

Vertigo in Primary Care: BPPV, Vestibular Migraine, Meniere's Disease

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Vertigo, the illusory sensation of movement, presents a common and often debilitating complaint in primary care, accounting for an estimated 3% of all primary care visits.¹ Distinguishing between peripheral and central causes, and then identifying the specific peripheral etiology, dictates effective management and prevents unnecessary referrals or investigations. General practitioners frequently encounter three primary peripheral vestibular disorders: benign paroxysmal positional vertigo (BPPV), vestibular migraine, and Meniere's disease.

KEY TAKEAWAYS

- **The Pivot:** A structured clinical assessment, focusing on symptom triggers and duration, can differentiate the most common vertigo syndromes without advanced imaging.
- **The Data:** BPPV is the most frequent cause, responsible for up to 20% of all vertigo presentations, with a lifetime prevalence of 2.4%.
- **The Action:** Clinicians should routinely perform the Dix-Hallpike maneuver for suspected BPPV and consider a trial of vestibular suppressants for acute, severe episodes.

Patients presenting with vertigo describe a sensation of spinning, swaying, or tilting, often accompanied by nausea, vomiting, and imbalance. The challenge for primary care clinicians lies in rapidly and accurately diagnosing the underlying cause, as treatment strategies vary significantly. Peripheral vestibular disorders, originating from the inner ear or vestibular nerve, constitute the vast majority of cases, while central causes, involving the brainstem or cerebellum, are less common but demand urgent investigation due to their potential for serious neurological sequelae. A careful history, focusing on the character, duration, and triggers of vertigo, alongside a targeted physical examination, remains the cornerstone of diagnosis.

Benign paroxysmal positional vertigo (BPPV) stands as the most prevalent peripheral vestibular disorder, with a lifetime prevalence of 2.4% and an incidence of 10.7 to 16.0 per 100,000 population per year. It is particularly common in older adults, with incidence increasing with age. The pathophysiology involves the displacement of otoconia (calcium carbonate crystals) from the utricle into one of the semicircular canals, most commonly the posterior canal. These displaced otoconia then move within the canal in response to head position changes, causing abnormal endolymphatic flow and triggering brief, intense vertigo episodes. Patients typically report sudden, brief (seconds to less than one minute) episodes of spinning vertigo provoked by specific head movements, such as looking up, lying down, or turning over in bed. Nausea and nystagmus often accompany these episodes, but hearing loss or tinnitus are absent. The diagnostic gold standard is the Dix-Hallpike maneuver, which, when positive, elicits vertigo and characteristic torsional nystagmus with a latency of a few seconds, lasting less than 60 seconds. The Epley maneuver, a series of head movements designed

to reposition the otoconia, is the primary treatment and demonstrates high efficacy, with resolution rates of 80-90% after one or two treatments.¹

Differentiating the common causes

Vestibular migraine, a distinct clinical entity, represents the second most common cause of spontaneous recurrent vertigo, affecting approximately 1% of the general population. It is more common in women and often has a personal or family history of migraine headaches. The diagnostic criteria for vestibular migraine require at least five episodes of vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours. These episodes must be associated with current or previous migraine with or without aura, and at least half of the vestibular episodes must be accompanied by at least one migraine feature: headache (of at least moderate intensity), photophobia, phonophobia, or visual aura. The vestibular symptoms themselves can be diverse, including spontaneous vertigo (internal or external), positional vertigo, visually induced vertigo, or head motion intolerance. Unlike BPPV, the vertigo in vestibular migraine is not necessarily triggered by specific head positions, and unlike Meniere's disease, hearing symptoms are not a primary feature. Acute treatment often involves vestibular suppressants (e.g., benzodiazepines, antihistamines) and antiemetics, similar to other vertigo types, but specific migraine abortive therapies (triptans) can also be effective. Prophylactic treatment, using medications like beta-blockers, tricyclic antidepressants, or topiramate, is indicated for frequent or severe attacks and has shown to reduce attack frequency and severity.²

Meniere's disease, while less common than BPPV or vestibular migraine, is a significant cause of recurrent vertigo, with an estimated prevalence of 0.2% in Europe. It is characterised by a classic triad of symptoms: episodic vertigo, fluctuating sensorineural hearing loss, and tinnitus, often accompanied by aural fullness. The pathophysiology involves endolymphatic hydrops, an excess of endolymphatic fluid in the inner ear, leading to distension of the endolymphatic sac. Vertigo attacks in Meniere's disease are typically severe, spontaneous, and last from 20 minutes to several hours, but rarely more than 24 hours. They are often preceded by an increase in aural fullness or tinnitus. Over time, the fluctuating hearing loss tends to progress to a permanent deficit, particularly in the low frequencies. Diagnosis relies on clinical criteria, including two or more spontaneous episodes of vertigo, each lasting 20 minutes to 12 hours, audiometrically documented low-to-medium frequency sensorineural hearing loss in the affected ear on at least one occasion, fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear, and no better explanation for the symptoms.³

Management of Meniere's disease focuses on symptom control and preventing disease progression. Acute attacks are managed with vestibular suppressants and antiemetics. Long-term management often involves dietary modifications, particularly sodium restriction, and diuretics (e.g., hydrochlorothiazide) to reduce endolymphatic fluid volume. For refractory cases, intratympanic injections of corticosteroids or gentamicin, or surgical interventions such as endolymphatic sac decompression or vestibular neurectomy, may be considered. The goal is to reduce the frequency and severity of vertigo attacks while preserving hearing function as much as possible. The progressive nature of hearing loss in Meniere's disease distinguishes it from BPPV and vestibular migraine, where hearing is typically unaffected. The clinical assessment of a patient presenting with vertigo must systematically exclude central causes, which, though rare, can mimic peripheral vestibular symptoms. Red flag signs and symptoms, such as new-onset headache, focal neurological deficits (e.g., diplopia, dysarthria, dysphagia, hemiparesis), altered consciousness, or gait instability disproportionate to the vertigo, necessitate immediate neurological evaluation and often neuroimaging. The HINTS (Head Impulse, Nystagmus, Test of Skew) examination, performed by experienced clinicians, can help differentiate

central from peripheral vertigo in the acute setting, particularly in patients with acute vestibular syndrome. A normal head impulse test, direction-changing nystagmus, and skew deviation are highly suggestive of a central lesion, even in the absence of other neurological signs. But this test requires proficiency and should not replace a comprehensive neurological assessment when central pathology is suspected.

Primary care clinicians must also consider less common peripheral causes, such as vestibular neuritis or labyrinthitis, which present as acute, sustained vertigo lasting days, often following a viral illness. Vestibular neuritis involves inflammation of the vestibular nerve, causing severe vertigo, nausea, and imbalance without hearing loss. Labyrinthitis is similar but includes hearing loss and tinnitus. Both are typically self-limiting, with symptomatic treatment and vestibular rehabilitation being the mainstays of management. The distinction from BPPV is the sustained nature of the vertigo, and from Meniere's, the absence of recurrent episodes and progressive hearing loss. The acute onset and prolonged duration differentiate these conditions from the episodic nature of BPPV and vestibular migraine.

The diagnostic process for vertigo in primary care therefore involves a careful stepwise approach. First, rule out central causes using a thorough neurological examination and by identifying any red flag symptoms. Second, if a peripheral cause is suspected, differentiate between episodic and sustained vertigo. For episodic vertigo, consider BPPV (brief, positional, no hearing loss), vestibular migraine (variable duration, migraine features, no hearing loss), and Meniere's disease (20 minutes to hours, hearing loss, tinnitus, aural fullness). For sustained vertigo, consider vestibular neuritis or labyrinthitis. The judicious use of diagnostic maneuvers, such as the Dix-Hallpike, and a detailed history are often sufficient to guide initial management. Referral to an otolaryngologist or neurologist is appropriate for atypical presentations, refractory symptoms, or when central pathology cannot be confidently excluded. The goal is to provide timely and effective relief for patients, many of whom experience significant functional impairment and anxiety due to their vertigo.

CLINICAL IMPLICATIONS

The prevalence of vertigo in primary care demands a systematic diagnostic approach. Over-reliance on imaging for every dizzy patient is inefficient and often unhelpful, particularly when a clear peripheral pattern emerges from the history and examination. Clinicians should feel confident in diagnosing and initiating treatment for BPPV based solely on a positive Dix-Hallpike maneuver, as the Epley maneuver offers immediate, low-risk relief.

Vestibular migraine, often underdiagnosed, requires a higher index of suspicion, especially in patients with a history of headaches. Recognising its diverse vestibular manifestations means that not all vertigo is positional. A trial of migraine-specific therapies, alongside vestibular suppressants, can significantly improve patient quality of life.

Meniere's disease, while less common, presents a more complex management challenge due to its progressive nature and impact on hearing. Early diagnosis and lifestyle modifications, particularly sodium restriction, are critical. Primary care's role here is to identify the classic triad and initiate basic management, referring for specialist input when symptoms are severe or refractory.

Ultimately, a structured clinical assessment, prioritising history and physical examination over immediate investigations, remains the most effective strategy for managing vertigo in primary care. This approach not

only streamlines patient care but also conserves resources, ensuring that specialist referrals are reserved for complex or ambiguous cases.

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