Vision Research 114 (2015) 122–134

Contents lists available at ScienceDirect

Vision Research

journal homepage: www.elsevier.com/locate/visres

The effect of occlusion therapy on motion perception deficits in amblyopia

Deborah Giaschi [a,*], Christine Chapman [a], Kimberly Meier [b], Sathyasri Narasimhan [a], David Regan [c]

[a] Department of Ophthalmology and Visual Sciences, University of British Columbia, 4480 Oak Street, Vancouver, BC V6H 3V4, Canada
[b] Department of Psychology, University of British Columbia, 2136 West Mall, Vancouver, BC V6T 1Z4, Canada
[c] Department of Psychology, York University, 4700 Keele Street, Toronto, ON M3J 1P3, Canada

ARTICLE INFO

Article history:
Received 9 September 2014
Received in revised form 7 May 2015
Available online 4 June 2015

Keywords:
Amblyopia
Motion perception
Motion-defined form
Multiple-object tracking
Visual development
Occlusion therapy

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).

* Corresponding author.
E-mail addresses: giaschi@mail.ubc.ca (D. Giaschi), cmcchapman@gmail.com (C. Chapman), kmeier@psych.ubc.ca (K. Meier), sathyasrinarasimhan16@neco.edu (S. Narasimhan), dregan@yorku.ca (D. Regan).

http://dx.doi.org/10.1016/j.visres.2015.05.015
0042-6989/

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).