Available online at www.sciencedirect.com
SCIENCE DIRECT®
Vision Research 45 (2005) 1615–1627
Vision Research
www.elsevier.com/locate/visres

Deficient motion perception in the fellow eye of amblyopic children

Cindy S. Ho [a,*], Deborah E. Giaschi [a,b,*], Catherine Boden [c], Robert Dougherty [d], Roy Cline [b], Christopher Lyons [b]

[a] Department of Psychology, University of British Columbia, Vancouver, Canada V6T 1Z4
[b] Department of Ophthalmology and Visual Sciences, University of British Columbia, Children's and Women's Health Centre of British Columbia, BC's Children's Hospital, 4480 Oak Street, Vancouver, BC, Canada V6H 3V4
[c] Department of Ophthalmology, University of California, San Diego, La Jolla, 92093-0946, USA
[d] Department of Psychology, Stanford University, Stanford, 94305-2130, USA

Received 3 September 2003; received in revised form 14 July 2004

Abstract

The extent of motion processing deficits and M/dorsal pathway involvement in amblyopia is unclear. Fellow eye performance was assessed in amblyopic children for motion-defined (MD) form, global motion, and maximum displacement (D_{max}) tasks. Group performance on MD form was significantly worse in amblyopic children than in control children. Global motion deficits were significantly related to residual binocular function. Abnormally elevated D_{max} thresholds were most prevalent in children with anisometropia. Our findings from these three uncorrelated tasks implicate involvement of binocular motion-sensitive mechanisms in the neural deficits of amblyopic children with strabismic, anisometropic, and aniso-strabismic etiologies.

Keywords: Amblyopia; D_{max}; Form; Motion; Binocular vision

1. Introduction

Amblyopia is a developmental condition that may affect a healthy eye during childhood if it is deprived of normal visual stimulation due to ocular misalignment (strabismus), unequal refractive errors (anisometropia), or both. M/dorsal and P/ventral pathways, the parallel neural pathways governing, respectively, temporal and spatial aspects of visual perception (Merigan & Maunsell, 1993; Ungerleider & Mishkin, 1982; Zeki, 1978), have different periods of development (Atkinson, 1992) and likely have different critical periods or windows of neural plasticity when they are vulnerable to changes such as those induced by abnormal visual stimulation or by amblyopic treatment (Daw, 1998). Therefore, abnormal visual experience early in development could cause deficits to any of the subcortical pathways before the primary visual cortex (V1) and/or the cortical streams at V1 and beyond.

Clinically, reduced visual acuity (VA) on standard tests involving letter or shape recognition, is the diagnostic indicator of amblyopia. Unilateral amblyopia is characterized by reduced VA in the amblyopic eye with normal VA in the fellow eye when tested through an optimal refractive correction. Motion perception is rarely tested clinically but emerging research evidence suggests that it is not spared in amblyopic eyes (Buckingham, Watkins, Bansal, & Bamford, 1991; Ellemberg, Lewis, Maurer, Brar, & Brent, 2002; Giaschi, Regan, Kraft, & Hong, 1992; Hess, Demanins, & Bex, 1997; Kelly & Buckingham, 1998; Paul, Giaschi, Cavanagh, & Cline, 2001; Schor & Levi, 1980a, 1980b; Steinman,

* Corresponding authors. Address: Department of Ophthalmology, University of British Columbia, Room A146, BC's Children's Hospital, 4480 Oak Street, Vancouver, BC, Canada V6H 3V4.
E-mail addresses: cindyh@interchange.ubc.ca (C.S. Ho), giaschi@interchange.ubc.ca (D.E. Giaschi).

0042-6989/$ - see front matter © 2005 Elsevier Ltd. All rights reserved.
doi:10.1016/j.visres.2004.12.009

Levi, & McKee, 1988). It has been suggested that motion perception deficits may provide a measure of neural change and visual loss more sensitive than form perception deficits (Kelly & Buckingham, 1998).

The fellow eye is often assumed to have normal visual function because it demonstrates normal VA. This assumption is likely not valid as numerous reports have claimed abnormal form (Davis et al., 2003; Kandel, Grattan, & Bedell, 1980; Kovacs, Polat, Pennefather, Chandna, & Norcia, 2000; Leguire, Rogers, & Bremer, 1990; Lewis, Maurer, Tytla, Bowering, & Brent, 1992) and motion (Ellemberg et al., 2002; Giaschi et al., 1992; Kelly & Buckingham, 1998; Paul et al., 2001) perception in the clinically unaffected fellow eye.

Fellow eye deficits likely reflect abnormalities associated with binocular mechanisms. Binocular neurons are not dependent on specific input from only one eye but instead can be stimulated through input from either eye. One might speculate that deficits in the fellow eye could result from (a) transfer between the amblyopic and fellow eye through remaining binocular neurons (Leguire et al., 1990); and/or (b) abnormal or modified development of neurons responding to fellow eye stimulation due to abnormal binocular interactions and/or competition (Crewther & Crewther, 1993; Kiorpes & McKee, 1999 (review), McKee, Levi, & Movshon, 2003). Furthermore, perceptual deficits in the fellow eye could be induced, at least in part, by visual deprivation of that eye during occlusion therapy.

As one progresses through the visual pathway beyond area V1, a higher proportion of neurons are binocular (Zeki, 1978). It is widely accepted that amblyopia occurs because of reduced numbers of binocular neurons in V1 (reviewed in Hess, 2001), but the extent to and manner in which binocular neurons in higher visual processing areas are affected by abnormal binocular experience during development is not yet clear. Thus, perceptual deficits in the fellow eye could suggest involvement of extra-striate cortex. It has been suggested, however, that binocular neurons higher in the visual pathway, such as area V5/MT, are less vulnerable to unequal monocular visual input than binocular neurons in area V1 (McColl & Mitchell, 1998). If this were true, one might expect motion perception in the fellow eye to show only subtle deficits in cases of unilateral amblyopia.

Giaschi et al. (1992) previously reported very robust deficits in the fellow eyes of amblyopic children (aged 4–14 years) on a motion-defined (MD) letter identification task that measured minimum speed thresholds. Whether the highly prevalent MD form deficits observed represent a general deficit in motion perception or a specific deficit in form processing that exists despite normal VA in the non-amblyopic fellow eye is still unclear. To investigate the possibility of general motion processing deficits, we looked at performance in the fellow eyes of amblyopic children on three specific psychophysical tasks chosen to represent different aspects of motion processing: coherence thresholds for direction discrimination of global motion, minimum speed thresholds for identification of motion-defined (MD) form, and maximum displacement thresholds for direction discrimination of coherent motion (D_{max}). Performance between amblyopic and control groups on the three motion tasks was compared. D_{max} and global motion stimuli shared the same display parameters. The D_{max} task varied dot displacement and held coherence constant and the global motion task varied coherence but held dot displacement constant. Thus, these two tasks represented orthogonal 1-D slices through the 2-D coherence/displacement motion space. The MD form task used in this study was similar to that used previously by Giaschi and colleagues with the exception that our task involved identification of shapes that preschool children could identify rather than letters, and involved vertical instead of horizontal relative motion cues to minimize the influence of nasal-temporal oculomotor asymmetries.

2. Methods

2.1. Subject selection

2.1.1. Amblyopic group

The amblyopic group consisted of 21 children ranging in age from 4.4 to 11.0 years ($M = 6.9$ years, SD = 1.7 years). The subjects were referred from the Department of Ophthalmology at the Children's and Women's Health Centre of British Columbia. Patients were assessed (by author RC or CL) and classified, based on clinical evaluation and history, into three amblyopic subgroups: strabismic (S) [$M = 7.0$ years, SD = 2.0 years], anisometropic (A) [$M = 6.8$ years, SD = 1.7 years], or aniso-strabismic (A + S) [$M = 7.4$ years, SD = 1.3 years]. None of the subjects included had eccentric fixation, latent or manifest nystagmus, anomalous retinal correspondence, or oculomotor dysfunction with the exception of strabismus. Only the fellow eye was tested. The ages and clinical diagnoses of the amblyopic group are summarized in Table 1. The age range of subjects was kept similar to that used by Giaschi et al. (1992) because their results suggest that children and adults may perform differently on the MD form task.

The Regan 96% contrast letter chart was used to measure VA because it has letter spacing designed to minimize crowding effects and has a logarithmic progression of letter size (Regan, 1988). Line VA was measured monocularly and recorded as a decimal VA measure. For example, a VA measure of 6/7.5 would be expressed as a decimal VA of 0.80. VA in children aged 3–5 years who were unfamiliar with letters was