

Higher Dependency on Visual Inputs During Posture Control: Another Long-Term Effect of mTBIs

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Abstract

This study investigated the hypothesis that individuals living with long-term effects of mild traumatic brain injury (mTBI) develop increased dependency on their visual inputs to control their vertical posture. To test this hypothesis we quantified visuo-postural dependency indices (*VPDIs*) calculated independently for multiple postural behavioral markers extracted from the body's center of pressure coordinates signals recorded during the execution of a quiet bipedal stance. One hundred and twenty-nine volunteers participated in this study. An *mTBI* ($n = 50$) and a neurotypical *Control* group were formed ($n = 79$). *VPDIs* were calculated as the normalized pair-wise subtraction of recordings obtained under *Vision* and *No-Vision* experimental conditions. Consistent with our hypothesis the results of this study show that balance behavior of mTBI participants deteriorate more abruptly in the absence of visual inputs when compared to neurotypical controls. These impairments may increase the likelihood of recurrent traumas when fast reactions are needed in daily activities, sports practice, or military operations. Additionally, the methodology used in this study showed to be potentially useful to aid future investigations of neural circuitry impaired by mTBI and provide indices of recovery in future clinical trials testing mTBI-related clinical interventions.

Introduction

Impairment to body vertical control is a common finding in patients suffering from the long-term effects of mild traumatic brain injuries (mTBIs)¹⁻³. Its effects are captured several months after an injury (> 6 months) and they indicate deterioration to one's ability to control its vertical posture. Significant increases in indices of body sway amplitudes accompanied by increases on body sway velocities corroborates with this view⁴. Despite the fact these postural behavior abnormalities are subtle and may go unnoticed for years, they are a key factor for the development of re-occurring injuries during young adult age as well as higher risks of falling on later stages of life. Multiple efforts are currently underway to clarify the neural mechanisms behind these behavioral modifications but current evidence suggests they stem from suboptimal multi-muscle control by the central nervous system (CNS)²⁻⁴.

This rationale finds support on the notion that multi-muscle control is a complex neural process depending on the optimal functioning of its conduction system⁵⁻⁷. This view is supported by the large number of neural substrates involved in the process of integrating sensory inputs from the visual, somatosensory, and vestibular organs into coherent motor outputs sent to the axial skeletal muscles. As a result, sensory neural potentials must travel relative long distances to be integrated. To cope with such complexity and allow for a fast integration of sensory information into motor commands the CNS counts with a fast and reliable conduction system. In fact, severe reduction in velocity conduction will make impossible for someone to respond to sudden environmental changes (e.g., changing direction to avoid collision with a tree while skiing) increasing the risks of severe injuries.

Conduction functions have shown to be compromised by mTBIs. For example, a recent study performed on patients suffering from concussive syndrome showed significant increases in the response latencies

invoked by both simple visual and auditory stimuli forty-eight months post-injury⁸. These findings are considered of importance since they suggest a wide-spread reduction in conduction of information across CNS substrates. This point of view has gained support from multiple observations showing significant reduction in the volume of important tracts associated with intra- and inter-hemispherical neural transmission⁹⁻¹². For example, Hulkover et al (2013)¹¹ compiled results from a large cohort of studies that utilized diffusion tensor imaging and suggested the existence of white-matter abnormalities in important areas for the integration of neural information within and between distinct brain areas including the superior longitudinal fascicule (SLF). Interestingly, the SLF is an important intra-hemisphere association pathway establishing connections between postrolandic regions and the frontal lobe¹³⁻¹⁵ by conveying important information about the perception of the visual space (SFL subdivision II, SFL-II). Therefore, the construction of a reliable neural representation of body configuration needed for optimal vertical posture control is dependent on the functional integrity of these tracts¹⁶⁻²⁰.

An important characteristic of the abnormal postural behavior observed in mTBI patients regards to their subtle progression during the initial years after an mTBI episode. During this period, despite the absence of reported symptoms a series of underlying modifications to the mechanisms of posture control may be under way to cope with the delays found in the neural processing of sensory-motor integration. Currently, the extent of the sensory functional re-weighting among major sensory systems after an mTBI is unclear however, we hypothesized that under such constraints a reorganization on the level of reliance on sensory modalities will occur. This hypothesis is based on previous reports supporting the notion the CNS is able to re-weight the role of each sensory modality on generating motor commands²¹⁻²⁵. Direct evidence for a re-weighting process comes from previous studies using manipulations of characteristics of sensory stimuli and their resulting effect to postural behavior indices (e.g. body sway amplitude and frequency)²⁶⁻²⁸. Collectively, these studies suggest that a healthy central nervous system can reduce its dependency on a less reliable sensory modality while increasing its dependency on the remaining channels. However, mTBI patients suffer from delays in information transmission of multiple sensory modalities therefore increasing the complexity of such adaptations. In this study we tested the hypothesis that individuals living with long-term mTBI effects develop a larger dependency on their visual inputs to control their vertical posture. This expectancy is based on the rationale that under the scenario of multiple delays in information processing, the CNS will increase its reliance on the visual system due to its capacity to produce a more complete description of its environment.

Materials & Methods

Participants.

One hundred and twenty-nine volunteers participated in this study (\bar{x} = 25.3 years of age; s = 7.2 years). All provided their informed and written consent based on procedures approved by the local Institutional Review Boards (University of Montana and Western Michigan University) and conforming to The Declaration of Helsinki. Informed consent was also provided to publish any images reported here.

An *mTBI* group was formed by fifty individuals (twenty-one females; twenty-nine males; \bar{x} = 28.8 years of age; s = 9.4 years) with history of single or multiple mild traumatic brain events. This cohort included occurrences from sports related activities, motor vehicle accidents, and military operations. The average number of previous concussive events was 2.15 (s = 2 events) and the average time elapsed from the last traumatic episode was 89.9 months (s = 90 months). Inclusion criteria for this group followed the standard classification proposed by the American Psychiatric Association as published in the Diagnostic and Statistical Manual of Mental Disorder²⁹ and included (a) mTBI caused by either direct impacts or blast-induced closed head trauma; and (b) presence of one or more of the following manifestations after the mTBI episode: confusion or disorientation, loss of consciousness for thirty minutes or less, post-traumatic amnesia for less than twenty-four hours, Glasgow Coma Scale score of 13–15 after thirty minutes post-injury or later on presentation for health care, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery. Exclusion criteria included the following: (a) manifestations of mTBI caused by drugs, alcohol, medications, and other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries, or intubation); (b) other potentially confounding issues (e.g. psychological trauma, language barrier, or coexisting medical conditions); (c) history of penetrating cranial injury; (d) history of any type of neurosurgery; and (e) abnormalities of cranial nerve functions.

A control group (*Control*) was formed by seventy-nine healthy individuals (fifty-seven females; twenty-two males; \bar{x} = 23.4 years of age; s = 4.5 years). The exclusion criteria for the control group included the following: (a) no previous history of head trauma, traumatic brain injury (including cerebral vascular accident), epilepsy, or seizure disorders; (b) no history of substance abuse (drugs, alcohol, or controlled medication); (c) no symptoms of psychological trauma such as panic attacks, frequent nightmares, insomnia, loss of self-esteem, anxiety, anger, frequent depression, feeling of despair, or emotional detachment; (d) no previous history of peripheral neuropathy (including loss/decreased sensory or motor function in the upper extremity), acute upper extremity injury, recurring and/or unexplained headaches, cardiac pacemaker, pulmonary disease, or metallic implants in head, spine, or upper extremity; (e) no history of any type of neurosurgery; and (f) no abnormalities of cranial nerve functions.

Materials and Procedures.

A force platform (AMTI BP400600, AMTI Inc.) was used to acquire the vertical and horizontal components of the ground reaction force (GRF) as well as moments of force around the frontal and sagittal axes. These signals were used for computations of the body's center of pressure coordinates in anterior-posterior and medial-lateral directions (COPap and COPml, respectively). Consistent with previous studies COPap and COPml were defined by:

$$COP_{ap} = \frac{-hF_x - M_y}{F_z} \quad (1)$$

$$COP_{ml} = \frac{-hF_y - M_x}{F_z} \quad (2)$$

where h is the height of the base of support above the force plate, F_x is anterior-posterior component of the GRF, F_y is medial-lateral component of the GRF, F_z is vertical component of the GRF, M_x is the moment of force around the sagittal axis and M_y is the moment of force around the frontal axis. All signals were sampled at 50 Hz with a 12-bit resolution. Prior to data recording, each participant went through a general neurological screening then provided self-reports utilizing Neurobehavioral Symptom Inventory (NSI) to record their prevailing current symptoms. Participants who met the established inclusion and exclusion criteria were tested in an area free of noise and distractions.

Protocol and Variables of Interest.

All participants were asked to perform the simple task of standing still for one-hundred and twenty seconds on a force platform with their either eyes opened or closed (*Vision* and *No-Vision* conditions, respectively). Feet were placed in parallel and fifteen centimeters apart and consistency of foot placement across participants was achieved by markings drawn on the surface of the force plate (Fig. 1a). Since participants did not move their feet during the entire experimental session this position was consistent along the entire experiment. Participants were barefoot during the entire experiment and instructed to cross their upper limbs against the chest. During execution of the *Vision* condition participants focused their sight to a physical static point placed at eyes level and at approximately one meter. Once this initial position was taken, participants were asked to remain steady, as vertical as possible, and to distribute their body weight evenly between the two feet. To avoid recording of any transient effects during the initial moments of a trial, data recording started after five seconds from the moment the subject took position. During execution of the *No-Vision* condition participants these same procedures were followed however participants kept their eyes fully closed during the entire time of data

recording. The average duration of the entire experimental session was approximately forty minutes and none of the participants reported fatigue or dizziness as an issue.

Signals from the force plate (Fig. 1b) were analyzed off-line with customized routines (BalanceLab vs 3.0, Synergy Applied Medical and Research Inc, USA). COPap and COPml coordinate signals were down sampled to 10 Hz and detrended by the mean of each time series. The following postural indices were computed for both *Vision* and *No-Vision* conditions: area of COP displacement (*COP_area*), total length of COP displacement (*COP_total_length*), maximum amplitude of COP displacement in each direction (*COPap_range* and *COPml_range*), root mean square estimations of COP signals (*COPap_rms* and *COPml_rms*), mean velocities of COP displacement (*COP_total_mean_vel*, *COPap_mean_vel*, and *COPml_mean_vel*), mean jerkiness of COP displacement (*COP_total_mean_jerk*, *COPap_mean_jerk*, and *COPml_mean_jerk*), frequency containing 80% of signal spectral power (*COPap_F80* and *COPml_F80*), amount of regularity and predictability of COP displacement in time quantified by sample entropy of COP signals (*COP_sent_ap* and *COP_sent_ml*), and degree of asynchrony or dissimilarity between COPap and COPml signals in time quantified by cross sample entropy (*COP_crosssent*).

COP_area was defined similarly to procedures employing the sector formula of Leibniz previously described and used in the literature^{30,31}. *COP_total_length* was computed as the total length of the COP displacement during the whole stance trial. The maximum amplitudes of the COP displacement in each direction (*COPap_range* and *COPml_range*) were computed by the difference between their maximum and minimum coordinates recorded. *COP_total_mean_vel*, *COPap_mean_vel*, and *COPml_mean_vel* were computed as the length of the COP trajectory divided by the duration of the trial. *COP_total_mean_jerk*, *COPap_mean_jerk*, and *COPml_mean_jerk* represented the rate of change of COP acceleration. They were computed as the third derivative of the COP position with respect to time. *COPap_sent*, *COPml_sent*, and *COP_crosssent* were computed through estimations of the correlation, persistence, and regularity of the COP signal in time. Smaller sample entropy estimates indicate many repetitive patterns of COP fluctuation in time, whereas larger estimates indicate a more irregular, random, and unpredictable pattern. For cross-sample entropy, higher estimates indicate larger levels of asynchrony of postural sway between the two directions, whereas lower estimates indicate more co-dependence^{1,32,33}.

A visuo-postural dependency index (*VPDI*) was then calculated for each postural index by a normalized pair-wise subtraction of recordings obtained under *Vision* and *No-Vision* conditions, as follows:

$$VPDI (\%) = ((NoVision - Vision) / Vision) * 100 \quad (3)$$

Under this approach, a *VPDI* represents the normalized effect of vision on a postural index of interest and its value can be either zero, positive, or negative. *VPDI* zero values are interpreted as a null effect of vision to postural sway behavior. Positive departures from a null effect represent a positive impact of vision to control of the vertical posture.

Statistical Analyses.

Due to the disparity in the number of males and female participants in both groups, two one-way MANOVA with factor *Gender* (Female and Male) and *VPDs* as variables of interest were used to define potential effects of *Gender* to these variables. Since *Gender* had no significant effect to the modulation of *VPDs* (Control: $F_{17,60}=1.291$, *Wilk's Lambda* = 0.735, $p = 0.229$; Control: $F_{17,34}=0.737$, *Wilk's Lambda* = 0.705, $p = 0.743$), data recorded from females and males on each group were combined into single data sets. Independent two-tailed t-tests were utilized to identify the effects of mTBI to symptoms scored by the NSI. Wilcoxon's and Mann-Whitney's tests were used to identify the potential effects of visual disruption to all postural indices and *VPDs* of interest on both groups. All tests were performed by the IBM SPSS statistics software suite (version 20, IBM® SPSS®) while keeping a level of significance at 5% ($\alpha = 0.05$).

Results

Recordings of current symptoms confirmed the presence of long-term effects on our *mTBI* cohort. Table 1 shows averages and standard deviations of NSI scores obtained across participants. They represent symptoms felt by the participants along the two weeks preceding the experimental session. Multiple symptoms affected our *mTBI* cohort including difficulties to fall or stay asleep, headaches, irritability, poor concentration, difficulty remembering information and making decisions, slowed thinking, poor coordination, changes in appetite, and hearing and vision difficulties. These findings were confirmed by a series of two-tailed student's T tests. A significant increase in the NSI's total score recorded for the *mTBI* participants was found ($p < 0.0001$).

Table 1

Averages and standard deviations recorded by the Neurobehavioral System Inventory (NSI) for participants in the *Control* and *mTBI* groups. Significant values from inferential tests are bolded.

	<i>Control</i>	<i>mTBI</i>	<i>T-test</i>
	<i>Mean ± SD</i>	<i>Mean ± SD</i>	<i>(p-value)</i>
<i>Feeling Dizzy</i>	0.15 ± 0.40	0.81 ± 1.02	< 0.0001
<i>Loss of balance</i>	0.12 ± 0.32	0.74 ± 0.99	0.0002
<i>Poor coordination, clumsy</i>	0.10 ± 0.30	0.83 ± 1.01	0.0003
<i>Headaches</i>	0.43 ± 0.62	1.36 ± 1.23	< 0.0001
<i>Nausea</i>	0.17 ± 0.59	0.33 ± 0.82	< 0.0001
<i>Vision problems, blurring, trouble seeing</i>	0.17 ± 0.42	1.07 ± 1.11	0.2609
<i>Sensitivity to light</i>	0.35 ± 0.94	0.69 ± 0.92	< 0.0001
<i>Hearing difficulty</i>	0.10 ± 0.30	1.00 ± 1.17	0.0717
<i>Sensitivity to noise</i>	0.13 ± 0.50	0.74 ± 1.19	< 0.0001
<i>Numbness or tingling on parts of my body</i>	0.17 ± 0.46	0.69 ± 1.118	0.0031
<i>Change in taste and/or smell</i>	0.03 ± 0.18	0.21 ± 0.65	0.0085
<i>Loss of appetite or increased appetite</i>	0.20 ± 0.61	0.45 ± 0.97	0.1392
<i>Poor concentration, can't pay attention, easily distracted</i>	0.28 ± 0.58	1.50 ± 1.27	< 0.0001
<i>Forgetfulness, can't remember things</i>	0.32 ± 0.62	1.48 ± 1.27	< 0.0001
<i>Difficulty making decisions</i>	0.23 ± 0.53	0.90 ± 1.14	0.0008
<i>Slowed thinking, difficulty getting organized, can't finish things</i>	0.20 ± 0.55	1.21 ± 1.30	< 0.0001

	<i>Control</i>	<i>mTBI</i>	<i>T-test</i>
	<i>Mean ± SD</i>	<i>Mean ± SD</i>	<i>(p-value)</i>
<i>Fatigue, loss of energy, getting tired easily</i>	0.47 ± 0.77	1.24 ± 1.34	0.0013
<i>Difficulty falling or staying asleep</i>	0.38 ± 0.58	1.55 ± 1.37	< 0.0001
<i>Feeling anxious or tense</i>	0.58 ± 0.70	1.19 ± 1.23	0.0055
<i>Feeling depressed or sad</i>	0.28 ± 0.49	0.64 ± 1.08	0.0484
<i>Irritability, easily annoyed</i>	0.30 ± 0.62	1.07 ± 1.11	0.0001
<i>Poor frustration tolerance, feeling easily overwhelmed by things</i>	0.40 ± 0.74	0.98 ± 1.16	0.0060
Total Score	5.58 ± 6.95	21.02 ± 18.18	< 0.0001

Figure 2 shows stabilograms recorded from representative participants from the *Control* (panels A and C) and *mTBI* (panels B and D) groups under the *Vision* and *No-Vision* conditions. Note that for both participants the transition between these two conditions had a significant impact to their sway magnitude. However, the impact of visual disruption to *mTBI* participants was disproportionate larger and impacted several other indices of postural sway. Such observations were held true for both cohorts and are presented on Tables 2 and 3.

Tables 2 and 3 show the medians and quartiles (25th and 75th) across participants for all seventeen postural indices of interest and VPDIs recorded under *Vision* and *No-Vision* conditions. Significant higher modulations for the *mTBI* group were found for *VPDI COP_area* ($p = 0.013$), *VPDI COPap_range* ($p = 0.005$), *VPDI COPml_rms* ($p = 0.012$), *VPDI COP_total_mean_vel* ($p < 0.001$), *VPDI COPap_mean_vel* ($p = 0.013$), *VPDI COPml_mean_vel* ($p = 0.002$), *VPDI COP_total_mean_jerk* ($p = 0.008$), and *VPDI COPml_mean_jerk_ml* ($p < 0.001$). These results were confirmed by the Mann-Whitney U tests ran with factor *Group* (*Control* vs *mTBI*) and each *VPDI*.

Table 2

Medians and quartiles (Q25th and Q75th) recorded for all postural indices under *Vision* and *No-Vision* conditions and for both *Control* and *mTBI* groups. Significant values from inferential tests are bolded.

	<i>Control</i>		<i>mTBI</i>			
	<i>Vision</i>	<i>No-vision</i>	<i>Vision</i> <i>x</i>	<i>Vision</i>	<i>No-vision</i>	<i>Vision</i> <i>x</i> <i>No-Vision</i>
	<i>Median</i> <i>(Q25th – Q75th)</i>	<i>Median</i> <i>(Q25th – Q75th)</i>	<i>p-value</i>	<i>Median</i> <i>(Q25th – Q75th)</i>	<i>Median</i> <i>(Q25th – Q75th)</i>	<i>p-value</i>
<i>COP_area</i>	1.01 (0.69, 1.80)	1.71 (1.05, 2.60)	< 0.001	1.40 (0.98, 1.89)	2.41 (1.65, 5.24)	< 0.001
<i>COP_total_length (cm)</i>	390 (326, 517)	480 (375, 596)	< 0.001	444 (371, 531)	542 (428, 875)	< 0.001
<i>COPap_range (cm)</i>	1.98 (1.64, 2.41)	2.48 (2.01, 3.20)	< 0.001	2.14 (1.64, 2.64)	2.85 (2.12, 4.83)	< 0.001
<i>COPml_range (cm)</i>	1.04 (0.81, 1.40)	1.34 (0.96, 1.77)	< 0.001	1.31 (0.96, 1.73)	1.97 (1.35, 2.65)	< 0.001
<i>COPap_rms (cm)</i>	0.35 (0.29, 0.44)	0.40 (0.32, 0.51)	< 0.001	0.36 (0.29, 0.49)	0.46 (0.34, 0.72)	< 0.001
<i>COPml_rms (cm)</i>	0.16 (0.13, 0.22)	0.21 (0.14, 0.26)	< 0.001	0.20 (0.15, 0.27)	0.28 (0.19, 0.37)	< 0.001
<i>COP_total_mean_vel (cm/s)</i>	0.60 (0.51, 0.70)	0.86 (0.72, 1.15)	< 0.001	0.62 (0.52, 0.85)	1.01 (0.79, 1.47)	< 0.001
<i>COPap_mean_vel_ap (cm/s)</i>	0.46 (0.40, 0.55)	0.70 (0.56, 0.92)	< 0.001	0.49 (0.39, 0.57)	0.81 (0.54, 1.15)	< 0.001
<i>COPap_mean_vel (cm/s)</i>	0.28 (0.23, 0.36)	0.36 (0.29, 0.45)	< 0.001	0.28 (0.21, 0.38)	0.41 (0.27, 0.65)	< 0.001
<i>COP_total_mean_jerk (cm/s³)</i>	90.90 (77.5, 105)	113.89 (94.3, 148)	< 0.001	87.66 (65.64, 111)	123.58 (80.71, 162)	< 0.001

	<i>Control</i>			<i>mTBI</i>		
<i>COPap_mean_jerk</i> (<i>cm/s³</i>)	68.02 (58.6, 81.9)	91.51 (75.4, 122)	< 0.001	67.09 (49.6, 92.7)	95.28 (60.91, 145)	< 0.001
<i>COPml_mean_jerk</i> (<i>cm/s³</i>)	43.06 (35.8, 52.2)	50.69 (39.42, 60.23)	< 0.001	39.29 (33.0, 50.7)	49.87 (37, 85.2)	< 0.001
<i>COPap_F80</i> (Hz)	0.15 (0.09, 0.26)	0.30 (0.22, 0.43)	< 0.001	0.20 (0.10, 0.31)	0.32 (0.22, 0.48)	< 0.001
<i>COPml_F80</i> (Hz)	0.37 (0.24, 0.48)	0.38 (0.27, 0.50)	0.123	0.30 (0.20, 0.35)	0.32 (0.23, 0.40)	0.061
<i>COPap_sent</i> (a.u)	0.56 (0.46, 0.66)	0.65 (0.57, 0.83)	< 0.001	0.55 (0.39, 0.60)	0.58 (0.49, 0.78)	< 0.001
<i>COPml_sent</i> (a.u)	0.67 (0.54, 0.84)	0.67 (0.56, 0.82)	0.849	0.59 (0.48, 0.72)	0.60 (0.43, 0.75)	0.854
<i>COP_Cross_Sent</i> (a.u)	1.25 (0.9, 1.54)	1.17 (0.87, 1.41)	0.071	1.03 (0.73, 1.40)	0.91 (0.43, 1.31)	0.025

Table 3

VPDI's medians and quartiles (Q25th and Q75th) recorded across participants for the *Control* and *mTBI* groups. Significant values from inferential tests are bolded.

	<i>Control</i>	<i>mTBI</i>	<i>Mann-Whitney test</i>
	<i>Median</i>	<i>Median</i>	<i>p-value</i>
	<i>(Q25th - Q75th)</i>	<i>(Q25th - Q75th)</i>	<i>(U, Z)</i>
<i>VPDI COP_area (%)</i>	46.38 (14.19, 103.74)	97.85 (36.01, 154.17)	0.013 (1398, -2.48)
<i>VPDI COP_total_length (%)</i>	11.34 (-3.32, 41.21)	23.40 (4.50, 46.56)	0.071 (1533, -1.80)
<i>VPDI COPap_range (%)</i>	26.37 (7.11, 51.36)	33.77 (12.76, 73.87)	0.176 (1624, -1.35)
<i>VPDI COPml_range (%)</i>	14.34 (-5.68, 47.81)	38.14 (18.16, 80.82)	0.005 (1329, -2.88)
<i>VPDI COPap_rms (%)</i>	13.45 (-3.54, 41.10)	23.31 (-0.67, 56.38)	0.088 (1533, -1.70)
<i>VPDI COPml_rms (%)</i>	13.42 (-4.14, 44.65)	36.06 (7.22, 72.04)	0.012 (1388, -2.53)
<i>VPDI COP_total_mean_vel (%)</i>	38.51 (23.96, 53.81)	60.45 (36.16, 82.02)	< 0.001 (1240, -3.26)
<i>VPDI COPap_mean_vel (%)</i>	46.81 (30.28, 64.18)	64.13 (37.66, 95.22)	0.013 (1396, -2.49)
<i>VPDI COPml_mean_vel (%)</i>	17.46 (6.32, 39.44)	46.01 (13.14, 69.35)	0.002 (1268, -3.12)
<i>VPDI COP_total_mean_jerk (%)</i>	22.03 (14.28, 35.65)	37.94 (14.94, 56.80)	0.008 (1366, -2.63)
<i>VPDI COPap_mean_jerk_ap (%)</i>	29.63 (16.41, 43.69)	41.50 (14.52, 63.70)	0.071 (1533, -1.80)
<i>VPDI COPml_mean_jerk_ml (%)</i>	12.45 (1.52, 21.16)	26.56 (8.71, 49.60)	< 0.001 (1255, -3.19)

	<i>Control</i>	<i>mTBI</i>	<i>Mann-Whitney test</i>
	<i>Median</i>	<i>Median</i>	<i>p-value</i>
	<i>(Q25th - Q75th)</i>	<i>(Q25th - Q75th)</i>	<i>(U, Z)</i>
<i>VPDI COPap_F80 (%)</i>	90.00 (21.54, 229.17)	75.00 (7.16, 182.73)	0.351 (1708.5, -0.93)
<i>VPDI COPml_F80 (%)</i>	6.25 (-17.79, 73.53)	12.69 (-14.46, 55.49)	0.962 (1886, -0.05)
<i>VPDI COPap_sent (%)</i>	24.26 (1.25, 41.98)	18.89 (-1.18, 43.89)	0.609 (1793, -0.51)
<i>VPDI COPml_Sent (%)</i>	0.99 (-14.79, 20.56)	1.72 (-16.04, 21.69)	0.996 (1895, -0.00)
<i>VPDI COP_crossent (%)</i>	-4.92 (-25.43, 14.59)	-20.97 (-40.59, 15.20)	0.151 (1607, -1.44)

Discussion

In this study we tested the hypothesis that individuals living with long-term effects of mild traumatic brain injury (mTBI) develop a larger dependency on their visual inputs to control their vertical posture. Consistent with this hypothesis our results show balance behavior of mTBI participants to deteriorate more abruptly in absence of visual inputs when compared to neurotypical controls. This deterioration was quantified by visuo-postural dependency indices (*VPDIs*) calculated independently for seventeen postural behavioral markers extracted from the body's center of pressure coordinates signals recorded during the execution of quiet bipedal stance.

As expected, our control group showed significant changes in body sway dynamics when visual inputs were disrupted. We observed significant increases of body sway amplitude and body sway velocity in both directions as well as increases to the total area and total length of sway. These findings are well aligned with previous reports demonstrating similar effects on both young and older healthy adults^{21,25,34-37}. These results support the rationale that vision is a sensory modality able to produce a reliable source of information for the control of the vertical posture. Under this rationale, visual inputs are constantly fed into the CNS resulting in the construction of an internal representation of one's relation to its surroundings (exteroception). This information is then utilized for the elaboration of motor outputs sent to postural muscles responsible for stabilization of major joints along the axial skeleton (e.g., ankle, knee, hip, intervertebral joints). Therefore, once vision is disrupted the quality of exteroception information is reduced and production of motor outputs is performed under reduced levels of certainty regarding the

current state of body dynamics. As a result, the vertical position becomes less stable resulting in larger amplitudes and faster body sway. This rationale is particularly supported by evidence showing that short-term disruptions of visual inputs are indeed related to modifications observed to the usual mechanisms of muscle activation utilized to maintain the vertical posture³⁸ as well as the presence of phase-locked body sway to motion of the visual field³⁹⁻⁴².

In addition to changes in the indices representing the temporal domain of analysis, we also observed significant increases to the distribution of the power spectrum density content of the COPap signal as well as increases in its temporal regularity. This finding is well aligned with several previous investigations^{27,28,43}. For example, Sim et al (2018)²⁷ utilized a discrete wavelet transform to study the energy content of the COPap signal within frequency bands below and above 1Hz. Their report highlights that a temporary interruption of visual inputs can cause significant shifts in the energy content of the signal in frequency bands up to 1Hz. Specific changes include reductions of energy content below 0.1Hz and increases on frequency bands up to 1Hz. These findings become relevant because the lower frequency content embedded in the COP signals has been linked to the visual neural loops involved in postural control. The relation of specific modalities of sensory information to the energy content of the COP signal has been an area of interest in human motor control. Studies of this nature are based on the effects of neural networks complexity to the time for completion of recurring neural loops. Under this principle, more complex sensory systems (composed by longer circuits) require longer delays to achieve their targeted structures resulting in the addition of lower frequencies components to the COP signal. Based on previous experimentation, visual inputs are credited to add energy content to frequencies below 0.1Hz, while vestibular and somatosensory inputs are linked to frequency bands of 0.1-0.5Hz and 0.5-1.0Hz, respectively^{28,44-46}. Our results corroborate with this body of evidence since the significant increase of *COPap_F80* are indicative of energy increase occurring towards larger frequencies when both vestibular and somatosensory neural relays become the main sources of sensory inputs. The lack of such modifications of *COPml_F80* needs to be further investigated.

Observations presented in Table 2 show that both groups responded with significant increases in most markers included in this study. However, analyzes of *VPDIs* indicated our mTBI cohort showed a disproportionate larger effect of visual disruption to body sway indices recorded in medial-lateral direction (Table 3). Specifically, we found significant increases in *VPDIs* indicating larger medial-lateral amplitude of sway accompanied by larger sway velocities, larger variations along the time (*COPml_rms*), and a larger presence of corrective actions during sway (*COPml_mean_jeark*). Even though we expected a significant deterioration of postural stability on our mTBI cohort, we did not expect a directional selectivity effect.

Disproportionate deterioration of body postural control in medial-lateral direction has been described in postural studies conducted in other populations^{47,48}. However, its driving mechanisms remain unclear. We speculate this effect may be driven by a combination of anatomical aspects of the axial skeleton and the segmental distribution of its postural muscles. Specifically, the presence of two lower limbs

positioned side-by-side and the reduced range-of motion of the knee joints in the frontal plane allow for relatively better medial-lateral stability. The parallel positioning of the two lower limbs increases the size of our base of support in the medial-lateral direction while allowing an effective transmission of reactive forces to partially counter-act the tendency of the body to lean on its side. As a result of this mechanical stability the number of muscles dedicated to control medial-lateral balance can be reduced therefore, making the muscular system dedicated to this type of control less redundant. Under our interpretation, even though this less redundant system functions well in health, it may reduce the ability of the central nervous system to adapt its neuromechanisms of control when the controller itself is compromised by pathologies. Instability will then follow.

Based on this rationale, a more redundant system, such as the one formed by the muscles controlling anterior-posterior sway will be able to respond better to impairments while delaying the progression of instability. This idea of a redundant system of control is well-known and has been demonstrated in multiple levels of analysis and in several types of motor actions⁴⁹⁻⁵¹. According to its principles, the CNS functions by exploring the motor redundancy offered by groups of muscles by co-vary their level of activity in a way that the final mechanical constraints of the motor task are respected. Despite its speculative status, the results presented here support this rationale. Note, that despite the fact the majority of *VPDIs* reaching significant threshold for inference, we also uncovered sizeable increases in *VPDIs* computed for anterior-posterior sway.

It is also important to note that mTBI affects a younger population who's professional and leisure activities involve faster reactions to environmental changes and failures to comply with their temporal constraints will increase the risk of segmental injuries and mTBI recurrences. This rationale is supported in part by the long-term effects mTBIs have to the CNS' white matter. Modern imaging studies have revealed significant reductions in the volume of important neural tracts. For example, Hulkover et al (2013)¹¹ compiled results from a large cohort of one hundred studies that utilized diffusion tensor imaging indices (e.g., fractional anisotropy) and suggested the existence of white-matter volume reduction in important areas for the integration of neural information within and between distinct brain areas including the superior longitudinal fascicule (SLF). SLF is responsible for intra-hemispherical exchange of neural information exchanged among the occipital, parietal, and frontal cortices, including their important areas of association located in the parietal and frontal cortices. Such reductions in volume occur due demyelination of these tracts resulting in extended delays in information exchange and integration of multiple sensory modalities intended to the creation of an internal representation of the body's current status. Delays of this nature have been recorded in mTBIs for visual and auditory stimuli⁸.

More recent studies have documented subtle but significant reductions in infratentorial areas of the CNS (ex. brainstem and cerebellum) in more severe cases of traumatic brain injury⁵²⁻⁵⁵. These observations suggest that a potential wide-spread effect of TBI to the CNS' nuclear and conduction system is likely. We interpret our results as corroborating with this rationale.

Conclusions

In this study we found evidence for a higher dependency of visual inputs to postural control in patients suffering from the long-term effects of mild traumatic brain injury. In addition, the utilization of quiet stance visuo-postural dependency indices (*VPDIs*) showed to be a robust method to investigate visual input dependency to one's postural control. Due to its multidimensional nature, this approach allows the assembly of a larger comprehensive panel of body sway characteristics.

Declarations

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Author contributions

A.DS, A.D., conceived the study and performed the experiments. A.DS carried out the analyses and wrote the main text. All authors critically revised the manuscript and approved its final version.

Competing financial interests and availability

The authors declare no competing financial interests. The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

a)



b)

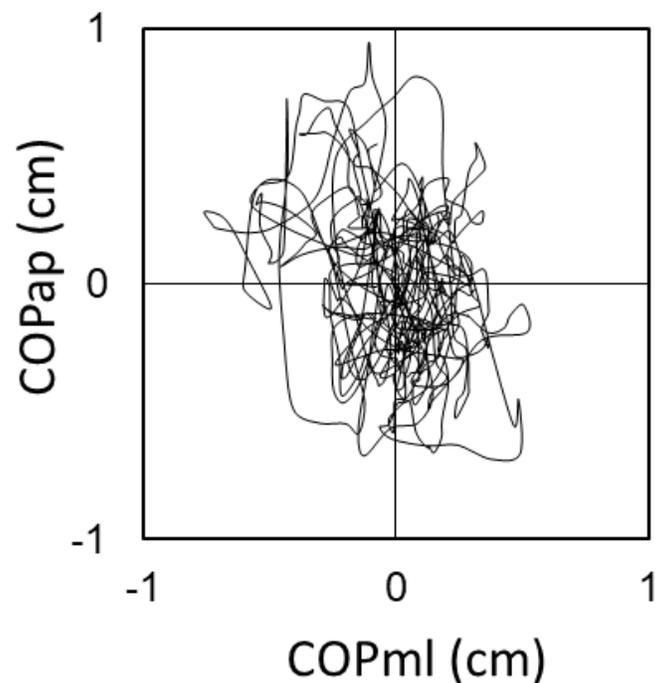


Figure 1

Panel a: Participants positioning during Vision and No-Vision trials. Panel b: example of posturographic recordings showing the migration of the COP coordinates in anterior-posterior (COPap) and medial-lateral (COPml) directions on the top of the force platform during the 120s of data recording.

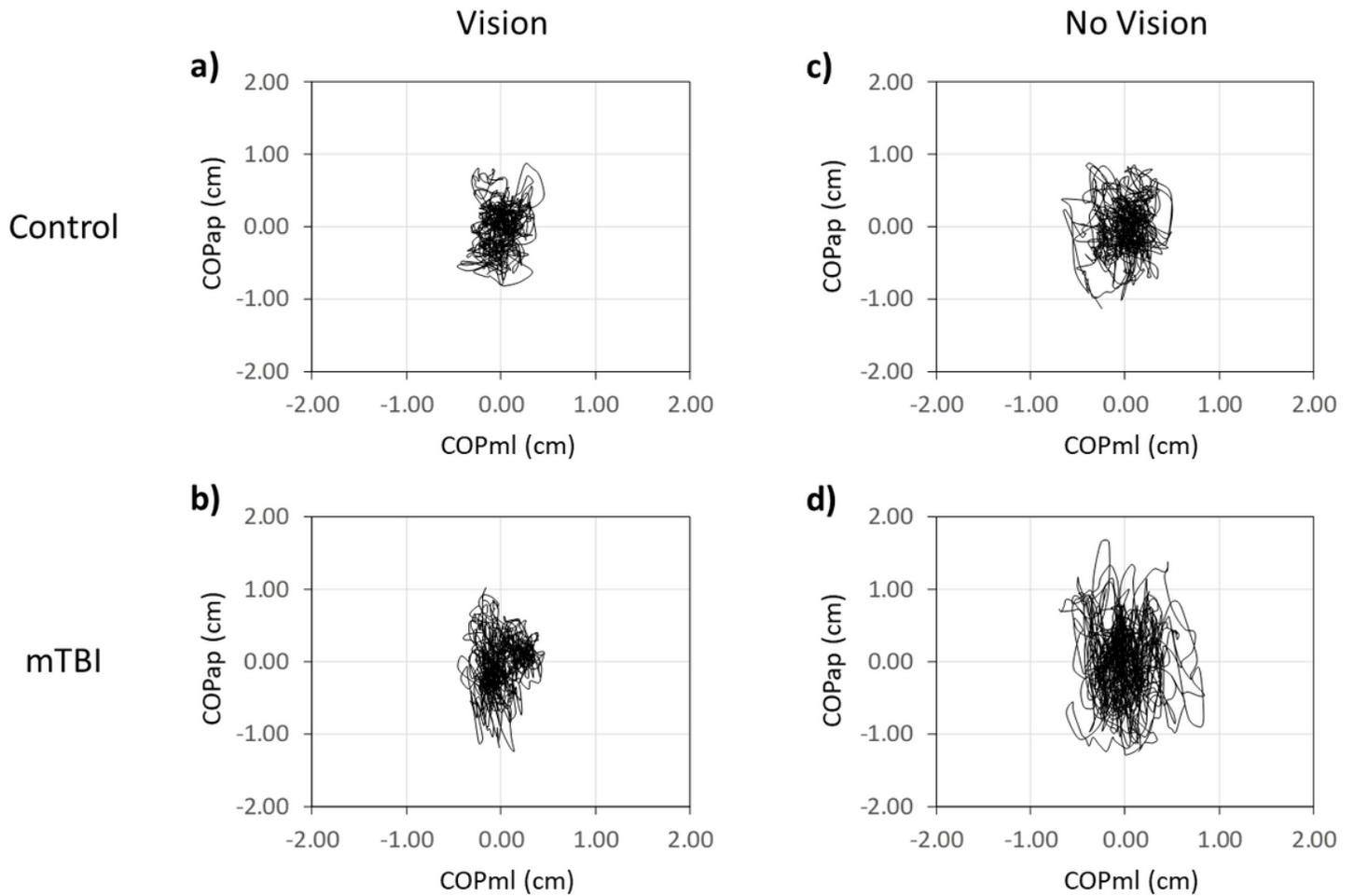


Figure 2

Panels a and c: Stabilograms recorded from a participant in the Control group under BEO and BEC conditions, respectively. Panels b and d: Stabilograms recorded from a participant in the mTBI group also under BEO and BEC conditions, respectively.