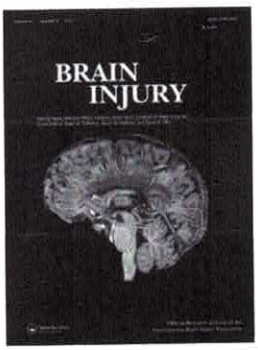

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Vestibular consequences of mild traumatic brain injury and blast exposure: a review

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ABSTRACT

The purpose of this article is to review relevant literature on the effect of mild traumatic brain injury (mTBI) and blast injury on the vestibular system. Dizziness and imbalance are common sequelae associated with mTBI, and in some individuals, these symptoms may last for six months or longer. In war-related injuries, mTBI is often associated with blast exposure. The causes of dizziness or imbalance following mTBI and blast injuries have been linked to white matter abnormalities, diffuse axonal injury in the brain, and central and peripheral vestibular system damage. There is some evidence that the otolith organs may be more vulnerable to damage from blast exposure or mTBI than the horizontal semicircular canals. In addition, benign paroxysmal positional vertigo (BPPV) is a common vestibular disorder following head injury that is treated effectively with canalith repositioning therapy. Treatment for (non-BPPV) mTBI-related vestibular dysfunction has focused on the use of vestibular rehabilitation (VR) augmented with additional rehabilitation methods and medication. New treatment approaches may be necessary for effective otolith organ pathway recovery in addition to traditional VR for horizontal semicircular canal (vestibulo-ocular reflex) recovery.

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Dizziness is a common and sometimes persistent symptom following a concussion or mild traumatic brain injury (mTBI). The term 'dizziness' is often used to describe multiple symptoms including vertigo (illusion of motion), disequilibrium (unsteadiness or imbalance), and lightheadedness (pre-syncope). Post-concussion dizziness presents a clinical challenge because there are multiple causes of dizziness and the clinical management is dependent on the aetiology [1–3]. Damage to the peripheral vestibular (inner ear) system is one potential cause of post-concussion dizziness. In war-related injuries, mTBI is often associated with a blast exposure that can damage the inner ear. Understanding the vestibular consequences of blast-related mTBI is important because TBI is the signature injury of the recent wars in Iraq and Afghanistan and is often associated with blast exposure [4] .

In many individuals with blast-related mTBI, the cause(s) of their dizziness or imbalance is unclear as many studies limit evaluation to a symptom-based questionnaire [5]. A limitation of this method is that non-vestibular disorders may cause dizziness or imbalance, and many individuals who complain of dizziness or imbalance have normal vestibular function. Another shortcoming is that many studies on dizziness (particularly studies of sports-related concussion) have limited their assessment to bedside screening and balance tests. Although loss of vestibular function may result in postural instability, postural stability involves the dynamic interplay between multiple body systems, including the sensory, central nervous, and musculoskeletal systems. Abnormal balance function, therefore, may not be a sensitive clinical indicator of vestibular dysfunction [6].

The purpose of this article is to provide an overview of chronic dizziness related to concussion-mTBI and blast injury with a focus on the vestibular consequences of blast-related mTBI. Specifically, studies that utilized vestibular laboratory testing were reviewed in order to understand the frequency of vestibular dysfunction in individuals with concussion-mTBI-related dizziness. In addition, current literature on the effectiveness of treatment of dizziness in concussion and blast-related TBI is summarized.

The terms 'brain injury' and 'head injury' have been used synonymously, although not all injuries to the head result in TBI, and, previously, the term 'concussion' has been used synonymously with mTBI. In an effort to develop a common definition of TBI, the DoD/VA Definition and Symptomatic Taxonomy Working Group and other joint consensus panels defined TBI as 'a traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately following the event: (1) any period of loss of or a decreased level of consciousness; (2) any loss of memory for events immediately before or after the injury; (3) any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc.); (4) neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.) that may or may not be transient; and (5) intracranial lesion [3]'. The DoD/VA definition of mTBI includes (1) loss of consciousness lasting 0 to 30 minutes, (2) alteration of consciousness/mental state lasting up to 24 hours, (3) post-traumatic amnesia lasting 0 to 1 day, and if available, (4) normal structural imaging, and (5) 13 to 15 on the Glasgow Coma scale [3].

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Mild TBI in combat veterans is often related to blast exposure which can also cause trauma to the inner ear (i.e., peripheral damage). A blast is caused by the detonation of an explosive (e.g., IED) that causes a peak positive pressurization (shock wave) followed in time by a negative pressurization. Primary blast injuries resulting from the impact of the shock wave affect air- and fluid-filled organs such as the lungs and sensory structures of the middle and inner portions of the ears. Secondary blast injuries can result from flying debris and bomb fragments, and tertiary blast injuries can result from the impact with another object when thrown by a blast wind. In general, the severity of blast injury is reduced the farther away the victim is from the blast. Otologic injuries due to blast exposure include tympanic membrane perforations, hearing loss, tinnitus, and otalgia. Although the consequence of blast on the auditory system is well established [7], there is less known about the impact of blast on the vestibular organs. Dizziness and imbalance can also occur following blast exposure [8,9], and histological studies have described damage to the vestibular sensory organs in blast victims [10]. The vestibular sensory organs (semicircular canals and otolith organs) are also located in the inner ear and have sensory epithelia similar in structure and function to the cochlea. Although the sensory epithelia of the semicircular canals (cristae) are encased in the bony labyrinth, the maculae of the otolith organs are located in the vestibule and may be more vulnerable to pressure waves than the cristae [11].

There is some evidence that the majority of individuals with blast-related mTBI experience significant dizziness within 72 hours after the injury [12]. Fortunately, for most individuals acute dizziness resolves within four to thirty days [12]; however, numerous studies have demonstrated that dizziness can last for six months or longer following head trauma in some individuals [13,14]. The cause of chronic post-concussion dizziness is unclear and inconsistent with vestibular compensation or recovery from peripheral vestibular dysfunction [15]. The presence of dizziness at six months after injury is an adverse prognostic indicator and may be the most persistent symptom adversely affecting clinical outcome and disease course [16,17]. Although post-concussion symptoms of dizziness may resolve over time in many individuals with TBI [18], the likelihood of dizziness worsening at three months post injury is greater than other post-concussion symptoms [19].

The diagnosis of dizziness and imbalance is complex as the balance system involves a multi-modal process. The vestibular system is one of three sensory systems (vision, vestibular, and proprioception or somatosensory) that contribute to balance. Information from these three sensory systems is processed at the brainstem (vestibular nuclear complex) and the cerebellum resulting in motor and perceptual outputs. Motor output is primarily mediated via two pathways: the vestibulo-ocular reflex (VOR) and the vestibulo-spinal reflex (VSR). The VOR controls eye movement to keep gaze steady when the head is in motion, and the VSR mediates postural control during head movement. In general, peripheral vestibular loss can occur in one or both labyrinths, in one or both branches of the vestibular nerve, and in one or more vestibular sensory organs. The central vestibular system includes the brainstem and cerebellum but pathways also project to higher centres in the midbrain and cerebral cortex. Injury to any of the vestibular sensory organs (peripheral dysfunction) or central vestibular pathways can cause postural instability, visual blurring, and subjective complaints of dizziness and/or imbalance. Rotational vertigo (spinning sensation) most often implicates the semicircular canal pathways which sense angular acceleration; whereas, damage to the otolith organs (gravito-inertial sensors) may cause unsteadiness and lateropulsion (tilting, pulling, pushing sensations) [20,21].

Central vestibular dysfunction

A common assumption is that dizziness following mTBI is caused by brain injury or central dysfunction [22,23], and diffusion-tensor neuroimaging (DTI) studies have demonstrated white matter abnormalities and diffuse axonal injury in individuals with mTBI [24,25]. Cerebellar DTI abnormalities have been observed in individuals with vestibular symptoms following mTBI, suggesting injury to the central vestibular system [26]. Using susceptibility-weighted imaging and DTI, Gattu et al. [27] described diffuse axonal injuries, microhaemorrhages, or vascular anomalies in four individuals with dizziness and/or imbalance following blast exposure. Interestingly, three of the four individuals also had abnormal peripheral vestibular function.

Ocular motor testing such as smooth pursuit tracking and volitional saccades can be used as a screening measure for determining CNS function independent of peripheral vestibular system function. Abnormal ocular motor function (e.g., saccadic dysmetria, gaze-evoked nystagmus, or saccadic pursuit) can indicate damage to CNS pathways that include the cerebral hemispheres, cerebellum, and brainstem. Fixation suppression of vestibular nystagmus requires intact connections between the cerebellum and vestibular nuclei, and, thus, has been used as a clinical test for central vestibular involvement or visual-vestibular interaction. Specifically, failure of fixation suppression indicates a patient is unable to visually suppress nystagmus by ≥50% of the peak response during vestibular stimulation, and suggests central pathology that may include the parietal-occipital cortex, brainstem, or cerebellum.

Most studies [28–30] suggested that ocular motor abnormalities occurred in ≤8% of individuals with post-concussion dizziness (Table 1); however, a greater prevalence of ocular motor abnormalities has been reported 0–4 days after injury [18]. Abnormal findings varied across studies but primarily included gaze-evoked nystagmus, failure of fixation suppression, and break-up of smooth pursuit. Similar findings have been reported in studies that examined blast-related dizziness (Table 2). Several studies have reported a higher incidence of ocular motor abnormalities in individuals with mTBI or blast exposure; however, these studies did not focus on participants with symptoms of dizziness [32–33]. A limitation of ocular motor tests for detecting CNS pathology is the use of prescribed medications that impact ocular motor function [20,31].

Table 1. Summary of studies examining the frequency (%) of vestibular, balance, and ocular motor dysfunction in individuals with dizziness/imbalance/vertigo following traumatic brain injury (TBI).

	N	TBI severity	Symptom duration	BPPV	Caloric test	Ocular motor tests	cVEMP	SOT
Berman and Frederickson, 1978 [29]	140	44 mild	≤60 mos	14%	6%	3%	DNT	DNT
		96 mod		15%	21%	8%		
Gannon et al., 1978 [74]	50	NA	*X* = 1.9 mos	NA	14%	NA	DNT	DNT
Tuohimaa, 1978 [18]	82	Mild	0–4 days	11%	9%	45%	DNT	DNT
			6 mos	5%	3%	7%		
Davies and Luxon, 1995 [28]	100	72 mild 24 moderate, 4 severe	*X* = 28 mos	15%	51%	8%	DNT	DNT
Ernst et al., 2005 [30]	63	NA	≤24 hrs	57%	19%	5%	DNT	NA*
			X = 1.5 mos	NA	NA	NA	25%	27%
Lee et al., 2011 [75]	28	NA	>1 mos	NA	7%	NA	32%	DNT

TBI = traumatic brain injury; BPPV = benign paroxysmal positional vertigo; cVEMP = cervical vestibular evoked myogenic potentials; mos = months; yr = year; SOT = sensory organization test; NA = data not available, DNT = did not test.
*Only group means provided (no individual data).

Table 2. Summary of studies examining the frequency (%) of vestibular, balance, and ocular motor dysfunction in individuals with dizziness/imbalance following blast exposure.

	N	TBI	BPPV (%)	Caloric test (%)	Ocular motor test	cVEMP	SOT
Shupak et al., 1993 [8]	5	NS	20	40	NA	DNT	DNT
Van Campen et al., 1999 [9]	27	16/27	11	7	4%	DNT	44%
Cohen et al., 2002 [13]	17	NS	6	0	NA	DNT	46%
Akin and Murnane, 2011 [38]	31	15/31	3	23	3%	52%	52%
Scherer et al., 2011 [31]	11	5/11	9	9	45%	29%*	NA**

TBI = traumatic brain injury; BPPV = benign paroxysmal positional vertigo; cVEMP = cervical vestibular evoked myogenic potentials; mos = months; yr = year; SOT = sensory organization test; NA = data not available; NS = not specified; DNT = did not test.
*Based on absent response as cVEMP amplitudes not available.
**Only group means provided (no individual data).

Semicircular Canal/VOR dysfunction

In addition to CNS or central vestibular dysfunction, dizziness/imbalance following mTBI may be related to peripheral vestibular system damage. The peripheral vestibular system is comprised of two types of sensory organs. Three semicircular canals are orthogonally positioned in each inner ear labyrinth and detect angular acceleration during head rotation. Two otolith organs (saccule and utricle) are positioned at right angles in each vestibular labyrinth and contribute to postural and gaze stability by providing sensory input regarding linear acceleration and head tilt (or changes in gravity). The activation of vestibular sensory cells produces an electrical signal that is relayed to the eye muscles via the VOR. The VOR response is often measured clinically by recording eye movement during vestibular stimulation to determine peripheral vestibular function. The binaural bithermal caloric test stimulates the horizontal semicircular canal primary afferents using a temperature gradient that induces a thermal convection current and activates the VOR pathway. Although the test has limitations, caloric testing is considered the gold standard for determining peripheral horizontal semicircular canal/superior nerve vestibular dysfunction [35].

It has been documented that a blow to the head can result in peripheral vestibular hypofunction (or unilateral weakness on the caloric test) [36] and is reasonable to presume that damages to peripheral vestibular system are likely due to the head injury rather than the resulting brain injury. Numerous studies have examined vestibular function in individuals with post-concussion dizziness, and Table 1 summarizes individual vestibular test findings for several studies that have reported individual data. Vestibular function was determined by measuring the VOR/horizontal semicircular canal response using the caloric test in all studies, and the number of individuals with abnormal findings on these tests ranges from 3 to 51%. Most studies demonstrated that horizontal semicircular canal dysfunction occurs in less than a quarter of individuals with dizziness following head injury [29,30]. Davies and Luxon, however, reported a greater incidence of post-concussion horizontal semicircular canal dysfunction (51%) and this finding may be related to the low critical value (>13%) used to define canal paresis for caloric testing [28].

Table 2 summarizes findings for several studies that have reported vestibular function in individuals with blast-related dizziness, and these studies reported that caloric abnormalities occurred in 0–40% of cases. There is some evidence that the incidence of vestibular dysfunction increases with the severity of head injury [29]. In contrast, Davies and Luxon [28] reported that vestibular abnormalities occurred at the same rate in individuals with minor and more severe head injury (although auditory abnormalities occurred more often in severe cases). Although there is evidence that vestibular (horizontal semicircular canal/VOR) dysfunction may resolve over time in some individuals with TBI [18], more longitudinal studies are needed to determine the long-term impact of head injury/blast exposure on vestibular function.

Several studies have examined the impact of mTBI or blast exposure on other tests of VOR function. Using active and passive head impulse testing, Scherer et al. [31] demonstrated

reduced horizontal VOR gain (<0.7) in five of twelve service members with TBI and dizziness. Balaban and colleagues [34] demonstrated significantly lower horizontal VOR gain and greater VOR gain asymmetry on a passive head impulse test in individuals with acute mTBI (non-blast) compared to healthy controls.

Gottshall and colleagues [37] determined that patients with acute mTBI scored higher (i.e., worse) on dynamic visual acuity (DVA) testing compared to healthy controls and recommended the use of DVA test as an outcome measure in the assessment of individuals with dizziness related to TBI. Caution should be exercised, however, in the use of the DVA test as a diagnostic tool, because the score reflects central compensation for vestibular dysfunction and as such is not a pure measure of peripheral vestibular function.

Otolith organ dysfunction

Until recently, the literature on vestibular consequences of head injury was restricted to the assessment of horizontal semicircular canal function via its connections to the eyes (VOR). Peripheral vestibular loss, however, can occur in the inferior and/or superior branches of the vestibular nerve and/or in the semicircular canals and/or the otolith organs. More recently, tests such as vestibular-evoked myogenic potentials are available to measure otolith organ function (i.e., gravitational receptor organs within the inner ear). Cervical vestibular-evoked myogenic potentials (cVEMP) are recorded from the activated sternocleidomastoid muscle in response to sound or vibration. Cervical VEMPs assess the saccular-collic pathway and have been used in only a few studies over the past decade to determine the impact of TBI on otolith organ function. Two studies reported 25 and 32% of individuals with post-concussion dizziness had abnormal cVEMPs, suggesting otolith organ dysfunction in individuals with dizziness following mTBI (Table 1). Akin and Murnane [38] reported 52% of individuals with blast-related dizziness had abnormal cVEMPs (Table 2). Scherer et al. [31] recorded cVEMPs in seven service members with blast/concussion-related dizziness and reported that two (29%) had absent responses. It is interesting to note that otolith organ dysfunction (abnormal cVEMP) occurred more often than horizontal canal (VOR) dysfunction (abnormal caloric test) in each study that employed both semicircular canal and otolith organ assessment. Similar findings have also been reported by Serrador et al. [39] in a group of veterans with blast exposure and/or mTBI using unilateral centrifugation to assess otolith function; approximately 30% of the veterans with blast/mTBI demonstrated unilateral otolith dysfunction without horizontal canal impairments . These abnormal otolith findings are supported by histological and cVEMP studies in humans and animals, which suggest that the saccule (one of the otolith organs) may be particularly susceptible to blast-related damage and noise exposure [11,40–44]. These data suggest that limiting vestibular testing to the assessment of horizontal semicircular function may miss important otolith impairments in patients with dizziness related to mTBI or blast exposure. These findings suggest the need to include cVEMP as a component of the clinical vestibular test battery in this patient population.

Benign Paroxysmal Positional Vertigo (BPPV)

In addition to damage to the sacculo-collic pathway discussed above, a head injury can cause the otoconia (calcium carbonate crystals in the otolith organs that result in gravity sensitivity) to become detached from the utricle (one of the otolith organs) and migrate into a semicircular canal causing BPPV [45,46]. BPPV is characterized by recurrent brief episodes of vertigo (a spinning sensation) associated with changes in head position (e.g., looking up or rolling over in bed). The presumed cause of BPPV is canalithiasis or free-floating otoconial debris in semicircular canal fluid (endolymph), which causes endolymph flow and activation of the vestibular sensory cells inducing nystagmus and vertigo [47]. The studies summarized in Table 1 suggest that 5–57% of individuals with post-concussion dizziness are diagnosed with BPPV. In a cohort of 100 patients, Davies and Luxon [28] reported that BPPV was the most common vestibular disorder following head injury. These findings are consistent with the earlier work of Barber [46] who described BPPV in approximately 25% of 165 individuals who experienced dizziness following a head injury. Similarly, Hoffer and colleagues [12] reported that 28% of individuals with post-concussion dizziness had BPPV.

BPPV is relatively easy to diagnose with the Dix-Hallpike and roll tests to localize the involved semicircular canal [48]. Similar to idiopathic BPPV, post-traumatic BPPV most often occurs in the posterior semicircular canal [49]. Treatment for BPPV is performed by placing the patient in a series of positions with the goal of moving the otoconial debris out of the semicircular canal and into the vestibule [50,51]. Canalith repositioning therapy (CRT) is quick and well tolerated by patients. Its efficacy is well established as several randomized control trials have demonstrated a significant benefit of CRT in a specialized clinic setting [52]. Although non-traumatic BPPV is often resolved with a single treatment, individuals with traumatic BPPV may require multiple treatment sessions to resolve BPPV [49,53,54]. Although BPPV can recur following resolution of symptoms, it is unclear if traumatic BPPV recurs more often than non-traumatic BPPV [53,54]. Two clinical practice guidelines on BPPV are available to the interested reader [48,55].

Balance and gait dysfunction

Postural instability or imbalance is a common symptom in patients with head trauma. Postural stability is a multi-sensory motor task that depends on reliable input from the vestibular, somatosensory, and visual systems. The vestibular system (particularly the otolith organs) provides important information about gravity, and postural control is modulated via the vestibulospinal reflexes. The lateral vestibulospinal tract receives a majority of input from the otolith organs and cerebellum and aids in tonic contractions of the antigravity muscles in the lower extremities.

The sensory organization test (SOT) using computerized dynamic posturography may be the most widely used quantitative clinical test to measure postural stability. The SOT assesses the integration of sensory information for static balance by measuring postural sway under conditions in which visual and somatosensory feedback is altered. The SOT was used as a measure of balance function in several of the studies in Tables 1 and 2, and the findings suggest that 27–52% of individuals with dizziness related to mTBI and/or blast have abnormal postural stability. Numerous studies have examined postural stability using SOT, and demonstrated higher magnitudes of anterior-posterior movement when individuals are deprived of accurate visual cues [56,57]. Similar to the chronic nature of post-concussion dizziness, postural stability can remain abnormal after other neurological symptoms are resolved [9,58]. Some investigators have concluded that abnormal postural stability suggests a multi-sensory or central cause of imbalance [59]. There is emerging evidence that increased postural sway and increased fall risk may occur in individuals with impaired otolith function [21,60,61]. Because the VSR uses otolith input to a greater extent than the VOR, this finding is not surprising and suggests that otolith dysfunction may be one source of post-traumatic imbalance.

Treatment

Vestibular rehabilitation (VR) is the treatment of choice for patients experiencing dizziness and imbalance related to vestibular dysfunction [62,63]. VR typically includes gaze stability exercises, gait and balance training, and general conditioning. VR has also been recommended for patients with dizziness related to mTBI/blast exposure [64]; however, there is conflicting evidence on its effectiveness [65–67]. Most individuals with blast-related mTBI suffer from multiple post-concussion symptoms that may include physical, emotional, and cognitive deficits. These comorbidities may impact the efficacy of VR for individuals with vestibular dysfunction related to mTBI/blast exposure. The use of anxiety management and behavioural and cognitive therapy to augment VR may improve the effectiveness of VR for this population [68].

There is evidence to suggest that otolith organ dysfunction can occur in patients with mTBI or blast exposure. This is important because VR is based upon principles of vestibular adaptation of semicircular canal/VOR input. There is some evidence that combined otolith dysfunction and canal dysfunction does not negatively impact rehabilitation outcomes [69]; however, the study did not include individuals with mTBI or otolith-only dysfunction. In contrast, Basta et al. [66] have shown that traditional VR is not effective for many patients with otolith disorders. Alternative methods of VR, such as using auditory feedback, may be more effective than traditional VR in patients with otolith disorders [66].

Other rehabilitation methods that have been shown to have some usefulness in individuals with mTBI and vestibular symptoms include a virtual reality-based environment task [70] and cervical spine physiotherapy in combination with VR [71]. Pharmaceuticals have also been used to treat individuals with vestibular symptoms following TBI or blast exposure. Betahistine dihydrochloride used in conjunction with VR enhanced recovery from trauma-related vestibular symptoms in a preliminary study [72]. In a randomized double-blind, placebo-controlled study on active duty service members, N-acetyl cysteine was beneficial in reducing the severity and increasing the resolution of post-concussion symptoms including balance dysfunction [73].

In summary, dizziness and imbalance are common sequelae associated with mTBI and these symptoms may last for six months or even longer. In war-related injuries, mTBI is often associated with blast exposure. The causes of dizziness or imbalance following mTBI and blast injuries have been linked to white matter abnormalities, diffuse axonal injury in the brain, and central and peripheral vestibular system damage. There is evidence to suggest that the otolith organs may be more vulnerable to damage from blast exposure or mTBI than the horizontal semicircular canals. In addition, BPPV is a common vestibular disorder following head injury that is treated effectively with CRT. Treatment for (non-BPPV) mTBI-related vestibular dysfunction has focused on the use of VR augmented with additional rehabilitation methods and medication. New treatment approaches may be necessary for effective otolith organ pathway recovery in addition to traditional VR for horizontal semicircular canal (VOR) recovery.

Declaration of interest

The authors report no declarations of interest.

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