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RESEARCH ARTICLE

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Ocular fixation and visual activity in the monkey lateral intraparietal area

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Abstract The macaque lateral intraparietal area (LIP) has been implicated in visuospatial attention and saccade planning. Since area LIP also contains a representation of the central visual field, we investigated its possible role in fixation and foveal attention in a visual fixation task with gap (momentary disappearance of fixation point). In addition to the expected visual neurons (n=119), two main categories were identified: (1) cells responding tonically both during the presence and momentary absence of the fixation stimulus(n=47); a subset of these neurons studied in a saccade task showed perisaccadic inhibition in half of the cases (14/27). The timing of this inhibition, however, is only loosely related to saccade timing; (2) cells responding mainly to the *absence* of the fixation stimulus, with either abrupt or gradual onset of activity during the gap (n=62). During saccades, these neurons showed presaccadic buildup and/or postsaccadic activity, which was spatially tuned in about half of the tested cells (28/53). Ninety-one percent of the cells in the first category and 59% of the cells in the second category were located in the dorsal portion of area LIP (LIPd). These results are consistent with the hypothesis of an oculomotor-attentional network contributing to fixation engagement and disengagement in a subregion of LIP.

Keywords Lateral intraparietal area · Fixation · Saccade · Enhancement · Buildup · Electrophysiology · Monkey

Introduction

During visual exploration, steady fixation alternates with gaze shifts to peripheral objects, selected because of their intrinsic saliency or through covert decisional processes. The lateral intraparietal area (LIP) has been implicated mostly in visuospatial attention (Colby et al. 1996; Ben Hamed et al. 1997, 2001b; Gottlieb et al. 1998) and voluntary saccades (Gnadt et al. 1986; Barash et al. 1991a, 1991b; Colby et al. 1996). This is consistent with anatomical data showing strong interconnections between LIP and two brain regions important for visual orienting: frontal eye field (FEF; Schall et al. 1995; Stanton and Goldberg 1995; Bullier et al. 1996) and superior colliculus (SC; Asanuma et al. 1985; Colby et al. 1988; Lynch and Graybiel 1985; Paré and Wurtz 1997). Both contain distinct groups of neurons with saccade or fixation-related activities.

Fixation activities, most extensively investigated in the rostral SC (SCr; Munoz and Wurtz 1993b; Everling et al. 1998), are characterized by a tonic discharge during active fixation, even if the fixation target is momentarily extinguished, and an inhibition time-locked to saccades. Fixation cells mainly have foveal or parafoveal visual RFs and may discharge before saccades of small amplitude. Paré et al. have shown recently that LIP neurons are antidromically activated by stimulation of SCr (Paré and Wurtz 1997; also see Gnadt and Beyer 1998). Previous studies have also reported neurons in area 7 and possibly in LIP, active during the fixation of a stimulus, and pausing during saccades (Lynch et al. 1977; Sakata et al. 1980; Bremmer et al. 1997; Read and Siegel 1997; Gnadt 2000). This is compatible with fixation-related signals, but also with tonic foveal visual signals, eye-position sensitivity or a combination of both.

LIP contains a representation of both central and peripheral visual field, and only a coarse topography has been described (Blatt et al. 1990; Ben Hamed et al. 1999, 2001a). However, connectivity studies suggest a functional heterogeneity of this area. Its portion most strongly connected with the FEF and SC is located deep in the intraparietal sulcus and matches a heavily myelinated region (Lynch and Graybiel 1985; Blatt et al. 1990; Schall et al. 1995; Bullier et al. 1996), labelled LIPv (Colby et al. 1993). The frontal projections of the dorsal part of LIP (LIPd) are much lighter in core FEF and are scat-

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tered over the arcuate and periprincipal cortex. LIPv and LIPd also have distinct, overlapping extrastriate visual area efferents, LIPv receiving projections mostly from dorsal stream areas and LIPd being connected to both dorsal and ventral streams (Ungerleider and Desimone 1986; Boussaoud et al. 1990; Andersen et al. 1990a; Blatt et al. 1990; Stanton and Goldberg 1995).

Here we describe neuronal activities recorded in LIP during fixation. We found two main types of cells segregating differentially between LIPd and LIPv in addition to the expected visual phasic or tonic neurons responding to the appearance or extinction of the fixation point: first, neurons responding tonically whether a stimulus is present or not; second, neurons discharging specifically during the blink interval. Subsets of these two groups were studied during saccade tasks, and their patterns of activity are contrasted to similar neurons described in the FEF and SC. These observations are interpreted in the context of oculomotor behavior and spatial attention. Preliminary results have been reported elsewhere (Ben Hamed et al. 1999).

Methods

Animal preparation, and physiological and histological methods

Experiments were conducted in one rhesus monkey (*M. mulatta*, 4.6 kg) and one fascicularis monkey (*M. fascicularis*, 3.8 kg). All animal care, housing, and surgical procedures were in accordance with European published guidelines on the use of animals in research (European Community Council Directive 86/609/ECC). During training and single-cell recording experiments, the animals were under fluid intake control. Monkeys were prepared for recordings by implanting a head-holding device under general anesthesia (Ketamine and Propofol) and sterile surgical conditions. Scleral search coils were placed, and a recording chamber for microelectrode penetrations through the intact dura was anchored flat to the skull centered at AP –11 and ML 16 mm for the first chamber and at AP 5 and ML 11 for the two other ones. Recording chamber, eye coil plug, and head holder were all embedded in dental acrylic connected to the skull by self-tapping screws. Extracellular recordings used tungsten-in-glass electrodes (Frederick Haer; impedance, 1–2 MW at 1 kHz) advanced by means of a hydraulic microdrive (Narishige) mounted on the recording chamber. During recording sessions, area LIP was identified by its location within the intraparietal sulcus and by its typical physiological response characteristics (LIP cells have visual and oculomotor properties as well as specific delay activity during memory-guided saccade tasks; see Barash et al. 1991a, 1991b; Ben Hamed et al. 2001a), with regard to the neighboring areas 7a (visual responses in 7a are more difficult to evoke than in LIP, and saccadic responses are mainly postsaccadic; see Andersen et al. 1990b), ventral intraparietal area VIP (VIP cells are sensitive to stimulus direction of movement and optic flow responses and can have bimodal or polymodal responses to tactile, visual, auditory, or vestibular stimulation; see Colby et al. 1993; Bremmer et al. 1999, 2001), and medial intraparietal area MIP (MIP cells have visual responses as well as responses to hand reaching movements by the monkey; see Colby and Duhamel 1991; Johnson et al. 1996). One animal is still used in ongoing experiments; in the other monkey, detailed histological analysis verified that recording sites in the two hemisphere had been located either in the dorsal or ventral portions of area LIP, respectively LIPd and LIPv. For this last monkey, microlesions (50 μA for 15 s) were made at specific locations in the lateral banks of the intraparietal sulci of both hemispheres. Fixation was carried out with a buffered solution (pH 7.4) of 4% paraformaldehyde. Marking pins were inserted to mark the limits and orientation of the grid, and the head was postfixed in paraformaldehyde solution for several days. The pins were then removed, and the brain was extracted. A block of tissue containing the intraparietal sulci and neighboring regions was cut from both hemispheres and immersed in 0.4 M phosphate buffer/10% sucrose solution for 2 days. The tissue was cut on a freezing microtome (50 μm sections) along planes parallel to the marking pins. Sections were counterstained with thionine. One in ten sections were myelin-stained using the Schmued method (Schmued 1990). Sections were digitized using a microscope coupled to a Neurolucida system. Two-dimensional flattened reconstructions of the intraparietal sulcus were subsequently obtained using a software developed in our laboratory.

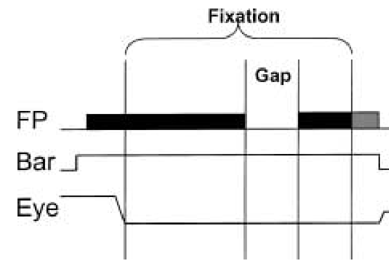

Fig. 1 Gap task paradigm. A fixation point appears at the center of the screen (*FP trace*) and the monkey has to fixate it (*Eye trace*) and manually hold a lever (*Bar trace*). After 1,200–1,600 ms, the fixation point is momentarily turned off and the monkey has to maintain fixation. This "gap" interval was fixed and lasted 200 ms or 400 ms in most experiments. Following its reappearance and after a second delay, the fixation point changes color and the monkey has to respond by releasing of lever in less than 700 ms

Behavioral tasks

During training and recording sessions, the animal was restrained in a primate chair. A PC running the REX software package (Hays et al. 1982) controlled behavioral paradigms and data acquisition. The monkeys were initially trained to manually grasp a handle mounted on the chair, look at a central spot of light, and release the handle when the spot changed color, to obtain a drop of water or fruit juice. When the monkeys performed at 80% correct responses, they were surgically prepared for extracellular recordings and eye-position monitoring, and further trained on the following tasks.

Fixation and "gap" fixation

The monkey had to manually grasp the chair-mounted handle in order to initiate a trial in the fixation and "gap" fixation tasks. This triggered the appearance of a 1°-wide square stimulus back-projected at the center of a translucent tangent screen. In the standard fixation task, the fixation stimulus was continuously present on the screen. The monkey was rewarded for releasing the handle within 700 ms of the color change of the fixation stimulus. The gap fixation task was similar to the standard task, with the difference that, in the course of the trial, the central stimulus was briefly extinguished and the monkey had to continue to fixate (Fig. 1). In both tasks, the use of color discrimination served to enhance the behavioral salience of the fixation point and focused the animal's attention at the fovea. The duration of a given fixation trial (until the color change) varied randomly between 2,000 and 2,400 ms. In the gap task, the central stimulus was turned off about 1,200–1,600 ms from fixation onset, and the gap itself generally lasted 400 ms (range 200–800 ms). The change in target color was unpredictable but occurred on average about 600 ms after the re-

appearance of the fixation target. Eye position was continuously monitored at a rate of 1 kHz, and the trial was interrupted if the eyes moved out of a 2°×2° tolerance window centered on the fixation target. Furthermore, during offline data analysis, trials in which eye movements larger than 1° were made inside the window were eliminated. Standard and gap fixation trials were run in separate blocks.

Memory-guided saccades

The memory-guided saccades task was used to detect changes in neuronal activity around the time of saccade execution. Briefly, as in the fixation task, the monkey was required to manually grasp a handle to trigger the appearance of a central stimulus. Four hundred milliseconds after the achievement of fixation, a second, peripheral stimulus was flashed for 100 ms at one of either four or eight possible locations radially arranged around the fixation point at an eccentricity of 7.5°. Following a delay period varying between 1,200 and 1,600 ms, the fixation stimulus disappeared, cueing the monkey to execute a saccade, in complete darkness, to the spatial location of the peripheral stimulus. When the saccade landed inside a 2°×2° tolerance window, the stimulus reappeared 50–200 ms later and became the new fixation target, which the monkey monitored until it changed color. As in the fixation task, the monkey responded to the color change by releasing the handle in order to obtain a reward. By dissociating in time visual target presentation and execution of the saccade, this task allowed us to distinguish neuronal activity that might occur in relation to these two events. Also, because the saccade was made in total darkness and the reappearance of the target was delayed relative to the end of the saccade, it was possible to measure perisaccadic activity independent of any response to changes in visual stimulation parameters. Finally, having the monkey perform the visual discrimination and bar release in this task allowed us to contrast directly the end-of-trial activity following a saccade with that following the gap in the gap fixation task.

Receptive mapping

Whenever possible, care was taken to map the recorded neuron's visual receptive field (RF) using a computer-driven automated procedure. Briefly, while the monkey was performing a central fixation task, a series of flashed visual stimuli were presented in a pseudorandom order across a predefined grid of locations. The visual characteristics (size, color, orientation) of the mapping stimulus were defined on the basis of prior manual testing. For most LIP neurons, an achromatic 1°-wide square provided an optimal stimulation. The stimulus grid consisted of a square array of 7×7 or 9×9 locations. The size and location of this grid was defined so as to encompass completely the spatial extent of the manually estimated RF. Six to eight stimuli were presented within a single trial, and each location of the grid was stimulated 9–12 times (the minimal requirement to obtain a statistically reliable RF map is 6–7 times). With this procedure, the RF of any given neuron can be mapped in 2–3 min. Data obtained with this technique were analyzed offline by a reverse correlation method, and interpolated color-coded RF maps were generated and used to compute different RF parameters such as location of peak, center of mass, and width. The eccentricity of a given RF was taken as the distance of its center of mass from the fovea and its width was taken as the width of the portion of the RF area whose activity exceeded half the peak activity. Only well-defined RFs were retained for analysis, i.e., RFs with a coherent shape and totally included in the mapping grid (see Ben Hamed et al. 2001a for detailed quantitative analysis of RFs).

Data analysis

Unit recordings and eye movement traces collected during the fixation and saccade tasks were processed offline. Statistical analyses (*t*-tests and ANOVAS) were performed in order to categorize the neurons into different functional subclasses on the basis of their discharge rate in different phases of the gap fixation task, as compared to their spontaneous firing rate. One particular type of comparison involved computing (multiple successive) bin-wise ANOVAS on the number of spikes in two time windows, with either one fixed window and one sliding window, or two paired sliding windows (adapted from Oram and Perrett 1992; and Thiele et al. 1999). In the first set of comparisons, the first window was fixed and placed just before the spike alignment event (such as the beginning of the gap interval), and the second window was placed after the extinction of the fixation point and successively shifted in 5-ms steps. In the second set of comparisons, the initial position of the two windows was the same, but subsequently both were shifted by the same amount in 5-ms steps. In both comparisons the first window was 100 ms wide and the second 50 ms wide. The obtained *F*-values (and the related *P*-values) were measures of the distance between the cell activity in the two epochs being compared. The larger the *F*-value, the larger the difference in the cell's responses between the two conditions. These *F*-values were plotted continuously on the same time base as the spike histograms and were evaluated against a significance threshold set at $P<0.01$. This method allowed the detection of differences in activity between two contiguous phases of the gap fixation tasks, and was also used as a statistical basis for determining the latency of this change. Both the one-fixed/one-sliding and the two-sliding windows ANOVAS can detect abrupt increases or decreases in neuronal activity. Gradual variations such as the progressive buildup in firing rate are essentially undetected by the two-sliding windows ANOVA. On the contrary, the one-fixed/one-sliding window ANOVA can detect a point at which the firing rate in the second window rises above the firing rate in the first window. Thus the contrast between the two curves of *F*-value was used in particular to distinguish abrupt-onset responses from buildup responses during the gap interval. Finally, standard statistical procedures were applied to the memory-guided saccade data, to establish the existence of spatially selective saccade-related activity or, in the case of tonic-fixation neurons, the presence of perisaccadic inhibition.

Results

A total of 180 cells were studied in the gap fixation paradigm. Of these, 145 showed significant task-related activity and were subdivided into 2 main groups: (1) cells that responded to the fixation stimulus were classified as phasic or tonic. Within the group of tonic cells, we distinguished between cells whose activity diminished during the gap interval and cells whose activity was maintained; (2) cells that became selectively active during the gap. Some of these cells showed an abrupt onset of activity at the extinction of the fixation spot and remained active throughout the gap. The others showed a progressive buildup of activity during the gap interval. A summary of the identified classes of neurons and of the average characteristics of their RFs is given by Table 1.

In the following paragraphs we first describe in detail these categories of neurons as defined from their responses to the gap fixation task. Then we present the responses of these same categories to a guided-memory saccade task. Thirdly, we look at task effects on the prereward fixation responses. Finally, we provide evidence for a topographical segregation of the above-described cell categories between a ventral and a dorsal portion of LIP.

Table 1 Characteristics of the population of lateral intraparietal area (LIP) neurons studied. Distribution of the recorded population of LIP cell subcategories defined on the basis of their response pattern during a gap-fixation task. For the two main defined subgroups, the median visual receptive field (*RF*) eccentricity and size are indicated

	Cells		Median RF eccentricity	Median RF size
	n	%		
Cells active during fixation	83	57.2	2.9°	7.5°
Tonic: sustained during the gap	47	32.4		
Tonic: inhibited during gap	15	10.3		
Phasic: on, off, on/off	21	14.5		
Cells with gap-triggered responses	62	42.8	7.8°	11.4°
Abrupt onset	30	20.7		
Buildup	32	22.1		
Total	145			

Neuronal activity during the fixation task

Tonic responses

Many neurons showed an increase in discharge rate with the appearance of a foveal stimulus and subsequently maintained a significant level of activity during fixation and are considered to be potentially involved in foveal visual processing, foveal attention, and ocular fixation. We distinguish here between neurons whose activity was maintained during the gap interval, when the stimulation was momentarily interrupted (47/145, 32.4%; Fig. 2A), and neurons whose activity was dependent upon the continuous presence of a stimulus on the fovea (15/145, 10.3%; Fig. 2B). The criterion for tonic activity in both cell types was a mean firing rate during the first period of fixation that was significantly higher than the mean spontaneous activity in the dark. Persistent activity during the gap is defined by the lack of significant drop in discharge rate during the gap period, as measured by the sliding-window ANOVA, and by a discharge rate that remained significantly higher than the spontaneous firing rate. In cells that stop firing during the gap, the response was extinguished within 180–220 ms of the beginning of the gap. Both categories of cells showed a brisk, short-latency increase in activity at the reappearance of the fixation target.

Phasic responses

Another class of foveal cells (21/145, 14.5%) showed mostly phasic responses to visual stimulation and no significant tonic activity. These responses can be triggered by the foveation of the fixation stimulus at the beginning of the trial, by the extinction and/or reappearance of the fixation stimulus at gap time, and, more rarely, by the change in color of the fixation stimulus at the end of the trial. Some cells showed phasic on, phasic off, or both types of responses. Phasic on responses had a relatively short latency in the range of 50–80 ms, while off responses occurred within 70–120 ms from fixation extinction.

Gap-triggered responses

In contrast to these patterns of neuronal activity, we found a surprisingly large number of cells whose responses were selectively triggered by the gap interval during foveal fixation. Two types of gap-related activity were distinguished: abrupt onset and buildup. This distinction was based on the dynamics of *F*-value curves for the two types of sliding-window ANOVAS described in the Methods section. A parallel rise in either curve corresponded to a sharp increase in activity and defined an abrupt-onset gap response. In contrast, a significant rise only in the *F*-curve of the one-fixed one-sliding ANOVA was thus the defining criterion for buildup neurons.

Gap-related abrupt-onset responses. An abrupt-onset pattern was found in 30 of 145 cells (20.7%) and was characterized by a sudden increase in activity when the foveal stimulus was extinguished. The majority of cells in this subclass had a relatively long response latency, compared with the phasic on-off neurons, in the 120–200 ms range. In all cells, this activity was tonic, remaining at a sustained high level throughout the gap (Fig. 3A). Because the activity of these neurons during the gap was not similar in magnitude to their spontaneous activity in the dark, they cannot be considered as “foveal off” cells. For the neuron shown in Fig. 3B, the gray histogram in the background shows the activity recorded during intervals in which the monkey held its gaze steady at the same mean orbital position as during foveal fixation, but in total darkness and in the absence of any task or visual stimulation. The mean spike activity in this condition was significantly less than that measured during the gap. This was true for a majority of gap-related neurons (21/30, 70%), which showed significantly higher firing rates during the gap interval than during the intertrial interval when spontaneous activity was sampled (*t*-test, $P<0.01$ or less). Only one cell showed lower activity during the gap; the remaining cells showed no significant differences between their gap-related abrupt-onset response and their spontaneous activity at equivalent eye positions. A subset of the neurons that showed an abrupt increase in activity at the beginning of the gap (8/30) were not fully inhibited but instead maintained a high level of tonic activity following the reappearance of the fixation target until the end of

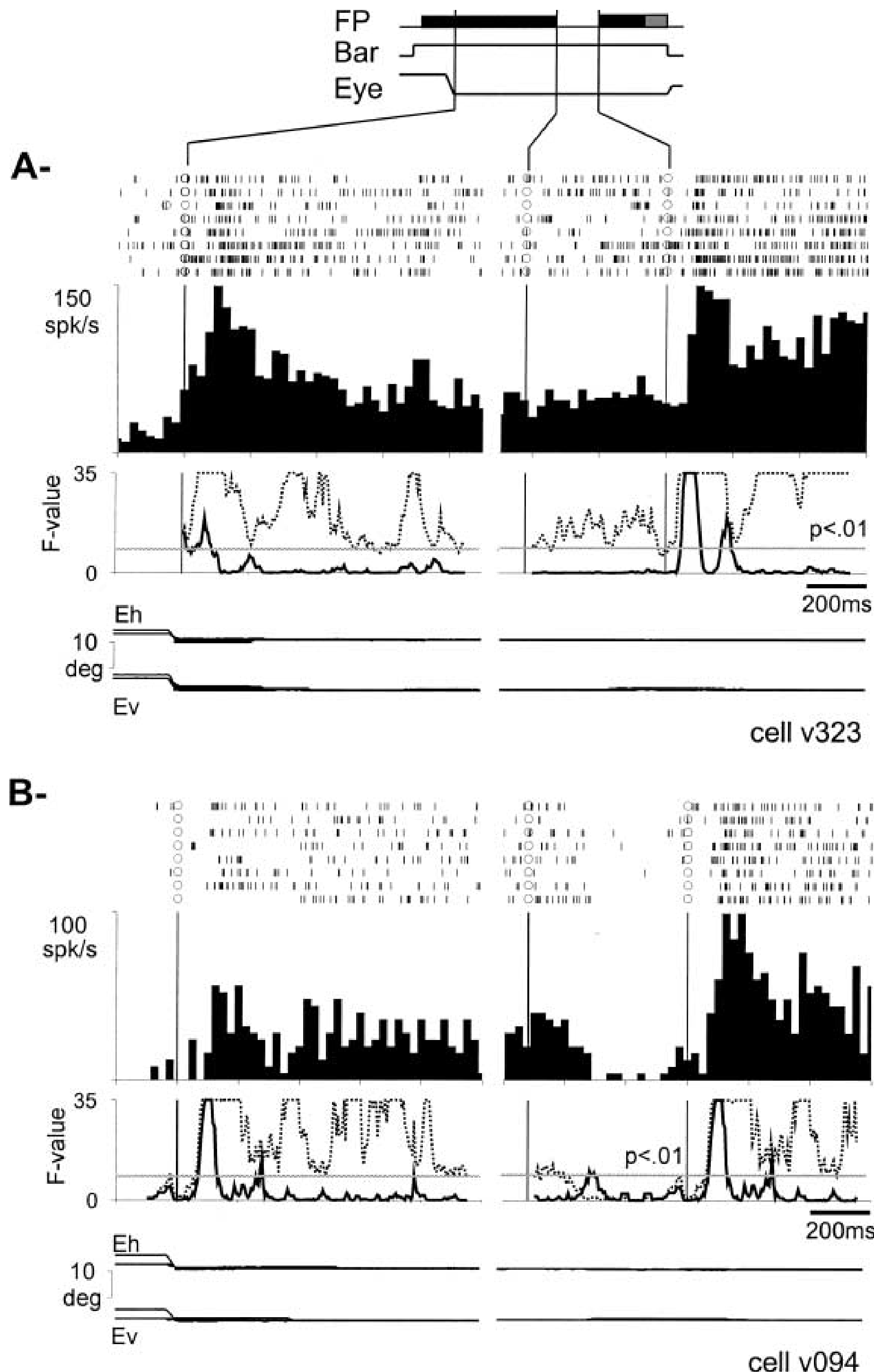

Fig. 2A, B Two representative neurons with tonic activity during fixation. The upper schema shows the task course and the spike alignment events (also represented by *circles in the raster plots* of **A** and **A**. The *upper part* of **B** and **B** shows spike rasters for individual trials, the *middle part* the cumulated response histogram (25 ms bins), and the *bottom part* the evolution of the *F*-value statistic computed with the sliding-window ANOVAs (see Methods). These *F*-value curves are obtained from the comparison of the response of the neuron between two sliding time windows. A first *F*-value is obtained with a first window being fixed at the beginning of the trial and a second window sliding forward in 5-ms steps (*dashed curve*), and a second *F*-value is obtained with two contiguous sliding windows (*solid curve*, see Methods section for details). The *horizontal line* indicates the *F*-value level for which the responses in the two time-windows reach a probability of significant difference of 0.01. **A** Unit v323 shows tonic activity to the appearance of the fixation point that is maintained through the *gap* interval. **B** Unit v094 shows tonic activity following the appearance of the fixation point and a return to baseline during the *gap* interval. The time scale is indicated at the *bottom left*

the trial, a phenomenon also difficult to account for on the basis of a simple visual off-response.

Gap-related buildup responses. The second type of gap-related activity was found in cells that, rather than showing an abrupt onset time-locked to the extinction of the fixation point, had more variable response latencies (32/145, 21%). When averaged across successive trials, the firing rate of these neurons expressed a progressive increase in activity that reached a peak and terminated with a sharp return to baseline shortly after the reappearance of the fixation stimulus (Fig. 3B). In 14 of 32 cells of this class, the activity culminated with a sharp phasic burst whose latency was compatible with a response to the reappearance of the foveal stimulus. In the remaining cases (18/32), no phasic burst was detected, and the activity peaked just as the fixation stimulus reappeared, with a latency that was too short to be attributed to the

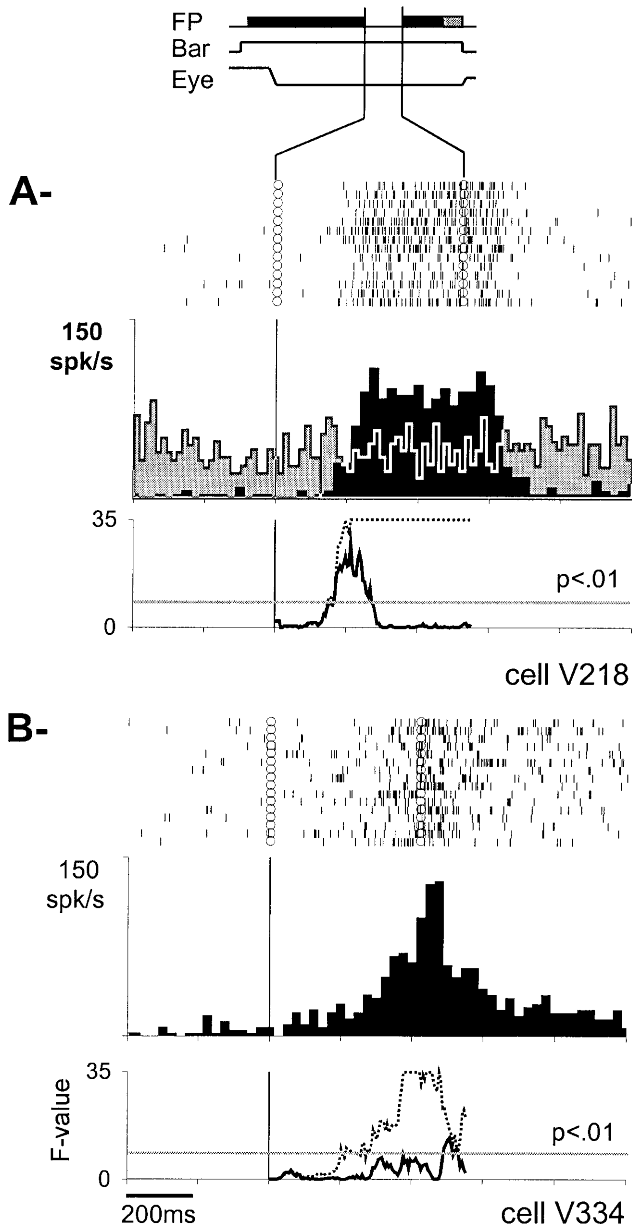

Fig. 3A, B Two representative neurons with activity triggered by the onset of the gap. Conventions as in Fig. 2. Unit v218 has an abrupt-onset response to the disappearance of the fixation point at the beginning of the gap interval (**A**), while unit v354 shows a progressive build up of activity (**B**). In **A**, the light gray histogram in the background represents mean spontaneous firing rate of the neuron in total darkness while the eyes were still and occupied an average horizontal and vertical position close to that required during foveal fixation

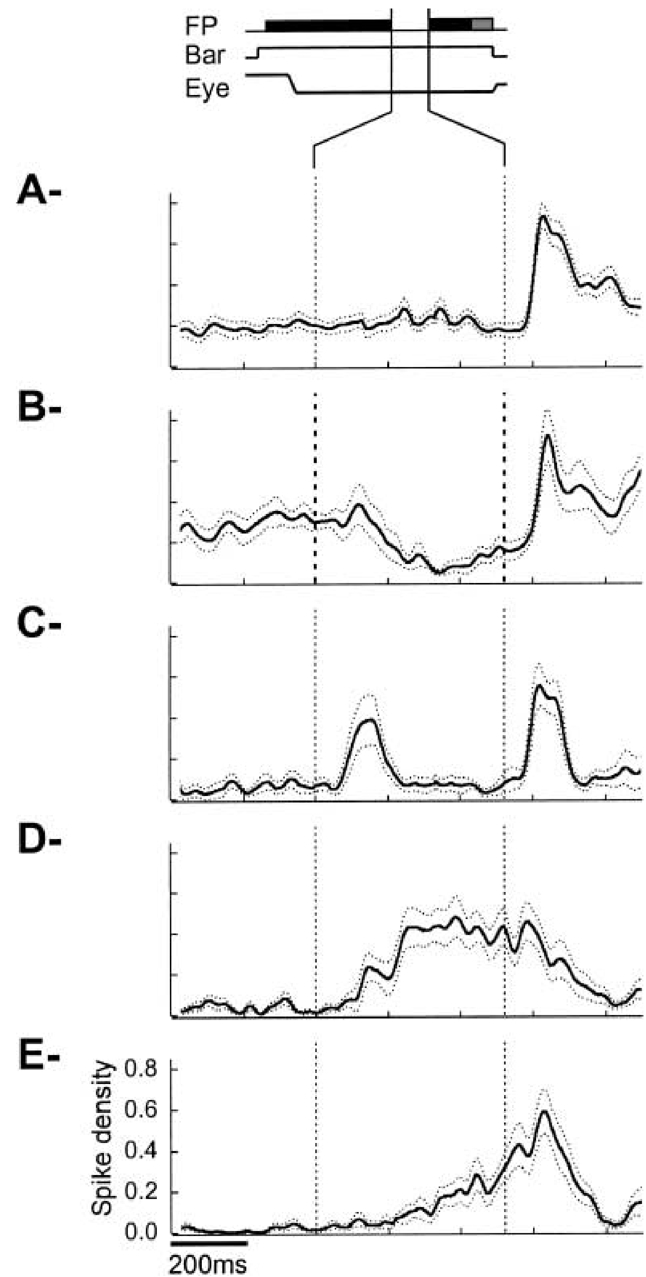

Fig. 4A–E Population spike density functions for each defined cell subcategory, representing activity near the gap interval in the fixation task. **A** Tonic gap-sustained cells (15 cells); **B** tonic gap-inhibited cells (47 cells); **C** phasic visual cells (21 cells); **D** abrupt onset gap-triggered cells (30 cells); **E** buildup cells (32 cells). Eight to twelve trials were available per cell. *Solid curves* represent the mean of normalized spike density functions and dotted curves represent the population upper and lower standard errors. Similar curves are obtained when population activity profiles were computed from nonnormalized data and show no difference

reafferent foveal stimulus. The mean latency of the activity buildup wsa quite long, ranging 200–260 ms from gap onset.

Population spike density functions were constructed by averaging the normalized gap-onset-aligned individual spike density functions of all the cells of a given category that were recorded for similar gap durations. These population spike density functions enabled us to appreciate the timing relations between the activity of the above-defined cell subcategories during the gap (Fig. 4). In particular the time course of the “off” response of the phasic visual neurons paralleld closely the decay of the tonic gap-inhibited neurons at the beginning of the gap. Both cell subpopulations became silent at the same time,

indicating a possible functional relationship between the two types of cells, e.g., the sharp and short-lived burst of the phasic cells might serve to truncate the response of the tonic gap-inhibited neurons. Another observation was the reciprocal pattern of activity of the tonic gap-inhibited neurons and of the abrupt onset gap-triggered neurons between 150 and 250 ms of the gap onset, with the first becoming silent as the second reached their maximal discharge during the gap, and vice versa at the end of the gap. Finally, the time course of activity of the two subpopulations of gap-related neurons was quite similar, with the main difference being found in their initial dynamics.

Saccade-related activity

In order to better define the relation between the activity of tonic neurons and ocular fixation, a subset of neurons was tested in a memory-guided saccade task. Targets were presented in the near periphery (4° or 7.5°) in one of either four or eight possible directions, radially arranged around the fixation point. An important difference between this task and the visually guided saccade tasks is that the saccadic target is specified prior to the disappearance of the fixation target and that the foveal stimulus is removed before the saccade is initiated, and the saccade ends before a new foveal stimulus is provided. Statistical tests were performed on neuronal activity recorded within several temporal windows related to these sensory and motor events. Activity was measured: (a) after the appearance of the saccade target (50–200 ms from target onset); (b) during the delay (250–1,000 ms from target onset); (c) just before the saccade (–150 ms to 0 ms from saccade onset); (d) during the execution of the saccade (saccade onset to saccade end); (e) after the saccade (50–150 ms from saccade end); (f) following the reappearance of the saccade target at the fovea (50–350 ms from target reappearance). ANOVAS were also performed on each of these temporal windows across the different saccade directions in order to determine the presence of spatial tuning of the observed neuronal responses.

Tonic neurons

Perisaccadic inhibition An important defining criterion for the identification of fixation-related activity is the presence of inhibition around the execution of eye movements. Different response patterns were expected in this task from tonic neurons that are solely driven by retinal signals, and from neurons that carry extraretinal information related to the monkey's oculomotor status. Cells that are purely visually driven should show inhibition (after a visual latency) following the extinction of the fixation stimulus but prior to saccade onset, and should not become active again until the saccade target reappears after an accurate eye movement is made. As expected, tonic cells whose activity stopped during the gap interval were not active before or during saccadic eye movements. Their activity decreased shortly after the fixation target disappeared and resumed when a new foveal stimulus was provided by the reappearance of the saccade target, following a visual latency.

About half of the tonic neurons tested (14/27) showed significant inhibition at the time of eye movements: in 8 cells an inhibition was present for all tested saccade directions, and in 6 cells inhibition was present for some but not all saccade directions. In general, however, the timing of perisaccadic inhibition was only loosely related to the oculomotor events. One example of a cell showing inhibition during eye movements in all directions is shown in Fig. 5. A computer-driven RF mapping procedure was used to obtain the RF outline shown in Fig. 5A. The RF was restricted to the foveal region. The saccades were thus made to locations well outside the cell's RF, and indeed none of the saccade targets evoked a visual response. This is the same cell as the one shown in Fig. 2A. It maintained a sustained level of activity throughout the gap period of the fixation task (Fig. 5B). Vertical upward and downward saccade directions are shown in Fig. 5C. The mean saccadic reaction time for these trials was about 275 ms. The tonic discharge paused about 205 ms after the fixation point was extinguished and 70 ms before the saccade began. Activity resumed to its presaccadic level about 130 ms after the saccade ended. Thus the pause of the neuron's activity lasted almost 200 ms, 4 times the mean duration of the saccades themselves. For the sample of LIP cells that were tested in the memory-guided saccade experiment, the mean time from saccade onset to pause onset was –64 ms (range –100 to –20 ms), and the mean time from saccade end to pause end was 102 ms (range 56ms–146 ms). Thus LIP cells paused relatively early and resumed their activity relatively late compared with superior colliculus fixation neurons (respectively –31 ms to saccade start and +31 ms to saccade end) and brainstem omnipause neurons (respectively –10 ms to saccade start and +9 ms to saccade end; Everling et al. 1998; Table 2). This suggests that the inhibition of LIP neurons might be more closely related to perisaccadic events, visual and/or attentional in nature, than to saccade initiation and termination proper.

Another example of a cell that pauses during saccades is shown in Fig. 6. This cell responded tonically to foveal stimulation but also responded to stimuli located in the upper perifoveal region as shown on the outline of its RF. Some of the saccade directions tested were located near or, in the example shown in the upper panel of Fig. 6C, inside the RF. Consistent with the location of the RF mapped during fixation, the cell responded with a phasic burst (not shown) to the saccade target flashed early in the trial in the upper left visual field. During the delay period, the cell discharged at a rate similar to that during simple fixation and, for this particular eye movement direction, the cell was not inhibited during the saccadic interval. However, it was inhibited for a saccade

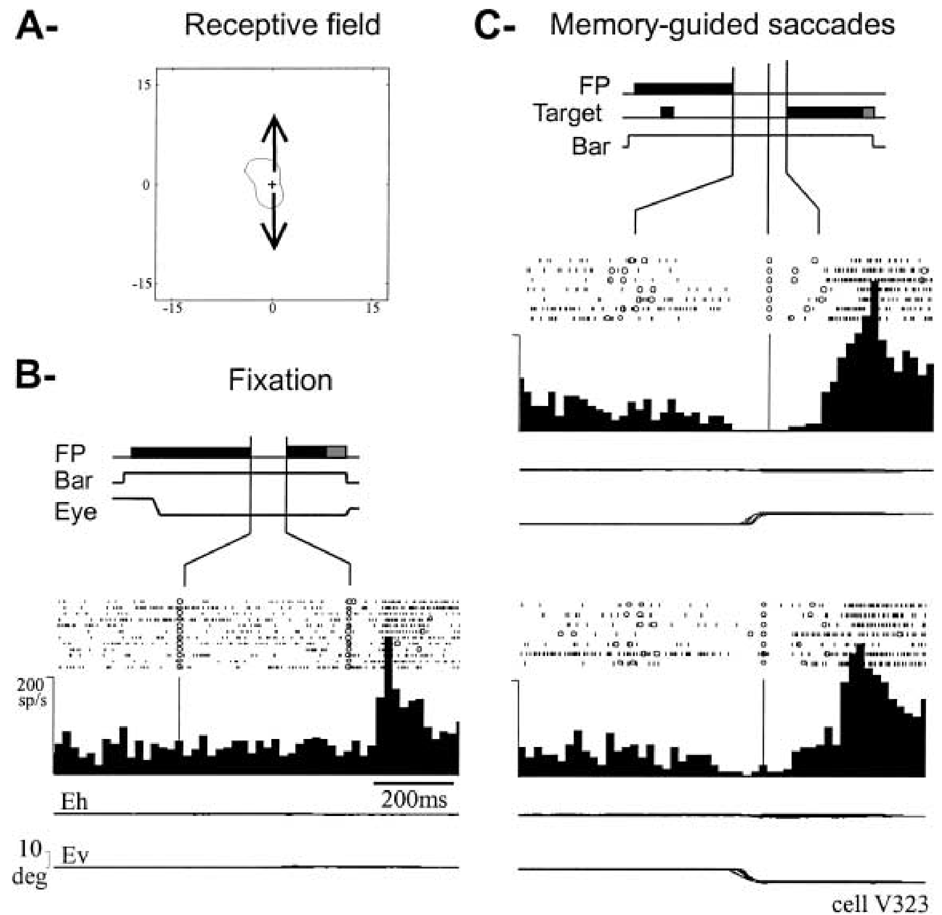

Fig. 5A–C Visual and saccadic properties of tonic-fixation unit v323. **A** Receptive field outline of the cell obtained using computer-driven mapping and calculated as the retinal field for which visually evoked activity rises above half the maximum firing rate (see Methods). A schematic representation of the two saccade directions shown in **C** is superimposed on the RF outline. **B** Response of the neuron in a gap fixation task. Conventions as in Fig. 2. **C** Response of the neurons during saccades to 10° right (*middle panel*) and 10° left (*lower panel*) memorized locations. Cell response is inhibited for both saccade directions

Table 2 Time course of pause in activity during saccades. Relation between discharge activity of tonic neurons and saccade dynamics. Mean of inhibition onset relative to saccade initiation, and of recovery relative to saccade termination across individual neurons, in three different structures: lateral intraparietal area (*LIP*; present study); superior colliculus (*SC*) and pontine nuclei (from Everling et al. 1998)

	Time from saccade onset to pause onset		Time from saccade end to pause end	
	Mean (ms)	Range (ms)	Mean (ms)	Range (ms)
Area LIP	–64	–100 to –20	+102	56 to 146
SC fixation neurons[a]	–31	–74 to –1	+31	–12 to 173
Omnipause neurons[a]	–10	–16 to 3	+9	–13 to 65

[a] From Everling et al. 1998

made in the opposite direction, as shown in the lower panel of Fig. 6C. It can also be noted that there was an overall relative decrease in activity during the memory period for a saccade outside the RF compared with a saccade into the RF or compared with the activity during the fixation task. This suggests that prespecification of a saccade target can modulate the activity of a neuron responding during fixation, even during periods in which the eyes are not moving.

Presaccadic activation. Tonic cells that did not show saccadic inhibition could express different types of activity at the time of saccade. In 8 of 27 cells a spatially selective, saccade-related discharge was present. These cells invariably had in addition a visual response to the saccade target that was superimposed on the tonic response to the fixation stimulus, consistent with the presence of a visual RF encompassing the foveal region and a portion of the adjacent periphery. The spatial tuning of the saccade-related discharges was congruent with that of visual discharge. The remaining cells (5/27) had spatially nonselective responses to the saccade.

Saccade-related activity in neurons with gap responses

The activity of a subset of neurons that responded selectively during the gap was also investigated in relation to

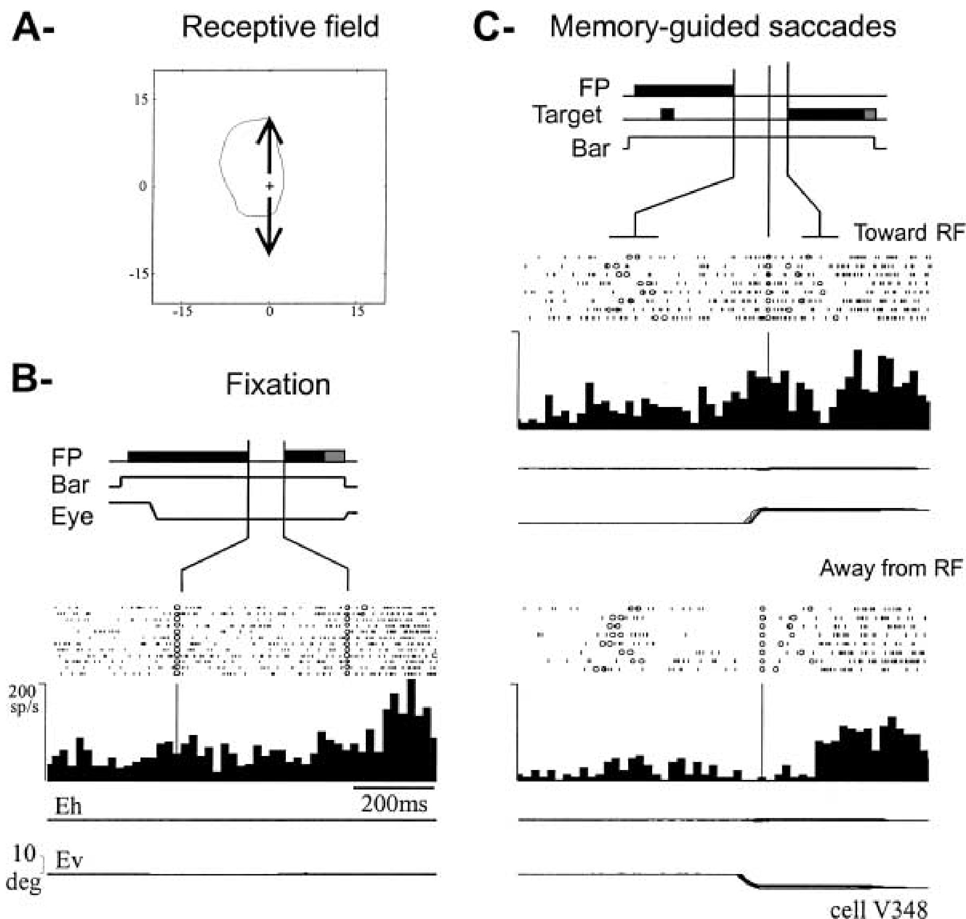

Fig. 6A–C Visual and saccadic properties of tonic-fixation unit v348. Conventions as in Fig. 5. Perisaccadic inhibition is present only for a saccade away from the receptive field

saccadic eye movements. Neurons with progressive onset in the gap fixation task systematically responded after a saccade in all direction (26/27). For most of these cells (20/26), the peak level of "postsaccadic" activity was similar to or smaller than that observed during the gap fixation task, when no saccade occurred. Interestingly, more than half of the neurons (17/27) showed in addition a spatially selective presaccadic buildup of activity. This subset of cells also showed visual responses to the saccade target, i.e., they had a visual RF extending into the near periphery, and the visual and buildup response were spatially congruent. An example of this pattern of activity is shown in Fig. 7. For saccades made in the optimal direction, the activity buildup began during the delay period of the memory-guided saccade task, while the response was mostly postsaccadic for saccades made in the antipreferred direction. The fact that activity buildup for the preferred saccade direction showed some anticipation with respect to gap onset in the gap task suggests that removal of a foveal stimulus is not the only or the main triggering event for such activity. Saccade planning or attention shift to the periphery might also be a sufficient condition.

Virtually all neurons with abrupt onset during the gap in the fixation task (25/26) responded during the perisaccadic interval and were inhibited shortly after the reappearance of the saccade target at the fovea. In a third of these cells (9/26), this activity following fixation point extinction in the memory-guided saccade task began at a latency consistent with the latency measured in the gap fixation task, irrespective of saccade direction, and the magnitude of the responses in the two tasks were also similar. In a majority of cases, however, saccade preparation and execution modified the temporal characteristics of the response with respect to the gap fixation trials. In 15 of 26 cells, the latency of the response was delayed with respect to that observed in the gap fixation task, beginning only after saccade onset. In 11 of 26 cells, the pattern of activity depended on saccade direction. Figure 8 shows an example of a cell modulated in a spatially selective manner. For saccades made in the upper right field, the onset of activity was not time-locked to and begins before the fixation point disappearance, while, for saccades made in the opposite direction, no such buildup occurred and the cell began discharging after the disappearance of the fixation point. Note that this cell did not have a peripheral visual RF and did not respond to the appearance of any of the peripheral saccade targets. Interestingly however, 16 cells with gap-triggered responses showed a spatially selective response to the saccade target. The combination of visual responses to peripheral saccade targets and responses to removal of a foveal fixation point suggests that these cells could have nonuniform RFs with excitatory peripheral and in-

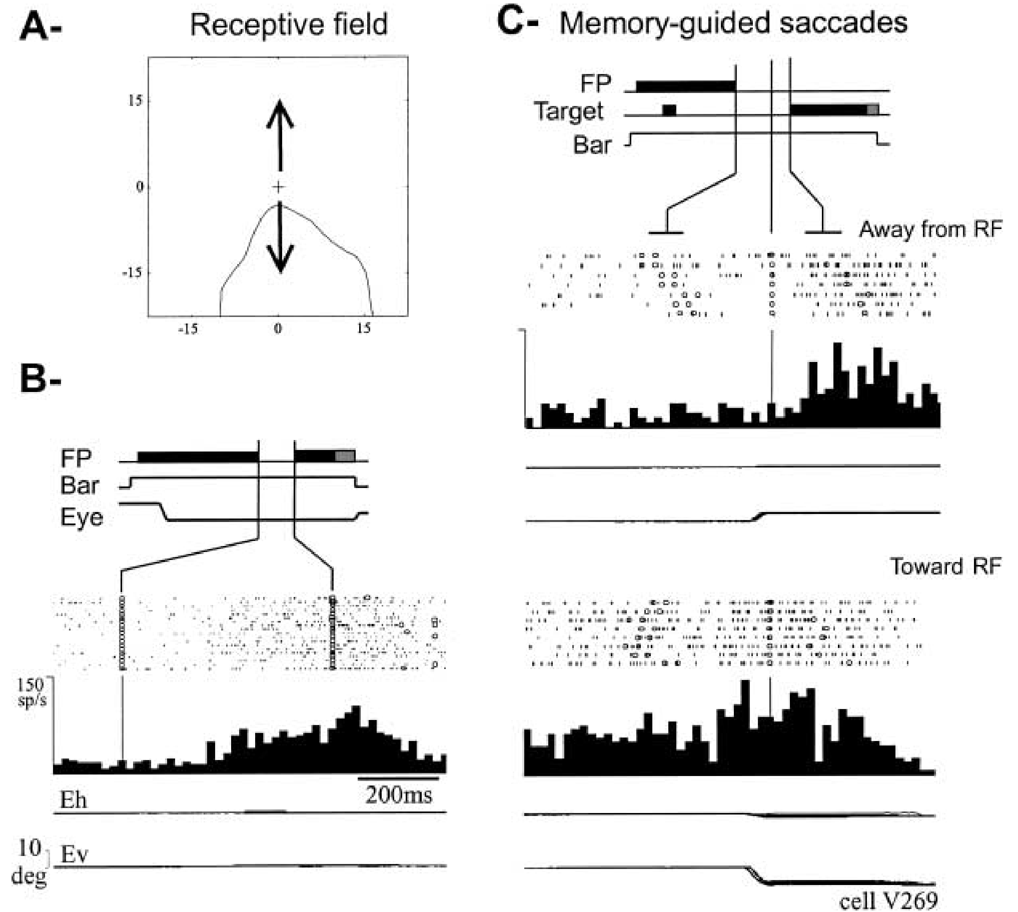

Fig. 7A–C Saccadic properties of gap-triggered buildup cell v269. Conventions as in Fig. 5. When the saccade is aimed inside the RF, there is significant activity during the presaccadic delay, while for other directions significant activity change occurs only postsaccadically

hibitory foveal regions, a possibility which was confirmed in some cases.

Receptive field characteristics

The locations of the center of mass and width of visual RFs were calculated for subsets of neurons from all of the categories reported here. The plots in Fig. 9 include data from cells with a well-defined RF, as measured with a computer-driven mapping procedure carried out during a standard fixation task (see Methods for details). Cells for which the existence of a RF can only be inferred on the basis of visual responses to saccade targets in the memory-guided saccade task are not represented. All of the cells with phasic and/or tonic activity during fixation had a visual RF. The general trend that can be observed is that these neurons have more central and smaller RFs than those with gap-related activity. The median RF eccentricity for the group of visual phasic and tonic neurons (35 cells) was 2.9° versus 7.5° for the gap-related neurons (40 cells; $P<0.001$, Mann-Whitney rank test). The median RF width was 7.8° and 11.4° for the former and latter categories of neurons, respectively, a difference which is also significant ($P<0.002$, Mann-Whitney rank test).

The RF characteristics of these cells cannot account completely for the pattern of activity that we report in the fixation and saccade experiments. Although in some cases, such as the one illustrated in Fig. 8, the location and size of the RF were consistent with the spatial selectivity of the response to saccade targets and to movement preparation in the memory-guided saccade task, these factors cannot explain nonspatially selective postsaccadic activity or activity buildup during gap fixation trials. Such apparent contradiction between the coexistence of "on" responses to peripheral saccade targets and "off" responses to fixation target may be resolved if one can identify an underlying, nonhomogeneous RF structure. This may not always be possible to demonstrate with RF mapping carried out during active fixation, since a neuron that is tonically inhibited by a behaviorally important foveal stimulus might have its responsivity to peripheral RF probes masked or diminished. For a different experiment (Ben Hamed et al. 1997, 2001b), we developed a method whereby visual RFs were mapped with stimuli flashed for 80 ms at locations defined with respect to the continuously monitored gaze direction, while the monkey was freely gazing on an otherwise blank screen. Figure 10 shows such a "free gaze" RF computed for a cell characterized by both abrupt onset activity during the gap fixation task and spatially selective visual and presaccadic responses in a memory-guided saccade task. This cell had both a large "on" peripheral subfield and a smaller "off" central sub-

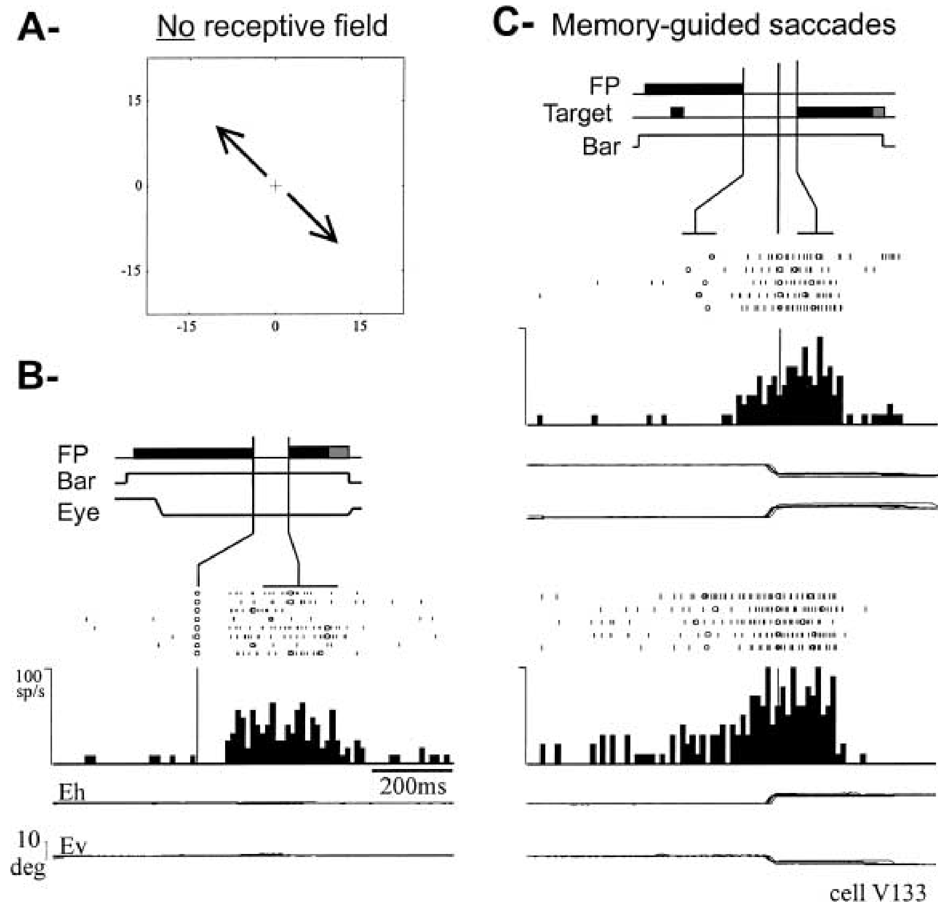

Fig. 8A–C Saccadic properties of gap-triggered abrupt-onset cell v133. The cell has no identified receptive field, otherwise conventions as in Fig. 5. During memory-guided saccades, the onset time of activity around fixation disappearance and saccade beginning varies slightly as a function of saccade direction

field, as illustrated by the "early" and "late" RF maps (Fig. 10A and B, respectively). This reciprocal pattern of activity over time is illustrated for probes at (0°,0°) and at (12°,0°) in Fig. 10C.

Modulation of fixation activity at the beginning and in the end of the task

Gap fixation task

The effect of the gap splits the fixation trial into two phases that are identical in terms of their visual parameters and motor requirements. Yet we found that a majority of cells showed differential responses to the fixation stimulus before and after the gap interval (see, for example, Fig. 2A, B). During fixation trials of the same duration in the absence of an intervening gap, these neurons do not typically show an increase in activity near the end of the trial, although a phasic response to the change in color of the fixation point can be observed as in the gap condition (data not shown).

As can be seen in Fig. 11, virtually all cells tested were enhanced following the reappearance of the fixation point. This enhancement was found to be statistically significant in about two-thirds of the tonic and phasic cells. In tonic neurons, we observed an enhancement of the tonic response (200–600 ms postgap), accompanied by an enhancement of the phasic response (0–200 ms postgap), when present. This enhancement was found in 10 of 15 tonic cells inhibited during the gap and in 26 of 47 tonic cells with sustained activity during the gap. In phasic neurons, there was an enhancement of the phasic postgap response relative to the response to the foveation of the fixation target at the beginning of the trial (9/21). By definition cells that discharge only during the gap should not show differences in activity before and after this event. However, some of the neurons with gap-related responses showed a more complex pattern of activity. As mentioned above, a proportion of the cells that were completely or almost completely inhibited during the first phase of fixation and that became active following the extinction of the fixation target were not reinhibited after its reappearance, but instead maintained a high level of tonic activity. This phenomenon was observed in about one-fourth of the abrupt-onset and buildup neurons (8/30 and 7/32, respectively).

Since the fixation tasks used in the present study always involved a manual response to a stimulus change at the end of each trial, we tested whether the gap could be used to predict the moment at which the fixation point would change color and allow a gain in reaction times. Manual reaction times following a gap were consistently shorter for gap than for no-gap trials. Mean reaction

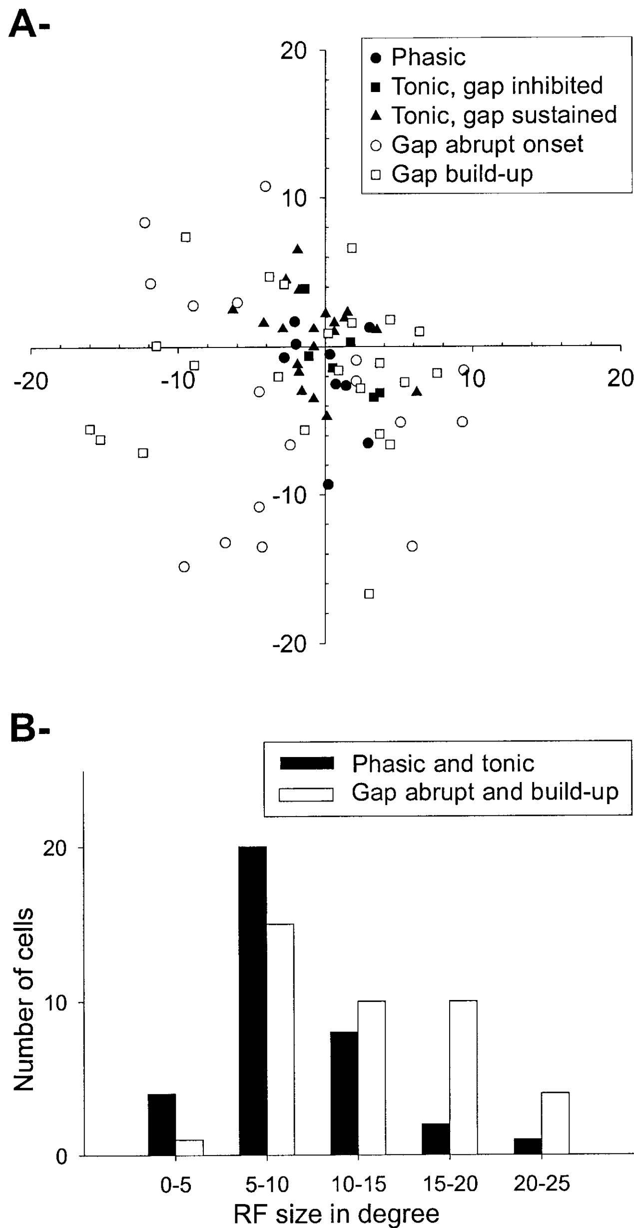

Fig. 9A, B Receptive field characteristics of the different cell categories. **A** Distribution of the RF centers of the different neuron subtypes as defined by their activity in the gap fixation task. Phasic cells and gap-inhibited and gap-sustained tonic cells significantly segregate from abrupt onset and buildup cells, and have RF with essentially central centers of mass. **B** Frequency distribution of RF size of the two broad categories of studied neurons

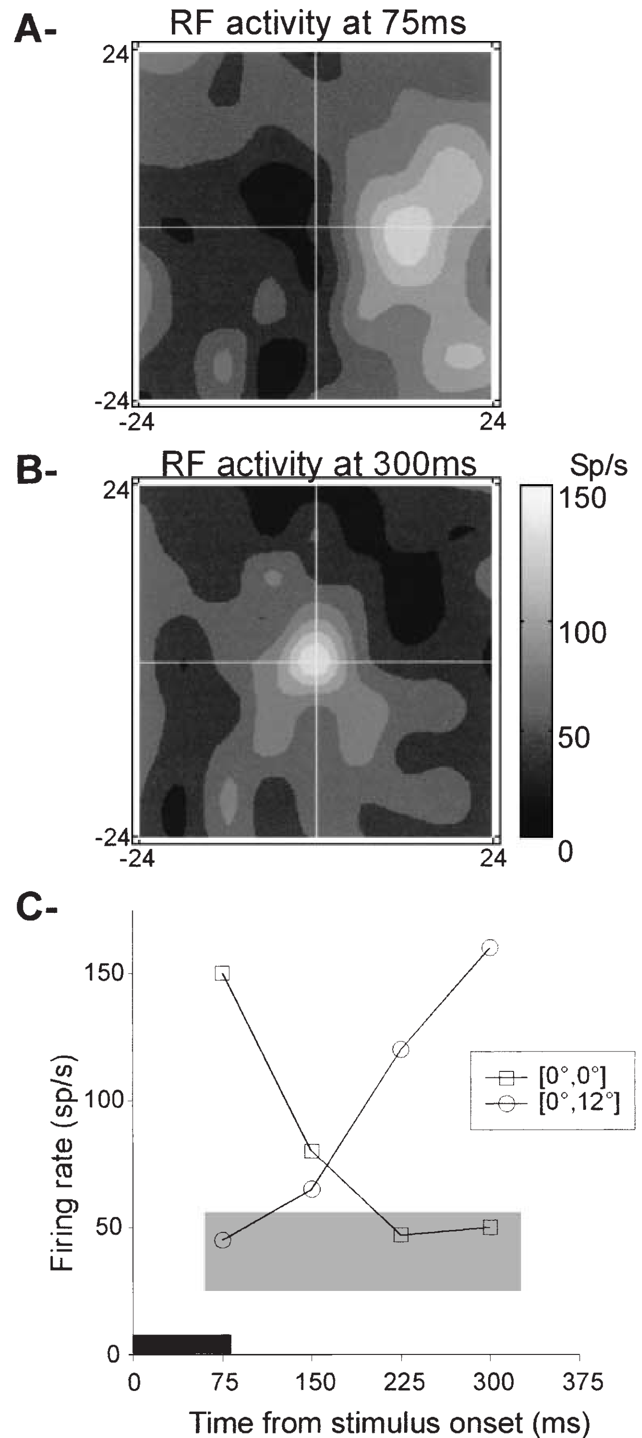

Fig. 10A–C Complex spatiotemporal profile of receptive field of an abrupt onset gap-triggered unit measured during free gaze, i.e., in the absence of a fixation target. **A** RF of the cell constructed by averaging the visual response in a 75-ms window beginning 75 ms after the appearance of the mapping stimulus. **B** RF of the cell in a 75-ms window beginning 300 ms after the appearance of the mapping stimulus (and 220 ms from its disappearance). **C** Time course of the mean response of the neuron for a stimulus presented at (0°,0°) – *circles* – and at (0°,12°) – *squares*. The level of variation (mean ± standard deviation) of spontaneous activity is shaded in *gray*, and the *black bar at the bottom left* of the graph indicates the time course of the RF probe stimulus

times were measured during 36 different recording sessions in which gap and no-gap fixation experiments were conducted (for a total of more than 700 trials of each type). Mean reaction times in the gap condition were shorter than in the no-gap condition by 77 ms or 22.8%. It should be noted that the timing of color change with respect to the gap varied randomly from trial to trial. However, trials lasted on average 2,500 ms and the gap occurred about two-thirds into the fixation period. Thus given the effects on reaction time, it is likely that the

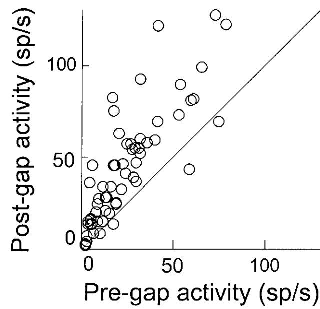

Fig. 11 Scatter plot of the mean discharge rate for all recorded cells during the pregap period (*x*-axis) and during the postgap period (*y*-axis). In tonic cells, the pregap period was taken as the 400 ms period centered on the fixation interval preceding the extinction of the fixation point and the postgap period was taken as the 200- to 600-ms interval following the reappearance of the fixation point just after the gap. In phasic cells, the pregap period was taken as the 0- to 200-ms interval following the onset of the phasic response to the fixation point, and the postgap period was taken as the 0- to 200-ms interval following the onset of the phasic response to the reappearance of the fixation point following the gap interval

presence of the gap afforded some degree of temporal prediction and was used as a general warning signal for the upcoming color discrimination.

We also measured the number of spikes produced by single neurons in a 500-ms window just before the change in color of the fixation target, for each individual fixation trial, and computed the correlation of this parameter to the monkey's reaction times. For all neurons tested, correlation coefficients were weak and nonsignificant (mean r=0.01, range –0.28–0.19), indicating that the modulation of LIP activity is not directly related to the manual reaction times. These results suggest that although the enhanced activity of tonic LIP cells following the gap could reflect an overall heightened state of behavioral arousal, focal attention or reward expectation, and may indirectly lead to fast reaction times, it is probably not a major contributing factor to motor programming of hand movements.

Saccade task or postsaccadic reactivation

As in the gap fixation task, the memory-guided saccade task involved two distinct fixation periods, a presaccadic phase and a postsaccadic phase. As can be seen in Figs. 5 and 6, a common feature of the response pattern of these neurons was an increase in their level of activity following the saccade and the reappearance of a foveal stimulus. Postsaccadic responses were found in virtually all tonic cells. The response generally occurred at a la-

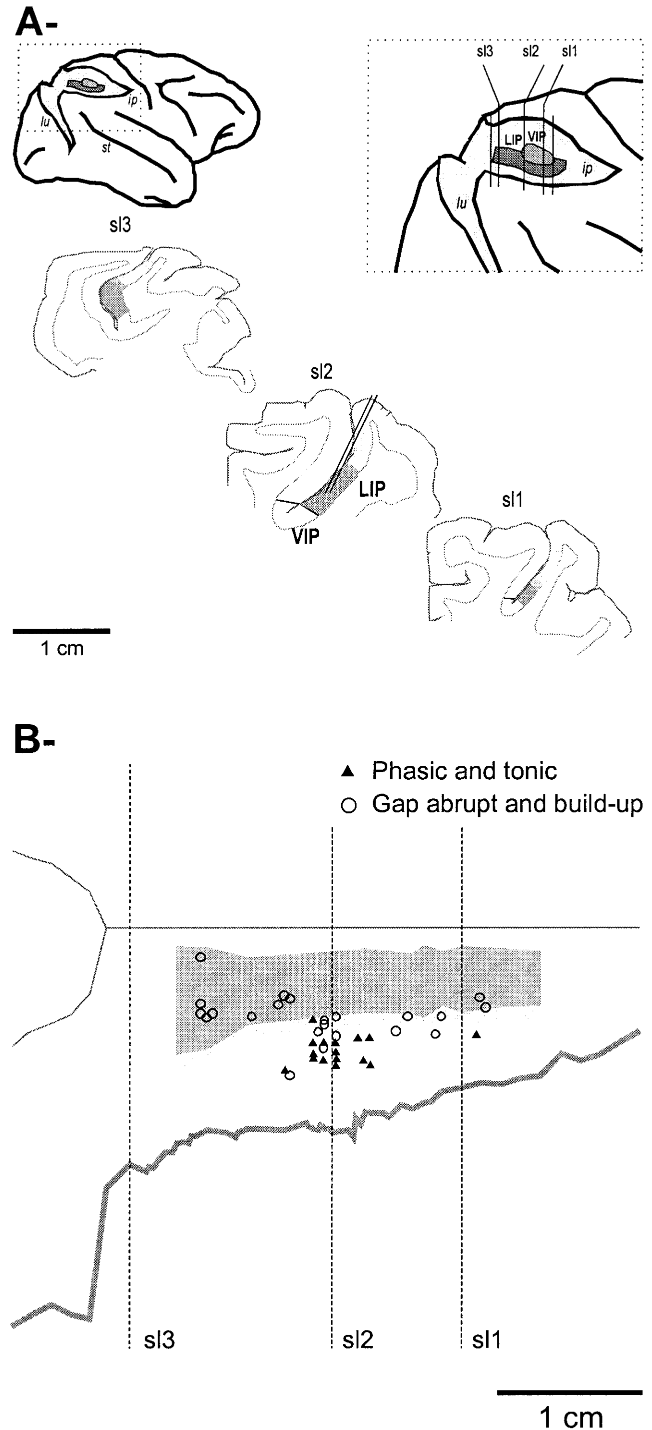

Fig. 12A, B Histological reconstruction of the recording sites along the lateral bank of the intraparietal sulcus. **A** Drawing of one of the recorded hemispheres (*top left*), enlarged view of the opened-up intraparietal sulcus (*right inset*). Limits of chamber position are indicated by the *extreme left and right straight lines*, locations of sections *sl1*, *sl2*, and *sl3* (*lower*) by the other lines. On each of these three quasi-coronal sections, the lateral intraparietal area (*LIP*) is in *gray* and reconstructed electrode tracks in *black*. **B** Flattened representation of the lateral bank of the intraparietal sulcus. IPS fundus is indicated by the *top straight horizontal line*. The *dark gray area* corresponds to the densely myelinated portion of the lateral bank of the IPS proposed to be equivalent to LIPv, the *light gray* is the putative transition zone between LIPv and LIPd. The level of sections *sl1*, *sl2*, and *sl3* are indicated. Sites where the different functional subclasses of neurons were recorded are indicated by *symbols*. (*VIP* Ventral intraparietal area)

tency consistent with a foveal visual response to the reappearing saccade target and frequently had a large phasic component. As in the fixation task, this response was significantly enhanced relative to the presaccadic fixation period. This enhancement could be similar in magnitude to the postgap activity level in the fixation task (12/27), smaller (3/27), or larger (12/27). In a majority of cases the postsaccadic activity was not spatially specific. The finding that cells which respond after a saccade could also show a response after a mere blink of the fixation target in the absence of any eye movement may lead to a reconsideration of the significance of the postsaccadic responses previously reported in parietal neurons. In 9 neurons, the postsaccadic activity varied significantly as a function of saccade direction (see Fig. 6C for an example). For the latter cells, the question is whether this postsaccadic activity occurred as a result of the saccade itself or as an eye position-modulated visual response. Indeed, it is also well known that many parietal neurons display firing-rate modulations as a function of orbital eye position, and the level of activity of tonic cells after a saccade could express this phenomenon. The present data cannot distinguish between the two possibilities.

Topographic distribution of recorded neurons in area LIP

One of the monkeys is still used in ongoing experiments. In the second monkey, after recording sessions were terminated, microlesions were made at specific locations in the lateral banks of the intraparietal sulci of both hemispheres, and the brain was subsequently processed using standard histological techniques. Two-dimensional, flattened reconstructions of the intraparietal sulcus were obtained from digitized, thionine-stained coronal sections, using software developed in our laboratory. On the basis of penetration records, we plotted the estimated recording sites where cells in each of the different categories were found (Fig. 12). Consistent with the RF distribution and the coarse topography previously described in area LIP (Ben Hamed et al. 2001a), tonic and phasic neurons responding during fixation were concentrated above the densely myelinated zone of the lateral bank of the IPS, in the dorsal part of area LIP, which seems to correspond to the anatomically defined LIPd subregion. In contrast, neurons with buildup or abrupt-onset of activity appearing during the gap interval were distributed across both the dorsal and ventral regions of area LIP.

Discussion

The activity of LIP neurons is strongly modulated by attentional (Colby et al. 1996; Platt and Glimcher 1997), memory (Gnadt and Andersen 1988), decisional, and movement parameters (Barash et al. 1991a, 1991b; Duhamel et al. 1992; Mazzoni et al. 1996; Platt and Glimcher 1998), as well as by static parameters such as eye position and vergence (Andersen et al. 1985; Bremmer et al. 1997; Gnadt and Beyer 1998; Gnadt 2000), in relation to the role of LIP in encoding visual targets for saccades and in saccadic behavior itself. We have described neuronal responses in LIP that cannot be interpreted exclusively in terms of saccade programming, since recordings were made essentially during simple fixation tasks. Different categories of neurons were distinguished on the basis of their pattern of responses during the gap fixation paradigm. In the following, they will be discussed with regard to their possible functional significance and to the similarities and differences with cells identified in other related cortical areas. The close relationship between the spatial attention dynamics and the oculomotor behavior will then be stressed.

What does LIP say when the eyes are fixed?

Visual phasic and tonic activities

Responses were mainly determined by stimulation of a foveal RF. These neurons were phasically activated by transient states of the fixation spot (appearance and/or disappearance) or were tonically activated by its presence and became silent during the gap and the intertrial interval. This visual activity was modulated by nonretinal factors and was often enhanced in the interval between the reappearance of the fixation target and the reward delivery. This amplification could show on the phasic and/or tonic component of the response. These cells showed no saccade-related responses for the range of studied saccades (very small parafoveal saccades were not tested). Histological reconstruction showed the cells to be essentially situated in LIPd.

Tonic-fixation activities

Cells discharged during fixation in both the presence and the absence of a fixation target, indicating that part of their activity was not due to retinal stimulation. These neurons were also confined to LIPd. Such cells had already been described in parietal cortex (Lynch et al. 1977; Sakata et al. 1980), as well as in FEF (Bruce and Goldberg 1985; Burman and Bruce 1997), supplementary eye field (Schall 1991; Bon and Lucchetti 1992; Schlag et al. 1992; Lee and Tehovnik 1995), prefrontal cortex (Suzuki and Azanuma 1977), and several subcortical structures (Hikosaka and Wurtz 1983; Schlag and Schlag-Rey 1984; Matsumura et al. 1992; Ma 1996).

Several possible sources can contribute to sustained extraretinal response. The first is an eye-position signal. The resting activity of many neurons in the parietal cortex is modulated by eye-position signals (Andersen et al. 1985, 1990b; Bremmer et al. 1997). This cannot account for the present findings because of the criteria on which this tonic activity is defined.

A second possibility is that this activity is an oculomotor fixation signal. Rostral SC has been characterized as a fixation zone (Munoz and Wurtz 1992, 1993b). Neurons in this region are active during fixation in total darkness, and pause completely or partially during saccades (Munoz and Wurtz 1993b). About half of the LIP neurons we tested during both fixation and saccade tasks exhibited a similar pattern of activity. However, the mean latencies of initiation and termination of the perisaccadic inhibition of LIP neurons is only loosely related to the onset and termination of saccades as compared to the SC and pons omnipause cells. The onset of this inhibition is leading in LIP, suggesting that it might contribute early in the process of saccade generation to attentional disengagement or fixation unlocking. The reciprocal is not true, since the perisaccadic pause terminates more than 70 ms later in LIP than in the two other structures, and thus cannot have a functional causal link with the reengagement of fixation. Monosynaptic connections from LIP to the saccade-related regions of the SC have been described (Paré and Wurtz 1997), but there is no direct evidence as yet of connections between the rostral SC and LIPd, where the foveal cells and the "fixation cells" described here are found.

A third possibility is a signal of cognitive origin, subserving the maintenance of a memory representation of a behaviorally relevant location (here, the center of the visual field). Sustained activity during the delay of a memory-guided saccade task in LIP has been suggested to reflect a motor plan for the upcoming saccade or intention (Gnadt and Andersen 1988; Bracewell et al. 1996; Mazzoni et al. 1996). But LIP neurons also maintain their response to a static stimulus in their RF after its disappearance, which is truncated when a saccade brings the location of the stimulus outside the RF (Kusunoki et al. 1997). This is interpreted as a memory representation of the stimulus. Tonic-fixation cells could reflect such a phenomena in foveal or parafoveal RFs.

Buildup activities

Activity is triggered by the disappearance of the fixation point but builds up progressively in time, culminating at or just before the reappearance of the fixation point. On memory-guided saccade tasks, these neurons have a characteristic response pattern, having a visual RF and discharging during the delay period for saccades into the RF. In addition, they respond postsaccadically in all directions.

"Buildup cells" were first described in the SC during gap saccade tasks (Munoz and Wurtz 1995a). Their buildup progresses into a saccadic burst if the target is in the neuron's movement field but is truncated if the target falls outside. This spatially nonspecific signal has been interpreted as a general saccade-facilitation/fixation-disengagement signal. Although we did not use a gap saccade task, the presence of a buildup signal in the gap fixation task suggests a possible functional relation between SC buildup cells and the neurons described here. Interestingly, Paré et al. show monosynaptic projections from LIP to the SC, and suggest that LIP cells preferentially project on *buildup* SC cells (Paré and Wurtz 1997).

Abrupt-onset gap-triggered cells

Likewise, abrupt-onset gap-triggered cells respond to foveal events, but in a very abrupt way. They can also respond to perifoveal and peripheral stimuli, and their inhibitory foveal responses and excitatory peripheral ones define nonhomogeneous RFs, whose functional significance is unclear (see Fig. 10). The response to the absence of foveal stimuli is tonic, even for long gap durations, but is enhanced during fixation as compared to spontaneous activity in darkness, suggesting a modulation by attention and/or fixation. These cells discharged during memory-guided saccades and their activity varied as a function of saccade direction. Their temporal dynamics differed from that observed during fixation. This suggests once again a complex interplay of modulatory signals related both to fixation and to saccadic behavior.

Postgap and postsaccadic enhancement

Amplification of responses following the reappearance of the fixation point and the acquisition of a new fixation target by a saccade was a frequent observation. In saccade tasks, such amplification is essentially independent of the direction of the upcoming saccade. The fact that the monkeys reacted faster to a change in the color of the fixation target, when a gap interval predicted the approximate timing of change, suggests that this postgap enhancement could be the correlate of a heightened focal attention or a reward expectation.

The concomitant presence of postsaccadic discharges suggest that this amplification could also be a correlate of fixation reengagement, preventing a new saccade to be executed during a period dedicated to visual analysis. This amplification pattern has been described in other structures such as SC (Lynch and Graybiel 1985) and FEF (Bruce and Goldberg 1985; Bruce et al. 1985; Goldberg and Bruce 1990). A similar role for amplification to the one given here has been suggested (Dias and Bruce 1994).

Toward reconciling attentional processing and oculomotor programming in LIP

Studies of rostral and caudal SC reveal an orderly, reciprocal neural mechanism used to control oculomotor exploration by means of fixation and saccades (Munoz and Wurtz 1992, 1993a, 1993b, 1995a, 1995b; Dorris et al. 1997). The dynamics of this fixation-saccade-refixation sequence is reflected in the timing of the responses of fixation, buildup and burst SC neurons. In LIP, a number of previous investigations have mostly emphasized neu-

rons with peripheral RFs studied during memory-guided saccades (Gnadt and Andersen 1988; Barash et al. 1991b; Colby et al. 1996). These neurons commonly discharge to a stimulus flashed in their RF, to the corresponding saccade made later in total darkness and during the intervening delay, and therefore must be carrying multiple signals related to sensory events, movement planning, and execution. Other studies have shown that LIP neurons also show strong modulation by selective spatial attention, independent of eye movement performance (Gottlieb et al. 1998) These studies show that the timing of the specific saccade-related signals is unrelated to saccade dynamics. Here, we have described in LIP two main categories of cells in relation to their response pattern on fixation and on gap-fixation: (1) cells that maintain a tonic response during fixation in both the presence or the absence of a foveal visual stimulus; and (2) cells that respond specifically during the fixation in the absence of a visual stimulus. The first class of cells that we hypothesize to take part in the oculomotor fixation behavior are essentially found in the most dorsal portion of LIP (LIPd) and show a high degree of inhibition on saccadic execution. The second class of cells that we hypothesize to take part in the process of engagement and disengagement of attention are mostly found in the ventral portion of LIP (LIPv) and have both a perifoveal or peripheral visual and oculomotor field. The dynamics of the responses of these two classes both to the disappearance of the fixation point while fixation is maintained (see Fig. 4) and to saccade execution suggest the existence of an oculomotor-attentional network between LIPd and LIPv subserving the function of coordination eye movement patterns to visual analysis. Since reversible lesions of LIP in monkeys produce only subtle impairments of oculomotor behavior (Li et al. 1999) but clear attentional deficits such as extinction or visual search impairments, even when no oculomotor deficits can be detected (Wardak et al. 2002), we suggest that this oculomotor-attentional coordination is a central function of LIP in monkeys.

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