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RESEARCH ARTICLE

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Expression of a re-centering bias in saccade regulation by superior colliculus neurons

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Abstract In previous studies of saccadic eye movement reaction time, the manipulation of initial eye position revealed a behavioral bias that facilitates the initiation of movements towards the central orbital position. An interesting hypothesis for this re-centering bias suggests that it reflects a visuo-motor optimizing strategy, rather than peripheral muscular constraints. Given that the range of positions that the eyes can take in the orbits delimits the extent of visual exploration by head-fixed subjects, keeping the eyes centered in the orbits may indeed permit flexible orienting responses to engaging stimuli. To investigate the influence of initial eye position on central processes such as saccade selection and initiation, we examined the activity of saccade-related neurons in the primate superior colliculus (SC). Using a simple reaction time paradigm wherein an initially fixated visual stimulus varying in position was extinguished 200 ms before the presentation of a saccadic target, we studied the relationship between initial eye position and neuronal activation in advance of saccade initiation. We found that the magnitude of the early activity of SC neurons, especially during the immediate pre-target period that followed the fixation stimulus disappearance, was correlated with changes in initial eye position. For the great majority of neurons, the pre-target activity increased with changes in initial eye position in the direction opposite to their movement fields, and it was also strongly correlated with the concomitant reduction in reaction time of centripetal saccades directed within their movement fields. Taking into account the correlation with saccadic reaction time, the relationship between neuronal activity and initial eye position remained significant. These results suggest that eye-position-dependent changes in the excitability of SC neurons could represent the neural substrate underlying a re-centering bias in saccade regulation. More generally, the low frequency SC pre-target activity could use eccentric eye position signals to regulate both when and which saccades are produced by promoting the emergence of a high frequency burst of activity that can act as a saccadic command. However, only saccades initiated within ~200 ms of target presentation were associated with SC pre-target activity. This eye-dependent pre-target activation mechanism therefore appears to be restricted to the initiation of saccades with relatively short reaction times, which specifically require the integrity of the SC.

Keywords Saccadic eye movement · Saccade initiation · Saccade selection · Reaction time · Express saccade · Superior colliculus · Motor preparation · Fixation · Eye position · Monkey

Introduction

Behavioral studies in head-restrained subjects (humans: Fuller 1996; Zambarbieri et al. 1995; monkeys: Albano and Wurtz 1982; Paré and Munoz 1996a) have shown that the initiation of saccadic eye movements is systematically modified by the starting position of the eyes in the orbits: movements made from eccentric initial eye positions and towards central positions have a shorter reaction time (RT) than those made to further eccentric positions. The implication of this re-centering bias for eye–head coordination was examined by Fuller (1996), who suggested that it helps to alter the relative timing of the initiation of eye and head movements, given the existing "orbital reserve", i.e. the extent of eye movement possible either by counter-rotation at the beginning of a head movement, or by per-rotation at the end of the gaze shift. With the visual field extending beyond the range that can be explored with saccades, this strategy can also help to minimize excursions away from the central position, preventing the eyes from reaching their boundaries even when the head is restrained. Unless this re-centering bias is caused by a peripheral muscular phenomenon,

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the neural mechanisms implicated in both the selection and the initiation of saccades may therefore be influenced by eye position. In this paper, we investigate this hypothesis by recording from saccade-related neurons in the intermediate layers of the primate superior colliculus (SC), a structure critical for both the initiation and the execution of saccades.

The intermediate layers of the SC contain an ensemble of neurons that display a saccade-related pattern of activity: a burst of action potentials time-locked to saccades of specific metrics that define a neuron's movement field (Sparks et al. 1976; Wurtz and Goldberg 1971). Saccade-related neurons are topographically organized and, within this neural map, the metrics of saccades are encoded in relative coordinates, i.e. neurons discharge for a particular range of saccade vectors irrespective of initial eye position (Schiller and Koerner 1971; Wurtz and Goldberg 1972). A recent study by van Opstal and colleagues (1995), however, showed that eye position does influence the magnitude of the saccade-related burst of activity of several SC neurons, thereby revealing their access to eye position signals. The same neurons can additionally display low frequency activity well in advance of saccade onset (Glimcher and Sparks 1992; Mohler and Wurtz 1976; Munoz and Wurtz 1995; Sparks 1978), and it was recently demonstrated that the level of this early activation could predict RT (Dorris et al. 1997). The hypothesis that the SC plays a role in saccade initiation was further supported by results obtained in microstimulation (Paré et al. 1994; Robinson 1972; Stanford et al. 1996) and pharmacological experiments (Aizawa and Wurtz 1998; Hikosaka and Wurtz 1985, 1986; Quaia et al. 1998; Schiller et al. 1987; Sparks et al. 1990). The question arises as to whether eye position can exert its effect on RT through the early activation of SC neurons?

We studied the activity of SC neurons using a simple reaction time paradigm wherein the initially fixated visual stimulus varied in position. The extinction of this fixation stimulus 200 ms before the presentation of the saccadic target provided a warning signal sufficient for the expression of 'pre-target' activity in these neurons. We examined the relation linking this neuronal activation to both initial eye position and RT to determine whether it is influenced by eye position and whether this influence is consistent with the known relationship between RT and eye position. We found that the SC pre-target activation could indeed contribute to the facilitation of movements towards the orbital center, consistent with the hypothesis that the central processes regulating the selection and the initiation of saccades use eye position signals.

Some results have been reported in abstract form (Paré and Munoz 1996b).

Materials and methods

Animal preparation and experimental procedures

Three male rhesus monkeys (*Macaca mulatta*, 5–10 kg) were prepared for behavioral training along with eye position and extracellular single-neuron recording in a single surgical procedure carried out under general anesthesia and aseptic conditions as described previously (Dorris et al. 1997; Munoz and Istvan 1998; Paré and Munoz 1996a). Animals received both antibiotics and analgesic medications during the post-surgery recovery period that lasted about 2 weeks. Animal health status was monitored closely and under supervision of the university veterinarian. All animal care and experimental procedures were approved by Queen's University Animal Care Committee and were in accordance with the Canadian Council on Animal Care policy on use of laboratory animals.

Two stereotaxic craniotomies were made to access both SC with microelectrodes. Stainless steel recording cylinders were positioned over each craniotomy: one centered on the midline and tilted 38° posterior of vertical and the other centered on the interaural axis and tilted 25° lateral. A PC-compatible computer running a UNIX-based real-time data acquisition system (REX) (Hays et al. 1982) controlled behavioral paradigms, visual displays, and reward delivery. Eye movements were monitored by the magnetic search coil technique using phase angle detection (Collewijn 1977), which permitted the absolute calibration of the horizontal eye position signals. Neuronal recordings were made with tungsten microelectrodes that were inserted into the brain via guide tubes positioned in the cylinders with the aid of a grid system (Crist et al. 1988). Action potentials of single neurons that met distinct amplitude and time constraints were isolated with the use of a window discriminator. The resulting output pulses were digitized at 1 kHz, whereas horizontal and vertical eye position signals were digitized at 500 Hz.

During the experiments, the monkeys were seated in a primate chair with the head restrained and facing an opaque tangent screen positioned 86 cm away. Each behavioral task was initiated by the appearance on the screen of a light spot, referred to as the fixation point, followed after a fixation period by a second light spot serving as a saccade target. The light spots (light-emitting diodes, 2.0 cd/m^2) were back-projected onto the tangent screen and their positions controlled by an x–y mirror galvanometer positioned 86 cm from the screen. This arrangement ensured that any angular deviations in the light source equaled the angular deviations in the cyclopean view of the monkey, and therefore fully corrected for any tangent errors. Except for the light spots, the room was kept dark during the experimental trials. To prevent dark-adaptation, the screen was illuminated with diffuse white light during the inter-trial intervals.

Behavioral paradigms

The activity of SC neurons was studied while the animal performed a warned, simple RT paradigm commonly known as the gap saccade task (Fig. 1A) (Saslow 1967). Once the fixation point appeared at the center of the screen, the monkey had to look at it and maintain visual fixation for 500–800 ms. The fixation point was then extinguished, and the monkey had to maintain steady non-visual fixation during a 200-ms gap interval before the saccade target appeared. The gap period was kept constant, allowing it to act as a warning interval. The monkey was required to make a saccade to the target within 500 ms of its presentation, and then maintain fixation for 300–500 ms before a liquid reward was given.

We ascertained the general properties of the neuronal discharge with respect to saccades within a few trials and then varied systematically (in steps of 1°) the position of the saccade target to determine the movement field of saccade-related neurons (Wurtz and Goldberg 1971; Sparks et al. 1976; Munoz and Wurtz 1995). This was accomplished by graphically displaying online rasters and histograms of the spike occurrences aligned on the onset of the saccades made to each target position. After the center of the movement field (the saccade vector for which neurons discharged optimally) was well defined, we introduced the *orbital* gap saccade task (Fig. 1B). In this task, the initial position of eye fixation was varied randomly between three different positions, each spatially separated by ~15° and distributed along the same axis as the saccade targets: central (straight-ahead), contralateral or ipsilateral

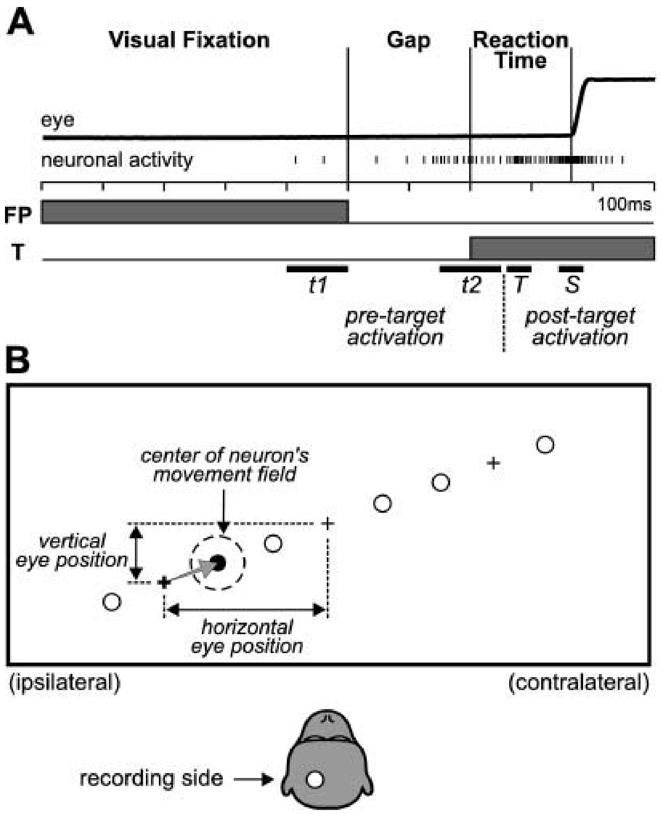


Fig. 1 **A** Gap saccade task: presentation of the visual stimuli and eye position trace as a function of time, along with the corresponding neuronal activity from one SC saccade-related neuron. The duration of the visual fixation interval was randomized between 500 and 800 ms, whereas the gap was kept constant at 200 ms. Reaction time is the time that has elapsed between target onset and saccade initiation. The pre-target neuronal activity was estimated during the final 100 ms of visual fixation (*t1* epoch) and from 50 ms before to 50 ms after the end of the gap (*t2* epoch). Post-target activation was estimated by measuring the magnitude of the target-related (*T* epoch) and of the saccade-related (*S* epoch) bursts of activity. **B** Orbital gap saccade task: spatial arrangement of the visual stimuli as seen from the animal's perspective. The crosses and circles represent the possible locations of the fixation point and saccade target (*FP* and *T* in panel A), respectively. The initial eye positions as specified by the fixation points were distributed along the same axis as the targets, and were located central, contralateral or ipsilateral relative to the recording side. The initial eye position associated with the eccentric fixation points were computed from the horizontal and vertical eye position. Two possible target positions were randomly interleaved for each initial eye fixation position. One target was located contralateral to the recording side, in the center of the neuron's movement field, whereas the other was equidistant relative to the fixation point but in the diametrically opposite direction. The fixation point and target for the current trial are highlighted and the *open arrow* represents the corresponding saccade

relative to the recording side; the movement fields of these neurons are situated contralateral to the recording side. We minimized large and systematic variations in target retinal error due to positional changes in the fixation point by setting the maximum initial eye-position error allowed by the computer-controlled fixation window to be ±1°. Blocks of trials were run in which, for each of the three initial eye fixation positions (+, Fig. 1B), two possible target positions (❍, Fig. 1B) were randomly interleaved (10–30 repetitions). For saccade-related neurons, one target was located contralateral to the side of the recording in the center of the neuron's movement field, whereas the other was equidistant relative to the fixation stimulus position but in the diametrically opposite direction. Data were collected from 90 SC neurons recorded during the orbital gap saccade tasks. However, in 11 additional cases (when the center of the movement fields was >15° eccentric), instead of the orbital gap saccade task, separate blocks of gap saccade trials were run from two or three different initial eye-fixation positions and the target was presented only in the center of the neuron's movement field, i.e. at the contralateral location. Typically, for the eye positions used, the resulting saccades were centrifugal, symmetrical, and centripetal. For neurons with fixation-related activity, targets were presented 10° to the right or left of the fixation point.

Data analysis

During off-line analyses, saccades were detected and marked using a computer program that identified the beginning and end of each saccade, using velocity and acceleration threshold criteria and template correlation (described by Waitzman et al. 1991). An experimenter verified these events to ensure accuracy. RT was measured as the interval from target appearance to the beginning of the saccade (Fig. 1A). Horizontal and vertical eye position signals were used to compute (with the Pythagorean theorem) the value of the different initial eye positions assumed in each experimental session and relative to the central orbital position (Fig. 1B). Rasters of neuronal discharge and continuously varying spike density functions (MacPherson and Aldridge 1979; Richmond et al. 1987) were aligned on specific paradigm events. To generate the spike-density function, a Gaussian pulse (σ=4 ms) was substituted for each spike and all Gaussians were summed together to produce a continuous function in time.

Unless specified otherwise, we analyzed only neurons with at least 10 trials distributed within each of the initial eye fixation and target positions of the task. The *pre-target* activity of individual neurons was measured from the number of action potentials occurring during two different intervals (Fig. 1A). Mean discharge rate was obtained from (1) the final 100 ms before the end of the visual fixation period (visual fixation epoch, *t1*); and (2) the interval from 50 ms before target appearance to 50 ms after target appearance (end of gap epoch, *t2*). Given the minimal visual afferent delay in our sample of SC neurons (~60 ms), the measure of activity during the *t2* epoch represents the latest estimate of pre-target activation. Following target presentation within their movement fields, SC neurons can exhibit two main types of *post-target* activity: a burst of activity time-locked to the appearance of the visual target and one aligned with the onset of the saccade (Fig. 1A). The level of the target-related burst of activity was calculated by taking the maximum value in the peak of the target-aligned spike density function that occurred between 60 and 100 ms after target presentation (*T* epoch). The level of the saccade-related burst of activity was considered as the peak rate in the saccade-aligned spike density function that occurred within ±20 ms from saccade initiation (*S* epoch).

Before statistical analyses, we performed a Kolmogorov-Smirnov test on each data set to determine whether it was drawn from a population with a normal distribution, and statistical comparisons within and between non-normal data were performed with non-parametric tests. For comparison of several samples, we used an analysis of variance (ANOVA) followed by an all-pairwise multiple comparison procedure (Student-Newman-Keuls of Dunn's method). Note, however, that the relationships between neuronal activity and either eye position or RT were estimated quantitatively using linear regression models, even though some data sets did not compose normal distributions. Statistical analysis results were considered significant at a P<0.05 level. All statistical tests were performed with SigmaStat (Jandel Scientific Corp.).

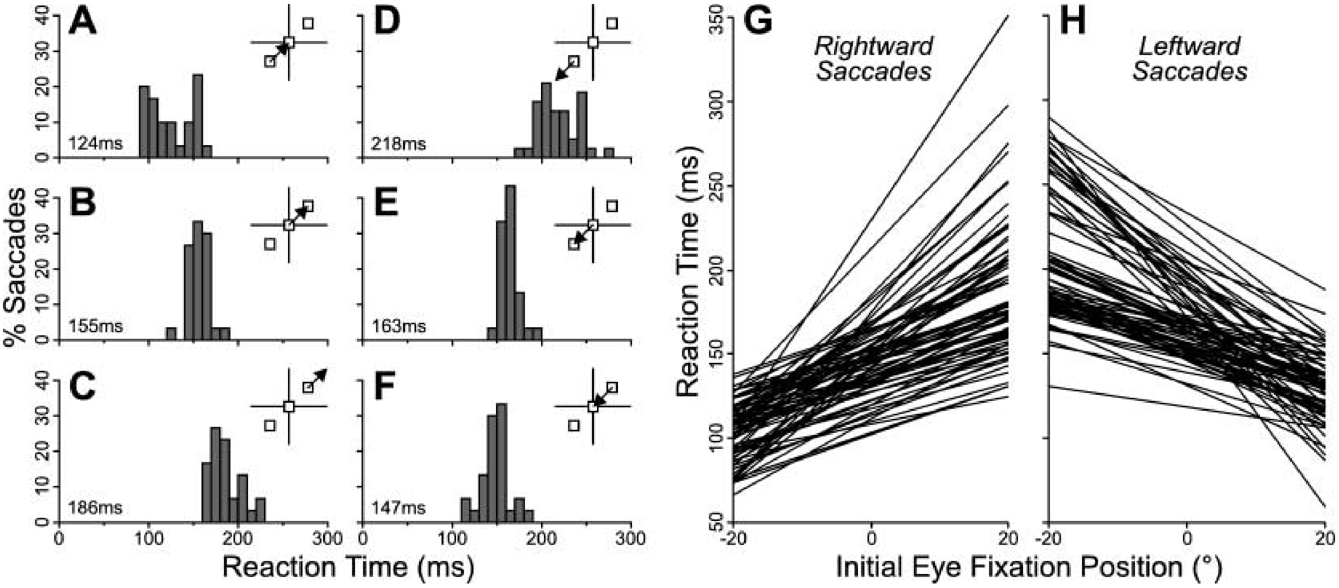


Fig. 2 **A–F** Histograms of representative distributions of reaction time for saccades of identical metrics but initiated from different initial eye fixation positions. Results from one session of the orbital gap saccade task, while buildup neuron *lx05i* was recorded (cf. Fig. 3, Fig. 4). Schematic in right-up portion of each histogram depicts saccades (→) made in each condition; □, different initial eye fixation positions. The mean reaction time is indicated in the bottom-left of each panel. **G, H** Linear regression curves (n=75) fitting the significant relationships between reaction time and initial eye position for saccades with rightward (**G**) and leftward (**H**) horizontal components. Left and right eye positions have negative and positive values, respectively. The Pearson correlation coefficient of the regressions had a mean of 0.64 (**G**) and –0.61 (**H**)

Neuron classification

The recorded SC neurons were separated into three different classes (Munoz and Wurtz 1993, 1995; Dorris et al. 1997). We classified as saccade-related neurons all neurons whose peak rate in the saccade-aligned spike density function exceeded 100 Hz for saccades to the center of the neuron's movement field. Among these saccade-related neurons, a *buildup neuron* exhibited pre-target activity during the end of gap (*t2*) epoch significantly greater than during the visual fixation (*t1*) epoch for at least one initial eye position, whereas a *burst neuron* lacked significant increases in pretarget activity. A neuron recorded in the rostro-lateral portion of the SC (within the representation of very small saccades) was classified as a *fixation neuron* if it discharged >10 Hz during both *t1* and *t2* epochs and displayed saccade-related pauses in activity.

Results

Behavioral performance

For the present behavioral data collected in the orbital gap saccade task, the modulation of saccadic RT distributions by changes in initial eye fixation position was similar to that described previously (Paré and Munoz 1996a). Figure 2A–F illustrates, for one data set, that shifting the initial eye position in a direction opposite to the saccade direction caused a systematic decrease in the distribution of RTs (Kruskal-Wallis ANOVA on ranks, $P<0.0001$). Thus, the shortest RTs were obtained with the eyes eccentric in the orbits and the saccades being centripetal (Fig. 2 A, F), whereas the centrifugal saccades displayed the longest RTs (Fig. 2C, D). In addition, the RT distribution of the centripetal saccades was often bimodal, consisting of short-latency *express* saccades (RT >70 ms and <120 ms) and longer-latency *regular* saccades (Fig. 2A) (Paré and Munoz 1996a; see also Fischer and Boch 1983). Data from 90 experimental sessions (10,767 correct saccades) were available and significant RT modulations were observed in 90% of them (one-way ANOVA or Kruskal-Wallis ANOVA on ranks, $P<0.05$).

To estimate quantitatively the change in RT as a function of initial eye position, the individual RTs for each session were fitted with a linear regression equation. Even though the saccadic vectors varied between sessions, we considered separately saccades with rightward and leftward horizontal components. By convention, right and left eye positions have positive and negative values, respectively. The regressions were found to be statistically significant in the great majority of the experimental sessions: 82/90 sessions for rightward saccades and 79/90 sessions for leftward saccades ($P<0.05$). Figure 2G, H shows the regression lines of the sessions for which the regressions were significant in both directions (n=75). The mean slope of the regressions was 2.07 ms/° for rightward saccades and –1.96 ms/° for leftward saccades. The corresponding y-axis intercept averaged 146 ms and 169 ms, and it was directly related to the regression slope (F-test, $P<0.0001$): when minimal, the slope approached zero. This outcome was due to the RT reaching its lower limit, as indicated by the occurrence of express saccades, whose minimal RT approaches the conduction time of the shortest neural pathways from the retina to the eye muscles (Carpenter 1981). In summary, mean RT decreased, on average, by 2 ms for each 1° change in initial eye fixation position; a value similar to our previous report, which included data collected in

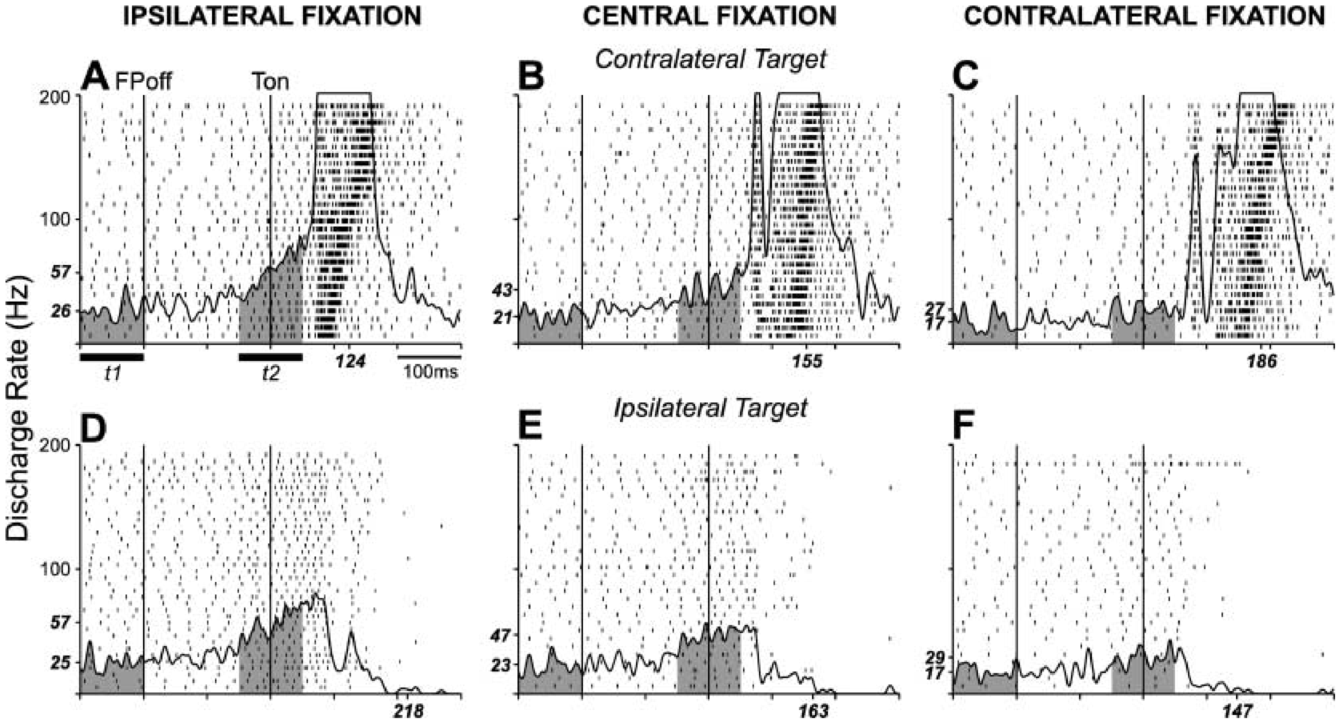


Fig. 3A–F Effects of initial eye fixation position on the pre-target activation of buildup neurons. Example from neuron *lx05i* recorded in the left SC of monkey *1* while it performed the orbital gap saccade task. The activity of this neuron was maximal for ipsilateral fixation position (**A** and **D**) and minimal for contralateral fixation position (**C** and **F**), regardless of the direction of the saccades. The presentation of the contralateral target elicited both a target- and saccade-related burst of action potentials (**A–C**). A cessation of activity followed the presentation of the ipsilateral target (**D–F**). Spike density functions are superimposed on the rasters of action potentials (*vertical tic-marks*; ranked according to increasing reaction time from bottom to top) and both are aligned on target onset (*Ton*), which occurred 200 ms after the disappearance of the fixation point (*FPoff*). The *shaded areas* under each spike density functions indicate the analysis epochs: visual fixation (*t1*) and end of gap (*t2*). The *bold ordinate values* indicate the mean levels of activity observed for the analysis epochs. The *bold abscissa values* indicate the mean reaction time of saccades made from each fixation position. The initial eye positions were referred relative to the recording side, whereas the target positions were referred relative to the visual field

eight extensive behavioral sessions (Paré and Munoz 1996a).

Among the saccades that were rejected from the above analysis, we identified two types of movements: anticipatory saccades and targeting errors. Anticipatory saccades were defined as saccades made toward the location of the visual target but not guided by the latter, i.e. they had RTs <70 ms (see Paré and Munoz 1996a). The percentage of anticipatory saccades with respect to the total number of saccades was only 1.4% (152/11,227), and the great majority (88%) were centripetal saccades produced when the initial eye position was eccentric, the same condition that facilitated the occurrence of short-latency express saccades. Targeting errors were saccades made in the direction opposite to the visual target, i.e. toward the other possible location of the target. Their incidence was about twice that of anticipatory saccades (2.7%, 308/11,227), and the great majority (81%) were centripetal saccades made in trials where the initial eye fixation position was eccentric and the target was presented further eccentric. Not all targeting errors had RTs <70 ms (67%), indicating that several of them occurred even though the information about the saccadic goal should have been available for a correct response to occur. In summary, saccade production was affected by changes in the initial eye position, with the RT of correct saccades being modulated and the likelihood of targeting errors influenced.

Neuronal sample

From recordings performed in the two superior colliculi of each of the three monkeys, 89 neurons fulfilled our neuron classification scheme; 12 neurons recorded in the orbital gap saccade task were rejected from the physiological analysis. According to our criteria 49% (n=44) were buildup neurons, 36% (n=32) were burst neurons, and 15% (n=13) were fixation neurons. Data from 35 buildup, 30 burst, and all fixation neurons were collected in the orbital gap saccade task. Data from nine buildup and two burst neurons were obtained only in separate blocks of trials with a single (contralateral) target position and a constant initial eye position that varied between blocks because their movement fields were >15° eccentric. Because our goal was to study the link between the SC early activity and both initial eye position and RT, we first describe the activity that occurred before target onset, i.e. the pre-target activation exhibited by buildup neurons. Next, we present the post-target activation of saccade-related neurons.

Pre-target activation of buildup neurons

Relationship with eye position

The majority of buildup neurons modulated their early low-frequency activity with respect to changes in initial eye position. Figure 3 illustrates the discharge of an example buildup neuron recorded in the left SC of *monkey 1* while it performed the orbital gap saccade task. This neuron displayed pre-target activity during both the visual fixation and gap periods at all initial eye positions. Targets presented in the contralateral hemifield (within the movement field) led to target- and saccade-related bursts of activity (A–C, most evident in panels B and C), whereas the low-frequency activity was abolished after targets were presented ipsilaterally (D–F). For each initial eye position, the early activity built up during the gap period to reach a level during the end of gap (*t2*) epoch that was about twice that of the visual fixation (*t1*) epoch, regardless of the location of the target to be presented. However, the absolute level of pre-target activity was maximal for the ipsilateral initial eye positions (Fig. 3 A, D). From ipsilateral to contralateral eye positions, the mean discharge rate decreased from 26 to 17 Hz during the *t1* epoch and from 57 to 27 Hz during the *t2* epoch. Using a two-way ANOVA, with initial eye position and target location as factors, the eye position effect on neuronal activity was statistically significant (*t1*, F=10.6; *t2*, F=66.9; P<0.0001). As expected, because it is an event occurring after the analysis epochs, the location of the target had no influence on pre-target buildup of activity (*t1*, F=0.1, P=0.78; *t2*, F=1.0, P=0.32).

Figure 4 summarizes the level of activity reached by all buildup neurons recorded in the orbital gap saccade task. Because target location never affected the level of pre-target activity of these neurons (see above), we pooled together the data obtained with the target presented at both the contralateral and the ipsilateral positions. During the *t1* epoch, the mean discharge rate did not vary significantly (Kruskal-Wallis ANOVA on ranks, P=0.17). During the *t2* epoch, the rate significantly decreased from 46 Hz (ipsilateral) to 32 Hz (central) and 20 Hz (contralateral) (Kruskal-Wallis ANOVA on ranks, p<0.0001; Student-Newman-Keuls test, P<0.05).

Given the modulation of the pre-target activity by initial eye position, an important issue is whether buildup neurons had a significant increase in their early discharge (*t2* >*t1*; paired *t*-test or Wilcoxon signed rank test, P<0.05) at each initial eye position. For the 35 buildup neurons tested in the orbital gap saccade task, significant buildup in activity at all three positions was found for 13 neurons (e.g. Fig. 3), at two positions (central and ipsilateral) for 12 neurons, and 9 neurons had significant buildup only at the ipsilateral position. One neuron showed significant buildup only at the central eye position. Similar results were obtained for the nine buildup neurons tested in blocks of trials where the initial eye position was kept constant. Overall, 98% of the neurons showed significant buildup activity at the ipsilateral eye position, 68% at the central position, and 30% at the contralateral position. In summary, both the level of pre-target buildup activity and its presence generally were modulated by initial eye position.

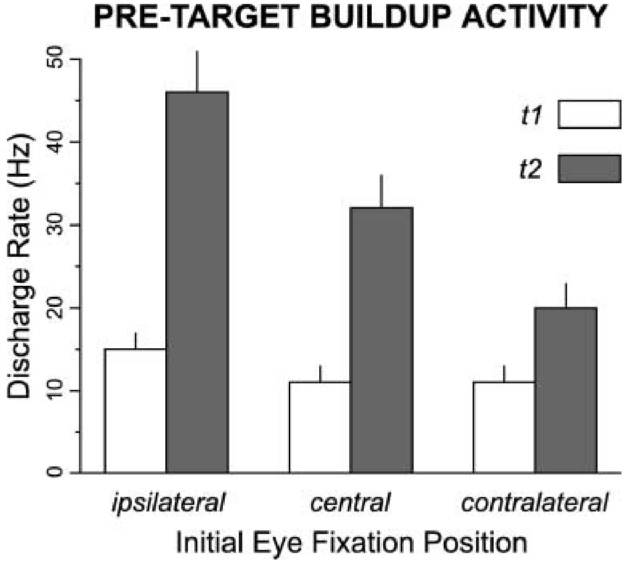


Fig. 4 Average level of pre-target buildup activity (and SE) as a function of initial eye position during visual fixation (*t1*) and end of gap (*t2*) epochs. Data from the sample of 35 buildup neurons recorded in the orbital gap saccade task. Trials with contralateral and ipsilateral targets were condensed

Linear regression analyses were performed to estimate quantitatively the change in discharge rate of the pre-target activity as a function of initial eye position. Figure 5 (A, B, E, F) illustrates this analysis for the example buildup neuron appearing in Fig. 3, while Fig. 6 and Table 1 summarize the correlation coefficients and the slope of the regression equations obtained from the sample. For the activity present during the *t1* epoch, the average correlation coefficient was –0.14 and the slope –0.16, with 52% of the neurons displaying a statistically significant relationship. For the activity present during the *t2* epoch, the average correlation coefficient was –0.35 and the slope averaged –0.8 Hz/°. A linear rate–position relationship was statistically significant in 75% of the buildup neurons. As in the example neuron of Fig. 3 and Fig. 5, the pre-target activity decreased with increasing contralateral positions for nearly all the neurons with a significant correlation. A positive relationship was observed in five neurons in the *t1* analysis and in only one neuron in the *t2* analysis (Fig. 6). In summary, SC pre-target activation was strongly and consistently modulated by changes in initial eye position.

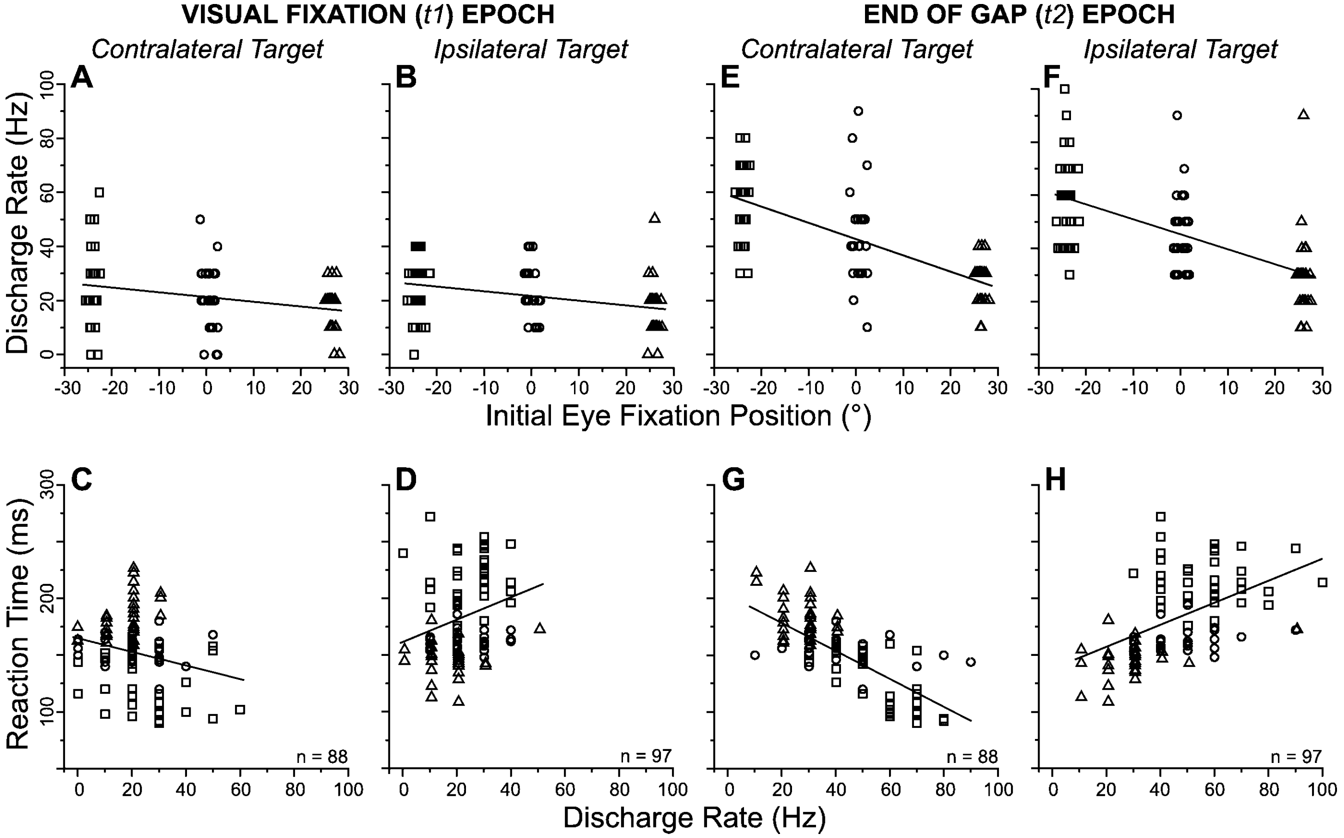


Fig. 5A–H Relationships between discharge rate of example buildup neuron *lx05i* as a function of initial eye fixation position (**A**, **B** and **E**, **F**) and saccadic reaction time (**C**, **D** and **G**, **H**). Each data point was obtained from a single trial and sampled during either the visual fixation (*t1*) epoch (**A–D**) or the end of gap (*t2*) epoch (**E–H**). Shown are the data from trials in which the target was presented either contralateral (**A**, **C** and **E**, **G**) or ipsilateral (**B**, **D** and **F**, **H**) in the visual field. The symbols indicate the different initial eye positions relative to the recording side: □, ipsilateral; ○, central; Δ, contralateral. The relationships between discharge rate and initial eye position and between reaction time and discharge rate were obtained by linear regression analysis. For the relationship between rate and eye position, the parameters of the linear regression equations are: *b*=21, slope=–0.18, *r*=–0.30 (**A**); *b*=22, slope=–0.18, *r*=–0.36 (**B**); *b*=43, slope=–0.60, *r*=–0.69 (**E**); *b*=44, slope=–0.56, *r*=–0.62 (**F**). For the relationship between reaction time and rate, the regression parameters are: *b*=168, slope=–0.64, *r*=–0.24 (**C**); *b*=156, slope=1.04, *r*=0.30 (**D**); *b*=209, slope=–1.28, *r*=–0.74 (**G**); *b*=133, slope=1.02, *r*=0.54 (**H**). All regressions were significant (*P*<0.05)

Relationship with reaction time

Besides its eye-position dependence, the pre-target activity of buildup neurons was also correlated with RT. For instance, Fig. 5 illustrates how this activity in the example buildup neuron (Fig. 3) was inversely related to the RT of contraversive saccades made into the movement field (Fig. 5C, G) and directly related to the RT of ipsiversive saccades made in the opposite direction (Fig. 5D, H). Linear regression analyses were again used to estimate quantitatively the change in RT as a function of the change in pre-target buildup activity. Figure 7 and

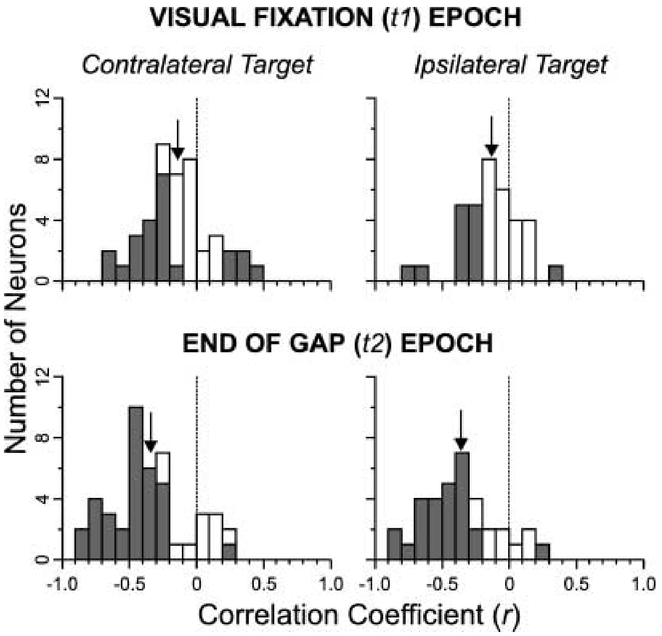


Fig. 6 Histograms of the distribution of the correlation coefficients of the linear regressions between initial eye position and rate of pre-target buildup activity during the visual fixation (*t1*) and end of gap (*t2*) epochs and before saccades to targets located contralateral and ipsilateral relative to fixation. Data are from 44 buildup neurons. *Shaded bar* represents statistically significant correlation (*P*<0.05)

Table 1 Relationship between pre-target activity of buildup neurons and initial eye fixation position (linear regression analysis). *Top*: correlation coefficient (*r*) values (mean ±SD and range in brackets). *Middle*: slope values (mean ±SD and range in brackets). *Bottom*: respective proportion of neurons showing a statistically significant correlation ($P<0.05$). Thirty-five neurons were tested in the orbital gap saccade task (randomized contralateral and ipsilateral targets) and nine neurons in blocks of trials with only a contralateral target and at least three different initial eye fixation positions

	Contralateral target		Ipsilateral target	
	Visual fixation (t1)	End of gap (t2)	Visual fixation (t1)	End of gap (t2)
Correlation coefficient	–0.14±0.25 [–0.64 to 0.43]	–0.34±0.30 [–0.84 to 0.25]	–0.13±0.22 [–0.71 to 0.34]	–0.36±0.27 [–0.81 to 0.21]
Slope (Hz/°)	–0.16±0.36 [–1.60 to 0.56]	–0.79±1.03 [–4.20 to 0.48]	–0.16±0.43 [–2.10 to 0.71]	–0.81±1.01 [–4.31 to 0.52]
Significant correlation*	52% (23/44)	75% (33/44)	37% (13/35)	74% (26/35)

*A non-parametric correlation analysis (Spearman rank order correlation) gave similar percentages: 45%, 80%, 40%, and 71%, respectively

Table 2 Relationship between reaction time and pre-target activity of buildup neurons (linear regression analysis). *Top*: correlation coefficient (*r*) and slope values (mean ±SD and range in brackets), along with the respective proportion of neurons showing a statistically significant correlation (*F*-test, $P<0.05$). Thirty-five neurons were tested in the orbital gap saccade task (randomized contralateral and ipsilateral targets) and nine neurons in blocks of trials with only a contralateral target. *Bottom*: proportion of neurons that showed a statistically significant correlation ($P<0.05$) in the study of Dorris et al. (1997), wherein all the saccades were generated from the central straight-ahead position

	Contralateral target		Ipsilateral target	
	Visual fixation (t1)	End of gap (t2)	Visual fixation (t1)	End of gap (t2)
Correlation coefficient	–0.16±0.20 [–0.57 to 0.35]	–0.41±0.24 [–0.83 to 0.21]	0.11±0.19 [–0.28 to 0.65]	0.24±0.21 [–0.10 to 0.69]
Slope (ms/Hz)	–0.64±0.81 [–2.91 to 1.38]	–0.82±0.68 [–3.46 to 0.38]	0.39±0.69 [–0.66 to 3.02]	0.46± 0.58 [–0.18 to 1.82]
Significant correlation*	41% (18/44)	82% (36/44)	20% (7/35)	54% (19/35)
Dorris et al. 1997	14% (4/29)	41% (12/29)	11% (2/19)	11% (2/19)

*A non-parametric correlation analysis (Spearman rank order correlation) gave similar percentages: 48%, 82%, 28%, and 54%, respectively

Table 2 summarize the correlation coefficients of all the regression equations. For the activity preceding saccades into the movement fields, 41% and 82% of the buildup neurons had significant correlation during visual fixation (*t1*) and end of gap (*t2*) epochs, respectively (Fig. 7, left; Table 2). In general, increases in buildup activity were accompanied by significant decreases in RT. For the neurons showing a significant negative correlation during the *t2* epoch (*n*=36), the average coefficient was –0.50 and the average slope was –0.99 ms/Hz. However, this relationship appeared to be restricted to the initiation of saccades with relatively short RTs. This restricted relationship derives from the absence, or near-absence (e.g. Fig. 5G), of pre-target activity for saccades with RTs of about 200 ms and greater. For the sample of neurons, the mean intercept of the significant linear regressions was 175 ms (range 106 to 228). The SC pre-target activation could therefore not possibly be effective in influencing the initiation of saccades with longer RTs.

For the saccades made in the direction opposite to the movement fields, RT was significantly correlated to the discharge rate of 20% and 54% of the neurons during the *t1* and *t2* epochs, respectively (Fig. 7, *right*; Table 2). In these cases, increases in buildup activity generally were associated with increases in RTs and the correlation coefficients were lower. Among the neurons with a significant positive correlation during the *t2* epoch (*n*=19), the average coefficient was 0.40 and the average slope was 0.75 ms/Hz.

In our previous report, we analyzed the relationship between pre-target buildup activity and RT when saccades were produced only from central initial eye position (Dorris et al. 1997). Not only was this relationship preserved when multiple initial eye fixation positions were used, but the percentage of neurons showing a significant correlation between their discharge rate and RT was increased (Table 2). One striking difference was the large increase in the percentage of significant correlations between buildup activity and the RT of saccades made in the direction opposite to the movement fields (11% versus 54%). The originally low percentage was taken to indicate that the early activity of buildup neurons might only be implicated in the process of initiating saccades made into the movement field. Our results therefore suggest a broader role in the saccade initiation process (see also Dorris and Munoz 1998). One major

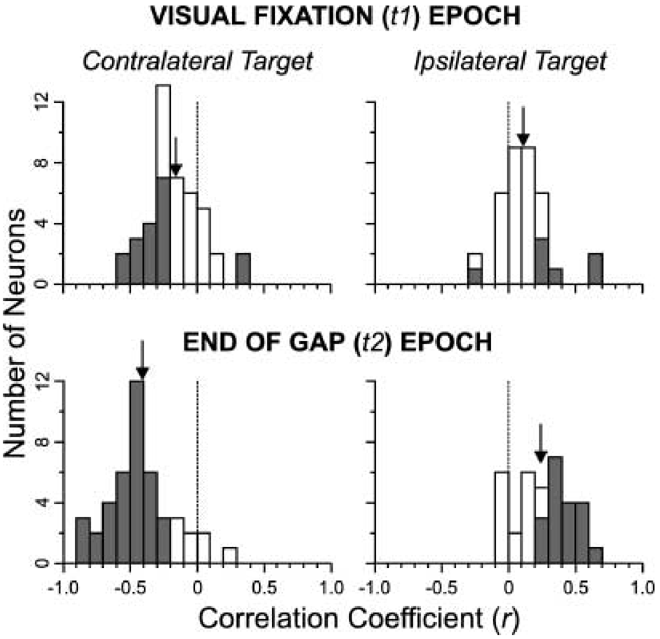


Fig. 7 Histograms of the distribution of the correlation coefficients of the linear regression between saccadic reaction time and rate of pre-target buildup activity during the visual fixation (*t1*) and end of gap (*t2*) epochs and before saccades to targets located contralateral and ipsilateral relative to fixation. Data are from 44 buildup neurons. *Shaded bar* represents statistically significant correlation ($P<0.05$)

factor contributing to the increase in the percentage of significant correlations between RT and buildup activity appeared to be the wider RT distributions. Indeed, the introduction of eccentric initial eye positions always increased the range of RTs and generally doubled it. For the 35 buildup neurons recorded in the orbital gap saccade task, the range in RTs averaged 76 ms (SD=41, range 14 to 230) for the central initial eye position alone, whereas it averaged 179 ms (SD=77, range 63 to 400) with the additional two eccentric positions.

In summary, a high level of pre-target buildup activity predicted well the occurrence of relatively short RT saccades made within the neuron’s movement field and it generally anteceded ipsiversive saccades with long RTs. This relationship between RT and neuronal activity persisted in spite of changes in initial eye position and, in fact, improved considerably.

Relationship with saccade probability

We showed that the pre-target activity of buildup neurons was related to both initial eye position and RT. Even after removing the influence of RT from the relationship between pre-target activity and initial eye position, the latter two parameters continued to be significantly correlated in most neurons: 61% and 54% for contralateral and ipsilateral target trials, respectively (ANCOVA, $P<0.05$). The target-position independence of the eye position effect (Table 1) and the unequal relationship between pre-target activity and RT (contra versus ipsi, Table 2) predicted this outcome. Altogether, these observations suggest that initial eye position contributes to the central processes underlying saccade initiation, for which buildup neurons are believed to be important (Dorris et al. 1997). Nevertheless, some additional observations suggest that the neuronal modulation might not be strictly related to initial eye position. To dissociate activity related to eye position from activity related to saccade production, we recorded nine of our neurons in separate blocks of *catch* gap trials, wherein saccades never followed the gap period because the next visual stimulus was presented at the fixation position. In this condition, the neuronal activation rapidly vanished, usually within the first five-ten trials (not shown). The same phenomenon was observed when saccades were made from an ipsilateral eye position, but repeatedly away from the neurons’ movement fields. We take these observations to signify that the pre-target SC activity is related to the probability that a saccade will be made within a neuron’s movement field (Dorris and Munoz 1998, see their Figs. 1 and 2; see also Basso and Wurtz 1998), and prior probability is a prerequisitefor the eye position influence. Thus, when different saccades are available, the probability of producing centripetal saccades may become *implicitly* augmented. In other words, the monkeys appeared to attribute more importance to visual targets requesting a centripetal saccade, despite the fact that at each initial eye position the *explicit* probability that the target appeared within the neuron’s movement field was always 50%.

Relationship with targeting errors

The existence of targeting errors in the orbital gap saccade task provided us with the opportunity to investigate whether the pre-target buildup activity, whose level is related to RT, can also predict the occurrence of a certain saccade out of two possible ones. Hypothetically, high early activity should be related to short RT and increased probability of saccades directed within the movement field. As reported above, the incidence of targeting errors was low and a quantitative analysis of the associated buildup activity was limited. Given the frequency distribution of these errors, we concentrated on the condition wherein both the initial eye position and the target locations were ipsilateral to the recording side. In this condition, the buildup activity was generally maximal, but it declined quickly starting ~80 ms after target appearance and the correct long-latency saccades were later generated away from the movement field (Fig. 8 A). On error trials (Fig. 8B), centripetal instead of centrifugal saccades were generated within the movement field, and the neuron discharged a saccade-related burst of activity. Because only the targeting errors were made within the neuron’s movement field, the buildup activity during the error trials was hypothesized to be higher than during the correct trials. As shown in Fig. 8, this is exactly what we observed.

Targeting errors could include responses with RTs ranging from 0 to 70 ms (Fig. 8B, *dotted traces*), and measuring the pre-target activity during the *t2* epoch was not valid because it could also include the saccade-related activity. We therefore considered a 50-ms epoch ending at target appearance and, because this interval is short, we quantified the buildup activity by computing the mean value of the spike density function during this epoch. The analysis excluded trials with RT <20 ms and included only neurons (*n*=10) recorded during sessions that yielded, in the condition that we examined, at least four error trials. For the neuron shown in Fig. 8, this activity was 78 and 148 Hz during the correct and error trials, respectively. For the 10 neurons that were analyzed, the buildup activity reached, on average, 60 Hz during the correct trials and 92 Hz during the error trials (paired *t*-test, *df*=9, *t*=–3.66, *P*<0.01). Consistent with our hypothesis, increased SC early activity associated with ipsilateral shifts in eye position was related to the occurrence of erroneous centripetal saccades into a neuron's movement field.

Discharges of fixation neurons

In the gap saccade task, fixation neurons discharged tonically during periods of visual fixation, often reduced their activity during the gap period, and exhibited a saccade-related pause in activity (Munoz and Wurtz 1995; Dorris and Munoz 1995). In the orbital gap saccade task, the tonic activity of fixation neurons during the visual fixation (*t1*) and end of gap (*t2*) epochs averaged, respectively, 56 and 42 Hz, and was not significantly modulated by initial eye position (Fig. 9) (Kruskal-Wallis ANOVA on ranks, *P*=0.95). These results corroborate the reported absence of eye-position effect on these neurons in a fixation task (Munoz and Wurtz 1993). In addition, the activity of fixation neurons was not correlated with RT on a trial-by-trial basis (not shown), thereby confirming our previous results (Dorris et al. 1997). Furthermore, the constant reduction in fixation-related activity during the gap could not predict the different mean RTs observed for the saccades produced from each initial eye position. These results further support the hypothesis that the early activity of saccade-related neurons, as opposed to the potential disinhibition afforded by a reduction in the activity of fixation neurons, provides the predominant signal determining saccade initiation (Dorris et al. 1997).

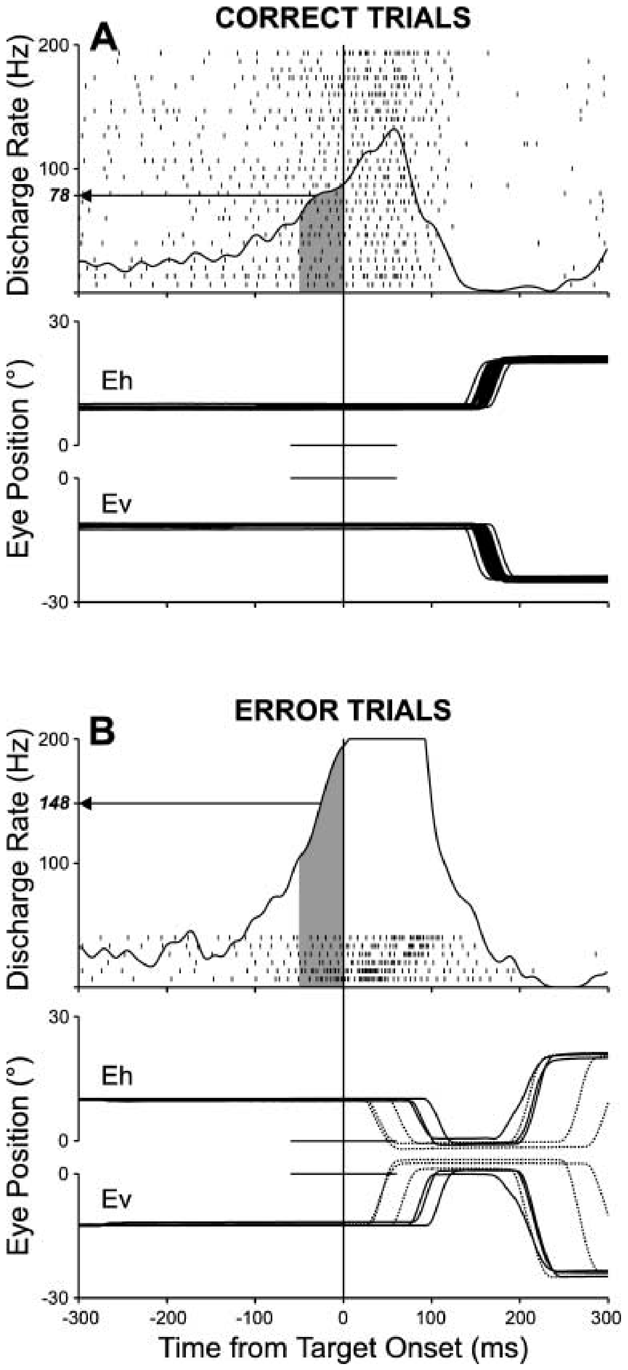


Fig. 8A, B Relationship between targeting errors and SC pre-target buildup activity. Example buildup neuron (*ma62d*) recorded in the right SC of monkey *3* performing the orbital gap saccade task. Panels show its activity associated with trials where correct (**A**) and erroneous (**B**) saccades were produced as both the initial eye position and the target location were ipsilateral to the recording side, i.e. opposite to the movement field. In this condition, the presentation of the visual stimulus elicited a cessation of neuronal activity (**A**) unless a targeting error occurred, in which case the saccade was made within the movement field and preceded by a higher level of buildup activity and a saccade-related burst of action potentials (**B**). Spike density functions are superimposed on the rasters of action potentials (*vertical tic-marks*; ranked according to increasing reaction time) and both are aligned on target onset along with the horizontal (*Eh*) and the vertical (*Ev*) eye position traces. The *bold ordinate values* indicate the mean value of the spike density function during the 50-ms analysis epoch (*shaded area*) that ends at the onset of the target. *Dotted position traces* indicate trials with a saccadic (anticipatory) reaction time <70 ms. Notice that each erroneous saccade was followed by a corrective saccade that brought the eyes toward the target location but after the typically long reaction time of the correct saccades

Discharges of burst neurons

The majority of burst neurons (66%, 21/32) displayed no significant activity during visual fixation or during the gap period. Most of them were, in fact, totally silent during fixation periods. Of the minority of neurons (34%, 11/32) that discharged tonically during all fixation periods, only one had a tonic activity significantly modulated by eye position. This neuron's modulation, however, differed from what we observed in buildup neurons as its level of activity increased with contralateral shifts in initial eye position; only one buildup neuron showed a significant positive correlation (Fig. 6, bottom). With the exception of a few neurons (n=4), which at times exhibited gap-related discharges for ipsilateral eye positions, most activity changes in burst neurons occurred after the target appearance.

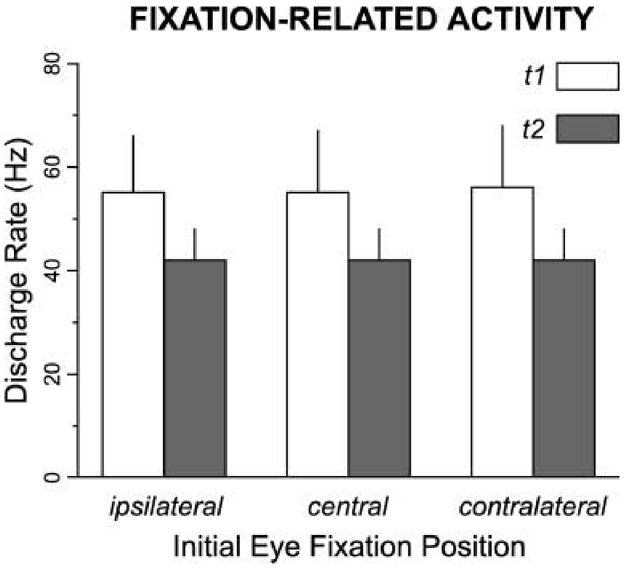


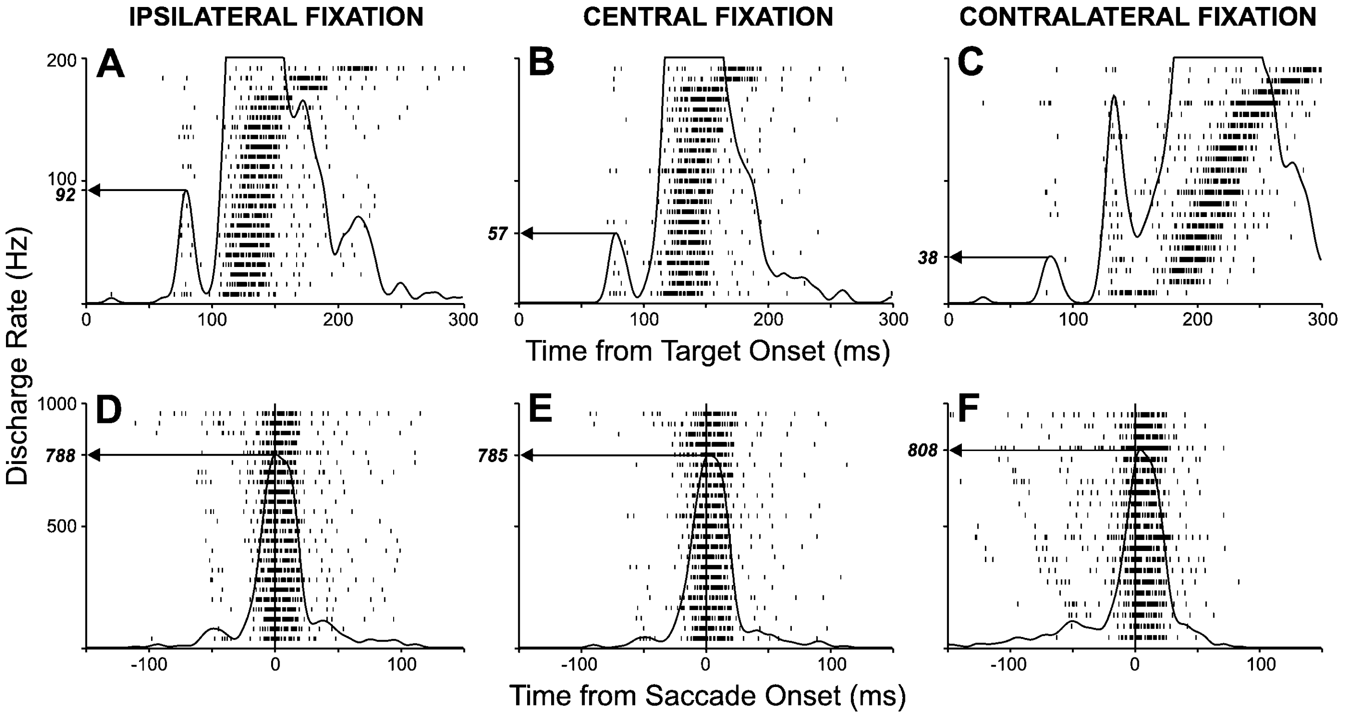
Fig. 9 Average level of fixation-related activity (and SE) as a function of initial eye position during visual fixation (*t1*) and end of gap (*t2*) epochs. Data from the sample of 13 fixation neurons recorded in the orbital gap saccade task. Trials with contralateral and ipsilateral targets were condensed

Post-target activation of saccade-related neurons

Target-related burst of activity

Saccades made within the movement fields of saccade-related neurons were preceded by a high frequency burst of activity, which often followed a lower frequency burst that was time-locked to the target presentation. We fre-

Fig. 10A–F Effects of initial eye fixation position on the post-target activation of burst neurons. Example from neuron *jh13f* recorded in the right SC of monkey *2* performing the orbital gap saccade task. Spike density functions are superimposed on the rasters of action potentials (*vertical tic-marks*; ranked according to increasing reaction time) and aligned on the presentation of the contralateral targets (**A–C**) and on the start of the associated saccades (**D–F**) produced within the movement field. The presentation of the contralateral target elicited both a target- and saccade-related burst of action potentials. The modest target-related burst (~80 ms) was modulated by initial eye position, being maximal for ipsilateral position (**A**) and minimal for contralateral position (**C**). The intense saccade-related burst did not change in magnitude (**D–F**). The *bold ordinate values* indicate the peak value of the spike density function for the target- (**A–C**) and saccade-related (**D–F**) bursts of activity



quently observed systematic changes in the magnitude of this target-related activity in both buildup and burst neurons. Figure 10A–C illustrates, for a burst neuron, how a contralateral shift in initial eye position caused a decrease in this initial burst, which reached its peak ~80 ms after target presentation. We performed an analysis of the magnitude of the target-related activity of all the saccade-related neurons recorded in the orbital gap saccade task and whose target-related activity was >50 Hz (relative to the *t2* activity) when the initial eye position was central. This was the case for 70% of the burst neurons (21/30) and 66% of the buildup neurons (23/35). Figure 11A illustrates the average peak discharge rate reached by both burst and buildup neurons, whose modulation was very similar. With the initial eye position shifting contralaterally, the target-related activity of the two classes of neurons significantly decreased from 377 Hz (ipsilateral) to 339 Hz (central) and 280 Hz (contralateral) (Kruskal-Wallis ANOVA on ranks, $P<0.005$; Student-Newman-Keuls test, $p<0.05$). Thus, changes in the level of both target-related and pre-target activities concur to reveal that there was an increase in the excitability of saccade-related neurons when the initial eye position was shifted ipsilaterally.

The short RT-saccades associated with the increased SC excitability occasionally included express saccades, whose initiation coincided with the target-related activity coalescing with the saccade-related activity (Edelman and Keller 1996; Dorris et al. 1997). Although this is consistent with the idea that an ipsilateral shift in initial eye position both augments the excitability of SC saccade-related neurons and shortens saccadic RT, we sought to control for the possible confound that the target-related bursts associated with express saccades was contaminated by saccade-related activity. In a second analysis, we excluded trials in which express saccades were produced. For the available burst and buildup neurons (n=39), the average peak discharge rate then decreased from 346 Hz (ipsilateral) to 316 Hz (central) and 275 Hz (contralateral), with the initial eye position shifting contralaterally. This was still a statistically significant modulation (Kruskal-Wallis ANOVA on ranks, $P<0.05$; Student-Newman-Keuls test, $P<0.05$).

Saccade-related burst of activity

As exemplified in Fig. 10 (D–F), initial eye position (or RT) seemingly did not modify the magnitude of the saccade-related burst of activity produced by SC neurons. Such lack of variability in the saccade-related activity was a common observation for the sample of 30 burst and 35 buildup neurons recorded in the orbital gap saccade task. On average, the saccade-related burst reached 522 Hz (range 107 to 869), and initial eye position had no significant influence (one-way ANOVA, P=0.88) (Fig. 11 B). Our data therefore further support the widely recognized notion that SC neurons encode the metrics of saccades in relative coordinates (Sparks and Mays 1990).

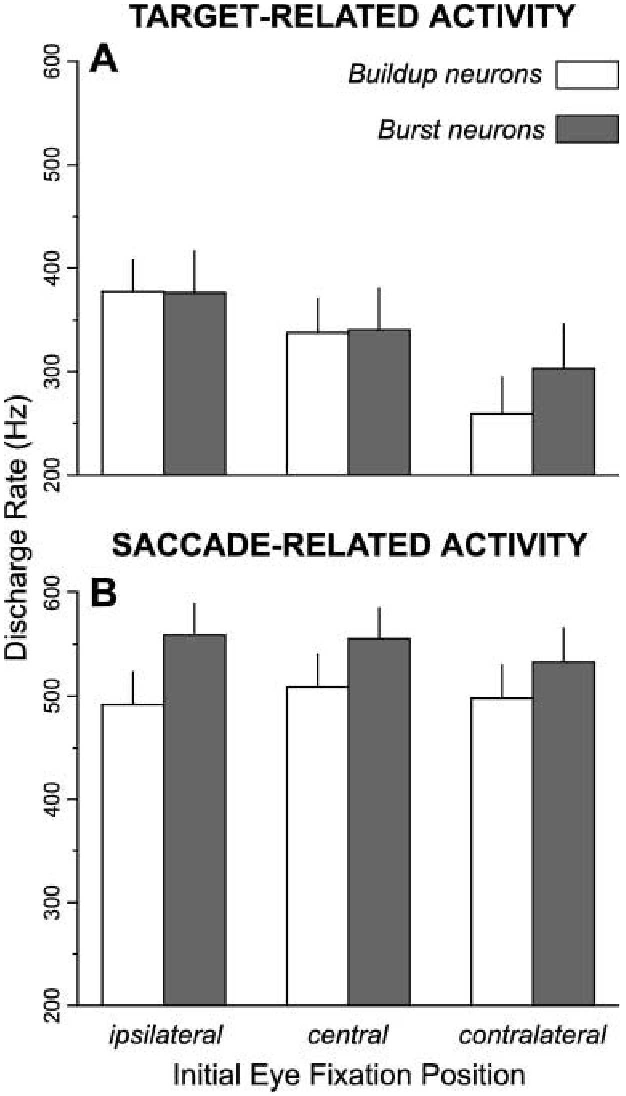


Fig. 11A, B Average level of target- (**A**) and saccade-related (**B**) activity (and SE) as a function of initial eye fixation position for buildup neurons and burst neurons recorded in the orbital gap saccade task

Only in a minority of neurons (20%, 13/65) was the magnitude of the saccade-related burst of activity systematically modulated (repeated t-tests with corrected degrees of freedom, $P<0.0167$). The activity of four neurons increased monotonically from ipsilateral to contralateral initial eye position, whereas the activity of nine neurons decreased. Note that this variability could simply have originated from some small differences in saccade metrics partially caused by the changes in initial eye position (Collewijn et al. 1998; Pélisson and Prablanc 1988).

Discussion

We showed that, with changes in initial eye position in the direction opposite to a neuron's movement field, the excitability of SC neurons increased, while the RT of centripetal saccades made within the neuron's movement field decreased. The change in neuronal excitability was conspicuous in the pre-target buildup of activity (see Figs. 3–6), but was also manifest in the burst of activity that occurred time-locked to target presentation (Fig. 11A). In contrast, the saccade-related burst of activity remained invariant (Fig. 11B). This eye position influence on SC neuronal excitability could facilitate the initiation of re-centering saccades (Fig. 7 and Fig. 8). These findings indicate that the eye position effect on saccade production is not a peripheral phenomenon due to the stretching of ocular muscles. Rather, they suggest that the neural control of saccade initiation, as reflected in SC activity patterns, can make use of eye position signals.

Such a *postural*[1] influence on SC neurons, whose discharges are intimately linked to saccade production (Sparks 1978), represents an additional factor regulating the neural processes of saccade selection and initiation. The modulated neuronal activity, however, can be dissociated from eye position signals and it appears more closely related to *implicit* saccade production probability. The re-centering bias in saccade regulation created by changes in initial eye position may therefore be analogous to manipulating the likelihood that a certain saccade will be executed (Carpenter and Williams 1995; Dorris and Munoz 1998; see also Basso and Wurtz 1998; Lecas et al. 1986). Although we did not manipulate initial eye position in a parametric way (only ipsilateral, central, and contralateral positions were employed), the independent observation that RT does vary systematically with initial eye position (Paré and Munoz 1996a) suggests that the effect of initial eye position on SC neuronal activity may not be determined by a simple 'binary' assessment of being either eccentric or not from the central position. Is this behavioral bias due entirely to the fact that our monkeys received extensive experimental training, in which the repetitious presentation of a fixation light in the central visual field might have induced an artificial strategy? The exact contribution of training cannot be easily estimated, but we do not think that it is an indispensable factor because an eye position–RT relationship has also been observed in human subjects without comparable training (Fuller 1996; Zambarbieri et al. 1995).

Several lines of evidence have already suggested that SC neurons have access to eye position signals (for review see van Opstal et al. 1995). It is therefore possible that saccadic programs are combined with eye position signals within the SC. Alternatively, saccadic programs could be modified beforehand, e.g. within cortical areas providing inputs to the SC (cf. Andersen et al. 1990). Two particular physiological studies are directly related to our findings. First, Peck et al. (1995) reported that, in the cat SC, the neuronal responses to visual stimuli are influenced by initial eye position. This is consistent with our results, which incidentally showed more systematic effects. Second, van Opstal et al. (1995) reported that the saccade-related activity of several monkey SC neurons (38%, 22/57) could increase significantly with a shift in eye position in the direction toward (19%), opposite (4%), or orthogonal (16%) to the neuron's movement field. Our comparatively simpler analysis (and slightly different experiments) identified a similar fraction of neurons with co- and anti-linear modulations (20% versus 23%). However, the lack of systematic modulation in both our sample and that of van Opstal and colleagues suggests that the eye position effect on SC saccade-related activity impacts behavior less significantly than its effect on the pre-target activity.

The consequence of an eye position effect on SC activity in advance of saccades may restrict visual exploration by limiting eccentric excursions of the eyes in the orbits, via an influence on the saccade selection and initiation processes. One possible benefit of this behavioral constraint may be to reduce sustained muscle work, which increases with ocular eccentricity (Robinson 1981). The most obvious advantage has to do with a visuo-motor optimizing strategy. With saccades bringing the eyes to appreciable eccentric orbital positions, the saccadic system loses the flexibility to respond to incoming stimuli if the latter require movements toward further eccentric positions. Therefore the eyes are optimally poised for the next movement only when they are at the center of the oculomotor range (Tweed 1997). This optimizing strategy has been shown to be pertinent particularly in the control of head-unrestrained gaze shifts (Fuller 1996), which may involve the SC (Freedman and Sparks 1997; Munoz et al. 1991).

When an observer's head is unrestrained, the range of positions that the eyes can take in the orbits no longer delimits the extent of visual exploration by saccadic shifts of the visual axis (gaze). Thus, the facilitation of centripetal saccades emphasized here may be downplayed by the involvement of the head enabling greater exploration capability. Nevertheless, the timing of the two mobile segments necessitates precise adjustments that must take into account the current eye position as well as the position of the head relative to the body (see Fuller 1996). Theoretically, gaze shifts can be executed by dividing the contribution of eye and head movements in many different combinations. However, these combinations are constrained by regulatory interactions between the mobile segments (Becker and Jurgens 1992; Delreux et al. 1991; Stahl 1999; Volle and Guitton 1993). In the head-unrestrained situation, the eye position modulation of SC neuronal activity could then con-

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[1] The term posture emphasizes that the observed changes in neuronal activity and behavior are attributed to changes in the position of the eyes within the orbits (body reference) and not to changes in the position of the eyes in space (spatial reference). Varying the position of the head would be necessary to corroborate this hypothesis.

tribute to optimizing eye–head coordination timing (Fuller 1996). Access to neck muscle afferents may further enable the SC in this function (Abrahams and Rose 1975; Abrahams and Turner 1981).

Neural signals for saccade initiation

An adequate description of most behavioral and physiological data that relate to saccade initiation can be found in the linear rise-to-threshold model developed by Carpenter (Carpenter 1981; Carpenter and Williams 1995). In this model, a saccadic target initiates the rise of a decision signal, which represents an estimate of the likelihood of the hypothesis that the target is present. This signal grows at a constant rate until a fixed threshold value is reached and the saccade is triggered. Variability in RT can be caused by variability in the initial value and in the rate of growth of the decision signal. Possible neural correlates of these signals have been identified. The *pre-target* buildup of activity of SC neurons undoubtedly contributes to setting the initial value of the decision signal, particularly in warning RT tasks like the gap task (Dorris et al. 1997). The *post-target* buildup of activity of saccade-related neurons in the frontal eye field (Hanes and Schall 1996) could be associated with the rising decision signal; analogous signals in the SC need to be documented. Lastly, the RT-invariant threshold can be discerned in the constant value reached by the saccade-related burst of activity (Dorris et al. 1997; Edelman and Keller 1996; Hanes and Schall 1996).

In previous reports, our group has hypothesized that express saccade generation requires advanced preparation of motor programs (Paré and Munoz 1996a) subserved by augmented SC pre-target activity that generally shorten RT and ultimately can allow SC target-related bursts of activity to trigger the movement (Dorris et al. 1997). Neuronal pre-target activity can be observed almost exclusively in warned RT tasks such as the gap task. Moreover, our study showed that the SC pre-target activity could only predict speeded RTs, which generally are produced in this gap task and specifically require the integrity of the SC, as demonstrated by the ablation study of Schiller and colleagues (Schiller et al. 1987). This experimental condition therefore corresponds to a special case of neural control of saccade initiation, as pre-target activity generally is absent in other RT tasks that normally yield longer RT. The latter would instead rely exclusively on decision processes reflected in coordinated post-target activation among cooperating neurons within and between oculomotor structures (Schall and Thompson 1999).

Conclusion

The SC is a site of convergence for sensory representations and pre-motor signals. Both categories of neural signals are influenced by the position of the eyes within the orbits during the period of fixation preceding a saccade. This influence renders the saccadic system capable of transforming the spatial coordinates of sensory targets into appropriate commands for saccades (see for review Sparks and Groh 1999). It also permits, as we suggest here, an adaptive strategy for optimizing saccade regulation by modifying the neural processes underlying the selection and the initiation of saccades. The orbital influence on SC neurons, however, seems to be limited to activity patterns in advance of saccade initiation since the saccade-related bursts of activity represent invariant saccadic commands that perhaps are modified by downstream elements to generate the adequate innervation of eye muscles.

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