# Effects of Early Monocular Deprivation on Visual Input to Cat Superior Colliculus

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DUE LARGELY TO the pioneering work of Wiesel and Hubel (10, 11, 24, 25), we have been aware for some time of many functional effects of early visual deprivation at the single-neuron level. Their results from normal, monocularly deprived (MD), and binocularly deprived (BD) cats have demonstrated that cells of the striate cortex developed permanently abnormal receptive-field properties in the deprived cats. For instance, they reported that most cells in the normal cat's striate cortex had receptive fields which could be activated by appropriate visual stimuli applied to either eye, and that these fields generally had orientation and/or direction selectivity (10). In the MD cat, the striate neurons' receptive fields appeared normal for the nondeprived eye, but stimuli applied to the deprived eye activated very few of these cells (11, 24, 25). In the BD cat, only about half of the striate neurons had normal receptive-field properties, the remainder either being unresponsive to visual stimuli or lacking orientation and direction selectivity (25). These results have since been substantially confirmed by others (2, 5).

More recent studies of functional pathways in the cat geniculocortical system have yielded new and fruitful directions of research into the functional effects of early deprivation. For instance, data were compiled suggesting that two parallel pathways, largely functionally independent, transmit visual information in the geniculocortical system (8, 9, 18, 19). These can be termed the X-pathway and the Y-pathway. For the former, retinal X-cells synapse onto dorsal lateral geniculate nucleus (LGN) X-cells (3, 9), which in turn project to area 17 (20), possibly onto simple cells (8, 20). For the latter, retinal Y-cells synapse onto LGN Y-cells (3, 9), which project both to areas 17 and 18, possibly onto complex cells (8, 20). Analogous to the receptive-field differences between cortical simple and complex cells (10), X- and Y-cells differ in retina and LGN both by virtue of their receptive-field properties and the fact that X-cell axons conduct more slowly than Y-cell axons (3, 9).

In this context, an investigation of the LGN in MD and BD cats disclosed that Y-cell activity could only rarely be recorded, although X-cells were frequently encountered (16). In the LGN of MD cats this Y-cell "loss" was very severe in laminae receiving direct retinal afferents from the deprived eye and located in the medial, binocular segment,[1] but not at all apparent either in laminae receiving direct retinal afferents from the nondeprived eye or in the lateral, monocular segment.[1] In BD cats, there was a moderate loss of Y-cells throughout the LGN. Subsequent data from MD and BD cats revealed both that

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[1] The binocular segment of the visual field is that central portion normally seen by both eyes, each monocular segment being the peripheral crescent seen only by the ipsilateral eye. In the cat, the binocular segment of visual field is mapped onto the binocular segment of the LGN, this being the medial portion including all of lamina $A_1$, and the corresponding parts of lamina A and the C laminae. The monocular segment of the LGN is then the most lateral portion of lamina A and the C laminae where these extend beyond lamina $A_1$. Likewise, the binocular and monocular segments of the visual cortex and SC each has mapped onto it the binocular and monocular segments of visual field.

retinal ganglion cell populations and their axons were normal (17). Consequently, the effects of visual deprivation seem to occur central to the optic tract.

A recent investigation (7) of visual afferents to the normal cat's superior colliculus (SC) was analogous to the above-mentioned geniculocortical analysis. Data from electrical stimulation of the visual pathways revealed that, among SC units: *1*) most (73%) were directly activated by axons of retinal W-cells in the W-direct pathway (cf. ref 21), *2*) a few (9%) were directly activated by the axons of retinal Y-cells in the Y-direct pathway, and *3*) the remainder (18%) were activated through the corticotectal loop in the Y-indirect pathway. The Y-indirect pathway involves retinal Y-cells, LGN Y-cells, and cortical complex cells, the last sending axons into the corticotectal pathway (12).

Based on this classification of normal visual input to the cat SC, we investigated these same pathways in MD cats. Evidence is presented below that in such cats a distinct abnormality exists among these pathways. The Y-indirect input transmitting information from the deprived eye was missing among SC afferents, while the other inputs were routinely found to respond normally. This finding is in accord with the earlier demonstration in MD cats that very few Y-cells driven by the deprived eye could be recorded from the LGN (16) and that complex cells (as well as other cortical cells) become unresponsive to visual stimuli (2, 5, 11, 24, 25).

## MATERIALS AND METHODS

### *Subjects*

Electrophysiological experiments were performed on six cats which were 6–12 mo of age. They were born and reared in the laboratory under conditions of monocular deprivation. The deprivation resulted from suturing together the lids of one eye during the 8th postnatal day. The lids were parted just prior to the experimental sessions so that the deprivation extended throughout the "critical period" as defined by Hubel and Wiesel (11).

### *Preparation, recording, and stimulation*

Cats were anesthetized initially with ether for the surgical preparation and with $N_2O/O_2$ (60/40) for the remainder of the session. They were then immobilized by continuous intravenous infusion of gallamine triethiodide and artificially ventilated. The pupils were dilated with atropine and Neo-Synephrine. A zero-power contact lens protected each cornea and served as a 3-mm-diameter artificial pupil. The eyes were focused onto a 1-m frontal tangent screen with spectacle lenses chosen by use of a Rodenstock refractometer.

Activity of SC units was recorded either with 4 M NaCl micropipettes having impedances of 4–10 MΩ at 50–300 Hz or with electrolytically sharpened tungsten wire electrodes varnished with Insl-X and having a similar impedance range. Rectangular pulses up to 25 or 30 mA and 50–100 μs in duration were delivered with bipolar stimulating electrodes in various positions along the visual pathway (see below). One cat had no stimulating electrodes and was studied solely for receptive-field features. The remaining five all had an electrode pair on the optic chiasm (OX). Of these five, three also had electrodes in the deprived eye's optic nerve (ON), one had electrodes in the nondeprived eye's ON, and one had electrodes in the optic tract (OT) contralateral to the deprived eye. The ON electrodes were placed extracranially at the exit of the nerve from the eyeball. This was accomplished by carefully dissecting parts of the surrounding periorbital tissue in such a manner as to preserve excellent optics for the eye.

In a previous study of normal cats, Hoffmann (7) devised a means for distinguishing three types of afferent input to SC neurons using criteria based on the conduction velocity of the retinofugal axons and the latency of the collicular neurons' responses to electrical activation of the OX. Conduction velocity for a given neuron was determined by the latency difference between responses to OX and either ON or OT shock (7). Consequently, the three types of afferent input are: *1*) the W-direct with a conduction velocity of less than 15 m/s, *2*) the Y-direct with a conduction velocity of greater than 35 m/s and an OX latency of less than 3.0 ms, and *3*) the Y-indirect with a conduction velocity of greater than 35 m/s and an OX latency of greater than 3.0 ms (see INTRODUCTION). Figure 1*A* summarizes Hoffmann's data on the distribution of SC afferents in normal cats (7).

### *Visual stimulation*

Targets were moved by hand across the tangent screen to map SC unit receptive fields. These targets were spots and light bars 1–5° in width, with varying luminances against a

background luminance of either 35 or 70 cd/m$^2$. Target movements were monitored by a light-sensitive resistor (9). As previously described (4), three receptive-field characteristics were noted: *1*) ocular dominance, *2*) direction selectivity, and *3*) speed selectivity. Only excitatory receptive fields were considered; no attempt was made to study inhibitory components.

In addition to the above, direction selectivity in some units was analyzed in more detail with the aid of poststimulus histograms which relate frequency of cell discharge to stimulus position. For this we adhered closely to previously described techniques (1, 4).

## RESULTS

A total of 419 SC neurons from six MD cats were studied in this experiment. All were recorded within 1,500 μm of the collicular surface, and thus were presumably either in the superficial gray layer or the stratum opticum. Not every unit could be investigated for the full range of features described below due to technical limitations.

### *Electrical activation*

In the present study five MD cats were implanted with two pairs of stimulating electrodes, and we thus successfully identified the afferent input to 131 SC units by measuring the response latencies to stimulation of the OX and either the ON or OT. These units were all recorded from the binocular segment of the SC contralateral to the deprived eye. Because we were interested in differences between SC inputs from the deprived and nondeprived eyes, we concentrated on units with monocular receptive fields in this section of the experiment (with our methods we were unable to determine the contribution of each eye to binocular SC neurons; see also below). Therefore, most of our latency data is from monocular units (i.e., with monocular receptive fields), and these comprised 100 of the 131 units. There was no difference in the latency range for monocular and binocular units, but latency data from the 31 binocular units will not be further considered in this section and are not included in Fig. 1*B, C*. However, receptive-field data from these neurons are discussed below.

In addition to determining the type of

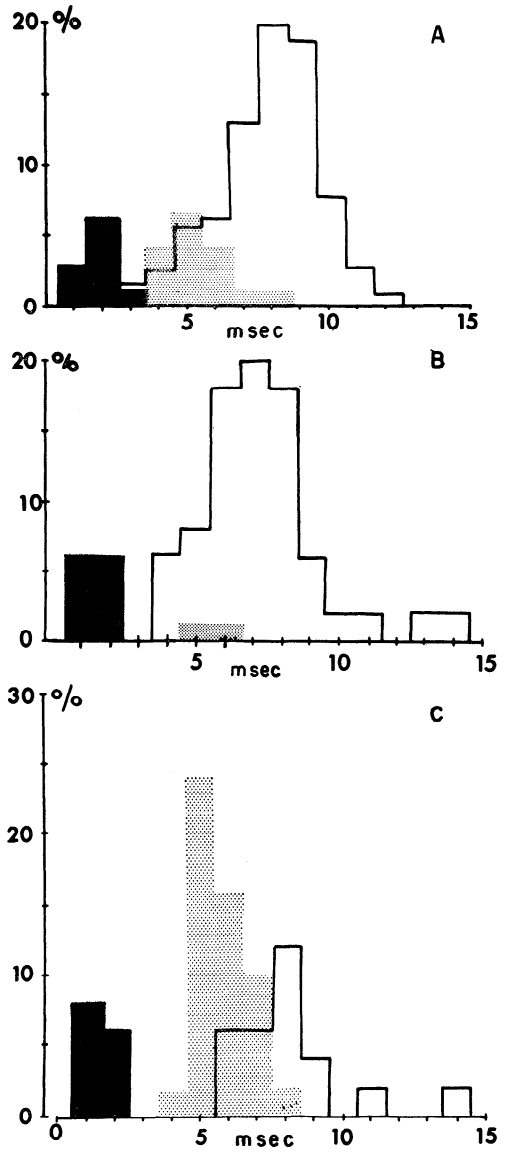


FIG. 1. Diagrams showing the relative latency-frequency distribution for SC units in normal (*A*) and MD cats (*B, C*). For each of *A–C*, the horizontal axis represents the latency of SC unit discharge in milliseconds after electrical activation of the OX, and the vertical axis shows the percentage of units in each 1-ms-wide latency group. The units' afferents were classified according to both their conduction velocities and latencies of evoked discharge (see text) as: W-direct (open bars), Y-direct (black bars), and Y-indirect (stippled bars). *A:* 170 units recorded from normal animals (data from ref 7). *B:* 50 units driven by the deprived eye in MD cats. *C:* 50 units driven by the nondeprived eye in MD cats. For *B, C*, the data were all collected in the SC contralateral to the deprived eye.

afferent input for each SC neuron, we also determined the eye of origin of that input. From the four cats with OX and ON electrodes, 50 of the monocular units could be activated from both stimulation sites, and an additional 31 monocular units could be activated only by the OX electrodes. These 31 SC units thus could not be used for data on afferent type, but they are used below for receptive-field analysis. It is important to note that the receptive fields of the 50 units activated by the ON electrodes were restricted to the eye from which the ON originated. In contrast, the 31 units driven only by the OX electrodes had receptive fields restricted to the eye associated with the other nerve. Given this, we could determine the eye of origin for the afferent input to the 50 SC units activated by both OX and ON electrodes. Of these 50 units, 30 were driven by the nondeprived eye and 20 by the deprived eye. We also concluded that monocular SC units are electrically activated only through the ON corresponding to the visually active eye. That is, there is a perfect correlation between the visual and electrical identification of the active eye.

We then made use of this conclusion to determine the eye of origin for 50 additional monocular units which could be activated by OX and OT stimulation in the final MD cat of the series; 30 of these were driven by the deprived eye, 20 by the nondeprived eye. There was no apparent difference in the distribution of afferent types among the 50 monocular units classified with ON and OX electrodes and the 50 monocular units classified with OX and OT electrodes. Thus the two populations were pooled to give the 100 units shown in Fig. 1*B, C*. Figure 1 includes only OX latencies which, for the MD cats, are within normal bounds for each afferent group. Although ON and OT latencies are not illustrated they too fall within the normal bounds (cf. ref 7).

Figure 1*B, C* clearly indicates that in MD cats the deprived eye drove many fewer SC units via the Y-indirect pathway than did the nondeprived eye ($P < 0.001$).[2] However, comparing data from MD cats to those from normal cats (Fig. 1*A*) shows not only that the deprived eye had fewer than normal Y-indirect afferents ($P < 0.002$), but also that the nondeprived eye had more than normal ($P < 0.001$). These data imply three possible consequences of early monocular deprivation: *1*) the deprived eye provides a greatly reduced Y-indirect afferentation of the SC, *2*) the nondeprived eye provides extra Y-indirect afferentation of the SC, or *3*) both of the above. However, it is emphasized that data from Fig. 1*B, C* are from the same MD cats, whereas Fig. 1*A* represents data from a different experiment. Therefore this could obscure comparisons between the data of Fig. 1*A* and *B, C*, a point reiterated below. Our tentative conclusion is that in MD cats the deprived eye drives very few units via the Y-indirect pathway. Whether or not the nondeprived eye's afferentation to SC is also affected (i.e., by a relative increase in the Y-indirect pathway) must remain an open question.

*Ocular dominance*

Figure 2 shows the ocular dominance distribution for SC neurons in normal cats (Fig. 2*A;* data from ref 7) and in MD cats (Fig. 2*B–D*). All units which reliably responded to visual stimulation of each eye separately were counted as binocular. A cell which responded reliably only to stimulation of one eye was counted as ipsilateral or contralateral according to which eye drove the cell. Three points emerge from these data. First, for cells located in the binocular segment of the SC (Fig. 2*A, B, D*), a smaller proportion had input from the deprived eye than had input from the nondeprived eye in MD cats or either eye in normal cats ($P < 0.001$). Second, the deprived eye, even with this reduced drive, was much more effective in activating units in the contralateral than ipsilateral SC ($P < 0.001$), and this presumably obtains from the normal preponderance of the contralateral retinotectal pathway over the ipsilateral one (13, 18a, 22). Third, for the monocular segment of the SC contralateral to the deprived eye, visual stimulation of the eye drove every unit encountered (Fig. 2*C;* see also below). In the five binocularly

[2] Unless otherwise indicated, all probability levels given in this paper were derived by means of the $\chi^2$ test.

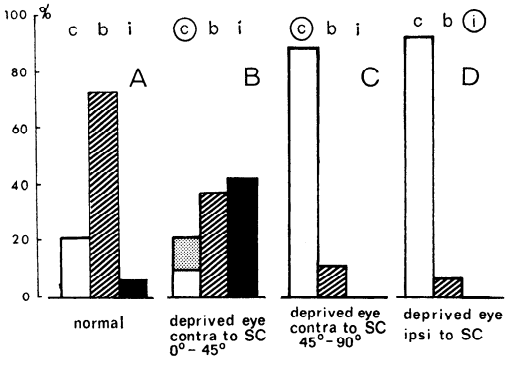


FIG. 2. Ocular dominance distribution for SC neurons from normal (*A*) and MD cats (*B–D*). For each of *A–D,* the horizontal axis represents the ocular dominance grouping: c represents units activated only by the contralateral eye (open bars); b, units activated by either eye (striped bars); and i, units activated only by the ipsilateral eye (black bars). A circle around the c or i indicates the deprived eye. The vertical scale shows the percentage of c, b, or i units in each group. *A:* 188 units from the SC of normal cats; data partly from Fig. 6 of ref 7. *B:* 278 units from the binocular segment of the SC contralateral to the deprived eye. The contralaterally driven units represented by the stippled part of the open bar were all recorded from one animal in three electrode penetrations. We found these in "monocular islands" of the binocular segment; that is, no units influenced by the nondeprived eye were found in these penetrations. More typical penetrations for all MD cats are shown in Fig. 3. *C:* 46 units from the monocular segment of the SC contralateral to the deprived eye. Of these 46 units, 5 were binocular presumably because the medial edge of their receptive fields extended into the binocular segment of visual field. *D:* 95 units from the binocular segment of the SC ipsilateral to the deprived eye.

driven units, portions of their receptive fields actually extended into the binocular segment. That is, the ipsilateral (nondeprived) eye's fields by definition were confined to the binocular segment, but the corresponding fields for the contralateral (deprived) eye were much larger and extended peripherally well beyond the ipsilateral eye's fields into the monocular segment.

In the binocular segment an inverse correlation existed between recording depth, measured in microns from the collicular surface, and the strength of input from the deprived eye, measured by the ocular dominance classifications as in Fig. 3 ($r = -0.75$ $P < 0.001$ for the SC contralateral to the deprived eye; $r = +0.41$, $P < 0.05$ for the other SC). This was seen throughout the experiment, but was most obvious for one cat which provided the results illustrated in Fig. 3. Figure 3*a–d* represents the relationship between ocular dominance and recording depth in the SC contralateral to the deprived eye, and Fig. 3*e, f* represents this relationship for the SC ipsilateral to the deprived eye.

*Direction selectivity*

Figure 4 illustrates the loss of direction selectivity among SC cells driven by visual stimuli to the deprived eye, and all of these data are from the binocular segment. A cell was considered to be direction selective if its discharge to stimulus movement in the preferred direction exceeded its discharge to movement in the opposite direction by at least 2:1. By this criterion, 57% (216/384) of SC units had direction selectivity in normal cats (Fig. 4*A;* data from ref 4, 7).

For monocularly activated neurons in the SC of MD cats (Fig. 4*B, C*), the deprived eye clearly drove fewer cells with direction selectivity (19%, 12/63) than did the nondeprived eye (68%, 76/112), and there was also less direction selectivity for the deprived eye than found for normal cats ($P < 0.001$). There is a suggestion that the nondeprived eye activated more direction-selective SC neurons than normal ($P < 0.05$), but as mentioned above this comparison between experiments could suffer from sampling errors. It may be, however, that monocular neurons driven by the nondeprived eye represent a different population than that of all cells in the normal cat (i.e., the normal population includes binocular cells which could include a lowered proportion of direction-selective units). Therefore, while it is clear that for monocularly driven cells in MD cats, the deprived eye drives fewer with direction selectivity, it is not clear whether or not the nondeprived eye has an abnormal proportion (i.e., higher) of direction-selective units.

Data from the binocular neurons in MD cats also indicate a loss of direction selectivity for units driven by the deprived eye. When tested with both eyes open, only 38% (39/95) of these units were direction selective (Fig. 4*D*), a proportion less than

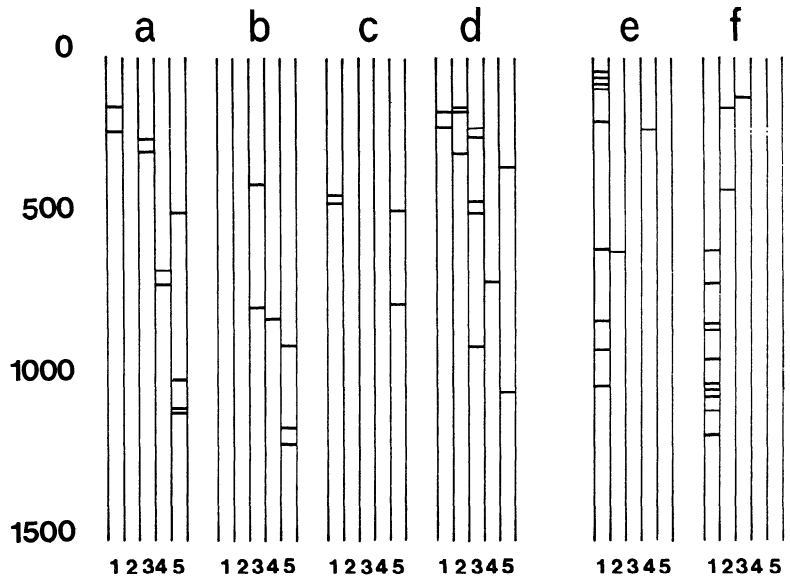


FIG. 3. Representative ocular-dominance distributions in penetrations through the SCs of one MD cat. The horizontal scale indicates five ocular-dominance groups: 1 indicates exclusive drive from the contralateral eye; 2, strong drive from the contralateral eye and weak drive from the ipsilateral eye; 3, equal drive from both eyes; 4, weak drive from contralateral eye and strong drive from ipsilateral eye; 5, exclusive drive from ipsilateral eye. The binocular units in Fig. 2 comprise groups 2–4 of this figure. The vertical scale indicates depth in microns from the SC surface which was determined by the first appearance of background activity. a–d: 33 units recorded in the SC contralateral to the deprived eye. e, f: 24 units recorded in the SC ipsilateral to the deprived eye.

normal ($P < 0.001$). Of considerable interest is the result of testing each eye independently for direction selectivity in 26 binocular units (Fig. 4*E–G*). Of these, direction selectivity was ascertained in 6 cells for either eye, in 15 cells for neither eye, and in the remaining 5 cells only for the nondeprived eye. Examples of this last group were never found in normal cats, and the responses from one such unit are illustrated in Fig. 4*F, G*.

In contrast, no loss of direction-selective units driven by the deprived eye was detected in the monocular segment of the SC. In the normal cat, 50% (17/34) were direction selective, and of units driven by the deprived eye in MD cats, 55% (21/38) were direction selective ($P > 0.05$). These data and those of Fig. 2 would suggest that the effects of early monocular deprivation in the cat's SC are limited to the binocular segment.

*Speed selectivity*

In the normal cat (see Fig. 5*A*, redrawn from Fig. 6 of ref 7) most SC units respond preferentially to slower stimulus speeds ($<50°$/s), although some respond well to fast stimulus speeds as well ($>100°$/s). In a previous experiment (7), responses in the SC to high stimulus speeds were correlated with input from retinal Y-cell axons, and the same correlation was seen in this experiment. Among all groupings of SC units in the MD cats (monocular units driven by either the deprived (Fig. 5*B*) or nondeprived eye (Fig. 5*C*), and binocular units (Fig. 5*D*)), most preferred slower stimulus speeds, and there is no significant difference among these populations ($P > 0.05$). There is some suggestion of a higher proportion of cells preferring fast stimulus speeds in the normal cat, and when this is compared to the other three groups of cells a slight difference is apparent ($P < 0.02$). However, as mentioned above, data from the normal cat were taken from a separate experiment, and comparisons with data in this experiment may not be valid. In any case, there is little if any change in the speed selectivity of SC cells due to monocular deprivation.

## DISCUSSION

Evidence has been presented in this paper for three effects on cells in the cat's SC following early monocular deprivation. Comparing cells driven by the deprived eye either with those driven by the nondeprived eye or with those from normal cats, these effects are: *1*) a large reduction in the Y-indirect input from the deprived eye to the SC, whereas the direct retinotectal input is apparently normal; *2*) a reduced drive of cells by the deprived eye, more severe in the contralateral than in the ipsilateral SC; and *3*) a reduction in the number of directionally selective neurons driven by the deprived eye. Selectivity for stimulus speed apparently was unaffected.

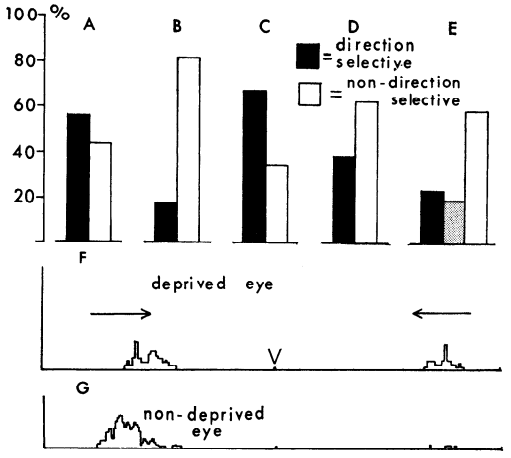


FIG. 4. Direction selectivity of units from the binocular segment of the SC in normal (*A*) and MD cats (*B–G*). The black bars represent direction-selective units (see text), and the open bars represent units lacking direction selectivity. The vertical scale in *A–E* shows the percentage of units in each group. *A:* 384 units from normal cats (data mostly from ref 4). *B:* 73 monocular units driven by the deprived eye. *C:* 112 monocular units driven by the nondeprived eye. *D:* 95 binocular units tested with both eyes open for the same stimuli. *E:* 26 binocular units tested with each eye separately. In this last group, five neurons (indicated by the stippled bar) showed direction selectivity for the nondeprived eye but not for the deprived eye. The black bar represents units with direction selectivity for either eye and the open bar represents units with direction selectivity for neither eye. *F, G:* average response histograms of one of the five units indicated by the stippled bar in *E*. Each histogram consists of 200 bins (100 for each direction of stimulus movement). The bin width is 40 ms, sweep amplitude 40°, and thus the stimulus speed is 10°/s. V indicates the turn-around point in the movement of a 2°-diameter spot of light. *F:* 50 sweeps of the stimulus to the deprived eye. The first bin has 50 counts. *G:* 25 sweeps of the stimulus to the non-deprived eye. The first bin has 25 counts.

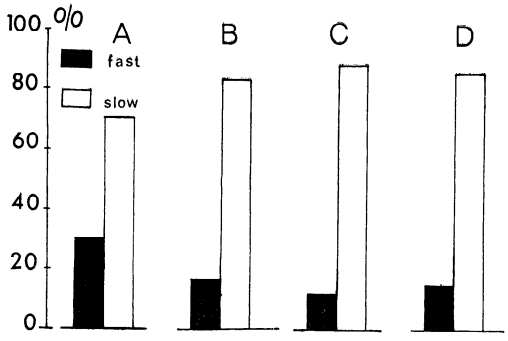


FIG. 5. Selectivity for stimulus speed measured in SC units of normal (*A*) and MD cats (*B–D*). Units responding to speeds well above 100°/s are represented by the black bars, and the open bars indicate neurons responding well only to stimulus speeds below 50°/s. *A:* 115 units from normal cats (data from ref 4 and 7). *B:* 64 units driven only by the deprived eye. *C:* 108 units driven only by the nondeprived eye. *D:* 86 binocular units.

### *Comparison with other studies*

Although much normal data are available for SC units using essentially the same methodology (4, 7), it proved impossible to determine if monocular deprivation affected only the deprived eye's input to SC or if the nondeprived eye's input was also altered. This failure could be due to sampling differences in the different experiments (see RESULTS). For these reasons, a comparison between these data and those of Wickelgren and Sterling (23) becomes somewhat tenuous. Yet it is interesting to point out that where comparable data were compiled (i.e., for direction selectivity and binocular interaction) good agreement exists as to the effects of monocular deprivation.

Wickelgren and Sterling (23) indicate in their study that most of the 23 SC units which were influenced by the deprived eye lacked direction selectivity. The present study quantitates and adds more detail to this reduction of direction selectivity in the responses originating from the deprived eye. It is interesting to note that in monocularly as well as binocularly driven units the direction selectivity has dropped to less

than 20% when tested through the deprived eye (Fig. 4*B, D*). Another 20% of the binocular units were direction selective when tested through the nondeprived eye, but nonselective when tested through the deprived eye, so that this interocular difference could be demonstrated in single SC units (Fig. 4*E–G*). Finally, when both eyes were tested together, such units revealed direction-selective properties, possibly because, for the nonpreferred direction, input from the nondeprived eye via the visual cortex inhibited input from the deprived eye. Some form of inhibition is suggested by the fact that in the nonpreferred direction we consistently found a less brisk response with binocular testing than with monocular testing of the deprived eye.

This study, as well as that of Wickelgren and Sterling (23), indicates a greater decrease in input from the deprived eye to the ipsilateral than to the contralateral SC. These intercollicular differences were perhaps expected, since: *a*) the SC receives visual input from both the retinotectal and corticotectal pathways; *b*) the retinotectal pathway is predominantly crossed whereas the corticotectal is not (cf. ref 13, 18a, 22); and *c*) as shown, monocular deprivation appears to affect the corticotectal (i.e., Y-indirect) but not the retinotectal input. Supporting this explanation of the intercollicular differences is the finding that the loss of input from the deprived eye increases with depth in the superficial gray of the SC (see Fig. 3). With Sterling's report (17) that retinal fiber terminals are particularly concentrated in the upper part of the superficial gray, these data suggest that the effect of deprivation is most severe in the zone where corticotectal fiber terminations predominate, i.e., in the deep part of the superficial gray.

However, there is another possible explanation; namely, that the intercollicular differences reflect interhemispheric differences in visual cortex instead of, or in addition to, the contralateral dominance of the retinotectal pathway. This possibility is raised because of the recent demonstration in MD cats that, for striate cortex units with receptive fields well away from the area centralis, the deprived eye is considerably more effective in driving units in contralateral than in ipsilateral cortex (15). Previous studies not showing interhemispheric differences were largely limited to the area centralis representation in the striate cortex (11, 23, 24). SC units of the present study had receptive-field locations equally spread over the binocular segment, including the area centralis. No differences in ocular dominance distribution with eccentricity of receptive fields were detected. However, the determination of afferent input to the SC in MD cats (Fig. 1) were all made from neurons in the SC contralateral to the deprived eye, and only 2 of 50 units driven by that eye had a Y-indirect input. It thus seems unlikely that the cortex contralateral to the deprived eye provides significantly more of a deprived-eye corticotectal input than does the ipsilateral cortex. Until these various factors are more clearly understood, however, the cause of the intercollicular differences in ocular dominance should remain an open question.

### *Differential effects in binocular and monocular segments*

Previous data indicate that, for MD cats, the deficits in the geniculostriate system are limited to the binocular segment, whereas the monocular segment develops normally. For LGN laminae innervated by the deprived eye, the binocular segment was found to have histologically smaller cells and Y-cells were recorded only rarely. In contrast, the monocular segment appeared histologically normal with the normal proportion of Y-cells (6, 17). For the striate cortex, neurons driven by the deprived eye were rarely found in the binocular segment (24, 25), but were routinely identified in the monocular segment (15). Sherman (14) recently described analogous behavioral results for MD cats, reporting that the deprived eye's deficiencies were limited to the binocular segment of visual field. He suggested that in the MD cat this represented ". . . normal development of the monocular segment of visual cortex (and perhaps also the superior colliculus) . . ." The results presented in this paper tend to confirm this suggestion.

*Development of visual inputs of SC*

Three separate visual inputs have been identified for the normal cat SC (7). Two of these arrive via the retinotectal pathway and include axons of retinal W-cells and Y-cells; these were termed the W-direct and Y-direct pathways. The present study provides no evidence that either of these inputs is affected by early monocular deprivation. The third, Y-indirect, input to the normal SC travels via the LGN and visual cortex. Y-cells in the LGN and a class of cortical complex cells are involved (7, 8, 12, 20). In MD cats, this Y-indirect pathway is largely missing for the deprived eye but intact for the nondeprived eye. The site of disruption of this pathway must remain speculative. It is probably central to the optic tract (17), and could be in the LGN due to the loss of Y-cells there (15). Alternatively, it could be in the visual cortex because geniculocortical synapses on complex, and perhaps other, cells related to the deprived eye fail to develop properly, and this in turn could cause retrograde changes in the LGN which are manifested as a failure to record Y-cells.

Figure 6 summarizes the above hypothesis with the suggestion that the main developmental abnormality in MD cats is located somewhere in the geniculocortical region. Affected are the Y-axons (and perhaps X-axons) plus their postsynaptic complex (and perhaps simple) cells. As a consequence, these complex cells, which are the cells of origin of the corticotectal pathway (12), can no longer be visually or electrically driven from the deprived eye, and the Y-indirect input to the SC is lost for that eye. Since the nondeprived eye maintains normal connections via the geniculocortical system (11, 16, 24, 25), this eye develops a Y-indirect input to the SC. Finally, the deprived eye maintains a Y-

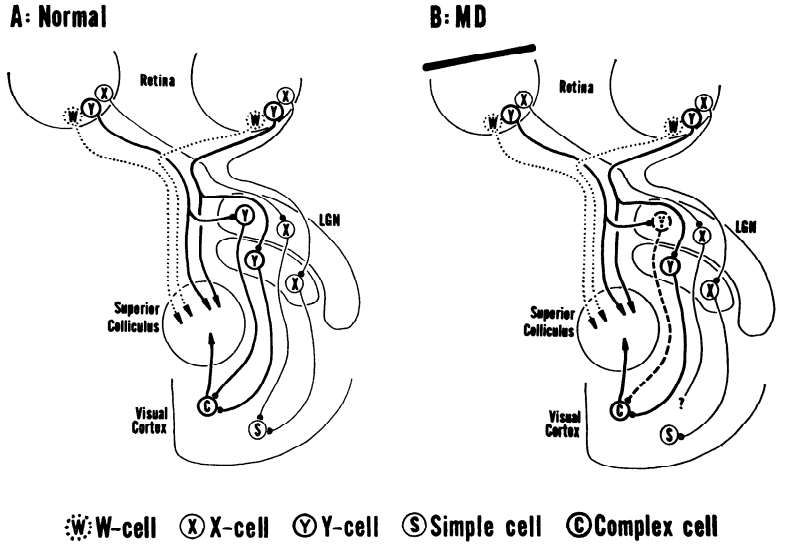


FIG. 6. Schematic diagram of afferents to the SC in normal (*A*) and *MD* cats (*B*). It is stressed that these diagrams are partially speculative. *A:* normal afferentation of the SC. Both eyes contribute to all three pathways: *1*) the W-direct originating from retinal W-cells, *2*) the Y-direct originating from retinal Y-cells, and *3*) the Y-indirect originating from retinal Y-cells and relayed through the LGN Y-cell and the cortical complex cell. The retinal X-cells synapse onto LGN X-cells which, in turn, project to cortical simple cells (see text). *B:* afferentation of the SC in MD cats. All three branches of visual input are maintained to the SC via the right nondeprived eye. For the left deprived eye, the two retinotectal (W-direct and Y-direct) pathways remain intact, but the Y-indirect pathway is lost. This is presumably due to a defect in the geniculocortical portion which is indicated by broken lines. We have placed a question mark in the geniculocortical portion of the deprived eye's pathway involving X-cells, since these cells appear physiologically normal in the LGN, yet very few cortical cells have receptive fields for the deprived eye. The defect shown for the deprived eye is apparently limited to the binocular segment of the Y-indirect pathway since available evidence suggests that the monocular segment for the deprived eye develops normally in MD cats.

indirect (and thus normal) input to the monocular segment of the SC, because in MD cats the geniculocortical system develops normally in this segment (see above and ref 15, 16). This hypothesis implies that in the cat's visual system the major functional effect of early visual deprivation is limited to the geniculocortical portion. Thus the retinogeniculate, retinotectal, and corticotectal pathways develop normally despite early deprivation. Regarding corticotectal development, it is worth noting that BD cats also seem to lose the Y-indirect input despite the fact that electrical activation of their visual cortex drives the SC cells normally (unpublished observations). We stress that this hypothesis is very tentative and that others may well be consistent with the available data.

## CONCLUSIONS

The SC normally receives visual input both from the retinotectal (W-direct and Y-direct) pathway terminating in the upper part of the superficial gray layer and the corticotectal (Y-indirect) pathway terminating in the lower part of this layer (7, 18). Three separate experimental results in this study point to the disruption of corticotectal influences originating from the deprived eye. First, electrical stimulation data indicate that the Y-indirect pathway from this eye is lost. Second, SC neurons driven by the deprived eye were least-frequently recorded in the lower part of the superficial gray where the corticotectal terminals predominate. Third, the receptive-field properties affected by early visual deprivation—ocular dominance and direction selectivity—are those which seem most dependent on the corticotectal pathway (13, 22, 23). Taken together, the available evidence strongly suggests severe disruption of the corticotectal pathway in MD cats.

## SUMMARY

*1.* Previous work has demonstrated three separate visual pathways to the superior colliculus (SC) in cats: *a*) the W-direct retinotectal pathway; *b*) the Y-direct retinotectal pathway; and *c*) the Y-indirect pathway involving retinal Y-cells, LGN Y-cells, and cortical complex cells which send axons into the corticotectal pathway. We investigated the afferentation to the SC in six monocularly deprived (MD) cats by studying electrically and visually elicited responses in 419 neurons.

*2.* The nondeprived eye had grossly normal input to the SC through all three afferent limbs. However, our data suggest the possibility that a slight relative overdevelopment of the Y-indirect pathway may occur for this eye. The three receptive-field features which were tested (ocular dominance, direction selectivity, and speed selectivity) were normal for the nondeprived eye.

*3.* For the deprived eye, the two retinotectal pathways appeared intact, but the Y-indirect pathway was virtually lost. Furthermore, the deprived eye drove fewer cells than normal, and of the cells driven, fewer had direction selectivity. Interestingly, the deprived eye drove many more neurons in the contralateral SC than in the ipsilateral SC. Speed selectivity among units driven by this eye was normal.

*4.* The above abnormalities for the deprived eye were limited to the binocular segment of the SC, since the monocular segment had normal physiological properties.

*5.* A hypothesis is put forward which suggests that in MD cats the developmental abnormalities in visual afferentation to the SC are located in the binocular segment of the geniculocortical system.

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