J Neurophysiol 116: 1507–1521, 2016.
First published July 6, 2016; doi:10.1152/jn.00268.2016.

Oculomotor inhibition covaries with conscious detection

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Submitted 31 March 2016; accepted in final form 5 July 2016

White AL, Rolfs M. Oculomotor inhibition covaries with conscious detection. *J Neurophysiol* 116: 1507–1521, 2016. First published July 6, 2016; doi:10.1152/jn.00268.2016.—Saccadic eye movements occur frequently even during attempted fixation, but they halt momentarily when a new stimulus appears. Here, we demonstrate that this rapid, involuntary "oculomotor freezing" reflex is yoked to fluctuations in explicit visual perception. Human observers reported the presence or absence of a brief visual stimulus while we recorded microsaccades, small spontaneous eye movements. We found that microsaccades were reflexively inhibited if and only if the observer reported seeing the stimulus, even when none was present. By applying a novel Bayesian classification technique to patterns of microsaccades on individual trials, we were able to decode the reported state of perception more accurately than the state of the stimulus (present vs. absent). Moreover, explicit perceptual sensitivity and the oculomotor reflex were both susceptible to orientation-specific adaptation. The adaptation effects suggest that the freezing reflex is mediated by signals processed in the visual cortex before reaching oculomotor control centers rather than relying on a direct subcortical route, as some previous research has suggested. We conclude that the reflexive inhibition of microsaccades immediately and inadvertently reveals when the observer becomes aware of a change in the environment. By providing an objective measure of conscious perceptual detection that does not require explicit reports, this finding opens doors to clinical applications and further investigations of perceptual awareness.

microsaccades; oculomotor inhibition; perceptual awareness; contrast sensitivity; visual adaptation

NEW & NOTEWORTHY

The eyes freeze in response to stimulus onsets. We developed a novel method to compare the sensitivity of this involuntary reflex to that of explicit perceptual detection. The two responses had similar contrast thresholds and were similarly affected by pattern adaptation. They also covaried across individual trials: the eyes froze if and only if the observer reported seeing a stimulus, even when none was present. Oculomotor inhibition therefore rapidly reveals the state of conscious perception.

THE EYES are rarely still. Even when an observer fixates on a single point, spontaneous microsaccades correct gaze position, prevent and counteract visual fading, and explore tiny image details (Martinez-Conde et al. 2013; McCamy et al. 2014; Otero-Millan et al. 2013; Rolfs 2009; Rucci and Victor 2015). The onset of an irrelevant stimulus inhibits these movements within 100 ms, followed by a rebound in the microsaccade rate (Engbert and Kliegl 2003; Hafed and Ignashchenkova 2013). The particular timing and magnitude of these microsaccade rate changes depend on stimulus parameters (Bonneh et al. 2015; Rolfs et al. 2008). Similarly, irrelevant stimulus onsets delay the execution of large voluntary eye movements (Bompas and Sumner 2009; Reingold and Stampe 2002; Sumner et al. 2006; Walker et al. 1997). We refer to these rapid inhibitory reflexes collectively as "oculomotor freezing."

Oculomotor freezing is an instantiation of stimulus detection by the oculomotor system, yet it is unknown whether it covaries with explicit perceptual detection: the observer's perception of a change in the environment that he or she can voluntarily report (by pressing a button, for instance). By "explicit perceptual detection," we refer to a concept related, but not necessarily identical, to perceptual awareness: the observer's conscious experience of a stimulus (Hochstein and Ahissar 2002; Kihlstrom et al. 1992). Visual awareness is selective: we fail to see many objects that appear in front of our eyes if they are physically below sensitivity thresholds, obscured by other salient stimuli, or filtered out by selective attention (Kim and Blake 2005). The internal mechanisms that determine whether or not a stimulus will be consciously detected are hotly debated (e.g., Dehaene and Changeux 2011; Tononi and Koch 2008).

Indeed, awareness per se is notoriously difficult to define and measure (Dehaene and Changeux 2011; Dehaene et al. 2003, 2006; Reingold and Merikle 1988; Sandberg et al. 2010; Seth et al. 2008; see DISCUSSION). In the present report, we measured "explicit perceptual detection" by having observers report whether or not they saw a visual pattern flashed briefly on a computer screen. Given some assumptions about this perceptual task (outlined in the DISCUSSION), we may infer from the observer's explicit reports whether or not he or she consciously detected a stimulus. We asked whether or not the observer's explicit perceptual reports correlate with oculomotor behavior. That is, do the mechanisms that give rise to conscious detection also trigger the oculomotor system's inhibitory response to sensory stimulation?

On the one hand, there are known dissociations between explicit perception and oculomotor control, cases in which eye movements respond differently to visual input than perceptual reports do (Spering and Carrasco 2015). Oculomotor freezing could be another such case: the rapid, reflexive inhibition of tiny involuntary eye movements could well be dissociated from the sluggish and selective phenomenon of perceptual awareness (the presumed basis of explicit perceptual reports). Indeed, some researchers have speculated that oculomotor freezing is controlled by subcortical circuits independent of conscious perception, such as the direct retinotectal pathway to the superior colliculus (Engbert 2006; Reingold and Stampe 2002; Ro et al. 2004; Rolfs et al. 2008; Sumner et al. 2006).

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On the other hand, oculomotor freezing and explicit perception might share fundamental detection mechanisms, such that one provides a robust proxy of the other. Such a link would be equally informative about the underlying brain circuitry but more useful for future applications (Martinez-Conde and Macknik 2008). The oculomotor reflex could provide an objective window into perceptual awareness without relying on subjective reports from the observer, an approach that is increasingly recommended in cognitive neuroscience (Hannula et al. 2005; Tsuchiya et al. 2015). Consistent with that hypothesis, two recent studies have reported that perceptual contrast thresholds can be inferred from stimulus-induced changes in the microsaccade rate (Bonneh et al. 2015; Scholes et al. 2015).

However, two important questions remain unanswered. First, how does oculomotor freezing relate to explicit perceptual detection on individual trials? Similar effects of stimulus intensity in the aggregate do not imply that microsaccade patterns track trial-by-trial fluctuations in conscious stimulus detection. In other words, the perceptual and oculomotor systems may show weak correlations across multiple presentations of identical stimuli, although, on average, they respond similarly to manipulations of stimulus parameters. The alternate possibility is that each stimulus that the observer reports seeing also triggers the oculomotor reflex, whereas unseen stimuli do not. Second, what is the locus of covariation between explicit perceptual detection and oculomotor freezing? Do the two outputs have similar contrast sensitivity because they share subcortical sensory processing (such as in the retina) or because they share detection mechanisms in the cortex?

We investigated how the rapid inhibition of microsaccades relates to explicit perception of the stimulus that triggers it. To do so, we simultaneously measured perceptual detection and oculomotor freezing, assessing their *1*) sensitivity to luminance contrast, *2*) susceptibility to orientation-specific adaptation, a hallmark of cortical processing (Kohn 2007; Solomon et al. 2004), and *3*) covariation across identical trials. The first two of those investigations demonstrated that the two behavioral outputs rely on similar sensory processing. The third investigation demonstrated that the oculomotor freezing reflex does not depend directly on physical stimulation but rather is contingent on the observer seeing a stimulus, even when none was present. In a proof-of-principle demonstration, we then used oculomotor behavior in individual trials to "decode" *1*) whether a stimulus was physically present or absent and *2*) whether the observer reported that a stimulus was present or absent. Decoding accuracy was higher for the subjective report than for physical stimulus presence, consistent with the conclusion that oculomotor freezing tracks fluctuations in perceptual awareness.

MATERIALS AND METHODS

Participants

A total of 33 observers (age: 20–42 yr old) with normal or corrected-to-normal vision participated in exchange for a fixed monetary payment. All but three observers were naive as to the research aims, and all gave informed consent. The Ethics Committee of the German Society for Psychology approved the study.

Apparatus and Stimuli

Observers sat in a darkened room with their head on a chin rest, 57 cm from a gamma-corrected ViewPixx 3D Display (VPixx Technologies), which uses a LCD screen with a scanning backlight to control stimulus presentation with high temporal resolution. We recorded the gaze position of both eyes at 500 Hz with a head-mounted Eyelink 2 system (SR Research, Kanata, ON, Canada). Stimuli were controlled and data were collected with Psychophysics and Eyelink toolboxes (Brainard 1997; Cornelissen et al. 2002; Pelli 1997). The grayscale display (1920 × 1080 pixels, 120-Hz refresh rate) had 10 bits of resolution in luminance. The background luminance was set to 35% of its maximum (32.5 cd/m^2), permitting 358 equally spaced values of Michelson luminance contrast.

The fixation mark was a 2 × 2-pixel black-and-white checkerboard pattern of width 0.055 degrees of visual angle (dva). In between trials, this mark was replaced by a circle (0.083 dva radius) of alternating black and white pixels. The target stimuli were Gabor patterns: sinusoidal gratings windowed by a two-dimensional Gaussian (σ = 0.67 dva).

Procedures for Experiments

Procedure for experiment 1. Twelve observers (4 men and 8 women, age: 23–36 yr old, 2 authors) participated. Observers began each trial (Fig. 1, *top* row) by fixating on the central mark. After 0.5–2.5 s, the target Gabor stimulus flashed for 3 ms (the rise to fall time of 1 frame). The target's onset time had a roughly flat hazard rate: it was set to 0.5 s plus a value drawn from an exponential distribution (mean: 0.65 s) that was clipped at 2 s. The target Gabor was always vertically oriented, with the spatial frequency (SF) set to 0.75 cycles per degree of visual angle (cpd). Its phase on each trial was randomly set to either 0° or 180°. On 50% of the trials, the target had nonzero contrast (present trials). On the remaining trials, its contrast was set to 0, causing no change on the screen (absent trials). The fixation mark remained visible at the center of the Gabor; 492 ms after the target onset, a beep (400 Hz, 50 ms, delivered through headphones) indicated that the trial was over.

The task was to indicate whether the target was present or absent by pressing the up or down arrow, respectively, with the right hand. The response time was unlimited, but responses were not allowed before the beep. A high- or low-pitched tone (600 or 180 Hz) indicated whether the response was correct or incorrect. After an intertrial interval (700 ms) containing only the circular fixation mark, the next trial began. Each observer completed 10 ± 1 testing sessions of 1 h. The first session began with practice and then two blocks of staircase trials to estimate the observer's contrast threshold.

During the staircase blocks, the contrast was adjusted after each trial according to the single interval adjustment matrix staircase procedure (Kaernbach 1990). The contrast adjustment depended on the stimulus and response: after a hit, −0.3 $\log_{10}$ units; miss, +0.3 $\log_{10}$ units; false alarm, +0.6 $\log_{10}$ units; and correct rejection, no adjustment. The magnitudes of these steps were halved after the first and second staircase reversals. In each block, we interleaved two staircases, one starting at a relatively high and the other at a low level of contrast. The block ended when both staircases underwent 10 additional reversals. The mean contrast of all but the first two reversal points provided a threshold estimate. We defined the observer's contrast threshold as the mean of four threshold estimates (2 from each of 2 blocks).

In the main experimental blocks, the target's contrast level on each target-present trial was drawn randomly from a set of eight, equally spaced in $\log_{10}$ units. These contrast levels spanned a range of 1.2 $\log_{10}$ units, with the maximum contrast being 0.48 $\log_{10}$ units above the observer's staircase threshold (greater range below the threshold to allow for learning and ensure accurate measures of low-contrast oculomotor responses).

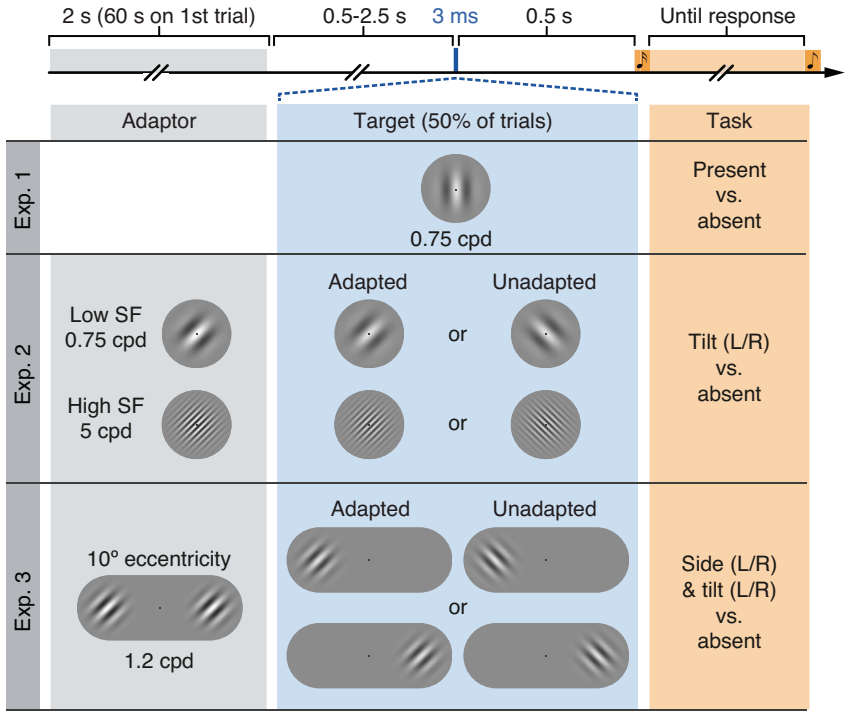

Fig. 1. Stimuli and trial sequence in *experiments 1–3*. The trial time-course is represented at the top of the figure, with example stimuli below, in separate rows for each of the 3 experiments. SF, spatial frequency; L, left; R, right.

Each block of the main experiment consisted of 96 trials: 48 trials with 0 contrast and 6 trials with each of the 8 nonzero contrasts, randomly interleaved. Each observer completed between 48 and 50 blocks, with an average of 6 blocks/day, leading to a total of ~4,900 trials. In one-third of the blocks, we played an additional "click" sound 492 ms before each trial's end, simultaneous with the target. The purpose of the click was to reduce all temporal uncertainty about the target's onset, and, indeed, the perceptual thresholds were slightly lower than without the click [means: 6.3% vs. 6.9%, $t(11) = 4.66$, $P < 0.001$]. These trials were not analyzed further because the sound itself inhibits microsaccades (Rolfs et al. 2005, 2008), preventing a meaningful calculation of oculomotor sensitivity.

Procedure for experiment 2. Twelve observers (3 men and 9 women, age: 20–33 yr old) participated, including two observers who also did *experiment 1*. The stimuli and procedure matched *experiment 1* with the following exceptions (Fig. 1, *middle* row). We tested two SFs of the target Gabor, 0.75 and 5 cpd. Each trial was preceded by the presentation of a 100% contrast adaptor grating, with the same size and SF as the target Gabor. Its orientation was constant for a block of trials, either +45° or −45° relative to vertical. To cancel out any retinal luminance adaptation, its phase changed every 83 ms, cycling between 0, 90, 180, and 270° at 3 Hz in steps alternating between 90° and 180°. The adaptor was presented for 60 s before the first trial of each block and for 2 s before each succeeding trial.

The target was present on 50% of trials, and its orientation was either +45° or −45°, intermixed randomly across trials. Thus, its orientation was equally likely to be parallel or orthogonal to the adaptor. The target's contrast was fixed to the observer's detection threshold (measured in initial staircase blocks) and adjusted if necessary across testing sessions if performance exceeded an average of 90% correct or dropped below 70% correct. There was never a click simultaneous with the target. The observer's task was to press the down arrow key if there was no target or the left or right arrow key if there was a target titled clockwise or counterclockwise of vertical, respectively.

Observers completed eight 1-h sessions, conducted on separate days. In the first two sessions, we measured contrast thresholds for both SFs (low SF: 4.5 ± 0.7% and high SF: 10.1 ± 2.4%). Each of the following sessions consisted of 8 blocks of 36 trials. The SF alternated across sessions, with the order counterbalanced across observers. Each observer provided ~1,650 trials.

Procedure for experiment 3. Twelve observers participated (6 men and 6 women, age: 23–42 yr old), including one observer from *experiment 2*. The stimulus and procedure differed from *experiment 2* as follows: two adaptor gratings with the same orientation were presented simultaneously, one on the left and one on the right side of fixation at 10 dva eccentricity (Fig. 1, *bottom* row). We tested only one SF, 1.21 cpd, scaled for cortical magnification to match the foveal 5 cpd gratings in *experiment 2* (Rovamo and Virsu 1979). On 50% of the trials, we flashed a single target Gabor at one of the adapted locations, tilted either +45° or −45°. Its contrast was fixed to each observer's detection threshold (20.0 ± 9.5%). If observers saw the target, they reported its location (left vs. right side) and tilt (left vs. right of vertical) by pressing one of four keys: two keys for the left hand, to report the orientation of targets on the left side, and similarly two keys for the right hand and right side. If they saw no target, they pressed the space bar. Observers completed four 1-h sessions, for a total of ~775 trials each. In the first session, we trained the observer and measured contrast thresholds with two blocks of staircases.

Eye Tracking

At the start of each block, we performed a 9-point calibration within a central square region 21 dva wide. Every 24 trials, we performed a calibration drift correction by having the observer press a key while fixating a dot at the screen's center. If either eye's gaze position deviated >2 dva from the fixation mark between the start of a trial start and the beep, that trial was immediately terminated and repeated at the end of the block. In *experiments 2* and *3*, if the eye-tracker had to be recalibrated and >150 s had elapsed since a long-duration adaptor, another 30-s adaptor was presented before the trials resumed. In addition, the radius of the acceptable region was expanded to 3 dva, to accommodate drift during the prolonged fixation periods. We also detected fixation breaks offline by defining, for each trial, the fixation position as the median gaze coordinates during the adaptor and fixation breaks as deviations >2 dva from that. Trials with offline-detected fixation breaks were excluded from the analysis (2% of trials on average).

Perceptual Data Analysis

We excluded trials with reaction times >3 SDs above the observer's median (1–2% of trials). In *experiment 1*, we computed perceptual sensitivity (d') at each contrast using the observer's hit rate (HR) for that contrast and false alarm rate (FAR) from all target-absent trials as follows:

$$d' = z(\mathrm{HR}) - z(\mathrm{FAR})$$

where z is the normal z-score function. To avoid undefined d' values, HR and FAR were not allowed to fall below $1/(2N)$ nor exceed $[1 - 1/(2N)]$, where N is the number of target-present or target-absent trials. For example, if the HR was 1, we assumed that had we run twice as many trials there would have been 1 miss. We fit d' with a Naka-Rushton function (Albrecht and Hamilton 1982; Naka and Rushton 1966) of contrast (c). The function had three free parameters: the slope (n), upper asymptote (A), and half-maximum contrast threshold (c_{50}):

$$d'(c) = Ac^n/(c^n + {c_{50}}^n)$$

In *experiments 2* and *3*, we computed d' for the adapted and orthogonal orientations separately. The HR was the proportion of target-present trials with a particular target orientation in which the observer reported seeing that orientation. The FAR was the proportion of trials when that orientation was not present (either no target or the other orientation was present) but the observer reported seeing it. In *experiment 3*, observers also reported the location of the target, primarily to avoid conflicts in preparing single response for a stimulus that could vary both in its orientation (left/right) and location (left/right). For simplicity, the accuracy of the location report was not considered in analyzing perceptual sensitivity, for which we were primarily interested in the effect of orientation-specific adaptation.

Microsaccade Detection

We first transformed the raw gaze positions into velocities (in dva/s) and smoothed them by averaging over neighboring pairs of two samples. We then identified microsaccadic events as shifts in gaze position with two-dimensional velocities that exceeded, for at least three samples, an ellipse with horizontal and vertical radii equal to five times the horizontal and vertical median-based SDs, respectively (Engbert and Mergenthaler 2006). Monocular microsaccadic events <10 ms apart were merged together. We defined binocular microsaccades as those with at least one sample of overlap between the two eyes, and, again, we merged binocular microsaccades <10 ms apart. We defined microsaccade onset as the time the first of the two eye velocities exceeded the threshold and offset as the timepoint just before the last eye's velocity dropped below threshold. Other parameters (e.g., amplitude) were averaged over the two eyes.

We included in the analysis only binocular microsaccades with durations ≥6 ms, amplitudes ≤1 dva, and peak velocities ≤250 dva/s. In *experiment 1*, we found that the majority of detected saccades were <30 min of arc in amplitude (89%), with peak velocities <65 dva/s (93%) and durations <30 ms (85%) In *experiment 2*, those percentages were 79%, 83%, and 88%, respectively. In *experiment 3*, they were 82%, 91%, and 78%, respectively.

For two observers (one observer each in *experiments 1* and *2*), the fixed velocity threshold of 5 SDs yielded very few microsaccades (and a poor oculometric function fit in *experiment 1*). For these observers, we lowered the threshold (λ) using an adaptive procedure (Engbert and Mergenthaler 2006). For a wide range of λ values, we compared the microsaccade rate detected from the true data with the microsaccade rate detected in phase-randomized amplitude-adjusted surrogate data (Theiler et al. 1992). At the optimal λ, the true microsaccade rate is equal to the maximum difference in true and surrogate rates (procedure fully described in Mergenthaler 2008). The resulting velocity thresholds for these two observers were 3.0 and 3.2 SDs. If we adjusted the thresholds individually for all observers, they tended to be <5, and we found all the same patterns in the data but with considerably more noise.

Microsaccade Analysis

We then determined the time-varying microsaccade rate for each experimental condition (e.g., contrast level) with a smoothing procedure (example in Fig. 2*A*). First we counted the number of microsaccades detected at each millisecond (t) relative to the target onset across all trials in a given condition. For each time point t, we then computed a weighted sum of microsaccades in the local interval, using the "causal" kernel, as follows:

$$\omega(\tau) = \alpha^2 \tau e^{-\alpha\tau}$$

where ω describes the weight given to microsaccades τ ms before time point t. We shifted the filter by $1/\alpha$ ms to avoid a temporal bias and give the most weight to microsaccades at point t (Rolfs et al. 2008; Widmann et al. 2014). Parameter α was set to 1/25. The smoothed rate $r(t)$ is the weighted sum of microsaccades divided by the total number of trials in the sample and converted into Hz by multiplying by 1,000. Microsaccade rates were computed from −350 to +500 ms relative to the target onset.

To estimate the statistical significance of changes in microsaccade rates, we bootstrapped them by simulating 1,000 repetitions of the experiment (Efron and Tibshirani 1993). On each repetition, we resampled with replacement from the set of observers. For each resampled observer, we generated new data by transforming the microsaccade rates into probabilities of a microsaccade at each millisecond and then drawing from binomial distributions with those probabilities. After smoothing the new rates, we generated distribu-

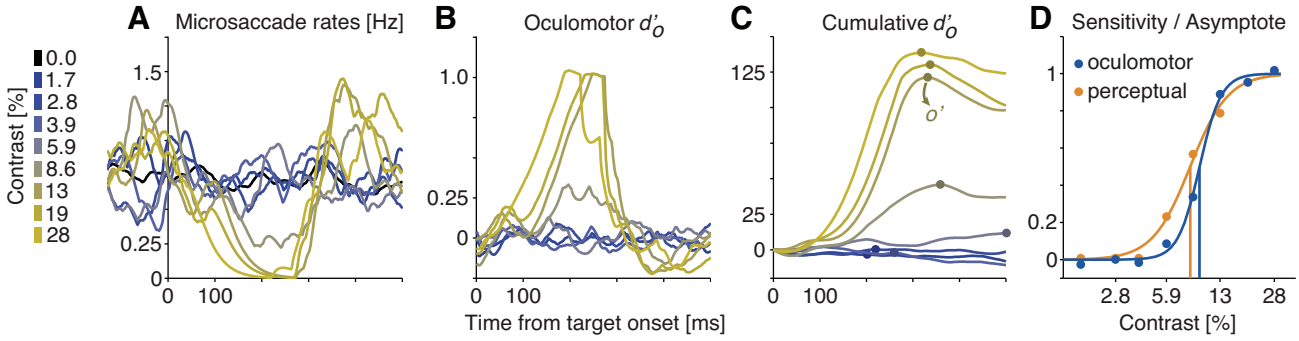

Fig. 2. Data from an example observer in *experiment 1*. *A*: microsaccadic rates as a function of target contrast and time from the target onset. *B*: oculomotor sensitivity d'_o. *C*: the cumulative sum of d'_o. Dots indicate the maxima, o'. *D*: oculometric (o'; blue) and psychometric (d'; orange) functions of contrast, normalized to the best-fitting upper asymptotes. The vertical lines indicate half-maximum thresholds (c_{50}).

tions of differences between microsaccade rates across conditions (e.g., target present vs. absent). The two-tailed bootstrapped P value was defined as twice the proportion of differences that fell below zero. When evaluating differences at many time points, we applied the false discovery rate correction (Benjamini and Hochberg 1995). Two conditions were deemed significantly different if the 95% confidence interval of differences did not include zero (corrected $P < 0.05$).

To directly compare the microsaccade rate changes to perceptual sensitivity, we computed an analogous estimate of oculomotor sensitivity (Fig. 2*B*). At each millisecond, the lack of a microsaccade after a stimulus was a "hit" and the lack of a microsaccade after no stimulus was a "false alarm." From the resulting oculomotor HR and FAR, we computed oculomotor sensitivity (d'_o) at each time point t relative to the stimulus onset as follows:

$$d'_o(t) = z\left[\mathrm{HR}(t)\right] - z\left[\mathrm{FAR}(t)\right]$$

Like perceptual d', this measure requires correction if HR or FAR reach extreme values. This can happen if no microsaccade was detected during a period around t as wide as the base (B) of the filter (~200 ms). Therefore, both rates were not allowed to fall below $1/(2NB)$ or to exceed $[1 - 1/(2NB)]$, where N is the number of target-present or target-absent trials, respectively. That is, we assumed that had we run twice as many trials, we would have found at least one microsaccade (a "miss") in the 200-ms time window surrounding any given time point. Nonetheless, because microsaccades occur only about once or twice every second, both HR and FAR at individual (ms) time points tended to be high (above 0.999). But because HR rose even higher than FAR after stimulus presentation, we found positive values of d'_o that are immune to any stimulus-independent changes in a bias to produce microsaccades or not (similar to how perceptual d' is immune to changes in the observer's bias to report present or absent).

To extract a single oculomotor sensitivity measure from an entire rate time course for a given condition, we defined a value for o', the maximum of the cumulative sum of d'_o values across time (Fig. 2*C*). o' is unaffected by rate rebounds after inhibition, which result in negative d'_o. In *experiment 1*, we fitted a Naka-Rushton function of contrast to these o' values, just like for d' (Fig. 2*D*). However, by taking the maximum of the cumulative sum, we were biasing o' to be high and, due to noisy fluctuations, likely overestimating it, especially at low contrasts. Accordingly, we fitted the Naka-Rushton functions with an additional parameter for the lower asymptote, which we then subtracted from all o' values before refitting them. Although perceptual d' and oculomotor o' values and their upper asymptotes cannot be compared directly, the estimated c_{50} values can.

In *experiments 2* and *3*, when we computed perceptual d', we controlled for any difference in bias or criterion between the parallel and orthogonal orientations by measuring separate FARs for each. However, we do not know which orientation the oculomotor system "saw" when microsaccades were inhibited on any given trial. It is therefore possible, in principle, that the adaptation effect in o' is at least in part due to a change in criterion for deciding a particular orientation was present. However, if the oculomotor response relied on neural substrates independent of perception (such as a direct subcortical route), it would be unlikely to have an orientation-dependent criterion.

Single Trial Classification

In this analysis, we used the information contained in microsaccade timing to classify individual trials as stimulus present/absent and perceptual report as present/absent. One recent study classified stimulus presence (but not the percept) by applying a linear support vector machine to temporally coarse microsaccade patterns (Scholes et al. 2015). We took a different approach, based on the principles of Bayesian inference, and we compared the classification of stimulus presence with the classification of the observer's percept.

Microsaccades are more informative if they occur at times relative to target onset when the mean microsaccade rates strongly differ between present and absent trials. Moreover, the absence of any microsaccade after stimulus onset provides some evidence in favor of stimulus (or, report) presence, given the general occurrence of inhibition on present trials.

For each observer, we first computed "priors" on a "training set" of the data. This began by computing smoothed microsaccade rates in each condition, which we transformed into the probability of observing a microsaccade (M) at each time point t, contingent on trial type: "yes" (Y) or "no" (N). For the classification of stimulus presence, trials with target stimuli present were Y trials and trials without targets were N trials. For the classification of perceptual reports, trials with reports of target presence were Y trials and reports of target absence were N trials. Those probabilities were expressed as $p[\mathrm{M}(t) \mid \mathrm{Y}]$ and $p[\mathrm{M}(t) \mid \mathrm{N}]$. We also computed the prior probability of "yes" trials $[p(\mathrm{Y})]$ and of microsaccades (or lack thereof) in the final 500 ms of each trial $[p(\mathrm{M})]$.

On independent "test" trials, we then computed the posterior probability $[p(\mathrm{Y} \mid \mathrm{E})]$ that each trial was a "yes" given the observed pattern of eye movements (E) in the last 500 ms. If E contained no microsaccades,

$$\begin{aligned} p(\mathrm{Y} \mid \mathrm{E}) &= p(\mathrm{Y} \mid \sim \mathrm{M}) \\ &= p(\sim \mathrm{M} \mid \mathrm{Y}) \times p\left(\mathrm{Y}\right) / p\left(\sim \mathrm{M}\right) \end{aligned}$$

where $p(\sim \mathrm{M} \mid \mathrm{Y})$ is the probability of observing no microsaccade on "yes" trials, $p(\mathrm{Y})$ is the prior probability of "yes" trials, and $p(\sim \mathrm{M})$ is the prior probability of observing no microsaccades.

On trials with n microsaccades at times $T = \{t_1, \ldots, t_n\}$, for each microsaccade i, we computed the following:

$$p[\mathrm{Y} \mid \mathrm{M}(t_i)] = p[\mathrm{M}(t_i) \mid \mathrm{Y}] \times p\left(\mathrm{Y}\right) / p[\mathrm{M}(t_i)]$$

where $p[\mathrm{M}(t_i) \mid \mathrm{Y}]$ is derived from the smoothed microsaccade rate on "yes" trials and $p[\mathrm{M}(t_i)]$ is derived from the smoothed rate collapsed across "yes" and "no" trials. Note that this analysis takes into account both the initial inhibition and subsequent rebound in microsaccade rate (unlike our computation of oculomotor d'_o which relies only on the inhibition).

In the case of $n > 1$, we then integrated the posterior probabilities across individual microsaccades such that:

$$p(\mathrm{Y} \mid \mathrm{E}) = \Pi p[\mathrm{Y} \mid \mathrm{M}(t_i)] / p\left(\mathrm{Y}\right)^{n-1}$$

We then classified each trial as "yes" or "no" by comparing $p(\mathrm{Y} \mid \mathrm{E})$ to some criterion (C). To estimate a criterion-free measure of the accuracy, we conducted a receiver operating characteristic (ROC) analysis by varying C across the full range, computing HR and FAR, and then the area (A') under this ROC curve. A' varied from 0.5 (chance) to 1.0 (perfect) and is therefore similar to percent correct but bias free.

For each analysis, we divided the trials into 10 sets, using each set in turn as the "test set" while the remaining 9 sets pooled together served as the "training" set. In *experiment 3*, we had fewer trials per condition. Test sets with 1/10 of the data often had 100% "yes" or "no" trials, leading to unstable accuracy measures, so we divided the data into only 5 test sets.

In *experiment 1*, we classified stimulus and report presence versus absence collapsed across all contrast levels. For stimulus classification, we also tested each contrast level individually, using as the "training set" all contrasts together. (When we "trained" using each contrast level separately, accuracy levels were slightly lower on average.) Similarly, in *experiments 2* and *3*, we classified stimulus and report for all orientations and stimulus presence for adapted and unadapted orientations independently (using all trials together as training).

In a final analysis, when classifying trials in each observer, we used the average of all other observers to estimate the priors: $p[\mathrm{M}(t) \mid \mathrm{Y}]$,

p[M(t)], p(Y), and p(~M). This classification tested whether one observer's microsaccades can predict stimulus/report presence even when we didn't know his or her own typical microsaccade rates but rather assume that he or she was like the average of all other observers.

RESULTS

Experiment 1: Contrast Sensitivity

In *experiment 1*, we measured how perceptual and oculomotor sensitivity vary with luminance contrast. (By "perceptual sensitivity," we refer to the sensitivity of explicit, voluntary perceptual reports, as opposed to the involuntary eye movement responses). We recorded the observers' binocular gaze position as they fixated a small point and monitored for a target stimulus. The target was a Gabor patch: a vertical sinusoidal luminance grating with a contrast level chosen randomly on each trial from a set of eight. The target appeared briefly (duration: 3 ms) at fixation at an unpredictable time on a random 50% of trials (Fig. 1, *top* row). Each trial ended with a beep 0.5 s later that prompted the observer to report whether or not they saw a target, providing our measure of perceptual sensitivity (d').

To estimate oculomotor sensitivity from each observer's eye movement traces, we first computed smoothed microsaccade rates at each time point relative to the target onset (example in Fig. 2*A*). As luminance contrast increased, the inhibition of microsaccades became stronger and began earlier (average shown in Fig. 3*A*). Only the upper four contrast levels caused significant inhibition compared with the spontaneous baseline rate (corrected $P < 0.05$, indicated by thicker lines in Fig. 3*A*), becoming significant by 104, 113, 125, and 175 ms after the target onset, respectively, in descending order. Based on previous reports that microsaccades are inhibited by stimulus onsets, we treated the lack of a microsaccade as a "report" of stimulus presence and the occurrence of a microsaccade as a "report" of stimulus absence. We then used classic signal detection theory to transform the microsaccade rates at each time point into oculomotor sensitivity in d'_o units (see MATERIALS AND METHODS). The maximum of the cumulative sum of oculomotor d'_o across time provided a single sensitivity measure for each contrast: o' (Fig. 2*B*).

Note that the absolute value of o' has no meaning but can be used to compare oculomotor responses across conditions. In addition, this measure captures only the initial microsaccadic inhibition and not the later rebound in microsaccade rate, because we are primarily interested in the most rapid oculomotor responses to the stimulus. Moreover, we observed relatively little rebound in the 500 ms between the stimulus and the beep prompting the observer's response, because manual response preparation itself inhibits eye movements (Betta and Turatto 2006). Other studies, in contrast, have investigated the rate rebound triggered by irrelevant stimuli that observers did not respond to (Bonneh et al. 2015; Scholes et al. 2015).

We then fit both perceptual d' and oculomotor o' values with independent Naka-Rushton functions of contrast (average $r^2 = 0.96$ and 0.99, respectively; Fig. 3*B*), which allowed us to compare their slopes and their thresholds (the contrast values needed to reach half of the upper asymptote).

The oculomotor contrast thresholds (mean ± SD: 7.4 ± 0.4%) were indistinguishable from the perceptual thresholds [6.9 ± 0.2%, $t(11) = 1.26$, $P = 0.24$; Fig. 3*C*]. On average, the oculometric slopes were steeper than the psychometric slopes, but the difference was not statistically reliable [$t(11) = 2.0$, $P = 0.07$; Fig. 3*C*]. The slope difference could indicate that the oculomotor response was more consistent across trials. Overall, however, perception and the oculomotor system respond very similarly to luminance contrast, detecting stimuli at about the same intensity levels. This is consistent with the results of two recent studies that predicted perceptual contrast thresholds from microsaccade patterns (Bonneh et al. 2015; Scholes et al. 2015).

Experiments 2 and 3: Orientation-Specific Adaptation

Next, we probed the locus of the sensory processing that underlies microsaccadic inhibition by exposing the observer to an oriented adaptor stimulus for several seconds before each trial. Such adaptation selectively reduces perceptual sensitivity for stimuli with orientations similar to the adaptor, especially when both have high spatial frequency (Blakemore and Campbell 1969; Heinrich and Bach 2002). We therefore reasoned that if microsaccadic inhibition relies on similar detection mechanisms as explicit perception, then it should also be weakened in response to stimuli with adapted orientations, in particular for high SFs.

In *experiment 2* (Fig. 1, *middle* row), we measured perceptual and oculomotor sensitivity for foveal stimuli of two different SFs, with orientations either parallel or orthogonal to a preceding adaptor stimulus of the same SF. In *experiment 3* (Fig. 1, *bottom* row), we repeated the adaptation paradigm for stimuli of a single SF placed 10 dva from fixation, in case the contribution of direct retinotectal projections is stronger outside the fovea (Perry and Cowey 1984). In both *experiments 2* and *3*, we fixed the stimulus contrast at each individual observer's detection threshold.

Microsaccade rates (Fig. 4*A*) showed clear signs of orientation adaptation: targets parallel to the preceding adaptor

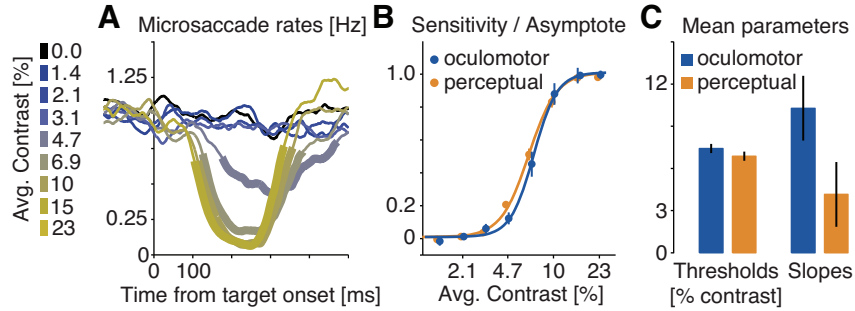

Fig. 3. Perception and oculomotor freezing are similarly sensitive to luminance contrast. *A*: mean microsaccade rates in *experiment 1*. The thicker lines indicate significant differences from the baseline rate with contrast zero ($P < 0.05$ from bootstrapping, false discovery rate corrected). *B*: Naka-Rushton function fits to the average oculomotor (o'; blue) and perceptual (d'; orange) sensitivity values, normalized by their upper asymptotes. *C*: means of threshold and slope parameter estimates from observers' individual fits. Error bars are ±1 within-subject SEs (Morey 2008).

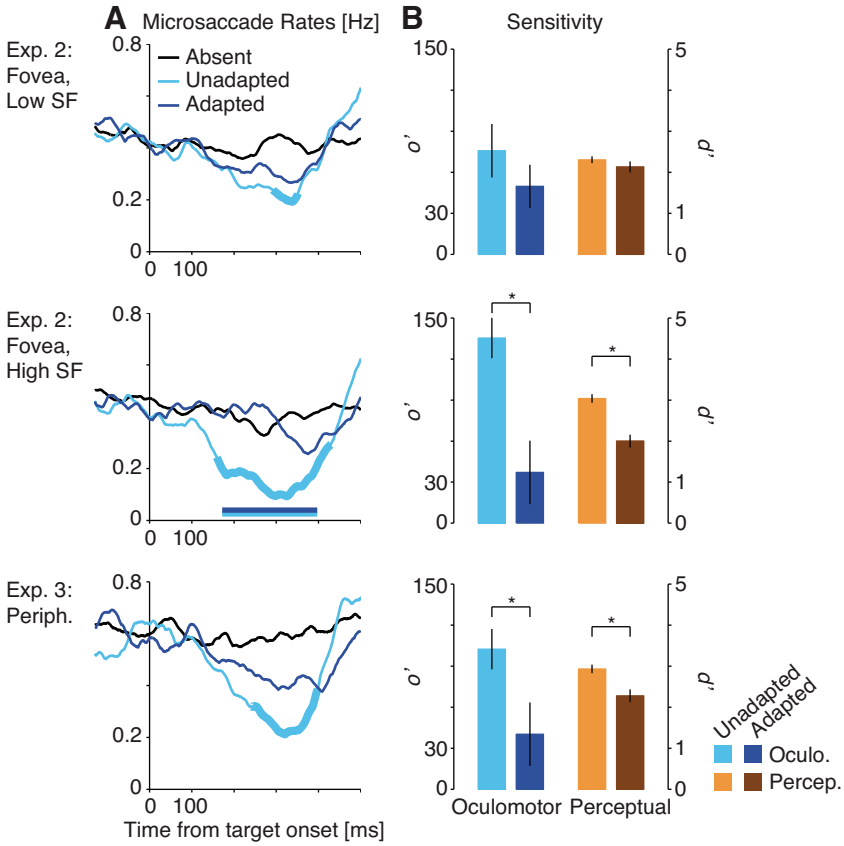

Fig. 4. Orientation-specific adaptation of microsaccadic inhibition. *A*: mean microsaccade rates in *experiments 2* and *3*. The thicker lines indicate significant differences from the target-absent condition (black line); horizontal blue bars indicate significant differences between the rates for targets orthogonal ("unadapted"; light blue) and parallel ("adapted"; dark blue) to the adaptor. *B*: mean oculomotor (o'; blue) and perceptual (d'; orange) sensitivity. *Significant pairwise differences. Error bars are ±1 within-subject SEs.

("adapted") caused weaker microsaccadic inhibition than orthogonal ("unadapted") targets. Only in the unadapted condition did the rates dip significantly below the baseline rate at any individual time point after the target onset.

Foveal oculomotor sensitivity (o'; Fig. 4*B*) was reduced for adapted orientations. A 2 × 2 repeated-measures ANOVA with factors of orientation (adapted or unadapted) and SF (high or low) demonstrated an effect of orientation [$F(1,11) = 19.6$, $P = 0.001$], which interacted with SF [$F(1,11) = 8.35$, $P = 0.01$]. The adaptation effect was significant when SF was high [$t(11) = 4.76$, $P < 0.001$; Fig. 4, *top*] but not when SF was low [$t(11) = 1.56$, $P = 0.15$; Fig. 4, *middle*]. In the periphery as well, o' was lower for the adapted than unadapted orientation [$t(11) = 3.77$, $P = 0.003$; Fig. 4, *bottom*].

Perceptual d' consistently mirrored the oculomotor sensitivity (Fig. 4*B*, orange bars). A 2 × 2 repeated-measures ANOVA on d' in the fovea showed that it was affected by orientation [$F(1,11) = 43.8$, $P < 0.0001$] and the interaction with SF [$F(1,11) = 24.0$, $P < 0.001$]. That is, d' was lower for adapted than unadapted targets, but only for the high foveal SF [$t(11) = 6.95$, $P < 0.0001$] and not for the foveal low SF [$t(11) = 1.72$, $P = 0.11$]. In the periphery, perceptual sensitivity was also lower for the adapted orientation [$t(11) = 2.32$, $P = 0.04$].

These orientation-specific adaptation effects suggest that for both foveal and peripheral stimuli, the signals that trigger microsaccadic inhibition are first processed by the same sensory units that lead to explicit perceptual detection, before reaching oculomotor control centers (including subcortical ones). Those sensory units are most likely in the visual cortex, where neurons are tuned for orientation, adapt selectively to stimuli like ours, and correlate strongly with perception (Fang et al. 2005; Kohn 2007; Ress and Heeger 2003). Orientation-specific adaptation has not been found in subcortical neurons (Solomon et al. 2004), and neurons in the superior colliculus have little if any orientation selectivity (White and Munoz 2012, but see Tailby et al. 2012). Orientation tuning is weak in the human lateral geniculate nucleus (Ling et al. 2015) and primate retinal ganglion cells (Passaglia et al. 2002; Schall et al. 1986) and most apparent for orientations that are cardinal or radial to the fovea, which our stimuli were not. Therefore, the strong adaptation effects we found in both perceptual and oculomotor sensitivity were almost certainly of cortical origin.

Contingence on Perceptual Reports

The effects of stimulus contrast and orientation adaptation on sensitivity (d' and o') demonstrate that explicit perceptual reports and oculomotor freezing rely on similar (if not the same) sensory detection mechanisms. To look more closely at the relation between perceptual and oculomotor responses, we examined whether they covary at the level of individual trials. To do so, we split the oculomotor data by whether the observer reported seeing a target or not. In *experiment 1*, target stimuli (collapsed across all contrast levels) inhibited microsaccades only in "hit" trials when the observer correctly reported target presence (Fig. 5*A*, *top left*). On incorrect "miss" trials (red line), the microsaccade rate was indistinguishable from the baseline rate (black line) from correct trials with no stimulus [at all individual time points, bootstrapped $P > 0.25$; collapsing across 500 ms poststimulus, $t(11) = 1.07$, $P = 0.31$]. The rate on hit trials was significantly lower in almost the entire half-second interval after the stimulus onset. This strong co-

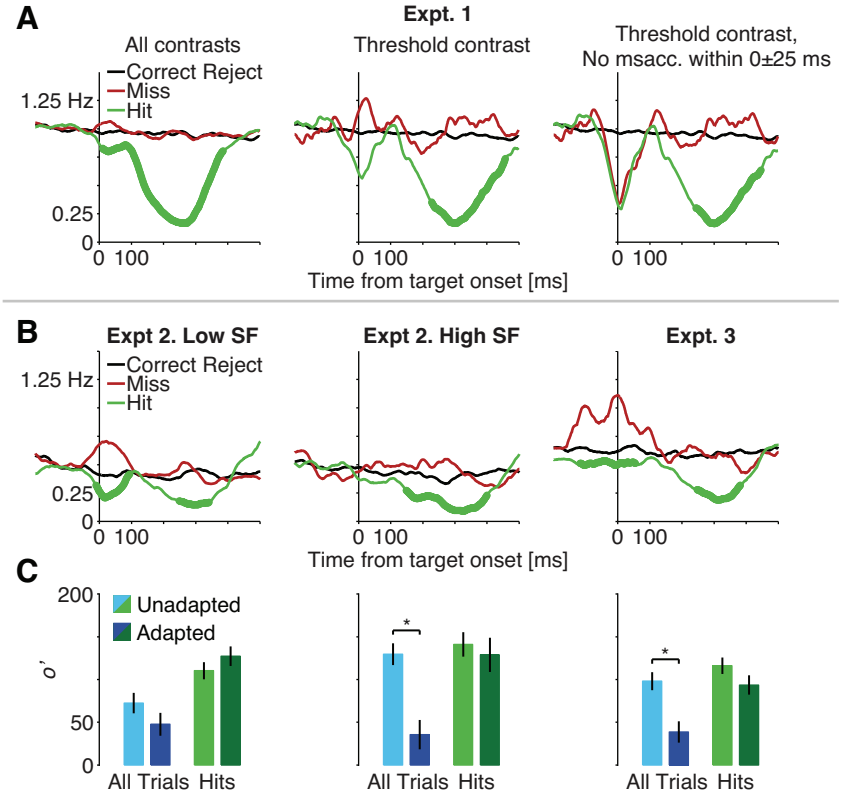

Fig. 5. Effect of perceptual report on target-present trials. *A*: mean microsaccade rates in target-present trials of *experiment 1*, contingent on whether the observer reported stimulus presence ("hit"; green) or absence ("miss"; red). The baseline rate from target-absent trials with correct responses is in black. The thicker lines indicate when the hit and miss curves differ significantly ($P < 0.05$ from bootstrapping). *Left*: all contrast levels. *Middle*: only trials with the single target contrast level nearest each individual observer's perceptual detection threshold. *Right*: the same data with threshold contrasts but excluding all trials with any microsaccade within 25 ms before or after the stimulus onset at *time 0*. *B*: same analysis of *experiments 2* and *3*. For a trial to count as a hit, the observer must have also correctly reported the target's orientation (and, in *experiment 3*, location). To count as a miss, the observer must have reported stimulus absence. This analysis collapses over adapted versus unadapted orientations, which showed similar patterns individually. *C*: mean o' from *experiments 2* and *3*. All trials together are plotted in blue and hit trials only in green. *Significant pairwise difference between unadapted (light bars) and adapted (dark bars) orientations. Error bars are ±1 within-subject SEs.

variation suggests that explicit perception and microsaccadic inhibition are triggered by the same detection mechanism.

The difference in rates was not due to the fact that missed stimuli tended to have lower contrasts, because the same pattern emerged even for the single contrast level nearest to each observer's detection threshold (Fig. 5*A*, *middle*). Again, the poststimulus drop in microsaccade rate occurred only on hit trials (green line), being significantly lower than baseline from 191 to 471 ms posttarget (corrected $P < 0.05$). The rate on miss trials never differed from baseline [all P values > 0.3; collapsing over 500 ms posttarget, $t(11) = 1.46$, $P = 0.17$]. Hit and miss trials differed significantly from each other between 228 and 458 ms (corrected $P < 0.05$; Fig. 5*B*, thicker line).

We also compared microsaccadic inhibition for the threshold-level contrast and maximum contrast used for each observer. Overall, there was much stronger inhibition for the maximum contrast [mean oculomotor sensitivity $o' = 143.7$ vs. 56.8, $t(11) = 6.47$, $P < 0.0001$]. That difference was largely reduced, however, when we only included hit trials for both contrast levels [$o' = 140.3$ vs. 121.2, $t(11) = 1.8$, $P = 0.095$]. This result suggests that microsaccadic inhibition is an all-or-none response that occurs whenever a stimulus is detected, irrespective of its contrast. The marginal remaining difference could indicate that some of the physical intensity difference is carried through into the strength of oculomotor inhibition or that some of the hits at threshold contrast were lucky guesses without a clear percept and without oculomotor inhibition.

Note also that around the time of target onset, the microsaccade rate was higher on miss trials than on hit trials (Fig. 5*A*, *middle*). This is because perceptual sensitivity is strongly reduced during saccades and microsaccades (Martinez-Conde et al. 2013; Rolfs 2009; Zuber et al. 1964), so missed stimuli were more likely to be accompanied by a microsaccade at roughly the same time. In principle, this contingency could cause differences between hit and miss trials in the microsaccade rates at later time points, because microsaccades may have a natural rhythm (Bosman et al. 2009; Gaarder et al. 1966; Hafed and Ignashchenkova 2013). However, we found the same difference in microsaccade rates between hit and miss trials at threshold contrast even when excluding all trials with any microsaccade in the 50-ms window centered on the time of target onset (Fig. 5*A*, *right*). Both rates dropped around *time 0*, due to the exclusion of trials with microsaccades in that window. But only on hit trials did the rate drop below baseline beginning ~100 ms later, whereas the rate on miss trials hovered around baseline.

In *experiments 2* and *3*, we found very similar differences between hit and miss trials, with oculomotor freezing only apparent on hit trials (Fig. 5*B*). The effect of saccadic suppression was also apparent for low frequencies in *experiment 2* and in *experiment 3*: around *time 0*, a peak in the microsaccade rate on miss trials and drop on hit trials. Again, however, the lack of inhibition on miss trials was preserved when we removed trials with microsaccades in the 50-ms window around the target onset (not shown).

Analysis of o' revealed that the effect of orientation adaptation on the oculomotor response was attenuated when only hit trials were considered (Fig. 5*C*, green bars). On hit trials, there was no reliable difference in oculomotor sensitivity for orientations parallel (dark green bars) and orthogonal (light green bars) to the adaptor [$t(11) = 1.37$, 0.52, and 1.59, $P = 0.20$, 0.61, 0.14, for the foveal low-SF, foveal high-SF, and peripheral conditions, respectively]. Therefore, although ob-

servers were less likely to see a target with the adapted orientation, when they did see it, their oculomotor systems responded to it as strongly as to the unadapted orientation. This is similar to the attenuation of the effect of contrast in *experiment 1* when only hit trials were included, again suggestive that oculomotor freezing is an all-or-none response that occurs when a stimulus is detected.

Critically, the dependence of microsaccade rates on perceptual reports appears to generalize to trials in which no stimulus was present. Observers occasionally reported seeing a target that was not there (FAR: ~6% in *experiments 1* and *2* and ~7% in *experiment 3*). On these trials, microsaccades were inhibited around the time the target would have appeared. Figure 6*A* shows this effect on microsaccade rates in *experiment 1*. The overall microsaccade rate in the last 500 ms of false alarm trials (green point) was significantly lower than on trials with correct rejections [black circle; $t(11) = 2.67$, $P = 0.022$; bootstrapped $P = 0.04$]. This difference remained significant even when controlling for the fact that there were far fewer false alarm trials than correct reject trials: over 1,000 repetitions, we drew a random subsample of correct reject trials equal in number to the false alarm trials and recomputed overall microsaccade rates in the last 500 ms of each trial. Averaging across participants, the 95% confidence interval on the distribution of rate differences did not include 0 (0.04, 0.24).

The display was physically identical (blank) on correct reject and false alarm trials. Therefore, the difference in microsaccade rate must be due to processes internal to the observer. However, we also compared microsaccade rates on miss trials (plotted in red in Fig. 6*A*) and false alarm trials (plotted in green in Fig. 6*A*). If oculomotor freezing is determined by stimulus presence, then microsaccade rates should be lower on miss trials (when there was a stimulus present). Alternatively, if oculomotor freezing covaries more strongly with explicit perception than the physical stimulus, then microsaccade rates should be lower on false alarm trials (when there was no stimulus present but the observer reported seeing one). Consistent with the latter hypothesis, the overall microsaccade rate was significantly lower on false alarm trials than on miss trials [$t(11) = 2.9$, $P = 0.015$]. Therefore, the eyes were more likely to freeze when the observer hallucinated a stimulus than when there really was a stimulus that the observer didn't see.

Note that the reduction in microsaccade rate on false alarm trials appears more diffuse than on target-present trials. Observers presumably based their decision to report "present" on a sensory signal registered roughly one half-second before the beep at the trial's end, because that is when the target always appeared on present trials. However, we do not know the exact time of each internal event that caused a false alarm and cannot lock our analysis to it, which could explain why the resulting pattern of microsaccadic inhibition is blurred in time.

We found the same pattern in all three experiments (Fig. 6*B*). Microsaccade rates were lower in false alarm trials than in correct reject trials for 10 of 12 observers in *experiment 2* [$t(11) = 3.0$, $P = 0.01$, bootstrapped $P = 0.01$] and 9 of 12 observers in *experiment 3* [$t(11) = 1.3$, $P = 0.22$, bootstrapped $P = 0.27$]. Because the display in all target-absent conditions contained only the fixation mark, we collapsed these data across all three experiments. On average, there were 22.3 ± 7.1% fewer microsaccades in the last 500 ms of false alarm trials than correct rejection trials [$t(32) = 3.2$, $P = 0.003$, bootstrapped $P = 0.01$]. Again, this across-experiment difference remained significant when subsampling from the correct reject trials to be equal in number to the false alarm trials [95% confidence interval: (0.051, 0.172)]. The same applies to the difference between miss trials and false alarm trials across all three experiments [95% confidence interval: (0.069, 0.189)].

The magnitude of the difference between microsaccade rates on false alarm and correct reject trials depended on the observer's perceptual false alarm rate (Spearman's $\rho = -0.58$, $P < 0.001$; Fig. 6*B*). The amount of inhibition of false alarm trials also correlated with the observers' decision criterion, expressed as the distance in d' units from the neutral point of $d'/2$ ($\rho = 0.36$, $P = 0.039$). Observers tended to be quite conservative (mean decision criterion in each experiment = 0.85 ± 0.07, 0.64 ± 0.04, and 0.48 ± 0.09). However, the most conservative observers (with

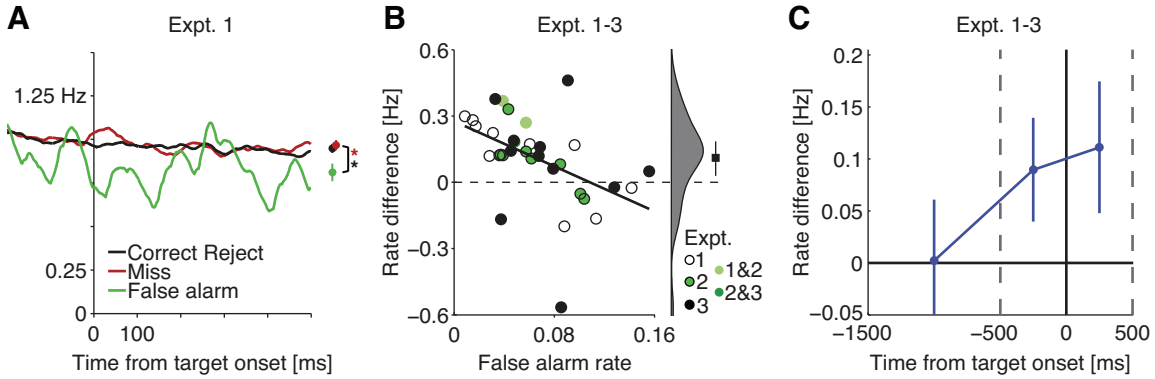

Fig. 6. Effect of perceptual report on target-absent trials. *A*: mean microsaccade rates in *experiment 1* divided by perceptual report in target absent trials (correct reject in black vs. false alarm in green) and miss trials in red. The rightmost points are the overall microsaccade rates in the 500 ms after the target onset, ±1 SE, which differ significantly between correct reject and false alarm trials and between miss and false alarm trials (*$P < 0.05$). *B*: summary of target-absent trials in all three experiments, plotting magnitude of inhibition [difference in microsaccade rate (in Hz) in the last 500 ms of correct rejection vs. false alarm trials] as a function of the perceptual false alarm rate. Each point is an individual observer. Three observers participated in two experiments, and we averaged their microsaccade rates across experiments, weighted by the number of trials. The black line is the best linear fit. On the *right*, oriented vertically, is the probability density function of inhibition magnitudes, with the mean (black square) and its bootstrapped 95% confidence interval. *C*: mean difference in microsaccade rates between correct reject and false alarm trials in three time windows. Data were aggregated across experiments in the same way as in *B*, taking into account the three observers who did two experiments. Error bars are 95% confidence intervals.

higher criteria and lower FARs) had the strongest microsaccadic inhibition when they did make a false alarm. One interpretation is that conservative observers with high criteria and low FARs reported "present" only when they had a strong percept that caused them to go against their bias to report "absent." That percept, being truly "visual," caused microsaccadic inhibition whether there is a stimulus physically present or not. In contrast, less conservative subjects may have occasionally false alarmed when they were less confident that they actually saw a target. In those false alarm trials, microsaccadic inhibition would be less likely, and so overall the difference compared with correct reject trials is diluted.

There could be other explanations for the observed differences between false alarm and correct reject trials. Some other factor that varies across trials, such as the overall level of task vigilance, could both make false alarms more likely and microsaccades less likely overall. However, the relative inhibition of microsaccades on false alarm trials (compared with correct reject trials) was specific to end of the trial, when the target would have appeared, despite the fact that trial duration was unpredictable. Figures 6C shows the difference in overall microsaccade rates between correct reject and false alarm trials in three time windows: −1,500 to −500 ms, −500 to 0 ms, and 0 to 500 ms. (We did not examine any earlier time bins because there were few trials with >1,500 ms of fixation before the target onset, due to the exponential distribution of this interval.) The rate difference was statistically significant only in the later two time bins [both $t(32) > 3.0$, $P < 0.01$] and was greater in the last time bin than the earliest [$t(32) = 2.32$, $P = 0.027$]. Therefore, any factor that explains the relative inhibition of microsaccade rates on false alarm trials could not be general in time to entire trials. This makes more likely the explanation that spurious internal visual signals soon before the trial's end can cause both oculomotor freezing and erroneous perceptual reports of target presence.

In summary, these analyses revealed that microsaccades are inhibited almost immediately after the observer detects a stimulus, regardless of whether a stimulus was presented or not. When the observer does not detect a stimulus (i.e., on trials when the observer reports stimulus absence), microsaccades continue at the usual rate. Oculomotor freezing and explicit perceptual detection therefore share a source of noise in their responses to luminance contrast.

Single Trial Classification Analysis

The patterns observed in the mean microsaccade rates suggest that the timing of microsaccades on individual trials may contain information as to whether a stimulus was present and, perhaps more reliably, whether the observer consciously perceived a stimulus. We tested this idea by dividing the data into independent training and test sets (see MATERIALS AND METHODS). We used training trials to estimate the prior probabilities of stimulus/report presence and the prior probabilities of microsaccades at each time point given stimulus/report presence or absence. On each test trial, we then used Bayes' rule to compute the posterior probability that a stimulus was present or that the observer reported stimulus presence, given the observed microsaccades. For instance, a microsaccade that occurs 250 ms after the time of potential stimulus onset would provide strong evidence that no stimulus was present or perceived, because microsaccadic inhibition on present trials is strongest around 250 ms. We computed classification accuracy using the criterion-free measure A', the area under the ROC curve. For a similar analysis applied to single neuron firing rates to classify perception on individual trials, see Quiroga et al. (2008).

In *experiment 1* (Fig. 7*A*), the mean A' for classifying stimulus presence, regardless of contrast, was 0.55 ± 0.008, significantly above chance [$t(11) = 6.73$, $P < 0.0001$]. The corresponding A' for classifying the perceptual report was 0.60 ± 0.01 significantly higher than for the stimulus [$t(11) = 5.19$, $P < 0.001$]. In these analyses, overall accuracy was limited by the fact that microsaccades are relatively rare, so even on target-absent trials there may be no microsaccades at all. Accordingly, we also estimated classification accuracy using only test trials that contained at least one microsaccade in the final 500 ms, yielding A' of 0.58 ± 0.01 for stimulus presence/absence and 0.68 ± 0.01 for the perceptual report [stimulus vs. report: $t(11) = 7.43$, $P < 0.0001$]. We also tested stimulus classification accuracy for each contrast level individually, using independent training sets with a mix of all contrast levels. A' increased with contrast, consistent with stronger microsaccadic inhibition at higher contrast. We did not classify the report as a function of contrast, because at extreme contrasts reports were nearly 100% present or 100% absent. However, we could classify perceptual reports at the contrast level nearest each observer's detection threshold: A' was significantly above chance both across all trials [0.59 ± 0.014, $t(11) = 6.19$, $P = 0.0001$] and when we included only trials with at least one microsaccade [0.61 ± 0.027, $t(11) = 4.12$, $P = 0.002$].

In *experiment 2* (Fig. 7*B*), the mean A' for stimulus presence (regardless of orientation) was 0.53 ± 0.007 and above chance [$t(11) = 4.3$, $P = 0.001$]. A' for the report was significantly

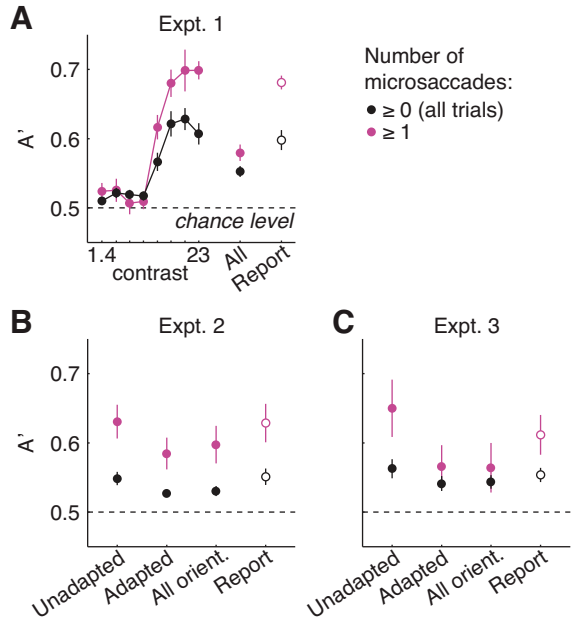

Fig. 7. Stimulus and report presence on individual trials can be predicted based on microsaccades in *experiment 1* (*A*), *experiment 2* (*B*), and *experiment 3* (*C*). Pink circles are accuracy only for trials with at least one microsaccade; black circles are for all trials together. Filled symbols are for classifying the stimulus presence/absence; open symbols are for classifying the observer's perceptual report (present/absent). A', area under the ROC curve. Error bars are ±1 SE.

higher [0.55 ± 0.01, $t(11) = 3.51$, $P = 0.005$]. Again, accuracy was higher for the subset of test trials containing at least one microsaccade both for stimulus (0.60 ± 0.027) and report classification [0.63 ± 0.028; comparison with stimulus classification: $t(11) = 2.17$, $P = 0.05$]. The effect of orientation adaptation was also visible in stimulus classification accuracy, being higher for the unadapted orientation [using all test trials: 0.55 vs. 0.53, $t(11) = 2.96$, $P = 0.013$].

For the peripheral stimuli in *experiment 3* (Fig. 7*C*), the mean A' for stimulus presence (regardless of orientation) was 0.54 ± 0.01 [higher than chance: $t(11) = 4.41$, $P = 0.001$], which was marginally lower than A' for the report [0.55 ± 0.01, $t(11) = 1.89$, $P = 0.08$]. For test trials containing at least one microsaccade, the difference in accuracy between stimulus classification ($A' = 0.56 \pm 0.036$) and report classification ($A' = 0.61 \pm 0.029$) increased but did not reach significance [$t(11) = 1.30$, $P = 0.22$]. As in *experiment 2*, A' for stimulus presence was higher for the unadapted than adapted orientation [using all test trials: 0.55 vs. 0.51, $t(11) = 2.34$, $P = 0.04$].

These results demonstrate that the timing of microsaccades contains information about whether or not a new stimulus had appeared within one-half second prior. Although overall classification accuracy is far from perfect, microsaccades contain even more information about whether the observer will report that a stimulus was present, suggesting that oculomotor freezing is causally related to conscious perception rather than to retinal stimulation. Classification of the stimulus is less reliable because it mixes together trials in which a stimulus was and was not perceived.

These results held up even if we used no prior information about a particular observer's microsaccade rates and based classification solely on average data from the rest of the observers. For classifying the stimulus using all trials, A' was significantly above chance in *experiment 1* [0.54 ± 0.010, $t(11) = 4.4$, $P = 0.001$] and in *experiment 3* [0.523 ± 0.008, $t(11) = 2.75$, $P = 0.019$] but not in *experiment 2* [0.51 ± 0.009, $t(11) = 1.57$, $P = 0.14$]. When only test trials with microsaccades were used, those A' values were somewhat higher (0.57 ± 0.012, 0.56 ± 0.013, and 0.552 ± 0.019, respectively) and above chance also in *experiment 2* [$t(11) = 4.19$, $P = 0.0015$]. For classifying the report, A' was above chance and higher than stimulus classification in all experiments, using all test trials [*experiment 1*: 0.58 ± 0.018, $t(11) = 3.75$, $P = 0.003$; *experiment 2*: 0.53 ± 0.01, $t(11) = 2.77$, $P = 0.018$; and *experiment 3*: 0.54 ± 0.009, $t(11) = 3.34$, $P = 0.007$]. Using only test trials with microsaccades, report classification accuracy was higher still (*experiment 1*: 0.65 ± 0.018, *experiment 2*: 0.61 ± 0.018, and *experiment 3*: 0.55 ± 0.017). Accuracy in this analysis tended to be lower than when training with each individual observer's data, but that difference was only reliable in *experiment 1* [stimulus A': 0.54 vs. 0.55, $t(11) = 2.38$, $P = 0.037$; report A': 0.58 vs. 0.60, $t(11) = 2.57$, $P = 0.026$]. Thus, even in the absence of knowledge of one observer's microsaccade rates, we were able to infer above chance whether a particular stimulus was present and explicitly perceived.

DISCUSSION

We found that the spontaneous microsaccade rate drops soon after the onset of a stimulus if and only if the observer explicitly perceived the stimulus. Microsaccades are also inhibited even when no stimulus was presented but the observer makes a false alarm. Therefore, oculomotor freezing is not determined directly by the physical characteristics of the triggering stimulus or even the presence of a stimulus. Rather, it indexes the internal registration of a new sensory event that can be voluntarily reported. We infer that the visual cortex is involved in triggering the oculomotor freezing response because the response depends on whether the orientation of the stimulus had previously been adapted. This conclusion contradicts the view that oculomotor freezing likely relies on specialized subcortical circuits (Engbert 2006; Reingold and Stampe 2002; Ro et al. 2004; Rolfs et al. 2008; Sumner et al. 2006).

Our data demonstrate covariation between two distinct behavioral responses to a brief visual stimulus: oculomotor inhibition (which begins involuntarily roughly 100 ms after the stimulus onset) and explicit perceptual reports, produced voluntarily at the end of the trial. To interpret this covariation, we need to first interpret the perceptual reports. What do they indicate about the perceptual or cognitive state of the observer on particular trials? One interpretation is that the perceptual reports index conscious perceptual awareness of the target stimulus. That interpretation rests on the assumption that when the observer has a conscious experience of a target Gabor patch, he or she reports "stimulus present," and when the observer has no such experience, he or she reports "stimulus absent." If we accept that assumption, we conclude that involuntary oculomotor inhibition reveals visual awareness. This conclusion is based on our analyses of hit versus miss trials (and false alarm versus correct reject trials) and our single trial classification performance. In other words: the eyes briefly stop making saccades when a new visual stimulus reaches conscious awareness.

This assumption that yes/no detection reports index conscious detection may not always be warranted (Dehaene and Changeux 2011; Reingold and Merikle 1988; Seth et al. 2008). For instance, the phenomenon of "blindsight" demonstrates that "objective" stimulus discrimination performance can be dissociated from subjective visual awareness. Therefore, it is possible that some correct reports of stimulus presence had no corresponding conscious experience. Other measures of visual awareness (such as subjective visibility ratings or postdecision wagering) may be more robust (Dehaene and Changeux 2011; Sandberg et al. 2010; Seth et al. 2008). Future experiments should explore their relation to oculomotor freezing, for instance by examining how oculomotor freezing relates to variable decision criteria or finely grained stimulus visibility ratings.

However, in our experiment, in which healthy subjects were asked to honestly report whether or not they saw a simple stimulus, there is no definite reason to suppose that their true conscious experiences deviate systematically from what they report. In such conditions, the various objective and subjective measures of stimulus detection usually agree well (Dehaene and Changeux 2011; Peters and Lau 2015). Indeed, there is some evidence that in normal observers there is no dissociation between discrimination performance and subjective awareness of a stimulus (Peters and Lau 2015). Moreover, in *experiment 1*, observers did not discriminate any stimulus attribute but merely reported its presence or absence. Therefore, responses

in our task are more similar to "subjective" ratings of visibility than the "objective" discriminations that can be dissociated from consciousness under conditions of blindsight (Lau and Passingham 2006; Weiskrantz 1986). The fact that false alarm rates remained very low (5–7%) despite trial-by-trial error feedback also suggests that observers remained conservative and only reported "present" when they were confident in their conscious experience of a stimulus.

Therefore, in the discussion that follows we assume that the observer's explicit perceptual reports index their conscious percepts on individual trials. Due to the caveats described above, the reader could replace the terms "conscious perception" and "visual awareness" with "explicit perception." The latter concept is limited to perception that can be voluntarily reported in our detection task and is not definitely (but probably) a sign of visual awareness.

Remarkably, we found that microsaccadic inhibition begins to reveal conscious (explicit) detection roughly 100 ms after the stimulus onset, reaching a maximum at ~200 ms (for high luminance contrast). This allows enough time for a sensory signal to be routed to oculomotor control centers via the visual cortex, but just barely, given that response latencies in macaque V1 are roughly 60 ms (Schmolesky et al. 1998). Perceptual reports are produced after several hundred more milliseconds of cognitive and motor processing. Nonetheless, our data suggest that whatever processes determine awareness of a stimulus must be finished in significantly less than 100 ms.

Two pieces of evidence suggest that oculomotor freezing is an all-or-none response that occurs whenever a new stimulus is consciously detected. Overall, stimulus parameters appear to have a graded effect on the degree to which the microsaccade rate drops after the stimulus onset. We saw this as a function of contrast in *experiment 1* (Fig. 3), as have others (Scholes et al. 2015). However, we found that the effect of contrast is nearly eliminated when we included only trials in which the observer correctly reported stimulus presence (hit trials). Similarly, in our adaptation experiments, stimuli with adapted orientations caused overall less microsaccadic inhibition than unadapted orientations, but that effect was also eliminated when only hit trials were included (Fig. 5). Therefore, physically weak or adapted stimuli are detected relatively infrequently, but when they are detected they cause strong oculomotor freezing. Similarly, some researchers have posited that conscious detection is an all-or-none phenomenon (Dehaene and Changeux 2011; Dehaene et al. 2003; Quiroga et al. 2008). We argue that across-trial aggregate measures of perceptual sensitivity (d') and the oculomotor response show similar effects of stimulus parameters (such as contrast, as shown in our *experiment 1* and by Bonneh et al. 2015 and Scholes at al. 2015) precisely because of the trial-by-trial covariation in all-or-none explicit perception and all-or-none oculomotor freezing, both of which are less likely for low contrast (or adapted) stimuli.

Based on the present data, we cannot determine whether conscious perceptual detection per se causes microsaccadic inhibition or vice versa. They may have a common cause: one detection mechanism triggers both the perceptual report "present" and the inhibition of eye movements. The resulting trial-by-trial covariation allowed us to decode the observer's subjective percept based on the timing of microsaccades in single trials. Although that classification accuracy was not particularly high (maximum ~70% correct), it provides evidence that such decoding is possible in principle. Importantly, decoding accuracy for the subjective report was higher than for classifying physical stimulus presence, within the same trials and constant stimulus intensity. In comparison, Scholes et al. (2015) trained a support vector machine to classify trials as stimulus present versus absent with up to 95% accuracy. However, they classified groups of trials, not individual trials, and the data were downsampled into coarse temporal chunks.

By classifying individual trials, we proved that microsaccade events within one half-second contain reliable information about explicit (conscious) perception. The technique could be improved, for instance, in conditions when microsaccades occur more frequently. Our novel Bayesian approach, with its transparent mathematical relation to the raw microsaccade time series, could also be translated to other applications (such as inferring perceptual states from neuronal spike trains). At the moment, however, aggregating microsaccade rates over many trials and computing oculomotor d' provides a better implicit diagnostic for how well particular stimuli can be perceived.

Other lines of research have investigated the relation between stimulus-driven eye movements and explicit perception (Kowler 2011; Spering and Montagnini 2011). Curious dissociations can occur, suggestive of independent sensory pathways (Spering and Carrasco 2015). However, perception-action correlations have proven equally informative. For instance, optokinetic nystagmus (Fox et al. 1975) and smooth pursuit (Braun et al. 2006; Madelain and Krauzlis 2003) reveal the perceived direction of motion in ambiguous or illusory displays. Our findings complement these continuous oculomotor indicators of perceived motion by providing an implicit measure of the conscious registration of stimulus onset. We chose to study responses to luminance contrast because it is the most fundamental aspect of visual input and seemed likely to affect eye movements via sensory channels independent of perception. In fact, we found the opposite.

In contrast to many other studies, we did not demonstrate a sustained effect of a cognitive state on microsaccade rates (Cui et al. 2009; Poletti et al. 2013; Steinman et al. 1967; Valsecchi et al. 2007) or an influence of microsaccades on perceptual performance (Deubel and Eisner 1986; Martinez-Conde et al. 2006). Rather, we showed that trial-by-trial variations in perception are linked (within 1/10 of a second after the stimulus onset) to these involuntary eye movements.

Our analysis of contrast thresholds in *experiment 1* confirms the results of two recent studies. Bonneh et al. (2015) found a correlation between perceptual contrast sensitivity and the latencies of microsaccades in particular time bins. Scholes et al. (2015) found that contrast thresholds for stimulus-induced microsaccade rate changes could predict contrast thresholds for explicit perceptual sensitivity. Both of those studies measured microsaccades during passive viewing and therefore could not examine trial-by-trial correlations between perceptual and oculomotor responses. Our data reveal that link, and our adaptation experiments suggest that processes in the visual cortex mediate oculomotor freezing.

Finally, another study used pupil dilation to investigate motor correlates of perceptual processes during a simple detection task (de Gee et al. 2014). They found that the pupil dilates more on trials when the observer reports "present" than "absent," especially for conservative observers. These results mirror ours, but with a sluggish motor response during pro-

longed decision formation rather than an immediate sensory reflex. By manipulating stimulus contrast and by adapting particular orientations, we were able to show that in addition to its correlation with detection criteria, oculomotor freezing tracks stimulus visibility.

The superior colliculus is causally involved in generating microsaccades (Hafed et al. 2009). One model for oculomotor freezing is that a sensory signal arriving in the superior colliculus suppresses ongoing activity or raises the threshold for saccade execution (Hafed and Ignashchenkova 2013; Rolfs et al. 2008). The direct retinotectal pathway to the superior colliculus (Marrocco and Li 1977; Perry and Cowey 1984) has previously been a tempting explanation for oculomotor freezing, given that the inhibition occurs rapidly and in response to irrelevant stimuli (Engbert 2006; Reingold and Stampe 2002; Ro et al. 2004; Rolfs et al. 2008; Sumner et al. 2006). One study found that saccadic response times to a peripheral target were slowed by a foveal distractor, even when transcranial magnetic stimulation applied to the occipital cortex rendered the distractor invisible (Ro et al. 2004). The authors concluded that the retinotectal pathway triggers oculomotor freezing independently of the visual cortex and conscious perception. Our results are inconsistent with that conclusion, although we studied spontaneous microsaccades rather than voluntary saccades.

Previous studies have used purely chromatic stimuli to argue that the retinotectal pathway, being insensitive to chromatic contrast (Marrocco and Li 1977; Perry and Cowey 1984), is not necessary for oculomotor freezing (Bompas and Sumner 2009; Rolfs et al. 2008; Sumner et al. 2006; Valsecchi and Turatto 2007). Our study goes an important step further by demonstrating that the retinotectal pathway is not sufficient for oculomotor freezing, even for luminance contrast. If it were, microsaccadic inhibition would have remained strong for adapted orientations and for targets that were missed.

Note that this conclusion does not preclude the involvement of subcortical processes in the oculomotor freezing response. To the contrary: we propose that the stimulus signal is first transmitted to the cortex via the geniculostriate pathway and is then relayed to subcortical oculomotor control centers. The signal from the cortex could be forwarded to the superior colliculus or directly to the latest stages of oculomotor control in the brain stem. For instance, it could inhibit microsaccades by activating "omnipause" neurons that normally cease firing during (micro)saccades (Brien et al. 2009; Otero-Millan et al. 2011) and thus prevent the activation of burst neurons that could otherwise trigger these movements (Van Gisbergen et al. 1981).

Our results imply potential confounds in studies of the neural correlates of consciousness (Tononi and Koch 2008). We have shown that explicit stimulus detection is selectively accompanied by a rapid decrease and then rebound in microsaccade rate. Microsaccades cause robust responses in the human visual cortex, and differences in microsaccade rates between conditions can have far-reaching consequences for interpretations of neural activity (Dimigen et al. 2009; Tse et al. 2010; Yuval-Greenberg et al. 2008). Therefore, correlations between a pattern of neural activity (e.g., the power of oscillations in some frequency band) and conscious detection could be contaminated by neural activity produced by microsaccades.

On the positive side, future research can take advantage of the perceptual information contained in microsaccades. Requiring an observer to make explicit judgments about a stimulus may introduce response biases, change the state of attention, and elicit complex brain activity patterns (Tsuchiya et al. 2015). Using oculomotor freezing as an implicit behavioral index of conscious detection would circumvent these complications. That would be especially useful when the stimuli ought to be unexpected or unattended. Oculomotor freezing would also enable measures of perception in nonverbal subjects, such as nonhuman primates, human infants, or those with incapacitating medical conditions. In fact, one patent has been filed to use fluctuations in microsaccade rate and direction to infer stimulus detection and the state of attention (Otero-Millan et al. 2014). Our data show that such an approach could be practically useful, although with some limitations.

We conclude that the reflexive freezing of saccadic eye movements is contingent on the observer consciously perceiving a stimulus. Therefore, perceptual awareness (or, more conservatively, explicit perception) shares rapid detection mechanisms with an obligatory, reflexive motor response. This link results in opponent behavior: the eyes move when nothing is seen and freeze when a new percept emerges.

ACKNOWLEDGMENTS

The authors are grateful to Niko Busch, Patrick Cavanagh, William Harrison, Sven Ohl, and Miriam Spering for comments on the manuscript.

GRANTS

This work was supported by Deutsche Forschungsgemeinschaft Grant RO 3579/2-1 and National Eye Institute Grant T32-EY-007031.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

A.L.W. and M.R. conception and design of research; A.L.W. performed experiments; A.L.W. analyzed data; A.L.W. and M.R. interpreted results of experiments; A.L.W. and M.R. prepared figures; A.L.W. drafted manuscript; A.L.W. and M.R. edited and revised manuscript; A.L.W. and M.R. approved final version of manuscript.

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