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Storage of an oculomotor motion aftereffect

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Abstract

Adaptation to motion produces a motion aftereffect (MAE), where illusory, oppositely-directed motion is perceived when viewing a stationary image. A common hypothesis for motion adaptation is that it reflects an imbalance of activity caused by neuronal fatigue. However, the perceptual MAE exhibits storage, in that the MAE appears even after a prolonged period of darkness is interposed between the adapting stimulus and the test, suggesting that fatigue cannot explain the perceptual MAE. We asked whether neural fatigue was a viable explanation for the oculomotor MAE (OMAE) by testing if the OMAE exhibits storage. Human observers were adapted with moving, random-dot cinematograms. Following adaptation, they generated an oculomotor MAE (OMAE), with both pursuit and saccadic components. The OMAE occurred in the presence of a visual test stimulus, but not in the dark. When the test stimulus was introduced after the dark period, the OMAE reappeared, analogous to perceptual MAE storage. The results suggest that fatigue cannot explain the OMAE, and that visual stimulation is necessary to elicit it. We propose a model in which adaptation recalibrates the motion-processing network by adjusting the weights of the inputs to neurons in the middle-temporal (MT) area.

Keywords: Eye movements; Smooth pursuit; Human

1. Introduction

The motion aftereffect (MAE) occurs when an observer adapts to motion that is viewed for an extended time. It is commonly manifest as the perception that a static surface moves opposite the direction of the adapting motion. The MAE has been extensively studied (e.g., Addams, 1834; Bex, Bedingham, & Hammett, 1999; Gibson, 1937; Levinson & Sekuler, 1976; Purkinje, 1820; Spigel, 1960, 1962a, 1962b, 1964; Thompson & Wright, 1994; Verstraten, Fredericksen, Grusser, & Van de Grind, 1994; Wohlgemuth, 1911), however the neural substrate of this phenomenon remains unknown. An early theory suggested that fatigue occurs in neurons that encode the direction of the adapting stimulus, leading to an imbalance in activity favoring the opposite motion direction. This has been hypothesized to occur because the fatigued neurons are unable to sustain high firing rates for a long time, or neurotransmitters are depleted, resulting in the fewer action potentials (Barlow & Hill, 1963; Wohlgemuth, 1911). A competing theory is that the synaptic weights of the neuronal network subserving motion processing are recalibrated by adaptation (Gibson, 1937; Harris, Morgan, & Still, 1981; Wiesenfelder & Blake, 1992).

Many physiological studies have assessed V1 (Giaschi, Douglas, Marlin, & Cynader, 1993; Hammond, Mouat, & Smith, 1985, 1986; Maffei, Fiorentini, & Bisti, 1973; Marlin, Hasan, & Cynader, 1988; Vautin & Berkley, 1977; vonderHeydt, Hänny, & Adorjani, 1978) and MT neurons (Kohn & Movshon, 2004; Petersen, Baker, & Allman, 1985; van Wezel & Britten, 2002) during the course of adaptation but the results are mixed and do not clearly differentiate between the fatigue and recalibration models. However, the perceptual phenomenon of MAE storage is strong evidence against neuronal fatigue as the mechanism of adaptation. Storage occurs when a period of darkness is imposed

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between the adapting stimulus and the test stimulus, postponing the MAE until the test stimulus appears (Spigel, 1960, 1962a, 1962b, 1964; Thompson & Wright, 1994; Verstraten et al., 1994; Wohlgemuth, 1911). This occurs even when the duration of the dark period is much longer than that of the usual MAE, and can leave the strength of the MAE relatively undiminished. If neuronal fatigue was the mechanism of adaptation, the adapted neurons should recover their activity level during the dark period thus preventing the expression of an MAE at a later time.

Evidence of a smooth pursuit MAE, which has characteristics similar to the perceptual MAE, has been found recently (Braun, Pracejus, & Gegenfurtner, 2006; Gardner, Tokiyama, & Lisberger, 2004). One might expect that the pursuit MAE would also exhibit storage, because motion pathways in the middle temporal/medial superior temporal (MT/MST) complex that are commonly thought to underlie motion perception provide input to the pursuit system (Komatsu & Wurtz, 1988; Newsome, Wurtz, & Komatsu, 1988). Moreover, smooth pursuit has been shown to reflect motion perception (Beutter & Stone, 1998; Heinen & Watamaniuk, 1998; Stone & Krauzlis, 2003; Watamaniuk & Heinen, 1999). However, these results cannot be taken as unequivocal evidence that neuronal changes that underlie the smooth pursuit MAE are restricted to the motion pathways that subserve perception. Changes in the pursuit system itself, including fatigue, could contribute to the pursuit MAE. To determine if neuronal fatigue is involved in the pursuit MAE, we investigated whether adaptation was preserved following a period of no visual stimulation.

2. Method

All experiments were approved by the California Pacific Medical Center institutional review board. Three human observers (two naïve) gave informed consent and participated in the experiments.

2.1. Stimuli

The adapting stimulus was a random-dot cinematogram (RDC) with component dots displayed at a density of 3.0 dots/deg^2. The pursuit stimulus was also an RDC (0.05 deg dot diameter, dot luminance = 14.4 cd/m^2) that moved against a dark background. Stimuli were presented on a 17 in. high-resolution computer monitor (1.76 min arc/pixel) at a rate of 60 Hz and RDCs were viewed through a 20 deg diameter aperture. The background luminance was (0.46 cd/m^2) and all stimuli were viewed from a distance of 80 cm. When the stimulus was an RDC, all dots moved in the same direction and at the same speed and virtually "wrapped around" when the border of the RDC was reached.

2.2. Procedure

Two separate types of trial blocks were used, *no-gap* and *gap*. Each block of trials began with the observer fixating a spot in the center of the screen (see Fig. 1). Simultaneous with the appearance of the spot, the adapting RDC appeared with component dots that moved upward at 10 deg/s. After 60 s, the RDC was turned off. On *no-gap* trials, the adapting RDC was followed immediately by a second RDC, the pursuit target, with component dots that moved at 0.5 deg/s. One way to minimize the chance that observers will predict the stimulus is to include, in a block of trials, stimuli that move at several different speeds in the same or opposite direction of the adapt stimulus, a technique used by previous investigators to reveal a pursuit MAE (Braun et al., 2006). However, anticipatory pursuit movements that are based on previous motion occur even when target motion is unpredictable (Heinen, Badler, & Ting, 2005; Kowler, Martins, & Pavel, 1984), and could add noise to the measured velocity. Therefore, while we adopted the multiple velocity paradigm, we modified it so that the principle direction of the test stimuli was orthogonal to the upward adapt stimulus so that anticipatory eye velocity generated by the adapt stimulus would have a direction roughly orthogonal to the test. Pursuit target motion direction was randomly set to one of five possible directions (−20, −10, 0, 10, 20 deg), with zero being directly rightward. Observers were required to follow the target with their eyes, which they did with a combination of smooth pursuit and saccades. After 1500 ms, the pursuit RDC disappeared and the next trial began. *Gap* trials were identical to *no-gap* trials, except that a 1000 ms blank period was interposed between the adapting and pursuit stimuli, and the pursuit RDC was presented for only 1000 ms.

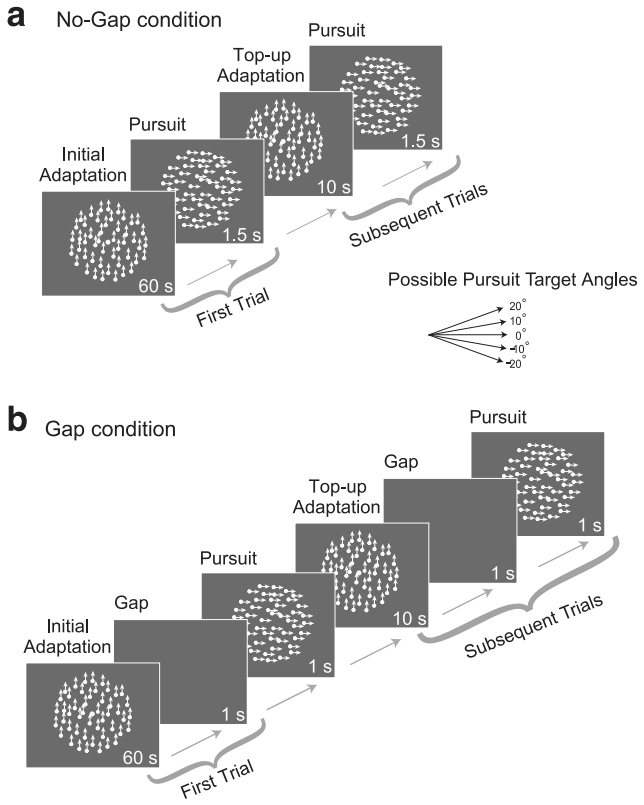

Fig. 1. Schematic representation of the adaptation protocol for experiments without and with a gap period. Each block of trials began with 60 s adaptation (a static spot was provided for fixation). (a) When there was no gap, a 1.5 s RDC pursuit stimulus moving orthogonal to the adaptation stimulus was next presented. Each trial was immediately followed by 10 s of top-up adaptation. (b) In the gap condition, a blank, dark screen (1 s) was presented immediately following the initial and top-up adaptation epochs, followed by a 1 s pursuit stimulus. The pursuit stimulus in all conditions moved in one of five directions spaced every 10 deg from −20 to +20 deg, centered about rightward (0 deg). In control trials, the stimulus presentation was the same except the adapt stimuli were static.

All trials after the first trial were preceded by a 10 s "top-up" adaptation period to maintain the level of motion adaptation. Control trial blocks were also performed for both *no-gap* and *gap* conditions. These were the same as the adapt blocks except that the adapting RDC was stationary. To avoid possible long-term adaptation effects, control blocks were never run sooner than 30 min after completion of an adapt block. The first trial from every block was excluded from the data analysis, due to possible "surprise" effects from the sudden appearance of the target after

the relatively long adapt period. The total number of trials in each block was 45, and a minimum of 2 blocks were run for each condition. Note that an equipment failure made the data file for one of the gap/no-gap blocks for HY unreadable and thus only one block for HY was used in the analysis.

2.3. Eye movement measurement and analysis

Observers viewed the display monocularly with one eye patched. Horizontal and vertical eye position were recorded using a Generation V dual-Purkinje-image eyetracker and sampled by computer at 1000 Hz. In tests of our instrument with an artificial eye, the overall noise of the system was less than 1 min arc. Eye position was calibrated to tracker output before each session by having the subject fixate several times at each of four 5-deg-eccentric cardinal positions, two 10-deg eccentric positions along the horizontal axis, and at the center while offsets and gains were adjusted. Eye velocity was obtained by digital differentiation of eye position signals and filtered to reduce 60 Hz noise (2 pole Butterworth filter, cutoff = 50 Hz). Saccades were located using a proprietary algorithm, which detected when the variance of eye velocity over a small time window (10 ms) exceeded a threshold, 150 $(\text{deg/s})^2$. Saccades were then excised from the data and replaced with a line using an interpolation algorithm. All eye movement records were manually checked by human operators to ensure that no residual saccade artifacts contaminated the velocity traces. Records contaminated by eye blinks were not included in the analyses.

Since subjects tracked using a combination of pursuit and saccades, the tracking direction was determined using eye position data (with saccades intact). Horizontal and vertical eye position were filtered (cutoff = 25 Hz). The median position value of the first 50 ms was subtracted from each trace to correct for fixation offsets. Traces were further subsampled every 25 ms. A linear regression was then performed on the subsampled data. The tracking angle was computed by taking the arctangent of the regression slope.

3. Results

Fig. 2a shows mean vertical smooth eye velocity difference traces after adaptation to upward motion for each observer. To obtain these traces, eye velocity was averaged over all trials and test velocities, removing saccades from the records before averaging. Mean eye traces from control trials in which observers pursued the same test stimuli after adapting to a static RDC were then subtracted from the mean adapt traces. Notice that all observers showed a weak, but consistent, downward deflection of eye velocity, indicative of an OMAE elicited by the upward adapting stimulus. However, the predominant OMAE resulted from a combination of pursuit and saccadic movements.

Plotted in Fig. 2b are two-dimensional eye position traces showing combined pursuit and saccadic eye movements of observer HY during pursuit of a rightward-moving test stimulus in both the control and adapt conditions. Eye position for the adapt trials (black lines) shows a robust downward trend relative to control (grey lines). To quantify the conglomerate oculomotor response, we performed a linear regression on each two-dimensional eye position record, and computed the slope of the best fitting regression line (see Section 2).

The eye velocity difference traces in Fig. 2a suggest that the duration of the OMAE was short, diminishing about 800 ms after target onset. Because of this, and since the OMAE did not manifest itself until approximately 200 ms,

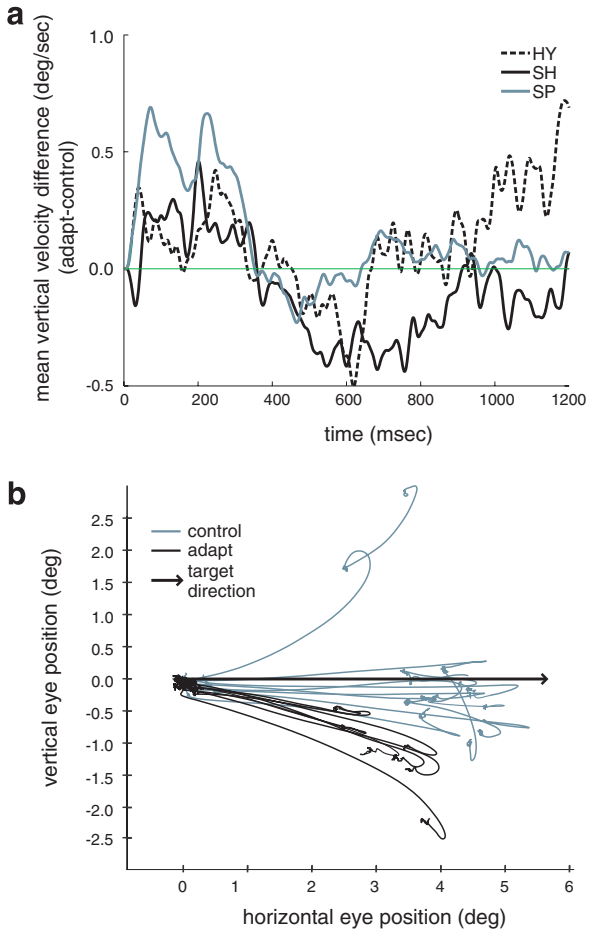

Fig. 2. Eye movement data for the *no-gap* condition. (a) Mean vertical velocity difference plotted as a function of time for three observers. Smooth eye velocity showed a deflection in the downward direction (~200–800 ms), opposite the adapt direction. The downward velocity is suggestive of an oculomotor MAE. (b) 2-dimensional position plot of pursuit and saccadic eye movements made to rightward-moving (0 deg) targets either after adaptation to a static RDC (control) or an RDC moving upward (adapt) for one observer (HY). For clarity, only the first eight trials of each condition are shown.

we chose to use the regression fits to quantify the OMAE over the 200–800 ms interval. Plotted in Fig. 3a are the values of the median control and adapt slopes from the regressions for all observers and each of the five different pursuit test directions. Note that the data for the adapt condition lie consistently below that of the control condition, characterizing a downward deflection of the eye movements, or an OMAE. Analyzing the later 800–1200 ms period revealed that this trend was no longer present (Fig. 3b). Therefore, the OMAE appeared to dissipate quickly during visual stimulation.

The average difference in regression slopes between the adapt and control conditions are summarized for each observer for both the early (200–800 ms) and late (800–1200 ms) periods of the pursuit phase in Fig. 3c. All three observers showed an OMAE in the early period that went away by the late period. An ANOVA verified this trend: for the early period, the median slopes for the adapt condition

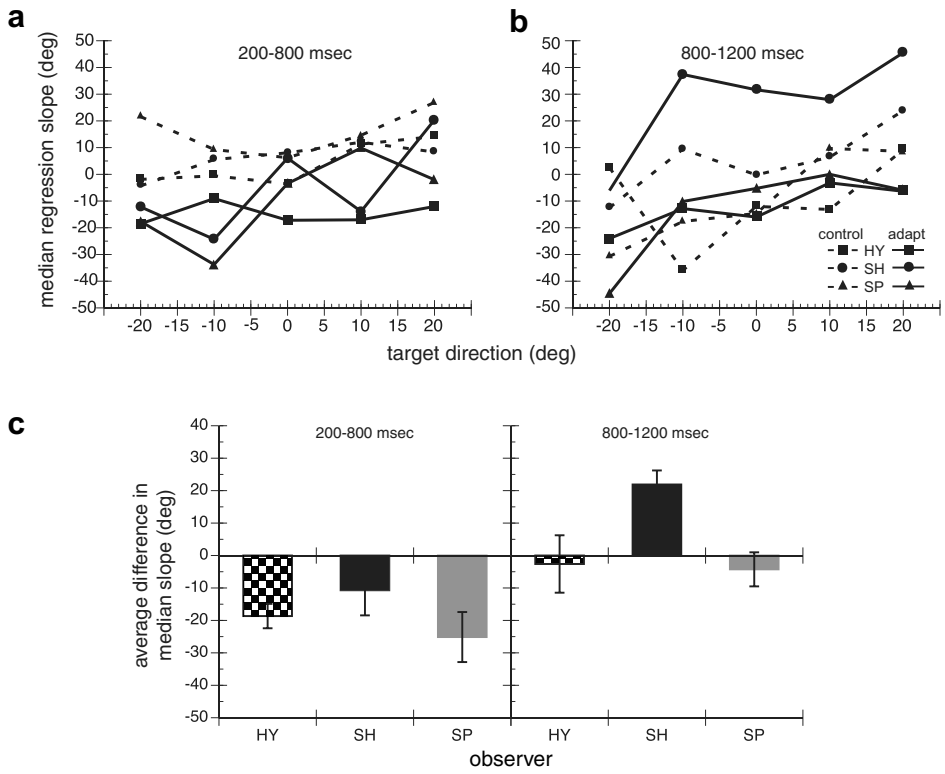

Fig. 3. Summary of the OMAE eye movements. (a) Median regression slope values for each target direction for both control (dashed lines) and adapt (solid lines) conditions in the no-gap experiment 200–800 ms after target onset (early period) for all three observers. Note that with few exceptions overall, for all target directions, adapt trials produced larger downward slopes for all observers. (b) Median regression slope values for each test direction 800–1200 ms after target onset (late period). Note that the control and adapt trials now show similar direction eye movements. (c) The difference in median slope (adapt – control) of eye position traces averaged over all target directions in the early and late periods. Note that all observers' eye movements exhibited a downward bias indicative of an oculomotor MAE early but that the effect dissipates quickly, within about 1 s of pursuing the visual target motion.

were lower than those for the control condition ($F(1,20) = 21.393$, $p = .0002$) while no difference between the adapt and control conditions was found for the late period ($F(1,20) = 0.463$, $p = .504$).

Given the smooth pursuit system receives input from MT, which processes motion for perception (Komatsu & Wurtz, 1988; Newsome et al., 1988), the expression of an OMAE following adaptation was not surprising. However, although the pursuit system receives input from the motion processing system, there are many other structures in this (e.g., Keller & Heinen, 1991; Lisberger, Morris, & Tychsen, 1987) and the saccadic system (Wurtz & Goldberg, 1989.) that are likely not part of the motion perception system. Therefore, it is not safe to assume that the neural processes responsible for the perceptual MAE are the same as those responsible for the OMAE, and previous studies demonstrating storage in the perceptual system do not rule out fatigue as a mechanism for the OMAE. To test this, we performed a second experiment in which eye movements were measured within the same adaptation paradigm as the previous experiment, except that now a 1 s blank period (gap) was introduced immediately after the adapting stimulus was extinguished. To insure that placing observers in the dark did not somehow disturb the eventual expression of the OMAE, the same test pursuit stimulus used in the first experiment was presented after the gap.

Surprisingly, during the gap the eyes failed to move in the direction of the previously observed OMAE. If anything, there was a tendency for a slow, upward drift that resembled the oculomotor phenomenon of optokinetic after-nystagmus (OKAN) (Fig. 4a). As the figure shows, when the test stimulus was presented after the gap, vertical eye velocity was deflected downwards, consistent with the OMAE. However, this effect was weak, and may instead reflect a correction for the vertical velocity error produced by the upward drift during the gap and the appearance of the test. However, when we analyzed the position traces, it became clear that the OMAE was still present after the gap. Fig. 4b shows two-dimensional eye position traces of observer HY after adaptation but during the gap for both adapt and control conditions. Here, the eyes appear to move in a random fashion. Fig. 4c shows eye position when the test stimulus was presented following the gap. Here, the adapt traces show a clear downward bias. Note that the eyes do not start at the same vertical position as controls because of the drift that occurred during the gap period. However, the downward eye movement was not a correction for a position error that may have occurred during the gap because RDCs do not have consistent position cues

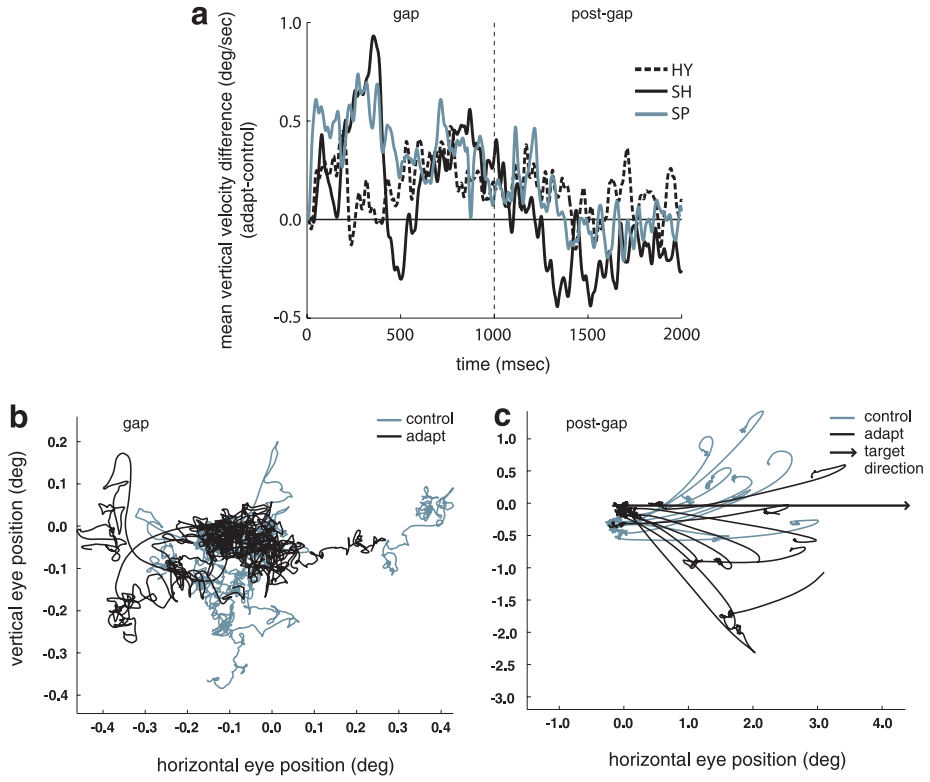

Fig. 4. Eye movement data for the *gap* condition. (a) Mean vertical eye velocity difference (adapt – control) plotted as a function of time for three observers. During the gap, smooth eye velocity initially showed an upward deflection that was in the same direction as the adapt stimulus. When the rightward-moving test stimulus was presented (post-gap), eye velocity abruptly reversed to downward, opposite the adapt direction, again suggestive of an OMAE. Panels (b) and (c) show position plots of the first eight control and adapt trials for one observer (HY). Note different axis ranges. (b) A 2-dimensional position plot of pursuit and saccadic eye movements made during the gap (following upward adaptation) when the screen was blank. The eyes appear to move randomly, showing little difference between control and adapt trials. (c) A 2-dimensional position plot of pursuit and saccadic eye movements for control and adapt trials made to rightward-moving (0 deg) targets presented after a 1.0 s gap. Note that as in the no-gap condition, the eyes show a clear downward bias relative to controls after the test stimulus was presented.

(e.g., Ball & Sekuler, 1979) making any position error difficult to detect in the RDC test stimulus.

We again summarized the position data by performing a linear regression on each two-dimensional eye position record and computing the slope of the best fitting regression line. The regression analysis showed no systematic bias in slope for the eye movements during the gap relative to the controls (Fig. 5a). However when the visual test stimulus was introduced after the gap, the eye movements again displayed a downward component. As can be seen from the regression slopes, all observers showed this trend (Fig. 5b). Therefore, the OMAE was preserved, or stored, through the dark period analogous to storage that occurs with the perceptual MAE.

Fig. 5c summarizes OMAE storage by showing the difference in median regression slope of the eye movements for all observers and test angles during both the gap and post-gap period. From the figure it appears that there was no systematic bias in the conglomerate eye movements during the gap, and an ANOVA conducted on the median slope data confirmed this ($F(1,20) = 0.0157$, $p = .696$). However, when the test was presented, the eye movements again showed the downward bias that was apparent during the no-gap condition, indicating that the OMAE had been stored during the dark period. An ANOVA conducted on the post-gap median slope data confirmed that post-gap, the eye movements were significantly more downward than during the gap ($F(1,20) = 23.139$, $p = .0001$). The analysis also showed a significant effect of test direction ($F(4,20) = 6.464$, $p = .0017$). Specifically, test directions with a greater downward angle produced larger downward eye movements, a result that was not found in the no-gap experiment. As further evidence of the prevalence of the OMAE after the gap, a t-test revealed that there was no significant difference in the magnitude of the OMAE (as measured by the average median slope differences) between the no-gap and post-gap ($t(28) = 1.166$, $p = 0.25$).

4. Discussion

In summary, adapting observers to visual motion biased their eye movements in a direction opposite that of the adapt stimulus when they viewed a visual test stimulus. This result is consistent with an oculomotor analogue of the perceptual MAE, which we term the OMAE. When observers were placed in the dark immediately after adaptation, no evidence of this oculomotor effect was found. However, when a test stimulus was presented after the dark

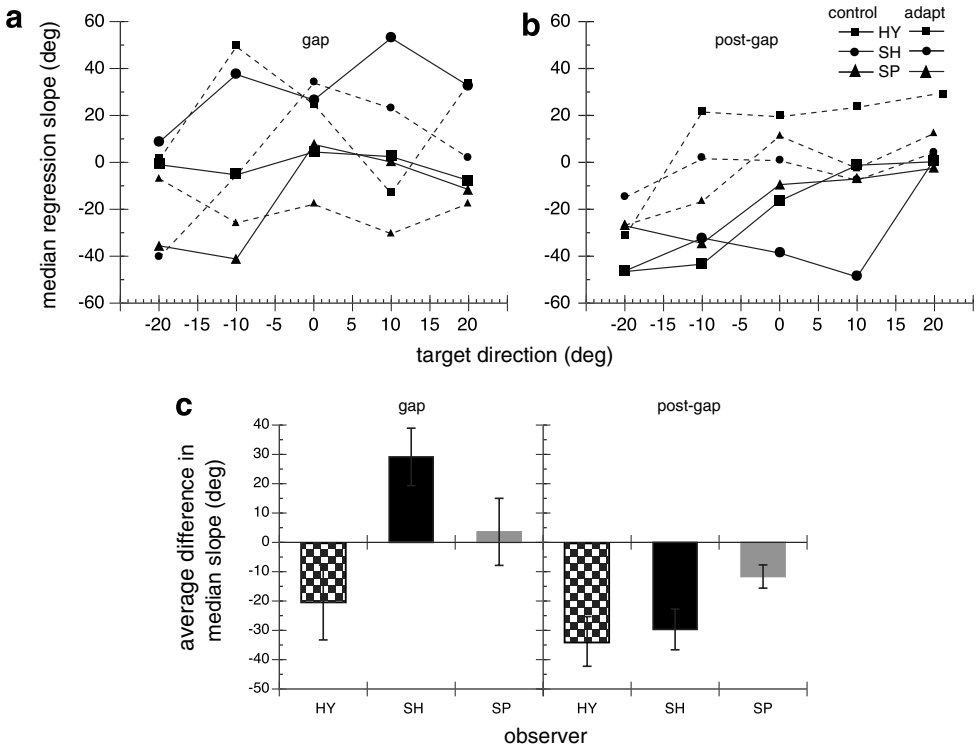

Fig. 5. Comparison of gap and post-gap eye movements. (a) Median regression slope values for each test direction for both control (dashed lines) and adapt (solid lines) conditions for the *gap* condition for all three observers for the time period 200–800 ms after extinction of the adapt stimulus. Note that eye movement data in adapt and control trials have similar slopes. (b) Median regression slope values for the post-gap period, 200–800 ms after visual target onset. Here, the eyes show a downward deflection consistent with an OMAE, demonstrating storage of the adaptation effect. (c) The difference in median slope (adapt – control) of eye position traces averaged over all target directions during the gap and post-gap periods. All observers' eye movements exhibited a downward bias indicative of an oculomotor MAE with the visual test stimulus.

period, the OMAE was again observed, demonstrating that the OMAE, like the perceptual MAE, exhibits storage.

This study was designed to probe the mechanism that subserves the MAE, utilizing a motor system rather than a purely perceptual system. The phenomenon of perceptual MAE storage essentially disproved neural fatigue theories and storage of the OMAE demonstrates that fatigue models also fail in the oculomotor system. Currently, the commonly held view is that some form of recalibration underlies adaptation though we know of no explicit models reflecting this view. It has been suggested that recalibration might serve as a mechanism for error correction (Andrews, 1964) to compensate for damage to the visual or oculomotor system. Error correction might also be used to calibrate the visual system during development. Alternatively, the purpose of recalibration might be to allow the visual system to have greater sensitivity in an optimal range (Barlow & Földiák, 1989; Krekelberg, van Wezel, & Albright, 2006; Tolias, Smirnakis, Augath, Trinath, & Logothetis, 2001). For example, this might be useful to improve acuity during extended periods of self-motion. Range adjustment occurs in photoreceptors during light or dark adaptation for a similar purpose in the luminance domain (Fain, Matthews, Cornwall, & Koutalos, 2001).

We measured eye movements to probe the adapted substrate during visual stimulation in the presence of a perceptual MAE, and in the dark, in the absence of a perceptual MAE. During test stimulation, we found an oculomotor aftereffect that was manifest as an eye movement response (smooth and saccadic) biased away from the direction of the adapting stimulus. Only a few studies have successfully measured a smooth pursuit MAE (Braun et al., 2006; Gardner et al., 2004). Our results extend these, and provide further evidence that the saccadic system is also privy to the neuronal signals that drive motion perception, which has been suggested before (deBrouwer, Yuksel, Blohm, Missal, & Lefèvre, 2002; Keller & Johnsen, 1990).

In the dark period between the termination of the adapting stimulus and the onset of the test stimulus, we found that the eye movements were not biased away from the direction of the adapt stimulus; if anything, they paradoxically moved toward it. These eye movements might be related to OKAN, which normally occurs following extended periods of visual motion (Cohen, Matsuo, & Raphan, 1977). OKAN is expressed in the same direction of the moving stimulus, and occurs after the stimulus is turned off. While commonly evoked with full-field motion, smaller stimuli have been used to produce it (van Die & Collewijn, 1982).

Our results, observing an OMAE and storage of the aftereffect, are in good agreement with perceptual MAE results and are consistent with the idea that recalibration underlies motion adaptation. A model of a motion-processing substrate that could explain our results is shown in

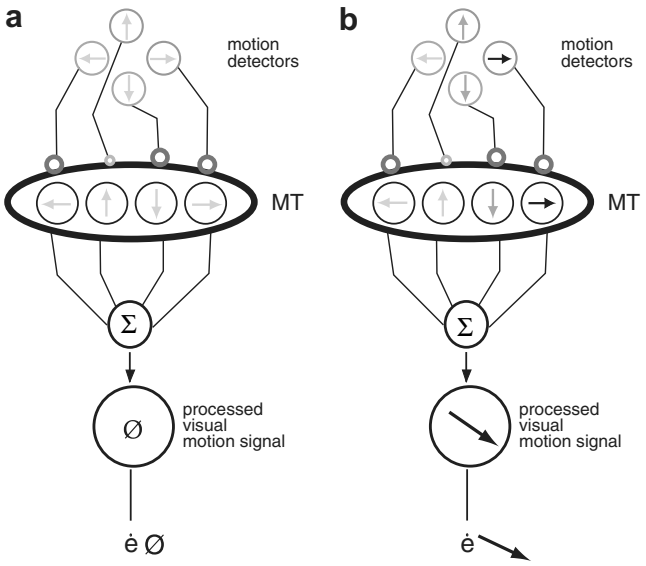

Fig. 6. Schematics of a model for the oculomotor MAE showing responses at various levels. (a) The state of the model in the dark, following adaptation to upward motion. Four example motion detectors representing cardinal directions of motion are not activated without visual stimulation (small grey arrows). The synapse that the upward unit makes with MT has been changed by adaptation (smaller, light grey circle; larger medium grey circles, other synapses). Because the motion detectors are not active, the effect of the synaptic adjustment is not realized in the MT neurons. When their output is summed, the processed motion signal is zero and the eyes do not move. (b) The model during rightward test stimulation. The rightward motion detector is activated, as well as the up and down ones to a lesser degree. Now, the upward MT unit is less activated than the downward one, resulting in a downward bias of the processed visual motion signal and the resultant eye movement.

Fig. 6. According to the model, adaptation to upward motion changes the weighting on the synapses between the motion detectors and MT, decreasing the efficacy of the upward channel (Fig. 6a). In the dark, the motion detectors and thus area MT are inactive. Since there is no net processed visual motion signal, the eyes do not move. Note that the effect of the altered synaptic weights is dormant in this condition. During exposure to a rightward-moving visual test stimulus, rightward motion detectors are activated (Fig. 6b). Since motion channels have sensitivity profiles that extend up to 135 deg to each side of their preferred direction (Maunsell & Van Essen, 1983; Snowden, Treue, & Andersen, 1992), the upward and downward detectors would also be mildly stimulated. Now, the altered synaptic weights decrease the activity of upward MT units and the vector average is a downward-biased rightward signal that is fed to the oculomotor system, producing the OMAE.

This scenario assumes that the adaptation effect seen in the eye movement system originates in the driving signals arriving from area MT which would be consistent with Culham et al. (1999) who showed that area MT+ was activated during perception of the MAE both immediately after adaptation and after a dark storage period. Alternatively, it is possible that adapting to visual motion produces adaptation effects at multiple cortical levels and that the effects on eye movements are due to adaptation at a level different from that responsible for the perceptual MAE. Testing between these alternatives is the topic for further research.

Competing interests statement

The authors declare that they have no competing financial interests.

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