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Visual Cortical Inputs to Deep Layers of Cat's Superior Colliculus

DAVID M. BERSON AND JAMES T. McILWAIN

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

SUMMARY AND CONCLUSIONS

1. In the superior colliculi of cats anesthetized with ketamine, 84% of identified output cells of the deep layers could be driven by shocks to the contralateral optic disk, optic chiasm, or ipsilateral optic tract; 75% of these deep-layer cells had response latencies reflecting a polysynaptic influence of retinal Y-cells. Following large, acute lesions of the ipsilateral occipital cortex (including visual areas 17, 18, 19, and the posteromedial lateral suprasylvian area (PMLS)), only 18% of deep-layer output cells were driven by electrical stimulation of the optic pathway and only 4% exhibited an indirect Y-cell influence. Thus, one or more of these visual areas may be important for the relay of retinal information, and particularly of Y-cell information, to the deep layers of the superior colliculus.

2. This hypothesis is supported by the observation that intracortical stimulation in areas 17, 18, 19, and PMLS activated many cells of the ipsilateral, deep tectal layers at latencies consistent with those exhibited by the indirect Y-cell pathway. The distributions of activation latencies were similar to those observed in the superficial layers, raising the possibility that at least some of the cortical influence on the deep layers may be mediated by direct connections.

3. Cells of the deep layers were more likely to be excited by a cortical stimulus that activated cells immediately above them in the superficial layers than by a stimulus that did not. This indicates that the functional connections between visual cortex and the deep collicular layers exhibit a topographic orderliness similar to that previously described for corticotectal projections to the superficial layers.

4. These results provide further evidence that the visual cortex exerts a significant influence on cells of the deep collicular strata and that the pathways involved are capable of mediating the indirect, retinal Y-cell input to these neurons.

INTRODUCTION

It is well established that the deep layers of the superior colliculus (i.e., those lying below the stratum opticum) participate in visually guided shifts of gaze (26, 49, 59, 68, 74, 81, 91), but it is not clear how retinal information reaches those deep tectal neurons that are thought to project to saccadic mechanisms in the brain stem. There is a direct input from the retina to the deep collicular layers, but this is apparently sparse (5, 18). The existence of fiber projections from the visually responsive superficial layers to the deeper strata, though long inferred, has recently been called into question (15). Circuitous routes through the basal ganglia have also been suggested on the basis of morphological findings (45, 63), but the visual responsiveness of the cells in these pathways has not been documented sufficiently to permit this possibility to be assessed.

Recently, we showed that most deep-layer cells in the cat receive excitatory influences by way of a polysynaptic pathway originating in retinal Y-cells (5). Because of its striking similarity to the "Y-indirect pathway" reaching the superficial collicular layers (30), we hypothesized that this multisynaptic retinal influence might reflect a major input to neurons of the deep tectum from the visual cor-

tex. Here we report the results of two sets of experiments testing this hypothesis. We first examined the effect of occipital cortical ablation on the Y-indirect input to deep tectal neurons. We then electrically stimulated each of several visual cortical areas to see if activity in their efferents would influence cells in the deep collicular laminae. The results support the hypothesis of a cortical role in the Y-indirect influence on the deep layers. They demonstrate that most deep-layer cells receive robust excitatory influences from the visual cortex and suggest that at least a part of this influence may reach the deep layers directly.

METHODS

Preparation

Adult cats were administered an initial dose of ketamine hydrochloride (30–40 mg/kg, ip), which was supplemented intravenously as needed to suppress spontaneous, organized limb movements. Since different cats require quite different maintenance doses of ketamine (unpublished observations), we did not administer paralyzing agents to these animals. (In an early attempt to use paralyzed animals, anesthetized with nitrous oxide supplemented with pentobarbital (25), unit responses in the deep collicular layers appeared depressed, so the approach was abandoned.) Salivation was controlled with atropine sulfate (0.04 mg/kg, im). Core temperature was automatically maintained at 38°C and the corneas were protected with plano contact lenses. The cat was placed in a stereotaxic instrument modified to permit visual stimulation, and the skull overlying the right superior colliculus and visual cortex was removed. The brain was covered with warm mineral oil.

Unitary action potentials, recorded in the right superior colliculus with varnished tungsten microelectrodes (5–15 MΩ at 1 kHz), were amplified and displayed by conventional methods, and traces of interest were recorded on magnetic tape. Electrode contact with the surface of the superior colliculus was recognized by the appearance of "juxtazonal potentials" (52) and the ensuing penetration involved all collicular layers. Units with action potentials characteristic of axons (6) were excluded from the data analysis. One or more electrolytic lesions were made in each track to aid subsequent histological reconstruction. Antidromic action potentials, evoked in deep-layer collicular cells from a pair of electrodes in the left predorsal bundle, were identified by their short and stable latencies and by collision with evoked orthodromic spikes. In this report, data and conclusions derived from this sample of antidromically activated cells will be specifically noted. Otherwise, reference is to the entire sample of deep-layer cells, which includes neurons not antidromically driven from the brain stem.

Ablation studies

In seven cats, suction lesions of the right occipital cortex were made immediately before recording. The lesions were large, including nearly all of the cortex known to be visually responsive (Fig. 1). Concentric bipolar electrodes were placed in the left optic disk (OD) and either the optic chiasm (OX) or the right optic tract (OT), as previously described (5). Single shocks 0.05 ms in duration and less than 5 mA in intensity were used. Latencies to evoked action potentials were measured from the onset of the shock artifact to the foot of the spike. Stimulus intensities were measured with a Hewlett-Packard 1110 current monitor. For comparison with the results of these ablation studies, we have included data from 13 cats without cortical lesions ("intact cats"), which were part of a study reported previously (5). In two cats, single units were also recorded in the colliculus contralateral to the cortical lesion and stimulating electrodes were placed in the optic chiasm and both optic disks.

Cortical stimulation studies

In the 15 animals of this series, we limited the mobility of the left eye, usually by attaching the conjunctiva with sutures and a tissue adhesive to a brass ring fixed rigidly to the stereotaxic frame. In a few cases the eye was stabilized with an intraocular electrode that impaled the optic disk. Residual shifts of eye position were limited to about 5° in amplitude, as determined from the projection of retinal landmarks on a tangent screen 75 cm distant from the cat (60). This degree of stability permitted us to estimate roughly the retinotopic location of recording sites in the superior colliculus and visual cortex. The pupils were dilated with atropine, the nictitating membranes contracted with phenylephrine, and the right eye was covered with an opaque patch.

Low-impedance tungsten microelectrodes (<2 MΩ at 1 kHz) were positioned within areas 17, 18, 19, or the posteromedial lateral suprasylvian area (PMLS) of visual cortex by reference to published maps (58, 84, 85). Usually one or two cortical areas were stimulated in a single experiment with from one to four electrodes placed in a particular area. Recording multiunit responses through these electrodes, we could confirm their placement within areas 17, 18, and often within area 19 by observing the shift of receptive fields as the electrodes were moved through the area. In these preparations, possibly due to the anesthesia, we usually could not plot receptive fields for sites

in PMLS and so have relied exclusively on histological confirmation of these placements. Cortical electrodes were fixed in position and connected to a constant-current stimulator that delivered single cathodal pulses of 0.05 ms duration and up to 2 mA in intensity. Effects of cortical stimulation on collicular units were studied in both deep and superficial layers.

Histology

At the end of the experiment, animals were given a lethal dose of pentobarbital and perfused through the carotids with 10% Formalin. Electrode tracks in the colliculus and suction lesions or sites of stimulation in the cortex were reconstructed from 100-μm frozen sections stained with cresyl violet. Sites of collicular unit recordings were attributed to the superficial or deep laminae (respectively, laminae I–III or IV–VII of Ref. 39). Results of stimulation at cortical sites lying in white matter or outside the target area have been excluded from the analysis.

Conduction velocity estimates

The average conduction distance between OD and OT electrodes was estimated to be 35.6 mm, a figure that combines the distance of 12.7 mm from the OT electrode to the midpoint of the chiasm (mean of 22 cats) with an average disk-to-chiasm midpoint distance of 22.9 mm (11 cats). Average disk-to-colliculus conduction distance was estimated to be 57.9 mm, a figure that combines the disk-to-chiasm distance of 22.9 mm with an average distance of 35 mm from the chiasm to the center of the colliculus (mean in eight cats, Ref. 52).

Retinal fibers in the cat conducting more rapidly than 20 m/s include nearly all of the Y-cell axons and few axons of other ganglion cell classes (10). Collicular responses mediated by such rapidly conducting axons should differ in latency by less than 1.2 ms after OD and OX shock and by less than 1.8 ms after OD and OT shock, given our electrode placements. Impulses in these axons evoked by stimulation at the OD should reach the colliculus in less than 2.9 ms and, assuming a synaptic delay of 0.5 ms, should activate collicular cells monosynaptically less than 3.4 ms after the OD shock. We therefore assume that a collicular unit response evoked more than 3.4 ms after OD shock but exhibiting an OD-OT latency difference less than 1.8 ms or OD-OX latency difference less than 1.2 ms reflects an indirect influence of retinal Y-cells. Using 25 m/s as a more conservative estimate of minimal Y-cell axonal conduction velocity reduces the maximal expected latency difference between OD and OX stimulation by only 0.2 ms and that between OD and OT by only 0.4 ms and does not substantially alter the interpretation of our results.

RESULTS

Ablation studies

In cats anesthetized with ketamine, electrical stimulation of the optic nerve or tract drives most cells in the deeper collicular layers (5). Typically, such stimulation evokes a burst of action potentials that occurs too late to be due to monosynaptic excitation by rapidly conducting axons but that begins only slightly earlier after OT shock than after OD shock. The small difference in latency together with the relatively long absolute latency of response indicate that the activation is initiated indirectly by the rapidly conducting axons of retinal Y-cells (5, 30). Following extensive ablation of occipital cortex (Fig. 1), deep-layer collicular cells were far less likely to be driven by stimulation of the OD, OX, or OT than were deep-layer units in intact cats. The reduction in retinal activation at latencies reflecting indirect Y-cell influence was particularly striking. Figure 2 illustrates this result for the subpopulation of deep-layer units that could be antidromically driven from the brain stem and will be called deep-layer output cells in this report. In intact cats, 84% (53/63) of such neurons could be driven at some latency by stimulation of the optic pathway and 75% (47/63) had latency behaviors reflecting indirect Y-cell activation. By contrast, in cats

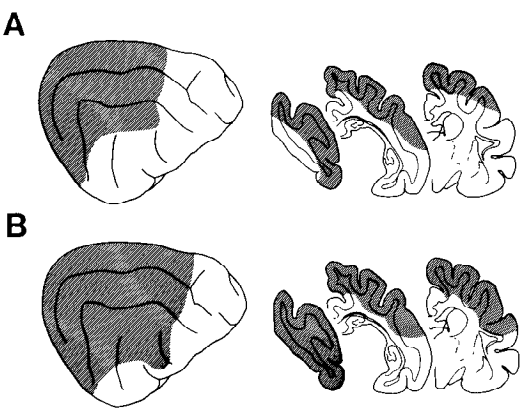

FIG. 1. Histological results from two representative experiments illustrating minimal (*A*) and maximal (*B*) extent of acute neocortical ablations made in the experiments of *Ablation studies*. Lesions (hatched areas) are shown on standardized drawings of three transverse sections through the neocortex (right) and, in reconstruction, on standardized lateral views of the hemisphere (left).

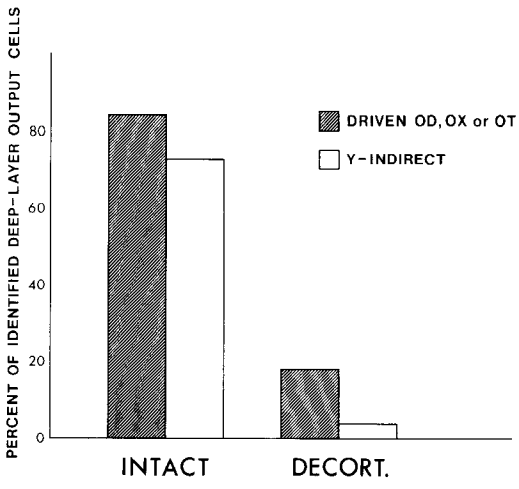

FIG. 2. Effects of acute lesions of the ipsilateral visual cortex on retinal input to deep-layer tectal cells antidromically activated from the brain stem ("deep-layer output cells"). Hatched bars: percentage of deep-layer output cells driven at any latency by stimulation of retinal fibers. White bars: percentage of output cells driven at latencies reflecting indirect Y-cell influence. These cells are a subset of those represented in the hatched bars. Left: animals with intact visual cortex; right: animals sustaining large acute ablations of the ipsilateral occipital cortex.

with cortical ablations, only 18% (9/50) of deep-layer output cells could be driven by stimulation of retinal fibers and only 4% (2/50) at Y-indirect latencies.

Similar effects of cortical ablation were obtained in our total sample of deep-layer cells, which includes units not driven antidromically from the brain stem. In animals with cortical lesions, only 10% (8/79) of cells in this larger sample had Y-indirect latency behaviors as compared to 69% (50/72) in a comparable sample from intact cats. Forty-two percent of the total sample of deep-layer neurons in animals with lesions could still be driven by electrical stimulation of the visual pathways but this measure is biased, since many of these cells were detected by their responses to such stimulation. The data of Fig. 2 are not subject to this bias because collicular output cells were detected by their antidromic responses to stimulation of the predorsal bundle.

Table 1 compares our results for deep-layer output cells with those obtained for cells of the stratum opticum. As described above, cortical lesions reduced the number of deep-layer output cells responding at Y-indirect latencies and rendered most of these cells unresponsive to stimulation of the visual pathways (Not Driven column, Table 1). However, in the stratum opticum many cells continued to respond to stimulation of the optic pathway after the ablation but now with latency differences exceeding those expected for Y-indirect activation (Other column, Table 1). These relatively large latency differences, ranging from 2.3 to 8.5 ms for OD-OT stimulation, seem likely to reflect the action of more slowly conducting retinal fibers, perhaps those of retinal W cells (30).

In the stratum opticum, units exhibiting the direct influence of retinal Y-cells were about as common in animals with lesions (22%; 5/23) as in intact cats (17%; 8/46). Fewer deep-layer output cells exhibited Y-direct input in cats with cortical ablation (8%; 5/60) than in intact animals (38%; 24/64). The Y-direct influences on deep-layer cells are tenuous, even under optimal conditions (5), so the reduced incidence of these responses may be related to nonspecific effects of the surgery. However, two observations make it unlikely that the deficit in the normally robust Y-indirect activation (Fig. 2, Table 1) arose entirely from generalized depression of collicular excitability. First, the majority of cells in the overlying superficial collicular layers (58/60; 97%) responded well to stimulation of optic nerve or tract fibers after occipital cortical ablation. This is in agreement with evidence from other studies that large ablations of the visual cortex do not eliminate, though they may modify, the responses of most superficial-

TABLE 1. *Effects of occipital decortication on collicular unit responses to stimulation of visual pathway*

	Y-Indirect	Other	Not Driven
	Cells of stratum opticum		
Intact	36 (87)	1 (2)	2 (5)
Decort	1 (5)	13 (68)	4 (21)
	Deep-layer output cells		
Intact	47 (75)	5 (8)	10 (16)
Decort	2 (4)	2 (4)	44 (88)

Y-direct responses excluded. Cells driven only from one site are not included, so totals for each row do not equal 100%. Values in parentheses are percentages.

layer cells to visual input (31, 33, 53, 64, 66, 88). Second, in recordings made in two animals from the colliculi contralateral to the cortical lesions, all of the deep-layer cells recorded (11/11) were driven by stimulation of the optic pathway and nearly all (10/11) had latency behaviors reflecting Y-indirect input. Thus, the reduction in Y-indirect responses was limited to the colliculus ipsilateral to the cortical lesions and was observed in tectal laminae containing cells that continued to respond to stimulation of other components of the visual pathway.

Cortical stimulation studies

The large lesions used in the first part of this study removed many distinct cortical areas interconnected by a complex network of association fibers (20, 22, 28, 40) and giving rise to multiple, parallel corticotectal projections (20, 22, 41, 86). To determine which of the visual areas might be involved in the Y-indirect route, we used focal intracortical stimulation to excite their efferents, while recording from cells in the deep collicular laminae. We restricted our survey to areas 17, 18, 19, and the posteromedial lateral suprasylvian area (PMLS), which are known, or seem likely, to receive input from the Y-cell component of the geniculocortical pathway (9, 11, 13, 21, 22, 32, 34, 35, 42, 44, 46, 47, 56, 65, 79, 82, 83, 87, 89).

CORTICAL ACTIVATION OF DEEP-LAYER NEURONS. Single electrical pulses to visual cortex evoked excitatory responses in many cells of the deep collicular layers, as has been reported for the hamster (62) and squirrel monkey (38); most of these neurons could be activated antidromically from the brain stem. The responses of one such deep-layer cell are illustrated in Fig. 3. The upper two traces show that, like most cells of the deep laminae (5), this unit was influenced indirectly by retinal Y-cell axons. Stimulation of retinal fibers produced a burst response that differed only slightly in onset latency after OD and OT stimulation and that had an absolute latency too long to be mediated by direct retinal Y-cell inputs. A single shock to the ipsilateral striate cortex evoked in the same cell a burst of action potentials at a distinctly shorter latency (2.0 ms, bottom trace). At higher stimulus currents, such cortical stimulation could provoke larger bursts of spikes,

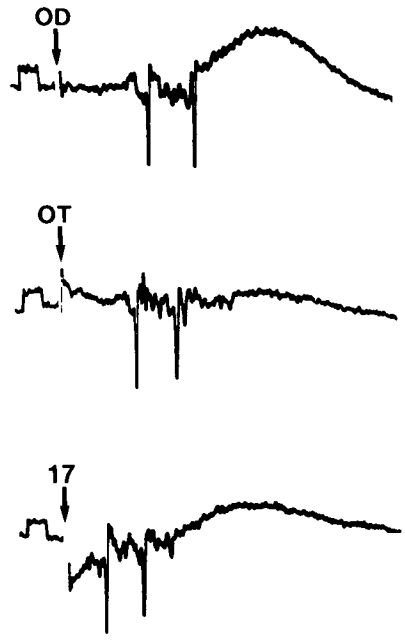

FIG. 3. Responses of a unit in the intermediate gray layer to electrical stimulation of the optic pathway and striate cortex. Single shocks to the contralateral optic disk (OD, top trace) evoked a burst of spikes beginning only 1 ms later than that following shock to the ipsilateral optic tract (OT, middle trace). The small-latency difference, together with the relatively long absolute latency (about 4 ms), reflects indirect activation by rapidly conducting axons of retinal Y-cells. Single shocks to the ipsilateral striate cortex (17, bottom trace) evoked a burst of spikes at a latency distinctly shorter than that elicited from retinal fibers. All stimulus pulses, 0.05 ms; current strengths: OD, 1.8 mA; OT, 0.2 mA; area 17, 1.3 mA. Positive-going square-wave calibration pulses at beginning of each trace are 200 μV, 1 ms.

as has been described for cells in the superficial collicular layers (51).

Stimulation of the extrastriate cortex produced similar short-latency excitatory responses in deep-layer collicular neurons. The traces of Fig. 4*A* and *B* illustrate the responses of a cell in the intermediate gray layer, which was driven from both area 18 and area 19.[1] The unit illustrated in Fig. 4*C* was driven

[1] Responses such as those of Fig. 4*A* and *B*, suggesting convergent inputs from several areas of visual cortex onto single deep-layer cells, were observed for all combinations of electrode placement. However, we did not systematically explore the question of convergence, since this would have involved distinguishing between effects mediated by corticocortical and corticotectal pathways, an undertaking beyond the scope of the present experiments.

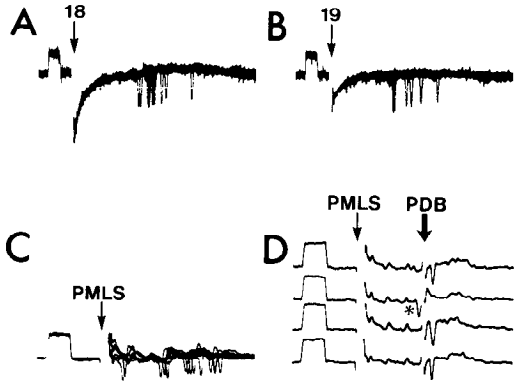

FIG. 4. Activation of deep-layer cells by stimulation of the extrastriate visual cortex. *A, B*: unit at border of intermediate white and deep gray layer (39) driven by intracortical stimulation both of area 18 (*A*) and area 19 (*B*). *C*: unit of intermediate gray layer orthodromically activated from area PMLS. Records in *A–C* show 10 superimposed traces. *D*: same unit as in *C*. Antidromic activation from region of predorsal bundles (PDB) and collision of antidromic spike with orthodromic action potential (asterisk; spike retouched) elicited from PMLS and falling within the critical interval. All stimulus pulses, 0.05 ms; current strengths, <2 mA for areas 18, 19, and PMLS; <1 mA for PDB. Positive-going square-wave calibration pulses at beginning of each trace are 500 μV, 1 ms in *A* and *B*; 300 μV, 1 ms in *C* and *D*.

at very short latency from area PMLS; like most deep-layer cells that could be orthodromically driven from the visual cortex, this unit was also activated antidromically from the predorsal bundle (Fig. 4*D*).

LATENCIES AND THRESHOLDS OF ACTIVATION. Figure 5 illustrates the distribution of minimum activation latencies for cells of the deep collicular layers (black bars) that responded to a standard intracortical stimulus pulse (50 μs, 2 mA, cathodal). The distributions of activation latencies for superficial-layer cells (white bars) recorded in the same experiments are included for comparison. Mean activation latencies from the cortex were shorter in deep-layer cells (4.0 ms from area 17, 4.0 ms from 18, 3.9 ms from 19, and 2.7 ms from PMLS) than in superficial-layer cells (5.0 ms from area 17, 4.9 ms from 18, 4.7 ms from 19, and 2.8 ms from PMLS), but this difference reached statistical significance only for stimulation in area 17 ($P < 0.05$, Mann-Whitney U test). Stimulation of area PMLS drove cells in deep and superficial layers at significantly shorter latencies than did stimulation in areas 17, 18, and 19 ($P < 0.01$, Mann-Whitney U test).

In the intervals between paroxysmal episodes of unresponsiveness induced by ketamine anesthesia (5, 55), single-pulse stimuli of 200–500 μA reliably activated deep-layer cells from each of the four areas of visual cortex tested. Unfortunately, because of the cyclic changes in excitability in this preparation, it was meaningless to define thresholds in the conventional way, i.e., as response probability within a fixed number of stimulus presentations. We note, though, that the minimum effective intensities, observed here for superficial- and deep-layer neurons, were only slightly higher than those required to drive superficial-layer neurons from areas 17, 18, and 19 in the unanesthetized pretrigeminal animal (51).

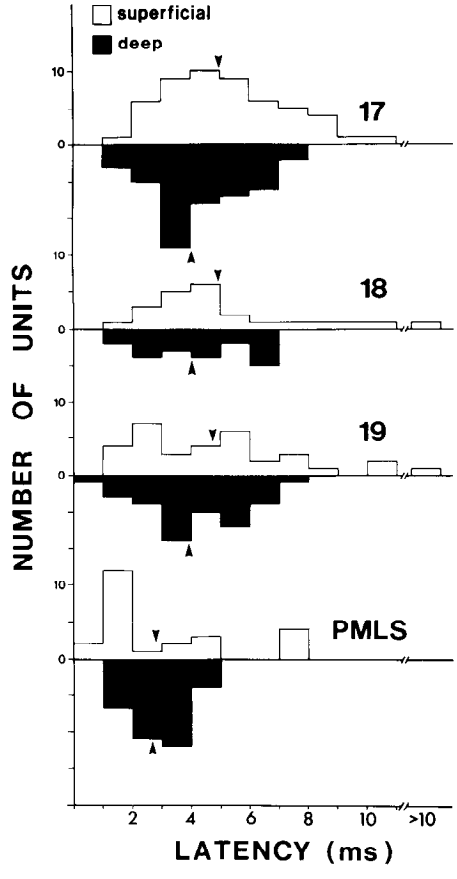

FIG. 5. Distribution of minimum latencies of activation of cells in the superficial (white bars) and deep collicular layers (black bars) after electrical stimulation of ipsilateral visual cortical areas 17, 18, 19, and PMLS. Mean for each distribution marked by arrowhead. (Note similarity between superficial and deep strata in latencies of activation from each area and especially short latencies of activation from PMLS.)

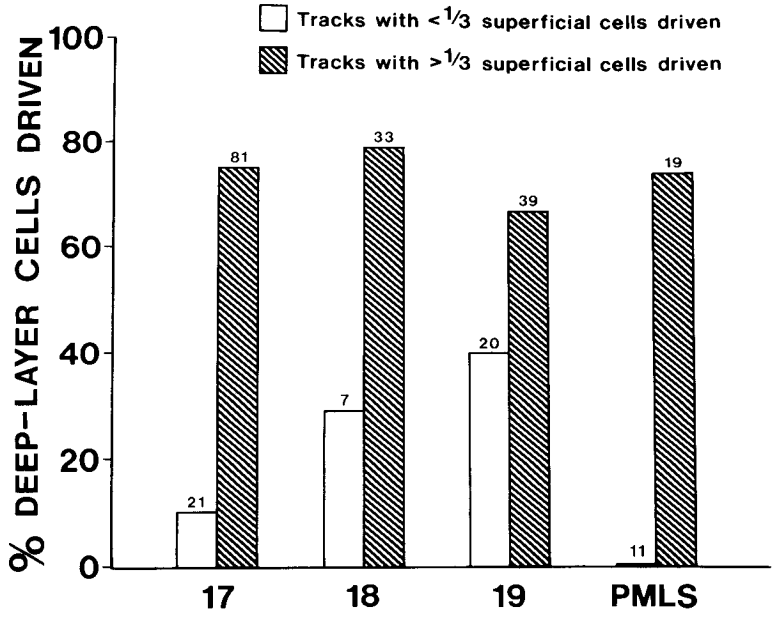

FIG. 6. Correlation of activation of deep-layer cells and overlying superficial-layer cells by stimulation at individual cortical sites. The number of superficial- and deep-layer units driven or not driven at 2 mA from individual cortical stimulus sites was determined for each recording track. At least four superficial layer cells were tested in each penetration. Tracks were sorted according to cortical area tested and according to whether more or fewer than one-third of the superficial-layer cells were activated. Data were then pooled for tracks in the several categories. White bars: percentage of deep-layer cells driven in penetrations in which fewer than a third of superficial-layer cells were driven from a given cortical electrode. Hatched bars: percentage of deep-layer cells driven in tracks in which more than a third of cells in the superficial layers were driven by the cortical stimulus. Shown over each bar is the number of deep-layer cells tested in that group. The distribution of cells between categories symbolized by the white and hatched bars differed significantly from chance for areas 17 and PMLS ($P < 0.001$, χ^2 test) and for area 18 ($P < 0.05$, Fisher exact probability test).

SPATIAL CORRESPONDENCE OF SUPERFICIAL AND DEEP-LAYER ACTIVATION. In individual recording tracts there was a clear correlation between the excitatory effects of a cortical stimulus on deep-layer cells and its ability to activate overlying superficial-layer neurons. This is illustrated in Fig. 6, which compares deep-layer neurons from penetrations in which more (Fig. 6, hatched bars) or fewer (white bars) than one-third of the superficial-layer cells were driven from a cortical site by single pulses of 0.05 ms, 2 mA. When a cortical stimulus drove more than a third of the cells in the superficial layers, on average about three-fourths of the deep-layer cells in that tract were activated from the same site. This correlation, which applied as well to the sample of deep-layer output cells, was observed for each of the individual cortical areas tested, although in the sample of cells studied during stimulation in area 19, it fell just short of being statistically significant ($0.05 < P < 0.10$, χ^2 test).

DISCUSSION

Y-indirect pathway

In a previous study, we found that retinal activation of deep-layer cells in the cat's superior colliculus is initiated predominantly by a multisynaptic pathway originating in retinal Y-cells; many of the tectal cells activated by this pathway were shown to be collicular output cells (5). The results reported here support the view that most of this retinal influence is cortically mediated, as was suggested by Hoffman (30) for the Y-indirect pathway reaching the superficial layers. Ablation of the occipital cortex reduced markedly the number of deep-layer cells that were driven from the visual pathways with latency patterns attributable to a polysynaptic Y-cell route (Fig. 2). Also, most cells of the deep collicular strata could be activated by stimulation of visual cortical areas known or suspected to receive input from the Y-cell component of the geniculocortical pathway. Geniculate Y-cell inputs to areas 17

and 18 are well documented (9, 32, 79, 83) and there is some evidence for similar input to area 19 (13, 42, 56). For the lateral suprasylvian areas, the question has apparently never been studied directly, but area PMLS, like areas 17, 18, and 19, is known to receive a substantial afferent projection from the medial interlaminar nucleus of the geniculate complex (22, 35, 44, 46, 65, 82) and perhaps also from magnocellular lamina C (44, 82), both targets of retinal Y-cells (7, 11, 14, 43, 48, 57, 89). Also, many units in PMLS respond to visual stimuli moving at high velocity, even after ablation of areas 17, 18, and 19 (75), a property suggestive of Y-cell input (10, 79). Finally, the latencies of activation of deep-layer collicular cells from visual cortex are compatible with cortical participation in the Y-indirect pathway. The polysynaptic Y-cell input drives collicular cells on average 7 ms after OD shock (5). The total transmission time from OD to cortical cells monosynaptically driven by geniculate Y-cells can be estimated to average about 3 ms (8, 9, 11, 13, 14, 19, 27, 32, 43, 73, 79, 83, 89). This would leave about 4 ms, on average, for corticotectal transmission and any additional synaptic delays, either intracortical or subcortical. Mean activation latencies for deep-layer cells from the cortical areas we tested ranged from 2.7 to 4.0 ms (Fig. 5). Thus, the average latencies of cortically evoked discharges in deep-layer cells are short enough to support a cortical role in the Y-indirect pathway. Our results do not indicate if one or more of the visual cortical areas tested are primarily responsible for relaying Y-cell signals to deep-layer collicular cells.

Are there direct inputs from visual cortex to deep collicular layers?

While fibers originating in areas 17, 18, and 19 terminate most densely in the stratum griseum superficiale (20, 41, 86), they do distribute a few axons deep to the stratum opticum (41). Axons from the lateral suprasylvian area project prominently to the stratum opticum, but nests of degenerating terminals have also been observed in the intermediate and deep gray layers following lesions in this cortical region (41). Segal et al. (71) have recently demonstrated retrograde labeling of cells in PMLS although not in areas 17, 18, and 19, with injections of tracers confined to the deep collicular layers. Taken together, these studies provide evidence for direct although sparse morphological links between each of the cortical areas we stimulated and the deep collicular strata. It is also possible that corticotectal axons contact the dendrites of deep-layer neurons in the stratum opticum (see, for instance, Figs. 13–16 in Ref. 36).

The results of the present experiments support the notion that at least some cells in the deep layers are contacted directly by axons from the visual cortex. Average latencies of activation following stimulation in a given cortical area were similar in deep-layer and superficial-layer neurons and were comparable to mean latencies of antidromic activation of corticotectal cells after stimulation in the superior colliculus (27; Table 1 of Ref. 51). Also, a substantial number of deep-layer cells could be driven at latencies as short as 2 ms, which is close to those observed in superficial cells activated from visual cortex in unanesthetized, midpontine pretrigeminal cats (51). It is noted, though, that average latencies of cortical activation for deep- and superficial-layer cells recorded here were about 1 ms longer than those observed for superficial-layer cells in the absence of anesthesia (51). This may reflect depressant effects of ketamine on the collicular cells.

A significant projection to the deep colliculus arises from the cortex on the crown of the suprasylvian gyrus (41), and one must ask if these fibers might have been activated inadvertantly in our experiments by the spread of stimulating current. The physical distance separating this pathway from our stimulus sites in areas 17, 18, and 19 make this an unlikely explanation for the deep-layer responses evoked from these regions. One can be less confident about this for stimulus sites in PMLS, which lies adjacent to axons descending from the suprasylvian crown. In several instances, deep-layer neurons were activated by stimulation in PMLS at intensities as low as 200 μA and from sites confirmed histologically to be in gray matter. Stimuli of this intensity and lasting only 0.05 ms are unlikely to excite axons traveling 0.5–1 mm away in the white matter of the suprasylvian gyrus (2, 80), but the possibility cannot be dismissed entirely. Thus, our interpretation of the effects of electrical stimulation in PMLS, while consistent with what is known about the con-

nections of this region with the lateral geniculate and the superior colliculus, must be considered tentative until the possibility of current spread to adjacent white matter can be definitely excluded.

Implications for effects of cortical lesions

In cats, lesions of the striate cortex leave many deep-layer cells responsive to visual stimulation (31, 33, 53, 64, 78), whereas similar lesions in monkeys are reported to eliminate visual responses in deep-layer neurons (70). Striate cortex lesions also leave much of the extrastriate cortex (including the lateral suprasylvian areas) visually responsive in the cat (12, 42, 72, 75), but eliminate most visual responses in the extrastriate cortex of the monkey (69). These observations undoubtedly reflect the fact that extrastriate projections of the lateral geniculate nucleus are far more prominent in the cat than in the monkey (3, 90). Since electrical excitation of extrastriate areas in the cat activates deep tectal neurons, these areas could mediate those visual responses that survive striate cortex ablation in this animal. In the monkey, on the other hand, ablation of striate cortex effectively deprives extrastriate cortex of relayed geniculate input so visual activity no longer has access to the deep colliculus by a cortical route.

Consistent with this view in a report by Hoffmann and Cynader (31) that larger cortical lesions involving the lateral suprasylvian areas eliminate deep-layer responsiveness to visual stimulation in the cat. However, we observed that some retinal influence persists in the cat following such lesions (Fig. 2 and Table 1), so the deep tectum is not totally dependent on occipital cortex for visual input. In addition to a sparse direct retinal projection to the deep layers (5, 18), pathways involving the pretectum, ventral lateral geniculate, and perhaps even the superficial layers may provide this residual excitation (4, 16, 17, 24, 37, 45).

Retinotopic organization

Cells in the superficial layers of the cat's superior colliculus tend to be driven by low-intensity stimulation in visual cortex if their receptive fields overlap those of cortical neurons near the stimulating electrode (50, 51). Since cells in a given electrode penetration through the superficial layers usually have overlapping receptive fields, the probability is high that a cortical stimulus that drives some of them will also drive the others. Cells of the deep laminae tend to have large receptive fields that contain the smaller fields of overlying superficial-layer cells (23). Thus, a cortical site related retinotopically to cells recorded above the stratum opticum should be similarly related to cells recorded in the deeper laminae in that same track. We have shown here that stimulation at a particular cortical site tends to drive deep-layer cells if neurons in the overlying superficial layers are also activated from the same cortical electrode. This suggests that functional cortical connections to the deep collicular strata obey visuotopic constraints similar to those that govern corticotectal inputs to the superficial layers. The morphological substrate of this visuotopic relationship between superficial and deep collicular cells is not apparent, since the direct visual cortical projections to the deep layers are said not to preserve the spatial order of the projections to the superficial layers (41) and a direct link between superficial and deep layers is not established (15).

It should be emphasized that our evidence for visuotopic order in the cortical projections to the deep tectal laminae is indirect, particularly as it relates to the projection from PMLS. The conditions of these experiments precluded a direct comparison of the receptive fields of neurons at stimulation and recording sites. In any case, it is unlikely that the geometry of the functional connections between PMLS and deep tectum will fit neatly into a conventional point-to-point frame of reference, since the representation of the visual field in this cortical area appears to be fundamentally different from that of areas 17, 18, and 19 (58, 84, 85). Even for areas 17, 18, and 19, the visuotopy of connections to the superficial layers has been shown not to be point to point when viewed at the cellular level. Neighboring neurons with dissimilar receptive fields are functionally related to cortical areas of different size. What is preserved in these projections is the connection between cortical and tectal neurons whose receptive fields share a common point in visual space (51). Perhaps detailed analysis of the corticotectal projections from

the lateral suprasylvian areas will disclose a similar property.

What does deep tectum do?

These results support the venerable but recently disputed (15) view that the deep layers of the cat's superior colliculus are closely linked to the visual system. Cells in these laminae also discharge in close association with movements, particularly those that orient the head and eyes toward external stimuli (26, 59, 81). This characteristic of the deep tectal layers finds its purest expression in the primate, where visual inputs seem neither necessary or sufficient for most of the cellular discharges that accompany saccadic eye movements (49, 54). In the cat, though, collicular cells that send their axons into the brain stem are the targets of effective visual inputs, especially those arising from retinal Y-cells and relayed in one or more areas of visual cortex. Moreover, destruction of the superior colliculus in this animal eliminates eye movements evoked by electrical stimulation of the occipital lobe (76; see also Ref. 67), which suggests that the visual cortex is linked to the oculomotor system through the superior colliculus. From our own observations (e.g., Fig. 4*A, B*) it appears that the several corticotectal projections may converge onto the same deep-layer cells, as if they share control of a common output system. Does the collicular circuitry perform some common service or services for all of its cortical masters? One obvious function would be to transform spatial information, received in what are probably several different formats, into a common code appropriate to the organization of the target motor structures of the brain stem and spinal cord. The deep colliculus could mediate a similar transformation of somatosensory and auditory information that reaches it. The idea that the tectum is a site of sensorimotor transformation is not new (1, 29, 61, 77) but it has fallen into disfavor, largely because deep tectal cells in the primate appear generally to have inconsequential visual responses (49, 54, 74, 91). This may mean simply that in the highly encephalized primate brain, the tectal output systems are buffered against direct sensory commands by more elaborate control structures than exist in other vertebrates. Our present and previous findings (5) and observations of others (23, 26, 59, 81) suggest that the cat's deep colliculus may yet provide a useful model of how neural tissue converts sensory information into a signal appropriate for motor control.

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