The extra-retinal motion aftereffect

Tom C. A. Freeman School of Psychology, Cardiff University, Cardiff, UK

Jane H. Sumnall School of Psychology, Cardiff University, Cardiff, UK

Robert J. Snowden School of Psychology, Cardiff University, Cardiff, UK

Repetitive eye movements are known to produce motion aftereffect (MAE) when made to track a moving stimulus. Explanations typically centre on the retinal motion created in the peripheral visual field by the eye movement. This retinal motion is thought to induce perceived motion in the central test, either through the interaction between peripheral MAE and central target or by adaptation of mechanisms sensitive to the relative motion created between centre and surround. Less attention has been paid to possible extra-retinal contributions to MAE following eye movement. Prolonged eye movement leads to afternystagmus which must be suppressed in order to fixate the stationary test. Chaudhuri (1991, *Vision Research*, 131, 1639-1645) proposed that nystagmus-suppression gives rise to an extra-retinal motion signal that is incorrectly interpreted as movement of the target. Chaudhuri's demonstration of extra-retinal MAE depended on repeated pursuit to induce the aftereffect. Here we describe conditions for an extra-retinal MAE that follows more reflexive, nystagmus-like eye movement. The MAE is extra-retinal in origin because it occurs in part of the visual field that received no retinal motion stimulation during adaptation. In an explicit test of the nystagmus-suppression hypothesis, we find extra-retinal MAE fails to store over a 30s delay between adaptation and test. Implications for our understanding of motion aftereffects are discussed.

Keywords: eye movement, afternystagmus, motion aftereffect, nystagmus-suppression, extra-retinal

Introduction

The motion aftereffect (MAE) has a long and venerable history within vision science. Recent reviews show that our understanding of the mechanisms contributing to MAE is quite sophisticated (Anstis, Verstraten & Mather, 1998; Clifford, 2002; Mather, Verstraten & Anstis, 1998). They also emphasise the view that the critical stimulus is motion on the retina. However, many of the classic stimuli that give rise to MAE, including waterfalls, rivers, cavalries and gratings, can produce tracking eye movements whose primary function is to stabilise objects in the image. Such eye movements bring about wholesale changes to the distribution of velocities across the retina. For instance, accurate eye movement renders the image of a waterfall more or less stationary for the majority of the adaptation period. Yet observers are known to experience compelling MAE when allowed to pursue part of the adapting pattern with their eyes (Anstis & Gregory, 1965; Chaudhuri, 1990; Chaudhuri, 1991a; Chaudhuri, 1991b; Mack, Goodwin, Thordarsen, Benjamin, Palumbo & Hill, 1987; Mack, Hill & Kahn, 1989; Morgan, Ward & Brusell, 1976; Swanston & Wade, 1992). This type of eye-movement MAE cannot be due to the retinal motion of the target being pursued because the retinal slip present in that part of the visual field is either too small or in the wrong direction (Morgan et al., 1976). Alternatively, the presence of stationary landmarks adjacent to the pursuit target may explain the effect because they move across the retina during eye movement and so create their own MAEs in the peripheral visual field. Some authors believe these peripheral MAEs can induce motion in a central test (Mack et al., 1987; Morgan et al., 1976), whereas others believe that eye-movement MAE results from the relative motion between centre and surround (Swanston & Wade, 1992). This cannot be the whole story, however, because MAE following eye movement can occur in the absence of peripheral retinal stimulation (Chaudhuri, 1990; Chaudhuri, 1991a; Chaudhuri, 1991b). This suggests a wholly separate extra-retinal contribution to eye-movement MAE. Here we examine this little-discussed contribution to MAE in more detail, asking in particular whether what we shall call *extra-retinal* MAE can be induced by the more reflexive eye movement typically associated with prolonged periods of repetitive tracking.

In the key condition of Chaudhuri's experiments, observers pursued a moving target displayed in complete darkness, thus removing any influence of peripheral retinal MAE on the subsequently viewed test (a small dot). After adaptation observers reported MAE in the opposite direction to the previous tracking eye movement. While this provides strong support for an extra-retinal component to MAE, the precise nature of the extra-retinal signal may well depend on the type of eye movement made during adaptation. Eye movements can be divided into two broad classes. One corresponds to a

doi:10.1167/3.11.11 Received April 29, 2003; published December 5, 2003

more reflexive response and is typically associated with image stabilisation during self-motion (e.g. optokinetic nystagmus, vestibulo-ocular reflex). The other corresponds to a more intentional, pursuit-like following response and is typically made to fixate small targets moving with respect to the observer. Different control mechanisms are known to accompany reflexive and intentional eye movement, such as the visual processes estimating target motion (e.g. Beutter & Stone, 2000; Harris & Smith, 1992, 2000; Ilg, 1997; Pola & Wyatt, 1985) and the predictive mechanism that accompanies pursuit (e.g. Kowler, 1989; Krauzlis & Adler, 2001). It is therefore not immediately clear whether the aftereffect Chaudhuri discovered is peculiar to prolonged intentional tracking or whether it extends to more reflexive, nystagmus-like eye movements.

Indeed, to explain the extra-retinal MAE, Chaudhuri suggested a mechanism more commonly associated with reflexive eye movement. As is well documented, the eyes continue to move following repetitive eye movement so long as the stimulus is removed and the observer left in complete darkness (Clement & Lathan, 1991; Gizzi, Raphan, Rudolph & Cohen, 1994; Muratore & Zee, 1979; Schor & Westall, 1986). Called *afternystagmus*, the residual eye movement is thought to result from a slowly-dissipating velocity-storage mechanism that aids image stabilisation when eye and surround move relative to one another (Cohen, Matsuo & Raphan, 1977). According to Chaudhuri's argument an observer must inhibit afternystagmus in order to fixate the stationary test. One possible by-product of *nystagmus-suppression* is generation of an extra-retinal motion signal that gives rise to MAE. The idea is not without precedent as a number of motion phenomena are thought to result from nystagmus suppression in its various forms (e.g. oculogyral illusion, Evanoff & Lackner, 1987; see Sumnall, Freeman & Snowden, in press, for exceptions). Chaudhuri provided direct support for the nystagmus-suppression hypothesis in the form of the close correlation between the respective time-course of afternystagmus and extra-retinal MAE. However, some care needs to be taken with this finding because real-time velocity nulling was used to measure the time-course of the aftereffect, using a single dot as test (Chaudhuri, 1991b). The physical motion of the dot almost certainly gave rise to pursuit-like eye movements, so it is not entirely clear what motion percept is being nulled, nor how eye movement, retinal slip and aftereffect interacted during test.

We investigated the possibility that optokinetic nystagmus (OKN) results in extra-retinal MAE by presenting prolonged large-field motion to our observers and examining adaptation conditions with and without eye-movement. The stationary test stimulus was presented in part of the visual field that had not previously received retinal motion stimulation. We confirmed the reflexive nature of the eye movement by analysing the distributions of fast-phase frequencies (Cheng & Outerbridge, 1974; Crognale & Schor, 1996; Schor & Narayan, 1981). We also examined whether extra-retinal MAE exhibits 'storage'. This refers to the remarkable finding that classic retinal MAE persists if one delays the time between adaptation and test beyond the aftereffect duration reported with no delay (e.g. Thompson & Wright, 1994). The nystagmus-suppression hypothesis predicts that extra-retinal MAE should not store, simply because afternystagmus eye movements fade in the dark and so should cease to exist at test if the delay is made long enough.

Methods

All stimuli were created using small, dim dots on a black background displayed on a large screen via a VSG 2/3 graphics board (see Movie 1 for an example). Adaptation stimuli consisted of a vertically moving, striped stimulus depicting two strips of horizontal lines measuring 20° by 72° and separated by 10°. A line consisted of a horizontal row of dots at 2° intervals, with each line separated vertically by 4°. In eye-movement conditions, the stimulus moved at 12°/s and observers were asked to keep their eyes located in the central black area. To elicit reflexive eye movement, observers were asked to stare straight ahead at the central blank but not inhibit any felt eye movement (this more reflexive type of eye movement is sometimes referred to as *stare-nystagmus*). In eye-stationary conditions, a small, central fixation point was provided and the stimulus made to move at 3°/s. The decrease in speed created eye-moving and eye-stationary conditions that were approximately equal in the degree of retinal motion stimulating the periphery during adaptation (see below for further discussion). A baseline condition was also run in the first two experiments, in which the striped pattern was made to hop vertically with random displacements at a frequency of 0.6 Hz. Adaptation lasted for 60s and was preceded by 40s of a bright, homogeneous field followed by 20s of darkness. The bright field helped maintain light adaptation, making any extraneous visual references less visible to the observer.

The test pattern for most experiments consisted of a single dot presented in isolation at the centre of the black screen. In principle, the dot coincided with an area of the visual field that had not received previous retinal-motion stimulation. In practice, the degree of motion stimulation at this location depended on the ability of observers to maintain gaze on the central blank area during adaptation. Eye movement recordings showed that by and large they were able to do so (see Movie 1 and Figure 2). Following adaptation, observers were asked to judge either MAE duration or direction depending on the experiment.

Eye movements were recorded using a head-mounted, video-based eye-tracker (ASL Series 4000). The visible red

LED of this equipment prevented us from recording afternystagmus because it served as a legitimate target with which to 'dump' the post-nystagmus eye movement (cf. Chung & Bedell, (1995); as yet we have been unable to filter out the light-source successfully). Eye movements were sampled at 50 Hz and analysed offline using custom software written in MatLab. Position records were low-pass filtered and then velocity and acceleration profiles determined from first and second time derivatives. Three aspects of eye movement were examined. The horizontal component was used to determine an observer's ability to maintain fixation on the central area. The vertical component was used to determine both gain and duration of ocular following during OKN slow phase. To do this, eye movement recordings were first segmented into the slow and fast phases by locating fast-phase peaks (i.e. saccades). These were defined as zero-crossings in the acceleration profile, coincident with eye speed exceeding a fixed velocity threshold. Samples 110ms either side of the peak were excluded from further analysis, as were the first three slow phases and the one just before test. The mean of the remaining samples was used to determine slow-phase gain. The distribution of slow-phase durations was examined to assess the degree of reflexive eye movement exhibited by observers during adaptation (see Results section for details). Performance of the algorithm was closely monitored by visual inspection of each recording.

Results

Figure 1 depicts sample eye movement traces for upward and downward eye-moving conditions, with slow-phase components (as defined by our algorithm) highlighted in black. Both waveforms resemble the classic saw-tooth profile of optokinetic nystagmus. Movie 1 lends support to this interpretation by showing a portion of the eye movement recordings superimposed on the adaptation stimulus. The traces in Figure 1 also show that any gross eye movement after adaptation was confined to the initial fixation of the test dot. We saw little evidence of afternystagmus during this phase – in most cases eye movement in this period was similar to the eye tremor accompanying fixation of the test in the baseline condition. Perceiving consistent motion of the test could not therefore be the result of the target moving on the retina.

Analysis of the horizontal eye movements confirmed observers were able to keep their gaze within the central blank area during ocular following. Figure 2 shows summary histograms of all horizontal-position samples recorded during vertical slow-phases. Foveation of the peripheral strips was infrequent, with 43% of observers in the upward eye-moving condition and 57% in the downward eye-moving condition never doing so at all. Of the remaining observers, only 17% and 12% of the samples collected in the two respective conditions wandered into the prohibited area. This means that the fovea of around half of the observers did not receive any retinal-motion stimulation during eye-movement adaptation, an impression that can be gleaned from Movie 1. For the remaining observers, only a small proportion of their time was spent fixating the moving strips. Of course, the analysis does not tell us how accurately these 'rule-breakers' fixated the strips and so gives no indication of the effective retinal slip the fovea received during adaptation. Movie 1, for instance, shows one clear rule-breaker (green symbol) fixating the inner edge of the right-hand strip. However, as this observer was able to follow the motion quite well, the degree of retinal motion stimulating this individual's fovea was arguably

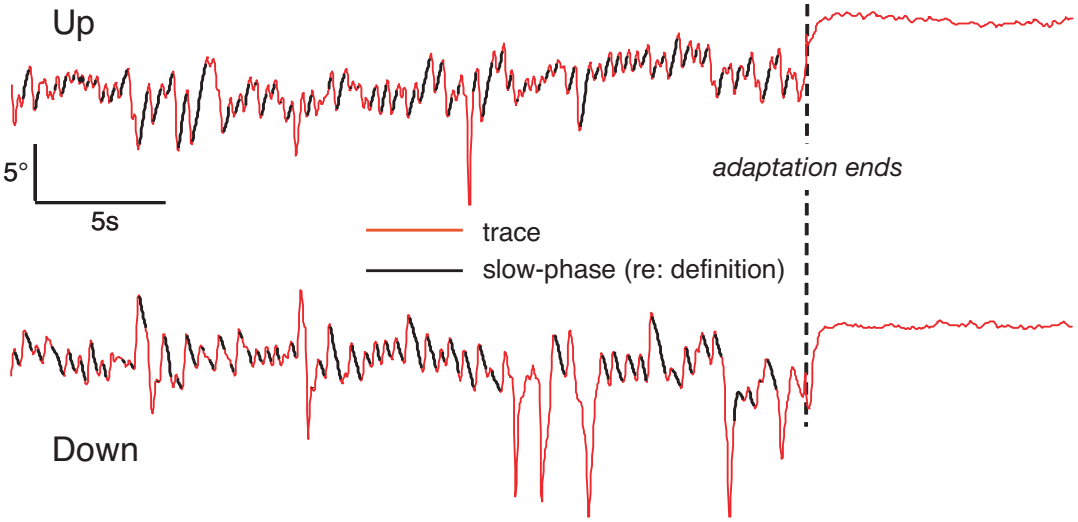


Figure 1: Sample vertical eye movements of one observer for a portion of the adaptation period in the two eye-moving conditions. The red is the original trace and the black segments indicate slow-phases identified by the algorithm described in the text. Broken vertical line shows the end of adaptation.

low.

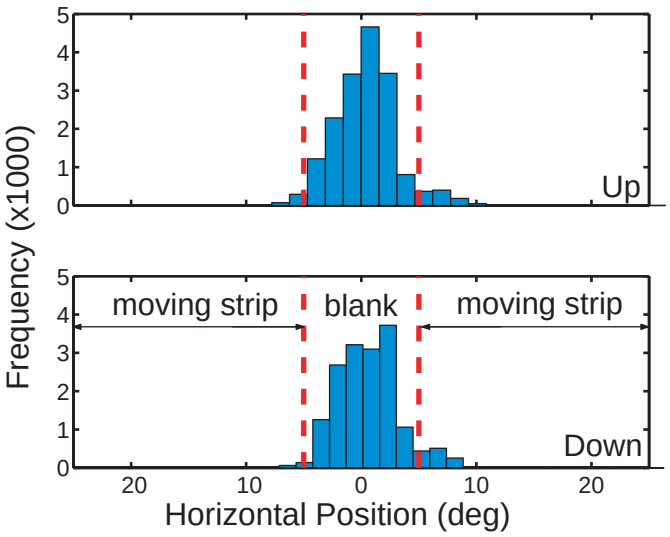


Figure 2: Histogram of horizontal positions sampled during slow-phase of eye movement. Dashed vertical lines indicate location of central blank region, with horizontal axis scaled to the overall width of adaptation stimulus.

Most observers reported compelling motion of the stationary test dot at the end of 60s of vertical eye movement. The direction reported was opposite to the stimulus motion shown in the adaptation period, such that a nystagmus eye movement with upward slow-phase produced a downward MAE. Like Chaudhuri (1991b), most observers also reported that upward eye movement gave rise to stronger MAE than downward eye movement. This was reflected in the duration data of the eye-moving condition (labeled OKN in Figure 3) and may result from the increased slow-phase gain found for upward eye movement (0.80) compared to downward (0.61), an asymmetry that has been reported previously (van den Berg & Collewijn, 1988). The middle pair of bars shows duration data for upward and downward eye-stationary conditions, in which a stationary fixation point was displayed at the centre of the blank area. To reiterate, the speed of the adapting pattern was set to 25% the speed in the eye-moving conditions, in an attempt to equate the average retinal-motion stimulation overall. As it turned out, this was a reasonably good guess at the average retinal slip present in the eye-moving conditions (mean slow-phase gain of ~0.7 yields an average retinal slip that is ~30% of the stimulus speed i.e. 3.6°/s). The key difference between eye-moving and eye-stationary conditions was therefore the presence of an ocular-following response as opposed to any gross change in the degree of retinal motion stimulation (eye movements in the eye-stationary condition were negligible).

The extra-retinal nature of the MAE our observers reported is supported by the analysis of the duration data, which showed a significant difference between eye-moving and eye-stationary conditions. However, the eye-stationary

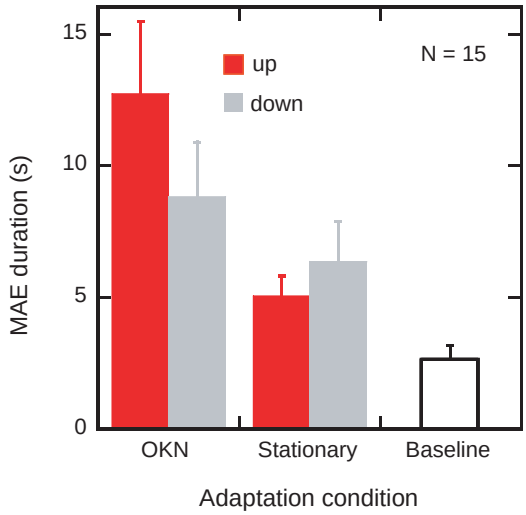


Figure 3: MAE duration for eye-moving (OKN) adaptation, eye-stationary and jitter baseline. Stimulus speed was 12°/s for OKN conditions and 3°/s for eye-stationary. Red bars correspond to upward slow-phase eye movement or upward retinal motion. Grey bars correspond to downward. ANOVA showed significant effect of eye movement ($F(1,14) = 5.88$, $p = .03$) but not direction ($F < 1$) or the interaction ($F(1,14) = 3.67$, $p = .08$). Eye-stationary MAE durations were significantly different from baseline (upward: $t(14) = 3.22$, $p = .006$; downward: $t(14) = 2.76$, $p = .015$). Error bars are ±1 SE.

data also revealed a small but significant MAE compared to baseline (see legend of Figure 3 for relevant statistics). This is surprising, not only because measured eye movements were negligible in the eye-stationary conditions but also because the retinal motion was peripheral to the test location during adaptation. One might be tempted to cite this as evidence of motion induction by peripheral MAE. However, the test stimulus was devoid of peripheral landmarks deemed critical for the induction process to operate (Wade, Spillman & Swanston, 1996). Some observers reported the presence of a short-lived moving afterimage coincident with the strips' location (see Thompson, 1998, for discussion). It is possible that a form of 'phantom' induction may have occurred between afterimage and test dot. However, not all observers experienced MAE in the eye-stationary conditions and our impression was that its direction was not consistently reported for those that did. For this reason, we ran a separate experiment designed to increase the number of direction judgments made in each of the five conditions, thus gaining a clearer picture as to the consistency of MAE directions perceived.

A new set of observers were therefore asked to judge the direction of MAE for a test dot presented for 4s following 60s adaptation. Each of the five conditions described above were repeated 4 times per observer in random order. Observers were allowed to respond 'up', 'down' or 'none'. Figure 4 shows the percentage of 'up'

and 'down' responses made (for clarity the 'none' responses are not shown). In both eye-moving conditions MAE is seen on most occasions and is consistently reported in a direction opposite to the slow-phase of the preceding nystagmus. In the eye-stationary condition MAE was reported less than 50% of the time, with direction preference following upward retinal-motion adaptation especially erratic. This aside, the direction most commonly reported was *opposite* to the direction of the adapting motion and hence the wrong way round for any induction effect to be implicated (phantom or otherwise). One possibility is that the weak eye-stationary MAE results from adaptation of motion mechanisms with large receptive fields (Snowden & Milne, 1997). Another is that the effect is extra-retinal in origin because retinal motion can, under certain conditions, lead to afternystagmus with slow-phase in the same direction as the adapting motion (Schor & Westall, 1986). Unfortunately, within our current set-up we are unable to measure afternystagmus with any success and so cannot determine whether slow large-field motion gives rise to the appropriate post-adaptation eye movement.

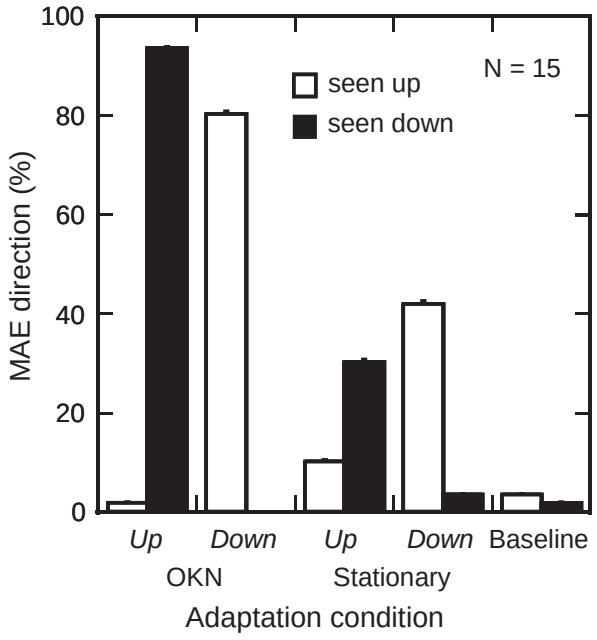


Figure 4: Direction judgments for the conditions described in Figure 3. White bars correspond to percent MAE seen to move up, dark bars percent MAE seen down. Error bars are ±1 SE.

One important feature of nystagmus is the distribution of slow-phase durations. This is thought to vary with the intention of the observer to follow the stimulus (Cheng & Outerbridge, 1974; Crognale & Schor, 1996; Schor & Narayan, 1981; van den Berg & Collewijn, 1988). Slow-phase duration is equal to the reciprocal of fast-phase frequency (though note for the algorithm employed here, the reciprocal relationship only holds once the assumed duration of a saccade is added back in). When using instructions thought to elicit more reflexive eye movement (*stare-nystagmus*), Cheng & Outerbridge (1974) found the distribution moved from approximately Gaussian to positively-skewed and multi-modal as stimulus speed lowered. The pattern did not depend on the type of stimulus used. The primary mode centred on 0.3s regardless of speed or stimulus type, a value which is similar to that found for vestibular nystagmus (Carpenter, 1988) and one that has been used as a way to characterise nystagmus-like eye movements as either reflexive or voluntary (e.g. Crognale & Schor, 1996). Conversely, when instructed to intentionally follow the stimulus (*look-nystagmus*), the distribution exhibited a far more extreme multi-modal shape resembling disparate 'islands' of eye movement activity. Again, Cheng & Outerbridge found the lower mode centred on 0.3s. Figure 5 shows histograms of slow-phase durations of all observers for the two eye-movement conditions in the duration experiment of Figure 3. Both distributions resemble those reported by Cheng & Outerbridge for stare-nystagmus at this stimulus speed (note that neither histogram contains slow-phase durations less than 220ms because saccades separated by less than this were automatically excluded by our algorithm). There is also some evidence of multi-modality, especially in the upward condition (top panel). In both cases the majority of the slow-phase durations were less than 1000ms, a criterion that has been used to segregate reflexive from intentional eye movement (Crognale & Schor, 1996; Schor & Narayan, 1981). We conclude the MAEs reported by our observers were induced primarily by reflexive eye movement.

Figure 6 shows the results of the final experiment, in which we examined the storage properties of extra-retinal MAE. Aftereffect duration for retinal and extra-retinal conditions was investigated following 0, 5 or 30s delay

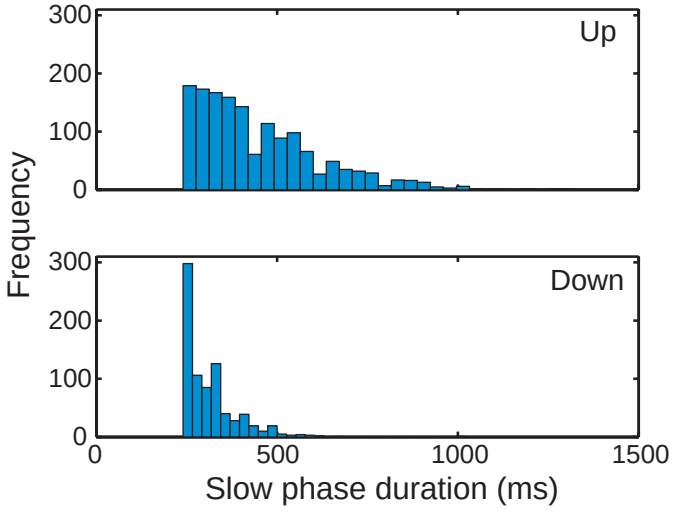


Figure 5: Distributions of slow-phase durations for observers contributing to Figure 3.

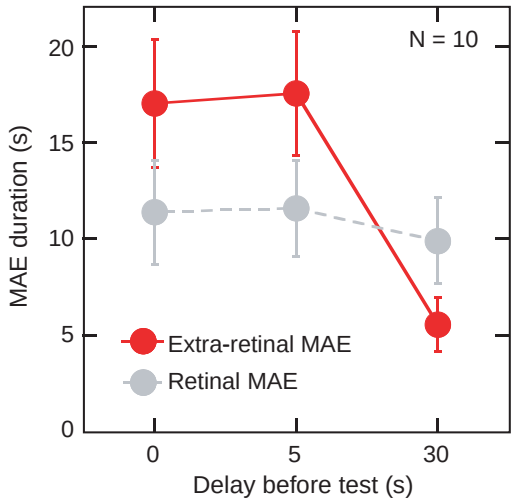


Figure 6: Storage characteristics of the extra-retinal MAE (solid lines) and retinal MAE (dotted lines). The stimulus moved upward with a stimulus speed of 12°/s (extra-retinal condition) and 3°/s (retinal condition). Test patterns consisted of a dot or last frame of adaptation, respectively. ANOVA showed significant effect of delay ($F(2,18) = 9.18$, $p = .002$) and interaction ($F(2,18) = 4.60$, $p = .024$) but not adaptation condition ($F < 1$). Error bars are ±1 SE.

between adaptation and test. During the delay observers remained in complete darkness. Retinal MAE was elicited using upward eye-stationary adaptation run at 3°/s but this time followed by the final frame of the display. We did not specify the type of MAE observers should base their judgment on in this condition, be it relative motion between dot and surround, movement of the surround, movement of the central dot or some combination. Extra-retinal MAE was elicited using upward eye-moving adaptation run at 12°/s, combined with stare-nystagmus instructions. The test pattern was a single dot. The duration data reveals a marked difference in the relationship between duration and delay for retinal and extra-retinal MAE. The retinal condition produced almost complete storage over 30s. However, the extra-retinal condition shows a steep decline in MAE duration between 5 and 30s. The extra-retinal MAE appears not to store over the same time period as the retinal MAE. The lack of storage provides support for the nystagmus-suppression hypothesis. The extra-retinal MAE disappears after 30s because afternystagmus is no longer present (see Chaudhuri, 1991b, for relevant afternystagmus data).

Discussion

These experiments provide further evidence for an extra-retinal component to MAE. Eye movements do more than simply distort the retinal stimulus during adaptation: they create non-visual signals that contribute to the aftereffect. The particular signal we are concerned with is one that is generated by the need to suppress afternystagmus in order to fixate a stationary test. Chaudhuri (1991b) found a good correlation between an MAE following repeated pursuit eye movements and the ensuing afternystagmus eye movement. Because the adapting stimulus contained no retinal motion (other than that to drive the eye movement), Chaudhuri concluded the MAE was extra-retinal in origin. We have extended his findings to nystagmus eye-movements more reflexive in nature. We too find MAE in a part of the visual field that has not received prior retinal stimulation during eye-movement adaptation. In a direct test of the nystagmus-suppression hypothesis we found the extra-retinal MAE fails to store appreciably following 30s delay between adaptation and test. In comparison, using the same observers we found almost complete storage of an equivalent retinal MAE.

MAE duration is, we accept, a somewhat subjective dependent measure, although it is still widely in use today. A more sophisticated technique is to null the perceived motion by opposing it with physical motion of the test, either by adjusting its speed (see Pantle, 1998) or by altering motion energy using added noise (e.g. Hiris & Blake, 1992; Snowden & Milne, 1997). Another method is to match the speed of the aftereffect with another visual stimulus located in a previously unadapted position. Unfortunately, the extra-retinal MAE affects all visual locations (Chaudhuri, 1991a) and so visual matching is ruled out. Nulling is also problematical. Moving the small test dot used in our experiments would necessarily give rise to eye movements and associated retinal slip, both of which might interfere with the percept. This problem could be partially resolved using a larger stimulus viewed with a stationary fixation point. However, our preliminary observations confirm perceived motion of window and fixation point (the extra-retinal MAE is not retinotopic). This gives rise to a complex relative motion between the moving test pattern being used to null the aftereffect and the window / fixation point. Attempts to use motion nulling have thus far proved unsuccessful.

The existence of extra-retinal MAEs raises several intriguing issue. The first concerns the interaction between retinal and extra-retinal MAE. Some authors have reported MAE opposite to retinal motion created by repetitive pursuit during adaptation (Anstis & Gregory, 1965). Others have claimed central MAE in the same direction as the retinal motion created in the periphery (Mack et al., 1987; Morgan et al., 1976). The debate has partially been resolved in terms of the degree to which peripheral MAEs can induce central ones (Wade et al., 1996). However, the existence of extra-retinal MAE during test has been largely ignored. How might this impact on the above interpretations? Retinal motion accompanying pursuit over a stationary background is opposite to the eye movement. When no peripheral landmarks are visible, the size and direction of subsequent MAE arguably depends on the relative size of retinal and extra-retinal MAEs because in principle these

move in opposite directions at the same visual location. Of course, this presupposes that retinal and extra-retinal MAE can interact with one another, an area we know nothing about. Introducing peripheral stimulation makes the situation even more complicated because now peripheral MAEs may introduce an induced motion component to the central test.

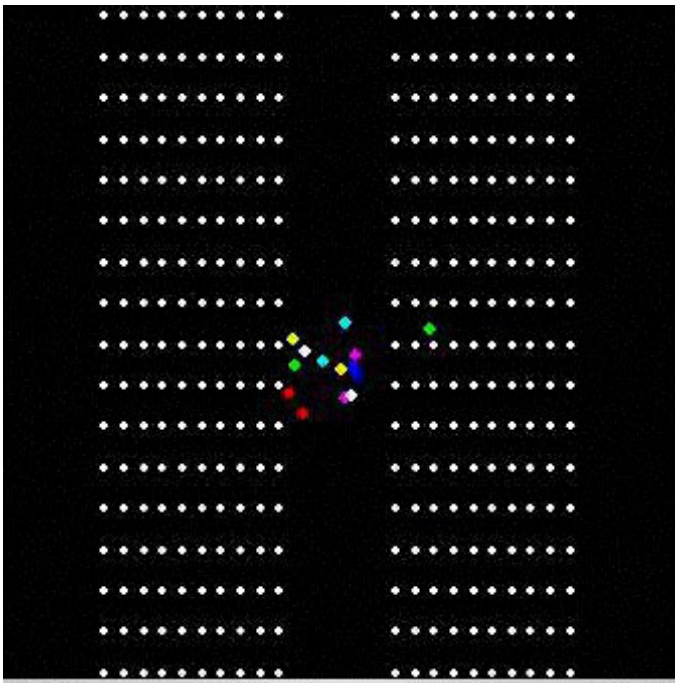
A related issue concerns the motion perceived during adaptation. Mack, Hill & Kahn (1989) argued that MAE was determined not by the degree of retinal MAE induced by the repetitive eye movement but by the perceived motion experienced during the adaptation phase. They created stimuli in which a vertical eye movement was made repeatedly over a stimulus moving horizontally. This produces oblique retinal motion – hence, MAE should also be oblique if all that determines subsequent aftereffect is motion stimulating the retina. However, they found all observers reported horizontal MAE, an effect we have confirmed on ourselves. Mack et al concluded that the MAE was consistent with the motion perceived during adaptation because all their observers correctly reported the horizontal motion of the adapting stimulus. However, the same aftereffect could equally be the result of an oblique retinal MAE summed with vertical extra-retinal MAE. This idea remains to be explored.

A third issue concerns the relationship between the extra-retinal MAE we report and the perception of self-motion, or vection. It is well known that observers placed inside a rotating drum experience vection even though they are physically stationary. Moreover, there is evidence that vection continues once the stimulation ceases, either by stopping the stimulus (Brandt, Dichgans & Koenig, 1973) or turning the lights out (Schor & Westall, 1986). The conditions are quite distinct from those described here, however: in the former condition, retinal adaptation may play a role, while in the latter, afternystagmus is seen. Moreover, when prompted none of our observers reported the sensation of self-motion during adaptation, nor attributed the motion of the test dot to the ego. The absence of vection may partly have been because adapting motion and aftereffect were vertical.

A final issue concerns the influence of extra-retinal MAE in judgements that may rely on the use of concurrent extra-retinal information concerning eye velocity. One of these is the perception of heading during eye movement. Evidence suggests that extra-retinal signals are an important component of the process that compensates for the retinal effect of eye movement (Ehrlich, Beck, Crowell, Freeman & Banks, 1998; Freeman, 1999; Freeman, Banks & Crowell, 2000; Li & Warren, 2000; Royden, Banks & Crowell, 1992). Extra-retinal MAEs may alter these signals and so lead to measurable distortions in perceived heading (e.g. slalom illusion; go to first author's home page for online demonstration). In similar vein, extra-retinal signals are thought to contribute to the perception of object motion during eye movement (Freeman, 2001; Freeman & Banks, 1998; Freeman & Sumnall, 2002; Mack & Herman, 1978; Turano & Massof, 2001; Wertheim, 1994). Preliminary experiments suggest that perceived stationarity during eye movement is modified following eye-movement adaptation (Sumnall & Freeman, 2002).

Appendix

The following movie contains a 20s sample of the eye movements made by all observers contributing to Figure 3's upward eye-moving condition. The eye movements of individual observers are shown as coloured symbols and are superimposed on the adaptation stimulus. One might notice the clear outlier (the green point on the right) who insists on tracking the right-hand strip despite instructions to maintain gaze on the central blank area. Despite this, their tracking is reasonably accurate, suggesting only small amounts of retinal slip stimulating the fovea.



Movie 1. Sample of observer eye movements.

Acknowledgements

The research was supported by a project grant from the Wellcome Trust. Commercial relationships: none.

References

Anstis, S., Verstraten, F. A. J., & Mather, G. (1998). The motion aftereffect. *Trends in Cognitive Sciences, 2* (3), 111-117.

Anstis, S. M., & Gregory, R. L. (1965). The aftereffect of seen motion: the role of retinal stimulation and of eye movement. *Quarterly Journal of Experimental Psychology, 17*, 173-174.

Beutter, B. R., & Stone, L. S. (2000). Motion coherence affects human perception and pursuit similarly. *Visual Neuroscience, 17* (139-153) [PubMed]

Brandt, T., Dichgans, J., & Koenig, E. (1973). Differential effects of central versus peripheral vision on egocentric and exocentric motion perception. *Experimental Brain Research, 16*, 476-191. [PubMed]

Carpenter, R. H. S. (1988). *Movements of the Eyes*. London: Pion.

Chaudhuri, A. (1990). A motion illusion generated by afternystagmus suppression. *Neuroscience Letters, 118*, 91-95. [PubMed]

Chaudhuri, A. (1991a). Eye-movements and the motion aftereffect - alternatives to the induced motion hypothesis. *Vision Research, 31* (9), 1639-1645. [PubMed]

Chaudhuri, A. (1991b). Pursuit afternystagmus asymmetry in humans. *Experimental Brain Research, 83*, 471-476. [PubMed]

Cheng, M., & Outerbridge, J. S. (1974). Inter-saccadic interval analysis of optokinetic nystagmus. *Vision Research, 14*, 1053-1058. [PubMed

Chung, S. T. L., & Bedell, H. E. (1995). 'Dumping' of rebound nystagmus and optokinetic afternystagmus in humans. *Experimental Brain Research, 107*, 306-314. [PubMed]

Clement, G., & Lathan, C. E. (1991). Effects of static tilt about the roll axis on horizontal and vertical optokinetic nystagmus and optokinetic after-nystagmus in humans. *Experimental Brain Research, 84*, 335-341. [PubMed]

Clifford, C. W. G. (2002). Perceptual adaptation: motion parallels orientation. *Trends in Cognitive Sciences, 6* (3), 136-143. [PubMed]

Cohen, B., Matsuo, V., & Raphan, T. (1977). Quantitative analysis of the velocity characteristics of optokinetic nystagmus and optokinetic afternystagmus. *Journal of Physiology, 270*, 321-344. [PubMed]

Crognale, M. A., & Schor, C. M. (1996). Contribution of chromatic mechanisms to the production of small-field optokinetic nystagmus (OKN) in normals and strabismics. *Vision Research, 36* (11), 1687-1698. [PubMed]

Ehrlich, S. M., Beck, D. M., Crowell, J. A., Freeman, T.C.A., & Banks, M.S. (1998). Depth information and perceived self-motion during simulated gaze rotations. *Vision Research, 38* (20), 3129-3145. [PubMed]

Evanoff, J. N., & Lackner, J. R. (1987). Influence of maintained ocular deviation on the spatial displacement component of the oculogyral illusion. *Perception & Psychophysics, 42* (1), 25-28. [PubMed]

Freeman, T. C. A. (1999). Path perception and Filehne illusion compared: model and data. *Vision Research, 39* (16), 2659-2667. [PubMed]

Freeman, T. C. A. (2001). Transducer models of head-centred motion perception. *Vision Research, 41*, 2741-2755. [PubMed]

Freeman, T. C. A., & Banks, M. S. (1998). Perceived head-centric speed is affected by both extra-retinal and retinal errors. *Vision Research, 38* (7), 941-945. [PubMed]

Freeman, T. C. A., Banks, M. S., & Crowell, J. A. (2000). Extraretinal and retinal amplitude and phase errors during Filehne illusion and path perception. *Perception & Psychophysics, 62* (5), 900-909. [PubMed]

Freeman, T. C. A., & Sumnall, J. H. (2002). Motion versus position in the perception of head-centred movement. *Perception, 31*, 603-615. [PubMed]

Gizzi, M., Raphan, T., Rudolph, S., & Cohen, B. (1994). Orientation of human optokinetic nystagmus to gravity: a model-based approach. *Experimental Brain Research, 99*, 347-360. [PubMed]

Harris, L. R., & Smith, A. T. (1992). Motion defined exclusively by 2nd-order characteristics does not evoke optokinetic nystgmus. *Visual Neuroscience, 9*, 565-570. [PubMed]

Harris, L. R., & Smith, A. T. (2000). Interactions between first- and second-roder motion revealed by optokinetic nystgmus. *Experimental Brain Research, 130*, 67-72. [PubMed]

Hiris, E., & Blake, R. (1992). Another perspective on the visual motion aftereffect. *Proceedings of the National Academy of Sciences, 89*, 9025-9028. [PubMed]

Ilg, U. J. (1997). Responses of primate area MT during the execution of optokinetic nystagmus and afternystagmus. *Experimental Brain Research, 113*, 361-364. [PubMed]

Kowler, E. (1989). Cognitive expectations, not habits, control anticipatory smooth oculomotor pursuit. *Vision Research, 29* (9), 1049-1057. [PubMed]

Krauzlis, R. J., & Adler, S. A. (2001). Effects of directional expectations on motion perception and pursuit eye movements. *Visual Neuroscience, 18*, 365-376. [PubMed]

Li, L., & Warren, W. H. (2000). Perception of heading during rotation: sufficiency of dense motion parallax and reference objects. *Vision Research, 40* (28), 3873-3894. [PubMed]

Mack, A., Goodwin, J., Thordarsen, H., Benjamin, D., Palumbo, D., & Hill, J. (1987). Motion aftereffects associate with pursuit eye movements. *Vision Research, 27* (4), 529-536. [PubMed]

Mack, A., & Herman, E. (1978). The loss of position constancy during pursuit eye movements. *Vision Research, 18*, 55-62. [PubMed]

Mack, A., Hill, J., & Kahn, S. (1989). Motion aftereffects and retinal motion. *Perception, 18* (5), 649-655. [PubMed]

Mather, G., Verstraten, F., & Anstis, S. (1998). *The motion aftereffect: a modern perspective*. Cambridge, Massachusetts: Bradford Book.

Morgan, M. J., Ward, R. M., & Brusell, E. M. (1976). The aftereffect of tracking eye movements. *Perception, 5*, 309-317.

Muratore, R., & Zee, D. S. (1979). Pursuit after-nystagmus. *Vision Research, 19*, 1057-1059. [PubMed]

Pantle, A. (1998). How do measures of the motion aftereffect measure up? In G. Mather, F. Verstraten, & S. Anstis (Eds.), *The Motion Aftereffect: a Modern Perspective*. Cambridge, Massachusetts: Bradford Book.

Pola, J., & Wyatt, H. J. (1985). Active and passive smooth eye movements: effects of stimulus size and location. *Vision Research, 25* (8), 1063-1076. [PubMed]

Royden, C. S., Banks, M. S., & Crowell, J. A. (1992). The perception of heading during eye-movements. *Nature, 360* (6404), 583-587. [PubMed]

Schor, C., & Narayan, V. (1981). The influence of field size upon the spatial frequency response of optokinetic nystagmus. *Vision Research, 21*, 985-994. [PubMed]

Schor, C. M., & Westall, C. (1986). Rapid adaptation of the vestibulo-ocular reflex and induced self-motion perception. *Perception & Psychophysics, 40* (1), 1-8. [PubMed]

Snowden, R. J., & Milne, A. B. (1997). Phantom motion aftereffects - evidence of detectors for the analysis of optic flow. *Current Biology, 7*, 717-722. [PubMed]

Sumnall, J. H., & Freeman, T. C. A. (2002). Pursuit adaptation alters perceived head-centred motion [Abstract]. *Journal of Vision, 2*(7), 504a, http://journalofvision.org/2/7/504/, DOI 10.1167/2.7.504. [Abstract]

Sumnall, J. H., Freeman, T. C. A., & Snowden, R.J. (2003). Optokinetic potential and the perception of head-centred speed. *Vision Research, 43*(16), 1709-18. [PubMed]

Swanston, M. T., & Wade, N. J. (1992). Motion over the retina and the motion aftereffect. *Perception, 21*, 569-582. [PubMed]

Thompson, P. (1998). Tuning of the motion aftereffect. In G. Mather, F. Verstraten, & S. Anstis (Eds.), *The Motion Aftereffect: a Modern Perspective*. Cambridge, Massachusetts: Bradford Book.

Thompson, P., & Wright, J. (1994). The role of intervening patterns in the storage of the movement aftereffect. *Perception, 23*, 1233-1240. [PubMed]

Turano, K. A., & Massof, R. W. (2001). Nonlinear contribution of eye velocity to motion perception. *Vision Research, 41*, 385-395. [PubMed]

van den Berg, A. V., & Collewijn, H. (1988). Directional asymmetries of human optokinetic nystagmus. *Experimental Brain Research, 70*, 597-604. [PubMed]

Wade, N. J., Spillman, L., & Swanston, M. T. (1996). Visual motions aftereffects: critical adaptation and test conditions. *Vision Research, 36* (14), 2167-2175. [PubMed]

Wertheim, A. H. (1994). Motion perception during self-motion - the direct versus inferential controversy revisited. *Behavioral and Brain Sciences, 17* (2), 293-311.