

UNDERSTANDING THE ORIGINS OF DIZZINESS IN PEOPLE WITH MILD TRAUMATIC BRAIN INJURY

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Each year, about 2.5 million people sustain a traumatic brain injury², and the majority of those injuries are mild traumatic brain injury (mTBI)¹. People with mTBI commonly report numerous symptoms ranging from cognitive, ocular-motor, migraine, anxiety/mood, and sleep¹. Currently, treatment for mTBI is based on specific symptoms the individual reports. The main issue with current treatment strategies is that symptoms overlap across many treatment options. This overlap introduces uncertainty in subtype classifications that drive treatment decisions. As result, people may receive incorrect, or delayed treatments.

The most overlapped symptom of mTBI is the symptom of dizziness. Dizziness is a common unmanageable symptom that has been an ongoing problem in individuals with mTBI. While often associated with vestibular and balance-oriented problems, dizziness can stem from multiple sources, including physiological and vestibular-ocular dysfunction after mTBI. The origin of dizziness remains unclear in the mTBI population.

To further assess the origin of dizziness, this study analyzed individual and combinational associations of physiological measures including blood pressure (BP), and heart rate (HR), balance measurements often associated with vestibular dysfunction, including turning velocity, gait speed, and postural sway, and vestibular-ocular measurements with reported levels of dizziness on the Dizziness Handicap Inventory (DHI).

Twenty-two participants, ages 22-50 diagnosed with mTBI, were tested 25-484 days post mTBI. Participants completed a tilt table test to assess changes in BP and HR from a supine to head up tilt (HUT) position to capture autonomic measures. A one-minute walk, and quiet standing (30 seconds, eyes closed) task were completed using wearable sensors (APDM Opals, Portland, OR) to compute balance and mobility measures. Vestibular-Ocular Motor Screening (VOMS) was completed to measure self-reported symptomatic changes before and after vestibular-ocular tasks, scored out of 7 task categories. Finally, participants completed the DHI, a 25-item self-assessment to evaluate the self-perceived handicapping effects imposed by dizziness.

Pearson correlation coefficients assessed the relationship between individual measurements and DHI total scores. Additionally, a backwards-stepwise regression model was used to determine the combination of variables that associated with the DHI score. Results indicated small-to-moderate correlations between some individual measurements and DHI (R^2 ranged from 0.02 to 0.36). Turning velocity, mean BP during the HUT, and total VOMS scores were retained in the final regression model, which had an R^2=0.68. These results support the

idea that dizziness can have varied etiologies in people with mTBI, including physical movement, autonomic dysfunction, and vestibular-ocular dysfunction.

Current mTBI treatments commonly focus on vestibular rehabilitation, as most dizziness-associated research is completed in this domain of mTBI. However, individuals who do not display vestibular deficits, and continue to experience dizziness, often do not receive adequate treatments to combat their symptoms¹. Our results suggest offering a battery to test multiple functions associated with dizziness including dynamic balance, autonomic, and vestibular-ocular functions to further understand the source(s) of an individual's dizziness to prescribe tailored, effective treatments.

References

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