

Perisaccadic visual perception

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Primates use frequent, rapid eye movements to sample their visual environment. This is a fruitful strategy to make the best use of the highly sensitive foveal part of the retina, but it requires neural mechanisms to bind the rapidly changing visual input into a single, stable percept. Studies investigating these neural mechanisms have typically assumed that perisaccadic perception in nonhuman primates matches that of humans. We tested this assumption by performing identical experiments in human and nonhuman primates. Our data confirm that perisaccadic visual perception of macaques and humans is qualitatively similar. Specifically, we found a reduction in detectability and mislocalization of targets presented at the time of saccades. We also found substantial differences between human and nonhuman primates. Notably, in nonhuman primates, localization that requires knowledge of eye position was less precise, nonhuman primates detected fewer perisaccadic stimuli, and perisaccadic compression was not towards the saccade target. The qualitative similarities between species support the view that the nonhuman primate is ideally suited to study aspects of brain function—such as those relying on foveal vision—that are uniquely developed in primates. The quantitative differences, however, demonstrate the need for a reassessment of the models purportedly linking neural response changes at the time of saccades with the behavioral phenomena of perisaccadic reduction of detectability and mislocalization.

Introduction

To make optimal use of the highly sensitive central part of the retina, primates use frequent, rapid eye movements to sample their visual environment. Even though this active sampling strategy has many benefits (Gegenfurtner, 2016), it also results in numerous challenges. The rapid motion across the retina should not be interpreted as motion in the world, the brain must keep track of where the eyes are pointing to know

where objects are in the world, and the successive snapshots of the world must somehow be combined to provide a continuous, stable percept and the illusion of perceived detail across the visual field.

In humans, perceptual stability has been studied primarily using behavioral methods that probe visual perception around the time of eye movements. Two well-documented phenomena that are thought to provide a glimpse of the operation of the mechanisms underlying perceptual stability have been particularly well documented. First, around the time of eye movements, visual sensitivity is temporarily reduced (saccadic suppression). Second, objects presented around the time of eye movements are mislocalized (perisaccadic mislocalization).

In nonhuman primates, related research has primarily used electrophysiological techniques to describe how impending and ongoing saccades affect neural response properties across the visual hierarchy. Most notable are saccade-related biphasic changes in neural responsiveness, changes in receptive field location, and changes in the representation of eye position. Some of these neural changes have also been identified in humans, using functional imaging and electroencephalography. For reviews that cover both the human and animal subjects literature see (Ibbotson & Krekelberg, 2011; Krock & Moore, 2014; Wurtz, Joiner, & Berman, 2011).

Computational models link behavioral phenomena and electrophysiological findings, and have led to new insights and new experiments (Hamker, Zirnsak, Ziesche, & Lappe, 2011; Pola, 2004, 2011; Teichert, Klingenhoefer, Wachtler, & Bremmer, 2010). However, given the paucity of behavioral data in nonhuman primates and the relatively coarse neural data that can be obtained in humans, such models must assume that the behavioral phenomena observed in humans are substantially similar to those in nonhuman primates. This assumption is largely untested. Building on four previous studies in nonhuman primates (Dassonville, Schlag, & Schlag-Rey, 1992; Hass & Horwitz, 2011;

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Jeffries, Kusunoki, Bisley, Cohen, & Goldberg, 2007; Mohler & Cechner, 1975), we developed a behavioral paradigm to quantify perisaccadic perception. We use the word perisaccadic here to denote a brief (~200 ms) time window centered on saccade onset. Our paradigm measures visual detection and localization for targets presented during this perisaccadic time window using identical paradigms in human and nonhuman primates.

Qualitatively, our behavioral experiments confirm the similarity in perisaccadic visual perception of macaques and humans. Specifically, we found a clear reduction in detectability (saccadic suppression), and substantial mislocalization of targets presented at the time of saccades (perisaccadic mislocalization). This further supports the claim that the nonhuman primate is well suited to study aspects of brain function—such as those relying on foveal vision—that are uniquely developed in primates. At the same time, however, our results show substantial differences between human and nonhuman primates—both in the magnitude of perisaccadic changes in detectability and the spatial pattern of perisaccadic mislocalization. This suggests that explaining human behavioral data with nonhuman primate neural response properties should only be done with caution and that a fuller understanding of the mechanisms underlying visual perception requires the simultaneous recording of neural and behavioral responses.

Materials and methods

All animal procedures were approved by the Rutgers University animal care and use committee and were in agreement with the National Institute of Health's guidelines for the humane care and use of laboratory animals, and with the animal research statement of the Association for Research in Vision and Ophthalmology. All human subjects provided written informed consent and reported normal or corrected-to-normal vision. Human experimental procedures were approved by the Institutional Review Board of Rutgers University and followed international guidelines for the ethical treatment of human subjects as expressed in the Declaration of Helsinki.

Subjects and apparatus

Three male macaques (*M. mulatta*), and five humans (*H. sapiens*) participated in these experiments. One macaque (M3) was head-fixed using an implanted titanium head post; the two other macaques (M1 and M2) performed the experiments head-free. To ensure an identical visual environment, the human subjects

(one female, four male) performed the experiments in the same setup using a chin rest.

After several months of acclimation and training to exit their home cages using the pole-and-collar method, the macaques received training to perform simple fixation tasks, and then training to perform the localization task. In each experiment, the animals sat in a custom primate chair and humans on a stool, with their eyes at a distance of 57 cm from the CRT monitor that displayed all visual stimuli (Sony GDM 520, 100 Hz, 1024 × 768 pixels, 40 × 30 cm). The monitor was the only source of light in the recording booth.

Eye movements were tracked with an infrared eye tracker (Monkey M3: Eyelink II; 120 Hz, nominal resolution 0.1°; Monkey M1 and M2 and the human subjects: Eyelink 1000, 500 Hz, nominal resolution 0.5°; SR Research, Ottawa, Canada). Animals, but not humans, received liquid reward for correct performance.

Visual stimuli

Monitor luminance was calibrated and linearized using the built-in procedures of the Sony GDM 520. The equal energy white background luminance of the screen was 5 cd/m².

The visual elements used in the tasks described below are the fixation target (F: red dots, 0.5° diameter, 25 cd/m²), always placed on the horizontal midline of the monitor. The localization target (T) was an equal energy white 1.25° square, (15 cd/m²), and the array of choice targets (below) consisted of squares identical to T. The choice arrays always appeared both 8.0° above and 8.0° below the horizontal midline and always spanned the width of the monitor. To create trials with different levels of difficulty, the spacing of the targets in the fixation conditions was chosen at random from 2°, 2.5°, 3°, and 3.5°. In the perisaccadic conditions, the spacing was always 2°.

Task

In the localization task (Figure 1), the fixation point F1 appeared 8° to the left of the vertical midline of the monitor. A variable delay (500–700 ms) after fixation was achieved, a new fixation point (F2) appeared on the opposite side of the monitor (+8°). The subject had to execute a saccade to this new position within 400 ms and maintain fixation (within ±1.5°) for 600 ms. This description covers rightward saccades, but analogous leftward saccades were also used (on separate days); data from sessions with leftward saccades were mirrored, and pooled with the data from rightward saccades.

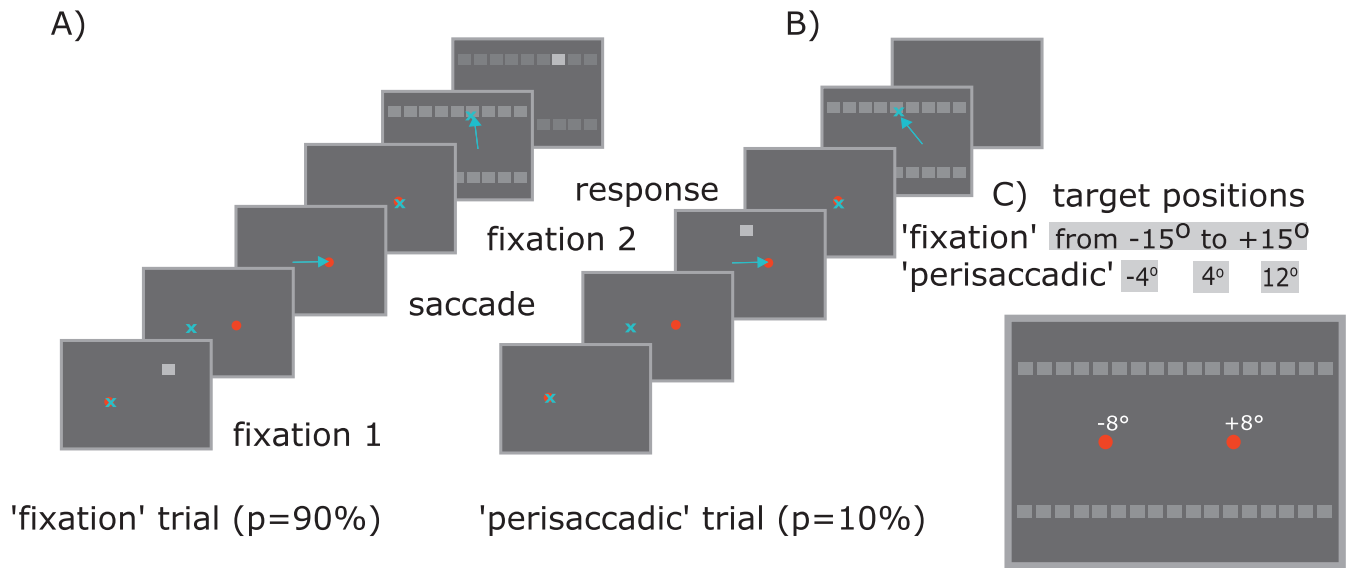


Figure 1. Experimental paradigm. (A) Timeline of a fix-pre trial with a rightward saccade. The subject fixates (F1), some time later, the target (T) is briefly flashed, and then, at least 250 ms later, the fixation point (F2) shifts and the subject follows it by making a saccade. An array of potential choices appears after 600 ms and the subject makes a saccade to one of the squares in the choice array and receives visual feedback about the correct location. In the analogous fix-post trial, the target is flashed at least 400 ms after the saccade to F2. (B) Perisaccadic trial. The main difference with the fixation trials in (A) is that the target (T) is presented within ± 150 ms from saccade onset. Because the perceived location of the flash is expected to differ from its physical location, no feedback about the correct location is given. (C) Spatial layout. Red points show the location of the fixation point before (F1) and after (F2) the saccade. Gray squares show the choice array; the subject can choose one (by fixating it) to indicate the perceived location of the target T. Gray squares at top show the physical location of the targets in perisaccadic trials with a rightward saccade. In fixation trials, the target locations were chosen randomly between -15° and 15° . The spatial layout in leftward saccade trials was mirror symmetric.

In randomly interleaved trials, the localization target (T) appeared while the animal was fixating F1 (T presented at least 400 ms before saccade onset; *fix-pre* trials), in the perisaccadic window (*perisaccadic trials*; within ± 150 ms from saccade onset), or while the animal was fixating F2 (T presented at least 400 ms after saccade onset; *fix-post* trials). The presentation duration of T was 30 ms (three monitor frames) in both fixation conditions and 10 ms (one frame) in the perisaccadic condition. The target T appeared 8.0° above or below the horizontal midline equally often in randomized order. In fix-pre and fix-post trials (collectively referred to as fixation trials for short), the horizontal position was randomly drawn from the range $[-15^\circ, 15^\circ]$. In perisaccadic trials T appeared at horizontal positions -4° , $+4^\circ$, or 12° for rightward saccades and at the mirror symmetric positions $+4^\circ$, -4° , and -12° for leftward saccades (see Figure 1).

After the saccade, two linear arrays of squares (choice arrays) appeared on the screen and remained visible until the end of the trial. One array was positioned at the same vertical position as the target. The second array was placed at the vertically mirrored location. The subject was trained/instructed to make a saccade to one of the elements of the choice arrays (choice targets) within 2000 ms. Once a subject maintained fixation for more than 400 ms within a

window of $\pm 1^\circ$ around a choice target this was considered the subject's perceived position of the flashed target T. (Often subjects made two to three saccades to reach this endpoint). In the fixation trials, one of the choice targets always coincided with the position of the target T. To increase the spatial resolution of the average measurements in the perisaccadic conditions, the choice arrays were shifted by $\pm 1^\circ$ on half the trials.

In fixation trials, subjects received visual feedback at the end of each trial; the square at the target position became brighter while the others were dimmed. In addition, a tone was used to indicate the correctness of the response (high tone: correct response, fixation within $\pm 1^\circ$ of the actual location of T; low tone: incorrect response). Monkey subjects additionally received liquid reward after providing the correct answer.

In perisaccadic trials, no visual or auditory feedback was provided. This was done to avoid cues to the subjects that could allow them to develop a strategy in which they made a saccade to a different location than the perceived location in order to maximize reward. Liquid reward was given randomly in 40% of trials in which the animal chose a square from the choice array with the same vertical position as the target (irrespective of the horizontal position of the choice). No

reward was given on trials in which the monkey chose a square from the array at the vertically mirrored position.

If the subject did not meet the eye position requirements (within $\pm 1^\circ$ during any of the fixation phases or within $\pm 1.5^\circ$ for the saccade to F2), auditory feedback was provided (buzzer sound), the trial was terminated, not used in the analysis, and the condition was repeated later during the session.

Training procedure

Human subjects were given verbal instructions and performed 100 practice trials to master the task. Animal subjects were first trained on fix-post trials only (i.e., the fixation point did not move between presenting the target and the animal's choice). In this phase, spacing of the choice array was 3.5° and targets were presented for up to 100 ms. After several weeks of training, we introduced the eye movement requirement by stepping the fixation point from F1 to F2. In this phase, target presentation remained confined to the fixation periods before and after the saccade (i.e., fix-pre and fix-post trials only). Gradually, over the course of several months, the duration of T was reduced to 30 ms and the spacing between answer targets was reduced to 2° . Once the animal reached consistent (i.e., multiple sessions) high performance ($>40\%$ correct) on localization of targets at all positions, we introduced perisaccadic trials (10 ms duration of T).

On these trials, we expect the subjects to mislocalize the targets. If we gave the animals reward based on the veridical location of the targets, they could develop a response strategy in which they corrected for their illusion—obviously an undesirable outcome. If, on the other hand, we gave reward based on a predicted illusory (e.g., shifted) location of the targets, we would simply be training the animals to report a shifted location, and not measure their percept. In other words, to quantify an illusory percept, reward must be provided independent of the animal's report (Krekelberg, Dannenberg, Hoffmann, Bremmer, & Ross, 2003). The animal, however, is of course unaware of this decoupling of report and reward. Consequently, it will adjust its report from trial to trial in a doomed attempt to find the response that maximizes reward. In our experience, this can lead to peculiar response strategies (e.g., picking the same location every trial, or alternating between a few stereotypical locations). It is therefore key to use report-independent rewards on only a small fraction of trials and embed them in a large number of trials in which veridical report can be rewarded. These latter trials reinforce the desired response strategy “report the perceived location of the target” and the expectation is that the animal will then

use the same strategy in the critical trials in which reward is independent of the animal's report. Specifically, we randomly interleaved fixation trials (90% of trials; rewarded for correct localization of the veridical location, as above) and perisaccadic trials (10% of trials; randomly rewarded) in all experiments.

Data analysis

For the moving averages in Figure 2, we binned the physical target position in 2° bins and then averaged the reported position for each of those targets, separately for each subject. To estimate variability around this estimated mean, we then subtracted the moving average from the reported target position and determined the standard deviation of these residuals (error bars in Figure 2).

For the moving averages of detection performance (Figures 3 and 4) and perisaccadic localization (Figures 5, 6, and 7), the window size was 20 ms, and data points are plotted at the center of the window.

For the statistical analysis of the time course of detection and mislocalization, we first binned the measure of interest in 40 ms wide nonoverlapping time bins ($[-80, -40]$, $[-40, 0]$, $[0, 40]$, $[40, 80]$ ms). These windows were chosen such that they contained approximately the same number of data points. We used a Fisher exact test to assess the statistical significance of detection performance and an analysis of variance (ANOVA) with time-bin, species, and physical target location as fixed effects and subject as a random effect for the localization data.

To show group averages (Figures 4 and 7) we bootstrap resampled the detection/localization data with replacement (10,000 times). The shaded areas around the curves represent the bootstrap estimates of the 95% confidence limits. Before averaging localization data across subjects, we removed the subject-specific localization biases that were not closely time-locked to saccade onset. Visual inspection of individual subject data suggested that perceived location was approximately constant for targets presented more than 40 ms before saccade onset (Figure 5). We therefore estimated, per physical target location and per subject, the average perceived location of targets presented more than 40 ms before saccade onset and subtracted this number from the individual localization curves to create bias-corrected localization curves.

Results

We assessed perisaccadic visual perception in three male macaques and five humans (four male). They were

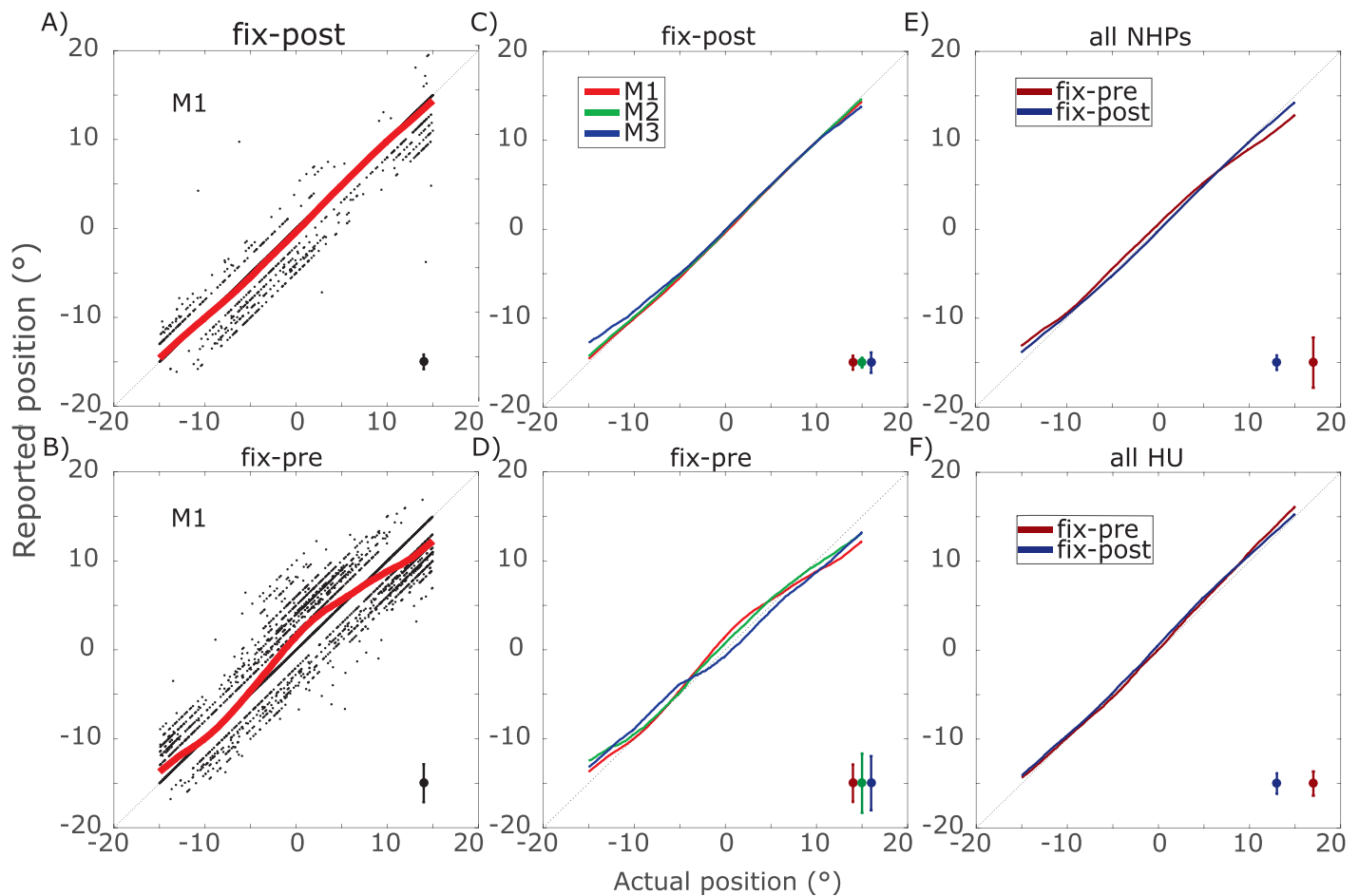


Figure 2. Fixation trial performance. (A) Performance of a single monkey subject (M1) on the fix-post trials. Each dot represents a trial, actual position is on the horizontal axis, and reported perceived position on the vertical axis. The solid line shows the average perceived location determined as a sliding average across trial. Error bars, shown in the bottom right of each panel, represent the standard deviation of the residuals (Materials and methods). (B) Performance of the same monkey subject (M1) on the fix-pre trials. Same conventions as in (A). (C) Average perceived location for fix-post trials in all three monkeys. (D) Same as (C), now for fix-pre trials. (E) Perceived location for fix-post and fix-pre trials averaged across three monkey subjects. (F) Same as (E), averaged over five human subjects. This figure shows that brief flashes were, on average, localized veridically when presented well before or after a saccade. The variance in localization across trials was typically larger in the conditions that required the integration of eye position information (fix-pre).

trained/instructed to report the location of a briefly flashed target by choosing an item from an array of possible positions (Figure 1).

Localization during fixation

In fixation trials, the flashed targets were presented long before (fix-pre) or after (fix-post) a saccade. In the fix-post trials, there was no saccade between target presentation and the subjects' choice. These were the easiest trials as illustrated by the performance in Figure 2. The subjects chose the correct location in the majority of trials (monkeys: 87% humans; 54%). Using a more lenient criterion for correct performance (an error less than 2°) changes these performance measures

to 91% correct (monkeys) and 81% correct (humans). These estimates of targeting errors in memory guided saccades are within the expected range based on previous reports (Gnadt, Bracewell, & Andersen, 1991; Opris, Barborica, & Ferrera, 2003).

In the fix-pre trials, the target was flashed before the saccade, and the subject reported this position after executing the saccade. On average, localization performance was still quite accurate in these trials (monkeys: 45% of trials with 0° errors, 54% of trials with $<2^\circ$ error; humans: 42% and 67%, respectively). We did not observe any consistent biases in localization when the targets were presented during fixation.

Figure 2 shows that there was more trial-to-trial variability in the fix-pre trials, especially in the monkeys. We quantified this with the standard

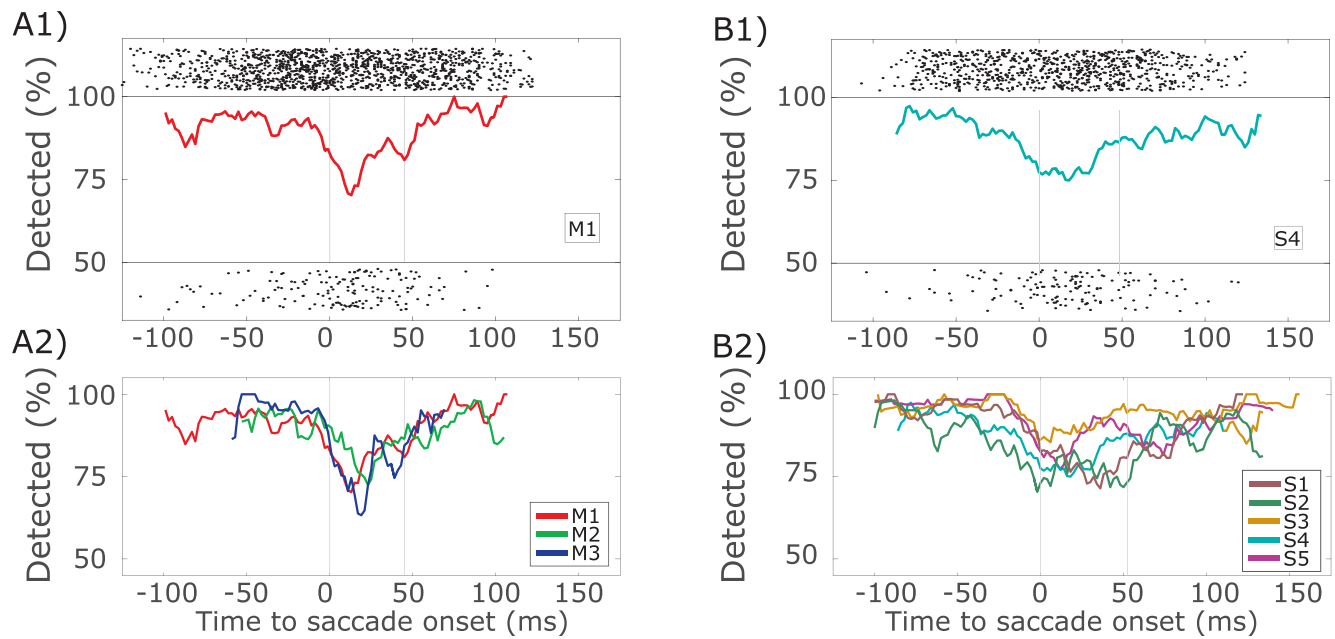


Figure 3. Perisaccadic detection performance. (A1) Results from an individual monkey subject (M1). Each dot represents a trial; each row a daily session. Dots in the top of the display represent likely detected targets (see main text for definition); dots at the bottom of the display represent undetected targets. The solid line (detectability curve) was calculated by determining the percentage of detections for each target presentation time (relative to saccade onset) using a 20-ms sliding window; data points are plotted at the center of the sliding window. (A2) Detectability curves for all monkey subjects for 15 cd/m^2 targets (monkey subjects color-coded as in Figure 2). (B1) Results for an individual human subject (S4), tested at the lower target contrast. Conventions as in Panel A1. (B2) Detectability curves for all human subjects for 8 cd/m^2 targets. Same format as Panel A2. Vertical lines in all panels mark average saccade start and end times of the subject or group (average 46 ms for monkeys, 53 ms for humans). This figure shows that both monkeys and humans are less likely to detect flashes presented around the time of saccade onset.

deviation of the data points around the moving average (i.e., variation around the solid lines in Figure 2). In the monkeys, this estimate of variability was 0.8° for the fix-post trials, and 2.8° for the fix-pre trials. In the humans, it was 1.2° and 1.4° , respectively. A two-way ANOVA of these variability estimates showed a significant effect of species, $F(1, 12) = 6.9$, $p = 0.02$; a significant effect of trial type, $F(1, 12) = 33.2$, $p <$

0.001 ; and a significant interaction, $F(1, 12) = 21.4$, $p < 0.001$.

Perisaccadic detection

In the perisaccadic time window, our first goal was to document whether detectability of flashed targets was affected by saccades. Targets were presented pseudorandomly above or below the horizontal midline, but the choice arrays were always presented in both locations (Figure 1). We used this to divide trials into undetected and (likely) detected targets. If the subject reported a perceived location on the incorrect array (i.e., top array when the target was presented in the bottom half, or bottom when the target was presented in the top half), we interpreted that as indicating that the target was undetected (and that the subject while guessing its vertical location, guessed wrong). Trials in which the subject chose the correct array (e.g., any location on the top array for a top half target location) were interpreted as a detection. (Given that random choices of top/bottom for truly undetected targets would lead the subject to choose the correct array on half of the trials, this estimate is necessarily

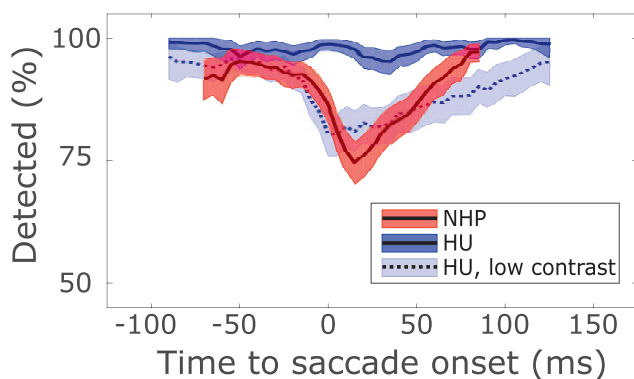


Figure 4. Average perisaccadic detection. Each curve shows the average perisaccadic detectability (i.e., the average of the data in Figure 3). Shading represents bootstrap estimates of the 95% confidence intervals. NHP: nonhuman primates; HU: humans.

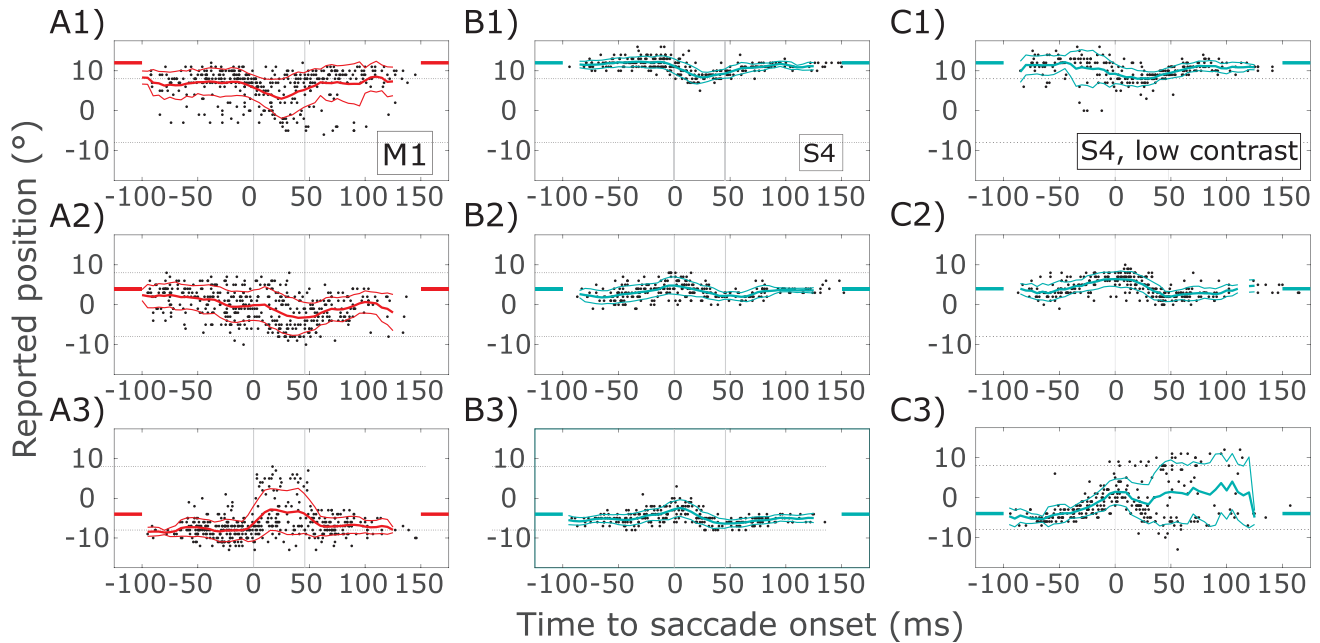


Figure 5. Perisaccadic localization in individual subjects. Each dot represents a trial. The horizontal axis corresponds to the presentation time of the flashed target (relative to saccade onset); the vertical axis is the reported perceived location. Thick lines show a moving average; thin lines represent the standard deviation around the moving average. Color-coding represents subjects. Each column corresponds to a subject and a target contrast (high/low). Each row corresponds to a target location (+12°, +4°, -4°). (A1) Monkey subject M1, high contrast, +12°. (A2) M1, high contrast, +4°. (A3) M1, high contrast, -4°. (B1) Human subject (S4), high contrast, +12°. (B2) S4, high contrast, +4°. (B3) S4, high contrast, -4°. (C1) S4, low contrast, +12°. (C2) S4, low contrast, +4°. (C3) S4, low contrast, -4°. Both the monkey and the human subject show perisaccadic mislocalization, but for the +4° target (middle row), the direction of mislocalization was opposite.

only an upper bound on detection. For ease of notation, we nevertheless refer to this as detection).

Qualitatively, the fraction of detected targets decreased as the presentation time approached saccade onset; detectability was lowest for targets presented during the saccade (Figure 3A). Quantitatively, we compared the monkeys' detection performance in the presaccadic [-80, -40] time window to the three subsequent 40-ms windows. Detection probability was significantly lower in the time windows during ([0, 40]) and immediately after the saccade ([40, 80]; Fisher's exact test; $p < 0.02$). The difference with the window immediately before the saccade ([-40, 0]), however, was not statistically significant ($p = 0.34$).

Somewhat surprisingly, human subjects' detection performance in the identical setup, using identical visual stimuli, was nearly 100% irrespective of target presentation time (Fisher exact tests, all $p > 0.69$, individual data not shown). To determine whether this was merely a reflection of lower contrast thresholds in the human subjects, we repeated the experiment in the human observers at a lower luminance of the flashed stimuli (8 cd/m² instead of 15 cd/m²). Detectability of the lower contrast targets was significantly reduced in all time windows compared to the [-80, -40] time window (Fisher's exact test; $p < 0.001$).

To summarize these results, we averaged the detectability curves per species and contrast condition. Figure 4 shows that the saccade did not affect the human subjects' detection of the high contrast targets while it reduced detection rates to ~75% for the low contrast targets. In the monkeys, detection of the high contrast targets was similarly reduced to ~75% (monkeys did not perform the experiment at low contrast).

Perisaccadic localization

We analyzed the reported location of the flashed targets as a function of their presentation time relative to saccade onset. In the example data sets shown in Figure 5, the saccades were rightward and the targets were flashed at -4°, +4°, or 12°. Well before or after the saccade, flashes were localized fairly close to their veridical position (indicated by the horizontal bars in the plots).

The figure is organized to allow a direct comparison of a single monkey subject (Column A), a human subject localizing targets with the same high contrast (Column B), and the same human subject localizing targets at the lower contrast (Column C). The rows

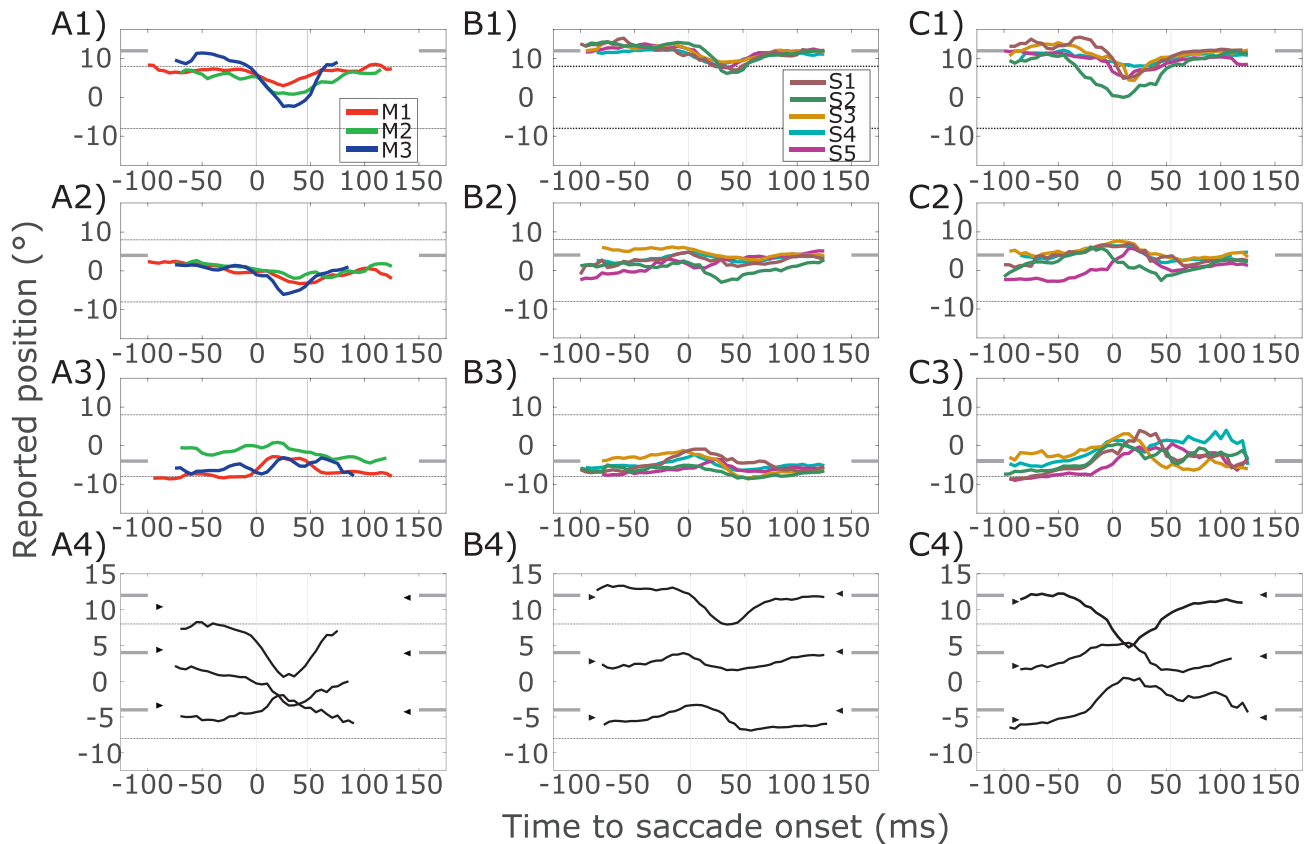


Figure 6. Perisaccadic mislocalization. (A1–A3) Average localization curves for three monkey subjects using the high luminance target at locations $+12^\circ$ (A1), $+4^\circ$ (A2), or -4° (A3). (B1–B3) Average localization curves for five human subjects, using the high luminance target. (C1–C3) Average localization curves for five human subjects, using the low luminance target. Conventions as in Figure 5. (A4) Group average localization curve for monkeys subjects using high luminance targets. (B4) Group average localization curves for human subjects using high luminance targets. (C4) Group average localization curves for human subjects using low luminance targets. Horizontal bars show veridical target location, triangles show the (average) perceived location during fixation (fix-pre on the left, fix-post on the right axis). This figure shows that both humans and monkeys showed perisaccadic mislocalization, but that the spatial pattern differed qualitatively.

represent the physical target locations. Comparing Columns A and B shows that both subjects showed clear mislocalization. The spatial pattern of mislocalization, however, appeared to differ between the human and monkey subject. Consider first the results from the example human subject (Panels B1, B2, and B3). Panel B1 corresponds to targets flashed beyond the saccade target (i.e., at 12°). Around the time of the saccade these were mislocalized towards the saccade target ($+8^\circ$). Panels B2 and B3 shows the data for targets flashed at $+4^\circ$ and -4° , respectively. These were also mislocalized towards the saccade target at $+8^\circ$. Taken together these data reflect the expected perisaccadic compression of space towards the saccade target.

The monkey subject showed a qualitatively similar pattern of mislocalization for flashes at $+12^\circ$ (Panel A1) and -4° (Panel A3), but the mislocalization of the target at -4° (Panel A2) was away from the saccade target. We will refer to this as backward mislocalization

to contrast it with the more typical mislocalization in the direction of the saccade—forward mislocalization.

As discussed above, monkey subjects detected only $\sim 75\%$ of the 15 cd/m^2 targets when presented close to saccade onset, while human subjects detected nearly 100% (Figure 4). It is conceivable that these differences in detectability caused differences in localization. To address this, Figure 5C1–C3 shows the localization data for the low contrast targets, which were approximately matched in detectability (Figure 4). As expected, mislocalization was more pronounced at the lower level of target contrast (Michels & Lappe, 2004), but the spatial pattern did not change. More specifically, unlike the monkey subject in Figure 5A2, the human subject still mislocalized the $+4^\circ$ target in the direction of the saccade (Figure 5C2), and the focus of the perisaccadic compression remained the saccade target.

Figure 6 shows the analogous results for all subjects. There was a marked difference between species for one of the target locations between fixation and saccade

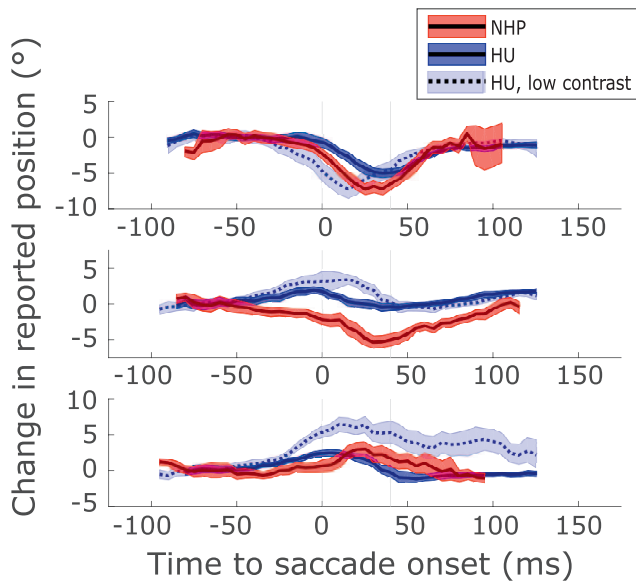


Figure 7. Bias-corrected perisaccadic mislocalization. (A) Mislocalization of targets $+12^\circ$ targets. (B) Mislocalization of $+4^\circ$ targets. (C) Mislocalization of -4° targets. Shaded area shows 95% confidence limits. NHP: nonhuman primates; HU: humans.

target (Panels A2, B2, and C2). All three monkeys showed backward mislocalization for these target locations, while the human subjects showed forward mislocalization. Lowering the contrast for the human subjects (and thus approximately matching the detectability of the stimuli across species) increased this difference in the pattern of mislocalization (right column).

The raw reported locations used in Figure 6 show that individual subjects (of either species) had substantial mislocalization even for flashes presented as early as 100 ms before or as late as 100 ms after saccade onset. To obtain a clearer picture of the average mislocalization that is time-locked to the saccade, we removed these biases per subject, and then averaged across subjects (Materials and methods).

This normalization (Figure 7) allows a direct comparison of perisaccadic mislocalization across species under physically matched conditions (red: monkeys; dark blue: humans) or detectability-matched conditions (red: monkeys; light blue: humans). Qualitatively speaking, the patterns of mislocalization were substantially similar, especially for the $+12^\circ$ target location (Figure 7A), and the -4° target location (Figure 7C). However, there were also notable differences in magnitude and timing of the effects. The largest qualitative difference was found for the $+4^\circ$ target location: Human subjects mislocalized this in the forward direction, while monkey subjects mislocalized in the backward direction. The 95% bootstrap confidence limits (shaded areas surrounding the curves) allow the reader to assess by eye which differences are

unlikely to be caused by random variation in subject responses.

We also analyzed these data with an ANOVA with fixed effects of time (using the same four time bins as in the analysis of perisaccadic detection), target position, and species, and subjects as a random effect. We performed these ANOVAs separately for the physically matched contrasts and the detectability-matched contrast. The significant interaction of time and position confirmed the presence of perisaccadic mislocalization (physically matched: $F[6, 4474] = 15.47$, $p < 0.001$; detectability-matched: $F[6, 4215] = 12.50$, $p < 0.001$). The claim that the pattern of mislocalization was significantly different in humans and monkeys was supported by the main effect of species (physically matched: $F[1, 4474] = 6.54$, $p = 0.020$; detectability-matched: $F[1, 4215] = 13.86$, $p = 0.005$), and the interaction of target position and species which was a trend for physically matched targets, $F(2, 4474) = 2.90$, $p = 0.082$, and a significant effect for detectability-matched targets, $F(2, 4215) = 4.12$, $p = 0.031$.

Discussion

Our experiments showed that monkeys are less likely to detect stimuli presented around the time of a saccade (saccadic suppression), and systematically misreport the location of such stimuli (perisaccadic mislocalization). The pattern of perisaccadic mislocalization in monkeys included both shifts in and against the direction of the saccade, but it was qualitatively different from that observed in humans under the same conditions.

We compare our main results with previous studies investigating perisaccadic perception in monkeys and humans and discuss how each of these findings serves to inform our understanding of the underlying neural mechanisms.

Perisaccadic reductions in detectability

A reduction in stimulus detectability at the time of rapid eye movements has previously been demonstrated in monkeys for stimuli presented at the onset of the fast-phase of the optokinetic nystagmus (Mohler & Cechner, 1975), and after the onset of a microsaccade (Hass & Horwitz, 2011). Our study adds that detectability at the time of instructed, large saccades is also reduced in monkeys. Under physically identical conditions, detectability was not reduced in our human subjects; only after reducing target luminance did we obtain a similar pattern of reduced detectability. A parsimonious interpretation of these findings is that the

influence of a saccade was qualitatively similar in both species, but that the human subjects had higher contrast sensitivity that allowed them to detect stimuli that would be below threshold in the monkeys. Note that human subjects typically spent less time in the recording booth than the monkeys, hence differences in contrast adaptation cannot account for these differences, as they would predict higher, not lower thresholds in the human subjects.

On average, the trough of reduced detectability coincided with saccade onset in the human subjects, while it was ~ 20 ms later in the monkeys. Given the variability across subjects (Figure 3) and the resulting uncertainty associated with the averages (shading in Figure 4), this difference should not be overemphasized. In fact, the timing of reduced detectability is quite variable across studies. Some of this variation may be attributable to factors such as the contrast of the target, the background luminance, and the retinal location of the target, but most studies also show substantial (within-species) individual differences in the timing of the trough of detectability (e.g., Majj, Brenner, & Smeets, 2011; Michels & Lappe, 2004).

Neural mechanisms of detection

Multiple factors likely contribute to the changes in detectability around the time of saccades. First, the small stimuli flashed during a saccade generate motion signals (on the moving retina) with spatiotemporal properties for which even visual neurons specialized for motion have low sensitivity (e.g., Krekelberg & van Wezel, 2013; Nover, 2005). Second, shearing forces in the retina generated by the rapid acceleration of the eye could further reduce sensitivity to light (Castet, Jeanjean, & Masson, 2001, but see Ross, Morrone, Goldberg, & Burr, 2001). Third, behavioral experiments show that backward masking by the postsaccadic scene strongly reduces detectability of intrasaccadic visual input (Campbell & Wurtz, 1978; Castet, Jeanjean, & Masson, 2002; García-Pérez & Peli, 2011). At the neural level, backward masking has been attributed to the interaction between the V1 response to the mask and the transient off-response to the target (Macknik & Livingstone, 1998). Whether this neural mechanism can fully account for reduced detectability at the time of saccades, however, has not yet been investigated. Fourth, across the visual hierarchy, changes in neural sensitivity have been documented that cannot be explained by either of the first three factors, because they occur in complete darkness (Kagan, Gur, & Snodderly, 2008), or well before the eyes start to move (Bremmer, Kubischik, Hoffmann, & Krekelberg, 2009; Rajkai et al., 2008). Assuming that any change in the response properties of visual neurons potentially

contributes to changes in detectability, these findings support the view that so-called central or extraretinal signals contribute to perisaccadic changes in detectability. Here too, a direct link between specific neural and behavioral changes is lacking (for review, see Ibbotson & Krekelberg, 2011). In the future, we plan to use the paradigms developed here to perform combined behavioral and electrophysiological studies that provide more specific insight into which of these mechanisms (or their interaction, Ibbotson & Cloherty, 2009) dominates perisaccadic perception.

Perisaccadic mislocalization

Two previous studies investigated perisaccadic localization in monkeys. The first showed uniform forward mislocalization (Dassonville et al., 1992), the second uniform backward mislocalization (Jeffries et al., 2007). Our study shows a mixture of forward and backward mislocalization that depends on the location of the stimulus on the screen.

In humans, the presence of visual references tends to evoke a compression mislocalization, while flashes in complete darkness are typically mislocalized in the forward direction (Lappe, Awater, & Krekelberg, 2000). This fits with the difference between the Dassonville et al. (1992) study, which was performed in complete darkness, and ours, which used a continuously lit monitor that provided a spatial frame of reference. The discrepancy with the Jeffries et al. (2007) study, however, is more difficult to explain.

The main difference with our design appears to be that Jeffries et al. (2007) used flashes with a longer duration. To address this, we performed a control experiment in M3 that matched the main experiment except for the use of 100-ms target flashes. We found no evidence of mislocalization; the animal subject reported location veridically throughout the perisaccadic period (data not shown). This veridical localization of long duration stimuli is consistent with previous studies in humans (Honda, 2006; Schlag & Schlag-Rey, 1995), although others have reported uniform forward mislocalization for long-duration flickering stimuli (Watanabe, Noritake, Maeda, Tachi, & Nishida, 2005).

Another difference is that Jeffries et al. (2007) used the first saccade to the flash as the animal's report of perceived position. In our main analysis, we followed Dassonville et al. (1992) and used the final eye position—often the result of two or more saccades—to quantify the animal's perceived flash position. Reanalyzing our data using the first saccade landing position as the animal's report, however, resulted in mislocalization curves highly similar to those in Figure 6, and not a uniform backward mislocalization. In summary, we cannot explain the discrepancy with the Jeffries et

al. study. This leaves us with the hypothesis that multiple subtle differences in the visual display, the training procedure, or intersubject differences, may have led to the different outcomes.

The most salient localization difference between human and monkey subjects was the direction of mislocalization of the $+4^\circ$ target. Humans consistently mislocalized this in the saccade direction, while all three monkeys mislocalized it against the direction of the saccade. Are there uncontrolled factors that could cause this difference? First, the detectability of targets could affect the pattern of mislocalization (Michels & Lappe, 2004). However, we observed the same qualitative difference between species for targets that were physically matched or (approximately) matched in detectability. Second, subtle differences in laboratory layout (e.g., visual references; Lappe et al., 2000) are known to affect perisaccadic localization. By letting human and monkey subjects perform the experiments in the same physical setup we avoided this potential confound. Third, monkeys worked for juice for many months, while humans volunteered only a few hours of their time. Therefore, motivation and its associated attentional state may well have differed between species. This could contribute to the different levels of accuracy in the fix-pre trials (Figure 2). However, because liquid reward was independent of the location of the target (and independent of the animals, report on perisaccadic trials), there seems little reason to assume that this could affect perisaccadic localization in a position-dependent manner. Based on these arguments we conclude that the difference in perisaccadic localization was a true difference between the monkey and human subjects in our experiments.

Neural mechanisms of localization

Spikes reaching the brain from the retina encode spatial information relative to the position of the eye, and this eye-centered neural representation is maintained across much of striate and extrastriate cortex (Hartmann, Bremmer, Albright, & Krekelberg, 2011; Ong & Bisley, 2011). Because the eyes move relative to the head, the body, and arms, the brain must consider these movements to act upon the visual input. In our experiments, subjects localized targets on a monitor, which was stationary with respect to the head but not with respect to the eye. To compute position information with respect to the head/monitor, the visual system must combine the information on the position of the target with respect to the eye that arrives from the retina with information on the position of the eye with respect to the head. Perisaccadic mislocalization has been used to generate insight into the neural signals used in this computation.

There are three conceptually different viewpoints that aim to explain perisaccadic mislocalization. The first two—attentional boosting and receptive field remapping—attribute mislocalization primarily to the errors in the neural representation of retinal location, while the third—eye position coding—attributes it to errors in the neural representation of eye position.

The attentional boost model hypothesizes that the excitability of neurons with receptive fields near the saccade target increases at the time of saccades. The goal of this increase is to improve processing at this newly important location in space (Hamker, Zirnsak, Calow, & Lappe, 2008; Hamker et al., 2011). Assuming a code in which each neuron votes for a retinal position in proportion to its firing rate, these neurons would cast a stronger vote, and thereby bias localization towards the saccade target. Data obtained in the middle temporal and medial superior temporal area (Krekelberg, Kubischik, Hoffmann, & Bremmer, 2003), area V4 (Tolias et al., 2001), and the frontal eye fields (Zirnsak, Steinmetz, Noudoost, Xu, & Moore, 2014) are consistent with such perisaccadic distortions of the neural representation. However, the model and the neural data predict that compression should be towards the saccade target; this does not appear to be the case in our monkey subjects.

The remapping model relates perisaccadic mislocalization to the temporary shift of receptive field locations that occurs around the time of a saccade in some parietal neurons (Duhamel, Colby, & Goldberg, 1992; Nakamura & Colby, 2002). In this model too, a spike is interpreted as a vote for a stimulus at the location of the neuron's receptive field during fixation, even when that receptive field is temporarily “re-mapped” somewhere else. For a neuron with a receptive field that is remapped in the direction of the saccade, this predicts that mislocalization should always be against the direction of the saccade because a flash in the remapped receptive field results in a spike that is decoded as a vote for the fixation receptive field. This model could account for the (Jeffries et al., 2007) result, but it is not consistent with our findings in monkeys or humans, or previous studies in humans (Honda, 2006; Schlag & Schlag-Rey, 1995; Watanabe et al., 2005).

The eye position model attributes perisaccadic mislocalization to temporary errors in the neural representation of eye position (Dassonville et al., 1992; Honda, 1989; Teichert et al., 2010) or a veridical eye position signal that is read out at the wrong time due to uncertainty about the presentation time of the target (Brenner, van Beers, Rotman, & Smeets, 2006; Majaj et al., 2009, 2011). Specifically, if the eye position signal is a damped version of the true eye position signal, this model predicts the biphasic patterns of mislocalization found in humans and monkeys (Dassonville et al.,

1992). In support of this, neural representations of eye position in parietal and extrastriate cortex indeed look like a damped version of the true eye position (Morris, Bremmer, & Krekelberg, 2013, 2016; Morris, Kubischik, Hoffmann, Krekelberg, & Bremmer, 2012). In its original form, however, this model cannot account for the compression pattern of mislocalization (e.g., Figure 6).

However, variants of this model can generate both forward and backward mislocalization depending on the latency and persistence of the signals representing the retinal input and the eye-position (Bischof & Kramer, 1968; Pola, 2004). Pola (2011) showed formally that assuming different dynamics at different retinal locations could account for perisaccadic compression of space towards the saccade target. With this assumption, however, the model could account equally for compression towards any location on the screen. Testing this hypothesis requires the measurement of the dynamics of visual and eye position signals across neurons that represent different locations of the retina.

In summary, the attention boosting and remapping models cannot account for the complexity of mislocalization we observed in our monkey subjects, while an extension of the eye position model can, at least in principle. An important caveat to this discussion is that the mismatch between the behavioral data and the attention boosting and remapping models only shows that they are incomplete, not that they are wrong. Put differently, given that all three underlying mechanisms (attentional boosting, receptive field remapping, and eye position signals) have substantial experimental support, it is not difficult to imagine that all three, and possibly others, contribute to mislocalization (Mohsenzadeh, Dash, & Crawford, 2016). The multitude of contributing factors, and the complexity of the computations that must be completed with each eye movement could explain why there is large intersubject variability, why the patterns of mislocalization are highly sensitive to extraneous visual elements in the display, and why they vary substantially even across apparently similar experiments.

Keywords: eye movements, saccade, position perception, saccadic suppression, perisaccadic mislocalization

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