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Projections of the Cochlea to the Dorsal Cochlear Nucleus in the Cat

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Degenerated axons have been traced to the dorsal cochlear nucleus with the Nauta method after cochlear ablations in cats. Collaterals of the descending branch of the auditory nerve project as very fine fibers to the molecular layer and as fine and coarse fibers to the fusiform cell and deep layers. The pattern of preterminal degeneration after short survivals is typical of both axosomatic and axodendritic endings on small and large cells in this nucleus, including the fusiform cells and giant cells. This nucleus probably has reciprocal connections with the posterior colliculus. Thus, the auditory signal transformations occurring between the cochlea and midbrain could be directly influenced by the fusiform and giant cells.

Introduction

The auditory nerve enters the cochlear nucleus and bifurcates into an ascending branch to the anteroventral and a descending branch to the posteroventral cochlear nucleus and the dorsal cochlear nucleus (DCN) (2, 5, 7, 15). Although it is thought that the DCN projects to the posterior colliculus (10, 21, and Warr, unpublished), some doubt exists regarding the exact terminations of the auditory nerve fibers within the DCN (6, 12, 14, 19). In order to define the functional relationship of the neuronal organization to auditory activity in the DCN, it is necessary to have an accurate account of the terminal sites of the primary auditory nerve fibers in the different parts of the nucleus and on particular cell types. We have undertaken studies of the axonal degeneration after cochlear ablations in cats to clarify this problem.

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Materials and Methods

Cochlear ablations were performed under aseptic conditions and pentobarbital anesthesia on five adult cats. The tympanic bulla was exposed through a ventral, midline incision of the neck. After partial removal of the bulla a probe, inserted in the round window, was used to ablate the cochlea. After postoperative survivals of 2, 3, 4, and 7 days (two cats) the brains were fixed by intracardiac perfusion of a formol–dichromate fixative, sectioned serially at 24 μ in the transverse plane on a freezing microtome, and prepared for study by the Nauta method (details in 8, 9). At least every other section was studied. In two cases (3- and 7-day survivals) both cochleas were destroyed. Series of Nissl- and protargol-stained sections, cut in the three standard planes, from normal adult cats were used for comparison The extent of the lesion was assessed in each case by microscopic dissection and inspection of the temporal bone. Findings could be verified in an animal without signs of damage to the vestibular ganglion or microscopic evidence of degeneration in the vestibular nuclei. There is no microscopic evidence of direct damage to the cochlear nucleus in any case.

Results

The DCN of the cat (Fig. 1) consists of four distinctive layers (Figs. 339, 340 in 15): the ependymal layer of the lateral recess of the fourth ventricle, the molecular layer (superficial plexiform layer in 15), the fusiform cell layer (granular layer in 15, Fig. 339), and the deep layer (deep plexiform layer in 15). The deep layer contains very large neurons, or giant cells (11), as well as small cells and granule cells, in a thick feltwork of fibers (Fig. 2A). The fusiform cell layer is the region of the cell bodies of the fusiform cells; this layer also contains the greatest concentration of granule cells (Fig. 3A). The molecular layer contains small cells and granule cells, embedded in a fine neuropil (Fig. 4).

After complete cochlear ablations, uniformly heavy preterminal degeneration appeared, on the lesioned side only, throughout the ventral cochlear nucleus and the deep layer of the DCN (Fig. 1). Heavy to moderate degeneration extended from the deep to the superficial portions of the entire fusiform cell layer. Sparse degeneration occurred throughout the molecular layer. Degenerated fibers were not found in the trapezoid body or in other auditory tracts and nuclei of the medulla. The most abundant and representative preterminal degeneration appeared 4 days postoperatively in the present series.

Throughout the deep layer a dense thicket of preterminal degeneration of coarse and fine fibers enveloped the giant cells, including their perikarya and dendritic trunks (Fig. 2A,B). Preterminal degeneration also appeared in association with small cell bodies and perhaps less clearly with granule

cells. In the neuropil the pattern of preterminal degeneration was characterized by more or less distinct clusters of coarse and fine axonal fragments, not clearly associated with cell bodies (Fig. 2A, B, D).

The fusiform cell layer was penetrated by coarse, medium-sized, and fine, degenerated fibers (Fig. 3). Degenerated axonal fragments occurred around the basal dendrites, the cell somas, and the apical dendrites of the fusiform

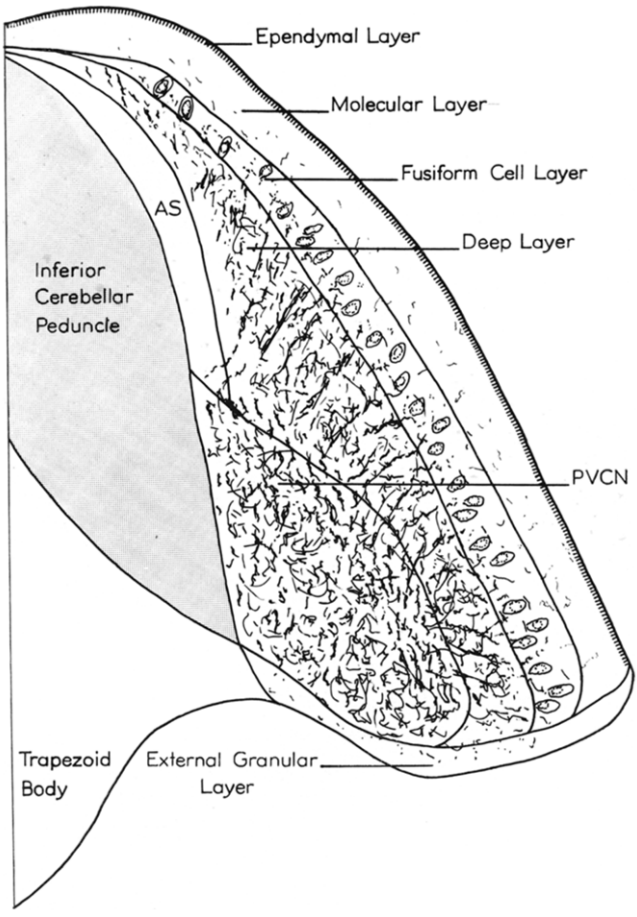


FIG. 1. Typical distribution and relative intensity of axonal degeneration, 4 days after a complete cochlear ablation, shown semischematically in a transverse section of the DCN at the level of the caudal region of the posteroventral cochlear nucleus (PVCN). Thick fibers of the descending branch of the auditory nerve appear in cross section in the PVCN and in the deep layer of the DCN along its deep border. Preterminal degeneration also occurs in the external granular layer. AS, dorsal and intermediate acoustic striae.

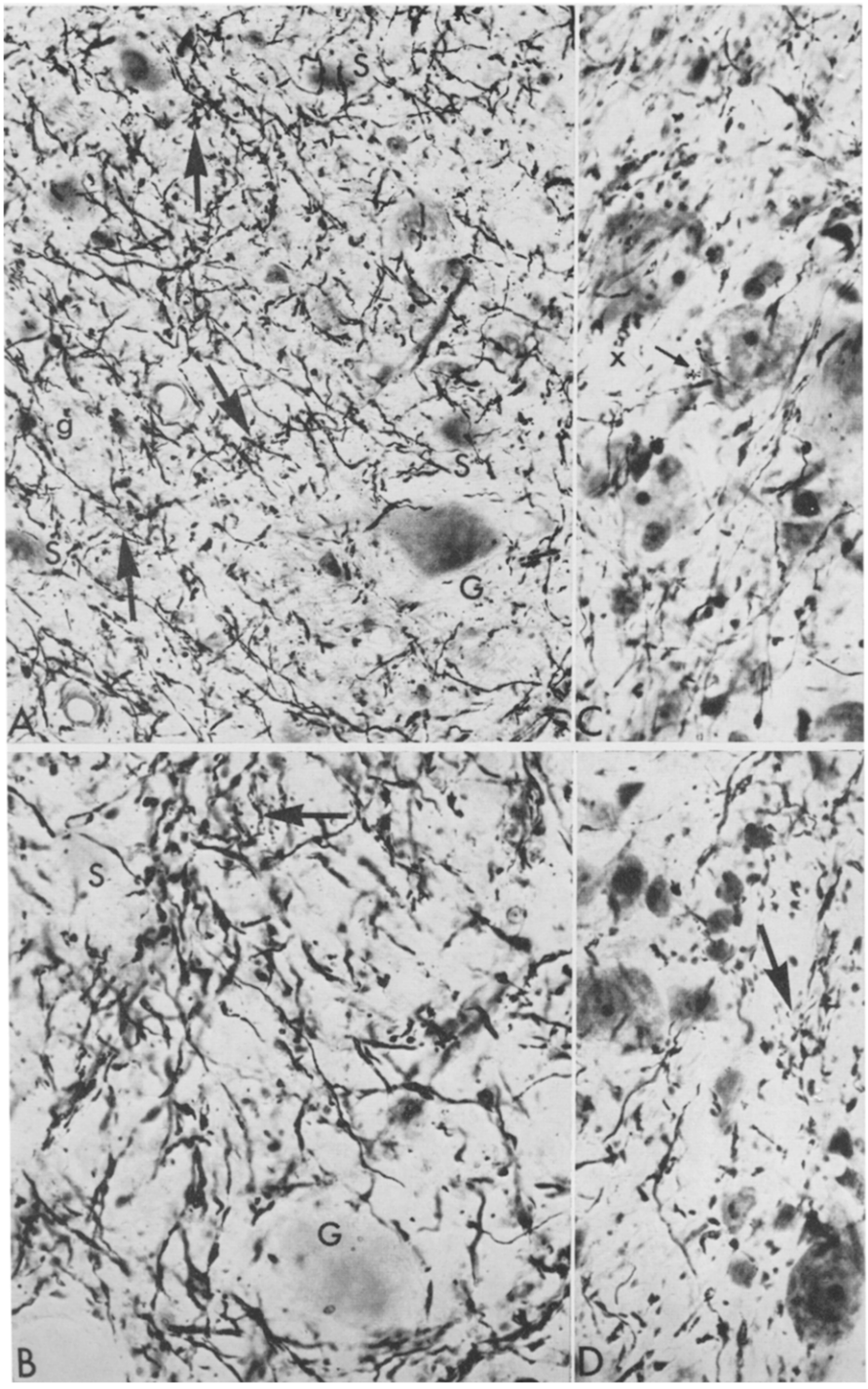
cells. Preterminal degeneration also was associated with the small cells of the fusiform cell layer. The degeneration was moderately dense in the deep region and gradually became sparse in the superficial region of the fusiform cell layer. Between the fusiform cells a characteristic pattern of preterminal degeneration appeared among the granule cells in the form of very small nests, or rosettes, consisting of coarse-to-medium masses, ringed by very fine, powdery debris (Fig. 3A, B). Some of these rosettes also appeared in the molecular layer.

The molecular layer contained a small number of medium-sized and very fine, degenerated fibers, entering from the fusiform layer (Fig. 4) only on the side of the lesion. The medium-sized fibers ran through the molecular layer perpendicular to the apical dendritic trunks of the fusiform cells. Many of the fine fibers ran toward the ependymal surface, parallel to the long axes of the fusiform cells. In a few cases very fine preterminal degeneration appeared around small cells.

Some differences in types and quantities of degenerated fibers were found at different survival times. At 7 days a heavy distribution of fine, as well as coarse, degenerated fibers appeared in the deep and the fusiform cell layers (Fig. 2C). Fine degenerated fibers within the molecular layer were scarce. In contrast, at 2 days mainly coarse fibers of passage and coarse fragments were associated in moderate intensity with giant and small cell somas and dendrites in the deep layer (Fig. 2D) and with fusiform cell bodies and dendritic trunks. The relatively little fine fiber degeneration was generally diffuse in both regions. Since fine fibers of passage were rarely recognized in the deep layer at this early stage of degeneration, it may be that many of the fine axonal fragments represent thin collaterals of the degenerating coarse fibers. The coarse preterminal degeneration already clearly aggregated in clusters in the early stages, even in the absence of fine preterminal fragments. These observations suggest that the coarse axons participate in the preterminal clusters, whatever the contributions of the finer axons.

Discussion

In a drawing by Ramón y Cajal (Fig. 340 in 15) collaterals of the descending branch of the auditory nerve in the cat appear to arborize around large fusiform cell bodies (bipolar cells in 7; pyramidal cells in 11). Moreover, Lorente de Nó (7) described primary fibers, projecting to these elongate cell bodies in the cat, but neither Ramón y Cajal nor Lorente de Nó traced auditory fibers to the molecular layer or to the giant cell bodies in the deep layer. In our rapid Golgi preparations from kittens and young cats, however, axons from the descending branch of the auditory nerve arborize in all three layers of the DCN (Cohen, unpublished.) The validity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of such observations can be verified experimentally by the degeneration methods.

Cochlear ablations have been used previously to demonstrate degeneration of the primary afferents of the DCN in cats. Using the Marchi technique, Lewy and Kobrak (6) observed some degeneration in the "superficial" DCN. Rasmussen, Gacek, McCrane, and Baker (20) had also recognized a cochlear projection to the DCN. Rasmussen (16) could detect no loss of endings in the DCN after cochlear ablation by inspection of sections, impregnated by his silver technique for synaptic endings. These findings were construed by Powell and Cowan (14) to indicate a total absence of a cochlear projection to the DCN. However, Rasmussen (18, 19) interpreted the findings to mean that the majority of endings in the DCN arises from autochthonous or from more centrally located neurons. Powell and Cowan (14), nevertheless, did show auditory fiber degeneration in the deep fusiform cell layer with the Nauta–Gygax technique. Osen (12), using the Nauta–Gygax, Fink-Heimer, and Glees methods, was loath to conclude that primary afferents are associated with fusiform cell bodies. Osen did, however, consider a possible association of primary afferents with the basal dendrites of fusiform cells.

The present results clearly demonstrate primary auditory nerve fibers at all levels of the fusiform cell layer, as well as the molecular layer of the DCN. Unlike previous investigations copious preterminal degeneration was frequently found around fusiform cell bodies, basal dendrites, and apical dendritic trunks. The rosettes of axonal degeneration among the granule cells suggest a special relationship between these cells and the primary afferents. Furthermore, fine degeneration was observed in the molecular region, where the more peripheral portions of the fusiform cell dendrites are located. In contrast to Osen (12), who reported no degeneration around giant cells, our study revealed both coarse and fine perisomatic and peri-

FIG. 2. Heavy preterminal degeneration of coarse-to-fine fibers in transverse sections of the deep layer of the DCN after an ipsilateral cochlear ablation. A. A perisomatic arrangement of preterminal degeneration occurs around small cells (e.g., S) and a giant cell (G). A possible relationship of degenerated axons with a granule cell (g) is also indicated. However, the dominant pattern of the degeneration would correspond to axodendritic relationships: fragments of coarse and fine axons outline dendritic trunks of the giant cell; clusters of coarse and fine preterminal degeneration occur throughout the neuropil (arrows). Four-day survival. ×384. B. Details of preterminal degeneration associated with giant cell (G) and small cell (S) bodies and axonal clusters (arrow). Four-day survival. ×629. C. After a 7-day survival preterminal degeneration of both coarse (x) and fine (arrow) axons occurs in relation to small cell bodies. Compared to a 2-day survival, there is much more fine degeneration, both preterminal and of passage. ×664. D. After a 2-day survival coarse preterminal degeneration predominates, especially in clusters (arrow), whereas fine preterminal degeneration is relatively sparse and diffuse. ×664.

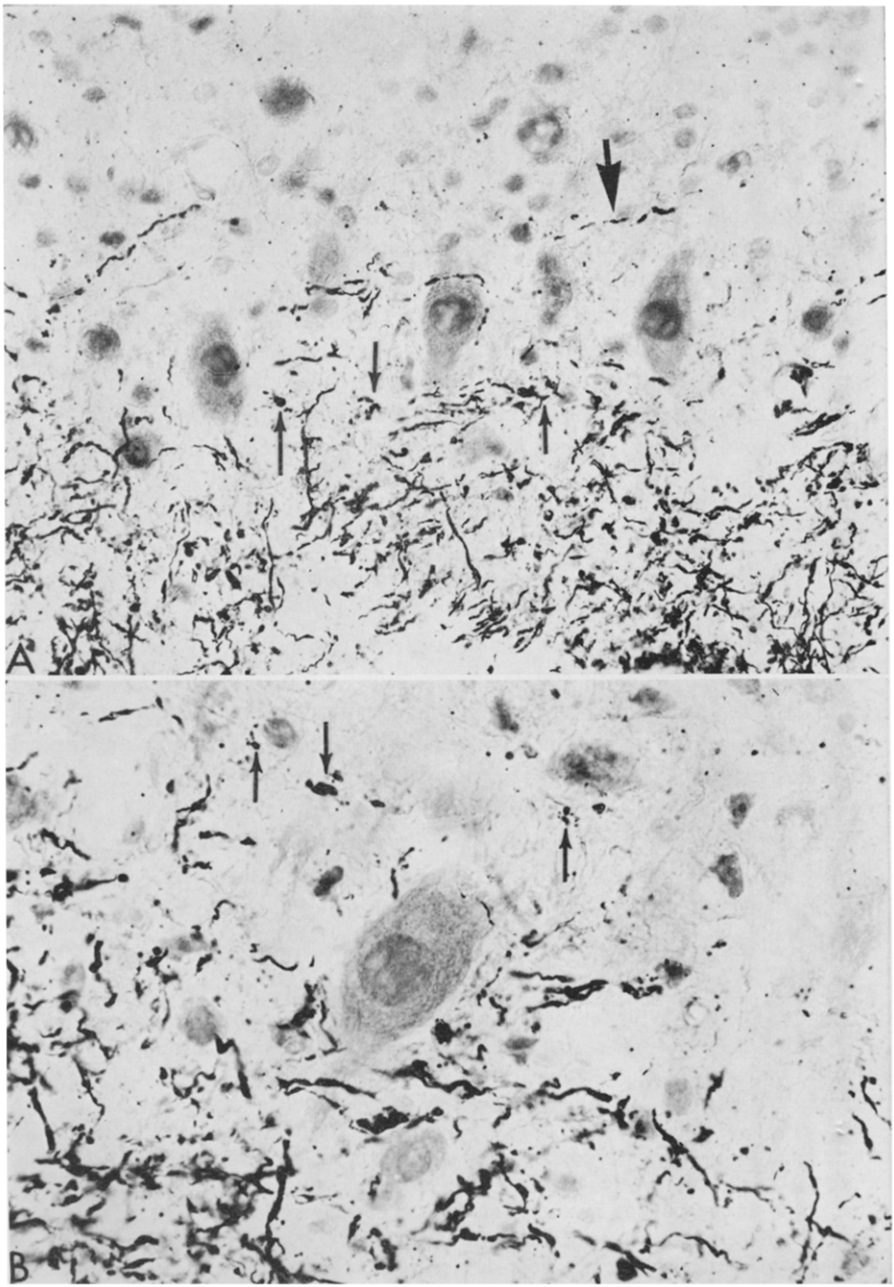

FIG. 3. Axonal degeneration in the fusiform cell layer 4 days after cochlear ablation. A. The fusiform cell bodies form a horizontal band across the middle of the field, above the heavy degeneration of the deep layer and below the sparse degeneration of

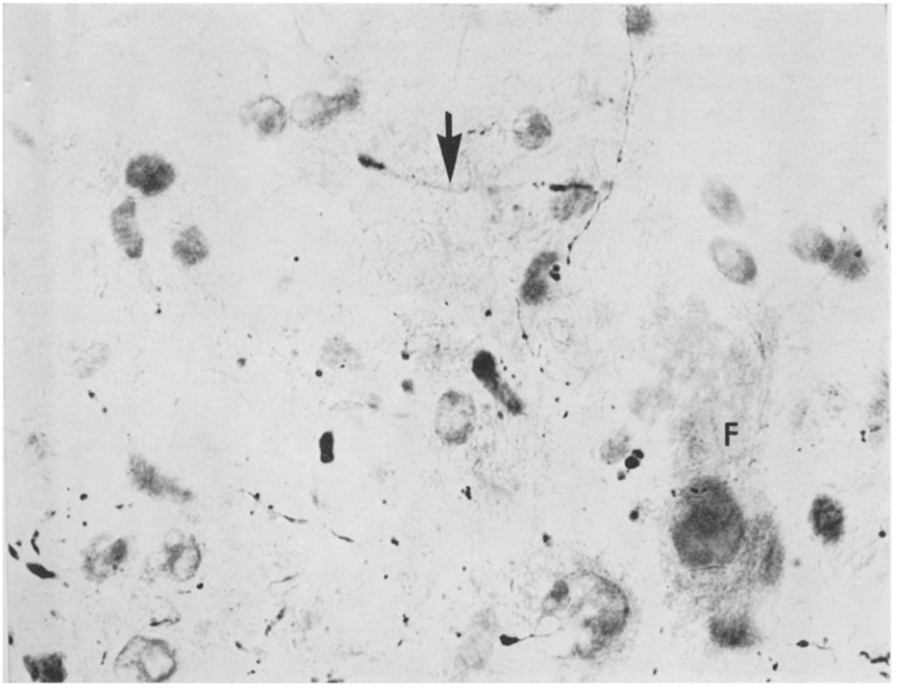

FIG. 4. In the molecular layer, 4 days after cochlear ablation, are a medium-sized degenerated axon (arrow), running perpendicular to the fusiform cell (F), and a fine degenerated axon, coursing vertically in the direction of the ependymal layer. ×744.

dendritic degeneration in relation to these cells. Moreover, the axonal clusters of the primary afferents in the neuropil of the deep layer probably reflect a special synaptic organization in this region.

Our findings suggest that the fusiform and the giant cells provide important links in the auditory pathway between the spiral ganglion, on the one hand, and the posterior colliculus, on the other. For the latter structure probably receives projections via the dorsal acoustic stria from the fusiform and possibly the giant cells (10, 12, 21, and Warr, unpublished). Primary

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the molecular layer. Many of the coarse fibers of passage of the deep layer are directed vertically to the fusiform cell layer, where moderate coarse-to-fine degeneration occurs in association with fusiform cells. Medium-sized degenerated fibers just above the fusiform cell bodies are oriented perpendicular to the apical dendritic trunks (thick arrow). Fine axonal degeneration is diffusely scattered in the fusiform and molecular layers. In the fusiform cell layer preterminal degeneration often gathers in very small, discrete clumps, or rosettes (thin arrows), among the granule cells. ×335. B. Fine and coarse axonal fragments appear to associate with a fusiform cell in the center of the field. Preterminal rosettes also occur (arrows). ×800.

afferent endings on the perikarya and dendrites of the fusiform and giant cells could provide a basis for direct influences of the DCN on the midbrain auditory centers, some of which, in turn, project back upon the DCN (2, 7, 17). However, the transformations of auditory signals in the DCN might well involve a more complicated pattern than that of the primarylike units of the anteroventral cochlear nucleus or of the auditory nerve (1, 3, 4). The unit discharge patterns in the anteroventral cochlear nucleus that resemble those of the auditory nerve apparently reflect the dominant activity of the large axosomatic end-bulbs of Held in this region (13). But the preponderance in the DCN of synaptic endings, having central or intrinsic origins (12, 19), suggests a less secure relationship of the secondary auditory neurons to the primary auditory input. Electron microscopic studies (Cohen, in preparation) should define the precise locations of the synaptic endings on the different cell types and in the axonal clusters and rosettes of the DCN.

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