Short- and long-term plasticity of eye position information: Examining perceptual, attentional, and motor influences on perisaccadic perception

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Spatial vision requires information about eye position to account for eye movements. But integrating eye position information and information about objects in the world is imperfect and can lead to transient misperceptions around the time of saccadic eye movements most likely because the signals are prone to temporal errors making it difficult to tell when the retinas move relative to when retinal images move. To clarify where this uncertainty comes from, in four experiments we examined influences of eye posture, attentional cueing, and trial history on perisaccadic misperceptions. We found evidence for one longer-term modulation of perisaccadic shift that evolved over the time of the test session due to biased eye posture. Another, short-term influence on perisaccadic shift was related to eye posture during preceding trials or the direction of the preceding saccade. Both perceptual effects could not be explained with visual delays, influences of attention or changes in saccade metrics. Our data are consistent with the idea that perisaccadic shift is caused by neural representations of eye position or space that are plastic and that arise from non-motor, extraretinal mechanisms. This suggests a perceptual system that continuously calibrates itself in response to changes in oculomotor and muscle systems to reconstruct a stable percept of the world.

Keywords: saccade, eye position signal, plasticity, oculomotor control, attention, spatial perception


Introduction

As you are reading these lines, your eyes move with quick saccades several times per second to obtain stable, highly resolved views of the words. But you also combine these views with retinal and/or extraretinal information about eye position to compensate for the movements of the retinas, otherwise the words would start jumping around in front of your eyes.

Under certain conditions, however, this spatial constancy is not completely achieved and visual stimuli do seem to jump: When a light flashes briefly in a dark room and someone makes a saccade, he/she will perceive the location of the flash as shifted in saccade direction even before saccade onset (Bockisch & Miller, 1999; Honda, 1991; Matin & Pearce, 1965; Schlag & Schlag-Rey, 1995). What causes these perisaccadic shifts?

As one possibility, eye position information might be fraught with one or two types of errors in time leading the brain to believe (a) that the eyes start moving before they actually do and/or (b) that they are moving more slowly, thus, resulting in a misinterpretation of vision (Dassonville, Schlag, & Schlag-Rey, 1992; Matin & Pearce, 1965; for a potential mechanism underlying these errors see Binda, Bruno, Burr, & Morrone, 2007). But then it remains unclear which source of eye position information perception uses. At least in darkness the information must come from sources other than the retina and, thus, are called extraretinal eye position signal (because only this case is considered here we simply use the term “eye position signal” or EPS). One extraretinal source of information about eye position is an “efference copy” of oculomotor commands that the superior colliculus projects onto mediodorsal thalamus from where it is sent to the frontal eye field (FEF; Sommer & Wurtz, 2002). This pathway carries information about saccade...
amplitudes or saccade goals. It does not represent an eye position signal in the strict sense of a continuous signal about eye position during and between eye movements, but perception could easily convert it into such information. In addition, continuous eye position information is directly available in other parts of ventral and intralaminar thalamus (Schlag-Rey & Schlag, 1984; Tanaka, 2007).

Alternatively, perception could extract eye position information from a corticale source: eye position could be predicted from oculomotor control signals in cortex, or from mechanisms programming shifts of attention. Saccades and attention seem tightly linked (Deubel & Schneider, 1996; Kowler, Anderson, Dosher, & Blaser, 1995), such as in the output from FEF (Hamker, 2005; Moore & Armstrong, 2003). FEF output, directly fed back into cortex, has been suspected to cause “perisaccadic compressions” (Hamker, Zirnsak, Calow, & Lappe, 2008), that is, under certain conditions people see perisaccadic flashes as crowded close to the saccade target rather than shifted (Lappe, Awater, & Krekelberg, 2000; Morrone, Ross, & Burr, 1997; Ross, Morrone, & Burr, 1997). Similarly, if perception used oculomotor commands to predict changes in eye position it might temporarily confuse oculomotor control signals with the actual onset of the saccade, hence causing perisaccadic shift.

An important difference between eye position information derived from oculomotor control signals as emitted from the FEF and eye position information derived from thalamic sources is that manipulating FEF output should change saccade metrics or saccade timing, but manipulating thalamic output should not. For example, Sommer and Wurtz (2002) inactivated mediodorsal thalamus and found that eye movements remained intact, but efference copy was impaired and led to errors when saccade targets had to be remapped. To emphasize the difference we will use the terms motor and non-motor EPS depending on whether it is emitted by a neural structure directly involved in oculomotor control or not.

Aside from EPS causing perisaccadic misperceptions, errors in the visual system have been recently proposed to contribute to perisaccadic shift (Pola, 2004), or to be the sole source of the problem (van Wetter & van Opstal, 2008).

To clarify what causes perisaccadic shifts here we sought to modulate shifts through manipulations of saccade latencies (Carpenter & Williams, 1995; He & Kowler, 1989; Posner, Snyder, & Davidson, 1980), eye-in-head effects (Paap & Ebenholtz, 1976; Park, 1969) and trial history (Dorris, Paré, & Munoz, 2000; Nakamura, Chung, Graziano, & Gross, 1999; Schlag-Rey & Schlag, 1984; Tanaka, 2007; Tanaka & Shimojo, 2000). We found that preceding saccades and eye posture influence perisaccadic shift, arguing against a purely visual account and in favor of an extraretinal EPS account of perisaccadic shift. Furthermore, saccade latencies were changed as well but neither this nor other saccade parameters could explain changes in shift, suggesting that the manipulated EPS was not identical with an oculomotor control signal, but arose from non-motor mechanisms.

### Methods

#### Subjects

Eleven participants (3 females, 8 males, 20–37 years old) gave their informed and written consent prior to their participation in the present study. Nine of them participated in Experiment 1, 8 in Experiment 2, and 3 participated in Experiments 2a and 3, respectively. Seven participants (including the authors) were tested in Experiments 1 and 2. Three of these (2 authors) also participated in Experiments 2a and/or 3. One person participated in Experiments 2a and 3. All participants were healthy and had normal or corrected-to-normal vision and all except the three authors were naïve to the experimental conditions and hypotheses. All procedures were approved by the Human Participants Review Sub-Committee of the University of Toronto and have therefore met the ethical standards laid out in the 1964 Declaration of Helsinki.

#### Apparatus and procedure

The experiments were performed in a dimly lit room. The participants’ head was fixed using a bite bar, 80 cm away from the center of a rear projection screen (203 cm by 152 cm). Onto the screen a Sony CRT projector (frame rate: 120 Hz, 1024 × 768 pixels) backprojected stimuli on a dark gray background (6 cd/m2). Eye movements were recorded with a head-mounted eye tracker (EyeLink II, SR Research, Ottawa; sampling rate: 500 Hz).

Both experiments were designed to yield perisaccadic misperceptions of spatial locations emphasizing perisaccadic shifts rather than compression (Lappe et al., 2000). Perisaccadic shifts and compressions seem to be at least partially independent phenomena (Michels & Lappe, 2004; Ostendorf, Fischer, Finke, & Ploner, 2007).

#### Experiment 1: Biased target locations

Trials in Experiment 1 started with a red fixation dot (0.8° across; 3 cd/m2) appearing at the center of the screen (Figure 1A). Participants were asked to fixate the dot and then press a mouse button to confirm fixation. 1700 to 1900 ms later the fixation point disappeared, and a red saccade target (1.2° across) appeared for 50 ms either 17° to the right or to the left. Participants moved their eyes as quickly as possible and around that time a white
vertical bar (0.5° by 87°; 41 cd/m²) was flashed for about 8 ms either 6.8° left or right of the saccade target (we used the minimum of positions to estimate perisaccadic compression so that sufficient numbers of trials would test each flash location in all conditions). Four-hundred milliseconds (500 ms in Experiments 2a and 3) after onset of the saccade target a mouse cursor appeared in the center of the screen and participants were asked to indicate the perceived location of the flash.

Importantly, as participants were informed, the location of saccade targets (and presumably of spatial attention) was biased such that in a series of four or more blocks, consisting of 80 trials each, saccade targets on the right side were more frequent (80%) than targets on the left (20%) while in other series targets on the left side were more frequent than targets on the right. This way, participants spent more time looking to one side than the other (of course, they also made more saccades in one than the other direction but only during trials; between trials they moved their eyes back to the central fixation point). In total, each participant performed about 4 test series.

Experiments 2 and 2a: Attentional cueing

Experiment 2 was similar to Experiment 1 (Figure 1B), except saccade target locations were not biased. However, to bias attention a black-outlined arrow inside a white square (3.2° across) appeared 200 to 600 ms after trial onset. It pointed left or right with equal probability and informed participants about the location at which the saccade target would appear 400 ms later with 80% probability (valid trials). Twenty percent of the arrows pointed in the opposite direction (invalid trials).

Because 400 ms stimulus asynchrony (SOA) might have been too short to process the arrow cue (though, see Posner et al., 1980), we conducted control Experiment 2a, with an SOA of 800 ms. Also, we used flash locations that were equally spaced apart (−22.5°, −7.5°, 7.5° and 22.5°) and saccade targets at ±15° to see whether this would increase the amount of perisaccadic shift. Shifts in Experiments 1 and 2 were rather small perhaps because flash locations were unevenly distributed and this might have eliminated an adaptation effect in Experiment 2.

Participants performed 20 or more blocks of trials in 2 or 3 sessions.

Experiment 3: Biased eye in head position without target bias

As one potential problem with Experiment 1, some flash locations were presented more often than others so that participants had more opportunity to learn those locations. To avoid this, we repeated Experiment 1 (with the saccade targets and flash locations from Experiment 2a), except saccades to the left and right side were equally likely. To bias eye-in-head position, each trial was preceded by a 5 s adaptation period during which participants visually tracked the saccade target as it jumped every 500 ms back and forth between the screen center and one side: 15° in some blocks and −15° in other blocks. Participants performed 3 or 4 blocks worth of 80 trials for each direction.

Data analysis

Eye movement data and mouse click responses (i.e., perceived flash locations) were stored on a PC for off-line analysis. Saccade reaction times were determined using a velocity criterion (20°/s). In addition, trials were visually inspected for appropriate fixation and saccade performance.
Trials containing eye blinks, saccades during the fixation interval, directional saccade errors, hypometric saccades (<50% of the final eye position), and trials with delayed saccade latencies (>1000 ms) were discarded. An average of 1361 (±435 SD) trials per participant were collected for Experiments 1 and 2. An average of 1819 (±433 SD) trials were collected for Experiment 2a, and for Experiment 3 the average was 532 (±92 SD).

Saccades and perceived flash locations for trials to the left and right side were then converted into a common reference frame. To examine the influence of saccades on spatial perception, we sorted perceived flash locations according to when the flash appeared relative to saccade onset. To average these data for each participant separately we then used a gliding window with a width of 42 ms (84 ms in Experiment 3) and a step size of 2 ms, and we repeated this procedure to further smooth the data.

**Results**

Figures 2 and 3 present perceived flash locations as a function of time centered on saccade onset. Figure 2 presents individual data sets from typical participants, Figure 3 averages across participants. In both experiments perceived flash location changed around the time of the saccade much like this has been described before (Bischof & Kramer, 1968; Bockisch & Miller, 1999; Honda, 1991; Lappe et al., 2000; Matin & Pearce, 1965; Morrone et al., 1997; Ross et al., 1997; Schlag & Schlag-Rey, 1995). Perhaps for stimulus-specific reasons, participants also underestimated locations of flashes presented on the outer side of the saccade target (upper panels in Figures 3A and 3D), and flash locations between fixation point and target were overestimated when appearing after the saccade (lower panels in Figures 3A and 3D). Of main interest, however, is that biasing target locations in Experiment 1 influenced the location at which the flashes were perceived (Figure 3A). Figures 3B and 3C show that at both flash locations the effect was significant and strongest before saccade onset, though there also was evidence for a time-constant effect (lower panel Figure 3B). Attentional cueing in Experiment 2 had no such effect (Figures 3D–3F). To examine the effect further we extracted two measures of misperception from the perceived-location function, perisaccadic shift and perisaccadic compression of flash locations (Lappe et al., 2000; Morrone et al., 1997).

**Perisaccadic shift**

To study perisaccadic shift we averaged perception of flashes across the two possible flash locations (Lappe et al., 2000). In both experiments participants perceived

![Figure 2. Perceived flash locations as a function of time. Saccade onset is at 0 ms. (A and B) Experiment 1 data for two participants. (C and D) Experiment 2 data for two participants. Blue: data for the frequent and valid conditions, red: data for the infrequent and invalid conditions.](https://jov.arvojournals.org/pdfaccess.ashx?url=/data/journals/jov/933553/)
flashes as shifted in saccade direction when the flashes appeared up to about 50 ms before saccade onset, and participants perceived a smaller shift in the opposite direction about 50 ms after saccade onset (Figures 4A and 4D). However, only when target locations were biased (Experiment 1) participants showed more shift during infrequent trials than during frequent trials. The difference peaked around 40–10 ms before saccade onset and

Figure 3. Perceived flash locations as a function of time. Saccade onset is at 0 ms. (A–C) Experiment 1. (D–F) Experiment 2. Upper panels in (A) and (D) present perceived locations for flashes beyond the saccade target; lower panels present perceived locations for flashes between target and initial fixation point. Dashed lines indicate true flash locations. Blue: data for the frequent and valid conditions, red: data for the infrequent and invalid conditions. (B and E) Difference curves between conditions in (A) and (D), respectively. (C and F) P-values of point-by-point t-tests comparing experimental conditions. Thick lines: p-values for data in upper panels, thin lines: p-values in lower panels in (A) and (D), respectively. Areas under curves = ±1 SE.
Figure 4. Perceived perisaccadic shift as a function of time. Saccade onset is at 0 ms. (A–C) Experiment 1. (A) Perceived shift of flash locations during frequent (blue) and infrequent (red) trials. (B) Difference curve between infrequent and frequent shift data. (C) $P$-values of point-by-point $t$-tests comparing infrequent and frequent shift data. (D–F) Experiment 2. (D) Perceived shift during valid (blue) and invalid (red) trials. (B) Difference curve between invalid and valid shift data. (C) $P$-values of point-by-point $t$-tests comparing invalid and valid shift data. Areas under curves $= \pm 1 SE$. 
Figure 5. Perceived perisaccadic compression of flash locations as a function of time. Saccade onset is at 0 ms. (A–C) Experiment 1. (A) Perceived compression during frequent (blue) and infrequent (red) trials. (B) Difference curve between infrequent and frequent compression data. (C) $P$-values of point-by-point $t$-tests comparing infrequent and frequent compression data. (D–F) Experiment 2. (D) Perceived compression during valid (blue) and invalid (red) trials. (B) Difference curve between invalid and valid compression data. (C) $P$-values of point-by-point $t$-tests comparing invalid and valid compression data. Areas under curves $\pm 1$ SE.
amounted to roughly 40% of presaccadic shift (Figure 4B). Point-by-point t-tests produced p-values smaller than 0.05 for 69 consecutive time units (Figure 4C), which is significant according to our criterion of 12 or more consecutive p-values smaller than 0.05 (Foxe, McCourt, & Javitt, 2003). The difference could not be explained by the smaller number of trials in the infrequent condition. To show this we repeated the analysis for the frequent condition 500 times, each time including only a randomly drawn quarter of the frequent trials but this had no effect on shift.

The difference in shift could have occurred because biased target locations in Experiment 1 required participants to move their eyes mostly to one side, or because the target bias let participants move their attention to that side and anticipate the target there (though, Awater & Lappe, 2004 have found no evidence for an influence of anticipation). To disentangle the two possibilities, Experiment 2 used arrow-shaped cues to bias attention while keeping saccades to the left and right side equally likely. However, perceived shift during validly and invalidly cued trials was largely identical (Figures 4D–4F), suggesting that biasing eye-in-head position but not attention influenced perisaccadic shift. This is first evidence that perisaccadic shift is associated with an EPS, probably independent of oculomotor control.

Perisaccadic compression

Did similar influences occur for perisaccadic compression? Though this second measure of perisaccadic misperception was not the main focus of our study, we calculated standard deviations of perceived flash locations to quantify compression (Lappe et al., 2000). Standard deviations were particularly reduced (and perceived flash locations maximally compressed) at saccade onset. However, perisaccadically there were no differences between experiments (Figures 5A vs. 5D) or conditions (Figures 5B, 5C, 5E, and 5F). At other times we did find trends for differences: both experiments showed differences between conditions some 100 ms before saccade onset. But these probably reflect fringe effects because we timed flashes so that they likely coincided with saccade onset and seldom 100 ms earlier, especially during the infrequent and invalid conditions. Furthermore, Figure 5F suggests a difference around ~50 ms but this was before perisaccadic compression began (Figure 5D), and it was only marginally significant because only 6 consecutive p-values were smaller than 0.05.

Precision

To see whether conditions differed in terms of localization precision, we calculated binwise standard deviations of perceived flash locations for each location separately and then averaged across locations. Imprecision peaked before saccade onset (Figures 6A and 6D) consistent with previous reports (Binda et al., 2007). In addition, precision was worse before than after the saccade perhaps because eye position was further away from flash locations or because locations had to be remembered longer. We also observed that precision was slightly worse for infrequent than for frequent trials (Figure 6B), though only after the saccade did the difference reach significance (as marked by the gray area in Figure 6A). For Experiment 2 we found no systematic differences between valid and invalid trials (Figures 6D and 6E). Furthermore, we found no evidence that precision was related to the increase in perisaccadic shift in Experiment 1 (or the lack thereof in Experiment 2; Figures 6C and 6F). That is, at no time did we find significant correlations with perisaccadic shift (dashed lines: point-by-point correlations of precision differences with shift differences; dotted lines: correlations for precision differences averaged across time; solid line in Figure 6C: correlations for precision differences averaged across the post-saccadic time during which precision differences were significant, i.e., the gray area in Figure 6A). In summary, we found no evidence for a connection between the effect on perisaccadic shift and localization precision, though it is possible that our data lacked statistical power. Further testing might be required in the future.

Saccade latencies

Because validity in Experiment 2 had no effect on perisaccadic misperception, we wondered whether our manipulation of attention had been insufficient. However, we found that in both experiments saccade latencies differed as expected: in Experiment 1 saccades were significantly faster in the frequent than in the infrequent condition (e.g., here we present group statistics based on individual median latencies: cueing effect = 16.5 ms, t(8) = 4.21, p = 0.0029; first pair of bars in Figure 7A), and in Experiment 2 valid cues resulted in faster responses than invalid cues (cueing effect = 34.6 ms, t(7) = 4.55, p = 0.0026; first pair of bars in Figure 7E), suggesting that Experiment 2 manipulated attention in a similar way as Experiment 1.

One difference between experiments becomes obvious in the distribution of saccade latencies (here we converted the discrete distribution of latencies into a continuous probability density function; we replaced each latency by a Gaussian kernel with standard deviation = 10 ms, summed over all kernels and then normalized the curve; Figures 7B and 7F). Only in Experiment 2 did valid trials yield clearly more saccades with latencies of 100 ms or less than invalid trials. However, we found that excluding these fast saccades had no influence on perisaccadic shift or compression. It slightly
changed group averages of latencies but the cueing effect remained (cueing effect = 25.6 ms, \( p = 0.013 \)).

With or without exclusion of fast saccades, the two experiments also differed in latency variability: in Experiment 1 latencies of infrequent saccades were more variable than latencies of frequent ones. For example, the interquartile range was larger (\( t(8) = 2.51, p = 0.036; \) second pair of bars in Figure 7A), and the continuous probability density function for infrequent trials was more spread with a smaller peak than the frequent curve (\( t(8) = 2.69, p = 0.027; \) third pair of bars in Figure 7A). By contrast, attentional cues in Experiment 2 had no such effect when including fast saccades (\( t's \leq 0.62, p's \geq 0.556; \) second and third pair of bars in Figure 7E) and remained insignificant when excluding fast saccades, though the peaks showed a similar trend as those in Experiment 1 (peak averages in Figure 7E appear to disagree with peaks in Figure 7F, but the latter graph confounds variability within and across participants).

Does the more prominent effect on the spread of latencies in Experiment 1 explain why perisaccadic shift changes there but not in Experiment 2? Larger variability in saccade latencies might make saccade onsets less predictable, and eye position information might become less precise in time, resulting in more perisaccadic shift, just as we observed for infrequent trials in Experiment 1. If so, people with more variable saccade latencies should show more shift.

To test this possibility we conducted a series of analyses in which we correlated various measures of saccade latencies with various measures of perisaccadic misperception. However, we found no relationship. For example, in Figure 7C we correlated differences in perceived shift associated with biased target locations (infrequent minus frequent trials) with differences in latency peaks (thick solid curve) and with differences in interquartile ranges (thin solid curve). Furthermore, we correlated differences in median latencies with differences in perceived shift (dotted curve) and in perceived compression (dashed curve). None of the correlations reached significance around the time of the saccade (Figure 7D). Also, we found no significances when repeating the analyses for the frequent and infrequent conditions separately. Lastly, we conducted equivalent analyses for attentional cueing in Experiment 2 (Figures 7G and 7H). But again, we found no association between saccade latencies and perisaccadic perception.

Was the SOA of 400 ms too short for a cueing effect to fully develop and influence perisaccadic shift? In Experiment 2a we prolonged SOA to 800 ms. Averaged across participants the cueing effect was of about the same magnitude as before (31.7 ms, \( t(2) = 6.12, p = 0.026; \) Figure 8A). Perisaccadic shift was more prominent than before, perhaps because flash locations were equally spaced, but again we found no cueing effect on perisaccadic shift. Only one participant showed such a trend (Figure 8E), the other two showed, if any, the opposite trend (Figures 8B–8D).

### Saccade amplitudes and velocities

Do the perceptual effects in Experiment 1 occur because biasing target locations changes oculomotor control? Performing more saccades in one direction might cause changes in saccade control to the opposite side and, indirectly, in perisaccadic perception (e.g., Bahcall & Kowler, 1999). However, we found no evidence for changes in saccade performance that could explain the perisaccadic effects.

In Experiment 1 saccades were influenced by flashes when they appeared before saccade onset: amplitudes were larger and velocities higher for flashes presented beyond the target than between target and fixation point (Figures 9A and 9B, and thin solid and dotted curve in Figure 9C, respectively), as reported before (Awater & Lappe, 2006). But frequency of saccade targets had no effect (thick solid and dashed curve in Figure 9C, respectively).

Effects in Experiment 2 were somewhat different. Just like before, saccade amplitudes and velocities varied with flash location for flashes before saccade onset (Figures 9D and 9E, and thin solid and dotted curve in Figure 9F, respectively). Yet in addition, the independent variable ‘cue validity’ had an effect; saccades after invalid cues tended to be larger and faster than validly cued ones (Figure 9F, thick solid and dashed curve). This could be due to a larger proportion of fast, but imprecise, saccades in the valid condition. However, we found the same metrical differences when we repeated the analysis without fast saccades (latencies < 100 ms). Though for now we cannot explain this difference, it appears unlikely that it explains the lack of a difference in perisaccadic shift in Experiment 2. If at all, larger amplitudes should have resulted in more shift. Though, the relationship between shift and amplitude appears to be rather weak (van Wetter & van Opstal, 2008).

Lastly, to examine potential changes in main sequence in the two experiments we plotted saccade velocities as a function of amplitude. The relationship was well approximated by linear regression functions, but slopes and offsets of these functions did not differ significantly between frequent and infrequent, or valid and invalid saccades (\( t's < 0.3, p's > 0.77 \)). Likewise, we found no differences when repeating the regression analyses with log transformed saccade amplitudes and when repeatedly subsampling random quarters of frequent and valid trials, respectively.

In sum, perisaccadic perception altered by target probability was not associated with changes in oculomotor control either. This further supports the idea that perisaccadic shift is governed by an EPS from non-motor sources.
Figure 6. Localization precision as a function of time. Saccade onset is at 0 ms. (A–C) Experiment 1. (A) Precision during frequent (blue) and infrequent (red) trials. The gray area indicates the time period during which conditions differed significantly. (B) Difference curve between infrequent and frequent precision data. (C) Correlation coefficients of differences in precision data with differences in perisaccadic shift data. Dashed curve: time-point-by-time-point correlations, dotted curve: correlations for precision differences averaged across time with shift differences at all time points, thick solid curve: correlations for significant precision differences with shift differences at all time points. (D–F) Experiment 2. (D) Precision during valid (blue) and invalid (red) trials. (B) Difference curve between invalid and valid Precision data. (C) Correlation coefficients as in (C). Note that no solid curve was included because differences between conditions were insignificant at all times. Areas under curves = ±1 SE.
Figure 7. Saccade latencies. (A–D) Experiment 1. (A) Medians, interquartile ranges and peak probability densities of saccade latencies averaged across participants for frequent (blue) and infrequent (red) trials. (B) Probability density curves of saccade latencies for frequent (blue) and infrequent (red) data. (C) Point-by-point correlations between measures of perisaccadic misperception (infrequent − frequent) and measures of saccade latencies (infrequent − frequent). (D) Point-by-point p-values of the correlations in (B). (E–H) Experiment 2. (E) Medians, interquartile ranges and peak probability densities of curves: perceived shift and interquartile range; dotted curves: perceived shift and median latencies; dashed curves: perceived compression and median latencies. Error bars = ±1 SE.
Is there other evidence that a non-motor EPS affects perisaccadic shift? Several studies have reported that neural activity carrying non-motor eye position information shows a history effect of preceding saccades (Nakamura et al., 1999; Schlag-Rey & Schlag, 1984; Tanaka, 2007). A similar short-term history effect might show up in perisaccadic shift if shift is caused by a non-motor EPS.

In search for a short-term effect, we repeated the analysis of perisaccadic shift perception in Experiment 1 but pooled all frequent and infrequent trials and instead sorted them into two new categories. A trial was called “one-back same” if it was preceded by a trial that required a saccade to the same target, and other trials were called “one-back different” because the preceding trial required a saccade in the opposite direction (a few trials at the start of test blocks were removed from the analysis). For these

Figure 8. Experiment 2a. (A) Saccade latencies. (B) Perceived flash locations as a function of time for one participant. (C–E) Perisaccadic shift as a function of time for the three participants. S7 was an author (VS). Gray areas indicate time periods during which conditions were significantly different. Blue: valid condition, red: invalid condition.
new data sets we determined the time at which presaccadic shift on average peaked (inside a time window of 50 ms before saccade onset), and for that time we then recorded shift in individual observers. Next, we again pooled all trials and sorted them into “two-back same” and “two-back different” data, that is, we separated trials depending on whether the target two trials earlier was the same or different (regardless of the target one trial earlier). We repeated the analysis for all n-back until 20-back; and we obtained equivalent n-back data for Experiment 2 while collapsing across valid and invalid trials.

We found that the “n-back same” curve in Experiment 1 settles at a lower level of presaccadic shift than the “n-back different” curve (Figure 10A), but not in Experiment 2 (Figure 10C). This is expected because in Experiment 1 the “same-curve” data contain more of the original “frequent” than “infrequent” trials (a trial counts as “same” either if that trial and the n-back trial are both “frequent” trials, $p = 0.8 \times 0.8 = 0.64$, or both “infrequent” trials: $p \approx 0.2 \times 0.2 = 0.04$) while the “different-curve” data contain equal portions of both categories (a trial counts as “different” if it belongs to the original “frequent” and the n-back trial belongs to the “infrequent” category or vice versa: $p \approx 0.8 \times 0.2 = 0.2 \times 0.8 = 0.16$).

Surprisingly however, for 1-back the “n-back same” curve showed more (not less) shift relative to the “different” data (Figures 10A and 10C). This short-term effect was significant for Experiment 2 ($t = 2.45, p = 0.044$). It was not significant for Experiment 1 ($t = 0.93, p = 0.282$), because it was eclipsed by the unequal proportion of frequent and infrequent trials as mentioned. To remove this confound we fit exponential functions to individual “same” and “different” curves ($f(i\text{-back}) = \text{asymptote} * (1 - b * \exp(-iback/c))$) and subtracted the asymptotes from the 1-back data. Afterwards the short-term effect was significant ($t = 3.14, p = 0.014$).
So, interestingly, preceding trials have a short-term influence on subsequent ones that is independent of attentional cues in Experiment 2 and that works opposite to the longer-term saccade frequency effect observed in Experiment 1. Visually inspecting Figure 10C suggests that in Experiment 2 the short-term effect disappears after the first trial. In Experiment 1 the effect faded more slowly (Figure 10A) perhaps reflecting an interaction between the two mechanisms underlying long- and short-term adaptation.

To try to find the point at which one effect would switch into the other, we repeated the n-back analysis with longer chains of “same” and “different” trials. But, pairs and triplets of “same” or “different” trials still produced about the same short-term effect, and longer chains were too rare to be analyzed in this way.

Finally, we tested whether the short-term effect on perisaccadic shift was associated with any measures of saccade performance. An analysis of saccade latencies (Figures 10B and 10D) did not mirror the short-term effect for shift, which could potentially have explained increased shift. “Same” data in Experiment 1 had shorter latencies than “different” data, again, because the proportions of original “frequent” and “infrequent” trials differed. But there was no evidence for a short-term effect reversing the overall trend, that is, preceding saccades in the same direction did not slow down latencies as reported by some (e.g., Tanaka & Shimojo, 2000), instead they slightly reduced latencies. Further research would be necessary to confirm whether this is consistent with similar findings that preceding saccades in the same direction reduce
latencies (Dorris et al., 2000), which is contrary to Tanaka and Shimojo (2000) but might happen after large numbers of trials (Fecteau & Munoz, 2003). Also, saccade latencies for “same” and “different” data were not correlated with corresponding measures of perisaccadic shift. And we observed no reliable n-back effects on saccade amplitudes and velocities.

**Experiment 3**

Is it possible that the adaptation effects are purely visual? For example, the long-term effect could be related to flash locations on the frequent side being trained more than on the infrequent side. Therefore, in Experiment 3 we presented flashes equally often at all locations, and we
biased eye-in-head positions through an adaptation period before each trial. Still, all three participants showed perisaccadic shift that was more pronounced for the unadapted side than the adapted side (red vs. blue lines in Figures 11A, 11D, and 11G), and the difference was most prominent around the time of the saccade (Figures 11B, 11E, and 11H). At the same time, we found no evidence that the adaptation period served as an attentional cue, biasing attention to the same or opposite side (Figures 11C, 11F, and 11I; average difference in saccade latencies across participants = 0 ms).

Discussion

Visual stimuli briefly presented around the time of a saccade are perceived as shifted in saccade direction, probably because it is difficult to tell when the retinas move relative to when retinal images move (Pola, 2004). To clarify whether the temporal uncertainty comes from vision or some sort of extraretinal eye position signal (EPS), here we examined how biased eye-in-head positions, attentional cueing, and trial history influence perisaccadic shift. We found evidence for adaptive mechanisms of perisaccadic shift on two different time scales. On a longer-term scale, while moving their eyes mostly to one side within a test session of ~30 min, participants perceived increased shift for saccades to the infrequent side. The increase was about 40% relative to shift during frequent trials in Experiment 1 and about 22% in Experiment 3 (though, in absolute terms it was larger there), and it was most pronounced for the time around the saccade. A trend for a time-constant offset was evident too but smaller (Figure 4B). This shows that our manipulation influenced perisaccadic perception in particular, suggesting that errors in time increased. Additional, rather unspecific changes in spatial perception might have occurred as well (Paap & Ebenholtz, 1976; Park, 1969). One possibility is that our experimental manipulations caused adaptation of neurons representing eye position or an effective shift in apparent midline. However, these effects could only explain the offset, not increased shift specifically for the time around the saccade.

Can the effect on perisaccadic shift be explained with a visual account of perisaccadic shift? Temporal delays and noise originating from the physiology of the visual system could add to perisaccadic misperceptions (Pola, 2004). It has even been argued that temporally noisy vision is the only cause of perisaccadic shift because shift does not increase with saccade amplitude (van Wetter & van Opstal, 2008). This finding is important as it suggests that temporal uncertainty in the EPS does not rise with saccade amplitude. It does not necessarily contradict previous findings that spatial uncertainty rises with saccade amplitude and other saccade metrics (Li & Matin, 1997; Niemeier, Crawford, & Tweed, 2003, 2007), because this reflects unsystematic errors in space, which alone would not cause errors in time and thus shift. Either way, it is possible that EPS had a consistent degree of temporal errors that caused shift.

In the present study changes in visual processing probably occurred because Experiment 1 stimulated the visual hemifield in which the frequent target was located more than the other field reducing contrast adaptation there. And both experiments biased attention, which should have increased contrast on that side. But it is unlikely that either of these contrary effects would have been more than subtle, while even substantial contrast changes do not change perisaccadic shift (Boucher, Groh, & Hughes, 2001; Michels & Lappe, 2004).

Still, solely visual errors could account for the Experiment 1 data: participants could have relied on vision only to practice localizing flashes and to reduce perisaccadic shifts on the frequent more than on the infrequent side (though, for flashes appearing before saccade onset participants would have had to refixate the center of the screen before responding). However, Experiment 3 showed the same difference in perisaccadic shift with unbiased flash locations. Of course, the adaptation period in Experiment 3 still biased saccadic targets to one side, but only relative to the head (relative to the line of sight targets on the left and right side were equally likely). Therefore, we cannot rule out that visual signals play a role, perhaps in the context of altered representations of space (also see below). However, the present data are difficult to explain with a purely visual account without changes in EPS. Instead, they could be interpreted in support of an EPS account of perisaccadic shift (Dassonville et al., 1992; Matin & Pearce, 1965). This way, our results would be consistent with a previous study that found very similar perisaccadic misperceptions for pro- and antisaccades, in support of a non-visual mechanism (Awater & Lappe, 2004).

Which source of eye position information might be involved in increased perisaccadic shift? In principle, there are two possibilities. Perception could extract eye position information from some stage of oculomotor control including programming of associated shifts of attention as long as the information re-enters the cortex. For example perception could extract eye position information from the oculomotor output from frontal eye fields (FEF), which directly feeds back into cortex (Hamker, 2005; Moore & Armstrong, 2003). Alternatively, perception could rely on eye position information arriving in cortex from structures that are not directly involved in oculomotor control, such as thalamic nuclei passing on eye position information from the superior colliculus (Sommer & Wurtz, 2002; Tanaka, 2007).

If perisaccadic shift was governed by FEF output (or output from some other cortical eye field; Lynch & Tian, 2006), changes in output should change oculomotor
performance together with perisaccadic perception. For example, one target location being more frequent or being predicted by a cue should allow participants to prepare saccades in that direction (Connolly, Goodale, Goltz, & Munoz, 2005; Connolly, Goodale, Menon, & Munoz, 2002; Thompson, Bichot, & Sato, 2005a; for a similar mechanism downstream from FEF: Dorris & Munoz, 1998), and to reduce saccade latencies for that target. This we did observe in Experiments 1, 2, and 2a, suggesting that our manipulations were sufficiently strong. However, preparatory activity and shortened delays should also reduce temporal uncertainty about when the eyes start moving, this way reducing perisaccadic shift for prepared saccades or increasing shift for unprepared saccades. Alternatively, one could postulate that perception confuses attentional shifts with saccades if it made poorly calibrated use of EPS, but then there should be more shift for expected targets locations. Here we found that perisaccadic shift was more pronounced for unexpected, infrequent targets than for frequent ones in Experiment 1. Note that this does not contradict a previous observation that biased target locations have no effect on perisaccadic perception because that study compared location frequencies of 100% and 50% only, so there was no infrequent condition with a target probability of 20% (Awater & Lappe, 2004). In contrast to Experiment 1, in Experiments 2 and 2a we did not find an equivalent increase for invalidly versus validly cued trials consistent with previous observations that perisaccadic perception does not differ for target predictability or pro- and antisaccades (Awater & Lappe, 2004; Bockisch & Miller, 1999). Also, perisaccadic misperceptions did not correlate with saccade latencies or latency variability in the first and the second experiment which argues against the possibility that cueing in Experiment 2 involved specialized attentional mechanisms. Furthermore, we observed no changes in other oculomotor measures that could explain the increase in shift. Finally, Experiment 3 produced changes in perisaccadic shift equivalent to those observed in Experiment 1 without changing saccade latencies. In sum, we failed to find evidence that oculomotor control signals serve as an EPS causing perisaccadic shift.

Could our experiments have modified two independent FEF outputs? Could target predictability in both experiments have modulated one output controlling saccade latencies, while only the first experiment modulated an EPS output causing misperceptions? FEF contains at least two subsystems (Sato & Schall, 2003; Thompson, Biscoe, & Sato, 2005b), one involved in visual selection much like a salience map and one involved in eye movement selection and control. Given this, it should be the latter subsystem governing both latencies and eye position information. Even if FEF contained yet another system that outputs a separate EPS, this would only support the idea that perisaccadic perception derives eye position from a signal not directly involved in oculomotor control.

Why does this EPS change with target probability biased to a frequent side? It is difficult to explain with biased saccade directions because between trials participants refixed the center of the screen. However, eye-in-head position was biased because participants spent more time looking to one side than the other. Maintaining a lateral eye-in-head position alters spatial perception (Park, 1969) and is an extraretinal factor involved in prism adaption (Paap & Ebenholtz, 1976). Here we argue that biased eye position, perhaps in combination with saccades, also impacts temporal precision and accuracy of the coding and processing of eye position information, perhaps for one or more of the following reasons.

First, efference copies of oculomotor commands projected from thalamus to cortical areas (Sommerr & Wurtz, 2002; Tanaka, 2007) might become less accurate or more sluggish in time when other eye-in-head positions are trained. Second, eye muscle proprioception might change, given that biased eye postures cause eye muscle potentiation (Paap & Ebenholtz, 1976). However, whether proprioception provides eye position information useful at the time of the saccade has been doubted (e.g., Guthrie, Porter, & Sparks, 1983). If at all, it might contribute only little to spatial perception (Bridgeman & Stark, 1991) and then it would be difficult to reconcile with relatively large effects on perisaccadic shift. What is more, misperceptions occur for stimuli presented before saccade onset while proprioceptive information is generated afterwards. Proprioception could still influence the past, but only if perception postponed its computations (similar to the postdiction model of the flash-lag effect, Eagleman & Sejnowski, 2000). In principle such delays do occur (Lappe et al., 2000; Watanabe, Noritake, Maeda, Tachi, & Nishida, 2005), but the present speculations require further investigation. As a third possibility, with biased eye postures cortical processing of EPS could trail off in temporal precision and accuracy for the untrained side. It is possible that in that case the effect would reside in an altered representation of space that combines extraretinal information with retinal signals with increased temporal sluggishness.

While these head-centered changes occur on a longer-term scale, we also found evidence for a short-term influence of trial history. In Experiment 1 as well as 2 participants saw increased perisaccadic shift when the preceding trial had required them to fixate the same eye-in-head position (and decreased shift for targets on the opposite side). Also possible is that increased shift was associated with the eye movements in the opposite direction during re-fixation that preceded the trial.

In Experiment 2 the short-term effect faded quickly. In contrast, in Experiment 1 it seemed to disappear after six or more trials, though this could have resulted from an interaction between the short-term and the longer-term effect, potentially indicating that in part the same neural structures are involved. Furthermore, we found no direct relationship to attention or oculomotor control. All in all,
our data could indicate that the effect is the perceptual correlate of the dynamic response patterns of thalamic and parieto-occipital neurons that carry non-motor eye position information (Nakamura et al., 1999; Schlag-Rey & Schlag, 1984; Tanaka, 2007). If so, it would provide a second line of evidence in support of non-motor mechanisms of perisaccadic perception. However, further research is necessary to confirm this potential connection between perception and physiology.

In contrast to these influences on perisaccadic shift, we failed to modulate perisaccadic compression—complementary to other studies showing influences of contrast and saccade velocity on compression but not shift (Michels & Lappe, 2004; Ostendorf et al., 2007). So one could argue that our results add to the notion that shift and compression are at least partially independent phenomena. However, this conclusion is premature because our paradigm was not optimally designed to study compression; it did not use a post-saccadic reference, and usually perisaccadic compression is estimated based on more and a larger range of flash locations (Lappe et al., 2000; Morrone et al., 1997). Therefore, further research will be necessary to investigate the role of eye posture on compression.

Conclusions

To conclude, in the present study we found evidence that perisaccadic shift is governed by an eye position signal that is not related to oculomotor control and that shows plasticity on different time scales as a function of eye positions and movements. Our data appear to suggest a non-motor system that constantly calibrates and adapts itself to compensate for changes in oculomotor (Goldstein & Robinson, 1986) and muscle systems (Paap & Ebenholtz, 1976) so as to reconstruct a coherent and stable percept of the outside world. Future research will explore the relationship between the effects reported here and changes in spatial processes observed after prism adaptation such as its ability to ameliorate spatial neglect (Rosetti et al., 1998).

Acknowledgments

The authors thank Matt Keough, Ada Le, and Boge Stojanoski for comments on an earlier version of the manuscript. This work was supported by grants from the Natural Sciences and Engineering Research Council of Canada, the Canada Foundation for Innovation, and the Ontario Innovation Trust. A.C. Kis was supported by a postdoctoral fellowship of the Academic Initiatives Fund of the University of Toronto.

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