



Deficits to global motion processing in human amblyopia

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Abstract

We investigated global motion processing in a group of adult amblyopes using a method that allows us to factor out any influence of the known contrast sensitivity deficit. We show that there are independent global motion processing deficits in human amblyopia that are unrelated to the contrast sensitivity deficit, and that are more extensive for contrast-defined than for luminance-defined stimuli. We speculate that the site of these deficits must include the extra-striate cortex and in particular the dorsal pathway.

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1. Introduction

Amblyopia is a developmental condition that is characterized by reduced vision in one eye due to the presence of a sensory impediment to visual development, such as strabismus (ocular misalignment) or anisometropia (unequal refractive error), occurring early in life. While there is some evidence to suggest that certain subcortical structures can be affected in amblyopia (Chino, Cheng, Smith, Garraghty, Roe, & Sur, 1994) the vast majority of studies in both humans (Hess & Baker, 1984; Hess, Baker, Verhoeve, Tulunay Keeseey, & France, 1985) and animals (Cleland, Crewther, Crewther, & Mitchell, 1982; Crewther, Crewther, & Cleland, 1985) point towards a cortical locus for the processing deficit. Additionally, recent imaging studies in humans show that the deficit involves both striate and extra-striate cortical function (Sireteanu, Tonhausen, Muckli, Zanella, & Singer, 1998; Barnes, Hess, Dumoulin, Achtman, & Pike, 2001). It is yet to be established if this is the result of independent striate and extra-striate deficits or whether it is simply the consequence of the 'down stream' striate deficit.

Previous animal studies have concentrated on striate function and in particular the contrast sensitivity deficit. Such studies have revealed deficits at the single cell level (Chino, Shansky, Jankowski, & Banser, 1983; Crewther & Crewther, 1990; Eggers & Blakemore, 1978; Kiorpes, Kiper, O'Keefe, Cavanaugh, & Movshon, 1998; Movshon et al., 1987) that include reduced spatial resolution, reduced contrast sensitivity and a reduced numbers of binocular cells. However, these deficits in the case of strabismic and anisometric amblyopia, as opposed to the more severe, deprivation amblyopia, have not been sufficient to account for the behavioural deficit measured with the same stimuli (Kiorpes et al., 1998). This led to the suggestion that additional deficits may exist beyond the striate cortex. Indeed a selectively reduced proportion of cells driven by the amblyopic eye in the extra-striate cortex of cats has been previously reported (Schroder, Fries, Roelfsema, Singer, & Engel, 2001; Sireteanu, 1991; Sireteanu & Best, 1992; Sireteanu et al., 1998).

The extra-striate cortex consists of a large number of identifiable areas that receive separate topographically organized inputs (Zeki, 1978). Two different extra-striate processing streams have been identified in primate, the dorsal stream leading to the parietal cortex and the ventral stream leading to the temporal cortex (DeYoe & Van Essen, 1988; Ungerleider & Mishkin, 1982; Van Essen, Anderson, & Felleman, 1992). Areas within these processing streams are organized in a roughly hierarchical

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way, with more complex processing occurring at sites further along the pathway. The dorsal stream involves areas MT and MST and is thought to mediate motion processing and the localization of objects in space. The ventral pathway, which includes areas V4 and IT is thought to mediate the processing of spatial form and may be associated with perceptual discrimination of shape and contour. Interestingly, evidence is emerging that areas MT/MST can also analyse object shape therefore this dichotomy may not be as marked as originally thought (Kourtzi, Bulthoff, Erb, & Grodd, 2002).

If the extra-striate cortex is selectively affected in humans with amblyopia one would expect to find a disruption to processing mechanisms unique to extra-striate cortex. In the present investigation we concentrated on global motion processing as a way of assessing dorsal stream function within the extra-striate cortex. We know from current neurophysiology that such a task could be adequately solved in primate areas MT/MST where individual receptive fields are sufficiently large to be able to integrate different local motions in adjacent regions of the field (Movshon, Adelson, Gizzi, & Newsome, 1985). Furthermore, not only do lesions to this cortical region in monkey (Newsome & Pare, 1988) and man (Baker, Hess, & Kihl, 1991) produce selective deficits for global motion but also micro-stimulation within this region can bias direction discrimination (Salzman, Murasugi, Britten, & Newsome, 1992) for this particular task.

There is support for the idea (Morrone, Burr, & Vaina, 1995) that global motion detection involves a two stage process: a contrast-dependent local motion signal extraction in V1 followed by a second stage, possibly in MT, where a global mechanism is limited primarily not by contrast but by the signal:noise ratio of moving dots.

No previous study has assessed global motion processing in humans with amblyopia let alone one where the known contrast sensitivity deficit has been taken into account. Since there is neurophysiological support (Chino et al., 1983; Crewther & Crewther, 1990; Eggers & Blakemore, 1978; Movshon et al., 1987) for the contrast sensitivity deficit being located in early visual areas, such as V1 (but also see Kiorpes et al., 1998), this leaves open the possibility that any observed reduction in global motion sensitivity might be the consequence of the well-documented striate deficit in area V1 at the *first* stage of the global motion process rather than a deficit at the level of the *second* stage located in extra-striate cortex. Indeed, Tang, Kiorpes, and Movshon (1998) have previously reported a global motion deficit in amblyopic monkeys, the magnitude of which could be directly related to the contrast sensitivity deficits present.

In the present investigation we have undertaken an analysis that distinguishes between first and second stage global motion deficits, in other words contrast-dependent (i.e. visibility-based) as opposed to sig-

nal:noise-dependent deficits in an attempt to ascertain whether any processing deficits observed are striate or extra-striate in origin.

The global motion task developed by Newsome and Pare (1988) was employed in which only a small percentage of the dots (“signal” dots) within a random-dot-kinematogram (RDK) move in the same direction (up or down), the remaining “noise” dots have random directions. For a given presentation, subjects have to discriminate the direction of the coherent global motion of the “signal” dots. Performance, which is quantified in terms of the minimum number of “signal” dots (coherence) required to support direction discrimination, is measured as a function of the dot modulation or contrast (visibility) of the dots. Since there is emerging evidence (Baker & Hess, 1998; Ledgeway & Hess, 2000), that global motion processing can utilize not only luminance-defined (so-called first-order) information but also contrast-defined (so-called second-order) information in RDKs, we assessed performance for both of these stimulus types (Fig. 1). This is particularly relevant to amblyopia because there is emerging evidence (Wong, Levi, & McGraw, 2001) that contrast-defined (i.e. second-order) form-processing might be affected to a greater degree.

2. Methods

2.1. Observers

Five strabismic, one anisometropic and five strabismic/anisometropic amblyopes (mean age 30.7 ± 13.4 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 eight 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 (RDKs) were computer generated and displayed on an SONY Multiscan 520 GS monitor (with a frame rate of 75 Hz), which was gamma-corrected with the aid of internal look up tables. As an additional precaution psychophysical procedures were used to confirm that any residual luminance non-

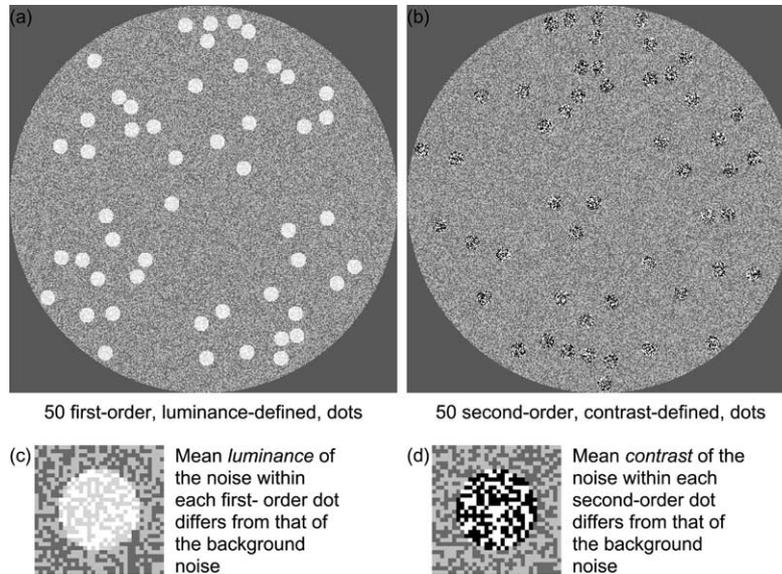


Fig. 1. Depiction of a single frame of the first-order (luminance defined) and second-order (contrast defined) global motion stimuli (a and b respectively). A magnified view of a single first-order dot (c) and a single second-order dot (d) are shown for clarity.

linearities were minimized (Ledgeway & Smith, 1994; Nishida, Ledgeway, & Edwards, 1997). In brief, motion

Table 1
Clinical characteristics of the amblyopic subjects

| Subject | Visual acuity | Spectacle prescription | Ocular alignment |
|---------|-----------------------|--|------------------|
| ● | RE 20/20 LE 20/50 | RE +0.75DS LE +1.00DS | R SOT 10Δ |
| ▼ | RE 20/15 LE 20/50 | Nil | L SOT 25Δ |
| ▲ | RE 1/200 LE 20/20 | Nil | R SOT 20Δ |
| ■ | RE 20/20 LE 20/200 | Nil | L SOT 14Δ |
| ◆ | RE 20/50 LE 20/20 | RE -1.00DS LE plano | R SOT 10Δ |
| ■ | RE 20/15 LE 20/200 | RE plano LE +3.25 × 90 | L SOT 10Δ |
| ◆ | RE 20/60 LE 20/20 | RE -5.25/ -2.25 × 180 LE -3.00/ -1.75 × 170 | L XOT 20Δ |
| ▼ | RE 20/30 LE 20/20 | RE +2.75/ -1.50 × 105 LE plano | L SOT 25Δ |
| ● | RE 20/15 LE 20/40 | RE +5.75/ -3.00 × 165 LE +6.75/ -2.00 × 10 | L XOT 8Δ |
| ▲ | RE 20/45 LE 20/20 | RE +5.50/ -1.00 × 180 LE +1.50DS | R SOT 4Δ |
| ● | RE 20/50 LE 20/20 | RE +3.25/ +1.00 × 90 LE +2.00DS | Straight |

Red symbols correspond to individual strabismic, green symbols to strabismic anisometropes and blue symbols to anisotropic amblyopes.

sequences were constructed in which consecutive images alternated between a sinusoidal variation in the luminance (first-order) of a two-dimensional (2-d), static noise field and an otherwise identical sinusoidal modulation in the contrast (second-order) of the noise field. When the spatial phases of the sinusoids in consecutive images differ by 0.25 spatial periods, observers cannot determine the direction of motion unless there is a significant luminance non-linearity contaminating the second-order images. We confirmed that the observers in our study were unable to identify motion direction under these conditions. Variants of this technique have been widely used by other researchers (e.g. Gurnsey, Fleet, & Potechin, 1998; Lu & Sperling, 2001; Papatomas, Gorea, & Chubb, 1996) to measure and/or check the efficacy of the gamma-correction applied to displays.

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 0.84 m. The mean luminance of the remainder of the display (which was homogeneous) was approximately 50 cd/m².

Each RDK was generated a new immediately prior to its presentation (on any one trial) and was composed of a sequence of eight 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 used previously to investigate the perception of first-order and second-order global motion (Edwards, Badcock, & Nishida, 1996). 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 $5.7^\circ/\text{s}$. When a dot reached the edge of the circular display window it was “wrapped-around” so that it immediately reappeared on the opposite edge 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).

Each dot was composed of 2-d, static¹ noise produced by assigning individual screen pixels ($1.41 \times 1.41'$) within the area of each dot to be “black” or “white” with equal probability. The dots were presented on a 2-d, static noise background which filled the entire circular display window (mean luminance of 50 cd/m^2 and Michelson contrast of 0.1), either the mean luminance (in the case of first-order dots as shown in Fig. 1a and c) or the mean contrast (in the case of second-order dots as shown in Fig. 1b and d) of which could be less than that of the noise within the dots.

The luminance modulation (visibility) of the first-order dots was defined as:

Dot luminance modulation

$$= (\text{DL}_{\text{mean}} - \text{BL}_{\text{mean}}) / (\text{DL}_{\text{mean}} + \text{BL}_{\text{mean}})$$

where DL_{mean} and BL_{mean} are the mean luminances of the noise within the dots and background respectively, averaged over pairs of noise elements with opposite luminance polarity. The luminance modulation of the first-order dots could be varied in the range 0–0.3.

The contrast modulation (visibility) of the second-order dots could be varied in an analogous manner according to the equation:

Dot contrast modulation

$$= (\text{DC}_{\text{mean}} - \text{BC}_{\text{mean}}) / (\text{DC}_{\text{mean}} + \text{BC}_{\text{mean}})$$

where DC_{mean} and BC_{mean} are the mean contrasts of the noise within the dots and background respectively, computed over pairs of noise elements with opposite luminance polarity. The contrast modulation of the second-order dots could be varied in the range 0–0.8.

¹ Static noise can sometimes give rise to local first-order luminance artifacts in contrast-defined patterns at threshold, as a consequence of local clustering of noise pixels with the same polarity (Smith & Ledgeway, 1997). However, such artifacts are minimal, or absent, when there is no spatial variation in luminance within each noise pixel and the contrast profile of the image is displaced by integer numbers of pixels (Nishida et al., 1997) as in this study.

2.3. Procedure

In the present task, elements in a field of random dots either moved coherently (signal dots) or in random directions (noise dots). The observer’s task was to indicate whether the signal dots had moved up or down. Global motion thresholds were measured 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 last six reversal points. At the beginning of each run of trials the staircase began with the maximum number of signal dots possible (i.e. all 50 dots moved in the same direction). The initial step size in signal dot number was eight dots and this was decreased after each of the first three reversals such that the step size for the last six reversals was only one dot. Each threshold reported is based on the mean of at least five such staircases. In those observers with amblyopia, measurements were repeated with both the amblyopic eye and non-amblyopic eye in random order. In normal observers the right or left eye was randomly assigned.

3. Results

Fig. 2a shows the mean normal result (black symbols) in which global motion thresholds for first-order dots are plotted against the dot modulation (contrast) of the dots. Global motion direction thresholds exhibit asymptotic behaviour at high levels of dot modulation but increase markedly as the magnitude of the dot modulation decreases (Edwards & Badcock, 1995; Edwards et al., 1996). In the case of normal observers, the relationship between the global motion threshold and the magnitude of the dot modulation is well described by a power function plus a constant (solid black line). $y = ax^b + c$, where a , b and c are constants. For the normal population the mean corresponding values for the first-order stimuli are $a = 7.11^{-4}$ ($\pm 5.88^{-3}$ s.e.m); $b = -2.26$ (± 0.17 s.e.m); $c = 7.34$ (± 0.24 s.e.m) and for the second-order stimuli are $a = 2.57^{-4}$ (± 1.45 s.e.m); $b = -2.78$ (± 0.56 s.e.m); $c = 5.06$ (± 2.55 s.e.m).

If performance on this task in amblyopia is limited by the contrast sensitivity deficit (i.e. due to reduced visibility), thought to reside in V1, then we would expect the response function (mean number of signal dots versus dot modulation (contrast)) for the amblyopic visual system to be well described by a laterally translated (to the right on this co-ordinate system) version of the normal response curve, as modeled by the dashed curves also depicted in Fig. 2a. Here we demonstrate hypothetically how a systematic difference in absolute sensi-

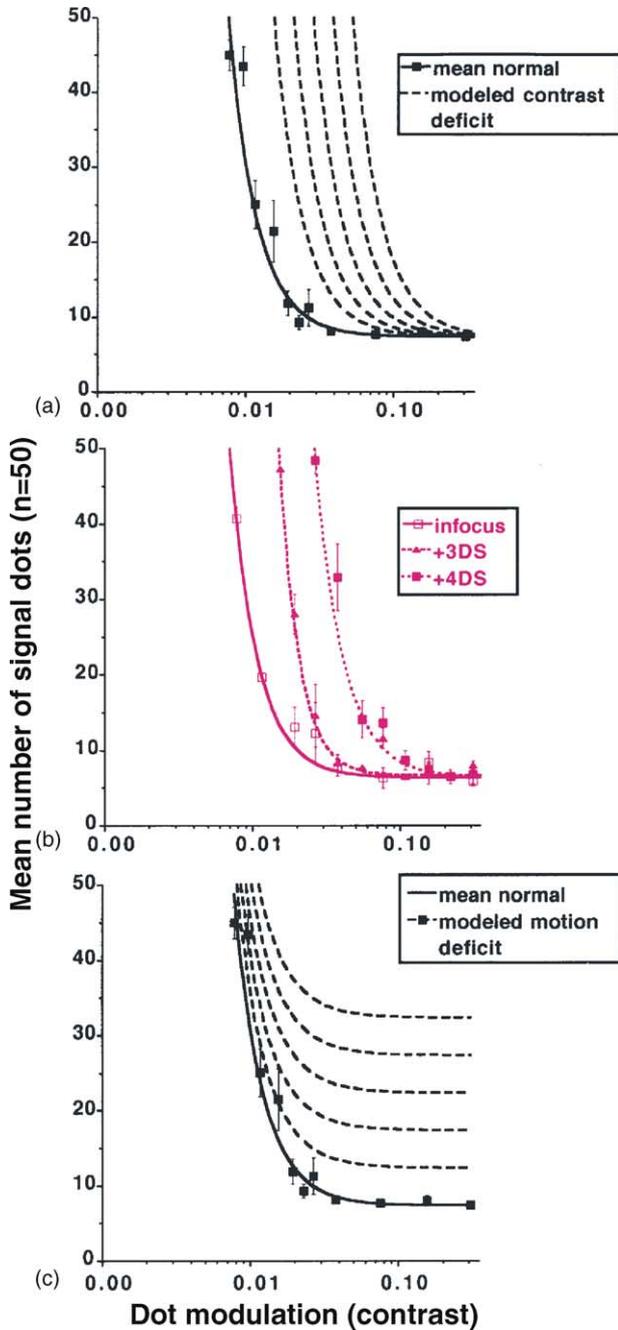


Fig. 2. The mean first-order (luminance-defined) global motion thresholds for the eight normal observers are plotted as a function of the dot modulation of the signal dots. The relationship between the global motion threshold and the magnitude of the dot modulation is fitted by a power function plus a constant. (a) The dashed curves demonstrate hypothetically how a systematic difference in absolute sensitivity can be predicted by a simple translation of the thresholds versus dot modulation function along the dot contrast axis (a contrast-specific deficit). (b) \square shows the mean response function (± 1 s.e.m) of a single normal observer from the normal observer group (infocus) and that same observer with the addition of dioptric blur of +3DS \blacktriangle and +4DS \blacksquare respectively, illustrating empirically the results of a laterally translated contrast response function. (c) The dashed curves again demonstrate in theory this time how a systematic difference in the ability to extract global motion direction, will manifest as a simple translation of the thresholds versus dot modulation function along the threshold axis (a global motion-specific deficit).

tivity can produce a simple translation of the response function along the dot modulation (contrast) axis (i.e. a contrast-specific deficit). A number of strictly local models of motion detection also exhibit a strong dependence on stimulus contrast. For example the output of a motion-energy detector (Adelson & Bergen, 1985) is proportional to the square of the effective contrast of the stimulus falling within its receptive field. Thus an early contrast-encoding deficit might be expected to produce a relatively impoverished output from these contrast-dependent models, leading to a concomitant lateral shift of the normal response curve like that shown in Fig. 2a.

A systematic difference in absolute sensitivity can also be mimicked empirically by means of optical defocus, which spatially filters and reduces the visible dot modulation (contrast). Fig. 2b shows the mean threshold of a normal observer (in-focus data) and that same observer with a dioptric blur of +3DS and +4DS respectively. The results of artificially blurring a normal observer can be seen to shift the whole curve laterally to the right, as was predicted above, amounting to a modulation depth re-scaling.

However, if on the other hand, performance is solely limited by deficient global motion processing, the response function for the amblyopic visual system should shift vertically on these axes, as shown by the model predictions (dashed curves) in Fig. 2c. Theoretically an early deficit in contrast sensitivity affecting local motion-energy detectors (c.f. cells in V1) would not be expected to produce a uniform vertical shift of the normal response function, since the responses to both the signal and the noise dots in the stimulus would be degraded to the same degree. An upwards translation of the response function along the y -axis indicates a change in the effective signal to noise ratio and presumably reflects the efficiency of the stage at which local motion signals are integrated to derive the global direction of image motion.

Both these predictions are well illustrated in Fig. 3. The filled green symbols in Fig. 3a show data for an individual amblyope (amblyopic eye) who exhibited reduced performance on this task as a result of a pure visibility deficit. While the filled red symbols in the Fig. 3b show data for another amblyope (amblyopic eye) who had reduced performance on this task because of a reduced sensitivity for the extraction of global motion.

Although these two individual amblyopes exhibited a selective loss in sensitivity it is plausible to expect that individual amblyopes might have reduced performance on this task for both of the above reasons. We therefore derived, for each subject, the relative extents of the contrast-based (visibility) deficit and the global motion-based deficit. We undertook this analysis separately for RDKs composed of first-order and second-order dots.

The raw data for individual amblyopic subjects (amblyopic eye) is displayed in Fig. 4 for both first-order

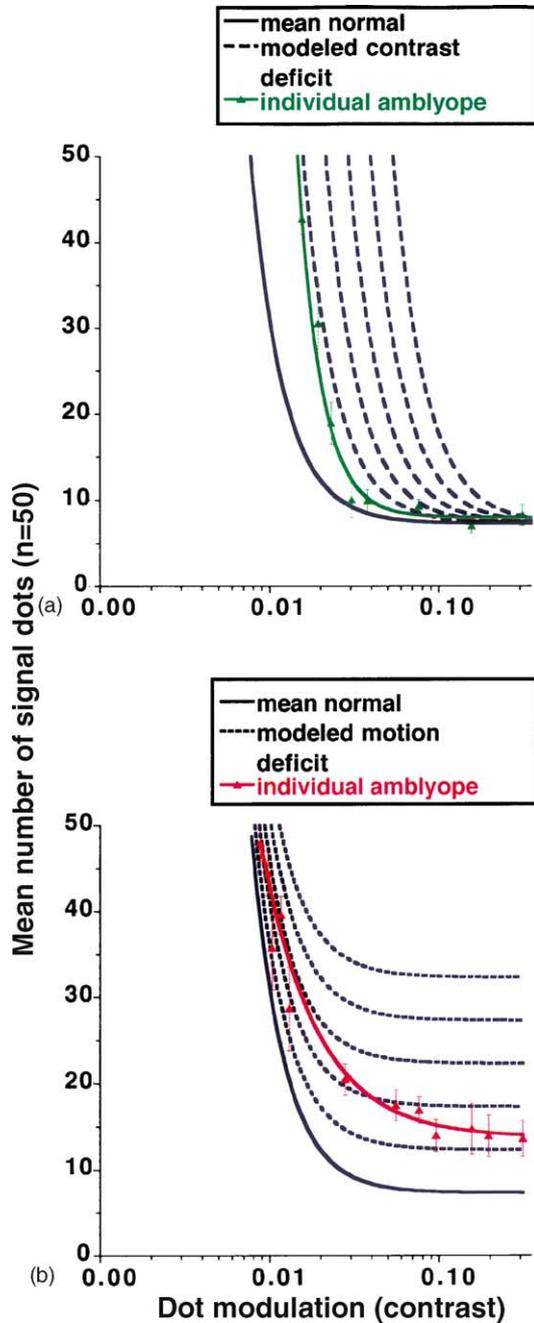


Fig. 3. Represents the mean response function (solid black line) for first-order global motion thresholds in the normal observer group. As in Fig. 2 the dashed lines represent the modeled (a) contrast-specific deficit and (b) a motion-specific deficit. (a) ▲ depicts the data of a single amblyope (± 1 s.e.m) who demonstrated a contrast-specific deficit only. (b) ▲ depicts the data of a single amblyope (± 1 s.e.m) who primarily demonstrated a global motion-specific deficit.

(Fig. 4a) and second-order (Fig. 4b) stimuli. While most amblyopes displayed reduced performance on this task, the underlying deficit was composed of both contrast (visibility), evident by a lateral shift in the response function, and global motion-based components evident by a vertical shift in the response function.

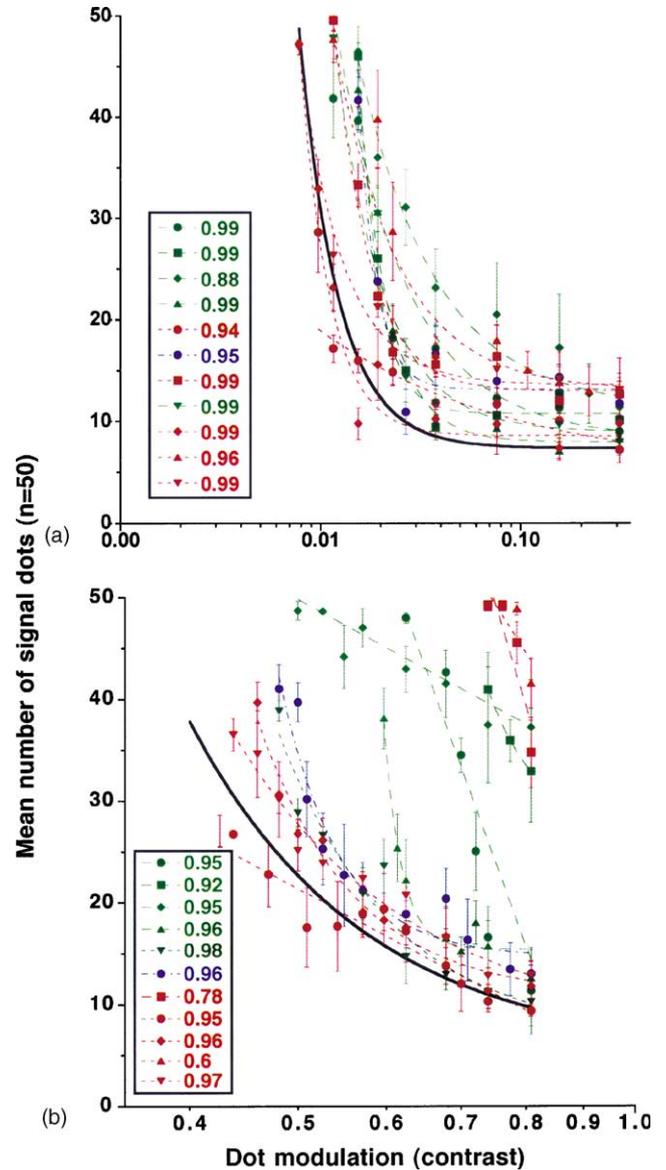


Fig. 4. Individual global motion thresholds. The solid black line represents the mean results for eight normal observers. (a) The global motion thresholds of each individual amblyope (amblyopic eye) for the luminance-defined, first-order dots and (b) shows the global motion thresholds of each individual amblyope (amblyopic eye) for the contrast-defined, second-order dots. Each datum represents the mean of five blocks of trials and error bars represent ± 1 s.e.m. Curves represent a power function fit to the data. An indication of the proportion of variance in the data that can be accounted for by the curve-fit/model is given by the individual r^2 values, inset within the plots.

Fig. 4 illustrates well the variability of deficit in the raw data for our group of amblyopic observers; it is difficult to assess the degree to which each response function is shifted either vertically or laterally. Therefore a summary of the relative contributions of visibility and global motion processing to the overall deficit is shown in Fig. 5 where the derived component anomalies for contrast and global motion deficits are plotted for each amblyope. These component anomalies were best de-

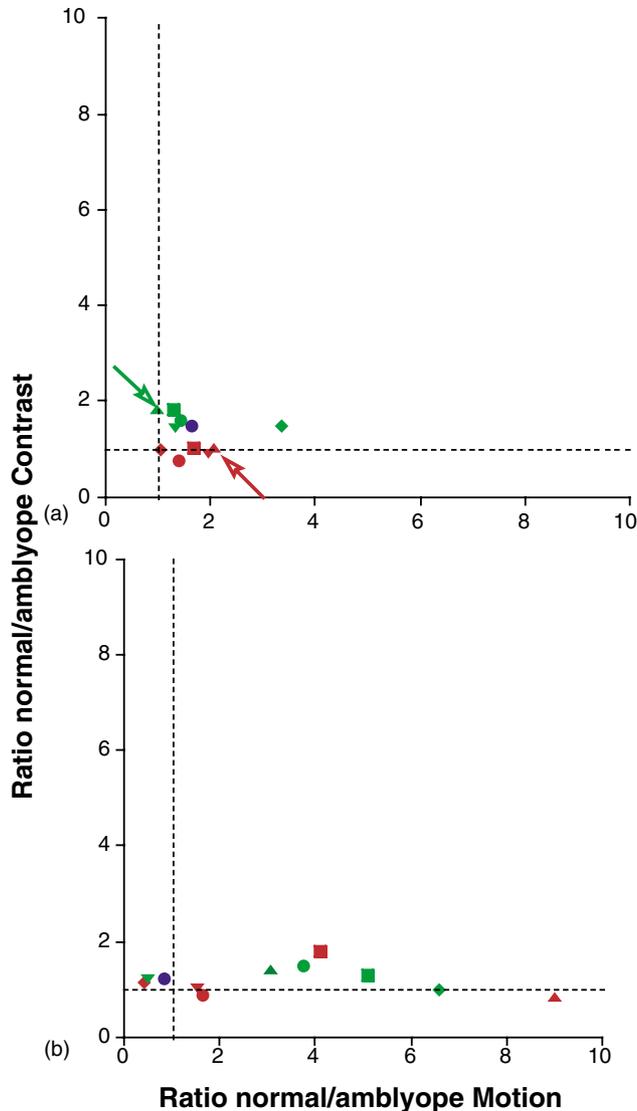


Fig. 5. Ratio of normal to amblyopic eye performance for both first-order (a) and second-order (b) stimuli. The dashed lines represent a ratio of one indicating no difference in threshold between the normal and amblyopic observers with respect to either the contrast or global motion extraction of the stimuli. Values falling along the horizontal dashed line are consistent with a global motion-specific deficit; values that fall along the vertical dashed line are consistent with a contrast-specific deficit. (Note: amblyope \blacktriangle previously depicted in Fig. 3a illustrates the presence of a contrast-specific deficit (data point lies along the vertical axis only) and amblyope \blacktriangle previously shown in Fig. 3b illustrates a global motion-specific deficit (data point lies along the horizontal axis only).)

scribed by independently fitting a two-parameter model, and taking the ratio of the best fitting parameters describing the lateral (contrast or visibility) and vertical (global motion sensitivity) shifts to the raw data, to bring them into correspondence with the mean performance exhibited by normal subjects. In all cases the numerator is represented by the mean values for the normal observers (see above) and the denominator is that of each individual amblyope. The dashed lines

represent a ratio of one indicating no difference in threshold between the normal and amblyopic observers with respect to either the contrast or global motion extraction of the stimuli.

For clarification the arrows in Fig. 5 indicate the relative contributions of visibility and global motion processing to the overall deficit for the two individual amblyopes shown earlier in Fig. 3a and b. In Fig. 3a amblyope \blacktriangle demonstrated a contrast-specific deficit; values that fall along the vertical dashed line in Fig. 5 are consistent with such a contrast-specific deficit. The raw data of amblyope \blacktriangle previously shown in Fig. 3b illustrated a global motion-specific deficit; values falling along the horizontal dashed line are consistent with a global motion-specific deficit.

To investigate whether or not there were any statistically significant differences in the patterns of deficits found for the amblyopic observers using first-order and second-order motion stimuli, a $2 \times 2 \times 2$ (mixed) analysis of variance (ANOVA) was performed on the data. The between-subject factor was *amblyopic subject group* (strabismic versus anisometropic strabimics). As there was only one anisometropic amblyope without measurable strabismus, this individual's data was excluded from the analysis in order not to violate the assumptions of ANOVA. The two within-subject factors were *stimulus type* (first-order versus second-order) and the *component anomalies* of the overall visual deficit (contrast versus global motion). There was no significant difference between the amblyopic subject groups [$F_{(1,8)} = 0.4$; NS], but there was a significant main effect of stimulus type [$F_{(1,8)} = 5.599$; $p = 0.0455$]. Thus overall, deficits in the processing of the second-order stimuli were significantly greater than those to the first-order patterns. The main effect of component anomalies was also significant [$F_{(1,8)} = 6.227$; $p = 0.0372$], indicating that global motion deficits, when collapsed across amblyopic subject group and stimulus type, were significantly greater than contrast (visibility) deficits. More importantly the interaction between stimulus type and component anomalies was significant [$F_{(1,8)} = 5.638$; $p = 0.0449$]. Exploration of this interaction, using an analysis of simple effects, revealed that the global motion deficit was significantly greater for second-order stimuli than first-order stimuli [$F_{(1,16)} = 11.22$; $p = 0.0041$] and also exceeded the contrast deficit for second-order stimuli [$F_{(1,16)} = 11.742$; $p = 0.0035$]. None of the other possible interactions reached significance.

Conventional visual acuity measures in the amblyopic eye were not found to be a reliable indicator of overall performance with no significant correlations between either first-order contrast [$r_{(11)} = 0.02$; NS] or motion [$r_{(11)} = 0.07$; NS] and second-order contrast [$r_{(11)} = 0.22$; NS] or motion [$r_{(11)} = 0.43$; NS]. This is evident in Fig. 5a and b where the poorest ($\blacktriangle/200$) and best ($\blacktriangledown/30$) individual visual acuities in the amblyopic subject group

do not then consequently dictate the upper and lower limits of either the contrast *or* motion based deficit.

It is important to add that in comparison to the normal population of non-amblyopic observers, several of the fellow fixing eyes of the amblyopes also demonstrated specific deficits in global motion processing (Fig. 6). With a mean ratio of normal to fellow eye performance of $1:1.4(\pm 0.6)$ for first-order increasing by up to a factor of three ($1:1.66\pm(1.05)$) for second-order global motion processing. The amblyopic results were therefore compared to those of non-amblyopes to avoid any assumption about the normality of the fellow eye in amblyopia (Giaschi, Regan, Kraft, & Hong, 1992).

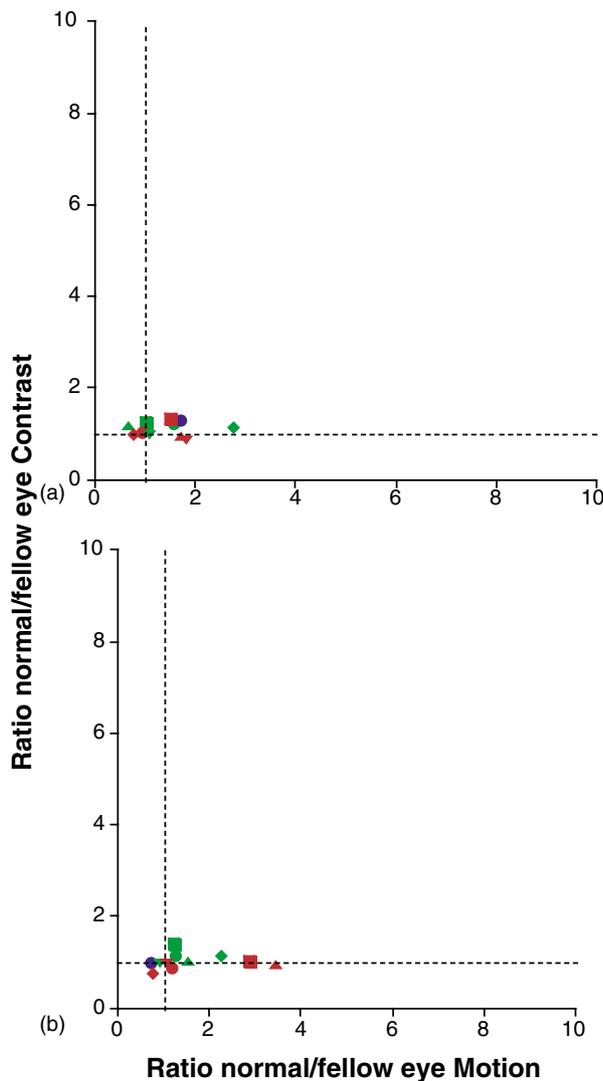


Fig. 6. Ratio of normal to fellow eye of amblyopic subjects performance for both first-order (a) and second-order (b) stimuli. The dashed lines represent a ratio of one indicating no difference in threshold between the normal and fellow eye of amblyopic observers with respect to either the contrast or global motion extraction of the stimuli. Values falling along the horizontal dashed line are consistent with a global motion-specific deficit; values that fall along the vertical dashed line are consistent with a contrast-specific deficit.

To summarize; first, there are global motion processing deficits in amblyopia that cannot be simply explained by reduced visibility as a result of the contrast sensitivity loss. Second, the global motion deficit is larger for second-order stimuli. Further analysis of the data also revealed that the deficits for global motion and visibility were not significantly correlated for both first-order [$r_{(11)} = -0.097$; NS] and second-order [$r_{(11)} = -0.158$; NS] stimuli.

4. Discussion

A number of previous studies have provided indirect evidence for a motion processing deficit in amblyopia (Donahue & Wall, 1994; Kubova, Kuba, Juran, & Blakemore, 1996; Levi, Klein, & Aitsebaomo, 1984; Tang et al., 1998). Our study, however, is the first to use global motion stimuli, to show directly that there is a deficit for global motion processing in human amblyopia. Firstly, we show that this overall deficit is composed of contrast- and signal:noise-dependent components. Secondly, we demonstrate that these global motion deficits are greater for second-order or contrast-defined stimuli. Finally we show that the fellow fixing eye is also affected, albeit to a lesser degree.

In terms of the currently accepted two-stage model for global motion detection, this suggests deficits involving both stages. The contrast-dependent deficit, on the basis of the available neurophysiology, is likely to be located in the striate cortex and to be related to the contrast sensitivity deficit. The signal:noise deficit is likely to involve the dorsal pathway of the extra-striate cortex where it has been shown that the integration of local motion directional signals takes place (Baker et al., 1991; Movshon et al., 1985; Newsome & Pare, 1988; Salzman et al., 1992).

In this present study we cannot definitively rule out the possibility that the striate cortex does not contribute to the signal:noise deficit observed in amblyopia. Even if the signal:noise ratio in the second (extra-striate) stage of global motion processing is lower in amblyopia, this may not rule out an explanation in terms of a striate deficit because performance in such a global motion task, could, in principle be reduced because of a primary deficit to motion detectors in V1 i.e. a reduction or larger bandwidth for directionally selective cells. Neither of which need imply a loss of contrast sensitivity. This seems unlikely for a number of reasons. Firstly, in human amblyopes, directional thresholds for simple grating stimuli of the type that optimally stimulate V1 neurons are normal (Hess & Anderson, 1993). Secondly, in amblyopic animals, the number, temporal properties and the directional selectivity of neurons in V1 driven by the amblyopic eye has also been shown to be normal (Movshon et al., 1987). Finally, as it cannot be excluded

that some of the fellow eyes show impaired performance, this suggests that the abnormality must at least in part affect the pathway at a point where the majority of the neurons are binocular (e.g. extra-striate cortex). Interestingly, we have also recently reported abnormalities in the fellow eye of amblyopes for a series of blur and orientation discrimination and inter-ocular matching tasks (Simmers & Bex, in review; Simmers, Bex, & Hess, in press).

We also show that the global motion processing deficit is larger for second-order stimuli and that this is not a consequence of the reduced visibility of the stimuli. Furthermore, the deficits for first-order and second-order stimuli are uncorrelated, suggesting at least two separate global motion-processing deficits. This dissociation in itself is suggestive of an extra-striate site because the available evidence suggest that, in the striate cortex, single cells do a double duty by processing both first- and second-order information (Baker, 1999; Zhou & Baker, 1994). Recently it has been shown that second-order spatial processing may also be more severely affected in amblyopia (Wong et al., 2001). Our results show that this is not limited to spatial processing but also includes motion processing mechanisms.

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