Development of the Lateral Geniculate Nucleus in Cats Raised with Monocular Eyelid Suture

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Since the pioneering work of Wiesel and Hubel (1963a,b; 1965), neurobiologists have appreciated the kitten's central visual pathways as an elegant model system for studies of the role of the postnatal environment in neural development. A particularly useful approach has been a comparison of the geniculocortical pathways in normally reared cats with those in cats raised with monocular eyelid closure. This paper concentrates on the developmental abnormalities seen in the lateral geniculate nucleus of such monocularly deprived cats. Although most studies of visually deprived cats have focused upon striate cortex, we have emphasized the lateral geniculate nucleus, because an understanding of cortical abnormalities requires a fairly complete description of the status of its geniculate inputs.

A simple version of the cat's retino-geniculo-cortical pathways is shown in Fig. 1. The dorsal two geniculate laminae, A and A1, provide a reasonably matched representation of each eye, and nearly all of our data are derived from this laminar pair. The ventral C complex includes laminae C, C1, and C2 (Guillery, 1970), and virtually nothing is known regarding the postnatal development of these laminae. Mainly for these reasons, this paper is further limited to a consideration of deprivation effects in laminae A and A1. However, before these are considered, it is useful to divide the lateral geniculate nucleus further into binocular and monocular segments and X- and Y-cells.

Binocular and Monocular Segments

Definition Each geniculate neuron has a small receptive field limited in visual space, and neighboring neurons tend to map neighboring spatial coordinates. As a consequence, an orderly, fairly precise point-to-point map of visual space

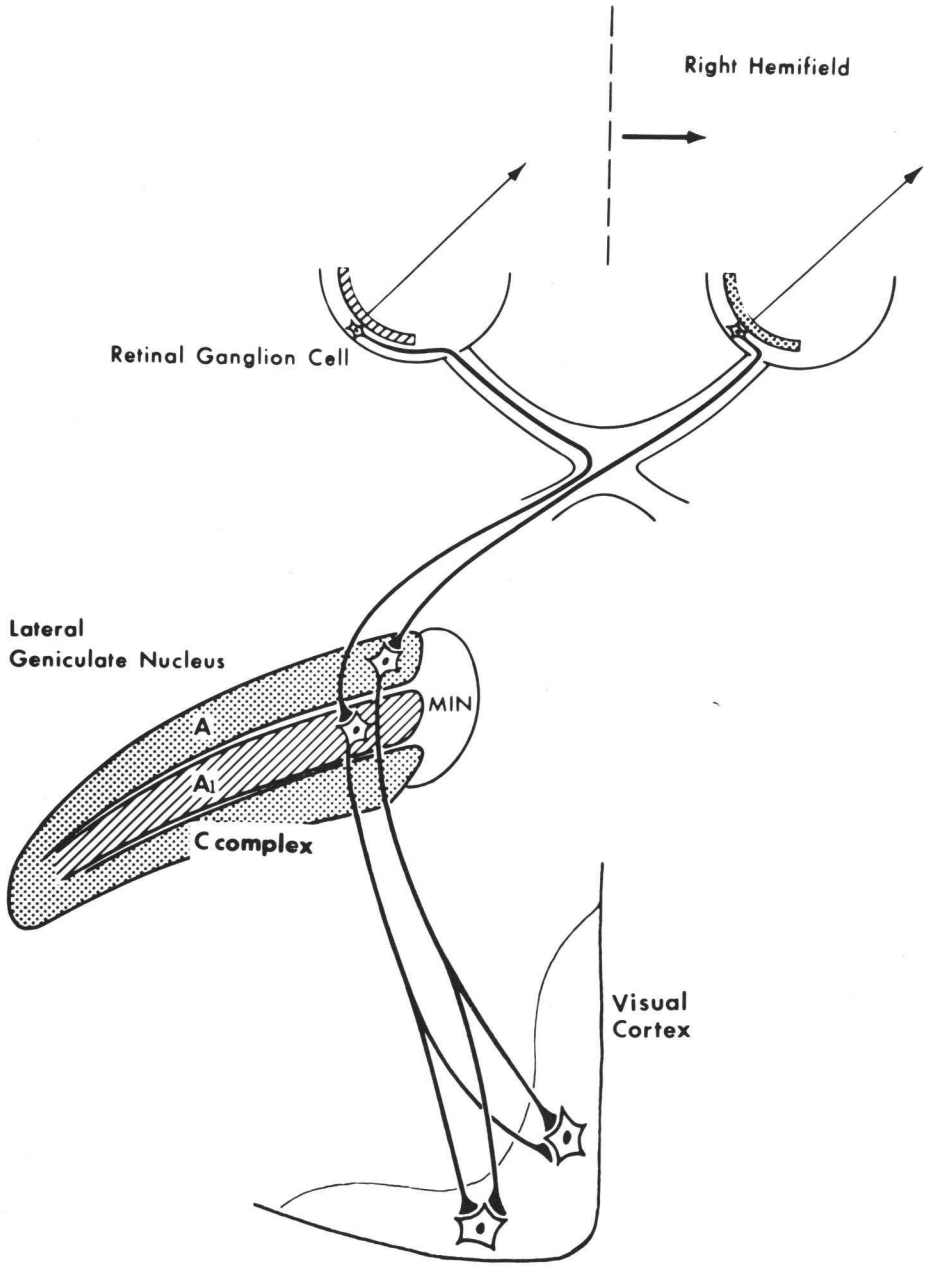

FIGURE 1 Retino-geniculo-cortical pathways in the cat (see text for details). The lateral geniculate nucleus is diagrammed in a coronal plane. The C complex refers to ventral laminae (C, C1, and C2), which receive input from retina and project to cortex, although these pathways are not drawn. Likewise, the medial interlaminar nucleus (MIN) contains cells which receive retinal input and project to cortex.

exists in the lateral geniculate nucleus (Sanderson, 1971): lateral (or medial) displacements in the nucleus map more peripheral (or central) visual space in the contralateral hemifield, and the medial edge of the nucleus represents the vertical meridian of visual field; rostral (or caudal) displacements map more inferior (or superior) visual space. The maps in laminae A and A1 are in register such that lines perpendicular to the laminae represent the same general area of visual space. As a result of this mapping, the binocular segment is that part of the nucleus which maps the central visual field seen by both eyes (roughly 45° to either side of the vertical meridian; Sherman, 1973). This includes all of lamina A1 and the corresponding portion (i.e., the medial three-fourths) of lamina A. The monocular segment is that part which maps the extreme peripheral crescent of visual field which can be viewed only by one eye (roughly 45-90° ipsilateral to that eye; Sherman, 1973). This is represented in the lateral one-fourth of lamina A which extends beyond lamina A1.

Binocular competition vs. deprivation per se Guillery and Stelzner (1970) first made use of this division into binocular and monocular segments in their histological studies of monocularly deprived cats. They confirmed and extended an earlier observation of Wiesel and Hubel (1963a). That is, Guillery and Stelzner (1970) reported that although cells in deprived laminae (i.e., those receiving direct retinal afferents from the sutured eye) were abnormally small, this effect was limited to the binocular segment of the nucleus. The deprived monocular segment of lamina A had cells of normal size which were indistinguishable from those in the nondeprived monocular segment on the other side (however, see Hickey, Spear, and Kratz, 1977).

The significance of this differential effect of monocular suture on the binocular and monocular segments of the nucleus is outlined in Fig. 2. The concept represented is that at least two different mechanisms can operate to produce the deprivation effects. We refer to one as "binocular competition" and the other as "deprivation *per se*," and they are described more fully below (see also, Sherman, Guillery, Kaas, and Sanderson, 1974).

The idea that a competitive mechanism is involved originated with Wiesel and Hubel (1965) and was elaborated by Guillery and coworkers (Guillery and Stelzner, 1970; Guillery, 1972; Sherman, Hoffmann, and Stone, 1972; Sherman, 1973; Sherman et al., 1974; Sherman, Wilson, and Guillery, 1975; Wilson and Sherman, 1977). Wiesel and Hubel (1965) suggested that during early postnatal development, pathways from each eye compete with one another for dominance of central connections. The actual site of this competition remains unknown, and for illustration purposes only, Fig. 2 is drawn as if the competition occurs between sets of geniculocortical synapses related to each eye. During development, these synapses proliferate in strength, number, or both, and they compete for total control of the cortical cell. If the visual environment is normal (Fig. 2, left), neither set of synapses related to one or the other eye has a competitive advantage conferred upon it, a balance is struck, and normal, binocular cortical neurons emerge (Hubel and Wiesel, 1962). However, if one

eye is sutured (i.e., the right eye in Fig. 2 (right), so that lamina A in the drawing is deprived), an advantage is somehow conferred upon the development of nondeprived geniculocortical connections. The advantage may be related to higher peak firing rates, more synchronous firing, etc., but in fact, we have no evidence as yet to suggest why nondeprived cells should be given an advantage. In any case, because of this advantage during competitive development, the nondeprived eye gains essentially total control over the cortical neurons, as is the case in monocularly deprived cats (Wiesel and Hubel, 1963b, 1965; Wilson and Sherman, 1977). Notice, however, that cells in the deprived monocular segment, by definition, cannot suffer the deleterious consequences of developing at a competitive disadvantage. Although they are deprived just as much as their counterparts in the binocular segment, they can form many stable

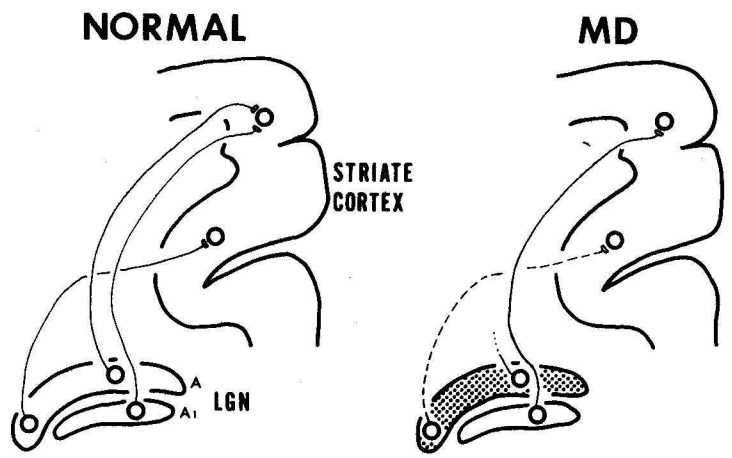

FIGURE 2 Diagram to illustrate developmental mechanisms of binocular competition and deprivation *per se* in monocularly deprived cats. The deprived example (MD) is shown as if the right eye and thus, lamina A, were deprived. It is suggested that during postnatal development, competition occurs among geniculo-cortical synapses for control of cortical neurons. During normal rearing (left), no competitive advantage is present, a balance is struck, and binocular cortical cells emerge. During monocular deprivation (right), the deprived cells are somehow placed at a competitive disadvantage such that the nondeprived eye develops nearly complete dominance over cortical cells. The deprived cells in the monocular segment of lamina A cannot by definition be placed at a competitive disadvantage, so they develop and/or maintain at least some cortical connections. To the extent that the deprived monocular segment cells are completely normal, whereas those in the binocular segment are not, a developmental mechanism of binocular competition is indicated. To the extent that equal deficits are seen throughout deprived lamina A, a noncompetitive developmental mechanism of deprivation *per se* is suggested. A combination of these two developmental processes is also possible.

geniculocortical connections simply because they are not fighting with a superior foe for these synaptic sites. Therefore, to the extent that the deprived monocular segment develops much more normally than does the deprived binocular segment, support for a mechanism of binocular competition is indicated. This is one explanation for the histological observations of Guillery and Stelzner (1970).

The concept of the noncompetitive mechanism of deprivation *per se* is much simpler. By this mechanism, the development of central pathways is determined solely by the quality of the visual environment experienced by each eye; and interocular interactions, such as binocular competition, play no role. As applied to Fig. 2, this mechanism would require that deprived cells develop equal abnormalities in the binocular and monocular segments.

From the consideration above, it should be clear that an important comparison to be made in these studies of monocularly deprived cats is between the deprived binocular and monocular segments. When deprivation-induced deficits are apparent, three possible conclusions can be drawn from such comparisons: (1) if the deprived monocular segment develops completely normally while the binocular segment does not, a mechanism of binocular competition alone can parsimoniously account for the results; (2) if the deprived monocular and binocular segments develop equal deficits, competitive mechanisms are not indicated, and deprivation *per se* can account for the results; and (3) if deficits are seen both in monocular and binocular segments, but the monocular segment deficits are less severe, a combination of binocular competition and deprivation *per se* is indicated. Two other points can be made. First, if only the binocular segment is studied, one cannot easily distinguish between effects due to binocular competition and those due to deprivation *per se* (cf. Sherman et al., 1974). Second, the deprived monocular segment may be the only place where deprivation *per se* without competition influences can be studied.

X- and Y-cells

The other important division of the cat's retino-geniculo-cortical pathways stems from the classical optic tract study of Enroth-Cugell and Robson (1966). They defined two distinct populations of retinal ganglion cells as X (linear spatial summation) and Y (nonlinear spatial summation).* Since then, numerous laboratories have concentrated on this distinction, and the scope of this literature is much too broad to cover in the present paper (for reviews, see Rowe and Stone, 1977; Rodieck, 1979). X- and Y-cells have been described in the

*Recently, a third cell type (W) has been described among retinal ganglion cells. Some of these cells project through geniculate neurons in the C complex to cortex (Wilson and Stone, 1975; Wilson, Rowe, and Stone, 1976). W-cells differ in many ways from X- and Y-cells, but relatively little is known of them in normal cats and virtually nothing is known of their properties following early visual deprivation. For this reason, plus the fact that nearly all of our analysis has been limited to laminae A and A1, which lies outside the W-cell pathway, W-cells are not considered further in this paper.

lateral geniculate nucleus with nearly identical properties to their retinal counterparts (Cleland, Dubin, and Levick, 1971; Hoffmann, Stone, and Sherman, 1972; Shapley and Hochstein, 1975). We now know that X- and Y-cells differ among many electrophysiological characteristics. Compared to X-cells, Y-cells possess: faster conducting axons, less linear spatial summation in the receptive field, larger fields, more phasic responses to standing contrasts, slightly better sensitivity to temporal changes, slightly poorer sensitivity to high spatial frequencies, and much greater sensitivity to low spatial frequencies (Cleland et al., 1971; Hoffmann et al., 1972; Shapley and Hochstein, 1975; Hochstein and Shapley, 1976a,b; Lehmkuhle, Kratz, Mangel, and Sherman, 1979a).

Although it seems clear that X- and Y-cells represent two parallel, fairly independent pathways from retina to cortex (Cleland et al., 1971; Hoffmann et al., 1972), the significance of X- and Y-cells for cortical processing remains unclear and somewhat controversial. In a strong departure from the Hubel and Wiesel (1962) "serial processing" hypothesis (Fig. 3A), whereby a single chain of cells from geniculate to cortical simple cell to cortical complex cell, etc., processed visual information, Stone and coworkers (Hoffmann and Stone, 1971; Stone and Dreher, 1973) suggested a hypothesis of "parallel processing" (Fig. 3B), whereby two independent cell chains—X-cells through cortical simple cells and Y-cells through cortical complex cells—processed different aspects of the visual scene in parallel.

Whatever the functional significance of the division of retinal and geniculate neurons into X- and Y-cells (see also below), it seemed reasonable to investigate the possibility that these two systems were differentially affected by deprivation in an analogous fashion to the differences seen between binocular and monocular segments. The importance of this possibility was underscored recently by the observations of Daniels, Pettigrew, and Norman (1978) who concluded that kitten geniculate X-cells normally attain maturity earlier than do Y-cells, and that Y-cells are thus more susceptible to environmental deficiencies during the "critical period" (Hubel and Wiesel, 1970) of early postnatal development.

Effects of Monocular Deprivation upon Y-cells

Sherman et al. (1972) reported that genicular Y-cells seemed much more affected by early lid suture than did X-cells. Deprived X-cells generally seemed normal both in numbers and response properties, although a subtle abnormality is described in the next section. Figure 4 represents a redrawing of Fig. 2 from Sherman et al. (1972) with added data points and limited to data from laminae A and A1. This shows that, with our recording techniques in monocularly deprived cats, few normal Y-cells were encountered throughout the binocular segment, whereas normal numbers were seen in the monocular segment. Furthermore, the receptive field properties of the encountered deprived Y-cells (mostly in the monocular segment) were completely normal (Sherman et al., 1972; Lehmkuhle et al., 1979b). These properties included response rate, field

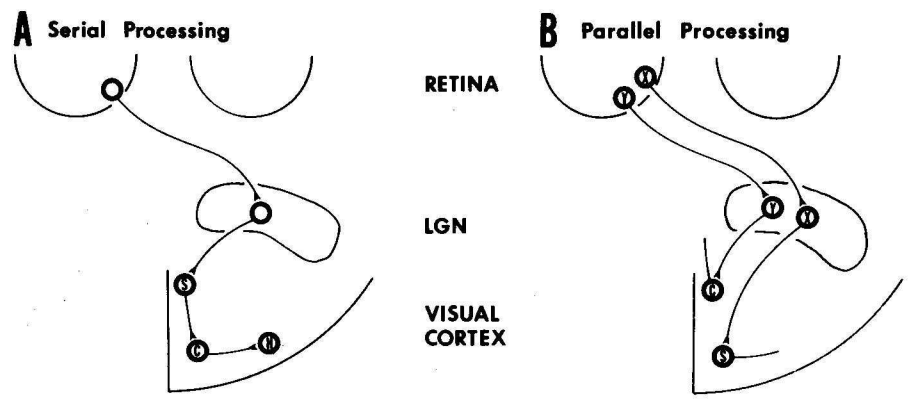

FIGURE 3 Hypotheses of serial and parallel processing. A: Wiring diagram for serial processing hypothesis of Hubel and Wiesel (1962). They suggested a single hierarchy of neurons from retina through cortex for visual processing. In this scheme, a fairly homogeneous population of geniculate cells feeds onto the first-order, or simple (S) cortical cell, then to the complex (C) and hypercomplex (H) cells. Note that this hypothesis was proposed before knowledge of X- and Y-cells. B: Wiring diagram for parallel processing scheme suggested by Stone and coworkers (Hoffmann and Stone, 1971; Stone, 1972; Stone and Dreher, 1973) and incorporating the concept of X- and Y-cells. At least two fairly independent pathways from retina through cortex which process different aspects of the visual scene in parallel are suggested. Retinal X-cells project to geniculate X-cells which project to cortical simple (S) cells. Retinal Y-cells project to geniculate Y-cells which project to cortical complex (C) cells. These diagrams represent simplified versions of hypotheses and should not be treated literally.

size, temporal and spatial contrast sensitivity (see also below), and area response functions. This pattern of few normal Y-cells in the deprived binocular segment and many perfectly normal Y-cells in the deprived monocular segment corresponds closely to the histological observations of Guillery and Stelzner (1970) and suggests a mechanism of binocular competition.

The interpretation of these results is not straightforward since uncontrolled electrode sampling biases are possible. Anatomical correlates, described below, help somewhat in our understanding of these results. One suggested by Eysel, Grüsser, and Hoffmann (1978) is that these results merely reflect a changed electrode sampling artifact caused by relatively selective shrinkage of Y-cells (see also, LeVay and Ferster, 1977; Garey and Blakemore, 1977). Even if this is the sole explanation, it supports the general notion that geniculate Y-cells are more affected by early lid suture than are the X-cells. Furthermore, whatever the reason for our failure to record deprived Y-cells, soma size alone cannot be the general explanation. This point is made most clearly in studies of cats reared in total darkness (Kratz, Sherman, and Kalil, 1979). In these animals, we found very few geniculate Y-cells (Fig. 4), yet the soma size distribution among laminae A and A1 neurons was completely normal in the same cats from which Y-cells went unrecorded. These cells clearly did not show the lack

of growth seen in monocularly deprived cats.* This suggests that Y-cell "losses" need not be correlated with changed soma size. Indeed, we have recently obtained evidence that deprived laminae contain abnormal cells with poor or no visual responsiveness that might represent the "missing" Y-cells (Kratz, Webb, and Sherman, 1978b; unpublished observations; and see Norton, Casagrande, and Sherman, 1977 for similar observations in monocularly sutured tree shrews).

In any case, there have been two types of anatomical studies which correlate with, but cannot yet explain, the physiological absence of recordable Y-cells from deprived laminae, which was described above. First, Garey and Blakemore (1977) and Lin and Sherman (1978) tried to isolate geniculate Y-cells for anatomical study by capitalizing on the observation (Stone and Dreher, 1973) that geniculate X-cells project only to area 17, while the Y-cells project both to areas 17 and 18. Horseradish peroxidase was injected into area 18 of monocularly deprived cats to label only a Y-cell population, and it was found that, in deprived laminae, labeled cells were smaller (Garey and Blakemore, 1977), much rarer, and more poorly stained (Lin and Sherman, 1978) than they were in nondeprived laminae. Area 17 injections provided relatively little asymmetry in labeling of deprived and nondeprived laminae, presumably because of the many fairly normal X-cells labeled in deprived laminae.

Second, LeVay and Ferster (1977) suggested a histological marker to distinguish between X- and Y-cells in the cat's lateral geniculate nucleus. They found that some cells had a curious cytoplasmic structure—à "cytoplasmic laminar body" (CLB)—while others did not. Based upon several lines of converging but indirect evidence, they concluded that cells with CLBs were X-cells. Larger cells without CLBs would thus be Y-cells; and the few smaller cells without CLBs, interneurons. Furthermore, they correlated these cell types with Golgi studies and concluded that Y-cells were Guillery's (1966) class 1 (large soma, extensive, cruciate dendritic arbor with few appendages or spines), X-cells were class 2 (intermediate soma size, curved dendrites with grape-like structures appended at dendritic branch points), and interneurons were class 3 (small soma, fine tortuous dendritic arbor with numerous stalked appendages of variable morphology). Although Guillery (1966) reported that 40% of his sample was intermediate or nonclassifiable, LeVay and Ferster (1977) do not mention such cells, so it is not clear whether these cells contain CLBs. LeVay and Ferster (1977) then applied their CLB classification to one monocularly deprived cat and concluded that, compared to deprived X-cells, deprived Y-cells were both fewer in number and considerably more shrunken (see also, Kalil and Worden, 1978).

*This raises another perplexing question. That is, what environmental factors and/or mechanisms control cell size? On the one hand, monocular suture retards cell growth in deprived laminae (Wiesel and Hubel, 1963a; Guillery and Stelzner, 1970; Hickey et al., 1977), whereas binocular suture or total dark rearing has little or no effect on cell size (Guillery, 1973; Hickey et al., 1977; Kalil, 1978; Kratz et al., 1978). Perhaps competitive mechanisms control cell size, so that only during appropriately unbalanced environmental conditions between the eyes will significant abnormalities in geniculate cell size develop.

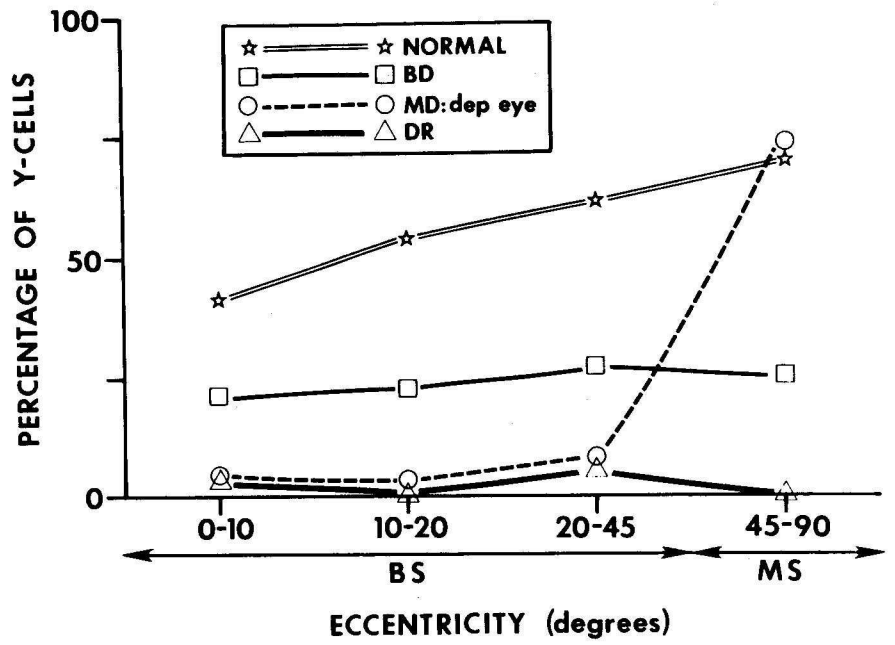

FIGURE 4 Percentage of Y-cells recorded in laminae A and A1 as a function of receptive field eccentricity from the area centralis. The few interneurons and unclassified relay cells (< 5%) are excluded, and the ordinate represents the percent fraction of Y-cells recorded among the total X- and Y-cell sample. The abscissa is broken into four eccentricity groups, and the binocular and monocular segments (BS and MS) are indicated. Each point on the graph for normal and monocularly deprived (MD) cats represents a sample of 62 to 212 cells. The data from nondeprived laminae of monocularly deprived cats (not shown) are indistinguishable from the normal data. Also shown are data from binocularly sutured (squares) and dark-reared (triangles) cats.

Such an anatomical means of identifying X- and Y-cells, based on a presumably nonselective histological method for locating CLBs, is of obvious potential importance for studying differential deprivation effects. For this reason, we have initiated a study to evaluate critically the LeVay and Ferster (1977) hypothesis by obtaining structure/function correlates at the single cell level for geniculate neurons in normal cats. Our method is to record intracellularly from these neurons with a fine micropipette filled with horseradish peroxidase, identify the cell as X or Y with conventional electrophysiological tests, and then iontophorese peroxidase into the cell for later morphological study. The filled cells present a Golgi-like appearance that allows ready identification into the classes described by Guillery (1966) and used by LeVay and Ferster (1977). To date, we have made such a correlation for ten Y-cells and eight X-cells (Friedlander, Lin, and Sherman, 1979). Of the Y-cells, six were class 1, two

were class 2, and two were intermediate or could not be classified. Of the X-cells, one was class 3, and the remaining seven were intermediate between classes 2 and 3, varying from nearly complete class 3 morphology to mostly class 2 structure. We thus tentatively conclude from a small sample that the LeVay and Ferster (1977) hypothesis requires some modification. Class 1 cells seem to be Y-cells, but cells with class 2 characteristics can also be Y-cells. X-cells seem to occupy the structural ground between classes 2 and 3. To the extent that class 2 cells seem to be Y-cells, and this was the only neuron class significantly affected by monocular eyelid suture (LeVay and Ferster, 1977), the anatomy again suggests that geniculate Y-cells are much more affected by early eyelid suture than are geniculate X-cells.

Effects of Monocular Deprivation upon Geniculate X-cells

Until recently, we were unable to detect any obvious effect of lid suture upon X-cell development. In deprived laminae, these cells were encountered in normal numbers and possessed fairly normal response properties (Sherman et al., 1972). Also, none of the anatomical studies suggested significant structural abnormalities for deprived X-cells (LeVay and Ferster, 1977; Garey and Blakemore, 1977; Lin and Sherman, 1978).

However, the recent literature suggested that more sensitive receptive field methods might uncover subtle deficits for deprived X-cells. For instance, Ikeda, Tremain, and Einon (1978) report that geniculate X-cells in cats raised with artificial esotropia have abnormally poor spatial acuity (defined as the highest spatial frequency to which the cell responds). Similarly, Maffei and Fiorentini (1976) and Hoffmann and Sireteneau (1977) reported poorer spatial acuity for deprived geniculate cells, but neither report distinguished between X- and Y-cells. We reinvestigated this question of geniculate X-cell normality in monocularly deprived cats by obtaining for these cells spatial and temporal contrast sensitivity functions to counterphased, sine-wave gratings (Lehmkuhle et al., 1978, 1979b). That is, we measured the grating contrast necessary to evoke a threshold neuronal response as spatial frequency (cycles/degree) and/or temporal frequency (cycles/sec counterphase rate) was varied. We found that, compared to nondeprived or normal geniculate X-cells, deprived X-cells had normal temporal sensitivity and normal sensitivity to lower spatial frequencies but were relatively insensitive to the higher spatial frequencies. Consequently, their spatial acuity was consistently reduced to the point that, on average, a normal X-cell could respond to a grating twice as fine as one that would excite a deprived X-cell (see Table 1). An important additional point also evident from Table 1 is the observation that deprived X-cells in the monocular segment were just as affected as were those in the binocular segment.

This last point is in stark contradistinction to the deprivation abnormalities described for Y-cells (compare Fig. 4 with Table 1) and suggests that different mechanisms are involved. Whereas some form of binocular competition effectively accounts completely for the Y-cell pattern of deficits, deprivation *per se* seems the simplest explanation for the X-cell pattern, since the abnormalities are equal for deprived binocular and monocular segments.

TABLE 1 Spatial resolution (highest spatial frequency sine-wave grating at 0.6 contrast and 2 cycles/sec counterphase rate to which the cell responds) for geniculate X-cells in deprived and nondeprived laminae A or A1 of monocularly deprived cats; data from Lehmkuhle et al. (1979b). These values (number of cells and mean ± standard error) are indicated for each of five eccentricity groups (receptive field eccentricity from the area centralis), including the monocular segment (> 45°), plus the total of all cells. No deprived X-cells were studied with a receptive field eccentricity between 20° and 45°. The reduction in resolution for each group is also shown and is fairly constant with eccentricity. This reduction is calculated as 100% [1 - (deprived resolution)/(nondeprived resolution)].

	0°-5°	5°-10°	10°-15°	15°-20°	> 45°	Total
Nondeprived						
N	32	29	8	9	11	89
Mean ± S.E.	2.8 ± 0.2	2.6 ± 0.2	2.5 ± 0.3	1.9 ± 0.2	1.2 ± 0.1	2.4 ± 0.2
Deprived						
N	5	17	9	7	17	55
Mean ± S.E.	1.5 ± 0.3	1.2 ± 0.1	1.1 ± 0.2	1.3 ± 0.2	0.6 ± 0.1	1.0 ± 0.2
Reduction	47%	54%	56%	33%	50%	58%

Further Evidence for Binocular Competition

The evidence presented above for binocular competition as a developmental mechanism is based upon differences between the reactions of the binocular and monocular segments to early monocular deprivation. The underlying assumption has been that these developmental differences are due to the binocular/monocular distinction between these segments. However, there are other differences that seem unrelated to this distinction. For instance, compared to centrally represented portions of the visual field (i.e., binocular segment), the peripherally represented portions (i.e., monocular segment) tend to have cells with less selective receptive field properties, and thus their development may be less sensitive to environmental irregularities. Also, differences in geniculocortical pathways between these areas have been suggested. Tusa, Rosenquist, and Palmer (1979) report that, whereas cortical area 17 includes a complete representation of the visual field, the area 18 map essentially covers only the binocular segment. Perhaps only the geniculocortical pathways to area 18, which involve Y-cells but not X-cells (Stone and Dreher, 1973), are affected by early lid suture, and this would not require a competitive mechanism.

Guillery (1972) designed an elegant experiment to demonstrate that the developmental differences between the binocular and monocular segments are due to the binocular/monocular distinction—and thus binocular competition—rather than other factors suggested above. He created a centrally located "critical segment" or "artificial monocular segment" by placing a neonatal retinal lesion centrally in the open eye at the time the other eye was sutured. Figure 5 summarizes the results obtained with this preparation which now includes two monocular segments for the deprived eye: the natural one related to extreme nasal retina and the artificial one related to central retina homonymous to the

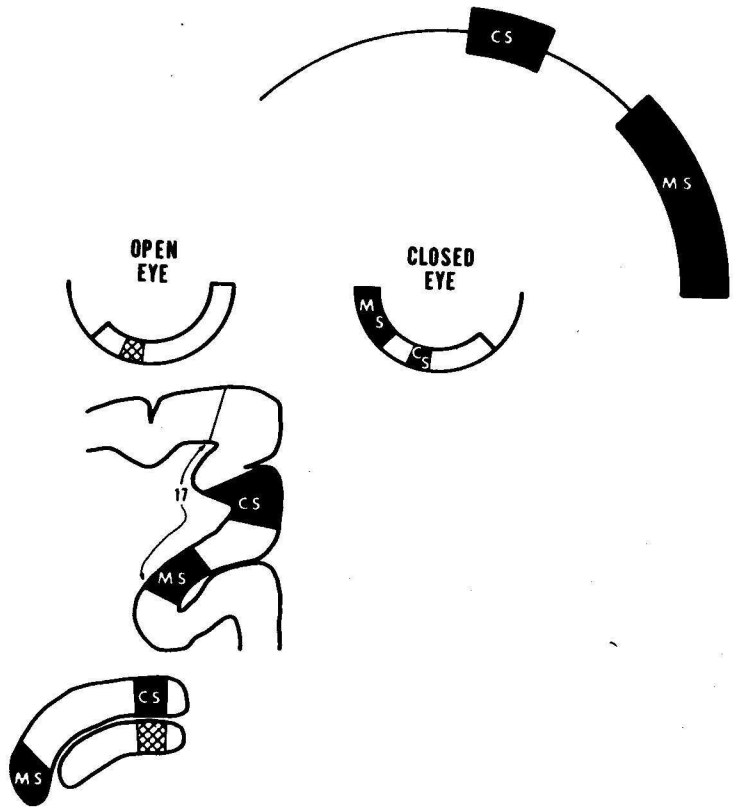

FIGURE 5 Summary of results from critical segment or artificial monocular segment preparation described by Guillery (1972) and studied also by Sherman et al. (1974, 1975). At the time the right eye is neonatally closed, a small lesion is placed in the left retina. This creates two monocular segments relative to the deprived eye: a natural one (MS) and an artificial one (critical segment, CS). Both segments develop in the same way (see text), and this supports the concept of binocular competition during development.

open eye's lesion. Note that the artificial monocular segment occupies regions in central pathways which, without the lesion, would have developed as binocular segment.

With this preparation, Guillery (1972) showed that in deprived geniculate laminae, cells were of normal size only in the natural and artificial monocular

segments. Sherman et al. (1974) then showed that while using the deprived eye, such a cat could visually orient to targets placed only in the natural or artificial monocular segments; also, only in the natural and artificial monocular segments of striate cortex did the deprived eye influence significant numbers of neurons. Finally, Sherman et al. (1975) reported that in the deprived laminae, only the natural and artificial monocular segments contained significant numbers of recordable Y-cells. The pattern of results illustrated in Fig. 5 indicates that the differential response of the binocular and monocular segments to early monocular lid suture is due to some form of binocular competition. While these studies clearly implicate such a developmental mechanism, we still know virtually nothing about the details of the mechanism or even its central site of action.

However, one additional speculation can be made based upon the observation that the geniculate Y-cells, but not X-cells, seem to develop by way of a mechanism of binocular competition. Recently, Ferster and LeVay (1978) suggested that axons from X-cells in layer IVc of cat striate cortex arborize within a single ocular dominance column. The Y-cells, on the other hand, seem to possess axons which ramify across many ocular dominance columns in layer IVab. X-cells may not show binocular competition simply because their projections from one geniculate lamina are not in a position to interact with those from another lamina. Y-cells alone may be in a position to compete binocularly along the lines suggested in Fig. 2, simply because of their more extensive axonal arborizations which permit interactions among axons and terminals from different geniculate laminae.

Effects of Monocular Deprivation upon Retinal Ganglion Cells

Theoretically, it is possible to account for deprivation defects in geniculate cells on the basis of similar defects in their retinal inputs. That is, Y-cells could be missing from the deprived retina, and retinal X-cells in the closed eye could develop poor spatial acuity. Any mechanism requiring interocular interactions (i.e., binocular competition for Y-cells) must almost certainly occur central to the retina, and an earlier study (Sherman and Stone, 1973) reported unchanged proportions of X- and Y-cells in the deprived retina. On the other hand, the noncompetitive deprivation *per se* mechanism implicated for X-cells could well have a retinal origin. We reinvestigated retinal ganglion cells in monocularly deprived cats by recording from optic tract, and we found no evidence for abnormalities in the spatial or temporal contrast sensitivity functions for deprived X- or Y-cells (Kratz, Mangel, Lehmkuhle, and Sherman, 1979). Thus, the retina seems to develop fairly normally despite the lid suture, and the defects described above have a more central origin.

If the data for X-cells have been correctly interpreted, this raises a difficult conceptual problem. Why should deprived geniculate X-cells display spatial deficits for only higher frequencies if their presumed retinal inputs have normal sensitivity throughout the spatial frequency domain? The population of X-cells

(and Y-cells) shows considerable scatter in terms of the sensitivity to high spatial frequencies or spatial acuity, and it may be that only the units with poorer spatial acuity sampled in the optic tract make or maintain effective connections in deprived geniculate laminae.

Summary and Conclusions

Patterns of X- and Y-cell effects It seems clear that geniculate cells do not develop normally during monocular lid suture, and that the consequences and underlying mechanisms of these deprivation effects are quite different for X- and Y-cells. These differences probably depend to some extent on the finding that when they enter the "critical period," X-cells have completed more of their development than have Y-cells (Daniels et al., 1978). Both physiological and anatomical evidence suggests that in the deprived, binocular segment, Y-cells are much more profoundly affected by lid suture than are X-cells. On the other hand, Y-cells seem completely normal in the deprived monocular segment, whereas X-cells are not. This suggests the very different deprivation mechanisms of binocular competition for Y-cells and deprivation *per se* for X-cells.

Functional implications In order to understand these results in a functional or clinical framework, we must first know what the significance of the X- and Y-cell division is for normal cats. Unfortunately, we have only intuitive speculations that can be addressed to this critical point. The most common suggestion (cf. Ikeda and Wright, 1972, 1975) is that X-cells are most concerned with the analysis of spatial patterns; and Y-cells with temporal patterns. However, our recent contrast sensitivity studies (Lehmkuhle et al., 1979a) suggested fairly small differences between these cell groups in terms of sensitivity to high spatial or temporal frequencies (X-cells were slightly more sensitive than were Y-cells to the former, Y-cells more than were X-cells to the latter). These data do not support a differential role for X- and Y-cells based upon spatial and temporal processing. The most dramatic difference in sensitivity between X- and Y-cells occurred in response to low spatial frequencies. To such stimuli, X-cells are fairly insensitive, whereas Y-cells are quite sensitive.

We have thus suggested a different functional dichotomy based upon the psychophysical observations that low spatial frequencies in a visual scene carry the basic form information, whereas the high frequencies add detail (Kabrisky, Tallman, Day, and Radoy, 1970; Ginsberg, Carl, Kabrisky, Hall, and Gill, 1976; Hess and Garner, 1977; Hess and Woo, 1978). Because of their unique sensitivity to these important low spatial frequencies, Y-cells are probably important to basic spatial analysis. X-cells, because of their better acuity and spatial phase dependency (Hochstein and Shapley, 1979a; Lehmkuhle et al., 1979a), probably add detail, such as better acuity, perhaps stereopsis, etc. (for a more complete discussion of this suggestion, see Lehmkuhle et al., 1979a,b). Without Y-cells, spatial vision might be at best rudimentary, but if only X-cells were affected, reasonable spatial vision might still be possible, since low spatial frequency analysis is possible. In support of the latter consequence of the

suggestion, Berkley and Sprague (1978) found that nearly total lesions of area 17, which destroy the X pathways but leave many or most of the geniculocortical projections of Y-cells intact (Stone and Dreher, 1973; Gilbert and Kelly, 1975; Kratz et al., 1977a), produce a cat with excellent spatial vision and only a 20% loss of spatial acuity.

These hypotheses might also explain some of the variability reported in clinical studies of amblyopia of central origin (cf. Hess and Woo, 1978; and many others). If X- and Y-cells are both affected, as in a lid sutured cat, the amblyopia might be maximal. If only the X-cells are affected at higher spatial frequencies, as seems to be the case in cats raised with esotropia or anisometropia (Ikeda and Wright, 1976; Ikeda and Tremain, 1978), the amblyopia would be much less severe and affect only the acuity level for fine details. Finally, the fact that Y-cells are very sensitive to low spatial frequencies could explain why lid suture, which attenuates all spatial frequencies, prevents their normal development, whereas anisometropia, which essentially attenuates only higher spatial frequencies, permits their normal development. X-cells, which are somewhat more sensitive to higher spatial frequencies develop abnormally under any deprivation condition, such as lid suture or anisometropia, which attenuates these frequencies.

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