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Low- and high-level motion perception deficits in anisometropic and strabismic amblyopia: Evidence from fMRI

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ABSTRACT

Maximum motion displacement (Dmax) is the largest dot displacement in a random-dot kinematogram (RDK) at which direction of motion can be correctly discriminated [Braddick, O. (1974). A short-range process in apparent motion. *Vision Research, 14*, 519–527]. For first-order RDKs, Dmax gets larger as dot size increases and/or dot density decreases. It has been suggested that this increase in Dmax reflects greater involvement of high-level feature-matching motion mechanisms and less dependence on low-level motion detectors [Sato, T. (1998). Dmax: Relations to low- and high-level motion processes. In T. Watanabe (Ed.), *High-level motion processing, computational, neurobiological, and psychophysical perspectives* (pp. 115–151). Boston: MIT Press]. Recent psychophysical findings [Ho, C. S., & Giaschi, D. E. (2006). Deficient maximum motion displacement in amblyopia. *Vision Research, 46*, 4595–4603; Ho, C. S., & Giaschi, D. E. (2007). Stereopsis-dependent deficits in maximum motion displacement. *Vision Research, 47*, 2778–2785] suggest that this "switch" from low-level to high-level motion processing is also observed in children with anisometropic and strabismic amblyopia as RDK dot size is increased and/or dot density is decreased. However, both high- and low-level Dmax were reduced relative to controls. In this study, we used functional MRI to determine the motion-sensitive areas that may account for the reduced Dmax in amblyopia In the control group, low-level RDKs elicited stronger responses in low-level (posterior occipital) areas and high-level RDKs elicited a greater response in high-level (extra-striate occipital–parietal) areas when activation for high-level RDKs was compared to that for low-level RDKs. Participants with anisometropic amblyopia showed the same pattern of cortical activation although extent of activation differences was less than in controls. For those with strabismic amblyopia, there was almost no difference in the cortical activity for low-level and high-level RDKs, and activation was reduced relative to the other groups. Differences in the extent of cortical activation may be related to amblyogenic subtype.

1. Introduction

Clinically, amblyopia is characterized by reduced visual acuity in one eye despite normal ocular health and optimal refractive correction. In unilateral amblyopia, the fellow (unaffected) eye demonstrates normal visual acuity. In addition to visual deprivation, amblyopia may be caused by strabismus, anisometropia or a combination of both strabismus and anisometropia.

Psychophysical tests showing visual losses other than reduced visual acuity implicate deficits in both P/ventral (form) and M/dorsal (motion) pathways (Milner & Goodale, 1995; Ungerleider & Mishkin, 1982). In addition to reduced visual acuity, there are well-documented deficits in other aspects of spatial vision such as low-contrast acuity, contrast sensitivity, positional acuity and spatial localization (for reviews see Asper, Crewther, & Crewther, 2000; Levi, 1991). There have also been reports of deficits in temporal and motion processing (Schor & Levi, 1980a; Schor & Levi, 1980b; Steinman, Levi, & McKee, 1988). Evidence for impairment of motion mechanisms in amblyopia has grown and includes reported deficits involving oscillatory movement displacement (Buckingham, Watkins, Bansal, & Bamford, 1991; Kelly & Buckingham, 1998), motion-defined form (Giaschi, Regan, Kraft, & Hong, 1992; Ho et al., 2005), motion after-effect (Hess, Demanins, & Bex, 1997), maximum motion displacement (Ho & Giaschi, 2006; Ho & Giaschi, 2007; Ho et al., 2005), and global motion (Ellemberg, Lewis, Maurer, Brar, & Brent, 2002; Simmers, Ledgeway, Hess, & McGraw, 2003). There have been numerous reports of abnormal motion perception in both the amblyopic and the fellow eye suggesting that these deficits are not well accounted for by reduced visual acuity (or other form perception deficits) in amblyopic eyes (Giaschi et al., 1992; Ho & Giaschi, 2006; Ho & Giaschi, 2007; Ho et al., 2005; Simmers et al., 2003).

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Our recent studies in amblyopia have focused on deficits of maximum motion displacement (Dmax). Dmax is the largest displacement at which the direction of a random-dot kinematogram (RDK) can be reliably discriminated (Braddick, 1974). If the displacement is small and all dots are shifted in the same direction (100% coherence), direction discrimination is not difficult because the motion perceived is smooth and continuous. As the displacement approaches the maximum displacement value (Dmax), direction discrimination of the apparent motion is still possible but more difficult because the motion appears to be less coherent. The value of Dmax may be restricted by the receptive field size of low spatial-frequency-tuned motion detectors at a low-level of motion processing and/or by the efficiency of spatial feature-matching at high levels of motion processing (Nishida & Sato, 1995; Sato, 1998; Snowden & Braddick, 1990).[1] It has been suggested that as dot probability is decreased or dot size is increased, motion processing involves low-level mechanisms to a lesser extent and is biased more toward high-level motion mechanisms (Sato, 1998; Smith & Ledgeway, 2001). There have been reports of amblyopic deficits in Dmax for both low-level and high-level RDKs (Ho & Giaschi, 2006; Ho & Giaschi, 2007). Our findings confirm that this mechanism "switch" is intact in amblyopia but it is associated with an overall decrease in Dmax.

Several other studies of amblyopia suggest that high-level motion processing is more impaired than low-level motion processing. The M pathway in the human visual system projects dorsally and includes high-level, motion-sensitive extra-striate areas: V3A (Tootell et al., 1997), V5/MT+ (Tootell et al., 1995; Zeki et al., 1991) and regions of the posterior parietal cortex (PPC) (Cheng, Fujita, Kanno, Miura, & Tanaka, 1995; Dupont, Orban, De Bruyn, Verbruggen, & Mortelmans, 1994; Orban et al., 2006; Sunaert, Van Hecke, Marchal, & Orban, 1999). Simmers and colleagues reported deficits in MT using first- and second-order global motion stimuli (Simmers, Ledgeway, & Hess, 2005; Simmers et al., 2003) as well as deficits in MSTd using translational, rotational, and radial optic flow patterns (Simmers, Ledgeway, Mansouri, Hutchinson, & Hess, 2006) in an amblyopic population. We have previously reported deficits in high-level attentive tracking (Ho & Giaschi, 2006). Attentive tracking (Cavanagh, 1992) is a high-level motion task that involves feature-matching mechanisms. The results of these studies implicate extra-striate motion-sensitive areas as part of the neural deficit underlying amblyopia. The attentive-tracking deficits seen in amblyopia (Ho et al., 2006) are likely associated with impairment of PPC (to which the dorsal visual pathway projects) because Culham and colleagues identified parietal activation using similar attentive-tracking tasks with functional MRI (Culham et al., 1998). Furthermore, PPC is implicated in high-level motion perception because patients with parietal lesions show deficits in motion perception for high- but not low-level tasks (Battelli et al., 2001).

Although several studies of amblyopia have demonstrated psychophysical deficits consistent with abnormal high-level motion mechanisms, there has been limited direct neuroimaging evidence to date associating extra-striate motion-sensitive brain areas with these behavioral deficits in amblyopic participants. The aim of this study was to investigate the extent to which the high-level (and likely the feature-based) motion system (and PPC) is impaired in amblyopia. The RDK stimulus parameters were kept consistent with those from our earlier studies (Ho & Giaschi, 2006; Ho & Giaschi, 2007). We assessed children with strabismic and anisometropic amblyopia and controls on two luminance-defined, high-level motion conditions (decreased dot density and increased dot size) as well as a low-level baseline (small dots, densely spaced) condition. Given our hypothesis that abnormal neural activity in extra-striate cortex may explain the reported behavioral Dmax deficits, less involvement of dorsal extra-striate areas in amblyopic participants relative to control participants during a direction discrimination task with high-level RDKs (compared to the low-level baseline RDK) was expected.

2. Methods

2.1. Participants

2.1.1. Control group

Four control children were tested, ranging in age from 14 to 16 years ($M = 15.4$ yrs, $SD = 0.9$ yrs). All of the subjects tested were visually mature as Dmax has been shown to reach adult levels between age 7 and 8 years (Parrish, Giaschi, Boden, & Dougherty, 2005). All children included had distance and near monocular line visual acuity (VA) equivalent to or better than 6/6 or 0.4 M, respectively (Jose & Atcherson, 1977). Both acuity cut-off values represent letter size with detail of 1 min when measured at 6 m and 40 cm, respectively. Distance line VA was measured using the Regan 96% contrast letter chart and near VA was measured using the University of Waterloo near vision test card. Stereoacuity, assessed using the Randot Stereotest (Stereo Optical Co., Inc.), was required to be equivalent to or better than 40″. Worth-4-Dot (W4D) testing (reviewed in Rutstein & Daum, 1998, chap. 5) was used to test for fusion and scored to give another measure of binocularity. The scoring was as follows:

5 = constant fusion
4 = intermittent fusion with intermittent diplopia
3 = constant diplopia
2 = intermittent suppression
1 = constant suppression.

All control subjects, when tested in the dark, were required to have a score of 5 when tested at 1 m. No control subject had a history of ocular pathology or abnormal visual development.

2.1.2. Amblyopic group

The subjects were referred from the Department of Ophthalmology at the Children's and Women's Health Centre of British Columbia, and from other local clinics. The ages and clinical details of the amblyopic children are summarized in Table 1. Data were collected from three amblyopic children with strabismus ($M = 14.4$ yrs, $SD = 1.0$ yrs) and four with anisometropia ($M = 14.2$

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[1] Feature-matching is a characteristic of the long-range (but not the short-range) motion system proposed by Braddick (1974). Since Braddick's short-range and long-range classification, several other theories of motion perception have evolved. For example, Cavanagh and Mather (1990) suggest that low-level mechanisms process first-order stimuli (luminance- or color-defined) and that high-level mechanisms process second-order motion stimuli (motion- and stereo-defined). Lu and Sperling (reviewed in 2001) propose three separate motion systems: a first-order system responding to luminance-defined stimuli, a second-order system responding to contrast- or motion-defined stimuli, and a third-order system which is based on the "salience map" of a moving stimulus. Nishida and Sato (1995) propose a model in which low-level and high-level mechanisms are based on spatial-frequency-tuned motion detectors and feature matching mechanisms, respectively (see also Sato, 1998). The mechanism that dominates is largely dependent on the stimulus parameters chosen (see also Smith & Ledgeway, 2001; Snowden & Braddick, 1990). Decreasing dot density and/or increasing dot size of first-order, luminance-defined RDKs create a bias towards high-level motion mechanisms. Nishida & Sato's model is most appropriate for this study given that all motion stimuli used are first-order. Because all stimuli are luminance-defined, this fMRI study differs from those looking at the neural substrates underlying first-order and second-order motion (see for example: Claeys, Lindsey, De Schutter, & Orban, 2003; Dumoulin, Baker, Hess, & Evans, 2003; Dupont, Sary, Peuskens, & Orban, 2003; Nishida, Sasaki, Murakami, Watanabe, & Tootell, 2003; Seiffert, Somers, Dale, & Tootell, 2003; Smith, Greenlee, Singh, Kraemer, & Hennig, 1998) which may not necessarily involve similar high-level mechanisms to those we are studying.