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Postsynaptic Potentials Recorded in Neurons of the Cat's Lateral Geniculate Nucleus Following Electrical Stimulation of the Optic Chiasm

STEWART A. BLOOMFIELD AND S. MURRAY SHERMAN

Department of Neurobiology and Behavior, State University of New York, Stony Brook, New York 11794-5230

SUMMARY AND CONCLUSIONS

1. We recorded intracellularly from X and Y cells of the cat's lateral geniculate nucleus and measured the postsynaptic potentials (PSPs) evoked from electrical stimulation of the optic chiasm. We used an in vivo preparation and computer averaged the PSPs to enhance their signal-to-noise ratio.

2. The vast majority (46 of 50) of our sample of X and Y cells responded to stimulation of the optic chiasm with an excitatory postsynaptic potential (EPSP) followed by an inhibitory postsynaptic potential (IPSP); these were tentatively identified as relay cells. We quantified several parameters of these PSPs, including amplitude, latency, time to peak (i.e., rise time), and duration.

3. Among the relay cells, the latencies of both the EPSP and action potential evoked by optic chiasm stimulation were shorter in Y cells than in X cells. Furthermore, the difference between the latencies of the EPSP and action potential was shorter for Y cells than for X cells. This means that the EPSPs generated in Y cells reached threshold for generation of action potentials faster than did those in X cells. The EPSPs of Y cells also displayed larger amplitudes and faster rise times than did those in X cells, but neither of these distinctions was sufficient to explain the shorter latency difference between the EPSP and action potential for Y cells.

4. The EPSPs recorded in relay Y cells had longer durations than did those in relay X cells. Our data suggest that the subsequent IPSP actively terminates the EPSP, which, in turn, suggests that the time interval between EPSP and IPSP onsets is longer in Y cells than in X cells. Furthermore, we found that, for individual Y cells, the latency and duration of the evoked EPSP were inversely related. These observations lead to the conclusion that the latency of IPSPs activated from the optic chiasm is relatively constant among Y cells and thus independent of the EPSP latencies. Thus the excitation and inhibition produced in individual geniculate Y cells may originate from different populations of retinogeniculate axons.

5. The IPSPs recorded in geniculate relay cells following optic chiasm stimulation could be divided into three groups based on their durations. The majority of both X and Y cells showed short-duration IPSPs, whereas the remainder of Y cells displayed medium-duration IPSPs, and the remaining X cells displayed long-duration IPSPs. A positive correlation was seen between the time to peak and duration of these IPSPs.

6. The reversal potential of short duration IPSPs, for both X and Y cells, was about −76 mV. In contrast, the reversal potentials of both medium- and long-duration IPSPs were about −102 mV. These data suggest that, in response to optic chiasm stimulation, both X and Y cells display two types of inhibition that differ in their ionic conductances. We found no evidence that any of the relay cells produced a mixed IPSP. Given prior evidence that γ-aminobutyric acid (GABA) is the dominant inhibitory neurotransmitter in the lateral geniculate nucleus, we propose that short-duration IPSPs reflect a chloride conductance that may be mediated by a $GABA_A$ receptor, whereas medium- and

long-duration IPSPs may each reflect a potassium conductance mediated by $GABA_B$ receptors.

7. We found that the standard deviations for 8 of 10 PSP parameters were greater for relay X cells than for relay Y cells. This finding extends to PSPs a previous assertion, based on other morphological and physiological data, that the X cell population is more heterogeneous than is the Y cell population.

8. We recorded from four neurons, all X cells, that did not display any obvious IPSP following stimulation of the optic chiasm. These cells could not be driven antidromically with electrical stimulation of the visual cortex. They have thus been tentatively identified as interneurons. In addition, the evoked EPSPs in these cells were roughly 10 times longer in duration than those generated in the relay cells. However, the other tested response properties of these neurons, which included an evaluation of their receptive field properties, were indistinguishable from those of relay X cells.

INTRODUCTION

Neurons in laminae A and A1 of the lateral geniculate nucleus have been a popular and fruitful subject of electrophysiological investigation, largely because of their strategic location for the transmission of visual information from retina to cortex. Studies using extracellular recording techniques have, for the most part, focused their attention on the parallel X and Y pathways. These studies have shown that the receptive field properties of geniculate X and Y cells are virtually identical to those of their afferent retinal inputs (10, 29, 34, 65). As a result, the lateral geniculate nucleus has often been regarded as a simple relay of visual information from retina to cortex, without significant elaboration of receptive field properties.

However, it has been demonstrated that retinal synapses represent only a small fraction of the inputs to geniculate relay cells (23, 24, 66). Activation of nonretinal inputs, such as those from the parabrachial region of the brain stem, can alter the activity of these neurons (2, 21, 60, 61). Thus it is now clear that the retinal synaptic inputs to geniculate relay cells are integrated with many nonretinal inputs, and it has been suggested that the lateral geniculate nucleus serves to gate the transmission of visual information from retina to cortex (11, 56, 62).

One approach to understand better how retinal and nonretinal signals are integrated within geniculate neurons is to use intracellular recording techniques to assess postsynaptic potentials (PSPs) in these cells. There exist a number of such studies of these neurons (1–3, 16, 35, 39, 43, 47, 60, 61, 63), and these have greatly advanced our understanding of the circuitry subserving excitation and inhibition of these cells. However, due to the technical difficulties in obtaining satisfactory intracellular recordings, there have been few investigations in which PSPs have been studied systematically, allowing for quantification of the various parameters of these synaptic potentials, such as their duration or amplitude. Knowledge of these parameters is essential for elucidation of the cellular mechanisms underlying the generation and integration of PSPs. Furthermore, the few quantitative studies have failed to identify cells as X or Y (e.g., Ref. 16), precluding a comparison of the synaptic events between these neuronal classes. Recent morphological evidence suggests that X and Y cells differ both in the distribution of retinal input and the relationship of this input to nonretinal synapses (25, 40, 66). Such differences should be reflected in the PSPs.

For the above reasons, we thought it timely to undertake a systematic study of the excitatory and inhibitory postsynaptic potentials (EPSPs and IPSPs) elicited from electrical stimulation of the optic chiasm in geniculate neurons identified as X or Y cells. We used computer averaging to quantify several parameters of both IPSPs and EPSPs, including latency, time to peak, amplitude, and duration. Analysis of these parameters has unveiled differences between X and Y cells in terms of the circuitry and cellular mechanisms underlying the activation and integration of postsynaptic potentials.

Portions of this study have been published previously in abstract form (4).

METHODS

General preparation

Many of the general methods used during these experiments have been described previously (5,

22), and a brief summary follows. Adult cats (2.0–3.5 kg) were anesthetized with 4% halothane delivered in a 50/50 mixture of nitrous oxide and oxygen. We cannulated the femoral vein for later infusion of paralytic agents and barbiturate, and we performed a tracheotomy to insert an endotracheal tube. After the animal was placed in a stereotaxic device, the level of anesthesia was changed to 1% halothane in a 70/30 mixture of nitrous oxide and oxygen; the cat was then paralyzed with 5 mg of gallamine triethiodide followed by an infusion of 3.6 mg/h of gallamine triethiodide, 0.7 mg/h of *d*-tubocurare, and 6 ml/h of 5% lactated Ringer solution. We artificially respired the cat and maintained its end-tidal carbon dioxide level at 4%. Small craniotomies and durotomies were made to introduce the stimulating and recording electrodes into the brain. Following surgery, all wounds and pressure points were infiltrated with 1% lidocaine. Halothane was then discontinued, and the animal was maintained for the remaining 24–36 h of the experiment on a 70/30 mixture of nitrous oxide and oxygen plus pentobarbital sodium infused at a rate of 1 $mg \cdot kg^{-1} \cdot h^{-1}$. We monitored heart rate and EEG throughout the course of the experiment to ensure adequate anesthesia; supplemental doses of pentobarbital sodium were administered as needed. Body temperature was maintained at 37°C with a thermostatically controlled heating blanket.

Visual stimulation

Atropine sulphate and phenylephrine were applied topically to the cat's eyes to dilate the pupils and retract the nictitating membranes. We fitted the corneas with contact lenses chosen by retinoscopy to ensure that each retina was focused on the visual targets. These targets were either located on a frontal tangent screen or generated on a cathode-ray tube. We projected the optic disk of each eye by the method of Fernald and Chase (19). This enabled us to locate each neuronal receptive field with respect to the position of the appropriate optic disk and, in turn, to that of the area centralis (52).

Responses to spatial sine-wave gratings, sinusoidally modulated in time, were used to determine the temporal and spatial summation properties of cells. We generated the gratings on a cathode-ray tube and could continuously vary spatial frequency, temporal frequency, and contrast; we used the maximum contrast, which was 0.6, for most tests, and the gratings had a mean luminance of 40 cd/m^2. Linear summation was determined by the criteria of Hochstein and Shapley (28). A cell was considered to sum linearly if: *1*) its responses occurred primarily at the fundamental temporal frequency of the stimulus, and *2*) if the responses displayed a sinusoidal spatial-phase dependency, with little or no modulated response at one spatial phase (the null position), and a maximal response to a phase angle of 90° from the null position. Cells with nonlinear summation displayed significant response components at twice the temporal frequency of the stimulus, seen as doubling responses, and these were essentially independent of the spatial phase of the stimulus.

Electrophysiological recordings

We inserted bipolar tungsten stimulating electrodes across the optic chiasm to effect orthodromic activation of geniculate neurons. We also placed an array of four stimulating electrodes into the cortical gray matter of areas 17 and 18, and we used pairs of these as bipolar stimulation sites to elicit antidromic activation of geniculate cells. Antidromic activation was verified by canceling the antidromic spike via collision with an orthodromic spike. The latency of activation from the stimulation site was measured as the time from the stimulus artifact to the foot of the action potential or EPSP in the geniculate cell.

Microelectrodes were fashioned from omega-dot glass tubing (od, 1.2 mm; id, 0.7 mm) on a vertical puller. Electrodes were filled with a solution of 4 M potassium acetate and then beveled to a final impedance (measured at 100 Hz) of 20–70 MΩ. We used a hydraulic microdrive to advance the electrodes through the brain to the lateral geniculate nucleus. We recorded electrophysiological activity through a high-impedance amplifier equipped with a bridge and current injection circuitry. All recordings were displayed on an oscilloscope and stored on an FM tape recorder and computer. Recordings of PSPs were computer averaged (50–150 times, 15–20 kHz) to enhance the signal-to-noise ratio (see RESULTS).

We studied several response properties of each geniculate neuron in addition to PSPs. These included response latency to electrical stimulation of optic chiasm and cortex, ocular dominance, receptive field position, center size, center-surround properties, responses to the modulated sine-wave gratings, responses to standing contrasts, and responses to fast-moving targets. From these response properties, we identified geniculate neurons as X or Y cells (38, 54, 65). X cells were not subtyped as lagged or nonlagged (30, 42). From a combination of penetration depth and the ocular dominance of each neuronal response to visual stimuli, we established the laminar location of the neurons.

During a penetration, geniculate neurons were first encountered while recording extracellularly. We then advanced the electrode slowly until we observed fluctuations in the recorded potential. Cells were impaled either by applying brief (50 ms) positive current pulses of 2–10 nA through the electrode, by oscillating the amplifier through overadjustment of the capacitance compensation

circuit, or by physically tapping the microelectrode holder. Intracellular impalement was indicated by a DC drop of 40–75 mV, the appearance of PSPs, depolarizing action potentials, and a measurable input resistance. For about 75% of our cells, all physiological measurements, including characterization as X or Y, were performed during intracellular recording. We first characterized the remainder of the cells in our study as X or Y while recording extracellularly, and we then impaled the cell for analysis of PSPs. Both EPSPs and IPSPs were recorded following electrical stimulation of the optic chiasm. Because Eysel (16) reported that high-frequency stimulation can effect parameters of the PSPs, we maintained frequencies of optic chiasm stimulation < 4 Hz.

We adopted a set of minimum requirements for acceptable intracellular recording. These included: *1*) a resting potential of at least −40 mV; *2*) a spike amplitude of at least 20 mV; and *3*) an input resistance of at least 12 MΩ (cf. Ref. 5). We also took precautions to minimize artifacts introduced into our data by the recording system. Data were collected only from electrodes that, with optimum capacitance compensation, displayed sufficiently short time constants. Our criterion was a voltage change with a rise time (i.e., the time interval between 10 and 90% of the maximum response amplitude) of no greater than 0.4 ms in response to a square-wave current pulse; this criterion was always rechecked at the level of the lateral geniculate nucleus. We also required that electrodes showed no sign of rectification when currents up to 5 nA were delivered across the tip. Recordings were terminated if these electrode characteristics changed appreciably, and any data collected after the electrode was last verified to behave stably were discarded.

Statistics

Unless indicated otherwise, statistical analyses were made using the Mann-Whitney *U* test (37).

RESULTS

We obtained intracellular recordings that met our criteria (see METHODS) from 50 geniculate neurons, including 30 X cells and 20 Y cells. Three of the Y cells were located in lamina C, and the remaining Y cells plus all of the X cells were recorded in lamina A or A1. Because we found no appreciable differences in either EPSPs or IPSPs that related to a cell's location in lamina A, A1, or C, we do not further distinguish laminar location as an important variable in the presentation of data.

Relay cells vs. interneurons

Most of the geniculate neurons in our sample (19 X and 13 Y cells) were positively identified as relay cells on the basis of antidromic activation from the visual cortex (see METHODS). For 14 of the neurons (seven X and seven Y cells) we never attempted antidromic activation, because this was one of the last parameters we tested, and the cells were lost before we could do so. However, all of the response properties and PSPs of these neurons were indistinguishable from those of their identified relay cell counterparts. We thus suggest that these seven X and seven Y cells were also relay cells, although we lack conclusive evidence to prove this. In any case, the PSPs of all of these 46 presumed relay cells are described together below.

However, the final four neurons, all X cells, could not be antidromically activated from cortex despite repeated attempts to do so; neighboring cells in the same penetrations could be antidromically activated. Instead, three of these were activated transynaptically in response to electrical stimulation of the visual cortex, presumably in an orthodromic fashion. We suspect that these are interneurons (cf. Refs. 15, 39), although definitive electrophysiological criteria for geniculate interneurons are not yet available (22, 55). Interestingly, as is documented below, these putative interneurons differed dramatically from the other 46 neurons in terms of the pattern of PSPs evoked from electrical activation of the optic chiasm. For this reason, quantitative analyses of the PSPs from these four putative interneurons are treated separately below.

Qualitative observations of PSPs

Because the PSPs recorded in geniculate neurons following stimulation of the optic chiasm were small in amplitude (see below), it was necessary to increase the signal-to-noise ratio to allow for quantitative measures of response parameters. In some previous studies of geniculate cells, the amplitudes of PSPs were often increased by shifting the membrane potential by means of current injected through the recording electrode, thereby increasing the driving force for ions forming the synaptic current (2, 3, 39). However, we felt that, in light of the recent demonstration that thalamic neurons have voltage-

sensitive conductances (13, 33, 34, 51), such artificial manipulation of membrane potential could trigger intrinsic conductances, which, in turn, would contaminate the PSPs. Consequently, instead of manipulating membrane voltage, we chose to use computer averaging of synaptic potentials to enhance their fidelity, although in rare cases it was necessary to inject small hyperpolarizing currents into the cell (see below). Such averaging techniques have been used in previous studies of geniculate responses (60, 64).

One problem with computer averaging of PSPs evoked from optic chiasm stimulation is the need to eliminate the possible contamination of the averaged record by action potentials. We did not consider spontaneous action potentials to be a problem, because their brief duration and relatively random occurrence allowed them to be "averaged" out of the final record like other background noise. However, it was necessary to ensure that optic chiasm stimulation did not elicit action potentials in the geniculate cells, even on occasional trials, for any such action potentials would be synchronized with evoked PSPs, which would distort the final, averaged record. The technique used most often was simply to reduce the current used to stimulate the optic chiasm until the action potential was eliminated, leaving only the PSPs. However, when this procedure occasionally failed to eliminate the evoked spike, a small current (−0.5 to −1.0 nA) was applied through the recording electrode to hyperpolarize the cell and thus bring the membrane potential away from spike threshold. The seven cells for which this limited current injection was necessary did not differ from the others in terms of the nature of their evoked PSPs, so we feel that this did not introduce artifacts in the form of inappropriate voltage-sensitive conductances.

Figure 1*A* shows two superimposed, nonaveraged responses of a geniculate Y cell to electrical stimulation of the optic chiasm. Following such stimulation with a 0.5-mA current pulse, the cell responded with an all-or-none action potential (SPIKE) followed by an afterhyperpolarization (AHP). When the stimulating current was reduced to 0.2 mA, the cell responded with an EPSP (EPSP), which failed to reach threshold for generation of action potentials. An IPSP (IPSP) can be seen clearly as the hyperpolarization following the initial EPSP. Note that although the IPSP and afterhyperpolarization are both hyperpolarizing events, they can readily be distinguished in terms of rise time (or time to peak), amplitude, and duration. Although it is unclear in Fig. 1*A*, IPSPs could often be seen following the action potentials, and they outlasted the shorter-duration afterhyperpolarization. With the exception of the four X cells that we have described above as putative interneurons (see also *Interneurons*), each of our sampled cells showed a similar EPSP/IPSP response sequence following electrical stimulation of the optic chiasm.

Figure 1*B*, which depicts a computer-averaged response of a geniculate Y cell to electrical stimulation of the optic chiasm, illustrates the various parameters of the postsynaptic responses that served as the data base for our quantitative analyses. The *vertical arrow* designated as *a* denotes the time at which the optic chiasm is stimulated; the stimulus artifacts have been removed from all figures to simplify the illustrations. The latency of the EPSP is represented as *b*, the time between optic chiasm stimulation and the foot of the EPSP. Unfortunately, measures for the duration of the EPSP and the latency and duration of the IPSP are obscured by the fact that the IPSP seems to start during the decaying portion of the EPSP. We have thus had to adopt operational definitions for these parameters as follows. The duration of the EPSP (*c*) is the time from the onset of the EPSP to the time at which the response returns to the resting membrane potential. The observed onset of the IPSP (or "crossover latency") is the point in time at which the repolarization of the EPSP crosses the base-line membrane potential and begins the IPSP (i.e., $b + c$). Finally, the duration of the IPSP (*d*) is the time between this crossover latency and the time at which the IPSP repolarizes to the resting potential.

It has been asserted that the IPSP has a lower threshold than the EPSP, and the former can be isolated by lowering the amplitude of current used to stimulate the optic chiasm (2, 63). However, despite repeated attempts, we could never obtain responses to optic chiasm stimulation that displayed only an IPSP without a preceding EPSP. We were thus unable to obtain a direct measure of the

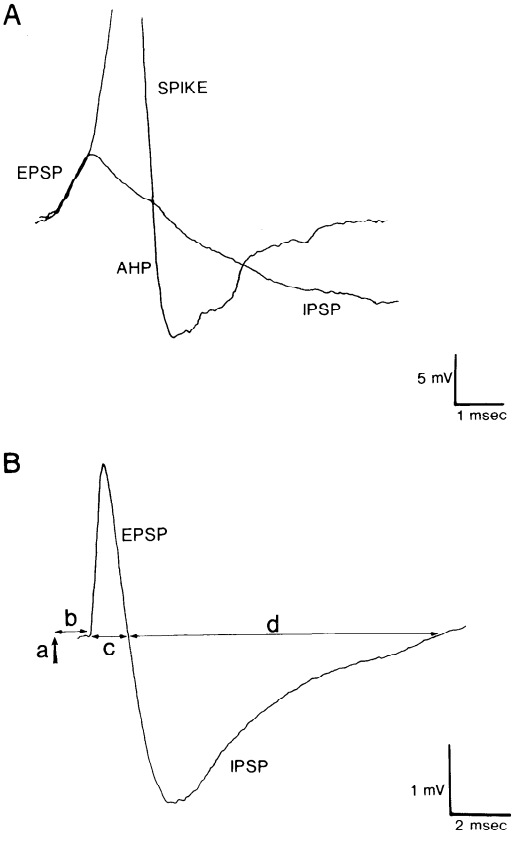

FIG. 1. Intracellular recordings from geniculate Y cells showing responses evoked from electrical stimulation of the optic chiasm. *A*: superimposed responses at two different levels of stimulation applied to the optic chiasm. Following stimulation with a 0.5-mA current pulse, the neuron responds with an action potential (SPIKE) followed by an afterhyperpolarization (AHP). When the stimulation current was lowered to 0.2 mA, the neuron's membrane potential did not reach threshold for initiation of an action potential, and its response consisted instead of an EPSP (EPSP) followed by an IPSP (IPSP). *B*: computer average of 100 responses to optic chiasm stimulation. The stimulus current was kept at a level low enough to prevent generation of action potentials in the neuron. The *arrow* (a) indicates the time of optic chiasm stimulation. This figure shows the temporal parameters of PSPs that were measured quantitatively in this study, which include EPSP latency (b), EPSP duration (c), and IPSP duration (d).

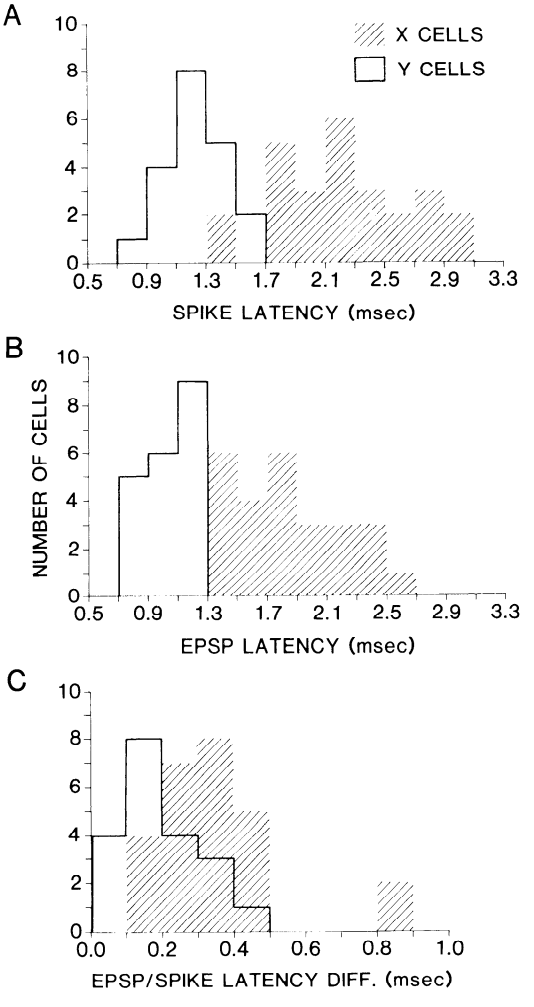

FIG. 2. Frequency histograms for X and Y cells of latency relationships of excitatory responses to stimulation of the optic chiasm. *A*: latency of intracellularly recorded action potentials. *B*: latency of EPSPs. *C*: difference between the latencies of EPSPs and action potentials as measured in the same individual neurons represented in *A* and *B*.

onset latency or duration for IPSPs. However, whereas our operational definitions as described above are clearly not the quantitative measures that would apply had the EPSPs and IPSPs occurred in isolation, they are nonetheless convenient and reliable indices that can be used as measures of PSPs among the neurons.

As noted above, only the four presumed interneurons among the X cells failed to display an IPSP in response to electrical stimulation of the optic chiasm. We have organized the remainder of RESULTS to treat these separately in a later section. Unless explicitly stated otherwise, all of the following data refer only to the remaining 46 geniculate neurons that exhibited both EPSPs and IPSPs in response to activation of the optic chiasm.

Relay cells

THRESHOLDS FOR ACTION POTENTIALS. An important parameter in signal transmission along any neuronal pathway is the membrane potential at which the conductances underlying the action potential reach threshold. Accordingly, although we did not systematically measure the threshold level for action potential initiation in our cells, we did make some preliminary measurements of the difference between the resting membrane potential and the potential at which the slowly rising EPSP changes to the fast-rising phase of the action potential. We measured this parameter for 12 cells (six X cells and six Y cells) chosen because they exhibited similar resting potentials, ranging from −58 to −62 mV. We found for these neurons that the thresholds for action potentials were 17 ± 4.2 mV (mean ± SD) above the resting potential, with no statistical difference between X and Y cells ($P > 0.1$).

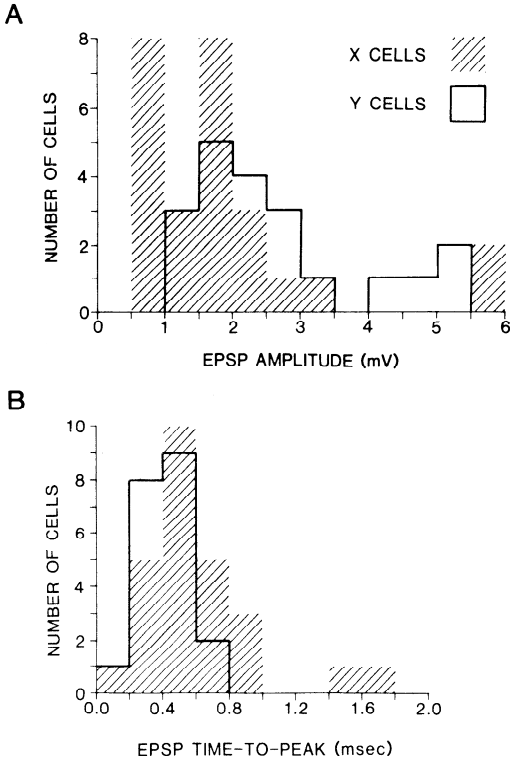

FIG. 3. Amplitudes and time-to-peak values of EPSPs in response to electrical stimulation of the optic chiasm for X and Y cells. *A*: frequency histogram of EPSP amplitudes. *B*: frequency histogram of EPSP time-to-peak values.

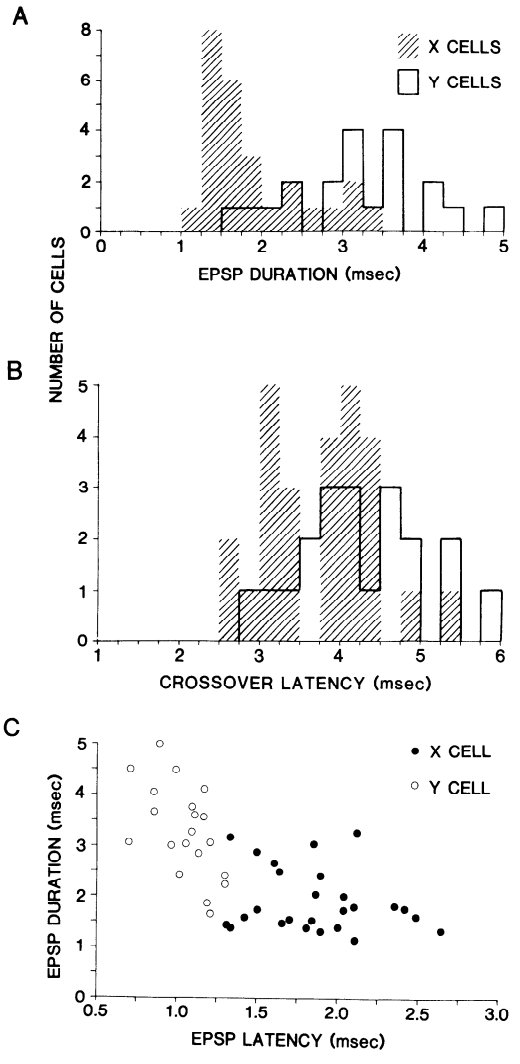

FIG. 4. Durations of EPSPs in response to optic chiasm stimulation for X and Y cells. *A*: frequency histogram of EPSP durations. *B*: frequency histogram of the crossover latencies (b + c in Fig. 1). *C*: relationship for individual neurons between latency and duration of EPSPs.

LATENCIES OF EPSPS AND ACTION POTENTIALS. Figure 2*A* illustrates for X and Y cells the latencies of the intracellularly recorded action potentials in response to electrical stimulation of the optic chiasm. As expected the latencies of X and Y cells overlapped slightly, and those for Y cells were significantly shorter than those for X cells (2.22 ± 0.43 ms for X cells vs. 1.24 ± 0.19 ms for Y cells; $P < 0.001$). Figure 2*B* shows that the latencies of EPSPs were also different between X and Y cells without overlap between

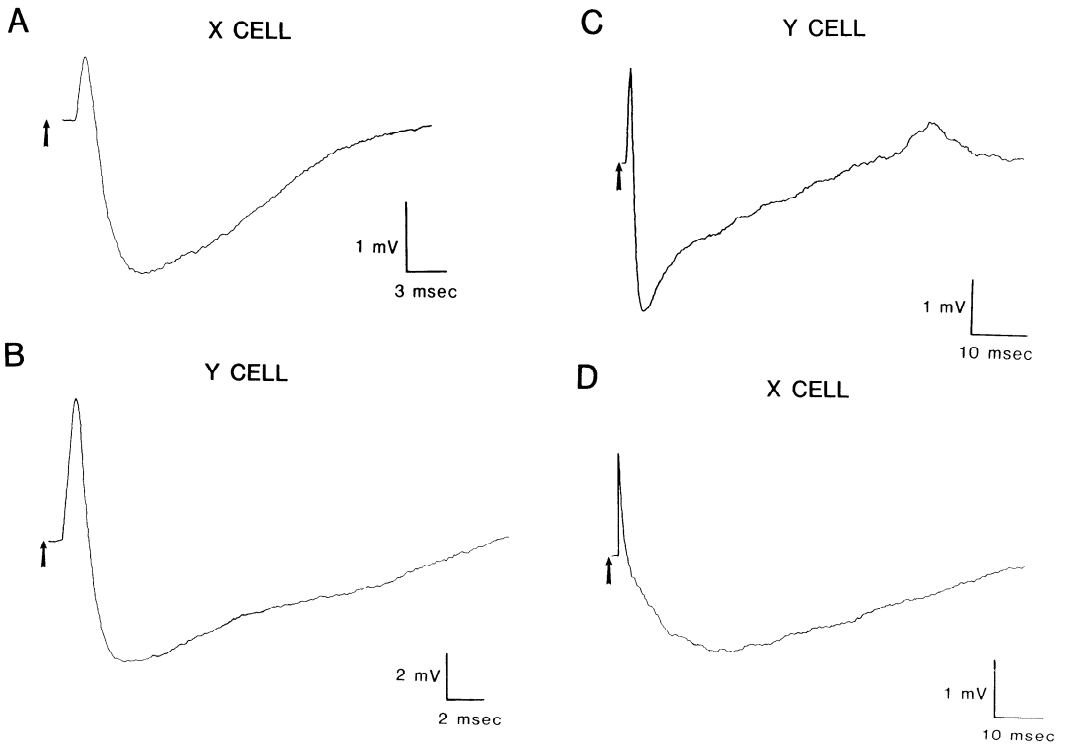

FIG. 5. Computer averages of responses in geniculate neurons to electrical stimulation of the optic chiasm showing examples of short-, medium-, and long-duration IPSPs; see text for details. The *arrow* preceding each trace indicates the onset of optic chiasm stimulation. Note the different time and voltage scales for each trace. *A*: short-duration IPSP for an X cell. *B*: short-duration IPSP for a Y cell. *C*: medium-duration IPSP for a Y cell. *D*: long-duration IPSP for an X cell.

cell types (1.86 ± 0.35 ms for X cells vs. 1.05 ± 0.17 ms for Y cells; $P < 0.001$).

A comparison of the difference in latency between the evoked EPSP and action potential for individual cells unveiled an interesting distinction between functional classes (Fig. 2*C*). This latency difference was significantly smaller for Y cells than for X cells (0.19 ± 0.10 ms for Y cells vs. 0.35 ± 0.17 ms for X cells; $P < 0.001$). This means that EPSPs generated in Y cells reach threshold for activation of action potentials sooner than those generated in X cells. Reasons for this are considered in DISCUSSION.

EPSP AMPLITUDES. Figure 3*A* shows that Y cells displayed EPSPs with larger amplitudes (2.60 ± 1.25 mV) than did X cells (1.87 ± 1.31 mV). This difference is statistically significant ($P < 0.01$). The quality of impalement could affect the EPSP amplitude, with poorer impalements leading to smaller EPSPs. However, we explain in DISCUSSION why we believe these EPSP amplitude differences between X and Y cells to be real and not an artifact of electrode impalement. Whereas it seems plausible that the greater amplitudes of Y-cell EPSPs might explain the shorter latency between EPSP and action potential onsets for these cells, we found no correlation in individual neurons between EPSP

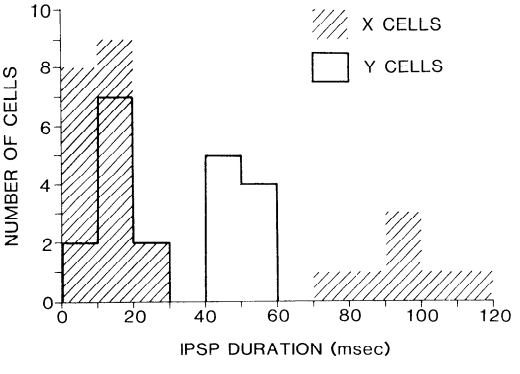

FIG. 6. Frequency histogram of IPSP durations in response to optic chiasm stimulation for X and Y cells.

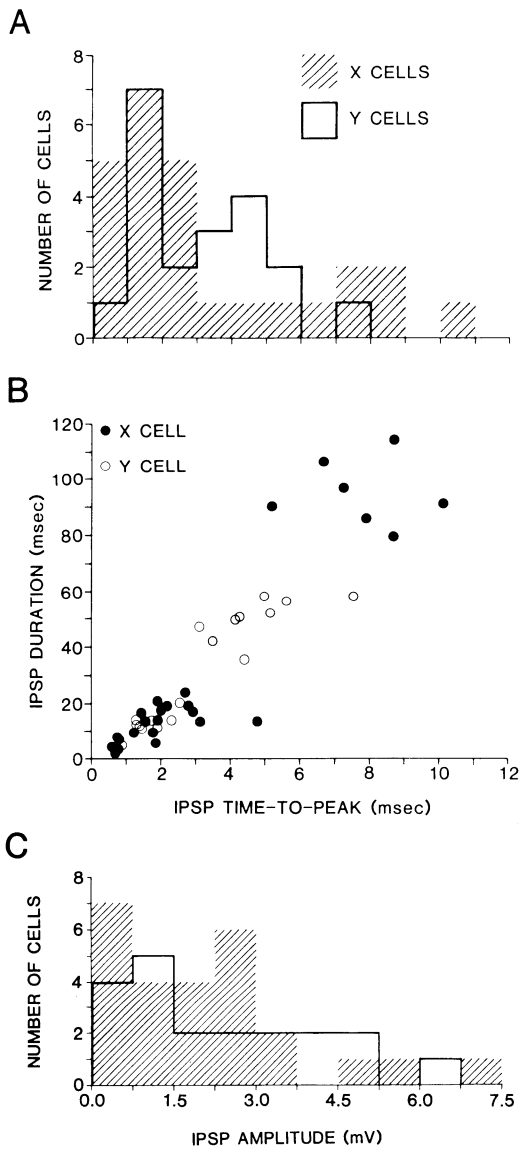

FIG. 7. Amplitudes and time-to-peak values of IPSPs in response to optic chiasm stimulation for X and Y cells. *A*: frequency histogram of IPSP time-to-peak values. *B*: relationship between IPSP duration and time-to-peak for individual neurons. C: frequency histogram of IPSP amplitudes.

amplitude and the latency difference between the EPSP onset and initiation of the action potential ($P > 0.1$ for all cells as well as for either subpopulation of X or Y cells). This is considered more fully in DISCUSSION.

EPSP TIME TO PEAK. The time to peak, or rise time, of an EPSP was defined as the time it took for the potential to grow from 10% of its maximum amplitude to 90% of this amplitude. As illustrated in Fig. 3*B*, Y cells display EPSPs with a slightly shorter response time to peak than do X cells (0.61 ± 0.33 ms for X cells vs. 0.43 ± 0.13 ms for Y cells; $P < 0.02$). However, we found no correlation in single neurons between rise time and the latency difference between the EPSP and action potential ($P > 0.1$ for all cells as well as for either subpopulation of X or Y cells). This suggests that faster rise times are not a major reason for the shorter action potential initiation times for Y cells.

The activation of synapses that are electrotonically farther from our recording site (i.e., presumably the soma) will produce PSPs that are both smaller in amplitude and slower in rise time. This is due to the electrotonic decay and charging of the capacitance across the membrane between the synaptic and recording sites (50). Thus all other factors being equal, both the larger amplitude and faster rise time seen in EPSPs recorded in Y cells would be produced if the excitatory synapses activated in Y cells were electrotonically closer to the soma than those for X cells. However, we found no correlation between rise time and amplitude of the EPSP ($P > 0.1$ for all cells as well as for either subpopulation of X or Y cells) (see DISCUSSION).

EPSP DURATIONS. We compared the duration of the EPSPs generated in X and Y cells following electrical stimulation of the optic chiasm (Fig. 4*A*). Although there was overlap in the values measured for X and Y cells, they were statistically different (1.91 ± 0.62 ms for X cells vs. 3.21 ± 0.83 ms for Y cells; $P < 0.001$). Given the shorter membrane time constants (5) and time to peak for the EPSPs seen in Y cells, the longer duration of the EPSPs for these cells requires another explanation. Analysis of four geniculate cells (two X cells and two Y cells) showed that the repolarization phase of the EPSPs follows a multiexponential decay. However, in contrast to what would be expected with a simple passive decay, the slope of the repolarization of these potentials increased with time, thereby indicating an active, hyperpolarizing process. This is most simply explained by the active termination of these EPSPs by the arrival of the subsequent IPSPs (see DISCUSSION).

Figure 4*B* shows for X and Y cells the crossover latencies (i.e., the sum of the la-

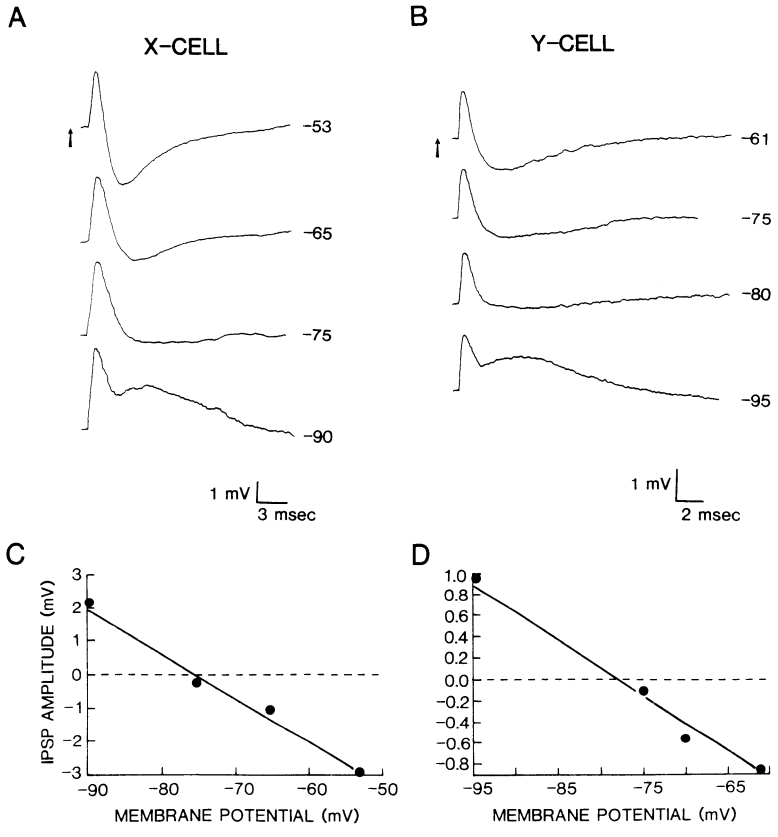

FIG. 8. Reversal potentials for short-duration IPSPs evoked by stimulation of the optic chiasm. *A*: computer-averaged responses of an X cell; *arrow* indicates onset of optic chiasm stimulation for all traces. The top trace shows the responses at the resting membrane potential, and in the traces below, the cell was hyperpolarized with injection of negative current through the electrode. The figures to right of all traces indicate membrane potential. Note the reversal of the IPSP as the membrane becomes increasingly hyperpolarized. *B*: computer-averaged responses of a Y cell; conventions as in *A*. *C*: plot of membrane potential vs. IPSP amplitude from the data in *A*. The reversal potential of the IPSP was extrapolated to −76 mV. *D*: plot of membrane potential vs. IPSP amplitude from the data in *B*. The reversal of the IPSP was extrapolated to −77 mV.

tency and duration of each EPSP as defined above). The crossover latency was significantly longer for Y cells than for X cells (3.77 ± 0.64 ms for X cells vs. 4.26 ± 0.77 ms for Y cells; $P < 0.05$). This longer crossover latency for Y cells means that, although their EPSPs are of relatively short latency (Fig. 2*B*), this is more than compensated by their longer durations (Fig. 4*A*).

Interestingly we found that the duration and latency of EPSPs were inversely related (Fig. 4*C*). This relationship was significant for geniculate neurons taken together ($P < 0.001$) and for Y cells alone ($P < 0.05$), but not for X cells alone ($P > 0.1$). A simple explanation for this observation, at least for Y cells, is that the EPSPs are terminated at a relatively fixed time after the stimulation of the optic chiasm. Thus EPSPs of shorter latency will have longer durations than those with longer latencies. If, as suggested above, the IPSP actively terminates the EPSP, this finding suggests further for individual Y cells that the latencies for onset of the EPSP and subsequent IPSP are independent (see DISCUSSION), with the latter occurring at a more fixed time among cells than the former.

IPSP DURATIONS. We were able to distinguish three types of IPSPs based on their durations. Examples are shown in Fig. 5. For 72% of the cells (88% of the X cells and 50% of the Y cells), these IPSPs were of fairly short duration, ranging from 0.7 to 23.8 ms (Fig. 5, *A* and *B*). The remaining Y cells displayed IPSPs with medium durations of 35.4–58.8

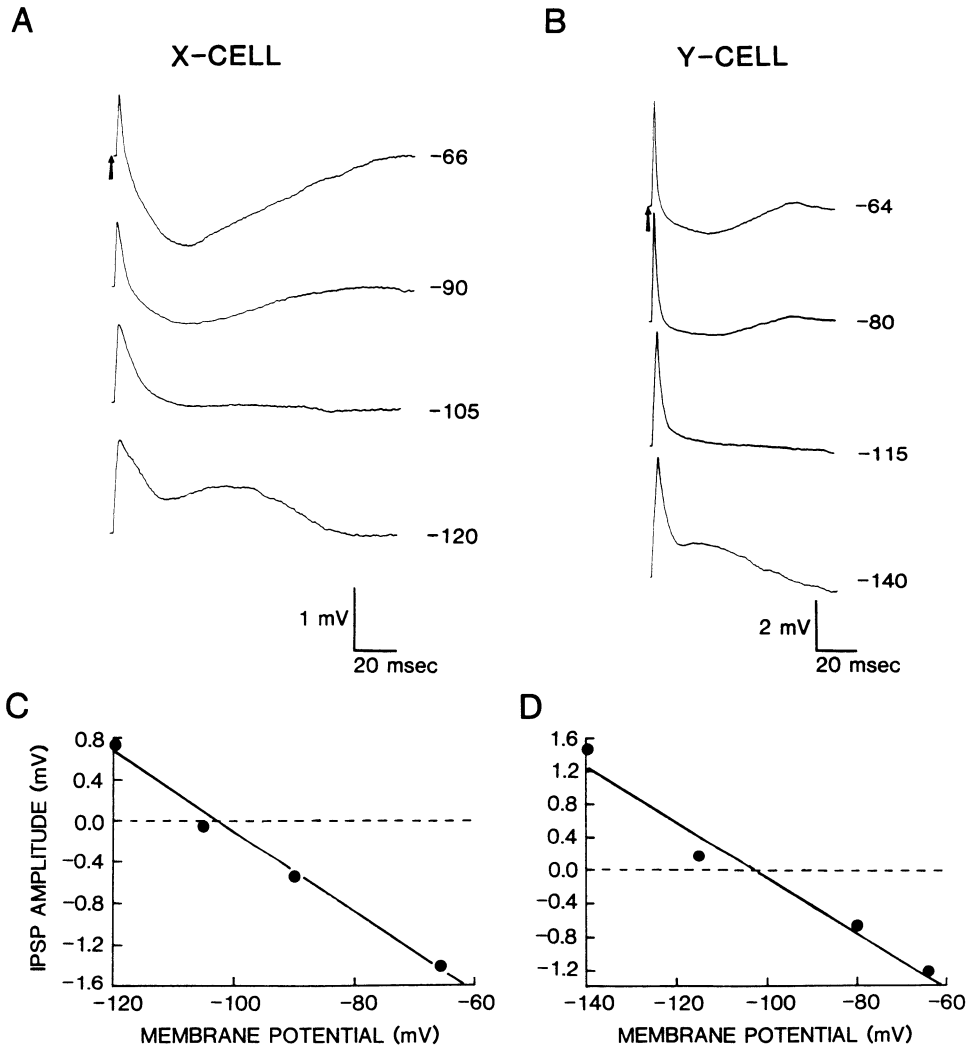

FIG. 9. Reversal potentials for medium- and long-duration IPSPs evoked from activation of the optic chiasm; all conventions as in Fig. 10. *A*: computer-averaged responses of an X cell displaying a long-duration IPSP. *B*: computer-averaged responses of a Y cell displaying a medium-duration IPSP. *C*: plot of membrane potential vs. IPSP amplitude from the data in *A*. The reversal potential of the IPSP was extrapolated to −102 mV. *D*: plot of membrane potential vs. IPSP amplitude from the data in *B*. The reversal of the IPSP was extrapolated to −102 mV.

ms (Fig. 5*C*), whereas for the remainder of the X cells these durations were quite long at 79.6–114.0 ms (Fig. 5*D*). Figure 6 shows the frequency histogram of these durations. We shall refer to these, respectively, as short-, medium-, and long-duration IPSPs. For the majority of neurons with only short-duration IPSPs, these were indistinguishable between X and Y cells, but for the remaining neurons, these potentials for Y cells were clearly shorter in duration than were those for X cells. Therefore, X cells had either short- or long-duration IPSPs, whereas Y cells had either short- or medium-duration ones.

IPSP TIME TO PEAK. We measured the time to peak for each IPSP to determine if there were any other differences that we could quantify in the three types of IPSP described above. As is summarized in Fig. 7*A*, we found that the time to peak of the IPSPs recorded in X cells (3.49 ± 2.86 ms) were slightly longer on average than those of Y cells (3.18 ± 1.76 ms), but

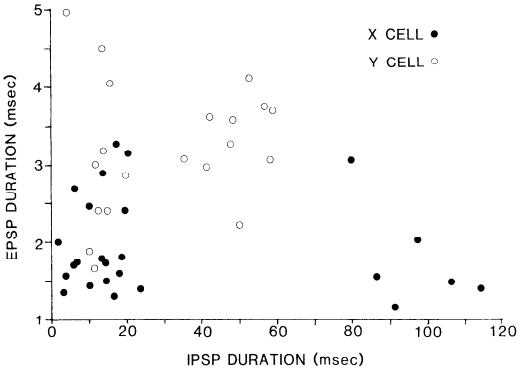

FIG. 10. Relationship in individual X and Y cells between the durations of the IPSP and EPSP evoked by stimulation of the optic chiasm.

this difference was not statistically significant ($P > 0.1$). However, Fig. 7*B* shows that the duration and time to peak of IPSPs were highly correlated ($r = +0.92$ and $P < 0.001$ for each of the X and Y populations alone as well as for the entire population). Inhibitory postsynaptic potentials with longer durations also took longer to reach peak amplitude, a point that can also be seen clearly in the records in Fig. 5. This provides an additional difference between the three types of IPSP.

Because the time to peak of the IPSP may be a simple reflection of a neuron's membrane time constant, we compared for individual geniculate cells the times to peak of the EPSPs and IPSPs. That is, cells with longer membrane time constants might be expected to exhibit slower rise times for all postsynaptic potentials. However, we found no correlation between the time-to-peak values of a neuron's evoked EPSP and IPSP ($r = +0.12$, $P > 0.1$). This suggests that the correlation seen between IPSP duration and IPSP time to peak is not an epiphenomenon of a cell's longer membrane time constant; it may instead reflect differences in the synaptic mechanisms generating the three types of IPSP (see DISCUSSION).

IPSP AMPLITUDES. The amplitudes of the IPSPs seen in geniculate neurons are illustrated in Fig. 7*C*. The amplitudes for X cells did not differ significantly from those for Y

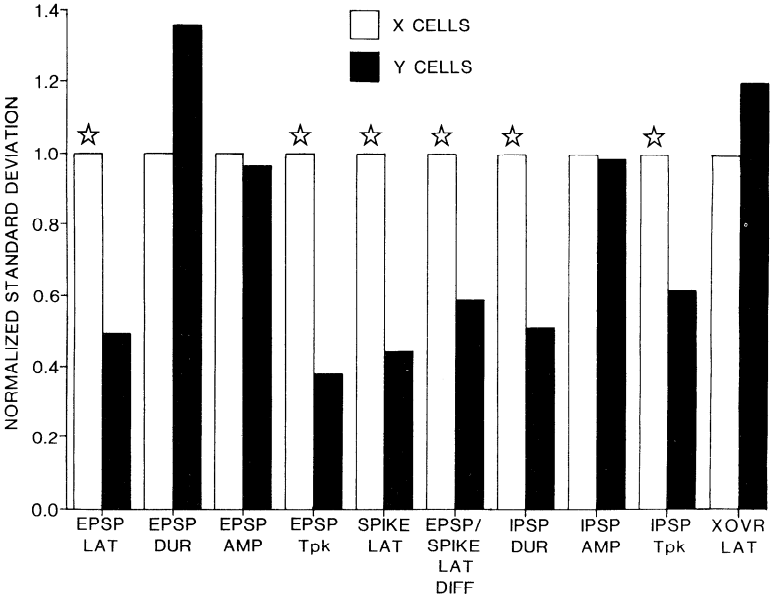

FIG. 11. Comparison for X and Y cells of the standard deviations for the 10 PSP parameters measured in this study (see text for details). The standard deviations have been normalized for easier comparison. *Stars* indicate those parameters for which a significant difference ($P < 0.01$–0.001) exists in variability. The parameters are as follows: EPSP LAT, latency of the EPSP; EPSP DUR, duration of the EPSP; EPSP AMP, EPSP amplitude; EPSP Tpk, EPSP time to peak; SPIKE LAT, latency of the action potential; EPSP/SPIKE LAT DIFF, latency difference between the onsets of the EPSP and action potential; IPSP DUR, duration of the IPSP; IPSP AMP, amplitude of the IPSP; IPSP Tpk, IPSP time to peak; XOVR LAT, crossover latency.

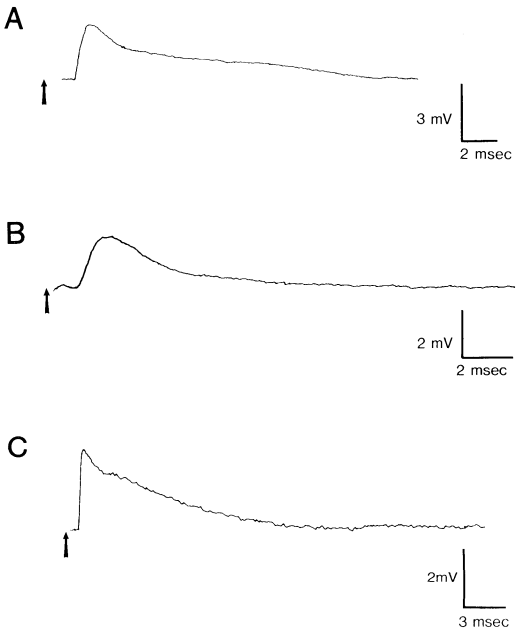

FIG. 12. Computer-averaged responses of putative interneurons to activation of the optic chiasm.

cells (2.12 ± 1.70 mV for X cells vs. 2.38 ± 1.68 mV for Y cells; $P > 0.1$). Even when we divided the IPSPs into short-, medium-, and long-duration types, we found no statistical differences among their amplitudes. In addition, we found no correlation between the amplitudes of EPSPs and IPSPs for individual neurons ($r = +0.24$, $P > 0.1$).

REVERSAL POTENTIALS OF IPSPS. In order to characterize further any differences among the various types of IPSP, we determined their reversal potentials by using current injection delivered intracellularly through the recording micropipette. Figure 8, *A* and *B*, illustrates the responses of an X and Y cell to electrical stimulation of the optic chiasm. Both cells exhibited only the short-duration type of IPSP. The top trace in each figure shows the response at the resting membrane potential, whereas in lower traces the membrane potential was varied as noted with injections of hyperpolarizing current. The IPSPs of both cells could be reversed at membrane potentials hyperpolarized with respect to the resting potential. Figure 8, *C* and *D*, shows the extrapolated reversal potentials, which are approximately −77 mV for both neurons. We similarly determined the reversal potentials for seven X cells and five Y cells that displayed only short-duration IPSPs. The average of the reversal potentials for these cells was −76.4 ± 7.9 (SD) mV. There was no significant difference found between the X and Y cells in this sample (−74.6 ± 6.4 mV for X cells vs. −79.0 ± 9.0 for Y cells; $P > 0.1$).

Figure 9 illustrates similar analyses that are typical of those for X cells with long-duration IPSPs and Y cells with medium-duration ones. For the X cell (Fig. 9, *A* and *C*), the reversal potential for the IPSP was extrapolated to −101 mV. We determined the reversal potential in a similar manner for two other X cells with long-duration IPSPs, and the average and standard deviation of the reversal potentials for the three X cells was −102 ± 6.4 mV. For the Y cell (Fig. 9, *B* and *D*), the reversal potential of the IPSP was likewise extrapolated to −101 mV. In this fashion, we determined the reversal potential for four Y cells with medium-duration IPSPs, and the average and standard deviation of the reversal potentials was −102.8 ± 6.3 mV.

The data of Figs. 8 and 9 indicate that the short-duration IPSPs for both X and Y cells reverse at the same membrane potential. Likewise, the long-duration IPSPs of X cells reverse at the same membrane potential as do the medium-duration ones of Y cells. Short-duration IPSPs reverse at a more positive potential than do the medium- and long-duration IPSPs. This suggests that two different ionic mechanisms may be involved in the inhibition of both geniculate X and Y cells following stimulation of the optic chiasm, but that each of the individual neurons apparently displays only one such mechanism (see DISCUSSION).

EPSP AND IPSP DURATIONS. As mentioned above and covered in more detail in DISCUSSION, a number of observations suggest that the EPSPs evoked by stimulation of the optic chiasm are abbreviated by the subsequent IPSPs. Thus the duration of the EPSP as we have defined it is largely determined by the latency of the IPSP. We thus thought it important to determine if the three types of inhibition we have seen control the duration of the preceding EPSP in different ways. Figure 10 shows the relationship between the durations of EPSPs and IPSPs for individual ge-

TABLE 1. *Parameters of postsynaptic potentials*

Cell Class	EPSP Lat., ms	EPSP Dur., ms	EPSP Amp., mV	EPSP Tpeak., ms	AP Lat., ms	AP-EPSP Lat., ms	IPSP Lat., ms	IPSP Dur., ms	IPSP Amp., mV	IPSP Tpeak., ms	XOVR Lat., ms
X	2.49	1.59	0.52	0.40	2.88	0.39	4.08	17.77	1.02	2.06	4.08
X	1.90	1.30	1.29	0.92	2.17	0.27	5.20	91.16	1.22	10.11	3.10
X	1.85	3.05	1.63	0.81	2.02	0.17	7.90	79.59	0.46	8.67	4.90
X	2.04	1.73	5.77	0.31	2.51	0.47	3.77	6.53	5.26	0.83	3.77
X	2.11	3.26	1.35	0.47	2.46	0.35	5.37	17.14	2.66	2.91	5.37
X	1.90	2.40	1.70	0.62	2.38	0.48	4.30	19.39	0.26	2.80	4.30
X	2.36	1.80	2.79	0.71	2.80	0.44	4.16	18.57	2.79	2.11	4.16
X	2.38	1.73	2.50	0.59	2.73	0.35	4.11	14.08	7.30	1.89	4.11
X	1.60	2.68	0.59	0.22	1.98	0.38	4.28	6.22	2.69	0.82	4.28
X	1.43	1.56	0.69	0.74	1.80	0.37	2.99	3.88	0.60	0.60	2.99
X_i	1.33	21.62	2.81	0.69	1.72	0.39					
X	1.50	2.89	0.85	0.52	1.81	0.31	4.39	13.61	0.38	4.79	4.39
X	2.04	1.99	1.60	0.48	2.29	0.25	4.03	1.75	0.38	0.62	4.03
X	1.50	1.70	1.87	0.18	1.73	0.23	3.20	5.85	3.75	1.87	3.20
X	1.80	1.39	1.80	0.64	2.61	0.81	3.19	23.80	1.92	2.77	3.19
X	2.01	1.36	0.96	0.42	2.24	0.23	3.37	3.00	0.48	0.80	3.37
X	2.63	1.31	1.44	1.63	2.91	0.28	3.94	16.32	2.50	1.52	3.94
X	1.31	1.44	3.50	0.87	1.42	0.11	2.75	10.00	1.75	1.32	2.75
X	1.87	2.01	1.73	0.61	2.12	0.25	3.88	97.16	2.71	7.21	3.88
X	2.11	1.15	2.01	0.58	2.92	0.81	3.26	91.01	1.76	5.20	3.26
X	2.11	1.78	0.90	0.30	2.50	0.39	3.89	13.12	2.71	3.17	3.89
X	1.63	2.45	0.95	1.48	1.81	0.18	4.08	10.00	0.79	1.81	4.08
X	1.33	3.14	5.77	0.31	1.47	0.14	4.47	20.41	0.58	1.92	4.47
X_i	2.10	17.50	1.27	0.56	2.45	0.35					
X_i	1.81	17.10	2.00	0.41	1.99	0.18					
X	1.34	1.40	1.71	0.51	1.78	0.44	2.74	114.02	3.18	8.72	2.74
X_i	1.64	19.08	3.42	0.49	1.92	0.28					
X	1.70	1.55	0.94	0.60	2.04	0.34	3.25	86.24	2.10	7.88	3.25
X	1.84	1.50	1.71	0.44	2.11	0.27	3.34	14.15	1.02	1.63	3.34
X	1.66	1.49	2.17	0.60	2.14	0.48	3.15	106.13	4.85	6.73	3.15
Y	1.13	2.86	1.20	0.62	1.33	0.20	3.99	19.64	0.34	2.51	3.99
Y	1.09	3.26	2.18	0.36	1.29	0.20	4.35	47.62	3.04	3.08	4.35
Y	1.16	4.10	5.24	0.52	1.20	0.04	5.26	52.38	0.90	5.11	5.26
Y	0.85	4.04	4.79	0.47	1.02	0.17	4.89	15.50	3.51	1.57	4.89
Y	1.00	3.01	1.82	0.41	1.32	0.32	4.01	11.55	0.74	1.40	4.01
Y	0.71	3.08	2.69	0.47	0.86	0.15	3.79	35.37	0.48	4.52	3.78
Y	1.16	3.57	1.75	0.58	1.24	0.08	4.73	48.00	1.84	4.22	4.73
Y	1.19	1.66	4.20	0.49	1.29	0.10	2.85	11.16	6.01	1.88	2.85
Y	0.99	4.49	2.12	0.21	1.30	0.31	5.48	13.39	1.02	1.27	5.48
Y	0.71	3.18	5.05	0.17	0.95	0.24	3.89	13.61	1.44	1.68	3.89
Y	1.30	2.40	2.47	0.47	1.62	0.32	3.70	14.60	4.20	2.31	3.70
Y	1.11	3.61	1.70	0.40	1.57	0.46	4.72	42.11	1.48	3.51	4.72
Y	1.06	3.06	2.81	0.51	1.33	0.27	4.12	58.01	2.38	7.59	4.12
Y	1.21	2.98	1.02	0.36	1.29	0.08	4.19	41.31	1.73	3.97	4.91
Y	0.88	4.98	1.71	0.71	1.10	0.22	5.86	3.92	0.71	0.81	5.86
Y	1.02	2.40	3.26	0.34	1.20	0.18	3.42	12.25	2.69	1.97	3.42
Y	1.19	1.87	2.36	0.37	1.36	0.17	3.06	9.80	1.01	1.40	4.06
Y	1.10	3.74	1.65	0.39	1.27	0.17	4.84	56.42	5.10	5.68	4.84
Y	1.30	2.21	1.30	0.46	1.38	0.08	3.51	49.97	4.91	4.22	3.51
Y	0.85	3.68	2.61	0.32	0.97	0.12	4.53	58.78	4.10	4.90	4.52
Totals (mean ± SD)											
X	1.86 ± 0.35	1.91 ± 0.62	1.87 ± 1.31	0.61 ± 0.33	2.22 ± 0.43	0.35 ± 0.17	3.97 ± 1.02	34.65 ± 37.48	2.12 ± 1.70	3.49 ± 2.86	3.77 ± 0.64
X_i	1.72 ± 0.28	18.83 ± 1.73	2.38 ± 0.81	0.54 ± 0.10	2.02 ± 0.27	0.30 ± 0.08					
Y	1.05 ± 0.17	3.21 ± 0.83	2.60 ± 1.25	0.43 ± 0.13	1.24 ± 0.19	0.19 ± 0.10	4.26 ± 0.77	30.77 ± 19.16	2.38 ± 1.68	3.18 ± 1.76	4.26 ± 0.77

Dur., duration; Lat., latency; Amp., amplitude; AP, action potential; Tpeak., time to peak; XOVR, crossover; X, X relay cells; X_i, interneurons; Y, relay Y cells.

niculate X and Y cells. We found no correlation between these variables ($r = -0.10$, $P > 0.1$ for all cells). Furthermore, there was no tendency among X cells for short- and long-duration IPSPs to be associated with different durations of EPSPs, and a similar lack of tendency existed for Y cells with short- vs. medium-duration IPSPs.

VARIABILITY IN PSP PARAMETERS. Considerable prior evidence exists that geniculate X cells display much more heterogeneity as a population than do Y cells (see DISCUSSION). We therefore compared the variability between X and Y cells in terms of their PSPs evoked from electrical activation of the optic chiasm. As illustrated in Fig. 11, the normalized standard deviations for these measures were larger for X cells than for Y cells in 8 of 10 cases, and in all 6 cases in which the differences were statistically significant (indicated by *stars*, $P < 0.05$–0.001 on F tests), X cells displayed more variability. These observations indicate that there is more heterogeneity among geniculate X cells than among Y cells with respect to the PSPs examined in the present study.

Interneurons

As previously mentioned, among our sample of 30 geniculate X cells we recorded from 4 neurons that could not be antidromically activated with electrical stimulation of the visual cortex. Unique among our neuron sample, these cells displayed no obvious IPSP following electrical stimulation of the optic chiasm. Examples of such responses are illustrated in Fig. 12. As far as we could tell, the receptive field properties of these cells were identical to those seen for the other X cells, and their response latencies to stimulation of the optic chiasm were clearly within the X cell range (see Table 1). Although we could not antidromically activate any of these cells from electrical stimulation of the visual cortex, three of the cells could be driven orthodromically from the visual cortex. These results suggest the possibility that these four X cells without evident IPSPs in our recordings were geniculate interneurons (15). We encountered no Y cell responses during this study which lacked an IPSP following electrical stimulation of the optic chiasm.

As might be expected from the difference in the presence of IPSPs, these putative interneurons and the putative relay X cells also differed in the durations of EPSPs evoked by stimulation of the optic chiasm (Fig. 13). Such durations for the relay X cells (1.91 ± 0.62 ms) were significantly shorter than those for the interneurons (18.83 ± 1.78 ms; $P < 0.001$). We also compared other EPSP parameters of relay X cells and interneurons and found no statistical differences between these cells types in terms of latency, amplitude, or time to peak of response ($P > 0.1$ for all measures). Thus, except for the duration of EPSPs and the presence or absence of IPSPs, the receptive field properties and responses to activation of the optic chiasm were indistinguishable between relay X cells and interneurons (55). Perhaps had we tested receptive field properties expected to be affected by IPSPs evoked by optic chiasm stimulation, properties such as peak firing rates or the tonicity of response, we would have documented differences between relay X cells and interneurons.

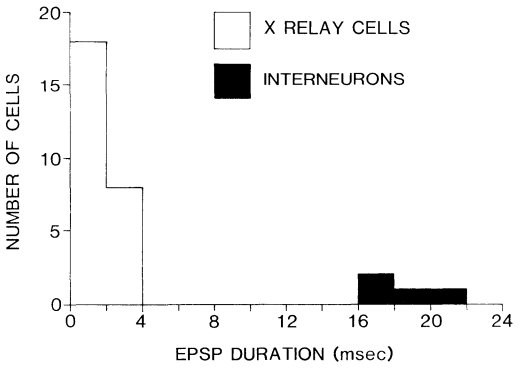

FIG. 13. Frequency histogram for all cells identified as X cells of the duration of the EPSP evoked by activation of the optic chiasm.

DISCUSSION

We have quantitatively examined several parameters of the PSPs elicited in geniculate neurons following electrical activation of the optic chiasm. Because intracellular recordings were obtained using an in vivo preparation, we were able to relate this synaptic activity of geniculate neurons to their identification as X or Y cells. We have found significant differences in the evoked PSPs between X and Y cells, as well as differences between neurons within the same functional

class. Our findings relevant to the retinogeniculate pathways lead to certain conclusions concerning synaptic circuitry, spatial and temporal integration of synaptic inputs, postsynaptic receptors, and ionic conductances. These conclusions are discussed below.

Relay cells

EPSP AMPLITUDE. We observed that electrical activation of the optic chiasm evoked EPSPs that were larger, on average, in Y cells than in X cells. We considered the possibility that this difference could be an artifact due to better impalements of Y cells than of X cells. Y cells have larger somata than do X cells (5, 22). Following electrode penetration, larger somata might experience less cell injury and/or develop a better membrane seal around the electrode tip, thereby producing a relatively higher input resistance. If so, then less of the synaptic current would be shunted through the less damaged soma membrane, and a larger amplitude EPSP would result.

However, for several reasons, we do not believe that this EPSP amplitude difference between X and Y cells is an artifact. First, if the quality of our intracellular impalements were a significant factor in our measured EPSP amplitudes, it should similarly affect IPSP amplitudes. However, we found no correlation between these two measures within individual neurons. Second, a lower input resistance due to shunting through damaged soma membranes would also result in a lower resting membrane potential. This did not appear to be the case, because we found no difference in this parameter between X and Y cells; we also observed no obvious differences in terms of ease of impalement or duration of intracellular recording between these cell types (see also Ref. 5). Third, we have found in this and previous studies that X cells have higher input resistances than do Y cells (5).

There may be many reasons why Y cells should develop larger EPSP amplitudes in response to optic chiasm stimulation than do X cells. One likely candidate for at least part of this difference is that the dendritic locations of synapses from retinogeniculate axons are electrotonically closer to the somata for Y cells than for X cells. Indeed, Y cells are slightly more compact electrotonically than are X cells (5). Furthermore, Y cells receive most of their retinal synapses on the large diameter shafts of primary dendrites quite close to the soma, whereas X cells tend to receive retinal input slightly farther from the soma on appendages of dendrites that are thinner than those of Y cells (25, 66). Not only are EPSPs in Y cells larger in amplitude than those in X cells (Fig. 3*A*), but they also exhibit faster rise times (Fig. 3*B*). Both of these features are consistent with retinogeniculate synapses that are electrotonically closer to the somata. Whereas part of the explanation for EPSP differences in amplitude and rise time between X and Y cells may be that retinogeniculate synapses are electrotonically closer to the somata of Y cells than of X cells, the lack of any correlation between EPSP amplitude and rise time among individual cells suggests that factors other than electrotonic parameters must also play a strong role in these differences.

EPSP AND ACTION POTENTIAL LATENCY DIFFERENCE. We observed that a significantly shorter interval existed in Y cells compared to X cells between the onset of the EPSP and that of the action potential evoked from optic chiasm stimulation. Several explanations can be considered for this difference between cell types. First, EPSPs of Y cells are greater in amplitude than are those of X cells (Fig. 3*A*). A larger-amplitude EPSP would permit the postsynaptic to reach its threshold for action potentials more quickly. Second, the EPSPs generated in Y cells might have a faster rise time (time to peak) than those for X cells, and thus they will reach the threshold for spike initiation sooner. A faster rise time is consistent both with a shorter electrotonic distance between the retinogeniculate synapses and the somata (see above) and with prior evidence that Y cells have shorter membrane time constants than do X cells (5). As noted above, the EPSP rise times are indeed faster for Y cells than for X cells (Fig. 3*B*). Third, if Y cells have lower thresholds for action potentials than do X cells, an EPSP could more rapidly depolarize the cell to its threshold level for action potential initiation. However, our observations from six X cells and six Y cells suggest no such difference (see RESULTS).

We can thus speculate that some combination of EPSP amplitude and rise time plays a role in action potential initiation times that are shorter for Y cells than for X cells. However, the lack of a strong correlation for individual neurons between the action potential

initiation times and either EPSP amplitudes or EPSP rise times indicates that neither EPSP factor strongly or solely determines action potential initiation times. Many other parameters, as yet undefined, may also play important roles in determining these initiation times.

EPSP DURATION. The duration of an evoked EPSP is obviously of considerable functional importance in the operation of neuronal circuits. We found that the durations of EPSPs evoked by electrical stimulation of the optic chiasm varied for the different neuronal groups. These durations were dramatically longer for the cells we suspect of being interneurons (i.e., the four X cells without evoked IPSPs; see also below) than for the presumptive relay cells; among the latter, these durations were longer for Y cells than for X cells. Given our operational definition of EPSP duration (see RESULTS), it is worth considering how this duration may be determined in our recording situation before considering the significance of these duration differences among cell types.

We believe that a major determinant of the EPSP duration is the subsequent arrival of the IPSP, which actively terminates the EPSP. Several lines of evidence are consistent with this view. First, the repolarization phase of the EPSP shows an increasing slope with time, indicative of an active process. Second, spontaneous EPSPs that are not followed by an IPSP display much longer durations than those evoked from optic chiasm stimulation, which are followed by an IPSP (35, 43; our own unpublished results). Third, the EPSPs recorded in the 4 putative interneurons, which lack IPSPs, last nearly 10 times longer than do EPSPs recorded in the remaining 46 neurons. Fourth, when the IPSPs were reversed using extrinsic current injection and their influence on the preceding EPSPs is thus diminished, the durations of the EPSPs were clearly increased; under these conditions of IPSP reversal, the EPSPs of X cells can even have a longer duration than those of Y cells (see Figs. 8 and 9).

If EPSP duration in our recording situation were determined largely by the latency difference between the onset of the EPSP and that of the later IPSP, then this latency difference must be greater for Y cells than for X cells. The EPSPs are clearly initiated in Y cells before they are in X cells. Given this, the EPSP duration differences we noted between cell types would result if the latencies of all IPSPs evoked from optic chiasm stimulation were relatively fixed in time compared with the EPSP latencies. Our evidence that, at least for Y cells, EPSPs that begin earlier have longer durations (Fig. 4*C*) is consistent with this interpretation; that is, with a subsequent IPSP of relatively fixed latency, an earlier EPSP would last longer. The significance of this correlation between EPSP latency and duration is considered more fully in the following section.

This explanation for the major determinant of EPSP duration focuses on IPSPs, but several other explanations for the longer EPSPs of Y cells than of X cells can also be considered. First, the conductance change underlying the EPSP may be longer for Y cells, either because the membrane channel openings are longer or because activity among the many retinal synapses is less well synchronized and thus spread over more time. However, at least the latter explanation would imply a more prolonged development of the EPSP that would be seen as a longer rise time for Y cells than for X cells, and, as emphasized in the above paragraphs, we have observed precisely the opposite (see Fig. 3*B*). Second, the IPSPs may be weaker in Y cells and thus repolarize the EPSPs more slowly. This seems unlikely, because we observed neither a difference in IPSP amplitude between X and Y cells (Fig. 7*C*) nor a relationship between IPSP amplitude and EPSP duration ($r = +0.17$, $P > 0.1$). Third, the EPSPs may be stronger in Y cells and thus are offset more slowly by the subsequent IPSP. Whereas we did observe larger amplitude EPSPs for Y cells than for X cells (Fig. 3*A*), we found no clear relationship between EPSP amplitude and EPSP duration ($r = +0.22$, $P > 0.1$).

LATENCY VS. DURATION OF EPSPS. A significant negative correlation exists for Y cells between the latency and duration of EPSPs evoked by optic chiasm activation (Fig. 4*C*). In view of the abovementioned conclusion that the IPSP terminates the EPSP, this suggests that the inhibition recorded in Y cells has a fixed latency from optic chiasm stimulation that serves to terminate the EPSP at a relatively fixed latency. This, in turn, implies that the EPSP and IPSP latencies are independent. Thus different retinogeniculate ax-

ons must subserve the excitatory and inhibitory inputs to these Y cells. Because these geniculate neurons are monosynaptically innervated by retinogeniculate Y axons, either other Y axons or possibly X axons must provide inputs to the inhibitory circuits that innervate the Y cells under consideration. The lack of correlation for X cells between EPSP latency and duration does not support such a fixed latency inhibition for these neurons.

Prior studies of the latencies of EPSPs and IPSPs evoked by optic chiasm stimulation have reached conflicting conclusions concerning these latency relationships. Singer and Bedworth (63) reported that the latency difference between EPSPs and IPSPs was greater for Y cells than for X cells, findings that are consistent with our conclusions. Eysel (16), however, found no clear relationship between the EPSP and IPSP latencies among individual cells, but because he did not explicitly identify his neurons as X or Y, it is not completely clear how to relate these conclusions to our own. Finally, Lindstrom (39) reported that the difference between the EPSP and IPSP latencies was always 0.7–0.8 ms for both X and Y cells, which directly contradicts our conclusions.

FEEDBACK VS. FEEDFORWARD INHIBITION. Two types of GABAergic neuron seem to be the source of local inhibitory inputs to geniculate relay cells of laminae A and A1 (20, 44, 45, 48). These types are the interneurons, which lie amongst the relay cells, and cells of the perigeniculate nucleus, which form a thin cellular layer just dorsal to lamina A. Recent electrophysiological studies have implicated these two cell types in different forms of inhibition evoked from activation of the optic chiasm: *1*) a feedforward inhibition over a disynaptic pathway consisting of a retinal axon innervating an interneuron that, in turn, innervates the relay cell; and *2*) a feedback inhibition over a trisynaptic pathway consisting of a retinal axon contacting a relay cell that innervates a perigeniculate cell (via an axon collateral) that, in turn, projects back to innervate geniculate relay cells (2, 3, 15, 39, 61, 63). Feedback inhibition evoked from activation of the optic chiasm usually should have a longer latency than does feedforward inhibition because of the additional synapse, although the faster conduction velocities of Y axons compared to X axons can complicate this.

Lindstrom (39) has used latency measurements to argue that both types of inhibition are present in both X and Y cells. Our own data bear on this indirectly. As we have noted in RESULTS, we were technically unable to measure directly the latencies of IPSPs evoked from electrical activation of the optic chiasm. However, if our argument that the IPSP terminates the prior EPSP is correct, then we can infer the relative difference in onset times between the EPSP and the IPSP from the EPSP duration. When we do so, we arrive at conclusions that differ subtly from the conclusions of Lindstrom (39).

As shown in Fig. 4*A*, EPSP durations were longer for Y cells than for X cells. Indeed, these durations were only 1–2 ms for most X cells, whereas they were greater than 2.5 ms for most Y cells. This suggests one of two hypotheses for most X cells: either the initiation of the EPSP precedes that of the IPSP by only a single synapse, which is consistent with feedforward inhibition; or feedback inhibition to X relay cells is activated by retinogeniculate Y axons, which conduct more rapidly than the X axons producing the EPSP, and this more rapid conduction permits the squeezing of two synaptic delays between the onsets of the EPSP and IPSP. A complementary set of hypotheses emerge for most Y cells: either the EPSP precedes the IPSP by two synaptic delays, which is consistent with feedback inhibition; or the IPSP is activated via retinogeniculate X axons, which conduct less rapidly than do the Y axons leading to the EPSP, and this adds an additional delay between the onsets of the EPSP and IPSP. In other words, this latter hypothesis for relay Y cells implies that these neurons are innervated by a feedforward inhibitory pathway involving retinogeniculate X axons. This is consistent with other evidence that most or all interneurons are innervated by retinogeniculate X axons (22, 26, 55).

MECHANISMS FOR IPSP GENERATION. Based on duration and time to peak, we found three distinct types of IPSP that could be evoked from electrical activation of the optic chiasm. In RESULTS, we refer to these as short-, medium-, and long-duration IPSPs. However, when neurons were classified, we found that two distinct types of IPSP occur in both X

and Y cells. Both classes contained cells that displayed either a shorter duration IPSP that reversed at −76 mV or a longer duration IPSP that reversed at −102 mV. Among the latter, X cells displayed only long-duration IPSPs, whereas Y cells displayed only medium-duration IPSPs. The differences in reversal potential suggest that there are at least two different types of ionic conductance changes that subserve IPSP generation.

As noted in the preceding section, the IPSPs seen in relay cells are thought to result from the activity of local, GABAergic neurons, which are the interneurons and perigeniculate cells (20, 44, 45, 48). Two types of GABAergic synapses have been encountered in the central nervous system. Many operate via a classic, bicuculline-sensitive, postsynaptic $GABA_A$ receptor by increasing the conductance to chloride ions (12, 14, 53). Because the equilibrium potential for chloride ions (i.e., −60 to −75 mV) is normally close to the resting potential, activation of these synapses polarizes the membrane minimally. Instead, they inhibit by shunting currents generated by EPSPs (for a discussion of this, see Refs. 36, 56). A second type of GABA synapse works via a bicuculline-insensitive, postsynaptic $GABA_B$ receptor by increasing the conductance of potassium ions (7–9, 46, 59). Because the equilibrium potential of potassium is normally −90 to −100 mV, the resultant IPSP is expressed as a large hyperpolarization. Finally, at least in hippocampal neurons, $GABA_A$ responses have faster rise times and shorter durations than do $GABA_B$ responses (46).

The different reversal potentials and temporal properties seen for the two types of inhibition displayed by X and Y cells suggest that they correspond to synaptic activation of $GABA_A$ and $GABA_B$ receptors. Although we did not find a difference in the amplitudes of these two IPSP types, this may not be surprising because many factors, such as amount of transmitter released and electrotonic distance of the synapse from the soma, can affect this variable. We thus conclude that both types of GABAergic inhibition (i.e., $GABA_A$ and $GABA_B$) are found in both classes of geniculate relay cell (i.e., X and Y). Considerable pharmacologic evidence exists to support the presence of $GABA_A$ responses among neurons of the cat's lateral geniculate nucleus. Much of the inhibition seen in these cells can be blocked by application of bicuculline (17, 49, 58) or reversed by intracellular injection of chloride ions (39). Although the existence of $GABA_B$ receptors has yet to be demonstrated unequivocally in the cat's lateral geniculate nucleus, $GABA_B$ receptors appear commonplace in the mammalian thalamus (9).

In our recordings, an individual cell displayed either a short-, medium-, or long-duration IPSP in response to activation of the optic chiasm. We found no differential receptive field properties that might correlate to such different IPSPs. It should be noted that each of these evoked IPSPs was actually the summed response of many individual synaptic events. This leads to the possibility that a single cell actually has more than one type of IPSP. An X cell could, for example, display a long-duration IPSP that obscures a short-duration IPSP. Two lines of evidence argue against this. First, the three IPSP types showed different time to peaks, so we would expect that, if two or more IPSP types were superimposed, there would be clear, multiple peaks in the response waveform. We never observed such multiple peaks. Second, the short-duration IPSPs displayed a different reversal potential than did either the medium- or long-duration IPSPs. If two different IPSP types were evoked in a cell, modulation of the membrane potential (as illustrated in Figs. 8 and 9) would reveal two inversions of the hyperpolarization, one at about −76 mV with a relatively fast rise time for its component, and a second one at about −102 mV with a longer rise time. Such multiple inversions were never seen.

If indeed the different IPSP types correspond to $GABA_A$ and $GABA_B$ responses, then we found no evidence of any IPSP for an individual neuron that might be a mixture of $GABA_A$ and $GABA_B$ responses. Perhaps each neuron expresses only either $GABA_A$ or $GABA_B$ receptors, with no mixing. This possibility needs to be explored more fully in future experiments in light of the recent in vitro study demonstrating both short- and long-latency IPSPs in single neurons of the rat's lateral geniculate nucleus (27).

VARIABILITY IN EPSPS AND IPSPS. A variety of morphological and electrophysiological observations suggest that geniculate X cells as a group display considerably more heterogene-

ity than do Y cells. X cells are much more variable than are Y cells in terms of dendritic morphology (5, 22, 31). The X cells display great variation in dendritic branching patterns, distributions of dendritic appendages, and shapes of individual dendrites and dendritic arbors, whereas Y cells seem to be scaled versions of one another, differing only in size. Related to this, X cells display much more heterogeneity in their passive cable properties than do Y cells (5). Physiological studies have described variability in the response properties of X cells to such an extent that subtypes have been proposed (e.g., the "normal" and "lagged" X cells; see Refs. 30, 41, 42). No such subtypes have yet been described for Y cells. Although it is not at all clear the extent to which the assorted morphological and electrophysiological variables relate to one another, there is a growing body of evidence that X cells are considerably more heterogeneous as a neuronal group than are Y cells.

We have noted a similar difference in variability between X and Y cells for the EPSP and IPSP parameters measured in the present study. Again, X cells display much more variability than do Y cells, as is summarized by Fig. 11. Our data thus adds to the evidence that X cells form a more heterogeneous class of neuron than do Y cells. Unfortunately, we cannot yet suggest a functional correlate for this variability.

Interneurons

We recorded from four X cells that lacked an IPSP following electrical stimulation of the optic chiasm. Instead, these neurons displayed only EPSPs with durations approximately 10 times that seen for EPSPs recorded in other X cells. Because of our inability to activate these cells antidromically from visual cortex, we consider these cells to be interneurons (see RESULTS). Previous intracellular recordings thought to be from interneurons, although few in number, have also failed to demonstrate IPSPs in response to stimulation of the optic chiasm (3, 47).

The lack of an IPSP in interneuron recordings might be taken to suggest that these cells are not the target of pathways subserving inhibition. However, intracellular recordings from geniculate interneurons can be most difficult to interpret because of their interesting and unique morphology (26, 55). Bloomfield and Sherman (6) have shown recently that major portions of the dendritic arbor of each interneuron are electrically isolated from the soma (and axon), and we presume that most or all of our intracellular recordings are from somata. (For relay cells, such isolation seems not to occur, because these cells and their dendritic arbors are electrotonically compact; see Ref. 5.) These electrically isolated dendrites of interneurons are not only a major postsynaptic target of afferents, they also maintain large numbers of output synapses (18, 26, 45). Presumably the recordings from the soma reflect only proximal synaptic inputs that control the cell's axonal output. Many of the important events in interneurons related to more distal dendritic locations, including many dendritic outputs and the EPSPs and IPSPs that control them, were possibly invisible to our recording electrodes. Interneurons might thus possess powerful inhibitory synaptic activity evoked from stimulation of the optic chiasm that remained unrecorded in our experiments.

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Present address of S. A. Bloomfield: Dept. of Ophthalmology, New York University Medical Center, 550 First Ave., New York, NY 10016.

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