Vestibular Migraine in Children:

Diagnosis, Pathophysiology, and Evidence-Based Management

Vestibular Medicine in Children

Topic 3 of 15

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How to Use This Review

This literature review is part of the Vestibular Medicine in Children series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, paediatricians, and emergency physicians who assess and manage children presenting with vestibular disorders.

The review is designed to be read as a deep-reference resource or used as a clinical desktop companion. It is supported by a clinical cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

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, emergencies, and critical safety points requiring immediate action.

Table of Contents

Criterion	ICHD-3 / Bárány Society requirement
A. Vestibular episodes	≥5 episodes of vestibular symptoms — spontaneous vertigo, positional vertigo, or visually induced vertigo
B. Duration	5 minutes to 72 hours
C. Migraine history	Current or previous history of migraine with or without aura (ICHD-3)
D. Migraine features	≥1 of: headache (migraine character), photophobia, phonophobia, visual aura during ≥50% of episodes
E. Exclusion	Not better accounted for by another vestibular or ICHD-3 diagnosis
Paediatric note	Duration criteria <5 min allowed in paediatric VM if other criteria met; ICHD-3 allows paediatric variants

Age group	Typical presentation	Distinguishing features
3–6 years	Episodic fright; clinging; pallor; balance loss — no headache report possible	Similar to BPVC; headache not reported at this age; family history key
6–10 years	Episodic vertigo ± headache; school absences; motion sensitivity	Headache begins to appear; photophobia elicitable; prodrome emerging
10–14 years	Classic VM presentation; dizziness ± headache; visual aura; motion sickness	Adult-equivalent features; sleep disruption; menstrual link in girls
Adolescence	Prolonged vestibular episodes; PPPD as complication; anxiety overlay	High PPPD risk if poorly managed; school disruption major impact

Diagnosis	Key differentiating features
BPVC	Age <6; seconds only; no headache; resolves spontaneously; normal family history not required
BPPV in children	Positive Dix-Hallpike; latency; fatigability; no headache; positional trigger only
Vestibular neuritis	Acute single prolonged episode; abnormal vHIT; postinfectious; no recurrence
Posterior fossa tumour	Progressive; headache; cerebellar signs; papilloedema; MRI required
Epilepsy	Altered consciousness; EEG abnormal; post-ictal phase; stereotyped brief episodes
Functional dizziness	Constant; not episodic; visual sensitivity; normal vestibular function; psychosocial overlay

VI. Differential Diagnosis: The Key Mimics

Category	Agent/strategy	Evidence in paediatric VM
Lifestyle	Sleep hygiene; regular meals; avoid triggers; stress management	Strong — trigger avoidance reduces episode frequency in children
Rescue — vestibular	Ondansetron 0.15 mg/kg (max 4 mg); dark room; hydration	Effective for acute phase; avoid regular antiemetics
Rescue — headache	NSAIDs (ibuprofen 10 mg/kg); sumatriptan nasal spray ≥12 years	Effective if headache prominent during episode
Prophylaxis — first-line	Propranolol 1 mg/kg/day (caution asthma); amitriptyline 0.5–1 mg/kg nocte	Limited paediatric RCT data; clinical evidence supports use
Prophylaxis — second-line	Topiramate 1–2 mg/kg/day; sodium valproate (avoid in girls of reproductive age)	Caution cognitive side-effects topiramate; specialist-only
VRT	Inter-episode vestibular rehabilitation; habituation exercises	Reduces vestibular sensitivity; reduces PPPD progression
Psychological	CBT for anxiety/school refusal; psychoeducation for family	High co-morbidity; often the rate-limiting factor in recovery

VII. Diagnostic Workup

VIII. Management: Acute and Preventive Strategies

Timepoint	Assessment	Action threshold
4 weeks	Episode diary review; trigger identification	Adjust lifestyle modification; confirm rescue therapy working
3 months	Frequency; school attendance; DHI-PC if applicable	If ≥2 episodes/month → prophylaxis
6 months	Response to prophylaxis; side-effects; school function	Trial off prophylaxis if 6 months episode-free
12 months	Long-term trajectory; puberty effects in adolescents	Girls: note menstrual link; adjust management plan
Any visit	Screen for PPPD: constant symptoms between attacks?	DHI-PC >40 + VSS-SF → PPPD complication; add VRT + psychology

IX. Prognosis, Comorbidities, and Long-Term Follow-Up

X. Summary and Key Clinical Takeaways

Indication	Urgency	Refer to
Diagnosis uncertain after initial workup	Routine	Vestibular physician + neurology
Prophylaxis required	Routine	Vestibular physician or paediatric neurologist
PPPD complication developing	Routine (within 4 weeks)	Vestibular physician + paediatric psychologist
Prolonged episodes >72 hours	Urgent	ED + neurology; rule out stroke/demyelination
Exam findings — abnormal	Soon	Vestibular physician; structural

VOR, nystagmus between attacks		cause to exclude

References

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I. Introduction and Diagnostic Landscape

Vestibular migraine (VM) represents the most common identifiable cause of episodic vertigo in adolescents, accounting for up to 20% of referrals to paediatric vestibular clinics [1,2]. Despite this prevalence, VM remains substantially underdiagnosed in clinical practice, particularly in emergency department settings where affected adolescents — most commonly teenage girls — are frequently labelled with anxiety, panic disorder, or functional neurological disorder. This diagnostic delay has meaningful consequences: adolescents with untreated VM are at risk of developing persistent postural-perceptual dizziness (PPPD) as a sequela of inadequately managed episodic vestibular dysfunction [3].

The formal recognition of VM as a distinct diagnostic entity has a relatively recent history. Neuhauser and colleagues described it as "migrainous vertigo" in 2001 [4], and Lempert and Neuhauser proposed the first widely-adopted diagnostic criteria in 2009 [5]. These criteria were refined and harmonised in 2012 [6] and subsequently formalised within the International Classification of Headache Disorders, Third Edition (ICHD-3) in 2018 as a classified vestibular disorder [7]. This process resolved longstanding classification confusion and enabled standardised epidemiological and clinical research.

In children, VM exists within a broader migraine continuum that begins in early childhood. Benign paroxysmal vertigo of childhood (BPVoC — PVM02) is now understood as a migraine equivalent and a precursor condition to VM: longitudinal cohort studies demonstrate that approximately 15–25% of children with BPVoC transition to definite VM during adolescence [8,9]. Clinicians assessing an adolescent with episodic vestibular symptoms should therefore enquire about a childhood history of BPVoC and a family history of migraine as key contextual features.

□ **Clinical Pearl:** VM is responsible for up to 20% of all referrals to paediatric vestibular clinics — yet is frequently missed or labelled anxiety in adolescents. A strong family history of migraine (particularly maternal) and co-occurring photophobia or phonophobia during vestibular episodes are the highest-yield discriminating features.

II. Epidemiology and Prevalence

The point prevalence of VM in children and adolescents has been estimated at approximately 2.7% in population-based studies [10]. Abu-Arafeh and Russell, in their landmark 1995 epidemiological work on childhood dizziness, identified migraine-associated vertigo as the most common cause of recurrent vertigo in children aged 5–15 years, with a marked female preponderance in the adolescent subgroup [11]. More recent systematic reviews confirm a female-to-male ratio of approximately 3:1 in adolescent VM, aligning with the well-established hormonal influences on migraine threshold.

A strong family history of migraine is present in up to 80% of children with VM [10,12]. The pattern is predominantly matrilineal: affected children most commonly have a mother or maternal grandmother with migraine. This familial aggregation reflects both the known polygenic heritability of migraine and the potential contribution of calcium and sodium channelopathies (CACNA1A, ATP1A2) that underpin familial hemiplegic migraine (FHM) and overlap with VM phenotypes [13]. Genetic counselling is appropriate for families with multiple affected members and severe phenotypes.

VM does not occur in isolation within the migraine spectrum. A substantial proportion of children with VM have comorbid migraine-equivalent disorders including cyclic vomiting syndrome (CVS) and abdominal migraine — conditions that often precede the emergence of headache-predominant migraine and VM by several years [14,15]. Recognition of these childhood migraine equivalents in the clinical history is diagnostically important and informs long-term management planning. Clinicians should specifically enquire about recurrent episodes of unexplained nausea and vomiting, periodic abdominal pain, and cyclical pallor in the childhood history.

□ **Key Point:** VM prevalence is approximately 2.7% in children, with a 3:1 female preponderance in adolescents. A matrilineal family history of migraine is present in up to 80% of cases and is the single most useful screening question in clinical practice.

III. Pathophysiology: Migraine and the Vestibular System

The pathophysiology of VM involves the convergence of cortical, trigeminovascular, channelopathy-mediated, and brainstem mechanisms that collectively dysregulate vestibular signal processing. Cortical spreading depression (CSD) — the slowly propagating wave of neuronal and glial depolarisation that underlies migraine aura — plays a central role [16]. In VM, CSD propagates beyond the occipital cortex to involve the parieto-insular vestibular cortex (PIVC), the principal cortical substrate for vestibular perception. Involvement of the PIVC and surrounding parietal association areas generates the subjective experience of vertigo and spatial disorientation that characterises VM episodes [17].

Concurrent activation of the trigeminovascular pathway results in the release of calcitonin gene-related peptide (CGRP) from trigeminal nerve terminals in the dura mater and trigeminovascular system. CGRP produces neurogenic inflammation and sensitises peripheral and central pain-processing pathways [18]. Crucially, CGRP has direct effects on vestibular end-organ and central vestibular pathways, contributing to vestibular sensitisation and the heightened vestibular hypersensitivity — including motion sensitivity and visual vertigo — that persists between attacks (the interictal period) [19]. This mechanistic insight explains why patients with VM frequently describe chronic interictal motion sensitivity that is disproportionate to their objective vestibular test findings.

Central sensitisation — the amplification of nociceptive and vestibular processing within the central nervous system — underlies several key clinical features of VM including cutaneous allodynia, vestibular hypersensitivity, and the persistence of low-grade dizziness between discrete VM episodes [20]. Central sensitisation also explains why standard vestibular function tests (vHIT, caloric testing) frequently demonstrate subtle interictal abnormalities in VM patients, even when the tests are performed during asymptomatic intervals. A normal or mildly abnormal vHIT does not exclude VM; conversely, an abnormal vHIT in the context of episodic vertigo and migraine features should prompt a definitive search for VM rather than a peripheral vestibular diagnosis [21].

Ion channelopathy represents an important aetiological dimension of VM, particularly in familial cases. Mutations in CACNA1A (encoding the P/Q-type voltage-gated calcium channel alpha-1 subunit) and ATP1A2 (encoding the Na^+/K^+-ATPase alpha-2 subunit) cause familial hemiplegic migraine (FHM1 and FHM2, respectively) and are associated with VM phenotypes in the broader migraine spectrum [13]. These channelopathies produce cortical hyperexcitability by altering the glutamatergic threshold for CSD, increasing the susceptibility to spreading depolarisation under permissive triggers.

Serotoninergic modulation is relevant both to VM pathophysiology and its pharmacological management. The 5-HT1B/1D receptor agonist mechanism of triptans — the mainstay of acute migraine therapy — suppresses trigeminovascular activation and inhibits CGRP release, providing the mechanistic basis for their efficacy in VM as well as headache-predominant migraine [22]. Brainstem vestibular nuclei receive dense serotonergic innervation from the dorsal raphe nucleus, and the locus coeruleus noradrenergic pathway modulates arousal and vestibular gain — both implicated in the prodromal phase of migraine [23].

□ **Important:** Central sensitisation explains interictal vestibular hypersensitivity in VM. A positive vHIT finding in the interictal period does NOT exclude VM — it may reflect persistent central sensitisation rather than a structural peripheral vestibular lesion. Clinical context and ICHD-3 criteria application are essential.

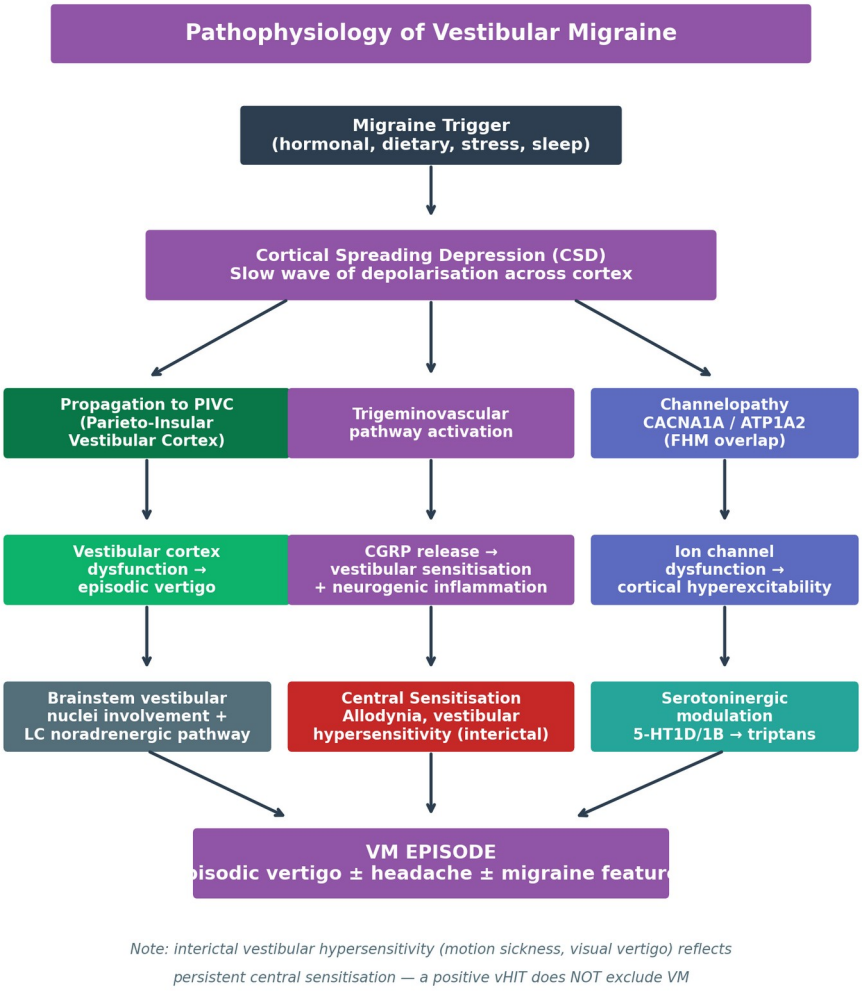

Figure 2. Pathophysiology of Vestibular Migraine — cortical spreading depression propagation to PIVC, trigeminovascular activation and CGRP, channelopathy arm, and convergence on central sensitisation and the VM episode.

Source: Australian Dizziness Clinics — clinical flowchart.

IV. ICHD-3 Diagnostic Criteria for Vestibular Migraine

Definite Vestibular Migraine — Full ICHD-3 Criteria

The 2018 ICHD-3 criteria for definite vestibular migraine (Appendix A1.6.5) require all of the following five criteria to be satisfied [7]:

A. At least five episodes of vestibular symptoms of moderate or severe intensity, lasting 5 minutes to 72 hours. Vestibular symptoms include spontaneous vertigo (internal or external), positional vertigo, visually-induced vertigo, head motion-induced vertigo, or head motion-induced nausea with unsteadiness.

B. Current or previous history of migraine with or without aura according to the ICHD-3 diagnostic criteria.

C. One or more migraine features occur with at least 50% of the vestibular episodes: (i) headache with at least two of: unilateral location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity; (ii) photophobia and phonophobia; (iii) visual aura.

D. Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder.

Probable Vestibular Migraine

Probable vestibular migraine applies when criteria A and D are met, and only one of B or C is satisfied (i.e., either a migraine history or migraine features are present, but not both). This classification is clinically important in paediatric practice, where the diagnostic history may be incomplete or where migraine features are difficult to elicit from younger children [24].

Paediatric Application and Caveats

Applying ICHD-3 criteria in children requires clinical adaptation. In pre-verbal or younger children (typically under 6 years), explicit reporting of photophobia or phonophobia may not be possible; however, behavioural correlates — such as seeking a dark, quiet room, covering the ears, or marked irritability during episodes — should be interpreted as equivalents of these migraine features [25]. Similarly, headache may not be reported or may follow the vestibular symptoms by hours rather than occurring simultaneously. Clinicians should not require simultaneous headache and vertigo: published data indicate that only approximately 30% of VM episodes in adults have both features concurrently [6,26]; this figure is likely even lower in children.

The sensitivity and specificity of ICHD-3 criteria for VM in paediatric populations have been evaluated in several clinic-based cohorts. Wiener-Vacher and colleagues demonstrated that a structured vestibular diary combined with ICHD-3 criteria achieved satisfactory diagnostic accuracy in children aged 8–16 years when applied over a minimum observation period of 3–6 months [27,28].

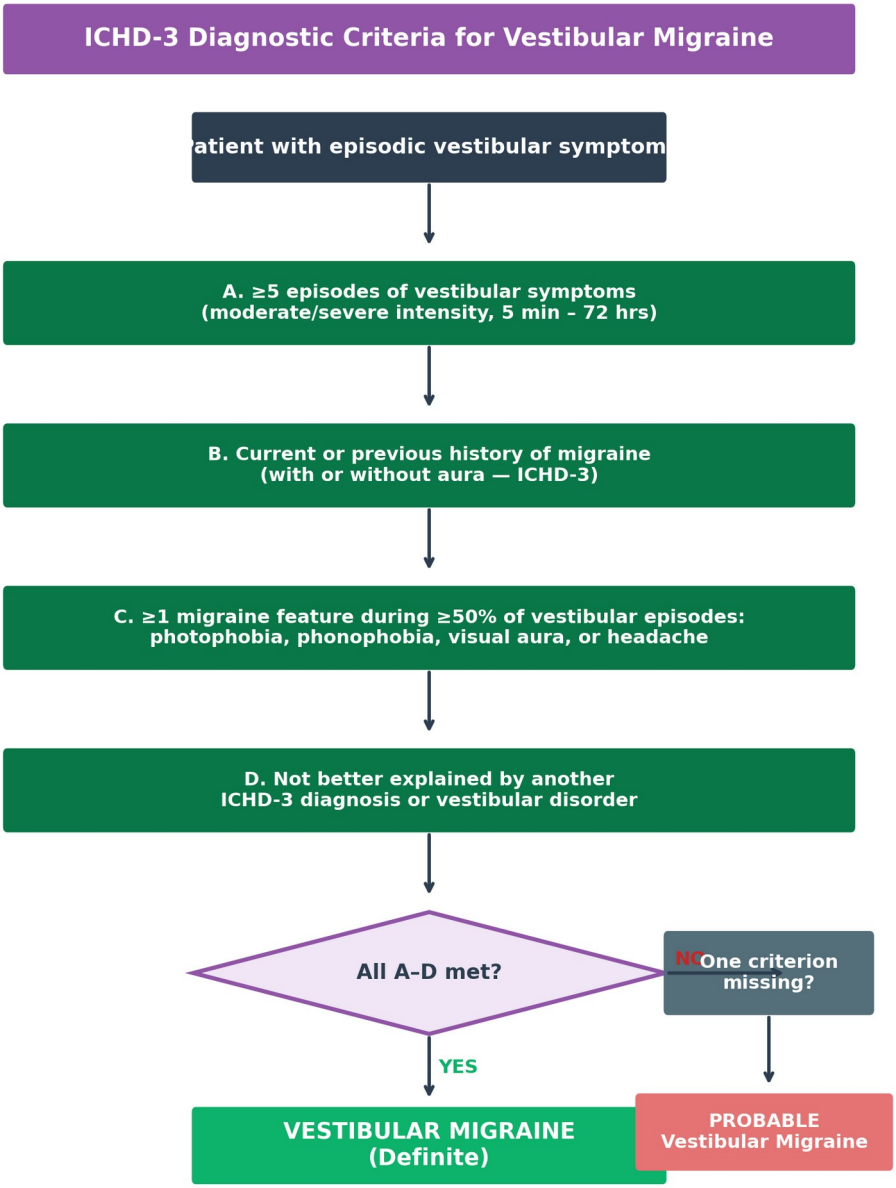

Figure 1. ICHD-3 Diagnostic Criteria for Vestibular Migraine — stepwise criteria checklist with branch to probable vestibular migraine when one criterion is absent.

Source: Australian Dizziness Clinics — clinical flowchart.

□ **Clinical Pearl:** The most common diagnostic error is requiring simultaneous headache and vertigo. Only 30% of VM episodes feature both concurrently. Migraine features (photophobia, phonophobia, nausea, need for rest) occurring in at least 50% of vestibular episodes are sufficient for Criterion C — the headache itself need not accompany every or any episode.

V. Clinical Features in Children: How VM Presents Differently

Episodic Vertigo: Duration and Quality

The cardinal feature of VM is episodic vestibular disturbance of moderate to severe intensity. In children, episodes typically last between 5 minutes and 72 hours, with most lasting 20 minutes to several hours

[28,29]. Spontaneous onset without a positional or movement trigger is common, though head movement and visual motion can both precipitate or exacerbate episodes. The vertigo is usually described as a sense of the room spinning or the child feeling as if they are moving; in younger or pre-verbal children, sudden cessation of activity, grasping for support, or clinging behaviour may be the presenting sign rather than a verbal complaint of vertigo.

Migraine Features During Episodes

Photophobia and phonophobia are the most consistently reported migraine features during VM episodes in children — and may be more prominent than headache itself [30]. Nausea is near-universal during acute episodes and is a diagnostically important feature. Visual aura — including scintillating scotomata, fortification spectra, hemianopia, and transient diplopia — may occur before, during, or independently of vestibular symptoms. Age-specific reporting of aura is important: younger children may describe visual disturbance as "seeing zig-zags", "sparkles", "a hole in my vision", or "things looking wavy" rather than using neurological terminology [25].

Interictal Symptoms

Between discrete VM episodes, a substantial proportion of affected adolescents experience persistent interictal symptoms including heightened motion sensitivity, visual vertigo (dizziness triggered by busy visual environments such as supermarkets or scrolling screens), and mild balance instability on challenging tasks [21,31]. These interictal symptoms reflect the central sensitisation mechanisms described in Section III and must be distinguished from PPPD — though the two conditions are not mutually exclusive and PPPD frequently develops as a consequence of inadequately treated VM.

Age-Specific Presentation

Adolescent girls present with the most recognisable adult-like VM phenotype, with well-characterised episodic vertigo, identifiable migraine features, and strong family history. A key trigger pattern in adolescent females is menstrual-cycle related: episodes clustering in the perimenstrual period are highly suggestive of VM and reflect oestrogen-withdrawal effects on the migraine threshold [32]. Pre-adolescent children more commonly present with shorter, less well-defined episodes and greater overlap with the BPVoC spectrum [8].

□ **Clinical Insight:** Adolescent girls presenting with dizziness triggered by menstruation, bright lights, busy visual environments, or sustained head movement should be actively screened for vestibular migraine before being labelled with anxiety or a functional diagnosis. The hormonal trigger pattern and photophobia are the highest-yield discriminators.

VI. Differential Diagnosis: The Key Mimics

The differential diagnosis of episodic vestibular symptoms in children requires systematic consideration of several conditions that can closely resemble VM. The key mimics are outlined below with the discriminating clinical features that separate each from VM.

Benign Paroxysmal Vertigo of Childhood (BPVoC)

BPVoC (see PVM02) overlaps significantly with VM in age range, family history, and episode character. The key distinction is that BPVoC episodes are typically very brief (seconds to 2 minutes), lack ICHD-3 migraine features (photophobia, phonophobia, headache), and occur predominantly in children under 5 years. Children with BPVoC do not meet ICHD-3 migraine criteria for VM; they have a migraine equivalent without migraine features. Transition to definite VM typically occurs in early adolescence [9].

Ménière's Disease

Ménière's disease presents with the triad of episodic vertigo, fluctuating sensorineural hearing loss, and aural fullness or tinnitus. It is rare in children and adolescents but does occur, particularly in association with endolymphatic hydrops secondary to autoimmune conditions or enlarged vestibular aqueduct syndrome [33]. The presence of documented hearing fluctuation on serial audiograms, aural pressure, and tinnitus distinguishes Ménière's from VM. Audiological investigation is essential in any child with episodic vertigo and any auditory symptoms.

Posterior Fossa Lesion

A posterior fossa neoplasm (medulloblastoma, ependymoma, brainstem glioma) or demyelinating process may present with episodic or progressive vestibular symptoms and headache. Features that mandate urgent neuroimaging include: progressive worsening of symptoms, symptoms that awaken the child from sleep, headache that is worst in the morning or with Valsalva, papilloedema, cerebellar signs (ataxia, dysmetria), or any cranial nerve deficit. Headache on exertion is a particularly important red flag in children.

PPPD and Functional Dizziness

PPPD is characterised by persistent non-spinning dizziness (lasting more than 3 months), postural sensitivity, and aggravation by visual motion — in the absence of discrete episodic vertigo as the dominant feature. PPPD may arise de novo or — critically — as a sequela of a primary episodic vestibular disorder including VM. In adolescents, anxiety and school avoidance are common comorbidities that both result from and further maintain persistent vestibular symptoms. PPPD and VM can coexist and both may require treatment [3].

Migraine with Brainstem Aura (Basilar Migraine)

Migraine with brainstem aura (previously termed basilar-type migraine) presents with aura symptoms clearly originating from the brainstem: vertigo, tinnitus, diplopia, ataxia, perioral paraesthesia, bilateral sensory symptoms, and decreased consciousness. This diagnosis overlaps with VM in its vestibular features but involves more prominent brainstem symptoms. Episodes of migraine with brainstem aura that include persistent neurological deficits must be distinguished from posterior circulation stroke using the HINTS examination (Head Impulse, Nystagmus, Test of Skew) and MRI brain with DWI [34].

□ **Important:** Migraine with brainstem aura (basilar-type migraine) can mimic acute posterior circulation stroke. If neurological deficits persist beyond the expected episode duration, the HINTS examination must be performed and urgent MRI with DWI is indicated. Do not assume a diagnosis of migraine in a child with acute vertigo and new neurological deficits.

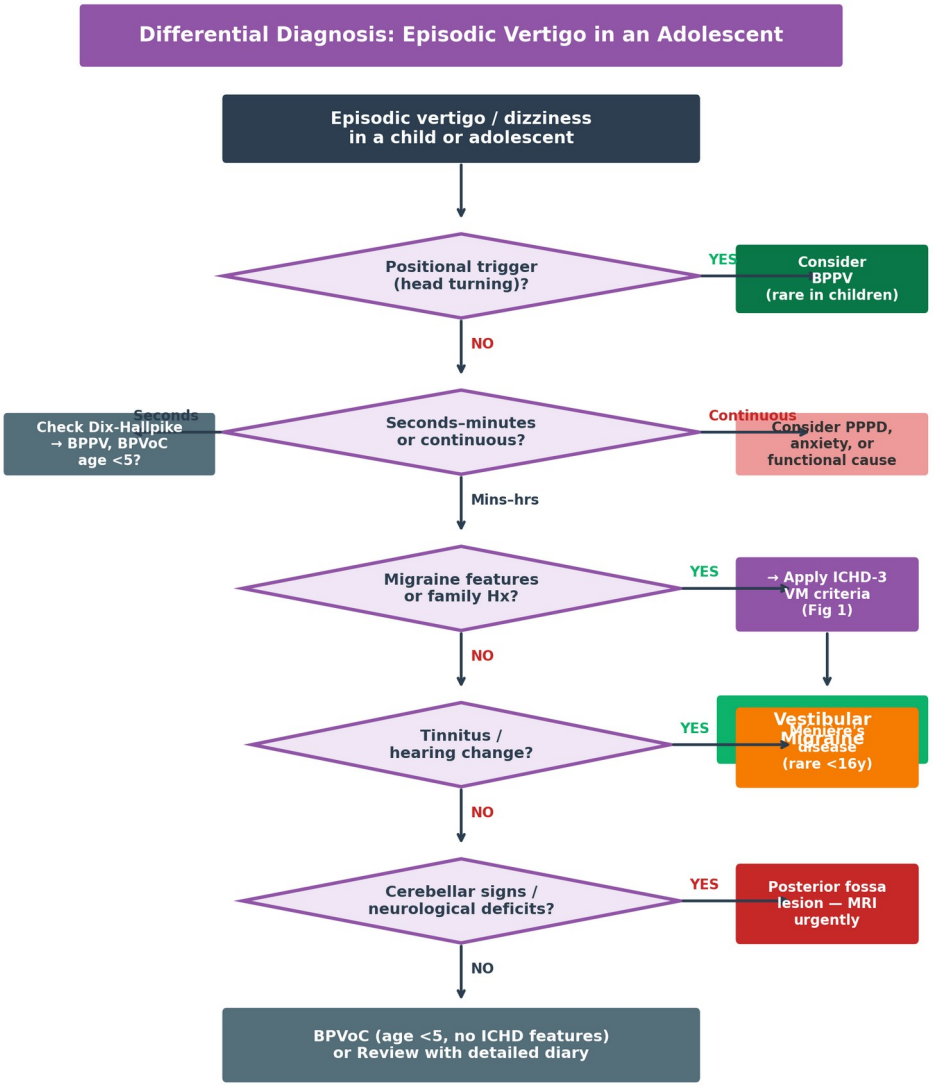

Figure 3. Differential Diagnosis Decision Tree: Episodic Vertigo in an Adolescent — systematic branching from positional trigger, duration, migraine features, hearing change, and neurological signs to final diagnoses.
Source: Australian Dizziness Clinics — clinical flowchart.

VII. Diagnostic Workup

Vestibular migraine is a clinical diagnosis: there is no diagnostic biomarker or pathognomonic test finding that confirms VM. The diagnostic workup serves three purposes: (1) to accumulate sufficient episodic data to satisfy the ICHD-3 minimum of five episodes; (2) to exclude structural and audiovestibular conditions that mimic VM; and (3) to characterise vestibular and psychological comorbidities that inform management planning.

Headache and Vestibular Diary

A prospectively maintained headache and vestibular diary is the most important diagnostic instrument in VM assessment. The diary should capture: episode date, duration, severity (moderate/severe), vestibular symptom type, associated migraine features (headache, photophobia, phonophobia, aura), potential triggers, and menstrual cycle phase in adolescent girls. A minimum of five documented episodes is required for ICHD-3 diagnosis; in practice, a 3–6-month diary observation period is often required in children with infrequent or variable episodes [27].

Audiological Assessment

A baseline pure-tone audiogram should be performed in all children with episodic vestibular symptoms. In VM, audiology is typically normal, serving primarily to exclude Ménière's disease and sensorineural hearing loss associated with enlarged vestibular aqueduct syndrome or labyrinthitis. Serial audiograms are indicated if hearing fluctuation is reported, as this changes the diagnostic priority toward Ménière's disease.

Vestibular Function Tests

Vestibular function testing in children with suspected VM serves primarily to exclude alternative diagnoses rather than to confirm VM. The video head impulse test (vHIT) assesses semicircular canal function via the vestibuloocular reflex (VOR). In VM, vHIT is normal or shows subtle interictal abnormalities that do not reflect true semicircular canal paresis [35]. Vestibular evoked myogenic potential (VEMP) testing — both cVEMP (saccular/inferior vestibular nerve) and oVEMP (utricular/superior vestibular nerve) — may show interictal abnormalities in VM but lacks specificity. Caloric testing assesses horizontal SCC function at low frequencies and is typically normal or shows mild asymmetry in VM. None of these tests has sufficient sensitivity or specificity to diagnose or exclude VM independently.

Neuroimaging

MRI brain (with and without contrast, including posterior fossa sequences and DWI) is indicated in the following clinical scenarios: first or worst headache of life; new neurological deficit; progressive symptoms; papilloedema; exertional headache; symptoms awakening from sleep; gait ataxia or cerebellar signs; and age under 3 years with unexplained episodic vestibular symptoms. CT brain is not adequate for posterior fossa assessment and should not substitute for MRI in children with vestibular presentations. In uncomplicated VM without red flags, neuroimaging is not routinely required for diagnosis.

Psychological and Hormonal Assessment

Psychological screening with validated tools — the PHQ-A (Patient Health Questionnaire for Adolescents) for depression and GAD-7 for anxiety — should be routine in adolescents with VM given the high prevalence of anxiety and depressive comorbidity [36]. In adolescent girls with a menstrual trigger pattern, correlation of the symptom diary with menstrual cycle phase is diagnostically and therapeutically important, as this identifies candidates for hormonal migraine management strategies.

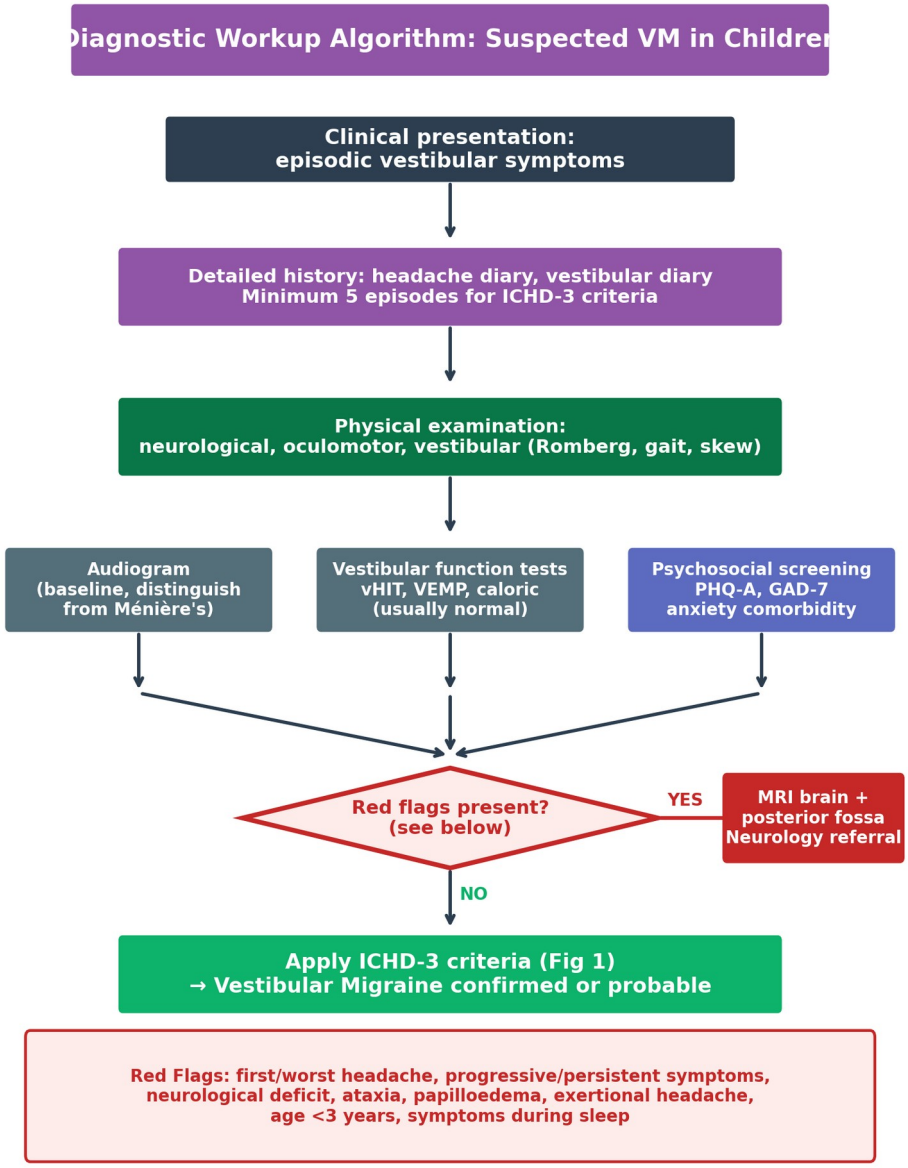

Figure 4. Diagnostic Workup Algorithm for Suspected Vestibular Migraine in Children — from clinical assessment through vestibular testing, red-flag evaluation, and ICHD-3 criteria application.

Source: Australian Dizziness Clinics — clinical algorithm.

□ **Clinical Insight:** In the majority of children with VM, the diagnosis rests on the clinical history and prospective diary data alone. Vestibular function tests have supporting rather than diagnostic roles. Avoid over-investigation with normal or non-diagnostic test results — focus diagnostic energy on accumulating sufficient episodic diary data to meet ICHD-3 criteria.

VIII. Management: Acute and Preventive Strategies

Acute Management

Acute VM episode management in children follows a stepwise approach. Environmental measures — rest in a dark, quiet room — should be the first-line intervention and are effective for mild to moderate episodes. NSAIDs, specifically ibuprofen at weight-based dosing, are the first pharmacological option for analgesia and have established efficacy in paediatric migraine [37]. Antiemetics — ondansetron (4–8 mg

oral or sublingual, weight-based) or prochlorperazine (in older children) — address nausea and vestibular symptoms during acute episodes and improve functional recovery.

Triptans are the gold-standard acute migraine therapy for children aged 12 years and over. Sumatriptan nasal spray (10–20 mg) and zolmitriptan nasal spray (2.5–5 mg) are the most commonly used formulations in adolescents and have demonstrated efficacy in randomised controlled trials of paediatric migraine [38,39]. Nasal spray formulations are preferred in VM given the frequent nausea and vomiting that limits oral bioavailability. Triptans should be used early in the episode for optimal efficacy; they are contraindicated in migraine with brainstem aura [22].

Non-Pharmacological Prevention

Non-pharmacological preventive strategies should be implemented in all children with VM as the foundation of the management plan, regardless of whether pharmacological prevention is also indicated. Sleep regulation — maintaining consistent sleep-wake cycles, targeting 8–10 hours per night in adolescents — is the most evidence-supported behavioural intervention for migraine prevention [40]. Trigger identification through diary review and systematic avoidance of modifiable triggers (dietary: tyramine, caffeine, MSG, skipped meals; environmental: bright or flickering lights, screen fatigue, dehydration) is individually tailored based on diary data.

Mindfulness-based stress reduction (MBSR) and cognitive behavioural therapy (CBT) have demonstrated efficacy in paediatric migraine prevention and are particularly important given the high comorbid anxiety burden in adolescent VM [41,42]. Vestibular physiotherapy is indicated for children with significant interictal motion sensitivity, visual vertigo, or balance impairment — it addresses the central sensitisation component through habituation and balance retraining and can meaningfully improve functional quality of life between discrete VM episodes [43].

Pharmacological Prevention

Pharmacological prevention is indicated when VM episodes occur four or more times per month, or when episodes cause significant functional impairment (school absence, sleep disruption, social withdrawal) even at lower frequencies [44]. The choice of agent is guided by patient age, comorbidities, tolerability, and available evidence. The Childhood and Adolescent Migraine Prevention (CHAMP) trial (Powers et al. 2017) — the largest RCT of preventive migraine therapy in children — compared topiramate and propranolol against placebo and found no significant superiority over placebo on the primary endpoint (50% reduction in headache frequency) [45]. However, a proportion of individual patients in both treatment arms responded meaningfully, and clinical practice has not abandoned these agents.

Topiramate (1–2 mg/kg/day in divided doses, maximum 100 mg/day) is the most widely used preventive agent in adolescent VM. Side effects include cognitive slowing ("dopamax"), word-finding difficulty, weight loss, paraesthesia, and teratogenicity (neural tube defects) — the last necessitating reliable contraception discussion in adolescent girls [46]. Propranolol (1–2 mg/kg/day) is contraindicated in asthma, Raynaud's disease, and diabetes and should be used cautiously in athletes.

Amitriptyline (10–25 mg nocte, titrating upward) is useful in older adolescents, particularly those with comorbid sleep disorder, anxiety, or depression. ECG screening for QTc prolongation is required prior to initiation. Cyproheptadine (0.25 mg/kg/day) is the preferred agent in younger children (under 12 years) and has a favourable side-effect profile; its appetite-stimulating effect can be beneficial in children with poor weight gain. Cinnarizine, a calcium channel blocker and antihistamine with specific vestibular suppressant properties, is widely used in Australian and UK paediatric vestibular practice for VM and is particularly useful when interictal vestibular symptoms are prominent [47].

CGRP pathway therapies (erenumab, fremanezumab, galcanezumab) represent an emerging preventive option. Paediatric data remain limited; however, open-label case series and early clinical trial data in adolescents show promising tolerability and efficacy in refractory VM [48]. Referral to a paediatric neurologist or vestibular physician with migraine expertise is appropriate when two adequate trials of preventive therapy have failed.

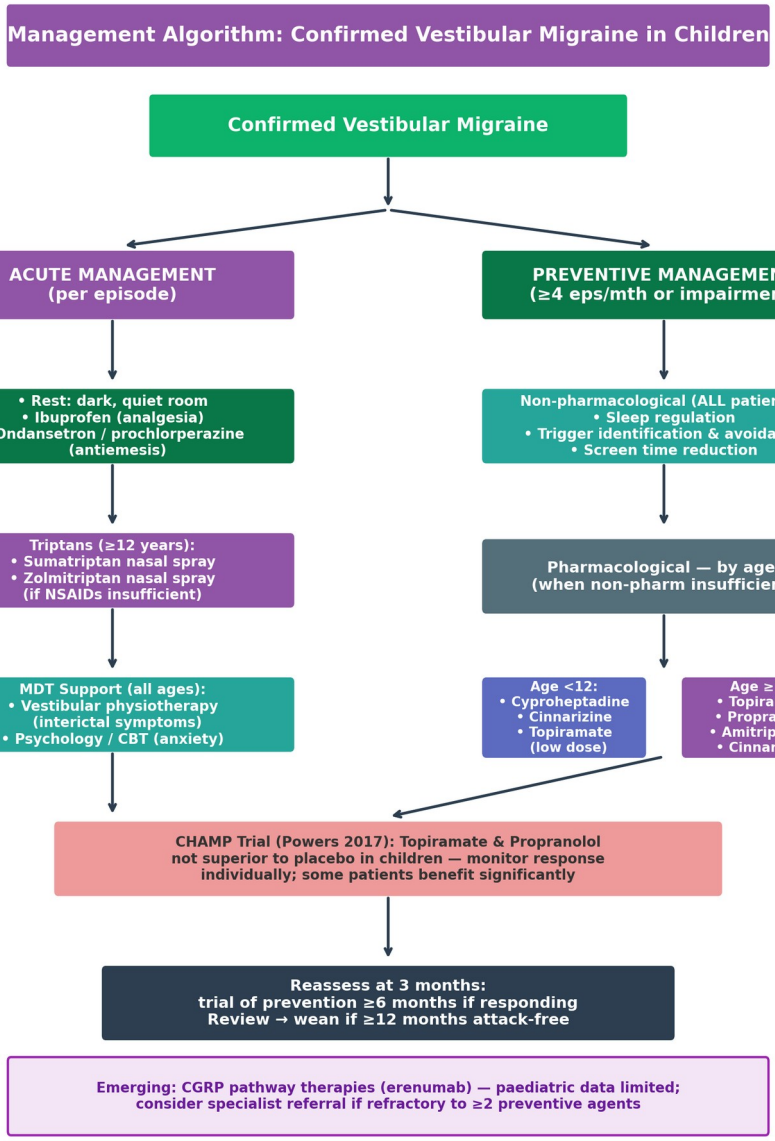

Figure 5. Management Algorithm for Confirmed Vestibular Migraine in Children — acute therapy and preventive therapy branches with pharmacological options stratified by age.

Source: Australian Dizziness Clinics — clinical algorithm.

□ **Key Point:** The CHAMP trial (Powers 2017) found topiramate and propranolol were not superior to placebo on the primary endpoint in children. However, both drugs showed response in individual patients and remain in clinical use. Monitor response at 3 months and continue for a minimum of 6 months if effective before considering weaning.

IX. Prognosis, Comorbidities, and Long-Term Follow-Up

The long-term prognosis of paediatric VM is generally favourable with appropriate management, though complete resolution is not universal. Published cohort studies report 50–60% of children experiencing significant reduction in episode frequency and severity within 12–18 months of initiating combined lifestyle modification and pharmacological prevention [49,50]. Children and adolescents who achieve good trigger identification and sleep regulation frequently reduce preventive medication requirements over time. However, VM is a chronic relapsing condition, and a subset of patients — particularly those with high psychiatric comorbidity or familial channelopathy — require long-term management into adulthood.

Psychological comorbidity — primarily anxiety and depression — is present in up to 40–50% of adolescents with VM and has a bidirectional relationship with disease severity: anxiety lowers the vestibular symptom threshold (central sensitisation), while recurrent disabling vestibular episodes drive anxiety and avoidance behaviours [36,51]. This bidirectional relationship mandates integrated psychological support as a component of all VM management plans, not as an afterthought or alternative to vestibular-specific treatment.

PPPD is an important sequela of inadequately treated or unrecognised VM. When episodic VM is not identified and managed, the persistent vestibular sensitisation and avoidance behaviours associated with recurrent episodes create the substrate for PPPD. Clinicians managing adolescents with persistent non-episodic dizziness and anxiety should always enquire about a prior history of discrete episodic vertigo to identify an underlying or concurrent VM aetiology [3,52].

Transition to adult care requires deliberate planning. VM managed by a paediatric neurologist or vestibular physician during adolescence should include a transition plan at approximately 16–18 years to an adult vestibular medicine or neurology service. The preventive medication regime, trigger profile, and functional status should be documented and communicated to the receiving adult service. Genetic counselling is appropriate for families with multiple affected members and documented channelopathy mutations, particularly CACNA1A-related phenotypes.

Long-term monitoring should include serial vestibular function assessment and audiological monitoring — particularly to detect any late-onset endolymphatic hydrops in patients with a prolonged VM history [53]. Hearing monitoring is especially important in patients with any auditory symptoms (tinnitus, aural fullness) that could signal the development of a Ménière's overlap phenotype.

□ **Clinical Insight:** PPPD frequently develops as a sequela of inadequately treated VM. Any adolescent with chronic daily or near-daily dizziness should be reviewed for an underlying episodic vestibular disorder — particularly VM — before a primary diagnosis of PPPD or anxiety is established. Treating the underlying VM may resolve or substantially reduce the persistent functional symptoms.

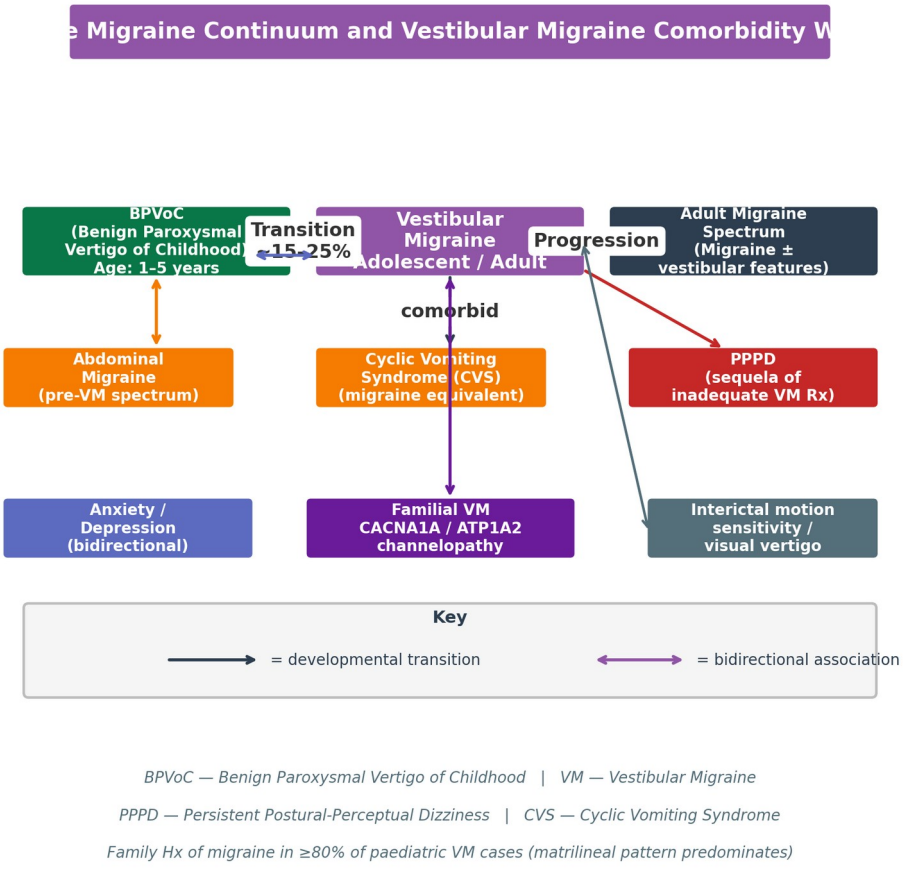

Figure 6. The Migraine Continuum and Vestibular Migraine Comorbidity Web — BPVoC transitioning to VM and adult migraine spectrum, with bidirectional links to PPPD, anxiety, CVS, abdominal migraine, and familial channelopathy.
Source: Australian Dizziness Clinics — clinical flowchart.

X. Summary and Key Clinical Takeaways

The following ten points represent the highest-yield clinical insights from this review of vestibular migraine in children and adolescents:

1. VM is the most common cause of episodic vertigo in adolescents and accounts for approximately 20% of paediatric vestibular clinic referrals. It is substantially underdiagnosed.

2. Do not require simultaneous headache and vertigo. Only 30% of VM episodes feature both concurrently. Migraine features present in ≥50% of vestibular episodes satisfy ICHD-3 Criterion C.

3. A matrilineal family history of migraine is the single most useful screening question and is present in up to 80% of paediatric VM cases.

4. BPVoC is a childhood migraine equivalent and precursor to VM. 15–25% of BPVoC cases transition to VM during adolescence — always take a BPVoC history.

5. Interictal vestibular hypersensitivity reflects central sensitisation — it is not diagnostic of a structural peripheral vestibular lesion. A mildly abnormal vHIT does not exclude VM.

6. Red flags mandate urgent MRI. Progressive symptoms, papilloedema, cerebellar signs, exertional headache, and persistent neurological deficits require urgent posterior fossa MRI with DWI.

7. Migraine with brainstem aura (basilar migraine) can mimic stroke. HINTS examination and MRI are required when neurological deficits persist beyond the expected episode duration.

8. The CHAMP trial showed no superiority of topiramate or propranolol over placebo on the primary endpoint in children — but individual patients respond. Monitor at 3 months and individualise pharmacological decisions.

9. Anxiety and VM are bidirectional. Psychological support (CBT, MBSR) is not an alternative to vestibular treatment — it is an integral component of the management plan for all adolescents with VM.

10. PPPD as a VM sequela: any adolescent with chronic persistent dizziness should be evaluated for an underlying episodic VM history before PPPD is diagnosed as a primary condition.

For clinical management of BPPV in children, refer to PVM04 — BPPV in Children.

□ **Key Point:** The diagnostic and management approach to VM in children requires integration of ICHD-3 criteria, prospective diary data, exclusion of structural mimics, and a multidisciplinary team involving vestibular physicians, physiotherapists, and psychologists. No single test confirms VM — the diagnosis is built on the clinical narrative.

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