Persistent alterations of cortical hemodynamic response in asymptomatic concussed patients

Allyssa K Memminia,1, Xin Sun2, Xiaosu Hu3,4, Jessica Kim2,4, Noelle K Herzog5,6, Mohammed N Islam5, Daniel H Weissman5,6, Alexander J Rogers1,7, Ioulia Kovelman3,4 & Steven P Broglio1

1Michigan Concussion Center, University of Michigan, Ann Arbor, MI 48109, USA
2Department of Psychology, University of Michigan, Ann Arbor, MI 48109, USA
3School of Dentistry, Department of Biologic and Materials Sciences & Prosthodontics, University of Michigan, Ann Arbor, MI 48109, USA
4Center for Human Growth and Development, University of Michigan, Ann Arbor, MI 48109, USA
5Department of Psychology, University of Toledo, Toledo, OH 43606, USA
6Department of Electrical and Computer Engineering, University of Michigan, Ann Arbor, Michigan MI 48109, USA
7Department of Emergency Medicine, Michigan Medicine, Ann Arbor, MI 48109, USA

*Author for correspondence: Tel.: +1 734 615 9330; amemmini@umich.edu

Aim: The underlying neurophysiological effects of concussion often result in attenuated cognitive and cortical function. To understand the relation between cognition and brain injury, we investigated the effects of concussion on attentional networks using functional near-infrared spectroscopy (fNIRS). Materials & methods: Healthy controls and concussed patients, tested within 72 h from injury (T1) and after symptoms resolved (T2) completed a computerized attention task during fNIRS imaging. Results: T1 patients exhibited slower reaction times and reduced brain activation pattern relative to healthy controls. Interestingly, the cortical oxygenation hemoglobin response at T2 was greater relative to T1 and healthy controls, while reaction time was normative. Conclusion: The exploratory findings of this study suggest once asymptomatic, a compensatory hemodynamic response may support the restoration of reaction time despite ongoing physiological recovery.

First draft submitted: 24 August 2020; Accepted for publication: 13 October 2020; Published online: 28 October 2020

Keywords: attention task • mild traumatic brain injury • neuroimaging • reaction time • recovery

Concussion is a transient, neurological dysfunction resulting from a direct or indirect blow to the head or body causing a rapid head acceleration–deceleration. This leads to alterations of mental status, cognitive function and, in some, loss of consciousness [1]. Concussion is often classified as a functional injury because it is not associated with abnormal structural findings on common neuroimaging techniques, such as standard magnetic resonance imaging or computed tomography scans [1]. Without objective measures, however, appropriate diagnostics and injury management for concussion are clinically challenging.

Following a head injury, as demonstrated in animal models, rapid changes in the neuronal membrane potential causes intense activation of the sodium-potassium pump, necessitating massive amounts of adenosine triphosphate, supported by glucose hypermetabolism [1,2]. Along with other neurochemical responses, the brain moves into a state of ‘energy crisis’ in which cerebral blood flow (CBF) is markedly decreased, further leading to an unbalanced energy supply and demand [2]. Previous work in humans has suggested several areas of decreased CBF following head injury in brain regions commonly associated with the attention network such as the frontal, parietal and temporal regions [3,4]. In addition, this work has revealed decreased task accuracy and slower reaction time when completing attention-based tasks relative to healthy controls [5].

To assess and manage concussions in humans, healthcare providers are encouraged to use a multifaceted approach, including cognitive tasks to measure reaction time, accuracy and working memory. A common sequela of
 concussive event. Despite a growing interest in the principal biologic effect of concussion, few studies have implemented neuroimaging techniques that can appropriately assess CBF post-concussion [4,8,9]. One such technique is functional near-infrared spectroscopy (fNIRS) [9], a cost-effective approach to objectively measure changes in the brain's hemodynamic response, a correlate of neural activity [10], during specific tasks such as neurocognitive assessments.

In the present study, we used fNIRS to assess the hemodynamic response within attention networks of the concussed human brain (1) shortly following injury and (2) after patients were behaviorally asymptomatic. In Experiment 1, we aimed to establish normative behavioral performance and cortical hemodynamic response in healthy adults performing an attentional temporal flanker task during fNIRS neuroimaging. In Experiment 2, we aimed to explore alterations of the hemodynamic response in concussed patients relative to a subgroup of controls from Experiment 1 using the same task. Concussed patients completed the temporal flanker task twice: once during the acute post-injury stage (T1) and again during the asymptomatic recovery stage (T2). Hemodynamic response patterns were compared between the three conditions: concussed T1, concussed T2 and controls. We hypothesized that at T1, concussed individuals would exhibit a reduced cortical hemodynamic response in frontal, parietal and temporal regions during the attention task, relative to controls. We further hypothesized that at T2, concussed individuals would continue to exhibit attenuated cortical hemoglobin responses during the attention task relative to matched controls. Collectively, this project aimed at uncovering neurophysiological alterations following concussion, which may not be completely mitigated after returning to normal symptom thresholds.

Experiment 1: HbO response during temporal flanker task in healthy adults

Methods

Participants

20 healthy adults (n = 20; 9 males, mean age: 26.30 ± 8.13 years, range: 18 to 51 years) were recruited from Southeastern Michigan, USA in 2018–2019. Participants were enrolled if they did not have a history of neurological or cognitive disorders, were not prescribed medications that may influence cognitive function and had not sustained a concussion within the previous 12 months [11–13].

Task description

We implemented a previously described [14] temporal flanker attention task consisting of a large letter at the center of a computer screen, followed by a second, smaller letter. In each trial, participants were asked to identify the second letter as quickly as possible. There were 9 s of fixation at the beginning of each run, with each trial lasting 2 s. Further, each of six 24 s blocks was preceded and followed by 18 s of fixation. Thus, the duration of a task ‘run’ was 279 s (4.65 min). Since there were three runs of the task, the total time was 837 s (13.95 min).

Procedure

Prior to enrollment, all participants provided written consent as approved by the university’s institutional review board. Upon consent, participants were fitted for the fNIRS cap and completed the temporal flanker task during fNIRS neuroimaging. This study was conducted in accordance of the Declaration of Helsinki.

fNIRS neuroimaging & data analysis

We used CW6 fNIRS system (TechEn Inc., MA, USA) with 690 and 830 nm wavelengths. The probe configuration consisted of eight light sources and ten light detectors, yielding 24 channels per hemisphere. The data channels covered areas typically engaged in attention processing (frontal, parietal and temporal regions; Figure 1) [3,15]. Data analyses [16] were conducted using NIRS Brain AnalyzIR Toolbox [17], with custom scripts written in MATLAB (MathWorks, MA, USA). Optical density data was converted into HbR/HbO signal for individual data analyses. For the purpose of this study, we focused on the hemoglobin oxygenation (HbO) data as it is a more common index of hemodynamic activity measured with fNIRS, in part, because the 830 nm wavelength that targets HbO is less susceptible to light diffusion than 690 nm [18]. Quantitative analysis indicated that HbR signal changes contributed 16–22%, while HbO signal changes contributed 73–79% to the measured total cortical hemoglobin
Concentration changes by fNIRS [18]. Individual-level data were analyzed by fitting a general linear model (GLM) with pre-whitening and robust least square solution [19]. A linear mixed effects model was then used to estimate task-related brain activations and brain–behavior relations at the group level using task > resting baseline contrasts. The data were thresholded at p < 0.05 uncorrected, due to the exploratory nature of the study.

**Results**

*Task performance*

Across all sessions, participants’ accuracy and reaction time were consistent with previously reported findings for healthy adults (accuracy: 97.791 ± 1.733%; reaction time: 0.533 ± 0.103 s) [20].

*Hemodynamic response*

Participants’ response patterns for the task relative to rest contrast were consistent with previously reported functional magnetic resonance imaging (fMRI) findings for attention [21]. These response patterns included significant activations in 13 channels, spanning the bilateral prefrontal cortex, bilateral parietal and left occipito-temporal regions (Table 1; Figure 1). Brain-behavior relation analyses revealed that participants with relatively fast overall reaction times showed significantly less activity in 18 channels, including those over bilateral prefrontal, left superior temporal and bilateral parietal regions (Table 1; Figure 2).

**Experiment 2: Effect of concussion on HbO response during temporal flanker task**

*Methods*

Concussed participants were recruited from a local emergency department in Southeastern Michigan, USA, and tested in a laboratory setting within 72 h from initial injury (n = 8; 2 males mean age: 18.38 ± 1.41 years, range: 16 to 21 years, mean concussion history = 0.50 ± 1.07, time from injury = 42.00 ± 16.97 h; symptom severity...
Table 1. Experiment 1 task versus rest cortical activation all control participants.

| Group/contrast | Regions of interest                     | β     | β-SE  | t     | p      | q      | Power (1-β) |
|----------------|----------------------------------------|-------|-------|-------|--------|--------|-------------|
| **Left hemisphere** |                                        |       |       |       |        |        |             |
| Task vs rest    | All controls                           | 7.809 | 2.802 | 2.787 | 0.006  | 0.060  | 0.869       |
|                 | IFG Pars Opercularis (DLPFC)           |       |       |       |        |        |             |
|                 | Middle Occipital Gyrus                 | 8.085 | 2.335 | 3.462 | 0.001  | 0.018  | 0.963       |
|                 | Superior Occipital Gyrus               | 4.657 | 2.286 | 2.037 | 0.044  | 0.234  | 0.647       |
|                 | MFG, DLPFC                             | 4.729 | 1.934 | 2.445 | 0.016  | 0.128  | 0.783       |
|                 | Premotor Cortex                        | 6.480 | 2.249 | 2.881 | 0.005  | 0.051  | 0.888       |
|                 | Primary Motor Cortex                   | 5.677 | 2.561 | 2.217 | 0.029  | 0.166  | 0.711       |
|                 | Primary Sensory Cortex                 | 6.793 | 2.821 | 2.408 | 0.018  | 0.130  | 0.772       |
| **Right Hemisphere** |                                        |       |       |       |        |        |             |
|                 | IFG Pars Triangular (DLPFC)            | 4.220 | 1.913 | 2.205 | 0.029  | 0.166  | 0.707       |
|                 | MFG, DLPFC                             | 4.540 | 2.264 | 2.006 | 0.047  | 0.239  | 0.635       |
|                 | Premotor Cortex                        | 8.057 | 2.742 | 2.939 | 0.004  | 0.050  | 0.898       |
|                 | Primary Motor Cortex                   | 10.089| 2.630 | 3.836 | <0.001 | 0.010  | 0.984       |
|                 | Primary Sensory Cortex                 | 6.940 | 2.371 | 2.927 | 0.004  | 0.050  | 0.896       |
|                 | Supramarginal Gyrus                    | 6.246 | 2.395 | 2.608 | 0.010  | 0.090  | 0.828       |
| **Predicted by RT** |                                        |       |       |       |        |        |             |
|                 | All controls                           | 45.948| 10.618| 4.327 | <0.001 | 0.001  | 0.996       |
|                 | IFG Pars Opercularis (DLPFC)           |       |       |       |        |        |             |
|                 | MFG, DLPFC                             | 34.352| 10.067| 3.412 | 0.001  | 0.012  | 0.959       |
|                 | IFG Pars Triangular (DLPFC)            | 62.741| 13.645| 4.598 | <0.001 | 0.000  | 0.998       |
|                 | IFG Pars Opercularis (DLPFC)           | 41.772| 13.396| 3.118 | 0.002  | 0.028  | 0.926       |
|                 | Anterior STG                           | 55.958| 11.232| 4.982 | <0.001 | 0.000  | 0.999       |
|                 | STG                                    | 52.214| 11.175| 4.672 | <0.001 | 0.000  | 0.998       |
|                 | Sensory Motor Cortex                   | 25.755| 12.038| 2.139 | 0.035  | 0.195  | 0.684       |
|                 | Posterior STG/MTG                      | 30.959| 11.504| 2.691 | 0.008  | 0.072  | 0.848       |
|                 | Supramarginal Gyrus                    | 23.078| 11.144| 2.071 | 0.041  | 0.217  | 0.660       |
|                 | Angular Gyrus                          | 29.659| 12.013| 2.469 | 0.015  | 0.101  | 0.790       |
|                 | MFG, DLPFC                             | 24.926| 9.772 | 2.551 | 0.012  | 0.089  | 0.813       |
|                 | MFG, DLPFC                             | 31.069| 10.913| 2.847 | 0.005  | 0.056  | 0.881       |
| **Right Hemisphere** |                                        |       |       |       |        |        |             |
|                 | IFG Pars Triangular (DLPFC)            | 31.501| 13.911| 2.264 | 0.025  | 0.153  | 0.727       |
|                 | IFG Pars Opercularis (DLPFC)           | 48.021| 12.762| 3.763 | <0.001 | 0.004  | 0.981       |
|                 | Supramarginal Gyrus                    | 45.810| 11.947| 3.834 | <0.001 | 0.004  | 0.984       |
|                 | Angular Gyrus                          | 23.713| 11.868| 1.998 | 0.048  | 0.223  | 0.633       |
|                 | Angular, Superior Occipital Gyri       | 26.776| 13.442| 1.992 | 0.049  | 0.223  | 0.630       |
|                 | MFG, DLPFC                             | 26.463| 9.723 | 2.722 | 0.008  | 0.072  | 0.855       |
|                 | Primary Motor Cortex                   | 23.793| 11.728| 2.029 | 0.045  | 0.223  | 0.644       |

Note: Data were analyzed by fitting a general linear model with pre-whitening and robust least square solution. A linear mixed effects model was then used to estimate task-related brain activations and brain–behavior relations at the group level.

DLPFC: Dorsolateral prefrontal cortex; IFG: Inferior frontal gyrus; MFG: Medial frontal gyrus; MTG: Medial temporal gyrus; STG: Superior temporal gyrus.

Control participants

A subset of eight healthy participants from Experiment 1 was matched to the concussed group on age and gender (n = 8; 2 males, mean age: 20.87 ± 1.86, range: 18 to 23 years).
Cortical hemodynamic response after concussion  Short Communication

Procedure & data analysis
Prior to enrollment, all participants provided written consent, or assent as appropriate, as required by the university’s institutional review board. Participants were then fitted for the fNIRS cap and completed the same neuroimaging task as in Experiment 1. Concussed participants completed the same experimental procedure at Time 1 (T1; symptom severity count = 16.13 ± 4.88, symptom severity score = 49.13 ± 31.89) and at Time 2 (T2; symptom severity count = 2.75 ± 3.20, symptom severity score = 2.88 ± 3.31) visits. After T1, participants’ symptoms were remotely monitored using the Sport Concussion Assessment Tool (third edition) symptom survey.[23]. Once asymptomatic, concussed participants were scheduled for a follow-up testing session where they completed a second measurement (T2; time between T1 and T2 = 25.50 ± 11.82 days). The control group only completed one testing session (Experiment 1) in order to establish normative data. Paired-samples t-tests were used to identify differences in the behavioral data between T1 and T2. Independent samples t-tests were used to explore differences between the concussed and control groups, with an alpha of 0.05 established a priori.

Results
Task performance
Accuracy
There were no conditional differences in accuracy. First, there were no differences within the concussed group between T1 and T2 (T1: 95.472 ± 4.210%; T2: 97.780 ± 0.961%; t(7) = 2.365; p = 0.189). Second, there were no differences between the concussed group at T1 and controls (controls: 97.001 ± 2.035%; t(7) = 2.145; p = 0.370) or between the concussed group at T2 and controls (t(7) = 2.145; p = 0.345).

Reaction time
There was one conditional difference in overall reaction time. Specifically, concussed participants were significantly slower at T1 than at T2 (T1: 0.595 ± 0.139 s; T2: 0.513 ± 0.106 s; t(7) = 2.365; p = 0.010); however, they performed similarly to controls (controls: 0.523 ± 0.087 s; t(7) = 2.145; p = 0.236). At T2, there was no difference between the concussed and control groups (t(7) = 2.145; p = 0.839).

Hemodynamic response
Concussed group: T1 versus T2
At T1, performing the temporal flanker task (vs. resting) was associated with significant activation in four channels within right prefrontal and bilateral parietal regions (Table 2; Figure 2). At T2, the same comparison was associated with significant activation in 23 channels throughout the attention network. A direct comparison between T1 and T2 showed greater activations in 22 channels at T2 compared with T1 (Table 2; Figure 2), covering bilateral frontal, parietal and temporal regions.

Concussed group versus control group
Controls showed greater activations in three bilateral frontal and three parietal channels than the concussed group at T1, while their activations were lower in one right frontal and one left parietal channel (Table 3; Figure 2). Contrary to our hypothesis at T2, the concussed group exhibited stronger activations than the control group in 12 channels within bilateral frontal, temporal and right parietal regions (Table 3; Figure 2).

Discussion
We compared attentional network activity in healthy adults to that in concussed patients. In Experiment 1, we found that the temporal flanker task engages bilateral prefrontal, bilateral parietal and left occipito-temporal regions in healthy adults. In Experiment 2, shortly after the head impact, concussed participants activated fewer channels and responded more slowly than controls (the former outcome may reflect post-injury vasoconstriction during the acute recovery phase [2]). We also found that after concussed patients became asymptomatic, they exhibited several areas of hyperactivation relative to the controls from Experiment 1 and responded more quickly than they did before. These exploratory findings suggest that after concussed patients become asymptomatic, a compensatory boost of the hemodynamic response supports their ability to respond quickly despite ongoing physiological recovery. This is noteworthy, as current clinical assessments are unable to reliably assess underlying recovery and therefore may put athletes at risk for prolonged recovery or secondary injury if they return to sport prematurely.
| Group/contrast       | Regions of interest                          | β     | β-SE  | t     | p     | q     | Power (1-β) |
|---------------------|----------------------------------------------|-------|-------|-------|-------|-------|-------------|
|                     | Left hemisphere                              |       |       |       |       |       |             |
| T1                  | Supramarginal/angular gyri                   | 18.369| 5.026 | 3.655 | <0.001| 0.018 | 0.976       |
|                     | MFG, DLPFC                                    | 11.063| 3.175 | 3.485 | 0.001 | 0.021 | 0.965       |
|                     | Primary sensory cortex                        | 9.604 | 4.338 | 2.214 | 0.029 | 0.263 | 0.711       |
|                     | Supramarginal gyrus                           | 8.805 | 4.270 | 2.062 | 0.041 | 0.263 | 0.657       |
|                     | Right hemisphere                              |       |       |       |       |       |             |
|                     | MFG, DLPFC                                    | 11.132| 3.531 | 3.153 | 0.002 | 0.019 | 0.932       |
|                     | Primary motor cortex                          | 6.594 | 3.331 | 1.980 | 0.050 | 0.184 | 0.627       |
|                     | Supramarginal gyrus                           | 9.191 | 4.113 | 2.412 | 0.017 | 0.087 | 0.774       |
|                     | Sensory motor cortex                          | 9.195 | 3.389 | 2.713 | 0.008 | 0.048 | 0.854       |
|                     | MTG posterior                                  | 9.682 | 3.199 | 3.026 | 0.003 | 0.024 | 0.914       |
|                     | Superior occipital gyrus                      | 10.705| 4.237 | 2.527 | 0.013 | 0.071 | 0.807       |
|                     | MFG, DLPFC                                    | 11.883| 3.869 | 3.071 | 0.003 | 0.247 | 0.920       |
|                     | Premotor cortex                               | 9.398 | 3.462 | 2.715 | 0.007 | 0.048 | 0.854       |
|                     | Primary motor cortex                          | 9.693 | 3.632 | 2.669 | 0.009 | 0.051 | 0.844       |
|                     | Supramarginal/angular gyri                    | 15.454| 3.971 | 3.891 | <0.001| 0.003 | 0.986       |
|                     | Right hemisphere                              |       |       |       |       |       |             |
|                     | MFG, DLPFC                                    | 7.121 | 2.790 | 2.553 | 0.012 | 0.250 | 0.814       |
|                     | Premotor cortex                               | 7.569 | 2.941 | 2.573 | 0.011 | 0.250 | 0.820       |
|                     | Primary motor cortex                          | 8.324 | 3.477 | 2.394 | 0.018 | 0.250 | 0.769       |
|                     | Supramarginal gyrus                           | 7.024 | 3.295 | 2.132 | 0.035 | 0.306 | 0.682       |

Note. Similar to Experiment 1, data were analyzed by fitting a general linear model with pre-whitening and robust least square solution. A linear mixed-effects model was then used to estimate task-related brain activations and brain–behavior relations at the group level.

DLPFC: Dorsolateral prefrontal cortex; IFG: Inferior frontal gyrus; MFG: Medial frontal gyrus; MTG: Medial temporal gyrus; STG: Superior temporal gyrus.

Brain–behavior relation analyses involving the healthy controls from Experiment 1 revealed that those who performed the flanker task more quickly exhibited less activity in 18 channels. In other words, as the healthy control participants’ task proficiency increased (i.e., faster reaction time), there was less activity observed in the bilateral prefrontal, left superior temporal and bilateral parietal regions, which has also been demonstrated in prior research using an analogous flanker task [24]. In Experiment 2, concussed individuals at T1 demonstrated less brain
activity and slower reaction times compared with the controls due to injury-related neurocognitive impairments. These findings are consistent with previous work using fMRI in concussed subjects, indicating areas of low activation of the attention network when assessed with a similar cognitive task [25].
By T2, concussed individuals performed similarly to controls, yet demonstrated activation increases across the attentional network. These results complement the work of Hammeke et al., which suggests injured athletes demonstrated hyperactivation (injured > controls) in the same networks at 7 weeks [25]. Despite exhibiting the same level of behavioral performance as controls, asymptomatic concussed patients may require additional resource allocation (i.e., from the attentional network) in order to perform similarly.

More broadly, the use of neuroimaging in conjunction with clinical behavioral assessments may assist clinicians who wish to determine whether a patient has neurologically recovered following a concussion. At present, many clinicians use subjective symptom inventories to determine whether they should initiate the return to sport protocol [22]. By using non-invasive techniques, such as fNIRS, clinicians may be able to determine whether a patient’s hemodynamic responses return to normative levels, which may index a more stable measure of injury recovery. Indeed, unlike fMRI, neuroimaging techniques such as fNIRS provide a cost-effective way to assess the hemodynamic response in patients with a suspected concussion across a wide variety of settings (e.g., an emergency department or sports medicine facility).

Although our findings are novel, we are aware of several limitations. Due to the SARS-CoV-2 outbreak, data collection was terminated prematurely, which led to the concussed and control groups to differ in terms of mean age. Further, concussed participants who sought treatment from the emergency department may have suffered from a more severe concussion than those who typically obtain care from a sports medicine or urgent care clinics (time to recovery for concussed participants = 25.50 ± 11.82 days). The authors also recognize the limited statistical power associated with the exploratory nature and limited sample size of Experiment 2. Lastly, although there is conflicting evidence regarding long-term effects of cognitive deficits after head injury [26], controls were enrolled in the present study only if they did not have a concussion in the previous 12 months. To control for this potential confound, future studies should consider only enrolling control participants without any concussion history. While we regard the study as exploratory given these limitations, the findings are nevertheless supported by previous findings of reduced attentional capabilities and cortical activity in the attention network following concussion [3,4] as well as those suggesting that neural alterations persist after individuals become asymptomatic [25].

In conclusion, the present results suggest pairing fNIRS with a temporal flanker task to assess attentional deficits may yield more nuanced indicators of concussion patients’ recovery trajectories than standard behavioral assessments. The use of neuroimaging techniques to assess changes in CBF throughout concussion recovery warrants further investigation as a diagnostic and recovery tool.

Future perspective
Researchers are eagerly pursuing the development a clinical tool to objectively assess brain structure and function after concussion. Although advanced imaging techniques, such as diffusion tensor imaging and single-photon emission computer tomography, have shown promise, these techniques are costly. Further, they cannot be used to conduct concussion screening on the sidelines, in athletic training facilities or rehabilitation clinics. In contrast, fNIRS is both cost-effective and portable, with recent models featuring wireless data transfer. Therefore, the use of such devices to assess concussion may one day complement current neurocognitive assessments and symptom inventories.

Executive summary
- Current concussion assessment protocols rely on clinical functioning and thus may not be sensitive to underlying neural deficits.
- Pairing neuroimaging techniques such as functional near-infrared spectroscopy with clinical behavioral assessments may further assist clinicians when determining if a patient is neurologically recovered.
- The present results indicate that shortly after their injury, concussed patients respond more slowly and exhibit less cortical activation than gender- and age-matched controls.
- In contrast, once concussed patients become asymptomatic, they respond just as quickly as controls and exhibit hyperactivity in bilateral frontal, temporal and right parietal regions.
- These findings suggest that after concussed patients become asymptomatic, a compensatory boost of the hemodynamic response supports their ability to respond quickly, despite ongoing physiological recovery.
Cortical hemodynamic response after concussion

Short Communication

Financial & competing interests disclosure

University of Michigan Exercise & Sport Science Initiative. The authors have no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript apart from those disclosed.

No writing assistance was utilized in the production of this manuscript.

Ethical conduct of research

The authors state that they have obtained appropriate institutional review board approval or have followed the principles outlined in the Declaration of Helsinki for all human or animal experimental investigations. In addition, for investigations involving human subjects, informed consent has been obtained from the participants involved.

Informed consent disclosure

The authors state that they have obtained verbal and written informed consent from the patient/patients for the inclusion of their medical and treatment history within this case report.

Open access

This work is licensed under the Creative Commons Attribution 4.0 License. To view a copy of this license, visit http://creativecommons.org/licenses/by/4.0/

References

Papers of special note have been highlighted as: • of interest; •• of considerable interest

1 Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery 75(Suppl. 4), S24–33 (2014).
2 Giza CC, Hovda DA. The neurometabolic cascade of concussion. J. Athl. Train. 36(3), 228–235 (2001).
• Describes the pathophysiology of concussion and its relation to disturbances in cerebral blood flow.
3 Fan J, McCandliss BD, Fossella J, Flombaum JI, Posner MI. The activation of attentional networks. NeuroImage 26(2), 471–479 (2005).
4 Wang Y, Nelson LD, Laroche AA et al. Cerebral blood flow alterations in acute sport-related concussion. J. Neurotrauma 33(13), 1227–1236 (2016).
5 Van Donkelaar P, Langan J, Rodriguez E et al. Attentional deficits in concussion. Brain Inj. 19(12), 1031–1039 (2005).
6 McCrea M, Prichep L, Powell MR, Chabot R, Barr WB. Acute effects and recovery after sport-related concussion. J. Head Trauma Rehabil. 25(4), 283–292 (2010).
7 McCrea M, Iverson GL, Mcallister TW et al. An integrated review of recovery after mild traumatic brain injury (MTBI): implications for clinical management. Clin. Neuropsychol. 23(8), 1368–1390 (2009).
8 Helmich I, Saluja RS, Lausberg H et al. Persistent postconcussive symptoms are accompanied by decreased functional brain oxygenation. J. Neuropsychiatry Clin. Neurosci. 27(4), 287–298 (2015).
9 Kontos AP, Huppert TJ, Beluk NH et al. Brain activation during neurocognitive testing using functional near-infrared spectroscopy in patients following concussion compared to healthy controls. Brain Imaging Behav. 8(4), 621–634 (2014).
10 Wray S, Cope M, Delpy DT, Wyatt JS, Reynolds EO. Characterization of the near infrared absorption spectra of cytochrome aa3 and haemoglobin for the non-invasive monitoring of cerebral oxygenation. Biochim. Biophys. Acta 933(1), 184–192 (1988).
• Identifies specific optode locations typically associated with attentional tasks within the frontal, parietal and temporal regions of the brain.
11 Tellier A, Marshall SC, Wilson KG, Smith A, Perugini M, Stiell IG. The heterogeneity of mild traumatic brain injury: where do we stand? Brain Inj. 23(11), 879–887 (2009).
12 Iverson GL, Lange RT, Waljas M et al. Outcome from complicated versus uncomplicated mild traumatic brain injury. Rehavisil. Res. Pract. 2012, 415740 (2012).
13 Slobounov SM, Zhang K, Pennell D, Ray W, Johnson B, Sebastianelli W. Functional abnormalities in normally appearing athletes following mild traumatic brain injury: a functional MRI study. Exp. Brain Res. 202(2), 341–354 (2010).
14 Hazeltine E, Lightman E, Schwab H, Schumacher EH. The boundaries of sequential modulations: evidence for set-level control. J. Exp. Psychol. Hum. Percept. Perform. 37(6), 1898–1914 (2011).
15 Zawadowska Vel Grajewska B, Sim E-J, Hoenig K, Herrnberger B, Kiefer M. Mechanisms underlying flexible adaptation of cognitive control: behavioral and neuroimaging evidence in a flanker task. Brain Res. 1421, 52–65 (2011).
16 Arredondo M, Satterfield T, Riobóo A, Gelman S, Kovelman I. Bilingual effects on lexical selection: a neurodevelopmental perspective. Brain Lang. 195, 104640 (2019).
17 Santosa H, Zhai X, Fishburn F, Huppert T. The NIRS Brain AnalyziR Toolbox. Algorithms 11(5), 73 (2018).
18 Gagnon L, Yucel MA, Dehaes M et al. Quantification of the cortical contribution to the NIRS signal over the motor cortex using concurrent NIRS-fMRI measurements. *NeuroImage* 59(4), 3933–3940 (2012).

19 Barker JW, Aarabi A, Huppert TJ. Autoregressive model based algorithm for correcting motion and serially correlated errors in fNIRS. *Biomed. Opt. Express* 4(8), 1366–1379 (2013).

20 Weissman DH, Egner T, Hawks Z, Link J. The congruency sequence effect emerges when the distracter precedes the target. *Acta Psychol. (Amst)* 156, 8–21 (2015).

**Establishes typical ranges for the accuracy and reaction time during the computerized flanker task in healthy adults.**

21 Prado J, Carp J, Weissman DH. Variations of response time in a selective attention task are linked to variations of functional connectivity in the attentional network. *NeuroImage* 54(1), 541–549 (2011).

**Indicates response patterns in during a behavioral task highlight specific areas of the attentional network such as the prefrontal, parietal and occipito-temporal regions.**

22 Echemendia RJ, Meeuwisse W, McCrory P et al. The sport concussion assessment tool 5th edition (SCAT5): background and rationale. *Br. J. Sports Med.* 51(11), 848–850 (2017).

23 McCrory P, Meeuwisse WH, Aubry M et al. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport, Zurich, November 2012. *J. Athl. Train.* 48(4), 554–575 (2013).

24 Casey BJ, Thomas KM, Welsh TF et al. Dissociation of response conflict, attentional selection, and expectancy with functional magnetic resonance imaging. *Proc. Natl Acad. Sci. USA* 97(15), 8728–8733 (2000).

**Identifies a positive correlation between cortical activation and reaction time using a similar flanker task.**

25 Hammeke TA, McCrea M, Coats SM et al. Acute and subacute changes in neural activation during the recovery from sport-related concussion. *J. Int. Neuropsychol. Soc.* 19(8), 863–872 (2013).

**Suggests athletes who sustained a sport-related concussion demonstrated areas of hyperactivation within the attentional network at seven weeks post-injury during a similar cognitive task.**

26 McInnes K, Friesen CL, Mackenzie DE, Westwood DA, Boe SG. Mild traumatic brain injury (mTBI) and chronic cognitive impairment: a scoping review. *PLoS One* 12(4), e0174847 (2017).