Context Contingent Signal Processing in the Cerebellar Flocculus and Ventral Paraflocculus During Gaze Saccades

T. Belton and R. A. McCrea
Department of Neurobiology, Pharmacology, and Physiology, University of Chicago, Illinois 60637

Submitted 4 March 2004; accepted in final form 30 March 2004


INTRODUCTION

Saccadic eye-head gaze shifts consist of rapid eye and head movements in the direction of the gaze shift, followed by a compensatory, rollback eye movement in the opposite direction that stabilizes gaze on the target when the head movement continues after the gaze shift is completed. The premotor signals that produce the saccadic gaze shift arise primarily from saccade-related burst neurons in the reticular formation that project to extraocular motor nuclei and to the cervical spinal cord (Grantly et al. 1993; Hepp et al. 1989; Moschovakis et al. 1996; Scudder et al. 2002). The premotor signals that produce gaze stabilizing eye movements at the end of the eye-head gaze shift arise primarily from cells in the vestibular nuclei that mediate the vestibulocochlear reflex (VOR) (McCrea and Gdowski 2003; McCrea et al. 1996; Phillips et al. 1996; Roy and Cullen 1998, 2001, 2002).

The eye-movement commands generated within brain stem VOR pathways during gaze shifts are complex (McCrea and Gdowski 2003). Most secondary VOR neurons are either insensitive to active head movements or cease firing altogether during saccadic gaze shifts. The insensitivity is due primarily to eye- and head-movement efference copy signals that cancel vestibular signals related to active head movements and produce saccade-related pauses in discharge (McCrea and Gdowski 2003; Roy and Cullen 2002). Near the end of the eye-head saccade, secondary VOR pathways begin producing signals that help stabilize gaze. One class of secondary vestibular neurons, the eye-head-velocity (EHV) neurons, have greater firing rate modulation during eye-head (gaze) saccade-related VOR eye movements than other secondary VOR neurons (Gdowski and McCrea 2000; McCrea and Gdowski 2003). Those cells putatively receive inputs from the cerebellar flocculus region (FLR), which includes the flocculus and the ventral paraflocculus (Lisberger et al. 1994a; Partsalis et al. 1995; Zhang et al. 1995b). The observation suggests the possibility that the cerebellar flocculus region might play an important role in stabilizing gaze immediately after saccades because many secondary EHV vestibular nucleus neurons receive inputs from FLR Purkinje (Pk) cells.

Although FLR Pk cells are largely insensitive to eye-head saccade gaze shifts (Belton and McCrea 1999), their firing behavior during the VOR evoked immediately after gaze saccades has not been examined in detail. In this study, we compared the signals generated by FLR Pk cells during the VOR evoked by gaze saccade-related active head movements to the signals generated during the VOR evoked by passive head movements. We report that FLR Pk cells respond differently to the VOR evoked by saccadic head movements than they do to the VOR evoked by passive head movements. We discuss this different sensitivity to VOR eye movements in relation to the previously described responses of VOR pathway neurons and the role of the flocculus in controlling gaze during saccadic eye-head movements.

METHODS

The methods used for recording and analyzing eye movements and single-unit activity in squirrel monkeys during combined eye-head saccades have been described previously (Belton and McCrea 2000a,b; McCrea and Gdowski 2003). All practices complied with National Institutes of Health principles of laboratory animal care (National Institutes of Health Publication No. 86-23).

Surgical preparation

Two adult squirrel monkeys were prepared for recording both single-unit activity and eye movements. A woven coil of fine, Teflon-
coated wire (Cooner) was sutured to the sclera of one eye for recording eye movements using the magnetic search coil technique. A Plexiglas well was fitted onto the parietal bone for the placement of microelectrodes, and a metal reference pin was affixed to the skull adjacent to the probe insertion site.

**Experimental recording conditions**

Animals were seated in a Plexiglas primate chair atop a vestibular turntable (Inland 832). A harness that was fitted over the shoulders and in front of the trunk inhibited arm-raising and trunk-twist movements. A rod was attached to a keyed metal stud affixed to the occipital bone. The rod was coaxial with the rotational axis of the turntable and positioned within 5 mm of the C1 occipital bone. The rod was made from coated wire (Cooner) was sutured to the sclera of one eye for recording eye movements using the magnetic search coil technique. A Plexiglas well was fitted onto the parietal bone for the placement of microelectrodes, and a metal reference pin was affixed to the skull adjacent to the probe insertion site.

**Location of recording sites**

Recordings were obtained from the flocculus and ventral paraflocculus in a region extending 4 mm caudal from the rostral end of the ventral paraflocculus and 2–3 mm medial from the lateral edge of the FLR. We did not find qualitative differences in Pk cell activity during pursuit or VOR behaviors across the flocculus-ventral paraflocculus border (posterolateral fissure) (Belton and McCrea 2000a). The most salient anatomical landmark during recording was the presence of VIIIth nerve axons 300–500 μm ventral to the cerebellar cortex. In one monkey, the position and orientation of microelectrode tracts was confirmed histologically.

**Data analysis**

**Analysis of Pk cell sensitivity to eye and head movements.** Pk cell sensitivity to eye velocity was assessed from the records of sinusoidal ocular pursuit (0.5 Hz, 40°/s). Their sensitivity to head velocity was assessed from the records made during sinusoidal WBR (0.5 Hz, 40°/s) in which the monkey suppressed the VOR by fixing a head stationary target. Cycles where the gaze was not within 2.5° of the visual target were discarded. Records from 20–100 selected cycles (mean = 26 at 0.5 Hz) were concatenated, desaccaded, cycle-averaged, and fit with sinusoidal functions. An iterative fitting technique was used to eliminate low firing frequency responses that deviated significantly from linearity during periods of inhibitory saturation (Chen-Huang et al. 1997). A significant minority of the Purkinje cells exhibited nonlinear responses during sinusoidal pursuit and VOR suppression that was not due to inhibitory saturation. In these cells, an estimate of sensitivity to eye and head movements was confined to the portion of the cycle in which the response was linearly related to the stimulus (Belton and McCrea 2000a; Lisberger et al. 1994a; Miles et al. 1980; Noda and Warabi 1982).

**Analysis of Pk cell responses during gaze saccade-related VOR.** Pk cells were selected for analysis that had relatively large responses during the VOR evoked by passive whole body rotation or during ocular pursuit. Pk cells that generated bursts of spikes during ocular saccades or had very large eye position-related changes in firing rate were excluded from analysis due to the difficulty of separating saccade-related eye movement signals from signals related to the compensatory eye movement at the end of eye-head saccades.

Records of eye-head (gaze) saccades were grouped according to direction and amplitude and whether the saccades were made to or in the absence of a visual target. Only gaze saccades that had peak head velocities >20°/s were included in the analysis. Saccade records were averaged after aligning the records on the peak compensatory eye velocity at the end of the gaze shift. For the saccades made to a visual target, gaze position at saccade-end was required to be within 2° of the target for inclusion in the average.

The firing rate modulation recorded during the epoch of VOR at the end of gaze saccades was quantified using linear regression analysis. For each saccade-related VOR epoch, the firing rate observed during VOR eye movements was regressed against the modulation predicted from the cell’s sensitivity to eye velocity measured during ocular pursuit, to eye position during steady fixation, and to head velocity during VOR cancellation. Firing rate modulation during saccade-related VOR eye movements was measured after subtracting the mean baseline firing rate within a 100-ms window that ended 30 ms before saccade onset. The eye-velocity sensitivity during the VOR related to gaze saccades was also calculated using linear regression analysis,
where for each saccade the firing rate modulation during the VOR epoch was regressed against recorded eye velocity.

For purposes of illustration in this paper, averages of 7–14 saccades are aligned on peak compensatory eye velocity at the end of the gaze shift. The spike rasters associated with each saccade are illustrated above the averaged response histogram.

**RESULTS**

The 48 FLR Pk cells chosen for analysis in this study were all strongly related to eye velocity during smooth pursuit eye movements (mean sensitivity re eye velocity during pursuit was 1.96 spikes/s per °/s, ±0.90). The sample included 27 eye-velocity (EV) Pk cells (mean eye-velocity sensitivity = 2.2 ± 1.2 spikes/s per °/s) and 21 eye-head velocity (EHV) Pk cells (mean eye velocity sensitivity = 1.6 ± 0.55 spikes/s per °/s). All of the cells were modulated during the VOR evoked by passive WBR. This modulation was stronger in EV Pk cells (mean sensitivity to VOR eye velocity: 2.0 spikes/s per °/s) than in EHV Pk cells (mean VOR eye velocity sensitivity: 0.86 spikes/s per °/s) because the latter cells receive vestibular inputs that tend to cancel signals related to eye velocity during the VOR. Some Pk cells were more sensitive to passive VOR eye movements in one direction than the other. In those cells, separate estimates of passive VOR eye velocity sensitivity in each direction were calculated.

**FLR Pk cells insensitive to VOR evoked by active head movements during gaze saccades**

Most (42/48) FLR Pk cells were insensitive to eye and head velocity during the VOR eye movements evoked during both ipsiversive and contraversive saccade-related head movements. This was true whether the saccades were spontaneously generated or made to the visual target. Figures 1 and 2 show the responses of an EV Pk cell and an EHV Pk cell during VOR eye movements related to active head movements during gaze saccades.

The EV Pk cell illustrated in Fig. 1 was sensitive to ipsilateral eye velocity (1.54 spikes/s per °/s) during ocular pursuit (Fig. 1A). When the monkey suppressed its VOR during passive whole body rotation by fixating a head stationary target (Fig. 1A), the cell’s firing rate modulated in relation to the unsuppressed ipsilateral VOR eye velocity. During passive whole body rotation, the cell’s firing rate was as sensitive to the eye velocity generated by the VOR as it was to eye velocity during pursuit (Fig. 1A). The absence of firing rate modulation during the VOR evoked by gaze saccade-related active head movements is shown in Fig. 1B, 1 and 2. The cell was neither excited during gaze saccade-related VOR eye movements in its on direction (Fig. 1B) nor inhibited during VOR eye movements in its off direction. The traces superimposed on the firing rate histograms are the expected responses if the cell had been insensitive to saccadic eye movements but had been as sensitive to VOR eye movements generated during, and immediately after, gaze saccades as it was during passive whole body rotation. Obviously compensatory VOR eye movements could not be accurately estimated during saccadic gaze shifts due to the large saccadic eye movement, and so the predicted responses were calculated based on the assumption that active head movements evoke VOR eye movements with a gain of 1.0 during the saccadic gaze shift.

Figure 1C plots the peak change in firing rate observed during the VOR related to individual gaze saccades versus the predicted change in firing rate expected from the cell’s sensitivity to eye velocity during pursuit and passive VOR. The expected response if the cell was equally sensitive to the VOR during passive and active head movements is represented by the dashed line. The linear regression of the observed sensitivity to saccade-related VOR is also shown (—). The slope of the regression was −0.03, which was not significantly different from zero. The probability that this cell was equally sensitive to passive and active VOR eye movements was <0.01 (Student’s t-test, P < 0.01).

The EHV Pk cell illustrated in Fig. 2 was sensitive to ipsilateral head velocity during VOR cancellation (0.61 spikes/s per °/s, Fig. 2A) as well as to eye velocity during ocular pursuit (1.43 spikes/s per °/s, Fig. 2A). The cell was, however, like most EHV Pk cells in the FLR, more sensitive to eye velocity than it was to head velocity. The traces superimposed on the firing rate histograms in Fig. 2 are the responses expected based on the cells sensitivity to eye movements during pursuit and to passive head movements during suppression of the VOR. Although the two signals tend to cancel one another during the VOR, the cell’s firing rate was still modulated during the VOR evoked by passive whole body rotation (Fig. 2A) due to the much stronger sensitivity to eye velocity. On the other hand, the cell’s firing rate was poorly modulated during gaze saccade-related VOR eye and head movements in both the eye movement on direction (2B1) and off direction (2B2). Figure 2C plots the peak change in firing rate observed during the VOR related to individual gaze saccades versus the predicted change in firing rate expected from the cell’s sensitivity to eye velocity during pursuit and passive VOR. The slope of regression (0.06) was not significantly different from zero.

Pk cells that were insensitive to VOR eye movements evoked by active head-on-trunk rotations during gaze saccades were sensitive to VOR eye movements evoked by passive head-on-trunk rotation. Figure 3A shows the responses of an EHV Pk cell during high-frequency passive head-on-trunk rotation at 3.5 Hz. The cell’s firing rate was modulated, although the response was smaller than that predicted from its responses during pursuit and VOR cancellation (trace superimposed on firing rate histogram), possibly because the cell received neck proprioceptive inputs or was sensitive to the absence of a visual target (Belton and McCrea 2000a; Gdowski et al. 2001). Figure 3B shows the cell’s response during on direction saccade-related VOR eye movements the temporal duration of which was comparable to a half cycle of the passive head-on-trunk rotation stimulus (3.5 Hz) shown in Fig 3A. Figure 3C plots the change in firing rate of the cell during individual saccade-related VOR epochs versus predicted change in firing rate based on its sensitivity to passive head-on-trunk rotation. The slope of regression (0.024) was not significantly different from zero. Similar analysis was carried out in 13 other Pk cells. The mean sensitivity of all 14 Pk cells to VOR evoked by high-frequency passive head-on-trunk rotation was 0.39 ± 0.27 spikes/s per °/s, whereas their sensitivity to gaze saccade-related VOR evoked by comparable head movements was not significantly different from zero.
FIG. 1. Eye-velocity (EV) Purkinje (Pk) cell that was insensitive to gaze saccade-related vestibuloocular reflex (VOR). The cell was sensitive to smooth eye velocity during ocular pursuit ($A_1$) as well as smooth eye velocity during the VOR ($A_2$). During VOR cancellation (VORC, $A_3$), the cell’s firing rate was related to unsuppressed VOR eye velocity. The models superimposed on the firing rate histograms are based on the cell’s sensitivity to eye velocity during ocular pursuit. $B_1$: averaged records of 8 contraversive gaze saccades when the compensatory VOR eye movements immediately after those saccades were in the cell’s on direction. Top: horizontal eye, head and gaze position (ipsiversive is upward). Middle: horizontal eye, head, and gaze velocity. Bottom: spike rasters related to individual saccades and average firing rate histogram. The model superimposed on the average firing rate histogram assumes the cell was sensitive to VOR eye velocity alone and not to ocular saccade-related eye velocity. $B_2$: averaged records of 8 ipsiversive gaze saccades when the compensatory eye movements were in the cell’s off direction. $C$: peak change in firing rate observed during VOR of individual gaze saccades is plotted vs. the predicted change in firing rate. The response expected if the cell was equally sensitive to VOR evoked by passive whole body rotation and to VOR evoked by saccadic head movements. The slope of the best linear fit to the observations (—) was not significantly different from zero. $T_v$, turntable velocity; $H_v$, head velocity; $E_v$, eye velocity; $G_v$, gaze velocity; $E_p$, eye position; $H_p$, head position; $G_p$, gaze position.
FIG. 2. Eye-head velocity (EHV) Pk cell that was insensitive to gaze saccade-related VOR. The cell’s firing rate was related to smooth eye velocity during ocular pursuit (A1). During VOR cancellation (VORC, A2) the cell’s firing rate was related to ipsilateral head velocity. The cell’s response during the VOR evoked by passive whole body rotation (A3) was similar to that predicted from its sensitivity to pursuit eye velocity and VORC head velocity (trace superimposed on response in A3). B, I and 2, shows the Pk cell’s lack of responsiveness during gaze saccade-related VOR eye movements in the on direction (1) and off direction (2). Top: average eye, head, and gaze position recorded during 8 (B1) and 7 (B2) saccades. Middle: average eye, head, and gaze velocity. Bottom: spike rasters related to individual saccades and average firing rate histogram. The model superimposed on the average firing rate histogram represents the response expected if the cell’s activity was related to head movements and VOR eye movements but not to saccadic eye movements. C: peak change in firing rate observed during VOR of individual gaze saccades is plotted vs. the predicted change in firing rate. - - - , the response expected if the cell was equally sensitive to VOR evoked by passive whole body rotation and to VOR evoked by saccadic head movement. The slope of the best linear fit to the observations (—) was not significantly different from zero. Abbreviations are the same as in Fig. 1.
Pk cells sensitive to gaze saccade-related VOR eye movements

Six EHV Pk cells generated bursts of spikes immediately after gaze shifts when the saccadic head movement produced VOR eye movements to the cell’s direction. The responses of one of them is shown in Fig. 4. The cell was sensitive to ipsilateral eye velocity during smooth pursuit eye movements (Fig. 4A1) and to ipsilateral head velocity during VOR cancellation (Fig. 4A3). During passive WBR, the firing rate of the cell was also strongly modulated (Fig. 4A1) because its sensitivity to VOR eye velocity was greater than its sensitivity to head velocity. Near the end of contraversive gaze saccades it generated a burst of spikes (Fig. 4B1). The burst began 81 ± 6 ms after the onset and 55 ± 12 ms prior to the end of saccades and continued as long as the active head movement and the compensatory eye movements were present. Because the EHV Pk cell was not sensitive to ocular saccades, the burst was presumably related to the active head movement component of the gaze shift and the VOR eye movements generated at the end of the saccade (defined in this case as the time at which the eye began to move opposite in direction to the saccade). The lines superimposed on the average firing rate histograms in Fig. 5B are the predicted firing rate of the cell based on its sensitivity to eye and head velocity during ocular pursuit and
FIG. 4. An example of 1 of the minority of EHV Pk cells that were sensitive to VOR eye movements after gaze saccades. The cell’s firing rate was sensitive to ipsiversive eye velocity during smooth pursuit (A1) and ipsiversive head velocity during VOR cancellation (A2) and was modulated in phase with ipsiversive eye velocity during the VOR evoked by passive whole body rotation (A3). After contraversive gaze saccades (B1), the cell generated a burst of spikes during ipsiversive VOR eye movements. The cell was not inhibited during contraversive VOR eye movements followed gaze saccades (B2). Traces superimposed on firing rate histograms are the responses expected based on the cell’s sensitivity to eye velocity during ocular pursuit and to head velocity during VOR cancellation. C: peak change in firing rate observed during VOR at the end of individual gaze saccades is plotted vs. the predicted change in firing rate. ——, the response expected if the cell was equally sensitive to VOR evoked by passive whole body rotation and to VOR evoked by saccadic head movements. Separate regressions (—) were done for ipsiversive, on direction VOR eye movements (positive values) and for contraversive, off direction VOR eye movements. See text for further description. Abbreviations are the same as in Fig. 1.
VOR cancellation. Each of the six EHV cells that were sensitive to gaze saccade head movements exhibited similar bursts. On average the beginning of the burst was 1.0 ± 110 ms (SD) after the end of the gaze saccade (which was the beginning of "rollback" or VOR eye movements). The latency to the beginning of VOR eye movements was, on average, 76 ± 11 ms after the onset of the gaze saccade.

Cells that generated bursts of spikes related to on direction VOR eye movements after contralateral active head movements were not inhibited when the VOR eye movements were to the cell’s nonpreferred direction. For the cell illustrated in Fig. 4B2, the expected reduction in firing rate during postsaccadic contralateral, or off direction VOR eye movements, is represented by the trace superimposed on the average firing rate histogram.

Figure 4C plots the change in the cells’ firing rate observed during the VOR during individual gaze saccades versus the predicted change in firing rate based on its sensitivity to eye velocity during pursuit and head velocity during VOR cancellation. Equal responsiveness during passive and active head movements is indicated by the dashed line. The slope of the linear regression for saccade-related VOR for 29 saccades in the cell’s on direction was 1.3, although the correlation between expected and actual peak firing rate was weak ($R^2 = 0.43$). The slope of the regression for 17 saccades in the cell’s off direction was −0.17, which was not significantly different from zero.
Similarity of Purkinje cell gaze saccade-related responses in the presence and absence of visual targets

FLR Pk cells are often more sensitive to eye and head movements in the presence of a visual target than when a target is absent (Belton and McCrea 2000a; Fukushima et al. 1996; Ghelarducci et al. 1975). However, during saccade-related VOR, most FLR Pk cells were insensitive to the compensatory eye movements regardless of whether saccades were directed to a visual target or not. Figure 5 shows the firing behavior of an EHV Pk cell during the VOR evoked by passive WBR (Fig. 5A) and during saccade-related VOR generated in the absence (Fig. 5B1) and in the presence (Fig. 5B2) of a visual target. The traces superimposed over the firing rate histograms are the responses expected based on the cell’s sensitivity to eye and head movements during ocular pursuit and VOR cancellation. The cell was insensitive to VOR eye movements related to gaze saccades both in the presence and in the absence of visual targets. Similar observations were made in 34 other Pk cells.

Population sensitivity of FLR Pk cells to saccade-related VOR eye movements

Considered as a population, FLR Pk cells were insensitive to saccade-related VOR eye movements. Figure 6A is a cumulative histogram of the responses of all the FLR Pk cells during individual saccade-related VOR eye movements. Pk cell response to each saccade-related VOR eye movement was calculated by linear regression of the change in firing rate versus predicted change in firing rate. Each value plotted is the estimated relative sensitivity of an individual Pk cell during a single saccade. Values <1.0 occurred when the change in firing rate was less than predicted. Values near zero correspond to no change in firing rate. Negative values were obtained when the cell’s response was opposite in direction to that predicted. The mean relative sensitivity of FLR Pk cells during the 1,710 saccades included in this analysis was 0.07 ± 0.36. The thickness of the hatched bar plotted at 1.0 represents the average cycle-to-cycle variance (1 SD) in Pk cell responses during passive WBR.

Figure 6B plots the average sensitivity of each of the 48 FLR Pk cells to active saccade-related VOR eye movements versus their predicted sensitivity to passive VOR eye movements. Each point represents the results of analysis of a single Pk cell like those shown in Figs. 1C–5C. On and off direction saccade-related VOR responses were calculated separately because some Pk cells exhibit directional differences in their responses to VOR eye movements (Belton and McCrea 2000a) (see above). The dashed diagonal line indicates equal sensitivity to VOR during passive and saccade-related head movements. Estimated in this manner, the sensitivity of FLR Pk cells to saccade-related VOR was attenuated by 97%. Considered together, the analyses in Fig. 6, A and B, suggest that the output of the FLR is attenuated by ~95% during on direction saccade-related VOR eye movements compared with its output during VOR eye movements evoked by passive rotation of the head. The small, residual sensitivity was due primarily to the six EHV Pk cells described above (indicated as square symbols in the plot). During off-direction saccade-related VOR eye movements the population response was not significantly different from zero.

Discussion

The FLR is an important part of the neural substrate that helps stabilize images on the retina by smoothly moving the eyes in the direction of expected image motion (Lisberger and Fuchs 1978; Noda and Warabi 1986; Noda et al. 1981; Stone and Lisberger 1990). The firing rates of most Purkinje cells in the cerebellar flocculus region are modulated during the VOR when it is evoked by passive movement of the head (Belton and McCrea 2000a; De Zeeuw et al. 1995; Fukushima et al. 1996, 1999; Hirata and Hightstein 2001). In the
context contingent signal processing in the FLR during gaze saccades.

**Role of the FLR in controlling the VOR during passive and active head movements**

FLR Pk cells generate smooth pursuit eye-movement command signals and vestibular signals that are essential for modifying the gain of the VOR during passive head movements, but the output of the FLR is apparently less important for this function during active head movements. Inactivation of the FLR strongly affects the ability to suppress or enhance the VOR during passive head movements (Belton and McCrea 2000a; Takemori and Cohen 1974; Waespe and Cohen 1983; Zee et al. 1981; Zhang et al. 1995b) but has little effect on this ability during active smooth tracking head movements (Belton and McCrea 2000b). Saccade-related head movements are probably at least as common as passively evoked head movements, and the function of the VOR would appear to be equally important in each circumstance. But the comparatively weak modulation of the firing rate of FLR Pk cells during active head movements during gaze saccades suggests that this region of the cerebellum is less involved in maintaining gaze stability during and immediately after active gaze shifts than it is when the head is passively perturbed.

The FLR provides visual, and vestibular sensory reafferent feedback signals, oculomotor feedback signals and possibly predictive signals that help stabilize gaze during passive head perturbations (Kettner et al. 2002; Miles et al. 1980; Stone and Lisberger 1990). These signals presumably would also be useful for producing gaze stability immediately after saccadic head movements. So why is the flocculus less active during the VOR eye movements following gaze saccades? One possibility is that different neural circuits regulate the gain of the VOR in different behavioral circumstances. The flocculus appears to be primarily involved in modifying VOR eye movements produced by passive perturbations of the head. Other cerebellar circuits, e.g., the vermis-fastigial pathway, may play the crucial role in modifying the VOR during active head movements.

**Conclusion**

The output of the flocculus and ventral paraflocculus plays an essential role in modifying signal processing in VOR pathways so that images of interest remain stable on the retina. This image stabilizing signal is highly dependent on the behavioral context and is called on primarily when external forces unrelated to self-generated eye and head movements are the cause of image instability.

**GRANTS**

This work was supported by National Institutes of Health Grants EY-08041 and DC-05056.

**REFERENCES**


Moschovakis AK, Scudder CA, and Hightstein SM. The microscopic anatomy and physiology of the mammalian saccadic system. Prog Neurobiol 50: 133–254, 1996.


