Benign Paroxysmal Positional Vertigo (BPPV):

A Vestibular Physician's Deep Review of Diagnosis, Mechanism, and Management

Vestibular Medicine for Vestibular Physicians

Peripheral Vestibular Pathology — Module 2.4

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How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of peripheral vestibular practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module.

Callout Box Guide

□ **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

Benign paroxysmal positional vertigo (BPPV) is the commonest peripheral vestibular disorder and the single most frequent cause of recurrent vertigo in adults [1,2]. It is mechanically driven, diagnostically efficient, and treatable in a single encounter — yet remains under-diagnosed, mis-treated, or wrongly attributed to vestibular suppressants in everyday practice [1,11].

Lifetime prevalence is approximately 2.4% in the adult population, with annual incidence near 0.6% (6 per 1,000) [6,7]. The female-to-male ratio is roughly 2:1 [6]. Prevalence rises sharply with age — one-year prevalence is up to seven-fold higher in patients over 60 compared with those under 40, with peak onset between the fifth and seventh decades [6,8,36]. BPPV is uncommon in children. Among older adults, active screening uncovers undiagnosed BPPV in 3–9%, with significant contribution to falls, hip fracture and depression [8].

Table 1. Epidemiology of adult BPPV at a glance.

Measure	Adult value	Notes
Lifetime prevalence	~2.4%	F 3.2% / M 1.6%; ≈2:1 [6]
1-year prevalence	~1.6%	~7× higher in age >60 vs <40 [8]
Annual incidence	~0.6% (6 / 1,000)	Rare before middle age [6]
Clinic share	20–30% of dizziness referrals	≈⅓ of all peripheral vertigo [10,11]

BPPV accounts for 20–30% of new dizziness-clinic referrals and roughly one-third of all peripheral vertigo diagnoses [10,11]. Despite its self-limited reputation, more than 85% of patients report interruption of work or daily activities during active episodes, and older patients have measurably higher rates of falls and mood disturbance until treated [8,46].

Functional impact is disproportionate to the brevity of each attack. Patients describe disabling anticipatory anxiety around triggering positions — turning over in bed, hair-washing, dental appointments — and frequently restrict daily activity for weeks before presenting. Disease-specific instruments such as the Dizziness Handicap Inventory consistently show moderate-to-severe scores in untreated BPPV that normalise rapidly with successful repositioning [46,47]. Untreated BPPV in older adults is associated with a measurable, dose-dependent rise in falls and fall-related injury, and a small but consistent excess of depressive symptoms — both reversible with treatment [8,46].

Economic cost is non-trivial. The mean number of medical visits per BPPV episode in pre-guideline cohorts exceeded four, with widespread inappropriate imaging, vestibular suppressant prescribing, and emergency presentations [1,11]. The single-encounter cure model is the cleanest cost-saving in vestibular medicine: a clinic equipped to diagnose and treat at the first visit removes downstream investigation in over 80% of presentations.

Identified risk factors for incident BPPV align tightly with the inferred pathophysiology. Age, female sex, post-menopausal status, low bone-mineral density, low serum 25-OH vitamin D, prior head trauma, migraine, and recent inner-ear inflammation each independently elevate risk [25,26,27,30]. Less consistently associated are hypertension, diabetes, hyperlipidaemia, and hypothyroidism; these should be addressed when present but are not necessarily causal. Genetic predisposition is suggested by familial clustering but no single gene has been confirmed [27].

Under-diagnosis remains the dominant epidemiological problem. Active screening of older adults in primary care, geriatric, and rehabilitation settings consistently identifies a sizeable cohort of patients with undiagnosed BPPV who have been labelled with 'cervicogenic dizziness', 'multifactorial imbalance', or attributed to medication side-effects [8]. A simple positional history coupled with a Dix–Hallpike, performed once during the assessment, captures most of these patients. The implementation gap — not the diagnostic difficulty — is the principal barrier to better population-level outcomes [1,8].

□ **Key Point:** BPPV is mechanical, peripheral, and curable in one encounter. Every vestibular service must be able to diagnose and treat all canal variants — posterior, lateral, and anterior — at the first visit.

II. Pathophysiology — Otoconia, Canalithiasis and Cupulolithiasis

BPPV is generated by otolithic debris that has separated from the utricular macula and entered a semicircular canal. In the canal, the displaced otoconia render the endolymph gravity-sensitive — either by free-floating motion (canalithiasis) or by adhesion to the cupula (cupulolithiasis) — producing positional vertigo and a stereotyped nystagmus [1,2,11].

Otoconia are biomineralised calcium-carbonate (calcite) crystals approximately 3–10 µm in length, embedded in a gelatinous matrix above the utricular and saccular maculae. Normal otoconia turn over across the life span; degenerating crystals are reabsorbed and replaced under the regulation of inner-ear calcium metabolism, otoconin and otopetrin signalling, and a vitamin D / parathyroid-hormone axis shared with bone [25,26,30,48]. Disturbance of this calcium economy — by ageing, oestrogen withdrawal, vitamin D deficiency, head trauma, or labyrinthine ischaemia — increases the probability that crystals detach prematurely and enter a canal. This explains the female predominance after menopause, the steep age gradient, and the rising recurrence risk in osteopenic patients [25,26,30].

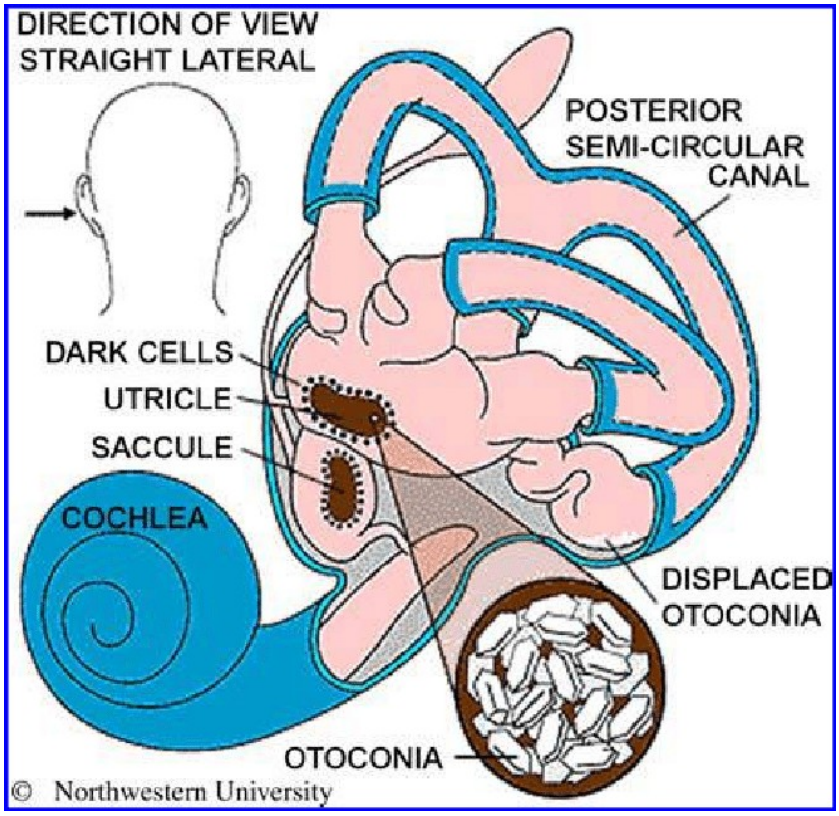

Figure 1. Inner-ear anatomy showing displacement of otoconia from the utricle into the posterior semicircular canal — the substrate of BPPV.

Source: Northwestern University Medical School educational diagram (used under educational fair use).

Canalithiasis

Free-floating otolithic particles within the semicircular canal lumen. With head movement in the plane of the canal, gravity pulls particles through the endolymph, generating a deflecting current on the cupula. This explains the characteristic latency of 1–5 seconds, crescendo–decrescendo nystagmus, transient duration (typically under 60 seconds), and fatigability with repeated provocation [1,4].

The hydrodynamics are well characterised. Particle drag through the viscous endolymph, combined with the inertia of the cupula, generates a force–time profile that matches the observed crescendo–decrescendo nystagmus. The latency represents the few seconds taken for particles to accelerate from rest and create a cupular deflection that exceeds the threshold for nystagmus generation. Fatigability —

diminishing response on repeat manoeuvres within a single sitting — is thought to reflect particle dispersion within the canal rather than habituation of central pathways [4,22].

Canalithiasis was the conceptual basis for Epley's canalith repositioning procedure [3], and histopathological evidence of particulate matter retrieved from the posterior canal during occlusion surgery confirms the mechanism [24,41]. Three-dimensional eye-movement recordings during positional testing demonstrate that the spatial vector of the nystagmus aligns precisely with the plane of the implicated canal — a finding that underpins canal-specific diagnosis and treatment [22].

Cupulolithiasis

Otoconia adherent to the cupula itself, rendering it gravity-sensitive. Positional change produces immediate-onset, persistent nystagmus lasting beyond 60 seconds in the provoking position, with little or no fatigability [5]. It is less common than canalithiasis overall but explains a defined subset of atypical horizontal-canal and anterior-canal presentations, as well as the apogeotropic horizontal variant.

Mechanically the persistence is straightforward: the adherent debris loads the cupula and deflects it for as long as the head remains in the provoking position, in contrast to free-floating particles whose stimulus dissipates once they settle. Therapeutically, cupulolithiasis typically requires a liberatory step — a rapid head movement designed to dislodge particles from the cupula — before the standard canalith repositioning sequence can be applied [15,19]. Cupulolithiasis is also the presentation most likely to be confused with central apogeotropic positional nystagmus, particularly in older adults; mechanism-aware bedside examination is the principal safeguard [33].

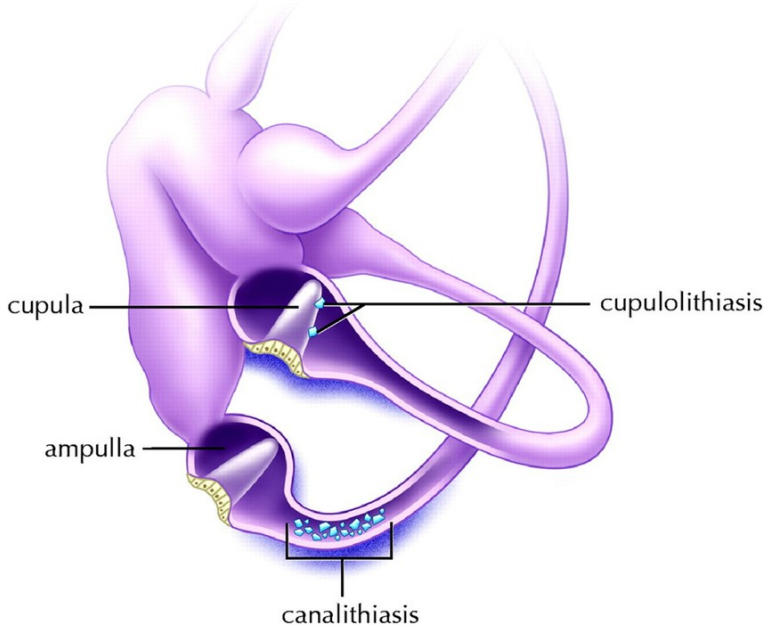

Figure 2. Left labyrinth showing the two mechanisms of BPPV — free-floating debris in the canal (canalithiasis, lower) versus debris adherent to the cupula (cupulolithiasis, upper).

Source: Adapted from Nuti et al., Benign paroxysmal positional vertigo (educational adaptation).

Canal distribution

The posterior canal is affected in 80–90% of cases by virtue of its dependent position, the horizontal canal in 5–15%, and the anterior canal in 1–5% [10,20,21]. The posterior canal predominates because in the upright head position its non-ampullary arm sits dependent within the labyrinth, behaving as a natural sump for shed otoconia. The horizontal canal is approximately coplanar with the earth in upright posture and therefore less likely to capture debris under physiological conditions, but is readily entered by particles displaced during supine immobility or rolling. The anterior canal is uppermost and so rarely receives otoconia — its rarity is anatomically explicable [10,11].

Multi-canal involvement occurs in approximately 2–10% — typically post-traumatic, post-Epley canal conversion, or following prolonged supine immobility [21,23,39]. A single patient may have canalithiasis in one canal and a contralateral cupulolithiasis in another. Bilateral simultaneous posterior-canal BPPV

is well described after head trauma and demands sequential, side-by-side treatment with brief intervals between Epley manoeuvres [23].

Atypical and secondary BPPV

Three atypical patterns warrant explicit recognition. The first is the apogeotropic posterior-canal variant, attributed to particles lodged in the short (non-ampullary) arm of the posterior canal; Dix–Hallpike provokes downbeating nystagmus with reversed torsion. The second is post-Epley canal conversion, in which otoconia migrate from the posterior into the horizontal canal during the manoeuvre, producing new geotropic or apogeotropic horizontal nystagmus that requires a different treatment plan [39]. The third is secondary BPPV — otoconia displacement triggered by another inner-ear pathology, most commonly Ménière's disease, vestibular neuritis, or recent otologic surgery; cure rates are lower and recurrence higher in this group [11,27,42].

A fourth, increasingly recognised pattern is canal jam — debris that wedges within a narrow canal segment and produces persistent positional nystagmus that does not respond to standard repositioning. Mechanical vibration, mannitol, or a mechanical repositioning chair sequence is sometimes effective; true jam is the principal indication for posterior-canal occlusion surgery in carefully selected patients [11,44].

□ **Clinical Insight:** Latency, duration and fatigability are the three bedside markers that separate canalithiasis from cupulolithiasis and, more importantly, from central positional nystagmus. Time the nystagmus — do not eyeball it.

III. Clinical Features by Canal Subtype

BPPV presents with brief, position-triggered vertigo. Patients describe vertigo on rolling in bed, lying down, sitting up, looking up, or bending forward. Episodes are sudden, last seconds to under a minute, and cluster in bouts over days to weeks before spontaneous remission or successful repositioning [1,2,11]. Nausea is common; vomiting suggests severe paroxysms. Between episodes patients are usually well, though older patients may report unsteadiness for hours after a bout. Neurological symptoms — diplopia, dysarthria, focal weakness, sensory disturbance — are not part of BPPV; their presence demands re-evaluation [10,11].

Three history elements are particularly informative. First, the trigger profile: pure positional triggers without spontaneous attacks point to BPPV; mixed positional plus spontaneous attacks suggest vestibular migraine, Ménière's disease, or a central disorder. Second, the attack length: BPPV attacks are seconds, never minutes — patients who report ten-minute vertiginous spells with positional triggers are describing a different syndrome regardless of how the attack 'feels'. Third, the auditory history: any ipsilateral hearing change, tinnitus or aural fullness redirects the work-up toward Ménière's, perilymph fistula, or superior canal dehiscence rather than BPPV [10,11,49].

Posterior canal BPPV (PC-BPPV)

The classic presentation. Dix–Hallpike to the affected side elicits a torsional-upbeating nystagmus, with the upper poles of the eyes beating toward the dependent ear and a vertical upbeat component. Latency 1–5 seconds, duration under 60 seconds, fatigues on repeat [1,22]. Reversal nystagmus on sit-up is supportive but not required. Sustained nystagmus beyond 1–2 minutes without fatigue suggests posterior-canal cupulolithiasis or a central mimic. Sleep position correlates with the side of posterior-canal involvement, supporting a gravity-dependent migration of otoconia during recumbency [9].

Quantitative three-dimensional recordings demonstrate that the torsional and vertical vectors of PC-BPPV nystagmus combine in a plane that mirrors the affected posterior canal, with the vertical component arising from the canal's elevation angle and the torsional component from its lateral deviation [22]. In clinical practice, the dependent-ear rule — that the upper poles of the eyes beat toward the lower ear — remains the most reliable bedside identifier of the affected side.

Horizontal (lateral) canal BPPV (HC-BPPV)

Patients describe vertigo on rolling side-to-side in bed or on horizontal head turning. The supine roll test (Pagnini–McClure) is diagnostic [10]. Two patterns:

- **Geotropic** — horizontal nystagmus beating toward the ground-ward ear on either side; canalithiasis of the long arm of the lateral canal. The stronger side identifies the affected ear (Ewald's law) [20].
- **Apogeotropic** — horizontal nystagmus beating away from the ground-ward ear; cupulolithiasis or anterior-arm canalithiasis. Counter-intuitively, the weaker side is the affected side [19,20].

Latency is typically zero or very brief in HC-BPPV. Canalithiasis fatigues in under a minute; cupulolithiasis tends to persist as long as the head is in the provoking position.

Side identification in HC-BPPV depends on Ewald's second law and the geotropic versus apogeotropic pattern. In the geotropic variant the stronger nystagmus identifies the affected ear, because rolling the affected canal down places the long arm of the canal in the gravity-favoured position for ampullopetal flow. In the apogeotropic variant the weaker nystagmus identifies the affected side, because the heavy cupula deflects in the opposite direction to a normal excitation [19,20]. Pseudo-spontaneous nystagmus — a horizontal nystagmus seen even in the upright position before any head turn — supports a horizontal-canal substrate, with the fast phase typically beating away from the affected ear in canalithiasis and toward the affected ear in cupulolithiasis [10,20].

HC-BPPV is also the principal substrate of post-Epley canal conversion. When new horizontal nystagmus develops within minutes to hours after a successful posterior-canal repositioning, the working diagnosis is canal conversion, not treatment failure; supine roll testing identifies the new pattern and informs the next manoeuvre [39].

Anterior canal BPPV (AC-BPPV)

Uncommon. The hallmark is downbeating positional nystagmus, often with a small torsional component, on Dix–Hallpike or straight head-hanging. The anterior canal of the ear opposite the torsion is usually implicated [22,31]. Anterior-canal BPPV is a diagnosis of exclusion — pure persistent downbeat without torsion, no latency, no fatigability, or any neurological sign points strongly to a central cause (craniocervical junction, cerebellar vermis, drug effect) and warrants MRI before therapeutic manoeuvres [32,33,45].

The series describing AC-BPPV are dominated by patients in whom central mimics were not formally excluded, and contemporary consensus is that a substantial proportion of reported AC-BPPV in older literature was central positional downbeat nystagmus from cerebellar pathology. In a dedicated vestibular service, the threshold for MRI in suspected AC-BPPV should be low [31,45].

□ **Important:** Treat persistent positional downbeat nystagmus without clear torsion as central until imaging excludes a posterior-fossa lesion. Repositioning manoeuvres performed on undiagnosed central pathology cause diagnostic delay.

IV. Diagnostic Manoeuvres and Bárány Society Criteria

Diagnosis is clinical, based on history plus a positive provocation manoeuvre with a canal-specific nystagmus pattern. The Bárány Society 2015 consensus formalised diagnostic criteria for each canal and mechanism, and introduced the categories of probable (spontaneously resolved) and possible BPPV for incomplete presentations [2,34].

Provocation testing

- **Dix–Hallpike —** the standard for posterior- and (sometimes) anterior-canal BPPV. Test both sides. Maintain the head-hanging position for at least 30 seconds before reading negative [1,11].
- **Supine roll test (Pagnini–McClure) —** essential when Dix–Hallpike is negative but history is positional. Roll 90° to each side, observe 30 seconds each [10,20].

- **Straight head-hanging —** targeted test for anterior-canal BPPV; also forms part of the Yacovino therapeutic sequence [14,31].

Technique matters. The patient must be moved briskly into the head-hanging position; a slow Dix–Hallpike may fail to provoke posterior-canal canalithiasis even when present. The neck is extended by 20° below horizontal — typically with a small pillow under the shoulders rather than under the head — and the head turned 45° toward the test side. The position is held for at least 30 seconds before declaring the test negative, and ideally for 60 seconds when a confident negative is needed [1,11]. Modifications for cervical-spine limitation include the side-lying (Semont-style) diagnostic test, in which the patient is moved from sitting to a side-lying position with the head rotated 45° away from the dependent shoulder; the head-hanging is avoided without sacrificing diagnostic yield [10,15].

Video-Frenzel goggles or VNG remove visual fixation and magnify the eyes, approximately doubling sensitivity in trained hands; their routine use is recommended in any dedicated vestibular service [10,11]. Goggles also allow recording of the nystagmus for documentation, peer review, and patient education — a measurable benefit in medico-legal practice.

Table 2. Bárány Society diagnostic criteria — abridged for vestibular-physician use [2,34].

Subtype	Provoking position	Nystagmus signature	Time profile
PC canalithiasis	Dix–Hallpike (affected side down)	Torsional-upbeat toward dependent ear	Latency 1–5 s; duration <60 s; fatigues
HC canalithiasis (geotropic)	Supine roll, both sides	Horizontal geotropic; stronger toward affected side	Brief or no latency; <60 s; fatigues
HC cupulolithiasis (apogeotropic)	Supine roll, both sides	Horizontal apogeotropic; weaker toward affected side	No latency; persists >60 s; no fatigue
AC canalithiasis	Straight head-hang or Dix–Hallpike (either side)	Downbeat with small torsion (top of eye toward affected ear)	No or brief latency; usually <60 s

Definite, probable, and possible BPPV

The Bárány Society criteria allow three diagnostic categories. Definite BPPV requires the full set: characteristic history, a positive provocation manoeuvre with a canal-specific nystagmus pattern, and no alternative explanation [2,34]. Probable (spontaneously resolved) BPPV is used when the history is unambiguous but provocation is negative at the time of testing — recognising the well-documented natural remission of attacks. Possible BPPV is reserved for incomplete or atypical presentations that do not meet definite criteria but where alternative diagnoses have been considered and excluded. This classification preserves diagnostic precision while accommodating the realities of late presentation and partial findings [2,34,35].

Beyond binary positive/negative

Provocation testing is not a yes/no test. A graded read is more useful in practice. Robust positive: characteristic latency, classic vector, brisk crescendo–decrescendo, and patient-reported vertigo matching the history — proceed directly to the matched manoeuvre. Equivocal positive: nystagmus present but vector imperfect, vertigo mild, fatigability uncertain — repeat the provocation after a 60-second interval, consider video-Frenzel recording, and test the unprovoked canal. Atypical: persistent nystagmus, wrong vector, or pure downbeat — escalate to imaging before any therapeutic manoeuvre. Negative with positive history: time the test (was 30+ seconds held?), test the supine roll, and consider 'probable BPPV (spontaneously resolved)' per the Bárány criteria [2,34].

Common diagnostic errors

Five recurring errors account for most of the misdiagnosis and 'refractory BPPV' that reaches the vestibular physician. Identifying them in routine practice halves the burden of avoidable referrals.

- **Premature termination of Dix–Hallpike —** the patient is sat up at the first complaint of dizziness, before the 1–5 second latency has elapsed and the diagnostic nystagmus has emerged. Hold the position for 30–60 seconds.
- **Failure to test the horizontal canal —** Dix–Hallpike alone misses HC-BPPV. A positional history with negative Dix–Hallpike mandates a supine roll before any 'refractory' label.
- **Misreading apogeotropic nystagmus —** the weaker side is the affected side in HC cupulolithiasis. Treating the wrong canal worsens rather than improves the patient.
- **Diagnosing AC-BPPV in pure persistent downbeat —** any persistent downbeat without torsion or fatigue is central until imaging excludes it.
- **Calling residual unsteadiness 'refractory BPPV' —** post-cure unsteadiness for 24–72 hours is normal, particularly in older patients. Confirm the provocation test is now negative before re-treating.

□ **Clinical Pearl:** If the Dix–Hallpike is negative but the history is unambiguously positional, the next test is the supine roll. Failing to test the horizontal canal is the single commonest reason for 'refractory BPPV' in primary care.

V. Investigations and the Role of Imaging

BPPV is a clinical diagnosis. Bárány Society and AAO-HNS guidelines explicitly recommend against routine vestibular function tests or imaging when the history and provocation findings meet criteria [1,2]. Caloric responses, vHIT, and VEMPs are normal between attacks and are not required for diagnosis [11].

Imaging is indicated only where atypical features raise the possibility of a central mimic: persistent downbeat without torsion, no latency or fatigability, additional neurological signs, or failure to respond to a correctly performed manoeuvre on more than one occasion [32,33,45]. MRI with high-resolution posterior-fossa imaging is the modality of choice; CT is reserved for the rare circumstance where MRI is contraindicated. Delayed-gadolinium hydrops MRI sequences have no role in the diagnostic work-up of BPPV but may be relevant where the differential includes Ménière's disease [50].

Audiometry should be considered if the patient reports any auditory symptom (hearing loss, tinnitus, aural fullness), which would point away from isolated BPPV. Bone-densitometry and serum 25-OH vitamin D are reasonable in recurrent BPPV, particularly in post-menopausal women, given the established link between low bone mineral density, vitamin D deficiency, and both first-episode BPPV and recurrence risk [25,26,30].

Routine vestibular function testing (caloric, vHIT, VEMP) adds nothing in typical BPPV and may generate misleading results — particularly when residual unsteadiness after Epley is interpreted as evidence of additional pathology. Reserve these tests for cases where the history or examination suggests a second vestibular diagnosis (vestibular migraine, vestibular neuritis sequelae, bilateral vestibulopathy) alongside the BPPV [11,29].

□ **Key Point:** Order vestibular tests and imaging only when the clinical picture does not fit. A normal MRI does not validate a BPPV diagnosis; the validation is a positive provocation test and a positive therapeutic response.

Table 3. Red flags warranting MRI before repositioning manoeuvres.

Red flag	Concern
Pure persistent downbeat nystagmus, no torsion	Cerebellar/craniocervical lesion
No latency, no fatigability, persistent >2 min	Central positional nystagmus
Direction-changing nystagmus in supine	Central or apogeotropic with central mimic
Any focal neurological sign (dysarthria, INO, ataxia)	Posterior-circulation lesion
Acute unilateral hearing loss with positional vertigo	AICA stroke or labyrinthitis
Failure to respond to two well-performed manoeuvres	Reassess diagnosis or canal; image if persistent

VI. Differential Diagnosis of Positional Vertigo

The principal differentials are central positional vertigo (CPV) and vestibular migraine (VM) with positional features. Less often, orthostatic dizziness, superior canal dehiscence (SCD) with Tullio phenomenon, or perilymph fistula can present with positional triggers — but with auditory features that BPPV lacks [11,32,33,49].

Superior canal dehiscence merits specific mention: a thin or absent bony covering of the superior semicircular canal exposes the labyrinth to pressure and sound, producing vertigo on Valsalva, loud sound (Tullio), or head movement. The pattern is usually accompanied by autophony, conductive hyperacusis, and the characteristic upbeat-torsional nystagmus in the plane of the dehiscent canal. High-resolution temporal-bone CT confirms the diagnosis; SCD does not respond to Epley [49].

Perilymph fistula presents with vertigo and hearing change after barotrauma or head injury and can mimic post-traumatic BPPV. The discriminating features are the auditory symptoms and the failure of repositioning to resolve the vertigo. Orthostatic dizziness is the most easily missed mimic in older patients on polypharmacy — lying-standing blood pressure is mandatory in any 'dizzy elderly' presentation before a vestibular label is applied [11].

Drug-induced positional symptoms — particularly with sedating antihistamines, benzodiazepines, tricyclic antidepressants, and serotonergic agents — can produce a chronic 'positional unsteadiness' that patients describe in BPPV-like terms but without true positional triggers or canal-specific nystagmus. The history (recent introduction or dose change) and a negative provocation test resolve the question. Cervicogenic dizziness is over-diagnosed: a structured BPPV work-up should be completed before this label is applied [11,29].

Persistent postural–perceptual dizziness (PPPD) frequently arises after a precipitating BPPV episode and presents as chronic non-positional unsteadiness with visual sensitivity, persisting beyond resolution of the underlying mechanical fault. Recognising the PPPD overlay is essential — repeated Epley manoeuvres on a structurally cured patient cannot resolve PPPD and the patient should be redirected to vestibular rehabilitation and PPPD-specific care [11,52].

Table 4. Differentiating BPPV from central positional vertigo and vestibular migraine.

Feature	BPPV	Central positional vertigo	Vestibular migraine
Latency	1–5 s (canalithiasis)	None	None or brief
Duration	<60 s	Persistent as long as position held	Minutes to hours
Nystagmus	Matches canal plane; fatigues	Often pure downbeat or pure torsional; no fatigue	Mild or absent; non-canalicular
Associated signs	None	Ataxia, dysarthria, INO, gaze-evoked nystagmus	Headache, photophobia, aura
Response to repositioning	Excellent	None	None (treat as migraine)

□ **Clinical Insight:** Vestibular migraine and BPPV co-exist more often than expected — VM patients have a measurably higher lifetime BPPV risk. When repositioning resolves the nystagmus but residual dizziness persists, screen for migraine biology before labelling 'refractory'.

VII. Repositioning Manoeuvres and Outcomes

Canalith repositioning procedures (CRPs) are the first-line treatment for every BPPV subtype. The manoeuvre is selected by the canal involved and the mechanism inferred from the nystagmus signature [1,17,18].

Posterior canal — Epley and Semont

The Epley manoeuvre relocates posterior-canal otoconia through sequential head-and-body rotations into the utricle [3]. A single Epley resolves BPPV in approximately 80% of patients; cumulative resolution exceeds 90% with one to three treatments [17,18,37,43]. The Semont liberatory manoeuvre achieves

comparable cure rates and is the manoeuvre of choice in patients with limited cervical extension [15,39,43].

Epley technique in detail: from sitting with the head turned 45° toward the affected side, the patient is moved briskly into the Dix–Hallpike head-hanging position and held until any provoked nystagmus settles, typically 30–60 seconds. The head is then rotated through 90° to the opposite side (now 45° from neutral toward the unaffected ear), held for a further 30–60 seconds, and the body and head rotated a further 90° so that the patient is in lateral decubitus with the face approximately 45° below horizontal. After a similar hold, the patient is returned to sitting with the head still rotated. Each phase should be quick rather than gentle — particle migration depends on a forceful gravitational swing through the canal [3,17].

The Semont liberatory manoeuvre takes the patient from sitting to side-lying on the affected side with the head turned 45° upward, then swings the patient rapidly through 180° to the opposite side-lying with the head now facing the bed; this rapid swing is the liberatory step. Outcomes are indistinguishable from Epley in head-to-head trials, but the Semont requires less neck extension and is preferred in patients with cervical spondylosis or vertebrobasilar insufficiency [15,39,43].

A common modification — the Semont-plus — increases the speed of the side-lying swing and extends the final position hold; a 2018 randomised comparison found a small but statistically significant improvement in first-session cure compared with the standard Semont. The principal limitation is operator force and patient tolerance in the elderly; the standard Semont remains the default in older patients [15,39].

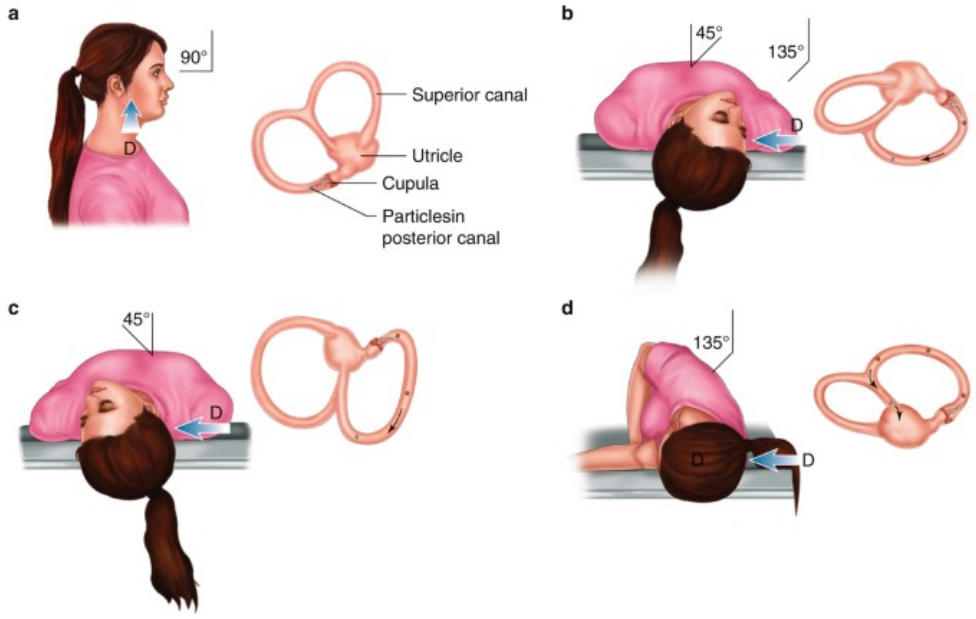

Figure 3. Epley canalith repositioning sequence for right posterior-canal BPPV. Particles are walked through the canal back to the utricle.

Source: Springer educational figure — used for clinician education only.

Figure 4. Semont liberatory manoeuvre for left posterior-canal BPPV — rapid side-to-side swing dislodges otoconia from the canal.

Source: Educational clinical photograph — used for clinician education only.

Horizontal canal — Lempert (BBQ) roll and Gufoni

For geotropic HC-BPPV, the Lempert 360° barbecue roll moves debris around the horizontal canal toward the utricle. Single-session cure rates are 70–75%, rising to 85–90% with two to three sessions [12,38]. The Gufoni manoeuvre is equally effective, requires less mobility, and is often better tolerated in older patients [13,19]. For apogeotropic (cupulolithiasis) patterns, a modified Gufoni or head-shaking can convert the pattern to geotropic, which is then treated by standard roll or Gufoni [19,39].

Lempert roll technique: from supine with the head elevated 30°, the patient rolls in 90° increments away from the affected ear, holding each position for 30–60 seconds, completing a full 360° rotation back to supine. The rationale is to carry debris around the long arm of the horizontal canal and into the utricle by sequential gravity vectors. The Gufoni manoeuvre is a single rapid side-lying step with the head rotated 45° downward, designed to lift debris out of the canal in one liberatory move [12,13,38].

Apogeotropic management is more nuanced. Vigorous horizontal head-shaking with the head pitched 30° forward can detach particles from the cupula and convert the nystagmus to a geotropic pattern, which then responds to standard manoeuvres. The Gufoni-Appiani variant performs the same function by a single rapid head turn while side-lying. Re-evaluation after each intervention is essential — conversion may occur in either direction during a single session [19,39].

Anterior canal — deep head-hanging (Yacovino)

The Yacovino deep head-hanging manoeuvre moves the patient rapidly from upright to deep head-hanging, then through staged head and body re-positioning [14]. It does not require knowledge of the affected side because the manoeuvre operates in the sagittal plane. Single-session cure rates approximate 80%; cumulative resolution after two to three sessions approaches 100% [14,31]. The deep head-hanging component is poorly tolerated in cervical-spine disease — modifications using inclined surfaces have been described and are reasonable where the standard manoeuvre cannot be performed [31].

Mechanically, the Yacovino works because the anterior canal in either ear shares a common sagittal plane with the head; a deep head-hanging position drops otoconia in the affected anterior canal toward the utricle through that shared plane regardless of left- or right-sidedness. Where the downbeat is clearly

torsional and lateralises to one side, the more selective reverse Epley to the opposite ear can also be used, but the side-independent Yacovino is procedurally simpler and equally effective in most series [14,31].

Pre-manoeuvre safety and consent

Before any positional therapeutic manoeuvre, screen for the small number of contraindications: unstable cervical-spine pathology (rheumatoid C1–C2 instability, atlanto-axial subluxation), severe carotid or vertebral artery stenosis with symptomatic ischaemia on neck extension, recent retinal detachment surgery, severe cardiac decompensation, and acute spontaneous spinal CSF leak. These are absolute or relative contraindications and should redirect to Semont-style or side-lying modifications, or to Brandt–Daroff exercises [10,11]. Provoked vertigo and transient nausea are expected during the manoeuvre and do not constitute adverse events; warn the patient explicitly, provide an emesis bag, and have an assistant on hand for older or frailer patients.

Multi-canal disease

Two-to-ten per cent of presentations involve more than one canal — typically post-traumatic, post-Epley conversion, or in secondary BPPV from another inner-ear disorder [21,23,39]. The treatment principle is sequential canal-by-canal repositioning: identify the most florid pattern and treat it first, repeat provocation testing once symptoms settle, then treat the residual pattern. Combined manoeuvres that purport to treat two canals in one sequence are generally less effective than serial single-canal manoeuvres and risk additional canal conversions. Where ambiguity persists, a mechanical repositioning chair allows precise canal isolation and is the practical solution.

Post-manoeuvre management

Older teachings instructing patients to sleep upright, avoid the affected side, or refrain from specific head positions for 24–48 hours after Epley have been formally tested and shown to confer no benefit. The 2017 AAO-HNS guideline explicitly recommends against post-manoeuvre postural restrictions [1]. Patients should be reassured that normal movement and sleep posture are safe and that residual unsteadiness for one to three days is usual, particularly in older patients, without representing treatment failure.

Outcome should be confirmed at the same encounter where possible — a negative repeat provocation test immediately after the manoeuvre is the strongest bedside marker of success and predicts low one-week recurrence [18,37]. Where same-encounter repeat is impractical (a heavily symptomatic patient), telephone or video follow-up at one to two weeks is appropriate.

Table 5. Subtype frequency, preferred manoeuvre, and expected outcome.

Subtype	Approx. share of BPPV	Preferred manoeuvre(s)	Cure rate
PC canalithiasis	80–90% [10,20]	Epley; Semont [3,15]	~80% (1 session); >90% (2–3) [17,18]
HC geotropic	5–15% [20]	Lempert BBQ roll; Gufoni [12,13]	70–75% (1); 85–90% (2–3) [38]
HC apogeotropic	~5%	Modified Gufoni; convert then treat [19]	50–70% (1); 80–90% (cumulative) [19]
AC canalithiasis	1–5% [22]	Yacovino deep head-hang [14]	~80% (1); ~95–100% (2–3) [14,31]
Multi-canal	2–10% [21,23]	Treat canal by canal	≥90% cumulative across canals

□ **Clinical Pearl:** Re-test in the same encounter after repositioning. A negative Dix–Hallpike immediately after Epley is the most reliable bedside marker of treatment success and predicts low one-week recurrence.

VIII. Adjuncts, Refractory Disease, and Surgical Options

Brandt–Daroff exercises

A habituation exercise performed by the patient at home, useful when the affected side is uncertain, when manoeuvres cannot be done in clinic, or as residual-symptom maintenance after successful repositioning [16]. They are slower than CRPs but durable, and an evidence-based option when a self-directed protocol is needed.

Standard scheme: the patient sits upright, rotates the head 45° away from the side to be tested, then rapidly assumes a side-lying position with the head still rotated upward. The position is held for 30 seconds or until vertigo resolves, the patient returns to upright for 30 seconds, then repeats on the opposite side. A complete cycle comprises five repetitions on each side, performed three times daily until two consecutive symptom-free days are achieved (typically 7–14 days). The mechanism is habituation rather than repositioning, which explains the slower response but durable result [16].

Mechanical adjuncts

Mastoid vibration during Epley has been studied as an adjunct to dislodge adherent otoconia; meta-analyses show a small, inconsistent benefit and routine use is not recommended [17,18]. Mechanical repositioning chairs (Epley Omniax and similar) deliver standardised, multi-axis rotations under controlled conditions; they are particularly useful in obese patients, those with mobility limitations, multi-canal disease, or refractory cases requiring precise canal-specific positioning. Cost and footprint constrain widespread adoption, but in dedicated vestibular services they enlarge the population for whom curative treatment is feasible.

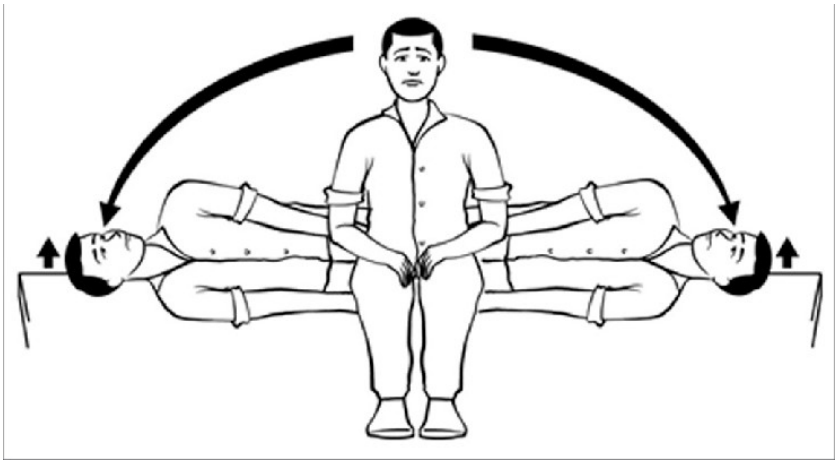

Figure 5. Brandt–Daroff habituation exercise — patient moves from upright to side-lying on each side in turn, with the head rotated 45° away from the dependent ear, until vertigo subsides.

Source: Educational illustration — used for clinician education only.

Pharmacotherapy

Vestibular suppressants (antihistamines, benzodiazepines) do not treat BPPV and should be avoided; they impair central compensation and add side-effect burden in the elderly [1,29]. Short-course antiemetic use is reasonable for severe nausea during paroxysms but should not delay definitive repositioning.

Refractory disease

Approximately 5% of patients fail two well-performed manoeuvres on separate occasions. In this group, re-confirm the canal and mechanism (often a missed HC apogeotropic pattern or post-Epley canal conversion to AC), screen for vitamin D deficiency and osteopenia [25,26,30], and re-examine for atypical features that warrant MRI. Mechanical repositioning chairs (Epley Omniax and similar) may help where comorbidity limits manual technique.

Surgery

Posterior-canal occlusion surgery is the last-resort option for disabling, truly refractory PC-BPPV. The procedure exposes the membranous posterior canal through a transmastoid approach and mechanically occludes it with bone pâté or fascia, abolishing endolymph flow within the canal while preserving the rest of the labyrinth. Cure rates exceed 90%; transient post-operative imbalance is expected and resolves within weeks. The principal risk is a small but real (~3–5%) incidence of sensorineural hearing loss, which is the basis for strict selection criteria — disabling, multi-year, manoeuvre-resistant, single-canal disease, with normal contralateral hearing and explicit informed consent [44]. Singular neurectomy (sectioning the nerve to the affected canal) is historical and is no longer performed. Surgery for HC- or AC-BPPV is essentially never required.

□ **Important:** Before invoking 'refractory BPPV', verify that the supine roll and head-hang have been performed, the manoeuvre matches the pattern, and the nystagmus has been timed. Most refractoriness is misdiagnosis or wrong-canal treatment.

IX. Prognosis, Recurrence, and Predisposing Factors

Without treatment, BPPV remits spontaneously in most patients within weeks to months, though episodes may persist far longer in the elderly [28,35]. With repositioning, immediate cure rates exceed 90% across subtypes [17,18].

Recurrence is common — 15–30% within one year, 40–50% over five years, with higher rates in older patients, women, and those with osteopenia or vitamin D deficiency [27,40]. Idiopathic BPPV is the predominant aetiology; identifiable triggers include head trauma, prolonged supine immobility, recent otologic surgery, ipsilateral migraine, and inner-ear pathology such as Ménière's disease or vestibular neuritis [23,27,42].

Table 6. Natural history and recurrence — key numbers for patient counselling.

Parameter	Adult value
Spontaneous remission (untreated, weeks–months)	~50% at 3 months; ~80% at 12 months [28,35]
Immediate cure with appropriate manoeuvre	~80% single session; >90% within 2–3 sessions [17,18]
Recurrence — 1 year	15–30% [27,40]
Recurrence — 5 years	40–50% [27,40]
Higher-risk subgroups	Female, >60 y, osteopenia, low vitamin D, migraine, post-trauma [25–27,30]

Recurrence is not failure of the previous manoeuvre — the repositioning resolved the index presentation, and the new event reflects fresh otoconial displacement. Patients counselled on this distinction are markedly less distressed by recurrence and re-attend earlier. Where recurrence is frequent (more than two episodes per year), screen for vitamin D deficiency and osteopenia, optimise any contributing comorbidity (migraine, sleep-deprivation, alcohol), and consider teaching a home self-Epley protocol for typical recurrences [37,52].

Vitamin D and bone-mineral density have an established association — low 25-OH vitamin D and osteopenia are independent risk factors for BPPV and recurrence, and vitamin D repletion modestly reduces recurrence in deficient patients [25,26,30]. Routine measurement is reasonable in recurrent cases. Migraine biology, particularly in women, independently increases BPPV risk and probably accounts for some of the apparent overlap between vestibular migraine and BPPV recurrence [27,42].

Special populations

Older adults: cumulative recurrence and falls risk both increase with age; a low threshold for treatment, vitamin D screening, and falls-prevention referral is appropriate. Cervical spondylosis may necessitate

Semont or side-lying modifications. Post-stroke patients with persistent positional complaints should be re-examined specifically for BPPV — secondary BPPV is over-represented in this group and is often missed in the broader rehabilitation context [27,42].

Post-traumatic BPPV: head injury is the most identifiable single trigger and accounts for a disproportionate share of multi-canal and bilateral BPPV [23]. Cure rates per session are slightly lower, and recurrence rates higher, than in idiopathic disease — a useful framing point for patient counselling.

Co-existing vestibular conditions

BPPV co-exists with other vestibular disorders more often than chance would predict. Ménière's disease, vestibular migraine, vestibular neuritis sequelae, and post-stroke vestibular dysfunction each elevate the risk of secondary BPPV [11,27,42]. The clinical consequences are practical. First, the patient may have two diagnoses; treating only one will leave residual symptoms. Second, the BPPV component may be less recognisable because the dominant disorder's symptoms blur the picture. Third, recurrence rates are substantially higher when an underlying disorder remains active, particularly Ménière's disease and vestibular migraine. A working principle: in any patient with chronic vestibular complaints and a new positional component, perform full provocation testing — the BPPV may be the most reversible part of the presentation.

Activity, work, and rehabilitation

After a successful manoeuvre most patients can resume normal activity immediately, including their usual sleeping posture. Residual unsteadiness over the first 24–72 hours is normal and not a reason for activity restriction. Vestibular rehabilitation is not routinely required after a single cured BPPV episode but adds value in two specific groups: older patients with measurable balance decrement and residual gait unsteadiness, and patients with a co-existing chronic vestibular disorder or PPPD overlay. Brief, targeted gaze-stabilisation and balance work over two to four weeks is sufficient in most cases [11,52].

Return to work is usually same-day for sedentary roles. Roles requiring head movement at height, operation of heavy machinery, or driving as core duties should be deferred until a confident negative provocation test and 24-hour symptom resolution have been demonstrated. The clinical record should specify the advice given, the criteria for return, and the documented outcome of the immediate post-manoeuvre re-test [11,51].

Fitness to drive

Active BPPV — by definition triggered by positional change rather than spontaneous — is generally compatible with private driving once symptoms are controlled with repositioning, in line with Austroads guidance [51]. Patients should be advised not to drive while attacks remain unprovoked or while severe nausea or anticipatory anxiety persist. Commercial drivers warrant a more conservative stance and individualised review. Document the advice given and the patient's understanding clearly in the clinical record [51].

□ **Clinical Insight:** Tell every patient post-Epley that recurrence is normal, not failure. A clear plan for self-recognition and re-attendance halves anxiety and shortens time-to-treatment on relapse.

X. Guidelines, Controversies and Future Directions

Modern practice is anchored by the AAO-HNS clinical practice guideline (2017 update) and the Bárány Society 2015 consensus diagnostic criteria [1,2]. Both endorse positional manoeuvre-based diagnosis, immediate repositioning as first-line treatment, and avoidance of routine imaging or vestibular suppressant medication. Post-manoeuvre postural restrictions are not required and were removed from the 2017 update [1].

Controversies

- **Post-manoeuvre restrictions —** multiple trials show no benefit; the 2017 AAO-HNS guideline explicitly recommends against instructing patients to sleep upright [1].

- **Pharmacological adjuncts —** betahistine, antihistamines, and benzodiazepines have no role in BPPV proper, though they are frequently and inappropriately prescribed [1,29].
- **Mechanical repositioning chairs —** equivalent to manual manoeuvres for outcome; useful in patients with mobility limitations or multi-canal disease, but cost and availability constrain adoption.
- **Vitamin D supplementation —** emerging trial evidence supports vitamin D repletion in deficient patients with recurrent BPPV, with a modest absolute reduction in recurrence over 12 months [26,30]. Reasonable as adjunctive care in osteopenic and post-menopausal cohorts.
- **Home self-Epley protocols —** safe and effective in patients with prior in-clinic cure who recognise classic recurrence; loses access to canal-conversion identification and is not appropriate for first presentations [52].
- **Anterior canal classification —** the Bárány criteria treat AC-BPPV as a distinct entity, but its prevalence is genuinely small and central mimics dominate the persistent-downbeat differential [31,32,45].

Patient counselling and education

Effective counselling has four elements. First, name the mechanism in plain language — 'small calcium crystals in the wrong part of the inner ear, moved out by the manoeuvre we just performed'. Second, set expectations for residual unsteadiness over the first 24–72 hours and for the safe return to normal activity, including sleep posture and driving. Third, frame recurrence as a physiological rebound risk, not a treatment failure, and define the simple criteria for re-attendance. Fourth, identify the small subset whose chronic dizziness will not resolve with repositioning — these patients need a different conversation about vestibular migraine, PPPD, or another co-existing disorder and a different care plan from the outset [11,52].

Telemedicine and home self-treatment

Video-based consultation has been validated for posterior-canal BPPV when the patient or carer can perform a Dix–Hallpike and the clinician can observe the eyes through a webcam or smartphone with adequate resolution [52]. Self-administered home Epley protocols, taught after an in-clinic cure, are effective in recurrent BPPV and are an evidence-based option for patients with recurrent stereotyped episodes [37,52]. The trade-off is loss of access to canal conversion detection — for first presentations, in-person evaluation remains the standard.

Future directions

Active research areas include: refined criteria for central positional nystagmus that distinguish cerebellar from peripheral apogeotropic patterns [32,33]; the role of bone metabolism and vitamin D biology in recurrence [26,30]; head-mounted video systems for home diagnosis and remote monitoring [52]; individualised post-Epley protocols that triage patients to in-clinic vs. self-administered home Epley by recurrence risk [45]; and machine-learning analysis of nystagmus video for automated canal identification, currently under prospective evaluation [52].

Two longer-horizon directions deserve mention. The first is mechanistic — better understanding of the bone-otoconia axis may yield a primary-prevention strategy for high-risk groups, particularly post-menopausal women with osteopenia. The second is system-level: vestibular-physician-led BPPV clinics, embedded within primary care or as standalone day-services, halve time-to-treatment and appropriate-use of imaging compared with usual care pathways. The model is replicable and the outcome data are persuasive for funders [11,52].

□ **Key Point:** BPPV is the cleanest condition in vestibular medicine — mechanism known, criteria defined, treatment validated, outcome measurable in a single visit. A vestibular service should resolve more than 90% of cases at first attendance.

Summary

BPPV is mechanically simple, diagnostically clean, and therapeutically rewarding. The vestibular physician's value sits in three places: at the front end, distinguishing peripheral BPPV from central mimics by mechanism-aware bedside examination; in the manoeuvre itself, choosing the right canal-specific technique and executing it briskly; and in the aftercare, framing recurrence as physiology rather than failure and identifying the small subset whose 'BPPV' is actually a central, migrainous, or PPPD-overlay condition. A service that owns all three steps delivers single-encounter cure rates above 90% and the cleanest outcome-cost ratio in vestibular medicine [1,11].

Five practice points consolidate this review:

- **Test every relevant canal —** Dix–Hallpike both sides, then supine roll if Dix–Hallpike is negative. Untested canals are the single largest source of false-refractory BPPV.
- **Time the nystagmus —** latency, duration, and fatigability separate canalithiasis, cupulolithiasis, and central mimics. Eyeballing it is the mistake.
- **Match manoeuvre to mechanism —** Epley/Semont for posterior canalithiasis; Lempert or Gufoni for horizontal canal, with conversion step for apogeotropic; Yacovino for anterior canal.
- **Image when the pattern does not fit —** persistent downbeat without torsion, no fatigue, or any focal sign is central until MRI proves otherwise.
- **Counsel for recurrence —** normalise the rebound risk, define re-attendance criteria, and screen bone health in recurrent disease.

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