Saccade-induced activity of dorsal lateral geniculate nucleus X- and Y-cells during pharmacological inactivation of the cat pretectum

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Abstract

The influence of neurons projecting from the pretectal nuclear complex to the ipsilateral dorsal lateral geniculate nucleus (LGNd) was investigated in awake cats. Responses from relay cells in the A-laminae of the LGNd were extracellularly recorded and analyzed during saccadic eye movements and visual stimulation in association with reversible inactivation of the ipsilateral pretectum with the GABA agonist, muscimol. Pretectal inactivation (PTI) resulted in spontaneous nystagmic eye movements in the dark with slow phases directed away from the injected side. In the control situation, all Y-cells and about two thirds of X-cells were excited during saccades or saccade-like visual stimulation but one third of X-cells were inhibited. During PTI all recorded X-cells were inhibited, either during saccades or saccade-like visual stimulation. The PTI-associated inhibition was stronger than in inhibited X-cells in control experiments only during saccades but not during stimulation with a moving pattern while the eyes were stationary. In Y-cells a reduction in the response peak width at half-height was seen during PTI, again only during saccades but not during stimulation with a moving pattern. These results indicate that during saccades the pretecto-geniculate pathway has a stronger influence on X LGNd relay cells than on Y-cells. The findings are discussed in terms of saccadic suppression and postsaccadic facilitation.

Keywords: Vision, Saccadic eye movements, Saccadic suppression, Postsaccadic facilitation

Introduction

The dorsal lateral geniculate nucleus (LGNd) is a highly integrative center in the pathway from the retina to the visual cortex. Thus, not surprisingly, only 10–20% of the synaptic input to the LGNd cells stems from retinal afferents. The remaining synaptic input of up to 80–90% derives from other sources including corticogeniculate axons, local interneurons, the thalamic reticular nucleus, hypothalamus, and the brain stem (for review see Guillery, 1969*a,b*, 1971; Sherman & Koch, 1986; Sherman, 1993). Functionally, this extraretinal input to LGNd neurons is thought to allow context specific processing of visual information, required during eye movements and other states of attention and wakefulness (Sherman & Koch, 1986; Steriade & Llinas, 1988; Steriade et al., 1990, 1993; Sherman, 1993; McCormick & Bal, 1994).

During saccadic eye movements the neuronal responses of single cells in the cat LGNd are specifically affected. Noda (1975*a,b*) described excitatory activity during saccades for Y-cells and inhibitory responses for X-cells. More recently, excitatory activity was also found for X-cells during saccades (Lee & Malpeli, 1994). In our own experiments, we found that about 30% of X-cells were

inhibited during saccades whereas the remaining X-cells showed excitatory responses (Fischer et al., 1996). A comparison of neuronal responses induced by saccade-like stimulus shifts while the eye was stationary, with actual saccade-evoked responses revealed that the latter had shorter latencies. From these results, a saccade-related nonretinal input to LGNd neurons was expected which most likely is mediated *via* subcortical centers involved in the control of eye movements.

In our studies, we concentrated on the projection from the pretectal nuclear complex to the LGNd. This projection originates from the nucleus of the optic tract (NOT) and the dorsal part of the posterior pretectal nucleus (NPP) (Kubota et al., 1987; Nabors & Mize, 1991; Cucchiaro et al., 1991, 1993; Wahle et al., 1994; Uhlrich & Manning, 1995; Schmidt et al., 1996). The NOT is most frequently referred to as a critical structure for the generation of the optokinetic nystagmus (Hoffmann, 1989). This could be demonstrated by recordings of single pretectal units (cat: Hoffmann & Schoppmann, 1981; monkey: Mustari & Fuchs, 1990; Ilg & Hoffmann, 1991), and by the effects of electrical stimulation (monkey: Schiff et al., 1988). Lesioning of the pretectal area resulted in directional deficits of the optokinetic system (Kato et al., 1986; Schiff et al., 1990) and of the pursuit system (Ilg et al., 1993). However, the neurons involved in this behavior, termed "retinal slip" neurons (Ballas & Hoffmann, 1985) do not project to the LGNd (Schmidt & Hoffmann, 1992). It was reported in cat that the

pretectal projection to the LGNd most likely consists of GABAergic neurons (Cucchiaro et al., 1991; Wahle et al., 1994) which seem to specifically contact inhibitory interneurons in the LGNd and in the perigeniculate nucleus (PGN) (Uhlrich & Cucchiaro, 1992; Cucchiaro et al., 1993). It has previously been shown in cats that the pretecto-geniculate cells are excited during saccadic eye movements with a duration exceeding the eye movement (Schmidt, 1996).

Several parallel neuronal streams from the retina to the LGNd are known including the X- and Y-pathways which are the best characterized. It is suggested that the X-pathway is involved in the analysis of fine spatial resolution whereas the Y-pathway is involved in the analysis of form vision and motion (reviewed in Sherman & Koch, 1986). From these facts it may be expected that these pathways are affected differently during saccadic eye movements.

Thus, the purpose of this study was twofold. First, we wanted to reveal the effect of the pretectal nuclear complex projection on LGNd relay neurons. This was achieved by measuring the saccade-related LGNd cell responses during pharmacological inactivation of the NOT and NPP. Second, we wanted to test whether there were differential effects of pretectal inactivation on the X- and Y-pathway.

Methods

Animal preparation

Our methods and equipment have been described in more detail elsewhere (Fischer et al., 1996) and are only briefly summarized here. Adult cats of both sexes were anesthetized with a mixture of Ketamine (20 mg/kg body weight) and thiazine hydrochloride (1 mg/kg). The trachea was intubated and anesthesia was maintained with 0.2–0.4% halothane in a 3:1 mixture of nitrous oxide and carbogen (95% O_2, 5% CO_2). Body temperature was monitored *via* a rectal probe and maintained at 37°C by a servo-controlled heating blanket. The CO_2-level of the expired air, intratracheal pressure, and electrocardiogram were continuously monitored throughout the surgery.

A craniotomy was performed above the LGNd, at Horseley-Clarke coordinates A0-A10/L4-L14, and a recording cylinder together with anchor screws were fixed to the skull with dental acrylic cement. To facilitate the localization of our injections, the NOT was first localized in electrophysiological recordings. When typical NOT unit activity was found to horizontal movement of large-field visual stimuli, the recording electrode was retracted and a 26-gauge stainless-steel guiding cannula was inserted at the same location with the tip 2 mm above the measured cell activity. In between experiments, the clearance of the guiding cannula was protected by a stainless-steel stylet and closed by a screw-top cap.

For electrical stimulation of the optic chiasm, a low-impedance bipolar electrode was implanted and fixed to the skull with dental acrylic cement. Eye movements were measured by an electromagnetic method employing a scleral search coil implanted in one eye (Judge et al., 1980).

After surgery, animals were treated with antibiotics and analgesics as required and were allowed to recover for at least one week before the first recording experiments.

Recording and stimulation

Cats were placed in a custom-made acrylic box to comfortably confine body movements and the head was fixed to the box by an elastic head holder. Horizontal and vertical eye movements were monitored using the phase-detection principle in a magnetic field (Fuchs & Robinson, 1966; Hartmann & Klinke, 1976; Kasper & Hess, 1991). Eye movements were measured relative to the stationary head, which was placed in the center of the magnetic field. Recording sessions typically lasted for 2.5 h or as long as no signs of discomfort for the cats were seen.

Extracellular recordings were made from single units in the A-laminae of the LGNd with tungsten-in-glass electrodes. The recording electrode was fixed to a holder which was mounted to the recording cylinder and was advanced by a hydraulic microdrive through the intact dura. Signals were amplified, band-pass filtered, and digitally stored for off-line analysis.

Single biphasic current pulses of 60–80 ms duration and of amplitudes up to 1.0 mA were applied to the stimulation electrode at a rate of 1/s. All latencies given in the text refer to the first negative peak of the evoked spikes.

For visual stimulation, a random square pattern (square size 1 deg; light square 43 cd/m^2; dark square 5 cd/m^2; contrast: 0.79) was projected onto a tangent screen located 80 cm in front of the cat. The pattern covered a visual field of 95 deg by 95 deg. Pattern movements were controlled by a computer and were presented in the horizontal direction.

Extracellular recordings were performed under the following stimulus conditions. (1) While the cats executed spontaneous saccades in front of the stationary random-square pattern and in complete darkness. (2) During stimulation with the pattern moving in a saccade-like fashion which mimicked a retinal image shift as it occurred during a spontaneous saccade but with the eye stationary. This stimulus was generated from the eye position signal during a horizontal saccade recorded from one of the cats (stimulus duration: 85 ms, mean velocity: 110 deg/s, peak velocity: 225 deg/s). (3) During visual stimulation moving the random-square pattern in a ramp-like fashion with a velocity of 440 deg/s and a duration of 80 ms across the screen. This stimulus was used as a control whether a high-velocity image shift over the retina is able to activate the pretecto-geniculate system. The ramp stimulus was identical to the sudden stimulus shift that drives pretectal neurons that project to the LGNd (Schmidt, 1996).

Inactivation of the pretectum

Functional inactivation of the pretectum was achieved by pressure injection of muscimol. Control injections consisted of a 0.9% saline solution. Injections were delivered through a stainless-steel needle inserted in the guide tube. A plastic tubing calibrated in 1-ml divisions was connected to the injection needle and the solution was applied with a manually driven 10-ml Hamilton syringe. The injected volume was controlled by observing the movement of a small air bubble placed in the plastic tube close to the injection needle. 1 ml of muscimol in saline (1 ml/mg) was injected at a rate of 0.2 ml/min. In the first experiment, the injection of the drug started 2 mm above the expected location of the pretectum. The application continued on separate days at increasing depth in 0.5-mm increments until the injection site was found at which muscimol elicited spontaneous nystagmus in darkness characteristic of NOT inactivation. Saline solution was injected to verify that the applied volume had no effect. All subsequent injections were made unilaterally into the NOT in the fully alert cat.

During control measurements, extracellular activity was recorded in cats without injections into the NOT. This pre-injection situation will be referred to as the "control" situation in the fol-

lowing. After muscimol application the occurrence of spontaneous nystagmic eye movements in the dark indicated the temporal inactivation of the NOT, further referred to as pretectum inactivation (PTI) measurement. During PTI neuronal activity induced by spontaneous saccades was recorded with the same stimulus conditions as during the control period before. Thus, we compared a set of cells the activity of which was measured during the control situation with a another set of individually different cells which were recorded during PTI.

Data analysis

The following tests were used to classify cells as X- or Y-cells (Cleland et al., 1971; Hoffmann et al., 1972). First, orthodromic latencies to electrical pulse stimulation of the optic chiasm was measured. Second, the random-square pattern was switched on and off for 1 s each to record transient and sustained responses. Examples of neuronal responses to pattern on or off are given in Figs. 3 and 4. Third, responses to fast movement ($>$100 deg/s) of a black bar for ON center and a white bar for OFF center cells were determined. A cell was classified as an "X-cell" if it had a latency between 1.6 and 2.5 ms, a sustained response to lights on or off for at least 500 ms, and did not response to the fast moving bar. A cell was classified as a "Y-cell" if it had a latency between 0.9 and 1.4 ms, a transient response to lights on and off for at most 100 ms, and responded to the moving bar. Cells were considered as unclassified if they did not satisfy all three criteria for either class.

Eye velocity and saccade detection were calculated off-line. A saccade was detected if the eye velocity exceeded 50 deg/s. Saccade onset was defined as the time when the velocity reached 10% of the peak velocity during the rising part of the velocity profile. Mean slow-phase eye velocity was calculated from "desaccaded" spontaneous nystagmic eye movements during darkness.

For neuronal activity to be related to the eye or stimulus movements, our data had to pass certain criteria. First, excitatory response activity had to contain at least four spikes with consecutive interspike intervals shorter than half of the mean interspike interval of the whole recording period (i.e. 30 s for each stimulus condition). Second, the probability of the defined excitatory response activity in a time window of 80 ms after saccade onset was tested against the probability of appearance in a time window of equal length before saccade onset (chi square test). Only at significance ($P < 0.01$) the occurrence of an excitatory response after saccade onset was regarded as saccade related. The onset of this excitatory response activity was defined to be the first interspike interval in which the instantaneous firing rate was less than the mean rate. The response latency of a single cell was taken as the mean of all saccade related onset activities for each cell.

The onset of inhibitory activity was determined by correcting the neuronal response for spontaneous activity and calculating the width at half-maximal inhibition. The onset of half-maximal inhibition is regarded as the onset of inhibition and the length of half-maximum as duration of the inhibition. For Y-cells, the peak amplitude was calculated as the ratio between the activity in a time window starting from saccade onset until the beginning of the excitation and the peak activity.

Single cell responses were aligned to either saccade or stimulus onset and, because the number of saccades was variable between individual recordings, mean response histograms were processed. The histograms were built up only from measurements in which no saccade appeared in the time window 100 ms before and 400 ms after saccade or stimulus onset. From all neuronal responses pop-ulation histograms were calculated by summing up the response histograms. There are at least three reasons for the use of population histograms. First, because LGN-projecting pretectal neurons show very uniform response properties, e.g. all cells are activated by saccadic eye movements (Schmidt, 1996), probably every saccade leads to a synchronous activation of the pretecto-geniculate projection. Second, single pretectal fibers terminate in LGN A-layers in a spatially unselective manner (Uhlrich & Manning, 1995), so that an individual LGN relay cell will receive input from several pretectal afferents. Finally, mostly because muscimol injections lead to an inactivation that lasts several hours, we could not switch between pre-injection and PTI situation during a single recording session so that we could not record from individual cells before and during PTI. The population histograms were calculated according to cell/response type, e.g. all Y-cells, all X-cells that were excited by saccades in the pre-injection situation, all X-cells that were inhibited by saccades in the pre-injection situation, and all Y- and X-cells during PTI.

Histological procedures

In some experiments, selected recording sites were marked by electrolytic lesions (5–10 μA $*$ 10 s, electrode tip negative) to obtain subsequent histological reconstruction. The cats were sacrificed at the end of the experiments by a pentobarbital overdose and perfused transcardially with saline and 10% phosphate-buffered formalin. Brain blocks were removed and 50-μm-thick frozen sections were cut. Nissl or Klüver-Barrera staining was used for localization of the electrolytic lesions and the position of the injection needle, respectively.

Results

Location of the injection site

The reconstruction of the injection site confirmed that the tip of the injection needle was situated in the pretectal area (Fig. 1, arrow). The darker shaded area (broken line) possibly marks the apparent extension of the drug spread around the tip of the injection needle. Muscimol injections at this site had strong effects on eye movements in darkness (see below). In the light, however, no significant changes of the metrics of eye movements, including spontaneous saccades, occurred.

Spontaneous nystagmus

In darkness, 5 to 10 min after application of muscimol into the left pretectal region, spontaneous nystagmus in the horizontal plane with slow phases directed towards the right (noninjected side) developed (Fig. 2). The mean eye velocity of this spontaneous nystagmus was 3.1 deg/s in cat 1 and 10.2 deg/s in cat 2. Slow-phase eye velocities were in the same range for each animal on consecutive days of drug application. This indicates that during the recording sessions a similar level of inactivation of the pretectal area was achieved on consecutive days. When spontaneous nystagmus occurred in darkness, visual stimulation elicited optokinetic nystagmus (OKN) only in the direction towards the noninjected side (e.g. to the right with an injection in the left NOT). During visual stimulation with a stationary random-square pattern, no nystagmus of the eyes was seen. Control injections of saline did not elicit spontaneous OKN and had no effects on eye movements.
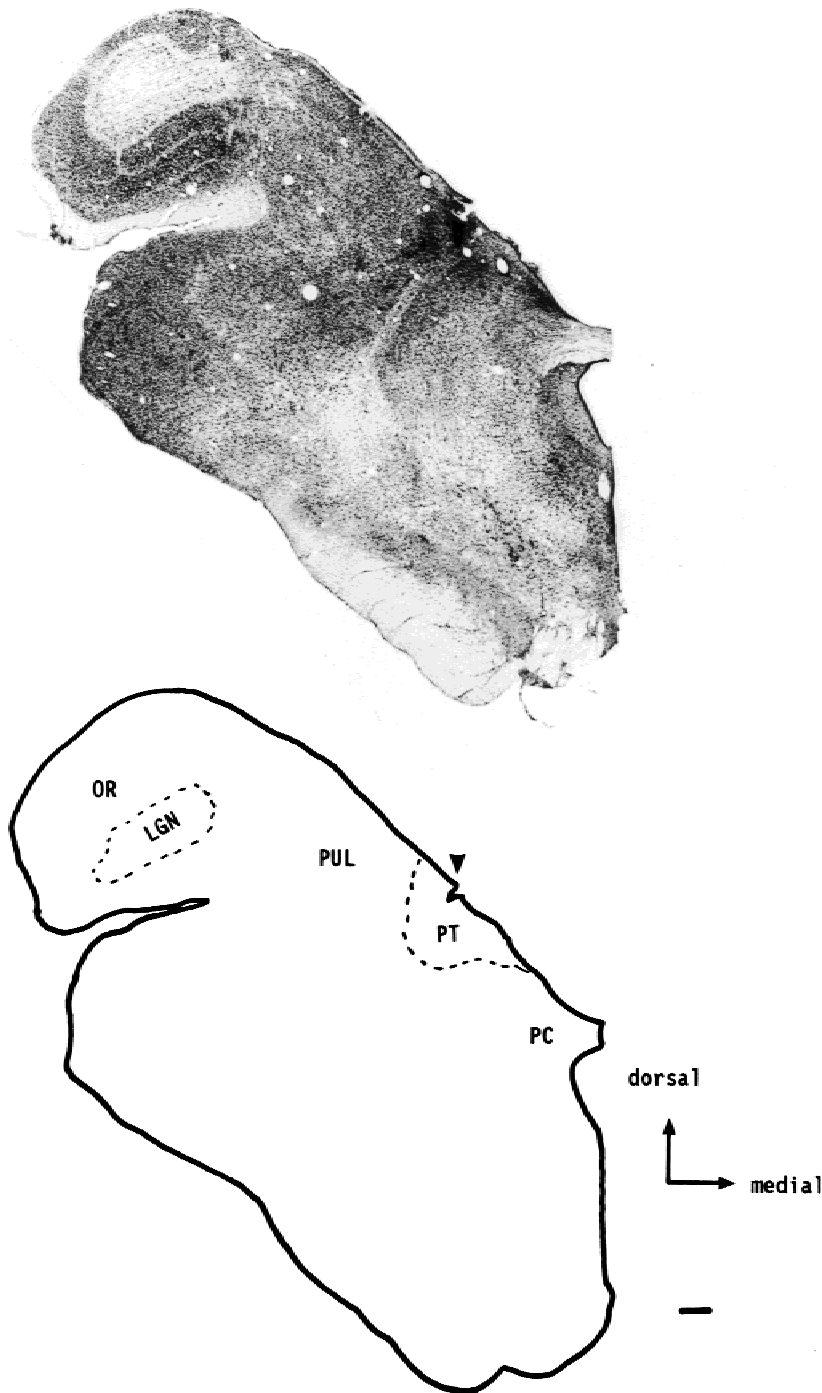
Fig. 1. Photomicrograph of a Nissl-stained frontal section through the right pretectum (top) and its reconstruction (bottom). The injection needle was inserted from above (arrow) and its tip was located in the pretectal area (PT). The darker region surrounded by a broken line seems to be the diffusion area of the muscimol. LGN: lateral geniculate nucleus; OR: optic radiation; PC: posterior commissure; PT: pretectal area; and PUL: pulvinar. Scale bar = 1 mm.

LGN cell responses

From a total of 282 cells recorded in the A-laminae of the LGNd, 260 were analyzed during saccadic eye movements. They could be subdivided into 109 Y-cells, 117 X-cells, and 34 unclassifiable cells. Neuronal activity was modulated in 89 Y-cells (82%) and 91 X-cells (78%) during saccades. The remaining X- and Y-cells showed no response to saccadic eye movements but could be driven by visual stimulation (Fischer et al., 1996).

Controls

In a previous report (Fischer et al., 1996), we have described the response properties of LGNd lamina A neurons during saccadic eye movements. The results could be confirmed in the present report. During saccades X-cells either were excited (70%) or inhibited (30%). Fig. 3 gives an example of an excited (A–C) and an inhibited (D–F) X-cell. Both cells showed saccade-induced modulation and also responded to saccade-like stimulus movements

horizontal eye movements
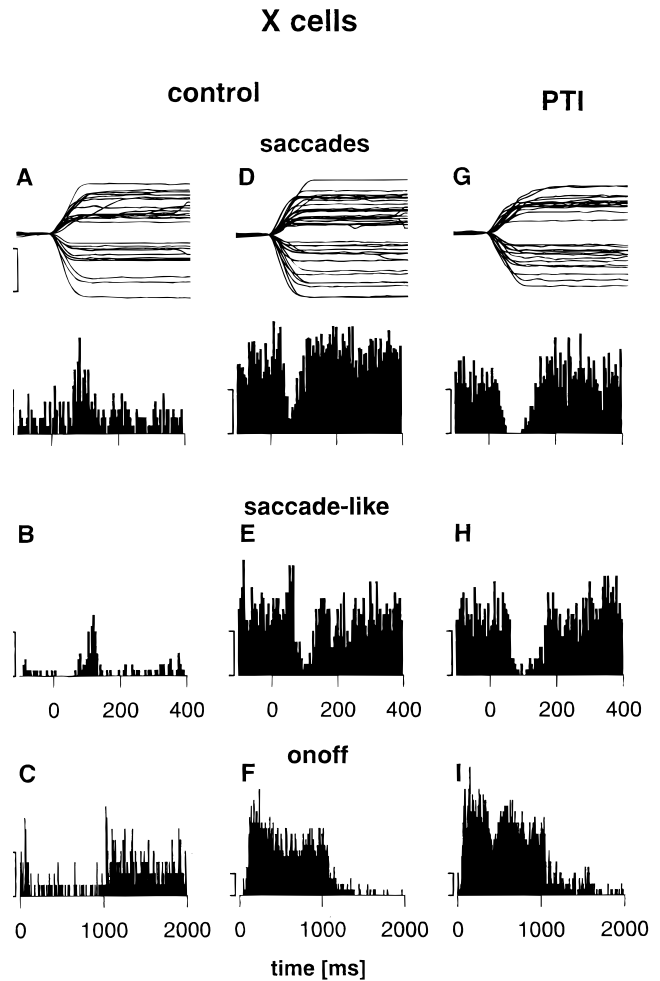

vertical eye movements

5 s
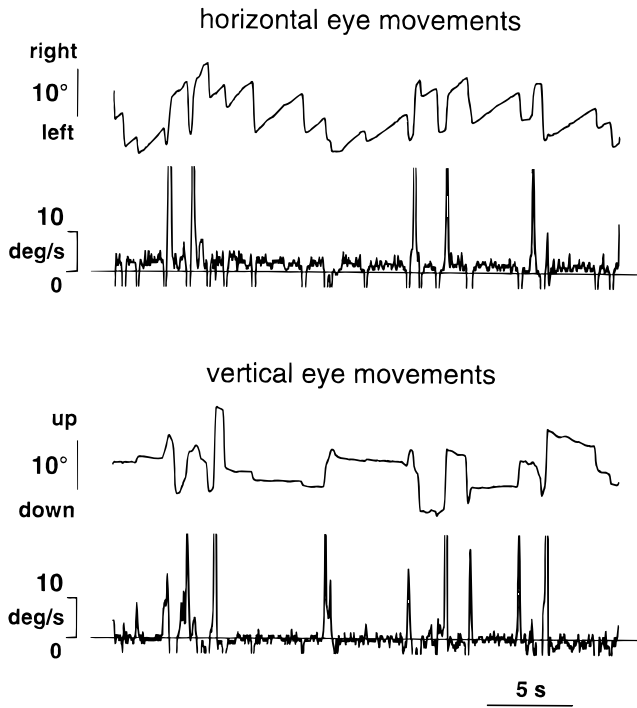
Fig. 2. Spontaneous nystagmus recorded in darkness 10 min after injection of muscimol into the left pretectal region of cat 1 during a 30-s recording period. Horizontal and vertical eye position are displayed (upper traces) together with the eye velocity (lower traces). A spontaneous nystagmus in the horizontal plane occurred with slow phases towards the right, i.e. noninjected side. In the vertical plane no nystagmic eye movements were seen.

X cells

control   **PTI**

saccades

Fig. 3. Examples of neuronal activity of X-cells during control (A–F) and during pretectal inactivation (G–I). Top row: Eye position traces and perisaccade time histogram with neuronal activity aligned to saccade onset (time 0). Middle row: Peristimulus time histogram, neuronal activity aligned to the onset of the saccade-like stimulus movement (time 0). Bottom row: Responses in the dark when the stimulus pattern was switched on (first 1000 ms) and off (second 1000 ms). In the control situation the first cell (A–C) was excited during saccades, the second cell (D–F) was inhibited. During pretectal inactivation, all X-cells were inhibited during saccades like the third cell (G–I). Histograms show responses to 30 (A), 37 (D), 26 (G) saccades and to 30 stimulus presentations (B,C,E,F,H,I). Latencies to saccade onset: 54.8 ms (A), 45.1 ms (D), 40.2 ms (G). Latencies to stimulus movement onset: 65.8 ms (B), 73.2 ms (E), 69.5 ms (H). In the eye position traces, rightward and upward saccades are represented by an upward deflection. Histogram binwidths: 5 ms. Scale bars: 10 deg and 50 spikes/s.

with differences in latencies as described previously (Fischer et al., 1996). A summed histogram of 27 X-cells that were excited during saccades is shown in Fig. 5A and of 32 inhibited X-cells in Fig. 6A. The summed histograms were calculated from individual neurons without weighting because we assume that the LGN output is best described by a simple summation of single cell responses. However, we also calculated summed histograms by using cell responses normalized to the mean response in the 100-ms period before saccade onset. These normalized population responses did not show significant differences to the summed histograms shown here. Mean latency to saccade-induced excitation was 35.5 ms (s.d. = 6.9, n = 27 in Fig. 5A) and to inhibition 46.3 ms (s.d. = 5.6, n = 32 in Fig. 6A), respectively.

Y-cells always were excited during saccades and saccade-like visual stimulation. An example is given in Figs. 4A–4C. A summed histogram of 48 Y-cell responses during saccades is depicted in Fig. 9A. Saccade-induced responses had a mean latency of 32.9 ms (s.d. = 6.8, n = 48).

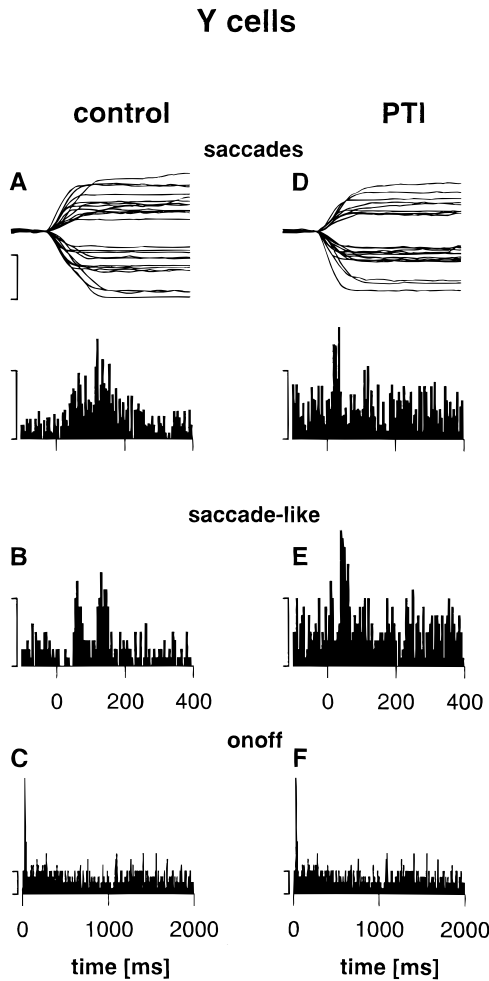### X-cells during pretectal inactivation

During inactivation of the pretectal region the most prominent changes in response properties occurred in X-cells. Under this condition no excitatory responses could be recorded in X-cells. Instead all X-cells were inhibited during saccades and also during visual stimulation with the random-square pattern moving in a ramp or saccade-like fashion (Figs. 3G and 3H). In contrast, responses to light on and off were not altered during inactivation of

the pretectum (Fig. 3I). The comparison of neuronal responses of X-cells during the control situation (control) and during pretectal inactivation (PTI) is shown in population histograms in Fig. 5. We will first consider the X-cells that were excited and then the X-cells that were inhibited during saccades and during stimulation with a moving pattern in the control situation.

The summed activity of X-cells that were excited during the control situation is shown in Fig. 5A. When the pretectum was inactivated all recorded X-cells were inhibited (Fig. 5B). The same effect was observed during stimulation with a pattern moving in a saccade-like fashion while the eye was stationary. The cells

Y cells

control   **PTI**

saccades

saccade-like

onoff

time [ms]   time [ms]

Fig. 4. Examples of neuronal activity of two Y-cells during control (A–C) and during pretectal inactivation (D–F). Same conventions as in Fig. 3. Histograms show responses to 25 (A), 23 (D) saccades and to 30 stimulus presentations (B,C,E,F). Latencies to saccade onset: 39.5 ms (A), 32.5 ms (D). Latencies to stimulus movement onset: 48.5 ms (B), 40.2 ms (E).

were excited during controls (Fig. 5D) and inhibited during PTI (Fig. 5E). To estimate the contribution of the pretectum to the activity of LGNd X-cells, we calculated the difference histograms between the control situation and pretectal inactivation (Figs. 5C and 5F). A strong excitatory difference in activity, between 50 and 150 ms, is revealed during saccades and saccade-like stimulation.

Calculating the difference between the difference histograms from Figs. 5C and 5F resulted in a histogram termed "effective PT-LGN activity" (Fig. 5G). This activity was calculated by subtracting the activity transferred by the pretectal input to LGN X-cell during saccade-like retinal image shifts from the activity during saccades. It is therefore thought to reflect the input from the pretectum to LGNd X-cells elicited by the actual eye movement.

In X-cells that were inhibited during controls, a more subtle effect during inactivation was observed. When the pretectum was inactivated, the summed X-cell activity during saccades was more strongly inhibited than during the control situation (Figs. 6A and 6B). The difference histogram of these two situations (Fig. 6C) shows a broad peak of activity around 100 ms after saccade onset which is the result of the shorter period of inhibition during the control situation. Thus, the effect of a stronger inhibition during

pretectal inactivation is mainly due to a prolongation of the inhibition. A prolonged inhibition during pretectal inactivation could not be seen during saccade-like stimulation (Figs. 6D and 6E) and, consequently, the difference histogram shows only low activity during the inhibition period (Fig. 6F). The effective PT-LGN (Fig. 6G) activity again may reflect the input from the pretectum to suppressed LGNd X-cells and shows enhanced activity around 80 ms. This may be the input elicited by the actual eye movement.

In further analysis, we compared the inhibition during inactivation with neuronal behavior of X-cells that were inhibited during the control situation. In Fig. 7, the peak strength of inhibition and the latency of inhibition are displayed for all inhibited X-cells in cumulative diagrams for the control situation (open circles) and during PTI (filled triangles). The strength of inhibition was calculated as the decrease in percent of cell activity during inhibition relative to maintained activity in a time window of equal width of the inhibition period prior to the saccade. Fig. 7A shows that, during saccadic eye movements, a reduction of up to 90% of the neuronal activity occurred during the inactivation of X-cells relative to spontaneous activity in both control and PTI situations. However, in the PTI situation the activity was significantly more reduced during the inhibition than in the control situation (Mann-Whitney rank sum test, $P < 0.05$; median during control: 57.8%, median during PTI: 67.4%). The distribution of saccade-induced X-cell inhibition, i.e. the shape of the cumulative plot did not change during PTI but shifted to the right. This indicates that the decrease of the population response most likely was not due to a strong PTI effect on only a small number of cells but that the entire population was affected. In contrast, the inhibition of X-cells, during saccade-like and ramp stimulation, was not stronger when the pretectum was inactivated (Figs. 7B and 7C).

The latency of the inhibition was not altered during pretectal inactivation as compared to the control situation, both for saccadic eye movements and visual stimulation (Figs. 7D and 7F). As found earlier (Fischer et al., 1996), the latency to inhibition in controls was smaller during saccadic eye movements than during visual stimulation (Mann-Whitney rank sum test, $P < 0.0001$) which indicates an input to X-cells during saccades that adds to the input caused by the stimulus movement across the retina.

As mentioned earlier, the majority of X-cells in the LGNd was not inhibited but excited during saccades and ramp-like and saccade-like stimulation (Figs. 5A and 5D). To take these response modes into account, we constructed the population histogram of all X-cells recorded during the control situation regardless of whether the X-cells were excited or inhibited during saccades (Fig. 8C, filled area). The second trace in Fig. 8C depicts the summed activity of all recorded X-cells during pretectal inactivation. The difference histogram between the control and the PTI situation for all X-cells is shown in Fig. 8D together with the mean eye velocity trace of 100 saccades registered during the recording sessions. The positive difference activity between 30 ms and 130 ms after saccade onset reflects the LGNd X-cell activity which is mediated by the activity of pretectal cells during saccades. The latency of about 30 ms with respect to the onset of the velocity trace is in agreement with our earlier study (Fischer et al., 1996).

Y-cells during pretectal inactivation

During pretectal inactivation, no obvious effects on Y-cells were found that were similar to those described for X-cells above. An example of a Y-cell response during PTI is depicted in Figs. 4D–4F. The population response of Y-cells during saccades in the
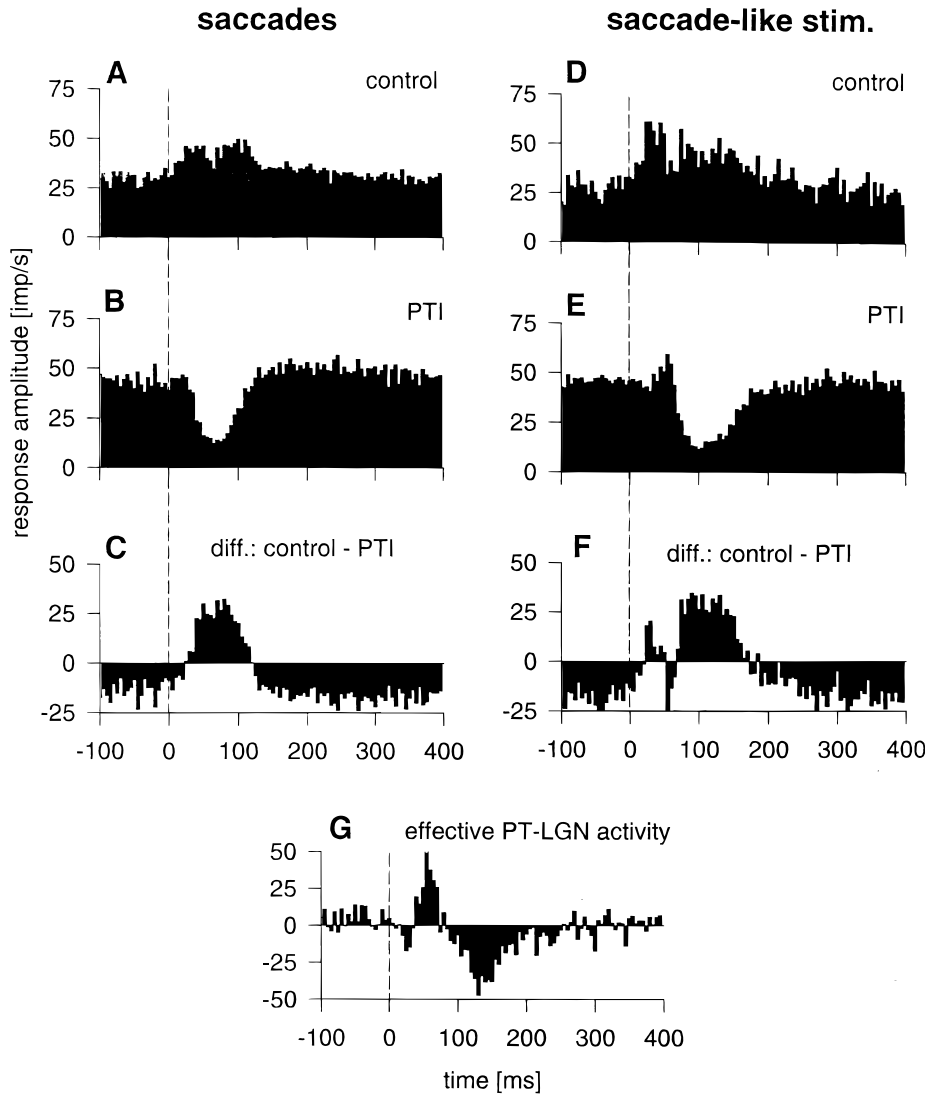
Fig. 5. Population response of LGNd X-cells which were excited during saccades and during visual stimulation with a random-square pattern moving in a saccade-like fashion in the control situation. Histograms are aligned to saccade onset (time = 0 ms, vertical line) in the left panel and to stimulus onset in the right panel. Population responses during the control situation (A,D; $n = 27$ cells) are compared with responses during inactivation of the pretectum (B,E; $n = 27$ cells) and the difference histograms of these two situations are given (C,F). The summed histogram of the effective PT-LGN activity in G is the difference of the histograms in C and F. Binwidth of histograms: 5 ms.

control situation (Fig. 9A) resembles the summed responses of Y-cells during PTI (Fig. 9B). However, in the difference histogram (Fig. 9C), there is a positive activity around 50 ms after saccade onset, indicating a shortened response of the initial peak in the neuronal response during PTI. This shortening of the initial response cannot be seen in the difference histogram of the saccade-like stimulation (Fig. 9F) and, thus, may be a saccade-induced effect. The difference of the histograms C and F (Fig. 9G) shows this effect around 50 ms and can be interpreted as the influence of pretectal cells on LGNd Y-cells during saccadic eye movements.

The consequences of PTI for Y-cells was investigated further on the single-cell level. In Fig. 10 the peak width at half-height, the peak amplitude, and the latency to excitation are shown in cumulative diagrams. A difference in neuronal behavior between controls and PTI was only found for the peak width at half-height of responses to saccadic eye movements (mean: control = 52.4 ms, PTI = 37.4 ms; Fig. 10A). During inactivation, the duration was much shorter (*t*-test, $P < 0.05$) than during controls but was not changed during visual stimulation with a moving pattern (Figs. 10B and 10C). These single-cell quantifications confirmed the results of the difference histograms of Fig. 9.

Both peak amplitude (Figs. 10D–10E) and latency of excitation (Figs. 10G–10H) were not altered during PTI compared to the control situation regardless of whether stimulated by saccadic eye movements or a visual pattern while the eyes were stationary.

Spontaneous activity of X- and Y-cells

To search for an overall effect of muscimol application into the pretectum, the spontaneous activity in controls and during NOT inactivation was compared (Table 1). The spontaneous activity did not change between X-cells that were inhibited during control and X-cells during PTI. Similarly, the inactivation had no effect on spontaneous activity in Y-cells. However, excited X-cells during control showed lower spontaneous activity than X-cells during PTI. This is also seen in Figs. 5C and 5F, where the negative response before time 0 ms and after 200 ms resulted from an increased spontaneous activity during PTI (Figs. 5B and 5E). We think this increase was not a functional effect of neuronal activity but resulted from the fact that the histogram of X-cell activity during PTI was composed of all X-cells recorded during the period of inactivation, containing cells that might be excited or inhibited during the con-

saccades
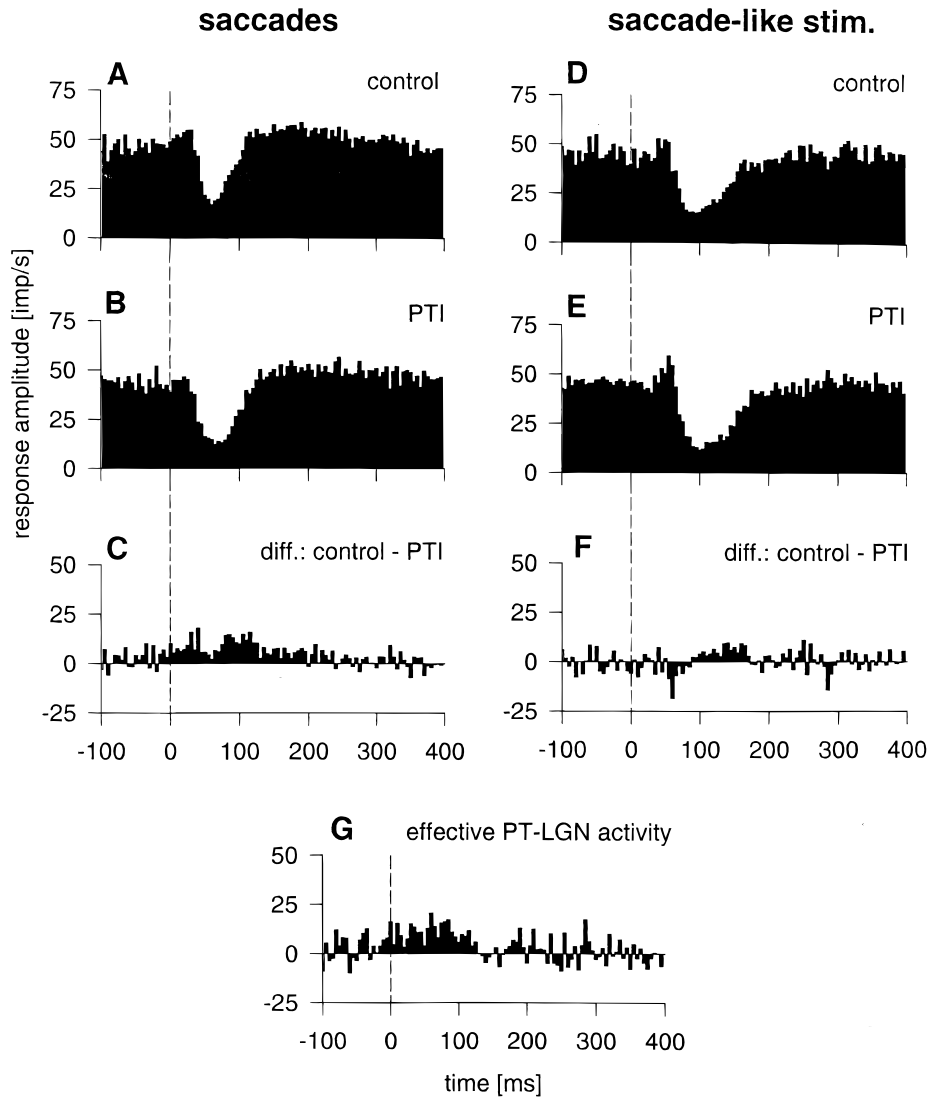
saccade-like stim.

Fig. 6. Population response of LGNd X-cells that were inhibited during saccades and during visual stimulation with a random-square pattern moving in a saccade-like fashion in the control situation. Same conventions as in Fig. 3. Number of cells in A,D,B,E: $n = 32$.

trol situation. Since the spontaneous activity of inhibited X-cells during control is higher than in excited X-cells, the spontaneous activity of the population response of X-cells during PTI should be also higher compared to excited X-cells during control.

Discussion

This study analyzes the influence of the pretecto-thalamic projection on LGNd cellular activity during saccadic eye movements.

Table 1. *Response amplitude of spontaneous activity of LGNd X- and Y-cells (impulses/sec)*

	X			Y	
	Control excited	Control inhibited	PTI	Control	PTI
Mean	30.2	46.9	42.9	20.2	19.7
s.d.	15.0	23.0	20.8	16.4	11.2
n	27	32	32	48	27

Inactivation of the pretectal area with the GABA agonist muscimol induced spontaneous nystagmus in darkness directed away from the injected side. During this inactivation, the activity of X-cells to saccades or saccade-like movements of a visual stimulus was always inhibited. This PTI associated inhibition was stronger than in inhibited X-cells in control experiments. In contrast, pretectal inactivation affected Y-cells only in a single parameter, i.e. during saccades the peak width at half-height was smaller than during control situations.

Recordings of neuronal activity in the LGNd during pretectal inactivation require a stable and prolonged effect of muscimol on pretectal neurons. Martin (1991) injected [^{3}H]muscimol into the cortex and measured the spread of the labelled drug. He reported a maximal spread of 1.7 mm from the site of injecting 1 ml within the first 20 min postinjection. The width of muscimol injections remained relatively constant up to 2 h. These results could be confirmed by our behavioral measurements. The latest appearance of spontaneous nystagmus was 20 min postinjection and nystagmic eye movements with similar amplitude could still be measured at the end of the recording experiments, i.e. about 2 h postinjection. These results indicate a constant level of pretectal inactivation throughout the daily recording sessions. Assuming a similar spread

strength of inhibition

latency to inhibition
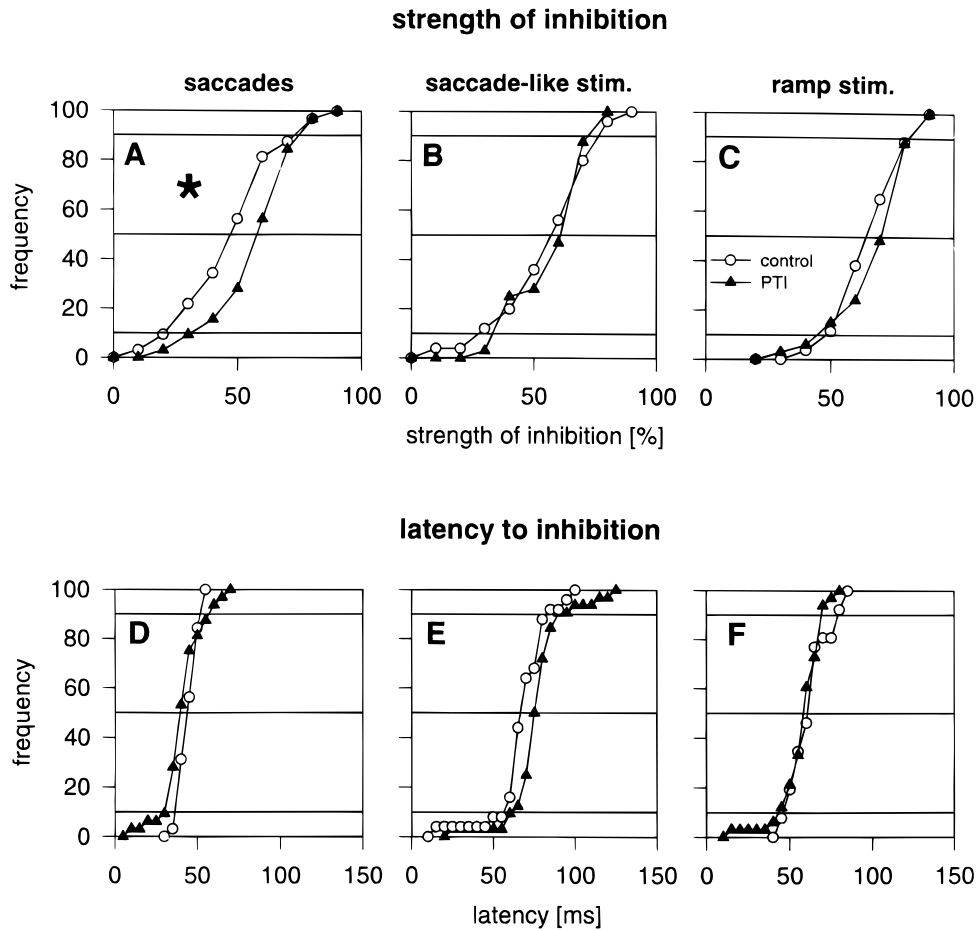
Fig. 7. Cumulative diagrams of two parameters of X-cells which were inhibited during control situation (open circles) and during pretectal inactivation (closed triangles). Strength of inhibition (A–C) and the latency to inhibition (D–F) during saccades (left column), during saccade-like movement of the stimulus pattern (middle column) and during ramp-like stimulus movement (right column). The horizontal lines at 50% frequency in each diagram give the median, the bottom lines the 10% fractiles, and the top lines the 90% fractiles. The asterisk marks the histogram with significantly different parameters between control situation and pretectal inactivation (Mann-Whitney rank sum test, $P < 0.05$). Values were derived from 32 cells during control and 32 cells during pretectal inactivation.

in the pretectal area as Martin measured in cortex, it can be seen from the histological reconstruction of the injection site that 20 min postinjection a large part of the pretectum must have been affected by the drug.

Effect of different stimulation modes and inactivation

We used a saccade-like stimulus that was derived from a spontaneous saccade of one of the cats and stimulated only in horizontal directions. However, Lee and Malpeli (1994) reported saccade-induced LGNd cell activity with directional bias in trained cats. This is in accordance with results from Thompson et al. (1994) who investigated the directionality of LGNd cells. They showed that only 30% of the relay neurons were direction selective and that this selectivity was only weak. Thus, we suppose that our restriction of the saccade-like stimulus movement to horizontal directions exerts only minor effects on our results. In addition, the LGNd-projecting pretectal neurons that might be preferably involved in the results described in this paper and will be further discussed below showed no correlation between response parameters and saccade direction (Schmidt, 1996). We think that this topic needs to be clarified with trained cats in the future.

From Figs. 7 and 10, it is evident that stimulation with a saccade-like and a ramp movement of the pattern did not result in differences between the controls and PTI situation. Despite the fact that a sudden stimulus shift which is similar to our ramp-like movement was able to drive pretectal cells (Schmidt, 1996), this excitation has little effect on the pretecto-geniculate system compared with the activation by saccade-induced retinal image shifts. During saccades, the response latencies of pretectal neurons identified as projecting to the LGNd were shorter and peak activities were increased compared to pure visual stimulation (Schmidt, 1996). These differences in response properties of pretectal cells, i.e. during saccadic and visual retinal stimulation, seem to be sufficient to cause the effect of different responses in the LGNd during saccades and during stimulation with a moving pattern while the eyes were stationary (Fischer et al., 1996).

Inactivation of the pretectum did not raise the spontaneous activity in X- and Y-cells, and during darkness no modulatory effect on LGNd relay cells could be seen even during saccades. From these two facts, it can be concluded that the influence of the pretectum on LGNd neurons is only effective if it occurs simultaneously with the retinal input to the relay cells. Indeed, saccadic eye movements and saccade-like stimulation, while the eye was

X cells during saccades
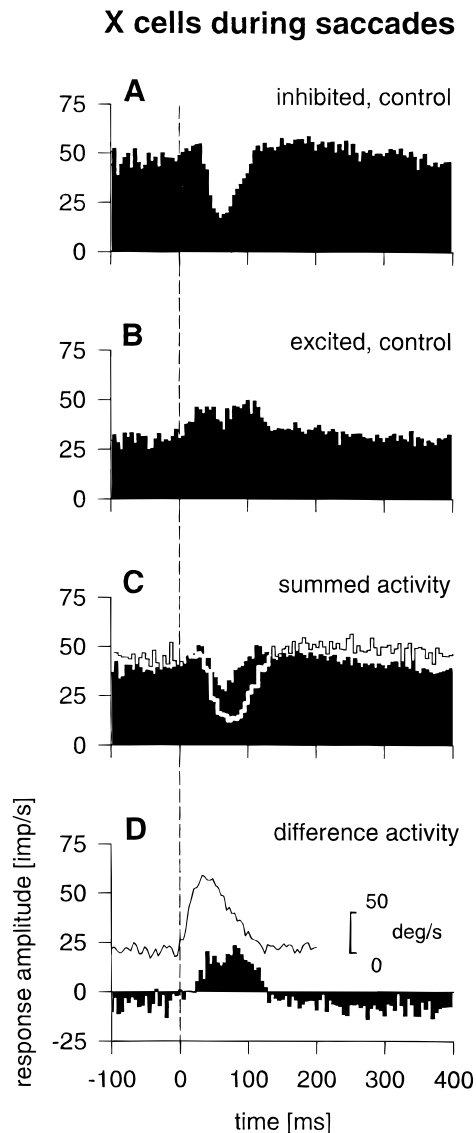
Fig. 8. Effect of pretectal inactivation onto the whole LGNd X-cell population recorded. The population responses of X-cells which were inhibited during control (A, $n = 32$) and which were excited during control (B, $n = 27$) were summed in C. The trace in C represents the population response of X-cells during pretectal inactivation (identical with Fig. 5B). D: Histogram of the difference activity of all recorded X-cells during control and pretectal inactivation. The curve above the histogram indicates the mean velocity of 100 saccades measured during the recordings. Binwidth of histograms: 5 ms.

stationary, had roughly similar effects on LGNd relay neurons in the control situation as well as during pretectal inactivation (see Figs. 5, 6, and 9). This is consistent with the results of Schmidt (1996) that LGNd-projecting pretectal neurons were activated by both, saccades and sudden stimulus shifts, the velocity of which were comparable with our saccade-like movement.

However, we found differences in response parameters between control and PTI situation, i.e. the strength of inhibition in X-cells and the peak width at half-height in Y-cells, that were almost exclusively restricted to saccade-induced neuronal activity and did not appear during saccade-like visual stimulation while the eye was stationary as shown in Figs. 7 and 10. It has been reported by

Schmidt (1996) that LGNd-projecting pretectal neurons had shorter latencies and increased peak activities during saccades compared to those observed during pure visual stimulation. These context-specific response properties of pretectal cells may be responsible for the LGNd relay cell responses especially regarding the strength of inhibition (X-cells) and the peak width at half-height (Y-cells) during saccades as compared to saccade-like stimulation.

Excited relay neurons

Thalamic relay cells are able to fire in a burst and in a tonic mode due to different voltage-dependent membrane conductances of the cells (Jahnsen & Llinás, 1984*a,b*; Lo et al., 1991; Huguenard & McCormick, 1992; McCormick & Huguenard, 1992; Guido et al., 1995). The burst mode is characterized by clusters of action potentials with interspike intervals of <4 ms and the bursts are separated by silent periods of more than 50 ms. During the tonic mode, the relay cells fire with a steady stream of action potentials. The burst mode was originally observed during certain phases of sleep, but burst responses were described by Guido and Weyand (1995) also in the behaving cat. They have found bursts about 150 ms or more after saccades and during the first phase of fixation when the cat was visually stimulated with a drifting grating. The cells changed to a tonic mode after the first cycle of the grating had passed the receptive field.

The excitatory responses we observed mostly occurred when cells showed firing response behavior of the tonic mode. Very rarely we measured interspike intervals of <4 ms during the initial phase of the visual stimulation. This may be due to the fact that the cats were not required to fixate a target but were allowed to make spontaneous eye movements. We think that the saccade-related excitatory responses of our LGNd cells were mainly generated in the tonic mode. This suggests that there may be a linear relationship between neuronal activity and stimulus parameters which is not the case in the burst mode (for review see Sherman, 1996). The bursts after saccades described by Guido and Weyand (1995) showed a far too long latency to add any influence to the saccade-related activity described in this study.

Other inactivation studies

The effect of inactivation of pretectal nuclei on LGNd relay cell responses in the anesthetized cat was tested by Funke and Eysel (1995). Similar to our results they described different effects on X- and Y-cells. Due to the lack of eye movements in the anesthetized preparation, they stimulated pretectal neurons with a grating moving in the background while stimulating the receptive field of a LGNd neuron and found a reduced inhibition in X-cells during inactivation of the pretectum. This is in contrast to our study which reveals a stronger inhibition of X-cells during pretectal inactivation. We think two explanations are possible. First, the visual background stimulation of pretectal neurons cannot be compared with saccadic eye movements in the awake cat. The stronger inhibition of X-cells during inactivation of the pretectal area in our study may result from blockade of the disinhibition induced by pretectal input during saccades in the control situation. Second, Funke and Eysel (1995) moved the background grating with a velocity of 50 deg/s which may be not sufficient to drive LGNd-projecting pretectal neurons since Schmidt (1996) has shown that these cells could not be activated by slow stimulus movements of 10 deg/s but only by sudden stimulus shifts of at least 400 deg/s or during saccades. The same reasons may be true for why the authors did not observe any effect on Y-cells.

saccades
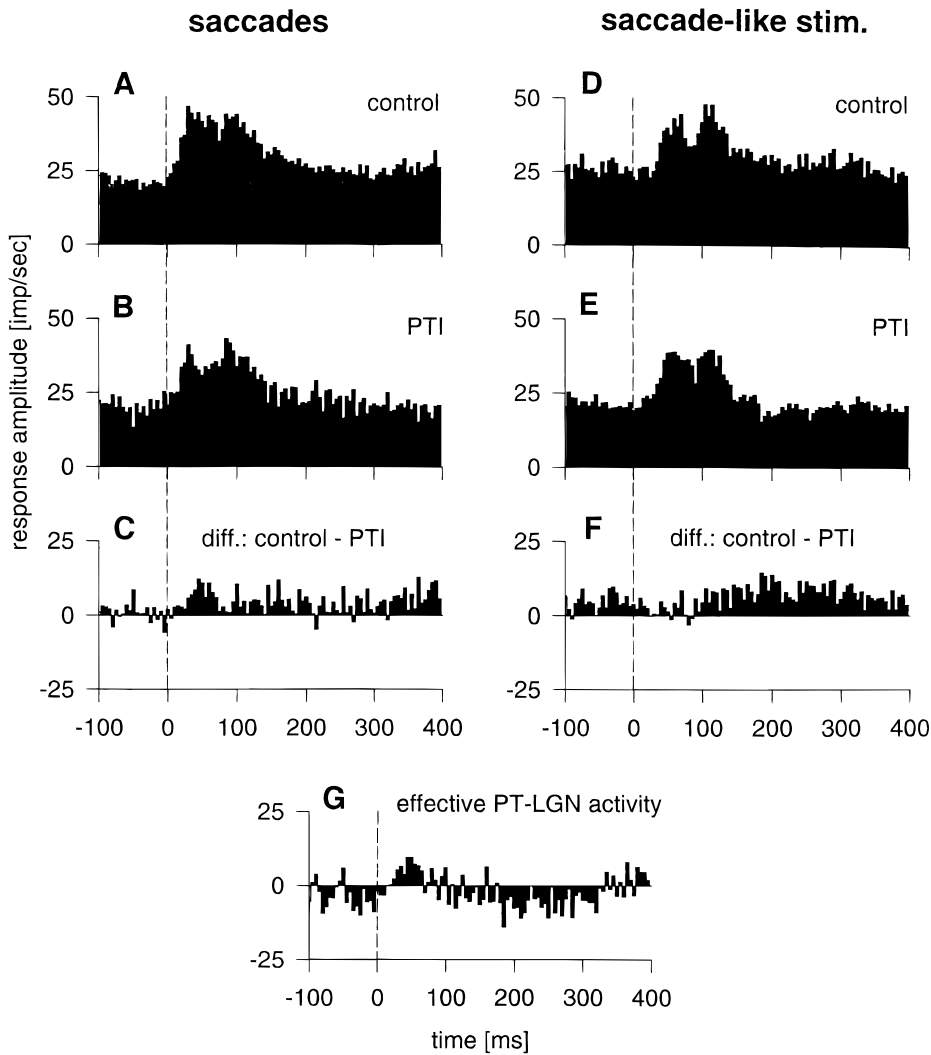
saccade-like stim.

Fig. 9. Population response of LGNd Y-cells during saccades (A–C) and during visual stimulation with a random-square pattern moving in a saccade-like fashion (D–F). Histograms are aligned to saccade onset (time = 0 ms, vertical line) in the left panel and to stimulus onset in the right panel. Population responses during the control situation (A,D; *n* = 48 cells) are compared with responses during inactivation of the pretectum (B,E; *n* = 27 cells) and the difference histograms of these two situations are given (C,F). The summed histogram of the effective PT-LGN activity in G is the difference of the histograms in C and F. Binwidth of histograms: 5 ms.

Functional considerations

The results of this study can be functionally interpreted in two ways. First, the observed neuronal behavior may be involved in the phenomenon of saccadic suppression. The main effect of the temporary inactivation of the pretectum was an inhibition of all recorded X-cells during saccades in light. Since the cat pretecto-geniculate projection is inhibitory and most likely terminates on inhibitory interneurons, this projection mediates a disinhibition onto the retinal input of X-cells. Also inhibitory neurons of the perigeniculate nucleus may be involved in this disinhibition (for review see Sherman & Koch, 1990). Because during saccades the excitatory X-cells are more strongly excited and the inhibitory X-cells are less inhibited as a result to the pretectal input, the influence of the pretectum on X-cells can be interpreted as a global modulatory effect on the geniculate relay. During a global activity increase of all X-cells, the activity of distinct groups of neurons, which may represent certain stimulus features, is lost. Thus, during saccadic eye movements X-cells are affected in a way which may lead to a functional disconnection from cortical areas.

A further source for an inhibition of X-cells may be mediated by Y-cells *via* the intrinsic connectivity of the LGNd itself (Singer & Bedworth, 1973; Mastronarde, 1992). During inactivation of the pretectum, a saccade-induced Y-cell response may lead to an additional activation of inhibitory interneurons which results in a strong inhibition of X-cells. This, however, is in contrast to our observation of disinhibition of X-cells. Though it is unknown whether saccadic suppression is paralleled by an inhibition or by a disinhibition of X-cells, it may be that a saccade-induced global disinhibition of LGNd X-cells may counteract an inhibition mediated by Y-cells.

We cannot rule out the possibility that some of our recordings may be from interneurons. In a study on this topic, Dubin and Cleland (1977) very rarely encountered intrageniculate interneurons, i.e. only 5.7% of their recorded cells. Since we used very similar recording techniques, the number of possibly recorded interneurons should not be higher in our study. However, if an interneuron is inhibited during saccadic eye movements, it should contribute to the above-described global modulatory effect on X-cells in the way that the inhibitory strength to X-cells is lowered. Thus, again excitatory X-cells should be more strongly excited whereas inhibitory X-cells should be less inhibited.

The only effect of the inactivation of the pretectum on Y-cells was a smaller peak width at half-height of responses to saccades. This is in accordance with a study of Fukuda and Stone (1976) who showed differential inhibitory influences on X- and Y-cells

peak width at half height

peak amplitude

latency to excitation
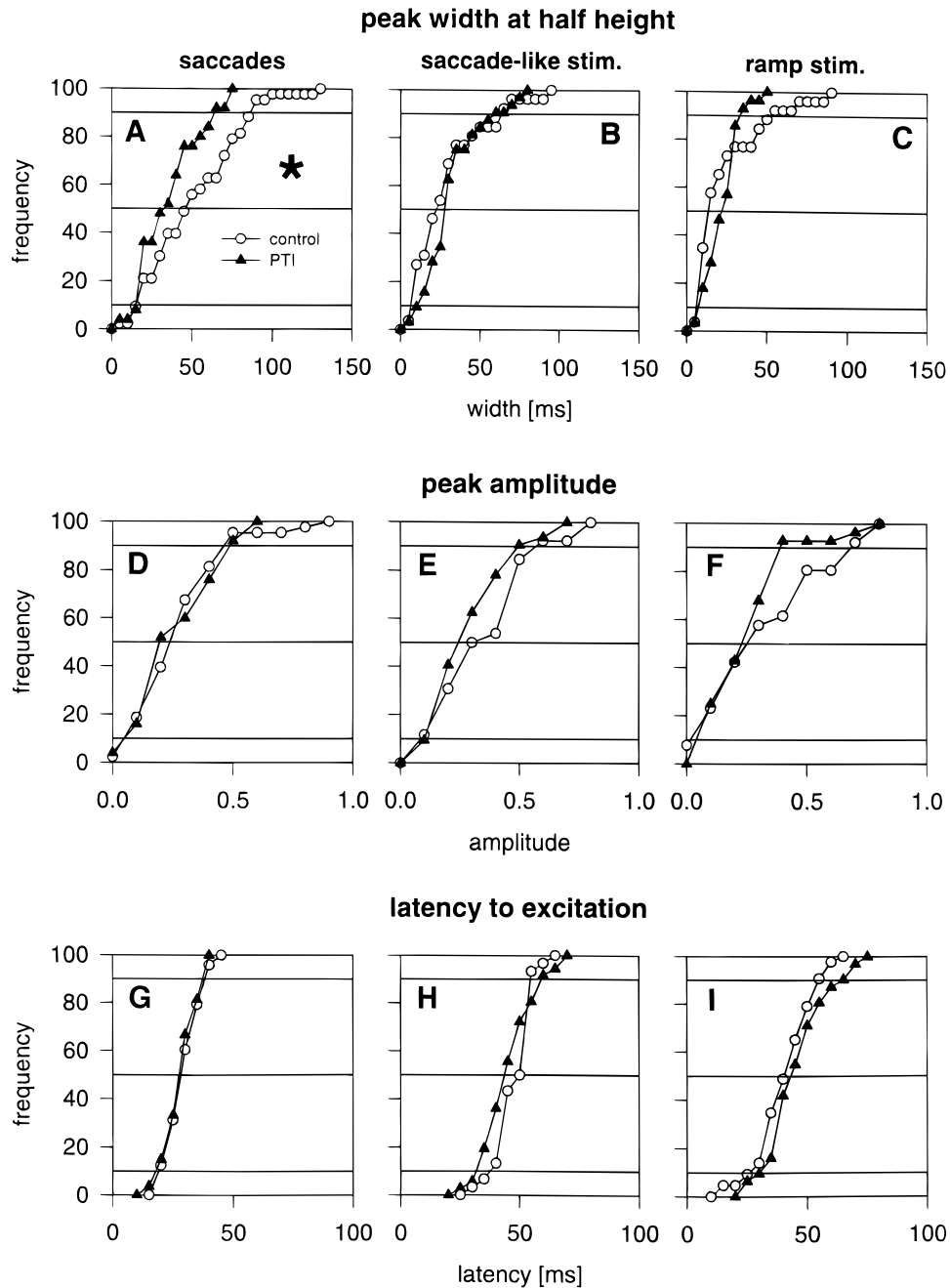
Fig. 10. Cumulative diagrams of three parameters of Y-cells which were excited during control situation (open circles) and during pretectal inactivation (closed triangles). Shown are the peak width at half-height (A–C), the peak amplitude (D–F), and the latency to excitation (G–I). Conventions are as in Fig. 7. The asterisk marks significant difference between control situation and pretectal inactivation (Mann-Whitney rank sum test, $P < 0.05$). Values were derived from 48 cells during control and 27 cells during PTI.

and that Y-cells were quantitatively less strongly inhibited than X-cells. Since the existence of Y-interneurons is a hotly debated question (e.g. Sherman & Friedlander, 1988), their contribution to the observed effect remains unknown. Another inhibitory connection on Y-cells involves the PGN, which receives Y-cell collaterals which in turn inhibit LGNd Y-cells. This inhibitory input may also be disinhibited by the pretecto-geniculate projection during saccades. When the pretectum is inactivated by muscimol, then Y-cells may be inhibited *via* the PGN. Since this is a feedback inhibition, it does not affect the whole neuronal activity during saccades but

only the activity after an initial response. This may be reflected in a shortened peak width at half-height of Y-cell responses during pretectal inactivation.

This study reveals a disinhibitory effect of the pretectum on LGNd relay cells during saccades. If the LGNd is the first site that plays a role in saccade-induced suppression of perception, it is obvious that this suppression is not mediated by an inhibition of neuronal activity. In contrast, the activity of the LGN-projecting pretectal cell population excited during saccades (Schmidt, 1996) inhibits LGNd interneurons which leads to a reduced inhibitory influence on the

relay cells. This may be sufficient to interrupt the relay of specific visual information to the cortex and may contribute to the perceptional effect of raised discrimination thresholds during saccades.

The different effects of pretectal inactivation on LGNd X- and Y-neurons is in accordance with a study of Burr et al. (1994) who found different psychophysical effects for the magnocellular and parvocellular pathway. However, they concluded that the saccadic suppression was confined to the magnocellular pathway. This is consistent with psychophysical results in humans which revealed a loss of motion perception which is thought to be associated with the Y-channel (see reviews of Matin, 1974; Volkmann, 1986). These observations are in contrast to our result of a more affected X-channel during pretectal inactivation. Our study was carried out in cats in which the pretecto-geniculate projection terminates exclusively on LGNd interneurons (Cucchiaro et al., 1993). In primates, however, pretectal fibers have been demonstrated to contact local interneurons as well as relay cells (Feig & Harting, 1994). This anatomical variation may result in a functional difference which accounts for the discrepancy between the studies.

Any contribution of the pretecto-geniculate input to saccadic suppression requires some, so far hypothetical, computation at higher, cortical levels. The prevailing effect, e.g. the selective influence of the pretectal inactivation onto LGNd X-cells, leads to the suggestion that another, more straightforward mechanism than saccadic suppression may be the result of this process. It has been proposed that a main effect of saccadic eye movements is a clearing of the visual screen for the next image (Jung, 1972) and that at the end of a saccade the inhibitory gradients, that have been built up during the saccade-induced retinal image shift, should be erased (Singer, 1977). This could be achieved by a diminishing of inhibitory influence on relay cells. Singer suggested the reticular control system and other brain-stem sources to be involved in this process of diminishing. Indeed, we have found a disinhibitory effect of the pretecto-geniculate projection on LGNd relay cells which resulted in a reduction of inhibition and, thus, led to an excitation of cells mainly of the X-, in part of the Y-channel.

Furthermore, Singer (1977) proposed that the reduction of the inhibitory coupling between retinogeniculate cells leads to lowered transmission thresholds and to a maximal broadening of the band-pass properties of individual channels. Our analysis revealed that the X-cells were more affected by a transient inactivation of the pretectum than the Y-cells. Thus, the disinhibitory effect of the pretecto-geniculate projection led to an excitation of X-cells at the end of saccades and may have resulted in a facilitation of the X-channel after saccades.

In psychophysical experiments, postsaccadic facilitation of a perceived image was reported by Burr and Morrone (1996). They found a postsaccadic enhancement of sensitivity for equiluminant, i.e. colored, stimuli. This raised sensitivity for colored stimuli, which is mediated *via* the parvocellular channel, is in accordance with our result of selective disinhibition and, thus, excitation of X-cells at the end of saccades. Perhaps especially X-cells have lowered transmission thresholds just after saccades which may lead to a higher sensitivity for colored stimuli during this period. If so, the LGNd could be regarded as a first site in which mechanisms that result in postsaccadic facilitation occur.

Facilitation of neuronal response in the LGNd that could be interpreted as postsaccadic enhancement was also observed by Lal and Friedlander (1990). They reported that this effect could take place *via* an afferent eye movement signal which may be an additional mechanism for postsaccadic facilitation to the one mediated by the pretecto-geniculate pathway.

Further evidence for an exceptional effect of pretectal cells on the X-channel may be derived from experimental results of Deubel et al. (1996). They have blanked a saccade target in a certain time window after the saccade with the result that a target displacement during the saccade was better detected than during fixation. They interpreted these findings as a saccadic enhancement of localization ability. The analysis of the localization of small objects is believed to be mediated by the X-channel. Thus, our findings of a preferred disinhibition of X-cells during saccades fits well with the psychophysical results of postsaccadic facilitation of the localization ability.

Our finding that inactivation of the pretecto-geniculate pathway leads to selective disinhibition of X-cells provides a model for postsaccadic facilitation. Further experimental evidence is needed regarding whether the effects reported in the LGNd are further processed in cortical areas.

References

BALLAS, I. & HOFFMANN, K.-P. (1985). A correlation between receptive field properties and morphological structures in the pretectum of the cat. *Journal of Comparative Neurology* **238**, 417–428.

BURR, D.C., MORRONE, M.C. & ROSS, J. (1994). Selective suppression of the magnocellular visual pathway during saccadic eye movements. *Nature* **371**, 511–513.

BURR, D.C. & MORRONE, M.C. (1996). Temporal impulse response functions for luminance and colour during saccades. *Vision Research* **36**, 2069–2078.

CLELAND, B.G., DUBIN, M.W. & LEWICK, W.R. (1971). Sustained and transient neurones in the cat's retina and lateral geniculate nucleus. *Journal of Physiology* **217**, 473–496.

CUCCHIARO, J.B., BICKFORD, M.E. & SHERMAN, S.M. (1991). A GABAergic projection from the pretectum to the dorsal lateral geniculate nucleus in the cat. *Neuroscience* **41**, 213–226.

CUCCHIARO, J.B., UHLRICH, D.J. & SHERMAN, S.M. (1993). Ultrastructure of synapses from the pretectum in the A-laminae of the cat's lateral geniculate nucleus. *Journal of Comparative Neurology* **334**, 618–630.

DEUBEL, H., SCHNEIDER, W.X. & BRIDGEMAN, B. (1996). Postsaccadic target blanking prevents saccadic suppression of image displacement. *Vision Research* **36**, 985–996.

DUBIN, M.W. & CLELAND, B.G. (1977). Organization of visual inputs to interneurons of lateral geniculate nucleus of the cat. *Journal of Neurophysiology* **40**, 410–427.

FEIG, S. & HARTING, J.K. (1994). Ultrastructural studies of primate lateral geniculate nucleus: Morphology and spatial relationships of axon terminals arising from retina, visual cortex (area 17), superior colliculus, parabigeminal nucleus, and pretectum of Galago crassicaudatus. *Journal of Comparative Neurology* **343**, 17–34.

FISCHER, W.H., SCHMIDT, M., STUPHORN, V. & HOFFMANN, K.-P. (1996). Response properties of relay cells in the A-laminae of the cat's dorsal lateral geniculate nucleus after saccades. *Experimental Brain Research* **110**, 435–445.

FUCHS, A.F. & ROBINSON, D.A. (1966). A method for measuring horizontal and vertical eye movement chronically in the monkey. *Journal of Applied Physiology* **21**, 1068–1070.

FUKUDA, Y. & STONE, J. (1976). Evidence of differential inhibitory influences on X- and Y-type relay cells in the cats's lateral geniculate nucleus. *Brain Research* **113**, 188–196.

FUNKE, K. & EYSEL, U.T. (1995). Pharmacological inactivation of pretectal nuclei reveals different modulatory effects on retino-geniculate transmission by X and Y cells in the cat. *Visual Neuroscience* **12**, 21–33.

GUIDO, W. & WEYAND, T.G. (1995). Burst responses in lateral geniculate neurons of the awake behaving cat. *Journal of Neurophysiology* **74**, 1782–1786.

GUIDO, W., LU, S.-M., VAUGHAN, J.W., GODWIN, D.W. & SHERMAN, S.M. (1995). Receiver operating characteristic (ROC) analysis of neurons in the cat's lateral geniculate nucleus during tonic and burst response mode. *Visual Neuroscience* **12**, 723–741.

GUILLERY, R.W. (1969*a*). The organization of synaptic interconnections in the laminae of the dorsal lateral geniculate nucleus of the cat. *Zeitschrift für Zellforschung und mikroskopische Anatomie* **96**, 1–38.

GUILLERY, R.W. (1969*b*). A quantitative study of synaptic interconnections in the dorsal lateral geniculate nucleus of the cat. *Zeitschrift für Zellforschung und mikroskopische Anatomie* **96**, 39–48.

GUILLERY, R.W. (1971). Patterns of synaptic interconnections in the dorsal lateral geniculate nucleus of cat and monkey: A brief review. *Vision Research* **3**, 211–227.

HARTMANN, R. & KLINKE, R. (1976). A method for measuring the angle of rotation (movements of body, head, eye in human subjects and experimental animals). *Pflügers Archiv* (Supp.) **362**, R52.

HOFFMANN, K.-P. (1989). Control of optokinetic reflex by the nucleus of the optic tract in primates. *Progress in Brain Research, Vol. 80*, ed. ALLUM, J.H.J. & HULLIGER, M., pp. 173–182. Amsterdam: Elsevier Science Publisher.

HOFFMANN, K.-P., STONE, J. & SHERMAN, M. (1972). Relay of receptive-field properties in dorsal lateral geniculate nucleus of the cat. *Journal of Neurophysiology* **34**, 518–531.

HOFFMANN, K.-P. & SCHOPPMANN, A. (1981). A quantitative analysis of the direction-specific response of neurons in the cat's nucleus of the optic tract. *Experimental Brain Research* **42**, 146–157.

HUGUENARD, J.R. & MCCORMICK, D.A. (1992). Simulations of the currents involved in rhythmic oscillations in thalamic relay neurons. *Journal of Neurophysiology* **68**, 1373–1383.

ILG, U. & HOFFMANN, K.-P. (1991). Responses of monkey nucleus of the optic tract neurons during pursuit and fixation. *Neuroscience Research* **12**, 101–110.

ILG, U., BREMMER, F. & HOFFMANN, K.-P. (1993). Optokinetic and pursuit system: A case report. *Behavioural Brain Research* **57**, 21–29.

JAHNSEN, H. & LLINÁS, R. (1984*a*). Electrophysiological properties of guinea-pig thalamic neurones: An *in vitro* study. *Journal of Physiology* **349**, 205–226.

JAHNSEN, H. & LLINÁS, R. (1984*b*). Ionic basis for the electroresponsiveness and oscillatory properties of guinea-pig thalamic neurons *in vitro*. *Journal of Physiology* **349**, 227–247.

JUDGE, S.J., RICHMOND, B.J. & CHU, F.C. (1980). Implantation of magnetic search coils for measurement of eye position: An improved method. *Vision Research* **20**, 535–538.

JUNG, R. (1972). Neurophysiological and psychophysical correlates in vision research. In *Brain and Human Behaviour*, ed. KARCZMAR, A.G. & ECCLES, J.C., pp. 209–258. Berlin: Springer-Verlag.

KASPER, H. & HESS, J.M. (1991). Magnetic search coil system for linear detection of three-dimensional angular movements. *IEEE Transactions of Biomedical Engineering* **38**, 466–475.

KATO, I., HARADA, K., HASEGAWA, T., IGARASHI, T., KOIKE, Y. & KAWASAKI, T. (1986). Role of the nucleus of the optic tract in monkeys in relation to optokinetic nystagmus. *Brain Research* **364**, 12–22.

KUBOTA, T., MORIMOTO, M., KANASEKI, T. & INOMATA, H. (1987). Projection from the pretectal nuclei to the dorsal lateral geniculate nucleus in the cat: A wheat germ agglutinin-horseradish peroxidase study. *Brain Research* **421**, 30–40.

LAL, R. & FRIEDLANDER, M.J. (1990). Effect of passive eye movement on retinogeniculate transmission in the cat. *Journal of Neurophysiology* **63**, 523–538.

LEE, D. & MALPELI, J.G. (1994). Effects of oculomotor behavior on visually-evoked activity in the cat LGN. *Society for Neuroscience Abstracts* **20**, 61.11.

LO, F.-S., LU, S.-M. & SHERMAN, S.M. (1991). Intracellular and extracellular *in vivo* recording of different response modes for relay cells of the cat's lateral geniculate nucleus. *Experimental Brain Research* **83**, 317–328.

MARTIN, J.H. (1991). Autoradiographic estimation of the extent of reversible inactivation produced by microinjection of lidocaine and muscimol in the rat. *Neuroscience Letters* **127**, 160–164.

MASTRONARDE, D.N. (1992). Nonlagged relay cells and interneurons in the cat lateral geniculate nucleus: Receptive-field properties and retinal inputs. *Visual Neuroscience* **8**, 407–441.

MATIN, E. (1974). Saccadic suppression: A review and an analysis. *Psychological Bulletin* **81**, 899–917.

MCCORMICK, D.A. & HUGUENARD, J.R. (1992). A model of the electrophysiological properties of thalamocortical relay neurons. *Journal of Neurophysiology* **68**, 1384–1400.

MCCORMICK, D.A. & BAL, T. (1994). Sensory gating mechanisms of the thalamus. *Current Opinion in Neurobiology* **4**, 550–556.

MUSTARI, M.J. & FUCHS, A.F. (1990). Discharge pattern of neurons in the pretectal nucleus of the optic tract (NOT) in the behaving primate. *Journal of Neurophysiology* **64**, 77–90.

NABORS, L.B. & MIZE, R.R. (1991). A unique neuronal organization in the cat pretectum revealed antibodies to the calcium-binding protein Calbindin-D 28k. *Journal of Neuroscience* **11**, 2460–2476.

NODA, H. (1975*a*). Depression in the excitability of relay cells of lateral geniculate nucleus following saccadic eye movements in the cat. *Journal of Physiology* **249**, 87–102.

NODA, H. (1975*b*). Discharge of relay cells in lateral geniculate nucleus of the cat during spontaneous eye movements in light and darkness. *Journal of Physiology* **250**, 579–595.

SCHIFF, D., COHEN, B. & RAPHAN, T. (1988). Nystagmus induced by stimulation of the nucleus of the optic tract in the monkey. *Experimental Brain Research* **70**, 1–14.

SCHIFF, D., COHEN, B., BÜTTNER-ENNEVER, J. & MATSUO, V. (1990). Effects of lesions of the nucleus of the optic tract on optokinetic nystagmus and after-nystagmus in the monkey. *Experimental Brain Research* **79**, 225–239.

SCHMIDT, M. (1996). Neurons in the cat pretectum that project to the dorsal lateral geniculate nucleus are activated during saccades. *Journal of Neurophysiology* **76**, 2907–2918.

SCHMIDT, M. & HOFFMANN, K.-P. (1992). Physiological characterization of pretectal neurons projecting to the lateral geniculate nucleus in the cat. *European Journal of Neuroscience* **4**, 318–326.

SCHMIDT, M., LEHNERT, G., BAKER, R.G. & HOFFMANN, K.-P. (1996). Dendritic morphology of projection neurons in the cat pretectum. *Journal of Comparative Neurology* **369**, 520–532.

SHERMAN, S.M. (1993). Dynamic gating of retinal transmission to the visual cortex by the lateral geniculate nucleus. In *Thalamic Networks for Relay and Modulation*, ed. MINCIACCHI, D., MOLINARI, M., MACCHI, G. & JONES, E.G., pp. 61–79. Oxford: Pergamon Press.

SHERMAN, S.M. (1996). Dual response modes in lateral geniculate neurons: Mechanisms and functions. *Visual Neuroscience* **13**, 205–213.

SHERMAN, S.M. & KOCH, C. (1986). The control of retinogeniculate transmission in the mammalian lateral geniculate nucleus. *Experimental Brain Research* **63**, 1–20.

SHERMAN, S.M. & FRIEDLANDER, M.J. (1988). Identification of X versus Y properties for interneurons in the A-laminae of the cat's lateral geniculate nucleus. *Experimental Brain Research* **73**, 384–392.

SHERMAN, S.M. & KOCH, C. (1990). Thalamus. In *The Synaptic Organization of the Brain*, 3rd edition, ed. SHEPHERD, G.M., pp. 246–278. New York: Oxford University Press.

SINGER, W. (1977). Control of thalamic transmission by corticofugal and ascending reticular pathways in the visual system. *Physiological Review* **57**, 386–420.

SINGER, W. & BEDWORTH, N. (1973). Inhibitory interaction between X and Y units in the cat lateral geniculate nucleus. *Brain Research* **49**, 291–307.

STERIADE, M. & LLINAS, R. (1988). The functional states of the thalamus and the associated neuronal interplay. *Physiological Review* **68**, 649–742.

STERIADE, M., JONES, E.G. & LLINAS, R. (1990). *Thalamic Oscillations and Signalling*. New York: Wiley.

STERIADE, M., MCCORMICK, D.A. & SEJNOWSKY, T.J. (1993). Thalamocortical oscillations in the sleeping and aroused brain. *Science* **262**, 679–685.

THOMPSON, K.G., ZHOU, Y. & LEVENTHAL, A.G. (1994). Direction-sensitive X and Y cells within the A laminae of the cat's LGNd. *Visual Neuroscience* **11**, 927–938.

UHLRICH, D.J. & CUCCHIARO, J.B. (1992). GABAergic circuits in the lateral geniculate nucleus of the cat. In *Progress in Brain Research, Vol. 90*, ed. MIZE, R.R., MARC, R.E. & SILLITO, A.M., pp. 171–192. Amsterdam: Elsevier Science Publishers.

UHLRICH, D.J. & MANNING, K.A. (1995). Projection of individual axons from the pretectum to the dorsal lateral geniculate complex in the cat. *Journal of Comparative Neurology* **363**, 147–159.

VOLKMANN, F.C. (1986). Human visual suppression. *Vision Research* **26**, 1401–1416.

WAHLE, P., STUPHORN, V., SCHMIDT, M. & HOFFMANN, K.-P. (1994). LGN-projecting neurons of the cat's pretectum express glutamic acid decarboxylase mRNA. *European Journal of Neuroscience* **6**, 454–460.