Fixation related shifts of perceptual localization counter to saccade direction

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Perisaccadic compression of the perceived location of flashed visual stimuli toward a saccade target occurs from about 50 ms before a saccade. Here we show that between 150 and 80 ms before a saccade, perceived locations are shifted toward the fixation point. To establish the cause of the "reverse" presaccadic perceptual distortion, participants completed several versions of a saccade task. After a cue to saccade, a probe bar stimulus was briefly presented within the saccade trajectory. In Experiment 1 participants made (a) overlap saccades with immediate return saccades, (b) overlap saccades, and (c) step saccades. In Experiment 2 participants made gap saccades in complete darkness. In Experiment 3 participants maintained fixation with the probe stimuli masked at various interstimulus intervals. Participants indicated the bar's location using a mouse cursor. In all conditions in Experiment 1 presaccadic compression was preceded by compression toward the initial fixation. In Experiment 2, saccadic compression was maintained but the preceding countercompression was not observed. Stimuli masked at fixation were not compressed. This suggests the two opposing compression effects are related to the act of executing an eye movement. They are also not caused by the requirement to make two sequential saccades ending at the initial fixation location and are not caused by continuous presence of the fixation markers. We propose that countercompression is related to fixation activity and is part of the sequence of motor preparations to execute a cued saccade.

Introduction

The apparent stability and constancy of the visual world is a testament to the extent of the visual processing going on "under the hood" of conscious awareness each and every moment. Our capacity to perceive the world "as it is," rather than perceiving the retinal image, is astonishing if you consider that our eyes (let alone the rest of the body) are never still but are in constant motion. Our eyes are always either drifting, making very small but high velocity shifts around a target position, following a moving target, rotating to give focus to different depth planes, or making large high velocity movements to acquire information about a completely new part of the visual scene. In general we are not aware of and in some cases are unable to become aware of these eye movements. Given everyday circumstances, we are unable to consciously monitor the motor act of moving our eyes and unable to access any conscious visual sign that the eye movement has been made. There is one particular situation where conscious visual awareness does become privy to the act of making an eye movement—in a lab setting when a small stimulus is flashed briefly around the time of a saccadic eye movement. Many studies have shown that from around 50 ms prior to the onset of a saccade a briefly presented stimulus is often mislocalized as though it had appeared closer to the target of the saccade (Bischof & Kramer, 1968; Ross, Morrone, & Burr, 1997; Lappe, Awater, & Krekelberg, 2000; Ostendorf, Fischer, Finke, & Ploner, 2007). The mislocalization is considered a perceptual consequence of the "under the hood" processing that is contributing to our ability to perceive the world as stable despite our own constant eye motion (Hamker, Zirnsak, Calow, & Lappe, 2008; Binda, Cicchini, Burr, & Morrone, 2009; Hamker et al., 2011).

Perisaccadic perceptual mislocalization could be the consequence of one or several possible processes. One of these is the motor plan associated with the saccade. Spatial perception is tightly linked to oculomotor control (Zimmermann & Lappe, 2010, 2016). Each saccade is preceded by preparatory activity in oculomotor areas such as the superior colliculus or the frontal eye field. A corollary discharge of this activity

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informs other brain areas about the upcoming saccade, affects spatial locations for transsaccadic perception (Melcher & Colby, 2008; Zirnsak & Moore, 2014) and future actions (Sommer & Wurtz, 2008), and produces a shift of attention to the saccade target location (Moore, Armstrong, & Fallah, 2003; Hamker et al., 2008; Arkesteijn, Belopolsky, Smeets, & Donk, 2019). As a consequence of this, the saccade target enjoys several perceptual benefits even before the eye starts moving (Deubel & Schneider, 1996; Kowler, Anderson, Dosher, & Blaser, 1995; Rolfs & Carrasco, 2012), each of which is consistent with a shift of attention to this region. The suggestion is that a feedback signal from oculomotor planning areas such as the frontal eye field or the superior colliculus produces a modulation of sensitivity at the target location in visual maps (Hamker et al., 2008). Neurons responsive to this retinotopic region of space increase their stimulus-driven activity relative to those responsive to other locations. In a readout of the whole population of location-tagged neurons, this increase in activity causes the perceived location to be skewed toward the stronger activity occurring in the region of the saccade target. The effect is to draw the perceived location toward the peak of the region of attention around the saccade target. The motor intention of the eye movement is likely to be a major cause of mislocalization. Lappe and Hamker (2015) found a bimodal pattern of localization errors when participants held two motor plans simultaneously. This was achieved by giving two possible saccade targets even though participants could eventually only execute one of the motor plans. This study also demonstrates that it is possible to hold two simultaneous motor plans that may play off each other for execution.

Despite being strongly implicated in saccadic mislocalization, further results suggest that motor planning is not the sole cause. Mislocalization is reduced when there are no other visual stimuli in the scene, indicating that visual references about the target location of the saccade contribute to the manifestation of compression (Lappe et al., 2000; Awater & Lappe, 2006; Zimmermann, Morrone, & Burr, 2014). This implies that a strong focus of attention may be necessary but it is not sufficient, in isolation, to drive strong saccadic mislocalization.

Another possible cause could be an inability of the visual system to accurately relate the retinal input to the information it has about the position of the eyes in the orbits (Matin, Pearce, & Pearce, 1969; Honda, 1991). If the information maintained by the visual system about the gaze position is updated ahead of the actual saccade and the estimated gaze was thought to be closer to the planned target prior even to saccade onset, this would cause a significant shift of the perceived location of stimuli in the direction of the saccade. This would be maximal when the eye position is considered to be at the saccade target but in reality the eye is still looking toward the initial fixation location, if this situation were to occur. This kind of eye position signal error would predict that even targets presented beyond the saccade trajectory are perceived as shifted further out in the same direction as the saccade. Instead, stimuli in these locations are seen as compressed toward the saccade target. An anticipatory shift of the eye position signal may contribute to the mislocalization effect but it cannot predict the effect on stimuli presented outside the trajectory of the saccade.

It seems then, that the influence of a presaccadic reallocation of attention is not sufficient to explain the full magnitude of mislocalization, nor is a nonsynchronous eye position and retinal signal necessarily the sole cause. What is required is a motor plan that produces presaccadic reallocation of attention and a subsequent disruption of visual continuity that requires a comparison between pre- and postsaccadic scenes (Awater & Lappe, 2006). The disruption of continuity about the stimulus in most studies might be caused by a combination of the brief duration of the stimulus, the sudden change of retinal input produced by the saccade, or any other disruption producing perisaccadic visual effects (e.g., gray out, backward masking). For compression to occur, the disruption appears to require that it occurs in the context of a motor plan with allocation of attention to the location of the motor goal. Two apparently contradictory findings support this idea. Zimmermann, Born, Fink, and Cavanagh (2014) have shown that perceptual mislocalization can be induced by a peripherally presented, attention-grabbing stimulus followed by an uncertainty inducing backward mask. Conversely, Atsma, Maij, Corneil, and Medendorp (2014) have shown that stimuli are more accurately localized if a saccade is planned and then not executed during a go/no-go task. In the first study there is a clear disruption via the backward mask but it may appear that there is no motor plan. It's likely, however, that the peripheral onset of the stimulus provides a sufficiently strong attentional capture that a motor plan is generated and the mask provides well enough timed uncertainty such that the motor plan need not be executed and yet mislocalization is induced. When cancelling a saccade in a go/no-go task, the motor plan is initially created and may be sufficiently reduced in order to stop the saccade occurring such that mislocalization is reduced. Even if the motor plan is not sufficiently reduced, the saccade induced disruption does not follow. In summary, perisaccadic mislocalization appears to require the attentional enhancement of a region of space that is the focus of a motor goal and at least the passive induction of significant disruption of visual continuity such as that provided by backward masking.

While saccadic and masking-induced mislocalization has generally been investigated using a motor end plan or attentional capture target that is away from the current fixation point, Haladjian, Wufong, and Watson (2015) found that when participants are asked to make two saccades, the second of which returns to the original fixation location, localization errors are toward the initial fixation point when the target stimulus is presented 50 ms ahead of the first saccade and then turn toward the end point of the first saccade when the stimulus is presented within 20 ms before the onset of or during the first eye movement. The early mislocalization in the direction of the current fixation location/final saccade target for stimuli presented ahead of the onset of the first saccade presents an opportunity to investigate whether this mislocalization is due to allocation of attention to the final landing location of the two-saccade sequence or might rather be due to a fixation maintenance–related modulation of attention at the fovea in the context of an impending eye movement. In other words, this counter mislocalization might be due to a motor plan involving a strong fixation signal that keeps the eyes at the initial saccade target prior to the cue to saccade or it might be due to the plan for attaining the end point of the second saccade. The following experiments were designed to investigate this issue.

Experiment 1: Single versus double overlap and step saccades

The purpose of Experiment 1 was to determine whether the mislocalization toward the initial fixation point requires a two-saccade outward-and-return sequence or whether it also occurs for single saccades. In addition, we wanted to manipulate the presence of the fixation point.

Method

Participants

Six participants, (two female, between 23 and 53 years of age) completed all three conditions. The experiment was approved by the Western Sydney University Human Research Ethics Committee and carried out in accordance with the guidelines in the Declaration of Helsinki.

Equipment

Stimuli were presented on a Sony Trinitron CRT monitor running at 100 Hz with a 1280×960 resolution. The experiment was controlled by a Mac Pro running OS X10.7 and using MATLAB 2011b (MathWorks, Natick, MA) and the Psychophysics Toolbox (Kleiner et al., 2007). Eye movements were recorded by an Eyelink 1000 desk-mounted display placed at the base of the monitor. Participants sat 57 cm from the screen with their head stabilized by a chin rest.

Stimuli

All stimuli were white presented on a gray background (luminance 5 cd/m^2). Plus-shaped fixation and target markers (luminance 59 cd/m^2) were presented 10 degrees of visual angle (dva) horizontally separated from the center of the screen. The initial fixation location was surrounded by a small ring that also acted as the cue to saccade when it was extinguished. The localization probe was a 1.56×0.31 dva vertical line (luminance 37 cd/m^2) presented at one of five locations: the center of the screen, 3.33 or 6.66 dva left or right of center. It was presented for 10 ms.

Probes were presented in the area between the fixation location and the target location rather than on both sides of the target location as is common in studies of saccadic compression. The reason for this placement was that we were interested to measure whether perceived positions shifted in the direction toward the fixation location or toward the target location. For probe positions beyond the target location, both of these direction would be the same. Hence these stimuli would not be informative for our study.

Procedure

The three saccade conditions were carried out in separate testing sessions and were completed in the same order by each participant: double overlap saccade, single overlap saccade, and single step saccade. For some participants a single condition was also completed across separate testing sessions on different days. Figure 1 illustrates the sequence of events during the trials in each condition. The single overlap saccade condition involved the participant making a single rightward saccade while both the initial and fixation marker and saccade target remained on screen throughout. The double overlap saccade condition involved the participant making one rightward saccade and immediately thereafter also making a leftward return saccade while both the initial and fixation marker and the saccade target remained on screen throughout. Disappearance of the ring around the initial fixation marker acted as the cue to saccade. The single-step saccade condition involved the participant making a single rightward saccade. The disappearance of the initial fixation point and the simultaneous appearance of the saccade target acted as the cue to saccade. The step saccade condition is known to produce saccades with a shorter latency than the

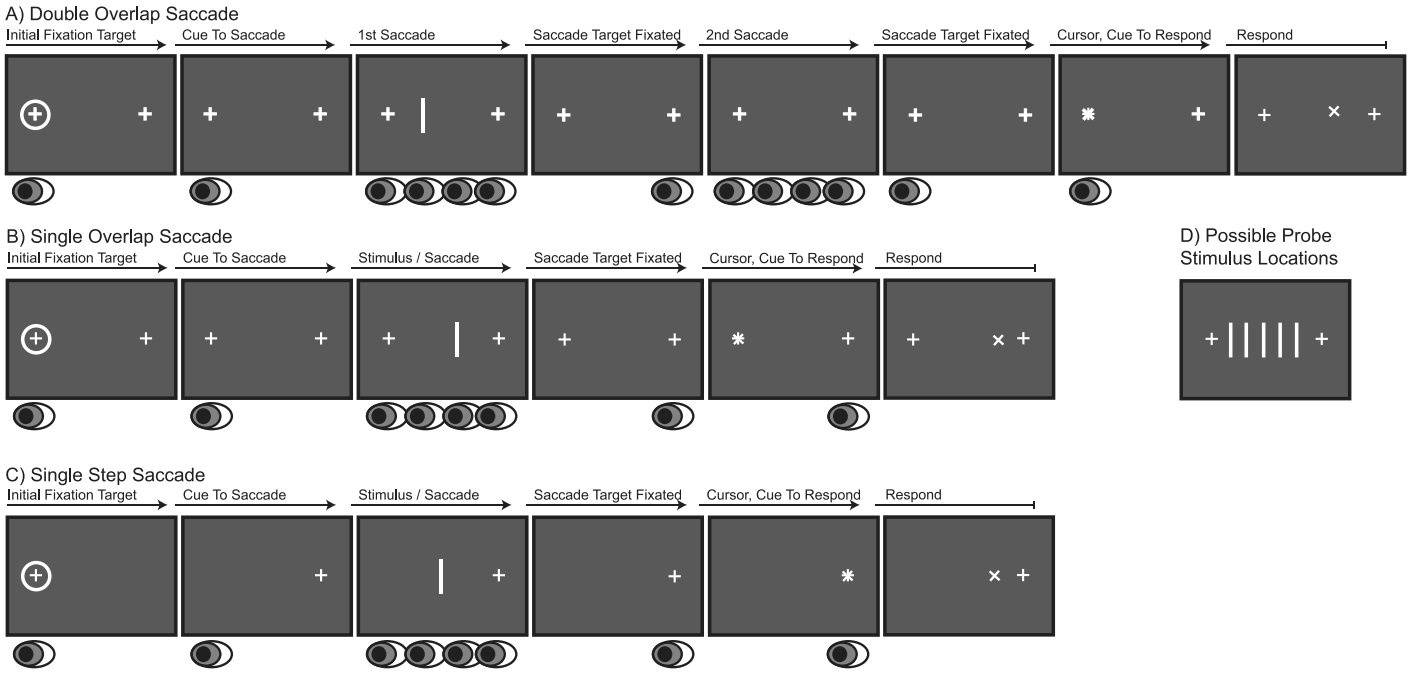

Figure 1. Sequence of events in the three conditions of Experiment 1.

overlap condition. In order to allow sufficient time for stimulus presentation before saccade onset, we asked participants in the step condition to make the saccade some time after the cue rather than immediately after the cue.

In all conditions, at the beginning of each trial participants were presented with the initial fixation marker and in the overlap saccade conditions also the saccade target. After fixating on the initial fixation point for a minimum of 1.5 s participants were cued to execute a 20 dva saccade to the saccade target and in the double saccade condition to immediately make a return saccade. A probe stimulus was flashed at any time from 300 ms prior to saccade onset to 300 ms postsaccade onset. At the end of the active trial period, the mouse cursor was presented and participants were asked to click on the perceived location of the probe. In case the participant did not see the probe due to saccadic suppression they were instructed to click the very top or bottom of the screen. In both the single and double overlap saccade conditions the cursor appeared at the location of the left fixation point. In the single-step saccade condition the cursor appeared at the location of the right saccade target. Participants completed an average of 550 trials in each condition, completed in blocks of 100 trials.

Results and Discussion

Prior to analysis of the perceptual localization data, each trial was analyzed to ensure participants executed a single large saccade to the saccade target after the cue was given. In the double overlap saccade condition, a further, large return saccade back to the initial fixation location was required after a short period of fixation. Trials not meeting these criteria were excluded from analysis. Trials where the participant clicked the very top or bottom of the screen were also excluded as indicating the participant did not see the target stimulus. Mean latencies were 236 ms ($SD = 27$ ms) in the double saccade overlap condition, 248 ms ($SD = 62$ ms) in the single saccade overlap condition and 196 ms ($SD = 32$ ms) in the step condition.

Trials were sorted according to the onset of the probe relative to the onset of the cued saccade (−300 to +300 ms). The measure of interest was the horizontal coordinate of the participant's localization of the probe. For each participant this location data was smoothed with a Gaussian-filter running average with a sigma of 30 ms. The localization through time, averaged across participants, can be seen in Figure 2.

In all three conditions, most localization curves show a mislocalization toward the fixation location that begins at around 200 ms before saccade onset and peaks between 100 and 50 ms before saccade onset, followed by a mislocalization toward the saccade target location peaking at around saccade onset (Figure 2). The mislocalization toward the saccade target location at saccade onset is consistent with the typical pattern of perisaccadic compression (Morrone, Ross, & Burr, 1997; Ross et al., 1997; Lappe et al., 2000; Kaiser & Lappe, 2002; Ostendorf et al., 2007). The preceding mislocalization toward the fixation location is consis-

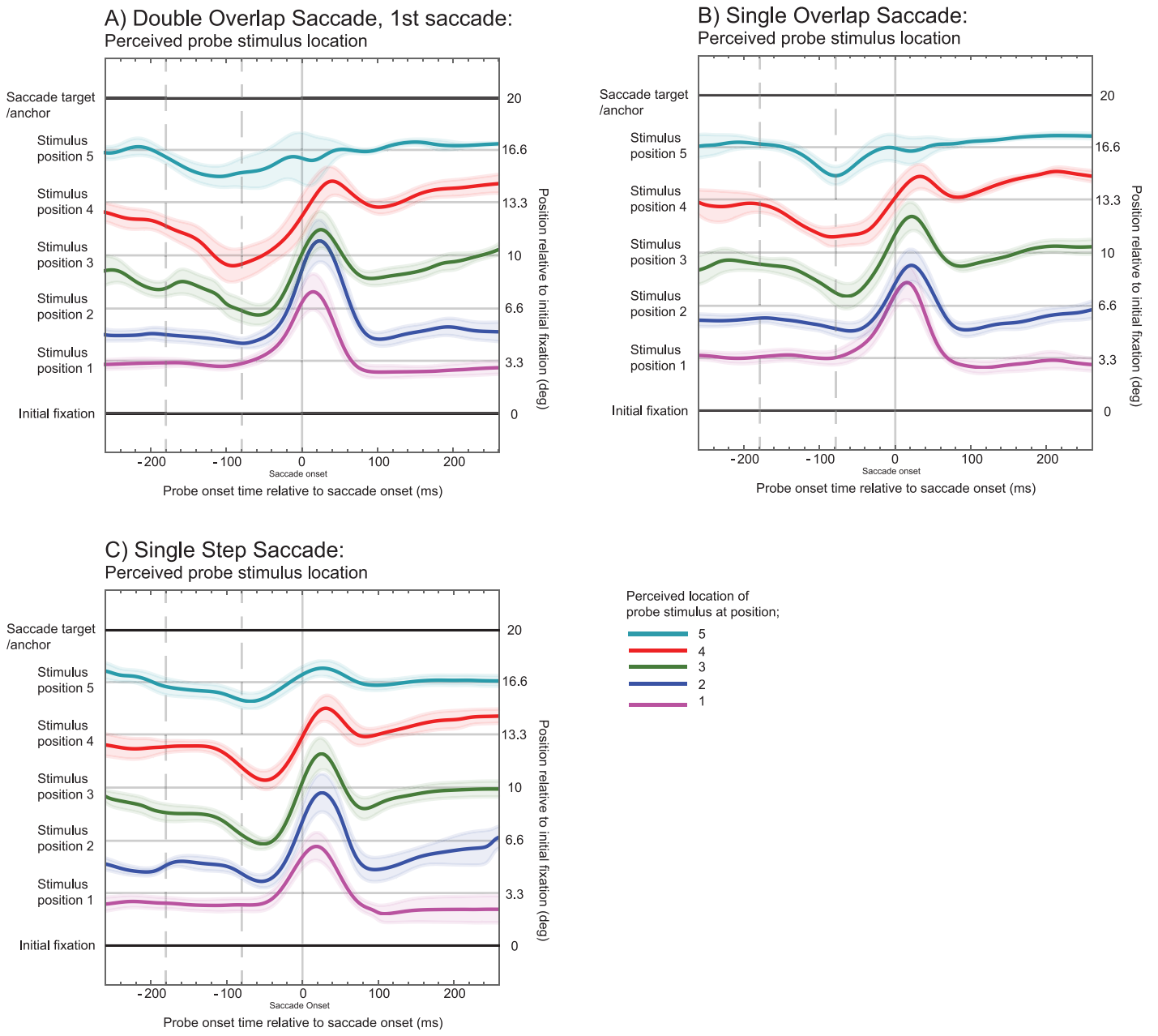

Figure 2. Results of three conditions of Experiment 1. The color lines show means and standard errors for the localization of the probe as a function of time relative to saccade onset. The vertical dashed lines at −180 and −80 ms show the points in time used in the analysis of the countercompression. All three conditions show a strong countercompression toward the initial fixation location for probe stimuli presented midway between the fixation and saccade target.

tent with localization errors toward the initial fixation point described by Haladjian et al. (2015) for double overlap saccades. Since this mislocalization occurred for single overlap saccades, it is not reliant on the presence of a return saccade.

To evaluate the data quantitatively we determined for each participant, condition, and probe position the values of the location report curve at 80 ms before saccade onset and compared it to a baseline location value taken from the location report curve at 180 ms before the saccade, the earliest point in time in which we had data from all participants in all conditions. The two time points used for data analysis are marked by vertical dashed lines in Figure 2. A repeated-measures analysis of variance (ANOVA) on these data with the factors condition (three levels), point-in-time (two levels), and probe position (five levels) showed no main effect of condition, $F(2, 10) = 1.77$, $p = 0.22$, $\eta_p^2 = 0.26$; a significant main effect of probe position, $F(4, 20) = 584.1$, $p < 0.001$, $\eta_p^2 = 0.99$; and a significant main

effect of point-in-time, $F(1, 5) = 36.4$, $p = 0.002$, $\eta_p^2 = 0.89$, confirming the mislocalization toward fixation at −80ms. There was also a significant interaction between point-in-time and probe position, $F(4, 20) = 31.3$, $p < 0.001$, $\eta_p^2 = 0.86$. The mislocalization toward fixation is strongest at the intermediate positions (2–4). These positions also show the strongest compression toward the saccade target at saccade onset.

The results of Experiment 1 show a mislocalization of briefly flashed stimuli against saccade direction within about 200 to 50 ms before saccade onset that somewhat mirrors the pattern of compression toward the saccade target at saccade onset. This "counter-compression" is not related to the planning of a return saccade since it also appears for single overlap and step saccades. Yet, it follows a time course that is related to the onset of the saccade, increasing from about 200 ms before and peaking around 80 ms before that onset.

If the compression toward the saccade target is driven by activity in oculomotor areas prior to saccade onset, then activity in these areas during fixation might produce a mislocalization toward the fixation location in a similar manner. The frontal eye fields and the superior colliculus contain fixation zones in which neurons are active during fixation (Munoz & Wurtz, 1993; Izawa, Suzuki, & Shinoda, 2009). This fixation activity commences before a saccade as activity of the target neurons increases. In the overlap task of Experiment 1, when the target appears in the periphery the participant is expected to keep fixation until the cue to release the saccade is given. In this situation, an increase in fixation activity might temporarily produce a compression toward fixation. Likewise, in the step task, we instructed our participants to make the saccade some time after the cue rather than immediately after the cue. This might also have led to a brief increase in fixation activity. In Experiment 2 we introduce a gap task that is known to release fixation activity. If our hypothesis is correct this should abolish the mislocalization toward the fixation point.

Experiment 2: Gap saccades in darkness

Two interlinked processes are involved in performing a saccade: release of fixation and the subsequent movement of gaze to the target. Normally these two processes are coordinated such that they occur at about the same time. The gap paradigm separates the two by introducing a temporal gap between the offset of the fixation point and the onset of the saccade target. In this case the activity of fixation neurons in the superior colliculus ceases during the gap (Dorris & Munoz, 1995). To test whether the mislocalization toward the fixation point is related to fixation activity, we measured presaccadic localization in a gap paradigm. To prevent any other visual reference on the fixation location besides the fixation point, we conducted this experiment in complete darkness.

Method

Participants

Four participants (three malese, between 23 and 53 years of age) completed the experiment. Three of the same participants had completed Experiment 1. The experiment was approved by Ethics Committee of the Department of Psychology and Sports Science of the University of Muenster and carried out in accordance with the guidelines in the Declaration of Helsinki.

Equipment

Stimuli were presented on a Eizo FlexScan 22-in. monitor running at 75 Hz with a 1152 × 864 resolution. The experiment was controlled by a Mac Mini running MATLAB (MathWorks) with the Psychophysics Toolbox (Kleiner et al., 2007). Eye movements were recorded by a fully infrared Eyelink 1000 desk-mounted system placed at the base of the monitor. Participants sat 57 cm from the screen with their head stabilized by a chin rest.

Stimuli

All stimuli were white presented on a black background. A filter was placed in front of the monitor to reduce light emitted from the black background to below 0.0006 cd/m^2 (Georg, Hamker, & Lappe, 2008). Fixation and target markers were small squares 0.4 × 0.4 dva (luminance 0.31 cd/m^2) located 10 dva horizontally separated from the center of the screen. The localization probe was a 1.56 × 0.31 dva vertical line (luminance 0.31 cd/m^2) presented at one of five locations: the center of the screen, 3.33 or 6.66 left or right of center. It was presented for 10 ms.

Procedure

Figure 3 illustrates the sequence of events during a trial. Throughout this experiment participants were seated in a completely dark room. All equipment lights were covered such that the only light in the room was emitted by the monitor. Use of the filter over the monitor made it possible to eliminate participants' ability to discern the edges of the monitor and any other visual structure in the room apart from the stimuli. Participants spent at least 5 min in total darkness before beginning data collection.

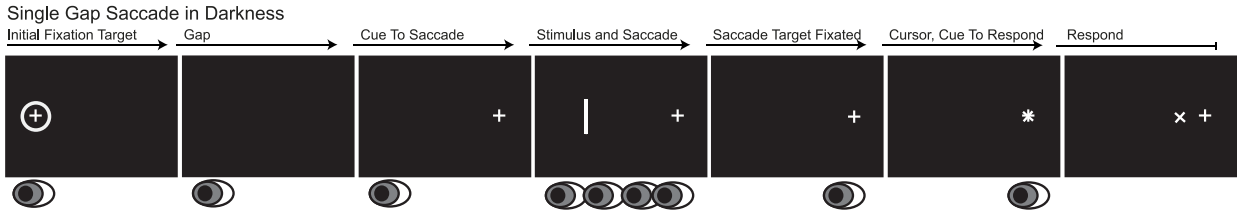

Figure 3. Sequence of events in the gap condition (Experiment 2).

Similar to the single reflexive saccade condition in Experiment 1, on each trial participants made a single rightward saccade. However, there was a 500-ms gap between the disappearance of the initial fixation point and the appearance of the saccade target. The saccade target stayed on the screen for the remainder of the trial. Participants were to execute a 20° saccade to the saccade target upon its appearance. A localization probe was presented at any time from 300 ms prior to saccade onset to 300 ms postsaccade onset. At the end of the active trial period, the mouse cursor was presented at the location of the right saccade target and participants were asked to click on the perceived location of the probe. Participants completed an average of 860 trials each, completed in blocks of 100 trials.

Results and discussion

Procedures for determining trial validity and calculating localization curves were the same as in Experiment 1. The localization curves (Figure 4) show the typical time course of perisaccadic compression peaking near saccade onset. However, mislocalization toward the fixation point is absent. An ANOVA with the factors point-in-time (80 vs. 180 ms before the saccade; see vertical dashed lines in Figure 4) and probe position (five probe locations) showed a significant main effect of position, $F(4, 12) = 402$, $p < 0.001$, $\eta_p^2 = 0.99$, but no effect of point-in-time, $F(1, 3) = 0.133$, $p = 0.74$, $\eta_p^2 = 0.04$, nor an interaction, $F(4, 12) = 2.62$, $p = 0.088$, $\eta_p^2 = 0.47$.

A comparison with Figure 2 shows that the time course of the localization in the gap condition differs from those of the single and double overlap saccade and the reflexive saccade, which are all very similar to each other. Notably, there is no mislocalization toward the fixation location while the compression toward the saccade target is of the same strength as that of the other conditions. We conclude that the early removal of the fixation point in the gap condition abolishes the mislocalization toward fixation, consistent with our hypothesis.

Removing the fixation point in the gap paradigm usually results in a reduction of saccade latency (Fischer & Boch, 1983). In our experiment, mean latency was 298 ms ($SD = 71$ ms) and thus longer than in the conditions of Experiment 1. This rather long latency might be explained by the low luminance of the target stimulus. Because we covered the monitor with a dark foil to reduce visibility of the luminous background edges of the monitor, all visual stimuli were reduced in luminance by about two log units (Georg et al., 2008). Saccade latency increases with decreasing luminance even in the gap paradigm (Marino & Munoz, 2009).

Experiment 3: Masked at fixation

Mislocalization of flashed stimuli can also occur during fixation. Some studies have reported that a briefly flashed stimulus followed by a mask is mislocalized toward a visual anchor in a pattern similar to saccadic compression (Zimmermann, Born, et al., 2014; Born, Krüger, Zimmermann, & Cavanagh, 2016). To test whether we could reproduce this effect, and whether it would also produce mislocalization toward the fixation point, we measured localization after backwards masking, closely following the experimental protocol of Zimmermann, Born, et al. (2014).

Method

Participants

Five participants, (two females, between 23 and 53 years of age) completed this experiment. Four participants had completed Experiment 1, one of whom also completed Experiment 2. The experiment was approved by the Western Sydney University Human Research Ethics Committee and carried out in accordance with the guidelines in the Declaration of Helsinki.

Equipment

Stimuli were presented on a Sony Trinitron CRT monitor running at 100 Hz with a 1280×960 resolution. The experiment was controlled by a Mac Pro running OSX 10.7, using MATLAB 2011b (MathWorks) and the Psychophysics Toolbox (Kleiner et al., 2007). Eye movements were recorded by an

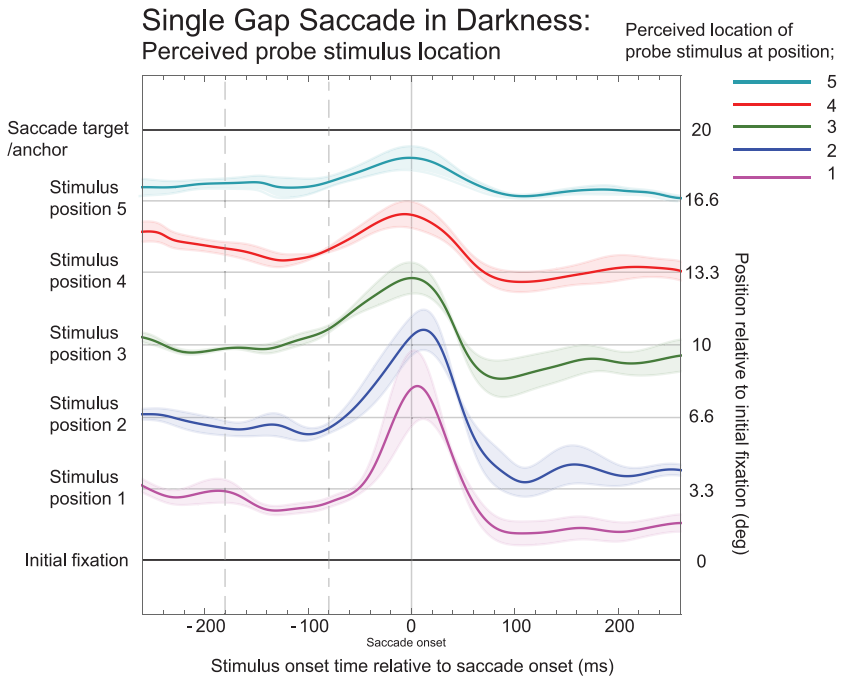

Figure 4. Results of the gap condition (Experiment 2). The color lines show means and standard errors for the localization of the probe as a function of time relative to saccade onset. The vertical dashed lines at −180 and −80 ms show the points in time used in the analysis of the countercompression. While compression toward the saccade target at saccade onset is similar to that in Experiment 1, there is no earlier mislocalization toward the fixation point.

Eyelink 1000 desk-mounted display placed at the base of the monitor. Participants sat 57 cm from the screen with their head stabilized by a chin rest.

Stimuli

All stimuli were white presented on a gray background (luminance 5 cd/m^2). Plus shaped fixation markers (luminance 59 cd/m^2) were presented 10 dva horizontally separated from the center of the screen. The initial fixation location was surrounded by a small ring which also acted as somewhat of a cue to probe and mask stimulus onset it was extinguished. The probe was a 1.56×0.31 dva vertical red line (luminance 35 cd/m^2) presented at one of five locations: the center of the screen and 3.33 or 6.66 left or right of center. It was presented for 10 ms. The mask was full screen, white noise comprised of 4 dva square blocks (average luminance 30 cd/m^2) presented for 50 ms.

Procedure

Figure 5 illustrates the sequence of events during a trial. Participants fixated on the left initial fixation marker while the right fixation marker remained on screen throughout. The disappearance of the ring surrounding the initial fixation acted as the cue to the onset of the mask stimulus. The mask appeared at either 160 or 320 ms after the cue. This timing was designed to replicate the execution of a saccade after the cue. A target stimulus was presented at any time from 300 ms prior to mask onset to 300 ms postmask onset. At the end of the active trial period, the mouse cursor was presented at the location of the left fixation point and participants were asked to click on the perceived location of the target stimulus. In case they did not see the stimulus due to the backwards masking, they were instructed to click the very top or bottom of the screen. Participants completed an average of 328 trials.

Results and discussion

We first determined whether the mask was effective in masking the stimuli. For this analysis we computed a continuous estimate of the percentage of trials in which the participants had seen the stimulus by a running average with a 30-ms window. Figure 6A shows that detection rate of the stimulus drops to about 50% around the time of the mask onset. Thus, at this time about half the probe stimuli were not seen by the participants. This indicates that the masking worked as expected and that it still produced a suitable number of trials for localization analysis.

We next determined the perceived positions of those stimuli that were seen by the participant, also with a running average with a 30-ms window. Figure 6A

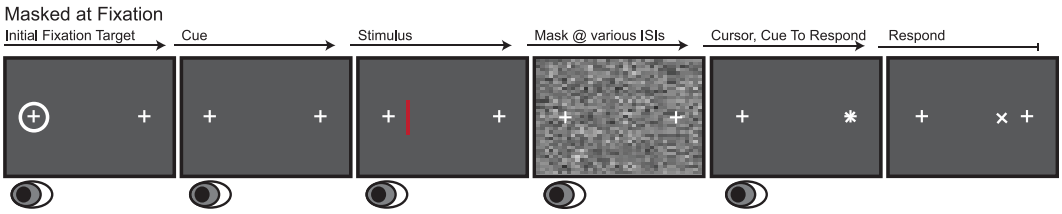

Figure 5. Sequence of events in the masking experiment.

shows that perceived locations remained constant over time. The localization curves neither showed mislocalization toward the target nor toward the fixation point. We conclude that in the conditions of our experiment no masking-induced compression occurred.

General discussion

The results of this study show that a presaccadic mislocalization toward the initial fixation point occurs in addition to the well-established perisaccadic compression toward the saccade target. This mislocalization begins about 150 ms before the saccade and peaks between 100 and 50 ms before saccade onset.

Haladjian et al. (2015) first reported evidence for compression toward fixation in a study involving a sequence of first outward and then return saccades. In that study, the fixation marker was also the target of the return saccade so that the compression might have been driven by the preplanning of the return saccade. The results of Experiment 1 show that this is not the case. Mislocalization toward the fixation point also occurred for single overlap and single step saccades. Since mislocalization toward fixation was not different between the overlap and step conditions, Experiment 1 further showed that mislocalization was not conditional to the presence of the fixation marker at the time of the flash or later.

Experiment 2 tested whether the holding of fixation, i.e., the presence of ongoing fixation activity in the oculomotor system, was needed to produce mislocalization toward the fixation locations. To test this, Experiment 2 employed the gap paradigm in which the participants were seated in the dark and the fixation marker was removed 500 ms before the onset of the saccade target. This condition is known to release fixation activity during the gap interval (Dorris & Munoz, 1995). Mislocalization toward fixation was much weaker in this condition in comparison to the three conditions of Experiment 1. We therefore conclude that mislocalization toward fixation is driven by fixation activity in the oculomotor system.

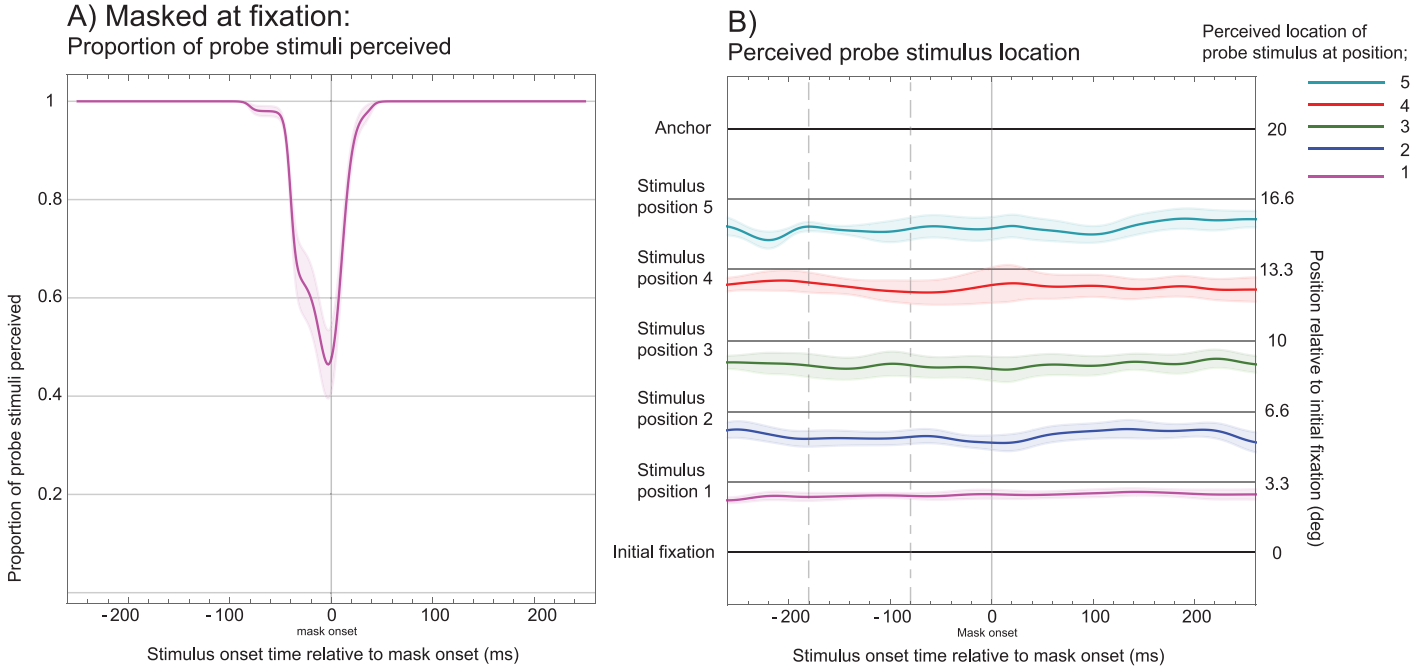

Figure 6. Results of the masking experiment. (A) Rate of probe detection. (B) The color lines show means and standard errors for the localization of the probe as a function of time relative to mask onset.

This finding is in line with a model of perisaccadic compression that explains mislocalization by the action of a feedback signal of the oculomotor plan on neural sensitivity in visual maps (Hamker et al., 2008; Zirnsak, Lappe, & Hamker, 2010; Zirnsak, Steinmetz, Noudoost, Xu, & Moore, 2014). In this model, preparatory saccade target information from the frontal eye fields (or the superior colliculus), i.e., the oculomotor plan, is fed back to visual cortical maps to enhance responses to visual input in the saccade target area. This increases spatial resolution in this area and provides benefits to visual processing that resemble attention shifts to the saccade target. At the same time, it distorts the population activity in the map such that briefly flashed stimuli appear closer to the target, thereby accounting for perisaccadic compression.

This model can be applied to the present data when considering that fixation is an active process that coincides with neural activity in the rostral pole of the superior colliculus (Munoz & Wurtz, 1992, 1993; Everling, Pare, Dorris, & Munoz, 1998; Krauzlis, Dill, & Kornylo, 2002) as well as with the activity of fixation neurons in FEF (Izawa et al., 2009) and many other oculomotor-related areas (Sakata, Shibutani, & Kawano, 1980; Bremmer, Distler, & Hoffmann, 1997; Read & Siegel, 1997; Ben Hamed & Duhamel, 2002). The same feedback gain modulation as proposed by Hamker et al. (2008) would then predict a distortion of the encoded flash position toward the fixation point at times in which fixation activity is high. Indeed, stimuli that are briefly flashed during fixation often appear closer to the fixation point than they truly are (Müsseler, van der Heyden, Mahmud, Deubel, & Ertsy, 1999; Lappe et al., 2000), a bias that may be produced by the fixation activity. In the overlap and step conditions, it seems likely that fixation activity is transiently increased in order to keep with instructions to not immediately look at the salient target in the periphery. Under the above model, such a transient increase in fixation activity would produce the observed transient countercompression. In contrast in the gap condition, fixation activity is known to be removed (Dorris & Munoz, 1995) and countercompression should not be observed, also consistent with the above model. Thus, our proposed model suggests that compression and countercompression are the same process with different attractive locations being active at different times.

Fixation neurons in the SC that are believed to be responsible for keeping fixation until a new saccade is prepared are also involved in the generation of microscopic saccades during fixation, and therefore resemble those neurons that are responsible for generating saccades. Since perisaccadic compression is also observed for such microsaccades (Hafed, 2013), it is likely that activity from fixation neurons is fed back to visual areas in the same manner as that of neurons that generate saccades. This feedback can be expected to generate compression toward the fixation location during times in which fixation activity is strong.

The time course of the mislocalizations in our experiments is consistent with this. Before a saccade is started, fixation activity has to cease and targeting activity has to build-up. This typically happens within 150 ms before saccade onset. The change in mislocalization over this time is consistent with the transition from fixation-related activity to target location–related activity during the time prior to saccade onset.

Two questions arise from the time course we observed for mislocalization toward fixation. First, why is it that mislocalization toward fixation appears to increase from ~200 ms before the saccade to ~80 ms before the saccade, despite the participant presumably fixating throughout this time. Second, why was the mislocalization toward fixation not reported in earlier studies on perisaccadic compression.

The second question has a simple answer. Prior studies on perisaccadic perception have all used reflexive saccades in the step paradigm. Participants were instructed to make a saccade as soon as the target appeared. This typically produces saccade latencies in the range between 150 and 200 ms and would not allow for sufficient baseline measurement before the saccade. In fact, prior studies have used the time up to 120 ms before saccade onset as the baseline for the localization. In the overlap paradigm (Experiment 1) latencies are longer and allow measurement of a broader picture of localization before saccades. To allow the same for the step condition in Experiment 1, we instructed participants not to react immediately on target appearance but to keep their gaze briefly on the initial fixation location and delay the saccade to the target.

These considerations also point to a possible answer to the first question, why there is an increase in fixation-related mislocalization from 200 to 80 ms before saccade onset. The instruction to briefly delay the saccade in the step condition might have led to a transient increase in fixation activity to counter the urge to move after target onset. Such a transient increase would, in our model, produce a transient increase in mislocalization. Likewise, in the overlap condition the continued presence of the fixation marker is known to increase latencies over the step paradigm. This is also likely to involve an increase in fixation activity until the cue to saccade is given.

An alternative explanation for the time course and direction of mislocalization before the saccade may presume that the modulation imposed by the saccade plan is biphasic in time, consisting first of a repulsion of the flash location from the target location and later of an attraction. However, such a model would not explain why the mislocalization toward fixation, i.e.,

the repulsion in this model, did not occur in the gap condition.

The mislocalization, moreover, does not appear to be related to saccadic suppression (reduced contrast sensitivity prior to saccade onset). Saccadic suppression starts only about 50 ms before a saccade (Diamond, Ross, & Morrone, 2000) while the mislocalization toward fixation appears much earlier. To the extent that both saccadic compression and saccadic countercompression are mediated by fixation and saccade motor plans, this would imply that saccadic suppression and saccadic compression are unrelated to each other.

Spatial uncertainty of eye position is also unlikely to account for the countercompression observed here. An anticipatory change in the extraretinal eye position signal toward the saccade target would appear to shift perceived position toward the target location, not toward fixation (Honda, 1991; Pola, 2004). At 80 ms prior to saccade onset it seems unlikely that there is any mismatch between the extra retinal eye position signal and the actual position of the eye.

We did not find any compression induced by visual masking in Experiment 3. The reason likely lies in the time course of our experiments. While we have attempted to closely follow the procedures of Zimmermann, Born, et al. (2014), one notable difference is that our experiment involved an overlap design in which both the fixation marker and the target/anchor were present on the screen from the beginning of the trial. In the experiments of Zimmermann, Born, et al. (2014), the anchor appeared and was followed by the mask with a timing typical for reflexive saccades. As Zimmermann, Fink, and Cavanagh (2013) have shown, the masking-induced fixational compression disappears if the anchor is presented more than 200 ms before the probe, as was the case in the present study. The lack of masking-induced compression in our study is thus consistent with the findings of Zimmermann et al. (2013). A possible explanation put forward for their masking-induced compression is that the sudden appearance of the target induced a covert motor plan or attention shift that would trigger a compression by a similar mechanism as saccades. Despite our attempt at inducing a shift of attention to the target location on the opposite side of the screen from fixation by having the response cursor appearing at that location, our experiment lacks the sudden onset of the anchor stimulus. Given this, we may not have been successful in inducing a covert motor plan in this study. The observation that saccades in the overlap paradigm did produce compression (Experiment 1) emphasizes the differences between masking-induced and saccade-induced compression.

A common finding between masking and saccades is that both reduce visibility of the probe stimulus. Thus, low visibility has been discussed as a common requirement of compression. Indeed, low visibility increases compression (Michels & Lappe, 2004; Georg et al., 2008), as is predicted by the model of Hamker et al. (2008). However, in our present countercompression paradigm we present stimuli more than 100 ms before the saccade while they are just as visible as when no eye movement is planned. Hence we believe that countercompression is sufficiently explained by needing to exert significant control over where the eye is fixating while also planning to move it at some point in the more distant than usual future.

Conclusion

In summary, we have demonstrated that a presaccadic countercompression toward the initial fixation location can occur at approximately 80 ms prior to saccade onset. This countercompression occurs even when a single saccade is planned whether or not the initial fixation point remains on screen while the saccade is cued and executed, suggesting the compression does not require that a saccade is planned toward that location and doesn't require a continuous visual input at that location. This suggests the compression is mediated by fixation related motor plan activity occurring ahead of saccade onset. This is supported by the finding that a saccade made in darkness after a 500-ms gap period does not produce strong countercompression. In this gap design the fixation related motor plan activity is found to weaken ahead of saccade onset. Finally, a lack of compression in the masked condition suggests a strong covert motor plan is required to produce saccade-like compression. We conclude that the interaction between both saccade- and fixation-related ocular motor plan activity and perception is strong and capable of producing complex distortions of the visual field.

Keywords: localization, perceptual stability, saccadic compression

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