Post-Concussive Dizziness

Recognition, Assessment, and Early Management After Mild Traumatic Brain Injury

Vestibular Medicine for General Clinicians

Topic 12 of 14

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How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with dizziness.

The review is designed to be read in a single 20–30 minute sitting, or used as a desktop reference. It is supported by an A4 one-page cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

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, emergencies, and critical safety points requiring immediate action.

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I. Scale of the Problem

Mild traumatic brain injury (mTBI) is one of the most common reasons for presentation to Australian emergency departments, with an estimated incidence of 100–300 per 100,000 population per year [1]. Sport, motor vehicle collisions, workplace injury, and falls in older adults account for the majority of cases. Although the injury is termed "mild", persistent symptoms are common and dizziness is among the most disabling.

Between 30% and 60% of patients experience dizziness or imbalance in the weeks following mTBI [2,3]. In most cases symptoms settle within the first two to four weeks, but a substantial minority — around 15–30% — have symptoms that persist beyond three months [4]. Chronic post-concussive dizziness is a well-recognised cause of inability to return to work, to sport, and to full driving, and it is a leading driver of medico-legal complexity after head injury [5].

□ **Key Point:** Post-concussive dizziness is common, heterogeneous, and treatable. The central clinical error is to ascribe all post-concussive dizziness to "the concussion" and to wait for it to settle — most persistent cases have a specific, identifiable, and treatable vestibular mechanism.

The shift in the evidence base over the past decade has been substantial. Prolonged rest — once routine — is now known to worsen outcomes, and early supervised vestibular rehabilitation is now endorsed as standard of care in international consensus statements including the Concussion in Sport Group (CISG) 6th International Consensus [6] and the Ontario Neurotrauma Foundation guidelines [7].

II. Mechanisms — Why Patients Are Dizzy After a Head Knock

Post-concussive dizziness is not a single entity. The head and neck are subjected to rapid acceleration-deceleration forces that can injure the peripheral vestibular apparatus, the central vestibular pathways, the cervical spine, and the autonomic nervous system simultaneously. A careful clinician asks not "is this patient dizzy from concussion?" but "which of the recognised mechanisms is operating in this patient?"

Mechanism	Typical pattern	Key clinical clue
BPPV (traumatic)	Brief positional vertigo, seconds, with rolling / lying / looking up	Positive Dix-Hallpike or supine roll test; often bilateral or multi-canal after trauma
Vestibular migraine (post-traumatic)	Episodic vertigo minutes to hours, often with headache, photophobia, visual triggers	Personal or family history of migraine; triggered by visual motion, sleep loss, stress
Cervicogenic dizziness	Non-specific unsteadiness worse with neck movement and sustained postures	Reduced cervical range, tender suboccipital and upper trapezius musculature
PPPD (post-concussive)	Persistent unsteadiness > 3 months, worse upright, in visual environments, with movement	Often preceded by an acute vestibular event or mTBI
Labyrinthine concussion	Acute vertigo, hearing loss or tinnitus on the impact side	Unilateral sensorineural hearing loss on audiogram
Autonomic / orthostatic	Lightheadedness on standing, fatigue, exercise intolerance	Orthostatic vitals abnormal; consider POTS, especially in adolescents

Most patients have more than one mechanism operating concurrently. Treatment directed at each mechanism individually is more effective than a single global approach.

□ **Clinical Insight:** The three most commonly missed mechanisms are traumatic BPPV (because it is not looked for), vestibular migraine (because the headache may not dominate the episode), and PPPD (because it is dismissed as "post-concussion syndrome" or "anxiety").

Patients with vascular risk factors or severe mechanism of injury warrant separate consideration for vertebral artery dissection and central injury. A new neurological sign, severe or evolving headache, or a Glasgow Coma Scale that does not normalise should prompt imaging and urgent assessment rather than conservative management.

III. Traumatic BPPV — The Diagnosis You Must Not Miss

Benign paroxysmal positional vertigo is the single commonest identifiable cause of dizziness after head injury, accounting for 20–30% of persistent post-concussive dizziness in systematic series [8,9]. The pathophysiology is straightforward: acceleration-deceleration forces dislodge otoconia from the utricle into the semicircular canals. Despite this, traumatic BPPV is under-diagnosed for three reasons: it is not looked for, the Dix-Hallpike is perceived as difficult, and traumatic cases are more likely to be atypical (bilateral, multi-canal, or horizontal canal rather than the classic posterior canal presentation).

□ **Important:** Every patient with post-concussive dizziness deserves a Dix-Hallpike manoeuvre and a supine roll test, regardless of whether their described symptoms sound typical of BPPV. Traumatic BPPV is the highest-yield diagnosis you can make in this population, and the treatment — a repositioning manoeuvre — is immediate and free.

Typical features of traumatic BPPV include:

- Brief (< 1 minute) vertigo triggered by rolling over in bed, lying flat, getting up from lying, looking up, or bending forward.
- Symptoms may appear a few days to weeks after the injury, not necessarily from day one.
- Bilateral posterior canal involvement is more common after trauma than in idiopathic BPPV.
- Horizontal canal BPPV is over-represented after trauma; if the Dix-Hallpike is negative but positional vertigo is described, a supine roll test must be performed.
- Recurrence rates are higher than in idiopathic BPPV — up to 40% within two years [10].

□ **Clinical Pearl: When documenting a post-concussive consultation, record Dix-Hallpike and supine roll test results in the examination section — not just the standard neurological screen. If these were not performed, say so and arrange them.**

Repositioning manoeuvres (Epley for posterior canal, Gufoni or BBQ roll for horizontal canal) are as effective in traumatic BPPV as in idiopathic cases, although more treatment sessions may be required and recurrence is more likely. If a clinician is not confident in diagnosis or treatment, prompt referral to a vestibular physiotherapist or vestibular physician should be arranged rather than symptomatic medication.

IV. Post-Traumatic Vestibular Migraine

Vestibular migraine is the second commonest cause of persistent post-concussive dizziness [11]. Head injury is a well-documented precipitant of migraine in individuals who have a migrainous predisposition, and patients with a prior history of migraine are particularly likely to develop chronic post-concussive symptoms.

The diagnosis is frequently missed because:

- Patients do not always associate their episodic dizziness with migraine, especially when the dominant symptom is vertigo rather than headache.
- Post-concussive headache and post-traumatic vestibular migraine overlap clinically and are often treated as a single entity.
- Clinicians ask about headache but not about the pattern of vestibular episodes.
- Triggers — screens, supermarkets, sleep deprivation, skipped meals — are characteristic but rarely elicited.

□ **Clinical Insight:** In any young or middle-aged adult with persistent episodic dizziness after head injury, ask specifically about personal or family history of migraine, photophobia and phonophobia during episodes, and visual motion triggers. A positive answer to two of these three makes post-traumatic vestibular migraine the most likely diagnosis.

Management follows standard vestibular migraine principles: lifestyle measures (sleep regularity, hydration, regular meals, caffeine moderation), trigger identification, and where episodes are frequent or disabling, preventive pharmacotherapy. First-line preventives with evidence in vestibular migraine include propranolol, candesartan, amitriptyline, nortriptyline, topiramate, and flunarizine (where available). Referral to a vestibular physician or neurologist experienced in vestibular migraine is appropriate where episodes remain frequent despite lifestyle measures or where preventive therapy is being considered.

V. Cervicogenic and Autonomic Contributions

The head and neck are injured together. Cervical soft tissue injury, loss of normal cervical proprioception, and muscle guarding all contribute to post-concussive dizziness, and cervicogenic mechanisms may predominate in patients with whiplash-type mechanisms of injury [12].

Features that raise the suspicion of a cervicogenic contribution:

- Dizziness described as unsteadiness or disorientation rather than vertigo.
- Symptoms worsened by sustained neck postures (desk work, reading, driving).
- Reduced active cervical range of motion, especially rotation.
- Tenderness in the suboccipital region, upper trapezius, and sternocleidomastoid.
- Improvement in dizziness with targeted cervical physiotherapy.

Cervicogenic dizziness is a diagnosis that should be made in conjunction with vestibular assessment rather than instead of it. Many patients have both a peripheral vestibular component and a cervical component, and treating only one leaves the patient only partially improved.

Autonomic Dysfunction

Autonomic dysregulation — particularly postural orthostatic tachycardia syndrome (POTS) — is increasingly recognised after mTBI, especially in adolescents and young adults [13]. Patients describe lightheadedness on standing, fatigue, exercise intolerance, and cognitive "fog", often with palpitations. The NASA lean test or a 10-minute active stand with heart-rate and blood-pressure measurement is a simple screening tool in primary care.

□ **Clinical Pearl:** In any adolescent or young adult with prolonged post-concussive symptoms including lightheadedness on standing and exercise intolerance, measure supine and standing heart rate and blood pressure. A sustained rise of >30 bpm (>40 bpm in under-19s) within 10 minutes of standing meets criteria for suspected POTS and warrants further assessment.

VI. PPPD After Concussion

Persistent Postural-Perceptual Dizziness (PPPD), formerly known as chronic subjective dizziness or phobic postural vertigo, is now a Bárány Society-defined disorder [14]. It is one of the commonest causes of persistent dizziness after an acute vestibular event of any type — including mTBI — and is found in 20–40% of patients with post-concussive dizziness persisting beyond three months [15].

The diagnostic criteria (Bárány, 2017) are:

- Dizziness, unsteadiness, or non-spinning vertigo present on most days for three months or more.
- Symptoms exacerbated by upright posture, active or passive motion, and exposure to moving or complex visual stimuli.
- The disorder typically follows a precipitating event — vestibular neuritis, BPPV, vestibular migraine, head injury, or a panic attack.
- Significant functional impairment.
- Symptoms not better explained by another disorder.

□ **Key Point:** PPPD is not a diagnosis of exclusion, nor is it "functional" or "psychological". It is a recognised chronic vestibular syndrome with specific diagnostic criteria and specific treatments. Labelling it as anxiety delays effective care by months or years.

Treatment of post-concussive PPPD is identical to PPPD from any other precipitant: patient education, vestibular rehabilitation with a therapist experienced in PPPD (habituation and visual-motion desensitisation rather than gaze-stabilisation alone), cognitive-behavioural therapy where indicated, and SSRI or SNRI pharmacotherapy (sertraline, escitalopram, venlafaxine) where symptoms are persistent or disabling.

VII. Early Vestibular Rehabilitation — The Standard of Care

Historical practice recommended prolonged physical and cognitive rest after concussion — so-called "cocoon therapy". The evidence base has now reversed this advice. Systematic reviews and randomised controlled trials show that prolonged rest is associated with worse outcomes, and that early symptom-limited return to activity and early supervised vestibular rehabilitation shorten recovery time and reduce the proportion of patients developing chronic symptoms [16,17].

Key principles of early rehabilitation, endorsed by the CISG 6th International Consensus [6] and Australian concussion position statements [18]:

- Relative rest (24–48 hours) rather than strict rest, with progressive return to low-intensity activity as tolerated.
- Referral to vestibular physiotherapy if dizziness persists beyond 7–10 days.
- Sub-symptom-threshold aerobic exercise (the Buffalo Concussion Treadmill Test protocol is one validated approach) reduces persistent post-concussive symptoms [19].

- Supervised gaze stabilisation, habituation, and balance retraining for identified vestibular dysfunction.
- Treatment of co-existing BPPV, vestibular migraine, cervicogenic and autonomic contributions in parallel.

□ **Important:** Advising a post-concussive patient to "rest in a dark room until dizziness settles" is now contrary to the evidence. It prolongs recovery and is a key driver of chronic symptoms. Early, supervised, graded return to activity and to vestibular rehabilitation is the standard of care.

Return-to-Sport and Return-to-Work Staging

The CISG staged return-to-sport protocol — light activity, sport-specific exercise, non-contact training, full-contact training, return to play — is now standard for athletes. A parallel staged return-to-work is appropriate for occupational concussion. In both, progression is symptom-limited: the patient advances to the next stage when the current stage is tolerated without significant symptom provocation.

VIII. When to Refer

Referral to a vestibular physician or dedicated vestibular service should be made without delay where any of the following apply:

- Dizziness persisting beyond 4 weeks from the injury.
- Confirmed or suspected BPPV that has not resolved after one or two repositioning attempts, or suspected non-posterior-canal (horizontal or anterior) BPPV.
- Recurrent BPPV after initial successful treatment.
- Episodic dizziness suggestive of vestibular migraine or Ménière's disease where preventive therapy is being considered.
- Chronic unsteadiness consistent with PPPD.
- Any central sign — new neurological symptoms, gait ataxia disproportionate to vertigo, direction-changing nystagmus, abnormal HINTS — requires urgent neurological assessment.
- Unilateral hearing loss or tinnitus following the injury — urgent ENT referral for audiogram and consideration of labyrinthine concussion.
- Failure to return to work, sport, or driving by 4–6 weeks.

Presentation	Action	Timeframe
Red flag neuro signs / worsening GCS / severe headache	ED, urgent imaging	Same day
Sudden hearing loss with vertigo	ENT emergency referral, audiogram	Within 72 hours
BPPV identified but not resolving	Vestibular physiotherapy or vestibular physician	Within 1–2 weeks
Persistent dizziness > 4 weeks	Vestibular physician, vestibular physiotherapy	Routine, 2–4 weeks
Suspected PPPD or vestibular migraine	Vestibular physician for diagnosis and management	Routine
Chronic symptoms affecting driving / work / sport	Vestibular physician, concussion clinic or dedicated service	Routine

Australian access to vestibular physicians varies by state. Telehealth models now extend access significantly beyond metropolitan centres.

□ **Clinical Insight:** Early referral is not a failure of primary care — it is the highest-yield intervention for patients who will otherwise drift into chronic disability. The 4-week mark is a useful operational trigger.

IX. Assessment and Management Pathway

The pathway below distils the evidence-based approach to the post-concussive dizzy patient in a form suitable for primary care and emergency use.

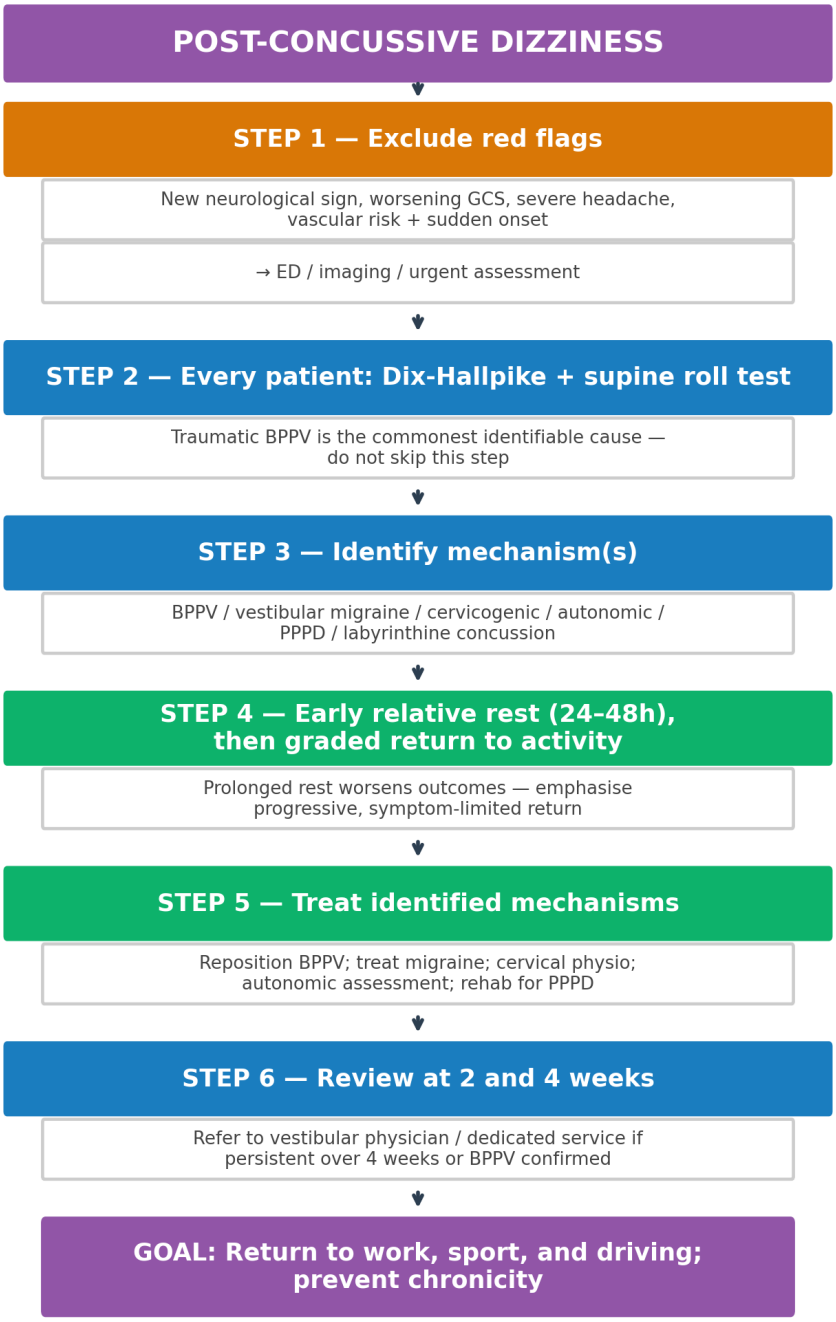

Figure 1. Post-Concussive Dizziness Assessment and Management Pathway
Source: Australian Dizziness Clinics — clinical algorithm.

X. Documentation, Driving, and Safety-Netting

A well-documented post-concussive consultation should include the mechanism of injury, loss of consciousness or amnesia, Glasgow Coma Scale, neurological examination, Dix-Hallpike and supine roll

test results, orthostatic vitals where appropriate, current symptom burden, identified mechanisms, advice given (including activity progression), and a clear review or referral plan.

Driving

Patients with ongoing significant dizziness should be advised not to drive until symptoms are controlled. Austroads Assessing Fitness to Drive guidance [20] requires the treating clinician to assess functional capacity and to advise the patient in writing where fitness to drive is in question. Document this advice.

Safety Netting

The patient and their family should be given clear written advice about warning signs that require urgent return — worsening headache, vomiting, drowsiness, confusion, seizure, weakness, visual change — and a named follow-up point. This is particularly important for patients discharged from ED with "mild head injury".

- Provide a written post-concussive symptom diary or digital tool (the SCAT6 symptom inventory is freely available).
- Schedule review at 7–10 days if symptomatic.
- Refer to vestibular physiotherapy early if dizziness dominates the symptom profile.
- Refer to vestibular physician at 4 weeks if symptoms persist, earlier if BPPV is identified or a complex mechanism is suspected.
- Document return-to-work, return-to-school, and return-to-driving advice explicitly.

□ **Key Point:** The post-concussive dizzy patient who does well has three things: a named clinician reviewing them, a specific mechanism identified and addressed, and a graded return to activity starting within days rather than weeks. These are all achievable in primary care.

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