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RESEARCH ARTICLE | *The Role of Eye Movements in Perception, Cognition, and Action*

Dynamics of fixational eye position and microsaccades during spatial cueing: the case of express microsaccades

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Tian X, Yoshida M, Hafed ZM. Dynamics of fixational eye position and microsaccades during spatial cueing: the case of express microsaccades. *J Neurophysiol* 119: 1962–1980, 2018. First published February 21, 2018; doi:10.1152/jn.00752.2017.—Microsaccades are systematically modulated by peripheral spatial cues, and these eye movements have been implicated in perceptual and motor performance changes in cueing tasks. However, an additional oculomotor factor that may also influence performance in these tasks, fixational eye position itself, has been largely neglected so far. Using precise eye tracking and real-time retinal-image stabilization, we carefully analyzed fixational eye position dynamics and related them to microsaccade generation during spatial cueing. As expected, during baseline fixation, microsaccades corrected for a foveal motor error away from the preferred retinal locus of fixation (the so-called ocular position "set point" of the oculomotor system). However, we found that this relationship was violated during a short period immediately after cue onset; a subset of cue-directed "express microsaccades" that were highly precise in time and direction, and that were larger than regular microsaccades, occurred. These movements, having <100-ms latencies from cue onset, were triggered when fixational eye position was already at the oculomotor set point when the cue appeared; they were thus error-increasing rather than error-decreasing. Critically, even when no microsaccades occurred, fixational eye position itself was systematically deviated toward the cue, again with ~100-ms latency, suggesting that the oculomotor system establishes a new set point at different postcue times. This new set point later switched to being away from the cue after ~200–300 ms. Because eye position alters the location of retinal images, our results suggest that both eye position and microsaccades can be associated with performance changes in spatial cueing tasks.

NEW & NOTEWORTHY Covert spatial cueing tasks are a workhorse for studying cognitive processing in humans and monkeys, but gaze is not perfectly stable during these tasks. We found that minute fixational eye position changes, independent of the more studied microsaccades, are not random in cueing tasks and are thus not "averaged out" in analyses. These changes can additionally dictate microsaccade times. Thus, in addition to microsaccadic influences, retinal image changes associated with fixational eye position are relevant for performance in cueing tasks.

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express microsaccades; eye position; fixational eye movements; microsaccades; spatial cueing

INTRODUCTION

Microsaccades interrupt periods of stable gaze position that drifts slowly and with small amplitude (Hafed et al. 2015; Krauzlis et al. 2017). Even though microsaccades have historically been believed to be random, it is now recognized that they are part of a deliberate oculomotor strategy to optimize gaze position at the fixated target (Guerrasio et al. 2010; Hafed 2011; Ko et al. 2010), and this optimization happens even in the face of peripheral stimulus onsets that might normally attract large, foveating eye movements (Tian et al. 2016). A highly studied example of such peripheral onsets in relation to microsaccades is that of spatial cueing (Engbert 2012; Engbert and Kliegl 2003; Hafed et al. 2011, 2013; Hafed and Clark 2002; Hafed and Ignashchenkova 2013; Meyberg et al. 2017; Peel et al. 2016; Rolfs et al. 2008; Wang et al. 2017; White and Rolfs 2016). In such cueing, a workhorse of cognitive studies of covert visual attention, cue onset causes robust and systematic modulations in microsaccade direction and frequency, and it is now clear that these modulations are not simply a probabilistic, or "dirty," readout of attentional state, but instead disruptions of an ongoing and precise oculomotor optimization process of fixational eye position (Engbert 2012; Hafed et al. 2015; Hafed and Ignashchenkova 2013; Tian et al. 2016).

The above-mentioned link between microsaccades and spatial cueing has garnered much attention, not only because it was recognized that microsaccades, an easily measurable biological parameter, could act as an "overt" measure of internal brain state (Engbert and Kliegl 2003; Hafed and Clark 2002) but also because neurophysiological studies have since revealed similarities between the mechanisms for generating microsaccades and larger saccades (Hafed 2011; Hafed et al. 2009; Hafed and Krauzlis 2012; Krauzlis et al. 2017). This has opened the intriguing possibility that individual microsaccades are associated with similar perimovement changes in visual

performance as large saccades. Indeed, correlates of saccadic suppression, compression, distortions of time perception, and alterations in peak velocity-to-amplitude relationships have all been reported for microsaccades (Bellet et al. 2017; Chen et al. 2015; Chen and Hafed 2017; Hafed 2013; Hafed et al. 2015; Hafed and Krauzlis 2010; Peel et al. 2016; Tian et al. 2016; Yu et al. 2017). This development has meant that it was now possible to consider as a distinct possibility that the mere occurrence of microsaccades in cueing tasks can cause performance changes qualitatively and quantitatively similar to those observed by the cognitive processes being probed by the tasks themselves (Bellet et al. 2017; Chen et al. 2015; Hafed 2013; Hafed et al. 2015; Tian et al. 2016).

However, a thus far neglected factor in studies of the links between spatial cueing and microsaccades has been the influence of fixational eye position per se. The implications of microsaccades in cueing tasks on fixational eye position dynamics are not explored even though microsaccades alter gaze position; conversely, the conditions of fixational gaze position that may or may not increase microsaccade likelihood in cueing tasks are unknown. This gap in our understanding exists because most modern studies of microsaccades have relied on video-based eye trackers, making it hard to reach reliable inferences about the role of fixational eye position dynamics. In this study, we used spatially and temporally precise scleral search coils combined with real-time retinal image stabilization (Chen and Hafed 2013; Tian et al. 2016) to investigate exactly these questions.

We uncovered a highly systematic relationship between instantaneous foveal eye position error (a direct consequence of instantaneous fixational eye position) and microsaccade occurrence in cueing tasks, and we discovered a new phenomenon of "express microsaccades" that critically depends on such a relationship. More importantly, we additionally found that cue onset causes reliable drifts in eye position to new foveal "set points" of the oculomotor system toward which microsaccades are directed. Instantaneous fixational eye position after cue onset is thus not a random variable. Instead, in addition to microsaccadic influences on performance alluded to above, retinal image position changes associated with foveal eye position itself may be relevant for performance in spatial cueing tasks.

MATERIALS AND METHODS

We performed behavioral experiments on three male rhesus macaque monkeys (*Macaca mulatta*; aged 6–11 yr and weighing 9–13 kg). These experiments were approved by the regional governmental offices of the city of Tuebingen. Laboratory setup was similar to recent descriptions (Chen and Hafed 2013; Hafed and Ignashchenkova 2013; Tian et al. 2016), and we measured eye movements with high temporal and spatial precision using the scleral search coil technique (Fuchs and Robinson 1966; Judge et al. 1980).

Behavioral Tasks

Experiment 1: spatial cueing task. The task was almost identical to that we recently described for the human experiments in (Tian et al. 2016), but it was now performed by monkeys. Briefly, the monkeys fixated a white, central fixation spot presented over a gray background (Chen and Hafed 2013; Tian et al. 2016). After a random delay of 500–1,000 ms, a white, 1° diameter circle appeared briefly (for ~35 ms) at 5° eccentricity along one of the four cardinal directions from the fixation spot (i.e., right, left, up, or down). After a random delay (~8–1500 ms), an identical white circle appeared either at the previously cued location ("same" condition) or the opposite location ("opposite" condition). In accordance with classic convention, we termed the first stimulus the cue and the second stimulus the target, and we defined the cue-to-target onset asynchrony (CTOA) as the time difference between their respective onsets (Fig. 1*A*). The fixation spot was extinguished at the same time as target onset, instructing the monkeys to generate a foveating saccade to the target. We measured saccadic reaction time (RT) to the target. We analyzed 8,195 trials from *monkey P*, 6,990 trials from *monkey N*, and 4,083 trials from *monkey F* in this task.

Experiment 2: spatial cueing task with real-time control of foveal motor error during initial gaze fixation. In *monkeys P* and *N*, we ran a second experiment comparing a control condition similar to that of *experiment 1* to a retinal image stabilization condition. The latter condition introduced a subtle change from the control condition, in the sense that we presented the peripheral stimulus when the retinal position of the fixation spot's image was experimentally controlled. The detailed procedures of this experiment are as follows. The control condition (see Fig. 11*A*, *left*) was identical to that described in the monkey experiments of Tian et al. (2016). In fact, we used the same control data from that study in the present study, and we compared them with data from randomly interleaved retinal image stabilization trials that were collected but not described or analyzed in the original study, and that constitute the novel contribution of the present study. Briefly, the control condition involved the monkeys fixating a small fixation spot. After some delay, a peripheral stimulus appeared at 5° eccentricity and remained on until fixation spot removal. The latter event instructed the monkeys to foveate the peripheral stimulus. In such a control condition, the stimuli were fixed on the display, as is classic in most cueing experiments, but due to fixational eye movements, they were variable in retinal coordinates. We compared these control trials with randomly interleaved retinal image stabilization trials. In the retinal image stabilization trials (see Fig. 11*A*, *right*,), after initial fixation, we stabilized the fixation spot on the retina by moving it with gaze position in real time for 100–550 ms before peripheral stimulus onset. When the peripheral stimulus appeared, the fixation spot position was frozen on the display, and the rest of the trial was identical to the control condition. In other words, we experimentally controlled and minimized foveal eye position error at the time of peripheral stimulus onset, with all other variables unaltered, and the peripheral stimulus was always presented relative to the instantaneous eye position at the end of the fixation interval containing retinal image stabilization. Thus, if predictions based on the results from *experiment 1* hold (e.g., see Figs. 1, 2, 5, and 7), then the brief period of retinal image stabilization, which allowed for experimental control over foveal eye position error at the time of peripheral stimulus onset, should alter microsaccade statistics. We carefully calibrated eye position and ensured maintenance of real-time (i.e., timely) stimulus updates as described earlier (Chen and Hafed 2013; Tian et al. 2016). Across both monkeys, we analyzed a total 13,973 control trials and compared them with 5,123 retinal image stabilization trials.

Data Analysis

Microsaccades and saccades were detected offline using velocity and acceleration criteria described recently (Chen and Hafed 2013), and microsaccade misdetections were checked manually for all trials. We defined as microsaccades all saccades occurring during stable fixation, and most movements were less than 30 min arc in amplitude. For example, median microsaccade amplitude was 12.9, 8.7, and 12.9 min arc in *monkeys P*, N, and *F*, respectively, in *experiment 1*.

Experiment 1: spatial cueing task. A primary observation in our study was the occurrence of so-called "express microsaccades" (see

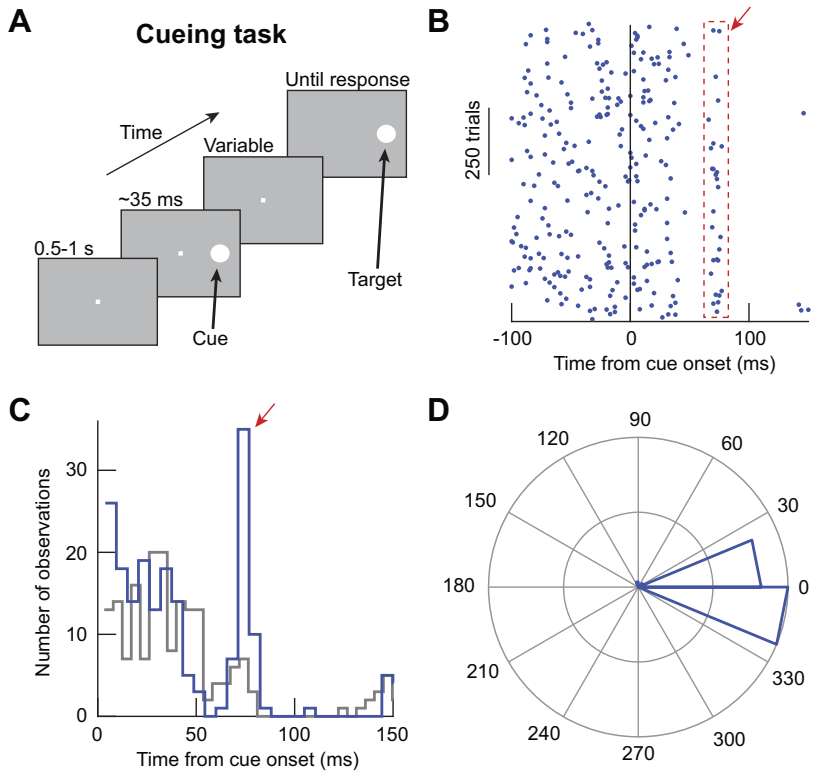

Fig. 1. Express stimulus-induced microsaccades. *A*: monkeys fixated a central spot, and a white circle appeared at 5° eccentricity for ~35 ms to the right or left of, or above or below, fixation. After a random cue-to-target onset asynchrony (CTOA), a similar white circle appeared either at the previously cued location or at the diametrically opposite one, and the monkeys generated a saccade to it. *B*: each row of dots is a trial from a sample monkey (*monkey P*) with a sample cue location (upward), and each dot indicates the onset time of a microsaccade. Shortly after cue onset, microsaccade frequency abruptly decreased to zero, as expected. However, there was a population of subsequent "express" movements triggered with latencies from cue onset of <100 ms (highlighted by red arrow and dashed rectangle). *C*: same data as in *B* but presented as a frequency histogram (blue) demonstrating the distinct population of movements with express latencies (red arrow) shortly after the onset of microsaccadic inhibition. For comparison, the gray histogram shows similar analyses for another cue location (downward) from the same monkey. Even though the express movements were fewer, they still occurred and shared properties with those observed for the upward cue (see Fig. 2). *D*: direction histogram of the express movements (with latencies of 60–100 ms from cue onset) shown in blue in *B* and *C*. We plotted the difference in direction between a given microsaccade and the direction of the cue relative to the fixation spot such that a value of 0 indicates perfect alignment between microsaccades and the cue. The directions of express microsaccades were highly aligned with cue location. Direction distributions for other cue locations and other monkeys are shown in Fig. 2.

RESULTS). We defined as express microsaccades movements with latencies of 60–100 ms after cue onset. We analyzed their onset times, directions, radial amplitudes, and peak velocities using standard techniques (Buonocore et al. 2017; Hafed et al. 2011, 2013; Hafed and Ignashchenkova 2013; Peel et al. 2016; Tian et al. 2016). For histograms of onset times, we used bin widths of 20 ms, and when we normalized histograms, we normalized by the total number of observations in a given analysis. To compare express microsaccade properties to properties of "regular" microsaccades, we defined the latter movements as those movements occurring during steady-state fixation of the central fixation spot during the interval 0–500 ms before cue onset (i.e., with no other stimulus on the display). We often referred to this interval as the "baseline" interval.

A second primary observation in our study was related to the instantaneous position of the eye at the time of microsaccade or cue onset. During steady-state baseline fixation before cue onset (0–300 ms before cue onset), we took all such intervals in which no microsaccades occurred and in which no express microsaccades were triggered after cue onset, and we used those as the preferred retinal locus of fixation (Nachmias 1959). Microsaccades during baseline steady-state fixation are expected to correct foveal errors that caused the eye to be relatively far away from this preferred locus (Guerrasio et al. 2010; Ko et al. 2010; Nachmias 1959; Tian et al. 2016), and we also confirmed this in our results (e.g., see Fig. 5*A*). We then explored the properties of eye position relative to this preferred retinal locus of fixation under a variety of conditions, such as the onset of an express microsaccade. We often referred to the preferred retinal locus of fixation as the oculomotor "balance point" or as the eye position set point, borrowing from control engineering terminology.

We also analyzed the time course of eye position set point variations at different times after cue onset. We picked all intervals in which there were no microsaccades occurring from −50 to 150 ms relative to cue onset (i.e., within a 200-ms interval), and we plotted eye position to explore any potential systematic eye position drifts immediately after cue onset. For later times after cue onset, we also picked successive 200-ms intervals not containing any microsaccades, and we plotted average eye position in the middle of these intervals to estimate eye position set points at different times after the cue. We moved these successive intervals in steps of 50 ms to map a time course of eye position set points. Of course, this means that for times longer than 100 ms after cue onset, there could have been a microsaccade (or more) occurring before our microsaccade-free 200-ms intervals in which we sampled eye position for this analysis. Therefore, in this particular analysis, the eye position set point could reflect potential contributions of previous microsaccades in addition to slow changes in gaze position (see RESULTS).

In one set of analyses, we also related the occurrence of express microsaccades to saccadic RT to the target (e.g., see Fig. 3). In this case, we obtained a measure of "cueing effect", which is the difference in RT to the target between trials in which the target was opposite the cued location and trials in which it was in the same cued location (Klein 2000; Lupiáñez et al. 2006; Posner 1980; Posner and Cohen 1984; Posner et al. 1985; Tian et al. 2016). A negative cueing effect indicates "inhibition of return," or the fact that RT to the target is faster for opposite, rather than same, target locations (Klein 2000; Lupiáñez et al. 2006; Posner 1980; Posner and Cohen 1984; Posner et al. 1985; Tian et al. 2016). We compared the cueing effect when trials contained express microsaccades immediately after cue onset with the cueing effect when trials did not contain any such express microsaccades. For this analysis, we only compared cueing effects for horizontal cue locations. The reason is that with vertical cues, same and opposite target locations have opposite vertical saccade directions (e.g., upward vs. downward saccades). Because it is known that there are strong asymmetries in vertical saccade RTs (Hafed and Chen 2016; Schlykowa et al. 1996; Zhou and King 2002), these asymmetries significantly complicate interpreting whether cueing effects were positive or negative for a given vertical cue location.

Finally, we theoretically explored what would happen if eye position during baseline fixation was solely determined by the cumulative effect of successive microsaccades, with no influence of eye position drift/control in between. We first selected, in each monkey, "baseline" microsaccades occurring within 500 ms before cue onset and having amplitudes <120 min arc. We then classified these microsaccades as

being directed toward one of the four visual quadrants (up/right, up/left, down/left, and down/right), and we calculated both the likelihood and amplitude of microsaccades going toward each quadrant. Each monkey exhibited some biases in microsaccade direction and amplitude during baseline fixation (e.g., see Fig. 8*A*), and we wanted to simulate trials in which the same biases existed. For each quadrant, we estimated the mean and variance of microsaccade amplitude directed toward that quadrant, and we also estimated the mean and variance of the likelihood that a microsaccade was directed toward a given quadrant. Similarly, we measured intermicrosaccadic intervals, and we fit the obtained distribution with a gamma function given the skewed nature of intermicrosaccadic interval distributions (e.g., see Fig. 8*A*). We then used each monkey's estimates of microsaccade direction bias (to one of the quadrants), amplitudes, and intermicrosaccadic intervals to create simulated eye position trials. In each such trial, starting at eye position "zero," a microsaccade could occur randomly, but in one of four directions (45°, 135°, 225°, or 315°, representing the 4 quadrants). The likelihood of which quadrant was the direction of the given microsaccade was drawn randomly from normal distributions with mean and variance matched to those estimated from the monkey's real data. Similarly, the amplitude of the microsaccade was picked randomly from a normal distribution having the same mean and variance as the amplitudes of the monkey for a given quadrant. After the microsaccade was generated in the simulated trial, an intermicrosaccadic interval was then picked randomly from the fitted gamma distribution of intermicrosaccadic intervals in the monkey, and the process was repeated successively to generate subsequent microsaccades. We simulated 2 s of fixation, and we repeated this process for 1,000 simulated trials in each monkey. In every simulated trial, we measured the final eye position, which was the cumulative sum of all consecutive microsaccades that had occurred in the simulated trial, and we compared the variance of simulated eye position with the variance in real data (e.g., see Fig. 8). To check whether square-wave jerks, or pairs of opposing microsaccades (Hafed and Clark 2002), could alter our simulation results, we repeated the above exercise, but only after forcing microsaccades to come in pairs of opposing eye movements with no direction biases (but maintaining the biases in amplitude and intermicrosaccadic intervals in the real data). This violated the biases in microsaccade directions present in the real data, but it tested the extreme case that square waves can recenter gaze. We also simulated a hybrid scenario in which the second movement in a square wave could sometimes not occur in the opposite direction, according to the direction biases present in the real data.

Experiment 2: spatial cueing task with real-time control of foveal motor error during initial gaze fixation. We performed the same time course analyses of microsaccade direction as those described in Tian et al. (2016). Briefly, we separated microsaccades as being either toward the peripheral stimulus, opposite it, or neither (orthogonal to the cue direction). We analyzed the time courses of toward and opposite microsaccades because they were the most modulated by stimulus onset and because orthogonal microsaccades were both infrequent and unmodulated by cue location. We also performed similar analyses, but now on microsaccade amplitude instead of direction (e.g., see Fig. 11*C* in RESULTS). We applied the same criteria for classifying microsaccades as being toward or opposite the peripheral stimulus location in our amplitude analyses.

Statistical analyses in both experiments included descriptive statistics along with measures of SE or 95% confidence and 95% prediction intervals (for linear regressions). We typically compared either regular microsaccades and express microsaccades or trials with express microsaccades and trials without express microsaccades, and the results of statistical tests for such comparisons are detailed in RESULTS and/or the figure legends.

RESULTS

Express Stimulus-Induced Microsaccades

We ran our monkeys on a spatial cueing task (*experiment 1*; Fig. 1*A*), similar to the one that we used on humans recently (Tian et al. 2016). Monkeys fixated on a small white spot, and a white circle (cue) appeared briefly at 5° eccentricity in one of the four cardinal directions. It was previously shown that cue onset in this task robustly modulates microsaccade frequency, resulting in an abrupt decrease in microsaccade probability immediately after stimulus onset (Engbert and Kliegl 2003; Hafed et al. 2011, 2013; Hafed and Clark 2002; Hafed and Ignashchenkova 2013; Meyberg et al. 2017; Peel et al. 2016; Rolfs et al. 2008; Tian et al. 2016; Wang et al. 2017; White and Rolfs 2016). We also observed such microsaccadic inhibition, but closer inspection of the data revealed a distinct population of microsaccades that were triggered within a narrow time window of ~60–100 ms after cue onset, and shortly after the onset of the microsaccadic inhibition phase. For example, in Fig. 1*B*, each dot represents the onset time of a microsaccade relative to cue onset (in this case, for the upward cues) in one of our monkeys (*monkey P*), with trials from the same monkey and cue location stacked as rows. Microsaccadic inhibition started at ~50 ms after cue onset, and it was followed on some trials (53/1311; 4.02%) with a population of eye movements reminiscent of "express saccades" that can be observed in larger visually guided saccade tasks (Boch et al. 1984; Carpenter 1988; Fischer and Boch 1983; Fischer and Ramsperger 1984). That is, these movements, highlighted in Fig. 1, formed a distinct population of movements from the microsaccades occurring in the preinhibition phase, and they had very short latencies relative to stimulus onset. These observations can be better appreciated with the same data plotted as a frequency histogram of microsaccade latencies from cue onset (Fig. 1*C*): there was a steady rate of microsaccade occurrence early after cue onset, followed by the onset of an inhibition phase, and then followed once again by a distinct peak of microsaccades with "express" latencies. Importantly, these microsaccades were also highly congruent in direction with the location of the cue. Specifically, Fig. 1*D* plots the distribution of angular differences between cue location and these microsaccades' directions (i.e., for the same movements highlighted in Fig. 1, *B* and *C*), and it shows that these movements had directions that were almost entirely within ±30° from the direction of the cue (the average directional difference between the microsaccades and cue direction was 2.14 ± 2.04°, mean ±SE, and it was not significantly different from 0; $P = 0.299$, *t*-test, $n = 53$ microsaccades). Because these movements were clearly triggered by cue onset in both time (Fig. 1*C*) and direction (Fig. 1*D*), and because they had very short latencies reminiscent of those associated with larger express saccades (Boch et al. 1984; Carpenter 1988; Fischer and Boch 1983; Fischer and Ramsperger 1984), we refer to these movements in this article as "express microsaccades."

Across all three monkeys, express microsaccades shared the above properties of tight temporal and directional correlations with cue location, and they were also greater than approximately four times larger in amplitude than normal microsaccades. In the same monkey as in Fig. 1, we plotted in Fig. 2*A* microsaccade frequency after cue onset for another sample cue location (this time, the rightward one); even though express

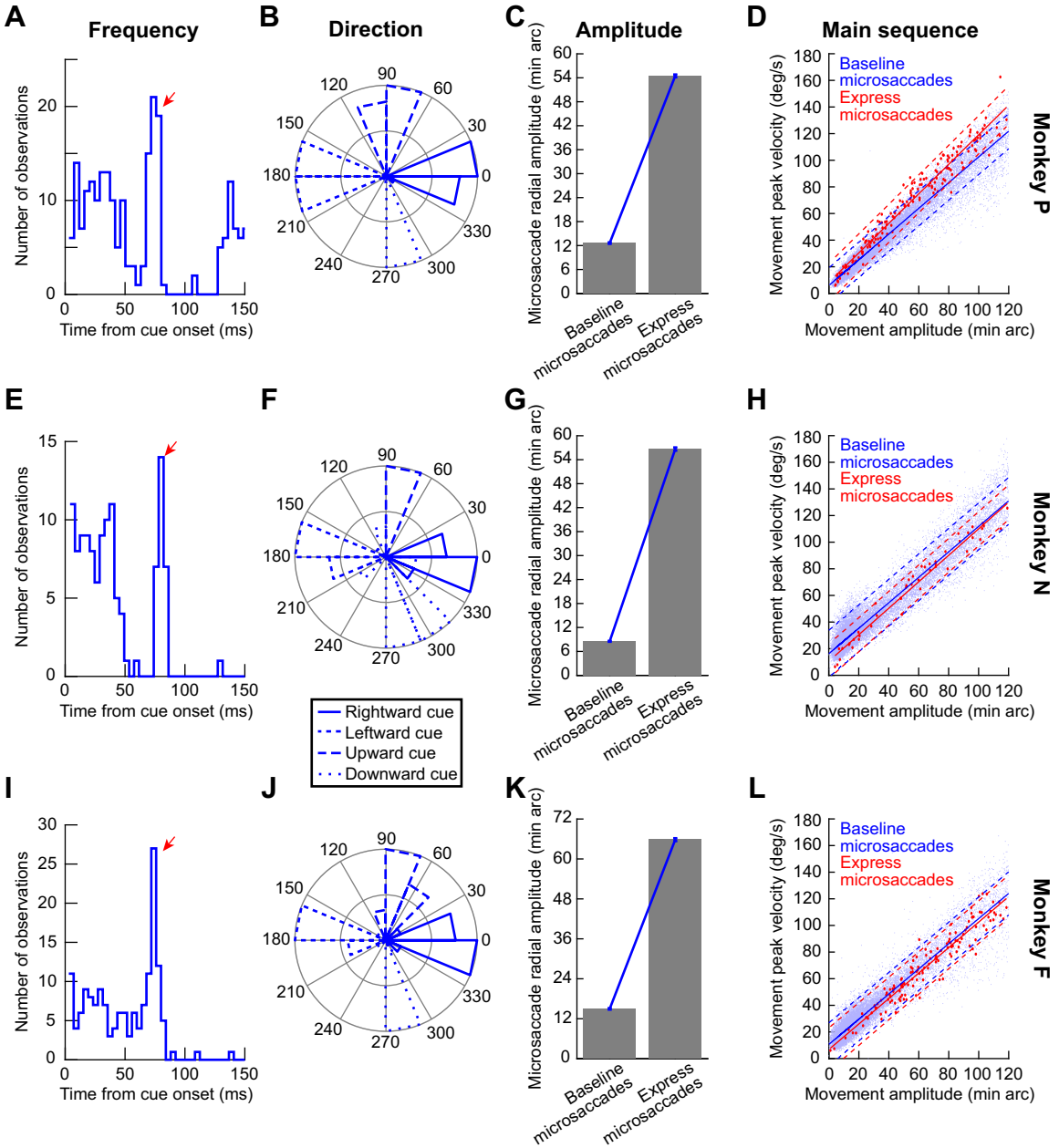

Fig. 2. Express microsaccades were precise in time and direction, and they were much larger than normal microsaccades while still showing normal saccadic kinematics. *A*: microsaccade frequency histogram relative to cue onset from *monkey P*, as described in Fig. 1*C*. Here we show data from the rightward cue location instead of the upward cue location shown in Fig. 1, to illustrate another cue direction for which express microsaccades were reliably triggered within a narrow time window after cue onset (red arrow). *B*: direction histograms of express microsaccades (occurring 60–100 ms after cue onset) in *monkey P* for the different cue locations. Even though express microsaccades were less likely to occur for some cue locations (e.g., Fig. 1*C*, gray histogram), when they did occur, their directions were strongly aligned with cue location. Each histogram shows absolute express microsaccade directions for a given cue location. For example, express microsaccades were predominantly leftward for the leftward cue, and so on. *C*: for all cue locations in the same monkey, bar at *left* shows average (±SE) microsaccade amplitude during a baseline fixation interval before cue onset (0–500 ms before cue onset), and bar at *right* shows average (±SE) express microsaccade amplitude (for movements occurring 60–100 ms after cue onset). Express microsaccades were >4 times larger than the monkey's regular microsaccades. *D*: main sequence relationship between peak velocity and movement amplitude for baseline (blue) and express (red) microsaccades in *monkey P*. Solid and dashed lines indicate linear regression line and accompanying 95% prediction intervals, respectively. Express microsaccades did not lie below the regular main sequence curve, as might be expected from kinematic alterations in a task such as ours (Buonocore et al. 2017). The slight increase in peak velocity for express microsaccades in this monkey might reflect increased alertness for these movements (see Fig. 3). *E–H*: same as *A–D* but for *monkey N* (histogram in *E* is for leftward cues, which resulted in the highest likelihood of express microsaccades in this animal). *I–L*: same as *A–D* but for *monkey F*, with the histogram in *I* showing data for downward cues (again, because they resulted in the most express microsaccades).

microsaccades were less frequent than for the upward cue (more on this below), they still occurred. Moreover, express microsaccade directions were always congruent with the cue location. This is indicated by the microsaccade direction histograms shown in Fig. 2*B*, in which we summarized the directions of all microsaccades occurring 60–100 ms after cue onset for each of the four cue locations; rightward cues triggered rightward express microsaccades, leftward cues trig-

gered leftward express microsaccades, and so on for the upward and downward cues. Finally, Fig. 2*C* shows that express microsaccade amplitudes in this monkey, regardless of cue location, were more than four times larger than the amplitudes of regular microsaccades, which we defined as those movements occurring during baseline fixation before cue onset (see MATERIALS AND METHODS). All of these observations were replicated in the two other monkeys tested in this experiment (Fig. 2*E–G*, *I–K*). Thus, express microsaccades were stimulus-induced and exhibited significantly larger amplitudes than regular microsaccades.

We also checked whether express microsaccades were genuine saccadic movements. Specifically, models of microsaccadic rate inhibition after cue onset often relate this inhibition to countermanding effects, in which some microsaccades shortly after cue onset might "escape" the resetting effects of this onset (Hafed and Ignashchenkova 2013; Salinas and Stanford 2013; Tian et al. 2016). These escape eye movements often violate (Buonocore et al. 2017) the well-known saccadic main sequence relationship (Zuber et al. 1965) and therefore exhibit altered kinematics. However, the express microsaccades that we observed in the present experiment exhibited apparently normal main sequence relationships in each of the monkeys. Specifically, Fig. 2, *D*, *H*, and *L*, shows the population of baseline microsaccades occurring before cue onset, along with a linear regression line and 95% prediction intervals, as well as express microsaccades (across all cue locations), also with a linear regression line and 95% prediction intervals. In all monkeys, express microsaccades did not appear larger in amplitude than their corresponding peak velocities would indicate, as might be expected from known kinematic alterations associated with peripheral cueing (Buonocore et al. 2017). If anything, *monkey P* exhibited higher peak velocities for the express microsaccades, which could reflect increased alertness for these eye movements. Therefore, express microsaccades were genuine saccadic eye movements in terms of kinematics.

These eye movements were also functionally relevant. This fact became clear when we analyzed behavioral performance in the cueing task (i.e., RT to the target onset at the end of the trial) on trials with and without express microsaccades. In this task, different CTOAs are known to cause differential saccadic RT effects for targets in the same and opposite cued locations (Fig. 3*A*); thus a measure of cueing effect in the task (Fig. 3*B*) is the difference in RT between opposite and same trials (Klein

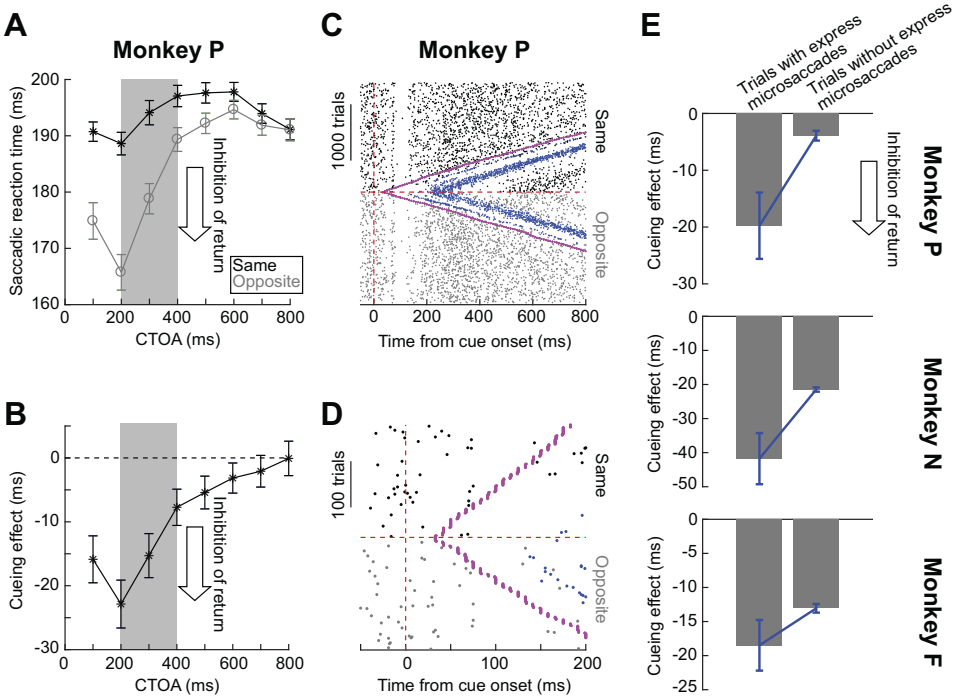

Fig. 3. Express microsaccades were associated with magnified cueing effects. *A*: saccadic reaction time (RT) as a function of cue-to-target onset asynchrony (CTOA) for an example monkey (*monkey P*). RT was faster for opposite than for same target locations, especially for CTOAs around ~200 ms, consistent with well-known inhibition of return. Note that with CTOA randomization as in our case, it is unlikely to observe short-CTOA RT benefits for same trials compared with opposite ones (Malevich et al. 2017), and this also depends on microsaccadic behavior (Tian et al. 2016). Thus the primary cueing effect to which we could relate express microsaccades in our data was inhibition of return. Error bars denote 95% confidence intervals, and the shaded rectangle defines an interval in which we explored the influence of express microsaccades on RT. *B*: cueing effect, defined as the RT difference between opposite and same trials (see MATERIALS AND METHODS), for the data in *A*. Error bars denote 95% confidence intervals. *C*: raster of microsaccade onset times as a function of CTOA for the same data in *A* and *B*. Each black or gray dot is a microsaccade onset, and each row is a trial. Magenta dots indicate target onset, and blue dots indicate response saccades. Trials were sorted by CTOA and target location relative to the cue (black means same); trials with no magenta dots had longer CTOA than shown in the figure. *D*: magnification of the short-CTOA subset of data from *C*, demonstrating how microsaccadic inhibition precludes analysis of relationships between express microsaccades and RT on very short CTOA trials. Moreover, for such trials, microsaccades are replaced by the real response saccades (Tian et al. 2016). Also note how RT was already shorter for opposite than same trials for early CTOAs (compare blue dots for same and opposite trials; Malevich et al. 2017). *E*: for all 3 monkeys, the cueing effect during the interval 200–400 ms after cue onset (shaded region in *A* and *B*) was magnified on trials with express microsaccades (error bars denote 95% confidence intervals). Note that for this analysis, we only considered horizontal cue and target locations. This is because vertical saccades have strong saccadic RT asymmetries (Hafed and Chen 2016; Schlykowa et al. 1996; Zhou and King 2002). Thus, for a given cue location, same and opposite saccades would necessarily be upward vs. downward, or vice versa, complicating any interpretation of cueing effects without RT asymmetry contamination.

2000; Lupiáñez et al. 2006; Malevich et al. 2018; Posner 1980; Posner and Cohen 1984; Posner et al. 1985; Tian et al. 2016). We found that the cueing effect was most negative ~200 ms after cue onset (Fig. 3*B*), indicating strong inhibition of return (Klein 2000; Lupiáñez et al. 2006; Posner 1980; Posner and Cohen 1984; Posner et al. 1985; Tian et al. 2016). Because express microsaccades were not very frequent in our experiment ($n = 97$ of 3,911 trials in the example data of Fig. 3*C*), and also because of microsaccadic inhibition reducing the number of microsaccades in very early CTOA trials (Fig. 3*D*), we could not measure the cueing effect for very short CTOAs with enough express microsaccade trials (microsaccades would be replaced with response saccades for short CTOAs; Tian et al. 2016). It was therefore difficult to relate the occurrence of express microsaccades to short-CTOA cueing effects. However, during well-known inhibition-of-return epochs (200–400 ms after cue onset; Fig. 3, *A* and *B*), we had sufficient trials with express microsaccades to compare cueing effects with and without these movements. In all three monkeys (Fig. 3*E*), trials with express microsaccades had significantly stronger cueing effects (in this case, inhibition of return) than trials without. This means that RTs on opposite trials got significantly faster when an express microsaccade was triggered earlier by the cue. We think that the effect of express microsaccade triggering lingered until 200–400 ms after cue onset because express microsaccades were almost always followed by an opposite movement (see Fig. 4) ~100 ms later. Thus, by the time of target onset in our analysis interval of Fig. 3*E*, the oculomotor system had already "flipped" toward the opposite location (Tian et al. 2016), and the target onset now appeared in the temporal vicinity of a directionally congruent microsaccade. This is a condition that is known to maximize microsaccadic influences on peripheral performance (Bellet et al. 2017; Chen et al. 2015; Tian et al. 2016). These observations of magnified cueing effects (Fig. 3*E*) therefore indicate that express microsaccades (Figs. 1 and 2) were not artifactual, but that they had significant functional relevance in the task when they did occur. Because of all of the above distinct properties of express microsaccades (Figs. 1–3), we next analyzed the reasons that such special eye movements might arise at all.

Time Since the Last Microsaccade and Instantaneous Fixational Eye Position Dictate the Occurrence of Express Microsaccades

Because express microsaccades did not occur on every single trial in our task, certain oculomotor factors must have existed for these special eye movements to be triggered. We hypothesized that one such factor was the time since the last microsaccade, especially because microsaccades are governed by temporal rhythmicity (Bellet et al. 2017; Hafed and Ignashchenkova 2013; Nachmias 1959; Tian et al. 2016), but we also discovered additionally that absolute fixational eye position at the time of cue onset was also critical.

In terms of time, we compared the temporal relationship between successive microsaccades during baseline fixation (i.e., before cue onset) with this relationship for express microsaccades. For each baseline microsaccade (i.e., occurring before cue onset; see MATERIALS AND METHODS), we plotted a frequency distribution of the times of all previous movements to the selected microsaccade and a similar frequency distribution of the times of all subsequent movements. The result, akin to a microsaccade-aligned autocorrelation function, revealed that during baseline fixation (i.e., before cue onset), microsaccade probability started to increase more than ~100 ms before or more than ~100 ms after the occurrence of any given movement, and this was true in all three monkeys. This is illustrated in Fig. 4, *A–C*, in which the raster plot above each histogram shows the times of individual microsaccades occurring either before (*left* histogram) or after (*right* histogram) a given movement, with all movements stacked on top of each other as rows. This expected behavior of microsaccades (Bosman et al. 2009; Hafed and Ignashchenkova 2013) was violated for express microsaccades. In each of the monkeys (Fig. 4, *D–F*), there was a noticeable scarcity of microsaccades occurring before any given express microsaccade, meaning that the latter movements were triggered when the cue appeared at a time in which no recent microsaccades had occurred for a substantial amount of time. Note that the analyses in Fig. 4, *D–F*, also revealed that express microsaccades were additionally followed by subsequent microsaccades with shorter average latencies than during baseline fixation before cue onset (compare the *right* histogram in each panel with the corresponding histogram above in Fig. 4, *A–C*). These additional subsequent movements occurred to correct for the large fixation error caused by express microsaccades, because these express movements could be as large as 1° in amplitude in all three monkeys (Fig. 2, *C*, *G*, and *K*), and they also had a significant impact on task performance (Fig. 3).

Thus, time since the last microsaccade was an important factor for whether the cue was effective in triggering an express microsaccade or not. However, there was another factor that we discovered, which was the instantaneous fixational eye position relative to the preferred retinal locus of fixation. To demonstrate the importance of this oculomotor factor, we related microsaccade direction to the absolute eye position that existed at microsaccade onset. For regular microsaccades occurring during baseline fixation before cue onset, we measured eye position while the monkeys fixated steadily without any microsaccades for at least 300 ms before cue onset and on trials without express microsaccades after cue onset (see MATERIALS AND METHODS). This position was deemed the current set point for the oculomotor system, and it was dictated by the preferred foveal retinal locus for fixation (Nachmias 1959). We then measured eye position for baseline microsaccades, which also occurred during baseline precue fixation, of different directions.

For example, in *monkey P*, for which express microsaccades were most likely for upward cues, we analyzed vertical eye position before and after cue onset (Fig. 5*A*). Before cue onset, upward microsaccades (Fig. 5*A*, *left*) were triggered when vertical eye position was spatially below the set point established without any microsaccades. That is, eye position in the 100-ms interval before microsaccade execution was significantly below (in spatial position) the baseline eye position without microsaccades ($P = 0$, rank sum test). Thus upward microsaccades acted to reduce foveal eye position error during baseline fixation, similar to our recent observations in Tian et al. (2016); for comparison, eye position for downward microsaccades in the same animal is also shown and again demonstrates the corrective nature of regular, precue microsaccades (i.e., eye position before microsaccade onset was spatially

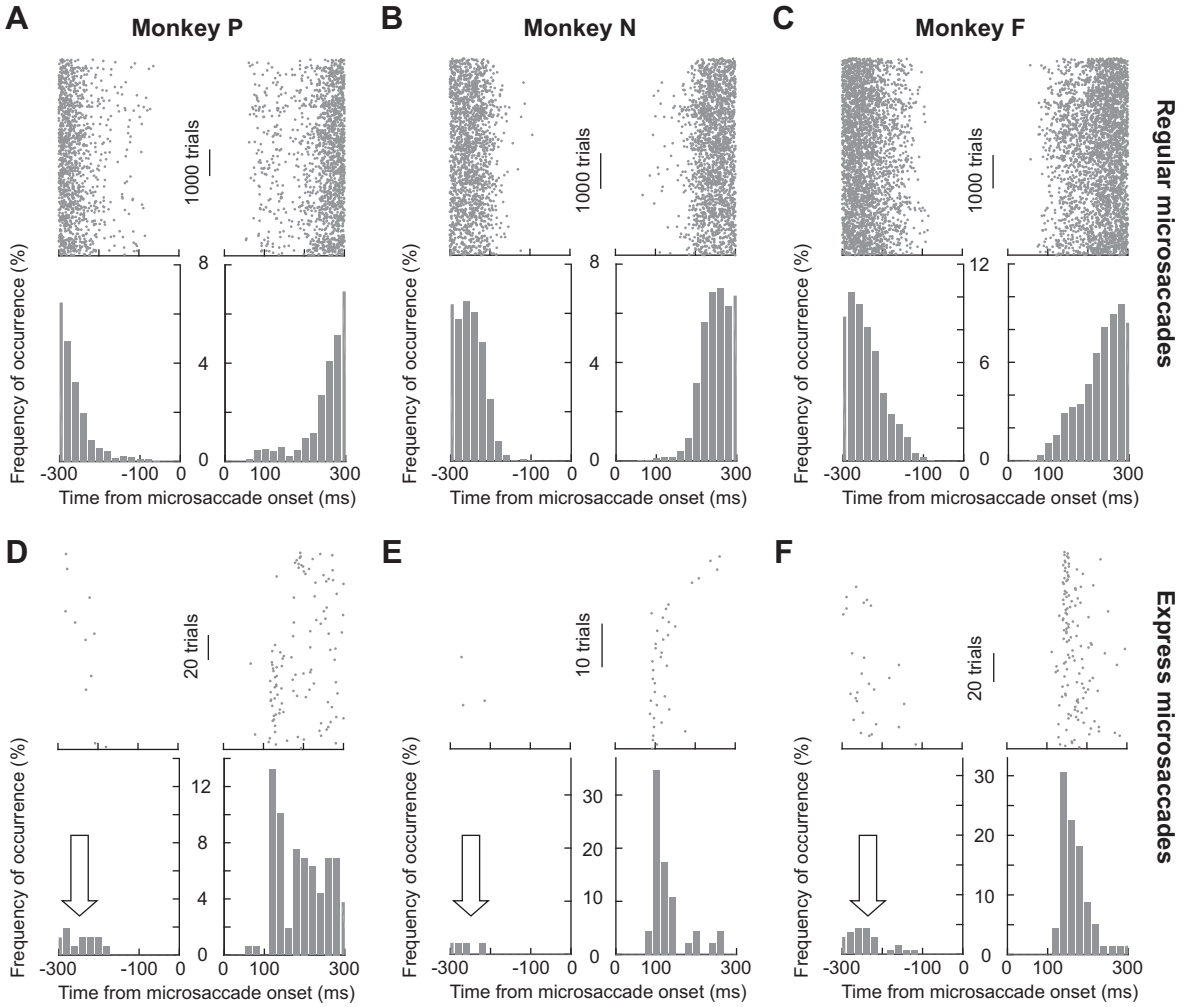

Fig. 4. Microsaccadic temporal structure influenced the likelihood of observing express microsaccades. *A–C*: for each monkey, we measured microsaccade probability either before (*left* histogram) or after (*right* histogram) the occurrence of a given microsaccade (akin to computing a microsaccadic autocorrelation function), and we did this for regular microsaccades occurring during a baseline fixation interval before cue onset (0–500 ms before cue onset). As expected, microsaccade probability increased more than ~100 ms before or more than ~100 ms after a given movement. Raw rasters above each histogram show microsaccade onset times across repetitions of this analysis. *D–F*: repeating the above analysis as in *A–C* but for express microsaccades (occurring 60–100 ms after cue onset for all cue directions) revealed that express microsaccades were most likely to occur if there was a particularly long interval of no microsaccades during fixation (see arrows and raster plots above each histogram). Note also that express microsaccades were often followed by a second population of low-latency microsaccades that were corrective back to the fixation spot given how big express microsaccades were (Fig. 2); these likely explain the magnified cueing effects shown in Fig. 3. Thus a particularly long fixation interval with no prior microsaccades is among the temporal conditions that can increase the likelihood of observing express microsaccades.

above baseline eye position without microsaccades; $P = 0$, rank sum test). However, after cue onset, when there was an upward express microsaccade (Fig. 5*A*, *right*), a very different relationship emerged; specifically, at the time of express microsaccade onset, there was minimal foveal eye position error from the baseline oculomotor set point. In other words, eye position in the 100-ms interval before express microsaccade execution was much closer to the baseline eye position without microsaccades than eye position in the 100-ms interval before regular microsaccades ($P = 5.1453 \times 10^{-10}$ for comparing the difference in eye position between regular microsaccades and baseline fixation to the difference in eye position between express microsaccades and baseline fixation, rank sum test).

Thus, when the cue appeared and the eye was almost perfectly balanced at its optimal set point, the cue was particularly effective in triggering a large, cue-directed express microsaccade. This property did not necessarily happen for all cue-directed microsaccades that are known to occur early after cue onset (i.e., at the onset of the microsaccade rate inhibition period of Fig. 1*C*; Hafed and Ignashchenkova 2013). For example, in Tian et al. (2016), we performed the same analysis as that shown in Fig. 5, *left*, for normal cue-directed microsaccades immediately after cue onset (see Fig. 10 in Tian et al. 2016), and there was no evidence that normal cue-directed microsaccades occurred with such minimal foveal eye position error at fixation as for the express microsaccades described in this study. Therefore, being at a near optimal eye position at the time of cue onset is critical for the triggering of express microsaccades in particular.

Our observations on the relationship between instantaneous foveal eye position error and express microsaccade occurrence were consistent across all three monkeys. For example, in *monkey N*, with the greatest likelihood of express microsaccades for leftward cues, we plotted horizontal eye position

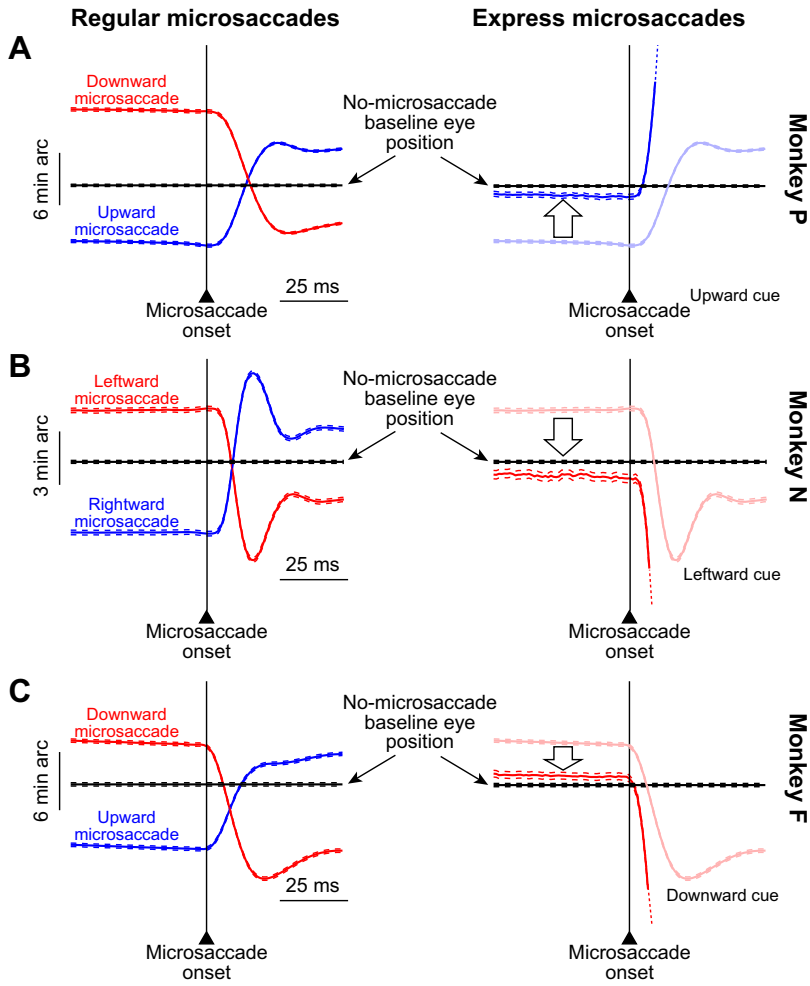

Fig. 5. Spatially, express microsaccades occurred when there was minimal eye position error to correct for at fixation. *A*, *left*: for *monkey P*, the relationship between eye position error during baseline fixation and microsaccade direction is shown. The black line shows average vertical eye position (±SE) during microsaccade-free fixation before cue onset (we ensured that there were no microsaccades 0–300 ms before cue onset, and this plot shows the middle of this interval). We next plotted average vertical eye position (±SE) aligned on microsaccade onset for all upward (blue) or all downward (red) microsaccades. Microsaccade directions were dictated by the sign of eye position error that existed before movement triggering. However, when express microsaccades happened (*right*), they did so when the eye was already almost "balanced" at its optimal fixation position (the black curve in *right* panel is identical to that in *left* panel). That is, the cue happened to appear when the eye was already at its optimal position, making the cue much more effective in triggering an eye movement away from this position. The faint blue curve in the *right* panel is a replica of the blue curve in the *left* panel, to facilitate comparison between regular and express microsaccades (open arrow). For *monkey P*, $n = 7{,}727$ baseline trials (black), 3,795 upward microsaccades (*left*), 3,965 downward microsaccades (*left*), and 53 express microsaccades (*right*). *B* and *C*: similar analyses for *monkeys N* and *F*, respectively. In all cases, express microsaccades were triggered when there was minimal eye position error at fixation when the cue appeared (express microsaccades were also much bigger than regular ones). For *monkey N* (*B*), $n = 9{,}552$ baseline trials (black), 2,497 rightward microsaccades (*left*), 2,173 leftward microsaccades (*left*), and 38 express microsaccades (*right*). For *monkey F* (*C*), $n = 3{,}329$ baseline trials (black), 2,050 upward microsaccades (*left*), 3,060 downward microsaccades (*left*), and 63 express microsaccades (*right*). Note that for each monkey, we analyzed eye positions along the direction resulting in the highest proportion of express microsaccades for clarity of presentation; it is for these directions that the eye was most likely to be near a balance point at the time of cue onset, and therefore most likely to be captured by cue onset in an express manner (see text).

before cue onset (Fig. 5*B*, *left*; $P = 0$, rank sum test for comparisons of baseline eye position with position before microsaccades) and also for express microsaccades (Fig. 5*B*, *right*; $P = 3.4765 \times 10^{-10}$ for comparisons of the difference between eye position before regular microsaccades and baseline with the difference between eye position before express microsaccades and baseline). Once again, leftward microsaccades during baseline fixation were error-reducing toward the baseline oculomotor set point (Fig. 5*B*, *left*; also shown are rightward microsaccades for demonstration of the predictable relationship between microsaccade direction during baseline fixation and instantaneous foveal eye position error). However, leftward express microsaccades (Fig. 5*B*, *right*) were error-increasing instead, and they occurred when foveal eye position error was again already minimal (i.e., with the eye almost balanced at its set point). Finally, for *monkey F*, with most express microsaccades occurring for downward cues, the same conclusion was reached (Fig. 5*C*; $P = 9.8253 \times 10^{-7}$ for comparison of the difference between eye position before regular microsaccades and baseline with the difference between eye position before express microsaccades and baseline). Therefore, in all three monkeys, express microsaccades were triggered when the eye was at a near-equilibrium position around the preferred retinal locus of fixation at the time of cue onset, meaning that the cue could easily tip the balance of fixation and trigger a large, cue-directed express microsaccade.

If eye position does indeed play a role in triggering express microsaccades, then the detailed patterns of instantaneous foveal eye position error at the time of cue onset can modulate express microsaccade likelihood even for a single cue location. Consider, for example, a scenario in which gaze is closer than average to the appearing cue location such that the fixation spot dictates a foveal motor error incongruent in direction with the direction of the appearing cue (see "Closer" condition in *inset* of Fig. 6*A*, *top*). In this case, the oculomotor system is faced with a spatial conflict between the direction needed to correct the current foveal motor error and the direction exerted by the attractive force of the peripheral cue. On the other hand, if fixational gaze happens to be farther away from the cue at the time of its onset, then the direction of the foveal error at fixation would be congruent with the direction of the attractive force of the cue (see "Farther" condition in *inset* of Fig. 6*A*, *top*). One might therefore expect that express microsaccades would be easier to trigger in this case than with the former one.

To demonstrate that this is indeed the case, we measured eye position at the time of cue onset in each of the monkeys, for a given cue location, and we performed a median split of the data based on whether the eye was closer to or farther away from the cue relative to the median eye position observed across trials. For example, in *monkey P* with an upward cue location, the distribution of vertical eye positions at the time of cue onset was that shown in Fig. 6*A*, *top*. Eye positions spatially above

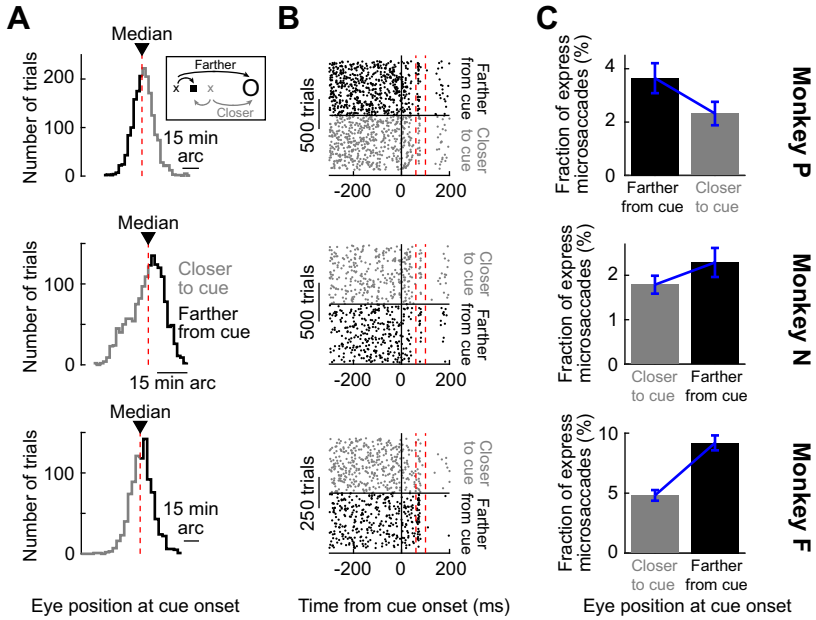

Fig. 6. On a finer scale, the location of the cue relative to instantaneous foveal eye position error at cue onset modulated express microsaccade likelihood. *A*: for each monkey (across rows), we measured foveal eye position error at cue onset for the cue location that elicited the most express microsaccades (same data as Fig. 5), and we obtained a median split of the trials according to whether gaze was closer to or farther away from the cue. When gaze was farther than the median, this meant that the cue location was congruent with the instantaneous foveal position error that microsaccades would hypothetically have to correct for; when gaze was closer, then foveal error relative to the fixation spot was opposite in direction to the cue location (see *inset* schematic where the cross is gaze, the square is the fixation spot, and the circle is the cue). *B*: microsaccade onset rasters for the "closer" or "farther" data subsets of *A*, with express microsaccades highlighted by the red dashed lines. In each of the monkeys, express microsaccades were more likely when the eye was farther from the median position than when it was closer. *C*: express microsaccade frequency for the 2 data subsets in each monkey. Error bars denote 95% confidence intervals. Farther trials had more express microsaccades than closer trials. This means that even though the eye was almost balanced at cue onset (Fig. 5), any remaining foveal error was such that the cue onset location was congruent with the direction of a corrective microsaccade needed to eliminate the existing small foveal error. Note that the color coding of closer or farther in was done to maintain the true directions of eye movements in each monkey. For example, in *monkey N*, the cue was to the left such that the farther trials from the median had eye position being more to the right (i.e., larger values of eye position in the graph). Similarly, for *monkey P*, the cue was upward such that closer trials had more upward eye positions (i.e., larger values of eye position in the graph).

the median position were closer to the cue than eye positions spatially below the median position. In Fig. 6*B*, we plotted the rasters of microsaccade onset times relative to cue onset for the two subsets of trials, and we analyzed the express microsaccades highlighted. There were more express microsaccades for the farther trials than for the closer trials, as we hypothesized ($P = 0.0275$, Wilcoxon signed-rank test; also see Fig. 6*C*, *top*, with 95% confidence intervals shown). In other words, when foveal motor error was minimal (Fig. 5) but congruent in direction with the cue location (Fig. 6*A*, *top*), more express microsaccades were triggered in *monkey P*. This was also true in the other two monkeys. Specifically, for *monkey N* with leftward cues, eye positions to the right of the median eye position at cue onset were farther from the cue than eye positions to the left (Fig. 6*A*, *middle*), and these eye positions were still associated with more express microsaccades (Fig. 6, *B* and *C*, *middle*; $P = 0.0413$). Similarly, in *monkey F* with downward cues exhibiting the highest express microsaccade likelihood, eye positions spatially above the median position were associated with more express microsaccades than eye positions spatially below the median position (Fig. 6, *A–C*, *bottom*; $P = 0.0096$).

Thus the role of eye position in relation to express microsaccades may be summarized by the "energy landscape" analogy shown in Fig. 7. If the eye is relatively far from the preferred retinal locus of fixation (i.e., the eye position set point), then this preferred locus is associated with a strong local minimum that attracts gaze toward it (Fig. 7*A*, with the ball rolling down the energy landscape toward the local minimum reflecting the tendency of gaze to correct eye position error). However, when the eye is already close to the preferred retinal locus of fixation, the system is quasi-balanced (similar to an unstable equilibrium; Fig. 7*B*). In this case, the cue onset exerts a much more effective attractive force on gaze than if foveal eye position error was large. Note that in Fig. 7*B*, even though the eye (ball in the figure) might be near optimal gaze position, it is still in a position in which the direction of (remaining) foveal eye position error is congruent with the attractive direction of the cue onset; that is, there is a weak local minimum at the preferred retinal locus that attracts gaze in the same direction as the direction caused by the peripheral cue location. Thus the scenario shown in Fig. 7*B* is the most effective scenario for the cue to trigger express microsaccades, as we showed in Fig. 6.

In the above results and figures, we have mentioned that express microsaccades were more likely for some cue locations than for others (e.g., Fig. 1*C*, compare the different-colored histograms), and the bulk of our analyses so far show results from the cue location eliciting the most express microsaccades in a given monkey (but see, for example, Fig. 2). However, we also wondered why one cue location was more effective than others in triggering express microsaccades. One possibility is related to the data shown in Fig. 5. Closer inspection of the regular microsaccade data (i.e., before cue onset) reveals that the average foveal eye position error before a given microsaccade was not the same for all movement directions. For example, for *monkey P*, foveal eye position before upward

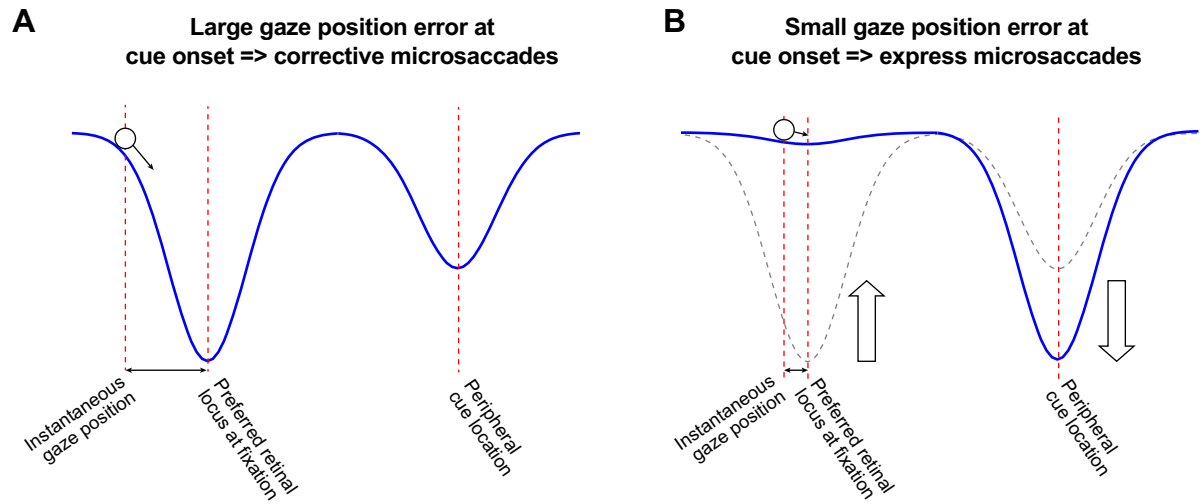

Fig. 7. Express microsaccade occurrence as an outcome of an "energy potential" landscape associated with instantaneous foveal eye position error at fixation. *A*: energy landscape analogy explaining the importance of gaze position at the time of cue onset. When there is a relatively large foveal eye position error at cue onset (as shown in Fig. 5*A*, *left*), there is a substantial local minimum that attracts gaze (ball in this analogy) to the optimal position needed to align the fixation spot with the preferred retinal locus in the fovea. Thus, even with the attractive influence of cue onset, microsaccades behave in a primarily corrective manner like the regular ones shown in Fig. 5*A*, *left* (also see Tian et al. 2016 for similar evidence). *B*: on the other hand, when gaze is almost balanced at the optimal preferred retinal locus for fixation, the local minimum associated with this locus is all but abolished, and cue onset exerts a much stronger attractive influence on eye movements. The dashed gray lines indicate the energy potential when gaze is not at its preferred locus (as in *A*). Note that when the eye is almost balanced but farther from the cue location (Fig. 6), this is equivalent to the scenario shown in this figure, where the (weak) local minimum at the optimal gaze position attracts gaze in the same direction as the attractive influence of the cue location. This explains why farther trials shown in Fig. 6 were the ones always associated with more express microsaccade likelihoods than closer trials. Thus near-optimal gaze fixation along with a remaining foveal error that is congruent with cue location are associated with the highest likelihoods of express microsaccades.

microsaccades was significantly smaller in amplitude than foveal eye position error before downward microsaccades (compare the premicrosaccade eye positions relative to the baseline fixation curve for upward and downward movements in Fig. 5*A*, *left*; $P = 3.3087 \times 10^{-18}$, rank sum test for average eye position in the 50-ms interval before microsaccade onset). This means that it was more likely for the eye to be closer to the optimal set point before upward than downward microsaccades, and it is exactly being close to the optimal set point that is associated with express microsaccade occurrence when a spatially congruent cue appears (Fig. 5*A*, *right*). Similarly, for *monkey N*, eye position error before leftward microsaccades in the precue interval was significantly smaller than before rightward movements ($P = 3.3037 \times 10^{-15}$), again increasing the likelihood of having a congruent cue appear with the eye closer to the set point than for other cue directions. Finally, for *monkey F*, it was eye position before downward microsaccades that was closest to the baseline set point during steady precue fixation ($P = 3.3037 \times 10^{-15}$). Thus one possible reason for why express microsaccades were more likely for some cue locations than others is the likelihood of gaze being near the balance point for these specific directions in which congruent microsaccades would be recruited by the cue (also see results for *experiment 2* below for a causal test of this interpretation, as well as Fig. 9 for why each monkey might drift to a position closer to the set point in one direction vs. others).

Fixational Eye Position Set Points Are Not a Simple Outcome of the Aggregate Impact of Prior Microsaccades

Our results so far indicate that fixational eye position set points are an important determining factor for the generation of express microsaccades. We next turned to the question of the establishment of eye position set points themselves, and whether/how they were modulated as a function of time relative to cue onset. We first asked whether microsaccades alone dictated baseline precue eye position set points or whether such set points were independent of the pattern of microsaccades that each monkey could exhibit. For example, when we inspected the distribution of microsaccade directions and amplitudes during steady-state baseline fixation before cue onset, we found that there were persistent asymmetries that were present in each monkey. An example of such asymmetries is illustrated in Fig. 8*A*, *top* row, for *monkey P* (the other 2 monkeys showed very similar asymmetries). In the leftmost histogram, we divided microsaccades according to whether they were directed toward one of the four quadrants around the fixation spot, and we measured the proportion of all microsaccades that were directed into a given quadrant. This monkey did not have perfectly uniformly distributed microsaccade directions, but it made more microsaccades toward the right visual field (upper and lower right quadrants). Similarly, microsaccade amplitudes were not the same in all four quadrants, but the monkey made slightly larger microsaccades into the left visual field (upper and lower left quadrants; *middle* histogram in Fig. 8*A*, *top* row). Might it then be the case that these asymmetries in microsaccade amplitudes and directions dictate the eye position set points in the precue interval, and therefore influence express microsaccade likelihood after cue onset? We suspected not, but we needed to explore this further.

To investigate this question, we created simulated data in which fixational eye position was solely dictated by microsaccadic displacements in eye position, with no other slow control of intermicrosaccadic gaze position. For each monkey, we measured direction and amplitude asymmetries as above, and we also measured intermicrosaccadic interval distributions (e.g., *right* histogram in Fig. 8*A*, *top* row, for *monkey P*). We

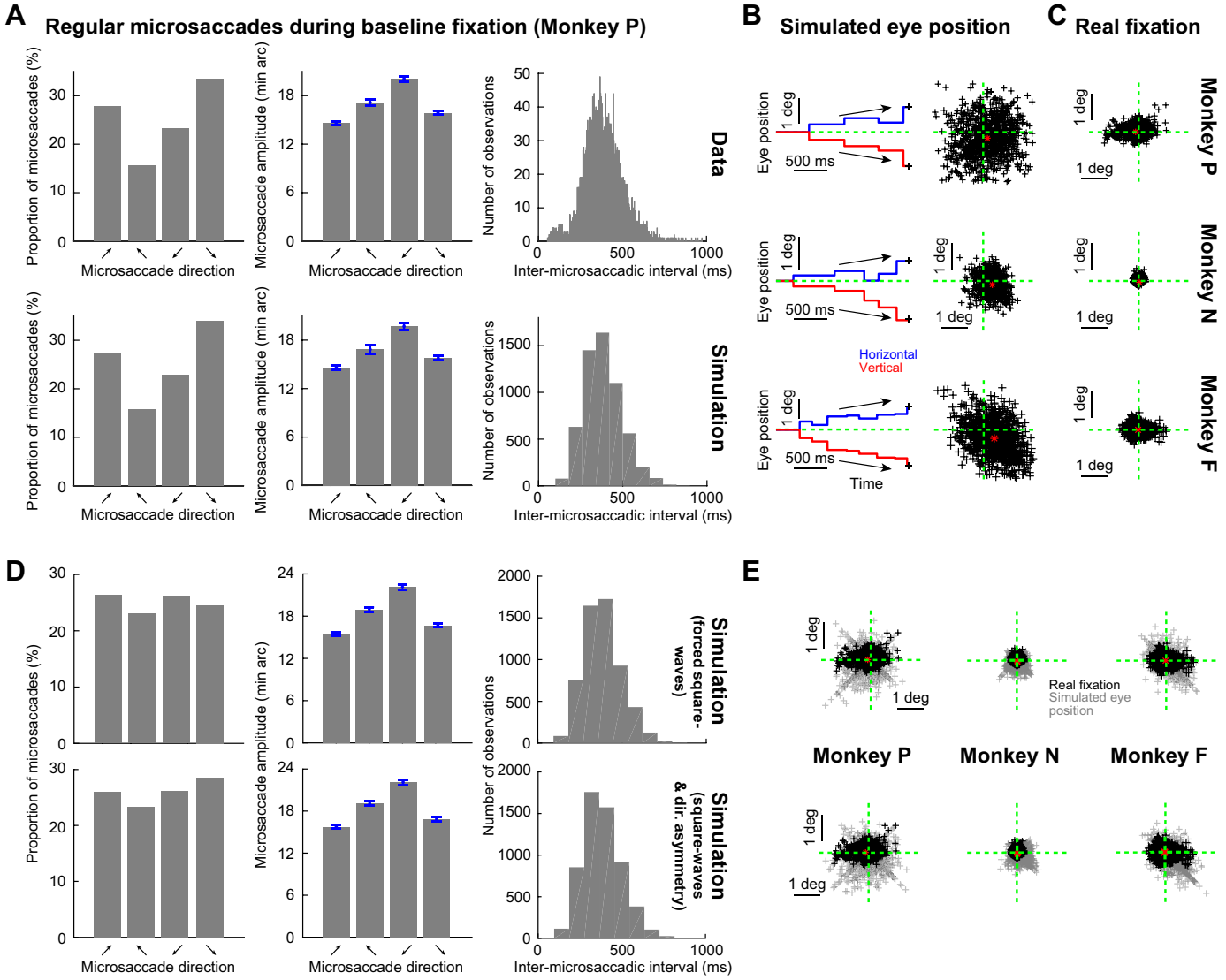

Fig. 8. Optimal baseline fixational eye position was not a simple outcome of the aggregate influence of successive microsaccades. *A*, *top* row: for each monkey (*monkey P* is shown as an example), we classified baseline microsaccades according to their direction (into 1 of the 4 quadrants), and we measured their intrinsic direction (*left* histogram) and amplitude (*middle* histogram) biases. This monkey made more microsaccades toward the upper and lower right quadrants, but these movements were smaller than those into the upper and lower left quadrants. We also measured intermicrosaccadic intervals (*right* histogram). *Bottom* row, we then created simulated data sets in which a microsaccade could occur in a given simulated trial at random with the same biases as in the monkey, and with the same intermicrosaccadic interval distribution (see MATERIALS AND METHODS). *B*: for each monkey, we simulated 2-s fixation trials in which the sole determinant of eye position was the outcome of microsaccades with intrinsic biases and times like those shown in *A* (i.e., there was no drift between microsaccades). *Left*, example simulated trials, demonstrating unstable, runaway fixation if the sole determinant of eye position were microsaccade amplitudes, directions, and times without any position control between microsaccades. *Right*, scatter points show the ending positions of simulated eye position after 2 s of fixation from 1,000 simulated trials. As can be seen, simulated eye position had a large amount of scatter and was biased away from "center." *C*: in contrast, real eye position at the end of the fixation period before cue onset was much more constrained in each of the monkeys (n = 7,727, 9,552, and 3,329 trials for *monkeys P*, *N*, and *F*, respectively). Thus eye position, an important determinant of express microsaccade occurrence (Figs. 5–7), was not a simple outcome of successive microsaccades shifting gaze in particular directions, but it was optimized despite microsaccade biases. *D* and *E*, *top* row: simulations for the same monkey as in *A* (*monkey P*) when microsaccades were forced to come as pairs of square waves (oppositely directed movements). This equalized microsaccade probabilities in all 4 directions (*D*, *left* histogram) but still resulted in runaway fixation due to amplitude asymmetries (*E*). *Bottom* row, similar conclusions for when square waves were still forced to happen but when, additionally, the second movement in a pair was allowed to not always be in the opposite direction such that direction (dir.) asymmetries (*D*, *left* histogram) could still occur as in the real data.

then created simulated trials in which fixational eye position was perfectly stable in between microsaccades. Moreover, the simulated microsaccades happened at random times, but with intermicrosaccadic interval distributions and direction/amplitude biases that were the same as those observed in the real data (Fig. 8*A*, *bottom* row, shows distributions from the simulated microsaccades having similar biases to those in the real data). We then simulated 2 s of fixation (left eye position traces in Fig. 8*B* with simulations matched to each monkey's microsaccade asymmetries). The cloud of dots in Fig. 8*B*, *right*, shows the final eye position after 2 s of simulated fixation from 1,000 simulated trials in each monkey. As can be seen in Fig. 8*B*, if eye position was solely dictated by the aggregate effect of consecutive microsaccades, then asymmetries such as those shown in Fig. 8*A* would result in runaway fixation in all three monkeys. In the real data, eye position at the end of the precue

interval was much tighter than might be predicted by an aggregate sum of prior microsaccades (Fig. 8*C*). Thus eye position set points are somewhat independent of microsaccades, and they arise due to a synergistic interaction between microsaccades and slow ocular drifts (Chen and Hafed 2013; Cherici et al. 2012; Nachmias 1959). Note that this conclusion can still be reached even if there is temporal sequencing of microsaccades (e.g., square-wave microsaccades; Hafed and Clark 2002) as long as overall asymmetries such as those in Fig. 8*A* persisted. For example, as shown in Fig. 8, *D* and *E*, *top* row, we repeated the simulations above, but this time by forcing microsaccades to come in pairs of oppositely directed movements. This violated the direction asymmetries present in the real data, but it still resulted in runaway fixation due to the amplitude asymmetries. We also obtained similar results when we performed additional simulations, again forcing microsaccades to come in pairs, but additionally also allowing the second movement in a pair to not always occur in the opposite direction such that direction asymmetries as in the real data could still occur (Fig. 8, *D* and *E*, *bottom* row).

If it turns out that fixational eye position set points are themselves time varying, as we show next, then the above results mean that fixational eye position alone, independently of perimicrosaccadic influences (Bellet et al. 2017; Chen et al. 2015; Chen and Hafed 2017; Hafed 2013; Hafed et al. 2015; Hafed and Krauzlis 2010; Yu et al. 2017), represents an additional important oculomotor factor that is worthy of consideration during analysis of performance changes in spatial cueing tasks; this would be worthy if for no other reason than at least to investigate the role of instantaneous fixational eye position in facilitating the likelihood of observing express microsaccades.

Fixational Eye Position Set Points Are Modulated by Cue Onset Independently of Whether Microsaccades Happen or Not

The results of Fig. 8 indicate that slow control of fixational eye position was critical in establishing the position set points alluded to above in Figs. 5–7. However, even though prior studies have focused primarily on the modulation of microsaccades in spatial cueing tasks, it remains unknown whether these fixational eye position set points can themselves also be systematically modulated in a time-varying manner after cue onset, independently of microsaccades.

To investigate this, we analyzed all microsaccade-free fixation trials in a short, 200-ms interval around cue onset. For every trial in which there were no microsaccades occurring from −50 to 150 ms relative to cue onset, we plotted average eye position and related it to cue location. We found a small but systematic influence of cue onsets on microsaccade-free eye position in all three monkeys such that the eye systematically drifted by a very small amount in the direction of the cue with a latency of <100 ms from the cue's onset. These results are shown in Fig. 9; for each monkey, and for rightward and leftward cues, Fig. 9*A* shows microsaccade-free horizontal eye

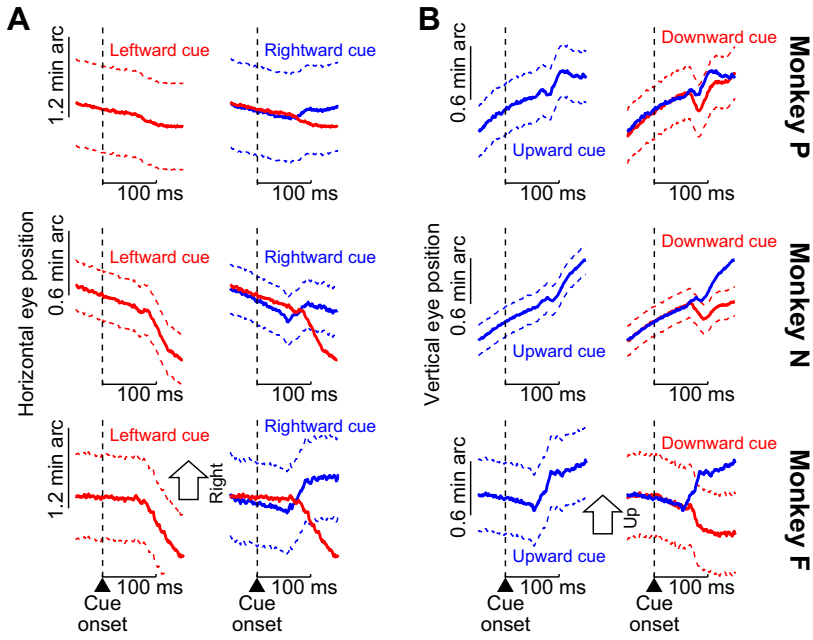

Fig. 9. Eye position was influenced by cue onset even when no microsaccades occurred. *A*: for each monkey (across rows), we plotted horizontal eye position aligned on cue onset for either rightward (blue; *right*) or leftward cues (red; *left*), when no microsaccades occurred in the shown intervals. Dashed lines indicate SE bounds. In all monkeys, cue onset resulted in a short-latency (~100 ms) deviation of eye position in the direction of the cue. This happened even though no microsaccades occurred in any of the shown intervals. The red curves at *right* are copies of those at *left* to illustrate the difference in eye trajectories between the opposing cue locations (error bars in the duplicated plots were omitted to avoid clutter, but they are shown for the originals). Note that *monkey N* had substantial nystagmus-like drift in eye position, but eye trajectory was still nonetheless influenced by cue location. Dashed lines indicate SE bounds; n = 1,289/1,304, 1,310/1,279, and 526/547 (rightward cue/leftward cue) for *monkeys P*, *N*, and *F*, respectively. *B*: similar analyses for vertical eye position trajectories after vertical cue onsets; n = 1,346/1,411, 1,351/1,400, and 686/686 (upward cue/downward cue) for *monkeys P*, *N*, and *F*, respectively. Again, vertical cues caused systematic deviations in eye trajectory during fixational drift (~100 ms after cue onset) that were in the direction of cue location and independent of microsaccades (no microsaccades occurred in any of the trials analyzed in this figure). This happened even in *monkeys P* and *N* with substantial nystagmus-like vertical drift, which was still nonetheless interrupted in a spatially specific manner by cue onset (compare blue and red curves for each monkey). Note that blue curves at *right* are duplicates of those at *left* (without duplication of the error bounds) to facilitate comparison of the different cue directions, as in *A*. Upward deflections in the curves in *A* denote rightward deflections in eye position, and upward deflections in *B* denote upward deflections in eye position.

position near cue onset (*left* panels show horizontal eye position for leftward cues, and *right* panels show horizontal eye position for rightward cues). In all three monkeys, leftward cues shifted eye position by up to ~1 min arc to the left, and rightward cues shifted eye position by up to ~1 min arc to the right, and with a short latency of <100 ms. This happened even in *monkey N*, which exhibited a steady leftward drift in eye position even before cue onset (likely due to a mini-form of nystagmus that some monkeys and humans exhibit during prolonged fixation). Similarly, Fig. 9*B* shows microsaccade-free vertical eye position for upward (*left*) and downward cues (*right*). Once again, note that the influence of cue onset on eye position emerged after <100 ms from such onset, and it was evident even in *monkeys P* and *N* with stronger vertical nystagmus-like drifts than in *monkey F*. Therefore, the results of Fig. 9 further indicate that fixational eye position set points can be systematically modulated independently of microsaccades, as we inferred from the results of Fig. 8. Of course, the systematic cue-induced drifts in eye position that we saw were expectedly small in size, especially because the monkeys were still fixating a salient foveal stimulus, but these systematic drifts in eye position were measurable and on a scale that would activate different foveal photoreceptors, on average, as a result of peripheral cue onsets.

It is also interesting to note the nystagmus-like drift directions in the three monkeys: *monkey N* showed strong leftward/upward drifts (Fig. 9, *middle* row), *monkey P* showed strong upward drifts (Fig. 9, *top* row), and *monkey F* had the most noticeable precue drift in the downward direction (Fig. 9, *bottom* row). These drifts potentially serve to bring the eye closer to the balance set point established in the precue interval (Fig. 5), supporting our interpretation in Figs. 5–7 that the same particular cue locations for each monkey (up for *monkey P*, left for *monkey N*, and down for *monkey F*) were associated with the most express microsaccades when compared with all other cue locations; it was for these cue locations and microsaccade directions that the eye was most likely to be near the optimal eye position set point of the monkey, a necessary condition for express microsaccade generation (Figs. 6 and 7).

Further evidence that the fixational eye position set point can change dynamically in time emerged when we extended the above analyses to much later postcue intervals. Because microsaccades inevitably occurred in such intervals, it was hard to identify particularly long microsaccade-free fixation periods as we did for the shorter intervals shown in Fig. 9. Instead, we picked successive 200-ms intervals of no microsaccades, and we plotted the average eye position in the middle of these intervals. For example, in Fig. 10*A*, each data point relative to cue onset plots the average horizontal eye position after a horizontal cue onset, but subject to the constraint that there were no microsaccades within ±100 ms from that particular data point. Similarly, Fig. 10*B* repeats this analysis for vertical eye position after vertical cue onsets. In all monkeys, eye position was not a stable entity after cue onset. For example, in *monkey P*, after ~200–300 ms from cue onset, eye position systematically shifted away from the cue location for both horizontal and vertical cues. Although this effect might reflect the fact that most microsaccades are known to bias away from cue location at these times (Engbert and Kliegl 2003; Hafed et al. 2011; Hafed and Clark 2002; Hafed and Ignashchenkova 2013; Tian et al. 2016), it does still nonetheless mean that eye position is not a static entity in spatial cueing tasks.

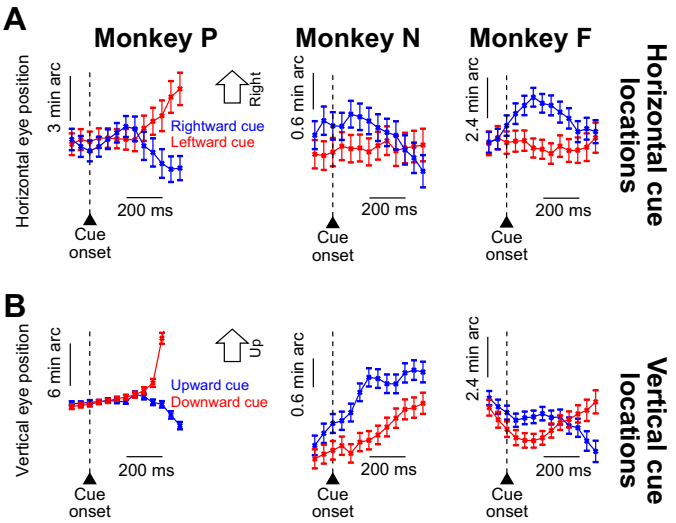

Fig. 10. Optimal baseline fixational eye position was deviated away from cue location in the longer term after cue onset. *A*: for each monkey (across columns), we picked successive 200-ms fixation intervals that did not contain any microsaccades, and we measured mean (±SE) eye position in the middle of these intervals. Eye position was not static after cue onset. For example, for times longer than ~200–300 ms after cue onset, eye position began to shift leftward for rightward cue locations in all 3 monkeys (i.e., opposite the cue location). Similarly, in *monkey P*, eye position shifted rightward for leftward cue locations, with a weaker trend in the other 2 animals. Thus the "baseline" to which microsaccades attempted to balance gaze was not a static entity but changed with time several hundred milliseconds after cue onset. *B*: similar analyses for vertical eye positions after vertical cue onsets. Note that in *monkeys P* and *F*, a clear reversal opposite cue location was evident (as in the horizontal cue conditions) such that eye position shifted upward for downward cues and downward for upward cues long after cue onset. *Monkey N*'s modulations were masked by consistent vertical nystagmus-like shifts in eye position (e.g., Fig. 8). Upward deflections in the curves in *A* denote rightward eye position deflections, and upward deflections in *B* denote upward eye position deflections.

The other two monkeys also showed similar reversals in eye position relative to cue location. For example, in *monkey N*, rightward cues eventually caused more leftward eye positions than leftward cues at the end of the shown interval, and in *monkey F*, upward cues caused more downward eye positions than downward cues at the end of the shown interval (Fig. 10). For vertical cues in *monkey N* and horizontal cues in *monkey F*, changes in the "direction" of eye position modulations as a function of time were consistent with a reversal away from the cue, although they were masked by systematic changes in position that were present even before cue onset (whether due to nystagmus-like drifts or to microsaccade asymmetries, or both). For example, in *monkey N*, the rate of upward drift was slowed down after >300 ms for upward cue locations but accelerated for downward cue locations. Similarly, in *monkey F*, a rightward drift in position switched to being leftward >300 ms after rightward cues.

Therefore, the combined results of Figs. 9 and 10 indicate that cue onset systematically deviated eye position toward its location with short latencies; for longer latencies, the net effect of both microsaccades and slow control of ocular drift meant that eye position was not a static entity, but dynamically shifted primarily away from the cue location under most circum-

stances. Such an "away" shift was also observed recently in similar experiments (Tian et al. 2016).

Experimentally Controlling Fixational Eye Position Set Points at Cue Onset Modulates the Statistics of Early Cue-Induced Microsaccades

Finally, on the basis of all the above results, we wondered whether we could experimentally place the oculomotor system in a state of unstable equilibrium (such as that shown in Fig. 7*B*) to artificially increase the likelihood of large, cue-induced microsaccades. We ran two of our monkeys in a second experiment (*experiment 2*; Fig. 11*A*), in which the monkeys fixated and a peripheral cue appeared (but persisted; see MATERIALS AND METHODS). We randomly interleaved control trials (Fig. 11*A*, *left*) with retinal image stabilization trials. The latter trials employed techniques, which we have used earlier (Chen and Hafed 2013; Tian et al. 2016), that allowed us to artificially move the fixation spot in real time with gaze position before cue onset (Fig. 11*A*, *right*). This ensured minimization of gaze position error at the time of cue onset. If such minimization was sufficient to trigger express microsaccades (Figs. 5 and 7), then we should have seen more cue-directed microsaccades 60–100 ms after cue onset than in the control condition, and these microsaccades should have also been significantly larger in amplitude. This is exactly what we found. In Fig. 11*B*, *left*, we plotted a time course of microsaccade directions after cue onset in the control condition. As we described recently (Tian et al. 2016), we divided microsaccades into movements toward the cue, opposite the cue, or orthogonal to the cue (see MATERIALS AND METHODS). This means that before cue onset, there was a 25% chance that microsaccades toward the cue occurred. Such movements then increased in likelihood early after cue onset, as expected from prior studies, before a reversal of microsaccade directions occurred. Such a reversal can be seen by the increase in movements opposite the cue. Importantly, with experimental control over eye position error at the time of cue onset in the retinal image stabilization trials (Fig. 11*B*, *right*), the increase of movements toward the cue in the critical interval of 60–100 ms was significantly more dramatic. Similarly, the amplitudes of these movements were also larger than in the control, as can be seen from the time courses of microsaccade amplitudes shown in Fig. 11*C*. Thus experimentally placing the oculomotor system at a point of equilibrium, albeit an unstable one (Fig.

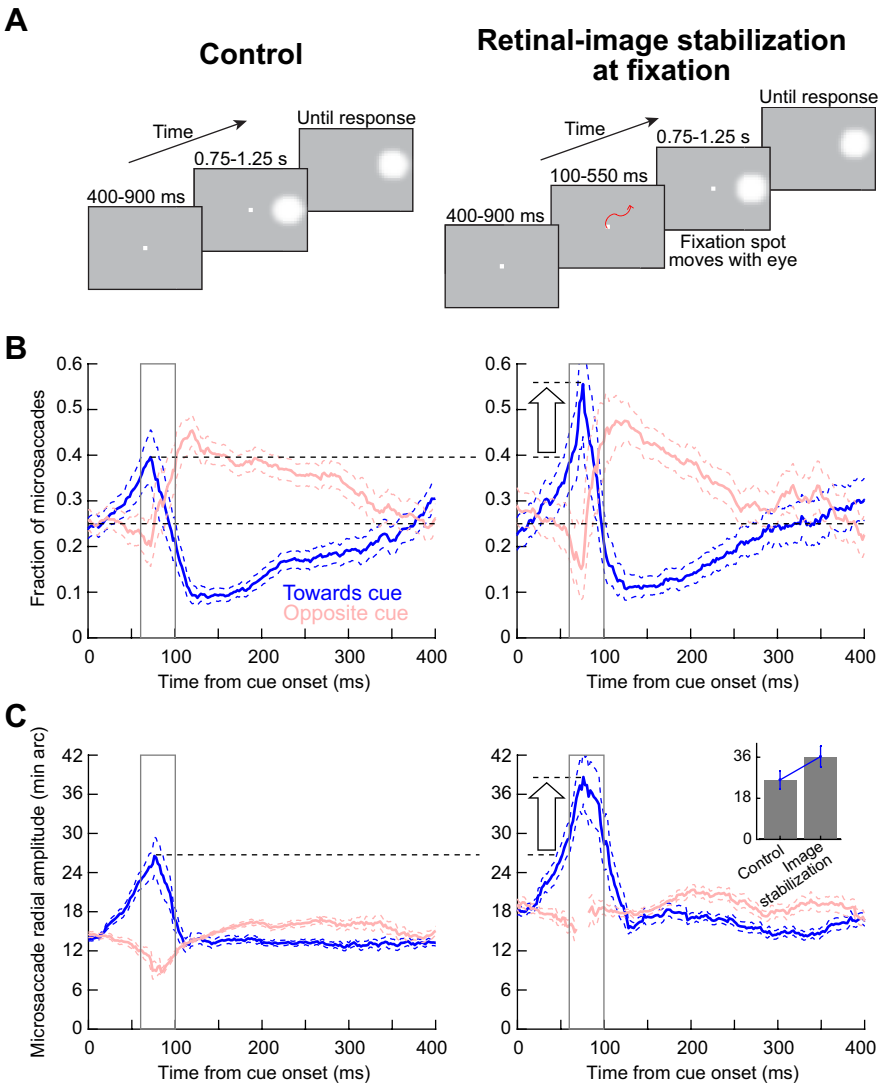

Fig. 11. Causally manipulating the properties of early cue-induced microsaccades by real-time stabilization of the instantaneous retinal image position of the fixation spot. *A*: in *monkeys P* and *N*, we ran control trials interleaved with retinal image stabilization trials, similarly to Tian et al. (2016). Monkeys fixated, and a peripheral cue appeared for a variable duration. In retinal image stabilization trials, the fixation spot was moved with gaze in real time such that foveal eye position error at the time of cue onset was minimized (red arrow). When the cue appeared, retinal image stabilization was stopped. *B*: time course of microsaccade directions after cue onset. *Left*, control trials; *right*, retinal image stabilization trials. Dashed lines denote 95% confidence intervals. In each condition, there was an increase in microsaccades toward the cue in the highlighted rectangle (also see Hafed et al. 2011, 2013; Hafed and Ignashchenkova 2013; Tian et al. 2016). However, in the retinal image stabilization condition, the increase was significantly stronger (open arrow), consistent with the mechanism shown in Fig. 7. That is, with foveal gaze position error in balance, the cue's attractive influence on gaze was more effective. All analysis methods for this figure are similar to those described in detail by Tian et al. (2016). *C*: similar time course analyses but for microsaccade amplitude. Graph at *right* shows that early cue-directed microsaccades were bigger when instantaneous foveal error was controlled than when it was not (open arrow). *Inset* shows microsaccade amplitudes in the interval 60–100 ms after cue onset, showing an increase in the retinal image stabilization condition. Error bars denote SE ($P = 0.034$, rank sum test). Thus controlling instantaneous foveal eye position error at the time of cue onset has a significant impact on the efficacy of the cue to influence subsequent microsaccades, consistent with the mechanism shown in Fig. 7.

7), was sufficient to increase the likelihood of express cue-directed microsaccades (and with larger amplitude), even though the manipulation was only a subtle and very brief manipulation at the fixation spot with all other stimulus conditions being identical to those in the control trials.

Our results from both experiments combined suggest that fixational eye position dynamics are an important, yet thus far largely neglected, aspect of analyzing fixational eye movements in spatial cueing tasks.

DISCUSSION

Using careful gaze position measurements, we found that fixational eye position is an important factor that is dynamically modulated in spatial cueing tasks, adding to the much more studied changes in microsaccades that are routinely observed in these tasks. Our observation of changes in microsaccade-free fixational gaze position immediately after cue onset (Fig. 9) is particularly intriguing, because it demonstrates fine stimulus-induced control over nonsaccadic ocular drifts during fixation, and also because it shows that dynamic changes in fixational eye position during spatial cueing tasks are not entirely accounted for by cue-induced changes in microsaccade directions and amplitudes alone (Fig. 8). Moreover, time since the last microsaccade (Nachmias 1959) can be used, in conjunction with eye position measurements, to predict in real time whether microsaccades are expected to occur in response to visual stimuli. Because microsaccades are associated with substantial visual performance changes (Bellet et al. 2017; Chen et al. 2015; Chen and Hafed 2017; Hafed 2013; Hafed and Krauzlis 2010; Tian et al. 2016; Watanabe et al. 2013, 2014; Yu et al. 2016, 2017), such predictions could be used to control behavioral performance and neural activity in a variety of paradigms requiring gaze fixation.

Express Stimulus-Induced Microsaccades

In *experiment 1*, we identified a special set of microsaccades that we termed "express stimulus-induced microsaccades." These movements were genuine responses to stimulus onset, even though they occurred with express latencies. For example, they were entirely directed toward the cue, and they occurred as a distinct population in latency histograms such as those shown in Fig. 2. These movements are thus not like escape microsaccades that are invoked in some models of the impact of spatial cueing on microsaccades (Hafed and Ignashchenkova 2013). These movements are also functionally relevant because they were associated with magnified task effects (Fig. 3), adding yet more evidence that microsaccades can alter performance in cueing tasks (Chen et al. 2015; Hafed 2013; Tian et al. 2016).

We found that express microsaccades were most likely to occur if no prior movements had occurred for a long time (Fig. 4) and also if the eye was already at a balance point during gaze fixation (Figs. 5–7). The former point is consistent with accounts of interactions between microsaccades and drifts (Nachmias 1959), but the latter is not. This latter point was also further supported by our use of real-time retinal image stabilization of the fixation spot to causally test the role of eye position on early cue-directed microsaccade statistics (Fig. 11), and it is consistent with Engbert's (2012) model of microsaccade triggering in tasks like ours. This idea of balance, of course, does mean that the balance is only momentary, because eye position continuously changes by minute amounts (Figs. 8 and 9). Thus the balance may be thought of as an unstable equilibrium state such that any perturbation of this state can push it away from balance. This is exactly what cue onset does, and this is why express microsaccades act to increase foveal eye position error as opposed to reducing it (Figs. 5 and 7).

The idea of momentary balance would also indicate that significant trial-to-trial variability in behavior in cueing tasks can be related to the instantaneous state of the oculomotor system. For example, momentary reductions of neural activity in the rostral superior colliculus (SC), a region related to the small eccentricities associated with microsaccades, are associated with increased visual bursts in more eccentric regions (Jagadisan and Gandhi 2016). If such reductions are correlated with oculomotor balance, then stronger visual bursts could contribute to express microsaccade generation and magnified cueing effects. This would be consistent with the notion that SC visual bursts have high correlation with saccadic RT (Chen and Hafed 2017; Hafed and Krauzlis 2010).

An additional intriguing property of express microsaccades is that they had variable likelihoods for different cue locations. We think that this is a consequence of how close eye position normally was to the optimal baseline eye position at any one moment, and with a remaining foveal error that is congruent with cue location. For example, in Fig. 5, *left*, eye position before individual microsaccade directions was associated with lower initial error (from the preferred retinal locus of fixation) compared with other microsaccade directions, even in the precue baseline interval. This means that for some microsaccade directions (e.g., upward in *monkey P* and leftward in *monkey N*), the eye was more likely to be closer to the balance point at the time of cue onset than in the other directions. This, in turn, increased the likelihood of express microsaccades in individual directions with the onset of directionally congruent cues. As for why the eye was closer to the optimal baseline position in some directions versus others, this could be due to minute ocular drifts as observed in Fig. 9 (also see Nachmias 1959). All of these observations demonstrate the importance of fixational eye position in spatial cueing tasks, because such position not only alters the position of retinal images but also can dictate the types of microsaccades that can be triggered.

Control Over Slow Ocular Drifts to Modulate Fixational Eye Position Set Points

Our results from Figs. 8 and 9 suggest that fixational eye position has a set point that can be modulated depending on task constraints and stimulus conditions. Even without any microsaccades, cue onset resulted in a consistent drift in eye position toward the cue with a latency of <100 ms (Fig. 9). This means that the fixational eye position set point was shifted after cue onset, contradicting recent suggestions (Poletti et al. 2017) that cue onset does not modulate eye position drifts. We think that these authors would have seen very similar effects to ours if they had analyzed eye position more closely. More importantly, our results suggest that slow ocular drift is not a random process but that it is directly influenced by visual stimuli, and therefore likely to be centrally controlled by the nervous system. For example, SC visual bursts, which can hypothetically even modulate the kinematics of microsaccades

in midflight (Buonocore et al. 2017; also see Jagadisan and Gandhi 2017 for similar ideas), could introduce a directional component to the drive of downstream premotor oculomotor nuclei and cause directional drifts in eye position, as we have seen. Indeed, it would be interesting to relate the properties of the transient, short-latency changes in eye position that we observed in Fig. 9 to the three known types of fixational eye movements that are classified in the literature (e.g., see Martinez-Conde et al. 2004), and ultimately to brain stem mechanisms.

In a similar vein, we observed that for even longer times after cue onset, the eye position set point was shifted primarily opposite the cue location (Fig. 10). We also noticed a phenomenon similar to that in Fig. 10 of Tian et al. (2016) in other experiments. Of course, the occurrence of previous microsaccades within a trial would contribute to the establishment of such a new set point, but what is intriguing is that it was shown in Tian et al. (2016) that late microsaccades (i.e., long after cue onset) corrected eye position errors toward the new shifted set point even though this new set point was deviated away from the preferred retinal locus of fixation that existed before cue onset. That is, the shifted set point was the new point to which microsaccades occurring long after cue onset corrected for. Moreover, in our Fig. 8, we demonstrated that an eye position set point was not necessarily trivially explained by the aggregate influence of previous microsaccades in a trial. Instead, slow drifts synergistically interact with microsaccades to establish the new set point (Chen and Hafed 2013; Herrmann et al. 2017; Nachmias 1959). All of these observations intriguingly suggest that the new eye position set point long after cue onset was deliberately established by the oculomotor system.

Gaze Fixation as Equilibrium

The above observations lead to questions of why the new set point was deviated away from cue location long after the cue. This could be related to the idea of establishing a new balance point of fixation given the "tipping" effect that the cue causes on eye movements (Fig. 7); that is, whether due to a lingering visual effect of the cue or due to a top-down signal associated with the cued location, the landscape of fixation is disrupted by cue onset, causing an imbalance in favor of the cued side. To rebalance fixation given this imbalance, the eye might deviate ever so slightly away from the cued location, perhaps reflecting a compensatory elevation of activity in the opposite hemifield (Tian et al. 2016). This is consistent with how the SC is believed to contribute to gaze fixation through balanced population activity (Goffart et al. 2012; Hafed et al. 2009; 2008).

An additional question related to this topic is how eye position can be controlled beyond the SC balance idea just mentioned, and what such control of eye position implies. Neurons in several brain areas, such as parietal cortex (Andersen et al. 1985, 1990), premotor cortex (Boussaoud et al. 1998), and prefrontal cortex (frontal eye field/supplemental eye field) (Boussaoud et al. 1993; Cassanello and Ferrera 2007; Schall 1991), exhibit so-called eye position gain fields. These neurons' various sensitivities are modulated as a function of absolute eye position. It could be that neurons with gain fields can contribute to establishment of the new fixational eye position set points at different times after cue onset, and given the small changes in eye positions that we observed, our results suggest that the resolution of eye position control, whether from parietal areas or elsewhere, has to be quite high. In a complementary fashion, it could be the case that fixational eye position set point shifts are implemented exactly to alter neurons' various sensitivities by exploiting these neurons' eye position gain fields. Either way, it would be interesting to better understand the detailed role of position control circuitry in not just eye position in cueing tasks but also in how the retinal implications of eye position can affect task performance. In the past, one of our primary foci in our laboratory was on the influence of perimicrosaccadic changes in vision on performance changes in attentional tasks (Chen et al. 2015; Chen and Hafed 2017; Hafed 2013; Hafed et al. 2015; Hafed and Krauzlis 2010; Tian et al. 2016), but our current results suggest that eye position itself, and its associated drifts in eye position, are important in their own right because they modify the statistics of slow changes in the retinal image of stimuli in the task.

Task Constraints and the Likelihood of Express Microsaccades

Finally, why is it that we saw express microsaccades in the frequency histograms (e.g., Figs. 1–2) even though earlier studies did not see them so prominently? We think that this is a function of task requirements. Specifically, our task design had some trials with very short CTOAs. Given that the monkeys had to generate a saccade at target onset, there were trials in which the monkeys had to generate saccades very rapidly after cue onset. Under these circumstances, it is useful to reduce microsaccade frequency because microsaccade occurrence can influence reaction time (Chen and Hafed 2017; Hafed and Krauzlis 2010; Sinn and Engbert 2011; Tian et al. 2016; Watanabe et al. 2014). Because we saw (Fig. 4) that express microsaccades were most likely to occur when there had been no microsaccades for a very long time, it makes sense that a task causing reductions in microsaccade frequency should be associated with a higher likelihood of express microsaccades. We also have anecdotal evidence of this in human subjects performing a highly demanding perceptual task. Leading up to stimulus onset in this task, microsaccade rate was reduced dramatically (similar to the late microsaccade rate in the attention task of Hafed et al. 2011). Under these conditions, stimulus onset caused a large peak of express microsaccades in the humans exactly like what we saw with our monkeys in the present study.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

X.T., M.Y., and Z.M.H. conceived and designed research; X.T., M.Y., and Z.M.H. performed experiments; X.T. and Z.M.H. analyzed data; X.T., M.Y.,

and Z.M.H. interpreted results of experiments; X.T. and Z.M.H. prepared figures; Z.M.H. drafted manuscript; M.Y. and Z.M.H. edited and revised manuscript; X.T., M.Y., and Z.M.H. approved final version of manuscript.

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