Activity of neurons in the lateral intraparietal area of the monkey during an antisaccade task

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The close relationship between saccadic eye movements and vision complicates the identification of neural responses associated with each function. Visual and saccade-related responses are especially closely intertwined in a subdivision of posterior parietal cortex, the lateral parietal area (LIP). We analyzed LIP neurons using an antisaccade task in which monkeys made saccades away from a salient visual cue. The vast majority of neurons reliably signaled the location of the visual cue. In contrast, most neurons had only weak, if any, saccade-related activity independent of visual stimulation. Thus, whereas the great majority of LIP neurons reliably encoded cue location, only a small minority encoded the direction of the upcoming saccade.

A class of rapid, brief eye movements—saccadic eye movements—is specialized for visual exploration in foveate animals. Although most often directed to visual or other sensory targets, saccades can also be generated at will, independently of direct sensory guidance. A clear experimental demonstration of the independence of the visual and saccadic systems is provided by the 'antisaccade' task, in which humans[1,2] and monkeys[3], make saccades to featureless, unmarked locations opposite a salient visual stimulus, in accordance to task instructions. The performance of non-stimulus-bound saccades in this and other situations is thought to depend on cortical, particularly frontal, structures[2,4].

In the monkey, a posterior parietal area, the lateral intraparietal area, is also thought to contribute to the planning of saccades. Anatomically[5–7] and physiologically, this area lies at a junction between the visual and saccadic systems. LIP neurons respond to salient or behaviorally relevant visual stimuli at restricted retinotopic locations (response fields), independently of saccade execution[8,9]. They also respond before saccades toward visible and, to a lesser extent[9,10], those toward remembered visual targets within their response field[11,12]. Unfortunately, most previous studies have not tested saccades when there is a conflict between the saccade goal and the spatial location of the cue specifying that goal. It has therefore remained unclear whether these presaccadic responses underlie a sensory-independent saccade-planning mechanism[13] or visual or attentional processes that normally precede saccades to visual targets[14].

In this study, we separated the visual and saccade-planning activity of LIP neurons using the antisaccade task. Monkeys were trained to execute, on interleaved trials, saccades toward a visual cue (prosaccade) or toward the opposite, unmarked location (antisaccade), in accordance with a centrally presented instruction. The vast majority of LIP neurons responded to the appearance of the cue in their response fields, regardless of the saccade dictated by that cue. However, only a minority became active before antisaccades to their response fields, although they did respond before prosaccades of equivalent metrics. Thus the vast majority of LIP neurons accurately encode the location of visual stimuli, but do not always reflect the formation of saccade motor plans independently of the confines of the immediate visual input. A report of these results has appeared in abstract form (J.G. & M.E.G., *Soc. Neurosci. Abstr.* 23, 14.9, 1997).

Results

We describe the activity of 105 neurons isolated from area LIP in two hemispheres in two monkeys. Each neuron responded significantly above baseline during at least one epoch—cue, delay or presaccadic—of the memory-guided saccade task (delayed-prosaccade trials).

Encoding of cue location and saccade direction

Neural activity was recorded during four types of prosaccade and antisaccade trials (**Fig. 1**). We first asked whether neurons responded more reliably as a function of cue location or saccade direction. Virtually all neurons encoded the location of the visual cue during the initial portion of the reaction time (**Fig. 2**): they responded to appearance of the cue in their response field, regardless of whether the impending saccade would be directed in their null or optimal direction. After this initial response, the activity pattern became more variable from cell to cell. Some neurons had no activity during the later portion of the reaction time (neuron A). More commonly, neurons were active before the saccade, although most did not reliably encode saccade direction during this epoch. Some neurons (such as neuron B) were active only if both cue and saccade goal were in their response field (V^+M^+ trials) and did not respond on any other trial type. Others, like neuron C, were active if the cue or the saccade goal, or both, were in their response fields (V^+M^+, V^+M^- and V^-M^+ trials). Only a minority of neurons, like neuron D, unambiguously specified the direction of the upcoming saccade. These cells responded during the presaccadic epoch only if the saccade was in their optimal direction, regardless of cue

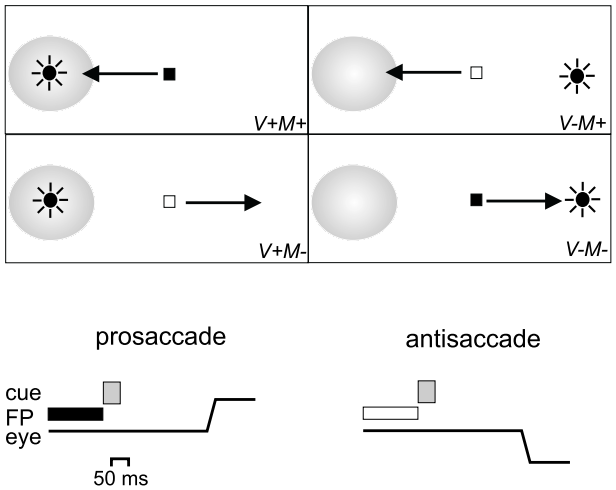

Fig. 1. The prosaccade/antisaccade task. Top, spatial locations of the cue (black circle) and saccade (arrow) in the four immediate trial types, for a putative neuron with response field to the left of fixation (shaded oval). Bottom, time course of task events on a prosaccade and an antisaccade trial.

location (V^+M^+ and V^-M^+ trials), and did not respond to a stimulus dictating a saccade away (V^+M^- trials).

To assess the reliability of the cue- and saccade-related signals, we calculated the information transmitted by each neuron's firing rate about the two possible cue locations and saccade directions, during early and late portions of the reaction time. Transmitted information estimates how consistently a particular response is correlated with a particular variable, independently of others. During a cue interval (40–160 ms after cue onset), neurons A–D in Fig. 2 transmitted 0.32, 0.21, 0.15 and 0.10 bits of information about cue location, with 1 bit being the maximum possible. During the presaccadic epoch (60 ms before saccade beginning), neuron D transmitted 0.26 bits about saccade direction, compared with only 0.05, 0.09 and 0.05 bits by neurons A, B and C.

The vast majority of neurons encoded the location of the cue during the cue epoch much more reliably than they did the direction of the saccade during the presaccadic epoch (**Fig. 3a**). The median instantaneous information transmitted about cue location during the early epoch was 0.15 bits, compared with a median of only 0.04 bits transmitted about the saccade in the presaccadic epoch ($p < 10^{-10}$). Information transmitted about the cue declined during the presaccadic epoch to a median of 0.05 bits, slightly more than the information transmitted about the saccade during the same interval (**Fig. 3b**; $p = 0.052$). Thus, although the vast majority of LIP neurons reliably encoded the locus of visual stimulation in their initial response, only a minority could indicate the direction of the saccade, even at the time of its initiation.

Target selection versus movement selection

Several previous studies have shown that LIP neurons can encode saccade direction in certain circumstances. Following presentation of a visual stimulus in their response field, the sustained activity of many LIP neurons is selective for the direction of the next intended saccade[9,12,17,18], as suggested in the present results also (**Fig. 2**). Neurons B–D had stronger presaccadic activity if the monkey prepared a saccade to the cue in their response field (V^+M^+ trials) than if the same cue dictated a saccade away (V^+M^- trials). However, this appeared critically dependent on the saccade target and did not consistently reflect the planning of the saccade itself: neurons B and C were not selective for the direction of saccades made without direct visual guidance in V^-M^+ versus V^+M^- antisaccade trials.

To determine if this was a general characteristic of our sample, we measured the modulation of the presaccadic activity by saccade direction in trials in which the cue was in the response field (V^+M^+ versus V^+M^- trials). We defined the contrast index $(R_{V+M+} - R_{V+M-})/(R_{V+M+} + R_{V+M-})$, where R is a neuron's presaccadic response on each trial type. An index of 1 represents a neuron perfectly selective for the saccade or, equivalently, for the saccade target in its response field, and an index of –1 indicates a neuron that responds only to a cue that dictates a saccade away. To determine whether this saccade-related modulation represented saccade motor planning, we compared it with the neurons' selectivity for the direction of an antisaccade (V^-M^+ versus V^+M^- trials). We defined an antisaccade selectivity index as $(R_{V-M+} - R_{V+M-})/(R_{V-M+} + R_{V+M-})$. Here a value of 1 represents a neuron perfectly selective for a saccade to its response field in the absence of a target and an index of –1, as above, a neuron that responds only for the cue dictating a saccade away. If neur-

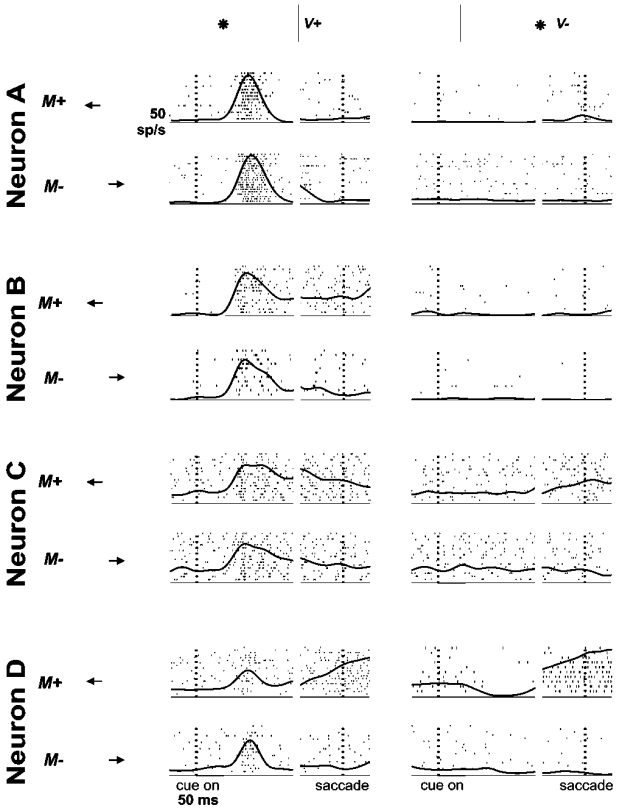

Fig. 2. The activity of four neurons on no-delay prosaccade and antisaccade trials. Trials are shown in the same format as in Fig. 1, with those in which the cue appeared in the response field (V^+) on the left and those in which the saccade goal was in the response field (M^+) on the top row for each neuron. For each trial type, activity is aligned first on the onset of cue presentation (gray horizontal bar) and again on the onset of the saccade. Average spike density histograms (solid lines) are superimposed on raster displays showing the activity on consecutive correct trials (first trial at the bottom).

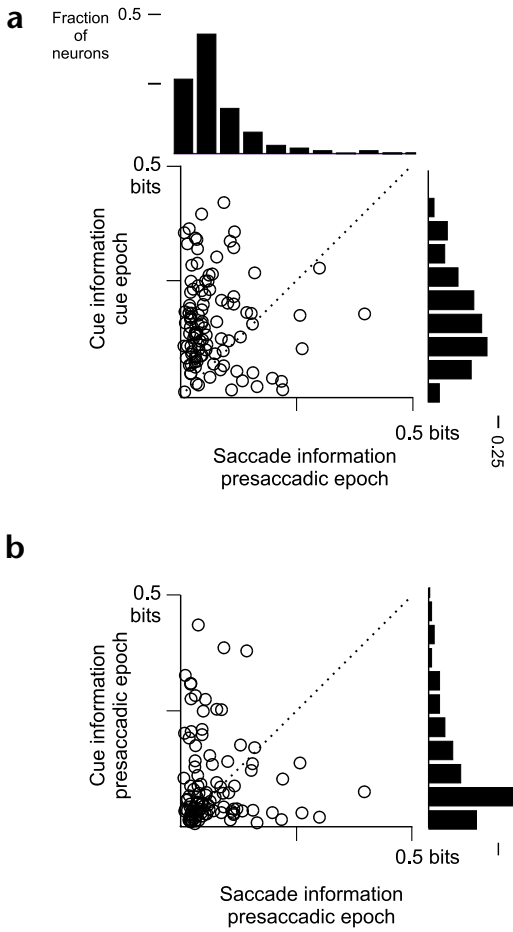

Fig. 3. Information transmitted about cue location and saccade direction across the entire sample (105 neurons). Information transmitted by each neuron during the presaccadic epoch (abscissa) is compared with the information transmitted about cue location during the cue epoch (**a**) and during the presaccadic epoch (**b**). All values represent instantaneous information averaged across the interval of interest (40–160 ms after cue onset, or the 60-ms epoch preceding saccade initiation). Histograms show the distribution of values along the abscissa and ordinate (bin size, 0.05 bits).

al activity reflected the planning of the saccade, neurons should have comparable selectivity on V^+ trials and on antisaccade trials. If, on the other hand, activity reflected the selection of a saccade target, neurons should be much more selective on V^+ trials, in which the cue appeared consistently in the response field, than on antisaccade trials.

Neurons were much more selective for saccade direction on V^+ trials than on antisaccade trials (**Fig. 4**; $p < 10^{-12}$). The median selectivity index, 0.18, was significantly greater than zero ($p < 10^{-7}$; abscissa and top histogram). In contrast, the median index on antisaccade trials, –0.09, was not different from zero ($p = 0.065$), and the entire distribution was shifted toward negative values, corresponding to no or opposite directional selectivity on antisaccade trials. Thirteen neurons (13/105, 12%) did have activity consistent with a saccade motor plan (shown in gray in **Fig. 4**). Like neuron D in Fig. 2, these neurons responded significantly more ($p < 0.025$) before prosaccades and before antisaccades to their response field than before antisaccades in the null direction (on V^+M^- trials).

Sustained visual and saccade-related activity

The results above suggest that the sustained activity of many neurons was strongly influenced by presentation of a visual target, as it was stronger on prosaccade than on antisaccade trials. To determine if the target's influence persisted at longer delays following its extinction, we compared activity preceding prosaccades and antisaccades to the response field on delay and no-delay trials.

Presentation of a visual target significantly enhanced activity across the sample at both short and long delays following extinction of that target (**Fig. 5**). Although the visual influence declined slightly with time (note the smaller effect of the cue during the presaccadic epoch relative to the delay epoch of delay trials), it remained significant throughout the time-span tested here. Of the neurons active before prosaccades, more than half responded significantly less before antisaccades (in the presaccadic epoch of no-delay trials, 42/64, 65%; in the delay epoch, 39/57, 68%; in the presaccadic epoch of delay trials, 23/44, 52%). In some of these neurons, the entire delay and presaccadic response—including in some cases activity bursts time-locked to saccade onset—were target dependent (**Fig. 6a**).

The sustained visual response continued unsuppressed even while the monkey prepared an antisaccade in the null direction, on no-delay V^+M^- trials. Across the sample, the presaccadic

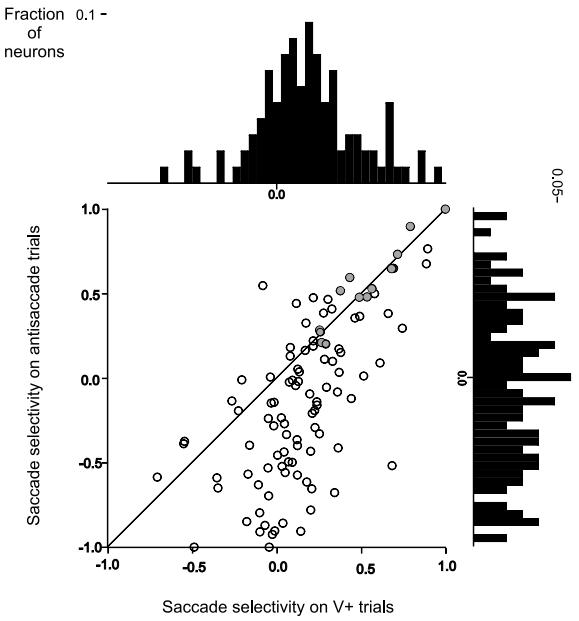

Fig. 4. Selectivity of the presaccadic activity for saccade direction in trials in which the cue appeared in the response field (abscissa) and in antisaccade trials (ordinate) for all 105 neurons. Positive indices indicate greater activity for saccades to the response field than for those directed away, negative indices show greater activity for saccades in the null direction, and indices of zero show no selectivity. (See text for details.) Gray symbols indicate neurons that had significant modulations for both measures. Histograms show the distribution of values along the abscissa and ordinate (bin size, 0.05).

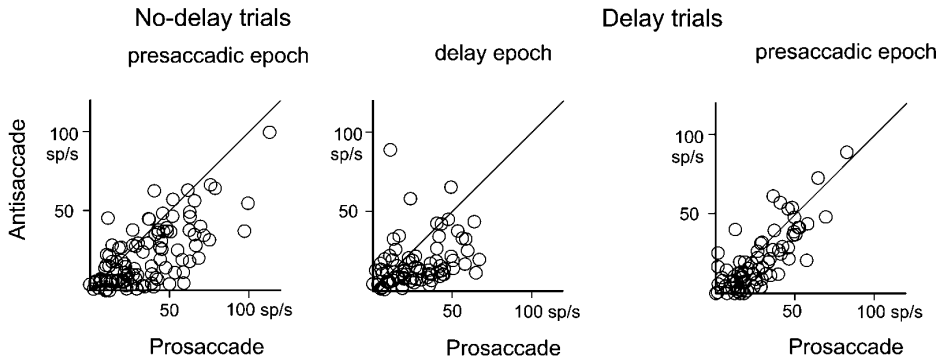

Fig. 5. Comparison of activity preceding prosaccades and antisaccades for the entire sample, for no-delay trials (105 neurons) and delay trials (79 neurons). Each data point represents the average firing rate of one neuron in the specified interval. Activity is greater in prosaccade than in antisaccade trials during the presaccadic epoch of no-delay trials ($p < 10^{-10}$), during the delay epoch of delay trials ($p < 10^{-12}$) and during the presaccadic epoch of delay trials ($p < 10^{-11}$).

response on no-delay V^+M^- trials was equivalent to ($p = 0.29$) and highly correlated with ($r = 0.76$, $p < 10^{-13}$) the delay activity measured during an analogous time interval on prosaccade trials. (This time window was defined, for each neuron, as the 50-ms epoch ending at the mean latency of saccades on V^+M^- trials.)

Approximately one quarter of all neurons tested had visually independent saccade-related activity. In particular, 30/105 (28%) responded above baseline before antisaccades to their response fields on no-delay V^-M^+ trials and 19/79 (24%) on delay V^-M^+ trials (for example, **Figs. 6b** and **1c** and **d**). This saccade-related activity was equivalent on immediate and delay antisaccade trials, showing that it was not significantly influenced by the requirement to remember a saccade motor plan (for presaccadic activity, $p = 0.14$ and $r = 0.89$; $n = 19$). Activity was also similar during the delay and presaccadic epochs of delay antisaccade trials ($p = 0.056$ and $r = 0.74$; $n = 19$).

Of neurons with saccade-related activity, slightly more than half also had sustained visual responses, as shown by their significantly higher firing rates before prosaccades than before antisaccades (for example, **Figs. 6b** and **1c**; 17/30, 57%, showed this difference on no-delay trials and 10/19, 52%, on delay trials). On no-delay antisaccade trials, these neurons had presaccadic activity when either the cue or saccade goal was in their response fields (V^+M^- and V^-M^+ trials) and consequently transmitted minimal information about saccade direction (mean, 0.04 bits about saccade direction versus 0.1 bits about cue location; $p = 0.048$, $n = 17$). The 13 remaining neurons had only saccade-related and no visual activity in the presaccadic epoch and transmitted more information about saccade direction than about cue location in this epoch (mean, 0.15 versus 0.04 bits; $p = 0.045$).

Quantitative analysis showed that neither the greater endpoint scatter nor the reduced velocities of antisaccades relative to prosaccades could account for the differences in neural activity (see Methods and eye movement records in **Fig. 6**). We wondered, however, whether the spatial profile of the neurons' activity might have varied between prosaccades and antisaccades. To examine this possibility, we tested 13 neurons with delayed prosaccades and delayed antisaccades spanning a wider range of locations (± 5° to ± 10° surrounding the center of the response field along both horizontal and vertical axes). The majority (11/13) did not respond before antisaccades, whether they had responded or not at the analogous locations on delayed prosaccade trials. The two remaining neurons responded equivalently at all locations before delayed prosaccades and antisaccades. Thus antisaccades were associated with overall lowered neural activity, without striking changes in the spatial properties of the response.

Saccade influences on the initial cue-evoked response

Although the neurons' initial response was primarily determined by the location of the cue, we noticed that many neurons tend-

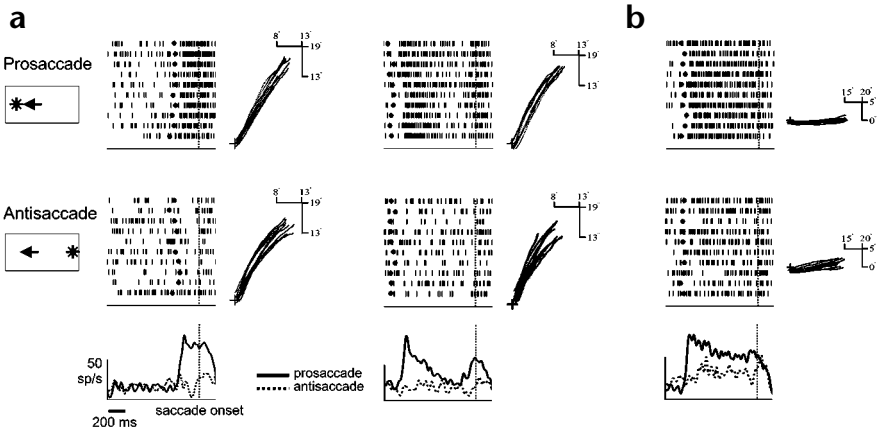

Fig. 6. Dependence of delay period and presaccadic activity on direct visual guidance, for two neurons. (**a**) Activity of one neuron on no-delay (left) and delay (right) trials, for prosaccades (top) and antisaccades (bottom). The trial-by-trial spike train and trajectories of the corresponding saccades (each with 1-ms resolution) are shown for each trial type. Raster lines are sorted in order of saccade accuracy, with the most accurate at the top (Methods). Black dots indicate cue onset. Bottom, superimposed spike density histograms corresponding to the rasters above. (**b**) Activity of a second neuron on delay prosaccade and antisaccade trials; same format as in (**a**). This neuron's activity on no-delay trials is shown in Fig. 2c. Both neurons were recorded using fixed delay intervals of 700 ms following extinction of the cue. Average presaccadic firing rates for neuron in (**a**) were 20 spikes/s on antisaccade trials and 58 spikes/s on prosaccade trials. During the delay period, neuron in (**b**) had an average firing rate of 33 spikes/s on antisaccade trials and 55 spikes/s on prosaccade trials. During the presaccadic epoch, its firing rates were 29 and 45 spikes/s, respectively.

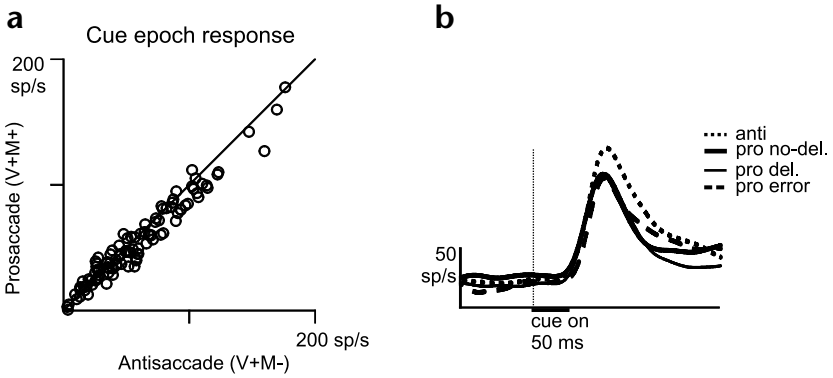

Fig. 7. Comparison of the cue-evoked response on antisaccade and prosaccade trials for all 105 neurons. (**a**) Each point represents one neuron's average firing rate, 50–150 ms after cue onset. (**b**) Averaged cue-aligned spike density histograms for 15 neurons that had a significantly greater response on antisaccade than on prosaccade trials.

ed to respond more strongly to cue onset when the monkey knew it would make a saccade away from the cue than when the cue directly marked the saccade's goal (for example, V^+M^- versus V^+M^+ trials, neurons A and D in **Fig. 2**). Although relatively modest in individual neurons, the trend in the cue epoch was highly significant for the sample as a whole (**Fig. 7a**, $p < 10^{-3}$). Note that this difference was in the opposite direction from that during the presaccadic epoch, when neurons responded more in V^+M^+ than in V^+M^- trials.

This effect reached statistical significance for 15 neurons, for which averaged spike density histograms are shown in Fig. 7b. The response enhancement reflected increased sensitivity to visual stimulation, as it did not occur in the baseline firing rate, before cue onset. Responses on V^+M^- trials also exceeded the cue-evoked activity on delayed prosaccade trials, showing that this effect could not be attributed simply to the requirement to inhibit an immediate, reflexive saccade to the cue. That this enhancement was important for the monkey's performance is suggested because it occurred specifically on correct antisaccade trials. The response on V^+M^- trials in which the monkey made a mistaken prosaccade to the cue was equivalent to that on other prosaccade trials and lower than that on correct V^+M^- trials (median response difference for the neurons in **Fig. 7b**, 12 sp/s, $p < 10^{-5}$).

Discussion

In the antisaccade task, a salient visual cue dictates an immediate saccade to an opposite spatial goal. This cue is thus task-relevant even though its spatial location differs from the goal of the saccade. We found, in this conflict situation, that the vast majority of LIP neurons unambiguously encode the location of the cue. However, they transmit much less information about the direction of the saccade, even at the time of the movement itself. Most LIP neurons thus encode significant visual stimuli, or events, much more reliably than they encode the direction of upcoming saccades.

The strong visual activity we found here confirms and extends a large number of prior observations that LIP neurons have strong visual responses independent of the monkeys' oculomotor strategy (maintained fixation or delayed saccades toward or away from the stimulus[9,12]). In the present task, LIP neurons responded more strongly to cues that triggered an immediate saccade away than to those that directly marked the saccade goal. The functional significance of this enhancement—which appears similar to that observed in monkey supplementary eye field[19] and in human scalp potential recordings[2]—remains unclear. It may reflect increased attention or arousal necessary for antisaccade performance, or a change in motor plan from the habitual prosaccade to the spatially incongruent response[19,20]. This modulation is clearly inconsistent, however, with the idea that the visual on response represents the formation of a covert saccade motor plan that is cancelled in downstream oculomotor structures.

Also consistent with previous studies[11,12] is the present finding that most LIP neurons had lower-level sustained activity that spanned the delay period between extinction of the target and a saccade to the remembered target location. We show here, however, that much of this activity depended on the memory of the visual target rather than on the preparation of the saccade itself. More than half of the neurons with sustained activity before memory-guided saccades responded significantly less, or not at all, before antisaccades of equivalent metrics (see also ref. 9). This sustained visual response was not suppressed even immediately before the monkey executed a saccade in the opposite direction. Most LIP neurons, therefore, seem to maintain visual representations on-line relatively independently of the monkey's oculomotor strategy, similarly to some neurons described in prefrontal cortex[21,22].

A minority of LIP neurons (approximately 25%) did have stimulus-independent presaccadic activity, which was equivalent before immediate and delayed antisaccades to the response field. This activity is analogous to that described before in the context of a learned-saccade task[8,9]. However, not all neurons with saccade-related activity could encode the direction of an antisaccade within behavioral reaction time. About half of these neurons also had sustained visual responses and responded throughout the presaccadic epoch when the cue appeared in their response field, even when the monkey made a saccade in the opposite direction. Thus, an even smaller percentage of neurons (in our estimate, 12%) reliably encoded saccade direction on no-delay trials. Because the sustained visual response appeared to decay with time, we cannot rule out the possibility that, given enough time, all 25% of the neurons with saccade-related activity would ultimately provide a reliable signal of saccade direction. However, the functional significance of this putative extra presaccadic activity, which would occur only at delays beyond a normal reaction time, is difficult to interpret.

It is clear from a large number of previous studies that LIP neurons can accurately reflect saccade planning under certain circumstances. Following presentation of a visual stimulus in their response field, most LIP neurons have sustained (delay and presaccadic) activity that is selective for the direction of the next intended saccade or, equivalently, for the spatial location of the next saccade's target[9,12,17,18]. However, the present data show that this selectivity is critically dependent on the saccade target and not on the saccade itself. In our experiment, most LIP neurons were indeed directionally selective in trials in which the cue appeared in their response field; however, they did not respond in relation to, or even showed the opposite directional selectivity

for, non-visually guided saccades (antisaccades). Like the initial visual responses, therefore, the sustained activity in LIP seems to be primarily determined by the presence and nature of a visual stimulus. Stimuli that are intrinsically salient[9,23] or task relevant strongly activate the vast majority of LIP neurons.

The visual dependence of the activity in area LIP contrasts with findings in two frontal saccade-related areas. Neurons in the supplementary eye field discharge before prosaccades and also, more strongly, before antisaccades to their response fields[19]. Whereas the projection from LIP to the superior colliculus conveys visual information[10], the efferent pathway from the frontal eye fields to the colliculus conveys primarily saccade-related activity independently of visual guidance[24,25].

These considerations suggest a functional distinction between frontal and parietal areas: whereas the main function of parietal areas such as LIP may be to describe the salient world, the main function of frontal structures such as the frontal eye field may be to decide how and when to act in that world (for evidence from the human literature, see refs. 26–28). Although the visual activity in LIP has privileged access to saccade-related structures, our experiments show that the saccadic system can simultaneously ignore the LIP signal and contravene it. If behavior were controlled only by the parietal lobe, it might never transcend the immediate demands of the sensory environment.

Methods

Experimental methods. Two male rhesus monkeys (*Macaca mulatta*) were prepared for physiological recording during sterile surgery under ketamine and isofluorane anesthesia. General behavioral and physiological methods were as described[8,9], with behavioral monitoring and data collection controlled by a 486 PC running the REX system[29], and visual stimuli projected upon a tangent screen by an Electrohome Video Projector driven by a second personal computer. All experimental protocols were approved by the NEI Animal Care and Use Committee as complying with the guidelines established in the Public Health Service Guide for the Care and Use of Laboratory Animals.

Behavioral tasks. Six different trial types were presented with equal probability in pseudo-random order. To initiate each trial, the monkey fixated a central fixation point, which could be either red or green. While the monkey maintained fixation, a visual cue flashed for 50 ms at a peripheral location. If the fixation point was red, the monkey was rewarded for making a saccade to the location of the cue (prosaccade); if it was green, he was rewarded for making a saccade to the location diametrically opposite the cue (antisaccade). The fixation point was a 0.5° square, and the cue was a white annulus 2° in diameter. Two cue locations were used for the testing of each neuron: one in the estimated center of the neurons' response field and the other at the diametrically opposite location, resulting in the four trial types (two cue locations × two mapping rules; **Fig. 1**). In these four trial types, the fixation point disappeared simultaneously with cue onset, instructing the monkey to make the required saccade without delay.

We used two additional trial types in which the fixation point remained lit for 450–700 ms after disappearance of the visual cue, thus instructing a delayed saccade. Half of these trials were prosaccade trials in which the cue and saccade goal were in the response field (memory-guided prosaccades), and half were antisaccade trials in which the monkey made a saccade to the response field in response to a cue presented at the opposite location (delay V^-M^+ trials).

Neural recording. Area LIP was identified physiologically by its significant visual, delay period and presaccadic responses on memory-guided saccade trials. Of the 105 neurons described here, 89%, 61% and 57% had significant activity (relative to baseline) during the cue, delay and presaccadic epochs, respectively, of the memory-guided saccade task, consistent with a previous report[11]. To ensure complete sampling of area LIP, we searched for responsive neurons up to 2 mm (along the grid axes) beyond the region yielding appropriate activity. All recording sites in one monkey were localized to area LIP as identified by myeloarchitectonic boundaries[5]. Magnetic resonance imaging was used to aid in chamber placement and to verify the location of electrode tracks.

Data analysis. Both monkeys performed pro- and antisaccades at more than 80% correct and with comparable latencies. Monkey 1 had a mean latency (± s.d., across all recording sessions, no-delay trials) of 293 ± 33 ms for prosaccades and 278 ± 43 ms for antisaccades. For monkey 2, the corresponding latencies were 265 ± 26 ms (prosaccades) and 265 ± 33 ms (antisaccades).

Saccade accuracy was controlled on-line by means of an invisible electronic window that could allow up to 50% overshoot or undershoot in saccade amplitude. We further tested the effects of saccade accuracy off-line. From each neurons' data set, we first calculated the mean endpoint of prosaccades to the response field and then measured the average distance between this prosaccade vector and the endpoints of antisaccades to the response field, separately for delay and no-delay trials. We excluded from analysis neurons (13/118) for which this distance was larger than 20% of mean prosaccade amplitude in either immediate or delay trials. For the remaining neurons, antisaccades to the response field deviated from the mean prosaccade amplitude by 10 ± 5% in no-delay trials and by 9 ± 18% in delay trials (mean ± s.d.).

To examine the effect of antisaccade scatter in the remaining data set, we compared the presaccadic activity associated with progressively more accurate subsets of antisaccades with the activity associated with all antisaccades. Antisaccades whose endpoints fell within 4, 3, 2.5, 2, 1.5 or 1 standard deviations of the prosaccade endpoint distribution were not preceded by significantly different activity than the entire sample of antisaccades in either immediate or delay trials. All antisaccades were therefore included in the present analyses. To assess the effect of saccade velocity, we fit the presaccadic activity using a linear regression model with experimental condition (prosaccade or antisaccade) and saccade velocity as independent variables. In only a minority of neurons did saccade velocity account for a significant proportion of response variance beyond that captured by the experimental condition (20/105, 19% neurons in no-delay trials and 16/79 (20%) neurons in delay trials).

Action potential trains, sampled at 1 kHz, were convolved with a Gaussian[15] of sigma 10 ms. Neural responses were measured as the average of this spike density trace for all correct trials, unless otherwise noted, in 4 different epochs: a baseline epoch, 200 ms before cue onset; a cue epoch, 50–150 ms after cue onset; a delay epoch, 150–450 ms after extinction of the cue; and a presaccadic epoch, 50 ms before saccade onset. Statistical significance ($p < 0.05$ unless otherwise noted) was inferred from the Wilcoxon rank test for paired or unpaired samples.

Instantaneous information was estimated with the cluster method[16], using spike count in 20-ms bins as the input code. To calculate information about cue location, responses on no-delay trials in which the cue appeared in the response field were compared with those in which it appeared at the opposite location (V^+M^+ and V^+M^- versus V^-M^+ and V^-M^- trials). To calculate information about the saccade, responses on no-delay trials in which the saccade goal was in the response field were compared with those in which it was at the opposite location (V^+M^+ and V^-M^+ versus V^+M^- and V^-M^- trials). Information in individual bins was averaged over a cue interval, 40–160 ms after cue onset (6 bins), and a saccade interval, 60 to 0 ms before saccade onset (3 bins). Unlike measures of cumulative information, this measure would not be expected to be systematically affected by the length of the measurement interval.

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