Identifying Vestibular Loss in Head and Brain Trauma

Presenter: Amy Lennox-Bowley, BSc (Audiology), MSc Equivalent
Hypatia Specialist Clinics & Hypatia Training | Hearing and Balance Academy / Natus Medical Incorporated

Learning Objectives

- Examine different forms of head injury and how they are categorized
- Identify common types of vestibular disorders associated with head trauma and the mechanisms behind them
- Discuss the complex nature of patients presenting with both NTBI and vestibular disorders
- Review and evaluate vestibular assessment strategies and their clinical utility
- Implement a dedicated and personalized vestibular rehabilitation plan for this patient cohort

1. Understanding Head Injury & Traumatic Brain Injury

Key Definitions

Head Injury	Damage to the head without necessarily causing damage to the brain or disruption to brain function (e.g., laceration without brain involvement).
Concussion	A mild traumatic brain injury (mTBI). Overlaps with TBI classification.
Traumatic Brain Injury (TBI)	Traumatically induced structural injury and physiologic disruption of brain function as a result of an external force. Categorized by loss of consciousness (LOC), post-traumatic amnesia (PTA), alteration of mental state, and Glasgow Coma Scale (GCS) score.

TBI Classification — Glasgow Coma Scale (GCS)

The GCS is the gold standard for grading TBI severity. It scores eye opening, best verbal response, and best motor response.

Severity	GCS Score	Loss of Consciousness	Post-Traumatic Amnesia	% of TBIs
Mild / Concussion	13–15	< 1 hour	< 24 hours	75–80%
Moderate	9–12	1–24 hours	1–7 days	~10%
Severe	< 8	> 24 hours	> 7 days	~10%

Important Clinical Note

Even 'mild' TBI can cause significant symptoms affecting sleep, mood, cognition, memory, and planning. Do not dismiss these patients. Significant improvements are possible with dedicated therapy — even in patients with severe initial scores or those over 60 years of age.

Causes of TBI

- Falls (most common cause in general population; highlights importance of vestibular prehabilitation)
- Motor vehicle accidents
- Blast injuries (10–20% of 2.5 million US military personnel deployed to Iraq/Afghanistan)
- Assaults and struck-by injuries
- Indirect trauma — e.g., rugby tackle via ankles causing shockwave without direct head contact

Types of Injury: Focal vs. Diffuse

- Focal injury: damage confined to a specific location (direct impact site)
- Diffuse injury: widespread damage across multiple brain regions — common in patients presenting to vestibular clinics
- Coup-contrecoup: injury can occur on both the side of impact AND the opposite side

Clinically, patients seen at later stages of recovery typically present with diffuse injury affecting multiple brain areas simultaneously.

2. Dizziness & TBI — Epidemiology

83%
report dizziness in acute TBI

37% still report non-rotational dizziness and imbalance 5 years post-injury

Dizziness and vertigo have independently been linked to poor recovery and disability outcomes in TBI — more than any other symptom.

3. Vestibular Disorders Associated with TBI

3.1 Post-Traumatic BPPV

The most common cause of dizziness in most populations — though potentially less so in TBI. Otoconia become displaced from the utricle into semicircular canals following head injury.

- Symptoms: dizziness when rolling in bed, changing head position
- Diagnosis: positional testing (Dix-Hallpike, roll test) — observe for nystagmus that fatigues
- Treatment: repositioning manoeuvres (e.g., Epley manoeuvre)
- Prevalence in TBI: ~17% in one study (Kerala, n=128); recurrence rate at 6 months ~40%
- Recurrence rate is notably higher in TBI patients than the general population

3.2 Labyrinthine Concussion

Damage to the inner ear from head trauma without skull fracture.

- Always presents with sensorineural hearing loss (SNHL)
- May present with or without vestibular symptoms
- Key feature: symptoms may be immediate OR delayed by days after injury — always consider trauma as a cause even if onset was delayed
- Mechanisms include reverse travelling waves affecting outer hair cells and cochlear cyanopathy

3.3 Traumatic Endolymphatic Hydrops (Post-Traumatic Ménière's Disease)

Rare condition (only ~7 publications since 1962, each describing 1–2 patients). Presents identically to typical Ménière's disease.

- Causes: fistulization of the bony labyrinth, direct injury to membranous labyrinth, injury to endolymphatic fluid drainage system
- May be missed and categorized simply as Ménière's disease if trauma history is not elicited
- Expected hearing loss: sensorineural, low-frequency

3.4 Superior Semicircular Canal Dehiscence (SSCD) — Third Window Syndrome

Thinning or a hole in the superior semicircular canal, often resulting from temporal bone fracture.

- Unusual triggers: dizziness with laughing, sneezing, coughing, lifting, sound (shower drops, music)
- Bone conduction hyperacusis: tuning fork on ankles heard in the head
- Audiological: low-frequency conductive hearing loss, but VEMP present despite conductive loss (sound attenuated via bone, not middle ear)
- VEMP that does not abolish at 75 dB HL suggests third window pathology

3.5 Perilymph Fistula

Proposed diagnostic criteria (Bárány Society — not yet finalized):

- Fluctuating or non-fluctuating SNHL, tinnitus, aural fullness (resembles Ménière's/hydrops)
- Must be preceded by: barotrauma, direct trauma (TBI), straining, or valsalva-type event
- Confirmed by: laboratory testing, imaging, or observation of perilymph leakage in middle ear
- The Hennebert (Fistula) Sign: nystagmus elicited by applying external pressure to the suspected ear
- Symptoms fluctuate as perilymph drains and refills — test results may vary day to day

3.6 Otolith Dysfunction

Particularly associated with TBI (prevalence ~30%) and blast injuries (~52%). High interest from military and NASA.

- Isolated otolith damage (without semicircular canal damage) = very atypical vestibular presentation
- Symptoms are vague: generalized unwellness, anxiety, poor spatial awareness, tilting, waxing/waning, swaying, rocking, bobbing
- 'Feels well in the car, but when the car stops, feels like the car is still moving'
- Normal caloric and VHIT; abnormal VEMP results (c/oVEMP)
- Often missed due to non-rotary, non-episodic presentation

3.7 Non-Vestibular Causes of Dizziness in TBI

- Central pathology: ongoing cerebellar/brainstem damage
- Medications (may be lifelong and ototoxic/vestibulotoxic)
- Visual processing changes
- Orthostatic hypotension (impaired autonomic homeostasis)
- Post-traumatic headache / chronic pain (pain prevents effective rehab — must be managed first)
- Proprioceptive disruption (e.g., limb fractures, traction, altered somatosensory input)

4. Clinical Presentation — What to Expect

Most patients presenting to specialist vestibular clinics are typically 18+ months post-injury. BPPV has often already been treated; these are the patients with ongoing, complex symptoms:

Vestibular / Balance Symptoms	Cognitive / Psychological Symptoms
• Vague, non-rotational dizziness • Postural instability • Visual vertigo / motion sensitivity • Poor spatial awareness • Tinnitus / hyperacusis • Post-traumatic headache • Truncal movement compensation	• Brain fog • Anxiety and low mood • Poor memory, planning, cognition • Personality changes / emotional lability • Agoraphobia / social withdrawal • Auditory processing difficulties • Maladaptive movement restriction

Visual Vertigo — A Key Concept

Defined as the result of being overstimulated by a complex visual environment. Occurs because:

- Normally, the vestibular system drives perception of movement — visual input is secondary
- After vestibular loss/asymmetry, the brain compensates by prioritizing visual input
- Result: if the patient SEES movement, they FEEL movement
- Common triggers: supermarkets, airports, tube stations, fast-moving screens, action films, TikTok

The TikTok Outcome Measure

A practical informal measure used in clinic: how quickly can the patient scroll through their TikTok feed? Faster tolerance = better vestibular compensation. Useful for tracking progress and engaging patients in self-monitoring.

5. Vestibular Assessment Battery

A typical assessment for a TBI patient takes 3–3.5 hours, including 1–1.5 hours for history taking. Allow breaks throughout.

5.1 Audiological Assessment

- Pure tone audiometry + speech audiometry
- Speech-in-noise testing (if auditory processing disorder suspected)
- Tympanometry and acoustic reflexes — critical before VEMP testing (confirms sound can conduct through middle ear)

5.2 Positioning Assessment

- Dix-Hallpike and roll test to identify and treat BPPV before further testing
- Treat BPPV first — this prevents confounded results on subsequent tests

5.3 Peripheral Vestibular Tests

VHIT	Video Head Impulse Test — assesses semicircular canal (VOR) gain; compensatory saccades indicate hypofunction. Gold standard for semicircular canal assessment.
SHIMP	Suppression Head Impulse Paradigm — assesses predictive/programmed saccades; asymmetry ratio is a sensitive marker. Still emerging in research.
cVEMP	Cervical VEMP — assesses saccule and inferior vestibular nerve function. Performed at 95 dB HL; compare both sides for asymmetry. Bilateral testing: abolishes at 75 dB normally.
oVEMP	Ocular VEMP — assesses utricle and superior vestibular nerve function. Complements cVEMP for comprehensive otolith assessment.
BVOR	Binaural bithermal caloric testing — useful but can provoke severe symptoms in central pathology patients. Courts value calorics in medico-legal cases. Always discuss with patient first.

5.4 Neuro-vestibular / Central Tests (VNG)

Smooth Pursuit	Central test — assesses ability to track a moving target smoothly
Saccades	Central test — assesses speed and accuracy of rapid eye movements
Subjective Visual Vertical (SVV)	Otolith test — utricular function assessment
Gaze / Spontaneous Nystagmus	Central and peripheral
Head Heave Test	Otolith assessment
Ocular Counter-Roll	Otolith (utricular) function

Hennebert Sign	Perilymph fistula / SSCD — nystagmus elicited by external ear pressure
Visual Vertigo Analysis (VVA)	Quantifies visually-induced dizziness

5.5 Patient-Reported Outcome Measures

- DHI (Dizziness Handicap Inventory) — primary outcome measure; 0 = no handicap, 100 = maximum handicap; scores ≥72 = severe
- Visual Vertigo Analogue Scale (VVAS)
- Pediatric DHI — for patients under 18
- TBI-specific questionnaires — consider for earlier-stage TBI assessment

VEMP Testing Tip

You are NOT testing hearing with VEMP — you are using sound to stimulate the vestibular organ. Middle ear conductivity matters, not hearing sensitivity. A patient with SNHL can still have a valid VEMP if the middle ear is conducting normally.

6. Rehabilitation Approaches

Rehabilitation should be multidisciplinary and personalized. Pain management must be prioritized — chronic headache significantly inhibits rehabilitation outcomes.

6.1 Foundation Principles

- 'Feed the body movement' — movement drives neural plasticity and compensation
- Start with gentle, graded activity (e.g., supervised gym sessions with a personal trainer)
- Address maladaptive avoidance behaviours early (motivational approach, CBT)
- Consider referral to: physiotherapy, occupational therapy, neuropsychology, pharmacy review
- Treat the whole person — cognitive, psychological, and physical domains

6.2 Gaze Stabilization & VOR Exercises

- Train the vestibulo-ocular reflex (VOR) to maintain stable gaze during head movement
- Begin with simple fixation exercises and gradually increase speed and complexity
- Essential for patients with VHIT deficits

6.3 Optokinetic (OPK) Retraining

- Uses moving visual patterns (black-and-white stripes, moving left/right or up/down)
- Patient follows the stripes to retrain visual-vestibular integration
- Particularly effective for visual vertigo and visually-induced dizziness
- Evidence: 5-day OPK + VRT protocol in athlete with concussion → VVAS scores near zero

6.4 Virtual Reality (VR) Rehabilitation

An innovative and highly engaging tool for vestibular rehabilitation in TBI patients.

- Mechanism: teaches the brain that visual-vestibular mismatch signals are safe and normalizes them; desensitizes the visual system to motion
- Progression — start easy, advance systematically:
  - Level 1: Plain backgrounds, slow movement, minimal visual noise (e.g., gentle snow scene)
  - Level 2: More colour, people moving through environments (e.g., a busy market)
  - Level 3: High stimulation — bouncing objects, snow, rapid head movement, lights, cars
- Gamification: fishing games, coordination tasks — especially effective with children and for sustained engagement
- Patients can use VR at home between sessions (e.g., daily practice)

Personalization Tip

Tailor VR content to the patient's interests. A patient who loves fishing can use a fishing game — this increased adherence dramatically (daily home practice). A child anxious about missing school can have physiotherapy delivered at school. Patient engagement is key.

7. Case Studies

Case 1 — Acute: Professional Ice Hockey Player (Vivi Moochie, published)

Patient	25-year-old male professional ice hockey player
History	3 concussions (2015, 2017, 2018); visually-induced dizziness, reduced gaze stabilisation
Treatment	5-day combined vestibular rehab + OPK retraining programme
Outcome	VVAS scores reduced from moderate-high pre-treatment to near-zero (≈0.x) at day 5. Returned to sport.
Key Learning	Short, targeted, combined programmes can produce dramatic results even in acute/sub-acute presentations

Case 2 — Chronic: Paul, 29-year-old male

History	Right vestibulopathy post-MVA. No prior treatment or investigation until 2 years post-injury
Symptoms	Continuous dizziness/unsteadiness, brain fog, anxiety, cognitive issues. DHI: 72 (severe)
Investigations	VHIT: right lateral catch-up saccades. cVEMP: absent right. VNG: multiple positive signs
Treatment	Rocksteady book, CBT, graded gym programme, gaze stabilization/OPK, then VR as improvement shown
Outcome	VHIT gains improved (catch-up saccades more time-locked). SHIMP asymmetry: 40% → 23%. DHI significantly reduced
Key Learning	A staged approach starting with education and gentle movement, progressing to technology-assisted rehab, yields measurable objective improvement

Case 3 — Paediatric: Emily, 9-year-old female

History	Left vestibulopathy + vestibular migraine since infancy. Now presenting with extreme fatigue, dizziness, falls
Symptoms	Feels permanently dizzy, very anxious, exhausted at school. Vestibular function tests normal; absent left cVEMP; reduced SHIMP gain
Scores	Pediatric DHI: 36 (moderate); Pediatric VVAS: moderate
Treatment	Pharmacology (migraine), neuropsychology (8 sessions every 2 weeks), physiotherapy delivered at school, then graded VR
Outcome	SHIMP asymmetry: 49% → 6%. DHI: 36 → 20. Continued improvement with ongoing VR treatment
Key Learning	Paediatric TBI exists and these patients can achieve meaningful recovery. Flexibility in delivery (school-based therapy) is key to compliance

8. Practical Clinical Recommendations

Service & Appointment Planning

- Allow extra appointment time — these patients fatigue easily and need breaks during assessment
- Use visual appointment letters (photos of clinic, staff, what to expect) — memory and planning difficulties are common
- Implement reminder systems — missed appointments often due to genuine forgetting, not disengagement
- Be aware of emotional lability — patients may become upset quickly; approach with empathy and patience

Caloric Testing in TBI

Calorics can provoke hyperresponsive results in patients with central pathology, which may be very distressing. Discuss risks with the patient, explain the value for medico-legal purposes if relevant, and use clinical judgement. Teach breathing techniques (nasal in, mouth out) to minimise anxiety during the test.

Medico-Legal Considerations

- Courts value caloric results — complete if patient consents and can tolerate
- SHIMP and other newer tests are still emerging in research — consider in context alongside established tests
- Comprehensive test battery strengthens evidence-based documentation of vestibular loss

9. Key Takeaways

Summary Points to Remember

- Vestibular problems are significantly underdiagnosed in TBI patients — dizziness is independently linked to poor recovery outcomes
- Multiple vestibular conditions can co-occur in TBI — a comprehensive test battery is essential
- Otolith dysfunction is common and frequently missed — VEMP is irreplaceable for assessing these organs
- Multidisciplinary collaboration is not optional — it is required for optimal outcomes
- Pain must be managed before effective vestibular rehab can begin
- Significant recovery IS possible — even years post-injury and in severe TBI. Don't write patients off
- Technology — VR, OPK, SHIMP — is opening new frontiers in both assessment and rehabilitation

- Paediatric patients are not immune to TBI — apply the same comprehensive approach

10. Notes & Contact

Amy Lennox-Bowley
Hypatia Specialist Clinics & Training
Liverpool, England
Contact for questions, data sharing, and collaboration on post-traumatic BPPV research

Notes