

# Changes in Neuronal Cell Bodies in N. Laminaris During Deafferentation-Induced Dendritic Atrophy

JEFFREY S. DEITCH AND EDWIN W. RUBEL

Departments of Otolaryngology and Physiology, and the Neuroscience Program, University of Virginia Medical Center, Charlottesville, Virginia 22908

---

---

## ABSTRACT

N. laminaris dendrites begin to atrophy almost immediately after they are deafferented. Accompanying this rapid change in shape is a loss of microtubules and neurofilaments at the base of the dendrite, and a decrease in the density of the dendritic cytoplasm. However, degenerative changes in the dendritic plasma membrane were not evident until 2 days after deafferentation. Thus it was unknown what happened to the volume and membrane lost from the atrophying dendrites before this time. The soma was investigated in this study as a possible recipient of the volume of the atrophying dendrite. Soma size increased significantly by 2 hours after deafferentation and continued to increase for 1-8 days after deafferentation. The nucleus, which is normally concentric with the soma, moved continuously to the dorsal pole of the soma, toward the innervated side of the cell. The cytoplasm on the ventral side of the soma showed a decrease in density and loss of cytoskeleton similar to what was found in the initial portion of the ventral primary dendrites in the accompanying paper.

These changes are interpreted as indicative of a rapid resorption of the ventral dendrite back into the soma following deafferentation.

**Key words:** cytoskeleton, auditory system, cell size, morphometry

---

---

The ventral dendrites of nucleus laminaris (NL) undergo a rapid and specific atrophy following deafferentation (Deitch and Rubel, '84). However, ultrastructural observations revealed no signs of degeneration of the dendritic membrane until 2 days after deafferentation, by which time the dendrites were already 30% shorter (Deitch and Rubel, '88). There was no evidence of glia engulfing the dendrites. Furthermore, the dendrites on the dorsal side of NL, whose innervation remained intact, did not change in either size or ultrastructure. Thus the question arose of how the cell disposed of the membrane and volume from the atrophying dendrites. The present study examined the somata of NL neurons for changes in size and ultrastructure that paralleled the loss of the ventral dendrites.

In an earlier study, Benes et al. ('77) described the swelling of perikaryal organelles on the ventral side of the soma 4 days after deafferenting the ventral dendrites. They also noticed the accumulation of more healthy looking organelles on the innervated side. In the present study we examined these neurons for similar events at earlier times after deafferentation.

## MATERIALS AND METHODS

### Preparation of tissue

Chickens (9-12 days old) and surgical procedures were the same as in the preceding paper (Deitch and Rubel, '88). Also, sections used for the electron microscopic analysis of NL somata were from the same material used in the preceding paper. Paraffin-embedded, Nissl-stained sections of brain stem were used for measurements of soma and nuclear cross-sectional area, and position of the nucleus within the cell body. Chickens surviving either 2 ( $n = 2$ ) hours, or 1

Accepted October 21, 1988.

J.S. Deitch is now at the Wadsworth Center for Laboratories and Research, New York State Department of Health, Empire State Plaza, Albany, NY 12201-0509.

The research reported here was from a dissertation presented in partial fulfillment of the requirements for the PhD degree from the University of Virginia (JSD). Preliminary results were presented at the Annual Meeting of the Society for Neuroscience, Washington, DC, November 1986.

Address reprint requests to Dr. Edwin Rubel, Department of Otolaryngology, University of Washington, RL-30, Seattle, WA 98195.

( $n = 2$ ), 8 ( $n = 4$ ), or 16 ( $n = 2$ ) days following crossed dorsal cochlear tract (XDCT) transection, and unoperated controls ( $n = 4$ ), were perfused transcardially with phosphate-buffered 10% formalin. Brains were removed and embedded in paraffin. Ten-micron-thick sections were cut through the brain stem. Every fourth section was mounted, stained with thionin, and coverslipped. Sections containing NL were observed under a 100X objective on a Leitz Orthoplan microscope. This image was transferred to a TV monitor via a Dage MTI Series 68 camera such that the cell body lamina of NL was always oriented horizontally on the screen. The soma and nucleus of each cell with a visible nucleolus was then outlined at its maximum perimeter. A total of 2,107 neurons were measured (minimum of 200 neurons per time point). Cells were sampled from throughout NL.

A Videoplan image analysis system (Carl Zeiss, Inc.) was used to calculate the center of gravity for the soma and for the nucleus, recorded as X,Y coordinates on a digitizing tablet with dorsal always being in the positive Y direction. The distance between the center of the nucleus and the center of the soma in the dorsal-ventral direction was calculated by subtracting the Y coordinate for the soma from the Y coordinate for the nucleus. Thus a dorsal displacement of the nucleus had a positive value, whereas a ventral shift had a negative value. Similarly, a medial shift (along the X axis) was positive and a lateral shift was negative. The maximum distance from the center that a nucleus could travel (the radius of the soma minus the radius of the nucleus) was estimated for each cell. By this method, the eccentricity of the nucleus was expressed as a percentage of the maximum distance the nucleus could travel from the center to the dorsal or ventral pole of the cell, and also mediolaterally. The cross-sectional area of the soma also was measured, and the relative position of each measured cell within the plane of NL was recorded (described in Deitch and Rubel, '84). Analysis of variance of soma size and nuclear movement was performed using SPSS programs. For measurements of soma cross-sectional area, NL was divided into eight "sectors" (isofrequency regions of NL of equal width; see Deitch and Rubel, '84), and the mean of the measurements within each sector of NL in each animal was used for the analysis; the sectors were treated as repeated measures. Thus there were 8 repeated measures (sector means) per animal, and 2-4 animals per group. Whereas the repeated measures are dependent samples, the time points are independent samples.

## RESULTS

### Ultrastructure of NL cell bodies

The most notable characteristics of the normal NL somata were a preponderance of mitochondria and a thick shell of organized rough endoplasmic reticulum (RER), which was distributed fairly evenly at the perimeter of the cell (Fig. 1). In fortunate sections an oriented complex of microtubules, neurofilaments, and mitochondria was seen forming a track into the dendrite from deep in the perikaryon, passing through the shell of RER at the cortex of the cell and into the base of a dendrite. This organization created the impression of a streaming of cellular contents between the soma and the dendrite. The cytoplasm at the base of the dendrites, and in the initial portion of the dendrites (as reported in the preceding paper), is highly packed with organelles and flocculent material. The Golgi apparatus was found evenly distributed interior to the RER and also was occasionally found at the base of a dendrite,

although never in the dendrite. The nucleus was predominantly concentric with the soma.

Four hours after XDCT transection, there was no qualitative evidence of a reaction in the soma. The continuity of material between the soma and dendrite appeared uninterrupted. In the initial portions of the ventral dendrites at this time, there was a decrease in microtubule content (Deitch and Rubel, '88), although qualitatively it was not yet obvious.

At 12 hours after deafferentation there was a noticeable thinning of the cytoplasm under the dendrites on the ventral side. The streaming of organelles into the dendrites was reduced, sometimes appearing as a gap in the perikaryon and extending into the dendrite. The initial portion of the ventral dendrite also had a decreased opacity, which in part was due to the decrease in cytoskeletal elements described in the preceding paper. By 1 and 2 days after deafferentation, this lucent area under the dendrites had spread in some cells, forming gaps around the cell between the RER and the plasma membrane. In some sections this space was limited to the ventral half of the cell body (Fig. 2), whereas in others the gap extended around the entire cell cortex (Fig. 3). The gap was never seen just on the dorsal side, and at no time did it interfere with the continuity of cytoplasm into the dorsal dendrites (Fig. 3). Since the contraction of the cytoplasm away from the plasma membrane can also be a fixation artifact, other neurons in the region (NM, vestibular neurons) were examined and were found not to have similar gaps under the plasma membrane.

Membrane-bound organelles in the NL cell body also did not show any signs of fixation artifact; nor did they exhibit any ultrastructural changes at the survival times examined in this study. Specifically, there was no obvious swelling of the Golgi complex nor any accumulation of vesicles, multivesicular bodies, or lysosomes. In some neurons an accumulation of RER on the ventral side was already apparent (Fig. 2).

### Size of the soma

A surprising result was a rapid change in the size of the soma following deafferentation. Whereas a decrease in soma size is a common reaction to deafferentation in many neural systems (see Born and Rubel, '85), the cell bodies of NL neurons initially *increased* in cross-sectional area for 1 and 8 days following XDCT transection, and then decreased toward control levels by 8 to 16 days (Fig. 4). The increase in soma area was statistically significant by 2 hours after deafferentation [ $F(1,3) = 400.7$ ,  $p < .001$ ], and the change in soma size over time is described by a quadratic relationship [ $F(4,13) = 227.5$ ,  $p < .001$ ]. This pattern of an increase and then decrease in soma area after deafferentation occurred in sectors 1-7; in the most posterolateral sector (8) the soma size remained significantly elevated at 16 days.

### Migration of the nucleus

In control neurons the nucleus, on the average, was concentrically situated within the shell of rough endoplasmic reticulum (RER). By 16 days after deafferentation the nucleus was more frequently found in the dorsal half of soma, and the RER accumulated on the ventral side (Fig. 5).

The position of the nucleus is represented here as the percentage of the maximum eccentricity of the nucleus within each cell (see Methods). The results are shown in Figure 6.

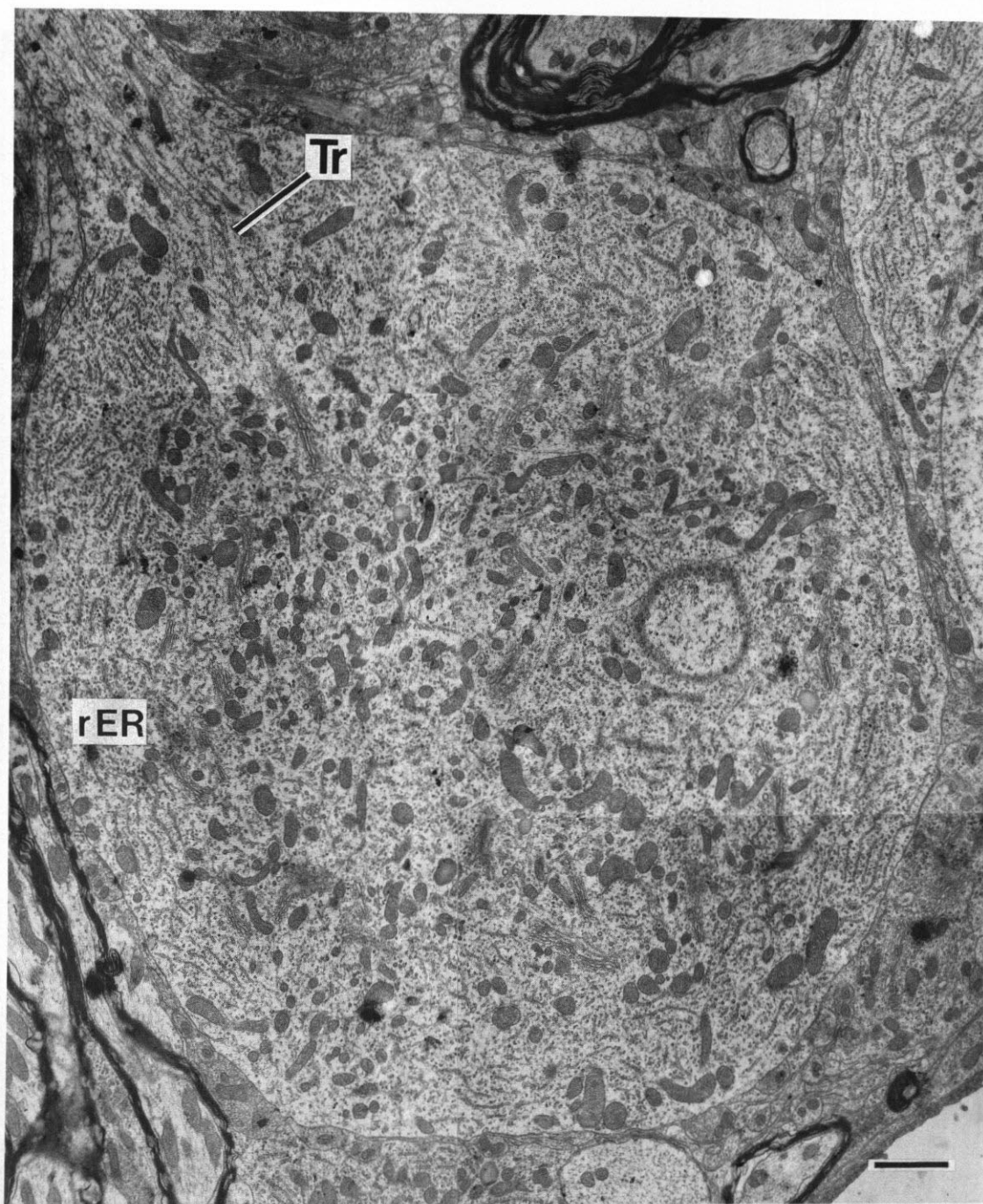


Fig. 1. Normal NL soma. Note the shell of RER at the periphery of the cell body. This pattern is interrupted by a track (TR) of perpendicu-

larly oriented organelles in continuity with the dendritic cytoplasm. In this and subsequent figures, dorsal is at top. Bar = 2  $\mu$ m.

In control animals the average NL nucleus resided  $0.00 \pm 5.0\%$  (s.e.m.) of the maximum possible shift from center to the dorsal pole; i.e., the position of the nucleus was distributed around the center of the soma. Two hours after deafferentation the average nucleus resided  $18.0 \pm 11.9\%$  of the maximum dorsal shift, thus showing a preference for the dorsal side. By 16 days the nucleus was an average  $67.0 \pm 20.0\%$  of the maximum distance dorsally. The shift in the position of the nucleus as a function of time was statistically significant [ $F(4,9) = 6.24$ ,  $p < .05$ ]. Although the mean distance of the nucleus from the center of the soma shifted continuously toward the dorsal side, only the animals surviving

8 and 16 days after deafferentation were statistically different from controls by *a posteriori* comparisons ( $p < .05$ , least significant difference). This was probably due to the large variability in this measure. Analysis of the position of the nucleus in the mediolateral axis revealed no reliable movement [ $F(4,7) = 0.61$ ,  $p > .50$ ].

## DISCUSSION

Electron microscopic examination of the atrophying ventral dendrites of NL following deafferentation revealed rapid changes in the cytoskeleton of the dendrites, but



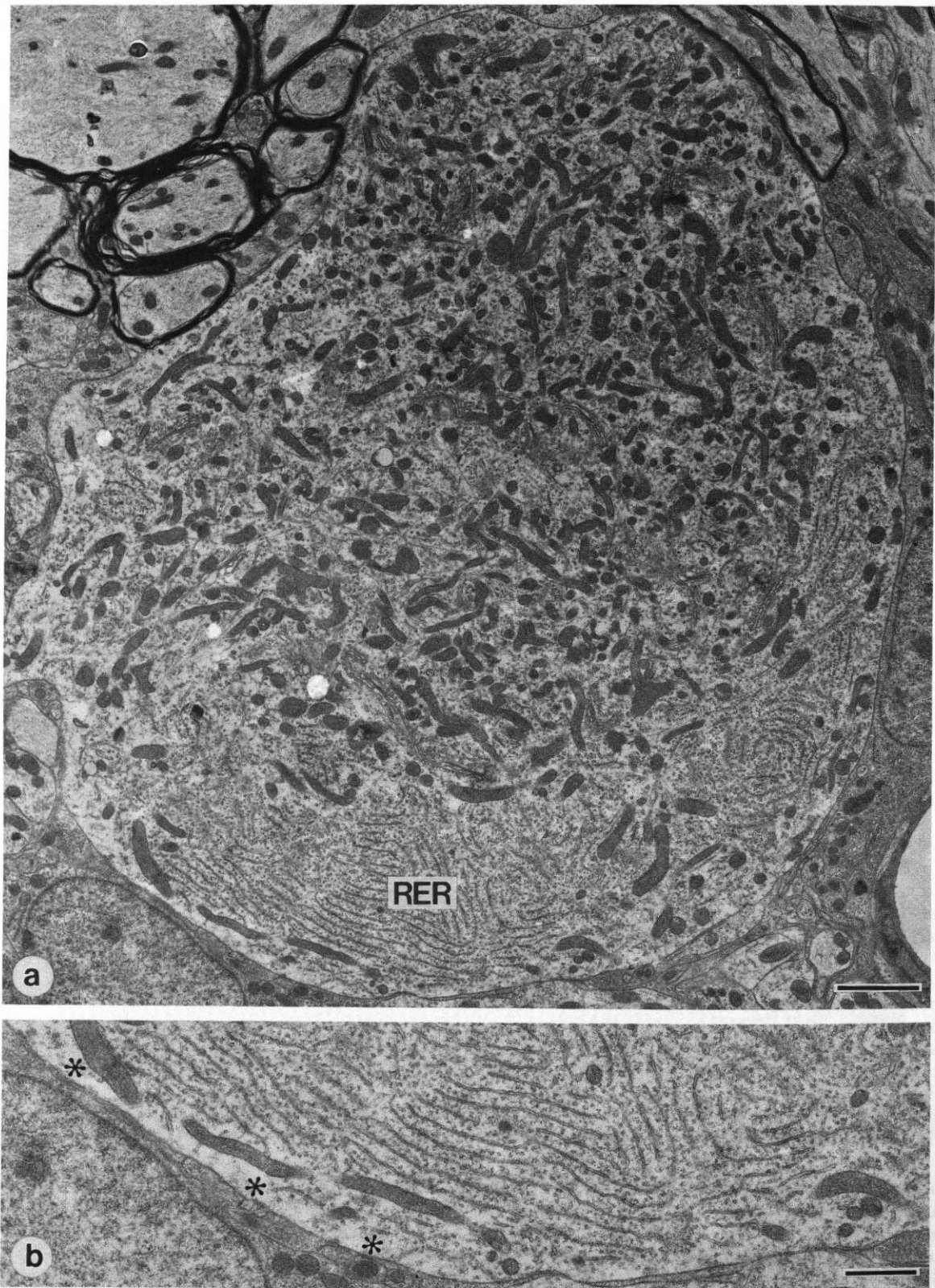


Fig. 2. (a) Example of an NL soma 1 day after XDCT transection, showing the relocation of RER from a circumferential distribution to a mass on the ventral side of the cell. Bar = 2  $\mu$ m. (b) Higher magnification of the ventral surface of the soma in (a). Compare the gap between

the organelles and the ventral plasma membrane (\*) with the abutment of organelles against the membrane on the dorsal side of the cell. Bar = 1  $\mu$ m.



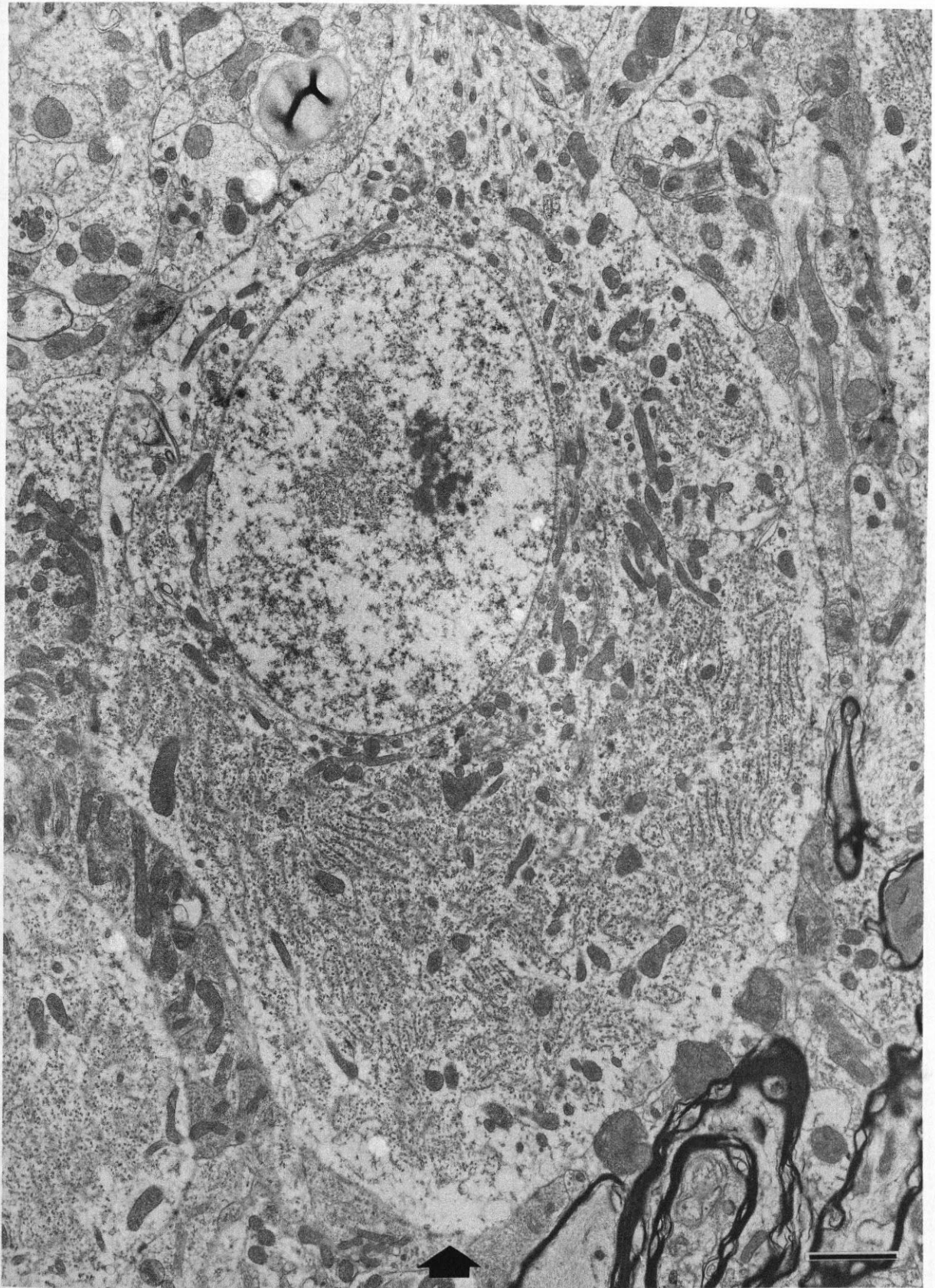


Fig. 3. Example of an NL soma 2 days after XDCT transection, showing a space formed at the periphery of the soma (arrow). Note that this space intrudes on the dorsal side of the cell but does not interfere

with the continuity of cytoplasmic organelles from the soma into the dorsal dendrites (top). Bar = 2  $\mu$ m.

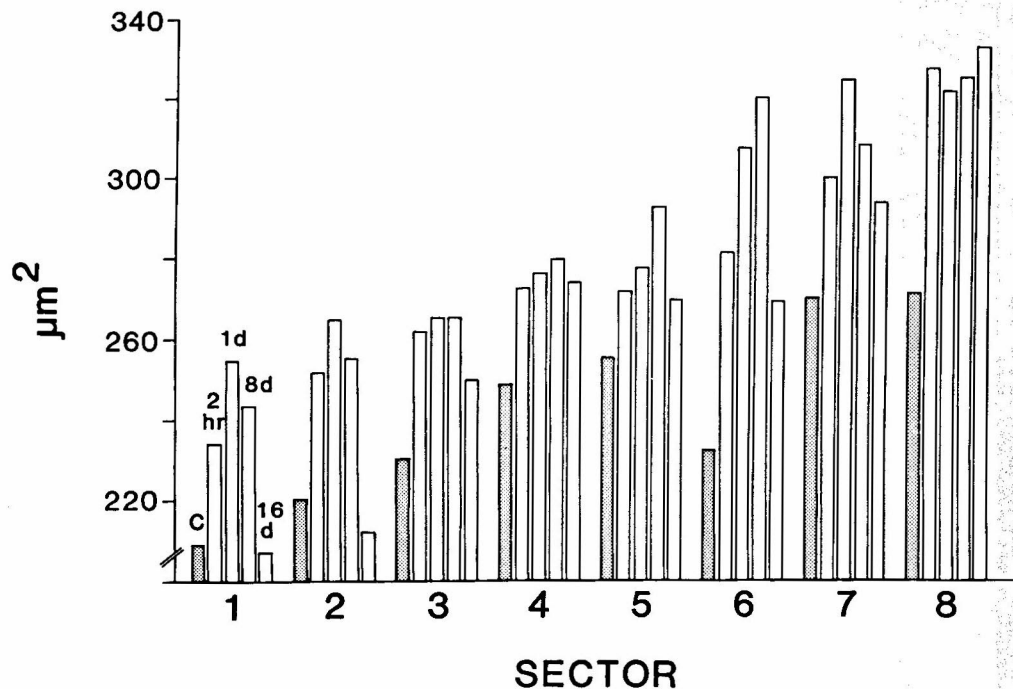


Fig. 4. Histogram of the change in average cross-sectional area of NL cell bodies over time after deafferentation (2 hours, and 1, 8, and 16 days) compared separately within each sector (1–8) of NL (mean  $\pm$  s.e.m.). Control values are represented by shaded bars. There is normally an increase in soma size from the anteromedial (sector 1) to

posterolateral (sector 8) regions of the nucleus. Soma cross-sectional area rapidly increases after XDCT transection—by 2 hours the soma is significantly larger ( $p < .001$ ). The increase and subsequent decrease in soma size over time fits a quadratic relationship ( $F = 227.5$ ,  $p < .001$ ).

failed to uncover a mechanism for the degradation of the dendrites during the first days after deafferentation (Deitch and Rubel, '88). Despite the loss of 30% of the dendrite, there was no sign of dendritic plasma membrane degeneration, no pinching off of dendrite (e.g., by glia), nor any detectable widening of the dendrite. Since the dorsal dendrites did not respond to deafferentation of the ventral dendrites with a change in either their size or ultrastructure, the NL soma was investigated as a possible depository for the membrane and volume lost from the ventral dendrites.

### Changes in the soma after deafferentation

There were no obvious changes in the size or integrity of the organelles in the cell body in the first 2 days after deafferentation. Benes et al. ('77) reported the swelling of organelles ventrally within the soma 4 days after XDCT transection. In the present study the organelles found ventrally after 2 days of deafferentation appeared normal, although their dense organization and continuity into the ventral dendrites was disrupted by gaps in the ventral perikaryon. The clearing of the cytoplasm at 2 days after deafferentation may precede the appearance of damaged organelles reported by Benes et al. ('77) 4 days after deafferentation. However, the chickens used by Benes et al. were 5–7 days old (and a different breed) and may have reacted faster or more severely to deafferentation.

The soma significantly increased in size only 2 hours after deafferentation. This increase peaked 1–8 days later and was largely reversed by 16 days postsurgery. In most other instances of deafferentation the size of the soma decreased a few days after deafferentation (e.g., Powell and Erulkar, '62; Guillery, '73; Born and Rubel, '85). However, in these examples the decrease in the average size of the soma was fol-

lowed by the death of a subpopulation of the deafferented cells and thus may have represented a different process. No loss of cells has been found in NL 16 days after XDCT transection in younger chickens (Rubel and Brandow, unpublished observations). Finally, the movement of the nucleus appeared to start within 2 hours following XDCT transection and continued dorsally through 16 days after deafferentation.

### Sites of regulation

Several aspects of the data presented here and in the preceding paper suggest that the initial response of the ventral dendrites to deafferentation is a disruption of structure at the base of the dendrites, rather than at more distal sites. Specifically, the cytology of the distal segments of dendrite (and their afferent connections) did not appear to change until 2 days after XDCT transection, by which time the dendrites were already over 30% shorter. However, in the more proximal portions of the dendrite and in the soma several changes occurred rapidly: microtubule density decreased by 60% in 12 hours, the dendroplasm became more lucent, with gaps in the continuity of materials from the soma, and by 2 hours after XDCT transection the soma had significantly increased in size (12%).

Our hypothesis suggesting the local regulation of dendrite by its afferents, i.e., acting directly at the innervated surface (Deitch and Rubel, '84), must be amended. Although it still remains true that the deafferented and innervated dendritic trees (ventral and dorsal, respectively) on the same cell are regulated separately, the results presented here do not support the idea that each terminal regulates the survival of the dendritic segment underneath it. Rather, they suggest that afferent innervation maintains entire dendritic trees by acting on the cytological integrity of the dendrite base.



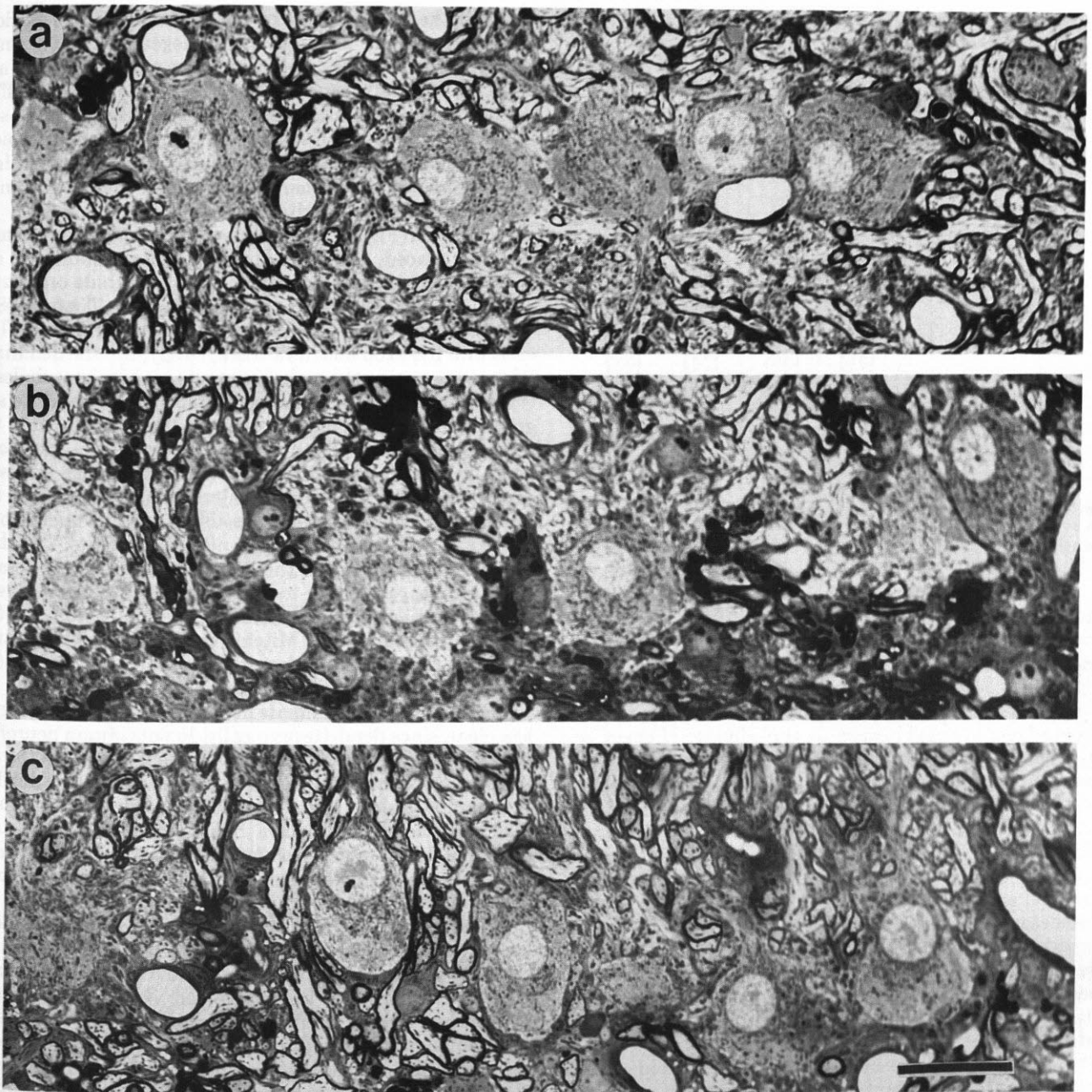


Fig. 5. Photomicrographs of 1- $\mu$ m-thick plastic sections stained with toluidine blue from a control animal (a), from an animal surviving 2 days (b), and from an animal surviving 16 days (c) after XDCT transec-

tion. Note the displacement of the nucleus from a central location, in (a), to a dorsal location, in (b) and (c). Bar = 20  $\mu$ m.

This does not rule out local effects of deafferentation. In many instances deafferentation results only in a loss of spines on the target dendrite (Globus and Scheibel, '66, '67; Valverde, '68; Parnavelas, '74). Studies by Steward and colleagues point to very complex local interactions between the distal dendrites of dentate gyrus neurons and their afferents (Caceres and Steward, '83; Steward and Vinsant, '83), whereas innervation persists at the base of these dendrites. These changes (such as protein synthesis) have not been examined in NL. It is possible that subtle changes in ultrastructure occur along the NL dendrites, but the gross changes are expressed first at the base.

The rapid atrophic response of the NL dendrites and soma to deafferentation may be revealing processes that

underly the normal ongoing regulation of dendritic size and shape. If dendrites are dynamically changing—constantly extending and retracting over a short range—then the balance of input at any given time would be altering the balance of this dynamic equilibrium. Those dendrites in the process of elongation will be slowed and eventually reversed by deafferentation. However, those dendrites in the normal process of retraction are already primed for the effects of a loss of afferents, and thus can act very quickly, as they do in NL. Although NL dendrites have nearly attained their adult morphology by 9–12 days old (Smith and Rubel, '79; Smith, '81; Deitch and Rubel, '84), the presence of filopodial extensions on some of the dendrites and bulbous endings on others suggest that they may still be actively extending and



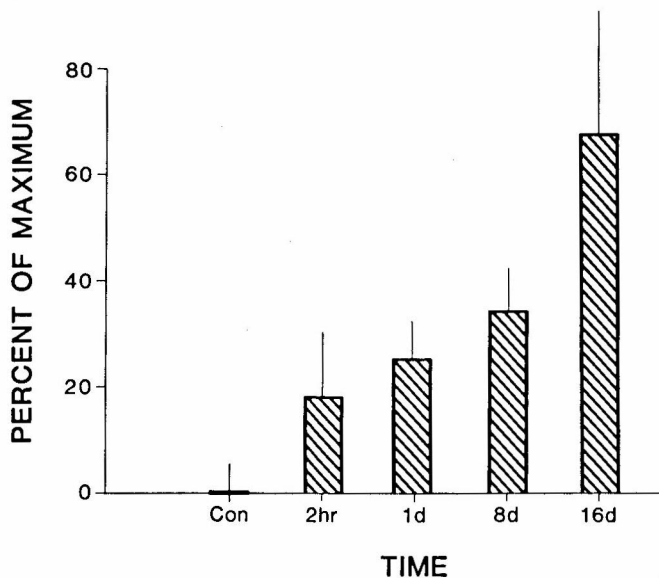


Fig. 6. Histogram showing the average position of the nucleus in the soma as a percentage of the maximum distance the nucleus can move off-center in the dorsal direction. (Mean  $\pm$  s.e.m.). The trend is significant ( $F = 6.24$ ,  $p < .05$ ) and is reliably different at 8 days ( $p < .05$ ).

withdrawing processes (see Deitch and Rubel, '88, Fig. 5). Some evidence already exists for a dynamic balance regulating dendritic morphology (Purves et al., '86).

Figure 7 is an initial attempt to schematically illustrate the cellular events that link the changes in dendritic size and cytoskeleton organization to the somatic changes reported here. The NM afferents (or the effects of synaptic activation) appear to exert a trophic influence on a subpopulation of microtubules, neurofilaments or their associated proteins, within the postsynaptic dendrite. If the structural equilibrium of the cytoskeleton at the dendrite base is required for stability of the dendrite (that is, the dendrites are not being "held out" by their tips), a breakdown of the cytoskeleton at the base will cause the dendrite to be resorbed back into the soma. As this process continues the more distal parts of the dendrite would be "reeled in," increasing the size of the soma and decreasing the size of the ventral dendrite, without affecting the size of the dorsal dendrites.

The movement of the nucleus is also consistent with a selective change in the ventral cytoskeleton. The nucleus is normally embedded in the cytoskeleton, so much so that it is held in place by the cytoskeleton after extraction of the cell in detergent (Marchisio et al., '78). Dissolution of the cytoskeleton on the ventral side after deafferentation might have created an imbalance in the cytoskeleton, favoring the intact side and pulling the nucleus toward it. This reorganization of the soma also may be responsible for the accumulation of RER on the ventral side of the cell body (Benes et al., '77).

In addition to deafferenting the ventral dendrites, XDCT transection also removes half of the NM terminals on the NL soma. The degenerating NM terminals, covering approximately 22% of the soma surface (from Parks et al., '83), may induce local changes under the plasma membrane (such as the subplasmalemmal gap, Figs. 2,3), but it is unlikely that they play a specific role in the larger morphological changes described here. The afferent endings from both the ipsi- and contralateral NM are intermixed over the cell body surface (and to a small extent on the initial den-

drites of the opposite side of the lamina). It is not obvious how a symmetric distribution of degenerating terminals would orchestrate the movement of the nucleus dorsally and the RER ventrally. Furthermore, one would expect deafferentation of the soma to result in a shrinkage in cross-sectional area (Born and Rubel, '85), rather than an expansion. Thus the increase in soma size and movement of the nucleus require the structural changes associated with deafferentation of the ventral dendrites.

As drawn in Figure 7, the nucleus in normal NL neurons is centrally placed, and the cytoplasmic microtubules are anchored at one end by a perinuclear microtubule organizing center (Spiegelman et al., '79; Jacobs and Thomas, '82). These microtubules presumably extend their growing (+) ends into the axon and, as a track, partly into the dendrites. We are suggesting the presence of one set of microtubules in the dendrites that is anchored distally in the dendrite and grows into the soma and another set that is oriented in the opposite direction. Both sets of microtubules can form cross-links with neurofilaments, with membrane, and with each other. The overlapping microtubular systems at the base of the dendrites would result in greater microtubule density in proximal vs. distal dendritic segments. Two days after deafferentation of the ventral dendrites, the loss of microtubules, according to our hypothesis, would be due to the loss of microtubule subunits sequentially from the (+) end (see Kirschner and Mitchison, '86) of the cytoplasmic and dendritic microtubules, both of which have their (+) ends at the base of the dendrite.

The role of neurofilaments in NL dendritic morphology is less clear, since dendrites typically do not contain neurofilaments. We have found that neurofilament density in the initial portion of the dendrites does decrease following deafferentation, at a slower rate than microtubules, but are more severely affected after 2 days than are microtubules. Neurofilaments do not undergo assembly/disassembly like microtubules and actin filaments; thus their decrease is most likely due to their destruction.

Finally, whereas the nucleus continued to move dorsally over 16 days after deafferentation, the increase in the mean size of the soma peaked between 1 and 8 days; in most parts of NL the process then reversed so that soma size approached that found in control animals. This most likely represents an upper limit on the amount of dendrite able to be resorbed, as well as a homeostatic control over the size of the soma returning it to its normal size. Thus the continued loss of dendrite past 1 day cannot be explained solely by resorption into the soma. In fact, the time course of dendrite loss determined from observations of Golgi-stained material (Deitch and Rubel, '84) revealed two phases of atrophy—an initial fast loss of dendrite (30% in 1 day) followed by a more gradual atrophy (another 30%) over the next 15 days. Thus the resorption of dendrite into the soma may underlie the rapid first phase of dendritic withdrawal, whereas the vesiculation of dendritic membrane slowly removes the remaining dendrite over the next 16 days.

The vesiculation of the dendritic membrane found at 2 days after deafferentation suggests that the additional loss of dendrite is due to degeneration of the dendrites, especially at their distal ends. Since membrane degeneration was not evident earlier than 2 days after XDCT transection, either the degenerating afferents suddenly began releasing a necrotic factor, or a delayed degenerative process took effect within the dendrite. In the latter case, the eventual degeneration of dendritic membrane could be a delayed effect of a

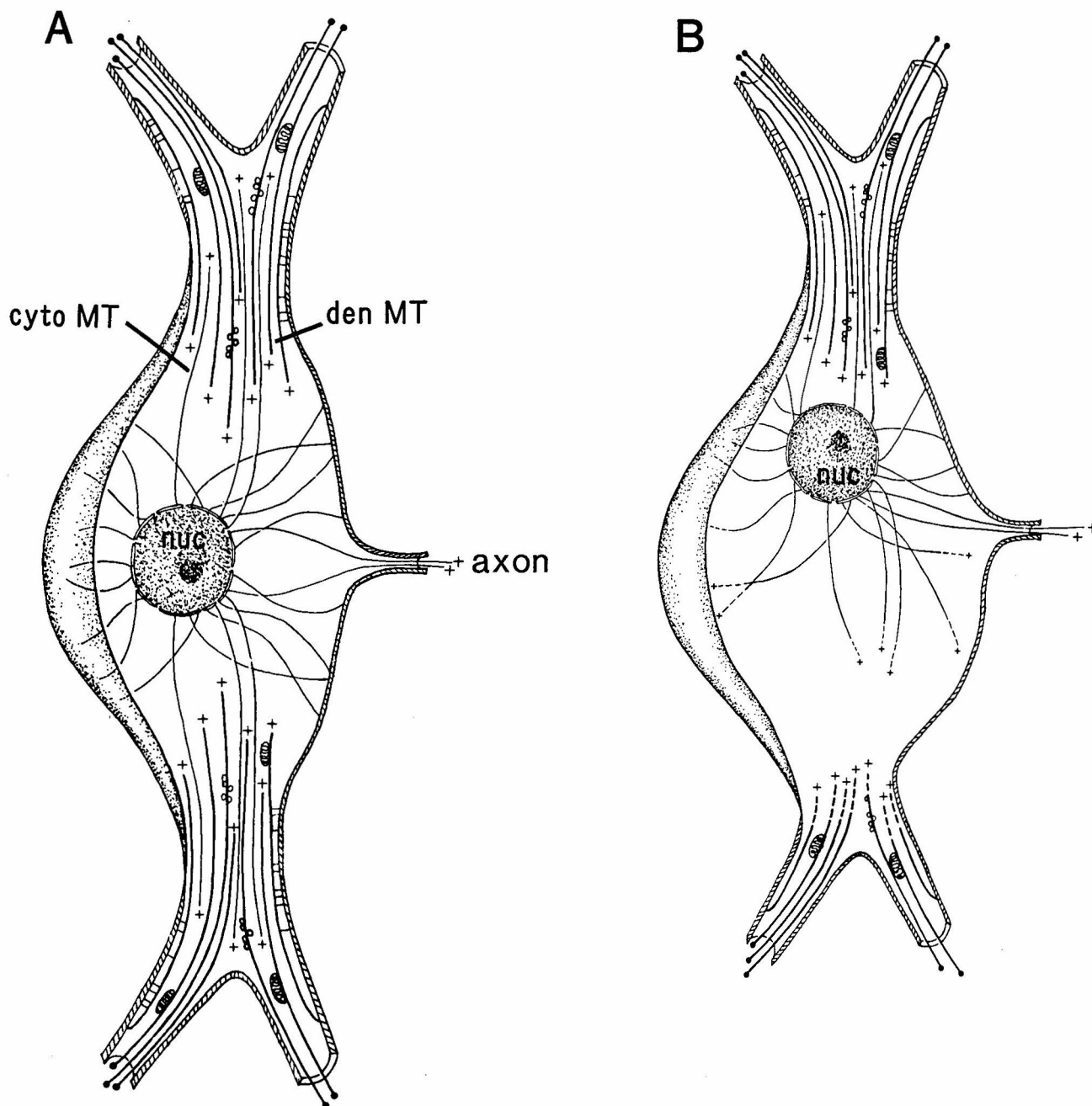


Fig. 7. Schematic representation of proposed organization and polarity of microtubules in normal and deafferented NL neurons. A. In normal NL neurons the nucleus (nuc) is centrally placed, where the cytoplasmic microtubules (cyto MT, thin lines) are anchored at one end by a perinuclear microtubule organizing center (Spiegelman et al., '79; Jacobs and Thomas, '82). These microtubules presumably extend their growing (+) ends into the axon and, as a track, partly into the dendrites. We hypothesize the presence of a separately organized set of microtubules in the dendrites (den MT, thick lines), which are anchored distally in the dendrite and grow into the soma. Both sets of microtubules can form cross-links with neurofilaments, membrane, and each other, and

the overlapping microtubular systems at the base of the dendrites results in the greater microtubule density found in the proximal vs. distal dendrite. B. One day after deafferentation of the ventral dendrites, the nucleus has drifted dorsally, the soma has expanded, and a zone sparse in organelles has formed at the base of the dendrite. The density of microtubules is reduced proximally but is normal distally in the ventral dendrites. The dorsal side remains identical to controls. The loss of microtubules, according to our hypothesis, would be due to the loss of microtubule subunits sequentially from the (+) end (see Kirschner and Mitchison, '86) of the cytoplasmic and dendritic microtubules, both of which have their (+) ends at the base of the dendrite.

loss of dendritic transport, caused by the disruption of the cytoskeleton at the base of the dendrite. The gap formed at the base of the dendrite by 2 days after deafferentation, as well as the lack of a buildup of organelles in the shortening dendrites, are consistent with a loss of transport into the ventral dendrites, but this has yet to be directly investigated.

## ACKNOWLEDGMENTS

We greatly appreciate the assistance of Drs. Lynne Olsho and Nancy Spetner for their help and guidance with the statistical analyses. This research was supported by PHS grant NS 19395, the Lions of Virginia Hearing Foundation, and the Pratt Foundation of the University of Virginia.

## LITERATURE CITED

- Benes, F.M., T.N. Parks, and E.W. Rubel (1977) Rapid dendritic atrophy following deafferentation: An EM morphometric analysis. *Brain Res.* 122:1-13.
- Born, D.E., and E.W. Rubel (1985) Afferent influences on brain stem auditory nuclei of the chicken: Neuron number and size following cochlea removal. *J. Comp. Neurol.* 231:435-445.
- Caceres, A., and O. Steward (1983) Dendritic reorganization in the denervated dentate gyrus of the rat following entorhinal cortical lesions: A Golgi and electron microscopic analysis. *J. Comp. Neurol.* 214:387-403.
- Deitch, J.S., and E.W. Rubel (1984) Afferent influences on brain stem auditory nuclei of the chicken: Time course and specificity of dendritic atrophy following deafferentation. *J. Comp. Neurol.* 229:66-79.
- Deitch, J.S., and E.W. Rubel (1988) Rapid changes in ultrastructure during deafferentation-induced dendritic atrophy. *J. Comp. Neurol.* (submitted).
- Globus, A., and A.B. Scheibel (1966) Loss of dendritic spines as an index of presynaptic terminal pathways. *Nature* 212:463-465.
- Globus, A., and A.B. Scheibel (1967b) Synaptic loci on parietal cortical neurons: Terminations of corpus callosum fibers. *Science* 156:1127-1129.
- Guillery, R.W. (1973) Quantitative studies of transneuronal atrophy in the dorsal lateral geniculate nucleus of cats and kittens. *J. Comp. Neurol.* 149:423-438.
- Jacobs, M., and C. Thomas (1982) The organization of 10 nm filaments and microtubules in embryonic neurons from spinal ganglia. *J. Neurocytol.* 11:657-669.
- Kirschner, M., and T. Mitchison (1986) Beyond self-assembly: From microtubules to morphogenesis. *Cell* 45:329-342.
- Marchisio, P.C., M. Osborn, and K. Weber (1978) The intracellular organization of actin and tubulin in cultured C-1300 mouse neuroblastoma cells. *J. Neurocytol.* 7:571-582.
- Parks, T.N., and E.W. Rubel (1975) Organization and development of brain stem auditory nuclei of the chicken: Organization of projections from n. magnocellularis to n. laminaris. *J. Comp. Neurol.* 164:435-448.
- Parks, T.N., P. Collins, and J.W. Conlee (1983) Morphology and origin of axonal endings in nucleus laminaris of the chicken. *J. Comp. Neurol.* 214:32-42.
- Parnavelas, J.G., G. Lynch, N. Brecha, C.W. Cotman, and A. Globus (1974) Spine loss and regrowth in hippocampus following deafferentation. *Nature* 248:71-73.
- Powell, T.P.S., and S.D. Erulkar (1962) Transneuronal cell degeneration in the auditory relay nuclei of the cat. *J. Anat.* 96:249-268.
- Purves, D., R.D. Hadley, and J.T. Voyvodic (1986) Dynamic changes in the dendritic geometry of individual neurons visualized over periods of up to three months in the superior cervical ganglion of living mice. *J. Neurosci.* 6:1051-1060.
- Smith, D.J., and E.W. Rubel (1979) Organization and development of brain stem auditory nuclei of the chicken: Dendritic gradients in nucleus laminaris. *J. Comp. Neurol.* 186:213-240.
- Smith, Z.D.J. (1981) Organization and development of brain stem auditory nuclei of the chicken: Dendritic development in n. laminaris. *J. Comp. Neurol.* 203:309-333.
- Spiegelman, B.M., M.A. Lopata, and M.W. Kirschner (1979) Aggregation of microtubule initiation sites preceding neurite outgrowth in mouse neuroblastoma cells. *Cell* 16:253-263.
- Steward, O., and S.L. Vinsant (1983) The process of reinnervation in the dentate gyrus of the adult rat: A quantitative electron microscopic analysis of terminal proliferation and reactive synaptogenesis. *J. Comp. Neurol.* 214:370-386.
- Valverde, F. (1968) Structural changes in the area striata of the mouse after enucleation. *Exp. Brain Res.* 5:274-292.