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SCIENCE DIRECT

Vision Research 46 (2006) 2571–2580

Vision Research

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The extent of the dorsal extra-striate deficit in amblyopia

A.J. Simmers [b], T. Ledgeway [c], B. Mansouri [a], C.V. Hutchinson [c], R.F. Hess [a,*]

[a] McGill Vision Research, McGill University, Montreal, Que., Canada
[b] Department of Visual Science, City University, London, UK
[c] School of Psychology, University of Nottingham, Nottingham, UK

Received 7 October 2005; received in revised form 21 December 2005

Abstract

Previously, we have shown that humans with amblyopia exhibit deficits for global motion discrimination that cannot be simply ascribed to a reduction in visibility or contrast sensitivity. Deficits exist in the processing of global motion in the fronto-parallel plane that suggest reduced extra-striate function (i.e., MT) in amblyopia. Here, we ask whether such a deficit also exists for rotation and radial components of optic flow that are first processed at higher sites along the dorsal pathway (i.e., MSTd). We show that similar motion processing deficits occur in our amblyopic group as a whole for translation, rotation, and radial components of optic flow and that none of these can be solely accounted for by the reduced visibility of the stimuli. Furthermore, on a subject-by-subject basis there is no significant correlation between the motion deficits for radial and rotational motion and those for translation, consistent with independent deficits in dorsal pathway function up to and including MSTd.

Keywords: Amblyopia; Optic flow; Contrast; MSTd; MT; Global motion

1. Introduction

Our understanding of the nature and site of the visual deficit in amblyopia has changed considerably over the last 3 decades. Initially it was thought that the contrast sensitivity loss provided an adequate explanation in humans (Hess & Howell, 1977; Levi & Harwerth, 1977) and that the loss of spatial resolution and contrast sensitivity of individual V1 cells provided an adequate neural model in animals (Kiorpes et al., 1987; Movshon et al., 1987). We now know that not only is there a range of suprathreshold deficits in amblyopia (Bradley & Skottun, 1984; Caelli, Brettel, Rentschler, & Hilz, 1983; Demanins, Hess, Williams, & Keeble, 1999; Hess, Burr, & Campbell, 1980; Hess & Holliday, 1992; Lawden, Hess, & Campbell, 1982; Pass & Levi, 1982; Treutwein, Rentschler, Zetzsche, Scheidler, & Boergen, 1996; Vandenbussche, Vogels, & Orban, 1986) but also there are processing deficits in amblyopia that do not involve contrast sensitivity and whose cortical locus is likely to be beyond the striate cortex (Simmers, 2003; Simmers, 2005; Sharma, 2000; Lerner, 2003).

Of particular relevance to the present study is the recent evidence that global motion processing is abnormal in amblyopia, be it strabismic, anisometropic or deprivation in origin (Constantinescu, Schmidt, Watson, & Hess, 2005; Ellemberg, Lewis, Maurer, Brar, & Brent, 2002; Simmers, Ledgeway, Hess, & McGraw, 2003). Using a novel contrast manipulation, Simmers et al. (2003) assessed the relative contrast vs. motion integration components of the deficit for global translational motion in a group of strabismic and anisometropic amblyopes. They showed that although there can be performance losses that have a purely contrast basis, more profound losses occur because of anomalous motion integration. On the basis of this and the current two-stage model of global motion detection (Morrone, Burr, & Vaina, 1995), they argued that the site of the contrast loss is likely to be the striate cortex and the site of the

* Corresponding author.
E-mail address: robert.hess@mcgill.ca (R.F. Hess).

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doi:10.1016/j.visres.2006.01.009

more profound integration loss, the extra-striate cortex. They showed that this was the case for both first-order (luminance based) and second-order (texture based) motion stimuli, although greater losses were found for the latter.

Our knowledge of where global motion processing occurs in normal cortex is also in a state of flux though it appears to involve a number of regions including areas MT (V5) and MST (Mikami, Newsome, & Wurtz, 1986a; Mikami, Newsome, & Wurtz, 1986b). Neurons in MT have larger receptive fields than their V1 counterparts, possibly containing many small V1 subunits (Movshon, Adelson, Gizzi, & Newsome, 1985a, 1985b) with extensive centre-surround interactions (Allman, Miezin, & McGuinness, 1985). Lesion studies in monkeys (Huxlin & Pasternak, 2004; Newsome & Pare, 1988; Rudolph & Pasternak, 1999) and clinical studies in humans (Baker, Hess, & Zihl, 1991) have implicated this area (i.e., MT/MST in the dorsal pathway) in global motion processing. Lesions to area MT for example result in elevated coherence (signal-to-noise) thresholds for global translational motion similar to that reported in human amblyopes (Huxlin & Pasternak, 2004; Newsome & Pare, 1988; Rudolph & Pasternak, 1999). On the basis of this previous work it would seem likely that cortical areas extending from V1 and including V3a and MT are implicated in the reported translational global motion deficits in amblyopia. The question addressed here is whether extra-striate regions higher up along the dorsal stream are also affected. Area MST receives its input from area MT (Maunsell & Van Essen, 1983; Ungerleider & Desimone, 1986) and in the dorsal part of MST there are cells whose properties suggest that they encode, along with global translation, other forms of global motion, for example rotation and radial motion (Duffy & Wurtz, 1991a; Duffy & Wurtz, 1991b; Geesaman & Andersen, 1996; Tanaka, Fukada, & Saito, 1989a; Tanaka, Fukada, & Saito, 1989b). We were particularly interested to know whether the global motion deficit in amblyopia that has already been shown for translation extends to these other forms of optic flow whose processing site is known to be located beyond area MT in MSTd.

Our approach is similar to that of Simmers et al. (2003) in that we measure coherence thresholds for global motion at a number of different stimulus contrasts. We do this for global translation, rotation, and radial motion so that we can not only compare their relative sensitivities in normal and amblyopic eyes but also so that we can separate out the relative low-level (contrast) and high-level (motion integration) contributions to any performance decrement. Our results suggest that similar losses in global motion processing occur for all three components of optic flow, suggesting that the loss of function along the dorsal stream of the extra-striate pathway extends at least as far as MSTd where all three components of global motion are known to be first processed.

2. Methods

2.1. Observers

Four strabismic, 3 anisometropic, and 4 strabismic/anisometropic amblyopes (mean age 30.4 ± 6.2 years) were recruited for the study (see Table 1 for clinical details). For the purpose of this study amblyopia was defined as a visual acuity of 20/30 or worse in the amblyopic eye and anisometropia was defined as an interocular difference of greater than 1.00 dioptre sphere or 1.0 dioptres of cylinder. A control group of 10 observers (mean age 29.4 ± 5.8 years) were selected with normal visual acuity and normal binocular vision. Viewing was monocular in all cases with the appropriate refractive correction. All experimental procedures followed the institutional guidelines, and informed consent was obtained after the nature and possible consequences of the experiment had been explained. All subjects were experienced in psychophysical testing.

2.2. Apparatus and stimuli

Global motion stimuli (either translational, radial or rotational random-dot kinematograms—RDKs) were computer generated and displayed on an *SONY Multiscan G520* monitor (with a frame rate of 75 Hz), which was γ-corrected with the aid of internal look up tables. The RDK stimuli were presented within a circular window at the centre of the display, the diameter of which subtended an angle of 12° at the viewing distance of 0.92 m. The mean luminance of the remainder of the display (which was homogeneous) was approximately 50 cd/m^2.

Each RDK was generated anew immediately prior to its presentation (on any one trial) and was composed of a sequence of 8 images, which when presented consecutively produced continuous apparent motion. The duration of each image was 53.3 ms, giving a total stimulus duration of 426.7 ms, conditions that are directly comparable to those we have used previously to investigate the perception of global motion (Simmers et al., 2003). Each image contained 50 non-overlapping dots (dot density of 0.44 dots/°^{2}) and the diameter of each dot was 0.47° (composed of ~314 screen pixels). On the first frame of each RDK the dot positions were determined randomly and on subsequent frames were shifted by displacing each dot by 0.3°, resulting in a drift speed, if sustained, of 5.7°/s. When a dot exceeded the edge of the circular display window it was immediately re-plotted in a random spatial position within the confines of the window. This combination of dot density, dot diameter and displacement magnitude was chosen on the basis of pilot studies to ensure that (1) the individual dots were readily visible to the observers and (2) there was a low probability of "false-matches" occurring between different dots on successive displacements (Williams & Sekuler, 1984).

The global-motion coherence level of the stimulus was manipulated by constraining a fixed proportion of the dots ("signal" dots) on each image update to move coherently along either a translational, radial or rotational (circular) trajectory and the remainder ("noise" dots) to move in random directions. In the case of translational motion the direction of the signal dots was chosen to be either upwards or downwards on each trial with equal probability. For radial motion the signal dots were displaced along trajectories consistent with either expansion or contraction on each trial and for rotational motion the signal dots depicted either clockwise or counter-clockwise rotation. In line with previous studies that have used comparable radial and rotational RDK stimuli (e.g., Burr & Santoro, 2001), the magnitude of the dot displacement was always constant across space (i.e., did not vary with distance from the origin as it would for strictly rigid global radial or rotational motion) so that performance could be directly compared with the translational RDK stimuli. This ensured that regardless of the type of global motion depicted, all stimuli were identical in terms of the local dot speeds present.

Table 1
Clinical details of amblyopic subjects

	Subject	Spectacle prescription	Visual acuity	Ocular alignment
	ML	RE +1.0/−0.75 × 590° LE −3.25 DS	20/80 20/25	R SOT 6°
	PH	RE −2.00/+0.50 × 90° LE +0.50 90°	20/20 20/65	L SOT 5°
	ED	RE +0.75 LE +0.75	20/16 20/63	L SOT 5°
	RA	RE +3.25 DS LE +4.75/−0.75 × 45°	20/15 20/40	L XOT 5°
	XL	RE −2.50 LE −2.75/ +0.75 × 110°	20/20 20/400	L SOT 15°
	AJ	RE +0.25/−0.25 × 180° LE +8.5/−3.5 × 180°	20/16 20/400	Straight
	CB	Nil	20/20 20/80	L SOT 6°
	DAY	RE +0.75 DS LE +1.00 DS	20/20 20/80	L SOT 6°
	LM	RE +2.25/−2.25 × 180° LE +4.25/−2.00 × 110°	20/16 20/50	L XOT 8°
	SI	RE +2.50 DS LE plano	20/40 20/16	Straight
	SM	RE −2.75 +0.75 × 170° LE +0.50/ +1.75 × 110°	20/25 20/80	Straight

The dots were presented on a homogenous mid-gray background (mean luminance 50 cd/m^2) that filled the entire circular display window. The luminance modulation (Michelson contrast) and hence visibility of the dots could be varied by increasing the luminance of the dots, with respect to the background, according to the following equation:

$$\text{Dot luminance modulation} = (L_{\text{dots}} - L_{\text{background}})/(L_{\text{dots}} + L_{\text{background}}),$$

where L_{dots} and $L_{\text{background}}$ are the luminances of the dots and background, respectively. The luminance modulation of the dots could be varied in the range 0.004–0.33.

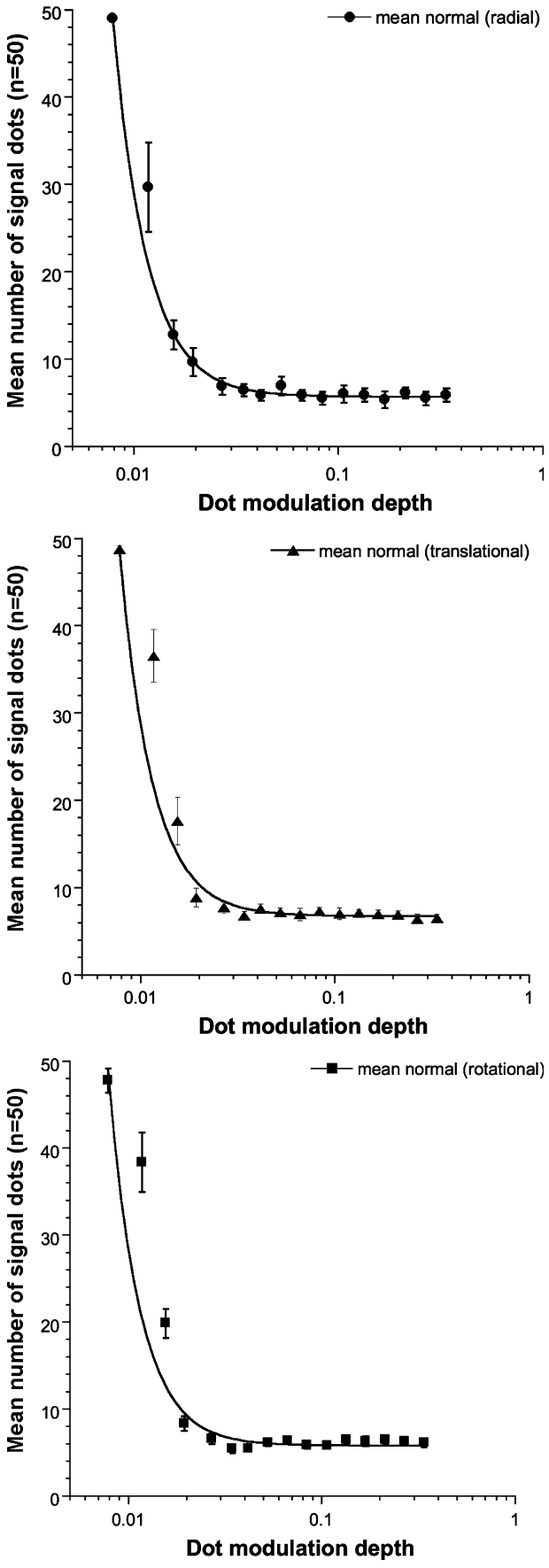
2.3. Procedure

Global motion thresholds were measured using a single interval direction-discrimination procedure. On each trial the observer was presented with a RDK stimulus in which the signal dots moved along either a translational, radial or rotational trajectory and the task was to identify its direction of motion from one of two known opposite motions: upwards vs. downwards, expansion vs. contraction or clockwise vs. counter-clockwise. Performance for each of the three types of motion (translational, radial, and rotational) was measured separately (the order of testing was pseudo-randomized for each observer) using an adaptive staircase procedure (Edwards & Badcock, 1995). The staircase varied the proportion of signal dots present on each trial, according to the observer's recent response history, to converge on (track) the 79% correct performance level. Eight reversals were collected before the staircase terminated and the threshold was taken as the mean of the final 6 reversal points. At the beginning of each run of trials the staircase began with the maximum number of signal dots possible (i.e., 50 dots). The initial step size in signal dot number was 8 dots and this was decreased after each of the first 3 reversals such that the step size for the last 6 reversals was only 1 dot. Each threshold reported is based on the mean of at least 5 such staircases. In those observers with amblyopia, measurements were repeated with both the amblyopic eye (AE) and non-amblyopic eye (fellow fixing eye) in random order. In normal observers the dominant eye was tested.

3. Results

Results for 10 normal observers for the three forms of global motion (i.e., translation, rotation, and radial motion) are displayed in Fig. 1. Here, we plot global motion thresholds, expressed in terms of the minimum number of signal dots (note that the total number of dots in the display was always 50) required to support reliable direction-discrimination performance, against the modulation depth (contrast) of the dots. It is clear that global motion thresholds are constant when the dot contrast exceeds a critical value but crucially depend upon dot contrast below this value (Simmers et al., 2003). The data have been fitted with a power function plus a constant (solid black line), $y = ax^b + c$, where a, b, and c are constants. This function was found to provide a good fit to previous global motion (Simmers et al., 2003) and global form data (Simmers, Ledgeway, & Hess, 2005). Here, the r^2 values of the fit were 0.89, 0.99, and 0.97 for radial, translation and rotation motion. The fit was poorer in the intermediate range where the variance was higher. Table 2 gives the mean corresponding values for these parameters for the normal subjects.

Similar motion/contrast dependencies are shown in Fig. 2 for the amblyopic eyes (AE) of each of our 11 ambly-



opic subjects (coloured curves). These amblyopic results are compared, for each of the three types of motion, to the averaged normal results (solid black curve re-plotted from Fig. 1).

Amblyopic subjects exhibit similar though not identical contrast/motion integration response functions and to a first approximation the departures from that of the normal curve do not appear to be different for the three different forms of global motion. We assume a two-stage model of global motion processing (Morrone et al., 1995) composed of an initial, local motion, contrast-sensitive stage that we associate with V1 and a second contrast-insensitive, motion integration stage that we associate with area or areas (e.g., MT, MSTd) in the dorsal stream of the extra-striate cortex. To separate out the relative striate/extrastriate components to the performance deficit we calculate (Simmers et al., 2005; Simmers et al., 2003) the relative horizontal and vertical translations needed to bring the results of each amblyopic subject into register with that of the average normal curve (solid black curve). The magnitudes of these derived shifts are shown in Fig. 3 and plotted against one another such that data falling on the vertical dotted line (scaling changes limited to the contrast axis in Fig. 2) would indicate a performance loss due purely to a visibility deficit, implicating the striate cortex. On the other hand, any data falling on the horizontal dotted line (scaling changes limited to the motion integration axis in Fig. 2) would be expected if the performance loss involves purely global motion integration processes and by implication, extra-striate cortex. Data falling on the diagonal would indicate combined deficits for visibility and global motion integration.

While in a few cases performance can be explained solely in terms of reduced visibility, in many cases both visibility and global motion integration are responsible for the performance deficit. In some cases, the deficit is purely due to an impairment of global motion integration.

In Fig. 4 group ratios are shown in the form of histograms separately for the global motion and visibility deficits for the three types of motion (i.e., radial, rotation, and translation). In the top histogram this comparison is between the results of amblyopic eyes and those of normal observers. In the bottom histogram, the fellow eyes of amblyopes are compared with those of normal observers. In terms of the amblyopic results, due to the limited sample size per amblyopic sub-group (strabismic, anisometropic and anisometropic strabismics), formal statistical analyses were carried out for a single generic subject group. Therefore, an analysis of variance (ANOVA) was carried out for *amblyopic subject group*

Fig. 1. The relationship between the modulation depth (contrast) of the individual dots and the global motion threshold (expressed as the mean number of signal dots required to support 79% correct responding) for radial, translation and rotation components of optic flow for 10 normal subjects. The resulting data are well-described by a power function plus a constant (solid black curves). The error bars are ±1 SD. See text for further details.

Table 2
Model parameters for the three types of motion stimuli

Complex motion	Constants		
	a	*b*	*c*
Radial	1.21e−04 (±1.35e−04 SEM)	−2.64 (±0.23 SEM)	5.76 (±0.25 SEM)
Rotation	8.9e−05 (±7.31e−05 SEM)	−2.7 (±0.17 SEM)	5.87 (±0.15 SEM)
Translation	1.36e−04 (±1.19e−04 SEM)	−2.61 (±0.18 SEM)	6.72 (±0.17 SEM)

In normal observers the relationship between the global motion threshold and the magnitude of the dot modulation is well described by a power function and a constant $y = ax^b + c$, where a, b, and c are constants.

(all amblyopes) and the 2 factors of *stimulus type* (radial vs. rotation vs. translation) and *component anomaly* (contrast vs. motion integration). ANOVA revealed no significant main effect of stimulus type [$F_{(2,20)} = 3.021$; ns] but a significant main effect of component anomaly [$F_{(1,10)} = 36.863$; $p < 0.0001$] with no significant interaction [$F_{(2,20)} = 1.194$; ns].

Amblyopes, as a group, do not appear to show a greater deficit or indeed a selective loss for any one type of complex motion. There was however a significant main effect of component anomaly, demonstrating that motion integration deficits, when collapsed across subject group and stimulus type, were significantly greater than contrast (visibility) deficits.

A similar analysis of variance for the *fellow eye* of the *amblyopic subject group* (all amblyopes) for the 2 factors of *stimulus type* (radial vs. rotation vs. translation) and *component anomaly* (contrast vs. motion integration) revealed a comparable pattern of findings in that there no significant main effect of stimulus type [$F_{(2,20)} = 2.506$; ns] but a significant main effect of component anomaly [$F_{(1,10)} = 35.863$; $p < 0.0001$]. Thus the fellow eyes of our amblyopes do not exhibit a selective loss for any one type of complex motion, but still demonstrate motion integration deficits that are significantly greater than contrast (visibility) deficits.

4. Discussion

The human visual system comprises both parallel and serial processing stages. At the level of the extra-striate cortex, two main parallel processing streams are evident, the ventral stream leading to the inferior temporal cortex and the dorsal stream leading to the parietal cortex (Ungerleider & Mishkin, 1982). It has been suggested that each of these streams is concerned with a fundamentally different type of visual analysis—the ventral stream with spatial analysis and the dorsal stream with temporal/motion analysis. Each cortical stream comprises a cascade of different hierarchical processing areas (Van Essen, Anderson, & Felleman, 1992) with higher levels processing more complex stimulus characteristics. The dorsal stream is concerned with the extraction of image motion. In V1, neurons exhibit directional sensitivity within highly localized regions of the field (Movshon et al., 1985a; Movshon & Newsome, 1996). At this stage the analysis is inherently local but with a strong dependence on stimulus contrast. The first cortical area in which these local estimates are combined is MT. Neurons in MT receive their input from several areas including V1, V2, V3a and possibly directly from the Lateral Geniculate Nucleus (Sincich, Park, Wohlgemuth, & Horton, 2004) and their responses saturate at relatively low contrasts. Their receptive fields are much larger than those in V1 being thought to comprise many smaller V1 subunits (Movshon et al., 1985a). This area combines many local motion estimates to derive a more global estimate of motion in the fronto-parallel plane (Baker et al., 1991; Newsome & Pare, 1988). In yet higher processing areas along the dorsal pathway, specifically in the dorsal part of the Medial Superior Temporal Area (MSTd), neurons with still larger receptive fields combine regional motion signals from MT to derive specific types of global motion or optic flow that relate to locomotion. Neurons in MSTd respond to translation, rotation and radial motion (Duffy & Wurtz, 1991a, 1991b; Geesaman & Andersen, 1996; Tanaka et al., 1989a, 1989b). Some cells respond preferentially to just one of these optic flow components, but others respond to combinations of components (Duffy & Wurtz, 1991a).

The purpose of the present investigation was to better define the extent of the deficit to the dorsal extra-striate pathway in human amblyopia. Previously, we have shown that amblyopes exhibit global processing deficits for the fronto-parallel motion of luminance defined stimuli and that this has both visibility and motion integration components (Simmers et al., 2003). This suggests that while there are deficits to neurons in area V1, there are also independent deficits to neurons in extra-striate motion processing areas such as MT. Here, we sought to establish if this deficit extends to the next processing stage along the dorsal pathway, namely MST where cells in the dorsal aspect are known to represent the first analysis site for rotation and radial components of optic flow (Duffy & Wurtz, 1991a, 1991b; Geesaman & Andersen, 1996; Tanaka et al., 1989a; Tanaka et al., 1989b). Our results show that there are comparable deficits in all three components of optic flow (i.e., translation, rotation and radial) and that this is not entirely due to a lower level visibility deficit. This suggests that, in amblyopia, the processing carried out by neurons in MSTd in the dorsal pathway is compromised. One cannot conclude however that there is an independent deficit at the level of MSTd

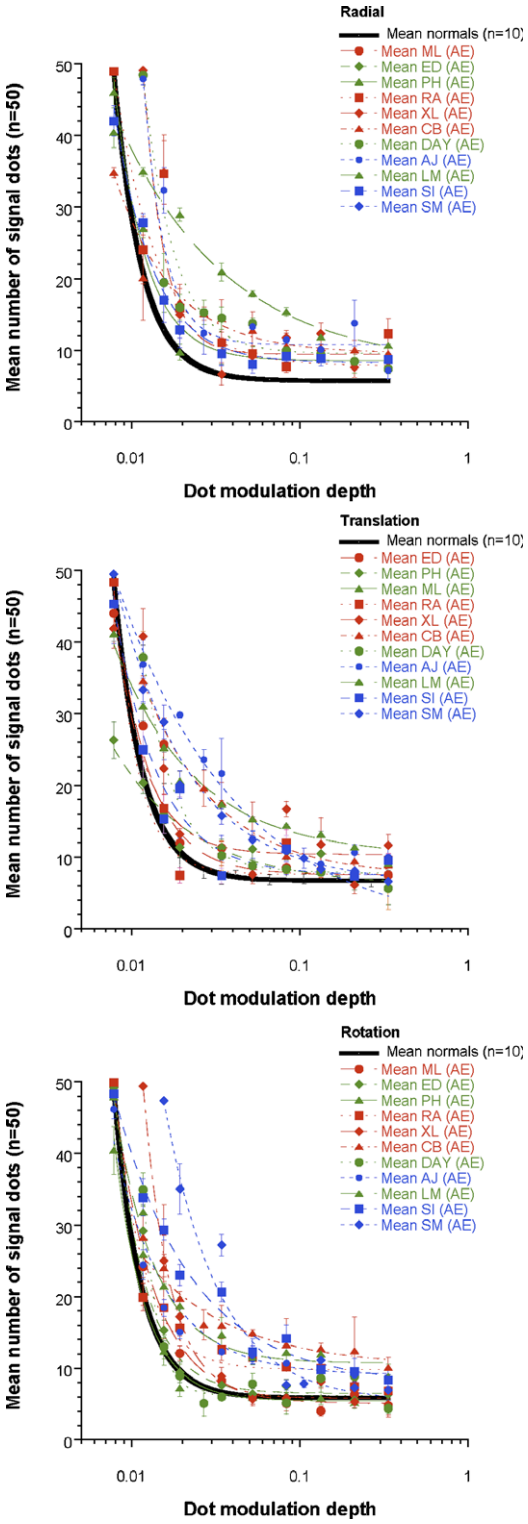


Fig. 2. The relationship between the modulation depth (contrast) of the individual dots and the global motion threshold (expressed as the mean number of signal dots required to support 79% correct responding) for radial, translation and rotation components of optic flow for 11 amblyopic subjects (observing was carried out using the amblyopic eye—AE). The results for each individual amblyope (coloured symbols) have been fitted by a power function plus a constant and are compared to the average performance of the normal observers (black solid curve re-plotted from Fig. 1). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this paper.)

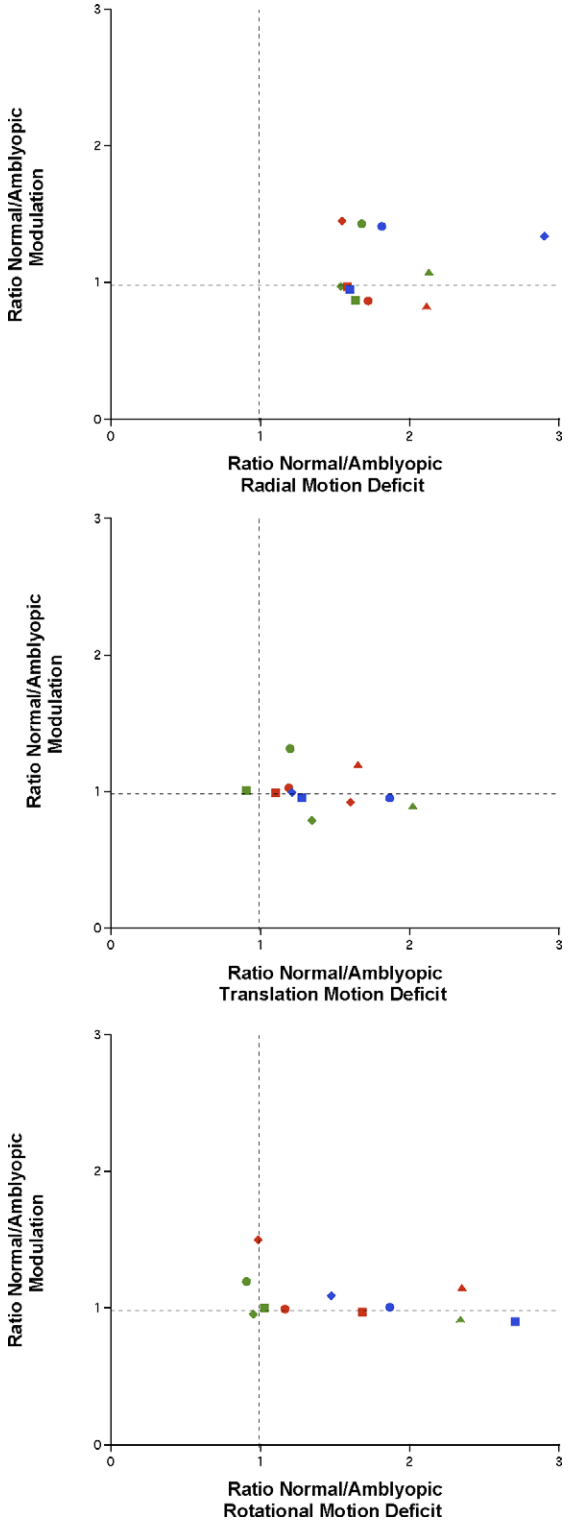


Fig. 3. Derived contrast/global motion components to the amblyopic performance losses shown in Fig. 2. The lateral and vertical shifts required to bring each amblyopic curve, shown in Fig. 2, into correspondence with that of the normal observers is plotted here. Data falling on the vertical dashed line represent reduced performance on the global motion task explicable in terms of reduced visibility alone (i.e., a contrast encoding deficit). Data falling in the horizontal dashed line represent reduced performance on the global motion task explicable in terms of global motion processing alone. Data falling on the diagonal line represent reduced performance as the result of combined visibility and global motion processing deficits.

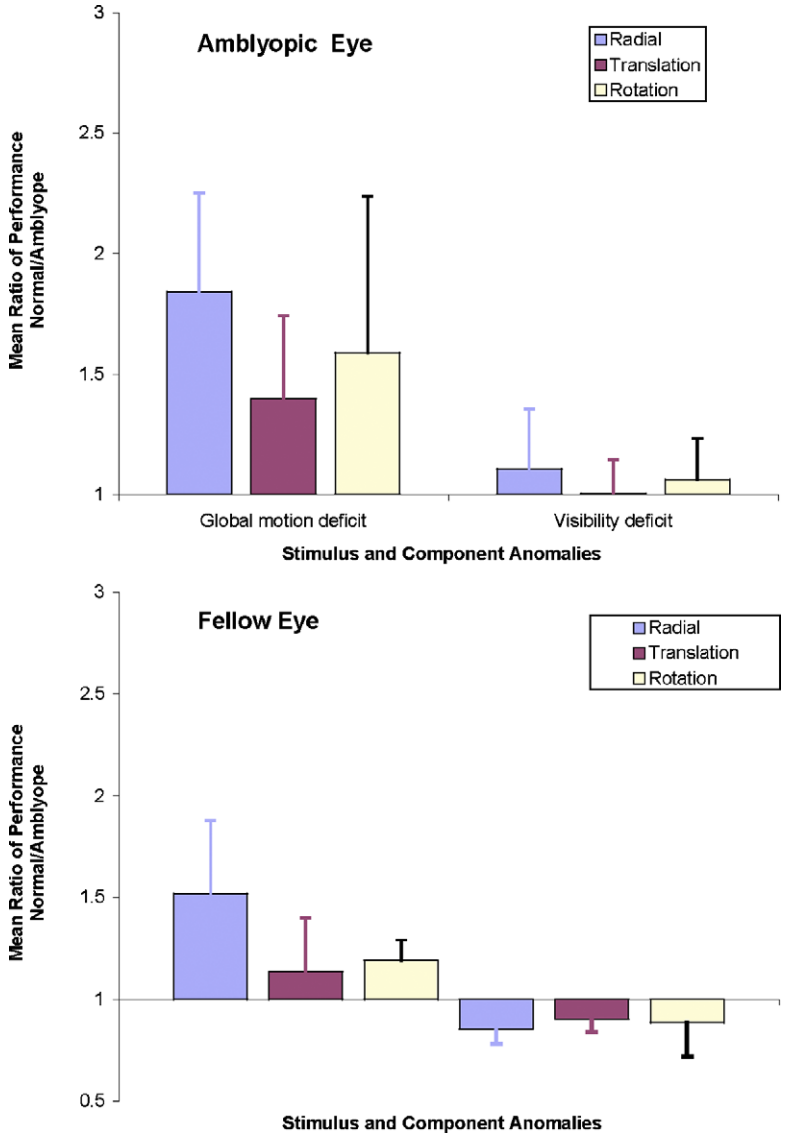


Fig. 4. Top, the mean ratio of normal/amblyopic eye for each type of motion (i.e., translation, rotation, and radial) and for the two derived component anomalies (i.e., visibility and global motion processing). The error bars represent 1 SD. Bottom, the mean ratio of normal/fellow fixing eye for each type of motion (i.e., translation, rotation, and radial) and for the two derived component anomalies.

in amblyopic humans. Since area MSTd receives input from area MT, any anomaly in MSTd function could simply be a consequence of the "upstream" deficit previously reported (Simmers et al., 2003) in area MT. Put another way, if the larger radial and rotational flow fields of MSTd neurons are constructed from the smaller fronto-parallel-responsive neurons in MT (Tanaka et al., 1989b) then the radial and rotational motion deficits reported here could originate in area MT. If this were the case, one would expect not only a strong correlation between the deficits for optic flow (MSTd) and fronto-parallel (MT) global motion but also the motion deficits for radial and rotational motion should be correlated if they have a common underlying cause (i.e., compromised global motion extraction in MT). In Fig. 5 we plot, on a subject-by-subject basis, the global motion deficit for translational vs. radial stimuli (Fig. 5A), translational vs. rotational stimuli (Fig. 5B) and radial vs. rotational motion (Fig. 5C). The finding that none of these comparisons exhibits a statistically significant ($p > 0.05$ in all cases) correlation [$r_{\text{translation/radial}} = 0.16$; $r_{\text{translation/rotation}} = 0.5$; $r_{\text{radial/rotation}} = 0.25$, respectively] is consistent with the idea that the optic flow deficits are due to independent processing anomalies further along the dorsal pathway (i.e., beyond MT) than those responsible for the previously reported translational deficit for global motion (Simmers et al., 2003). However, it is not conclusive evidence for independent deficits at the level of MSTd because the low correlations may be due to factors other than a complete independence. Specific tests of independence would need to be carried out before we can definitively conclude this.

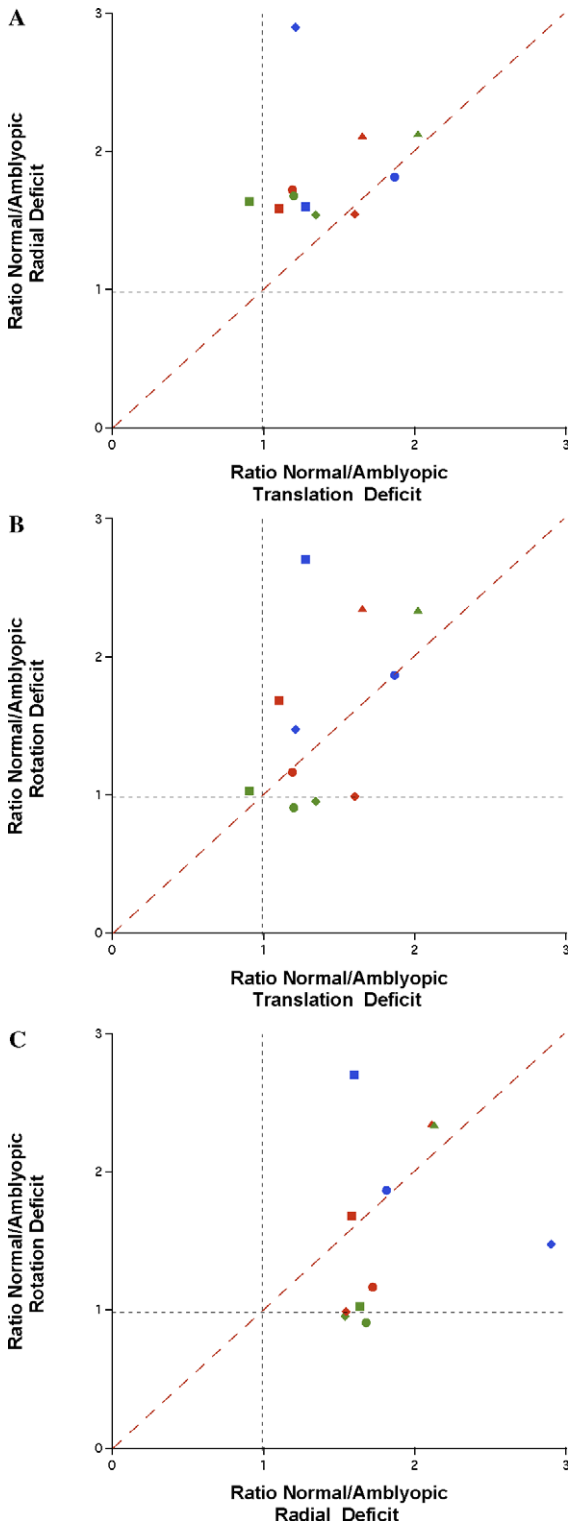


Fig. 5. (A–C) Global motion deficits for translation vs. radial motion [$r = 0.16$; ns], translation vs. rotation [$r = 0.5$; ns] and radial vs. rotational motion [$r = 0.25$; ns] respectively are compared for the amblyopic eye of all subjects (symbols refer to Table 1).

Two interesting differences were found between the performance of fellow fixing eyes of amblyopes and the dominant eyes of our normal population, one concerned the contrast component of the global motion deficit, the other the motion integration component. In terms of the contrast component of the deficit, the ratio between performance of the normal and fellow eye for contrast/visibility was found to be 1:0.88 ± 0.11, suggesting that the fellow eye could tolerate greater reductions in contrast compared to a normal eye and still reliably perceive global motion. In terms of the motion integration component of the performance deficit, similar though less severe motion deficits were seen in the fellow fixing eye of amblyopes.

Although present animal models of amblyopia have been preoccupied with the contrast sensitivity deficit and area V1, it is widely acknowledged that to explain other aspects of amblyopia would require deficits beyond V1 (Kiorpes & McKee, 1999). Two studies that have investigated cortical function beyond V1 in amblyopic animals have highlighted anomalies in extra-striate areas. While it is generally agreed that about the same number of cells in area 17 and 18 (Hubel & Wiesel, 1965; Kalil, Spear, & Langsetmo, 1984; Schroder, Fries, Roelfsema, Singer, & Engel, 2002) can be driven through the strabismic and non-strabismic eyes (but in the case of esotropia and severe amblyopia see Baker, Grigg, & von Noorden, 1974; Crawford & von Noorden, 1979; Kiorpes, Kiper, O'Keefe, Cavanaugh, & Movshon, 1998), this is not the case in extra-striate areas. There are not only many fewer binocular cells (as is also the case in area 17 and 18) but also many more cells are driven by the non-strabismic eye in both the dorsal (Schroder et al., 2002; Sireteanu & Best, 1992) and ventral (Schroder et al., 2002) pathways. It is possible that the loss of binocularity may account for the small deficit found in the fellow eyes in the present study and the reduced number of extra-striate cells driven by the strabismic eye may correlate with the integrative component of the deficit.

Acknowledgments

This work was supported by a CIHR grants to R.F.H. (MT 108-18 & MOP 53346). C.V.H. was supported by a Universitas 21 Prize Scholarship from the University of Nottingham.

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