# Loss of a Specific Cell Type From Dorsal Lateral Geniculate Nucleus in Visually Deprived Cats

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CATS REARED under various conditions of visual deprivation are deficient in their subsequent performance of certain visual tasks (6, 19). Wiesel and Hubel (15–17, 25–28) have sought the physiological basis of these effects by comparing receptive-field properties of single visual neurons of normally reared cats to those of visually deprived cats. Their consistent finding (17, 26–28), confirmed by others (7), is that cells of the striate cortex develop permanently abnormal receptive-field properties during deprivation rearing. In a preliminary study, Wiesel and Hubel (25) observed that cells of the dorsal lateral geniculate nucleus (LGNd) in visually deprived cats have essentially normal receptive fields despite the loss of many large cells in this nucleus. These findings have been substantially confirmed (11, 24). The present study follows a series of recent papers which note the presence of two functionally distinct types of cell in the cat's retina and LGNd: the X-cells (3, 13) (type II (5, 21) or sustained cells (2)) and the Y-cells (3, 13) (type I (5, 21) or transient cells (2)). This paper presents evidence that one effect of rearing cats with visual deprivation (achieved by neonatal eyelid suture) is the selective elimination from the LGNd of Y-cells. The remaining neurons appear to be functionally normal.

## MATERIALS AND METHODS

### *Subjects*

Seven cats, born and reared in the laboratory, were used in this experiment. They were chosen from seven different litters. All were reared under conditions of visual deprivation achieved by a previously described eyelid-suturing technique (25). Three of the cats were binocularly deprived (BD cats) by suturing both eyelids; the remaining four were monocularly deprived (MD cats) by suturing either the left (three cats) or right (one cat) eyelids. The deprivation period, which lasted from the 8th postnatal day for 8–12 months, included all of the "critical period" as defined by Hubel and Wiesel (17).

### *Physiological recording*

Conditions and techniques for single-unit recording, receptive-field analyses, and latency measurements were identical to those of the previous study (13) with the following three minor differences: *1*) all these cats underwent a bilateral cervical sympathectomy (20) just prior to single-unit recording to allow direct comparison of their optic disc projections with those of a previous study (23), *2*) the changes in responsiveness of LGNd neurons following optic chiasm (OX) stimulation were not studied in detail, and *3*) coronal histological sections were prepared from two of the MD cats and one of the BD cats.

### *Measurement of interocular alignment*

Interocular alignment is defined as the alignment of the visual axes and was measured in the cats both while conscious (by the pupil-corneal reflex method) and while paralyzed and anesthetized (by their optic disc projections) using techniques already described (23). Interocular alignment measured under these conditions provided as assessment of the presence or absence of strabismus (23). Three of the MD cats and two of the BD cats had their eyelids parted under pentabarbitone anesthesia 1–3 weeks prior to physiological recording to allow a sufficient period for measurement of their interocular alignments during consciousness. The remaining cats (one MD and one BD) had their eyelids parted minutes before commencement of the physiological recording.

Received for publication December 27, 1971.

## RESULTS

### *Interocular alignment*

All seven visually deprived animals showed evidence of strabismus (i.e., abnormal alignment of the visual axes), in close agreement with a previous report (23). Two BD cats were observed during consciousness and had a divergent strabismus, and all three BD cats had, after anesthesia and paralysis, a divergent misalignment of their optic disc projections. Of the three MD cats observed during consciousness, two had a divergent strabismus and a corresponding divergent misalignment after anesthesia and paralysis; the third had a convergent strabismus and a convergent misalignment after anesthesia and paralysis. The fourth MD cat had a convergent misalignment after anesthesia and paralysis.

### *Single-unit recording*

A total of 260 LGNd neurons from the seven visually deprived cats were studied by single-unit recording. Of these, 57 (see Fig. 5) were positively identified as relay cells, being activated both orthodromically by electrical stimulation of the optic chiasm (OX stimulation) and antidromically by electrical stimulation of the striate cortex (VC stimulation). The other 203 cells were activated only by OX stimulation, but had properties typical of relay cells, including appropriate OX latencies (see below) and concentric on- or off-center receptive fields; they are assumed, as in the previous paper (13) to be relay cells. All 260 units could be classified as either X-cells (having X-fields) or Y-cells (having Y-fields) by the criteria presented previously (13). As in the normal cat (13) a small number of units was found whose properties were distinct from those of relay cells, and these are not further considered here.

Visual deprivation dramatically reduced the percentage of Y-cells among neurons driven by the deprived eyes and two features of this result are elaborated in the following sections. First, whereas a paucity of Y-cells was seen in LGNd's of both MD and BD cats, the distribution of remaining Y-cells differed markedly between MD and BD cats. Second, all the LGNd neurons studied had properties within the normal range established in the preceding paper (13).

### *Relative frequency of X- and Y-fields in MD cats*

The receptive fields of 158 units were studied in MD cats, 89 from the "deprived" laminae and 69 from the "nondeprived" laminae. (Deprived laminae receive direct retinal afferents from the visually deprived eye, nondeprived laminae from the nondeprived eye.) Units were recorded in the LGNd both contralateral (three cats) and ipsilateral (one cat) to the deprived eye (see Fig. 3). Since no interlaminar differences in the effects of deprivation were detected, data obtained from the different laminae are pooled in Figs. 1, 2, 4 and 5, and Table 1.

Figure 1*A* and *B* shows the positions in the visual field of all the receptive fields studied in MD cats. Fields of units driven by the nondeprived eye (nondeprived eye fields) are shown in Fig. 1*A*. Figure 1*B* represents the deprived eye fields. X-fields are represented by open circles and Y-fields by closed circles. For simplicity all fields are presented as if recorded from the left LGNd and from the right eye, although both eyes and both LGNd's were used. Part of the peripheral limit of the binocularly viewed visual field is indicated in each section of Fig. 1, *A–C*, as a line which is vertical near the zero horizontal parallel and curves toward the zero vertical meridian in the lower portion of the visual field. The shape and position of this line were derived from Sanderson's (22) LGNd maps by estimating the lateral limit of the portion of the visual field which projects to lamina $A_1$ at different coronal levels. Areas of the visual field nasal to this limit (to the left of it in Fig. 1) are represented in the medial, laminated portion of the LGNd, while areas temporal to this limit (to the right of it) are represented in the lateral, unlaminated portion of the LGNd. Following previous conventions (11, 13) these are referred to, respectively, as the binocular segment and monocular segment of the visual field and the binocular seg-

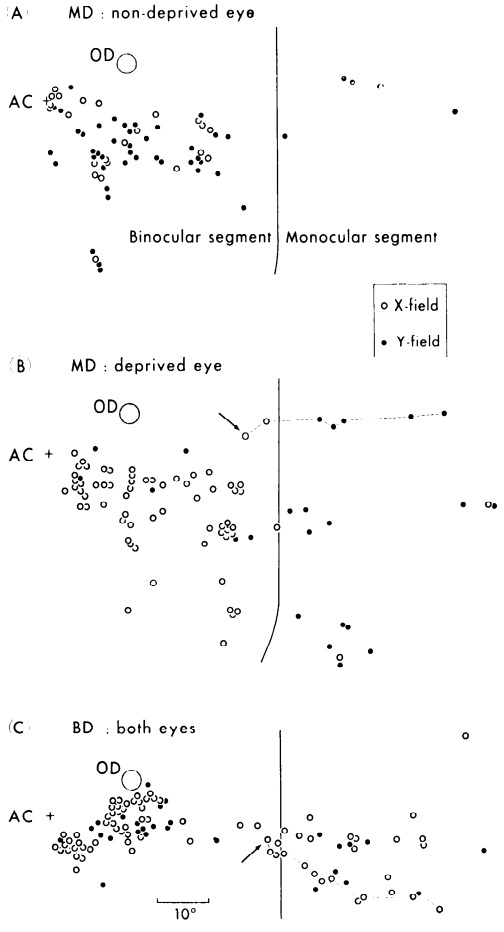


FIG. 1. Distribution of locations in the visual field of all 260 receptive fields plotted in this study. X-fields are indicated by open circles; Y-fields, by filled circles. For simplicity, the figure has been drawn as if all fields were found in the left LGNd and driven by the right eye, although fields from both eyes and both LGNds are, of course, included. Also shown are projections of the optic disc (OD) and area centralis (AC), which were determined by methods described in the preceding paper (13), and the border between the binocular and monocular segments of the visual field which was derived from Sanderson's (22) LGNd maps (see text). *A:* nondeprived eye receptive-field locations in MD cats. *B:* deprived eye receptive-field locations in MD cats. Fields joined by a dashed line represent the sequence of units encountered along the electrode penetration indicated by an asterisk in Fig. 3*A*. The first unit encountered in this penetration is marked by an arrow. *C:* receptive-field locations in BD cats. The sequence of units encountered along a single penetration is indicated as for *B*.

ment and monocular segment of the LGNd.

Figure 2 summarizes the data of Fig. 1

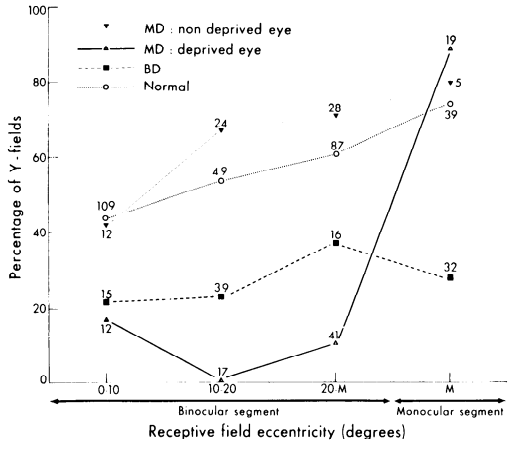


FIG. 2. Same data as in Fig. 1 showing the variation with eccentricity in the visual field of the frequency of Y-fields expressed as a percentage of the total population (X-fields and Y-fields). This relationship is shown separately, as indicated, for nondeprived eye receptive fields of MD cats, for deprived eye receptive fields of MD cats, for receptive fields of BD cats, and for receptive fields of normal cats. Data for normal cats were taken from Fig. 4*B* of the previous paper (13). The numbers next to each point indicate the total number of fields from which the percentage of Y-fields was calculated for that point. To minimize random sampling errors in these percentages (13), each point includes units pooled from at least four different penetrations using four different electrodes. The one exception to this is that the five units representing nondeprived eye fields of MD cats in the monocular segment were all encountered along a single electrode penetration.

Figure 2 summarizes the data of Fig. 1 above and of Fig. 4*B* of the preceding paper (13), and shows for normal, MD, and BD cats, the change in the relative frequency of Y-cells with receptive-field eccentricity. For MD cats the relative frequency of Y-cells among nondeprived eye fields follows closely the normal relationship with respect to eccentricity. However, considering the whole of the binocular segment of the visual field, the percentage of Y-fields among deprived eye fields is much less (9%, 6/70) than either the percentage among the nondeprived eye fields (63%, 41/64) or the percentage in normal animals (55%, 156/284). Both of these differences are statistically significant ($P < 0.001$ on $\chi^2$ tests). By contrast, considering the monocular segment of the visual field, the percentage of Y-fields among deprived eye fields did not differ significantly either from the percentage

TABLE 1. *Center sizes for X- and Y-fields of LGNd neurons in deprived cats*

| | X-Fields | | | | Y-Fields | | | |
|---|---|---|---|---|---|---|---|---|
| | No. of units | Range | Mean | SD | No. of units | Range | Mean | SD |
| Normal cats | 113 | 0.2–1.5 | 0.8 | 0.3 | 127 | 0.4–3.2 | 1.4 | 0.6 |
| MD cats: nondeprived eye | 19 | 0.3–1.4 | 0.8 | 0.3 | 31 | 0.6–2.6 | 1.3 | 0.5 |
| MD cats: deprived eye, monocular segment | 2 | 0.6–0.9 | 0.7 | 0.2 | 10 | 0.8–2.1 | 1.4 | 0.4 |
| MD cats: deprived eye, binocular segment | 39 | 0.4–1.6 | 0.9 | 0.4 | 4 | 0.7–1.5 | 1.2 | 0.4 |
| BD cats | 68 | 0.3–2.3 | 0.9 | 0.4 | 22 | 0.8–3.2 | 1.8 | 0.7 |

Values are in degrees. Data for deprived cats compared to data for normal cats taken from the previous paper (13). The number of fields does not include all the fields in Fig. 1 because, as in the previous paper (13), not all fields were sufficiently accurately measured before the unit was lost.

among nondeprived eye fields or from the percentage found in normal cats. The dashed line in Fig. 1*B* joins the receptive fields of neurons studied along a single electrode penetration in the sequence in which they were found. This sequence illustrates that the difference in relative Y-field frequency between the binocular and monocular segments could be seen in single electrode tracks (see also Fig. 3*A* and *B*). Thus, the loss of Y-fields following monocular deprivation was apparent only in the binocular segment of the visual field.

*Relative frequency of X- and Y-fields in BD cats*

A pattern of Y-field loss different from that found in MD cats emerged from the 102 units studied in BD cats. Figure 1*C* shows the positions in the visual field and X–Y classification of the receptive fields of these units, and Fig. 2 summarizes the relative frequency of Y-fields as a function of eccentricity. Considering first the receptive fields located in the binocular segment of the visual field, 29% (20/70) of the fields of BD cats were Y-fields, which is greater than the percentage among deprived eye fields of MD cats and less than the corresponding percentage both for the nondeprived eye fields of MD cats and for normal cats ($P < 0.001$ on a $\chi^2$ test for all three comparisons). On the other hand, the percentage of Y-fields found in the monocular segment of the visual field of BD cats, 28% (9/32) is not different from the percentage in its binocular segment, but is significantly less than the corresponding percentage in normal and MD cats ($P < 0.001$ on $\chi^2$ tests for both comparisons). The sequence of receptive fields for a single electrode penetration is illustrated in Fig. 1*C* (as in Fig. 1*B*) and shows that the paucity of Y-fields in both segments of the visual field could be seen in single electrode tracks. The effect of visual deprivation appears to be less severe in BD cats than in the binocular segment of the LGNd in MD cats, but to extend throughout the LGNd.

*Track reconstructions*

From the preceding, it is evident that in the LGNd of an MD cat an electrode can travel from a region with an abnormally low Y-cell population to a region with a normal Y-cell population in one of two ways: *1*) it can pass from a deprived to a nondeprived lamina within the binocular segment of the nucleus; or *2*) it can pass from a deprived lamina in the binocular segment to the unlaminated, monocular segment. Figure 3 shows a diagrammatic representation of the units in MD cats found along every electrode penetration which passed between two such regions of the LGNd and in the course of which at least two units were studied in each region. This figure emphasizes two points. First, all deprived laminae in the binocular segment of the LGNd, whether ipsilateral or contralateral to the deprived eye, showed the characteristic loss of Y-cells. This point was also substantiated for the BD cats. Second, the paucity of Y-cells in deprived laminae was apparent within single elec-

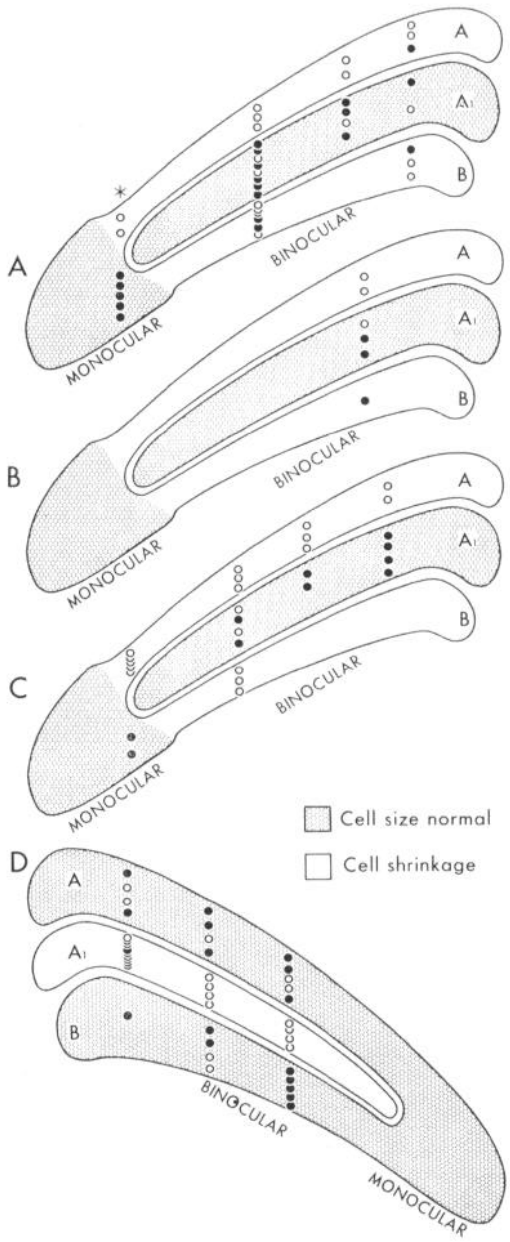


FIG. 3. Diagrammatic representation of every electrode penetration in MD cats which sampled at least two neurons each from regions of the LGNd with and without Y-cell loss (see text). X- and Y-cells encountered along these penetrations are represented as open and filled circles, respectively. The shaded portions of each LGNd are those showing no cell shrinkage as previously determined histologically (11). Each lamina A, $A_1$, and B is labeled; and the binocular and monocular segments are indicated. The sequence of neurons is correct as indicated, and they have been placed in the figure as follows. The placement in laminae of the binocular segment was determined both by stereotaxic readings of the electrode position and by determination of the eye whose visual stimulation activated the neuron. That is, all units placed in laminae A and B were driven by the contralateral eye, and those in lamina $A_1$ by the ipsilateral eye (1, 8, 10, 12, 18, 22). This lamination pattern has been previously reported for MD and BD cats (24). Placement of neurons in the monocular segment was derived from the stereotaxic position of the electrode track and from their receptive-field locations (see Fig. 1). One electrode track which traversed the monocular segment was reconstructed histologically and is marked by an asterisk in *A*. *A, B, C:* penetrations made contralateral to the deprived eye in three MD cats. The sequence of fields from units in the track marked with an asterisk in *A* is shown in Fig. 1*B*. *D:* penetrations made ipsilateral to the deprived eye in the fourth MD cat.

trode penetrations. The diagrammatic coronal sections used in Fig. 3 have shaded and unshaded portions which represent (following Guillery and Stelzner (11)) the regions of the monocularly deprived LGNd in which cell size and packing density are normal (shaded) and the regions in which there is cell shrinkage and an increase in cell density (unshaded). Histological sections of the LGNd made in this study confirm these observations. In the LGNd contralateral to the deprived eye of MD cats a discontinuity was particularly apparent in cell size between the monocular segment of the nucleus, where the cells appeared normal in size, and layer A of the binocular segment, in which the cells appeared shrunken and large cells, in particular, appeared to be lacking. No comparable discontinuity was apparent in the LGNd of a BD cat, an observation in agreement with previous cell body counts made by R. W. Guillery (personal communication).

### *Properties of LGNd cells driven by visually deprived eyes*

The responses to visual stimuli of LGNd cells driven by the deprived eye were essentially normal, in agreement with earlier reports (24, 25). Film records show no differences, for example, between the responses of any cell in this study and those of normal cells from the previous study (13) to the visual stimuli (stationary spots stimuli, hand-moved spots, and gratings) used to distinguish X- and Y-fields. However, these responses were not

quantified in either normal or visually deprived animals, and quantitative differences may exist. Recently, for example, Eysel et al. (4) presented evidence for a lowering in the surround inhibition of deprived eye fields in the LGNd of an MD cat. Those properties of deprived eye cells which were quantified, i.e., receptive-field center size and latency to electrical stimuli, also proved essentially normal, although minor statistical differences may exist. Table 1 shows that, for MD and BD cats, the mean center size of Y-cells was larger than that of X-cells as in the normal, and except for one X-cell each from an MD and a BD cat, the range of center sizes for deprived cats fell within the range of center sizes for normal cats.

Figure 4*A–D* shows that among all groups of units isolated in this study, Y-cells respond to OX stimulation at shorter latencies than X-cells, with little overlap, as in the normal (13). Figure 5*A–C* shows the correlation between OX and VC latencies in samples of LGNd units obtained from nondeprived and deprived laminae in MD cats and from BD cats. In each sample there is a statistically significant correlation between OX and VC latencies as in the normal cat (13). In all three samples (as in the normal) fast-conducting retinal afferents drive LGNd Y-cells which, in turn, have fast axons projecting to the visual cortex. Slow retinal afferents drive X-cells, which have slow axons to the visual cortex. The orthogonal regression line is drawn for each sample. The slopes and intercepts of these lines are close to the slope and intercept in the normal cat (13), although there appears to be more scatter in the OX/VC latency relationships for deprived eyes in MD and BD cats than for either the nondeprived eye of MD cats or normal cats (see also Fig. 3*C* of the previous paper (13)). Moreover, the OX and VC latencies in Figs. 4 and 5 were all within the range in normal cats (13) with the exception only of the unit in Fig. 5*C* with a VC latency of 3.1 msec.

In the preceding paper (13) two LGNd neurons were described which discharged at two latencies to OX stimulation, which suggested that both fast and slow retinal afferents converge to drive a single LGNd neuron (see also Cleland et al., ref 2). Five such units were seen in this study, one driven by the deprived eye in an MD cat, the other four in BD cats. For the first spikes the OX latencies ranged from 1.2–1.3 msec, which is in the latency range expected for Y-cells, and for the second spikes from 1.6–2.3 msec, which is in the latency range expected for X-cells. On the basis of receptive-field properties, two were classified as X-cells and three as Y-cells.

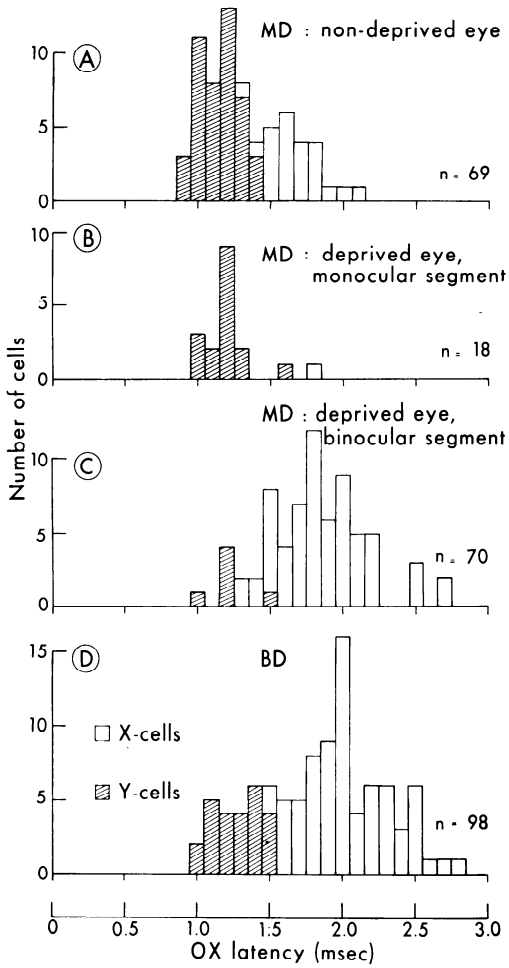


FIG. 4. Frequency histograms showing the distribution of response latencies to optic chiasm (OX) stimulation among single LGNd units. Numbers of X-cells are indicated by open bars; Y-cells by shaded bars. Not shown are latencies for five units which appeared to have two separate OX latencies (see text). *A:* neurons driven by the nondeprived eye in MD cats. *B:* neurons from the monocular segment driven by the deprived eye in MD cats. *C:* neurons from the binocular segment driven by the deprived eye in MD cats. *D:* neurons in BD cats.

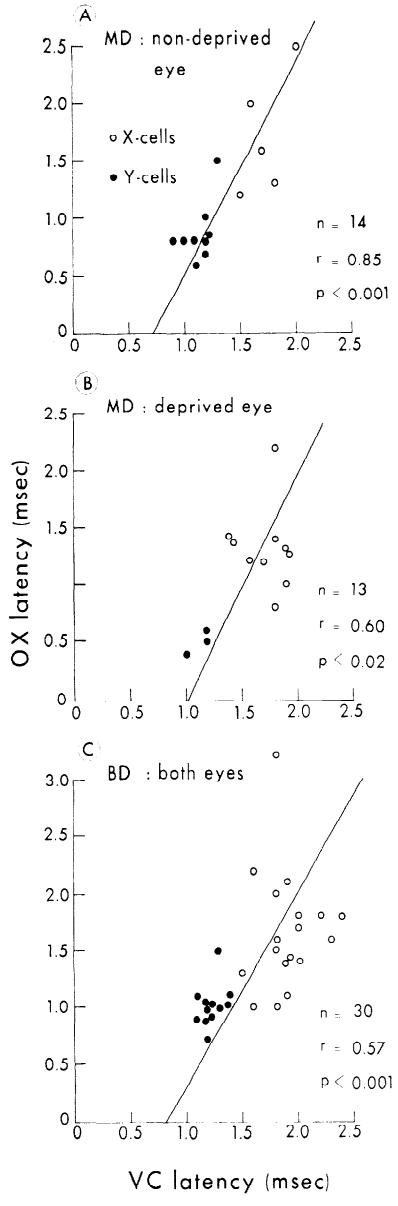


FIG. 5. Correlations between OX and VC latencies of LGNd neurons in MD and BD cats. X-cells are represented by open circles and Y-cells by filled circles. The orthogonal regression line, the correlation coefficient ($r$), and the confidence limit of the correlation ($P$) are indicated in each case. *A:* neurons driven by the nondeprived eye in MD cats. *B:* neurons driven by the deprived eye in MD cats. *C:* neurons in BD cats.

That is, in each case one input seemed to be functionally dominant. These cells are excluded from the latency data in Figs. 4 and 5 but have not been excluded from the other illustrations.

## DISCUSSION

The results of this study confirm previous reports that the functional properties of individual LGNd cells in visually deprived cats are essentially normal (24, 25). Nevertheless, the principal finding of this study, that visual deprivation results in a selective loss of Y-cells from the LGNd, indicates that the LGNd as a whole is markedly abnormal in function and thus runs counter to a previous suggestion (25, 26). It also seems to require reconsideration of the suggestion that visual deprivation principally disturbs synapses within the visual cortex (17, 26, 27). Before pursuing this point it seems important to consider the following three questions:

### *How do present data relate to histological data?*

There is a good correlation in both MD and BD cats between the areas of the LGNd from which Y-cells are lost and the areas in which histological changes are apparent. Guillery and Stelzner (11), for example, report that there is significant cell shrinkage in the deprived laminae of the binocular segment of the LGNd, but no significant shrinkage in the deprived monocular segment. In BD cats there is no comparable difference between monocular and binocular segments of the LGNd. However, Wiesel and Hubel (27), K. L. Chow and D. Stewart (personal communication), and R. W. Guillery (personal communication) all report evidence of cell shrinkage in the LGNd. Wiesel and Hubel reported the shrinkage to be as severe as in the binocular segment of the LGNd in MD cats. Chow and Stewart and Guillery consider the shrinkage to be less severe than in MD cats. Guillery (personal communication) notes that shrinkage is apparent in both binocular and monocular segments of the LGNd.

Clearly these results provide some morphological basis for the present physiological findings. It was suggested in the previous paper that Y-cells may be large cells. It is consistent with this idea and with the loss of Y-cells noted in this study to suggest that the decrease in mean size

of all cells noted by the above workers may be largely a result of the selective shrinkage of large cells.

*Could functioning Y-cells be present but be so reduced in size that they are missed by present electrodes?*

While it seems unlikely that such a sharp reduction in cell size could occur without a change in functional properties, there is no direct evidence against this possibility. If this is true, however, the Y-cells must be considerably smaller than normal Y-cells (smaller, too, than X-cells), and this would itself constitute a selective effect of deprivation on Y-cells. Of course, the possibility exists in any single-unit study with microelectrodes that the activity of a class of cells is missed. The following discussion assumes that the microelectrodes provided an accurate sample of cell types in the LGNd and that the number of functional Y-cells is reduced by deprivation.

*What is fate of lost Y-cells?*

It seems unlikely that their cell bodies disappear from the LGNd since counts made by Guillery (personal communication) indicate no cell loss from LGNds of MD cats. Two more likely possibilities are: *a*) the cells could be present physically but be nonfunctional; and/or *b*) the cells which would normally develop as Y-cells accept X-afferents and develop as X-cells.

*Possible mechanisms for effect of deprivation on LGNd*

Three possible mechanisms can be suggested to explain the effects of deprivation on the LGNd, but none alone seems adequate to explain all of the present results in MD and BD cats.

*1*) Deprivation might exert its primary effect on the retina, causing a functional loss of Y-cells there. The removal of their drive could either silence their intended target cells in the LGNd or it could cause them to develop as X-cells. In either case these LGNd cells could shrink or grow less. This suggestion, however, fails to explain either the persistence of Y-cells in the monocular segment of MD cats or the different pattern of cell loss in MD and BD cats.

*2*) The primary effect of deprivation might be to disrupt the synapses of Y-afferents in the LGNd. This possibility could have the same consequences as the first suggestion, and also would suffer from the same inadequacies.

*3*) The effects of deprivation might have their primary locus in the cortex, changing the LGNd secondarily. Two distinct mechanisms are included in this possibility. *a*) The first is an extension of Wiesel and Hubel's (26, 27) concept of "binocular competition" among geniculostriate synapses. That is, during development LGNd neurons compete for synapses on binocularly activated cortical cells. In an MD cat, LGNd neurons driven by the deprived eye are in some sense handicapped in this competition, and their terminals fail to form effective synapses. Their cells of origin undergo a subsequent anatomical atrophy or failure to develop. If this process were limited to Y-cells and include functional as well as anatomical defects, many of the results in MD cats can be explained: thus a small number of Y-cells driven by the deprived eye in the binocular segment and all Y-cells in the monocular segment of the LGNd survive because they have uncontested monocular control of their target cortical cells (16, 26, 27). However, this mechanism fails to explain the loss of Y-cells from the monocular segment of the LGNd in BD cats. *b*) The second mechanism involving a primary effect of deprivation on the cortex would require that the functional development of Y-cells depends on a normal corticofugal input to the LGNd (9, 14) which is disturbed by early visual deprivation. There is little evidence, however, for or against this suggestion, and it, too, fails to account easily for the differences between MD and BD cats.

To summarize, further work seems necessary to determine the nature and location of the primary effect or effects of deprivation on the developing visual system. A principal difficulty with any scheme for these effects is to explain how one effect (the loss of Y-cells) is less severe for BD cats than MD cats in the binocular

segment of the LGNd, but more severe for BD cats than MD cats in the monocular segment. An analogous qualitative difference in the development of interocular alignment has been noted between BD and MD cats (23), and this difference was also seen in the present study. It was then suggested (23) that development of interocular alignment is controlled by different mechanisms in BD and MD cats. The results of the present experiment likewise suggest that different mechanisms control development of the geniculostriate pathway in BD and MD cats.

## SUMMARY

*1.* Electrophysiological techniques were used to study responses of 260 single units in the dorsal lateral geniculate nucleus (LGNd) of seven adult cats visually deprived by eyelid suture from their 8th postnatal day. Three of the cats were binocularly deprived (BD), and four were monocularly deprived (MD). The latency of each cell's orthodromic response to electrical stimulation of the optic chiasm was measured; and the antidromic response latency to electrical stimulation of the visual cortex was also measured in 57 of these cells. The cells' responses to visual stimuli, including flashing spots of light, hand-held black or white targets, and square-wave black-and-white gratings, were also studied.

*2.* As in the normal cat, these LGNd neurons could be classified by their responses to the visual stimuli as X-cells or Y-cells. Moreover, the properties of all but two of the LGNd cells in this study were within the range of properties previously established for normal cats.

*3.* However, there was a marked reduction in the proportion of Y-cells found in the LGNd of visually deprived cats. In MD cats there was a severe loss of Y-cells from laminae in the binocular segment receiving direct retinal afferents from the deprived eye. Y-cell loss in MD cats was apparent neither in laminae receiving direct retinal afferents from the nondeprived eye nor in the unlaminated, monocular segment of the LGNd contralateral to the deprived eye. In BD cats, a less severe loss of Y-cells was apparent throughout the LGNd.

*4.* Three implications of these data are discussed: *a*) The location and severity of Y-cell loss from the LGNd correlates with the location and severity of cell shrinkage in the LGNd, described previously. *b*) This evidence of functional change in the LGNd following visual deprivation suggests that the primary effect of deprivation could be exerted peripheral to the cortex, although it is still possible that changes in the LGNd are secondary to primary changes in the cortex. *c*) An extension of an earlier concept of binocular competition during development is consistent with the pattern of Y-cell loss found in the LGNd of MD cats, but fails to explain the pattern found in the LGNd of BD cats.

*5.* These results suggest that different mechanisms guide visual development in MD and BD cats.

## ACKNOWLEDGMENTS

We thank Mrs. Joyce Campion and Miss Sandra Watson for their technical assistance and Mrs. Lyn Speight for her secretarial help. Professor P. O. Bishop gave valuable advice in the preparation of the manuscript.

S. M. Sherman was supported by National Institutes of Health Grant 1 FO2 NS4 3310.

K.-P. Hoffmann was partially supported by a travel grant from the Deutsche Forschungsgemeinschaft.

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