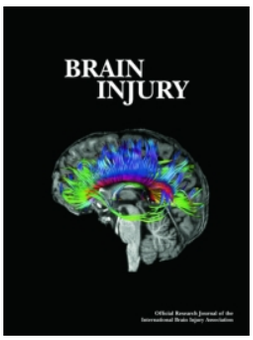

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BRAIN INJURY
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The effect of mild traumatic brain injury on the visual processing of global form and motion

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ABSTRACT

Cortical visual processing involves the ventral stream (form perception) and the dorsal stream (motion perception). We assessed whether mild traumatic brain injury (TBI) differentially affects these two streams. Eleven adults with mild TBI (28 ± 9 yrs, 17 ± 5 months post injury) and 25 controls (25 ± 5 yrs) participated. Participants completed tests of global processing involving Glass patterns (form) and random dot kinematograms (motion), measurement of contrast thresholds for motion direction discrimination, a comprehensive vision screening and the Post-Concussion Symptom Inventory (PCSI). Our results showed that the mild TBI group had significantly higher (worse) global form (mean ± SD: TBI 25 ± 6%, control 21 ± 5%) and motion (TBI 14 ± 7%, control 11 ± 3%) coherence thresholds than controls. The magnitude of the mild TBI group deficit did not differ between the two tasks. Contrast thresholds for motion direction discrimination did not differ between the groups, but were positively correlated with PCSI score (r^2 = 0.51. p = 0.01) in the mild TBI group. The mild TBI group had worse outcomes than controls for all clinical measurements of vision except distance visual acuity. In conclusion, mild TBI affects processing in both the dorsal and ventral cortical processing streams equally. In addition, spatiotemporal contrast sensitivity may be related to the symptoms of mild TBI.

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Mild TBI; ventral stream; dorsal stream; form perception; motion perception

Introduction

Traumatic brain injury (TBI) is one of the most common neurological disorders and a major cause of disability (1). Almost 90% of TBIs are considered to be mild, whereby symptoms are present, but no brain abnormalities can be detected by diagnostic imaging (2–4). Mild TBI significantly impairs activities of daily living, such as reading, driving, and moving (5,6). Common symptoms include nausea, dizziness, headaches, difficulty with balance, confusion, disorientation, and light sensitivity (7). In addition, vision problems such as strabismus, photosensitivity, visual field defects, and anomalies of accommodation and vergence, have been documented in individuals with mild TBI (8,9). Typically, these vision problems cannot be explained by ocular pathology and are attributed to impaired brain neurophysiology (10,11).

Brain trauma can cause both focal and diffuse injury (12). Focal injuries are most often caused by a direct brain impact that is sufficient to cause intracranial bleeding and subdural hematomas (12). Focal injuries typically result in moderate or severe TBI. In contrast, diffuse injury, commonly referred to as Diffuse Axonal Injury (DAI), is primarily caused by sheering damage to axons in brain areas that are subjected to acceleration/deceleration forces (13). DAI has been associated with the interruption of neuron axonal transport systems that move elements such as mitochondria and proteins between the cell body and its axon (14,15). This disruption leads to neuronal swelling and secondary physiological changes such as elevated intracranial pressure and cerebral oedema. DAI can cause mild, moderate or severe TBI and manifests acutely as a loss of consciousness or confusion. Chronic effects of DAI include Post-Concussion Syndrome PCS (16).

Although clinically observable brain injuries are absent, mild TBI has been linked to a number of physiological changes within the brain. These include a neurometabolic cascade starting immediately after the biomechanical injury (17). This cascade alters cellular metabolism by increasing the release of potassium (K+) and the absorption of toxic calcium ions (Ca2+) by affected neurons (18). Changes in brain connectivity that are correlated with symptomatology have also been observed in Mild TBI (14). For example, using functional magnetic resonance imaging, Palacios et al. observed reduced functional connectivity within cognition networks in mild TBI. The extent of reduced functional connectivity was significantly associated with neuropsychological test performance (19).

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The diagnosis of mild TBI is usually based on a history of head trauma resulting in lost or decreased consciousness and a Glasgow Coma Score (GCS) score (14,16). This makes the diagnosis of mild TBI challenging since it relies heavily on subjective patient report. Predicting recovery from mild TBI is also challenging. A study of high-school and student athletes indicted that up to 90% of mild TBI symptoms resolve after 2 weeks without medical intervention (20). However, recovery from oculomotor dysfunctions associated with mild TBI may take up to 3 months (21). Furthermore, a subset of individuals with mild TBI may continue to experience symptoms 1-year post injury (22–24). The cause of this variability in recovery from mild TBI is unknown.

The duel stream theory of vision proposes that higher-level cortical processing of visual information occurs in two parallel, but interconnected streams; the dorsal stream and the ventral stream (25–28). The dorsal stream receives input from the magnocellular layers of the lateral geniculate nucleus (LGN), includes motion sensitive areas such as V3A, MT and MST and projects to the posterior parietal lobe (29). The ventral stream receives input from the parvocellular layers of the LGN, includes form sensitive areas such as V4 and projects to the inferior temporal lobe (25). Functionally, the dorsal stream supports visuo-motor control whereas the ventral stream subserves object recognition (28,30).

Ventral and dorsal stream function can be measured psychophysically (31,32). This approach is based on the linking assumption that psychophysical tasks can target brain areas such as V4 in the ventral stream and MT in the dorsal stream. These areas integrate local signals from V1 and V2 into coherent, global representations of form (V4) or motion (MT) (33). Global form tasks designed to measure ventral stream function require the combination of local form cues into a coherent shape or pattern (32,34). Glass patterns, constructed from multiple pairs of dots that can be configured into a coherent pattern, are a common global form stimulus. Similarly, global motion tasks that are used to measure dorsal stream function involve the integration of multiple local motion signals into a coherent motion percept (31,35). Global motion tasks often utilize stimuli constructed from groups of moving dots called random dot kinematograms (RDKs), whereby a sub-set of "signal" dots move coherently in a common direction and the remaining "noise" dots move in random directions. The observer's task is to identify the direction of coherent motion and the signal to noise ratio in the stimulus is manipulated to determine a motion coherence threshold (31,35,36). Measurements of basic visual functions such as spatial or temporal contrast sensitivity can be used to assess whether deficits in global processing tasks are due to abnormal integration of local signals or impairments in the early-stage (pre-integration) processing of visual information (36,37).

The dorsal stream vulnerability hypothesis posits that the dorsal stream has a greater susceptibility to damage than the ventral stream (38). Evidence for this hypothesis comes primarily from psychophysical studies of children with neurodevelopmental disorders. For example, Williams's syndrome (a genetic disorder associated with cognitive and visuo-motor deficits) appears to be associated with impaired global motion but normal global form perception (39). Similar observations have been reported for children with autistic spectrum disorder or dyslexia (40,41). One possible explanation for dorsal stream vulnerability is that the magnocellular pathway that projects to the dorsal stream has substantially fewer cells and therefore less redundancy than the parvocellular pathway that projects to the ventral stream (26,42). This may also make the dorsal stream more vulnerable to the effects of mild TBI, even in adult patients.

Two previous studies have investigated the impact of mild TBI on global motion perception. Brosseau-Lachaine et al. (10), reported significantly elevated (poorer) motion coherence thresholds for radially moving RDKs in children with mild TBI compared to controls. This deficit could not easily be explained by impairments affecting the early-stage processing of motion signals because contrast sensitivity for direction discrimination of moving gratings was equivalent between the two groups. However, deficits in early-stage magnocellular processing have previously been reported in individuals with mild TBI (43) suggesting that part of the global motion processing deficit observed by Brosseau-Lachaine et al. could be due to abnormalities within the LGN or V1. Patel et al. (44), also observed a small but statistically significant elevation in motion coherence thresholds in a group of adults with mild TBI compared to controls. Lower level visual processing of motion was not assessed by Patel et al. Together these two studies of global motion processing suggest that mild TBI impairs dorsal stream function. To our knowledge, no previous studies have directly assessed ventral stream function in patients with mild TBI. Therefore it is unclear whether deficits are confined to the dorsal stream, as might be predicted by the dorsal stream vulnerability hypothesis, or whether mild TBI affects both the dorsal and ventral streams equally.

In the present study, the function of both the dorsal and ventral processing streams in individuals with mild TBI and controls was assessed psychophysically using measures of global form and motion perception. Based on the concept of dorsal stream vulnerability, our hypothesis was that mild TBI would impair global motion more than global form perception. In addition, we measured contrast sensitivity for static stimuli and for motion direction discrimination to assess whether lower-level processing deficits might account for any global processing abnormalities. Since patients with mild TBI exhibit many different vision problems, a range of basic visual functions were also assessed. Mild TBI symptoms were quantified using the post-concussion symptom inventory.

Methods

Participants

Eleven participants with mild TBI (mean age 28 ± 9 yrs, 17 ± 5 months post injury, 7 female) and 25 age-matched controls with no history of mild TBI (25 ± 5 yrs, 14 female) took part in this study. Participants were recruited via a database of patients with TBI within the School of Optometry and Vision Science, University of Waterloo and through flyers posted at the School of Optometry and Vision Science and The Guelph Vision Therapy Center. Therefore, participants with mild TBI were sampled from a population with vision-related symptoms. Inclusion criteria for controls were: 19 to 40 years of age, no self-reported history of mild

TBI, able to provide informed written consent. Inclusion criteria for participants with mild TBI were: self-reported medical diagnosis of mild TBI (either a single or multiple concussions), at least 3 months post-injury, less than 20 minutes of lost consciousness immediately following the injury, less than 24 hours of post-traumatic amnesia. Exclusion criteria for all participants were: self-reported neuropsychiatric condition, binocular visual acuity worse than 6/12, and a self-reported history of seizures or any other neurological disorder. Participants used their habitual vision correction during testing.

Vision assessments

Global form perception

Global form perception was tested psychophysically using a form detection task based on Glass patterns (Figure 1). The task was used to measure a form coherence threshold. Stimuli were generated by an Apple Macintosh computer using Psykinematix software and presented on 27-inch iMac display (1024 x 768 pixels resolution, 60 cm viewing distance). The stimuli were composed of bright pairs of dots presented on a grey background (100 cd/m^2) and were presented for 1 second within a rectangular aperture (10° diameter). There were two populations of dot pairs: "signal pairs" which were arranged to form concentric- or cross-shaped radial Glass patterns constructed in a manner similar to that described by Wilson and Wilkinson (1998), and "noise pairs" which were oriented randomly within the display aperture (Figure 1). Form coherence was modulated by varying the ratio of signal to noise dots in the stimulus.

Procedure

The global form task required participants to discriminate between the two different Glass patterns (concentric or cross) using button presses within a two-alternative-forced-choice procedure. Before starting the main task, participants completed a familiarization session where the stimuli were presented at 100% coherence. This was followed by blocks of 4 stimuli presented at 80, 70, and 60% coherence. Participants had to achieve an accuracy of at least 75% correct (three correct responses on four trials with the same level of coherence) on each coherence level prior to starting the main task. The main task involved a 2-down-1-up adaptive staircase procedure that started at 100% coherence (50% proportional step size before the first reversal, 25% thereafter). The staircase was terminated after 12 reversals and the average of the last 11 reversals was calculated to estimate the form coherence threshold. The staircase was repeated twice and the average threshold was used for data analysis.

Global motion perception

Global motion perception was tested psychophysically by using Random dot kinematograms (RDKs) to measure motion coherence thresholds (Figure 2). RDKs were generated and presented using the same apparatus as the form coherence task. RDKs consisted of 100 bright dots presented at 100% contrast on a grey background (100 cd/m^2). Dots had a diameter of 0.24° and a density of 1.27 dot/deg^2. The RDKs were presented for 1 second at a viewing distance of 60 cm within a circular aperture (10° diameter). Dots were displaced every 17 ms in order to achieve a speed of 6°/second. The parameters of the RDKs were chosen based on a previous study that assessed global motion perception in children (36). Signal dots moved up or down. Noise dots moved in random directions. Motion coherence was controlled by varying the proportion of signal to noise dots.

Procedure

Participants identified the direction of the signal dots (up or down) with a button press. Before starting the main task, participants completed a familiarization session during which blocks of 4 RDKs were presented with block-by-block coherence levels of 100, 80, 70 and 60%. An accuracy of 75% correct (three correct responses on four trials with the same level of coherence) for each coherence level was required before moving on to the main task which employed

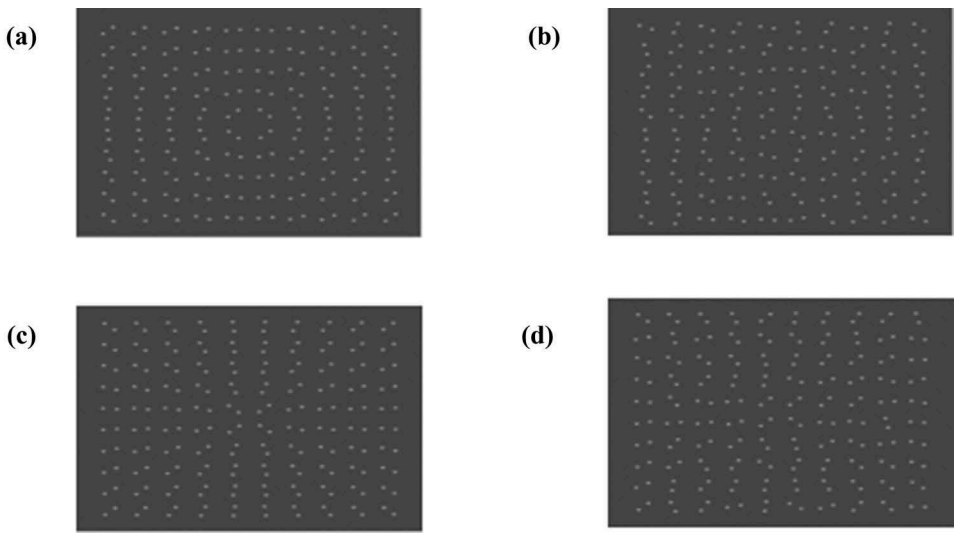

Figure 1. An example of the global form task stimulus. (a) A concentric pattern at 100% coherence, (b) a concentric pattern at 50% coherence, (c) a radial pattern at 100% coherence, (d) a radial pattern at 50% coherence.

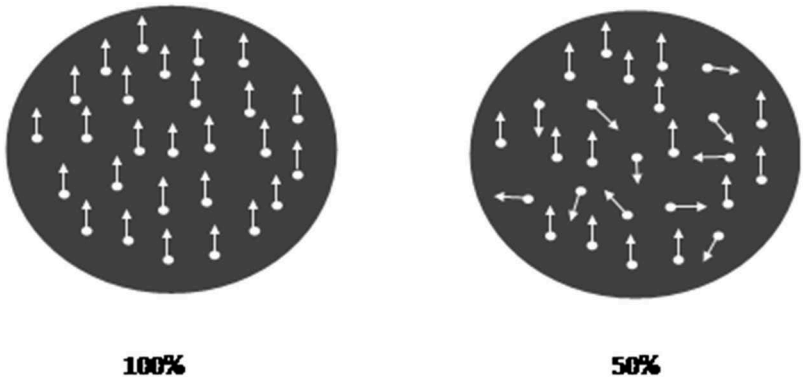

Figure 2. A schematic of the random dot kinematogram stimulus with coherence levels of 100% and 50% (arrows indicate the direction of the moving dots).

a 2-down-1-up adaptive staircase procedure to measure a motion coherence threshold. The staircase began with 100% coherent stimuli and had a proportional step size of 50% before the first reversal and 25% thereafter. The staircase terminated after 12 reversals and the average of the last 11 reversals was calculated to estimate the coherence form threshold. The staircase was repeated twice and the average threshold was used for data analysis.

Contrast sensitivity for motion direction discrimination

Contrast sensitivity thresholds for coherent motion direction discrimination were measured to control for the possibly that global motion perception might be affected by deficits in lower level visual functions such as contrast sensitivity. Stimuli used for motion direction discrimination contrast thresholds were generated using Psykinematix software with parameters identical to the global motion perception task. In this task, RDKs were always presented with 100% coherence and dot contrast was varied.

Procedure

RDKs with 100% coherence and 70% contrast were presented and the motion direction was varied (up/down) until the participant was able to correctly identify the direction of 2 consecutive trials. A 2-down-1-up adaptive staircase with a proportional step size of 50% before the first reversal and 25% thereafter was then used to assess the contrast threshold for motion direction discrimination. The staircase terminated after 5 reversals and the average of the last 4 reversals was calculated to estimate the contrast threshold.

Vision tests

Visual acuity

Monocular and binocular distance visual acuity was measured using the Freiburg Vision Test ("FrACT") at a distance of 3 meters (45). Landolt-Cs were presented on a 27-inch iMac display in one of 4 orientations. The participants were asked to indicate the Landolt-C orientation using a keypad. A best parameter estimation by sequential testing (PEST) algorithm was used to determine each acuity threshold in logMAR. Near binocular visual acuity was measured using the Bailey Lovie Near Visual Acuity Chart at 40cm (46). Participants read from the largest row letter by letter and were asked to guess the letters when they were not sure. Visual acuity was recorded using letter-by-letter scoring, where each letter was equal to 0.02 logMAR.

Contrast sensitivity

Contrast sensitivity was assessed using the Freiburg Vision Test ("FrACT") with a Landolt C target viewed from 3 meters (47). The participant used a directional keypad to indicate the orientation of the Landolt C within an 8-alterntive-forced-choice procedure and threshold was calculated using the PEST algorithm. Thresholds were recorded as log contrast sensitivity (log CS).

Stereo acuity

The VAC FLY Stereo Acuity Test (40 cm viewing distance) was used to estimate disparity thresholds for local stereopsis in the range of 400 to 20 seconds of arc with the graded circles optotypes. If the participant could not appreciate the depth of the circles, the housefly optotype was used to identify whether the participant had gross stereopsis. The Randot Preschool Stereo Acuity Test (40 cm viewing distance) was used to estimate global stereopsis disparity thresholds with the range of 800 to 4 arc sec. The smallest disparity at which a participant was able to correctly identify at least 2 of 3 circles or shapes within the set of test panels was recorded as the local or global stereo acuity threshold respectively.

Additional clinical vision tests

Participants also completed the following vision oculomotor and accommodation tests administered by a research optometrist: 1) confrontation visual fields whereby static, single-quadrant counting was used to identify any gross visual field defect in the peripheral visual field, 2) pupil reflexes to rule out any relative afferent pupillary defect, 3) near point of convergence (NPC) was measured using a RAF ruler, 4) vergence facility was tested using a 12BO/3BI prism flipper to assess a participant's ability to rapidly change vergence without changing accommodation, 5) accommodative amplitude was measured monocularly and binocularly using the RAF ruler (push-up method), and 6) accommodative facility was tested binocularly using a +/−2.00 D flipper to assess the participant's ability to change accommodation without changing vergence.

Symptom assessment

Participants with mild TBI were asked to complete a non-standardized questionnaire designed to obtain information about their medical history. In addition, they were asked to

complete the standardized self-report Post-Concussion Symptom Inventory (PCSI) (48,49).

Statistical analysis

Statistical analyses were conducted using SPSS statistical software. The Shapiro-Wilk test was used to assess whether data were normally distributed. Three analyses were then conducted. Firstly, a repeated measures ANOVA with factors of Group (mild TBI vs control) and Test Type (global motion vs. global form) was used to compare form and motion coherence thresholds between the two groups. Secondly, independent samples t-tests (parametric) or Mann-Whitney tests (non-parametric) were used to compare clinical measurements of vision between the two groups. Thirdly, relationships between the mild TBI group PCSI symptom scores and performance on vision measures were quantified using Pearson or Spearman correlations.

Results

All participants successfully completed the entire test battery. Demographic information and clinical vision test results for the mild TBI and control groups are shown in Table 1. Mild TBI was caused by motor-vehicle or sporting accidents in our sample. Participants with mild TBI details are provided in Table 2.

Global form and motion perception

Results are provided as mean ± SD. Coherence thresholds (Figure 3) for both global form and motion perception were higher (worse) in the mild TBI group (form 25 ± 6%, range 16 to 33%; motion 14 ± 7%, range 8 to 32%) compared to the control group (form 21 ± 5%, range 15 to 32%; motion 11 ± 3%, range 6 to 17%). Coherence thresholds were not normally distributed. Therefore, a log transformation was applied to enable parametric analysis. ANOVA revealed a significant main effect of Group ($F_{1,34}$ = 7.1, p = .01) whereby coherence thresholds were higher (worse) in the mild TBI group. The interaction between Group and Test Type was not significant ($F_{1,34}$ = 0.1, p = .51). This indicates that the mild TBI group deficit was equivalent for both global perception tasks.

Global form coherence thresholds were not significantly correlated with near visual acuity (rho = 0.04, p = .9) or static contrast sensitivity (rho = 0.16 p = .66) among participants with mild TBI. Similarly, global motion coherence thresholds were not significantly correlated with contrast thresholds for motion direction discrimination (rho = 0.31, p = .35). Therefore, impairments in the global processing of form and motion in the mild TBI group were not directly related to measures that targeted the early stages of form or motion processing within V1.

Contrast thresholds for motion direction discrimination

Contrast thresholds for motion direction discrimination did not differ significantly between the two groups (t_{34} = 0.54, p = .59; Figure 4).

Correlations with PCSI symptoms score and time since injury

A secondary analysis was conducted to assess the relationships among the global form coherence thresholds, global motion coherence thresholds, contrast thresholds for motion direction discrimination, PCSI symptom scores and time since

Table 1. Demographic and vision test result comparisons between mild TBI and control subjects.

	Normal controls	Mild TBI	t-test (p value)
Age (yrs) (mean ± SD)	25.48 (± 5.2)	28 (± 8.56)	0.28
Distance VA	−0.24 (± 0.06)	−0.20 (± 0.10)	0.26
Near VA	−0.13 (± 0.09)	−0.05 (± 0. 09)	0.02
Log contrast sensitivity	2.13 (± 0.23)	1.95 (± 0.16)	0.03
Stereo Acuity			
Local stereopsis	20.4 (± 1.38)	26.7 (± 10)	<0.01
Global stereopsis	40 (± 0)	60 (± 28.28)	<0.01
NPC	6.6 (± 3.1)	12 (± 9.9)	0.01
Vergence facility	15.32 (± 3.67)	12.7 (± 6.3)	0.13
Accommodative facility	9.8 (± 2.5)	4.9 (± 4.19)	<0.01
Accommodation amplitude difference	4.53 (± 2.9)	−0.04 (± 2.7)	<0.01

Table 2. Mild TBI participant characteristics.

Mild TBI subject #	Gender/Age	Time since injury (months)	Number of symptoms/20 Total	Total PCSI score/120	Medications
1	Female/19	3	16	80	No
2	Female/40	5	20	86	No
3	Female/40	5	19	46	No
4	Male/23	20	2	4	No
5	Female/26	36	1	1	No
6	Male/21	47	2	2	No
7	Male/22	17	7	15	No
8	Male/22	15	8	25	No
9	Female/24	32	11	27	No
10	Female/29	3	19	74	Yes
11	Female/42	16	19	78	Yes

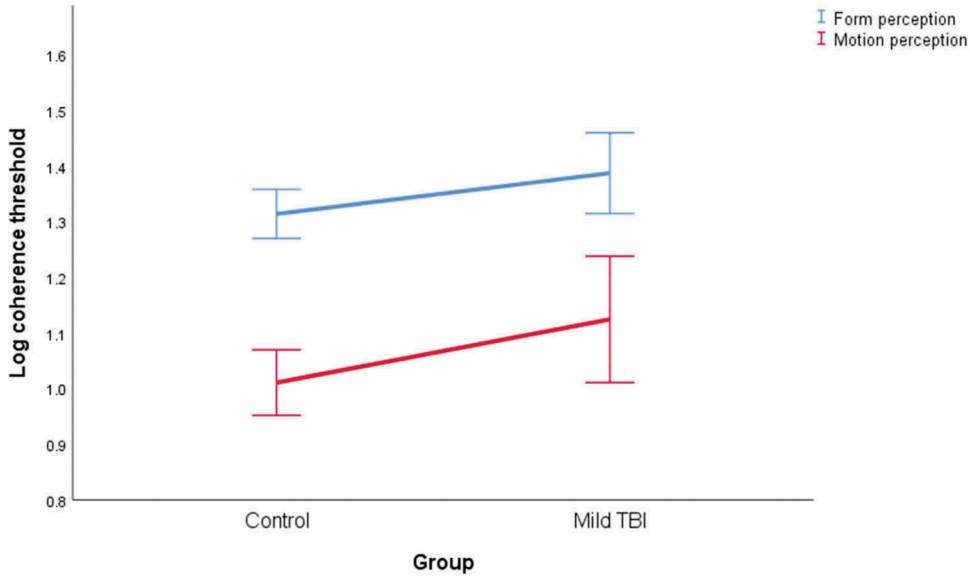

Figure 3. Form and motion coherence thresholds for the normal and mild TBI groups. Error bars represent 95% confidence intervals.

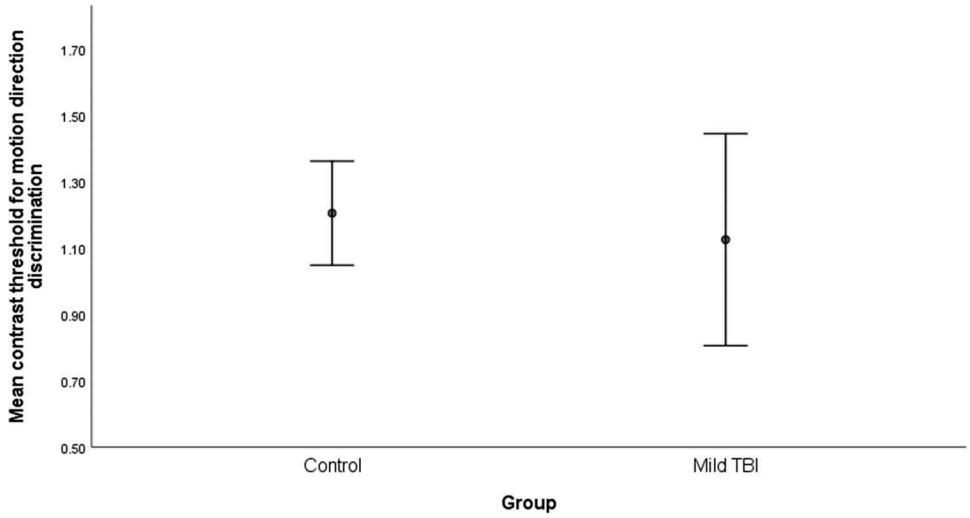

Figure 4. Comparison of contrast thresholds for motion direction discrimination between the mild TBI and normal control groups. Error bars represent 95% confidence intervals.

injury. PCSI symptom scores were not significantly correlated with log global form (R^2 = 0.008, p = .79) or global motion (R^2 = 0.29, p = .08) coherence thresholds. There was a significant positive correlation between PCSI symptom scores and contrast thresholds for motion discrimination (R^2 = 0.51, p = .01; Figure 5). There was also a significant negative correlation between PCSI symptom score and the time since injury (R^2 = – 0.59, p < .01). Time since injury did not correlate significantly with any visual outcome measure (all p > .05).

Discussion

Our results indicate that global form and motion perception, and, by extension, the dorsal and ventral cortical visual processing streams (31,32), are impaired equally by mild TBI. This pattern of results is not consistent with the dorsal stream vulnerability hypothesis, which proposes that the dorsal stream is more vulnerable to damage than the ventral stream (38), at least during visual development. The diffuse structural and metabolic changes caused by mild TBI appear to be sufficiently extensive to cause widespread disruption in visual processing that extends to both major extra-striate visual cortex processing streams (12,17). This is consistent with brain imaging studies that indicate generalized alterations in cortical functional connectivity following mild TBI (14,19).

Our findings are in agreement with those of Patel et al. (44) and Brosseau-Lachaine et al. (10) who reported a significant elevation of motion coherence thresholds in individuals with mild TBI compared to controls. We extend these previous results to show that the global processing deficit for motion processing in mild TBI also extends to form perception.

Elevated motion coherence thresholds can be caused by abnormal processing within dorsal stream areas such as V3A

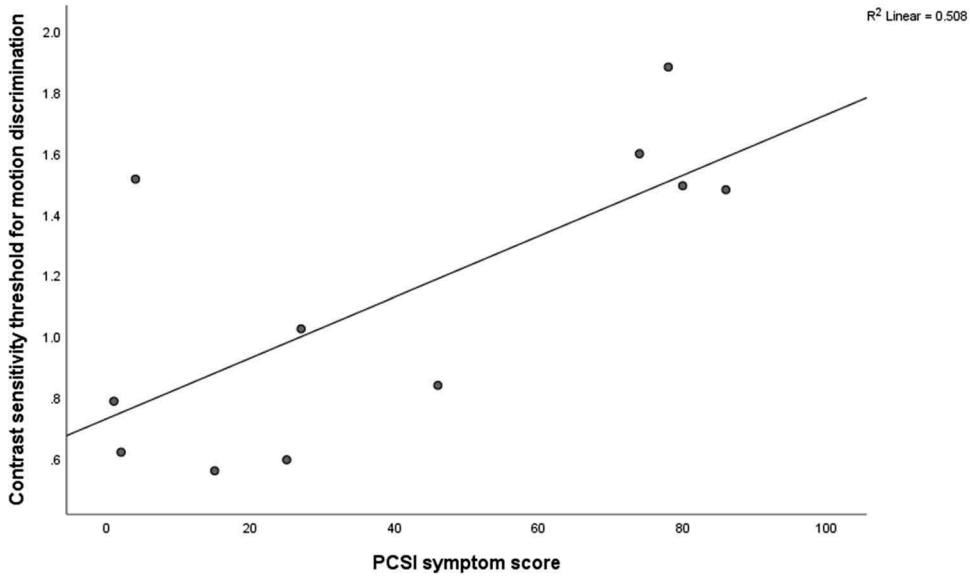

Figure 5. The relationship between PCSI symptom scores and contrast sensitivity for motion direction discrimination.

and MT/V5 or impaired local motion processing due to damage within the magnocellular retino-geniculate pathway and/or V1. In this study, we did not include a task that specifically targeted early processing within the retino-geniculate magnocelluar pathway such as flicker detection (50) due to time constraints within the test protocol. However, contrast thresholds for motion direction discrimination were assessed and provided a measure of local motion processing (36). We did not observe any statistically significant differences between the mild TBI and control groups for motion direction discrimination contrast thresholds. In addition, motion direction discrimination contrast thresholds were not correlated with motion coherence thresholds within the mild TBI group. Together, these results suggest that the global motion impairment in the mild TBI group was due to a specific motion integration deficit rather than impaired processing of local motion signals.

Deficits in global form processing can also occur as a result of impaired processing of local information in V1. We did not include a contrast sensitivity measure for our global form stimuli due to time constraints within the test protocol; however, we did include a measure of static contrast sensitivity along with an assessment of near visual acuity that involves static, high spatial frequency processing. The mild TBI group had significantly higher (worse) thresholds than the control group for both of these tests, however all thresholds fell within what is considered to be the normal range (1.65–1.95 log CS) (51). Neither static contrast sensitivity, nor near visual acuity were significantly correlated with form coherence thresholds in the mild TBI group. Therefore, as a whole, our data are consistent with the presence of a specific global form processing deficit in our mild TBI group. However, we cannot rule out the possibility that mild impairments in local form processing in V1 are amplified in extrastriate areas and contribute to the elevated form coherence thresholds we observed in the mild TBI group.

Mild TBI impairs attention (52). Therefore, it is possible that our results could be explained by a general attention deficit in the mild TBI group. Two pieces of evidence argue against this possibility. First, the mild TBI group were not impaired on the motion direction discrimination contrast threshold task relative to the control group. If group differences were due to an overall difference in attention, impairments for all psychophysical tasks would be expected. Second, scores on the PCSI components that specifically relate to cognitive symptoms including difficulty concentrating, difficulty remembering and feeling mentally foggy were not correlated with any of the psychophysical thresholds within the mild TBI group (all rho < 0.50, $p > .05$). Although not conclusive, these findings suggest that our results are due to deficits in vision processing rather than a general impairment in attention.

Although contrast thresholds for motion direction discrimination were not significantly different between the control and mild TBI groups, there was a significant positive correlation between motion contrast thresholds and PCSI scores (worse thresholds were associated with worse symptom scores). The question of whether there is a causal relationship between contrast thresholds for dynamic stimuli and mild TBI symptoms remains to be answered. However, our finding is consistent with that of Chang et al (43) who reported a significant relationship between the symptoms reported in patients with mild TBI and critical flicker frequency (CFF) thresholds whereby increased thresholds were associated with worse symptom scores. They suggested that the relationship between CFF and mild TBI symptoms might reflect altered gain control within the magnocellular pathway. In the present study we found no significant correlation between PCSI scores and either global form or global motion coherence thresholds suggesting that PCSI symptom scores are associated with lower-level visual function rather than performance on higher-level global integration tasks.

Although it has been reported that patients with mild TBI typically recover and return to normal from 1 to 3 months post injury, more than 70% of the participants who sustained mild TBI in our sample still had symptoms (53). Two participants in the mild TBI group reported in the medical history questionnaire that they were using medication such as antidepressants, and it is possible that these medications may have affected their visual function (54).

As expected (8,9,55,56), participants with mild TBI performed significantly worse than controls on all of the clinical vision tests that we administered with the exception of distance visual acuity (which was part of our inclusion criteria). This included static contrast sensitivity, although thresholds within the mild TBI group remained within the normal range (51). The contrast sensitivity test that we used involved a low spatial frequency optotype. Therefore, our results are consistent with Spiegel et al (57) who reported that the contrast sensitivity function is shifted toward high spatial frequencies in those with mild TBI. Secondly, our participants with mild TBI had reduced local and global stereo acuity. Deficits in the detection of retinal disparity in individuals with mild TBI have been linked to impaired cortical processing rather than oculomotor control abnormalities (58). Stereopsis is thought to be linked to both dorsal and ventral streams (59,60). Therefore, the reduction of stereopsis is possibly due to an impairment of both processing streams, which is consistent with our primary finding of defects in both the dorsal and ventral processing streams. We also observed that participants with mild TBI performed worse than controls in the NPC, accommodation facility and accommodation amplitude tests. These findings are consistent with previous studies of patients with mild TBI and may be due to a deficit within the neural pathways associated with the vergence and accommodative systems (8,9,55,56).

Our study has a number of limitations. The use of self-report rather than medical records to determine a diagnosis of mild TBI raises the possibility that not all participants in our mild TBI group had a formal medical diagnosis. Stringent screening was used to minimise this risk, but it cannot be ruled out. We also used the PCSI rather than the Adult Post Concussion Scale that may have been more appropriate for our sample population. In addition, there was considerable variability in time since injury and PCSI score within our mild TBI group. One advantage of this variability was that it allowed us to measure correlations between PCSI, time since injury and vision outcome measures. The only significant correlation we observed for vision measures was between contrast thresholds for motion direction discrimination and PCSI score, however our small sample size and the presence of multiple comparisons precludes strong conclusions. In addition, although we did not observe a correlation between vision outcome measures and PSCI scores relating to attention, we cannot completely rule out a generalized effect of mild TBI on attention because time constraints precluded the inclusion of visual-attention-specific tests within our protocol. Finally, our mild TBI sample population was biased towards participants with vision-related symptoms. It is not clear whether our results will generalize to individuals with mild TBI who do not seek clinical vision services.

Our results suggest a generalized visual integration deficit in individuals with mild TBI along with an association between spatio-temporal contrast sensitivity and PCSI score. Performance on visual tasks involving global integration or spatio-temporal contrast sensitivity can be improved by techniques such as perceptual learning and non-invasive visual cortex stimulation (61–65). It is conceivable that these techniques may also improve visual function in individuals with mild TBI.

In conclusion, mild TBI has widespread effects on the cortical processing of visual information that extend to both the dorsal and ventral processing streams and last at least 3 months post-injury. Measures of global form and motion perception may be useful in the assessment of recovery from mild TBI.

Declaration of interest

The authors report no conflicts of interest.

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