Bilateral Vestibulopathy — Rehabilitation Strategies

Substitution, Adaptation, and Quality-of-Life Outcomes

Vestibular Physiotherapy for Clinicians

Topic 5 of 12

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How to Use This Review

Bilateral vestibulopathy (BV) is one of the most challenging scenarios in vestibular rehabilitation. Unlike unilateral loss, bilateral loss eliminates the adaptive signal that drives gain re-training. This review equips physiotherapists with practical, evidence-based strategies for managing oscillopsia, dark-induced imbalance, falls prevention, and quality-of-life burden, organised across four clinical themes: diagnosis and functional impact, substitution strategy, exercise programming, and long-term compensation.

Audience: physiotherapists with a special interest in vestibular rehabilitation. 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.

Callout Guide

Clinical Pearl: Highlights a key clinical insight that materially changes management.

Pitfall: Highlights a common mistake or trap to avoid in practice.

Note: Provides a definition or framing detail for context.

Caution: Flags a safety issue or red flag requiring escalation.

Contents

I. Introduction: Definition and Epidemiology

Bilateral vestibulopathy (BV) is hypofunction of bilateral peripheral vestibular systems. Per the Bárány Society 2017 consensus criteria, definite BV requires bilaterally impaired horizontal VOR documented by video head impulse test (vHIT gain less than 0.6), caloric test (sum SPV less than 6 degrees per second on each side), or rotational chair (gain less than 0.1 at 0.1–0.32 Hz) [1,11].

Prevalence ranges between 28 and 50 per 100,000 in developed countries [2], but true incidence is likely under-estimated because mild bilateral loss is often missed in non-specialist settings [3]. Bilateral loss has profound functional, cognitive, and quality-of-life consequences [7,17].

Snapshot: BV affects roughly 30 per 100,000 adults; definite diagnosis requires bilateral angular-VOR impairment on vHIT, caloric, or rotational chair testing per Bárány Society 2017 criteria.

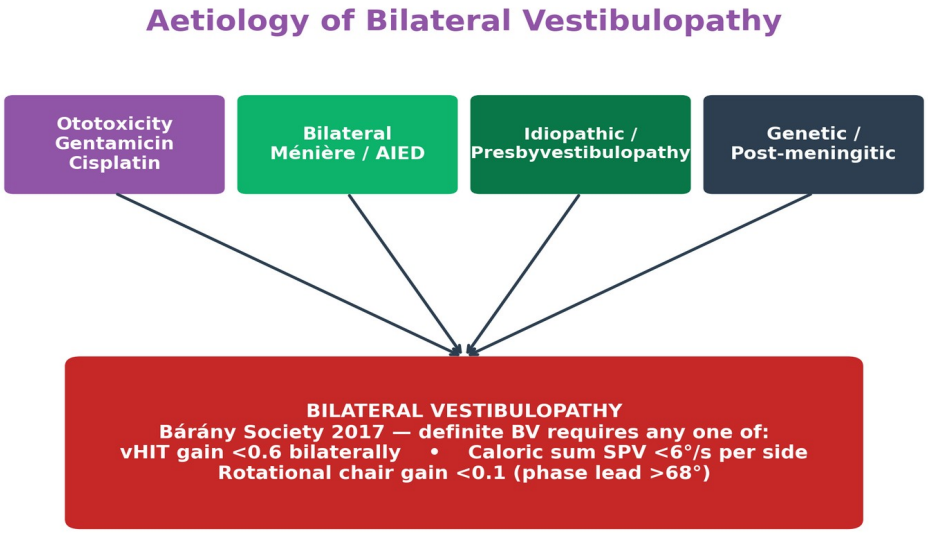

Figure 1. Common aetiologies of bilateral vestibulopathy and the Bárány Society 2017 diagnostic thresholds (any one of: vHIT gain less than 0.6 bilaterally, caloric sum SPV less than 6°/s on each side, or rotational chair gain less than 0.1).

Source: Australian Dizziness Clinics, 2026.

II. Aetiology of Bilateral Vestibulopathy

Ototoxicity: Gentamicin and Cisplatin

Aminoglycoside antibiotics accumulate in the labyrinth and cause irreversible hair-cell death. Gentamicin preferentially affects vestibular hair cells; cisplatin damages auditory hair cells but can also cause vestibular toxicity [6]. Risk factors for ototoxicity include high cumulative dose, renal impairment, prolonged exposure, and concurrent loop diuretics.

Caution: In any patient on aminoglycoside or platinum chemotherapy, document baseline gait and balance before starting, and screen for early bilateral vestibulopathy symptoms at every cycle.

Bilateral Ménière and Autoimmune AIED

Bilateral Ménière disease occurs in 15–30 percent of unilateral cases, typically manifesting in the contralateral ear 2–10 years after the initial diagnosis [3]. Autoimmune inner-ear disease (AIED) presents with bilateral, fluctuating, progressive sensorineural hearing loss and vestibular symptoms; immunosuppression may halt progression.

Idiopathic and Presbyvestibulopathy

A significant proportion of bilateral vestibulopathy cases remain idiopathic [3]. Age-related vestibulopathy (presbyvestibulopathy) is increasingly recognised in older adults and contributes to falls, gait instability, and cognitive decline [11].

III. Clinical Features and Diagnostic Criteria

The cardinal symptom is oscillopsia — illusory motion of the visual world during head movement [13,15]. Unlike the brief oscillopsia of unilateral loss, oscillopsia in bilateral vestibulopathy is persistent and severe, particularly during walking, running, or driving.

Gait instability is profound in darkness or on uneven surfaces, even though ambulation in bright lighting may appear nearly normal. This light-versus-dark dissociation is the diagnostic hallmark of bilateral vestibular loss [13,14].

Clinical Pearl: Ask every patient with non-specific imbalance — does the imbalance get worse in the dark or on uneven ground? A clear yes is highly suggestive of bilateral vestibulopathy and should trigger objective vestibular testing.

IV. Assessment Battery for Bilateral Loss

Objective confirmation is essential before designing a rehabilitation programme. A bilaterally pathological bedside head impulse test alone classifies the patient as probable bilateral vestibulopathy; vHIT, calorics, or rotational chair confirms the diagnosis [1,12]. Functional measures including the Dynamic Gait Index, Functional Gait Assessment [19], and patient-reported Dizziness Handicap Inventory [20] should be obtained at baseline to track rehabilitation progress.

Test	Threshold	Notes
Video head impulse	Horizontal VOR gain less than 0.6 bilaterally	Most sensitive bedside-portable test
Caloric	Sum bithermal max peak SPV less than 6°/s on each side	Reduced or absent canal response
VEMP (cervical and ocular)	Reduced amplitude or absent bilaterally	Confirms otolith involvement
Dynamic visual acuity	Greater than 3-line drop with active head movement	Functional gaze stability
Romberg / FGA	Falls in dark or on foam, FGA less than 22/30	Falls risk threshold
DHI / ABC	Baseline values for re-test	Self-report handicap and confidence

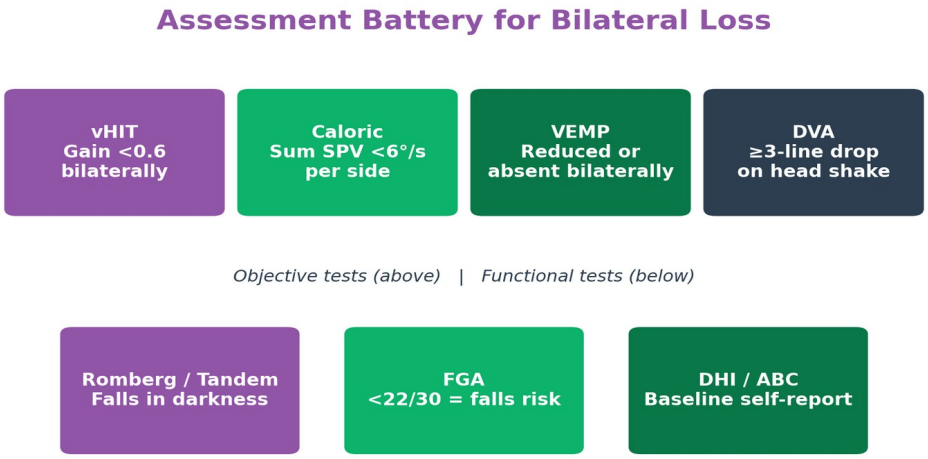

Figure 2. Assessment battery for bilateral vestibulopathy — objective tests above and functional tests below.
Source: Australian Dizziness Clinics, 2026.

Pitfall: A normal Dix-Hallpike does not exclude bilateral vestibulopathy. Patients with BV can also have superimposed BPPV, and many have subclinical untreated canalolithiasis at the time of vestibulopathy assessment.

V. Modified VRT: Why Adaptation Training Fails

Standard VRT relies on a vestibular signal being present to drive adaptive plasticity. In bilateral vestibulopathy both labyrinths are deficient, so classical gaze stabilisation exercises (X1 and X2 viewing) cannot drive VOR adaptation [4,5]. Patients who undertake unmodified VRT often report worsening rather than improvement, leading to disengagement.

Rehabilitation shifts from adaptation to substitution: teaching the brain to rely on vision, proprioception, and cervical proprioceptive (cervico-ocular) reflexes instead of vestibular input [4,8,14]. The substitution paradigm has emerged as the standard of care for definite bilateral vestibulopathy.

Clinical Pearl: Pure bilateral loss = substitution-led rehab; severe but incomplete BVH still benefits from limited VOR adaptation work added on top of the substitution programme.

VI. Substitution Strategies

Cervico-Ocular Reflex Enhancement

Cervical proprioceptors signal head position to the vestibular nuclei and partially fill the gap left by absent VOR signal [5,8]. The lead exercise is slow-frequency head movement against a fixed visual target, which reinforces cervico-ocular reflex contribution to gaze stability.

Visual Preprogramming and Saccadic Substitution

Train rapid, preprogrammed saccades toward targets before moving the head [8,12]. Eyes leap to the target pre-emptively instead of tracking smoothly during head movement. The cued preprogrammed saccade is the strongest functional substitute for absent VOR.

Proprioceptive Training in Darkness

When vision is unavailable, proprioceptive input from feet, ankles and trunk becomes the sole source of balance control [4,5]. Strengthen through graded exercises: standing on foam with eyes closed, single-leg stance with eyes closed, and progressive surface compliance challenges. Patients with comorbid migraine require migraine-aware adaptation of the substitution programme [10].

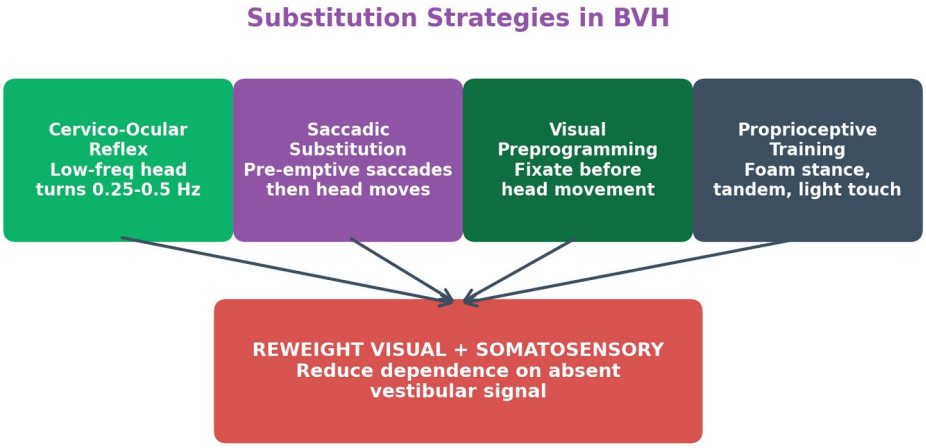

Figure 3. Four substitution strategies — cervico-ocular reflex, saccadic substitution, visual preprogramming, and proprioceptive training in darkness.

Source: Australian Dizziness Clinics, 2026.

VII. Oscillopsia Management

Oscillopsia is the most distressing symptom for many patients with bilateral vestibulopathy [13,15]. Unlike gait instability, oscillopsia persists because VOR loss cannot be overcome through compensation; substitution strategies provide partial mitigation but rarely eliminate the symptom completely.

Environmental and Optical Strategies

Increase lighting intensity and contrast at home and at work [13]. Install motion-sensor lights in hallways and use bright task lighting for reading. Prismatic glasses that move the visual field opposite head motion partially compensate for VOR loss.

Behavioural Strategies

Teach minimisation of rapid head movements during visual tasks [16]. When reading, move the eyes without moving the head; when changing direction, step-turn slowly. These behavioural changes preserve gaze stability and significantly reduce oscillopsia-related disability.

VIII. Falls Prevention and Home Safety

Fall risk in bilateral vestibulopathy is substantial — prospective studies report 30–50 percent annual fall rates in untreated patients [9,17]. Environmental hazards pose the major threat in the home, particularly stairs, bathrooms, and night-time mobility.

- Eliminate throw rugs, secure electrical cords, and remove under-foot clutter.

- Ensure stair handrails on both sides of every staircase.
- Install grab bars in bathrooms next to toilet and inside the shower.
- Add motion-sensor or timed nighttime lighting on the path to the bathroom.
- A cane or walker reduces fall risk significantly, especially outdoors and at night.

Caution: Outdoor falls in bilateral vestibulopathy carry a high fracture rate. A cane in darkness or outdoors is not optional in established BV — it is part of the standard prescription, even when clinic gait looks safe.

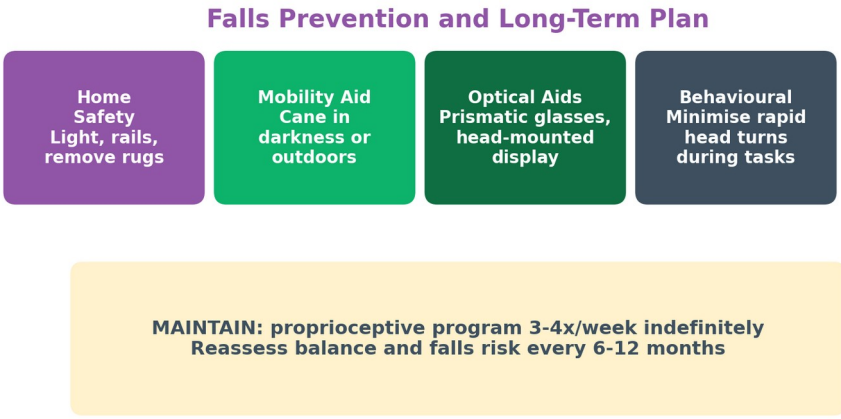

Figure 4. Falls prevention bundle for bilateral vestibulopathy — home safety, mobility aids, optical strategies, and behavioural change.

Source: Australian Dizziness Clinics, 2026.

IX. Prognosis and Long-Term Management

Prognosis depends on aetiology and age [13,18]. If caused by self-limited ototoxic exposure, vestibular function is static and central compensation plateaus within 6–12 months. If progressive (autoimmune, idiopathic, presbyvestibulopathy), function may continue to deteriorate, requiring ongoing reassessment and rehabilitation adjustments.

Most patients achieve a satisfactory quality of life within 6–12 months through substitution, behavioural modification, and environmental change [16,17]. Comorbid visual loss, peripheral neuropathy, and cognitive impairment significantly worsen prognosis; emerging evidence highlights cognitive consequences of bilateral vestibular loss including hippocampal atrophy and spatial memory deficits [7].

Maintenance and Relapse Prevention

Long-term success requires maintenance exercises — proprioceptive training three to four times per week indefinitely, periodic balance reassessment, and prompt intervention if function deteriorates [4]. Annual review with the multidisciplinary team is recommended.

Sensory Reweighting in Bilateral Vestibulopathy

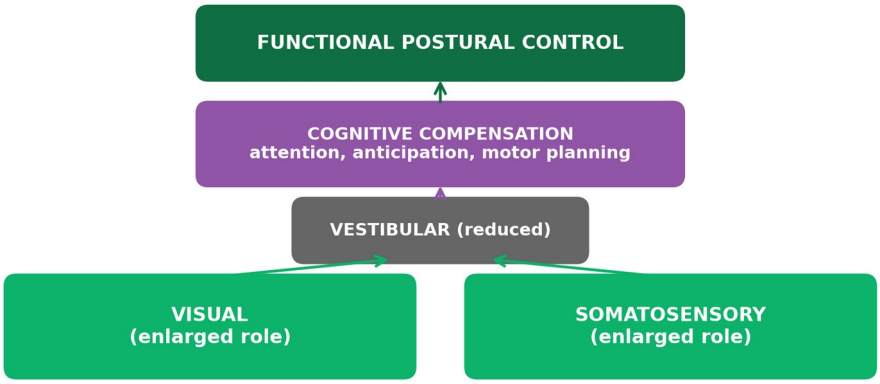

Figure 5. Sensory reweighting in bilateral vestibulopathy — vestibular layer reduced, visual and somatosensory layers enlarged, cognitive layer above for compensation.

Source: Australian Dizziness Clinics, 2026.

Snapshot: BV recovery is mostly substitution-driven with 6–12 months to plateau; maintenance exercise indefinitely is the critical adherence lever.

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