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Recommended Citation

Fife, Terry D. and Giza, Christopher, "Posttraumatic Vertigo and Dizziness" (2013). *Neurology*. 69.
<https://scholar.barrowneuro.org/neurology/69>

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Posttraumatic Vertigo and Dizziness

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Semin Neurol 2013;33:238–243.

Abstract

Keywords

- ▶ postconcussive syndrome
- ▶ postconcussive dizziness
- ▶ vertigo
- ▶ traumatic brain injury

Dizziness and vertigo are common symptoms following minor head trauma. Although these symptoms resolve within a few weeks in many patients, in some the symptoms may last much longer and impede ability to return to work and full functioning. Causes of persisting or recurrent dizziness may include benign paroxysmal positional vertigo, so-called labyrinthine concussion, unilateral vestibular nerve injury or damage to the utricle or saccule, perilymphatic fistula, or less commonly traumatic endolymphatic hydrops. Some dizziness after head trauma is due to nonlabyrinthine causes that may be related to structural or microstructural central nervous system injury or to more complicated interactions between migraine, generalized anxiety, and issues related to patients self-perception, predisposing psychological states, and environmental and stress-related factors. In this article, the authors review both the inner ear causes of dizziness after concussion and also the current understanding of chronic postconcussive dizziness when no peripheral vestibular cause can be identified.

Vertigo and dizziness are common symptoms following head trauma.¹ Trauma can affect the labyrinth and other vestibular structures by a variety of mechanical mechanisms resulting in labyrinthine concussion, benign paroxysmal positional vertigo, perilymphatic fistula, unilateral vestibular loss, traumatic endolymphatic hydrops, and utriculosaccular injury.

Concussion and other head injuries can also lead to chronic dizziness without evident labyrinthine injury or dysfunction. Patients with this type of dizziness may remain so for weeks, months, or years. Possible mechanisms include central post-traumatic vertigo or dizziness that includes postconcussive syndrome, posttraumatic migrainous vertigo, diffuse axonal injury, and anxiety-associated dizziness.

In this article, we review the structural and anatomic consequences of trauma on the labyrinth and related vestibular structures and discuss the possible cause and management of postconcussive dizziness that often lacks demonstrable anatomical findings by examination, routine vestibular tests, or magnetic resonance imaging (MRI).

Labyrinthine Traumatic Injury

Benign Paroxysmal Positional Vertigo

Benign paroxysmal positional vertigo (BPPV) is among the most common causes of vertigo resulting from head trauma.^{2,3} The forces applied to the skull during trauma cause traumatic dislodgement of otoconia from the macula of the utricle. Like idiopathic BPPV, episodes of vertigo lasting 10 to 30 seconds are evoked by turning in bed or vertical head movements.⁴ Benign paroxysmal positional vertigo should always be considered in patients with head trauma with complaints of positional vertigo. Paroxysmal positional nystagmus is seen with Dix-Hallpike positioning and treatment with canalith-repositioning maneuvers or Semont's liberatory maneuver is highly effective.⁵ Recurrence rates are similar to idiopathic BPPV, but it may take more positioning maneuvers to achieve success.^{6–8} In addition, traumatic BPPV is more likely to be bilateral, occurring in 25% compared with only 2% in idiopathic BPPV.⁸

Labyrinthine Concussion

Labyrinthine concussion is a term that presumes a non-specified injury to the membranous labyrinth resulting from acceleration–deceleration forces on the bony labyrinth from trauma. Hearing loss, dizziness, and tinnitus are the typical features and often these improve. Thus, this term is given to define a syndrome and presumed cause rather than a specific well-defined injury. The injury may include ruptures of portions of the membranous labyrinth or bleeding or traumatic ischemia.^{9,10} Labyrinthine concussion may also occur on the side opposite a temporal bone fracture.¹¹

Posttraumatic Ménière's Disease

Ménière's disease is thought to be caused by dysfunctional regulation endolymphatic fluid homeostasis that leads to periodic endolymphatic hydrops. This influx of fluid to the endolymph space causes attacks of vertigo, muffled hearing, and ear pressure with tinnitus all in the affected ear. Attacks may last as little as 20 minutes, but usually last 2 to 4 hours. Diagnostic criteria have been developed for the diagnosis.¹² Low-frequency pure-tone hearing loss is a characteristic feature; over time, hearing loss becomes permanent as does vestibular loss. The relationship between endolymphatic hydrops and trauma can be difficult to determine because the condition may come on *de novo* without trauma. In one series of 120 patients with Ménière's disease, less than 3% were found to have the condition due to trauma.¹³ Nevertheless, there seems to be support for the idea of microstructural changes in the labyrinthine membranes, which render them prone to producing endolymphatic hydrops.¹⁴ There does not seem to be good evidence supporting noise exposure or other acoustic trauma as a cause, however.¹⁵ Initial treatment consists of sodium restriction to < 1500 mg per day and the addition of a diuretic, but if this fails to control vertigo attacks, additional interventions are available and discussed elsewhere.¹⁶

Utriculosaccular Injuries

Until recently, patients with posttraumatic dizziness and normal testing of horizontal vestibular function by caloric or rotational chair testing were considered to have no vestibular dysfunction. However, recent studies have found that this assumption is sometimes erroneous. Isolated dysfunction of the otolith organs (utricle and saccule) may occur even when caloric responses are normal¹⁷; this can be measured by vestibular evoked myogenic potentials (VEMPs).¹⁸ Patients with acute loss of the otolith sensory organ function may acutely have severe postural imbalance or a sense of tilting. Otolith sensory dysfunction may also affect quick head position changes and may account for some cases of positional dizziness on Dix-Hallpike positioning with no visible nystagmus. The time necessary to achieve functional adaptation to unilateral utricular or saccular loss is unclear. The asymmetric standing posture may recover much sooner as a result of central nervous system plasticity,¹⁹ but postural regulation during locomotion and dynamic movements may take longer, perhaps months, to recover.

Perilymphatic Fistulas

Head trauma can sometimes result in a rupture or other abnormal opening of the fluid filled membranous labyrinth.²⁰ These membrane ruptures usually occur at the round or oval window due to increased membrane elasticity between the middle ear and inner ear. Barotrauma as from scuba diving, blasts, large pressure fluctuations, or head trauma, particularly when associated with temporal bone fracture, can lead to a traumatic fistula.²¹ The findings may include unilateral deafness or sensorineural hearing loss, fluid or blood from the ear, a perforated tympanic membrane, tinnitus, vertigo, or unsteadiness. The diagnosis is notoriously difficult as evidenced by the small number of documented fistulas seen at the time of surgery even among those in whom it has been suspected.²⁰ Some fistulas heal spontaneously, but surgical repair is needed in cases that do not improve with time.

Unilateral Vestibular Loss

Vestibular loss may occur after head trauma even in the absence of temporal bone fracture or hearing loss.²² Possible mechanisms include traction or injury-induced demyelination of the vestibulocochlear nerve, trauma-related bleeding or micro-ischemic changes, or direct trauma or injury to the labyrinth. Temporal bone fractures may result in vertigo from unilateral vestibular loss. Of 118 consecutive skull fractures, 22% were temporal bone fractures²³ with ~80% being longitudinal in the axis of the petrous bone and 20% being transverse. Longitudinal fractures usually involve the middle ear and are less likely to transect the vestibulocochlear and facial nerves; they are caused by blunt force to the temporal or parietal regions. Transverse fractures often involve the otic capsule and inner ear and vestibulocochlear nerve and injure the facial nerve; they are due to blunt force to the frontal-occipital axis. Other commonly observed findings associated with temporal bone fractures include otorrhea or hemotympanum, tympanic membrane perforation, cerebrospinal fluid otorrhea and vertigo, and hearing loss and facial paralysis.

Dizziness from Central Nervous System Traumatic Injury

Imbalance is common in the late aftermath of serious traumatic brain injury and may also be present in mild traumatic brain injury (mTBI).²⁴ Trauma to the pontomedullary region of the brain that is bilateral and visible by MRI tends to have very poor outcomes,²⁵ so this is not likely to account for the larger number of patients with chronic dizziness after TBI. Traumatic brain injury can lead to diffuse axonal injury, and traction on or contusion of the brainstem^{25,26} or cerebellum,^{27,28} any of which can disrupt vestibular and postural reflex pathways accounting for dizziness. Diffuse axonal injury refers to injury to axons within the white matter fiber tracts due to abrupt stretching that damages the axonal cytoskeleton, reduces elasticity, and impairs axoplasmic transport.²⁹ Diffuse axonal injury most commonly affects the gray–white matter junctions, the brainstem, corpus

callosum, cerebral and cerebellar peduncles, basal ganglia and thalamus, and the frontal and temporal lobe white matter.

Mild traumatic brain injury is most commonly defined as a closed head injury with¹ a Glasgow Coma Scale score of 13–15,² resolution of posttraumatic amnesia within 24 hours,³ and loss of consciousness for less than 30 minutes. A head computed tomography (CT) scan will show no hemorrhage or other acute traumatic abnormalities.^{4,30} Accordingly, the vast majority of patients with head trauma or concussion have mTBI.³¹ The challenge in most patients with chronic dizziness and general symptoms of postconcussive syndrome after mTBI is how to distinguish patients with microstructural brain injury from those without it. The assumption is that this distinction may inform better treatment and prognostication.

Posttraumatic Vestibular Migraine

Posttraumatic migraine-like headaches, and to a lesser degree dizziness, are common after minor trauma to the head and neck.^{32–34} In a study of 58 active duty and retired military personnel with mild TBI and resulting dizziness, 41% were diagnosed with posttraumatic vestibular migraine.³

Patients with posttraumatic migrainous vertigo often describe dizziness as spinning, rocking or to-and-fro oscillation sensations, floating, or drunkenness that in many ways makes it indistinguishable from vestibular migraine without prior trauma and also with postconcussive dizziness in general. In these patients, the presence of nausea and motion sensitivity and absence of any evident primary inner-ear cause somewhat favor posttraumatic migrainous vertigo. The symptoms may begin within days to several weeks of the concussion, but as with postconcussive syndrome, the severity of the head injury seems a poor predictor of whether a patient will develop migrainous vertigo. The poor correlation between the trauma severity and migraine has suggested that posttraumatic stress disorder, from the emotional trauma of the event, is a more important predictor of the development of migraine symptoms than is any actual physical brain trauma.³⁵ Others have speculated similarly that the cause may not be anatomical injury, but the same mechanisms at play for migraine syndrome in the absence of head injury.^{36,37} A better predictor is pre-existing personal or family history of migraine and a long history of motion sickness. Women are more likely afflicted by 2:1 to 3:1 over men.³⁸

Although there are randomized clinical trials addressing the treatment of migraine headaches, there are none addressing vestibular migraine *de novo* nor posttraumatic migrainous vertigo.^{39,40} Extrapolating from studies in the treatment of migraine headaches,^{41,42} it seems quite reasonable to try to manage chronic debilitating posttraumatic migrainous dizziness with migraine prophylactic medications.³² One retrospective chart review was unable to demonstrate a correlation between the use of several migraine prophylactic medications and improvement in headaches and vestibular measures.³⁴

Postconcussion Syndrome versus mTBI

Postconcussion syndrome includes persistence of at least three of the following symptoms after a concussion: head-

ache, dizziness, irritability, reduced concentration, fatigue, insomnia, and photophobia and phonophobia. The term *postconcussion dizziness* implies it is a component of postconcussion syndrome, an entity with a long history.⁴³ However, in a patient with a history of minor head trauma, it may be unclear whether the individual has dizziness due to microstructural abnormalities from trauma due to the postconcussive syndrome—without any actual injury to the brain—or whether there is actually an unrecognized labyrinthine cause. Indeed, the possibilities are not mutually exclusive; some patients with postconcussive dizziness may also have a labyrinthine cause, thus posing a particular challenge to clinicians. The diagnosis of posttraumatic dizziness is sometimes used to lump all entities together by simply indicating that the patient reports dizziness after a minor head injury.

Patients with mTBI often report postconcussive syndrome symptoms such as headache, memory difficulties, dizziness, and irritability; however, these same symptoms are very common in general and were as common in those with orthopedic injuries as in those with mTBI in a cohort of university students.⁴⁴ However, in a retrospective cohort study of military personnel with mild TBI versus nonhead injury, those with mild TBI ($n = 334$) were more likely than the nonhead injury ($n = 658$) group to report several symptoms, including tinnitus (odds ratio [OR] = 1.63, 95% confidence interval [CI] = 1.10–2.41) and dizziness (OR = 10.60, 95% CI = 3.48–32.27).⁴⁵ In other studies, the link between mTBI and persisting postconcussive syndrome symptoms lasting more than a year is tenuous. An online questionnaire study found postconcussive syndrome symptoms present among controls without mTBI at essentially the same rate as in those without mTBI.⁴⁶ Furthermore, the severity of the concussion or duration of loss of consciousness correlates very poorly with persistent postconcussive syndrome symptoms.⁴⁷ Interestingly, one study of 176 mTBI patients found that postconcussive syndrome was actually more prevalent in those without abnormalities on cerebral imaging, whereas those with imaging abnormalities were more likely to have auditory or vestibular abnormalities.⁴⁸

Even so, some studies have suggested some connection between postconcussive syndrome symptoms and microstructural brain changes in mTBI patients. Using an MRI technique to measure impaired structural integrity due to diffuse axonal injury, patients with mild traumatic brain injury with postconcussive syndrome showed more severe and persistent structural integrity changes than did those mTBI patients without postconcussive syndrome symptoms.⁴⁹ Furthermore, the severity of postconcussive syndrome symptoms after mTBI also correlates with the degree of microstructural brain injury using similar tract-based spatial statistics on MR diffusion tensor imaging (DTI).⁵⁰

Postconcussion Dizziness

Postconcussion dizziness is most commonly described as a rocking or swaying sensation, floating lightheadedness, a feeling of drunkenness, or general imbalance worsened by head motion. As such, the description is compatible with a

mixed bag of causes that could potentially include occasional patients with microstructural changes of the brain, but more commonly includes those with posttraumatic migrainous vertigo, labyrinthine dizziness, so-called cervical vertigo, anxiety-related dizziness, and some with combinations of these that is further aggravated by the poor coping ability. In many patients, the dizziness is either continuous or occurs on and off. There seems to be poor correlation between the severity of reported symptoms and the severity of head trauma, an observation true for postconcussive syndrome in general.⁴⁷

Sports-Related Postconcussion Dizziness

There is some value to considering sports-related concussion and postconcussion dizziness distinctly. First of all, the consequences of vestibular dysfunction and imbalance may play a role in an increased risk for repeated injury in the athlete returning to sport prematurely. Second, there are several studies investigating postconcussion dizziness as a symptom specifically in cohorts of concussed athletes, which may or may not be generalizable to other mechanisms of concussion or mTBI (including mTBI caused by a military blast injury).

Dizziness and imbalance are fairly common complaints after a sports-related concussion, reported in 43 to 81% of concussed athletes.^{51,52} When self-reported symptoms of dizziness and imbalance were compared with objective measurements of postural stability and vestibular function from the sensory organization test (SOT) in a concussed athlete cohort, moderate correlations (Spearman $R_s = -0.39$ to -0.57) were reported,⁵³ suggesting that symptoms are related to actual impairments, but also that other factors may weigh in with regard to symptom reporting in athletes. One cohort study of 107 high school athletes with concussion found that on-field dizziness was a significant predictor (OR = 6.34, 95% CI = 1.34–29.91) of prolonged (> 21 d) recovery after a sports-related concussion.^{52,54}

A small number of reports have also implicated autonomic dysfunction as a potential contributor to postconcussive dizziness and other symptoms.^{55–57} Recently, Goodman and colleagues reported autonomic dysfunction detected by tilt table testing in 20 concussed patients with symptoms of dizziness and lightheadedness.⁵⁸

Prognosis

Patients with chronic vertigo after concussion are substantially less likely to return to work.⁵⁹ Most patients improve within days to a few weeks, but a sizable proportion of patients continue to report postconcussive syndrome symptoms for months or years after minor head injuries. Patients with postconcussive syndrome symptoms at 3 months ended up having similar symptoms at 12 months.⁶⁰ In one Swedish cohort, 56% were recovered at 3 months postinjury.⁶¹

Psychological factors may worsen the impact of vestibular symptoms and delay functional recovery. Cultural issues may influence the endorsement of postconcussive syndrome symptoms in healthy individuals of differing cultural and language backgrounds.⁶² There is speculation that chronic dizziness and other symptoms of mTBI are a phenomenon of

Western Europe and the United States; However, similar persisting symptoms and lack of return to work even at 1 year has been reported in other regions as well.⁶³ One study of individuals evaluated for legal claims from whiplash injury reported a very low proportion (3.7%) having any vestibular symptoms or complaints.⁶⁴

It may be that the actual physical trauma of a mTBI is a less important factor than is female gender and the presence of preinjury depression and anxiety, posttraumatic stress disorder, and pain, which predispose to prolonged postconcussive syndrome symptoms.^{65–67} As one hypothesis goes, mild head trauma may be the initiating factor, but it is the pre-existing psychological condition that causes the prolonged postconcussive syndrome symptoms.⁶⁸

Treatment

Effective treatment requires elimination of as many of the factors that contribute to dizziness, headaches, concentration difficulties; minimization of stress whenever feasible is also recommended. Some have suggested that rest from cognitive and physical activities may improve recovery after a sports-related concussion,^{47,69} implying that time off work or away from stress may help. Practically speaking, the opposite is more common. Many patients must take off from work, which adds to work-related, household, and financial stressors. It has been suggested that managing anxiety may help to reduce the ongoing symptoms of postconcussive syndrome.⁷⁰ Even patients' perception of their own well-being and so-called all-or-nothing behavior appear to influence the perpetuation of postconcussive syndrome.⁷¹

In a patient with chronic dizziness after a concussion, one should evaluate for migrainous vertigo, labyrinthine causes, autonomic dysregulation, and medication side effects that might be adding to the patient's symptoms. It should not be assumed that signs of generalized anxiety are necessarily the cause of the dizziness because secondary anxiety is especially common in those with chronic dizziness of any cause.⁷² If the patient reports spinning vertigo, a labyrinthine cause should be sought; caloric testing, audiometry, and VEMPs, if available, should be done. If no labyrinthine cause is identified, one can consider prophylactic treatment for posttraumatic migraine. Anxiety should also be managed with counseling and/or medications because chronic vestibular symptoms may provoke secondary anxiety.^{73,74} It is also important to encourage patients to resume their normal activities and to shape their self-perceptions in a positive light to minimize the detrimental effect of being ill or infirm.

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