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Effects of speed, age, and amblyopia on the perception of motion-defined form

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ABSTRACT

We determined the effect of dot speed on the typical and atypical development of motion-defined form perception. Monocular motion coherence thresholds for orientation discrimination of motion-defined rectangles were determined at slow (0.1 deg/s), medium (0.9 deg/s) and fast (5.0 deg/s) dot speeds. First we examined typical development from age 4 to 31 years. We found that performance was most immature at the slow speed and in the youngest group of children (4–6 years). Next we measured motion-defined form perception in the amblyopic and fellow eyes of patients with amblyopia. Deficits were found in both eyes and were most pronounced at the slow speed. These results demonstrate the importance of dot speed to the development of motion-defined form perception. Implications regarding sensitive periods and the neural correlates of motion-defined form perception are discussed.

1. Introduction

The developing visual system is vulnerable to damage during sensitive periods of neural plasticity. The sensitive period for typical development can be shorter or longer than the sensitive period for damage, and there are different sensitive periods for different aspects of vision (reviewed in Lewis & Maurer, 2005). The sensitive period for damage to different aspects of vision can be assessed psychophysically by studying patients with amblyopia due to uncorrected ocular misalignment (strabismus) and/or unequal refractive error (anisometropia) during early childhood. The defining characteristic of unilateral amblyopia is poor visual acuity in one eye (amblyopic eye) while the other eye (fellow eye) retains normal visual acuity. A variety of other visual deficits have been identified, including both form (low-contrast acuity, contrast sensitivity, positional acuity, spatial localization: reviewed in Levi, 1991; spatial distortion: Barrett et al., 2003; contour integration: Kovacs et al., 2000; motion-defined form: Giaschi, Regan, Kraft & Hong, 1992; Ho et al., 2005; Wang, Ho, & Giaschi, 2007) and motion perception (motion aftereffects: Hess, Demanins, & Bex, 1997; oscillatory movement displacement: Buckingham et al., 1991; Kelly & Buckingham, 1998; global motion: Aaen-Stockdale & Hess, 2008; Aaen-Stockdale, Ledgeway, & Hess, 2007; Ellemberg et al., 2002; Simmers et al., 2003, 2006; maximum motion displacement: Ho & Giaschi, 2007; Ho et al., 2005; attentive tracking: Ho et al., 2006), but the nature of the underlying neural abnormalities remains largely unknown.

It has become common to interpret typical and atypical visual development in terms of the underlying cortical ventral and dorsal streams. The hypothesis of dorsal stream vulnerability has been put forward to account for the prevalence of deficits in global motion perception in developmental disorders such as autism, Williams syndrome, Fragile X, developmental dyslexia and developmental dyspraxia (reviewed in Braddick, Atkinson, & Wattam-Bell, 2003). Some of these disorders, however, show ventral stream deficits as well (reviewed in Grinter, Mayberry, & Badcock, 2010), and Bertone and colleagues (Bertone & Faubert, 2005; Bertone et al., 2003, 2008) caution that stimulus complexity (first-order versus second-order stimulus properties), rather than dorsal stream vulnerability, may be driving these deficits. Our finding of a deficit in motion-defined (second order) form perception in amblyopia, in patients with normal luminance-defined (first order) form perception (Giaschi et al., 1992), is support for this suggestion. However, motion-defined form has also been used to study the intactness of low-level motion mechanisms (Battelli et al., 2001) and the integrity of dorsal stream function (Downie et al., 2003; Felmingham & Jakobsen, 1995) in clinical populations, as well as the onset of direction-selective motion perception in human infants (Wattam-Bell, 1996).

Neuroimaging (Hotson & Anand, 1999; Malach et al., 1995; Orban et al., 1995; Schoenfeld et al., 2003) and human lesion studies (Blanke et al., 2007; Regan et al., 1992) have confirmed the importance of both ventral (linked to form) and dorsal (linked to motion) streams to the processing of motion-defined form. Although the dorsal/ventral dichotomy has fueled a great deal of research and improved our understanding of visual processing, the view that motion processing relies exclusively on the dorsal stream while form processing relies exclusively on the ventral stream is

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probably too simplistic (e.g. Farivar, 2009), and the cortical pathways show considerable cross-talk (Merigan & Maunsell, 1993).

The ability to detect motion contrast appears as early as 2–4 months of age (Johnson & Aslin, 1998; Johnson & Mason, 2002; Kaufmann-Hayoz, Kaufmann, & Stucki, 1986; Wattam-Bell, 1996). The age at which performance improves to adult levels is well into the school-age years, although maturation appears to depend heavily on stimulus parameters. Motion-defined form perception matures by 7 years of age when minimum speed thresholds are measured (Giaschi & Regan, 1997; Parrish et al., 2005), and by 10 or 15 years of age when coherence thresholds are measured at 6 deg/s (Gunn et al., 2002) or 1.3 deg/s (Schrauf, Wist, & Ehrenstein, 1999), respectively. We have suggested that it is mainly the form perception aspects of this task that are maturing during the school-age years and that are susceptible to disruption by amblyopia, based on several pieces of evidence. Shape identification matures earlier for motion contrast than for texture-contrast (Parrish et al., 2005). Fellow-eye deficits in motion-defined form are more prevalent than deficits in global motion and maximum motion displacement in children with amblyopia (Ho et al., 2005). Deficits in motion-defined and texture-defined form perception tend to occur together, and in the absence of global motion deficits, in children with amblyopia (Wang, Ho, & Giaschi, 2007). The effect of dot speed, however, on the development of motion-defined form perception has not been assessed directly. Speed-tuned development would indicate the importance of the motion aspects of this task.

The development of motion perception appears to depend on speed, with several aspects showing more immaturity for slow speeds than for fast speeds (Ahmed et al., 2005; Aslin & Shea, 1990; Ellemberg et al., 2004; Kaufmann, 1995; Volkmann & Dobson, 1976). For example, global motion direction discrimination was more immature in 5-year-olds for slower speeds (1.5 deg/s) than for faster speeds (6 or 9 deg/s). This immaturity was observed for both first-order and second-order stimuli, but was greater for second-order motion at the slowest speed (Ellemberg et al., 2004). In addition, the sensitive period for the disruption of fast global motion was found to be very short, based on performance in children deprived of normal vision by cataracts (Ellemberg et al., 2002). The effect of speed on visual deficits in strabismic or anisometropic amblyopia has not been assessed.

The purpose of the current study was to determine the effect of dot speed on the typical and atypical development of motion-defined form perception. Our previous technique of measuring minimum speed thresholds (Giaschi et al., 1992; Giaschi & Regan, 1997; Ho et al., 2005; Parrish et al., 2005; Wang, Ho, & Giaschi, 2007) masked the effect of speed on this aspect of vision. Instead, in the current study, coherence thresholds were measured for motion-defined form discrimination at three fixed speeds. This also allowed for easier comparison with previous studies (Annaz et al., 2010; Gunn et al., 2002; Reiss, Hoffman, & Landau, 2005; Schrauf, Wist, & Ehrenstein, 1999). Our first goal was to typify the normal development, and maturation to adult levels, of motion-defined form perception as a function of motion speed during childhood. We next used the speed tuning of the motion-defined form deficit in amblyopia to explore how development of the visual pathways is disrupted by abnormal visual stimulation.

2. Experiment 1: the affect of age

2.1. Methods

All research was approved by the University of British Columbia's Clinical Research Ethics Board and the Children's and Woman's Health Center of BC Research Ethics Board.

2.1.1. Participants

A total of 68 volunteers were recruited via posters from the community to participate in this experiment. They earned $10 for their participation. All participants gave written and informed consent after the test procedures had been explained. Signed consent for children was obtained from the accompanying parent. Verbal or written assent was obtained from each child. The participants were divided into four groups of unequal size: 4–6 year-olds ($N = 22$), 7–10 year-olds ($N = 15$), 11–17 year-olds ($N = 15$), and 18–31 year-olds ($N = 16$). Visual acuity (VA) was assessed using the Regan high-contrast letter chart (Regan, 1988). For a few children in the youngest age group who did not know the alphabet reliably, the Lighthouse picture chart was used (Lighthouse Low Vision Products). For 4–6 year old participants, best-corrected decimal VA was required to be at least 0.67. For participants 7 years and older, best-corrected decimal VA was required to be at least 1.0 (Jose & Atcherson, 1977). Stereoacuity was assessed using the Randot Circles test (Stereo Optical Co. Inc.) and was used as a measure of binocularity. For all age groups, a stereoacuity of 70 s of arc or lower was required for inclusion (Rutstein & Daum, 1998). All participants were free from amblyopia or other eye diseases based on parental report.

2.1.2. Apparatus

The tasks were generated on a Macintosh Powerbook G4 with a 17 in. Sony Trinitron monitor with a resolution of 1024 (horizontal) × 768 (vertical) pixels and a screen refresh rate of 75 Hz. Responses were given on a Mac Gravis game pad.

2.1.3. Stimulus

A randomly generated array of white square dots (98.5 cd/m^2) of 0.022° length was presented on a black background (1.0 cd/m^2) with a dot density of 8% 170 dots/deg^2. The array of dots formed a rectangular shape that was only visible because the signal dots within the shape moved in one direction while the signal dots outside the shape moved in the opposite direction at the same speed (see Fig. 1). The shape, a vertical or horizontal rectangle, remained stationary. The motion-defined rectangle had dimensions of 2 × 1° within the 7.45 (horizontal) × 5.53° (vertical) background dot display.

Each trial consisted of 48 monitor refreshes at a refresh rate of 75 Hz giving a total stimulus duration of 640 ms. The dots moved at one of three speeds, 0.1 deg/s (slow), 0.9 deg/s (medium) or 5.0 deg/s (fast). A proportion of dots inside the rectangle moved coherently in the same vertical direction (signal dots); the direction of coherent motion alternated randomly (up or down) across trials. Signal dots outside of the rectangle moved in the opposite direction with the same level of coherence. The remaining dots

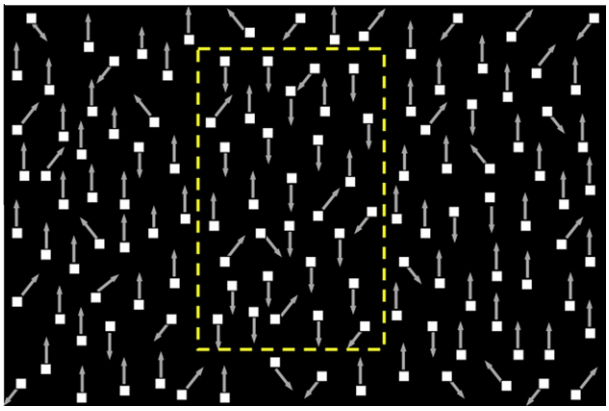

Fig. 1. Motion-defined form stimulus. The motion-defined form task representing a 'tall' rectangle in the center of the display. The signal dots within the bounds of the rectangle move in one direction while the signal dots in the surrounding area move in the opposite direction at the same speed.

inside and outside the rectangle moved in random directions at the same speed (noise dots). Each dot had a limited lifetime. Each dot had a 3% chance of appearing black, i.e. disappearing, on any given frame. Thus, the majority of extinct dots reappeared in the following frame at new coordinates in accordance with its previous path of motion. If any pixel of a dot overlapped with the border of the MD rectangle the entire dot disappeared.

2.1.4. Procedure

All testing was completed in one session that lasted approximately 1 h. The tasks were completed monocularly using the eye with the best visual acuity. The other eye was occluded with an opaque eye patch. The test environment was dimly lit with diffuse light to prevent glare on the computer screen. Participants sat 2.5 m from the monitor. The participant's task was to indicate the orientation of the rectangle, horizontal or vertical, by pressing the corresponding button on the gamepad.

To ensure that the participants understood the task and could correctly discriminate the orientation of the rectangle, the experiment began with a block of five trials with a motion coherence of 0.8 (0.8 signal dots, 0.2 noise dots). Performance accuracy of 80% on this task was required for the experiment to proceed. Next, the proportion of coherently moving dots was manipulated in a two-down, one-up staircase in which coherence level was decreased when two successive trials were correct or increased by the same step size when one trial was incorrect. A run started at a motion coherence of 1.0 with an initial step size of 0.1. After the third response reversal, step size was halved in both directions at each reversal. Three conditions were tested in each participant: slow, medium and fast. Staircases for each speed condition were completed separately (i.e. staircases were not interleaved). Each condition began with a practice staircase of 20 trials, followed by a longer staircase with a minimum of 40 trials and a stopping criterion of 10 reversals or 50 trials. The condition order was counterbalanced across participants and breaks were taken after each staircase to prevent fatigue.

2.1.5. Measures

Coherence thresholds for discriminating the orientation of the motion-defined rectangle were used as a measure of the participant's perceptual ability. Thresholds were determined by fitting a Weibull function to the data for each participant using a maximum-likelihood minimization procedure (Watson, 1979). Threshold was defined as the point of maximum slope on the fitted curve, which occurs at 82% correct in a two-alternative forced-choice procedure (Strasburger, 2001). A χ^2 test ($p > .05$) confirmed that the Weibull function accurately fit the data for each participant.

2.2. Results

The motion coherence scores were analyzed with a 4 between (age group) × 3 within (speed) mixed-model ANOVA. The ANOVA showed a significant age by speed interaction, $F(4.33,92.46) = 2.54$, $p = .04$ (degrees of freedom adjusted with Greenhouse–Geisser method due to non-spherical data; see Fig. 2). The effect size for this interaction was medium ($f = 0.31$; Cohen, 1977).

The significant interaction was followed up with simple effects analyses, with the age groups analyzed at each speed using univariate ANOVAs. The effect of age was significant at the slow speed, $F(3,64) = 15.17$, $p < .001$, and the medium speed, $F(3,64) = 2.94$, $p = .04$, but not at the fast speed, $F(3,64) = 1.48$, $p = .23$. The size of the age effect was large at slow speed ($f = 0.84$; Cohen, 1977), medium-large at medium speed ($f = 0.37$; Cohen, 1977), and medium at fast speed ($f = 0.26$; Cohen, 1977). Post-hoc pair-wise comparisons were conducted using a Bonferroni correction to maintain an overall α-level of .05. At the slow speed, 4–6 years olds performed worse than each of the three older age groups (all $p < .001$), while there were no differences between the older age groups (all $p > .15$). At the medium speed, 4–6 year olds performed worse than the 18–31 years olds only ($p = .005$).

We next expressed the mean coherence threshold for each child group as a threshold elevation relative to the mean coherence threshold for the adult group (Table 1). Threshold elevation was highest at the slow speed, particularly in the youngest group of children.

2.3. Discussion

The main objective of Experiment 1 was to evaluate motion coherence thresholds for motion-defined orientation discrimination as a function of speed and age. We observed a significant interaction between speed and age, suggesting different developmental trajectories at different motion speeds. Performance improved to adult levels by 7 years of age under the slowest speed condition whereas age related changes at the medium speed were subtle, gradually improving across the age range tested. No clear effect of age was observed at the fast speed. In our previous work, we found maturation to adult speed thresholds of 0.1 deg/s by 7 years of age (Giaschi & Regan, 1997; Parrish et al., 2005). This suggests that our previous technique of measuring minimum speed thresholds with coherence fixed at 100% and our current technique of measuring coherence thresholds with speed fixed at 0.1 deg/s are tapping into a similar developmental process.

Expressing the coherence thresholds as threshold elevation ratios allowed us to compare the relative immaturity as a function of speed and age group. Performance was most immature at the

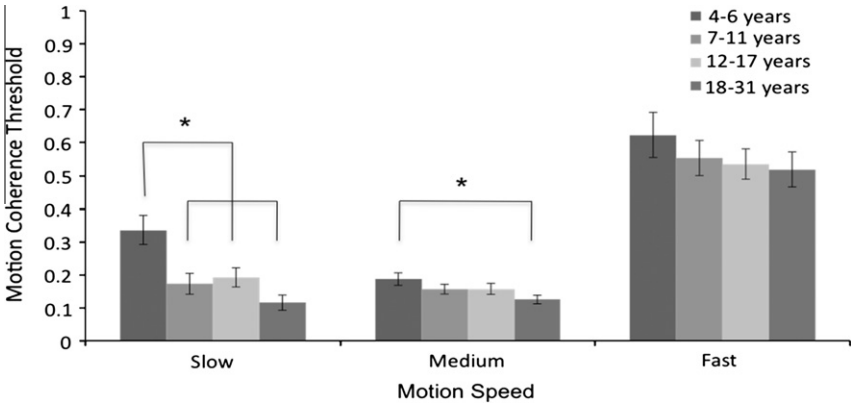

Fig. 2. The effect of age on motion-defined form perception. Mean motion coherence thresholds, showing a significant interaction between age and speed ($p = .04$). Error bars represent ±1 standard error. *Indicates a significant mean difference at an experiment-wide α of .05.

Table 1
Threshold elevation ratio relative to adult group mean.

Group	Speed		
	Slow	Medium	Fast
4–6 Years	3.2×	1.4×	1.3×
7–11 Years	1.5×	1.2×	1.1×
12–17 Years	1.5×	1.2×	1.1×
Adult	–	–	–

slow speed, and in the youngest group of children. These results suggest that motion-defined form perception is more immature at age 4–6 years for slower speeds than for faster speeds. A similar result has been reported for global motion perception (Ellemberg et al., 2004). In addition, maturation to adult levels appears to occur sooner during childhood at the fastest speed, possibly before 4 years of age.

It could be argued that the absence of an age effect at the fast speed represents a ceiling effect because thresholds were highest under this condition. Subjects did not find it difficult to perceive the direction of motion, given that a speed of 5.0 deg/s (or a relative speed of 10 deg/s) is far from the upper limit for human global motion perception (Van de Grind, Van Doorn, & Koenderink, 1983). Rather, the motion-defined edges became less salient at lower coherence levels. However, the mean adult threshold at the fast speed was 0.5, leaving room for a threshold elevation of up to two times.

The speed effect we observed may partially explain some previous inconsistencies about the maturation of motion-defined form. Schrauf, Wist, and Ehrenstein (1999) found significant improvement until 15 years of age at a slow speed (1.3 deg/s). Gunn et al. (2002) found slight improvement until 10 years of age at a fast speed (6 deg/s). Additional stimulus differences such as target size and shape, dot density, dot lifetime, interframe dot displacement and background dot motion must also be important, but this requires further investigation.

If slow and fast motion-defined form perception typically develops at different rates, then the susceptibility of this aspect of perception to damage during childhood may also depend on speed. In Experiment 2 we tested this hypothesis by measuring motion-defined form perception at different speeds in patients with amblyopia.

3. Experiment 2: the effect of amblyopia

3.1. Methods

The apparatus and stimuli were identical to those used in Experiment 1.

3.1.1. Participants

The patient group consisted of 12 participants who had been treated for amblyopia but were no longer undergoing occlusion therapy. Patients ranged in age from 7 to 25 years ($M = 13.18$, $SD = 6.43$). The clinical details of the patients with amblyopia are summarized in Table 2. The participants were referred from the Department of Ophthalmology at the Children's and Women's Health Centre of British Columbia. The control group consisted of the 46 participants aged 7–31 years ($M = 15.56$, $SD = 6.00$) from Experiment 1.

3.1.2. Procedure

Both the amblyopic and fellow eyes were tested at three speeds giving a total of six conditions. Testing lasted a total of 2 h and the order of testing was counterbalanced across patients. The procedure was identical to Experiment 1 in all other respects.

3.2. Results

3.2.1. Individual participant analysis

Individual patient coherence thresholds (Table 3) were compared to the control group data from Experiment 1. We expected abnormal performance to occur only at one tail of the normal distribution corresponding to higher coherence levels. A one-tailed 95% confidence interval suggests that 5 out of 100 people performing the task would obtain scores that differ by at least 1.64 standard deviations from the population mean. Abnormal performance was defined as a coherence threshold greater than 1.64 standard deviations away from the mean threshold obtained by the patient's age-matched control group. The prevalence of abnormal coherence thresholds in control participants did not differ significantly from that expected based on 95% confidence limits [$\chi^2(2) = 0.90$, $p = .64$]. This confirmed that comparisons were being made to control participants that indeed showed normal performance. Due to the extended length of testing in the amblyopic group three patients did not complete the task under all six conditions due to fatigue or limited time availability (see Table 3).

Eight out of 12 amblyopic participants (67%) showed abnormal performance in the amblyopic eye under at least one speed condition. Of these, 8 out of 11 (73%) were deficient at the slow speed, 1 out of 10 (10%) was deficient at the medium speed, and 1 out of 12 (8%) was deficient at the fast speed. In the fellow eye, a total of 7 out of 12 patients (58%) were deficient. Five out of 11 (45%) fellow eyes were deficient at the slow speed, 3 out of 10 (30%) at the medium speed, and 1 out of 12 (8%) at the fast speed. There were no obvious differences between amblyopic subtypes with respect to the overall prevalence of deficits or the distribution of deficits

Table 2
Summary details for the participants with amblyopia.

Patient	Diagnosis	Age (years)	Decimal visual acuity (amblyopic eye)	Decimal visual acuity (fellow eye)	Stereo acuity (seconds of arc)
1	A	25.89	0.8	1.2	30
2	A	16.92	1.07	1.2	20
3	A	9.33	0.65	1.28	100
4	A	9.7	0.88	1.28	25
5	A	10.19	0.8	1	40
6	A	7.56	0.8	1.1	40
7	A	9.05	1	1	20
8	A	16.89	1.1	0.9	20
9	A	10.5	0.25	1.33	>400
10	S	8.33	0.5	1.05	30
11	S	9.06	1	1	400
12	A + S	24.73	0.3	1.25	>400

A, anisometropic amblyopia; S, strabismic amblyopia; A + S, aniso-strabismic amblyopia.

Table 3
Test data for 12 amblyopic patients.

Patient	Coherence threshold					
	Amblyopic eye			Fellow eye		
	Slow	Medium	Fast	Slow	Medium	Fast
1	0.52[a]	0.09	0.67	0.29[a]	0.21[a]	0.53
2	0.42[a]	0.10	0.33	0.37[a]	0.14	0.40
3	0.44[a]	0.23	0.48	0.15	0.27[a]	0.85[a]
4	0.35[a]	0.15	0.40	0.10	0.16	0.49
5	0.24	0.13	0.53	0.13	0.13	0.28
6	0.43[a]	0.29[a]	0.43	0.32	0.18	0.50
7	0.24	0.11	0.31	0.19	0.10	0.31
8	0.38[a]	0.19	0.53	0.25	0.20	0.40
9	0.22	0.14	0.50	0.35[a]	0.15	0.49
10	n/a	0.24	0.29	n/a	0.36[a]	0.29
11	0.74[a]	n/a	0.90[a]	0.47[a]	n/a	0.48
12	0.99[a]	n/a	0.63	0.28[a]	n/a	0.44
Mean	0.45	0.17	0.50	0.26	0.19	0.45
SD	0.23	0.07	0.17	0.11	0.08	0.15

n/a Indicates subject was not tested on that condition.
[a] Result is greater than the normal upper limit for the appropriate age group. One-tailed normal limits for MD form are set at a 1.64 standard deviation (*SD*) limit for a 95% confidence interval.

across speed conditions, but only three subjects had strabismus and none of these completed all three conditions (see Table 3).

3.2.2. Analysis of variance

In this experiment, the amblyopic group was too small to split into separate age groups for comparison to controls. Therefore, in order to test the effects of amblyopia and speed on motion-defined form perception, we utilized an analysis of covariance (ANCOVA) procedure. Age was included as a covariate in order to partial out any effect of age. Amblyopic and fellow eye analyses were conducted separately using 2 between (group) × 3 within (speed) mixed-model ANCOVAs. Control participants from the three oldest age groups in Experiment 1 were assigned randomly to either the amblyopic eye control group ($N = 23$; $M = 15.93$ years, $SD = 6.20$) or the fellow eye control group ($N = 23$; $M = 15.18$ years, $SD = 5.91$). The difference between the mean ages of the control groups was not statistically significant, $t(44) = .42$, $p = .67$. Due to the extended testing length for the participants with amblyopia, there was a higher rate of fatigue in the amblyopic group than in the controls. Participants who did not complete all three speed conditions were excluded from the analysis; a total of three cases were excluded leaving an amblyopic group of nine participants.

3.2.2.1. Fellow eye. In the fellow eye, a significant interaction between group and speed was observed, $F(1.27, 36.90) = 4.23$, $p = .04$ (degrees of freedom adjusted with Greenhouse–Geisser method due to non-spherical data; see Fig. 3). The effect size of the interaction was medium-large ($f = 0.38$; Cohen, 1977). Follow-up pair-wise comparisons with a Bonferroni correction ($\alpha = .05$) revealed higher coherence thresholds (worse performance) for fellow eyes under the slow condition, $F(1,29) = 8.05$, $p = .008$. The difference between groups was not statistically significant at the medium speed, $F(1,29) = .57$, $p = .46$, or at the fast speed, $F(1,29) = 1.32$, $p = .26$. Additionally, the interaction between age and speed was not significant, $F(1.27, 36.90) = .37$, $p = .60$, and there was no main effect of age, $F(1,29) = .54$, $p = .47$.

3.2.2.2. Amblyopic eye. In the amblyopic eye, a significant interaction between group and speed was observed, $F(1.42, 41.21) = 9.64$, $p = .001$ (degrees of freedom adjusted with Greenhouse–Geisser method due to non-spherical data; see Fig. 3). The interaction effect size was large ($f = 0.58$; Cohen, 1977). Follow-up comparisons with a Bonferroni correction ($\alpha = .05$) revealed that the amblyopia group had higher coherence thresholds (worse performance) than the control group in the slow condition only, $F(1,29) = 23.00$, $p < .001$. There were no differences between groups at the medium, $F(1,29) = .02$, $p = .89$, or the fast speeds, $F(1,29) = .50$, $p = .49$. There was no interaction between age and speed, $F(1.42, 41.21) = .59$, $p = .51$, and there was no main effect of age, $F(1,29) = .68$, $p = .42$.

3.2.3. Correlational analysis

In order to determine if poor VA explained poor motion-defined form perception in the amblyopia group we analyzed correlations between VA and coherence threshold. We correlated amblyopic eye VA with amblyopic eye coherence threshold at each speed. The same procedure was performed for the fellow eye. We also correlated VA in the amblyopic eye (as a measure of depth of amblyopia) with coherence threshold in the fellow eye at each speed. All correlations were non-significant ($p > .05$). The same results were shown in control participants.

We also correlated stereoacuity with VA and with coherence threshold. In the patient group, poor stereoacuity was associated with poor amblyopic eye VA, $r(9) = -0.90$, $p = .001$. Non-significant correlations between stereoacuity and coherence threshold were observed for both eyes and all speed conditions ($p > .05$).

3.3. Discussion

Our main finding in Experiment 2 was that motion-defined form deficits in amblyopia were most pronounced at the slow speed. We found deficits in both the amblyopic and fellow eyes, implicating the disruption of binocular neural mechanisms. We previously reported a deficit in motion-defined form discrimination for both amblyopic and fellow eyes (Giaschi et al., 1992; Ho

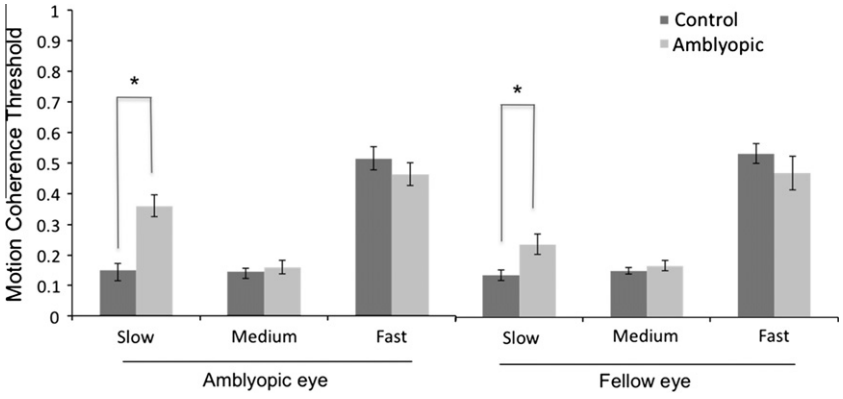

Fig. 3. The effect of amblyopia on motion-defined form perception. Mean coherence thresholds, showing a significant interaction between group and speed in the amblyopic ($p = .001$) and fellow eye ($p = .04$). Error bars represent ±1 standard error. *Indicates a significant mean difference at an experiment-wide α of .05.

et al., 2005), but we examined only very slow speeds at 100% coherence.

The speed tuning of motion deficits in amblyopia is consistent with previous studies in humans and monkeys. Steinman, Levi, and McKee (1988) reported especially large amblyopic deficits of temporal asynchrony discrimination at slow speeds. Hou, Pettet, and Norcia (2008) used visual-evoked potentials to measure coherent motion responses and found that tuning curves for observers with strabismic amblyopia were shifted toward higher speeds relative to control subjects, indicating a loss of sensitivity for slower speeds. Kiorpes, Tang, and Movshon (2006) found reduced sensitivity for global motion direction discrimination in the amblyopic eye of monkeys at slow speeds only. Fellow eyes deficits at slow speeds were found in strabismic but not anisometropic amblyopia. Thus, our results build on existing evidence that amblyopic deficits are speed-dependent, binocular, and are not explained by poor visual acuity. The sample size in the present study was too small to examine differences according to amblyopia subtype.

4. General discussion

The present experiments demonstrate the importance of dot speed in the development of motion-defined form perception. Experiment 1 typified normal development, revealing that performance in 4–6 year olds was more immature at slower than at faster speeds. Experiment 2 showed a speed-dependent deficit in amblyopia, demonstrating that abnormal visual input during childhood adversely affects slow but not fast motion-defined form perception.

4.1. Sensitive periods

The sensitive period for the *typical development* of motion-defined form perception appears to end sooner for fast motion (before 4 years) than for slow motion (after 6 years). A similar developmental pattern has been observed for global motion perception in infant monkeys (Kiorpes & Movshon, 2003) and suggested, although not demonstrated empirically, for global motion perception in human children (Lewis & Maurer, 2005). No other study has examined the speed tuning of this sensitive period for motion-defined form, but the exact age ranges may depend on additional stimulus parameters that we did not vary (Gunn et al., 2002; Schrauf, Wist, & Ehrenstein, 1999).

The sensitive period for *damage* to motion-defined form perception also appears to end sooner for fast motion than for slow motion. The coherence thresholds of the amblyopic group were similar to those of the 4–6 year-old control group for slow motion and did not differ from those of the older control groups for faster motion. The exact length of these sensitive periods cannot be estimated from the histories available on our patients with amblyopia. The sensitive period for the disruption of fast global motion perception is known to be very short (Lewis & Maurer, 2005). Global motion perception was disrupted in patients with congenital cataract but not in patients who developed cataract after 4 months of age. Disruption of slower motion has not been studied in this patient group.

The idea that amblyopia can be thought of as arrested visual development is not new. Our results support the "Detroit model" which asserts that different structures and functions are susceptible to damage at different times and that structures and functions that mature earlier are less susceptible to damage than those that develop later (Levi & Carkeet, 1993). Our patient group is not large enough to examine the hypothesis that follows from this that anisometropia arises later in development than strabismus and, thus, results in a different pattern of visual deficits, although there is evidence against this hypothesis (Birch & Swanson, 2000).

Although the results are interpreted in terms of the speed tuning of motion-defined form development, the immaturity and deficit at the slow speed could be due to coarser resolution of local motion detectors in young children and in patients with amblyopia. This possibility can only be ruled out through further experimentation.

4.2. Neural correlates of motion-defined form perception

Although our task involved orientation discrimination of second-order form, typical and atypical development depended on the speed of the motion contrast used to create this form. Contrary to our previous conclusions that it is mainly the form perception aspects of this task that are maturing during the school-age years (Ho et al., 2005; Parrish et al., 2005; Wang, Ho, & Giaschi, 2007), the present results suggest maturation of the motion component aspects as well. The results of Experiment 2 could be taken as evidence for dorsal stream vulnerability (Braddick, Atkinson, & Wattam-Bell, 2003), consistent with other reports of motion perception deficits in amblyopia (Aaen-Stockdale & Hess, 2008; Aaen-Stockdale, Ledgeway, & Hess, 2007; Buckingham et al., 1991; Ellemberg et al., 2002; Hess, Demanins, & Bex, 1997; Ho & Giaschi, 2007; Ho et al., 2005, 2006; Kelly & Buckingham, 1998; Simmers et al., 2003, 2006). If we assume a single perceptual system subserving human motion perception (Van Boxtel & Erkelens, 2006; Van Boxtel, Van Ee, & Erkelens, 2006), our results suggest that some components of this system mature earlier than others.

An alternative interpretation is that two independent speed-tuned systems mediate fast and slow motion perception (Burr, Fiorentini, & Morrone, 1998; Edwards, Badcock, & Smith, 1998; Gegenfurtner & Hawken, 1995; Gorea, Papathomas, & Kovacs, 1993; Hawken, Gegenfurtner, & Tang, 1994; Khuu & Badcock, 2002; Van de Grind et al., 2001; Van der Smagt, Verstraten, & Van de Grind, 1999; Verstraten, Van der Smagt, & Van de Grind, 1998). The 'slow' system is hypothesized to be active at speeds below 3 deg/s and the 'fast' system becomes more involved as speeds increase, to an upper limit of approximately 80 deg/s (Burr, Fiorentini, & Morrone, 1998; Khuu & Badcock, 2002; van de Grind et al., 2001). It has even been suggested that these slow and fast motion systems correspond to the ventral and dorsal cortical streams, respectively (Gegenfurtner & Hawken, 1996; Thompson, Brooks, & Hammett, 2006). According to this view, our results could be attributed to slower maturation and longer vulnerability of the ventral stream rather than the dorsal stream. Previous evidence about which pathway matures first is inconsistent (Bachevalier, Hagger, & Mishkin, 1991; Distler et al., 1996; Kovacs et al., 1999; Mitchell & Neville, 2004).

Further research is necessary to delineate the cortical mechanisms that mediate the effect of speed on motion-defined form perception.

4.3. Clinical implications

The mainstay of treatment for amblyopia is occlusion therapy, which consists of covering the fellow eye with an opaque eye patch so that the amblyopic eye might regain VA. Therapy is often considered successful if VA in the amblyopic eye improves by 2 or 3 lines on a Snellen chart (Stewart, Moseley, & Fielder, 2003). All of the patients tested in Experiment 2 had completed occlusion therapy, and in some cases had normal VA in the amblyopic eye. Still, the majority of amblyopic participants were impaired on the task at the slow speed. This result is consistent with previous studies demonstrating that some amblyopic deficits, including losses in motion-defined form perception, are resistant to occlusion therapy (Chatzistefanou et al., 2005; Constantinescu et al., 2005; Giaschi et al., 1992; Ho et al., 2005).

5. Conclusions

We present evidence that the typical development of motion-defined form perception depends on dot speed, with slower maturation for slower speeds. We show that atypical visual stimulation during childhood is most disruptive at slow speeds, possibly due to a very short sensitive period for damage at faster speeds.

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