



*Funct Neurol.* 2016 Apr-Jun; 31(2): 75–80.

PMCID: PMC4936800

Published online 2016 Jun 30. doi: [10.11138/FNeur/2016.31.2.075](https://doi.org/10.11138/FNeur/2016.31.2.075)

PMID: [27358219](https://pubmed.ncbi.nlm.nih.gov/27358219/)

## Vestibular disorders following different types of head and neck trauma

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### Summary

This review focuses on the published literature on vestibular disorders following different types of head and neck trauma. Current knowledge of the different causes and underlying mechanisms of vestibular disorders, as well as the sites of organic damage, is presented. Non-organic mechanisms are also surveyed. The frequency of occurrence of vestibular symptoms, and of other accompanying subjective complaints, associated with different types of trauma is presented and related to the specific causes. Hypotheses about the pathogenesis of traumatic vestibular disorders are presented, and the knowledge derived from animal experiments is also discussed.

We believe this to be a very important topic, since vestibular complaints in traumatic patients often remain undiagnosed or underestimated in clinical practice. This review article aims to suggest directions for additional research and to provide guidance to both the scientific and clinical practice communities.

**Keywords:** concussion, dizziness, imbalance, traumatic brain injury, vertigo, vestibular

### Introduction

The many published studies on traumatic brain injury indicate that post-traumatic dizziness or vertigo is one of the major complaints following head injuries ([Davies and Luxon, 1995](#); [Luxon, 1996](#); [Nacci et al., 2011](#)). Traumatic brain injury, which is often mild, affects, annually, between 1.8 and 3.8 million individuals in the United States alone ([Alhilali et al., 2014](#)). The causes of vestibular symptoms that appear after mild traumatic brain injury should be clarified, before routinely classifying them as a post-concussion syndrome ([Friedland, 2015](#)).

There are several types of post-traumatic dizziness and its pathogenesis remains controversial ([Brandt, 1999](#); [Shepard, 2013](#); [Tuohimaa, 1978](#)). Likewise, opinions on the important factors to evaluate are numerous, but in general there are three schools of thought. One considers dizziness, as well as the other symptoms of the post-traumatic syndrome, to be of psychogenic origin; according to a second school of thought, post-traumatic dizziness is exclusively of organic origin; finally, the third school emphasizes the importance of factors of both types ([Brandt, 1999](#)). Head trauma can directly damage the vestibular organ or vestibular nerve, as well as the brainstem and the visual and oculomotor

pathways, and lead to vestibular disorders ([Allison and Fuller, 2000](#)). Unfortunately, associated anxiety and secondary gain factors make it difficult to distinguish clearly between organic and psychogenic mechanisms ([Brandt, 1999](#)).

## Head trauma

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The frequency of dizziness and disequilibrium following head trauma is about 40–60% among non-hospitalized patients ([Gannon et al., 1978](#)). Even in cases of mild head trauma dizziness was reported to persist for at least two years in 18% of patients ([Cartledge, 1978](#)). These figures signify that there is a common etiology for a heterogeneous collection of peripheral and central vestibular disorders ([Brandt, 1999](#)). According to [Kushner \(1998\)](#), the organically conditioned dizziness that occurs after head trauma is usually peripheral rather than central in origin. The late onset of the symptomatology can be explained by the slow degeneration that sets in after concussion ([Brandt, 1999](#)). The most frequent peripheral form of vertigo after head trauma is benign paroxysmal positional vertigo (PPV), thought to be due to dislodgement of otoliths from the macula of the utricle ([Brandt, 1999](#); [Shepard, 2013](#)). Clinical experience and the most recent literature show that post-traumatic PPV (canalolithiasis) is usually unilateral, and less frequently bilateral. However, bilateral PPV is recognized to have a post-traumatic etiology. When canalolithiasis is bilateral, paroxysmal vertigo and related nystagmus are more pronounced on one side. The symptoms may persist for between a day and up to more than a year ([Shepard, 2013](#)).

Vestibular dysfunction after labyrinthine concussion has often been ascribed to unilateral microscopic hemorrhages in the labyrinth ([Davies and Luxon, 1995](#)). Labyrinthine concussion is often manifested by vertigo, nausea and/or vomiting, but the vestibular examination focuses on pathological nystagmus that occurs spontaneously and is exacerbated during rapid head movements. Generally, labyrinthine concussion resolves through adaptation over a period of weeks or months, a process known as vestibular compensation ([Shepard, 2013](#)). While vestibular suppressants dramatically improve the symptoms during the early period after trauma, they generally delay compensation and subsequently recovery ([Shepard et al., 1990](#)).

Petrous bone fracture can also lead to direct injury of the vestibular nerve or of the labyrinth ([Brandt, 1999](#)). Experimental head injuries in guinea pigs showed that the vestibular organ is disarranged with lysis, exfoliation and vacuolization of the sensory epithelia ([Zhou et al., 1994](#)).

Loss or reduction of function in a semi-circular canal can also lead to peripheral vestibular symptoms. This may be the result of the injury or also occur as a side effect of medication prescribed after head trauma (e.g., ototoxic aminoglycoside antibiotics) ([Halmagyi et al., 1994](#)). Trauma can also cause vertigo by creating a perilymphatic fistula between the middle and inner ear ([Schessel et al., 2005](#)). Rupture of the oval or round windows of the inner ear may lead to the development of a perilymphatic fistula and provoke inappropriate stimulation of labyrinthine receptors ([Grimm et al., 1989](#)). Such fistulas may even occur following minor head trauma or barotraumas (high altitude or underwater) ([Ildiz and Dündar, 1994](#); [Melamed et al., 1992](#); [Pullen, 1992](#); [Shepard, 2013](#)), strenuous exercise, suppressed sneezing, or air travel ([Gunesh and Huber, 2003](#); [Jaffe, 1979](#); [Kim et al., 2001](#)). Generally the symptoms are dizziness, fluctuating hearing loss, ear pressure and tinnitus, chronic nausea and exertional headaches ([Grimm et al., 1989](#)), and their expression depends on head position, movement or air pressure ([Brandt, 1999](#)). A perilymphatic fistula may, in rare instances, heal spontaneously, whereas a chronic perilymphatic fistula has to be corrected surgically ([Grimm et al., 1989](#)).

Acute evolving vertigo may be associated with even mild traumatic brain injury ([Davies and Luxon, 1995](#); [Fitzgerald et al., 1997](#); [Healy, 1982](#); [Oosterveld et al., 1991](#)). Frequently such patients also complain of headache and have difficulty concentrating (post-concussion syndrome).



Vestibular testing of patients with head trauma shows that head trauma may damage the peripheral and central vestibular structures, simultaneously or separately ([Brandt, 1999](#)). Many authors support the hypothesis that traumatic brain injury with vestibular symptoms affects both the peripheral and central vestibular structures. A recent study on patients after mild head trauma and subsequent vestibulopathy compared radiological findings and clinical assessment — this focused on reduction of cognitive functions, severity of symptoms and time to recovery — in these patients. They were shown to have significant axonal impairment irrespective of the prevailing peripheral vestibular symptoms. These findings support the hypothesis that post-traumatic vestibulopathy has a central axonal injury component ([Alhilali et al., 2014](#)).

Central vestibular syndromes are mostly due to concussion of vestibular nuclei or central vestibular pathways. Direct traumatic damage to the brainstem or cerebellum may be followed by the occurrence of imbalance and transitional vertigo ([Shepard, 2013](#)). All the parts of the brainstem and the cerebellum can be affected, but the mesencephalon somewhat more often ([Brandt, 1999](#)). Symptoms of a central origin may include nausea with non-positional vertigo and imbalance ([Kushner, 1998](#)).

Up to 50% of patients with mild traumatic brain injury develop a post-concussion syndrome ([Miller Fisher, 1966](#); [Rutherford, 1977](#); [Williams et al., 1990](#)) (dizziness, headache, tinnitus, hearing loss, blurred vision, diplopia, anxiety, irritability, depression, emotional lability, a decrease in intentional information processing, and fatigue). These symptoms are attributed to the diffuse microscopic changes that accompany mild concussion, the most common form of closed head injury. Even mild concussion can cause significant attentional and information processing impairments that last for months in the absence of any apparent neurological problem ([Hugenholtz et al., 1988](#)).

Animal experiments have shown that minor head trauma can produce petechial cerebral hemorrhages due to distortion forces, especially in the brainstem and the vestibular nuclei ([Jellinger, 1967](#); [Brandt, 1999](#)).

Head injury can occur through a variety of mechanisms. Even high-impact aerobics has been accused of injuring the vestibular system at different levels, causing dizziness, balance dysfunction, etc. ([Brandt, 1999](#)).

[Kortschot and Oosterveld \(1994\)](#) reported that 258 of 318 patients with whiplash trauma had a central-vestibular dysfunction ([Kortschot and Oosterveld, 1994](#)).

## Whiplash and blast exposure traumas

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Some authors have compared vestibular disorders due to mild traumatic brain injury and those following cervical whiplash, since, in both cases, the complaints can have both a peripheral and a central genesis ([Nacci et al., 2011](#)). According to some studies, approximately 10% of whiplash injury survivors have vestibular complaints that could be explained either as subjective, individual sensations or as objective damage to the vestibular structures. It is necessary to conduct specialized vestibulometric tests to exclude the possibility of exaggeration of symptoms, which has been observed in this patient group ([Tranter and Graham, 2009](#)).

Other authors found that about 50% of whiplash injury patients show objective abnormalities on vestibular testing, such as reduced caloric responses, positional nystagmus and, occasionally, increased “hyperactive” vestibular responses ([Fischer et al., 1995](#); [Healy, 1982](#); [Kelders et al., 2005](#); [Oosterveld et al., 1991](#)).

In whiplash injuries, dizziness and vertigo are the most probable symptoms (changes in vestibular function); other pathogenetic explanations are based on neuromuscular ([Gray, 1956](#)) and neurovascular mechanisms ([Weeks and Travelli, 1955](#)), overexcitation, or damage of the cervical and/or lumbar proprioceptors ([Hinoki, 1985](#); [Nacci et al., 2011](#)). Acceleration forces associated with traumatic



injury probably loosen the otoconia; this leads to unequal otolith masses on the two sides, thus causing a temporary disturbance of spatial orientation (Brandt, 1999). A comparative otoneurological study was conducted in two groups of dizzy whiplash patients: patients with dizziness after pure whiplash injury and those with minor injury associated with whiplash. This study showed that there were only a few cases in which whiplash caused central or peripheral vestibulopathy, which was more probable after minor head injury associated with whiplash. Impaired postural control in these patients was more often related to cervical proprioceptive disorders that could be mistaken for a vestibular symptom (Nacci et al., 2011).

There has recently been an increase in publications reporting blast exposure as the cause of head injury. Vestibular disorders are common clinical findings in people with blast-induced traumatic brain injury. The mechanisms underlying injury of the cortical and subcortical structures responsible for motion perception, spatial orientation, vertigo and postural control have not yet been fully clarified (Scherer and Schubert, 2009).

Similarly, surveys on vestibular disorders occurring after blast exposure head trauma have also become more common. It has been found that, for the most part, these disorders follow an atypical course and cannot be equated with those following conventional traumatic brain injury. Furthermore, a US military study showed that vestibular function in these subjects deteriorates significantly over time (Hoffer et al., 2010).

Other authors have drawn parallels between vestibular damage following blast injury and following traumatic brain injury. In a comparative study of the two groups of patients it was found that in both cases the otolith organs are the most vulnerable structures. Their injury results in loss of function of the horizontal semicircular canals and postural instability (Akin and Murnane, 2011).

Objective biomarkers of diagnostic and prognostic importance have recently been identified in post-traumatic disorders following blast injury. Awwad et al. (2015) reported a transient increase in cerebral glucose metabolism in certain brain structures associated with vestibular function. Positron emission tomography/computed tomography image fusion made it possible to visualize the presence of hypermetabolic states in vestibulomotor brain regions (motor cortex, caudate putamen, thalamus and vestibular nuclei).

Focused neuro-otological studies have also been conducted, by Scherer et al. (2011), in two groups of patients who experienced blast trauma: one group with and one without post-traumatic dizziness. The mean active yaw angular vestibulo-ocular reflex (aVOR) gain in the symptomatic group was lower than that established in the asymptomatic group, while the results from passive yaw impulses were similar in the two groups. For pitch head rotation, both active and passive aVOR gains were significantly lower in the symptomatic group compared with the asymptomatic group. In both groups there were patients found to have aVOR gains above 1.0 for active pitch up head rotations. This indicates that in the asymptomatic patients, too, there can be abnormality in vestibulometric testing although this is less marked than in the other group. The authors concluded that the finding of depressed active aVOR gain in symptomatic patients compared with non-dizzy patients implicates central processing of second-order vestibular afference and efference copy signals. This observation may explain the discrepancy between high rates of dizziness reported by blast-exposed subjects and the relative scarcity of evidence available to support a diagnosis of vestibulopathy. The findings may also suggest a differential destruction of type I hair cells or disruption of irregular afferent vestibular pathways, explaining the common pattern of normal vestibular function testing in patients with mild traumatic brain injury. The reduced pitch aVOR and significantly elevated symptom severity during exertional testing appear useful findings in blast-exposed subjects.



A recent study of vestibular function among veterans with blunt and blast trauma showed that vestibular disorders may be related to psycho-emotional causes (particularly noradrenergically modulated states) as well as to organic damage at supratentorial level ([Franke et al., 2012](#)). Vestibulosuppressants had a positive effect on the acute stage of blunt or penetrating inner ear trauma, but so far no specific treatment has been recommended ([Shepard, 2013](#)).

Non-vertiginous dizziness in the absence of true peripheral or central vestibular dysfunction following whiplash-type trauma may have a cervicospinal origin ([Kushner, 1998](#)). Cervical vertigo is commonly invoked as a cause of dizziness after head trauma, presumably due to damage to cervical muscle afferents that project to the vestibular nuclei ([Brandt and Bronstein, 2001](#); [Gdowski and McCrea, 2000](#)). This view is supported by the improvement of vestibular complaints after any intervention in the neck region, for example, injection of local anesthetic ([de Jong et al., 1977](#); [Dieterich et al., 1993](#); [Zee and Leigh, 2006](#)).

Other proposed mechanisms of cervicogenic dizziness include overstimulation of cervical sympathetic nerves ([Ellis et al., 2015](#); [Hinoki, 1985](#)), aberrant afferent input from positional proprioceptors in the cervical spine, and compromised vertebral artery blood flow ([Sandstrom, 1962](#)). Vibration of neck muscles in normal subjects can lead to illusions of motion ([Karnath et al., 1994](#)). The cervico-ocular reflex may be enhanced in patients with whiplash injury ([Kelders et al., 2005](#)).

Post-traumatic migraine can occur with an aura of dizziness which is not always immediately followed by a headache. In these cases it is difficult to make the differential diagnosis between migraine with aura and direct involvement of the vestibular structures.

Concomitant injuries (e.g., injuries to the visual system or injuries leading to significant restrictions of range of motion) can also cause complaints of dizziness and instability ([Shumway-Cook, 2000](#)).

All peripheral and most central vestibular syndromes gradually improve over time due to functional recovery or central compensation once a treatment regimen is begun. This may consist of exercises designed to readjust the vestibular responses and enhance central compensation, or sensory substitution of the vestibular deficit. Central compensation (rearrangement) would account for a gradual recovery within weeks, thus supporting the view that exercise is a very effective therapy ([Brandt, 1999](#)).

Secondary phobic postural vertigo following recovery of organic post-traumatic vestibular dysfunction should be suspected if dizziness lasts longer than 4–6 weeks without noticeable improvement but with normal otoneurological test results ([Brandt, 1996](#); [Huppert et al., 1994](#)).

Chronic post-traumatic dizziness or disequilibrium persisting for months or years without abnormal otoneurological or neuro-ophthalmological findings is most likely psychogenic, especially if accompanied by chronic headache (tension or cervicogenic) and depression ([Brandt, 1999](#)). However, even though it is psychogenic, it nevertheless has to have a microscopic structural or subcellular basis, which indicates that the genesis is complex in many traumatic patients.

There are some non-vestibular causes of dizziness occurring after traumatic brain injury. Orthostatic hypotension, as a cause for dizziness, may be related to physical deconditioning, medullary injury or be a side effect of medications such as some antihypertensives, analgesics or beta blockers. Other symptomatic drugs for post-concussion syndrome (e.g., benzodiazepines, diuretics and anti-convulsants) may also lead to sensations of dizziness ([Daroff and Carlson, 2005](#)). Dizziness may be related to pre-existing conditions such as neurological disease, labyrinthopathy, cardiac disease or diabetes. One frequent complication of traumatic brain injury, i.e. hyponatremia ([Agha et al., 2004](#); [Harrigan, 1996](#); [Zafonte and Mann, 1997](#)), can cause dizziness or orthostatic hypotension ([Armstrong, 2004](#); [Harrigan, 1996](#); [Taylor et al., 1995](#)). When investigating post-traumatic vertigo and dizziness it is useful to apply the specialized vestibular tests, for example, checking for spontaneous nystagmus with and without Frenzel's glasses, and for positional and positioning nystagmus, the head-shaking test, the



Halmagyi test (the head-thrust test), and the vestibular bithermal caloric test. It has been shown that posturographic examination with eyes open and closed is useful for dynamic monitoring of patients in the post-traumatic period. In addition, it has been proven that balance control is related to the vestibular-spinal reflex and cervical proprioception in whiplash patients (Nacci et al., 2011).

Audiological testing is also important for the evaluation of the dizzy patient after traumatic brain injury, especially for determining the degree and the site of the damage (Rubin, 1990). This includes not only subjective audiometry, but also objective audiometry, i.e., evoked potentials. It is also important to determine whether tinnitus is present.

## Concluding remarks

In conclusion, the present review provides an overview of the various mechanisms underlying the vestibular disorders that follow head trauma. They can be due to organic, structural or substructural (microscopic) damage localized at different vestibular levels, from the labyrinth to the cortical regions; they can also involve non-vestibular structures, such as neck proprioceptors, whose signals integrate with the vestibular afferentation, as well as humoral mechanisms. They can also be psychogenic, functional disorders. Thus, vestibular examination and other related tests are very useful for establishing the site, pathogenesis and severity of traumatic vestibular disorder. Such information will improve the results of therapy.

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