Second-Order Optic Flow Deficits in Amblyopia

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Purpose. Amblyopic observers show deficits for global motion discrimination that cannot be accounted for by their contrast sensitivity impairment. The processing of first- and second-order translational global motion is deficient, as is the processing of first-order optic flow, suggesting that cortical function in extrastriate areas is impaired. The authors sought to determine whether amblyopes show impairment in the processing of optic flow defined by second-order motion, whether these deficits are comparable in the two eyes, and whether these deficits are correlated with first-order deficits.

Methods. Eight amblyopic subjects (three strabismic, three strabismic-anisometropic, one anisometropic, one deprivation; mean age, 29 years) were tested. The authors used random dot kinematograms in which the dots were luminance or contrast modulations of background noise. The global pattern of dot motion within the stimulus area was translational, radial, or rotational. Coherence thresholds for direction discrimination were obtained across a range of dot modulation depths, allowing the separation of contrast and motion deficits.

Results. The present study showed that deficits in second-order optic flow processing were equivalent to those for first-order stimuli and that these were unrelated to the extent of the amblyopic contrast sensitivity deficit and were comparable in both eyes. Radial optic flow was more affected than rotational optic flow.

Conclusions. Global motion impairment appeared to have a high-level binocular locus and was independent of the depth of the contrast deficit. Results also support the idea that global motion and optic flow processing are form-cue invariant. (*Invest Ophthalmol Vis Sci.* 2007;48:5532–5538) DOI:10.1167/iovs.07-0447

The major features of amblyopia are loss of visual acuity and contrast sensitivity. A key factor determining the pathology seems to be whether binocular function is spared.[1] The locus of the acuity and contrast impairment is most likely visual area V1, where neurons show abnormalities in spatial scale and binocular organization.[2] Functional neuroimaging shows reduced V1 activation from the amblyopic eye.[3,4] In addition to the early contrast and acuity deficits in amblyopia, evidence is now mounting for separate, downstream impairments for "higher-level" processes traditionally associated with extrastriate visual areas. Amblyopes show deficits in numerating features,[5] facial representation,[6] and global motion.[7,8]

In this study we were interested in expanding the description of the global motion deficit so that the extent of the processing anomaly along the dorsal pathway could be gauged. Previous studies have shown that there is a primary deficit (not a consequence of defective low-level local motion processing) to the processing of global translational motion[7] and to the global processing of radial and rotational motion (optic flow).[9] This suggests that the functions attributed to extrastriate area MT, among others—namely its primary role in the integration of translational motion signals from V1[10–12]—and the functions that have been attributed to extrastriate area MSTd among others—namely its role in optic flow analysis[13–18]—may be compromised in amblyopia. Another piece of the puzzle regarding the extent of the extrastriate dorsal pathway deficit in amblyopia is that second-order global translational motion is more affected than its first-order counterpart.[7] First-order refers to stimuli in which the moving elements are defined by a change in luminance with respect to the background, whereas second-order refers to stimuli in which these elements are defined by a change in contrast.[19,20] This may implicate specific regions within the extrastriate cortex known to specialize in the integration of second-order motion (such as the anterior superior parietal lobule and lateral occipital regions) as opposed to first-order motion (precuneus and medial occipital cortex).[21]

Several pieces of information relevant to establishing the full extent of the dorsal pathway deficit in amblyopia are missing. First, we do not know whether the processing of second-order optic flow is deficient and, if it is, whether it is affected more than its first-order counterpart, which appears to be the case for translational motion. This bears on the full extent of the optic flow deficit because there is evidence that extrastriate areas exhibit specialization for either first- or second-order global motion processing. Given the psychophysical evidence for independence of first- and second-order optic flow processing,[22] second-order optic flow may be spared or affected to a greater extent than first-order optic flow. Second, we do not know the relationships among the deficits for translational, rotational, and radial motion. A better understanding of this would help to establish whether the loss of function for optic flow was merely a consequence of an impaired translational motion input from area MT[16,23] or whether separate deficits extend along the dorsal pathway as far as area MSTd. Recent findings of Simmers et al.[9] concerning first-order optic flow suggest the latter conclusion, but a more complete answer would be obtained by comparing the deficits for first- and second-order stimuli. Third, we do not fully understand why and to what extent the normal fixing eye of amblyopes is affected in global motion tasks. Comparing fixing and amblyopic eye deficits for translational, rotational, and radial motion and also for first- and second-order stimuli would provide a more complete picture of the loss of function that we know does occur for the fixing eye. The relevance of this information is that if the binocular connections to extrastriate cortex (e.g., MT) are spared, one would expect to find comparable motion

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TABLE 1. Clinical Details of the Amblyopic Observers

Subject	Age (y)	Acuity	Stereo	Prescription	Alignment	Amblyopia
EW	26	R: 20/40 L: 20/20	0/10	No prescribed correction	R: esotropic 10°	Strabismic
ED	46	R: 20/20 L: 20/70	5/10	R: +0.75 L: +0.75	L: esotropic 4°	Strabismic
XL	33	R: 20/20 L: 20/400	3/10	R: −2.5 L: −2.75/+0.75 × 110°	L: esotropic 15°	Strabismic
JL	29	R: 20/20 L: 20/63	0/10	R: plano L: +2.5	L: exotropic 5°	Strabismic-anisometropic
ML	24	R: 20/80 L: 20/25	0/10	R: +1.0/−0.75 × 590° L: −3.25	R: esotropic 6°	Strabismic-anisometropic
PH	36	R: 20/20 L: 20/65	0/10	R: −2.0/+0.5 × 90° L: +0.5 × 90°	L: esotropic 5°	Strabismic-anisometropic
WM	21	R: 20/20 L: 20/60	0/10	R: plano L: +1.75/−0.5 × 180°	—	Anisometropic
SA	18	R: 20/560 L: 20/100	0/10	R: plano L: −3.25 +0.5 × 90°	R: esotropic 23°	Deprivation

R, right eye; L, left eye.

deficits in fixing and fellow amblyopic eyes. Recent evidence suggests that the binocularity of global motion processing is derived from its local motion input from lower visual areas such as V3 rather than at the site of global integration in the extrastriate cortex.[24]

To address each of these three key issues, we measured performance for the direction discrimination of first- and second-order global motion stimuli undergoing translational, radial, and rotational motion in the fixing and amblyopic eyes of a group of amblyopes and controls. The results suggest that the processing deficits for optic flow also affect second-order processing, to the same extent (and not greater) than that of its first-order counterpart. Translational and radial global motion are affected to a greater extent than rotational motion, and the global motion deficit in the amblyopic and fellow fixing eye are of comparable magnitude for different types of global motion, be it first- or second-order, translational, or radial. This suggests that the mechanisms affected by the amblyopic global motion deficit are form-cue invariant.

METHODS

Observers

This study adhered to the tenets of the Declaration of Helsinki. All observers gave informed consent after an explanation of the study. The nonamblyopic observers consisted of one of the authors (CAS) and five experienced observers naive to the purposes of the experiment. Four observers were emmetropic and had 20/20 acuity; the other two observers were myopic and a mean correction of −4.25 D, which they wore for the course of the experiment. Average age of the nonamblyopic participants was 29.1 years (SD, ±5 years), and all had previously participated in psychophysical experiments. The details of the amblyopic observers are presented in Table 1. Average age of the amblyopic participants was 29 years (SD, ±9 years). Stereovision was assessed using the stereovision test (Randot SO-002; Stereo Optical, Inc., Chicago, IL). Acuity was measured using a logMAR chart.

Apparatus and Stimuli

Random dot kinematograms (RDKs) were generated by custom software on a computer (Macintosh G4; Apple, Cupertino, CA) and displayed on a 22-inch CRT monitor (Diamond Pro 2070SB; Mitsubishi, Irvine, CA). The resolution of the screen was 1078 × 768 pixels, and the frame rate was 75 Hz. The display was γ-corrected with the use of internal look-up tables by a psychophysical technique described elsewhere.[25]

The RDKs were "movies" composed of eight consecutively presented frames; each frame was presented for 53 ms. The total presentation duration was, therefore, 427 ms. The RDKs contained 50 nonoverlapping dots (radius, 0.235°), which were presented in a circular window with a diameter that subtended 12° of visual angle from the viewing distance of 93 cm. This resulted in an average dot density of 0.44 dots/deg^2. A circular portion of the display centered at fixation (radius, 0.7°) was occluded (i.e., set to mean luminance) to prevent the sudden appearance or disappearance of dots at fixation acting as a potential cue to global motion direction in the radial stimuli. A pilot study demonstrated that observers could use this cue, and this resulted in artificially low thresholds for radial motion. Inclusion of a foveal occlusion zone eliminated this advantage.

All the dots were displaced 0.3° on each frame, producing a speed of 5.6°/s. If a dot exceeded the boundary of the display area, it was wrapped around to reappear at the opposite edge of the stimulus area. The direction in which the dots were displaced depended on the condition and on whether a dot was assigned to be a "signal dot" or a "noise dot." In the *translational* condition, signal dots were displaced vertically, upward or downward. In the *radial* condition, signal dots were displaced outward or inward. In the *rotational* condition, signal dots were displaced clockwise or counterclockwise. Noise dots were always displaced in a random direction. On each frame, dots were randomly reassigned to be either a noise dot or a signal dot so that subjects could not complete the task by tracking a single dot.

The background of the stimulus presentation area was composed of two-dimensional, static, binary noise with a Michelson contrast of 0.1. Each noise element was assigned a single luminance value (randomly chosen to be either "black" or "white" with equal probability) and was composed of a single screen pixel to avoid potential luminance artifacts.[26] A different stochastic noise sample was used for every motion sequence that was generated. The remainder of the display was set to the mean luminance of the monitor (~40 cd/m^2). Each dot was a circular region of noise elements, and either the mean luminance (in the case of first-order stimuli) or the mean contrast (in the case of second-order stimuli) of the noise within the dot could be increased relative to that of the noise in the background. The modulation depth of the dots refers to this increase in luminance or contrast. In first-order stimuli, the modulation depth is defined as

$$\text{Modulation depth} = (DL_{mean} - BL_{mean})/(DL_{mean} + BL_{mean})$$

where DL_{mean} and BL_{mean} refer to the mean dot luminance and background luminance, respectively. In second-order stimuli the modulation depth is defined as

$$\text{Modulation depth} = (DC_{mean} - BC_{mean})/(DC_{mean} + BC_{mean})$$

where DC_{mean} and BC_{mean} refer to the mean dot contrast and background contrast, respectively. Figure 1 shows single frames of the first-order (left) and second-order (right) stimulus.

In line with previous studies that have used comparable radial and rotational RDK stimuli (e.g., Simmers et al.,[9] Burr and Santoro,[27] Aaen-Stockdale et al.[28]), 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 a strictly rigid radial or rotational flow field) so that performance could be directly compared with the translational RDK stimuli. Indeed many studies suggest that neurons in MSTd are relatively insensitive to the presence or absence of speed gradients within the receptive field.[17,29,30]

Procedure

A single-interval 2AFC staircase procedure was used to obtain observers' global motion thresholds for each of a range of dot modulation depths (visibility levels). The trials all began with presentation of a fixation cross in the center of the display, which was replaced by an RDK stimulus. The task of the subject was to identify the global motion direction (up/down, outward/inward, clockwise/counterclockwise according to the condition) and to respond with a button press. Initially, all dots were displaced in the "signal" direction. An adaptive 1-up, 3-down staircase procedure[31] was used to vary the percentage of signal dots to converge on the observers' motion coherence threshold, which was defined as the stimulus coherence (minimum number of signal dots) supporting 79% correct performance. The step size of the staircase was initially set to eight signal dots; this was subsequently halved for each reversal so that after the third reversal the step size was reduced to a single dot. The staircase terminated after eight reversals, and the threshold value was calculated as the mean of the last six reversals. Thresholds were obtained for all three motion types (translational, radial, and rotational) and stimulus classes (first-order and second-order) at each of a range of modulation depths. Observers repeated each condition five times, and the reported thresholds are the mean of these five staircases.

Analysis

For each subject, the mean of five staircases was taken as the threshold for each modulation depth. The mean motion coherence threshold across the normal observers as a group was calculated for each dot modulation depth condition that was tested. As in previous studies using this technique,[7,9,28,32] the individual *and* group data were fit well with a curve of the form $y = ax^b + c$, where y is the motion coherence threshold, x is the dot modulation depth (contrast) and a, b, and c are constants. Above a certain contrast, thresholds are limited by the efficiency with which the visual system can integrate the local motions. Below this value, thresholds depend on the contrast of the stimulus (Fig. 2, thick line). The results for the normal observers on this task have been presented and discussed previously.[28]

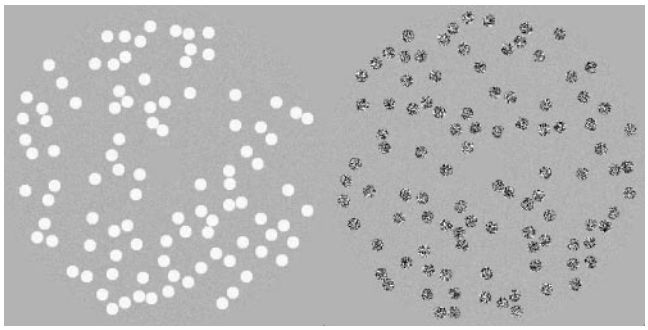

FIGURE 1. Example frames of the luminance-modulated first-order stimulus (*left*) and the contrast-modulated second-order stimulus (*right*).

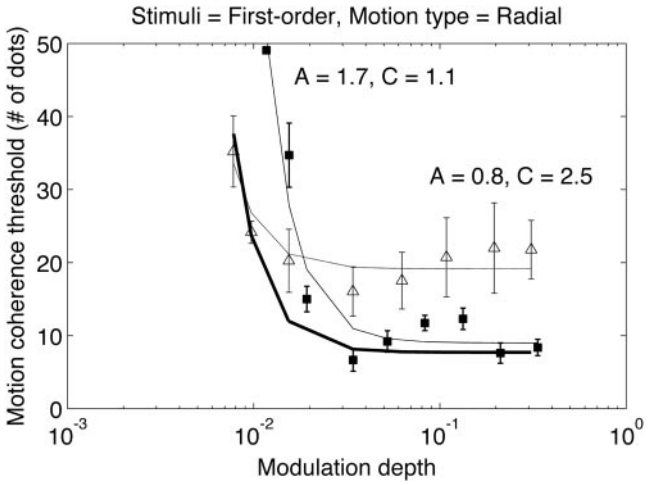

FIGURE 2. Data for two amblyopic observers viewing first-order radial motion with the amblyopic eye. *Thick line*: normal data. *Closed squares*, *thin curve*: $r^2 = 0.94$; "pure" contrast deficit; observer XL. *Open triangles*, *dashed curve*: $r^2 = 0.83$; "pure" motion deficit; observer EW. Corresponding values for parameters A and C are shown next to the fits.

To separate and quantify the contrast deficit and global motion deficit in the amblyopic observers, we calculated by how much we would have to shift the best-fitting curve for the observers along the x-axis (for contrast encoding deficits) and the y-axis (for motion-encoding deficits) to bring it into correspondence with the data for each *individual* amblyope. To achieve this, the coherence thresholds for an individual amblyopic observer were plotted as a function of the dot modulation depth, and the following curve was fit to the data:

$$y = a(x/A)^b + (c*C)$$

where a, b, and c are the constants obtained from the fit to the normal data for each condition. The values A and C thus attained encode the ratio of normal performance to amblyopic performance with respect to contrast and motion encoding, respectively, for that person. The model was fitted for each amblyopic observer individually for each condition. This technique is described in more detail in Simmers et al.[7] Figure 2 shows the first-order radial data from two amblyopic observers, one of whom shows a relatively pure contrast deficit and the other who shows a relatively pure motion deficit, and the obtained values of A and C.

R^2 values were calculated for each fit. Overall, the curve was a good fit to the data. Fifty-two percent of the fits had an R^2 value greater than 0.9, whereas 77% of the fits had an R^2 value greater than 0.8.

Statistical Analysis

ANOVA was carried out using SPSS software (SPSS; Cary, NC), and the correlation coefficients were calculated with a math program (Matlab; Mathworks, Natick, MA). Correlation coefficients were calculated from data that were averaged over the appropriate conditions so that each subject was only represented by one data point. This was done to avoid artificially inflating the resultant significance by assuming too many degrees of freedom.

RESULTS

The values of A and C that result from this fit are plotted in Figure 3 against one another for each condition, with first-order conditions in the left column and second-order condi-

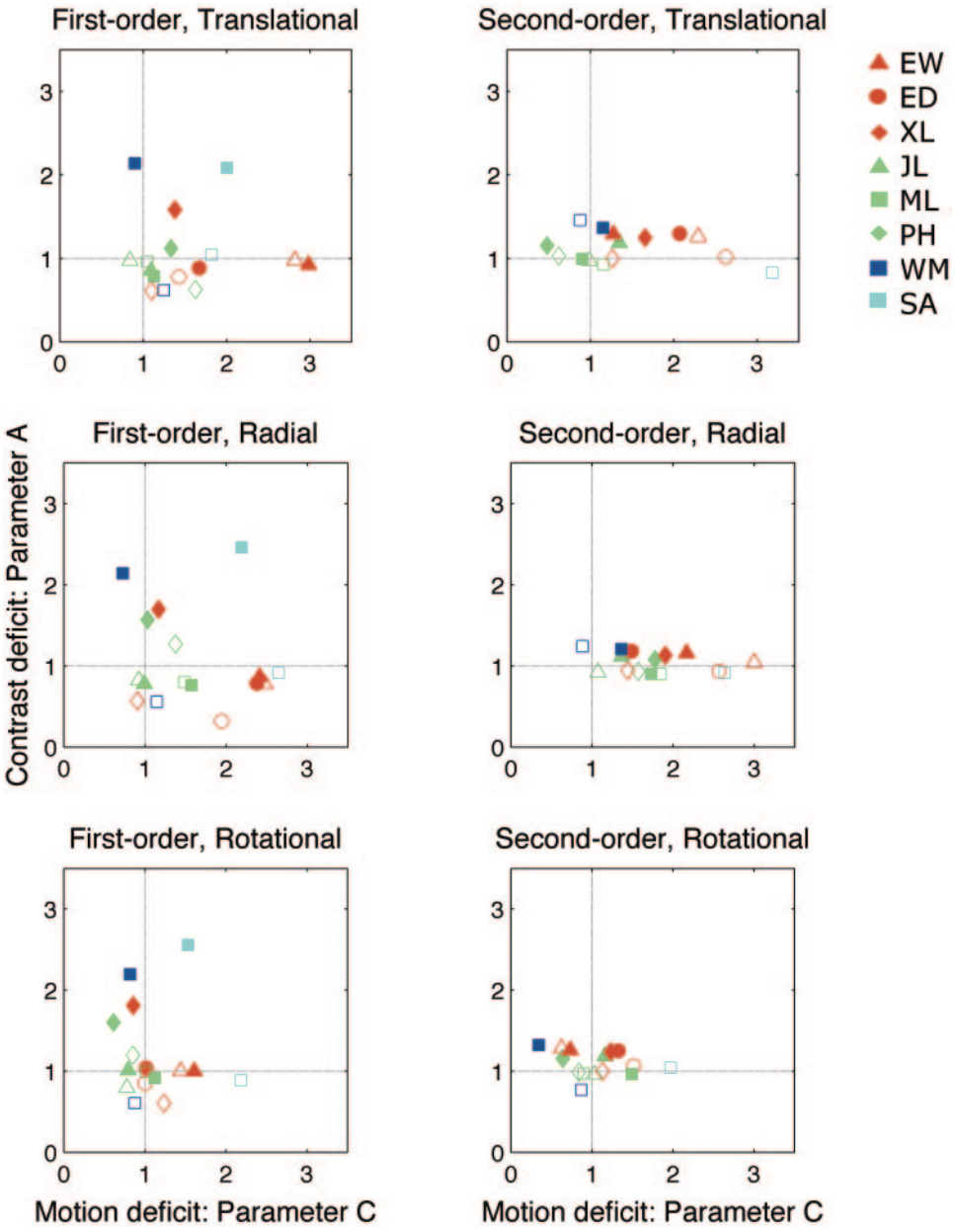

FIGURE 3. Contrast and motion deficits for all amblyopic observers in all conditions. *Red* symbols correspond to strabismic amblyopes, *green* symbols correspond to strabismic-anisometropic amblyopes, *blue* symbols correspond to anisometropic amblyopes, and *cyan* symbols correspond to deprivation amblyopes. *Solid* symbols: amblyopic eye (AE); *open* symbols: fellow eye (FE).

tions in the right column. Data falling on the vertical dotted lines indicated a deficit limited purely to contrast, and data falling on the horizontal dotted lines indicated a deficit involving only the computation of global motion. In a few cases, the observer's performance was limited only by the visibility of the stimulus, but often the observer's contrast threshold was approximately normal, and impairment was restricted to the processing of global motion. Data falling on the diagonal indicated equal deficits for contrast and motion. One observer (SA) with deprivation amblyopia could not discriminate second-order motion with her amblyopic eye; therefore, the data for this observer are missing for that condition. For the first-order conditions, this observer showed marked contrast *and* motion deficit in her amblyopic eye. Interestingly, she showed normal contrast threshold but a large motion deficit in her fellow eye.

Three-way ANOVA was carried out on the motion and contrast deficits to identify any differences in the group means. For motion deficits, a main effect was found of motion type ($F(2,12) = 9.7$, $P = 0.003$) because of the much smaller size of rotational motion deficits (Fig. 4, left). For contrast deficits, a main effect was found of eye ($F(1,6) = 5.9$, $P = 0.05$). No other main effects or interactions were significant.

Analysis of the group means might not have revealed important patterns in the data, so an analysis of correlations between the conditions was undertaken. First, it was important to establish whether the two deficits were independent by determining whether the motion deficit was correlated with the contrast deficit. The left-hand plot of Figure 5 shows the data from all conditions. The motion deficit does not appear to be correlated with the contrast deficit for either the amblyopic or the fellow eye. The deficits for each subject were averaged across motion type and motion class (Fig. 5, right), and a correlation coefficient was calculated on this averaged data. Correlation coefficients showed no significant correlation between the severity of the contrast deficit and the severity of the motion deficit in either the amblyopic ($r = 0.24$; $P =$ NS) or the fellow ($r = 0.38$; $P =$ NS) eye.

Figure 6 shows deficits for contrast (left) and motion (middle) in amblyopic and fellow eyes. Contrast deficits measured in the amblyopic eyes show no correlation to those measured

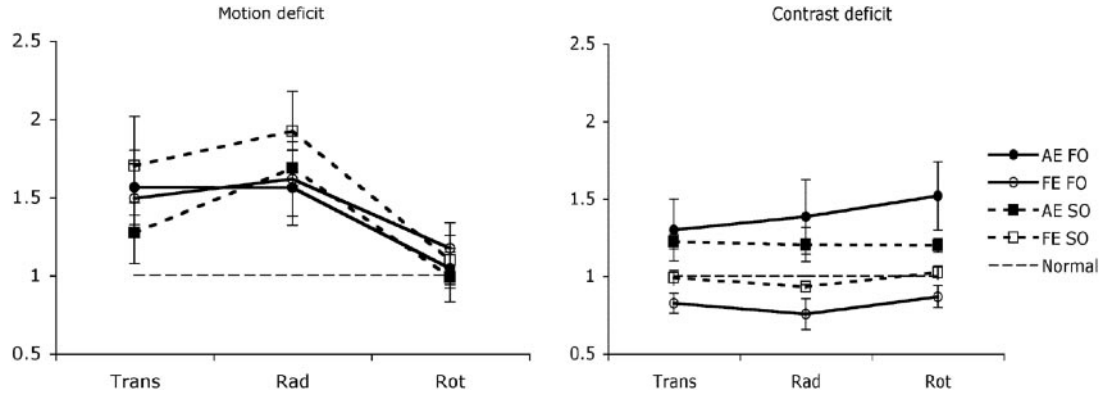

FIGURE 4. Group data.

in the fellow eyes, with virtually all the variation in the data captured by the amblyopic eyes. Motion deficits, on the other hand, showed a strong correlation between the amblyopic and fellow eyes. These relationships also hold if first-order (dashed lines) and second-order (dotted lines) conditions are analyzed separately. Correlation coefficients comparing the two eyes of the amblyopic observers were calculated on data averaged across motion type and class. This averaged data is shown in the right-hand plot of Figure 6. The correlation coefficient for contrast deficits was nonsignificant ($r = -0.23$; P = NS), whereas the correlation coefficient for motion deficits was highly significant ($r = 0.92$; $P < 0.001$) and close to unity. Because the motion deficits in each eye were so tightly correlated and close to unity, the following analyses averaged motion deficits across eyes.

Examination of the group means (Fig. 4) shows that the class of the stimulus (first- or second-order) did not greatly, or systematically, affect the group means across conditions. This is corroborated by the results of ANOVA, which showed no main effect of stimulus class. We wondered, therefore, whether first- and second-order deficits would be correlated. The plot of Figure 7 shows the first- versus second-order deficits for the three different types of motion, averaged across eye. For radial motion, first- and second-order deficits show a highly significant positive relationship ($r = 0.94$; $P = 0.0003$) that lies close to unity. Correlations between first- and second-order deficits for translational ($r = 0.43$; P = NS) and rotational ($r = 0.57$; P = NS) motion proved nonsignificant, despite also being close to unity. Previous work has suggested that second-order deficits, measured using this same technique, are greater than first-order deficits.[7] Although inspection of Figure 7 appears to support this assertion in that approximately twice as many data points lie above the unity line than below it, the results of the ANOVA and a *t*-test carried out on first- and second-order deficits revealed that second-order deficits are no worse, on average, than first-order deficits ($t(44) = -0.9$; $P = 0.4$).

Partial correlations were run on motion deficits according to motion type (translational, radial, and rotational motion). A partial correlation measures the degree of association between two random variables, with the effect of a set of controlling random variables removed. For example, a strong correlation between two variables (say, x and y) could be attributed to some relationship between x and y or it could be attributed to their individual relationship to a third variable, z. If first- and second-order deficits are investigated independently, none of the correlations prove significant ($P > 0.05$ in all cases). If first- and second-order deficits are averaged, then translational and radial motion deficits, controlling for rotational motion, show a significant correlation ($R = 0.84$; $P = 0.02$), but motion deficits for translational and rotational motion, controlling for radial motion ($R = 0.16$; NS), and motion deficits for radial and rotational motion, controlling for translational motion ($R = 0.26$; NS), do not show significant correlation.

DISCUSSION

The amblyopic deficit in global motion processing, be it for first- or second- order, translational, rotational, or radial motion, is independent of the depth of the contrast sensitivity deficit, supporting the previous conclusions of Simmers et al.[9] that the site of the impairment is beyond V1.

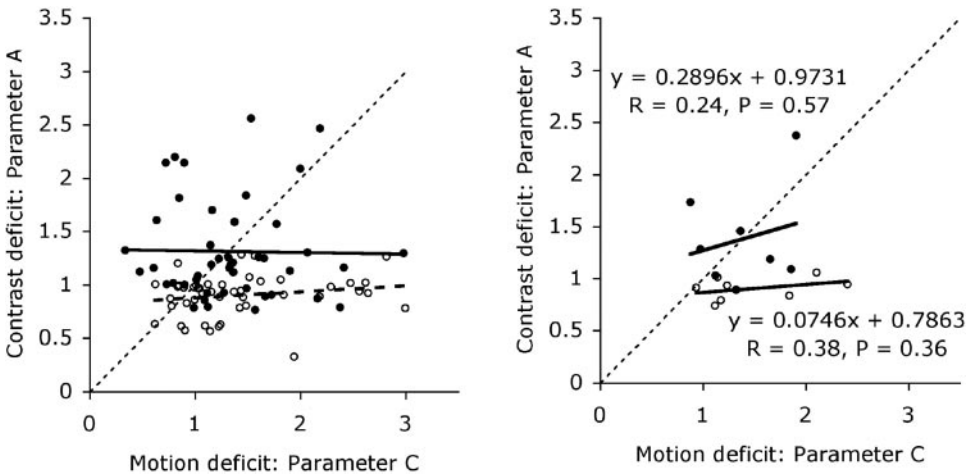

FIGURE 5. Contrast and motion deficits. *Left*: data from all conditions. *Right*: each subject's average deficits across motion type and motion class. *Solid circles*: amblyopic eyes; *open circles*: fellow eyes. Neither eye showed a strong correlation between the two deficits.

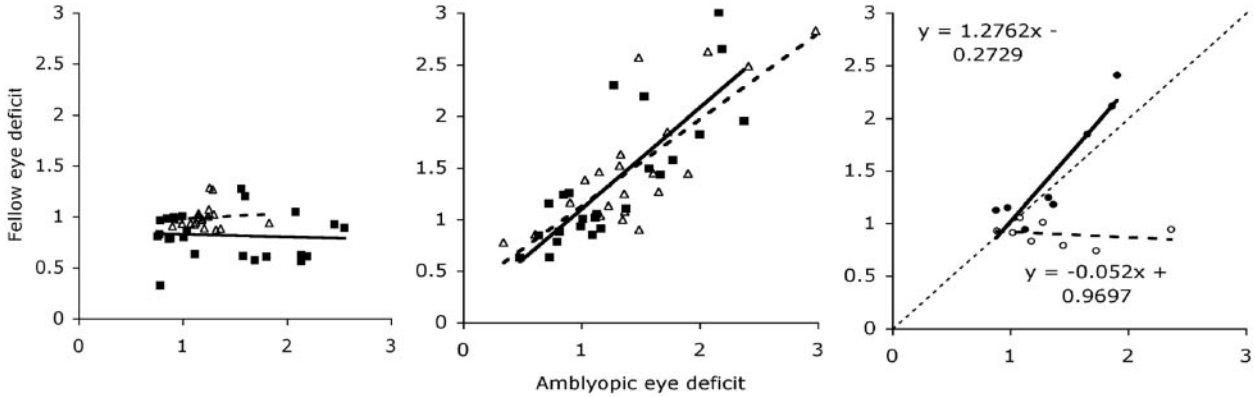

FIGURE 6. Comparison of contrast deficits (*left*) and motion deficits (*middle*) measured in observers' fellow eyes compared with their amblyopic eyes. *Solid squares*, *solid lines*: first-order conditions; *open triangles*, *dashed lines*: second-order conditions. *Right*: subjects' average deficits across motion type and motion class. *Closed circles*: motion deficits; *open circles*: contrast deficit. The same pattern of results holds in the raw and averaged data and in second- or first-order separately.

To What Extent Is the Fellow Eye Affected?

An interesting finding to come out of this study is the near unity relationship between the two eyes in the extent of the measured motion deficit. No such correlation occurs for the contrast deficit, with virtually all the variation occurring in the amblyopic eye. This suggests that the contrast deficit occurs as a result of a low-level impairment in monocular regions activated solely by the amblyopic eye, consistent with the established V1 deficit.[2] The motion deficit, however, seems to originate in a binocular region. It has recently been argued[24] that the binocularity of normal global motion perception originates from its inputs from lower-level areas such as V3. If this is so, the present results concerning the fixing eye suggest that the binocularity of these cortical regions is preserved in human amblyopia. Impairments in global motion perception are greater after early binocular form deprivation than after early monocular form deprivation,[8] and this sparing of global motion perception in the deprived eye suggests that areas involved in the integration of global motion benefit from input from the nondeprived eye. Ellemberg et al.[8] suggest that collaborative interactions beyond primary visual cortex enable relative sparing of some visual functions after monocular deprivation. It has also been found that the nonamblyopic eye can show impaired discrimination of motion-defined form.[33]

Another possibility is that the observers with deepest amblyopia might have received greater amounts of occlusion therapy of the fixing eye, leading to a motion deficit similar to that in the amblyopic eye. We looked at the size of the motion deficit in the fellow eye as a function of the duration of patching reported in the amblyopic observers' history. This turned out to be a poor predictor of the size of the motion deficit in the fellow eye ($r = 0.66$; P = NS); therefore, we find no support for this explanation.

One caveat should be added, however. Kiorpes et al.[34] showed in amblyopic monkeys that the relative global motion deficit in the two eyes changes as a function of displacement/speed in a translating RDK. The present study used a single displacement/speed so it would be interesting to establish whether the interocular correlation is similarly strong at higher or lower speeds or larger/smaller displacements.

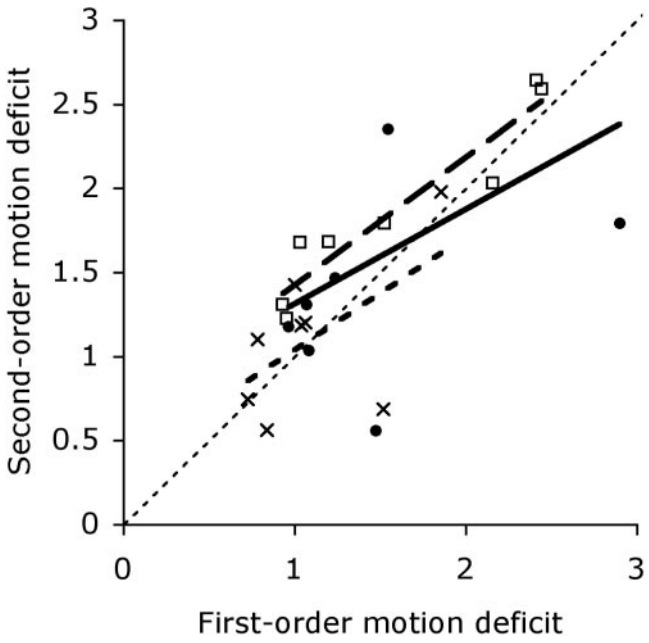

FIGURE 7. Correlation between first-order and second-order deficits for different types of motion. *Thin dashed line*: unity slope. *Thick, solid line*: result of a linear regression on translational deficits (*circles*). *Thick, dashed line*: result of a linear regression on radial deficits (*squares*). *Thick, dotted line*: result of a linear regression on rotational deficits (*crosses*).

What Is the Relationship among the Deficits for Translational, Rotational, and Radial Motion?

Our results suggest that deficits do occur for optic flow processing, with radial motion affected more than rotational motion. We also find a strong correlation between the deficits for translational and radial motion, suggesting interdependence. Simmers et al.[9] concluded that there was no strong correlation between translational motion deficits and radial/rotational deficits. Perhaps because of the greater statistical power in our study, a result of including second-order conditions and fellow eye data in the analysis, we found that radial (but not rotational) motion deficits were significantly correlated with translational motion, possibly suggesting that impairment at the level of global translational motion processing (i.e., V5) may underlie the radial motion deficit (i.e., MST).

The fact that the magnitude of the deficits for radial and rotational motion are different is consistent with the idea that they are analyzed by separate mechanisms, perhaps the cardinal mechanisms proposed by Morrone et al.[35] Some investigators, however, suggest that radial and rotational detectors

could simply be separate channels in a "generalized spiral" motion mechanism.[36] One ecological reason radial and rotational motion may be analyzed by different mechanisms is that both types of motion are caused by different types of self-motion. Although translational and rotational motion can be, and often are, caused by movements of the eyes and head, radial motion is usually caused by the movement of objects in depth or locomotion (i.e., three-dimensional [3D] motion). Aaen-Stockdale et al.[28] show that coherence thresholds for second-order radial motion are consistently higher than those for second-order translational or rotational motion because of a longer temporal integration period for second-order radial motion. It is, therefore, possible that radial motion undergoes additional processing by an entirely separate mechanism, such as motion-in-depth analysis, given its role in locomotion and 3D motion, and evidence suggests that such 3D analyses are impaired for second-order motion.[37]

Is Second-Order Optic Flow Processing Affected in Amblyopia?

Our findings suggest that second-order processing of optic flow is affected in amblyopia, but not to a greater extent than its first-order counterpart. Simmers et al.[7] suggested that, for translational motion, the deficit for second-order stimuli was much greater than that of its first-order counterpart. Our findings do not support this conclusion. Rather, the near-unity relationship between first- and second-order deficits suggests impairment of either form-cue invariant mechanisms or an extensive extrastriate dorsal anomaly if different extrastriate areas are specialized for first- or second-order global motion processing.[21]

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