12 Killackey HP, Belford GR. *The formation of afferent patterns in the somatosensory cortex of the neonatal rat.* J Comp Neurol 1979;183:285–304.
13 Land PW, Simons DJ. *Cytochrome oxidase staining in the rat SmI barrel cortex.* J Comp Neurol 1985;238:225–235.
14 Landry P, Dykes RW. *Identification of two populations of corticothalamic neurons in cat primary somatosensory cortex.* Exp Brain Res 1985;60:289–298.
15 Melzer P, Van der Loos H, Dörfl J, Welker E, Robert P, Emery D, Berrini JC. *A magnetic device to stimulate selected whiskers of freely moving or restrained small rodents: its application in a deoxyglucose study.* Brain Res 1985;348:229–240.
16 Nussbaumer J-C, Van der Loos H. *An electrophysiological and anatomical study of projections to the mouse cortical barrelfield and its surroundings.* J Neurophysiol 1985;53:686–698.
17 Price TR, Webster KE. *The corticothalamic projection from the primary somatosensory cortex of the rat.* Brain Res 1972;44:636–640.
18 Scheibel ME, Scheibel AB. *Patterns of organization in specific and nonspecific thalamic fields.* In: Purpura DP, Yahr MB, eds. *The thalamus.* New York: Raven Press, 1966;13–46.
19 Simons DJ. *Response properties of vibrissa units in rat SI somatosensory neocortex.* J Neurophysiol 1978;41:798–820.
20 Spacek J, Lieberman AR. *Ultrastructure and three-dimensional organization of synaptic glomeruli in rat somatosensory thalamus.* J Anat (Lond)1974;117:478–516.
21 Van der Loos H. *Barreloids in mouse somatosensory thalamus.* Neurosci Lett 1976;2:1–6.
22 Waite PME. *Somatotopic organization of vibrissal responses in the ventrobasal complex of the rat thalamus.* J Physiol (Lond) 1973;228:527–540.
23 Welker C. *Microelectrode delineation of fine grain somatotopic organization of SmI cerebral neocortex in albino rat.* Brain Res 1971;26:259–275.
24 Welt C, Steindler DA. *Somatosensory cortical barrels and thalamic barreloids in reeler mutant mice.* Neurosci 1977;2:755–766.
25 White EL, DeAmicis RA. *Afferent and efferent projections of the region in mouse SmI cortex which contains the posteromedial barrel subfield.* J Comp Neurol 1977;175:455–482.
26 Winer JA, Larue DT. *Patterns of reciprocity in auditory thalamocortical and corticothalamic connections: study with horseradish peroxidase and autoradiographic methods in the rat medial geniculate body.* J Comp Neurol 1987;257:282–315.
27 Wise SP, Jones EG. *Cells of origin and terminal distribution of descending projections of the rat somatic sensory cortex.* J Comp Neurol 1977;175:129–158.
28 Woolsey TA, Van der Loos H. *The structural organization of layer IV in the somatosensory region (SI) of mouse cerebral cortex. The description of a cortical field composed of discrete cytoarchitectonic units.* Brain Res 1970;17:205–242.
29 Wouterlood FG, Groenewegen HJ. *Neuroanatomical tracing by use of Phaseolus vulgaris-leucoagglutinin (PHA-L): electron microscopy of PHA-L filled neuronal somata, dendrites, axons and axon terminals.* Brain Res 1985;326:188–191.
30 Yamakado M. *Postnatal development of barreloid neuropile in the ventrobasal complex of mouse thalamus: a histochemical study for cytochrome oxidase.* Brain Nerve 1985;37:497–506.
31 Yuan B, Morrow TJ, Casey KL. *Corticofugal influences of SI cortex on ventrobasal thalamic neurons in the awake rat.* J Neurosci 1986;6:3611–3617.

Cellular thalamic mechanisms
M. Bentivoglio and R. Spreafico, editors

Functional organization of the cat's lateral geniculate nucleus

S. MURRAY SHERMAN

Department of Neurobiology, State University of New York, Stony Brook, NY, USA

Introduction

The lateral geniculate nucleus, which is the major way station between retina and visual cortex, has been the most intensely studied of all thalamic nuclei. Despite this, we are only now beginning to appreciate the role of geniculate circuitry in visual processing. This role seems to be the control or gating of retino-geniculo-cortical transmission [8,53,55]. That is, the neuronal circuitry of the lateral geniculate nucleus determines the extent to which retinal signals are relayed to visual cortex. However, unlike other regions of the visual system (e.g., retina or visual cortex), there is no significant elaboration of receptive field properties by this circuitry. In part, this is because each geniculate neuron receives its main retinal input from one axon, or from very few axons with the same receptive field properties, and thus geniculate neurons display virtually the same receptive field properties as do their retinal afferents [6,7,37,38,51].

Ever since the pioneering work of Hubel and Wiesel [26–28], the receptive field approach has proven to be a remarkably productive tool in understanding the functional organization of most visual structures. For instance, the observation in retina and visual cortex that receptive fields become more complex and selective as each synaptic hierarchy is ascended [13,17,27,28] has led to the obvious conclusion that synaptic circuitry in these areas is used for the elaboration of receptive field properties, which in turn is an important neuronal concomitant for the analysis of visual scenes. However, the role of geniculate circuitry was missed or misunderstood for many years, partly because this receptive field approach failed to provide useful insights into the functioning of this circuitry. That is, because no significant elaboration of receptive field properties occurs between retina and the lateral geniculate nucleus, many regarded this nucleus as a simple, machine-like relay of retinal information to cortex, an otherwise uninteresting neuronal structure. This has led to a paradox for nearly 20 years, because while most physiological studies (i.e., re-

ceptive field studies) uncovered no functionally important input to geniculate relay cells other than retinal axons*, morphological studies demonstrated that only 10–20% of the synapses formed onto these neurons actually derived from retina [19,20,63]. The functional significance of the vast majority of synaptic input to geniculate relay cells, which is clearly non-retinal in origin, can be attributed to a gating function [53]. Thus, the view that the lateral geniculate nucleus is a simple relay can be appreciated in retrospect as a failure of limitation of the receptive field approach.

While it now seems clear that non-retinal inputs to geniculate relay cells play an important and unique role in vision, surprisingly little is known about the morphological organization of these inputs at the single-cell level. Accordingly, our laboratory for the past few years has carried out a series of studies of this organization, with particular emphasis on the A-laminae of the cat's lateral geniculate nucleus (see Acknowledgements). The following is a brief summary of these studies.

General overview of the cat's lateral geniculate nucleus

Geniculate lamination

The cat's lateral geniculate nucleus is a mostly laminated structure that can be divided into four general areas: the A-laminae; the C-laminae; laminae 1, 2 and 3 of the medial interlaminar nucleus; and the geniculate wing. Fig. 1, which is a schematic view of a coronal section through the lateral geniculate nucleus, shows these laminae and their relationship to ocular input. The contralateral nasal retina innervates laminae A, C, C2, 1 and the geniculate wing; the ipsilateral temporal retina innervates laminae A1, C1, 2 and the geniculate wing; and the contralateral temporal retina innervates lamina 3 [21,25,48]. Thus, the geniculate wing is binocularly innervated, lamina C3 receives no direct retinal input, and each of the other laminae is retinally innervated by only one or the other eye. Laminae A and A1, which form a reasonably matched pair, have been the most intensely studied geniculate regions and are thus the best understood. They form the focus of the remainder of this paper.

X and Y pathways

Retinal ganglion cells of cats (as well as of all other mammals studied to date) are divided into several distinct morphological and physiological classes, which form points of departure for the parallel retinofugal pathways. The retino-geniculo-cortical X and Y pathways are the best understood of these parallel neuronal streams (see Fig. 2A). These pathways are fairly independent of one another, and they seem to remain segregated through at least the first few stages of circuitry in the visual

*To be fair, there has long been physiological evidence that some non-retinal inputs can alter geniculate relay cell responsiveness (reviewed in Refs. 8, 53 and 55), but until recently this has not had much impact on mainstream notions of geniculate circuitry and its functional significance.

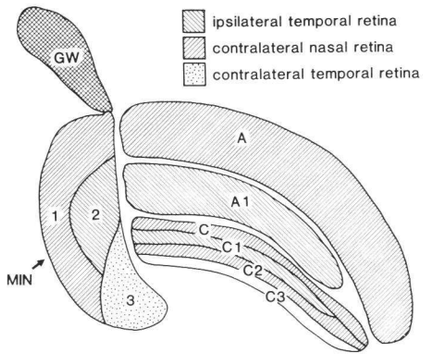

FIG. 1 Schematic drawing of coronal section through the cat's lateral geniculate nucleus. The various laminae and their ocular inputs are also shown. Letters and numbers depict the various laminae (see text for details), MIN indicates the medial interlaminar nucleus, and GW indicates the geniculate wing. Note that, with two exceptions, each lamina receives input exclusively from one retina; the exceptions are the geniculate wing, which receives binocular input, and lamina C3, which receives no direct retinal input.

cortex. It is likely that each of these pathways is involved in an analysis of somewhat different aspects of the visual scene and thus play different functional roles in visual perception (for hypotheses, see Refs. 29, 34, 52, 57 and 58). Although the X and Y pathways have been extensively studied at retinal and cortical levels, only their relationship to geniculate circuitry is of concern here. More complete ac-

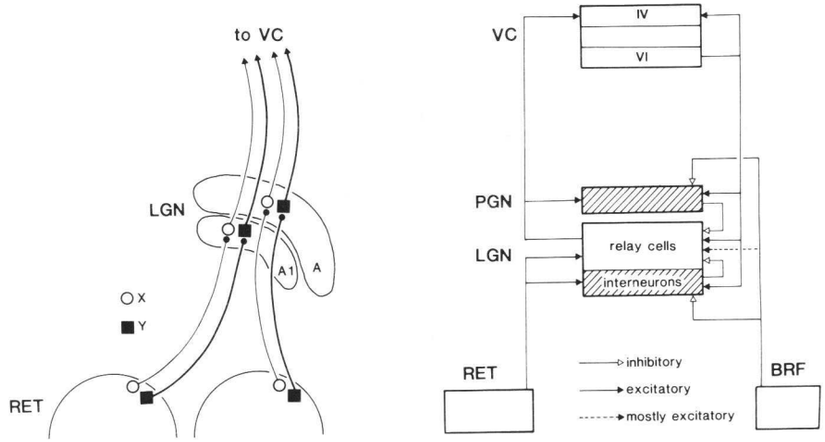

FIG. 2 Summary of neuronal circuitry involving the A-laminae of the cat's lateral geniculate nucleus (LGN). BRF, brainstem reticular formation; PGN, perigeniculate nucleus; RET, retina; VC, visual cortex. (Redrawn from Ref. 53.)

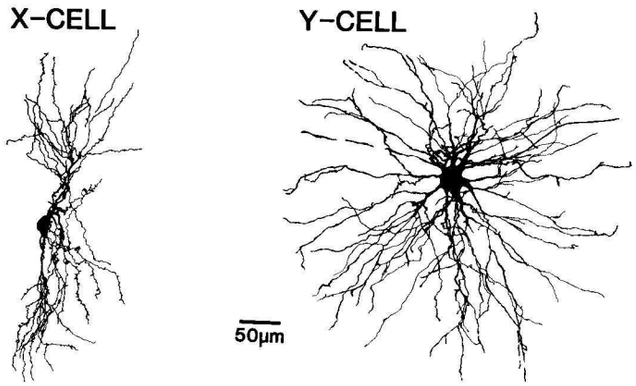

FIG. 3 Drawing of typical examples of relay X and Y cells in the geniculate A-laminae; coronal view. These cells were physiologically identified and labeled intracellularly with HRP. The laminar borders (not shown) run horizontally, so the predominant orientation of the X cell's dendritic arbor is perpendicular to these borders, and it extends roughly as far in the rostrocaudal direction as it does mediolaterally. The Y cell arbor is approximately spherical in shape. (Taken from Ref. 16.)

counts of these parallel visual pathways in cats and other mammals can be found in several recent reviews [34,51,52,54,57,58].

Relay X and Y cells are present in roughly equal numbers within the geniculate A-laminae (see Ref. 52). These cell classes differ in a large number of response properties which largely reflect similar differences among retinogeniculate X and Y inputs (see above). Compared to Y cells, X cells have smaller receptive fields, respond better to higher spatial frequencies but more poorly to lower ones, exhibit more linear spatial and temporal summation in response to visual stimuli, and have slower conducting axons. Many other differences in response properties have also been documented for X and Y cells.

The intracellular HRP-labeling technique has permitted a detailed study of relay X and Y cell morphology [3,15]. Both cell types have extensively branched dendritic arbors that contain 90–95% of the neuron's surface membrane, and the dendritic branching closely obeys the '3/2' power law. This means that, when the diameters of each daughter branch are raised to the 3/2 power and these products are then summed, this sum equals the diameter of the parent branch raised to the 3/2 power. Among other things, this suggests efficient electrotonic conductance of voltage signals across the dendritic branch points and allows the dendritic arbors to be collapsed to simple cylinders for easier modeling of neuronal functioning (see Ref. 44).

However, as is summarized by Fig. 3, in other ways these geniculate cell classes differ morphologically. The X cells have smaller somata with thinner more sinuous dendrites than do Y cells. The dendritic arbors of X cells tend to be oriented perpendicular to the geniculate laminae, while those of Y cells tend to be more radially

arranged. Finally, proximal dendrites of most geniculate X cells have numerous dendritic appendages, the significance of which is considered below, while Y cell dendrites tend to be quite smooth and appendage-free.

Retinal and non-retinal inputs to relay cells

Fig. 2 places retinal inputs to these geniculate relay cells in the context of non-retinal inputs (for a review of these non-retinal inputs, see Ref. 53). Local inhibitory neurons, which employ gamma-aminobutyric acid (GABA) as their neurotransmitter, form a major source of synaptic input to these cells. Two groups of such GABAergic neurons are known. One includes the local interneurons scattered among the relay cells of the A-laminae; 20–30% of all geniculate neurons in these laminae are GABAergic interneurons, the remaining being relay cells. The other group includes the cells of the perigeniculate nucleus**, which is a scattered cell group lying just dorsal to lamina A. All perigeniculate cells are GABAergic. The other two known sources of input to geniculate relay cells derive from beyond the thalamus. The first of these emanates from cells located in layer VI of visual cortex, particularly from areas 17, 18 and 19. The second arises from cell groups in the brainstem reticular formation. These issue mostly from cholinergic cells of the parabrachial region***, but also include noradrenergic neurons from this region as well as from the locus coeruleus and a few serotonergic cells of the raphe nucleus [12,53].

In addition to innervating relay cells directly, the pathways from visual cortex and the brainstem reticular formation also innervate both interneurons and perigeniculate cells. Furthermore, interneurons are directly innervated by retinal axons, and perigeniculate cells are innervated by collaterals of relay cell axons as they pass through *en route* to visual cortex. The cortical axons seem to excite all of their postsynaptic targets [1,60], but the brainstem inputs are more complex and may have

**It has been argued that the perigeniculate nucleus is a portion of the thalamic reticular nucleus, implying that it derives from ventral thalamus (e.g., Ref. 32). For instance, the connections described for various subdivisions of the reticular nucleus of the thalamus involving the brainstem reticular formation plus thalamocortical and corticothalamic axon collaterals are rather similar to those described for the perigeniculate nucleus; also, like perigeniculate cells, all cells of the reticular nucleus of the thalamus are GABAergic. Nonetheless, other embryological and phylogenetic origins for the perigeniculate nucleus cannot yet be ruled out.

***There has been some confusion about the terminology of these brainstem regions that innervate the thalamus. In the rat, where cytoarchitectonic boundaries are clearer than in the cat, the cholinergic and noradrenergic cell groups that project to thalamus are quite distinct. The former has been called the pedunculopontine tegmental nucleus, while the latter is called the locus coeruleus [22,49]. In the cat, the homologous nuclei seem almost to overlap somewhat, so that a mostly cholinergic cell group reminiscent of the rat's pedunculopontine tegmental nucleus contains some noradrenergic neurons. It is this zone that we call the parabrachial region. The presence of noradrenergic cells in this region raises some question about its precise homology with the pedunculopontine tegmental nucleus of the rat, and we thus wish to avoid using this loaded term. The use of the term 'parabrachial region' is in accord with recent terminology as applied to the cat's brainstem (e.g., Refs. 12 and 50). We avoid the term 'parabrachial nucleus', because this is used for a cell group that in many species is involved in the gustatory pathway [43]. 'Parabrachial region' refers merely to the territory adjacent to the brachium conjunctivum.

rather unconventional postsynaptic effects [39,53]. Generally, the brainstem inputs serve to increase the excitability of relay cells and decrease that of perigeniculate cells and interneurons.

Relay cells thus receive a lavish supply of non-retinal afferents. These afferents control the general excitability of geniculate relay cells, particularly with regard to the responsiveness of these relay cells to activation of their retinal inputs. These afferents can affect the relay cells via direct connections or indirectly via control of perigeniculate cells and interneurons. It is of obvious importance to work out any differences that occur in the pattern of these non-retinal inputs to relay X and Y cells. Preliminary data described below indicate that such differences indeed exist.

Intrinsic physiological properties of relay cells

From the above paragraphs, it should be abundantly clear that geniculate relay cells must integrate synaptic input from many sources, retinal as well as non-retinal. It is thus important to assess the intrinsic integrative properties of these neurons. These properties can be roughly divided into the passive-cable properties and active conductances that the postsynaptic relay cells exhibit.

Passive-cable properties. As noted above, the dendritic branching pattern of the relay cells makes them suitable subjects for modeling as passive cables. There exist subtle but important and consistent differences in the passive-cable properties between geniculate relay X and Y cells [4]. However, for the purposes of this discussion, the most important of these properties is the electrotonic length of the entire cell, including its dendritic arbor. Although values of electrotonic length are slightly but significantly higher for X than for Y cells, for both classes they are only about 1. This means that even the most distally located synaptic inputs will have its postsynaptic potential attenuated by only about 1/3 *en route* to the soma and axon hillock. In other words, all of the synaptic input located on the dendrites of these geniculate relay cells, even those most distally located, are in a position to influence the relay cells quite effectively.

Active conductances. It is also worth noting that these geniculate relay cells are endowed with a rich variety of membrane conductances, some of which are voltage-dependent and others of which can be controlled by neurotransmitter action (see Ref. 53). These conductances can also greatly affect retino-geniculo-cortical transmission and can be controlled by the non-retinal afferents. For instance, a low-threshold calcium conductance, which is voltage-dependent, can move the neuron from a tonic state of fairly faithful relay of retinal signals to a state of bursty firing that no longer reflects the incoming retinal signals. This conductance has been identified in relay neurons of virtually all mammalian thalamic nuclei, including the cat's lateral geniculate nucleus, with both *in vivo* and *in vitro* techniques (Refs. 30, 31, 39 and 56 and our own unpublished observations). The membrane voltage levels that regulate this conductance can be effectively controlled via activity of inputs from the parabrachial region and/or the locus coeruleus (cf. Ref. 39). Therefore, non-retinal inputs may regulate relay cell excitability not only by classical excitation and

inhibition, but also by unconventional control of these membrane conductances (for a further discussion of this, see Ref. 53).

Detailed morphology of geniculate circuitry

An understanding of the morphological basis for the above-mentioned gating of retino-geniculo-cortical transmission requires a detailed knowledge of the types of synaptic terminal found in the geniculate neuropil, their origins and the pattern of synaptic contacts they form onto the relay cells. We have sought to address this problem via detailed reconstructions with the electron microscope. Our general approach has been to reconstruct physiologically identified cells or axons that were previously labeled with an electron-opaque marker, most typically by the intracellular iontophoresis of horseradish peroxidase (HRP) during electrophysiological recording [15,23,24,63].

Such iontophoresis completely labels the physiologically identified cell without extending into the extracellular matrix to label other neural elements indirectly.

This labeling serves both to relate the neuronal morphology to a specific and defined physiological entity as well as to ease the burden of reconstruction. The latter benefit follows because it is possible to be confident that labeled processes, even when they are dendritic segments separated by 500 μm or more, belong to the same neuron, a conclusion that would otherwise be possible only after tedious and unrealistic serial reconstruction. Unfortunately, even with intracellularly labeled material, the time needed to perform adequate reconstructions generally precludes the possibility of sampling large numbers of labeled neurons. Our conclusions from these studies thus require qualification regarding their generality, although the consistency of results across neurons of the same type is an encouraging sign in this regard.

Types of synaptic profile

Fortunately for those interested in the detailed morphology of the cat's lateral geniculate nucleus, roughly 95% of all synaptic terminal profiles encountered in the A-laminae can be identified as belonging to one of four morphological classes designated as *RLP*, *RSD*, *F1* and *F2* terminals [19,20]. RLP terminals (*R*ound vesicles, *L*arge profile, *P*ale mitochondria) and RSD terminals (*R*ound vesicles, *S*mall profile, *D*ark mitochondria) form asymmetric synaptic contacts. In contrast, F1 and F2 terminals (*F*lattened vesicles) form symmetric synaptic contacts. F1 and F2 terminals can be distinguished because F1 terminals derive from axons, whereas F2 terminals emanate from dendrites; also, F1 terminals are exclusively *presynaptic* profiles, whereas F2 terminals are both *presynaptic* and *postsynaptic* (see below).

It is now known that RLP terminals are isomorphic with retinal terminals [20,21,23,47,59], but the sources of the other terminal types are less securely identified and remain a focus of our research. It has been suggested that most F1 and F2 terminals are GABAergic [40] and local in origin, with F2 terminals issuing from dendrites of interneurons [14,24] and F1 terminals deriving, from axons of interneurons and/or perigeniculate cells (Refs. 9, 24 and 41, and see also below). Many

and perhaps the vast majority of RSD terminals derive from visual cortex [10,19,20,33,46]. As will be demonstrated below, F1 and RSD terminals may also be formed from axons deriving from the brainstem reticular formation.

Distribution of synaptic inputs onto relay cells

The distribution of synaptic inputs onto relay X and Y cells labeled intracellularly with HRP was described by our laboratory [63]. We found similarities and differences in the pattern of innervation of these cell classes. Our conclusions are schematically summarized by Fig. 4 (see also Refs. 23 and 24).

For both cell types, the distal dendrites (i.e., >100 μm from the soma) are dominated by synapses from RSD terminals, while the proximal dendrites (<100 μm from the soma) contain nearly all of the synapses from RLP, F1 and F2 terminals. Also, nearly half of the synapses derive from RSD terminals, 10–20% from RLP terminals, and the remainder from F1 and F2 terminals. These relative numbers of terminal types that contact individual X and Y cells closely match prior estimates of their relative distribution in the neuropil [19,20].

Other features of innervation patterns differ dramatically between X and Y cells. The vast majority of F2 terminals contact X cells, and contacts from F2 terminals outnumber those from F1 terminals on X cells by roughly 2 to 1. On Y cells, the difference is even more striking, since contacts from F1 terminals outnumber those

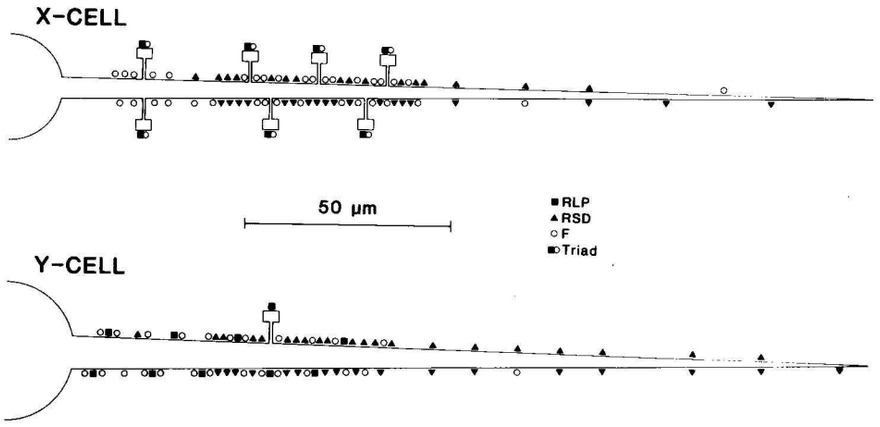

FIG. 4 Schematic summary of synaptic inputs and their terminals of origin for relay X and Y cells of the geniculate A-laminae. For simplicity, only a single unbranched dendrite is illustrated for each neuron. Also, no distinction is made between F1 and F2 terminals (both are shown as F terminals), but the F terminals contacting Y cells are predominantly of the F1 type whereas those contacting X cells are mostly of the F2 type. The triadic inputs shown for the X cell represent only those involving a retinal (RLP) terminal and an F2 terminal. The relative distribution and location of synapses is depicted fairly accurately, but only a fraction could be shown; on average, each of these neurons receives roughly 4000–5000 synapses. (Redrawn from Wilson et al. [63].)

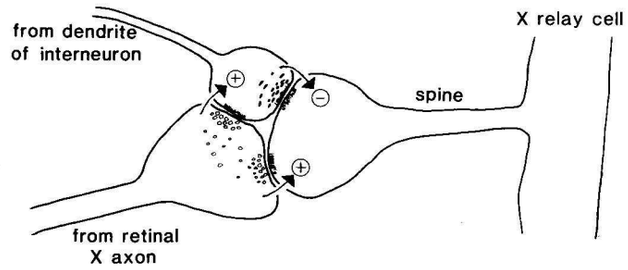

FIG. 5 Schematic drawing of a triadic synaptic relationship found in the geniculate A-laminae, commonly within complex synaptic glomerular zones (see Figs. 8 and 9). A retinal X axon contacts an F2 terminal arising from an interneuron's dendrite, and both terminals contact the same spine or appendage of a relay X cell.

from F2 terminals by approximately an order of magnitude. Another dramatic difference between cell types regards the nature of the retinal input. For Y cells, RLP terminals form simple synapses directly onto dendritic shafts, typically amongst input from the more numerous F1 terminals. For X cells, however, RLP terminals tend to contact dendritic appendages in a complex arrangement with F2 terminals: the RLP terminal contacts the F2 terminal, and both the F2 and RLP terminals contact the same dendritic appendage.

This arrangement which is a characteristic of F2 terminals and X cells but not of Y cells, is known as a *synaptic triad* and is schematically illustrated in Fig. 5. Another terminal type (e.g., an RSD terminal) may replace the RLP terminal in these triads (see below). Frequently, these synaptic interactions are embedded in a complex region known as a *synaptic glomerulus* [19,20,42,59], and glomeruli are common to geniculate circuitry in the X pathway but not in the Y pathway. It seems as if retinal input to X cells is often filtered through these glomeruli.

Sources of non-retinal inputs

While the above present the pattern of inputs onto the geniculate relay cells, what is still generally missing is an appreciation of the sources of these inputs. Until this is known, it will not be possible to understand how the various sources of non-retinal input control relay cell excitability and thus effect gating of retino-geniculocortical transmission. As noted above, we do know that RLP terminals derive from retina and that F2 terminals seem to issue from dendrites of interneurons, but the sources of the other inputs remains to be established. Preliminary work from our laboratory, as outlined below, has shed some light on the specific contributions of perigeniculate cells, interneurons, and axons from the brainstem reticular formation to these non-retinal inputs; inputs from visual cortex have yet to be characterized in detail.

Perigeniculate cells. As has been previously summarized in Fig. 2, perigeniculate cells receive input from axons of the brainstem reticular formation and from col-

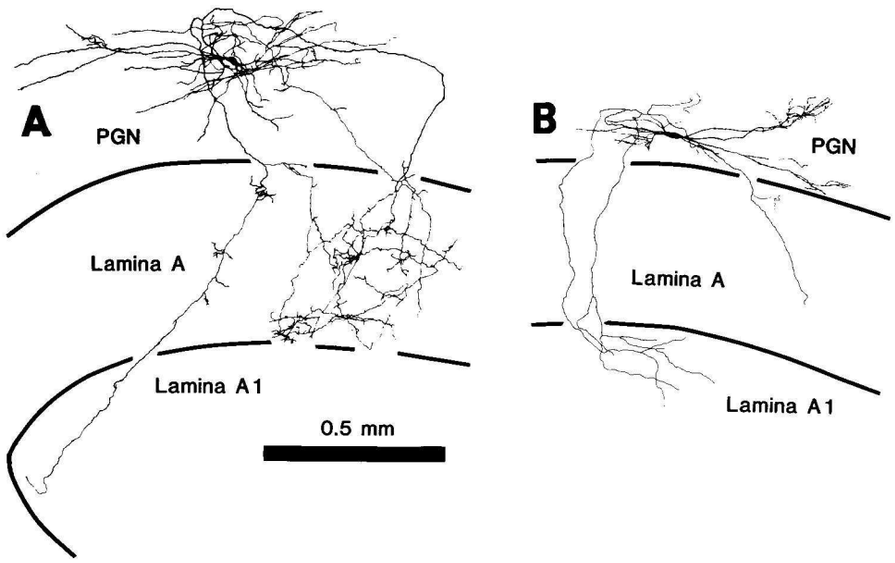

FIG. 6 Drawing of typical examples of perigeniculate (PGN) cells; coronal view. These cells were studied physiologically and labeled intracellularly with HRP. The dendritic arbors extend considerably along an axis parallel to the border with lamina A. The axons predominantly innervate the lateral geniculate nucleus, with the axon arbors there restricted to the A-laminae, but collaterals are also involved in local circuits in the perigeniculate nucleus. Within the lateral geniculate nucleus, each of these axons innervates both laminae A and A1, but each predominantly innervates either lamina A if the neuron's responses are much stronger to visual stimulation of the contralateral retina or lamina A1 if the cell responds predominantly to visual stimulation of the ipsilateral retina. A. Perigeniculate neuron with a receptive field dominated by the contralateral retina. B. Perigeniculate neuron with a receptive field dominated by the ipsilateral retina. The axon arbor in the A-laminae is incompletely labeled, but the general shape of the arbor there can still be discerned. (Drawing from unpublished data of Uhlrich et al. [61].)

laterals of both geniculocortical and corticogeniculate axons. Our preliminary studies of perigeniculate cells [9,61] have employed the intracellular labeling of individual cells with HRP. This labeling not only fills the soma and dendrites, but also labels the axon and much or all of its terminal arbor. Examples of labeled perigeniculate cells are shown in Fig. 6.

Each axon of these cells exclusively innervates laminae A and A1, but pointedly fails to innervate the C-laminae or the medial interlaminar nucleus. Each axon innervates one of the A-laminae more densely, and this correlates with the ocular dominance of the parent perigeniculate cell's receptive field properties. That is, although each of these labeled neurons can be binocularly activated, it responds better to visual stimulation of one eye than of the other; when the contralateral eye is dominant, the major projection is to lamina A, and when the ipsilateral eye dominates, lamina A1 receives the larger input [61]. Within the A-laminae, these axons display many *en passant* swellings or boutons. Our electron-microscopic investigations have demonstrated that these boutons are actually the synaptic terminals and that they are of the F1 variety [9]. Axons from perigeniculate cells are thus the source of many F1 terminals. Our preliminary evidence suggests that at least some perigeniculate cells directly innervate relay X cells, and other indirect evidence suggests that relay Y cells also receive direct innervation from perigeniculate cells.

Interneurons. Interneurons have a remarkable morphology (see Fig. 7), which generally corresponds to the *class 3* cell as described by Guillery [18] from Golgi impregnations (see also Refs. 14, 15, 35 and 41). The cells have small somata with long thin tortuous dendrites that run vertically through one of the A-laminae without crossing a laminar border. Most remarkable are the complex appendages found throughout the dendritic arbor. These appendages are often clumped together, but can occur singly, and they often involve swellings that are connected to each other and to the stem dendrite by long thin processes. These swellings have the general form of boutons, which in turn impart an 'axoniform' appearance to the dendrites (cf. Ref. 18). True axons can rarely be identified with the light microscope, because they are difficult to impregnate in Golgi material, and they could easily be confused with dendrites in HRP-labeled material. When identified, these axons seem to terminate close to or within the dendritic arbor. In general appearance, these interneurons are remarkably like the interneurons of the ventrobasal thalamic nuclei as described elsewhere in this volume [45].

Each of the 15 interneurons so far labeled with HRP in our laboratory has displayed rather uniform physiological characteristics, including the presence of a conventional action potential and receptive field properties that are generally indistinguishable from those of relay X cells. Furthermore, the response latency of these cells to electrical activation of the optic chiasm is not different from that of relay X cells, which indicates that these interneurons are directly innervated by retinogeniculate X axons. Presumably, more subtle electrophysiological tests, perhaps involving intracellular recording, will demonstrate clear differences between interneurons and relay X cells. In any case, all of these interneurons seem to be firmly entrenched in the X pathway (see also below), and we have as yet found no clear evidence for the presence of interneurons innervated by retinogeniculate Y axons (but see Ref. 36).

We have begun an electron-microscopic investigation of HRP-labeled interneurons and found many differences between their synaptic inputs and those of relay cells [24,63]. For instance, none of our sample of relay cells receives retinal inputs onto its soma, but this occurs for interneurons. More importantly, we have been able to confirm the conclusion reached by Famiglietti and Peters [14] that the bouton-like appendages along the dendrites are actually synaptic terminals of the F2 type. As respectable F2 terminals, they are both postsynaptic and presynaptic, since they receive inputs from other RLP, RSD and F1 terminals as well as contact appendages of relay X cells in triadic synaptic arrangements. After HRP iontophoresis into a single interneuron, we have found both labeled and unlabeled F2 terminals contacting the same postsynaptic profiles, which implies that synaptic input

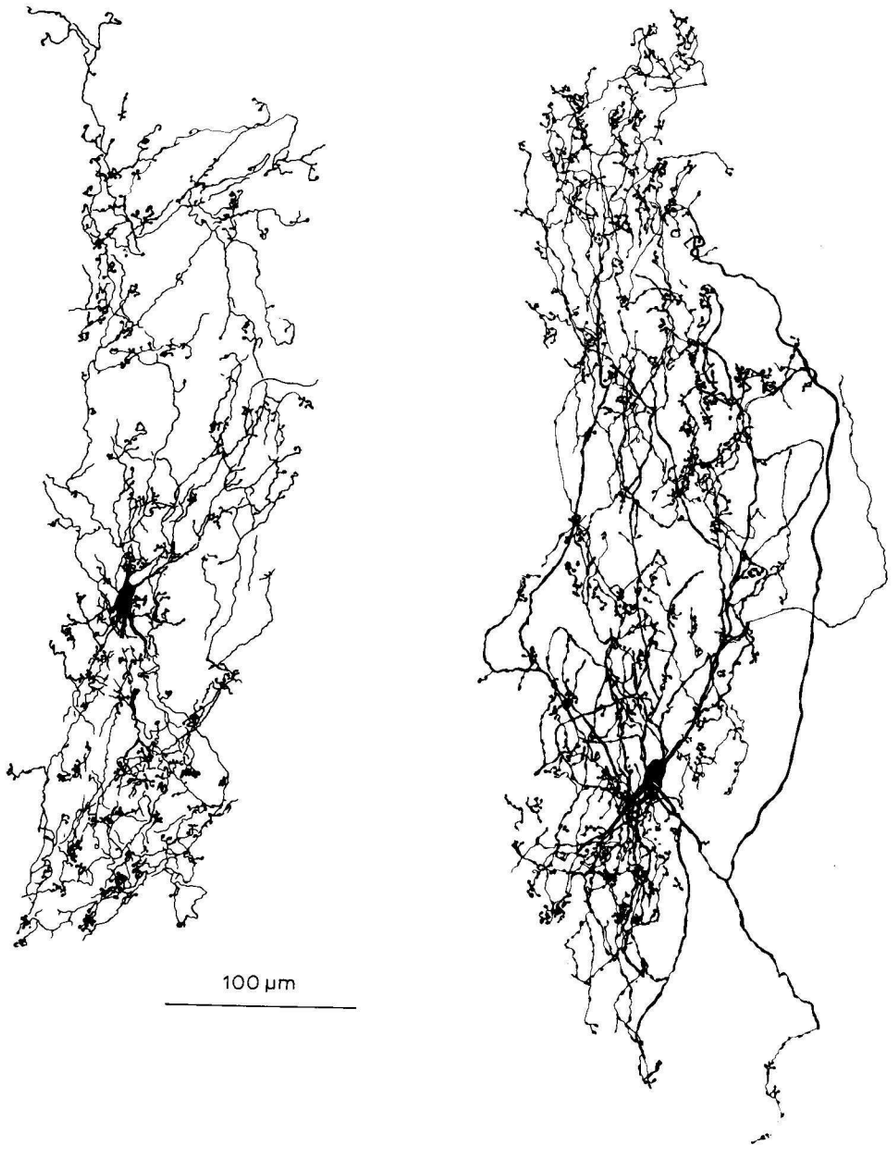

FIG. 7 Drawing of typical examples of interneurons in the geniculate A-laminae; coronal view. These cells were studied physiologically and labeled intracellularly with HRP. The laminar borders (not shown) run horizontally, so the long axis of each dendritic arbor runs perpendicular to these borders. All of the interneurons studied to date in this fashion exhibit conventional action potentials, and their receptive field properties are indistinguishable from those of relay X cells.

from several interneurons converges onto individual postsynaptic cells.

Fig. 8 shows a reconstruction of a cluster of appendages from a labeled interneuron along with its postsynaptic partner, which is a set of dendritic appendages from a relay X cell. The connections are richly embellished with inputs from other terminal varieties in a complex glomerular zone, which, as noted above, is a feature typified by retinogeniculate circuitry of the X pathway. Because F2 terminals from interneurons appear to be strategically sited to regulate retinal inputs in the glomerulus, and because most retinal inputs to relay X cells are involved in complex synaptic relationships with F2 (and other non-retinal) terminals [23,63], the interneurons seem capable of playing a major role in gating retino-geniculo-cortical transmission for the X pathway.

Although both the interneurons and the relay X cells have dendritic appendages, Fig. 8 shows a clear and perhaps functionally vital difference between them. The boutons on the interneuron are connected to each other and to the stem dendrite by exceedingly fine and long processes, whereas the appendages of the relay X cell are relatively short and stubby. We have modeled the significance of this morphology, with the very important and as yet untested assumption that these dendritic membranes are passive and do not conduct regenerative potentials [24]. By our model, the tips of the appendages on the relay X cell are within a fraction of a length constant from the stem dendrite, and thus postsynaptic potentials generated there will be faithfully conducted to the soma and axon hillock with relatively little attenuation (less than half). In contrast, the F2 terminals so far modeled that emanate from the interneuron's appendages are several length constants from the dendritic shaft, so that the synaptic inputs onto these processes would be attenuated by 10–100-times before reaching the stem dendrite. A consequence for interneurons is that each cluster of appendages along its dendritic arbor is effectively isolated electrically from all other clusters. Since the F2 terminals within the clusters are both presynaptic and postsynaptic, this implies that the interneurons dendritic arbor supports hundreds of local circuits.

Also, interneurons have a dendritic branching pattern that is not conducive to efficient propagation of voltage signals across dendritic branch points. Little of any postsynaptic potential generated beyond the first few branch points will reach the soma, and interneurons often have 15th or higher order dendritic branching. Indeed, most or all synapses formed onto the dendritic appendages, or F2 terminals, of these interneurons would have their signals attenuated by several orders of magnitude before reaching the soma. Our modeling of these neurons also suggests that dendritic sections separated by several branch points will be electrically isolated from one another. In contrast, as noted above, dendritic branching in relay cells seems designed for efficient propagation of such signals throughout the dendritic arbor [3,4].

If the soma, where electrodes most likely sample electrical activity of the neuron, is electrically isolated from the dendritic clusters of interneurons, then synaptic inputs onto these clusters cannot be appreciated in conventional recording situations. The prior statements about electrophysiology of interneurons may thus reflect only synaptic inputs onto proximal dendrites and not those more distally located. Obviously, this creates an important qualification to any conclusions concerning the

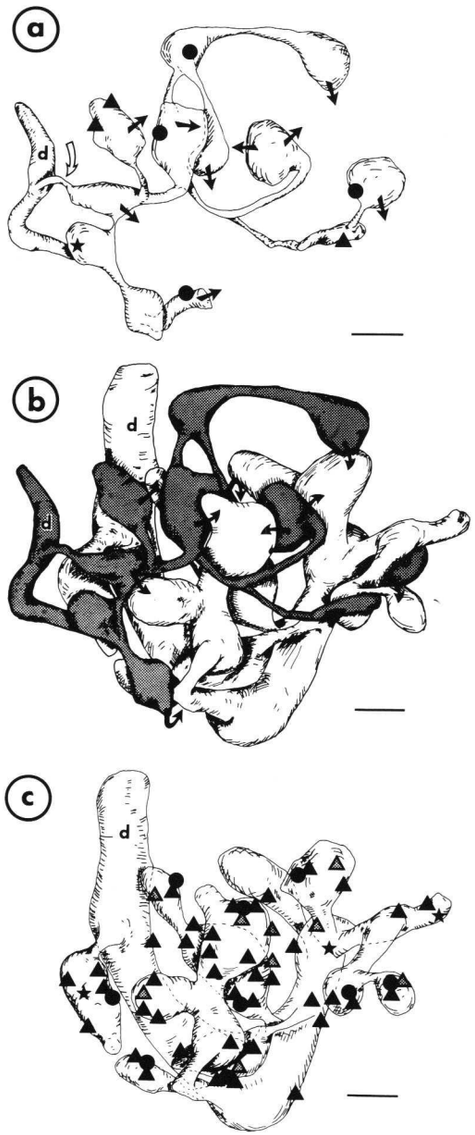

FIG. 8 Reconstruction of a glomerular zone in the geniculate A-laminae, showing the F2 terminals from an intracellularly labeled interneuron, the postsynaptic cluster of appendages from a relay X cell, and the location of synaptic contacts; each scale bar represents 1.0 μm. **a**. Labeled processes from the interneuron. A thin stem dendrite (d) emits an extremely fine process (open arrow) that arborizes into twelve F2 terminals connected by extremely fine processes. These terminals are postsynaptic to retinal or RLP terminals (circles), unlabeled F terminals (triangles; most or all of these may be F2 terminals, but they were not sufficiently reconstructed to be certain), and an RSD terminal (star). The labeled F2 terminals also form synaptic outputs (solid arrows). **b**. Combined reconstruction of the labeled interneuron's processes from **a** (stippled) and unlabeled postsynaptic processes from **c** (open). The synapses from the F2 terminals onto the relay X cell's appendages are illustrated (solid arrows; these represent the same solid arrows as in **a**). **c**. Unlabeled postsynaptic dendrite (d) from a relay X cell with eight appendages that receive all of the neuron's synaptic input in the reconstructed zone. These include nine synapses from RSD or retinal terminals (circles), nine from F2 terminals of the labeled interneuron (stippled triangles; these correspond to the solid arrows in **a** and **b**), 40 from unlabeled F terminals (solid triangles), and three from RSD terminals (stars). The 16 triadic synaptic arrangements are illustrated by overlapping pairs of symbols for synapses from RLP and F terminals. (Taken from Hamos et al. [24].)

response properties of interneurons, including the conclusion that retinogeniculate Y axons do not innervate them.

This unusual integrative behavior of interneurons based on cable modeling raises an interesting question: Why, if the F2 outputs of the interneuron are electrically isolated from the soma, should the neuron exhibit conventional action potentials? The answer seems to be that at least some of these interneurons have myelinated axons, and action potentials would be needed to conduct information along these axons. We have begun to reconstruct the contacts made by one of these axons. Our very preliminary data suggest the possibility that these axons culminate in F1 terminals within the dendritic arbor. We still cannot even speculate as to the identity of the postsynaptic elements, except for the observation that no element reconstructed as yet is innervated by labeled terminals of both F1 and F2 varieties. Interestingly, Montero [41] has made comparable observations for interneurons with *unmyelinated* axons, except that he describes their axonal boutons as having morphology subtly different from that of typical F1 terminals.

This creates an interesting and unconventional picture of the functioning of interneurons. They seem to be engaged in two separate and parallel computations with two separate outputs. The conventional output seems to involve action potentials conducted along a myelinated axon, and this would reflect integration of inputs limited to the proximal dendrites [4]. The unconventional output derives locally from dendritic appendages that are also postsynaptic processes and that usually assemble in electrically isolated clusters along the distal dendrites. The two outputs seem even to use different terminals: F1 terminals for the axon and F2 terminals for the dendrites.

Brainstem reticular formation. We have labeled and reconstructed at the light and electron microscopic levels individual axons from the brainstem reticular formation, mostly from the parabrachial region [10,62]. However, instead of intracellular iontophoresis of HRP, these were labeled by placing the anterograde tracer, *Phaseolus vulgaris*-leucoagglutinin (PHA-L), among their somata; the PHA-L is transported along the axons and their terminal arbors where immunohistochemical techniques can be used to visualize the label.

In addition to the dorsal division of the lateral geniculate nucleus, these axons

frequently innervate other thalamic nuclei that are within the central visual pathways. These include the ventral division of the lateral geniculate nucleus, the perigeniculate nucleus, and portions of the lateral posterior-pulvinar complex. We have never seen an axon that innervates the lateral geniculate nucleus plus a non-visual thalamic structure, such as the ventrobasal complex or medial geniculate nucleus. The terminal arbors of these axons within the lateral geniculate nucleus are sparsely branched with boutons mostly *en passant*.

Our initial electron microscopic studies of these labeled axons has supported the notion that the boutons seen at the light microscopic level are the exclusive site of synaptic contacts. Furthermore, these observations have generally proved complementary to those of De Lima et al. [11]. They labeled terminals within the A-laminae with an antibody directed against choline acetyltransferase and studied this material with the electron microscope. These cholinergic terminals, which presumably derive from the parabrachial region [12], have morphological features remarkably similar to those we have seen for terminals labeled from PHA-L injections into the parabrachial region.

We have reconstructed portions of single axons labeled from the parabrachial region. These axons frequently enter synaptic glomeruli to contact dendritic shafts and appendages of presumed relay X cells plus F2 terminals. These axons also innervate other dendritic segments that have not yet been identified as to cell type. Since electrophysiological evidence indicates that axons from the brainstem reticular formation directly innervate interneurons and relay Y cells as well as relay X cells (e.g., Ref. 1; reviewed in Ref. 53), it is plausible that some of these unidentified postsynaptic profiles belong to these other cell types. In any case, Fig. 9 summarizes some of the synaptic relationships we have documented for the X pathway. Frequently, a parabrachial axon enters into triadic synaptic relationships with F2 terminals and dendritic appendages of relay X cells; dendritic shafts of the same postsynaptic cell are also contacted. However, unlike the triadic relationships formed by retinal terminals, for which a single RLP terminal contacts both the dendritic appendage and the F2 terminal, those formed from parabrachial axons usually involve separate terminals that contact these postsynaptic elements. Interestingly, a retinal terminal and parabrachial axon often share the same F2 terminal and dendritic appendage for their own triadic relationships. Thus, at least for the X pathway, input from the parabrachial region is ideally sited to gate retino-geniculo-cortical transmission.

Perhaps most surprisingly, we documented several examples of an individual parabrachial axon that produces some terminals forming symmetric synapses and others forming asymmetric synapses. This challenges the conventional notion that any axon produces a morphologically homogeneous type of terminal and synapse. The dense PHA-L labeling within the terminals tends to obscure features such as vesicle morphology, and thus it is difficult to be certain how these terminals would be identified if unlabeled. With this proviso, we tentatively consider the terminals that form symmetric synapses to be F1 terminals and those forming asymmetric synapses to be RSD terminals (for a more complete discussion of this, see Ref. 10). We found a strong tendency for the putative RSD terminals to contact dendritic appendages, while the putative F1 terminals contact dendritic shafts and F2 terminals.

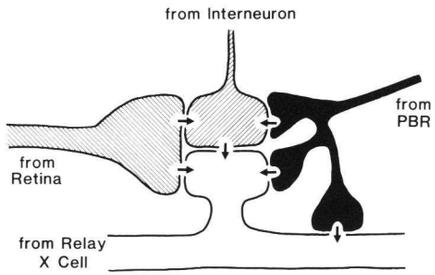

FIG. 9 Schematic drawing of the innervation by a single parabrachial axon of X cell circuitry within the lateral geniculate nucleus. The single axon contacts an F2 terminal from an interneuron plus a dendritic appendage and shaft of a relay X cell; the F2 terminal contacts the same dendritic appendage. Thus, the parabrachial axon engages in triadic relationships similar in many ways to those of retinal terminals. In fact, as shown, a parabrachial axon and retinal terminal often share the same F2 terminal and dendritic appendage in their triadic relationships. The synapses formed by the parabrachial axon onto dendritic appendages tend to be asymmetric, whereas those formed onto dendritic shafts and F2 terminals tend to be symmetric. (Taken from Cucchiaro et al. [10].)

Visual cortex. It has been known for many years that corticogeniculate axons form synapses from RSD terminals (e.g., Refs. 10, 19, 20, 33 and 46). Although other sources of RSD terminal have been recently documented, including our tentative conclusion that one such source is the innervation from the brainstem reticular formation, it is likely that the vast majority of RSD terminals derive from visual cortex, which implies that corticogeniculate axons produce the plurality of synapses onto geniculate relay cells. In fact, corticogeniculate axons outnumber geniculocortical axons by a factor of roughly 10. Despite this, depressingly little is known about the functional significance of this input (for a discussion of this, see Ref. 53). Likewise, insufficient morphological information is available to characterize this pathway at the single neuron level. We hope to address some of these morphological issues by applying the above-mentioned PHA-L-labeling technique to individual corticogeniculate axons.

Conclusions

Contemporary studies of the cat's lateral geniculate nucleus performed in a number of laboratories, including ours, has enabled us to begin to appreciate the functional organization of this nucleus. For nearly 20 years, students of the lateral geniculate nucleus have recognized the broad classes of synaptic terminal in the geniculate neuropil and have also realized that retinal terminals are a small minority there, although until recently this has not seemed to influence notions about the functional significance of geniculate circuitry very much. We now have a much better understanding of how these different terminal types contribute to the innervation of geniculate relay cells. We are also beginning to determine the different sources

TABLE 1 *Synaptic profiles in the A-laminae of the cat's lateral geniculate nucleus*

Type	Source	Percent
RLP	retinal axons	10–20
RSD	cortical and BRF[a] axons	40–45
F1	various axons[b]	20–25
F2	dendrites of interneurons	20–25

[a]Brainstem reticular formation.
[b]Axons from interneurons, from cells of the perigeniculate nucleus (and reticular nucleus of the thalamus), and from cells of the brainstem reticular formation.

for these terminal types (see Table 1), although much more still needs to be learned. Finally, we have clear evidence that these morphological features of geniculate circuitry differ significantly between the X and Y pathways.

The point of all of this morphological work is that it represents an attempt to understand the anatomical basis for an important and interesting function: namely, the control of retino-geniculo-cortical transmission. As noted elsewhere (see Ref. 53), the gating of this transmission may represent a key neuronal substrate for visual attention, although various areas of visual cortex are almost certainly also intimately involved in the control of visual attention. Since 'visual attention' is an umbrella term that covers many related but separate phenomena, there must be many distinct neuronal substrates for it. Perhaps the great complexity and variety of non-retinal inputs to geniculate relay cells represent different circuits for different attentional mechanisms.

Acknowledgements

The research attributed to my laboratory represents the combined efforts of many colleagues, some of whom have since left to establish their own laboratories. The most important contributors to the observations described above include, in alphabetical order, S.A. Bloomfield, J.B. Cucchiaro, M.J. Friedlander, J.E. Hamos, D. Raczkowski, L.R. Stanford, D.J. Uhlrich, S.C. Van Horn and J.R. Wilson. My laboratory's research has been supported by USPHS grants EY03038 and EY03604.

References

1 Ahlsen G, Grant K, Lindstrom S. *Monosynaptic excitation of principal cells in the lateral geniculate nucleus by corticofugal fibers.* Brain Res 1982;234:454–458.
2 Ahlsen G, Lindstrom S, Lo F-S. *Inhibition from the brain stem of inhibitory interneurons of the cat's dorsal lateral geniculate nucleus.* J Physiol (Lond) 1984;347:593–609.
3 Bloomfield SA, Hamos JE, Sherman SM. *Passive cable properties and morphological correlates of neurons in the lateral geniculate nucleus of the cat.* J Physiol (Lond) 1987;383:653–692.
4 Bloomfield SA, Sherman SM. *Dendritic current flow in X cells in the cat's lateral geniculate nucleus: a comparison of relay cells cells and interneurons.* Soc Neurosci Abstr 1987;13:203.

5 Bloomfield SA, Sherman SM. 1988 (in preparation).
6 Cleland BG, Duvin MW, Levick WR. *Sustained and transient neurones in the cat's retina and lateral geniculate nucleus.* J Physiol (Lond) 1971;217:473–496.
7 Cleland BG, Lee BB. *A comparison of visual responses of cat lateral geniculate nucleus neurones with those of ganglion cells afferent to them.* J Physiol (Lond) 1985;369:249–268.
8 Crick F. *Function of the thalamic reticular complex: the searchlight hypothesis.* Proc Natl Acad Sci 1984;81:4586–4590.
9 Cucchiaro J, Uhlrich DJ, Hamos JE, Sherman SM. *Perigeniculate input to the cat's lateral geniculate nucleus: a light- and electron microscopic study of single, HRP-filled cells.* Soc Neurosci Abstr 1985;11:231.
10 Cucchiaro J, Uhlrich DJ, Sherman SM. *Parabrachial innervation of the cat's dorsal lateral geniculate nucleus: an electron microscopic study using the tracer Phaseolus vulgaris-leucoagglutinin (PHA-L).* 1988 (in press).
11 De Lima AD, Montero VM, Singer W. *The cholinergic innervation of the visual thalamus: an EM immunocytochemical study.* Exp Brain Res 1985;59:206–212.
12 De Lima AD, Singer W. *The brainstem projection to the lateral geniculate nucleus in the cat: identification of cholinergic and monoaminergic elements.* J Comp Neurol 1987; 259:92–121.
13 Dowling JE. *Organization of vertebrate retinas.* Invest Ophthal 1970;9:655–680.
14 Famiglietti EV Jr, Peters A. *The synaptic glomerulus and the intrinsic neurons in the dorsal lateral geniculate nucleus of the cat.* J Comp Neurol 1972;144:285–334.
15 Friedlander MJ, Lin C-S, Stanford LR, Sherman SM. *Morphology of functionally identified neurons in the lateral geniculate nucleus of the cat.* J Neurophysiol 1981;46:80–129.
16 Friedlander MJ, Stanford LR, Sherman SM. *Effects of monocular deprivation on the structure–function relationship of individual neurons in the cat's lateral geniculate nucleus.* J Neurosci. 1982;2:321–330.
17 Gilbert C. *Microcircuitry of the visual cortex.* Annu Rev Neurosci 1983;6:217–247.
18 Guillery RW. *A study of Golgi preparations from the dorsal lateral geniculate nucleus of the adult cat.* J Comp Neurol. 1966;128:21–50.
19 Guillery RW. *The organization of synaptic interconnections in the laminae of the dorsal lateral geniculate nucleus of the cat.* Z Zellforsch 1969;96:1–38.
20 Guillery RW. *A quantitative study of synaptic interconnections in the dorsal lateral geniculate nucleus of the cat.* J Comp Neurol 1969;96:39–48.
21 Guillery RW, Geisert EE, Polley EH, Mason CA. *An analysis of retinal afferents to the cat's medial interlaminar nucleus and to its rostral thalamic extension, the 'geniculate wing'.* J Comp Neurol 1980;194:117–142.
22 Hallanger AE, Levey AI, Lee HJ, Rye DB, Wainer BH. *The origins of cholinergic and other subcortical afferents to the thalamus in the rat.* J Comp Neurol 1987;262:105–124.
23 Hamos JE, Van Horn S, Raczkowski D, Sherman SM. *Synaptic circuits involving an individual retinogeniculate axon in the cat.* J Comp Neurol 1987;259:165–192.
24 Hamos JE, Van Horn SC, Raczkowski D, Uhlrich DJ, Sherman SM. *Synaptic connectivity of a local circuit neuron in the cat's lateral geniculate nucleus.* Nature 1985;317:618–621.
25 Hickey TL, Guillery RW. *An autoradiographic study of retinogeniculate pathways in the cat and the fox.* J Comp Neurol 1974;156:239–254.
26 Hubel DH, Wiesel TN. *Integrative action in the cat's lateral geniculate body.* J Physiol (Lond) 1961;155:385–398.
27 Hubel DH, Wiesel TH. *Receptive fields, binocular interaction and functional architecture in the cat's visual cortex.* J Physiol (Lond) 1962;160:106–154.
28 Hubel DH, Wiesel TN. *Receptive fields and functional architecture in two nonstriate visual areas (18 and 19) of the cat.* J Neurophysiol 1965;28:229–289.

29 Ikeda H, Wright MJ. *Receptive field organization of sustained and transient retinal ganglion cells which subserve different functional roles.* J Physiol (Lond) 1972;227:769–800.
30 Jahnsen H, Llinás R. *Electrophysiological properties of guinea-pig thalamic neurones: an in vitro study.* J Physiol (Lond) 1984;349:205–226.
31 Jahnsen H, Llinás R. *Ionic basis for the electroresponsiveness and oscillatory properties of guinea-pig thalamic neurones in vitro.* J Physiol (Lond) 1984;349:227–247.
32 Jones EG. *The thalamus.* New York: Plenum Press, 1985
33 Jones EG, Powell TPS. *An electron microscopic study of the mode of termination of corticothalamic fibres within the sensory relay nuclei of the thalamus.* Proc R Soc B 1969; 172:173–185.
34 Lennie P. *Parallel visual pathways.* Vision Res 1980;20:561–594.
35 LeVay S, Ferster D. *Relay cell classes in the lateral geniculate nucleus of the cat and the effect of visual deprivation.* J Comp Neurol 1977;172:563–584.
36 Lindstrom S. *Synaptic organization of inhibitory pathways to principal cells in the lateral geniculate nucleus of the cat.* Brain Res 1982;234:447–453.
37 Mastronarde DN. *Two classes of single-input X-cells in cat lateral geniculate nucleus. I. Receptive-field properties and classification of cells.* J Neurophysiol 1987;57:357–380.
38 Mastronarde DN. *Two classes of single-input X-cells in cat lateral geniculate nucleus. II. Retinal inputs and the generation of receptive-field properties.* J Neurophysiol 1987; 57:381–413.
39 McCormick DA, Prince DA. *Actions of acetylcholine in the guinea pig and cat medial and lateral geniculate nuclei, in vitro.* J Physiol (Lond) 1987;392:147–165.
40 Montero VM, Singer W. *Ultrastructural identification of somata and neural processes immunoreactive to antibodies against glutamic acid decarboxylase (GAD) in the dorsal lateral geniculate nucleus of the cat.* Exp Brain Res 1985;59:151–165.
41 Montero VM. *Ultrastructural identification of synaptic terminals from the axon of type 3 interneurons in the cat lateral geniculate nucleus.* J Comp Neurol 1987;264:268–283.
42 Peters A, Palay SL. *The morphology of lamina A and A1 of the dorsal nucleus of the lateral geniculate body of the cat.* J Anat (Lond) 1966;100:451–486.
43 Pfaffmann C, Frank M, Norgren R. *Neural mechanisms and behavioral aspects of taste.* Annu Rev Psychol 1979;30:283–325.
44 Rall W. *Core conductor theory and cable properties of neurons.* In: Kandel ER, Geiger S, eds. *Handbook of physiology section 1: the nervous system. vol. 1, Cellular biology of neurons.* Bethesda: American Physiological Society, 1977;39–97.
45 Ralston HJ, III, Ohara PT, Ralston DD, Chazal G. *The neuronal and synaptic organization of the primate somatosensory thalamus.* 1988 (in the present book).
46 Robson JA. *The morphology of corticofugal axons to the dorsal lateral geniculate nucleus in the cat.* J Comp Neurol 1983;216:89–103.
47 Robson JA, Mason CA. *The synaptic organization of terminals traced from individual labeled retino-geniculate axons in the cat.* Neuroscience 1979;4:99–111.
48 Rowe MH, Dreher B. *W cell projection to the medial interlaminar nucleus of the cat: implications for ganglion cell classification.* J Comp Neurol 1982;204:117–133.
49 Rye DB, Saper CB, Lee HJ, Wainer BH. *Pedunculopontine tegmental nucleus of the rat: cytoarchitecture, cytochemistry and some extrapyramidal connections of the mesopontine tegmentum.* J Comp Neurol 1987;259:483–528.
50 Sakai K. *Some anatomical and physiological properties of ponto-mesencephalic tegmental neurons with special reference to the PGO waves and postural atonia during paradoxical sleep.* In: Hobson JA, Brazier MAB, eds. *The reticular formation revisited.* New York: Raven Press, 1980;427–447.
51 Shapley R, Lennie P. *Spatial frequency analysis in the visual system.* Annu Rev Neurosci 1985;8:547–583.

52 Sherman SM. *Functional organization of the W-, X- and Y-cell pathways: a review and hypothesis.* In: *Progress in psychobiology and physiological psychology, vol. 11,* Spraque JM, Epstein AN, eds. New York: Academic Press, 1985;233–314.
53 Sherman SM, Koch C. *The control of retinogeniculate transmission in the mammalian lateral geniculate nucleus.* Exp Brain Res 1986;63:1–20.
54 Sherman SM, Spear PD. *Organization of the visual pathways in normal and visually deprived cats.* Physiol Rev 1982;62:738–855.
55 Singer W. *Control of thalamic transmission by corticofugal and ascending reticular pathways in the visual system.* Physiol Rev 1977;57:386–420.
56 Steriade M, Deschênes M. *The thalamus as a neuronal oscillator.* Brain Res Rev 1985;8:1–63.
57 Stone J. *Parallel processing in the visual system.* New York: Plenum Press, 1983.
58 Stone J, Dreher B, Leventhal A. *Hierarchical and parallel mechanisms in the organization of visual cortex.* Brain Res Rev 1979;1:345–394.
59 Szentagothai J, Hamori J, Tömböl T. *Degeneration and electron microscope analysis of the synaptic glomeruli in the lateral geniculate body.* Exp Brain Res 1966;2:283–301.
60 Tsumoto T, Creutzfeldt OD, Legendy CR. *Functional organization of the corticofugal system from visual cortex to lateral geniculate nucleus in the cat.* Exp Brain Res 1978;32:345–364.
61 Uhlrich DJ, Cucchiaro JB, Sherman SM. *Innervation of the cat's lateral geniculate nucleus by individual cells of the perigeniculate nucleus.* Soc Neurosci Abstr 1987;13:1435.
62 Uhlrich DJ, Cucchiaro JB, Sherman SM. *The projection of individual axons from the parabrachial region of the brainstem to the dorsal lateral geniculate nucleus in the cat.* J Neurosci 1988 (in press).
63 Wilson JR, Friedlander MJ, Sherman SM. *Ultrastructural morphology of identified X- and Y-cells in the cat's lateral geniculate nucleus.* Proc R Soc B 1984;221:411–436.