Received: 14 December 2022 | Accepted: 8 March 2023

DOI: 10.1111/dmcn.15591

ORIGINAL ARTICLE

Motion and form coherence processing in individuals with cerebral visual impairment

Lotfi B. Merabet[1] | **Claire E. Manley**[1] | **Zahide Pamir**[1,2] | **Corinna M. Bauer**[3] | **Jan Skerswetat**[4] | **Peter J. Bex**[4]

[1]The Laboratory for Visual Neuroplasticity, Department of Ophthalmology, Massachusetts Eye and Ear, Harvard Medical School, Boston, MA, USA

[2]Department of Psychology, Bilkent University, Ankara, Turkey

[3]Lab for Neuroimaging and Vision Science, Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

[4]Translational Vision Lab, Department of Psychology, Northeastern University, Boston, MA, USA

Correspondence
Lotfi B. Merabet, Massachusetts Eye and Ear, Harvard Medical School, 20 Staniford Street, Boston, MA 02114, USA.
Email: lotfi_merabet@meei.harvard.edu

Funding information
National Eye Institute, Grant/Award Number: R01 EY030973 and R21 EY030587

Abstract

Aim: Using a visual psychophysical paradigm, we sought to assess motion and form coherence thresholds as indices of dorsal and ventral visual stream processing respectively, in individuals with cerebral visual impairment (CVI). We also explored potential associations between psychophysical assessments and brain lesion severity in CVI.

Method: Twenty individuals previously diagnosed with CVI (mean age = 17 years 11 months [SD 5 years 10 months]; mean Verbal IQ = 86.42 [SD 35.85]) and 30 individuals with neurotypical development (mean age = 20 years 1 month [SD 3 years 8 months]; mean Verbal IQ = 110.05 [SD 19.34]) participated in the study. In this two-group comparison, cross-sectional study design, global motion, and form pattern coherence thresholds were assessed using a computerized, generalizable, self-administrable, and response-adaptive psychophysical paradigm called FInD (Foraging Interactive D-prime).

Results: Consistent with dorsal stream dysfunction, mean global motion (but not form) coherence thresholds were significantly higher in individuals with CVI compared to controls. No statistically significant association was found between coherence thresholds and lesion severity.

Interpretation: These results suggest that the objective assessment of motion and form coherence threshold sensitivities using this psychophysical paradigm may be useful in helping to characterize perceptual deficits and the complex clinical profile of CVI.

Cerebral (or cortical) visual impairment (CVI) is a brain-based visual disorder associated with damage and/or maldevelopment of retrochiasmal visual processing areas in the absence of major ocular disease.[1,2] Associated etiologies are heterogeneous and include hypoxic–ischemic injury, trauma, infection, and genetic/metabolic disorders.[3] The clinical profile of CVI is also complex and visual deficits can include reduced visual acuity, visual field, and contrast sensitivities, as well as impaired ocular motor function.[4] However, for many individuals with CVI, higher order visual processing disorders represent the main visual deficit,[4–7] even in cases where visual acuity and visual field functions are at normal or near-normal levels.[8,9] Thus, without targeted assessment, visual perceptual deficits may be easily missed or misdiagnosed.[10,11]

It has been proposed that the visual symptom complex of CVI is consistent with dorsal stream dysfunction (DSD) in association with developmental damage affecting occipital-parietal pathways.[11,12] The dorsal visual stream is crucial for appraising and attending to elements within a visual scene, the perception of complex motion, and the visual guidance of movement. In contrast, the ventral stream (connecting occipital and temporal cortical areas) is responsible for the processing of shape, orientation, and form information as well as object and face recognition.[13,14] In the case of CVI, visual processing disorders attributed to dysfunction of the ventral stream appear less frequently.[11]

Abbreviations: CVI, cerebral visual impairment; DSD, dorsal stream dysfunction; FInD, Foraging Interactive D-prime.

The complex behavioral profile of CVI can be captured using a variety of means including careful history taking, use of structured questionnaires, as well as neuropsychological testing.[15,16] However, there remains the question as to whether the preponderance of DSD in CVI reflects the design of these assessments which may not necessarily evaluate nor disentangle dorsal and ventral processing functions in an even-handed manner.

Studies by Atkinson, Braddick, and colleagues have shown that visual processing deficits that are typically ascribed to the dorsal stream appear to be a common consequence of early neurodevelopmental damage.[17] Using a behavioral task called the 'ball in the grass',[18,19] these investigators measured relative sensitivities for global motion and form coherence signal integration, serving as indices of dorsal and ventral stream processing respectively.[18] In a variety of neurodevelopmental disorders (e.g. developmental dyslexia, Williams syndrome, autism spectrum disorder), motion processing appears to be more greatly impaired than form processing. This has led to the concept of 'dorsal stream vulnerability'.[17,18]

In this study, we developed and deployed a variant of this testing approach called 'Foraging Interactive in D-prime' (FInD)[20] that allows for relatively rapid assessment of motion and form coherence sensitivities based on psychophysical threshold functions. We then compared these indices of dorsal and ventral stream processing in individuals with CVI and neurotypical controls. Given that the motion and form coherence tasks were designed to be as similar as possible with respect to difficulty and cognitive demands, the presence of differential task performance would be highly suggestive of a selective processing deficit rather than a general impairment in global signal integration, attention, or task comprehension. As a secondary aim, we explored putative associations between motion and form coherence threshold measurements and underlying brain lesion severity (quantified from available morphometry magnetic resonance imaging [MRI]). Consistent with DSD, we hypothesized that participants with CVI would show higher motion, but similar form coherence thresholds compared to controls in association with impaired dorsal stream processing. Second, increased coherence thresholds would be positively correlated with increasing lesion severity.

What this paper adds

- In participants with cerebral visual impairment (CVI), motion (but not form) coherence thresholds were significantly higher compared to controls.
- These psychophysical results support the notion of dorsal stream dysfunction in CVI.

METHOD

Participants

Twenty individuals previously diagnosed with CVI (nine males; mean age = 17 years 11 months [SD 5 years 10 months], range = 8–31 years; mean Verbal IQ = 86.42 [SD 35.85], range = 22–148) and 30 individuals with neurotypical development (12 males; mean age = 20 years 1 month [SD 3 years 8 months], range = 11–26 years; mean Verbal IQ = 110.05 [SD 19.34], range = 74–139) participated in the study. Verbal IQ was assessed using subtests from the Wechsler Intelligence Scale for Children and Adults, Fourth Edition (specifically, the digit span, similarities, and vocabulary subtests of the Wechsler Intelligence Scale for Children, and the digit span, similarities, vocabulary, and information subtests of Wechsler Intelligence Scale for Adults to obtain an index of verbal comprehension).

All participants with CVI were previously diagnosed by eyecare professionals with extensive clinical experience working with this population. Diagnosis was based on a directed and objective assessment of visual functions (including visual acuity, contrast, visual field perimetry, color, and ocular motor functions), functional vision assessment (use of surveys, questionnaires, and activities), a thorough refractive and ocular examination, as well as an integrated review of medical history and available neuroimaging and electrophysiology records.[4,10,21] Causes of CVI were diverse and included hypoxic–ischemic injury related to preterm birth, periventricular leukomalacia, hypoxic–ischemic encephalopathy, seizure disorder, as well as genetic and metabolic disorders. Nine participants with CVI were born preterm (i.e. <37 weeks' gestation). Associated neurodevelopmental comorbidities included cerebral palsy and a history of developmental delays (according to the definition of 'slow to meet or not reaching milestones in one or more of the areas of development including communication, motor, cognition, social-emotional, or adaptive skills expected for the child's age'; https://sites.ed.gov/idea/statute-chapter-33). Best corrected binocular visual acuity ranged from 20/15 to 20/70 Snellen (−0.12 to 0.54 logMAR equivalent). All participants had visual acuities sufficient to perform the task and intact visual field function within the area corresponding to stimulus presentation, as well as sufficient motor ability to use a computer mouse or point to the screen to indicate their answer. Exclusion criteria included any evidence of oculomotor apraxia (i.e. apraxia of gaze or evidence of impaired visual orienting behavior), intraocular pathology (other than mild optic atrophy), uncorrected strabismus, as well as hemianopia or a visual field deficit corresponding to the area of testing (see Table 1 for complete participant demographic details).

Comparative controls had normal or corrected-to-normal visual acuities and no previous history of any ophthalmic (e.g. strabismus, amblyopia) or neurodevelopmental conditions.

Written informed consent was obtained from all participants and a parent/legal guardian (in the case of a minor) before data collection. The study was approved

TABLE 1 Demographics of the participants with CVI.

Participant ID	Etiology; comorbidities	Age (years)	Sex	Preterm/term	Visual acuity Snellen (OU)	Visual acuity LogMAR (OU)	Verbal IQ	Subcortical lesion ($n = 18$)	Hemispheric lesion ($n = 24$)	Global lesion ($n = 48$)
1	Seizure disorder	8	Female	Term	20/20	0.00	44			
2	PVL; CP	13	Female	Preterm	20/60	0.50	67			
3	Polymicrogyria	20	Female	Preterm	20/30	0.17	66			
4	Hypoxic-ischemic encephalopathy	23	Female	Preterm	20/40	0.30	44	2	1.5	3.5
5	Meningitis, infarct	20	Female	Term	20/40	0.30	114	4	5	10
6	Seizure disorder	15	Female	Term	20/60	0.50	63			
7	Perinatal head injury, hypoglycemia, anoxia	12	Female	Term	20/20	0.00	105	0	4	4
8	Genetic disorder	12	Female	Term	20/20	0.00	101			
9	Complication at birth	20	Female	Term	20/15	−0.12	148	1.5	4	7.5
10	PVL; CP	22	Female	Preterm	20/15	−0.12	135			
11	Decreased placental perfusion/ global developmental delay	23	Female	Term	20/70	0.54	100	1	0	1
12	Seizure disorder; focal cortical atrophy	21	Male	Term	20/40	0.30	75	2	17	19
13	Unspecified; developmental delay	22	Male	Preterm	20/25	0.10	37	0	2.5	2.5
14	Genetic disorder	18	Male	Term	20/20	0.00	120	3	1	4
15	PVL; CP	10	Male	Preterm	20/20	0.00	91	0	9	10
16	Unspecified; developmental delay	17	Male	Term	20/25	0.10		0	0	2
17	PVL; CP	16	Male	Term	20/20	0.00	94	4	11.5	16.5
18	Cystic PVL; CP	11	Male	Preterm	20/30	0.17	79	4	9	22
19	PVL; CP	25	Male	Preterm	20/25	0.10	137	8	16	26
20	PVL; CP	31	Male	Preterm	20/50	0.40	22	4	13	17

Abbreviations: CP, cerebral palsy; CVI, cerebral visual impairment; OU, oculus uterque; PVL, periventricular leukomalacia.

by the Investigative Review Board at the Massachusetts Eye and Ear in Boston, MA, USA and carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

Visual stimulus and psychophysical task

Global motion and form pattern coherence thresholds were assessed using a computerized, generalizable, self-administrable, and response-adaptive psychophysical paradigm called FInD.[20] Stimuli were presented as a 4×4 chart of 6° diameter cells, each containing a pattern (see Figure 1a). A random subset of cells contained a clockwise rotating moving (motion task) or static circular (form task) pattern and the remaining cells contained noise. The number and location of the cells containing the target stimulus were randomly generated and determined by the program as part of the thresholding procedure (see Appendix S1 for further details). Participants were asked to respond to two experimental prompts, for the motion task: 'are the dots spinning or just popping?' and for the form task: 'is the shape a spiral or just random?' A reference stimulus at 100% coherence was presented within the top left corner of the screen throughout the assessment to provide an example of the target pattern. Before commencing testing, comprehension of the task requirements was confirmed by having the participant verbally describe and/or draw in space (with their finger or hand) the general motion and shape of the reference stimulus perceived (i.e. a circular pattern) to the best of their ability. All participants were able to verbally describe and/or manually indicate the circular shape of the reference stimulus correctly before commencing the task.

Participants searched a total of three grids for the motion and three grids for the form pattern stimuli (a total of six grids, presented in alternating order; Figure 2b) and a computer mouse was used to click on any cells they determined to contain the signal pattern. To avoid any possible perceptual difficulties related to image crowding and simultanagnosia,[22,23] only one cell was revealed at a time underneath where the mouse pointer was located (all other cells remained hidden at the mean luminance background level; see Video S1 for a demonstration video of the task). A cell selected by the participant was marked by a dark circle and could be deselected by clicking on the same cell. The number of target signal cells present and the range of signal coherence levels on subsequent charts were updated on the next grid based on prior responses. Participants were given unlimited time to complete the task and were instructed to check their selections to maximize accuracy. Time taken to complete the task was also recorded. A higher coherence threshold value is indicative of poorer signal integration ability for both the motion and form pattern stimuli.

Structural imaging and lesion analysis

Structural morphometric data were available from a subset of participants with CVI ($n = 14$). A 3D T1-weighted scan (echo time [TE] = 2.9 ms, repetition time [TR] = 6.5 ms,

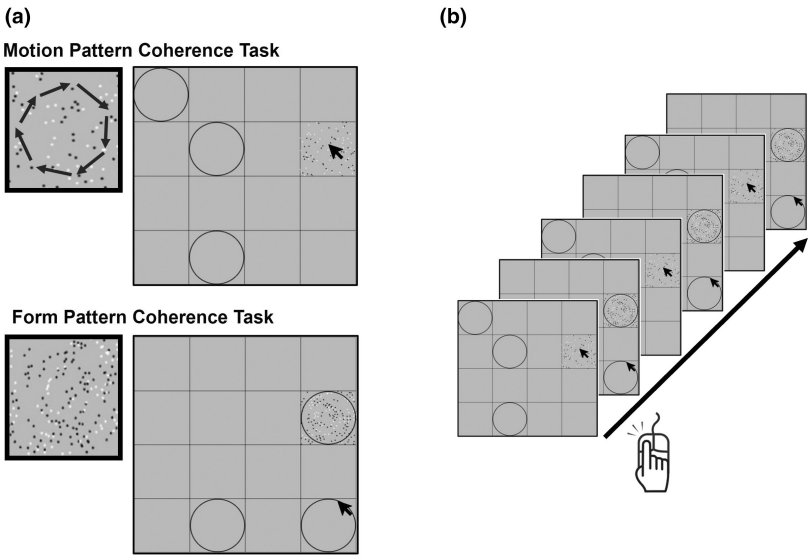

FIGURE 1 Visual stimulus and psychophysical task. (a) Schematic of the motion and form pattern coherence tasks (upper and lower panels respectively). A reference stimulus of the target pattern was presented at the top left corner of the screen (clockwise direction arrows are for illustration purposes only). Participants searched the 4×4 chart and clicked on any cells they perceived to contain a target stimulus (selected cells were indicated by a dark circle). (b) Participants viewed a total of six charts (three motion and three form) and used a mouse to make their responses. To avoid possible perceptual difficulties related to image crowding and simultanagnosia, individual cells were revealed one at a time underneath where the mouse pointer was located (see Video S1 for a demonstration of the task)

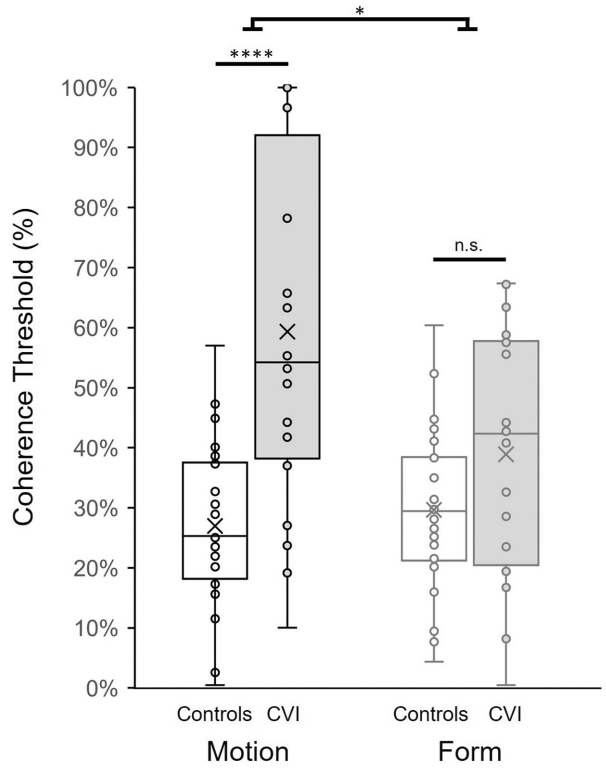

FIGURE 2 Motion and form coherence thresholds. Overall, motion coherence thresholds were significantly higher than form coherence thresholds. Participants with cerebral visual impairment (CVI) showed a significantly higher mean threshold (indicative of worse performance) for the motion, but not for the form task, as compared to controls. Results are shown as box plots with interquartile ranges as well as maximum and minimum values. Individual data (circles) are overlaid with the mean ('X') and median (line) values shown. Significance levels: *$p < 0.05$; ****$p \leq 0.001$; n.s. = non-significant

flip angle = 8°, isotropic 1 mm acquired voxel size, 0.47 x 0.47 x 1.00 mm reconstructed voxel size) and 3D-FLAIR (TE = 1650 ms, TR = 4800 ms, refocusing angle = 40°, isotropic 1.12 mm acquired voxel size, isotropic 0.74 mm reconstructed voxel size) was acquired with a 32-channel phased array head coil (3 T Philips Ingenia Elition X scanner, the Netherlands). Structural MRIs were assessed for brain lesion severity according to a reliable and validated semi-quantitative scale (see Fiori et al.[24] for complete details). Briefly, subscores from each category were summed to provide a subcortical (calculated as the sum of the basal ganglia and brainstem scores), hemispheric (calculated as the sum of the frontal, parietal, temporal, and occipital scores bilaterally), and global (sum of hemispheric, subcortical, corpus callosum, and cerebellum subscores) lesion index scores. A higher score is indicative of greater lesion severity.

Statistical analysis

Statistical analyses were carried out using SPSS Statistics package (version 24; IBM Corp., Armonk, NY, USA) and R (version 4.2.2; R Foundation for Statistical Computing, Vienna, Austria, https://www.r-project.org/). After confirmatory Shapiro–Wilk tests for normality (based on the primary outcomes of motion and form pattern coherence thresholds), a primary unadjusted analysis was carried out using a two-way repeated measures analysis of variance (ANOVA) with group (between-subject factor) and task (within-subject factor). This was followed by a secondary adjusted analysis exploring group and tasks effects while controlling for age and Verbal IQ as covariates, and an interaction analysis analyzing potential effect modification considering age and Verbal IQ as covariates. For this purpose, we employed a linear mixed model approach to account for the repeated measures design of our motion (dorsal) and form (ventral) task assessments. We included random effect for each participant acknowledging the multiple measurements and include a fixed effect for task and a fixed effect for group. In the adjusted model, we included fixed effects for age and Verbal IQ. In the age interaction model, we included multi-way interaction terms between age, task, and group, whereas in the Verbal IQ interaction model, we included multi-way interaction terms between Verbal IQ, task, and group. Time to complete the task was compared between groups using a Wilcoxon rank-sum test. Effect sizes were reported as partial η^2 and Cohen's d for ANOVA and *t*-tests respectively. Putative associations between coherence thresholds and lesion severity subscores were analyzed using non-parametric Spearman rank correlations followed by correction for multiple comparisons using false discovery rate. The false discovery rate corrected results account for six total tests of correlation coefficient: one for each of three subscores (subcortical, hemispheric, and global) for each of the two tasks (motion and form coherence). There was no missing data and no data outliers were removed as part of the analysis.

RESULTS

There was no statistically significant difference with respect to age (t[29.277] = 1.441, $p = 0.160$, d = 0.454) or the distribution of males/females ($\chi^2 = 0.1232$, $p = 0.726$) between the two groups. However, the group with CVI had a significantly lower mean Verbal IQ score compared to controls (t[26.736] = 2.568, $p = 0.016$, d = 0.838).

For the primary unadjusted analysis, the repeated measures ANOVA revealed a significant main effect of task (F[1,48] = 5.529, $p = 0.023$, $\eta_p^2 = 0.103$) and group (F[1,48] = 28.109, $p < 0.001$, $\eta_p^2 = 0.369$). There was also a significant interaction effect of task and group (F[1,48] = 9.313, $p = 0.004$, $\eta_p^2 = 0.162$). Post hoc comparisons (Bonferroni corrected) showed that the motion coherence threshold (mean = 59.33% [SD 29.54]) of participants with CVI was significantly higher than form coherence threshold (mean = 38.91% [SD 20.87]; t [19] = 2.432, $p = 0.025$, d = 0.544). No such difference was observed for the control

group (mean motion threshold = 26.99% [SD 12.75]; mean form threshold = 29.64% [SD 12.64]; t[29] = 0.998, $p = 0.326$). Performance on the motion coherence task was significantly different between the two groups (t[23.775] = 4.618, $p < 0.001$, d = 1.54, mean difference = 32.34%, 95% confidence interval [CI]: 17.88, 46.80), while no significant difference was observed for the form threshold task (t[28.313] = 1.781, $p = 0.086$) (Figure 2).

For the secondary adjusted analysis, we used a linear mixed model with age and Verbal IQ included as covariates, and random intercepts for each participant. In the adjusted model controlling for age and Verbal IQ, the effect of group ($\beta = 0.345$, $p < 0.001$, standard error = 0.062, CI: 0.224, 0.466) and the interaction effect of group and task ($\beta = -0.226$, $p = 0.003$, standard error = 0.084, CI: −0.428, −0.103) remained statistically significant, while the task effect was not significant ($\beta = 0.044$, $p = 0.443$, standard error = 0.057, CI: −0.067, 0.155). Holding group and task constant, Verbal IQ showed no significant association with coherence threshold ($\beta = -0.010$, $p = 0.197$, standard error = 0.008, CI: −0.025, 0.005), while age did show a statistically significant association ($\beta = 0.047$, $p = 0.035$, standard error = 0.021, CI: 0.005, 0.088). A subsequent interaction analysis revealed that interactions of group and task with age controlling for Verbal IQ were not statistically significant (overall likelihood-ratio test $p = 0.149$), while the corresponding overall test for interactions with Verbal IQ while controlling for age were statistically significant ($p = 0.026$). However, upon further visual inspection the estimated group differences in motion (dorsal) task threshold remained large in absolute terms across values of Verbal IQ, decreasing only slightly as Verbal IQ increased. Moreover, while the estimated group difference in form (ventral) task threshold increased for larger Verbal IQ values, this apparent relationship appeared to be driven by an outlying participant in the group with CVI with both the highest Verbal IQ score and largest form (ventral) task threshold value overall. Based on these analyses, there does not appear to be evidence of confounding or clinically meaningful effect modification of the group differences by either age or Verbal IQ.

The mean time taken to complete the task did not differ significantly between the two groups (CVI mean = 388.51 s, interquartile range [IQR] = 185.11; control mean = 326.58 s, IQR = 307.22; S = 563.0, z = 1.039, $p = 0.298$).

Finally, we explored putative associations between motion and form coherence thresholds and lesion severity as indexed by subcortical, hemispheric, and global lesion indices.[24] Spearman rank correlations (false discovery rate corrected) did not reveal a significant association for both motion and form coherence threshold values and lesion severity across all subscores (motion threshold vs subcortical [rho = 0.35, $p = 0.22$], hemispheric [rho = 0.002, $p = 0.99$], global [rho = −0.05, $p = 0.86$]; form threshold vs subcortical [rho = 0.25, $p = 0.4$], hemispheric [rho = 0.51, $p = 0.06$], global [rho = 0.51, $p = 0.06$]).

DISCUSSION

In this study, we assessed motion and form coherence serving as indices for dorsal and ventral stream processing sensitivities respectively. Consistent with previous reports describing DSD in CVI,[11,12] we found that mean global motion coherence thresholds were significantly higher in CVI compared to controls. In contrast, global form coherence thresholds were not significantly different between both groups. Based on the adjusted analysis, there does not appear to be evidence of confounding or clinically meaningful effect modification of the group differences by either age or Verbal IQ. Time taken to complete the task was also not significantly different between the CVI and control groups. An exploratory analysis of available MRI data from the group with CVI did not reveal any statistically significant association between motion and form coherence thresholds and lesion severity.

Previous studies have demonstrated that quantifying signal/noise thresholds with respect to global motion and form signals can serve as a useful index to assess dorsal and ventral visual stream processing sensitivities respectively. Motion and form coherence thresholds are largely similar in adults and reach adult levels for form coherence around 7 to 10 years of age for typically developing children.[18] In contrast, global motion sensitivity shows a slower developmental trajectory and reaches adult levels around 8 to 12 years.[18] There is also mounting evidence that the development and maturation of global motion perception is both delayed and more variable across a diverse range of neurodevelopmental disorders (e.g. developmental dyslexia, Williams syndrome, autism spectrum disorder) providing support for the concept of 'dorsal stream vulnerability'.[17,18] In the context of CVI, the selective deficit we observed with respect to global motion signal integration is in line with the notion of DSD.[11,12] This is despite the relatively heterogenous sample population tested here with respect to age, Verbal IQ, and lesion severity. Further studies with targeted recruitment and larger samples are needed to further characterize motion and form coherence processing with respect to specific etiologies of CVI.

The relationship between visual processing deficits and underlying structural and functional changes in CVI remains to be clearly established. Indeed, characterizing the neurophysiological basis of visual dysfunctions in CVI remains challenging given that early neurological and developmental damage to cerebral structures across individuals is highly variable with respect to cause, localization, and severity. Tinelli et al. recently explored the relationship between visual function impairments and brain lesion severity in a sample of children with bilateral cerebral palsy associated with periventricular leukomalacia.[25] The authors found that greater brain lesion severity (using the same semi-quantitative MRI scoring scale used in this study) was strongly correlated with greater levels of visual dysfunction. Specifically, visual acuity, visual field, stereopsis, and

color perception were all found to be impaired when cortical damage was present, while subcortical brain damage was associated with deficits with ocular motor functions (i.e. fixation and saccades).[25] In this study, we did not observe a statistically significant association with either motion or form coherence thresholds and all indices of lesion severity. The lack of a significant association is likely related to the specific nature of our task assessment as well as the relatively heterogeneous and small sample size of our population with CVI. Furthermore, the semi-quantitative method of scoring lesion severity used in this study may not be sufficiently sensitive compared to other quantitative lesion segmentation approaches. It is possible that greater lesion severity would be associated with greater impairments in both motion and form coherence processing. However, it is also important to differentiate changes in lesion severity that are specific to dorsal and ventral related areas. Furthermore, greater lesion severity may also be associated with other sensorimotor and cognitive deficits that may further confound observations with performance. Finally, it is important to note that while an association between visual processing impairments and brain injury is often suspected in CVI, evidence of observable structural damage is not always apparent.[5]

In the case of CVI, it is crucial to carefully assess both dorsal and ventral stream related functional abilities in a comprehensive and even-handed manner as they may be impacted differently, particularly at the individual level. At the same time, while it may be useful to conceptually separate visual processing impairments according to the classic two-stream organization, it is important to note that the dorsal and ventral streams do not function independently of one another but, rather, are closely interlinked. This makes the functional roles of the two streams difficult to disentangle, especially when considering everyday tasks (see Grinter et al.[26] and Boot et al.[5] for further discussion).

The main advantage of the psychophysical approach used in this study is that assessing selective threshold sensitivities can be carried out relatively quickly with a single testing platform. This is also supported by the fact that time to complete the task was comparable in both the CVI and control groups, and despite differences in Verbal IQ levels. As the motion and form assessments were designed to be as similar as possible, the presence of a selective deficit with regard to motion signal integration is consistent with impaired functioning along the dorsal stream rather than a generalized impairment in signal integration, attention, or task comprehension. As currently designed, the task requires a manual and/or verbal response, visual acuity level sufficient to discriminate the stimulus elements, as well as a sufficient level of cognitive functioning to confirm comprehension of the visual stimuli and task requirements. Thus, this task may not be appropriate for the broader population with CVI. Future studies will need to confirm our findings with a larger study sample and with task design modifications that can accommodate a wider range of visual and motor functioning, as well as cognitive abilities. Finally, large-scale longitudinal studies relating psychophysical thresholds, functional clinical assessments, and structural differences revealed by advanced neuroimaging methodologies should provide convergent evidence to help uncover the complex neurophysiological basis of CVI.

ACKNOWLEDGMENTS

The authors would like to thank the participants and their families for their participation in this study. This work was supported by grants from the National Institutes of Health/National Eye Institute (R21 EY030587 and R01 EY030973) to LBM. JS and PJB are founders of PerZeption Inc. A patent has been filled (pending status at the time of resubmission) for the FInD method, including FInD Motion and Pattern coherence, and is owned by Northeastern University, Boston, USA. This work was conducted with consultation regarding the statistical analyses from Harrison Reeder, through support from Harvard Catalyst, The Harvard Clinical and Translational Science Center (National Center for Advancing Translational Sciences, National Institutes of Health Award UL1 TR002541), and financial contributions from Harvard University and its affiliated academic healthcare centers.

DATA AVAILABILITY STATEMENT

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

ORCID


Lotfi B. Merabet https://orcid.org/0000-0002-8094-9536

REFERENCES

1. Dutton GN, Lueck AH. Impairment of Vision Due to Damage to the Brain. In: Dutton AHLaGN, editor. Vision and the Brain: Understanding Cerebral Visual Impairment in Children: AFB Press New York; 2015. p. 3–20.
2. Sakki HEA, Dale NJ, Sargent J, Perez-Roche T, Bowman R. Is there consensus in defining childhood cerebral visual impairment? A systematic review of terminology and definitions. Br J Ophthalmol. 2018;102(4):424–32.
3. Hoyt CS. Visual function in the brain-damaged child. Eye (Lond). 2003;17(3):369–84.
4. Fazzi E, Signorini SG, Bova SM, La Piana R, Ondei P, Bertone C, et al. Spectrum of visual disorders in children with cerebral visual impairment. J Child Neurol. 2007;22(3):294–301.
5. Boot FH, Pel JJ, van der Steen J, Evenhuis HM. Cerebral Visual Impairment: which perceptive visual dysfunctions can be expected in children with brain damage? A systematic review. Res Dev Disabil. 2010;31(6):1149–59.
6. Ortibus E, Lagae L, Casteels I, Demaerel P, Stiers P. Assessment of cerebral visual impairment with the L94 visual perceptual battery: clinical value and correlation with MRI findings. Dev Med Child Neurol. 2009;51(3):209–17.
7. Philip SS, Dutton GN. Identifying and characterising cerebral visual impairment in children: a review. Clin Exp Optom. 2014;97(3):196–208.
8. van Genderen M, Dekker M, Pilon F, Bals I. Diagnosing cerebral visual impairment in children with good visual acuity. Strabismus. 2012;20(2):78–83.
9. Williams C, Northstone K, Sabates R, Feinstein L, Emond A, Dutton GN. Visual perceptual difficulties and under-achievement at school in a large community-based sample of children. PLoS One. 2011;6(3):e14772.

10. Chandna A, Ghahghaei S, Foster S, Kumar R. Higher Visual Function Deficits in Children With Cerebral Visual Impairment and Good Visual Acuity. Front Hum Neurosci. 2021;15:711873.
11. Dutton GN. 'Dorsal stream dysfunction' and 'dorsal stream dysfunction plus': a potential classification for perceptual visual impairment in the context of cerebral visual impairment? Dev Med Child Neurol. 2009;51(3):170–2.
12. Macintyre-Beon C, Ibrahim H, Hay I, Cockburn D, Calvert J, Dutton GN, et al. Dorsal Stream Dysfunction in Children. A Review and an Approach to Diagnosis and Management. Current Pediatric Reviews. 2010;6(3):166–82.
13. Goodale MA. Separate visual systems for perception and action: a framework for understanding cortical visual impairment. Dev Med Child Neurol. 2013;55 Suppl 4:9–12.
14. Haxby JV, Grady CL, Horwitz B, Ungerleider LG, Mishkin M, Carson RE, et al. Dissociation of object and spatial visual processing pathways in human extrastriate cortex. Proc Natl Acad Sci U S A. 1991;88(5):1621–5.
15. Chang MY, Borchert MS. Methods of visual assessment in children with cortical visual impairment. Curr Opin Neurol. 2021;34(1):89–96.
16. McConnell EL, Saunders KJ, Little JA. What assessments are currently used to investigate and diagnose cerebral visual impairment (CVI) in children? A systematic review. Ophthalmic Physiol Opt. 2021;41(2):224–44.
17. Braddick O, Atkinson J, Wattam-Bell J. Normal and anomalous development of visual motion processing: motion coherence and 'dorsal-stream vulnerability'. Neuropsychologia. 2003;41(13):1769–84.
18. Atkinson J. The Davida Teller Award Lecture, 2016: Visual Brain Development: A review of 'Dorsal Stream Vulnerability'-motion, mathematics, amblyopia, actions, and attention. J Vis. 2017;17(3):26.
19. Atkinson J, Braddick O. Dorsal stream vulnerability and autistic disorders: the importance of comparative studies of form and motion coherence in typically developing children and children with developmental disorders. CAHIERS DE PSYCHOLOGIE COGNITIVE-CURRENT PSYCHOLOGY OF COGNITION. 2005;23(1–2):49–58.
20. Bex PJ, Skerswetat J. FInD - Foraging Interactive D-prime, a rapid and easy general method for visual function measurement. Vision Sciences Society Annual Meeting Abstract Journal of Vision 2021.
21. Boonstra FN, Bosch DGM, Geldof CJA, Stellingwerf C, Porro G. The Multidisciplinary Guidelines for Diagnosis and Referral in Cerebral Visual Impairment. Front Hum Neurosci. 2022;16:727565.
22. Das M, Bennett DM, Dutton GN. Visual attention as an important visual function: an outline of manifestations, diagnosis and management of impaired visual attention. Br J Ophthalmol. 2007;91(11):1556–60.
23. Dutton GN. Cognitive vision, its disorders and differential diagnosis in adults and children: knowing where and what things are. Eye (Lond). 2003;17(3):289–304.
24. Fiori S, Cioni G, Klingels K, Ortibus E, Van Gestel L, Rose S, et al. Reliability of a novel, semi-quantitative scale for classification of structural brain magnetic resonance imaging in children with cerebral palsy. Dev Med Child Neurol. 2014;56(9):839–45.
25. Tinelli F, Guzzetta A, Purpura G, Pasquariello R, Cioni G, Fiori S. Structural brain damage and visual disorders in children with cerebral palsy due to periventricular leukomalacia. Neuroimage Clin. 2020;28:102430.
26. Grinter EJ, Maybery MT, Badcock DR. Vision in developmental disorders: is there a dorsal stream deficit? Brain Res Bull. 2010;82(3–4):147–60.

SUPPORTING INFORMATION

The following additional material may be found online:
Appendix S1: Visual stimulus and psychophysical task details.
Video S1: Motion and form coherence task demo with cursor.

How to cite this article: Merabet LB, Manley CE, Pamir Z, Bauer CM, Skerswetat J, Bex PJ. Motion and form coherence processing in individuals with cerebral visual impairment. Dev Med Child Neurol. 2023;65:1379–1386. https://doi.org/10.1111/dmcn.15591