Benign Paroxysmal Vertigo of Childhood:

Diagnosis, Pathophysiology, and Clinical Management

Vestibular Medicine in Children

Topic 2 of 15

Australian Dizziness Clinics | www.AustralianDizzinessClinics.com

Version 1.0 | April 2026

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: Identifies the most clinically important take-home message in the surrounding text — the single fact or concept most likely to change practice.

Clinical Insight: Provides mechanistic or physiological context that deepens understanding of the clinical presentation or diagnostic approach.

Clinical Pearl: A practical, experience-based tip for direct application at the bedside or in the clinic — high-yield and immediately usable.

Important: Flags a safety-critical issue, diagnostic pitfall, or common error that must not be missed in clinical practice.

Table of Contents

I. Introduction: BPVoC in Context

Feature	BPVC finding	Clinical relevance
Age peak	18 months – 5 years	Rare after age 6; overlap with vestibular migraine in older children
Episode duration	Seconds to minutes (usually <2 min)	Distinguishes from VM (minutes to hours) and epilepsy
Nystagmus	Horizontal nystagmus during episode; normal between attacks	Confirms vestibular origin
Consciousness	Fully preserved; frightened appearance	Key: child is alert — NOT epilepsy
Pallor/vomiting	Common	Autonomic accompaniment; resolves completely
Interical examination	Completely normal	Essential feature — any deficit = investigate further
Audiogram/EEG	Normal	Required to confirm diagnosis and exclude structural cause

II. Epidemiology and Natural History

Criterion	ICHD-3 requirement
A. Episodes	≥5 attacks of sudden vertigo (without provoking cause)
B. Duration	Seconds to minutes, resolving spontaneously
C. Symptom-free intervals	Completely normal between attacks
D. Tests normal	Normal audiological examination and vestibular function
E. No other cause	Not better accounted for by another ICHD-3 diagnosis
Migraine family history	Not required but supports diagnosis; ≥50% have positive FHx

III. Pathophysiology: The Migraine Hypothesis and Beyond

IV. Clinical Features and Diagnostic Criteria

Diagnosis	Key differentiating features	Distinguishing test
Vestibular migraine (older child)	Longer duration; headache; photophobia; Dix-Hallpike negative	ICHD-3 criteria; age >6
Epilepsy (temporal lobe)	Altered consciousness; post-ictal phase; EEG abnormal	EEG; neurology referral
BPPV	Triggered by head movement; positive Dix-Hallpike; older children	Dix-Hallpike test
Posterior fossa tumour	Progressive; headache; cerebellar signs; papilloedema	MRI posterior fossa
Orthostatic hypotension	Triggered by standing; no nystagmus; BP drop >20 mmHg systolic	Orthostatic BP series

Anxiety/panic	No nystagmus; associated anxiety features; normal vestibular function	Clinical history; mental health screen

V. Differential Diagnosis: What Else Looks Like BPVoC?

Investigation	Purpose	Timing
Audiogram (pure-tone)	Exclude SNHL; rule out ototoxicity	At diagnosis
EEG	Exclude epilepsy if episodes have atypical features	If atypical features
MRI brain/posterior fossa	Exclude structural cause	If headache, focal neurology, or atypical course
vHIT	Peripheral vestibular function between attacks	If vestibular function suspected abnormal
Blood glucose	Hypoglycaemia-related episodic vertigo	If fasting episodes or metabolic risk
Maternal migraine history	Establishes migraine family history	Clinical history at diagnosis

VI. Diagnostic Workup: What to Investigate and Why

VII. Management: Acute Episode and Preventive Strategies

Timepoint	Assessment	Action if abnormal
6 weeks post-diagnosis	Diary review; episode frequency/duration	Revise diagnosis if duration increasing or new features
3 months	Symptom diary; school attendance	Consider prophylaxis if >2 episodes/month
6 months	Neurodevelopment; headache onset	If headaches developing → reclassify as vestibular migraine
12 months	Frequency trend; ICHD-3 criteria re-review	If persisting beyond age 7 → consider VM diagnosis
Resolution	>6 months symptom-free	Discharge; counsel that VM may emerge in adolescence

VIII. BPVoC, Vestibular Migraine, and the Migraine Continuum

IX. Long-Term Outcomes and Transition to Adulthood

Indication	Urgency	Refer to
Atypical features (altered consciousness, focal signs)	Urgent	Neurology; MRI
Abnormal audiogram or interical vestibular deficit	Soon	Vestibular physician + audiology
Failure to resolve by age 8	Routine	Vestibular physician; reclassify as vestibular migraine?
Parental anxiety; frequent school absence	Routine	Vestibular physician; paediatric psychologist
Progressive symptoms; new headache component	Urgent	Neurology; MRI posterior fossa

X. Summary and Key Clinical Takeaways

I. Introduction: BPVoC in Context

ICHD-3 Classification and Historical Context

Benign Paroxysmal Vertigo of Childhood (BPVoC) is classified by the International Headache Society in the third edition of the International Classification of Headache Disorders (ICHD-3) as an episodic syndrome that may be associated with migraine. It sits within the broader category of childhood periodic syndromes — disorders that share pathophysiological mechanisms with migraine but manifest differently in the developing nervous system. The ICHD-3 classification marked a pivotal conceptual advance: BPVoC is no longer viewed as a peripheral vestibular disorder of uncertain origin but as an age-specific expression of the migraine spectrum. [1]

The condition was first described by Basser in 1964 in a landmark paper reporting brief episodes of vertigo in young children with a strong family history of migraine and a benign, self-limited course. Basser correctly identified the condition as distinct from epilepsy and from structural peripheral vestibular disease. Subsequent decades saw considerable debate about aetiology — with early theories including viral labyrinthitis, perilymph fistula, and vestibular end-organ pathology — before the migraine hypothesis became dominant in the 1990s following population and family studies by Abu-Arafeh and Russell. [2,3]

Clinical Pearl: BPVoC is the most common cause of episodic vertigo in children under 5 years of age — yet most emergency physicians have never encountered the diagnosis. Failure to recognise BPVoC exposes children to unnecessary investigations, prolonged parental anxiety, and delayed reassurance.

Relationship to the Migraine Spectrum

BPVoC belongs to a family of episodic syndromes in childhood that are now understood as migraine equivalents. These include cyclic vomiting syndrome (CVS), abdominal migraine, and benign paroxysmal torticollis of infancy. All share a familial migraine background, a self-limited course, episodic pattern without identifiable structural pathology, and a tendency to evolve toward typical migraine or vestibular migraine as the child grows. The unifying mechanism involves cortical spreading depression and trigeminovascular sensitisation operating in an immature and hyperexcitable neural substrate. [4]

In paediatric vestibular clinics, BPVoC accounts for approximately 15–30% of all referrals for episodic dizziness or vertigo, making it the single most common vestibular diagnosis in the preschool age group. Despite this, a significant proportion of affected children are first seen in emergency departments where the diagnosis is not made, investigations are performed unnecessarily, and the family is discharged without a definitive explanation. This underdiagnosis reflects limited training in paediatric vestibular medicine at all levels of the medical workforce. [5]

Key Point: BPVoC is classified in ICHD-3 as an episodic syndrome associated with migraine — not a peripheral vestibular disorder. This classification carries direct management implications: the family history, the migraine background, and the prevention strategies all follow from the migraine framework.

Scope of This Review

This review covers BPVoC from epidemiology through pathophysiology, diagnostic criteria, differential diagnosis, diagnostic workup, management (both non-pharmacological and pharmacological), the relationship to vestibular migraine, and long-term outcomes. It is written for vestibular physicians, paediatricians, and emergency physicians who will encounter BPVoC in both dedicated vestibular clinics and general clinical settings. References to key clinical decision points are signposted throughout. Flowcharts for diagnostic criteria (Figure 1), differential diagnosis (Figure 2), pathophysiology (Figure 3), workup (Figure 4), management (Figure 5), and natural history (Figure 6) accompany the text.

II. Epidemiology and Natural History

Incidence, Prevalence, and Age of Onset

BPVoC has a reported prevalence of approximately 2–3% in the general childhood population, based on population-based questionnaire studies from Scotland and Scandinavia, although this figure likely underestimates the true prevalence given that many episodes go unrecognised or misdiagnosed. The condition typically begins between the ages of 2 and 5 years, with onset before age 2 being unusual and warranting careful consideration of alternative diagnoses including benign paroxysmal torticollis of infancy

or structural posterior fossa pathology. Onset after age 8 is increasingly likely to represent early vestibular migraine rather than classic BPVoC. [6]

Abu-Arafeh and Russell conducted the first large epidemiological study of BPVoC in 1995, surveying over 2000 schoolchildren in Aberdeen and identifying a point prevalence of 2.6%. Their data confirmed the migraine family history association — present in approximately 70% of affected children — and established that the condition was underrecognised by teachers, parents, and clinicians alike. Studies by Wiener-Vacher and colleagues at the Necker Enfants Malades Hospital in Paris subsequently provided detailed clinical characterisation data from a dedicated vestibular clinic cohort. [2,7]

Key Point: BPVoC typically presents between age 2 and 5. Onset before age 2 or after age 8, a progressive course, or associated neurological signs should prompt urgent reconsideration of the diagnosis — particularly posterior fossa tumour and epilepsy.

Sex Distribution and Family History

Most series report an approximately equal sex distribution in early childhood, with a slight female predominance emerging in the subgroup that transitions to vestibular migraine in adolescence — mirroring the female predominance in adult vestibular migraine (approximately 3:1 female to male). A family history of migraine is present in 60–80% of affected children across published cohorts, strongly supporting the migraine hypothesis. In some families, a clear autosomal dominant inheritance pattern is apparent with variable expressivity — the same CACNA1A mutation may produce familial hemiplegic migraine in a parent and BPVoC in a child. [8]

Natural History: Resolution and Transition

The natural history of BPVoC is predominantly benign, with spontaneous resolution in the majority of affected children by the age of 8–10 years. Longitudinal cohort studies report resolution rates of approximately 70–80% within 3–5 years of onset. However, a significant minority — estimated at 20–30% in published series — does not simply resolve but transitions to vestibular migraine in late childhood or adolescence. Langhagen and colleagues, in a long-term follow-up study of a paediatric vestibular cohort, found that 21% of children initially diagnosed with BPVoC subsequently met ICHD-3 criteria for vestibular migraine. Marcelli and colleagues reported similar rates in an Italian cohort. [9,10]

The transition from BPVoC to vestibular migraine is thought to reflect the maturing nervous system's capacity to generate the full clinical phenotype of vestibular migraine — including verbalised rotatory vertigo, phonophobia, photophobia, and headache — as the child's cognitive and language development allows more complete symptom expression. In practice, the clinician managing a child with BPVoC should explicitly counsel the family that while the condition is likely to resolve, the child carries an elevated lifetime risk of vestibular migraine and migraine-spectrum disorders into adulthood. [10]

Clinical Insight: Most children with BPVoC will outgrow the condition by 8–10 years. However, approximately 20–30% will transition to vestibular migraine in adolescence. Long-term follow-up is warranted even after apparent resolution — advising families to monitor for migraine symptoms as the child enters the teenage years is part of good clinical practice.

III. Pathophysiology: The Migraine Hypothesis and Beyond

Cortical Spreading Depression and Vestibular Expression

The prevailing pathophysiological model for BPVoC invokes the same fundamental mechanism as migraine: cortical spreading depression (CSD), a wave of sustained neuronal and glial depolarisation followed by prolonged suppression that propagates across cortical and subcortical structures at approximately 3–5 mm per minute. In adult vestibular migraine, CSD is thought to propagate into the parietoinsular vestibular cortex, the posterior insula, and the temporal parietal junction — regions responsible for vestibular cortical processing and multisensory integration. In the developing brain, the hyperexcitable and incompletely myelinated cortex may exhibit lower thresholds for CSD initiation and propagation. [11]

The brainstem plays a central role in the pathophysiology of BPVoC. The trigeminal nucleus caudalis — the principal relay nucleus for trigeminal pain — has extensive anatomical and functional connections with the vestibular nuclei in the medulla and pons. Convergent inputs from the trigeminal system can sensitise vestibular nucleus neurons, producing episodic vestibular dysfunction in the absence of any structural labyrinthine pathology. This trigeminovestibular convergence is the mechanistic basis for the

clinical observation that migraine attacks in both adults and children are frequently accompanied by vestibular symptoms. [12]

Clinical Insight: The absence of peripheral vestibular findings in BPVoC — normal vHIT, normal caloric, normal VEMP — is not a diagnostic failure. It is the expected finding, because the dysfunction is central (brainstem trigeminovestibular convergence and cortical CSD) rather than peripheral. Explaining this to families avoids unnecessary re-investigation.

Ionic Channelopathy Hypothesis: CACNA1A, ATP1A2, SCN1A

A growing body of genetic evidence supports a channelopathy contribution to the BPVoC phenotype. The CACNA1A gene, encoding the alpha-1A subunit of P/Q-type voltage-gated calcium channels, is the most extensively studied. Mutations in CACNA1A produce a clinical spectrum including: familial hemiplegic migraine type 1 (FHM1), episodic ataxia type 2 (EA2), spinocerebellar ataxia type 6 (SCA6), and — in a paediatric context — BPVoC. The shared genetic substrate across this spectrum suggests that the age at which a given CACNA1A mutation manifests is partly determined by developmental calcium channel expression patterns in the immature brain. [13]

ATP1A2 mutations (FHM2, Na+/K+-ATPase) and SCN1A mutations (FHM3, Nav1.1 sodium channel) have also been implicated in familial cases. These mutations impair the clearance of extracellular potassium and glutamate during and after CSD, creating a local ionic milieu that lowers the threshold for repeat spreading depression. In the vestibular system, where the endolymph already maintains a highly unusual ionic composition (high potassium, low sodium), ionic channel dysfunction may particularly disrupt hair cell and afferent nerve function. [13,14]

Important: CACNA1A mutations cause a clinical spectrum including BPVoC, episodic ataxia type 2, and familial hemiplegic migraine — often within the same family. When a child presents with BPVoC, always ask about family members with episodic ataxia, hemiplegic migraine, or unexplained episodic neurological symptoms. Genetic counselling is indicated in multiplex families.

Cerebellar Involvement and Serotonergic Modulation

The flocculonodular lobe of the cerebellum — the phylogenetically ancient 'vestibular cerebellum' — is a critical modulator of vestibular signal processing, VOR gain adaptation, and postural stability. Cerebellar Purkinje cells express high densities of P/Q-type calcium channels (the CACNA1A product), making the flocculonodular lobe particularly susceptible to ionic channelopathy-mediated dysfunction. During a BPVoC episode, transient flocculonodular dysfunction may disrupt ongoing vestibular signal processing, producing the acute unsteadiness and distress characteristic of the disorder. This model is supported by the overlap between BPVoC and episodic ataxia type 2, in which cerebellar dysfunction is the dominant phenotype. [15]

Serotonin (5-HT) plays a modulatory role in vestibular neurotransmission at multiple levels of the vestibular pathway. 5-HT1B/1D receptors on trigeminal afferents are the target of triptan medications, and the same receptor subtypes are expressed on vestibular nucleus neurons. Serotonergic modulation of vestibular processing may explain why non-pharmacological migraine prevention strategies — particularly sleep hygiene and dietary trigger avoidance — can reduce BPVoC episode frequency, as many triggers act via serotonergic pathways. [12,16]

IV. Clinical Features and Diagnostic Criteria

ICHD-3 Diagnostic Criteria in Detail

The ICHD-3 diagnostic criteria for BPVoC require: (A) at least 5 episodes fulfilling criteria B and C; (B) vertigo occurring without warning, maximal at onset, and resolving spontaneously after minutes to hours (typically less than 5 minutes) without loss of consciousness; (C) at least one of the following interictal features: nystagmus, ataxia, vomiting, pallor, or fearfulness; and (D) normal neurological examination, audiometric and vestibular functions between episodes; and (E) not attributed to another disorder. [1]

Several aspects of these criteria warrant clinical emphasis. The 'at least 5 episodes' threshold exists to distinguish BPVoC from isolated events — a single episode of acute vertigo in a toddler may represent the first presentation of BPVoC but should also prompt consideration of posterior fossa pathology. The criterion that episodes resolve spontaneously, without loss of consciousness, distinguishes BPVoC from epileptic events (which may involve altered consciousness and post-ictal confusion) and from cardiac

arrhythmia (which may produce true syncope). The requirement for normal interictal examination is absolute — any child with persistent nystagmus, abnormal eye movements, cerebellar signs, or hearing loss in the interictal period has a different diagnosis. [1,17]

Clinical Pearl: In toddlers, BPVoC often presents as sudden onset of crying, unsteadiness, and clinging to a parent — not as verbalised rotatory vertigo. The diagnosis requires a careful, structured history from the primary caregiver, asking specifically: 'What does the child look like? What do they do during the episode? How long does it last? How do they seem afterwards?' Without this structured approach, BPVoC is easily missed or mislabelled as 'funny turns'.

Age-Specific Presentation Differences

In toddlers (aged 2–3 years), BPVoC episodes are dominated by behavioural features: sudden onset distress, crying, clinging, reaching for support, truncal unsteadiness, and pallor. Nausea may be evident as a reluctance to feed or gagging. The child cannot verbalise that the world is spinning — this capacity requires both mature language development and the cognitive ability to describe an internal vestibular percept. Episodes are typically brief (under 2 minutes in this age group) and the child returns to normal activity almost immediately. The absence of post-ictal confusion or drowsiness is a key distinguishing feature from epilepsy. [6,17]

In school-age children (5–8 years), episodes begin to take on a more recognisable vestibular character. Older children can describe spinning or tilting sensations, and headache may accompany some episodes — at this point, the distinction between BPVoC and early vestibular migraine becomes clinically challenging and may require longitudinal observation rather than a single diagnostic encounter. Nystagmus, if present during an episode, should be characterised: horizontal direction-fixed nystagmus is consistent with a peripheral or benign central mechanism, whereas direction-changing, vertical, or purely torsional nystagmus demands urgent neuroimaging. [1,18]

Important: Any child with nystagmus during an episode, cerebellar signs on interictal examination, or a progressive course must not be diagnosed with BPVoC until central pathology has been excluded by MRI brain with gadolinium. The price of missing a posterior fossa tumour is catastrophic.

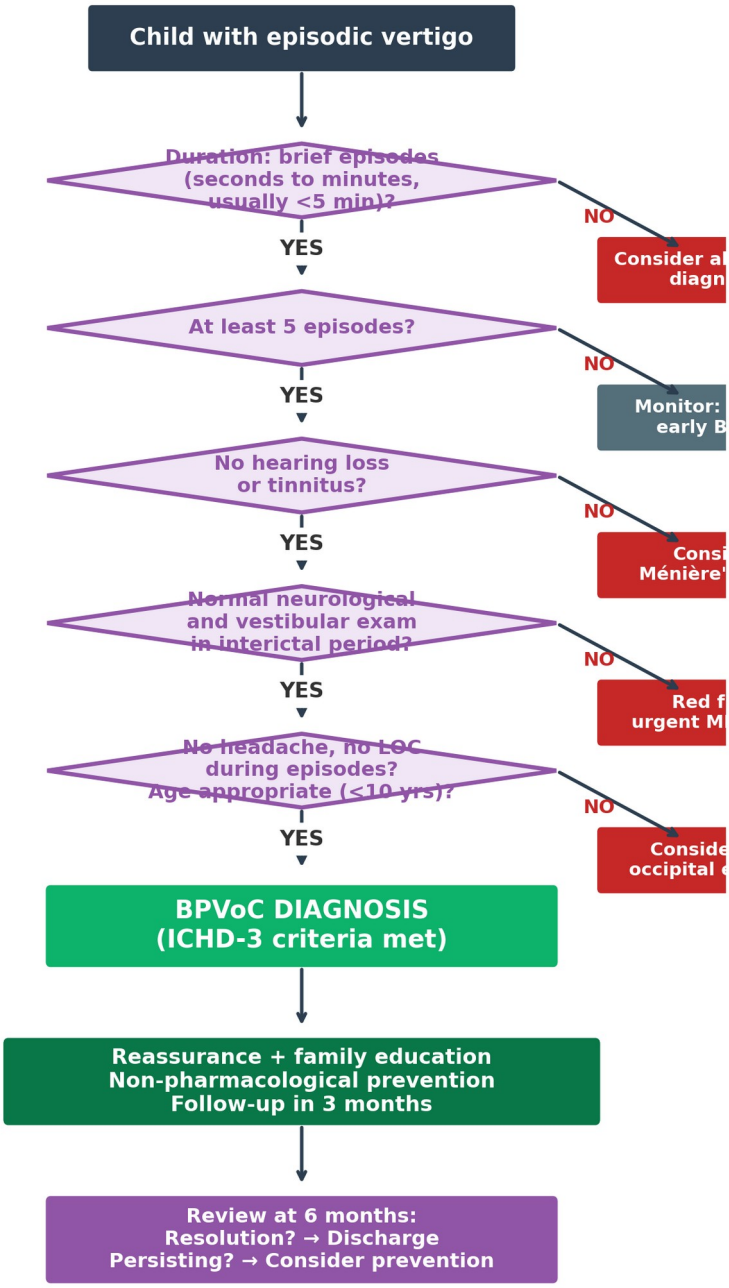

Figure 1. ICHD-3 Diagnostic Criteria for BPVoC — Stepwise diagnostic flowchart applying ICHD-3 criteria to a child with episodic vertigo.

V. Differential Diagnosis: What Else Looks Like BPVoC?

Vestibular Migraine

Vestibular migraine (VM) is the most important differential diagnosis for BPVoC, particularly in children over 6 years. The ICHD-3 criteria for vestibular migraine require at least 5 episodes of vestibular symptoms of moderate or severe intensity, a current or prior history of migraine, and at least 50% of episodes associated with migraine features (headache, photophobia, phonophobia, or visual aura). In school-age children, the distinction between BPVoC and vestibular migraine may be artificial — BPVoC in an 8-year-old with headache during some episodes and a strong family history of migraine is likely early vestibular migraine. The distinction matters for treatment: VM in older children may warrant a more aggressive preventive approach. [1,19]

Epilepsy: Panayiotopoulos Syndrome and Ictal Vertigo

Benign occipital epilepsy of childhood — particularly Panayiotopoulos syndrome — is a critical differential diagnosis because it presents with episodic events that may include vertigo, eye deviation, nausea, and pallor. Key distinguishing features include: longer episode duration (often 5–30 minutes), autonomic features (ictal vomiting is characteristic of Panayiotopoulos), impaired consciousness during episodes, post-ictal confusion or sleepiness, and abnormal EEG (occipital spike-wave discharges). A child with possible BPVoC who has any episodes with altered consciousness or prolonged duration exceeding 10 minutes should have an EEG. Ictal vertigo from temporal lobe epilepsy is rare in children but also merits consideration. [20,21]

BPPV in Children, Posterior Fossa Pathology, and Cardiac Causes

Benign paroxysmal positional vertigo (BPPV) does occur in children, though it is less common than in adults. The key distinguishing feature is the positional trigger — symptoms provoked by specific head movements and reproduced by the Dix-Hallpike manoeuvre with a characteristic upbeat-torsional nystagmus. BPVoC episodes are not position-triggered. Posterior fossa tumours (medulloblastoma, ependymoma, pilocytic astrocytoma) are the most important pathology to exclude in any child with episodic vertigo. Red flags — progressive course, persistent neurological signs, headache that wakes the child from sleep, cerebellar signs, abnormal eye movements, or papilloedema — mandate urgent MRI. Cardiac arrhythmia (particularly long QT syndrome) may present with episodic collapse that resembles vertigo; ECG is warranted when there is a syncopal quality to events. [22]

Important: Posterior fossa tumour must always be considered in a child with episodic vertigo. Any progressive course, cerebellar ataxia in the interictal period, persistent nystagmus, headache on waking, or papilloedema mandates urgent MRI brain. Never apply the BPVoC label to a child who has not had a thorough interictal neurological examination.

Additional Differentials and Figure 2

Other diagnoses to consider include orthostatic hypotension (symptoms provoked by standing, ameliorated by lying down; confirm with lying and standing BP), psychogenic or functional dizziness (PPPD) in school-age children (continuous or near-continuous symptoms, school avoidance, anxiety features, and normal vestibular testing), and perilymph fistula following head trauma (fluctuating hearing loss with positional symptoms). The differential diagnosis is summarised in Figure 2.

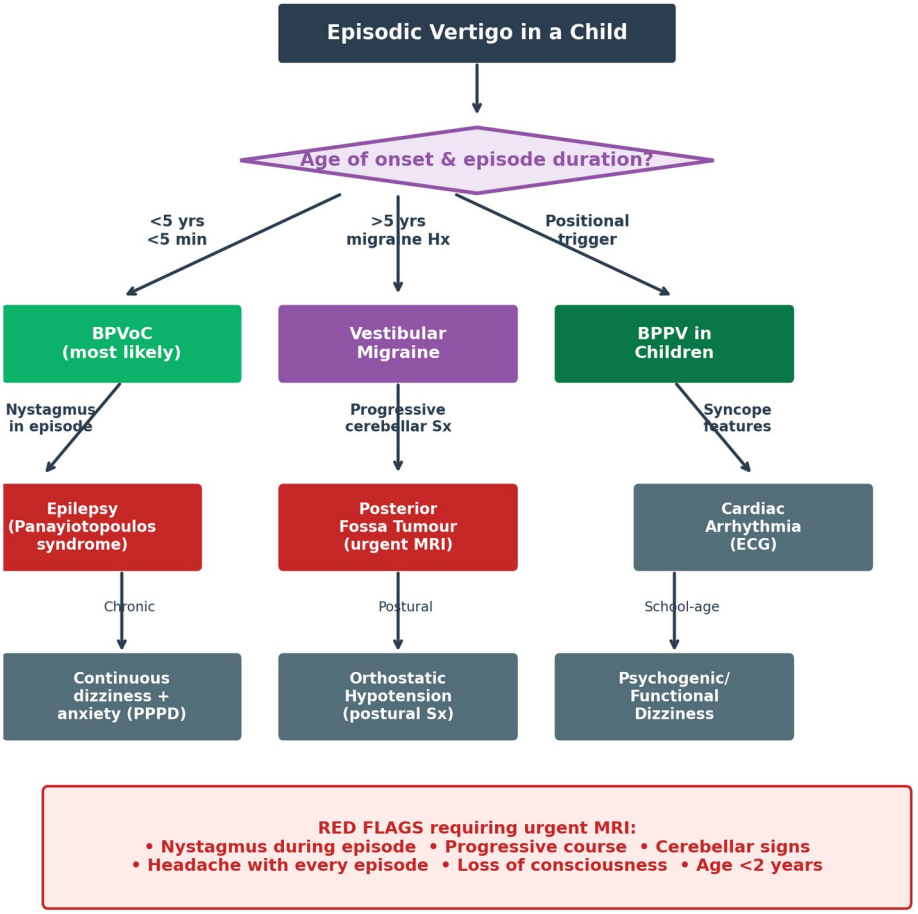

Figure 2. Differential Diagnosis of Episodic Vertigo in Children — Decision tree distinguishing BPVoC from vestibular migraine, BPPV, epilepsy, posterior fossa pathology, and other causes based on age, duration, triggers, and associated features.

VI. Diagnostic Workup: What to Investigate and Why

History: The Cornerstone of Diagnosis

In BPVoC, as in vestibular migraine, the history is the diagnostic instrument. A structured paediatric vestibular history should establish: exact episode duration (seconds, minutes, or hours), frequency over the past 3 months, whether episodes are increasing or decreasing in frequency, the presence of any prodrome or postdrome, associated symptoms (nausea, vomiting, pallor, nystagmus witnessed by carer, photophobia in older children), positional or exertional triggers, family history of migraine (maternal, paternal, first-degree relatives), and the child's developmental history including any motor delay. The HEADS-FIRST acronym (History, Episode character, Associated symptoms, Duration, Sleep, Family history, Investigations, Triggers) provides a structured framework for the paediatric vestibular history. [5,7]

The family history is particularly valuable. A maternal or paternal history of migraine — especially if the migraine included vestibular symptoms — significantly raises the prior probability of BPVoC. A family history of episodic ataxia or hemiplegic migraine raises the possibility of a CACNA1A channelopathy and

may prompt genetic referral. Absence of any family migraine history does not exclude BPVoC, but should increase vigilance for alternative diagnoses. [8]

Clinical Pearl: Ask parents to video an episode on their smartphone. Video evidence of the child's posture, eye movements, level of consciousness, and behaviour during an episode is often the most diagnostically informative piece of information in the entire workup — and it costs nothing.

Physical Examination and Vestibular Function Testing

The interictal neurological and vestibular examination must be normal in BPVoC. Examination should include: full cranial nerve assessment, cerebellar tests (finger-nose, heel-shin, rapid alternating movements — age-appropriate), assessment of gait and tandem gait, the head impulse test (age-appropriate vHIT or bedside head impulse), cover-uncover test and pursuit, fundoscopy for papilloedema in children with headache, and tympanometry. Video head impulse testing (vHIT) is feasible in cooperative children from approximately age 4 upward, and normative data by age are available. In BPVoC, vHIT is expected to be normal. [23]

Vestibular evoked myogenic potentials (cVEMP and oVEMP) are typically normal in BPVoC, though some studies have reported subtle asymmetries in a subset of children — these findings have not been shown to alter management and should not be over-interpreted. Caloric testing — technically challenging in preschool children — is generally normal but may show mild asymmetries in some series, again of uncertain clinical significance. The most important role of VFT in BPVoC workup is exclusion: an abnormal vHIT or caloric result should prompt reconsideration of the diagnosis, not confirmation of it. [24,25]

Clinical Insight: Vestibular function tests in the interictal period are typically normal in BPVoC. An abnormal vHIT (reduced gain with corrective saccades) or a significantly asymmetric caloric response in a child with episodic vertigo should prompt serious reconsideration of the diagnosis — consider vestibular neuritis with incomplete recovery, enlarged vestibular aqueduct syndrome, or perilymph fistula.

Audiometry, Neuroimaging, EEG, and Bloods

A baseline audiogram is recommended in all children with episodic vertigo, primarily to exclude hearing loss — which, if present, immediately shifts the diagnosis away from BPVoC toward Ménière's disease, enlarged vestibular aqueduct syndrome, or autoimmune inner ear disease. Hearing is expected to be entirely normal in BPVoC. MRI brain with gadolinium is not routinely required for a child who meets ICHD-3 criteria for BPVoC with a normal interictal examination, no red flags, and a positive family history of migraine. Indications for MRI include: any red flag feature, atypical presentation, age under 2 years, or failure to respond to standard management over 6 months. EEG is indicated when episodes involve altered consciousness, post-ictal symptoms, or other epileptiform features. A fasting blood glucose is appropriate in young children where hypoglycaemic episodes are possible. ECG should be obtained if any syncopal quality is noted. [22,26]

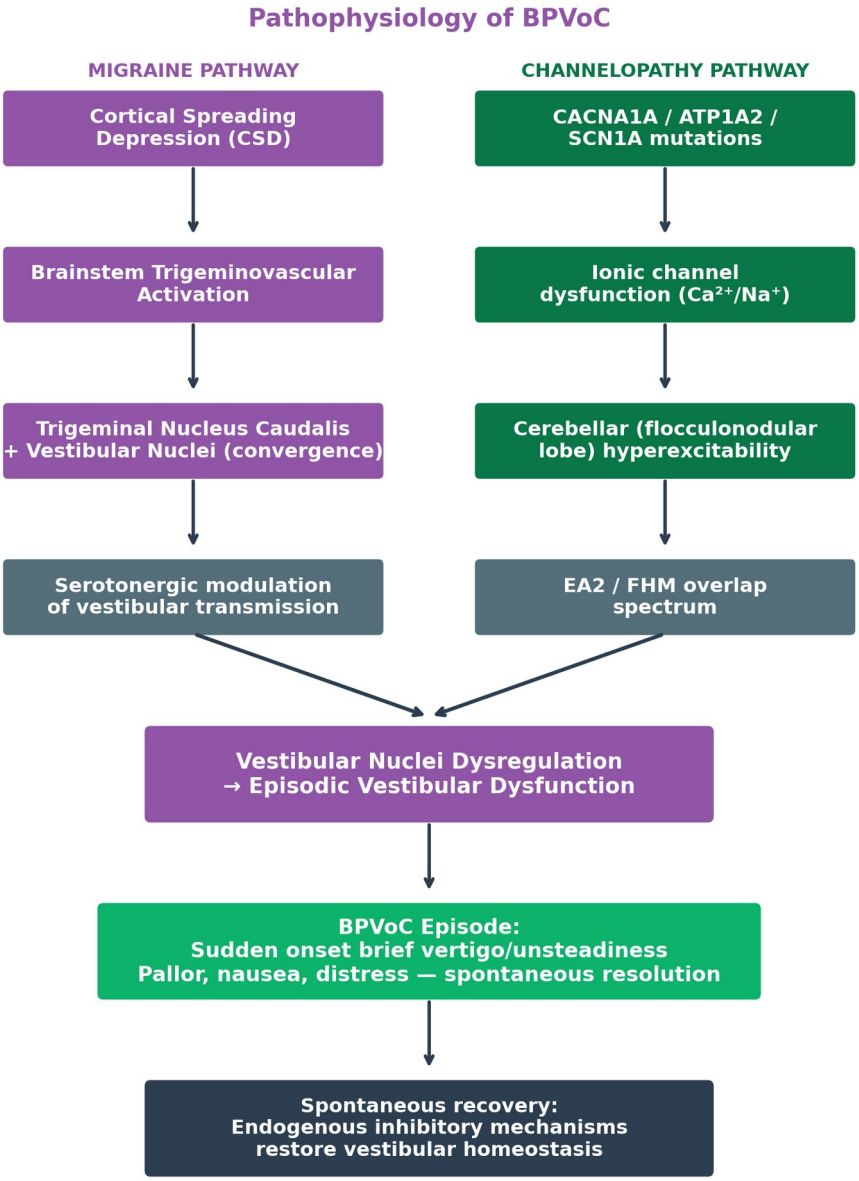

Figure 4. Diagnostic Workup Algorithm for BPVoC — Stepwise investigation pathway from history and examination through vestibular function testing to neuroimaging, stratified by red flag status.

VII. Management: Acute Episode and Preventive Strategies

Acute Episode Management

The acute management of a BPVoC episode primarily consists of parental reassurance, gentle physical support for the child (sitting or lying in a comfortable position in a low-stimulus environment), and waiting for spontaneous resolution. The typical episode is so brief — under 5 minutes — that pharmacological intervention is rarely initiated before the episode has already resolved. For more prolonged or more distressing episodes, antihistamines with vestibulolytic and antiemetic properties (notably promethazine hydrochloride, 6.25–12.5 mg orally or PR in children over 2 years, depending on weight) may be considered as rescue medication, though evidence for this indication specifically in BPVoC is extrapolated from adult vestibular migraine management rather than paediatric RCTs. [27]

Parental and family education is the most important acute management strategy. Parents should be coached to: remain calm during an episode, position the child safely to prevent falls, document the episode duration and key features for the clinician record, and avoid rushing the child to hospital unless the episode lasts more than 15 minutes, consciousness is impaired, or there are accompanying

neurological symptoms. A written action plan for parents significantly reduces ED presentations and parental anxiety in the published paediatric vestibular migraine literature, and the same principle applies to BPVoC. [5]

Clinical Pearl: Most families with a child newly diagnosed with BPVoC need three things: a clear diagnosis with an accessible explanation, written information about what to do during an episode, and a follow-up appointment. Pharmacotherapy should be offered only to the minority with frequent or functionally impairing episodes.

Non-Pharmacological Prevention

Non-pharmacological preventive strategies are the first-line approach for all children with BPVoC, regardless of frequency. These strategies directly target known migraine triggers and are without side-effects. The evidence base is extrapolated from paediatric migraine prevention trials, but the clinical rationale is strong given BPVoC's place on the migraine spectrum. Key strategies include: (1) sleep hygiene — consistent sleep and wake times, 10–12 hours per night in preschool children, avoidance of sleep deprivation (a potent migraine trigger); (2) dietary trigger avoidance — reduction in tyramine-rich foods (aged cheeses, processed meats, fermented products), caffeine (in older children), and MSG; (3) adequate hydration — dehydration is a well-established migraine trigger; (4) screen time moderation — in keeping with paediatric guidelines; (5) regular physical activity and stress management in school-age children. [28]

Pharmacological Prevention: Indications and Agents

Pharmacological prevention should be considered when episodes occur at a frequency of 3 or more per month, or when episodes cause significant functional impairment — school absence, sleep disruption, or severe parental anxiety — regardless of absolute frequency. The evidence base for paediatric pharmacological migraine prevention, while improving, remains primarily derived from open-label trials and observational series rather than large randomised controlled trials. [28,29]

Cyproheptadine (a serotonin and histamine antagonist with Ca2+-channel modulating properties) is the most widely used first-line agent for BPVoC and paediatric migraine prevention in children under 10 years. Doses of 2–4 mg at night are typical, titrating to 0.25 mg/kg/day in two divided doses if needed. The main side effects are appetite stimulation (clinically useful in underweight children, less so in those with obesity) and sedation. Evidence from two RCTs in paediatric cyclic vomiting syndrome and open-label series in BPVoC and migraine supports its efficacy. Propranolol (0.5–1 mg/kg/day divided twice daily, maximum 4 mg/kg/day) is an option in children over 5 years without asthma, cardiac conduction abnormalities, or diabetes. Topiramate (1–2 mg/kg/day) has evidence in children over 6 years from the CHAMP trial, particularly when there are accompanying migraine features. Pizotifen (0.5 mg nocte in young children) is used in some Australian centres. [29,30]

Key Point: Most children with BPVoC — particularly those seen in a dedicated vestibular or paediatric neurology clinic — do not require pharmacotherapy. The diagnosis itself, accompanied by reassurance, a sleep hygiene plan, and trigger diary, reduces episode frequency in a significant proportion. Reserve pharmacotherapy for children with frequent episodes causing measurable functional impairment.

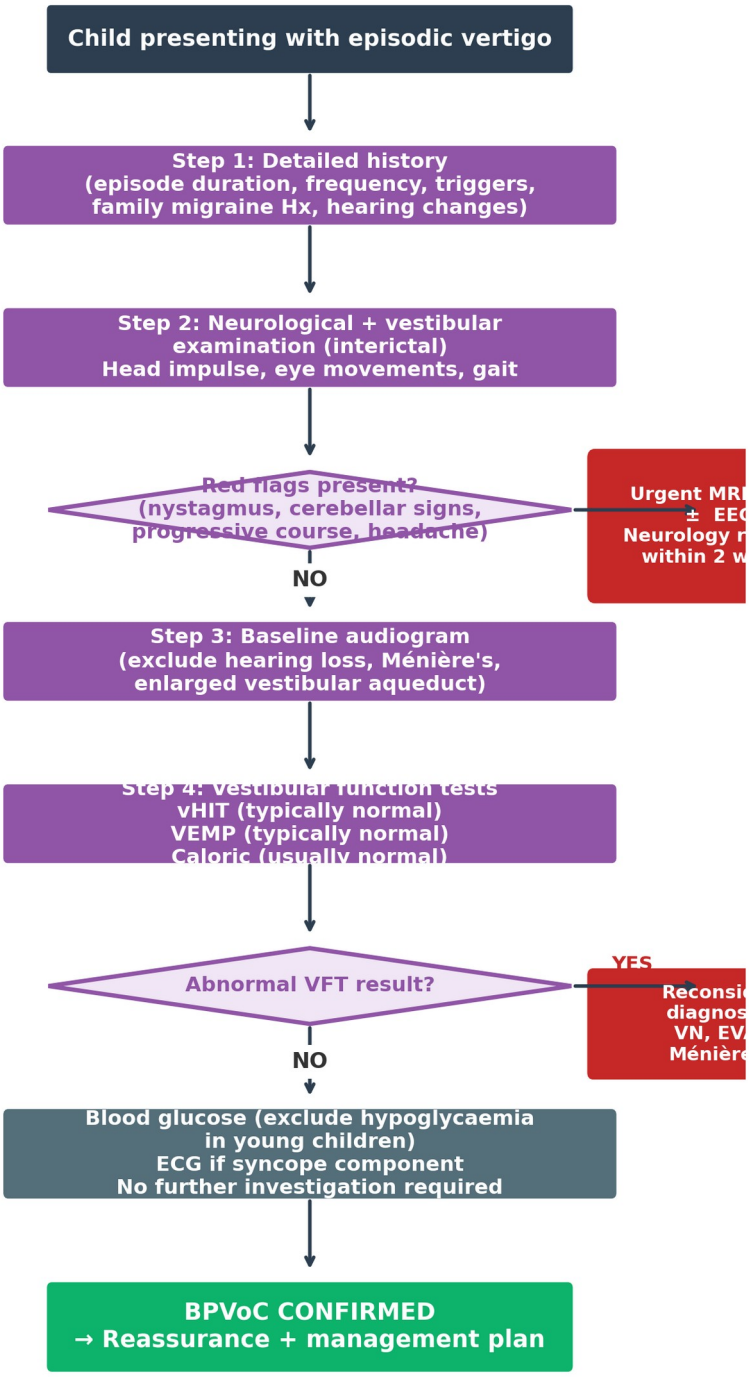

Figure 3. Pathophysiology of BPVoC — Dual pathway model showing cortical spreading depression and trigeminovascular activation (left arm) and ionic channelopathy contributions via CACNA1A/ATP1A2 (right arm), converging on vestibular nucleus dysregulation.

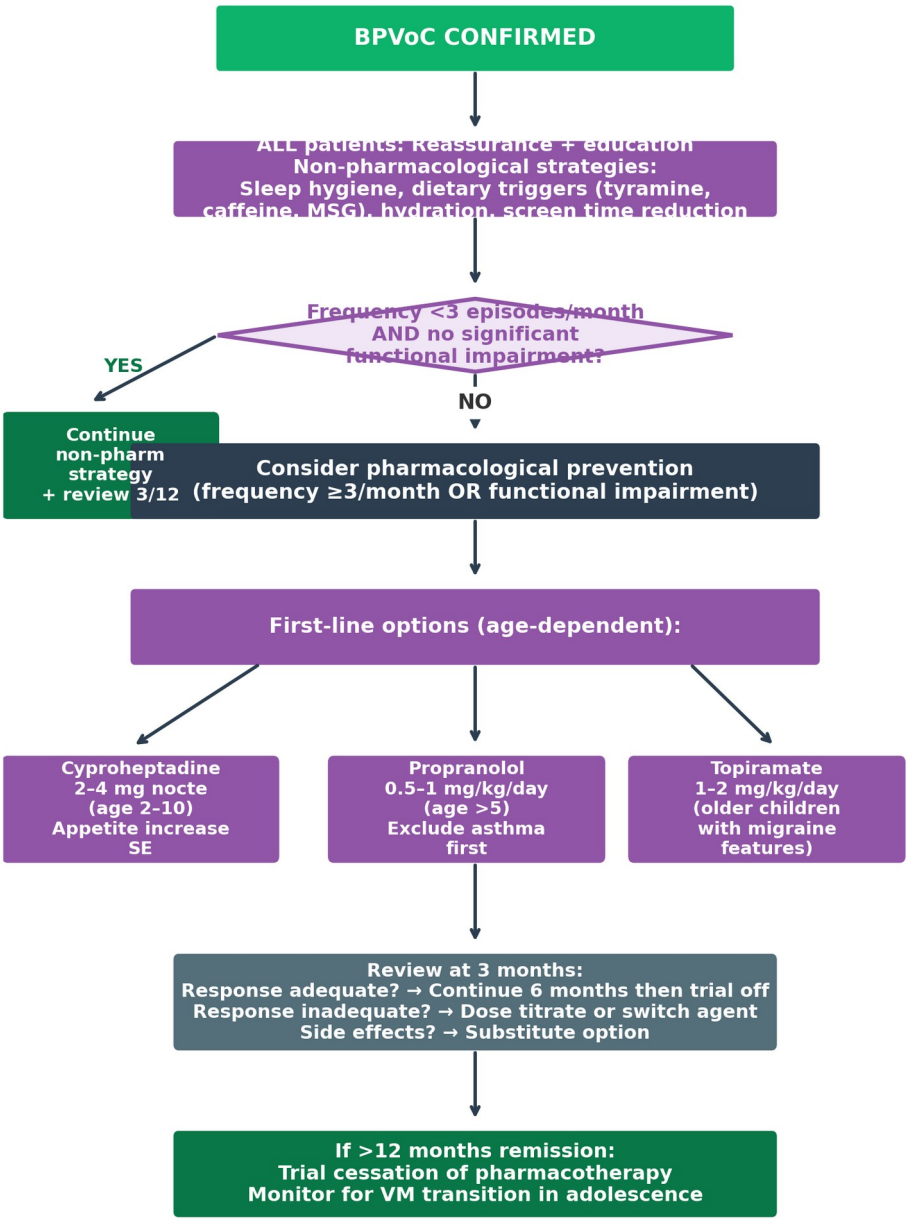

Figure 5. Management Algorithm for BPVoC — Decision tree from confirmed diagnosis through non-pharmacological strategies to pharmacological prevention, with medication options and review schedule.

VIII. BPVoC, Vestibular Migraine, and the Migraine Continuum

ICHD-3 Episodic Syndromes: A Unified Framework

The ICHD-3 classification formalises the concept of childhood periodic syndromes as a coherent group of migraine equivalents. BPVoC, cyclic vomiting syndrome (CVS), abdominal migraine, and benign paroxysmal torticollis of infancy share: a familial migraine background, episodic and paroxysmal onset, absence of identifiable structural or metabolic aetiology, age-appropriate clinical phenotype (vestibular symptoms when the vestibular system is the predominant expression; abdominal when the gastrointestinal autonomic system is predominant), and a tendency to transition to more typical migraine phenotypes as the nervous system matures. A single child may experience more than one episodic syndrome simultaneously or sequentially. [1]

The shared pathophysiology across these episodic syndromes — involving CSD, trigeminovascular sensitisation, ionic channel dysfunction, and hypothalamic modulation of episodic thresholds — provides the mechanistic basis for the family of conditions. Genetic studies of multiplex migraine families have found the same CACNA1A mutations cosegregating with BPVoC in children and vestibular migraine in their parents, providing direct evidence of the BPVoC-to-VM continuum at the genetic level. Understanding this continuum is not merely academic — it directly informs the management approach (lifestyle modification, trigger avoidance, migraine-specific preventives) and the counselling given to families. [8,13]

Key Point: BPVoC and vestibular migraine are points on the same paediatric migraine spectrum. The distinction is largely age-related: BPVoC is the age-specific phenotype in preschool and early school years, when the child cannot yet verbalise rotatory vertigo and when migraine features are absent or subclinical. As the nervous system matures, the full vestibular migraine phenotype emerges.

BPVoC-to-Vestibular Migraine Transition: Published Evidence

Longitudinal cohort data consistently show that a proportion of children with BPVoC will develop vestibular migraine. Langhagen and colleagues, in a prospective vestibular clinic cohort with a mean follow-up of 7 years, found that 21% of children initially diagnosed with BPVoC subsequently met ICHD-3 criteria for vestibular migraine. Risk factors for this transition included: older age at BPVoC onset (age 6 or above), strong maternal migraine history, high initial episode frequency (>4 per month), and persistence of episodic vertigo beyond age 8. Marcelli and colleagues reported similar transition rates in an Italian clinic cohort with 5-year follow-up. [9,10]

The clinical management at the point of transition from BPVoC to vestibular migraine involves: reclassifying the diagnosis, introducing or intensifying lifestyle preventive strategies, and reassessing the pharmacological prevention approach using evidence from the adult and adolescent vestibular migraine literature (where nutraceuticals — magnesium, vitamin B2 — and tricyclics have a stronger evidence base than in young children). Neuropsychological assessment should be considered in adolescents with vestibular migraine who have significant school impact or comorbid anxiety. [30]

Genetic Counselling Considerations

In families with a clear autosomal dominant inheritance pattern — multiple generations with BPVoC, vestibular migraine, episodic ataxia, or hemiplegic migraine — referral to a clinical genetics service should be considered. CACNA1A sequencing is the most diagnostically useful first-line test in such families. Identification of a pathogenic CACNA1A variant has both diagnostic and management implications: the affected child should be monitored for episodic ataxia features, and first-degree relatives should be counselled about the channelopathy spectrum. However, genetic testing is not indicated in sporadic BPVoC without a family history of the extended channelopathy spectrum. [13]

IX. Long-Term Outcomes and Transition to Adulthood

Resolution Rates and Residual Migraine Risk

The majority of children with BPVoC will achieve complete resolution of episodic vertigo episodes by the age of 8–10 years. Published series report resolution in 70–80% within 5 years of onset, with the remainder either transitioning to vestibular migraine or experiencing sporadic ongoing episodes at reduced frequency. Even in children who achieve apparent resolution, the underlying migraine biology persists — the lifetime risk of migraine (with or without aura) and vestibular migraine is substantially elevated compared to the general paediatric population. Adult follow-up data from the Abu-Arafeh cohort indicate that a significant proportion of BPVoC patients developed migraine with or without aura in adulthood. [2,10]

Families should be explicitly counselled at the time of the initial BPVoC diagnosis that resolution of childhood episodes is the expected outcome, but this does not confer lifelong freedom from migraine-spectrum disorders. Monitoring for migraine symptoms through adolescence — particularly around the time of puberty, when hormonal changes significantly lower migraine thresholds — is an important component of long-term management. Girls with a history of BPVoC should be counselled about the increased risk of menstrual migraine. [10]

Clinical Insight: Advise families at first diagnosis: 'We expect the BPVoC episodes to resolve by about age 8 to 10 in most children. But the underlying migraine tendency does not disappear — we recommend keeping an eye on headaches and dizziness through the teenage years, particularly at puberty.' This sets accurate expectations and keeps families engaged with follow-up.

Psychological Sequelae and School Impact

Episodic vertigo in childhood carries a psychological burden that is often underestimated. Children who experience frequent, unpredictable episodes of distress and unsteadiness may develop anticipatory anxiety, avoidance behaviours, and school reluctance. In school-age children, this may manifest as refusal to participate in physical education, reluctance to attend excursions or unfamiliar environments, and school absence during periods of high episode frequency. Systematic screening for anxiety using age-appropriate tools (for example, the Spence Children's Anxiety Scale) at the first clinic visit and at follow-up is recommended, particularly in children with high episode frequency or prolonged diagnostic delay. [31]

Referral to a paediatric psychologist is indicated for children with significant anxiety or avoidance behaviour. Cognitive-behavioural therapy (CBT) approaches adapted for paediatric vestibular disorders have shown efficacy in adult PPPD; similar principles — graded activity, exposure, and anxiety management — can be applied in older children and adolescents. Liaison with the school (with parental consent) to implement a vestibular management plan may reduce absenteeism and academic impact. [31]

Multidisciplinary Management and Transition of Care

Optimal management of BPVoC and the BPVoC-to-VM transition requires a multidisciplinary team that may include: a vestibular physician (coordinating the diagnostic pathway and pharmacological prevention), paediatric neurology (if epilepsy is on the differential, or for complex pharmacological management), audiology (baseline and monitoring audiometry), vestibular physiotherapy (in children with persistent postural instability or who have transitioned to vestibular migraine with interictal symptoms), and paediatric psychology (for anxiety and school avoidance). In most cases of straightforward BPVoC, the entire management pathway is adequately handled by the vestibular physician or general paediatrician in conjunction with the GP. [5,27]

Clinical Pearl: The transition from paediatric to adult care is a vulnerable period for young people with vestibular migraine. A structured transition document — summarising the diagnosis, pharmacological history, trigger profile, and current management — should be provided to the young person and their adult care provider at the point of transition, ideally at age 17 or 18.

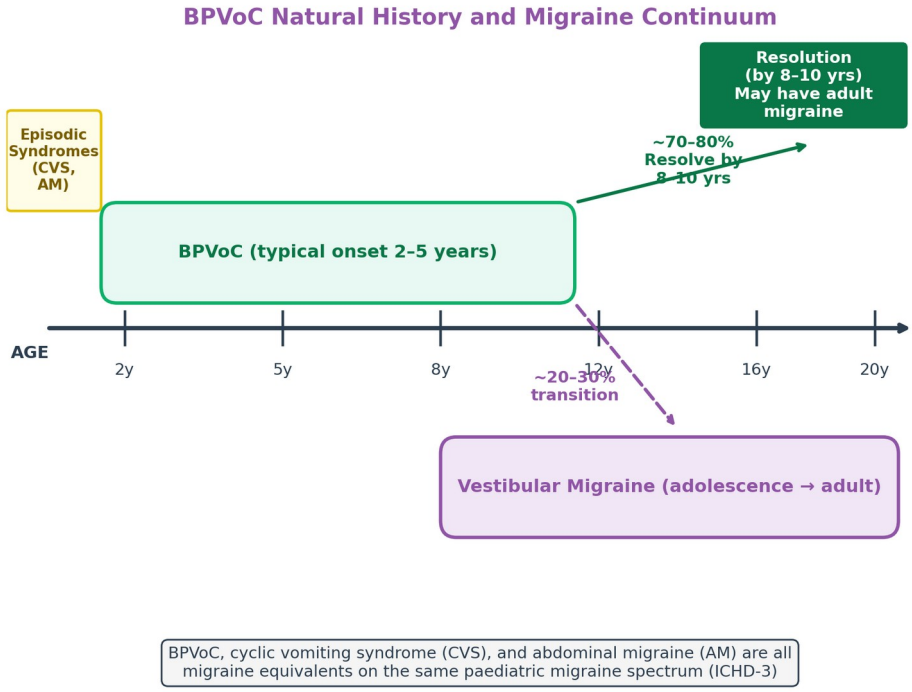

Figure 6. BPVoC Natural History and Migraine Continuum — Timeline from typical BPVoC onset at age 2–5 years through resolution pathway (70–80%) and transition to vestibular migraine pathway (20–30%), with the paediatric episodic migraine equivalents shown.

X. Summary and Key Clinical Takeaways

Ten Core Clinical Points from This Review

• BPVoC is classified in ICHD-3 as an episodic syndrome associated with migraine — it is not a peripheral vestibular disorder. Management follows the migraine framework.

• The typical presentation is in children aged 2–5 years: brief episodes (under 5 minutes) of distress, unsteadiness, pallor, and nausea, with complete spontaneous resolution and a normal interictal examination.

• In toddlers, BPVoC manifests as crying, clinging, and unsteadiness — not verbalised rotatory vertigo. A careful structured history from the caregiver is essential for diagnosis.

• A strong family history of migraine (in 60–80% of cases) is the most important corroborating historical feature. In families with episodic ataxia or hemiplegic migraine, consider CACNA1A channelopathy.

• The interictal vestibular examination — vHIT, VEMP, caloric — is expected to be normal. An abnormal VFT result should prompt reconsideration of the diagnosis.

• MRI brain is not routinely required but is mandatory for any child with red flags: progressive course, cerebellar signs, nystagmus in the interictal period, headache on waking, or papilloedema.

• Non-pharmacological strategies (sleep hygiene, dietary trigger avoidance, hydration) are first-line prevention for all children. Most families do not require pharmacotherapy.

• When pharmacotherapy is indicated (frequency ≥3/month or significant functional impairment), cyproheptadine is the preferred first-line agent in children under 10 years.

• Approximately 20–30% of children with BPVoC will transition to vestibular migraine in adolescence. Long-term follow-up and adolescent monitoring are part of good clinical care.

• Psychological sequelae — anxiety, school avoidance — should be screened for at every visit. Paediatric psychological support is indicated for children with significant avoidance behaviour.

Signpost to PVM03

The next topic in the Vestibular Medicine in Children series — PVM03: Vestibular Migraine in Children — covers the diagnostic criteria, pathophysiology, and management of vestibular migraine specifically in the

paediatric population, including the transition from BPVoC and the challenges of differentiating VM from other causes of episodic vertigo in school-age children. Clinicians managing children with BPVoC transitioning to vestibular migraine should proceed to PVM03 as the next reference in this series.

References

1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1–211.

2. Abu-Arafeh I, Russell G. Paroxysmal vertigo as a migraine equivalent in children: a population-based study. Cephalalgia. 1995;15(1):22–25.

3. Basser LS. Benign paroxysmal vertigo of childhood (a variety of vestibular neuronitis). Brain. 1964;87(1):141–152.

4. Giffin NJ, Benton S, Goadsby PJ. Benign paroxysmal torticollis of infancy: four new cases and linkage to CACNA1A mutation. Dev Med Child Neurol. 2002;44(7):490–493.

5. Wiener-Vacher SR, Quarez J, Siquier-Gauthier A. Epidemiology of vestibular disorders in a paediatric vestibular clinic in France. Clin Otolaryngol. 2019;44(4):518–527.

6. Lindskog U, Odkvist L, Noaksson L, Wallquist J. Benign paroxysmal vertigo in childhood: a long-term follow-up. Headache. 1999;39(1):33–37.

7. Wiener-Vacher SR. Vestibular disorders in children. Int J Audiol. 2008;47(9):578–583.

8. Ophoff RA, Terwindt GM, Vergouwe MN, et al. Familial hemiplegic migraine and episodic ataxia type-2 are caused by mutations in the Ca2+ channel gene CACNL1A4. Cell. 1996;87(3):543–552.

9. Langhagen T, Albers L, Heinen F, et al. Period prevalence of dizziness and vertigo in adolescents. PLoS One. 2015;10(9):e0136512.

10. Marcelli V, Russo A, Cristiano E, Tessitore A. Benign paroxysmal vertigo of childhood: long-term outcome and predictors of progression. Cephalalgia. 2020;40(8):819–825.

11. Charles A. Migraine. N Engl J Med. 2017;377(6):553–561.

12. Furman JM, Balaban CD. Vestibular migraine. Ann N Y Acad Sci. 2015;1343(1):90–96.

13. Pietrobon D. Familial hemiplegic migraine. Neurotherapeutics. 2007;4(2):274–284.

14. Vecchia D, Pietrobon D. Migraine: a disorder of brain excitatory-inhibitory balance? Trends Neurosci. 2012;35(8):507–520.

15. Kors EE, Terwindt GM, Vermeulen FL, et al. Delayed cerebral edema and fatal coma after minor head trauma: role of the CACNA1A calcium channel subunit gene and relationship with familial hemiplegic migraine. Ann Neurol. 2001;49(6):753–760.

16. Tighilet B, Lacour M. Serotonin modulates the activity of the medial vestibular nucleus: involvement of serotonin2A receptor-mediated activation of L-type calcium channels. Eur J Neurosci. 2001;14(12):1952–1960.

17. Balatsouras DG, Kaberos A, Assimakopoulos D, et al. Etiology of vertigo in children. Int J Pediatr Otorhinolaryngol. 2007;71(3):487–494.

18. Casani AP, Dallan I, Cerchiai N, Lenzi R, Cosottini M, Sellari-Franceschini S. Posterior fossa arachnoid cysts presenting as positional vertigo: three case reports and literature review. Otol Neurotol. 2013;34(1):e1–e5.

19. Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. J Vestib Res. 2012;22(4):167–172.

20. Ferrie CD, Caraballo R, Covanis A, et al. Autonomic status epilepticus in Panayiotopoulos syndrome and other childhood and adult epilepsies: a consensus view. Epilepsia. 2007;48(6):1165–1172.

21. Covanis A. Panayiotopoulos syndrome: a benign childhood autonomic epilepsy frequently imitating encephalitis, syncope, migraine, sleep disorder, or gastroenteritis. Pediatrics. 2006;118(4):e1237–e1243.

22. Riina N, Ilmari P, Kentala E. Vertigo and imbalance in children: a retrospective study in a Helsinki University otorhinolaryngology clinic. Arch Otolaryngol Head Neck Surg. 2005;131(11):996–1000.

23. Janky KL, Givens D. Vestibular, visual acuity, and balance outcomes in children with cochlear implants: a preliminary report. Ear Hear. 2015;36(6):e364–e372.

24. Maes L, De Kegel A, Van Waelvelde H, Dhooge I. Rotatory and collic vestibular evoked myogenic potential testing in normal-hearing and hearing-impaired children. Ear Hear. 2014;35(2):e21–e32.

25. Valente M. Maturational effects of the vestibular system: a study of rotary chair, computerized dynamic posturography, and vestibular evoked myogenic potentials with children. J Am Acad Audiol. 2007;18(6):461–481.

26. Pavone P, Longo MR, Parano E, et al. Benign paroxysmal vertigo of childhood: clinical aspects and EEG findings. Brain Dev. 2016;38(3):274–279.

27. Weisleder P, Fife TD. Dizziness and headache: a common association in children and adolescents. J Child Neurol. 2001;16(10):727–730.

28. Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S. Pathophysiology of migraine: a disorder of sensory processing. Physiol Rev. 2017;97(2):553–622.

29. Powers SW, Coffey CS, Chamberlin LA, et al. Trial of amitriptyline, topiramate, and placebo for pediatric migraine. N Engl J Med. 2017;376(2):115–124.

30. Fotuhi M, Glaun B, Quan SY, Sofare T. Vestibular migraine: a critical review of treatment trials. J Neurol. 2009;256(5):711–716.

31. Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the committee for the classification of vestibular disorders of the Bárány Society. J Vestib Res. 2017;27(4):191–208.

Disclaimer and Copyright

This literature review is published by Australian Dizziness Clinics for the continuing professional development of healthcare clinicians. It does not constitute individualised medical advice and is not intended for lay distribution. Clinical decisions must be made in the context of each clinician's professional judgement and the specific circumstances of each patient.