BPPV:

Posterior, Horizontal, Anterior, and Atypical Presentations

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How to Use This Review

This comprehensive review provides vestibular physiotherapists with detailed guidance on diagnosing and treating benign paroxysmal positional vertigo (BPPV) across all canal variants. It addresses pathophysiology, diagnostic manoeuvres, repositioning techniques, post-manoeuvre management, and treatment-resistant cases.

The document follows a structured clinical format with numbered sections, integrated callout boxes for rapid reference, summary tables, and a references section. It is designed both as a learning resource and a quick-reference tool for practising clinicians.

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

 Clinical Insight: Clinically relevant observations derived directly from the evidence — for direct application in assessment and diagnosis.

 Clinical Pearl: High-yield, memorable clinical points — the take-home messages most likely to influence management or examination performance.

Contents

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I. Introduction and Epidemiology

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo in the general population, accounting for 20-30% of all vertigo cases presenting to dizziness clinics [1,13]. Lifetime prevalence is approximately 2.4%, with peak incidence between 50-60 years of age and a 2-3:1 female predominance [1].

BPPV is characterised by brief episodes of vertigo (<1 minute typically) triggered by specific head movements, accompanied by characteristic nystagmus that depends on the affected canal [13]. Despite high prevalence, BPPV is frequently misdiagnosed, leading to unnecessary investigations and delayed treatment in many patients [12,13].

 Clinical Pearl: BPPV is highly treatable: 80-90% of cases achieve symptom resolution with single manoeuvre success rates of 70-90%, and 2-3 sessions achieve near 100% success in most cases.

The distribution of BPPV variants differs from historical literature. Posterior canal BPPV remains most common (80-90% of cases), but horizontal and anterior canal involvement is more readily recognised with modern diagnostic techniques [11,13]. Multi-canal BPPV occurs in approximately 5-10% of cases, often after head trauma or in older patients [11].

II. Pathophysiology of BPPV

Otoconia Lifecycle and Morphology

Otoconia (calcium carbonate crystals) are embedded in the otolithic membrane overlaying vestibular sensory hair cells [14]. They serve a critical biomechanical role: their mass and density allow detection of linear acceleration and head tilt by deflecting hair cell stereocilia.

Otoconia have a finite lifespan; they are continuously produced within the saccule and utricle and normally confined within the otolithic organs [14]. However, with ageing, trauma, or inner ear pathology, otoconia may dislodge and migrate into the semicircular canals, causing BPPV [13,14].

Histological studies reveal otoconia are roughly 3-30 micrometers in size, composed of calcium carbonate in a matrix protein [14]. Individual particles can clump together to form larger, more clinically relevant masses; aggregated otoconia have greater inertia and produce more sustained nystagmus.

 Key Point: *Otoconia dislodgement is usually a consequence of age, head trauma, or prior vestibular inflammation. The triggering event may occur days to weeks before symptom onset.*

Canalithiasis vs Cupulolithiasis

Canalithiasis occurs when otoconia migrate freely within the semicircular canal endolymph [13,14]. As head position changes, gravity causes particles to migrate, deflecting the cupula and triggering nystagmus and vertigo. This is the most common BPPV mechanism (canalithiasis variant).

Cupulolithiasis occurs when otoconia adhere directly to the cupula (the gelatinous structure overlying hair cells within the ampulla) [14,19]. This creates persistent cupular deflection regardless of head position, producing more sustained, less fatiguing nystagmus. Cupulolithiasis represents an important alternative pathophysiology, particularly for apogeotropic horizontal canal BPPV [19].

The distinction between canalithiasis and cupulolithiasis has practical implications: canalithiasis typically shows transient nystagmus that fatigues with repeated provocation, whilst cupulolithiasis shows persistent nystagmus that may not fatigue [13,14]. Treatment approaches may differ between these mechanisms.

 Clinical Insight: Canalithiasis is thought to be more common than cupulolithiasis based on the

nystagmus characteristics observed during diagnostic tests.

Particle Mechanics and Movement Within Canals

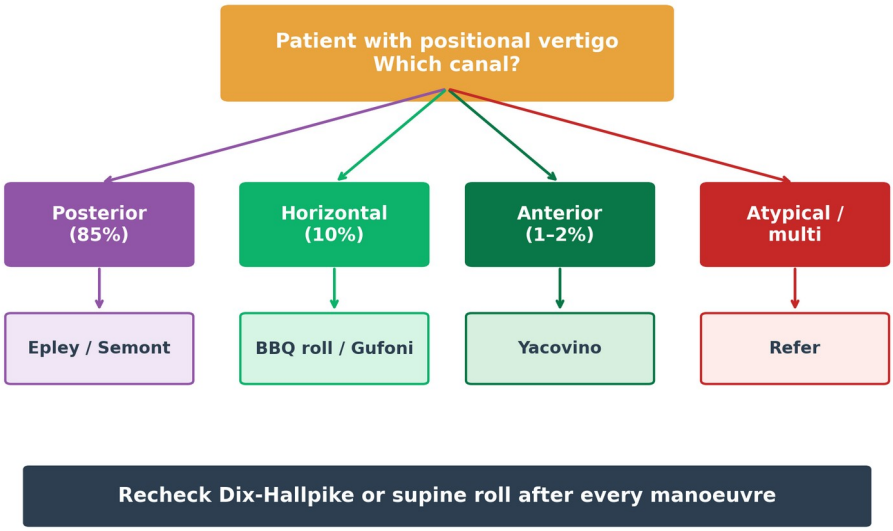
Within a semicircular canal, particles are acted upon by gravitational force and endolymphatic drag [14]. The balance between these forces determines particle movement velocity. As particles move through the canal, they create endolymphatic flow that deflects the cupula and triggers vestibular afferent firing.

The velocity of particle movement depends on particle size and density [14]. Larger particles move faster; smaller particles move slower. Particle clumping changes effective mass and consequently movement characteristics.

Particle redistribution occurs during repositioning manoeuvres as the canal orientation is systematically changed [2,3]. The goal of most manoeuvres is to migrate particles from the affected canal back into the utricle, where they are absorbed by macular epithelium and degraded.

 Key Point: *Successful repositioning requires gravity-dependent particle movement: particles must move to canal locations where gravity naturally migrates them toward exit points. Manoeuvre effectiveness depends on positioning the canal to facilitate this natural movement.*

Figure 1. BPPV Canal Distribution and Decision Diamond.



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III. Posterior Canal BPPV

Dix-Hallpike Manoeuvre: Technique and Interpretation

The Dix-Hallpike test is the gold standard diagnostic manoeuvre for posterior canal BPPV [8,15]. Patient starts sitting, clinician positions patient supine with head turned 45° toward affected ear and extended 20-30° below horizontal. Manoeuvre lasts 30-60 seconds with eyes open observing for nystagmus.

In posterior canal BPPV, this manoeuvre produces: (1) latency of 3-10 seconds before nystagmus onset, (2) characteristic up-beating (superior) nystagmus combined with torsional component beating toward the affected ear, (3) duration <1 minute typically, and (4) reversal of nystagmus direction when patient

sits back up [8,13]. Nystagmus fatigues with repeated provocation, distinguishing canalithiasis from central pathology [13].

Positive Dix-Hallpike test has sensitivity 80-90% and specificity >95% for posterior canal BPPV [15]. False positives can occur in anterior canal BPPV (though rarely) and in central pathology mimicking BPPV; central nystagmus typically lacks the latency, fatiguability, and characteristic torsional pattern of peripheral BPPV [16].

 Clinical Pearl: The presence of latency, fatiguing, and direction-appropriate nystagmus confirms posterior canal BPPV. The cardinal feature is the delayed onset; immediate vertigo without nystagmus suggests anxiety rather than true BPPV.

Epley Manoeuvre: Step-by-Step Technique

The Epley manoeuvre (canalith repositioning procedure) is the gold-standard treatment for posterior canal BPPV, achieving resolution in 80-90% of cases after 1-2 attempts [2,20]. The technique uses sequential head positions to roll otoconia particles from the posterior canal back into the utricle.

Technique: (1) Patient supine with head hanging, head rotated 45° toward affected ear (same position as Dix-Hallpike), (2) slowly rotate head 90° toward unaffected side (now facing away from affected ear), (3) roll patient onto unaffected shoulder whilst maintaining head rotation (head facing 135° from initial position), (4) sit patient back up with head still rotated [2]. Hold each position 30-60 seconds.

The mechanism: starting position (supine head-hanging) encourages particles into posterior canal. Progressive rotation and rolling moves particles from canal toward ampulla, then from ampulla toward common crus and ultimately back into utricle [2]. Each position change creates an endolymphatic current that helps move otoconia through the canal.

 Key Point: *The Epley is highly effective but technique precision matters: inadequate head extension, too-rapid movement, or deviation from prescribed positioning reduces efficacy. Clear communication and demonstration are essential.*

Semont Manoeuvre (Liberatory Manoeuvre)

The Semont manoeuvre offers an alternative to the Epley, achieving similar success rates (75-85%) [3,20]. It differs in mechanism: rather than rolling particles through canal positions, the Semont uses rapid lateral head movements to dislodge particles from canal walls.

Technique: (1) Patient sitting, head rotated 45° toward affected ear, (2) rapidly recline to supine (affected ear toward bed), hold 30 seconds, (3) rapidly transfer through sitting to supine on opposite side (unaffected ear toward bed) maintaining head rotation, hold 30 seconds, (4) return to sitting [3].

The Semont manoeuvre may be preferred in patients with cervical spine limitations, as it involves less head-hanging than Epley [3]. Some patients find Semont more uncomfortable due to rapid movement, though success rates are comparable.

 Clinical Insight: Both Epley and Semont are effective; choice depends on patient tolerance, cervical spine status, and clinician experience. Both should be in the competent physiotherapist's toolkit.

IV. Horizontal Canal Geotropic BPPV

Supine Roll Test: Diagnostic Approach

The supine roll test is performed with patient supine [11]. The head is then rapidly rotated side-to-side (ear-to-ear rolling) whilst eyes remain looking forward. Nystagmus direction and intensity are observed during each side-roll.

Characteristic features: (1) horizontal nystagmus beating toward affected (undermost) ear, (2) nystagmus present during roll test but direction reverses with head rotation [11,13]. Intensity may differ between sides, suggesting unilateral involvement; symmetric nystagmus suggests cupulolithiasis or bilateral pathology [11].

Geotropic variant represents the most common horizontal canal form. It often responds to repositioning manoeuvres with higher success rates (85-95%) compared to apogeotropic variants [4,5].

 Clinical Pearl: The geotropic pattern (nystagmus beating toward undermost ear) is gravity-dependent: particles are presumed to be free-floating in the canal (canalithiasis), not attached to cupula.

BBQ (Barbecue) Roll Manoeuvre

The BBQ (Barbeque) roll manoeuvre, also called the "rotation therapy," treats geotropic horizontal canal BPPV by rolling the patient in the direction that moves particles back toward the utricle [5]. The procedure is performed slowly to allow gravity-mediated particle migration.

Technique: (1) Patient supine, head neutral, (2) slowly roll patient 360° toward affected side (rolling continuously in one direction) at approximately 90° increments, holding each position 30 seconds [5]. Total time: approximately 4-6 minutes per cycle.

Mechanism: rolling toward affected ear encourages particles to migrate from canal back toward utricle through a gravity-dependent process [5]. The slow, continuous movement is essential — rapid rolling fails to allow particle redistribution.

 Key Point: *BBQ success rates for geotropic horizontal BPPV are 85-95% with single application, higher than for posterior canal variants. Some patients require 2-3 applications.*

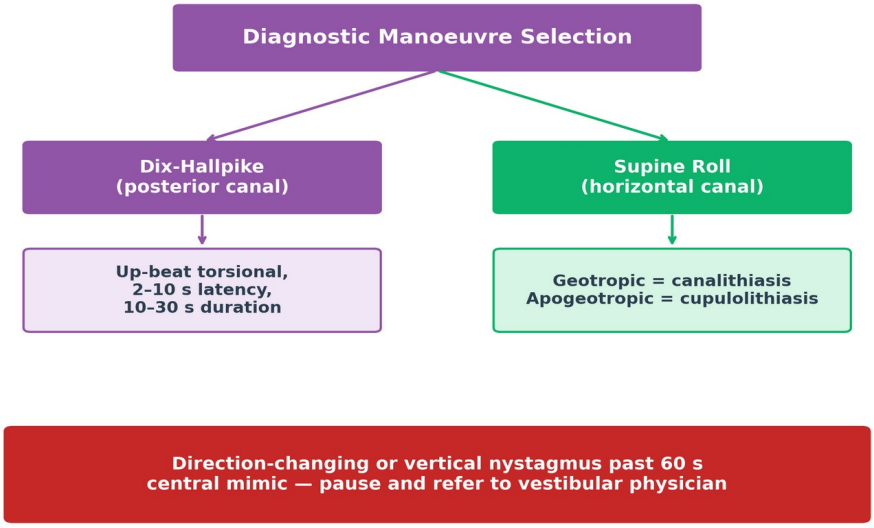
Gufoni Manoeuvre for Horizontal BPPV

The Gufoni manoeuvre offers an alternative for horizontal canal BPPV: (1) patient sitting, (2) rapidly recline to side-lying on affected ear (30-45 degrees), hold 30 seconds, (3) rapidly rotate head 45 degrees toward floor whilst maintaining side-lying, hold 30 seconds, (4) return to sitting [4].

The Gufoni manoeuvre may be more convenient in patients who cannot tolerate prolonged rolling. Success rates are comparable to BBQ (80-90%) [4].

 Clinical Insight: Choice between BBQ and Gufoni depends on patient tolerance and anatomical considerations (cervical spine limitations). Both are effective for geotropic horizontal BPPV.

Figure 2. Diagnostic Manoeuvre Selection.



Source: Australian Dizziness Clinics, 2026.

V. Horizontal Canal Apogeotropic BPPV

Identifying Apogeotropic Direction-Changing Nystagmus

Apogeotropic horizontal canal BPPV is less common but often more challenging to treat [11,18]. During supine roll test, nystagmus beats away from undermost ear (apogeotropic = away from earth).

Apogeotropic BPPV is thought to represent cupulolithiasis rather than canalithiasis: particles adhere to cupula rather than floating free [19]. The direction-changing nystagmus in apogeotropic variant has unique characteristics that distinguish it from geotropic horizontal canal BPPV.

Apogeotropic variant is associated with lower spontaneous resolution rates and higher recurrence rates compared to geotropic [18,19]. Treatment often requires modification of standard repositioning approaches.

 Key Point: *The direction-changing nystagmus pattern (beating away from undermost ear) indicates apogeotropic horizontal BPPV. This variant often responds less completely to standard manoeuvres.*

Modified Treatment Approaches for Apogeotropic BPPV

Standard BBQ rolling may be ineffective for apogeotropic BPPV [18]. Alternative approaches include: (1) rotating patient in direction opposite to standard BBQ, (2) using rapid side-lying manoeuvres to detach cupular particles, (3) employing the Casani-modified Semont, or (4) the head-down rolling manoeuvre [18].

Some cases of apogeotropic BPPV spontaneously convert to geotropic pattern after 1-2 weeks, suggesting particle migration or detachment from cupula [18]. Repeat assessment after 1-2 weeks is appropriate before declaring treatment failure.

 Clinical Insight: Apogeotropic BPPV requires individualised approach and clinical flexibility. Patient communication about realistic treatment expectations is important; multiple sessions may be needed.

VI. Anterior Canal BPPV

Deep Head-Hanging Positioning

Anterior canal BPPV is rare (2-10% of BPPV cases) and often missed diagnostically because the characteristic nystagmus (down-beating with slight clockwise torsional component for left anterior canal) is more subtle than posterior canal [11,13].

Anterior canal BPPV is suspected when down-beating nystagmus is provoked by head extension or when Dix-Hallpike provokes down-beating (rather than up-beating) [13]. The anterior canal is anatomically positioned such that head-hanging Dix-Hallpike can affect it; differential diagnosis from posterior canal requires careful nystagmus interpretation.

Anterior canal BPPV is often confused with posterior canal BPPV if the examiner misidentifies the nystagmus direction (confusing torsional component with rotational direction) [15]. Down-beating nystagmus, however, must always raise concern for central pathology and warrant neuroimaging review [7,16].

 Clinical Pearl: Anterior canal BPPV is uncommon enough that when suspected, careful examination and possibly video-nystagmography or VNG is warranted to confirm the diagnosis before pursuing treatment.

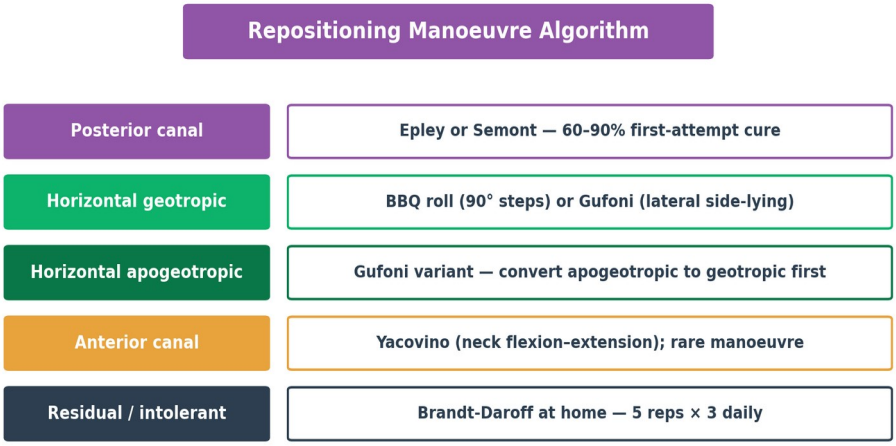
Reverse Epley Manoeuvre for Anterior Canal

Treatment for confirmed anterior canal BPPV involves reverse positioning compared to posterior canal: (1) patient supine with head extended (opposite of Epley starting position), head rotated 45° away from affected ear, (2) head returned to neutral whilst maintaining supine, (3) sit patient up with head rotated [2]. The mechanism: rolling particles from anterior canal back through ampulla into utricle.

Success rates for anterior canal manoeuvres are lower than posterior canal (60-75%), reflecting the rarity and diagnostic difficulty of anterior canal BPPV [13,20]. Recurrence rates may also be higher in anterior canal cases.

 Key Point: *Anterior canal BPPV requires correct diagnosis of nystagmus direction (down-beating with clockwise torsion when viewed from above). Misdiagnosis as posterior canal leads to treatment failure.*

Figure 3. Repositioning Manoeuvre Algorithm.



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VII. Multi-Canal BPPV

Simultaneous Multi-Canal Involvement

Multi-canal BPPV occurs in approximately 5-10% of cases: particles affect multiple semicircular canals simultaneously [11]. This may result from significant head trauma, severe vestibular pathology, or the natural progression of disease.

Diagnosis is suggested when multiple diagnostic tests show abnormal nystagmus (e.g., positive Dix-Hallpike plus abnormal supine roll test) [11,13]. Treatment requires sequential manoeuvres: address each affected canal separately, prioritising the more symptomatic or most amenable to repositioning.

Example: patient with posterior and horizontal canal involvement would receive Epley manoeuvre first, then re-evaluate supine roll test. If horizontal canal involvement persists, perform BBQ roll on a subsequent visit, allowing 24-48 hours between manoeuvres [2,5].

 Key Point: *Multi-canal BPPV is not simultaneously treated; rather, manoeuvres are sequentially applied, with re-testing between manoeuvres to confirm resolution of each canal's involvement.*

VIII. Post-Manoeuvre Management: Evidence on Restrictions

Activity and Positional Restrictions

Historically, patients were restricted to "head up" positioning (30° elevation) for 24-48 hours after repositioning manoeuvres, with advice to avoid bending forward, sleeping on the affected side, and certain head positions [6]. This was based on theoretical concerns about particle reflux into the canal.

A 2020 systematic review by Gans found minimal evidence supporting post-manoeuvre activity restrictions [15]. Randomised trials comparing restriction to no-restriction protocols found similar outcomes.

Current recommendations favour minimal restrictions: patients should avoid activities that provoke severe vertigo but need not maintain strict positional limitations [15,20]. The duration of restrictions, if used, can be reduced to 24 hours rather than 48-72 hours.

Table 1. Post-Manoeuvre Activity Recommendations

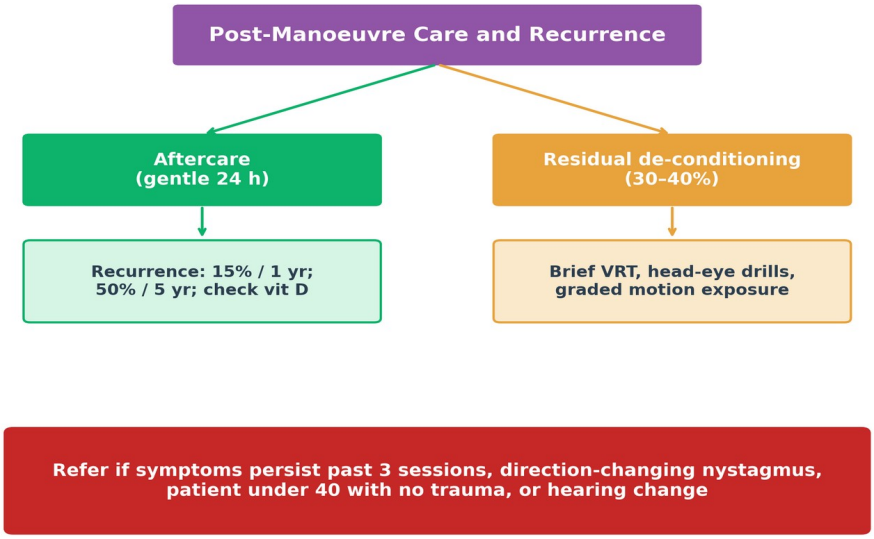
Activity Level	Recommendation	Evidence	Success Impact
No restriction	Continue normal activities	Similar to restriction	Minimal
Mild restriction	Avoid troublesome positions only	Comparable success	None to minimal
Strict restriction	30° elevation, avoid bending	No additional benefit	None

Evidence does not support strict post-manoeuvre restrictions for improving outcomes

Patient counselling should clarify that manoeuvre effectiveness does not depend on post-treatment activity modification [6,15]. This reduces anxiety and improves quality of life during the recovery period.

 Clinical Insight: Practical advice: avoid movements that are known to provoke symptoms, but routine activities, exercising, and even head motion are not contraindicated post-manoeuvre.

Figure 4. Post-Manoeuvre Care and Recurrence.



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IX. Treatment-Resistant BPPV

Diagnostic Uncertainty and Reassessment

Treatment-resistant BPPV (failure to achieve resolution after 2-3 appropriately performed manoeuvres) occurs in 10-15% of cases [13,20]. Common causes include: (1) misdiagnosis (alternative pathology mimicking BPPV), (2) incorrectly identified affected canal, (3) cupulolithiasis variant, (4) multi-canal involvement, or (5) underlying vestibular pathology [13].

Reassessment involves: thorough re-examination for alternative diagnoses, careful nystagmus re-assessment in multiple head positions (to identify missed canal involvement), consideration of vestibular neuritis, central vestibular pathology, or vestibular migraine [9,10,16,17]. The Halmagyi head impulse test and vHIT may help differentiate peripheral from central pathology [9,10].

 Clinical Pearl: Treatment failure should prompt diagnostic reassessment rather than repetition of the same manoeuvre. Careful physical examination often identifies missed canal involvement or different diagnosis.

Conversion and Recurrence Patterns

Some patients with initially posterior canal BPPV convert to horizontal canal BPPV after treatment [18]. This may reflect particle fragmentation or migration during the manoeuvre itself, or natural progression of disease.

Recurrence of BPPV is common: approximately 30-50% of patients experience symptom recurrence within 5 years [1,13]. Recurrence may represent same canal relapse, conversion to different canal, or new BPPV onset in contralateral ear.

 Key Point: *Recurrent BPPV is treated the same way as initial presentation: diagnostic testing followed by appropriate repositioning manoeuvre(s). Recurrence does not indicate a chronic disease requiring ongoing management.*

X. Outcome Data and Recurrence Rates

Table 2. BPPV Treatment Success Rates and Outcomes

Variant	Manoeuvre	1st Success	After 2-3 Attempts	Recurrence (5yr)
Posterior	Epley	85-90%	95-99%	30-40%
Posterior	Semont	75-85%	95-98%	28-38%
Horizontal geo.	BBQ	85-95%	98-99%	35-45%
Horizontal apo.	Variable	60-75%	85-95%	50-65%
Anterior	Reverse Epley	60-75%	80-90%	40-50%

Success and recurrence rates vary by variant and technique

Overall, BPPV has one of the best prognoses among vestibular disorders: >95% of patients achieve complete symptom resolution with 2-3 appropriately performed manoeuvres [20]. Resolution rates remain high even in older patients and those with comorbid conditions.

Despite high treatment efficacy, BPPV continues to be undertreated globally, with many patients receiving vestibular suppressants or continuing to suffer with untreated symptoms [12,13]. This may reflect inadequate clinician training in repositioning manoeuvres or systematic barriers to specialist referral.

XI. Conclusions

Benign paroxysmal positional vertigo remains the most common cause of vertigo and is highly treatable through mechanism-based repositioning manoeuvres [1,13,20]. Each canal variant has specific diagnostic features and tailored treatment approaches that should be matched to individual presentations.

Diagnostic accuracy requires careful physical examination with attention to nystagmus latency, direction, and fatiguing pattern [8,13,15]. Multi-canal involvement, apogeotropic variants, and treatment resistance require additional clinical reasoning and adapted protocols [11,18,19].

Post-manoeuvre activity restrictions have minimal evidence support and should be minimised in favour of practical activity guidance [15]. Patient counselling about high cure rates and likely recurrence improves long-term symptom management and quality of life.

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