated sodium channels in demyelinated axons and suppressing ectopic firing [6,36]. The vast majority of patients respond, often with a 90%-plus reduction in attack frequency, making VP one of the more gratifying chronic vertigo syndromes to treat [6,8].

First-line agents

Carbamazepine has been first-line since the original descriptions and is effective at doses well below those used in epilepsy — commonly 100 mg twice daily titrated towards 200 mg twice daily, occasionally higher, with relief often achieved around 300–400 mg per day [4,6]. Oxcarbazepine is widely used as an equivalent first-line option with fewer interactions, started at 150–300 mg and titrated to 600–900 mg per day [6,8]. A randomised double-blind, placebo-controlled crossover trial of oxcarbazepine (the VESPA trial) confirmed efficacy, with a marked fall in attack frequency, while also highlighting tolerability issues including dizziness, fatigue and hyponatraemia [35]. Many patients intolerant of one drug do well on the other, so having both options is valuable [6,8].

Carbamazepine warrants some vigilance: it can cause dose-related drowsiness, diplopia and ataxia during titration, hyponatraemia — particularly in older patients — and, rarely, rash or blood dyscrasias, and as a potent enzyme inducer it interacts with oral contraceptives, anticoagulants and many other drugs [6,36]. Baseline and interval full blood count and serum sodium are reasonable, and starting low with slow up-titration mitigates most early side effects [6,8]. Oxcarbazepine shares the hyponatraemia risk but has fewer interactions, which is partly why many clinicians prefer it despite a comparable efficacy profile [35,36].

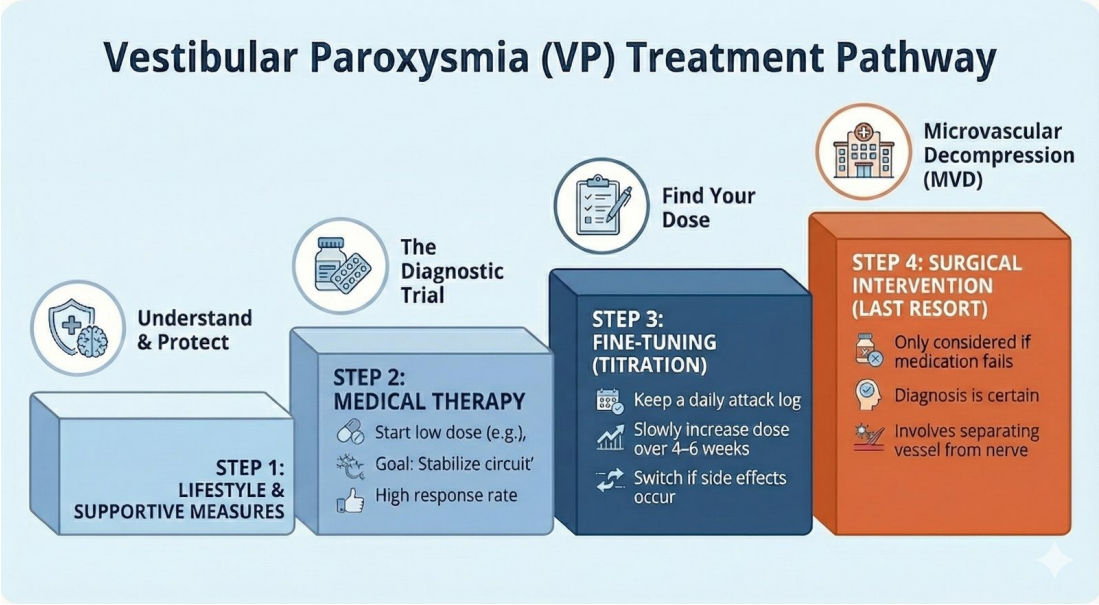

Figure 4. Stepwise treatment pathway for vestibular paroxysmia — from education and supportive measures through titrated medical therapy to microvascular decompression as a last resort.

Source: Australian Dizziness Clinics educational schematic.

Second-line and refractory options

When carbamazepine and oxcarbazepine fail or cannot be tolerated, other sodium-channel-active agents are tried. Lacosamide is emerging as effective and well tolerated, typically 50 mg twice daily increasing as needed, with no hyponatraemia and little cognitive impact, although PR-interval prolongation warrants caution [8,36]. Lamotrigine and phenytoin have anecdotal support only, and gabapentinoids — calcium-channel rather than sodium-channel agents — are not first-line but are occasionally tried off-label [6,8]. In children, very low weight-based carbamazepine doses are usually effective, and because paediatric disease may remit with growth the strategy is to treat for one to two years and then attempt withdrawal [22].

Practically, clinicians start one first-line agent, titrate slowly to limit side effects, and judge response over a 4–6 week therapeutic trial against a daily attack diary; partial responders escalate, non-responders switch agent or class, and a serum sodium check at 2–4 weeks is prudent in older patients or those on diuretics [6,8]. Because attacks are so brief, vestibular suppressants such as meclizine add little and are generally unnecessary once a sodium-channel blocker is effective [6].

Supportive care

Education is therapeutic: reassuring patients that VP is benign and highly treatable relieves the considerable anxiety generated by frequent unexplained vertigo [7,9]. Identifiable triggers are avoided where feasible, simple fall-prevention and driving precautions are advised until control is achieved, and vestibular rehabilitation is reserved for those with a residual interictal deficit rather than for the paroxysms themselves [6]. Co-morbid anxiety or anticipatory avoidance may warrant psychological support, and structured follow-up monitors diaries, doses and side effects [7,9]. Validated instruments such as the Dizziness Handicap Inventory and the Vertigo Symptom Scale quantify baseline burden and treatment response in clinic and research settings [43,44].

A pragmatic sequence works well in practice: choose one first-line agent, titrate over one to two weeks against a daily diary, and judge the response over four to six weeks at a therapeutic dose. Partial responders are escalated within tolerance, non-responders are switched to the alternative first-line drug or to lacosamide, and unacceptable side effects prompt a change of class rather than abandonment of treatment [6,8,35]. A serum sodium check at two to four weeks is prudent, particularly in older patients or those on diuretics, since asymptomatic hyponatraemia is common with carbamazepine and oxcarbazepine and occasionally becomes clinically significant [6,8].

Table 5. Sodium-channel-blocker pharmacotherapy for vestibular paroxysmia [6,8,35,36].

Agent	Starting dose	Usual target	Notes
Carbamazepine	100 mg twice daily	300–800 mg/day	First-line; enzyme inducer; monitor sodium, FBC
Oxcarbazepine	150–300 mg/day	600–900 mg/day	First-line; RCT-supported; hyponatraemia risk
Lacosamide	50 mg twice daily	100–200 mg twice daily	Well tolerated; no hyponatraemia; watch PR interval
Lamotrigine / phenytoin	Low, titrated	Variable	Third-line; anecdotal evidence only
Carbamazepine (paediatric)	2–4 mg/kg/day	Weight-based, divided	Treat 1–2 years then trial withdrawal

□ **Clinical Pearl:** Start low and go slow, and confirm with a diary. A patient whose daily attacks vanish on 200–400 mg of carbamazepine has both effective treatment and a secured diagnosis; persistent attacks at maximal tolerated dose should send you back to the differential, not higher up the dose ladder.

VIII. Microvascular Decompression and Secondary Vestibular Paroxysmia

Microvascular decompression (MVD) of the vestibulocochlear nerve is the definitive surgical treatment, performed through a retrosigmoid craniotomy with interposition of a Teflon cushion to separate the offending vessel from the nerve [3,39]. Because VP is usually well controlled medically and MVD carries non-trivial risk, surgery is reserved for selected cases: a certain diagnosis with clear medication responsiveness, debilitating attacks despite optimal therapy or intolerance of effective drugs, a definite vascular compression on the symptomatic side, and an informed patient who prefers a potentially curative operation to indefinite medication [39,40,42].

Early Jannetta-era reports found improvement in roughly three-quarters of cases, and subsequent series — including a 207-patient cohort — report 70–80% achieving complete or near-complete relief, with small modern series describing durable abolition of attacks at long-term follow-up [3,39,40]. Outcomes are better when a clear loop is identified on MRI, underscoring the importance of patient selection; figures are also likely inflated by publication and selection bias [24,39]. Risks reflect the posterior-fossa approach: hearing loss (the labyrinthine artery often arises from AICA), transient or rarely permanent facial weakness, cerebellar or brainstem infarction in the low single-digit percentages, CSF leak, and aseptic meningitis [39,40]. MVD is therefore a genuine last resort for severely impaired patients who cannot be managed medically [40,42].

Secondary VP demands a different pathway. When MRI reveals a cerebellopontine-angle tumour such as a vestibular schwannoma or meningioma, an arachnoid cyst, or extreme canal narrowing, treatment is directed at the underlying lesion — resection or radiosurgery — with medication only temporising [24,41]. Such cases may combine continuous disequilibrium from sustained pressure with brief positional attacks from intermittent irritation, and addressing the lesion can relieve both. Subarcuate-artery and other unusual vascular causes have also been reported and respond to targeted decompression [41]. Where decompression succeeds, relief tends to be durable; recurrent symptoms suggest incomplete decompression or a revised diagnosis [39,40].

Among the roughly three-quarters who improve after decompression, many are cured permanently while a minority retain less frequent attacks; non-response should prompt reconsideration of whether the correct vessel was addressed or whether the original diagnosis was wrong [39,40]. Where irreversible nerve injury has already occurred, vertigo may cease only at the cost of a permanent unilateral vestibular loss, a trade-off that must be discussed explicitly during consent [40,42].

□ **Important:** Always re-interrogate the diagnosis before referring for MVD. Failure of multiple sodium-channel blockers should raise the possibility of vestibular migraine or a central mimic rather than triggering surgery, and any new progressive hearing loss or sustained imbalance mandates fresh imaging to exclude a secondary lesion.

IX. Prognosis, Recurrence and Special Populations

The natural history of VP is incompletely mapped, and published long-term outcomes diverge. A prospective follow-up of 61 medically treated patients over about 3.4 years described a persistent course, with around 72% still experiencing attacks and a similar proportion reporting continued quality-of-life impact, leading the authors to caution that complete remission is not the rule [8,9]. In contrast, a larger cohort of 146 patients followed for a mean 4.8 years was more optimistic: about 75% were attack-free at last follow-up and more than half of those had stopped medication without relapse [9,10]. An earlier series confirmed that the roughly 90% reduction in attacks achieved on carbamazepine or oxcarbazepine is maintained while treatment continues, with no tachyphylaxis [37].

Spontaneous remission is difficult to quantify because definite VP is rarely left untreated, but some probable cases settle without medication, and the bimodal age structure hints that younger patients with congenital vascular contacts may age out as collateral pathways develop, while older patients may eventually lose enough nerve function that misfiring ceases — at the cost of a degree of permanent vestibular loss [7,9]. Hearing prognosis is generally reassuring: VP does not typically drive progressive cochlear decline, and the mild tinnitus that some patients experience often eases once ephaptic firing is suppressed [6,8].

The pragmatic strategy is to treat for one to two years and then cautiously taper: patients who remain attack-free can stop and be monitored, while those who relapse — around a quarter when medication is withdrawn over five years — simply resume effective therapy [9,37]. The divergent cohorts probably reflect different populations and definitions of “attack-free”; the truth likely lies between, with a substantial subset achieving remission and another requiring ongoing therapy [8,9]. After successful MVD, relief is generally durable and recurrent compression is uncommon [39,40].

VP does not cause progressive hearing loss or permanent neurological deficit; the principal hazard is injury from falls or accidents during an attack, so driving and activity precautions apply until control is achieved [9,45]. Quality of life improves markedly with successful treatment, and validated outcome measures document this change [43,44]. Special populations warrant tailored thinking: children are usually treated medically with an expectation of possible spontaneous remission and almost never operated upon [22], while older patients with vascular tortuosity are typically managed conservatively given operative risk [45].

□ **Clinical Insight:** Counsel two messages at diagnosis: most patients achieve excellent control, and a meaningful minority can stop medication after one to two attack-free years. Frame ongoing low-dose therapy as safe and reversible rather than lifelong and fixed.

Table 6. Long-term outcomes of vestibular paroxysmia across representative cohorts [8,9,37].

Cohort / follow-up	Key outcome	Implication
61 patients, ~3.4 years [8]	~72% still had attacks; QoL impact persisted	Often an ongoing condition under treatment
146 patients, ~4.8 years [9]	~75% attack-free; over half stopped medication	Remission and drug withdrawal achievable in many
32 patients, ~2.6 years [37]	~90% reduction maintained on therapy	No tachyphylaxis; relapse usually follows taper
Withdrawal cohorts [9,37]	~25% relapse off medication over ~5 years	Re-treat on relapse; regain control
Post-MVD series [39,40]	Durable relief when a clear loop decompressed	Surgery curative in selected refractory cases

X. Guidelines, Controversies and Future Directions

Several debates shape how aggressively VP is diagnosed and treated. The first is conceptual: because "definite" VP is partly defined by drug response, the criteria risk circularity and may exclude genuine cases that cannot tolerate a trial [6]. The counterargument is pragmatic — medication response remains the most reliable available marker until an imaging or electrophysiological biomarker matures [9,18]. A second debate concerns MRI: since vascular contact is common in asymptomatic people, distinguishing causative from incidental compression is unresolved, and work continues on quantitative signs such as nerve indentation, contact angle and diffusion changes to define a "significant" compression [13,24].

Further controversy surrounds the threshold for surgery — some European centres favour earlier MVD in young patients with clear compression, others reserve it strictly for refractory disease [39,40] — and the recognition of paediatric VP, where the emergence of narrow-canal syndromes is shifting opinion towards a treatable mechanical cause in selected children [22,24]. The evidence base remains thin: only one randomised trial exists (oxcarbazepine), and calls continue for head-to-head drug trials and prospective natural-history studies [35,9]. There is also a semantic legacy, with older literature using "disabling positional vertigo" and "microvascular compression syndrome of CN VIII" for the same entity [2,17].

VP belongs to a broader family of neurovascular cross-compression syndromes, and rare patients harbour simultaneous trigeminal neuralgia, hemifacial spasm and VP, implying a generalised predisposition to close vessel–nerve relationships [17,48]. Its closest auditory relative is carbamazepine-responsive typewriter tinnitus — brief staccato bursts of unilateral tinnitus attributed to ephaptic discharge in the cochlear nerve — which may occur alone or alongside VP when both divisions of the eighth nerve are involved [46,47]. Recognising these parallels lets the clinician reach for the same diagnostic framework and drug class across the group [13,48].

Looking ahead, VP sits within a family of neurovascular cross-compression syndromes, and patients occasionally harbour simultaneous trigeminal neuralgia, hemifacial spasm and VP, pointing to a shared predisposition [17,48]. Its auditory counterpart, carbamazepine-responsive "typewriter tinnitus" from cochlear-nerve compression, is an instructive parallel and may coexist with VP [46,47]. Advanced imaging — high-field and multimodal MRI combining structural, diffusion and hydrops sequences — is refining the picture, and a composite imaging "fingerprint" may eventually reduce reliance on therapeutic trials [24,26]. For now VP remains a clinical diagnosis whose greatest practical lesson is recognition: keeping it on the differential for brief, frequent, stereotyped vertigo spares patients years of mislabelling and delivers a treatment that works [9,28].

VP can also coexist with other vestibular disorders: a patient stable on a sodium-channel blocker may still develop BPPV or vestibular neuritis, and any change in attack character — particularly prolongation or new hearing loss — warrants fresh assessment rather than dose escalation [7,45]. From a service perspective, the main barrier to good outcomes remains recognition; embedding VP in vertigo teaching and maintaining a low threshold for high-resolution imaging of recurrent, unexplained vertigo are the highest-yield system-level changes [9,28].

□ **Key Point:** Vestibular paroxysmia is rare, frequently missed, and highly treatable. The clinician's task is pattern recognition — brief, frequent, stereotyped vertigo — followed by MRI to exclude mimics and a low-dose sodium-channel-blocker trial that is at once the treatment and the diagnostic test.

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