Benign Paroxysmal Positional Vertigo in Children:

Pathophysiology, Diagnosis, and Canal Repositioning Techniques

Vestibular Medicine in Children

Topic 4 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.

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Canal	Provocative test	Nystagmus finding	Latency
Posterior canal (90%)	Dix-Hallpike	Upbeat-torsional; fatigable; reverses on sitting	1–5 sec
Horizontal — canalolithiasis	Supine roll	Geotropic direction-changing; stronger to affected ear	Immediate
Horizontal — cupulolithiasis	Supine roll	Apogeotropic direction-changing; stronger to unaffected ear	Immediate
Anterior canal (rare)	Dix-Hallpike	Downbeat-torsional nystagmus	1–5 sec
Modified technique <8 years	Slower positioning; smaller head extension; parental support	Same latency; cooperation variable	Age-appropriate

Precipitant	Frequency in paediatric BPPV	Clinical notes
Head trauma / concussion	40–50%	Most common; BPPV may appear days–weeks post-injury
Vestibular neuritis	10–15%	Develops 4–8 weeks post-acute phase
Vestibular migraine	10–15%	Recurrent BPPV; VM prevention reduces frequency
Idiopathic	20–25%	Diagnosis of exclusion; always seek precipitant in children
EVA / labyrinthine malformation	5–10%	Part of episodic inner ear dysfunction; investigate for EVA
Ototoxicity	Rare	Post-aminoglycoside; during compensation phase

Diagnosis	Key differentiating feature
Vestibular migraine	Duration minutes–hours; no Dix-Hallpike positivity; headache/photophobia
BPVC	Age <6; seconds; spontaneous; no Dix-Hallpike positivity; pallor
Central positional vertigo	No latency; no fatigability; direction-changing in same head position
Orthostatic hypotension	Triggered by standing; BP drop; no nystagmus; presyncope not vertigo

PPPD	Dix-Hallpike triggers symptoms but no nystagmus; constant background dizziness

V. Diagnostic Manoeuvres: Dix-Hallpike, Supine Roll, and Adaptations for Children

Canal	Manoeuvre	Key steps	Success rate (single treatment)
Posterior canal	Epley manoeuvre	Dix-Hallpike position → head 90° opposite → body roll → sit up; 30–60 sec each	70–80%
Posterior canal (alt)	Semont liberatory	Rapid positioning affected side then rapid flip opposite	60–70%
Horizontal — canalolithiasis	Barbecue (360°) roll	Log roll: affected ear down → supine → unaffected → prone; 90° steps 1 min each	70–80%
Horizontal — cupulolithiasis	Forced prolonged position	Lie on unaffected side 12 hours; converts to canalolithiasis; then BBQ roll	Variable

VI. Canal-Specific Repositioning Manoeuvres

VII. Special Situations: Bilateral BPPV, Horizontal Canal BPPV, Recurrent BPPV

Aspect	Recommendation
First treatment	Epley in clinic; advise transient nausea during manoeuvre
Repeat treatment	Positive Dix-Hallpike at 1 week → repeat; consider canal conversion
Vestibular suppressants	NOT recommended — impair central compensation
Home Epley	Can teach parent/older child; confirm canal variant first
Recurrence	10–15% recur at 1 year; re-treat; ≥3 recurrences → vestibular physician
Precipitant treatment	Treat VM/post-neuritis — reduces recurrence frequency

VIII. Secondary BPPV: Causes and Investigation

IX. Outcomes, Recurrence, and Follow-Up

Indication	Urgency	Refer to
BPPV not resolving after 3 Epley manoeuvres	Routine	Vestibular physician; canal variant assessment
Recurrence ≥3 per year	Routine	Vestibular physician; investigate underlying cause
Post-concussion BPPV with persistent concussion symptoms	Routine	Concussion clinic + VOMS assessment

Central positional nystagmus features (no latency, no fatigability)	Urgent	MRI brain; neurology
BPPV + unilateral SNHL	Soon	EVA/labyrinthine malformation workup; paediatric ENT + MRI temporal bones

X. Summary and Key Clinical Takeaways

References

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I. Introduction: BPPV in the Paediatric Context

Benign paroxysmal positional vertigo (BPPV) is the most common cause of peripheral vertigo in adults, but its presentation in children differs in important ways that demand clinical attention. While BPPV can occur across all paediatric age groups, it is most commonly identified in school-aged children between 8 and 14 years and is distinctly uncommon in children under 5 years of age. This epidemiological pattern itself carries diagnostic significance: a young child presenting with apparent BPPV should prompt a systematic search for an underlying secondary cause. [1,2]

In adults, the majority of BPPV cases are idiopathic, attributed to age-related degeneration of the otolithic membrane and spontaneous detachment of otoconia. In children, this degenerative mechanism is largely absent. Instead, BPPV in the paediatric population tends to be either truly idiopathic (less common) or secondary to a recognisable precipitant — most frequently head trauma, enlarged vestibular aqueduct syndrome (EVAS), or prior viral labyrinthitis. The distinction between idiopathic and secondary BPPV has direct implications for investigation and long-term management. [2,3]

This literature review provides vestibular physicians, paediatricians, and emergency physicians with a comprehensive, evidence-based framework for the diagnosis and management of BPPV in children. It covers the pathophysiology of canalolithiasis and cupulolithiasis in the developing inner ear, the diagnostic application of positional testing adapted for paediatric practice, canal-specific repositioning manoeuvres, and an algorithmic approach to secondary BPPV investigation. Special attention is paid to the clinical challenges of performing and interpreting positional testing in young or anxious children.

> □ **Clinical Pearl:** BPPV in a child under 8 years is almost always secondary — investigate for an underlying cause including enlarged vestibular aqueduct, post-traumatic changes, or prior viral labyrinthitis before accepting an idiopathic diagnosis.

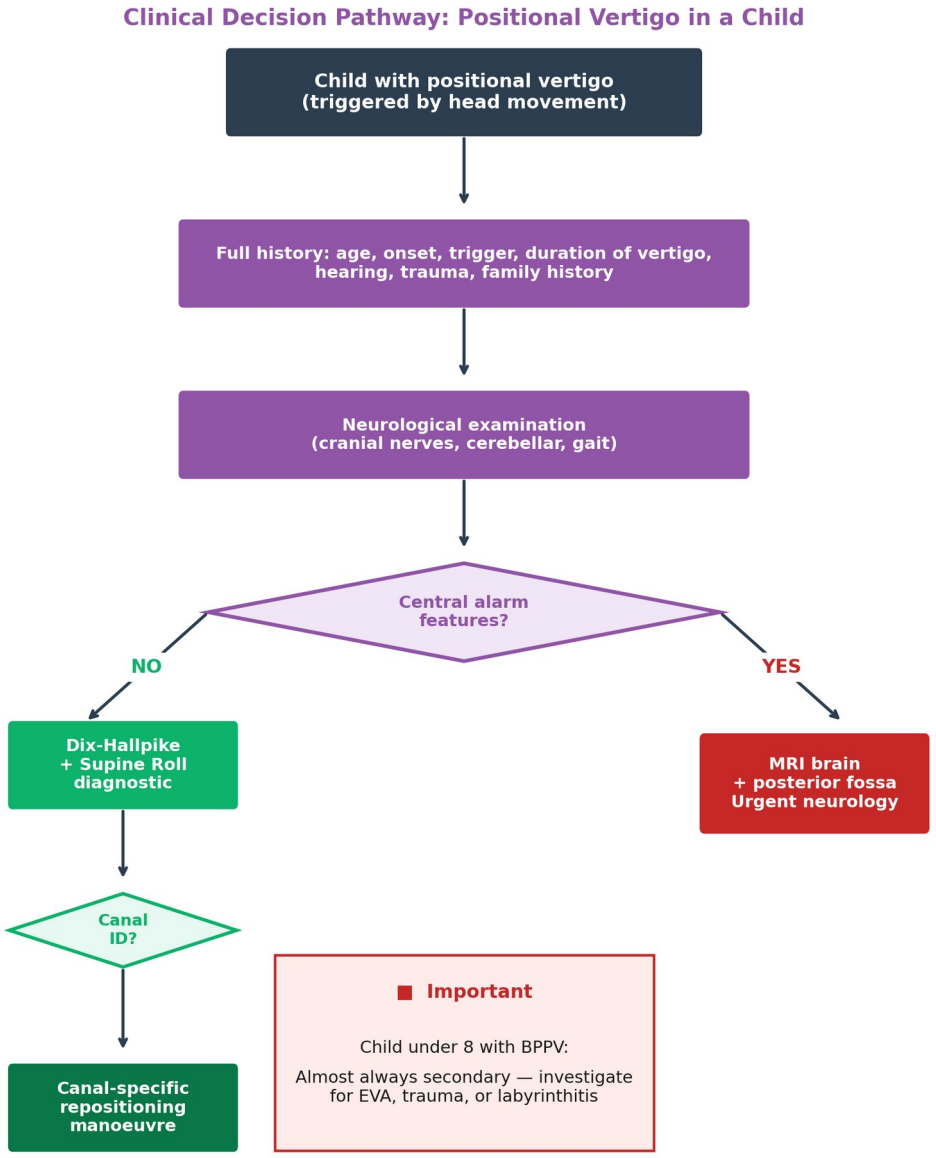

Figure 1. Clinical Decision Pathway for Positional Vertigo in a Child — structured approach from presentation through diagnostic testing to canal-specific management.
Source: Australian Dizziness Clinics — clinical flowchart.

II. Epidemiology: How Common is BPPV in Children?

Paediatric BPPV is substantially less common than its adult counterpart, and population-based epidemiological data in children remain sparse. The most comprehensive series have emerged from tertiary vestibular referral centres, and these studies suggest that BPPV accounts for approximately 5–17% of paediatric vestibular diagnoses, compared with 20–30% in adult vestibular clinics. [3,4]

Age and Sex Distribution

Korres and colleagues identified a paediatric BPPV cohort in which the mean age at presentation was 11.3 years, with the majority of cases occurring between 8 and 16 years. Cases under 5 years were rare and were predominantly associated with secondary causes. In contrast to the adult literature, where BPPV exhibits a clear female preponderance (approximately 2:1), the sex distribution in paediatric BPPV is more balanced, with several series reporting near-equal male-to-female ratios. [3,5]

Choung and colleagues described a cohort of 48 children with BPPV and found that the posterior semicircular canal was involved in 85% of cases, consistent with adult data. The horizontal canal

accounted for approximately 12% of cases, and anterior canal BPPV was rare at around 3%. Importantly, the proportion of secondary BPPV in paediatric series ranges from 20–50%, significantly higher than in adult cohorts where secondary causes account for less than 15% of presentations. [4,6]

Recurrence Rates

Recurrence rates in children with BPPV are generally reported to be lower than in adults. While adult BPPV has a 1-year recurrence rate of approximately 20–30%, paediatric series report recurrence rates of 10–20% at one year. Cases associated with secondary causes — particularly EVA and recurrent traumatic BPPV — carry higher recurrence rates and may require ongoing surveillance and vestibular rehabilitation. [4,7]

> □ **Key Point:** Paediatric BPPV accounts for 5–17% of vestibular diagnoses in children. The posterior semicircular canal is most commonly affected (85%). Secondary causes are present in up to 50% of paediatric cases — significantly higher than in adult populations.

III. Pathophysiology: Canalolithiasis, Cupulolithiasis, and Canal Anatomy in Children

BPPV arises from the inappropriate presence of otoconia — calcium carbonate crystals normally anchored to the otolithic membranes of the utricle and saccule — within the semicircular canals (SCC). Two distinct pathophysiological mechanisms have been characterised: canalolithiasis, in which free-floating otoconia move within the endolymphatic fluid of the SCC lumen; and cupulolithiasis, in which otoconia adhere directly to the cupula of an ampulla. [8,9]

Canalolithiasis

Canalolithiasis is the predominant mechanism in BPPV across both adult and paediatric populations. When the head is moved into a provocative position, free-floating otoconia within the SCC lumen are displaced by gravity, creating abnormal endolymph flow (ampullofugal or ampullopetal depending on canal orientation) and a deflection of the cupula that does not correspond to actual head velocity. This mismatch between the vestibular signal and proprioceptive/visual inputs produces the characteristic nystagmus and subjective vertigo. The nystagmus generated by canalolithiasis is typically transient (5–30 seconds), geotropic for the posterior SCC, and fatigable with repeated testing. [8,9]

Cupulolithiasis

Cupulolithiasis, in which otoconia adhere to the cupula, renders the cupula inappropriately gravity-sensitive. This produces persistent nystagmus (rather than transient) with a position-dependent pattern. Cupulolithiasis of the horizontal SCC produces the apogeotropic variant of horizontal BPPV — characterised by nystagmus that beats away from the ground (toward the ceiling) when the head is turned to either side in the supine position, with persistent rather than transient duration. Cupulolithiasis of the posterior SCC is uncommon. [8]

Canal Anatomy and Otoconia in Children

The semicircular canal geometry in children is functionally adult-equivalent by school age, though otoconia composition and anchoring may differ in younger children. Otoconia in children are subject to fragmentation and detachment through traumatic mechanisms (the most common secondary cause of paediatric BPPV), inflammatory processes (post-viral labyrinthitis), and structural vulnerability in EVA, where fragile otoconia are predisposed to spontaneous release. [6,10]

In EVA, mutations in the SLC26A4 gene (encoding pendrin) are associated with abnormal endolymphatic hydrops, increased fragility of the otolithic membranes, and a significantly elevated risk of BPPV — occurring at a much younger age than idiopathic BPPV. Children with EVA may experience recurrent BPPV episodes, often triggered by minor head trauma or pressure changes (Valsalva, diving), due to repeated otoconia fragmentation. [6,10]

Nystagmus Characteristics by Affected Canal

Canal	Test Used	Nystagmus Direction	Duration	Fatigable?
Posterior SCC	Dix-Hallpike	Upbeat-torsional (geotropic)	5–30 sec	Yes
Horizontal SCC (Canalolithiasis)	Supine Roll	Horizontal Geotropic	10–60 sec	Yes
Horizontal SCC (Cupulolithiasis)	Supine Roll	Horizontal Apogeotropic	Persistent	No
Anterior SCC	Dix-Hallpike	Downbeat-torsional	5–20 sec	Yes

■ Clinical Pearl: In children, nystagmus may be less pronounced than in adults. Upbeat-torsional nystagmus on Dix-Hallpike is pathognomonic for posterior SCC BPPV.

Figure 2. Nystagmus Characteristics by Affected Canal — diagnostic features of posterior and horizontal SCC BPPV including nystagmus direction, duration, and fatigability.
Source: Australian Dizziness Clinics — clinical flowchart.

□ **Clinical Insight:** The posterior SCC is the most gravitationally dependent canal in the upright and lying positions, making it the most common site of otoconia accumulation. The horizontal SCC becomes vulnerable when patients spend prolonged periods in recumbent positions — relevant in paediatric patients recovering from illness or hospitalisation.

IV. Clinical Features and Canal Identification

The clinical presentation of BPPV in children mirrors that of adults in its positional trigger and brief duration, but important differences exist in how children perceive and describe vestibular symptoms. Paediatric patients — particularly younger children — may lack the language to describe spinning vertigo and instead report symptoms such as feeling "wobbly", "my head hurts when I move", or may simply appear nauseated or reluctant to change position. This atypical symptom reporting can delay diagnosis, particularly if the examining clinician does not specifically elicit a positional trigger from both the child and the accompanying caregiver. [4,11]

Posterior SCC BPPV — Classic Presentation

The hallmark of posterior SCC BPPV is a brief (5–30 second) episode of vertigo and nausea triggered by head extension or turning to one side, typically when lying down, sitting up, or rolling over in bed. On Dix-Hallpike positioning, the characteristic nystagmus is upbeat-torsional — vertical upbeat with a torsional component toward the affected (down) ear. The nystagmus typically has a latency of 1–5 seconds after positioning, reaches its peak intensity within 5–10 seconds, and fatigues (diminishes in amplitude) with repeated testing. Associated symptoms include nausea, pallor, and occasionally vomiting — which may be more prominent in children than adults due to heightened autonomic sensitivity. [8,11]

Horizontal SCC BPPV — Presentation

Horizontal SCC BPPV presents with positional vertigo triggered by head rotation in the supine position (rolling in bed) or occasionally by rapid head turns. Nystagmus on Supine Roll testing is horizontal, with the pattern depending on the mechanism: geotropic (beating toward the floor) in canalolithiasis, and apogeotropic (beating toward the ceiling) in cupulolithiasis. The geotropic pattern involves stronger

nystagmus when the affected ear is down. Duration tends to be longer than posterior SCC nystagmus, and fatigability is variable. [9]

Absence of Neurological Signs

A critical feature distinguishing peripheral BPPV from central positional vertigo (central causes) is the absence of neurological signs. In BPPV, the head impulse test is normal (no corrective saccade), there is no skew deviation, and nystagmus is direction-fixed (not direction-changing) and conforms to the expected canal-specific pattern. The neurological examination — cranial nerves, cerebellar function, gait — should be normal. Any deviation from this pattern should raise the possibility of a central lesion and prompt neuroimaging. [12]

> □ **Clinical Insight:** Children may not report spinning sensation. They may instead describe feeling "wobbly", "dizzy when lying down", or simply exhibit reluctance to move their head. Take a careful positional trigger history from both the child and the parent — asking specifically whether symptoms occur when rolling over in bed, looking up, or getting in and out of bed.

V. Diagnostic Manoeuvres: Dix-Hallpike, Supine Roll, and Adaptations for Children

The diagnostic manoeuvres for BPPV — the Dix-Hallpike test for posterior SCC and the Supine Roll test for horizontal SCC — are the same in children as in adults, but their execution requires specific modifications to account for paediatric cooperation, body size, and anxiety. Establishing rapport and explaining the procedure clearly to both child and caregiver before beginning is essential. [3,11]

Dix-Hallpike Technique in Children

The Dix-Hallpike test is performed with the child seated on the examination table with the clinician standing to one side. The child's head is turned 45 degrees toward the ear being tested (the affected, or suspected affected, side). The child is then rapidly reclined to a position approximately 30 degrees below horizontal, with the head remaining at 45 degrees of rotation. For younger or smaller children, the clinician may need to support the head and neck more actively. The speed of recline is important — the manoeuvre should be performed rapidly to maximise the gravitational displacement of otoconia. The child should be instructed to keep their eyes open and look straight ahead during the test. [3,12]

In anxious children who cannot cooperate with the standard technique, a modified approach using a reclining chair or performing the test on a parent's lap (for very young children) may improve compliance. However, for children who cannot cooperate at all, diagnostic certainty may be limited, and a therapeutic trial of repositioning based on clinical suspicion is occasionally necessary.

Supine Roll Test for Horizontal SCC

The Supine Roll test is performed with the child supine with the head elevated approximately 20 degrees. The head is rapidly rotated 90 degrees to one side and the nystagmus is observed (direction, intensity, duration). The head is then returned to centre and rapidly rotated to the opposite side. The affected side is identified by the direction and relative intensity of the induced nystagmus: in the geotropic pattern (canalolithiasis), the nystagmus beating toward the floor is strongest when the affected ear is down. [9]

Interpreting Nystagmus Direction

Correct identification of the affected canal and side from nystagmus direction is the most critical diagnostic step. The clinician must observe both the beating direction (upbeat/torsional for posterior; horizontal for lateral) and the relationship to which ear is down (for lateralisation). In children, nystagmus may be less pronounced in amplitude than in adults and may require Frenzel goggles or video-Frenzel goggles to visualise reliably. [3,12]

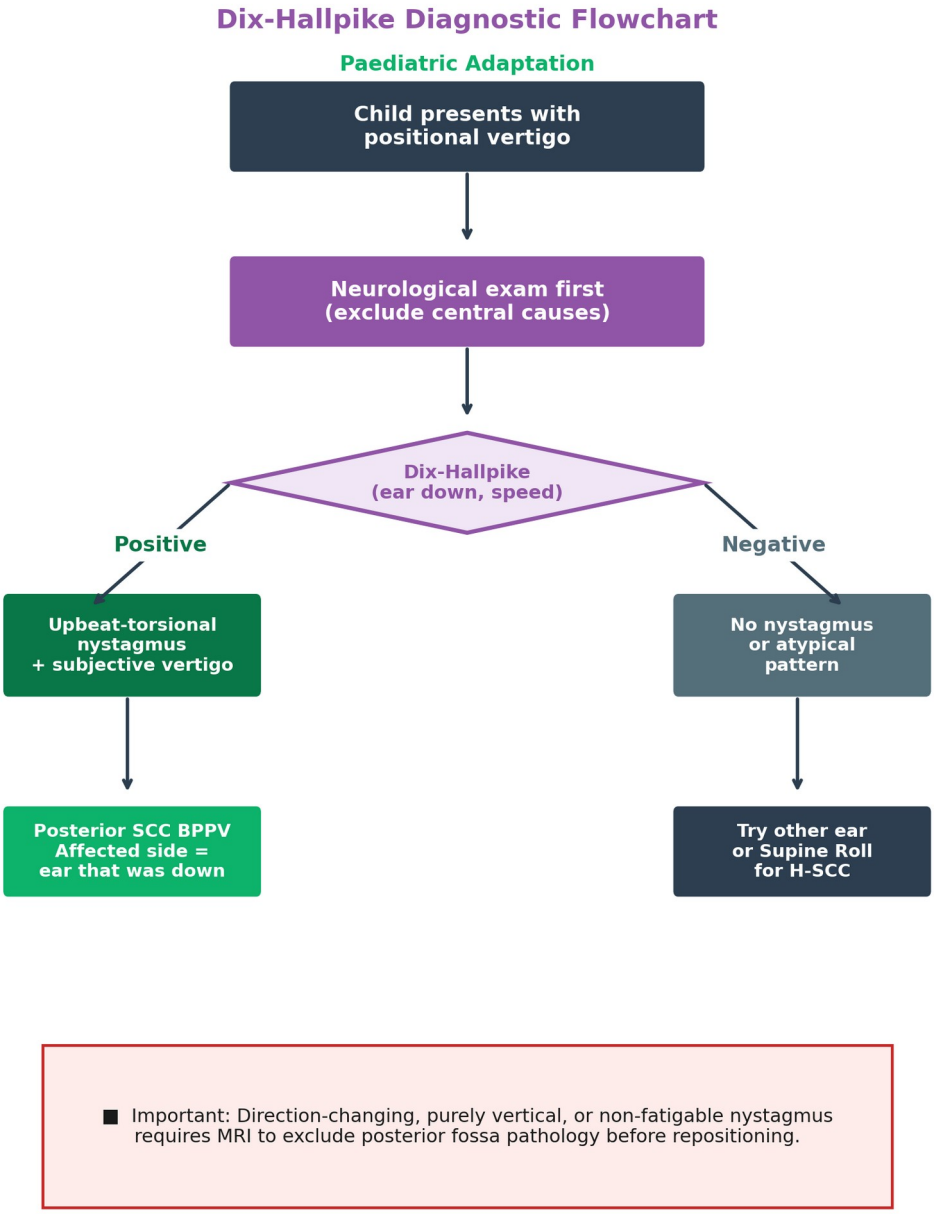

Figure 3. Dix-Hallpike Diagnostic Flowchart with Nystagmus Pattern Interpretation — paediatric adaptation including response to atypical nystagmus patterns.
Source: Australian Dizziness Clinics — clinical flowchart.

□ **Important:** Always perform a full neurological examination before repositioning manoeuvres. Positional nystagmus with direction-changing features, purely vertical nystagmus, skew deviation, absent fatigability, or neurological signs requires MRI of the posterior fossa before any repositioning is attempted.

VI. Canal-Specific Repositioning Manoeuvres

Canalith repositioning manoeuvres (CRMs) are the treatment of choice for BPPV across all age groups. The goal is to guide free-floating otoconia out of the affected SCC and into the utricle, where they are reabsorbed. Canal-specific manoeuvres have excellent efficacy in children, with success rates generally exceeding those in adults, reflecting the higher proportion of canalolithiasis (vs cupulolithiasis) in the paediatric population. [3,4]

Epley Manoeuvre — Posterior SCC BPPV

The Epley manoeuvre, described in 1992, remains the gold-standard treatment for posterior SCC canalolithiasis. The procedure involves four sequential head positions designed to rotate the otoconia 360 degrees around the posterior SCC and deposit them into the common crus and ultimately the utricle. In children, the manoeuvre is performed identically to the adult technique, with modifications as needed for size and cooperation. Key adaptations include performing the manoeuvre slowly, pausing at each position for 30–60 seconds (rather than 30 seconds as in the original description), and using reassuring verbal guidance throughout. [13]

Success rates with the Epley manoeuvre in paediatric BPPV are reported at 85–95% after 1–3 sessions, somewhat higher than the 80–85% reported in adult series. This may reflect the higher proportion of simple canalolithiasis (as opposed to cupulolithiasis) in children, which responds more predictably to repositioning. [3,4]

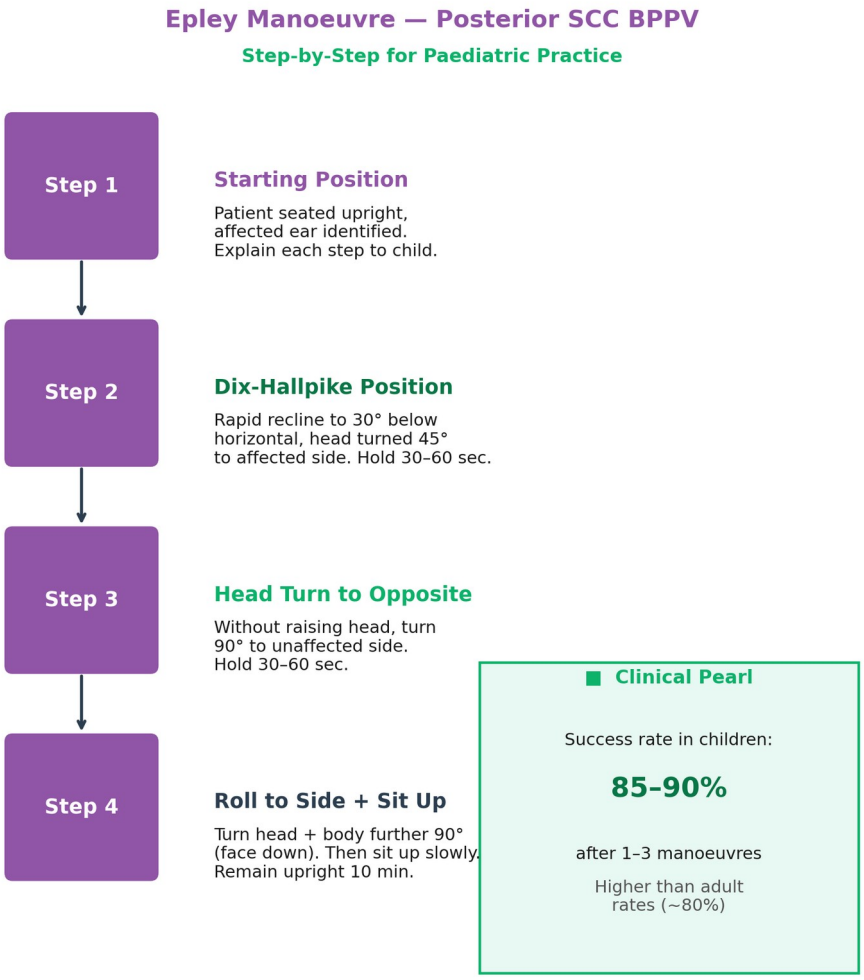

Figure 4. Epley Manoeuvre Step-by-Step for Posterior SCC BPPV — paediatric modifications including prolonged hold times and reassurance strategies.

Source: Australian Dizziness Clinics — clinical flowchart.

Semont Manoeuvre — Alternative for Posterior SCC

The Semont (or liberatory) manoeuvre is an alternative repositioning technique for posterior SCC BPPV that involves rapid lateral decubitus positioning and may be preferred for children who cannot tolerate the head-hang position required in the Epley. Evidence for the Semont manoeuvre in children is more limited, but single-session success rates of 70–80% have been reported. [14]

Gufoni and Barbecue Roll — Horizontal SCC BPPV

Horizontal SCC canalolithiasis (geotropic pattern) is treated with either the Gufoni manoeuvre or the Barbecue roll (360-degree rotation). The Gufoni manoeuvre involves a rapid lateral decubitus to the

affected side, followed by a rapid 45-degree nose-down head turn, held for 2 minutes, followed by a slow return to sitting. The Barbecue roll involves sequential 90-degree rotations from affected side through 270 degrees to return the patient to supine facing the unaffected side. [15]

For horizontal SCC cupulolithiasis (apogeotropic pattern), the Gufoni manoeuvre to the unaffected side or forced prolonged positioning on the unaffected side are the primary treatment options. Evidence in paediatric horizontal canal BPPV is primarily extrapolated from adult studies, with no large paediatric-specific RCTs for horizontal canal manoeuvres.

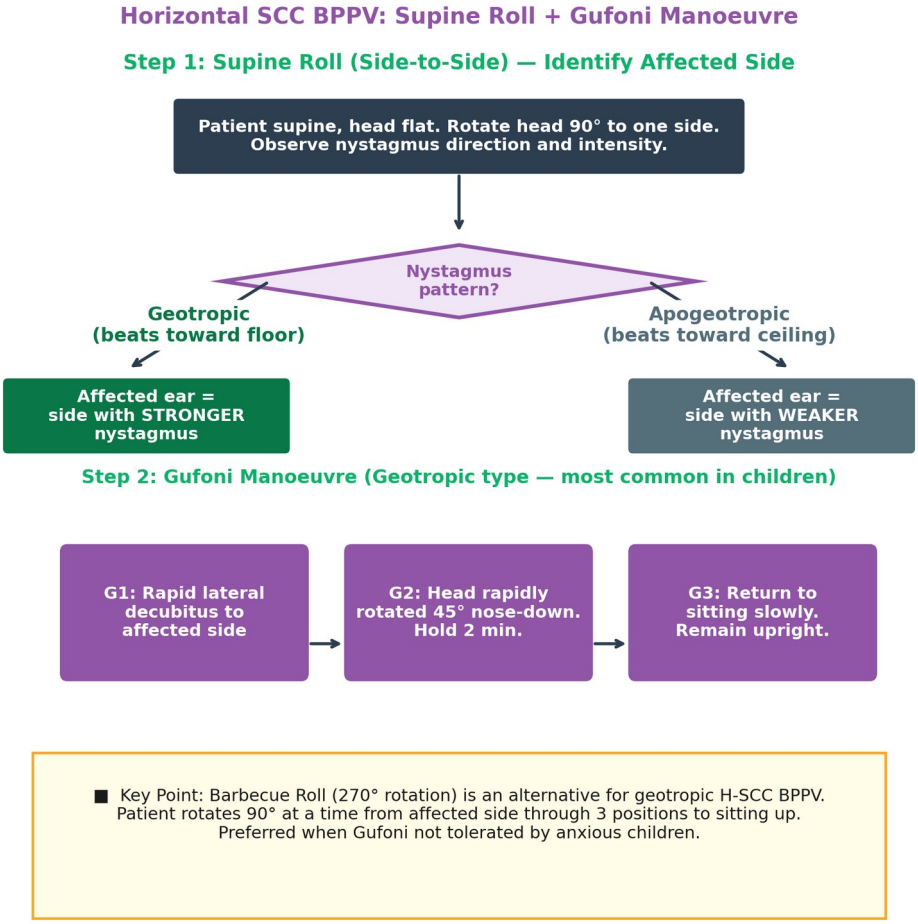

Figure 5. Horizontal SCC BPPV — Supine Roll Test and Gufoni Manoeuvre Steps — including identification of affected side and step-by-step repositioning.
Source: Australian Dizziness Clinics — clinical flowchart.

Post-Manoeuvre Instructions

Post-manoeuvre activity restriction (avoidance of lying flat for 48 hours, sleeping semi-recumbent) was historically recommended but is not supported by evidence from RCTs. Current evidence suggests that post-manoeuvre activity restrictions do not improve outcomes and may unnecessarily restrict normal paediatric activity. Children should be advised that mild unsteadiness for 24–48 hours post-manoeuvre is common and does not indicate treatment failure. Return to full activity, including school and sport, can be advised as tolerated.

VII. Special Situations: Bilateral BPPV, Horizontal Canal BPPV, Recurrent BPPV

Bilateral BPPV

Bilateral BPPV — defined as BPPV affecting SCCs on both sides — is rare in children but is significantly more common in secondary paediatric BPPV than in idiopathic cases. The most important secondary causes of bilateral paediatric BPPV include bilateral head trauma (e.g., road traffic accident), aminoglycoside ototoxicity (which causes bilateral hair cell loss and otoconia fragmentation), and bilateral EVA. Bilateral BPPV is treated with canal-specific manoeuvres for each affected side sequentially. [4,6]

> □ **Important:** Bilateral BPPV in a child should prompt investigation for aminoglycoside exposure (neonatal NICU admission), bilateral EVA, or prior bilateral head trauma. It is not expected to be idiopathic in childhood.

Recurrent BPPV

Recurrent BPPV — defined as two or more episodes within a 12-month period — warrants investigation for an underlying secondary cause, even if the initial episode was labelled idiopathic. Contributing factors include structural vulnerability of the otolithic membranes (EVA), chronic middle ear disease, ongoing exposure to vestibulotoxic agents, or a predisposing calcium metabolism disorder (hypovitaminosis D, osteoporosis — rare in children). Vestibular rehabilitation with balance training may reduce recurrence frequency by improving central compensation. [7]

Surgical Management

Surgical intervention for BPPV — specifically posterior SCC occlusion — is reserved for the extremely rare child with disabling, medication-refractory BPPV who has failed multiple CRMs. Surgical occlusion blocks the posterior SCC lumen, preventing otoconia movement. The procedure carries a small risk of permanent SNHL and is undertaken only after extensive conservative management has failed. This indication is exceptionally uncommon in the paediatric population. [16]

VIII. Secondary BPPV: Causes and Investigation

Secondary BPPV — in which an identifiable precipitant accounts for otoconia detachment — is proportionally much more common in children than in adults. Identifying the secondary cause is important for prognosis, recurrence prevention, and detection of serious underlying pathology. [2,6]

Head Trauma

Head trauma is the most common cause of secondary BPPV in children, accounting for 40–60% of secondary paediatric BPPV in referral series. Trauma-related BPPV may occur immediately after the injury or be delayed by days to weeks. The mechanism involves mechanical shearing of otoconia from the utricular macula. The posterior and horizontal SCCs may both be affected. Traumatic BPPV may be bilateral. Management with CRMs is effective, but recurrence rates are higher than in idiopathic cases. [2,17]

Enlarged Vestibular Aqueduct (EVA)

EVA/EVAS is the most common genetic cause of paediatric sensorineural hearing loss and is strongly associated with recurrent BPPV in children. Mutations in SLC26A4 (autosomal recessive) cause abnormal endolymph ion transport and mechanical instability of the otolithic membranes. Children with EVA may present with episodic vertigo, fluctuating hearing loss, and recurrent BPPV, often triggered by minor head trauma or pressure changes. Audiological monitoring and avoidance of head trauma and contact sports are recommended. [6,10]

Other Secondary Causes

Additional secondary causes in children include: post-viral labyrinthitis (HSV-1 reactivation, CMV, influenza) causing otoconia fragmentation; prolonged bed rest or NICU admission (gravity-independent otoconia settlement); ototoxic drug exposure (aminoglycosides, cisplatin); and rarely, Ménière's disease in adolescents (endolymphatic hydrops with otoconia instability). [2,7]

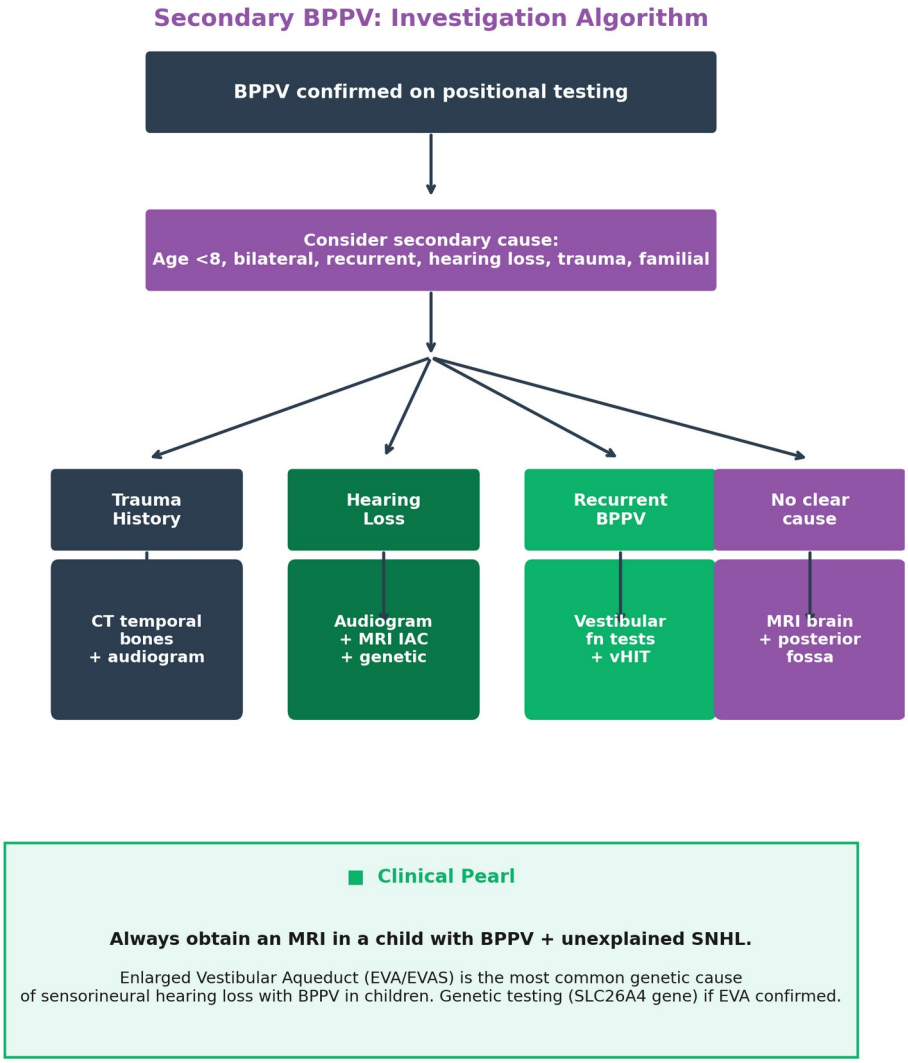

Figure 6. Secondary BPPV Investigation Algorithm — structured workup by presenting feature including trauma history, hearing loss, recurrent episodes, and unexplained cases.
Source: Australian Dizziness Clinics — clinical flowchart.

□ **Clinical Pearl:** Always obtain an audiogram and MRI in a child with BPPV and unexplained SNHL. Enlarged Vestibular Aqueduct (EVA) is the most common genetic cause and is identified on CT/MRI temporal bones. Genetic testing for SLC26A4 mutations should be considered if EVA is confirmed on imaging.

The investigation workup for secondary BPPV in children should be guided by clinical context. A full audiogram is mandatory in all paediatric BPPV cases — particularly when hearing loss, tinnitus, or fluctuating auditory symptoms are present. Vestibular function testing including video head impulse testing (vHIT) and vestibular evoked myogenic potentials (VEMPs) can characterise the degree of canal and otolith dysfunction. MRI of the internal auditory canals and posterior fossa with gadolinium is indicated when: an underlying cause is suspected; BPPV is bilateral; or the child is under 8 years. CT temporal bones assists in identifying EVA (cotillon sign) and structural anomalies.

IX. Outcomes, Recurrence, and Follow-Up

The overall prognosis for children with BPPV is excellent. Resolution rates with CRMs are high — approximately 85–95% achieve symptom resolution after 1–3 manoeuvres. Compared to adults, paediatric BPPV tends to resolve more quickly and with fewer treatment sessions, likely reflecting the

higher prevalence of pure canalolithiasis (vs cupulolithiasis), younger patient age, and superior vestibular compensation capacity. [3,4]

Recurrence and Monitoring

Recurrence rates in children (10–20% at 1 year) are lower than in adults (20–30%), but cases associated with secondary causes — particularly EVA, recurrent trauma, or bilateral involvement — carry higher recurrence risk. Children with recurrent BPPV should be assessed for modifiable risk factors (recurrent head trauma, medication exposure) and considered for structured vestibular rehabilitation to improve central compensation. Audiological review is recommended for any child with recurrent BPPV, given the association with EVA and progressive SNHL. [4,7]

Residual Symptoms and Rehabilitation

A subset of children will experience residual postural instability or non-specific dizziness following successful CRM — a finding analogous to "residual dizziness" in adults. This is thought to reflect incomplete central compensation after sustained canal dysfunction. Targeted vestibular rehabilitation exercises (gaze stabilisation, balance retraining) are effective in children from approximately 7–8 years of age and should be offered when residual symptoms persist beyond 4–6 weeks. Return to school and sport should be guided by symptom control and balance performance rather than a fixed time interval.

> □ **Key Point:** Children generally have faster and more complete BPPV resolution than adults — 85–95% after 1–3 repositioning manoeuvres. Residual dizziness persisting beyond 4–6 weeks should prompt vestibular rehabilitation referral and reassessment for inadequate repositioning, an additional diagnosis, or an undetected secondary cause.

X. Summary and Key Clinical Takeaways

BPPV in children is a clinically important and treatable cause of paediatric vertigo that differs from adult BPPV in its epidemiology, aetiology, and the higher proportion of secondary causes. The following ten points encapsulate the core clinical principles of this literature review:

1. BPPV is rare under 5 years. A child under 8 years presenting with apparent BPPV is likely to have a secondary cause — investigate accordingly.
2. The posterior SCC is affected in 85% of paediatric BPPV cases, producing upbeat-torsional nystagmus on Dix-Hallpike positioning.
3. Children may not describe spinning — they report being "wobbly" or vomit with head movement. A positional trigger history from both parent and child is essential.
4. Neurological examination must precede repositioning. Direction-changing, purely vertical, or non-fatigable nystagmus requires MRI.
5. The Dix-Hallpike and Supine Roll tests are the diagnostic gold standard and can be adapted for anxious or younger children.
6. The Epley manoeuvre achieves 85–95% resolution in paediatric posterior SCC BPPV after 1–3 sessions.
7. Horizontal SCC BPPV (10–12% of cases) requires the Gufoni or Barbecue Roll manoeuvre. Identify geotropic vs apogeotropic pattern before treating.
8. Bilateral BPPV or BPPV under age 8 should prompt structured investigation including audiogram, MRI, and genetic testing if EVA is suspected.
9. EVA is the most common genetic cause of recurrent BPPV with hearing loss in children — audiogram and MRI are mandatory when SNHL accompanies BPPV.
10. Prognosis is excellent — recurrence rates are lower than in adults. Residual dizziness beyond 4–6 weeks warrants vestibular rehabilitation.

This review is the fourth in the Vestibular Medicine in Children series. The next review (PVM05) covers Vestibular Neuritis and Labyrinthitis in Children, including the acute vestibular syndrome, HINTS examination in the paediatric setting, and vestibular rehabilitation following acute peripheral vestibulopathy.

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