Post-Concussion Dizziness (Post-Concussive Vestibular Syndrome):

A Vestibular Physician's Deep Review of Pathophysiology, Diagnostic Subtypes, Workup, and Management

Vestibular Medicine for Vestibular Physicians

Central Vestibular Pathology — Module 3.5

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How to Use This Review

This literature review forms part of the Vestibular Medicine for Vestibular Physicians series published by the Australian Dizziness Clinics Education Hub. It is written for vestibular physicians, neuro-otologists, advanced ENT trainees, and vestibular physiotherapists working at the deep end of central vestibular practice, where a working command of mechanism, criteria, and atypical presentations is expected rather than optional. Post-concussion dizziness is one of the most diagnostically heterogeneous presentations in the specialty — combining peripheral vestibular injury, central pathway disruption, cervicogenic dysfunction, autonomic dysregulation, and functional overlay in proportions that vary between patients and shift across recovery.

The review is dense by design — intended as a 30–40 minute deep read or a desktop reference. It is supported by an A4 clinician cheat sheet, short-form clinician videos, audio episodes, and a patient information leaflet within the same Education Hub module. Clinical algorithms are integrated throughout and a comprehensive vestibular function test interpretation guide is provided in Section VII.

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, atypical presentations, and critical safety points requiring escalation or imaging.

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I. Introduction and Epidemiology

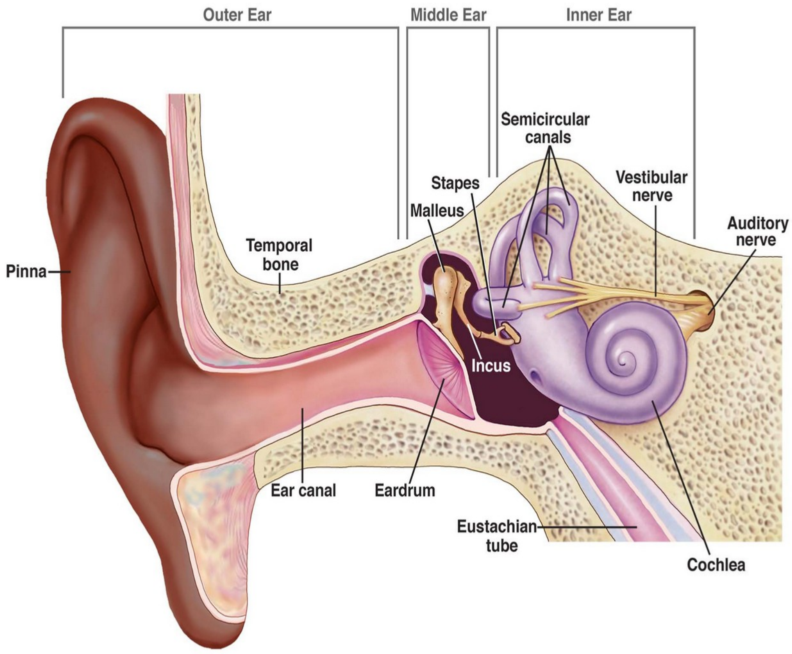

Figure 1. The inner ear (vestibular apparatus). The semicircular canals, utricle, and saccule constitute the peripheral vestibular organ susceptible to mechanical and metabolic injury following concussive head trauma.
Source: National Institute on Deafness and Other Communication Disorders (NIDCD/NIH), public domain. https://www.nidcd.nih.gov/news/multimedia/medical-illustration-parts-ear

Mild traumatic brain injury (mTBI) and concussion are among the most common neurological events encountered in clinical practice worldwide, with an estimated global incidence of 54–60 million episodes per year [1,2]. In Australia, mTBI accounts for approximately 100–300 hospitalisations per 100,000 population annually, with sport and recreation, road trauma, workplace injury, and falls in older adults representing the dominant mechanisms [3]. Dizziness or vestibular dysfunction is the second most reported symptom after headache, occurring in 50–80% of patients in the acute post-injury period and persisting beyond three months in 15–30% of cases — a syndrome variously termed post-concussion syndrome (PCS), post-concussive vestibular syndrome (PCVS), or post-traumatic dizziness [4,5,6].

The vestibular physician's role is distinct from that of the emergency clinician or general practitioner: where initial management focuses on exclusion of structural injury and safety-netting, the vestibular physician inherits a subgroup of patients with complex, multi-mechanism vestibular dysfunction that resists simple categorisation. These patients frequently carry unrecognised traumatic BPPV, labyrinthine concussion with partial VOR loss, subtle central vestibular pathway injury from diffuse axonal injury (DAI), cervicogenic dizziness, post-traumatic autonomic dysfunction, and developing PPPD — often simultaneously [7]. Each mechanism requires targeted assessment and specific management, and treating only the most prominent contributor leaves the patient only partially improved.

☐ **Key Point:** Post-concussion dizziness is not a single diagnosis. It is a clinical syndrome encompassing up to seven concurrent mechanisms, each requiring independent identification and targeted treatment. Failure to subtype accurately is the leading cause of prolonged disability in this population [4,8].

Epidemiology by Subtype

Table 1. Epidemiology and Prevalence of Post-Concussive Vestibular Subtypes

Subtype	Prevalence in Post-Concussive Cohorts	Time to Presentation	Key Risk Factors

Traumatic BPPV	20–30%	1 day – 6 weeks	Bilateral posterior canal; higher rate than idiopathic; older age
Labyrinthine concussion	10–15%	Immediate – days	High-velocity trauma; basilar skull fracture; blast exposure
Perilymph fistula (PLF)	5–10%	Immediate – weeks	Blunt trauma, barotrauma, Valsalva; oval window (blunt) vs round window (barotrauma)
Central vestibular / DAI	15–25%	Immediate	High-energy mechanism; loss of consciousness; CT-negative DAI
Post-traumatic VM	10–20%	Days – weeks	Pre-existing migraine predisposition; female sex; younger age
Post-traumatic PPPD	20–30% (of persistent PCS)	> 3 months	Anxiety, avoidant behaviour, prior vestibular disorder
Autonomic dysfunction (POTS-like)	10–30% in adolescents	Days – weeks	Adolescent female sex; pre-existing autonomic lability
Cervicogenic dizziness	15–25% (whiplash predominant)	Days – weeks	Whiplash mechanism; cervical soft tissue injury; suboccipital tenderness

The distinction between these subtypes has therapeutic and prognostic implications. Traumatic BPPV resolves rapidly with repositioning manoeuvres but recurs at higher rates than idiopathic BPPV — up to 40% within two years in some series [9,10]. Labyrinthine concussion may partially compensate over months with targeted vestibular rehabilitation but often leaves residual VOR gain asymmetry detectable on vHIT [11]. Post-traumatic PPPD can persist for years in the absence of specific management [12]. Accurate subtyping therefore determines not only initial management but also the expected trajectory of recovery and the need for specialist escalation.

II. Neurometabolic Cascade and Vestibular Pathophysiology

The mechanistic basis of concussion was substantially clarified by Hovda and colleagues in their seminal work on the neurometabolic cascade [13,14]. Following rapid acceleration-deceleration forces, non-specific neuronal depolarisation triggers a massive efflux of potassium and influx of sodium and calcium ions, with simultaneous release of excitatory amino acids including glutamate and aspartate. The resultant ionic disruption demands upregulated membrane pump activity, driving acute hyperglycolysis — a phase of increased cerebral glucose demand — paradoxically accompanied by reduced cerebral blood flow and impaired oxidative phosphorylation within the same timeframe [13,14].

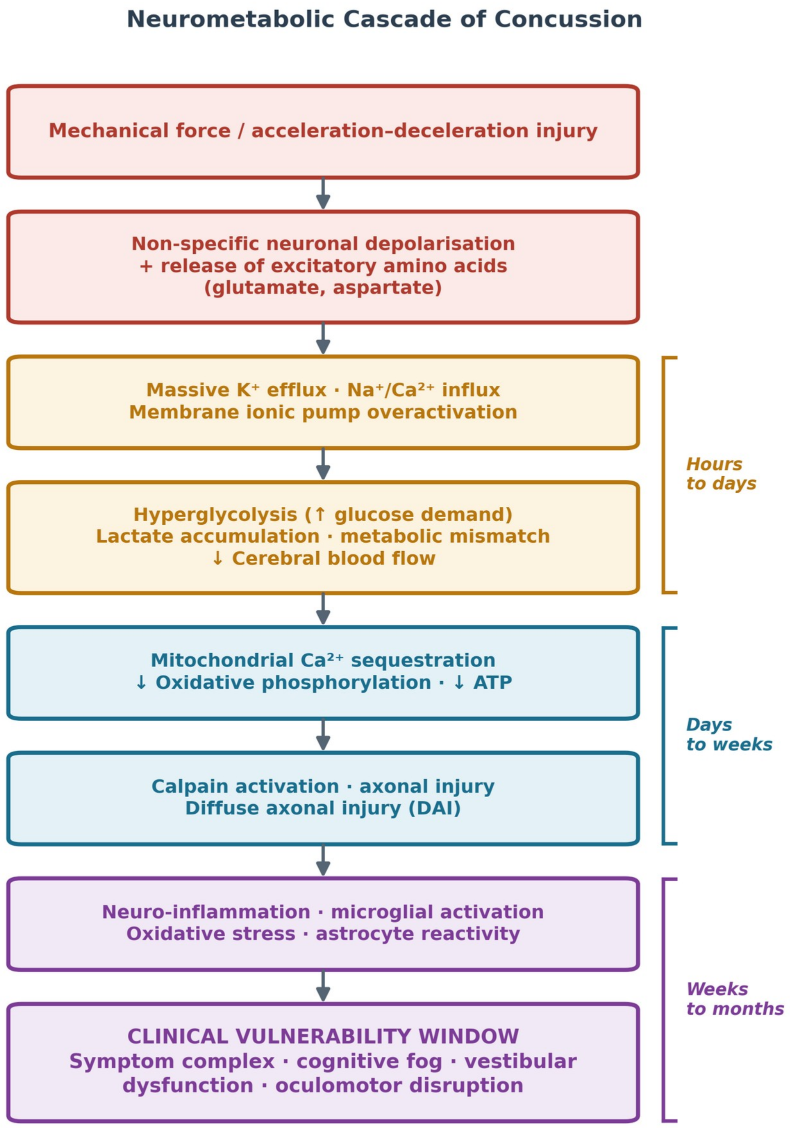

Figure 2. Neurometabolic cascade of concussion — from initial mechanical force to the clinical vulnerability window characterised by vestibular dysfunction, cognitive fog, and oculomotor disruption.

Source: Adapted from Giza and Hovda [13,14] and Barkhoudarian et al. [15].

The calcium influx phase, which can persist for 2–5 days in animal models and 2–4 weeks in human studies, is critical for the vestibular clinician [13]. Mitochondrial calcium sequestration impairs oxidative phosphorylation, reducing ATP availability in metabolically active vestibular hair cells, cochlear supporting cells, and vestibular nerve axons. The otolithic organs — utricle and saccule — are particularly susceptible to mechanical disruption, with otoconia displaced from the macula by the shear forces of the initial trauma, producing the substrate for traumatic BPPV [9,16]. Simultaneously, the endolymphatic fluid dynamics may be disrupted, generating a substrate for secondary endolymphatic hydrops in susceptible individuals [17,25].

Diffuse Axonal Injury and the Central Vestibular System

DAI represents a distinct and clinically underrecognised pathological substrate in mTBI. High-velocity rotational forces generate shear strains that exceed the tolerance of long white matter axons, particularly at sites of mechanical anisotropy — the junction between grey and white matter, the internal capsule, the brainstem tegmentum, and the superior cerebellar peduncles [18,19]. The vestibulo-ocular reflex (VOR) circuitry — encompassing the vestibular nuclei, the medial longitudinal fasciculus (MLF), the nucleus prepositus hypoglossi, and the cerebellum — traverses each of these DAI-vulnerable regions [20,21]. CT imaging is negative in the majority of mTBI cases with DAI; susceptibility-weighted MRI sequences (SWI) detect microhaemorrhages in the brainstem and corpus callosum in a significant minority, correlating with prolonged symptom duration [22].

□ **Clinical Insight:** Patients with central vestibular injury from DAI typically present with preserved vHIT VOR gain (VOR circuitry below the injury level is intact) but impaired smooth pursuit, saccadic hypometria, and bidirectional direction-changing nystagmus. This pattern — normal vHIT but abnormal HINTS exam — is the central fingerprint and mandates MRI with SWI sequences [22].

Neuro-Inflammation and the Prolonged Recovery Trajectory

Microglial activation and astrocyte reactivity following mTBI generate a prolonged neuroinflammatory state that contributes to the chronic symptom burden in post-concussion syndrome [23]. Pro-inflammatory cytokines including IL-1β, IL-6, and TNF-α have been measured in the CSF and peripheral blood of mTBI patients for weeks to months after injury [24]. Within the inner ear, inflammatory mediators may contribute to secondary endolymphatic hydrops and perilymph chemistry disruption, providing a mechanistic explanation for the late development of a Ménière-like syndrome in some post-traumatic patients [17,25].

III. Peripheral Vestibular Injury: Traumatic BPPV, Labyrinthine Concussion, and Perilymph Fistula

Traumatic BPPV

Traumatic BPPV is the most prevalent and most treatable cause of post-concussive dizziness, accounting for 20–30% of persistent vestibular symptoms in systematic series [9,10]. The mechanism is mechanical: acceleration-deceleration forces degrade the adhesive matrix holding otoconia to the utricular macula, releasing calcium carbonate crystals into the endolymphatic space, where they migrate into one or more semicircular canals [9,16]. Posterior canal variant predominates, but bilateral posterior canal involvement is more common after trauma — reported in 20–30% of traumatic BPPV series compared to < 10% in idiopathic cases [9]. Horizontal canal BPPV — both canalithiasis and cupulolithiasis variants — is similarly over-represented, and multi-canal BPPV (more than one canal affected) occurs in up to 15% of traumatic cases [10].

□ **Clinical Pearl:** Always perform both Dix-Hallpike (bilateral) and a supine roll test on the first assessment. A negative Dix-Hallpike does not exclude BPPV after trauma: horizontal canal and anterior canal variants, and bilaterally positive posterior canal disease, are over-represented and require the full positional screen to identify [9,10].

Treatment follows the same principles as idiopathic BPPV — Epley canalith repositioning procedure for posterior canal disease, Gufoni or barbecue roll manoeuvre for horizontal canalithiasis — but with the following caveats: multiple treatment sessions are more commonly required (mean 2.1 sessions vs 1.3 for idiopathic in one comparison cohort [10]); recurrence rates are elevated at approximately 40% within two years [9]; and the presence of co-existing central vestibular injury may modify positional nystagmus characteristics, making differentiation from central positional nystagmus challenging when atypical features are present — including sustained nystagmus, direction change on sitting up, or prominent instability [26].

Labyrinthine Concussion

Labyrinthine concussion refers to functional and structural injury to the membranous labyrinth occurring in the absence of temporal bone fracture, producing a spectrum from transient auditory and vestibular symptoms to permanent partial unilateral vestibular loss [27,28,29]. The mechanism involves the mechanical energy of the impact propagating through the fluid-filled otic capsule, generating hydraulic pressure waves that damage the metabolically active hair cells of the cristae ampullares and macular organs. The clinical presentation is an acute unilateral or bilateral peripheral vestibulopathy: continuous vertigo or severe imbalance from onset, often with accompanying sensorineural hearing loss and tinnitus on the affected side — distinguishing it from BPPV, which is purely positional [27,29].

Vestibular function testing reveals a characteristic pattern: reduced vHIT gain on the ipsilateral horizontal semicircular canal (with or without corrective saccades), reduced or absent ipsilateral cVEMP responses (reflecting saccular nerve involvement), reduced or absent ipsilateral oVEMP (utricular nerve), and asymmetric caloric responses confirming canal paresis on the affected side. Pure-tone audiometry typically shows a high-frequency sensorineural hearing loss on the affected side, reflecting concurrent cochlear hair cell injury [28,30]. The key prognostic distinction is between partial and complete loss: partial labyrinthine concussion carries a better prognosis for vestibular compensation with targeted gaze-stabilisation rehabilitation, whereas complete loss may leave permanent VOR asymmetry requiring long-term adaptation strategies [11,28].

Perilymph Fistula

Perilymph fistula (PLF) — an abnormal communication between the perilymph-filled inner ear and the air-filled middle ear cavity — is an underdiagnosed but clinically important cause of post-traumatic dizziness [31,32]. Even minor mTBI can produce sufficient pressure differential to rupture the oval window membrane, round window membrane, or both. The classic clinical triad is: pressure-dependent dizziness (worsened by straining, coughing, sneezing, lifting, or Valsalva manoeuvre); sensorineural or conductive hearing loss that may fluctuate; and tinnitus with a sense of aural fullness [31]. When blunt trauma is the mechanism, the oval window is the more commonly affected site; barotrauma characteristically affects the round window [32].

> ☐ **Important:** Pneumolabyrinth — air within the cochlea or vestibule — is pathognomonic of perilymph fistula and may be identified on high-resolution CT temporal bone. Its presence confirms the diagnosis without the need for exploratory tympanotomy. In the absence of pneumolabyrinth, diagnosis relies on the clinical history, pressure-provoked nystagmus on the platform pressure test or Valsalva test, and intraoperative perilymph flow observation [31,33].

Initial management of suspected PLF is conservative: strict physical rest with avoidance of straining, head elevation, and stool softeners for 7–14 days [33]. Carbonic anhydrase inhibitors (acetazolamide) and thiazide diuretics are used empirically in cases with a mixed PLF/hydrops presentation. Surgical exploration with fat plug or perichondrial patch repair of the oval and round window niches is reserved for patients who fail conservative management at 6–8 weeks with ongoing significant vestibular and auditory symptoms. Intraoperative confirmation of active perilymph flow remains the gold standard for diagnosis [32,33].

IV. Central Vestibular Injury: Diffuse Axonal Injury, Oculomotor Pathways, and Brainstem

Central vestibular injury is the mechanistically complex end of the post-concussive spectrum. Unlike peripheral labyrinthine injury — where the structural lesion is contained within the temporal bone — central injury involves the distributed neural circuitry of the brainstem, cerebellum, thalamus, and cortical multisensory integration areas that process vestibular input and generate coherent spatial orientation [18,20,21]. The protean manifestations of central vestibular injury — from oculomotor dysfunction and dynamic visual acuity loss to postural instability and cognitive-vestibular dissociation — reflect the anatomical breadth of this circuitry and its vulnerability to DAI [19,21].

Oculomotor Dysfunction

Oculomotor dysfunction is one of the highest-yield and most functionally disabling findings in post-concussive vestibular assessment. The supranuclear oculomotor pathways — spanning the frontal eye fields, posterior parietal cortex, superior colliculus, paramedian pontine reticular formation (PPRF), and medial longitudinal fasciculus (MLF) — are disrupted in a substantial proportion of patients with mTBI, often independently of any peripheral vestibular injury [34,35]. The VOMS battery provides a validated bedside screen: impairment in smooth pursuit, saccades, VOR cancellation, or near-point convergence each localises to distinct anatomical substrates and predicts prolonged recovery [36,37].

Convergence insufficiency — the inability to maintain binocular fusion at near working distance — is the most common isolated oculomotor finding, occurring in up to 40% of post-concussive patients and generating symptoms of diplopia, headache, and visual fatigue that are often attributed to screen use rather than vestibular pathology [35,38]. The brainstem convergence centre (midbrain periaqueductal grey and nucleus of Edinger-Westphal) is particularly susceptible to contusive injury. Impaired smooth pursuit — characterised by a catch-up saccadic pattern disrupting smooth sinusoidal tracking — localises the lesion to the dorsomedial pontine tegmentum, the flocculus, or the posterior parietal cortex [35,39].

□ **Clinical Insight:** Dynamic visual acuity (DVA) testing — measuring visual acuity during horizontal head oscillation at 2 Hz compared to static acuity — is a functional measure of VOR gain under ecologically valid conditions. A DVA decrement of two or more LogMAR lines is highly specific for unilateral vestibular hypofunction and corroborates vHIT gain reduction in labyrinthine concussion. In central injury, DVA may be preserved despite subjective visual motion sensitivity, reflecting the central-peripheral dissociation in this population [40].

Cerebellar and Brainstem Vestibular Pathways

The cerebellar flocculus and nodulus play a critical role in VOR gain calibration and gaze-holding. Contusive injury to the posterior fossa — or DAI disrupting the superior cerebellar peduncle input to the flocculus — produces a characteristic triad of preserved VOR gain on vHIT, impaired VOR cancellation (the cerebellar brake on the VOR is lost), and downbeat or torsional nystagmus in primary gaze — the central nystagmus fingerprint requiring MRI evaluation [22,41]. Gaze-evoked nystagmus (nystagmus intensifying on eccentric gaze) with Alexander's law behaviour is a further brainstem/cerebellar sign, distinct from peripheral spontaneous nystagmus which shows inhibition on visual fixation [26,41].

HINTS examination (Head-Impulse, Nystagmus, Test of Skew) — validated for distinguishing vestibular neuritis from posterior circulation stroke in the acute vestibular syndrome — applies directly to the post-concussive patient with acute spontaneous vertigo: a normal vHIT, direction-changing nystagmus, or skew deviation in a post-traumatic patient mandates urgent MRI with DWI to exclude posterior fossa haematoma, cerebellar contusion, vertebrobasilar dissection, or posterior fossa infarction before the presentation is attributed to a benign concussive mechanism [22,42].

V. Autonomic Dysfunction, Cervicogenic Dizziness, and Post-Traumatic Endolymphatic Hydrops

Post-Traumatic Autonomic Dysfunction

Dysautonomia following mTBI — ranging from subtle heart rate variability abnormalities to frank postural orthostatic tachycardia syndrome (POTS) — is increasingly recognised as a distinct mechanistic contributor to post-concussive dizziness, particularly in adolescents and young adults [43,44]. The medullary cardiovascular control centres, the insular cortex, and the hypothalamic-brainstem autonomic axis are disrupted by both DAI and the neuroinflammatory sequelae of mTBI. Clinical manifestations include lightheadedness on standing (orthostatic intolerance), exercise intolerance, palpitations, excessive fatigue, and cognitive fog — the POTS-like syndrome that overlaps significantly with the vestibular and cognitive trajectories of post-concussion syndrome [44,45].

Orthostatic vital signs — seated and standing heart rate and blood pressure at 1, 3, and 5 minutes — are the minimum evaluation. Tilt-table testing is the gold standard for formal dysautonomia quantification. The Buffalo Concussion Treadmill Test (BCTT), originally developed to identify exercise intolerance predicting prolonged recovery, operationalises autonomic threshold testing in a clinical setting: a symptom-exacerbation heart rate significantly below age-predicted maximum is a reliable indicator of autonomic dysregulation and guides sub-symptom-threshold aerobic exercise prescription, now endorsed as a core management strategy in the 6th International Consensus on Concussion in Sport [45,46].

Cervicogenic Dizziness

Cervicogenic dizziness — dizziness arising from dysfunctional afferent input from the cervical proprioceptive system — is common after concussive trauma involving the head and neck, and is particularly prominent in whiplash-mechanism injuries [47,48]. The cervical dorsal root ganglia (C1–C3), suboccipital muscles, and facet joint mechanoreceptors contribute proprioceptive signals to the vestibular nuclei via the medial vestibulospinal tract, integrating with semicircular canal and otolith input to produce coherent spatial orientation. Disruption of this afferent stream produces a mismatch between expected and received proprioceptive input, perceived as dizziness or disequilibrium [47].

☐ **Clinical Pearl:** Cervicogenic dizziness is a diagnosis requiring positive confirmation, not merely exclusion of other causes. The cervical relocation test (the patient attempts to return their head to neutral from rotation with eyes closed, and repositioning error is measured), the smooth pursuit neck torsion test (smooth pursuit measured with and without trunk rotation relative to the neck), and symptomatic improvement with targeted cervical physiotherapy together constitute the most clinically accepted confirmatory approach [47,48].

Post-Traumatic Secondary Endolymphatic Hydrops

A subset of patients who sustain labyrinthine concussion, temporal bone trauma, or perilymph fistula subsequently develop a Ménière-like syndrome characterised by episodic vertigo, fluctuating low-frequency sensorineural hearing loss, tinnitus, and aural fullness — post-traumatic secondary endolymphatic hydrops [17,25,49]. The pathophysiological mechanism involves disruption of endolymphatic homeostasis through otoconia displacement obstructing the endolymphatic duct, labyrinthine membrane disruption allowing perilymph-endolymph mixing, or neuroinflammatory damage to the ion-transporting cells of the endolymphatic sac [17,50]. MRI with gadolinium using inversion recovery sequences can visualise endolymphatic hydrops directly in a tertiary clinical setting, providing objective confirmation of hydrops distribution [51].

Management follows the principles of idiopathic Ménière's disease: low-sodium diet, vestibular suppressants restricted to acute attacks, and preventive pharmacotherapy (betahistine, thiazide diuretics, or acetazolamide) for frequent episodic disease. Post-traumatic hydrops may stabilise with conservative management in a higher proportion of patients than idiopathic Ménière's disease, though the evidence base for this distinction is limited to case series [49]. Intratympanic dexamethasone or gentamicin ablation follow the same decision thresholds as idiopathic Ménière's disease where conservative measures fail [50].

VI. PPPD After Concussion and Post-Traumatic Vestibular Migraine

PPPD After Concussion: Diagnostic Criteria and Pathophysiology

Persistent Postural-Perceptual Dizziness (PPPD) is the most common cause of chronic dizziness lasting more than three months after an acute vestibular precipitant of any type, including mTBI [12,52]. The Bárány Society diagnostic criteria (2017) define PPPD as: dizziness, unsteadiness, or non-spinning vertigo present on most days for three or more months; symptoms exacerbated by upright posture, active or passive motion, or exposure to complex or moving visual stimuli; onset following a precipitating vestibular, neurological, or anxiety-inducing event; significant functional impairment; and symptoms not better explained by another active disorder [52]. In the post-concussive context, the precipitating event is the head injury itself, and the early post-injury symptom burden creates the ideal neurological and psychological substrate for PPPD to develop [12,53].

Table 2. Bárány Society Diagnostic Criteria for PPPD (2017) with Post-Concussive Annotations

Bárány Criterion	Requirement	Post-Concussive Annotation
A. Symptom character	Dizziness, unsteadiness, or non-spinning vertigo on > 50% of days for 3 months or more	Head injury is a valid precipitating event; onset may be delayed 1–4 weeks as acute symptoms abate
B. Provocation	Worsened by: upright posture; active or passive motion; complex / moving	Screen time, busy environments, and driving are typical post-concussive

	visual stimuli	PPPD triggers
C. Triggering event	Onset after precipitating event: vestibular disorder, acute illness, neurological event, TBI, or anxiety	TBI is explicitly listed; document mechanism and timing of injury
D. Functional impairment	Significant distress or functional disability	Use Dizziness Handicap Inventory (DHI): moderate 36–59; severe 60 or above
E. Exclusion	Not better explained by another active disorder; other diagnoses may coexist	Coexisting traumatic BPPV or PT-VM does not exclude PPPD — treat concurrently

The neurobiological model of PPPD centres on maladaptive cortical recalibration of multisensory integration following the acute vestibular event [12,53]. Under normal conditions, the brain weights sensory inputs — visual, vestibular, and somatosensory — dynamically according to their reliability. After a vestibular precipitant, upweighting of visual input ("visual dependency") and increased cognitive monitoring of balance (high-gain postural control) produce a persistent state of vestibular hypersensitivity that perpetuates symptoms beyond structural recovery [52,53]. This model explains the characteristic visual motion sensitivity and the paradox of normal vestibular function testing in a patient with severe subjective dizziness.

☐ **Key Point:** PPPD and post-concussive structural vestibular injury are not mutually exclusive. Up to 40% of post-concussive PPPD patients have coexisting traumatic BPPV, partial labyrinthine concussion, or post-traumatic VM. Both diagnoses must be identified and treated concurrently; treating only the PPPD while leaving untreated BPPV or VM sustains the perpetuating precipitant [12,53].

Post-Traumatic Vestibular Migraine

Head injury is a potent precipitant of vestibular migraine (VM) in individuals with migrainous predisposition, and VM is the second most common cause of persistent post-concussive dizziness after BPPV [54,55]. The mechanism likely involves trigeminovascular sensitisation, cortical spreading depression, and disruption of the descending serotonergic pain-modulation pathways — all of which may be initiated or amplified by the neuroinflammatory and metabolic sequelae of mTBI [56]. Patients with a prior personal or family history of migraine are at significantly elevated risk, and the temporal pattern — acute concussive symptoms gradually transitioning to episodic vestibular migraine attacks weeks to months after injury — is characteristic [54].

The Bárány Society / International Headache Society diagnostic criteria for VM (2022 update) require: at least five episodes of vestibular symptoms of moderate or severe intensity lasting 5 minutes to 72 hours; a current or prior history of migraine with headache; one or more migraine features in at least 50% of episodes (headache, photophobia, phonophobia, or visual aura); and symptoms not better explained by another disorder [57]. In the post-concussive context, distinguishing post-traumatic headache (a direct consequence of the injury) from post-traumatic VM (a precipitated migraine disorder) requires careful temporal tracking: the headache of mTBI typically begins within 24 hours and follows a near-continuous pattern; VM attacks are episodic, triggered, and separated by symptom-free intervals [54,55].

Management of post-traumatic VM follows standard VM principles: lifestyle modification, acute treatment with triptans or NSAIDs for moderate-to-severe attacks, and preventive pharmacotherapy when attacks occur more than once monthly or are functionally disabling. First-line preventives with the strongest evidence include topiramate, propranolol, valproate, and amitriptyline [57,58]. The CGRP monoclonal antibodies (erenumab, fremanezumab, galcanezumab) are second-line agents with growing evidence in VM specifically [59]. Vestibular rehabilitation as an adjunct to pharmacotherapy improves functional outcomes in VM compared to either alone [57,58].

VII. History, Examination, and Vestibular Function Testing

History

A systematic history is the foundation of subtype classification in post-concussive vestibular assessment. The history must establish: mechanism and timing of injury (velocity, rotational component, direct vs indirect impact); loss of consciousness or post-traumatic amnesia (markers of injury severity); the temporal evolution of symptoms from the day of injury to the present; the precise character of dizziness (vertigo, disequilibrium, presyncope, or visual motion sensitivity); provocation profile (positional, exertional, orthostatic, visual); associated auditory symptoms (hearing loss, tinnitus, aural fullness) and their laterality; prior history of migraine, motion sickness, or vestibular disorder; and medication history including vestibular suppressants that may impede compensation [4,7].

Bedside Vestibular Examination

The examination of the post-concussive patient must be structured and systematic. Spontaneous nystagmus is assessed in primary gaze, lateral gaze, and vertical gaze with and without visual fixation (Frenzel goggles or video-oculography preferred). Direction-changing nystagmus, nystagmus enhanced by fixation removal, downbeat nystagmus, or torsional nystagmus in primary gaze all indicate central pathology. Smooth pursuit is assessed using a 0.4 Hz pendulum target: saccadic intrusions at any velocity warrant documentation. The HIT is performed bedside, but video head impulse testing (vHIT) provides quantitative gain and precise saccade identification superior to the clinical manoeuvre, particularly for low-gain losses in partial labyrinthine concussion [42,60].

Positional testing — bilateral Dix-Hallpike and supine roll test — is mandatory in every post-concussive vestibular assessment regardless of the presenting complaint, given the 20–30% prevalence of coexisting traumatic BPPV [9]. Skew deviation (vertical ocular misalignment on alternate cover test) is a HINTS-positive finding that localises to the brainstem otolithic pathways and mandates MRI. The VOMS battery provides rapid bedside quantification of oculomotor and vestibulo-ocular dysfunction, with any domain score rise of 2 or more out of 10 on a provocation visual analogue scale being clinically significant and predictive of prolonged recovery [36,37].

Table 3. Red Flags in Post-Concussive Vestibular Assessment Requiring Urgent Imaging

Red Flag Feature	Concern	Urgent Action
New focal neurological signs (weakness, dysarthria, dysphagia)	Structural intracranial lesion; brainstem injury	Emergency CT/MRI head; neurosurgery
Progressive or severe headache unresponsive to analgesia	Intracranial haemorrhage; cerebral venous thrombosis	Emergency CT head
Gait ataxia disproportionate to reported vertigo	Cerebellar contusion or haematoma	MRI with SWI; cerebellar stroke protocol
Direction-changing nystagmus or downbeat nystagmus in primary gaze	Central vestibular lesion; posterior fossa pathology	MRI with FLAIR and SWI
HINTS positive (normal vHIT + abnormal nystagmus or skew)	Posterior circulation stroke or structural brainstem lesion	MRI DWI within 24 hours
Unilateral sudden sensorineural hearing loss post-trauma	Temporal bone fracture; labyrinthine artery infarction	CT temporal bone; urgent audiometry and ENT
Loss of consciousness > 30 minutes or post-traumatic amnesia > 24 hours	Exceeds mTBI definition; moderate TBI; risk of intracranial complication	CT head; neurology admission assessment

Vestibular Function Test Battery

The vestibular physician's toolkit extends significantly beyond bedside examination. The full functional assessment of a complex post-concussive patient typically includes video head impulse testing (vHIT), cervical and ocular vestibular evoked myogenic potentials (cVEMP and oVEMP), videonystagmography (VNG) with bithermal caloric irrigation, computerised dynamic posturography (CDP) or the modified clinical test of sensory interaction on balance (mCTSIB), pure-tone audiometry and speech discrimination, and in selected cases electrocochleography (ECoG) for endolymphatic hydrops assessment [60,61,62].

Table 4. vHIT and VEMP Interpretation by Post-Concussive Vestibular Subtype

Subtype	vHIT Gain	Corrective Saccades	cVEMP	oVEMP	Caloric	Interpretation
Traumatic BPPV	Normal	Absent	Normal	Normal	Normal	Semicircular canal function preserved; otolith mechanically displaced
Labyrinthine concussion	Low (affected side)	Present (covert)	Reduced or absent	Reduced	Reduced unilateral	Combined SCC + otolith dysfunction; matches sensorineural hearing loss
Perilymph fistula	Variable	May be present	May be affected	Variable	Normal / slightly ↓	Pressure-provoked nystagmus on Valsalva; fistula test positive
Central (DAI / brainstem)	Normal (gain preserved)	Covert saccades rare	Normal	Normal	Symmetric	HINTS: smooth pursuit impaired; saccades bidirectional; HINTS-positive
PPPD / post-TBI PPPD	Normal	Absent	Normal	Normal	Normal	VFT entirely normal; diagnosis of exclusion; Bárány criteria must be met

cVEMP = cervical vestibular evoked myogenic potential; oVEMP = ocular VEMP; SCC = semicircular canal; DAI = diffuse axonal injury; PPPD = persistent postural-perceptual dizziness.

Source: Adapted from Agrawal et al. [60], Bartl et al. [61], and Schubert et al. [62].

Table 5. Vestibular Function Test Battery in Post-Concussive Assessment

Test	What It Assesses	Normal Range	Key Findings in mTBI
vHIT (horizontal)	Horizontal SCC — high-freq VOR gain	Gain 0.80–1.20; no corrective saccades	Reduced gain + covert saccades in labyrinthine concussion; preserved in central DAI
vHIT (vertical: LARP/RALP)	Anterior / posterior SCC — vertical VOR	Gain 0.75–1.20 per axis	Reduced in temporal bone fracture; preserved in PPPD
cVEMP (click / tone burst)	Saccule via inferior vestibular nerve	Symmetric amplitude; threshold below 100 dBnHL	Reduced or absent ipsilateral in labyrinthine concussion and PLF
oVEMP (bone conductor)	Utricle via superior vestibular nerve	Symmetric N10 amplitude	Reduced ipsilateral in labyrinthine concussion; may be normal in BPPV
Bithermal caloric	Horizontal SCC low-frequency response	Unilateral weakness below 25%	Unilateral canal paresis in labyrinthine concussion; may be

			normal in central DAI
CDP / mCTSIB	Sensory integration: visual / proprioceptive / vestibular	Condition 5 and 6 within age norms	Vestibular condition failure in peripheral lesions; visual condition failure in PPPD and central injury
Pure-tone audiometry	Cochlear function	Thresholds at or below 25 dBHL; symmetric	High-freq SNHL ipsilateral in labyrinthine concussion; conductive component in ossicular injury
ECoG	Endolymphatic pressure (SP/AP ratio)	SP/AP ratio below 0.45	Elevated SP/AP in post-traumatic hydrops

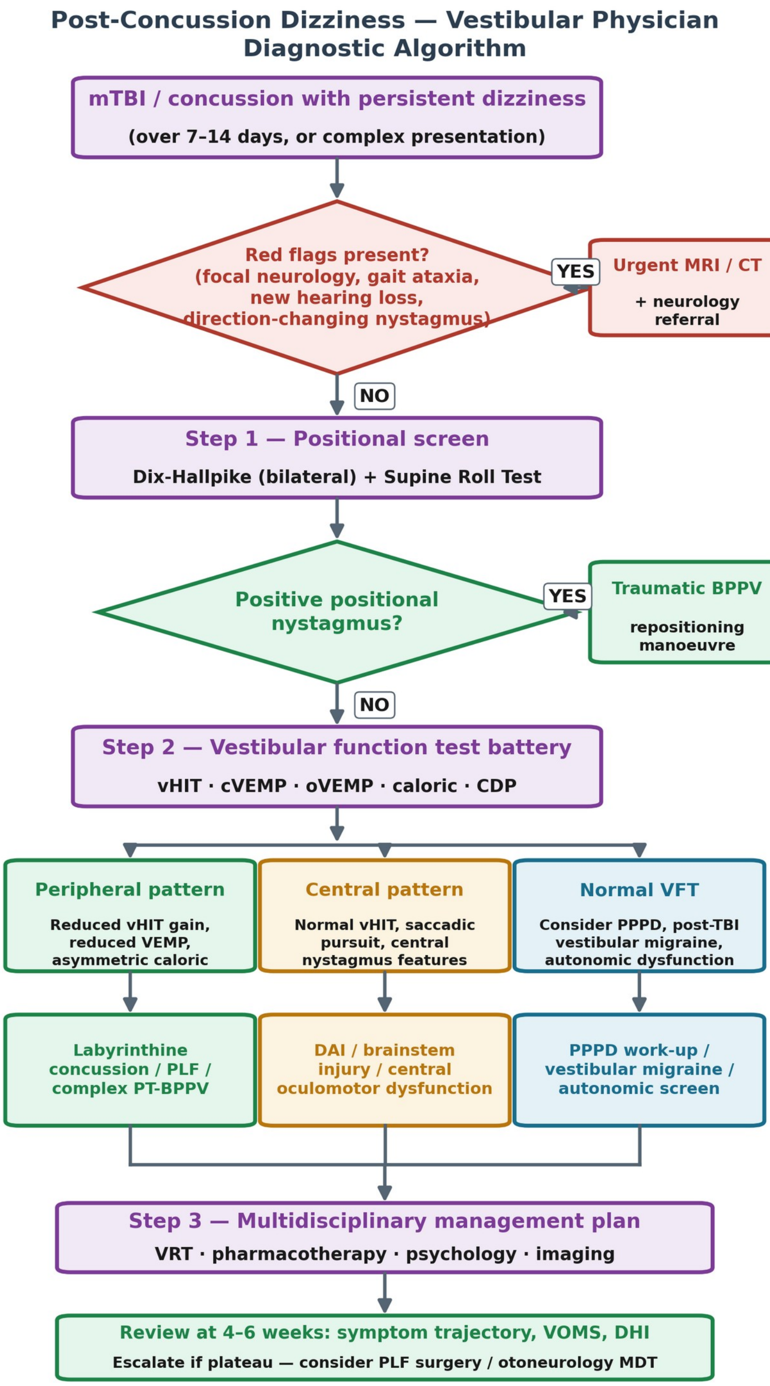

Figure 3. Post-Concussion Dizziness diagnostic algorithm for the vestibular physician. VFT = vestibular function testing; PLF = perilymph fistula; PPPD = persistent postural-perceptual dizziness; DAI = diffuse axonal injury.

Source: Adapted from Lan and Hoffer [7], Marcus et al. [63], and Patricios et al. [46].

VIII. Management: Peripheral Lesions, Central Rehabilitation, and Pharmacotherapy

Peripheral Vestibular Management

Traumatic BPPV responds to the standard canalith repositioning procedures as outlined in Section III, but the treating vestibular physician must anticipate: bilateral or multi-canal BPPV requiring sequential treatment within the same session; canal conversion (debris transiting from one canal to another during repositioning, most commonly posterior-to-horizontal canal in the Epley manoeuvre); and a higher burden of residual dizziness and oscillopsia following successful repositioning, reflecting co-existing central sensitisation and perceptual dysregulation that characterises post-concussive dizziness even after the mechanical BPPV is resolved [9,10].

Labyrinthine concussion is managed with directed vestibular rehabilitation targeting the specific functional loss identified on VFT. Partial unilateral vestibular loss requires gaze-stabilisation exercises (VOR times 1 and times 2 exercises in horizontal and vertical planes), dynamic visual acuity training, and progressive balance challenge on compliant surfaces and in dynamic environments. The principle of habituation (progressive exposure to provocative stimuli) is combined with adaptation (activities designed to elicit VOR gain recalibration by the cerebellum). Early initiation — within days of injury where tolerated — accelerates compensation and reduces the risk of PPPD superimposition [11,64].

Central Rehabilitation and Sub-Symptom-Threshold Exercise

For patients with central vestibular pathway injury or oculomotor dysfunction, the rehabilitation programme is tailored to the specific deficits identified. Convergence insufficiency responds to targeted exercises including pencil push-ups, Brock string exercises, and prism-based vergence training [35,38]. Smooth pursuit impairment is addressed with smooth pursuit training — visual targets moving sinusoidally or randomly in horizontal, vertical, and diagonal planes — while saccadic exercises address accuracy and velocity [35]. Dual-task cognitive-vestibular exercises (performing arithmetic simultaneously with gaze stabilisation or balance training) address the specific cognitive-vestibular integration deficits prevalent in central post-concussive injury [65].

Sub-symptom-threshold aerobic exercise — formalised by the Buffalo Concussion Treadmill Test protocol — is now endorsed in international consensus guidelines as a core management strategy for post-concussive syndrome regardless of vestibular subtype [45,46,64]. The target intensity is 80–85% of the BCTT symptom-exacerbation threshold heart rate. The dose-response relationship is reproducible: patients training consistently at or just below the threshold show the fastest recovery on serial BCTT and the lowest rate of symptom recurrence during exercise progression [45,66]. Vestibular suppressants impede cerebellar-mediated VOR compensation and should be restricted to acute symptomatic relief during the first 24–72 hours; continued use beyond this window delays functional recovery [7,67].

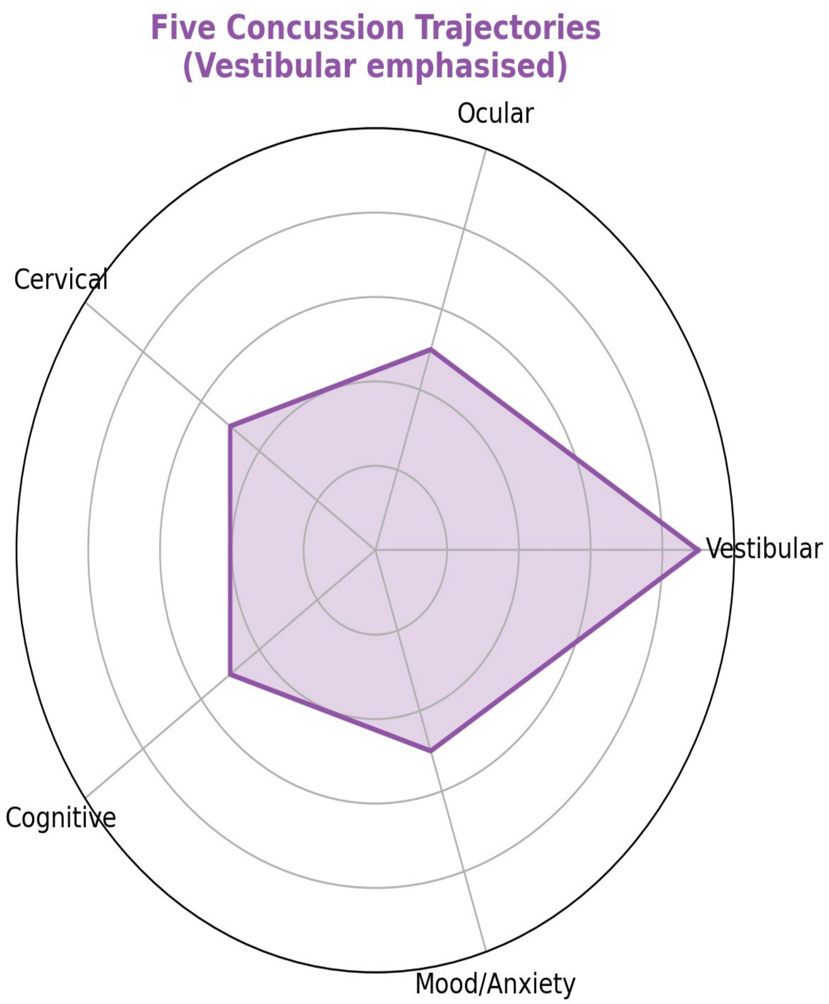

Figure 4. Five concussion trajectories — vestibular, ocular, cervical, cognitive, and mood/anxiety — each with distinct anatomical substrates and rehabilitation priorities. Most post-concussive patients have one dominant trajectory with one or two secondary contributors.

Source: Adapted from Ellis, Leddy, and Willer [68] and Patricios et al. [46].

Pharmacotherapy

Table 6. Pharmacotherapy in Post-Concussive Vestibular Management

Indication	Agent(s)	Dose / Regime	Evidence / Notes
Acute vestibular suppression (first 24–72 hours only)	Prochlorperazine Promethazine Meclizine	5–10 mg q6–8h 25 mg BD 25–100 mg/day	Short-term only; avoid beyond 48–72 h — impedes VOR compensation
Post-traumatic VM — acute	Sumatriptan (or other triptans) NSAIDs (ibuprofen 400–600 mg)	50–100 mg oral or 6 mg SC PRN with attacks	Triptans contraindicated in hemiplegic migraine and recent vascular events
Post-traumatic VM — prevention	Propranolol Topiramate Valproate Amitriptyline	40–160 mg/day 50–200 mg/day 400–1000 mg/day 10–75 mg nocte	Level A evidence for headache prevention; choose based on comorbidities
Post-traumatic VM — CGRP pathway	Erenumab Fremanezumab Galcanezumab	Monthly or quarterly SC injection	2nd line; TGA-approved for migraine prevention; growing VM-specific evidence [59]

PPPD	Sertraline Escitalopram Venlafaxine	50–200 mg/day 10–20 mg/day 37.5–150 mg/day (SNRI)	Serotonin receptors in vestibular pathways; combine with VRT and CBT; minimum 3–6 months treatment [52,53]
Post-traumatic secondary hydrops	Betahistine Hydrochlorothiazide Acetazolamide	16–48 mg TID 25–50 mg/day 250 mg BD–TID	Evidence largely from idiopathic Menieres disease; dietary sodium below 2 g/day first-line adjunct
PLF conservative management	Acetazolamide Sodium restriction	250 mg BD; dietary below 2 g/day	Reduces endolymphatic pressure empirically; evidence is case-series level only [32,33]
Post-traumatic POTS	Fludrocortisone Midodrine Propranolol (low dose)	0.1 mg/day 2.5–10 mg TID 10–20 mg BD	Combined with volume expansion (salt and fluid loading) and compression garments; first-line is exercise rehabilitation

IX. Refractory Disease, Special Populations, and Medicolegal Considerations

Refractory Post-Concussive Vestibular Syndrome

Persistent symptoms beyond six months despite targeted management constitute refractory post-concussive vestibular syndrome and warrant multidisciplinary review. The most common reasons for treatment resistance are: inadequate subtype classification (missed PLF, unrecognised post-traumatic VM, or undertreated PPPD); inadequate rehabilitation duration or intensity (evidence supports continued VRT for 3–6 months rather than short courses); untreated psychiatric comorbidity (anxiety and depression amplify PPPD and VM and predict poor outcome without specific psychological intervention); and persistent structural abnormality requiring surgical consideration (PLF with failed conservative management; severe temporal bone fracture with ossicular chain disruption) [12,64,67].

The role of intratympanic interventions in post-traumatic vestibular disease is evolving. Intratympanic dexamethasone has been used in post-traumatic secondary hydrops with the same rationale as idiopathic Meniere's disease — anti-inflammatory and osmotic stabilisation of the endolymphatic system — though controlled evidence in the post-traumatic cohort specifically is absent. Intratympanic gentamicin ablation remains a last resort in post-traumatic hydrops with intractable vertigo and useful hearing, applying the same thresholds as in idiopathic Meniere's disease [50,67].

Paediatric and Adolescent Populations

Children and adolescents represent a distinct post-concussive population with several clinically important differences from adults [69,70]. The developing vestibular and oculomotor systems may be more vulnerable to concussive injury, and recovery trajectories are consistently longer — paediatric sport-related concussion studies report median recovery times of 4–7 weeks compared to 2–4 weeks in adults [69]. Oculomotor dysfunction is over-represented, reflecting the developmental stage of the convergence-accommodation system, and has particular functional impact for school reintegration where near-vision reading tasks are ubiquitous [38,70]. Sub-symptom-threshold aerobic exercise rehabilitation is safe and effective in children aged 12 and above, with the BCTT protocol validated in paediatric sport-related concussion cohorts [66].

Post-traumatic PPPD in the paediatric population may develop rapidly following a single concussive event in adolescents with pre-existing anxiety, somatic symptom amplification, or school avoidance patterns. Early psychological screening and early referral to a paediatric clinical psychologist experienced in health anxiety and somatic disorders is a critical component of management, as untreated psychological perpetuating factors dramatically extend recovery duration in this age group [12,70].

Older Adults

Older adults present a mechanistically distinct post-concussive profile. Falls are the dominant mechanism, and the pre-existing vestibular deficits of age-related peripheral vestibular loss (presbyvestibulopathy), reduced visual acuity, and impaired proprioception compound the post-concussive vestibular dysfunction. The cervical spine is frequently involved in fall-mechanism injuries. A pragmatic approach — assess and treat all identified vestibular mechanisms regardless of attribution confidence — is appropriate, where falls risk is the primary safety concern [3,71].

Blast and Military mTBI

Blast-related mTBI is the signature injury of modern combat, generating a distinct pathophysiological substrate through primary blast wave, secondary blast (fragmentation), tertiary (body displacement), and quaternary (thermal and chemical) mechanisms. The inner ear is exquisitely sensitive to primary blast pressure waves: the cochlea suffers acoustic trauma, the labyrinthine membranes may rupture producing PLF, and temporal bones may fracture even without direct head contact [72,73]. Bilateral involvement is common. Blast-related mTBI also carries a disproportionate burden of PTSD, which interacts with post-traumatic PPPD and vestibular migraine in ways that complicate both diagnosis and treatment [73].

Medicolegal Considerations

Post-concussive vestibular presentations are among the most frequently encountered conditions in medicolegal practice, particularly in road trauma, workers' compensation, and sport injury litigation. The vestibular physician providing medicolegal opinion must adhere to the following standards: diagnosis must be based on objective vestibular function testing rather than symptom report alone; each identified subtype must be characterised by its specific test result signature (abnormal vHIT gain, cVEMP asymmetry, canal paresis percentage, ECoG SP/AP ratio); the causal link between the documented mechanism of injury and the identified vestibular lesion must be argued on the balance of probabilities with reference to the peer-reviewed literature; and the degree of functional impairment must be quantified using validated instruments (DHI, VOMS scores, DVA decrement) [7,74].

☐ **Clinical Insight:** A comprehensive vestibular physician report for medicolegal purposes should include: the mechanism of injury; timeline from injury to symptom onset; prior vestibular history; examination findings (nystagmus characteristics, HINTS result, VOMS score, positional test results); VFT battery results with normative data; diagnosis by Barany Society or equivalent criteria; prognosis with and without treatment; and the attributable fraction of the current vestibular impairment to the index injury. The absence of objective VFT abnormality does not exclude PPPD, which is a diagnosis of defined clinical criteria not requiring abnormal VFT [7,52,74].

X. Guidelines, Controversies, and Future Directions

Current Guidelines

The 6th International Consensus Statement on Concussion in Sport (Amsterdam, 2022) endorses: brief relative rest in the first 24–48 hours; early sub-symptom-threshold aerobic exercise commencing within the first week where tolerated; vestibular physiotherapy referral by 7–10 days for persistent dizziness; and a structured, symptom-limited return-to-sport and return-to-work protocol [46]. The Ontario Neurotrauma Foundation's concussion guidelines provide complementary evidence-based recommendations covering paediatric populations, occupational concussion, and pharmacotherapy in persistent cases [75]. Australian-specific guidance — including the Australian Institute of Sport's Brain

Health Position Statement (2023) — aligns with the CISG framework and provides specific thresholds for return to contact sport [76].

Table 7. Guideline Recommendations Relevant to Vestibular Physician Management of Post-Concussion

Recommendation Domain	Guideline / Source	Evidence Grade	Vestibular Physician Implication
Early aerobic exercise (sub-symptom threshold from day 2–7)	CISG 6th Consensus 2022 [46]; ONF 2018 [75]	Level 1 (RCT evidence)	Prescribe BCTT-guided exercise; document threshold HR
Vestibular physiotherapy by 7–10 days if dizziness persists	CISG 6th Consensus 2022 [46]	Consensus recommendation	Vestibular physician assessment concurrent with or directing VRT
Avoid prolonged rest beyond 24–48 hours	CISG 2022; ONF 2018 [46,75]	Level 1	Counsel patients against activity avoidance; document advice
BPPV: repositioning manoeuvres (Epley for posterior canal)	Evidence-based systematic review	Level 1	Perform at first visit; document result and follow-up plan
PPPD: SSRIs / SNRIs + VRT + CBT	Barany Society PPPD consensus [52]	Level 2–3	Initiate SSRI early in confirmed PPPD; refer psychology
VM prevention pharmacotherapy	IHS / Barany 2022 [57]	Level 1 (headache; extrapolated to VM)	Select preventive based on comorbidities; reassess at 3 months
Return-to-contact-sport clearance	CISG 2022; AIS 2023 [46,76]	Consensus	Clearance should include documented VFT normalisation
Avoid vestibular suppressants beyond 72 hours	Multiple vestibular society guidelines [7,67]	Level 2	Document cessation and patient counselling in the record

Controversies

Several areas of active controversy are relevant to the vestibular physician. First, the diagnostic validity of perilymph fistula remains contested: the absence of a non-invasive confirmatory test, historically wide variation in reported prevalence, and the difficulty of distinguishing PLF-related symptoms from labyrinthine concussion mean that PLF is both over- and under-diagnosed in different clinical environments [31,32]. The development of proteomics-based perilymph biomarkers (PROM1, OTOS, cochlin) detectable in middle ear fluid holds promise as a non-invasive diagnostic test but is not yet clinically available [77].

Second, the appropriate threshold for sport return after concussion — and the role of vestibular physician clearance as a gatekeeping function — remains debated. Some jurisdictions now mandate return-to-contact-sport clearance by a concussion-experienced clinician, but the vestibular-specific criteria for clearance are not uniformly defined, and the residual vestibular deficits acceptable for contact sport participation have not been prospectively validated [46,76]. Third, the relationship between repeated concussive injury and long-term vestibular neurodegeneration — the potential for cumulative post-traumatic vestibulopathy — is an emerging research question with significant implications for sport and occupational medicine but currently without definitive clinical evidence [18,19].

Future Directions

Emerging directions with potential to transform post-concussive vestibular management include: (1) quantitative MRI biomarkers — diffusion tensor imaging fractional anisotropy, MR spectroscopy of the hippocampus and brainstem, and gadolinium-enhanced hydrops MRI — providing objective markers of recovery independent of symptom report [22,78]; (2) wearable vestibular sensors (IMU-based head kinematic analysis, portable vHIT devices) enabling community-based real-world VOR monitoring for longitudinal tracking [79]; (3) pharmacological neuroprotection targeting the acute neurometabolic cascade — including NAD+ precursors, mitochondrial-targeted antioxidants, and microglial inhibitors — though no agent has yet achieved clinical trial evidence in human mTBI [14,80]; and (4) virtual reality-based vestibular rehabilitation platforms, enabling standardised, doseable vestibular-oculomotor exercise delivery with built-in outcome monitoring [65].

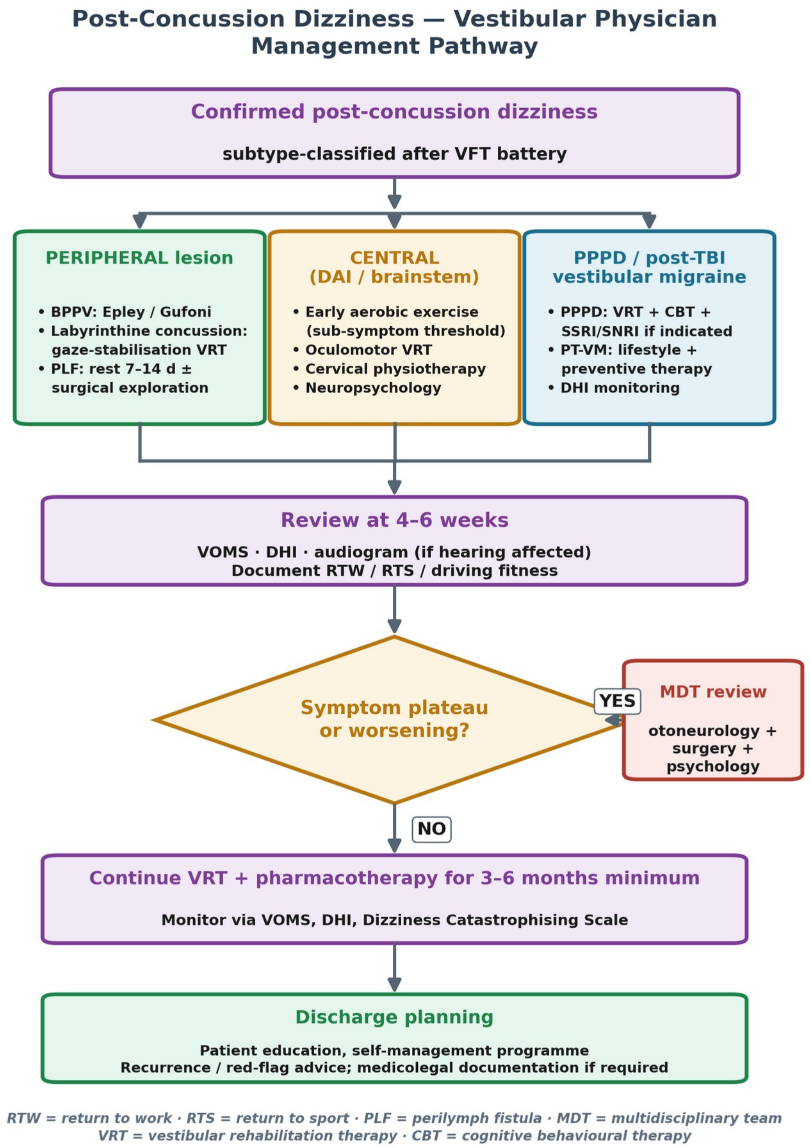

Figure 5. Management pathway for post-concussion dizziness at the vestibular physician level. RTW = return to work; RTS = return to sport; PLF = perilymph fistula; MDT = multidisciplinary team; VRT = vestibular rehabilitation therapy; CBT = cognitive behavioural therapy.

Source: Adapted from Patricios et al. [46], Lan and Hoffer [7], and Staab et al. [52].

References

[1] GBD 2016 Traumatic Brain Injury and Spinal Cord Injury Collaborators. Global, regional, and national burden of traumatic brain injury and spinal cord injury, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Neurol. 2019;18(1):56-87.
[2] Cassidy JD, Carroll LJ, Peloso PM, et al. Incidence, risk factors and prevention of mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med. 2004;(43 Suppl):28-60.
[3] Australian Institute of Health and Welfare. Hospitalised injuries among Australians 2021-22. AIHW; 2023.
[4] Hoffer ME, Balaban C, Gottshall K, et al. Blast exposure: vestibular consequences and associated characteristics. Otol Neurotol. 2010;31(2):232-236.
[5] Marcus HJ, Paine H, Sargeant M, et al. Vestibular dysfunction in acute traumatic brain injury. J Neurol. 2019;266(10):2430-2433.
[6] Akin FW, Murnane OD, Hall CD, Riska KM. Vestibular consequences of mild traumatic brain injury and blast exposure. J Vestib Res. 2017;27(5-6):297-305.
[7] Lan MY, Hoffer ME. Post-traumatic dizziness. In: Babu S, Schutt C, Bojrab D, editors. Diagnosis and Treatment of Vestibular Disorders. New York: Springer; 2019. p. 301-309.
[8] Master CL, Katz BP, Arbogast KB, et al. Vision and vestibular system dysfunction predicts prolonged concussion recovery. Clin J Sport Med. 2018;28(2):139-145.
[9] Ahn SK, Jeon SY, Kim JP, et al. Clinical characteristics and treatment of benign paroxysmal positional vertigo after traumatic brain injury. J Trauma. 2011;70(2):442-446.
[10] Gordon CR, Levite R, Joffe V, Gadoth N. Is posttraumatic benign paroxysmal positional vertigo different from the idiopathic form? Arch Neurol. 2004;61(10):1590-1593.
[11] Alsalaheen BA, Mucha A, Morris LO, et al. Vestibular rehabilitation for dizziness and balance disorders after concussion. J Neurol Phys Ther. 2010;34(2):87-93.
[12] Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the Barany Society. J Vestib Res. 2017;27(4):191-208.
[13] Giza CC, Hovda DA. The neurometabolic cascade of concussion. J Athl Train. 2001;36(3):228-235.
[14] Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery. 2014;75(Suppl 4):S24-S33.
[15] Barkhoudarian G, Hovda DA, Giza CC. The molecular pathophysiology of concussive brain injury. Clin Sports Med. 2011;30(1):33-48.
[16] Liu H. Presentation and outcome of post-traumatic benign paroxysmal positional vertigo. Acta Otolaryngol. 2012;132(8):803-806.
[17] Ishiyama G, Ishiyama A, Jacobson K, Baloh RW. Drop attacks in older patients secondary to an otological cause. Neurology. 2001;57(8):1103-1106.
[18] Smith DH, Meaney DF, Shull WH. Diffuse axonal injury in head trauma. J Head Trauma Rehabil. 2003;18(4):307-316.
[19] Griesbach GS, Hovda DA, Tio DL, Taylor AN. Heightening of the stress response during the first weeks after a mild traumatic brain injury. Neuroscience. 2011;178:209-219.
[20] Baloh RW. Vestibular neuritis. N Engl J Med. 2003;348(11):1027-1032.
[21] Dieterich M, Brandt T. Functional brain imaging of peripheral and central vestibular disorders. Brain. 2008;131(10):2538-2552.
[22] Kinnunen KM, Greenwood R, Powell JH, et al. White matter damage and cognitive impairment after traumatic brain injury. Brain. 2011;134(2):449-463.
[23] Faden AI, Loane DJ. Chronic neurodegeneration after traumatic brain injury: Alzheimer disease, chronic traumatic encephalopathy, or persistent neuroinflammation? Neurotherapeutics. 2015;12(1):143-150.
[24] Kaur P, Sharma S. Recent advances in pathophysiology of traumatic brain injury. Curr Neuropharmacol. 2018;16(8):1224-1238.
[25] Yazdi AK, Babak MR. Post-traumatic secondary endolymphatic hydrops: a case series. B-ENT. 2015;11(1):65-70.
[26] Newman-Toker DE, Kattah JC, Alvernia JE, Wang DZ. Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. Neurology. 2008;70(24 Pt 2):2378-2385.
[27] Segal BN, Alford EL. Labyrinthine concussion: definition and mechanism. Otolaryngol Head Neck Surg. 1995;112(5):688-692.
[28] Katsarkas A. Dizziness in aging: a retrospective study of 1194 cases. Otolaryngol Head Neck Surg. 1994;110(3):296-301.
[29] Hoffer ME, Gottshall K, Moore R, Balough BJ, Wester D. Characterizing and treating dizziness after mild head trauma. Otol Neurotol. 2004;25(2):135-138.

[30] Merchant SN, Gopen Q. A human temporal bone study of acute bacterial meningogenic labyrinthitis. Am J Otol. 1996;17(3):375-385.
[31] Kohut RI, Waldorf RA, Haenel JL, Thompson RS. Perilymph fistulas: review and management. Ear Nose Throat J. 1992;71(7):310-319.
[32] Fitzgerald DC. Perilymphatic fistula: a review of classification, etiology, diagnosis, and treatment. Front Neurol. 2020;11:1046.
[33] Seltzer S, McCabe BF. Perilymph fistula: the Iowa experience. Laryngoscope. 1986;96(1):37-49.
[34] Lovell MR, Collins MW, Iverson GL, et al. Recovery from mild concussion in high school athletes. J Neurosurg. 2003;98(2):296-301.
[35] Master CL, Scheiman M, Gallaway M, et al. Vision diagnoses are common after concussion in adolescents. Clin Pediatr. 2016;55(3):260-267.
[36] Mucha A, Collins MW, Elbin RJ, et al. A brief Vestibular/Ocular Motor Screening (VOMS) assessment to evaluate concussions. Am J Sports Med. 2014;42(10):2479-2486.
[37] Whitney SL, Eagle SR, Marchetti G, et al. Association of acute VOMS scores to prolonged recovery in collegiate athletes. Brain Inj. 2020;34(6):840-845.
[38] Convergence Insufficiency Treatment Trial Study Group. Randomized clinical trial of treatments for symptomatic convergence insufficiency in children. Arch Ophthalmol. 2008;126(10):1336-1349.
[39] Suh M, Kolster R, Sarkar R, McCandliss B, Ghajar J. Deficits in predictive smooth pursuit after mild traumatic brain injury. Neurosci Lett. 2006;401(1-2):108-113.
[40] Tusa RJ, Herdman SJ. Unilateral vestibular hypofunction. Neurology. 1993;43(7 Suppl 3):S48-S54.
[41] Glasauer S, Strupp M, Kalla R, Buttner U, Brandt T. Effect of 4-aminopyridine on upbeat and downbeat nystagmus. Ann N Y Acad Sci. 2005;1039:528-531.
[42] Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome. Stroke. 2009;40(11):3504-3510.
[43] Goodman BP, Vargas BB, Dodick DW. Autonomic disorders following traumatic brain injury. Semin Neurol. 2015;35(1):70-77.
[44] Kanjwal K, Karabin B, Kanjwal Y, Grubb BP. Postural orthostatic tachycardia syndrome following traumatic brain injury. J Cardiovasc Electrophysiol. 2010;21(4):413-416.
[45] Leddy JJ, Haider MN, Ellis MJ, et al. Early subthreshold aerobic exercise for sport-related concussion: a randomized clinical trial. JAMA Pediatr. 2019;173(4):319-325.
[46] Patricios JS, Schneider KJ, Dvorak J, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport — Amsterdam 2022. Br J Sports Med. 2023;57(11):695-711.
[47] Reneker JC, Clay Moughiman M, Cook CE. The diagnostic utility of clinical tests for differentiating between cervicogenic and other causes of dizziness after a sports-related concussion. Musculoskelet Sci Pract. 2015;20(1):82-89.
[48] Reid SA, Rivett DA. Manual therapy treatment of cervicogenic dizziness: a systematic review. Man Ther. 2005;10(1):4-13.
[49] Yazdi AK, Babak MR. Post-traumatic Meniere's disease: a case series. B-ENT. 2015;11(2):143-148.
[50] Magnan J, Ozgirgin ON, Trabalzini F, et al. European position statement on diagnosis, and treatment of Meniere's disease. J Int Adv Otol. 2018;14(2):317-321.
[51] Naganawa S, Nakashima T. Visualization of endolymphatic hydrops with MR imaging in patients with Meniere's disease and related pathologies. Jpn J Radiol. 2014;32(4):191-204.
[52] Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD). J Vestib Res. 2017;27(4):191-208.
[53] Popkirov S, Staab JP, Stone J. Persistent postural-perceptual dizziness (PPPD): a common, characteristic and treatable cause of chronic dizziness. Pract Neurol. 2018;18(1):5-13.
[54] Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria (update). J Vestib Res. 2022;32(1):1-6.
[55] Versino M, Colnaghi S, Sances G, et al. Dizziness and migraine: a causal relationship? Funct Neurol. 2003;18(2):97-101.
[56] Diener HC, Dodick D, Goadsby PJ, et al. Utility of topiramate for the treatment of patients with chronic migraine. Cephalalgia. 2009;29(10):1021-1027.
[57] Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. J Vestib Res. 2012;22(4):167-172.
[58] Strupp M, Versino M, Brandt T. Vestibular migraine. Handb Clin Neurol. 2010;97:755-771.
[59] Goadsby PJ, Reuter U, Hallstrom Y, et al. A controlled trial of erenumab for episodic migraine. N Engl J Med. 2017;377(22):2123-2132.
[60] Agrawal Y, Carey JP, Della Santina CC, Schubert MC, Minor LB. Disorders of balance and vestibular function in US adults. Arch Intern Med. 2009;169(10):938-944.

[61] Bartl K, Lehnen N, Kohlbecher S, Schneider E. Head impulse testing using video-oculography. Ann N Y Acad Sci. 2009;1164:331-333.
[62] Schubert MC, Della Santina CC, Shelhamer MJ. Optimizing the sensitivity of the head thrust test for unilateral vestibular hypofunction. J Vestib Res. 2008;18(5-6):285-292.
[63] Marcus HJ, Paine H, Sargeant M, et al. Vestibular dysfunction in acute traumatic brain injury. J Neurol. 2019;266(10):2430-2433.
[64] Schneider KJ, Meeuwisse WH, Nettel-Aguirre A, et al. Cervicovestibular rehabilitation in sport-related concussion: a randomised controlled trial. Br J Sports Med. 2014;48(17):1294-1298.
[65] Slobounov SM, Gay M, Zhang K, et al. Alteration of brain functional network at rest and in response to YMCA physical fitness test in concussed college athletes: RsFMRI study. Neuroimage. 2011;55(4):1716-1727.
[66] Leddy JJ, Baker JG, Willer B. Active rehabilitation of concussion and post-concussion syndrome. Phys Med Rehabil Clin N Am. 2016;27(2):437-454.
[67] Herdman SJ, Schubert MC, Tusa RJ. Role of central preprogramming in dynamic visual acuity with vestibular loss. Arch Otolaryngol Head Neck Surg. 2001;127(10):1205-1210.
[68] Ellis MJ, Leddy JJ, Willer B. Physiological, vestibulo-ocular and cervicogenic post-concussion disorders: an evidence-based classification system. Brain Inj. 2015;29(2):238-248.
[69] Lau B, Lovell MR, Collins MW, Pardini J. Neurocognitive and symptom predictors of recovery in high school athletes. Clin J Sport Med. 2009;19(3):216-221.
[70] Halstead ME, Walter KD; Council on Sports Medicine and Fitness. Sport-related concussion in children and adolescents. Pediatrics. 2010;126(3):597-615.
[71] Rubenstein LZ. Falls in older people: epidemiology, risk factors and strategies for prevention. Age Ageing. 2006;35(Suppl 2):ii37-ii41.
[72] Hoffer ME, Balaban C, Gottshall K, Balough BJ, Maddox MR, Penta JR. Blast exposure: vestibular consequences and associated characteristics. Otol Neurotol. 2010;31(2):232-236.
[73] Fausti SA, Wilmington DJ, Gallun FJ, Myers PJ, Henry JA. Auditory and vestibular dysfunction associated with blast-related traumatic brain injury. J Rehabil Res Dev. 2009;46(6):797-810.
[74] Shepard NT, Handelsman JA, Clendaniel RA. Balance and dizziness. In: Zasler ND, Katz DI, Zafonte RD, editors. Brain Injury Medicine. 2nd ed. New York: Demos; 2012. p. 779-793.
[75] Ontario Neurotrauma Foundation. Guidelines for Concussion/Mild Traumatic Brain Injury and Persistent Symptoms. 3rd ed. Toronto: ONF; 2018.
[76] Australian Institute of Sport. Concussion and Brain Health Position Statement 2023. AIS; 2023.
[77] Peus D, Dobrev I, Bijnen D, et al. Comparative proteome analysis of human perilymph: searching for biomarkers of inner ear function. J Proteomics. 2020;221:103759.
[78] Jorge RE, Acion L, White T, et al. White matter abnormalities in veterans with mild traumatic brain injury. Am J Psychiatry. 2012;169(12):1284-1291.
[79] MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS. The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology. 2009;73(14):1134-1141.
[80] Prins ML, Giza CC. Induction of monocarboxylate transporter 2 expression and ketone transport following traumatic brain injury in juvenile and adult rats. Dev Neurosci. 2006;28(4-5):447-456.

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