Effects of Early Binocular Deprivation on Visual Input to Cat Superior Colliculus

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A CAT RAISED with monocular or binocular eyelid suture develops severe abnormalities in its visual system. These include well-documented effects on the histology (1, 6, 7, 24, 25) and physiology (16) of the lateral geniculate nucleus, on the physiology of the striate cortex (1, 15, 25), and on the animal's overall visual capabilities (1, 2, 4, 14).

In contrast to the many studies of deprivation effects on the geniculocortical system of cats, these effects on the superior colliculus have been less completely investigated (cf. ref 20). Hoffmann (8) provided additional control data for such studies by analyzing the visual afferents to the normal cat's colliculus. He determined that at least three pathways are involved: *1*) the W-direct pathway (which innervates 73% of collicular cells) involves retinotectal axons originating from W-cells;[1] *2*) the Y-direct pathway (9% of collicular cells) involves retinotectal axons originating from Y-cells;[1] and *3*) the Y-indirect pathway (18% of collicular cells) involves a cortical loop consisting of a retinal Y-cell, geniculate Y-cell, and cortical complex cell. The complex cell sends an axon into the corticotectal pathway (11). According to Hoffmann (8), X-cells[1] participate in retinogeniculocortical pathways and not retinotectal pathways (see also ref 3).

Recently we used this framework of analysis to study the collicular afferents in monocularly deprived cats (9). We found that afferents originating in the nondeprived eye were substantially normal. For the deprived eye, the W-direct and Y-direct pathways appeared normal, but the Y-indirect pathway was essentially missing. This abnormality, however, could be documented only in the binocular segment of the colliculus (i.e., that portion which maps the central, binocularly viewed segment of visual field). The collicular monocular segment, which maps the peripheral, monocularly viewed crescent of visual field, appeared by receptive-field criteria to be unaffected by the deprivation. We concluded (9) that this effect on the Y-indirect pathway was related to the previously reported (16) decrease in the relative frequency of Y-cells in the geniculate nucleus after monocular deprivation, since: *1*) the proportion of Y-cells driven by the deprived eye is reduced in the binocular segment, but not in the monocular segment of the geniculate; and *2*) geniculate Y-cells form an integral link in the Y-indirect pathway.

In the present study, we extended this analysis to binocularly deprived cats. These, unlike monocularly deprived cats, suffer a severe loss of Y-cells throughout the geniculate nucleus (i.e., in binocular and monocular segments; cf. ref 16). We found a correlated loss of the Y-indirect pathway to the binocularly deprived cat's colliculus. In addition, we found evidence that this loss occurred between the optic tract and corticotectal pathway, since cortical stimulation elicited normal collicular activation.

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[1] Based on electrophysiological criteria, the cat's retinal ganglion cells have been classified into three functional groups, called W-, X-, and Y-cells. X- and Y-cells appear to innervate the geniculocortical pathway in a parallel fashion, and W- and Y-cells project into the retinotectal pathway. For details see text and ref 3, 8, 10, 21.

MATERIALS AND METHODS

Subjects

Five cats which were born and raised in the laboratory were used in this experiment. At 8–12 days of age, each had the lids of both eyes sutured together. Each cat then had both eyes opened at 10–12 mo of age, immediately prior to the terminal study of the superior colliculus.

Electrophysiological methods

We have previously described our methods (8, 9) and will only briefly outline them here. During the recording session, the cats were anesthetized with N_2O/O_2 (60/40), paralyzed with a continuous infusion of Flaxedil (40 mg/h) in saline (6 ml/h), and the corneas were covered with zero-power contact lenses which included a 3-mm-diameter artificial pupil. The eyes were focused with spectacle lenses, if needed, onto a 1-m frontal tangent screen.

We recorded extracellular activity of collicular units using either 4 M NaCl-filled micropipettes or tungsten electrodes varnished with Insl-X. Bipolar stimulating electrodes were placed in the optic chiasm and optic tracts of three of the cats. The remaining two had chiasm electrodes plus three pairs of wire electrodes (1-mm bare tips) inserted 2 mm into the lateral gyrus of visual cortex. The electrodes provided rectangular stimulating pulses of 50–100 μs and up to 30 mA.

We determined the afferent input to each collicular neuron by applying Hoffmann's criteria (8) based on the conduction velocity of the retinofugal axons and the neuron's response latency to orthodromic activation from the chiasm. The former allows a determination of whether retinal W-cells or Y-cells are involved, and the latter, discriminations between the Y-direct and Y-indirect pathways. The conduction velocity was based on the cell's response-latency difference between optic chiasm and tract stimulation plus the measured separation of the two electrode pairs. That is: *1*) the W-direct pathway has a conduction velocity of less than 15 m/s; *2*) the Y-direct pathway has a conduction velocity of greater than 35 m/s and a latency to chiasm stimulation of less than 3 ms; and *3*) the Y-indirect pathway has a conduction velocity of greater than 35 m/s and a latency to chiasm stimulation of more than 3 ms (see ref 8 and 9 for details).

We analyzed each collicular cell's receptive field with conventional techniques by moving visual targets across the tangent screen while monitoring the cell's activity and, for some neurons, we used a computer to prepare poststimulus histograms which relate firing rate to stimulus position (9).

RESULTS

We analyzed the properties of 164 superior collicular neurons from five binocularly deprived cats. Control data from normal cats, collected with identical techniques to those used here, are provided for comparison. Much of the control data has been previously published (8, 9). We found no detectable differences between the effects of deprivation on the 154 cells in the binocular segment of the colliculus and the 10 cells in the monocular segment. Therefore, data below are pooled from both segments.

Electrical stimulation

Stimulating electrodes were placed in the optic chiasm and tract of three of the five deprived cats, and 84 collicular neurons were then studied for afferent input (see MATERIALS AND METHODS). Figure 1*A* (data from ref 8) shows afferent input data from normal cats, and Fig. 1*B* shows this analysis from the deprived cats. Hoffmann (8) found that among 170 collicular neurons in normal cats, 125 had W-direct afferents, 15 had Y-direct afferents, 30 included 12 cells and 18 axons representing the Y-indirect input, and therefore all had identifiable latency patterns to chiasm stimulation. We found in the binocularly deprived cats' colliculi (Fig. 1*B*) that 56 cells (67%) had W-direct input, 2 cells (2%) had Y-direct input, 0 cells (0%) had Y-indirect input, and 26 cells (31%) had no identifiable input. This is statistically[2] different ($P < 0.001$) from the normal distribution, and indicates that binocular deprivation produces: *1*) no change in the W-direct input; *2*) a moderate loss of Y-direct input; *3*) a severe loss of Y-indirect input; and *4*) a concomitant appearance of many neurons with no identifiable input from the optic chiasm.

From the above and previous work (9), we concluded that one substantial result of early visual deprivation is a failure of

[2] Unless otherwise noted, the χ^2-test is used for all statistical analyses in this paper.

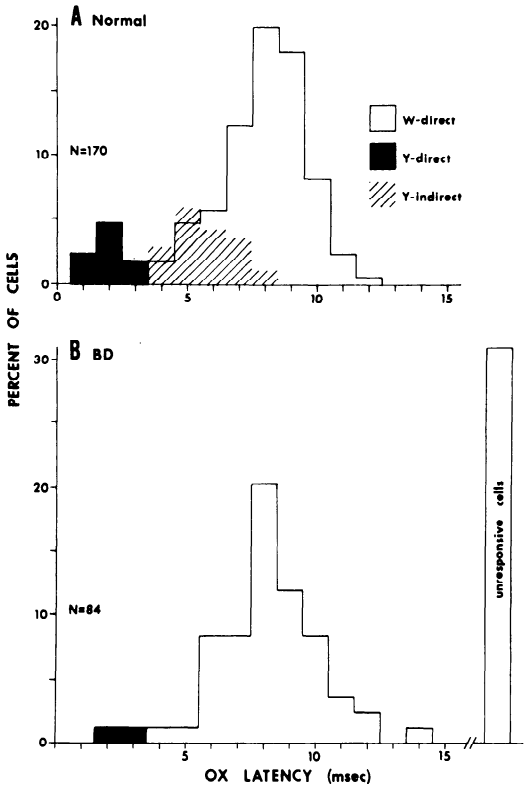

FIG. 1. Histograms showing the relative latency-frequency distribution for neurons of the superior colliculus after electrical activation of the optic chiasm (OX) in normal (*A*) and binocularly deprived (*B*) cats. In each histogram, the horizontal axis represents the latency of collicular cell discharge in milliseconds, and the vertical axis shows the percentage of cells in each 1-ms-wide latency group. The afferent to each neuron was classified according to both the retinofugal conduction velocity and the neuron's discharge latency to OX stimulation (see text) as: W-direct (open bars), Y-direct (black bars), and Y-indirect (hatched bars). *A*: 170 collicular neurons recorded from normal cats (data from ref 8). *B*: 84 collicular neurons recorded from binocularly deprived cats. Of these neurons, 26 were unresponsive to OX stimulation.

the Y-indirect pathway to develop input to collicular neurons. Given the normal distribution of Y-cells in the optic tract of deprived cats (17), this could represent a failure of the Y-indirect pathway at the retinogeniculate, the geniculocortical, and/or the corticotectal synapse. To test for this final possibility, we applied electrical stimuli directly to the visual cortex and optic chiasm in the remaining two deprived cats, and monitored activity to these stimuli in 51 collicular neurons.

Of these 51 neurons, 43 (84%) responded with action potentials to electrical stimulation of cortex. For purposes of comparison, we recorded from 30 collicular neurons in a normal cat and used identical techniques of electrical stimulation. We found that 24 of the 30 cells (80%) responded to cortical stimulation so that the responsive population of collicular neurons in the binocularly deprived cats seems normal.

Figure 2*A* shows the latency to cortical stimulation of activity in the 43 collicular neurons from the two deprived cats. The black bars represent 11 cells which were responsive to cortical but not to chiasm stimulation. Of these, 5 cells had clear receptive fields mapped with visual stimuli. The remaining 6 cells had no detectable visual fields, although neighboring cells did. Figure 2*B* shows the relationship between latencies to chiasm and cortical shock in the remaining 32 cells and it indicates a weak, positive correlation. A similar correlation obtained in normal cats ($r = +0.69$, $P < 0.001$, N = 30) (unpublished data). Note that the chiasm-unresponsive neurons of Fig. 2*A* cluster at the short latency end of the histogram. This plus the correlation of Fig. 2*B* suggests that these neurons might be expected to have short-latency (i.e., Y-direct) retinal input.

Two of the neurons indicated in Fig. 2*B* had sufficiently short chiasm latencies to be classified as having Y-direct input (see MATERIALS AND METHODS and ref 8, 9). Most or all of the remainder had such long latencies that they were presumably activated by W-direct input since Y-indirect input is functionally missing in these animals (see above).

Of the eight cells visually driven but unresponsive to cortical shock, only two were driven from the chiasm, at latencies of 8 and 9 ms, respectively. It is interesting that in the five binocularly deprived cats, 43 of 135 collicular cells (32%) were unresponsive to chiasm shock, but only 8 of 51 (16%) were unresponsive to cortical shock. In the normal cat, each of 170 cells was driven by chiasm stimulation (ref 8) and only 6 of 30 cells (20%) failed to respond to cortical shock.

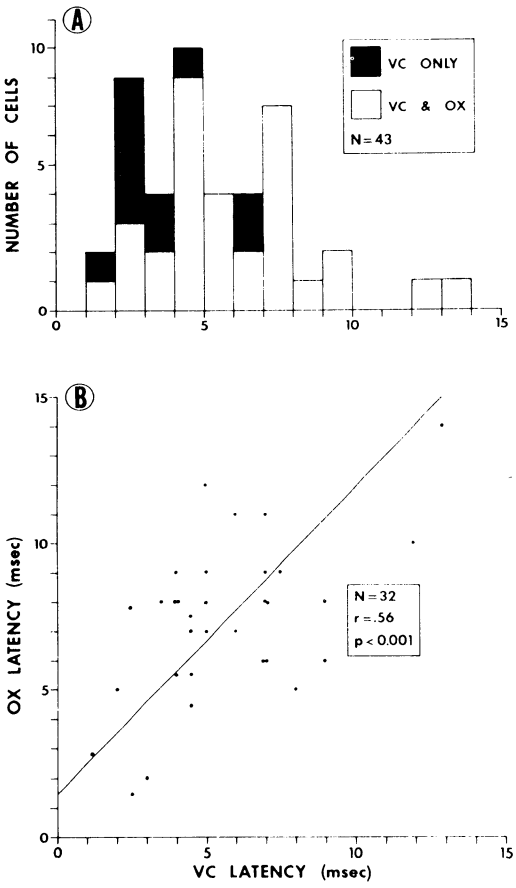

FIG. 2. Response latencies of collicular neurons in binocularly deprived cats to electrical activation of the visual cortex (VC). *A*: 43 cells activated by VC stimulation. The horizontal axis represents the latency in milliseconds of collicular neuronal discharge to VC stimulation, and the vertical axis gives the number of cells in each 1-ms-wide latency group. Open bars: 32 neurons responsive to stimulation of both VC and optic chiasm (OX). Black bars: 11 neurons responsive to stimulation only of VC and not of OX. *B*: graph showing the positive correlation between latencies to OX and VC stimulation for the 32 collicular neurons responsive to both stimuli. VC latencies are plotted on the horizontal axis in milliseconds; OX latencies, on the vertical axis. The line of best fit is shown with a slope of 1.04 and a Y-intercept of 1.5 ms ($r = +0.56, P < 0.001$).

In summary, these data indicate that for binocularly deprived cats: *1*) collicular neurons are responsive in a normal fashion to cortical shock; *2*) many fewer such neurons respond to chiasm shock, presumably representing the loss of Y-indirect and, to a lesser extent, Y-direct input. This, in turn, suggests that the breakdown in the Y-indirect pathway occurs between the chiasm and cortex, i.e., either at the retinogeniculate or geniculocortical synapse.

Visual stimulation

Receptive-field properties were studied in 164 collicular neurons in the five binocularly deprived cats. These include the 135 cells described above plus 29 cells which were lost after some receptive-field data were gathered but before electrical stimuli were applied. For receptive fields, we concentrated on ocular dominance, direction selectivity, and selectivity for stimulus speeds.

OCULAR DOMINANCE. Collicular cells in the normal cat are driven nearly equally by either eye (Fig. 3*A*, data redrawn from ref 8; see also ref 12, 19). However, as Fig. 3*B* illustrates, the contralateral eye dominates in the binocularly deprived cat's colliculus. Of 142 neurons tested, 58 (41%) were driven exclusively by the contralateral eye and 24 (17%) were clearly dominated by that eye. Twenty-seven cells (19%) were equally driven from either eye. Only 9 cells (6%) were driven more strongly or exclusively by the ipsilateral

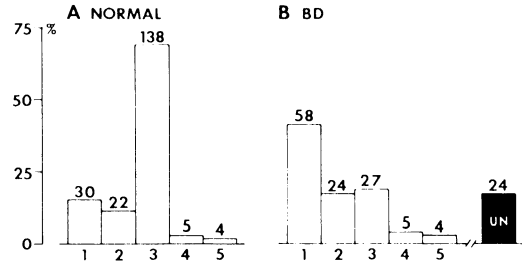

FIG. 3. Ocular dominance distributions for collicular neurons from normal (*A*) and binocularly deprived (*B*) cats. For each, the horizontal axis indicates the following ocular dominance groups: 1, neurons activated exclusively by the contralateral eye; 5, neurons activated exclusively by the ipsilateral eye; 2, 3, and 4, binocularly activated neurons such that 2 represents neurons dominated by the contralateral eye, 3 represents neurons driven nearly equally well by each eye, and 4 represents neurons dominated by the ipsilateral eye. The vertical scale shows the percentage of 1-, 2-, 3-, 4-, or 5-type neurons. *A*: 199 neurons from the colliculus of normal cats. *B*: 142 neurons from the colliculus of binocularly deprived cats. Of these, 24 were unresponsive to visual stimuli.

eye. Also, 24 cells (17%) had no detectable receptive field for either eye, a class of cells found neither in the normal colliculus nor in the colliculus of monocularly deprived cats (9, 23). The deprived cats' ocular dominance distribution for collicular neurons reported here is significantly different from normal ($P < 0.001$), and is in substantial agreement with the data of Sterling and Wickelgren (20).

DIRECTION SELECTIVITY. Direction selectivity was determined in 147 collicular neurons having clear receptive fields in the deprived cats. We used our previous criteria for direction selectivity. That is, the response in the preferred direction must be at least twice that in the opposite direction for the cell to be classified as direction selective (8, 9). Figure 4*A* shows that in the normal cat, 216 of 384 collicular neurons (57%) displayed direction selectivity. However, only 26 of 147 (18%) were direction selective in the deprived cat (see Fig. 4*B*), and this is lower than normal ($P < 0.001$). We saw considerable variation in the proportion of direction-selective cells among the five binocularly deprived cats. The values for individual animals were 35% (12 of 30), 27% (8 of 30), 18% (2 of 11), 12% (2 of 15), and 3% (2 of 61). As in the normal cat, most of these directionally selective neurons (22 of 26) preferred movement directed away from the area centralis. Sterling and Wickelgren (20) also reported a reduction in directionally selective collicular neurons following binocular deprivation.

SPEED SELECTIVITY. On the basis of their speed preference for moving stimuli, collicular neurons were placed into one of four categories: *a*) cells responding optimally to stimuli moving at speeds less than 5°/s; *b*) cells responding well to stimuli at 5–50°/s; *c*) cells responding well to stimuli at speeds up to 100°/s; and *d*) cells responding briskly to stimuli moving at speeds well above 100°/s. Figure 5 shows that for 115 cells from normal cats, about half of the collicular neurons fall into category *a*, whereas the great majority of cells (92 of 119; 75%) in the binocularly deprived cats preferred this slowest range of stimulus movements. Only 4 cells (3%) in the binocularly deprived cats were found to respond to stimulus speeds well over 100°/s, whereas in normal cats, 22 cells (19%) responded to such stimuli. This represents a significant abnormality

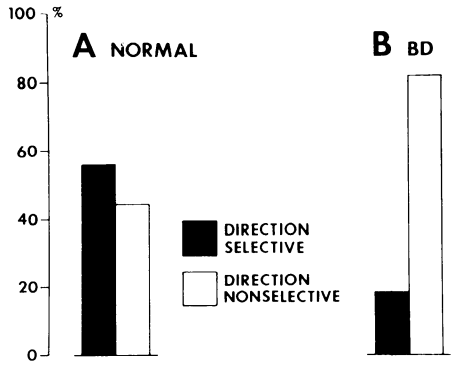

FIG. 4. Proportion of direction-selective neurons from the superior colliculus of normal (*A*) and binocularly deprived (*B*) cats. The black bars represent direction-selective cells (see text for definition), and the open bars represent cells responding nearly equally well for all directions of stimulus movement. The vertical scale shows the percentage of neurons in each group. *A*: 384 collicular cells from normal cats (data mostly from ref 8). *B*: 147 collicular cells from binocularly deprived cats.

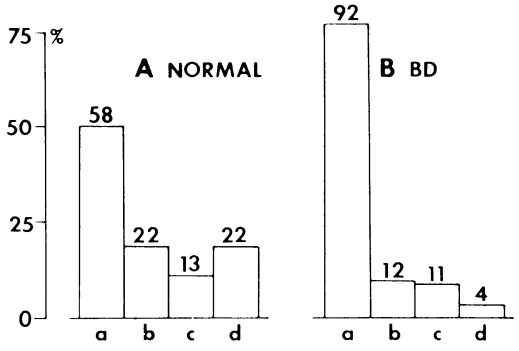

FIG. 5. Selectivity for speed of stimulus movement measured in superior collicular neurons of normal (*A*) and binocularly deprived (*B*) cats. For each, the horizontal axis indicates the preferred stimulus speed groupings as follows: a, neurons preferring speeds of less than 5°/s (in binocularly deprived cats, these cells mostly responded only to stimulus speeds less than 1°/s); b, neurons preferring speeds of 5–50°/s; c, neurons responding to speeds up to 100°/s; and d, neurons responding to speeds well over 100°/s. The vertical scale gives the percentage of a, b, c, or d neurons in each group. *A*: 115 collicular neurons from normal cats. *B*: 119 collicular neurons from binocularly deprived cats.

in speed selectivity for the deprived cats ($P < 0.001$).

DISCUSSION

In the five binocularly deprived cats, we found that the population of collicular neurons had several electrophysiological deficits. First, whereas the W-direct pathway seemed normal, the Y-indirect input was entirely missing and the Y-direct input was unusually small. Second, this neuron population displayed receptive-field abnormalities including: *1*) an ocular dominance distribution strongly biased toward the contralateral eye; *2*) a reduced number of directionally selective cells; *3*) few neurons which responded to any but the slowest stimulus speeds; and *4*) a class of neurons without detectable responses to visual stimuli.

Site of deficit in Y-indirect pathway

On the basis of collicular neuronal responses to optic chiasm and tract shock, we determined that the Y-indirect pathway was missing from binocularly deprived cats. This pathway involves the following chain of neurons and synapses: retinal Y-cell to geniculate Y-cell to cortical complex cell (the corticotectal neuron) to collicular cell (see introduction and ref 8). Since the optic tract in these cats has the normal complement of Y-cells (17), the deficit in the Y-indirect pathway must occur central to this structure. Furthermore, the corticotectal limb of the pathway seemed grossly normal since collicular neurons in deprived cats were activated in a normal fashion by electrical stimulation to cortex (of course, this does not rule out potentially major corticotectal abnormalities which were not tested). Therefore, the major deficit may occur between the optic tract and cortex. The simplest explanation is that the defect in the Y-indirect pathway in binocularly deprived cats is a consequence of the loss of geniculate Y-cells (16), since these form an integral link in the pathway (see also Fig. 6*C*). Interestingly, W. Singer (personal communication) in a study of area 17 and 18 in normal and binocularly deprived cats, found that the deprivation resulted in an abnormally small percentage of cortical neurons which could be activated by stimulation of the chiasm or optic radiation.

This implies that, in binocularly deprived cats, the superior colliculus is functionally decorticate for retinal stimulation since retinotectal but not retinogeniculocorticotectal pathways are intact. It is interesting to note the similarities in collicular receptive-field deficits in binocularly deprived and normally reared but decorticate cats. Both have collicular fields with reduced direction selectivity and contralateral eye dominance (cf. ref 12, 20, 22).

We emphasize the speculative nature of the above conclusion and, in fact, note at least one incongruity. In these deprived cats, no Y-indirect pathway was detected, yet about one-fifth of the geniculate Y-cells survived deprivation (16). This could mean: *1*) that further deficits occur between these surviving Y-cells and the colliculus; or *2*) that the surviving Y-cells are a subset not involved in collicular afferentation. Furthermore, the Y-indirect input may not be the only corticotectal pathway since many collicular neurons respond at relatively long latencies to cortical stimulation (8).

Comparison of decorticate and binocularly deprived cats

The receptive-field properties of collicular cells in cats with lesions of visual cortex are markedly similar to those of binocularly deprived cats, presumably because the loss of the Y-indirect pathway due to deprivation functionally mimics decortication. Lesions of visual cortex produce a shift in the ocular-dominance distribution of the collicular neurons in favor of the contralateral eye, and this shift closely resembles that seen in binocularly deprived cats (12, 20, 22). Likewise, the proportion of directionally selective collicular neurons is roughly 60% in normal cats, but only 10% and 18%, respectively, in decorticate and binocularly deprived cats.

Despite these changes in ocular dominance and proportion of directionally selective neurons, decortication produced

no obvious shift in neuronal speed sensitivity among collicular neurons. Of 283 neurons in decorticate cats, 40 (14%) responded well to stimulus speeds of over 100°/s (unpublished data). As can be seen from Fig. 5, this proportion is not significantly less than normal but is significantly greater than that for binocularly deprived cats ($P > 0.05$ and $P < 0.01$, respectively). Hoffman (8) concluded that the Y-inputs to colliculus (direct and indirect) provide sensitivity to fast stimuli. The Y-indirect pathway is abolished in the decorticate and deprived cat. However, because the Y-direct input presumably is unaffected by decortication but is reduced by binocular deprivation, sensitivity to fast stimuli is retained more completely for the former than for the latter cat. A similar conclusion obtained after monocular deprivation. That is, the deprived eye lost Y-indirect input to the colliculus while Y-direct input was unaffected, and a normal proportion of neurons driven by that eye displayed sensitivity to fast stimuli (9).

Similarities between monocularly and binocularly deprived cats

We have previously described deficits among collicular neurons in monocularly deprived cats (9), and it is interesting to compare these deficits to those described in the present account. Three main anomalies were noted for the deprived eye in monocularly reared cats: *1*) a severe reduction obtained in the Y-indirect input, while the retinotectal W- and Y-direct pathways seemed normal; *2*) fewer neurons were activated by visual stimulation, but among those driven by the deprived eye, more were in the colliculus contralateral than ipsilateral to that eye; and *3*) a reduction in the proportion of directionally selective neurons was seen.

These abnormalities also comprise the most obvious collicular deficits in the binocularly deprived cas, a fact which suggests related developmental mechanisms consequent to both types of deprivation. These similarities between cats are obvious in the lack of Y-indirect input and reduced direction selectivity, but they are more subtle for ocular dominance. In binocularly deprived cats, the dominance of the contralateral eye resembles the dominance of the contralateral over the ipsilateral input following decortication (12, 22), and this presumably obtains from the preponderance of the contralateral portion of the retinotectal pathway (18). In monocularly deprived cats, the deprived eye has stronger receptive-field input to the contralateral than to the ipsilateral colliculus, and this, too, probably reflects this contralateral retinotectal preponderance (18) since retinotectal inputs from the deprived eye seem normal.

Differences between monocularly and binocularly deprived cats

Despite the above similarities, some developmental differences between monocularly and binocularly deprived cats are suggested by certain important differences in their collicular deficits. One difference is that the deficits in the monocularly deprived cat seemed limited to the binocular segment, while after binocular deprivation, the monocular and binocular segments of the colliculus had apparently equal deficits. This difference is most easily explained in terms of the different effects of the two types of deprivation on the lateral geniculate nucleus and striate cortex. Y-cells appear to be functionally missing throughout the geniculate following binocular deprivation, but are missing only from binocular segment of deprived laminae after monocular deprivation (16). Many neurons throughout the visual cortex of binocularly deprived cats seem unresponsive (1, 25). However, in monocularly deprived cats, the deprived eye drives few cells in the binocular segment (1, 15, 25) but many in the monocular segment (15).

Another difference between collicular effects of the two forms of deprivation is seen in the neurons' visual responsiveness to varying stimulus speeds. This responsiveness seems normal for cells driven via the deprived eye after monocular deprivation but in binocularly deprived cats, these neurons were generally unresponsive to moderate or high speeds. This receptive-field difference may be related

to a possible difference in the Y-direct input. After monocular deprivation, there was no sign of a reduction of Y-direct input from the deprived eye, while there may have been such a reduction with binocular deprivation. We emphasize the possibility of sampling errors in such comparisons (9), but the proportion of collicular neurons with Y-direct input is less with binocular deprivation than is associated with the deprived eye following monocular deprivation ($P < 0.001$). Given the loss of Y-indirect input with both forms of deprivation, this suggests that the binocularly deprived cat has nearly only W-direct fibers to the colliculus, whereas the deprived eye in monocularly deprivation has W-direct plus Y-direct fibers. As is noted above, this difference would subserve the difference in stimulus-speed sensitivity.

Finally, if several assumptions are permitted, the ocular-dominance distributions reveal interesting differences in the effects of both types of deprivation (see also ref 9, 20). In the binocularly deprived cats, over four-fifths of the collicular neurons had detectable receptive fields, and this suggests that the presence of such fields is not overly dependent on corticotectal input since these cats have no Y-indirect (and thus less or no visually responsive corticotectal) pathway. In the colliculus contralateral to the nondeprived eye of monocularly deprived cats, more than 90% of the cells were driven solely by that eye. Yet in the other colliculus, almost 50% of the neurons were driven solely by the nondeprived eye despite the apparently normal retinotectal pathway. Only the Y-indirect (and thus corticotectal) pathway showed such preference for the nondeprived eye, and this, in turn, suggests a relatively major role for corticotectal input in determining the presence of receptive fields. From other data, Wickelgren and Sterling (23) also concluded that the corticotectal pathway in monocularly deprived cats strongly affected the collicular ocular dominance pattern. This leads to the conclusion that the corticotectal pathway seems much more important to collicular ocular dominance in monocularly deprived cats than in binocularly deprived cats.

CONCLUSIONS

Figure 6 summarizes the major conclusions from this study of binocularly deprived cats plus previous data from normal (8) and monocularly deprived cats (9). The major collicular afferentation (W-direct, Y-direct, and Y-indirect) is shown for normal, monocularly deprived, and binocularly deprived cats. Since X-cells play no obvious role in collicular afferentation (3, 8), they have been omitted from consideration here. Figure 6*A* shows the normal disposition of the three collicular inputs. We have indicated the Y-indirect as the "dominant" input to the colliculus for receptive-field properties since many of these (i.e., binocularity and direction selectivity) depend on corticotectal integrity (12, 22). Figure 6*B* shows for the monocularly deprived cat the normal inputs from the nondeprived eye and the loss of Y-indirect (but not W-direct or Y-direct) input from the deprived eye. In this case, the dominant input is the Y-indirect (as it is in the normal cat) for reasons given above. Figure 6*C* shows the total loss of Y-indirect input in binocularly deprived cats, and the Y-direct pathway is shown as possibly abnormally small. Because of this and the relatively minor role played by the corticotectal pathway in ocular dominance (see above), the W-direct pathway is indicated as the dominant input. The loss of Y-indirect input to the colliculus from the deprived eyes can be related to the associated decrease in the relative frequency of geniculate Y-cells and/or to the cortical deficits associated with visual deprivation.

This question of input dominance merits further speculation. The corticotectal pathway develops dominance in the normal and monocularly deprived cat, but not in the binocularly deprived cat. Perhaps this dominance is determined by early competition between retinotectal and corticotectal pathways, a competition analogous to the binocular competition indicated in the geniculocortical system (5, 15). It has already been suggested that the geniculocortical system in normal and monocularly deprived cats develops by way of the normal mechanism of binocular competition, while the geniculocortical

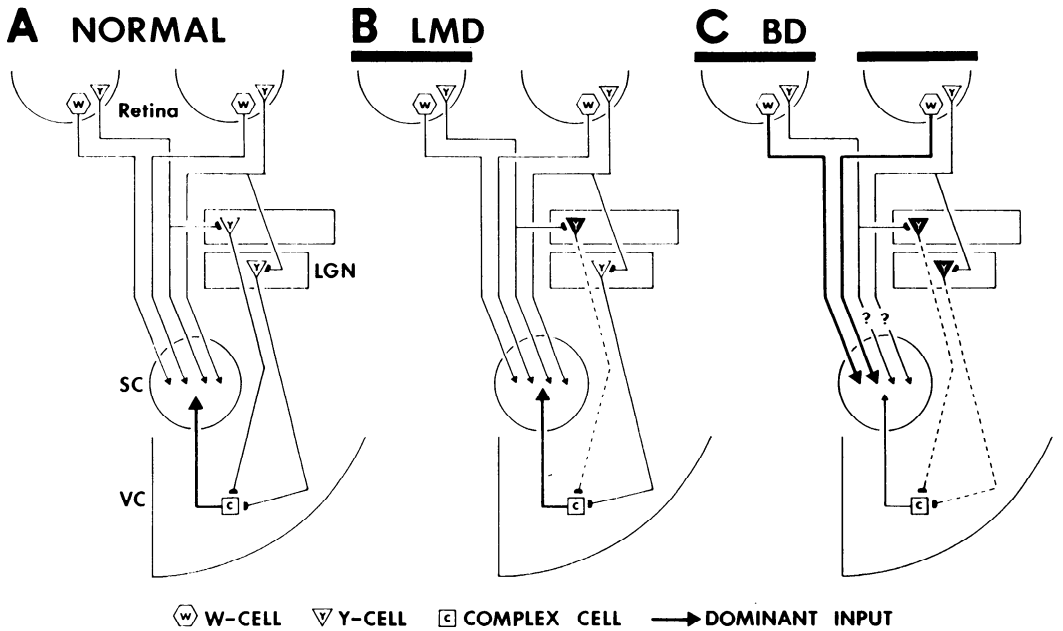

FIG. 6. Summary schematic diagrams showing afferents to the superior colliculus (SC) in a normal (*A*), a left monocularly deprived (*B*), and a binocularly deprived (*C*) cat. These diagrams are hypothetical explanations of much of data from this and related studies. W-cells, Y-cells, and complex cells are shown as indicated. Unrecordable Y-cells in the lateral geniculate nucleus (LGN) are shown as black. X-cells and cortical simple cells are omitted since they play no major or obvious role in collicular afferentation. See text for details. *A*: pathways to the SC in a normal cat. Each eye provides W-direct and Y-direct retinotectal input plus Y-indirect corticotectal input. The last involves Y-cells from retina and LGN plus complex cells from visual cortex (VC). The Y-indirect input seems to dominate many neuronal properties in the SC. *B*: pathways to the SC in a left monocularly deprived cat. All three inputs (W- and Y-direct plus Y-indirect) are evident from the nondeprived (right) eye, and the retinotectal inputs (W- and Y-direct) are normal from the deprived (left) eye. However, the deprived eye provides no Y-indirect input because Y-cells are no longer available in the LGN. Although not illustrated, this deficit is limited to the binocular segment of the LGN. As in the normal cat, the Y-indirect input (only from the nondeprived eye in the binocular segment) is dominant among afferents to the SC. *C*: pathways to the SC in a binocularly deprived cat. From each eye, the W-direct input is normal, the Y-direct input is probably reduced, and the Y-indirect input is completely missing. The Y-indirect input loss is due to a loss of recordable Y-cells throughout binocular and monocular segments of the LGN. However, unlike normal and monocularly deprived cats, the W-direct pathway dominates among afferents to the SC.

system in binocularly deprived cats generally fails to develop (13, 15). Perhaps this normal form of cortical development in normal and monocularly deprived cats leads to or is related to the eventual corticotectal dominance, and the general lack of cortical development results in retinotectal dominance. Therefore, this difference in input dominance between monocularly and binocularly deprived cats may be a reflection of another major difference between them in terms of developmental mechanisms (13).

SUMMARY

1. Recent work has demonstrated at least three distinct inputs to the superior colliculus in normal cats: *a*) the W-direct retinotectal pathway; *b*) the Y-direct retinotectal pathway; and *c*) the Y-indirect pathway which involves Y-cells in retina and lateral geniculate nucleus plus complex cells in cortex, the last being the corticotectal cells.

2. We investigated these inputs in five cats raised with binocular eyelid closure by studying the electrophysiological properties of 164 collicular neurons. After such binocular deprivation, the Y-indirect pathway was missing and the Y-direct pathway appeared reduced, although the W-direct input seemed unaffected.

3. Despite the loss of the Y-indirect input, collicular activation to electrical

stimulation of cortex seemed normal in these cats. This suggested that the Y-indirect loop was affected between the optic tract and cortex, and this, in turn, correlated to the previously described reduction in recordable Y-cells from the lateral geniculate nucleus of binocularly deprived cats.

4. We found receptive-field correlates to this loss of Y-direct and Y-indirect input in the binocularly deprived cats. Compared to collicular neurons in normal cats, those in deprived cats exhibited abnormally strong dominance by the contralateral eye, loss of directional selectivity, and loss of responsiveness to fast visual stimuli.

5. These and other data lead to the suggestion that in normal and monocularly deprived cats, the corticotectal input dominates collicular receptive-field properties, whereas in binocularly deprived cats, the remaining retinotectal input dominates these properties.

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