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Convergence of Retinal W-Cell and Corticotectal Input to Cells of the Cat Superior Colliculus

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SUMMARY AND CONCLUSIONS

1. Conduction velocities of retinotectal W-cell afferents were estimated from differences among latencies of collicular unit responses to supramaximal stimulation of the contralateral optic disk (OD), optic chiasm (OX), and ipsilateral optic tract (OT). W-cell afferents driving collicular neurons had very slowly conducting axons, nearly all below 8 m/s (mean = 5.3 m/s). These match the conduction velocities of W-cell axons terminating in the uppermost superficial gray layer and triggering juxtazonal potentials (JZPs). Such slow conduction velocities are typical of W-cells belonging to the W2 subclass ("phasic W-cells"), but are slower than nearly all W1 cells ("tonic W-cells").

2. Most W-driven cells were activated at latencies longer than expected for monosynaptic input from these W-cell afferents. However, comparable delays were observed among JZPs, which signal monosynaptic excitation of collicular neurons by W-cell terminals. This suggests that the delayed activation of W-driven cells reflects slowed conduction in the preterminal segments of W-cell afferents rather than polysynaptic transmission of W-cell signals through intermediary neurons in the brain stem or cortex. Thus monosynaptic inputs from retinal W2 cells appear to drive most neurons of the superficial collicular layers.

3. Convergence of retinotectal W-cell and corticotectal pathways was assessed by recording responses of W-driven collicular cells to intracortical stimulation of area 17. The great majority of W-driven collicular cells were activated by cortical stimulation (41/52; 79%), indicating that such convergence is widespread.

4. The population of corticotectal cells influencing W-driven collicular cells may differ from that mediating Hoffmann's Y-indirect pathway (24). W-driven collicular cells were activated from the striate cortex at longer latencies (mean = 6.3 ms) than cells driven by the Y-indirect pathway (mean = 2.5 ms). In addition, cortically activated W-driven cells were common throughout the superficial gray layer, whereas cells driven by the Y-indirect input were encountered only in the deepest part of the superficial gray and below.

5. W2 cells, apparently the dominant retinotectal cell type, nearly all project contralaterally and are tuned for slow stimulus velocities. Thus the binocularity of W-driven collicular cells and their sensitivity to moderately fast stimulus motion probably reflect the convergent cortical input described here.

INTRODUCTION

The superficial layers of the mammalian superior colliculus represent a major constituent of the extrageniculate visual system. Little is known of the role played by the superficial layers in visual function, but their anatomy suggests they may execute several operations in parallel. The major outputs of the superficial layers arise from more or less distinct populations of neurons that differ in their sublaminar distributions (see Ref. 26a for review). This sublaminar specificity is mirrored in the afferents to the colliculus, with individual components of the retinotectal and corticotectal projections terminating at characteristic depths within the superficial strata (7, 15, 24, 27, 30, 36, 52, 64). Thus collicular output to a particular target is presumably shaped by selective convergence of a

subset of retinal and cortical signals on a class of efferent collicular cells.

Direct retinal input to the cat's colliculus originates primarily in ganglion cells of the W- and Y-cell classes (10, 11, 16, 24). Though a few X-cell axons may innervate the colliculus (e.g., Ref. 49), they have never been shown to activate collicular cells. Retinal Y-cell afferents terminate in the deep stratum griseum superficiale (SGS), the stratum opticum (SO), and probably sparsely in the deeper collicular layers (1, 8, 15, 24, 27, 38, 43). Collicular cells monosynaptically excited by the Y-direct input occupy these same layers, but are not found in the upper half of the superficial gray (8, 24, 43).

W-cell afferents clearly dominate the retinotectal projection and drive most cells of the SGS (12, 24, 27, 33, 36, 43, 51, 60, 61, 65), but the functional nature of this input is poorly understood. Retinal W-cells have been formally divided into two subclasses (48). W1 cells (sometimes called "tonic W-cells"; Refs. 16 and 55) are distinguished by their sustained responses to light stimuli, small-to-medium-size cell bodies, moderately slow conduction velocities, and uncrossed projections from the temporal retina. W2 cells (also known as "phasic W-cells") give transient responses to light stimuli and have very small cell bodies, extremely slow conduction velocities, and almost exclusively crossed projections from the temporal retina. The relative contributions of W-cell subtypes to collicular unit properties are unknown. It seems likely, though, that W2-cell axons outnumber other fiber types in the retinotectal projection because most ganglion cells innervating the colliculus are extremely small (33, 47, 55, 60, 61, 65), project contralaterally (2, 3, 18, 20, 28, 57), and have exceptionally slow axonal conduction velocities (7, 15, 36, 38). Nonetheless, W1 cells also contribute at least modestly to the retinotectal pathway. For example, there is an uncrossed W-cell projection to the colliculus arising predominantly from W-cells with tonic responses to visual stimuli (16), relatively large somas (33, 55, 60, 61, 65), and relatively rapidly conducting axons (15). It is not known whether individual collicular neurons receive convergent input from W1 and W2 cells or whether these two inputs are segregated at the postsynaptic level.

Nor is it certain that the main W-cell influence on the colliculus is a direct one. Indeed, most collicular cells driven by the W-cell pathway are activated from the optic nerve and tract at latencies longer than expected for a monosynaptic input (43, 51). This may reflect oligosynaptic circuitry involving other W-cell targets, such as the pretectum or lateral geniculate nucleus, or intrinsic networks within the colliculus itself. One goal of the present study was to clarify the composition of W-cell signals driving collicular neurons and the circuits by which they do so.

There is conflicting evidence regarding the convergence of W-cell and corticotectal input to single collicular neurons. On the one hand, such convergence seems almost certain. Among superficial-layer cells, more than half receive W-cell input (12, 24, 36, 43, 51), and more than half can be activated from the visual cortex or affected by its ablation (26, 35, 43, 46, 68). Thus at least some cells must get both inputs. On the other hand, paradoxical anatomical and physiological evidence suggests that such convergence may be rare. Latency-difference studies have identified in some collicular cells a polysynaptic Y-cell influence that is mediated by corticotectal input (8, 9, 12, 24, 50, 51). Such "Y-indirect" activation has come to be equated at least implicitly with corticotectal influence. Most superficial-layer cells, however, exhibit latency behavior indicative of direct W-cell input rather than of Y-indirect influence (12, 24, 51). Moreover, retinal and cortical inputs to the SGS exhibit considerable segregation, both in their sublaminar distribution and their synaptic contacts on single neurons (23, 40, 41). These results might be interpreted to mean that most W-driven collicular cells lack corticotectal influence. Thus a second goal of this study was to assess possible convergence between these dominant visual influences on the superficial collicular layers.

METHODS

Experiments were carried out on 14 adult cats using methods that have been documented more fully elsewhere (7–9). In brief, animals were anesthetized either with Nembutal supplemented with small doses of ketamine hydrochloride (n = 11) or with a combination of ketamine (30 mg/kg ip,

supplemented iv as needed) and acepromazine maleate (0.5 mg/kg; $n = 3$). Data from ketamine-anesthetized animals did not differ appreciably from the main sample, so the two have been combined. Animals were placed in a stereotaxic apparatus, and the skull and meninges were removed to provide access to the right superior colliculus, visual cortex, and optic pathway. The brain was covered with mineral oil. Concentric bipolar stimulating electrodes were placed in the right optic tract (OT), optic chiasm (OX), and left optic disk (OD). In nine animals, a comb of three concentric bipolar electrodes with tips separated by 2 mm was placed in the striate cortex on the medial surface of the hemisphere between Horsley-Clarke coordinates P3.0 and A3.0. A stimulus isolator delivered constant-current square-wave pulses (<5 mA, 50–350 μs) between the core and sleeve of single electrodes or, in some instances of cortical stimulation, between the cores of electrode pairs.

Single units and field potentials of the colliculus were recorded throughout the SGS and SO with tungsten microelectrodes, amplified, monitored, and stored conventionally. Approximate retinotopic alignment of collicular penetrations with cortical stimulus sites was guided initially by stereotaxis and existing maps of cortical and collicular retinotopy (14, 63). The alignment was subsequently refined to maximize the amplitude of field potentials evoked in the superficial gray layer by cortical stimulation. Selected recording sites were marked with electrolytic lesions.

At the end of the experiment, animals were given a lethal dose of Nembutal and perfused through the carotids with 10% Formalin. The brain and left optic nerve were removed from the skull. Conduction distances separating stimulus sites in the optic pathway from one another and from the colliculus were measured in each brain by dissection. Locations of cortical stimulus sites and collicular recording sites were determined from frozen sections stained with cresyl violet.

Latencies of evoked collicular unit responses were measured from the onset of the stimulus artifact to the beginning of the shortest-latency spike evoked in a series of at least 20 stimulus presentations. Above threshold, evoked spikes occurred reliably in a narrow window of latencies and were easily distinguished from spontaneous activity, which was generally low. Afferent conduction velocities were estimated as described previously from the differences in minimum latency of the earliest responses evoked from ≥2 stimulus sites (8, 24). Collicular units were considered to be driven by a Y-cell pathway if they exhibited afferent conduction velocities in excess of 20 m/s (9, 24). This influence was considered Y-indirect if the cell was activated from the OX at latencies exceeding 2 ms (cf. Refs. 9 and 24). Given the ambiguous origin of later spikes in multiple-spike bursts, no systematic attempt was made to analyze them. Consequently, the present studies provide no evidence either for or against possible convergence of retinal W- and Y-cell pathways. Observations on juxtazonal potentials (JZPs) included in this report were drawn from an earlier study (7).

RESULTS

Most neurons recorded in the superficial gray layer were driven by retinal W-cells. Such neurons, here termed "W-driven cells," exhibited characteristic patterns of evoked response that were first described by Hoffmann (24). As shown for a typical neuron in Fig. 1*A*, W-driven cells responded at long latencies to shocks of the optic pathway, and their response latencies shifted many milliseconds as the site of stimulation passed from the OD to the OX or OT. The conduction velocity of the retinal fibers driving such neurons can be estimated by dividing the conduction distance between two stimulus sites by the difference in the latency of the responses evoked from those sites (24). For most collicular cells, however, including that illustrated in Fig. 1, latencies varied with stimulus strength, decreasing by as much as several milliseconds as the stimulus was increased above threshold intensity (Fig. 1*B*). This latency reduction presumably resulted from the recruitment of additional retinal afferents with excitatory inputs to this cell. Hence, there was no unique "latency-difference" value for any pair of stimulus sites, and therefore, no single estimate of afferent conduction velocity. However, above some suprathreshold stimulus level, response latencies reached an asymptotic minimum, and further increments in intensity had little effect. I have based all conduction-velocity estimates on these minimum latencies, which presumably reflect the activity of the most rapidly conducting retinal fibers capable of driving the cell under these conditions. For the cell illustrated in Fig. 1, minimum latency values from the three stimulus sites yielded estimates of retinal afferent conduction velocity ranging from 3.5 to 5.1 m/s. These values are typical of W-cell axons and slower than virtually all X- or Y-cell axons (10, 11, 59), so this neuron is clearly activated by a W-cell pathway. Such W-cell activation was typical of

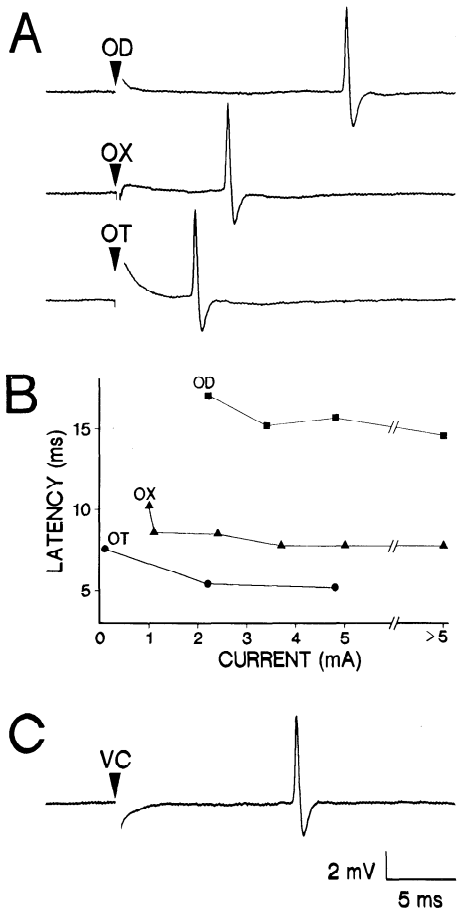

FIG. 1. Electrically evoked responses of a W-driven cell of the superficial gray layer. *A*: action potentials evoked by shocks to the contralateral optic disk (OD), optic chiasm (OX), and ipsilateral optic tract (OT). Large shifts in latency with changes in the site of stimulation and long absolute latencies are typical of W-driven cells. *B*: dependence of same cell's response latency on stimulus intensity. Latency of response evoked from each stimulus site was several milliseconds longer at threshold (*leftmost point*) than at supramaximal intensity. Estimates of the conduction velocity of retinal afferents driving this unit, based on differences among minimum response latencies, ranged from 3.5 to 5.1 m/s, velocities typical of retinal W-cell axons and slower than virtually all X- and Y-cell axons. *C*: activation of the same cell at long latency (12.7 ms) by electrical stimulation of the visual cortex (VC); stimulating electrode was located in area 17. All stimuli 150 μs in duration, 4.4–4.6 mA. Calibration marker (*bottom*) applies to both *A* and *C*. Bandpass of recording system: 100 Hz–10 kHz.

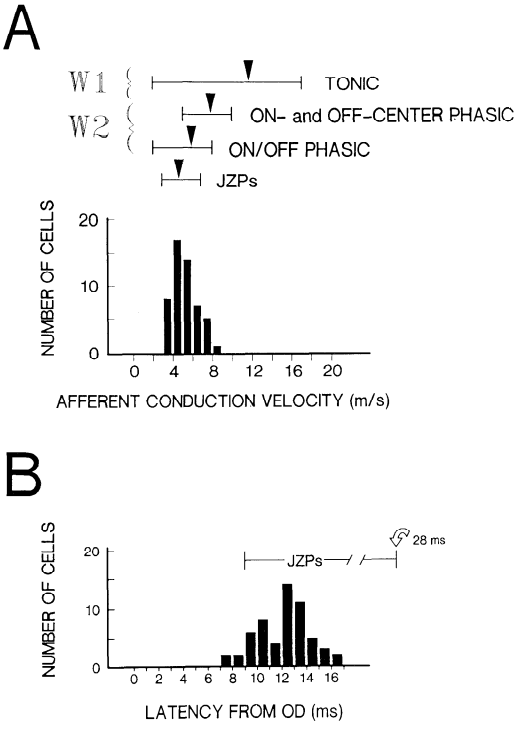

FIG. 2. Evidence for activation of superficial collicular neurons by very slowly conducting W-cell afferents. *A*: distribution of maximal conduction velocities of retinal afferents driving superficial-layer collicular cells. Estimates based on differences in minimum latency of responses evoked from the optic disk (OD) and optic tract (OT). *Top*: summary of data on axonal conduction velocities of major functional subtypes of W-cells (59) and of W-cells triggering collicular juxtazonal potentials (JZPs; Ref. 7). JZPs signal the monosynaptic excitation of collicular cells by slowly conducting W-cell afferents to the upper superficial gray layer (7, 36). *Horizontal bars* indicate total range of conduction velocities; *arrowheads* mark mean values. *B*: distributions of minimum latency of response evoked in superficial-layer cells by shocks to the OD. *Horizontal bar* above the histogram indicates the total range of latencies observed for JZPs evoked from the OD (7). Data from cells demonstrably driven by direct or indirect Y-cell pathways have been excluded in both *A* and *B*.

neurons in the superficial collicular layers: of 171 cells tested, 107 (63%) were activated by retinal fibers conducting at <13 m/s.

Very slowly conducting W-cell input to W-driven cells

Figure 2*A* illustrates the distribution of maximal conduction velocities among retinal afferents driving the recorded superficial-layer collicular cells, excluding those driven by the Y-direct or -indirect paths. Estimated afferent conduction velocities ranged from 3 to 9 m/s. Comparison with Stone and Fukuda's data for various types of W-cells (Fig. 2*A*,

top; Ref. 59) reveals that these velocities fall within the low end of the W-cell conduction-velocity range, at values characteristic of W-cells of the W2 subclass but too slow to be typical of W1 cells.

The conduction-velocity estimates of Fig. 2*A* were derived from OD-OT latency differences. Of the available estimates, these were probably the most accurate, because the large OD-OT conduction distance would have minimized the effects of measurement errors. A virtually identical distribution was obtained using estimates based on OD-OX latency differences (not shown). When OX-OT differences were used, the conduction-velocity distribution peaked at a similar value (6 m/s), but had a tail of higher values that was not evident in the other distributions: roughly one-third of the cells had estimated afferent conduction velocities in the range 8–16 m/s, well within the range of W1 cells. These relatively fast velocities are probably largely artifactual,[1] but it is apparent in any case that the great majority of W-driven cells are activated by retinal axons conducting at <10 m/s.

JZPs constitute an index of monosynaptic collicular excitation by certain W-cell afferents to the upper SGS (7, 36). The conduction velocities of these afferents are extremely slow (7) and Fig. 2*A* shows their close correspondence to those of the W-cell afferents that activate collicular cells. This suggests that a common population of slowly conducting W-cell afferents (probably those of W2 cells) trigger both the JZPs and collicular spike responses.

This is supported by the results of Fig. 2*B*, which illustrates for W-driven collicular cells the distribution of minimum latencies of activation from the OD (Fig. 2*B*). The response latencies of nearly all cells (53/57; 93%) fell within the range of latencies of JZPs. Only 7% (4/57) discharged before the earliest JZPs, suggesting possible input from a more rapidly conducting W-cell population. The scant evidence for fast W-cell activation is remarkable because the method, based on the earliest spike evoked by supramaximal stimuli, favored the detection of the fastest retinal inputs to the recorded cells. It is this methodological bias that presumably accounts for the concentration of unit responses at the short-latency end of the spectrum of JZP latencies: among the multiple convergent W-cell afferents that apparently drive single collicular cells (cf. Fig. 1*B*), it is the most rapidly conducting whose influence will be reflected in Fig. 2*B*.

Are collicular cells driven monosynaptically by retinal W-cell afferents?

Most collicular cells driven by the W-cell pathway are activated at latencies longer than expected for a monosynaptic input (43, 51). The most commonly cited explanation for this delay is that W-cell signals affect tectal neurons indirectly through intermediary neurons within the colliculus or other visual nuclei (43, 51). However, the delay may occur largely within the retinal afferents themselves. Spike conduction apparently slows substantially in W-cell axons near their terminations in the upper SGS, presumably due to reductions in preterminal caliber and myelination (7). Thus JZPs, which signal monosynaptic W-cell excitation of collicular neurons, should themselves be delayed from the

[1] The OX and OT electrodes were separated by the smallest conduction distance of any pair of stimulating electrodes (only about one-third that separating the OD and OT, for example). As a result, any small errors in the measurement of conduction distances or of response latencies would have introduced relatively large errors in conduction-velocity estimates based on OX-OT latency differences. In addition, the OX-OT distribution may reflect in part the conduction velocities of nonretinal fibers. For example, shocks to the OX and OT may have stimulated afferents from the contralateral parabigeminal nucleus (19). Parabigeminal fibers would not be activated at the OD, so their conduction velocities would not contribute directly to the histograms of Fig. 2*A*. They could, however, shift the distribution toward lower conduction velocities by reducing response latencies from the OT, thus increasing OD-OT latency differences. However, the effect would be relatively minor. Suppose, for example, that the higher velocity tail in the OX-OT distribution reflected contamination at the OT and OX by fibers 50% faster than contralateral retinal afferents; this would cause the conduction velocities shown in Fig. 2*A* to be underestimated by no more than 8%. Correcting the distribution by this amount would not substantially alter the conclusion that very slowly conducting W-cells dominate the retinotectal projection. Note, also, that if afferent conduction velocity were being systematically underestimated, collicular cells should have exhibited absolute latencies that were paradoxically short, that is, too short to have been mediated by monosynaptic inputs from the fibers whose conduction velocities were estimated. The results of Fig. 3 show that this did not occur. The possibility of contamination is rendered still more remote by preliminary evidence (unpublished observations) that few collicular cells respond to stimulation of contralateral parabigeminal afferents.

expected monosynaptic latency as calculated from the conduction velocity of the axon's main trunk.

Figure 3 compares directly the delays from the expected monosynaptic latencies of collicular unit responses (*top*) and JZPs (*bottom*). For each of 43 W-driven collicular neurons, the latency expected for monosynaptic activation from the OD was obtained by dividing the estimated afferent conduction velocity for that cell (based on its OD-OT latency difference) into the conduction distance separating the OD and colliculus in that animal and adding 0.5 ms as an estimate of the synaptic delay. The upper histogram of Fig. 3 plots the distribution of the *differences* between these expected monosynaptic spike latencies and the observed latencies of activation from the OD. Expected latencies of monosynaptic responses were estimated in the same way for each of 20 JZPs evoked from the OD in an earlier study (7). The conduction velocities of JZP-triggering axons were determined by a collision method that, like the latency-difference method of the present study, reflected the properties of the main trunk of the axon (7). The differences between the expected and observed latencies of JZPs (Fig. 3, *bottom*) represent delays attributable to preterminal slowing of conduction. The results confirm earlier reports (43, 51) that most W-driven collicular responses are delayed from their expected monosynaptic latencies, but indicate that these delays closely match those of JZPs, which signal monosynaptic W-cell excitation of the colliculus. This suggests that the delay in W-driven unit responses is largely attributable to slowed spike conduction in the W-cell axons themselves and that many of these units receive monosynaptic W-cell input. If preterminal slowing accounts for the apparent delay in collicular responses, comparable delays should have occurred for responses evoked from the OX or OT; this was confirmed both for unit responses and for JZPs. The present data do not exclude the possibility that some collicular neurons receive only polysynaptic W-cell inputs, but such neurons would seem to be rare in the superficial layers.

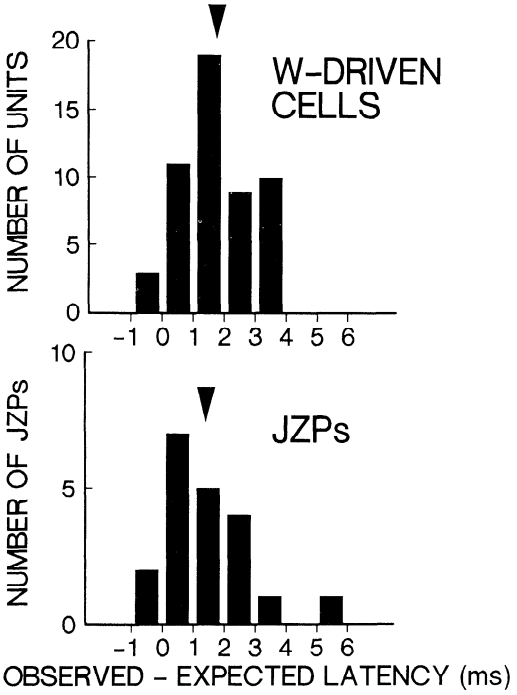

FIG. 3. Distribution of delays in the observed latency of collicular responses with respect to the latency expected for monosynaptic W-cell input. *Top*: delays in evoked spike latency of collicular W-driven cells. Expected monosynaptic latencies were determined for each cell by dividing the disk-to-colliculus conduction distance by the estimated maximal afferent conduction velocity (based on optic disk-optic tract latency differences) and adding 0.5 ms for synaptic delay. Subtracting this expected latency from the observed minimum activation latency from the optic disk yielded for each unit an estimate of the delay in the spike response from the expected monosynaptic activation latency. *Bottom*: delays among juxtazonal potentials (JZPs), which constitute an index of monosynaptic excitation of collicular cells by very slowly conducting W2 afferents (7, 36). Delays determined as for unit responses except that conduction velocities were estimated using a collision technique described elsewhere (7). Mean delays (*arrowheads*) were 1.77 ms for W-driven unit responses and 1.40 for JZPs.

Cortical activation of W-driven collicular cells

Cortical stimulation activated the vast majority of collicular neurons that were driven by retinal W-cell input. The W-driven cell illustrated in Fig. 1 was typical in this regard, being activated from the striate cortex at a latency of 12.7 ms (Fig. 1*C*). Among cells meeting a stringent criterion for W-cell activation (every individual estimate of afferent conduction velocity under 10 m/s), 79% (41/52) were driven from the cortex. This is comparable to the percentage of cortically driven cells overall in the superficial collicular layers (195/225; 87%; see also Refs. 26 and 35). These data probably underestimate the prevalence of corticotectal influence on W-driven collicular cells because retinotopic alignment of cortical stimulus sites with collicular re-

cording penetrations was only approximate in this study. When misalignments occurred, they undoubtedly reduced the probability of cortical driving among recorded collicular neurons (34, 35). W-driven cells could be activated from sites in area 17 many millimeters from the area-18 border with currents as low as 300 μA. Nonetheless, extrastriate areas were probably activated by cortical shocks in most cases, if not through current spread then by synaptic connections.

Relationship to Y-indirect pathway

It is noteworthy that W-driven cells, though activated from the cortex, gave no indication of receiving input from the Y-indirect pathway, which is known to reflect corticotectal influence (9, 24). If they had received Y-indirect influence, their initial response to stimulation of the optic nerve or tract should have exhibited the small latency differences characteristic of this input, because the Y-indirect influence actually reaches the colliculus before the W-direct volley (Ref. 24 and unpublished observations). Instead, they had the very large latency shifts that signal W-cell influence.

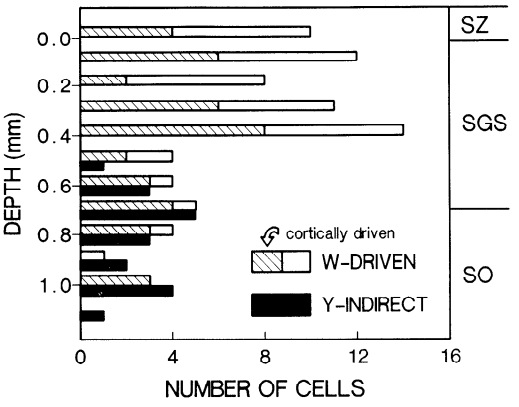

FIG. 5. Comparison between the laminar distributions of collicular cells driven by the Y-indirect route (*black bars*) and by retinal W-cell input (*light bars*). W-driven cells activated from the cortex are indicated by *hatching*. W-driven cells were common in the upper two-thirds of the stratum griseum superficiale (SGS) but relatively rare in the stratum opticum (SO). Cells with Y-indirect input were never recorded in the upper half of the SGS, but were frequently encountered in the bottom third of the SGS and in the SO. Y-indirect activation is also common in the intermediate and deep collicular layers (Ref. 8; not shown). Criteria for identifying cells with Y-indirect or W-cell input as for Fig. 4; not all W-driven cells were tested for response to cortical stimulation.

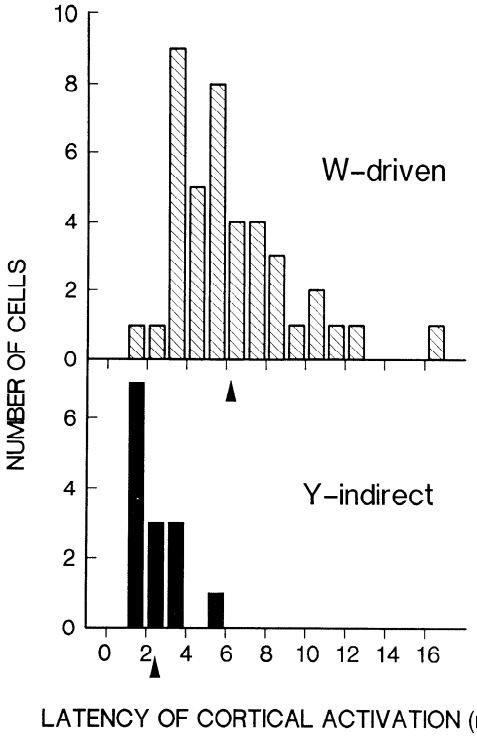

FIG. 4. Latencies of activation from striate cortex among collicular cells driven either by W-cell input (*above*) or by the Y-indirect pathway (*below*). Mean cortical activation latencies (*arrowheads*) were much longer for W-driven cells (6.3 ms) than for cells exhibiting Y-indirect activation (2.5 ms). For W-driven cells included here, all latency-difference measurements (disk-chiasm, disk-tract, and chiasm-tract) yielded estimated afferent conduction velocities <10 m/s. Cells identified as receiving Y-indirect input had estimated afferent conduction velocities >20 m/s and OX latencies >2 ms.

This suggests that the corticotectal circuit influencing W-driven collicular cells may be distinct from that mediating Y-indirect activation. This inference is further supported by two observations. First, as shown in Fig. 4, W-driven collicular cells were activated from the striate cortex at longer latencies on average (mean = 6.3 ms) than were collicular cells exhibiting Y-indirect input (mean = 2.5 ms). Second, cortically activated W-driven cells were encountered throughout the superficial layers and were especially prevalent in the upper half of the SGS; by contrast, cells activated by the Y-indirect pathway were recorded only in the deepest part of the SGS and below (Fig. 5).

The most parsimonious explanation for these results is that electrical stimulation of the striate cortex activates at least two corticotectal inputs in parallel: a long latency

component terminating superficially on collicular neurons with W-cell input; and a shorter latency component terminating deeper on cells exhibiting Y-indirect input. Of course, depth segregation of postsynaptic unit types need not reflect a stratification of input. The proposed laminar segregation of the two corticotectal systems is supported, however, by current source-density evidence that Hoffmann's Y-indirect pathway synapses selectively near the border of the SGS and SO (15), where cells with Y-indirect input are found (Fig. 4). Likewise, several interpretations may be offered for the differing transmission times of the two components. The longer-latency activation of W-driven cells as compared with Y-indirect cells may reflect input from more slowly conducting corticotectal cells or through a more indirect, polysynaptic circuit.[2]

DISCUSSION

The results of this study support the view that most of the retinal input to the superior colliculus comes from W-cells with extremely slowly conducting axons, presumably W2 cells. These cells evidently activate most collicular neurons directly, rather than through polysynaptic circuits as previously suggested. The findings also show that nearly all collicular cells driven by W-cell input receive convergent excitatory influence from the visual cortex. This influence is apparently distinct from the Y-indirect pathway identified by Hoffmann (24).

W-cell input to the superior colliculus

Since the pioneering work of Hoffmann (24), W-cells have been known to provide most of the retinal input to the cat's superior colliculus. More recent evidence suggests that the bulk of the retinotectal projection arises from a subset of W-cells—the W2 cells—whereas a smaller component is supplied by W1 cells (see INTRODUCTION). The present findings suggest that the functional dominance by W2 cells may be even stronger than expected at the postsynaptic level: under the conditions of these experiments, most superficial-layer cells could be driven only by retinal afferents with the slow conduction velocities typical of W2 cells. Remarkably few cells appeared driven by faster-conducting W1 afferents. It should be noted that these experiments were not suitable for detecting possible subthreshold excitatory influences from W1 afferents. Nor do they rule out a contribution from the small numbers of W1 cells with axons as slowly conducting as those of W2 cells. Nonetheless, in light of the crossed projections and extremely small size of most ganglion cells innervating the colliculus, these data leave little doubt that W2 cells dominate the retinotectal projection.

The present findings confirm earlier reports (43, 51) that most W-driven cells are activated at latencies longer than expected for a monosynaptic input. It has been suggested that W-cell signals may affect these cells only indirectly, after relays in the colliculus itself, in other subcortical W-cell targets or, by analogy with the Y-indirect pathway, in the W-cell stream of the geniculocortical pathway (43, 51). The findings reported here indicate that most of the delay can be attributed instead to properties of the retinal afferents themselves. Paradoxically long latencies just like those exhibited by W-driven cells are typical of juxtazonal potentials, which signal monosynaptic W-cell excitation of collicular neurons (Fig. 3). This is presumably because the expected monosynaptic latencies of both JZPs and postsynaptic units were figured from the estimated conduction velocity of the main trunk of the afferent axons, whereas conduction in these axons is probably not uniform, slowing considerably near their terminal ramifications. Their caliber and myelination undoubtedly decrease in the colliculus itself, and they may also undergo thinning in or near the brachium of the superior colliculus because they may distribute collaterals to the geniculate C-layers or pretectum. Such preterminal slowing seems adequate to account for the delays illustrated in Fig. 3. Fine unmyelinated axons of the central nervous system are known to propagate action potentials as slowly as 0.1–0.3 m/s (39, 45,

[2] It seems unlikely, however, that a single population of corticotectal neurons in area 17 is responsible for both pathways, driving Y-indirect cells monosynaptically and W-driven cells through collicular interneurons. This would require monosynaptic input from the striate cortex to reach cells only relatively deep in the superficial layers, whereas anatomic evidence shows it to be concentrated superficially (4, 30, 40, 64). Nor would it explain why inactivation of areas 17 and 18 affects cells in the upper SGS, but not those in the deeper layers receiving the Y-indirect influence (42).

62). A preterminal segment conducting at 0.1 m/s would only have to be 300 μm in length to introduce an apparent delay of 3 ms in a JZP or unit response evoked by a W-cell axon conducting at 5 m/s. Thus the apparent delay in W-driven collicular responses seems likely to reflect the nonuniform conduction velocity of W-cell afferents rather than any polysynaptic circuitry mediating W-cell influence on the colliculus.

The major collicular target of W2 input appears to correspond to the tier of densest retinal input, namely the upper 50 μm of the SGS (7, 15). On the other hand, collicular neurons driven by this input are encountered throughout the superficial layers (Fig. 5 and Ref. 43). If W2 activation is initiated largely by monosynaptic contacts, then most W-driven collicular cells apparently receive their major retinal input within the upper SGS, whatever the depth of their somas. Anatomic studies confirm that many cells throughout the superficial gray extend dendritic processes into the upper SGS (31, 43, 56). This is in keeping with evidence that the current sink induced in the upper SGS by excitatory W-cell synapses has a corresponding source distributed throughout the underlying SGS (15, 36). The secure activation by W2 afferents of cells in the middle and deeper SGS implies that synaptic inputs in the upper SGS have a far more profound excitatory impact at the axon hillock than might have been predicted from their distal dendritic position. This may reflect some active property of collicular dendrites or simply the sheer density of W-cell synapses on postsynaptic elements in the upper SGS. In any case, it suggests that sublaminar specificity in afferent input to the SGS may be substantially smeared at the postsynaptic level. For example, monosynaptic input from W2 cells may be expected to drive not only the very superficial collicular cells that project to the lateral geniculate nucleus, but also cells of the lower SGS that innervate the pulvinar complex (26a).

Corticotectal convergence on W-driven collicular neurons

This study has demonstrated that most collicular cells driven by retinal W-cell input receive convergent excitatory influences from the visual cortex. This result is not entirely unexpected. W-driven collicular cells predominate in the SGS (12, 24, 51), a layer heavily innervated by the visual cortex and containing cells that are in many cases dependent on cortical input for their selectivity or responsiveness to visual stimuli (42, 46, 68). Indeed, in Hoffmann's original report (24), evidence suggesting such convergence is mentioned in passing; the present findings show it to be widespread.

On the other hand, the finding is seemingly at odds with certain anatomic evidence. Cortical and retinal inputs to the SGS exhibit largely complementary laminar and topographic distributions (17, 40). Retinal inputs, presumably W-cell afferents, are densest in the upper 50 μm of the SGS and are especially dense outside the representation of the area centralis (2, 18, 20, 40, 57); inputs from the striate cortex, by contrast, are densest just beneath the main retinal terminal layer and within the representation of the area centralis (40, 64; but see Ref. 4). Moreover, ultrastructural studies have revealed little evidence of synaptic convergence of retinotectal and corticotectal afferents on single collicular neurons (23, 41). A plausible explanation for the apparent conflict between the anatomy and physiology is that retinal and cortical afferents simply synapse on largely separate dendritic sectors of collicular neurons. Convergence of this sort need not be apparent at the level of light microscopy nor in any but the most complete serial electron microscopic reconstructions. Another possibility, of course, is that most W-driven cells do lack monosynaptic cortical input and receive corticotectal influence instead through polysynaptic circuits.

The present evidence for convergence of retinal W-cell and corticotectal input is also apparently in conflict with certain electrophysiological findings. Many cortical cells projecting to the colliculus are driven by the geniculocortical Y-cell stream (6, 22, 54); none are known to be driven by the X- or W-cell systems. Corticotectal projections mediate the multisynaptic Y-cell influence that is detectable in some collicular cells by the latency-difference method (9, 24). This Y-indirect pattern of activation has come to be equated at least implicitly with corticotectal influence. Yet W-driven collicular cells, most of which can be activated from the cortex, fail to exhibit Y-indirect latency behavior. This suggests that a substantial component of corticotectal influence eludes detection by the la-

tency-difference technique. The failure may stem from the method's reliance on the shortest-latency response to detect afferent inputs. The well-established Y-indirect pathway, though polysynaptic, actually activates collicular cells at shorter latencies than the direct W-cell input. This need not be true for the retinocorticotectal input to W-driven cells. Indeed, transmission times in the corticotectal segment alone are substantially longer for this circuit than for the Y-indirect pathway (Fig. 4). Another limitation of the latency-difference technique is that it can detect only those pathways that are reliably evoked by stimulation of the peripheral visual pathway. Yet as many as two-thirds of corticotectal cells in area 17 fail to respond to such stimuli (22, 54); among these may be those innervating W-driven collicular cells.

Because W-driven collicular cells do not exhibit frank Y-indirect activation, the nature of the geniculocortical drive to the corticotectal cells influencing them is an open question. Geniculocortical Y-cell signals seem likely to be at least partly responsible. Many corticotectal cells in areas 17 and 18 are known to receive Y-cell influence (22, 54), and the response properties of W-driven collicular cells are substantially altered by manipulations thought to disrupt geniculocortical Y-cell pathways preferentially (12; see also Ref. 25). On the other hand, the possibility that cortical influence on W-driven collicular cells represents an X-indirect or W-indirect pathway has not been ruled out. Though neither X- nor W-cell inputs have been shown by latency analysis to drive corticotectal cells, this negative result is inconclusive given the refractoriness of most corticotectal cells to electrical stimulation of the visual pathway (22, 54). Indeed, the X-cell and W-cell components of the retinogeniculocortical pathway seem particularly refractory to such electrical stimuli (13, 53).

Even if its geniculate drive does come from Y-cells, the circuit mediating corticotectal influence on W-driven cells is almost certainly distinct from that which mediates the Y-indirect pathway of Hoffmann (24). These two corticotectal influences differ not only in their transmission time from the cortex (Fig. 4) and in their detectability by the latency-difference method, but also in their laminar distribution (Fig. 5). The two pathways might originate in separate populations of neurons in the striate cortex because corticotectal cells of area 17 are anatomically and physiologically heterogeneous (22, 29, 66, 67) and vary in conduction velocity by as much as an order of magnitude (22, 44, 54, 66). On the other hand, it is possible that some of the collicular activation that follows striate stimulation is relayed through the extrastriate areas, which receive strong inputs from area 17 and have independent corticotectal projections.

An extrastriate origin seems particularly likely for Hoffmann's Y-indirect pathway (24). This pathway targets cells within the deep SGS and below (Fig. 5 and Ref. 15), closely matching the stratum of densest input from the lateral suprasylvian (LS) areas and well beneath that from areas 17 and 18 (4, 30, 40, 52, 64). The projection from LS does carry the requisite Y-cell signals because corticotectal cells in LS are driven polysynaptically by geniculate Y-cell inputs (6). Moreover, inactivating LS alters or abolishes the visual responses of cells in collicular layers receiving Y-indirect signals (Fig. 5 and Refs. 8, 9, 15), whereas cooling the striate cortex has no effect on these cells (42). A contribution from the striate cortex to Hoffmann's Y-indirect pathway cannot be excluded: Y-driven corticotectal cells in area 17 (22, 54) might contact cells of the deep SGS or SO through weak projections to those layers or through synapses on distal dendrites extending more superficially. Nonetheless, present evidence weighs heavily in favor of an origin for Hoffmann's Y-indirect pathway primarily in the extrastriate cortex rather than in area 17.

The cortical influence on W-driven collicular cells seems likely to originate at least in part in the striate cortex. This influence reaches cells in the upper half of the SGS, in which corticotectal input from the striate cortex is dense (4, 30, 40, 64) and in which the visual properties of collicular cells are sensitive to inactivation of areas 17 and 18, but not of LS (42). An apparent paradox posed by this anatomic model is that area 17 stimulation drives collicular cells at shorter latencies by the proposed multisynaptic pathway (striate to extrastriate cortex to collicular Y-indirect cells) than by a direct pathway (striate cortex to collicular W-driven cells). In fact, though, this is fully consistent with properties of these corticotectal systems. Many corticotectal cells in area 17 have slowly conducting axons (22, 44, 54, 66), whereas the

pathway from LS to the colliculus is extremely fast (6), and the same appears to be true for transcortical inputs to LS (32).

Relationship of inputs to collicular receptive-field properties

Many of the receptive-field properties of superficial collicular cells may simply reflect convergent input from retinal W2 cells, which include direction-selective W-cells and on-center, off-center, and on/off-center phasic W-cells (48). Simple convergence of input from these cell types could account for the phasic responses and slow velocity preference of collicular W-driven cells (12, 24, 37, 58), because these are also features of W2 cells (10, 11, 59). In view of the present evidence that multiple W2 afferents converge on single collicular cells, it comes as no surprise that collicular receptive fields are generally much larger than those of W2 cells (10, 11, 37, 58, 59). The preference of many collicular cells for stimuli substantially smaller than their excitatory receptive fields (5, 37, 58) may also be attributable to W2 cell convergence, because W2 cells have rather small receptive fields and low sensitivity to extended stimuli (10, 11, 59).

On the other hand, several properties of W-driven collicular cells point to convergent influences from sources other than W2 cells. Most W-driven cells are binocular (12, 24, 51), whereas W2 cells would be expected to supply information almost exclusively from the contralateral eye. Despite the preference of W-driven cells for slow-stimulus velocities, many of these cells are also responsive to stimuli moving faster than the cutoff velocity of W2 cells (about 50°/s; Refs. 10–12, 59). These properties may be conferred in large part by the corticotectal inputs identified here. For example, most corticotectal cells are binocular and are responsive to rapidly moving stimuli (22, 44, 54). Both inactivation of the cortex (42, 46, 68) and degradation of geniculocortical Y-cell function (12, 25) reduce the ipsilateral-eye responsiveness and high-velocity sensitivity of many collicular cells, including those known to be W-driven. The fact that these effects are incomplete may imply still other convergent influences on W-driven cells, perhaps from the parabigeminal nucleus, from other subcortical nuclei, or from weak W1, X- or Y-cell inputs.

Corticotectal influence is probably also partly responsible for the direction-selectivity that is typical of W-driven collicular cells (12, 24; see also Ref. 51). It would be premature to rule out any role for direct retinal input because some direction-selective W-cells have been antidromically activated from the colliculus (11, 16); in fact, as members of the W2 subclass (48), direction-selective ganglion cells may be among the receptive-field types dominating the retinotectal projection. Still, it is generally accepted that the corticotectal pathway plays a pivotal role in the expression of collicular direction-selectivity: cortical ablation or inactivation drastically reduces the incidence of direction-selective tectal neurons (42, 46, 68). A cortical role appears to apply specifically to W-driven collicular cells, because they become largely unselective for stimulus direction when Y-indirect pathways are disrupted (12).

Whatever their functional contribution, corticotectal signals must now be recognized to influence collicular cells regardless of retinal input type or laminar position. The failure of the latency-difference technique to reveal much of this influence underscores the method's inherent limitations and serves notice that functional convergence among various tectal afferent pathways may prove even more extensive than is now apparent.

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