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Ultrastructural Changes to the Cochlea Resulting from Impulse Noise*

N. Slepecky, R. Hamernik[1], D. Henderson[1], and D. Coling[2]

Department of Otolaryngology and Communication Sciences, State University of New York, Upstate Medical Center, Room 89, Weiskotten Hall, Syracuse, NY 13210, USA, and Department of Mechanical and Aerospace Engineering, Syracuse University, Syracuse NY, USA

Summary. Following impulse noise trauma to chinchillas, observation of plastic-embedded surface preparations of the organ of Corti showed no consistent relationship between cochlear hair cell loss and permanent hearing loss (Hamernik et al. 1980). In some animals there was a loss of hearing when hair cells were present. The cochleas from that experiment were examined with transmission electron microscopy to determine at the ultrastructural level if there was damage to the sensory cells that would explain the change in threshold sensitivity.

Ultrastructural changes in cochlear hair cells include an increase in lysosomes, multivesicular bodies, vacuolization of subsurface cisternae, and proliferation of Hensen bodies. These changes are observed in all experimental animals. Alterations to the ultrastructure of the stereocilia vary from animal to animal and on the outer hair cells, the changes include loosening of the stereocilia membranes, loss of stiffness, fusion of the stereocilia and disintegration of the rootlets. These changes are observed only in animals that have a permanent threshold shift after noise trauma.

Key words: Acoustic trauma – Cochlear pathology – Hair cell – Stereocilia

Introduction

In an earlier experiment designed to assess the effects of impulse noise on hearing (Hamernik et al. 1980), plastic-embedded surface preparations of the

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1 *Present address:* University of Texas at Dallas, Callier Center for Communication Disorders, 1966 Inwood Road, Dallas, TX 75235, USA

2 *Present address:* Bruel and Kjaer Inst. Inc., 185 Forest Street, Marlborough, MA 91752, USA

Offprint requests to: N. Slepecky (address see above)

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organ of Corti were observed in the light microscope. In several chinchillas, there was no consistent relationship between hair cell loss and permanent hearing loss. In some animals there was a loss of hearing without a significant loss of hair cells. Since the information obtained with the light microscope did not explain the changes in hearing thresholds, these plastic embedded cochleas were sectioned for observation with the transmission electron microscope. We were interested to see whether there was damage to the sensory cells at the ultrastructural level that might explain the change in threshold sensitivity.

Methods

Four chinchillas were exposed to impulse noise from a compressed air driven source. Fifty impulses were presented, one per minute at an intensity of 155 dB peak sound pressure level. Hearing was measured before and after exposure with the auditory evoked response. The noise exposure and methods for measuring hearing thresholds have been described in detail by Hamernik et al. (1980). The cochleas were perfused with 1% osmium tetroxide in Zetterqvist veronal acetate buffer, pH 7.0, and prepared as surface preparations for examination with the light microscope (Bohne 1972). Specimens from a point along the basilar membrane 35–40% of the distance from the apex were selected for detailed electron microscopic analysis. Although hair cells were present in this region of the cochlea, the animals differed in the extent of hearing loss.

Results

The cochleograms for the experimental animals are shown in Fig. 1. Final audiograms taken 30 days after the exposure to the impulse noise, just prior to perfusion of the cochlea, are superimposed on the cochleograms according to the place-frequency map for the chinchilla (Eldridge et al. 1977).

In the basal half of the cochlea, there are areas that show hair cell loss, and these areas correspond with a loss of hearing. In the apical half, there is less damage and hearing loss is more variable. There are areas where hearing thresholds are worse than would be expected from the cochleograms. In one animal (chin. 620) there is hearing loss in the absence of any significant hair cell loss throughout the cochlea. In all cases, the remaining hair cells and supporting cells appear normal when viewed by light microscopy.

When sections of sensory hair cells are examined by transmission electron microscopy, signs of possible sensory cell pathology are present in hair cell somata from all four cochleas sectioned. The inner hair cells display only an increase in lysosomes and multivesicular bodies (Fig. 2a). In all cells examined the synaptic region of the inner hair cells appears normal, with synaptic bodies and afferent nerve terminals present (Fig. 2b). Signs of possible sensory cell pathology in outer hair cells include vacuolization of subsurface cisternae and an increase in lysosomes, multivesicular bodies and Hensen bodies (Fig. 2c).

Only three of the four experimental animals (chin. 622, 611, and 620) show alterations in hair cell stereocilia. Stereocilia of the normal chochlea appear

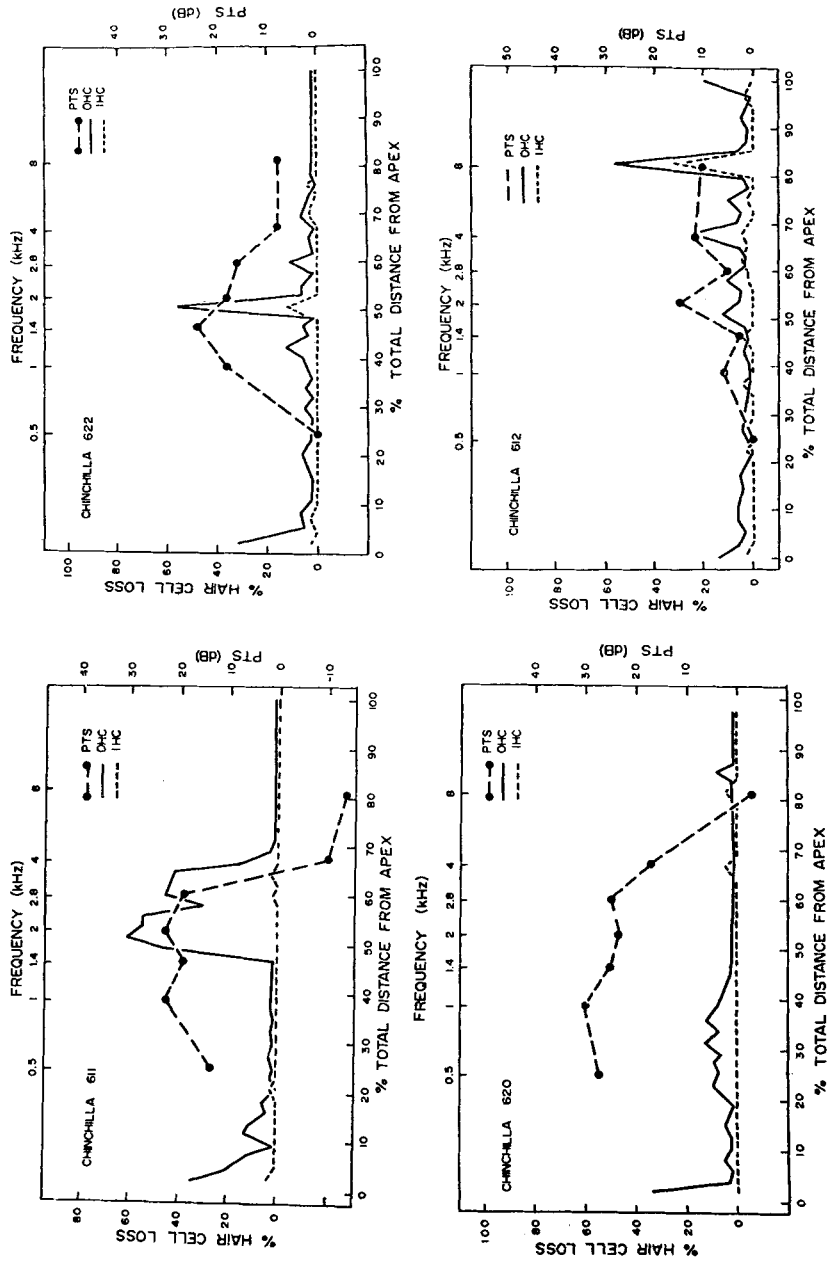
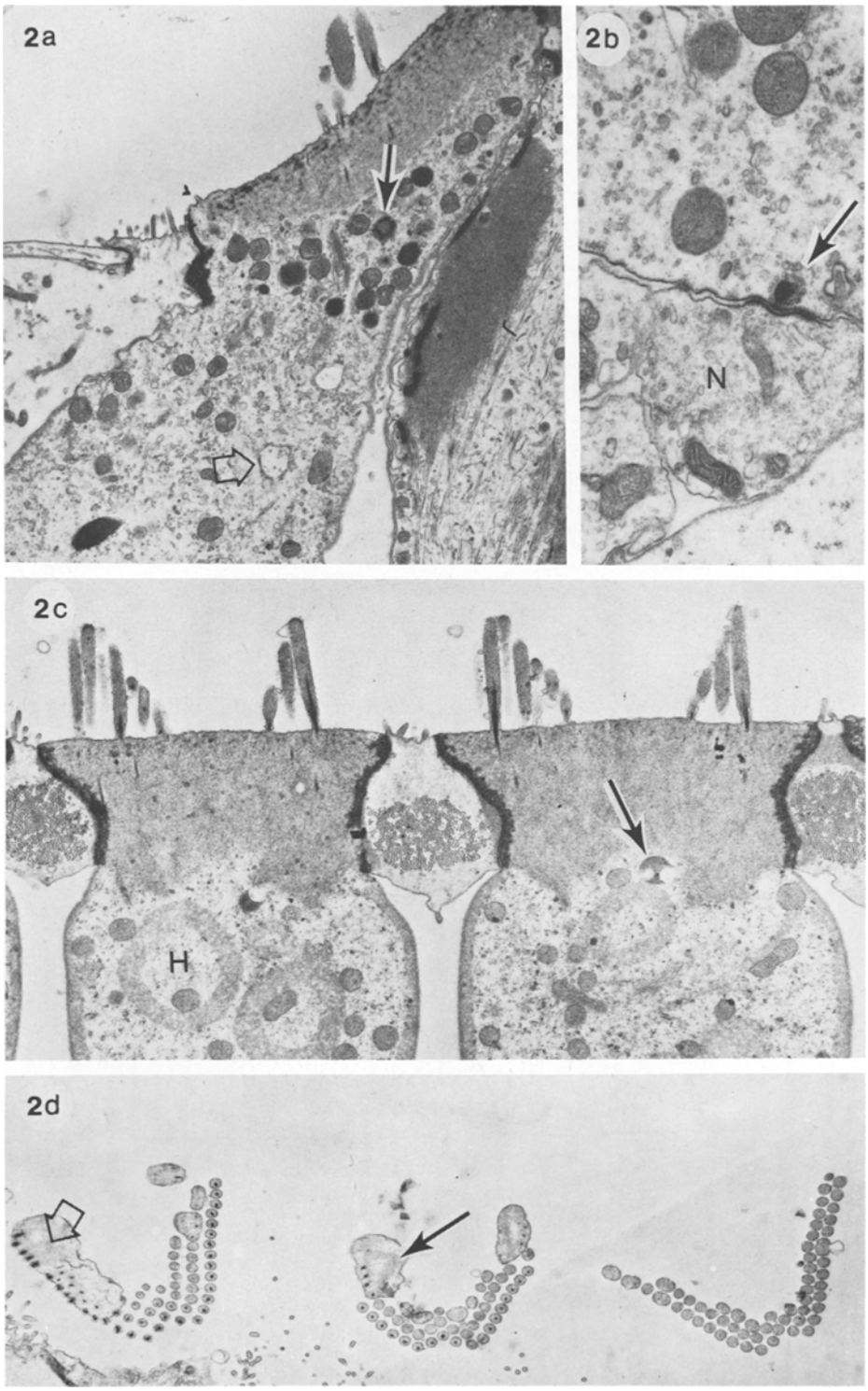


Fig. 1. Cochleograms and auditory evoked response audiograms from chinchillas exposed to impulse noise. Percentage inner (– – –) and outer (———) hair cell loss is plotted as a function of percent total distance from the apex. Hearing loss (——) is expressed as permanent threshold shift in decibels


2a
2b
N
2c
H
2d

erect with the membrane closely apposed to the stereocilia. Changes to the outer hair cell stereocilia include a loosening and wrinkling of the stereocilia membrane, loss of stiffness, fusion to form giant stereocilia and disintegration of the dark staining rootlets (Fig. 2d). It is of interest that the stereocilia are damaged only in the three experimental animals that show changes in threshold sensitivity after impulse noise.

Discussion

There have been several reports of ultrastructural damage to the cochlea following exposure to noise (Spoendlin 1971; Lim and Melnick 1974). Moreover, other investigators have reported a loss of hearing in the presence of hair cells that appear normal at the light-microscopic level (Ades et al. 1974; Hunter-Duvar and Bredberg 1974). We have observed that after impulse noise trauma there are areas of the cochlea where hearing thresholds are altered in the absence of any significant hair cell loss (Hamernik et al. 1980). We now show that in these regions of the cochlea where hair cells are present and appear normal, there is damage to the sensory cells observed at the ultrastructural level which could account for the change in threshold sensitivity. Signs of cochlear pathology include an increase in lysosomes, multivesicular bodies, vaculization, and disorganization of subsurface cisternae and proliferation of Hensen bodies. However, in the region of the cochleas sectioned, there are similar changes in the ultrastructure of the hair cell somata in all experimental animals while the amount of threshold shift varies from 0–30 dB. Thus, hearing loss cannot be explained by these changes alone.

Only three of the four experimental animals show ultrastructural changes to the stereocilia. There are alterations to the cell membrane which result in loosening and wrinkling of the membrane surrounding the stereocilia as well as fusion to form giant stereocilia. There are also changes to the actin filaments within the stereocilia as reflected in the loss of stiffness and in the disappearance of the rootlets.

The results presented in this paper demonstrate that after impulse noise, although the remaining hair cells appear normal by light microscopy, there are signs of cochlear pathology at the ultrastructural level, and that noise induced hearing loss may be associated only with changes to the stereocilia.

Fig. 2. Electron micrographs of cochlear hair cells from animals exposed to impulse noise. **a** A radial section through the apical region of an inner hair cell. There is an increase in lysosomes (*closed arrow*) and multivesicular bodies (*open arrow*). **b** A radial section through the synaptic region of an inner hair cell. The synaptic body (*arrow*) is present, and the afferent nerve terminal (N) is normal. **c** A section perpendicular to the reticular lamina, tangential to the organ of Corti. Outer hair cells show an increase in lysosomes (*arrow*) and Hensen bodies (H). **d** A section parallel to the reticular lamina through stereocilia of first row outer hair cells. There is fusion to form giant stereocilia (*closed arrow*) and the rootlets have disappeared (*open arrow*)

References

Ades HW, Trahiotis C, Kokko-Cunningham A, Averbuch A (1974) Comparison of hearing thresholds and morphological changes in the chinchilla after exposure to 4 kHz tones. Acta Otolaryngol (Stockh) 78: 192

Bohne BA (1972) Location of small cochlear lesions by phase contrast microscopy prior to thin sectioning. Laryngoscope 82: 1

Eldridge DH, Mills JH, Bohne BA, Clark WW (1977) Frequency-position maps for the chinchilla cochlea. J Acoust Soc Am 62: S35

Hamernik RP, Henderson D, Coling D, Slepecky N (1980) The interaction of whole body vibration and impulse noise. J Acoust Soc Am 67: 928

Hunter-Duvar IM, Bredberg G (1974) Effects of intense auditory stimulation: hearing losses and inner ear changes in the chinchilla. J Acoust Soc Am 55: 795

Lim DJ, Melnick W (1974) Acoustic damage of the cochlea. Arch Otolaryngol 94: 294

Spoendlin HH (1971) Primary ultrastructural changes in the organ of Corti after acoustic overstimulation. Acta Otolaryngol (Stockh) 71: 166

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