Journal of Physiology (1990), 429, *pp.* 259—280
With 9 figures

Printed in Great Britain

PRIVATE INHIBITORY SYSTEMS FOR THE X AND Y PATHWAYS IN THE DORSAL LATERAL GENICULATE NUCLEUS OF THE CAT

By SIVERT LINDSTRÖM AND **ANDRZEJ WRÓBEL***

From the Department of Physiology, University of Göteborg, Box 33031, S-40033 Göteborg, Sweden

(Received 2 February 1990)

SUMMARY

1. Inhibitory connections of X- and Y-type principal cells in the cat's dorsal lateral geniculate nucleus were studied with intracellular recording techniques in barbiturate-anaesthetized animals. Cells were identified as principal cells by antidromic activation from the visual cortex and as X or Y types by their responses to visual stimulation.

2. Graded electrical stimulation was used to obtain selective activation of X and Y ganglion cell axons. The optic nerves were stimulated through ring electrodes behind the eye bulbs and the evoked nerve volley was monitored by an optic tract electrode. The nerve volley consisted of two well-segregated components, an early, low-threshold Y component and a late, high-threshold X component.

3. All principal cells received monosynaptic excitation and disynaptic feed-forward inhibition from optic nerve fibres. The excitatory and inhibitory post-synaptic potentials were evoked by Y axons in Y cells and by X axons in X cells. Thus, the feed-forward inhibitory pathway to principal cells is type selective.

4. Recurrent inhibition was evoked in all cells by antidromic activation of principal cell axons in the visual cortex. The recurrent inhibitory potentials had significantly shorter latencies in Y than in X cells but with considerable overlap between the two samples. This overlap presumably reflects a similar overlap in antidromic conduction times for X and Y principal cell axons.

5. Recurrent inhibitory potentials evoked in the orthodromic direction by optic nerve stimulation originated from Y axons in Y principal cells and from X axons in X cells as would be expected for a type-selective recurrent inhibitory pathway.

6. It is concluded that X and Y principal cells in the dorsal lateral geniculate nucleus have similar but functionally separate inhibitory circuits.

INTRODUCTION

It is widely agreed that cat retinal ganglion cells of X and Y types contact separate classes of principal cells in the dorsal lateral geniculate nucleus (dLGN) thereby defining their functional properties (Cleland, Dubin & Levick, 1971; Stone &

* **Present** address: Department of Neurophysiology, Nencki Institute of Experimental Biology, Pasteur 3, 02-093 Warsaw, Poland.

Hoffman, 1971; Sherman, 1985). The segregation of the X and Y pathways seems to be maintained at the first synapses of the visual cortex. Here most target cells receive selective excitation and inhibition from either X or Y geniculate fibres (Bullier & Henry, 1979; Ferster & Lindström, 1983; Ferster, 1990). In contrast to this segregation, it has been proposed in several studies that the intrinsic inhibitory connections of the dLGN are less specific. All types of inhibitory interactions have been reported: Y inhibition of X cells (Singer & Bedworth, 1973), X inhibition of Y cells (Burke, Burne & Martin, 1985; Bloomfield & Sherman, 1988) and a mixture of both (Hoffman, Stone & Sherman, 1972).

Most of these studies were completed before the inhibitory circuits of the dLGN were properly characterized. It is now known that principal cells receive two types of local inhibition; feed-forward inhibition via intrageniculate interneurones and recurrent inhibition via perigeniculate cells (Dubin & Cleland, 1977; Lindström, 1982; Ahlsdn, Lindström & Lo, 1985). Inhibitory neurones of both types have been identified and found to be activated from either the X or Y system (Dubin & Cleland, 1977; Ahlsén, Lindström & Lo, 1983; Lindström, 1983; Wróbel & Tarnecki, 1984; see, however, Sherman & Friedlander, 1988). This input selectivity of the interneurones suggested to us that the dLGN inhibition might be more specific than so far believed. Or could it be so that the output of one or both classes of interneurones is less type selective than their input?

In an attempt to answer this question we have recorded intracellularly from visually classified principal cells and tried to identify the afferent origin of both feed-forward and recurrent inhibitory postsynaptic potentials. The synaptic responses were evoked by electrical stimulation of the visual pathway and threshold and conduction velocity separation methods were used to determine whether X or Y ganglion cell axons were responsible for the effects. It will be shown that X and Y principal cells are equipped with similar but private inhibitory circuits. A brief preliminary report has been published (Lindström & Wróbel, 1984).

METHODS

Animal preparation. Experiments were performed on twenty-three young adult cats ([9—29 kg). Anaesthesia was induced with an alphaxalone—alphadolone mixture (Saffan, Glaxovet Ltd 12 mg/kg I.M.) followed by sodium pentobarbitone (Apoteksbolaget; 25—30 mg/kg i.v.). Additional small doses of pentobarbitone were given as needed to keep the animal in a state of slow-wave sleep as judged by EEG recordings. The animals were paralysed by gallamine triethiodide (Flaxedil, May and Baker Ltd), continuously infused with a bicarbonate-buffered Ringer—glucose solution. They were artificially ventilated through a tracheotomy with a positive end-expiratory pressure of 1—2 cmH_2O. Tidal volume was adjusted to maintain end-expiratory CO_2 at 3•53•7%. Temperature was kept at 38 ^{0}C. To reduce respiratory-linked movement of the brain the animals were suspended by a clamp placed on a midthoracic vertebrae and a pneumothorax was performed prior to the start of the recording. The corneas were covered with contact lenses of a curvature appropriate to focus them on a tangent screen 15 m in front of the cat. Pupils were fully dilated, accommodation paralysed and nictitating membranes retracted by local application of 1 % atropine and .10% neosynephrine.

Intracellular recordings. Intracellular recordings were obtained from dLGN principal cells with glass micropipettes filled with 3 M-sodium or potassium acetate. The electrodes had their tips broken back to a diameter of approximately 05 μ*m* giving a DC resistance of 15—30 MÙ. Electrodes filled with sodium acetate were exclusively used in later experiments since better intracellular

recordings were obtained with such electrodes. Presumably, the leakage of sodium ions from their small tips was too slow to induce a significant depolarization of the cells. The microelectrodes were advanced with a stepping motor micromanipulator through the overlying cortex down to the dLGN. After the beginning of each track the exposed cortex in the small craniotomy over the

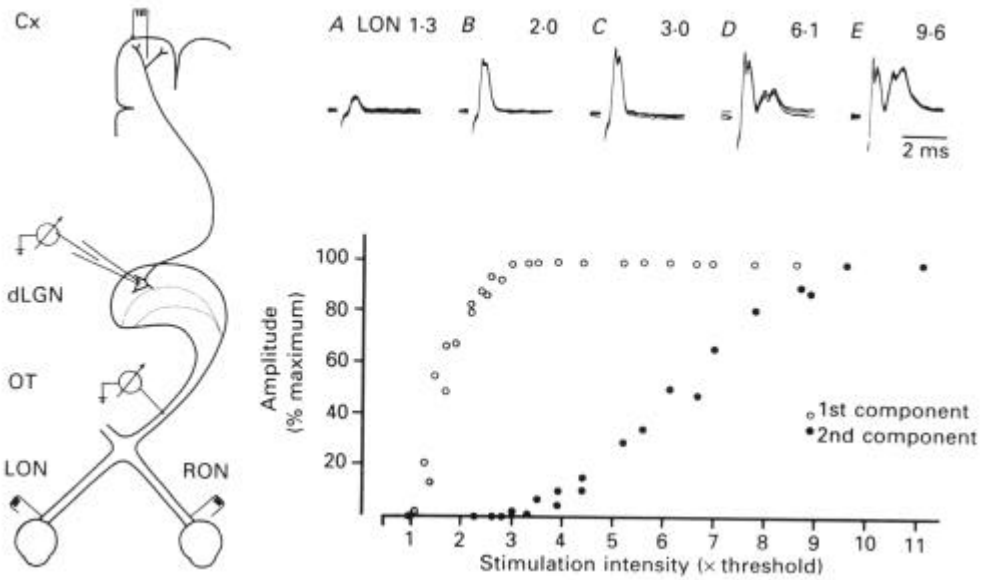

Fig. **1.** Threshold separation method used in the experiment. Diagram to the left shows stimulation (LON, OT, Cx) and recording (OT, dLGN) sites along the visual pathway. *A—F,* compound action potentials evoked in the right optic tract by left optic nerve (LON) stimulation. Negativity is upwards in nerve volley recordings of all figures. The stimulation intensity, indicated in multiples of threshold for the first detectable volley, was gradually increased to obtain the consecutive records in *A—F.* The nerve volley consists of two components with different thresholds and conduction times. In the diagram below the amplitudes of the 1st (Y volley) and 2nd (X volley) components are plotted against the stimulation intensity in multiples of threshold intensity. LON, left optic nerve; OT, optic tract; dLGN, dorsal lateral geniculate nucleus; Cx, primary visual cortex.

dLGN was covered by body-warm agar to reduce pulsations. In most experiments only the anterior half of the dLGN was explored but penetrations were spaced so that dLGN cells with inputs corresponding to retinal eccentricities from 1 to 55 deg were studied.

Principal cells were identified as such by their location in the dLGN, by their receptive field properties and by antidromic activation from the visual cortex. Only typical principal cells with clear all or nothing antidromic spikes of fixed latency were accepted for the study (cf. Figs 3 and 4). Most impaled principal cells were damaged by the penetration. To prevent injury discharges and to stabilize the recordings the cells were initially hyperpolarized by current injection through the recording electrode. Current injection was also routinely used to reverse or enhance postsynaptic potentials (PSPs). The fine electrodes were polarized by this procedure so reliable measurements oUDC membrane potentials could not be obtained. Despite this difficulty, it was still possible to measure latencies and thresholds of synaptic potentials quite accurately. For each class of cells we also obtained several stable penetrations lasting up to 1 h with 70—80 mV action potentials. Measured latencies in these cells were in the same range as for the rest of the population.

Electrical 8timulation procedures. Three stimulation sites were used to characterize the connectivity of the recorded principal cell as shown schematically in Fig. 1. For selective stimulation of Y and X axons we adapted the procedure of Bishop & McLeod (1954) with a

retrobulbar ring electrode, leaving, however, the eyebulbs intact for visual stimulation. The optic nerves (ON) were layed in a U-shaped silver-wire electrode melted into a piece of protecting plastic tube and covered by an additional wire to close the loop. The ring electrode was used as the cathode and a silver wire hooked through the conjunctiva behind the lower eye lid served as the anode. The purpose of this arrangement was to generate a uniform electrical field through the optic nerve at the level of the cathode and thereby to ascertain optimal threshold separation between the Y and X fibres.

To place the ring electrode around the optic nerve the zygomatic arch was first removed together with the retrobulbar fat surrounding the nerve. The eye was then gently rotated medially and downwards and the electrode slipped between the recti muscles and around the nerve. The eye bulb was allowed to rotate back to the original position while the flexible wire was carefully adjusted to avoid any external pull on the nerve. Finally the exposed tissue was covered with cotton wool soaked in warm saline. During the entire dissection and placement procedure great care was taken not to disturb the blood supply of the retina or the optic nerve.

The optic nerve was stimulated with an isolated constant-current stimulator giving rectangular pulses of variable amplitude and duration. We preferred short pulses (005 ins) since they gave the best threshold separation between the fibre groups. With these short pulses the threshold intensity varied between 05 and 15 mA with maximal (X) response attained at 6—10 times threshold. In our routine procedure the stimulation intensity was gradually increased to activate first the thickest, fast conducting axons and then progressively more slowly conducting fibres. The evoked nerve volley was monitored with a unipolar tungsten electrode placed in the optic tract (OT) 3—5 mm from the optic chiasm. The same electrode could be used to stimulate the optic tract fibres.

The conduction velocities of stimulated fibres were calculated from the difference in latencies of synaptic potentials evoked by ON and OT stimulation and from the conductance distance measured after dissection of the nerves at the end of the experiment. All latency measurements were taken at a stimulation intensity about two times threshold for the appropriate potential. With this procedure the time of spike initiation at the stimulation site (about 02 ins; Lindström, 1982) is not included in our estimates which explains our comparatively high conduction velocity values (cf. Stone, Dreher & Leventhal, 1979). For cortex stimulation an array of seven tungsten electrodes, 2 mm apart, was placed 2 mm from the mid-line in the anterior—posterior direction between A6 and P6 and inserted to a depth of 2—3 mm below the surface of the cortex (Cx). Any of these electrodes could be used for antidromic activation of principal cell axons or to record evoked potentials and an EEG.

Visual stimulation. Receptive fields were plotted against a mesopic background on a tangent screen V5 m in front of the cats. The principal cells were first classified as X or Y using extracellular recordings. A number of visual tests were used for the classification (in order of importance):

receptive field centre size in relation to eccentricity (Hoffman *et al.* 1972; Cleland, Harding & Tulunay-Keesey, 1979); spatial resolution tested with moving gratings of different spatial frequencies (Cleland *et al.* 1971; So & Shapley, 1979; Troy, 1983); the presence of a 'null position' (Enroth-Cugell & Robson, 1966) in a spatial summation test with contrast reversal of two hemifields. Once classified (only a few cells required the whole battery of tests for type specification) the cell was impaled and the receptive field remapped using the auditory response of unitary EPSPs as an indicator. To establish the eccentricity the area centralis and optic disc positions were mapped with each penetration. The expected progression of receptive field positions (Sanderson. 1971) and the changes of ocular preference at the dLGN laminar border made histological confirmation unnecessary.

RESULTS

Selective activation of X and Y ganglion cell axons

This study required a simple and reliable procedure for selective activation of X and Y inputs to dLGN principal cells in order to identify the source of their feed-forward and recurrent inhibitory postsynaptic potentials (IPSPs). It occurred to us that the well-established difference in axonal diameter of X and Y ganglion cells might be utilized for selective electrical activation of the two fibre groups. The thick Y fibres should have lower thresholds for stimulation than the thinner X fibres,

provided that the electrical field is evenly distributed through the nerve. This later condition was achieved using a ring electrode around the optic nerve behind the eye bulb (Bishop & McLeod, 1954). The records in Fig. 1*A—E* show the result of graded stimulation with such an electrode. The evoked nerve volley, monitored by an

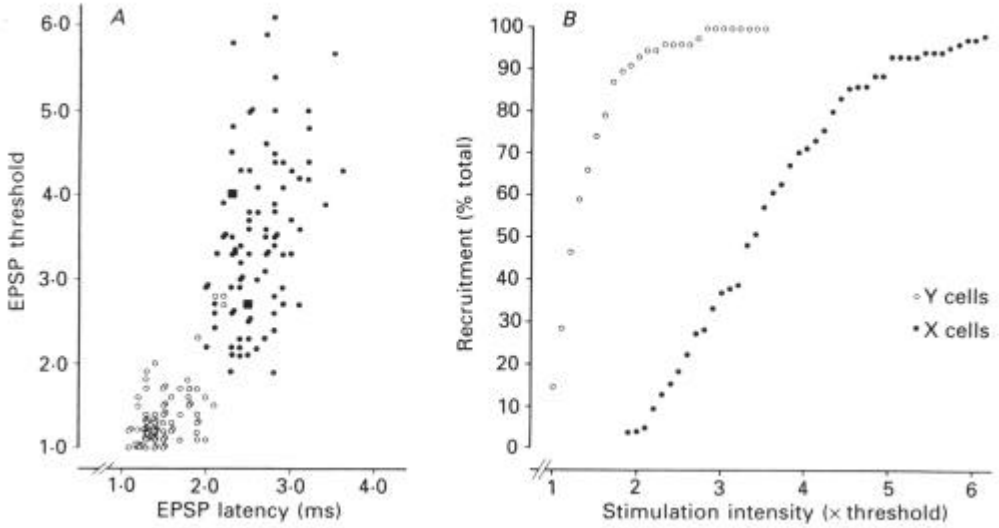

Fig. 2. *A,* threshold—latency relation for unitary EPSPs evoked in visually identified dLGN principal cells. The EPSPs were evoked by electrical stimulation of the optic nerves behind the eye bulbs. The thresholds are expressed in multiples of threshold for the nerve volley. The latency was measured from the onset of the stimulus shock artifact using an intensity of about two times threshold for the unit. Only the shortest latency unit was plotted for cells with multiple inputs. In this and following figures: **,** X cells *(n* = 89); , Y cells (n= 77). Two X cells with atypical inhibition are indicated by squares. *B,* cumulative recruitment of X and Y unitary EPSPs based on the same sample as in *A*. The relative number of recruited unitaries as a percentage of the total X or Y subsample has been plotted against the stimulation intensity.

electrode in the contralateral optic tract, consisted of an early, low-threshold component from fast conducting fibres *(A—C)* and a late, high-threshold component from more slowly conducting fibres *(D* and *E;* Bishop & McLeod, 1954). The estimated conduction velocity for the fastest fibres in each component was 93 and 31 m/s indicating that they represented the activation of Y and X axons respectively. A third later component appeared at even higher intensities (not illustrated); this component presumably originated from W fibres.

The stimulation intensity required to obtain a maximal first component varied considerably between experiments (range 1•7—3•O times nerve threshold) and so did the threshold intensity for the second component (range 2•0—4•0). Part of this variability can be ascribed to difficulties in determining the absolute nerve threshold. The threshold separation was quite good in most animals, however, with minimal overlap between the two components. The plot in the lower right diagram of Fig. 1 is typical for the contralateral, left optic nerve (LON). In this case the first

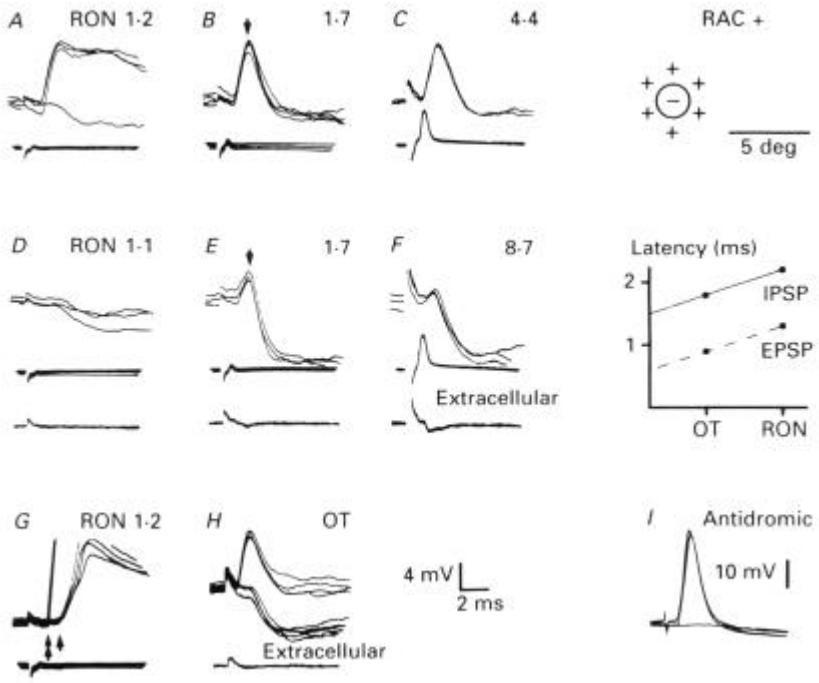

Fig. 3. Intracellular recordings of EPSPs and feed-forward IPSPs in a Y principal cell. The recordings are from an off-centre cell activated from the ipsilateral right eye. Its receptive field is plotted in the upper right corner (RAC, right area centralis). Upper trace in *A*—*G* shows intracellularly recorded synaptic potentials evoked by stimulation of the right optic nerve (RON) at the indicated intensities (in multiples of nerve threshold). The second trace shows the nerve volley recorded simultaneously by the optic tract electrode. Extracellular field potentials evoked at the same stimulation intensities and recorded with the microelectrode just outside the cell are shown below in *D*—*F* and *H*. The responses in *D*—*F* were obtained with the cell artificially depolarized by the injection of a steady positive current (2 nA) through the recording microelectrode. This depolarization enhanced the IPSPs at the expense of the EPSPs. The response in & shows reversed IPSPs evoked at EPSP threshold with the cell hyperpolarized by a 10 nA negative current. Only two stimuli evoked an EPSP as shown by the first truncated, upward deflection (double arrow in *U).* The small arrows in *B, E* and *U* point to the IPSP onset. The corresponding PSPs evoked from the optic tract (OT) at EPSP threshold are shown in *H* and the antidromic spike evoked at threshold by visual cortex stimulation in *I.* In the extrapolation diagram the PSP latencies from the right optic nerve and the optic tract have been plotted against the conductance distance to the cell in the dLGN. The estimated conduction velocity was the same (60 m/s) for the optic nerve fibres responsible for the EPSP and the IPSP. Voltage calibrations between *H* and *I* refers to intra- and extracellular recordings in *A*—*H;* time calibration to all records. Further details in the text.

component was maximal at about three times threshold for the nerve at which intensity the second component was just beginning to grow. For the ipsilateral, right optic nerve (RON) the separation was slightly worse but only in one experiment did the range of overlap include more than 10% of the fibres as judged by the size of the nerve volleys.

To verify that the first and second components originated from Y and X fibres we plotted the threshold against the latency for unitary excitatory postsynaptic potentials (EPSPs) in a number of visually characterized principal cells (Fig. *2A)*. Each unitary EPSP represents the synaptic effect of a single activated optic nerve fibre (see below). For cells with more than one input fibre only the first activated EPSP was included. As expected, the unitary EPSPs had lower thresholds and shorter latencies in Y cells ( ) than in X cells ( ). In terms of latency there was very little overlap between the two populations. Since the synaptic delay in the dLGN is the same for X and Y fibres (0 3 ms; Lindström, 1982; Wang, Cleland & Burke, 1985), the different latencies mainly reflect the different conduction velocities of the afferent optic nerve fibres.

There appears to be a greater overlap for threshold intensities between the two fibre groups but most of this results from the pooling of data from several experiments in the same plot. As pointed out above, the threshold ranges varied somewhat from cat to cat and even between the two optic nerves in the same animal. When the threshold of a particular unitary EPSP was compared with the simultaneously recorded nerve volley the unitaries of Y cells had thresholds within the intensity range of the first component and those of X cells within the range of the second. The cumulative recruitment curves in Fig. 2B show that even for the pooled sample there were quite few units in the threshold overlap zone. Not more than 10 % of the unitaries in Y cells had higher thresholds than the lowest threshold unitaries of X cells. These results clearly demonstrate that the first nerve volley component originates from Y axons and the second from X axons. Accordingly, the first low-threshold component will be referred to as the 'Y volleys and the second component as the 'X volley'.

Feed-forward inhibition

Intracellular recordings of EPSPs and IPSPs were obtained from 178 principal cells classified as X and Y cells on the basis of visual testing (99 X cells and 79 Y cells). Sixteen additional cells had mixed excitatory inputs from both X and Y axons. These cells are not included in the main material but will be discussed separately below. Of the main sample 74 cells were activated from the ipsilateral and 104 from the contralateral eye. Twelve of the latter cells, all with Y type excitation, were recorded in the upper C layer. These C layer cells were in all studied aspects, except lamina position, similar to Y cells in lamina A and Al. They are therefore included in the material without specific labels. All principal cells received monosynaptic excitation and disynaptic feed-forward inhibition following electrical stimulation of one of the optic nerves. For identified Y cells both the EPSPs and the IPSPs were recruited with the first, Y component of the nerve volley while for X cells the PSPs came with the second, X component.

Y cells

Typical recordings from a Y principal cell are shown in Fig. 3. The cell had a large off-centre receptive field and no 'null position' in the linearity test. It responded well to a large bar, rapidly moved through its receptive field and produced 'on — off' bursts to a small light spot flashed at the centre—surround border. Its excitatory

input came from a single retinal ganglion cell in the ipsilateral right eye. The unitary nature of the excitatory input was easily verified with electrical stimulation of the optic nerve. With the stimulation intensity adjusted to the threshold level of the responsible fibre, an EPSP was evoked in an all-or-nothing manner. The record of Fig. *3A* shows four superimposed responses, three of which gave rise to an EPSP of the same amplitude. One stimulus failed to activate the axon and thus no EPSP was evoked. The data underlying the threshold—latency plot in Fig. 2A were obtained from unitary EPSPs identified in this way.

The threshold intensity for the unitary EPSP in Fig. *3A* was P2 times threshold for the most sensitive fibres in the optic nerve, i.e. in the lower threshold range for Y fibres. No additional EPSP component was added when the stimulus strength was increased to obtain a maximal Y volley (Fig. *3B)* or further, to recruit also X fibres (Fig. *3C).* The EPSP decay became faster when the stimulus strength was increased from the threshold level *(A)* to that of a maximal Y volley *(B).* This change was due to the recruitment of feed-forward IPSPs.

To better reveal the IPSP the cell was steadily depolarized by current injection through the recording microelectrode, a procedure that enhances the IPSP at the expense of the EPSP (Fig. *3D—F).* The records in *D* show that already a stimulus of 11 times threshold for the optic nerve evoked a small disynaptic IPSP. This low-threshold IPSP can be seen also in the failure trace in *A.* Maximal IPSP amplitude was attained at a stimulus strength below threshold for the X volley *(E).* No further IPSP increase was observed when the intensity was adjusted to evoke a maximal X volley *(F).* This lack of additional effect was not caused by the JPSIP approaching its reversal level. A much larger summed IPSP could be obtained by double stimulation (not illustrated).

The inhibitory response in *E* was a compound IPSP. When the stimulus strength was carefully graded within the Y range the LPSP grew in at least five discrete steps with different thresholds (not illustrated). We take this to indicate that at least five ganglion cells with axons of different thresholds contributed to the inhibitory response, each one recruiting a new inhibitory interneurone. This figure is a minimum number. Our analysis of the number of unitary IPSPs was much hampered by the on-going 'spontaneous' activity of the input ganglion cells. For this principal cell we could not, for instance, determine whether its excitatory input ganglion cell also contributed to the inhibitory response. This was clearly the case for many other principal cells (cf. below). Even with these limitations it can be concluded that the feed-forward inhibitory pathway displayed more convergence than the excitatory one to the cell.

The latency of the PSPs were determined at twice threshold for each response and measured from the onset of the stimulus shock artifact to the start of the PSPs. The extracellular field potential (lowermost traces in Fig. *3D—F)* was always subtracted if it was large enough to influence the measurements. For the illustrated cell it was not necessary. The inflexion point near the peak of the EPSP was taken as the onset of the IPSP (arrow, Fig. *3B* and K), since this was a clear pivot point for the IPSP when the membrane potential was changed (cf. Fig. 3A, *B* and K). That this point represents the true onset of the LPSP was confirmed by reversing the IPSP with a strong hyperpolarizing current (Fig. 3.0). The reversed IPSP was evoked at

threshold for the EPSP and the EPSP failed in all but two traces (double arrow). This failure makes the latency difference between the two PSPs quite evident. The IPSP latency from the optic nerve was 0·9 ms longer than that for the EPSP (2·2 ms compared to 1·3 ms). The same latency difference was also found when the two responses were evoked from the optic tract (Fig. *3H)*, suggesting that it represented the interneuronal delay in the inhibitory pathway (Lindström, 1982).

The measured latency from two sites of stimulation allowed us to apply an extrapolation procedure to estimate the axonal conduction velocity and local geniculate delay (Ferster & Lindström, 1983). In the diagram of Fig. 3 the latency of the PSPs has been plotted against the conductance distance from the stimulation sites to the recording site in the dLGN. The slope of the lines connecting the optic nerve and optic tract points give the conduction velocity which was the same (60 m/s) for the axons giving the EPSP and IPSP. This value is within the conduction velocity range for Y axons (see above).

The intercept of the lines (0·6 ms for the EPSP and 1·5 ms for the IPSP) corresponds to the geniculate delay plus the spike initiation time at the site of stimulation. From our earlier work it is known that these intercept values correspond to monosynaptic and disynaptic connections (below 1 ms for monosynaptic and between 1 and 2 ms for disynaptic). The spike initiation time was estimated with a similar extrapolation procedure but monitoring the nerve volley in the dLGN rather than PSPs (not illustrated). It was 0·2 ms, a quite typical value (Lindström, 1982). The remaining time to the EPSP onset (0·4 ms) includes a true synaptic delay of about 0·3 ms (Lindström, 1982; Wang *et al*. 1985) and a small extra delay due to slowing of the impulse in the terminal branches of the ganglion cell axon.

The IPSP value includes an additional synaptic delay of 0·3 ms plus the time for EPSP rise to spike threshold in the interneurone (Lindström, 1983) and its spike propagation. Together this sequence easily accounts for a delay of 0·9 ms in the inhibitory pathway. There is certainly no time for a recurrent inhibitory loop which would have required another 0·5–1 ms (Lindström, 1982). This somewhat elaborate treatise serves to emphasize that the early IPSP evoked in this and all other Y cells by optic nerve stimulation were indeed mediated by a disynaptic feed-forward pathway.

One more technical aspect deserves to be mentioned in relation to Fig. 3. A larger IPSP was evoked from the optic tract at the EPSP threshold than from the optic nerve (cf. Fig. 3A and *H)*. This larger IPSP implies that with optic tract stimulation more axons providing an inhibitory input to the cell were recruited at lower intensity than the excitatory fibre. A similar change in recruitment order between the optic nerve and optic tract electrodes was a common finding. The explanation is that the optic tract electrode generated a non-uniform field through the nerve. In this situation not only fibre size but also the distance between the tip of the stimulation electrode and the activated axons play a role in determining its threshold intensity.

X cells

Recordings from a characteristic X principal cell are shown in Fig. 4. The receptive field of this unit had a small centre, a strong inhibitory surround and a 'null position' in the contrast reversal test. The cell received excitation from two retinal ganglion

cells in the contralateral left eye. The unitary EPSPs are seen in the upper row of records (Fig. *4A—C)*, taken with the cell hyperpolarized to the IPSP reversal level. The first unitary EPSP was evoked at a stimulus strength of 3•5 times threshold for the nerve (Fig. *4A)*. This intensity was well above the stimulus strength required for

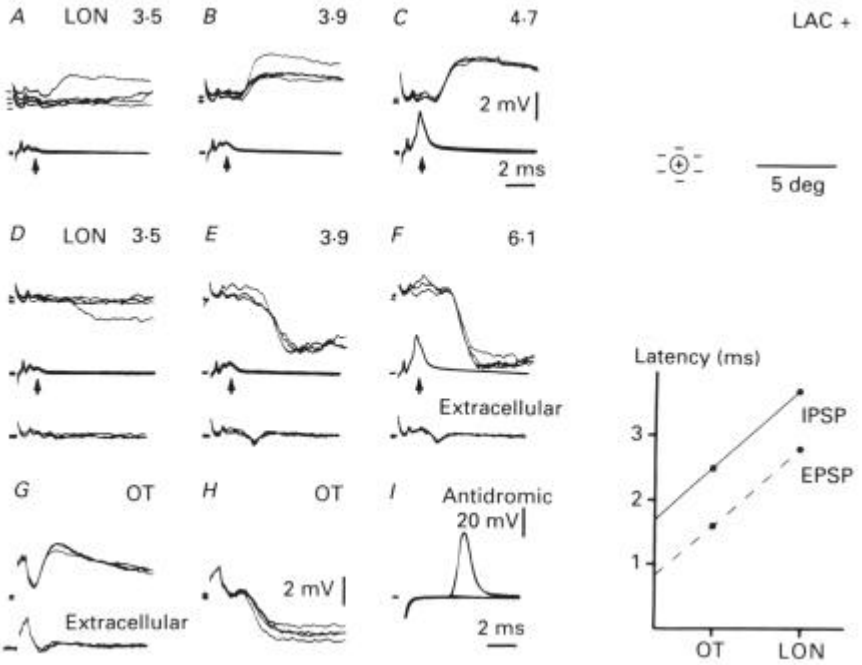

Fig. 4. Intracellular recordings of synaptic potentials in an X principal cell activated from the contralateral left eye. Details as in Fig. 3. Arrows below the nerve volley recordings point to the X component. The cell was hyperpolarized for the EPSP responses in *A—C* and *C* and depolarized to reveal the IPSPs in *D—F* and *H* (5 nA). Note the longer latency of the IPSPs compared to that of the EPSPs. The conduction velocity of the responsible optic nerve fibres was the same for both PSPs (24 m/s).

a maximal Y volley and within the lower X intensity range as indicated by a small X component in the nerve volley recording (arrow, lower trace). The other unitary EPSP was recruited at still higher intensity (Fig. *4B)*. No additional EPSPs were obtained with the X volley reaching its maximum (Fig. *4C)*.

The disynaptic feed-forward IPSP evoked in the cell by optic nerve stimulation was revealed by the injection of a strongly depolarizing current (Fig. *4D—F)*. The longer latency of the IPSP precludes that this hyperpolarizing response is a reversed EPSP. Note that both PSPs in this X cell had longer latencies than the analogous responses of the Y cell illustrated in Fig. 3. The IPSP threshold for the X cell was the same as for the first unitary EPSP, i.e. well within the X range (Fig. *4D)*. In fact, it was evident from records taken at intermediate levels of polarization that the unitary EPSP and IPSP were evoked from the very same optic nerve fibre. At threshold stimulation intensity and also during spontaneous activity they occurred together in an all or nothing fashion. Similar observations have been made for the

majority of well-analysed X cells. In the present cell the IPSP grew further with the recruitment of more X fibres (Fig. *4E* and *F),* indicating its convergent nature. At least four unitary IPSPs could be resolved by more carefully graded stimulation. From the slope of the lines in the extrapolation diagram it is seen that the described

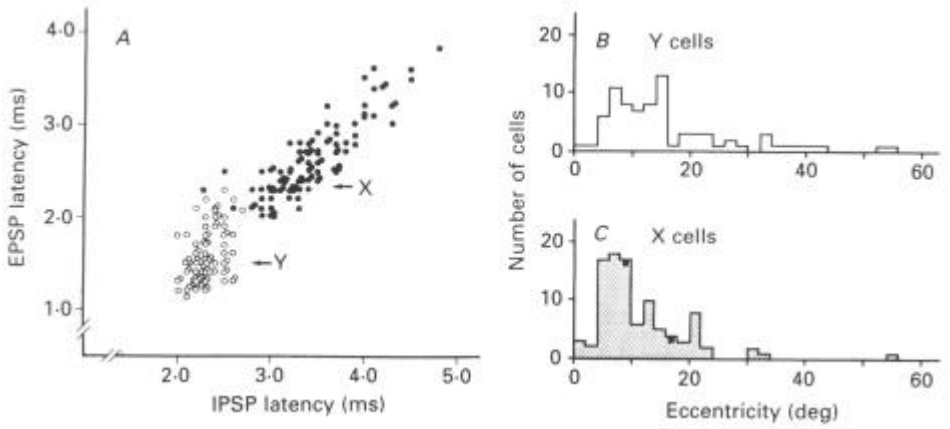

Fig. 5. *A,* latencies of monosynaptic EPSPs and disynaptic IPSPs evoked by optic nerve stimulation in visually identified X and Y principal cells. For each cell the shortest latency of the EPSP has been plotted against that of the IPSP. , X cells *(n* = 99); , Y cells *(n* = 79). Two X cells with feed-forward IPSPs from both Y and X axons are marked by filled squares. *B* and *C,* distribution of receptive field eccentricities for the sample of Y and X cells in *A*.

PSPs were evoked by slowly conducting optic nerve axons. The estimated conduction velocity was the same (24 m/s) for both responses.

Comparison of IPSPs in X and Y ce118

A similar, but not always as extensive analysis, was performed for all the recorded Y and X cells. The plot in Fig. *5A* summarizes our main findings with respect to the feed-forward inhibition. Here the latency of the EPSP evoked from the optic nerve has been plotted against that of the IPSP for each cell. The data points for the vast majority of the X and Y cells form two well-segregated subpopulations with minimal overlap in the inhibitory dimension. All X cells had excitatory latencies equal to or longer than 2~O ms and, with the exception of three cells discussed below, IPSP latencies at or above 28 ins. Additional unitary PSPs, if present, had very similar latency values (cf. Fig. 4). With the exception of the three mentioned cells, none of the X cells had inhibitory inputs that could originate from large-diameter, fast-conducting Y axons. Several of our X cells fitted the criteria of lagged X cells as defined recently by Mastronarde (1987). There was no obvious difference between the feed-forward IPSPs in these and other X cells.

All Y cells had IPSP latencies compatible with a disynaptic input from Y axons. In the large majority the IPSPs reached a maximum at intensities below threshold for the X volley. A few cells had IPSPs that grew beyond this point. They were all found in experiments with a certain amount of threshold overlap between the X and

Y nerve volleys. In these cells the IPSPs increased with stimulation intensities in the lower X range, corresponding to the zone of overlap, but not beyond the level for the maximal Y volley. We therefore assume that this inhibition also originated from Y fibres with somewhat higher thresholds. We found no Y cell with a clear inhibitory input from X fibres.

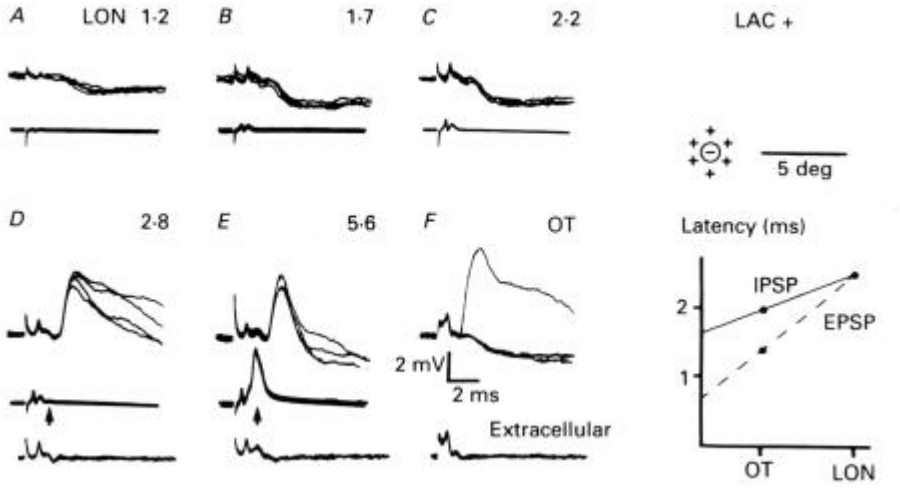

Fig. 6. Intracellular recordings from an exceptional X cell with mixed inhibitory input. The sequence of responses in *A—E* was obtained by graded stimulation of the left optic nerve. The cell was activated by a single X fibre with its threshold in the lower X range (2•8 times the nerve threshold; *D)*. Small arrows in *I)* and *F* point to the X volley. Low-threshold Y fibres evoked a small feed-forward IPSP with appropriate Y latency *(A—C)*. Additional IPSPs were evoked by X fibres as seen from the change in time course of the EPSP decay when the stimulation intensity was increased within the X range (cf. *D* and *E)*. The conduction velocities were 25 m/s for the monosynaptic EPSP and 53 m/s for the early disynaptic IPSP. Other details as in Fig. 3.

A similar plot as in Fig. *5A* was obtained when instead of latency, PSP thresholds were compared (not illustrated). For X cells there was a linear relation between the EPSP and IPSP values both in terms of threshold intensities and latencies (cf. Fig. *5A)*. The Y cell population deviated somewhat from this linear relationship. Many Y cells with EPSPs in the long-latency, high-threshold end of the sample had IPSPs with comparably short latencies and low thresholds. It is reasonable to assume that this behaviour reflects a larger convergence within the Y inhibitory pathway.

Cells with atypical convergence patterns

Among 178 visually characterized X and Y cells we found only two with mixed inhibitory input, both classified as X cells (, Figs 5A and *C* and *9C)*. Recordings from one of these cells is illustrated in Fig. 6. This off-centre cell was classified as an X cell by its small receptive field centre as compared to neighbouring Y cells. It gave a tonic discharge to the offset of a centred light spot but, unfortunately, it was impaled before a linearity test could be applied. It had a single excitatory input fibre with a threshold and latency in the lower X range (Fig. *6D)*. The sequence of

recordings in *A—E* were obtained with gradually increasing strength of stimulation of the left optic nerve. The upper row of records were taken within the Y volley range (cf. nerve volleys in lower traces) and yet there is a small disynaptic IPSP evoked at these low intensities. Both its threshold and latency point to a Y fibre origin. An

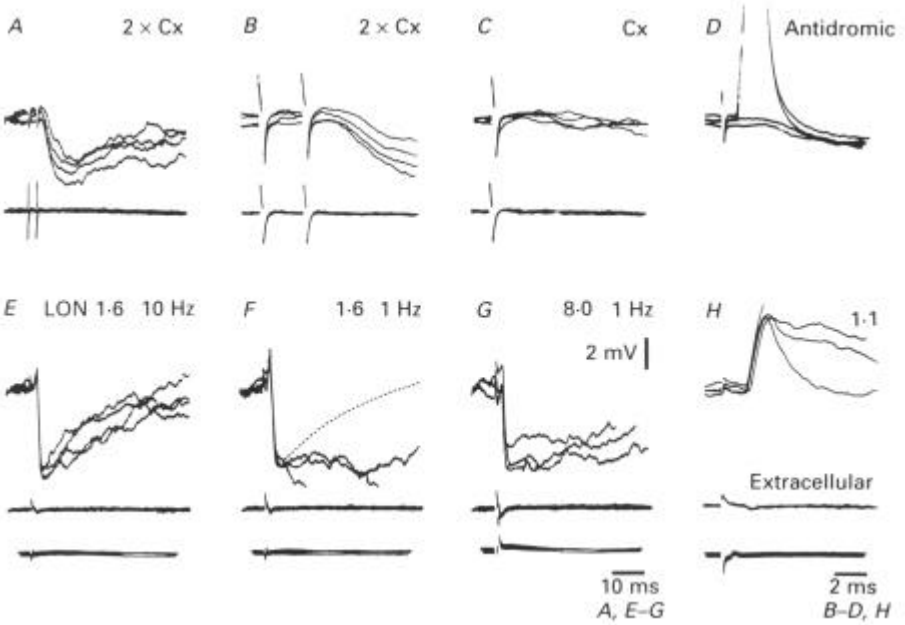

Fig. 7. Recurrent IPSPs in a Y principal cell. *A* and *B*, recurrent IPSP evoked by double stimulation of the visual cortex (Cx) and recorded with two different time bases. Temporal facilitation was used to obtain a distinct JPSP onset with minimal latency. The stimulation intensity was adjusted so that a single stimulus was subthreshold for an JPSP *(C)*. *E—G*, recurrent JPSP evoked in the orthodromic direction in the same cell by left optic nerve stimulation. The stimulation intensity was maximal for Y fibres in *E* and *F* (l~6 times threshold) and for X fibres in U (80 times threshold). At a stimulation frequency of 10 Hz only a feed-forward IPSP with a simple exponential decay was evoked (F). At lower stimulus repetition rate (1 Hz) an additional late irregular JPSP component is added to the response. This late IPSP component is a recurrent JPSP evoked by orthodromic activation of recurrent inhibitory interneurones in the perigeniculate nucleus. The recurrent LPSP was maximal with activation of Y fibres and no further IPSP was added to the response when X fibres were also stimulated (cf. *F* and U). The responses in *A—C* and *E—U* were obtained with the cell depolarized by current injection (2 nA). *D*, truncated antidromic spike at threshold; *H*, monosynaptic EPSP evoked by left optic nerve stimulation; *D* and *H* recorded with the cell unpolarized. Lower traces in *A—C* and middle traces in *F—H* are extracellular field potentials, lower traces in *F—U* optic tract nerve volleys. Other details as in Fig. 3.

additional IPSP component was evoked by X fibres as indicated by the change in the EPSP decay slope between the records in *D* and *E*.

The difference in conduction velocity of the fibres responsible for the EPSP and early IPSP is obvious from the extrapolation diagram. For the IPSP the conduction velocity was 53 m/s, i.e. within the Y range, while the corresponding value for the EPSP was typical for X fibres, 25 m/s. This exceptional X cell undoubtedly received

a mixture of inhibition from both X and Y optic nerve fibres. So did the other X cell indicated by a square. (The third deviating X cell in Fig. 5*A* had a short-latency IPSP that grew with the X volley only.) The rarity of X cells with Y inhibitory input suggests that this type of convergence might result from an error of connections in the dLGN network.

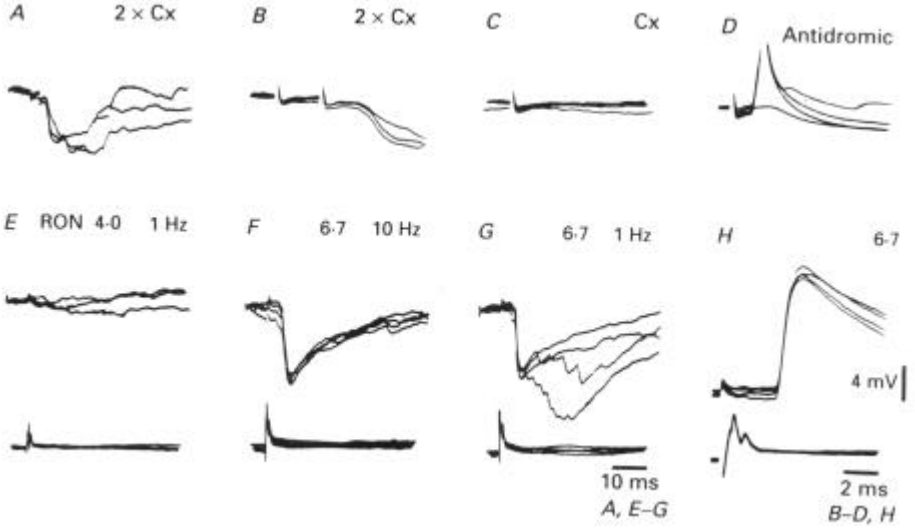

Fig. 8. Recurrent LPSPs in an X off-centre principal cell. *A* and *B,* recurrent JPSP evoked by double stimulation of the cortex and recorded with two different time bases. *C,* lack of LPSP after single stimulation with the same intensity. *D,* truncated antidromic spike evoked at threshold intensity from a neighbouring cortex electrode. *F—F,* recurrent IPSP evoked in the orthodromic direction by right optic nerve stimulation, same procedure as in Fig. 7. Note lack of IPSPs at 4~O times threshold, 1 Hz *(F)* and the late recurrent component at 6~7 times threshold, 1 Hz *(C). H,* monosynaptic EPSP evoked by X fibres in the right optic nerve. Note the long latency compared to the EPSP in Fig. 7 *H.* Lower traces in *F—H* are nerve volleys simultaneously recorded from the optic tract. Extracellular field potentials were insignificant and therefore not illustrated. Responses in *A—C* and *F—C* were obtained with the cell depolarized by current injection (6 nA). Other details as in Fig. 3.

Sixteen cells in our total sample received mixed excitation from X and Y optic nerve fibres. Thirteen of these cells had a dominant EPSP from Y axons and three from X axons. The EPSPs from the non-dominant fibre group were always quite small and most cells were visually classified in accordance with their dominant excitatory inputs. Twelve of these cells had feed-forward IPSPs that grew with both the Y and X components of the nerve volley; the other four seemed to receive IPSPs from the dominant fibre group only.

Recurrent inhibition

The recurrent inhibitory system was studied in a smaller sample of cells. The IPSPs were evoked by antidromic activation of principal cell axons from the visual cortex. A stimulation electrode could usually be selected that produced a good recurrent IPSP in the recorded cell without contamination of an antidromic spike

(Lindström, 1982). Double stimulation was used to obtain temporal facilitation at the level of the recurrent inhibitory interneurones in the perigeniculate nucleus (Ahlsén *et al.* 1983). This procedure gives recurrent IPSPs with more distinct onset and minimal latency. Recurrent IPSPs were evoked in all of the cells tested.

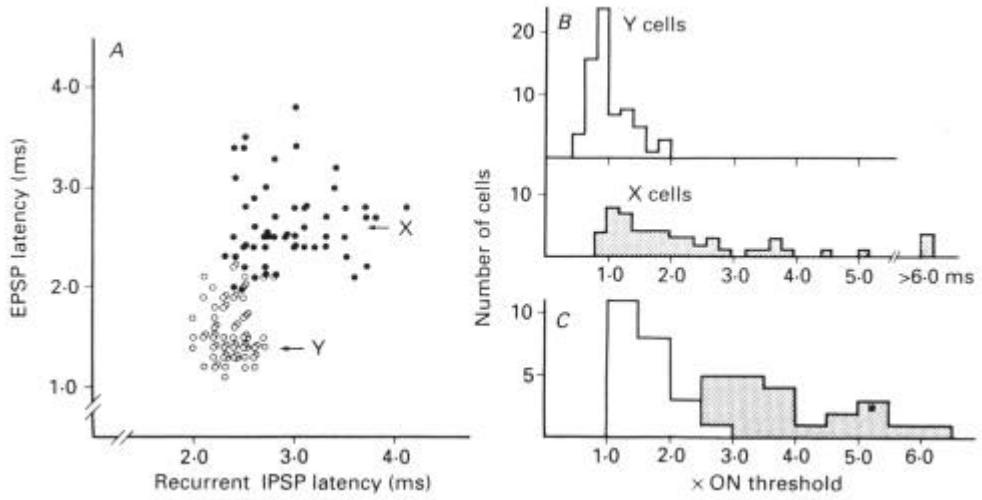

Fig. 9. *A,* scatter distribution of recurrent IPSP latencies in visually classified X and Y principal cells. For each cell the latency of the EPSP evoked by optic nerve stimulation has been plotted against the latency of the recurrent IPSP evoked by cortex stimulation. The latency of the recurrent IPSP was measured from the effective stimulus (cf. Fig. *7B* and *C).* are X cells *(n* = 55); , Y cells *(n* = 68). *B,* latency distribution for antidromic spikes evoked by cortex stimulation in the same sample of Y and X cells. *C,* distribution of thresholds for orthodromically activated recurrent IPSPs in X and Y principal cells. The IPSPs were evoked by optic nerve stimulation as in Fig. *7E—G,* and the thresholds measured in multiples of nerve threshold as in Fig. 1. Open histograms, Y cells *(n* = 23); shaded histograms, X cells *(n* = 21), as in *B.* , an X cell with mixed feed-forward IPSPs from X and Y fibres. Further details in the text.

Examples of such IPSPs in a Y and an X principal cell are shown in Figs 7 and 8. The records in *A* and *B* are the same IPSPs displayed with two different time bases. The stimulation intensity was adjusted so that a single stimulus was subthreshold for a response *(C).* It follows that the IPSP was triggered by the second stimulus (producing a summed EPSP above spike threshold in the interneurones). The IPSP latency was accordingly measured from the second stimulus shock artifact and it was 2•4 ms for the Y cell in Fig. 7 and 2•5 ms for the X cell in Fig. 8.

In Fig. 9A the latency of the recurrent IPSP in all studied X and Y cells is plotted against their EPSP latency from the optic nerve. The latter parameter was used to allow a comparison with previous plots. The shortest recurrent latency (20 ms) was found among the Y cells and they had, as a population, significantly shorter latency values than the X cells. However, there was a sizeable range of overlap between the two groups (from 2•3 to 2•8 ms). This overlap in latency reflects a similar overlap in antidromic conduction times from the cortex to the same cells (Fig. 9B; cf. also Stone & Hoffman, 1971; Stone & Dreher, 1973; So & Shapley, 1979). Although it lowers the

analytical sensitivity of the procedure this overlap does not exclude a type-selective recurrent inhibition. Note that more than half of the X cell population had longer recurrent latencies than any Y cell. These cells at least seemed to receive a selective recurrent input from the X system.

To improve the certainty of our conclusion we also evoked recurrent IPSPs in the orthodromic direction by stimulation of the optic nerves (Lindström, 1982). In this way we could take advantage of the threshold separation between X and Y fibres. The procedure is illustrated by the recordings of Fig. *7E—G*. The record in *E* shows a maximal feed-forward IPSP with slower time base than in previous figures. At 10 Hz of stimulation the feed-forward IPSP had a smooth exponential decay. When the stimulus repetition rate was lowered to 1 Hz (Fig. *7F)* a late additional IPSP component appeared. This IPSP component results from orthodromic activation of perigeniculate neurones. In this Y cell the orthodromically elicited recurrent JJ)5J) developed with the Y component of the nerve volley (Fig. *7F)*. It did not increase further in amplitude when the stimulus strength was increased to recruit the X fibres also (Fig. 7G).

A similar analysis for the X cell in Fig. 8 revealed that the orthodromically elicited recurrent IPSP appeared only at high X stimulus intensities. Stimulation of the right optic nerve at 4•0 times threshold, well above Y maximum but below threshold for the excitation of the cell, evoked no inhibitory response (Fig. *SE)*. A recurrent IPSI~ was evoked in the orthodromic direction, however, when the intensity was increased to recruit the excitatory and feed-forward inhibitory input to the cell (Fig. 8F and G).

The same kind of measurements were obtained for forty-four visually characterized principal cells (twenty-three Y cells and twenty-one X cells) and the result is presented in Fig. *9C*. All Y cells had orthodromic thresholds for the recurrent IPSP in the Y volley range and the X cells in the X range. For the majority of the Y cells we could be certain that the IPSP grew exclusively with the Y volley (as for the cell in Fig. *7)*, indicating that the recurrent input was Y selective. The lack of low-threshold IPSPs in the X cells precludes that these cells received a Y-type recurrent inhibition. Interestingly, one of the exceptional X cells with mixed X-Y feed-forward inhibition was tested and found to receive an exclusive X-type recurrent inhibition. Although a rather small number of cells were studied with this procedure it seems safe to conclude that there is no significant Y-type recurrent input to X principal cells or the reverse. Thus, the recurrent inhibitory pathway seem to be as type selective as the feed-forward inhibitory pathway.

Binocular inhibition

Many principal cells also receive inhibition from retinal ganglion cells in the nonexcitatory eye (Suzuki & Kato, 1966). Such non-dominant inhibition is mediated both by the feed-forward and the recurrent pathway (Lindström, 1982). We only occasionally studied this non-dominant inhibitory input in our sample of cells. Among thirty-one tested Y cells twenty-six had disynaptic feed-forward IPSPs from the non-dominant nerve. All these IPSPs developed with the Y component of the nerve volley. Only two of five tested X cells had a clear disynaptic IPSP from the non-dominant eye and this IPSP appeared together with the X volley. Since most

perigeniculate interneurones receive binocular excitation, the recurrent inhibitory pathway is typically binocular. This was confirmed for fifteen Y and four X principal cells with recurrent inhibition activated in the orthodromic direction (see above). Also the recurrent IPSPs from the non-excitatory optic nerve were evoked by the same fibre type as the EPSPs in the cells.

Receptive field position

Singer & Bedworth (1973) suggested that the degree of inhibitory interaction between the X and Y pathway differed between principal cells with central and more peripheral receptive fields. To be more specific, peripheral X cells were believed to receive stronger Y inhibition than those with central receptive fields. We found no such difference. The lack of Y inhibition of X cells in our material cannot be explained by a heavy bias in the sample towards central units. The distribution of receptive field eccentricities for all our Y and X cells is shown by the histograms in Fig. *5B* and *C*. More than 40 % of our X cells had receptive fields at eccentricities larger than 10 deg, the maximal value being 55 deg. The only two X cells with mixed inhibition from the X and Y systems (see above) had their receptive fields at intermediate eccentricities, 8 and 17 deg **(** in Fig. *SC)* There was likewise no difference in inhibitory input to Y cells with central or peripheral receptive fields.

DISCUSSION

Two main conclusions emerge from our results: (1) both X and Y principal cells in the cat's dLGN receive feed-forward and recurrent inhibition; (2) the inhibitory circuits are similar but functionally independent for the two cell types. Both conclusions are at odds with some views in the literature and require consideration.

Similar inhibitory circuits for X and Y *cells*

The first intracellular studies of the dLGN revealed prominent IPSPs in principal cells after optic tract stimulation (Suzuki & Kato, 1966; Mcllwain & Creutzfeldt, 1967; Singer & Creutzfeldt, 1970). The IPSPs occur in X and Y cells (Singer & Bedworth, 1973) and are, for both cell types, mediated by feed-forward and recurrent inhibitory pathways (Lindström, 1982). This view is supported by recordings from the inhibitory neurones of these pathways (intrageniculate interneurones and perigeniculate cells), both of which are found with selective excitation from either the X or Y system (Dubin & Cleland, 1977; Lindström, 1983; Ahlsén *et al*. 1983; Wrdbel & Tarnecki, 1984; Xue, Carney, Ramoa & Freeman, 1988).

The symmetrical organization of the inhibitory pathways to X and Y principal cells has recently been questioned (Sherman & Friedlander, 1988). These authors propose that the feed-forward pathway might be exclusive for X cells and the recurrent for Y cells. The idea has apparently evolved from their failure to identify Y- type intrageniculate interneurones by intracellular injection of horseradish peroxidase. On the basis of this strictly negative finding they disclaim the existence of Y-type intrageniculate interneurones. Previous physiological identification of such cells is dismissed as based on a single negative criterion, lack of antidromic activation from the visual cortex. This discussion fails to acknowledge that

intrageniculate interneurones also differ from principal cells in aspects that provide positive identification criteria: duration of action potentials (Lindström, 1983; McCormick & Pape, 1988), synaptic response to cortical (Dubin & Cleland, 1977; Lindström, 1983) or brain stem stimulation (Ahlsén, Lindström & Lo, 1984).

Independent anatomical support for two types of intrageniculate interneurones have been obtained with GAD and GABA immunohistochemistry (Montero & Zempel, 1985). Presumed Y-type intrageniculate interneurones are few in number, about 3—6 % of the neuronal population in the A laminae of the cILGN. That such a small group of cells is missed in a limited sample of intracellularly stained interneurones (Sherman & Friedlander, 1988) may not be entirely surprising. Anyhow, the present findings should leave no doubt that Y principal cells receive disynaptic inhibition of the feed-forward type just like X cells.

The proposition that X cells lack recurrent inhibition is puzzling since X principal cells are known to issue axon collaterals in the perigeniculate nucleus (Ahlsén, Lindström & Sybirska, 1978; Friedlander, Lin, Standford & Sherman, 1981), to excite perigeniculate cells by such collaterals and to receive recurrent IPSPs (references above). The present finding of recurrent IPSPs in all studied X cells reiterates these observations. It is worth noting that there were no obvious quantitative differences between the IPSPs in X and Y principal cells. Recurrent and feed-forward IPSPs were as large and easy to reveal in X cells as in Y cells. This observation does not preclude the possibility that the two inhibitory circuits differ in importance for X and Y cells during adequate visual activation but any such differences remain to be shown (Sillito & Kemp, 1983).

Lack of inhibitory interaction between X and Y systems

All but two principal cells in this study received feed-forward and recurrent inhibition exclusively from the same afferent system as their excitatory input. Thus, we were unable to confirm previous suggestions that the X and Y pathways have reciprocal or partially interactive inhibition in the dLGN (Hoffman *et al*. 1972; Singer & Bedworth, 1973; Burke *et at*. 1985; Bloomfield & Sherman, 1988).

Our analysis rests on the established method of fibre threshold separation with graded electrical stimulation. The procedure has been extensively used in studies of specific neuronal connections in the spinal cord (Baldissera, Hultborn & Illert, 1981) and its usefulness is also well documented for the retino-geniculate pathway (Bishop & Leary, 1940; Bishop & McLeod, 1954; Bishop, Clare & Landau, 1969). Our control recordings fully confirmed the conclusion of Hoffman *et at*. (1971) that the first low-threshold component of the optic nerve volley originates from Y axons and the later component from X axons. Using a ring electrode for stimulation an almost perfect threshold separation between the two fibre groups was obtained. Identical results with the same technique have been presented by Ferster (1990). In our view the threshold separation technique is as powerful as any visual procedure for X and Y segregation. Combined with intracellular recordings it offers a unique opportunity to identify all the excitatory and inhibitory inputs from retinal ganglion cells to a single principal cell.

The disynaptic feed-forward IPSPs in most X cells originated from ganglion cell axons with thresholds and conduction velocities in the X range only. The lack of

significant Y contribution was not due to poor resolution in the recordings. JPSPs as small as 5 % of the maximal response were reliably detected; in some cells even smaller responses could be seen. Neither can our failure to reveal Y-type IPSPs in X cells be explained by hypothetical shunting synapses or synapses at remote dendritic locations. Such synaptic effects would have been disclosed by the routine polarization of the cells since the dendritic trees of dLGN principal cells are quite compact (Crunelli, Leresche & Parnavelas, 1987; Bloomfield, Hamos & Sherman, 1987). EPSPs from cortico-geniculate fibres, known to terminate at distant dendrites, were also readily observed (S. Lindström & A. Wróbel, unpublished observations). Neither were Y-type IPSPs concealed by an excitatory input since the Y axons could be stimulated selectively without contamination from X axons.

Other possibilities such as conduction failure at the interneuronal level in the Y inhibitory pathway due to a depressive effect of the anaesthesia or inhibition from the brain stem can also be excluded. Such schemata would require two classes of Yactivated intrageniculate interneurones with separate projection to X and Y cells since Y-type IPSPs were readily evoked in neighbouring Y principal cells of the same preparation. The feed-forward IPSPs are also quite robust to changes in depth or type of anaesthesia (S. Lindström & A. Wrdbel, unpublished observations). All of these considerations lead us to believe that even small Y-type inhibitory inputs to the X cells would have been revealed should they exist. The finding of mixed IPSPs in two exceptional X cells supports this view.

Comparable results were obtained for Y cells, most of which had disynaptic IPSPs growing below threshold for the X volley. Some Y cells had additional unitary I PSPs appearing within the threshold overlap zone, where the first X axons were recruited. These IPSPs had Y-type latencies and they were never evoked at intensities above that of the maximal Y volley. Therefore, we assume that all unitaries forming the compound IPSP in Y cells were mediated by Y fibres.

The lack of detectable X contribution to these IPSPs was not due to occlusion at the interneuronal level since X and Y fibres activate separate intrageniculate interneurones. Neither is it due to saturation of IPSPs approaching their reversal level. Most IPSPs were recorded with the membrane potential artificially shifted away from this potential and larger IPSPs were easily obtained with temporal summation. Note also that convergence of Y- and X-type feed-forward IPSPs were observed in several cells with mixed excitatory inputs from X and Y fibres. The conclusion that Y cells lack feed-forward inhibition from X axons seems inevitable.

The selectivity of the recurrent pathway was more difficult to determine. As expected from perigeniculate cell recordings (Ahlsén *et al*. 1983), the shortest IPSP latencies were found in Y principal cells and the longest in X cells. Many X cells had latency values longer than any of those in Y cells suggesting a recurrent input exclusively from the X system. Unfortunately, about half the cells had latencies within a sizeable range of overlap between the two populations. Since the threshold separation procedure is useless with cortex stimulation, recurrent IPSPs were instead evoked in the orthodromic direction by optic nerve stimulation. All units checked in this way had fibre type specific inhibition via the recurrent loop. Taken together these findings convincingly demonstrate that the recurrent inhibitory pathways to principal cells are as type specific as the feed-forward pathways.

Given the consistency of our observations it may be asked why other investigators have reached different conclusions. One explanation might be their use of optic chiasm stimulation (Hoffman *et al*. 1972; Singer & Bedworth, 1973; Bloomfield & Sherman, 1988). This mode of stimulation results in a substantial overlap between the X and Y fibre populations both in latency and threshold (So & Shapley, 1981; see also Results). From the published records of Singer & Bedworth (1973) it can be seen that they also underestimated the IPSP latency in X cells. The latency was measured to the deviation point of PSPs obtained at two membrane polarization levels, this point being set mainly by EPSP depression (see their Fig. *4D* and *E)*. IPSPs evoked in X cells by stimulation of the superior colliculus were taken as further evidence for Y-type inhibition of dLGN X cells (Singer & Bedworth, 1973). At the time only Y axons were believed to bifurcate to both structures. This argument has been waived by a later demonstration of retinal X cells with bifurcating axons (Illing & Wässle, 1981).

The experiments of Burke *et al*. (1985) are different. They blocked impulse conduction in Y axons by applying pressure to the optic nerve. Stimulation of remaining fibres induced two phases of reduced responsiveness in both X and Y geniculate neurones as judged by field potential recordings. The early phase was ascribed to reduced excitability in the optic nerve fibres while the later phase had a much longer latency (more than 10 ins) and duration (several hundred milliseconds) than any IPSP we have observed in principal cells. Whatever the source of these subnormalities it seems unlikely that they were generated by the local inhibitory circuits of the dLGN.

Functional and developmental implications

It is generally believed that the X and Y pathways carry different types of information to higher brain centres, although the exact nature of this information is debated (Stone *et al*. 1979; Sherman, 1985). If two separate messages are required at the cortical level, it would seem strange to distort them by inhibitory interaction in the dLGN. Why else would the pathways be segregated again in the cortex? Separate inhibitory circuits for the X and Y principal cells, as found here, ascertain that the information reaches the cortex undisturbed. Private inhibitory systems also allow for separate control of the pathways by cortico-geniculate or brain stein neurones (Ahlsén, 1984). Such a selective control might be used to change the balance between the X and Y systems so that the brain obtains optimal information for each behavioural task.

Anatomically, X and Y principal cells, intrageniculate interneurones, their dendrites and axons, terminal branches of perigeniculate neurones and retinal ganglion cells all intermingle in the dLGN neuropile. Yet with this anatomical disorder all synaptic connections seem to be functionally specific. Not only the excitatory input to principal cells but also their inhibitory circuits are well segregated. This precise wiring is undoubtedly of fundamental importance for the function of the structure. To understand how this impressive precision in neuronal connections comes about is a real challenge for future studies of neuronal development.

The present study was supported by the Swedish Medical Research Council (Project No. 04767) and by Stiftelsen Sigurd och Elsa Goljes Minne. A. W. was supported by a Swedish MRC Visiting Scientist Fellowship (No. 7110).

REFERENCES

AHLSEN, G. (1984). Brain stem neurones with differential projection to functional subregions of the dorsal lateral geniculate complex in the cat. *Neuroscience* 12, 817—838.

AIILSEN, G., LINDSTRöM, S. & Lo, F.-S. (1983). Excitation of perigeniculate neurones from X and Y principal cells in the lateral geniculate nucleus of the cat. *Acta physiologica scandinavica* 118, 445—448.

AHLSEN, G., LINDSTRöM, S. & Lo, F.-S. (1984). Inhibition from the brain stem of inhibitory interneurones of the cat's dorsal lateral geniculate nucleus. *Journal of Physiology* 347, 593—609.

AHLSEN, *G.*, LINDSTRöM, S. & Lo, F.-S. (1985). Interaction between inhibitory pathways to principal cells in the lateral geniculate nucleus of the cat. *Experimental Brain Research* 58, 134—143.

AHLSEN, G., LINDSTRöM, S. & SYBTRSKA, E. (1978). Subcortical axon collaterals of principal cells in the lateral geniculate body of the cat. *Brain Research* 156, 106—109.

BALDISSERA, F., HULTBORN, H. & ILLERT, M. (1981). Integration in spinal neuronal systems. In *Handbook of Physiology,* section I, *The Nervous System,* vol. II, *Motor Control,* ed. BROOKS, V. B., pp. 509—595. American Physiological Society, Bethesda, MD, USA.

BIsHoP, G. H., CLARE, M. H. & LANDAU, W. M. (1969). Further analysis of fiber groups in the optic tract of the cat. *Experimental Neurology* 24, 386-399.

BISHOP, P. 0. & MCLEOD, J. G. (1954). Nature of potentials associated with synaptic transmission in lateral geniculate of cat. *Journal of Neurophysiology* 17, 387—414.

BISHOP, *G.* H. & O'LEARY, J. L. (1940). Electrical activity of the lateral geniculate of cats following optic nerve stimuli. *Journal of Neurophysiology* 3, 308—322.

BLOOMFIELD, S. A., HAMOS, J. E. & SHERMAN, S. M. (1987). Passive cable properties and morphological correlates of neurones in the lateral geniculate nucleus of the cat. *Journal of Physiology* 383, 653—692.

BLOOMFIELD, S. A. & SHERMAN, S. M. (1988). Postsynaptic potentials recorded in neurons of the cat's lateral geniculate nucleus following electrical stimulation of the optic chiasm. *Journal of Neurophysiology* 60, 1924—1945.

BULLIER, J. & HENRY, *G.* H. (1979). Neural path taken by afferent streams in striate cortex of the cat. *Journal of Neurophysiology* 42, 1264—1270.

BURKE, W., BURNE, J. A. & MARTIN, P. R. (1985). Selective block of Y optic nerve fibres in the cat and the occurrence of inhibition in the lateral geniculate nucleus. *Journal of Physiology* 364, 81—92.

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

CLELAND, B. *G.*, HARDING, T. H. & TULUNAY-KEESEY, U. (1979). Visual resolution and receptive field size: examination of two kinds of cat retinal ganglion cell. *Science* 205, 1015—1017.

CRUNELLI, V., LERESCHE, N. & PARNAVELAS, J. G. (1987). Membrane properties of morphologically identified *X* and Y cells in the lateral geniculate nucleus of the cat *in vitro*. *Journal of Physiology* 390, 243—256.

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.

ENROTH-CUGEL, C. & ROBSON, J. G. (1966). The contrast sensitivity of retinal ganglion cells of the cat. *Journal of Physiology* 187, 5 17—552.

FERSTER, D. (1990). *X*- and Y-mediated synaptic potentials in neurons of areas 17 and 18 of cat visual cortex. *Visual Neuroscience* 4, 115—133.

FERSTER, D. & LINDSTRöM, 5. (1983). An intracellular analysis of geniculo-cortical connectivity in area 17 of the cat. *Journal of Physiology* 342, 181—2 15.

FRIEDLANDER, M. J., LIN, C.-S., STANFORD, L. R. & SHERMAN, S. M. (1981). Morphology of functionally identified neurons in the dorsal lateral geniculate nucleus of cat. *Journal of Neurophysiology* 46, 80—129.

HOFFMANN, K. P., STONE, J. & SHERMAN, S. M. (1972). Relay of receptive field properties in dorsal lateral geniculate of the cat. *Journal of Neurophysiology* 35, 518—531.

fLUNG, R. B. & WXSSLE, H. (1981). The retinal projection to the thalamus in the cat: a quantitative investigation and a comparison with the retinotectal pathway. *Journal of Comparative Neurology* 202, 265—285.

LINDSTRöM, S. (1982). Synaptic organization of inhibitory pathways to principal cells in the lateral geniculate nucleus of the cat. *Brain Research* 234, 447—453.

LINDSTRöM, 5. (1983). Interneurones in the lateral geniculate nucleus with monosynaptic excitation from retinal ganglion cells. *Acta physiologica scandinavica* 119, 101—103.

LINDSTRöM, S. & WRóBEL, A. (1984). Separate inhibitory pathways to X and Y principal cells in the lateral geniculate nucleus of the cat. *Neuroscience Letters Supplement* 18, 8234.

MCCORMICK, D. A. & PAVE, H-C. (1988). Acetylcholine inhibits identified interneurons in the cat lateral geniculate nucleus. *Nature* 334, 246—248.

MCILWAIN, *J.* T. & CREUTZFELDT, 0. D. (1967). Microelectrode study of synaptic excitation and inhibition in the lateral geniculate nucleus of the cat. *Journal of Neurophysiology* 30, 1—2 1.

MASTRONARDE, D. M. (1987). Two classes of single-input X-cells in cat lateral geniculate nucleus. I. Receptive-field properties and classification of cells. *Journal of Neurophysiology* 57. 357—380.

MONTERO, V. M. & ZEMPEL, J. (1985). Evidence for two types of (}ABA-containing interneurons in the A-laminae of the cat lateral geniculate nucleus: a double-label 1-IRP and CABAimmunocytochemical study. *Experimental Brain Research* 60, 603—609.

SANDERSON, K. J. (1971). The projection of the visual field to the lateral geniculate and medial interlaminar nuclei in the cat. *Journal of Comparative Neurology* 143, 101—118.

ShERMAN, S. M. (1985). Functional organization of the W-, X- and Y-cell pathways in the cat: a review and hypothesis. In *Progress in Psychobiology and Physiological Psychology,* vol. 11. pp. 233—314. Academic Press, New York.

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

SILuT0, A. M. & KEMP, J. A. (1983). The influence of GABAergic inhibitory processes on the receptive field structure of X and Y cells in cat dorsal lateral geniculate nucleus (dLGN). *Brain Research* 277, 63—77.

SINGER, W. & BEDWORTH, N. (1973). Inhibitory interaction between X and Y units in the cat lateral geniculate nucleus. *Brain Research* 49, 29 1—307.

SINGER, W. & CREUTZFELDT, 0. D. (1970). Reciprocal lateral inhibition of on- and off-center neurons in the lateral getiiculate body of the cat. *Experimental Brain Research* 10, 311—330.

So, Y. T. & SHAPLEY, R. M. (1979). Spatial properties of X and Y cells in the lateral geniculate nucleus of the cat and conduction velocities of their inputs. *Experimental Brain Research* 36, 533—550.

STONE, J. & DREHER, B. (1973). Projection of X- and Y-cells of the cat's lateral geniculate nucleus to areas 17 and 18 of visual cortex. *Journal of Neurophysiology* 36, 55 1—567.

STONE, *J.*, DREHER, B. & LEVENTHAL, A. (1979). Hierarchical and parallel mechanisms in the organization of visual cortex. *Brain Research Review* 1, 345—394.

STONE, J. & HOFFMANN, K. P. (1971). Conduction velocity as a parameter in the organization of the afferent relay in the cat's lateral geniculate nucleus. *Brain Research* 32, 454—459.

SUZUKI, H. & KATO, E. (1966). Binocular interaction at cat's lateral geniculate body. *Journal of Neurophysiology* 29, 909—920.

TROY, *J.* B. (1983). Spatial contrast sensitivities of X and Y type neurones in the cat's dorsal lateral geniculate nucleus. *Journal of Physiology* 344, 399—417.

WANG, C., CLELAND, B. G. & BURKE, W. (1985). Synaptic delay in the lateral geniculate nucleus of the cat. *Brain Research* 343, 236—245.

WRdBEL, A. & TARNECKI, R. (1984). Receptive fields of cat's non-relay lateral geniculate and perigeniculate neurons. *Acta neurobiologica experimentalis* 44, 289—299.

XUE, J. T., CARNEY, T., ROMOA, A. S. & FREEMAN, R. D. (1988). Binocular interaction in the perigeniculate nucleus of the cat. *Experimental Brain Research* 69, 497—508.