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RAPID PUBLICATION

A Neural Correlate for the Gap Effect on Saccadic Reaction Times in Monkey

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SUMMARY AND CONCLUSIONS

1. The reduction in saccadic reaction time associated with the introduction of a period of darkness between the disappearance of an initial fixation point and the appearance of a new peripheral saccade target is known as the gap effect. Fixation cells in the rostral pole of the monkey superior colliculus have been implicated in the control of active visual fixation and suppressing saccadic eye movements. To determine whether specific variations of fixation cell discharge was correlated to the gap effect, we recorded the activity of fixation cells while a monkey generated visually guided saccades with various temporal gaps between the disappearance of the initial fixation point and the appearance of a peripheral saccade target.

2. The saccadic reaction times of the monkey were shortest with gap durations of 200–300 ms and increased with shorter or longer gap durations. The activity of fixation cells followed a similar time course, having a minimum discharge rate 200–300 ms into the gap, and increased activity at the time of target appearance with smaller or larger gap durations.

3. We propose that the activity of fixation cells in the monkey superior colliculus provide a neural correlate of the gap effect. The decrease in activity of fixation cells 200–300 ms into the gap weakens the powerful state of inhibition which they normally exert upon the saccade generating system, allowing targets to be acquired at shorter reaction times.

INTRODUCTION

Saccades are high-velocity eye movements used to look from one point to another in the visual field. Saccadic reaction times can be reduced by introducing a temporal gap of no stimuli between the disappearance of the initial target being fixated and the appearance of the new saccade target in what has become known as the *gap effect* (Braun and Breitmeyer 1988; Fischer and Boch 1983; Fischer and Ramsperger 1984; Kalesnykas and Hallett 1987; Saslow 1967). Using this gap saccade paradigm, Fischer and colleagues described a separate population of saccades that emerged from the saccadic reaction time histograms, which they named *express saccades* because they had latencies of only 80–100 ms in monkey (Fischer and Boch 1983) and 100–130 ms in humans (Fischer and Ramsperger 1984). Gap durations ~200 ms were found to be optimal in demonstrating the gap effect by producing both the shortest latency saccadic reaction times (Braun and Breitmeyer 1988; Saslow 1967) and the highest percentage of express saccades (Mayfrank et al. 1986). The attentional disengagement hypothesis maintains that saccadic reaction times are reduced during this gap paradigm because visual attention can be disengaged from the current point of fixation before the appearance of the visual target (Fischer and Weber 1993; Mayfrank et al. 1986). If this disengagement occurs during the gap, the time saved bypassing this process will be expressed as a decrease in reaction time.

The primate superior colliculus (SC) is critical for saccade control (for review see Sparks and Hartwich-Young 1989). It has been hypothesized (visuomotor hypothesis) that SC visuomotor cells may trigger express saccades if the saccade generating circuit has been disinihibited partially at the time of target appearance (Dias and Bruce 1994; Edelman and Keller 1993; Sommer 1994). This disinhibition is presumed to occur during the gap of no stimuli in the gap saccade paradigm.

Both the attentional disengagement and visuomotor hypotheses predict the existence of neurons in the brain that exhibit a change in their activity level during the gap period, reflecting the disengaging of active visual fixation or the disinhibition of visuomotor cells in the SC, respectively. A subset of cells in the rostral pole of the SC discharges tonically when the eyes are fixated on a target and pauses during saccades (Munoz and Guitton 1989, 1991; Munoz and Wurtz 1992, 1993a; Peck 1989). When these fixation cells are inhibited artificially after microinjection of muscimol, an agonist of the endogenous inhibitory neurotransmitter γ-aminobutyric acid (GABA), monkeys exhibit shorter saccadic reaction times and have difficulty suppressing unwanted eye movements (Munoz and Wurtz 1992, 1993b). In contrast, when fixation cells are activated artificially by electrical stimulation or injection of bicuculline, a GABA antagonist, monkeys exhibit prolonged saccadic reaction times (Munoz and Wurtz 1993b). It has been hypothesized that collicular fixation cells exert powerful inhibition over the brain stem saccade generating circuit and a prerequisite step in saccade generation is the cessation of fixation cell activity (Munoz and Guitton 1989, 1991; Munoz and Wurtz 1993a,b).

We hypothesize that the activity of collicular fixation cells will be reduced during the gap period thereby disinhibiting the saccade generating circuit leading to a reduction in saccadic reaction times. To test this hypothesis, we recorded the activity of fixation cells in the rostral pole of the SC while a monkey generated saccades in the gap paradigm, and we found that fixation cells reduced their rate of discharge during the gap in a manner consistent with producing the gap effect.

METHODS

Single-cell activity was recorded from the rostral SC of a juvenile male monkey (*Macaca mulatta*) weighing 5 kg. All procedures

were approved by the Queen's University Animal Care Committee and complied with the guidelines of the Canadian Council on Animal Care.

Procedures for recording eye movements with the magnetic search-coil technique and single-cell activity from head-restrained monkeys have been described previously (Munoz and Wurtz 1993a). The monkey was trained to perform several saccade and fixation-related tasks, including the gap saccade paradigm. The monkey faced a tangent visual screen 86 cm away. Light emitting diodes (2.0 cd/m^2) were back projected onto the translucent screen. The monkey was required to look at a fixation point (FP) that appeared in the center of the screen and then maintain fixation for 500–1,000 ms. The FP then was extinguished and there was a period of no stimuli (gap) during which the animal was in total darkness before a visual target (T) was presented randomly either 10 deg to the left or right of the FP. Within a block of trials, the gap duration was randomized between 0, 100, 200, 300, 400, 600, and 800 ms. A liquid reward was given if the monkey maintained fixation on the FP until T presentation, at which time, it made a saccade to T and fixated upon it for ≥300 ms. During the intertrial intervals (500–1,000 ms), the screen was illuminated with diffuse white light (1.0 cd/m^2) to prevent dark adaptation.

Behavioral paradigms, visual displays, and storage of data were under the control of a 486 PC computer running a real-time data acquisition system (REX) (Hays et al. 1982). Horizontal and vertical eye and target positions were digitized at 500 Hz. Single-cell discharges were collected at 1 kHz via a window discriminator that produced a pulse for each valid spike that met both amplitude and time constraints. Data were stored on hard disk and analyzed off-line.

Rasters of cell discharge and continuously varying spike density functions (MacPherson and Aldridge 1979; Richmond et al. 1987) were aligned on such events as FP disappearance, T appearance, or saccade onset. We convolved the spike train of each cell with a Gaussian pulse of 10 ms to generate the spike density function for each of the gap durations. This value was chosen because it provided sufficient smoothing of the envelope of cell discharge without losing important details. Using a smaller Gaussian pulse (e.g., 4 ms) added more noise to the shape of the spike density function without altering the overall shape. The discharges of several cells were combined into a population response by first generating the spike density functions for each cell. The waveforms from each cell were grouped according to gap duration and then averaged together to show the activity of the sample of fixation cells during each of the gap intervals. The computation of the mean saccadic reaction time of the monkey for each gap duration was calculated by averaging the reaction times from all trials in which fixation cell activity was recorded during the randomized gap trials. To quantify the change in discharge of each fixation cell during the gap, we computed the average discharge frequency in three separate 100-ms epochs in trials having gap durations of ≥600 ms (Fig. 1*B*). The mean rate of discharge for each fixation cell was obtained for the 100-ms interval immediately before fixation point disappearance (t_1), during the interval 200–300 ms into the gap (t_2), and during the interval 500–600 ms into the gap (t_3). A one-way analysis of variance (ANOVA) was performed for the mean discharge rates of all three intervals to determine whether there was a significant change in the discharge rate of each fixation cell during the gap interval. For those cells with statistically significant differences in their mean values, an all pairwise multiple comparison procedure (Student-Newman-Keuls method) was performed to determine which of the means differed from one another. To determine whether this change in fixation cell discharge was significant for our entire sample during the gap interval, a Kruskal-Wallis ANOVA on ranks for nonnormal samples was carried out on the mean firing rates of each epoch of all the cells followed by an all pairwise multiple comparison procedure (Student-Newman-Keuls method).

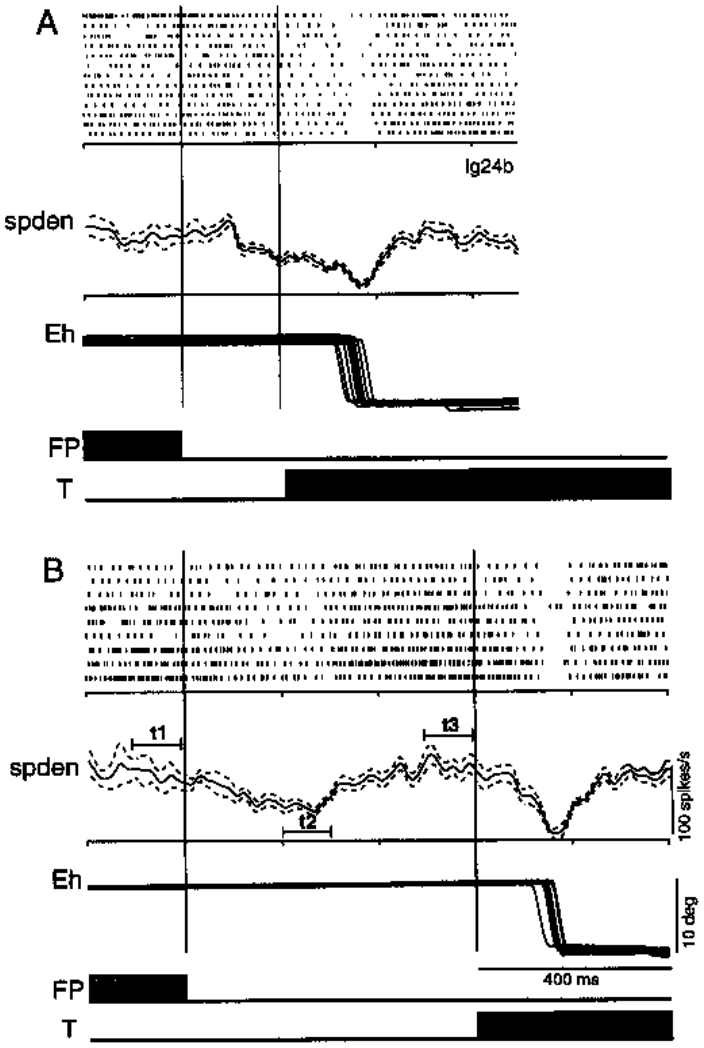

FIG. 1. Activity of a fixation cell (lg24b) located in the right superior colliculus while the monkey performed the gap saccade paradigm. Fixation point (FP) was extinguished 200 (*A*) and 600 ms (*B*) before the appearance of the target (T) presented 10 deg to the left. Individual rasters of cell discharge, the spike density function (spden), and the horizontal eye position traces (Eh) are aligned on FP disappearance (left vertical line). The standard error of the spike density function is represented by the dashed lines. The average discharge rates of fixation cells were computed at 3 times to quantify the extent of the change in activity during the gap: 100-ms interval before FP disappearance (t_1); 200- to 300-ms interval into the gap (t_2); and 500- to 600-ms interval into the gap (t_3).

RESULTS

We recorded the activity of 53 fixation cells in the rostrolateral pole of the SC of one monkey. To be classified as a fixation cell and included in our sample, a cell had to be located 1.5–3.0 mm below the dorsal surface of the rostrolateral pole of the SC and had to possess the following discharge properties as previously described by Munoz and Wurtz (1993a): (1) tonic activity while the monkey fixated a visible target spot, even when this target spot was momentarily removed and the monkey was required to maintain the same eye position, and (2) a pause in discharge when the monkey generated saccades. The activity of a typical fixation

cell is shown in Fig. 1 while the monkey made leftward saccades in trials with gap durations of 200 ms (Fig. 1*A*) and 600 ms (Fig. 1*B*). The cell had a decrease in activity during the gap interval. For trials with longer gap durations (Fig. 1*B*), the activity decreased to a minimum ~250 ms into the gap and then increased again later in the gap. Shortly after target appearance, the monkey generated a saccade that was associated with the characteristic pause in cell discharge (Munoz and Wurtz 1993a).

To quantify the degree to which the fixation cells exhibited this gap-related decrease in discharge frequency, the average firing frequency of each cell was computed for three separate epochs during trials with gap durations of ≥600 ms (see METHODS). Sufficient data was obtained from 20 cells to allow a detailed analysis of the effect of gap duration on fixation cell discharge and saccadic reaction times (see Table 1). Most cells (18/20) decreased their discharge rate 200–300 ms after fixation point disappearance (i.e., $t_2 < t_1$). This reduction was significant for 14 of 20 cells (Student-Newman-Keuls test, $P < 0.05$). Most of the cells (18/20) subsequently increased their rate of discharge later in the gap (i.e., $t_2 < t_3$). Eight of these cells had a significant increase in discharge rate from t_2 to t_3 ($P < 0.05$). In addition, 12 of the cells had a significant decrease in discharge rate between t_1 and t_3 ($P < 0.05$). When the mean discharge rates from all 20 cells were compared, there was a significant difference among the three epochs (Kruskal-Wallis ANOVA for nonnormal samples, $P < 0.001$). Furthermore, an all pairwise multiple comparison procedure revealed that the means of each of the epochs differed significantly from each other (Student-Newman-Keuls test, $P < 0.05$).

TABLE 1. *Quantification of fixation cell activity during the gap saccade paradigm*

Cell	t_1	t_2	t_3
li77c	24 ± 3	24 ± 3	20 ± 2
lj17b	32 ± 3	11 ± 2*†	19 ± 2‡
li71b	36 ± 5	38 ± 3†	51 ± 4‡
li88b	41 ± 8	27 ± 2	30 ± 3
lm18c	43 ± 4	30 ± 2*	36 ± 2
ln38a	44 ± 4	17 ± 1*†	30 ± 2‡
ln15d	52 ± 7	29 ± 2*	33 ± 3‡
ln21c	53 ± 6	30 ± 3*	37 ± 2‡
ll05a	58 ± 4	17 ± 2*†	26 ± 3‡
li57a	54 ± 9	31 ± 4	44 ± 6
ll11c	61 ± 5	36 ± 4*†	62 ± 5
li64a	52 ± 15	30 ± 7	33 ± 6
lg27e	66 ± 5	43 ± 4*	49 ± 3‡
ln25c	70 ± 5	32 ± 5*	42 ± 4‡
li40e	68 ± 7	49 ± 8	42 ± 6‡
lg26b	94 ± 8	61 ± 4*†	99 ± 5
lg24b	95 ± 11	52 ± 4*†	103 ± 9
ln36a	113 ± 17	46 ± 6*	71 ± 8‡
ln43b	119 ± 16	28 ± 4*†	78 ± 10‡
ln22c	128 ± 14	36 ± 6*	42 ± 6‡
mean	65 ± 7	33 ± 3	47 ± 5

Intervals t_1, t_2, t_3 are defined in Fig. 1*B*. There is a statistically significant difference in the median values among the groups (Kruskal-Wallis one way analysis of variance on ranks for nonnormal samples, $P < 0.001$). An all pairwise multiple comparison procedure (Student-Newman-Keuls method) indicated that all of the mean parameters are significantly different from each other ($P < 0.05$). Values are means ± SE in spikes per second. * Statistically significant difference between t_1 and t_2 (all pairwise multiple comparison, Student-Newman-Keuls method, $P < 0.05$). † Statistically significant difference between t_2 and t_3 (all pairwise multiple comparison, Student-Newman-Keuls method, $P < 0.05$). ‡ Statistically significant difference between t_1 and t_3 (all pairwise multiple comparison; Student-Newman-Keuls method, $P < 0.05$).

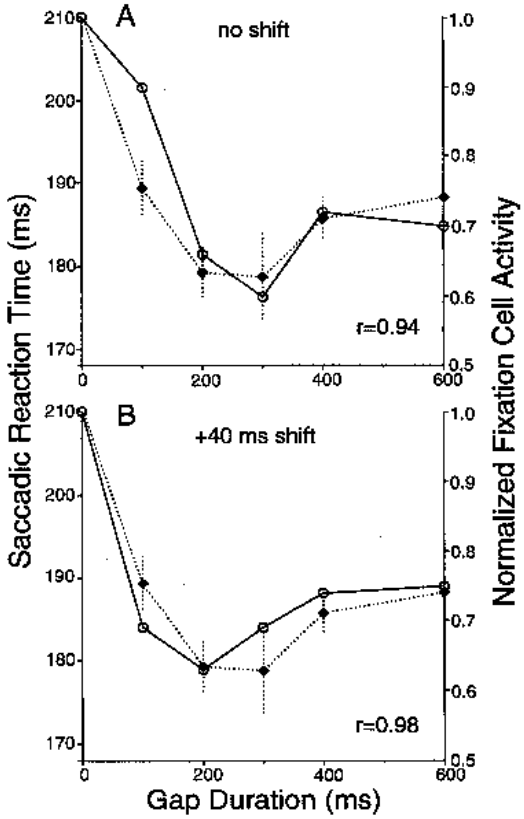

FIG. 2. Time course of the monkey's saccadic reaction times closely followed fixation cell discharge sampled at target appearance (*A*) and 40 ms after target appearance (*B*). Fixation cell activity for each gap duration (○—○) was calculated by dividing the value of the spike density function at T appearance (or T appearance + 40 ms) by the value of the spike density function at FP disappearance (or FP disappearance + 40 ms) for individual cells and then averaging these values together for all 20 fixation cells. The mean saccadic reaction time (mean ± SE bars) for each gap duration (♦ · · · ♦) was calculated by averaging all the individual trials during recording of the same 20 fixation cells. The correlation values were obtained from a linear regression analysis between the fixation cell activity and the saccadic reaction times.

If the collicular fixation cells provide a neural correlate of the gap effect, then the population response should correlate to the monkey's behavior during the gap. Figure 2 shows the close relationship between our sample of fixation cells and the corresponding saccadic reaction times. The filled diamonds, linked by the dotted line, represent the mean saccadic reaction times for each of the different gap durations that were obtained concurrently with the recording of the activity of the sample of 20 fixation cells. The open circles connected by the solid line is a measure of the activity of the sample of fixation cells at T appearance (Fig. 2*A*). The change in fixation cell activity closely followed the time course of the change in the animal's behavior. A linear regression analysis between fixation cell activity and saccadic reaction times revealed a strong correlation ($r = 0.94$). When fixation cell discharge was at its lowest at target appearance (~200–300 ms into the gap), the mean saccadic reaction times were shortest. For gap durations <200 ms

and >300 ms, fixation cell discharge was greater at target appearance and the corresponding saccadic reaction times were also longer.

A more accurate measurement of fixation cell activity as it relates to saccadic reaction times would take in to account the afferent delay for transmission of visual stimuli to the superior colliculus. Because this visual afferent time to the monkey SC is 40–50 ms (Goldberg and Wurtz 1972), we also measured fixation cell activity at T appearance plus 40 ms. Measuring fixation cell activity at this time after target presentation provides an estimate of the level of inhibition being imposed at the instant when the retinal afferent signal reaches the superior colliculus. These results are shown in Fig. 2*B* in which the time of fixation cell activity sampling was shifted forward in time 40 ms for both FP disappearance and T appearance. The main effect of sampling with a 40 ms shift is that it allows time for the FP disappearance to modify the fixation cell's firing frequency at the 100 ms gap duration interval (i.e., compare fixation cell activity at 100 ms gap duration in Fig. 2, *A* and *B*). The correlation between fixation cell activity and saccadic reaction times was improved ($r = 0.98$).

DISCUSSION

We have provided evidence for a neural correlate for the gap effect on saccadic reaction times in the monkey superior colliculus. Collicular fixation cells decreased their rate of tonic activity 200–300 ms into the gap, corresponding to the gap durations that produced the shortest saccadic latencies observed in this study and in close agreement with other studies employing human subjects (Braun and Breitmeyer 1988; Kalesnykas and Hallett 1987; Saslow 1967).

A decrease in activity of some fixation cells was reported previously during a fixation-blink paradigm (see Fig. 5 of Munoz and Wurtz 1993a), in which a fixation target was momentarily extinguished and then reinstated creating a 300- to 600-ms gap period during which the monkey was required to maintain steady fixation. Because the monkey was not performing the gap saccade paradigm, it was not possible to correlate the decrease in activity to saccadic reaction times. The average firing frequency of each fixation cell was computed during the final 200 ms of the 300- to 600-ms gap in the earlier study and not 200–300 ms into the gap that was specific to this study. Sampling outside the 200- to 300-ms boundaries had the effect of diluting the greatest decrease in activity that may be found at this gap interval. However, qualitative examination of individual spike density functions from cells in the previous study (e.g., see Fig. 4 Munoz and Wurtz 1993a) reveals a transient decrease in activity in some fixation cells during the 200- to 300-ms gap interval.

Despite the decrease in saccadic reaction times associated with the introduction of a temporal gap, it is important to clarify that it is unclear whether any express saccades were elicited in this study. Express saccades in the monkey are usually defined as visually triggered saccades with latencies <100 ms (Fischer and Boch 1993), and these were not observed. This leads to the fundamental differences between the gap effect and express saccades that must be stated. The gap effect is the reduction in saccadic reaction times associated with the introduction of a temporal gap between fixation point disappearance and target appearance. This latency reduction is a "global" effect of the paradigm itself in that it occurs irrespective of the subject, previous training, and target amplitude and direction. Express saccades, however, cannot be made by all subjects, and unlike the gap effect, their appearance is highly dependent upon previous training. Express saccades generally are elicited only after extensive training to targets of a particular direction and amplitude and do not extrapolate to other untrained areas of the visual field (Fischer et al. 1984; Rohrer and Sparks 1993). Studies reporting express saccades traditionally have required subjects to execute the gap paradigm at one stereotyped amplitude. In our experiments, the monkey was performing various paradigms with target eccentricities requiring saccades of many different amplitudes and directions that were interweaved with the gap saccade paradigm at 10-deg target locations used for this particular study. This may account for the appearance of the gap effect without a clear presence of express saccades.

Despite these differences, the conditions that produce the gap effect are the same conditions in which express saccades may be elicited. Mayfrank et al. (1986) have shown that the percentage of express saccades as a function of gap duration in humans followed a remarkably similar shape as the activity of fixation cells shown in Fig. 2. These findings, taken in conjunction with the findings of this paper, suggest a causal link between fixation cell activity, the gap effect, and express saccade generation.

Recently, the question of the neurophysiological mechanism(s) that produce express saccades have been debated (Fischer and Weber 1993). Local ibotenic acid lesions in the prelunate gyrus (area V4) of the monkey increased the percentage of express saccades (Weber and Fischer 1990). Express saccades can be made without intact frontal eye fields (FEF) but ablation of the SC, while still permitting saccades, prevents express saccade generation (Schiller et al 1987). Furthermore, patients with prefrontal lesions have shown increased percentages of express saccades (Braun et al. 1992; Guitton et al. 1985; Pierrot-Deseilligny et al. 1991). Dias and Bruce (1994) recently have argued that the FEF may in fact have a role in the generation of the gap effect and express saccades because some cells in the FEF have an increase in their discharge rate associated with the disappearance of the fixation target in the gap task. Visuomotor cells in the deeper layers of the SC discharge bursts of action potentials 45–65 ms after target presentation and 20–25 ms preceding the onset of saccades (Sparks and Hartwich-Young 1989 for review). Most of these cells display only one burst, at the visual response latency, during express saccades (Edelman and Keller 1993), suggestive of a disinhibition of the saccade generating circuit at target appearance.

Both the attentional disengagement hypothesis (Fischer and Weber 1993; Mayfrank et al. 1986) and derivations of the visuomotor hypothesis (Dias and Bruce 1994; Edelman and Keller 1993; Rohrer and Sparks 1986; Sommer 1994) of express saccade generation predict a neural correlate for disengagement of attention and disinhibition of the saccade generating circuit at the time of target appearance, respectively. The observed gap-related decrease in activity of collicular fixation cells can satisfy these requirements. Furthermore, modification of fixation cell activity with injection of

GABAergic drugs has very predictable effects on saccadic reaction times and occurrence of express saccades (Munoz and Wurtz 1992, 1993b). We conclude that the decrease in activity of fixation cells during the 200- to 300-ms gap interval relaxes the proposed powerful state of inhibition that this area normally exerts upon the saccade generating system (Munoz and Guitton 1989, 1991; Munoz and Wurtz 1993a,b). This creates a temporal window during which active visual fixation has been disengaged partially or is "free-floating" (Braun and Breitmeyer 1988). Any visual target presented during this time can be acquired at reduced saccadic reaction times because the process of disengaging active fixation has been at least partially accomplished before target appearance. Express saccades, presumably, would require the same general disinhibition of the SC motor map but, in addition, would require a localized, training-dependent, increase in activity of saccade-related cells during the gap. Possible sources of this increased presaccadic activity could be the visual burst of visuomotor cells in the SC (Edelman and Keller 1993; Rohrer and Sparks 1986), the gap-related discharge of buildup neurons in the intermediate SC (Munoz and Wurtz 1995) or cells in the FEF (Diaz and Bruce 1994). We currently are examining the role of various collicular cells in express saccade generation.

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