

Morphology and Axonal Projection Patterns of Individual Neurons in the Cat Perigeniculate Nucleus

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SUMMARY AND CONCLUSIONS

1. The lateral geniculate nucleus is the primary thalamic relay through which retinal signals pass en route to cortex. This relay is gated and can be suppressed by activity among local inhibitory neurons that use γ -aminobutyric acid (GABA) as a neurotransmitter. In the cat, a major source of this GABAergic inhibition seems to arise from cells of the perigeniculate nucleus, which lies just dorsal to the A-laminae of the lateral geniculate nucleus. However, the morphological characteristics of perigeniculate cells, and particularly the projection patterns of their axons, have never been fully characterized. We thus examined the morphology of these cells: individually by intracellular injection of horseradish peroxidase (HRP) and en masse with the anterograde tracer *Phaseolus vulgaris* leucoagglutinin (PHAL).

2. We recorded from 12 perigeniculate cells that we impaled and successfully labeled with HRP. These cells exhibited response properties generally consistent with those described previously. They had long response latencies to stimulation of the optic chiasm and relatively large, often diffuse, receptive fields. The visually evoked responses of most of the cells were dominated by one eye. Compared with cells of the lateral geniculate nucleus, perigeniculate cells had large somata ($517 \pm 136 \mu\text{m}^2$ in cross-sectional area, mean \pm SD), which were fusiform or multipolar in shape, and dendritic arbors that extended a considerable distance ($1,095 \pm 167 \mu\text{m}$) parallel to the border between the perigeniculate and lateral geniculate nuclei. Terminal arbors of some dendrites were quite complex and beaded.

3. The axons of six perigeniculate cells were labeled sufficiently well to trace and reconstruct over a considerable distance. Each of these axons formed branches that descended to innervate the lateral geniculate nucleus, and this geniculate innervation was exclusively limited to the A-laminae. Terminal boutons within the A-laminae were nearly all en passant, which gave the axons a beaded appearance. Furthermore, branches of five of these six axons provided local innervation of the perigeniculate nucleus, generally within each labeled cell's own dendritic arbor. Three of the cells also exhibited an axon branch that extended medially and caudally away from the soma, but we were unable to trace these axon branches to their targets.

4. Within the lateral geniculate nucleus, each arbor of perigeniculate axons derived from two main components. One was a narrow, sparse medial component that innervated laminae A and A1. The other was a wider, more robust lateral component that innervated only one of these laminae, as determined by ocular dominance: if the parent perigeniculate cell was dominated by the contralateral eye, this more robust lateral component innervated only lamina A; if by the ipsilateral eye, its target seemed to be limited to lamina A1. Although we recorded from perigeniculate cells with a fairly balanced binocular input, none was successfully labeled with HRP. Finally, the extents of these medial and lateral components of the terminal arbors are comparable with those of retinogeniculate arbors, which suggests a surprising retinotopic precision in the projection of individual perigeniculate axons to the A-laminae.

5. We confirmed and extended our intracellular HRP results by bulk labeling the projection using PHAL placed into the perigeniculate nucleus. PHAL-labeled terminal arbors were beaded, and the geniculate A-laminae were the overwhelming target of these PHAL-labeled axons. Furthermore, although we could not fully reconstruct individual axons that were labeled with PHAL, we did reconstruct portions of axons that closely resembled the medial or lateral components observed with the intracellular HRP labeling.

6. However, these PHAL injections labeled a very small number of arbor segments not seen in the HRP-labeled material. These had boutons appended to the axon by short stalks (i.e., as occurs in type 1 axons) rather than being en passant, and some of these arbors extended beyond the A-laminae (e.g., into the C-laminae). We found that corticogeniculate axons labeled from PHAL injections placed into the striate cortex had type 1 morphology, confirming earlier evidence, and that these extended through the A- and C-laminae. These data are consistent with the interpretation that the sparse labeling of type 1 axons in the A-laminae and beyond seen after placement of PHAL into the perigeniculate nucleus is due to labeling of corticogeniculate axons passing through the perigeniculate nucleus.

7. We conclude that perigeniculate axons innervate the lateral geniculate nucleus exclusively or nearly so in the A-laminae. They produce boutons en passant in their retinotopically restricted terminal arbors. There is also a curious division of the axon arbor into medial and lateral components. Although this division seems to relate to ocular dominance, we see no other clear functional correlate for this morphological feature.

INTRODUCTION

The retinogeniculocortical pathway is the primary afferent pathway subserving visual perception in most mammals. As visual information is transmitted through the thalamic level of this pathway at the lateral geniculate nucleus, it is modulated by extrinsic inputs (Burke and Cole 1978; Sherman and Koch 1986; Singer 1977; Steriade and Llinás 1988). Inhibitory mechanisms play a crucial role in this modulation. In the cat, a major source of inhibition to the lateral geniculate nucleus is thought to arise from the perigeniculate nucleus.

The perigeniculate nucleus, which lies directly dorsal to the lateral geniculate nucleus, contains cells that stain positively for γ -aminobutyric acid (GABA) and glutamic acid decarboxylase (Fitzpatrick et al. 1984; Montero and Singer 1984; Rinvik et al. 1987). It is present in many carnivores (Sanderson 1974) and seems to represent a subregion of the thalamic reticular nucleus. Perigeniculate cells are innervated by collaterals of geniculocortical and corticogeniculate axons and by several pathways ascending from the brain stem reticular system (e.g., of Ahlsén and Lindström

1978; Ahlsén and Lo 1982; Friedlander et al. 1981; Robson 1984; Stanford et al. 1983; Uhlrich et al. 1988; Updyke 1975). These varied inputs make the perigeniculate nucleus an important station through which diverse areas of the brain may indirectly influence geniculate transmission of retinal inputs.

A number of functional roles have been proposed for the perigeniculate nucleus. Suggested inhibitory effects of perigeniculate cells on geniculate relay cells include recurrent inhibition, lateral inhibition, long-range inhibition, and binocular inhibition (e.g., Dubin and Cleland 1977; Eysel et al. 1986; Guido et al. 1989; So and Shapley 1981; Xue et al. 1988). In addition, the perigeniculate nucleus has been implicated in the transmission of eye-movement information (Singer 1977), in mediating selective attention (Crick 1984), and in the genesis of state-dependent oscillatory rhythms in the visual thalamus (Hu et al. 1989; Steriade and Deschênes 1984).

A clear picture of the projection from the perigeniculate nucleus to the lateral geniculate nucleus is essential for understanding how the transmission of visual information is modulated in the lateral geniculate nucleus, but such a description is lacking. Previous studies are limited to Golgi impregnations of perigeniculate cells from kittens (O'Leary 1940; Szentágothai 1972) and an unelaborated abstract (Ahlsén and Lindström 1978). We have thus analyzed this projection in adult cats at the single-cell level, using intracellular labeling with horseradish peroxidase (HRP), and en masse, using the anterograde tracer, *Phaseolus vulgaris* leucoagglutinin (PHAL). We have presented preliminary results of these experiments in abstract form (Cucchiari et al. 1985; Uhlrich et al. 1987).

METHODS

Because many of the procedures used in these experiments have been described in detail previously (Cucchiari and Uhlrich 1990; Friedlander et al. 1981; Humphrey et al. 1985; Uhlrich et al. 1988), they are only briefly outlined here.

Intracellular HRP experiments

GENERAL PREPARATION. Adult cats were initially anesthetized with 4% halothane in a 1:1 mixture of N₂O and O₂. We then performed a tracheotomy, cannulated the femoral vein, and placed the animal in a stereotaxic apparatus, where paralysis was induced with 5 mg of gallamine triethiodide. The cat was artificially respired thereafter and maintained on a continuous infusion of gallamine triethiodide (3.6 mg/h) and *d*-tubocurarine (0.7 mg/h) in 5% lactated Ringer solution (6 ml/h). Vital signs were continuously monitored. We maintained end-tidal CO₂ at 3.8–4.2% and rectal temperature at 37.5–38.0°C. During surgical procedures, animals were anesthetized with 1.5–2.5% halothane in a 7:3 mixture of N₂O and O₂. We infused all wound margins and pressure points with 2% lidocaine. After the surgery, we discontinued halothane and maintained the animal on the N₂O-O₂ mixture, and we added pentobarbital sodium (Nembutal) to the infusion solution, generally at a rate of 1 mg · kg⁻¹ · h⁻¹, although supplements were sometimes given to maintain synchronized activity in the animal's EEG.

We placed bipolar tungsten stimulating electrodes in the optic chiasm for electrical stimulation of retinogeniculate axons. These electrodes were separated by 3–4 mm. During placement, we recorded through these electrodes, and their final depth and rostrocaudal position (typically near A14 and 20–26 mm below the cor-

tical surface) were determined by maximizing a visually evoked potential. Finally, a craniotomy 1 cm in diameter was opened over the lateral geniculate nucleus, and a Plexiglas chamber that surrounded the opening was affixed to the skull with dental acrylic. We minimized brain pulsations during recording by filling the chamber with 3% agar in 0.9% saline and sealing it with dental wax.

ELECTROPHYSIOLOGICAL RECORDING AND INTRACELLULAR LABELING. We used a 3 M KCl electrode with an impedance (at 100 Hz) of 4–15 MΩ to locate the lateral geniculate nucleus. We then switched to a micropipette filled with a solution of 5% HRP (Sigma Type VI) in 0.2 M KCl and 0.05 M tris(hydroxymethyl)aminomethane (Tris) at a pH of 7.4. The pipette tips were beveled such that their impedance in the brain (at 100 Hz) was 55–110 MΩ. We used these HRP-filled electrodes to search for perigeniculate cells at a depth of 0–1,000 μm dorsal to the top of lamina A. These cells were identified by their characteristic responses that distinguished them from geniculate neurons: perigeniculate cells had large, ill-defined receptive fields and poor visual driving; excitatory receptive fields were usually binocular, although visual driving was generally dominated by one eye; and response latencies from optic chiasm stimulation were generally long and quite variable.

Once a perigeniculate cell was characterized, we impaled it by slowly advancing the electrode and passing short pulses of positive current (2–5 nA) or lightly tapping the stereotaxic apparatus. On a successful impalement, indicated by a 25- to 60-mV drop in the DC level and large (15–40 mV) monophasic action potentials, we reconfirmed that we were in the same cell that was recorded extracellularly. We then iontophoretically injected HRP into the cell by passing 4–15 nA positive current of variable frequency and duty cycle for 1–7 min. We never injected more than one perigeniculate cell in a penetration, and penetrations were widely spaced (i.e., >1.5 mm) so that we could easily match a histologically recovered cell to its physiological counterpart. We made use of Sanderson's (1971) retinotopic maps of the lateral geniculate and perigeniculate nuclei to achieve these matches.

HISTOLOGY. At least 2 h after the last injection, the animals were given an overdosage of pentobarbital sodium (100 mg) and perfused through the heart with 2 l of 1% paraformaldehyde and 2% glutaraldehyde in 0.1 M phosphate buffer (pH 7.4). We blocked the brain stereotaxically and refrigerated it overnight in either phosphate buffer or 30% sucrose in phosphate buffer. Serial coronal sections were cut at 50 or 100 μm, and the HRP was visualized by the use of diaminobenzidine with cobalt chloride intensification (Adams 1977).

Bulk labeling with PHAL

GENERAL PREPARATION AND INJECTIONS. We made small extracellular injections of PHAL into the perigeniculate nucleus of four cats. Each animal was anesthetized intravenously with pentobarbital sodium (initial dose of 15 mg/kg with 5- to 10-mg supplements as needed), placed in a stereotaxic apparatus, and surgically prepared under sterile procedures. We administered atropine sulphate (0.15–0.20 mg) to reduce salivation, infused all wound margins and pressure points with 2% lidocaine, and covered the corneas with contact lenses. Vital signs were continuously monitored.

We first located the lateral geniculate nucleus with a low-impedance electrode as described above. We then replaced this electrode with a double-barreled pipette in which each of the tips was broken back to a diameter of 2–5 μm. One barrel was filled with a PHAL solution (2.5% in 0.05 M sodium phosphate buffer, pH 7.4), and the other contained either 3 M KAc or 3 M KCl. The latter barrel provided excellent single- or multiunit recordings, and we were able to accurately resolve the border between geniculate lamina A and the perigeniculate nucleus. We then retracted

the electrode to a position 300–500 μm dorsal to the top of lamina A and iontophoretically injected the PHAL through the other barrel (5 μA positive current pulsed on and off at 0.07 Hz for 15 min).

In a fifth cat, similar procedures were used to make an injection of PHAL into area 17 of visual cortex. However, in this case, the electrode placement was made under visual and stereotaxic control.

HISTOLOGY. After a survival period of 1–3 days for the perigeniculate injections and 10 days for the cortical injection, each cat was deeply anesthetized, given heparin, and perfused through the heart with saline followed by two fixatives (1 l of 4% paraformaldehyde in 0.1 M acetate buffer at pH 6.5, then 1 l of 4% paraformaldehyde and 0.05% glutaraldehyde in 0.05 M borate buffer at pH 9.5). The brain was then removed and thalamic blocks were post-fixed overnight at 4°C in 20% sucrose dissolved in the second fixative. The next day, we cut sections at 40 μm and processed the tissue for PHAL, using the immunoperoxidase procedure described by Gerfen and Sawchenko (1984). The peroxidase was visualized with diaminobenzadine and cobalt chloride intensification (Adams 1977).

RESULTS

Intracellular labeling with HRP

RESPONSE PROPERTIES OF LABELED CELLS. We recorded from 12 perigeniculate cells that we impaled and successfully labeled with HRP. The electrophysiological properties

of these cells were similar to those described previously for such neurons (Ahlsén et al. 1982a,b; Dubin and Cleland 1981; So and Shapley 1981). The cells responded with long, probably disynaptic latencies to electrical stimulation of the optic chiasm. We were unable to drive three of the cells from chiasm stimulation, but for the others the latency values were 2.8 ± 0.6 ms (here and below, this refers to the mean \pm SD) with a range of 1.8–3.5 ms. These cells often displayed relatively large or ill-defined receptive fields. For four of the cells, we could not plot receptive field borders, although they were clearly driven via visual stimuli; and for a fifth cell, we were unable to elicit clear responses to visual stimuli. The receptive field diameters of the other seven cells were $3.8 \pm 3.6^\circ$ with a range of 0.5–10.5°. Only 4 of the 11 visually driven cells had clearly binocular receptive fields; of the remaining 7, 5 were dominated by the contralateral eye. Other response properties to visual stimuli varied among the cells: some responded in a transient fashion, others more tonically; some responded to the onset of light flashed in their visual fields, others to light offset, and still others to both light onset and offset. Except for ocular dominance (see below), there were no obvious morphological correlates to these physiological properties.

SOMA AND DENDRITIC MORPHOLOGY. The labeled perigeniculate neurons exhibit many morphological features that

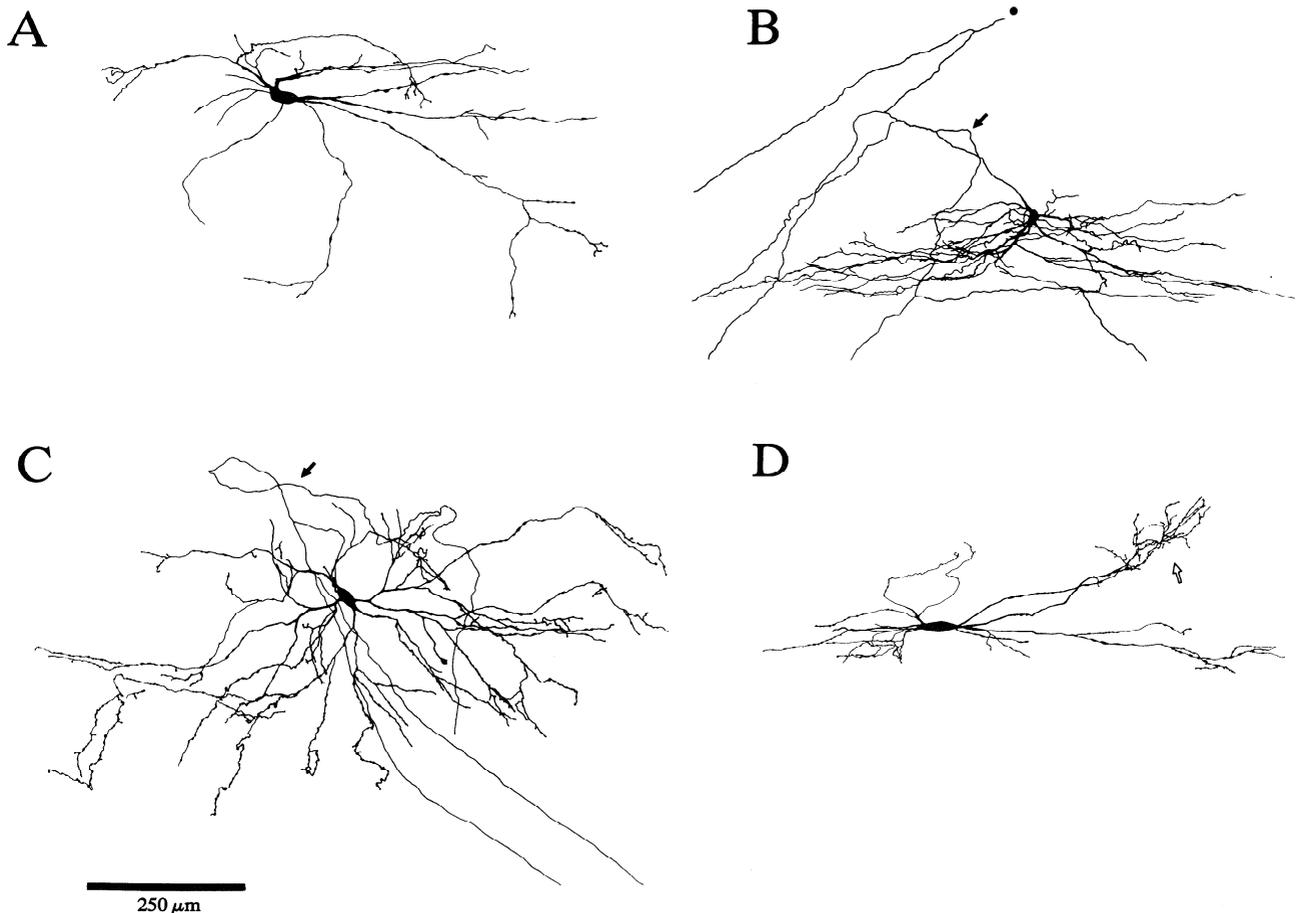


FIG. 1. Camera lucida reconstructions of 4 perigeniculate cells labeled intracellularly with HRP. Solid arrows in *B* and *C* indicate labeled axons, whereas the open arrow in *D* indicates an elaborate dendritic arbor. Filled circle in *B* indicates an axon branch that extended caudally and medially away from the cell to an unknown target. Scale bar applies to all 4 reconstructions.

have been previously described for this population (Ide 1982; O'Leary 1940). Figure 1 illustrates four representative examples. Somata are usually fusiform or multipolar, and are $517 \pm 136 \mu\text{m}^2$ in cross-sectional area, with a range of $284\text{--}765 \mu\text{m}^2$. These perigeniculate cells are thus relatively large compared with geniculate neurons labeled with HRP in this laboratory (Friedlander et al. 1981; Sherman and Friedlander 1988; Stanford et al. 1983): relay X cells are $281 \pm 100 \mu\text{m}^2$, relay Y cells are $439 \pm 147 \mu\text{m}^2$, relay W cells are $231 \pm 69 \mu\text{m}^2$, and interneurons are $129 \pm 36 \mu\text{m}^2$. These differences in soma size between perigeniculate and geniculate neurons are statistically significant ($P < 0.001$ on a Mann-Whitney U test).

Although dendritic morphology is quite varied, the dendritic arbors of all the labeled perigeniculate cells are most extensive in the direction parallel to the border between the perigeniculate and lateral geniculate nuclei. Along this axis, for the eight cells with particularly well-labeled dendritic arbors, these arbors extend $1,095 \pm 167 \mu\text{m}$, with a range of $873\text{--}1,397 \mu\text{m}$; this is much larger than that of thalamic relay cells, which seldom exhibit a dendritic arbor longer in any dimension than $400 \mu\text{m}$ (Friedlander et al. 1981; Stanford et al. 1983). Most of the dendrites, particularly distally, are beaded, and the distal dendrites often form elaborate arbors (e.g., Figure 1D). These elaborations may be the sites of considerable synaptic interaction, and, although we need to verify this at the electron microscopic level, they may be the source of presynaptic dendrites described previously in the cat's perigeniculate nucleus (Ide 1982) and thalamic reticular nucleus (Deschênes et al. 1985).

Figure 2 illustrates a labeled neuron that physiologically and morphologically resembled all the other perigeniculate cells in our sample. We have identified it as a perigeniculate neuron for four reasons. First, it responded sluggishly to visual stimuli, with a long latency to activation of the optic chiasm (2.6–2.9 ms). Second, although quite near the lateral geniculate nucleus, its soma was located in the perigeniculate nucleus. Third, except for a primary dendrite that descended into lamina A, the soma and dendritic morphology of the cell resembled that of all other perigeniculate cells. Fourth, the axon, although not completely labeled (and not illustrated in Fig. 2), had medial and lateral components like those of other perigeniculate cells (see below).

Dendrites of perigeniculate cells that extend into the lateral geniculate nucleus have been described before (Ide 1982; Szentágothai 1972), but never to the degree seen here. Because there is no direct evidence of retinal terminals located in the perigeniculate nucleus proper (Bowling and Michael 1984; Sur et al. 1987; Tamamaki et al. 1990), such dendrites afford one mechanism by which perigeniculate cells may be directly innervated by the retina (cf. Schmielau 1979). This cell, then, would be a strong candidate for innervation by the contralateral retina. However, the visual driving of this cell was dominated by the ipsilateral eye, because we found no evidence of responses to our handheld stimuli through the contralateral eye. Furthermore, although this cell's long latency from electrical activation of the optic chiasm is at the extreme long end of the range for monosynaptic X axon innervation (Hoffman et al. 1972; So and Shapley 1979), it is more consistent with disynaptic innervation via collaterals of geniculate relay cells.

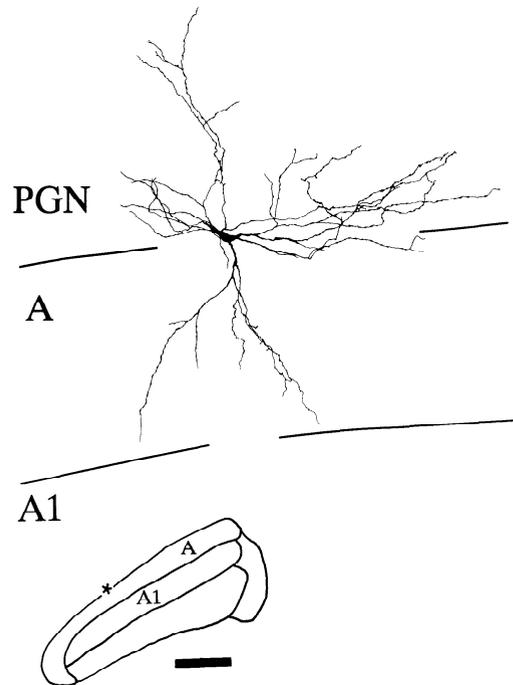


FIG. 2. Reconstruction of an HRP-labeled perigeniculate cell showing dendrites extending through most of lamina A. *Inset*: a lower-power outline of the lateral geniculate nucleus and the location of the perigeniculate soma (*). The cell was driven only by the ipsilateral eye; the cell responded to activation of the optic chiasm at a latency of 2.6 ms. The axon, which was not fully labeled and is not shown, innervated the A-laminae in a fashion similar to other perigeniculate axons, with separate medial and lateral components (see text for details). Scale bar represents $100 \mu\text{m}$ for the cell reconstruction and 1.0 mm for the *inset* of the lateral geniculate nucleus.

FINE DETAILS OF AXON ARBORS. One can never be sure that an axon is fully labeled. However, we have concluded that an axon process is fully labeled if it is very dark all the way to a terminal bouton. In some cases, the label in an axon or dendrite clearly fades out before the termination. We note below when this occurs. Our sample size of labeled axon arbors is small, because we found the axon to be the most difficult neuronal element to label adequately. However, the axons of six of the perigeniculate cells were labeled sufficiently to trace them into the lateral geniculate nucleus, where they innervate the A-laminae.

These six axons displayed consistent features as regards the fine details of their terminal arbors. In particular, within the lateral geniculate nucleus, most of the terminal boutons are located en passant (Fig. 3, B and C; see also below). We have shown in a separate study that these boutons are the sites of synaptic contact (Cucchiari et al. 1985, 1991). To quantify this, as is summarized in Table 1, we randomly sampled 981 boutons from two of the labeled axons and determined that 913 of these (93%) were located en passant, the remainder being found on short side branches. Furthermore, the results from the two axons were quite consistent, with one axon displaying 489 of 529 (94%) and the other 424 out of 462 (92%) boutons that were en passant ($P > 0.1$ on a χ^2 test). We thus confirm the suggestion by Robson (1984) that perigeniculate cells give rise to beaded axons in

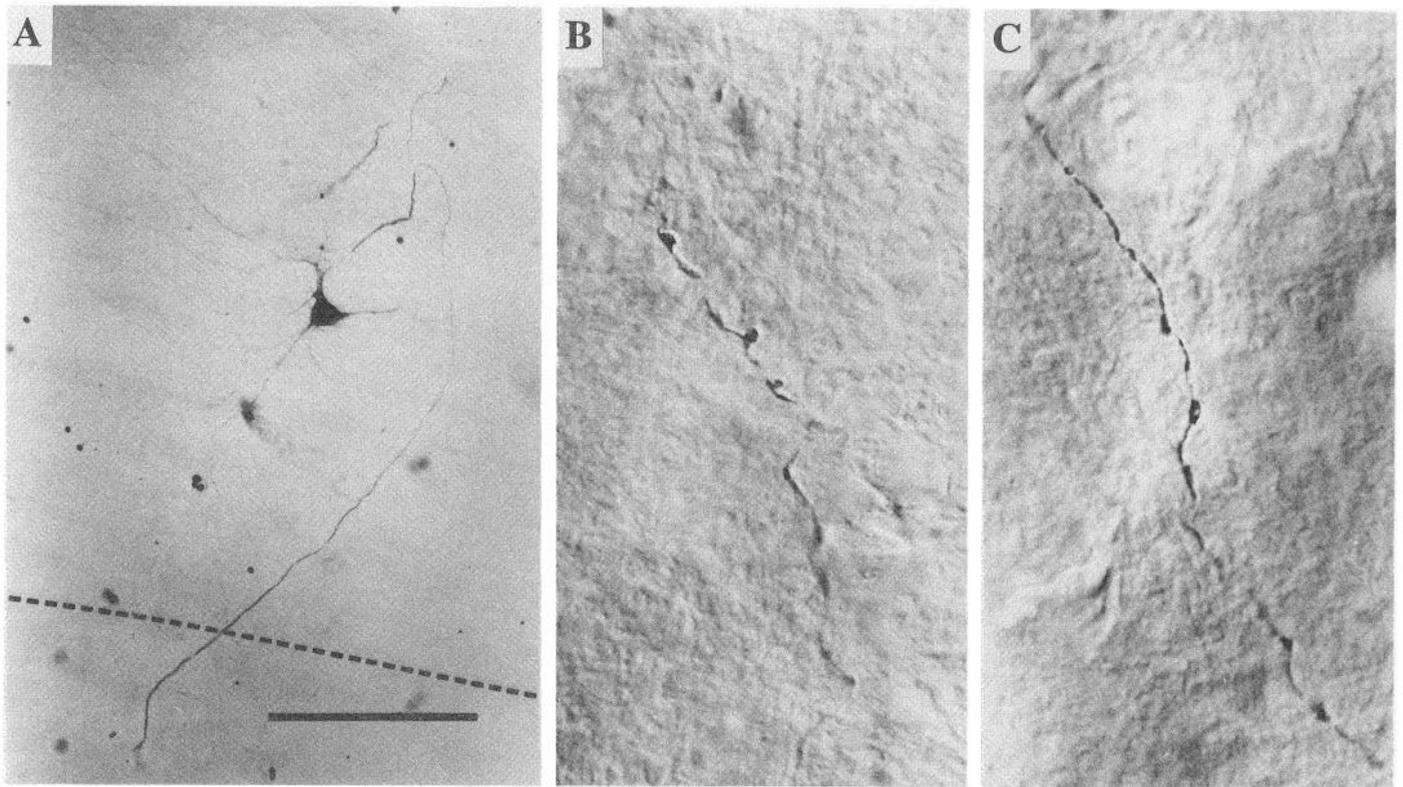


FIG. 3. Photomicrographs of a perigeniculate cell that was intracellularly labeled with HRP. Cell was well-driven only by the contralateral eye. Its on-center receptive field, measuring $2.5 \times 2.0^\circ$, was located 8.5° lateral to and 1.0° below the area centralis. Its response latency to chiasm activation was variable, ranging between 3.2 and 4.0 ms. *A*: low-power photomicrograph of the cell in the perigeniculate nucleus. Note that the axon originates from a large proximal dendrite and bifurcates within $100 \mu\text{m}$ of the soma. In this section, 1 branch descends into the lateral geniculate nucleus, lamina A of which is bounded dorsally by the dashed line. *B* and *C*: high-power photomicrographs of axon terminal processes in lamina A from the perigeniculate cell shown in *A*. Note the beaded appearance of the axons. Scale bar in *A* is $125 \mu\text{m}$ for *A* and $20 \mu\text{m}$ for *B* and *C*.

the lateral geniculate nucleus. Finally, from measurements of 234 boutons from four of the labeled axons, we ascertained that they are $1.0 \pm 0.3 \mu\text{m}$ in diameter.

PATTERNS OF AXON TERMINATIONS. The six labeled perigeniculate axons also display a fairly consistent pattern of terminal arborization. One example, which arises from a multipolar perigeniculate cell, is shown in Figs. 3 and 4. As with all of the axons in our sample, this one originates from a large dendrite (see Fig. 3*A*), branches within $200\text{--}300 \mu\text{m}$ of the soma, and descends into the lateral geniculate nucleus. Although the dendritic arbor is not fully labeled, a robust axonal projection is apparent (see Fig. 4). There is a sparse axon arbor within the cell dendritic arbor, but the primary axon target appears to be the lateral geniculate nucleus. Within the lateral geniculate nucleus, the axon arbor has medial and lateral components. The medial component is a relatively narrow and sparse arbor in laminae A and A1, whereas the lateral component is much more robust and is restricted to lamina A. Thus the medial component innervates both laminae, whereas the lateral component innervates only one. Interestingly, this cell's visual driving was strongly dominated by the contralateral eye, and, via the lateral component of its axon arbor, it projects primarily to lamina A, which is innervated by the contralateral retina.

Figure 5 shows another perigeniculate cell for which visual driving was dominated by the contralateral eye. The projection to the lateral geniculate nucleus from this cell also has medial and lateral components. The medial component is relatively narrow, and it innervates both laminae A and A1. The lateral component is much broader, contains many more boutons, and is restricted to lamina A. Thus far, we have recovered four perigeniculate cells with labeled

TABLE 1. *Bouton distribution*

	En Passant	Appended
PGN terminals labeled with HRP		
A-laminae	913	68
C-laminae	0	0
PGN terminals labeled with PHAL		
A-laminae	1,015	134
C-laminae	46	54
Cortical terminals labeled with PHAL		
A-laminae	157	255
C-laminae	139	220

PGN, perigeniculate nucleus; HRP, horseradish peroxidase; PHAL, *Phaseolus vulgaris* leucoagglutinin.

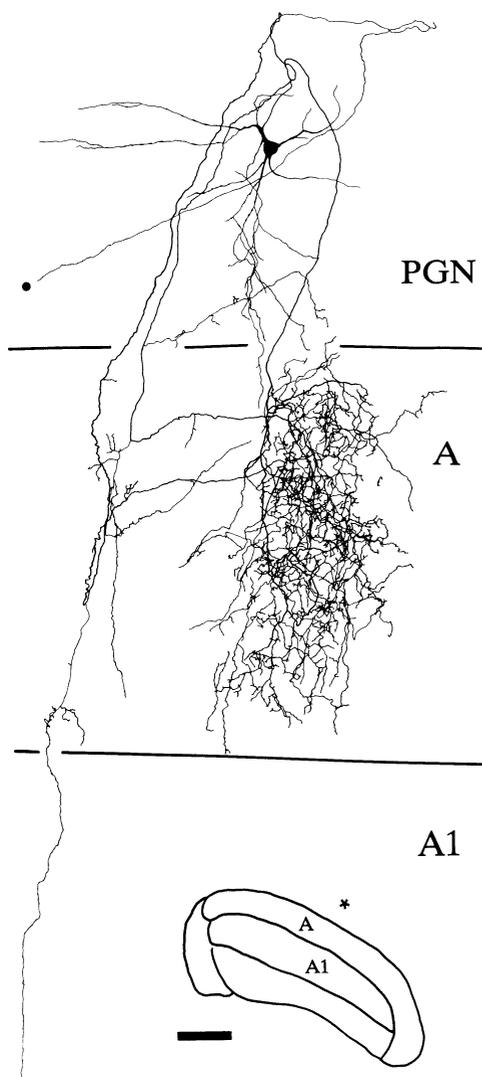


FIG. 4. Camera lucida reconstruction of the HRP-labeled perigeniculate cell illustrated in Fig. 3. Although the axon is well labeled, the dendritic arbor of this cell is not. Filled circle in the perigeniculate nucleus indicates an axon branch that extends medially and caudally away from the cell body toward an unknown destination. Scale bar is 100 μm for the cell reconstruction and 1.0 mm for the inset of the lateral geniculate nucleus. Other conventions are as in Fig. 2.

axons for which visual driving was strongly dominated by the contralateral eye. Each of these cells projects to the lateral geniculate nucleus with this same pattern: a robust lateral arbor that is restricted to lamina A and a narrow medial arbor that, when sufficiently well labeled, can be traced through both laminae A and A1.

We have recovered two perigeniculate cells with labeled axons in which visual driving was dominated by the ipsilateral eye. Figure 6 illustrates one example. Although the axon arbor is not fully labeled, medial and lateral components in the cell's projection to the lateral geniculate nucleus are apparent. The lateral branch appears to bypass lamina A to terminate in lamina A1, which is innervated by the ipsilateral eye. The full extent of the medial branch is not known, and it seems plausible that it continues into lamina A1. What is clear is that, like the medial component

from the contralaterally driven perigeniculate cells, the medial axon branch of this cell also innervates the geniculate lamina that corresponds to the nondominant eye (i.e., lamina A). The other perigeniculate cell dominated by the ipsilateral eye displayed a similar pattern of axon arbor termination.

Our results from the intracellular labeling suggest that perigeniculate cells project exclusively, or nearly so, to the A-laminae of the lateral geniculate nucleus, because we found no evidence of terminations in the C-laminae or medial interlaminar nucleus. We also noted that the projection to the A-laminae has a consistent pattern of medial and lateral components. The medial component is rather sparse and it innervates both laminae A and A1. The lateral component is more robust, and its projection is limited to the geniculate lamina that corresponds to the eye that dominated the visual driving of the perigeniculate cell. In addition to cells with obvious ocular dominance by one or the other eye, we have recorded from and labeled two perigeniculate cells that were driven fairly equally by each eye. Unfortunately, the axons of these cells were not sufficiently labeled that we could trace their arbors into the lateral geniculate nucleus. Thus the terminal arbor pattern for such cells remains unknown. Arbors of such binocularly driven perigeniculate cells would be particularly interesting with regard to the pattern we have seen that relates strongly to ocular dominance.

As noted above, five of the six perigeniculate axons, in addition to contributing a large arbor in the A-laminae of the lateral geniculate nucleus, sparsely innervate the perigeniculate nucleus within their dendritic arbors via small axon collaterals. We have also observed three perigeniculate cells with an axon branch that extends medially and caudally away from the soma (e.g., Figs. 1, 3, and 4). We were unable to trace these axon branches to their target in this material, but it is possible that some of these branches are destined for the medial interlaminar nucleus, the pulvinar, or some other target (see below).

Bulk labeling with PHAL

Although the technique of intracellular HRP labeling provides a powerful structure/function description of perigeniculate cells, it yields only a small number of cells for study. Furthermore, because we have had difficulty with this technique in completely labeling the perigeniculate cells and particularly their axons, the full extent of their axon arbors remains unresolved. We have thus complemented our intracellular HRP experiments with bulk labeling experiments using PHAL to characterize more fully the projection of perigeniculate cells. Figure 7 illustrates examples from such material.

We found many features in common between the HRP- and PHAL-labeled material. The diameters of 181 randomly selected boutons labeled with PHAL were $1.0 \pm 0.4 \mu\text{m}$, which is virtually the same as the diameter spectrum of HRP-labeled boutons ($P > 0.1$ on a Mann-Whitney U test; see above). Also, although we were unable to reconstruct complete axon arbors after PHAL-labeling, as is illustrated in Fig. 8, we did observe PHAL-labeled axon segments that

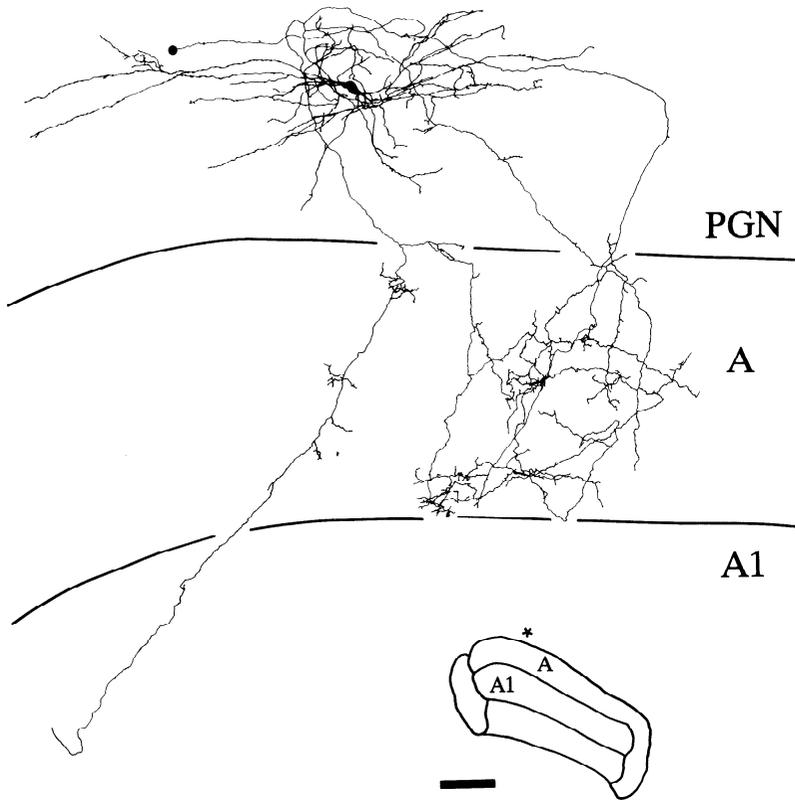


FIG. 5. Reconstruction of an HRP-labeled perigeniculate cell driven only by the contralateral eye. Both the dendritic arbor and the axon arbor in the lateral geniculate nucleus are well labeled. Cell had an ill-defined receptive field located roughly 2° lateral and 1° below the area centralis. Its variable response latency to chiasm activation was 2.7–3.3 ms. Scale bar is $100 \mu\text{m}$ for the cell reconstruction and 1.0 mm for the inset in the lateral geniculate nucleus. Other conventions are as in Fig. 2.

appeared to correspond to either the medial or lateral component of an axon arbor. Other similarities in projection pattern between HRP- and PHAL-labeled axons are described in the paragraphs below.

PERIGENICULATE TARGETS REVEALED WITH PHAL. Each injection of PHAL into the perigeniculate nucleus produced a dense column of axon terminal label nearly exclusively limited to the A-laminae of the lateral geniculate nucleus. However, we did detect extremely minute labeling beyond the A-laminae, including other geniculate regions, such as the medial interlaminar nucleus and C-laminae, as well as the lateral posterior-pulvinar complex. Figure 9 summarizes two representative projection patterns after PHAL injections in the perigeniculate nucleus. The location of each bouton in every fourth section throughout the entire projection column through the A- and C-laminae is shown. In both cases, the projection columns are fairly narrow through these laminae. In one case, 4,421 of the total of 4,652 boutons (95%) are located in the A-laminae, and in the other the proportion is 7,055 of 7,335 boutons (96%). These values between cases are consistent ($P > 0.1$ on a χ^2 test), and together they indicate that 96% of the label is confined to laminae A and A1. It should be noted that the label is as dark in the C-laminae as in the A-laminae, and thus the sparser labeling in the C-laminae does not appear to result from a waning of label further from the injection site. This nearly complete confinement of label to the A-laminae is in close agreement to the more limited observations from intracellular labeling of perigeniculate cells.

As is the case for HRP-labeled axons, most of the PHAL-labeled axons are fine, with many swellings en passant,

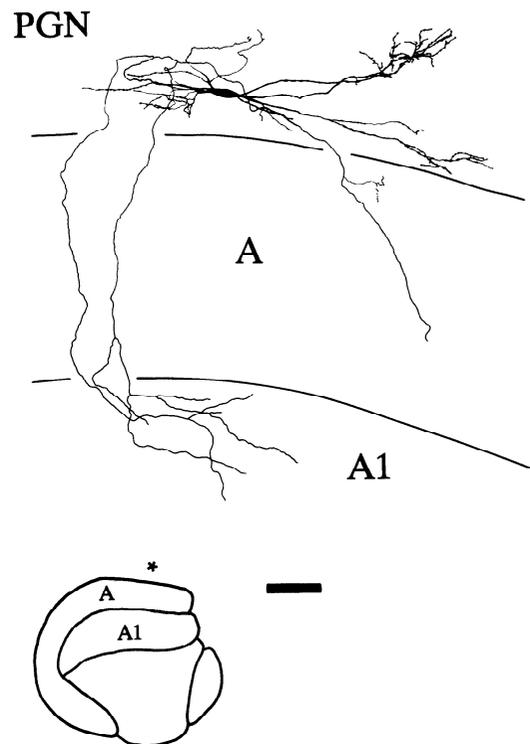


FIG. 6. Reconstruction of an HRP-labeled perigeniculate cell driven only by the ipsilateral eye. Cell had a large, diffuse, on-off receptive field measuring $11 \times 10^\circ$ and located near the area centralis. It responded to chiasm stimulation with a latency of 2.6 ms. The dendritic arbor is well labeled, but the axonal arbor is incompletely labeled in the A-laminae. Scale bar is $100 \mu\text{m}$ for the cell reconstruction and 1.0 mm for the inset of the lateral geniculate nucleus. Other conventions are as in Fig. 2.

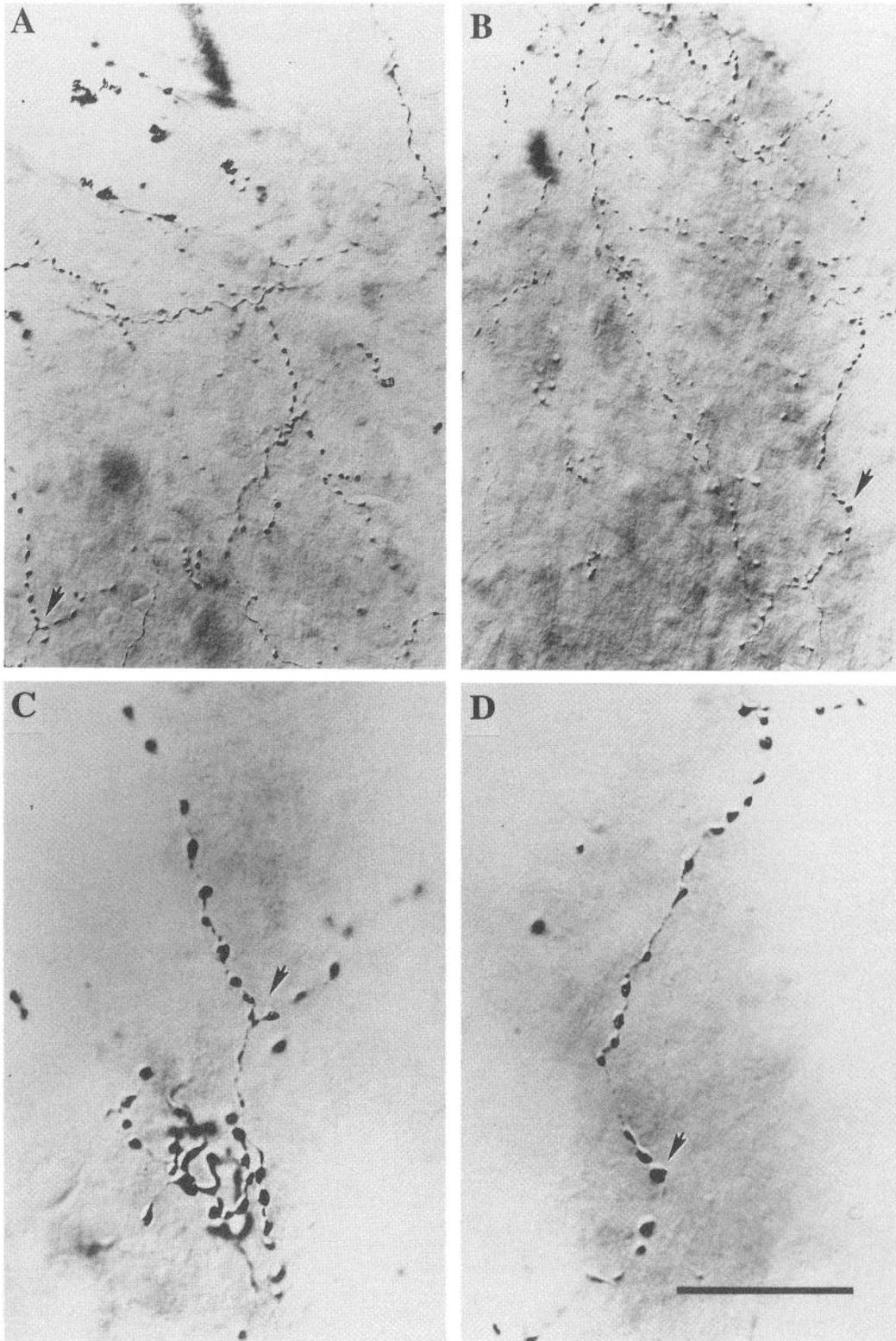


FIG. 7. Photomicrographs of axons in the A-laminae labeled from PHAL injected into the perigeniculate nucleus. Note the beaded appearance of the axons and the relative paucity of appended boutons. *A* and *B*: low-power photomicrographs. *C* and *D*: higher-power photomicrographs of selected processes from *A* and *B*. Arrows in *A* and *C* indicate the same processes, as do the arrows in *B* and *D*. Scale bar in *D* is 50 μm for *A* and *B* and 20 μm for *C* and *D*.

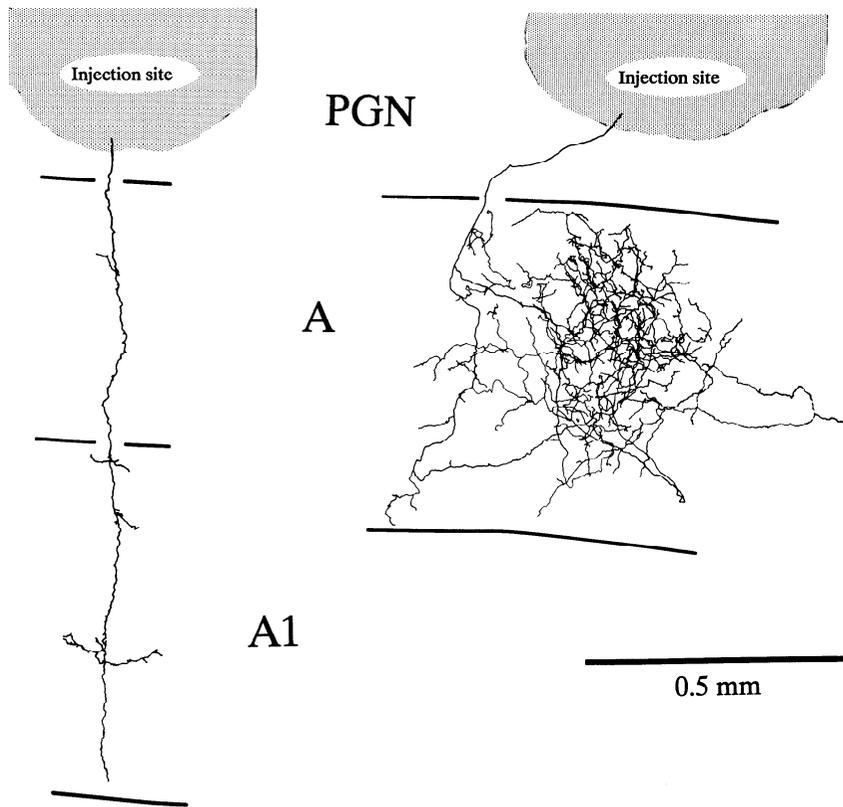


FIG. 8. Partially reconstructed axons that were labeled by PHAL injections in the perigeniculate nucleus. The process on the *left* innervates laminae A and A1 and resembles the medial axon component that we have described for perigeniculate cells labeled intracellularly with HRP. The process on the *right* is restricted to lamina A and resembles the lateral axon component of HRP-labeled perigeniculate cells in which visual driving was dominated by the contralateral eye. Scale bar applies to both reconstructions.

which gives the axons a beaded appearance (cf. Figs. 3 and 7). Within the A-laminae, we randomly sampled 575 boutons from one case and 574 from another; we found that 505 boutons in the former (88%) and 510 in the latter (89%) are en passant, the remainder found on short side branches (see Table 1). The two cases are thus comparable ($P > 0.1$ on a χ^2 test) and indicates an overall value of 88% of boutons being located en passant. While very similar to the value of 93% noted above for HRP labeled axons, it is significantly smaller ($P < 0.001$ on a χ^2 -test), suggesting a slightly different population of labeled axons between the two labeling techniques. Indeed, as is illustrated by Fig. 10, *A* and *B*,

we noted that rare PHAL-labeled axons in our material displayed boutons that were mostly on short side branches, and this renders the axons very similar to the type 1 axons described previously (Guillery 1971). Such type 1 axons were not seen after HRP labeling (see also below).

Interestingly, the few axons labeled with PHAL in the C-laminae had a different appearance, being mostly of type 1 morphology. We found, in the same two cases as used above, that 46 out of 100 randomly sampled boutons (46%) were en passant, the rest being on short side branches (see Table 1). This is a significantly lower percentage than seen in the A-laminae of the same cases ($P < 0.001$ on a χ^2 test).

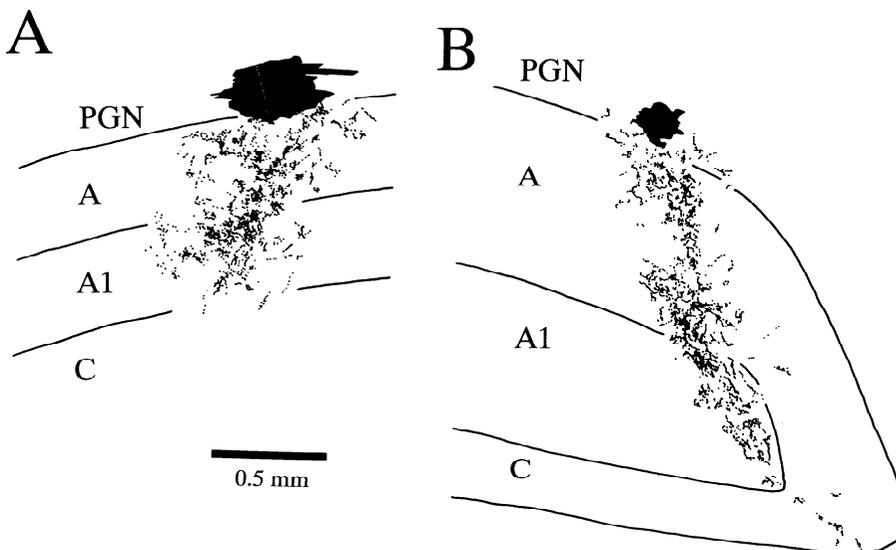


FIG. 9. Camera lucida reconstructions of 2 PHAL injections in the perigeniculate nucleus (solid filled areas). Dots indicate the location of every bouton on sections taken $160 \mu\text{m}$ apart through the entire projection column. Rostrocaudal extent of each of these projection columns is $\sim 600 \mu\text{m}$. Scale bar in *A* applies also to *B*.

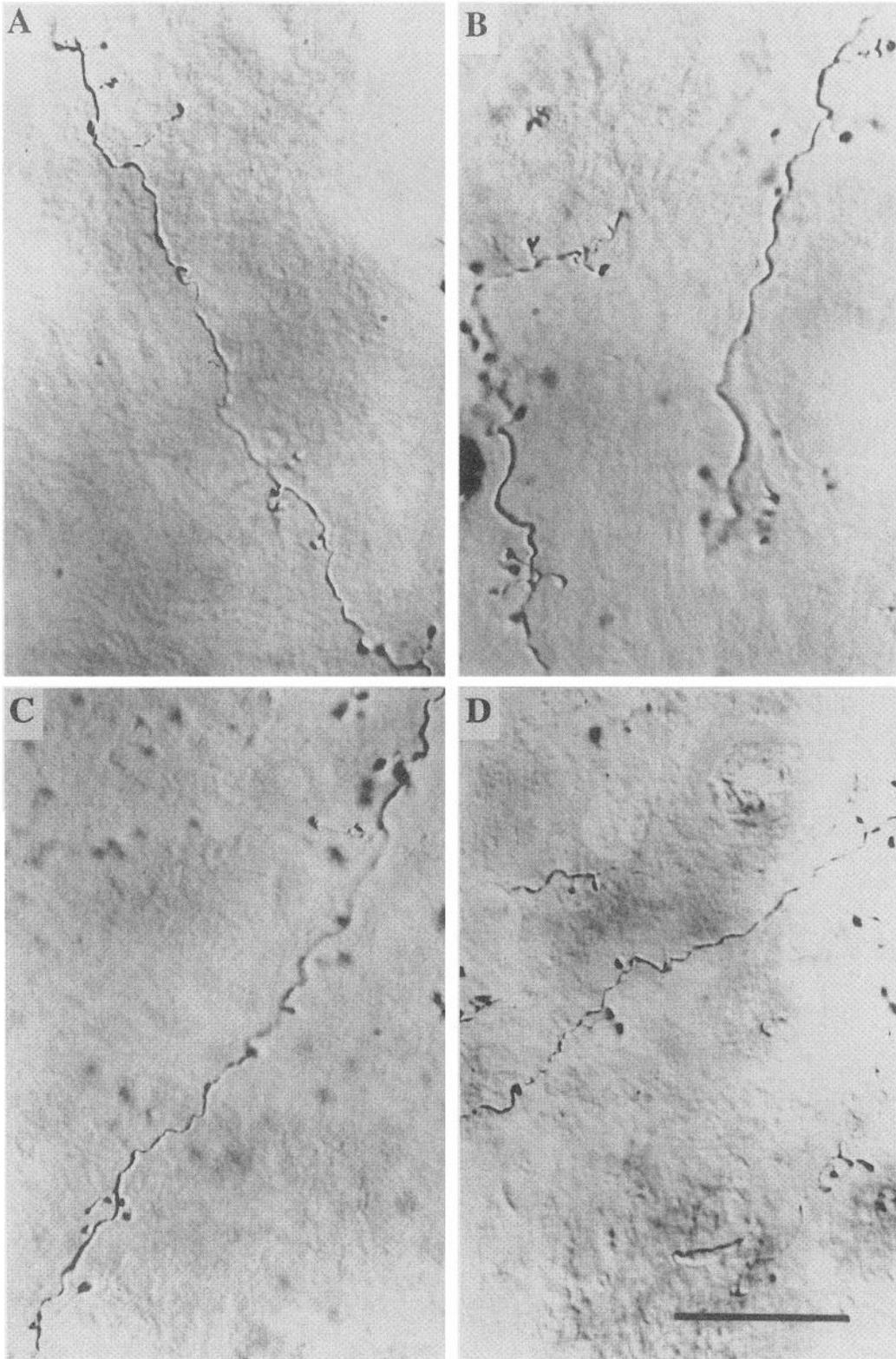


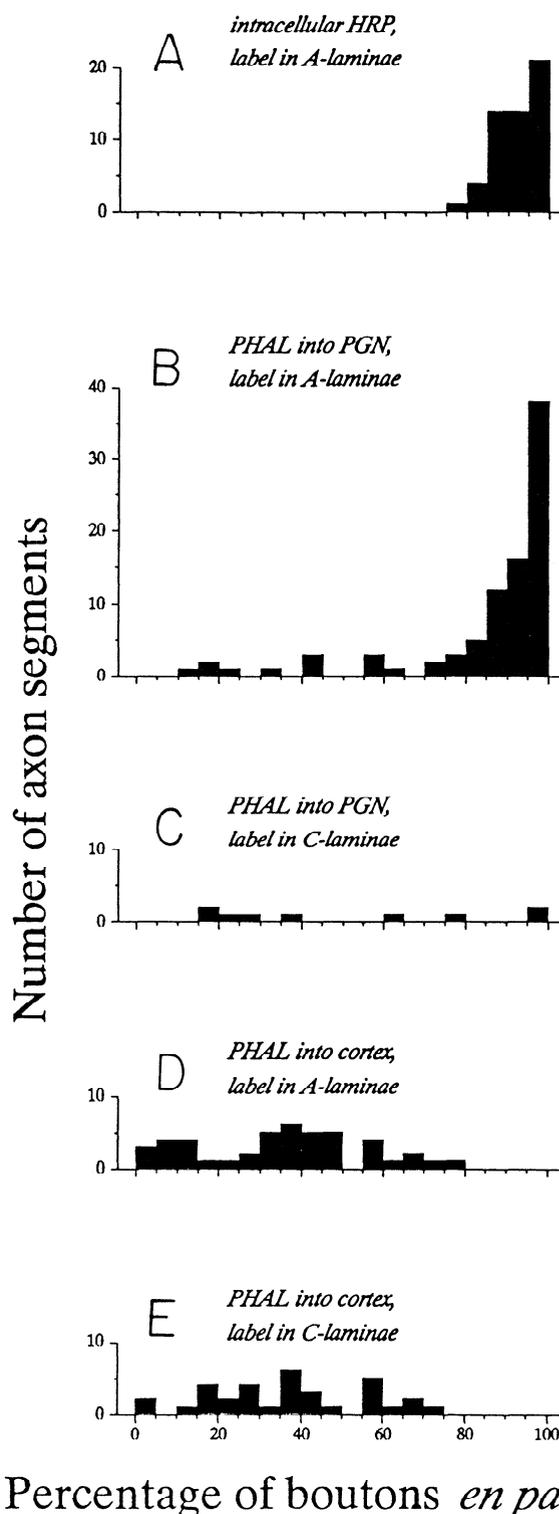
FIG. 10. Photomicrographs of axon terminations with type 1 morphology (Guillery 1971) that were labeled after PHAL injections in the perigeniculate nucleus (*A* and *B*) and area 17 of visual cortex (*C* and *D*). Note the fine appearance of the axons and the relatively high number of appended boutons. Scale bar in *D* is 20 μ m, and it applies also to *A-C*.

PHAL LABELING OF FIBERS OF PASSAGE. An important limitation of the PHAL technique is that it labels some fibers passing through the injection site (Cliffer and Giesler 1988; Cucchiaro and Uhlrich 1990; Gerfen and Sawchenko 1984). Thus, although the PHAL technique provides a more complete characterization of the projections of perigeniculate cells than does the limited sample of intracellularly labeled axons, this characterization may be contaminated by axons that do not originate from the perigeniculate nucleus. This may have contributed to the very slight differences seen between HRP and PHAL labeling, as if PHAL labels an extra axon population, displaying type 1 morphology, that innervates structures beyond the A-laminae (i.e., the C-laminae, the medial interlaminar nucleus, and the lateral posterior-pulvinar complex).

An important issue is whether this extra labeling represents an additional, relatively rare type of perigeniculate cell not sampled by our intracellular HRP technique or a small population of labeled fibers of passage. The largest population of axons passing through the perigeniculate nucleus en route to the lateral geniculate nucleus is the corticogeniculate pathway (Guillery 1967), and there is some basis for believing that type 1 axons in the lateral geniculate nucleus are corticogeniculate axons (Guillery 1971).

To test this possibility further, we analyzed one case in which PHAL was injected into the striate cortex to label axon terminals in the lateral geniculate nucleus. Figure 10, *C* and *D*, shows axons labeled from cortex within the A-laminae. It is clear that these are quite different from the predominantly beaded axons seen from the perigeniculate nucleus, because most boutons from these cortical axons are on short side branches, rather than being en passant. We quantified these impressions after cortical labeling as above, as is summarized in Table 1. In the A-laminae, we found that 157 of 412 randomly sampled boutons (38%) are en passant. Analogous values from the C-laminae, 139 of 359 (39%), are similar ($P > 0.1$ on a χ^2 test). Interestingly, compared with the material in which PHAL was injected into the perigeniculate nucleus, these C-laminae values are not significantly different ($P > 0.1$ on a χ^2 test), but, for the A-laminae, the percentage of en passant boutons is much higher after perigeniculate than cortical injections ($P < 0.001$ on a χ^2 test).

We probed this further in an analysis of labeling in the A- and C-laminae, which is summarized by Fig. 11. For this analysis, we randomly selected from each case sprigs of terminal arbor chosen so that each sprig contained ≥ 10 boutons. For each sprig, we determined the percentage of boutons that were on short terminal side branches (i.e., as in type 1 axons), the remaining boutons being en passant (i.e., as in beaded axons). After both intracellular HRP labeling of perigeniculate cells and injection of PHAL into the perigeniculate nucleus, nearly all of the sprigs labeled in the A-laminae were beaded (Fig. 11, *A* and *B*), and these distributions do not differ significantly ($P > 0.1$ on a χ^2 test). Conversely, the labeled sprigs in both the A- and C-laminae after placement of PHAL into cortex appeared to be of type 1 axons (Fig. 11, *D* and *E*), and these distributions do not differ significantly either ($P > 0.1$ on a χ^2 test). This labeling after cortical injection also appeared very similar to the la-



Percentage of boutons *en passant*

FIG. 11. Frequency histograms showing the percentage of boutons en passant in different conditions. All boutons not en passant in this analysis were appended to the terminal axon by a short side branch. These percentages were computed for randomly selected sprigs of terminal arbors containing ≥ 10 boutons (see text for details). *A*: boutons in A-laminae labeled after intracellular injection of perigeniculate cells with HRP. *B*: boutons in A-laminae labeled after PHAL injection into perigeniculate nucleus. *C*: boutons in C-laminae labeled after PHAL injection into perigeniculate nucleus. *D*: boutons in A-laminae labeled after PHAL injection into visual cortex. *E*: boutons in C-laminae labeled after PHAL injection into visual cortex.

being seen in the C-laminae after PHAL placement into the perigeniculate nucleus (Fig. 11C; $P > 0.1$ on a χ^2 test). Finally, Fig. 11, B and D, shows that the PHAL labeling after injections of cortex differs markedly from that after perigeniculate injections ($P < 0.001$ on a χ^2 test).

These data help to confirm earlier evidence (Guillery 1971) that corticogeniculate axons are mostly of type 1 morphology and that they innervate both the A- and C-laminae. What we have shown is that the corticogeniculate labeling in the C-laminae is quite similar to what is seen after PHAL injections into the perigeniculate nucleus, but we did not see it after intracellular labeling of perigeniculate cells with HRP. Although we cannot rule out the possibility that this extra labeling seen in the PHAL material reflects a relatively rare morphological type of perigeniculate axon not captured by our small HRP sample, the data are consistent with the notion that most or all of these type 1 axons in our PHAL material are corticogeniculate fibers labeled as they pass through the perigeniculate nucleus. We also conclude from Fig. 11, A and B, that perigeniculate axon terminals are beaded, but this does not imply that all beaded axons in the lateral geniculate nucleus derive from the perigeniculate nucleus. That is, some of the beaded axons depicted in Fig. 11B that were labeled after the PHAL injections into the perigeniculate nucleus may represent fibers of passage, perhaps from other regions of the thalamic reticular nucleus (Cucchiari et al. 1990).

DISCUSSION

We used two complementary tracing techniques to examine the axonal projections of perigeniculate cells, and both methods yielded similar results. We have confirmed prior suggestions that the perigeniculate nucleus projects densely to the lateral geniculate nucleus and that the terminal axons have a beaded morphology. Within the lateral geniculate nucleus, perigeniculate cells project exclusively or nearly so to the A-laminae. Furthermore, the projection to the A-laminae exhibits two distinct components for each axon: a narrow and sparse medial component innervates both laminae, whereas a wider and more robust lateral component innervates only the lamina innervated by the same eye that dominates the receptive field of the perigeniculate cell in question. Finally, we found that the beaded morphology of perigeniculate axons is distinct from the type 1 morphology of corticogeniculate axons.

Connections with the lateral geniculate nucleus

Although prior work has indicated that perigeniculate cells innervate the A-laminae of the lateral geniculate nucleus, this has been limited to an abstract of HRP-filled cells (Ahlsén and Lindström 1978) and Golgi work in kittens (O'Leary 1940; Szentágothai 1972); and the full extent of this projection in the adult was undetermined. Our data not only confirm the earlier conclusion but also reveal that little if any further projection exists from the perigeniculate nucleus to other regions of the lateral geniculate nucleus. This indicates reciprocity for the A-laminae, because nearly all of the geniculocortical axons arising from these laminae branch to innervate the perigeniculate nucleus en route to cortex (Friedlander et al. 1981). However, such reciprocity

is not evident for other regions, because a minority of projection axons emanating from the medial interlaminar nucleus innervate the perigeniculate nucleus (Raczkowski and Sherman 1985), and most W and Y cells in the C-laminae do so (Stanford et al. 1983). This further suggests that neurons of the medial interlaminar nucleus and C-laminae can influence the A-laminae through the perigeniculate nucleus, but the A-laminae cannot similarly influence these other geniculate regions via an analogous perigeniculate route.

Projection patterns of individual perigeniculate axons

Three features of the perigeniculate axon arbors within the lateral geniculate nucleus merit further discussion: they have distinct medial and lateral components; they exhibit a strong correlation between ocular dominance and the laminar location of their arbors; and their extent is surprisingly small across the retinotopic map within the lateral geniculate nucleus.

MEDIAL AND LATERAL COMPONENTS. The observation that each of the perigeniculate axons displayed a distinct medial and lateral component to its terminations in the lateral geniculate nucleus was completely unexpected. There is no clear functional correlate known for this morphological feature either in the visual responses of perigeniculate cells or in the inhibitory responses seen in geniculate neurons. This requires further study. We can speculate that such a medio-lateral asymmetry might be considered a morphological substrate for direction selectivity. That is, a visual stimulus moving horizontally toward the fixation point from the periphery (i.e., lateral to medial as seen from the maps of Sanderson 1971) would evoke a different pattern of inhibition in the A-laminae from that of a stimulus moving the opposite direction. Although there is recent evidence of direction selectivity among cells of the A-laminae (Thompson et al. 1990), both the morphological and physiological details are too sketchy at present to speculate further.

OCULAR DOMINANCE. We also observed a strong correlation between ocular dominance of the perigeniculate cell and its projection to the A-laminae. Perigeniculate cells with receptive fields dominated by the contralateral eye project predominantly to lamina A, which is innervated by that eye, whereas cells dominated by the ipsilateral eye mostly innervate lamina A1, which is innervated by the ipsilateral eye. Because the perigeniculate input is presumed to provide inhibitory modulation of geniculate neuronal response properties (Sherman and Koch 1986; see INTRODUCTION), this pattern of connectivity suggests that this inhibition is mostly related to one eye. Compared with their retinal inputs, receptive fields of neurons in the A-laminae express enhanced inhibition for the dominant eye (Cleland and Lee 1985; Hubel and Wiesel 1961), or monocular inhibition, as well as an inhibitory receptive field for the nondominant eye (Sanderson et al. 1971), or binocular inhibition. Thus the connectivity patterns of our limited sample of HRP-filled perigeniculate cells suggest that they mainly contribute to monocular, rather than binocular, inhibition.

However, there are three reasons to qualify this conclu-

sion. First, although dominated by one eye, these perigeniculate cells do respond, albeit weakly, to the other eye. Second, the medial component of the axonal projection, although relatively sparse, does equally innervate the two A-laminae. These factors do provide a limited substrate for binocular inhibition. Third, we did observe perigeniculate cells with fairly balanced binocularity, and although we recovered none of their axons for morphological analysis, these cells clearly provide a substrate for binocular inhibition of geniculate cells.

RETINOTOPIC EXTENT. Retinotopically, the perigeniculate projection to the lateral geniculate nucleus is surprisingly restricted. The entire lateral component of a perigeniculate axon arbor in the lateral geniculate nucleus is often <500 μm across, and the medial component is even less extensive, often <100 μm across. These dimensions are comparable with those of individual retinogeniculate arbors (Bowling and Michael 1984; Sur et al. 1987; Tamamaki et al. 1990). This is surprising, because perigeniculate circuitry is generally thought to provide diffuse inhibitory inputs to the lateral geniculate nucleus, in keeping with the relatively large receptive fields and dendritic arbors of perigeniculate cells; and our expectation was that their axonal arbors should reflect this by extending much more broadly across the retinotopic map than do retinogeniculate arbors, which clearly provide a retinotopically precise input to geniculate cells.

The relatively precise morphological pattern of perigeniculate axon arbors seems inconsistent with the large receptive fields of perigeniculate cells. Perhaps these diffuse fields are an artifact of anesthesia and/or paralysis (see also above) such that these receptive fields in an awake, alert cat might be more restricted. The alternative conclusion is that perigeniculate circuitry enables the convergence of a relatively large portion of the visual field onto a more restricted region of the lateral geniculate nucleus, such as may occur with long-range inhibition (Eysel et al. 1984). Regardless of the specific functional correlate, this morphological feature of perigeniculate axon arbors suggests that this input provides a much more retinotopically restricted pattern of inhibition than is generally thought to be the case.

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REFERENCES

- ADAMS, J. C. Technical considerations on the use of horseradish peroxidase as a neuronal marker. *Neuroscience* 2: 141–145, 1977.
- AHLSÉN, G. AND LINDSTRÖM, S. Axonal branching of functionally identified neurones in the lateral geniculate body of the cat. *Neurosci. Lett. Suppl.* 1: 156, 1978.
- AHLSÉN, G., LINDSTRÖM, S., AND LO, F.-S. Functional distinction of perigeniculate and thalamic reticular neurons in the cat. *Exp. Brain Res.* 46: 118–126, 1982a.
- AHLSÉN, G., LINDSTRÖM, S., AND LO, F.-S. Excitation of perigeniculate neurones from X and Y principal cells in the lateral geniculate nucleus of the cat. *Acta. Physiol. Scand* 118: 445–448, 1982b.
- AHLSÉN, G. AND LO, F.-S. Projection of brainstem neurones to the perigeniculate nucleus and the lateral geniculate nucleus in the cat. *Brain Res.* 238: 433–438, 1982.
- BOWLING, D. B. AND MICHAEL, C. R. Terminal patterns of single physiologically characterized optic tract fibers in the cat's lateral geniculate nucleus. *J. Neurosci.* 4: 198–216, 1984.
- BURKE, W. AND COLE, A. M. Extracortical influences on the lateral geniculate nucleus. *Rev. Physiol. Biochem. Pharmacol.* 80: 105–166, 1978.
- CLELAND, B. G. AND LEE, B. B. A comparison of visual responses of cat lateral geniculate nucleus neurones with those of ganglion cells afferent to them. *J. Physiol. Lond* 369: 249–268, 1985.
- CLIFFER, K. D. AND GIESLER, G. J. PHA-L can be transported anterogradely through fibers of passage. *Brain Res.* 458: 185–191, 1988.
- CRICK, F. Function of the thalamic reticular complex: the searchlight hypothesis. *Proc. Natl. Acad. Sci. USA* 81: 4586–4590, 1984.
- CUCCHIARO, J. B. AND UHLRICH, D. J. *Phaseolus vulgaris* leucoagglutinin (PHA-L): a neuroanatomical tracer for electron microscopic analysis of synaptic circuitry in the cat's dorsal lateral geniculate nucleus. *J. Electron Microsc. Tech.* 15: 352–368, 1990.
- CUCCHIARO, J. B., UHLRICH, D. J., HAMOS, J. E., AND SHERMAN, S. M. Perigeniculate input to the cat's lateral geniculate nucleus: a light and electron microscopic study of single, HRP filled cells. *Soc. Neurosci. Abstr.* 11: 231, 1985.
- CUCCHIARO, J. B., UHLRICH, D. J., AND SHERMAN, S. M. A projection from the thalamic reticular nucleus to the dorsal lateral geniculate nucleus in the cat: a comparison with the perigeniculate projection. *Soc. Neurosci. Abstr.* 16: 159, 1990.
- CUCCHIARO, J. B., UHLRICH, D. J., AND SHERMAN, S. M. An electron-microscopic analysis of synaptic input from the perigeniculate nucleus to the A-laminae of the lateral geniculate nucleus in cats. *J. Comp. Neurol.* In press.
- DESCHÊNES, M., MADARIAGA-DOMICH, A., AND STERIADE, M. Dendrodendritic synapses in the cat reticularis thalami nucleus: a structural basis for thalamic spindle synchronization. *Brain Res.* 334: 165–168, 1985.
- DUBIN, M. W. AND CLELAND, B. G. Organization of visual inputs to interneurons of lateral geniculate nucleus of the cat. *J. Neurophysiol.* 40: 410–427, 1981.
- EYSEL, U. TH., PAPE, H.-C., AND VAN SCHAYCK, R. Excitatory and differential disinhibitory actions of acetylcholine in the lateral geniculate nucleus of the cat. *J. Physiol. Lond.* 370: 233–254, 1984.
- FITZPATRICK, D., PENNY, G. R., AND SCHMECHEL, D. E. Glutamic acid decarboxylase-immunoreactive neurons and terminals in the lateral geniculate nucleus of the cat. *J. Neurosci.* 4: 1809–1829, 1984.
- FRIEDLANDER, M. J., LIN, C.-S., STANFORD, L. R., AND SHERMAN, S. M. Morphology of functionally identified neurons in the lateral geniculate nucleus of the cat. *J. Neurophysiol.* 46: 80–129, 1981.
- GERFEN, C. AND SAWCHENKO, P. E. An anterograde neuroanatomical tracing method that shows the detailed morphology of neurons, their axons and terminals: immunohistochemical localization of an axonally transported plant lectin, *Phaseolus vulgaris* leucoagglutinin (PHA-L). *Brain Res.* 290: 219–238, 1984.
- GUIDO, W., TUMOSA, N., AND SPEAR, P. D. Binocular interactions in the cat's dorsal lateral geniculate nucleus. I. Spatial-frequency analysis of responses of X, Y, and W cells to nondominant-eye stimulation. *J. Neurophysiol.* 62: 526–543, 1989.
- GUILLERY, R. W. Patterns of fiber degeneration in the dorsal lateral geniculate nucleus of the cat following lesions in the visual cortex. *J. Comp. Neurol.* 130: 197–222, 1967.
- GUILLERY, R. W. Patterns of synaptic interconnections in the dorsal lateral geniculate nucleus of cat and monkey: a brief review. *Vision Res. Suppl.* 3: 211–227, 1971.
- HOFFMANN, K.-P., STONE, J., AND SHERMAN, S. M. Relay of receptive-field properties in dorsal lateral geniculate nucleus of the cat. *J. Neurophysiol.* 35: 518–531, 1972.
- HU, B., STERIADE, M., AND DESCHÊNES, M. The effects of brainstem peribrachial stimulation on perigeniculate neurons: the blockage of spindle waves. *Neuroscience* 31: 1–12, 1989.
- HUBEL, D. H. AND WIESEL, T. N. Integrative action in the cat's lateral geniculate body. *J. Physiol. Lond.* 155: 385–398, 1961.

- HUMPHREY, A. L., SUR, M., UHLRICH, D. J., AND SHERMAN, S. M. Projection patterns of individual X- and Y-cell axons from the lateral geniculate nucleus to cortical area 17 in the cat. *J. Comp. Neurol.* 233: 159-189, 1985.
- IDE, L. S. The fine structure of the perigeniculate nucleus in the cat. *J. Comp. Neurol.* 210: 317-334, 1982.
- MONTERO, V. M. AND SINGER, W. Ultrastructure and synaptic relations of neural elements containing glutamic acid decarboxylase (GAD) in the perigeniculate nucleus of the cat: a light and electron microscopic immunocytochemical study. *Exp. Brain Res.* 56: 115-125, 1984.
- O'LEARY, J. L. A structural analysis of the lateral geniculate nucleus in the cat. *J. Comp. Neurol.* 73: 405-430, 1940.
- RACZKOWSKI, D. AND SHERMAN, S. M. Morphology and physiology of single neurons in the medial interlaminar nucleus of the cat's lateral geniculate nucleus. *J. Neurosci.* 5: 2702-2718, 1985.
- RINVIK, E., OTTERSEN, O. P., AND STORM-MATHISEN, J. Gamma-aminobutyrate-like immunoreactivity in the thalamus of the cat. *Neuroscience* 21: 781-805, 1987.
- ROBSON, J. A. Reconstructions of corticogeniculate axons in the cat. *J. Comp. Neurol.* 225: 193-200, 1984.
- SANDERSON, K. J. The projection of the visual field to the lateral geniculate and medial interlaminar nuclei in the cat. *J. Comp. Neurol.* 143: 101-118, 1971.
- SANDERSON, K. J. Lamination of the dorsal lateral geniculate nucleus in carnivores of the weasel (Mustelidae), raccoon (Procyonidae) and fox (Canidae) families. *J. Comp. Neurol.* 153: 239-266, 1974.
- SANDERSON, K. J., BISHOP, P. O., AND DARIAN-SMITH, I. The properties of the binocular receptive fields of lateral geniculate neurons. *Exp. Brain Res.* 13: 178-207, 1971.
- SCHMIELAU, F. Integration of visual and nonvisual information in nucleus reticularis thalami of the cat. In: *Developmental Neurobiology of Vision*, edited by R. D. Freeman. New York: Plenum, 1979, p. 205-266.
- SHERMAN, S. M. AND FRIEDLANDER, M. J. Identification of X versus Y properties for interneurons in the A-laminae of the cat's lateral geniculate nucleus. *Exp. Brain Res.* 73: 384-392, 1988.
- SHERMAN, S. M. AND KOCH, C. The control of retinogeniculate transmission in the mammalian lateral geniculate nucleus. *Exp. Brain Res.* 63: 1-20, 1986.
- SINGER, W. Control of thalamic transmission by corticofugal and ascending reticular pathways in the visual system. *Physiol. Rev.* 57: 386-420, 1977.
- SO, Y. T. AND SHAPLEY, R. Spatial properties of X and Y cells in the lateral geniculate nucleus of the cat and conduction velocities of their inputs. *Exp. Brain Res.* 36: 533-550, 1979.
- SO, Y. T. AND SHAPLEY, R. Spatial tuning of cells in and around lateral geniculate nucleus of the cat: X and Y relay cells and perigeniculate interneurons. *J. Neurophysiol.* 45: 107-120, 1981.
- STANFORD, L. R., FRIEDLANDER, M. J., AND SHERMAN, S. M. Morphological and physiological properties of geniculate W-cells of the cat: a comparison with X- and Y-cells. *J. Neurophysiol.* 50: 582-608, 1983.
- STERIADE, M. AND DESCHÊNES, M. The thalamus as a neuronal oscillator. *Brain Res.* 8: 1-63, 1984.
- STERIADE, M. AND LLINÁS, R. R. The functional states of the thalamus and the associated neuronal interplay. *Physiol. Rev.* 68: 649-742, 1988.
- SUR, M., ESGUERRA, M., GARRAGHTY, P. E., KRITZER, M. F., AND SHERMAN, S. M. Morphology of physiologically identified retinogeniculate X- and Y-axons in the cat. *J. Neurophysiol.* 58: 1-32, 1987.
- SZENTÁGOTHAJ, J. Lateral geniculate body structure and eye movement. *Bibl. Ophthalmol.* 82: 178-188, 1972.
- TAMAMAKI, N., UHLRICH, D. J., AND SHERMAN, S. M. Morphology of physiologically identified retinal axons in the cat's thalamus and mid-brain as revealed by intra-axonal injection of biocytin. *Soc. Neurosci. Abstr.* 16: 711, 1990.
- THOMPSON, K. G., ZHOU, Y., AULT, S. J., AND LEVENTHAL, A. G. Direction sensitive relay cells in the LGNd of cats and monkeys. *Soc. Neurosci. Abstr.* 16: 158, 1990.
- UHLRICH, D. J., CUCCHIARO, J. B., AND SHERMAN, S. M. Innervation of the cat's lateral geniculate nucleus by individual cells of the perigeniculate nucleus. *Soc. Neurosci. Abstr.* 13: 1435, 1987.
- UHLRICH, D. J., CUCCHIARO, J. B., AND SHERMAN, S. M. The projection of individual axons from the brainstem parabrachial region to the dorsal lateral geniculate nucleus in the cat. *J. Neurosci.* 8: 4565-4575, 1988.
- UPDYKE, B. V. Patterns of projection of cortical areas 17, 18 and 19 onto the dorsal lateral geniculate nucleus in the cat. *J. Comp. Neurol.* 163: 377-396, 1975.
- XUE, J.-T., CARNEY, T., RAMOA, A. S., AND FREEMAN, R. D. Binocular interaction in the perigeniculate nucleus of the cat. *Exp. Brain Res.* 69: 497-508, 1988.