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TEMPORAL RESPONSE CHARACTERISTICS OF CELLS IN MONKEY STRIATE CORTEX MEASURED WITH METACONTRAST MASKING AND BRIGHTNESS DISCRIMINATION

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SUMMARY

After measuring metacontrast masking psychophysically in two monkeys, recordings from parafoveal striate cortex of the monkeys were made while they performed a simultaneous brightness discrimination and while they judged the apparent brightness of a stimulus masked by metacontrast. The size and orientation of the stimuli were held constant regardless of receptive field parameters. In both tasks, the single-cell activity immediately following the presentation of flashed discrimination stimuli reflected only stimulus parameters, and was independent of the monkey's behavioral choice. Later activity (up to 400 msec post-stimulus) was significantly greater if the monkey was about to press the correct panel in the discrimination, or if he pressed the unmasked side (with greater apparent brightness but identical intensity) in the masking paradigm. One quarter of the cells showed a change in firing rate during the 250 msec preceding the behavioral response, though the difference in overall firing level between correct and incorrect brightness discrimination trials was diminished in this epoch, and the corresponding firing difference in metacontrast trials was not significant. The temporal pattern of firing also differed between correct and incorrect trials in the pre-response interval. The results suggest an iterative or recurrent coding of visual information, where the same cells participate in early, late, and pre-response coding in different ways.

INTRODUCTION

When two discrete visual stimuli sharing a common contour are flashed on a screen about 60 msec apart in time, the brightness of the first stimulus appears to be reduced. This 'metacontrast' masking phenomenon is of interest because it appears to

work backwards in time. The backward masking function is commonly 'U-shaped': there is little or no masking when the two stimuli are presented simultaneously, and masking reaches a maximum at a stimulus onset asynchrony (SOA) of 50–150 msec[1].

Metacontrast tells us that the neural code of a stimulus remains vulnerable to interruption from the activity elicited by a second stimulus, even after a delay of 100 msec or more. The delay allows metacontrast to be used as a tool to analyze the temporal aspects of visual coding.

Metacontrast has been investigated at the retinal level in cats[20], where masking was found only at SOAs too short to be responsible for the effects observed in humans. In the lateral geniculate nucleus (LGN) of the dark-adapted cat, Schiller[32] obtained only monotonic masking functions. This result would be expected if metacontrast requires inhibition, for surround inhibition disappears at low light levels. Monotonic masking also occurs, however, in LGN cells of the light-adapted cat[9]. The psychophysics of metacontrast also argues against a locus of the effect in the LGN, for strong masking can be obtained dichoptically[34,45] while binocular interactions in LGN cells are weak or absent. For these reasons we have begun the search for physiological mechanisms of metacontrast in the striate cortex.

Other studies have measured evoked potentials in human subjects during metacontrast masking. Vaughan and Silverstein[44] found a decrease in a 200-msec latency component of the visual evoked potential; the area under this component closely paralleled the psychophysical apparent brightness of the test stimulus.

At the single-cell level, Bridgeman[9] studied masking functions in the cat's retina, LGN, and striate cortex, using stimuli which elicit metacontrast in humans. Unit responses were analyzed into early and late components, with the latency and duration of the late component matching the late interval defined by Vaughan and Silverstein[44]. At lower anatomical levels only monotonic masking was found, in agreement with the results of others, but in the striate cortex a reduction of late activity occurred at target-mask SOAs which correspond with those eliciting metacontrast in humans. Early components of the response were not affected.

The finding of correlates of metacontrast only in late responses is not surprising — indeed, it is a logical necessity, for the earliest response of most cortical cells to the target stimulus is already dissipated by the time the delayed 'mask' stimulus appears. Wagman and Battersby[45] first pointed out that the early response is a necessary condition for perception but not a sufficient one. The implication is that the early and late components of the single-cell response have different functions, the first component being more closely related to physical stimulus parameters while the second is influenced by other factors as well. This was also the conclusion of the earlier study on the mechanism of metacontrast in single cells of the cat[9]. The psychophysical characteristics of metacontrast masking in the cat have never been assessed, however, so that the correspondence of physiological data with psychophysical characteristics was inferred on the basis of the general parallel between cat vision and human vision. By investigating mechanisms of metacontrast in the monkey, psychophysical and electrophysiological data can be compared directly and metacontrast can be used as a tool to examine the temporal aspects of stimulus coding in single cells of visual cortex.

The little attention which has been given to temporal aspects of stimulus coding in single cells of the monkey is primarily in terms of the sustained–transient continuum[33] or the similar modulated vs fusional responses[26]. These distinctions concern only the first response of a neuron to a stimulus, however, and do not extend to later activity which can be differentiated from the first burst of a neuron's stimulus-induced response.

Reports of late secondary bursts in monkey visual cortex are limited to a few examples fortuitously published in illustrations intended to demonstrate other effects. Fig. 4 of Wurtz[49], for example, shows a peak of single-cell activity about 200 msec after the larger primary burst of firing elicited by a long bar moving across the receptive field at 900°/sec. The 200-msec late peak also appears when the monkey makes a saccadic eye movement across the bar stimulus. The peak consists of only 1–3 spikes in each trial, however, and is missing completely in some trials, so that it is doubtful that the phenomenon would have been apparent from simply listening to the cell's activity. A similar late peak is apparent in Fig. 4 of another paper by Wurtz[48] when a saccade is made across a bar stimulus, especially when the bar is only 0.5° wide. The role of these late peaks in the coding of brightness will be analyzed here.

The principal hypothesis to be examined, then, is that information necessary for stimulus detection is carried in the primary peak of the single-cell evoked response, while a second later peak carries information related to apparent brightness. The primary emphasis is on stimulus coding rather than on metacontrast per se. A secondary emphasis is the role of striate cortex neurons in organizing the behavioral response.

METHODS

Training

In the first phase of training, two sub-adult *Macaca fascicularis* monkeys were shaped to perform a simultaneous brightness discrimination of briefly flashed stimuli. Two discs 3.6° in radius were back-projected for 40 msec on a tangent screen, with each center 8.05° from the centerline of the screen. One disc was 80% as bright as the other, and the water-deprived monkey was rewarded with juice for pressing a panel on the side of the screen where the brighter disc had appeared. A ring surrounded each disc, equal in area to the disc and with its inner edge 3 mm from the edge of the disc. The disc and ring were flashed simultaneously, and monkeys were run 300 trials per day. The intertrial interval (ITI) was 2 sec and the penalty delay 4 sec. Following this part of the training procedure, which was completely automated, each monkey was overtrained for at least 3000 trials to stabilize performance.

The monkey was then shifted to a fixation task, where he was rewarded for a brief glance at the fixation light. Fixation was assessed using two methods: at first we mounted a mirror directly in front of the monkey at a 45° angle to his line of sight and observed the corneal reflection of the fixation light[2,23]. Later we observed the monkey through a closed-circuit TV system consisting of an IR-sensitive camera and a monitor, with the monkey illuminated by an IR light source. This method was used in

all single-unit recording trials. The camera was mounted close enough that only part of the monkey's head was visible on the screen. When the monkey was reliably performing on the fixation task, the required fixation interval was increased until several seconds of fixation were necessary to receive reward. This aspect of training was controlled directly by the experimenter.

During testing a trial was aborted if fixation was not maintained. Because receptive fields were superimposed on a homogeneous screen after stimulus presentation, an eye movement of at least 4° would have been required to move the receptive field to the nearest contoured area (the fixation point); this was well within the resolution of our system. Smaller eye movements should have no effect on the activity of striate neurons, since only a very small proportion of striate cortex neurons (less than 5%) respond to eye movements in the absence of stimuli on their receptive fields[7,24,49].

Masking

To assess the properties of metacontrast masking in the monkey, the fixation task was chained to the brightness task. At the start of a trial the fixation light would come on, and when the monkey fixated it the experimenter instead of giving a reward would initiate a brightness trial with a switch connected to the computer. All aspects of the testing procedure except for trial initiation were under computer control, so that the experimenter did not know which type of trial would be presented next. If fixation did not occur within 2.4 sec, the fixation light would go out for 0.6 sec before the light cycle started again. If the monkey did not respond with a panel press within 2 sec of the presentation of the flashed stimuli, the trial was repeated. Viewing was binocular.

A trial consisted of one of 3 types of displays (Fig. 1): (1) a projector would project on one side of the screen an equally bright concentric disc and ring, and on the other side a ring filled with a filtered disc. The monkey was rewarded only for pressing the side corresponding to the brighter disc, and incorrect trials were repeated ('brightness trials'); (2) a projector would flash a concentric, equally bright disc and ring on one side (the 'simultaneous side') and a disc only on the other side (the 'delay side'). After a variable SOA another projector would flash a 'mask' ring concentric with the disc which had been flashed alone ('metacontrast trials'). The delayed-ring condition is a stimulus configuration adequate to demonstrate metacontrast masking in humans[1]. On these trials the monkey was rewarded for pressing either panel; and (3) these trials were the same as the metacontrast trials except that the mask on the delay side was a large ring, 13.3 cm in inside diameter and of the same width as the small ring ('large-ring trials'). Under these conditions apparent motion and double stimulation are seen by human observers, but masking of brightness does not occur[1]. Thus the large-ring condition is a control for these factors.

Electrophysiology

During single-cell recording monkeys were tested in blocks with the design shown in Fig. 1, except that all metacontrast trials were run at the previously determined optimum SOA.

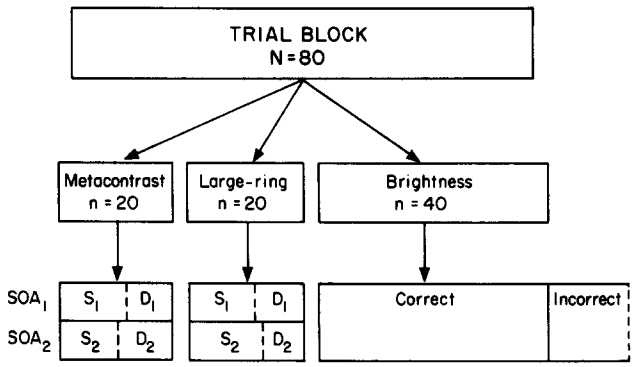

Fig. 1. Breakdown of an 80-trial block into 3 trial types. Solid lines represent independent variables, while dashed vertical lines represent dependent variables and change in each block according to the monkey's behavioral responses. S, psychophysical response on the simultaneous side (disc and ring together); D, response on the delay side (disc followed by ring at the corresponding SOA).

Preparatory to single-cell recording each monkey was accustomed to performing the psychophysical testing procedure in a primate chair. The monkey was then surgically implanted with a head plug for chronic physiological recording[6]. Wells were located so that all recording took place in the part of area 17 posterior to the lunate sulcus and superior to the lateral calcarine sulcus where the cortical surface is nearly parallel to the skull surface.

Single-cell data were obtained either with laboratory-produced microelectrodes[37] or with commercial tungsten electrodes (F. Haer). The electronic apparatus followed standard techniques.

Receptive fields were mapped with two methods. The first used a small hand-controlled back-projected line or spot, which could be swept across the screen in any direction, orientation or speed while another experimenter administered fixation trials. The receptive field was determined from the cellular activity on an audio monitor. The second method used a back-projected bar of variable orientation, which was swept across the tangent screen at variable speed by a computer-controlled mirror. The experimenter would initiate a trial by illuminating the monkey's fixation light. When the monkey fixated on the light the experimenter would initiate a pair of sweeps; the bar would sweep in one direction for 500 msec, pause for 100 msec, and then return in 500 msec. If the monkey maintained fixation he was rewarded with juice and the trial was stored in the computer, while a failure to maintain fixation resulted in no reward and discarding of that trial's data.

A recording session began with chairing the monkey, fixing the head, and attaching recording hardware. Body temperature, blood gases, accommodation, pupil size and corneal condition were maintained at optimum levels by the monkey's natural mechanisms. When a cell was isolated its response was briefly characterized by hand-mapping, and recording was begun with a block of 80 trials. If necessary, the stimuli were moved vertically to fall into the cell's receptive field. The data collection process was automatic except for eye movement monitoring and trial initiation, as described above. During recording one experimenter continuously monitored the single-cell

record on an oscilloscope while another monitored eye fixation and initiated trials. At the conclusion of an 80-trial block the single-cell data were displayed on a second oscilloscope, and if possible the cell's receptive field was then mapped with the computer technique. A complete analysis of one cell occupied several hours, and only one cell was held long enough to record two complete blocks of trials. (These blocks were combined for statistical analysis because independence could not be assumed.)

PSTHs beginning 100 msec before onset of the first (target) stimulus and extending 400 msec beyond it in 10 msec bins were recorded separately for brightness, metacontrast and large-ring conditions. Trials on which the response had been correct (or on the 'simultaneous' side) were collated separately from those on which the monkey had been incorrect (or had pressed the 'delayed' side), so that 6 PSTHs were collected for each 80-trial block. Similarly, response-synchronized activity was collected separately for each of the 6 conditions beginning 250 msec before the monkey's response and ending at the time of response. Background activity was determined from the 100 msec sample collected before each trial, when the monkey was in the state of arousal and attention which would occur during the trial itself. The total sample in each cell consisted of at least 8 sec of background activity (100 msec/trial × 80 trials), plus additional data collected before incorrect brightness trials.

Evoked responses were divided into 'early' and 'late' analysis intervals using a priori criteria based on earlier work on physiological correlates of metacontrast in humans and cats[9,44] (Fig. 2).

When recording was no longer possible from the second implant site, the implanted plug was removed surgically and the monkey used in other experiments.

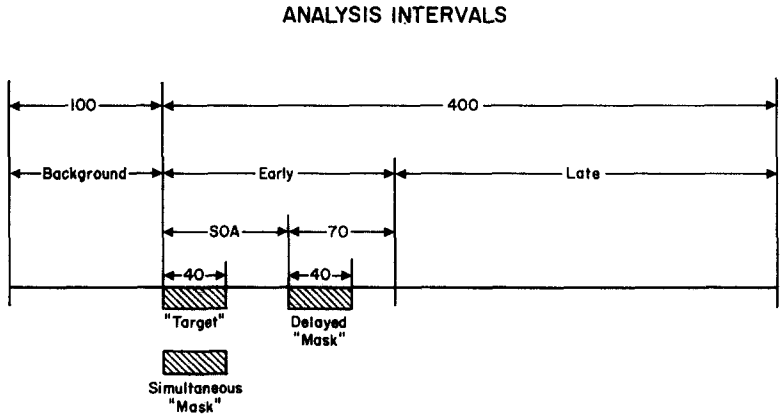

Fig. 2. Analysis intervals for metacontrast and large-ring trials. Data collection began 100 msec before the onset of the first 'target' stimulus, a light disc. The ring-shaped 'mask' stimulus surrounded the target spatially but was delayed in time. Data analysis intervals for the brightness trials were the same as those shown here even though the mask stimulus was simultaneous with the target on both sides of the screen. All numbers are times in msec. SOA, stimulus onset asynchrony. To avoid possible contamination from the primary response to the mask, the late interval always began 70 msec after mask onset.

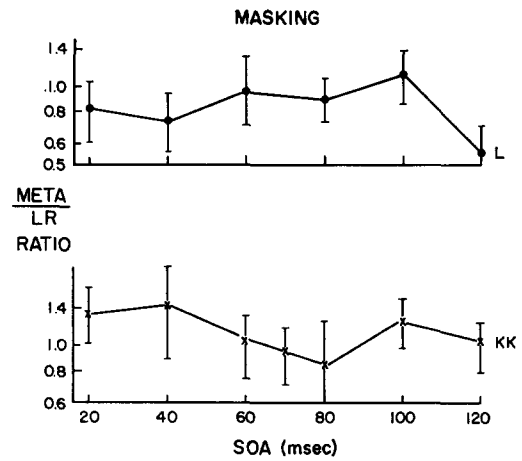

Fig. 3. Metacontrast masking functions in two monkeys. The y-axis is the ratio of responses to the masked side in the metacontrast condition (delayed mask) to responses on the masked side in the large-ring condition (identical temporal parameters but with an annulus distant from the target disc). An extra data point was obtained in monkey KK at 70 msec SOA to verify the location of the peak masking. Each point in the figure is based on 100 metacontrast trials and 100 large-ring trials. The length of the vertical bars is one standard deviation.

RESULTS

Psychophysical metacontrast

Masking curves (Fig. 3) show that the two monkeys had rather narrow temporal masking ranges, one peaking at 80 msec and the other 120 msec. Since each point in Fig. 3 is a mean of data from 10 trial blocks, standard deviations could be calculated from the 10 resulting meta/LR ratios. Thus the standard deviations give an impression of the day-to-day variability in performance.

The statistical significance of the minimum in the U-shaped function for each monkey was assessed with a post-hoc *t*-test, to determine whether the difference in the distributions of meta and LR trials in the 10 trial blocks at the minimum SOA was significantly different from the mean of the meta/LR biases at the other SOAs. This difference was significant in both monkeys (for monkey L, $t_{18} = 3.92$, $P < 0.001$; for monkey KK, $t_{18} = 2.87$, $P < 0.01$), indicating that the minima in the masking functions do not represent random variations in discrimination behavior. None of the other SOAs are significantly below the mean, using the same statistical procedure. Though it is possible that monkey L's maximum of masking was greater than 120 msec, this value was used in subsequent single-cell recording because it yielded a significant masking.

Receptive fields

The locations of the receptive fields of the neurons ranged from 4° to 8° from the center of the fovea on the side contralateral to the recording site, and near or slightly below the horizontal meridian. These were the locations expected from maps of area 17[42] under the recording wells.

We performed an intensive analysis of a small sample of cells rather than a brief analysis of a larger sample; the present results are based on 30 cells from 4 implanted recording wells in 2 monkeys. In general we recorded from only 1 cell per day, so that the number of penetrations nearly equals the number of cells.

Preliminary receptive-field analysis with hand-controlled stimuli revealed a nearly equal number of oriented and nonoriented fields. The proportion is similar to that often found in foveal and parafoveal striate cortex[12,13,17,25,41], though some estimates of the proportion of oriented fields in alert monkey range as high as 83–87%[26,27,47]. The proportion of oriented cells is somewhat lower in an awake preparation such as ours than in paralyzed and anesthetized preparations[33]; the difference between studies in observed proportions of nonoriented receptive fields can be partly attributed to the length of the stimuli used[26], an explanation consistent with our use of relatively small stimuli for hand-mapping.

Four of the oriented cells (29%) were direction-selective, i.e. responsive only to stimuli moving in one direction. The frequency of direction-selective cells corresponds closely to the 22% found by Poggio et al.[26]. The same authors comment on the extraordinary difficulty of hand-mapping receptive fields in the awake monkey, a result fully corroborated in the present study. Thus the hand maps of receptive fields can serve only as a preliminary guide, and the principal results of this study stem from statistical analysis of machine-collected and averaged responses.

Brightness discrimination

The present results were analyzed differently from those of most other studies of single units because our stimuli were not adjusted to be optimal for a given cell. The move away from 'optimal' stimuli has been begun and commented upon by others[3,26], but is carried further here to the analysis of responses to only two configurations of stimuli, the metacontrast and the large-ring patterns. The use of standardized stimuli enabled us to examine the ensemble response of many neurons operating in parallel on the same stimulus. The non-optimal stimuli also meant that single-cell responses were often weak, however, requiring averaging and statistical analysis to bring out regularities of response patterns. A similar requirement for averaging has been noted in the effect of saccadic suppression on single-cell background activity[14].

The brightness data can be examined for differences between trials on which monkeys were rewarded for responses on the bright side and those on which they encountered a penalty delay for responses on the filtered side. The spike count per trial was first calculated separately for the early and late post-stimulus intervals in the correct trials. The same statistics were then calculated for the incorrect trials, yielding a total of 4 conditions per cell. The distribution of spikes/trial in the early interval for the correct trials was then compared with the corresponding distribution in the incorrect trials; there was no significant difference between these distributions ($t_{18} = 1.44$, $P > 0.1$), showing that the difference in behavior on the correct and incorrect trials was not due to differences in sensitivity caused, for instance, by small differences in fixation or by differences in state of arousal. A similar comparison of distributions of activity in the late interval, however, shows a significant enhancement of activity in

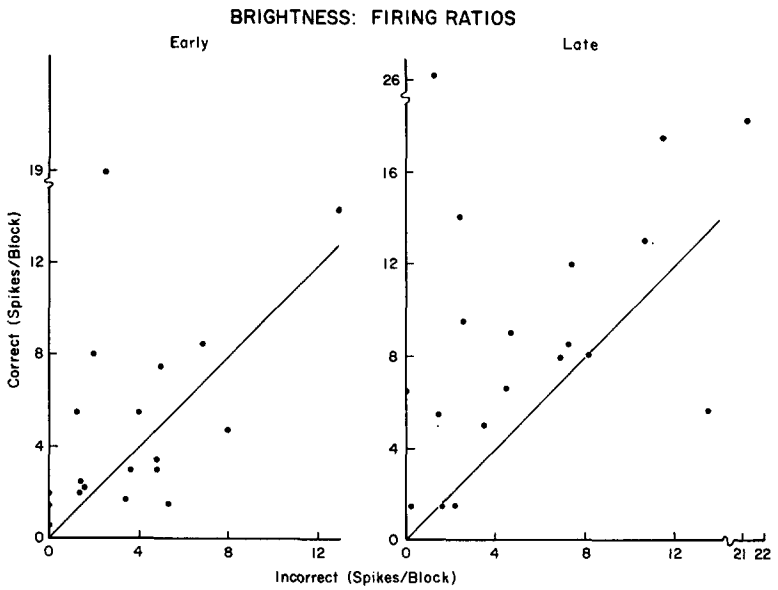

Fig. 4. Firing in the correct brightness trials vs incorrect trials for the early post-stimulus interval (left) and the late interval (right). Because the proportion of correct and incorrect trials is a dependent variable whose value changes from cell to cell, the ordinate and abscissa are normalized to show the amount of activity which would have been observed if each type of trial had been recorded for a 20-trial block in each cell. The resulting activity levels are labeled 'spikes/block' in this and subsequent figures. The diagonal line indicates equal response under the two conditions: dots below the diagonal indicate cells which fire more during incorrect trials, while dots above the diagonal indicate more firing during correct trials.

the correct trials ($t_{18} = 2.36$, $P < 0.04$). Thus the late activity was greater for correct than for incorrect trials, while the early activity did not significantly influence trial outcome. These results are illustrated in Fig. 4.

It is important that the late cellular activity which differentiated correct and incorrect trials occurred before the behavioral response which defined the trial type. The late response may have played a role in organizing the subsequent behavioral choice, but the behavior could not influence the late single-cell activity.

Visual masking

The results in metacontrast trials were similar to those in the brightness trials. Early activity was not significantly different in responses on the simultaneous ('correct') side vs the delayed ('incorrect') side ($t_{18} = 0.49$, N.S.), though activity in the late interval was significantly enhanced in trials on which the monkey chose the simultaneous side ($t_{19} = 3.41$, $P < 0.005$). The enhancement occurred despite the physical identity of light intensity on the simultaneous and delay sides, showing that the late responses corresponded to apparent brightness rather than physical intensity.

Ratios of activity in simultaneous and delay conditions are shown for both early and late intervals in Fig. 5. Fig. 5 also shows that activity in the simultaneous and the delay trials is correlated, especially in the late post-stimulus interval; cells which

respond vigorously under one condition tend to have a large response under the other condition as well.

The slope from the graph's origin to a point in Figs. 4 and 5 defines the ratio of a cell's firing in correct trials to its firing in incorrect trials (C/I ratio). Each cell is represented by one such point in the left graphs of the figures and by another point in the right graphs. Early and late activity can be compared directly by plotting the C/I ratios in the early and the late intervals respectively for each cell (Fig. 6). The shift of the points to the right of the vertical centerline in this figure shows that during the late interval almost all cells fired more in the correct trials than in the incorrect ones, while the more symmetrical distribution about the horizontal centerline shows the lack of bias between correct and incorrect trials in the early interval. This plot allows the early and late data to be compared directly at the price of eliminating information about absolute firing levels of the cells. The bias between early and late activity shows that most cells are carrying information in this task, despite the lack of 'optimal' receptive-field stimuli.

The early burst of firing after presentation of a briefly flashed stimulus is usually considered to encompass most of the stimulus-evoked activity of a cell, and it has been the most intensively studied. Under the conditions of the present experiment, however, there is actually greater spike density in the late interval than in the early interval for most cells. The difference in total firing summed over both simultaneous and delay trials is statistically significant ($t_{20} = 2.36$, $P < 0.04$), and is illustrated by the location of most cells below the diagonal in Fig. 7. The relatively low intensity of response in the early interval may be due to the non-optimal nature of the stimulus for each receptive field, and the relatively strong late response may result from the information content of the stimuli.

Pre-response changes

Several cells showed dramatic changes in activity immediately preceding the behavioral response. These changes were assessed by summing the pre-response

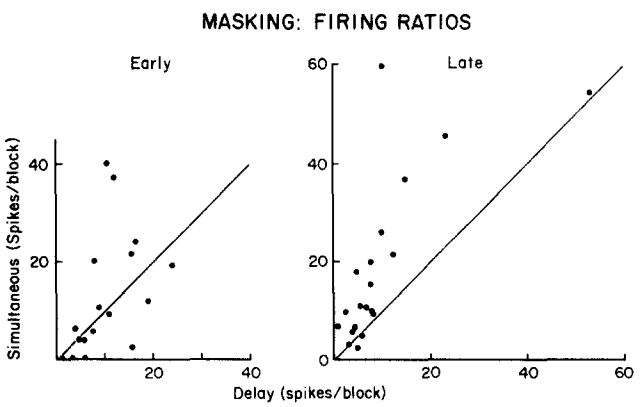

Fig. 5. Firing ratios in simultaneous vs delay masking trials, plotted separately for the early post-stimulus interval (left) and the late interval (right). Display format is similar to that of Fig. 4.

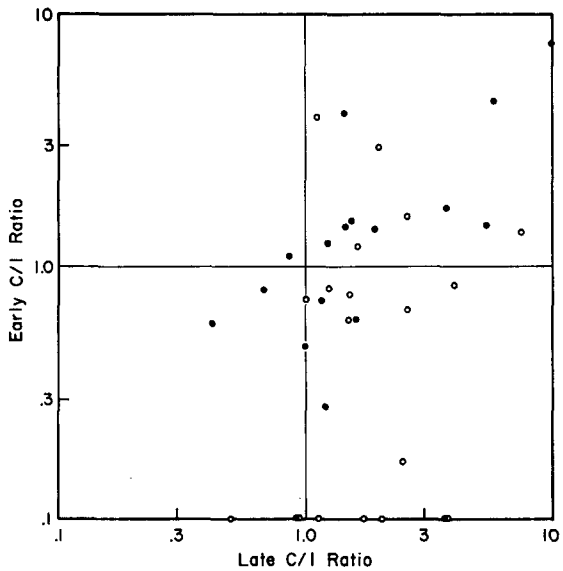

Fig. 6. C/I (correct/incorrect) firing ratios in the early interval vs the late interval for all trials, in log–log coordinates. The vertical centerline indicates equal firing density in correct (or simultaneous) and incorrect (or delay) trials during the late interval, while the horizontal centerline shows equal firing during the early interval. Cells whose points would fall beyond the extremes of the plot are shown at the edge of the diagram. Each cell is indicated by two symbols, a filled circle for brightness trials and an open circle for masking trials. The clustering of points on the right side of the diagram indicates that in the late interval most cells fired more during correct trials than during incorrect trials, while the approximate symmetry of points about the horizontal centerline shows the lack of significant firing bias in the early interval.

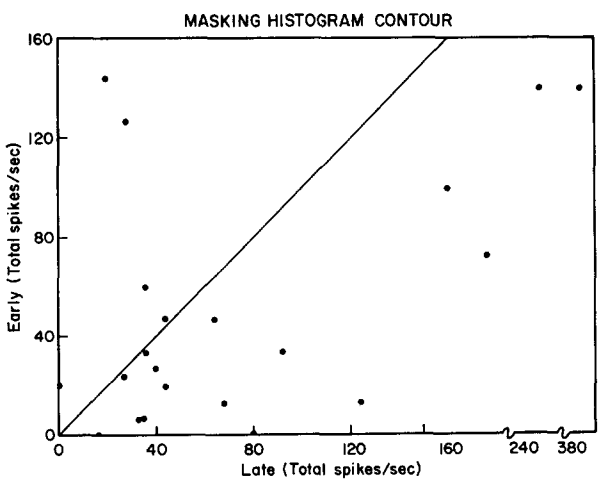

Fig. 7. Comparison of activity in the early post-stimulus interval vs the late interval, after correction for the longer duration of the late interval. The diagonal indicates equal spike density in the two intervals. Most cells have a greater spike density in the late interval (points below the diagonal) despite large differences among cells in intensity of response.

activity in all trials for each cell into a single histogram, and then computing the best-fit linear regression line through the last 250 msec of activity preceding the response. Non-zero slopes significant at $P < 0.05$ were present in 5 (25%) of the cells; 3 (15%) of them were significant at $P < 0.01$ (Fig. 8). Changes were evenly divided between positive slopes (activity increasing just before the response) and negative slopes. The correlation coefficients of the cells did not seem to be grouped into different classes, but showed a continuous and symmetrical distribution.

Changes in activity before the behavioral response thus occurred more frequently than would be expected by chance, though not all cells showed monotonic pre-response changes. The pre-response characteristic was not related to other cell properties such as orientation or C/I ratio.

The total activity of each cell in the last 250 msec before the response was also calculated for each of 4 trial types (masking, simultaneous response; masking, delay response; brightness, correct response; brightness, incorrect response). In metacontrast trials, the difference between total activity in the simultaneous condition and the delay condition was greatly diminished ($t_{19} = 1.70$, N.S.). This reduced differentiation of total activity in simultaneous and delay trials is in contrast with the much greater firing rate in simultaneous trials for the late post-stimulus interval, and shows that the late interval and the pre-response interval do not sample the same data.

A similar result was obtained in the pre-response epoch of the brightness trials,

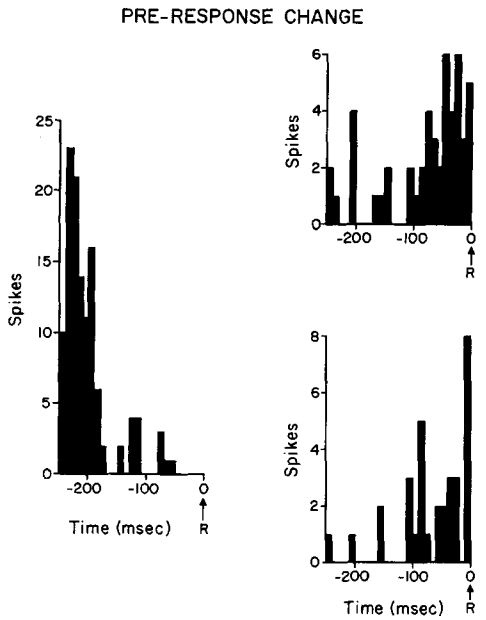

Fig. 8. Pre-response histograms for 3 cells. Histograms are composites of summed activity in all trials, averaged backward from the behavioral response (arrows at 'R'). The cell on the left decreased its activity before the response and was completely inhibited for the 50 msec preceding the response (this characteristic probably had no effect on the response itself, which is already coded in motor cortex by this time). Background firing rate in this unit was 4.4 spikes/sec. The two units on the right show an increase in firing just before the response.

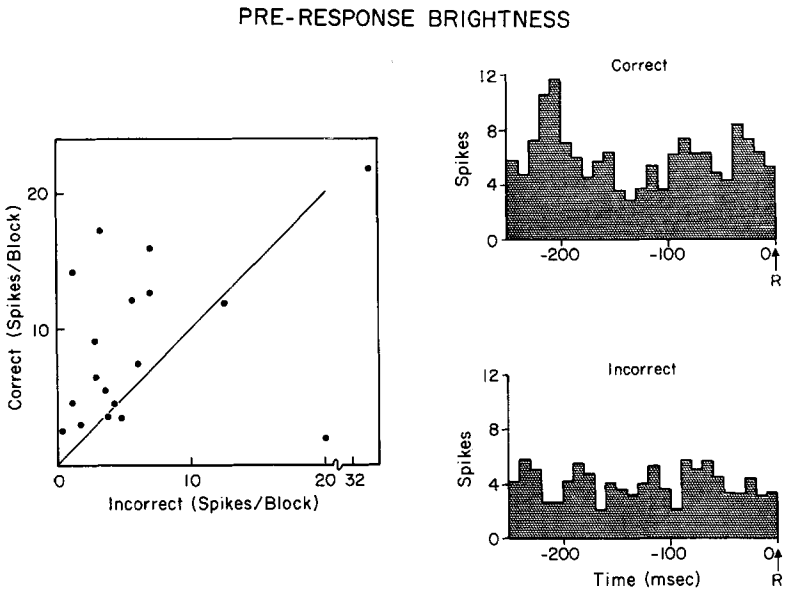

Fig. 9. Left: firing during correct vs incorrect brightness trials for the 250 msec preceding the behavioral response. The relative excess of firing before correct responses is statistically significant. Right: pre-response histograms for correct and incorrect trials.

where normalized activity for correct trials is still significantly greater than the activity for incorrect trials ($t_{18} = 2.18$, $P < 0.05$), though the significance of the difference is reduced in comparison to the results for the late post-stimulus interval. The remaining difference in firing is almost entirely due to redistribution of the activity in the pre-response interval (Fig. 9). Specifically, the peaks of firing at —200 msec and just before the response in the correct trials are absent in the incorrect trials, and a less distinct peak just after —100 msec seems to be present in both conditions. The sharpness of these peaks indicates that they are truly response-synchronized rather than very late stimulus-synchronized features of the pre-response pattern.

Background activity

Background activity epochs long enough to analyze were obtained from 20 cells, one with a 4-sec sample and the others with samples of more than 8 sec. The overall background rate was 1.3 spikes/sec, a figure which compares to the 1.2 spikes/sec obtained by Schiller et al.[33] for their S-type cell group. Their other major group, the CX-type cells, had a rate of 4.9 spikes/sec. It is likely that the enhanced alertness in our monkeys, who were performing a psychophysical eye fixation task during the collection of background activity samples, resulted in a lowering of the background activity in many cells[8]. Background activity in the present experiments often seemed to decrease during the course of a psychophysical trial block. None of the infrequent cells reported by Bridgeman[8], which showed prolonged inhibition with visual attention, were encountered. The distribution of background firing rates showed an exponentially decreasing cell frequency with increasing rate, a pattern which is typical of cell populations in monkey visual cortex[33].

Background rate was positively correlated with spike density: the correlation between early activity and background was 0.798 ($t_{17} = 5.48$, $P < 0.001$, n = 19), while the correlation of late activity with background was 0.750 ($t_{17} = 4.68$, $P < 0.001$, n = 19). These tests compared overall background rate with spike density in correct brightness trials, the condition where the number of trials available for the sample was largest and most consistent from cell to cell. Background rate did not correlate significantly with other properties of the neurons such as oriented vs non-oriented receptive fields, zero vs non-zero pre-response regression slope, or correct vs incorrect brightness trials. Only one cell showed no background activity at all in an 8-sec sample.

DISCUSSION

The general finding that the striate cortex codes more than simply the stimulus attributes of a situation confirms results obtained with implanted gross electrodes[28,31,40], though the finer-grained analysis of the microelectrode yields a different pattern of temporal coding. In the gross electrode studies rhesus monkeys were implanted in striate cortex with bipolar electrodes, and correlates of stimulus, response, and reinforcement were visible in the averaged evoked response on some (though not all) electrodes. In agreement with the present single-unit results, the first components of the gross cortical response were invariant over correctness of response or degree of training. These studies reported no change in late stimulus-locked activity, however, while the single-unit results show significant differences in the amount of late activity as a function of correctness of behavioral choice. Response-synchronized gross evoked activity was also independent of what pattern had been recognized or the correctness of the behavioral response.

Some of these discrepancies may be due to differences in stimuli and design; the gross electrode studies used successive rather than simultaneous discriminations. They also examined the foveal projection area, and fixation, though not tightly controlled, was probably foveal also; the present study used parafoveal stimulation and recording.

An important extension of the earlier work concerns the overlap of stimulus- and response-related events in single cells. The present data reveal that the same cell can participate in early, late and pre-response phases of the cortical response, demonstrating a true multiplexing in single-cell coding, while the gross evoked potential work left open the possibility that spatially adjacent but distinct and specialized subpopulations participated in different functions.

The issue of spatial distribution in the brain of the early and late components of cortical response, as well as the temporal properties of the response, is best developed in the literature on event-related potentials (ERPs) recorded from the scalp, primarily in humans. A general principle emerges from this work that early ERPs following a brief stimulus are restricted to primary sensory areas and reflect only stimulus parameters, while later components are less regular and are modified by task variables such as the information content of the stimuli and the subsequent behavioral pattern[21,29,30]. The later components typically have a wider spatial distribution as well.

Vaughan[43] has shown that the later components of the ERP have a substantially different spatial distribution than the early components; he inferred from scalp recordings that the late components originate not in sensory areas, but in intrinsic cortex. This implies a recoding of information carried in stimulus-evoked early peaks, probably outside area 17, to combine it with endogenous information about the task. The present data show that the recoded information is again represented in the same cells at a later time, now influenced by the eventual behavioral choice in the same trial. The spatial distributions of early and late activity overlap in parafoveal cells of area 17.

The wider spatial distribution of late activity has also been demonstrated in gross potentials recorded from area 17 of the monkey by Gouras and Padmos[18]. In their Fig. 1 several components are visible in the ERP, including an early peak which leaves the baseline about 40 msec after the flashed stimulus and returns to baseline by about 75 msec. As the flashed stimulus is gradually moved away from the receptive field center the early wave disappears while the later waves remain nearly unchanged. This work suggests that even on a more microscopic level within striate cortex the spatial distribution of late activity is wider than that of the early activity which defines the classical receptive field.

Functions of late activity: iterative coding

These results show that one cell can code information in different ways at different times: how does this come about? These cells exist in a cortical milieu which consists of large numbers of similarly organized synaptic structures, each imposing only a few logical layers between cortical input and output[35]. In sensory cortex these probably correspond to cortical columns. Most afferent and efferent fibers from the cortex are not motor or sensory, but are U-shaped fibers densely connecting cortical areas[4]. Evidence from the results presented here combined with the anatomy reviewed above suggests that visual information enters the primary cortex in an early burst of activity, then is carried by corticocortical connections to other areas, perhaps outside the striate cortex, where it is convolved with other types of (endogenous) information, and is then returned to the same striate cortex cells for further processing. The process may be repeated again just before the behavioral response.

Responses of visually excitable neurons in several non-striate areas are consistent with this hypothesis. In inferior temporal cortex of monkey, Gross, Bender and Gerstein[19] found single units with enhanced discharges during performance of a same–different task. The units had post-stimulus latencies typically more than 80 msec, longer than the typical post-stimulus latency of the first burst of activity in striate neurons but earlier than the late integration interval in the present study. These neurons did not differentiate correct from incorrect trials, however, so that they are intermediate between early and late striate activity in their specificity of correlation to task variables. Many units of parietal cortex (area 7) in monkey discharge in the presence of a stimulus which will lead to reward[22], a property which has a logical 'and' relationship with the late activity in the present results. The properties of neurons in both of these studies are consistent with a reciprocal (probably indirect) connection with striate neurons, though further experiments could clarify this idea.

A similar process has been proposed by Eccles[15,16] for the organization of motor responses, where motor cortex, sub-cortical motor structures, and cerebellum engage in repeated reverberations of activity before and during a motor response. This can be interpreted as an iterative process as opposed to a hierarchical one, where the same cells, possibly in different configurations, are involved in several different kinds of processing at different times.

In this interpretation information moves not only from one area to another in an ever more 'central' direction, but also moves in the reciprocal direction. The principle is similar to the well-known examples of efferent control in sensory systems[11,36,38,39], where information is transferred from a subcortical system to a cortical area which in turn modulates the activity of the same subcortical structure.

Implications for mechanisms of metacontrast

The physiological mechanism of metacontrast masking proposed by Bridgeman[9], based on single-cell results in the cat, was a simple decrease in a late peak of the unit response caused by an inhibitory interaction. The present results suggest that a decrease in a late peak, while it accompanies metacontrast masking, is only part of a more complex and widespread mechanism and cannot itself provide an explanation of the effect. The reduction in the late peak observed here in masking trials is probably due to partially endogenous activity which is determined not only by the stimulus but also by other influences usually subsumed under a 'cognitive' label. This might provide a point at which higher-order variables have their influence on the metacontrast effect. Some properties of metacontrast, however, such as its changes with retinal eccentricity, can be accounted for by properties of receptive fields[10]. The physiological results obtained here, where early components of the response were unaffected by masking, exclude such theories as Breitmeyer and Ganz's[5] proposal that metacontrast might be mediated by the interaction of the early components of activity in sustained and transient cells. The basic phenomenon of metacontrast might depend on simple mechanisms such as lateral inhibition, but the many other influences on the late component mean that information from other sources must be superimposed on the basic mechanism.

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REFERENCES

1 Alpern, M., Metacontrast, *J. Opt. Soc. Amer.*, 43 (1953) 648–657.
2 Bagshaw, M. H., Mackworth, N. H. and Pribram, K. H., Method for recording and analyzing visual fixations in the unrestrained monkey, *Perceptual and Motor Skills*, 31 (1970) 219–222.
3 Baizer, J. S., Robinson, D. L. and Dow, B. M., Visual responses of area 18 neurons in awake, behaving monkey, *J. Neurophysiol.*, 40 (1977) 1024–1935.

4 Braitenberg, V., *On the Texture of Brains*, Springer-Verlag, New York, 1977.
5 Breitmeyer, B. G. and Ganz, L., Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing, *Psychiol. Rev.*, 83 (1976) 1–36.
6 Bridgeman, B., A simple system for extracellular microelectrode recording from awake animals, *Electroenceph. clin. Neurophysiol.*, 33 (1972) 116–118.
7 Bridgeman, B., Receptive fields in single cells of monkey visual cortex during visual tracking, *Int. J. Neurosci.*, 6 (1973) 141–152.
8 Bridgeman, B., Background activity in single cells of monkey visual cortex during visual tracking, *Int. J. Neurosci.*, 6 (1973) 153–158.
9 Bridgeman, B., Correlates of metacontrast in single cells of the cat visual system, *Vision Res.*, 15 (1975) 91–99.
10 Bridgeman, B. and Leff, S., Interaction of stimulus size and retinal eccentricity in metacontrast masking, *J. exp. Psychol.*, 5 (1979) 101–109.
11 Brooks, B. and Jung, R., Neuronal physiology of the visual cortex. In R. Jung (Ed.), *Handbook of Sensory Physiology, Vol. VII/3, Central Visual Information B*, Springer-Verlag, Berlin, 1973, pp. 325–440.
12 Dow, B. M. and Gouras, P., Color and spatial specificity of single units in rhesus monkey foveal striate cortex, *J. Neurophysiol.*, 36 (1973) 79–100.
13 Dow, B. M., Functional classes of cells and their laminar distribution in monkey visual cortex, *J. Neurophysiol.*, 37 (1974) 927–946.
14 Duffy, F. and Burchfiel, J., Eye movement-related inhibition of primate visual neurons, *Brain Research*, 89 (1975) 121–132.
15 Eccles, J. C., The dynamic loop hypothesis of movement control. In K. N. Leibovic (Ed.), *Information Processing in the Central Nervous System*, Springer, New York, 1969, pp. 245–269.
16 Eccles, J. C., *The Understanding of the Brain*, McGraw-Hill, New York, 1977.
17 Gouras, P., Opponent-colour cells in different layers of foveal striate cortex, *J. Physiol. (Lond.)*, 238 (1974) 583–602.
18 Gouras, P. and Padmos, P., Identification of cone mechanisms in graded responses of foveal striate cortex, *J. Physiol. (Lond.)*, 238 (1974) 569–581.
19 Gross, C., Bender, D., and Gerstein, G., Activity of inferior temporal neurons in behaving monkeys, *Neuropsychologia*, 17 (1979) 215–229.
20 Grüsser, O.-J., Peterson, A. and Sasowski, R., Neurophysiologische Grundlagen des Metakontrastes, *Pflügers Arch. ges. Physiol.*, 283, R50 (1965).
21 Klinke, R., Fruhstorfer, H. and Finkenzeller, P., Evoked responses as a function of external and stored information, *Electroenceph. clin. Neurophysiol.*, 25 (1968) 119–122.
22 Lynch, J., Mountcastle, V., Talbot, W. and Yin, T., Parietal lobe mechanisms for directed visual attention, *J. Neurophysiol.*, 40 (1977) 362–389.
23 Mackworth, N. H., The wide angle reflection eye camera for visual choice and pupil size, *Percept. Psychophys.*, 3 (1968) 32–34.
24 Noda, H., Freeman, R. and Creutzfeldt, O.-D., Neuronal correlates of eye movements in the visual cortex of the cat, *Science*, 175 (1972) 661–663.
25 Poggio, G. F., Baker, F. H., Mansfield, R. J. W., Sillito, A. and Grigg, P., Spatial and chromatic properties of neurons subserving foveal and parafoveal vision in rhesus monkey, *Brain Research*, 100 (1975) 25–59.
26 Poggio, G. F., Doty, R. W., Jr. and Talbot, W. H., Foveal striate cortex of behaving monkey: single-neuron responses to square-wave gratings during fixation of gaze, *J. Neurophysiol.*, 40 (1977) 1369–1391.
27 Poggio, F. G. and Fischer, B., Binocular interaction and depth sensitivity in striate and prestriate cortex of behaving rhesus monkey, *J. Neurophysiol.*, 40 (1977) 1392–1405.
28 Pribram, K. H., Spinelli, D. N. and Kamback, M., Electrocortical correlates of stimulus response and reinforcement, *Science*, 157 (1967) 94–94.
29 Ritter, W., Simson, R. and Vaughan, H. G., Jr., Association cortex potentials and reaction time in auditory discrimination, *Electroenceph. clin. Neurophysiol.*, 33 (1972) 547–555.
30 Ritter, W., Simson, R., Vaughan, H. G., Jr. and Friedman, D., A brain event related to the making of a sensory discrimination, *Science*, 203 (1979) 1358–1361.
31 Rothblat, L. and Pribram, K. H., Selective attention: input filter or response selection? An electrophysiological analysis, *Brain Research*, 39 (1972) 427–436.

32 Schiller, P. H., Single unit analysis of backward visual masking and metacontrast in the cat lateral geniculate nucleus, *Vision Res.*, 8 (1968) 855–866.
33 Schiller, P. H., Finlay, B. L. and Volman, S. F., Quantitative studies of single-cell properties in monkey striate cortex. I. Spatiotemporal organization of receptive fields, *J. Neurophysiol.*, 39 (1976) 1288–1319.
34 Schiller, P. H. and Smith, M., Monoptic and dichoptic metacontrast, *Percept. Psychophys.*, 3 (1968) 237–239.
35 Shepherd, G. M., *The Synaptic Organization of the Brain*, Oxford University Press, New York, 1974.
36 Singer, W., Temporal aspects of subcortical contrast processing, *Neurosci. Res. Program Bull.*, 15 (1979) 358–369.
37 Spinelli, D. N., Bridgeman, B. and Owens, S., A simple single-unit microelectrode recording system, *Med. biol. Engng*, 8 (1970) 599–602.
38 Spinelli, D. N. and Pribram, K. H., Changes in visual recovery functions produced by temporal lobe stimulation in monkeys, *Electroenceph. clin. Neurophysiol.*, 20 (1966) 44–49.
39 Spinelli, D. N. and Pribram, K. H., Changes in visual recovery function and unit activity produced by frontal cortex stimulation, *Electroenceph. Clin. Neurophysiol.*, 22 (1967) 143–149.
40 Spinelli, D. N. and Pribram, K. H., Neural correlates of stimulus response and reinforcement, *Brain Research*, 17 (1970) 377–385.
41 Spinelli, D. N., Pribram, K. H. and Bridgeman, B., Visual receptive field organization of single units in the visual cortex of monkey, *Int. J. Neurosci.*, 1 (1970) 67–74.
42 Talbot, S. and Marshall, U., Physiological studies on neural mechanisms of visual localization and discrimination, *Amer. J. Ophthalmol.*, 24 (1941) 1255–1264.
43 Vaughan, H. H., Jr., The relationship of brain activity to scalp recordings of event-related potentials. In E. Donchin and D. Lindsley (Eds.), *Average Evoked Potentials*, NASA, Washington, D.C., 1969, pp. 45–94.
44 Vaughan, H. G., Jr. and Silverstein, L., Metacontrast and evoked potentials: a reappraisal, *Science*, 160 (1968) 207–208.
45 Wagman, I. H. and Battersby, W. S., Neural limitations of visual excitability — V. cerebral after-activity evoked by photic stimulation, *Vision Res.*, 4 (1964) 193–208.
46 Weisstein, N., W-shaped and U-shaped functions obtained for monoptic and dichoptic disk-disk masking, *Percept. Psychophysiol.*, 9 (1971) 275–278.
47 Wurtz, R., Visual receptive fields of striate cortex neurons in awake monkeys, *J. Neurophys.*, 32 (1969) 727–742.
48 Wurtz, R., Responses of striate cortex neurons to stimuli during rapid eye movements of the monkey, *J. Neurophysiol.*, 32 (1969) 975–986.
49 Wurtz, R., Comparison of effects of eye movements and stimulus movements on striate cortex neurons of the monkey, *J. Neurophysiol.*, 32 (1969) 987–994.