Effects of Convergent Strabismus on the Development of Physiologically Identified Retinogeniculate Axons in Cats

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ABSTRACT

We have studied the effects of surgically induced convergent strabismus (esotropia) on the morphological development of retinogeniculate X and Y axon arbors in cats. Single axons were recorded in the lateral geniculate nucleus or in the optic tract adjacent to the nucleus, classified physiologically, and injected intracellularly with horseradish peroxidase. The arbors of recovered axons were compared with X and Y axon arbors from normally reared adult cats. Our data demonstrate that while X axon arbors are relatively normal, the arbors of Y axons are profoundly affected by rearing with strabismus. Y axons, whether originating from the deviated or the nondeviated eye, have substantially smaller arbors and fewer boutons in the A-laminae of the lateral geniculate nucleus compared to Y axons in normal cats. The C-lamina terminations of contralaterally projecting Y axons in the strabismic cats are unaffected. These results suggest that the postnatal development of retinogeniculate Y axon arbors in the A-laminae is strongly influenced by abnormalities in postnatal visual experience. Furthermore, the present data suggest that, in addition to intraocular competitive interactions between X and Y axons previously proposed to account for the effects of other rearing conditions, interactions between afferents from the two eyes must also be involved in the development of at least Y axons.

Key words: X-cells, Y-cells, esotropia, axon arbors, lateral geniculate nucleus

The cat's visual system is composed of no fewer than three parallel streams of retinogeniculate afferents arising from W-, X-, and Y-cells in the retina (see Sherman, '85, for review). These cell classes differ physiologically and anatomically, and presumably make different contributions to visual perception. Over the past few years, we have used intracellular staining techniques to study the relationship of structure to function in normal adult retinogeniculate X and Y axons (Sur et al., '87). We have also studied aspects of their normal postnatal development (Sur et al., '84) and how their development is affected by manipulations in sensory experience (Sur et al., '82, '85; Garraghty et al., '86a,b, '87). On the basis of these observations, we have identified important differences in the developmental histories of X and Y axons. For example, during postnatal development the arbors of X axons appear to grow exuberantly, attaining their adult form via subsequent pruning or retraction (Sur et al., '84; Garraghty et al., '86a). Y axon arbors, on the other hand, apparently achieve their adult form via steady expan-

sion without growing exuberantly or being pruned (Sur et al., '84; Friedlander et al., '85; Garraghty et al., '86a; see Garraghty and Sur, '88, for review). These differences in normal development, together with observations made in cats reared with monocular lid suture (Sur et al., '82) or monocular enucleation (Garraghty et al., '86b), led us to develop a hypothesis of intraocular competition between X and Y axons during postnatal maturation (see Sur, '88; Garraghty and Sur, '88, for reviews). Moreover, because Y axons, but not X axons, sprout into denervated zones of the lateral geniculate nucleus (LGN) after prenatal or postnatal monocular enucleation, we have suggested that the development of Y axons is particularly sensitive to binocular competition (Garraghty et al., '86b, '88b). In the present experiments, we

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have continued our investigation of the factors influencing X and Y retinogeniculate development by studying cats reared with a convergent strabismus (esotropia). With this rearing procedure, binocular alignment is perturbed; both eyes remain intact (unlike monocular enucleation), and both eyes receive patterned visual inputs of roughly comparable quality (unlike monocular eyelid suture). We have found that Y axons from both eyes are affected by rearing with esotropia while X axons apparently are not. These results reinforce the notion that the development of retinogeniculate Y axon arbors is profoundly influenced by binocular interactions. A preliminary report of these data has appeared in abstract form (Garraghty et al., '88a).

MATERIALS AND METHODS

Data reported here were taken from four cats reared with convergent strabismus. The procedures for surgical induction of strabismus have been described in detail elsewhere (Chino et al., '83, '88). The deviations were induced during the third postnatal week by simple transection of the lateral rectus muscle of the right eye. The kittens were anesthetized with ketamine hydrochloride (35 mg/kg) during the surgery (see White et al., '82). They were then permitted to recover, and they survived for no less than 9 months before being prepared for the terminal experiment.

Our surgical and electrophysiological procedures have been described elsewhere (Garraghty et al., '86b, '87; Sur et al., '87). Briefly, the animals were anesthetized, paralyzed, artificially respirated, and placed in a stereotaxic apparatus. Stimulating electrodes were placed across the optic chiasm so that optic chiasm latencies could be measured for all encountered retinogeniculate axons. Our recording electrodes were beveled micropipettes (95–100 MΩ at 100 Hz) filled with 5–10% horseradish peroxidase (HRP) in 0.2 M KCl and 0.05 M Tris.

During our experiments, micropipettes filled with HRP were lowered through the LGN. When retinogeniculate axons were encountered in the LGN or in the optic tract adjacent to the LGN, they were classified as X or Y by using a standard battery of tests (Garraghty et al., '86b, '87; Sur et al., '87), most prominently linearity of spatial summation and latency of spikes to optic chiasm stimulation. Following classification, the axons were impaled, key physiological properties were quickly reassessed, and HRP was injected iontophoretically into them.

Several hours after the last HRP injection, the cats were deeply anesthetized with sodium pentobarbital and perfused transcardially with 0.9% saline followed by a mixture of 1% paraformaldehyde and 2% glutaraldehyde in 0.1 M phosphate buffer (pH 7.6). The thalamus was blocked in situ and stored overnight in 0.1 M phosphate buffer with 30% sucrose. Blocks were sectioned at 100 µm on a freezing microtome in either the coronal or parasagittal plane. Sections were processed for HRP histochemistry with 3–3' diaminobenzidine with cobalt intensification (Adams, '77). By using Sanderson's ('71) retinotopic maps, each labeled axon could be matched to one from which receptive field location and ocular dominance data were obtained. Well-filled axons were reconstructed by using a microscope equipped with a 50× oil immersion objective and a camera lucida.

Two quantitative measures were taken for each axon (see Garraghty et al., '86b, '87; Sur et al., '87). First, the number of boutons in each terminal arbor within the laminated portion of the LGN was counted. Second, terminal field volumes were estimated by measuring the area enclosed by the outermost boutons in the drawing of each section, multiplying by section thickness, and summing over sections. Comparisons are made to previously reported data from normal X and Y axons that were analyzed in identical fashion (Sur et al., '87). Unless otherwise noted, all statistical comparisons were performed by using the Mann-Whitney U test.

RESULTS
Extracellular recording

In the four strabismic cats we recorded a total of 87 axons, of which 41 were X and 46 were Y, an X/Y recording ratio of about one. In contrast, in normal cats, we have reported recording 138 axons, of which 41 were X and 98 were Y (Sur et al., '87). These X/Y ratios differ significantly (chi-square $= 7.19; P < .01$), reflecting a reduction in the recording rate for Y axons in the strabismic cats. This reduction was evident for Y axons from both eyes. Twenty of the 41 recorded X axons but only 13 of the 46 recorded Y axons were from the ipsilateral retina. These differences in the recording rates of contralaterally and ipsilaterally projecting X and Y axons need not reflect an effect of strabismus. In normal cats, we also record roughly equal numbers of contralaterally and ipsilaterally projecting X axons, but find that contralaterally projecting Y axons are recorded more frequently that ipsilaterally projecting ones (Sur, Esguerra, Garraghty, Kritzer, and Sherman, unpublished observations).

Our sample of X axons had an average optic chiasm latency of 0.84 msec with receptive fields at an average eccentricity of 23.2°. Receptive field centers of the X axons averaged 1.7° in diameter, and, as in normally reared cats (e.g., Cleland et al., '79), were larger at greater eccentricities ($r = .42$, $P < .01$). The Y axons composing our sample responded to optic chiasm stimulation with an average latency of 0.52 msec and had receptive fields averaging 30.7° in eccentricity. Their receptive field centers averaged 2.6° in diameter, and, as with the X axons, receptive field center sizes increased with eccentricity ($r = .36, P < .02$).

Intracellular recording and staining

Of the 87 recorded axons, 20 were sufficiently well-filled with HRP to be reconstructed. Of the 20 recovered axons, eight were X and 12 were Y. Four of the X axons were from the deviated eye and four were from the nondeviated eye. These X axons responded to stimulation of the optic chiasm with latencies ranging from 0.7–1.1 msec (mean, 0.94 msec). Their receptive field center diameters ranged from 0.5–2.6° (mean, 1.2°) at eccentricities ranging from 3–36° (mean, 19.9°). Of the Y axons, eight were from the deviated eye and four were from the nondeviated eye. Contralaterally and ipsilaterally projecting axons were equally represented in our sample of X axons, but most (n = 10) of our sample of recovered Y axons projected contralaterally. These Y axons responded to optic chiasm stimulation with latencies ranging from 0.4–0.7 msec (mean, 0.53 msec). Their receptive field center diameters ranged from 0.7–8.5° (mean, 2.8°) at eccentricities ranging from 6–69° (mean, 30.0°). Thus, the axons we have recovered are physiologically quite representative of the ones we recorded.

X axons. Figure 1 shows the camera lucida reconstruction of an ipsilaterally projecting X axon from the nonde-
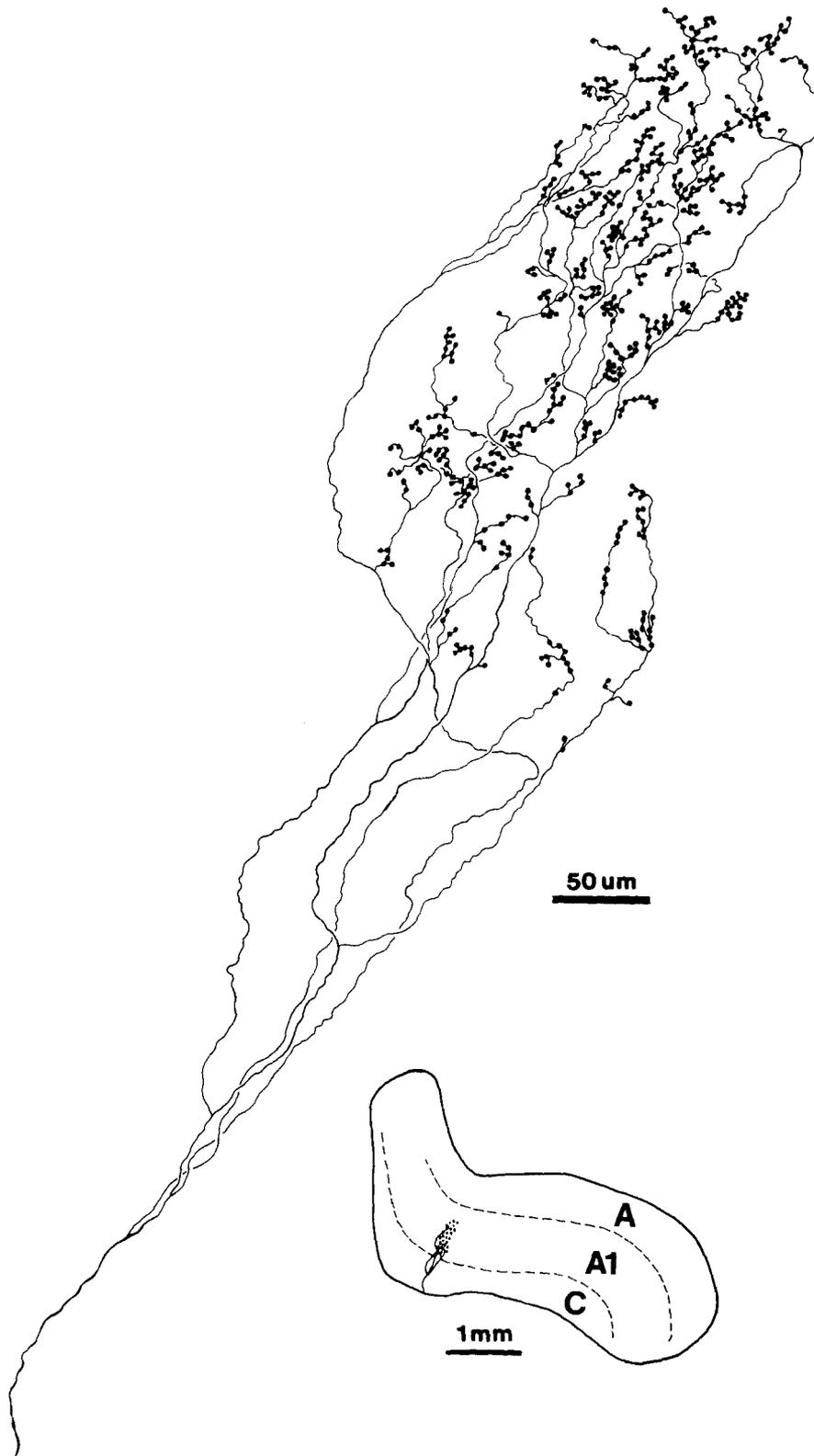
50 um

A

A1

C

1mm

Fig. 1. Representative example of an HRP-labeled retinogeniculate X axon in a strabismic cat. This axon projected ipsilaterally to lamina A1 from the nondeviated eye. The location of this arbor is represented in the smaller drawing of the lateral geniculate nucleus in the lower part of the figure. Abbreviations: A, lamina A; A1, lamina A1; C, C laminae.
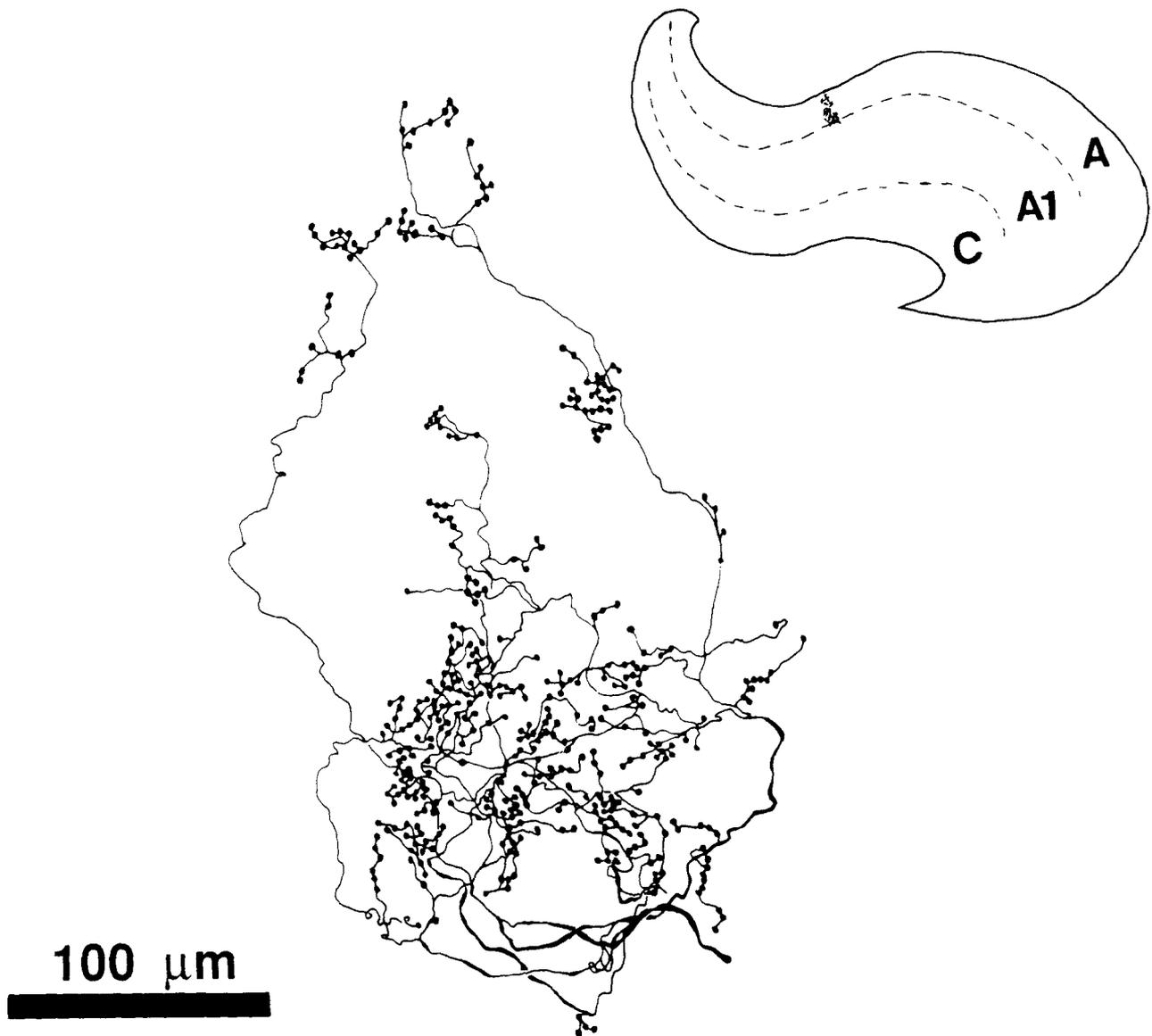
Fig. 2. The reconstruction of an X axon from the deviated eye pro-
jecting contralaterally to lamina A. Conventions as in Figure 1.

viated eye of a strabismic cat. This axon responded to optic
chiasm stimulation with a spike at a latency of 1.1 msec. Its
receptive field center measured 0.8° in diameter, and was
located at an eccentricity of 8°. This axon's appearance is
normal in comparison to X axons from normal cats (cf. Fig.
4; Sur et al., '87). It is also within the normal range with
respect to both the number of boutons in its arbor (n = 762)
and its arbor volume (3.64 mm³ × 10⁻³).

Figure 2 shows the reconstruction of a contralaterally pro-
jecting X axon from the deviated eye of a strabismic cat.
The latency of this axon's response to optic chiasm stimula-
tion was 1.1 msec. Its receptive field center was 0.5° in diam-

eter at an eccentricity of 3°. This terminal arbor is also nor-
mal in terms of bouton number (n = 512) and volume (2.92
mm³ × 10⁻³).

Data from all of the X axons we have recovered from stra-
bismic cats are represented in a scatterplot in Figure 3. Data
from normal cats are presented for comparison. The average
terminal arbor volumes are nearly identical for the two
groups. The mean number of boutons was 15% less in stra-
bismic than in normal cats, but this is not a statistically sig-
nificant difference (P > .10). We also detected no differ-
ences within our sample in bouton number or terminal arbor
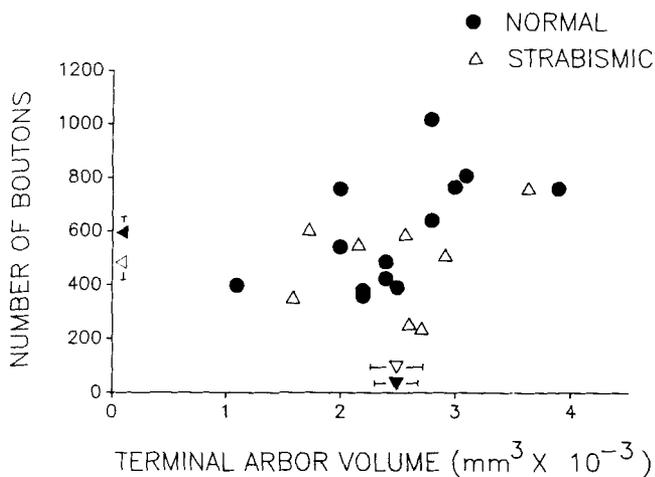volume between axons from the deviated eye and those from

Fig. 3. Scatterplots of the number of boutons for each retinogenicu-late X-cell axon vs. terminal arbor volume. The plots represent data for X axons from strabismic cats (open triangles) and data for X axons from normal cats (closed circles) for comparison. These normal data have been previously reported (Sur et al., '87). Symbols on the ordinate and abscissa represent mean values ± the standard error of the mean. On average, X-cell axons from strabismic cats do not differ from normal in terms of either terminal arbor volume or bouton number.

the nondeviated eye; nor were there any differences between contralaterally and ipsilaterally projecting axons. There were also no differences between X axons as a function of eccentricity, but our sample includes only one axon with a receptive field within the central 5° of visual space (cf. Chino and Kaplan, '88).

Y axons. In contrast to the apparently normal development of X axons in strabismic cats, we have found that the development of Y axon arbors is noticeably affected. The camera lucida reconstruction of an ipsilaterally projecting Y axon from the deviated eye of a strabismic cat is shown in Figure 4. This axon responded to stimulation of the optic chiasm with a latency of 0.6 msec. Its receptive field center was 3.7° in diameter and was located at an eccentricity of 45°. Compared to normal Y axons, this axon has both fewer boutons (n = 391) and a smaller arbor volume (6.09 mm^3 × 10^{-3}).

Figure 5 presents the reconstruction of a contralaterally projecting Y axon from the deviated eye of a strabismic cat. Its latency of response to optic chiasm stimulation was 0.6 msec and its receptive field center measured 1.5 in diameter at an eccentricity of 14°. This axon is even more abnormal than the one represented in Figure 4 in that it has no arbor in lamina A. Its arbor in the dorsal part of the C-laminae, on the other hand, appears completely normal. We (Sur et al., '87), and others (Bowling and Michael, '84) have never recovered a Y axon from the contralateral retina of a normal adult cat without a termination in lamina A. We have, however, seen similar axons in cats reared with eyelid suture (Sur et al., '82; Garraghty et al., '86a). This is the only Y axon without an A-lamina termination that we have recovered in strabismic cats.

The effect of strabismus on the development of Y axons is not restricted to the deviated eye. Figure 6 presents the

reconstruction of a contralaterally projecting Y axon from the nondeviated eye. This axon responded to optic chiasm stimulation with a latency of 0.4 msec. Its receptive field was located at an eccentricity of 22° and its center was 1.2° in diameter. While this axon has retained a termination in lamina A, it is much smaller than normal arbors with respect to bouton number (n = 420) and arbor volume (5.47 mm^3 × 10^{-3}). Overall, Y axons from both eyes of strabismic cats were equally affected by this rearing procedure.

Our Y axon data are summarized in Figure 7. The values represented in Figure 7 are for A-laminae terminations only. We have detected no differences between the C-laminae arbors of strabismic and normal cats either in terms of bouton number or terminal arbor volume ($P > .10$ for both comparisons). We should note that the axon which had no lamina A termination at all is not represented in this figure; its zero values were not included in computations of mean bouton number or arbor volume, or in performing statistical comparisons. Compared to normal Y axons, Y axons from strabismic cats have about 40% fewer boutons ($P < .005$) and arbor volumes that are about 35% smaller ($P < .025$). Since the reductions in these two parameters are roughly equal, bouton density (number of boutons per unit volume) within the Y axon arbors of strabismic cats remains in the normal range.

Another index of the effects of strabismus can be obtained by comparing bouton numbers and arbor volumes of X axons with those of Y axons. In normal cats, Y axon arbors in the A-laminae contain substantially more boutons than X axon arbors ($P < .01$; Sur et al., '87). Y axons also have terminal arbor volumes which are substantially larger than those of X axons ($P < .001$). Bouton densities, however, are greater within X axon arbors than within Y arbors ($P < .001$; Sur et al., '87). Thus, the differences between X and Y arbors are more substantial for terminal arbor volume than for bouton number. When similar comparisons are made for the axons from strabismic cats, X and Y arbors in the A-laminae do not differ in terms of bouton number ($P > .10$) because of the substantial decrease in bouton number for the Y axons. Y arbors in the strabismic cats retain larger terminal arbor volumes than X axons ($P < .005$), but the difference is smaller than in normal cats. While the Y axon arbors are, on average, 2.3 times larger in volume than the X arbors in strabismic cats, the comparable value is 3.5 for normal cats.

DISCUSSION

In the present experiments, we have studied the effects of convergent strabismus on the development of retinogeniculate X and Y axon arbors. Our results demonstrate that rearing with strabismus does indeed affect the development of retinal axons, and does so for axons from both surgically deviated as well as nondeviated eyes. Moreover, this effect is selective in that Y axons are affected while X axons apparently are not. Thus, strabismus, like visual deprivation by eyelid suture (Sur et al., '82; Raczkowski et al., '88), results in developmental anomalies in relatively peripheral components of the visual system.

Extracellular recordings

In normal cats (Sur et al., '87) and in cats reared with various perturbations (Garraghty et al., '86a,b, '87), we have recorded about twice as many Y axons as X axons, despite the fact that retinogeniculate X axons probably outnumber
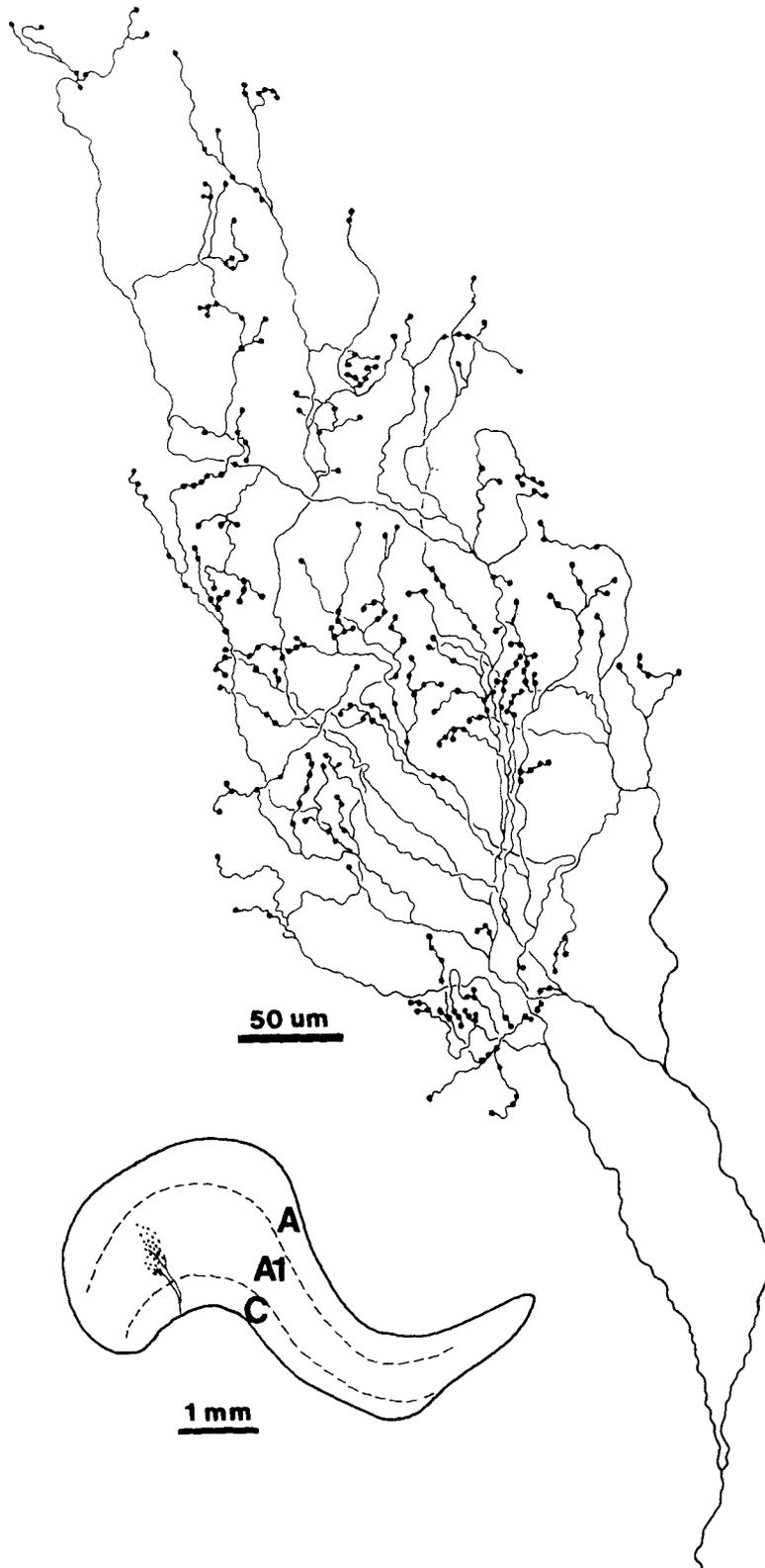
50 um

A

A1

C

1 mm

Fig. 4.   The reconstruction of a Y axon from a strabismic cat. This axon projected ipsilaterally to lamina A1 from the deviated eye. Conventions as in Figure 1.
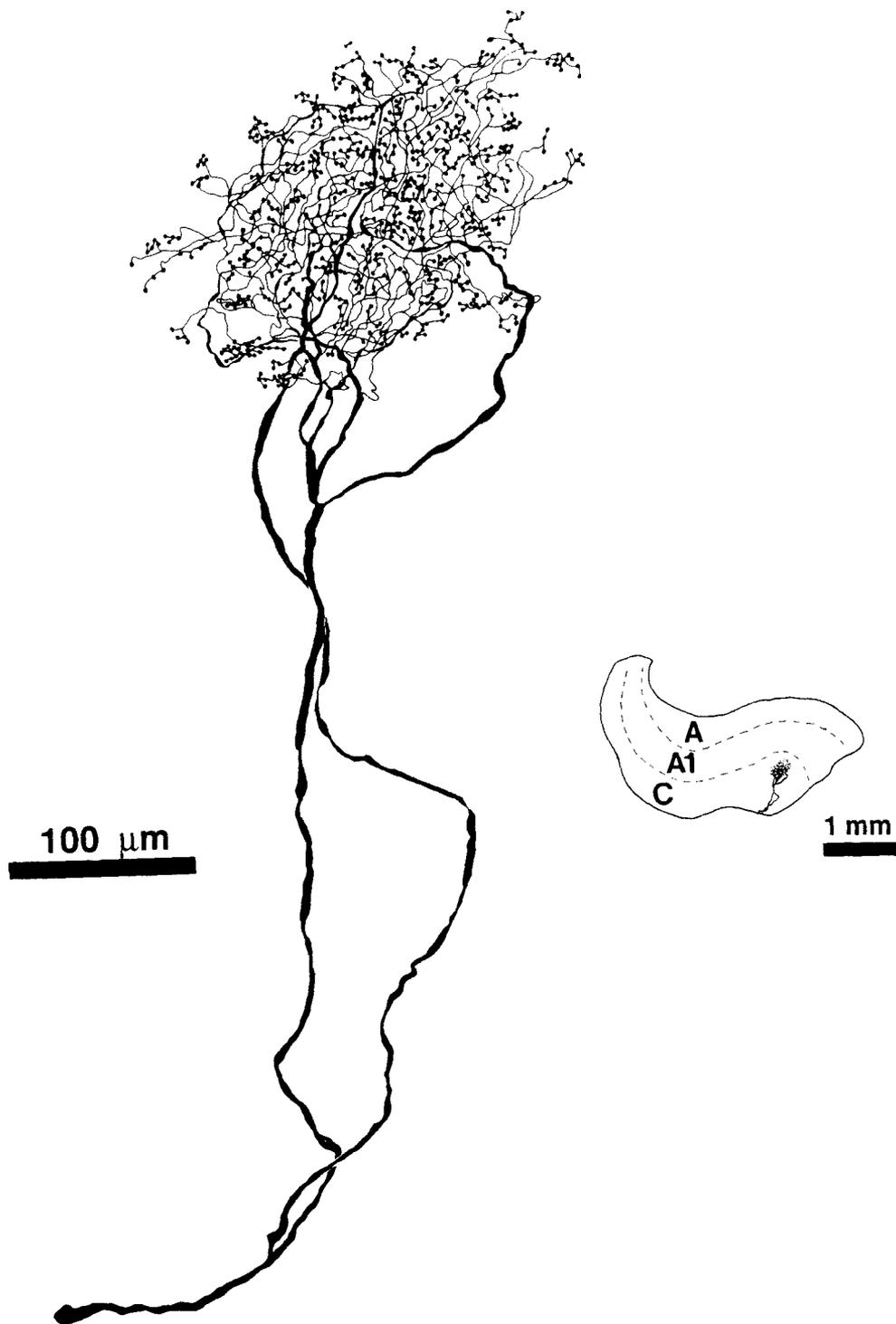
100 μm

A
A1
C

1 mm

Fig. 5.   Contralaterally projecting Y axon from the deviated eye of a strabismic cat. This axon has an arbor in the dorsal part of the C-laminae only and no termination in lamina A, a pattern never found in normal cats. Conventions as in Figure 1.
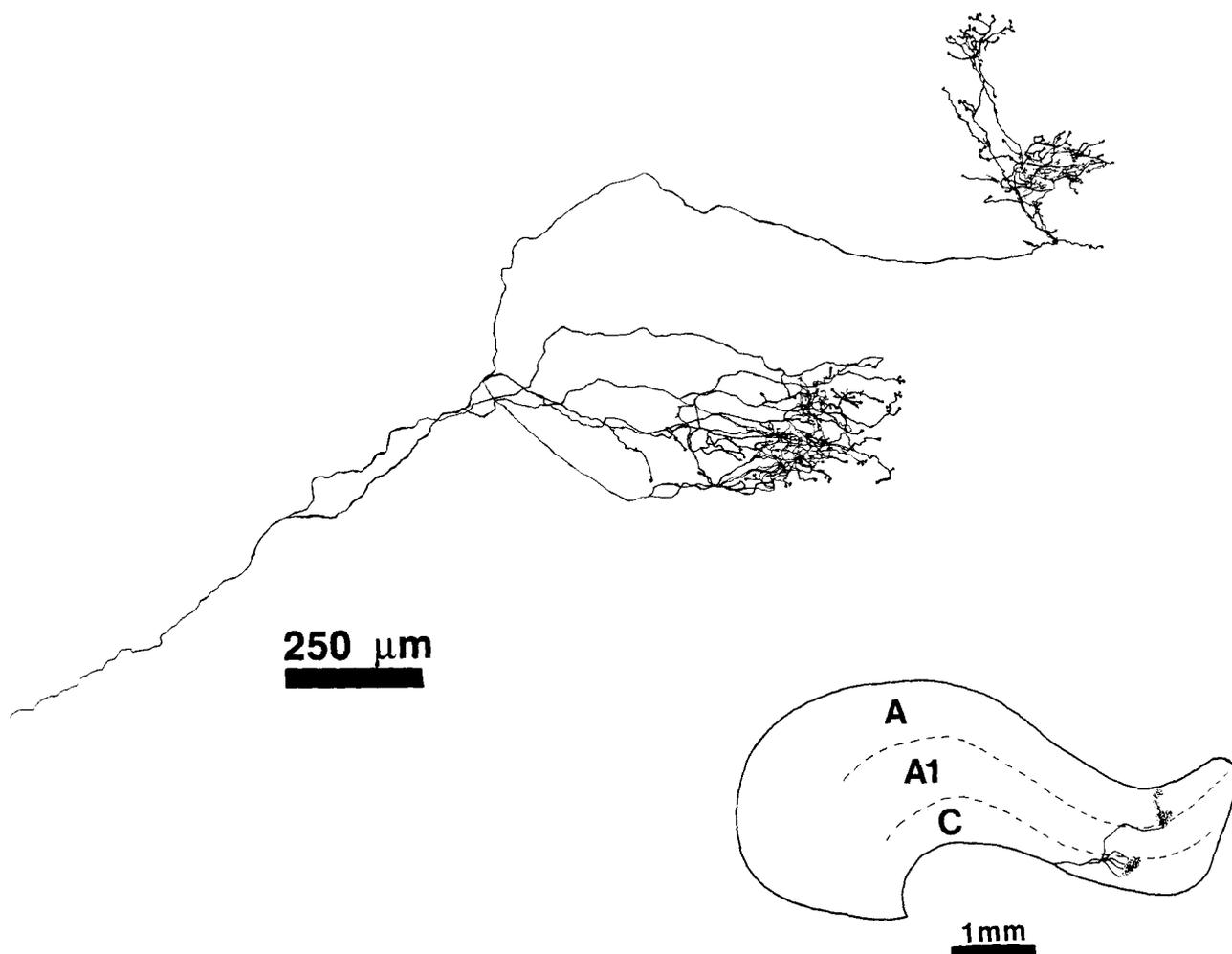
250 μm

A

A1

C

1mm

Fig. 6.   A contralaterally projecting Y axon from the nondeviated eye of a strabismic cat. While this axon terminates in both lamina A and the C-laminae, its arbor in lamina A is much smaller than normal. Conventions as in Figure 1.

Y axons by about ten to one (e.g., Fukuda and Stone, '74; Stone, '78; Hughes, '81; Illing and Wässle, '81). These findings indicate that the recording bias of our micropipettes has been stable across experimental conditions. In contrast, in our four strabismic cats, we have recorded roughly equal numbers of X and Y axons, regardless of whether the recorded axons arose from the deviated or the nondeviated eye. This difference in the recording probabilities for X and Y retinogeniculate axons is intriguing because it replicates an earlier report (Chino et al., '80) in which a "loss" of Y axons from both eyes was found in the optic tract of strabismic cats. Presumably Y axons are encountered less frequently either because they are smaller than normal or because they are less active than normal or both. Paradoxically, this finding demonstrates that the recording probabilities of X and Y retinogeniculate axons are inadequate predictors of the recording rates of X- and Y-cells in the LGN

because no changes have been reported in X/Y ratios in the LGN of strabismic cats (Mower et al., '82; Jones et al., '84).

Why are LGN Y-cells not "lost" in strabismic cats?

One might expect changes in retinogeniculate axonal morphology like those seen in the Y axons in the present study of esotropic cats to have substantial effects on LGN cell physiology. For example, the A-laminae terminations of deprived retinogeniculate Y axons are significantly affected by eyelid suture (Sur et al., '82; Raczkowski et al., '88), and the proportion of Y-cells recorded in deprived geniculate A-laminae is also severely reduced (see Sherman and Spear, '82, for review). Interestingly, the C-lamina terminations of retinogeniculate Y axons are seemingly unaffected by deprivation (Sur et al., '82), and Y-cells in the deprived C layer

● NORMAL
△ STRABISMIC

NUMBER OF BOUTONS

2100 —
1800 —
1500 —
1200 —
900 —
600 —
300 —
0 —

0   2   4   6   8   10   12   14

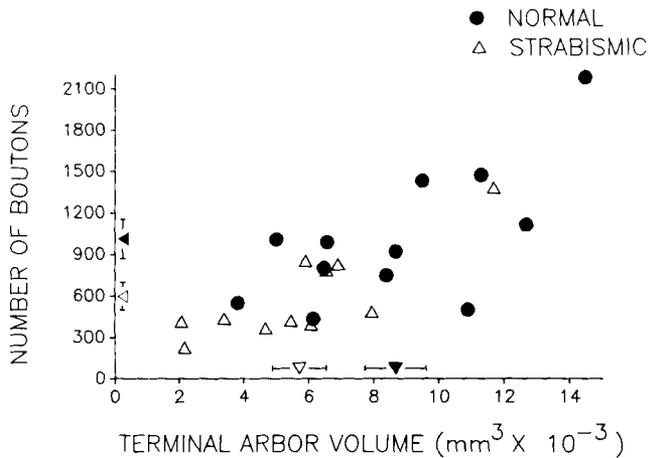TERMINAL ARBOR VOLUME (mm^3 X 10^{-3})

Fig. 7. Scatterplots of bouton number vs. terminal arbor volume for Y axons from strabismic and normal (Sur et al., '87) cats. These values are for terminations in the A-laminae only; C-laminae data are not included. The Y axon shown in Figure 5 which had no layer A arbor at all is also not represented here. Y-cell axons from strabismic cats had both fewer boutons and smaller arbor volumes than normal. Conventions as in Figure 3.

are also apparently unaffected (Spear et al., '89). This pattern of results obviously supports the hypothesis that the Y-cells in the deprived A-laminae of lid-sutured cats are not recorded because of insufficient retinal drive. On the other hand, geniculate A-laminae Y-cells are also "lost" in dark-reared cats (Kratz et al., '79; Mower et al., '81; Kratz, '82; Garraghty et al., '87), but the retinogeniculate terminations of Y axons in dark-reared cats appear normal (Garraghty et al., '87). The present results show that strabismus, like eyelid suture, also "shrinks" the arbors of Y axons terminating in the A-laminae, but the electrophysiological encounter rate for geniculate Y-cells has been reported to be normal (Mower et al., '82; Jones et al., '84). It is possible, therefore, that as far as the probability of recording LGN cells is concerned, other morphological features of retinogeniculate connectivity are more important than our measures of axon arbor morphology. Such features might include the location of retinal axon terminals on LGN cells and the integrative properties of the postsynaptic LGN neuron.

Comparison with visual deprivation: interactions between X and Y axons

We have previously suggested that X and Y axons from one eye interact competitively in the LGN during postnatal development (Sur et al., '82, '84; Garraghty et al., '86a; see Sur, '88; and Garraghty and Sur, '88, for reviews). For example, in cats reared with monocular eyelid suture, deprived Y axons have shrunken arbors in the A layers, where X axons also terminate, but have normal arbors in the dorsal C-laminae, where X axons do not terminate. Furthermore, deprived X axon arbors are larger than normal as if they had retained the exuberant form characteristic of their early development (Sur et al., '82, '84). The present results show that rearing cats with a convergent strabismus selectively impedes the development of retinogeniculate Y axons; however, the X axons are certainly not larger than normal even

though Y axons are shrunken. It is possible, therefore, that the failure of the Y axons to reach normal size in the strabismic cats is not solely due to their being placed at a competitive disadvantage with respect to the X axons. Our results then suggest that factors other than intraocular competition between X and Y axons in the LGN must also play a role in Y axon development.

Other experiments also lead to similar conclusions. Binocular lid suture, for example, causes shrinkage of Y axon arbors in the A-laminae of the LGN, leaving X axon arbors relatively unaffected (Raczkowski et al., '88). Furthermore, the C-laminae terminations of some Y axons in binocularly sutured cats are larger than normal. It is difficult, a priori, to directly compare strabismus and eyelid suture because there are fundamental differences in the nature of the stimulus disruptions arising from them. Eyelid suture simply deprives the retina of patterned visual stimulation. With a misalignment of the visual axes, on the other hand, both eyes receive patterned visual stimulation of roughly comparable quality. Yet with either monocular or binocular suture, and with strabismus, the development of Y axons is affected. Clearly, therefore, the development of retinogeniculate Y axon terminations in the LGN is sensitive to disturbances in normal (binocular) vision even when pattern vision is available. The fact that Y axons from both eyes of esotropic cats are equally affected serves to underscore the developmental sensitivity of this class of retinal afferents to departures from normal binocular experience.

Why are inputs from both eyes affected?

The induction of artificial convergent strabismus involves the manipulation of the lateral rectus muscle of only one eye; yet Y axons from both eyes are equally affected (cf. Chino et al., '83; Holopigian and Blake, '83; Sireteanu and Singer, '84; Garraghty et al., '85). But why should this presumably asymmetric manipulation have bilateral effects? Monocular lid suture also has substantial effects on central visual structures, but its deleterious consequences are confined to inputs arising from the deprived eye (see Sherman and Spear, '82, for review). Because the effects of monocular lid suture are far more pronounced than would be predicted from a knowledge of the effects of binocular lid suture, it has been surmised that the effects of monocular lid suture are due largely to the establishment of a competitive imbalance between inputs from the two eyes (see Sherman and Spear, '82, for review). Artificial strabismus also creates abnormalities in binocular interactions (perhaps because of the mismatch of the two monocular retinal images), but, unlike with monocular lid suture, neither eye has any apparent competitive advantage (Hubel and Wiesel, '65; see Garraghty et al., '85). Thus Y axons from both eyes of strabismic cats might suffer because both eyes are disadvantaged by the deviation of one eye.

Implicit in this argument is the presumption that despite the fact that only one eye is manipulated, the immature nervous system weights the two sets of monocular inputs equally (at least during early postnatal development when retinogeniculate arbors are maturing). It may do this because of an initial inability to discriminate which eye is the source of the disruption. The mature nervous system can identify which eye is the source of such a disruption because it fails to conform to "expectations" associated with eye movement commands (e.g., von Holst, '54). Such expectations could develop during infancy with the development of eye alignment and the experience of predictable changes in

the retinal image with eye movements (Hein et al., '79), for which normal binocular experience is required (e.g., Sherman, '72; Blake et al., '74; Cynader, '79; Cynader and Harris, '80). Without this experientially generated calibration, the immature visual system may be unable to determine that only one eye has been manipulated. If so, this inability to identify the source of the disruption, together with the Y system's dependence upon normal experience for its development, may act together to affect retinogeniculate development in both eyes. A mechanism of this sort would presumably be cortical, and hence the effects on retinogeniculate arbors would be both retrograde and transynaptic.

Alternatively, it is possible that the present results are attributable to direct interactions between the Y axons and do not require a more central arbiter. For example, under normal conditions, axons innervating adjacent laminae along a projection column would tend to fire in a correlated fashion since their receptive fields would be homonymous, and such correlated activity might be an important stimulus for normal development. If the visual axes are misaligned, the activity of axons innervating adjacent laminae would be asynchronous, and lacking information about which set of axons arises from the deviated eye, the development of both sets could be affected. Perhaps comparable experiments in cats reared with exotropia or anisometropia might help in refining our understanding of the mechanisms affecting the development of Y axons in esotropic cats.

CONCLUSIONS

We have shown that rearing cats with convergent strabismus leads to a severe reduction of Y axon arbors in the A laminae of the LGN. Y axons from both the surgically deviated as well as nondeviated eyes are affected, while X axons appear comparatively normal. These results, along with those from cats reared under a variety of other conditions, indicate strongly that the postnatal development of retinogeniculate Y axon arbors can be influenced markedly by disturbances in visual experience.

Our results also suggest that multiple factors are involved in the postnatal shaping of retinogeniculate arbors during normal development. While previous studies have implicated competitive interactions between X and Y arbors from the same eye, at least early in development, the present results indicate that binocular interactions are also involved in the morphological development of at least Y axon arbors. Such interactions may occur in cortex and influence retinogeniculate arbors retrogradely, or they may occur in the LGN itself during development.

ACKNOWLEDGMENTS

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