

Interference during eye movement preparation shifts the timing of perisaccadic compression

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Our perception of the surrounding environment remains stable despite the fact that we frequently change the retinal position of input by rapid gaze shifts (saccades). There is a long-standing debate whether visual stability depends on an active mechanism using an efference copy of the impending saccadic motor command. Behavioral studies showing changes in perception around the time of saccades are consistent with a predictive mechanism, but previous studies of perceptual effects in humans confounded saccade programming with the resulting physical eye movement. In three experiments, we used a saccadic inhibition (SI) paradigm to delay saccadic onset while participants were performing a perisaccadic localization task. As expected, the perceived position of the probe stimulus was systematically biased (compressed) toward the saccadic goal, already during the presaccadic interval. In the SI condition, the localization error was shifted in time, in line with it following saccade intention rather than execution. The pattern was not the consequence of the probe being captured by the timing of the flashed distractor, but depended instead on the delay in saccadic onset time caused by SI. Importantly, the same configurations of perceptual probes presented with a flashed backward mask when participants maintained fixation did not lead to similar localization errors as saccade trials. This pattern of results is consistent with an active, sensorimotor explanation for perisaccadic mislocalization and, more generally, theories emphasizing the role of motor prediction in visual stability.

Introduction

A basic question in visual neuroscience is why we perceive a stable world despite frequent saccadic eye movements that shift the position of objects on the

retina. One hypothesis is that stability is maintained across saccades by means of a predictive efference copy of the impending motor command (also known as corollary discharge) that it is sent back to cortical areas informing about the incoming change in the eye position (Balslev, Himmelbach, Karnath, Borchers, & Odoj, 2012; Castet, Jeanjean, & Masson, 2002; Ibbotson & Cloherty, 2009; Sherrington, 1900; Sperry, 1950; von Helmholtz, 1866/1924; Von Holst & Mittelstaedt, 1954; Wurtz, Joiner, & Berman, 2011). Neurophysiological recordings showed that there is a precise pattern going upstream from the superior colliculus (SC) to the frontal eye field (FEF) and passing by the mediodorsal thalamus that is involved in carrying this efference signal (Sommer & Wurtz, 2006). Direct stimulation of this circuit causes deficits in tasks that require updating of the visual information, such as double step saccades (Becker & Jürgens, 1979). Moreover, in the SC, the FEF and the lateral intraparietal area (LIP), a preparatory signal for saccades has been recorded. Neurons in these areas start their responses prior to saccadic onset when a stimulus will be located in the postsaccadic receptive field (predictive remapping: Duhamel, Goldberg, Fitzgibbon, Sirigu, & Grafman, 1992; Nakamura & Colby, 2002; Walker, Fitzgibbon, & Goldberg, 1995). More recently, it has been proposed that the preparatory activity in the FEF converges toward the saccadic goal from all nearby locations in the visual field (Zirnsak, Steinmetz, Noudoost, Xu, & Moore, 2014). Nonetheless, in both circumstances these active mechanisms have been proposed to play a critical role in maintaining visual stability (e.g., Hamker, Zirnsak, & Lappe, 2008; Zirnsak, Gerhards, Kiani, Lappe, & Hamker, 2011; Zirnsak et al., 2014).

Citation: Buonocore, A., & Melcher, D. (2015). Interference during eye movement preparation shifts the timing of perisaccadic compression. *Journal of Vision*, 15(15):3, 1–16. doi:10.1167/15.15.3.

An interesting behavioral phenomenon that has been linked to visual stability is that stimuli presented in darkness are perceived in the incorrect location (mislocalized) when displayed around the time of an eye movement (Bockisch & Miller, 1999; Dassonville, Schlag, & Schlag-Rey, 1995; Honda, 1985, 1989, 1991; Matin, Matin, & Pearce, 1969; Matin, Matin, & Pola, 1970; Miller, 1996; Shlag & Schlag-Rey, 1995). Typically, the perceived location is compressed towards the saccadic goal. This phenomenon is referred to as perisaccadic mislocalization or compression (Honda, 1999; Lappe, Awater, & Krekelberg, 2000; MacKay, 1970; Morrone, Ross, & Burr, 1997; Ross, Morrone, & Burr, 1997; Zimmermann, Fink, & Cavanagh, 2013; Zimmermann, Born, Fink, & Cavanagh, 2014). In these localization paradigms, participants are required to make an eye movement toward a lateralized target. A probe is then presented either between the current fixation and the saccadic goal or in a position more eccentric compared to the saccadic target. In conditions in which there are some visual references (not complete darkness), it has been consistently shown that the perception of the probe is shifted toward the target location (compression), even for flashes presented during the presaccadic interval (Ross et al., 1997). Overall, consistent compression curves are recorded starting from 50–60 ms before saccadic onset, peaking at the time of the saccade and persisting up to 30–40 ms thereafter. It has been suggested that the activity recorded in SC and FEF neurons prior to saccadic movements might be responsible for the error in the localization pattern observed behaviorally. For example, it has recently been shown that visual receptive fields in the FEF compress toward the saccadic target during the perisaccadic time period (Zirnsak et al., 2014). These studies suggest that eye movement preparation and the efference copy signal may underlie perisaccadic compression (Cicchini, Binda, Burr, & Morrone, 2013; De Pisapia, Kaunitz, & Melcher, 2010; Hamker et al., 2008).

In contrast to active, efference copy theories of visual stability and perisaccadic mislocalization, an alternative view proposes that passive mechanisms, such as interference or masking by the postsaccadic stimulus are involved in mislocalization (Castet et al., 2002; Dorr & Bex, 2013; Ibbotson & Cloherty, 2009; Maij, Brenner, Li, Cornelissen, & Smeets, 2010; Ostendorf, Fischer, Gaymard, & Ploner, 2006; Zimmermann et al., 2013; Zimmermann et al., 2014). For example, Ostendorf and colleagues (2006) showed that it was possible to generate similar shift in the perceived position of a briefly presented stimulus by simulating retinal input of the moving eye during fixation. The authors obtained this effect by quickly shifting the visual display in a similar manner to how the image would move on the retina while producing an eye movement. This result

suggests that mislocalization can be elicited even without an extraretinal signal. Similar findings were also found by Dorr and Bex (2013). The authors showed that it was possible to reduce visual sensitivity by shifting the visual input while participants were maintaining fixation, suggesting that the main role was played by the retinal speed rather than the extraretinal signal. Atsma, Maij, Corneil, and Medendorp (2014) recently reported that canceling a saccade reduces mislocalization, suggesting that the eye movement itself is a necessary prerequisite to mislocalize a probe stimulus.

In line with this idea, Zimmermann et al. (2013; Zimmermann et al., 2014) showed that compression effects could also be obtained in absence of eye movements. In particular, localization error arose when a visual probe was presented and subsequently masked by a large field transient lasting 50 ms, similar to the transient loss of information due to saccadic eye movements. The authors proposed that the compression effect can be explained as a consequence of interrupting and reconnecting the visual input, with similar mechanisms to what happens when we move our eyes. However, compression from a mask during fixation (Zimmermann et al., 2013; Zimmermann et al., 2014) would not seem to involve an active, efference copy mechanism, raising the question of whether motor prediction plays a role in saccadic compression.

A limiting factor in this debate has been that previous studies of perceptual and motor effects of saccades in humans either have confounded motor programming with the movement itself or have used artificial methods to simulate the saccadic visual input while the eyes were resting. In order to properly distinguish between an active (efference copy) and passive (retinal smear or postsaccadic masking/anchoring effects) mechanism it would be necessary to dissociate the intention to move the eyes with the actual movement itself. A novel approach to pursue this idea could be to make use of paradigms whereby a delay is introduced in the motor execution. Among the many “distractor” paradigms, one that has been shown to precisely interfere with eye movement onset is saccadic inhibition (SI). In SI paradigms, an unexpected visual transient inhibits the production of saccades between approximately 60 to 125 ms after the transient onset (Bompas & Sumner, 2011; Buonocore & McIntosh, 2008, 2012; Edelman & Xu, 2009; McIntosh & Buonocore, 2014; Reingold & Stampe, 2002), generating a consistent delay in saccadic reaction times (SRT). The momentary pause of the motor program induced by SI might then allow dissociation of the motor programming from saccadic execution. Contrary to standard distractor paradigms, SI has the power to introduce a highly reliable delay in each participant by calibrating the timing of the suppressor (i.e., the flash)

to the performance of the participants during no flash trials. This paradigm gives the considerable advantage of allowing us to time the secondary perceptual stimulus exactly during the time window of inhibition. A similar methodology was already employed to study the effect of SI on double-step saccades. By combining the two paradigms, we showed that temporally interfering with the first eye movement generated a pattern of spatial errors in the secondary eye movement, congruent with the prediction of an efference copy impairment (Buonocore & Melcher, 2015).

In the present study we combined the SI method with a perisaccadic localization paradigm to introduce a delay between intention and execution. One possible outcome would be that the efference copy would be passed intact from the SC or FEF to cortical areas (Wurtz et al., 2011) but that the saccade would be delayed (Munoz & Wurtz, 1993a, 1993b), causing a leftward shift in the mislocalization curve with respect to saccade onset (Figure 1A). An alternative hypothesis might be that the timing of the probe is captured by the flash onset. In this case, the probe would be associated with a lower localization error (i.e., as if the probe would have been presented earlier in time) shifting the localization curve rightward (Figure 1B). Finally, the SI delay might be already implemented in the efference copy itself, realigning the intention with the execution. In this case no shift would be recorded. In three experiments, we report a consistent shift in the perisaccadic localization error curves during SI trials in line with the first (motor intention), rather than the latter, hypothesis. These findings are consistent with an important role for motor planning (and efference copy) in compression effects, as they show that the time course of mislocalization is tied to intended saccade onset rather than the actual onset/execution of the eye movement itself (Figure 1A).

Materials and method

Participants

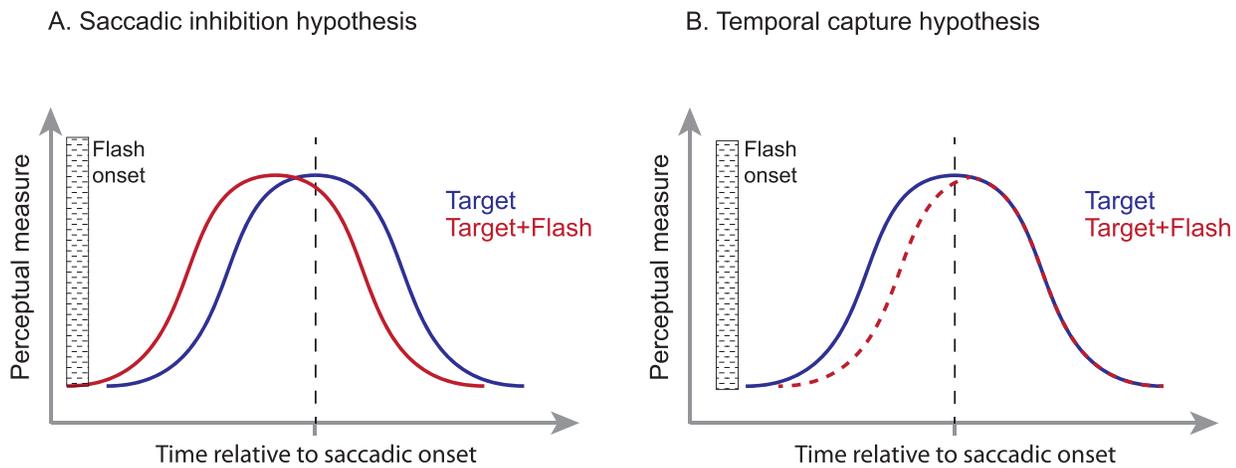
A total of 32 volunteers aged between 18 and 34 years participated in two experiments (Experiment 1, $N = 13$; Experiment 2, $N = 10$, two participants had also taken part in Experiment 1; Experiment 3, $N = 11$). All participants reported they were free from neurological and visual impairments. These experiments were conducted in accordance with the 1964 Declaration of Helsinki, and in accordance with the guidelines for the University of Trento Ethics Committee. All participants gave informed written consent and received a small monetary compensation for their time.

Apparatus and stimuli

Stimuli were white (107 cd/m^2) on a gray (10 cd/m^2) background presented on a 19-in. CRT monitor (1024×768 pixels) at 85 Hz (100 Hz in Experiment 3). In all the experiments, participants were seated with their head resting on a chin and forehead rest in order to reduce head movements. The eyes were horizontally and vertically aligned with the center of the screen at a distance of 60 cm. Eye movements were recorded with the EyeLink 1000 system (SR Research, Ottawa, ON, Canada; detection algorithm: pupil and corneal reflex; 1000 Hz sampling; saccade detection was based on a $30^\circ/\text{s}$ velocity and $9500^\circ/\text{s}^2$ acceleration thresholds). Perceptual responses were recorded with a standard mouse and keyboard connected to the display computer.

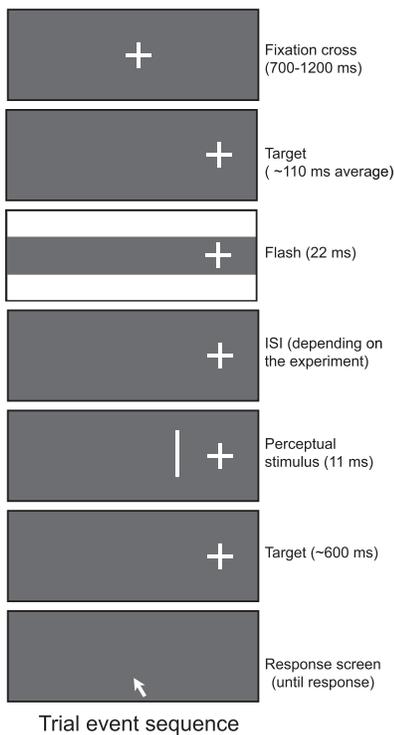
General procedure

The experimenter started each trial with a drift correction and a tone accompanying the onset of an approximately 0.50° fixation cross. In Experiments 1 and 2, the fixation cross was placed at the center of the screen while in Experiment 3 it was shifted 10° of visual angle to the left (Figure 1C, D, E). After a delay interval varying randomly between 500 and 1200 ms, the saccadic target was presented either to the left or the right of the visual field with equal probability (Experiments 1 and 2, Figure 1C) or 16.5° on the right (Experiment 3, Figure 1D and E). Participants were instructed to fixate the cross and to move their eyes to the target as soon as it appeared. In a preliminary baseline block, the target was presented without any distractor for 40 or 50 trials (for Experiments 1 and 2, respectively). The median baseline SRT was calculated from the baseline block in order to determine the timing of the flash and the perceptual stimulus (PS) in the main experiment. In Experiment 3, the median SRT was calculated online at the end of each trial and there was no preliminary block of target-only trials. For the flash, the onset time was calculated by subtracting a constant value from the median baseline SRT. The PS followed the flash onset by a constant delay (see experimental procedure for each experiment below). Thus, the timing of the flash was arranged so that the expected maximum dip of SI (approx. 90 ms after distractor onset) would coincide approximately with the peak of the baseline SRT distribution (for similar procedures, see Buonocore & McIntosh, 2012; Reingold & Stampe, 2002). In the main experiment, the target was presented either alone or with a flash, in a random order within each block of trials so that participants could not predict whether or not a flash would occur. A 5-point calibration on the horizontal and vertical axes was run at the beginning of each session and after three consecutive

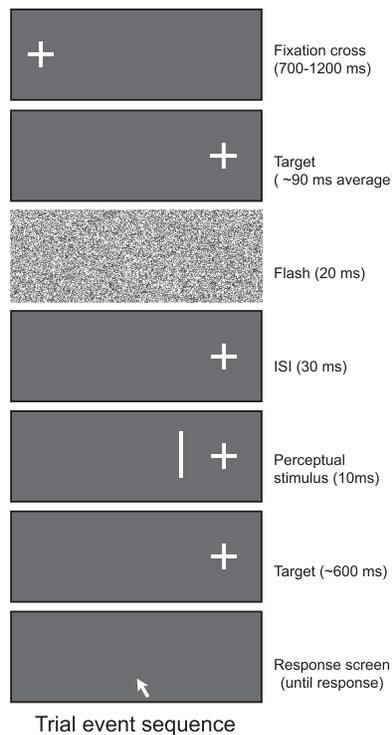


General methods

C. Experiment 1 and 2



D. Experiment 3: Saccade



E. Experiment 3: Fixation

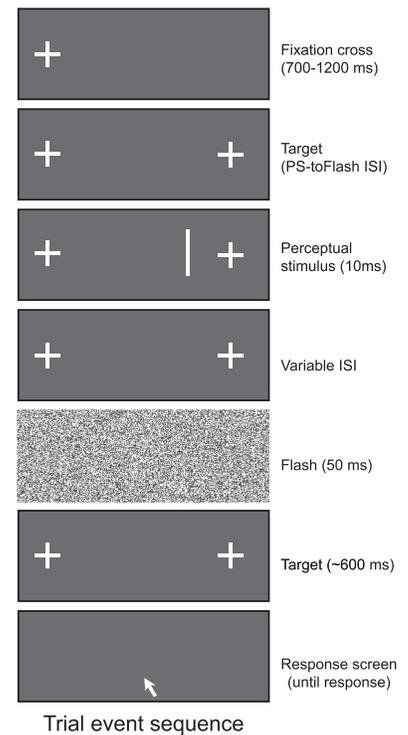


Figure 1. Predicted perceptual changes after saccadic inhibition (SI) and Methods for Experiment 1, Experiment 2, and Experiment 3. Perisaccadic perceptual effects, such as saccadic mislocalization, are proposed to result from a predictive copy of the motor command. Thus, SI of the motor command should affect performance. (A) If the efference copy of the command is sent prior to saccade onset and causes the anticipatory change in perception but the eye movement is delayed, this should cause a leftward shift in the curve as shown in the left panel. (B) Alternatively, if the probe is captured by the onset of flash it would be perceived as if it was presented at an earlier time, where mislocalization would be lower, hence shifting the mislocalization curve rightward. (C) In Experiments 1 and 2, participants were instructed to make a saccade as soon as the fixation cross stepped either to the left or to the right side of the screen and then use a mouse to report the location of a briefly presented vertical bar presented at 6.6° of visual angle on the same side as the saccade target. In some trials we presented a white rectangle covering one-third of the top and one-third of the bottom of the screen for 22 ms. The flash onset was time-locked to the median saccadic reaction time (SRT) of each participant collected in a preliminary block of 40 target-only trials by subtracting 80 ms in order to maximize the inhibitory effect of the flash. Participants were asked to ignore the flash and focus on the saccadic and the perceptual task. In Experiment 1, the time of

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the perceptual stimulus (PS) was time-locked to flash onset and presented 117 ms thereafter. In Experiment 2, we varied the time between flash and PS so that it could have appeared 141, 117, and 35 ms before or 70 ms after. (D) The time course of Experiment 3 was identical to that of Experiments 1 and 2, but now participants were asked to make a 16.5° saccade from left to right. In the saccadic version of the task, a flash made of random noise, either covering the entire screen or the one-third top and bottom of the screen, was presented 50 ms before the PS. (E) In the fixation version of the task, the sequence of events and the stimuli used were identical to the saccadic task but now participants were required to not make any eye movement. The flash was presented 165 ms on average after the displacement of the target. The PS was presented either 110, 80, or 20 ms before the flash, or 60 or 90 ms thereafter. Stimuli are not on scale.

trial blocks; additional calibrations were run if the participant moved their head from the chinrest.

Experiment 1

After the initial fixation period, the fixation cross was displaced by a step of 13.3° to the right or to the left side of the monitor along the horizontal axis (Figure 1C). The saccadic target remained visible for 700 ms. The perceptual stimulus (PS), a white vertical bar (0.1° by 1.3°), was also presented on the same side of the saccadic target at 6.6° of eccentricity (Figure 1C). The timing of the flash varied across participants (see above). Participants were asked to first move their eyes to the new fixation location and then at the end of the trial to report the location where they perceived the bar to have been presented. At the end of the trial, the mouse cursor appeared at the center of the screen, 3.5° below the fixation cross, and participants were able to adjust the mouse cursor and then click at the perceived location of the PS. In flash trials, a flash occupying one-third of the top and one-third of the bottom of the screen was presented for 23 ms (Figure 1C). The onset of the flash was set to occur 80 ms before the median SRT calculated in the preliminary baseline block, in order to evoke saccadic inhibition. On these trials, the PS was time-locked to the presentation of the flash, being displayed 117 ms after the flash. The timing was arranged so that the PS was presented just after the point of maximum inhibition (where there are virtually no saccades) during the recovery phase of the SI dip. In target-only trials there was no flashed distractor. Each of the four (target left, target right, target plus flash left, target plus flash right) occurred 12 times per block, shuffled randomly. Each participant completed two sessions of three blocks, for a total of 288 trials. Data were then collapsed by side and analysis focused on target only versus target flash condition.

Experiment 2

We ran a supplementary control experiment testing different flash times with respect to the onset of the

perceptual stimulus. The experimental design was identical to the one described above (Figure 1C) with the difference that we introduced four flash delay conditions. In flash trials, the flash was time-locked to the PS onset, being presented either 141, 117, or 35 ms *before* the PS or 70 ms *after*. The presentation onset time of the PS was also jittered by adding a multiple of 11.7 ms (our refresh rate resolution), starting from 11.7 to 58.8 ms (1 to 5 refresh rate). This jitter was aimed to optimize the number of trials per each time bin used in the analysis. Each of the 10 conditions (factorial combination of condition by target side) occurred eight times per block, shuffled randomly. Each participant was meant to complete two sessions of 15 blocks, for a total of 1,200 trials divided over two days. Six participants were not able to complete all 30 blocks (three participants completed 22 blocks, two participants completed 25, and one participant completed 26 blocks). Data were then collapsed by side and analysis focused on target only versus target flash condition.

Experiment 3

In Experiment 3, we measured the pattern of saccadic compression for targets in different locations to replicate previous studies showing that probes presented more eccentric than the saccadic target were mislocalized towards the target (Figure 1D). Moreover, we tested if localization errors were also present when participants were asked to maintain fixation for the whole length of the trial, in line with recent results from Zimmermann and colleagues (2013; Zimmermann et al., 2014).

In the *saccade* version of Experiment 3, the fixation cross was always presented on the left side of the screen at 10° of visual angle from the central point (Figure 1D). After a delay interval varying randomly between 500 and 1200 ms, the fixation made a step of 16.5° of visual angle. On saccade trials, participants were instructed to fixate the cross and to move their eyes to the target as soon as it made a step. In flash trials, a flash made of random noise (i.e., at each pixel was assigned a random gray value varying from 0 [black] to 125 [white]) covered for 20 ms either the entire screen (full-flash) or two-

thirds of the screen (2/3-flash). Flash onset always preceded the onset of the PS by 50 ms. The onset of the PS was calculated online by subtracting from the ongoing median SRT a value extracted at random from an array of SOAs. The set of SOAs contained values spanning from 20 to 140 ms in steps of 20 ms. In this experiment, we also varied the position of the PS relative to the saccadic target. In particular, the PS could have been presented either approximately 10° or 5° of visual angle on the left of the saccadic target or approximately 5.8° to the right. Each of the nine conditions (factorial combination of flash [no-flash, full-flash, 2/3-flash] by PS location [-10 , -5 , and $+5.8$]) occurred eight times per block, shuffled randomly. Each participant completed two sessions of 10 blocks, for a total of 1,440 trials divided over two days.

In the *fixation* version of the experiment, the setup was identical to the saccadic experiment but participants were asked to maintain their gaze on the left fixation cross for the whole length of the trial (Figure 1E). After a random delay between 500 and 1200 ms, a second fixation cross appeared at 16.5° of visual angle on the right side of the screen followed by the presentation of the flash 150 to 180 ms thereafter (mimicking the average saccadic reaction time for this type of stimulus). The locations of the PS were identical to the saccade experiment. In line with recent studies of compression effects without saccades (Zimmermann et al., 2013; Zimmermann et al., 2014), the PS was presented 20, 80, or 110 ms before the flash or 60 or 90 ms after the flash. In this way, the PS was aligned to flash onset, rather than to saccade onset as in the previous experiments. Each of the 30 conditions (factorial combination of flash [full-flash, 2/3-flash] by PS location [-10 , -5 , and $+5.8$] by PS time [-110 , -80 , -20 , $+60$, or $+90$]) occurred two times per block, shuffled randomly. Each participant completed one session of 12 blocks, for a total of 720 trials. The order of the experiment condition, saccade or fixation, was counterbalanced across participants.

Data screening

Saccades with a latency of less than 70 ms (Experiment 1: approx. 1.5%; Experiment 2: approx. 0.2%; Experiment 3: 1.4%); of more than 500 ms (Experiment 1: approx. 1.2%; Experiment 2: approx. 0.5%; Experiment 3: 0.9%); of less than 1° amplitude (Experiment 1: approx. 0.8%; Experiment 2: approx. 2.2%); or less than 10° (Experiment 3: 9.7%), and saccades made in the wrong direction (Experiment 1: approx. 0.8%) were excluded. In Experiment 3, since we asked for a saccadic task different from the previous two (i.e., larger saccade starting from the left side, block direction and multiple PS locations), we excluded

trials in which saccadic latency was of less than 70 ms (approx. 1.4%), more than 500 ms (approx. 0.9%), less than 10° (approx. 9.7%), or with a duration longer than 150 ms (approx. 0.8%). In the fixation version of Experiment 3 we excluded all the trials in which participants made an eye movement (13.1%).

In Experiments 2 and 3, when participants did not perceive the PS, they reported this failure by clicking with the mouse on the center of the screen. This happened for approximately 6.6% and 1.87% of trials, so these trials were subsequently removed from the analysis (in Experiment 1 this happened for approx. 0.8%, but we did not remove those trials since participants were not explicitly instructed to report missed perceptual targets and inspection of the data showed that only three of these trials fell in the presaccadic period).

Analysis of saccadic inhibition

For the saccade trials not excluded (see above), a full analysis of the SRT distributions was conducted according to the method of Buonocore and McIntosh (2012) modified according the Bompas and Sumner (2011) procedure for dip ratio calculation (see also McIntosh & Buonocore, 2014). Once the saccadic data were aligned to flash onset for each participant and condition (target-only and flash), a probability density estimate was evaluated within a 500 ms interval, spanning from -150 to 350 ms with 1 ms precision, using a kernel-smoothing window of eight points. The magnitude and the latency of SI was calculated by using the proportional change for each point in time in the flash distribution relative to the baseline distribution; that is, by using the formula: (target – flash)/target. We then extracted the maximum inhibition and its latency in the first 150 ms before distractor onset. A full description of SI goes beyond the aims of the current paper, since SI was used only as a method to interfere with the execution of the saccade. In brief, we expected SI to cause suppression during approximately 60 to 110 ms and cause an overall delay in saccades. We confirmed this prediction empirically, and the analysis showed that we successfully induced SI in each participant and experiment.

General analysis of perceptual performance

We followed the convention of displaying performance (localization) in the task as a function of the timing of the stimulus (flashed bar) with respect to saccade onset. For each participant and condition, SRTs were subtracted from the time of the PS (i.e., Experiments 1, 2, and 3, offset of the vertical bar) so

that negative values represented stimuli that were flashed before the onset of the eye movement, while positive values indicated that the PS was shown after saccadic onset. For the present study, the focus of interest was mainly on the presaccadic trials, which would seem more likely to reflect the effect of a predictive signal. For each participant, we fit the raw data with a robust local logistic regression, locally weighted scatterplot smoothing (LOWESS), with a linear polynomial function and a smoothing window containing the 15% of the data points. From the fit, we extracted data points every 20 ms (Experiments 1 and 2) or 10 ms (Experiment 3) that were then submitted to the statistical analysis. The use of SI to delay saccades had the collateral effect of generating bimodal SRT distributions, a well-known feature of the SI paradigm. Nonetheless, the consequence of this manipulation was that along the presaccadic interval there might have been sudden drops of sampled trials, compared to the target-only condition. The fitting procedure helped to overcome this limitation and, for clarity, we report the average number of trials in bins across participant and along the presaccadic interval (see caption for Figure 2). At the individual level, if the fit returned a null value in the presaccadic interval (between -90 and 0 ms), the participants were excluded from statistical analysis. This happened for two participants in Experiment 1, one participant in Experiment 2, and one participant in Experiment 3.

In Experiments 1 and 2, we ran a two (flash: no-flash vs. flash) by ten (bin: -110 to $+70$ in steps of 20 ms intervals) repeated-measures ANOVA. In Experiment 3, we run three repeated-measures ANOVAs, one for each PS location (-10° , -5° , and $+5.8^\circ$), with three (flash: no-flash, full-flash, and partial-flash) by 18 (bin: -110 to $+70$ in steps of 10 ms intervals). A Greenhouse-Geisser adjustment to the degrees of freedom was performed where sphericity was violated. Significant interactions were followed up by a series of paired samples t tests comparing no-flash versus flash conditions at each time bin. Considering that adjacent time bins are likely to be correlated, we performed the Benjamini and Hochberg (1995) and the Benjamini and Yekutieli (2001) procedures controlling the false discovery rate (FDR) of a family of hypothesis tests. Corrected p levels are reported in the text.

Results

Experiment 1

We first investigated the time course of perceived localization using a standard approach (Figure 2A). As

shown in Figure 2A, we were able to replicate the classic presaccadic compression profile in which the flashed probe is perceived closer to the saccadic target (Honda, 1999; Lappe et al., 2000; MacKay, 1970; Matin & Pearce, 1965; Morrone et al., 1997; Ross et al., 1997), even if the PS was mislocalized only about halfway between its physical location and the saccadic target. Formally, the repeated measure ANOVA confirmed this observation with a main effect of time bin, $F(2.7, 26.8) = 21.47$, $p < 0.0001$, suggesting that for both conditions there was a reliable increment in localization error that was peaking just a mere 30 ms after saccadic onset. No effect of flash was observed, $F(1, 10) < 1$, ns]. On half of the trials, chosen at random, we induced inhibition by flashing rectangular bars at the top and bottom of the screen (Figure 1C). The SRT histograms show that in flash trials there was a sudden drop in saccadic frequency soon after flash onset (Figure 3A, red curve) and the overall inhibitory profile is depicted in Figure 3B. Overall, more than 85% of saccades were suppressed about 80 ms after flash onset, creating a shift in SRTs towards later values since these saccades were delayed. We extracted from each individual SI profile two parameters: the maximum inhibition and the latency of the maximum inhibition (Figure 3C and D). From the two bar graphs it is possible to appreciate the consistency of the SI for each individual in both the magnitude (green bars) and the latency (orange bars) of the inhibition. Robust SI was recorded in each participant about 80 ms after flash onset.

Consistent with our hypothesis, the SI manipulation altered the time course of the presaccadic localization (Figure 2A). This was confirmed by an interaction between flash and bin, $F(2.7, 26.7) = 7.572$, $p < 0.001$. In particular, in the flash condition, the curve of the localization error was delayed by about 20 ms, consistent with the hypothesis that SI would effectively create a brief pause or delay prior to saccade initiation (Figure 1A). The significance of this time-specific shift was tested via a series of post hoc pairwise t test on the bins comparing the difference between no-flash and flash conditions. In the presaccadic period, we report a significant difference for the bin starting at -30 ms, bin -30 : $t(10) = 4.6948$, $p < 0.0042$, and persisting at -10 ms, bin -10 : $t(10) = 5.3450$, $p < 0.0033$. Moreover, we found a significant reduced localization error, compared to the no-flash condition, in the postsaccadic period for the $+30$, bin 30: $t(10) = 2.8996$, $p < 0.0396$, and $+50$ ms, bin 50: $t(10) = 3.8770$, $p < 0.0102$, time bin after saccadic onset. While the difference, between no-flash and flash (SI) trials during the presaccadic period indicates a specific effect of SI on the timing of localization error, its persistence in the postsaccadic period suggest an extended effect of SI upon saccades, so that delayed saccades tends to carry over an error in

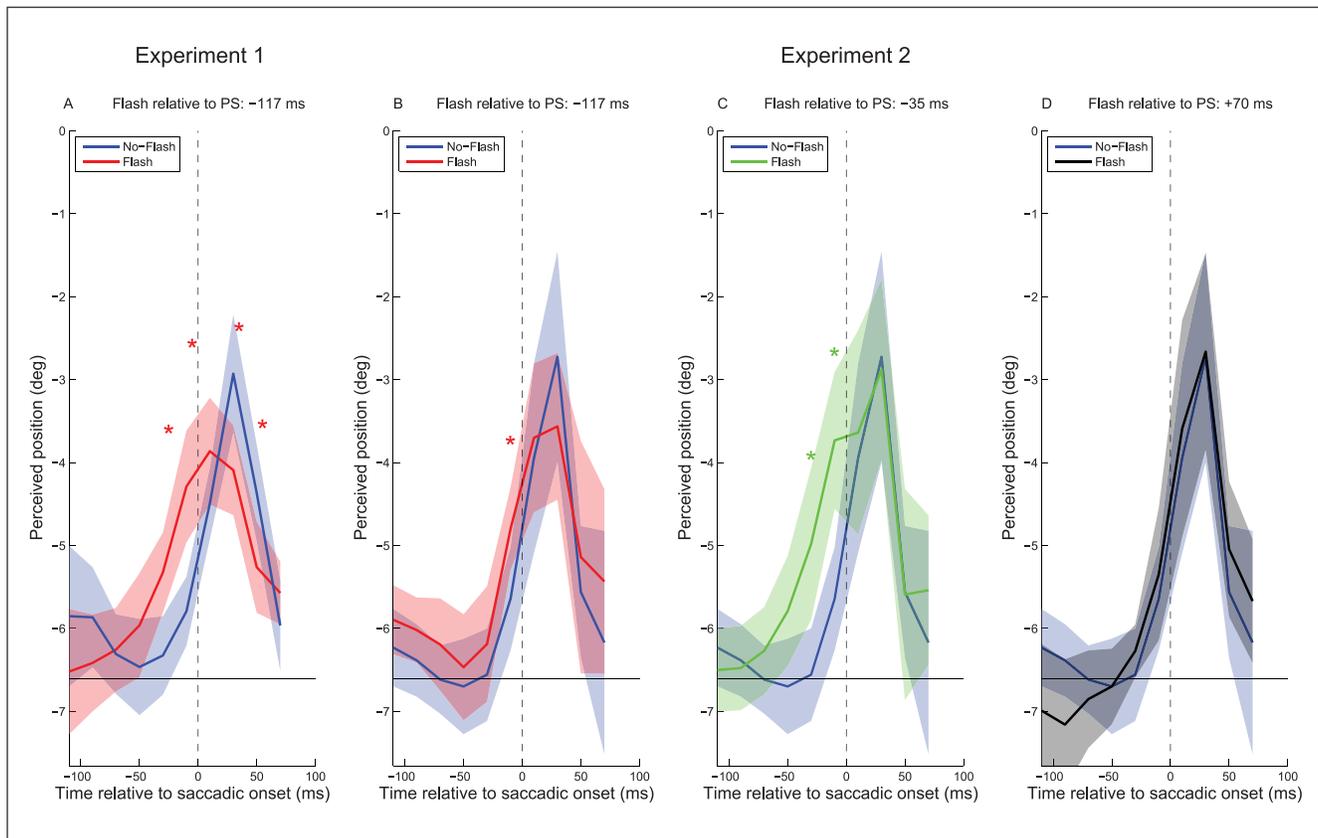


Figure 2. Perisaccadic localization errors in Experiments 1 and 2. (A) Perisaccadic localization errors for baseline (blue) and SI (red) trials in Experiment 1. (B–D) Perisaccadic localization errors in Experiment 2 for baseline (blue) and SI trials (either red, panel B, green, panel C, or black, panel D). Each panel represents the time-course of the perisaccadic compression effect relative to the time difference between flash and PS (panel B: -117 ms; C: -35 ms; D: $+70$ ms). In all the graphs, negative time values indicate that the bar was presented *before* the onset of the saccade. The shaded area represents the 95% confidence interval corrected for between-subjects variability. Asterisks indicate a significant difference between the baseline and the saccadic inhibition (SI) time point. Average number of trials between -90 to $+90$ ms relative to saccade onset in steps of 30 ms: Experiment 1: no-flash: 4, 9, 15, 25, 44, and 30 and flash: 14, 20, 29, 20, 16, and 19. Experiment 2: no-flash: 7, 19, 41, 50, 49, and 21 and flash -117 : 23, 47, 61, 29, 13, and 10; flash -35 : 16, 14, 28, 51, 50, and 19; flash $+70$: 5, 21, 43, 50, 51, and 20.

perception. Overall, the temporal shift in the time course of mislocalization is consistent with the hypothesis that perisaccadic mislocalization can be linked to the efference copy signal (the intention to move the eye) rather than the physical eye movement itself.

Experiment 2

This second experiment was run to check that the effect of SI was temporally specific and that the onset of the flash was not “capturing” the onset of the perceptual stimulus due to a more general effect on spatial localization during uncertainty (Figure 1B; Majaj, Brenner, & Smeets, 2009). It is important to note that, in our design, any such effects would have been expected to cause a shift in the opposite direction (i.e., the PS would have been associated with an error in

localization at the time of the flash, shifting the curve rightward), and that the type of suprathreshold stimuli used here have been shown to be immune from such capture effects (Binda, Morrone, & Burr, 2010). Nonetheless, in order to completely rule out this alternative explanation and to show the specificity of the SI effect on mislocalization, we ran a control experiment using different ranges of SOA between the perceptual stimulus and the flash. In particular, in Experiment 2 the flash could be presented at 141 ms, 117 ms, or 35 ms before, or 70 ms after the perceptual stimulus. Participant reaction times for this task were quite fast, so the 141-ms condition coincided with, or was lower, than the onset of the perceptual stimulus; there were few or no presaccadic trials in that condition.

First, inspection of the SRT distributions (Figure 3E) highlighted a drop in frequency associated with the

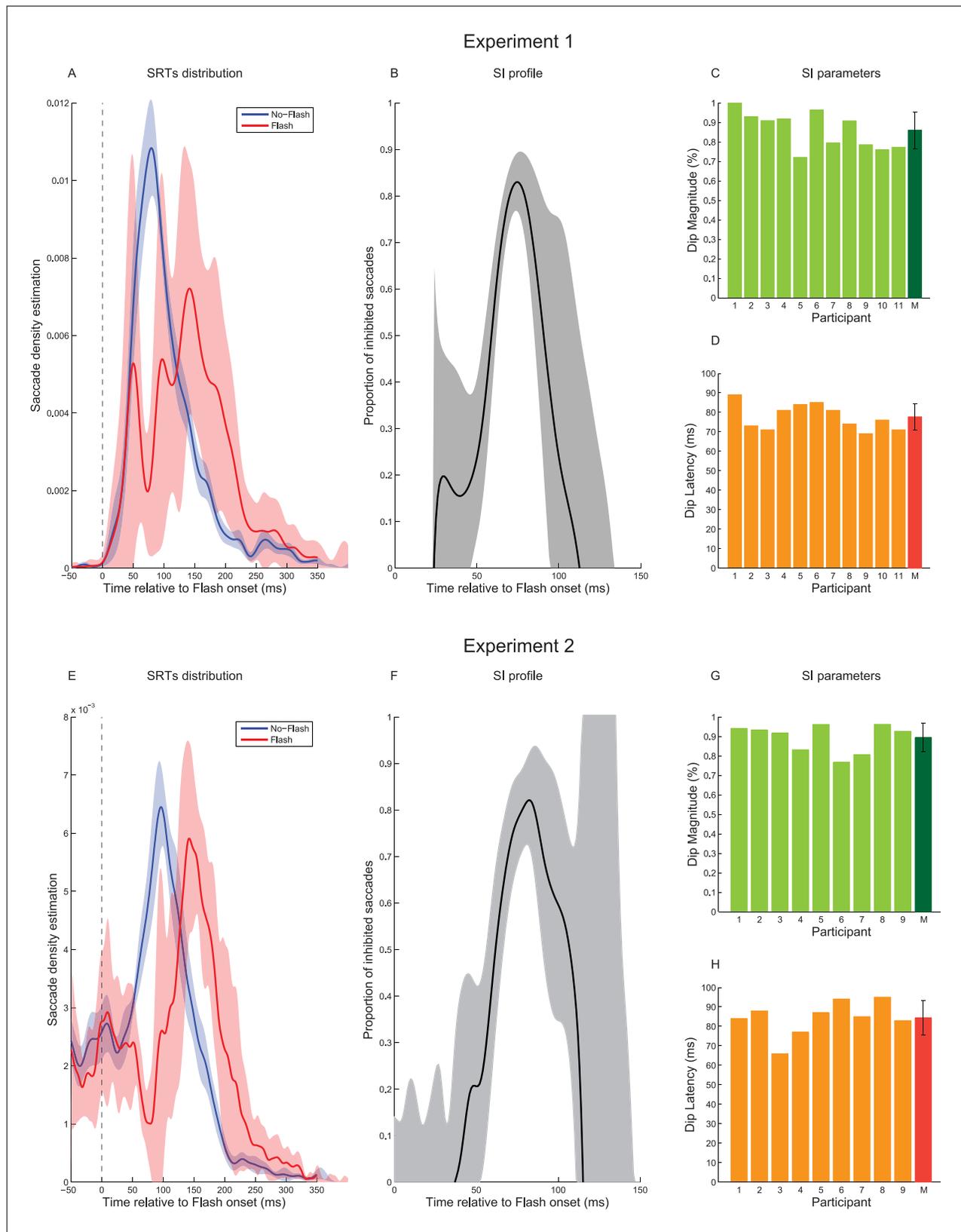


Figure 3. Saccadic inhibition (SI) analysis in Experiments 1 and 2. (A–E) Saccadic reaction times (SRT) distributions in the target-only and flash conditions for data aligned to flash onset. The shaded area indicates the standard deviation of the data. (B–F) For each individual, the SI profile was obtained as the proportion of inhibited saccades. SI profiles were then averaged together to express the

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mean inhibition. Overall, in both experiments, more than 80% of saccades were suppressed about 80 ms after flash onset. The shaded area indicates the 95% confidence intervals. From each individual, SI profiles were extracted two parameters: the maximum inhibition and the latency (C–G) of the maximum inhibition (D–H). From the two bar graphs, it is possible to appreciate the stability of the SI for each individual in both the maximum (green bars) and the latency (orange bars) of the inhibition. The darker bars in each of the two panels represent the overall mean (M) and standard deviation (SD).

flash condition (red curve), similar to the one recorded in Experiment 1. The development of the inhibition is depicted in Figure 3F, replicating a general robust SI peaking about 80 ms after flash onset. Moreover, each participant showed a strong inhibitory effect at a constant timing (Figure 3G, H) Hence, SI altered again the time course of the perisaccadic localization, in a way compatible to Experiment 1. Considering the conditions in which each participant had sufficient trials in the presaccadic condition (excluding the 141-ms condition), we replicated the main effect of time bin, $F(9, 72) = 13.180$, $p < 0.0001$, indicative of how the localization error varied over time. Thus, as in the previous experiment, participants were reporting the location of the perceptual probe toward target location already during the presaccadic interval, as can be seen in Figure 2B, C, and D. As for the previous experiment, the probe was mislocalized only about 60% of the distance between PS and saccadic target physical distance. Then, the repeated measure ANOVA unveiled a main effect of the flash-to-PS timing, $F(3, 24) = 5.053$, $p < 0.01$, confirming that the localization pattern was modulated according to the time of the flash. But more importantly, we found again on interaction between flash and bin, $F(27, 216) = 4.618$, $p < 0.0001$, which was further investigated using a paired-sample t test between no-flash and flash at each time bin. As predicted, for the -117 -ms flash, the localization curve was shifted leftward (i.e., as in Experiment 1), with a significant difference at 10 ms before saccadic onset, $t(8) = 5.0181$, $p < 0.0103$ (Figure 2B). Moreover, in the -35 -ms condition, we recorded a more pronounced leftward shift in the localization error, starting at -30 ms and persisting at -10 ms before saccadic onset, $t(8) = 3.294$, $p < 0.05$ and $t(8) = 5.140$, $p < 0.0088$, respectively (Figure 2C). This finding of an earlier shift in the mislocalization curve is coherent with the SI hypothesis. In fact, when the probe was presented 30 ms before saccadic onset, the relative time of the flash was 75 ms before saccadic onset, already in the range of SI. We can then hypothesize that the -30 ms PS was associated with delayed saccades. Overall, the results of Experiment 2 expand and confirm the pattern found in Experiment 1, consistent with the idea that SI saccades are delayed and this added delay drives the shift of the mislocalization curve at different intervals before saccadic onset.

Finally, as expected, we did not find any shift in the opposite direction for the SI flash presented 70 ms after the perisaccadic stimulus (Figure 2D). Taken together, these results confirm that temporal capture (Maij et al., 2009) was not the explanation for the pattern of results found here. Instead, our findings are consistent with a previous report showing a lack of attentional capture for high-contrast stimuli (Binda, Morrone, & Burr, 2010).

Experiment 3

Experiment 3 was run to test if our design could capture the pattern of saccadic compression for PS presented at different locations, more and less eccentric relative to the saccadic goal. The second aim of this experiment was to test if a similar pattern of localization errors was also present in absence of eye movements (Zimmermann et al., 2013; Zimmermann et al., 2014).

As for Experiments 1 and 2, the SRT distributions were highly different between the no-flash and flash conditions, with a pronounced drop in saccadic frequency in flash trials (Figure 4A). This was true for both the full-flash (red) and the 2/3-flash (green) conditions. Overall, the inhibitory profile was identical for the two types of flashes (Figure 4B) and this became even clearer by looking at the magnitude (Figure 4C) and the latency of inhibition (Figure 4D) for each participant. On the basis of these observations and the finding of the previous experiments, we analyzed the perceptual performance on the localization of the PS in both the saccade and fixation experiments.

In the saccadic version of the task, to test the effect of the different flash on the localization performance, we ran three separate repeated measure ANOVAs, one for each location of the PS. For the PS at location one (-10° of visual angle), we found a main effect of bin, $F(17, 153) = 57.803$, $p < 0.0001$, underlying the strong compression of the probe toward target location (Figure 5A), no main effect of flash, $F(2, 18) < 1$, ns , but more importantly we replicated the interaction between flash and bin, $F(34, 306) = 5.481$, $p < 0.0001$. Post hoc comparisons between the no-flash and full-flash conditions at each time bin showed localization errors shifted in time by about 20 ms, compatible with the previous experiments. The shift started at -40 ms,

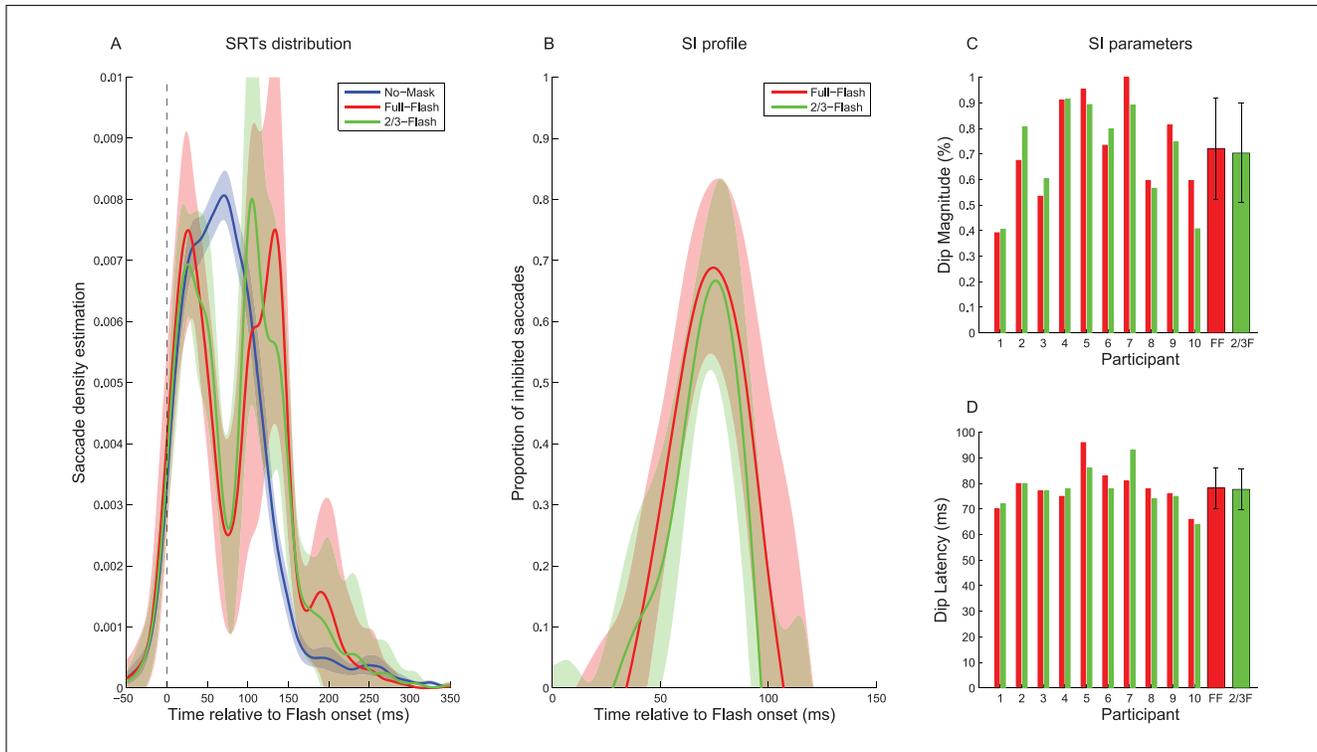


Figure 4. Saccadic inhibition (SI) analysis in Experiment 3. Same panel arrangement of Figure 3. (A) SRT distributions in the target-only (blue) and flash conditions (red: full-flash; green: two-thirds-flash) for data aligned to flash onset. The shaded area indicates the standard deviation of the data. (B) SI profiles for full-flash (red) two-thirds-flash (green). Overall, in both conditions, more than 60% of saccades were suppressed about 80 ms after flash onset. The shaded area indicates the 95% confidence intervals. SI parameters: the maximum inhibition (C) and its the latency (D) for each individual and the overall mean (FF: full-flash; 2/3F: two-thirds-flash) and standard deviation. As in the previous experiments, it is possible to appreciate the stability of the SI across participants and conditions.

bin -40 : $t(9) = 3.3987$, $p < 0.0355$, and persisted at -30 ms, bin -30 : $t(9) = 3.0476$, $p < 0.0498$, before saccadic onset. As in Experiment 1, there was a subsequent reduction in the mislocalization error starting at $+20$ ms and persisting up to $+40$ ms after saccadic onset with bin $+20$: $t(9) = 3.9541$, $p < 0.0285$; bin $+30$: $t(9) = 4.1534$, $p < 0.0285$; and bin $+40$: $t(9) = 3.7226$, $p < 0.0285$, respectively. We found a similar shift in the localization curve also in the 2/3-flash condition, affecting the presaccadic period at -50 and -40 ms, bin -50 : $t(9) = 3.1170$, $p < 0.0402$, and bin -40 : $t(9) = 3.6027$, $p < 0.0258$, with the subsequent reduction in compression in the postsaccadic period, bin $+20$: $t(9) = 3.7424$, $p < 0.0258$; bin $+30$: $t(9) = 3.7716$, $p < 0.0258$; bin $+40$: $t(9) = 4.1393$, $p < 0.0258$; and bin $+50$: $t(9) = 3.0685$, $p < 0.0402$.

For location two (-5° of visual angle), we found an effect of the bin, $F(17, 153) = 17.433$, $p < 0.0001$, suggesting that these probes were also compressed toward the saccadic goal (Figure 5B). We found a main effect of the flash, $F(2, 18) = 3.908$, $p < 0.05$, highlighting that the compression was overall less powerful in the two flash conditions. For location three

($+5.8^\circ$ of visual angle), we report only a main effect of the bin, $F(17, 153) = 13.474$, $p < 0.0001$, and no main effect of flash, $F(2, 18) < 1.4$, ns . (Figure 5C). This finding underlies that, with our stimuli, participants were localizing the probe toward the saccadic goal, even when it was presented more eccentric than the saccadic target. Nonetheless, neither of the two conditions had an interaction between flash and bin suggesting a clear shift in time during flash trials, location -5° : $F(34, 306) = 1.3$, ns , and location $+5.8^\circ$: $F(34, 306) = 1.2$, ns .

In the second version of the task, seven out of the 11 participants were tested in absence of eye movements. In order to see if there were any localization errors relative to the flash onset, we tested each time bin against the first one recorded, at -110 ms from flash onset (Figure 5D). We did not report any statistical difference aside for a mild compression effect in the full flash condition at -20 ms: $t(6) = 3.0548$, $p < 0.025$, and $+90$: $t(6) = 3.3114$, $p < 0.025$. This result suggests that, with our set of stimuli, a different pattern of localization errors were made in absence of eye

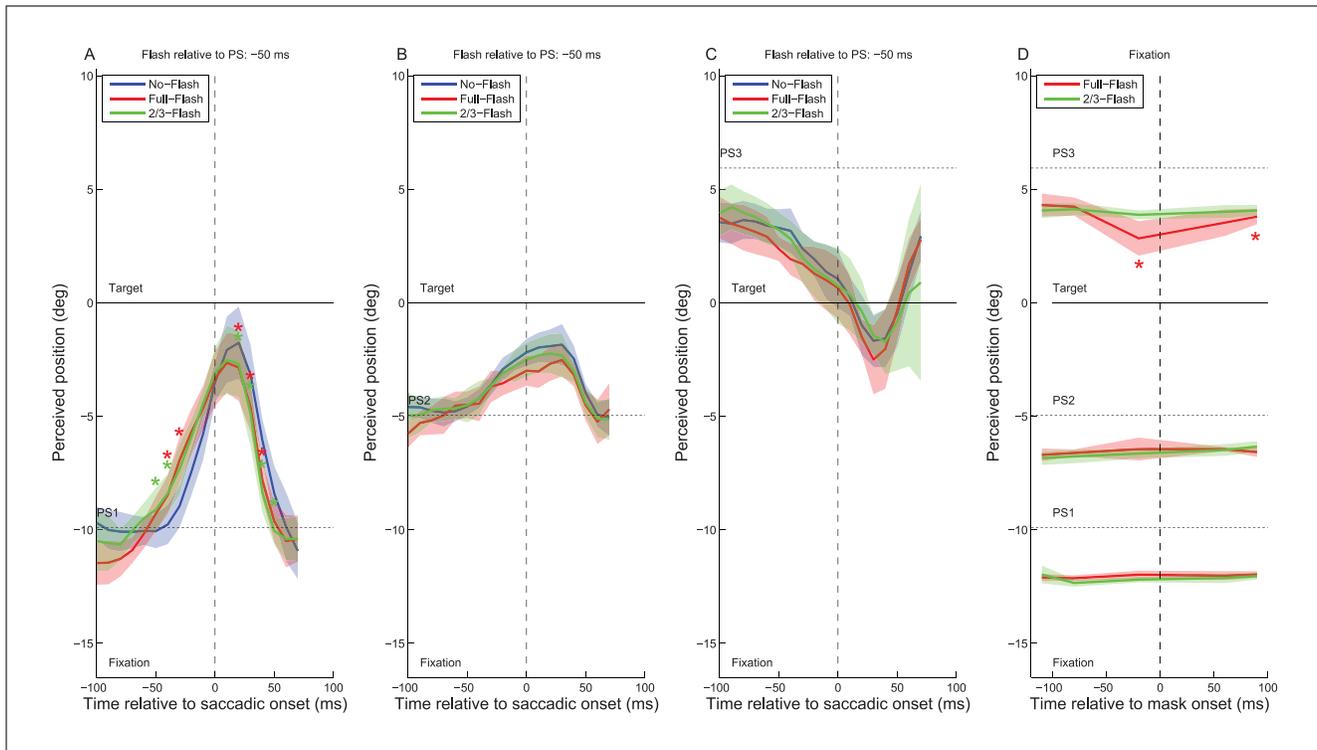


Figure 5. Perceptual performance in Experiment 3. Localization errors during saccadic trials for baseline (blue) and SI (red: full-flash; green: 2/3-flash) conditions in Experiment 3. Each panel represents a different position of the PS, respectively presented at -10° (A), -5° (B), and $+5.8^\circ$ (C) relative to the saccadic target. (D) Localization errors in Experiment 3 in which participants were restrained from making eye movements. Data are aligned to flash onset. The comparison between the saccadic with the fixation condition highlights how the pattern of errors is much more pronounced when eye movements were allowed. The shaded area represents the 95% confidence interval corrected for between-subjects variability. Asterisks indicate a significant difference between the baseline and the SI time point. Average number of trials between -90 to $+90$ ms relative to saccade onset in steps of 30 ms: PS at location 1, no-flash: 7, 23, 35, 36, 27, and 5; full-flash: 9, 28, 35, 32, 28, and 6; 2/3-flash: 17, 28, 35, 32, 25, and 6. PS at location 2, no-flash: 20, 24, 12, 26, 27, and 5; full-flash: 32, 26, 11, 22, 26, and 7; 2/3-flash: 23, 31, 12, 26, 25, and 5. PS at location three, no-flash: 17, 28, 35, 32, 25, and 6; full-flash: 34, 24, 13, 27, 26, and 7; 2/3-flash: 27, 32, 14, 28, 25, and 7.

movements, which is different from what was observed in the saccadic version of the experiment.

Discussion

In the present paper we tested whether interfering with timing of an impending saccade, via an irrelevant flashed distractor, could modulate perisaccadic mislocalization (Honda, 1999; Lappe et al., 2000; MacKay, 1970; Matin & Pearce, 1965; Morrone et al., 1997; Ross et al., 1997). We hypothesized that if an active mechanism was used to maintain visual stability then temporal interference with saccade execution could lead to changes in perception. In our study, we achieved temporal interference by flashing an irrelevant stimulus during saccadic programming and successfully inducing SI about 80 ms later (Bompas & Sumner, 2011; Buonocore & McIntosh, 2008, 2012; Reingold &

Stampe, 2002). First we replicated the typical mislocalization curve, reliably starting 40–30 ms before and peaking just after saccadic onset. Second, we report a strong interaction between mislocalization and SI. In particular, the leftward shift in the compression curve was consistent with extending the delay between motor intention and motor execution. This effect was particularly acute when the probe was presented toward the end of the SI (Experiments 1 and 2), but it was also found in the earlier part of the inhibitory period (Experiments 2 and 3). This pattern of results is consistent with our hypothesis that SI would shift the time course of perisaccadic mislocalization.

A number of previous studies have concluded that passive mechanisms, such as masking by the post-saccadic stimulus, might be sufficient to maintain visual stability (Castet et al., 2002; Dorr & Bex, 2013; Ibbotson & Cloherty, 2009; Majj et al., 2010; Ostendorf et al., 2006). Nonetheless, one potential issue with those studies was that the perceptual effects were recorded

under quite different conditions, in which participants were asked either to maintain fixation (Dorr & Bex, 2013; Ostendorf et al., 2006) or to cancel eye movements (Atsma et al., 2014). There are obviously a number of differences between making a saccade and not making one, such as the tight temporal and spatial link between attention and the saccade target. In the current study, our paradigms tried to minimize these issues by testing mislocalization effects only during overt orienting: participants always made a saccade on each trial, with the only manipulation affecting the timing of the motor execution.

Additional evidence for an active role of efference copy in perisaccadic effects comes from Experiment 3, in which we directly compared the effects of masking the perceptual target on localization judgments with the pattern of compression found with saccades. Consistent with recent reports (Zimmermann et al., 2013; Zimmermann et al., 2014), we did find a small compression in one of the conditions with fixation, but the magnitude of these effects was miniscule with respect to the strong compression found on saccade trials. Thus, while we agree that errors in localization for masked perceptual targets are potentially interesting, our data are not consistent with the strong claims made about masking or reduced visibility of the perceptual target being a potential explanation for the robust and often replicated finding of near complete compression of spatial judgments towards the saccade target.

It is important to consider whether the pattern of results found here could be explained by a more general effect on spatial localization during uncertainty (Maij et al., 2009). However, the attention capture hypothesis would have led to a shift of the mislocalization curve in the opposite direction. We directly tested, and disconfirmed, this alternative explanation in our second experiment in which the timing of the flash with respect to the eye movement was directly manipulated. Reinforcing this view, it has been reported that suprathreshold stimuli, similar to those used in the present experiments, are immune from such capture effects (Binda et al., 2010). Thus, our results can be more parsimoniously explained by the dissociation between a delayed motor program (as demonstrated clearly by measures of SI) and its execution. Our findings support the claim that mislocalization reflects, at least in part, a motor intention/prediction (Burr & Morrone, 2011; Hamker et al., 2008). Overall, our results are consistent with theories that posit an active mechanism for spatial updating across saccades, such as spatio-temporal transformation of receptive fields (Burr & Morrone, 2011; Cicchini et al., 2013; Zimmermann et al., 2013), predictive mapping of object pointers (Melcher & Colby, 2008; Melcher, 2011) or attention pointers (Cavanagh, Hunt, Afraz, & Rolfs, 2010; Rolfs, Jonikaitis, Deubel, & Cavanagh, 2011).

In terms of the size of the temporal shifts found here, which was on the order of 20–30 ms, it is important to remember that our analyses necessarily included trials in which there was a strong SI and those in which saccades escaped from any flash-induced delay. We did not characterize SI on a trial-by-trial basis, but instead grouped all trials in the flash condition together in order to show that SI occurred. It is likely that a large proportion of trials were included in our analysis of the flash condition in which saccades were not delayed, thus strongly diluting the size of the effects since both SI and no-SI trials were averaged together.

On a neurophysiological level, the present data can be explained by inhibitory mechanisms within the SC and their interactions with the FEF. It has been proposed that SI derives either from lateral interactions in the intermediate layers of the SC (Olivier, Dorris, & Munoz, 1999) or stimulation of omnipause neurons via fixation related neurons (Findlay & Walker, 1999; Munoz & Wurtz, 1993a, 1993b). The motor signal arising within the SC is thought to split into an ascending pathway to the mediodorsal thalamus (and to the FEFs; Wurtz et al., 2011) and a descending pathway to the brainstem and pons for executing the motor program, where it also receives signals from the FEF, that is combined with the SC signal by means of efference copy. It is also well known that the receptive field changes in the FEFs are modulated by the efference copy signal from the SC (Sommer & Wurtz, 2006), bringing information about the programmed saccade. Consequently, the modulation of the SC signal by flash appearance (and here recorded as SI) might vary the dynamics of the FEF signal, leading to temporal changes in the mislocalization pattern. Given that the ascending pathway is thought to carry the efference copy signal, it would be interesting in future studies to measure the influence of SI on these two pathways. The congruency between the substrates implicated in SI and efference copy, together with the timing of these signals, is highly suggestive for an account of the behavioral effects found here in terms of the efference copy (corollary discharge).

In the present experiments, we aimed to introduce a temporal delay between the efference copy and saccade execution. It has been argued that perception and motor systems might be differentially influenced by efference copy (Bays & Husain, 2007). In the current study, we found that SI affected both action and perception, consistent with theories linking perceptual effects with a predictive motor signal. Similarly, recent studies showing remapping patterns in the FEFs match the pattern of compression found in mislocalization (Zirnsak et al., 2014) are consistent with an active, sensorimotor explanation for at least some perisaccadic effects.

The current results imply that SI could be a fruitful tool for studying the role of motor intention (and

hence, indirectly, efference copy) in behavioral measures of visual stability (see also Buonocore & Melcher, 2015). Other psychophysical paradigms that have been tied to visual stability include spatial updating across a double-step saccade, saccadic suppression, and spatial remapping of visual features (for review, see Melcher, 2011). Converging evidence, from computational modeling, psychophysics, neuroimaging and neurophysiology, would be needed to determine which of these behavioral effects are determined by an active, predictive mechanism that takes into account the intention to move the eyes, and which behavioral effects reflect the influence of the saccadic eye movement itself.

Keywords: visual stability, mislocalization, eye movements, saccadic inhibition, efference copy

Acknowledgments

This research was funded by a European Research Council Starting Grant award (Grant Agreement n. 313658) to D. M. In addition, A. B. was funded by a research fellowship from the Autonomous Province of Trento through the call, Grandi Progetti 2012 project “Characterizing and improving brain mechanisms of attention—ATTEND.” The authors declare no competing financial interests.

Commercial relationships: none.

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