Electrophysiological and Psychophysical Studies on Microsaccades

by

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ABSTRACT

Our eyes never stop moving, even during attempted gaze fixation. Fixational eye movements, which include tremor, drift, and microsaccades, are necessary to prevent retinal image adaptation, but may also result in unstable vision. Fortunately, the nervous system can suppress the retinal displacements induced by fixational eye movements and consequently keep our vision stable. The neural correlates of perceptual suppression during fixational eye movements are controversial. Also, the contribution of retinal versus extraretinal inputs to microsaccade-induced neuronal responses in the primary visual cortex (i.e. area V1) remain unclear. Here I show that V1 neuronal responses to microsaccades are different from those to stimulus motions simulating microsaccades. Responses to microsaccades consist of an initial excitatory component followed by an inhibitory component, which may be attributed to retinal and extraretinal signals, respectively. I also discuss the effects of the fixation target's size and luminance on microsaccade properties. Fixation targets are frequently used in psychophysical and electrophysiological research, and may have uncontrolled influences on experimental results. I found that microsaccade rates and magnitudes change linearly with fixation target size, but not with fixation target luminance. Finally, I present ion a novel variation of the Ouchi-Spillmann illusion, in which fixational eye movements may play a role.

ACKNOWLEDGMENTS

I would like to express my deepest appreciation to my advisor, Dr. Martinez-Conde, who welcomed me to her lab. Dr. Martinez-Conde's lab provided with me great opportunities to learn about neuroscience and specifically eye movements. With the help of Dr. Martinez-Conde and her team at Barrow Neurological Institute, I was able to extend my knowledge in the field of neuroscience and my research skills in electrophysiology and psychophysics. Her lab is world leading in eye movement research and it has been my pleasure to contribute to her research projects. I should mention that she spent a lot of her time teaching me to set up primates for neuronal recordings.

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Chapter 1

INTRODUCTION TO FIXATIONAL EYE MOVEMENTS

It might be surprising to know that our eyes never stop moving. Even when we try to keep our eyes still, they are making involuntary small movements called fixational eye movements. Fixational eye movements are necessary to overcome neuronal adaptation, which can occur in retinal photoreceptors due to unchanging visual stimuli. In other words, if our eyes stopped moving, stationary or unchanging objects would disappear from our vision due to the adaptation of retinal photoreceptors. Retinal stabilization under experimental conditions, or even due to intense careful fixation, causes the fading of visual stimuli from awareness (Martinez-Conde, Macknik, Troconso, & Dyar, 2006 and Simons, Lleras, Martinez-Conde, Slichter, Caddigan, & Nevarez, 2006). This phenomenon, called visual or perceptual fading, was reported by Troxler more than 200 years ago (Troxler, 1804). We fixate our gaze about 80% of the time, so learning the mechanisms underlying the generation of fixational eye movements, and their effects on vision and oculomotor control, is crucial to our understanding of visual neuroscience. There are three different classes of fixational eye movements: microsaccades, tremor, and drift.

Tremor is a aperiodic, wave-like motion of the eyes (Riggs, Cornsweet, & Cornsweet, 1953). It has an amplitude equivalent to the diameter of a cone in the fovea, which makes its accurate recording challenging (Yarbus, 1967; Ratliff & Riggs, 1950 and Carpenter, 1988). The contribution of tremor to the maintenance of vision is unclear (Martinez-Conde, 2006).

Drift is a slow motion of the eye that takes place between microsaccades and occurs simultaneously with tremor. Drift amplitudes are equivalent to approximately a dozen photoreceptors (Ratliff & Riggs, 1950).

Microsaccades –i.e. small-magnitude saccades that occur while attempting to fixate– are the largest and fastest fixational eye movements. They and occur with a frequency of 1-2 Hz (Martinez-Conde et al, 2013) and have durations around 25 ms (Ditchburn, 1980) Retinal image displacement due to microsaccades range from several dozen (Ratliff & Riggs, 1950) to several hundred photoreceptor widths (Martinez-Conde, Macknik, & Hubel, 2000; Martinez-Conde, Macknik, & Hubel, 2002; Martinez-Conde, Macknik, & Hubel, 2004; Hafed & Clark, 2002; Moller, Laursen, Tygesen, & Sjolie, 2002; Engbert & Kliegl, 2003; Engbert & Kliegl, 2003a and Engbert & Kliegl, 2004). .

Here I will discuss part of the studies on microsaccadic eye movements in which I was involved during my graduate studies in Dr Martinez-Conde's lab, one of the world leading labs in eye movements research, at the Barrow Neurological Institute. In the first section, I will discuss electrophysiological experiments on rhesus monkeys that address the neural correlates of microsaccadic suppression and the importance of retinal versus extraretinal signals in V1neuronal responses. In the second section, I will present psychophysical experiments conducted to determine the effect of a fixation target's physical features on the characteristics of human microsaccades. In the third section, I will introduce a novel version of a visual illusion in which fixational eye movements might play a role.

Chapter 2

NEURONAL RESPONSE IN AREA V1 TO MICROSACCADES

INTRODUCTION

Fixational eye movements, in particular microsaccades, have been shown to counteract visual fading and to increase visibility (Martinez-Conde, Macknik, Troconso, & Dyar, 2006; Troncoso, Macknik, & Martinez-Conde, 2008 and McCamy M. B., et al., 2012). Even though microsaccades result in displacements of retinal images that are large enough to be perceived, we are not aware of them. The visual system suppresses such displacements from our awareness through a mechanism called "microsaccadic suppression", whose neural correlates are largely unknown (Martinez-Conde , Otero-Millan, & Macknik, 2013). To date, it remains controversial if the visual system suppresses the retinal motion induced by microsaccades using either retinal or extraretinal signals (inputs from sources other than the retina). Even more surprisingly, it is not known whether the neural correlates of microsaccadic suppression are located at high or low levels of visual processing.

I explored the role of neurons in the area V1 in microsaccadic suppression by comparing their responses to microsaccades in the presence of a stationary stimulus to their responses to a moving stimulus mimicking the retinal displacements induced by microsaccades. The interplay between receptive field and visual stimulus could explain thoroughly the responses recorded from neurons in area V1 in response to microsaccades. If that is the case, real microsaccades should elicit the same responses as stimuli motions that mimic microsaccades. Alternatively, there might be other sources contributing to V1 neurons' responses. Corollary discharges from the oculomotor system in association with

microsaccades, proprioceptive signals from the eye muscles, and/or global motion integration are possible candidates. In that event, the responses to microsaccade-induced retinal displacements should be different from the responses to equivalent stimulus displacements.

I found that V1 neurons' responses to real microsaccades differed from their responses to equivalent retinal motion in the world (i.e. induced by simulated microsaccades). The responses to real microsaccades were biphasic with an excitatory component appearing almost immediately after microsaccade onset, followed by an inhibitory trough. In contrast, the responses to simulated microsaccade did not show the trough component. The difference in responses to real and simulated microsaccades suggests that V1 can distinguish between retinal motion induced by eye movements and motion in the world. This finding may help to clarify the role of V1 in stimuli visibility and microsaccadic suppression.

METHODS

Surgical and recording procedures. I recorded neuronal responses from area V1 of two adult male rhesus monkeys (*Macaca Mulatta*), at the same time as the monkey's eye positions. The neuronal population (N = 184 neurons) included 76 neurons from monkey H and 108 neurons from monkey Y, with receptive field (RF) eccentricities ranging from 0.2 degrees to 35 degrees. I analyzed 70 neurons from monkey H: 69 neurons had data in all experimental conditions, and 6 neurons were discarded due to technical problems; see **Data Analysis** section for details. I analyzed 76 neurons from monkey Y: 52 neurons had data in all conditions, and 32 neurons were discarded due to technical problems.

Before the experiments began, each monkey was implanted with a head post for head stabilization, a scleral eye coil to monitor eye movements and a recording chamber mounted over the occipital operculum to gain access to area V1. All the animal procedures were approved by the Institutional Animal Care and Use Committee (IACUC) at the Barrow Neurological Institute and followed the recommendations of the NIH Guide for the Care and Use of Laboratory Animals and the Animal Welfare Act of 1986 and its revisions.

Single units were recorded extracellularly with lacquer-coated electropolished tungsten electrodes (FHC Inc). A small portion of the dura mater was removed to facilitate the penetration of electrode to the brain. After isolating each individual neuron, I mapped its receptive field (RF) and determined the optimal orientation and width of a bar stimulus based on seeing/hearing each neuron's activity while manually changing the stimulus' dimensions, contrast, and/or orientation. Eye movements were sampled at 1Khz with a Riverbend system.

Experimental design. Monkeys were trained to fixate their gaze on a small red cross (with a diameter of 0.5°) on a video monitor (BarcoReference Calibrator V, 100Hz refresh rate) placed at a distance of 57 cm from the monkeys' eyes. Fruit juice rewards were provided for every ~1.5-2 seconds of fixation. Eye movements exceeding a 2x2° fixation window were recorded but not rewarded. The experiment had three different experimental conditions:

Stationary stimulus condition. In this condition, a stationary bar of optimal characteristics (including dimensions, contrast, and orientation) was positioned over the neuron's RF.

Moving stimulus condition. A bar with the same physical characteristics as above was positioned over the RF of the same neuron. Here the bar was not stationary, however, but it moved to simulate the fixational eye movements recorded during the Stationary stimulus condition. To do this, the x and y eye-position records were taken from the Stationary stimulus condition and fed back into the system to produce the motion of the bar. This condition allowed us to compare neural responses to "real" microsaccades, generated by monkey while the moving bar was over the neuron's RF (**Figures 1-2**, blue) with neural responses triggered by bar's "simulated" microsaccades that occurred simultaneously (**Figures 1-2**, red).

No stimulus condition. For a subset of the neurons (n=116) tested under both the Stationary stimulus condition and the Moving stimulus condition, I run a third condition

in which no stimulus was placed on the RF. Area V1 firing rates can be very low in the absence of visual stimulation, making it difficult to monitor the shape of the spike waveform during this condition; thus, to ensure that neurons were not lost during the recordings, I re-ran the Stationary stimulus condition at the end of the No stimulus condition, and compared the neuronal responses to those obtained during the first run of the Stationary stimulus condition. For a No stimulus condition to be valid, the baseline and the peak in the PSTH of the final Stationary stimulus condition were required to be within 30% of the baseline and the peak recorded during the initial Stationary stimulus condition. Also, because only neurons with a minimum ongoing activity can show a potential response decrement, I only considered neurons tested in the No stimulus condition eligible for this analysis if their responses met a minimum "quorum" of data in at least 20 out of the 1,000 1-ms-bins around the microsaccade time. 62 neurons (35 in monkey H and 27 in monkey Y) met these requirements.

I recorded eye positions and neural activity for 5-10 minutes in each condition, which resulted in ~700 microsaccades per condition.

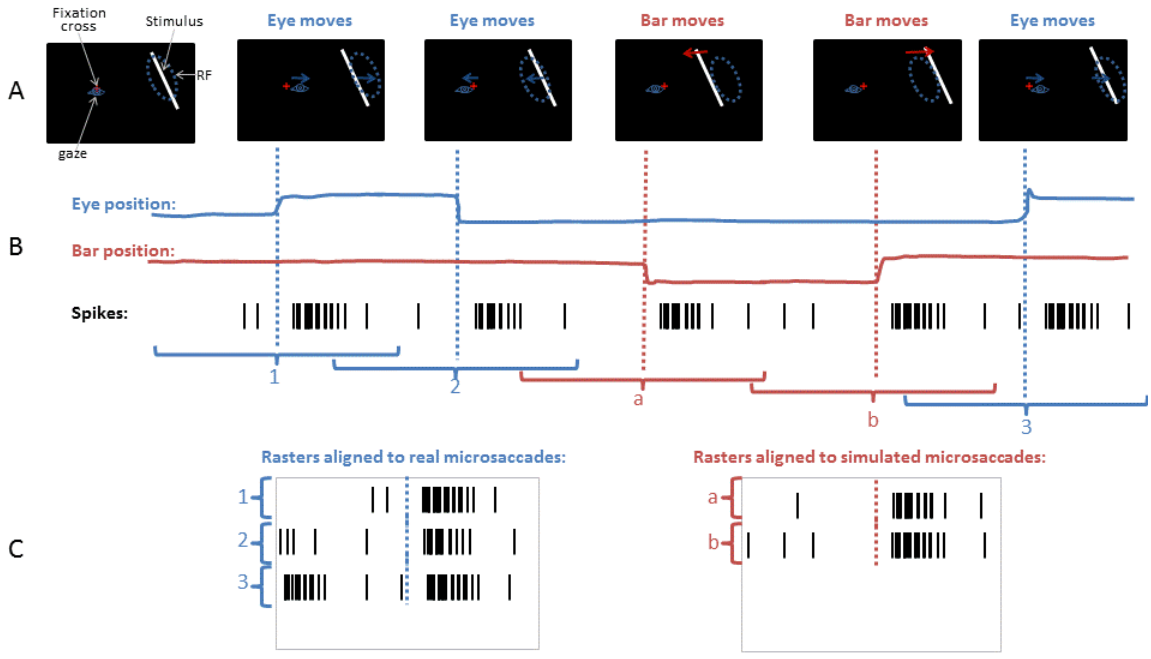


Figure 1. ***Experimental design and data analysis.*** (**A**) Schematic of the stimulus monitor (not to scale) showing the fixation target (red cross), center of gaze (eye), stimulus (white bar) and receptive field position (dashed ellipse). Blue arrows indicate gaze displacement ('real' microsaccades) and red arrows indicate stimulus displacement ('simulated' microsaccades). (**B**) Schematic of data collected during a few seconds of the experiment: eye position trace (blue), bar position trace (red) and spikes from the neuron being recorded (short black lines). Blue dotted lines indicate the places were a 'real' microsaccade was detected in the eye position trace. Red dotted lines indicate the places were a 'simulated' microsaccade was detected in the bar position trace. Brackets at the bottom indicate the amount of time around each event ('real 'or 'simulated 'microsaccades) used to calculate the PSTHs. (**C**) Rasters of spikes (from (**B**)) aligned at the different events: 'real' microsaccades (left) and 'simulated' microsaccades (right).

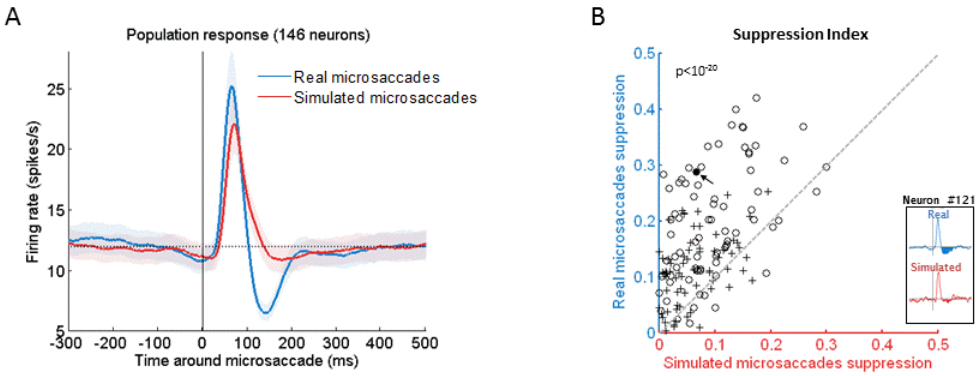


Figure 2. ***Spike rate modulation after 'real' and 'simulated' microsaccade.*** **(A)** Population data showing the peri-microsaccade modulation of V1 responses for 146 neurons, both for 'real' microsaccades (blue) and for simulated microsaccades (red). Both show a large increase in firing rate immediately after the microsaccade, but only in the case of 'real' microsaccades this enhancement is followed by a period of suppression (firing rate below baseline). The dotted horizontal line represents baseline firing rate and the shaded areas are the SEM across the 146 neurons. **(B)** Comparison of the suppression index between 'real' and 'simulated' microsaccades. Each point represents data from a single neuron: N=76 for monkey Y (o) and N=70 for monkey H (+). The inset illustrates the time course of microsaccade-related modulation for real and simulated microsaccades (as in panel A) for single neuron #121 (in black and pointed by the arrow in the scatter plot). The suppression index is the normalized sum of the negative values in these curves (shaded areas), and yields the ordinate and abscissa of each data point in the scatter plot. The line of unit slope (dashed gray line) indicates balanced real versus simulated microsaccade suppression. Most data points (83%) fall above the line, indicating a predominance of suppression after 'real' microsaccades. The p-value ($p<10^{-20}$) represents the statistical significance of differential suppression index after 'real' and 'simulated' microsaccades for the entire population measured by a two-tailed Wilcoxon signed rank test.

Microsaccade detection.

Eye movements were recorded with a scleral eye coil and sampled at 1Khz with a Riverbend system. Saccades were identified with a modified version of the algorithm developed by Engbert and Kliegl (Engbert & Kliegl, 2003a; Engbert, 2006; Engbert & Mergenthaler, 2006; Laubrock, R, & Kliegl, 2005; and Rolfs, Laubrock, & Kliegl, 2006) with l = 8 (used for the velocity threshold detection) and a minimum saccadic duration of 6 ms. Microsaccades were defined as saccades with magnitude < 2 degrees in each eye (Beer, Heckel, & Greenlee, 2008; Betta & Turatto, 2006; Hafed, Goffart, & Krauzlis, 2009; Martinez-Conde, Macknik, Troconso, & Dyar, 2006; Martinez-Conde, Macknik, Troncoso, & Hubel, 2009; and Troncoso X. , Macknik, Otero-Millan, & Martinez-Conde, 2008).

Data analysis.To examine the peri-microsaccade modulation of neural responses, I extracted the spike times that occurred from 500 ms before until 500 ms after the onset of microsaccade for each neuron in all conditions, and calculated the perisaccade time histogram (PSTH) for both 'real' and 'simulated' microsaccades. It is important to note that I calculated both PSTHs from the same exact train of spikes, only changing the trigger event to 'real' or 'simulated' microsaccade onset (**Figure 1**). I calculated the PSTHs for individual neurons (**Figure 3B**) using 1ms (1 sample) bins and smoothed them with a Savitzky-Golay filter of 35 ms. The population responses in **Figure 2A** and **3A** are the averages of the PSTHs of the individual neurons (unfiltered), smoothed with a Savitzky-Golay filter of 35 ms.

I considered that neuronal responses showed modulation to microsaccades if the PSTH had a peak (or a trough) above (or below) 3 standard deviations of the baseline (defined as the mean activity in the range 500 - 100ms before microsaccades).

Peri-microsaccade modulation of the spike rate was summarized with two indices: the normalized enhancement index and the normalized suppression index (Reppas, Usrey, & Reid, 202). For each individual neuron, normalized enhancement was calculated from the PSTH by integrating the area that fell above baseline. Suppression was similarly calculated from all values that fell below baseline (shaded in **Figure 2B** inset). These areas were normalized by the integral of the baseline in the interval [30-350] ms; so that a value of 1 would mean that during the entire interval the response doubled (for the enhancement index) or fell to 0 (for the suppression index).

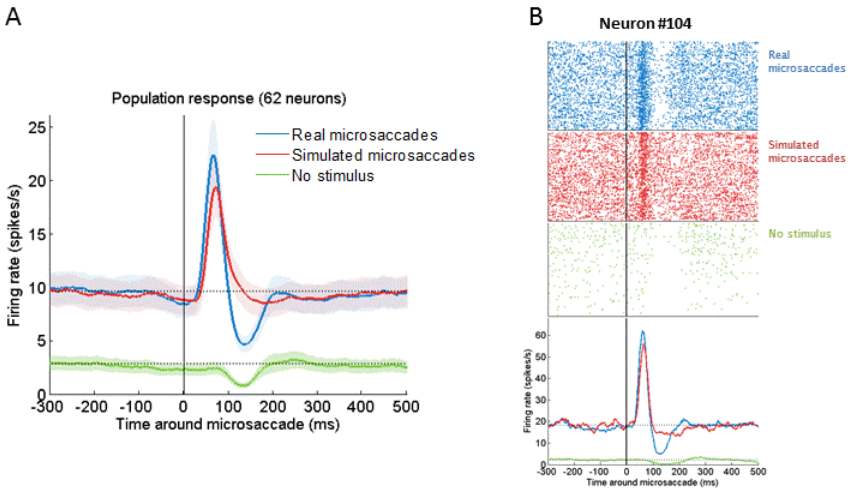


Figure 3. ***Neuronal response in "no stimulus condition".*** **(A)** Population data showing the peri-microsaccade modulation of V1 responses for the 62 neurons where I also tested the 'no-stimulus' condition (green). The dotted horizontal lines represent baseline firing rate and the shaded areas are the SEM across the 62neurons. **(B)** Example for an individual neuron: PSTH (bottom) and spike rasters for 'real' (blue) and 'simulated' (red) microsaccades during the 'moving bar' condition and during the 'no stimulus' condition (green). Each dot in the rasters represents a spike and there is one line per microsaccade.

RESULTS

In order to understand whether V1 neurons can distinguish between internal and external motion, I compared V1 neuronal responses to real microsaccades with those to simulated microsaccades. In addition, I set out to determine whether the interplay between receptive field and visual stimulus can explain in full the neural responses to microsaccades in area V1.

Different neural responses to real versus simulated microsaccades. I compared the responses of each V1 neuron to microsaccades with those to equivalent stimulus motion. To produce realistic microsaccade-like motion, monkeys first fixated a small cross in the presence of a stationary bar over the neuron's RF, while their eye movements were being recorded (Stationary stimulus condition; see Methods for details). Then, the bar was moved according to the eye position data previously collected, while the same neuron was being recorded (Moving stimulus condition). That is, the formerly sampled eye-position data was sign-reversed and used to specify the x- and y-position of the bar in each frame of the Moving stimulus condition. The sign reversal of the eye position yielded a retinal image motion equivalent to that in the Stationary stimulus condition (see Methods, **Figure 1** for details). Thus, it was possible to compare the neural responses to the "simulated" microsaccades produced by the motion of the bar (**Figure 1**, red) with the neural responses to the "real" microsaccades that occurred concurrently during the Moving stimulus condition (**Figure 1**, blue).

Real microsaccades included retinal motion signals (receptive field displacement over the stimulus), in addition to other potential signals, such as a corollary discharge

produced by the oculomotor system in association with microsaccades, proprioceptive signals from the eye muscles, and/or global motion integration computations (I refer to all these possible sources generically as "non-retinal" signals). In the simulated microsaccades, only the retinal signals produced by the motion of bar stimulus over the visual field were present.

Responses to real microsaccades (**Figure 2A**, blue) were generally biphasic: a quick and dramatic increase over baseline (peaking ~70ms after microsaccade onset) was followed by a smaller and slower trough below baseline (minimum below baseline at ~145ms after microsaccade onset). The Stationary stimulus condition (when the bar was not moving) produced an equivalent response profile.

Responses to simulated microsaccades (**Figure 2A**, red) (i.e. responses to stimulus motion simulating microsaccade-induced displacement) differed from the responses to real microsaccades in that they lacked the inhibitory (trough) component of response. That is, both real and simulated microsaccades produced large increases in firing rate shortly after the microsaccade onset, but this enhancement was followed by suppression (firing rate below baseline) only in the case of 'real' microsaccades. The neuronal population results (**Figure 2A**) were consistent with those of individual neurons, in that the majority (83%) of neurons lacked a trough (or the trough was greatly reduced) for simulated versus real microsaccades (**Figure 2B**; see Methods for details on the calculation of the suppression index).

In a subset of the recorded neurons, there was no visible excitatory component in response to real microsaccades, but the inhibitory component was present and had a

similar latency after microsaccade onset as previously observed (with a minimum below baseline at ~145ms).

I ran a subsequent control experiment to assess whether the lack of excitatory responses found in this subset of neurons might have been an artifact due to suboptimal positioning of visual target over RF. In this experiment, I changed the properties of stimulus (orientation, width, contrast and position) in a gradual fashion. As expected, neurons decreased their responses as the visual target became less optimal (and ultimately stopped responding when it was outside of the RF). The shape of the neuronal response never switched from excitatory to inhibitory, however, indicating that target positioning over RF was appropriate and validating the results described above (data not shown).

The combined results indicate that V1 neuronal responses to real microsaccades are not purely because of eye motion sweeping the neuron's RF over stimulus, but are influenced by non-retinal signals as well. Potential sources could be a corollary discharge produced by the oculomotor system in association with microsaccades, proprioceptive signals from eye muscles, and/or global motion integration computations (motion relative to the static fixation cross or to the edges of the monitor). Future research should investigate the origin of these signals.

Inhibitory responses to microsaccades also occur in the absence of visual stimulation. In a subset of neurons, I recorded neural responses in the No stimulus condition. Here I could investigate the neuronal responses to real microsaccades in the absence of any visual stimulation in the RF. Any response modulation seen in this condition would be

due to extraretinal signals, because there was no visual stimulus to cause retinal displacements in conjunction with microsaccades.

In the absence of visual stimulation, I found diminished neuronal activity in response to (real) microsaccades with the same timing as in the presence of visual stimulation (**Figure 3**). This finding supports the hypothesis that extraretinal signals contribute to microsaccade-induced responses in area V1.

DISCUSSION

We can distinguish between object movements and self-movement (including eye, head, or body movement) despite of retinal image displacement in both. The fact is that the image of external world is never stable on our retina even when we fixate our eyes. During fixation, microsaccades displace the retinal images of visual stimuli continuously. Even if these retinal displacements are very small they are still in the range of our perceptual abilities however. But our visual perception is quite stable despite these jittery movements on the retina. Therefore, there might be a mechanism of "microsaccadic suppression" to block the microsaccade-induced retinal motion from our awareness. Psychophysical experiments have shown elevation of perceptual thresholds during microsaccades (Zuber & Stark, 1966 and Beeler, 1967), but the underlying neural correlates are not well understood. Here I studied the responses of area V1 to microsaccades, as well as the contribution of retinal versus extra-retinal signals to such responses.

The experimental paradigm offered the advantage of dissociating the retinal motion induced by microsaccades from that induced by equivalentstimulus motion, without stabilizing the eye. Stabilization methods such as extraocular muscle paralysis may not eliminate eye movement perfectly, they may interfere with perception, and they cause fading of retinal image due to adaptation (Stevens, et al., 1976). Further, the motor command to generate a microsaccade can potentially contribute to neuronal responses in visual neurons, even if no actual microsaccade can be made due to eye paralysis.

In the current study, I compared the responses of area V1 neurons to retinal motion due to real and simulated microsaccade and I investigated the contribution of

retinal versus extraretinal signals to microsaccade-induced responses. The results indicate that neuronal responses in area V1 to real microsaccades have an excitatory peak followed by an inhibitory trough (**Figure 2A**). Responses to simulated microsaccades also included the excitatory peak (reflecting comparable retinal signals to those in real microsaccades), but lacked the inhibitory trough.

Thus, the trough component (also seen in the "no stimulus" condition) may be attributed to extra-retinal signals. These combined findings are consistent with the hypothesis that both retinal and extraretinal signals contribute to microsaccade-triggered neural activity in area V1. Further studies are needed to find the source and importance of the inhibitory response. Potential candidates are corollary discharges (directly coming from oculomotor system or from higher areas in the visual system), proprioceptive signals (from extra ocular muscles), or global motion integration.

One hypothesis is that the inhibitory component contributes to microsaccadic suppression, a process in which the nervous system prevents vision from becoming unstable due to microsaccades. Future investigation is required to explore this topic.

Chapter 3

THE EFFECTS OF FIXATION TARGET SIZE AND LUMINANCE ON MICROSACCADES

INTRODUCTION

In visual neuroscience psychophysical and electrophysiological experiments, human subjects or monkeys frequently need to keep their eyes focused on a fixation target. For example, single cell recording experiments in awake monkeys usually require a fixation target to stabilize the monkey's gaze and thus keep the neuron's RF in the position within the visual field during the recordings. The use of a fixation target also provides a comparable experimental condition among different psychophysical and physiological studies. The physical properties of fixation targets such as size, luminance, color, and shape usually vary across studies, however. Presently, there is no such thiug as a standard fixation target. Presently, fixation targets are either chromatic or achromatic, their sizes range typically from 0.05 to 2 degrees of visual angle (°), and their shapes are as diverse as circles, concentric rings, squares, and crosses (Bonneh, et al., 2010; Hsieh & Tse, 2009; Kanai & Kamitani, 2011; Laubrock, Kliegl, Rolfs, & Engbert, 2010; McCamy M. , et al., 2012; Mergenthaler & Engbert, 2010; Otero-Millan, Macknik, & Martinez-Conde, 2012; Pastukhov & Braun, 2010; Rolfs, Jonikaitis, Deubel, & Cavanagh, 2011 and Thaler, Schütz, Goodale, & Gegenfurtner, 2012). Because fixational eye movements affect the visibility of visual stimuli, they may influence experimental results in an uncontrolled way. The effect of fixation target physical properties on fixational eye movements, especially microsaccades, is not well studied, however. There are some

conflicting reports on the effects of fixation target size and luminance on fixation position control (Rolfs, 2009). Differences in eye-tracking techniques, the use of limited numbers of naïve subjects, and small sample sizes might contribute to the inconsistency of previously reported results (Steinman, 1965 and Rattle, 1969). Steinman (1965) found conflicting effects of target size on fixation accuracy in two subjects, although larger targets led to fewer microsaccades in both subjects. Rattle (1969) found a modest decrease in fixation accuracy for large targets, and a larger reduction in fixation accuracy for targets of foveal size. Studies on the effects of target luminance on fixation parameters have had more consistent results, but few luminance levels have been tested (Steinman, 1965).

In the present study, I conducted experiments to investigate the effects of the fixation target's size and luminance on the characteristics of microsaccades over a large range of stimulus parameters, among a large number of human subjects. Participants fixated a circular target of varying luminance and size while their eye movements were recorded with an infrared video tracker. The results showed a linear decrease in microsaccade rate versus a linear increase in microsaccade magnitude with target size. In the absence of the fixation target, larger and scarcer microsaccades were produced. These findings suggest that the physical properties of fixation targets can affect the outcome of visual and oculomotor studies. Thus, fixation target properties should be reported in all studies (which is not always the case. e.g. see (Bonneh, et al., 2010; Kanai & Kamitani, 2011 and Murakami, 2010). This possible effect needs to be considered in the future studies in the field, as well as in the interpretation of research results.

METHODS

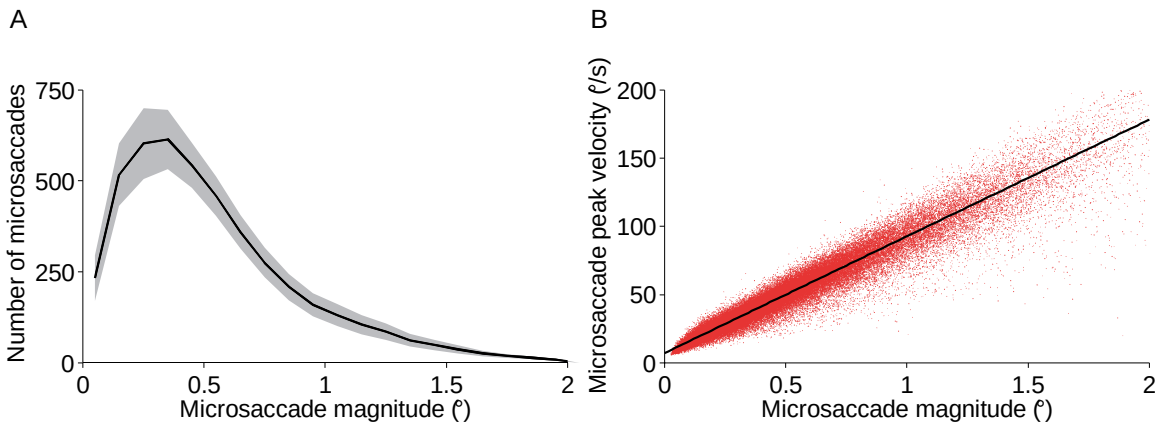
***Subjects*.** Seventeen adult subjects (12 male, 5 female) with normal or corrected-to-normal vision participated in the experiment. Age and education information was not obtained. Naïve subjects (16) were paid $15/session. Experiments were carried out under the guidelines of the Barrow Neurological Institute's Institutional Review Board (protocol number 04BN039). Written informed consent was obtained from each subject.

***Experimental design*.** Subjects were asked to rest their forehead and chin on the EyeLink 1000 head/chin support 57 cm away from a linearized video monitor (Barco Reference Calibrator V, 75 Hz refresh rate). They were instructed to look at the center of a circular target presented on the center of the monitor's screen, on a 50% gray background. Target luminance and size varied randomly across trials. Eleven possible luminance levels (ranging from 5% to 95% in 9% steps) and six possible radius sizes (0.033°, 0.067°, 0.133°, 0.267°, 0.533°, and 1.067°) resulted in a total of 66 experimental conditions. Note that, for a luminance level of 50%, there was no fixation target, and in this case the subjects were instructed to look at the center of the monitor. The experiment consisted of 4 sessions of ~30 minutes, with each session including 33 randomly interleaved trials of 30 seconds each. Each subject saw each fixation target twice (i.e. 60 seconds of presentation time for each visible fixation target condition) and the no fixation target condition 12 times (i.e. 360 seconds: 6 sizes at 50% luminance, with each size seen twice). Subjects took short (~2-5 min) breaks after each 11 trials. Subjects' eye position was calibrated at the beginning of the experimental session, and re-calibrated after each break. I used custom code and the Psychophysics Toolbox (Brainard, 1997; Kleiner,

Brainard, Pelli, Ingling, Murray, & Broussard, 2007 and Pelli, 1997) to display visual stimuli. To disregard the potential effect of the initial stimulus onset transient at the start of each trial, I conducted analyses only on data recorded after the first second of the trial.

***Eye movement analyses*.** Binocular eye position was acquired noninvasively with a video eye tracker at 500 Hz (EyeLink 1000, SR Research, instrument noise 0.01o RMS). Blinks were identified and removed as portions of the raw data where pupil information was missing. Also, portions of data where very fast decrease and increase in pupil area occurred (> 50 units/sample, such periods are probably semi-blinks where the pupil is never fully occluded) (McCamy M. , et al., 2012 and Troncoso, Macknik, & Martinez-Conde, 2008) were removed. 200 ms before and after each blink/semi-blink were added to eliminate the initial and final parts where the pupil was still partially occluded (Troncoso, Macknik, & Martinez-Conde, 2008). Saccades were identified with a modified version of the algorithm developed by Engbert and Kliegl (Engbert & Kliegl, 2003a; Engbert, 2006; Engbert & Mergenthaler, 2006; Laubrock, R, & Kliegl, 2005 and Rolfs, Laubrock, & Kliegl, 2006) with l = 4 (used for the velocity threshold detection) and a minimum saccadic duration of 6 ms. To reduce the amount of potential noise, only binocular saccades were considered, that is, saccades with a minimum overlap of one data sample in both eyes (Engbert, 2006; Engbert & Mergenthaler, 2006; Laubrock & Kliegl, 2005 and Rolfs, Laubrock, & Kliegl, 2006). Microsaccades were defined as saccades with magnitude < 2 degrees in each eye (Beer, Heckel, & Greenlee, 2008; Betta & Turatto, 2006; Hafed, Goffart, & Krauzlis, 2009; Martinez-Conde, Macknik, Troconso, & Dyar, 2006; Martinez-Conde, Macknik, Troncoso, & Hubel, 2009 and Troncoso X. ,

Macknik, Otero-Millan, & Martinez-Conde, 2008). This threshold was selected to accommodate the shift to increased microsaccade magnitudes that occurred with larger target sizes (**Figure 5C Inset**). When calculating microsaccade properties such as magnitude, peak velocity, and direction I averaged the values for the right and left eyes. See **Figure 4** for microsaccade descriptive statistics and the microsaccadic main sequence (peak-velocity relationship).



C

Microsaccade number (N)	Microsaccade rate (N/sec)	Magnitude (°)
4,495 (346)	1.17 (0.09)	0.52 (0.04)

Figure 4. ***Microsaccade characteristics.*** (**A**) Average microsaccade magnitude distribution across subjects and experimental conditions. Shadow indicates the SEM across subjects (n = 17) (**B**) Microsaccadic peak velocity-magnitude relationship for all subjects combined. Each red dot represents a microsaccade. (**C**) Microsaccade descriptive statistics. Numbers in parentheses indicate the SEM across subjects (n = 17).

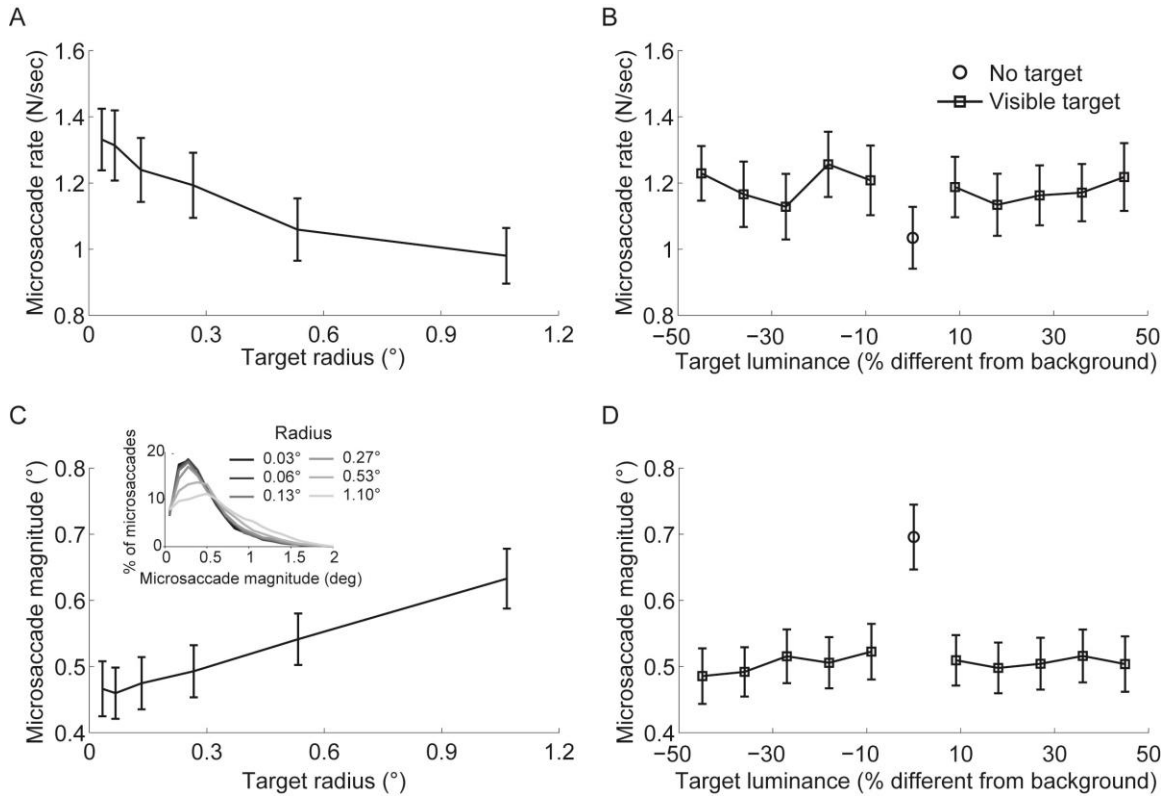


Figure 5. ***Effect of size and luminance on microsaccade rate and magnitude.*** (**A**) Microsaccade rate decreased linearly with target size ($F(5, 80) = 20.24$, $p < 0.0001$; linear trend $F(1, 16) = 16.00$, $p < 0.0001$). (**B**) Microsaccade rate did not change significantly with target luminance ($F(9, 144) = 2.14$, $p = 0.082$). (**C**) Microsaccade magnitude increased linearly with target size ($F(5, 80) = 28.96$, $p < 0.0001$; linear trend $F(1, 16) = 39.20$, $p < 0.0001$). (**D**) Microsaccade magnitude did not change significantly with target luminance ($F(9, 144) = 1.6$, $p = 0.121$). Microsaccade rate (**B**) was lower and microsaccade magnitude (**D**) was higher when the fixation target was absent compared to present (rate: $t(16) = 2.96$, $p = 0.009$; magnitude: $t(16) = -5.64$, $p < 0.001$). Error bars represent the SEM across subjects ($n = 17$).

Statistical methods. To assess the effects of target luminance and size on microsaccades, I performed a repeated measures ANOVA on each of the dependent variables: microsaccade rate, microsaccade magnitude. Target luminance (10 levels, I excluded the luminance level of 50% because it matched the background luminance, thus the target was invisible) and size (6 levels) were the within subjects factors variables. For violations of the ANOVA assumption of sphericity, *p*-values were adjusted using the Greenhouse-Geisser correction. To compare microsaccade characteristics with versus without a fixation target, I performed two tailed paired *t*-tests on the same dependent variables. In this case, I collapsed all fixation target conditions with luminance ≠ 50% for the fixation target condition; a fixation target with luminance = 50% corresponded to the no fixation target condition. Significance levels were set at $\alpha = 0.05$ throughout.

RESULTS

Effects of target size and luminance. Microsaccade rate decreased linearly with target size, whereas microsaccade magnitude increased linearly (**Figure 5A,C,E**). Target luminance did not affect microsaccade rate and magnitude (**Figure 5B,D,F**). There was no interaction between target luminance and size for any of these variables (all *F*-values < 1.4). The data found in this study showed for the first time that microsaccade magnitude increases with target size. Steinman (1965) found that larger fixation targets lead to fewer microsaccades— consistent with the present results—but did not investigate whether target size had an effect on microsaccade magnitude. The lack of effects of target luminance on microsaccade rate is consistent with previous results (Steinman, 1965).

Effects of presence versus absence of a fixation target. I investigated the effect of not having a fixation target on microsaccade parameters during fixation. To do this, I collapsed all the conditions with a fixation target (i.e. target luminance ≠ 50%) and compared them with the condition where there was no target (i.e. target luminance = 50%). Microsaccades were scarcer and larger without a target than with a target (**Figure 5B,D**). These findings extend and are consistent with, those of a recent report of smaller rates and larger microsaccade magnitudes during attempted fixation to the center of a black screen, compared to attempted fixation to a 0.0667° target with maximum contrast on a black background (Cherici, Kuang, Poletti, & Rucci, 2012). The present data also agree with the previous observation of lower microsaccade rates during the free-viewing exploration of blank scenes than during that of natural scenes (Otero-Millan, Troncoso, Macknik, Serrano-Pedraza, & Martinez-Conde, 2008).

DISCUSSION

I investigated the effect of fixation target size and luminance on microsaccade characteristics in 17 subjects, using a large number of size and luminance variables. Microsaccade magnitudes increased linearly with fixation target size, whereas microsaccade rates decreased linearly with fixation target size. Target luminance had no effect on microsaccade parameters. In the absence of a fixation target, microsaccade rates decreased and magnitudes increased.

A simple theory, similar to that described in (Timberlake, Wyman, Skavenski, & Steinman, 1972), may explain the effect of target size on microsaccades. When fixation targets are small enough to be confined within fovea, the role of visual errors signaled by deviations of the target image from fovea might be more essential to fixation stability, and proprioceptive inputs may not play a big role. When the fixation target is large and its border falls on more peripheral parts of retina, which have less spatial resolution, the role of proprioceptive inputs might be more significant than that of the retinal signals in fixation stability. The drawback is that proprioceptive signals are characterized with less spatial and temporal resolution than retinal signals (Hansen & Skavenski, 1977 and Van Beers, Sittig, & Denier van der Gon, 1998), which might contribute to delayed detection of fixation errors and subsequently production of less frequent and larger corrective microsaccades. This theory may account for the microsaccade rate decrease and magnitude increase with larger fixation target sizes. Also, it is consistent with the results found in the no fixation target condition, because in this condition the fixation target can be assumed to be as large as the entire monitor screen. One of the limitations of this hypothesis is that subjects might have relaxed their fixation with larger target sizes,

however despite being instructed to focus their gaze at the center of the fixation target (or the monitor, when the fixation target was not present).

The most important conclusion from this study is that fixation target properties should be tuned to the desirable microsaccade size in any given study. Also, because microsaccades influences object visibility, the characteristics of fixation targets may have uncontrolled and unwanted effects on the results of many psychophysical and physiological experiments. Thus, the characteristics of fixation targets characteristics should always be reported, so as to ensure replicability and facilitate data interpretation.

Chapter 4

A NOVEL VARIANT OF THE OUCHI-SPILLMANN ILLUSION

Classic Ouchi-Spillmann Illusion. Op artist Hajime Ouchi's book on "Japanese Optical and Geometrical Art", first published in 1973, featured a striking motion illusion in which a black-and-white checkered disk appeared to float and shift against a checkered background (**Figure 6**) (Ouchi, 1977). The illusion languished in obscurity until vision scientist Lothar Spillmann (1986) stumbled upon it and introduced it to the vision research community (Spillmann, Heitger, & Schuller, 1986), where it has enjoyed enormous popularity.

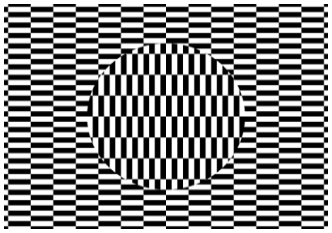


Figure 6. ***Classic Ouchi-Spillmann illusion.*** The central disk appears to float and shift against the background. The disk pattern has an opposite orientation to that of the background.

A new variant of the Ouchi-Spillmann Illusion. Here I report a novel variant of the Ouchi-Spillmann illusion. Whereas in the classical version the disk pattern has an opposite orientation to that of the background, the new illusion consists of a single

checkered pattern with an overlaid solid ring (that is either black or white). The ring's position becomes unstable upon observation, shifting from side to side if the checkered pattern is horizontal (i.e. in its longest orientation axis), or moving up and down if the checkered pattern is vertical (**Figure 7A, and B**).

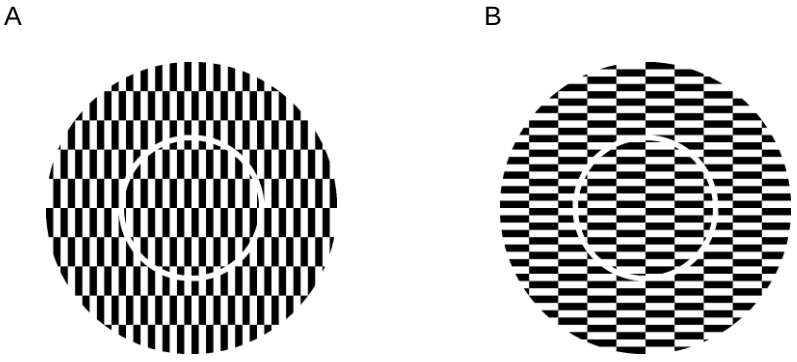


Figure 7. ***Novel variant of illusion and the effect of orientation.*** The ring motion is influenced by the orientation of checkered pattern. (**A**) The ring shifts from side to side if the pattern is horizontal, and (**B**) moves up and down if the pattern is vertical.

The strength of the illusion is enhanced for patterns made with elongated checks, and diminished for patterns with more symmetrical (i.e. square) checks. (**Figure 8 A and B**) As in the classical Ouchi-Spillmann's illusion, the observer's head and eyes movements appear to facilitate the perception of motion. I propose that the original Ouchi-Spillmann illusion and the novel variant reported here are modulated by the observer's exploratory and fixational eye movements, including both saccades and microsaccades. Further, the illusory motion in the new variant may result from eye

position changes along the same axis as the perceived direction of motion. Future work will determine whether exploratory and fixational eye movements, including both saccades and microsaccades, facilitate the perception of motion in these illusions.

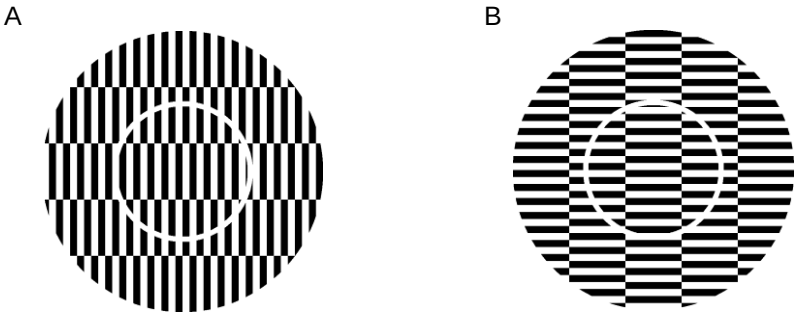


Figure 8. ***The effect of checker dimensions on illusion strength.*** The strength of the illusion is enhanced for patterns made of elongated checks vertically (A), or horizontally (B).

Chapter 5

CONCLUSIONS

Neuronal responses to microsaccades in area V1 are biphasic, with an excitatory component (peak) followed by an inhibitory component (trough). The peak may be attributed to retinal image displacements, and the trough to extraretinal signals. Thus, both retinal and extraretinal signals contribute to microsaccade-triggered neuronal responses in area V1. s. The inhibitory extraretinal component may contribute to perceptual suppression and visual stabilization during microsaccades (i.e. microsaccadic suppression). These findings further indicate that V1 neurons can distinguish between self-generated (i.e. ocular) motion and world motion.

Microsaccades characteristics are influenced by the physical properties of fixation targets. Microsaccade magnitudes increase linearly with fixation target size, while microsaccade rates rate decrease linearly with fixation target size. Luminance has no effect on microsaccade parameters. Because microsaccades affect the visibility of objects, the characteristics of fixation targets can have unwanted influences on many psychophysical and electrophysiological studies in which a fixation target is used. Thus, the physical properties of fixation targets should be both reported and tuned to the experiment's purposes.

Fixational eye movements, and particularly microsaccades, may play an essential role in the perception of visual illusions such as the classical Ouchi-Spillmann illusion and the novel variant reported here. More research is needed to determine the role of microsaccades and other fixational eye movements in these visual phenomena.

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