



Impaired spatial and binocular summation for motion direction discrimination in strabismic amblyopia

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ABSTRACT

Amblyopia is characterised by visual deficits in both spatial vision and motion perception. While the spatial deficits are thought to result from deficient processing at both low and higher level stages of visual processing, the deficits in motion perception appear to result primarily from deficits involving higher level processing. Specifically, it has been argued that the motion deficit in amblyopia occurs when local motion information is pooled spatially and that this process is abnormally susceptible to the presence of noise elements in the stimulus. Here we investigated motion direction discrimination for abruptly presented two-frame Gabor stimuli in a group of five strabismic amblyopes and five control observers. Motion direction discrimination for this stimulus is inherently noisy and relies on the signal/noise processing of motion detectors. We varied viewing condition (monocular vs. binocular), stimulus size (5.3–18.5°) and stimulus contrast (high vs. low) in order to assess the effects of binocular summation, spatial summation and contrast on task performance. No differences were found for the high contrast stimuli; however the low contrast stimuli revealed differences between the control and amblyopic groups and between fellow fixing and amblyopic eyes. Control participants exhibited pronounced binocular summation for this task (on average a factor of 3.7), whereas amblyopes showed no such effect. In addition, the spatial summation that occurred for control eyes and the fellow eye of amblyopes was significantly attenuated for the amblyopic eyes relative to fellow eyes. Our results support the hypothesis that pooling of local motion information from amblyopic eyes is abnormal and highly sensitive to noise.

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1. Introduction

Amblyopia is a developmental visual disorder caused by a discrepancy in the images falling on each retina during early development. Under such conditions the visual system develops abnormally resulting in a loss of visual function, typically in one eye (Holmes & Clarke, 2006). Importantly, once amblyopia has developed, correcting the original amblyogenic factor will not fully restore vision to the amblyopic eye, as the visual deficit is cortical in nature (Anderson & Swettenham, 2006; Barnes, Hess, Dumoulin, Achtman, & Pike, 2001; Barrett, Bradley, & McGraw, 2004; Kiorpes, 2006; Kiorpes & McKee, 1999). While amblyopia is primarily thought of as a disorder of spatial vision (Barrett, Pacey, Bradley, Thibos, & Morrill, 2003; Levi, 2006), amblyopes do also exhibit anomalous motion perception (Aaen-Stockdale & Hess, 2008;

Aaen-Stockdale, Ledgeway, & Hess, 2007; Buckingham, Watkins, Bansal, & Bamford, 1991; Constantinescu, Schmidt, Watson, & Hess, 2005; Ellemberg, Lewis, Maurer, Brar, & Brent, 2002; Hess, Demanins, & Bex, 1997; Ho & Giaschi, 2006, 2009; Ho et al., 2005; Kelly & Buckingham, 1998; Kiorpes, Tang, & Movshon, 2006; Levi, Klein, & Aitsebaomo, 1984; Schor & Levi, 1980; Simmers, Ledgeway, Hess, & McGraw, 2003; Simmers, Ledgeway, Mansouri, Hutchinson, & Hess, 2006; Steinman, Levi, & McKee, 1988; Thompson, Aaen-Stockdale, Mansouri, & Hess, 2008). However there is an interesting difference between the spatial and temporal visual deficits that occur in amblyopia. The spatial deficit is known to affect low level visual functions, such as contrast sensitivity and visual acuity (Bradley & Freeman, 1981; Hess, 1979; Hess & Howell, 1977; Levi & Harwerth, 1980), as well as higher level visual functions that require global processing such as contour integration (Chandna, Pennefather, Kovacs, & Norcia, 2001; Hess & Demanins, 1998; Kozma & Kiorpes, 2003). However, based on the current evidence described below, the motion deficit appears to primarily affect tasks that require global integration of

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motion information with local motion perception remaining largely intact.

There is increasing evidence that amblyopia is associated with a deficit in the perception of global motion (Aaen-Stockdale & Hess, 2008; Aaen-Stockdale et al., 2007; Constantinescu et al., 2005; Elleberg et al., 2002; Ho et al., 2005; Kiorpes et al., 2006; Simmers et al., 2003, 2006) and that this deficit is not limited to the amblyopic eye but also affects the fellow fixing eye (Elleberg et al., 2002; Giaschi, Regan, Kraft, & Hong, 1992; Ho & Giaschi, 2006; Ho et al., 2005; Simmers et al., 2003, 2006). Global motion perception typically requires the integration of distributed signal elements and the segregation of these signal elements from noise elements. Therefore it is likely that global motion tasks rely upon processing in extrastriate dorsal stream visual areas such as V5/MT (Britten, Shadlen, Newsome, & Movshon, 1992; Newsome & Pare, 1988). Further psychophysical evidence implicating an extrastriate motion processing deficit in amblyopia includes abnormalities in the motion after-effect (Hess et al., 1997), impaired perception of motion defined form (Giaschi et al., 1992) and elevated D_{\max} thresholds (Ho & Giaschi, 2006, 2007) that persist when the stimuli are high pass filtered (Ho & Giaschi, 2007), therefore implicating abnormal function of high-level, possibly feature tracking mechanisms in amblyopia (Cavanagh & Alvarez, 2005; Ho et al., 2006).

In contrast to the pronounced deficits found for higher level motion perception, local motion perception appears to be largely unaffected by amblyopia (Hess & Anderson, 1993; Hess, Howell, & Kitchin, 1978; Hess, Mansouri, Dakin, & Allen, 2006; Thompson, Hansen, Hess, & Troje, 2007). In addition, the local motion deficits that have been found mainly concern the detection of stimuli with high spatial frequencies (Hess & Anderson, 1993) or low temporal frequencies (Schor & Levi, 1980), therefore implicating low level losses in acuity and contrast sensitivity rather than motion perception per se (Hess & Anderson, 1993). There is also evidence to suggest that in the absence of noise elements, integration of motion information is normal (Hess et al., 2006) or even excessive (Thompson et al., 2008) in the amblyopic visual system and that the motion processing deficit is associated with poor segregation of signal elements from noise elements (Mansouri & Hess, 2006; Thompson et al., 2007). It would appear therefore that while that the spatial impairments in amblyopia extend from low level processing within the primary visual cortex through to extrastriate visual areas, motion impairments may be primarily due to abnormal pooling of visual information within the extrastriate visual cortex (Kiorpes et al., 2006; Simmers et al., 2003).

Consistent with this distinction, neurophysiological investigations have demonstrated that neurons within the primary visual cortex of amblyopic monkeys show abnormalities in their spatial but not their temporal responses when driven by the amblyopic eye (Kiorpes, Kiper, O'Keefe, Cavanaugh & Movshon, 1998). In contrast, extrastriate visual areas appear to demonstrate deficits in both spatial (Movshon et al., 1987) and motion processing (El-Shamayleh, Kiorpes, Kohn, & Movshon, 2010), with motion deficits being most evident for stimuli that require global processing (El-Shamayleh et al., 2010). Human neuroimaging has also indicated both striate and extrastriate deficits for amblyopic eye viewing of grating stimuli (Barnes et al., 2001; Hess, Li, Lu, Thompson, & Hansen, 2010; Hess, Li, Mansouri, Thompson, & Hansen, 2009; Muckli et al., 2006), whereas motion specific deficits appear to be most pronounced in extrastriate visual areas (Bonhomme et al., 2006; Ho & Giaschi, 2009; Thompson, Villeneuve, Casanova, & Hess, 2010).

In order to further investigate the nature of the motion processing deficit in amblyopia we employed a motion direction discrimination task for two-frame motion sequences in which an abruptly presented Gabor patch of suprathreshold contrast was offset by a

variable phase step from the first frame to the second (Nakayama & Silverman, 1985). This paradigm was chosen because task performance is limited by noise that is inherent in the stimulus itself rather than due to the addition of noise elements to which amblyopic vision is known to be highly sensitive (as described above). Specifically, the abrupt presentation of the stimulus generates motion energy in multiple directions (Churan, Richard, & Pack, 2009; Maunsell, Nealey, & DePriest, 1990). Therefore in order identify the direction of motion presented in the stimulus, the visual system must detect the directional signal generated by the small phase displacement against the noise generated by the stimulus onset. In other words the task is limited by the signal/noise ratio of the motion stimulus (Churan et al., 2009; Nakayama & Silverman, 1985).

We quantified motion discrimination thresholds in terms of phase step size (Churan et al., 2009; Nakayama & Silverman, 1985; Tadin, Lappin, Gilroy, & Blake, 2003) for both normal observers and a group of strabismic amblyopes. We measured thresholds at a range of stimulus sizes (5.3–18.5°), under both monocular and binocular viewing conditions and at a fixed high and suprathreshold low contrast in order to assess the effects of spatial summation, binocular summation and contrast on motion discrimination. The spatial frequency of our stimuli was fixed at 0.5 cpd to minimise spatial contrast sensitivity differences between amblyopic and non-amblyopic eyes (Hess, 1979) and to optimize the effects of binocular summation on motion perception (Rose, 1978, 1980). We found that for high contrast stimuli, task performance was equivalent for the amblyopic observers and the control observers confirming that local motion measurements are normal in amblyopia. However for the low contrast stimuli we observed a pronounced binocular summation effect for normal observers that was absent for the amblyopic observers. In addition we found a strong spatial summation effect for the normal eyes of controls and the fellow fixing eyes of amblyopes. However the effect of spatial summation was significantly attenuated for amblyopic eyes relative to fellow fixing eyes, and this loss of spatial summation was independent of any reduction in task performance due to impaired contrast sensitivity. Given that sensitivity to spatial displacement is similar between V1 and MT (Pack, Conway, Born, & Livingstone, 2006), we suggest that our results are due to a specific impairment in the extrastriate mechanisms responsible for spatial summation.

2. Methods

2.1. Participants

Five observers with strabismic amblyopia and five control observers with normal or corrected to normal vision took part in this study. Details of the amblyopic observers can be found in Table 1. Acuity in the amblyopic eye ranged from 20/40 to 20/70, thus our amblyopic sample can be characterised as mild to moderate in terms of their acuity loss. All amblyopic and control participants were experienced psychophysical observers and all were naive to the purpose of the study. All study protocols were approved by the institutional ethics committee and were in accordance with the Declaration of Helsinki.

2.2. Apparatus and procedure

Stimulus design, apparatus and general procedural details were that same as those used by Churan et al. (2009). Stimuli were generated using a standard Pentium 3 PC computer using Matlab v7.0 and the Psychophysics Toolbox (Brainard, 1997; Pelli, 1997) and displayed using a CRT Electrohome 8000 projector with a spatial

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