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Cortical activity during cognitive and motor tasks in mild traumatic brain injury: a pilot study using functional near-infrared spectroscopy

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Abstract

Background

Mild traumatic brain injury (mTBI) is a complex condition associated with persistent motor and cognitive dysfunction. Standard clinical tools such as the Glasgow Coma Scale and Sports Concussion Assessment Tool (SCAT) are used to inform diagnosis but may not detect subtle or persistent neurophysiological alterations. However, laboratory-based assessments, including motion capture and computerised cognitive testing, have demonstrated that motor and cognitive deficits can persist beyond apparent clinical recovery. Functional near-infrared spectroscopy (fNIRS) measures oxygenated haemoglobin (HbO_2) as a proxy marker of cortical activity during ecologically valid tasks and may provide objective biomarkers of lasting neural dysfunction following mTBI. Assessment of prefrontal, motor and visual cortical regions may provide insight into alterations in the processing required during cognitive and motor task performance.

Methods

Sixteen individuals with mTBI (at varying stages post-injury, range 7 days to 7.5 years) and 18 age-matched healthy controls completed a battery of seated computerised cognitive tests, standing balance tasks, and dual-task walking under simultaneous fNIRS recording. A 26-channel fNIRS system measured relative changes in HbO_2 across the pre-frontal cortex (PFC), supplementary motor area (SMA), primary motor cortex (M1), primary somatosensory cortex (S1), and primary visual cortex (V1). Gait and balance were concurrently assessed using body-worn inertial sensors.

Results

Groups were well-matched on demographic, neuropsychological, and clinical assessments. During computerised cognitive testing, the mTBI group demonstrated significantly lower M1 activation (p=.020) and lower V1 activation (p=.016) compared to controls, with no corresponding difference in reaction time performance. During dual-task walking, healthy controls significantly increased SMA recruitment relative to single-task walking (p=.011), whereas the mTBI group showed no such modulation (Group × Task interaction: p=.003). In addition, mTBI participants demonstrated greater foot strike angle during walking (p=.040) and higher sway frequency during foam standing with eyes open.

Conclusions

Despite equivalent neuropsychological and clinical scores, individuals with mTBI (at varying stages post-injury) exhibited abnormal cortical activity during cognitive and motor tasks and subtle gait and balance differences. The dissociation between normal task performance and aberrant cortical recruitment suggests that fNIRS is sensitive to neural dysfunction following mTBI irrespective of injury chronicity. These findings support fNIRS as a candidate objective digital biomarker of residual neural dysfunction following mTBI, warranting further validation in larger, stage-stratified cohort studies.

Introduction

In the United Kingdom, over one million people annually receive a diagnosis of mild traumatic brain injury (mTBI), commonly referred to as concussion, following head or neck trauma [1, 2]. Despite the term *mild*, mTBI is associated with a spectrum of neurological, cognitive, and motor dysfunction, including impaired memory, executive function, balance, and gait [3–14]. Historically regarded as a self-limiting injury with full recovery, mTBI is now increasingly recognised as a condition with potentially lasting neurophysiological sequelae that are difficult to detect using conventional tools and can last beyond the acute injury phase (≤ 72hours or <7days post injury, depending on the guideline used) [15].

Current diagnosis and recovery monitoring rely predominantly on self-report symptom inventories and subjective clinical rating instruments, such as the Glasgow Coma Scale (GCS) and the Sport Concussion Assessment Tool (5th Edition) (SCAT-5) [4, 16, 17].

These tools are susceptible to symptom under-reporting, effort-related variability, and insensitivity to subtle functional deficits, particularly in the post-acute phase when overt symptom burden has often resolved [18]. There is therefore a clinical imperative to develop objective, portable assessments capable of detecting residual neural dysfunction in individuals with mTBI across the recovery continuum.

Disruption to cerebral blood flow (CBF) is among the most durable neurophysiological sequelae of mTBI, documented in animal models within hours of injury and persisting over weeks to months [19–21]. In the acute and sub-acute stages, a period of cerebrovascular vulnerability is characterised by suppressed CBF coupled with elevated metabolic demand, representing a potentially dangerous mismatch between neural activity and its haemodynamic support [22]. Traditional methods for quantifying CBF, including transcranial Doppler ultrasonography, positron emission tomography, single photon emission computed tomography, and functional magnetic resonance imaging, are effective but impose significant practical limitations (i.e., they are non-portable, expensive, require stationary conditions, and cannot be applied during dynamic motor tasks) [23]. These constraints preclude their use as routine clinical assessments for mTBI.

Functional near-infrared spectroscopy (fNIRS) offers a complementary approach to CBF measurement that addresses many of these limitations. As a mobile, wearable brain imaging modality, fNIRS measures relative changes in cortical oxygenated (HbO_2) and deoxygenated haemoglobin via near-infrared light transmitted through the scalp. This provides a proxy measure of regional cortical activity during ecologically meaningful tasks, including walking and balance assessments [24–28]. Furthermore, examining cortical activity during motor tasks with fNIRS has been shown to be reliable and discriminates between young adults, older adults, and individuals with neurological conditions, supporting its utility as a functional neuroimaging tool in movement contexts [29].

The application of fNIRS to mTBI has yielded a growing but not fully developed body of evidence across cognitive, balance, and gait paradigms. For example, during cognitive testing, Kontos et al. [30] previously examined nine symptomatic concussed athletes, 15–45 days post-injury, during a computerised neurocognitive battery, finding reduced prefrontal cortex (PFC) HbO_2 across memory, digit-symbol substitution, and working memory tasks relative to healthy controls. Similarly, Urban et al. [31] examined patients with a history of mTBI longitudinally using a computerised attention task, first within 72 hours of injury and again once asymptomatic (mean ~ 21 days post-injury), finding reduced cortical activation and slower reaction times acutely. However, once asymptomatic, participants demonstrated a compensatory haemodynamic response that was greater than both the acute time point and healthy controls, while reaction time had normalised. This suggests that cortical upregulation may support cognitive recovery despite ongoing physiological disruption.

Additionally, Hocke et al. [32] demonstrated reduced prefrontal functional connectivity during a working memory task in adults with persistent post-concussion symptoms several months post-injury, with greater symptom severity correlating with lower coherence. During standing balance tasks, Helmich et al. [33] examined 62 concussed athletes (symptomatic and asymptomatic, timepoint not specified beyond current symptom status) across four standing conditions, finding significantly reduced left hemispheric frontopolar HbO_2 in symptomatic athletes during eyes-closed postural conditions. Conversely, Singh and Helmich [34] demonstrated increased parietal cortex activation in symptomatic athletes, interpreting this pattern as compensatory upregulation that nevertheless failed to resolve postural deficits.

During walking, Martini et al. [35] demonstrated elevated HbO_2 in the PFC in individuals with persistent post-concussion symptoms, within three months of injury. During dual-task walking, Hagen et al. [36] found that college-aged athletes with a history of two or more sport-related concussions, assessed at least one year post-injury, showed altered neural recruitment across the frontoparietal attention network, and primary motor and sensory cortices, despite comparable gait performance to controls, with compensatory PFC recruitment persisting well beyond symptom resolution.

Furthermore, an fNIRS study conducted by Hocke et al. [32] found that people with persistent post-concussive symptoms showed reduced connectivity between prefrontal and motor areas compared with healthy controls and that symptoms severity was associated with reduced coherence. In addition, one magnetoencephalography study [37] found altered activation following

concussion across frontoparietal and occipitotemporal regions, including abnormal recruitment of areas involved in visuoperceptual processing, despite comparable behavioural performance to controls in visual working memory tasks.

Collectively, these studies point toward altered cortical haemodynamics across multiple cortical regions following mTBI across task contexts, though directional inconsistency likely reflects differences in injury chronicity (i.e., studies span acute (≤ 72 hours) through to several years post-injury), symptom status, and the cortical regions examined (i.e., largely only PFC), underscoring the need for multi-region assessment across standardised paradigms.

The present study aimed to examine relative HbO_2 across five cortical regions of interest (PFC, supplementary motor area (SMA), primary motor cortex (M1), primary somatosensory cortex (S1), and primary visual cortex (V1).) during seated cognitive, standing, and walking tasks in individuals with mTBI at varying stages post-injury compared to healthy controls. These regions were selected based on their established involvement in cognitive control, motor planning, sensorimotor integration, and visual processing during complex motor behaviours [38, 39]. Hypotheses were task-specific, informed by the existing fNIRS concussion and wider neurological literatures.

Specifically, we hypothesised that:

1. During cognitive testing, individuals with mTBI would demonstrate reduced HbO_2 relative to healthy controls, consistent with previous fNIRS concussion studies reporting attenuated cortical activation during neurocognitive tasks.
2. During motor tasks, individuals with mTBI would demonstrate increased HbO_2 relative to healthy controls, consistent with compensatory cortical upregulation observed during walking and balance tasks in mTBI and wider neurological populations.
3. Compensatory increases in HbO_2 would be most pronounced under conditions of increased task demand, including dual-task walking and foam-surface standing.

Methods

Study Design and Ethical Approval

This cross-sectional pilot study recruited individuals with mTBI and healthy controls. All procedures were conducted in accordance with the Declaration of Helsinki, and ethical approval was obtained from the Northumbria University research ethics committee. Written informed consent was obtained from all participants prior to enrolment.

Participants

Participants were eligible for inclusion if they were aged 18–50 years. Individuals in the mTBI group were required to have received a clinical diagnosis of mTBI from a qualified healthcare professional based on standard criteria including SCAT-5 and GCS assessment with clinical judgment, and to be ≥ 7 days post-injury. Given the absence of a minimum chronicity criterion, the sample spans the post-acute to chronic recovery continuum (range: 7 days to 7.5 years); time since injury is reported in Table 1. Exclusion criteria included any concurrent musculoskeletal, neurological, or sensory impairment that could independently explain cognitive or mobility dysfunction. Participants were also excluded if they had a history of neurological conditions (unrelated to mTBI for that group), major psychiatric diagnosis, or uncorrected visual impairment.

Table 1

Demographic, cognitive, visual, and clinical characteristics of participants.

Demographics		**HC (n = 18) Mean (SD)**	**mTBI (n = 16) Mean (SD)**	**P**
	Age (years)	**28.83 (9.78)**	**26.62 (7.76)**	**0.597c**
	Gender	13f / 5m	8f / 8m	0.183a
	Education (years)	18.33 (3.18)	19.44 (5.82)	0.798c
	Height (cm)	172.41 (8.22)	176.90 (9.50)	0.177b
	Weight (kg)	70.57 (25.04)	82.50 (15.19)	0.224c
	FES-I	21.82 (7.44)	19.85 (6.19)	0.377c
Cognition	MoCA	27.56 (1.78)	27.20 (2.04)	0.711c
	DT walk error %	21.59% (1.88)	35.68% (2.28)	0.086b
	Max digit span (sitting)	6.12 (1.32)	6.33 (1.11)	0.455c
	SRT (ms)	294.94 (45.01)	295.34 (41.43)	0.825b
	SRT CV%	15.26%	14.03%	0.861d
	CRT (ms)	397.99 (61.08)	395.54 (71.28)	0.862b
	CRT CV%	15.34%	18.02%	0.218d
	DV (ms)	427.70 (36.43)	442.39 (43.18)	0.263b
	DV CV%	8.52%	9.56%	0.480d
	TMT-A (s)	21.65 (6.77)	22.32 (11.50)	0.766c
	TMT-B (s)	44.08 (18.03)	44.04 (12.24)	0.994b
Visual	Contrast sensitivity	1.72 (0.11)	1.76 (0.01)	0.302c
	Visual acuity	-0.06 (0.08)	-0.05 (0.13)	0.363b
Clinical	SCAT5 symptom score	8.33 (14.19)	14.00 (11.82)	0.299b
	Time since injury (days) – Median (IQR)	-	1119.50 (55.25–2742.75)	-

[Values are mean (SD) unless indicated. Significant values in bold. Footnotes: aX², b independent samples t-test, c Mann-Whitney U test, d Levene's test for equality of variances. Abbreviations: CV = coefficient of variation; CRT = choice reaction time; DT = dual-task; DV = digit vigilance; FES-I = Falls Efficacy Scale—International; HC = healthy controls; MoCA = Montreal Cognitive Assessment; mTBI = mild traumatic brain injury; SCAT-5 = Sport Concussion Assessment Tool—5th Edition; SRT = simple reaction time; TMT = Trail Making Test. Units: cm = centimeters; kg = kilograms; ms = milliseconds; s = seconds}

Outcome Measures and Assessments

Demographic data were collected including age, sex, height, weight and years of formal education. Baseline global cognition was assessed using the Montreal Cognitive Assessment (MoCA) [40], attention and executive function using Trails Making Test Parts A and B (TMT-A/B) [41], and working memory using the backward digit span subtest [42]. Falls-related self-efficacy was quantified with the Falls Efficacy Scale (International version) (FES-I) [43]. Visual contrast sensitivity (via Mars Perceptrix) and visual acuity (via LogMar) were additionally assessed to characterise any between-group visual differences that could confound cortical findings. Clinical assessment involved the current post-concussion symptom burden assessed with the SCAT-5 symptom inventory and the time (in days) post injury [16].

Task Protocol

Task Protocol
All tasks were performed under simultaneous fNIRS recording. A 20-second baseline period was recorded at the start of each task and each task lasted 2 minutes. Tasks were performed in three sequential blocks:

Cognitive testing: Participants completed three computerised cognitive tests in a seated position: Simple Reaction Time (SRT), Choice Reaction Time (CRT), and Digit Vigilance (DV) [44].

Balance: Standing tasks were performed with feet hip-width apart (standardised using an APDM positional template) with arms side by side, across four conditions: eyes open on a firm surface (EO-Firm), eyes closed on a firm surface (EC-Firm), eyes open on a foam surface (EO-Foam), and eyes closed on a foam surface (EC-Foam).

Walking: Participants walked back and forth over a 10-metre course with 180-degree turns under two conditions: single-task walking (ST) and dual-task walking (DT), with a concurrent forward digit span task calibrated to each participant's individual sitting span.

Inertial Sensor Measures

Participants wore Opal V2 inertial measurement units (APDM Wearable Technologies, a Clario company, Portland, OR, USA) at the lower back, sternum, bilateral wrists, and bilateral feet [45]. Sensors were time-synchronised with the fNIRS system via Portasync (Artinis Medical Systems, Doetinchem, The Netherlands). Gait outcomes were selected a priori based on previous work[46] identifying gait characteristics across pace, rhythm, variability and asymmetry domains as relevant to cognitive decline, and included gait speed, cadence, stride length, stride time, foot strike angle, and stride length variability. Balance outcomes included sway area, jerkiness (mean square jerk), sway velocity, root mean square (RMS), and 95th percentile sway frequency.

fNIRS Acquisition and Processing

Cortical activity was recorded using a continuous-wave fNIRS system (OctaMon+ Brite24, Artinis Medical Systems) at 50 Hz via Oxysoft software. The montage comprised 20 long-separation channels (inter-optode distance 3 cm) and 6 short-separation channels (inter-optode distance 1 cm). A 3D digitiser (Polhemus Patriot, Colchester, VT, USA) was used to register optode positions relative to standard anatomical landmarks. Channel coordinates were entered into NIRS-SPM (Statistical Parametric Mapping for NIRS; http://www.nitrc.org/projects/nirs_spm) and registered to Montreal Neurological Institute (MNI) standard brain space, enabling assignment of channels to cortical regions of interest (ROI): PFC, SMA, M1, S1, and V1.

fNIRS data processing was performed in line with our previous fNIRS studies and consensus guidance via MATLAB 2022b [47–50]. Steps included: (1) low-pass filtering (cut-off 0.14 Hz); (2) baseline correction—for walking trials, the mean of the 20-second pre-walk standing period was subtracted; for standing trials, the mean of the initial 20 seconds of the seated baseline was subtracted; for cognitive trials, the mean of the 10-second pre-trial period was subtracted; (3) short-channel regression to remove systemic physiological noise (1.5 cm short-separation channels subtracted from corresponding 3 cm channel signals); (4) visual signal inspection; and (5) channel averaging within each ROI.

Statistical Analysis

Statistical analyses were conducted in SPSS (v26, IBM, Chicago, IL, USA). Data normality was assessed using Kolmogorov-Smirnov tests, and parametric or non-parametric tests were selected accordingly for descriptive comparisons (independent t-tests or Mann-Whitney U tests; chi-square for categorical variables). Group differences in demographic and clinical characteristics were examined using these tests (Table 1).

Separate linear mixed effects models (LMEM) were fitted for each cortical ROI and for each class of outcome measure (gait, balance) across the three task paradigms (cognitive, walking, balance). Each model included fixed effects of Group (HC, mTBI), Task (e.g., SRT/CRT/DV for cognitive testing; ST/DT for walking; EO-Firm/EC-Firm/EO-Foam/EC-Foam for balance), and their interaction (Group × Task). A random intercept per participant was included to account for the repeated measures structure of the

data. A random intercept structure was selected as the most parsimonious specification for this pilot sample. Given the pilot and exploratory nature of this study, no correction for multiple comparisons was applied, and p<.05 was considered statistically significant.

Results

Participant Characteristics

Thirty-four participants were enrolled, specifically 18 healthy controls and 16 individuals with mTBI at varying stages post-injury (range: 7 days to several years; median 1190.5 days (IQR: 55.25 to 2742.75 days) since injury; see Table 1). Groups were well-matched on age (mTBI: 26.62 ± 7.76 years; HC: 28.83 ± 9.78 years), sex distribution (mTBI: 8f/8m; HC: 13f/5m), and education. There were no significant between-group differences on any demographic, cognitive, visual, or clinical assessment (all p>.05), including MoCA, TMT-A/B, SCAT-5 symptom score, reaction time measures, contrast sensitivity, or visual acuity (Table 1). Of note, dual-task walk error rate showed a numerically large difference (mTBI: 35.68% vs HC: 21.59%) that approached but did not reach statistical significance (p=.086), potentially reflecting limited statistical power in this pilot sample.

Cortical Activity During Computerised Cognitive Testing

Table 2 presents cortical HbO_2 across all ROIs during the three computerised cognitive tests. The LMEM revealed a significant main effect of Group for M1 (F = 5.672, p=.020) and V1 (F = 6.085, p=.016), with the mTBI group demonstrating lower HbO_2 in both regions compared to healthy controls across cognitive tasks. These group differences were consistent across all three reaction time tests (SRT, CRT, DV), with no significant Group × Task interaction observed for any ROI, indicating that the cortical suppression in mTBI was not task-specific but generalised across the cognitive battery. No significant group, task, or interaction effects were observed for PFC, SMA, or S1 during cognitive testing. A moderate negative correlation was observed between SCAT-5 symptom score and M1-HbO_2 during the SRT (r=-.476, n = 19, p=.039).

Table 2

Cortical HbO_2 (mean [SD]) during computerised cognitive testing in healthy controls and mTBI. Values reflect relative change from baseline.

ROI	HC SRT	mTBI SRT	HC CRT	mTBI CRT	HC DVT	mTBI DVT	Group F(p)	Task F(p)	Group × Task F(p)
PFC	-0.194 (0.113)	-0.008 (0.234)	0.015 (0.176)	-0.092 (0.134)	-0.025 (0.177)	0.002 (0.117)	0.365 (.550)	0.522 (.596)	2.517 (.089)
SMA	0.027 (0.215)	-0.081 (0.168)	0.062 (0.196)	0.021 (0.184)	0.033 (0.156)	-0.022 (0.233)	0.338 (.563)	0.184 (.832)	1.553 (.219)
M1	0.018 (0.184)	-0.028 (0.204)	0.022 (0.182)	-0.101 (0.137)	0.093 (0.145)	-0.009 (0.189)	5.672 (.020)*	1.535 (.222)	0.363 (.697)
S1	-0.029 (0.216)	-0.054 (0.200)	-0.049 (0.136)	-0.099 (0.158)	0.052 (0.139)	-0.046 (0.193)	2.544 (.114)	1.633 (.201)	0.419 (.659)
V1	0.016 (0.195)	-0.046 (0.253)	0.055 (0.227)	-0.130 (0.204)	0.019 (0.157)	-0.039 (0.132)	6.085 (.016)*	0.188 (.829)	1.100 (.337)

[indicates significant Group main effect (p<.05).Abbreviations: CRT = choice reaction time; DV = digit vigilance; HC = healthy controls; M1 = primary motor cortex; mTBI = mild traumatic brain injury; PFC = pre-frontal cortex; ROI = region of interest; S1 = primary somatosensory cortex; SMA = supplementary motor area; SRT = simple reaction time; V1 = primary visual cortex}*

Gait and Cortical Activity During Walking

Table 3 presents gait kinematics and cortical HbO_2 during single-task and dual-task walking. The mTBI group exhibited a significantly greater foot strike angle than healthy controls across both walking conditions (Group main effect: F = 4.621, p=.040). Both groups showed significant task effects: cadence, stride length, stride time, and gait speed all differed between ST and DT conditions (all p<.01), reflecting the anticipated cost of dual-task performance. There were no significant between-group differences in any other gait metric.

Table 3

Gait metrics and cortical HbO_2 (mean [SD]) during single-task (ST) and dual-task (DT) walking.

Gait	HC ST	mTBI ST	HC DT	mTBI DT	Group F(p)	Task F(p)	Group × Task F(p)
Foot strike angle (°)	24.67 (4.48)	27.47 (3.51)	24.03 (4.32)	27.15 (3.62)	4.621 (.040)*	2.065 (.161)	0.215 (.646)
Cadence (steps/min)	108.58 (8.00)	105.32 (5.60)	107.74 (9.71)	102.16 (7.33)	2.743 (.108)	8.533 (.007)*	2.865 (.101)
Stride length (m)	1.22 (0.12)	1.26 (0.12)	1.20 (0.12)	1.23 (0.13)	0.503 (.483)	9.674 (.004)*	0.117 (.735)
Stride time (s)	1.11 (0.08)	1.14 (0.06)	1.12 (0.10)	1.18 (0.09)	2.573 (.119)	10.263 (.003)*	2.976 (.095)
Gait speed (m/s)	1.12 (0.13)	1.11 (0.13)	1.08 (0.15)	1.05 (0.16)	0.375 (.544)	11.089 (.002)*	0.814 (.374)
Stride length variability	0.05 (0.04)	0.08 (0.06)	0.07 (0.07)	0.06 (0.04)	0.452 (.507)	0.003 (.957)	2.543 (.121)
Cortical Activity (HbO_2)							
PFC	0.021 (0.104)	-0.004 (0.095)	0.027 (0.092)	0.020 (0.143)	2.895 (.969)	1.345 (.811)	0.856 (.465)
SMA	-0.069 (0.149)	0.050 (0.137)	0.200 (0.182)	0.022 (0.074)	0.332 (.575)	8.981 (.011)*	13.558 (.003)*
M1	0.010 (0.082)	-0.023 (0.257)	0.095 (0.214)	0.001 (0.123)	1.759 (.200)	1.097 (.307)	0.511 (.483)
S1	-0.027 (0.072)	0.015 (0.181)	0.048 (0.068)	-0.001 (0.105)	0.052 (.822)	1.970 (.176)	2.849 (.107)
V1	-0.028 (0.098)	0.012 (0.152)	0.042 (0.064)	-0.050 (0.196)	0.185 (.672)	0.219 (.645)	3.276 (.086)

[indicates statistical significance (p<.05). Abbreviations: DT = dual-task; HC = healthy controls; M1 = primary motor cortex; mTBI = mild traumatic brain injury; PFC = pre-frontal cortex; ROI = region of interest; S1 = primary somatosensory cortex; SMA = supplementary motor area; ST = single-task; V1 = primary visual cortex.* Units: ° = degrees; cm = centimetres; s = seconds; steps/min = steps per minute; m/s = metres per second.*}*

A significant Group × Task interaction was found for SMA (F = 13.558, p=.003). Post-hoc inspection revealed that healthy controls significantly increased SMA-HbO_2 from single-task to dual-task walking (F = 8.981, p=.011), consistent with the additional motor planning and attentional demands of concurrent cognitive task performance. By contrast, individuals with mTBI showed no modulation of SMA activity across conditions (ST and DT values were similar). No significant group effects or Group × Task interactions were observed for HbO_2 in the PFC, M1, S1, or V1.

Balance and Cortical Activity During Standing

Table 4 presents balance metrics and cortical HbO_2 across the four standing conditions. Sway area and jerk showed highly significant task effects (both p<.001), consistent with expected increases in postural challenge across conditions (firm to foam surface; eyes open to eyes closed). Sway frequency was significantly higher in the mTBI group during foam standing with eyes open relative to controls, though no significant Group main effects were observed for any other balance metric.

Table 4

Balance metrics and cortical HbO_2 (mean [SD]) during standing across four conditions.

Balance	Firm EO HC	Firm EO mTBI	Firm EC HC	Firm EC mTBI	Foam EO HC	Foam EO mTBI	Foam EC HC	Foam EC mTBI	Group F(p)	Task F(p)	Group × Task F(p)
Sway area (m^2)	0.04 (0.10)	0.08 (0.20)	0.07 (0.09)	0.06 (0.08)	0.06 (0.04)	0.12 (0.20)	0.15 (0.14)	0.19 (0.15)	0.636 (.431)	11.941 (< .001)*	1.000 (.396)
Jerk (m^2/s)	3.78 (6.96)	7.13 (16.91)	8.03 (8.00)	7.86 (9.25)	5.86 (4.84)	14.17 (20.04)	21.09 (18.18)	29.95 (26.69)	1.436 (.240)	25.142 (< .001)*	1.529 (.212)
Sway velocity (m/s)	0.57 (0.39)	0.60 (0.35)	0.99 (1.19)	0.80 (0.71)	0.68 (0.39)	0.71 (0.63)	0.90 (0.57)	0.95 (1.22)	0.001 (.972)	2.026 (.116)	2.026 (.833)
RMS (m^2)	0.08 (0.05)	0.09 (0.35)	0.19 (0.37)	0.10 (0.06)	0.095 (0.04)	0.11 (0.08)	0.14 (0.05)	0.15 (0.07)	0.133 (.717)	1.614 (.192)	1.036 (.381)
Frequency 95% (Hz)	0.87 (0.14)	0.88 (0.15)	0.85 (0.18)	0.88 (0.23)	0.92 (0.17)	1.07 (0.20)	0.96 (0.13)	1.07 (0.22)	2.705 (.110)	10.188 (< .001)*	2.382 (.075)
Cortical Activity (HbO_2)											
PFC	-0.062 (0.137)	-0.063 (0.134)	-0.057 (0.132)	-0.078 (0.136)	-0.055 (0.133)	-0.063 (0.135)	-0.050 (0.139)	-0.048 (0.134)	0.882 (.364)	2.312 (.081)	1.042 (.378)
SMA	-0.038 (0.380)	-0.162 (0.334)	-0.395 (0.397)	-0.172 (0.348)	-0.048 (0.403)	-0.143 (0.349)	-0.052 (0.355)	-0.167 (0.341)	0.726 (.402)	0.726 (.539)	1.200 (.315)
M1	-0.087 (0.351)	-0.210 (0.356)	-0.083 (0.361)	-0.215 (0.361)	-0.101 (0.367)	-0.221 (0.346)	-0.081 (0.355)	-0.199 (0.331)	0.797 (.381)	1.856 (.145)	0.219 (.883)
S1	0.088 (0.519)	-0.239 (0.401)	-0.075 (0.523)	-0.235 (0.419)	-0.086 (0.538)	-0.245 (0.404)	-0.083 (0.528)	-0.239 (0.393)	0.857 (.362)	0.517 (.672)	0.079 (.971)
V1	0.057 (0.290)	-0.276 (0.384)	-0.054 (0.308)	-0.289 (0.380)	-0.082 (0.293)	-0.289 (0.392)	-0.077 (0.323)	-0.282 (0.383)	2.895 (.100)	1.345 (.265)	0.856 (.467)

[indicates statistical significance (p<.05). Abbreviations: EC = eyes closed; EO = eyes open; HC = healthy controls; M1 = primary motor cortex; mTBI = mild traumatic brain injury; PFC = pre-frontal cortex; RMS = root mean square; ROI = region of interest; S1 = primary somatosensory cortex; SMA = supplementary motor area; V1 = primary visual cortex. Units: m^2 = square metres; m^2/s = square metres per second; m/s = metres per second; Hz = hertz.}*

A consistent pattern of lower cortical HbO_2 in mTBI compared to healthy controls was observed across all standing conditions for V1 and S1, and to a lesser extent M1. Notably, V1 HbO_2 approached significance as a Group main effect (F = 2.895, p=.100), and S1 showed a numerically large and directionally consistent group difference across all four standing conditions (mTBI consistently ~ 0.32 units below HC across all conditions). While these did not reach the p<.05 threshold, the consistency and magnitude of this pattern across conditions is worthy of note given the pilot sample size.

Discussion

To our knowledge, this is the first study to employ fNIRS to simultaneously examine cortical activity across five regions of interest during cognitive and motor tasks in individuals with mTBI. Hypotheses were task-specific, we predicted reduced HbO_2 during cognitive tasks, consistent with prior fNIRS concussion literature showing attenuated cortical activation during neurocognitive testing, and increased HbO_2 during motor tasks (gait and balance), consistent with compensatory upregulation reported across mTBI and wider neurological populations. The mTBI and healthy control groups were not different on clinical or cognitive tests, but the cognitive cortical findings largely confirmed our hypotheses, while the motor task cortical findings did not, and represent the most novel and unexpected contribution.

Cortical activity during cognitive testing

The significant reduction in M1 and V1 HbO_2 during computerised cognitive testing in the mTBI group, in the absence of any reaction time performance difference, is consistent with prior fNIRS concussion literature. Kontos et al. [30] were the first to demonstrate this pattern, reporting reduced prefrontal HbO_2 during neurocognitive testing in concussed athletes at 15–45 days post-injury, with poorer performance accompanying the cortical reduction. Our findings extend this to a sample assessed at substantially longer post-injury intervals, and notably in M1 and V1 rather than PFC, which suggests that cortical suppression during cognitive engagement is not confined to the prefrontal regions. The preservation of task performance alongside reduced cortical activity suggests altered neural recruitment following mTBI. One possible framework for interpreting such changes is the Compensation-Related Utilisation of Neural Circuits Hypothesis (CRUNCH)[51], which proposes that individuals with reduced neural efficiency recruit greater cortical resources at lower task demands to maintain performance, but reach a ceiling in recruitment at lower levels of task difficulty than healthy controls. Once this compensatory capacity is exhausted, further increases in task demand may be accompanied by a plateau or reduction in cortical activation [51, 52]. This pattern has been demonstrated in mTBI using fNIRS by Hagen et al. [36], who found that concussed individuals showed a reduction in PFC activation at higher cognitive loads while performance remained intact, consistent with depletion of cognitive processing capacity rather than preserved neural efficiency. Reduced M1 activation during seated cognitive tasks may reflect impaired motor preparation circuitry, consistent with TMS evidence of altered intracortical inhibition in mTBI [53, 54]. While reduced V1 activity during visually-presented cognitive tasks may indicate disrupted visual cortical processing, which is coherent with documented oculomotor and visual pathway dysfunction following mTBI [55, 56]. The negative correlation between SCAT-5 symptom score and M1-HbO_2 during SRT further supports the clinical relevance of this suppression, linking residual symptom burden to measurable cortical dysfunction even in the absence of performance differences. Similarly, Hocke et al. [32] demonstrated that greater symptom severity correlated with reduced cortical coherence in adults with persistent post-concussion symptoms during a working memory task.

Cortical activity during walking

Contrary to our hypothesis of increased cortico-motor recruitment during walking, the mTBI group did not demonstrate increased SMA recruitment relative to controls during either walking condition. The most compelling finding was the significant Group × Task interaction for SMA, as the SMA is a key cortical region for motor planning, and its recruitment during complex walking tasks has been demonstrated across neurological populations, where increased SMA engagement reflects the additional planning demands of challenging gait [25, 57, 58]. Healthy controls in the current study appropriately upregulated SMA from single to dual-task walking, consistent with established models of cognitive-motor integration during dual-task paradigms. The mTBI group showed no such modulation with a flat SMA profile across both conditions, which may represent a failure of adaptive cortical upregulation that was entirely invisible to gait kinematics. Hagen et al. [36] similarly found altered neural recruitment across the frontoparietal network and primary motor and sensory cortices in athletes with repeat concussions during dual-task walking, with compensatory PFC upregulation persisting beyond medical clearance, despite normal gait performance. This reinforces that gait metrics alone are insufficient to detect the neurophysiological cost of walking in this population [13]. Furthermore, athletes with repeat concussions showed reduced S1 and M1 activation during dual task walking compared with single task whereas controls showed the opposite pattern with increased M1 and S1 activation during the dual-task condition.

The failure to upregulate SMA in our sample may reflect a reduced capacity for adaptive motor planning under increased cognitive load, which combined with the near-significant increase in dual-task cognitive error rate, suggests a genuine motor-cognitive integration impairment that standard assessments do not capture. Martini et al. [35] reported increased PFC-HbO_2 during gait in those with persistent post-concussion symptoms within three months of injury, which we did not replicate at longer post-injury intervals. This may reflect the wide chronicity range of our sample attenuating any compensatory PFC signal, or stage-dependent differences in the cortical response to walking that require longitudinal investigation to resolve.

Cortical activity and balance

The balance findings also ran contrary to our compensation hypothesis. A directionally consistent pattern of lower HbO_2 in mTBI was observed across V1 and S1 for all four standing conditions, approaching significance for V1, with S1 showing a numerically large group difference (~ 0.32 units lower in mTBI) across all conditions. This contrasts with the compensatory parietal

upregulation reported by Singh and Helmich [59], who found increased parietal cortex activation in symptomatic concussed athletes across all balance conditions, but is directionally consistent with Helmich et al.[33], who found reduced frontopolar HbO_2 in symptomatic athletes during eyes-closed balance conditions. Reduced S1 activity across standing conditions in our sample may indicate impaired somatosensory cortical processing, consistent with evidence that mTBI disrupts central sensorimotor integration and proprioceptive weighting during balance [60, 61]. Sensory phenotyping studies in mTBI have demonstrated that individuals with chronic injury show impaired proprioceptive contribution to balance, often relying disproportionately on visual and vestibular inputs (i.e., a pattern that would be particularly destabilising under foam-surface conditions that reduce somatosensory reliability) [62]. This is consistent with the elevated sway frequency observed in our mTBI group during foam-surface, eyes-open standing, a condition that maximally challenges sensory reweighting. The discrepancy between our balance cortical findings and the Singh and Helmich [59] compensatory upregulation pattern may reflect differences in symptom status, ROI coverage, or the wide chronicity range of our sample, and underscores the need for longitudinal, stage-stratified studies in this domain. The lack of group differences in the remaining sway metrics may reflect limited task difficulty, as participants stood with their feet apart rather than together, potentially reducing the challenge to postural control.

Gait kinematics

The greater foot strike angle observed in the mTBI group across both walking conditions warrants careful interpretation. A larger foot strike angle indicates a more heel-dominant contact pattern, which may represent a more cautious, proprioceptively guided gait strategy. This could reflect a compensatory adaptation, whereby individuals with mTBI adopt a more deliberate heel-strike pattern to accommodate persistent subtle sensorimotor deficits. Such an interpretation is consistent with the broader findings of this study, in which preserved behavioural performance was accompanied by evidence of altered neural function. Further research using larger samples and comprehensive kinetic and kinematic analyses is needed to clarify the mechanisms underlying this finding.

Limitations

The sample size (n = 34) is modest and limits statistical power, which likely accounts for the number of near-significant trends that did not reach threshold. Additionally, due to the exploratory nature of this work, no correction for multiple comparisons was applied, which could be addressed in future work. The wide range in time since injury (7 days to 7.5 years) represents an important limitation, as participants at different stages of recovery may exhibit distinct cortical activation profiles. Combining these individuals into a single group may attenuate or obscure stage-specific effects. Future studies should examine time since injury as a covariate and either stratify participants by injury chronicity or recruit within defined post-injury windows. This cross-sectional design precludes causal inference or characterisation of the cortical response trajectory within individuals, which could be examined in longitudinal work in future research.

Conclusions

This pilot study provides the first multi-region fNIRS characterisation of cortical activity during cognitive, balance, and walking tasks in individuals with mTBI. Despite comparable clinical and neuropsychological performance, participants with mTBI exhibited reduced M1 and V1 activation during cognitive testing, failed to upregulate SMA activation during dual-task walking, and demonstrated reduced somatosensory cortical engagement during balance. These alterations were not detected by standard clinical assessments, highlighting a dissociation between preserved behavioural performance and altered cortical recruitment. Collectively, these findings support the potential of fNIRS as a biomarker of residual neural dysfunction following mTBI. Larger, longitudinal studies stratified by time since injury are now needed to validate these findings, define the trajectory of cortical recovery, and establish the clinical utility of fNIRS in mTBI assessment.

Declarations

Author Contributions

PT and SS conceived and designed the study. PT, LG, DP collected data. PT, DP, AG performed data analysis. PT, SS drafted the manuscript. RV, DM, LK, RM revised the manuscript critically for intellectual content. All authors approved the final version for

submission.

Declaration of Conflicting Interests

The authors declare no conflicts of interest with respect to the research, authorship, or publication of this article.

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Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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