

BPPV in the Emergency Department: Rapid Diagnosis and Treatment

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How to Use This Review

This literature review provides emergency medicine clinicians with evidence-based, practical clinical knowledge for rapidly diagnosing and managing benign paroxysmal positional vertigo (BPPV) in the acute care setting.

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.

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I. Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disorder, accounting for approximately 20-30% of all dizzy presentations to the emergency department. Despite its high prevalence, BPPV remains frequently misdiagnosed in acute care settings [1,15].

The hallmark of BPPV is brief episodes of severe vertigo triggered by specific head position changes. Understanding BPPV pathophysiology and mastering rapid, targeted diagnostic techniques are essential skills for emergency medicine clinicians [4].

BPPV arises from displacement of otoconia within the semicircular canals, creating abnormal endolymphatic flow. Fortunately, BPPV is highly treatable through simple positional manoeuvres that can be performed in the ED [4,16].

□ **Key Point:** BPPV accounts for ~20% of ED dizzy presentations. It is the most common vestibular disorder and is highly treatable with bedside repositioning manoeuvres.

Clinical Significance in the Emergency Department

BPPV presentation in the ED often causes significant patient anxiety. However, distinctive features allow rapid bedside differentiation from central causes: [2,15]

- **Latency:** Nystagmus appears 5-20 seconds after provocative head movement
- **Fatiguability:** Repeated testing produces progressively weaker nystagmus
- **Duration:** Episodes are brief, typically <1 minute
- **Rotation:** Nystagmus is typically upbeat with rotatory component

In-ED repositioning manoeuvres have success rates >80% for posterior canal BPPV, often providing complete symptom relief within a single ED visit [3,5] [6].

□ **Clinical Pearl:** If nystagmus has latency (>5 seconds onset), fatigues with repeated testing, and resolves within 1 minute, BPPV is confirmed and imaging is not indicated.

Epidemiology and Prevalence

BPPV is the most common cause of peripheral vestibular vertigo. Lifetime prevalence estimates range from 1.6% to 2.4% in the general population, but in the ED setting, prevalence among all dizzy presentations is 20-30% [1,15].

Age is the strongest demographic risk factor, with incidence increasing markedly after age 60. Women are affected 1.5-2 times more frequently than men. Idiopathic BPPV accounts for approximately 50% of cases [1,12].

The ED is a critical point in the BPPV care pathway. Early, accurate diagnosis and in-visit treatment reduce diagnostic uncertainty and unnecessary advanced imaging [2].

□ **Clinical Insight:** In a cohort of ED patients with dizziness, ~1 in 5 will have BPPV. Bedside diagnostic testing takes <2 minutes and can confidently establish the diagnosis without imaging.

II. Pathophysiology

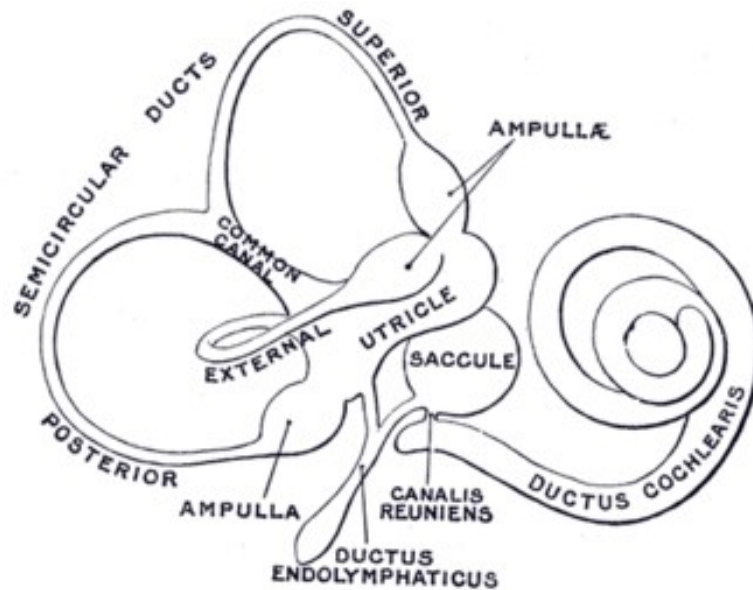


Figure A. Membranous labyrinth — three semicircular ducts, ampullae, utricle, and saccule. Otoconia dislodged from the utricular macula migrate into the canals and cause BPPV.

Source: Wikimedia Commons

BPPV results from displacement of otoconia—small crystals of calcium carbonate and protein embedded within the utricular membrane of the inner ear. When otoconia become detached and fall into the semicircular canals, they abnormally stimulate the vestibular sensory epithelium (crista ampullaris), triggering false signals of rotation [10,16].

The mechanical consequence depends on the relationship between the otoconia and canal anatomy. In canalithiasis, free-floating otoconia move through the canal fluid (endolymph) in response to head acceleration. In cupulolithiasis (less common), otoconia adhere directly to the cupula [4,16].

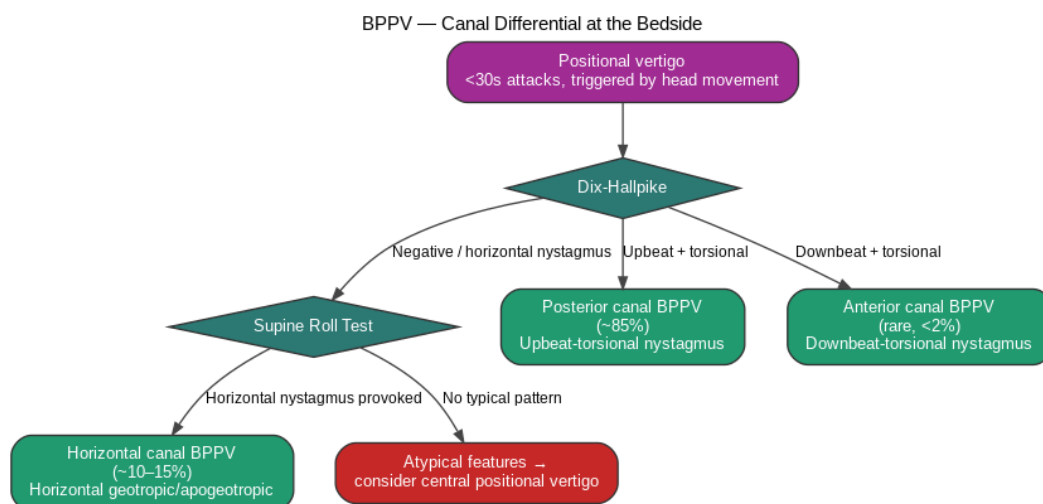


Figure 1. Bedside canal-differential pathway for positional vertigo.

Canalithiasis vs Cupulolithiasis

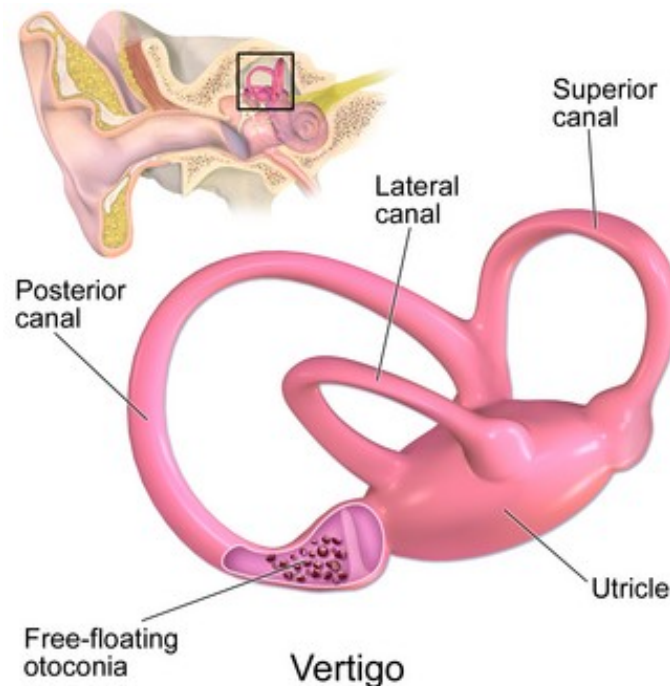


Figure B. Three-dimensional view of the semicircular canals (superior, lateral, posterior) with free-floating otoconia shown in the posterior canal — the canalithiasis mechanism underlying BPPV.

Source: Wikimedia Commons

Canalithiasis is the predominant mechanism in BPPV. Free-floating otoconia within a canal move passively with the endolymph in response to head acceleration. This creates a bolus of high-density particles that stimulates the cupula, generating intense, transient nystagmus with characteristic latency and fatigue [4,13].

Cupulolithiasis occurs when otoconia stick to the cupula itself, increasing its density and altering its mechanical properties. Cupulolithiasis typically produces longer-duration vertigo and nystagmus (up to several minutes) with less pronounced fatigue. It is less common and more difficult to treat with standard manoeuvres [10,16].

□ **Key Point:** Canalithiasis (free particles) accounts for >90% of BPPV and produces brief vertigo with marked fatigue. Cupulolithiasis (particle adhesion) is rarer and produces more prolonged symptoms.

Otoconia and Semicircular Canal Mechanics

The utricle contains a sensory epithelium covered with the utricular membrane—a gelatinous structure embedded with otoconia. These crystals detect linear head acceleration and gravity. Disruption of the utricle can cause otoconia to fragment and dislodge [16].

Free fragments then migrate into the semicircular canals—typically the posterior canal due to anatomical susceptibility. Within a canal, the otoconia respond to head acceleration, creating endolymphatic flow that stimulates the cupula abnormally [4,16] [6].

The semicircular canals are oriented in three orthogonal planes: posterior (nearly vertical), anterior (nearly vertical), and horizontal (nearly horizontal). Understanding canal anatomy is essential for predicting which movements provoke symptoms and selecting appropriate diagnostic and therapeutic manoeuvres [16].

□ **Clinical Insight:** Otoconia fragments are denser than endolymph, so they settle and move with gravity. In the posterior canal, they typically accumulate at the lowest dependent portion.

Canal Prevalence and Posterior Canal Predominance

The posterior semicircular canal is affected in 80-90% of BPPV cases. This anatomical predominance reflects the posterior canal's geometry—its long slender shape predisposes to otoconia settling and accumulation [1,4] [6].

Horizontal canal BPPV accounts for 5-15% of cases and typically presents with more persistent symptoms. Anterior canal BPPV is rare (<3%) and can be challenging to diagnose due to atypical nystagmus and symptom patterns [9,10].

Multiple canals can be simultaneously affected (mixed-canal BPPV), though this is uncommon. Recurrent or bilateral BPPV is possible but suggests a secondary cause.

Table 1. Semicircular Canal Involvement in BPPV

Canal	Prevalence	Duration	Nystagmus
Posterior	80-90%	Seconds-1 min	Upbeating, torsional
Horizontal	5-15%	Minutes-hours	Horizontal; geotropic or apogeotropic
Anterior	<3%	Seconds-minutes	Downbeating; atypical
Mixed	<5%	Variable	Mixed patterns

III. Posterior Canal BPPV

Posterior canal BPPV is the most common variant, accounting for 80-90% of all BPPV cases. The diagnosis is confirmed by the Dix-Hallpike manoeuvre, which reliably provokes the characteristic nystagmus [2,4] [3].

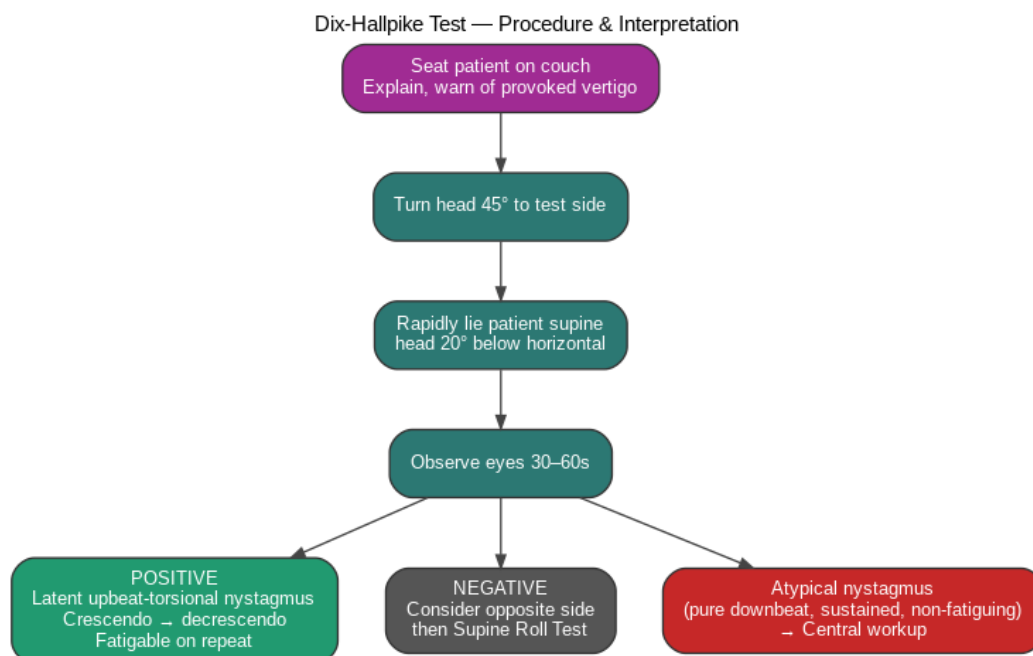


Figure 2. Dix-Hallpike test — procedure and interpretation [3].

The Dix-Hallpike Testing Technique

The Dix-Hallpike manoeuvre is the gold-standard bedside test for posterior canal BPPV. It is simple to perform, takes <30 seconds, and reliably provokes symptoms if BPPV is present [2,5] [3].

- **Position:** Patient sits upright on examination table
- **Grasp:** Clinician stabilizes patient's head with both hands
- **Turn:** Rotate patient's head 45 degrees toward one shoulder
- **Lower:** Quickly lower patient backward until head hangs below horizontal
- **Observe:** Watch the eyes carefully for nystagmus onset (typically 5-20 seconds)
- **Hold:** Maintain position for up to 60 seconds
- **Return:** Slowly return patient to upright
- **Repeat:** Test the opposite side to determine laterality

The Dix-Hallpike should be performed cautiously in patients with contraindications (severe cervical spondylosis, recent stroke, or hemodynamic instability) [2] [3].

□ **Key Point:** *Dix-Hallpike should be performed slowly and deliberately. Smooth positioning allows true BPPV nystagmus to manifest clearly.*

Expected Nystagmus Patterns

If otoconia are present in the posterior canal, the Dix-Hallpike provokes a characteristic nystagmus pattern with three key features: upbeating component with a rotatory component, latency before onset, and fatigability with repetition [2,4] [3].

The primary nystagmus is upbeating combined with a torsional component. The torsion is typically in the plane of the affected canal—the superior pole of the eye on the side of the affected canal rotates toward the affected ear [2,4].

The nystagmus does not begin immediately. Instead, there is a latency period of typically 5-20 seconds before nystagmus onset. This latency is crucial for diagnosis: it distinguishes BPPV from central positional nystagmus [2,10].

When the same manoeuvre is repeated immediately after the first test, the nystagmus becomes progressively weaker with each repetition. By the third or fourth repetition, the nystagmus may disappear entirely [2,4].

In posterior canal BPPV, the total nystagmus duration is typically <1 minute. If nystagmus persists for >2 minutes, consider alternative diagnoses [2] [6].

□ **Clinical Insight:** The combination of latency + fatigue + upbeating-torsional pattern + duration <1 minute is pathognomonic for posterior canal BPPV.

The Epley Canalith Repositioning Manoeuvre

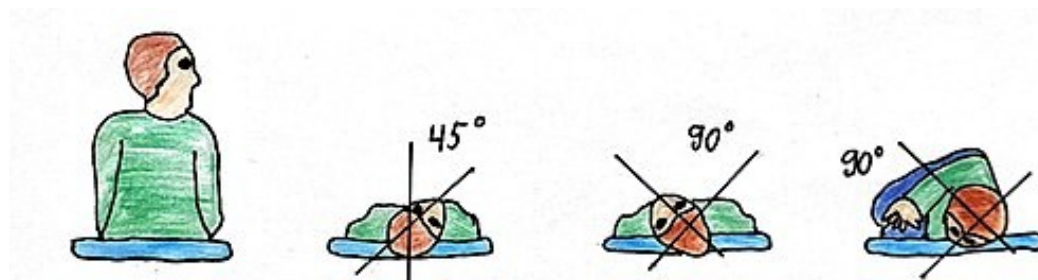


Figure C. The Epley canalith repositioning manoeuvre — sequential head positions (upright → 45° extension → 90° contralateral roll → further 90° forward roll) that guide otoconia back into the utricle.

Source: Wikimedia Commons

The Epley canalith repositioning manoeuvre (CRM) is the standard first-line treatment for posterior canal BPPV. It has efficacy rates >80% and takes approximately 5-10 minutes [3,5,6] [6].

The Epley manoeuvre is performed in a series of five sequential head positions. At each position, the clinician allows time for the otoconia to move through the endolymph [6] [5].

- **Position 1:** Perform Dix-Hallpike position for 30 seconds [3]
- **Position 2:** Rotate head 90 degrees toward opposite shoulder for 30 seconds
- **Position 3:** Roll body until fully supine while maintaining head rotation for 30 seconds
- **Position 4:** Sit up slowly with head moving from extension to flexion for 30 seconds

After the manoeuvre, some sources recommend keeping the head upright for 24 hours. However, recent evidence suggests these restrictions provide minimal benefit [3,5].

Epley Canalith Repositioning Manoeuvre — Posterior Canal

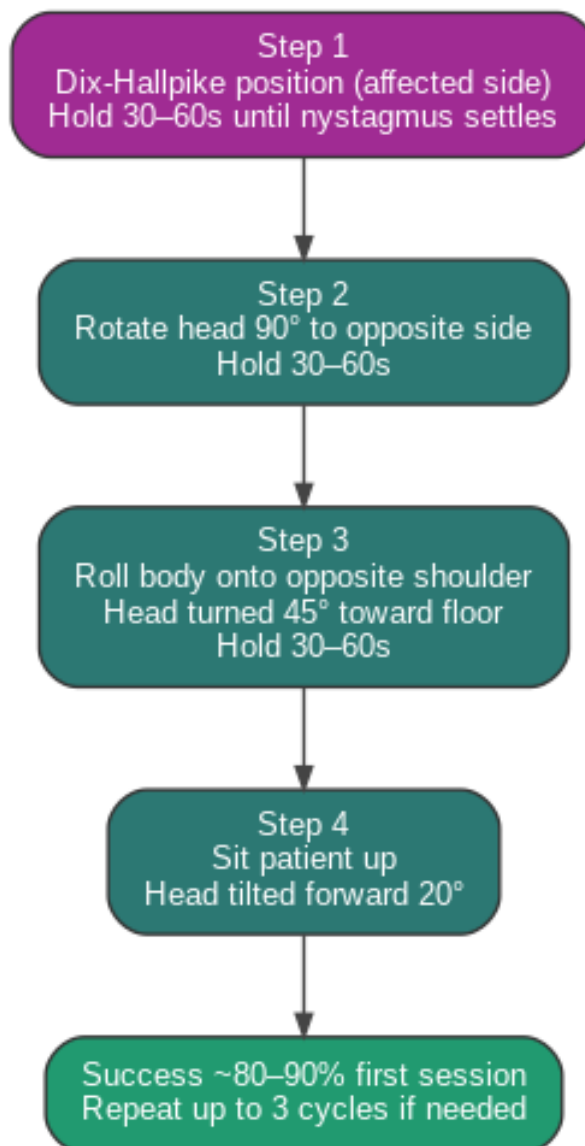


Figure 3. Epley canalith repositioning manoeuvre — four-step sequence.

□ **Clinical Pearl:** Epley CRM is simple enough to teach to patients for home use. Self-administered Epley manoeuvres achieve similar efficacy and can be repeated daily if symptoms persist.

The Semont Manoeuvre as Alternative

The Semont manoeuvre is an alternative repositioning technique with similar efficacy to the Epley (70-80% success rate). It uses rapid lateral head movements and rapid positional changes to dislodge particles [7].

Semont can be used if Epley is contraindicated (severe cervical spondylosis or limited neck motion) or if Epley fails after initial attempts [7].

□ **Key Point:** Both Epley and Semont manoeuvres are effective with >70% success rates. Epley is preferred first-line.

Success Rates and Treatment Outcomes

Immediate success following a single in-ED Epley manoeuvre occurs in 50-80% of posterior canal BPPV patients. An additional 10-20% experience significant improvement [3,5,6] [5].

Failed manoeuvres may reflect: cupulolithiasis rather than canalolithiasis, incorrect diagnosis, improper technique, or patient anxiety. Repeating the manoeuvre a second time in the ED increases success rates to >80% [3,10].

Patients with failed ED treatment should be referred for specialist vestibular assessment. Home-based Epley repetition often achieves resolution within days if the initial ED attempt was unsuccessful [2].

□ **Clinical Insight:** Even if a single in-ED manoeuvre does not provide complete symptom resolution, most patients improve significantly and can be discharged with home manoeuvre instructions.

IV. Horizontal Canal BPPV

Horizontal canal BPPV accounts for 5-15% of BPPV presentations and typically produces more persistent vertigo (minutes to hours) compared to posterior canal BPPV [9,10] [6].

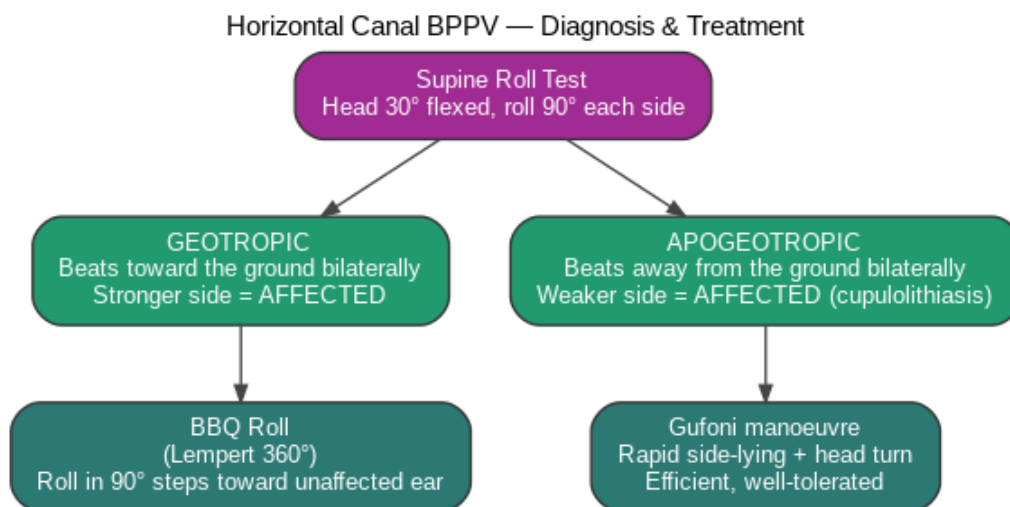


Figure 4. Horizontal canal BPPV — geotropic vs apogeotropic differentiation and treatment.

The Supine Roll Test

The supine roll test is the primary diagnostic manoeuvre for horizontal canal BPPV. The patient begins supine, and the head is rotated 90 degrees from center toward one ear, then toward the other [9,10] [7].

- **Position:** Patient lies flat supine on examination table with head in neutral
- **Rotate right:** Rotate head 90 degrees to the right for 30-60 seconds
- **Return:** Return head to center
- **Rotate left:** Rotate head 90 degrees to the left for 30-60 seconds

Nystagmus from horizontal canal BPPV appears with shorter latency and longer duration compared to posterior canal BPPV [9,10] [6].

□ **Key Point:** *Horizontal canal BPPV should be suspected if supine roll testing provokes horizontal nystagmus or if a patient with posterior canal diagnosis has persistent symptoms after successful Epley treatment.*

Nystagmus Patterns: Geotropic vs Apogeotropic

Horizontal canal BPPV produces two distinct nystagmus patterns depending on particle location [9,10].

The nystagmus beats toward the ground (affected ear). When head is rolled right with right horizontal canal affected, nystagmus beats rightward. This pattern is consistent with free-floating particles (canalithiasis) and has better treatment prognosis [9] [7].

The nystagmus beats away from the ground (opposite affected ear). This pattern is consistent with particle adherence near the cupula (cupulolithiasis) and has more variable treatment response [10].

Table 2. Geotropic vs Apogeotropic Horizontal Canal BPPV

Pattern	Mechanism	Nystagmus	Treatment
Geotropic	Free particles in lateral canal	Beats toward ground	Good; BBQ roll
Apogeotropic	Particles near ampulla	Beats away from ground	Variable; Gufoni

BBQ Roll Manoeuvre

The BBQ (360-degree) roll manoeuvre is the first-line treatment for geotropic horizontal canal BPPV. The patient rolls 360 degrees supine in the direction of the nystagmus [10,16] [7].

- **Start:** Patient supine with head rotated toward affected side
- **Roll:** Roll patient continuously 360 degrees in direction matching nystagmus
- **Target:** Complete full 360-degree rotation
- **Repeat:** May be repeated 1-2 more times

Success rates for the BBQ manoeuvre in geotropic horizontal canal BPPV are approximately 70-80% after 1-2 repetitions [10] [7].

□ **Clinical Insight:** The "BBQ" name derives from the 360-degree rotation resembling rotating meat on a grill. It is mechanically simple and can be performed by patients at home.

Gufoni Manoeuvre

The Gufoni manoeuvre is used for apogeotropic horizontal canal BPPV, where particles are thought to be lodged near the ampulla or adhered to the cupula [8,9] [7].

- Horizontal nystagmus beats away from the ground (opposite affected side)
- BBQ manoeuvre failed to improve symptoms
- Symptoms more persistent than typical geotropic BPPV

Success rates are variable (50-70%), and some cases require repeated manoeuvres or vestibular medication.

□ **Key Point:** *Geotropic horizontal BPPV → BBQ roll. Apogeotropic horizontal BPPV → Gufoni manoeuvre or medication if manoeuvre fails.*

V. Anterior Canal and Atypical Variants

Anterior canal BPPV is rare, accounting for <3% of BPPV cases. It is diagnostically challenging because nystagmus patterns are atypical [4,19].

Epidemiology and Diagnosis

Anterior canal BPPV is uncommon but may be underdiagnosed due to unfamiliarity with its nystagmus patterns. It can occur in isolation or with posterior canal BPPV [19] [6].

The deep head-hanging manoeuvre or supine-head-hanging position can provoke anterior canal BPPV.

Nystagmus Characteristics

Anterior canal BPPV produces a downbeating nystagmus—a vertical nystagmus that beats downward. This differs distinctly from the upbeat-torsional pattern of posterior canal BPPV [4,19] [6].

The nystagmus can be provoked by head-hanging position. The pattern typically has less obvious latency and fatigue, making it more challenging to identify as BPPV rather than central nystagmus.

□ **Clinical Insight:** *Downbeating nystagmus in a patient with positional vertigo should raise suspicion for anterior canal BPPV. Central causes of downbeating nystagmus are uncommon in acute presentations but always warrant consideration.*

The Reverse Epley Manoeuvre

The reverse Epley manoeuvre (anti-Epley) is used to treat anterior canal BPPV. It is performed in reverse sequence compared to standard Epley [19] [5].

The reverse Epley starts with the patient sitting upright and fully flexed forward (chin toward chest), then proceeds through positions moving the head progressively from flexion to extension. Success rates are variable (50-70%) [19].

□ **Key Point:** *Anterior canal BPPV is rare (<3% of cases) but should be suspected if downbeating nystagmus is observed. The reverse Epley manoeuvre is the primary treatment.*

VI. Differential Diagnosis: BPPV vs Central Positional Vertigo

A critical ED challenge is distinguishing BPPV from central positional nystagmus due to brainstem or cerebellar lesions. Central positional nystagmus can appear similar but has distinctly different characteristics [2,4].

BPPV vs Central Positional Vertigo — Red Flags

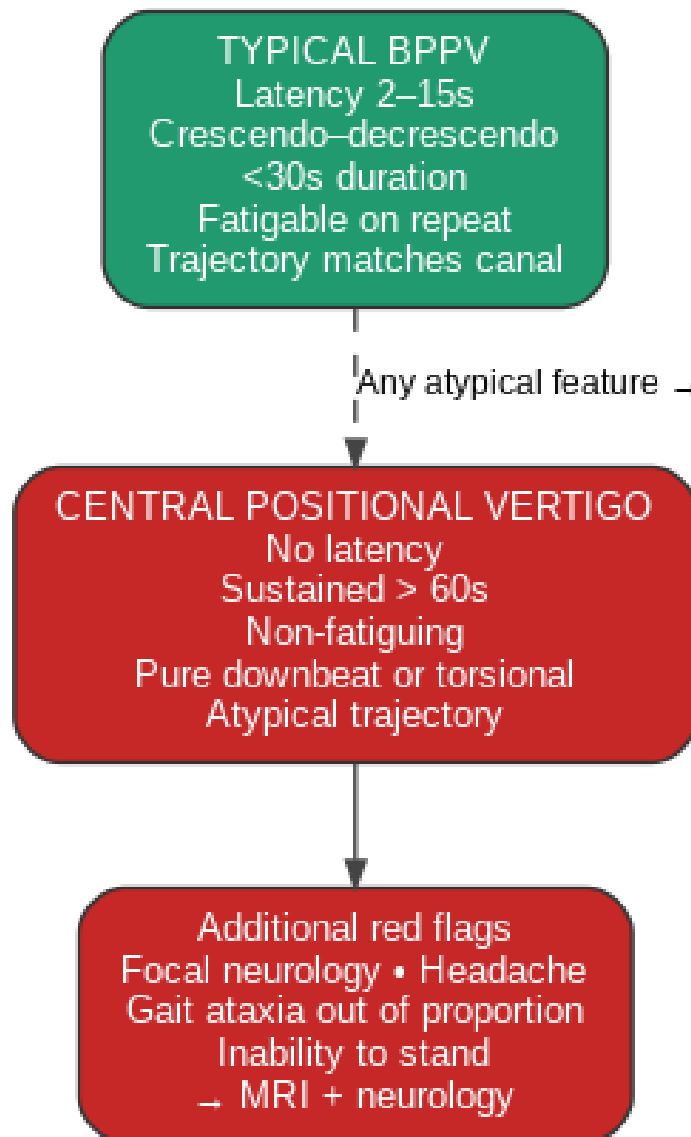


Figure 5. BPPV vs central positional vertigo — red flags.

Key Distinguishing Features

Table 3. BPPV vs Central Positional Nystagmus

Feature	Peripheral BPPV	Central Positional
Latency	Yes (5-20 sec)	No (immediate)
Fatigability	Yes (decreases)	No (persists)
Duration	<1 min typical	>1 min; often minutes
Direction	Unidirectional; rotatory	Often direction-changing
Associated signs	None; normal exam	Often present

Feature	Peripheral BPPV	Central Positional
Nystagmus type	Upbeating-torsional	Downbeating common
Imaging	Not indicated	MRI indicated

Central Positional Nystagmus Patterns

Central positional nystagmus often has different morphology. Downbeating nystagmus (vertical nystagmus beating downward) is much more common in central lesions than in peripheral BPPV [4].

Direction-changing nystagmus—where nystagmus direction changes depending on head tilt direction—is highly suspicious for central positional nystagmus and should prompt immediate MRI [4].

Cerebellar nodulus lesions are particularly associated with central positional nystagmus. Nodular lesions produce persistent, direction-changing nystagmus that does not fatigue [4].

□ **Key Point:** *Downbeating nystagmus, direction-changing patterns, or absence of fatigue should raise red flags for central positional nystagmus and warrant MRI to exclude brainstem or cerebellar stroke.*

VII. ED-Specific Management and Disposition

Once BPPV is confirmed, ED management should prioritize in-visit repositioning manoeuvres as first-line treatment. Antiemetic medications should not replace positional manoeuvres [2,5].

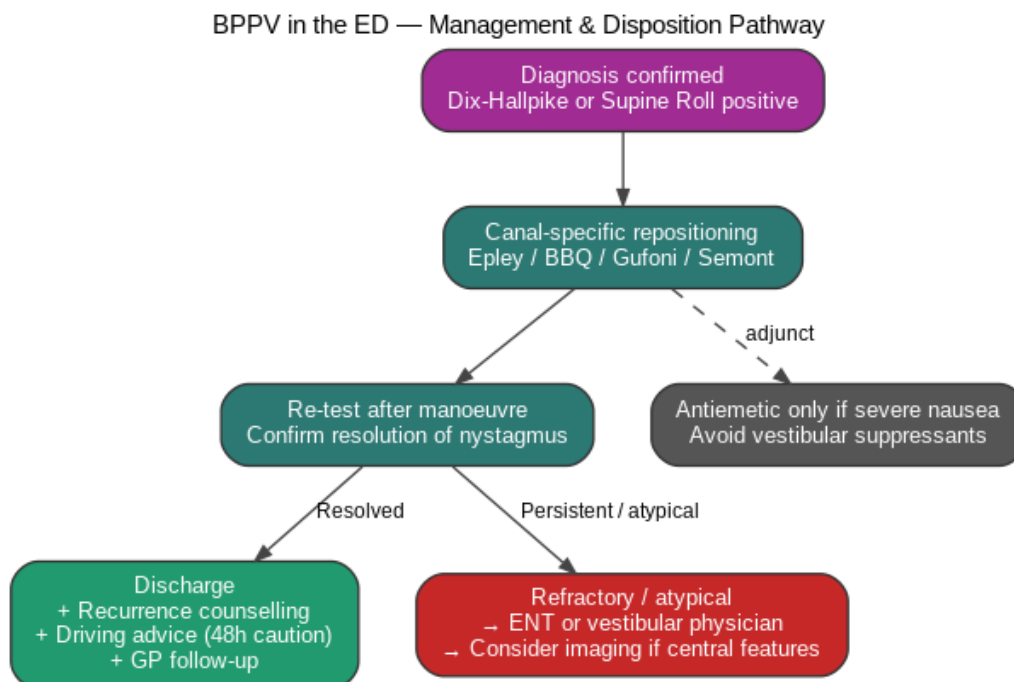


Figure 6. BPPV in the ED — management and disposition pathway.

Repositioning Manoeuvres vs Medication

Repositioning manoeuvres provide immediate, direct mechanical relief by moving otoconia out of the affected canal. Success rates are 70-80% for a single in-ED manoeuvre [3,5].

Antiemetic medications (ondansetron, metoclopramide, prochlorperazine) suppress nausea but do not treat underlying BPPV pathology. They do not move otoconia out of the canal [5].

Clinical trials consistently show:

- Repositioning manoeuvres are superior to medication alone
- Combining manoeuvre + medication provides modest additional benefit
- In-ED repositioning reduces time to resolution from weeks to minutes to hours
- In-ED repositioning reduces ED length of stay and improves patient satisfaction

□ **Clinical Pearl:** If repositioning is successful in the ED, the patient leaves with resolved symptoms and does not require medication.

When to Use Antiemetic Medication

Medications should be reserved for patients in whom:

- Repositioning manoeuvre is contraindicated
- Repositioning is attempted but fails
- Patient has severe nausea/vomiting limiting cooperation

Common antiemetics include:

- **Ondansetron:** 4-8 mg IV/IM/SL; highly effective for nausea
- **Metoclopramide:** 10 mg IV/IM; dopamine antagonist
- **Prochlorperazine:** 5-10 mg IV/IM; phenothiazine antipsychotic

□ **Clinical Insight:** Antiemetic medication should never delay or replace repositioning manoeuvres.

Discharge Advice and Recurrence Counselling

Patients successfully treated with repositioning in the ED can be safely discharged with clear advice.

- **Activity:** No specific head position restrictions required
- **Home manoeuvre:** Teach patient Epley manoeuvre to perform 1-2 times daily if symptoms persist [5]
- **Head movements:** Use cautious, deliberate movements for first 48 hours
- **Medication:** Antiemetic prescription for home use if needed

BPPV has a natural recurrence rate of approximately 15-30% within 1 year. Patients should be advised they can self-manage using home manoeuvre if symptoms return [1,12] [13].

□ **Key Point:** After successful in-ED treatment, most patients do not require specialist referral. Home Epley education and clear recurrence counselling optimize outcomes.

Driving Advice

Patients with acute BPPV should not drive immediately after a symptomatic episode. Vertigo and nausea significantly impair reaction time and safety [2].

However, once the acute vertigo has resolved, driving is safe. Most ED patients treated successfully with repositioning manoeuvres can resume normal activities—including driving—within hours of ED discharge [2].

□ **Clinical Insight:** Clear, practical discharge advice reduces patient anxiety and ED revisits.

VIII. Conclusions and Key Takeaways

BPPV is the most common peripheral vestibular disorder and accounts for approximately 20% of ED dizzy presentations. Recognition and treatment require no advanced imaging and can be accomplished within minutes using bedside examination and simple repositioning manoeuvres [1,2,15].

Posterior canal BPPV (80-90% of cases) is diagnosed by Dix-Hallpike testing and treated with Epley CRM with >80% success rates. Horizontal canal BPPV (5-15%) is diagnosed by supine roll testing and treated with BBQ roll or Gufoni manoeuvres. Anterior canal BPPV is rare but should be suspected if downbeating nystagmus is observed [2,3,6] [3].

The key diagnostic features that distinguish BPPV from central positional nystagmus are latency, fatiguability, torsional nystagmus, and brief duration (<1 minute) [2,4].

In-ED repositioning manoeuvres are far more effective than antiemetic medications alone. Most patients can be safely discharged after successful ED repositioning with home exercise education [3,5].

□ **Clinical Pearl:** Bedside mastery of BPPV diagnosis and treatment is a high-value ED skill that improves patient outcomes, reduces unnecessary imaging, and enhances ED efficiency.

□ **Clinical Insight:** The presence of latency and fatigue on repeat positional testing is pathognomonic for peripheral BPPV.

□ **Key Point:** BPPV is common, simple to diagnose with bedside examination, and highly treatable in the ED. Mastering these diagnostic and treatment skills is essential for all emergency medicine clinicians.

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Accuracy and Currency

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