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The amblyopic deficit for global motion is spatial scale invariant

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ABSTRACT

Humans with amblyopia display anomalous performance for global motion discrimination. Attempts have been made to rule out an explanation based solely on the visibility loss in lower visual areas. However, it remains a possibility that the altered scale over which local motion is processed in V1 might lead to reduced efficiency of global motion processing in extra-striate cortex. We use stimuli composed of spatial frequency bandpass elements, equated for visibility, to show that the global motion deficit in amblyopia for both fellow and amblyopic eyes is still present once impairments in low-level processing have been factored out. This residual deficit appears to be spatial scale invariant and the relative deficit between the eyes shows a dependence on stimulus speed. We believe that this rules out an explanation of the amblyopic global motion deficit based solely on local motion input. We suggest instead that, in addition to low-level deficits, motion processing in a broadband, extra-striate, global motion mechanism is impaired in amblyopia.

1. Introduction

The major features of amblyopia ("lazy eye") are a typically unilateral loss of visual acuity of at least a factor of two and a loss of contrast sensitivity in the affected eye. It is a developmental disorder normally associated with, but not necessarily caused by, strabismus, anisometropia or form deprivation (Asper, Crewther, & Crewther, 2000a, Barrett, Bradley, & McGraw, 2004) and is not connected to any evident pathology in the visual system. Individuals with amblyopia show abnormal distortions of spatial stimuli (Bedell & Flom, 1981) and a marked contrast sensitivity loss at high spatial frequencies (Hess & Howell, 1977). They also show some abnormalities of temporal processing (Asper, Crewther, & Crewther, 2000b) and unsteady and inaccurate fixation (Brock & Givner, 1952).

The locus of the acuity and contrast impairment is most likely visual area V1 (Hess, 2001), where neurons show abnormalities in spatial scale, eye dominance and binocular organisation (Kiorpes, 2006; Levi, 2006). Contrast sensitivity for motion appears to be normal in amblyopia at low spatial frequencies (Hess, Howell, & Kitchin, 1978) but contrast thresholds are impaired at high spatial frequencies and low temporal frequencies (Hess & Anderson, 1993; Manny & Levi, 1982; Schor & Levi, 1980). Oscillatory motion detection requires larger displacements in amblyopia, and this is probably related to the shift in spatial resolution (Buckingham, Watkins, Bansal, & Bamford, 1991). Functional magnetic resonance neuroimaging (fMRI) shows reduced activation of V1 from the amblyopic eye (Barnes, Hess, Dumoulin, Achtman, & Pike, 2001). There is also fMRI evidence of active suppression of V1 by the fellow eye (Conner, Odom, Schwartz, & Mendola, 2007).

In addition to the early contrast and acuity deficits in amblyopia, evidence is now mounting for separate, downstream impairments of "higher-level" processes traditionally associated with extra-striate visual areas. Amblyopes show deficits in numerating features (Sharma, Levi, & Klein, 2000) and representation of faces (Lerner et al., 2003), and motion-defined form may be impaired in amblyopia (Giaschi, Regan, Kraft, & Hong, 1992). However, the proposed extra-striate impairment in amblyopia that has perhaps generated the most interest is the deficit to global motion processing.

In a series of recent studies, it has been demonstrated that human strabismic amblyopes exhibit a performance deficit for the direction–discrimination of global motion, measured as signal-to-noise ratios for random dot kinematograms (RDKs), that cannot be accounted for by the amblyopic contrast sensitivity loss (Simmers, Ledgeway, Hess, & McGraw, 2003). This deficit is extensive, occurring for translational, rotational and radial motion, for first- and second-order motion as well as affecting both fellow and amblyopic eyes equally (Aaen-Stockdale, Ledgeway, & Hess, 2007; Simmers, Ledgeway, Mansouri, Hutchinson, & Hess, 2006). Similar results have also been reported in deprivation amblyopia (Constantinescu, Schmidt, Watson, & Hess, 2005; Ellemberg, Lewis, Maurer, Brar, & Brent, 2002) and in amblyopic children (Ho et al., 2005).

By manipulating the contrast of the global motion stimulus and assessing whether the deficit is best explained by a shift along the contrast as opposed to the motion coherence axis, Simmers et al.

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(2003), (2006) provided an assessment as to whether the global motion deficits could be simply explained by the known contrast sensitivity loss in V1. It was concluded that in the majority of cases there were global motion deficits that could not be so explained, providing evidence for a primary processing deficit in extra-striate cortex (e.g., middle temporal visual areas V5/MT). Neuroimaging has recently shown that motion-selective areas V5 and V3A show reduced activation to a motion stimulus by the amblyopic eye (Bonhomme et al., 2006).

While the above analysis is suggestive that the loss of global motion sensitivity is not a consequence of altered visibility of the elements or the scale over which the low-level analysis occurs, it is not definitive because the elements used were spatial frequency broadband and the scale over which the motion was computed by low-level detectors, as a consequence, was indeterminate. Ideally one would use spatially bandpass elements (e.g., Baker & Hess, 1998) whose contrast is equal multiples above motion threshold for any comparison of the normal and amblyopic performance. This would ensure that the elements themselves were of equal detectability (to the low-level motion system) and that the scale of motion analysis would be the same between eyes.

We studied a group of strabismic and strabismic-anisometropic amblyopes using global motion stimuli (RDK) in which the individual 'dots' were radial log-Gabors. In this manner we ensured that the local elements were spatially bandpass and DC-balanced (Field, 1987). We have used this same stimulus recently to demonstrate that global motion extraction in normal observers is equally efficient throughout the visual field, once spatial resolution and contrast sensitivity differences are taken into account (Hess & Aaen Stockdale, 2008). We presented all stimuli at the same multiple of motion direction–discrimination threshold to ensure that any global motion deficit that we measure is uncontaminated by the contrast detection anomaly present at that scale in early visual areas. This approach allows a direct estimate of the spatial scale dependence of the global motion deficit believed to reside in extra-striate cortex.

2. Methods

2.1. Observers

The study conformed to the Declaration of Helsinki regarding the use of human subjects. All observers gave informed consent after an explanation of the study. The six non-amblyopic observers consisted of the two authors and four other experienced observers naïve to the purpose of the experiment. All normal observers had normal visual acuity or wore their prescribed correction. Average age of the non-amblyopic observers was 34 (*SD* 10) years.

The details of the eleven amblyopic participants are given in Table 1. Average age of the amblyopic participants was 31 (*SD* 8) years. Most of the amblyopes collected data at more than one speed. Due simply to the availability of amblyopic observers, four amblyopes collected data at all three speeds, four amblyopes collected data at two speeds and three amblyopes collected data at only one speed. This meant that there were nine amblyopic observers for the two lower speeds and six observers for the highest speed.

One strabismic amblyopic observer (ADG) improved her acuity remarkably during the course of testing. When we began testing, she showed acuity of 20/32 in her fellow eye (FE) and 20/50 in her amblyopic eye (AE). This improved to the point where she could not be considered amblyopic (FE: 20/20, AE: 20/25). As she was amblyopic when she began the experiment her data were retained in the analysis. However, this observer's individual data are highlighted in blue in Fig. 2. We can, for the moment, only speculate as to how this subject may have improved her vision. This subject had participated in several experiments over some months, therefore perceptual learning may have played a role (Levi, 2005; Li, Young, Hoenig, & Levi, 2005; Polat, Ma-Naim, Belkin, & Sagi, 2004).

Most of the amblyopic observers had previously participated in other experiments in our lab, whilst some of the normal observers were new to psychophysical experiments. We therefore find it unlikely that differential experience of psychophysical tasks is responsible for any differences between the groups.

Table 1
Clinical details of the amblyopic observers

Observer	Birth year	Acuity with correction	Stereo	Prescription	Alignment	Type	Speeds tested (°/s)
ED	1960	R +0.75 L +0.75	5/10	R +0.75 L +0.75	L ESO 4°	S	5.7, 11.4, 22.8
JL	1977	R 20/20 L 20/63	0/10	R plano L +2.5	L E × O 5°	SA	5.7, 11.4, 22.8
ML	1982	R 20/80 L20/25	00/10	R +1.0/−0.75 × 590° L −3.25	R ESO 6°	SA	5.7, 11.4
GN	1976	R 20/60 L 20/25	0/10	R −9 L −1/−0.5 10°	R E × O 10°	SA	5.7
VD	1982	R 20/20 L 20/63	0/10	None	L ESO 1°	SA	11.4
SDP	1971	R 20/20 L 20/40	6/10	R −0.5/−0.25 0° L −0.75/−0.5 0°	R corrected with surgery	S	5.7
ADS	1984	R 20/20 L 20/80	0/10	R +0.25 L −4.75	R ESO 15°	SA	5.7, 11.4, 22.8
ADG	1977	R 20/20 L 20/25	5/10	R −3.75 +1.25 55° L −4 −1.75 35°	L corrected with surgery	Rec	5.7, 11.4
KG	1984	R 20/40 L 20/25	5/10	R +4.00 −1.75 30° L +3.75 −0.75 150°	R E × O 2°	S	11.4, 22.8
AM	1963	R 20/20 L 20/400	0/10	None	L ESO 23°	SA	5.7, 11.4, 22.8
BH	1980	R 20/20 L 20/63	5/10	R +0.75 L +0.75	L ESO 4°	SA	5.7, 11.4
GH	1962	R 20/20 L 20/63	?		L E × O 6°	SA	22.8

R, right eye; L, left eye; ESO, esotropic; EXO, exotropic; S, strabismic; SA, strabismic-anisometropic; Rec, recovered.

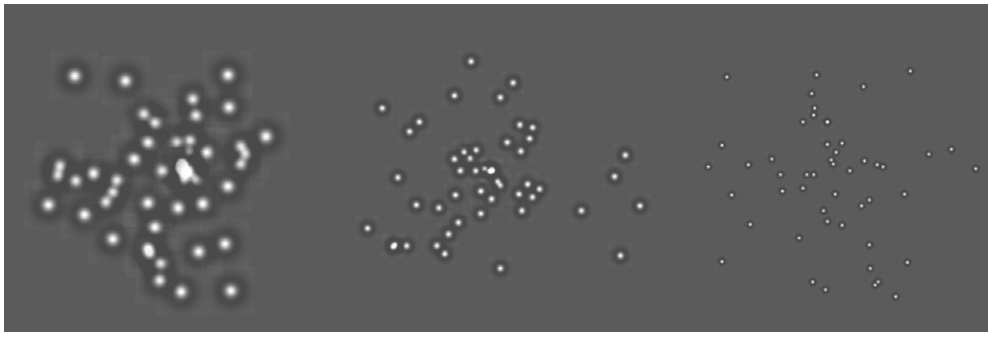

Fig. 1. Single frames from RDK stimuli composed of isotropic log-Gabor elements. From left to right: peak frequencies of 1, 2 and 4 c/deg.

2.2. Apparatus and calibrations

Stimuli were displayed on a Sony Trinitron E500 monitor driven by a *Bits++* device (Cambridge Research Systems, Rochester, UK) with 14 bits contrast resolution, connected to a Macintosh G4 computer running the PsychToolBox software (Brainard, 1997; Pelli, 1997) under *Matlab* 5.2. Display resolution was 1024 × 768 pixels and frame rate was 60 Hz. The monitor was gamma-corrected in software with lookup tables using luminance measurements obtained from an *Eye-One Display 2* calibration device (*Gretag Macbeth, Grand Rapids, MI*). The monitor was viewed in a dimly lit room. The mean luminance of the display was 21 cd/m2. The stimuli were viewed at 60 cm, and the display subtended 35 × 27 deg of visual angle. Stimuli were generated on-line, and a new stimulus was generated for each presentation.

2.3. Stimuli

Stimuli were random dot kinematograms (RDK), in which the 'dots' were limited lifetime, isotropic, bandpass elements (Fig. 1). The signal direction was either left or right (translational motion). Stimulus area, dot number, duration, and interstimulus interval were the same for all experimental conditions. Each stimulus frame had 50 elements, initially randomly distributed over a circular aperture area of radius 6 deg when viewed from a distance of 60 cm. Each element was displaced every frame in either a signal or random direction for 100 ms (life-time of 6 frames). Once the lifetime of an element expired, the element was randomly relocated inside the circular aperture. When moving outside the circular aperture, the elements were repositioned on the opposite side of the circular aperture relative to their motion direction. The temporal phase of each element was randomized in the first frame of each motion sequence, so they appeared and disappeared randomly in time according to a uniform distribution. All elements in the display moved at the same speed: either 5.7, 11.4 or 22.8 °/s depending on the condition, resulting in displacements of 0.095, 0.19 and 0.38° each frame.

The signal-to-noise ratio of the stimulus was reduced in a staircase fashion as described below to ascertain the motion coherence threshold for that condition. The entire stimulus presentation lasted 500 ms (30 frames). Responding initiated the next trial.

The dot elements were isotropic log-Gabors (Field, 1987; Hess & Aaen Stockdale, 2008). These DC-balanced elements were generated in the Fourier domain according to:

$$\mathrm{IsoLogGauss}(f) = exp((-(log(f/fo))^2)/(2 * log(sigmaOnf)^2))$$

with the constraint of IsoLogGauss(0) = 0, then converted to the spatial domain after inverse Fourier transform:

$$\mathrm{IsoLogGabor}(r) = \mathrm{invFFT}(\mathrm{IsoLogGauss})$$

where r is the radial position, f is the radial frequency, fo is the peak frequency, *sigmaOnf* defines the spatial bandwidth (1/1.5 corresponding to about 1.5 octaves). When we refer to "spatial frequency" being varied between conditions, this refers to the peak frequency (fo).

If overlapping of the elements occurred, luminance was added, with the constraint that the luminance could not exceed the maximum value of a single element.

2.4. Procedure

The task was a 2AFC direction–discrimination of an RDK composed of isotropic log-Gabor elements of various spatial frequencies (1, 2 and 4 c/d) and speeds (5.7, 11.4 and 22.8 °/s). Subjects provided their responses by pressing keyboard keys associated with left and right motion, respectively. Auditory feedback was given after each trial. A white fixation mark was briefly presented at the beginning of each trial in the centre of the display. For the amblyopes, both eyes were tested monocularly. The normals viewed the stimuli monocularly with a randomly assigned eye.

Contrast thresholds were measured at an RDK coherence of 70%. In the staircase procedure used, the stimulus contrast was reduced after three correct responses (by 50% before the first reversal, and 12.5% after the first reversal), and increased after one wrong response (by 25%).[1] Each session ended after five reversals and the mean of the last four reversals was taken. For the measurement of contrast thresholds, three staircases were simultaneously interleaved. The thresholds reported are the average of 3–5 staircases for each condition. A few practice staircases were run before the experiments commenced.

Coherence thresholds were then measured using the same staircase procedure, but reducing the ratio of signal- to noise-dots rather than the contrast. Coherence threshold staircases were not interleaved. Instead, several (typically 3–5) staircases were obtained and spatial frequency, speed and eye were randomised between staircases. The contrast of the elements was held constant at a multiple of contrast threshold for the relevant spatial frequency and eye. Typically this was 5× contrast threshold, but for a few observers we needed to drop this to 4× or 3× for the higher speeds. Crucially, the contrast of the stimulus was always the same multiple of discrimination threshold across

[1] This 3-down-1-up staircase procedure has been assumed by many authors to converge on a performance level of 79.37%, but Garcia-Perez (1998) points out that this only occurs when steps up and down are equal in size. For the step sizes used here, the threshold is closer to 86% and this may vary according to a variety of factors: the spread of the underlying psychometric function, the number of trials and the number of reversals. Our staircase technique implements recommendations made by Garcia-Pérez to maximise reliability.

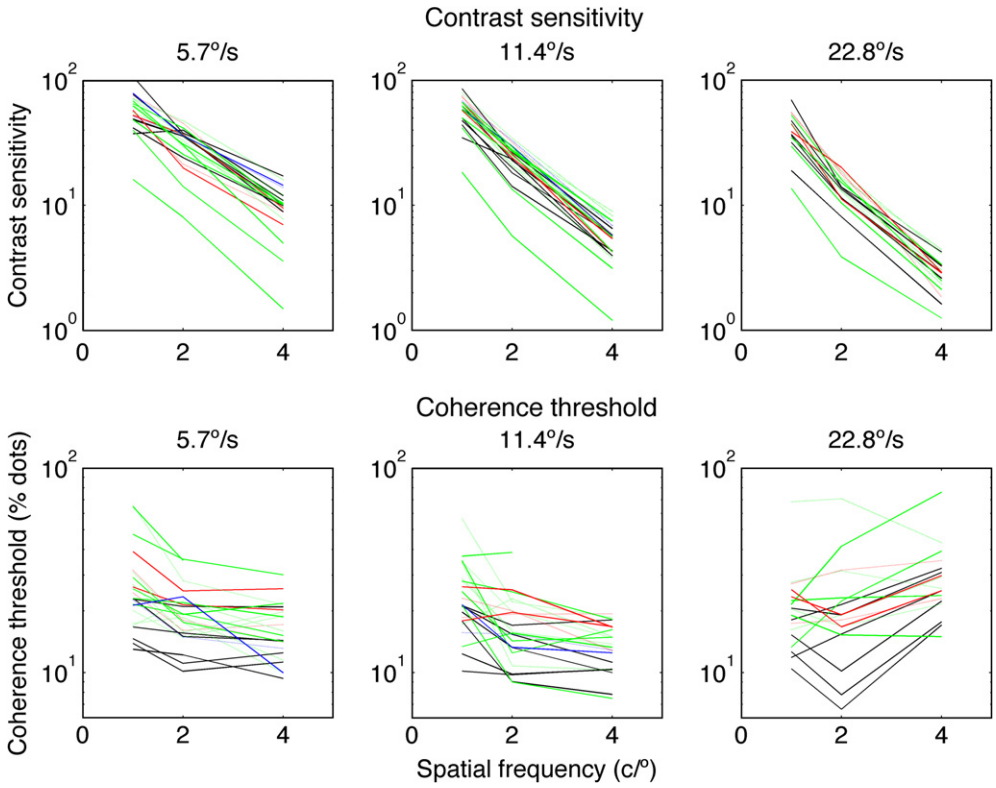

Fig. 2. Contrast sensitivity (top row) and Coherence thresholds (bottom row) for all observers. Solid black lines show data from normal observers, red lines show strabismic observers, green lines show strabismic-anisometropic observers and blue lines show the data from one subject who recovered acuity to normal levels during the course of the experiment. Solid lines are from amblyopic eyes, dotted lines are from fellow eyes.

spatial frequency, controlling for any differences caused by differential contrast sensitivity.

3. Results

3.1. Contrast sensitivity

Results for all amblyopic and normal subjects are shown in Fig. 2 (top). Average results are shown in Fig. 3 (top). Contrast sensitivity (1/threshold) for fellow eyes is similar to or above that for normal eyes at all spatial frequencies and speeds. For amblyopic eyes, performance is worse than normal and this difference is more obvious at low speeds and high spatial frequencies, supporting previous work (Hess & Howell, 1977; Manny & Levi, 1982).

Two-way ANOVAs were run on the contrast sensitivity data showing a main effect of spatial frequency at all speeds (low: $F(2,62) = 9.43$, $p < 0.01$; moderate: ($F(2,62) = 13.37$, $p < 0.01$; high: $F(2,45) = 81.1$, $p < 0.01$), with performance falling off with increasing spatial frequency.

Although viewing eye (amblyopic, fellow or normal) did not show a main effect for any speed (low: $F(2,62) = 2.88$, NS; moderate: $F(2,62) = 1.44$, NS; high: $F(2,45) = 1.45$, NS), there was a significant interaction between eye and spatial frequency at both low ($F(2,62) = 2.93$, $p < 0.05$) and moderate ($F(2,62) = 3.58$, $p < 0.025$) speeds. This appears to be the result of a greater AE deficit at higher spatial frequencies. This high spatial frequency selective deficit had disappeared at high speeds ($F(2,45) = 0.43$, NS).

3.2. Coherence thresholds

Results for all amblyopic and normal subjects are shown in Fig. 2 (bottom). Average results are shown in Fig. 3 (bottom). Coherence thresholds for global motion in normal observers decrease gradually with increasing spatial frequency at low and moderate speeds (fig. 3, bottom). At higher speeds, thresholds show a more u-shaped profile.

At low speeds, amblyopic eyes show an increase in coherence thresholds of ~1.7× that of normal eyes. Fellow eyes also show a smaller increase of ~1.3×. This results in a significant main effect for viewing eye ($F(2,62) = 7.79$, $p < 0.01$). At moderate speeds, there is a main effect of viewing eye ($F(2,62) = 5.73$, $p < 0.01$), with amblyopic observers demonstrating a global motion deficit of around 1.5× compared to normal eyes, but the difference between the amblyopic and fellow eyes has completely disappeared. At high speeds, amblyopic eyes still demonstrate a global motion deficit of around 1.5× relative to normal, whilst fellow eyes (at least for spatial frequencies 1–2 c/deg) show a deficit of around 2× ($F(2,45) = 4.39$, $p = 0.01$).

Crucially, although thresholds show a main effect of spatial frequency at both low ($F(2,62) =$, $p < 0.025$) and moderate ($F(2,62) = 9.91$, $p < 0.01$) speeds, there is no statistically significant interaction between eye and spatial frequency (low: $F(2,62) = 2.16$, NS; moderate: $F(2,62) = 1.28$, NS). Instead, the amblyopic deficit for global motion appears to remain constant across the range of spatial frequencies tested.

In the fast condition, there is no statistically significant main effect of spatial frequency ($F(2,45) = 1.95$, NS) and no interaction between eye and spatial frequency ($F(2,45) = 0.63$, NS). Amblyopic eye performance is a constant 1.5× normal performance across spatial frequency.

At the highest spatial frequency tested in the fast condition, fellow eye performance appears to have improved to near-normal levels. However, normal performance on this condition was already quite poor and we believe that this merely reflects a ceiling effect. Additionally, the size of the displacements relative to the spatial frequency of the elements in this condition means that mo-

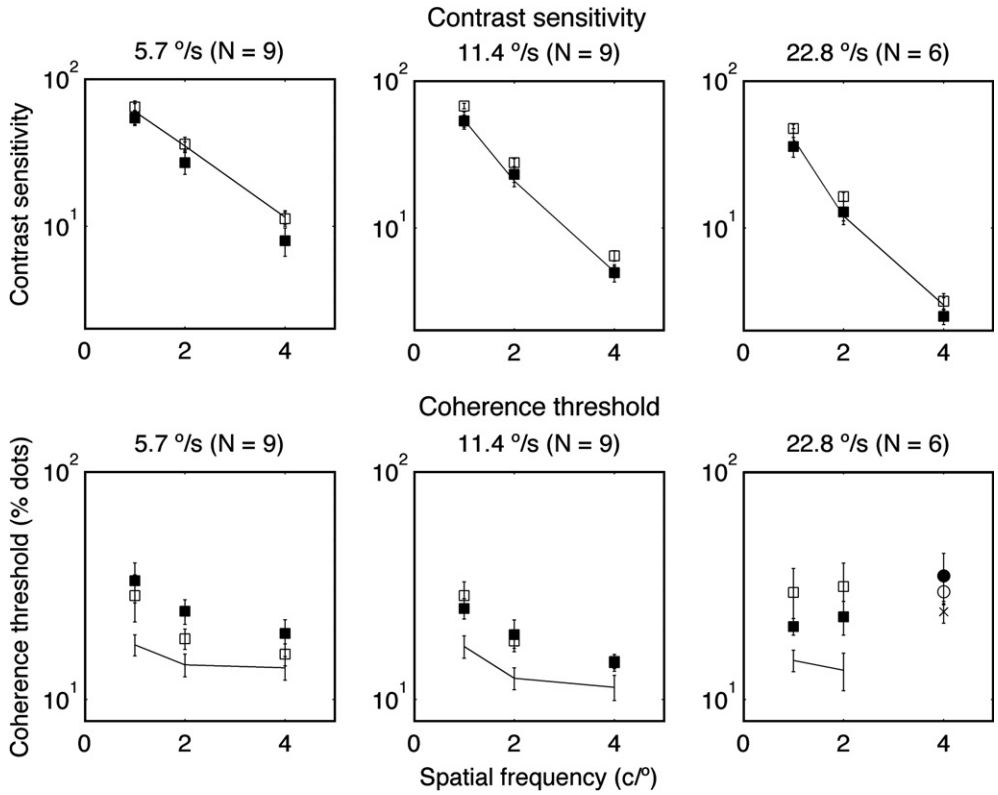

Fig. 3. Contrast sensitivity (top row) and Coherence thresholds (bottom row) for amblyopic and normal observers at three different speeds. Datapoints show group means and error bars show ±1 standard error. Solid symbols refer to amblyopic eye data and open symbols show fellow eye data. Normal data are shown by the solid line. The datapoints at the highest spatial frequency in the highest speed condition are shown with different symbols, as this stimulus was only visible to second-order mechanisms.

tion analysis of this particular stimulus was probably restricted to second-order mechanisms.

4. Discussion

Local motion processing in amblyopia shows some abnormalities related to acuity and contrast sensitivity deficits. Displacement thresholds show a dependence upon grating acuity (Levi, Klein, & Aitsebaomo, 1984). The contrast sensitivity measurements obtained here support previous work that has shown greater impairments in local processing at high spatial frequencies and low speeds (Hess & Anderson, 1993; Hess & Howell, 1977; Manny & Levi, 1982; Schor & Levi, 1980). In addition, there are reports (Ho & Giaschi, 2006; Ho et al., 2005) of anomalous local motion processing in human amblyopes under specific conditions (i.e., near D_{max}).

Our approach to measure contrast thresholds for motion direction–discrimination and to present all stimuli at comparable suprathreshold levels for the subsequent measurement of global coherence was designed to compensate for any contrast- or acuity-dependent losses in local motion processing.

4.1. Spatial scale of global motion processing

By the use of spatial frequency narrowband stimuli equated for detectability we have been able to show that the global motion deficit in amblyopia for both fixing and fellow amblyopic eyes is sizeable (1.5–2 times normal threshold) and spatial scale invariant. The fact that we could measure a deficit at all for stimuli equated for motion visibility adds further weight to the conclusions of Simmers et al. (2003), suggesting that the global motion deficit in amblyopia is not a consequence of the already documented low-level deficits in contrast sensitivity or local motion sensitivity. This research also supports the findings of Constantinescu et al. (2005), but with a larger population rather than a single subject. Thus there is likely to be a primary deficit at the level of the extra-striate cortex where global motion is processed.

The spatial scale of global motion processing has been investigated previously by Kiorpes, Tang, and Movshon (2006). They measured behavioural responses to global motion stimuli from the fellow and amblyopic eyes of monkeys made artificially strabismic or anisometropic. They measured signal-to-noise ratios for discrimination of the global motion of broadband random dot kinematograms. Global motion sensitivity functions from the amblyopic eye were shifted toward larger dot displacements relative to the fellow eye. The authors argued that this shift was correlated with the shift in spatial resolution measured by the contrast sensitivity function, thereby implying that at least part of the global motion deficit can be explained by low-level factors (i.e., a change in the spatial scale of low-level contrast sensitivity or local motion detection). It is likely that the observed *shift* in spatial scale is the result of low-level abnormalities in motion detection (Hess & Howell, 1977; Levi et al., 1984; Manny & Levi, 1982), whilst the observed *reduction* in motion sensitivity in the amblyopic eye is a result of higher-level impairments. Our results are entirely consistent with this. By using narrowband elements presented at constant suprathreshold contrasts, we hope we have eliminated local motion abnormalities and obtained a better measure of the global motion deficit.

Both eyes in amblyopic observers show similar global motion deficits and these deficits are similar across spatial scale. Similar performance on both eyes implicates the impairment of a high-level binocular area, and has been noted previously (Aaen-Stockdale

et al., 2007; Ho et al., 2005; Simmers et al., 2003). Indeed, some studies have found better performance in the amblyopic eye on temporal tasks (Fahle & Bachmann, 1996). The fact that the fellow eye may be equally impaired in global motion tasks, perhaps due to deficits in binocular areas such as V5/MT (Born & Bradley, 2005), suggests that comparisons should be made not just between the two eyes of amblyopes, but also with the eyes of normal control observers.

One limitation of our approach of maintaining all stimuli at a constant multiple of motion detection threshold is that we were not able to test a wider range of spatial scales. However, in the context of the isotropic log-Gabor stimuli used, the range of spatial frequencies covered is as broad as we could make it without varying other factors such as dot density and stimulus area. Testing a larger range with narrowband stimuli such as ours was simply not feasible due to the overlapping of individual elements at low frequencies and the inability of observers to resolve elements of higher spatial frequencies. Other studies have achieved greater ranges of spatial frequency. Kiorpes et al. (2006) tested over a range of 0.5–30 c/deg, but this was not spatial frequency *per se,* but rather is the *equivalent* spatial frequency computed from the displacement of otherwise broadband dots. A study with a single deprivation amblyope showed a global motion impairment in both amblyopic and fellow eyes between 3 and 12 c/deg, a range similar to that used in this study (Constantinescu et al., 2005). Our study supports their conclusion that the global motion deficit is nonselective for spatial scale.

It could be argued that the stimuli of highest spatial frequency, in the fastest condition, are not activating the same motion pathway as the other stimuli. These log-Gabors were displaced more than one cycle of their wavelength, which would render them invisible to first-order motion sensors (Adelson & Bergen, 1985; van Santen & Sperling, 1985; Watson & Ahumada, 1985), but not to mechanisms that could detect the motion of the contrast envelope of each element. The size of the displacements relative to the spatial frequency of the elements in this condition means that this particular stimulus was probably restricted to analysis by second-order mechanisms (Cavanagh & Mather, 1989; Chubb & Sperling, 1988), which have been shown to play a role in global motion detection (Baker et al., 1998). This dependence upon second-order mechanisms may explain the higher thresholds for this condition. This may not be a problem for our study since there is widespread agreement that the two pathways are combined previous to, or at the level of, global motion analysis (Albright, 1992; Ledgeway, Hess, & McGraw, 2002; Lu & Sperling, 2001; Stoner & Albright, 1992; Wilson, Ferrera, & Yo, 1992) and as such the magnitude of global motion deficits measured with first- or second-order stimuli are highly correlated (Aaen-Stockdale et al., 2007).

Although, in our study, both amblyopic and fellow eyes showed similar global motion deficits, the relative size of the global motion deficit between the eyes varies with speed. At low speeds, amblyopic eyes are worse than fellow eyes, but at higher speeds this difference disappears, then reverses. Our data suggest that the relative size of the amblyopic global motion deficit between amblyopic and fellow eyes varies with speed, not spatial scale, but this warrants further investigation.

4.2. Nature of the deficit

It would be counter-productive to blindly integrate all motion signals, when some of those motion signals may be irrelevant or belong to a different object. In "noise-less" global tasks, where all local elements contribute to completing the task, amblyopes display normal performance for form (Mansouri, Allen, Hess, Dakin, & Ehrt, 2004) and motion (Hess, Mansouri, Dakin, & Allen, 2006; Thompson, Aaen-Stockdale, Mansouri, & Hess, 2008) but display anomalous performance in tasks involving orientation or motion noise (Aaen-Stockdale et al., 2007; Simmers, Ledgeway, & Hess, 2005; Simmers et al., 2003; Simmers et al., 2006). Speculatively, the underlying deficit, that we here show is spatial scale invariant, may involve an upset in the balance between the two complementary processes of integration and segregation. Mansouri and Hess (2006) present a model of segregation/integration of global orientation and motion based on the equivalent noise model of Dakin (2001). In this model, normal observers can extract the mean direction/orientation of a population, which is likely to signify the signal, and exclude from integration local elements that are very different from this mean (segregation). Amblyopic observers, however, are impaired at this segregation stage and integrate all local elements inappropriately. The model predicts amblyopic performance, relative to normal, on form and motion tasks and suggests that normals can "ignore" or segregate noise that is more than two *SD* from the mean orientation/direction, whilst amblyopes integrate across all orientations/directions reducing their thresholds predictably.

The present results suggest that when broadband dots are used to measure global motion processing in amblyopia, that the different spatial frequency components of the stimulus are equally affected. Having controlled for the influence of the spatial frequency deficit in lower visual areas, we found a uniform impairment across spatial scale. Such a result would be expected if the information from different sized receptive fields in V1 were summed at, or prior to, global motion analysis. There is some psychophysical evidence that global motion is processed by a mechanism that is spatially broadband (Bex & Dakin, 2002; Yang & Blake, 1994). Impairment of a broadband global motion mechanism in amblyopia may explain our data. Another possibility is that there is some degree of spatial scale preservation within the extra-striate region where global motion is processed and that the amblyopic deficit affects this processing, independent of its spatial scale, however, the former explanation seems the more parsimonious.

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