Benign Paroxysmal Positional Vertigo (BPPV):

Diagnosis and Treatment of the Most Treatable Vertigo at the Bedside

Vestibular Medicine for General Clinicians

Topic 4 of 14

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How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with dizziness.

The review is designed to be read in a single 20–30 minute sitting, or used as a desktop reference. It is supported by an A4 one-page 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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I. Why BPPV Matters in General Practice

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo across all age groups, with a lifetime prevalence of approximately 2.4% and a cumulative incidence rising sharply after the age of 50 [1,2]. It accounts for 20–30% of all dizziness presentations in primary care, and for approximately half of all vertigo diagnosed in otolaryngology clinics [3]. It is the single most treatable vertigo condition in medicine — a correctly performed repositioning manoeuvre resolves symptoms in 80–90% of patients at the first treatment [4,5].

Despite this, BPPV is under-diagnosed and under-treated in general practice. Audits in Australian and international primary care settings consistently show that the Dix-Hallpike is performed in fewer than 25% of eligible patients, and that formal repositioning manoeuvres are offered to fewer than 10% [6,7]. Patients are often prescribed vestibular suppressants (prochlorperazine, betahistine) that provide no durable benefit, while the definitive treatment is available at the bedside in five minutes.

□ **Key Point:** BPPV is the only vestibular condition where a correctly performed bedside manoeuvre — taking 3 to 5 minutes and requiring no equipment — cures 80–90% of patients at a single visit. General practitioners should be confidently able to diagnose and treat posterior canal BPPV.

The consequences of missed BPPV are not trivial. Untreated BPPV in older adults is associated with a substantially increased risk of falls, fall-related fracture, depression, and loss of independence [8]. Effective treatment at the primary care level has measurable benefit at the population health level.

□ **Clinical Insight:** If a GP learns only one vestibular manoeuvre in their career, it should be the Epley. Its diagnostic cousin, the Dix-Hallpike, is equally simple, and together they identify and cure the commonest cause of vertigo in a single consultation.

II. Pathophysiology — Canalithiasis and Cupulolithiasis

BPPV is caused by displacement of otoconia — calcium carbonate crystals — from the utricular macula into one of the semicircular canals. When the patient moves into a position that places the affected canal in a gravity-dependent plane, the otoconial debris moves within the canal and generates an aberrant endolymph flow. This is interpreted by the central vestibular system as head rotation, producing vertigo and a characteristic nystagmus.

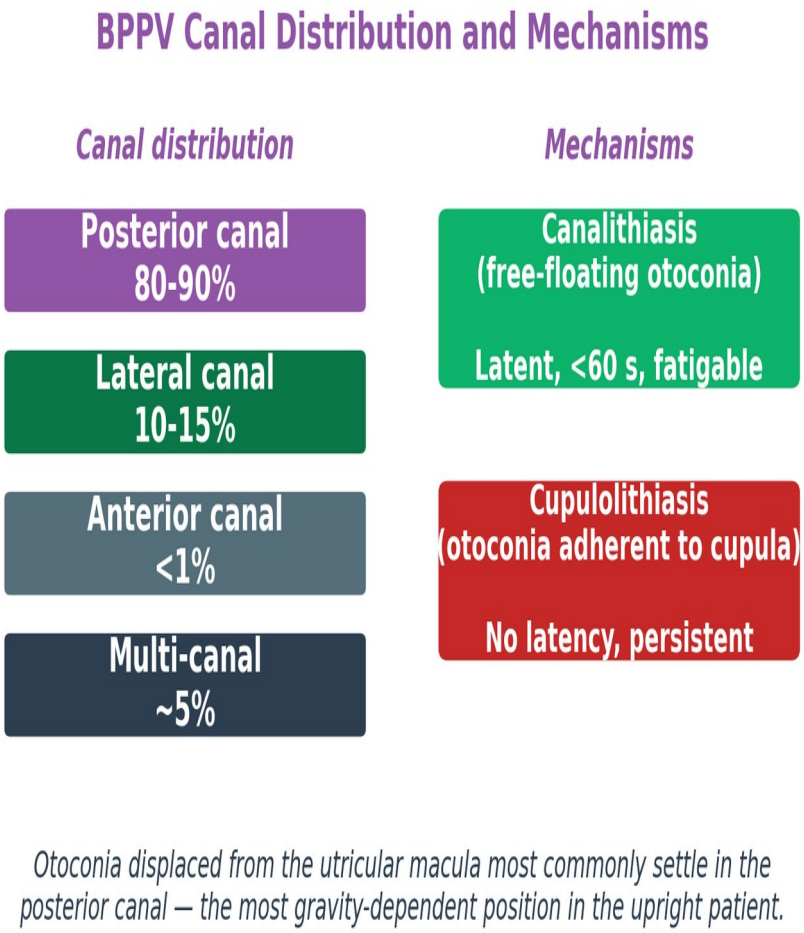

Figure 1. BPPV canal distribution and mechanisms — otoconia displaced from the utricular macula most commonly settle in the gravity-dependent posterior canal.
Source: Australian Dizziness Clinics — clinical flowchart.

Canalithiasis (Free-Floating)

Canalithiasis is the more common form. Otoconia float freely in the canal lumen and settle when gravity permits. Nystagmus has a characteristic latency (1–5 seconds), builds to a crescendo, and resolves within 60 seconds as the debris reaches the lowest point of the canal. It is fatigable on repeat testing. Canalithiasis is highly responsive to repositioning manoeuvres.

Cupulolithiasis (Adherent)

Cupulolithiasis is less common. Otoconia adhere to the cupula itself, rendering it gravity-sensitive. Nystagmus begins without latency, is persistent (>60 seconds), and does not fatigue. Cupulolithiasis is less responsive to standard repositioning and may require specific manoeuvres (Gufoni, forced prolonged position) [9].

Canal Distribution

- Posterior semicircular canal: 80–90% of all BPPV. Most gravity-dependent canal in the upright position; otoconia readily settle into it.
- Lateral (horizontal) canal: 10–15%. Often secondary to treatment of posterior canal BPPV (canal conversion).
- Anterior semicircular canal: <1%. Rare; a downbeat-predominant nystagmus on positional testing should raise suspicion for a central mimic before diagnosing anterior canal BPPV [10].

- Multicanal: occurs in approximately 5% of patients, typically post-trauma or bilateral disease.

Aetiology and Risk Factors

Most BPPV is idiopathic. Identifiable secondary causes include head trauma, vestibular neuritis or labyrinthitis (otoconial disruption), prolonged recumbency (post-operative, post-hospitalisation), Ménière's disease, migraine, vitamin D deficiency, and osteoporosis [11,12]. Recurrence rates are 15–30% at one year and 40–50% at five years, higher in secondary BPPV [13].

□ **Clinical Pearl:** Vitamin D deficiency is associated with both incidence and recurrence of BPPV. In Australian patients with recurrent BPPV — particularly older adults with osteoporosis — measuring and replacing vitamin D is a reasonable adjunct to repositioning therapy [12].

III. Clinical Presentation — The Classic History

The history of BPPV is often so characteristic that an experienced clinician can make a provisional diagnosis before the patient finishes speaking. The key features:

- Episode duration: seconds — typically 10 to 30 seconds, rarely more than 60 seconds per attack.
- Clear positional trigger: rolling over in bed (especially to one side), lying down, getting up from lying, looking up ("top shelf"), bending forward.
- Complete recovery between attacks: when the head is still and upright, patient is asymptomatic. A residual unsteadiness or "foggy" sensation between attacks is common, particularly in older adults.
- Absence of hearing symptoms: tinnitus, hearing loss, or aural fullness are not features of BPPV. If present, reconsider the diagnosis.
- Absence of neurological symptoms: no diplopia, dysarthria, weakness, or severe headache.

The Diagnostic "Pseudo-Constant" Pattern

Patients often describe BPPV as "constant dizziness" because every time they move their head the symptom re-triggers. The clarifying question — "between the individual attacks, when you hold your head still, do you feel completely normal?" — is essential. True BPPV produces discrete attacks of seconds interspersed with asymptomatic periods, not continuous vertigo.

□ **Key Point:** Episode duration of seconds plus a clear positional trigger equals BPPV until proven otherwise. Confirm with Dix-Hallpike at the same consultation.

Features That Should Prompt Reconsideration

- Episodes lasting minutes or longer — consider vestibular migraine, Ménière's, TIA.
- Associated neurological or severe headache symptoms — consider central positional vertigo.
- Progressive unilateral hearing loss — consider vestibular schwannoma.
- No resolution between attacks — consider AVS (vestibular neuritis, stroke).
- First episode in a patient with vascular risk factors — perform and document a focused neurological examination.

□ **Clinical Insight:** BPPV is the commonest cause of vertigo, but central positional vertigo (cerebellar or fourth-ventricular lesions) is the most important mimic. Downbeat-predominant nystagmus, lack of latency, lack of fatigability, and persistence beyond 60 seconds should shift suspicion to central cause and prompt imaging [14].

IV. Diagnostic Manoeuvres — Dix-Hallpike

The Dix-Hallpike manoeuvre is the diagnostic test of choice for posterior canal BPPV. Correctly performed and interpreted, it has a sensitivity of approximately 79% and specificity of 75% for posterior canal BPPV, with higher performance when combined with clinical history [4].

Technique

- Position the patient seated on the couch, legs extended, so that when lying supine the head will extend 20 degrees below horizontal.
- Explain the test, warn the patient that vertigo may be provoked, and provide an emesis bag.
- Rotate the head 45 degrees toward the side being tested.
- Supporting the head firmly, lie the patient rapidly supine with head extended 20 degrees below horizontal and still rotated 45 degrees.
- Observe the eyes for at least 30 seconds — ideally 60 — in this position.
- Return the patient slowly to sitting, observing for reversal nystagmus.
- Allow a brief rest, then repeat on the opposite side.

Expected Positive Findings — Posterior Canal BPPV

- Latency: 1–5 seconds before nystagmus begins.
- Direction: upbeat (fast phase toward forehead) with torsional component (top poles of eyes beat toward the downward / affected ear).
- Duration: less than 60 seconds.
- Fatigability: decreases on repeat testing.
- Reversal: nystagmus reverses direction on returning the patient to sitting.
- Symptom concordance: reproduces the patient's presenting vertigo.

Table 1 — Nystagmus Interpretation on Dix-Hallpike

Nystagmus pattern	Diagnosis
Upbeat-torsional, latent, <60 s, fatigable	Posterior canal BPPV (side tested)
Horizontal nystagmus on Dix-Hallpike	Consider lateral canal BPPV — proceed to supine roll test
Pure downbeat, no latency, persistent	Central positional nystagmus — consider cerebellar lesion, imaging required
Purely torsional, persistent	Rare — anterior canal BPPV or central mimic
No nystagmus despite reproduction of vertigo	Equivocal — retest; if still negative, consider alternative cause
Nystagmus without vertigo	May be "silent" residual BPPV or central pattern — interpret cautiously

Features raising concern for central cause: no latency, persistent >60 seconds, pure vertical / downbeat, lack of fatigability, purely torsional without canal pattern.

□ **Clinical Pearl:** The commonest cause of a false-negative Dix-Hallpike is premature termination. The nystagmus may take 5–10 seconds to emerge. Hold the position for at least 30 and ideally 60 seconds, with the eyes continuously observed.

Modifications for Patients with Limited Neck Mobility

In older adults or those with cervical spine pathology, the side-lying test (Semont diagnostic position) is a reasonable alternative. The patient sits on the couch, head turned 45 degrees to the unaffected side, then is brought into side-lying on the affected side. Provocation of the same nystagmus pattern confirms the diagnosis [15].

V. Diagnostic Manoeuvres — Supine Roll Test for Lateral Canal

Lateral (horizontal) canal BPPV is the second-commonest variant. It is frequently missed because the Dix-Hallpike is insensitive for it — lateral canal otoconia do not move meaningfully during the Dix-Hallpike position. Any patient with typical BPPV history and a negative Dix-Hallpike should proceed to a supine roll test [16].

Technique

- Lie the patient supine with head elevated approximately 20 degrees (pillow under shoulders).
- Rapidly turn the head 90 degrees to one side; observe for nystagmus for at least 30 seconds.
- Return the head to neutral; allow at least 30 seconds for symptoms to settle.
- Rapidly turn the head 90 degrees to the opposite side; observe for at least 30 seconds.
- Compare the intensity and direction of nystagmus between the two sides.

Interpretation

- Geotropic nystagmus (beats toward the ground-ward ear on each side, with greater intensity on one side): canalithiasis of the lateral canal. The more intense side is the affected side.
- Apogeotropic nystagmus (beats away from the ground-ward ear on each side): cupulolithiasis of the lateral canal, or canalithiasis in the anterior arm of the canal. The less intense side is the affected side.
- Persistent direction-changing horizontal nystagmus without clear positional dependence: central cause must be considered.

Table 2 — Lateral Canal BPPV — Sub-type and Affected Side

Nystagmus pattern	Sub-type	Affected side
Geotropic, both sides	Canalithiasis (long arm)	Side with stronger nystagmus
Apogeotropic, both sides	Cupulolithiasis OR canalithiasis (anterior arm)	Side with weaker nystagmus
Unilateral nystagmus only	Variable	Requires repeat testing and clinical correlation

Ewald's second law guides side determination in lateral canal BPPV [16].

□ **Key Point:** If positional vertigo persists after apparently adequate Epley treatment, perform a supine roll test. Untreated or mis-directed lateral canal BPPV is one of the commonest causes of "failed" BPPV treatment in primary care.

VI. Treatment — The Epley Manoeuvre

The Epley (canalith repositioning) manoeuvre is the treatment of choice for posterior canal BPPV. Systematic reviews and randomised trials report single-treatment success rates of 80–90%, with cumulative success exceeding 95% after two or three treatments [4,5]. It is endorsed as first-line treatment by the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) clinical practice guideline [4].

Figure 2. The Epley (canalith repositioning) manoeuvre for right posterior canal BPPV — five sequential head positions; mirror for left-sided disease.

Source: Australian Dizziness Clinics — clinical flowchart.

Technique (Right Posterior Canal Example)

- Position 1 — Sit upright with head rotated 45 degrees to the right (toward the affected side).
- Position 2 — Rapidly lie supine, head still rotated 45 degrees right and extended 20 degrees below horizontal (Dix-Hallpike position). Hold for 30–60 seconds, or until nystagmus resolves, plus a further 30 seconds.
- Position 3 — Rotate head 90 degrees to the left, so head is now rotated 45 degrees to the left of midline. Hold for 30–60 seconds.
- Position 4 — Ask the patient to roll onto the left shoulder and turn the head further so the face is 45 degrees below horizontal. Hold for 30–60 seconds.
- Position 5 — Sit the patient up with the head still rotated to the left, then bring the head to neutral.

Mirror the technique for left-sided BPPV. The AAO-HNS guideline specifies that post-treatment activity restriction (sleeping upright, avoiding the affected side) is not required and does not improve outcomes [4].

Expected Outcomes

- Single treatment: 80–90% symptom resolution [4,5].

- Second treatment at 1–2 week review: cumulative 95%+.
- Residual unsteadiness without vertigo post-treatment: common, resolves with mobilisation and balance exercises over days to weeks.
- Recurrence at 1 year: approximately 15–30% [13].

□ **Clinical Insight:** A single, correctly performed Epley manoeuvre resolves 80–90% of posterior canal BPPV. This is one of the most effective therapeutic interventions in medicine for a common condition. It is free, takes less than five minutes, and is deliverable in any clinical setting without equipment.

Alternative Manoeuvres for Posterior Canal BPPV

- Semont (liberatory) manoeuvre: non-inferior to Epley in randomised trials; preferred in patients with significant cervical pathology [17].
- Brandt-Daroff exercises: inferior to Epley for acute treatment; reasonable as a home-based adjunct or for patients who decline in-clinic manoeuvre [18].

Treatment of Lateral Canal BPPV

- Gufoni manoeuvre: first-line for both geotropic and apogeotropic lateral canal BPPV; 70–80% single-treatment success [19].
- Barbeque (Lempert 360°) roll: traditional treatment for geotropic lateral canal BPPV; comparable success rates to Gufoni [19].
- Forced prolonged position: overnight lying on the unaffected side; used when in-clinic manoeuvres fail.

□ **Clinical Pearl:** Vestibular suppressants (prochlorperazine, betahistine, diazepam) are not effective treatments for BPPV and are actively unhelpful. They suppress symptoms without treating the cause, delay central compensation, and — in older adults — increase fall risk. Avoid them in confirmed BPPV [4].

VII. Clinical Algorithm — Diagnosis and Treatment at the Bedside

The algorithm below outlines the end-to-end bedside pathway for suspected BPPV, integrating history, diagnostic manoeuvre, treatment, and follow-up.

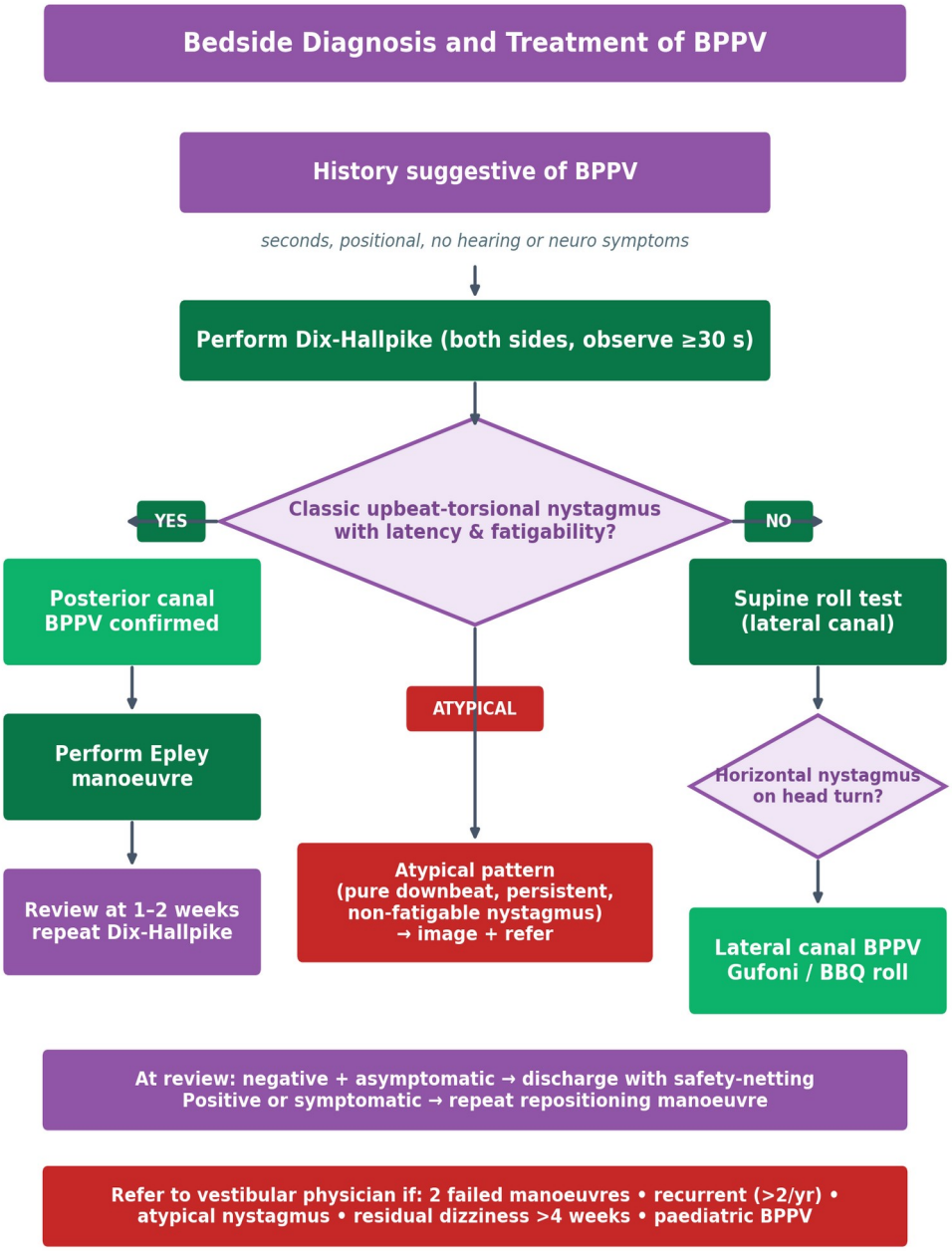

Figure 3. BPPV bedside diagnosis and treatment algorithm — a complete encounter (history, manoeuvre, treatment, follow-up) is deliverable in 10–15 minutes in a standard GP consultation.

Source: Australian Dizziness Clinics — clinical flowchart.

□ **Key Point:** Diagnosis and treatment of BPPV is a single consultation event. The clinician who can deliver this end-to-end pathway offers substantial value, particularly to older patients where untreated BPPV increases fall risk.

VIII. Follow-Up, Recurrence, and Persistent Symptoms

Review at 1–2 weeks after treatment, with repeat Dix-Hallpike (and supine roll if lateral canal suspected), is the standard of care [4]. The aim of review is to confirm resolution objectively, not only symptomatically.

Residual Symptoms After Successful Repositioning

A substantial minority of patients report residual unsteadiness or "fogginess" for days to weeks after successful Epley, despite negative Dix-Hallpike. This is not treatment failure. Proposed mechanisms include partial utricular dysfunction from otoconial disruption, mild central vestibular de-compensation, and co-existing anxiety or hypervigilance. Management is reassurance, resumption of normal activity, and vestibular rehabilitation if symptoms persist beyond 2 weeks [20].

Recurrence

Recurrence rates are approximately 15% at 1 year and 40–50% at 5 years [13]. Risk factors for recurrence include older age, osteoporosis and vitamin D deficiency, Ménière's disease, migraine, and secondary (traumatic) BPPV. Patients should be advised to return early at any recurrence — repeat Epley at first recurrence is appropriate primary care.

True Treatment Failure — When to Reconsider

- Consistently negative Dix-Hallpike despite typical history: consider lateral canal BPPV (supine roll test), vestibular migraine (episode duration longer than seconds), or a central mimic.
- Recurrent positive Dix-Hallpike with the same canal despite two correctly performed Epley manoeuvres: consider cupulolithiasis, multi-canal involvement, or a secondary cause (intercurrent vestibular neuritis, migraine).
- Nystagmus with atypical features (downbeat, persistent, non-fatigable): treat as central until imaging excludes.
- Frequent recurrence (>2 episodes / year): consider secondary causes (migraine, Ménière's, osteoporosis, vitamin D deficiency) and referral to a vestibular physician.

□ **Important:** A patient labelled as "chronic BPPV" who has failed multiple treatments deserves reconsideration. The commonest explanations are (a) unrecognised lateral canal BPPV, (b) technically inadequate Epley manoeuvre, and (c) a misdiagnosis — most often vestibular migraine or PPPD. Referral for dedicated assessment is appropriate.

□ **Clinical Insight:** BPPV is the commonest cause of chronic positional vertigo but chronic positional symptoms without objective BPPV findings are rarely BPPV. Vestibular migraine and PPPD can mimic positional symptoms and need different treatment pathways.

IX. Special Populations and Contraindications

BPPV occurs across all age groups but has particular features in specific populations that alter diagnosis and management.

Older Adults

BPPV incidence rises sharply after 50 years and peaks in the seventh decade [2]. In older adults, presentation is often atypical — patients may describe "imbalance" or "falls" rather than vertigo, and episodes may be less dramatic. Untreated BPPV in this group is associated with a threefold increased fall risk and with functional decline [8]. Screening dizzy older patients with Dix-Hallpike is an under-utilised falls prevention intervention.

Patients with Cervical Spine Pathology

Severe cervical spondylosis, rheumatoid atlanto-axial instability, or vertebral artery disease are relative contraindications to Dix-Hallpike and Epley in the fully extended position. The side-lying (Semont) test and liberatory manoeuvre achieve the same canal orientations without neck extension and are appropriate alternatives [15,17].

Paediatric Patients

BPPV in children is uncommon but under-recognised; it is typically secondary to head trauma or migraine. Standard diagnostic and therapeutic manoeuvres, appropriately scaled, are effective. Suspect central positional vertigo (posterior fossa tumour) in any child with persistent positional symptoms and perform imaging with a low threshold [21].

Pregnancy

BPPV is increasingly recognised in pregnancy, possibly related to hormonal effects on otoconial metabolism. Diagnostic and therapeutic manoeuvres are safe in all trimesters. Avoidance of anti-emetics during repositioning is usually feasible given the brief duration.

Post-Traumatic BPPV

Head trauma (including mild concussion) is a recognised precipitant. Post-traumatic BPPV has higher rates of bilateral and multi-canal disease and a correspondingly higher rate of treatment failure at first manoeuvre. More manoeuvres and longer follow-up are often required [11].

Contraindications and Precautions

- Unstable cervical spine pathology — use modified manoeuvres.
- Severe carotid or vertebrobasilar stenosis — seek specialist input.
- Recent retinal detachment or ophthalmic surgery — consult treating ophthalmologist.
- Acute vestibular neuritis with co-existing BPPV — treat the BPPV, but expect residual symptoms from the underlying neuritis.

□ **Clinical Pearl:** Age is not a contraindication to Epley — age is an indication. Older adults have the highest BPPV prevalence, the greatest fall-related morbidity from untreated BPPV, and the most to gain from a single successful repositioning manoeuvre.

X. When to Refer and Key Messages

Most BPPV can and should be diagnosed and treated in general practice. Referral to a vestibular physician or dedicated vestibular service is indicated in a minority of cases.

Indications for Referral

- Diagnostic uncertainty — atypical nystagmus, red-flag features, equivocal Dix-Hallpike in a patient who clinically "should" have BPPV.
- Failure of two correctly performed Epley (or Gufoni) manoeuvres on separate visits.
- Suspected lateral canal or multicanal BPPV where the clinician is not confident in identifying affected side or selecting manoeuvre.
- Frequent recurrence (>2 episodes per year) suggesting secondary cause.
- Post-traumatic BPPV with incomplete resolution after two treatments.
- Any suspicion of central positional vertigo — imaging plus vestibular physician review.
- Persistent residual unsteadiness beyond 4 weeks despite negative Dix-Hallpike — consider PPPD.
- Paediatric BPPV — lower threshold to refer and image.

Key Clinical Messages

- BPPV is the commonest cause of vertigo and the most treatable — in many healthcare systems, it is the single most cost-effective vertigo intervention.
- The diagnostic test — Dix-Hallpike — and the treatment — Epley — are bedside manoeuvres taking a combined 5–8 minutes.
- 80–90% of posterior canal BPPV resolves with a single correctly performed Epley manoeuvre.
- The Dix-Hallpike is insensitive for lateral canal BPPV; a supine roll test should follow a negative Dix-Hallpike in a patient with typical history.
- Vestibular suppressants are not effective treatments for BPPV and may do harm in older adults.
- Red-flag nystagmus patterns (downbeat, persistent, non-fatigable, pure vertical) should prompt imaging, not another Epley.
- Recurrence is common but individual episodes respond as well to treatment as the first.
- Untreated BPPV in older adults is a modifiable fall risk factor.

□ **Key Point:** The general clinician who can reliably diagnose and treat posterior canal BPPV at the first consultation delivers one of the highest-yield interventions in outpatient medicine. It is a

core competency for GPs and hospital generalists.

□ **Clinical Insight:** A patient who walks into a consultation with positional vertigo and walks out without it — after a five-minute bedside manoeuvre, no prescription, no imaging, no referral — is the signature success story of bedside vestibular medicine. It is entirely achievable in routine primary care.

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