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Role of Competitive Interactions in the Postnatal Development of X and Y Retinogeniculate Axons

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ABSTRACT

The cat's retinogeniculate pathway is largely composed of X and Y axons, which represent two distinct neuronal streams organized in parallel. Our earlier data, summarized in the previous paper, suggest that the postnatal development of retinogeniculate axon arbors is characterized by competitive interactions between the X and Y axons. Thus, during development, X arbors in lamina A or A1 are initially broad or exuberant before the Y arbors begin to develop adultlike arbors; the X arbors then shrink to their adult form as the Y arbors grow and establish their mature complement of connections; monocular lid suture prevents the rapid growth of Y arbors, which in turn prevents the pruning of X arbors; and monocular enucleation at birth allows X arbors from the remaining eye to retain their exuberance although completely confined to their appropriate lamina A or A1, whereas the Y arbors develop aberrant extensions into adjacent, previously denervated laminae. We now provide additional evidence for the role of competition between retinogeniculate X and Y axons during development. The addition of visual deprivation by lid suture of the remaining eye to monocular enucleation at birth causes no apparent change in the morphology of X arbors in laminae A and A1. In contrast, the Y arbors of such cats continue to form extensive translaminar sprouts in the previously denervated laminae despite severely reduced terminations in the lamina A or A1 normally innervated by the remaining eye. We interpret these new data, in conjunction with our earlier data, as follows. If retinogeniculate X and Y arbors compete for synaptic space during postnatal development, terminations of Y axons can be affected by lid suture only in geniculate laminae where terminations of X axons are also present. Thus, Y axon arbors are severely reduced in deprived lamina A or A1 following lid suture whether or not the other eye is removed. Where X arbors are not present, such as in lamina C or the laminae inappropriate for the remaining eye after removal of the other, the lid suture has no obvious effect on development of the Y arbors.

Key words: monocular enucleation, visual deprivation, X-cells, Y-cells, visual system development, retinogeniculate axons

Retinogeniculate X and Y axons in cats not only display different morphological features in the adult (Bowling and Michael, '80, '84; Sur and Sherman, '82), but they also mature with different time courses (Sur et al., '84) and are affected in different ways by early sensory manipulations such as lid suture (Sur et al., '82) or monocular enucleation (Garraghty et al., '86). On the basis of these observations, we have formulated a working hypothesis regarding interactions between these retinogeniculate X and Y axons during normal and perturbed postnatal development (see also

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Sherman, '85). According to this hypothesis, X axons are exuberant early in postnatal life and are pruned to their normal adult form as a consequence of competitive interactions with later-maturing Y axons (Sur et al., '82, '84). This competition occurs during the first few months of life (Sur et al., '84) and thus coincides with the "critical period" defined by studies of visually deprived kittens (reviewed in Movshon and Van Sluyters, '81; Sherman and Spear, '82). However, if Y axons are placed at a competitive disadvantage by means of lid suture, they fail to develop mature terminal arbors in geniculate laminae A and A1, which in turn permits the X axons to retain their exuberant arbors (Sur et al., '82). These deprived Y axons do, however, develop normal terminal arbors in lamina C, where X axons never seem to form arbors that could compete with those of developing Y axons. Monocular enucleation at birth also allows X axons to retain their neonatal exuberance, but it now does so not at the expense of Y axons that form translaminar sprouts to innervate the previously denervated laminae (Garraghty et al., '86). Thus early monocular enucleation results in abnormally large terminal arbors for both axon classes, but those of X axons are entirely confined to the appropriate laminae whereas the Y axons are not so confined. Presumably, the later-maturing Y axons find it easier to expand into the deafferented laminae than to displace X axons within the normal target layer.

Since lid suture seems to shift the competitive advantage from the later-developing Y axons to the exuberant X axons, and since monocular enucleation leads to sprouting of only the Y axons, we wondered what the combined effects of these manipulations might produce. Two distinct and opposite possibilities seemed worth exploring. First, perhaps the lid closure in monocularly enucleated kittens would prevent the Y axons from developing translaminar sprouts and even permit the X axons to do so. Second, perhaps the lid closure in these cats would encourage a partial segregation of X and Y axon terminal arbors, because while the deprived Y axons might still form translaminar sprouts (while the X axons cannot), these Y axons might compete unsuccessfully with the X axons for terminal space in the normally innervated laminae. In this case, one might expect retinogeniculate Y axons to develop their terminal arbors chiefly in the inappropriate, previously denervated laminae, whereas the X axon arbors remained confined to the appropriate laminae.

To test these possibilities, we reared kittens to adulthood with one eye removed at birth and the other sutured, and we then studied the morphological characteristics of the retinogeniculate axon arbors that developed in these animals. Our results tend to support the second of the above-mentioned possibilities. We believe that these results lend further credence to the hypothesis that retinogeniculate X and Y axon arbors compete with one another for the development of terminals in laminae A and A1 of the cat's lateral geniculate nucleus. We have published a preliminary report of these results in abstract form (Garraghty et al., '85).

MATERIALS AND METHODS

With the one minor methodological exception involving eyelid suture, all of the methods employed in the present study are identical to those described in the previous paper for a similar study of monocularly enucleated kittens (Garraghty et al., '86). Briefly, five kittens from our breeding colony underwent surgery within 24 hours of birth to remove one eye and have the lids of the remaining eye sutured closed. The surgery was performed under Metofane (Pitman-Moore) anesthesia, and the orbits were infiltrated with a long-lasting, local anesthetic agent. Frequent inspections of each kitten ensured that the lids remained closed over the remaining eye during the animal's life.

These animals ranged from 14 to 24 months of age at the time they were prepared for acute intracellular recording. The methods used were identical to those reported in the previous paper (Garraghty et al., '86). We used micropipettes filled with horseradish peroxidase (HRP) to record single retinogeniculate X and Y axons. After physiological study, an axon was impaled, and HRP was iontophoresed into it to permit subsequent visualization of its entire arbor in the lateral geniculate nucleus. For all axons, we measured the volume of arbors and the number of terminal boutons contained in these arbors. These parameters were then compared to those of monocularly enucleated cats without lid suture of the remaining eye (Garraghty et al., '86) and to normal cats from our previously reported experiments that employed the same techniques (Sur and Sherman, '82; Esguerra et al., '85; Sur et al., '86).

All statistical comparisons were made by using the Mann-Whitney *U*-test.

RESULTS

We recorded extracellularly from 76 axons of retinal ganglion cells in the optic tract of the five monocularly enucleated-plus-lid-sutured cats. These included 26 X and 50 Y axons. Of these, nine X and 12 Y axons were successfully and sufficiently labeled with HRP for subsequent quantitative morphological analysis. As noted in Materials and Methods, we compared these data to similar data obtained from eight X and 11 Y axons of normal cats (Sur and Sherman, '82; Esguerra et al., '85; Sur et al., '86) plus eight X and 15 Y axons of cats raised with monocular enucleation without additional deprivation by lid suture.

Physiology of retinogeniculate X and Y axons

As expected from normal cats, receptive field center sizes varied with eccentricity. In the enucleated-plus-lid-sutured cats, the 26 X axons had an average receptive field center diameter of 0.8° with an average eccentricity from the area centralis of 16°, and the analogous values for the 50 Y axons were 2.1° for average center diameter and 26° for average eccentricity. Response latency to optic chiasm stimulation for the X axons ranged from 0.7 to 1.2 msec with an average of 0.9 msec, and for the Y axons the range was 0.4 to 0.7 msec with an average of 0.5 msec. These values differ neither from those of retinogeniculate axons in normal cats (Hoffmann et al., '72; Kratz et al., '79; So and Shapley, '79; Sur and Sherman, '82, '84; Schroeder et al., '86) nor from those in monocularly enucleated cats without additional deprivation by lid suture (the previous paper, Garraghty et al., '86). We also detected no abnormality in general responsiveness or spatial summation properties among our sample of recorded retinogeniculate axons. Table 1 summarizes many of these response properties for our sample of recorded axons.

The subpopulation of nine X and 12 Y axons that were successfully labeled in the monocularly enucleated-plus-lid-sutured cats had physiological properties that were representative of the larger sample of recorded axons. For the labeled retinogeniculate X axons, the average values for receptive field center size, eccentricity, and latency to optic

TABLE 1. Summary of the Physiological Properties of the Retinogeniculate Axons From Monocularly Enucleated and Lid-Sutured Cats That are Analyzed in Detail in the Present Study, and the Figure(s) in Which They Are Illustrated

Axon class	Center type	Center size	Optic chiasm latency (msec)	Eccentricity	Figure
X	Off	1.1°	0.9	11°	1, 5
X	On	0.4°	0.8	12°	2
X	On	0.6°	0.8	22°	3
X	Off	0.9°	0.8	24°	4, 6A
X	Off	0.9°	0.85	12°	6B
X	On	0.3°	0.9	9°	6C
X	On	0.4°	1.0	7°	7A
X	On	1.5°	0.75	26°	7B
X	On	0.3°	1.0	19°	7C
Y	On	1.4°	0.55	27°	8
Y	On	0.4°	0.7	12°	9
Y	On	1.5°	0.45	28°	10a, b, c (left), 12B
Y	Off	1.4°	0.6	23°	10c (right), d, e, 11B
Y	Off	2.9°	0.5	20°	11A
Y	On	1.4°	0.65	36°	12A
Y	Off	3.6°	0.5	30°	12C
Y	Off	2.9°	0.5	18°	13A
Y	On	3.3°	0.5	54°	13B
Y	Off	0.9°	0.65	6°	13C
Y	On	3.5°	0.45	29°	14
Y	Off	3.3°	0.4	38°	15

chiasm stimulation were, respectively, 0.71°, 15.8°, and 0.9 msec. These respective values for the labeled Y axons were 2.2°, 25°, and 0.5 msec.

Qualitative morphology of retinogeniculate X and Y axon arbors

The morphology of retinogeniculate X and Y axon arbors in normal cats has been well documented (Bowling and Michael, '80, '84; Sur and Sherman, '82). Briefly, retinogeniculate axon arbors are normally confined strictly to their appropriate laminae based on their eye of origin (e.g., laminae A and C if from the contralateral retina and lamina A1 if from the ipsilateral retina). X axons innervate only lamina A (if from the contralateral retina) or lamina A1 (if from the ipsilateral retina) in relatively small zones including roughly 500–1,000 terminal boutons. Each of the Y axons typically branches to innervate several geniculate regions: lamina A, lamina C, and the medial interlaminar nucleus if from the contralateral retina; lamina A1 and the medial interlaminar nucleus if from the ipsilateral retina. The Y arbors are much larger than those of X axons and develop roughly twice as many terminal boutons in lamina A or A1 alone than do the X axons.

Most of the following documentation is limited to the new observations of retinogeniculate axons from the experimental cats of the present study. These observations are compared to our analogous observations made with identical techniques from normal cats (Sur and Sherman, 1982; Esguerra et al., '85; Sur et al., '86) and cats raised with monocular enucleation alone (Garraghty et al., '86). Our analysis of axon arbors in monocularly enucleated-plus-lid-sutured cats are limited to terminal arbors in the patently laminated region of the lateral geniculate nucleus (i.e., the A- and C-laminae) and do not include terminal zones in the medial interlaminar nucleus.

X axons. Of the nine X axons recovered for detailed anatomical study in the monocular-enucleated-plus-lid-sutured cats, four were ipsilaterally projecting (i.e., the remaining eye was ipsilateral to the lateral geniculate nucleus in question) and five were contralaterally projecting. The terminal arbors of the contralaterally and ipsilaterally projecting X axons are essentially the same in shape and general appearance.

Figure 1 represents the *camera lucida* reconstruction of an ipsilaterally projecting X axon recovered from a monocularly enucleated-plus-lid-sutured cat. As with X axons from normally reared cats (see Fig. 1 of Sur and Sherman, '82) or from monocularly enucleated and visually experienced cats (see Figures 1, 4, and 5 of the previous paper, Garraghty et al., '86), the terminal field of this axon is relatively restricted in the plane parallel to the laminar borders and is clearly confined to its appropriate target lamina (laminar borders can be clearly assigned in monocularly enucleated-plus-lid-sutured cats; see the previous paper, Garraghty et al., '86; and see Discussion). Figures 2 and 3 present the reconstructions of two contralaterally projecting X axons from a monocularly enucleated-plus-lid-sutured cat. Most of the terminal arbors of these two axons were found in the same anteroposterior extent of lamina A and thus were reconstructed from the same coronal sections. These axons also display appropriately restricted terminal arbors that appear grossly normal. Although the axon illustrated in Figure 3 may seem somewhat unusual, because its terminal arbor is split into two discrete clumps, such a geometry has also been seen in retinogeniculate X axon arbors from normal cats (Hamos, et al., in preparation).

The photomicrographs presented in Figures 4 and 5 further indicate that the pattern of terminal boutons is qualitatively normal for these X axon arbors. Both of these examples illustrate X axons that project ipsilaterally to lamina A1. The terminal boutons tend to occur in clusters at the end of short stalks, and they are relatively consistent in shape and size.

Figures 6 and 7 display the terminal bouton patterns for the remainder of the sample of X axons from the monocularly enucleated-plus-lid-sutured cats. Ipsilaterally projecting axons are represented in Figure 6 and contralaterally projecting axons in Figure 7. Again, it is clear that all of these axons have terminal arbors restricted to the appropriate laminae. In comparison to X axons from normally reared cats (see Fig. 1 of Sur and Sherman, '82; and Figs. 5, 6 of Bowling and Michael, '84), the gross morphologies of these axons appear normal, despite their abnormally large size (see below).

Y axons. Unlike the retinogeniculate X axons in the monocularly enucleated-plus-lid-sutured cats, the Y axons formed arbors that were clearly abnormal. Each of these showed at least some evidence of "sprouting" into the previously denervated lamine A or A1. This innervation of inappropriate laminae was more dramatic for the ten contralaterally projecting Y axons than for our two ipsilaterally projecting examples.

Contralaterally projecting Y axons from monocularly enucleated and lid-sutured cats are shown in Figures 8 and 9. These axons have strikingly abnormal terminal arbors with three discrete regions of termination in laminae A, A1, and C. The inputs to lamina A1 represent inappropriate terminal zones for contralaterally projecting retinogenicu-

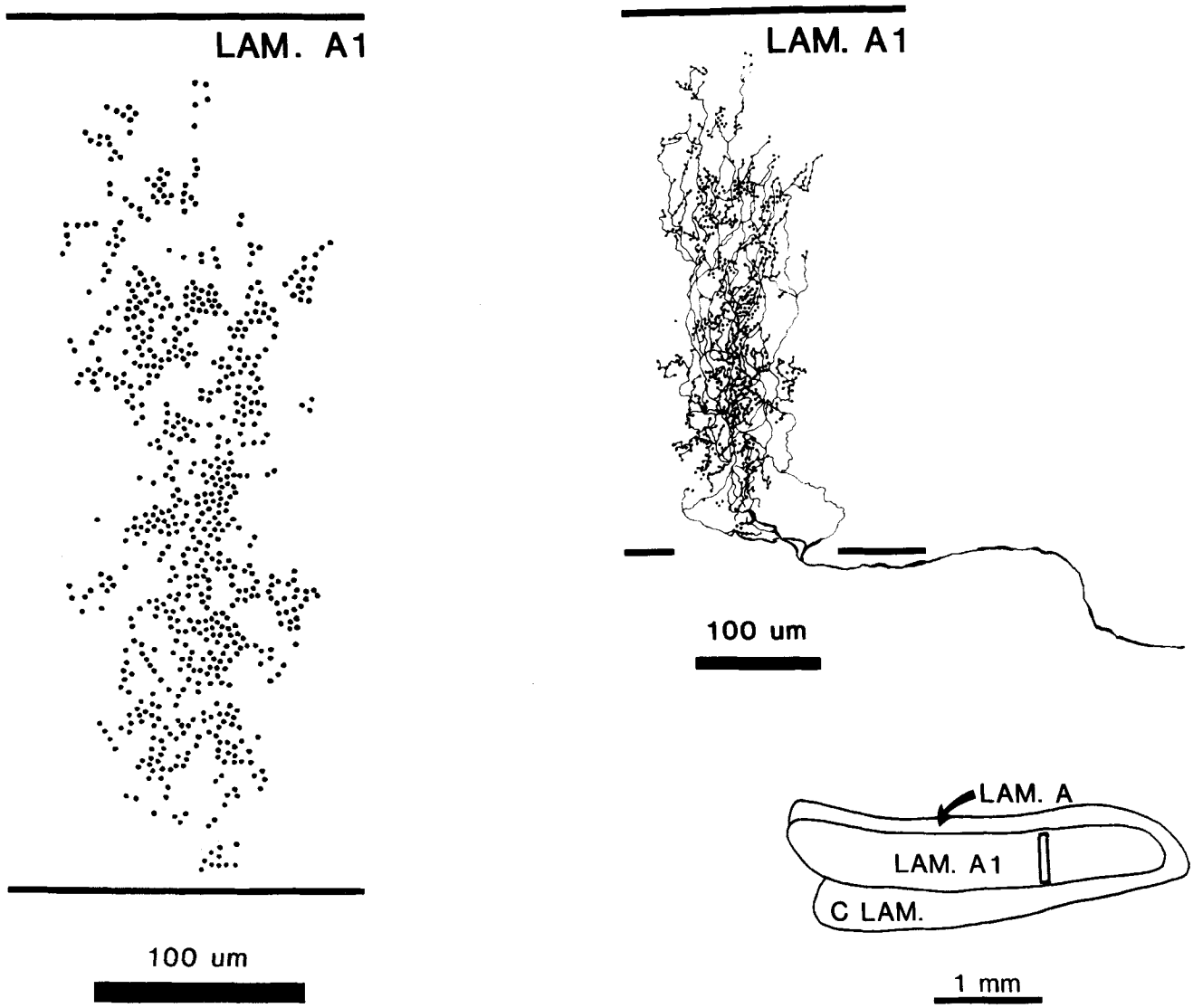


Fig. 1. *Camera lucida* reconstruction of an ipsilaterally projecting X axon recovered from a monocularly enucleated-plus-lid-sutured cat. In the small drawing of the lateral geniculate nucleus in the lower right, the rectangle shown in lamina A1 depicts the location of the terminal arbor. The arbor is completely reconstructed in the drawing on the right, and the pattern on the left illustrates the location of the terminal boutons with dots. The solid black lines above and below the representations of this arbor reflect laminar borders, in this case for lamina A1. This axon's terminal arbor is appropriately restricted to lamina A1.

late axons. In addition to the very obvious translaminar sprouting into lamina A1, it should be noted that the terminal arbors in lamina A are very small relative to those of Y axons in normally reared (cf. Fig. 2 of Sur and Sherman, '82; Figs. 5, 6 of Bowling and Michael, '84) or monocularly enucleated cats without additional deprivation by lid suture (cf. Figs. 9, 11, 12 of the previous paper, Garraghty et al., '86). Indeed, the terminal zones in the inappropriate lamina A1 are larger than those in the appropriate lamina A. Such a pattern was never seen with enucleation alone. Instances of sprouting in cats with enucleation and no additional deprivation by lid suture always took the form of expansion of existing terminal arbors without any intervening gaps (Garraghty et al., '86).

Two contralaterally projecting Y axons terminating at the same parasagittal level are illustrated in Figure 10. The photomicrographs of Figure 10, which illustrate the translaminar invasion of the inappropriate lamina A1 by both Y axons, also serve to demonstrate the grossly normal appearance of individual terminal boutons. As is the case for normal retinogeniculate Y axon arbors, the terminal boutons in Figure 10 are irregular in shape and size, and they are often found *en passant* along axons.

Figures 11–13 present the terminal bouton distributions of the remaining contralaterally projecting retinogeniculate Y axons in our sample. As can be seen from Figures 8–13, *all* of the contralaterally projecting Y axons exhibited some degree of abnormal growth into the previously dener-

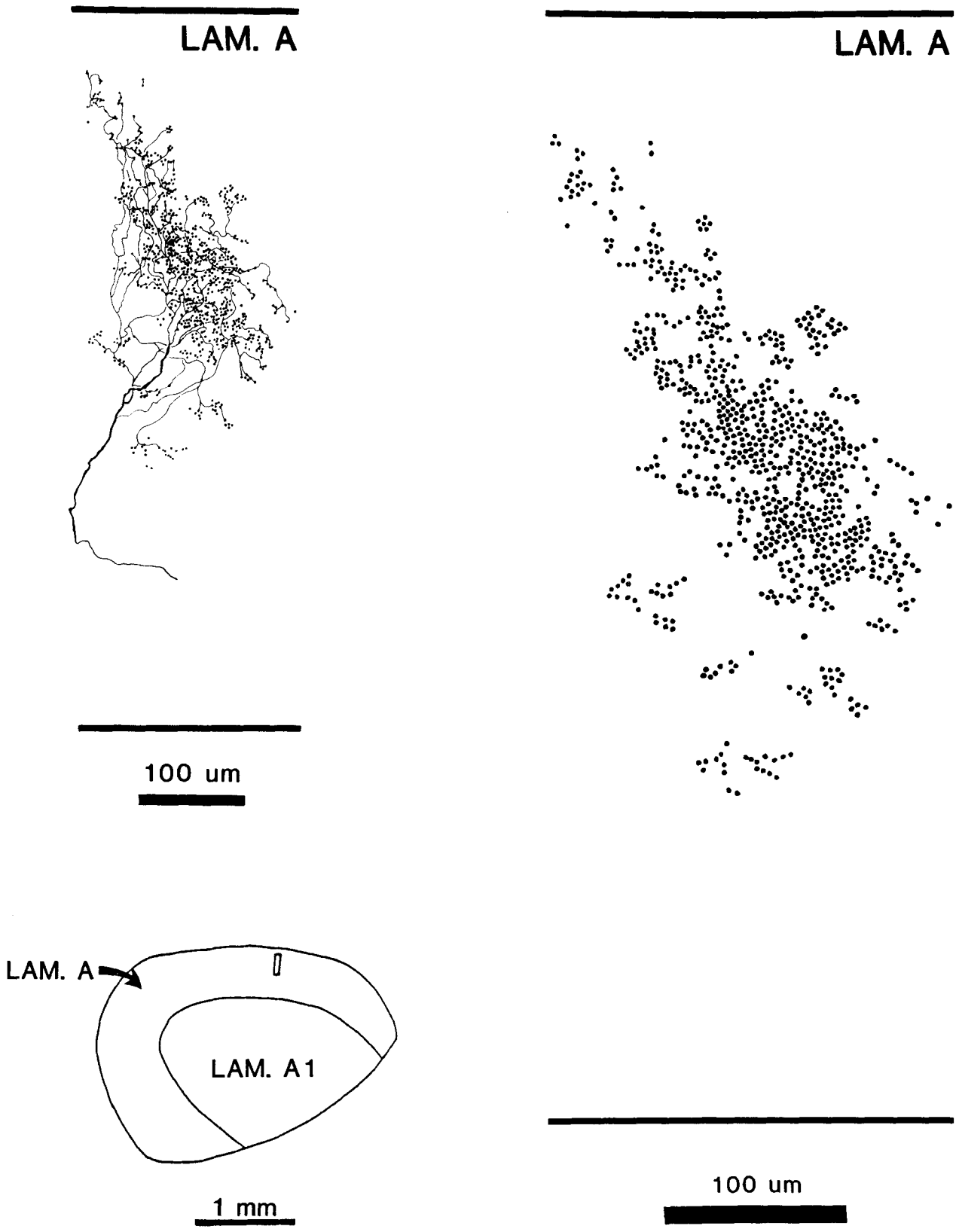


Fig. 2. The reconstruction of a contralaterally projecting X axon from a monocularly enucleated-plus-lid-sutured cat. Conventions as in Figure 1. The terminal arbor of this axon is appropriately restricted to lamina A.

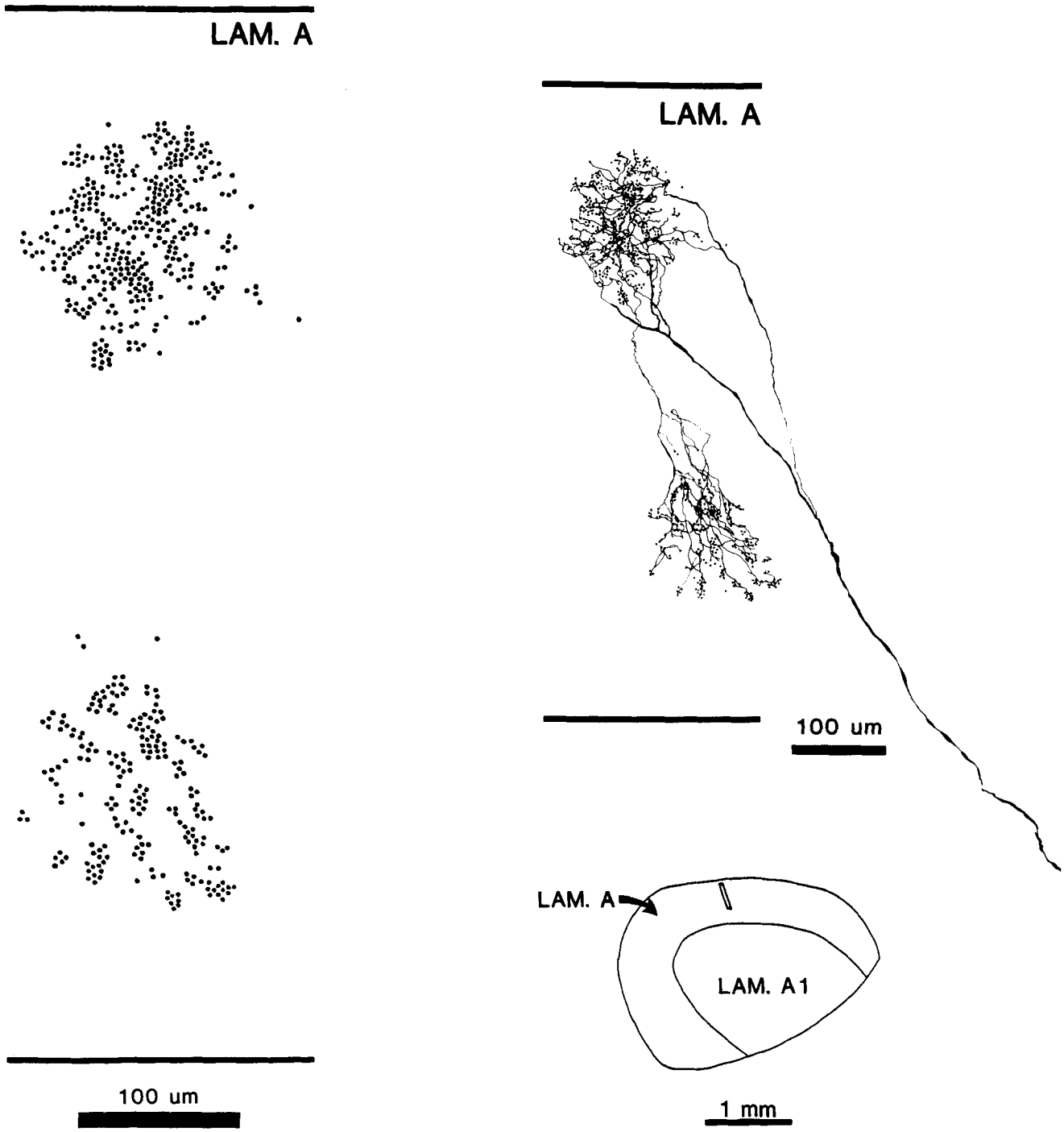


Fig. 3. Another example of a contralaterally projecting X axon reconstructed from the same coronal sections as the axon illustrated in Figure 2. Conventions as in Figure 1. The terminal arbor of this axon is also appropriately restricted to lamina A. This arbor is somewhat unusual in that it is split into two discrete patches, but such patterns of termination have also been seen in X axons from normal cats.

vated lamina A1, and this growth followed the retinotopically correct lines of projection (see Sanderson, '71). This abnormal growth ranged from quite substantial (e.g., Fig. 11A,B) to slight (e.g., Fig. 12B). Of particular interest is the observation that the lamina A terminations of most of the contralaterally projecting Y axons were sparse relative to normal (see Fig. 2 of Sur and Sherman, '82), and frequently smaller than the aberrant terminations in the denervated

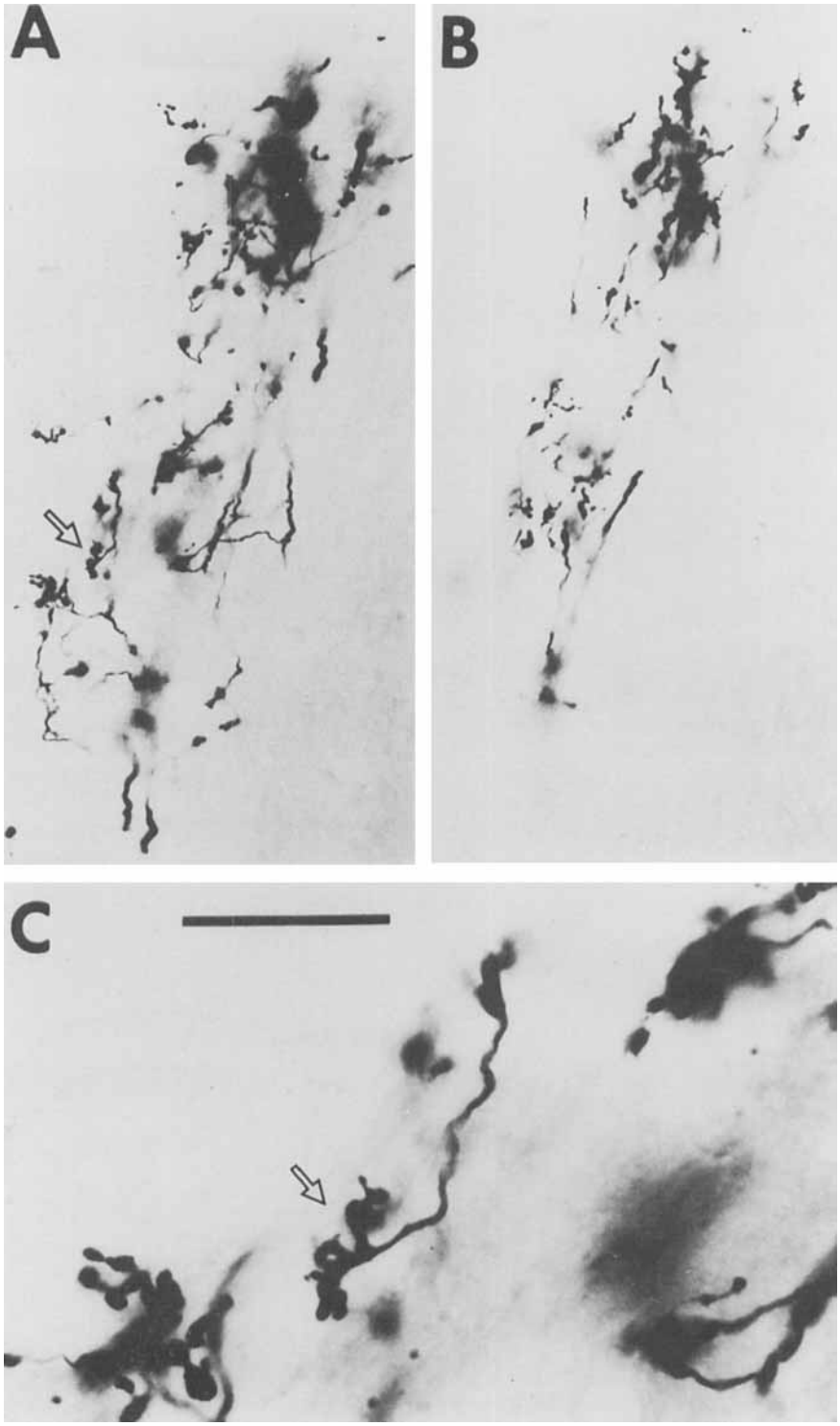


Fig. 4. Photomicrographs of a retinogeniculate X axon innervating lamina A1 from the remaining, ipsilateral retina of a monocularly enucleated-plus-lid-sutured cat. A, B. Lower-power views of the same part of the axon arbor at two different focal planes. The scale represents 100 μm for A and B and 25 μm for C. C. Higher-power view. Arrows in A and C point to the same part of the arbor. Note that the boutons frequently occur in clumps and are relatively consistent in size and shape, characteristic features of normal X axons.

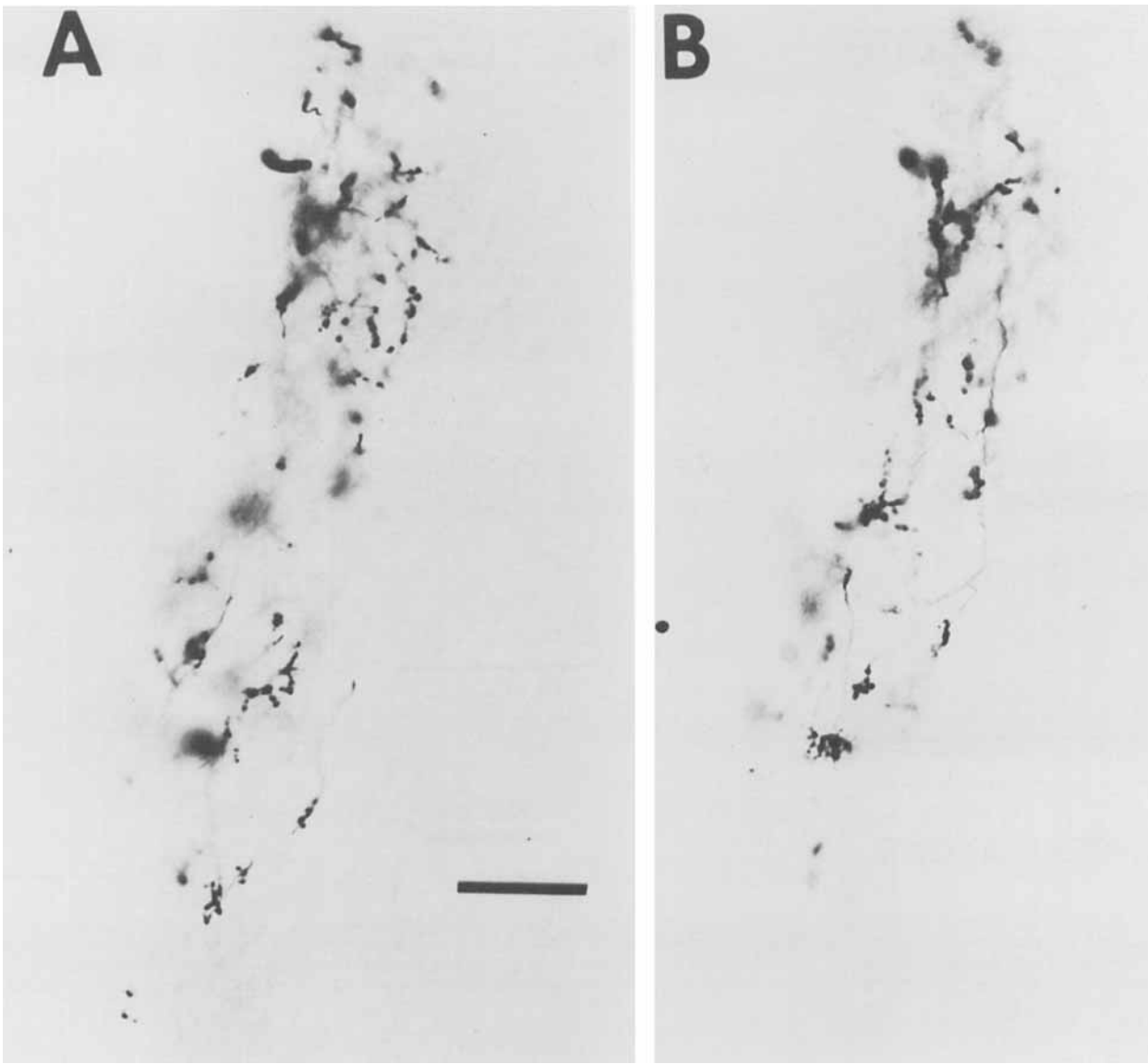


Fig. 5. Photomicrographs of another retinogeniculate X axon innervating lamina A1. A and B are views of the same part of the axon arbor at different focal planes. The scale represents 50 μm. Boutons again occur in clumps and are relatively consistent in size and shape.

lamina A1. This is particularly true for the axons illustrated in Figure 13. Note that in one instance, illustrated in Figure 13A, no terminal arbor at all was found in lamina A (cf. Fig. 2b, Sur et al., '82).

Finally, it should be noted that the separate subregions of aberrant terminal arbor found in lamina A1 for the axons illustrated in Figures 8 and 9 are not the rule. Rather, there appears to be a continuum from discrete subregions to reasonably uninterrupted growth (e.g., Fig. 11A). A separate subregion of terminal arbor for each lamina is thus seen in some axons while in others there is essentially one continuous zone of terminations extending from lamina A through the denervated lamina A1 and into lamina C.

Aberrant growth into lamina A was less evident for the two ipsilaterally projecting Y axons in the monocularly enucleated and lid-sutured cats than was such growth for the contralaterally projecting Y axons. Figure 14 presents the bouton distribution of one of the ipsilaterally projecting Y axons, and the invasion of the arbor into lamina A is quite limited although retinotopically correct. Figure 15 illustrates the second ipsilaterally projecting arbor, which in this case was sectioned parasagittally. Two unusual features were noted. First, there was no translaminar sprouting of this axon into a retinotopically matched portion of lamina A or anywhere else in the binocular segment of lamina A. This represents our only example of a retinogen-

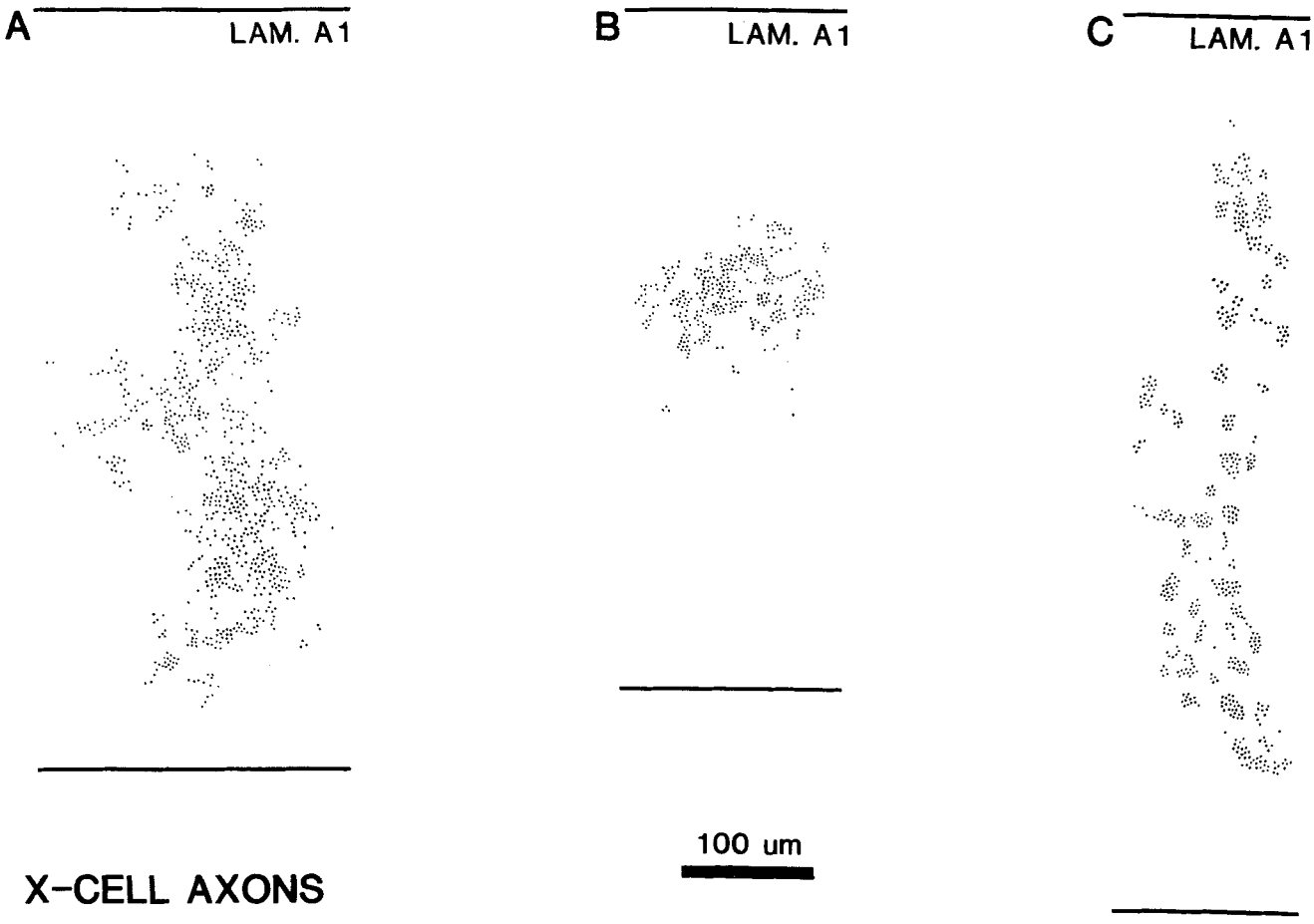


Fig. 6. Summary of terminal boutons from retinogeniculate X axons in monocularly enucleated-plus-lid-sutured cats. These represent ispilaterally projecting axons that innervate lamina A1. As in all figures, the laminar borders are depicted by solid, horizontal lines. Each dot represents a single bouton in the same fashion as the drawing on the left in Figure 1. Letters A, B,and C in the upper left of each illustrated arbor identify these axons with respect to their physiological characteristics presented in Table 1. Note that all of these terminal arbors are completely confined to lamina A1.

iculate Y axon from these enucleated and lid-sutured cats that has failed to develop an aberrant translaminar input to a retinotopically correct region. Second, however, the axon did form a small, aberrant translaminar sprout into the denervated monocular segment some 400 μm lateral to the arbor in lamina A1 (see inset to Fig. 15).

Quantitative observations

While it is evident from the above qualitative descriptions that rather dramatic abnormalities develop for the retinogeniculate Y axon arbors of monocularly enucleated and lid-sutured cats, there also seemed to be abnormalities in the sizes of terminal arbors and density or number of terminal boutons involving both X and Y axons. To assess this possibility, we measured the terminal arbor volumes and counted the number of terminal boutons for the labeled axons in our sample. For comparison, we also include previously reported data from retinogeniculate axons found in cats raised with monocular enucleation without additional deprivation by lid suture (see Fig. 13 of the previous paper, Garraghty et al., '86). The scatter diagrams of Figure 16 summarize this analysis. Closed symbols represent data from cats raised with monocular enucleation and lid suture (MLS+E): open symbols represent data from cats raised with monocular enucleation alone (ME; redrawn from Fig. 13 of the previous paper, Garraghty et al., '86). Each point represents a single axon.

For the X axons (Fig. 16A), no obvious difference was seen in terms of terminal bouton numbers between cats with monocular enucleation alone and cats with monocular enucleation plus lid suture (the respective means are 803 and 741; $P > .1$). However, the terminal arbor volumes of these axons were significantly larger for the former than for the latter cats (the respective means are 0.00614 mm^3 and 0.00303 mm^3; $P < .05$). Consequently, the density of boutons was much higher for the cats with monocular enucleation plus lid suture than for cats with monocular enucleation alone (the respective means are 257,000/mm^3 and 127,000/mm^3; $P < .001$).

A different pattern is evident for the Y axons (Fig. 16B). The average number of terminal boutons for these axon arbors was much lower in the enucleated and lid-sutured

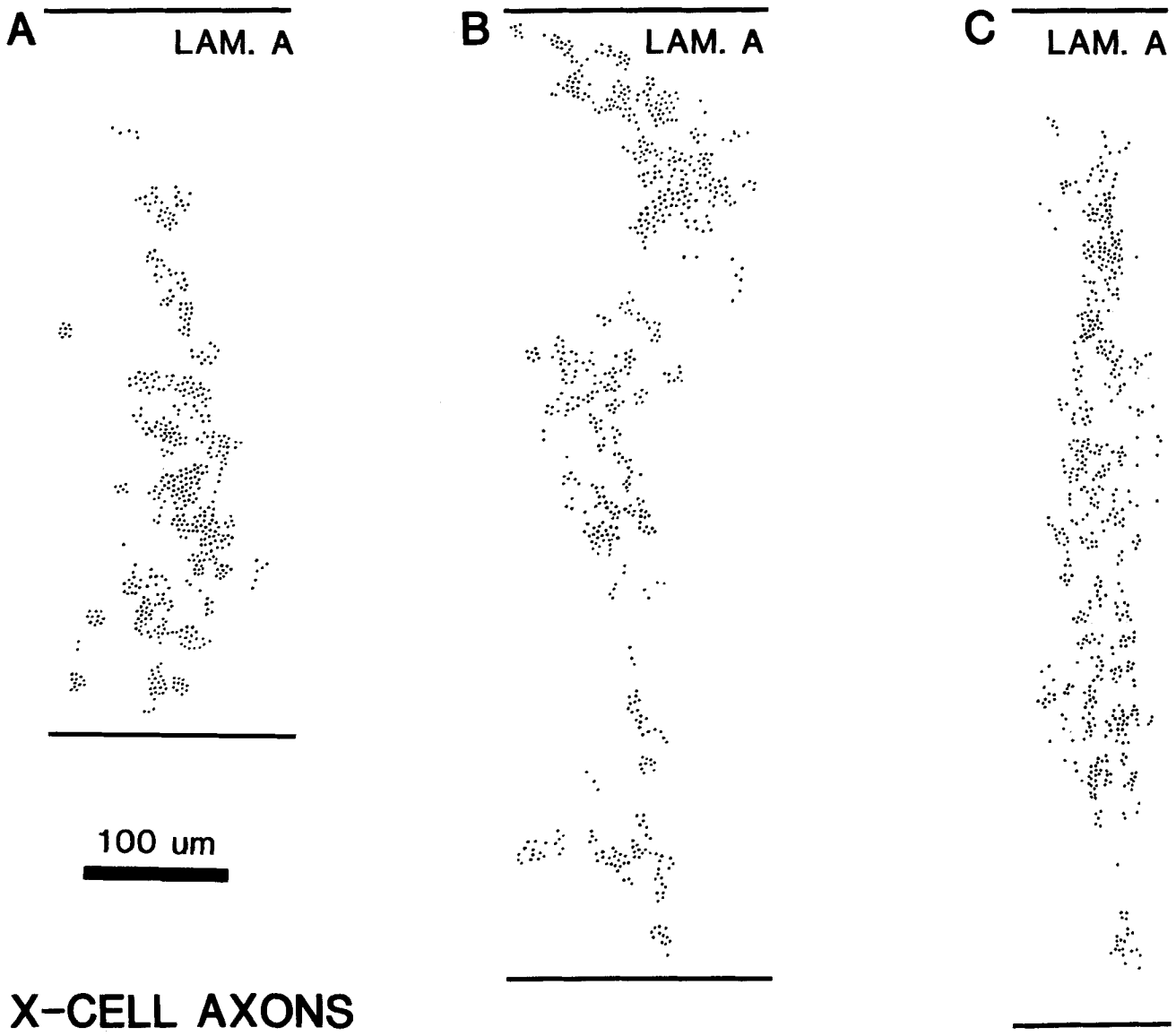


Fig. 7. Summary of terminal boutons from retinogeniculate X axons in monocularly enucleated-plus-lid-sutured cats; conventions as in Figure 6. These are contralaterally projecting axons that terminate in lamina A. The arbors of all of these axons are appropriately confined to lamina A.

cats than in the cats with enucleation alone (the respective means are 882 and 1258; $P<.01$). Similarly, the average terminal arbor volumes of these axons were smaller in the former than in the latter cats (the respective means are 0.0116 mm^3 and 0.0194 mm^3; $P<.02$). As a result, no significant difference was noted between these groups of experimental cats in the density of terminal boutons derived from retinogeniculate Y axons (the respective means are 86,000/mm^3 and 73,000/mm^3; $P>.1$).

DISCUSSION

It was previously demonstrated that rearing cats with one eye removed results in abnormal translaminar growth of retinogeniculate axons (Guillery, '72; Hickey, '75; Robson et al., '78; Robson, '81). Our major contribution to this issue has been the demonstration that this abnormal growth is restricted to the Y class of retinogeniculate axons (see the previous paper, Garraghty et al., '86). Since visual deprivation by lid suture affects the development of retinogeniculate Y axons (Sur et al., '82), the present experiment was designed to assess the effects of a combination of lid suture and enucleation on the development of physiologically identified retinogeniculate afferents. Our major findings are twofold. First, closing the intact eye of monocularly enucleated cats neither alters the morphology of X axons further nor prevents Y axons from sprouting into adjacent dener-

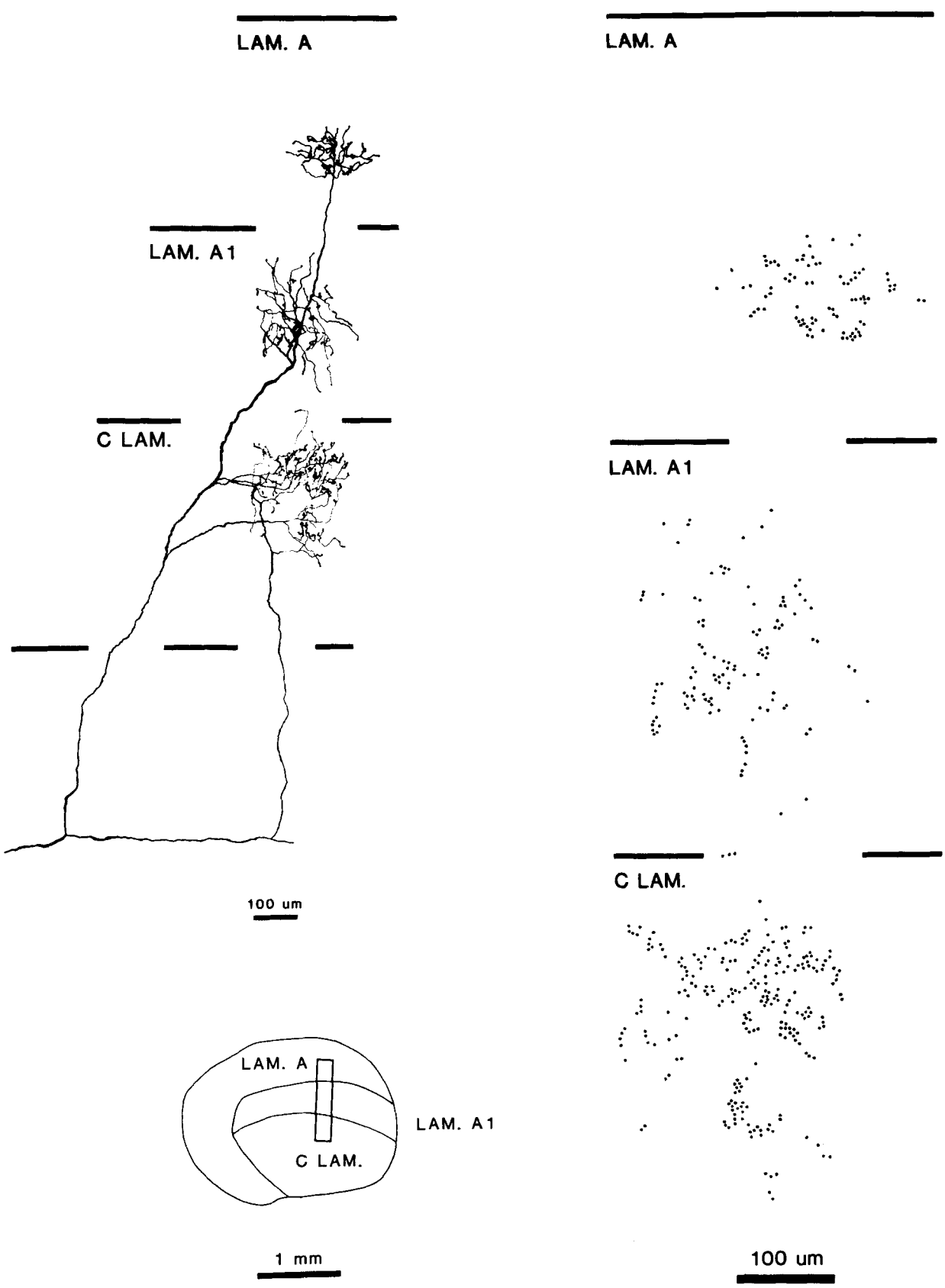


Fig. 8. Example of an HRP-labeled retinogeniculate Y axon from a monocularly enucleated-plus-lid-sutured cat; conventions as in Figure 1. This axon projected contralaterally. Unlike normal contralaterally projecting Y axons, this axon has terminal boutons in lamina A1, which is normally the target of ipsilaterally projecting axons. The lamina A termination of this axon is also very small.

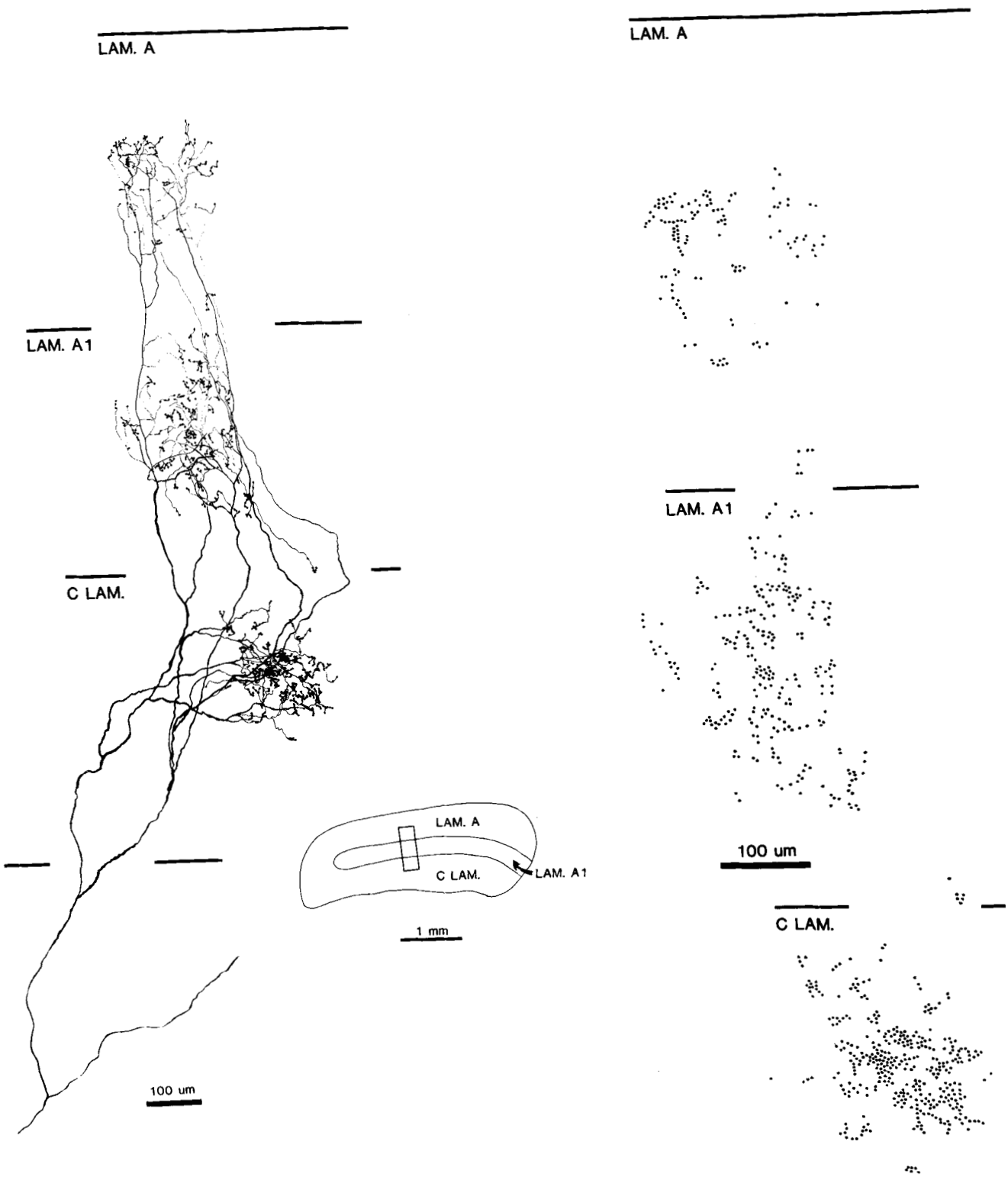


Fig. 9. Another contralaterally projecting Y axon from a monocularly enucleated-plus-lid-sutured cat; conventions as in Figure 1. As with the axon illustrated in Figure 8, this axon also had three discrete patches of termination in lamina A, lamina A1, and lamina C. Again the "normal" lamina A termination is small, and in this case, noticeably smaller than the aberrant termination in lamina A1.

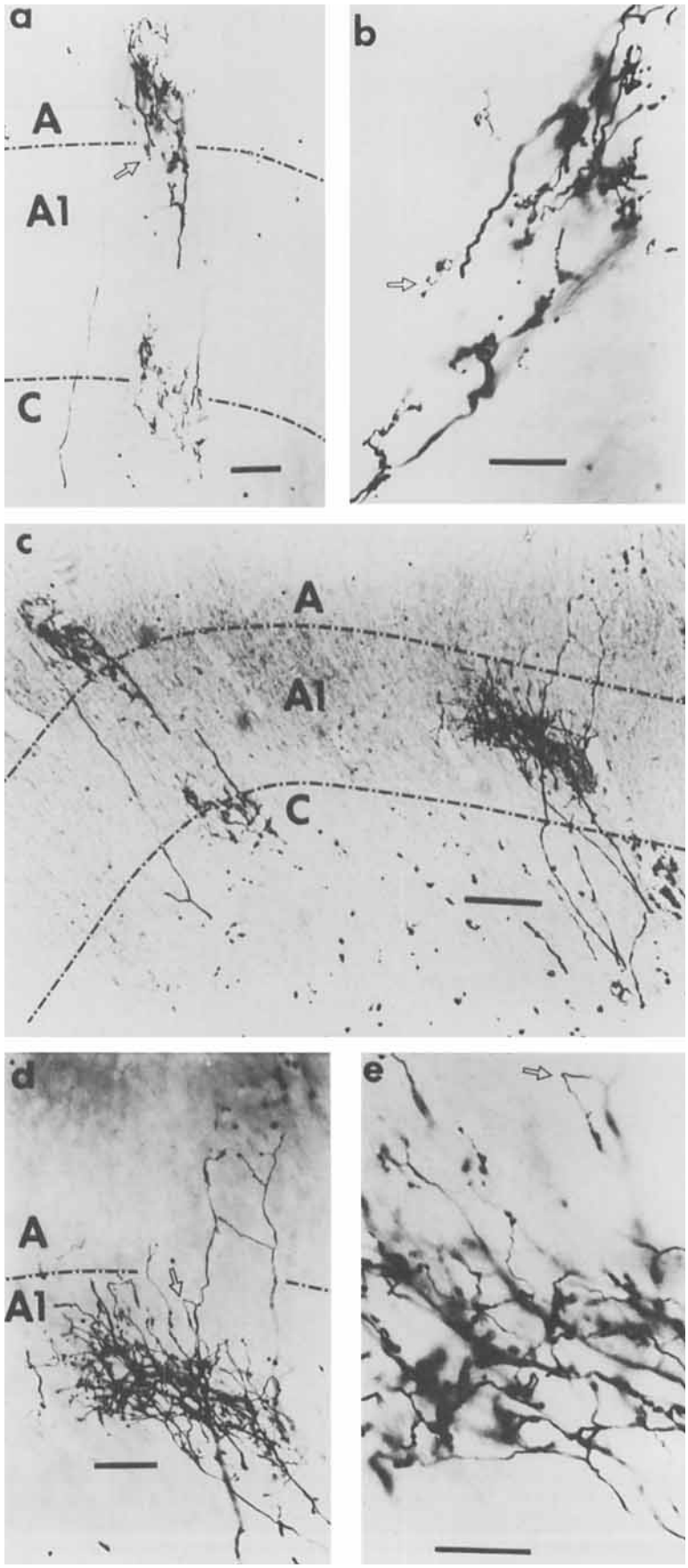


Figure 10

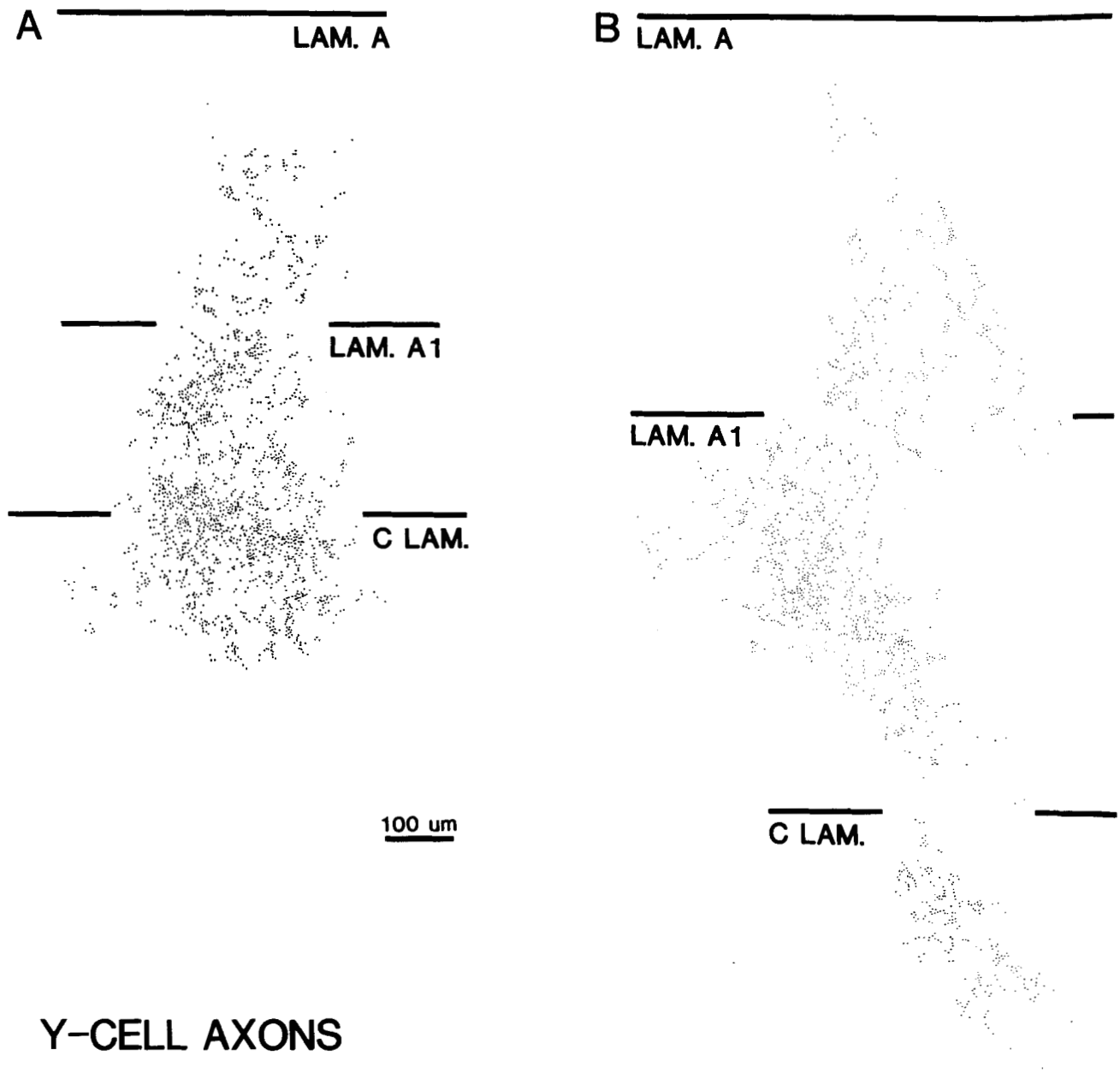


Fig. 11. Summary of terminal boutons of other contralaterally projecting Y axons from monocularly enucleated-plus-lid-sutured cats; conventions as in Figure 6. These two examples sprout extensively into the denervated lamina A1. Unlike the Y axons illustrated in Figures 8 and 9, these arbors are not characterized by discrete clumps of terminations in the three geniculate laminae, but are, rather, relatively continuous.

Fig. 10. Photomicrographs of two retinogeniculate Y axons from a monocularly enucleated-plus-lid-sutured cat. These axons projected contralaterally from the remaining eye. c. The relative positions of these two axons in the parasagittally sectioned lateral geniculate nucleus. Rostral is to the left, dorsal is up. A, A1, and C label the geniculate laminae. In this case lamina A1 was denervated by the early enucleation. Sprouting into lamina A1 is noticeable, particularly in the axon on the right. The scale bar represents 200 μm. a, b. Two views of the more rostral (i.e., left-most in c) of these axons at different powers (scales, 100 μm in a and 50 μm in b). Arrows in a and b point to the same part of the arbor, in this instance an aberrant termination into the denervated lamina A1. The boutons are irregular in size and shape, as is characteristic of normal Y axons. d, e. Two views of the more caudal (i.e., right-most in c) of these Y axons at different powers (scales, 100 μm in d and 50 μm in e). As is evident, this axon sprouted heavily into the denervated lamina A1. Again, the boutons themselves appear normal.

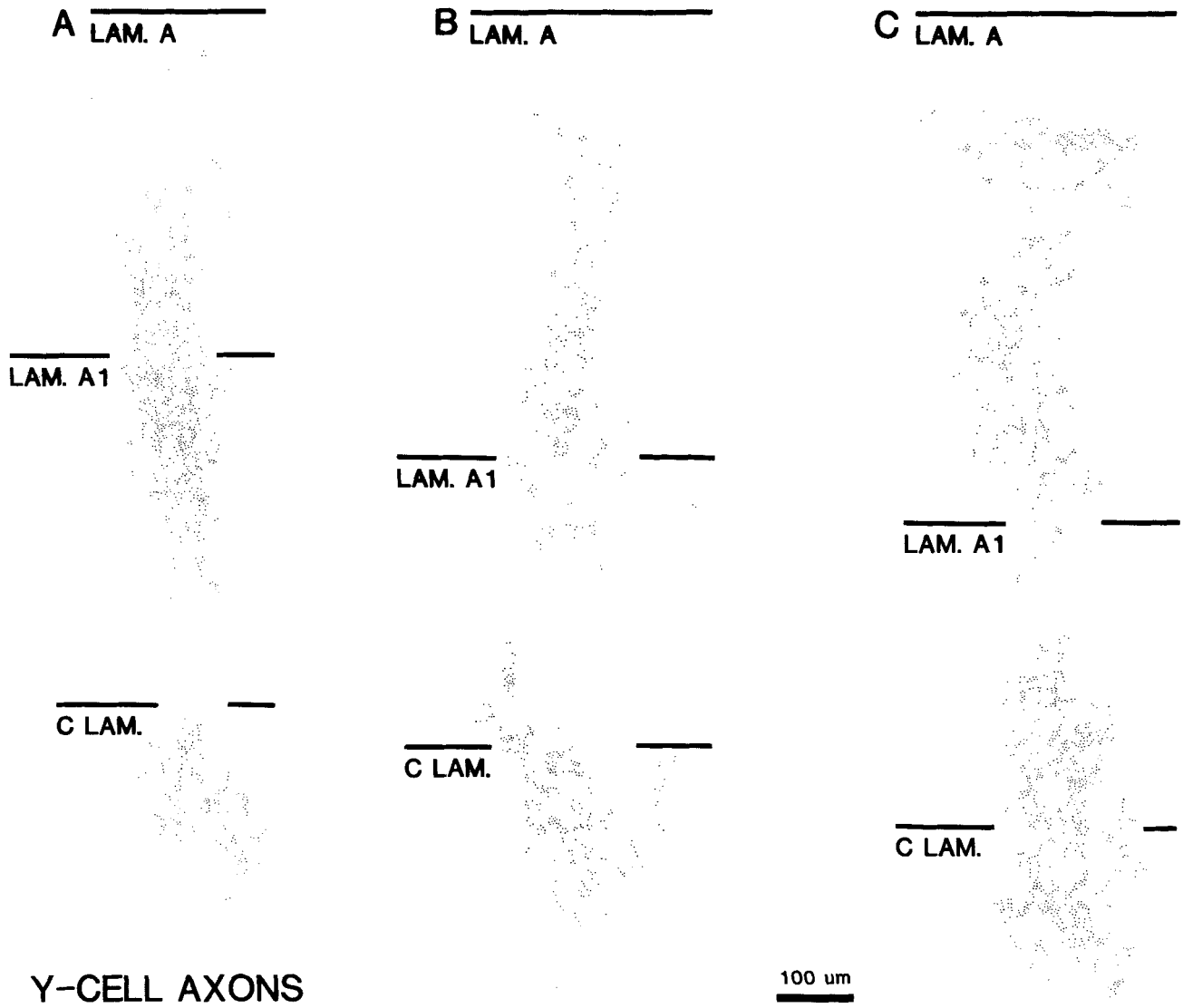


Fig. 12. Summary of terminal boutons of three additional contralaterally projecting Y axons from monocularly enucleated-plus-lid-sutured cats; conventions as in Figure 6. In each instance, aberrant terminations are evident in the previously denervated lamina A1. While these arbors are not continuous like the example presented in Figure 11, they are also not discrete in the same way as the axons illustrated in Figures 8 and 9. In these axons, the aberrant growth can be characterized as extensions from a normal target lamina.

vated laminae. Second, the lid suture does cause a reduction in the extent of Y axon arbors in lamina A or A1 innervated by the remaining eye, although no such reduction is seen for Y axons projecting contralaterally to lamina C. The reduction thus occurs where synaptic terminals from X axons are present in large numbers.

Retinogeniculate axon arbors following enucleation paired with lid suture

After both enucleation and enucleation paired with lid suture, retinogeniculate X axons are restricted to their appropriate target lamina. Several possible explanations, three of which are considered in some detail below, can be offered singly or in combination for this failure of X axons to sprout. First, perhaps the development of translaminar sprouts represents a competitive process during which the developing Y axons have such an advantage that X axons are effectively barred from the previously denervated laminae. Second, perhaps, the enucleation performed during the first postnatal day is already too late for the development of sprouts from X axons. Third, perhaps the X axons never had the potential to develop stable translaminar sprouts. These possibilities are considered in more detail below.

The possibility of X axons being competitively disadvantaged. Prior studies of the development of retinogeniculate axon arbors in normal and monocularly sutured cats indicate that the final sizes of the terminal arbors result from the extent to which the later-expanding Y arbors are able to reduce the exuberant arbors formed earlier by X

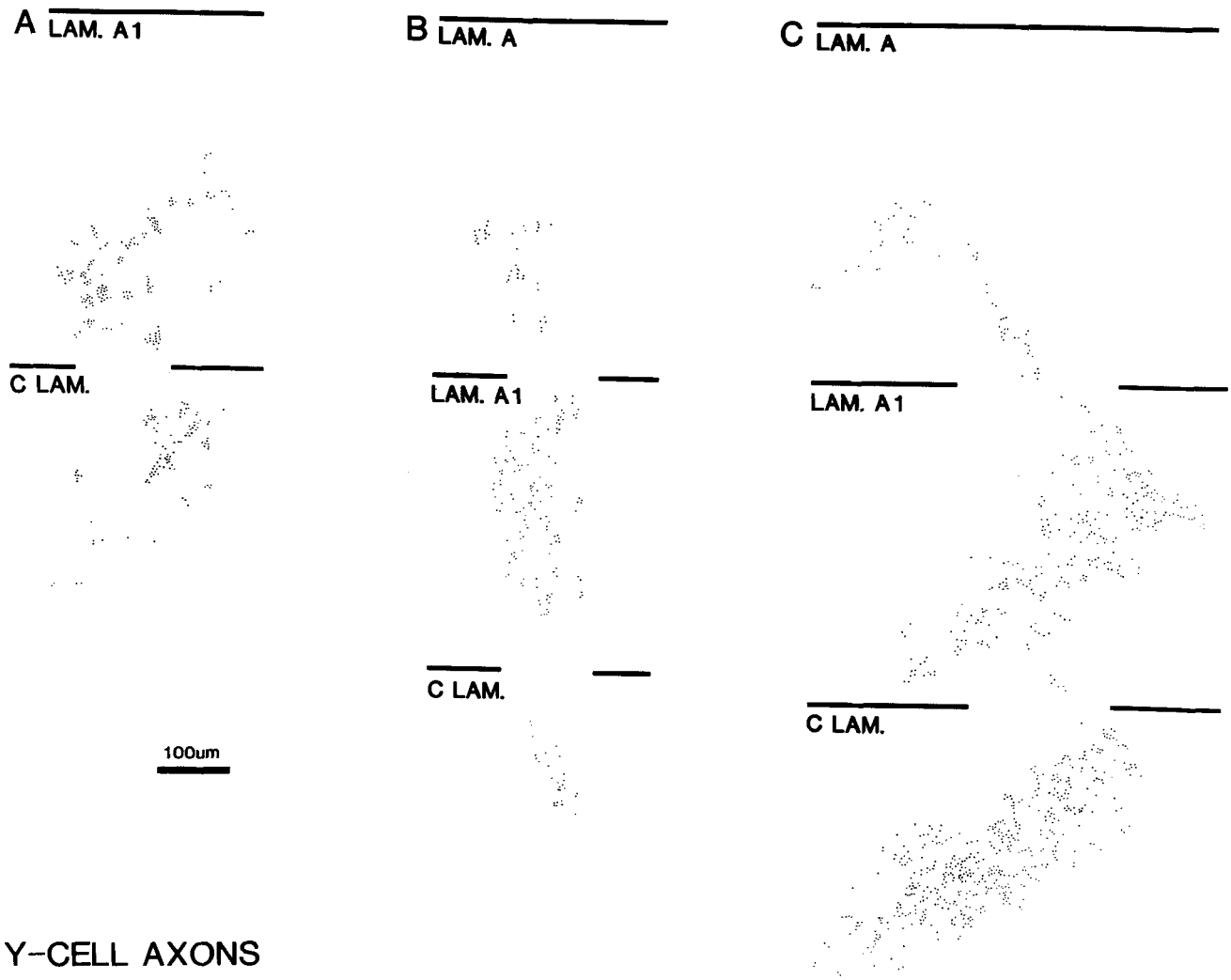


Fig. 13. Summary of terminal boutons of the remaining contralaterally projecting Y axons from monocularly enucleated-plus-lid-sutured cats; conventions as in Figure 6. These particular axons were much more obviously affected by deprivation than those illustrated in Figures 11 and 12 insofar as their terminations in lamina A were very sparse (B, C) or absent (A) even though that was their normal target. The aberrant lamina A1 terminations are in each case larger than the small or absent termination in lamina A.

axons (Sur and Sherman, '82; Sur et al., '82, '84). Development in a normal visual environment (i.e., no lid suture) maximizes the competitive advantage of the Y axons, whereas lid suture dramatically reduces this advantage. If a similar process of competition between the developing X and Y axons were involved in the establishment of translaminar sprouts following monocular enucleation, one might argue that the limitation of these sprouts to Y axons reflects the competitive advantage of these axons as long as the remaining eye is nondeprived.

Thus our present experiment, which involved depriving the remaining eye by lid suture, should reduce this advantage of the Y axons sufficiently to permit some translaminar sprouting of X axons. The observation that, in the enucleated-plus-lid-sutured cats, many Y axons developed arbors in their normal target laminae that were considerably smaller than those developed in cats raised with enucleation alone (see the previous paper, Garraghty et al., '86), suggests that a shift in competitive advantage was actually produced. Nonetheless, these same Y axons in the enucleated-plus-lid-sutured cats developed extensive translaminar sprouts (see Fig. 8–13). On the other hand, this clear shift in favor of retinogeniculate X axons in their competitive development with Y axons did not produce translaminar sprouts among these axons. Although it is possible that X axons need a greater advantage than that provided by lid suture alone to establish translaminar sprouts, this strongly suggests that the failure of these axons to form such sprouts is not a simple matter of their being competitively disadvantaged.

The possibility of a "critical period" for forming sprouts. The establishment of any detectable translami-

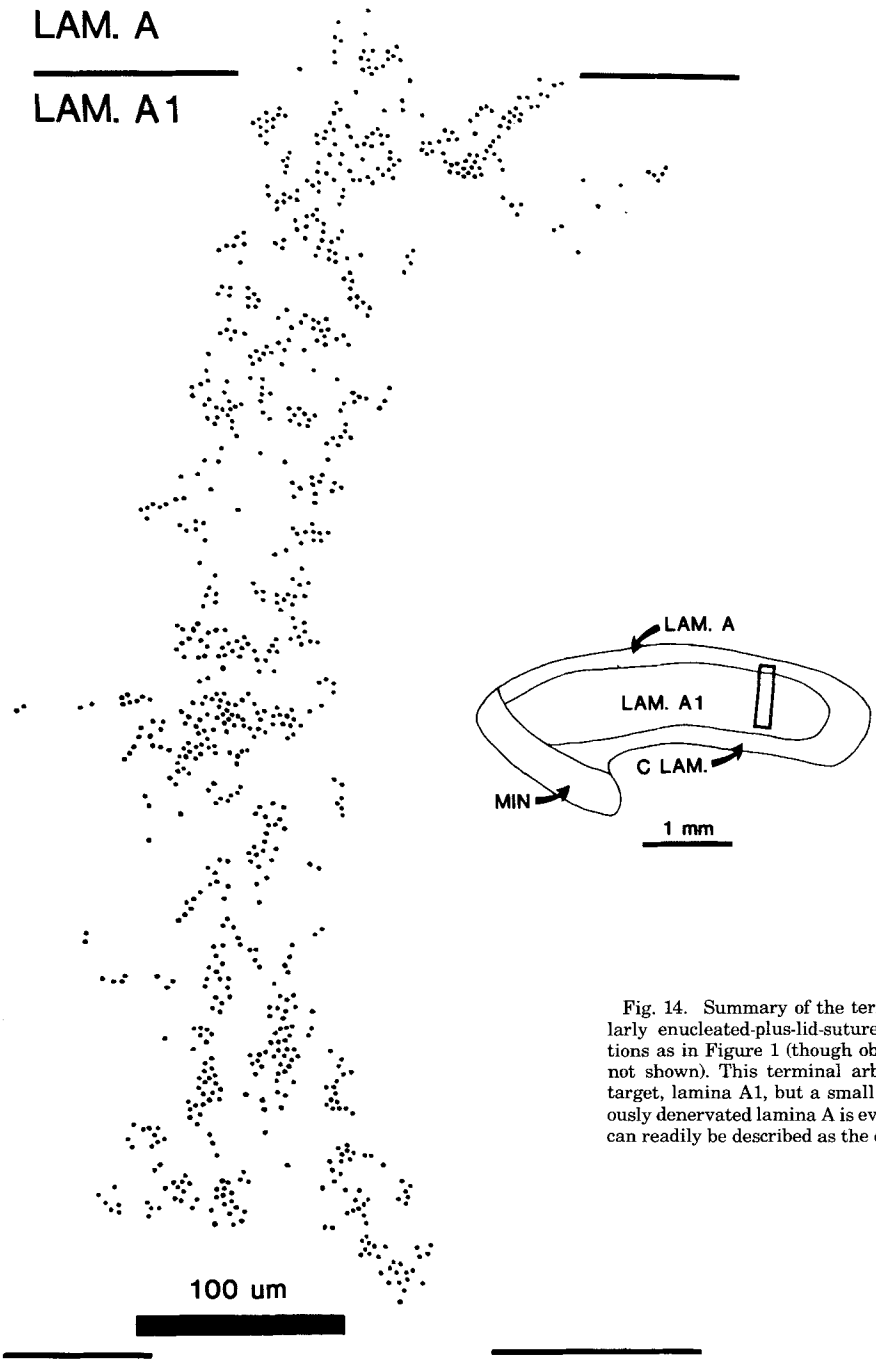
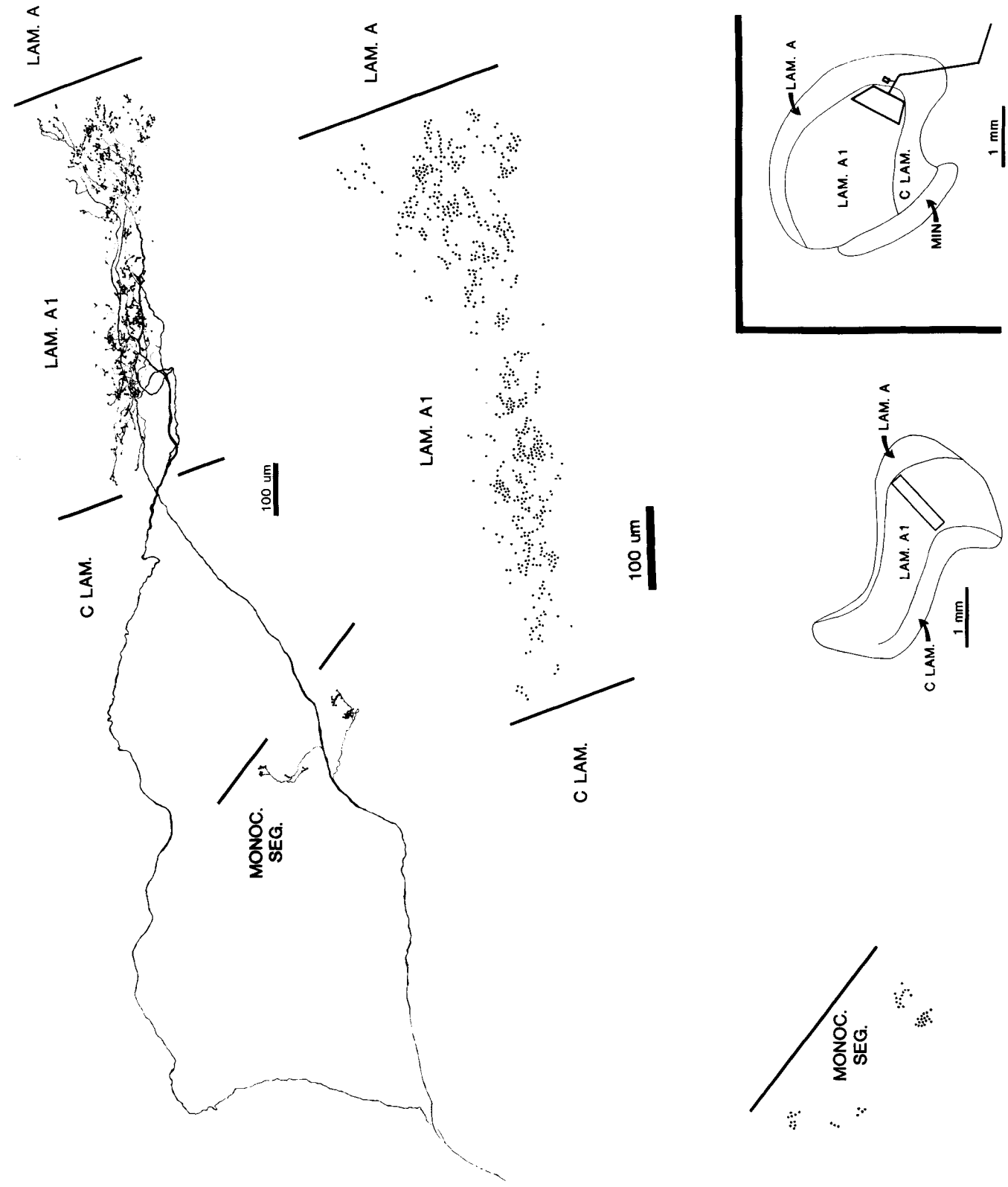


Fig. 14. Summary of the terminal boutons of a Y axon from a monocularly enucleated-plus-lid-sutured cat that projected ipsilaterally; conventions as in Figure 1 (though obviously a line drawing of the axon itself is not shown). This terminal arbor is largely restricted to its appropriate target, lamina A1, but a small amount of aberrant growth into the previously denervated lamina A is evident. The abnormal growth in this instance can readily be described as the expansion of the existing arbor.

Fig. 15. The remaining ipsilaterally projecting Y axon from a monocularly enucleated-plus-lid-sutured cat; conventions as in Figure 1. Note that the brain was sectioned parasagittally. In this case, no abnormal growth was evident in the binocular segment of the lateral geniculate nucleus. A small aberrant termination was present, however, in the denervated monocular segment some 400 μm lateral to the main termination in lamina A1. The inset in the lower right-hand part of the figure is a depiction of the location of this terminal arbor superimposed upon a coronal view of the lateral geniculate. To accomplish this, the dorsoventral extent of the arbor in each sagittal section was measured. These measurements were then superimposed upon the coronal view. The shape of the coronal view was inferred from Sanderson's ('71) coronal maps given the coordinates of this axon's receptive field.


LAM. A
LAM. A1
C LAM.
MONOC. SEG.
100 um
LAM. A
LAM. A1
C LAM.
100 um
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C LAM.
MIN
1 mm
LAM. A
LAM. A1
C LAM.
1 mm
MONOC. SEG.

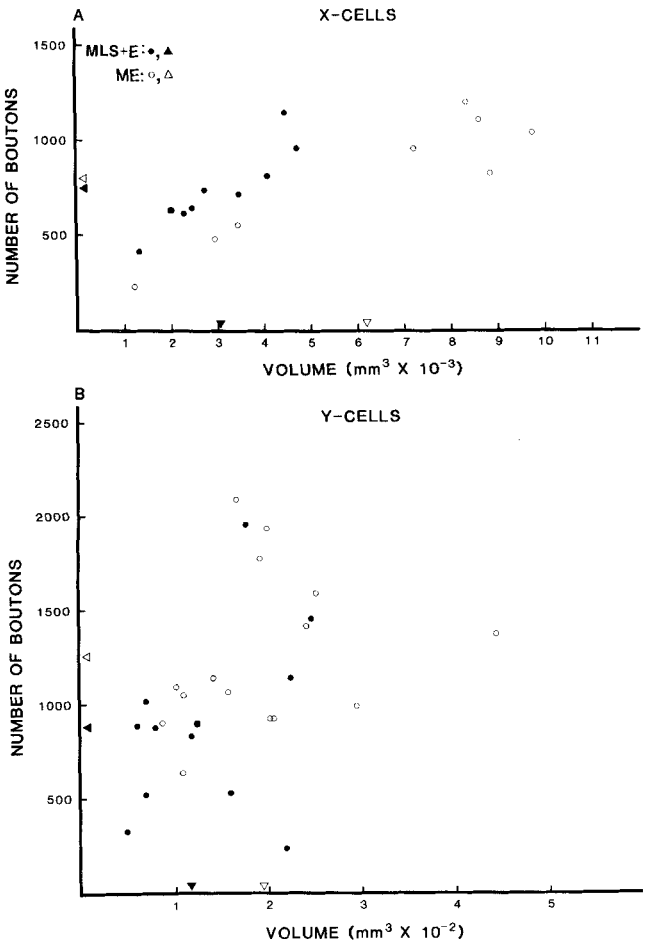

Fig. 16. Scatterplots representing the X and Y axons recovered from monocularly enucleated-plus-lid-sutured cats (MLS+E) and monocularly enucleated visually experienced cats (ME, Garraghty et al., '86). Closed symbols represent data from MLS+E cats; open symbols represent data from ME cats. Symbols on the ordinates and abscissae represent mean numbers of boutons and terminal field volumes, respectively. Note the difference in the scales of the abscissae. A. X axons in MLS+E and ME cats have comparable numbers of boutons, but the volumes of the terminal fields in the MLS+E cats are much smaller. B. Y axons in the MLS+E cats have both fewer boutons and smaller terminal field volumes than Y-cell axons in ME cats (cf. Fig. 13, Garraghty et al., '86).

nar sprouts requires that the monocular enucleation in a kitten be performed within the first 2 postnatal weeks (Guillery, '72; Hickey, '75). The retinogeniculate Y axons thus pass through a developmental stage that is essentially analogous to the "critical period" defined in terms of the postnatal period during which the visual system is susceptible to the effects of deprivation by lid suture (reviewed in Movshon and Van Sluyters, '81; Sherman and Spear, '82). Perhaps the X axons simply have an earlier period of susceptibility for the induction of translaminar sprouts.

This suggestion is bolstered by considerable evidence that retinogeniculate X axons begin their development and maturation before Y axons do. This applies to the birth dates of their parent cells in the retina (e.g., Walsh et al., '83), to the arrival of their axons in the optic tract (e.g., Torrealba et al., '82), and to the development of terminal arbors in the lateral geniculate nucleus (Sur et al., '84; Friedlander et al., '85). The arbors of X axons are certainly not rigidly set by birth, because they can be considerably pruned during the second and third postnatal months (Sur et al., '84). They may, however, have lost the ability to form translaminar sprouts by this time. If so, earlier, prenatal enucleations could demonstrate sprouting among X axons. Preliminary results suggest that this is not the case when enucleation is performed at embryonic day 44. At this stage of development, retinogeniculate axons from the two eyes still have extensively overlapped terminal zones in the lateral geniculate nucleus (Shatz, '83); enucleation does not induce the development of abnormal morphology in the X axons but seems to do so in Y axons (Stretavan et al., '85). Therefore, either X axons do not possess the capacity to form stable translaminar sprouts or even earlier enucleation is required.

The possibility of fundamental developmental differences between X and Y axons. Perhaps retinogeniculate X axons are developmentally constrained in a way that Y axons are not to form stable terminal arbors limited to their appropriate geniculate laminae. For instance, afferents from X axons might play some special role in the process of afferent segregation characteristic of normal development (e.g., Shatz, '83). Y axons apparently fail to recognize this afferent geometry when other avenues for abnormal growth are provided by denervating adjacent territory or by blockade of normal retinal impulse activity with intraocular injections of tetrodotoxin (Sur et al., '85). The growth and sprouting of Y axons, therefore, may result from a combination of following the laminar path laid down by X axons plus a tendency to grow along paths of least resistance (i.e., ones not requiring the competitive displacement of X axons). Since Y axons observe the integrity of laminar boundaries in normally reared cats, it seems likely that abnormal sprouting reflects the absence of interlaminar competition between afferents from the two eyes. That is, perhaps during normal development, retinogeniculate Y axons do not sprout abnormally because those synaptic sites in inappropriate laminae to which they grow after enucleation or enucleation-plus-lid-suture are already occupied by afferents from the appropriate eye that enjoy a competitive advantage at those sites.

Even the normal process of interocular segregation of Y axon arbors may have little or nothing to do with the segregation of X axons arbors. Whereas X axon arbors obey rules of laminar fidelity even if one eye is removed, the arbors of Y axons do not. Rather, it appears that the normal process of laminar segregation of Y axon arbors depends upon competitive interactions between axons from the two eyes (see, however, Shatz and Sretavan, '86; Sretavan and Shatz, '86). If this last suggestion represents the explanation for the dramatically different developmental response of retinogeniculate X and Y axons to removal of one eye, it implies a fundamental difference between these axon classes that goes beyond different rates of development and different levels of success in competitive interactions.

Determination of laminar boundaries

For two reasons, it seems noteworthy that geniculate laminar boundaries were easily determined in cats raised with enucleation paired with lid suture, but that these boundaries were often obscure in cats reared with enucleation alone.

First, we were concerned in our prior study of enucleation alone that we might have mislocated laminar borders in our reconstructions of axonal arbors (see the previous pa-

per, Garraghty et al., '86). We were fairly conservative to ensure that we did not overestimate the extent of the denervated laminae, so that any perceived translaminar growth of axonal arbors was truly translaminar. Consequently, we may have somewhat underestimated the extent of these denervated laminae and thereby failed to see any slight translaminar growth of X axons that might have been present (see Discussion of the previous paper, Garraghty et al., '86). While this would not change the main conclusion that X and Y axons possess qualitatively different capacities to form translaminar sprouts, the results of the present study suggest that no such error in missing potentially present translaminar terminals of X axons was made. As noted in Results, we had no difficulty in confidently determining laminar boundaries in the cats of the present study, and no evidence of translaminar arbors was seen for X axons. This despite the fact that, as noted in the above section, we would expect the more favorable competitive environment for X axons to produce more translaminar sprouting when lid suture is added to enucleation than when enucleation occurs alone. In other words, failure to detect translaminar sprouting for X axons in the present study renders it most unlikely that we failed to see any such sprouting that might have occurred in our previous study (Garraghty et al., '86). This, in turn, supports our general determination of laminar boundaries in the cats raised with enucleation alone.

Second, it seems curious that the addition of lid suture to enucleation leads to the development of a clearer lamination pattern in the lateral geniculate nucleus than occurs with enucleation alone, as if the deleterious consequences of two abnormal situations, enucleation of one eye and lid suture of the other, tend to balance one another during the development of geniculate lamination. Enucleation alone causes transneuronal atrophy of the denervated geniculate laminae and may cause hypertrophy of neurons in the other laminae, although we did not specifically test for this latter possibility. Monocular lid suture alone is known to cause a failure of cells to grow to normal size in deprived laminae (i.e., those innervated by the closed eye) and hypertrophy of cells in nondeprived laminae (e.g., Hickey et al., '77). That these two processes may offset each other may not seem particularly surprising in view of our notions of the competitive nature of retinogeniculate development. However, the more complete development of lamination seen in the present study indicates that the fiber plexus that develops between geniculate laminae is also more completely formed, and it is not presently at all clear why this should depend on the interaction between enucleation and lid suture.

Bouton numbers of retinogeniculate axons

X axons. Retinogeniculate X axons from the deprived retinas of cats raised with monocular lid suture and from normal kittens at 3–4 weeks of age exhibit an abnormally large number of terminal boutons in geniculate lamina A or A1 (Sur et al., '82, '84). As noted above, this has been interpreted as a failure of the deprived Y axons to prune back the exuberant arbors of the earlier-developing X axons. A similar explanation can be used to explain the abnormally large bouton numbers of retinogeniculate X axons in cats raised with monocular enucleation (Fig 16A). The opportunity offered Y axons to innervate denervated laminae reduces their pressure to prune the X axon arbors.

Since lid suture further reduces the ability of Y axons to prune X axon arbors, one might expect the X arbors to contain the greatest number of boutons in the enucleated-plus-lid-suture cats of the present study. However, there is no difference between cats raised with enucleation alone or enucleation coupled with lid suture in terms of bouton numbers (Fig. 16A). Thus, it seems likely that the number of boutons found in the X axon arbors after these two rearing procedures reflects the maximum number that they can sustain in adulthood. Perhaps by 3–4 weeks of age, these X axons have already formed the largest number of boutons they can maintain, and the only alteration they are capable of undergoing involves a reduction in these arbors via competitive pressure from developing Y axons. This reduction through competitive development can be minimized or avoided by various experimental manipulations, such as enucleation and/or lid suture. We should note that the fact that X axons can retain any number of excess boutons demonstrates that the pruning process that they undergo during normal development is not intrinsically programmed (e.g., Brown et al., '76), but rather depends upon extrinsic factors—most likely, we think, competitive interactions with Y axons.

Finally, although there is no difference in bouton numbers between cats raised with enucleation alone and enucleation paired with lid suture, the former cats exhibit much larger volumes of retinogeniculate X axon arbors than do the latter cats (Fig. 16A). We suggest that this is a fairly trivial consequence of the fact that the previously denervated A-laminae are obviously smaller after enucleation plus lid suture than after enucleation alone (see above).

Y axons. The average number of boutons found in the retinogeniculate arbors of Y axons was somewhat smaller in cats raised with enucleation plus lid suture than either in cats raised with enucleation alone or in normal cats (Fig. 16B; see also Fig. 13 of the previous paper, Garraghty et al., '86). This result occurs despite the obvious translaminar sprouts of the Y axons in enucleated-plus-lid-sutured cats, and it reflects the reduced number of boutons in the laminae still innervated by X axons (i.e., lamina A contralateral to the remaining eye and lamina A1 ipsilateral). This seems to be a fairly straightforward result of the lid suture (cf. Sur et al., '82), that prevents the deprived Y axons from forming substantial arbors where X axons are already established, but does not retard the development of Y axon arbors where the X arbors are not present, such as in lamina C and in the denervated laminae.

Nature of Y axon sprouting

Retinogeniculate Y axons in the monocularly enucleated-plus-lid-sutured cats consistently sprout across laminar borders. Indeed, every Y axon in the present study developed abnormal translaminar sprouts. It is thus evident that normal patterned visual experience is not a prerequisite for the occurrence of sprouting, a conclusion already reached by Hickey et al. ('77). Furthermore, in the monocularly enucleated-plus-lid-sutured cats, we obtained our only unambiguous example of abnormal innervation of the denervated monocular segment (see Fig. 15). Similar sprouting of ipsilaterally projecting axons into the denervated monocular segment has already been documented in monocularly enucleated and visually experienced cats (Hickey, '75; Robson, '81). With this single exception, all of our examples of translaminar growth generally follow lines of projection (Sanderson, '71) and consequently lead to innervation of retinotopically matched zones in all laminae.

Robson ('81) has suggested that translaminar growth along projection lines involves the contiguous expansion of existing terminal arbors while growth into the denervated monocular segment involves the formation of "new" arbors that do not abut the arbor in the normally innervated laminae. We remain unconvinced, however, that the distinction drawn between translaminar and monocular segment growth is valid. In our material from monocularly enucleated-plus-lid-sutured cats, some of the contralaterally projecting Y axons produced three discrete patches of termination in laminae A, A1, and C (see Figs. 8–10), whereas others produced translaminar sprouts that were contiguous with the arbor in normally innervated laminae (see Figs. 11–13). The innervation of lamina A1 in the former arbors cannot be characterized as expansions of the arborizations in lamina A or C, although the translaminar growth represented in Figures 11–13 might be so characterized. Unfortunately, our data cannot distinguish between the possibility that the different types of translaminar sprouting (i.e., separate versus contiguous) represent ends of a continuum of a single process leading to such sprouting, and the possibility that these are two separate processes, as Robson ('81) has suggested. In any case, we can conclude that a process leading to discrete patches of terminal arbor is not limited to the formation of sprouted arbors into retinotopically inappropriate regions, such as the denervated monocular segment.

A hypothesis for X and Y retinogeniculate development

Figure 17 summarizes the data from the present experiment and previous experiments involving normal adult cats (Sur and Sherman, '82; Bowling and Michael, '80, '84; Esguerra et al., '85; Sur et al., '86), developing kittens (Sur et al., '84; Friedlander et al., '85), cats raised with monocular lid suture (Sur et al., '82), and cats raised with monocular enucleation (the previous paper, Garraghty et al., '86). These data serve as a basis for our hypothesis regarding the development of retinogeniculate arbors. We emphasize the speculative nature of the hypothesis and offer it mainly as a useful theoretical framework for our experiments and as a heuristic model to be tested with further experimentation.

As shown in Figure 17, the developmental histories of retinogeniculate X and Y axons differ. X axon arbors display a relative exuberance at 3–4 weeks of age followed by a decline in size so that normal adult morphology is achieved by 12 weeks of age (Sur et al., '84). Y axon arbors, on the other hand, are relatively immature at 3–4 weeks of age (Sur et al., '84; Friedlander et al., '85) and grow monotonically to adult size by 12 weeks of age (Sur et al., '84). Therefore, unlike those of X axons, Y axon arbors grow to their adult size without any obvious process of retraction. Finally, there is no evidence that X axons ever innervate lamina C in substantial numbers (Sur and Sherman, '82; Sur et al., '84; Bowling and Michael, '84), and Y axon arbors from the contralateral eye begin to mature in lamina C before they do in lamina A (Friedlander et al., '85).

Rearing with monocular lid suture affects development of X and Y axons in quite different ways. The arbors of retinogeniculate X axons innervating deprived laminae A or A1 are abnormally large, as if they managed to retain their immature exuberant form (Sur et al., '82, '84). Y axon arbors innervating deprived lamina A or A1 are abnormally small, as if they failed to grow from their neonatal state (Sur et al., '82, '84). However, Y arbors in deprived lamina C develop normally after lid suture (Sur et al., '82). This is a crucial observation, because it indicates that deprived Y arbors cannot develop normally where X arbors are already established (i.e., in lamina A or A1) but can do so where X arbors are never present (i.e., in lamina C; see above). This further suggests that the development of X and Y arbors is controlled, at least in part, by a competitive process. That is, during normal development, X axons arrive at the lateral geniculate nucleus before Y axons do, and the X axons are thus able to form exuberant arbors in lamina A or A1 that innervate more than their normal complement of postsynaptic geniculate neurons. The decline in X bouton numbers then stems from their competitive displacement by the later-maturing Y axons in lamina A or A1 (Sur et al., '82, '84). During lid suture, arbors of retinogeniculate Y axons in lamina C are spared the deleterious effects of deprivation because they are never placed at a competitive disadvantage with respect to previously formed arbors from X axons. Lid suture, however, somehow prevents the later-maturing Y axons from gaining the competitive edge needed to displace portions of the exuberant X arbors, with the result that X arbors in deprived laminae A and A1 remain abnormally large while those of Y axons remain abnormally small.

After monocular enucleation (Garraghty et al., '86), X arbors seem again to retain their immature exuberance. However, this retention is not now at the expense of developing arbors from the Y axons. Rather, many Y axons sprout into adjacent denervated laminae. This pattern is best explained with the conclusion that the X axons retain their exuberance because the Y axons are provided with a less-resistant avenue for growth by the enucleation. That is, the later-maturing Y axons apparently find it easier to expand into the deafferented laminae than to displace X axons within their normal target layer. The X axons are, therefore, again freed from debilitating competitive interactions with the Y axons, and consequently can retain the exuberance normally lost because of this competition.

The experimental paradigm is further complicated, but the underlying developmental mechanisms are perhaps clarified, by the addition of visual deprivation to the enucleation. Again, the X arbors retain their immature exuberance, but after monocular enucleation plus lid suture, unlike enucleation alone, the Y arbors in the normally innervated laminae A and A1 are also affected. The contralaterally projecting Y axons in the present experiment display the same differential pattern of laminar sensitivity to the effects of deprivation as is found for monocular lid suture alone (i.e., without removal of the other eye; see Sur et al., '82). That is, the lamina A arbors of the contralaterally projecting axons are sparse or absent while the arbors within lamina C are unaffected. Furthermore, we find no evidence that the addition of lid suture to enucleation produces smaller abnormal arbors in the previously denervated lamina A1 than would occur with enucleation alone. Since we have never seen an abnormal translaminar sprout from an X axon after either enucleation alone or monocular enucleation plus lid suture, it seems likely that the denervated lamina A1 in the monocularly enucleated and lid-sutured cats is devoid of terminal arbors from X axons. In this sense, the denervated lamina A1 is similar to lamina C, which would imply that competitive interactions be-

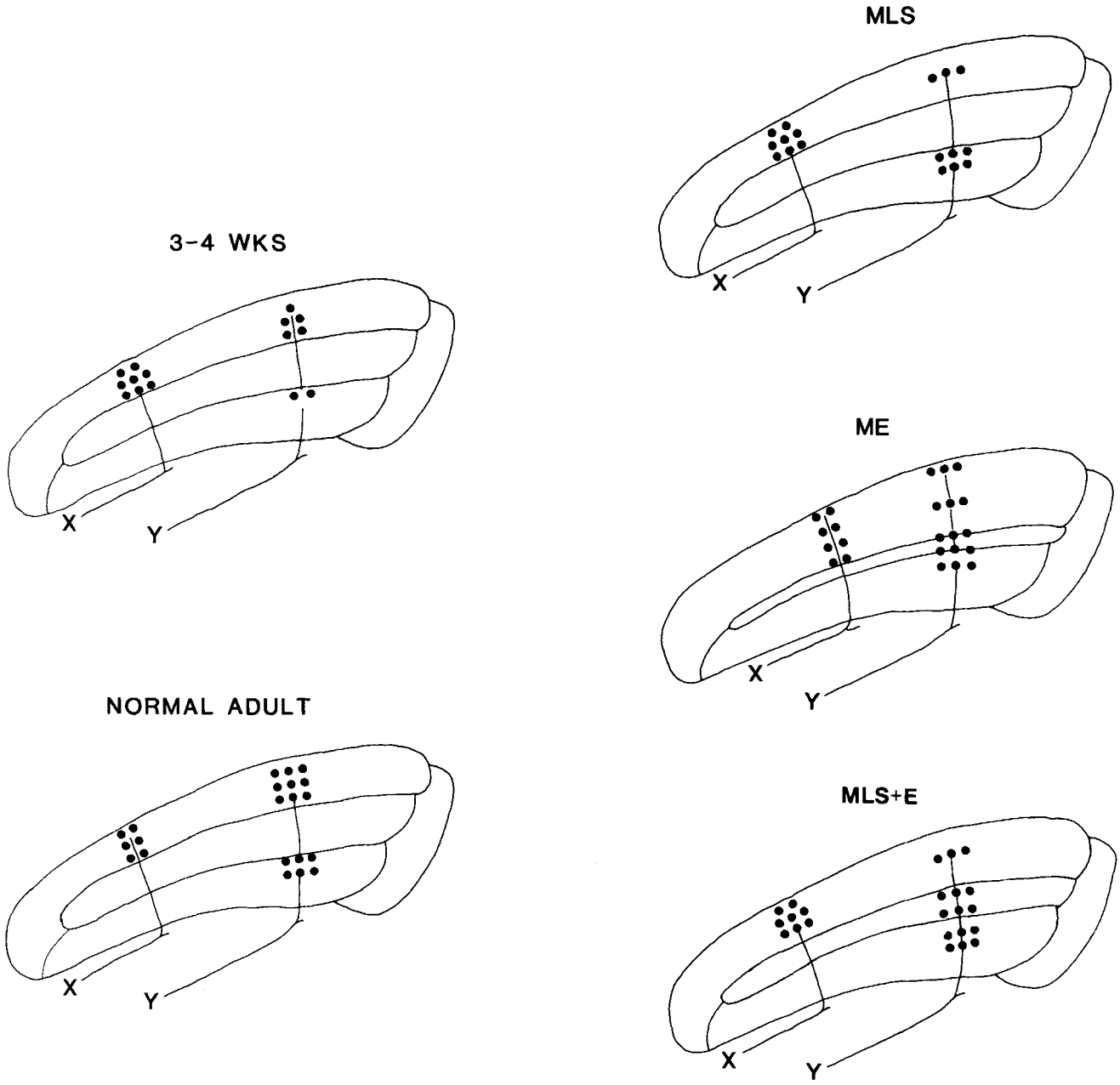

Fig. 17. Schematic representation of X and Y axons in the lateral geniculate nuclei of kittens, adult cats, and cats reared with perturbations. In each instance, the geniculate represented is contralateral to the eye of origin of the axons displayed. The dots represent terminal boutons, and only arbors in laminae A, A1 and C are depicted. In the kitten (3–4 WKS), X axon arbors are exuberant relative to their normal adult form, while Y axons are much smaller than their adult form. X axons achieve their mature form via retraction of the exuberant arbor, whereas Y axons achieve their mature form through continued growth. In cats reared with monocular lid suture (MLS), X axons are presumably placed at a competitive advantage over Y axons and retain their exuberant form while the Y axons fail to develop, but this only happens in laminae where X inputs are present. In lamina C, which is devoid of X afferentation, Y axon arbors develop normally. In cats reared with monocular enucleation (ME), X axons again retain their exuberant form, but in this case not at the expense of Y axons that frequently sprout into the laminae denervated by enucleation. It seems that the Y axons find this to be a less-resistant avenue for growth than the competitive displacement of the already-exuberant X axons in the normal target lamina. X axons are never found to sprout into the denervated lamina. Finally, in cats reared with monocular enucleation paired with lid suture of the remaining eye (MLS+E), X axons again remain exuberant, but no more so than after either enucleation or lid suture alone. This size, therefore, probably reflects the maximum that can be maintained by X axons. Y axons again sprout into the previously denervated laminae while the X axons do not. The terminations of the Y axons in the normally innervated A-laminae are, however, smaller than normal, presumably because of the lid suture.

tween X and Y axons for terminal space would not be possible there. Thus, the aberrant lamina A1 terminations of Y axons can develop freely, albeit abnormally, in the absence of patterned visual input because X axons are not present.

Conclusions

After rearing cats with monocular enucleation alone or paired with lid suture of the remaining eye, retinogeniculate X axons exhibited no translaminar sprouting whereas nearly every Y axon formed translaminar sprouts. This leads to two obvious conclusions. First, normal visual experience is not required for X axon arbors to develop or retain projections restricted to lamina A or A1 when nearby territory is denervated. Second, normal visual experience is equally unnecessary for Y axons to develop translaminar sprouts.

Even after the addition of monocular lid suture to enucleation of the other eye, which shifts the competitive advantage to favor growth of X axon arbors over those of Y axons within the appropriately innervated laminae, we still failed to detect evidence of translaminar sprouting by X axon arbors. We tentatively conclude that the different pattern of X and Y axon arbors seen in experimental cats reflects a qualitative difference in the development of these axon arbors: the earlier-developing X axons are constrained to innervate only a predetermined lamina (A or A1), perhaps to set up laminar boundaries within the lateral geniculate nucleus.

In addition to indirect evidence for such qualitative differences between retinogeniculate X and Y axons with respect to their developmental mechanisms, our results suggest competitive interactions between X and Y axons in the development of their terminal arbors. The evidence for such competition has already been discussed. During such competition, the X arbors are constrained, as noted above, to their appropriate lamina A or A1, whereas the Y arbors are not so constrained. The X arbors remain large, as in their immature exuberant condition, whenever Y axons fail to prune them. This can be brought about by several experimental manipulations: (1) by lid suture alone, because the deprived Y axons are somehow unable to develop the needed competitive advantage to displace the already established X arbors; (2) by removal of one eye alone, because Y arbors grow along a path of less resistance that leads them into the denervated lamina, thereby reducing competitive pressure on the X arbors; and (3) by the combination of monocular enucleation and suture of the remaining eye. In two-eyed cats, retinogeniculate Y axons never terminate in a lamina inappropriate for their eye of origin. This suggests the presence of some unspecified preference for all retinogeniculate axons to form arbors in the appropriate laminae, and the presence of X (and Y) axons already there from the other eye would help to discourage the establishment of such inappropriate connections.

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