Activity Linked to Externally Cued Saccades in Single Units Recorded From Hippocampal, Parahippocampal, and Inferotemporal Areas of Macaques

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Previous work from this laboratory (Ringo et al. 1994) found that approximately one-quarter of the units recorded in the inferotemporal cortex, the parahippocampal gyrus, and the hippocampal formation showed significantly altered activity with spontaneous saccadic eye movements. This was termed saccadic modulation. The activity occurred in the dark as well as in the light. The activity in the light could be measured in a time period before the slip of an image on the retina due to the eye movement could have affected unit activity. Thus its origin is extraretinal.

The role of saccadic modulation is currently unknown. Saccades have been found, however, to approximately double the amplitude of a widespread memory effect found in this region (Nowicka et al. 1995). This effect is a reduction in response to the representation of specific images, without any general fatigue in the neuron’s responsiveness, which we have called a stimulus-specific adaptation (for review, see Brown 1996; Ringo 1996).

The saccadic eye movements (saccades) in the previous study were all spontaneous. This situation limits the control over variables internal to the brain that might correlate with saccades. As an example, we found many units that showed a spatial selectivity in the magnitude of their saccadic modulation. It is conceivable, however, that a buildup of attention or interest in one target position changes both the activity in a unit and causes a saccade, i.e., creates a situation where the unit activity and the saccade are not causally linked but are both simply influenced by a shared underlying variable, the attentional increase.

Some of this concern may be eliminated by examining units for saccadic modulation using saccades that are triggered by an external cue. This was done in the present study by training the monkeys to make saccades to targets presented briefly on a display. The use of externally cued saccades also allowed us to examine saccadic modulation systematically for its directional selectivity as well as its sensitivity to the target position.

We found abundant saccadic modulation in single units recorded from the hippocampal formation, parahippocampal gyrus, and inferotemporal cortex with these externally driven saccades. Many of these units also showed directional selectivity and sensitivity to target position.

METHODS

Subjects

Two adult female macaque monkeys (Macaca nemestrina) were used in the experiments. They had limited access to water but received the necessary fluids as rewards in the behavioral task. Hydration levels were carefully monitored, and free water access was allowed at least once each week.

Surgery

Each monkey was prepared for single-unit recording. Nine stainless steel guide tubes (1.5 mm OD) were implanted stereotactically and directed to inferotemporal cortex, hippocampal formation, and posterior parahippocampal gyrus of one hemisphere. These guide
it was necessary to separate responses to the cued saccade from spontaneous and cued saccades. The correct response was rewarded with a squirt of fruit juice. Trials were repeated correctly by 1,000 ms after the onset of the cross, the trial was ended. A dot was presented, for 200 ms, 7.5° to the left of center. The monkey then was required to fixate gaze inside a 3° window around the cross. This window is shown in the figure as a dotted circle. That dotted circle and the dotted line showing the axis did not appear on the display. After a 600- to 1,000-ms delay, a dot was presented, for 200 ms, 7.5° to the left, right, above, or below the position of the cross. The monkey then was required to make a saccade to the dot and keep a steady gaze within a 3° window for ≥500 ms. Sequence of events in the control trial was similar to that in the experimental trial. Difference was that peripheral presentation of 1 dot was replaced with 4 dots in the periphery, for monkey M1 (as shown) or 1 central dot, for monkey M2, (not shown). To get its reward, the monkey had to withhold a saccade and keep a steady gaze (during ≥500 ms within a 3° window) on the position where the cross was presented previously. During recording sessions, 300 experimental and 150 control trials were run. For spontaneous saccades and that occurred during a 500-ms period starting 100 ms after the target dot was presented on the screen were treated as externally cued saccades.

Response to the dot in the control condition (without eye movements)

Because the cued saccades always followed a dot presentation, it was necessary to separate responses to the cued saccade from responses to the dot per se (e.g., a visual response). This was done by use of control trials (33% of all trials) in which the monkey was presented with dot patterns designed to be as good or better at eliciting visual responses as the saccade-cuing dots, while at the same time, the monkey was required to refrain from making a saccade. In these control trials, the monkey was required to maintain gaze on the position defined by the placement of the initial

Unit recording

Single-unit recordings were made with parylene coated electrodes (Microprobe), etched from 75 μm tungsten wire with an impedance of ~1 MΩ (measured at 1 kHz). During insertion, the electrodes were protected by a 0.3-mm stainless steel guard tube and lowered to within 10 mm of the recording site. A unit then was sought while advancing the electrode with a hydraulic microdrive. In each session, one, or sometimes two simultaneous, units were recorded using commercial recording and spike separation software (Datawave, Longmont, CO). Unit activity was separated from noise and the activity of other cells, off-line, on the basis of latency and amplitude of different spike components, fit to template, and first and second principal components were used.

Experimental procedure

The monkey was seated in a primate chair. Its head was held by a padded face mask mounted over the snout and by a plate behind the head, preventing withdrawal. Recordings were done in darkness. The display screen, a computer monitor, was positioned in front of the monkey’s eyes. Each session consisted of 450 trials. At the beginning of each trial, a cross (1° diam) was presented for 750 ms, 7.5° to the left or right of the center of the screen (see Fig. 1). For the trial to proceed, the monkey had to fixate its gaze on the cross and hold this eye position (within a circular window of 3° radius) after the cross disappeared. If the monkey had not fixated correctly by 1,000 ms after the onset of the cross, the trial was repeated. After a randomly determined interval (between 600 and 1,000 ms), a dot (0.5° diam) was presented for 200 ms. The center of the dot was 7.5° to the left of, right of, above, or below the point where the center of the cross was previously presented. The monkey was required to make a saccade to the dot and hold this eye position for ≥500 ms (within a circular window, 3° radius). A correct response was rewarded with a squirt of fruit juice.

Spontaneous and cued saccades

Before each trial, spontaneous saccadic eye movements were recorded. There was no illumination, from the display or otherwise, in that period. Changes in gaze position ≥4° within a 20-ms period (200°/s) were treated as saccades. In all cases, the beginning of a saccade was defined by the first time point in which the speed of the eye movement was >15°/s. Changes in gaze position that met the criteria described above for spontaneous saccades and that occurred during a 500-ms period starting 100 ms after the target dot was presented on the screen were treated as externally cued saccades.
fixation cross. Only trials in which the monkey’s gaze remained within the 3° window were rewarded.

For one monkey, control trials used the simultaneous presenta- tion of four dots (7.5° to the left, right, above, and below the center of the fixation cross). This configuration instructed the monkey to withhold any eye movement (Fig. 1). The second monkey had difficulty learning this paradigm, so for its control trials, we presented a dot that occupied the same place as the cross. All dots were the same color, intensity, and size.

Localization of recorded areas

At the end of the experiment, one monkey had electrolytic lesions made through each of the guide tubes and was killed with an overdose of barbiturate, then perfused transcardially with saline followed by 10% formalin. The brain was blocked in situ, extracted, embedded in wax, sectioned, and stained. The regions from which the recorded units were isolated were derived from histological data.

The second monkey continues to participate in other experiments. The guide tubes were implanted stereotactically. Assistance in estimating the regions from which units were recorded in this monkey was provided by profiles of cell distribution. Before the first cell recording from each well, the distance from the top of the guide tube to the bottom of the skull was measured with a fine tungsten probe (125 μm). Then the cell density distribution as a function of recording depth was established during the first several electrode penetrations. This method gave us a measure of the depth of layers of gray matter (with many firing units) separated by layers of white matter. Figure 2 presents the regions from which the units were recorded in both monkeys. The regions are shown on standard brain drawings from the atlas of Winters et al. (1969).

Statistical analysis

The existence of saccadic modulation in each unit was tested statistically. However, a simple spike count was not used because that method is poor at detecting biphasic activity, (i.e., discharge patterns after a trigger event that show an increased rate following or followed by a decreased one) and because such biphasic activity patterns are common in ventral temporal lobe units following saccades (Ringo et al. 1994). Instead, we used an analysis that examined how repeatable the shape of the unit response was after the cued saccade in different trials. We measured whether the spike distribution in a single trial could be predicted from the averaged spike distribution from all other trials (excluding the trial in question). The ability of this template to predict the spike pattern at a level greater than chance was used as the indication of a significant response. This method had the additional advantage, compared with a spike count in a fixed time bin, that it adjusts to the time course of the response of each unit.

Visual inspection of the averaged spike density histograms around the cued saccade suggested that a 300-ms period after the saccade includes the strongest saccadic modulation. Therefore, this period was used, and spike counts were broken into 15 bins each of 20 ms. Baseline activity, determined as the average spike rate during the 300 ms before the saccade, was subtracted from each bin. This 15-element vector was calculated for each goal-directed saccade. To find whether there was any consistent response related to these saccades, the vector for a particular saccade was multiplied (inner or dot product) by the 15-element vector created by averaging all other response vectors (i.e., excluding the particular saccade under consideration). Multiplications from all bins were added together (Eq. 1). This process was repeated for each saccade.

\[
M_i = \sum_{j=1}^{15} (y_{ij} \times \sum_{k=1}^{n} y_{k})
\]

where

- \( n \) is the number of saccades in the data set for the unit
- \( M_i \) is the inner product measure for a particular, \( i \), saccade,
- \( y_{ij} \) is the amplitude of the response for a particular saccade, \( i \), and an individual 20 ms bin, \( j \).
- The amplitude is measured with respect to a presaccade baseline (defined from the 300-ms period just before saccade onset).
- \( y_{kj} \) same as \( y_{ij} \), index \( k \) substituting for index \( i \).

We determined, with a test of proportions (Sokal and Rohlf 1969), if the percentage of saccades for which the inner product measure, \( M_i \), had positive values was significantly \( >50\% \). Fifty percent is the value expected by chance if there is no consistent response, and hence no predictive power from calculating the average response to all other saccades. A value significantly \( >50\% \) indicates predictive power, hence a consistent response.

Response to the dot in the control condition was examined using the same method that was used for testing saccadic modulation. The only difference was that the 300-ms period of analysis started 100 ms after the dot was presented on the screen (and not when the saccade occurred since there were no saccades in these control trials). This time period was chosen to maximize the measured response. Only trials in which the monkey did not make any eye movement \( >1° \) after the onset of visual image were used in this analysis.

RESULTS

The monkeys performed the task well, making crisp saccades to the location of the dot on experimental trials while gaze remained fixed on control trials. Figure 3 shows a typical set of eye position records. The unit was selected for its good unit spike activity but was not selected for good saccades. All the eye position records for movements in the
different directions and alternate records for control trials (i.e., a comparable number) are shown.

We recorded 290 units in the present experiment. Of these, 209 units (149 cells from monkey M1 and 60 from M2) had an average spike frequency of \( \approx 2 \) Hz. Only these more active units were analyzed to limit the number of trials with zero spikes in the period of interest. The numbers of units recorded from different regions are presented in Table 1. The anterior/posterior distinction used here was with reference to a coronal level at the posterior end of the anterior medial temporal sulcus and the anterior end of the occipital temporal sulcus. This divides area TE (Seltzer and Pandya 1978; Von Bonin and Bailey 1947) into approximately anterior and posterior halves, labeled aIT and pIT in the Table. The dorsal/ventral distinction (dorsal including TE1, TE2 and TE3) follows Seltzer and Pandya (1978).

About 20% of the units (39/209) showed significant \( (P < 0.01) \) responsiveness to the image used in control trials (4-dot pattern in 1 monkey or 1 dot in the other monkey). The most responsiveness was observed in inferotemporal cortex, less in posterior parahippocampal gyrus, and the least in the hippocampal formation (Table 1).

For further analysis, we took the 134 cells (from the 209 units with firing rates \( > 2 \) Hz) that did not show even a trend of significant response to the dot in control trials (i.e., for which \( P > 0.1 \)). The intent of this selection was to eliminate units with visual response to the dot so that any response with the saccade triggered by the dot presentation would be unlikely to be caused by a visual response. From this group of 134 visually nonresponsive units, 71 (53%) showed significant \( (P < 0.01) \) activity linked to externally cued saccades, and 39 (29%) units showed significant \( (P < 0.01) \) activity linked to spontaneous saccades. The criterion level of \( P < 0.01 \) was used here for categorizing units as showing saccadic modulation to limit the effects of multiple testing (i.e., in testing 134 units with a \( P < 0.05 \) criterion, one expects about 7 false inclusions or type I errors, whereas for a \( P < 0.01 \) criterion, one expects only \( \sim 1 \) such erroneous classifications). Overall, the group of units showing saccadic modulation for externally cued saccades and the group showing modulation for spontaneous saccades showed no statistically significant correlation (Fisher exact test, \( P = 0.57 \)). Correlation is evident among the strongest responding units from each group (Fisher exact test, \( P = 0.0012 \), when the groups are defined by including only those with a significance test level of \( P < 0.0001 \)).

Interestingly, there was no correlation (Fisher exact test, \( P = 0.28 \)) between groups of units responsive to the dot presentation in control trials and those that showed significant modulation related to target directed saccades. This would suggest that the saccadic modulation effect is not a result of the visual response to the dot. Several examples of units that showed particularly clear saccadic modulation on trials with goal directed saccades and no response to the control dot(s) are presented in Fig. 4.

For comparison, a simple spike count method was used to examine the responses to the cued saccades in the group of 134 units that showed no visual responses. This analysis used a \( t \)-test \( (P < 0.01 \) criterion) to look for a significant difference between the spike count in the same 300-ms time periods (1 before and 1 after the cued saccade) as used in the template matching procedure. In this case, 42 units (42/134, 31%) showed significant saccadic modulation. Thus as one might expect, the spike count method was less sensitive in detecting responses than the template method.

Statistical significance of the modulation related to spontaneous saccades also was calculated (using the same template matching method as for the cued saccades). The number of such cells found in different areas is listed in Table 1. The number of cells that showed significant modulation related to spontaneous saccades was greater in posterior \( (38.0\% ; 35/92) \) than in anterior \( (9.5\%; 4/42) \) inferotemporal cortex. The difference was statistically significant (test of proportion, \( t = 3.7, P < 0.001 \)). This agrees with the relative paucity of such units in the more anterior regions in the
previous study (Ringo et al. 1994). However, there was no such difference in units with saccadic modulation linked to externally cued saccades (54 and 50%, respectively).

We next analyzed whether unit activity occurring just after the goal-directed saccade showed gaze-position and saccade-direction sensitivity. The 71 units that showed significant saccadic modulation were tested for sensitivity to the direction of saccade to the dot (left, right, up, or down) after collapsing the data across the two starting positions (left and right). For each of these four groups of data, the template matching method was used to test for responses that were significantly different between directions. Thus the proportion of trials with (for example) upward saccades, that produced a positive result when multiplied by the template resulting from all other upward trials, was compared (via a test of proportions) with the corresponding proportions derived from trials with the other directions. If the responses for saccades of different directions did not differ, no difference in these proportions would be expected. The problem of underestimating the type I error rate due to use of more trials is avoided by use of Bonferroni adjustment (Harris 1985). Results are presented in Table 2. There were 12 units (17%) that showed direction sensitivity at a $P < 0.01$ level (probability after Bonferroni adjustment). Using a lower criterion ($P < 0.05$, Bonferroni corrected), 22 units (31%) showed directional sensitivity. Two clear examples of cells showing different target directed saccadic modulation for different directions are presented in Fig. 5.

Similarly, the 71 units that showed significant saccadic modulation were tested for sensitivity of that saccadic modulation to the (left or right) starting gaze position for the saccade to the dot. For this analysis, data were collapsed across the four directions. For each of these two groups of data (left and right starting gaze-position), the template matching method was used again to test for responses that were significantly different between the two starting positions. Thus the proportion of trials starting from a left fixation point, which produced a positive result when multiplied by the template resulting from all other left fixation trials, was compared (via a test of proportions) with the corresponding proportions derived from trials with the right starting point. If the responses for saccades from different starting positions did not differ, no difference in these proportions would be expected. Results are presented in Table 2. There were nine units (9/71, 13%) that showed position sensitivity at a $P < 0.01$ level. With a lower criterion ($P < 0.05$), 20 units

### Table 1. Results of statistical testing of saccadic modulation in units recorded from different brain regions

<table>
<thead>
<tr>
<th>Structure</th>
<th>$n^{*}$</th>
<th>$P &lt; 0.01$</th>
<th>$P &lt; 0.1$</th>
<th>$n^{†}$</th>
<th>$P &gt; 0.1$</th>
<th>Cued</th>
<th>Spontaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>aHF</td>
<td>11</td>
<td>1 (9.1)</td>
<td>3</td>
<td>8</td>
<td>1 (12.5)</td>
<td>1 (12.5)</td>
<td></td>
</tr>
<tr>
<td>pHF</td>
<td>67</td>
<td>7 (10.4)</td>
<td>20</td>
<td>47</td>
<td>27 (57.4)</td>
<td>21 (44.7)</td>
<td></td>
</tr>
<tr>
<td>aIT ventral</td>
<td>22</td>
<td>5 (22.7)</td>
<td>11</td>
<td>11</td>
<td>5 (45.5)</td>
<td>1 (9.1)</td>
<td></td>
</tr>
<tr>
<td>aIT dorsal</td>
<td>37</td>
<td>10 (27.0)</td>
<td>14</td>
<td>23</td>
<td>15 (65.2)</td>
<td>2 (8.7)</td>
<td></td>
</tr>
<tr>
<td>pIT ventral</td>
<td>5</td>
<td>3 (60.0)</td>
<td>4</td>
<td>1</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>pIT dorsal</td>
<td>31</td>
<td>8 (25.8)</td>
<td>14</td>
<td>17</td>
<td>10 (58.8)</td>
<td>7 (41.2)</td>
<td></td>
</tr>
<tr>
<td>pPG</td>
<td>36</td>
<td>5 (13.9)</td>
<td>9</td>
<td>27</td>
<td>13 (48.1)</td>
<td>7 (25.9)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>209</td>
<td>39 (18.7)</td>
<td>75</td>
<td>134</td>
<td>71 (53.0)</td>
<td>39 (29.1)</td>
<td></td>
</tr>
</tbody>
</table>

* Dot response refers to the number of units that showed significant responses ($P < 0.01$ or $P < 0.1$) to the control dot(s). Only trials without saccades (no eye movement $>1^*$) were analyzed. Saccade modulation refers to the number of units showing significant ($P < 0.01$) response to cued or spontaneous saccades. Values in parentheses are percentages. aHF, anterior hippocampal formation; pHF, posterior hippocampal formation; aIT, anterior inferotemporal cortex; pIT, posterior inferotemporal cortex; and pPG, posterior parahippocampal gyrus. † Number of units that had an average frequency of firing $> 2$ spikes/s. Only these were analyzed.

**FIG. 4.** Four examples (A–D) of units showing activity linked to externally cued saccades. Responses of the units to saccades in experimental trials are presented (top). Vertical line denotes the beginning of a saccade (defined as the first time point in which the speed of the eye was $> 15^*$/s). Corresponding response of the units in control trials also are presented (bottom). Vertical line denotes the onset of the control dot(s), which instructed the monkey to withhold eye movements. Only trials without any eye movements $> 1^*$ for a 800-ms period beginning with the onset of the dot(s) were included in the analysis. A–C were recorded from monkey M1 (4-dot control). D was recorded from monkey M2 (1-dot control presented in the same position as the cross).
TABLE 2. Number of units with saccadic modulation that showed significant directional and positional specificity of postsaccadic activity

<table>
<thead>
<tr>
<th>Structure</th>
<th>Direction†</th>
<th>Position‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>aHF</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>pHF</td>
<td>27</td>
<td>3</td>
</tr>
<tr>
<td>aIT ventral</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>aIT dorsal</td>
<td>15</td>
<td>4</td>
</tr>
<tr>
<td>pIT ventral</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>pIT dorsal</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>pPG</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>71</td>
<td>12 (16.9)</td>
</tr>
</tbody>
</table>

Values in parentheses are percents. * Number of cells that showed significant (P < 0.01) saccadic modulation to the cued saccades. † The direction of saccade made to the dot (left, right, up, down). ‡ The position of the fixation cross on the screen (left, right).

(28%) showed position sensitivity. Almost half (33/71) of the units showed some spatial influence over the saccade modulation with the lower (P < 0.05) criterion.

The position sensitivity seen above is an influence of starting position on the modulation caused by the saccade. We also examined the spike trains to find whether the cells showed simple gaze-position sensitivity as such. That is, was there a difference in rate of discharge due simply to gaze position regardless of saccades. Such simple position sensitivity was examined by a t-test of the spike count (for left vs. right fixation trials) in the 300-ms bin immediately before the cue presentation. Seven units (7/71) were found to show significant simple position dependence at the P < 0.01 level.

The results of these analyses show that the position of initial fixation, i.e., the direction of gaze, can change the general level of some units’ activities and that saccadic modulation itself, in some units, shows direction and/or position specificity.

DISCUSSION

The experiment described in this paper has revealed a strong saccadic modulation widely spread in the ventral temporal lobe. Roughly half of the units recorded in the hippocampal region, the parahippocampal gyrus, and the inferotemporal cortex showed significant modulation of their activity linked to target-directed saccades.

This saccadic modulation often had a spatial component. Using the simple saccade task of this study, the direction of the saccade significantly affected about one-sixth of the cells that show saccadic modulation (one-third if a lower criterion of significance is used). In another spatial characteristic, unit activity in about one-seventh of the cells in hippocampal formation and parahippocampal gyrus depended significantly on the starting position of gaze. Only a single inferotemporal unit showed this effect. Various position effects have been seen in hippocampal and parahippocampal recordings previously (Feigenbaum and Rolls 1991; Nowicka et al. 1996). The current study has the advantage that it clearly could separate influences of gaze position from the saccades that preceded those fixation episodes. If one equates the place viewed by the monkey with the place occupied by the rat, these units may, in some ways, be reminiscent of head-direction cells recorded in the rat subiculum, and elsewhere (Taube et al. 1990), or hippocampal place cells (Muller and Kubie 1987; O’Keefe and Speakman 1987). However, our experiments do not provide conditions to separate the direction the eye is pointed with respect to the head and/or the rest of the body from the direction of the eye in spatial coordinates independent of the body.

In the current experiments, a dot, briefly flashed, cued the monkey to make the saccade. It is logically possible that the responses we analyzed were to that light flash rather than to the saccade. There are, however, two reasons we think this is unlikely. First, in our detailed analysis, we excluded units that showed even a trend (P < 0.1) toward a significant response to the control dot(s). Second, the groups of units significantly responding to trials with saccade and in trials without saccade were not correlated (indicating that the two groups were not responding to the same stimulus).

Because our analysis method was slightly unusual, the question may arise if our results were somehow spuriously due to this choice. Our analysis method was basically a template matching operation. This had the advantage of ac-

FIG. 5. Directional sensitivity. Two examples (A and B) of units that show differential saccadic modulation for different directions of eye movement. A: greatest response was to saccades directed downward. B: saccades to the left produced an inhibition of the unit activity, whereas saccades to the right produced an increase in the unit activity. In neither unit was there any statistically significant response to dots in control trials. Both units were recorded from monkey M1.
commodating differing temporal characteristics in the different unit responses. It is, however, not as manifest an operation as a simple spike count. It is therefore reassuring that when a traditional spike count analysis was performed it also found many units with significant saccadic modulation (RESULTS). The fact that the template matching method discovered more such units than did the spike count method is to be expected because template matching is sensitive to consistent differences in some temporal aspects of the response in addition to the sensitivity to simple spike count difference.

In our previous work (Ringo et al. 1994), we found robust saccadic modulation related to spontaneous eye movements. The current work shows that saccades directed by an external command also produce extensive saccadic modulation in the same areas. This makes it clear that the modulation was not peculiar to saccades in a monkey unoccupied by any task. The current work also suggests that the saccadic modulation is not due to some internal state change, which itself is not directly related to saccades but which simply correlates with saccades. From previous work, it was possible to suppose that both spike activity in the temporal lobe recordings and spontaneous saccades stemmed from a shared preceding event but were not themselves causally linked. For example, one might suppose that the neural circuitry had reached a “decision” or a break point in some internal state, perhaps related to attention, that would lead to both activity in units in the temporal lobe recording regions and to an increased tendency to make a saccade but that the temporal lobe unit activity and the saccade were not directly related. The current work, showing robust and short-latency activity with externally commanded saccades, indicates that these are liable to be related more directly.

One simple implication of this work is a methodologic one. In any work with units from the areas in which we recorded, care must be taken to ensure that results are not confounded with eye movements.

The functional significance of saccadic modulation is currently unknown. There has been no work that we are aware of suggesting it plays a substantial role in oculomotor control nor is it tightly coupled to known oculomotor circuitry (Baizer et al. 1993; Büttner and Büttner-Ennever 1988).

Our recordings were made in inferior temporal cortex, hippocampal formation, and the parahippocampal gyrus, areas that usually are associated with high level memory and visual perception. It seems plausible that saccadic modulation is part of the mnemonic and attentional processing usually associated with the ventral temporal lobe (Mishkin 1982; Riches et al. 1991; Richmond et al. 1983; Rolls 1987; Sobotka and Ringo 1993). In agreement with such a possibility, we have found recently that saccades approximately doubled the stimulus specific adaptation seen widely in inferior temporal cortex (Nowicka et al. 1995). Indeed, such effects may be part of the mechanism of the cognitive role saccades have long been thought to play (see, for example, the still cogent arguments for a role of eye movements in perceptual learning made in Chapter 2 of Hebb 1949).

The saccadic modulation recorded in ventral temporal areas partly changes character between spontaneous and cued saccades. Changes occur in the fraction of units showing effects in the regional distribution and in the particular units displaying effects. This suggests the modulation is malleable to the current brain state or task; this is certainly compatible with a role as a controlling signal, synchronizing the processing with the saccades. If so, then in future work, experimental designs that incorporate more complex images in more complex tasks may help discover the role played by saccadic modulation in the memory/attentional temporal stream system.

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The saccadic modulation recorded in ventral temporal areas partly changes character between spontaneous and cued saccades. Changes occur in the fraction of units showing effects in the regional distribution and in the particular units displaying effects. This suggests the modulation is malleable to the current brain state or task; this is certainly compatible with a role as a controlling signal, synchronizing the processing with the saccades. If so, then in future work, experimental designs that incorporate more complex images in more complex tasks may help discover the role played by saccadic modulation in the memory/attentional temporal stream system.


