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Retinal Y-Cell Activation of Deep-Layer Cells in Superior Colliculus of the Cat

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SUMMARY AND CONCLUSIONS

1. Visual inputs to layers of the superior colliculus (SC) beneath the stratum opticum have been studied in ketamine-anesthetized cats by recording extracellular unit responses to electrical stimulation of the optic pathway.

2. In most cells (83%), single shocks to the contralateral optic disk (OD) or ipsilateral optic tract (OT) elicited a burst of 1–3 spikes beginning 3–10 ms ($\bar{x}$ = 6.1) after OD stimulation and 2–9 ms ($\bar{x}$ = 5.2) after OT shock. The small latency difference appears to reflect mediation by rapidly conducting Y-cell axons, but the absolute latencies indicate that these axons do not synapse directly on the recorded cells and may not proceed directly to the colliculus ("Y-indirect pathway").

3. In a smaller number of deep tectal cells (34%), this burst was preceded intermittently, but at a consistent latency, by a single spike that followed OD shock by 1.1–3.4 ms ($\bar{x}$ = 2.4) and OT shock by 0.8–2.5 ms ($\bar{x}$ = 1.4). The small latency difference and short absolute latencies suggest that a component of the retinotectal Y-cell projection may make monosynaptic contact with deep-layer cells ("Y-direct pathway").

4. This short-latency excitation of deeper layer cells survived transection of the IIIrd, IVth, ophthalmic Vth, and VIth cranial nerves as well as midpontine pretrigeminal brain stem transection. Hence, the early spike following OD stimulation could not be attributed to inadvertent activation of non-visual fibers in the orbit.

5. Cells with apparent Y-direct input, as well as those with Y-indirect input, were found throughout the deeper collicular layers. Most of these cells could be antidromically driven from the region of the predorsal bundle.

6. Thus, the present findings demonstrate a major retinal Y-cell influence on output cells in the deep layers of the cat's superior colliculus. Part of this influence may be mediated by a direct, monosynaptic projection from the retina.

INTRODUCTION

Within the superior colliculus, a clear contrast is evident between the marked visual responsiveness of superficial-layer cells (stratum opticum and above) and the polymodal and premotor properties of neurons in the deeper layers (below the stratum opticum; for recent reviews see Refs. 26, 75, and 84). Nonetheless, many deeper layer cells can be shown to possess visual receptive fields and, because some of these neurons appear to participate in the control of visually guided saccadic eye movements (53, 60, 62, 68, 70, 74, 85), the sources of their visual inputs are of considerable interest.

Direct retinal projections to the deep strata have not been observed in anatomical studies, but retinotectal axons terminate heavily in the stratum griseum superficiale and to a lesser extent in the stratum opticum (7, 14, 29, 33, 37, 39, 78, 80). In a physiological study of the cat, Hoffmann (35) identified two classes of collicular cells receiving direct retinal input. One class, found throughout the superficial layers, receives direct input from the slowly conducting axons of retinal W-cells (W-direct pathway). The second class, lying in the lower half of the stratum griseum superficiale and in the stratum opticum, receives monosynaptic in-

put from rapidly conducting Y-cell axons (Y-direct pathway). In addition to these direct retinal projections, superficial layer cells receive input from visually responsive areas of the cortex and brain stem (8, 20, 21, 25, 28, 30, 40, 54, 81).

Thus, one possible source of visual information for the deeper layers is the superficial tier of the colliculus itself. This idea is reinforced by the striking spatial correspondence of the visual receptive fields of superficial- and deep-layer cells and by the congruence of the retinotopic sensory map of the upper strata with the retinocentrically organized "motor map" of the deep layers (2, 60, 67, 68, 70, 85). Despite the plausibility of this connectional scheme, there is no compelling anatomical or physiological evidence for direct communication between the superficial and deep strata (19, 53).

Descending projections linking visual areas of the cortex with the deep tectal laminae (8, 25, 40, 81) provide a second possible source of retinal information for the deep cells. In monkeys (71) and squirrels (58), destruction or inactivation of the visual cortex are reported to eliminate the visual excitability of cells below the stratum opticum. A similar result has been observed in the cat (77), but other investigators report persistence of visual responses in the deep layers after massive lesions of the visual cortex (36, 56, 59, 66). For the cat, then, a crucial role of the corticotectal pathways in deeper layer visual responsiveness remains in doubt.

In the present study, we have used an electrophysiological approach similar to that of Hoffmann (35) to investigate visual inputs to the deep collicular layers. We have carried out these experiments in cats anesthetized with ketamine, for we have found that deep collicular cells respond well to visual stimulation in this preparation. The results reveal a substantial retinal Y-cell influence on the deeper collicular strata. For the most part, this influence is mediated polysynaptically, but some deep-layer cells may receive monosynaptic input from retinal Y-cells.

METHODS

Preparation

Animals in the main group of experiments received an initial dose of 30 mg/kg ketamine, ip, supplemented intravenously as required to suppress spontaneous limb movements. No paralytic agents were used and adequacy of anesthesia could easily be monitored. Salivation was controlled with a single dose of atropine sulfate (0.04 mg/kg, im). The corneas were protected with plano contact lenses to prevent them from drying, and rectal temperature was maintained automatically at 38°C. The cat was placed in a stereotaxic instrument and portions of the skull were removed to provide access to the right superior colliculus and optic tract and left midbrain tegmentum. Scalp edges were sutured to a brass ring and the exposed cortex covered with a pool of warm mineral oil.

A general indication of the direction of gaze was obtained by back projecting the optic disk of the left eye onto a tangent screen (22). The visual responsiveness of collicular units was informally tested using projected stimuli or dark targets on wands. Receptive fields of tectal cells and response fields of juxtazonal potentials (55) provided a rough idea of the location of the recording electrode within the retinotopic collicular map, but systematic receptive-field analysis was not attempted. Precise mapping was precluded in any case by slow drifts in eye position that occurred in this preparation. Nystagmus, which may be observed in cats lightly anesthetized with ketamine (42; unpublished observations), was not apparent at the plane of anesthesia used here.

Electrical stimulation

A pair of bipolar concentric electrodes with tips separated 1–2 mm was positioned in the left predorsal bundle, about 2 mm posteroventral to the trochlear nucleus and 0–1.5 mm from the midline. The depth of this electrode pair was adjusted to yield consistent antidromic responses in deeper layer collicular cells. Action potentials were judged as antidromic on the basis of short, stable response latencies and the collision test; high-frequency stimulus trains evoked body movements and were not used. A single bipolar stimulating electrode was placed in the right optic tract (OT) at Horsley-Clarke coordinates anterior 8.0–9.0 and lateral 9.0–10.0. Visually evoked activity recorded through the electrode permitted its accurate placement in the OT. Another concentric bipolar electrode was inserted into the left eye through a scleral slit and positioned in the optic disk (OD) under ophthalmoscopic guidance. In two cats a similar electrode was also placed in the right OD. Adequate placement of the OT and OD electrodes was confirmed in all cases by recording field potentials evoked at one electrode from stimulation through the other. Stimulating electrodes were connected to a constant-current stimulator and current strength was monitored by measuring the

voltage drop across a 1,000-Ω resistor in series with each electrode. Single current pulses of 0.05 ms duration and less than 5 mA were generally used.

Recording

Unit activity in the superior colliculus, recorded with extracellular tungsten microelectrodes (5- to 15-MΩ impedance at 1,000 Hz), was amplified and monitored by conventional methods. Recording band pass was varied as required to minimize shock artifact. Traces of interest were preserved on FM tape. The electrode was lowered until it contacted the collicular surface, as signaled by the appearance of juxtazonal potentials or a prominent negative evoked potential (55), and the electrode was then advanced an additional 0.5 mm. Beyond this point in the penetration, every unit encountered was characterized as to its response to stimulation at the OD, OT, and brain stem electrodes. Minimum latencies from OD and OT shock to unit responses of interest were determined from 10 or more successive stimulus presentations. Latencies were measured from the onset of the stimulus artifact to the foot of the action potential. One or more small electrolytic lesions were placed in each recording track.

Data on relative frequency of particular classes of deep-layer unit responses (Tables 1 and 2) were obtained from seven cats. In these, special care was taken to advance the recording electrode slowly and to alternate frequently between stimulus sites to maximize the chances of detecting collicular cells having low spontaneous activity. Units with waveforms characteristic of axons (10) were excluded from the sample.

We frequently observed in ketamine-anesthetized animals a rhythmic waxing and waning of unit activity (1–2 Hz) in the superior colliculus. This periodic suppression is presumably related to the rhythmic, synchronous activity observed in the neocortex and hippocampus in this preparation (61), but we have not investigated this directly.

Histology

At the end of the experiment, the animals were given an overdose of pentobarbital and perfused through the carotid arteries with 10% Formalin. Electrode tracks were reconstructed from 100-μm frozen sections stained with cresyl violet, and the laminar location of each recorded unit was estimated. For purposes of reporting, units have been assigned either to the superficial or to the deep collicular strata (respectively, laminae I–III or IV–VII of Kanaseki and Sprague, Ref. 39). The positions of stimulating electrodes in the OT and brain stem were verified. The average conduction distance between OD and OT electrodes was estimated to be 38.5 mm, a figure arrived at by combining the average measured distance from the OT electrode to the midpoint of the chiasm (13.5 mm) and an average disk-to-chiasm midpoint distance of 25 mm (J. T. McIlwain, unpublished observations). Average disk-to-colliculus conduction distance was estimated to be 60 mm, a figure that combines a disk-to-chiasm distance of 25 mm with the average distance of 35 mm from the chiasm to the center of the colliculus measured in eight cats (55).

Control experiments

In one animal the left IIIrd, IVth, ophthalmic Vth, and VIth cranial nerves were transected at their point of entry into the orbit by means of a ventral approach. Following surgery, the left eyelids were sealed to prevent corneal drying and the animal was allowed to survive for 3 days prior to recording. Eye movements and corneal reflex were abolished and mydriasis was evident on the left side; the right eye appeared normal. Stimulation and recording were as described above. The completeness of the transection was verified by postmortem dissection of the nerves.

In another animal, trigeminotectal pathways were eliminated by midpontine, pretrigeminal transection of the brain stem (6) under halothane on the day of recording. Artificial respiration was administered and anesthesia discontinued. Stimulation and recording procedures were as described above except that the predorsal bundle was not stimulated.

RESULTS

Most deep-layer cells (70/80; 88%) could be driven by electrical stimulation of the ipsilateral optic tract or contralateral optic disk. Their responses were similar to those observed within the lower stratum griseum superficiale and stratum opticum. The most typical response (83% of deeper layer cells) was a burst of one to three spikes beginning about 3–10 ms ($\bar{x} = 6.1$) after OD stimulation and about 2–9 ms ($\bar{x} = 5.2$) after OT stimulation. We shall refer to this as the "late burst" to distinguish it from an earlier response seen in some units. In about one-third of the recorded deep-layer cells (27/80), the late burst was preceded intermittently, but at a consistent latency, by a single "early spike." Figure 1*A* and *B* shows the early spike and late burst elicited by OD and OT stimulation in a cell of the intermediate gray layer. The latency to the early spike in this unit was extremely short—about 2.3 ms from the OD (Fig. 1*A*) and 1.2 ms from the

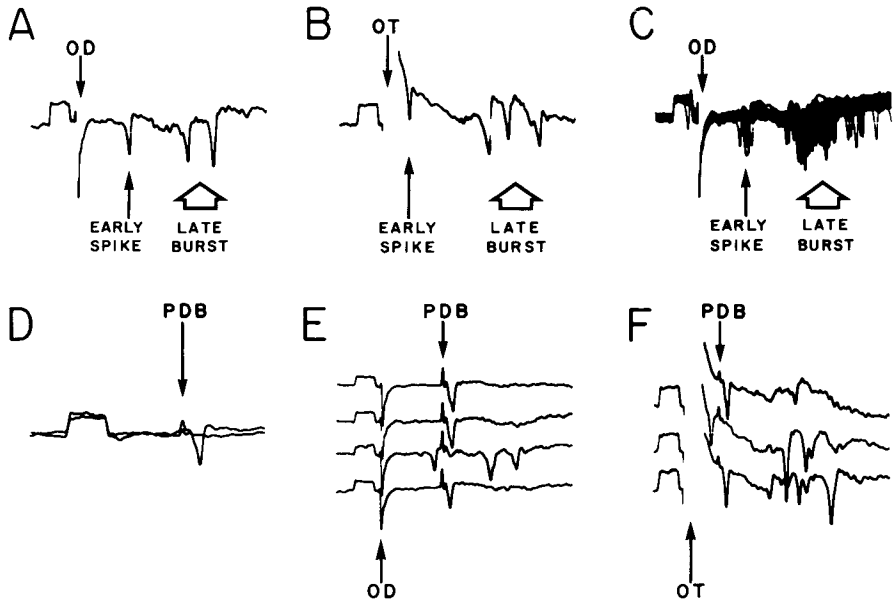

FIG. 1. Responses of a unit in the intermediate gray layer to electrical stimulation of the optic pathway and predorsal bundle. *A*: single short-latency early spike and subsequent late burst of spikes evoked by single shock to contralateral optic disk (OD). Spikes of the late burst were often superimposed on irregular slow potentials and low-amplitude multiunit activity, sometimes obscuring a unit action potential clearly isolated as the early spike and by antidromic activation (e.g., *D–F*). *B*: early spike and late burst evoked at shorter latency from ipsilateral optic tract (OT). *C*: fifty superimposed traces showing variable latency of early spike, evoked here by OD stimulation at 1 Hz. *D–F* illustrate antidromic activation of the same unit from the predorsal bundle (PDB). *D*: all-or-none nature of spike elicited at short latency from near-threshold PDB stimulation. *E, F*: collision of antidromic spike elicited from PDB with orthodromic early spike evoked from OD (third trace in *E*) or from OT (middle trace in *F*). All stimulus pulses, 0.05 ms; current strengths, 4.6 mA for OD; 3.2 mA, for OT; and less than 0.3 mA for PDB stimuli. Calibration pulses in all records, +200 μV, 1 ms.

OT (Fig. 1*B*)—and showed considerable variability (Fig. 1*C*), suggesting that the response was synaptically mediated. Despite the relatively large distance separating the two stimulus sites, the shift in latency was small (about 1 ms), reflecting activation by rapidly conducting optic axons. Stimulation of the predorsal bundle elicited a single spike at a short and stable latency suggestive of antidromic activation (Fig. 1*D*). The antidromic origin of this response was confirmed by its collision with the short-latency orthodromic spike elicited from either the OD or OT (Fig. 1*E, F*).

Relative frequency of deep-layer cells exhibiting short-latency optic inputs

Cells exhibiting an early spike following OT or OD stimulation appeared to be at least as common in the deeper collicular layers (34%) as in the superficial layers (18%) (Table 1). The unexpected occurrence of these responses in the deep strata and their relative lability (see below) prompted us to observe deeper layer units for longer periods of time than we routinely allotted for superficial cells. This may account for the somewhat higher relative frequency of early spikes recorded in the deep as compared to the superficial cell samples. Optic tract shock elicited the early spike more reliably and in a greater number of deep-layer cells than did OD shock. Of 27 deep-layer cells exhibiting an early spike, 26 (96%) were driven at this short latency from the OT, but only 18 (48%) from the OD (Table 1). Of cells that could be driven antidromically from the predorsal bundle, more than a third exhibited an early spike after OT stimulation. Half of these units could also be activated at short latency from the OD (Table 1). Except for one unit, which lay in the

TABLE 1. *Laminar distribution and relative frequency of cells exhibiting an early spike*

	Site of Effective Stimulation				
	OD only	OT only	Both	Neither	Total
Superficial layers*	1 (1)	2 (3)	11 (14)	66 (82)	80 (100)
Deep layers†	1 (1)	14 (18)	12 (15)	53 (66)	80 (100)
Antidromic from PDB	0 (0)	15 (19)	15 (19)	50 (62)	80 (100)

Values in parentheses are percentages. PDB, predorsal bundle. * Lower stratum griseum superficiale and stratum opticum. † Stratum griseum intermediale and below.

stratum opticum, all units driven antidromically from the predorsal bundle were located in the deep collicular layers.

Conduction velocity of afferents mediating early spike

Figure 2 illustrates the distribution of minimum latencies to the early spike for all cells driven at short latency by OD or OT stimulation. Among deep-layer cells (Fig. 2, black bars), the average latency to the early spike was 2.4 ms (range, 1.1–3.4 ms) after OD and 1.4 ms (0.8–2.5 ms) after OT stimulation. There was no significant difference between these values and those we observed in the superficial layers (Fig. 2, open bars; $P > 0.05$, Mann-Whitney U test). Our latency values correspond closely to those reported by Hoffmann (35) for a population of superficial-layer cells receiving apparent monosynaptic input from retinal Y-cell axons. His Y-direct cells were driven on average at 2.0 ms (1–4 ms) after OD shock and 1.2 ms (0–2.5 ms) after OT shock. For the combined superficial and deep samples shown in our Fig. 2, the mean latency to the early spike from the OD was 2.2 ms (1.1–3.4 ms) and from the OT was 1.4 ms (0.8–2.5 ms).

In our combined sample, the average response latency after OD and OT stimulation differed by 0.8 ms. Dividing this into the average estimated OD-OT conduction distance (38.5 mm) yields 48 m/s as the average conduction velocity of the fibers mediating the early spike. Note that errors as great as 5 mm in the estimation of conduction distance alter the conduction-velocity estimate by only 6 m/s. Thus, there is no doubt that these fibers are among the most rapidly conducting of the optic nerve and tract (9, 11, 15, 23, 79).

Further support for the notion that Y-cell afferents mediate the early spike in deep-layer cells is provided in Fig. 3*A*, which gives individual OD-OT latency differences for 17 units driven at short latency from both sites. At the top of Fig. 3 are indicated the latency shifts expected from responses mediated by W-, X-, and Y-cell pathways, activated extraretinally at our OD-OT electrode separation. The expected values were calculated from data of Cleland and Levick (15), who report mean extraretinal conduction velocities for the three axon classes as well as the fastest and slowest velocities observed in each class. It is evident from Fig. 3 that the latency shift for virtually all cells in the sample fell within the range predicted for Y-

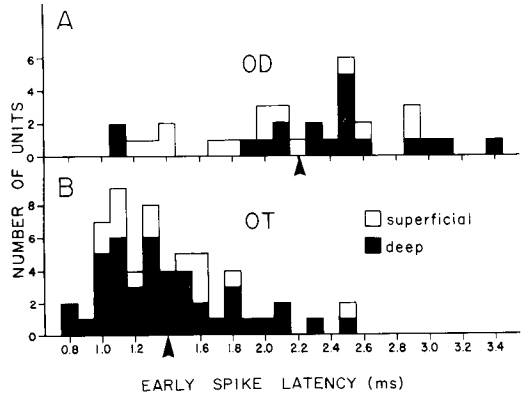

FIG. 2. Distribution of minimum latencies of early spike evoked from optic disk (*A*) or optic tract (*B*). Latencies measured from onset of shock artifact to foot of shortest latency early spike in 10 or more stimulus presentations. Superficial-layer cells (open bars) recorded more than 0.5 mm below surface, but above intermediate gray; deep-layer cells (black bars) recorded below stratum opticum. Arrowheads indicate mean latencies; superficial and deep samples are lumped. Note shift of mean latency of about 0.8 ms.

cells, and the mean shift (0.84 ms) was very close to that expected for a Y-cell input (0.74 ms).

Thus, the early spike evoked in deep-layer cells appears to be mediated by a retinal Y-cell pathway. The short absolute latencies of the responses suggest that the retinal fibers proceed directly to the colliculus, as do those of the Y-direct pathway of Hoffmann (35). We will discuss later the possibility that the early spikes in deep-layer cells are mediated by monosynaptic contacts from this Y-cell projection.

Factors influencing efficacy of fast retinotectal inputs

The probability of occurrence of the early spike in tectal cells could sometimes be increased by the use of paired shocks (separation, 0.1–0.4 ms) or by concurrent sensory stimulation (Fig. 4*A*). The latter usually consisted of vigorously waving visual stimuli (a hand or black cardboard disk) in front of the animal, although auditory and somatosensory stimulation was occasionally found to be effective. The sensory stimuli were given in a random pattern and never produced apparent early spikes in control sweeps when electrical stimulation was omitted. Presumably, such sensory stimulation increased the base-line excitability of the recorded cells and permitted excitatory synaptic effects of the fast direct inputs to exceed threshold.

Deep-layer cells often appeared to habituate rapidly to visual stimuli, and it was frequently easier to evoke early spikes when no shocks had been delivered for several minutes. In some cells, periods in which early spikes were reliably elicited could be separated by intervals of many minutes during which only the late burst was observed. This periodic waning of early-spike responsiveness was easily distinguished from the transient suppression of all unit activity observed during episodes of ketamine-induced hypersynchrony (61).

As noted above, the early spike was usually more readily elicited by OT than by OD stimulation. To determine if this was due to the activation of binocular inputs at the OT electrode, we tried stimulating both optic disks, either simultaneously or at various pairing intervals. We never found stimulation of the ipsilateral OD to evoke an early spike or to increase noticeably the probability with which the early spike was elicited from the contralateral OD. Ipsilateral stimulation did, in some cases, enhance the late burst.

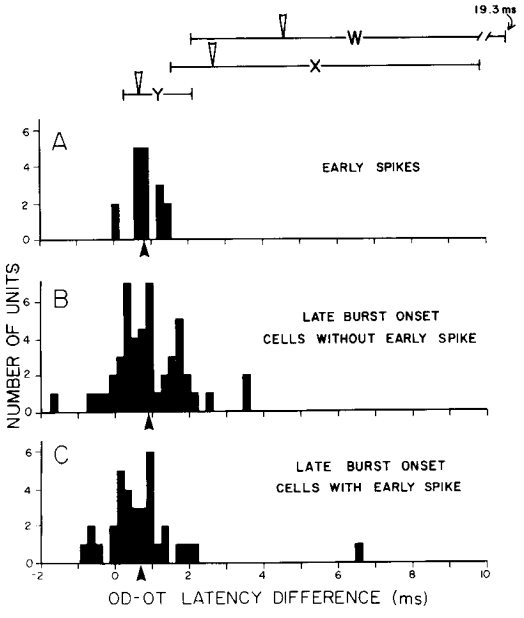

FIG. 3. Distributions of OD-OT latency shifts for the early spike and late burst of individual deep-layer cells (below) and ranges of shifts expected for inputs mediated by retinal Y-, X-, and W-cell axons (above). In each histogram, data are drawn from cells recorded below stratum opticum that were driven from both OD and OT. Latency differences were determined for single units by subtracting the minimum OT latency from the minimum OD latency. Histograms show distribution of such latency shifts for early spike (*A*), for onset of late burst in cells exhibiting no early spike (*B*), and for late burst onset in cells exhibiting an early spike after OD or OT shock (*C*). Filled arrowheads indicate mean latency shifts. Above: means (open arrowheads) and ranges (horizontal lines) of latency shifts expected for tectal unit responses mediated directly or indirectly by retinal Y-, X-, or W-cell axons. Values determined by dividing average electrode separation in present study by fastest, slowest, and mean conduction velocities for each fiber population (15). Latency shifts for early spike and late burst onset fall predominantly in Y-cell range. Note close correspondence of observed mean latency shift in each distribution to expected mean for Y-cell input.

Do fibers of retinal origin mediate early spike?

Not all axons in the optic tract arise from retinal ganglion cells. For instance, axons from cells in the parabigeminal nucleus des-

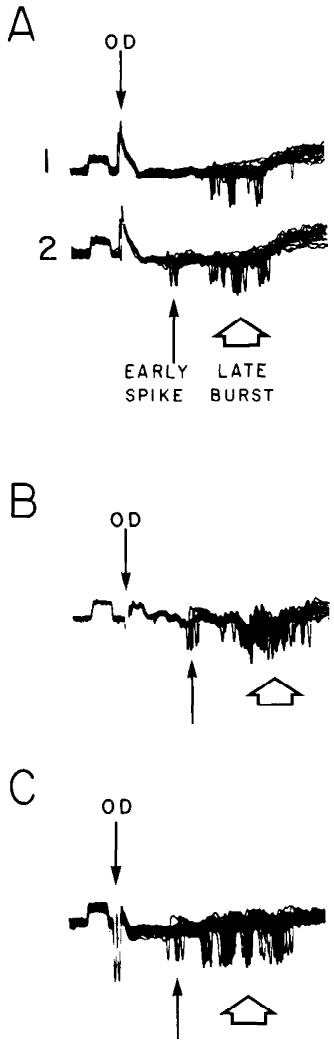

FIG. 4. *A*: facilitation of early spike in intermediate gray-layer cell by concurrent visual stimulation. In A_1, no visual stimulation is used; shock to optic disk (0.05 ms, 3.0 mA) elicits only late burst. Twenty superimposed traces. Calibration pulse, +100 μV, 1 ms. A_2 same as A_1 except that concurrent asynchronous visual stimulation is used. Note occurrence of early spike. *B, C*: persistence of early spike and late burst evoked from optic disk after interruption of nonretinal sensory inputs to the colliculus from the orbit. Record in *B* from intermediate gray of unanesthetized cat with complete transection of the brain stem at pretrigeminal level. Twenty superimposed traces; single pulse to OD (1 Hz, 0.05 ms, 4.2 mA). Record in *C* taken from intermediate gray-layer unit in a cat with complete transection of IIIrd, IVth, ophthalmic Vth, and VIth cranial nerves. Forty-seven superimposed traces. Early spikes evoked by double pulse to OD (separation, 0.3 ms; duration, 0.05 ms; 4.2 mA). Calibration in *B* and *C* is 1 ms, +200 μV.

tined for the contralateral superior colliculus traverse both optic tracts and the supraoptic commissure (30). Thus, the early spike elicited in collicular cells from OT stimulation could reflect activation of these or possibly other nonretinal axons coursing in the optic tract. For this reason, the finding that OD stimulation evokes the early spike is key evidence for its origin from a retinotectal input. On the other hand, the deep layers of the superior colliculus are known to receive nonretinal sensory input from the orbit, including a sizable proprioceptive input from the extraocular muscles (1, 5, 17). Therefore, it was important to show that current spread from the OD electrode to these nonretinal sensory fibers could not account for the early spike.

In one animal we completely transected the brain stem just rostral to the entry of the trigeminal nerve. Because virtually all extraocular proprioceptive input is thought to enter the brain stem through the Vth, and to a lesser extent VIth, cranial nerves (3, 4, 48, 49), this transection presumably interrupted these proprioceptive inputs to the superior colliculus as well as all other trigeminotectal pathways. Nonetheless, deep-layer cells driven at short latency from the disk in this preparation (Fig. 4*B*) were at least as numerous as in the main group of experiments. Ten units recorded below the stratum opticum could be reliably driven from the OD at latencies under 4 ms, implying that the responsible afferents conduct in the Y-cell range (15, 24, 35). Eight of these cells were also driven at short latency from the optic tract.

Any sensory fibers coursing into the brain over the IIIrd or IVth cranial nerves would not have been eliminated in this pretrigeminal control animal. Since a small number of extraocular proprioceptive fibers may enter over the IIIrd nerve in some species (50), we cut in one animal the IIIrd, IVth, and VIth cranial nerves and the ophthalmic branch of the Vth at their point of entry into the orbit. During recording from this cat, an early spike was elicited by OD stimulation in two intermediate gray-layer cells. One of these cells was also driven at short latency from the OT electrode and could be antidromically driven from the brain stem (Figs.

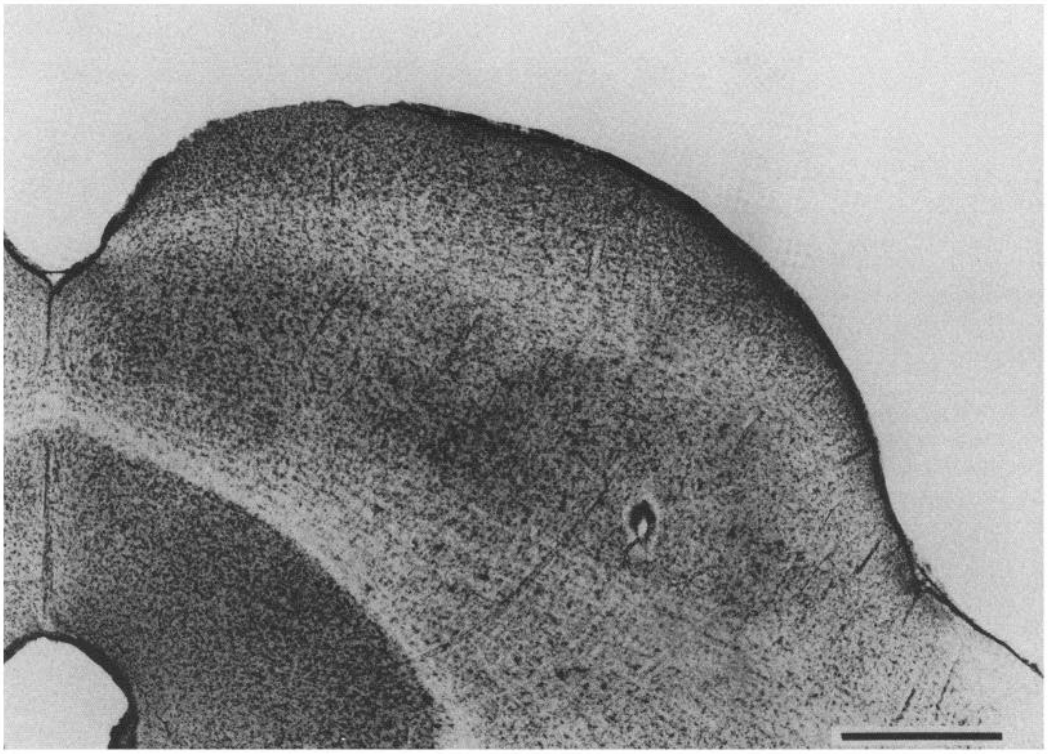

FIG. 5. Nissl-stained transverse section through the superior colliculus. Lesion in the intermediate gray layer marks site at which recording in Fig. 4*C* was made. Scale line, 1 mm.

4*C*, 5). These observations leave little doubt that the early spikes in deep tectal cells are at least partly attributable to activation of retinofugal axons.

We note that cells with early spikes were more difficult to find in the nerve-sectioned animal than in the main group or pretrigeminal control animal. This could mean that nonretinal sensory fibers in the orbit do enter the brain over the IIIrd or IVth cranial nerve and that activation of these fibers accounts for some of the short-latency tectal activation from the OD. On the other hand, this would not explain why the early spike was also more difficult to elicit from the OT site in this animal. It seems more likely that the difficulty in eliciting the early spike is attributable to trauma, ischemia, or residual barbiturate effects resulting from surgery. Any such residuum would be especially likely to affect the tenuous functional connections producing the early spike. It is noteworthy in this regard that early spikes were equally difficult to obtain in several other animals undergoing the same surgery but in which section of the nerves was incomplete.

Other properties of deep tectal cells exhibiting early spikes

Cells excited at short latency from OD or OT did not differ from other cells encountered in the deep collicular layers with respect to spontaneous discharge rate or latency to antidromic activation from the predorsal bundle. Nor did the variability in onset latency of these early responses differ significantly from that observed among superficial cells. Deep-layer cells with and without early spikes were equally likely to be driven antidromically from the brain stem. Like other deep-layer cells in the ketamine-anesthetized cat, most units receiving fast retinal input could be excited by visual stimulation and were especially responsive to rapid stimulus motion. Although some of these cells clearly responded to somatosensory and auditory stimulation, we did not investigate these inputs systematically. Cells driven at short latency from OD and OT

TABLE 2. *Laminar distribution and relative frequency of cells exhibiting a late burst*

	Site of Effective Stimulation				
	OD only	OT only	Both	Neither	Total
Superficial layers*	0 (0)	11 (14)	62 (78)	6 (8)	79 (100)
Deep layers†	0 (0)	7 (9)	57 (74)	13 (17)	77 (100)
Antidromic from PDB	0 (0)	9 (12)	56 (74)	11 (14)	76 (100)

Values in parentheses are percentages. PDB, predorsal bundle. * Lower stratum griseum superficiale and stratum opticum. † Stratum griseum intermediale and below.

were found throughout the deep layers of the colliculus (laminae IV–VII of Kanaseki and Sprague, Ref. 39) and were not confined to any particular sector of its retinotopic map.

Late burst

As noted earlier, most units observed in the stratum opticum and deeper layers responded to OT or OD shock with a burst of action potentials at latencies distinctly longer than those of the early spike (Fig. 1 and Table 2). When both stimulation sites were effective in eliciting the burst, it was clear that the latency of the initial spike was only slightly greater after OD than after OT stimulation (compare Fig. 1*A* and *B*). We will consider first the minimum latencies to the initial spike of the late burst in those units exhibiting no early spike (Fig. 6). For this group of cells, the distributions of onset latency did not differ significantly between superficial and deep layers ($P > 0.10$, Mann-Whitney U test) although the superficial layers appeared to contain a few cells with unusually long response latencies (Fig. 6). If superficial and deep cells are considered together as a single population, their mean response latencies were 6.7 ms from the OD stimulus and 5.9 ms from the OT stimulus. The difference in mean latency (0.8 ms) for an average electrode separation of 38.5 mm indicates that the retinal fibers mediating the onset of the late burst had an average conduction velocity of 48 m/s between the OD and OT electrodes, i.e., close to the mean conduction velocity of retinal Y-cell axons (52 m/s, Ref. 15). We determined the OD-OT latency differences for late burst onset in individual deep-layer cells that could be driven from both sites. For the vast majority of these cells, the latency shifts fell within the range expected if activity in a Y-cell pathway evokes the first spike of the late burst (Fig. 3*B*). Since we measured latencies only to the first spike of the late burst, we cannot rule out contributions of more slowly conducting pathways to later components of the burst. Indeed, a few of our units exhibited latency shifts consistent with mediation by X- or W-cell pathways (see DISCUSSION).

Most (53 of 59) superficial- and deep-layer cells that did emit an early spike also showed a late burst following stimulation of the optic pathway. The late burst in these

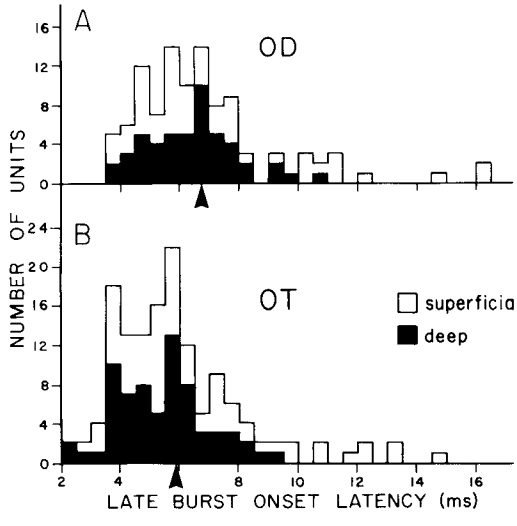

FIG. 6. Distribution of minimum onset latencies of late burst elicited from stimulation of optic disk (*A*) or optic tract (*B*). Units in this sample exhibited no early spike. Latencies measured from onset of shock artifact to foot of first action potential of late burst in 10 stimulus presentations. Laminar location of superficial (open bars) and deep samples (black bars) as in Fig. 2. Mean minimum latencies for combined superficial and deep sample indicated by arrowheads. Note shift in mean latencies of about 0.8 ms.

cells began on the average 5.5 ms after OD shock (range, 3.7–9.3; $n = 45$) and 4.6 ms after OT shock (range, 2.7–9.6; $n = 47$). The difference between these mean latencies indicates that this burst was mediated by retinal fibers having an average conduction velocity of 44 m/s. This value falls within the Y-cell range and corresponds closely to that determined for the early spike (48 m/s) and for late burst onset in cells exhibiting no early spike (48 m/s). This correspondence is reflected also in the distribution of individual OD-OT latency differences in early-spiking deep-layer cells (Fig. 3*C*).

Although initiation of the late burst appeared to depend on a Y-cell pathway in cells both with and without early spikes, the late burst began on average more than 1 ms earlier in cells that exhibited an early spike than in cells that did not. The difference was especially prominent in the superficial layers, where mean late burst onset in early spiking cells was 4.7 ms (range, 3.7–5.8; $n = 11$) after OD shock and 3.6 ms (range, 2.8–5.0; $n = 10$) after OT shock. These data are drawn from too small a sample to be considered conclusive, but if confirmed they would suggest that the pathway mediating the late burst has two components, the faster (and/or shorter) of which terminates on cells showing early spikes.

The data indicate that the pathway responsible for the late burst in most of the cells observed here involved Y-cell axons, but it is clear from the absolute latencies that these axons did not synapse directly on the recorded neurons and perhaps did not even proceed directly to the tectum. Hoffmann (35) observed in some superficial collicular units a similar paradoxical association of long response latencies with rapid afferent-fiber conduction velocities. He concluded that Y-cell inputs reached these neurons by a circuitous route through visual cortex, a route he termed the Y-indirect pathway.

DISCUSSION

The major finding of the present study is that retinal Y-cells can exert a substantial influence on neurons of the cat's superior colliculus lying in laminae deep to the stratum opticum. The results indicate that rapidly conducting retinal Y-fibers provide excitatory input to the majority of deep-layer cells by way of a polysynaptic pathway. In addition, our findings confirm two previous reports of very-short-latency excitation of some deep tectal cells following stimulation of the optic pathway (45, 56) and suggest that this excitation may be mediated by retinal Y-cell axons that project directly to the tectum.

Origin of early spike

A question of considerable functional and structural interest is whether the Y-cell axons responsible for the early spike make monosynaptic contact with the deep-layer cells. A direct projection of retinal Y-cells to the SC has been well established by both anatomical and electrophysiological methods (12, 15, 24, 35, 38, 41, 46, 83), so one may assume that some population of tectal cells receives monosynaptic Y-cell input. Hoffmann (35) observed a population of units in the superficial layers that could be activated at very short latency from the optic pathway and concluded that these units received monosynaptic contacts from Y-cell axons. Our experiments show that the early spikes recorded deep to the stratum opticum were also mediated by a Y-cell pathway and that their response latencies after OD and OT stimulation did not differ significantly from those observed in the superficial layers by us or by Hoffmann (35). Furthermore, the early spikes were undoubtedly postsynaptic responses and not antidromic spikes, since they occurred with low probability and showed considerable latency jitter. Nor were they recorded from optic tract axons or corticotectal fibers, for the early spike could be made to collide with antidromic spikes elicited from the predorsal bundle. In summary, the early spikes were recorded from neurons located in the deep layers, were synaptically mediated, and had latencies comparable to those observed in superficial cells that Hoffmann supposed to receive monosynaptic Y-cell input.

A compelling argument for monosynaptic mediation of the early spike must rest on the demonstration that the observed latencies allowed insufficient time for two synaptic steps to occur. The shortest disynaptic activation latency for an early spike would pre-

sumably be mediated by the fastest Y-cell axons, which conduct at about 122 m/s (15). If this velocity is maintained from the optic disk to the superior colliculus over a 60-mm conduction distance, conduction time would amount to 0.5 ms. Adding to this 0.4 ms for each of two synaptic delays and 0.2 ms for interneuron conduction time yields 1.5 ms as an estimate of the earliest possible tectal activation by a disynaptic pathway. Early spike latencies from OD stimulation ranged from 1.1 to 3.4 ms in our sample of deep tectal cells and from about 1–4 ms in Hoffmann's Y-direct cells. Thus, the shortest observed latencies in both of these samples appear to leave insufficient time for a disynaptic relay.[1] Early spikes with latencies longer than about 1.5–2 ms could, however, be mediated either by mono- or disynaptic Y-cell pathways.

Further evidence on this point is provided by Maeda et al. (45) who recorded intracellularly from identified tectospinal and tectoreticular neurons in the cat and found that stimulation of the contralateral OD elicited excitatory postsynaptic potentials (EPSPs) at latencies as short as 2.1 ms. These authors argued that the short-latency excitatory events were mediated by a disynaptic pathway through the superficial layers, where they recorded EPSPs with latencies of 1.4–1.9 ms following OD shock. It may be noted, though, that a latency of 2.1 ms is quite compatible with monosynaptic Y-cell activation, since impulses conducted at 36 m/s—well within the Y-cell range—would travel the 60 mm from OD to SC in 1.7 ms. Assuming with Maeda et al. (45) a 0.4-ms synaptic delay, one obtains a total latency of 2.1 ms. These authors also illustrate a sequence of EPSPs evoked in a tectoreticular neuron by OD stimulation (their Fig. 1B); the onset latency of this potential exhibits no jitter, again suggesting a monosynaptic relay.

Taken together, these physiological observations suggest that, in the cat, retinal axons project monosynaptically to at least some deep-layer cells. Excitation of deep tectal cells by electrical stimulation of optic nerve fibers has been reported in other species, but such activation has generally been attributed to polysynaptic pathways (51, 52, 65). As noted already, a direct retinal projection to the deep collicular strata finds no support in anatomical studies, which have consistently reported that retinotectal axons terminate exclusively in the superficial layers (29, 33, 39, 78; but cf. Ref. 13). This discrepancy would be explained if the contacts occurred not in the deep layers but rather on dendrites of deep-layer cells that reach into the stratum opticum. Although dendritic arborizations of most deep-layer neurons lie in the same lamina as their parent cell body, the processes of some cells do rise into the stratum opticum (44, 63, 82), where Y-cell axons are believed to terminate (35, 38, 57). A second possibility is that the deep layers receive collaterals of Y-cell axons that are too fine or few in number to be detected by present methods. Either of these possibilities would be consistent with the low synaptic security of the early spike.

Late burst: indirect retinal projections to deep-layer cells

By contrast with the relative rarity and lability of the early spike, the late burst was a robust response, easily elicited in the majority of deep-layer tectal cells in our sample, and indicative of a pathway providing significant excitatory input to these neurons. This input was evident in the unanesthetized midpontine-pretrigeminal preparation, which means that the effectiveness of the responsible pathway is not heightened abnormally by some action of ketamine. The distributions of onset latencies and latency shifts following OD and OT stimulation (Figs. 3*B*, *C* and 6) indicate that the initiation of the burst was mediated primarily by retinal Y-cell axons, but that these axons did not synapse on the recorded cells and probably did not proceed directly to the colliculus.

As emphasized already, we cannot exclude contributions to the late burst by slower conducting pathways, because the influence of these inputs would have been masked by effects of earlier impulses. Indeed, the possibility that single tectal cells receive input from both slow direct and fast

[1] Similarly, assuming the same synaptic delays and interneuron conduction time and an estimated OT-SC conduction distance of about 22 mm, the earliest disynaptic activation of tectal cells from the OT site would occur at 1.2 ms. Many early spikes are evoked from the OT at latencies shorter than this (Fig. 2*B*).

indirect pathways (35) is a reason for caution in interpreting latency-shift data. If such convergence were to occur, then the pathway responsible for triggering the first spike in a particular collicular cell could depend on the stimulus site.[2] This is evident from Hoffmann's (35) data on the superficial layers, which indicate that the Y-indirect volley largely precedes the W-direct volley after OD shock, but that a part of the W-direct volley may lead the Y-indirect volley following OT shock (see his Fig. 2). If such a "crossover" of the two volleys occurred in the convergent inputs to a single cell, the early arrival of the direct volley after OT shock would cause a spuriously large OD-OT latency shift to be attributed to the fast indirect pathway. Thus, the conduction velocity of the fast indirect pathway would be underestimated. In the present study, the estimated conduction velocities for the fibers mediating the late burst were so fast as to make it virtually certain that most were Y-fibers, regardless of whether their actual conduction velocities were somewhat higher. Therefore, the potential ambiguity produced by the crossover effect does not change our main conclusion that the late burst was initiated by a Y-indirect pathway.

Our results show that the Y-indirect pathway regularly excited those cells in the superficial and deep layers that exhibited an early spike. Such convergence was observed only rarely by Hoffmann (35) in his study of superficial-layer cells. Also, the percentages of cells driven by the indirect pathway were far larger in our sample than in that of Hoffmann. It is likely that these discrepancies are an effect of anesthesia, since Hoffmann used ether followed by nitrous oxide, while we used ketamine or brain stem transection. Most deep-layer cells respond well to visual stimuli in unanesthetized cats (27, 62), whereas such responses are much reduced and altered when nitrous oxide is employed (18, 47). Since our evidence shows that the deep layers receive a substantial Y-indirect input, the suspicion must be strong that this pathway is particularly susceptible to depression by nitrous oxide anesthesia. This is consistent with the report of Marrocco (51), whose observations in the macaque under nitrous oxide anesthesia indicate that very few deep-layer cells respond to electrical stimulation of the most rapidly conducting axons in the optic pathway. Furthermore, in a comparable study of the hamster anesthetized with pentobarbital, Rhoades and Chalupa (65) apparently found no cells below the stratum opticum that were driven by rapidly conducting retinal afferents.

Hoffmann (35) presented evidence that the Y-indirect pathway to the superficial layers involves a relay through visual cortex, and subsequent observations on corticotectal projections are consistent with this (34, 73). Since visual cortical areas send at least sparse projections to the deep collicular layers (8, 25, 40, 81), it is possible that the fast indirect pathway observed here also employs a cortical loop. Schiller et al. (69) have shown, in fact, that inactivation of the Y-like magnocellular layers of the primate geniculate eliminates visual activation of deep collicular neurons. In the cat, however, a late burst may be recorded in collicular cells following ablation of visual cortices (56) and visual responses persist in deep-layer tectal cells during cortical inactivation (76) or after decortication (36, 59, 66). These residual responses may represent the effects either of retinal fibers running directly to the colliculus or of indirect retinotectal projections that involve the pretectum, ventral lateral geniculate nucleus, or other retinal targets.

Functional considerations

The present results, and those of several previous studies (27, 52, 56, 62, 65, 77), demonstrate that the discharge of deep-layer cells can be powerfully affected by signals of retinal origin. Moreover, many of these deep-layer units are output cells that send their axons into the contralateral predorsal bundle, presumably to terminate on cells of the reticular formation and cervical spinal cord. The retinal signals relayed by such cells may account for the visual responses of reticular "omnipause" neurons, which re-

[2] This is true even if all retinal fibers directly or indirectly affecting the recorded cell are activated at both the OD and OT sites. If stimulation at these sites influences different populations of fibers, the interpretation of such data becomes even more difficult. Such differential stimulus effects may account for the few instances of negative OD-OT latency shift observed here (Fig. 3*B*, *C*).

ceive monosynaptic input from the SC (43, 64) and which can be excited at relatively short latency from the optic chiasm (43). In the cat, the timing of this excitatory visual input is consistent with mediation by the late burst observed here in deep tectal cells. Furthermore, bilateral destruction of the colliculi eliminates the responses of omnipause neurons to optic chiasm stimulation (43).

There is abundant evidence that the deep-layer cells participate in the generation and guidance of saccades in primates (53, 60, 67, 68, 70, 72, 74), and comparable evidence is emerging for the cat (16, 31, 32, 62). The notion that retinal signals can decisively influence these preoculomotor mechanisms has been seriously challenged (53, 60, 85), but the evidence crucial to this argument has been obtained almost exclusively from primates. Peck and her colleagues (62), in a recent study of tectal cells in the waking cat, noted that "many SC neurons responded lawfully before visually triggered (eye movements), but not before spontaneous saccades in the dark" (p. 99). It is possible that, in the cat, visual and eye movement-related discharges in deep layer cells are not as clearly dissociated as they appear to be in the primate. Further work is needed in the cat to determine the balance of control over deep-layer cells exercised by retinal inputs and by signals from other systems that interact with visual responses or act independently on the collicular output circuitry.

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