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Saccade-Related Activity in the Lateral Intraparietal Area I. Temporal Properties; Comparison With Area 7a

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SUMMARY AND CONCLUSIONS

1. The cortex of the inferior parietal lobule (IPL) contains neurons whose activity is related to saccadic eye movements. The exact role of the IPL in relation to saccades remains, however, unclear. In this and the companion paper, we approach this problem by quantifying many of the spatial and temporal parameters of the saccade-related (S) activity. These parameters have hitherto been largely unstudied.

2. The activity of single neurons was recorded from *Macaca mulatta* monkeys while they were performing a delayed-saccade task. The analysis presented here is based on 161 neurons recorded from the lateral intraparietal area (LIP), a recently defined subdivision of the IPL; and 54 neurons recorded from the neighboring part of the IPL, area 7a. Overall, 409 IPL neurons were isolated in this study.

3. The typical activity of IPL neurons during the delayed-saccade task has three basic phases: light sensitive (LS), memory (M), and S. These basic phases are common to neurons of both areas LIP and 7a. In each phase (LS, M, and S), individual neurons may or may not be active. Most LIP neurons, however, are active in more than one phase.

4. To compare the activity levels of different neurons, the actual firing rate was weighted by each neuron's background level, yielding an "activity index" for each neuron, in each phase of the task. We calculated the activity index for the LS and M phases and for three phases related to the saccade: a presaccadic (Pre-S), a saccade-coincident (S-Co), and a postsaccadic (Post-S) phase. For area LIP neurons the median values of the activity index were high for the LS, M, Pre-S, and S-Co activities, and slightly lower in the Post-S period. In area 7a the median values were low for the LS phase and, in particular, for the M and Pre-S phases, somewhat higher coincident with the saccade, and high post-saccadically.

5. In area LIP, in each phase, 49–63% of the neurons had excitatory activity, and 10–17% had inhibitory responses.

6. In contrast, in area 7a excitatory responses were most frequent in the Post-S phase (56%). Excitation was particularly infrequent during M (28%) and Pre-S (22%). The incidence of inhibitory responses varied too (4–18%). The time course of inhibition was roughly opposite that of excitation; the highest frequency of inhibitory responses occurred during the saccade.

7. The latency of the S activity was defined as the time, relative to the beginning of the saccade, when the activity became significantly higher than background. In area LIP, latencies ranged from 200 ms before the saccade to 200 ms after the saccade. The mean latency in area LIP was 10.5 ms before the saccade. In area 7a the latencies ranged from 50 ms before the saccade to 380 ms after the saccade. The mean latency in area 7a was 140 ms after the saccade.

8. A neuron was considered presaccadic if its activity varied significantly from background by the time the saccade started. In other words, the latency of the neuron's S activity had to be ≤ 0 ms. In area LIP, 72% (61/85) of the neurons were presaccadic. In area 7a, only 18% (6/33) of the units were presaccadic.

9. The offset time of the saccadic activity was defined as the time the activity outlasted the saccade. The S activity usually lasted well beyond the saccade. The median offset time for LIP neurons was 120 ms. The median offset time for 7a neurons was 300 ms (but note that the S activity of most area 7a neurons only started postsaccadically).

10. The duration of the S activity was computed from its latency and offset and from the duration of the saccadic movement (typically 60 ms). For both areas LIP and 7a the distribution of durations was approximately Gaussian. The parameter values of the distributions were also similar for both areas (median 210 ms in both LIP and 7a). Thus the duration of the S activity is usually considerably longer than the duration of the saccade itself.

11. In area LIP the onset latencies of the LS activity ranged from 50 to 270 ms, with median 110 ms. In area 7a, although the range was similar to that of LIP, typically the LS latencies were longer (median 160 ms).

12. In summary, area LIP contains vigorous activity in anticipation of the saccade, during the LS, M, and Pre-S periods. Area 7a is active mainly during and after the saccade. These results suggest that area LIP participates in the planning of saccades. Area 7a, however, probably primarily subserves different functions.

INTRODUCTION

Ample evidence suggests that the inferior parietal lobule (IPL) of primates plays a role in visually guided saccadic eye movements. Lesions to the IPL in humans often produce deficits in these movements. These deficits are neither purely sensory nor purely motor in origin. Balint, the first to describe these deficits, coined the phrase "psychic paralysis of gaze" (Balint 1909; see Husain and Stein 1988). Similar deficits have also been observed after IPL lesions in monkeys [for reviews of these deficits in humans and monkeys see Andersen (1987) and Hyvarinen (1982)]. These deficits in saccades are similar in strength to those that follow frontal eye-field lesions; combined lesions result in much more severe deficits (Lynch et al. 1986; Lynch and McLaren 1989). Saccades can be evoked by low-threshold electrical stimulation of the IPL (Shibutani et al. 1984). Electrophysiological studies of the IPL have revealed saccade-related (S) single-unit activity (Hyvarinen and Poranen 1974; Lynch et al. 1977; reviewed in Andersen 1987).

In spite of the general acceptance of the involvement of

the IPL in saccades, its exact role remains unclear. A major source of ambiguity has been the insufficient determination of the sensory and motor components of the functions of the IPL. For instance, parallel to their effect on saccades, parietal lesions often result in a deficit in visual attention (Critchley 1953). A confounding factor for these distinctions may be the subtle sensorimotor interactions that are at the core of visually guided saccades. Although visually guided saccades are movements, and must be planned and executed like any other movement, they are both initiated by and affect visual perception. Also, saccades are intimately tied to visual attention.

The ambiguity of the sensory and motor components has not been entirely resolved by electrophysiological studies. Mountcastle and his colleagues proposed that the IPL contains "a command apparatus for the behavioral acts of manual and visual exploration of the immediately surrounding extrapersonal space" (Mountcastle et al. 1975). An alternative hypothesis, however, was offered by Robinson et al. (1978), stating that the IPL is a sensory association area. Robinson et al. maintained that the movement-related IPL neural activity was not motor but sensory; that is, that presaccadic activity in visually guided saccades is a visual response to the target stimulus for the saccade.

To distinguish between the sensory and motor components of the responses of IPL fixation and saccade neurons, Andersen et al. (1987) introduced new paradigms to the study of the IPL. This and other studies led Andersen (1987) to propose that a major function of the IPL is sensorimotor integration. One paradigm was the delayed saccade, in which the visual and S responses are separated in time. Many IPL neurons were found to have both responses. Hence the IPL carries not only visual but also oculomotor-related signals. However, only a small minority of the neurons reported by Andersen et al. (1987) had oculomotor activity that preceded the eye movement. Thus most of the S activity that was reported could not reflect motor planning, because it started after the movement.

Recently, anatomic studies of the IPL have shown that it contains at least two distinct areas. Andersen et al. (1985) defined a new area in the caudal third of the lateral bank of the intraparietal sulcus [lateral intraparietal area (LIP)]. The remainder of the caudal IPL, area 7a, is restricted to the gyral surface. Area LIP has much stronger connections than area 7a to established saccade centers: the superior colliculus, the frontal eye field, and dorsal and dorsolateral pons (Andersen et al. 1990a; May and Andersen 1986). These connections suggest that the subdivision of the IPL that is most likely to be involved in saccades is area LIP. The five presaccadic neurons found by Andersen et al. (1987) were, indeed, all located in LIP.

The electrophysiological differences between areas LIP and 7a have not been studied. The anatomic connections of these areas lead to several important questions: is area LIP specifically related to movement planning? A necessary condition for an area to participate in the planning of saccades is to have neurons that are presaccadic. Considering this condition, we ask, first, if there is a difference between areas LIP and 7a in terms of the latencies of the S activity. Second, are the patterns of neural activity, evoked during the delayed-saccade task in areas LIP and 7a, similar or different from each other? Third, is there, in either area, sensorimotor integration taking place on the level of the single neurons? Fourth, are there differences between these areas in terms of the visual response, in particular, its timing? Areas LIP and 7a are known to have different connections with the extrastriate visual cortical areas (Andersen et al. 1990a), suggesting that their visual response latencies may be different. These and other issues are addressed in this paper by examining, in detail, the response of area LIP neurons in a memory saccade task. This task was designed to tease apart activities related to the visual stimulus, the planning of the eye movement during the memory (M) period, and the eye movement itself.

Preliminary reports of parts of this work have been published elsewhere (Andersen and Gnadt 1989; Barash et al. 1988, 1989; Gnadt and Andersen 1988).

METHODS

Animals, surgery, and animal care

Two 5-kg male *Macaca mulatta* monkeys were used in this study. Before the beginning of training, a head post was mounted in a dental acrylic skull cap, and a scleral search coil was implanted (Judge et al. 1980; Robinson 1963). A second surgical procedure took place when the animals became proficient in their task; in this procedure a recording chamber was mounted over the posterior parietal cortex (Brodmann's areas 5 and 7). Four to 8 mo later, in a third procedure, a chamber was mounted over the posterior parietal cortex of the second hemisphere. All surgical procedures were carried out under general anesthesia [10 mg/kg im Ketamine followed by 10 mg/kg iv pentobarbital sodium (Nembutal)] and with sterile conditions. An analgesic was given postoperatively. The postoperative care consisted of close monitoring of the animal until after full recovery from the anesthesia. After each surgical procedure the monkeys rested for a week and were treated with systemic antibiotics.

During the training and recording periods the animals were deprived of water in their home cages. We carefully monitored the amount of apple juice received by the animal as reward; if the full daily ration was not reached, it was supplemented by water after the sessions. Each week the animals had at least two days of rest, with unrestricted water supply.

During the recording period the recording chamber was flushed daily with sterile saline, and antibiotics were applied to the dura before closing the chamber for the night. Occasionally the animals were lightly anesthetized with Ketamine, and their dura was scraped to prevent an accumulation of granulation tissue.

Routine veterinarian care was given to the animals. The general well-being of the animals was observed in accordance with National Institutes of Health guidelines.

Experimental setup: data collection

Both training and recording sessions were conducted in a completely darkened room. The animal was monitored continuously with a remote camera and an infrared light source. The monkey was positioned with its head fixed 57 cm in front of a large, featureless back-projection screen. Stimuli appeared on the screen as 0.5° diam spots, 45 cd/m^2 against a totally dark background. Stimulus timing and location were controlled by fast-response electronic shutters and x,y galvanometer mirror systems under computer control. To correct for the error in eccentricity caused by the use of a tangent screen, the computer adjusted the command to the x,y galvanometers so that stimuli with the correct eccentricities were displayed to the animal.

Eye position was measured with the use of a scleral search coil system (Judge et al. 1980; Robinson 1963). Eye position was typi-

cally sampled at 500 Hz; in some cases 100 Hz. Each daily session was started by a calibration run, in which the animal fixated stimuli presented at nine locations. These locations, in degrees, were $(x,y) = (20i, 20j)$ where $i,j = -1,0,1$, and (0,0) is the animal's fixation point. These locations will henceforth be called "20° grid." Linear approximations for the horizontal and vertical eye-position signals were calculated from this calibration run. Eye-position calibration varied from day to day by, at most, a few percent.

Spike recordings were made with platinum-iridium electrodes covered with glass, with 1–5 MΩ impedance at 1 KHz. The electrodes were advanced through the dura into the parietal cortex with the use of a Chubbuck microdrive with 1-μm resolution in depth. Signals were fed into an amplifier, and single units were isolated with the use of a variable-delay window discriminator. The time of spike acceptance was determined by the computer with 0.1-ms resolution.

Training: delayed-saccade task

One week after the first surgery (for the acrylic skull cap and the eye coil), training was begun. Initially the animals learned to fixate a fixation spot, and then to make visually guided saccades (in which the onset of a peripheral target coincided with the offset of the fixation spot). Subsequently, the animals were trained to withhold their saccade in the delayed-saccade paradigm. Finally, they were trained in the other paradigms used in this and related studies (Andersen et al. 1990b; Barash et al. 1991). Throughout all training and recording sessions, the heads were fixed. After a successful trial the monkeys received as reinforcement a drop of apple juice. The monkeys performed 1,000–3,000 trials daily, usually 4 days a week.

The experiment was controlled by a laboratory computer. Trials of the delayed-saccade task were organized in the following way (illustrated in Fig. 2): the appearance of a fixation spot signaled to the monkey the beginning of a trial. The monkey had to fixate the spot within 1 s. The moment the eye foveated, the fixation spot was called "fixation onset." If the monkey failed to fixate the spot within the given 1 s, the trial was aborted, declared a "miss," and an intertrial interval was begun.

At a specified time, usually 800 ms after fixation was attained, a peripheral target spot appeared on the screen. The target stimulus was presented briefly (typically 300 ms, occasionally 500 ms), and then disappeared. The fixation spot was retained through the peripheral stimulation, and the monkey was not allowed to break fixation. After the peripheral target spot was extinguished, the fixation spot remained on the screen for some time. The interval from the offset of the target to the offset of the fixation spot is called the "delay" period. Its duration was usually 400 or 2,000 ms. During the delay period the monkey was required to continue to fixate the fixation spot.

The offset of the fixation spot served as the "go" signal for the saccade. Within 500 ms the monkey had to move his eyes into a window centered on the location at which the peripheral target had previously appeared. After this eye movement the monkey had to keep his eye in the new position for at least 500 ms. If the monkey fulfilled all these conditions, the trial came to a successful end (a "hit"), and the animal was rewarded with a drop of apple juice. If a trial had reached the fixation onset stage but subsequently the monkey either moved his eyes before the go signal, or did not enter the target window soon enough after the go, or moved out of the target window before 500 ms, the trial was aborted and declared an "error."

The subsequent intertrial interval was a pause (randomly varied from 500 to 2,000 ms), during which the collection program accomplished bookkeeping and data display tasks.

Data was collected in "runs." A run was a sequence of trials from nine classes, the order of which was randomly interleaved. Trials of the same class had identical stimulus presentations and behavioral requirements. In the delayed-saccade task, eight of the nine classes had the temporal pattern of stimulation and behavior that was described above. The only difference among the classes was the location of the stimulus. The target could be presented in any of the eight locations on a 15° grid (described in the previous section), that is, in any of eight directions separated by 45°. The ninth class was a control class, and it is described next.

Because the go signal in the protocol described above was the offset of the fixation spot, if a neuron had a (visual) foveal off response, this visual activity would have occurred just before the saccade and might have thus been mistakenly considered saccade related. To preclude this possibility the following protocol was applied in the ninth class: as in the other classes, trials in this class began with the onset of the fixation spot. The monkey had to fixate the spot within 1 s; otherwise, a miss was declared and the trial aborted. After fixation onset the fixation spot stayed on for the same duration as the other classes, and the monkey had to continue to fixate it. However, no peripheral target was flashed during this fixation, and after the offset of the fixation spot, the monkey had to keep his eye within the fixation window for another 500 ms. If the monkey broke fixation before this period was over, the trial was declared an error. One of the following two variations of the described basic paradigm was used. In the first variation (see Fig. 2*B*), after the dark fixation, the fixation spot reappeared for another 500 ms. The monkey had to continue fixating the spot as long as it was on. This variation allowed us to examine whether the neuron had a foveal on response. In the other variation, from 800 to 1,100 ms after key down, the target projector flashed at location (0,0), overlapping the fixation spot. The monkey had to maintain his fixation for the whole duration of the trial. In this case the total illumination in the control trials was the same as that of the delayed saccades. If a fixation-off response was observed, the neuron was not further analyzed.

Another piece of evidence that the saccade response was not due to the offset of the fixation point was that almost all saccade responses were selective for the directions of the eye movements. If the cells were simply responding to the offset of the fixation light, they would have responded to all subsequent directions of eye movements.

Once a neuron was isolated, it was studied in a run of delayed saccades, usually with 400-ms delay. The run continued until 8–10 hits were collected in each class. Additional types of runs, including delayed saccades with longer delays, were subsequently recorded (see Andersen et al. 1990b; Barash et al. 1991).

The spatial windows used for determining adequate eye position were squares with side length ±5–10° centered on the fixation spot or on the target. The reason for these relatively large windows is that delayed saccades made in complete darkness exhibit systematic spatial errors. In particular, up-going saccades tend to be hypermetric, whereas down-going saccades are almost invariably hypometric. These effects are described in detail by Gnadt et al. 1991. This contrasts with the accuracy of visually guided saccades, for which virtually flawless performance could be achieved with ±1.5° side-length windows.

Histology: allocation of units to cortical areas

After recording from both hemispheres, one monkey was killed. In the last few weeks of the experiment involving this monkey, several marking lesions were made in both hemispheres by passing small DC currents through the recording electrode at different depths. At the conclusion of the experiment, the monkey was given an overdose of pentobarbital sodium and then perfused transcardially with heparinized saline, followed by buffered Formalin. Guide wires were lowered into the brain at selected record-

ing-chamber coordinates immediately after the animal was killed. The wires were used as landmarks for blocking the posterior parietal cortex and for determining the locations of the recording tracts made early in the experiments. Good agreement was found between the locations of the guide wires and the coordinates of the marking lesions, indicating that the locations of the early recordings (determined from the coordinate system of the microdrive) were reasonably accurate.

Thirty-micron-thick sections were cut and alternately stained with thionin for cytoarchitecture and by the Gallyas method for myeloarchitecture (Gallyas 1979). Areas within the IPL were identified on architectural and physiological criteria. In particular, we differentiated between areas LIP and 7a primarily on myeloarchitectural grounds (see Andersen et al. 1990b for details).

One hemisphere was recorded from a second monkey. This animal is currently being used in related experiments. We defined a functional criterion for determining whether a given neuron belongs to area LIP or to area 7a. This criterion was based on the analysis of the results from the sacrificed monkey, and on data from previous monkeys not included in the present study.

We recorded many neurons from the gyral cortex, that is, with depth less than 2,500 μm from the top of neural activity, at different chamber coordinates. Because the cortex of the superior parietal gyrus (area 5 of Brodmann) is somatosensory, whereas the cortex of the IPL is visual oculomotor (Hyvarinen 1982; Mountcastle et al. 1975), we were able to draw with relative precision the line of the sulcus on the chamber map. Neurons that were lateral to this line, and whose depth was at most 2,500 μm, were classified to area 7a. Because of the inclination of the recording chamber, penetrations that reached area LIP had either to pass through area 5 or to descend along the sulcus. If the electrode passed through area 5, a subsequent completely "quiet" zone followed (without neural activity), as the electrode traversed the intraparietal sulcus before entering area LIP. On entering LIP, visual or oculomotor neurons were found. This zone was presumably the sulcus. The subsequent cortex, at least 2,500 mm under the top of neural activity, was considered area LIP.

Figure 1 shows the sites of penetrations, in which area LIP and area 7a neurons were encountered, made into the two hemispheres of the first monkey. Representative sections illustrate the estimated locations of neurons recorded in these hemispheres.

Data analysis

The responses we recorded typically depend on many parameters. Each phase can be excitatory or inhibitory (or neither); each phase can have its own preferred direction, latency, and level of activity. Unbiased analysis of the activity of these neurons is therefore quite complex, particularly when groups of neurons are compared (that is, areas LIP and 7a). Toward this end a relational data base was constructed (4th Dimension, Acius). Several analyses were made on the raw data on the laboratory computer, and the results were extracted and imported into the data base. Because this arrangement is not standard, we will briefly describe it.

The data base contained four files important for the present discussion. A "Penetrations" file contained the date and chamber coordinates of each penetration, depth of the dura, and the top of neural activity, electrode impedance, intervals in the track of the electrode suspected as white matter, and several additional parameters. A "Cells" file, linked to the Penetrations file, included primarily the depth in which each neuron was recorded, its run list, and evaluation of the quality of the recording. A "Fields" file, linked to the Cells file, had a record for every run that used the delayed saccade task; the record contained 104 parameters imported from the laboratory computer (11 phases times 9 parameters for each phase, plus some additional data). Only the parameters that were found to be interesting are described here. A fourth file, "Timing," also linked to the Cells file, contained light-sensitive (LS) and S latencies, duration and offset of the S activity, and additional parameters for each cell. Selections of neurons (such as "area LIP neurons") were exported to various statistical and graphical Macintosh programs. In particular, we used Data Desk (Odesta) for data exploration. We feel that this arrangement helped us reach reliable quantitative characterization of the activity in LIP.

Definition of activity index

To compare levels of activity evoked in different neurons, we decided that we must take into consideration that the compared neurons typically differ not only in their "responses," but also in their background levels. Hence, to compare "net responses", we first assigned to each neuron a "net" level of activity (its activity index), on the basis of its response and background levels. For each neuron, in a given phase, we measured the mean response rates for saccades in the eight directions $r_1, \ldots, r_8$, and the corresponding mean background rates, $b_1, \ldots, b_8$. Each r_i and b_i are the means of at least eight trials in the given direction. We then took as a general mean background the mean of $b_1, \ldots, b_8$. We also determined the maximal rate $r_{max} = \max\{r_1, \ldots, r_8\}$ and the minimal rate r_{min}. Neurons satisfying $r_{max} \geq 1.2 \cdot$ background were considered potentially excitatory, and their activity index was defined in the following manner

$$\text{activity index} = \begin{cases} (r_{max} - \text{background})/\text{background}^{1/2} & \text{if background} \geq 1 \text{ imp/s} \\ r_{max} - \text{background} & \text{otherwise} \end{cases}$$

A good correspondence was found between neurons that were subjectively classified as having a clearly excitatory response and those having an activity index higher than 2.0.

Otherwise (i.e., if $r_{max} < 1.2 \cdot$ background), neurons were considered potentially inhibitory, and their activity index was calculated thus

$$\text{activity index} = \begin{cases} (r_{min} - \text{background})/\text{background}^{1/2} & \text{if background} \geq 1 \text{ imp/s} \\ r_{min} - \text{background} & \text{otherwise} \end{cases}$$

Neurons were usually considered clearly inhibitory if their activity index was -2.0 or less.

Neurons showing excitation in some directions but inhibition in other directions were considered excitatory. Often such mixed responses were in opposing directions ("push-pull"). Careful distinction between the excitatory and inhibitory components of mixed fields is beyond the scope of the present analysis.

An advantage of the activity index is that it is a continuous function. It can be used to select subsamples of units with various "quality" levels of response. We often wondered if our findings would be different if we limited our sample only to the "best" neurons (that fulfilled some criteria). We examined most of the questions discussed in these two papers with higher activity index thresholds. We did not find any significant effects of these different selections. Hence we feel reassured that our results do not reflect an artifact of our method of response selection.

We have also repeated parts of the analysis described in these two papers employing a statistical acceptance criterion, the *t* test used for latency measurements with large bins (>100 ms). The results were consistent with those based on the activity index.

Definition of phases

The activity index was utilized to evaluate the level of activity of the population in several phases. A list of the standard phases used in this paper follows (in a few neurons the times of the slices were

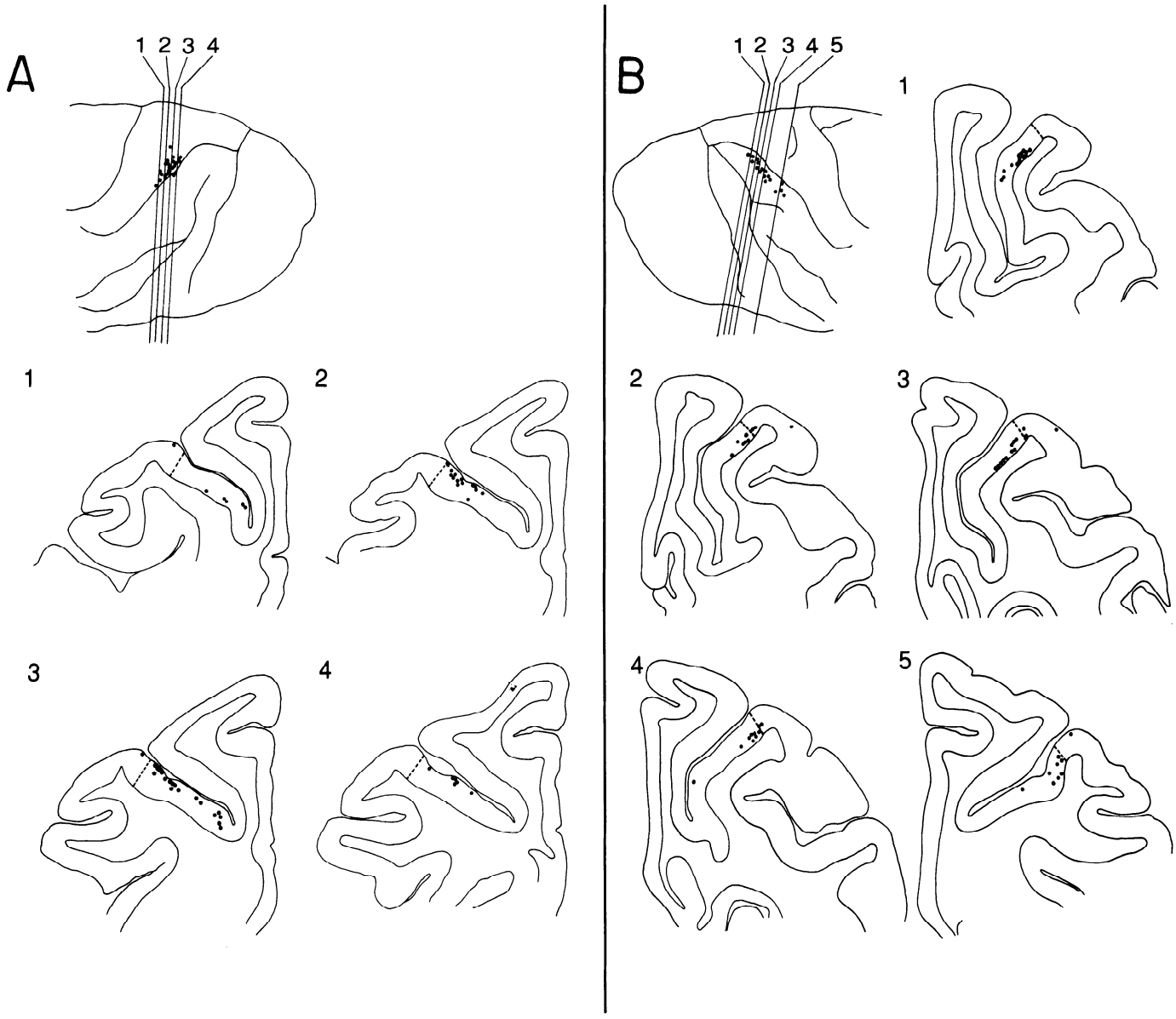

FIG. 1. Reconstructions of the locations of neurons recorded in the left (*A*) and right (*B*) hemispheres of the 1st monkey. Panels on the *top left* of *A* and *B* show the entry points of electrode penetrations. Other panels show the reconstructed locations of recording sites drawn onto representative coronal sections. Dotted lines indicate the border between area LIP, in the bank of the intraparietal sulcus, and area 7a, on the gyral surface.

slightly changed to fit their pattern of activity): *1*) background, 300–700 ms from fixation onset; *2*) LS, 75–275 ms from target onset; *3*) M, 200–400 ms from target offset; *4*) presaccadic activity (Pre-S), 200–25 ms *before* the beginning of the saccade; *5*) saccade-coincident (S-Co), 25 ms before the saccade to 75 ms after the beginning of the saccade; and *6*) postsaccadic (Post-S), 200–500 ms after the beginning of the saccade. The last three phases are based on alignments of the trials on the beginning of the saccade. (Additional phases were tested but are not presented because they did not yield significantly different results from those we do present.)

The phases Pre-S, S-Co, and Post-S, defined above, are fixed with respect to the saccadic movement. Together, these phases cover most of the interval from 200 ms before the saccade to 500 ms after the saccade. Typically, a saccadic discharge lasts for only part of this 700-ms interval; but the timing of the discharge varies from one neuron to another. We refer to this discharge as "saccadic" (S). We investigate some of its attributes (such as latency). Examining S, on the basis of its timing variability, is complementary to the study of the fixed intervals Pre-S, S-Co, and Post-S.

Determination of the start and end of the saccadic movement

Eye position was usually sampled at the rate of 500 Hz. Eye velocity was computed from the eye-position signal with the use of a two-point central difference algorithm modeled as an ideal differentiator in series with a low-pass filter (Bahill et al. 1982; Usui and Amidror 1982). A fluctuation of the tangential eye velocity was recognized as a saccade if it was higher than a threshold, usually 50°/s, for at least 25 ms. In this case the beginning of the saccade was defined as the time at which the velocity increased to >10°/s, and the end of the saccade as the time at which the velocity decreased to <50°/s (to screen out postsaccadic drift).

Determination of the time the LS and S activities begin (or end)

Onset and offset times were determined for the LS and S activities by the use of the same procedure. To simplify its description, we illustrate the procedure for the case of the latency of the S activity (see Fig. 8*A*).

The spike records from a given class (i.e., trials in a single given direction) were aligned on the beginning of the saccade. A "baseline" interval was determined. It was always well before the onset of the activity (for S activity latency, typically from 400 to 100 ms before the saccade). A second, "detect," interval was also defined; it started well before the saccade and continued well after it (for S activity latency, typically from 100 ms before the saccade to 400 ms after it).

Both intervals were partitioned into bins, usually 20 ms long. The number of spikes in each bin, in each trial, was counted. For the baseline interval, all these counts were grouped together into a sampled baseline distribution. The number of samples in the baseline distribution was, thus, (duration of baseline interval/bin duration) × (number of trials in class).

In the detect interval each bin was treated separately. A sampled detect distribution thus had the same number of samples as trials in the class.

Each bin in the detect interval was compared separately with the baseline interval. The means of these two distributions (i.e., baseline and detect) were compared by the use of a standard t test with an α level of 0.05. The programs were adapted from Press et al. (1988). We determined, for each bin in the detect interval, whether its activity was significantly different from that of the baseline interval. Significantly different bins were marked (↑ in Fig. 8*A*).

If the activity starts abruptly (as in Fig. 11*A*), a continuous series of bins was recognized as significantly different from the baseline. In this case the latency of the activity was defined as the midpoint of the first recognized bin (relative to the onset of the saccade). If the rise in activity was less abrupt, or the data more noisy, an isolated bin was occasionally recognized as significantly different from the baseline. Therefore usually only the first recognized bin that was followed by another recognized bin was defined as the latency of the response.

Note that this definition of latency determines when the activity is already significantly different from that in the baseline. It is an upper bound for the time of start of the activity: the activity may begin slightly before its specified latency.

RESULTS

Data base

We isolated 409 single units from two hemispheres of one monkey and one hemisphere of a second monkey. The present analysis is based on 215 units, selected according to the following criteria: *1*) reliable classification of the unit into either area LIP (161 units) or area 7a (54 units); *2*) lack of a foveal off response; and *3*) duration and quality of the recording and the resulting completeness of study.

Activity of IPL neurons in the delayed-saccade task

Figure 2*A* shows activity recorded from an LIP neuron while the monkey was performing the delayed-saccade task. The response of this neuron shows phases of activity that are typical of IPL neurons in this task. Many neurons, however, show only some of these phases.

A target stimulus in the receptive field of the neuron triggers a strong discharge. Such discharge is often maintained

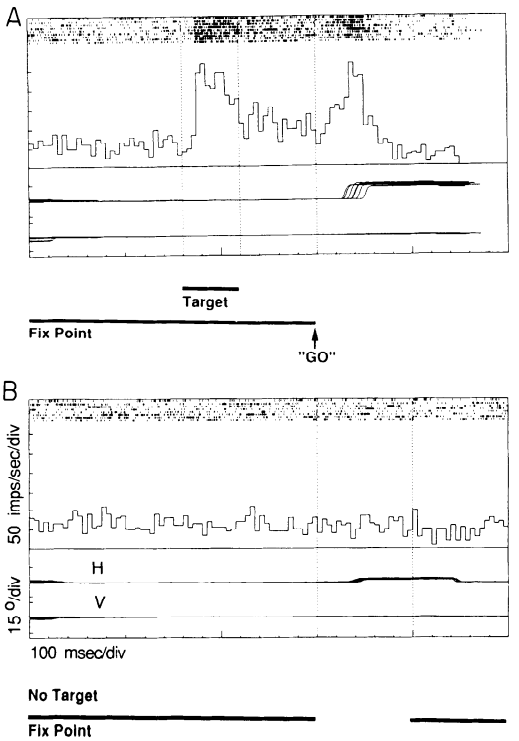

FIG. 2. *A*: sequence of events in a M saccade and a typical response of an LIP neuron. *B*: sequence of events and response of the same LIP neuron to a control task, demonstrating that the S activity is not a response to the offset of the fixation spot. Onset and offset times, for both target and fixation spot, are indicated both in the schematics at the *bottom* of the figures, and by the dotted vertical lines above. Shown, from the *top*, are the spike rasters, where each horizontal trace represents a trial, and each tick within a line marks the time of occurrence of a spike, the resulting histogram (binwidth, 25 ms), and the horizontal and vertical eye-position traces of the various trials, superimposed. Trials are aligned on the sensory events (note variable saccadic latencies). *A* illustrates the 3 typical phases of activity (LS, M, and S) described in the text. Lack of activity in *B* demonstrates that this neuron has neither on nor off visual response to the fixation spot.

until the stimulus is extinguished (as it is in Fig. 2*A*). Because no eye movements are made during this visual stimulation, nor for some time after it, the discharge is LS.

During the delay period, after the target stimulus is turned off, activity often remains elevated above the background level until the saccade is made, although it may be somewhat reduced relative to the LS activity. This phase of activity is called memory (M) because, in this part of the task, the monkey has to remember the location in which the target had previously appeared, and to which, subsequently, he must saccade. Note that during the M phase there is neither visual stimulation nor any eye movement.

Around the time of the saccade, another burst is often observed. In Fig. 2*A* it begins somewhat before the saccade and continues after the saccade is completed. Because the saccade is made in the dark, long after the target stimulus was extinguished, this burst is not visual, and it is termed saccade-related (S). Hence the delayed saccade paradigm allows us to separate temporally the visual and oculomotor responses of these neurons.

Nevertheless, because the go signal (the offset of the fixation spot) is visual, a control has to be made to preclude the possibility that this visual stimulation is the cause of the S discharge. This control is shown in Fig. 2*B*; trials of this

class were always randomly interleaved with the delayed saccades. Here no target stimulus is presented to the monkey before the fixation spot is turned off. The monkey may not move his eyes even after the fixation spot is extinguished. The background activity in Fig. 2*B* remains unchanged when the fixation spot disappears and then reappears. "Fixation-off" responses were observed in a few neurons; these neurons were not further analyzed. None of the neurons discussed in this paper had a fixation-off response.

In conclusion, the delayed saccade task temporally separates the sensory (LS) and motor-related (S) responses. In visually guided saccades, these responses are coincident. In addition, the delayed saccade reveals a new type of activity, sensorimotor memory (M).

Sensorimotor nature of the M activity

Figure 3 shows that the M activity is not simply a long-lasting LS response. Had the M activity been an LS response, it would not be affected by the length of the delay period. Figure 3, *A* and *C*, depicts data from an LIP neuron that was tested with different delay periods (500 ms in *A*, 1,300 ms in *C*). The duration of the M activity in these two cases is clearly different: in the trials of Fig. 3*A*, the activity is cut off at the end of the saccade, whereas, at the same point of time in Fig. 3*C*, the activity is maintained.

In trials of both delay periods (Fig. 3, *A* and *C*), the activity begins with the sensory event and is maintained until the completion of motor act; in both, the saccade is accompanied by activity that subsequently declines to baseline. It follows that the M activity in this neuron is sensorimotor in nature.

Figure 3, *B* and *D*, displays data recorded from a different LIP neuron, showing that the M response can also be inhibitory. For this neuron, too, the duration of the M inhibition is determined by the length of the delay period. Another aspect of the activity of this neuron is a rise in the level of activity that starts at the beginning of the trial and proceeds until the presentation of the target stimulus. Such anticipatory activity was observed in a few neurons. It is, of course, not spatially selective, because it occurs before the appearance of the peripheral target and is thus common to all target locations.

Population profile of the activity

So far, only examples of single neurons have been presented. Next, we ask how is the activity distributed over the population of neurons of areas LIP and 7a. Here we face the following problem: we cannot compare directly the rates of firing of different neurons because their background rates may be different. We must weight the response rate of each neuron according to its own background rate. Toward this aim, we defined in METHODS an "activity index" that is a function of a neuron's response and background rate and quantifies the strength of the response.

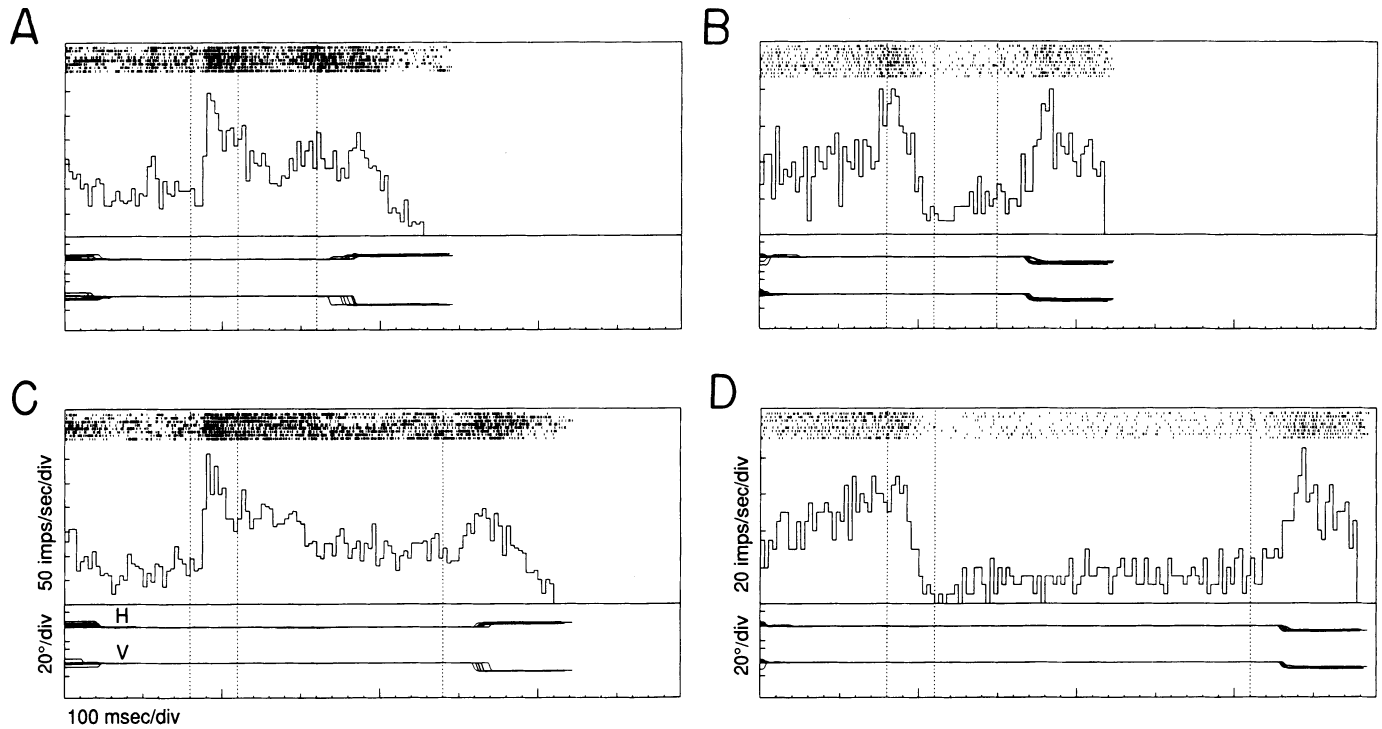

FIG. 3. Delayed-saccade tasks with delay periods of different durations, showing that the M activity is initiated by the visual stimulation and is terminated by the saccade. *A* and *C*: response of an area LIP neuron in M saccade trials with delays of 500 and 1,300 ms. The increased level of activity is maintained throughout the delay period until the saccade is made. *B* and *D*: response of another area LIP neuron, which shows an inhibition of activity during the delay period. The shown M saccades are with delays of 400 and 2,000 ms. M inhibition is maintained until the saccade is made. Shown, from the *top*, are the spike rasters and the histograms; the horizontal and vertical eye-position traces of the various trials are superimposed. Trials are aligned on the sensory events. The vertical dotted lines denote, from the *left*, the onset and offset of the target and the offset of fixation spot.

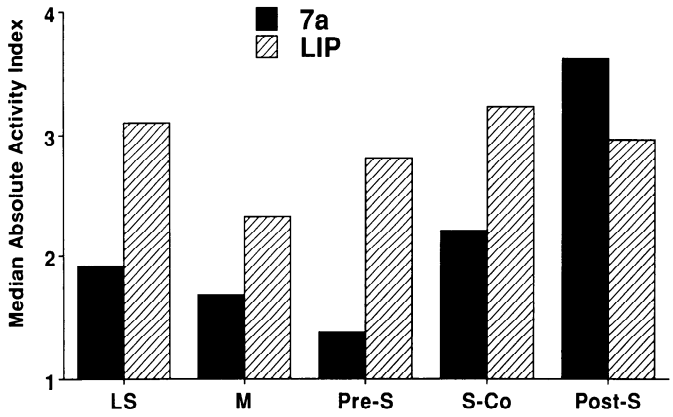

FIG. 4. Median of the absolute value of the activity index is a measure for the overall level of response. It is calculated for each phase of the delayed-saccade task, for neurons from areas 7a and LIP. Activity in area LIP remains relatively high throughout the trial. In contrast, activity in area 7a is low until the saccade is made.

Figure 4 illustrates the median values of the activity index in the various phases of the task. In area LIP the median activity index is high throughout the trial, peaking for the LS and S-Co phases. Note that this result does not imply whether the same or different neurons are active in each phase. This question will be discussed below.

In area 7a, the profile of the median activity index values is very different (Fig. 4). The medians are low for LS, M, and, in particular, for Pre-S. During S-Co the median is somewhat higher; it peaks after the saccade (Post-S). Thus, unlike area LIP, which is active before, during, and after the saccade, area 7a is relatively inactive before the saccade, and activity in it increases during the saccade and peaks after the saccade. This pattern suggests that area LIP may play a role in the planning of saccades, whereas area 7a does not. In the next sections we shall examine this pattern of activity in more detail.

Figure 4 displays the median of the absolute values of the activity indexes, thus reflecting both excitatory and inhibitory responses. Selection of only excitatory responses (positive activity indexes) yields similar results. The means of the activity indexes also show the same characteristics.

It should not be inferred that area 7a has smaller visual responses, in general, than area LIP. Only in this specific task can we say that the visual responsiveness of area 7a is lower than that of LIP. Note also that the Post-S activity in area 7a is high, and thus the low LS and M median activity indexes of area 7a do not reflect a general unresponsiveness of this area.

Background rates

The activity index is lower in area 7a than in area LIP before the saccade because activity is lower in area 7a in these phases, not because of the background rates in the two areas. Figure 5 shows that the background rates in area LIP are indeed somewhat higher than those of area 7a. Because the background rate reflects fixation behavior, Fig. 5 may indicate that fixation activity is stronger in area LIP, at least in the specific conditions of the present task. A similar result was observed in a different series of experiments by Andersen et al. (1990a).

Distribution of the activity in phases and in combinations of phases

The following criterion was used to characterize a neuron as "active": an excitatory response is defined by an activity index ≥ 2.0; an inhibitory response, by activity index -2.0 or smaller (see METHODS for details).

We repeated the analysis of this section with a second criterion: a neuron was considered to have a response if its firing rate was significantly different from baseline, with the use of the t test described in the METHODS section, with long bins (at least 200 ms). Because the results were similar, we present only the results obtained by the first criterion.

Figure 6 shows, for both areas LIP and 7a, the fractions of neurons that had excitatory or inhibitory responses. In area LIP, the fraction of excitatory neurons is similar in all phases (49–63%). During the LS and S-Co phases, excitation is most frequent (both 63%). The fraction of inhibitory neurons in LIP also does not vary much from phase to phase (10–17%). The total fraction of responding neurons, excitatory or inhibitory, was, in all phases, 62–77%. The fraction of inhibitory neurons, out of the total number with excitatory or inhibitory responses, also did not vary much: from 14% (15/106) in the LS to 25% (24/95) in the Pre-S phase (n = 145 for area LIP).

In contrast, in area 7a the fraction of neurons with excitatory responses varied considerably from phase to phase. Before and during the saccade, these fractions were small relative to LIP: from 44% of neurons with excitatory activ-

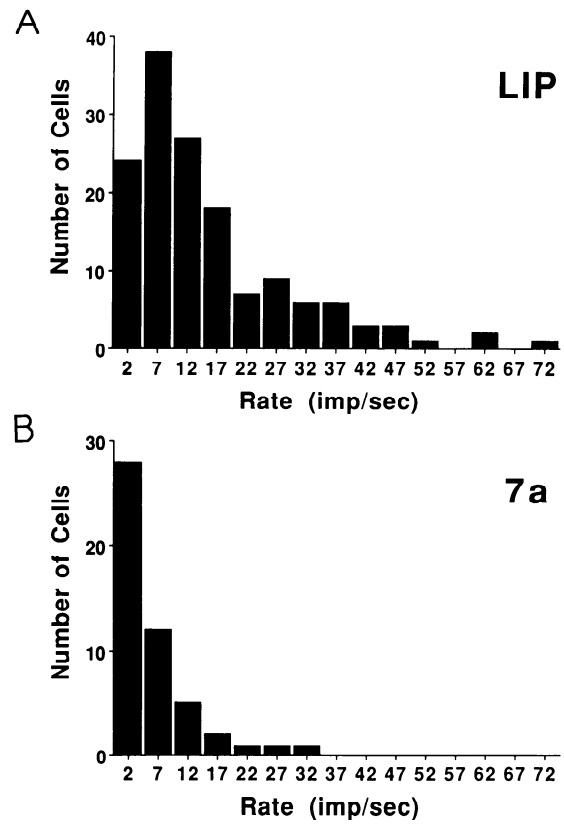

FIG. 5. Distributions of background spike rates for neurons in areas LIP and 7a. The background rate of a neuron is measured during the initial fixation in delayed-saccade trials, before target presentation. Area LIP background rates, thus defined, tend to be higher than those of area 7a.

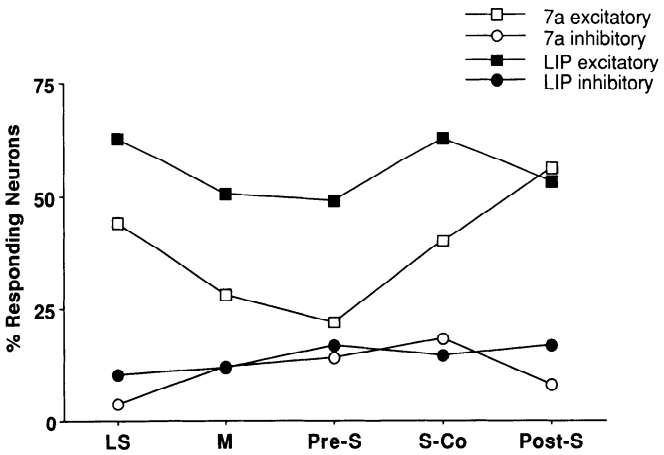

FIG. 6. Percentage of neurons from areas LIP and 7a showing excitatory or inhibitory responses, during each phase of the delayed-saccade task. A neuron was deemed to show an excitatory (or inhibitory) response during a given phase if its activity index during that period was >2.0 (or less than −2.0).

ity in the LS, down during M to only 22% in Pre-S, and up again to 40% for S-Co. Only during the Post-S phase was the frequency of excitation (56%) as high as that in LIP. The incidence of inhibitory responses varied too (4–18%). The time course of inhibition was roughly opposite that of excitation; the highest frequency of inhibitory responses (18%) occurred during the saccade. Consequently, the total fraction of responsive neurons (excitatory or inhibitory) varied from 48% in LS, down to 36% in Pre-S, and up to 64% in Post-S (n = 50). The fraction of inhibitory responses out of the total responding varied in area 7a from 8% for LS (2/24) to 30% for M (6/20), peaking for Pre-S at 39% (7/18), and dropping to 12% (4/32) postsaccadically.

In individual neurons each of the phases of the activity may or may not appear; when they appear, they may be excitatory or inhibitory, and in varying intensities. Figure 7 shows that, for 145 LIP neurons studied, 124 (86%) had excitatory activity in *at least* one phase. Out of these neurons, 83 (67%) had excitatory activity in more than one phase; 48 (39%) were excitatory in LS, M, and S-Co phases. If one also counts the inhibitory phases, these fractions become even larger (65/145 or 45% responsive in the 3 phases). It thus becomes clear that the LS, M, and S activities are integrated in LIP at the level of the single neurons.

A similar analysis to the one of Fig. 7 was not done for area 7a, because most of its neurons have little LS and M activities in this task.

Although it is clear that inhibitory responses occur in the IPL in the delayed-saccade task, we shall not discuss them in detail in the subsequent sections, because their low spike rates make them hard to study quantitatively.

Latency of the S activity

We determined the latency of the S activity with respect to the onset of the saccade in 85 area LIP and 33 area 7a neurons. Neurons with clear, robust S activity were selected for this analysis. The procedure is described in METHODS. Note that negative latency values represent activity starting before the saccade, and positive values represent activity that follows the saccade.

The analysis is illustrated for an area LIP neuron in Fig. 8*A*. The latency determined here was 10 ms. Note that the last histogram bin before the saccade shows that the mean rate has already increased; however, this bin is not recognized statistically. This example illustrates that the latency values obtained in this method are higher bounds: they describe when the S activity has sufficiently developed to be significantly different from the baseline; but the saccadic activity could start earlier, and take time to grow.

Figure 8*B* shows that, in area LIP, the S activity of 61/85 units (72%) has started by the time the saccade begins (these units have latency ≤0 ms). In contrast, in area 7a (Fig. 8*C*) only 6/33 units (18%) have S activity starting by that time.

Even though the distribution of latencies in area LIP (Fig. 8*B*) is centered on the beginning of the saccade, its range is large: −200–200 ms. In area 7a the range of the observed latencies is shifted to later values: −50–380 ms. (Indeed, even higher latencies might exist in some neurons; they would not show in our data because trials were terminated 500 ms after the completion of the saccade.) The distribution of latencies in area 7a is broader; this is reflected in a higher value of its standard deviation (134 ms; cf. 78 ms in LIP).

The mean latency in area LIP is presaccadic (−10.5 ms). The mean latency in area 7a is postsaccadic (140 ms). This difference is statistically significant (2-sample t test with separate variance estimates, t = 6.06, df = 40, null hypothesis rejected with α = 0.01).

Activity offset and duration of the S activity

We determined how long after the end of the saccade the S activity lasted. Figure 9*A* illustrates the procedure. The statistical method used for this comparison is identical to the one used for the measurement of the latencies (see METHODS). Here, the trial traces are aligned on the end of the saccade. The detect interval begins at the end of the

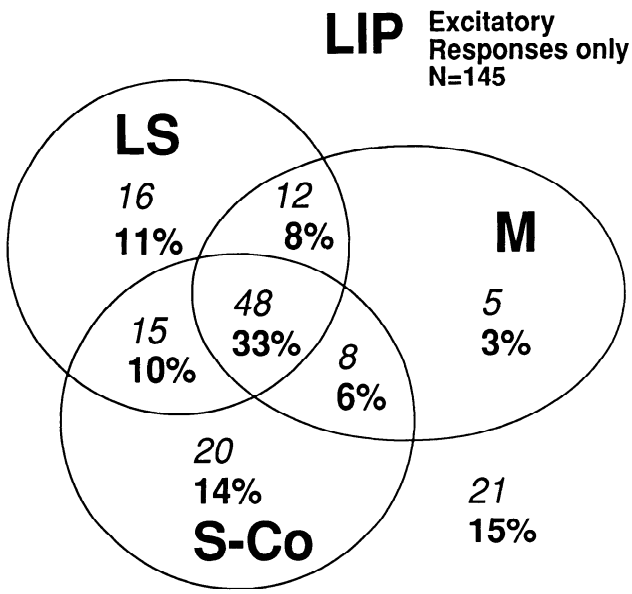

FIG. 7. Numbers and percentages of LIP neurons with excitatory responses in each phase LS, M, and S-Co and combinations thereof. Of the 145 LIP neurons, 63% show excitatory LS responses, 50% excitatory M responses, and 63% excitatory S-Co responses.

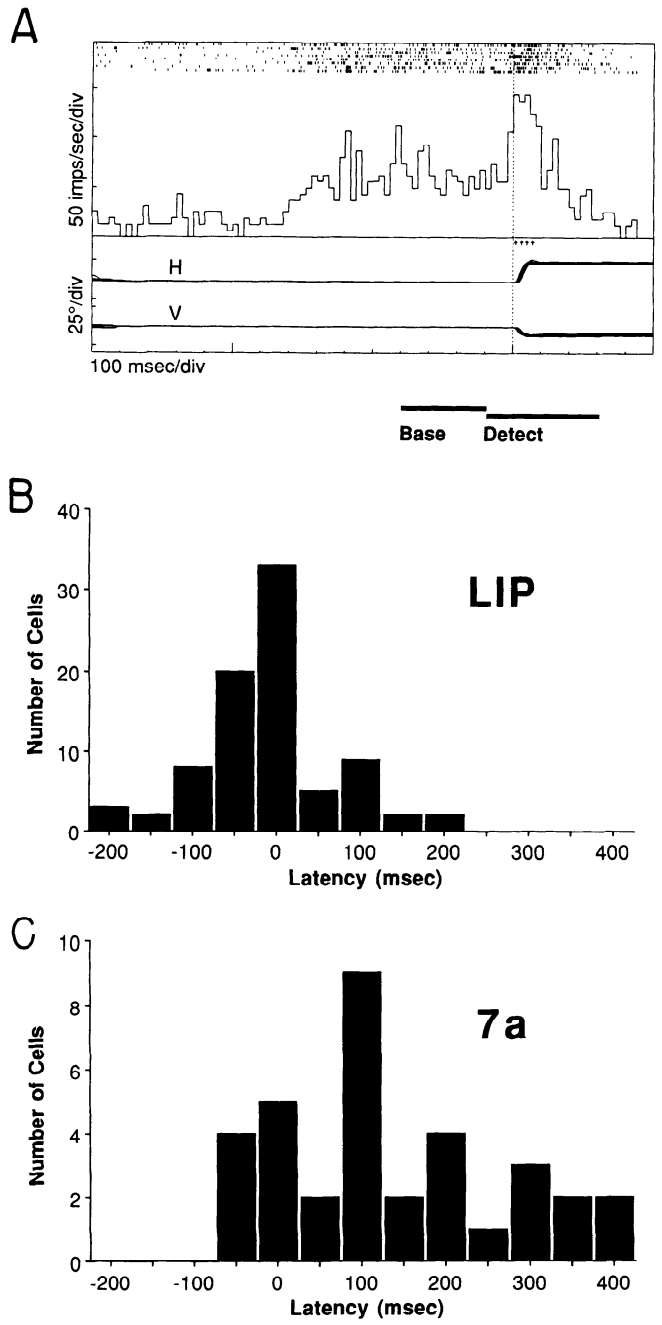

FIG. 8. Latency of the saccade-related activity. *A*: determination of the latency for an individual LIP neuron. Delayed-saccade trials, in the neuron's preferred direction, were aligned on the beginning of the saccade. Those histogram bins within the detect interval that showed significantly higher activity than the "base" period were marked (by ↑ beneath histogram bins). The latency of the S activity was defined as the time from the beginning of the saccades to the midpoint of the 1st marked bin. (Hence negative latency implies presaccadic start of the S activity.) Shown, from the *top*, are the spike rasters and the histograms; and the horizontal and vertical eye-position traces of the various trials, superimposed. Vertical dotted line denotes beginning of the saccades. *B*: distribution of latencies of the S activity for neurons in area LIP. *C*: distribution of latencies of the S activity for neurons in area 7a.

saccade and continues close to the end of the trials. The baseline interval in this case is taken from the fixation interval in the beginning of the trial, before the presentation of the target stimulus; the reason is that we test when the activity level returns to background. Note that at the end of the trial the monkey had to keep his eye for 500 ms where the stimulus had been, i.e., to "fixate" a remembered location, although without a visual stimulus. The time of offset of the S activity is defined as the midpoint of the last 20-ms bin marked by the *t* test, relative to the end of the saccade.

Figure 9*B* illustrates the results for area LIP. The S activity clearly outlasts the saccade, often by >100 ms (the median offset is 120 ms).

The results for area 7a (not shown) are similar, although the graph spreads out to higher values (the median is 300 ms) because many area 7a neurons are postsaccadic.

Now that we have the latency and the offset times of the S activity for each saccade, we can add them to the time of the saccade itself to obtain the duration of the S activity. The distribution of these durations, for area LIP and for area 7a, are shown in Fig. 10, *A* and *B*, respectively.

For both areas LIP and 7a, the distributions obtained for the S activity durations were approximately Gaussian. The parameters of these distributions were also similar: the median duration was 210 ms in both LIP and 7a, and the mean ±SD was 211 ± 104 ms for area LIP and 235 ± 113 ms in area 7a.

The similarity of the distribution of the durations in the two areas indicates that the main difference between the S

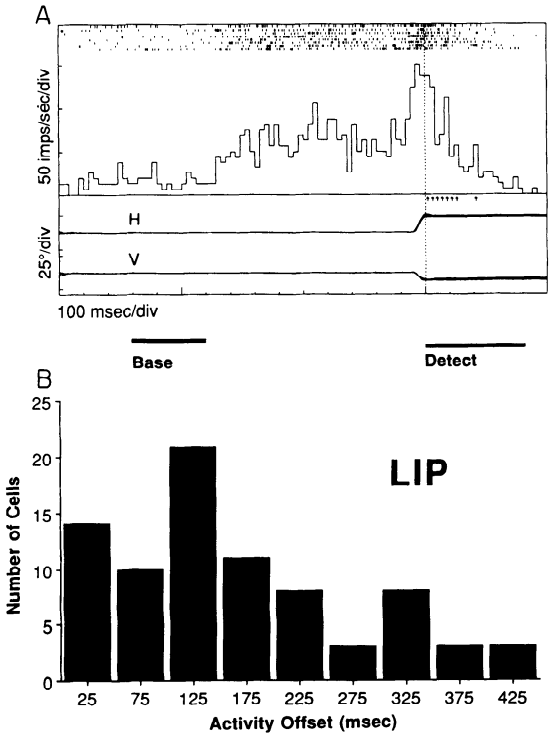

FIG. 9. Activity offset of saccade-related activity in area LIP neurons. *A*: determination of the S activity offset for an individual LIP neuron. Delayed-saccade trials, in the neuron's preferred direction, were aligned on the end of the saccade. Those histogram bins within the detect interval that showed significantly higher activity than the base period were marked (by ↑ beneath histogram bins). The offset of the S activity was defined as the time from the end of the saccades to the midpoint of the 1st marked bin. Shown, from the *top*, are the spike rasters and the histograms; and the horizontal and vertical eye-position traces of the various trials, superimposed. Vertical dotted line denotes the end of the saccades. *B*: distribution of the S activity offsets for neurons in area LIP.

activity in the two areas is in the time they start, that is, their latency with respect to saccade onset. Note that the duration of the burst is usually considerably longer than the duration of the movement that is normally ~60 ms for the 15–20° saccades used in the present study. Because, for more than one-half the LIP units, the S activity begins before the saccade, we conclude that typically in area LIP the S activity is perisaccadic. In area 7a, however, it is typically post-saccadic.

Latency of the LS activity

The latency of the LS activity was defined as the time, relative to the onset of the target stimulus, when the activity became significantly higher than that of the previous fixation interval (background). This procedure is illustrated in Fig. 11*A* for an area LIP neuron. The final part of the initial fixation (at least 300 ms), immediately preceding the presentation of the target, was used as the baseline interval. The detect interval started when the target light was turned on, and continued at least for the duration of the target stimulus (in some neurons, with late LS activity, it was continued even further). The onset latency for this cell was 90 ms.

Sixty-three neurons from area LIP that had clear LS responses were used for this analysis. Figure 11*B* shows the distribution of LS latencies obtained from these neurons. The shortest LS latency found is 50 ms; the sample median is 110 ms. Fifty-one LIP neurons (81%) have LS latencies less than 140 ms; the rest of the units had their LS latencies spread in a range of much higher values (max 270 msec), giving the distribution of the LS latencies a skewed shape.

The LS activity recorded from area 7a is, at least in the present behavioral task, considerably weaker than that of area LIP, as was illustrated earlier. Only 13 of the neurons studied in area 7a had a response robust enough for their LS latencies to be reliably evaluated. The distribution of the LS latencies of these neurons is presented in Fig. 11*C*. Comparison of Fig. 11, *B* and *C*, shows clearly that the typical LS

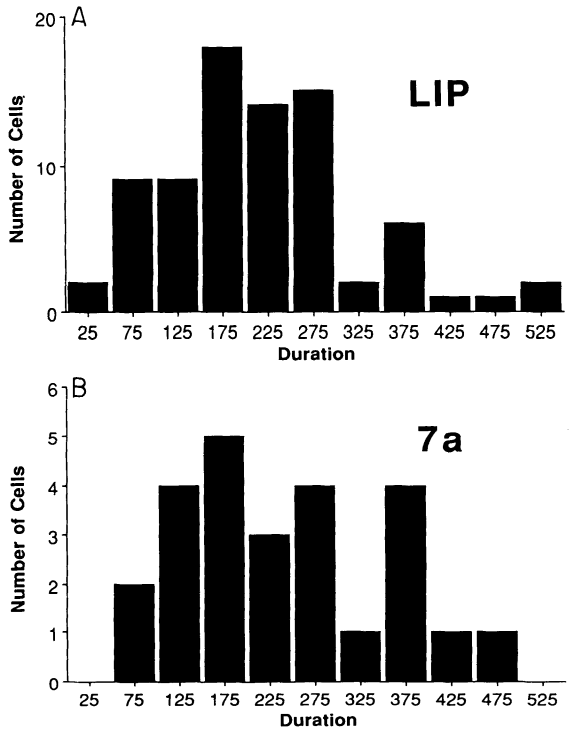

FIG. 10. Duration of the saccade-related activity. *A*: distribution of durations in area LIP. *B*: distribution of durations in area 7a.

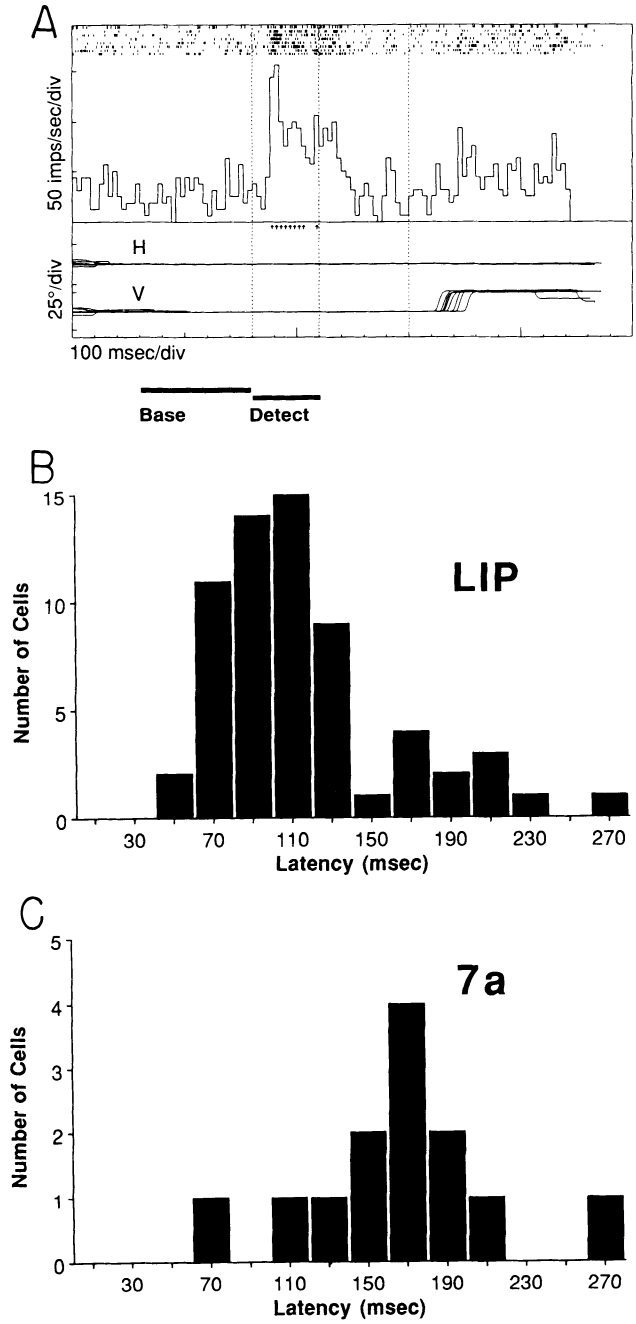

FIG. 11. Latency of the LS activity. *A*: determination of the LS latency for an individual LIP neuron. Delayed-saccade trials, in the neuron's preferred direction, were aligned on the time of presentation of the target stimulus. Those histogram bins within the detect interval that showed significantly higher activity than the base period were marked (by ↑ beneath histogram bins). The latency of the LS activity was defined as the time from the target presentation to the midpoint of the 1st marked bin. Shown, from the *top*, are the spike rasters and the histograms; and the horizontal and vertical eye-position traces of the various trials, superimposed. Vertical dotted lines denote, from the *left*, the onset and offset of the target, and the offset of fixation spot. *B*: distribution of latencies of the LS activity for neurons in area LIP. *C*: distribution of latencies of the LS activity for neurons in area 7a.

latency values of area 7a are higher than those of area LIP; the median LS latency of area 7a is 170 ms. The difference in means between the distributions of these two areas is statistically significant (2-sample t test with separate variance estimates, $t = 3.405$, df = 16, $P < 0.01$; mean ± SD for LIP, 116 ± 46 ms; for 7a, 165 ± 48 ms).

The functional significance of this difference in LS latencies is not clear. It may reflect a longer anatomic pathway, because LIP occupies a lower position than 7a in the hierarchy of visual cortical areas. [Area LIP can be reached from striate cortex with only 1 stop in extrastriate cortex, e.g., in area MT; whereas reaching area 7a requires at least 2 stops (see Andersen et al. 1990a).] It could be argued that the difference in latencies may also be due to the difference in strength of the response between areas LIP and 7a, because weak responses typically have longer latencies. However, the area 7a neurons included in this analysis all had an activity index over two, the same requirement as for area LIP neurons, and thus the overall difference in the level of activity does not directly explain the different LS latencies.

DISCUSSION

Sensorimotor integration in area LIP

In the present study we have shown that in area LIP there are both visual and saccade activities. Moreover, the integration of these types of activity occurs on the level of single neurons. The sensorimotor nature of the activity in area LIP is exemplified by the existence of memory responses within it. Because these responses are initiated by a visual stimulus and terminated by a motor act, they are sensorimotor in nature.

Area 7a, in contrast, shows weaker activity in the periods up to and including the saccade. The most prominent activity in the population of area 7a units studied was postsaccadic. This suggests that, unlike area LIP, area 7a does not play a major role in the preparation of saccades.

In area LIP 63% (91/145) of units have clear, excitatory LS responses. The same percentage of neurons have clear, excitatory S-Co responses. If a neuron has excitatory LS activity, there is a 69% (63/91) probability that it will also have S-Co activity (and vice versa). These percentages are even higher if one counts units with either excitatory or inhibitory responses. Clearly, then, some form of visuomotor integration is occurring at the level of individual LIP neurons.

In the present study we have confirmed and extended the results of Gnadt and Andersen (1988) on memory responses in IPL neurons. Many (57%, 83/145) area LIP neurons show a maintained activity during the delay period of a delayed saccade trial, that is, after offset of the target until the saccade is made. Furthermore, 76% (48/63) of the neurons having both LS and S-Co responses also have M activity. It is important to recall that there is no visual stimulation nor movement during this delay period. Typically the M activity is excitatory, but in some units the firing is suppressed to below background (see RESULTS; Fig. 3). The M activity is spatially tuned, typically closely matching the LS and presaccadic tuning of the unit. We suggest in the companion paper (Barash et al. 1991) that this activity reflects the planning of saccades.

Functional differences between areas LIP and 7a

Until recently, area 7 has been treated as a single area in physiological investigations (e.g., Hyvarinen and Poranen 1974; Mountcastle et al. 1975; Robinson et al. 1978). However, in the last few years several anatomic studies have suggested that area 7 may be partitioned into subdivisions, including areas LIP and 7a. (e.g., Andersen et al. 1985, 1990a; Asanuma et al. 1985; Barbas and Mesulam 1981; Blatt et al. 1990; Lynch et al. 1985; Pandya and Seltzer 1982; Seltzer and Pandya 1980). However, the functional significance of these subdivisions had not been carefully investigated physiologically.

In the present study we have found clear differences in the patterns of activity between areas LIP and 7a, when tested in the same animals, and in the same paradigm (the delayed saccade). First, in terms of the average activity of the population, in area LIP there is a strong response to the visual stimulus, and the activity remains high until the completion of the saccade itself. In contrast, in area 7a the most prominent activity in the population of units studied was postsaccadic (see *Population profile of the activity*). Second, clear presaccadic activity was found in 72% (61/85) of the neurons tested in area LIP, but only in 18% (6/33) of the units from area 7a. The mean latency of the S activity was −10.5 ms in area LIP, but 140 ms in area 7a (see *Latency of the S activity*). The duration of the S activity was nevertheless similar (median, 210 ms in both areas). Third, the visual response was also quicker in area LIP than in 7a; the median latency for the LS response was 110 ms in LIP and 170 ms in 7a.

These differences between the activity in areas LIP and 7a are consistent with the different connections of the two areas. Area LIP was originally defined on the basis of its much stronger connections than area 7a (or, indeed, other IPL subdivisions) with known saccade centers such as the superior colliculus and the frontal eye fields (Andersen et al. 1985, 1990a; Lynch et al. 1985). This pattern of connections supports the hypothesis that area LIP, specifically, plays a role in the preparation of saccades. Also, area LIP occupies a lower position than area 7a in the hierarchy of visual cortical areas; area LIP can be reached from striate cortex with only one stop in extrastriate cortex [e.g., in the middle temporal area (MT)], whereas reaching area 7a requires at least two stops (Andersen et al. 1990a). This is consistent with the shorter LS latencies in LIP, as compared with those of area 7a.

The apparently weak LS responses in area 7a reported in this paper are probably due to the specific paradigm that we have used. We did not make any attempt to find the "optimal" visual stimuli for the neurons we recorded. We do not wish to suggest that area 7a is not a visual area.

In summary, we have shown that there are clear differences between areas LIP and 7a in the timing and strength of the responses of neurons during delayed saccades. Although typically the ranges of these parameters (e.g., S activity latencies) in LIP and 7a overlap to some degree, the differences in their distributions are significant. We believe that these differences establish the existence of a difference in function between these two areas. More specifically, these findings suggest that a major function of area LIP, but

not of area 7a, is in the planning of visually guided saccadic eye movements.

Planning of saccades versus dynamic control

Evidence presented in this and the companion paper suggests that the large proportion of presaccadic LIP neurons that have tonic M activity may be involved in the planning of saccades. The S activity of many LIP neurons starts before and lasts at least for the duration of the saccade. Do LIP neurons with such S activity have a role in the "real-time" dynamic control of saccades [in the sense of Robinson (1975) or related models of the execution of saccades]? We did not set out to investigate this issue in the present experiment, and we cannot rule out the possibility that a minority of LIP neurons do participate in dynamic control of saccades. Nevertheless, we believe that this is not the case for most LIP neurons for the following reasons. First, the S activity of most LIP neurons is much longer (mean, 211 ms) than the duration of the saccadic movement itself (~60 ms). Second, the firing rate usually does not vary much during the saccade. We therefore conclude that LIP is likely to play a role in higher level processes related to the planning of saccades, rather than in the control of the execution of such movements.

Relation to previous delayed-response studies

Various delayed-response tasks have been used to investigate neuronal mechanisms of short-term memory in several other areas of the brain, e.g., the prefrontal cortex (Fuster 1973; Joseph and Barone 1987; Watanabe 1986), the nucleus dorsalis (Fuster and Alexander 1973), the inferotemporal cortex (Fuster and Jervey 1982), and the hippocampus (Watanabe and Niki 1985). Neurons in the premotor cortex (e.g., Wise and Mauritz 1985), area 5 (Crammond and Kalaska 1989), and the putamen (Alexander 1987) have been shown to demonstrate directionally specific delay period activity in tasks requiring the monkey to make arm movements to the remembered locations of visual targets. Sustained activity during the delay period of the delayed-saccade task has been recorded in the substantia nigra pars reticularis (Hikosaka and Wurtz 1983), the caudate nucleus (Hikosaka et al. 1989), and in the prefrontal cortex and the frontal eye fields (Funahashi et al. 1989, 1990). These high-level motor areas, like area LIP, may participate in the planning of movements, and the delay period activity may be a common manifestation of this role.

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