JOURNAL OF NEUROPHYSIOLOGY
Vol. 53, No. 2, February 1985. *Printed in U.S.A.*

Cat Lateral Suprasylvian Cortex: Y-Cell Inputs and Corticotectal Projection

DAVID M. BERSON

Division of Biology and Medicine, Section of Neurobiology, Brown University, Providence, Rhode Island 02912

SUMMARY AND CONCLUSIONS

1. Retinal Y-cells activate most cells in the deep layers of the cat's superior colliculus via an indirect pathway involving the occipital cortex (4, 5). The lateral suprasylvian area seems to be an important source of visual input to the deep collicular strata (5, 34, 57, 76, 77), but it is unclear whether Y-cell influences reach this extrastriate area and, hence, whether this area participates in the indirect Y-cell pathway.

2. In this study, retinal influences on the posteromedial lateral suprasylvian area (PMLS) were studied in anesthetized cats. Responses to electrical stimulation of the optic disk (OD) and optic chiasm (OX) were recorded in single units in PMLS and in neurons of the dorsal lateral geniculate nucleus (LGNd) that were antidromically driven from PMLS.

3. Virtually all PMLS cells (99%; 99/100) exhibited small differences ($\leq$0.8 ms) between OD- and OX-activation latency, indicating that they were driven by a pathway originating in rapidly conducting Y-cell axons.

4. A small number of PMLS cells (17%; 20/118) had very short activation latencies ($\leq$3.2 ms from OX), comparable to those of cells in areas 17 and 18 receiving monosynaptic inputs from geniculate Y-cells (13, 16, 30, 53, 88). Further, LGNd cells with latency behaviors typical of Y-cells could be antidromically driven from PMLS, confirming that geniculate Y-cells project directly to PMLS.

5. Most PMLS cells (83%; 98/118), though exhibiting small OD-OX latency differences, had absolute latencies too long to be attributed to direct inputs from geniculate Y-cells (3.3–8.5 ms from OX). Thus Y-cells in the LGNd influence most PMLS cells by way of a multisynaptic pathway.

6. PMLS cells antidromically activated from the superior colliculus were driven only by this multisynaptic Y-cell input. Total conduction time from the retina through PMLS to the colliculus corresponds closely to the latency of the indirect Y-cell activation observed in the deep collicular layers (4).

7. These results support the view that the lateral suprasylvian cortex constitutes an important source of visual input to the cat's deep collicular layers and, more generally, that the extrastriate visual cortex may figure prominently in the cortical control of gaze.

INTRODUCTION

The neocortex constitutes a major source of visual input to cells in the deep layers of the mammalian superior colliculus, i.e., those lying below the stratum opticum (5, 38, 57, 67, 75, 83). Because these neurons in turn influence neural circuits controlling eye and head movements (1, 15, 23, 29, 42, 66), visual cortical inputs to the deep collicular layers may be important in the initiation and guidance of orienting movements to visual stimuli.

In the cat, we have identified a polysynaptic pathway that originates in retinal Y-cells, synapses in the occipital cortex, and drives the great majority of deep collicular cells, including those with axons that descend in the predorsal bundle (4, 5). It is not known which areas of the visual cortex contribute

to this "indirect Y-cell pathway," but several observations implicate the lateral suprasylvian (Clare-Bishop) area. First, the corticotectal projections of the lateral suprasylvian area terminate in the optic and intermediate layers (40, 76, 77), laminae containing the dendritic arborizations of many deep-layer cells (56, 90). Electrical stimulation of the posteromedial lateral suprasylvian area (PMLS) activates most deep-layer cells, and at latencies short enough in many cases to suggest a monosynaptic connection (5). Second, large cortical lesions that include the lateral suprasylvian area eliminate the indirect Y-cell influence on the deep colliculus (5) and selective inactivation or destruction of this area renders deep collicular cells largely unresponsive to visual stimulation (34, 57). Finally, the lateral suprasylvian area's projections to the colliculus likely carry visual signals originating at least partly in retinal Y-cells, since this cortical area receives input from the medial interlaminar nucleus (MIN) and lamina C of the lateral geniculate nucleus (3, 19, 37, 45, 47, 55, 63, 64, 68, 87) and from cortical areas 17 and 18 (9, 18, 19, 36, 62), all of which receive major inputs from Y-cell pathways (7, 8, 10, 12, 14, 30, 43, 49, 53, 58, 79, 85, 88, 91).

The experiments reported here document prominent Y-cell influences on neurons of PMLS, including those projecting to the superior colliculus. The findings further implicate the lateral suprasylvian cortex in the transmission of visual information to the deep collicular strata and thus in the generation of visually guided shifts of gaze.

METHODS

Preparation

After the induction of surgical anesthesia, cats were placed in a stereotaxic instrument. Core temperature was maintained automatically near 38°C. The nictitating membranes were retracted with phenylephrine and the corneas were protected with plano contact lenses. The skull and dura overlying the optic chiasm (OX), lateral suprasylvian cortex, and superior colliculus or lateral geniculate nucleus were removed and the brain was covered with warm mineral oil or agar. Concentric bipolar electrodes were placed in the OX and left optic disk (OD) as previously described (4). Square-wave, constant-current pulses of 50 μs and <5 mA (measured with Hewlett-Packard 1110 current monitor) were delivered through these electrodes. Wound margins and pressure points were infiltrated with a long-lasting local anesthetic (Duranest).

In some cases, paralysis was induced with Flaxedil (20 mg, iv) and maintained by continuous infusion of Flaxedil (5 $mg \cdot kg^{-1} \cdot h^{-1}$) and *d*-tubocurarine (0.5 $mg \cdot kg^{-1} \cdot h^{-1}$) in 5% dextrose in water. In these cases, bilateral pneumothorax was performed, the animals were artificially ventilated with an N_2O/O_2 mixture (70:30), and end-tidal CO_2 was maintained near 4%. The electroencephalogram was monitored and small doses of barbiturates were infused as needed to maintain slow-wave activity (27).

The activity of single units in the cortex or thalamus was recorded with varnished tungsten microelectrodes (5–15 MΩ at 1 kHz), amplified and displayed by conventional techniques, and stored on magnetic tape. Units with action potentials characteristic of axons (6) were excluded from the data analysis. Antidromic spikes, recorded either in the cortex or in the thalamus, were identified by their stable latencies, their collision with evoked orthodromic spikes, and their ability to follow stimulation at frequencies >500 Hz (17, 46). One or more small electrolytic lesions were placed in each recording track to aid histological reconstruction.

Cortical experiments

In 10 cats, recordings were made in the lateral suprasylvian visual area. In early experiments, surgical anesthesia was induced with pentobarbital sodium (35–40 mg/kg, ip) or ketamine HCl (30–40 mg/kg, ip); recordings were carried out under nitrous oxide or pentobarbital sodium anesthesia. Cortical responsiveness was best in later experiments in which ketamine HCl alone was used for surgery and recording. These animals were not paralyzed and ketamine HCl was infused as needed to suppress spontaneous limb movements. Atropine sulfate (0.04 mg/kg, im) was administered to control salivation.

Under stereotaxic guidance, a comb of three concentric bipolar stimulating electrodes (Rhoades, type NE-100; diam, 0.2–0.5 mm; tip exposure, 0.5 mm; tip-to-sleeve separation, 1 mm) was lowered by a vertical approach into the rostral half of the right superior colliculus or the brachium of the superior colliculus. The final placement of these electrodes was optimized by stimulating through them and recording the field potentials evoked at the OD or OX. Current pulses were passed either between the core and sleeve of a single electrode or between two electrodes. Geniculocortical fibers were not stimulated directly at

the dorsal lateral geniculate nucleus (LGNd) or optic radiation since this could have resulted in concurrent activation of other fiber systems, such as those originating in the lateral posterior nucleus (25, 68).

Single units in PMLS (59) were recorded on the right side, between Horsley-Clarke levels A5 and P2; electrodes were angled 30–45° from vertical in the frontal plane.

Lateral geniculate experiments

In 9 cats, unit activity was recorded in the LGNd. Surgery was carried out under pentobarbital sodium anesthesia (35–40 mg/kg, ip and supplemented iv as needed). During recording, animals were paralyzed and maintained as described previously.

A comb of three concentric bipolar stimulating electrodes identical to that used for collicular stimulation was inserted into PMLS on the right side. The electrodes were separated from one another by 1–2 mm and were angled 35–40° from vertical in the frontal plane. Tips were usually inserted <2 mm into the sulcal cortex and never extended more than halfway down the medial bank of the middle suprasylvian sulcus. In every case, electrode tips were confirmed by histology to lie within the gray matter of PMLS between Horsley-Clarke levels A5 and P1. Shocks were delivered either between the core and sleeve of single electrodes or between electrode pairs.

Retinal landmarks for both eyes were plotted by tapetal reflection (61) on a tangent screen 75 cm distant. Recording electrodes were lowered vertically into the right LGNd, and the receptive fields of single units or multiunit clusters were plotted on the tangent screen using small spots of light from a hand-held projector. These plots, together with retinotopic maps of the geniculate (69), were helpful in positioning recording electrodes within the MIN.

Histology

Animals were given an overdose of barbiturate and perfused through the carotids with 10% formalin. The brain was cut at 100 μm on a freezing microtome and sections were stained with cresyl violet. Recording tracks and sites of stimulation were reconstructed. Units recorded in PMLS were assigned to cortical layers according to the terminology of Sanides and Hoffmann (70), and those in the LGNd were assigned to subdivisions of the geniculate according to the scheme of Hickey and Guillery (33).

The average conduction distance between OD and OX electrodes was estimated to be 23.1 mm, a figure that combines the average length of the optic nerve, as determined by postmortem dissection, with the average displacement of the OX electrode from the center of the chiasm, as measured in histological sections.

RESULTS

Recordings from lateral suprasylvian area

RETINAL ACTIVATION OF PMLS. Electrical stimulation of retinal fibers typically evoked a prominent biphasic field potential in PMLS (Fig. 1). The initial negative deflection of this response, recorded at 8 sites in 5 animals, occurred 3.8–4.7 ms after OD shock and 3.4–4.1 ms after OX shock. The difference between OD and OX latency at individual recording sites (n = 8) ranged from 0.3 to 0.6 ms. Dividing these OD-OX latency differences into the conduction distance between the OD and OX (23.1 mm) yields 39–77 m/s as an estimate of the range of conduction velocities of retinal fibers initiating this cortical response. These results are in accord with earlier suggestions, based on extracellular field potentials (9, 89), that the lateral suprasylvian cortex is influenced by signals originating in the most rapidly conducting class of retinofugal axons.

Single units recorded in PMLS responded to OD or OX shock with one or more action potentials. These spikes were most commonly superimposed on the negative phase of the field potential. Figure 2, *A* and *B*, illustrates

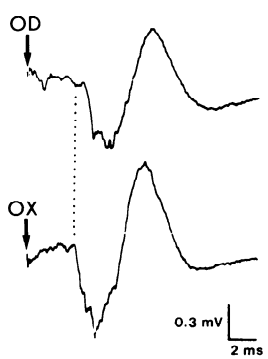

FIG. 1. Field potentials evoked in posteromedial lateral suprasylvian area (PMLS) by electrical stimulation of contralateral optic disk (OD) and optic chiasm (OX). Single sweep is shown in both cases. Prominent negative phase of field potential evoked by OX shock (*dotted line*) began <1 ms earlier than that evoked from OD. Recorded at border of layers IV and V. Both stimuli were 1.8 mA for 50 μs. Bandpass of recording system: 100 Hz–30 kHz. Upward deflection is positive.

the responses of a single unit in PMLS to stimulation of retinal fibers. The spike evoked by OD stimulation (Fig. 2*A*) occurred at ~5 ms, and the spike evoked by OX stimulation (Fig. 2*B*) occurred only ~0.7 ms earlier. The small OD-OX latency difference suggests that, like the field potential, this response was mediated by rapidly conducting retinal axons. This was a corticotectal cell because stimulation of the superior colliculus (Fig. 2*C*) elicited a spike of antidromic origin, as shown by its short, stable latency (0.8 ms) and by its collision with orthodromic potentials evoked from the OD (Fig. 2*D*). Collicular stimulation also evoked a second, longer-latency action potential (4–5 ms), which showed substantial latency jitter and was presumably orthodromic in nature.

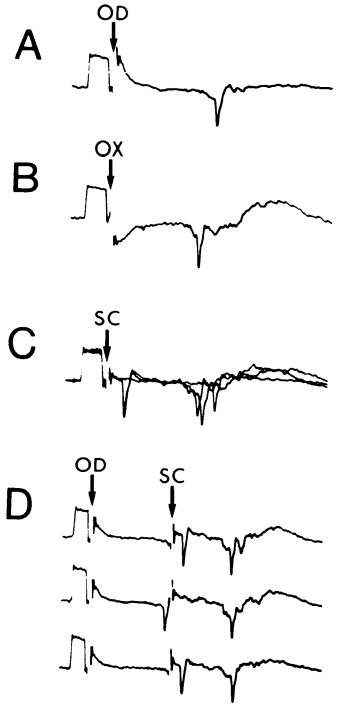

FIG. 2. Responses of identified corticotectal cell in PMLS. *A* and *B*: stimulation of OD (*A*, 1.6 mA) evoked action potential <1 ms later than spike evoked from OX (*B*, 0.2 mA). *C*: stimulation of ipsilateral superior colliculus (SC) evoked both short latency antidromic spike (in 2/3 superimposed traces) and longer latency orthodromic spikes (all 3 traces). Stimulus was near threshold for antidromic response (0.2 mA). *D*: collision test. SC shock (0.4 mA) followed OD shock (1.0 mA) by fixed interval. Short-latency antidromic spike (*top and bottom traces*) evoked from SC collides with orthodromic spike (*middle trace*) evoked from OD. All stimuli in *A–D* were 50 μs in duration. Square-wave calibration pulses at beginning of each trace: +400 μV, 1 ms; sweep speed is slower in *D* than in *A–C*. Amplitude of field potentials reduced by high-pass filtering (bandpass: 3–100 kHz).

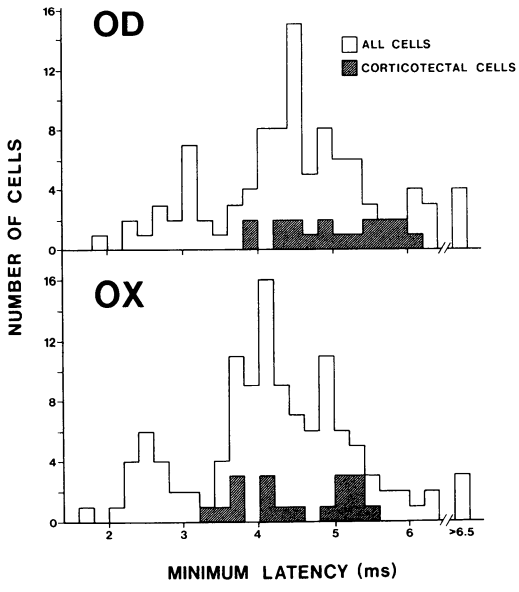

FIG. 3. Distribution of minimum latencies of activation of PMLS cells by stimulation of OD or OX. *Open bars* indicate total sample of units; *hatched bars* indicate subset antidromically activated from SC.

Figure 3 illustrates the distribution of minimum response latencies of units in PMLS after stimulation of the OD and OX. In both histograms, a small peak appears at ~2–3 ms and a broad, larger peak occurs at longer latencies. For the purposes of this report, cells discharging at short latency (OD latencies ≤ 3.5 ms, OX latencies ≤ 3.2 ms) are said to exhibit an "early response," and those discharging at longer latencies, a "late response." Evidence is presented below that the early response reflects a direct input from geniculocortical axons, whereas the late response is mediated by indirect geniculate inputs to PMLS.

The late response was common among PMLS neurons, occurring in 83% (98/118) of the cells overall, and in every cortical layer except layer I. Cells that were antidromically activated from the superior colliculus ("corticotectal cells") exhibited exclusively this late response. Spikes of the late response

occurred in synchrony with the negative phase of the prominent evoked potential illustrated in Fig. 1.

The early response appeared in only 17% (20/118) of PMLS units. These units were encountered most frequently in layer IV, though a few were recorded in the infragranular layers. Spikes of the early response were not usually associated with any field potential, though a small negative wave beginning 2.3–2.6 ms after OD shock and 1.9–2.0 ms after OX shock was occasionally recorded in PMLS (cf. Ref. 89). In about half the cells exhibiting the early response, the short-latency action potential was followed by a second spike at a delay of 1–2 ms. The latter corresponded in latency to the late response observed in other PMLS cells and sometimes appeared in isolation after OD and OX shocks.

CONDUCTION VELOCITY OF RETINAL FIBERS MEDIATING CORTICAL ACTIVATION. Responses of individual PMLS cells, whether early or late, exhibited small OD-OX latency differences ($\leq$1.5 ms). As a result, the OX-latency histogram in Fig. 3 is shifted slightly and uniformly to the left with respect to the OD-latency histogram. Corticotectal cells, for example, were excited on average 5.0 ms after OD shock (range, 3.9–6.0 ms) and 4.4 ms after OX shock (range, 3.2–5.4 ms). Dividing this difference in mean response latency (0.6 ms) into the estimated mean OD-OX conduction distance (23.2 mm) yields 39 m/s as an estimate of the average conduction velocity of retinal fibers mediating the excitation in these corticotectal units; this is a velocity typical of retinal Y-cell axons (11).

The distribution of individual OD-OX latency differences for all cells of PMLS is plotted in Fig. 6*A*. From a comparison with the expected latency differences for W-, X-, and Y-cell mediated responses, it is evident that the latency differences observed were typical, almost without exception, of a Y-cell pathway and that the mean latency difference (0.47 ms) was very close to that expected for such a pathway (0.45 ms). Thus virtually all recorded PMLS cells were driven by a Y-cell input.

THE CORTICOTECTAL PATHWAY. Units in PMLS antidromically activated from the superior colliculus were recorded almost exclusively within layers IV and V (97%; 96/99).[1] Latencies of the antidromic responses ranged from 0.3 to 6.4 ms (median, 1.7 ms).

If PMLS participates in the indirect Y-cell input to the colliculus (4, 5), then conduction time through the retina-PMLS-colliculus circuit should correspond to the latency of indirect Y-cell activation of collicular cells after the stimulation of retinal fibers. The time required for retinal output to reach the superior colliculus by way of PMLS may be estimated by adding the OD activation latency of individual corticotectal cells in PMLS to their latency to antidromic activation from the colliculus. The mean value of this sum was 6.1 ms (range, 4.4–7.8 ms; n = 18). If 0.5 ms is taken as the synaptic delay at the colliculus, then a volley set up at the OD and transmitted through PMLS should excite collicular cells with a mean latency of 6.6 ms. This value corresponds closely to the mean latency for indirect Y-cell activation of collicular cells after OD shock [6.7 ms; (4)].

Stimulation of the superior colliculus evoked orthodromic responses in many units recorded in PMLS, including corticotectal cells (see Fig. 2*C*). Comparable responses have been observed in areas 17 and 18, where the orthodromic responses are thought to result from stimulation of Y-cell axons in the colliculus, antidromic conduction to their branchpoint in the optic tract, and subsequent orthodromic activation of geniculocortical Y-cell pathways (16, 79, 88). For cells in areas 17 and 18, this interpretation is supported by the strong positive correlation between the latency of the orthodromic response evoked from the OX and that evoked from the colliculus (79, 88). The same strong correlation characterizes PMLS cells in this study (r = +0.95; n = 25), so that in this extrastriate area too, activation of Y-cell pathways may be responsible for orthodromic responses evoked by collicular stimulation. However, other circuits, such as tectothalamocortical pathways or intracortical collaterals of corticotectal axons, may also play a role.

[1] This sample is larger than that represented by the hatched bars of Fig. 3 because it includes cells (primarily from barbiturate-anesthetized animals) that were unresponsive to OD or OX shocks.

Recordings from lateral geniculate nucleus

The findings of part I indicate that PMLS receives visual input from a pathway originating in retinal Y-cells. To determine if some of this input is transmitted directly to PMLS from the lateral geniculate nucleus, I sought to record from cells in the LGNd that could be activated antidromically from the lateral suprasylvian area. A special effort was made to record from cells in the MIN, since this component of the LGNd is known to contain Y-cells (14, 43, 49, 58) and to have a substantial projection to the lateral suprasylvian cortex (3, 19, 37, 45, 47, 64, 68, 87).

Recordings from a cell lying at the lateral border of MIN are illustrated in Fig. 4. This unit was identified as a geniculate Y-cell on the basis of its short latency of activation from the OD (1.4 ms, Fig. 4*A*) and OX (0.9 ms, Fig. 4*B*) and its small OD-OX latency difference (0.5 ms). Stimulation of ipsilateral PMLS evoked a short-latency action potential (Fig. 4*C*), whose antidromic origin was confirmed by its collision with orthodromic spikes evoked from the OX (Fig. 4*D*) and by its ability to follow faithfully three shocks to PMLS at 1,000 Hz (Fig. 4*E*).

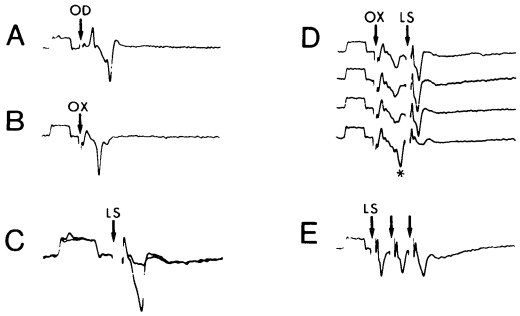

FIG. 4. Unit responses of cell of dorsal lateral geniculate nucleus (LGNd) antidromically driven from PMLS. *A* and *B*: stimulation of OD (*A*) evoked spike with latency under 2 ms; this was <1 ms longer than latency of spike evoked from OX (*B*). *C*: antidromic activation of same unit from PMLS; 2 superimposed traces show all-or-none nature of short-latency spike evoked by stimulation of lateral suprasylvian (LS) area; gain and sweep speeds are greater than in *A* and *B*. *D*: collision test. Orthodromic action potential evoked from OX in *bottom trace* (*asterisk*) collides with antidromic spike evoked from LS. *E*: antidromic action potential evoked from LS follows 3 shocks at 1 kHz. Stimulus intensities: OD, 1.0 mA; OX, 0.1 mA; and LS, 3.2 mA. All stimuli were 50 μs in duration. Square-wave calibration pulses at beginning of each trace: +400 μV, 1 ms.

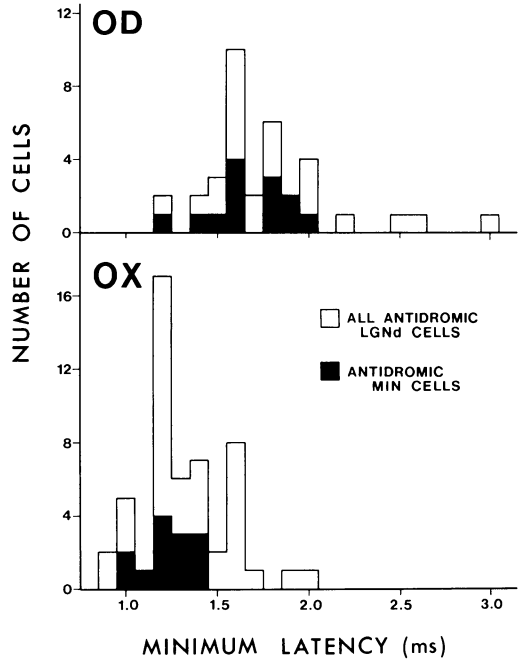

FIG. 5. Distribution of minimum latencies of activation from OD or OX of cells in LGNd that were antidromically driven from PMLS. *Open bars*, total sample of such geniculate cells; *filled bars*, subset lying within medial interlaminar nucleus (MIN).

Stimulation of PMLS evoked antidromic responses not only in MIN cells, but in cells of the laminar LGNd as well. A few of these were recorded in lamina C, which has at least a weak projection to PMLS (37, 45, 87), but most were encountered in layers A and A1, which have been thought to project exclusively to areas 17 and 18. No antidromic responses were recorded in the parvocellular C-laminae, but units were rarely encountered in these layers, probably because the electrodes used were not optimal for recording small cells. Antidromically driven cells of the laminar LGNd were comparable to those of the MIN both with respect to latency of antidromic activation from PMLS (laminar LGNd cells: 0.2–0.8 ms, $\bar{x}$ = 0.52, n = 38; MIN cells: 0.3–2.4 ms, $\bar{x}$ = 0.81, n = 26) and in terms of their retinal input (see below).

Fibers originating in the MIN and the lateral posterior nucleus and destined for the

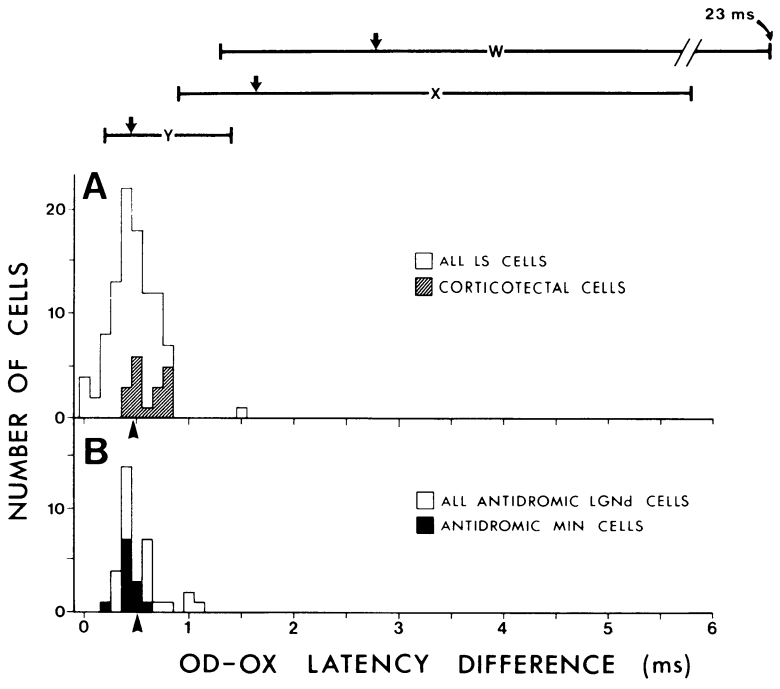

FIG. 6. Distribution of differences in response latency after OD and OX stimulation for individual cells recorded in LS (*A*) or LGNd (*B*). *A*: *open bars,* total sample of lateral suprasylvian cells; *hatched bars,* subset of cells antidromically activated from colliculus. *B*: *open bars,* all LGNd cells antidromically driven from lateral suprasylvian area; *filled bars,* subset lying in MIN. *Arrowheads* below *abscissae* in *A* and *B* indicate mean values for total samples. Top of figure, ranges (*horizontal lines*) and mean latency differences (*arrows*) expected for responses mediated by W-, X-, and Y-type retinal fibers; values were determined by dividing average electrode separation in present study by fastest, slowest, and mean conduction velocities for each fiber population (11).

lateral suprasylvian area traverse the A-laminae (25, 68), but it is unlikely that these axons generated the antidromic responses recorded in the A-layers. These A-layer units had spike waveforms indicative of recordings from cell bodies, not axons (6), and their ocularity and receptive-field locations matched those of neighboring cells recorded in the same lamina. Considerable suspicion must exist, however, that the antidromic responses were produced by current spread from the cortical stimulus site to the underlying optic radiations. This possibility is taken up in the DISCUSSION.

Figure 5 plots, for all LGNd cells antidromically activated from PMLS, the distribution of minimum activation latencies from the OD and OX. The observed latencies fell almost entirely within the range reported for geniculate Y-cells (13, 14, 35, 85, 91). The difference between the mean latency after OD shock (1.8 ms) and OX shock (1.3 ms) was only 0.5 ms, indicating that the retinal axons driving these geniculate cells had an average conduction velocity of 46 m/s, close to the mean conduction velocity of Y-cell axons [51.9 m/s; (11)]. Figure 6*B,* which plots the distribution of individual OD-OX latency differences, suggests that virtually all of these units are Y-cells. All of the differences lay within the range predicted for Y-cells, and were in most cases too small to be attributed to any other class of cells; in addition, the mean latency difference (0.51 ms) fell close to that expected for Y-cell mediated responses (0.45 ms).

DISCUSSION

The present study provides the first direct physiological evidence that neurons in the cat's lateral suprasylvian visual area are driven by a Y-cell pathway. Among the recipients of this Y-cell influence are cells of PMLS

that send axons to the superior colliculus. This supports the idea that PMLS may be a major component of the polysynaptic Y-cell pathway to the deep collicular layers (4).

Y-cell inputs to lateral suprasylvian area

Units in PMLS exhibited, almost without exception, the small OD-OX latency differences expected for cells driven by a Y-cell pathway (Fig. 6*A*). This preponderance of Y-cell influence is comparable to that reported for area 18 (30, 85, 88). PMLS undoubtedly receives additional excitatory input from more slowly conducting geniculocortical pathways, because it receives direct input from the parvocellular C-laminae and geniculate wing (3, 37, 45, 63, 64, 87), which are driven almost exclusively by W-cell input (12, 26, 39, 45, 50, 91, 92). The failure to detect the influence of W-cell (and X-cell) pathways may have resulted from biases in this study against detecting such inputs. Cortical cells were characterized primarily on the basis of minimum response latency, which favors the detection of faster retinal inputs; pathways contributing to later components of the cortical response are more difficult to isolate. In addition, stimulation of retinal fibers is relatively ineffective in driving polysynaptic X- and W-cell pathways, in part because synaptic transmission through these pathways is vulnerable to inhibition mediated by Y-cells at the LGNd and cortex (13, 16, 35, 78, 79, 85, 88). Nonetheless, X- and W-cell influence has been detected in areas 17 and 19 with comparable methods (8, 13, 16, 79, 85), so that the present results may imply a special association of PMLS with the Y-cell stream.

Direct geniculate Y-cell projections to lateral suprasylvian area

More than 40 years ago, Marshall, et al. (48) demonstrated that retinal inputs to the suprasylvian cortex survived bilateral destruction of the striate area. They suggested that direct projections from the lateral geniculate body to this extrastriate region might exist, and their hypothesis has been amply confirmed by anatomical methods (3, 19, 37, 44, 45, 47, 63, 64, 68, 87).

In an early field potential study, Vastola (89) argued that these direct geniculate inputs to the lateral suprasylvian cortex conveyed retinal signals originating in the most rapidly conducting optic tract fibers. He was unable to rule out possible contamination of his suprasylvian recordings by activity in the underlying geniculostriate radiations, but the present study suggests that his inference was correct. First, cells in PMLS exhibiting the early response to stimulation of retinal fibers have latency behaviors indistinguishable from those of cells in areas 17 and 18 receiving direct geniculate Y-cell input (13, 16, 30, 88). Further, cells in the LGNd with latency behaviors characteristic of geniculate Y-cells could be antidromically driven from PMLS (Figs. 4–6). These geniculate neurons were driven orthodromically from the OX with an average latency of 1.3 ms and were antidromically activated from PMLS with a mean latency of 0.6 ms. Assuming a 0.5-ms synaptic delay at the cortex, these geniculate Y-cells should therefore drive cells in PMLS monosynaptically on average 2.4 ms after OX shock. This is very close to the peak of the short-latency hump in the latency histogram of Fig. 3.

Indirect geniculate Y-cell inputs to lateral suprasylvian area

Monosynaptic inputs from geniculate Y-cells would be expected to drive cells in PMLS at latencies no longer than $\sim$3.2 ms after OX shock; this value combines the largest sum of orthodromic OX latency and antidromic PMLS latency among geniculate cells of the present study (2.7 ms) with an estimated synaptic delay at the cortex of 0.5 ms. Cells in PMLS exhibiting a late response, though unquestionably driven by a Y-cell pathway (Fig. 6*A*), respond to OX shock at latencies $>$3.2 ms. These neurons, which constitute a majority of the PMLS cells encountered, must therefore be activated indirectly by geniculate Y-cells.

Neurons in PMLS getting monosynaptic geniculate input undoubtedly make local synaptic contacts and are probably an important source of polysynaptic Y-cell inputs to other PMLS cells. Such local intracortical circuitry has been suggested to underlie comparable indirect geniculate Y-cell activation of cells in areas 17 and 18 (16, 30, 79, 88). A second likely source of polysynaptic Y-cell

input is the corticocortical projection from areas 17 and 18 to PMLS (9, 18, 19, 36, 62); cells of area 17 projecting to the lateral suprasylvian area belong to a physiological class that is driven primarily by Y-cell input (8, 32). Whatever pathways are responsible, it is clear that monosynaptic and multisynaptic Y-cell input may converge in the lateral suprasylvian area, since some PMLS neurons exhibited both early and late responses (cf. Refs. 16, 79, 88).

All recorded corticotectal cells appeared to receive excitatory visual input by the indirect Y-cell pathway; there was no evidence of monosynaptic input from geniculate Y-cells, nor of driving by X- or W-cell pathways. These results are similar to those obtained for corticotectal cells in areas 17 and 18 of the cat (16, 31). Whether corticotectal cells as a class are devoid of any influence from the X- or W-cell streams is uncertain, but such inputs have not been detected with intracellular recordings (16). Moreover, the visual activity of corticotectal cells in the macaque appears to be effectively silenced by selective inactivation of the "Y-like" magnocellular layers of the geniculate (74).

Do geniculate A-laminae project to lateral suprasylvian cortex?

In this study, stimulation of PMLS evoked antidromic responses in many cells of the geniculate A-layers. The finding is at odds with a substantial body of anatomical literature indicating that direct projections from the LGNd to the lateral suprasylvian area arise almost exclusively from the C-layers and MIN (3, 19, 37, 44, 45, 47, 55, 63, 64, 68, 87) and that the cortical projections of the A-layers are restricted to areas 17 and 18 (44, 63, 68).

The shocks used in these experiments were relatively strong and there is some likelihood that the antidromic responses in the A-layers resulted from spread of stimulating current to the geniculostriate radiation, which lies just under the fundus of the middle suprasylvian sulcus. On the other hand, several observations raise doubt that the phenomenon is merely a reflection of unwanted current spread. First, it can be estimated that single 2-mA, 50-μs current pulses should directly excite myelinated axons no farther than 2 mm from the electrode tips;[2] such pulses evoked antidromic responses in the A-laminae even when delivered to sites more than 5 mm distant from the optic radiation. Second, barring marked differences in activation threshold[3] or trajectory among geniculocortical axons, current spread from PMLS to the optic radiation should have activated geniculate principal cells more or less indiscriminately. Instead, antidromic responses were recorded almost exclusively in geniculate Y-cells (Figs. 5 and 6*B*).

A convincing demonstration that the phenomena reported here reflect bona fide projections of A-layer cells to PMLS would entail a detailed quantitative evaluation of current spread that is beyond the scope of this study. It is noteworthy, however, that at least isolated labeled cells may be found in the A-laminae after retrograde tracer injections restricted to the lateral suprasylvian cortex [Refs. 3 (Figs. 18–20) and 45; see also Ref. 52], and a reexamination of the question seems warranted as more sensitive tracing methods are developed.

Lateral suprasylvian area and visual input to deep collicular layers

Most cells of the cat's deeper collicular layers receive effective visual input from a polysynaptic pathway originating in retinal Y-cells and transmitted by the visual cortex (4, 5). Several recent findings, including those reported here, implicate the lateral suprasylvian cortex in this indirect Y-cell input to the deep superior colliculus. First, stimulation of PMLS drives most deep collicular cells, in many cases at latencies that are short enough to be mediated by monosynaptic contacts

[2] This estimate is based on Ranck's comprehensive review of the literature on spread of stimulating current in the CNS (65). I have assumed that the 50-μs pulses used in the present report require about twice as much current to excite as the 200-μs pulses used to generate his estimates. Currents in excess of 10 mA should be required to excite axons 5 mm from the electrode.

[3] Axonal activation threshold and conduction velocity are inversely related (2). Though mean conduction velocity of X- and Y-type geniculocortical axons differ, there is substantial overlap between the populations (10, 91). Moreover, the distance from cortical stimulus sites to the radiations varied by a factor of two in these experiments, and the resulting variation in current density at the radiations presumably dwarfed any systematic threshold differences between the two axon classes.

(5). In addition, the time required for retinal Y-cell output to reach the colliculus by way of PMLS is very close to the latency of the indirect Y-cell activation recorded in the colliculus after stimulation of the optic disk (see RESULTS). Finally, large lesions of the occipital cortex that include PMLS drastically reduce indirect Y-cell influence on the deep layers (5), and local inactivation of the lateral suprasylvian cortex depresses visual responses in these layers (57).

The functional significance of visual cortical input to the deep layers lies in its ability to drive collicular cells emitting descending axons (5). These cells in turn provide input to the oculomotor tegmentum and cervical spinal cord (1, 15, 23, 24, 29, 42, 66), and hence appear to provide corticotectal cells with rather direct access to neural circuitry subserving orienting movements of the eyes and head. Consistent with this view, collicular ablations in cats interrupt cortical visual influences on certain preoculomotor neurons (42) and abolish the saccadic eye movements evoked by stimulation of the occipital cortex (82). Though these observations point to a major role for the visual cortex in the visuomotor functions of the colliculus, the widespread convergence of corticotectal projections raises the possibility that neither the lateral suprasylvian region, nor any other single cortical area, plays an essential role in the visuomotor operations of the colliculus. Behavioral studies are so far equivocal on this point: lateral suprasylvian lesions resulted in profound sensory neglect in one study (84) but had no detectable effect on visual localization or attention in another (81).

It is not known whether the visual cortex, particularly the extrastriate areas, will prove of comparable importance in deep collicular function in other species. This is particularly so for primates, in which frank visual responses are less robust and presaccadic activity less dependent on visual input than seems true for deep-layer cells in the cat (20, 22, 28, 51, 54, 60, 72, 80, 93). The similarities between feline and primate corticotectal organization are nonetheless striking. As in the cat, extrastriate areas in monkeys project to deeper tectal layers than does the striate cortex (21), and may therefore contact deep-layer cells directly. Corticotectal cells in the visual cortex of the macaque are driven by the "Y-like" (magnocellular) geniculocortical stream (74), and silencing these cells (73, 75) effectively eliminates the visual responses of deep collicular neurons (75). Finally, collicular ablation abolishes saccades evoked by electrical stimulation of the parieto-occipital cortex in the monkey (41, 71), just as it does in the cat. These parallels identify the role of the visual cortex, especially that of the extrastriate areas, in colliculo-oculomotor function as a promising avenue for further research.

ACKNOWLEDGMENTS

I am grateful to James T. McIlwain for his advice and support and for his comments on the manuscript. I thank Rose Davis for secretarial assistance.

This work was supported by a National Institutes of Health Postdoctoral Research Fellowship to the author (EY-05425) and Grant EY-02505, and a Grass Foundation Trustees Grant to J. T. McIlwain.

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