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The effect of occlusion therapy on motion perception deficits in amblyopia

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ABSTRACT

There is growing evidence for deficits in motion perception in amblyopia, but these are rarely assessed clinically. In this prospective study we examined the effect of occlusion therapy on motion-defined form perception and multiple-object tracking. Participants included children (3–10 years old) with unilateral anisometropic and/or strabismic amblyopia who were currently undergoing occlusion therapy and age-matched control children with normal vision. At the start of the study, deficits in motion-defined form perception were present in at least one eye in 69% of the children with amblyopia. These deficits were still present at the end of the study in 55% of the amblyopia group. For multiple-object tracking, deficits were present initially in 64% and finally in 55% of the children with amblyopia, even after completion of occlusion therapy. Many of these deficits persisted in spite of an improvement in amblyopic eye visual acuity in response to occlusion therapy. The prevalence of motion perception deficits in amblyopia as well as their resistance to occlusion therapy, support the need for new approaches to amblyopia treatment.

1. Introduction

In the clinic, unilateral amblyopia is typically defined as reduced visual acuity that cannot be optically corrected in an otherwise healthy eye, with at least a two-line difference in Snellen or logMAR visual acuity between the eyes (Holmes & Clarke, 2006; Ohlsson, 2005). It can be caused by anything that deprives an eye of normal visual experience for a prolonged period before the age of 8 years (von Noorden, 1990). The most common causes are untreated strabismus, which is a misalignment of the eyes, or anisometropia, which is a difference in the refractive error between the eyes, or both strabismus and anisometropia. The fellow eye usually has normal visual acuity.

Amblyopia is commonly associated with disruptions in binocular vision, including fusion and stereopsis, particularly when strabismus is involved (McKee, Levi, & Movshon, 2003). In the psychophysics laboratory, several other deficits in spatial vision have been well established, and some of these are assessed clinically. The spatial vision deficits include contrast sensitivity (Hess & Howell, 1977; Levi & Harwerth, 1977), Vernier acuity (Birch & Swanson, 2000; Levi & Klein, 1985), as well as spatial distortions (Barrett et al., 2003; Bedell & Flom, 1981; Hess, Campbell, & Greenhalgh, 1978), crowding (Bonneh, Sagi, & Polat, 2004; Flom, Weymouth, & Kahneman, 1963; Giaschi et al., 1993; Levi, Hariharan, & Klein, 2002; Schapero, 1971) form integration (Mansouri & Hess, 2006), orientation processing (Husk & Hess, 2013), contour integration (Chandna et al., 2001) and static angle discrimination (Levi & Tripathy, 2006).

There is growing evidence for motion perception deficits in amblyopia that are independent of the spatial vision deficits. Deficits have been reported in: gaze control (Giaschi et al., 1992a); motion aftereffects (Hess, Demanins, & Bex, 1997); oscillatory movement displacement (Buckingham et al., 1991; Kelly & Buckingham, 1998); global motion (Simmers et al., 2003, 2006); optic flow (Aaen-Stockdale, Ledgeway, & Hess, 2007); motion-defined form (Giaschi et al., 1992b; Hayward et al., 2011; Ho et al., 2005; Wang, Ho, & Giaschi, 2007); structure-from-motion (Husk, Farivar, & Hess, 2012); maximum motion displacement (Ho & Giaschi, 2006, 2007; Ho et al., 2005) and attentive motion tracking (Ho et al., 2006). Many of these deficits are found in the fellow eye with normal visual acuity, as well as in the amblyopic eye (Aaen-Stockdale, Ledgeway, & Hess, 2007; Davis et al., 2008; Giaschi et al., 1992b; Ho & Giaschi, 2006, 2007; Ho et al., 2005, 2006; Simmers et al., 2003).

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The current study focused on motion-defined form perception and on multiple-object tracking, two aspects of motion perception that show robust deficits in each eye in children with amblyopia. After reviewing the published psychophysical evidence for deficits in amblyopia, we present new results on the effect of amblyopia treatment on these two aspects of motion perception.

1.1. Motion-defined form perception

Motion-defined form or motion contrast can be created by moving dots inside a central shape in one direction while dots outside the shape move in the opposite direction at the same speed. The shape itself is stationary and lacks luminance contours (Regan & Hong, 1990). Thresholds for correct identification or discrimination of the shape can be measured by fixing the coherence level of the dots at 100% and reducing dot speed, or by fixing the dot speed and reducing the coherence of the dot pattern both inside and outside the shape. 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). Maturation to adult performance levels depends on the stimulus parameters chosen (Giaschi & Regan, 1997; Gunn et al., 2002; Parrish et al., 2005; Schrauf, Wist, & Ehrenstein, 1999). For example, discrimination thresholds are adult-like at 4 years of age for faster speeds of motion and after 6 years of age for slow speeds (Hayward et al., 2011). Motion-defined form tasks activate posterior occipital regions as well as regions of both ventral and dorsal streams including fuisform gyrus, cuneus and MT+ (Bucher et al., 2006; Chen et al., 2003; Giaschi, 2006).

We assessed 20 children with anisometropic and/or strabismic amblyopia (age 4–14 years) on a motion-defined letter identification task (Giaschi et al., 1992b). Compared to a group of 30 age-matched control children, the children with amblyopia showed elevated speed thresholds for identifying letters in both their amblyopic and fellow eyes. This deficit was not due to poor visual acuity because all fellow eyes and many treated amblyopic eyes had normal visual acuity. The fellow eye deficit in motion-defined form identification was confirmed in a different group of children with amblyopia who had global dot motion perception within normal limits (Ho et al., 2005). Most of the children with deficits in motion-defined form identification also showed deficits in texture-defined form identification (Wang, Ho, & Giaschi, 2007). Taken together, these studies suggest that mechanisms involved in figure-ground segregation are deficient in amblyopia.

The role of speed-tuned motion mechanisms in this deficit was confirmed by our more recent work (Hayward et al., 2011). We measured minimum coherence thresholds for motion-defined form discrimination at three fixed speeds: slow (0.1 deg/s), medium (0.9 deg/s), and fast (5 deg/s) in 12 participants with anisometropic and/or strabismic amblyopia (age 7–25 years) and 46 age-matched controls. We found abnormal performance in both amblyopic and fellow eyes at the slow speed only. The slow-speed version of this motion-defined form task was used in the current study. Given the later maturation of motion-defined form perception for slow speeds relative to fast, our results suggest that the deficit in amblyopia reflects a disruption of mechanisms that are still developing at the onset of amblyopia.

1.2. Multiple-object tracking

In a typical multiple-object tracking task (Pylyshyn & Storm, 1988), attention is used to track cued moving targets among moving distractors. Adults with normal vision can track four to five targets simultaneously, but the task becomes increasingly difficult as the number of targets increases. This task has been used with children as young as 3 years of age (O'Hearn, Hoffman, & Landau, 2010). Until age 11, children show a pattern of results similar to that of adults (Brodeur et al., 2013; O'Hearn, Landau, & Hoffman, 2005), but with lower accuracy (Trick, Hollinsworth, & Brodeur, 2009; Trick, Jaspers-Fayer, & Sethi, 2005). Performance on this task is believed to reflect the high-level motion system that depends primarily on attention (Cavanagh, 1992) and involves the posterior parietal cortex (Battelli et al., 2001; Culham et al., 1998; Howe et al., 2009; Jovicich et al., 2001), but low-level motion areas such as MT+ are also involved (Culham et al., 1998; Howe et al., 2009; Jovicich et al., 2001).

We assessed 18 children with anisometropic or strabismic amblyopia (age 9–17 years) and 30 age-matched controls on a multiple-object tracking task (Ho et al., 2006). Participants viewed eight dots surrounding a central fixation target in a random array. At the beginning of each trial, up to four dots were cued by briefly turning red. Then, the dots moved in random directions at a speed of 6 deg/s. After 5 s, the dots stopped moving, and participants had to click on the dots they had been tracking (full report). Accuracy for identifying the tracked dots decreased as more dots were required to be tracked for both children with amblyopia and controls, which replicates the typical finding. The performance of children with amblyopia, however, was poorer than that of controls, regardless of which eye they used. In addition, the deficit increased as more dots were required to be tracked. There was no difference between children with strabismic or anisometropic amblyopia. The children with amblyopia also showed a deficit on a single-object tracking task in which one of four targets was tracked along a circular path. Performance on a low-level global dot motion task was within normal limits in all but 3 children with amblyopia.

The multiple-object tracking deficit in amblyopia was replicated, using an easier partial-report task, in a group of 7 participants (age 9–37 years) with anisometropic and/or strabismic amblyopia (Secen et al., 2011). This version of the task was used in the study described below. In a different type of tracking task, in which the ability to track deviations in linear trajectories was assessed, a small deficit was observed in the amblyopic but not the fellow eye in a group of six adults with strabismic and/or anisometropic amblyopia (Tripathy & Levi, 2008). The deficit in multiple-object tracking in amblyopia is consistent with other deficits on high-level tasks requiring attentive processing, including object enumeration (Sharma, Levi, & Klein, 2000) and the attentional blink (Asper, Crewther, & Crewther, 2003; Popple & Levi, 2008).

1.3. Motion perception and amblyopia treatment

The motion perception and fellow-eye deficits summarized above imply that the amblyopic visual system can be more severely compromised than originally thought at a time when the only deficits considered were deficits in visual acuity and other aspects of spatial vision. It is possible that treatment may be more difficult and may take a longer time in patients with motion deficits. In typical treatment for amblyopia, after the amblyogenic factors such as anisometropia or strabismus are corrected, the clinically unaffected fellow eye is occluded with a patch to improve the visual acuity of the amblyopic eye.[1] This often works quite well, particularly in children under the age of 7 years (Fronius et al., 2014), but occlusion fails to restore visual acuity in the amblyopic eye in up to one third of cases (Clarke et al., 2003; Flynn et al., 1999). This is partly because children and their parents do not always follow the treatment instructions they are given, so their compliance is poor (Fronius et al., 2014). However, failures can also occur when

[1] Atropine drops or a fogged lens (Bangerter foil) may also be used to penalize the fellow eye, with similar results (Pediatric Eye Disease Investigator Group, 2008, 2010).

compliance is good (e.g. Chekitaan, Karthikeyan, & Meenakshi, 2009; Hiscox et al., 1992). Even with successful patching, there can be a decrease in visual acuity after the patching stops in up to 25% of cases (Holmes et al., 2007). We hypothesize that treatment failures and post-treatment "slippage" occur because occlusion treats only some of the visual deficits in amblyopia.

Different aspects of visual function respond differently to occlusion therapy. Contrast sensitivity improves alongside visual acuity (Wali et al., 1991), but contour integration improves faster and more completely than visual acuity (Chandna, Gonzalez-Martin, & Norcia, 2004). On the other hand, abnormalities in visual evoked potentials have been observed even after patching restores visual acuity to normal levels (Weiss & Kelly, 2004). Simmers et al. (1999) showed that occlusion therapy had a different effect on contour interactions, contrast perception, eye movements, positional uncertainty and visual acuity. There are two reasons for suggesting that motion deficits may not be treatable by occlusion therapy. First, the sensitive period for the development of motion perception in animal models is short (Mitchell, Kennie, & Kung, 2009). This implies that the neural mechanisms underlying motion perception may be mature and not very plastic by the time patching is undertaken. Secondly, we found that motion-defined form deficits were more prevalent in children who had undergone multiple strabismus surgeries and several rounds of occlusion therapy (see Giaschi et al., 1992b versus Ho et al., 2005), suggesting their amblyopia was difficult to treat.

To assess how motion deficits are affected by occlusion therapy, we conducted a prospective study. Children with amblyopia were followed through their occlusion treatment to determine if the deficits in performance on motion-defined form and multiple-object tracking tasks were present before the start of occlusion therapy, if the deficits improved with patching, and if the deficits predicted poor occlusion therapy outcomes.

2. Methods

This study was approved by the University of British Columbia's Clinical Research Ethics Board and the Children's and Woman's Health Centre of BC Research Ethics Board, and carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki). Informed consent was obtained from a parent or guardian and verbal assent was obtained from the child at each visit.

2.1. Participants

Forty-five children receiving treatment for unilateral amblyopia between the ages of 3 and 10 years were recruited from the ophthalmology clinic at BC Children's Hospital; 61 control children with healthy vision in the same age range were recruited from the community. The diagnosis of unilateral amblyopia was based on ophthalmologist report, and included a 2-line difference in Snellen acuity (in most cases) with a best-corrected visual acuity of 20/30 (0.2 logMAR acuity) or worse in the amblyopic eye and within the normal range for age in the fellow eye (Table 1). The upper limit for normal visual acuity was 20/40 (0.3 logMAR) for age 3 years, 20/30 for age 4–6 years and 20/25 (0.1 logMAR) for ages 7 and older (Dobson et al., 2009; Drover et al., 2008). The initial clinical assessment was performed on the same day as or prior to the first laboratory visit.

A child was considered to have anisometropic amblyopia if in addition to the visual acuity loss, the spherical equivalent difference[2] between the eyes was 1 diopter in the absence of any ocular manifest deviation (Weakley, 2001). Strabismic amblyopia was diagnosed if the patient showed any constant or intermittent manifest deviation $\geqslant 1$ prism diopter with an accommodative or non-accommodative involvement (Pediatric Eye Disease Investigator Group, 2003). Aniso-strabismic amblyopia was diagnosed if the patient met the criteria for both anisometropic and strabismic amblyopia. Based on this categorization, 22 children were classified with anisometropic amblyopia, 11 with strabismic amblyopia and 12 with aniso-strabismic amblyopia (see Table 1).

All children with amblyopia were receiving occlusion therapy at the time of testing, a few in combination with atropine penalization or Bangerter foils (see Table 1). Treatment started prior to or within one month after the first laboratory test date. Throughout this paper we refer to the eye that was treated by the ophthalmologist as the amblyopic eye, even if acuity in the treated eye reached normal levels during the study. At the end of the study, each child's treatment status (completed or in progress) was determined from their ophthalmologist's records.

All participants were requested to return at 6-month intervals after the first visit for a total of three test visits. For children with amblyopia, we attempted to schedule the first session prior to patching. Most children, however, had started occlusion therapy well before entering the study. On average, the children had been prescribed 1600 h (range: 0–8133 h, SD = 1940, Mdn = 823.3) of occlusion therapy, with a mean compliance of 83% based on parental report, prior to the first test session. This is similar to the compliance level of 77% reported with an electronic occlusion dose monitor (Fronius et al., 2014). At each test visit, visual acuity was assessed using the Regan high-contrast letter chart (Regan, 1988). The Lighthouse Picture Chart (Lighthouse Low Vision Products) or PattiPics Symbol Chart (Precision Vision) was used with young children who could not recognize alphabet letters. Stereoacuity was assessed using the Randot Preschool test (Stereo Optical Co. Inc.).

To be included in the study, control children needed a best-corrected logMAR acuity, at first test visit, within the normal limits for age as defined above, and stereoacuity within the normal limit for their age (<6 years: 200 arc sec or better; 6 years: 100 arc sec or better; >7 years: 60 arc sec or better; Birch et al., 2008). Children with amblyopia were also required to have a best-corrected logMAR acuity in the amblyopic eye of 1.0 (20/200) or better at the first laboratory visit to ensure that the stimuli were visible, although their initial visual acuity at clinical assessment could have been worse than this.

For analyses, we compared the first and the last visits. We selected the third visit to be the last visit except in cases where there were only two visits (16 controls, 7 children with amblyopia) or the child had stopped patching prior to the third visit (2 children with amblyopia).

2.2. Apparatus

Tasks were run using a Macintosh Powerbook G4 laptop. The motion-defined form stimuli were generated using MATLAB R2006b (The MathWorks, Inc.) with the Psychophysics Toolbox extension 3.0.8 (Brainard, 1997; Kleiner, Brainard, & Pelli, 2007; Pelli, 1997) and displayed on a 17″ Apple Studio Display CRT monitor with a resolution of 1024 × 768 and a refresh rate of 75 Hz. The multiple-object tracking stimuli were generated using MATLAB 5.2 with the Psychophysics Toolbox extension 2.52 and displayed on a 15″ flat panel Apple Studio Display LCD color monitor at a resolution of 800 × 600 and a refresh rate of 60 Hz. Responses in both tasks were collected with a Logitech gamepad.

2.3. Procedure

After the visual and stereoacuity assessment on each visit, performance was measured on either the multiple-object tracking or

[2] Spherical equivalent = full spherical correction + 1/2 (cylindrical correction).

Table 1
Clinical and first test visit data for children with amblyopia.

ID	Clinical assessment						First test visit		Time of analysis
	Diagnosis	Snellen acuity AE	Snellen acuity FE	Optical correction	Age (years)	Treatment type	Age (years)	Stereoacuity (arc sec)	Treatment status
S02*	A	20/30−	20/20	AE: +2.50 FE: +0.50 + 0.25 × 90	5.19	Patch	5.31	100	C
S03*	A	20/50	20/30	AE: +3.00 FE: +1.50	3.82	Patch	5.37	100	C
S04	A	20/50	20/20	AE: +5.00 FE: +1.25	5.04	Patch & atropine	6.49	>800	C
S05	A	20/200	20/20	AE: +3.00 FE: plano	6.26	Patch & foil	6.78	100	P
S06*	A	20/80	20/40	AE: −1.75–2.25 × 004 FE: +0.50–0.25 × 007	3.47	Patch	5.9	40	C
S07	A	20/80	20/20	AE: +4.00 + 0.50 × 80 FE: +1.25	5.07	Patch	6.56	100	P
S08*	A	20/30	20/20	AE: +4.50 + 1.50 × 40 FE: +0.50 + 100 × 90	4.76	Patch	8.05	>800	P
S09	A-S	20/70	20/20	AE: +2.00 + 3.50 × 75 FE: +1.25 + 1.25 × 90	4.06	Patch	8.22	Not tested	C
S10*	A-S	20/50	20/25	AE: +4.25 + 1.25 × 110 FE: plano	4.28	Patch & atropine	7.33 (MOT) 8.31 (MD)	800 (MOT) >800 (MDF)	C
S11*	A-S	20/100	20/10	AE: +3.25 + 1.00 × 180 FE: +2.00	3.96	Patch	8.41	>800	C
S12	S	20/50	20/30	AE: +5.25 + 1.0 × 90 FE: +4.75 + 1.0 × 90	3.97	Patch	6.47	>800	C
S13	A	20/50	20/20	AE: −2.50 + 5.50 × 100 FE: plano	8.42	Patch	8.91	100	C
S14	A	20/40	20/20	AE: −3.0 + 5.5 × 100 FE: −1.5 + 3.0 × 85	5.09	Patch	5.16	800	C
S15	A	20/70	20/25	AE: +4.50 + 1.00 × 115 FE: plano	3.81	Patch	3.93	>800	P
S16	A	20/400	20/20	AE: +5.50 + 1.50 × 110 FE: plano	5.42	Patch & atropine	8.55	>800	U
S17	A	20/200	20/25	AE: +6.75 FE: +2.00	4.59	Patch	6.25	40	C
S18	A	20/60	20/20	AE: +4.0 + 1.00 × 100 FE: plano	5.63	Patch	6.08	>800	U
S19	A	20/30	20/20	AE: −0.75 + 3.25 × 90 FE: plano	4.30	Patch	4.49	60	C
S20*	A	20/40	20/20	AE: +3.50 + 1.00 × 100 FE: plano	5.73	Patch & atropine	8.33	>800	C
S22*	A-S	20/40	20/25	AE: +2.75 + 1.00 × 179 FE: +1.75 + 0.50 × 2	4.45	Patch	4.59	>800	P
S23*	A-S	20/100	20/20	AE: −0.25 + 1.25 × 60 FE: −0.75 + 0.25 × 110	6.82	Patch & atropine	7.15	>800	U
S25	S	20/40	20/25	AE: +1.00 FE: +1.00	4.15	Patch	4.89	>800	P
S26	A	20/40	20/30	AE: +6.25 + 0.80 × 90 FE: +4.50 + 0.50 × 90	4.28	Patch	4.79	800	P
S27	A-S	20/80	20/25	AE: +5.25 FE: +0.25	5.11	Patch	5.6	>800	P
S28	A	20/60	20/20	AE: +6.00 FE: +2.80	5.24	Patch	7.13	Not tested	P
S29	S	20/40	20/25	AE: +2.00 + 0.75 × 95 FE: +2.25 + 0.75 × 85	4.86	Patch & atropine	5.82	>800	C
S30	A-S	20/60	20/30	AE: +2.00 FE: plano	3.44	Patch	4.03	800	C
S31	S	20/40	20/25	AE: +3.75 + 1.75 × 95 FE: +3.00 + 1.75 × 85	3.66	Patch	4.01	>800	C
S32	A	20/50	20/25	AE: −3.75 + 3.75 × 90 FE: −2.50 + 3.75 × 90	5.09	Patch	6.07	200	C
S33	A	20/200	20/20	AE: +5.75–0.25 × 10 FE: plano	3.19	Patch	4.56	>800	C
S34	S	20/70	20/30	AE: +2.5 + 1.5 × 95 FE: +2.5 + 0.5 × 90	4.83	Patch	4.83	>800	C
S35	A-S	20/40	20/20	AE: +2.75 + 1.50 × 90 FE: +2.25 + 0.75 × 95	4.27	Patch	4.83	400	U
S36	A	20/40	20/30	AE: +3.00 + 0.75 × 105 FE: +2.00 + 0.25 × 135	5.02	Patch	5.38	60	U
S37	A-S	20/60	20/20	AE: −6.75 + 1.00 × 105 FE: plano + 1.00 × 10	4.94	Patch	5.28	400	C

(*continued on next page*)

Table 1 (*continued*)

ID	Clinical assessment						First test visit		Time of analysis
	Diagnosis	Snellen acuity AE	Snellen acuity FE	Optical correction	Age (years)	Treatment type	Age (years)	Stereoacuity (arc sec)	Treatment status
S38	A	20/100	20/40	AE: +5.25 + 2.50 × 100 FE: +2.00 + 0.50 × 90	3.80	Patch	4.32	>800	C
S39	S	20/50	20/20	AE: +2.00 FE: +2.00	4.24	Patch	4.5	>800	P
S40	S	20/50	20/30	AE: +2.75 + 1.00 x080 FE: +2.75	4.73	Patch & atropine	4.73	>800	P
S41	S	20/50	20/30	AE: +5.50 + 1.75 × 95 FE: +5.00 + 2.00 × 90	2.70	Patch	3.87	>800	U
S42	A-S	20/200	20/40	AE: +6.25 FE: +3.25 + 0.75 × 95	2.50	Patch	3.22	>800	C
S43	A-S	20/40	20/25	FE: +4.75 AE: +8.00	5.01	Patch	5.2	800	C
S44	A-S	20/400	20/30	AE: +4.75 + 0.25 × 180 FE: +2.50 + 0.50 × 180	3.40	Patch & atropine	3.92	Not tested	P
S45	A	20/200	20/20	AE: +4.50 FE: +5.00 + 10.50 × 155	6.32	Patch & foil	7.74	100	C
S46	S	20/30	20/20	AE: +3.00 FE: +3.00	3.98	Patch	5.07	800	U
S47	S	20/100	20/20	AE: +1.50 + 1.25 × 180 FE: +1.50 + 0.50 × 105	4.25	Patch	5.35	>800	U
S48	S	20/40	20/25	Never prescribed glasses	5.10	Patch	5.49	100	C

Note: S02-S25 performed the multiple-object tracking (MOT) task; S26-S49 performed the motion-defined form (MDF) task; *participated in both tasks. Subtypes: A = anisometropic, A-S = aniso-strabismic, S = strabismic. Acuities: AE = amblyopic eye, FE = fellow eye. Optical correction: R = right eye, L = left eye, Stereoacuity: >800 = unable to measure; converted to 1200 arc sec for data analysis. Treatment status: C = completed treatment, P = occlusion treatment still in progress, U = unknown.

the motion-defined form task, or both (for 9 children). For both tasks, participants were given child-friendly instructions followed by practice runs with binocular viewing. During the testing phase, all participants wore a dark patch to occlude the non-tested eye. Eye testing order was counterbalanced. Diffuse illumination was used in the test room to prevent glare on the screen. Audio-visual feedback was provided after every trial, the trials were self-paced and guessing was encouraged.

The motion-defined form display was presented at a viewing distance of 2.5 m. The stimulus was a randomly generated array of white square dots (1.74 arc min^2, 94.5 cd/m^2) presented on a black background (7.45 × 5.53 deg, 0.12 cd/m^2) with a dot density of 8% (170 dots/deg^2) and speed of 0.08 deg/s for 640 ms (Fig. 1, right). These parameters were chosen from our previous study in which we found that the motion-defined form deficit in amblyopia was largest at this speed of motion (Hayward et al., 2011). Dots within an invisible rectangle (2 × 1 deg) at the center of the screen moved coherently in one direction (up or down), and dots outside this rectangle moved in the opposite direction. The direction of coherent motion was random across trials. The rectangle could be oriented horizontally or vertically, had no luminance-defined contours and remained stationary within the display. The participant's task was to identify the orientation of the rectangle. The proportion of coherently moving dots was controlled using a two-down, one-up staircase. 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. A run had a minimum of 40 trials and stopped after 10 reversals or 50 trials were reached, whichever occurred first. One run was conducted for each eye. A coherence threshold was estimated by fitting a Weibull function to the data using a maximum-likelihood minimization procedure and χ^2 test to assess adequacy of fit ($p > .05$) (Watson, 1979). The point of inflection (82% correct in a two-alternative procedure) was taken as the measure of coherence threshold (Strasburger, 2001).

The multiple-object tracking task was similar to the partial report one described previously (Secen et al., 2011). Participants were seated 57 cm from the display. A cartoon character was presented in the center of the screen and children were instructed to maintain fixation on this character throughout the task. At the beginning of each trial, eight randomly-positioned dots (1 deg diameter) appeared inside a dark square (14 × 14 deg, 0.52 cd/m^2). One to four target dots were cued by appearing as a cartoon character, while the remaining dots were green (20.4 cd/m^2) (Fig. 1, left). After 1.5 s, all dots reverted to green and moved around the screen at a velocity of 6 deg/s for 5 s, with the motion direction perturbed randomly every 45 ms such that the dots never occluded or collided with each other. The participant's task was to track all of the cued dots in motion while maintaining central fixation. When the dots stopped moving, one dot was surrounded by a red square, and the child had to indicate if this dot had been one they were tracking. The prompted dot was one of the target dots 50% of the time. One or two runs of 24 trials were tested for each eye, with six trials for each number of targets tracked (one to four) ordered randomly. Accuracies for each number of targets tracked were averaged across trials.

2.4. Data analysis

Analysis of variance (ANOVA) was used to compare the control and amblyopia groups on each task. For each control participant, one eye was chosen randomly to act as a control for the amblyopic eye, and the other eye acted as a control for the fellow eye. For the motion-defined form task, we conducted an ANOVA investigating the effect of group (amblyopia, control) on motion coherence thresholds at first visit; and for the children followed through occlusion therapy, we conducted an ANOVA investigating the effect of group and visit (first visit, last visit) on motion coherence thresholds. Similarly, for the multiple-object tracking task, we conducted an ANOVA investigating the effect of group and number of

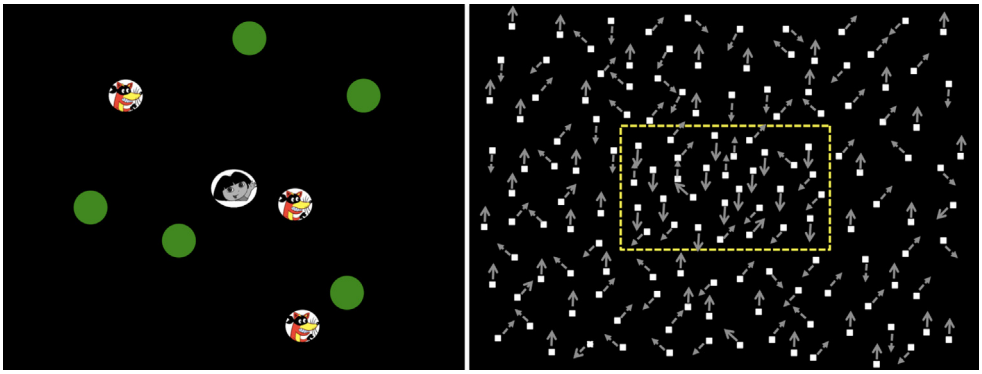

Fig. 1. Left: First frame of the multiple-object tracking animation sequence showing the cuing of three of eight dots to be tracked and the central fixation character. Right: The motion-defined form stimulus showing a horizontal rectangle. The solid arrows represent the coherent motion direction of the signal dots and the dashed arrows represent the random motion direction of the noise dots. The rectangle border was made visible by motion contrast between signal dots inside and outside the rectangle.

targets tracked (one through four) on tracking accuracy at first visit; and for the children followed through occlusion therapy, we conduced an ANOVA investigating the effect of group, number of targets tracked, and visit on tracking accuracy. Because some children had data available for one eye only, each of these analyses was conducted twice: once for the amblyopic eye, and once for the fellow eye.

In addition, the average performance of the control participants was used to identify motion deficits in individual children with amblyopia. One eye was chosen from each control participant to create norms against which to compare the children with amblyopia. Since we expected performance to vary with age, we classified children into two age categories, young (<7.0 years) and old ($\geqslant$7.0 years), and computed values separately for children in each age category for each dependent measure at the first and last visit. These age categories were selected based on our previous work showing motion-defined form perception for the stimuli used here is mature by age 7 (Hayward et al., 2011); for consistency, the same age categories were used for the multiple-object tracking task. We confirmed that the age groups were appropriate for the current study by examining the data for the control children in 1-year age bins. There were no age differences between children with amblyopia and controls on either task (motion-defined form: young $t(55) = 1.94$, $p = .06$, old $t(22) = 1.64$, $p = .12$; multiple-object tracking: young $t(30) = 0.97$, $p = .34$, old $t(24) = 1.76$, $p = .09$).

Consistent with a one-tailed 95% confidence interval, a deficit on the motion-defined form task was defined as a coherence threshold that fell 1.64 SD above a patient's age-matched control group mean. Because higher values represent better performance in multiple-object tracking, a tracking deficit was defined as an accuracy score that fell 1.64 SD below a patient's age-matched control group mean. Tracking deficits were calculated for each number of targets tracked (one through four). In addition, intercepts and slopes were calculated for each participant using the four tracking conditions at first visit where intercept was defined as the point at which the best-fit regression line intersected the *y*-axis and slope was defined as the slope of this line. Deficits were defined as an intercept 1.64 SD below the control group mean, or a negative slope 1.64 SD steeper than the control group slope. Participants were considered to have a tracking deficit in a particular eye if they had at least one deficit at any tracking level, or had an intercept or a slope deficit.

2.5. Calculation of occlusion doses, effects, and outcomes

Patient clinic charts were reviewed to collect information relevant to amblyopia treatment. Weighted sums and averages were used to calculate prescribed and parental self-reported occlusion dose (h) and occlusion dose rate (h/day) respectively, between patching start date, first laboratory test date, last laboratory test date and patching stop date. Reported patching compliance was calculated as the reported dose divided by the prescribed dose multiplied by 100. Response to patching was determined using task performance measures: coherence threshold (motion-defined form) and accuracy (multiple-object tracking), and logMAR visual acuities for each eye collected at first and last visit. In addition, amblyopic and fellow eye logMAR acuities were collected from the clinic charts at the occlusion therapy start and stop dates and were used to calculate three treatment outcomes. Following Stewart, Moseley, and Fielder (2003), we calculated: (1) residual amblyopia as the interocular difference between the amblyopic and fellow eye acuity, and (2) proportion of improvement in the amblyopic eye,[3] for last-visit and patching stop date. Following Lin and Chen (2013), we used (3) a categorical measure of successful treatment defined as 0.1 logMAR acuity or better in the amblyopic eye at patching stop date.

3. Results

3.1. Motion-defined form

3.1.1. First visit

We initially assessed motion-defined form perception in 32 children with amblyopia (age 3.22–8.41 years; $M = 5.54$, SD = 1.43) and 49 controls (age 4.01–8.07 years; $M = 6.09$, SD = 1.31). A separate ANOVA was conducted for each eye to investigate the effects of group (amblyopia, control) on motion coherence thresholds. Each eye of control participants was compared to either the amblyopic or the fellow eye of the children with amblyopia.

Group average motion coherence thresholds are shown in Fig. 2. For the amblyopic eye, data were available for 25 children with amblyopia and 40 controls. Motion coherence thresholds were significantly elevated in children with amblyopia, $F(1,43.2) = 42.1$, $p < .001$. For the fellow eye, data were available for 29 children with amblyopia and 41 controls. Again, coherence thresholds were significantly elevated in children with amblyopia, $F(1,47.2) = 14.5$, $p < .001$.

We assessed children with amblyopia on an individual basis, comparing their performance to control children in the same age category. Twenty-two of the 32 children had deficits in

[3] Proportion improvement = (amblyopic eye acuity at time 1 – amblyopic eye acuity at time 2)/(amblyopic eye acuity at time 1 – fellow eye acuity at time 2).

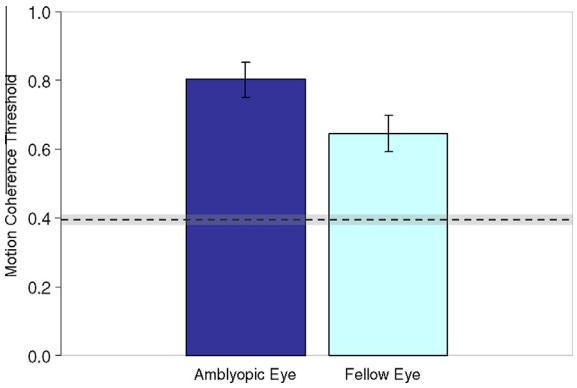

Fig. 2. First visit motion coherence thresholds on the motion-defined form task in the amblyopic and fellow eyes of children with amblyopia. Error bars represent standard error. Dashed line represents control mean coherence threshold + one standard error.

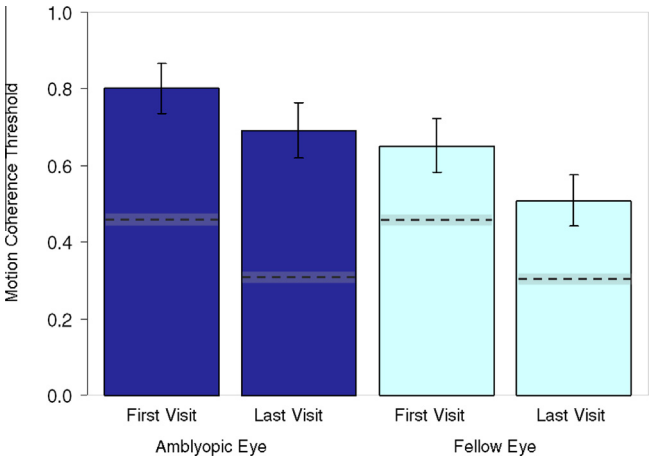

Fig. 3. First- versus last-visit motion coherence thresholds on the motion-defined form task. Error bars represent standard error. Dashed lines represent control mean coherence threshold + one standard error.

motion-defined form perception. Of these 22 children, 13 had data available for both eyes: six showed a deficit in both eyes and seven showed a deficit in only the eye treated for amblyopia. Six children with a deficit had data available for only the fellow eye, and the remaining three children had data available for only the eye treated for amblyopia. Ten children had normal motion coherence thresholds: nine in both eyes and one in the fellow eye, but with no data available for the eye treated for amblyopia. Compared to children without any motion-defined form deficits, children with a deficit in any eye had significantly worse visual acuity in the amblyopic eye, $t(30) = 2.09$, $p = .05$ (0.41 vs. 0.25 logMAR), but not the fellow eye, $t(30) = 1.59$, $p = .12$), and significantly worse stereoacuity, $t(29) = 2.47$, $p = .02$ (947 vs. 520 arc sec).

3.1.2. Occlusion effects on motion-defined form perception

For the motion-defined form task, we followed 22 children with amblyopia through occlusion therapy, and 34 controls were tested at the same intervals. There was no difference in number of days between first and last visit (amblyopia, $M = 407$, SD = 155; controls, $M = 360$, SD = 161; $t(55) = 1.08$, $p = .29$). To assess group differences in motion-defined form perception over time, an ANOVA was conducted on motion coherence threshold as a function of group (amblyopia, control) and visit (first, last) for each eye.

For the amblyopic eye, data from 18 children with amblyopia and 30 controls were available. There was a significant effect of group, $F(1,46) = 45.8$, $p < .001$, such that children with amblyopia had elevated thresholds compared to controls, and a significant effect of visit, $F(1,46) = 8.33$, $p = .01$, such that children performed better at the task on their last visit. There was no group × visit interaction, $F(1,46) = 0.01$, $p = .92$. For the fellow eye, data from 20 children with amblyopia and 33 controls were available. Results were similar to the amblyopic eye: there was a significant effect of group, $F(1,51) = 17.86$, $p < .001$; a significant effect of visit, $F(1,51) = 14.50$, $p < .001$; and no group × visit interaction, $F(1,51) = 0.08$, $p = .78$. Thus, while children with amblyopia did improve over time (Fig. 3), the improvement was equivalent to the improvement seen in the controls. Therefore, the decrease in coherence threshold is likely due to practice or to normal development.

Assessing the children with amblyopia on an individual basis, 14 of 22 children showed motion coherence deficits on their first visit: these deficits persisted in eleven and resolved in three children. Moreover, of the eight children who had normal motion coherence thresholds at first visit, one had developed a deficit by last visit.

3.1.3. Motion-defined form deficits and response to occlusion therapy

Children with amblyopia showed a significantly greater improvement in logMAR visual acuity in their amblyopic eye between their first and last visit ($M = 0.082$, equivalent to 6.6 letters) than control children, ($M = 0.015$, 1.2 letters), $t(54) = 2.11$, $p = .02$, 1-tailed. The improvement in visual acuity in the fellow eye ($M = 0.064$, 5.1 letters, for children with amblyopia; $M = 0.022$, 1.7 letters, for controls), failed to reach significance, $t(54) = 1.74$, $p = .09$, 2-tailed. This pattern of improvement suggests that occlusion therapy had a positive impact on visual acuity in the amblyopic eye. However, nineteen children with amblyopia had suboptimal amblyopic eye visual acuity at last visit.

We examined the proportion of children with persistent, developed, or resolved motion-defined form deficits in each eye as a function of response to treatment (Table 2). Eight children (36%) showed a positive response to treatment: three maintained normal motion-defined form perception in both eyes, two had persistent deficits in at least one eye, and three resolved a deficit in at least one eye. Of the fourteen children who did not respond to occlusion therapy: four maintained normal motion-defined form perception in both eyes, nine retained or developed deficits in at least one eye and one resolved a deficit in the amblyopic eye. There were no significant correlations between the number of hours of prescribed patching before the first visit and coherence thresholds in either eye (both $p > .05$).

Compliance with occlusion therapy, occlusion dose (h), and occlusion dose rate (h/day) between patching start and first visit, and between first visit and last visit, were not correlated with having a persistent or developed motion-defined form deficit in at least one eye (all $p > .05$), nor were they correlated with change in motion coherence thresholds between first and last visit (all $p > .05$). Finally, children with and without an initial motion deficit did not differ significantly between amount of residual amblyopia ($p = .17$) or proportion of improvement in the amblyopic eye ($p = .99$) at last visit.

To determine if motion-defined form deficits predict a poorer response to occlusion therapy, we compared first-visit thresholds in children with normal visual acuity on their last visit to those of children with suboptimal visual acuity on their last visit. On average, children with amblyopia who had suboptimal visual acuity by last visit had significantly higher initial coherence thresholds than those who had normal visual acuity by last visit, $t(16) = 5.07$, $p < .001$ ($M = 0.916$ vs. 0.402). In addition, beyond our last-visit data, we were able to obtain follow-up clinical data on treatment success at occlusion stop date for seventeen children who came

Table 2
Motion-defined form deficits and visual acuity in children with amblyopia between first and last visit.

ID	Amblyopic eye			Fellow eye			Occlusion therapy***					
	Acuity first visit (logMAR)	Acuity last visit (logMAR)	Motion deficit	Acuity first visit (logMAR)	Acuity last visit (logMAR)	Motion deficit	Prescribed cumulative dose from patch start to first visit (h)	Prescribed cumulative dose from first to last visit (h)	Prescribed dose rate from patch start to first visit (h/day)	Prescribed dose rate from first to last visit (h/day)	Compliance from patch start to first visit (%)	Compliance from first visit to last visit (%)
S02	0.16**	0.21**	NONE	0.11**	0.00	NONE	366.0	249.0	1.1	2.5	68.0	84.5
S03	0.19**	0.15**	PERSIST	0.11**	0.06	DEVELOP	265.4	138.9	1.5	0.6	100.0	Unknown
S06	0.10	0.03	NONE	−0.03	−0.05	NONE	489.3	60.7	0.6	0.1	100.0	78.3
S08	0.48**	0.40**	NONE	0.15**	−0.05	NONE	8133.0	2514.0	5.2	6.0	69.4	100.0
S11	0.38**	0.30**	PERSIST	0.00	−0.10	NONE	6213.0	1427.5	3.8	3.1	84.0	89.5
S22*	0.70**	0.28**	PERSIST	0.37**	0.25**	Deficit†	851.0	1462.5	6.0	3.2	62.6	57.4
S26	0.30**	0.24**	NONE	0.17**	0.21**	NONE	230.0	1045.0	2.5	1.5	100.0	91.1
S27	0.40**	0.38**	PERSIST	0.18	−0.01	No data	588.0	1554.5	4.0	4.0	87.5	66.9
S28	0.17	0.30**	PERSIST	0.00	−0.01	RESOLVE	4666.0	4928.0	6.8	8.0	78.0	42.5
S29	0.12	0.26**	Deficit†	0.15	0.20**	DEVELOP	310.0	553.0	0.9	1.6	27.9	59.8
S30*	0.54**	0.29**	Deficit†	0.17	0.08	RESOLVE	899.9	1226.0	1.3	1.5	24.9	97.7
S31*	0.28**	0.18	No data	0.36**	0.18	RESOLVE	188.0	412.0	1.4	1.0	100.0	100.0
S32*	0.49**	0.31**	PERSIST	0.30**	0.20**	PERSIST	3968.0	2232.0	8.0	7.8	45.6	Unknown
S33*	0.40**	0.30**	PERSIST	0.00	0.00	NONE	1805.0	627.0	3.6	3.0	86.2	0.0
S34*	0.54**	0.23**	No data	0.18	0.08	RESOLVE	3055.0	525.0	3.0	1.0	53.5	68.3
S35*	0.40**	0.15**	NONE	0.15	−0.06	NONE	975.0	1518.5	4.8	2.9	52.6	157.8
S36*	0.18	0.05	NONE	0.00	−0.06	NONE	168.0	1094.0	2.0	2.0	200.0	136.8
S37	0.18	0.36**	PERSIST	0.18	0.15**	PERSIST	5523.9	840.0	3.1	2.0	97.5	169.2
S38	0.51**	0.51**	RESOLVE	0.00	0.00	NONE	1124.0	2136.0	5.9	6.0	96.3	100.0
S39*	0.48**	0.38**	NONE	0.00	0.08	NONE	534.0	687.6	1.7	1.8	131.5	68.9
S40	0.38**	0.30**	PERSIST	0.20**	0.08	PERSIST	952.0	1097.5	2.8	3.1	86.9	80.6
S41	0.70**	0.65**	PERSIST	0.25**	0.40**	PERSIST	679.0	525.0	1.7	3.0	88.5	100.0

* Showed a positive response to treatment (acuity improved 0.1 logMAR or better in the amblyopic eye between lab visits).
** Suboptimal acuity (worse than 0.1 logMAR on Regan chart or 0.2 logMAR on pediatric chart).
*** Calculated from medical charts where information was available.
† First visit data not available, therefore could not determine if deficit developed or persisted.

for at least one motion-defined form visit. However, there was no difference in initial motion-defined form thresholds for those who had suboptimal acuity vs. normal at occlusion stop date, $t(15) = 0.078$, $p = .94$ ($M = 0.521$ vs. 0.500). By occlusion stop date, thirteen children (76%) were classified as successfully treated (Lin & Chen, 2013). Fig. 4 displays the percentage of children with motion-defined form perception deficits as a function of treatment success. Most of the children with motion deficits were in the successful treatment group. There was no significant association between having any deficit at the first visit and the amount of residual amblyopia or proportion of amblyopic eye improvement at patching stop date (both $p > .05$). For eleven children who returned for a last test visit, there were also no associations between having a persistent or developed deficit and these measures (both $p > .05$).

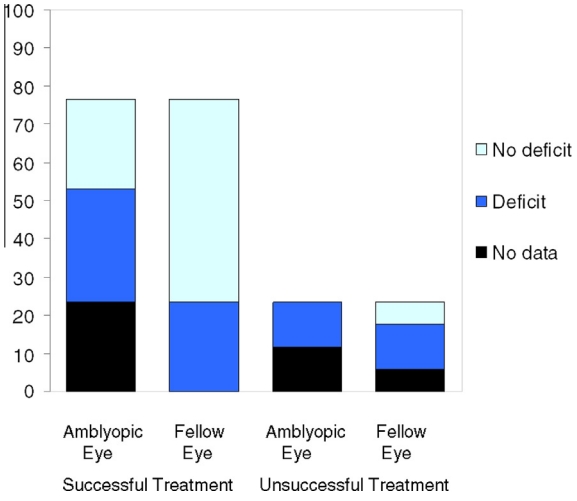

Fig. 4. The relationship between first-visit motion-defined form deficits and treatment outcome in the 17 children who had completed occlusion therapy by the end of the study. Successful treatment was defined as an amblyopic eye visual acuity of 0.1 logMAR or better at patching stop date.

3.2. Multiple-object tracking

3.2.1. First visit

We initially examined multiple-object tracking in 22 children with amblyopia (age 3.93–8.91 years; $M = 6.51$, SD = 1.47) and 35 control children (age 4.54–9.22 years; $M = 6.75$, SD = 1.11). Two ANOVAs were conducted to investigate the effects of group (amblyopia, control) and number of targets tracked (one through four) for each eye. Half of the control children were randomly assigned to have their left eye compared to the amblyopic eye and their right eye to the fellow eye, and the remaining half were assigned the opposite. Degrees of freedom were adjusted with a Huynh–Feldt correction where the assumption of sphericity was violated.

Tracking performance is shown in Fig. 5. For the amblyopic eye, data were available for 20 children with amblyopia and 29 controls. There was a significant effect of number of targets tracked, $F(2.4, 111.3) = 15.21$, $p < .001$; but no significant effect of group, $F(1, 47) = 3.45$, $p = .07$. This was qualified by a significant interaction between number of targets tracked and group, $F(2.4, 111.3) = 3.29$, $p = .03$. A simple main effects analysis investigating the effect of group at each number of targets tracked revealed that children with amblyopia had a deficit when tracking one target, $F(1, 188) = 5.98$, $p = .02$, and two targets, $F(1, 188) = 5.75$, $p = .02$, but performed the same as controls when tracking three targets, $F(1, 188) = 1.88$, $p = .17$, and four targets, $F(1, 188) = 0.03$, $p = .87$.

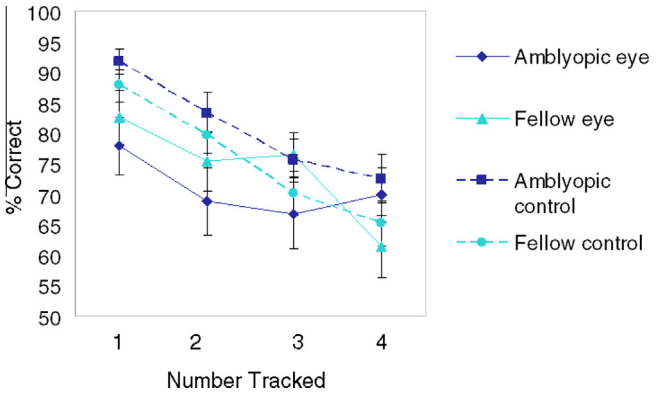

Fig. 5. First-visit mean accuracy scores plotted against number of targets tracked. Error bars represent standard error.

For the fellow eye, data were available for 19 children with amblyopia and 30 controls. There was a significant effect of number of targets tracked, $F(2.6,120.5) = 20.67$, $p < .001$; and no significant effect of group, $F(1,47) = 0.30$, $p = .59$, nor a significant interaction between number of targets tracked and group, $F(2.6,120.5) = 1.88$, $p = .15$. All differences in accuracy between number of targets tracked were significant (all $p < .05$), except between tracking two and three targets ($p > .05$).

We assessed children with amblyopia on an individual basis for tracking deficits by comparing their performance to control children in the same age category, using the method described in Section 2.4. Fourteen of the 22 children with amblyopia had a deficit in tracking performance: six had a deficit in only the eye treated for amblyopia (though three of these had no data for the fellow eye), five had only a fellow eye deficit (though two of these had no data for the amblyopic eye), and three had a deficit in both eyes. Eight children had normal tracking performance in both eyes. Compared to children without tracking deficits, children with any tracking deficit had significantly worse visual acuity in the amblyopic eye (0.37 vs. 0.20 logMAR), $t(20) = 2.34$, $p = .03$, but not the fellow eye, $t(20) = 1.12$, $p = .28$, and significantly worse stereoacuity (360 vs. 897 arc sec), $t(20) = 2.46$, $p = .02$.

3.2.2. Occlusion effects on multiple-object tracking performance

For the multiple-object tracking task, we followed 11 children with amblyopia through occlusion therapy, and 28 controls were tested at the same intervals. There was no group difference in number of days between first and last visit (amblyopia, $M = 362$, SD = 130; controls, $M = 388$, SD = 146; $t(37) = 0.42$, $p = .67$). To assess group differences in tracking accuracy over time on the multiple-object tracking task, an ANOVA was conducted on the accuracy scores to assess the effects of group (amblyopia, control), visit (first, last) and number of targets tracked (one through four). Again, separate analyses were performed, one for the amblyopic eye and one for the fellow eye. Degrees of freedom were adjusted with a Huynh–Feldt correction where the assumption of sphericity was violated.

For the amblyopic eye, data from 10 children with amblyopia and 21 control children were available. There was a significant effect of visit, $F(1,31) = 5.80$, $p = .02$, such that children performed better on the last visit compared to the first visit. There was also an effect of number of targets tracked, $F(3,87) = 17.8$, $p < .001$. Bonferroni-corrected pairwise comparisons revealed that participants were significantly better at tracking one target vs. two ($p = .04$), three ($p < .001$), or four ($p < .001$) targets; and at tracking two targets vs. three ($p = .01$) or four ($p = .01$) targets. There was no difference in performance for tracking three vs. four targets ($p = .99$). There was no significant interaction between visit and number of targets tracked, $F(3,87) = 1.59$, $p = .20$. There was also no effect of group, $F(1,29) = 0.12$, $p = .74$; nor did group interact with any other factors: for visit × group, $F(1,29) = 0.28$, $p = .61$; for number of targets tracked × group, $F(3,87) = 1.09$, $p = .36$; for visit × number of targets × group, $F(3,87) = 0.37$, $p = .78$.

For the fellow eye, data were available from 11 children with amblyopia and 24 controls. There was a significant effect of visit, $F(1,33) = 36.7$, $p < .001$, and a significant effect of number of targets tracked, $F(2.6,85.0) = 27.15$, $p < .001$. This was qualified by a significant interaction between visit and number of targets tracked, $F(2.8,91.8) = 2.94$, $p = .04$. Simple main effects analysis examining the effect of visit at each number of targets tracked revealed that participants performed better at last visit for all number of targets tracked, with a greater increase in performance for tracking four compared to tracking one target ($p < .05$). Again, there was no effect of group, $F(1,33) = 0.28$, $p = .60$; nor did group interact with any other factors: for visit × group, $F(1,33) = 0.38$, $p = .54$; for number of targets tracked × group, $F(2.6,85.0) = 0.25$, $p = .83$; for visit × number of targets × group, $F(2.8,91.8) = 2.03$, $p = .12$.

Assessing the 11 children followed through occlusion therapy on an individual basis, four showed normal tracking performance, three showed a persistent tracking deficit and three developed a deficit. Amblyopic eye data were not available for one child.

3.2.3. Tracking deficits and response to occlusion therapy

The 11 children with amblyopia who were followed through occlusion therapy showed a significant improvement in logMAR visual acuity between their first and last visit for the amblyopic eye ($M = 0.081$, equivalent to 6.5 letters; 95% CI: −.0048 to .104), compared to controls ($M = 0.035$, 2.8 letters), $t(37) = 1.85$, $p = .04$, 1-tailed. The improvement in visual acuity in the fellow eye ($M = 0.039$, 3.1 letters) was not significantly different from controls ($M = 0.043$, 3.4 letters), $t(37) = 0.14$, $p = .89$, 2-tailed. This suggests that occlusion therapy had a positive impact on visual acuity in the amblyopic eye. Despite the improvement, ten of these eleven children continued to have suboptimal amblyopic eye visual acuity (worse than 0.1 logMAR for Regan chart, or 0.2 logMAR for pediatric chart) at last visit.

A positive response to occlusion therapy was defined as improvement in amblyopic eye logMAR acuity of 0.1 (one line) or more. Table 3 displays the proportion of children with persistent, developed or resolved tracking deficits in each eye as a function of response to treatment. Three children (27%) showed a positive response to treatment between their two visits: one maintained normal attentive tracking in both eyes, one resolved a tracking deficit, and one child with a persistent deficit in one eye developed a deficit in the other. Of the remaining eight children who did not respond to occlusion therapy, three maintained normal attentive tracking in both eyes and five retained or developed deficits in at least one eye. There were no significant correlations between the number of hours of prescribed patching before the first visit and tracking accuracy for any number of targets or either eye (all $p > .05$).

Occlusion therapy compliance, occlusion dose (h), and occlusion dose rate (h/day) between patching start and first test visit, and between first and last test visit, were not correlated with having a persistent or developed tracking deficit in at least one eye (all $p > .05$). Change in tracking accuracy between first and last visit was not correlated with compliance, patching dose or dose rate between patching start and first test visit or between first and last test visit (all $p > .05$). The amount of residual amblyopia and the proportion of improvement in the amblyopic eye visual acuity at last visit (both $p > .05$) did not differ between children with or without an initial tracking deficit.

Table 3
Multiple-object tracking deficits and visual acuity in children with amblyopia between first and last visit.

ID	Amblyopic eye			Fellow eye			Occlusion therapy***					
	Acuity first visit (logMAR)	Acuity last visit (logMAR)	Tracking deficit	Acuity first visit (logMAR)	Acuity last visit (logMAR)	Tracking deficit	Prescribed cumulative dose from patch start to first visit (h)	Prescribed cumulative dose from first to last visit (h)	Prescribed dose rate from patch start to first visit (h/day)	Prescribed dose rate from first to last visit (h/day)	Compliance from patch start to first visit (%)	Compliance from first visit to last visit (%)
S02	0.16**	0.21**	NONE	0.11**	0.00	PERSIST	366.0	399.0	1.0	3.0	68.0	75.7
S03	0.19**	0.15**	DEVELOP	0.11**	0.06	DEVELOP	265.4	138.9	1.5	0.6	100.0	Unknown
S04*	0.26**	0.15**	No data	−0.03	−0.06	RESOLVE	698.0	1907.0	1.3	3.1	89.1	69.7
S05*	0.29**	0.15**	NONE	0.04	−0.01	NONE	1170.0	2722.0	6.0	5.3	38.1	52.6
S06	0.10	0.03	NONE	−0.03	−0.05	NONE	489.3	60.7	0.6	0.1	100.0	78.3
S07	0.29**	0.20**	DEVELOP	0.06	0.05	NONE	1505.0	658.0	2.8	3.5	78.2	25.1
S08	0.48**	0.40**	NONE	0.15**	−0.05	NONE	8133.0	2514.0	5.2	6.0	69.4	100.0
S09*	0.45**	0.28**	PERSIST	0.06	−0.14	DEVELOP	Unknown	882.0	Unknown	2.3	Unknown	79.1
S10	0.44**	0.40**	PERSIST	0.05	0.06	RESOLVE	3669.5	1074.0	2.3	3.0	93.9	97.5
S11	0.38**	0.30**	DEVELOP	0.00	−0.10	RESOLVE	6213.0	1427.5	3.8	3.1	84.0	89.5
S12	0.18**	0.26**	NONE	0.00	0.14**	NONE	2644.0	399.0	2.0	1.0	86.7	100.0

* Showed a positive response to treatment (acuity improved 0.1 logMAR or better in the amblyopic eye between lab visits).
** Suboptimal acuity (worse than 0.1 logMAR on Regan chart or 0.2 logMAR on pediatric chart).
*** Calculated from medical charts where information was available.

We were able to obtain follow-up clinical data on treatment success at occlusion stop date for thirteen children who came for at least one multiple-object tracking visit. Nine children (69%) showed 0.1 logMAR acuity or better in the amblyopic eye at patching stop date and were classified as successfully treated (Lin & Chen, 2013). Fig. 6 displays the percentage of children who showed a tracking deficit in each eye at first visit as a function of treatment success. Tracking deficits occurred in both the successful and the unsuccessful treatment groups, but were proportionately more frequent in the unsuccessful group. However, there was no significant association between the presence of a deficit at the first visit and the amount of residual amblyopia or the proportion of amblyopic eye improvement at patching stop date (both $p > .05$). For the eight children who returned for a last test visit, there were also no associations between having a persistent or developed deficit and these measures (both $p > .05$).

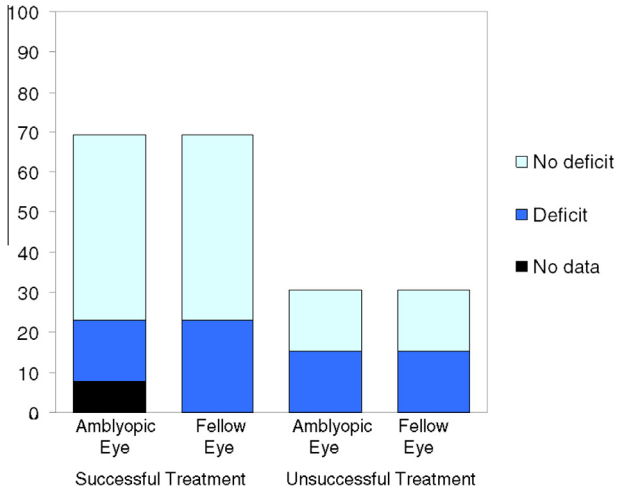

Fig. 6. The relationship between first visit tracking deficits and treatment outcome in the 13 children who had completed occlusion therapy by the end of the study. Successful treatment was defined as an amblyopic eye visual acuity of 0.1 logMAR or better at patching stop date.

3.3. Motion deficits in subtypes of amblyopia

We did not have a large enough sample to statistically assess differences in motion perception deficits by amblyopia subtype. Deficits, however, appeared across all subtypes of amblyopia. For example, Fig. 7 shows persistent deficits in motion-defined form perception in all three amblyopia subtypes (anisometropic, strabismic, or aniso-strabismic amblyopia). The qualitative data suggest that on the motion-defined form task, strabismic and aniso-strabismic subtypes may have a greater percentage of deficits at first visit (83% each) than anisometropic subtypes (44%). By last visit, the aniso-strabismic subtype may have responded the least to occlusion treatment, as 83% had persistent or developed motion-defined form deficits, compared to 44% for anisometropic and 33% for strabismic subtypes. On the multiple-object tracking task, all children with aniso-strabismic amblyopia had tracking deficits at first visit, compared to 29% for anisometropic and 0% for strabismic subtypes. Moreover, all children with aniso-strabismic amblyopia had persistent or developed tracking deficits, compared to 43% anisometropic subtypes and 0% strabismic subtypes.

3.4. Comparison of motion-defined form and multiple-object tracking deficits

Table 4 shows the percentage of deficits for each task in each eye at first and at last visit. There was no significant difference in percentage of participants with initial deficits on multiple-object tracking and motion-defined form tasks ($p = .52$). In addition, logMAR acuities at clinical assessment date, and at first visit were not significantly different between the multiple-object tracking and the motion defined form groups (all $p > .05$).

Nine children with amblyopia completed both the multiple-object tracking and motion-defined form tasks, five of whom came for multiple visits. At first visit, five children had deficits in both tasks, one had a deficit in multiple-object tracking only, one had a deficit in motion-defined form perception only, and the remaining two had normal performance in both tasks. At last visit, two had deficits in both tasks, one had a deficit in multiple-object tracking only, and the remaining two had normal performance on both tasks.

4. Discussion

We examined motion-defined form perception and/or multiple-object tracking deficits in 45 children with amblyopia,

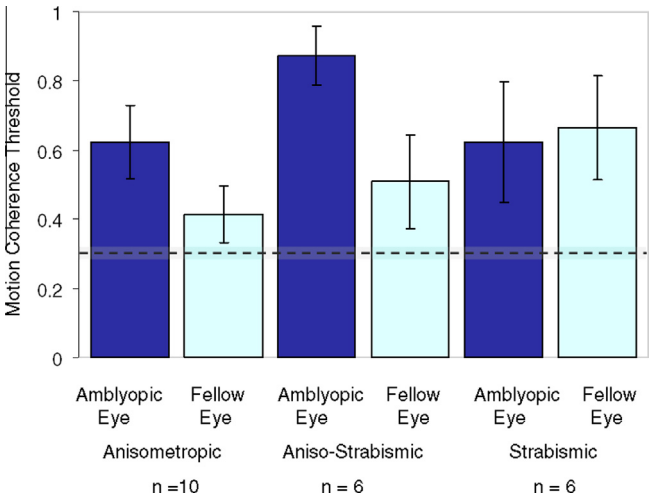

Fig. 7. Last visit motion coherence thresholds on the motion-defined form task as a function of amblyopia subtype. Error bars represent standard error. Dashed line represents control mean coherence threshold + one standard error.

and followed some of them over a year as they underwent occlusion therapy. On average, occlusion therapy led to significant improvement in visual acuity in the amblyopic eye. While many of the children we tested were in the middle of their occlusion therapy and had suboptimal visual acuity, most of the children who finished treatment by the time of data analysis were considered successfully treated based on improved visual acuity.

On the motion-defined form task, we found that children with amblyopia performed significantly worse than controls both with their amblyopic eye and with their fellow eye. Deficits were present in 69% of children in the amblyopia group on their first visit. This confirms our previous findings (Giaschi et al., 1992b; Hayward et al., 2011; Ho et al., 2005; Wang, Ho, & Giaschi, 2007), and extends them to younger children with amblyopia.

On the multiple-object tracking task, we found no robust group differences between children with amblyopia and controls. We did find that 64% of children in the amblyopia group had a tracking deficit in at least one eye on their first visit, and that children with amblyopia had a deficit in tracking one or two targets at their first visit, but these group deficits did not appear in the fellow eye nor did a group effect persist throughout occlusion therapy. The lack of a group difference on the multiple-object tracking task is not consistent with our previous work (Ho et al., 2006; Secen et al., 2011) that did find a deficit in children with amblyopia. Two factors may account for this: first, Ho et al. (2006) used a full-report multiple-object tracking task instead of the partial-report task used here. It may be that the partial-report task is easier and less sensitive to disruptions in attentional processing, resulting in a type of ceiling effect. In addition, the participants in the current study were younger (3–9 years) than those in our two previous studies (Ho et al., 2006: 9–17 years; Secen et al., 2011: 9–36 years). Attentional processes involved in multiple-object tracking are still developing at age 8 years (Trick, Jaspers-Fayer, & Sethi, 2005; Trick, Hollinsworth, & Brodeur, 2009). Therefore, it may be that group differences will emerge at a later age if visual development is arrested in the children with amblyopia. The current results suggest that the deficit in motion-defined form perception is more robust than the deficit in multiple-object tracking.

Only two children with amblyopia were tested before the start of occlusion therapy. We were, therefore, unable to confirm that the motion deficits are present before the start of occlusion. Deficits, however, were observed to develop during treatment in only a few children. Thus, it seems unlikely that treatment is the main cause of the motion deficits reported previously.

In both tasks, children with a deficit at first visit had significantly worse visual acuity in the amblyopic eye, and significantly worse stereoacuity, compared to children with amblyopia who had no motion perception deficits. This suggests that motion perception deficits are more common in children with deeper amblyopia. The motion deficits are not likely to be simply a byproduct of poor visual acuity because many of them occurred in the fellow eye that had good visual acuity (Table 4). In addition, our inclusion criterion of 1.0 logMAR acuity or better in the amblyopic eye was selected to ensure the stimuli would be resolvable, although suboptimal visual acuity can lead to elevated thresholds on motion-defined form tasks (Zwicker et al., 2006).

For the children we followed through occlusion therapy, very few motion deficits resolved on either task. At the last visit, tracking deficits and motion-defined form deficits were present in 55% of children in the amblyopia group. This was not related to compliance or to occlusion dose. The resistance of motion deficits to occlusion therapy is consistent with the finding that the sensitive period for the development of motion perception ends early in life in animal models (Mitchell, Kennie, & Kung, 2009). Thus motion perception may be mature and not very plastic by the time occlusion therapy begins.

First-visit motion deficits were more common in children who had suboptimal visual acuity on their last visit for both multiple-object tracking and motion-defined form tasks. This suggests that such deficits may predict a poor response to occlusion therapy. However, when we examined visual acuity at occlusion stop date, which was sometimes several years after the last visit, most children had achieved visual acuity within normal limits. This questions the predictive value of initial motion deficits, but requires further investigation into clinical versus psychophysical measurement of visual acuity. It is possible that amblyopia takes longer to treat when motion deficits are present.

Although occlusion therapy is simple and relatively non-invasive, it has a broad range of disadvantages: it is distressing to the child, unpopular with parents, and may interfere with educational performance. There is also growing evidence of persistent

Table 4
Percentage of participants with motion deficits for each task.

Visit	Deficit category	% Participants*	
		MD	MOT
First visit motion-defined form (n = 32) tracking (n = 22)	Amblyopic eye deficit	50.0	40.9
	Fellow eye deficit	37.5	36.4
	Deficit present in at least 1 eye	68.8	63.6
Last visit motion-defined form (n = 22) tracking (n = 11)	Amblyopic eye deficit	54.5	45.5
	Fellow eye deficit	31.8	27.3
	Deficit present in at least 1 eye	54.5	54.5

MD = motion-defined form task; MOT = multiple-object tracking task.
* Includes nine first- and five last-visit participants who completed both tasks.

negative effects of patching on aspects of a child's well-being, such as self-esteem (Sabri et al., 2006; Webber et al., 2008). Negative psychosocial effects have been reported in education, occupation, lifestyle and sport in up to 50% of cases (Packwood et al., 1999). It is therefore important to identify children who have perceptual deficits, aside from visual acuity, that are unlikely to be treated successfully with occlusion therapy. Our results suggest that multiple-object tracking and motion-defined form perception fall into this category of untreatable deficits.

McKee, Levi, and Movshon (2003) suggest that a proper assessment of binocularity may be critical to establishment of an appropriate treatment protocol for amblyopia. Amblyopia is conventionally treated as a monocular problem, but new treatments target binocular mechanisms. As described in other articles in this issue, these new treatments are based on dichoptic training to reduce suppression of the amblyopic eye by the fellow eye (Birch, 2013; Hess, Mansouri, & Thompson, 2010a, 2010b; Hess, Mansouri, & Thompson, 2011; Knox et al., 2012; Mansouri, Thompson, & Hess, 2008). These new treatments for amblyopia may be particularly beneficial to children with motion perception deficits.

5. Conclusion

The current prospective study and prior psychophysical studies indicate that many children with amblyopia have deficits in motion perception. The work presented here focused specifically on motion-defined form perception and multiple-object tracking. Deficits in motion-defined form perception appear to be more robust than multiple-object tracking deficits. These deficits may occur in either or both the amblyopic and fellow eye, and unlike visual acuity deficits in the amblyopic eye, they appear to be quite resistant to occlusion therapy. It is yet to be seen if dichoptic training can alleviate motion deficits.

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