

*Research Note***Superior colliculus neurons provide the saccadic motor error signal**

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Summary. Studies of the intermediate layers of the superior colliculus have suggested that it provides a desired change in eye position signal (ΔE) for the generation of saccadic eye movements. Recent evidence, however, has shown that some neurons in these layers may be related to the velocity of saccades. We present single cell recordings from the intermediate layers of monkey superior colliculus that are consistent with the hypothesis that many superior colliculus neurons provide instead a motor error signal, e_m . Our hypothesis about the function of these cells places them inside the local feedback loop controlling the waveform of the saccade.

Key words: Eye movements – Saccades – Superior colliculus – Feedback – Control – Single cell recording

Introduction

Generation of visually guided saccades requires a transformation from visual signals to motor signals. Robinson has proposed a model of saccadic control that uses a local feedback loop to generate this transformation (Robinson 1975). In this model, the input to the local feedback loop is the desired eye position in a craniotopic coordinate frame, i.e., where the eye moves in the orbit. Failure to find this signal in craniotopic coordinates has led to consideration of a model that uses a signal in retinotopic coordinates as input to the local feedback loop (Jürgens et al. 1981).

Figure 1 shows a simplified block diagram of Robinson's local feedback loop as modified by Jürgens et al. (1981). The command ΔE signals how far the eye must move to foveate the target. This is converted to a motor error signal (e_m) by subtracting how far it has already moved during the saccade, $\Delta E'$. In this model, ΔE , $\Delta E'$ and e_m are in motor coordinates, and thus, like retinotopic signals, are independent of orbital position. Note that in Robinson's original model the signal ΔE does not appear. In his model the motor coordinate signal (e_m) is obtained directly as the difference between two signals in craniotopic coordinates: the desired eye position and the efference copy of current eye position.

In a local feedback model, the saccadic waveform is determined solely by the elements within the local feedback loop, whereas the amplitude of the movement is determined by the input. It has frequently been assumed that this input, ΔE , was derived from the intermediate layers of the superior colliculus (SC) (Wurtz and Albano 1980; Sparks 1986). Hikosaka and Wurtz found that muscimol and lidocaine injections in the SC caused a slowing of saccades, as well as a reduction in saccade amplitude (Hikosaka and Wurtz 1985, 1986). Berthoz et al. (1986), in the cat, and Rohrer et al. (1987), in the monkey, have shown that activity in some cells in SC is related to saccadic eye velocity. This suggests that the SC is providing velocity as well as change in eye position information.

Our current study has concentrated on the correlation of the discharge of neurons in the intermediate layers of the SC with the waveform of the saccadic eye movement. While these neurons were active prior to a saccade, their discharge usually declined during the saccade and ended abruptly at about the same time the saccade ended. From these observations, we infer that the superior colliculus activity is

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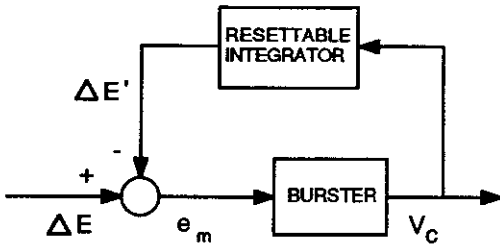


Fig. 1. A simplified block diagram of the local feedback loop for the retinotopic control of saccades. The difference between the desired eye position (ΔE) and the feedback copy of current change of eye position ($\Delta E'$) is motor error (e_m) how much further the eye has to go. The signal e_m is applied to the burst neurons in the brainstem to obtain the velocity command, V_c . The velocity signal is fed back through a leaky integrator, which is reset to zero after each saccade, to monitor current eye position during the saccade ($\Delta E'$)

not only the ΔE signal. Instead, we hypothesize that the activity of some collicular neurons is the motor error signal, e_m .

Methods

A trained monkey was prepared for single cell recording in the superior colliculus as previously described (Hikosaka and Wurtz 1983). Eye movements were recorded using the magnetic search coil technique. Unit data was collected with 1 ms resolution, whereas eye movements and paradigm data were collected with 2 ms resolution. Trains of spike activity were converted to continuous estimates of the probability density function of spike occurrence by substituting gaussians 8 ms wide for each spike. The sum of these gaussians is called the spike density function (Richmond et al. 1987).

A complete, one-dimensional model of the saccadic system (Optican and Miles 1985; Zee et al. 1976) was simulated. This model contained the local feedback loop shown in Fig. 1 and was used to generate the signals ΔE , $\Delta E'$, and e_m .

Results

Figure 2 shows a typical burst neuron recorded in the intermediate layers of the SC. In Fig. 2A the top two traces show horizontal and vertical eye position while the monkey made a saccade to a target light. Below the eye movements are the corresponding individual spike train and spike density function. Figure 2B shows 10 trials from the same cell for saccades to that target, displayed as both a raster and an averaged spike density function. All traces are aligned on the end of the saccade. The presaccadic activity began 26.1 ± 2.2 ms and reached a peak 2.2 ± 0.9 ms prior to the start of the saccade. The activity of the cell declined with the execution of the saccade and had returned to background level by the end of the saccade.

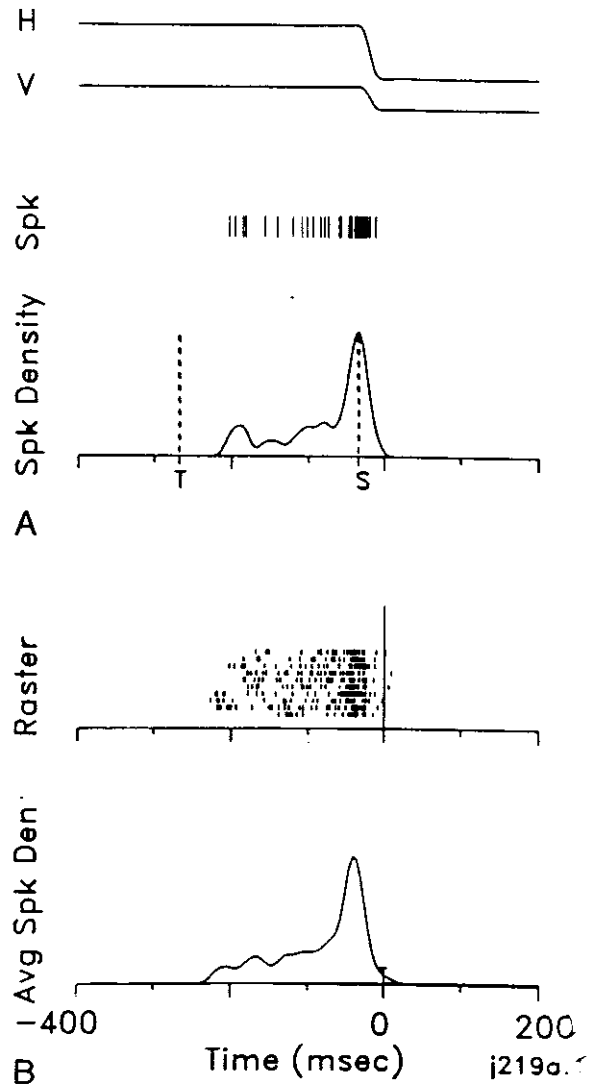


Fig. 2A, B. Discharge of a cell located in the intermediate layers of the superior colliculus during a saccadic eye movement. **A** The sample eye movement shown in the top two traces marked **H** and **V**, was a visually guided saccade to a target 10.5 deg left and 4 deg down from the fixation point and was within the movement field of this cell. The next trace shows the discharge of the cell. The lowest trace shows the spike density function derived by replacing each spike with a gaussian 8 ms wide. The **T** indicates the onset of the target light to which there was a small visual response. Just prior to the onset of the saccade (**S**) there was a large burst of activity which is completed by the time the saccade had come to an end. **B** Neuronal discharge of a series of ten trials to the same target position as in **A**, aligned on the end of the saccade, is shown as a raster (above) and as an averaged spike density function (below)

Figure 3 juxtaposes an example of neuronal activity with simulations of ΔE and e_m . Figure 3A shows the spike density from 90% of peak to the end of the saccade for the same trial as Fig. 2A. Figure 3B shows the simulated ΔE and e_m signals during a saccade of the same amplitude.

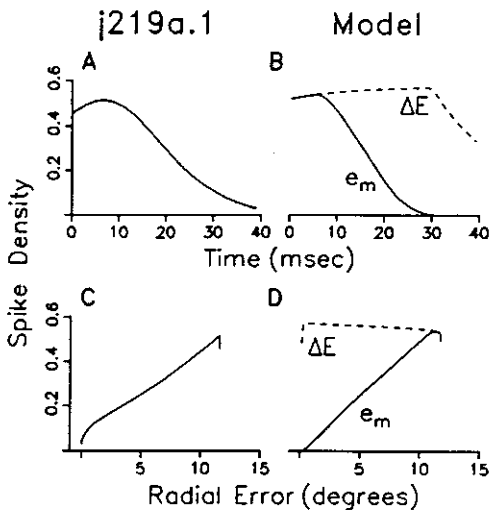


Fig. 3A–D. Time courses (A–B) and trajectories (C–D) for neuronal response and simulations of ΔE and e_m signals. **A** The single spike density function from Fig. 2A is plotted from 90% of its peak value to the end of the saccade. **B** The simulated control signals for a saccade of the same size and direction. The signal ΔE remained constant until the saccade was almost completed while e_m declined smoothly during the movement. **C** The trajectory of spike density vs radial error. Radial error is the distance between the current and final eye positions. Spike density was shifted by 8 ms to align it with the eye movement. In this trajectory, time runs from right to left along the curve. The decline in neuronal activity was proportional to the radial error. **D** The simulated trajectories of ΔE and e_m signals. These signals were also shifted to align them with the eye movement. The trajectory of e_m matches that of the activity in C.

In the simplest model of the saccadic system, shown in Fig. 1, ΔE is the input, and can be considered constant for the duration of the saccade. Simulation of a complete model of the saccadic system (Zee et al. 1976) will produce more realistic eye movements if the input to the inner feedback loop of Fig. 1 is allowed to change gradually. This causes the ΔE trace in Fig. 3 to rise gradually during the eye movement, and then drop off after the saccade is over. The details of this rise and fall are not important to this discussion, and will be deferred to a later presentation.

In either model, the activity of a cell that coded desired change in eye position (ΔE) should remain high throughout the duration of the saccade, otherwise the saccade would not reach the target. In contrast, the response of a neuron which coded motor error (e_m) should decline during the movement, otherwise the saccade would continue past the target. The simulated motor error (e_m) closely approximated the neuronal response.

This relationship was explored further in Fig. 3C–D, which emphasize the relationship of the neuronal response to radial error, the distance of the

eye from the target. Note the striking linear trajectory in Fig. 3C. This implies that spike density declines in proportion to radial error. The simulations (Fig. 3D) demonstrated that the motor error (e_m) was proportional to the radial error, whereas ΔE and radial error were uncorrelated. The small residual activity in the neuron (Fig. 3C) is not considered significant, since it could be eliminated by threshold effects. Such quantitative details are not relevant to this discussion, which concentrates instead on the striking qualitative differences between the simulated ΔE and e_m activities.

We characterized the degree to which a particular cell matched the motor error signal of the model by evaluating the slope of the linear relationship between spike density and radial error. We looked at the response of 26 neurons and have found only four cells whose trajectories had slopes near zero (like ΔE in Fig. 3D). The other 22 neurons had linear trajectories with steep slopes that were consistent with the interpretation that they were encoding the motor error signal e_m .

Discussion

Our hypothesis is that many superior colliculus neurons contribute a motor error signal and are therefore within the local feedback loop that controls saccades. In contrast, most previous studies suggested that the SC was in front of the local negative feedback loop, acting as the source of the ΔE signal, because SC neurons are topographically mapped in relation to the desired change in eye position (Wurtz and Albano 1980; Sparks 1986).

In the original, spatial coordinate version of Robinson's model, the internal signals leading up to saccades must be in craniotopic coordinates, namely desired eye position and the efference copy of current eye position. Keller (1979) suggested that some SC neurons represented the differences of these two signals, motor error, and he noted that this error signal would be reduced to zero with the end of the saccade. Furthermore, he suggested that the quasi-visual cells of Sparks et al. (1977), might fulfill this function. Mays and Sparks pointed out, however, that the quasi-visual cells do not turn off by the end of the saccade (Mays and Sparks 1980). Instead, Sparks (1986) suggested that these neurons may be signaling the command for the motor error, ΔE .

We have now found that a majority of cells in the intermediate layers of SC have a discharge that declines during the saccade in proportion to motor error. Our model explains a number of previously puzzling experimental results. The slowing of eye

movements following muscimol or lidocaine injections in SC (Hikosaka and Wurtz 1985, 1986) would be expected since the SC is inside the feedback loop generating the velocity command (V_c in Fig. 1). Furthermore, the close relationship of intermediate SC neurons to velocity (Berthoz et al. 1986; Rohrer et al. 1987) fits with the placement of these cells in the feedback loop, since the e_m signal is separated from V_c only by the nonlinear response characteristic of the burst neurons (Keller 1974; Van Gisbergen et al. 1981).

If the decline in activity of these neurons in SC is encoding the motor error signal during the saccade, what is the activity during the long build-up before the saccade encoding? One suggestion is that the rapid rise in presaccadic activity is related to the selection of the target. Also, since monkeys are able to make saccades in the absence of the superior colliculus (Wurtz and Goldberg 1972), presumably based on input from the frontal eye fields (Schiller et al. 1980), we would expect to find a similar motor error signal somewhere in the frontal cortex to brainstem pathway.

Our results emphasize that neuronal activity during the saccade allows for a functional classification of collicular neurons. Based on such activity we hypothesize that the superior colliculus provides the dynamic motor error signal, in addition to the goal of the saccade. Thus, the superior colliculus is part of the local feedback loop that determines the waveform of the saccade.

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