Excitability Changes in Cat Lateral Geniculate Cells during Saccadic Eye Movements

Abstract. *The excitability of lateral geniculate cells to orthodromic volleys decreased during saccadic eye movements. This decrease was caused by retinal impulses generated by a quick displacement of the image of the visual field associated with eye movements. This may be a mechanism for saccadic suppression.*

Psychophysical studies have shown that the visual threshold is substantially increased during saccadic eye movements (*1*). This effect is known as "saccadic suppression" and is accompanied by a diminution or a modification of visually evoked responses (*2*). Similar changes in evoked responses have also been reported in experimental animals (*3*), with suppression of orthodromic volleys at the lateral geniculate nucleus (LGN) (*4*). Because these modifications are associated with eye movements, they have been frequently regarded as a manifestation of a corollary discharge (*5*) that may arise in the oculomotor system and signal information on eye movements.

However, MacKay (*6*) has questioned whether eye movements are necessary for a saccadic suppression, since he has demonstrated a threshold elevation for flashes that occurs whenever the visual field is abruptly displaced. He suggests that displacement of the retinal image itself generates a transient "neural disturbance" which may interfere with subsequent detection of the test flash.

In this study, by testing single unit responses during eye movements in chronically prepared cats, we found that the excitability of LGN cells to orthodromic volleys was markedly decreased. We could further demonstrate that it was caused by impulses arising in the retina which was excited transiently by a quick displacement of the image during an eye movement. We propose that this may be a mechanism for saccadic suppression. As suggested by MacKay (*6*) from psychophysical observations, the saccadic suppression would then be independent of any corollary mechanism.

A total of 73 units was tested in 18 experiments on four cats. About 1 week prior to the first experiment, a stimulating electrode was inserted in the optic tract under pentobarbital anesthesia, and a vertical cylinder was fixed under an opening in the skull for later insertion of microelectrodes into the LGN. At the same time, two transverse tubes were attached to the skull by acrylic cement. During testing, the cat's head was held rigidly in the stereotaxic frame by inserting two pairs of bars into the transverse tubes. The cat sat quietly on a hammock and its eyes were free to move. Neuronal discharges were recorded with stainless steel microelectrodes, insulated with Insl-X, driven through the intact dura. Details of the preparation and the recording procedures have been described (*7*).

Eye movements were recorded by electrooculograms in both horizontal and vertical directions with silver–silver chloride electrodes (*8*) implanted in the bone surrounding the orbit.

The firing probability of LGN cells to stimulation of the optic chiasm was evaluated during an eye movement by triggering a stimulator from potential shifts in the horizontal electrooculogram and altering delays in the stimulus pulse. The testing shock was a rectangular 50-μsec pulse with an intensity selected for each cell by finding the intensity which gave a firing probability of 80 percent in room light while the eyes were steady. Experiments were performed in three visual environments: in a patterned field with stationary grating, in diffuse illumination, and in complete darkness. The stationary pattern was a vertically oriented grating with dark stripes 2° wide (5 cd/m^2) separated by light stripes 4° wide (70 cd/m^2). Luminance over the diffusely lit screen was approximately uniform at 50 cd/m^2.

Changes in firing probability of an LGN cell following eye movements in the three visual environments are shown in Fig. 1A. As indicated by reduced probability, excitability of the cell decreased notably for about 200 msec when the eyes moved in the patterned field (*P*). The probability was evaluated from 20 consecutive spike responses. Examples of the original records tested at a stimulus delay of 30 msec are shown in Fig. 1B.

A prominent decrease in cellular excitability occurred in all classes of relay cells of the LGN. At least three

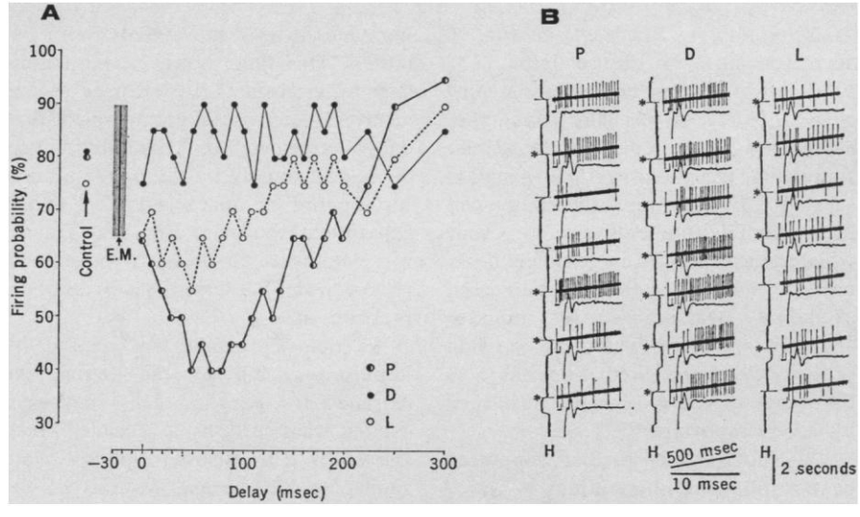

Fig. 1. Changes in firing probability of an LGN cell upon stimulation of the optic chiasm during the period following saccadic eye movements. (A) Firing probability was tested by changing the delay of the stimulus pulse at 10-msec intervals (the testing order was shuffled randomly) in the patterned field (*P*, half-black circles), in complete darkness (*D*, black circles) and in diffuse light (*L*, open circles). Firing probability was calculated from 20 consecutive spike responses. *E.M.*: The onset of eye movements measured retrograde from the triggering moment (delay 0). The vertical line represents the average of 30 events and the shaded area shows 1 standard deviation of the time variation. (B) Original film records tested at a stimulus delay of 30 msec in three visual environments. The spike responses are shown by vertical deflections in both faster (10 msec) and slower (500 msec) horizontal sweeps, and the potential shifts in the horizontal electrooculogram (*H*) are displayed horizontally as a moving spot and recorded continuously on a running film. To avoid triggering of the stimulator by subsequent eye movements occurring within 2 seconds of the first movement, a gating circuit inhibited activation of the stimulator until an eye movement occurred more than 2 seconds after the preceding pulse. Positive responses are marked by stars on the left.

classes of cells have been found (*9*). As seen in the slower sweeps (500 msec) in Fig. 1B, the firing rate of this cell in complete darkness (*D*) was higher than in diffuse light (*L*). When the eyes moved in the patterned field, the cell showed sustained response, either an increased or a decreased firing, reflecting the luminance of stripes in the grating. The sustained response continued until it was finally interrupted by the next eye movement. Cells showing this type of sustained response sient response to saccades. They do not show transient response to saccades. They have receptive fields near the area centralis and latencies to chiasmatic stimulation are usually longer than 1.5 msec. Another class of cells is called T units (*9*). They show transient response to saccades. They do not change firing level in response to the luminance of the visual field. Their latencies are usually shorter than 1.5 msec. It seems most probable that S- and T-units correspond to X- and Y-cells (*10*), respectively. There are also cells of intermediate properties showing mixed responses, that is, transient responses to saccades and sustained responses to local luminance (M-units) (*9*).

Excitability of the cell in Fig. 1 decreased also in diffuse light (*L*). This, however, was due to an incomplete *Ganzfeld*. It was later found that black bars at the edge of the screen stimulated peripheral retinal ganglion cells (*9*). Furthermore, the effect seen in the lit environments did not occur when the eyes moved in complete darkness. This indicates that the decreased excitability was caused by impulses arising in the retina where the ganglion cells would be excited transiently as the image of the grating was displaced by an eye movement.

This notion was further supported by the following observations in which the visual field was moved abruptly while the eyes were stationary. The image motion which might occur during an eye movement was simulated by reflecting the grating image from a front surface mirror attached to the spindle of the galvanometer actuated by the horizontal electrooculogram potentials previously recorded on magnetic tape. The projected grating pattern was then moved in front of the stationary eyes at the speeds and amplitudes identical with retinal image motion as might be occurring during saccades. The same procedures as for

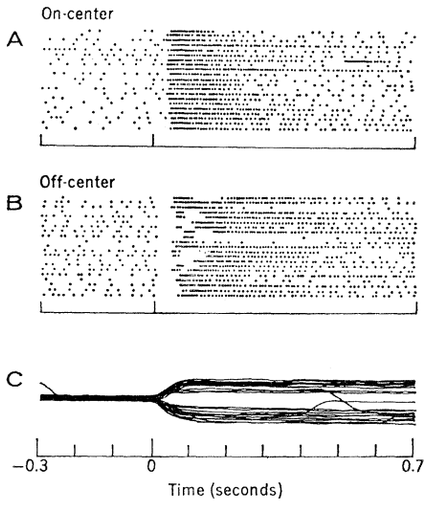

Fig. 2. Transient responses of an on-center (A) and an off-center (B) T-retinal ganglion cell to saccadic eye movements scanning the stationary grating pattern. Neuronal discharges were recorded in the optic tract, a few millimeters posterior to the optic chiasm. They are represented by horizontal rows of dots aligned to the start of saccadic eye movements. The on- or off-center property of the receptive field center was assessed by testing the cell with three parallel light stripes moving across the screen. (C) Horizontal electrooculograms showing 21 consecutive saccades.

spontaneous eye movements were repeated. This time, however, the stimulator was triggered by changes in the recorded electrooculogram which produced target motion. Excitability was depressed notably by the image motion for a period comparable to that of the experiment shown in Fig. 1A. Thus it was demonstrated that eye movements per se were not essential for the depression.

As the depression was caused by impulses arising in the retina, we searched for ganglion cells that were excited transiently by a saccade. Three classes of cells whose responses were similar to S-, T-, and M-units of the LGN were found also in the retinal ganglion cells. Responses of an on-center and an off-center T-ganglion cell to saccades are shown in Fig. 2. They are aligned to the start of saccades (time 0), and spikes are represented by horizontal rows of dots. The time course of saccades is shown in (C), where the superimposed electrooculograms show 21 consecutive saccades. On-center cells usually showed simple bursts (A), and off-center cells tended to show an initial suppression followed by a prolonged burst (B).

Information on quick motion of retinal image, coded on the transient responses of T-ganglion cells, would be transferred to T-geniculate cells. The fact that the response properties of S-, T-, and M-units are preserved both in the retina and LGN may indicate that the channels for these direct excitatory impulses are well separated in the LGN. The depression was found in all classes of LGN relay cells; therefore, pathways for the inhibitory impulses seem to be much less specifically organized (*10*). It may be possible that LGN cells receive inhibitory influence from the same pool of interneurons, as is frequently suggested (*11*).

The suggestion has been made that oculomotor-visual integration occurs in the LGN (*12*). However, we failed to detect any signs of the corollary discharge during saccadic eye movements in our alert cats. As most previous discussions were based on observations in either encéphale isolé cats or in sleeping cats, the disagreement might depend on states of alertness. As suggested by Bizzi and Brooks (*13*), it may be possible that subtle modification occurs in the functional connections between the LGN and the oculomotor centers during rapid eye movements in sleep. A similar explanation may be given for the different observations in spontaneous firing of LGN cells. The majority of LGN cells discharged in bursts with rapid eye movements in sleep (*14*). However, after testing approximately 450 LGN cells, we did not find any cells which changed activity with eye movements in complete darkness, although all LGN cells showed either transient or sustained changes, or both, in their firings when the eyes moved in the light (*9*). This confirms the recent observation that most LGN cells in alert rhesus monkeys fail to discharge with eye movements in complete darkness (*15*).

Another possible basis for the disagreement might relate to observations during different types of eye movement. When eye movements were elicited by direct or indirect stimulation of the brainstem oculomotor centers, it is possible that such stimulation itself causes not only changes in levels of alertness, but also that aberrant volleys might impinge upon the LGN cells. During eye movements induced, for example, by vestibular stimulation, nystagmic impulses might be transferred to the LGN. However, it is not clear whether such impulses are com-

parable to any corollary discharges which may subserve normal visual perception. During the period of such involuntary eye movements, we are aware that the perceived world is never stabilized, suggesting that a corollary mechanism would no longer be operating effectively. In summary, it would appear that in normal conditions of active eye movements, the LGN of the cat does not participate in oculomotor-visual integration. The saccadic suppression may simply reflect the decreased excitability of LGN cells that would be caused by impulses conveyed by axons of the retinal ganglion cells.

HIROHARU NODA
W. ROSS ADEY
Department of Anatomy and
Brain Research Institute,
University of California,
Los Angeles 90024

References and Notes

1. R. W. Ditchburn, *Optica Acta* **1**, 171 (1955); F. C. Volkmann, *J. Opt. Soc. Am.* **52**, 571 (1962); P. L. Latour, *Vision Res.* **2**, 261 (1962); B. L. Zuber and L. Stark, *Exp. Neurol.* **16**, 65 (1966); G. W. Beeler, *Vision Res.* **7**, 769 (1967); A. Starr, R. Angel, H. Yeetes, *ibid.* **9**, 195 (1969); F. C. Volkmann, A. M. L. Schick, L. A. Riggs, *J. Opt. Soc. Am.* **58**, 562 (1968); L Mitrani, N. Yakinoff, St. Mateef, *Vision Res.* **10**, 411 (1970).
2. E. G. Gross, H. G. Vaughan, Jr., E. Valenstein, *Electroencephalogr. Clin. Neurophysiol.* **22**, 204 (1967); J. A. Michael and L. Stark, *Exp. Neurol.* **17**, 233 (1967); R. Chase and R. Kalil, *Vision Res.* **12**, 215 (1972).
3. H. Collewijn, *Vision Res.* **9**, 803 (1969); J. A. Michael and L. Stark, *Electroencephalogr. Clin. Neurophysiol.* **21**, 478 (1966).
4. E. Bizzi, *J. Neurophysiol.* **29**, 861 (1966); H. Kawamura and P. L. Marchiafava, *Arch. Ital. Biol.* **106**, 141 (1968); L. J. Malcolm, I. S. C. Bruce, W. Burke, *Exp. Brain Res.* **10**, 283 (1970); T. Ogawa, *Brain Res.* **38**, 211 (1972).
5. R. W. Sperry, *J. Comp. Physiol. Psychol.* **43**, 482 (19→ E. von Holst and H. Mittelstaedt, *Naturwissenschaften* **37**, 464 (1950); H. L. Teuber, in *Handbook of Physiology*, section 1, *Neurophysiology*, J. Field, H. W. Magoun, V. E. Hall, Eds. (Williams & Wilkins, Baltimore, 1960), vol. 3, pp. 1595–1668.
6. D. M. MacKay, *Nature* **225**, 90 (1970).
7. H. Noda, R. B. Freeman, Jr., B. Gies, O. D. Creutzfeldt, *Exp. Brain Res.* **12**, 389 (1971).
8. Similar to those used for the monkey by P. H. Schiller and M. Stryker, *J. Neurophysiol.* **35**, 915 (1972).
9. H. Noda and W. R. Adey, in preparation.
10. B. G. Cleland, M. W. Dubin, W. R. Levick, *J. Physiol. (Lond.)* **217**, 473 (1971); K.-P. Hoffmann, J. Stone, S. M. Sherman, *J. Neurophysiol.* **35**, 518 (1972).
11. W. Singer and O. D. Creutzfeldt, *Exp. Brain Res.* **10**, 311 (1970); W. Singer, E. Pöppel, O. D. Creutzfeldt, *ibid.* **14**, 210 (1972); K.-P. Hoffman, J. Stone, S. M. Sherman, *J. Neurophysiol.* **35**, 518 (1972).
12. E. Bizzi, *J. Neurophysiol.* **29**, 861 (1963); H. Kawamura and P. L. Marchiafava, *Arch. Ital. Biol.* **106**, 141 (1968); M. Jeannerod and P. T. S. Putkonen, *Exp. Brain Res.* **13**, 533 (1971); R. Corazza and C. Lombroso, *Electroencephalogr. Clin. Neurophysiol.* **19**, 323 (1970).
13. E. Bizzi and D. C. Brooks, *Arch. Ital. Biol.* **101**, 666 (1963).
14. E. Bizzi, *J. Neurophysiol.* **29**, 1087 (1966); H. Sakakura, *Jap. J. Physiol.* **18**, 23 (1968).
15. U. Büttner and A. F. Fuchs, *J. Neurophysiol.* **34**, 127 (1973).
16. Supported in part by U.S. Air Force contract AFOSRF 44620-70-C-0017, NIH grant 1 RO1 EY01051-01A1, and NASA grant 05-007-195.

9 October 1973

Torpor in an Andean Hummingbird: Its Ecological Significance

Abstract. *Field studies on an Andean hummingbird showed that nocturnal torpor occurs more frequently and lasts longer in the winter. Energy depletion does not seem to cause this yearly torpor cycle, and a photoperiodically controlled rhythm that enables the birds to automatically conserve energy in early evening for possible metabolic expenditures required later in the winter night is suggested.*

Daily torpor, in which the body temperature is lowered to near ambient, is known to occur in several groups of small endotherms, including some birds, bats, and rodents. Many workers have conducted laboratory studies on the physiology of torpor (*1, 2*), and some have calculated its savings in energy (*3*). However, because of the difficulty of studying organisms in their roosts or burrows, few field data are available that have been systematically gathered on torpid animals on a yearly basis. Nocturnal torpor may be employed occasionally in the field by energy-stressed incubating female hummingbirds, indicating that torpor may be used by normal, healthy individuals in certain emergency situations (*4*). However, I know of no study on birds that reveals the importance of torpor under natural conditions to males and nonbreeding females and to the population as a whole during the nonbreeding season. The need for this kind of information has been noted (*2, 4, 5*). This report shows that torpor occurs naturally in the field in both sexes of the Andean hillstar hummingbird, *Oreotrochilus estella estella,* that often its occurrence is not related to any detectable emergency conditions, and that its frequency and duration are seasonal.

In 1968 and 1970, I conducted field studies on several populations of *O. estella* in the southern Peruvian Andes between 3800 and 4300 m elevations. This species roosts in caves at night, clinging to surface irregularities in the walls and ceilings with their claws (*6*). Individuals are thus easily located and studied in their roosts with little disturbance. I located individuals by flashlight at night, determined their metabolic condition (nontorpid, entering torpor, torpid, and arousing from torpor) by observing breathing rates, erection of plumage, and general alertness, and took ambient and body temperatures (T_a and T_b, respectively) with a Schultheis quick-recording thermometer whenever feasible. When the difference between ambient and body temperatures was likely to be large, the bulb of the thermometer was first warmed in the hand before insertion in the bird. Unless a bird was just entering torpor, its T_b was usually only 0° to 2°C higher than T_a except when T_a was lower than 7°C (Fig. 1). I also noted the time of night, the sex (by plumage), and the identification number of the individual. I checked on each individual one to three times a night and recorded "torpor" or "nontorpor" for each night. During both winter and summer, evening observations were made between ½ hour after darkness and 23:00, and early morning observations were made between 02:00 and 05:00 (first light occurred at 05:15 to 05:30). Observations were made during the rainy summer months of September to March and the dry winter months of June to August. No individual was observed in both seasons. Within a season each individual was observed one to eight different nights, except one summer individual that was observed 15 nights and one winter individual that was observed 12 nights. Sample sizes and results are given in Fig. 2A. The summer individual observed 15 nights was torpid twice and nontorpid 13 nights; the winter individual observed 12 nights was torpid all 12 nights.

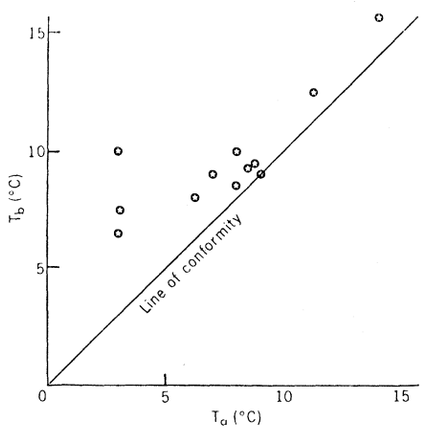

Fig. 1. Body temperatures of torpid *O. estella* at various ambient temperatures; T_a, ambient temperature; T_b, body temperature.

In summer and winter there were no differences between the sexes in the