presense of [^{35}S]uridine 5′-(α-thio) triphosphate (>1200 Ci/mmol) (DuPont, Biotechnology Systems) at 5 × 10^7cpm/ml. Hybridization and washing conditions were as recommended by the membrane supplier. Before autoradiography, tissue prints were stained with India ink (*7*). Autoradiograms were made on Kodak Tmax 400 film exposed for 10 days at −70°C and developed in Tmax developer for 11 min at 24°C. A detailed description of the tissue print and autoradiography methods used in this study will be presented elsewhere (B. A. McClure and T. Guilfoyle, in preparation).

15. Tissue localization of RNAs by hybridization of antisense probes to tissue prints was suggested by J. E. Varner.

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Gating of Retinal Transmission by Afferent Eye Position and Movement Signals

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Vision in most vertebrates is an active process that requires the brain to combine retinal signals with information about eye movement. Eye movement information may feed forward from the motor control areas of the brain or feed back from the extrinsic eye muscles. Feedback signals elicited by passive eye movement selectively gate retinal outflow at the first relay, the dorsal lateral geniculate nucleus. The gating predominantly facilitates retinogeniculate transmission immediately after eye movement and inhibits transmission when a new steady-state eye position is achieved. These two gating effects are distributed in a complementary fashion across the dorsal lateral geniculate nucleus such that the spatiotemporal activity profile could contribute to object detection and localization.

THE EXTRAOCULAR MUSCLES (EOM) of mammals are richly innervated by stretch receptors (*1, 2*) that, when activated, produce phasic bursts of activity (*2, 3*) that may interact with visual signals in neurons in brain regions that receive monosynaptic retinal input (*4*). Interaction of phasic eye movement signals from the EOM with retinal signals has also been reported in higher centers such as visual cortex (*5*). However, information is not available on the effects of EOM-mediated tonic eye position changes and phasic eye movements on early visual processing in functionally identified neurons with respect to their positions on the retinal map. Thus, it is not known how the nervous system could use such information to facilitate spatial localization of objects. This is of particular interest in light of reports that feed-forward signals may be sufficient to signal eye position during performance of certain oculomotor tasks (*6*).

We have examined the effects of passive changes in eye position and eye movement on retinogeniculate transmission in the cat. General surgical methods, anesthesia, and electrophysiological recordings were as described (*7, 8*). The experimental procedure is shown in Fig. 1. The same visual stimulus was repeatedly delivered to the right eye while the steady-state position or time of a brief phasic movement of the left eye was randomly varied by a computer (*9*). The left eye was prevented from receiving visual stimulation by occlusion with an opaque contact lens and an intravitreal injection of tetrodotoxin (TTX) (*10*).

Extracellular recordings made with fine-tipped micropipettes (*7*) were obtained from 209 single X or Y (*11*) neurons of the A layer of the left dorsal lateral geniculate nucleus (LGN_d) (contralateral to the eye being visually stimulated but ipsilateral to the eye being moved). Thus, LGN_d layer A received a modulated retinal signal only from the stationary eye. Two types of experiments were performed: a tonic eye position experiment in which the steady-state position of the left eye was varied (n = 133 neurons) and a phasic eye movement experiment in which a brief movement of the left eye occurred at various times before or after the visual stimulus onset (n = 76 neurons). A subset of the neurons that showed significant changes in their visual response after eye movements (n = 12) was also tested for

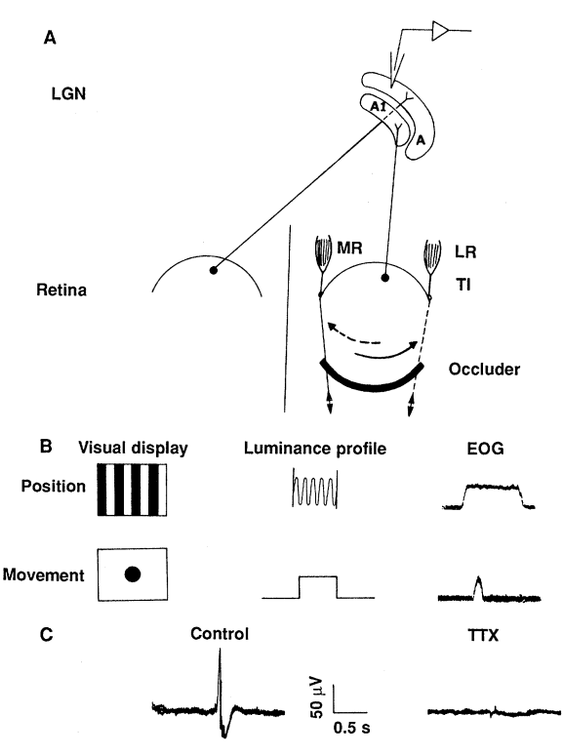

Fig. 1. Experimental procedure. (**A**) Schematic top view of the cat's eyes and relevant projection of the retinas to the LGN (animal looking toward bottom of figure). The locations of the medial rectus (MR) and lateral rectus (LR) muscles and their tendons of insertion (TI) on the globe of the left (moving) eye are indicated. The right eye, which remained stationary and received visual stimulation, was fitted with a contact lens. By application of the correct spectacle lens, the right retina was made conjugate with a display monitor positioned at a viewing distance of 57 cm. The left eye, which was moved but received no visual stimulation, was fitted with an opaque contact lens occluder and received an intraocular injection of TTX in most cases. (**B**) Visual stimuli (delivered to right eye) and eye movement signals (of left eye). For the eye position experiment, the visual display (a drifting grating pattern) and electrooculogram (EOG) indicating a steady-state change in eye position are indicated. For the eye movement experiment, the visual display (an abrupt onset circular spot positioned within the receptive field of the LGN_d neuron) and brief (75 ms) phasic eye movement are indicated. Neuronal responses were collected for six eye positions or ten times of eye movement in a randomized, interleaved fashion. Blocks of five trials were collected for each eye position or time of movement in an interleaved fashion. The new position or time was selected by the computer with a new randomization from a table of preselected values during a 1-s pause. During the pause period, the visual display remained spatially and temporally homogeneous. (**C**) Visually evoked potential recorded from optic nerve of left (moving) eye before and after TTX injection into vitreous humor.

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directional preference. Neuronal responses were analyzed for changes in both the number and frequency of action potentials.

Responses of an LGN_d X cell to identical visual stimuli for two different eye positions are shown in Fig. 2, A through D. A significant reduction in the response of the neurons to the visual stimulus was seen when the response at the deviated (10° nasal) position (Fig. 2, C and D) was compared to that at the control (5° temporal) position (Fig. 2, A and B). At the deviated position, the number of action potentials per trial was reduced by 40%, whereas the mean peak firing rate was reduced by 53%. This difference in both the measures as a function of eye position was significant when compared between individual positions and over the full range of eye positions tested [$P < 0.001$, analysis of variance (ANOVA) test]. A conservative measure ($P < 0.05$, trend analysis), as evaluated by both the number of action potentials and firing rate, was applied in order to evaluate the spatial extent over which these effects occurred (*12*). Examples of these relations for several individual LGN_d neurons are shown in Fig. 3A (*13*). A reduction in the visual response when the eye position was deviated in either direction from rest was the predominant effect observed. The mean gain or slope across the range of eye positions for the population of neurons studied is shown in Fig. 3A. This represents a mean reduction of 2.2% (±0.4% SEM) in visual response per degree change in eye position. However, if only the range of 5° deviation [more within the range of typical saccadic eye

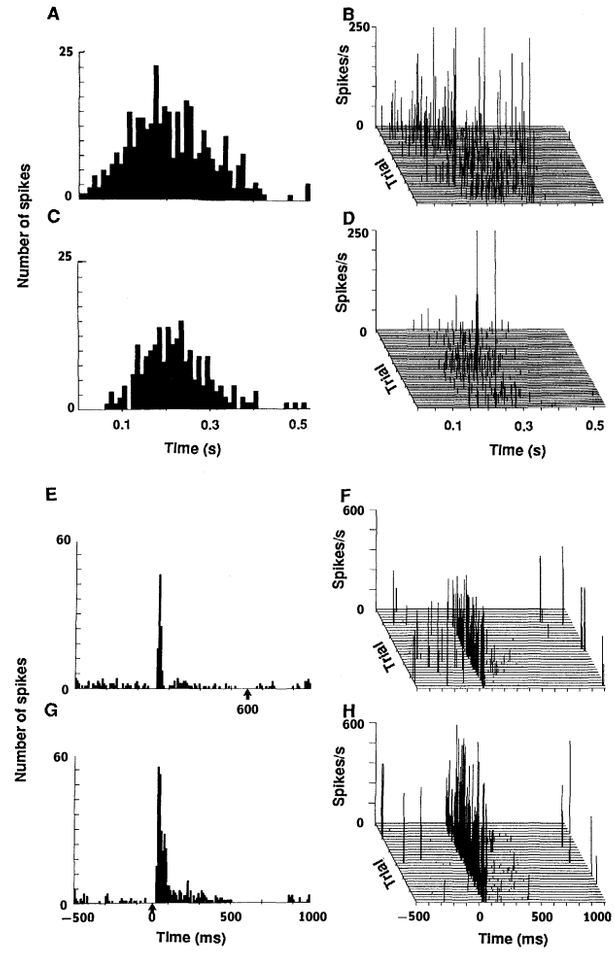

Fig. 2. The effect of changing eye position (A through D) and eye movement (E through H) on visual responses of two LGN_d neurons. The effect of eye position is illustrated for an X cell with an off-center receptive field at an eccentricity of 10° from central vision. The effect of eye movement is illustrated for a Y cell with an on-center receptive field at an eccentricity 4° from central vision. Left column (A, C, E, and G) shows time histograms of accumulated action potentials for 50 (eye position) or 30 (eye movement) trials. Bin width, 5.0 ms. Right column (B, D, F, and H) shows firing rate plots on a trial-by-trial basis. Eye position effects on visual response were evaluated at six positions, whereas eye movement effects were evaluated at ten times. In each case, the appropriate variable (eye position or time of eye movement) was varied in a randomized interleaved fashion. (**A** through **D**) Eye position: Responses for two of the eye positions tested [(A and B) control, 5° temporal position; (C and D) deviated, 10° nasal position] for which the difference in the cell's visual response was greatest are illustrated. A significant reduction in the number of action potentials [212 in (C) versus 354 in (A)] and the peak firing rate [75.0 ± 25.0 Hz in (D) versus 160.0 ± 25.0 Hz in (B)] was observed. (**E** through **H**) Eye movement: Responses for two of the ten times of eye movement are indicated [(E and F) control, 600 ms after visual stimulus onset; (G and H) maximal effect, 0 ms or coincident with visual stimulus onset]. Visual stimulus onset was always at time 0, and duration of visual stimulus was 500 ms. Arrows in (E) and (G) indicate time of phasic eye movement (200° per second, 75 ms duration, 15° amplitude). The visual response was facilitated as demonstrated by an increase in the number of action potentials [284 in (G) versus 121 in (E)] and by the increase in firing rate [455.0 ± 33.0 Hz in (H) versus 275.0 ± 27.0 Hz in (F)] when the eye movement was initiated at time 0. Similar but less dramatic increases in the visual response occurred for other times of eye movement initiated within 50 ms of the visual stimulus onset (Fig. 3).

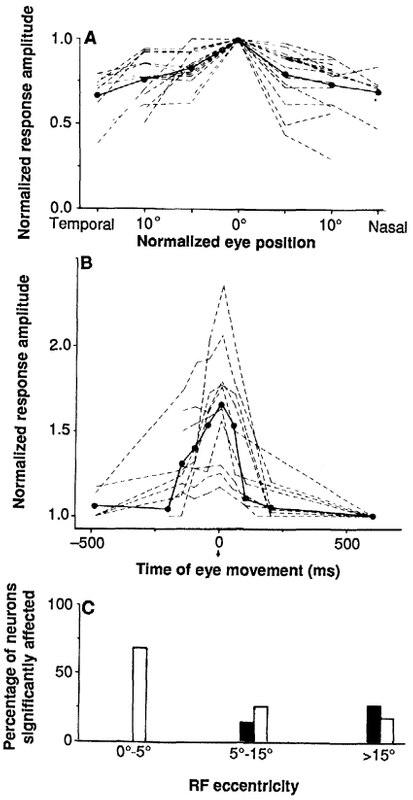

Fig. 3. Summary of magnitude and distribution of eye position (A and C) and eye movement (B and C) effects on visual responses of LGN_d neurons with respect to their RF location. (**A**) Summary of the relation of visual response amplitude to eye position for all of the individual cells ($n = 13$) that had a significant reduction ($P < 0.05$, ANOVA) in both the number of action potentials and the firing rate for the visual response when the eye was positioned to either side of an optimum position [quadratic trend (*12*)]. Ten other neurons (not shown) were significantly affected by eye position but had other types of spatial profiles [such as those fitted by linear trends (*12*)]. The visual response was normalized with respect to the optimum position (which was 0° for 10 of 13 of these neurons and 5° nasal or temporal for the other 3 neurons). The solid line represents the mean function. (**B**) Summary of the temporal range for eye movement facilitation for all ten neurons with visual responses that were significantly ($P < 0.05$, ANOVA) and maximally facilitated when the phasic eye movement onset was coincident with the visual stimulus onset (indicated by arrow). (Fifteen other neurons had visual responses that were also significantly affected by appropriately timed eye movements, but these effects varied in optimum time and polarity of response and are not shown.) The visual response amplitude is the number of action potentials occurring in the 250-ms block beginning at the visual stimulus onset. The control visual response amplitude is taken when no eye movement occurs until 600 ms after visual stimulus onset. The mean facilitatory temporal range for the ten neurons is shown by the solid line. (**C**) Distribution of LGN_d neurons (both X and Y cells combined) with significant ($P < 0.05$, ANOVA by both number of action potentials and firing rate criteria) eye position (solid bars, $n = 23$ of 133 tested) or movement (open bars, $n = 25$ of 76 tested) effect on visual response with respect to range of grouped RF eccentricities.

movements in cat (*14*)] from the position of maximal response is considered, the average slope is 4.0% (±0.5% SEM) reduction per degree. Such a relation of visual response magnitude to eye position has also been observed for neurons in the parietal cortex of alert monkeys (*15*).

The responses of an LGN_d Y cell to identical visual stimuli when a phasic eye movement occurred at two different times are shown in Fig. 2, E through H. The number of action potentials and the peak firing rate were significantly greater ($P < 0.001$, ANOVA test, 135% and 65% increase, respectively) when a brief temporal movement (*16*) of the ipsilateral eye occurred coincident with the visual stimulus onset (Fig. 2, G and H) than in the control condition when the eye movement occurred 600 ms after the visual stimulus onset (Fig. 2, E and F). The specificity of this effect for the visual response of the neuron is indicated in Fig. 2E. At 600 ms after visual stimulus onset the eye movement had no effect on the neuron's spontaneous rate of discharge. This selective gating of the visual response with lack of an effect on the spontaneous discharge of the neurons was observed for 88% (22 of 25) of the affected neurons. Moreover, no effects of eye position changes or movement were seen for the visual responses of retinal ganglion cell axons ($n = 24$), implying that the selective gating occurred at the retinogeniculate synapse (*17*). The time window for eye movement–induced facilitation of the visual response is shown in Fig. 3B. The average profile had a 60% increase in the visual response when the phasic eye movement was coincident with visual stimulus onset.

The magnitude and likelihood of the eye position and movement effects on visual responses are unevenly distributed with respect to cell type and receptive field (RF) eccentricity in the LGN_d (Fig. 3C). Although both types of effects are more likely to occur for X than for Y cells ($P < 0.01$, χ^2 test), the complementary distribution of the two effects with respect to RF eccentricity is more striking (*18*). Inhibition of the visual responses as a function of deviations in eye position is stronger and more likely ($P < 0.01$, χ^2 test) to occur for neurons with RF eccentricities >15° (Fig. 3C). Facilitation of visual responses by phasic eye movement is stronger and more likely ($P < 0.01$, χ^2 test) for neurons with RFs within 5° of central vision (Fig. 3C).

On the basis of our results, the effects of changes in eye position or appropriately timed eye movements on the visually elicited activity profiles of layer A of the right and left LGN_d are shown in Fig. 4. The EOM signals initially enhance the visual responses in and near central vision for a brief period after the eyes have centralized a novel visual target, potentially providing a postsaccadic alerting mechanism.

The spatial activity profiles of the A layers of the LGN_d with the eyes in a deviated steady-state position but with identical visual stimulation are shown in Fig. 4B. The nonuniform reduction in the spatial activity profile that occurs with increasing angle of gaze could contribute to target localization in a head-centered coordinate system by combining retinal and afferent eye position signals. Any one LGN_d neuron could not reliably encode target location because of many possible combinations of eye position and visual stimulus strength. However, the nonuniform distribution of the eye position effect with respect to the retinal map would cause the response of the entire population of LGN_d neurons to differ for the same visual scene viewed at differing angles of gaze. If only the strength (contrast) of the textured visual stimulus was reduced without changing eye position, the spatial activity profile would simply be a copy of the control response (Fig. 4A) with a negative offset. The directional selectivity (*16*) of the eye movement effect could contribute the vector component necessary for target localization. Alternatively, the directional component may come from a corollary discharge (*6*) of motor signals that initiate eye movement.

Averaging of activity across a neuronal population has been suggested as a mechanism for the generation of eye movements to different positions [saccade generation by the primate superior colliculus (*19*)] and for the encoding of the head-centered positional informaiton in higher cortical areas [primate cortical area 7a (*20*)]. Such a mechanism provides for accurate motor localization of the eyes to a target with minimization of the noise effects due to a single neuron's variability (*19*). We suggest that a similar mechanism may contribute to localization of visual targets in early visual processing.

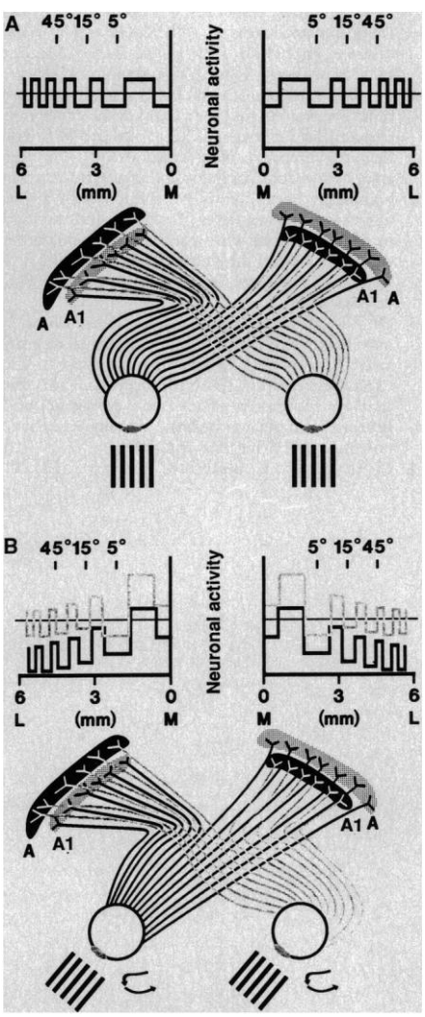

Fig. 4. Schematic representation of the differential effects of varying eye position and time of eye movement on the activity profile in layer A of the right and left LGN_d. The drawings of the two eyes illustrate the known projection pattern from the nasal and temporal hemiretinas to contralateral LGN_d layer A and the ipsilateral LGN_d layer A1, respectively. The shaded pupils indicate the position of the eyes, and the alternating grating patterns modulated in one dimension represent a textured visual simulus that spans the entire retina. The plots above the LGN_d represent the predicted activity profile across the mediolateral dimension of layer A of each LGN_d for an appropriate subset of neurons (for example, on-center neurons). The solid straight lines in (A) and (B) represent the mean activity level, and the representations of the corresponding position of the retinas with respect to the representation of central vision (the area centralis) are given in degrees. The varying activity profile expands as the representation of central vision is approached in accordance with the known magnification of the retinal surface on the LGN_d. (**A**) (Control) The predicted activity profiles with the eyes at rest and straight ahead. (**B**) Effect of eye position and movement. The predicted activity profiles in response to the identical visual stimulus at the same retinal location when the eyes are held in a position deviated from rest (solid lines on graph) or when the eyes have just completed (within 50 ms) a phasic movement (stippled lines on graph). The change in eye position causes an inhibition such that the likelihood and magnitude of the inhibition increases at more peripheral receptive field representations, whereas the appropriately timed eye movement causes a nonuniform facilitation such that the likelihood and magnitude of the facilitation decreases at more peripheral RF representations in the LGN_d. Abbreviations: L, lateral; and M, medial.

REFERENCES AND NOTES

1. S. Cooper and P. M. Daniel, *Brain* **72**, 1 (1949); H. Hosokawa, *Tex. Rep. Biol. Med.* **19**, 405 (1961); R. M. Alvarado-Mallart and M. Pincon-Raymond, *Tissue Cell* **11**, 567 (1979). Although the EOMs of

most mammals lack typical neuromuscular spindles found in other skeletal musculature and do not have a myotatic reflex [E. L. Keller and D. A. Robinson, *J. Neurophysiol.* **34**, 908 (1971)], they have a high density of sensory nerve terminals including palisade end organs.
2. S. Cooper, P. M. Daniel, D. Whitteridge, *J. Physiol. (London)* **113**, 463 (1951).
3. S. Cooper and M. Fillenz, *ibid.* **127**, 400 (1955); P. Bach-y-Rita and F. Ito, *ibid.* **186**, 663 (1966).
4. V. C. Abrahams and P. K. Rose, *J. Neuophysiol.* **38**, 10 (1975); C. Batini and G. Horcholle-Bossavit, *Prog. Brain Res.* **50**, 345 (1979); I. M. L. Donaldson and R. A. Dixon, *Exp. Brain Res.* **38**, 245 (1980); I. M. L. Donaldson and A. C. Long, *J. Physiol. (London)* **298**, 85 (1980); H. Enomoto, M. Matasumura, J. Tsutsui, *Neuro-ophthalmology* **13**, 48 (1983); W. Guido, W. L. Salinger, C. E. Schroeder, *Exp. Brain Res.* **70**, 417 (1988).
5. P. Buisseret and L. Maffai, *Exp. Brain Res.* **28**, 421 (1977); P. Buisseret and E. Gary-Bobo, *Neurosci. Lett.* **13**, 259 (1979); J. A. Ashton, A. Boddy, I. M. L. Donaldson, *Neuroscience* **13**, 635 (1984).
6. B. L. Guthrie, J. D. Porter, D. L. Sparks, *Science* **221**, 1193 (1983); O. Bock and G. Kommerell, *Vision Res.* **26**, 1825 (1986); A. L. Graves, Y. Trotter, Y. Fregnac, *J. Neurophysiol.* **58**, 816 (1987); M. J. Steinbach and D. R. Smith, *Science* **213**, 1407 (1981); A. Fiorentini, N. Berardi, L. Maffei, *Exp. Brain Res.* **48**, 113 (1982).
7. M. J. Friedlander, C.-S. Lin, L. R. Stanford, S. M. Sherman, *J. Neurophysiol.* **46**, 80 (1981); J. S. Tootle and M. J. Friedlander, *ibid.* **56**, 523 (1986).
8. Adult cats were initially anesthetized with 3% halothane delivered in a mixture of N_2O and O_2 (1 : 1) for all surgical procedures. Anesthesia was maintained during electrophysiological recording with a 70 : 30 mixture of N_2O and O_2 and intravenous alphaxalone (1 to 2 mg kg^{-1} $hour^{-1}$) (Glaxovet) or sodium pentobarbital (2 to 4 mg kg^{-1} $hour^{-1}$) (Abbott) in 5% lactated Ringer solution, and paralysis was maintained with gallamine triethiodide (20 mg kg^{-1} $hour^{-1}$) (Davis and Geck) plus *d*-tubocurarine chloride (0.25 mg kg^{-1} $hour^{-1}$) (Squibb). No differences in results were noted between experiments with either method of anesthesia. Animals were mechanically ventilated with expired CO_2 pressure regulated at 3.8 to 4.2%; body temperature was controlled with a feedback blanket at 38.0°C; heart rate and blood pressure were monitored with an intra-arterial cannula in the abdominal aorta. Heart rate and mean blood pressure were kept between 150 and 190 beats per minute and 90 to 110 mm-Hg, respectively. After any increase in these values, whether it was spontaneous or in response to periodic application of mechanical stimuli (as noted by continuous monitoring of heart rate and blood pressure), we supplemented the intravenous anesthesia. Animals were maintained in a stereotaxic frame with a custom-designed apparatus for passively manipulating eye position by computer control.
9. The tendons of insertion of the rectus muscles were moved by application of force applied tangential to the globe from a vertex in front of the eye. The eye rotated about a vertical axis in the horizontal plane. The duration, velocity, and amplitude of the eye movements were controlled by a computer-driven optical scanner as verified by electrooculogram recordings and repeated verification of the position of retinal landmarks of the moving left eye by reverse ophthalmoscopy [R. Fernald and R. Chase, *Vision Res.* **11**, 95 (1971)] during the initial calibration procedure of each experiment.
10. TTX blocks the voltage-dependent Na^+ channels in the membranes of the retinal ganglion cells, thus preventing the initiation of action potentials. Therefore, even if the opaque contact lens on the moving eye allowed any differential illumination of the retina during eye movements, no signal reaches the brain from the retina of the moving eye. This was verified by recording the visually evoked mass response of the left optic nerve before and after TTX injection, thus ensuring that no retinal signals were elicited in the moving eye (Fig. 1). Although the direct monosynaptic projection of the retinal ganglion cell axons to the LGN_d is monocular, with right and left eye afferents segregated into different cell layers in the LGN_d, we took the precaution of injecting TTX into the moving eye because indirect binocular effects, particularly inhibitory ones, may be mediated polysynaptically into LGN_d neurons [K. J. Sanderson *et al.*, *Exp. Brain Res.* **13**, 178 (1971)].
11. S. M. Sherman and P. O. Spear, *Physiol. Rev.* **62**, 738 (1982). Cells were classified as X or Y by a standard battery of electrophysiological tests including (i) linearity of spatial summation within the receptive field as evaluated by counterphase of sinusoidal gratings at appropriately high spatial frequency and (ii) latency of response to electrical stimulation of the optic chiasm.
12. The overall effect of eye position signals on the visually elicited responses was tested with a one-factor, repeated measure ANOVA test [B. J. Winer, *Statistical Principles in Experimental Design* (McGraw-Hill, New York, 1962)] at a significance level of $P < 0.05$. All neurons that had a statistically significant effect were tested for a trend of the effect with a trend analysis program [R. R. Sokal and F. J. Rohlf *Biometry* (Freeman, New York, 1981)]. The trend analysis was done for linear and quadratic functions. Significant linear trends were fitted with a linear regression (least-squares method), and nonlinear trends were fitted with a second-order polynomial (quadratic) with the curve fitter program [P. K. Warma, *Curve Fitter* (Interactive Microwave, Philadelphia, 1980)].
13. Of 38 cats tested, eye position changes or eye movements significantly affected the visual responses in LGN neurons in 33 cats. Significant eye position effects occurred for 23 of 133 neurons, and significant eye movement effects occurred for 25 of 76 neurons (as evaluated by both the number of action potentials and firing rate criteria).
14. M. Stryker and C. Blakemore, *Vision Res.* **12**, 2005 (1972); M. Crommelink and A. Roucoux, *Brain Res.* **103**, 574 (1976).
15. R. A. Andersen, G. K. Essick, R. M. Siegel, *Science* **230**, 456 (1985).
16. A similar set of test conditions with movements occurring at various times but with the movement in the nasal direction had no effect on the neuron's visual response. Of 12 neurons that had a significant facilitatory effect of eye movement and were subsequently tested for effects of movement in the opposite direction, 9 showed a directional preference with 7 preferring nasalward movement and 2 preferring temporalward movement.
17. Although our experiment demonstrates that afferent eye position and movement signals gate retinogeniculate signal transfer (and thus the nature of the signal that reaches the visual cortex), we have not verified the route by which this information reaches the LGN_d. Because the LGN_d receives a reciprocal innervation from the visual cortex, the signal could be a recurrent one. Alternatively, the ascending projections to the LGN_d from sites in the mesencephalic reticular formation [W. Singer, *Brain Res.* **61**, 35 (1973); S. M. Sherman and C. Koch, *Exp. Brain Res.* **63**, 1 (1986); W. Francesconi, C. M. Müller, W. Singer, *J. Neurophysiol.* **59**, 1690 (1988)], which also receives afferent eye movement signals [S. Cooper, P. M. Daniel, D. Whitteridge, *Brain* **78**, 564 (1955)], may be the source. The effect of eye position and movement on visual responses of LGN_d neurons was determined to occur central to the optic tract axons. In control experiments ($n = 7$ cats, $n = 24$ axons), no effects of eye movement were observed on the visual responses or spontaneous activity of single retinal ganglion cell axons. In additional control experiments we used local anesthetic blockade of the eye position and movement effect by applying lidocaine (4%) to EOM ($n = 4$ cats, $n = 4$ LGN_d neurons that had previously shown a significant eye position or movement effect). These results support our suggestion that the effects are mediated via afferent EOM signals.
18. For example, in the 0° to 5° RF range, many (7 of 8 X cells and 3 of 6 Y cells) of the cells tested had a significant eye movement effect without regard to cell class, whereas in the same region none of the cells tested (0 of 15 X cells and 0 of 9 Y cells) showed an effect of eye position change on their visual response.
19. C. Lee *et al.*, *Nature* **332**, 357 (1988).
20. D. Zipser and R. A. Andersen, *ibid.* **331**, 679 (1988).
21. Supported by NIH grant EY-05116 (M.J.F.), the National Society to Prevent Blindness (R.L.), and the Alfred P. Sloan Foundation (M.J.F.). We thank K. Ramer for software development, J. Rodda for technical assistance, Y. Fregnac and K. A. C. Martin for comments on the manuscript, D. L. Sparks and J. D. Porter for discussions of the experiments, and R. Enge for processing the manuscript.

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