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A Reduced Motion Aftereffect in Strabismic Amblyopia

R. F. HESS,* R. DEMANINS,*‡ P. J. BEX†

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The motion aftereffect was measured using both static and dynamic test stimuli in a group of normal observers and a group of strabismic amblyopes. Amblyopes exhibited a reduced direct aftereffect for both static and dynamic stimuli and only two of the eight amblyopes exhibited any measurable interocular transfer for either test stimulus. It is hard to explain these results in terms of either the known spatial (contrast sensitivity and positional sensitivity) or motion deficits previously reported in amblyopia. These results suggest a primary motion deficit in amblyopia affecting both the static and dynamic motion aftereffects. © 1997 Elsevier Science Ltd. All rights reserved.

Amblyopia Motion aftereffect Adaptation Motion Static Dynamic

INTRODUCTION

Strabismic amblyopia is characterized by reduced contrast sensitivity and increased positional uncertainty. The site of the deficit is thought to be cortical and there is evidence to suggest anomalies in area V1. It is not known to what extent extra-striate areas are also affected. In this respect the current controversy over whether strabismic amblyopes exhibit anomalous motion perception is relevant. The processing of visual motion involves well defined extra-striate pathways (Maunsell & Newsome, 1987; DeYeo & Van Essen, 1988; Newsome & Pare, 1988; Zihl *et al*., 1983) which could be implicated if motion processing was abnormal in amblyopia.

The results from previous psychophysical studies are conflicting. Some studies argue for a selective impairment of motion processing (Tychsen & Lisberger, 1986; Schor & Levi, 1980a,b; Woods & Kulikowski, 1978; Norcia *et al*., 1991; Donahue & Wall, 1994; Kommerell *et al*., 1995; Graemiger *et al*., 1995), others argue for a selective sparing (Hess *et al*., 1978b; Levi *et al*., 1984; Hess & Anderson, 1993; Kubova *et al*., 1996) and yet others argue that form and motion processing are equally affected (Rentschler *et al*., 1981; Steinman *et al*., 1987; Banton & Levi, 1991).

One of the main early pieces of evidence that motion is a primary attribute in primate vision comes from the finding that prolonged viewing of a moving stimulus results in a percept of illusory motion for subsequent viewing of a stationary pattern [see Wade (1994) for review]. The site of this phenomenon is thought to be cortical and possibly extra-striate (e.g. Tootell *et al*., 1995). A recent distinction has been drawn between motion aftereffects (MAEs) measured using "static" and "dynamic" stimuli (Hiris & Blake, 1992). For example it is claimed that a MAE is not measurable with a non-Fourier adapting stimulus if tested with a static stimulus (Anstis, 1980; Derrington & Badcock, 1985; Nishida *et al*., 1994) but is measurable if tested with a dynamic (flickering) test stimulus (McCarthy, 1993; Ledgeway, 1994; Nishida *et al*., 1994). This difference has led to the proposal that the MAEs registered with these two types of test stimuli (static and dynamic) originate at different sites along the motion processing pathway. Nishida and Sato (1995) suggested V1 as a possible candidate for the site of the static MAE and MT or MST as the candidate site for the dynamic MAE. Further support for this dichotomy came from the work of Ashida and Osaka (1994, 1995) in their investigation of the spatial and temporal dependencies of the aftereffect measured with static and dynamic stimuli although this has recently been questioned (Bex *et al*., 1996).

One way of addressing whether there is a deficit to motion processing in amblyopia is to assess whether strabismic amblyopes experience a normal MAE. Although there have been studies on the MAE in amblyopia they have previously been directed towards the degree of interocular transfer [e.g. among others, Mitchell *et al*. (1975); Keck & Price (1982); O'Shea *et al*. (1994)] and not whether the direct effect is normal.

*McGill Vision Research Unit (H4–14), 687 Pine Avenue West, Department of Ophthalmology, McGill University, Montreal, Quebec, Canada H3A 1A1.

†Center for Visual Science, University of Rochester, 274 Meliora Hall, Rochester, NY 14627, U.S.A.

‡To whom all correspondence should be addressed.

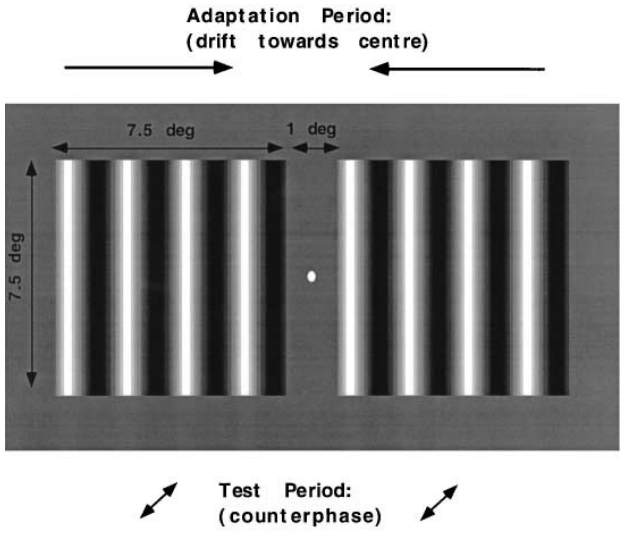

FIGURE 1. Spatial configuration of display.

Ideally, for the reasons outlined above, the MAE should be separately investigated for static and dynamic test stimuli. Here we report such an investigation.

The results suggest that strabismic amblyopes exhibit a reduced MAE for both static and dynamic stimuli which cannot be accounted for on the basis of what we already know of their contrast sensitivity and positional deficits. This suggests a primary deficit to motion processing, one which does not seem to simply follow from previously suggested motion deficits in amblyopia.

METHODS

Apparatus and stimuli

Stimuli were generated using a VSG 2/1 graphics card (Cambridge Research Systems) in a host PC microcomputer (DELL 333D) and were presented on a Nanao Flexscan 6500 monitor with P4 phosphor and with a frame rate of 118 Hz. The mean luminance of the display was 32 cd/m^2. The luminance of the display was linearized using an ISR attenuator (Pelli & Zhang, 1991) and calibrated using a UDT Photometer. The image was 16 deg horizontally (512 pixels) by 13.4 deg vertically (428 pixels) and was viewed from a distance of 118 cm in a dim room. The spatial layout of the display is shown schematically in Fig. 1. There were two square stimulus windows, each subtending 7.5 deg $\times$ 7.5 deg. The windows were separated horizontally by a 1 deg strip of mean luminance, in the centre of which was a prominent fixation point. The remainder of the display was blank and at the mean luminance.

Adapting and test stimuli were vertical, 1 c/deg sinusoidal gratings of 50% peak Michelson contrast, which were presented in the stimulus windows. The adapting gratings drifted towards the fixation point at a temporal frequency of 2 Hz. The test grating was either static or was sinusoidally counterphasing at a temporal frequency of 1 Hz. The starting phase of all gratings was randomized before each presentation. These conditions were chosen because they have been shown to elicit robust MAEs (Bex *et al.*, 1996). The adapting and test gratings were viewed by either both eyes, by the amblyopic eye (AE) or by the fixing eye (FE).

Procedure

The subject was instructed to maintain steady fixation during adapting and testing phases and initiated each trial with the press of a mouse button. This was followed by a 20 sec adaptation period during which the adapting stimulus was observed with the adapting eye(s). The adapting grating always drifted towards the centre of the screen to facilitate steady fixation. The adaptation period was immediately followed by a brief tone and the test period. During the test period, the test grating was presented in both windows and was either static or counterphasing. The subject maintained steady fixation with the test eye(s) and was required to press a mouse button when the MAE had finished. If the subject did not experience a MAE, the duration was recorded as zero seconds. Subjects practiced the task many times before formal data collection. The direction of the MAE was always seen in the opposite direction to that of the adapting grating (it always appeared to move away from the fixation point) and it was not necessary to record the perceived direction of MAE.

Each trial was followed by an inter-trial recovery interval of not less than 1 min. The whole procedure was repeated for each of the combinations: adapting eye(s) and test eye(s). The presentation sequence for the various conditions was randomized. The mean and standard errors of at least four estimates of MAE duration for each condition were recorded.

Control experiments

One-dimensional spatial noise, consisting of a random one-dimensional noise pattern moving, rather than a sinusoid, was used to ascertain the effect of using a spatially broadband stimulus on MAE durations in amblyopia. To determine whether our results were due to previously reported naso-temporal asymmetries in strabismic amblyopia, we changed the orientation of the display screen by 90 deg, such that we could compare the results for vertical as opposed to horizontal motion.

Subject details

Five normal subjects were used. All were experienced psychophysical observers and with the exception of subject EF none had extensive experience with MAEs. All normal observers had right eye sighting dominance. Eight strabismic amblyopes, four of whom were mixed (strabismic and anisometropic amblyopes) were also tested. Their individual clinical details are given in Table 1. All were experienced psychophysical observers although none had extensive experience with the MAE. Most subjects were tested on more than one occasion.

RESULTS AND DISCUSSION

Figure 2 shows results for the direct effect for both static and dynamic test stimuli for a group of normals and

TABLE 1

Subject	Age	Sex	Eye	Refraction	Letter acuity	Grating acuity (c/deg)	Fixation	Ocular alignment	Stereo acuity (sec)	History
OA strab/aniso	18	M	RE	$-4.50/-5.00\times030$	6/24	9	3 deg nasal	5 deg RET	Gross	Diagnosed age 3 yr, Rx age 3 yr, patching age 3 yr, no surgery
			LE	$-1.75/-1.75\times150$	6/6		Centred			
CT strab/aniso	40	F	RE	Plano	6/6		Centred	5 deg LET	Nil	LET aged 2 yr, Rx age 6–25 yr, patching at 2 yr, no surgery
			LE	$+3.25/-3.25\times180$	6/60	15	3 deg nasal			
MonS strab	24	F	RE	+0.75 DS	6/6		Centred	10 deg LET	Nil	Amblyopia age 9 yr, Rx age 9–20 yr, no patching, no surgery
			LE	+1.00 DS	6/18	24	1 deg nasal/inf.			
MS strab	27	M	RE	−1.25 DS	6/5		Centred	10 deg LET	Nil	Surgery at age 8 yr
			LE	Plano	6/9	36	0.5–1 deg nasal, unsteady	1 deg RHT		
SB strab	50	F	RE	Plano	6/6		Centred	2 deg LET	Gross	Microtropia diagnosed age 4 yr, no treatment
			LE	+0.50 DS	6/60	18	2 deg nasal			
VE strab/aniso	65	M′	RE	+0.75 DS	6/6+2		Centred	6 deg LXT	Gross	LXT diagnosed age 7 yr, Rx since age 7 yr, no patching, no surgery
			LE	+3.00 DS	6/24	11	Centred			
MarS strab/aniso	21	F	RE	+7.00 DS	6/6		Centred	L.esoflick on unilateral CT, 8 deg exophoria on alternate CT	800	Accommodative/anisometropic esotropia (microtropia) diagnosed at 1 yr. Rx since then but worn full-time only since age 5 yr. Patching <1 month at 1 yr, no surgery
			LE	+9.25 DS	6/15	24	Centred			
CC strab	24	M	RE	+1.00 DS	6/12	21.6	Centred	6 deg RET	Nil	RET diagnosed at age 3 yr, patching and visual training age 5 yr for 1 yr, first Rx age 5 yr, no surgery
			LE	+1.00 DS	6/4.5		Centred			

LET, left esotropia; RET, right esotropia; LXT, left exotropia; Rx, eyewear prescription; RHT, right hypertropia; CT, cover test.

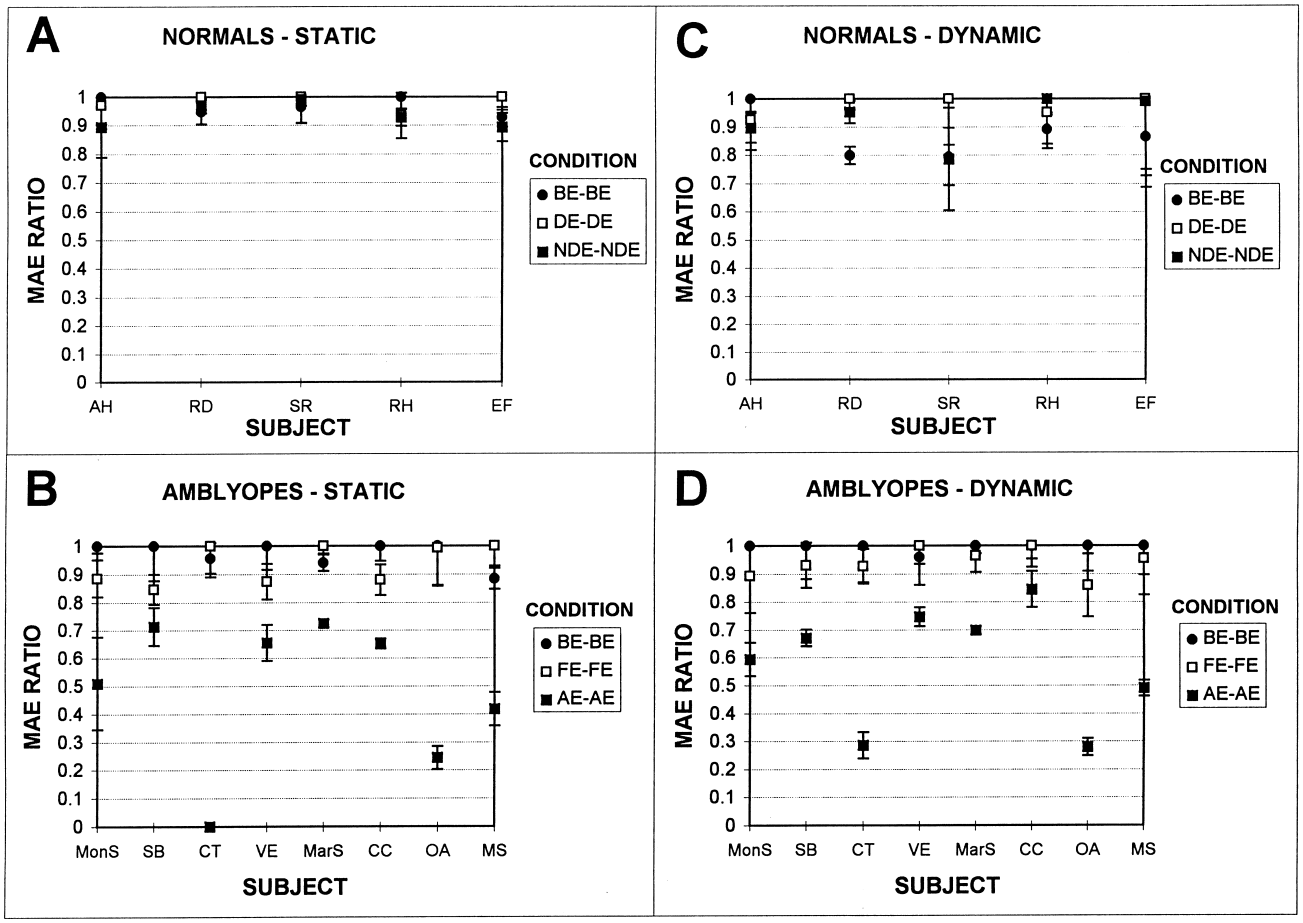

FIGURE 2. Comparison of the static (A and B) and dynamic (C and D) motion aftereffects for a group of normal observers (A and C) and a group of strabismic amblyopes (B and D). For the normal observers, responses are compared for three different viewing conditions [adapting eye(s)–test eye(s)]: both eyes (BE–BE), dominant sighting eye (DE–DE) and the other non-dominant eye (NDE–NDE). For the amblyopes, responses are compared for both eyes (BE–BE), the fixing eye (FE–FE) and the fellow amblyopic eye (AE–AE). The ratio is obtained for each subject by normalizing all their individual mean durations for a given viewing condition by the viewing condition giving rise to the maximum mean duration obtained for that subject. The normalization constant and the viewing condition giving rise to this value are given in Table 2 for each subject. The error bars represent the SEM for this ratio and are at times smaller than the symbol sizes. Strabismic amblyopes display a reduced duration of motion aftereffect for both static and dynamic conditions.

for the fixing and fellow amblyopic eyes of eight strabismic amblyopes. For each graph, results are shown for each eye separately (dominant, sighting eye, DE; non-dominant eye, NDE) and for both eyes (BE) together. Since the threshold criterion for abolition of the MAE differs across subjects, we normalized each subject's response separately for static and dynamic test conditions. The normalization is done separately for each subject across the viewing conditions for that subject and separately for the static and dynamic test conditions. The normalization values (i.e. the maximum MAE duration and the viewing condition that gave rise to this value) for each subject in the static and dynamic conditions are given in Tables 2 and 3. The results are plotted in terms of a ratio which is obtained by normalizing the durations to the maximum obtained for that subject. For normals, the MAE ratio for static test stimuli is between 0.9 and 1.0 [Fig. 2(A)]. Dynamic test stimuli produce results around 0.8–1.0 [Fig. 2(B)]. The fellow FE of strabismic amblyopes [open symbols in Fig. 2(B and D)] fall within this normal range for both static and dynamic test conditions. For the static test, with the exception of amblyope SB, all amblyopic eyes exhibit significantly reduced MAEs [Fig. 2(B) filled squares; $P < 0.05$, one-tailed *t*-test]. For the dynamic test, with the exception of amblyope CC, all amblyopic eyes exhibit significantly reduced MAEs [Fig. 2(D) filled squares; $P < 0.05$, one-tailed *t*-test].

Results for the interocular transfer of the MAE for both static and dynamic test stimuli are shown in Fig. 3. All normal subjects exhibited significant interocular transfer for both static and dynamic test conditions although the degree of transfer was slightly larger in the static case. The claim that the interocular transfer for dynamic stimuli is 100% [Nishida *et al*. (1994); also see Raymond (1993)] was not replicated here under our stimulus conditions. On the other hand, the majority of amblyopes exhibited no transfer in either direction (FE to AE; AE to FE) regardless of the type of test stimulus used (static or dynamic). The exception was amblyope OA whose

TABLE 2. Normalization constants for amblyopic and normal subjects; horizontal motion

	Normalization constant–static MAE (sec)	Viewing condition	Normalization constant–dynamic MAE (sec)	Viewing condition
Amblyopic subject				
MonS	26.4	BE–BE	12.1	BE–BE
SB	18.5	BE–BE	12.8	BE–BE
CT	22.4	FE–FE	16.6	BE–BE
VE	16.8	BE–BE	11.0	FE–FE
MarS	25.0	FE–FE	19.7	BE–BE
CC	18.8	BE–BE	13.3	FE–FE
OA	20.0	BE–BE	19.6	BE–BE
MS	36.3	FE–FE	20.9	BE–BE
Normal subject				
AH	22.7	BE–BE	20.0	BE–BE
RD	21.7	DE–DE	22.2	DE–DE
SR	22.0	DE–DE	19.2	DE–DE
RH	16.6	BE–BE	13.3	DE–DE
EF	31.9	DE–DE	8.6	DE–DE

Maximum MAE durations obtained and the viewing condition (adapting eye–test eye) giving rise to this value for each subject. These values were used as the normalization constant in obtaining the MAE ratio for each subject separately in Figs 2–4.

transfer was significantly reduced compared with normal observers ($P < 0.01$, one-tailed *t*-test) for static test stimuli. Only CT and OA exhibited transfer for dynamic test stimuli, both of which were significantly reduced from the average normal result ($P < 0.05$, one-tailed *t*-test). There was no strong correlation between the subjects who exhibited transfer and those with residual stereoacuity (see Table 1).

Before one jumps to the conclusion that a reduced MAE in amblyopia necessarily implicates anomalous motion processing it is first prudent to consider other possible explanations. *First, could it be a consequence of the stimuli being reduced in their visibility owing to the known contrast sensitivity deficit?* This seems an unlikely explanation because the duration of the aftereffect is unaffected when the contrast of test and adapting stimuli is reduced by a factor of four (50% to 12.5%), so long as the relative contrast between adapting and test stimuli does not change (Bex and Mareschal, unpublished data). Since the test and adapting stimuli are spatial frequency narrowband and of the same spatial frequency, their visibility would be affected equally by any contrast sensitivity deficit in amblyopia. Furthermore, prior to testing we had ensured that the spatial frequency of the stimulus (1 c/deg) was well within the amblyopic passband (see grating acuities in Table 1). Consistent with this, the correlation between their grating acuity and the magnitude of the MAE deficit is weak ($r = 0.20$ for static and $r = 0.16$ for dynamic).

Could it be that the spatial scrambling that has been postulated in amblyopia (Hess *et al.*, 1978a; Hess & Field, 1993) *is responsible for the reduced duration of the MAE?* It is difficult to control for this since we do not at present have a quantitative model for the proposed spatial disarray. What we can say is that all subjects reported that they unambiguously perceived the motion and direction of the adapting stimuli. It is possible that as a consequence of their spatial disorder the sinewave appeared more spatially noisy to the amblyopic visual system, however, this is unlikely to have reduced the duration of the aftereffect. We verified this in two amblyopes (OA and MS) by adapting and testing with

TABLE 3. Normalization constants for amblyopic and normal subjects; vertical motion

	Normalization constant–static MAE (sec)	Viewing condition	Normalization constant–dynamic MAE (sec)	Viewing condition
Amblyopic subject				
OA	20.0	BE–BE	19.6	BE–BE
MS	36.7	BE–BE	16.7	FE–FE
SB	14.0	FE–FE	9.4	FE–FE
Normal subject				
RD	25.2	DE–DE	20.9	DE–DE

Maximum mean of the MAE durations (four trials) obtained for each subject and the viewing condition (adapting eye–test eye) giving rise to this value. These values were used as the normalization constant in obtaining the MAE ratio for each subject separately in Fig. 5(B and D). Normalization constants for horizontal motion [Fig. 5(A and C)] can be found in Table 2.

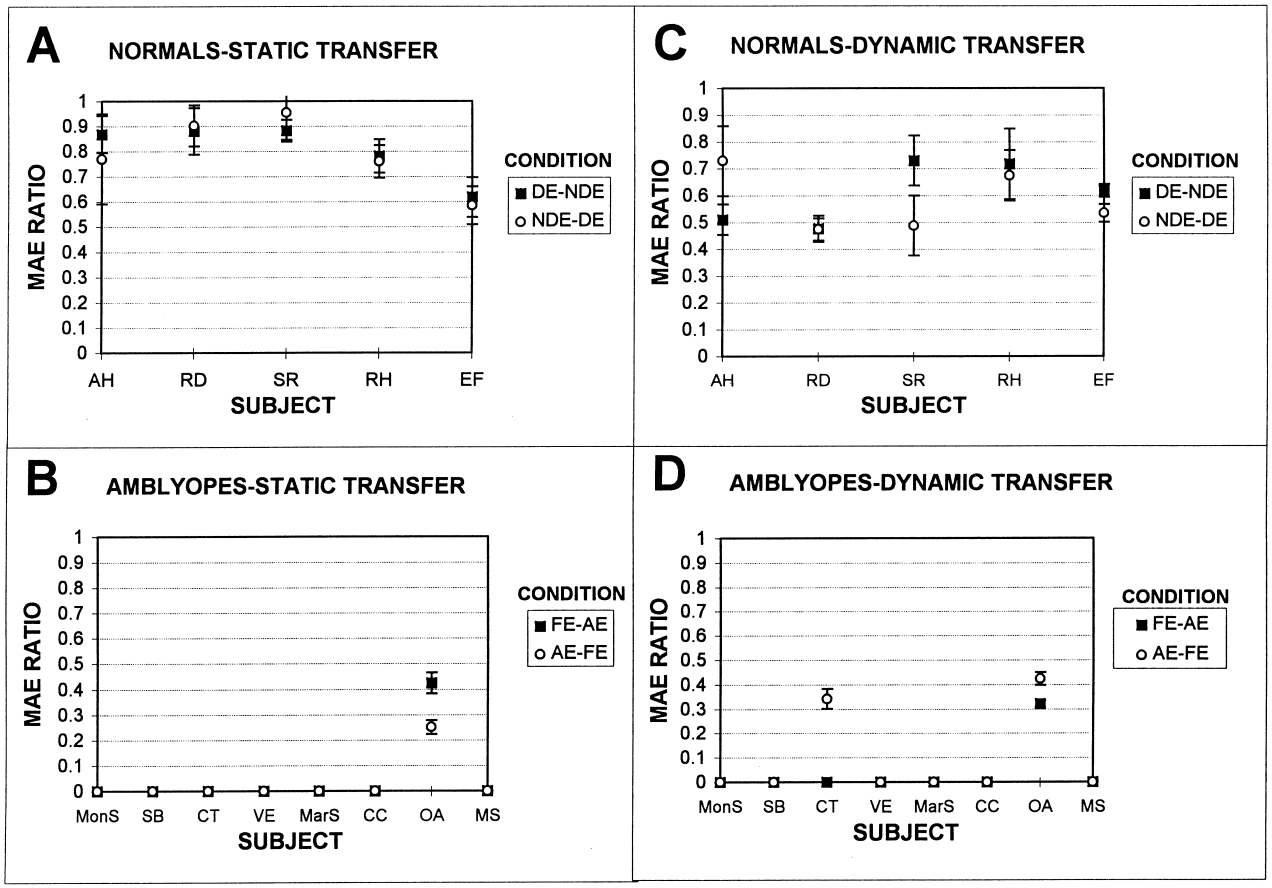

FIGURE 3. Comparison of the interocular transfer of the static (A and B) and dynamic (C and D) motion aftereffects for a group of normal observers (A and C) and a group of strabismic amblyopes (B and D). For the normal observers, responses are compared for two different viewing conditions: transfer from the sighting DE, to the NDE (DE–NDE) and transfer in the other direction (NDE–DE). For the amblyopic observers, responses are compared for two different viewing conditions: transfer from the FE to the AE (FE–AE) and transfer in the other direction (AE–FE). The ratio is obtained for each subject by normalizing the mean duration for the transfer viewing conditions (e.g. in the case of normals DE–NDE and NDE–DE) by the maximum duration obtained for that subject. These normalization constants and the viewing condition giving rise to this value are given for each subject in Table 2. The error bars represent the SEM for this ratio and are at times smaller than the symbol sizes. Normals exhibit less transfer for the dynamic test whereas amblyopes exhibit reduced or zero transfer (data on abscissa) for both static and dynamic tests.

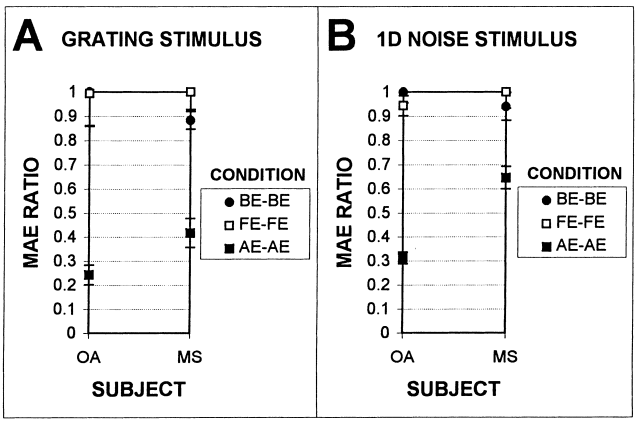

FIGURE 4. The static motion aftereffect is compared for a sinewave spatial stimulus and a one-dimensional noise spatial stimulus for two amblyopic subjects (OA and MS). Results are shown for BEs (BE–BE), the FE (FE–FE) and the fellow AE (AE–AE). The normalization constants for the noise stimulus were 12.8 sec (OA; BE–BE) and 24.4 sec (MS; FE–FE). The MAE is significantly reduced for the AE compared with that of the FE for both subjects for both grating and noise stimuli.

one-dimensional spatial noise rather than a single sinusoid. We reasoned that any spatial scrambling within the amblyopic visual system would disrupt a spatially incoherent noise stimulus less than it would a spatially coherent sinewave. We found significantly reduced (one-tailed *t*-test; $P < 0.005$) MAE durations for the amblyopic eyes compared with that of the fellow FE of both subjects for noise stimuli in the static condition (Fig. 4). Consistent with this is that there is only a weak correlation between the positional deficits in these subjects measured with a three Gabor alignment task at a spatial scale a factor of two within their resolution limit (Hess & Holliday, 1992; Demanins & Hess, 1996) and the duration of the MAE ($r = 0.41$ for static and $r = 0.59$ for dynamic).

A number of previous studies have reported deficits to motion processing in amblyopia. While some of these have more to do with temporal threshold sensitivity (Woods & Kulikowski, 1978; Rentschler *et al*., 1981: Schor & Levi, 1980a) others (Tychsen & Lisberger, 1986; Steinman *et al*., 1987; Levi *et al*., 1984; Hess &

Anderson, 1993) can be more directly interpreted in terms of motion processing *per se* because either motion direction or motion speed was assessed. In all of these studies anomalous function was revealed in the high-spatial low-temporal frequency range. *Is the reduced MAE merely a consequence of this previously reported anomaly?* This seems unlikely for two reasons. First, the stimulus spatial frequency was well within the resolution limit for all amblyopes (see Table 1). For those with better acuity, the stimulus should be regarded as a mid–low spatial frequency. Second, previous reports involved either elevated thresholds for direction discrimination (Levi *et al*., 1984; Hess & Anderson, 1993), reduced perceived speed (Tychsen & Lisberger, 1986) or elevated thresholds for speed discrimination (Steinman *et al*., 1987), none of which would necessarily diminish the duration of the MAE for suprathreshold stimuli since there is only a very weak dependence of the aftereffect duration on the temporal frequency of the adapting stimulus (Bex *et al*., 1996). Furthermore, the perceived speed deficits in amblyopia (Tychsen & Lisberger, 1986; Steinman *et al*., 1987) while being clearly present are small (less than a factor of two) and unlikely to significantly reduce the duration of the MAE (Bex *et al*., 1996). However this presupposes that the MAEs' dependence on the temporal properties of the test stimulus are similar in normal eyes and amblyopic eyes.

Previous studies have demonstrated a naso-temporal asymmetry for pursuit eye-movements and judgement of target velocity [Tychsen & Lisberger (1986); Graemiger *et al*. (1995); but also see Steinman *et al*. (1987)]. Because of our stimulus arrangement, both nasal and temporal fields were tested simultaneously and it is therefore difficult to say how this affected our results. Some, though not all, subjects commented on a difference in the perceived velocity of the nasal and temporal segments in our adapting stimulus. *Could the duration of the aftereffect be reduced due to this previously reported naso-temporal asymmetry?* This seems unlikely because similar durations of MAE are obtained in normals with only half of the stimulus field visible (Hess and Demanins, unpublished). To verify that the MAE deficit was not restricted to horizontal motion we repeated measurements for three amblyopes for an

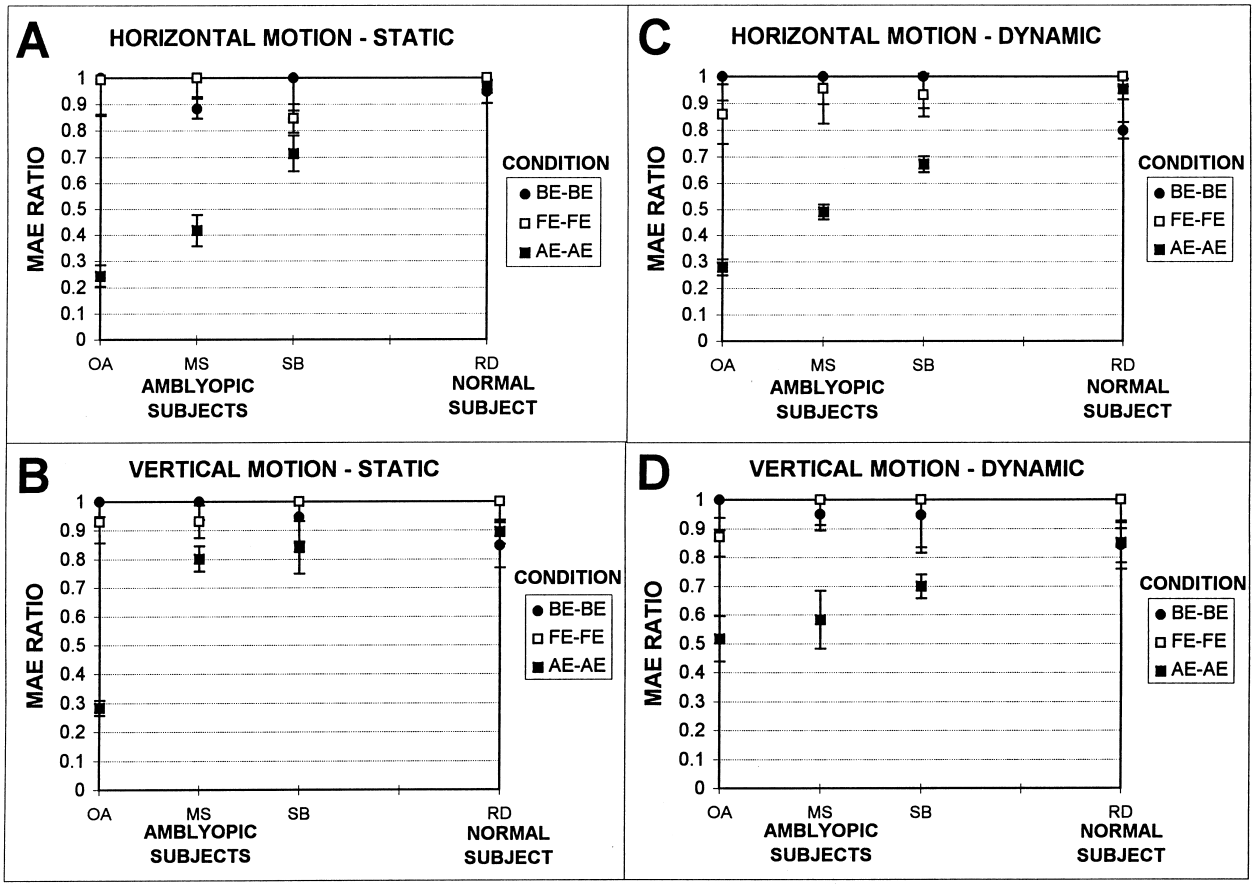

FIGURE 5. Comparison of the static (A and B) and dynamic (C and D) MAEs for a group of three amblyopic (OA, MS, and SB) and one normal observer (RD). The MAE is compared for horizontal motion (A and C) and for vertical motion (B and D). Responses are compared for each eye separately (FE–FE and AE–AE for the amblyopes or DE–DE and NDE–NDE for the normal observer) and for both eyes (BE–BE). The ratio is obtained for each subject by normalizing all means by the maximum duration obtained. The normalization constant is given in Table 3 for each subject. The error bars represent the SEM for this ratio and are at times smaller than the symbol sizes. Our previous conclusions for horizontal motion (A and C) are seen to hold for vertical motion (B and D). Amblyope SB did not exhibit a significantly reduced static aftereffect for either horizontal or vertical motion. She did exhibit a significantly reduced dynamic aftereffect for both horizontal and vertical motion.

otherwise identical stimulus arrangement involving only vertical motion. The normalization constant (i.e. the maximum MAE duration) obtained for each subject and the subject's viewing condition giving rise to this value are provided in Table 3. The results shown in Fig. 5 suggest that similar static and dynamic MAE deficits occur for vertical and horizontal motion. In one subject (MS), the deficit, though significant (one-tailed *t*-test; $P < 0.05$), was reduced for vertical motion.

Amblyopes are known to have more unstable fixation (Schor, 1973). *Could it be that unstable fixation* per se *reduces the duration of the aftereffect?* We verified that if fixation is made artificially unstable by asking normal observers to make continual, rapid eye movements within a 3 deg dia central zone during both adaptation and test phases, it did not affect the duration of either the static or the dynamic MAE. Therefore, we feel that this is not a satisfactory explanation.

Could it be that that the deficit is of a more general form and involves the mechanism of adaptation independent of modality? This is unlikely because we know that the strength, dynamics and properties of adaptation produced by at least some other modalities are normal in amblyopia (Hess, 1980).

We are left to conclude that the reduced MAEs reported here represent evidence, complementary to that of others (Tychsen & Lisberger, 1986: Steinman *et al*., 1987), of a deficit which involves the processing of visual motion in amblyopia. Based on differences in the properties of the static and dynamic MAEs (McCarthy, 1993; Ledgeway, 1994; Nishida *et al*., 1994; Ashida & Osaka, 1994; Ashida & Osaka, 1995; Nishida & Sato, 1995; Verstraten *et al*., 1996) it has been suggested that static and dynamic test stimuli reflect activity of either different aspects of motion processing or different sites of motion processing. The present results which demonstrate deficits for both static and dynamic stimuli suggest that the motion deficit in amblyopia affects both of these aspects or sites of motion processing.

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