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Behavioral Enhancement of Visual Responses in Monkey Cerebral Cortex. I. Modulation in Posterior Parietal Cortex Related to Selective Visual Attention

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SUMMARY AND CONCLUSIONS

1. Clinical and experimental data suggest that area 7 of posterior parietal cortex plays a role in visual attention and eye movements. We have operationally defined attention as a stimulus-selection process independent of the specific movement used to respond to the stimulus. We trained monkeys to make various hand or eye movements and recorded from single neurons in area 7 while the monkeys were performing these tasks.

2. As demonstrated previously (19, 20, 46, 47, 63), many cells in area 7 respond to visual stimuli independent of any behavior. The discharge to a stimulus may be enhanced when the animal makes an eye movement to the stimulus.

3. We used two paradigms to study the modulation of visual responses when the animal used a stimulus without making an eye movement. The first was a peripheral-attention task in which the animal had to signal the occurrence of a peripheral stimulus without making an eye movement to it. Half of the cells studied gave an enhanced response in this task. In the second task, the animal had to reach out and touch a stimulus without making an eye movement to it. Visually responsive parietal neurons also yielded enhanced responses in this task.

4. The enhancements demonstrable in the saccade, peripheral-attention, and hand-reach tasks probably represent the same underlying process because their frequency of occurrence in our sample is almost identical, the intensities are quite similar, and cells that give an enhanced response on one task give an enhanced response in the others.

5. These results show that in posterior parietal cortex, the behavioral enhancement of a visual response is independent of the specific movement used to respond to the stimulus. The physiological mechanism of enhancement, which is movement independent and spatially selective, resembles the psychological phenomenon of selective spatial attention. We suggest that the role of area 7 in visual attention may be mediated by the enhancement of visual responses to selected stimuli.

INTRODUCTION

Since the 19th century clinical and experimental studies have suggested that posterior parietal cortex plays a role in the neural processes underlying visual attention (9). Humans with lesions in posterior parietal cortex neglect their contralateral visual fields, and this had been considered to be an attentional deficit (24). When a patient with damage to the right parietal lobe is presented with a visual stimulus in each visual hemifield, he reports seeing only the one contralateral to the normal hemisphere. However, when he is presented only with a stimulus contralateral to the damaged hemisphere and is instructed to attend to that field, he can detect the stimulus. Thus the deficit is

not purely sensory, since under certain circumstances the patient can perceive the stimulus, nor is it motor, since the patient is capable of making all movements required for orientation and response. Instead, it is an inability to select the stimulus from a complex environment, and it is this deficit that we and others (9, 24, 59) consider to be an attentional one.

Monkeys with posterior parietal lesions also display deficits that appear to be related to visual attention. These include a failure to avoid noxious stimuli in the field contralateral to the lesion (10, 25), deficits in visually guided reaching (23, 34, 42, 52), and difficulties in performing discriminations that require rapid shifts in attention (37).

Other work has associated posterior parietal cortex with eye movements under several experimental and clinical situations. Thus humans with posterior parietal lesions have difficulty making eye movements to explore the visual environment (54). Monkeys with posterior parietal cortex lesions have difficulty beginning to track moving targets and have increased latencies for saccade initiation (35). Transcortical stimulation of this area results in eye movements (15, 55). Single neurons have been identified whose activity is associated with eye movements (26, 36, 39). Mountcastle and colleagues (36, 39) suggested the inferior parietal lobule mediates the process of visual attention by directing eye movements. Since the primate retina can best analyze visual stimuli in the fovea, the process of attention is intertwined with the process of gaze shifting (62), and the eye movement relationship for area 7 may be related to shifting attention rather than shifting the eyeball.

In a previous study (47), we found that in our sample every neuron associated with eye movements could be driven by some visual stimulus. The response of half these neurons was enhanced when the animal made the appropriate eye movement to respond to the stimulus. The neurons did not discharge when the animal made the same response in the absence of the stimulus. Because the activity of the neurons depended on the stimulus and did not depend on the movement, we proposed that their activity did not serve as a direct and exclusive precursor of the eye movement. Instead, we postulated that it indicated a response to an attended stimulus and, since the animal made eye movements to attended stimuli, the cells discharged in association with those eye movements.

This presaccadic enhancement of visual responsiveness was first described in the superior colliculus (21) and has been found in other areas of the monkey visual system, including striate cortex (60), prestriate cortex (45), frontal eye fields (17, 18, 60), posterior parietal cortex (47, 63), and pulvinar (28). It was first suggested that the enhancement process could be an excellent physiological mechanism for the psychological process of attention, since attention intensifies the effect of a stimulus on an animal's behavior (21). However, for each area it is important to determine whether enhancement is related to the eye movement or to the shift of attention that precedes and facilitates the eye movement. In the superior colliculus, frontal eye fields, and pulvinar, enhancement of visual responses occurs only before eye movements, not before other modes of attentive behavior (17, 18, 28, 61). Therefore, in these areas enhancement may be an excellent mechanism for facilitating a visually guided eye movement, but it cannot by itself be a mechanism underlying a more general process of visual attention.

In all previous studies of area 7, eye movements were an integral part of attentive behavior. It was impossible to determine whether the relationship of neuronal response to eye movements was specific to that movement or more related to the attentional mechanisms that are associated with the eye movement. We therefore used a series of tasks in which the animals made an eye movement to a stimulus or performed other behaviors in which they had to attend to the stimulus but not make an eye movement to it. We found that in area 7 response enhancement is easily dissociable from the oculomotor process and occurs in several conditions in which the stimulus is important to the animal but is not the target for an eye movement. These results imply that area 7 has a role in selective visual attention that is frequently intertwined with the oculomotor processes but dissociable from them. Brief reports of these results have been presented previously (6, 46, 59).

METHODS

Behavioral training

Four rhesus monkeys, weighing 3.5–5.0 kg, were trained on several visual tasks similar to those developed by Wurtz (58) and subsequently modified by others (21, 47). A PDP-11 computer was used for animal training and behavioral control.

A leather harness was placed around the chest of each monkey under ketamine anesthesia (47). The monkey was trained to jump from its home cage into a primate chair, restrained only by a cane attached to the harness. Monkeys remained in the chair only during actual training and recording periods, and rested unrestrained in the home cage each day. The animals were water deprived and their weight monitored daily to prevent excessive dehydration. Water was used as a reward, and the monkeys were allowed to work to satiation each day.

The monkeys were trained on four tasks: fixation, saccade, peripheral attention, and hand reach. They were first trained on the fixation task. In this task (shown in Fig. 1*A*) the monkey pressed a lever to bring a small spot of light (5′ of arc) onto a tangent screen 57 cm in front of him. After a variable and unpredictable interval (1–4 s), this fixation point dimmed and remained dim for 400–600 ms. If the monkey released the bar while the light was dim, he received a drop of water. If the monkey released the bar either before or after the dim period, he received neither reward nor punishment. At the end of a variable and unpredictable interval (usually 400–1,200 ms) after the fixation-point onset, a second visual stimulus came onto the screen. This second stimulus was behaviorally irrelevant to the monkey. If the animal broke fixation to look at the peripheral stimulus, the computer automatically aborted the trial and extinguished the fixation point and second stimulus, thus depriving the animal of a chance to earn a reward on that trial. The peripheral stimuli were produced by a Leitz projector with a quartz-iodide bulb and were adjusted to an intensity of 1.0–1.5 log units above a background illumination of 1 cd/m^2 using Kodak Wratten neutral-density filters. The size, shape, hue, and location of the stimuli were easily changed so that receptive fields and response characteristics of neurons could be determined. Trials were separated by an intertrial interval of 0.5 s, during which the monkey's lever presses had no effect.

All other tasks began like the fixation task, with the monkey pressing the lever and fixating a central point. In the saccade task (Fig. 1*B*), the central fixation point went out and a small peripheral stimulus came on simultaneously. The monkey then made a saccade to fixate the second stimulus

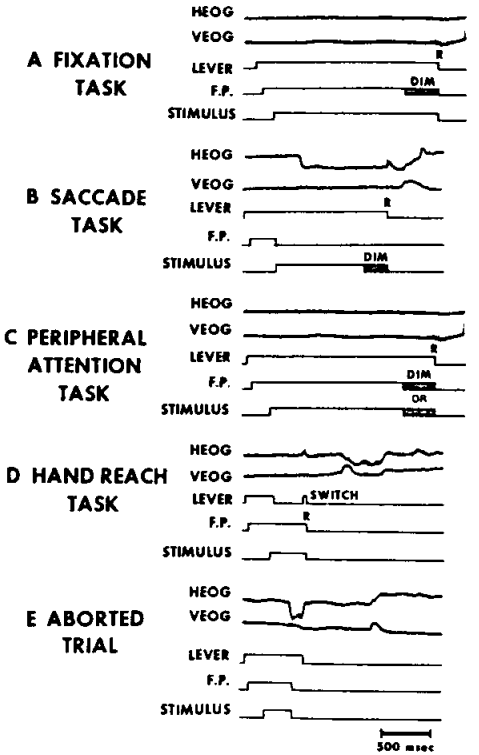

FIG. 1. Schematic illustration of the behavioral tasks used to study parietal neurons. In the fixation task (*A*), the monkey is required to fixate a spot of light (FP) turned on by pressing a lever. The animal learns not to respond to the subsequent onset of a stimulus light and releases the lever when the fixation point dims to obtain a reward (R). After the reward the monkey is free to make spontaneous eye movements. Horizontal and vertical electrooculogram traces (HEOG, VEOG) document that no eye movements were made to break fixation. For the saccade task (*B*), the trial begins similarly but the fixation point is turned off when the stimulus comes on, and the monkey makes a saccadic eye movement to fixate the stimulus. The deflection of HEOG depicts this eye movement; the monkey releases the bar when the stimulus dims. During the peripheral-attention task (*C*), the monkey initiates trials as before but the animal must release the lever when either the fixation point or the stimulus dims. Each dims 50% of the time in random distribution, and the monkey cannot make a saccadic eye movement to the stimulus. The lack of deflection on EOG traces indicates continued fixation. The hand-reach task (*D*) begins with a lever press, resulting in appearance of the fixation point. Next a panel switch is illuminated and the monkey must release the lever and contact the panel without making an eye movement. As a control against inappropriate eye movements, the computer terminates trials (extinguishes the fixation point and the stimulus) whenever a saccade greater than 2° is made during a fixation period (*E*).

in expectation that it would dim so that he could release the bar and get a reward. In order to prevent predictive saccades, we varied the time at which the fixation point changed and we maintained a 0.25 probability that no fixation-point change would occur.

The peripheral-attention task (Fig. 1*C*) resembled Wurtz and Mohler's (61) hand-response task. This paradigm required the monkey to detect a dimming of a peripheral stimulus while he continued to look at the central fixation point. The task was like the fixation task except that on 50% of trials the peripheral stimulus dimmed but not the fixation point. The monkey had to release the lever in response to dimming of either the fixation point or the peripheral stimulus. The monkeys learned the task without eye movement control, and all monkeys spontaneously adopted the strategy of looking at the fixation point and allowing the peripheral stimulus to fall on peripheral retina. During the actual experiments the computer monitored the monkey's eye movements and aborted the trial whenever an eye movement greater than 2° occurred (Fig. 1*E*). This ensured fixation on every trial.

In the hand-reach task (Fig. 1*D*), as in all tasks, the monkey pressed the lever to initiate the trial and illuminate the fixation point. On 25% of the trials, the central fixation point dimmed and a correct lever release was rewarded. On the remaining 75% of trials at a random interval after fixation, the Leitz projector illuminated a touch panel. In these cases the monkey had to reach up through the hand hole in the primate chair and touch the illuminated panel without moving his eyes from the fixation point. Monkeys would naturally solve this hand-reach task by looking at the stimulus and then touching it, but the computer program forced them to reach without looking by aborting any trial in which a saccade greater than 2° was made. If we relaxed the eye movement restriction, the monkeys quickly resumed making the eye movement before the hand movement. This suggests that the hand-reach task requires a rather artificial behavior, whereas the other three tasks are naturally occurring behaviors in the monkey's normal repertoire. Monkeys were overtrained for several weeks on the three natural tasks. They learned to switch with relative ease from task to task, and they learned that the same stimulus might be irrelevant during one series of trials, a saccade target during another, and a significant peripheral stimulus during a third. In these cases, the behavioral context endowed identical stimuli with different significances for the monkey.

Experiments were usually run in blocks of no fewer than 16 trials of one task. We provided no cue or interruption between trials when we switched tasks. The only way the animal could discover that the task had changed was by appreciating differences in the action of the fixation point or receptive-field stimulus. The monkeys seldom missed the first saccade trial. For the transition from fixation to saccade this was simple: the fixation point disappeared as the peripheral stimulus came on. For the transition from fixation to peripheral attention this was difficult: the only clue was that the fixation point did not dim and the peripheral stimulus did. The monkeys frequently missed the first trial or two on which the peripheral stimulus dimmed. In order to prevent predictive behavior on the monkey's part, we did not use a standard sequence of tasks.

Physiological methods

We used standard recording techniques (47). Briefly, monkeys were prepared under ketamine-pentobarbital anesthesia for chronic single-unit recording, using the methods of Evarts (13, 14) and their modifications (39, 58). Recording cylinders, 2 cm in diameter, were placed normal to the skull approximately over the intraparietal sulcus. The position of the cylinder relative to the underlying sulci was verified using a mold of the sulcal markings on the underside of the calvarium (57). A single pair of bolts was implanted and a broad base of Codman neurosurgical acrylic was attached to these bolts and to the recording cylinder. A socket for attachment to the head-holding apparatus was attached to the first layer of acrylic and the skin was left open around the acrylic cap. The acrylic kept the bolts rigid, and the bone remained intact. Silver-silver chloride electrodes were implanted on the outer canthi and above and below one eye for electrooculographic (EOG) recording (4). After each monkey recovered from surgery he was placed each day in a primate chair with head restrained, and a platinum-iridium (5–6 microelectrode was advanced through the dura into the cortex for extracellular single-unit recording. The recording cylinder was irrigated with antibiotic solutions. Daily electrode placement and single-unit recording were performed without anesthesia. The EOG records were accurate to 1° in the horizontal plane and 2° in the vertical. The EOGs were calibrated by having the monkey make eye movements 20° apart on the tangent screen.

On-line data analysis and storage

On-line analysis of single-unit discharge patterns was performed with the PDP-11 computer. This allowed the neuronal discharge pattern to be synchronized with various events such as stimulus onset, eye movement initiation or termination, lever press, or fixation-point onset. Sixteen-line rasters or their histograms were continually available for display on an oscilloscope, as was a running display of monkey behavior, eye movements, and unit discharge. The computer could be set to discard automatically trials in which extraneous eye movement or inaccurate lever release occurred, or these erroneous trials could be examined exclusively. For each block of 16 trials the computer stored three rasters on the magnetic disk. The rasters were calculated from the same trials but synchronized on different events or calculated using different time bases.

Calculation of enhancement index

The rasters were subsequently used off-line as data for the calculation of a measure of enhancement of visual responses, the enhancement index, E. E is the ratio of the number of discharges evoked by the stimulus in an active task, such as saccade or peripheral attention, to the number of discharges evoked by the same stimulus in the fixation task. Background activity is subtracted from the responses in each condition. The enhancement index provides a measure of how behavior modulates the intensity of the response to a visual stimulus. It allows a comparison of the effects of different tasks on the same cell or the same task on different cells. Since it is a measure of the relative intensity of responses, it reveals nothing about the qualitative nature of the response of a neuron. It does not describe the absolute intensity of an individual response, its regularity or length, or any fine structure within a train of discharges.

Figure 3 shows the technique of computation of E. The neuron used as an example in the figure gave an enhanced response to a saccade target. The computer was used to display a cumulative histogram, and the investigator positioned cursors by hand to define a window around the neuronal response. To minimize experimenter bias, we used several procedural rules. The window was opened just before the inflection in the cumulative histogram line that signified the onset of the response to the stimulus. For the saccade task the window was closed at either the end of the neuronal response or the beginning of the eye movement, whichever came first. For the peripheral-attention task the window was closed at the end of the neuronal response. On those cells in which enhancement indices were studied for both saccade and attention tasks, the smaller of the above-determined windows was used for both tasks. A window of equal width for background activity was positioned to end less than 100 ms before the onset of the stimulus. The computer then counted the total number of discharges in each window for the 16 trials and subtracted the number in the background window from the number in the response window. This yielded the number of action potentials above background evoked by the stimulus. Using identical window placements, we computed a similar number for the response to the same stimulus in the fixation case. The ratio of the response number in the active task to the response number in the fixation task was defined as E, the enhancement index.

The enhancement index was only computed for blocks of trials in which the behavior was successful in all cases. If the monkey failed to release the bar or make the proper eye movement, that trial was not included in the raster.

An enhancement index of 1.0 signifies equivalent responses in the active and fixation tasks, and an enhancement index of 2.0 indicates that the response to a relevant stimulus in an active task is twice that in the fixation task. We selected an enhancement index of 1.5 to represent an enhanced response. This increase is always beyond the variability of indices calculated between two repetitions of the same task. In addition, Mann-Whitney *U* tests calculated on a limited number of cells were always significant at least to $P < 0.05$ for $E > 1.5$. When we calculated enhancement indices between blocks of different trials of the same task, the enhancement index was always less than 1.5. Therefore, we selected 1.5 as the criterion to signify an enhanced response.

A few cells had enhancement indices less than 0.67, which would signify a 50% decrement in the active case. This attenuation of the visual response may reflect an underlying partial suppression mechanism active under certain circumstances. However, in these experiments we could not distinguish a physiological attenuation related to the task from two other possibilities: the first is that the responses of many neurons in the parietal cortex tend to habituate after multiple stimulus presentations. Consequently, a decrement of neuronal response as the monkey performs various tasks could be due to the animal's behavior or due to habituation. We did not examine this distinction in the present experiments. The second is that in our sample, Mann-Whitney *U* tests in cases of $E < 0.67$ were not significant. This may be because the small number of discharges in each trial resulted in enough overlap to preclude significant rank ordering.

A negative enhancement index would imply that the cell was excited by the stimulus in the active task and inhibited by the same stimulus in the fixation task, or vice versa. We never saw this phenomenon.

Anatomical technique

For each monkey, many penetrations were made over a period of several months of neuronal recording. Consequently, not every penetration

was marked. Instead, the areas that were physiologically responsive to visual stimuli were located and these areas recorded on a grid map of the cylinder. Near the end of experimentation on a given monkey a few electrolytic lesions (10 uA × 60 s) were made at the sites of visual cells in the margins of the grid map of the visually active area. For control purposes we also marked anterior penetrations where we could evoke activity by somatosensory but not visual stimulation. These penetrations were in area 5. Just before perfusion, the animal was deeply anesthetized with pentobarbital and four pins were inserted into the brain at the margins of the cylinder, using a specially designed cap that fit over the cylinder and kept the pins parallel to the longitudinal axis of the cylinder. The animal was perfused with saline and Formalin with the pins in place. The gross brains were photographed with the pin placements marked and then sectioned and stained with cresyl violet. The areas from which the recordings were made were extrapolated from the location of the border-marking lesions, the pin placements, and the cylinder chart. Figure 2 shows the reconstructions of the two most disparately placed cylinders. The aggregate properties of visual cells did not differ among the cylinders.

RESULTS

A total of 534 neurons were recorded in area 7 of four monkeys. The recording area included the dorsum of the inferior parietal lobule and the posterior bank of the intraparietal sulcus. The surface of the visually responsive area is shown diagrammatically in Fig. 2. Of these neurons, 404 cells (75%) produced an excitatory visual response, 11 (2%) showed an inhibitory visual response, 26 cells (5%) showed both visual and somatosensory responses, and 61 cells (12%) showed only a somatosensory response. We were unable to drive 32 cells (6%). Since we were particularly interested in visual cells, we tended to spend more time studying them, frequently using up the monkey's appetite for water before we exhausted the cells available for study in a particular penetration. We rarely made penetrations at the same point twice, so we may have exaggerated the percentage of visually responsive neurons. Nonetheless, we did not find any neurons associated with eye movements that could not be driven by some visual stimulus (47).

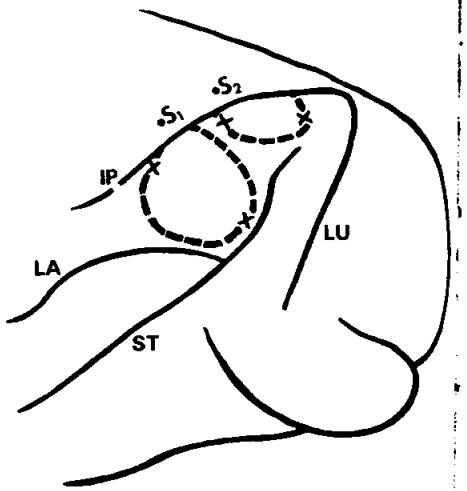

FIG. 2. Reconstruction of the two disparate-placed recording cylinders, superimposed on an outline of the lateral surface of the monkey brain. LU, lunate sulcus; LA, lateral sulcus; IP, intraparietal sulcus; and ST, superior temporal sulcus. The dashed outlines delineate the parts of area 7 within which visual cells were found. ×'s, penetrations in which lesions were made at sites of enhanced visual cells; S, locations of two penetrations that had only somatosensory receptive-field properties.

Presaccadic enhancement of visual responses

We obtained enhancement indices for 100 cells with excitatory responses in the saccade task. We did not test enhancement in cells inhibited by visual stimuli. As shown in Table 1, 39% (39 of 100) of neurons tested on the saccade task produced an enhancement index of at least 1.5, indicating a 50% increase in neuronal response when the stimulus was the saccade target. Such presaccadic enhancement has been observed previously (19, 20, 47, 63) and is illustrated by the cell analyzed in Fig. 3. Figure 4, top, shows distribution of enhancement indices for the saccade task. Distribution is unimodal with a peak around 1.0 (no enhancement) and a skew into the enhanced region. On those cells for which we were able to calculate enhancement indices for several sets of fixation and saccade trials, the indices remained in the same range.

TABLE 1. *Percentage of enhanced neurons*

Monkey	Saccade Task	No-Saccade Task
006	38 (8/21)	42 (11/26)
008	33 (12/36)	37 (9/24)
009	50 (9/18)	50 (5/10)
010	40 (10/25)	39 (14/36)
Total	39 (39/100)	41 (39/96)

Figures in parentheses are number of neurons enhanced/number tested.

A small percentage of the cells had a response attenuation large enough to produce enhancement indices of less than 0.67. This attenuation could indicate a mechanism actively suppressing the visual response under certain task conditions. However, these differences are not significant by Mann-Whitney *U* tests, and could be due to habituation or the fluctuation inherent in low background rates. Further work is under way to determine if there is indeed a statistically significant task-related response attenuation in certain parietal neurons.

Enhancement of visual responses in absence of saccades

We studied responses of 96 cells in the peripheral-attention task. Figure 5 shows the response of a cell that gave an enhanced response in the peripheral-attention task. Figure 5*A* shows the response of the neuron to onset of a behaviorally irrelevant spot of light in its receptive field. There is a burst of activity beginning approximately 100 ms after onset of the receptive-field stimulus. Figure 5*B* shows response of the same neu-

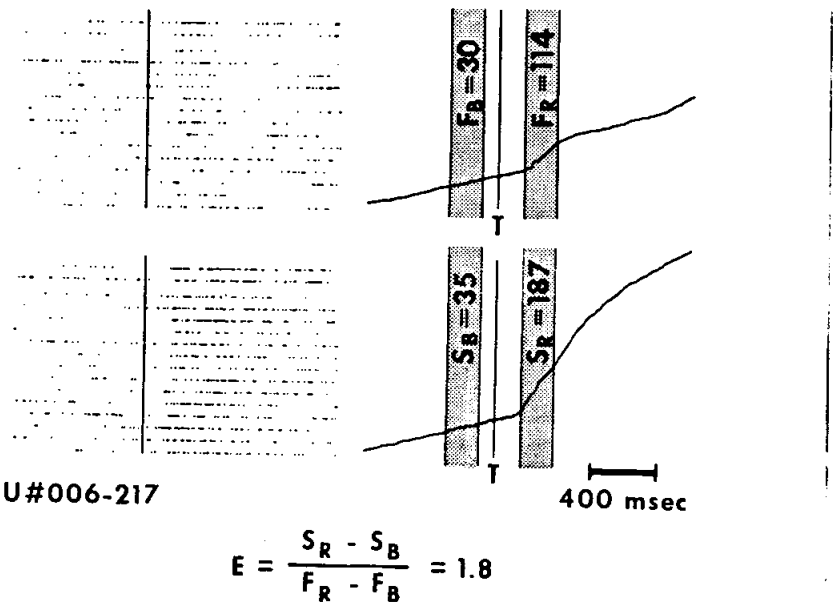

FIG. 3. Rasters show raw data for a parietal neuron during a series of fixation trials (upper raster) and during a series of saccade trials (lower raster). To the right of each raster there is a cumulative histogram computed from same data by summing the total number of unit discharges moving left to right. Superimposed on each cumulative histogram trace are trigger lines (T) corresponding to stimulus onset and two windows of equal width, one before the stimulus onset and the other after. These windows are labeled SB (for saccade background) and (SR for saccade response), respectively, in the saccade histogram, and FB (for fixation background) and FR (for fixation response), respectively, in the fixation histogram. Windows are identical with respect to width and position in each histogram. Numbers of spikes within each window are shown on diagrams. The enhancement index E is defined as ratio of number of spikes above background evoked by the stimulus in the saccade case to the number of spikes above background evoked by the stimulus in fixation case. In this example, E = 1.80 and $P < 0.01$ by Mann-Whitney *U* test. The height of the trigger line in the cumulative histogram corresponds to 921 discharges total.

ron to an identical receptive-field stimulus during the peripheral-attention task. When the monkey must detect a change in receptive-field stimulus without making a saccade to it, there is a larger discharge than when the stimulus is behaviorally irrelevant. Note that the receptive-field stimulus provides the signal for lever release in only half the trials, and the fixation point provides the signal for lever release in the remaining trials. Since the animal cannot predict whether the fixation point or the receptive-field stimulus will be significant at onset of the stimulus, it must attend to both all the time; the stimulus evokes in an enhanced response at each presentation.

Figure 4, bottom, shows distribution of enhancement indices for the cells studied in the peripheral-attention task. Thirty-eight cells (40%) had an enhancement index of 1.5 or greater.

Spatial specificity of enhancement

Enhancement in area 7 studied using the saccade task is spatially specific; the visual on-response is enhanced only when the animal makes a saccade to the stimulus in the receptive field but not when it makes a saccade to a stimulus outside the receptive field of the neuron (47). We therefore designed an experiment to see if the enhancement demonstrable with the peripheral-attention task is also spatially specific. We used a modification of the peripheral-attention task. While the monkey fixated the central fixation point, two peripheral stimuli appeared simultaneously: one in the receptive field of the neuron and one outside the receptive field. Trials were run in blocks of at least 32. In some blocks the fixation point or the receptive-field stimulus dimmed, and in other blocks the fixation point or the stimulus outside the receptive field dimmed. Visual stimulation in all these types of trials was the same: the same two stimuli and the fixation point always appeared on the screen. However, significance of the stimuli changed from block to block. Figure 6*A* shows the response of a neuron in area 7 to the onset of two behaviorally irrelevant stimuli: one within and one without the receptive field. Figure 6*B* shows the lack of enhanced response of the same neuron to the same stimuli after the animal had learned that the stimulus outside the receptive field might dim. Figure 6*C* shows the enhanced response of the neuron to the same stimuli after the animal had learned that the stimulus inside the receptive field might dim. The enhancement index computed for Fig. 6*A* and *B* was 1.13, and that computed for Fig. 6*A* and *C* was 2.04. We conclude that the enhancement that is independent of saccadic eye movements is also spatially selective. It is therefore unrelated to general behavioral effects such as arousal.

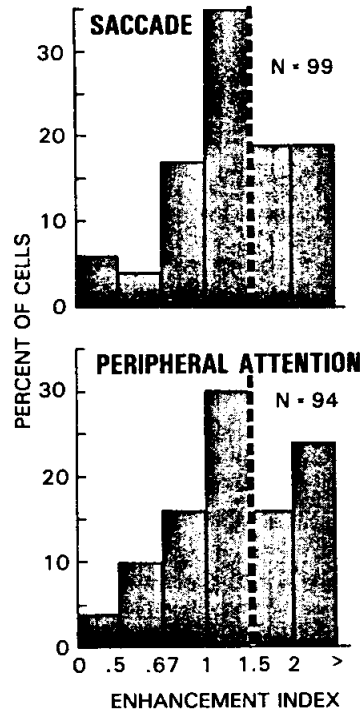

FIG. 4. Distribution of enhancement indices. The abscissa is range of enhancement indices as computed according to technique illustrated in Fig. 3, and ordinate represents number of cells in sample with that index. The top graph shows distribution of indices for cells studied when the animal made an eye movement to stimulus; the bottom shows distribution when monkey had to use stimulus but not make an eye movement to it. Dashed line marks an enhancement index of 1.5, corresponding to a 50% increase in firing, the selected criterion for an enhanced response. This graph illustrates cells for which either enhancement index was calculated. Not every cell has enhancement indices calculated for both saccade and peripheral-attention tasks.

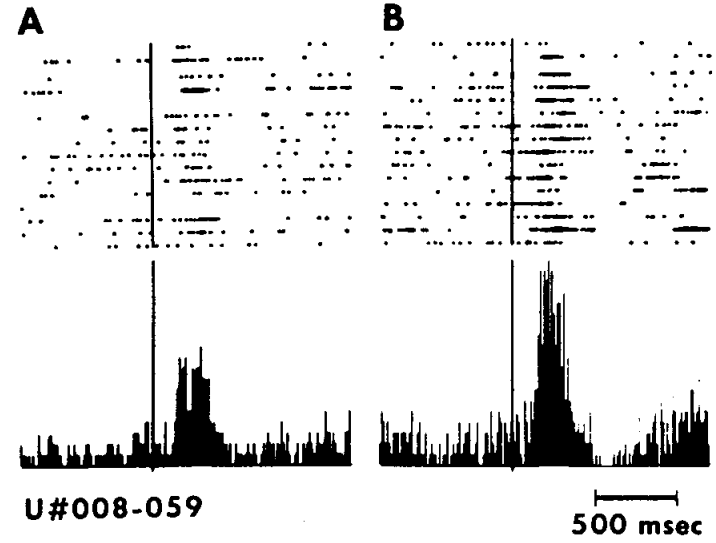

FIG. 5. Enhancement independent of any eye movement. *A:* response of a parietal neuron to the onset of a spot of light in its receptive field while the monkey fixated. *B:* enhanced response to the onset of same stimulus when the monkey was required to detect a dimming of the peripheral stimulus while fixating. Vertical line denotes when stimulus was turned on, and histogram sums data in adjacent raster. Conventions and format of this figure will be used for subsequent figures. Enhancement index is 1.65. The height of the vertical line corresponds to a discharge frequency of 114 Hz.

This experiment could be performed only on neurons with limited receptive fields that responded to fairly small stimuli, since one stimulus had to be placed outside the receptive field. The stimuli had to be small and far apart so that the monkeys could not easily attend to both stimuli at once. Because this experiment required the monkey to learn to attend to first one stimulus and then the other while the single neuron remained isolated, we were able to test successfully only eight enhanced cells. Seven of those cells produced an enhanced response only when the receptive-field stimulus dimmed, and one cell showed enhancement when either stimulus dimmed.

Equivalence of saccade and saccade-independent enhancement

Several aspects of our data suggest that cells with discharges enhanced before eye movements are the same population as those enhanced during attentional processes not involving eye movements. First, the percent of enhancements on the saccade task and peripheral-attention task are similar for each monkey (Table 1). Second, the distribution of enhancement indices for the peripheral-attention task resembles that for the saccade task (Fig. 4). Both distributions are unimodal with peaks around 1.0 (no enhancement) and skew into the enhanced region.

In order to test directly if the enhancement before eye movements and the enhancement in association with eye movement-free attention were indeed the same, we studied the responses of 31 neurons in both the saccade and peripheral-attention tasks. Comparison of these two tasks required a visual stimulus large enough for the monkey to detect its dimming peripherally yet small enough for the monkey to use as a saccade target. Figure 7 shows the response of a cell to the receptive-field stimulus on the fixation task (Fig. 7*A*) and the enhanced response to the same stimulus used in the saccade task (Fig. 7*B*) or the peripheral-attention task (Fig. 7*C*). For this cell the response was slightly larger on the pe-

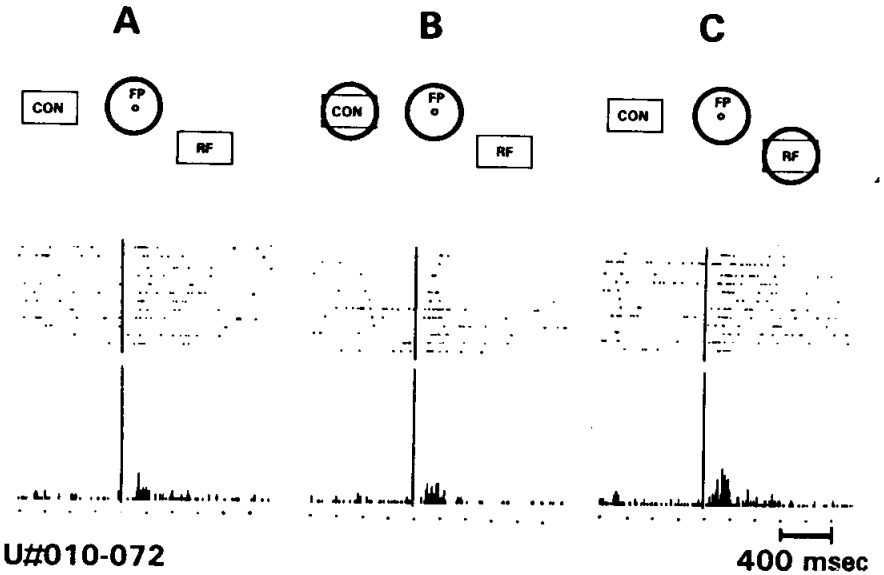

FIG. 6. Spatial selectivity of eye movement-independent enhancement. The monkey looked at the fixation point (FP) and was presented with two stimuli: RF in the receptive field and CON, a control stimulus beyond the receptive field. *A:* discharge of a parietal neuron to the onset of both stimuli while the monkey fixated and detected the dimming of only the fixation point (circled). *B:* response of this cell to the same stimuli when the monkey was required to use the stimulus outside the receptive field without making any eye movement to it. In this case the fixation point or the control stimulus might dim (both are circled). *C:* enhanced response under the same stimulus conditions when the monkey used the stimulus in receptive field. Either the fixation point or the receptive-field stimulus could dim. Enhancement indices are 1.13 for *A/B* and 2.04 for *A/C*. The height of the vertical line corresponds to discharge frequency of 152 Hz.

ripheral-attention task than on the saccade task, but for other cells the reverse was true. Twelve (39%) of the cells studied produced an enhanced response on both tasks, 18 (58%) did not produce an enhanced response on either task, and one cell had an enhance-

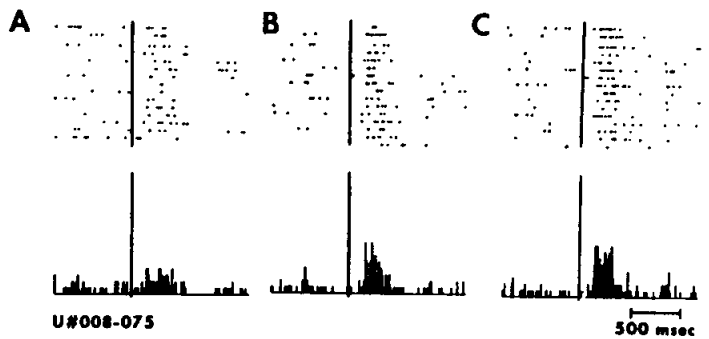

FIG. 7. Demonstration of presaccadic and eye movement-independent enhancement in the same cell. Data in *A* show response of a cell to the onset of a visual stimulus while monkey fixated; *B* illustrates the enhanced response to onset of the same stimulus when monkey is going to make an eye movement to it. *C* shows that the same cell has an enhanced response when animal must attend to it but cannot make an eye movement toward it. Enhancement indices are 2.54 for *A/C* and 1.97 for *A/B*. The height of the vertical line corresponds to discharge frequency of 114 Hz.

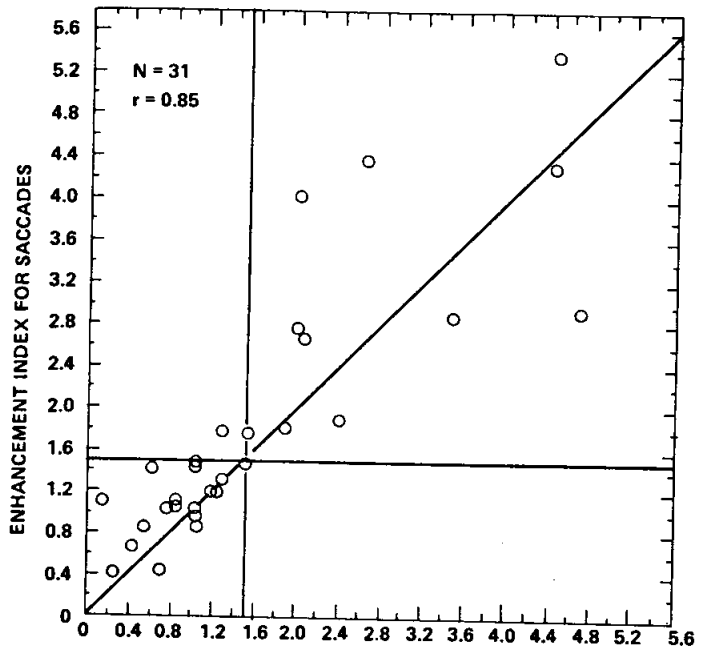

FIG. 8. Scatter diagram for saccadic and attentional enhancement. Each circle represents a cell tested for both saccadic and attentional enhancement. The ordinate corresponds to enhancement indices determined for the saccade task; abscissa lists indices for the peripheral-attention task. The horizontal and vertical lines within the graph at 1.5 depict criterion level for classifying cells as enhanced.

ment index of 1.52 on the saccade task and an enhancement index of 1.46 on the peripheral-attention task. Figure 8 is a scatter diagram showing the enhancement indices for the saccade and the saccade-free tasks for each cell studied on both tasks. The correlation coefficient is 0.85 between the two indices. In general, cells either yielded enhanced responses for both tasks or neither. We did not find a population of cells with attention-only or saccade-only enhancement. This differs from the frontal eye fields (17) and the superior colliculus (61) where all enhancement is saccade only. Thus, the processes of presaccade and eye movement-free enhancement in area 7 are either identical or very tightly linked.

Hand-reach enhancement

As a further test of the independence of enhancement from the eye movement response, eight cells in two monkeys that produced an enhanced response on the saccade task were tested on the hand-reach task. All of these neurons produced an enhanced response when the monkey touched the receptive-field stimulus without making an eye movement. Figure 9 shows the results of a typical hand-reach experiment during fixation. The neuron gave a weak response to the stimulus onset (Fig. 9*A*) and a much more robust response to the stimulus when it was the target for a saccade (Fig. 9*B*) and also when it was the target for a hand reach (Fig. 9*C*). It is important to emphasize that in the hand-reach task the visual situation is changed by the hand traveling through the receptive field to reach the panel. However, the enhancement clearly begins before the hand begins to move.

Independence of latency and enhancement

The latencies of responses to receptive-field stimuli ranged from 40 to 236 ms, with

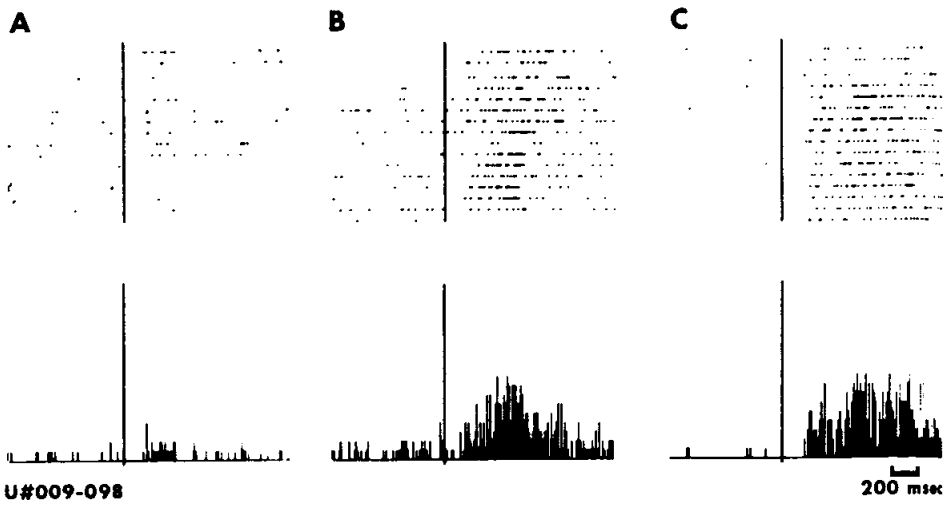

FIG. 9. Enhancement in same cell for eye and hand-reaching movements. The cell responds weakly to onset of a visual stimulus while monkey fixates (*A*). The response to onset of same stimulus was enhanced when the stimulus was target for a saccadic eye movement (*B*) or a hand reach (*C*). Enhancement indices are 2.37 for *A/B* and 2.47 for *A/C*. The height of the vertical line corresponds to discharge frequency of 114 Hz.

a mean of 98 ms and a mode of 76 ms. This unimodal latency distribution has been reported previously (47). There was no correlation between response latency and enhancement index. Figure 10 is a scatter diagram showing the latency and the enhancement index for each cell studied. There is no correlation between latency and saccade enhancement ($r = 0.097$) or between latency and peripheral-attention enhancement ($r = 0.001$).

DISCUSSION

Attention as a physiologically approachable phenomenon

Psychologists have used attention to mean various things, all of which probably have different psychological correlates (3, 43). Attention may refer to intensive phenomena such as alertness, vigilance, or arousal that regulate how the organism interacts with the environment as a whole. Alternatively, attention may refer to selective phenomena that involve how the organism responds to competing stimuli. This type of attention can select among different sensory modalities: for example, choosing auditory stimuli over visual stimuli. It can also involve the abstraction of a specific stimulus property, such as a color or one conversation at a cocktail party. Finally, attention can be selective in terms of stimulus location; one can pay attention to a specific part of his visual field. Our experiments deal only with the latter form of spatially selective visual attention.

Usually, when an animal directs its attention to a specific location it makes a receptor-adjusting or orienting response. This may be a gaze shift, but it may also involve pinna orientation (8, 53) or hand orientation to further tactile exploration (22). It is also possible to direct one's attention without making an orienting response (30, 44). Responding to tachistoscopically flashed stimuli is one example of this sort of dissociation. Posner and his group (43, 44) have shown that human subjects have faster manual reaction times to the onset of a peripheral stimulus when they are told to attend to the location of that stimulus while they fixate a light elsewhere than when they are told to attend elsewhere. Thus, there must be a neural mechanism that underlies spatially selective visual attention that is not obligately tied to a receptor-adjusting response. We suggest that enhanced visual responses

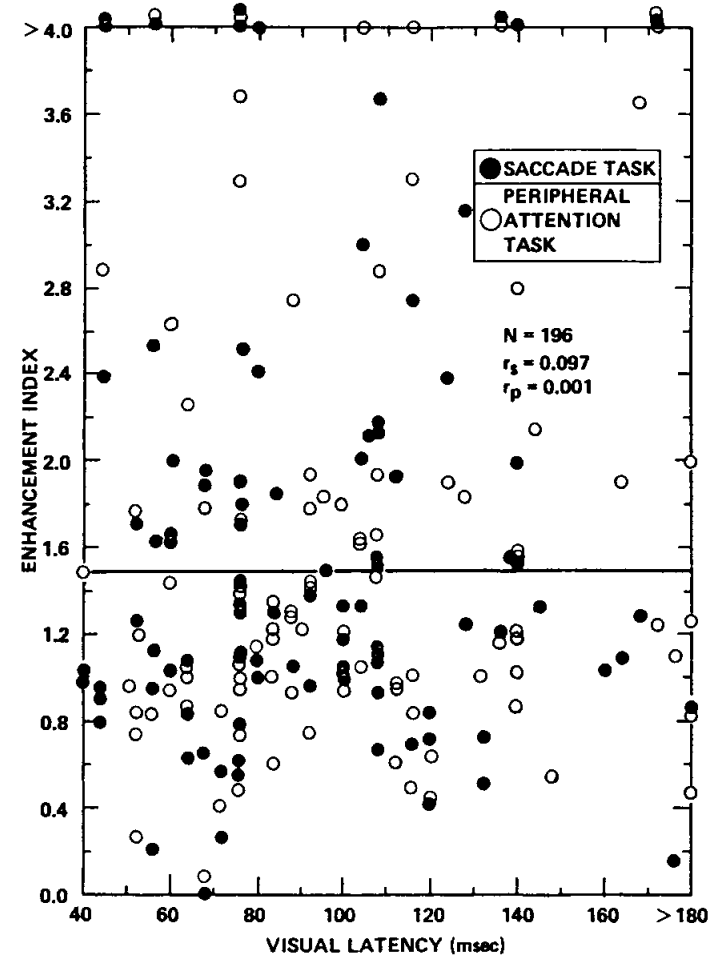

FIG. 10. Scatter diagram for visual on-response latency and enhancement index. The ordinate corresponds to the range of enhancement indices with the horizontal line at 1.5 indicating the criterion level for classifying a cell as enhanced. The abscissa represents range of visual response latencies. The filled circles correspond to data for cells tested with eye movement task; the open circles indicate results from the peripheral-attention task. Cells tested for both tasks are represented by two circles.

in area 7 may participate in such a mechanism.

Enhancement of visual response in area 7 was first described in association with eye movements (19, 20, 47, 63). Our experiments now show that when an animal attends to a visual stimulus the response to that stimulus is enhanced. This enhancement occurs when the monkey makes a saccade to a stimulus, when he reaches to touch it, or when he might have to use it as a cue for some behavior that does not require a targeted movement. If a cell in area 7 yields an enhanced response in one task the overwhelming probability is that it will yield an enhanced response in all tasks studied. Conversely, a cell that does not yield an enhanced response on one task will not yield an enhanced response on any task. This is markedly different from the superior collic-

ulus (61) and the frontal eye fields (17) where cells that do not give an enhanced response in the peripheral-attention task may well give an enhanced response in the saccade task. Since enhancement is spatially selective, it does not occur when the monkey attends to or makes a saccade to a stimulus far from the receptive field of the neuron (47). Thus, activity in area 7 is not associated with general arousal or specific movement, but is associated with selective spatial attention. Since purposeful movement is usually associated with spatial attention, it is not surprising that the enhancement occurs in association with eye or hand movements to the attended stimulus. The fact that the enhancement occurs with three different kinds of behavior in which the animal attends to the stimulus makes it likely that the enhancement is involved in a very general process of selective visual attention. Of course it is always possible to consider the parietal signal as an oculomotor signal, which is canceled later on. This is unlikely for two reasons. The first is that it occurs in the peripheral-attention task, a task that the animals naturally learn to perform without ever making eye movements to the stimulus. Second, the general attentional nature of parietal enhancement contrasts strongly with the specific oculomotor enhancement of the frontal eye fields (17) and the superior colliculus (61). It would be strange indeed if the brain were forced constantly to cancel a specific parietal oculomotor signal yet produce a frontal signal sufficiently differentiated not to require such cancellation.

Varieties of response enhancement

Mountcastle and his group (36, 39, 63) described neurons in area 7 that discharge in association with saccadic eye movements, and they postulated that these neurons direct attention by commanding saccades to attended stimuli. They also stated that these neurons were not light sensitive, although they subsequently described light-sensitive cells that they considered to provide a visual input for the oculomotor mechanism (63). In our previous study (47) we demonstrated that some passive visual response could be found for every saccade-related neuron that we encountered. We now show that there is an eye movement-independent process of enhancement in area 7 that may certainly underlie visual attention. We did not, in the population studied, find evidence for a saccade-related process that was absent when the animal attended to the stimulus without making an eye movement to it. It is possible that such a mechanism does occur in area 7 and that its representatives eluded our sample. Such a sparse distribution would be in dramatic distinction to the preponderance of cells exhibiting saccade-specific enhancement in the frontal eye fields (17, 18) and the superior colliculus (61). Conversely, these areas that contain the specific oculomotor enhancement give no evidence of a more general attentional enhancement. Of course, the attentional processes in area 7 play an important part in the guidance of eye movements, just as the visual processes in the retina play an equivalently important role. However, neither plays a specific role in the sense that one cannot predict from the discharge of an enhanced visual neuron in area 7 that an eye movement will take place.

In order to understand the significance of enhancement of visual responses in the posterior parietal cortex, it is important to consider the characteristic of enhancement in other areas in which it has been studied. Enhancement was first demonstrated in the superior colliculus (21). Enhancement in the frontal eye fields has properties similar to collicular enhancement (17, 18, 60). In both of these cases, since the enhancement occurs only before an eye movement, it has been considered as a mechanism by which the visual information implicit in the neuron's discharge can be efficiently transmitted to the oculomotor system (47, 59). Enhancement has been described in striate cortex (60) and prestriate cortex (45), where it is spatially nonselective and response independent. Thus, if the discharge of a neuron in striate cortex is enhanced before a saccade to the stimulus in the receptive field, it will be enhanced before all saccades, even those to targets far from the receptive field. Thus the enhancement does not encode the direction of eye movement. In addition, the discharge to the stimulus will also be enhanced when the animal attends to the stimulus without making an eye movement (60). This kind of enhancement clearly cannot participate in spatially selective attention or in a specific motor behavior, but it could easily be involved in the processes underlying arousal. Recently the pulvinar has been shown to have spatially nonselective enhancement that occurs in association with eye movements but not in association with other attentive behavior. Such enhancement may be used in processing the changes in visual information associated with an eye movement (28).

We have shown that enhancement in area 7 is associated with visual attention, but in order to direct the monkey's attention to a stimulus we paired that stimulus with a reward. Rolls and his colleagues (48) showed that area 7 neurons respond equally well to attended stimuli that are aversive. These findings suggest that enhancement that we observe in area 7 is dependent more on the monkey's selective attention than on the stimulus' specific motivational significance.

Anatomic substrates of visual enhancement

The behavioral enhancement of visual responsiveness in area 7 has two different components. The first is the visual input responsible for driving the cell. The second is a behavioral input that modulates the visual response. Previous studies have shown that the parietal neurons do not discharge when the proper eye movements spontaneously are made in total darkness (36, 47). Therefore, the behavioral input does not excite the cell by itself: only the visual input can serve that purpose.

Anatomic studies of the afferents to area 7 can provide candidates for the sources of both the visual and behavioral components of the enhanced visual response. Visual responses could come from several areas: medial and oral pulvinar, lateral posterior nucleus (1, 2, 11, 27, 38, 50, 51), the frontal eye fields (31, 50, 51), and prestriate cortex (11, 33) all send projections to area 7.

Enhancement has been demonstrated in the striate cortex (60), prestriate cortex (45), and the pulvinar (28), but does not have the properties of spatial selectivity and task independence that we described in area 7. Consequently, the enhancement in area 7 probably arises from another source. Such a source could be the rich limbic inputs to area 7, those from the substantial innominata, claustrum, and cingulate gyrus (29, 38, 50, 51). Neurons in the cingulate gyrus respond to the onset of a reward-associated stimulus with a latency of 80–120 ms (40). Other neurons in this area discharge when the monkey anticipates the onset of a reward-associated stimulus and cease discharging when the stimulus appears. Some neurons in substantia innominata respond to stimuli not associated with food (49). Others respond specifically to aversive stimuli with comparable latencies. Activity of these limbic neurons could set up a tonic state of readiness in area 7 so that when the attended stimulus appears the visual response would be enhanced. Since the limbic neurons seem to be specific for quality of the motivation, that is, whether it is appetitive or aversive, area 7 may serve to integrate motivation-specific information into a general attentional mechanism (48). The fact that the latency distribution of enhanced neurons is similar to that of unenhanced neurons implies that there is not a large amount of serial processing occurring in area 7 before the enhancement is manifest.

Functional considerations for area 7

Area 7 projects to areas involved with eye movement (the intermediate and deep layers of the superior colliculus and the frontal eye fields) (32), other movements (premotor cortex (41) and basis pontis (16)), and to the prefrontal areas involved with integration of complicated behaviors such as delayed response (7). Presumably, a target that results in an enhanced visual response in area 7 could then contribute to an eye movement, a hand movement, or the sensory data for a delayed response. We have not found populations of neurons that are movement specific in their discharge pattern. Since different parts of area 7 do have different projection fields (7), it is possible that different areas that give the same sort of visual and behavioral discharges have different functions by virtue of their different projec-

tions (12). A weakness of the extracellular method is that we do not know the anatomical efferents of the cells from which we record, and the functional significance of these cells may be limited by their efferent connections as well as by their discharge patterns.

In studying neuronal discharge patterns, we can only propose that area 7 participates in a general mechanism for visual attention by providing an amplified signal corresponding to an important visual stimulus. The nature of the response that the animal will make to that stimulus is generated by other areas.

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Behavioral Enhancement of Visual Responses in Monkey Cerebral Cortex. II. Modulation in Frontal Eye Fields Specifically Related to Saccades

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SUMMARY AND CONCLUSIONS

1. We studied the activity of visually responsive neurons in the frontal eye fields of awake rhesus monkeys while the animals performed a variety of visual tasks in order to assess the role of these neurons in visually guided behavior.

2. The majority of neurons in a small region of the most posterior part of the prearcuate gyrus have visual receptive fields. The visual response of over half of these neurons is enhanced when the animal uses the stimulus in the visual receptive field as a target for an eye movement. This burst of enhanced visual activity is time-locked to the stimulus onset, and it precedes the eye movement.

3. The response of these neurons is not enhanced when the monkey performs a task that requires it to attend to the stimulus without making an eye movement toward it. In addition there is no enhanced response when the animal reaches out and touches the stimulus if he is not allowed to make a concurrent eye movement.

4. When the monkey makes an eye movement to a stimulus that is on continuously, there is a burst of activity that precedes the eye movement even though the visual response to the stimulus may have already adapted. However, there is no discharge before similar eye movements when a visual stimulus is absent.

5. These results suggest that the visually responsive cells in frontal eye fields may provide a retinal error signal to the brain stem gaze-shift centers, and the presaccade enhancement of these visual responses may be a cortical component of the neural events preceding purposeful, visually guided saccades.

INTRODUCTION

Since the 19th century the frontal eye fields have been thought to be involved in the neural events preceding eye movements (8). Supporting evidence comes from both clinical and electrophysiological observations. Thus, Holmes (18) noted that frontal tumors result in seizures that begin with contraversive eye movements and that patients with frontal lesions tend not to make eye movements into the visual field contralateral to their lesions. Ferrier (13) observed eye movements into the contralateral field with electrical stimulation of the monkey frontal eye fields. More recently, Robinson and Fuchs (40), Schiller (44), and Marrocco (30) produced saccades by stimulation of monkey frontal eye fields. These saccades are stereotyped and their direction depends on the location of the stimulating electrode in the frontal eye fields. Prolonged stimulation leads to multiple, defined saccades rather than single, prolonged ones. Simultaneous stimulation of two frontal eye field sites result in an eye movement that is the vector sum of the eye movements produced from either site alone (40).