Visual Psychophysics and Physiological Optics

Sensitivity to Binocular Disparity is Reduced by Mild Traumatic Brain Injury

Gunnar Schmidtmann,[1,2] Tatiana Ruiz,[1,2] Alexandre Reynaud,[2] Daniel P. Spiegel,[1,2] Maude Laguë-Beauvais,[1,3] Robert F. Hess,[2] and Reza Farivar[1,2]

[1]Traumatic Brain Injury Program, The Research Institute of the McGill University Health Centre, Montreal, Canada
[2]McGill Vision Research Unit, Department of Ophthalmology, McGill University, Montreal, Canada
[3]Department of Neurology and Neurosurgery, McGill University, Montréal, Canada

PURPOSE. The impairment of visual functions is one of the most common complaints following mild traumatic brain injury (mTBI). Traumatic brain injury–associated visual deficits include blurred vision, reading problems, and eye strain. In addition, previous studies have found evidence that TBI can diminish early cortical visual processing, particularly for second-order stimuli. We investigated whether cortical processing of binocular disparity is also affected by mTBI.

METHODS. In order to investigate the influence of mTBI on global stereopsis, we measured the quick Disparity Sensitivity Function (qDSF) in 22 patients with mTBI. Patients with manifest strabismus and double vision were excluded. Compared with standard clinical tests, the qDSF is unique in that it offers a quick and accurate estimate of thresholds across the whole spatial frequency range.

RESULTS. Results show that disparity sensitivity in the mTBI patients were significantly reduced compared with the normative dataset ($n = 61$). The peak spatial frequency was not affected.

CONCLUSIONS. Our results suggest that the reduced disparity sensitivity in patients with mTBI is more likely caused by cortical changes (e.g., axonal shearing, or reduced interhemispheric communication) rather than oculomotor dysfunction.

Keywords: traumatic brain injury, stereopsis, disparity sensitivity

The ability to perceive depth information from binocular disparity (stereopsis) is achieved by multiple cortical areas starting with the primary visual cortex (V1).[1-3] Unlike local stereopsis, global stereopsis can occur in the absence of monocularly perceived cues by integrating local stereoscopic information over large spatial regions.[4-7]

Research on both monkeys and humans has shown that cortical lesions can produce a marked impairment in stereopsis.[8,9] Lesion studies indicate that different types of stereopsis are processed at different cortical loci by separate mechanisms. For example, damage to the temporal lobe in macaque monkeys compromised global stereopsis while damage to V1 and V2 did not.[9] Conversely, severing V2 had a substantial detrimental effect on local stereopsis. In humans, right temporal lobectomy significantly worsened global stereopsis while local stereoacuity remained unchanged[10-12] (but see Kim et al.[13]). In addition to lesion studies, there is growing evidence that deficits in stereopsis occur in neurodegenerative diseases such as Parkinson's[13-15] and Alzheimer's.[16]

Disparity processing may also be affected by traumatic brain injury (TBI). In the United States alone, TBI affects up to 5.3 million people annually,[17-19] which makes it one of the most common causes of hospitalization and disability.[20,21] Miller and colleagues[22] examined local stereopsis in 93 TBI patients using a standard clinical stereo test (Stereo Optical Company Test 004; Stereo Optical Company, Inc.). They reported that 24% of patients had a total lack of stereopsis and 41% performed considerably worse than control participants. The degree of impairment was related to TBI severity, memory abilities, and presence of brain lesion.[22] The deficits reported by Miller et al.[22] were based on a coarse clinical test in a population with more severe brain injuries, thus raising the question of whether disparity processing is also impaired in mild TBI (mTBI). Note that the standard clinical tests for stereopsis, such as the one used by Miller et al.,[22] are limited in a number of ways: the disparity scale is coarsely quantized, and there is no way of estimating the variance associated with these measurements because psychophysical procedures involving multiple presentations of the same stimulus are not practical.

Depth processing may thus be one of several aspects of cortical visual processing that appears to be affected in TBI, and even mTBI. For example, Brosseau-Lachaine et al.[23] have demonstrated that children with mTBI have a decreased sensitivity to static and dynamic contrast-defined second-order stimuli, whereas sensitivity to first-order (luminance-defined) stimuli were not affected. Piponnier et al.[24] showed that reaction times on a motion direction discrimination task were longer in mTBI patients for both first- and second-order stimuli and compared with the control group, the reaction times for second-order stimuli were longer than for first-order stimuli in the mTBI group. Traumatic brain injury patients have elevated thresholds for global motion.[25] Finally, we have recently reported decreased sensitivity for static contrast and texture-defined second-order stimuli,[26] as well as reduced interhemispheric transfer for visual signals.[27]

2630

TABLE. Participant Details

ID	Age	Sex	VA RE	VA LE	Bino VA	Heterophoria/tropia
T1	29	Female	−0.16	−0.08	−0.18	Ortho
T2	33	Female	0.02	−0.04	−0.16	Exophoria 2 PD
T3	57	Female	0	0	-0.1	Ortho
T4	32	Female	−0.2	−0.2	−0.24	Ortho
T5	63	Female	0.06	0.06	0	Ortho
T6	18	Female	0	0.16	0	Exophoria 3 PD
T7	40	Male	0.08	−0.08	−0.1	Esophoria 8 PD
T8	23	Female	−0.14	−0.18	−0.18	Ortho
T9	44	Female	−0.1	−0.1	−0.12	Ortho
T10	24	Female	0.08	−0.04	−0.06	Exophoria 4 PD
T11	31	Female	0.04	−0.2	−0.2	Ortho
T12	26	Male	−0.1	−0.2	−0.12	Ortho
T13	24	Male	−0.02	−0.04	−0.04	Ortho
T14	50	Male	−0.02	−0.18	−0.22	Exotropia 2 PD
T15	28	Female	−0.06	−0.08	−0.1	Exophoria 3 PD
T16	44	Male	0.04	−0.06	−0.14	Ortho
T17	19	Female	−0.1	−0.1	−0.18	Ortho
T18	38	Female	−0.08	−0.18	−0.2	Exophoria 2 PD
T19	18	Female	−0.08	−0.04	−0.1	Ortho
T20	24	Male	0.22	0.24	0	Ortho
T21	39	Female	0.12	0.14	0.12	Ortho
T22	20	Male	0.02	−0.08	−0.14	Ortho

Visual acuity (VA) is expressed as logMAR. RE, right eye; LE, left eye; Bino, binocular; PD, prism dioptres.

Detection of disparity sensitivity changes after mTBI may contribute to diagnosing the injury and to characterizing the nature of the cortical loss. Because the changes after mTBI are likely to be subtle and because we cannot know what aspect of disparity processing is likely affected, it is crucial that we adopt a measure that remains comprehensive with regard to the stimulus range, but is also precise so as to be sensitive to subtle changes. We have previously used the quick Contrast Sensitivity Function paradigm (qCSF[26,28,29]) to characterize the CSF across a large range of spatial frequencies after mTBI. Here, we adopt a similar procedure, the quick Disparity Sensitivity Function (qDSF[30]), to characterize disparity sensitivity across a range of spatial frequencies for global stereopsis. With the qDSF approach, we remained sensitive to subtle changes that may accompany mTBI, while also remaining unbiased with regard to the range of spatial frequencies tested. We asked whether disparity sensitivity was affected after mTBI, and whether mTBI affected the overall sensitivity to disparity, or disrupted sensitivity to a specific spatial frequencies.

METHODS

Subjects

TBI Group. A group of 22 participants (7 males, 15 females, mean age 32 years, ±12.7 SD) with a history of mTBI were recruited from the McGill University Health Centre Out-Patient TBI Program or via public advertisements. Participant details are summarized in the Table. The criteria of the diagnosis were: (1) any amnesia of events immediately before or after the accident lasting no longer than 24 hours, and (2) a Glasgow Coma Score ranging between 13 and 15. If loss of consciousness was present, it had to be shorter than 30 minutes. All participants completed a short neuropsychological screening, including (1) visual attention using the Trail Making Test A and B,[31] the Bells Test,[32] and (2) spatial neglect by using the Clock-drawing test.[33] Prior to data collection, a short verbal screening for relevant medical history was performed, which included questions regarding recurrent migraines, psychiatric disorders, or vertigo.

The exclusion criteria were general anesthesia within the past 6 months, other acquired brain injuries in the past, severe tremors, and/or epilepsy.

Subjects were also assessed for the presence of a strabismus by the "Cover-Uncover" and "Alternating Cover Tests." The magnitude of any heterophoria was measured with the "Maddox Rod Test." Patients with double vision or manifest strabismus were excluded. Participants underwent an assessment of their monocular and binocular visual acuity (Logarithmic Visual Acuity Chart; Precision Vision, Lasalle, IL, USA) at a viewing distance of 4 m and their ocular dominance was determined (Miles Test). We collected visual dysfunction data from 16 of 22 subjects contacted after the study. The subjects responded to a questionnaire adapted from Assessment and Management of Visual Dysfunction Associated with Mild Traumatic Brain Injury for the Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury.[26]

Normative Dataset. The TBI group was compared with the normative dataset, previously recruited by our Department to validate the qDSF paradigm.[30] The normative dataset consisted of 61 subjects (25 males, 36 females, mean age 26 years, ±5.7 SD). All subjects had normal or corrected-to-normal visual acuity. All procedures were in accordance with the Declaration of Helsinki and were approved by the Research Ethics Board of the McGill University Health Centre. Informed consent was obtained from all participants prior to data collection.

The TBI group and normative dataset were tested for normality by using Shapiro-Wilk tests and revealed that the normative dataset was normally distributed, whereas the TBI group was not (Normative: $W(61) = 0.950$, $P = 0.0143$, TBI: $W(22) = 0.916$ $P = 0.0619$). A nonparametric Mann-Whitney U test showed that both groups were not statistically significantly different with respect to their age distribution ($U = 527.5$, $P = 0.066$).

Apparatus

The stimuli were generated within the MATLAB (MATLAB R 2012a; The MathWorks, Natick, MA, USA) environment on a PC
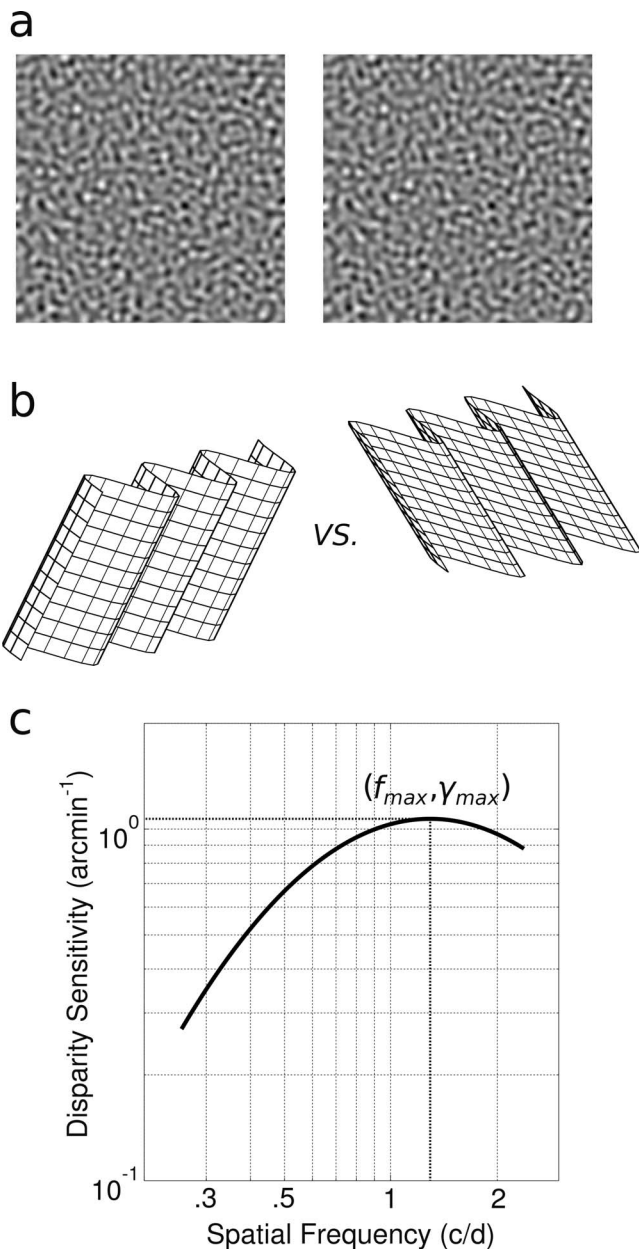
FIGURE 1. Stimulus examples, experimental paradigm and disparity sensitivity function (DSF). (**a**) Two-dimensional fractal noise stereograms filtered with different frequency bands, viewed through passive polarized glasses through a 3D monitor. (**b**) The task was to indicate the direction of the disparity modulation (left oblique versus right oblique; 45° or 135°). (**c**) The log-log parabola DSF is defined by four parameters: peak SF f_{max}, max gain γ_{max}, the bandwidth β, and the truncation δ. However, only f_{max} and γ_{max} were kept for further analysis. Reprinted from Reynaud A, Gao Y, Hess RF. A normative dataset on human global stereopsis using the quick Disparity Sensitivity Function (qDSF). *Vision Res*. 2015;113:97–103, with permission from Elsevier. Copyright © 2015 Elsevier Ltd.

(Intel Core i7 processor, 4 GB RAM, 2.67 Hz, ATI Radeon HD 3400 8-bit graphics card; DELL, Round Rock, TX, USA) and presented on a calibrated, gamma-corrected 2300 3D-Ready LED monitor ViewSonic V3D231 (ViewSonic Corporation, Brea, CA, USA) with a mean luminance of 100 cd/m². The stereo image was displayed in interleaved line stereo mode at a resolution of 1920 × 1080 pixel and a refresh rate of 60 Hz. Stimuli were presented at a viewing distance of 70 cm in a dim-

lit room. The observers viewed the stimuli stereoscopically through passive polarized three-dimensional (3D) glasses. The polarized filters lead to a luminance reduction of approximately 40%, which was measured with a photometer.

Stimuli

The stimuli consisted of dichoptically presented fractal noise (carrier), band-pass filtered with central spatial frequencies of 0.94, 1.31, 1.83, 2.54, 3.54, 4.93, 6.87, or 9.57 c/deg, and one octave bandwidth. The disparity between the two eyes was modulated by oblique (45°: left; or 135°: right) sinusoidal corrugation at spatial frequencies corresponding to one-forth of the carrier spatial frequency (i.e., the ratio between the central spatial frequency of the filter and the disparity modulation was always kept at 4-to-1). A circular Gaussian aperture of Sigma = 7.5° on a gray background was then applied. The carrier spatial frequency (and consequently disparity modulation spatial frequency) and disparity were determined by the qDSF routine.[30] Example stimuli and experimental paradigm are shown in Figures 1a and 1b.

The Quick Disparity Sensitivity Function (qDSF)

We used the qDSF paradigm for assessment of global stereopsis.[30] The qDSF, based on the qCSF,[28,29] is a Bayesian adaptive procedure that estimates multiple parameters of psychometric function allowing for quick estimates of thresholds across the whole frequency range. Within an experimental run, the qDSF algorithm searches in real-time and based on previous responses for the optimal carrier spatial frequency and disparity in order to maximize the information gain about the subjects' disparity function.

The qDSF is based on a truncated log-log parabola model of the CSF (Fig. 1c).[34,35] The function is defined by four parameters: peak SF f_{max}, max gain γ_{max}, the bandwidth β, and the truncation δ.

$$S'(f) = \log_{10}(\gamma_{max}) - \kappa \left(\frac{\log_{10}(f) - \log_{10}(f_{max})}{\frac{\beta'}{2}} \right)^2$$

$$S(f) = \log_{10}(\gamma_{max}) - \delta \quad \text{if} \quad f < f_{max} \wedge S'(f) < \log_{10}(\gamma_{max}) - \delta$$

(1)

$$S(f) = S'(f) \text{ else}$$

with $\kappa = \log_{10}(2)$ and $\beta' = \log_{10}(2\beta)$.

Similar to Reynaud et al.,[30] we have not analyzed the bandwidth and truncation parameters, because these parameters were usually out of range and could not be reliably estimated.

Procedure

The experimental procedure was similar to the one introduced by Reynaud et al.[30] Observers were asked to identify the orientation of the disparity modulation (i.e., left oblique versus right oblique [45° or 135°]) in a single-interval identification paradigm (see Fig. 1b). The monitor was initially set to a mean gray luminance. An experimental trial consisted of the following sequence: (1) a green fixation dot appeared on the screen, (2) the fixation dot disappeared and the stimulus was presented for 1 second, (3) a red fixation dot appeared until the subject responded by pressing one of two keys on a numeric keypad, and (4) the fixation dot disappeared and
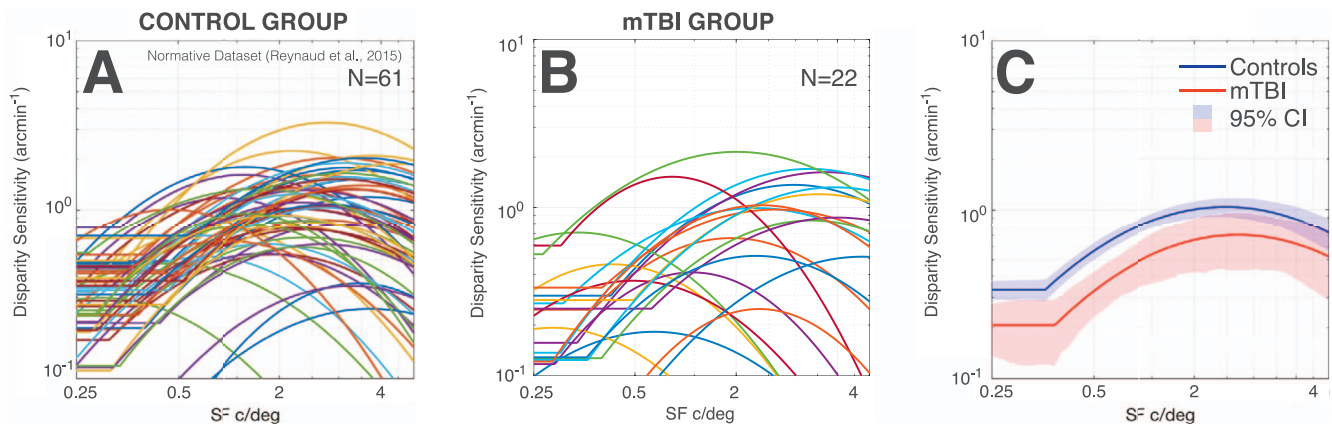
FIGURE 2. (**A**) Individual qDSFs for the normative dataset ($N = 61$, Reynaud et al.[30]). (**B**) Individual qDSFs for the mTBI group ($N = 22$). (**C**) Average qDSFs expressed as the nonparametric pseudomedian for the normative dataset in *blue* and the mTBI group in *red*. The *shaded areas* represent $\pm$ nonparametric 95% confidence intervals. *$P < 0.05$ Mann-Whitney U test.

audio feedback was provided. Dot luminance was matched to that of the background.

RESULTS

Figure 2A shows the individual qDSFs for the normative dataset ($N = 61$, Reynaud et al.[30]) and Figure 2B for the mTBI group ($N = 22$) investigated here. In each graph, the disparity sensitivity in arcmin^{-1} is plotted against the spatial frequency of the disparity modulation in cycles per degree of visual angle. For each sensitivity function, we derived two key parameters: the height of the function (max gain, γ_{max}) and the position of the maximum sensitivity (peak SF, f_{max}) (see Fig. 1c).

A Kolmogorov-Smirnov test was employed to test the relevant dependent variables (max gain, peak SF) for normality. This test revealed that with the exception of max gain for the normative dataset ($W(61) = 0.949$, $P = 0.0140$), the qDSF parameters are not normally distributed for both groups (peak SF Normative: $W(61) = 0.990$, $P = 0.9043$; max gain TBI: $W(22) = 0.934$, $P = 0.1458$; peak SF TBI: $W(22) = 0.967$, $P = 0.6312$).

The resulting average qDSFs, expressed as the nonparametric pseudomedian, are shown in Figure 2C where the average qDSF for the normative dataset is shown in blue and the corresponding function for the TBI group in red. The shaded regions refer to nonparametric ±95% confidence intervals.

To validate the results statistically, we performed Mann-Whitney U tests, which revealed a statistically significant

difference between TBIs and controls for max gain ($U = 490$, $P = 0.034$), but not the peak SF ($U = 628$, $P = 0.461$), as evident in the downwards shift of the median qDSF for the TBI group (Fig. 2C).

The group summary results for max gain and peak SF are presented as bar plots in Figure 3.

Additional Spearman correlation analyses were performed between the relevant qDSF parameters (peak SF and max gain) versus age, and the optometric and neuropsychological screening tests results. The correlations and the corresponding r and P indices are presented in each graph of Figure 4. None of these correlations are significant.

The Visual Dysfunction questionnaire that the 16 subjects completed contained questions about: (1) general changes in vision after the injury, (2) problems related to blurred vision, (3) visual acuity changes, (4) dizziness or balance problems, (5) clear vision, (6) computer work problems, and (7) headaches during computer work. Three patients reported moderate to severe overall changes in vision. Two patients reported moderate to severe problems caused by blurred vision. Moderate visual acuity changes were experienced by one patient. Moderate to severe visual problems related to balance and dizziness were reported by two patients and two subjects mentioned moderate to severe problems while reading or working on a computer screen.

Spearman correlation analyses were performed between the peak SF and max gain versus blurred vision and all abovementioned visual problems. None of these correlations were significant.
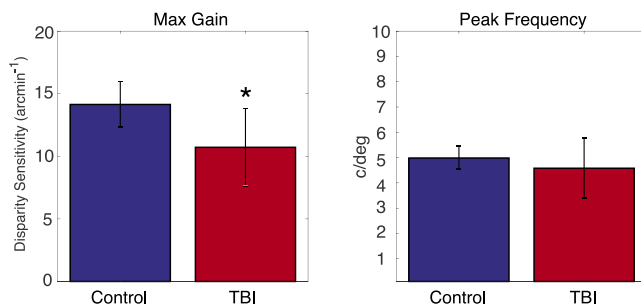
DISCUSSION

The main aim of the current study was to systematically investigate the integrity of global stereopsis in patients with mTBI across a range of spatial frequencies. Global stereopsis involves the integration of local depth values across large regions of the visual field, and as such most likely relies on extrastriate processing, where neurons have larger receptive fields composed of many smaller V1 subunits. Previous studies in this field have focused on more local measures of stereo vision that likely reflect processing by early cortical areas (V1/V2). Unlike previous studies, we did not confine our assessment to the smallest detectable disparity (i.e., stereoacuity), but rather measured disparity sensitivity over the full SF range.
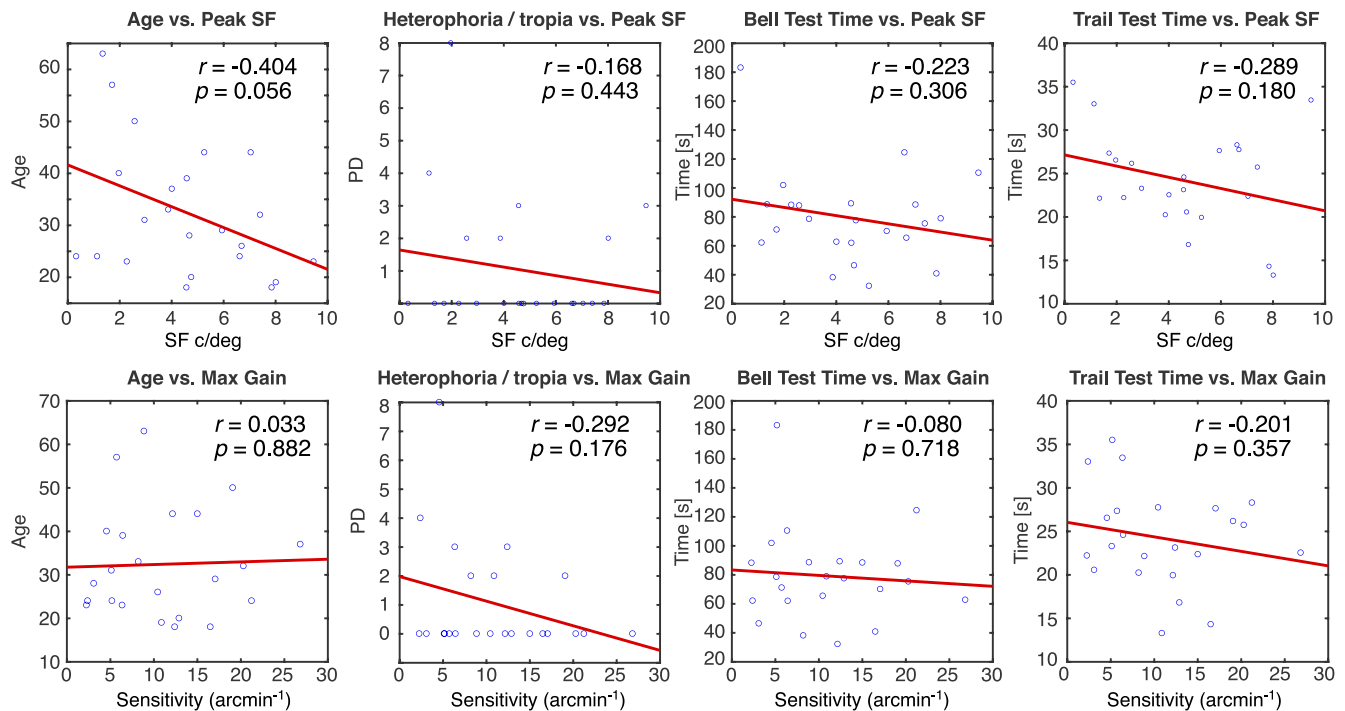

FIGURE 3. Pseudomedian estimates of max gain (*left*) and peak SF (*right*) shown for the control group in *blue* and the TBI group in *red*. The *error bars* represent ± nonparametric 95% confidence intervals. The *asterisk* (*) refers to statistically significant differences between control and TBI group ($P < 0.05$ Mann–Whitney U test).

FIGURE 4. Correlations between the peak SF (*top*) and Max gain (*bottom*) versus age and results from optometric and neuropsychological screening tests. Left to right: age, heterophoria/tropia, Bell Test time, and Trail Test time.

To do so, we employed the novel qDSF paradigm. Compared with standard clinical approaches (e.g., Stereo Fly Test, Butterfly Test or Random Dot Tests), this Bayesian adaptive procedure provides a quick and accurate estimate of thresholds across the full spatial frequency range. The qCSF, which is the methodologic basis for the qDSF algorithm, has already been successfully applied to assess vision in patients suffering from mTBI, demonstrating its clinical applicability.[26]

The main result from this study is that mTBI patients have a small but significant reduction in disparity sensitivity compared with the control group, and that this is a general loss occurring over the full spatial frequency range.

This raises two questions. First, what causes the decreased disparity sensitivity in TBI patients? And second, how does the decreased sensitivity relate to the commonly described visual problems that accompany mTBI?

Traumatic brain injury–associated visual deficits are diverse and include blurred vision, double vision, reading problems, increased sensitivity to motion and flicker, and eye strain.[20,21,27,36,37] None of these symptoms can explain the impairment that we report for stereopsis. The most relevant symptom in this regard is "blurred vision," however this would be expected to selectively affect disparity sensitivity for high spatial frequencies and result in a displacement of the peak to lower spatial frequencies, which was not observed. Other studies have found evidence that TBI can lead to more general oculomotor problems that could in turn affect binocular function, specifically vertical heterophoria.[38] Further studies reported vergence dysfunctions[39] and double vision after TBI.[20] While we specifically excluded patients with profound manifest oculomotor dysfunction of this kind, it remained a possibility that large compensated horizontal or small-but-significant compensated vertical heterophorias may have played a part in the stereo-sensitivity deficits we report. However, our correlation analysis presented in Figure 4 shows that there are no significant correlations between the amount of heterophoria and the key qDSF parameters.

Our results support our initial hypothesis that the likely cause of this stereo deficit in mTBI is a sensory rather than a motor loss.

Previous studies proposed a neuronal cause, specifically diffuse axonal shearing as the cause for impaired stereopsis in TBI.[22] The brain injuries caused by this axonal shearing have been shown to affect brain structures that are involved in midline stereovision, such as the corpus callosum.[40] By employing the travelling wave paradigm,[41] we have recently demonstrated that the interhemispheric communication is impaired in patients suffering from mTBI.[27] However, an impairment to midline stereopsis is unlikely to explain the current results where the stereo information is distributed over a large part of the visual field (stimulus sigma was 7.5°). These results are nevertheless consistent with a hypothesis that the reduced disparity sensitivity is the result of sensory loss due to cortical damage subsequent to brain trauma (e.g., axonal shearing).

In summary, we demonstrate that patients with mTBI experience significant impairments in global stereopsis. We argue that the reduced disparity sensitivity in patients with mTBI might be caused by sensory loss as the result of cortical damage (e.g., axonal shearing or reduced interhemispheric communication) rather than oculomotor dysfunction.

References

1. Barlow HB, Blakemore C, Pettigrew JD. The neural mechanism of binocular depth discrimination. *J Physiol*. 1967;193:327–342.

2. Howard IP, Rogers BJ. *Seeing in Depth*. Vol 2. Toronto, Canada: University of Toronto Press; 2002.

3. Neri P. A stereoscopic look at visual cortex. *J Neurophysiol*. 2005;93:1823–1826.

4. Fricke TR, Siderov J. Stereopsis, stereotests, and their relation to vision screening and clinical practice. *Clin Exp Optom*. 1997;80:165–172.

5. Witz N, Hess RF. Mechanisms underlying global stereopsis in fovea and periphery. *Vision Res*. 2013;87:10–21.

6. Tyler CW. Depth perception in disparity gratings. *Nature*. 1974;251:140–142.

7. Julesz B. Binocular depth perception of computer-generated patterns. *Bell Sys Tech J*. 1960;39:1125–1162.

8. Cowey A. Disturbances of stereopsis by brain damage. In: Ingle DJ, Jeannerod M, Lee D, eds. *Brain Mechanisms and Spatial Vision*. Dordrecht: Springer Netherlands; 1985:259–278.

9. Cowey A, Porter J. Brain damage and global stereopsis. *Proc R Soc Lond B Biol Sci*. 1979;204:399–407.

10. Ptito A, Zatorre RJ, Larson WL, Tosoni C. Stereopsis after unilateral anterior anterior temporal lobectomy. *Brain*. 1991;114:1323–1333.

11. Ptito A, Zatorre RJ, Petrides M, Frey S, Alivisatos B, Evans AC. Localization and lateralization of stereoscopic processing in the human brain. *Neuro Report*. 1993;4:1155–1158.

12. Verhoef B-E, Decramer T, van Loon J, et al. Stereopsis after anterior temporal lobectomy. *Cortex*. 2016;82:63–71.

13. Kim S-H, Park J-H, Kim YH, Koh S-B. Stereopsis in drug naïve Parkinson's disease patients. *Can J Neurol Sci*. 2011;38:299–302.

14. Koh S-B, Suh S-I, Kim S-H, Kim JH. Stereopsis and extrastriate cortical atrophy in Parkinson's disease: a voxel-based morphometric study. *Neuro Report*. 2013;24:229–232.

15. Lee C-N, Ko D, Suh Y-W, Park K-W. Cognitive functions and stereopsis in patients with Parkinson's disease and Alzheimer's disease using 3-dimensional television: a case controlled trial. *PLoS One*. 2015;10:e0123229.

16. Mendez MF, Cherrier MM, Meadows RS. Depth perception in Alzheimer's disease. *Percept Mot Skills*. 1996;83:987–995.

17. Coronado VG, Xu L, Basavaraju SV, et al. Surveillance for traumatic brain injury-related deaths–United States, 1997–2007. *MMWR Surveill Summ*. 2011;60:1–32.

18. Corrigan JD, Selassie AW, Orman JAL. The epidemiology of traumatic brain injury. *J Head Trauma Rehabil*. 2010;25:72–80.

19. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury. *J Head Trauma Rehabil*. 2006;21:375–378.

20. Greenwald BD, Kapoor N, Singh AD. Visual impairments in the first year after traumatic brain injury. *Brain Inj*. 2012;26:1338–1359.

21. Kapoor N, Ciuffreda KJ. Vision disturbances following traumatic brain injury. *Curr Treat Options Neurol*. 2002;4:271–280.

22. Miller LJ, Mittenberg W, Carey VM, McMorrow MA, Kushner TE, Weinstein JM. Astereopsis caused by traumatic brain injury. *Arch Clin Neuropsychol*. 1999;14:537–543.

23. Brosseau-Lachaine O, Gagnon I, Forget R, Faubert J. Mild traumatic brain injury induces prolonged visual processing deficits in children. *Brain Inj*. 2009;22:657–668.

24. Piponnier J-C, Forget R, Gagnon I, McKerral M, Giguère J-F, Faubert J. First- and second-order stimuli reaction time measures are highly sensitive to mild traumatic brain injuries. *J Neurotrauma*. 2016;33:242–253.

25. Patel R, Ciuffreda KJ, Tannen B, Kapoor N. Elevated coherent motion thresholds in mild traumatic brain injury. *Optometry*. 2011;82:284–289.

26. Spiegel DP, Reynaud A, Ruiz T, Laguë-Beauvais M, Hess R, Farivar R. First- and second-order contrast sensitivity functions reveal disrupted visual processing following mild traumatic brain injury. *Vision Res*. 2016;122:43–50.

27. Spiegel DP, Laguë-Beauvais M, Sharma G, Farivar R. Inter-hemispheric wave propagation failures in traumatic brain injury are indicative of callosal damage. *Vision Res*. 2015;109:38–44.

28. Hou F, Huang C-B, Lesmes L, et al. qCSF in clinical application: efficient characterization and classification of contrast sensitivity functions in amblyopia. *Invest Ophthalmol Vis Sci*. 2010;51:5365–5377.

29. Lesmes LA, Lu Z-L, Baek J, Albright TD. Bayesian adaptive estimation of the contrast sensitivity function: the quick CSF method. *J Vis*. 2010;10(3):17.

30. Reynaud A, Gao Y, Hess RF. A normative dataset on human global stereopsis using the quick Disparity Sensitivity Function (qDSF). *Vision Res*. 2015;113:97–103.

31. Giovagnoli AR, Del Pesce M, Mascheroni S, Simoncelli M, Laiacona M, Capitani E. Trail making test: normative values from 287 normal adult controls. *Ital J Neurol Sci*. 1996;17:305–309.

32. Gauthier L, Dehaut F, Joanette Y. The Bells Test: a quantitative and qualitative test for visual neglect. *Int J Clin Neuropsychol*. 1989;11:49–54.

33. Ishiai S, Sugishita M, Ichikawa T, Gono S, Watabiki S. Clock-drawing test and unilateral spatial neglect. *Neurology*. 1993;43:106–110.

34. Albert J Ahumada J, Peterson HA. Luminance-model-based DCT quantization for color image compression. *Proc SPIE*. 1992;1666:365–374.

35. Watson AB, Ahumada AJ. A standard model for foveal detection of spatial contrast. *J Vis*. 2005;5(9):6.

36. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. *Optometry*. 2008;79:18–22.

37. Capó-Aponte JE, Urosevich TG, Temme LA, Tarbett AK, Sanghera NK. Visual dysfunctions and symptoms during the subacute stage of blast-induced mild traumatic brain injury. *Mil Med*. 2012;177:804–813.

38. Doble JE, Feinberg DL, Rosner MS, Rosner AJ. Identification of binocular vision dysfunction (vertical heterophoria) in traumatic brain injury patients and effects of individualized prismatic spectacle lenses in the treatment of postconcussive symptoms: a retrospective analysis. *PM R*. 2010;2:244–253.

39. Thiagarajan P, Ciuffreda KJ, Ludlam DP. Vergence dysfunction in mild traumatic brain injury (mTBI): a review. *Ophthalmic Physiol Opt*. 2011;31:456–468.

40. Inglese M, Makani S, Johnson G, et al. Diffuse axonal injury in mild traumatic brain injury: a diffusion tensor imaging study. *J Neurosurg*. 2005;103:298–303.

41. Wilson HR, Blake R, Lee SH. Dynamics of travelling waves in visual perception. *Nature*. 2001;412:907–910.