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Alteration of Visual Perception prior to Microsaccades

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SUMMARY

Gaze fixation is an active process, with the incessant occurrence of tiny eye movements, including microsaccades. While the retinal consequences of microsaccades may be presumed minimal because of their minute size, a significant perceptual consequence of these movements can also stem from active extraretinal mechanisms associated with corollaries of their motor generation. Here I show that prior to microsaccade onset, spatial perception is altered in a very specific manner: foveal stimuli are erroneously perceived as more eccentric, whereas peripheral stimuli are rendered more foveal. The mechanism for this perceptual "compression of space" is consistent with a spatially specific gain modulation of visual representations caused by the upcoming eye movements, as is hypothesized to happen for much larger saccades. I then demonstrate that this perimicrosaccadic perceptual alteration has at least one important functional consequence: it mediates visual-performance alterations similar to ones classically attributed to the cognitive process of covert visual attention.

INTRODUCTION

Visual and cognitive neuroscience experiments rely almost exclusively on gaze fixation to avoid several ambiguities caused by eye movements. First, eye movements alter retinal images. Second, eye movements are not independent of behavioral state, which makes it impossible to average out their retinal consequences in experiments. Third, eye movements are associated with active, extraretinal mechanisms, such as "saccadic suppression" (Zuber and Stark, 1966) and "saccadic compression" of space (Ross et al., 1997), that dramatically alter perception even well before movement onset.

The requirement of "gaze fixation" per se does not necessarily eliminate these sources of ambiguity, because tiny eye movements continue to occur (Barlow, 1952). "Microsaccades" constitute one component of these movements, and they are called such because they are scaled-down versions of larger saccades (Zuber et al., 1965). Microsaccades not only alter retinal images (Verheijen, 1961), but they are also not random and instead are biased by stimulus presentations routinely used in experiments (Engbert and Kliegl, 2003; Hafed and Clark, 2002; Hafed et al., 2011). The results I describe below show that microsaccades are also associated with active extraretinal mechanisms that significantly alter spatial perception even before they occur. This perceptual alteration can influence visual locations much farther away from the movements' endpoints. Thus, even if the movements themselves cause small retinal-image changes, the active mechanisms associated with them can still significantly alter vision.

In the experiments described below, I also tested for a functional consequence of perimicrosaccadic changes in perception. Consider, for example, the cognitive process of "covert visual attention" (Carrasco et al., 2002; Yeshurun and Carrasco, 1998). Covert attention is believed to change spatial perception, altering visual acuity (Carrasco, 2011; Carrasco et al., 2002), resolution (Carrasco, 2011; Carrasco and Frieder, 1997; Carrasco et al., 2002; Yeshurun and Carrasco, 1999), and even "appearance" (Carrasco, 2011; Carrasco et al., 2004). Such alteration is classically uncovered during "spatial cueing" paradigms, in which a spatial location is first cued with a brief visual stimulus and then perceptual performance is probed shortly afterward (Carrasco et al., 2000, 2002, 2004; Nakayama and Mackeben, 1989; Posner, 1980; Yeshurun and Carrasco, 1998). Even though it is assumed that eye movements do not occur in these paradigms (Carrasco, 2011), microsaccades still take place, and they do so in a remarkably reflexive, machine-like manner (Hafed et al., 2011). Thus, if microsaccades are preceded by altered percepts, as I show in this paper, then might it be the case that changes in perception attributed to covert attention are simply mediated by microsaccades? In the second half of this paper, I provide evidence that supports this notion and potentially explains a recent paradoxical observation that covert attention did not shift when microsaccades did not occur (i.e., in most trials; see Figure 9 in Hafed and Clark, 2002).

RESULTS

Foveal Stimuli Are Mislocalized in the Direction of Upcoming Microsaccades

I first asked whether spatial perception is altered around the time of microsaccades. I performed an experiment conceptually similar to those used to study perception around large saccades (Ross et al., 1997). The rationale behind these experiments is to present a brief probe stimulus around the time of eye movement and to ask the subjects to localize this probe. If perception is

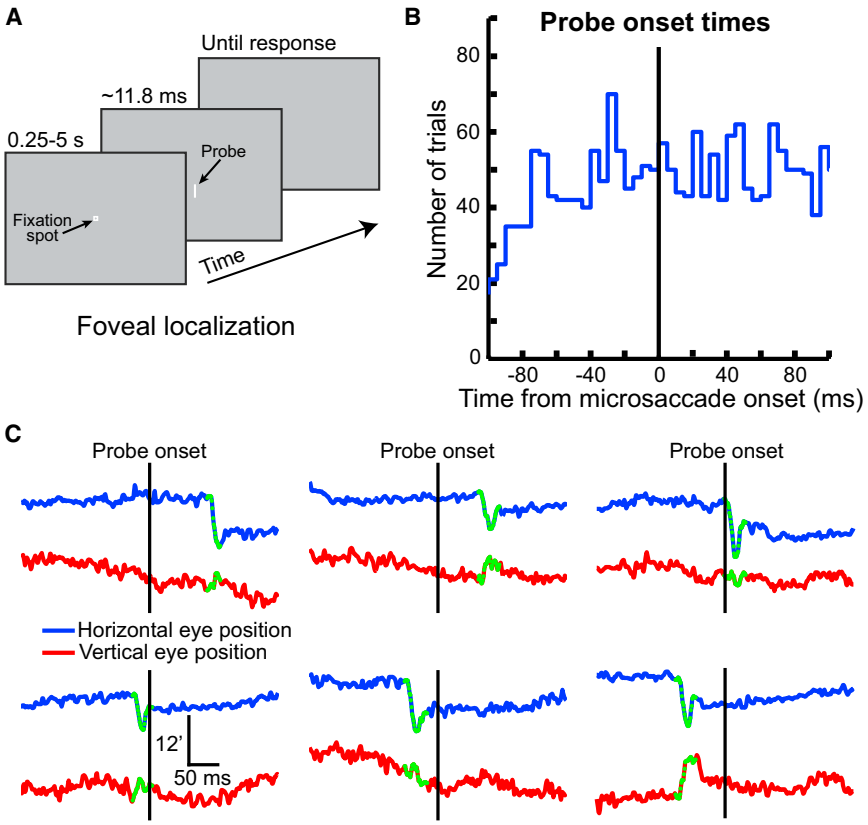

Figure 1. Studying Perception around the Time of Microsaccades

(A) Subjects fixated a central spot for a random time, after which a brief probe was presented. Subjects reported the perceived horizontal position of the probe (right or left of the spot).

(B) Distribution of probe times relative to microsaccade onset across trials. The task design resulted in a relatively uniform distribution of probes near microsaccades, which allowed me to analyze percepts at different times relative to the movements.

(C) Sample individual trials showing the relative timing between probe and microsaccade onset. Each panel shows horizontal (blue) and vertical (red) eye positions. Portions of eye position highlighted in green are individual microsaccades. The probe (black vertical line) could appear at different times before (top row) or after (bottom row) microsaccades. Upward deflections in the plots denote rightward or upward eye movements. See also Figure S6.

momentarily altered near eye movements, the subjects will mislocalize the stimulus. In this case, I simply asked the subjects to fixate a central spot and I presented the probe at a completely random time, regardless of whether a microsaccade occurred or not (Figure 1A). After probe onset, the subjects reported whether its horizontal position was displaced to the right or left of the central fixation spot. Extreme randomization in the timing of the probe (Figure 1A) helped prevent the subjects from anticipating its onset, which would have reduced microsaccade rate (Hafed et al., 2011; Pastukhov and Braun, 2010).

Unbeknownst to the subjects, they generated microsaccades, and across trials my task resulted in a uniform distribution of probe times relative to microsaccade onset (Figure 1B). Thus, in post hoc analyses, I could identify trials in which the probe appeared during a specific time window relative to the movement, and I could then ask how spatial localization was altered during this time window. Examples of such trials can be seen in Figure 1C, which plots horizontal and vertical eye positions from a sample subject. In each sample trial shown, a microsaccade smaller than ~12′ occurred near probe onset, and across trials this microsaccade could occur either before or after the probe. Thus, after collecting many trials, I was able to analyze perceptual localization during perimicrosaccadic intervals.

Even before microsaccades occurred, spatial localization was altered, suggesting an active extraretinal mechanism that modifies perception before miniscule eye movements. Consider, for example, Figure 2A. In this analysis, I plotted the proportion of "right" responses given by subjects when the probe appeared at a horizontal displacement of 0′ or 4.5′ to the right or left of the fixation spot. When I measured the subjects' percept on trials with no microsaccades within ±175 ms from probe onset (Figure 2A, black), I observed the expected veridical percept: the subjects easily discriminated between rightward and leftward probe displacements, and they were guessing for no displacements (Figure 2A, black curve). However, when the probe appeared within 50 ms before a rightward microsaccade (Figure 2A, blue curve), even the leftward probe location was more often perceived as displaced rightward (i.e., there were more "right" reports than without the upcoming movement; $p < 0.05$, χ^2 test). When the microsaccade was leftward instead (Figure 2B, red curve), the rightward probe location was more often perceived as displaced leftward (i.e., there were less "right" reports than without the movement; $p < 0.05$, χ^2 test). Thus, within 50 ms before microsaccade onset, brief foveal probes were mislocalized in the direction of the upcoming miniscule eye movement.

This perceptual mislocalization was consistent for nearby foveal locations. I repeated the above analysis, but now for all locations tested, by constructing full psychometric curves of perceptual reports as a function of probe location. The black curve in Figure 2C shows data from trials with no microsaccades within ±175 ms from probe onset, and the blue curve shows data from trials in which the probe appeared within 50 ms before microsaccade onset. Note that in this analysis, I remapped the probe locations and perceptual reports to a coordinate system relative to microsaccade direction, in order to combine rightward and leftward movements (see Experimental Procedures). As can be seen, the point of subjective equality (PSE) in the blue curve was shifted such that probes were more often perceived as being displaced in the direction of the upcoming microsaccade than when no movements occurred ($p < 0.05$ for the difference

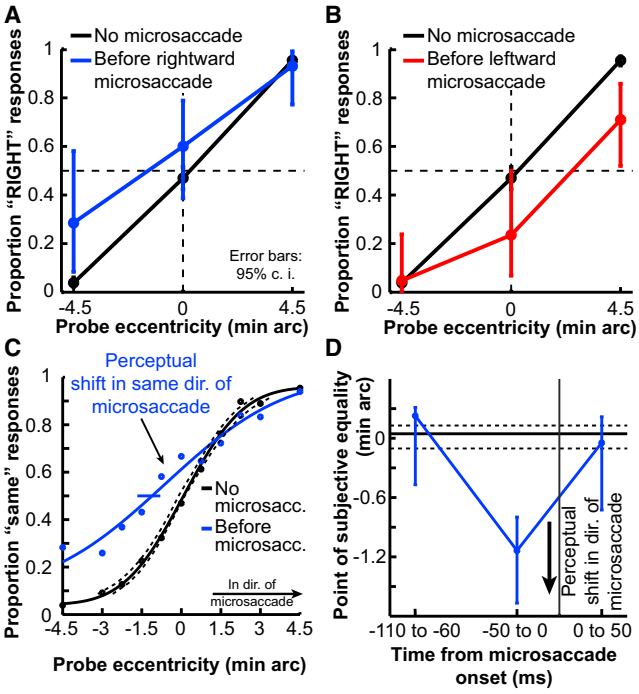

Figure 2. Mislocalization of Foveal Stimuli in the Direction of Upcoming Microsaccades

(A) Proportion of "right" responses as a function of probe location. When the probe appeared without microsaccades (black curve), a veridical percept was observed. The subjects had no difficulty in correctly discriminating between right and left probes; they were guessing for zero probe eccentricity. However, when the probe appeared <50 ms before a rightward microsaccade (blue curve), there was an increase in "right" responses even for leftward probes.

(B) The same analysis before a leftward microsaccade (red curve) shows a decrease in "right" reports even for rightward probes. Thus, (A) and (B) show a mislocalization in the direction of an upcoming movement.

(C) Psychometric curves of perceptual localization with (blue curve) and without (black curve) upcoming microsaccades. The blue curve was obtained from trials in which the probe appeared <50 ms before microsaccade onset. A mislocalization in the direction of the upcoming movement occurred, as evidenced by the shift in the blue psychometric curve. Note that I remapped the physical probe locations (*x* axis) and subjective reports (*y* axis) from absolute coordinates (left/right) to ones relative to microsaccade direction to clarify the result (see Experimental Procedures).

(D) Points of subjective equality from psychometric curves as in (C) but collected at different times relative to microsaccades. Black shows the no-microsaccade case (black curve in C). The effect in (C) was time-locked to microsaccade onset. All error bars indicate 95% CIs.

See also Figure S1.

between blue and black PSEs, bootstrapping test; Wichmann and Hill, 2001a, 2001b). Thus, the foveal space I tested was distorted in a very specific manner within 50 ms before microsaccades occurred.

When I next probed perceptual localization during other time windows, I found that this effect was time-locked to microsaccades. In Figure 2D, I plot the PSE from psychometric curves similar to those in Figure 2C but collected during different time windows (Figure 2D, blue). I also plot the PSE from the baseline condition without microsaccades (Figure 2D, black). As can be seen, mislocalization was strongest in the 50-ms period before microsaccade onset ($p < 0.05$, bootstrapping test) and it disappeared during other times. This time course was similar to that observed in classic saccadic compression experiments (Ross et al., 1997) in which probes were also mislocalized before saccades, except that it now happened for miniscule movements. Moreover, this effect was robust across individual subjects (Figure S1A available online), and it was also not due to subjects becoming momentarily blind to the probe, possibly through microsaccadic suppression (Zuber and Stark, 1966), because I used bright probes, and also because I tested the subjects' ability to see the probes in a separate control experiment (Figures S1B and S1C).

Thus, microsaccade generation was associated with a concomitant mislocalization of foveal visual stimuli immediately before movement onset.

Peripheral Stimuli Are Mislocalized Opposite the Direction of Upcoming Microsaccades

I hypothesized that the pattern of mislocalization described above is a correlate of saccadic compression (Ross et al., 1997), in which probes are mislocalized as if space is compressed toward the saccade endpoint. In the case of microsaccades, compression would be toward an imaginary, virtual goal associated with the tiny movements. If this is the case, then repeating the same perceptual localization experiment above, but now in the periphery, should reveal qualitatively different results, i.e., it should reveal mislocalization in the opposite direction from a microsaccade because compression toward the movement's virtual goal would now be back toward the fovea. I thus tested localization at 5° (Figure 3A), an eccentricity that is much larger than the actual microsaccade endpoints (median: 12′). In this experiment, subjects fixated the same central spot and the probe was presented near a reference spot located at 5°. The subjects reported whether the probe was displaced to the right or left of the eccentric reference. Again, the subjects consistently mislocalized probes relative to the no-microsaccade baseline if these probes appeared immediately before the onset of a microsaccade directed toward their location (Figure 3B; black and blue curves have different PSEs; $p < 0.05$, bootstrapping test). This time, however, the mislocalization was opposite the movement direction, consistent with a foveally shifted representation of space right before the tiny eye movement. That is, physically more eccentric probe locations were reported more often as being "more foveal than the reference spot" when these locations were tested within 50 ms before microsaccades than when they were probed without them. This also occurred during an earlier time window centered on 85 ms before microsaccade onset (Figure 3C, $p < 0.05$, bootstrapping test), and my time-course analysis of Figure 3D showed that this phenomenon, again just like in Figure 2D, was time-locked to the movement.

Taken together, Figures 1, 2, and 3 therefore suggest that microsaccades are associated with a compression of visual space similar to the well-known saccadic compression associated with much larger saccades (Ross et al., 1997). In the case of microsaccades, this phenomenon amounts to foveal locations being misperceived as displaced in the direction of an upcoming movement and peripheral ones as shifted back toward the fovea.

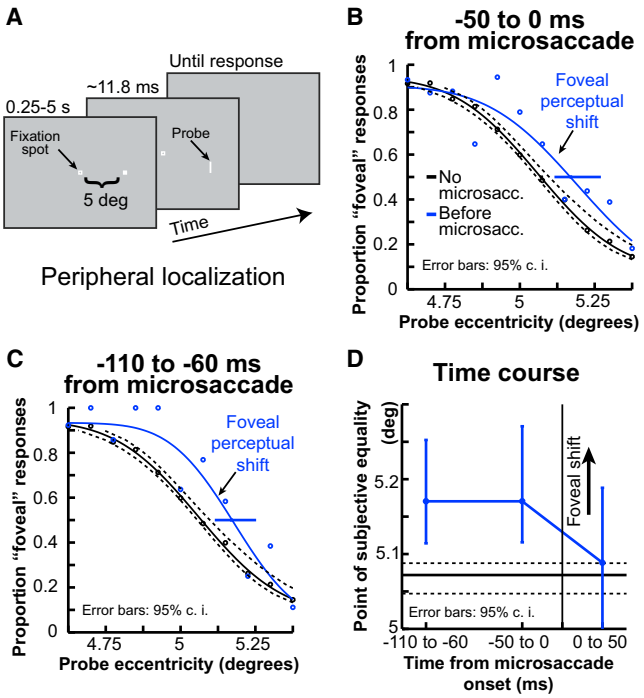

Figure 3. Mislocalization of Peripheral Stimuli in the Opposite Direction of Upcoming Microsaccades

(A) The same localization task as in Figure 1, but now testing perception in the periphery. Subjects fixated the central spot, and there was a second reference spot at 5°. After a random time, a probe appeared near the eccentric spot and the subjects reported its horizontal displacement relative to the spot.

(B and C) Similar to Figure 2C, but for localization at 5° and at two 50-ms windows centered on −25 (B) and −85 (C) ms from microsaccade onset. Subjects consistently perceived probes as being more foveal relative to the no-microsaccade case (black curve).

(D) Similarly to Figure 2D, the effect was time-locked to microsaccades. Error bars indicate 95% CIs.

See also Figure S4.

In the following section, I test whether this similarity is plausible by implementing a neurally based model of saccadic compression and applying it to microsaccades. In the section following that, I test the generality of this phenomenon during other visual conditions in which microsaccades occur but are assumed to be inconsequential.

A Model of Saccadic Compression Can Describe Microsaccadic Compression

Hamker and colleagues (2008) hypothesized that spatial compression for large saccades (Ross et al., 1997) may be thought of as reflecting the influence of saccade motor generation on visual sensitivity. The concept of their model is simple: movement-related neural activity (say, an efference copy from the superior colliculus [SC]; Sommer and Wurtz, 2002) increases the gain of visual neurons responding to the flashed probe in a spatially specific manner (Hamker et al., 2008). For example, for a 20° saccade, visual neurons representing 20° retinal eccentricities are gain modulated by the oculomotor activity generating the 20° eye movement. Such gain modulation is sufficient to explain perisaccadic compression effects for large saccades, and the model is also attractive because the same modulation additionally explains the relationship between saccades and attention (Hamker et al., 2008), as I also test later in this work for microsaccades. Moreover, the model is strongly supported by neuronal evidence of changes occurring in visual receptive fields (RFs) around the time of large saccades (Tolias et al., 2001; Zirnsak et al., 2011). Given the conceptual similarity of my results to saccadic compression, I asked whether a similar idea is sufficient to explain microsaccadic compression, especially since microsaccade generation in the SC is similar to larger saccades (Hafed, 2011; Hafed et al., 2009; Hafed and Krauzlis, 2012).

I implemented a simplified, one-dimensional (1D) version of the Hamker et al. (2008) model at a snapshot of time in which the oculomotor system (specifically the SC) is preparing for an upcoming microsaccade. My goal was to test whether the concepts hypothesized for saccadic compression can qualitatively explain microsaccadic compression.

The details of the model are described in Experimental Procedures. Briefly, microsaccade-related activity from the SC implements a gain increase on neurons representing the probe location. I simulated this gain signal according to the published literature on microsaccade generation in the SC (Goffart et al., 2012; Hafed, 2011; Hafed et al., 2009; Hafed and Krauzlis, 2012). In particular, according to this literature, SC neurons involved in microsaccade generation are tonically active during fixation, and they exhibit a spatially specific premicrosaccadic increase that is almost identical to presaccadic increases (Hafed et al., 2009; Hafed and Krauzlis, 2012). Thus, any microsaccade-related gain signal derived from these neurons must reflect the neurons' changes from baseline. I thus assumed in my model that the microsaccade-related gain signal that affects visual representations is derived based on the change in SC activity that is specific to microsaccades (Figure 4C, equal to the difference between panels A and B). Moreover, I used the published literature on the spatial profile of population activity in the foveal/parafoveal SC during the presence of a foveal goal to estimate the spatial extent of Figures 4A–4C (Goffart et al., 2012; Hafed, 2011; Hafed et al., 2008, 2009; Hafed and Krauzlis, 2008, 2012). Therefore, in my implementation of the model, before microsaccade onset, visual neurons responding to the flashed probe are gain modulated by the oculomotor feedback signal shown in Figure 4C. The strength of this gain modulation at a given eccentricity depends on the strength of the oculomotor feedback signal at the same location.

My neurally inspired implementation of the model can explain mislocalization of foveal stimuli in the direction of an upcoming microsaccade (Figure 2). Consider, for example, the scenario in Figure 4D, corresponding to the experiment of Figure 1. A foveal probe would normally activate visual neurons with foveal RFs (Figure 4D, blue activity profile). The center of mass of this retinotopic population activity may be read out as identifying the probe location. When a microsaccade is about to be generated, foveal and parafoveal SC neurons exhibit increases in firing rate that modulate the tonic activity in this structure during fixation (Hafed, 2011; Hafed et al., 2009; Hafed and Krauzlis, 2012; compare Figures 4A and 4B). This creates a spatially extended

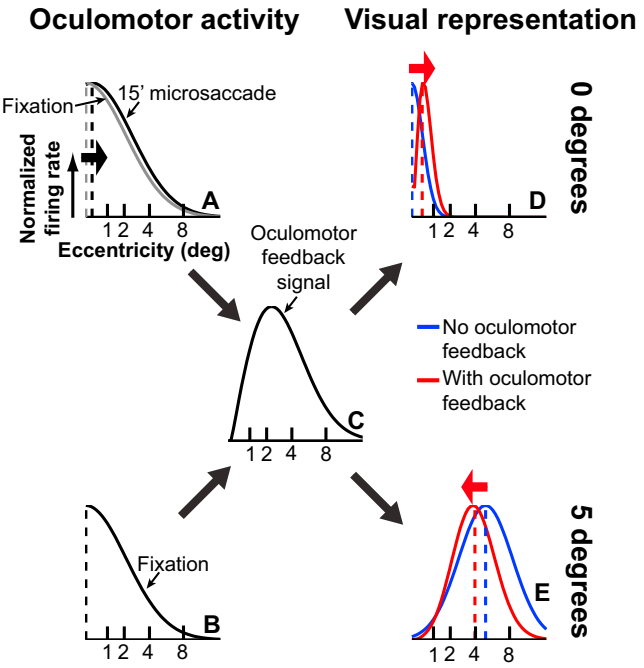

Figure 4. Microsaccadic Compression Predicted by a Model of Saccadic Compression

Implementation of a 1D version of a model of saccadic compression (Hamker et al., 2008), explaining the results of Figures 2 and 3.

(A and B) Microsaccade-related SC activity (A) modifies tonic activity during fixation (B, repeated in gray in A; Hafed et al., 2009).

(C–E) The difference between the two (C) provides oculomotor feedback, implementing a gain modulation on visual neurons representing the probes (D and E; blue, representations without microsaccades; red, after oculomotor feedback). The red arrows above (D) and (E) indicate the resulting shift in percept. The model predicts the opposing directional effects in Figures 2 and 3 (compare red arrows). The x and y axis labels in (A) apply to all panels.

oculomotor feedback signal (Figure 4C) that when applied to the visual representation of the probe deviates it more eccentrically (red activity profile in Figure 4D). This means that individual visual neurons appear to be recruited slightly more eccentrically by the upcoming microsaccade (also see Figure 6). Thus, the center of mass of the resulting gain-modulated probe representation is now shifted (compare the blue and red population profiles).

The model can also explain the opposite, foveally shifted mislocalization of peripheral space before microsaccades (Figure 3). In this case (Figure 4E), the visual neurons responding to the peripheral probe have peripheral RFs (blue activity profile). Before an impending microsaccade, the oculomotor feedback signal is spatially more foveal than the center of mass representing the peripheral probe. Therefore, this feedback signal more effectively modulates the neurons on the foveal end of the population activity, resulting in a more foveal bias of the overall center of mass of the population (Figure 4E, red population activity profile).

Thus, an interaction between a spatially specific visual representation of the probe with a second spatially specific oculomotor feedback signal can account for the patterns of mislocalization I observed in Figures 1, 2, and 3. However, this result raises an important unanswered question: Where does perceptual mislocalization flip from being in the direction of a given microsaccade (Figure 4D) to being opposite it (Figure 4E)? According to my model, this flip can occur at an eccentricity significantly larger than the microsaccade amplitude itself, because of the spatial shape of the oculomotor feedback signal associated with microsaccade generation (Goffart et al., 2012; Hafed, 2011; Hafed et al., 2009; Hafed and Krauzlis, 2012; Figure 4C). For example, consider the scenario in which the same model is run, but now for a probe presented at the intermediate eccentricity of 2.5°. In this case, I found that mislocalization can potentially still be in the direction of a microsaccade, as in the 0° case, and not opposite it, as in the 5° experiment (Figure 5A). This is exactly what I also found experimentally when I repeated the same task of Figure 3A but now at the intermediate eccentricity of 2.5° (see Figures 5B and 5C, which plot the results in a format identical to that used in Figures 3B–3D).

Thus, the results so far show that not only is perception altered before microsaccades, but this alteration is also very specific: foveal locations (0° and 2.5°) are perceived as more eccentric, whereas more peripheral locations (5°) are foveally shifted. The mechanism for this effect is consistent with the previously proposed mechanism for larger saccadic compression (Hamker et al., 2008). Given that microsaccades occur in most, if not all, experiments requiring fixation, the implication of these results is that active perceptual alterations associated with microsaccades likely appear in such experiments even if the experiments themselves are not designed to investigate microsaccades. In the following section, I illustrate this by testing the generality of premicrosaccadic changes in perception to a seemingly unrelated behavioral task.

Microsaccades Alter Performance in Cueing Experiments

The finding that foveal locations are rendered more eccentric before microsaccades and that peripheral locations are rendered more foveal is reminiscent of observations that covert attention alters spatial perception (Carrasco et al., 2002; Yeshurun and Carrasco, 1998). Since cue onsets reflexively trigger microsaccades, I wondered whether premicrosaccadic changes in perception could be part of the mechanism by which visual performance is changed after cueing in classic attention studies, i.e., could microsaccadic compression contribute to cueing effects?

The motivation behind this question is simple. Before large saccades occur, RFs in visual areas such as V4 and FEF are compressed toward the saccade goal (Tolias et al., 2001; Zirnsak et al., 2011), a predicted consequence of the gain modulation in Hamker et al. (2008) (see also Zirnsak et al., 2010; Richard et al., 2009). Besides providing a possible substrate for saccadic compression, such modulation also explains presaccadic attention shifts because more neurons are now effectively dedicated to processing locations near the saccade target (Hamker et al., 2008; Tolias et al., 2001). In the case of microsaccades, my results are consistent with the model of Hamker et al. (2008) (Figure 4) and thus with the idea that visual representations may also be altered before microsaccades occur, as schematized in Figure 6. This figure shows the putative individual

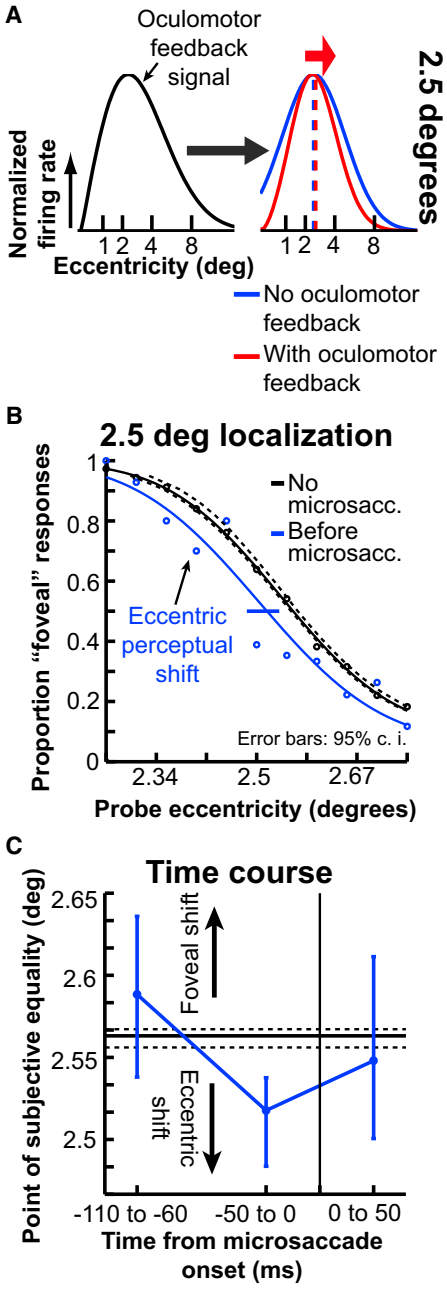

Figure 5. Mislocalization at 2.5°: Model and Experiments

(A) Result of simulating the model of Figure 4 for a 2.5° probe location. For the same microsaccade as in Figure 4, mislocalization in this case is toward a more eccentric shift, similar to probes at 0° and opposite to probes at 5°.

(B and C) The experiment of Figure 3A, but now repeated with the eccentric reference spot placed at 2.5°. Consistent with the model (A), microsaccades are now associated with a more eccentric mislocalization: physically foveal locations are perceived more frequently as less foveal before a microsaccade than with no microsaccades, and the effect is time-locked to movement onset. Error bars indicate 95% CIs (other conventions similar to those in Figures 2C, 2D, and 3B–3D).

See also Figure S5.

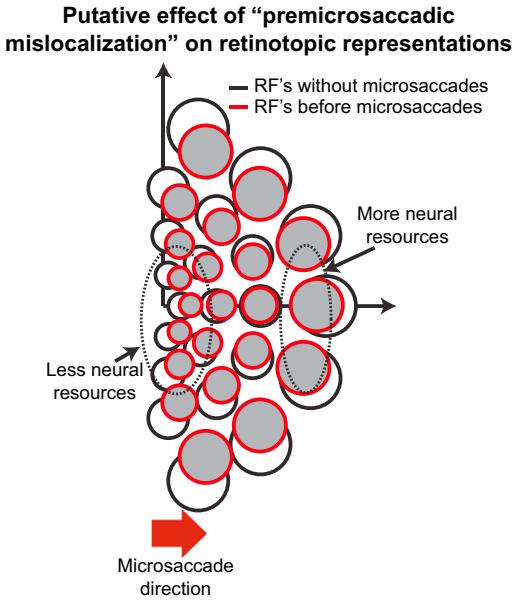

Figure 6. Schematic Demonstrating a Potential Consequence of Microsaccadic Compression

For near eccentricities (dashed ellipse on the left), RFs would shift in the direction of an upcoming microsaccade (compare red and black RFs), leaving ever so slightly less neural resources at these eccentricities than without microsaccades. In the periphery (dashed ellipse on the right), foveal shifts would recruit resources from the many neurons that are normally more peripheral than the target location. Thus, consistent with Hamker et al. (2008) and Figure 4, perceptual mislocalization (Figures 1, 2, 3, 4, and 5) can be correlated with visual performance changes in other tasks beyond just localization because of altered neural recruitment (Figures 7 and 8).

neuron perspective of the population-level changes in Figure 4. As can be seen, at a given peripheral location (right dashed ellipse), a premicrosaccadic foveal shift would recruit the many neurons that would normally be tuned more peripherally to help process the location. This would endow the location with slightly more neuronal resources and alter visual performance. On the other hand, at foveal eccentricities (left dashed ellipse), foveal neurons would be recruited to process ever so slightly more eccentric locations, leaving the original locations with less neuronal resources. Interestingly, small shifts in visual RFs during spatial attention were indeed observed in previous studies (Womelsdorf et al., 2006; Connor et al., 1997), although an explicit link to microsaccades was not explored. If this putative link between premicrosaccadic alteration of perception and attentional performance is true, then one would expect to see premicrosaccadic alteration of performance in classic covert attention tasks, and with specific predictions depending on the cued eccentricity.

To test this, I replicated a classic cueing task used as a workhorse of covert attention (Carrasco et al., 2002). Subjects fixated a central spot and a cue was presented at 5° or 2.5°. After a random time from cue onset, a brief spatial-acuity target (Landolt square) appeared at the previously cued location (Carrasco et al., 2002) and subjects discriminated this stimulus (Figure 7A). In a minority of trials (see Experimental Procedures), the cue

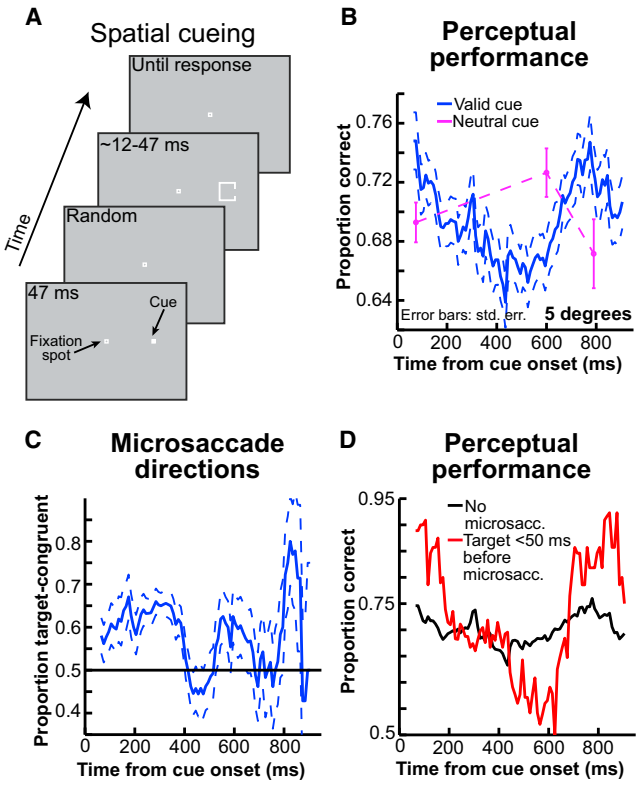

Figure 7. Interaction between Perceptual Performance and Microsaccades in Spatial Cueing

(A) Classic cueing task.

(B) At 5°, subjects had high performance immediately after cue onset and their performance oscillated dynamically, first decreasing and then increasing again. The magenta data points show performance in the minority of neutral cue trials and demonstrate that the main effects (blue) replicated previous reports regarding attention (e.g., Carrasco et al., 2002).

(C) Microsaccade directions, analyzed as in Pastukhov and Braun (2010), also oscillated after valid cue onset. Note how perceptual performance in (B) was high whenever microsaccades were biased toward the target.

(D) Whenever the target appeared immediately before microsaccades, the performance oscillations in (B) were very strongly magnified (red) compared with when it appeared without movements (black). Figure 8 provides a possible explanation. Error bars indicate SE.

See also Figure S2.

consisted of two identical spots, one at the upcoming target location and another at the opposite one. This spread-neutral cue (Carrasco et al., 2002) was noninformative and allowed me to assess whether I could indeed replicate previous modulations in perceptual performance after valid cue onset.

As expected, cueing altered performance. Figure 7B shows the time course of perceptual performance when the cue appeared at 5° (blue curve). The magenta points show performance when the cue was noninformative about the target location. Shortly after cue onset (~75 ms), performance was higher in cued trials than otherwise ($p < 0.05$, χ^2 test; Carrasco et al., 2002; Nakayama and Mackeben, 1989). When the target appeared later (~600 ms), performance declined, and the subjects showed a worse performance than in the neutral cueing condition (middle magenta point; $p < 0.05$, χ^2 test). Both early enhancement (Nakayama and Mackeben, 1989) and late reduction (related to a so-called inhibition of return; Klein, 2000) have previously been observed, confirming that my cueing paradigm was effective in reproducing previous reports. Perhaps most surprisingly, I also observed that performance increased again even longer after cue onset: subjects performed better than they did in the neutral cueing condition (rightmost magenta point; $p < 0.05$, χ^2 test). This oscillation in performance did not happen for neutral cues ($p > 0.05$, χ^2 test comparing the three neutral cue times to each other), and it was repeatable across individual subjects (Figure S2). Thus, my cueing task replicated previous modulations in perceptual performance and also demonstrated an oscillation in performance, with a late rise long after the cue. Similar cue-induced oscillations were recently observed (Koenig-Robert and Vanrullen, 2011; Landau and Fries, 2012), although, to date, no mediating mechanism for them has been revealed.

I hypothesized that this mechanism could be related to premicrosaccadic alteration of visual perception (Figures 1, 2, 3, 4, 5, and 6). I thus analyzed microsaccade directions after cue onset in the same task and found similar oscillations (Figure 7C), such that performance (Figure 7B) was always high whenever the target appeared at a time of more frequent microsaccades toward its location (Figure 7C). Importantly, whenever the acuity target appeared immediately before a microsaccade (Figure 7D), the performance oscillations were hugely magnified (red curve) compared with when the same target appeared without any movements (black curve), suggesting a possible causal interaction with the upcoming movement.

To investigate this interaction further, I again plotted perceptual performance but now as a function of target time relative to microsaccade onset and compared this performance with that observed when the target appeared without any movements. Perceptual performance consistently increased immediately before a microsaccade toward the peripheral target (Figure 8A, blue) compared with the no-microsaccade baseline (Figure 8A, black), as well as with the neutral-cue trials (Figure 8A, magenta). For example, whenever the acuity target appeared within ~34 ± 25 ms before microsaccades, performance was significantly higher than without microsaccades ($p < 0.05$, χ^2 test). The time course of this effect was similar to the time course of foveal perceptual shifts observed earlier (Figure 3D) using a different behavioral task. Thus, impending microsaccades effectively rendered spatial perception momentarily higher in acuity, an effect previously attributed to covert attention (Carrasco et al., 2002). This analysis also explains the oscillation in perceptual performance seen in Figures 7B and 7D, since it shows that during late-target epochs after cue onset, the increase in performance (Figure 7B) resulted from the microsaccades being predominantly directed toward the cued location (Figure 7C; i.e., the acuity target was likely to appear within the critical premicrosaccadic interval that is expected to alter performance). In fact, when I repeated the analysis of Figure 8A but only for trials with target onsets >500 ms after cue onset, I found an identical result (Figure S3B). Thus, oscillations in attentional performance (Landau and Fries, 2012) may simply reflect the temporal dynamics of microsaccades triggered by the cues and their influence on perception.

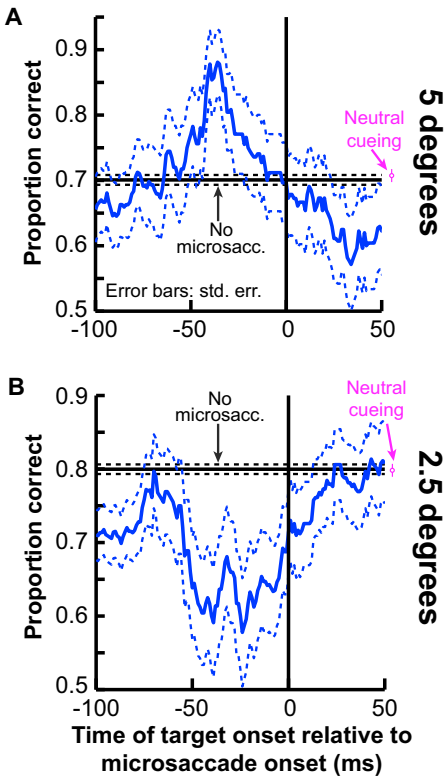

Figure 8. Premicrosaccadic Alteration of Performance during Spatial Cueing

(A) Proportion of correct responses in the cueing task as a function of time of acuity-target onset from microsaccade onset. The black line shows performance when the target appeared without microsaccades within ±150 ms, and the magenta point (± SE) shows performance in the neutral cueing trials (both serve as a comparison baseline). With a similar time course as in Figure 3D, performance increased before a microsaccade toward the target, as if space was rendered higher in acuity (consistent with the foveally directed perceptual shift in Figure 3).

(B) At 2.5°, again with timing similar to that in Figure 5C, performance decreased, as if space was rendered lower in acuity (consistent with the eccentric shift in Figure 5). Thus, the same microsaccade caused higher performance at 5° but lower performance at 2.5°, as predicted by Figures 3 and 5. Error bars indicate SE.

See also Figure S3.

I then analyzed perceptual performance after spatial cueing but at 2.5°. In this case, when the acuity target appeared before a microsaccade toward its location, a very different result emerged: performance decreased (i.e., the subjects performed worse than they did with no microsaccades or during neutral cueing for targets 25 ± 25 ms before the movement, $p < 0.05$, χ^2 test; Figure 8B). Thus, perception was rendered lower in acuity, which again is consistent with the more-eccentric perceptual shifts I observed earlier using a different paradigm (Figures 5B and 5C) and also consistent with Figures 4 and 6. And again, this effect was previously attributed to covert attention (Yeshurun and Carrasco, 1998). Thus, the same microsaccade was associated with either increased (at 5°; Figure 8A) or decreased (at 2.5°; Figure 8B) performance, exactly consistent with the spatial pattern of microsaccadic compression I observed earlier (Figures 1, 2, 3, 4, 5, and 6). Moreover, the similarity between localization performance and cueing performance persisted even when microsaccades opposite the target location were considered (Figures S4 and S5).

Thus, perimicrosaccadic changes in perception (Figures 1, 2, 3, 4, and 5) can be observed in cueing tasks, suggesting that these alterations can account for at least part of the performance changes that classically have been attributed to covert attention (Figures 6, 7, and 8). In fact, I also found that microsaccades alter performance even in monkeys and with other perceptual tasks. To show this, I reanalyzed data from a recent cueing study (Hafed et al., 2011) in which a monkey reported the direction of a brief motion pulse rather than the orientation of a Landolt square (Figure S3C). Upcoming microsaccades toward the peripheral pulse were associated with increased perceptual performance as in my human subjects using very different perceptual stimuli, further demonstrating the generality of premicrosaccadic alteration of visual perception.

To summarize, attentional performance in cueing tasks was altered during the time period preceding microsaccades, and this alteration was manifested in a manner consistent with the premicrosaccadic changes in perception I observed earlier using very different behavioral tasks. The observation of performance changes during attentional cueing paradigms had never before been considered to be related to the microsaccades that occur during these paradigms.

DISCUSSION

In this work, I have shown that microsaccades are associated with an altered spatial percept before movement onset, and demonstrated that this alteration also occurs in different experimental conditions, including ones, such as cueing, that are routinely used to study cognitive and perceptual phenomena. Taken together, my results allow two main conclusions: first, microsaccades are associated with the same active mechanisms for saccadic compression as are much larger saccades (Ross et al., 1997). Second, the existence of these mechanisms nullifies the very same reasons that we traditionally cite when enforcing gaze fixation in experiments. My results thus call for a careful evaluation of any phenomenon in which visual performance is altered, but under the assumption that fixational eye movements are inconsequential (also see Kuang et al., 2012, for a similar sentiment).

Implications for Experiments Requiring Fixation

The observation of altered percepts before microsaccades extends previous investigations of the relationship between saccade amplitude and perceptual mislocalization (Kaiser and Lappe, 2004; Lavergne et al., 2010) down to the smallest possible movements. These results thus support the recently emerging picture of fundamental similarity between microsaccades and saccades (Hafed, 2011): the neural mechanisms for microsaccade generation seem to be the same as those for larger saccades, at least at the level of the brainstem (Hafed et al., 2009; Hafed and Krauzlis, 2012; Van Gisbergen et al., 1981). Moreover, microsaccades are associated with suppressed visual responses in the SC (Hafed and Krauzlis, 2010),

as happens for larger saccades (Goldberg and Wurtz, 1972). Finally, the present results show that microsaccades momentarily alter spatial perception, again just like larger saccades (Ross et al., 1997).

These results are significant particularly given my observation that microsaccade-related changes in perception also appear in cueing experiments and thus have real functional consequences. Previous analyses demonstrated behavioral and neural modulations around microsaccades that were generally consistent with microsaccadic suppression (Bosman et al., 2009; Hafed and Krauzlis, 2010; Hafed et al., 2011; Herrington et al., 2009). However, my present results show a much more fine-grained influence, and also demonstrate that microsaccades in seemingly unrelated tasks (such as spatial cueing) can account for a significant fraction of performance changes. In fact, when J.J. Clark and I first studied the effects of cues on microsaccades 10 years ago (Hafed and Clark, 2002), we found that attentional performance at a cued location or away from it was not different except in (the minority of) trials in which microsaccades occurred (see Figure 9 in Hafed and Clark, 2002). While these early results may have suggested that attention does not shift in most trials after cue onset, the present results clarify a potential reason for why this could be the case: in the trials with microsaccades, the perceptual target likely appeared in the critical premicrosaccadic interval in which visual perception is altered.

The fact that attentional cueing effects may reflect premicrosaccadic changes in perception is significant given the relatively small magnitude of these effects in the first place. For example, in spatial cueing paradigms, the influence of cues on performance is typically small (<5%–10% changes in perceptual performance; Koenig-Robert and Vanrullen, 2011). Thus, even if microsaccades do not occur in every single trial, all it takes for microsaccades to account for the majority of cueing effects is a few movements occurring at the right time. Thus, the component of performance changes attributed to premicrosaccadic changes in perception can be a huge fraction of the overall attentional performance changes seen in cueing tasks.

Implications for Our Understanding of Attention

It may be argued that an alternative interpretation of my results could be that attention influenced both visual performance and microsaccades, and that it did so even in spatial localization. That is, it may be argued that perceptual mislocalizations (Figures 1, 2, 3, 4, and 5) simply reflected the influence of rapid covert attention shifts exogenously driven to the probes. Such probes could attract attention, and this could somehow both alter spatial perception and trigger a microsaccade in <50 ms. However, if this were the case, then it would mean that attention shifts only in a very small minority of trials. For example, only ~10% of the trials shown in Figure 2C contained a microsaccade <50 ms after probe onset, which is the critical period during which the percept was altered. If attention was really the cause of the altered percept in these trials, that would mean that attention did not shift in ~90% of the trials. Moreover, this interpretation would also mean that probe onset can both attract attention and then trigger a microsaccade in <50 ms, which is faster than even the fastest saccadic reaction times (Edelman and Keller, 1996). Finally, I did not see any evidence that microsaccades that occurred within 50 ms after probe onset were biased by probe location or eccentricity. Thus, it is unlikely that my mislocalization results reflected the influence of rapid attention shifts to the probes. Alternatively, a more parsimonious explanation, which is commonly invoked for large saccades (Hamker et al., 2008; Ross et al., 1997), is that spatial perception was altered actively by the oculomotor signals associated with movement generation.

If that is the case, then why does cueing alter microsaccades? That is, why are spatial cues so effective in inducing microsaccades? One likely possibility is that cueing triggers a reflexive, default orienting response that is actively suppressed by the instruction to fixate. Several lines of evidence support this possibility. First, SC activity is highly sensitive to cue onsets (Boehnke and Munoz, 2008), and the close proximity of the SC to the motor output (Gandhi and Katnani, 2011) creates an efficient path for rapid orienting reflexes. Second, neck muscles that are part of the body's "head-turning synergy" are subliminally recruited during covert attentional cues (Corneil et al., 2008), as if the system wants to orient toward these cues by default. Third, reversible inactivation of the SC was recently shown to disrupt the influence of peripheral cues on microsaccades (Hafed et al., 2013). Finally, in my neutral-cue trials with two alternative orienting locations, the microsaccades were equally likely to go to either side, which explains why performance in these trials differed from that in the valid-cue trials. Thus, it appears that microsaccades simply reflect the influence of suppressed orienting reflexes to the presented spatial cues. Such reflexes can also explain the rebound of microsaccade directions toward the cued location long after cue onset (Figure 7C), because subjects could anticipate that the target was about to appear in these long trials.

This raises the potential for an alternative (albeit less neurally inspired) model of the oculomotor feedback signal compared with the one I used in Figure 4C. In particular, it could be argued that the oculomotor feedback signal really at play in my data is nothing more than a pure saccade signal (say to 5°), and that a separate fixation command suppresses the actual saccade without inhibiting its feedback signal from affecting perception. While such a model can be functional, it is not in line with existing literature on the function of the foveal portion of the SC in fixation and microsaccade generation. In particular, at the level of the SC, the neurons that presumably would implement the fixation command are exactly those that are involved in microsaccade generation (Hafed, 2011; Hafed and Krauzlis, 2012). Second, even if there was a separate area implementing the fixation command, this alternative model would not predict the opposite results I observed between the 2.5° and 5° locations. This is so because there is no reason to expect that the pure saccade signal (à la Hamker et al., 2008) would be conceptually different for a 2.5° saccade relative to a 5° one. Additional assumptions and/or parameters would be needed to predict the opposing effects I observed at these two eccentricities, whereas these effects are an emergent property of my implementation of the model.

Finally, the idea that cueing activates a default orienting reflex can reconcile a variety of converging evidence on the almost

identity relationship between saccade generation and visual attention. The classic premotor theory of attention (Berlucchi and Rizzolatti, 1987; Rizzolatti et al., 1994) predicted that attention may be a manifestation of eye movement generation. Later behavioral and neural results demonstrated that visual performance is indeed altered prior to eye movements. For example, in addition to saccadic compression effects (Ross et al., 1997), neural data in visual area V4 show presaccadic enhancement of activity (Moore, 1999; Moore et al., 1998; Tolias et al., 2001), and SC neurons have enhanced contrast sensitivity before saccades (Li and Basso, 2008). According to the models of Hamker and colleagues (2008), these phenomena may all be linked through the influence of corollaries of motor generation on visual representations (Figure 6), and the apparent obligatory link between attention and saccade targets (Deubel and Schneider, 1996) is an emergent property of the gain modulation of visual activity by corollary discharge. My results show that the exact same mechanism remarkably also links attention and microsaccades.

EXPERIMENTAL PROCEDURES

Experiments in this work were approved by the ethics commission of Tuebingen University.

Behavioral Tasks

Subjects sat in a dark room 57 cm in front of a CRT monitor (85 Hz, 41 pixels/degree). For foveal localization (Figure 1A), a white central fixation spot appeared for 0.25–5 s over a uniform gray background. The spot was ~7.3′ × 7.3′ and its luminance was 97.3 cd/m^2. Background luminance was 20.5 cd/m^2. When the spot disappeared, a brief white probe appeared simultaneously and lasted for 11.8 ms. The probe consisted of a vertical line (15′ × 1.5′) centered 37′ above/below the fixation spot; its horizontal position varied across trials. Subjects fixated and reported whether the probe was displaced to the right or left of the central spot. The eccentric localization tasks (at 5° or 2.5°; Figures 3A, 5B, and 5C) were similar except that the probe appeared at a location centered on 5° or 2.5° horizontally (right or left of fixation). The fixation spot always remained on, and there was a second reference spot at 5° or 2.5° that disappeared with probe onset. Subjects reported whether the probe was displaced to the right or left of this spot.

The cueing task (Figure 7A) involved a cue spot at either 5° or 2.5° (right or left of fixation) for 47 ms. A Landolt square (Carrasco et al., 2002) appeared 58–1,000 ms after cue onset, and the subjects reported the direction of the square's opening. The Landolt square was 31′ × 31′, with a gap size of 2.9′ at 2.5° and 4.4′ at 5°. In one-fourth to one-third of the trials, I used a spread-neutral cue (Carrasco et al., 2002), i.e., both the target location and an identical location on the opposite side were cued simultaneously. In these trials, the square appeared 71 ms, 600 ms, or 788 ms after spread-neutral cue onset. Subjects initially did a practice session in which the Landolt square appeared 400 ms after cue onset and lasted for a variable duration. I then picked for each subject the duration of the Landolt square that resulted in ~70%–75% correct identification of the gap location at 5°. I then ran the main experiment shown in Figures 7 and 8. Similar to previous studies, the target duration was ~12–47 ms, being ~12, 24, or 35 ms for two-thirds of the subjects (~12 ms for two of them). Thus, the duration of the Landolt square was one in which perisaccadic changes in perception were still expected to occur (Van Wetter and Van Opstal, 2008).

I collected thousands of trials in each condition (8,116 in the foveal localization experiment, 14,524 at 2.5°, 11,212 at 5°, and 10,154 in the cueing task). Such large numbers of trials were necessary to compile full psychometric curves and time courses of perimicrosaccadic effects. Eight subjects participated in the foveal localization experiment, 16 at 2.5°, 18 at 5°, and 21 for spatial cueing. Each subject participated in one to three sessions, with some subjects completing more than one experiment. In my analyses, I pooled data across subjects to increase statistical confidence, after first confirming the robustness of the effects for individual subjects (see Figures S1A, S2, and S3C).

I tracked eye movements using a high-speed camera (EyeLink 1000, 1 KHz sampling). In order to stabilize the head and maximize eye-tracking performance, I fixed the subjects' heads at five different points using a custom-made device.

Behavioral Analysis

For the behavioral analysis, I fitted psychometric data with sigmoids. To obtain psychometric curve confidence intervals (CIs; e.g., Figure 2C), I used bootstrapping (1000 iterations; Wichmann and Hill, 2001a, 2001b). I assessed shifts in psychometric curves by assigning significance to those cases in which the 95% CIs for the no-microsaccade PSE did not overlap with the 95% CIs with microsaccades. I remapped the right/left responses to label them as either "in the direction of a microsaccade" (Figure 2C) or "more foveal" (e.g., Figure 3B) to clarify the influence of the microsaccades on spatial perception. For example, for the analysis shown in Figure 2C, if the microsaccade was rightward and the subject indicated "right," then the percept was that of a probe appearing displaced in the direction of the microsaccade. For Figures 2C and 2D, I also remapped the physical right/left locations of the probe according to microsaccade direction, such that positive locations on the x axis in Figure 2C are locations displaced in the direction of the movement. This allowed me to combine rightward and leftward microsaccades to simplify the data presentation. In this case, the no-microsaccade curve was plotted as the fraction of "right" responses (y axis) as a function of physical probe location.

In the cueing task, I obtained the time course of perceptual performance after valid cue onset (Figure 7) by binning trials according to target onset time. I slid the bin (130 ms width) by 10-ms steps. For Figure 8, I used a finer resolution (50-ms bins stepped by 1 ms). I also performed the analysis of Figure 8 for the minority neutral cue trials and still found premicrosaccadic changes, consistent with the idea that microsaccades alter visual performance in general. I used the χ^2 test to compare binomial proportions (Figures 7 and 8).

Microsaccade Analysis

I detected microsaccades using velocity/acceleration criteria (Hafed et al., 2009) and manually inspected all trials to correct for misdetections. I checked the characteristics of the detected movements by plotting movement peak velocity against amplitude (Zuber et al., 1965), as well as by plotting amplitude distributions (Hafed et al., 2009; Figure S6). I detected ~37,000–47,000 microsaccades per experiment.

I only included predominantly horizontal movements (directions within 45° from horizontal) in the analyses because of the spatial arrangement of the stimuli in the tasks. Preliminary analyses revealed that, as in larger saccades, the perimovement changes I observed depended on the movement direction.

Model of Spatial Mislocalization

I sought to investigate whether premicrosaccadic changes in perception are mediated by a mechanism similar to that hypothesized for presaccadic changes. Thus, I implemented a simplified model (shown in Figure 4) based on one previously described for larger movements (Hamker et al., 2008) but maintaining the same conceptual components, and staying in line with published results regarding microsaccade generation in the SC.

My model contains a 1D map of visual space, including foveal magnification (Hamker et al., 2008). I used the following equation to represent visual space:

$$X = 1.4 * \ln\left(\mathrm{sqrt}\frac{(r^2 + 2 * 3 * r + 3^2)}{3}\right) \qquad \text{(Equation 1)}$$

where X is the transformed space (millimeters of tissue) and r is the original visual eccentricity. This is a model of visual mapping in the SC (Ottes et al., 1986), and I only used it to simplify the model. Visual space in cortical areas has a similar foveal magnification (Hamker et al., 2008). In this visual space, brief luminance probes induce a population response centered at the probe location (see Figure 4, rightmost column, blue curves). Localization is assumed to depend on readout of this population (Hamker et al., 2008), and I

implemented readout in my model by a center of mass computation. For 0°, 2.5°, and 5°, I assumed that the probe activates populations in visual space based on RF sizes in different areas, including the SC (Hamker et al., 2008; Krauzlis, 2004). I thus modeled the visual responses to 0°, 2.5°, and 5° probes as gaussians in model space with SDs equivalent to ~1°, 3°, and 3° (Krauzlis, 2004). The exact numerical choices do not necessarily alter the conceptual results of the model.

To investigate the role of microsaccades on visual representation, I implemented a snapshot of the model when an oculomotor signal is present to modify this representation (Hamker et al., 2008). I assumed that oculomotor feedback acts as a gain on visual representation such that the output of a visual neuron, i, is modified as follows:

$$Y_i = (1 + \text{gain}) * y_i \quad \text{(Equation 2)}$$

where gain is the oculomotor feedback at a given time relative to movement onset. Because the neurons involved in microsaccade generation in the SC are tonically active without microsaccades, the oculomotor feedback signal that is specific to microsaccades is the difference between the activity for a microsaccade and the tonic activity during fixation (Figure 4, panel C = panel A minus panel B). I modeled the latter activity as a Gaussian in SC space, with the SD based on previously collected neural recordings (0.75 mm) and centered on the 0° retinotopic position (Hafed and Krauzlis, 2008). Activity before a microsaccade would involve increases in neurons preferring the microsaccade endpoint, thus causing a shift in the whole population activity profile in the direction of the microsaccade (Figure 4A). Thus, the center of mass of premicrosaccadic activity reflects the endpoint of the upcoming movement, consistent with previous results from SC experiments (Goffart et al., 2012; Hafed et al., 2008; Hafed and Krauzlis, 2008; Lee et al., 1988) and explaining Figure 4A (also see Hafed, 2011). All model outputs were normalized to one.

SUPPLEMENTAL INFORMATION

Supplemental Information includes six figures and can be found with this article online at http://dx.doi.org/10.1016/j.neuron.2012.12.014.

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