Dependence of Saccade-Related Activity in the Primate Superior Colliculus on Visual Target Presence

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Edelman, Jay A. and Michael E. Goldberg. Dependence of saccade-related activity in the primate superior colliculus on visual target presence. J Neurophysiol 86: 676-691, 2001. Neurons in the intermediate layers of the superior colliculus respond to visual targets and/or discharge immediately before and during saccades. These visual and motor responses have generally been considered independent, with the visual response dependent on the nature of the stimulus, and the saccade-related activity related to the attributes of the saccade, but not to how the saccade was elicited. In these experiments we asked whether saccade-related discharge in the superior colliculus depended on whether the saccade was directed to a visual target. We recorded extracellular activity of neurons in the intermediate layers of the superior colliculus of three rhesus monkeys during saccades in tasks in which we varied the presence or absence of a visual target and the temporal delays between the appearance and disappearance of a target and saccade initiation. Across our sample of neurons (n = 64), discharge was highest when a saccade was made to a still-present visual target, regardless of whether the target had recently appeared or had been present for several hundred milliseconds. Discharge was intermediate when the target had recently disappeared and lowest when the target had never appeared during that trial. These results are consistent with the hypothesis that saccade-related discharge decreases as the time between the target disappearance and saccade initiation increases. Saccade velocity was also higher for saccades to visual targets, and correlated on a trial-by-trial basis with perisaccadic discharge for many neurons. However, discharge of many neurons was dependent on task but independent of saccade velocity, and across our sample of neurons, saccade velocity was higher for saccades made immediately after target appearance than would be predicted by discharge level. A tighter relationship was found between saccade precision and perisaccadic discharge. These findings suggest that just as the purpose of the saccadic system in primates is to drive the fovea as an immediate response to the appearance of a target as well as after an instructed delay. To help assess the time course of the perisaccadic discharge in neurons in the intermediate layers of the superior colliculus that they termed “visually-triggered movement cells.” These neurons discharged before and during saccades made to light spots but not for movements made across a dark or featureless background. These results were corroborated qualitatively by Mays and Sparks (1980). More recently, Everling et al. (1999) observed that saccades to visual targets (pro-saccades) are accompanied by greater perisaccadic superior colliculus discharge than saccades directed away from a visual target (anti-saccades).

INTRODUCTION

Saccades move the eye rapidly from one position to another so that new objects can be imaged on the fovea. Their primary function is to render visual processing more efficient. Nonetheless, the experimental analysis of saccades has shown that they can be divorced from the visual world, made spontaneously in the dark, to auditory or tactile stimuli (Frens and Van Opstal 1995; Groh and Sparks 1996; Jay and Sparks 1984; Whittington et al. 1981; Zambarbieri et al. 1995), and to the spatial location of recently vanished visual objects (Gnadt et al. 1991; Hikosaka and Wurtz 1983; Smit et al. 1987; White et al. 1994). Such nonvisually guided saccades tend to be slower and less precise than visually guided movements (Gnadt et al. 1991; Smit et al. 1987), suggesting that the presence of the target contributes to its generation in a fundamental way. Despite this, much research into the neural mechanisms underlying the generation of saccadic eye movements has relied on various nonvisually guided saccades, because the separation of stimulus from response when a saccade is made in the dark is an analytic convenience for dissociating “visual” from “motor” activity (Bruce and Goldberg 1985). Little effort has been made to understand the neural underpinnings of the higher performance achieved through visual guidance. One striking example of this discrepancy is that, although the superior colliculus is probably the most thoroughly studied sensorimotor structure in the mammalian brain, with considerable evidence that it provides signals coding saccadic timing and vector (Sparks and Hartwich-Young 1989), there has been little focus on whether the colliculus contributes to visually guided movement per se. Indeed, there is a tacit assumption that perisaccadic signals in the superior colliculus are independent of the reason for a saccade (Goldberg et al. 1991).

There have been some observations that question this assumption. Mohler and Wurtz (1976) described neurons in the intermediate layers of the superior colliculus that they termed “visually-triggered movement cells.” These neurons discharged before and during saccades made to light spots but not for movements made across a dark or featureless background. These results were corroborated qualitatively by Mays and Sparks (1980). More recently, Everling et al. (1999) observed that saccades to visual targets (pro-saccades) are accompanied by greater perisaccadic superior colliculus discharge than saccades directed away from a visual target (anti-saccades).

In the experiments described in this report, we examined how the perisaccadic discharge in neurons in the intermediate layers of the superior colliculus depends on visual target presence. We assessed the robustness of visual target dependence by recording neurons during visually and nonvisually guided saccades made both as an immediate response to the appearance of a target as well as after an instructed delay. To help assess the time course of target dependence, we also recorded neurons during saccades...
made in a task requiring an immediate response to a target flashed so briefly that it was no longer present at movement initiation.

Portions of these results have been reported previously in abstract form (Edelman and Goldberg 1997).

METHODS

Surgical and neurophysiological methods

We recorded neurons in the superior colliculi of three adult rhesus monkeys (Macaca mulatta). In preparation for these experiments, the monkeys were surgically implanted with a recording chamber above the superior colliculus, a head holder that could be coupled to an animal chair to fix the head during recordings, and scleral search coils in each eye (Judge et al. 1980). The head holder and recording cylinder were embedded in an implant made of dental acrylic attached to the skull by surgical-grade titanium screws. General anesthesia was induced using ketamine (10.0 mg/kg), diazepam (1.0 mg/kg), and glycopyrrolate (0.01 mg/kg), and maintained with isoflurane. For two of the monkeys, the recording chamber was implanted several months after the implant, head-holder device, and eye coils. After surgery, animals were given flunixin meglumine as analgesia as needed (2.0 mg/kg). The antibiotic ampicillin was administered every other day for the 2 wk following surgery. Monkeys were allowed to recover for at least 1 wk after surgery before their participation in these experiments. The animal’s fluid intake, weight, and general health status were carefully monitored. All procedures were approved by the Animal Care and Use Committee of the National Eye Institute in compliance with the Public Health Service Guide for the Care and Use of Laboratory Animals.

Monkeys were housed unrestrained, in pairs, in between recording sessions. During recording sessions the monkey sat comfortably in a primate chair. The monkeys’ heads were restrained by attaching a metal post to the implanted head holder and using a metal sleeve to couple the post with another post attached to the chair. Neural activity was monitored using tungsten microelectrodes manipulated by an electromechanical microdrive system. Electrodes were positioned within the recording chamber by being placed in a 23-gauge stainless steel guide tube inserted into a hole of a plastic grid fastened inside the chamber (Crist et al. 1988).

Experimental control and data acquisition

Experiments were controlled and data were collected using the REX (Real Time Experimental) control language (Hays et al. 1982) running on a Hewlett-Packard HP Vectra 486 computer under the QNX operating system. Eye position and neuronal activity were sampled and stored at 1,000 Hz.

Stimulus presentation

Visual stimuli consisted of red light emitting diodes (LEDs) back-projected onto a tangent screen located 57 cm away from the monkey (Crist and Robinson 1989). Position of the projected light spots was controlled using two pairs of mirror galvanometers. The intensity of the LEDs was 1.0–1.3 cd/m². The entire screen was dimly illuminated (0.1 cd/m²) to enhance the monkeys’ alertness and motivation.

Movement field estimation and trial types

After isolating a single neuron the approximate center of its movement field (the loci of endpoints of saccades accompanied by perisaccadic neural discharge) was estimated. To do this, neural activity was monitored while monkeys made 30–40 trials of visually guided delayed saccades to targets whose position was manipulated by a joystick in between trials. Nearly all neurons were recorded using four, and usually five, experimental tasks (Fig. 1). Trials for all five

FIG. 1. Temporal and spatial schematics for the 5 tasks used to elicit saccades. A: spatial and temporal schematic of Step, Visual delay, Memory delay, and Flash tasks. Targets were presented in the center of a neuron’s movement field, and monkeys were rewarded for saccades that landed near the locations of these targets (left). Rectangular bars (right) indicate that a fixation point or target is illuminated. B: spatial and temporal schematic of Anti task. Targets were presented opposite the center of a neuron’s movement field, and monkeys were rewarded for saccades that landed near the center of the movement field. In the Memory delay and Anti tasks, targets were reilluminated at the saccade goal after the saccade landed. See text for details.
region of space centered at the estimated center of the neuron’s movement field. The width of this window was generally set to 30% of the target eccentricity for the Step, Visual delay, and Flash tasks. For a saccade to fall in this window, its amplitude had to be at least 85% (100% − 30%/2) of target eccentricity. For the Memory delay and Anti tasks this window was sometimes set larger since such saccades are known to be less accurate than saccades made to visual targets. Monkeys were required to maintain fixation within this square for 250 ms after the end of the saccade, after which they received a drop of water or juice. Since monkeys were well trained in the different tasks by the time neural recordings began, errors caused by saccades not landing in the reward window occurred very rarely (<2% of trials), except in the Anti task when monkeys occasionally made saccades to the location of the visual target. The size of the windows was not adjusted to require optimal accuracy, but merely to ensure that saccades were not grossly misdirected.

Different trial types were intermixed pseudorandomly. Since monkeys never knew whether an immediate response would be required, and since there was no temporal gap between fixation point disappearance and target appearance, fewer than 1% of trials elicited express saccades (Fischer and Boch 1983). In the Anti paradigm the only clue that the monkey had to make an anti-saccade was the appearance of the target opposite the location to which the animal had been making saccades. Indeed, we use the term “anti-saccade” loosely here, since clearly the monkey did not need to use mental rotation or other high-level cognitive processes to calculate the desired saccadic endpoint; he merely had to remember to make a saccade with an identical vector to all the others in the block of trials. In this sense, this task is similar to the learned saccade task used to distinguish true presaccadic activity from tonic visual activity (Bruce and Goldberg 1985).

Neuron criteria

We recorded from all neurons with saccade-related discharge that we were able to isolate extracellularly in the superior colliculus. To qualify as having saccade-related discharge, neurons had to have a statistically significant increase in discharge rate in the perisaccadic period (20-ms window centered at the beginning of the saccade) in the Step or Visual delay tasks relative to the level of discharge immediately after the transient visual response. Such a criterion excluded neurons with tonic visual activity that persisted up to the time of the saccade but did not increase perisaccadically, such as the quasi-visual cells of Mays and Sparks (1980). Since express saccades were virtually absent from our data, visual and motor bursts were temporally distinct (see Dorris et al. 1997; Edelman and Keller 1996). Across all recording sessions we attempted to sample neurons with saccade-related discharge at various depths throughout what were presumably the intermediate and deep layers of the superior colliculus (Sparks and Hartwich-Young 1989).

Data analysis

SELECTION OF TRIALS FOR COMPARISONS OF NEURAL DISCHARGE. To help ensure that differences in neural activity between different trial types were not due to systematic differences in vector between two types of saccades, we used a graphical user interface program written for MATLAB (MathWorks) to select subsets of our data to be used in comparing neural discharge. For each neuron’s data set, the chosen subset consisted of trials whose saccade endpoints were within a region whose angular extent was at most 15% of the average saccade amplitude. For a given neuron’s data set, the distance between the vector-averaged saccade displacements of one trial type and that of any other trial type was at most 7% of average saccade amplitude across all trial types. For most data sets the endpoints of chosen saccades occupied an even smaller region. For each neuron’s data set, we analyzed data for a particular trial type if at least six trials met the above criteria.

Similar to previous studies (Gnadt et al. 1991; White et al. 1994), we found that conducting these experiments in dim illumination resulted in very little, if any, of a systematic, dysmetric, upward endpoint shift of the memory-guided saccades, in contrast with that observed when memory-guided saccades are performed in complete darkness (Gnadt et al. 1991; White et al. 1994). Reillumination of the target after the end of the saccade in the Memory delay and Anti tasks, as well as keeping the saccade goal constant within a block of trials, may have also contributed to the reduction of this dysmetria.

SPIKE DENSITY TRANSFORM AND ESTIMATE OF PERISACCADIC DISCHARGE. To assist both the graphical comparison and quantitative analysis of the visuomotor burst waveform, the raw spike data were convolved with a Gaussian of fixed standard deviation (5 ms) to yield a spike density trace (Richmond and Optican 1987). This transform yielded a continuous measure of neural activity.

CURVE FITTING OF SLICES THROUGH MOVEMENT FIELDS. The spatial properties of isoradial slices of movement fields (perisaccadic discharge as a function of saccade direction) for a subset of sampled neurons recording during the Step and Memory delay tasks were estimated using a Gaussian curve fitting procedure with three free parameters. The equation for the curve fit is

$$Z(\theta) = M \ast \exp\left[-\frac{1}{2 \ast \sigma^2} \ast (\theta - \theta_0)^2\right]$$

where $Z$ is the predicted neural discharge (spikes/s), $\theta$ is the saccade direction (zero is right and horizontal, deg), and the three free parameters are $M$, magnitude of response at center of movement field (spikes/s); $\theta_0$, direction of center of movement field (deg); and $\sigma$, directional standard deviation, or spread (deg).

Similarly, for a few neurons isodirectional slices of movement fields (discharge as a function of saccade amplitude) were estimated using a log-Gaussian curve fit (Bruce and Goldberg 1985)

$$Z(\rho) = M \ast \exp\left[-\frac{1}{\rho_0} \ast \left[\ln\left(\frac{\rho + \alpha}{\rho_0 + \alpha}\right)\right]^2\right]$$

where $Z$ and $M$ are as defined above, $\rho$ is the saccade radial amplitude (deg), and the two free parameters (in addition to $M$) are $\rho_0$, radial eccentricity of center of movement field (deg); and $\rho_0$, spread of surface along radial dimension [unitless]. The parameter, $\alpha$ [unitless], ensured that the surface would pass through zero amplitude at the origin. This parameter was fixed at 3, a value taken from the result of a surface-fit procedure used to map visual space onto collicular space (Ottes et al. 1986). The curve-fitting procedures were performed using a nonlinear regression algorithm (Edelman and Keller 1998; Marr and Card 1963) run in MATLAB.

MEASURES OF NEURAL SELECTIVITY. To assess the importance of stimulus presence, we calculated a contrast index, the target dependence factor (TDF) for neurons recorded in both the Step and Anti tasks

$$TDF = \frac{Z_s - Z_a}{Z_s + Z_a}$$

where $Z_s$ is the level of perisaccadic discharge for a particular neuron in the Step paradigm and $Z_a$ is the level of discharge in the Anti task. Such an index can vary from 1 to −1. Thus if a neuron discharged only in the Step or only in the Anti task, its TDF would equal 1 or −1, respectively. We used the levels of discharge in the Step and Anti tasks to describe target dependence because, across our sample, discharge was on the average highest in the Step task and lowest in the Anti task.

NORMALIZATION OF SACCADE VELOCITY. To examine how saccade velocity depends on trial type, we normalized for saccade am-

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plumity by computing a “velocity index” for all saccades in our data set. To calculate this index we first calculated the dependence of peak velocity on saccade amplitude (or saccade “main-sequence”) (Bahill et al. 1975) for all Visual delay trials in all of our data sets by regressing peak velocity against saccade amplitude to fit a rising and saturating first-order exponential curve (Becker 1989)

$$V_{\text{peak}} = V_{\text{max}} \times [1 - \exp(-k \times A)]$$

where $V_{\text{peak}}$ and $A$ are the peak saccade velocity and amplitude, and $V_{\text{max}}$ and $k$ are parameters determined by the regression. The velocity index for each saccade, $i$, in our data set was then calculated

$$\text{velocity index} = \frac{V_{\text{peak},i}}{V_{\text{max}}[1 - \exp(-k \times A_i)]}$$

where $V_{\text{peak},i}$ and $A_i$ are the peak velocity and amplitude of each saccade. Therefore a saccade with a velocity faster than that of the main sequence for a given amplitude would have a velocity index greater than one, whereas a saccade with a velocity slower than the main sequence would have a velocity index of less than one.

MULTIVARIATE REGRESSION ANALYSIS. Part of our data analysis required assessing whether discharge depends on target presence (or, more specifically, the duration of target absence prior to saccade initiation, or “blank time”) independent of other saccade characteristics. Such regression analysis is complicated by the correlation between target presence on other saccade characteristics, such as saccade velocity, since conventional regression analysis requires that the independent factors in the regression equation are themselves uncorrelated (Glantz and Slinker 1990). A demonstration that each factor independently contributes to the level of discharge can be made by computing the marginal sum-of-squares for the regression. This procedure determines the significance of a factor in a regression equation after the other factor(s) have been entered into the equation (Glantz and Slinker 1990).

For example, to establish whether some dependent variable, $Z$, depends on a factor, $B$, independent of factor $A$, requires first regressing $Z$ against $A$ (and including a $y$-intercept term)

$$Z = d_a \times A + d_b$$

where the coefficients, $d_a$ and $d_b$ (both scalars), are determined through a linear least-squares error procedure. The mean square statistic of the regression, $MS_{\text{reg}},$ is then computed (Glantz and Slinker 1990).

Next, discharge is regressed against both factors

$$Z = d_a \times A + d_b \times B + d_i$$

Again, a linear least-squares error procedure determines the coefficients. The mean square statistic for this second regression, $MS_{\text{reg},2}$ is then computed. Next the $F$ statistic

$$F = MS_{\text{reg},2}/MS_{\text{reg},1}$$

is computed (Glantz and Slinker 1990). A statistically significant value of $F$ would indicate that the dependent variable, $Z$, is dependent on factor $B$ independent of factor $A$.

SACCade SCATTER. We used saccade scatter as a measure of saccadic precision. For each neuron’s data set we analyzed the endpoint scatter for all saccades that landed in the reward window for each trial type. Scatter ($S$) of trials for each trial type for each neuron’s data set was defined as

$$S = \frac{1}{A} \sqrt{\frac{\sum ((x_i - \bar{x})^2 + (y_i - \bar{y})^2)}{n}}$$

where $x_i, y_i$ are the horizontal and vertical endpoints of each saccade that landed in the reward window for a given trial type, $\bar{x}, \bar{y}$ are the means of the horizontal and vertical endpoints of these saccades, and $n$ is the total number of saccades. We then normalized the scatter by dividing by $A$, the average amplitude of saccades in the data set.

STATISTICAL ANALYSES. Unless otherwise noted, all pairwise (task vs. task) comparisons of neural discharge or saccade metrics were performed using the nonparametric Wilcoxon signed-rank test (Glantz 1992) across the subset of 64 neurons recorded in a given pair of tasks. For correlating one measurement to another, the Pearson product-moment correlation coefficient was computed (Glantz 1992). In all statistical tests, $P$ values $<0.05$ were taken to indicate statistical significance. Unless otherwise noted, perisaccadic discharge for a task was defined as the mean spike density in a 20-ms window centered on saccade onset. Multiple comparisons were corrected using the Bonferroni adjustment, where necessary.

RESULTS

Visual target dependence of saccade-related discharge in the superior colliculus

We recorded from 64 neurons in the superior colliculi of three monkeys. Data were collected from a vast majority of these neurons during performance of all four of the Step, Visual delay, Memory delay, and Anti tasks. Across this sample, neurons had a larger presaccadic burst before saccades to a visual target (Step and Visual delay tasks) than for saccades of equivalent vector not made to a target (Memory delay and Anti tasks). Discharge of neurons representative of our sample are shown in Fig. 2. For a few neurons, target dependence was quite extreme, with considerable discharge in the two target-present tasks and little or no activity before saccades made in the two target-absent paradigms (Fig. 2A). Most neurons had considerable discharge when saccades were not made to a visible target, although this level of discharge was much less than that for saccades to a target (Fig. 2B). Finally, a few neurons had virtually the same amount of discharge in all four tasks (Fig. 2C).

Across our sample of neurons, discharge was greater for saccades made to a visual target regardless of the interval between target onset and the signal to make the saccade as determined by the disappearance of the fixation point. The Step and Anti tasks both required saccades to be made immediately, but peak saccade-related activity (defined as the average spike density in a 20-ms window centered on saccade onset) was much greater across the sample when a target was present in the movement field (Median discharge: Step, 215 spikes/s; Anti, 82 spikes/s; $P < 0.001$; Fig. 3). The Visual delay and Memory delay trials both required saccades to be withheld during an instructed delay, but here also saccade-related activity was much greater across the sample when a target was present (Median discharge: Visual delay, 197 spikes/s; Memory delay, 102 spikes/s; $P < 0.001$; Fig. 3). It is also clear from this figure that, across our sample, neurons exhibited target-dependent perisaccadic activity regardless of their overall discharge level.

It is clear in Fig. 2, B and C, that in the tasks where there was no visual target the length of the burst of discharge did not increase to compensate for their lower peak discharge rates. However, it is possible that for some neurons peak activity is inversely proportional to the duration of the burst, so that the total number of action potentials was invariant. To test this possibility, we recomputed the pairwise comparisons described above, but rather than calculating a measure of peak spike
FIG. 2. Spike rasters and spike density traces for discharge of 3 neurons representative of our sample. For each of the 3 neurons, we show rasters and spike density traces of discharge in the Step, Visual delay, Memory delay, and Anti tasks. For each task, discharge aligned on target onset (represented temporally by dashed line) is shown on the left, and discharge aligned on saccade onset is shown on the right. Horizontal bar represents 50 ms. Vertical bar represents 250 spikes/s. Frame on right for A–C shows the saccade-aligned spike density traces for the 4 tasks superimposed.
density, we instead counted the total number of spikes in an interval of longer duration than the 20 ms used in the calculation of peak discharge. This interval started 30 ms before saccade onset [slightly earlier than the approximate start of the burst (Dorris et al. 1997; Edelman and Keller 1996; Sparks 1978)] and ended 8 ms before the end of saccade [8 ms being the efference delay for SC signals to affect eye position during a saccade (Miyashita and Hikosaka 1996)]. These time limits were set to provide a more liberal estimate of the interval of perisaccadic activity while excluding virtually all of the visual response. Across our sample of neurons, the results of these analyses were virtually identical to those using the 20-ms time window described above.

**Is the degree of a neuron’s target dependence related to its level of visual and delay period activity?**

Most neurons in our sample had a distinct visual on-response as well as a perisaccadic burst. It would seem reasonable that neurons with a large visual response also have a stronger visual target dependence of perisaccadic activity. We assessed the relation between a neuron’s visual response and its perisaccadic target dependence for the 57 neurons in which we obtained at least 6 trials in the Step and Anti tasks. The strength of a neuron’s visual response was defined as the peak spike density occurring within 90 ms of target onset in the Step task (a separate analysis revealed no significant differences in visual response among the Step, Visual delay, and Memory delay tasks). Target dependence was quantified by calculation of a target dependence factor (TDF; see METHODS), which could range from 1 for neurons that discharged in the Step but not the Anti tasks and −1 for those that discharged in the Anti but not Step tasks. Across the sample we found a modest positive correlation between a neuron’s TDF and visual response ($r = 0.44, P < 0.001$, Fig. 4). However, visual target dependence of saccade-related discharge was evident not only for neurons with distinct visual responses to the appearance of the stimulus, such as those whose discharge is shown in Fig. 2, A and B, but also for neurons with little if any visual on-response (Fig. 5). For this neuron we again observed considerable and comparable discharge in the two target-present tasks but very little discharge in the two target-absent tasks. It is evident from Fig. 4 that the modest correlation between a neuron’s perisaccadic target dependence and a neuron’s visual response was mostly due to the weak visual responses of neurons whose perisaccadic discharge was independent of target presence; in contrast, target-dependent neurons had widely varying visual response intensities.

Neurons with relatively high firing rates in the delay period and more truncated perisaccadic bursts, characteristic of the buildup neurons of Munoz and Wurtz (1995), also had target-dependent perisaccadic activity (Fig. 6). Indeed, overall such neurons displayed approximately the same level of target dependence as neurons quiescent during the delay period, as we found little relation ($r = 0.06, P > 0.5$) between TDF and the average level of delay period activity in the two instructed delay tasks (Visual delay and Memory delay). Therefore just as both neurons with and without strong visual responses show perisaccadic target dependence, target dependence is unlikely to distinguish burst from buildup neurons.

**Dependence of perisaccadic activity on immediacy of response**

As described earlier, discharge was greater for saccades to a visual target regardless of the immediacy of response after target appearance. Indeed, we found little evidence of an effect of instructed delay on discharge. For the two tasks in which a saccade was made to a visible target, discharge was slightly higher when a saccade had to be made immediately (Step), than when the saccade was delayed (Visual delay), although this difference was not statistically significant (Median discharge: Step, 220 spikes/s; Visual delay, 208 spikes/s; $P = 0.08$, Fig. 7). When a saccade was not made to a visible target,
discharge was somewhat higher when the task was delayed than when an immediate saccade was required (Median discharge: Memory delay, 102 spikes/s; Anti, 84 spikes/s; \( P < 0.001 \), Fig. 7), although we did not test whether discharge might be lower still in a delayed antisaccade task.

**Dependence of saccade-related discharge on the time between target disappearance and saccade beginning**

Our observation that saccade-related discharge was highest when the saccade was made to a visible target (Step, Visual delay tasks), much lower when a target had disappeared several hundred milliseconds prior to saccade onset (Memory delay), and lower still when a target did not appear in the movement field in that trial (Anti) suggests that discharge is inversely dependent on the time between target disappearance and saccade initiation, an interval we will refer to as the “blank time.”

To test this idea further, while recording from 47 neurons we ran Flash trials (see METHODS) in which a target flash (50 ms) was the cue to make a saccade that, because of latency constraints, was not initiated until well after the target disappeared. Blank time in these trials was generally 150–200 ms, intermediate to that of the Step and Memory delay tasks (Fig. 8A).

For many neurons whose perisaccadic activity depended on the presence of a visual target, discharge in the Flash task was intermediate to that of the Step and Memory delay tasks (Fig. 8B). For this neuron the reduced perisaccadic discharge in the Flash task is not a result of a reduced visual response to the briefly flashed target, since the visual responses are virtually

![Fig. 5. Spike rasters and spike density traces for discharge of a neuron with little visual response that had virtually no perisaccadic activity when a visual target was not present at saccade endpoint. Other conventions as for Fig. 2.](J. A. EDELMAN AND M. E. GOLDBERG)

![Fig. 6. Spike rasters and spike density traces for discharge of a neuron with a sluggish perisaccadic response. Other conventions as for Fig. 2.](J Neurophysiol • VOL 86 • AUGUST 2001 • www.jn.org)
Fig. 10. Dependence of SC activity on target presence.

Dependence of saccade velocity on target presence and saccade latency

It is well-known that memory-guided and anti-saccades are slower than visually guided saccades (Amador et al. 1998; Becker and Fuchs 1969; Bell et al. 2000; Fischer and Weber 1992; Gnadt et al. 1991; Hallett and Adams 1980; Hikosaka and Wurtz 1985; Smit et al. 1987; White et al. 1994). Our finding that perisaccadic discharge is greater for saccades to visible targets than for memory-guided and anti-saccades thus poses the question of whether in monkey, as for a subset of collicular neurons in cat (Berthoz et al. 1986), saccade velocity correlates with discharge in the superior colliculus. For many neurons, we found that peak saccade velocity depended on task in a manner similar to that of saccade-related discharge, although the differences in saccade velocity were generally not as great. This can be seen for the data from one neuron’s data sets depicted in Fig. 11. Just as perisaccadic activity is higher when a saccade is made to a visual target (Fig. 11A), so too is peak velocity higher (Fig. 11B).

To help assess the relationship between saccade velocity, target, and perisaccadic activity across our sample of neurons, we calculated a velocity index for each saccade by comparing its velocity with those of the main sequence at the corresponding saccade amplitude (see METHODS). Saccades faster or slower than those on the main sequence would have velocity indexes of greater or less than one, respectively. We calculated grand averages of both velocity indexes and saccade-related discharge for the (44/64) neural data sets for which we obtained sufficient data (≥ 6 trials) for all five tasks (Fig. 11D). Although the pattern of results for velocity and saccade-related discharge rate was similar, the velocities of Visual delay saccades were somewhat slower than those of Step saccades, and Anti saccades were somewhat faster than Memory delay saccades. As in the case for the neuron of Fig. 11, A–C, percentage differences in saccade velocity across the sample are dwarfed by the percentage differences in perisaccadic discharge rate.

Since the velocity of Visual delay and Memory delay saccades were somewhat slower than would be predicted if saccade velocity were strictly dependent on collicular discharge,
we wondered whether some factor other than discharge might affect their velocity. The obvious candidate for such a factor would be the target-saccade interval, defined as the time between target disappearance and saccade initiation, since, of our five tasks, only the Visual delay and Memory delay tasks involved delayed responses.

We used a linear multivariate regression model to determine what, if any, of the variance of the velocity index could be related to target-saccade interval independent of velocity.

\[
\text{Velocity index} = v_0 + v_1 \times \text{discharge rate} + v_2 \times \text{target-saccade interval} + v_3 \quad (1)
\]

Since we are interested in the effect of collicular output as a whole in affecting saccade velocity, we conducted this analysis by grouping data for the 44 neurons we recorded in all 5 tasks.
Using this grouped data set for each of the five tasks, we first computed the coefficient of determination of the relationship between velocity index and discharge rate (omitting the target-saccade term in the equation above). Most of the variance in the velocity index ($R^2 = 0.75$) was due to variation in discharge rate. However, when we added target-saccade interval we found that a sizable additional component of the variance was now accounted for ($R^2 = 0.99$ for the combination of discharge and latency). That the $R^2$ values are so close to one is also a result of grouping the neural data sets for the analysis, which eliminated both intertrial and interneuronal variability. We found no such significant increments in $R^2$ when we added target-duration or blank time terms to Eq. 1.

Does discharge depend on visual guidance independent of other saccade characteristics?

Although the analysis described above demonstrated that not all of the variance of saccade velocity was related to collicular discharge, it is certainly possible that the target dependence of neuronal discharge was primarily related to saccade velocity rather than to characteristics of our tasks per se. For a few neurons the effect of target presence on discharge was so dramatic (e.g., Fig. 2A) that it is doubtful that these differences depend on velocity. However, for other neurons, such as the neuron whose levels of discharge and velocity are shown in Fig. 11, A and B, a distinct relationship between peak saccade velocity and discharge was observed saccade-by-saccade (Fig. 11C).

To test whether perisaccadic discharge was dependent on target presence independent of saccade velocity, we examined the relationship of perisaccadic discharge to blank time, peak saccade velocity, and target-saccade interval for each of the 44 neurons recorded in all 5 tasks, using multivariate regression analysis. The data sets for each regression included trials from all five tasks. Note that for the antisaccade trials, blank time included at least one intertrial interval, since a target had not appeared in the neuron’s receptive field since a previous trial. For the neurons recorded in all 5 tasks, 26/44 had a statistically significant dependence of perisaccadic discharge on blank time.
independent of saccade velocity and latency. Moreover, for all 26 of these neurons, discharge was negatively correlated with blank time, confirming that discharge decreased with increasing blank time. Perisaccadic discharge was significantly dependent on saccade velocity independent of blank time and latency for 23 neurons, with all correlations positive. In contrast, the dependence of discharge on saccade latency independent of the other 2 factors was much less systematic, with the discharge rate of 10 neurons positively dependent on saccade latency and 4 neurons negatively dependent. These findings suggest that perisaccadic discharge of most neurons is dependent on the length of the time that the target has been absent regardless of other saccade characteristics. Conversely, these results, like the results of the preceding section, suggest that to some extent saccade velocity and perisaccadic discharge are dissociated.

Dependence of saccade precision and accuracy on target presence

Although we did not observe substantial systematic dysmetria for nonvisually guided saccades, saccade precision was also higher, and thus saccade endpoint scatter lower, for saccades made to a visual target. We analyzed precision for trials in the data sets corresponding to the 44 neurons recorded in all 5 tasks. As opposed to the spatially restricted data sets used for analyzing neural discharge, to assess saccade precision we used all rewarded trials (>98% of all trials, except for saccades in the Anti task, which occasionally were directed to the visual target; see Methods). Saccades to visual targets (Visual delay and Step) were the most precise, saccades to briefly appearing targets (Flash) were less precise, saccades to locations at which targets had disappeared 500–700 ms before saccade initiation (Memory delay) were less precise still, and saccades to locations at which no target appeared (Anti) were the least precise (Fig. 11D). This pattern of differences was the inverse of the pattern observed for neural discharge for these 44 neurons, suggesting that the relation between superior colliculus discharge and saccade precision is even tighter than that between discharge and peak velocity.

Size and location of movement fields of visually guided and memory-guided saccades

Although we have demonstrated that discharge for a particular saccade vector is greater for saccades to a visual target, it is possible that nonvisually guided saccades compensate for this difference by having larger movement fields. It is also possible that the center of the movement fields for Step and

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**FIG. 11.** A–C: the relationship between trial type, neural discharge, and saccade velocity for a single neuron. To construct this plot, for this neuron’s data set we selected Step, Visual delay, Memory delay, and Anti trials such that the mean saccade amplitude for each task’s trials was within 3% of the average of the means. A: mean spike density for the Step, Visual delay, Memory delay, and Anti tasks. Spike density traces are aligned on saccade onset, denoted by zero on the x-axis. B: average radial velocity profiles for saccades the 4 paradigms. Traces are aligned on saccade onset, denoted by a zero on the x-axis. C: plot of peak saccade velocity vs. perisaccadic discharge for the 4 tasks. D: summary for perisaccadic discharge, velocity, and scatter for neurons for which we obtained data from all 5 tasks (n = 44). For comparison, data are arranged in order of average magnitude of discharge from Step, on the left, to Anti, on the right.
Memory delay trials are different (see Stanford and Sparks 1994). To see whether the presence of a visual target affects movement field center or extent, we compared the activity in the Memory delay and Step tasks at multiple target positions within the movement field. For 12 neurons we varied target direction across 5 positions with the central position at the estimated center of the neuron’s movement field, keeping target eccentricity constant. Target directions were spaced by 22.5°. For five neurons we varied target eccentricity across five positions that spanned the movement field while keeping target direction constant (for 1 neuron we varied both target direction and eccentricity in separate blocks of trials). We fit the relationship of response to saccade direction with Gaussian curves, using three free parameters: height (neuronal response), center (movement field location), and width (movement field extent).

We studied movement fields of neurons with minimal (Fig. 12A), moderate (Fig. 12B), and strong (Fig. 12C) perisaccadic responses in the Memory delay task. We found that Gaussian height was much greater in the Step task while the Gaussian center was unchanged, i.e., the movement field centers were in similar locations in the Step and Memory tasks. We found a small yet statistically significant ($P = 0.047$) difference in Gaussian width (movement field extent; Table 1A). To determine whether this difference in movement field width could compensate for the dramatically weaker burst in the Memory delay task, we compared the area under the Gaussians in both conditions (Table 1A). The increase in movement field size was nowhere near large enough to compensate.

We performed a similar analysis for the five isoradial comparisons, except that we used a log-Gaussian curve to fit the relationship between discharge and saccade amplitude (see METHODS). Log-Gaussian curve fits for one of the neurons in which we varied target eccentricity while keeping direction constant also were of similar shape; again, the Memory delay movement field appeared to be a scaled-down version of the Step movement field (Fig. 12D). Statistically significant fits were obtained for both trial types for data from four of five neurons. Again, the height parameter was much higher for Step trials than for Memory delay trials, while the center and width parameters were more similar (Table 1B).

Dependence of activity during the instructed delay period on target presence

Given that discharge in the superior colliculus around the time of the saccade was dependent on target presence, could discharge in our two instructed delay conditions (Visual delay and Memory delay) occurring after the visual response but before the saccade-related burst also depend on target presence? And could such a delay period dependence be predictive of perisaccadic dependence? Across our sample, discharge in this delay period (defined as the 300-ms interval ending 100 ms prior to saccade onset) was greater when a saccade was made to visible target (Visual delay task) than when a saccade was made to the remembered location of a target (Memory delay task) (Visual delay, 23.6 spikes/s; Memory delay, 14.9 spikes/s; $P < 0.001$). To assess whether target dependence in the delay period was predictive of perisaccadic target dependence, we compared for each neuron the contrast index assessing the relative strengths of activity in the Visual delay and Memory delay tasks in the delay period with the contrast index assessing their relative strengths in the perisaccadic period. The contrast indexes were calculated for each period using the formula $(Z_{VD} - Z_{MD})/(Z_{VD} + Z_{MD})$, where $Z$ is the level of discharge for the appropriate period (1.0: only Visual delay discharge; 0.0: equal discharge; −1.0: only Memory delay discharge).
discharge). For the 16/64 neurons that had delay period discharge over 30 spikes/s in either the Visual delay or Memory delay task, the measures of target dependence in these two epochs were highly correlated ($r = 0.90$, $P < 0.001$, Fig. 13). Therefore, although, as stated previously, the average level of delay period activity (across the Visual delay and Memory delay tasks) was not predictive of perisaccadic target dependence, the target dependence of delay period activity was highly predictive of the target dependence of perisaccadic activity.

**DISCUSSION**

These experiments demonstrate that the intensity of the perisaccadic burst of neurons in the intermediate layers of the superior colliculus is dependent on the presence of a visual target. Neurons discharge more for saccades to visual targets regardless of a neuron’s visual response intensity, level of delay period activity, and overall level of perisaccadic activity. Discharge is greater for saccades to visual targets regardless of whether the saccades are made immediately on target presentation, or only after an instructed delay. Finally, target-enhanced discharge is not all-or-nothing, but tends to decrease over the course of several hundred milliseconds after target disappearance, a continuum mirrored by changes in saccade endpoint scatter, and, to a lesser extent, saccade velocity (see Becker and Fuchs 1989; Gnadt et al. 1991; White et al. 1994).

These findings suggest that visual target presence has an influence on saccade-related activity in the superior colliculus second only to saccade vector.

**TABLE 1. Curve fits of saccade-related discharge**

<table>
<thead>
<tr>
<th></th>
<th>Step</th>
<th>Memory Delay</th>
<th>Wilcoxon Test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. Discharge versus direction</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response magnitude (M), spikes/s</td>
<td>358</td>
<td>156</td>
<td>$P &lt; 0.001$</td>
</tr>
<tr>
<td>Directional center ($\theta_c$), deg</td>
<td>1.55</td>
<td>1.46</td>
<td>$P &gt; 0.5$</td>
</tr>
<tr>
<td>Directional SD ($\theta_s$), deg</td>
<td>23.8</td>
<td>26.8</td>
<td>$P = 0.047$</td>
</tr>
<tr>
<td>Area of movement field slice, deg $\times$ spikes/s</td>
<td>21,843</td>
<td>12,394</td>
<td>$P &lt; 0.001$</td>
</tr>
<tr>
<td>Coefficient of determination ($R^2$)</td>
<td>0.76</td>
<td>0.53</td>
<td>$P &lt; 0.001$</td>
</tr>
<tr>
<td><strong>B. Discharge versus amplitude</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response magnitude (M), spikes/s</td>
<td>231</td>
<td>107</td>
<td></td>
</tr>
<tr>
<td>Radial center ($r_c$), deg</td>
<td>12.8</td>
<td>13.6</td>
<td></td>
</tr>
<tr>
<td>Radial spread ($r_s$) [dimensionless]</td>
<td>0.60</td>
<td>0.45</td>
<td></td>
</tr>
<tr>
<td>Coefficient of determination ($R^2$)</td>
<td>0.46</td>
<td>0.41</td>
<td></td>
</tr>
</tbody>
</table>

Number of neurons in A is 12, and in B is 4. Values in left 2 columns are averages across all neurons. Values for the directional center were normalized in A for every neuron’s data by subtracting the direction of the central target. Upward displacements were defined as positive and downward displacements as negative. A positive value in the 3rd column indicates a greater value for the Step paradigm. Values in bold in the left 2 rows indicate the greater value. Values in the far right column are $P$ values resulting from a Wilcoxon signed-rank test comparing values for the Step and Memory delay paradigms. Numbers in this column are bold if $P < 0.05$. Conventions are identical for B, although we did not perform statistical tests due to the small sample size.

Factors other than visual target presence had a much weaker influence on saccade-related activity. Our data suggest that the time between the appearance of the target and the initiation of the saccade has a much smaller effect on saccade-related discharge. We also did not observe an effect on discharge by visual target duration, although since we did not systematically vary target duration in any of our conditions, we cannot rule out that discharge and duration are independent, particularly for very short durations (see Aitsebaomo and Bedell 1992; Hill et al. 1997; Sommer 1994). Furthermore, although we found that peak saccade velocity is also dependent on task, these differences in velocity only partially accounted for differences in discharge. Finally, as mentioned in METHODS, our trained monkeys made few errors in any of the five tasks, suggesting that the differences in discharge we observed were not due to differences in the ability to perform these tasks.

**Visual target-dependent discharge in the superior colliculus**

Mohler and Wurtz (1976) first made the observation that certain neurons in the intermediate layer of the superior colliculus, which they termed visually triggered movement cells, have perisaccadic bursts for visually guided saccades but not for spontaneous saccades made in darkness or across a featureless background. Mays and Sparks (1980) extended these findings, using a double saccade task to show qualitatively that a small fraction of superior colliculus neurons failed to discharge for purposive movements when no target appeared in the neuron’s movement field, including neurons that had little or no visual response (see also Sparks and Porter 1983). A much more recent study has shown that superior colliculus neurons discharge more for saccades to a visual target than for spatially equivalent antisaccades (Everling et al. 1999).
Our results suggest that these previous findings are specific instances of a general phenomenon of visual dependency of saccade-related discharge in the superior colliculus. Given that discharge for memory-guided saccades is only modestly stronger than that for anti-saccades, it is likely that the reduced activity for anti-saccades is due primarily to the absence of a visual saccade target rather than to the requirement to make a saccade away from a sudden visual onset, although inhibition from the contralateral colliculus may play a small role (Everling et al. 1998, 1999). The visually triggered movement neurons of Mohler and Wurtz (1976) and Mays and Sparks (1980) appear simply to be located at one extreme of a spectrum of target dependence rather than being a distinct class of neurons. Indeed, the term “visually triggered” to describe these neurons may be misleading, as we found that perisaccadic discharge was reduced for saccades “triggered” by a briefly flashed target.

Neural mechanisms underlying target dependence

For perisaccadic discharge in the superior colliculus to be influenced by the presence of a visual target, information relating to the visual target must be present in the superior colliculus at the time of the saccade. Recent work has shown that the phasic visual response present 40–70 ms after target onset for many superior colliculus neurons appears to serve as the perisaccadic burst for express saccades (Dorris et al. 1997; Edelman and Keller 1996). In such a case, the mechanism for target presence affecting perisaccadic discharge is straightforward. But how does target presence affect perisaccadic discharge if the phasic component of the visual response is long gone?

One possibility is that visual target dependence may result from visual input to the superior colliculus from visual and parietal cortex. Neurons in the lateral intraparietal area (LIP) with visual and perisaccadic responses project to the intermediate layers of the superior colliculus (Paré and Wurtz 1997) and have higher perisaccadic discharge for saccades made to a visual target than for saccades to blank space (Gottlieb and Goldberg 1999; Gottlieb et al. 1998; Nakamura and Colby 2000; Paré and Wurtz 1997). Other extrastriate visual areas have been shown to be at least modestly active prior to saccadic eye movements in monkey (Fischer and Boch 1981b; Moore et al. 1998). Tonic visual input from these areas to the superior colliculus may be subthreshold for initiating bursts of activity (or even individual action potentials) prior to a command to make a saccade. Such a command, originating from frontal cortex or within the superior colliculus itself, may inhibit fixation-related activity (Munoz and Wurtz 1993) and thereby disinhibit collicular saccade neurons. At such a time low-level collicular discharge resulting from tonic visual input may breach threshold and cause a saccade-triggering burst of discharge. Neurons in the superior colliculus with perisaccadic discharge that depends on target presence may receive input mainly from visual and parietal cortex, so that in the absence of a visual target there is insufficient input to lead to the generation of a burst, even if the neurons are disinhibited.

It is less clear whether the frontal eye field projection to the superior colliculus affects collicular visual dependence. The projection from the frontal eye field to the superior colliculus is less visual than that from LIP to the colliculus (Segraves and Goldberg 1987; Sommer and Wurtz 2000). However, an analysis similar to ours has not been performed for frontal eye field neurons, and in this area the presaccadic burst could also be visually dependent (Everling and Munoz 2000). The supplementary eye field, another frontal cortical area that projects to the superior colliculus (Leichnetz 1981), actually discharges more for antisaccades than saccades to a visual target (Schlag-Rey et al. 1997). Neurons in the superior colliculus whose discharge is not dependent on the presence of a visual target may rely much more on inputs from frontal cortex. Indeed, the continuum of perisaccadic target dependence we observed might be explained by a continuum in relative strengths of visuoparietal and frontal inputs to superior colliculus neurons. Paradoxically, however, reversible lesions of the lateral intraparietal area have less effect on visually guided saccades than they do on memory-guided saccades (Li et al. 1999).

Relation between superior colliculus discharge and saccade metrics

Our results also demonstrate for the first time in monkey that the activity of many superior colliculus neurons positively correlates on a trial-by-trial basis with saccade velocity. As such, this study complements earlier work that provided evidence that the level of superior colliculus activity influences saccadic velocity in cat (Berthoz et al. 1986) and that the parameters of electrical stimulation can influence saccade velocity (Stanford et al. 1996). However, saccade velocity can only partially account for the differences we observed in perisaccadic discharge among the tasks. A majority of neurons for which we collected data in all five tasks (26/44) discharged less the longer the target had been absent prior to saccade initiation, regardless of saccade velocity and latency. Indeed, given the known anatomy and physiology underlying saccadic generation, our findings may be a consequence of the prolonged absence of a visual target decreasing collicular activity, which in turn contributes to a decrease in saccade velocity. Moreover, discharge does not appear to be the sole determinant of saccade velocity, as our analysis revealed that the requirement to delay a saccade after target onset contributed to a decrease in saccade velocity independent of saccade-related discharge. This suggests the possibility that eye movement signals from the frontal eye fields or other cortical pathways that bypass the superior colliculus to reach the cerebellum and/or brain stem may influence saccade velocity independent of collicular discharge.

In contrast, we found that the relative amount of saccade scatter observed in the five paradigms was predicted well by the relative level of discharge in the tasks. As mentioned previously, we did not observe systematic inaccuracy for the nonvisually guided saccades. Increased perisaccadic activity in the superior colliculus may result in a higher signal-to-noise ratio for visually guided saccades, thereby increasing their endpoint precision. The tighter relationship between collicular discharge and saccade precision than between discharge and saccade velocity suggests that the colliculus may play a more critical role in determining saccade vector than in determining saccade velocity.
Movement fields of visually and nonvisually guided saccades

Our results suggest that the overall magnitude of the neuron’s response depends on whether the saccade is made to a visible target, while the shape and location of its movement field depend much less on target presence. At first glance, this may seem to contradict the results of Stanford and Sparks (1994), who found that the movement fields of memory-guided saccades were shifted upward relative to those of visually guided saccades. However, they also found an upward asymmetry of memory-guided saccades that matched this movement field shift, in effect dissociating saccade vector from target. We did not find such an upward shift, perhaps because the monkeys were informed of the accuracy of their saccades by a reillumination of the target, or because our experiments were not performed in total darkness. One can interpret their data to suggest that, regardless of task, movement neurons in the superior colliculus code the location of the saccade target, not saccade vector, as if the colliculus were coding a saccadic command in visual coordinates. Other recent work dissociating saccade vector from target location has demonstrated similar results (Edelman and Keller 1998; Frens and Van Opstal 1997; Keller et al. 1996). Since in our tasks memory-guided saccades were generally not dysmetric, target position and saccade vector were (on the average) identical. Therefore we would not expect memory-guided movement fields to shift upward, and, as this is the case, our results are consistent with the idea that the superior colliculus codes for visual target. Indeed, they demonstrate that not only does the visual target determine the locus of perisaccadic discharge (except, of course, for antisaccades), but that it also strongly influences the intensity of perisaccadic discharge.

Influence of vision on the role of the superior colliculus in movement generation

The superior colliculus and its evolutionary ancestor and homologue, the optic tectum, have long been thought to participate in the generation of rapid movements of eye, head, and body both toward and away from peripheral sensory stimuli (Huerta and Harting 1984). The evolution of the fovea and a sophisticated cortical visual system has placed a new demand on the superior colliculus: the facilitation of rapid, precise visual exploration using eye movements. The purpose of this exploration is to bring peripheral visual targets to the fovea, regardless of whether they have appeared suddenly or have long been present in the visual scene. The dependence of superior colliculus activity on target presence provides evidence for a sensorimotor mechanism that participates selectively in the generation of all visually guided saccades.

Our data agree with all previous studies of the intermediate layers of the superior colliculus in showing that there is a baseline of activity in the superior colliculus that precedes every saccade, regardless of its genesis. This baseline activity drives the eyes to a rough approximation of desired location in space, with sufficient precision to satisfy a nonvisual purpose such as the quick phase of vestibular nystagmus or a cognitively driven saccade in total darkness. However, the raison d’etre of the saccadic system is much more likely to be moving a stimulus to the fovea than it is merely driving the eyes to a visually useless position. When the goal of a saccade is to place a small visual target on the fovea, greater motor precision is necessary. We suggest that increased activity in the colliculus preceding saccades to visible targets serves to make saccades faster and more precise. As such, it represents a subcortical reflection of the evolution of the fovea and the cortical visual system. These results also suggest that any study of the saccadic system that relies exclusively on nonvisually guided saccades may be incomplete.

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REFERENCES


