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Morphology of Retinogeniculate X and Y Axon Arbors in Cats Raised With Binocular Lid Suture

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SUMMARY AND CONCLUSIONS

1. We examined the terminal arbors of single, physiologically identified retinogeniculate X and Y axons in 13 adult cats raised from birth with binocular lid suture. We recorded in the optic tract from 146 retinogeniculate axons. We studied the response properties of each axon encountered and attempted to penetrate it for labeling with horseradish peroxidase.

2. We attempted to classify each retinogeniculate axon as X or Y on a standard battery of tests. We thus identified 46 X and 91 Y axons; 5 axons had unusual response properties, and 4 axons were lost before they could be adequately identified. The X and Y axons had response properties that were completely normal by our criteria. The 5 unusual axons exhibited linear spatial and temporal summation, which is a property of X cells, despite all of their other tested response properties being consistent with those of Y cells.

3. We achieved complete, dark labeling of 13 X and 13 Y axons that form the data base for all of our qualitative and quantitative morphological observations. All of these labeled axons had response properties entirely normal for their X or Y class. Nine of the labeled X axons arise from the contralateral retina and 4 from the ipsilateral retina, whereas the respective numbers for the Y axons are 8 and 5.

4. Each of the individual retinogeniculate X axons form terminal arbors that appeared essentially normal in terms of location within geniculate lamina A or A1, shape, volume, and number of terminal boutons.

5. In contrast, the retinogeniculate Y axons form clearly abnormal arbors with diminished projections, both in terms of bouton numbers and arbor volumes. For Y axons from the contralateral retina, a roughly normal arbor is formed in the C-laminae, despite greatly diminished or absent projections formed in lamina A, something never seen in normal cats. For Y axons from the ipsilateral retina, the projections to lamina A1 are also diminished, and the arbors there are all limited to the ventral half of the lamina, a pattern rarely seen for normal Y axons.

6. The selective reduction in retinogeniculate Y axon arbors in these binocularly lid-sutured cats is consistent with similar observations reported for monocularly lid-sutured and strabismic cats but is quite different from the apparently normal development of retinogeniculate axon arbors in cats raised in complete darkness. At least for deprivation via lid suture, the abnormal development of retinogeniculate Y axons may account for the failure of many geniculate Y cells to develop normal response properties and morphological features in such cats.

INTRODUCTION

The cat's retinogeniculocortical system is comprised of at least three parallel and distinct neuronal pathways known as the W, X, and Y pathways (for reviews, see Refs. 29, 39, 42, 43). A great deal of attention has been focused on the postnatal development of these pathways. In particular, many studies have addressed the abnormalities that develop in these pathways when conditions of visual deprivation are present during an early postnatal critical period (31, 42).

The best understood paradigm of visual deprivation is that produced by monocular lid

suture, a procedure that deprives one retina of all spatial patterns and that produces permanent and dramatic abnormalities in the lateral geniculate nucleus and visual cortex. The Y pathway seems particularly vulnerable to this form of early monocular deprivation. In geniculate laminae innervated by the sutured or deprived eye (i.e., the deprived laminae), somata grow less than normal (21, 23, 55), few normal Y cells can be recorded (14, 28, 41), and many neurons with poor responsiveness that might be abnormal Y cells develop morphologically abnormal dendritic arbors (15). Sur et al. (45) used the technique of intra-axonal labeling with horseradish peroxidase (HRP) of physiologically identified retinogeniculate axons to establish that many Y axons from the deprived eye fail to develop normal terminal arbors in the lateral geniculate nucleus. Thus at least some of the effects seen on Y cells in deprived geniculate laminae must be due to the failure of their retinal input to develop properly (see also DISCUSSION).

Following early binocular deprivation, whether by binocular lid suture or by maintaining the kittens in a completely dark environment, the number of recorded Y cells is also considerably reduced in the lateral geniculate nucleus (27, 33, 41). Given the above-mentioned pattern of results for monocular lid suture, one might predict that the abnormalities of geniculate Y cells produced by early binocular lid suture would also be correlated with abnormalities among retinogeniculate Y axon arbors. However, in a recent study of dark-reared cats, Garraghty et al. (17) examined the morphology of retinogeniculate axon arbors labeled intracellularly with HRP and reported them to be completely normal.

This reported difference in retinogeniculate axon arbors between monocularly lid-sutured and dark-reared cats is surprising and raises many questions. For instance, is this difference due to dissimilarities between lid suture, which permits considerable light but no spatial patterns to reach the retina (30), and total darkness? Is it due to the dissimilar effects of monocular vs. binocular deprivation? To answer these questions, and for other reasons, we thought it appropriate to analyze retinogeniculate arbors in cats reared with binocular lid suture. We used the above-mentioned technique of physiologically identifying retinogeniculate axons and then labeling them with HRP. Whereas retinogeniculate X axon arbors seem to develop essentially normal morphological characteristics after rearing with binocular lid suture, most retinogeniculate Y axon arbors do not. We have published a preliminary report of these findings (51).

METHODS

Subjects

Since many of the procedures used in these experiments have been described in detail previously (for details, see Refs. 12, 41, 44), we provide only a brief outline here. Data were obtained from thirteen cats born in our colony. Before eye opening, they underwent surgical closure of the lids of both eyes and were raised in this fashion with binocular lid suture until they were at least 1 yr of age. They were then prepared for acute experiments.

Electrophysiological methods

We first anesthetized each cat (initially with 4% halothane in a 1:1 mixture of nitrous oxide and oxygen that was replaced by a 7:3 mixture of nitrous oxide and oxygen supplemented with a 1 $mg \cdot kg^{-1} \cdot hr^{-1}$ infusion of pentobarbital sodium), cannulated a femoral vein for infusion of the barbiturate plus paralytic agents (3.6 mg/hr gallamine triethiodide and 0.7 mg/hr *d*-tubocurarine), and performed a tracheotomy. The animal was placed in a stereotaxic apparatus, paralyzed, and prepared for visual neurophysiology. We dilated the cat's pupils, retracted its nictitating membranes, and fitted contact lenses to its corneas. The lenses were chosen by retinoscopy to focus the eyes on the visual stimuli employed in our receptive field analysis of retinogeniculate axons. These stimuli consisted either of targets presented on a tangent screen or of gratings that were generated on a cathode ray tube and sinusoidally modulated in space and time. Bipolar stimulating electrodes were placed across the optic chiasm. We recorded electrophysiological responses both extracellularly and intracellularly through fine-tipped glass micropipettes filled with 3% HRP in 0.2 M KCl buffered to pH 7.4. Both the stimulating electrodes and the recording electrodes were inserted into the brain through hydraulically sealed chambers.

Axons were recorded in the optic tract and identified as X, Y, or other on a battery of the following tests (6, 10, 24, 25): latency of orthodromic response to optic chiasm stimulation; the linearity of spatial and temporal response summation to counterphased sine wave gratings; sustained or transient responsiveness to standing contrasts; receptive field size; ocular dominance; and the polarity (on or off) of the receptive field center. Fol-

lowing extracellular classification, we impaled each axon, confirmed that the impaled axon was the same as the extracellularly studied axon, and iontophoresed HRP into the axon until its resting potential fell to less than one-half of its initial value.

After every penetration, the optic disk was projected onto the plotting screen (11). We never attempted to inject more than three axons from the same eye, and these would only be injected if their receptive fields were widely separated from one another. Because of the precise retinotopic mapping of retinogeniculate arbors within geniculate laminae and the restriction of axons from each eye to a unique set of geniculate laminae (38, 44), we were able unambiguously to assign each labeled axon to one from which electrophysiological data were taken.

Morphological methods

Several hours following the last intracellular injection, each cat was deeply anesthetized with barbiturate (50 mg/kg) and perfused transcardially with 0.9% saline followed by fixative (1% paraformaldehyde and 2% glutaraldehyde in a 0.15 M phosphate buffer). We removed the brain, trimmed it to a block containing the thalamus, and refrigerated this block overnight in a solution of 5% dextrose in phosphate buffer. On the following day, 100-μm thick sections were cut frozen in the coronal plane, and these were reacted with diaminobenzidine intensified with cobaltous chloride (1).

Each well labeled axon was reconstructed by using a drawing tube attachment on a microscope with an oil immersion objective (either a $\times$40, $\times$50, or $\times$63 objective with numerical apertures ranging from 0.95 to 1.40). The relationship of each reconstructed axon to geniculate laminar borders was determined by using dark field illumination and/or by counterstaining selected sections with cresyl violet. For each reconstructed axon, two quantitative measures were made. First, the number of boutons found in the A- and C-laminae were determined. Second, the volume of each terminal arbor was estimated using the same procedure as described previously (44). That is, we measured the area enclosed by the outermost boutons in the drawing of each section and multiplied this area by the 100-μm section thickness to estimate the arbor volume for each section. We summed these volumes for all sections containing the terminal arbor. We compared our data to those reported for normal retinogeniculate X and Y axons that were obtained with identical techniques and that were similarly analyzed (44).

Statistics

Unless otherwise indicated, all statistical comparisons were based on the Mann-Whitney U test.

RESULTS

Electrophysiological properties of retinogeniculate axons

In 13 adult cats raised from birth with binocular lid suture, we recorded extracellularly from 146 retinogeniculate axons. Of these, 46 were X axons, 91 were Y axons, 5 were axons that we believe to be Y axons with unusual spatial and temporal summation properties (see below), and 4 were not identified. These last 4 were not recovered histologically and are not further considered.

On all of our tests, the 137 axons unambiguously identified as X or Y seemed completely normal, as defined by comparison with an analogous population of axons from normal cats studied with identical techniques (44). The X axons displayed linear spatial and temporal summation, whereas the Y axons summed nonlinearly (cf. Ref. 24). Response latencies to optic chiasm stimulation were consistently longer for the X axons than for the Y axons. The range for X axons was 0.5–1.0 ms, with a mean of 0.75 ms; for Y axons, these respective values were 0.25–0.65 ms and 0.45 ms. Receptive field center diameters of the X axons were smaller than those of the Y axons at every eccentricity. The range for X axons was 0.25–3.5° over an eccentricity range of -1.5–41°. For Y axons, the range was 0.5–8.5° over an eccentricity range of -3.5–54°.

Five of our sample of recorded axons were unusual. By most of our criteria, these were Y axons, because their receptive field dimensions, phasic responses to standing contrasts, and response latencies to optic chiasm stimulation were indistinguishable from those seen in the unambiguously identified 91 retinogeniculate Y axons. However, these 5 axons displayed linear spatial and temporal summation in response to sinusoidal gratings, and this linear summation was seen at every spatial frequency to which these cells reliably responded. Although we injected and recovered one of these unusual, presumptive Y axons, it was incompletely labeled and not included for quantitative analysis. However, on the basis of qualitative observations, which included preterminal axonal branching and arbor shape, the morphological features of this axon were indistinguishable both from the other Y axon arbors of the present study as well as from those of normal cats (44).

Of our larger sample of electrophysiologically recorded retinogeniculate axons, 37 were labeled by intra-axonal injection of HRP and recovered for morphological analysis. These include 17 X axons and 20 Y axons. The terminal arbors of 13 X axons (4 from the contralateral retina and 9 from the ipsilateral retina) and 13 Y axons (8 contralateral and 5 ipsilateral) were labeled particularly well, and we focused on these for all of our morphological analyses. Table 1 summarizes many of the response properties seen in these 26 labeled axons. The data from Table 1 indicate that this subpopulation of well-labeled axons is reasonably representative of our larger electrophysiological sample, at least in terms of response properties.

TABLE 1. *Response properties of labeled retinogeniculate axons from binocularly lid-sutured cats*

Eye of Origin	Center Sign	Optic Chiasm Latency, ms	Eccentricity, deg	Center Size, deg	Phasic/ Tonic
X-cell axons, $n = 13$					
C*	On	0.8	46	3.1	T†
C	On	1.0	14	0.5	T
C	On	1.0	24	1.6	T
C	Off	0.75	9	1.5	T
I	On	0.6	8	0.7	T
I	On	0.6	15	0.8	T
I	On	0.7	26	0.8	T
I	On	0.75	17	0.9	T
I	On	0.8	14	0.8	T
I	Off	0.5	19	1.3	T
I	Off	0.6	9	2.0	T
I	Off	0.7	16	0.6	T
I	Off	0.8	3	0.9	T
Y-cell axons, $n = 13$					
C	On	0.4	8	3.0	T
C	On	0.4	6	0.9	P
C	On	0.4	12	1.5	T
C	On	0.45	2	3.8	T
C	On	0.45	4	1.0	T
C	On	0.45	30	3.8	P
C	Off	0.3	21	2.1	P
C	Off	0.6	16	3.8	P
I	On	0.3	20	1.4	T
I	On	0.35	29	1.5	P
I	On	0.5	52	2.8	T
I	Off	0.45	37	6.7	P
I	Off	0.55	13	1.9	P

* C, contralateral; I, ipsilateral. † T, tonic; P, phasic.

Morphology of retinogeniculate X and Y axon arbors

QUALITATIVE OBSERVATIONS. Figure 1 illustrates many of the morphological features seen in the terminal arbors of retinogeniculate X and Y axons following binocular lid suture. These features and the differences between X and Y arbors are qualitatively reminiscent of those seen for X and Y arbors of normal cats (4, 44, 46). Terminal boutons from X axons (Fig. 1*C*) are typically found on short stalks. These boutons, which are relatively spherical and uniform in size, tend to occur in clusters like bunches of grapes. Conversely, boutons from Y axons (Fig. 1*B*) are irregular in size and shape, and they tend to occur en passant along short axon branches. Although, as we shall document below, the extents of retinogeniculate Y axon arbors are abnormally small in the binocularly lid-sutured cats, these arbors nonetheless contain populations of boutons with qualitatively normal morphology.

X axons. X axons appear not to be affected in any significant fashion by binocular lid suture, since the morphological features of these axons are virtually the same as those observed in normal cats (see Refs. 4, 5, 44, 46). Figure 2 illustrates the complete reconstruction within lamina A of one of these X axons that is contralaterally projecting (i.e., it arises from the contralateral retina). As is the case for every contralaterally projecting retinogeniculate X axon in normal cats, the arbor of this axon consists of a dense collection of preterminal axon branches and terminal boutons found primarily in a narrow, cylindrical zone that is strictly limited to lamina A. Within the optic tract, the axon emits a branch that courses medially and innervates the medial interlaminar nucleus (not shown). Furthermore, this axon innervates regions beyond the lateral geniculate nucleus. as evinced by a branch of the axon entering the brachium of the superior colliculus. Figure 3 shows a reconstruction of an ipsilaterally projecting retinogeniculate X axon in a binocularly lid-sutured cat. This axon densely innervates lamina A1 in a normal fashion, and it neither branches in the optic tract nor innervates the medial interlaminar nucleus.

The morphology of the remaining retinogeniculate X arbors also appears qualitatively

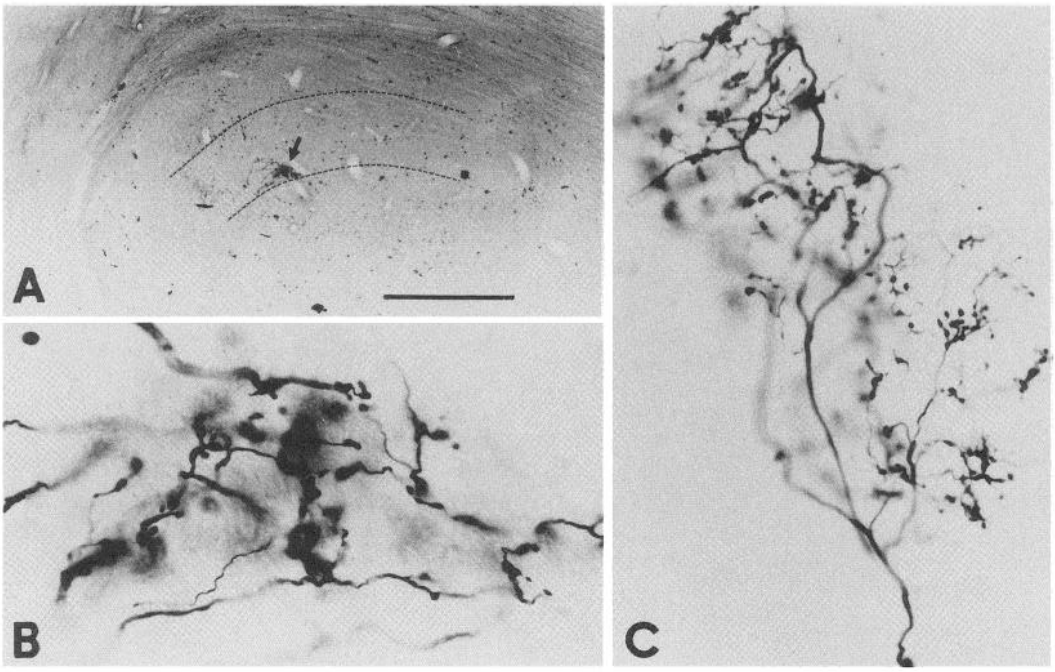

FIG. 1. Photomicrographs of labeled retinogeniculate X and Y axon arbors in binocularly lid-sutured cats. *A*: lower power view of Y axon from left retina terminating in ipsilateral lamina A1. The *dashed lines* bound lamina A1, with lamina A above and the C-laminae below. Note that the terminal arbor is limited to the ventral half of lamina A1. *B*: higher power view of same terminal arbor as depicted in *A*. Most of the boutons here are fairly evenly distributed and occur en passant. *C*: X axon from contralateral retina terminating in lamina A. The boutons here tend to occur in prominent clusters with gaps between. The scale in *A* represents 1 mm for *A* and 50 μm for both *B* and *C*.

normal. For each, the boutons extend dorsoventrally across most of lamina A or A1 in a narrow column. Not a single bouton was located in an inappropriate lamina: boutons were found neither in lamina A from any ipsilaterally projecting axon nor in lamina A1 from any contralaterally projecting axon.

Y axons. In contrast to X axons, many retinogeniculate Y axons exhibit morphological anomalies following binocular lid suture. These abnormalities, which occur in axons from both retinas and with receptive fields representing all retinal eccentricities, show a wide range of severity. Retinogeniculate Y axons from the contralateral retina seem to be more clearly affected by binocular lid suture, and two examples with extreme abnormalities are illustrated in Figs. 4 and 5. In normal cats, contralaterally projecting retinogeniculate Y axons always terminate in lamina A (cf. Refs. 4, 5, 44, 46). However, the arbors illustrated in Figs. 4 and 5 are strictly limited to the C-laminae; no boutons are located in lamina A. The projection to the C-laminae in both of these axons also appears to cover much more territory than is seen for any normal Y axon.

Figures 6 and 7 show reconstructions from two additional contralaterally projecting retinogeniculate Y axons from cats raised with binocular lid suture. Although these axons innervate both lamina A and the C-laminae, they do so in an abnormal fashion. The terminal arbors are strikingly sparse in lamina A, sparser than for any such arbors described in normal cats (4, 5, 44, 46). They are also denser in the C-laminae than in lamina A, a relationship reported so far for only one normal retinogeniculate Y axon (44). Furthermore, the abnormally small arbors seen for the deprived axons in lamina A involve both an abnormally small terminal arbor volume and abnormally few boutons (see below).

Figure 8 illustrates an example of a qualita-

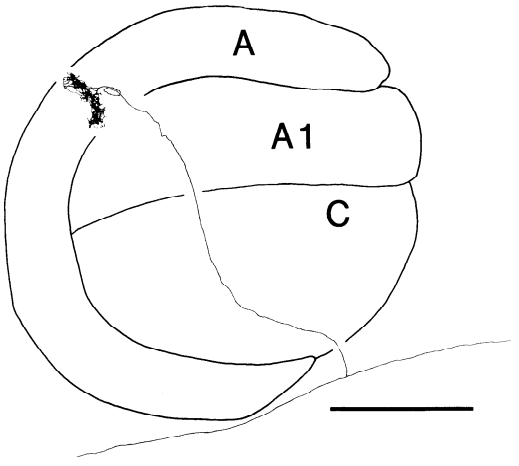

FIG. 2. Reconstruction from serial coronal sections of retinogeniculate X axon in a cat reared with binocular lid suture. The axon arises from the contralateral retina and the arbor reconstructed here is limited to lamina A. Not shown is a sparse terminal arbor in the medial interlaminar nucleus, which arises from a branch in the optic tract in a section far posterior to the series shown here. A, lamina A; A1, lamina A1; C, C-laminae. Scale is 1 mm.

tively normal retinogeniculate Y axon that originates from the contralateral eye in a cat reared with binocular lid suture. This axon branches in the optic tract, with one branch heading into the lateral geniculate nucleus and the other coursing medially and posteriorly toward the brachium of the superior colliculus. The axon terminates densely in lamina A and the C-laminae, but not at all in lamina A1. Portions of its terminal arbor originate from multiple axon branches that converge on the site of dense innervation. In

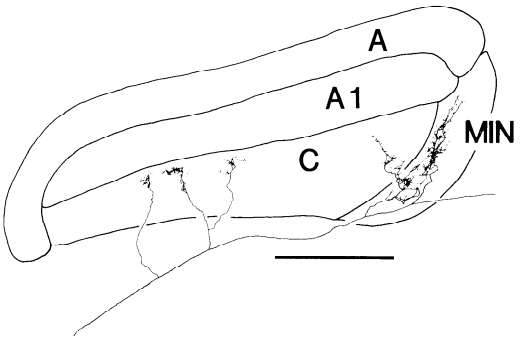

FIG. 4. Reconstruction of a retinogeniculate Y axon arising from the contralateral retina; conventions as in Figs. 2 and 3. Note that the axon innervates the C-laminae, but not lamina A. Scale is 1 mm.

lamina A, the boutons show a tendency to concentrate in the lower half of layer A, but this tendency is seen as well in normal Y axons (5, 44). This axon also innervates the medial interlaminar nucleus.

Figures 9 and 10 display reconstructions of two ipsilaterally projecting retinogeniculate Y axons recovered in cats raised with binocular lid suture. Figure 1, *A* and *B*, shows photomicrographs of another ipsilaterally projecting retinogeniculate Y axon, and the other such axons (not illustrated) are basically similar in morphological features to the three that are illustrated. Although these axons terminate in lamina A1, which is the appropriate geniculate lamina, their arbors are unusual because they only innervate the ventral portion of the lamina. It should be noted that even Y axons in normally reared cats produce more boutons ventrally than dorsally in

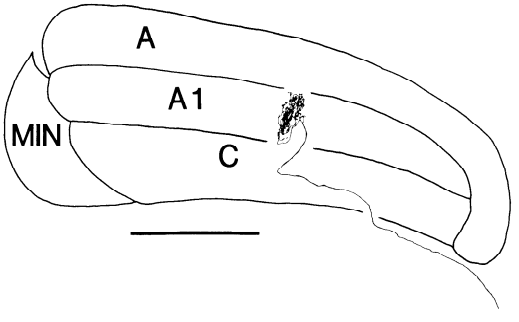

FIG. 3. Reconstruction of a retinogeniculate X axon arising from the ipsilateral retina; conventions as in Fig. 2, and MIN indicates the medial interlaminar nucleus. Scale is 1 mm.

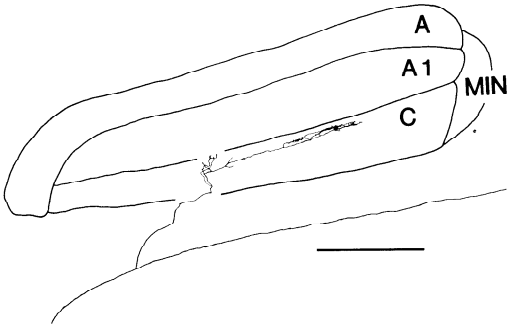

FIG. 5. Reconstruction of another retinogeniculate Y axon similar to that illustrated in Fig. 4; conventions as in Figs. 2 and 3. Scale is 1 mm.

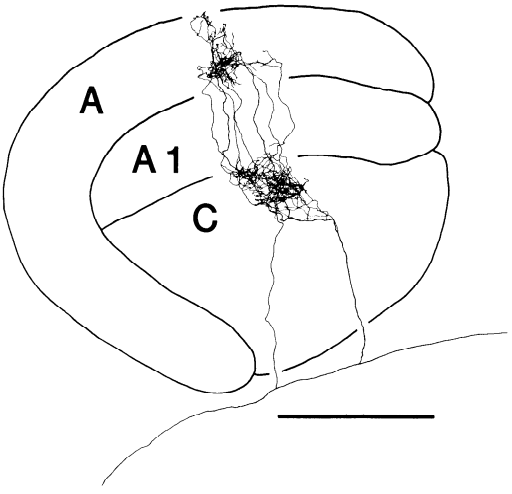

FIG. 6. Reconstruction of a retinogeniculate Y axon arising from the contralateral retina; conventions as in Figs. 2 and 3. Although both the C-laminae and lamina A are innervated, the latter terminal arbor is abnormally small and sparsely populated with boutons. Scale is 1 mm.

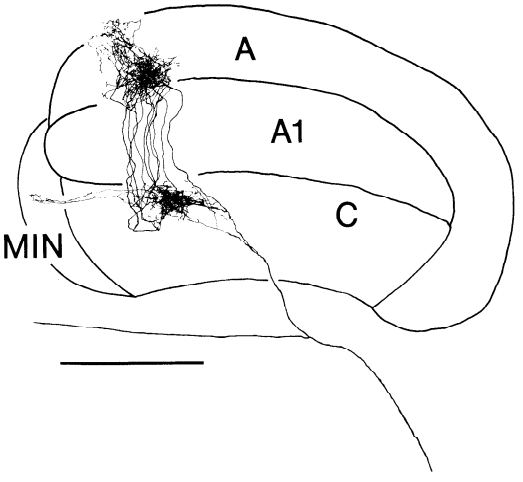

FIG. 8. Reconstruction of a relatively normal retinogeniculate Y axon arising from the contralateral retina; conventions as in Figs. 2 and 3. Scale is 1 mm.

the A-laminae. However, this tendency is fairly subtle, and six of seven ipsilaterally projecting retinogeniculate Y axons in normal cats provide substantial innervation to the dorsal half of lamina A1 (44). This difference between normal and binocularly lid-sutured cats is statistically significant ($P < 0.02$).

QUANTITATIVE ANALYSIS. Our qualitative observations indicate that the majority of Y axons in binocularly lid-sutured cats show a reduced innervation of the geniculate A-laminae when compared to their counterparts in normal cats. Conversely, the terminal arbors of deprived X axons appear qualitatively normal. We quantified these observations by measuring the bouton numbers, terminal arbor volumes, and bouton densities for 13 X and 13 Y axons from binocularly lid-sutured cats and comparing these values with analogous data taken from 14 X and 12 Y axons

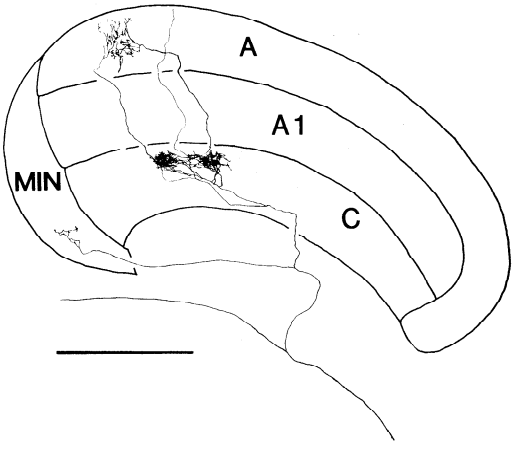

FIG. 7. Reconstruction of another retinogeniculate Y axon similar to that illustrated in Fig. 6; conventions as in Figs. 2 and 3. Scale is 1 mm.

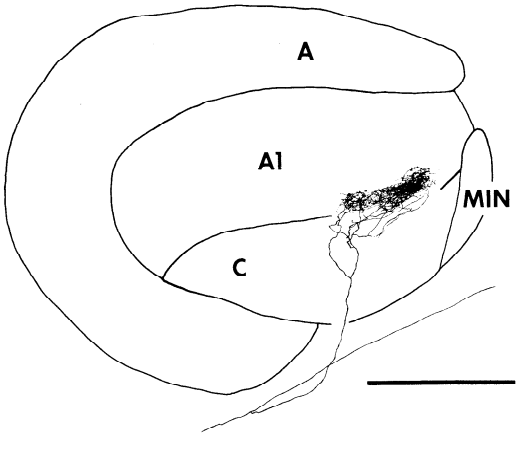

FIG. 9. Reconstruction of a retinogeniculate Y axon arising from the ipsilateral retina and innervating lamina A1; conventions as in Figs. 2 and 3. Note that the terminal arbor is limited to the ventral half of lamina A1. Scale is 1 mm.

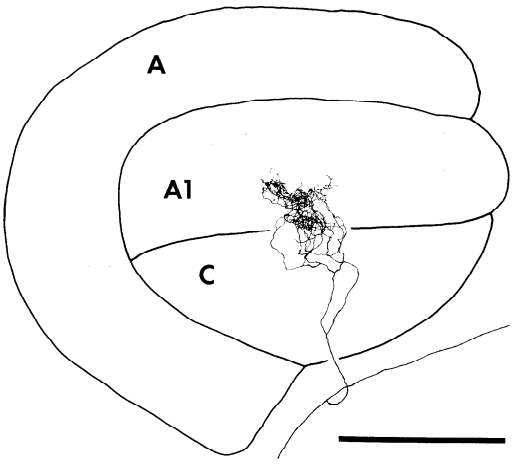

FIG. 10. Reconstruction of another retinogeniculate Y axon similar to that in Fig. 9; conventions as in Figs. 2 and 3. Scale is 1 mm.

obtained from normally reared cats (data from Ref. 44).

X axons. Figure 11 illustrates three measures of retinogeniculate axon arbor extent as a function of the retinal eccentricity of each axon's receptive field. These measures are number of boutons, arbor volume, and bouton density; the last measure is simply the first divided by the second for each axon. X axons from normal cats show no consistent trend with eccentricity for any of the three measures, and our sample from binocularly lid-sutured cats is firmly embedded within the normal axon distribution. No significant differences were found between normal and deprived X axons with regard to bouton numbers (a mean of 584 from normals vs. 515 from deprived; $P > 0.1$), arbor volume (a mean of 2.42×10^{-3} mm from normals vs. 2.62×10^{-3} mm from deprived; $P > 0.1$), or bouton density (a mean of 2.49×10^{5} boutons/mm^3 from normals vs. 2.05×10^{5} boutons/mm^3 from deprived; $P > 0.1$). The same lack of difference between normal and deprived axons was observed even when the analysis is limited to lamina A or A1.

One curious difference between retinogeniculate X axons from normal and binocularly lid-sutured cats did appear in the relationship between arbor volume and the number of boutons; the former divided by the latter represents the bouton density values of Fig. 11*C*. These relationships are shown in Fig. 12*A*. For normal X axons, a significant positive correlation exists between arbor volume and bouton number ($r = +0.59$; $P < 0.05$). However, no such correlation exists for the sample of six X axons from binocularly lid-sutured cats ($r = -0.04$; $P > 0.1$), and the difference between the two populations is statistically significant ($P < 0.01$ on a difference of correlation coefficients). Thus whereas larger arbors tend to contain more boutons in normal cats, no such relationship exists for binocularly lid-sutured cats.

Y axons. In contrast to the essentially normal appearance of retinogeniculate X axon arbors in binocularly lid-sutured cats, deprived Y arbors are quite abnormal when quantitatively compared with their normal counterparts. These differences are readily apparent when numbers of boutons in the terminal arbors are compared. The mean numbers of total boutons, which includes lamina A1 for ipsilaterally projecting axons and lamina A plus the C-laminae for contralaterally projecting axons, are 620 for cats raised with binocular lid suture and 1,200 for normally reared cats. This difference is statistically different ($P < 0.01$), and it is evident even if the comparison is limited to the subpopulations of ipsilaterally and contralaterally projecting axons. For ipsilaterally projecting Y axons innervating lamina A1, the mean values are 502 for deprived axons and 1,103 for normal axons ($P < 0.02$). For contralaterally projecting Y axons innervating lamina A and the C-laminae, the respective mean values are 693 and 1,333 ($P < 0.02$).

The difference between contralaterally projecting Y axons from normal and binocularly lid-sutured cats arises primarily from a reduced projection to lamina A in the latter. Contralaterally projecting Y axons in normal cats project an average of 881 boutons there, whereas this value is only 310 following binocular lid suture ($P < 0.01$). Conversely, there is only a slight, statistically insignificant, reduction in the number of boutons formed by deprived axons in the C-laminae (452 in normal axons vs. 384 in deprived axons; $P > 0.1$). When ratios are formed for each of these axons by dividing the total number of boutons found in lamina A and the C-laminae into the number formed only in lamina A, the mean values are 0.71 for normal axons and 0.36 for deprived axons ($P < 0.02$). Fi-

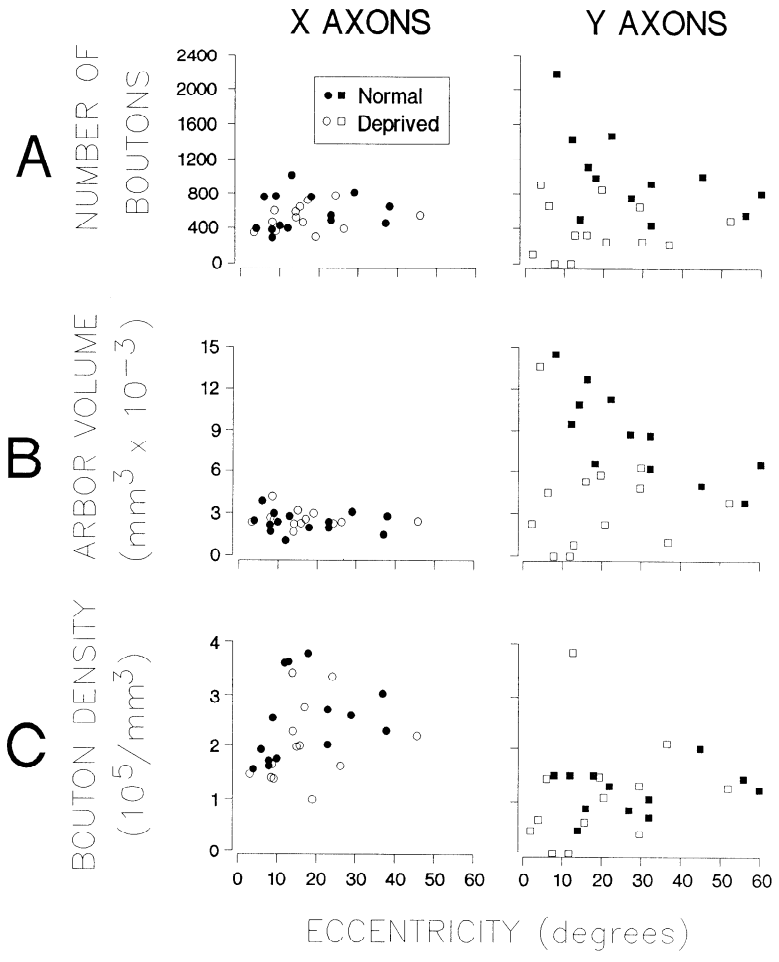

FIG. 11. Quantitative analyses of terminal arbors in lamina A or A1 for retinogeniculate axons. Separately illustrated are values for X and Y axons and for normal and binocularly lid-sutured (Deprived) cats. The normal data were taken from Sur et al. (44). *A*: relationship between eccentricity of the axon's receptive field location and the number of terminal boutons. *B*: relationship between receptive field eccentricity and the volume of the terminal arbor. *C*: relationship between receptive field eccentricity and the density of boutons.

nally, the mean volume for deprived axon arbors is smaller than that of normal axon arbors (8.71×10^{-6} mm^3 for normal arbors vs. 3.89×10^{-6} mm^3 for deprived arbors; $P < 0.001$).

From these data, we conclude that retinogeniculate Y axons in binocular lid-sutured cats provide an abnormally reduced innervation to the A-laminae. Figures 11 and 12 summarize additional quantitative details for Y axon arbors. For normal Y arbors within lamina A or A1, there exist significant, inverse correlations between eccentricity and either bouton number or terminal arbor volume (44). These correlations disappear following rearing with binocular lid suture (Fig. 11, *A* and *B*). Furthermore, bouton number and arbor volume appear to be smaller for deprived Y axons across the entire eccentricity range, although there is some overlap between the two distributions. Nonetheless, since normal Y axons with receptive field locations closer to the area centralis tend to possess larger arbors with more boutons, the abnormalities seen after binocular lid suture tend to be greater for axons with such central receptive field locations.

Figure 11*C* shows that no significant difference in average bouton density was observed between deprived and normal retino-

geniculate Y axons (1.20×10^5 boutons/μm^3 for normal axons vs. 1.12×10^5 boutons/μm for deprived axons; $P > 0.1$). This follows because the abnormalities in bouton numbers and terminal arbor volume for the deprived retinogeniculate Y axons are similar. Figure 12*B* shows further that both normal and deprived Y axons maintain a significant positive correlation between arbor volume and the number of boutons ($r = +0.67$ and $P < 0.02$ for normal Y axons vs. $r = +0.78$ and $P < 0.01$ for deprived Y axons). We find it both curious and inexplicable that, after rearing with binocular lid suture, the more severely affected retinogeniculate Y axons display a normal relationship between arbor volume and bouton numbers, whereas the otherwise unaffected X axons do not.

VARIABILITY IN DEPRIVATION EFFECTS ON Y AXONS. Our analysis of Y axons indicates considerable variability in the morphological abnormalities caused by rearing with binocular lid suture. Some Y axons are severely affected, others seem to be essentially normal, and others are in between. This raises the possibility that the depth of the abnormalities might be related to other properties of the axons and that certain subtypes of Y axon are immune to the deleterious effects of this form of visual deprivation. Thus we looked for relationships between the tested physiological properties (e.g., receptive field size or location, center sign, tonic or phasic responses, and response latency to activation of the optic chiasm) of these axons and their morphological features. We found none. In fact our evidence suggests that *all* Y axons may be affected by binocular lid suture, at least to some degree. This is exemplified by Fig. 13, which illustrates the relationship between number of boutons and latency to activation of the optic chiasm for deprived and normal Y axons. Bouton numbers include only those that occur in lamina A or A1. At every latency, the values for axons recovered in our binocularly deprived cats are smaller than those obtained in normal cats.

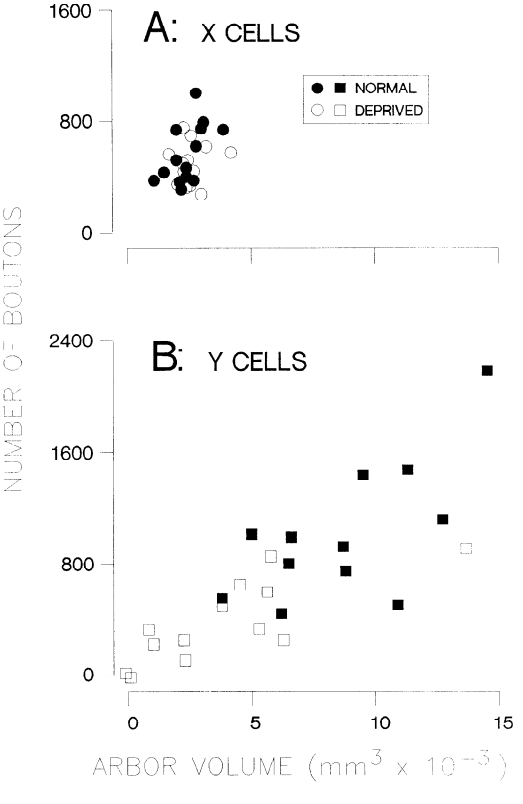

FIG. 12. Relationship between volume of terminal arbor in lamina A or A1 and the number of boutons located there for retinogeniculate axons. Shown separately are X and Y axons from normal and binocularly lid-sutured (Deprived) cats; the normal data were taken from Sur et al. (44).

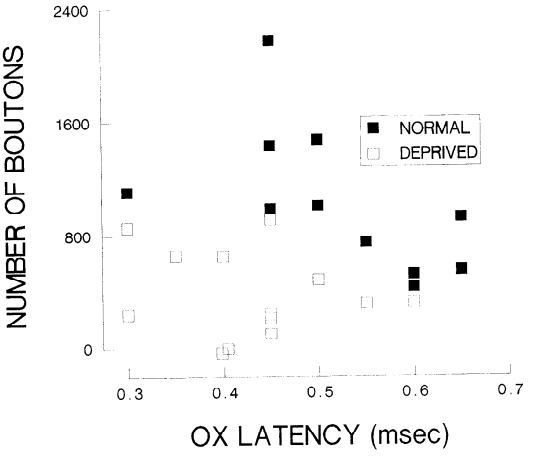

FIG. 13. Relationship for retinogeniculate Y axons between response latency to stimulation of the optic chiasm and the number of boutons located within the axon's terminal arbor in lamina A or A1. Shown separately are axons from normal and binocularly lid-sutured (Deprived) cats; the normal data were taken from Sur et al. (44).

DISCUSSION

We used the technique of intra-axonal HRP labeling to examine the morphology of

single, physiologically identified retinogeniculate axons in adult cats raised from birth with binocular lid suture. Our data suggest that X axon arbors enjoy a relatively normal morphological development during this form of visual deprivation. However, the projection from Y axons to the A-laminae of the lateral geniculate nucleus is severely reduced or absent in these cats. Although some Y axons show greater anomalies than others, our preliminary data suggest that, compared to their counterparts in normal cats, the projections of *all* Y axons are reduced somewhat following binocular lid suture. These data add to the growing body of evidence (reviewed in Refs. 39, 40, 42; see also Refs. 18, 19) that deprivation seriously interferes with the development of the Y pathway and that significant deficits in the central connections of this pathway are apparent as peripherally as the retinogeniculate synapse.

Retinogeniculate axon arbors in other experimental conditions

The results of the present study can be compared against analogous results from a variety of other rearing conditions. These generally indicate that the Y pathway is considerably more susceptible to the deleterious consequences of rearing with visual deprivation than is the X pathway (reviewed in Refs. 39, 40, 42). We can most directly compare our present results with those of other studies that employed similar techniques of intra-axonal labeling with HRP. Such studies have defined the morphology of individual retinogeniculate axon arbors in normal kittens (13, 47), in cats raised with monocular lid suture (45), in cats raised with neonatal removal of one eye (18, 19), in cats raised in total darkness (17), and in cats reared with convergent strabismus (16).

NORMAL DEVELOPMENT. Indirect evidence suggests that, during normal development, retinal X cells are born before Y cells and that retinogeniculate X axons enter the optic tract to innervate the lateral geniculate nucleus earlier than do Y axons (reviewed in Ref. 40; see also Refs. 50, 52–54). Likewise, retinogeniculate X axon arbors seem to develop and mature earlier than do the Y arbors (13, 47). In fact, by 3 wk of age, many X arbors are larger than their adult counterparts, whereas the Y axons are just beginning to establish their arbors in the A-laminae. Sur et al. (47) have suggested that the earlier maturing X axons form exuberant arbors that are pruned as the later maturing Y axons arrive on the scene. By 12 wk of age, both X and Y axons have essentially achieved their adult forms. This postnatal period of 3–12 wk corresponds roughly to the postnatal critical period as defined on the basis of susceptibility to visual deprivation (see Refs. 2, 26). Therefore, the X axons have finished much of their growth by the beginning of the critical period, whereas Y axons develop most of their innervation of the A-laminae during this period.

MONOCULAR LID SUTURE AND CONVERGENT STRABISMUS. The results of the present study from binocularly lid-sutured cats are, with some exceptions, similar to those that have been obtained from cats raised with monocular lid suture (45) or convergent strabismus (16). All three rearing conditions result in a sharp reduction in the innervation of the A-laminae by retinogeniculate Y axons from the lid-sutured or deviated eye. This reduction can either be complete or take the form of a markedly diminished and sparse innervation. Despite this, the same axons, if from the contralateral retina, seem to form a normal number of boutons in the C-laminae. This finding suggests that competition exists between X- and Y-cells during development (see also Refs. 18, 19, 40, 45). It is as if the deprived Y axons can form arbors where they need not compete with the earlier maturing X arbors (i.e., in the C-laminae), but they cannot do so where these X arbors are already established (i.e., in the A-laminae). With a normal visual environment, the later maturing Y axons can successfully compete to establish their arbors in the A-laminae. Monocular lid suture, binocular lid suture, and convergent strabismus place these Y axons at a devastating competitive disadvantage.

Although the data base is still rather small and the results should be regarded as preliminary, the contralaterally projecting Y axons seem differentially affected in their projection to the C-laminae by the different forms of visual deprivation. This projection seems to develop completely normally during either monocular lid suture or convergent strabismus (16, 45). However, whereas contralaterally projecting Y axons in binocularly lid-sutured cats form a normal number of boutons

in the C-laminae, the projection can be abnormally wide (see Figs. 4 and 5). This is particularly true in severely abnormal Y axons that fail completely to innervate lamina A. The functional significance of this unusually expansive projection to the C-laminae is not yet clear, and it is also puzzling why this should be apparent after binocular but not monocular lid suture. Nonetheless, this abnormality of developing retinogeniculate Y arbors in a region normally devoid of X arbors suggests that factors other than X vs. Y competition can affect development (see also below).

MONOCULAR ENUCLEATION. Early postnatal removal of one eye leads to "sprouting" of retinogeniculate axon arbors into the previously denervated laminae (20, 22, 36, 37). That is, for the lateral geniculate nucleus contralateral to the remaining eye, retinal axons are induced to extend their terminal arbors from lamina A into lamina A1; for the ipsilateral side, these arbors spread from lamina A1 to lamina A. By labeling individual retinogeniculate axons with HRP, Garraghty et al. (18) recently showed that all of this sprouting is due to Y axons. Every Y axon showed precisely this pattern of sprouting, whereas each X axon had all of its terminal arbor confined to the laminae appropriate for the remaining eye (i.e., lamina A contralaterally and lamina A1 ipsilaterally). When the remaining eye was lid sutured to favor retinogeniculate X axons in any competitive development, it was still only the Y axons that demonstrated such sprouting into the inappropriate laminae, even though they often failed to develop significant arbors in their appropriate laminae due to unfavorable competition with the X arbors already established there (19). Thus as is the case with monocular or binocular lid suture and convergent strabismus, it is the retinogeniculate Y axons that demonstrate significant plasticity and susceptibility during their postnatal development.

DARK REARING. Garraghty et al. (17) have recently reported that cats reared in total darkness throughout their critical period develop what appear to be morphologically normal terminal arbors among labeled retinogeniculate X and Y axons. To date, this stands as the only example of an abnormal visual environment that fails to affect the development of Y axons. This result is also hard to reconcile with other known abnormalities caused by complete darkness during development (see below).

The fact that dark rearing seems to promote normal development of Y arbors whereas binocular lid suture does not is particularly remarkable. Indeed, part of the rationale for our present study was to determine if this discrepancy reflects the difference between monocular and binocular deprivation or one between dark rearing and lid suture. Since we have also seen dramatic effects of binocular lid suture on the development of retinogeniculate Y axon arbors, we conclude that it is not a difference between monocular and binocular deprivation that determines the development of these arbors. Why lid suture (and convergent strabismus) prevents many retinogeniculate Y axons from developing normal terminal arbors in the A-laminae, whereas complete darkness does not, remains a mystery, although some suggestions are offered below.

Other effects of visual deprivation

In the above paragraphs, we have outlined the deleterious effects of various forms of visual deprivation on the development of retinogeniculate axon arbors. The resultant abnormalities among these arbors should be associated with anomalies in the Y pathway more centrally, such as at the levels of the lateral geniculate nucleus and visual cortex. It is still difficult to relate functional organization at the cortical level differentially to the X or Y pathway, and evidence of cortical abnormalities in visually deprived cats that can be related to deficits in the Y pathway remains indirect (for a discussion of this, see Ref. 42). Thus the paragraphs below concentrate on the status of neurons in the lateral geniculate nucleus of visually deprived cats.

LID SUTURE. For the cases of monocular and binocular lid suture, the abnormalities that develop in retinogeniculate Y axons closely match the abnormalities seen among geniculate neurons. Few Y cells in the geniculate A-laminae develop with normal response properties in such cats; indirect evidence suggests that many geniculate neurons normally fated to develop as Y cells never develop effective retinal inputs, and these cells become morphologically quite abnormal as well (14, 15, 35, 41). It seems likely that these

more central geniculate effects are largely secondary to the failure of retinogeniculate Y axons to develop normal innervation in the A-laminae.

DARK REARING. In view of the above discussion and the earlier assertion that cats raised in total darkness have morphologically normal retinogeniculate axon arbors (17), one might predict that geniculate neurons in dark-reared cats are also quite normal. However, this is not the case. The geniculate A-laminae of dark-reared cats display abnormally few responsive Y cells, a deficiency roughly comparable to that seen after monocular or binocular lid suture (27, 33).

If all of the conclusions from the various experiments are correct as stated, this implies that, despite the apparently normal morphology of retinogeniculate Y axon arbors in these cats, they do not attain a normal functional innervation of their target cells. This apparent discrepancy between the status of retinogeniculate axon morphology and geniculate neuronal response properties also suggests that the above correlations between these parameters for lid-sutured cats may be an epiphenomenon. It is worth noting here that both the conclusions of geniculate neuronal response properties (27, 33) and of retinogeniculate axon arbor morphology (17) are based on a relatively small data base. Given the importance that can be placed on these results, it would seem desirable that both sets of results should be confirmed and the data bases extended.

Contralaterally vs. ipsilaterally projecting Y axons

The effect of binocular lid suture is most obviously seen in some contralaterally projecting Y axons. Although these axons form a normal number of boutons in the C-laminae, they do not innervate lamina A. Normally, all contralaterally projecting Y axons innervate lamina A (4, 5, 44, 46). Thus our results coupled with those from other studies of retinogeniculate axon development suggest that different developmental mechanisms operate in lamina A vs. the C-laminae (see below). While we have also observed reduced arbors in ipsilaterally projecting Y axons, we have not observed any such axons that fail completely to innervate lamina A1. Such a failure would be manifested either by an axon that innervates only the medial interlaminar nucleus or by a parent axon in the optic tract that bypasses the lateral geniculate nucleus altogether. Whereas one might expect from our material that such ipsilaterally projecting axons might be more easily missed than those from the contralateral retina with clear innervation of the C-laminae, this did not seem to be the case. We could account for every ipsilaterally projecting axon that we attempted to label, and none failed to innervate lamina A1.

However, it is possible that a situation comparable to the one seen in contralaterally projecting Y axons does exist for the ipsilaterally projecting ones. This is based partly on the observation of an apparent imbalance between contralaterally and ipsilaterally projecting Y axons (4, 5, 44, 46). The latter fail to innervate the C-laminae to any appreciable extent, focusing instead on lamina A1 plus the medial interlaminar nucleus. In contrast, nearly every contralaterally projecting Y axon branches to innervate lamina A, the dorsal, magnocellular tier of lamina C (which contains a nearly pure population of Y cells), and the medial interlaminar nucleus. Colby (7) recently suggested that lamina A1 contains elements for the ipsilaterally projecting Y pathway that are analogous to those representing the contralaterally projecting pathway in lamina A and magnocellular lamina C. She based this on her observation that magnocellular lamina C (for the contralateral eye) and lamina A1 (for the ipsilateral eye) provide essential innervation to the corticotectal pathway, whereas lamina A does not (7). Perhaps Y cells in the dorsal portion of lamina A1 are functionally related to those in lamina A, while those in ventral lamina A1 are analogous to Y cells in magnocellular lamina C. If so, then ipsilaterally projecting Y axons that innervate the ventral, but not the dorsal, portion of lamina A1 in binocularly lid-sutured cats could reflect developmental mechanisms similar to those that cause contralaterally projecting Y axons to innervate the C-laminae fairly normally despite a greatly reduced innervation of lamina A.

Whereas this hypothesis seems reasonable, it also raises questions about the developmental mechanisms of these axon arbors. As noted above, evidence from related studies has suggested that developing retinogeniculate X and Y axons compete with one an-

other for terminal space, and that visual deprivation can upset the competitive balance in favor of the X axons. Thus Y axons fail to develop normal arbors where X axon arbors are present (i.e., the A-laminae) but can do so where X arbors never exist (i.e., the C-laminae). Since X arbors clearly exist throughout the dorsoventral extent of lamina A1, the relatively normal innervation of ventral lamina A1 by retinogeniculate Y axons in binocularly lid-sutured cats would not be expected on the basis of X vs. Y competitive mechanisms.

Conclusions

We have presented clear evidence that rearing cats with binocular lid suture causes abnormalities to develop in retinogeniculate axon arbors, at least for the Y axons. These results are remarkably similar to analogous results reported for monocularly lid-sutured cats (45) and cats raised with convergent strabismus (16). In the context of other results aimed at understanding the development of retinogeniculate axons in cats, such as retinal ganglion cell birthdates, time of entry of retinofugal axons in the optic tract, normal development of retinogeniculate axon arbors, and the effects of neonatal enucleation on the development of these arbors, three major conclusions emerge: first, compared to X cells, retinogeniculate Y axons develop and mature later; second, the maturation of retinogeniculate axon arbors in the A-laminae may involve a competitive interaction between X and Y axons, although our observation that ipsilaterally projecting Y axons are effected by binocular lid suture more in the dorsal portion of lamina A1 than in the ventral portion can be raised as a challenge to this hypothesis; and third, during the postnatal critical period, these Y axons are much more plastic and susceptible to environmental influences than are the X axons.

However, these results for lid suture and convergent strabismus during postnatal development contrast starkly to the normal morphological appearance of retinogeniculate axon arbors as reported for dark-reared cats (17). At present, these results suggest that, as far as retinogeniculate axon development is concerned, a total absence of photic stimulation is better than the presence of abnormal photic stimulation. Other studies of visual cortex and visual perception (8, 9, 32, 34, 48, 49) have led to a possibly comparable conclusion (however, see Refs. 3, 27). These studies suggest that, whereas abnormal photic stimulation leads to permanent abnormalities, dark rearing merely delays the critical period. Thus a normal visual environment imposed following dark rearing permits visual cortex to develop in a substantially normal fashion. Perhaps a prerequisite to this is the presence of morphologically normal retinogeniculate Y axon arbors.

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