Post Concussive Vestibular Outcome Measures and Interventions

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**Post Concussive Syndrome**

The persistence of mild traumatic brain injury symptoms beyond a normal recovery timeframe despite appropriate physical and cognitive rest is termed Post Concussive Syndrome (PCS).1 According to the World Health Organization’s International Classification of Diseases, a PCS diagnosis requires at least three of the following symptoms: headache, dizziness, fatigue, irritability, insomnia, concentration difficulty, or memory difficulty.2 PCS can be a reflection of continued anatomic, physiologic, or neurometabolic pathology, which is likely if initial symptoms are exacerbated with exertion but improved with rest.1,3 Conversely, if exertional symptoms no longer respond to rest, then they may be propagated by concurrent non-organic psychological symptoms resultant of prolonged inactivity or frustration with symptom unremittance.1 Additionally, damage resulting in visual dysfunction, cognitive dysfunction, autonomic nervous system dysfunction, vestibular dysfunction, or any unique combination thereof contributes to the manifestation of PCS.1

Specifically, vestibular pathology commonly presents in the form of dizziness and resultant nausea, vomiting, migraine, and/or impaired balance.1,4 However, cervicogenic dizziness or headache from a whiplash injury can mimic PCS.1,4 With cervical injury, extreme flexion or extension may compress vasculature or motion may be undetected by injured joint receptors.4 Impaired cervical proprioception causes conflicting signals at the vestibular nucleus that result in dizziness, differentiated from vestibular dizziness as it often presents with *active* head movement and neck pain.4

The complexity of even strictly vestibular symptoms demands the application of specific and sensitive outcome measures for accurate diagnosis and effective treatment. While treatment of PCS relies on a clinician’s ability to differentiate among various pathologies, this paper exclusively addresses outcome measures and treatment activities for vestibular issues relevant to PCS. These issues can be divided into two categories: peripheral dysfunction due to damage along the vestibulocochlear nerve or within the labyrinth, or central dysfunction due to central damage to the vestibular nuclei or higher pathways. Both categories share common intervention components.

**Common Intervention Components**

 First and foremost, there are several common themes in vestibular rehabilitation. Education of both patient and caregiver is key to ensuring their understanding of and adherence to the prescribed intervention(s). Cognitive behavioral therapy and mild to moderate aerobic exercise are also commonly integrated into the overall intervention strategy.4 Likewise, general guidelines include only progressing a treatment activity once the patient has been asymptomatic for at least 24 hours.4 These are important considerations for developing plans of care to address the following vestibular issues.

**Peripheral Vestibular Dysfunction**

**Benign Paroxysmal Positional Vertigo**

BPPV is an inner ear condition in which calcium carbonate crystals called otoconia are dislodged from the utricle’s otolithic membrane and travel into the semicircular canals, which are oriented at right angles to each other. This orientation provides information on the head’s position in space, as endolymph pressure is registered by each canal’s capula relative to the other.4 BPPV usually occurs unilaterally, with the presence of otoconia disrupting canula signals occurring in only one ear. Thus, mismatched signals between the right and left inner ear result in a feeling of spinning known as vertigo. Mismatched signals simultaneously disrupt the Vestibular Ocular Reflex (VOR), resulting in temporary nystagmus accompanied by associated nausea, vomiting, and imbalance.4

Outcome measures for monitoring BPPV include the Dix-Hallpike test and the Pagnini-McClure (Supine Roll) test, which involve moving the head through arcs of motion that follow the path of each semicircular canal. These tests measure symptom provocation specific to each canal in which otoconia remain present.4

 Epley’s canalith repositioning maneuver is a treatment that uses gravity to move otoconia out of the posterior semicircular canals and back to the utricle. The maneuver begins with the Dix-Hallpike test procedure, followed by the patient slowly rolling to their side as the therapist holds their head to align the targeted canal so that otoconia flow out. Unique maneuvers are required for involvement of otoconia in each of the other two semicircular canals. For otoconia adhered to the cupula, Brandt-Daroff’s liberatory maneuver is preferable. One to five sessions over two or three weeks typically remedy BPPV, but residual VOR dysfunction may require additional intervention.4-7

**Gaze Instability**

Concussion can cause VOR disruption independent from BPPV, and disturbed VOR as a result of unilateral vestibular hypofunction causes an inability to stabilize one’s gaze during movement. This creates symptoms of spontaneous nystagmus, blurred vision, dizziness, vertigo, and feeling that the environment is moving, all of which contribute to postural instability and imbalance.4,9 The functional impact of VOR deficits may be measured through the Dynamic Visual Acuity Test (DVAT).4

 Treatment of VOR disruption includes the VORx1 exercise of focusing on a fixed object while moving one’s head up and down or side to side and the VORx2 exercise of tracking a moving object up and down or side to side by moving the eyes and head. The goal of these exercises is to habituate the vestibular system by provoking mild to moderate symptoms. To do this, progression techniques include increasing speed of head movement, increasing activity duration, and placing the fixed object in front of a busy background. To further challenge VOR’s role in balance, the patient may sit and then stand on increasingly unstable surfaces while performing dynamic activities.8

**Motion Sensitivity**

 Also known as visual vertigo, motion sensitivity occurs when environmental movement exceeds comfort level, such as passing trees outside a moving car, passing shoppers within a crowded mall corridor, or flashing scenes on a television screen.4,10

As previously mentioned, motion sensitivity can be clinically evaluated via the VOMS.8 Additionally, motion sensitivity can be clinically evaluated via DVAT or subjectively measured through self-report questionnaires such as the Dizziness Handicap inventory (DHI), Motion Sensitivity Quotient (MSQ), or Activities-Specific Balance Confidence Scale (ABCS).3,4

Treatment hinges on habituation through optokinetic interventions, with many patients inadvertently worsening their condition by avoiding noxious stimuli.4,10 Introduction to habituation should be gradual, beginning in a quiet space as many patients suffering PCS are also sensitive to noise.4,10 Diaz recommends the following habituation protocol, which synthesizes findings from her literature review. Phase I begins in a visual and auditory free environment, in which the patient sits and focuses on a fixed object while large intricately designed sheets move at their sides and to their front.4 Increased speed and change in direction of the sheets may be used to provoke mild to moderate symptoms, as this is the goal of treatment. As tolerance for the activity improves, Phase II progresses functional tasks performed under the same stimuli, and these tasks include standing on a stable surface, standing on one leg, standing on an unstable surface, etc.4 Phase II integrates auditory stimuli, at which point the environment may be changed to a loud and dynamic venue that is more representative of daily life.4 Similar interventions may be progressed via computer screens, advancing to larger screens as patients improve.

**Impaired Postural Control, Balance, and Gait**

 In addition to aforementioned vestibular dysfunction, damaged sensory processing centers can impair the brain’s ability to integrate vestibular information.

As opposed to generic balance outcomes such as tandem gait, Rhomberg Test, and High Level Mobility Assessment Tool alone, the Sensory Organization Test (SOT), Clinical Test of Sensory Integration in Balance (CTSIB), modified CTSIB, and computerized posturography are discretely differentiate among visual, somatosensory, and vestibular deficits’ contributions to postural sway and loss of balance.4 Treatment can then be tailored to address the appropriate deficits.

For vestibular deficits, Witney and Herdman’s proposed exercise plan for patients with vestibular hypofunction appears applicable to patients with PCS.4 Beginning by standing still on a stable surface with feet together and eyes open, hand support may be utilized only as needed.10 When hand support is no longer needed, patients progress to eyes closed, while other conditions remain the same.11 Next, patients progress to walking with eyes open while repeatedly turning their head, progressing further from a wide stance to a narrow stance.11 Ultimately, patients progress to standing on an unstable surface, such as foam, with feet together and eyes open and may perform dynamic reaching tasks, followed by a final progression to eyes closed.11 More advanced agility drills may be incorporated to further progress young athletic populations for return to sport or work.

**Central Vestibular Dysfunction**

**Vestibular Nuclei Damage**

 Concussive damage to the vestibular nuclei or damage along higher pathways communicating to the vestibular nuclei also present as vestibular dysfunction, with similar symptoms of dizziness, nausea, vomiting, postural instability, and balance impairment.4 Although outcome measures such as the VOMS, DVAT, MSQ, SOT, CTSIB, and computerized posturography may demonstrate consequential vestibular dysfunction, computerized tomography and functional magnetic resonance imaging may more discretely identify damage and recovery at vestibular nuclei or along higher pathways.4,9

**Vestibular Dysfunction and Pharmaceutical Interventions**

 It is worth noting that vestibular suppressants should no longer be relied upon for the treatment of post-concussive vertigo, as these suppressants actually delay recovery.12 Thus suppressants have been and must continue to be replaced by vestibular rehabilitation techniques, similar to those outlined above, which are proven to reduce dizziness and improve balance and gait in both children and adults.13

 Additionally, tricyclic antidepressants are commonly prescribed to aid sleep and reduce headaches attributed to PCS, despite lack of evidence that these or any other medications significantly restore function in humans.1 Thus, rehabilitation appears better supplemented by educational sleep hygiene interventions.14

**Conclusion**

Although much of the research focusing on vestibular dysfunction is focused on pathology, many case studies, case series, and randomized clinical trials are available to inform best practice. However, most of these studies are not exclusive to PCS, requiring extrapolation of results to formulate conclusions in the treatment PCS. To provide the most informed recommendations, several systematic reviews have synthesized the effects of these and other PCS-specific vestibular studies. While a metanalysis of existing PCS-specific vestibular randomized controlled trials would confirm assumptions and fill gaps in current recommendations, current evidence supports the efficacy of aforementioned outcome measures and interventions in reducing vestibular PCS symptoms and impairments.

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