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Module 1

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**Vestibular Dysfunction in Individuals with Moderate-to-Severe Traumatic Brain Injury (TBI)**

**Why and How Often Does Vestibular Dysfunction Occur in People with Moderate-to-Severe TBI?**

Vestibular dysfunction is common following moderate-to-severe traumatic brain injury (TBI) and can be caused by injury to both peripheral and central vestibular structures. Common peripheral vestibular disorders following TBI include benign paroxysmal positional vertigo (BPPV), labyrinthine concussion, perilymphatic fistula, and acute peripheral vestibular loss. Common central vestibular disorders following TBI include post-traumatic vestibular migraine, post-traumatic persistent-postural perceptual dizziness (PPPD), damage to the cerebellum, and damage to the brainstem. 1, 2, 3

Vestibular dysfunction resulting from TBI can have significant repercussions for recovery. For instance, ambulatory individuals with acute TBI who experience symptoms such as dizziness and imbalance have worse physical, psychological, and socioeconomic outcomes. Vestibular dysfunction, whether peripheral or central in origin, affects as many as 80% of ambulatory individuals with moderate-to-severe acute TBI.2 Around five years post TBI, approximately 50% of individuals still experience vestibular symptoms.1 Despite the prevalence of vestibular dysfunction in people with moderate-to-severe TBI, early diagnosis and treatment remain insufficient. Thus, vestibular screening should be implemented in the acute phase of care for individuals post-TBI as the nature and degree of symptoms can help guide treatment strategies and improve long-term outcomes.2

**Pathophysiology of Vestibular Dysfunction following Moderate-to-Severe TBI**

The underlying mechanism of vestibular dysfunction in individuals with moderate-to-severe traumatic brain injury varies depending on whether the dysfunction originates from peripheral or central factors.2 After brain injury, vestibular dysfunction can occur because of injury to the inner ear, vestibular nerve, brainstem, cerebellum, or cortical networks responsible for processing vestibular information.3

Common Peripheral Pathophysiology

BPPV is one of the most common peripheral vestibular disorders with traumatic BPPV accounting for approximately 8-20% of all BPPV cases.4 Following head injury, otoconial debris may become dislodged and separate from the macula which can lead to sedimentation of this debris in the semicircular canals (typically the posterior canal). When the particles are shifted during head and neck movement, it stimulates ampullar receptors and leads to vertigo symptoms. Because of its high prevalence, BPPV should be considered in all patients following head injury who complain of positional vertigo.4

Additionally, acute peripheral vestibular loss following TBI can occur due to injury to the peripheral vestibular structures. For example, acute peripheral vestibular loss may result from fracture of the bony or membranous labyrinth, microstructural injuries to the inner ear, or injury to vestibular afferent neurons. Similarly, labyrinthine concussion may originate from the movement of the membranous labyrinth on the bony labyrinth during the acceleration-deceleration phase of brain injury or from compression and vibration forces from blunt trauma. Both mechanisms previously mentioned can result in hemorrhaging, disruptions in circulation, and epithelial damage within the inner ear.5 Perilymphatic fistula typically occurs after a traumatic event and is an abnormal rupture that allows perilymph fluid to leak out of the inner ear into the middle ear space. The abnormal fluid leakage from the inner ear into the middle ear causes vestibular symptoms.6

Common Central Pathophysiology

The pathophysiology of vestibular migraine is largely unknown, but Espinosa-Sanchez et al7 reports that abnormal brain sensitization can lead to abnormalities in thalamo-cortical processing and vestibular hypersensitivity. The abnormal processing of both vestibular information and nociceptive information could lead to vestibular dysfunction with simultaneous migraine symptoms.7 In PPPD, another central vestibular disorder, the individual has a triggering vestibular event which leads to high anxiety and caution over their acute physical symptoms. Overtime, this leads to maladaptive postural reflexes, excessive self-monitoring, and avoidance behaviors. This maladaptation leads to disturbed somatosensory information about the body’s position which causes them to experience increased dizziness and gait disturbances.8

It also should be noted that individuals with moderate-to-severe TBI may have impairments in visual acuity, eye movements (including convergence), saccades, smooth pursuits, and visual perception. These visual impairments may occur following TBI due to damage to the optic nerve, optic tracts, or occipital lobe. Visual impairments may manifest as symptoms like headache, dizziness or vertigo, and imbalance.9, 10

**Symptoms Associated with Vestibular Dysfunction in People with Moderate-to-Severe TBI**

It is evident that vestibular dysfunction following TBI occurs through various mechanisms, so symptom presentation is also variable depending on the structures that are involved. Common vestibular symptoms following TBI include balance deficits, vertigo, dizziness, headache, visual impairments, and auditory changes.9, 11 The most consistent way to categorize vestibular dysfunction following TBI is peripheral versus central vestibular dysfunction.11

Individuals with peripheral vestibular disorders typically present with vertigo, lightheadedness, nausea, vomiting, auditory changes, ringing in the ears, fullness in the ears, sweating, bradycardia, and other autonomic symptoms. Patients with peripheral vestibular disorders also demonstrate nystagmus to the contralateral side which can be suppressed with visual fixation. Vestibular compensation occurs quickly in peripheral vestibular disorders but occurs slowly in central vestibular pathology. Individuals with central vestibular dysfunction typically complain of more severe disequilibrium and ataxia and have decreased ability to ambulate due to instability. In central vestibular pathology, nystagmus changes direction with gaze, is unaffected by gaze fixation, and is typically vertical or torsional in direction. Another symptom characteristic of central vestibular pathology is visual blurring or oscillopsia.11

Although there are characteristic features differentiating peripheral from central vestibular pathology, there are also unique symptoms among the previously mentioned vestibular disorders. Momentary vertigo that lasts less than one minute and is triggered by head movements is suggestive of BPPV. Patients with BPPV will also experience nystagmus, lightheadedness, nausea, and increased falls. Further, patients present with a positive Dix-Hallpike Test or Supine Roll Test based on the semicircular canal involved. Patients with labyrinthine concussion will have transient vertigo or disequilibrium and hearing loss at 4,000 Hz. Typically, symptoms resolve within days to weeks but may persist longer.11 Patients with perilymphatic fistula may complain of disequilibrium with increased cerebrospinal fluid pressure or when exposed to loud noises. These patients may have nystagmus with positive pressure applied to the ear during pneumatic otoscopy. Individuals with vestibular migraine may complain of episodic vertigo, gait ataxia, nausea, auras, sensitivity to light, sensitivity to noise, and sensitivity to head movements. Vestibular symptoms may occur with the headache, after the headache, or between headache attacks.11 Finally, patients with PPPD present with persistent dizziness, unsteadiness, or non-spinning vertigo for greater than three months. Symptoms last for hours and are worsened by upright postures and environments with complex visual stimuli.8

Determining whether the individual has peripheral versus central vestibular dysfunction influences the treatment strategy, patient goals, and the individual’s prognosis in physical therapy. The goal of physical therapy in vestibular rehabilitation is to promote central nervous system compensation to improve symptoms and reduce impairments. Pathology of peripheral vestibular structures impairs sensory information regarding the person’s head position and movement. Peripheral vestibular impairments may be transient or permanent, but these individuals can achieve compensation faster because their central vestibular structures are intact. Individuals with central vestibular disorders, on the other hand, have limited recovery of vestibular dysfunction because pathology of the central vestibular structures impacts the individual’s ability to process and integrate vestibular, visual, and somatosensory information. Thus, damage to central vestibular structures results in slower compensation. The average length of vestibular therapy for peripheral vestibular disorders is 6-12 weeks, but the average length for central vestibular disorders is typically longer.12

**Assessment and Outcome Measures for Vestibular Dysfunction in People with Moderate-to-Severe TBI**

A general vestibular screen should be performed on individuals with moderate-to-severe TBI in the acute phase of treatment as able.1 The screen should first include an assessment of neck range of motion and assess for the presence of spontaneous nystagmus when the patient is looking straight ahead. Additionally, the therapist should assess smooth pursuit, ocular motion, saccades, and near point convergence.9, 10 If patient complaints suggest positional vertigo, the Dix-Hallpike Maneuver and Supine Roll Test should be performed.13 The Dix-Hallpike Manuever is considered to be the gold standard test for posterior canal BPPV. Frenzels glasses, which remove the eyes’ ability to focus on an object, can be used during the tests to reduce optic fixation. The Supine Roll Test is used to detect the presence of horizontal canal BPPV.4

The HINTS (head impulse, nystagmus, and skew deviation) test is useful in differentiating peripheral versus central causes of vertigo. The head impulse test looks at high-velocity VOR. In the presence of peripheral vestibular pathology, VOR gain will be decreased resulting in corrective eye movements or saccades during the test. The VOR Suppression Test (also called the VOR Cancellation Test) is used to detect central vestibular pathology because VOR gain is typically normal in central vestibular disorders, but VOR suppression is abnormal.14 The characteristics of nystagmus also give insight to whether the individual has central versus peripheral pathology. Nystagmus in peripheral vertigo is unidirectional and rotary whereas the nystagmus in central vertigo is vertical or direction changing. Presence of skew deviation, or one eye higher than the other, indicates central vertigo. This can be assessed using the Alternate Cover Test. During this test the therapist covers one of the patient’s eyes and assesses for a vertical corrective saccade which indicates central pathology.14 Further, the Dynamic Visual Acuity Test is a functional test for VOR and is used to assess dynamic visual acuity (visual acuity while turning the head) compared to static visual acuity.15 Finally, the Bucket Test is a quick and easy measure of the patient’s subjective visual vertical which is often impaired in unilateral vestibular loss or dysfunction. Patients with BPPV have also been shown to have impaired subjective visual vertical due to impaired spatial orientation.13 Additional assessments can be performed based on the patient’s specific symptoms and the results of the previously mentioned tests for oculomotor and vestibular function.

Outcome measures administered to people with vestibular dysfunction following moderate-to-severe TBI should aim to assess dizziness, static and dynamic balance, sensory integration, functional mobility, and falls risk. Outcome measures recommended for use in the inpatient and outpatient setting by both the TBI EDGE taskforce and the vestibular EDGE taskforce include the Dizziness Handicap Inventory (DHI) and Berg Balance Scale (BBS).16, 17

Measures that assess static and dynamic postural stability and are indicated for use in people with vestibular dysfunction include the Clinical Test of Sensory Interaction on Balance (CTSIB), the Functional Gait Assessment (FGA), and the Dynamic Gait Index (DGI).17 Additional outcome measures that assess balance indicated for use in individuals with TBI are the Community Balance and Mobility Scale (CB&M) and Balance Error Scoring System (BESS).16 The BESS assesses static postural stability but is typically used in people with mild TBI.18

Further, the Timed Up and Go test is a reliable and valid measure for assessing functional mobility and predicting falls risk.19 The Motion Sensitivity Quotient (MSQ) is useful for measuring the duration and intensity of dizziness with various head and body positions.20

Self-reported outcome measures that are recommended by the Vestibular EDGE taskforce that may be useful for TBI patients with vestibular dysfunction are the Activities Specific Balance Confidence (ABC) Scale, and the Vestibular Rehabilitation Benefit Questionnaire (VRBQ).17

**Physical Therapy Treatments and Mechanisms for Improvement**

Treatment approaches for vestibular dysfunction in individuals with moderate-to-severe TBI differ based on the underlying cause, specific symptoms experienced by the patient, and the patient’s overall functional status. The primary principles of vestibular rehabilitation include compensation, adaptation, habituation, and substitution.

Compensation for the loss of vestibular function occurs in response to permanent vestibular damage and is a process of recovery.21 Compensation involves adaptation of residual VOR gain, habituation of symptoms, substitution with alternative strategies, and regaining postural control.22, 23 Habituation is one mechanism of compensation that results from repeated exposure to a provocative stimulus and attenuation of the individual’s response to that stimulus. Habituation has been found to be effective for the treatment of both central and peripheral vestibular disorders. With that said, habituation was found to be less effective for individuals with central vestibular pathology due to head injury and individuals with bilateral vestibular loss.21 Adaptation, another mechanism of compensation, is the long-term recovery of neuronal responses by normalizing gaze and postural stability during head movements which ultimately reduces symptoms. Adaptation exercises involve head movement with a stationary target, movement of the target and head, target placement with distracting visual stimuli, and challenging postures. Substitution is another mechanism of compensation. Substitution exercises attempt to “facilitate the use of alternative strategies” for missing vestibular function.21

Gaze stabilization exercises are used during vestibular rehabilitation and are an example of both substitution and adaptation.Gaze stabilization exercises based on vestibular adaptation include head movements while focusing on a target such as VORx1 or VORx2. Conversely, gaze stabilization exercises based on vestibular substitution include smooth pursuit eye movements or central pre-programming eye movements (saccades). Examples of gaze stabilization exercises based on substation are eye-head movements between targets or remembered target exercises.23

Ultimately, treatment strategies for individuals with peripheral vestibular disorders often include gaze stabilization to improve VOR gain, Canalith Repositioning Maneuvers (CRM) to improve positional vertigo in BPPV, habituation exercises to improve sensitivity to body movements, and optokinetic exercises to improve visual motion sensitivity.21 The Epley maneuver and Barbeque maneuver are used to treat BPPV of the posterior canal and horizontal canal respectively and have shown a success rate of approximately 85% with the first treatment.4, 24 Nonetheless, it has been found that around 30-50% of cases of BPPV spontaneously recover so it may not be advisable to perform repositioning maneuvers in severely injured trauma patients such as individuals with acute TBI. Severity of trauma and duration of ICU treatment are two factors that may delay treatment of BPPV in TBI patients.24

Vestibular rehabilitation for central vestibular disorders uses the same treatment principles as those used in peripheral vestibular disorders (adaptation, substitution, and habituation), but typically incorporates VOR suppression, VOR memory, anti-saccade, and memory-guided saccade exercises.25

Vestibular rehabilitation for PPPD includes habituation exercises, gaze stabilization, balance training, relaxation techniques, and dynamic gait training. Habituation exercises are used to reduce abnormal postural reflexes and decrease sensitivity to provocative stimuli.8 Typically, in patients with PPPD,habituation exercises are more effective in improving symptoms provoked by head and body movements rather than symptoms provoked by visually complex environments.25 Slow progression is key for patients with PPPD.8 Vestibular rehabilitation may be used in conjunction with cognitive behavioral therapy and pharmacological intervention to target psychological symptoms. Lastly, the aim of vestibular rehabilitation for vestibular migraine includes habituation exercises to desensitize the vestibular system, coordination between head and eye movements with gaze stabilization exercises, posture and balance retraining, and dynamic gait training. Vestibular rehabilitation can be used in conjunction with prophylactic medication to reduce the frequency and severity of attacks. Vestibular suppressants can be used to reduce symptoms but should be discontinued due to their ability to hinder compensation.25

**Influence on Future Practice**

The high prevalence of vestibular dysfunction following moderate-to-severe TBI highlights the importance of early diagnosis and management. Vestibular dysfunction is often missed in the acute phase of treatment due to ambiguous objective signs and symptoms that may be difficult to detect for non-vestibular therapists. Additionally, vestibular dysfunction may be confused with other post-traumatic neurological conditions like epilepsy. Finally, vestibular dysfunction is often overlooked because vestibular signs and symptoms may not be a priority to the patient or clinician in that specific phase of treatment.2 It is important, however, to understand the immense physical, psychological, and economic impact that vestibular dysfunction can have in individuals following TBI. Early vestibular screening in the inpatient rehabilitation or outpatient settings could improve patient symptoms, prevent falls, enhance quality of life, and optimize recovery.2 When treating individuals with moderate-to-severe TBI in the future, these insights will help me adopt new treatment and assessment behaviors, improve my outcome measure selection for post-traumatic vestibular patients, and enhance my patient and caregiver education about vestibular disorders and prognosis. It is crucial to continue to use clinical decision making to determine the correct patients to screen for post-traumatic vestibular disorders, but having this knowledge will better prepare me to recognize and treat these dysfunctions.

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