Vestibular Consequences of Mild Traumatic Brain Injury (mTBI)

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Introduction

The vestibular organs are crucial for motion sensation and maintenance of balance. Imbedded in the temporal bones, they are well protected and yet vulnerable to concussive shock with abrupt force applied to the head through blunt trauma or overpressure from blasts. A variety of injuries can occur to the vestibular organ with traumatic brain injury (TBI), both acute and chronic. Dysfunction of the vestibular organs results in continuous or intermittent vertigo and reduction of balance, increasing the risk of falls. In addition, injury can occur in a number of places in the central vestibular pathway, which can also cause impairment in balance function and equilibrium.

It is our opinion that vestibular dysfunction is often unrecognized after TBI, due to the attention paid to primary injuries to the scalp, skull, and brain. Rapid recognition of the possibility of vestibular disorders after TBI should lead to screening for these problems and their prompt treatment. The great advantage of such screening and recognition is that appropriate treatment can often be immediately rendered. For example, lack of identification of the presence of benign positional vertigo (BPV) can mean that as a head injury patient tries to mobilize they are struck with debilitating vertigo. This vertigo can result in falls and drastic exacerbation of patients' other symptoms, such as headache and memory loss. The patient is thus bedridden or mobility impaired for a long periods, even months. Fortunately, a simple treatment, the canalith repositioning maneuver [1], can immediately cure BPV and hugely improve mobilization and even mental status. Other vestibular disorders can also be detected and managed expeditiously to improve recovery. For example, a study of blast-injured service members in Operation Iraqi Freedom demonstrated improvement if their blast-triggered migraine-related vertigo was diagnosed and treated [2].

For the purposes of this chapter, we will confine our discussion to mild traumatic brain injury (mTBI), otherwise known as concussion. mTBI is the most common disorder seen in the wars in Southwest Asia and is increasingly becoming a more important topic due to the number of sports-related episodes of mTBI [2, 3]. The symptoms

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of this disorder can be myriad, but one of the most common is dizziness. Assessment for vestibular disorders should be part of standard clinical doctrine for acute and chronic management of head-injured patients. In this chapter, we will review this assessment from an anatomic and physiologic point of view and for the appropriate clinical approaches. We will briefly outline treatment approaches to the various disorders.

Vestibular Anatomy and Physiology

The vestibular organs are simply accelerometers that provide information to the brain about the motion of the head. Inside the utricle and the macula are the otolith organs. The otoliths are calcium carbonate crystals fixed in a gelatinous matrix that rest on hair cells. The otoliths are detectors of linear acceleration, either motion in a straight line or slow tilting of the head relative to horizontal. In contrast, the semicircular canals (SCC) are rotational or angular accelerometers. They are hollow and fluid-filled. Inertia of fluid in the canals as the head turns results in the deviation of the cupula, the acceleration sensor in each canal. Signals from the hair cells in the otoliths and the SCCs are transmitted along the vestibulocochlear nerve, in parallel with the signals from the cochlea that encode sound stimuli. In the brainstem, vestibular signals are combined, modulated, and adapted by cerebellar circuits. Disruption of the otoliths, SCCs, vestibulocochlear nerves, and brainstem circuits are all potential sites of dysfunction of the vestibular system. Active disturbance of these systems results in vertigo, and damage to the systems mean loss of acceleration information to the brain and loss of balance. Understanding of the pathophysiology, loss of function, and neural adaptation of the vestibular system is key to the management of TBI-induced disorders.

Mild Traumatic Brain Injury (mTBI) from Blunt Versus Blast Trauma

In this discussion we will examine two types of mTBI. We will first look at mTBI secondary to blunt head injury (closed head injury); then we will examine the vestibular disorders associated with mTBI seen after blast.

Blunt head injury is by far the most common cause of mTBI in the civilian world and is receiving increased attention due to sports-related etiologies. Such sports-related injuries can occur in high-profile professional athletes as well as the young soccer prodigy playing at the local park on Saturday morning. Work in our laboratory over the last several years has allowed us to characterize the neurosensory symptoms as a whole and individual vestibular disorders that were seen after closed head injury [4–6].

There are five well-described symptom clusters that individuals who have suffered mTBI will likely fall within [6]. These include (1) dizziness/mild cognitive impairment, (2) post-traumatic headache/migraine, (3) emotional/affective, (4) fatigue/malaise, and (5) nausea. The mTBI patients that fall within the dizziness/mild cognitive impairment cluster describe symptoms of balance problems, dizziness, difficulty concentrating, difficulty remembering, confusion, and blurred vision. There are also well-known differences between each sex, with more males exhibiting dizziness/mild cognitive impairment symptoms and more females exhibiting post-traumatic headache/migraine symptoms. Other factors likely play a role, such as the higher prevalence of headaches in women, but all of these symptom clusters are important to consider when assessing a recently injured patient in the emergency room or clinic setting.

Table 1 shows the characteristics of the four classes of balance disorders seen after blunt trauma. Post-traumatic benign positional vertigo (PTBPV) is identical to idiopathic benign positional vertigo. It is characterized by short episodes of vertigo that occur when changing head or body position (rolling over in bed, looking up, etc.). The episodes last only a few seconds. PTBPV is discussed in more detail below. Post-traumatic exercise-induced dizziness (PTEID) is dizziness that occurs after the completion of physical activity. These individuals complain of unsteadiness or feeling off balance after they finish a period of physical exertion. They do not generally complain of vertigo. The third class of dizziness seen is post-traumatic

Table 1 Vestibular disorders after closed head injury

Entity	History	Physical exam	Vestibular tests
Positional Vertigo (PTBPV)	Positional Vertigo	Nystagmus on Dix-Hallpike test or modified Dix-Hallpike test	No other abnormalities
Exertional Dizziness (PTEID)	Dizziness during and right after exercise	Abnormalities in challenged gait testing	No other abnormalities
Migraine-associated dizziness (PTMAD)	Episodic vertigo with periods of unsteadiness Headaches	Abnormalities in challenged gait testing +/− Abnormalities on head impulse testing. Normal static posture tests	VOR gain, phase, or symmetry abnormalities. High-frequency VOR abnormalities Normal posturography
Spatial disorientation (PTSpD)	Constant feeling of unsteadiness worsened by standing but still present when sitting or lying down Drifting to one side while walking. Shifting weight when standing still	Abnormalities on standard gait tests +/− Abnormalities on head impulse testing Abnormalities on static posture tests	VOR gain, phase, or symmetry abnormalities. High-frequency VOR abnormalities Abnormal posturography Central findings on rotation chair testing

migraine-associated dizziness (PTMAD). In this classification, which has received increasing attention over the last several years, individuals complain of a variety of transient types of dizziness. Individuals can have vertigo, unsteadiness, or visual flow abnormalities. The episodes are intermittent and can last from seconds to hours. Most have more than one type of dizziness episode. In this disorder, migraine headache (either coincident with or distinct from the dizziness) is one of the hallmark symptoms. PTMAD is discussed in more detail below. The final class of dizziness seen after blunt head trauma is post-traumatic spatial disorientation (PTSpD). In this symptom complex, individuals complain of unsteadiness when standing still or moving quickly. They also have unsteadiness on uneven surfaces or when walking in poor light conditions. Similar to the migraine-associated dizziness patients, this group of individuals may have headaches, but unlike that group, headaches are rarely one of the dominant symptoms. The hallmark of this condition is the need to use light touch when standing still to avoid wobbling. We have been able to describe the frequency of these disorders, and these data are shown in Fig. 1. It should be noted that the frequency of PTBPV is likely underestimated in this group since many of these individuals may have resolved the BPV prior to presenting to clinic.

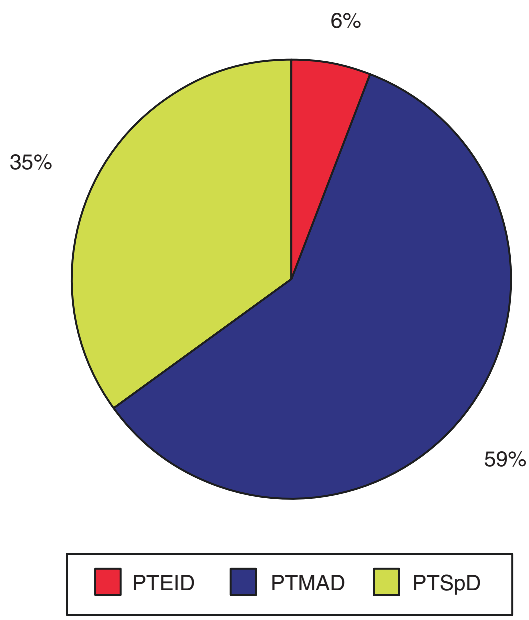

Fig. 1 Comparisons of dizziness. Blunt head trauma

While blast-related mTBI may seem less relevant, it is becoming an increasingly important etiology of mTBI. Well over 80% of all war injuries are blast-related mTBI in isolation. In the civilian world, air bags, compressors, pneumatic tools, and a number of other job site risks have resulted in a sharp rise in the number of blast-related mTBI cases. Dizziness is the leading symptom of blast-related mTBI [2]. Blast-induced mTBI differs from blunt mTBI in a number of ways [5]. The

Table 2 Balance disorders seen after blast exposure

Entity	History	Physical exam	Vestibular tests
Positional Vertigo (PBBPV)	Positional vertigo	Nystagmus on Dix-Hallpike test or modified Dix-Hallpike test	No other abnormalities
Exertional dizziness (PBED)	Dizziness during exercise	Abnormalities in challenged gait test	No other abnormalities
Blast-induced disequilibrium (PBD)	Constant feeling of unsteadiness when standing and walking worse with challenging environments Constant headache	Abnormalities in challenged gait Abnormalities in tandem Romberg Abnormalities with quick head motion	Abnormal posturography Abnormal target acquisition, dynamic visual acuity, and gaze stabilization +/-VOR gain, phase, or symmetry abnormalities
Blast-induced disequilibrium with Vertigo (PBDV)	Constant feeling of unsteadiness when standing and walking worse with challenging environments Constant headache Episodic vertigo	Abnormalities in challenged gait Abnormalities in tandem Romberg Abnormalities with quick head motion	Abnormal posturography Abnormal target acquisition, dynamic visual acuity, and gaze stabilization VOR gain, phase, or symmetry abnormalities

classes of dizziness demonstrate the differences between blast and blunt TBIs effects. Table 2 shows the classes of dizziness seen after blast-induced mTBI. The post-blast benign positional vertigo (PBBPV) is identical to that of PTBPV with transient positional-induced vertigo episodes. On the other hand, post-blast exertional dizziness (PBED), which was formerly termed post-blast exercise-induced dizziness, is dramatically different from the PTEID in that post-blast individuals get unsteady upon starting to exercise (rather than at the completion of the episode). The symptoms of unsteadiness and disequilibrium as well as headaches are the same but the temporal relationship of these symptoms to the exercise is much different and, hence, more troubling to the patient. The final two classes, post-blast dizziness (PBD) and post-blast dizziness with vertigo (PBDV), are characterized by the following two symptoms – constant unsteadiness, which is made worse by more challenging balance environments (uneven surfaces, poor light conditions, moving quickly, etc.) and constant headaches which fluctuate in severity. The presence of additional episodic vertigo separates the two disorders. The relative frequency of these dizziness types is shown in Fig. 2. Unlike after blunt head injury, the frequency of PBBPV, while likely slightly higher than zero, is very small. The classification systems have proved helpful in a variety of ways. They can be understood and are essential to guide treatment and rehabilitation. They also provide prognostic details that help in patient management. Equally as important is that they provide a diagnosis for patients who have too often been told that the dizziness is "something they got from the head injury" and "give it time – it will go away."

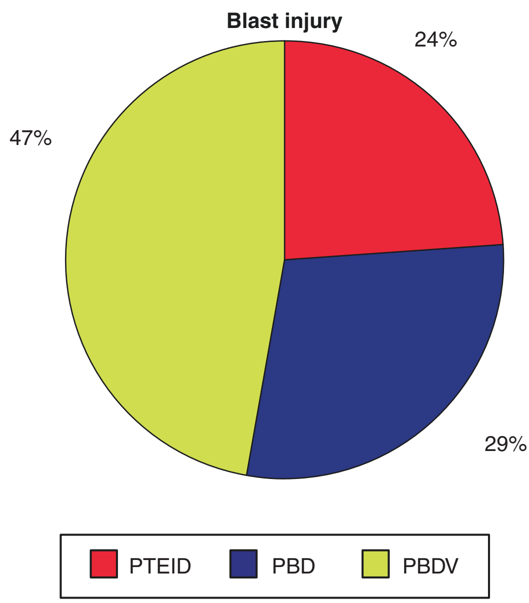

Fig. 2 Comparisons of dizziness. Blast injury

Post-traumatic Benign Positional Vertigo (PTBPV)

BPV is the most common condition causing vertigo. BPV is simply the result of calcium carbonate crystals broken loose from the otolith organ ending up in the semicircular canals. Clinicians involved in the diagnosis or treatment of patients that present with vertigo or imbalance should know how to elicit a history of BPV and carry out the Dix-Hallpike test for the diagnosis. The canalith repositioning maneuver (CRM or, as it is commonly known, the Epley maneuver) is a simple, safe procedure that can immediately cure BPV. If one is familiar with the diagnosis of BPV, the CRM is a powerful addition to one's armamentarium. A full description of the Dix-Hallpike test and the CRM is given in Viirre and colleagues [7]. In summary, one looks for a history of brief vertigo attacks that are provoked by head movements: rolling over in bed, bending over, or reaching up. The vertigo lasts seconds and should stop as soon as the patient lies still. Note that motion sickness and imbalance from a spell of BPV can last for minutes or hours after the actual vertigo episode. The Dix-Hallpike test is carried out with a patient on an examining bench. While being held securely by the examiner, the patient's head is turned 45 degrees to the right or left. The patient is then thrust backward rapidly. Onset of a vertigo sensation accompanied by torsional nystagmus (a clockwise or anticlockwise rotation of the eyes as the patient looks straight ahead) is diagnostic. The CRM is a simple continuation of movements once a positive Dix-Hallpike test is elicited. With the head extended back so it is below the horizontal plane, a sequence of turns of the head and body will remove the offending calcium carbonate crystals from the semicircular canal.

Post-traumatic Migraine-Associated Dizziness (PTMAD)

Migraine is one of the most common genetic disorders present in approximately one in six women and one in ten men. Migraine is a disease complex of which headache is only the most common symptom. Migraine aura is well recognized and its presence is diagnostic of migraine. Almost half of migraineurs have dizziness and vertigo episodes [8]. The high incidence of migraine in the general population suggests that a high percentage of people with TBI will have concomitant migraine, even if they were not symptomatic prior to their injury. The physical and emotional stress of TBI – and perhaps the release of neurohumoral factors during the injury – are powerful triggers for migraine symptoms. The post-traumatic headache, dizziness, cognitive difficulties, and symptoms not localized to the head may well be present in TBI patients as the result of activation of migraine.

Migraine headache is diagnosed by using the International Headache Society criteria for headache [9]. There are no diagnostic criteria for migraine-related dizziness, but vertigo in a patient who meets the migraine headache criteria must have migraine considered in the differential diagnosis. Because of the variable penetrance of the condition, review for a family history of recurrent headaches, dizziness, and/or motion sensitivity may be fruitful, even if a formal diagnosis of migraine is not reported in the family.

Treatment of migraine can be effectively carried out by lifestyle and medical management. In TBI patients, particular attention must be paid to provision of adequate regular sleep, regular meals, and a well-designed activity program (see below). Medical management includes use of beta-blockers (propranolol) and carbonic anhydrase inhibitors (topiramate). Topiramate in particular has been studied in the TBI dizziness population and has been found to be effective not only for the dizziness and vertigo, but also for headache control in patients.

Diagnosis

Aside from a thorough medical history and a standard vestibular physical exam, there are recently described techniques to more accurately diagnose mTBI. These specialized vestibular function tests are identical to the standard clinical vestibular exam but instead objectively measured with infrared goggles and standardized visual stimuli [10]. These tests can be separated into oculomotor tasks (vertical and horizontal smooth pursuit, vertical and horizontal saccades, antisaccade, predictive saccade, optokinetic response, saccade-reaction time test), vestibular tasks (head impulse test [HIT], subjective visual vertical and horizontal), and oculovestibular reaction time (OVRT) tasks.

A test battery consisting of a subset of these tests (antisaccade [increased error rate percentage], predictive saccade [decreased absolute number], and HIT tasks [increased absolute gain symmetry, decreased average gain]) can sensitively and specifically (89% and 95%, respectively) identify individuals with acute mTBI [10]. These tasks reflect underlying pathophysiologic differences in individuals who have suffered acute mTBI compared to healthy individuals. The increased antisaccade error rate suggests impaired inhibitory contributions of frontal cortical regions and GABAergic output from various brain regions [11]. The abnormal HIT results are presumed to be a result of disruption to neuroanatomical pathways involving the vestibular nuclei, related cerebellar connections, and direct projections from the oculomotor, trochlear, and oculomotor nuclei. Other sets of these tests yield similar specificity and sensitivity measurements and have been formatted to work on a pair of portable goggles. This portable system provides objective, point-of-injury testing and should also yield the best prognostic information for return to play as well. This body of recent work has begun to show that objective vestibular testing is considered to be an efficient and effective method to determine the presence or absence of mTBI.

Treatment

The vestibular physical therapy rehabilitation strategy employs specific exercises designed to decrease dizziness, increase balance function, and increase general activity levels. Exercises to decrease dizziness focus on exposure to specific stimuli for habituation or attenuation of the dizziness response in the brain. Balance retraining involves exercises designed to improve organization of sensory information for balance control and coordination of muscle responses. General activity exercise involves a daily aerobic exercise program of progressive walking, cycling, or swimming.

A vestibular physical therapy (VPT) program for mTBI patients consists of exercise procedures that target the vestibulo-ocular reflex (VOR), cervico-ocular reflex (COR), depth perception (DP), somatosensory retraining (SS), dynamic gait, and aerobic function. The VOR, COR, and DP exercises are graded in difficulty, based on velocity of head and object motion and progression of body positioning from sitting to standing to walking. The SS exercises are graded in difficulty by narrowing the base of support, making the surface uneven, or changing the surface from firm to soft. Large amplitude head and trunk movements are also employed to increase somatosensory input. These exercises include the proprioceptive neuromuscular facilitation techniques of slow reversal head and neck patterns, modified chopping and lifting for head and trunk in progression from supine to sitting to standing postures, and total body mass rolling activities. Varied walking exercises are graded in difficulty by changing direction, performing with the eyes closed, increasing speed of ambulation, walking on soft surfaces, or navigating stairs. An aerobic exercise home program progressively increases the time, speed, or distance that the patient can tolerate. All persons are encouraged to work at their maximum tolerance while performing the VPT. Patients are instructed to perform the exercises twice daily at home. Patients are monitored by the physical therapist twice the first week and once a week for the subsequent 7 weeks. Patient compliance to the home exercise program is surveyed by the physical therapist during patient visits.

An objective assessment is performed for all mTBI patients by the vestibular physical therapist. A functional test battery consisting of an impulse head thrust test, Fukuda step test, Romberg test, tandem Romberg test, and Dynamic Gait Index (DGI) [12] is administered to each patient. In addition, the Dizziness Handicapped Index (DHI) [13] and the Activities-Specific Balance Confidence Scale (ABC) [14] surveys are administered. The above measurements are obtained pre-treatment, during treatment, and post-treatment (6–8 weeks after beginning treatment). Subjective patient reports of degree and length of imbalance perception are documented throughout treatment. The length of time required for patients to return to work after the initiation of physical therapy is monitored.

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As we have noted, vestibular complaints are the most frequent sequelae of blast-induced mTBI [1]. VPT has been established as the most important treatment modality for this group of patients. Nevertheless there is little work objectively documenting the impact of VPT on this group of patients. Studies have been completed in the past examining clinical measures, like the Glasgow Coma Score (GCS), on overall recovery pattern after TBI, but outcome measures specifically aimed at examining the adequacy of vestibular tests to track vestibular recovery have remained lacking. Scherer and Schubert reinforced the need for best practice vestibular assessment for formulation of appropriate VPT treatment strategies [15]. Now the application of vestibular testing and rehabilitation in this patient population is needed to provide information on objective outcome measures [15]. VPT is most effective when applied in a customized fashion. While we and others have developed VPT procedures that are applied in "best practices" for blast mTBI vestibular patients, these therapies must be customized for the patient entry level of function and expectation level of recovery. Knowledge of the patient's disability and diagnosis is critical to build the foundation for return to activity, work, or sport. There has been documentation on the reliability of both the Center of Dynamic Pressure and the Dynamic Gait Index as diagnostic tools [12, 16–18], but those studies have not looked at the head injury population, which tends to have a different type of vestibular profile than those tested in previous studies. The head injury population is also a younger population than the previous studies represent. Similarly, there are several studies [19–22] examining the GCS as an outcome measure and correlating this with postural stability. In these studies the patient groups were small and again far different from our mTBI blast patients, both in terms of vestibular dysfunction and age. What might be considered normal for an older vestibular patient (post-stroke, etc.) would still be wholly unacceptable in this young military population intent on returning to active duty. Our study represents a demonstration of a suite of vestibular tests successfully utilized to judge outcomes in patients with both blunt and blast-induced mTBI with vestibular disorders. Vestibular clinical centers will establish their own normal levels on patients of similar age and activity level. The standard results of these tests can be used to determine return to duty/work status as well as return to physical activity status. While the entire suite of tests provides valuable information, our data indicate that the vertical GST is the most sensitive outcome predictor for our population. This likely indicates that recovery of vestibular function is frequency and velocity dependent. This observation agrees with the work of Paige [23] in which linearity and symmetry of the VOR were examined.

Advanced Concepts in Vestibular Consequences of TBI

Blunt and blast mTBI have been demonstrated to result in a variety of medical conditions and syndromes. These vary from simple diagnosis and management, such as BPV, to the more complex, such as post-traumatic spatial disorientation. Fortunately, observers can be readily trained to recognize these various conditions and initiate management. Since dizziness is the leading complaint post-mTBI, deployment of formalized protocols and training programs should be activated throughout the military and even in civilian environments, such as organized football, where mTBI is frequent. Variants, such as migraine-related mTBI syndromes should be trained on for recognition and screened for management.

Despite recent work in the area, there is still a great deal of research with respect to mTBI that needs to proceed. Critical among these include deploying known countermeasures for mTBI, determining the pathophysiology of mTBI so even more specific treatments can be developed, studying the effects of multiple blasts and head impacts, and developing diagnostic and therapeutic tools that are mobile, rugged, and easy to use.

References

1. Helminski JO, Zee DS, Janssen I, Hain TC. Effectiveness of particle repositioning maneuvers in the treatment of benign paroxysmal positional vertigo: a systematic review. Phys Ther. 2010;90(5):663–78.
2. Hoffer ME, Balaban C, Gottshall K, Balough BJ, Maddox MR, Penta JR. Blast exposure: vestibular consequences and associated characteristics. Otol Neurotol. 2010;31(2):232–6.
3. McCrory P, Meeuwisse W, Johnston K, Dvorak J, Aubry M, Molloy M, et al. Consensus statement on concussion in sport – the third international conference on concussion in sport held in Zurich, November 2008. Phys Sportsmed. 2009;37(2):141–59.
4. Hoffer ME, Gottshall KR, Moore R, Balough BJ, Wester D. Characterizing and treating dizziness after mild head trauma. Otol Neurotol. 2004;25(2):135–8.
5. Hoffer ME, Donaldson C, Gottshall KR, Balaban C, Balough BJ. Blunt and blast head trauma: different entities. Int Tin J. 2009;15(2):115–8.
6. Hoffer ME, Szczupak M, Kiderman A, Crawford J, Murphy S, Marshall K, et al. Neurosensory symptom complexes after acute mild traumatic brain injury. PLoS One. 2016;11(1):e0146039.
7. Viirre E, Purcell I, Baloh RW. The Dix-Hallpike test and the canal repositioning maneuver. Laryngoscope. 2005;115(1):184–7.
8. Vuković V, Plavec D, Galinović I, Lovrencić-Huzjan A, Budisić M, Demarin V. Prevalence of vertigo, dizziness, and migrainous vertigo in patients with migraine. Headache. 2007;47(10):1427–35.
9. International Headache Society Classification: ICHD-III. https://www.ichd-3.org/.
10. Balaban C, Hoffer ME, Szczupak M, Snapp H, Crawford J, Murphy S, et al. Oculomotor, vestibular and reaction time tests in mild traumatic brain injury. PLoS One. 2016;11(9):e0162168.
11. Munoz DP, Everling S. Look away: the anti-saccade task and the voluntary control of eye movement. Nat Rev Neurosci. 2004;5(3):218–28.
12. Herman T, Inbar-Borovsky N, Brozgol M, Giladi N, Hausdorff JM. The Dynamic Gait Index in healthy older adults: the role of stair climbing, fear of falling and gender. Gait Posture. 2009;29(2):237–41.
13. Jacobson GP, Newman CW. The development of the dizziness handicap inventory. Arch Otolaryngol Head Neck Surg. 1990;116(4):424–7.
14. Powell LE, Myers AM. The Activities-Specific Balance Confidence (ABC) scale. J Gerontol A Biol Sci Med Sci. 1995;50A(1):M28–34.
15. Scherer MR, Schubert MC. Traumatic brain injury and vestibular pathology as a comorbidity after blast exposure. Phys Ther. 2009;89(9):980–92.
16. 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.
17. Mishra A, Davis S, Speers R, Shepard NT. Head shake computerized dynamic posturography in peripheral vestibular lesions. Am J Audiol. 2009;18(1):53–9.
18. Nashner LM, Peters JF. Dynamic posturography in the diagnosis and management of dizziness and balance disorders. Neurol Clin. 1990;8(2):331–49.
19. Whitney SL, Marchetti GF, Pritcher M, Furman JM. Gaze stabilization and gait performance in vestibular dysfunction. Gait Posture. 2009;29(2):194–8.
20. Goebel JA, Tungsiripat N, Sinks B, Carmody J. Gaze stabilization test: a new clinical test of unilateral vestibular dysfunction. Otol Neurotol. 2007;28(1):68–73.
21. Badaracco C, Labini FS, Meli A, Angelis ED, Tufarelli D. Vestibular rehabilitation outcomes in chronic vertiginous patients through computerized dynamic visual acuity and gaze stabilization test. Otol Neurotol. 2007;28(6):809–13.
22. Pritcher MR, Whitney SL, Marchetti GF, Furman JM. The influence of age and vestibular disorders on gaze stabilization: a pilot study. Otol Neurotol. 2008;29(7):982–8.
23. Paige GD. Nonlinearity and asymmetry in the human vestibule-ocular reflex. Acta Otolaryngol. 1989;108(1–2):1–8.