VESTIBULAR DYSFUNCTION (AFTER BRAIN INJURY)

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1. DISEASE/DISORDER:

Definition

Vestibular disorders arise from damage to the peripheral and/or central vestibular system and can cause balance deficits, vertigo, dizziness, vision impairments and/or auditory changes. The primary focus of this article is on vestibular disorders following brain injury.

Etiology

In the general population vestibular disorders are most commonly caused by a traumatic brain injury, an infection (viral), and aging. Vestibular dysfunction after a traumatic brain injury (TBI) is the result of peripheral injury and/or central injury.

Epidemiology including risk factors and primary prevention

Reports of dizziness, changes in balance and/or coordination have been estimated to be 15-78% in mild TBI. In blast-related TBI, the incidence of vestibular deficits has been reported in 30% of military personnel. The incidence could be as high as 100% when the patient has sustained a temporal bone fracture. Vestibular symptoms experienced are most commonly attributed to the coexisting brain injury as opposed to a result of a direct injury to the vestibular system.

Patho-anatomy/physiology

The vestibular system controls balance via the peripheral and central system, which can both be affected by a TBI.

1. Ocular motor control and perceptions of motion (Peripheral system)

The labyrinth is located in the temporal bone, which houses three semicircular canals (lateral, posterior, superior) and responds to angular accelerations/decelerations in all planes, and two otolithic organs (the utricular and saccular macula) which control response to linear acceleration/deceleration, including gravity. Together they adjoin the cochlea and are both innervated by cranial nerve (CN) VIII. CN VII is also located in this area and is vulnerable to trauma, such as a temporal bone fracture.

Benign Paroxysmal Positional Vertigo (BPPV)

Due to alteration in mechanics of the semicircular canals caused by free-floating calcium debris (canalithiasis) which creates a pathologic sensitivity to gravity.

Labyrinthine Concussion

Caused by metabolic deterioration and cell death after a violent movement of fluids and tissues within the labyrinth.

Temporal Bone Fracture

Due to the close anatomical location, longitudinal temporal bone fractures can travel along the external auditory canal, middle ear, and anterior to the labyrinth, terminating in either the foramen lacerum or foramen ovale. Transverse fractures travel through the petrous bone between the foramen magnum and foramen lacerum and disrupt the peripheral vestibular system and internal auditory canal. Mixed or oblique fractures have been noted in penetrating trauma.

Perilymphatic Fistula

An aberrant communication between the middle and inner ear allowing backflow of endolymph. It can be congenital, traumatic or spontaneous in nature.

2. Postural and motor system control (Central system)

Input regarding head movement sensed by the peripheral vestibular organs is relayed from CN VIII to the central nervous system (CNS). Three tracts originate from the vestibular nuclei, the medial and lateral vestibulospinal tracts and the reticulospinal tract. The medial vestibulospinal tract is a contributor to the cervical vestibulospinal tracts and the others contribute to the coordination of the head and upper body movement with lower extremities. The cerebellar cortex influences lower motor pathways via the corticospinal tract and the extra-pyramidal system.

Disease progression including natural history, disease phases or stages, disease trajectory (clinical features and presentation over time)

Peripheral Disorders

Benign Paroxysmal Positional Vertigo

Patients experience frequent bouts of paroxysmal vertigo lasting less than one minute, as well as nystagmus, lightheadedness and a tendency to fall.

Labyrinthine Concussion

Sudden onset of continuous vertigo and hearing loss after trauma in the absence of a temporal bone fracture. Vertigo can improve to last less than five minutes over days and becoming position-provoked. Hearing may improve.

Temporal Bone Fractures

Presentation, prognosis and duration varies depending on the axis and severity of the fracture. Patients may experience hearing loss, vertigo, imbalance, sanguinous otorrhea and extreme pain. Hearing loss secondary to external and middle trauma is conductive, often reversible and amenable to surgical therapy. Sensorineural hearing loss is usually permanent.

Perilymphatic Fistula

Episodic vertigo and/or hearing loss provoked by sneezing, lifting, straining, coughing, and loud sounds. Clinical tests are insensitive. Can recur in 10% of individuals affected despite surgical intervention.

Central Disorder

Direct Trauma to Brainstem/Cerebellum

Auditory function will usually be spared. Symptoms include nausea, vomiting, nystagmus, and disequilibrium with preservation of the vestibulo-ocular reflex.

Specific secondary or associated conditions and complications

Migraines have been a source of vertigo when an aura is present. Anxiety and post-traumatic stress disorder (PTSD) can also be associated with vestibular dysfunction. Other psychological factors may be associated with symptoms suggestive of vestibular dysfunction.

2. ESSENTIALS OF ASSESSMENT

History

Obtaining a detailed history can differentiate various vestibular disorders. Many disorders change over time and understanding the first episode of dizziness assists with diagnosis. Quality of the vertigo, mechanism and severity of injury, associated or aggravating factors, concomitant otologic symptoms, and other neurologic symptoms associated should be assessed. The Dizziness Handicap Inventory can be to quantify the effects and perception of dizziness as a handicap by quantifying on their functional, physical and emotional well-being.

Physical examination

Examination of vertigo includes a comprehensive neurologic exam, observation for spontaneous nystagmus, bedside vestibulo-ocular reflex (VOR) testing, Dix-Hallpike maneuver (which can be both diagnostic and curative in BPPV), postural sway on firm and soft surface, and stepping test. Other special tests include an ocular-motor exam, an otologic exam including pneumatic otoscopy, and bedside hearing assessment including finger rub, Weber's and Rhinne test, and disequilibrium with preservation of the VOR. The Dynamic Visual Acuity Test (DVAT) can also be used to assess visual acuity during head movement relative to baseline static visual acuity.

Functional assessment

The Functional Balance Assessment can be used to determine function and risk of falling. Other tests include Dynamic Gait Index, Clinical Test of Sensory Integration and Balance, Berg Balance Scale, Modified Gait Abnormality Rating Scale, Physical Performance Test, Gait Speed and Fives Times Sit to Stand. These tools provide subjective and objective measurements of patients' functional status and determine if they are improving.

Imaging

Brain imaging is commonly ordered in patients complaining of dizziness and vertigo in the setting of TBI. A CT scan can rule out temporal bone fractures as well as a large mass. MRI is the test of choice for small lesions, if indicated.

Supplemental assessment tools

Vestibular Testing:

- 1. Electro-oculography (EOG).
- 2. Video-oculography (VOG)
- 3. Saccades
- 4. Smooth pursuit
- 5. Optokinetic nystagmus
- 6. Caloric testing
- 7. Rotational testing
- 8. Posturography
- Vestibular-evoked myogenic potentials

Early predictions of outcomes

In high school football players, dizziness at the time of injury was associated with a 6.34 odds ratio of developing a protracted recovery from concussion.

The DVAT and DHI can be used as reliable outcome measures in evaluating the progress of patients with balance disorders associated with TBI.

3. REHABILITATION MANAGEMENT AND TREATMENTS

Available or current treatment guidelines

Vestibular disorders after traumatic brain injury are often treated with a combination of medications, vestibular and balance rehabilitation therapy (VBRT) and sometimes with surgery. VBRT promotes functional balance recovery and compensation by using existing neural mechanisms for adaption, plasticity and compensation. Patients with uncompensated and stable (nonfluctuating) vestibular function where symptoms provoked by head motion or environmental cues will likely improve with vestibular rehabilitation.

Types of VBRT:

- 1. Habituation Repeatedly exposing one to a noxious stimulus (e.g., head movements), reducing symptoms to that stimulus.
- 2. Adaption Uses head movements to produce long-term plastic changes in the neural response, improving postural changes and decreasing symptoms.
- 3. Substitution Uses alternative strategies for gaze stability and postural control.
- 4. Dix-Hall Pike Series of positioning maneuvers designed to move offending otoconia out of the involved canal.

Medications are also used to suppress vestibular symptoms of vertigo, lightheadedness and imbalance. These medications should be utilized short term or "as needed," as they can significantly slow the natural compensation process and effectiveness of VBRT.

- 1. Antihistamines (Meclizine & Promethazine) Usually the drug of choice. Meclizine is safest to use in pregnant patients.
- 2. Anticholinergics (Scopolamine)
- 3. Phenothiazine (Prochlorperazine)
- 4. Benzodiazepines (Diazepam, Lorazepam & Clonazepam) Those with prostatism and glaucoma are given benzodiazepines over anticholinergics, despite sedating side effects.

Surgery is uncommon, but considered in temporal bone injuries and perilymphatic fistulas.

At different disease stages

Medications are effective in suppressing vertigo in the acute and early stages of VBRT.

With temporal bone fractures, surgery can be considered after imaging rules out dural or brain exposure to avoid external canal trauma manipulation. Once ruled out, the ear can be microdebrided or the post-traumatic ear canal stenosis can be surgically repaired to improve hearing. Perilymphatic fistulas are intially treated with bed rest, head elevation and avoidance of straining. Surgical exploration can be performed if hearing fluctuates, or vertigo continues for greater than 2-3 days with conservative treatment.

Coordination of care

Cognitive deficits, such as impaired memory/concentration, difficulty in language comprehension, poor judgment and mood lability may hinder successful treatment in patients with severe traumatic brain injury. VBRT are a supplement to the comprehensive multidisciplinary program that patients with brain injuries should receive and clinical judgment should be used to adapt the basic exercise program for the individual patient.

Patient & family education

Patients and family members should be educated that vestibular exercises provoke symptoms of vertigo, dizziness and disequilibrium and that the exercises are beneficial in their recovery. Medications can be used for short-term symptomatic relief and exercises can be tailored to help patients better tolerate symptoms. The intensity of exercises should be increased to provide long-lasting benefits. Those who are unsteady are at a higher risk for falls and environmental modifications should be recommended, such as improving lighting, removing throw rugs, or installing hand railings. Family involvement is essential in a successful home exercise program.

Emerging/unique Interventions

Factors that affect outcome of VBRT include:

- 1. Age
- Combined central or peripheral vestibular disorders
- 3. Duration and chronicity of illness
- 4. Medical comorbities such as diabetes, kidney disease or liver disease
- 5. Migraines

- 6. Neck dysfunction
- 7. Ongoing litigation
- 8. Peripheral neuropathy
- 9. Preexisting eye movement disorders such as strabismus or amblyopia
- 10. Psychiatric disorders

4. CUTTING EDGE/EMERGING AND UNIQUE CONCEPTS AND PRACTICE

Use of alternative sensory input (such as vibratory or auditory feedback) to improve balance and gait in vestibular dysfunction is being explored further.

5. GAPS IN THE EVIDENCE-BASED KNOWLEDGE

Gaps in the evidence-based knowledge

There is a lack of comprehensive studies that identify VBRT outcomes in patients with traumatic brain injury. Some studies suggest that vestibular exercises can reduce symptoms and improve function up to 85% of the time.

REFERENCES

Bibliography

Daroff RB. Bradley's Neurology in Clinical Practice. 6th ed. St. Louis, MO: WB Saunders; 2012:644-664.

Furman JM, Cass SP, Whitney SL. Vestibular Disorders: A Case Study Approach. 3rd ed. New York: Oxford University Press; 2010.

Ropper A, Samuels M. Adams and Victor's Principles of Neurology. 10th ed. Philadelphia: McGraw Hill; 2014: Ch. 15, pp 290-316.

Zasler ND, Katz DI, Zafonte RD. Brain Injury Medicine Principles and Practice. 2nd ed. New York: Demos Medical Publishing; 2013:779-793.

Schultz BA, Cifu DX, McNamee S, Nichols M, Carne W. Assessment and treatment of common persistent sequelae following blast induced mild traumatic brain injury. *NeuroRehabilitation*. 2011;28(4):309-20.

Lau BC, et al. Which on-field signs/symptoms predict protracted recovery from sport-related concussion among high school football players? *Am J Sports Med.* Nov 2011;(39):2311-2318.

Gottshall K, Drake A, Gray N, McDonald E, Hoffer ME. Objective vestibular tests as outcome measures in head injury patients. *Laryngoscope*. 2003; 113(10):1746-50.

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Author Disclosure

Rosanna Sabini, DO Nothing to Disclose