Ultrastructure of the Normal, Drug Damaged and Sound Damaged Cochlea

Ivan M. Hunter-Duvar, Ph. D.
Professor and Director Otology Research Lab.
Department of Otolaryngology
The Hospital for Sick Children
Toronto, Ontario MSG 1X8
Canada

Normal Cochlea

The true appearance of the ultrastructure of the normal cochlea is still an unknown entity and shall likely remain so. This is due to its inaccessibility and the necessity of distorting it with processes such as fixation, decalcification, drying, etc. required to prepare the tissue for the instruments used to observe it. Differences in preparation processes probably account for many of the unresolved differences in opinion that exist regarding cochlear ultrastructure.

The cochlea is composed of many different types of cells with functions not fully known but often surmised from either their structure or their location relative to theories of how the cochlea works. The scanning and transmission electron microscopes allow us to look at the ultrastructure of these cells in great detail after they suffer the preparation distortions already discussed. This paper will detail some of the opinions obtained from SEM and TEM study of the chinchilla cochlea.

Similarity can be found in the surface areas of both the mesothelial cells on the outer surface of Reissners membrane and the mesothelial cells of the tympanic layer which covers the under surface of the basilar membrane. Both groups interface with perilymph and their rough surfaces, with few microvilli and large bulges from cell nuclei, contrast with the microvilli covered surfaces of the cells which interface with the endolymph of the scala media. (Fig. 1) Surface areas of Claudius cells, dark cells of the stria vascularis and endothelial cells of Reissners membrane all appear similar under the SEM although they probably perform quite different functions. Although their surface areas look similar, the Claudius cells with round nuclei and interiors almost completely devoid of organelles contrast strongly with the cells of the stria vascularis with their large lobulated nuclei and their dense packing of mitochondria. We have previously shown that the endothelial cells of Reissners membrane which are presumed to have an ion filtering function are capable of removing from the endolymph, by ingesting it, the debris from organ of Corti damage. (Fig. 1)

In recent years the finding that almost all afferent innervation was to inner hair cells combined with the lack of neural connections between outer and inner hair cells has caused renewed interest in the tectorial membrane and its attachments. A firm attachment of outer hair cell stereocilia to the tectorial membrane has been demonstrated (Fig. 2) but no compelling evidence for an attachment of inner hair cells has appeared. The existence and location of Hensen’s stripe would suggest some interaction between it and inner hair cell stereocilia and speculation has it intermittently touching inner hair cell stereocilia or directing a flow of fluid against them but its true functional significance remains unknown.

Researchers have suggested an attachment of the tectorial membrane to the Hensen-Deiters cell area lateral to the outer hair cells. Our finding with the adult chinchilla preparation indicated the outermost portion of the randfasernetz or marginal net is firmly attached to the stereocilia of the outermost hair cells. (Fig. 3) This attachment of the fibrous part of the tectorial membrane does not preclude an additional lateral attachment by a covering gel, however, any separation of fluids by the tectorial membrane, required for some theories, would necessitate special properties similar to those recently proposed by Steel.

Findings of actin in the stereocilia and the cuticular plates of hair cells allows postulation of control of organ of Corti movement through the firm attachment of outer hair cells to the tectorial membrane. This mechanical concept considerably eases the theoretical pressure from the lack of synaptic contact between nerve fibers of outer and inner hair cells.

Sound Damage

A growing body of data indicates that one of the first signs of acoustic overstimulation is disruption of actin structure leading to a distortion of hair cell stereocilia. (Fig. 4) Some question exists as to the ability of the stereocilia to recover from distortion. We have suggested that there is a point at which stimulation can be withdrawn and stereocilia which have collapsed
can recover their upright position. This conclusion is based on our finding that the collapsed stereocilia seen immediately after overstimulation are not found at later examination times when the picture is one of erect stereocilia, missing stereocilia or fused stereocilia. We suggest the missing or fused stereocilia are the results of not withdrawing the stimulation in time to allow actin recovery. We suspect that the dislocation of erect stereocilia after acoustic stimulation (Fig. 5) reported by Tilney in the lizard and Engstrom et al in the rabbit may have an explanation in terms of the stereocilia having collapsed and later regaining an erect position.

Fusion of stereocilia from acoustic overstimulation may be accompanied by deterioration of the hair cell's inner structure. With continued overstimulation the fused stereocilia appear to autolize (Fig. 6) and the contents of the hair cell are ejected out into the scala media. After a period of hours cuticular plates of partially damaged hair cells, sometimes with nearly intact stereocilia, may be ejected from the organ of Corti in the damaged area as it stabilizes after overstimulation.

We have seen no evidence of leakage between organ of Corti fluid spaces and scala media in the initial stages of damage from acoustic trauma. In cases of severe trauma from either very intense stimulation or prolonged stimulation we feel such leakage must occur.

In the chinchilla the order of sensitivity of hair cells to acoustic trauma is usually well defined with first row outer hair cells being the first damaged. These are generally followed in order by second and third row outer cells with inner hair cells appearing the most resistant to damage. It may be that this pattern is variable with the sound stimulus and with species. In older, unstimulated cochleas the most missing hair cells are often seen in the third row of outer hair cells. On occasion we have noted that the damage pattern to the stereocilia of the first outer row of hair cells gives the appearance of the result of a collision between the stereocilia of adjacent cells. The first row cells differ from second and third row cells in that they are separated by processes of the outer pillar cells rather than processes of Deiters cells.

Drug Damage

The chinchilla shows the same pattern of hair cell loss from the ototoxic antibiotics as originally reported for the guinea pig. Basal hair cells deteriorate in the order of first, second, third row outer and then inner hair cells. Loss in continued exposure moves from base to apex with apical inner hair cells being the last survivors. Large populations of anatomically undamaged inner hair cells may remain in the total absence of outer hair cells. In such areas, organ of Corti structure and fluid spaces may remain relatively intact with Deiters cells filling the void left by the lost outer hair cells. With continued exposure to the ototoxic drug, the deterioration of inner hair cells leads to the demise of the supporting cells. This is followed by the disappearance of the dendrites leaving only a basilar membrane covered with ancillary cells.

REFERENCES

11) Tilney, L. G., Saunders, J. C., Egelman, E. and DeRosier, D. J. (1982): Changes in the organization of actin filaments in
the stereocilia of noise-damaged lizard cochleas. Hearing Res. 7, 181-197.

Figure 1 TEM micrograph of Reissners membrane showing the bulge in the thin perilymphatic layer cells caused by the cell nuclei. Debris from a damaged organ of Corti is seen on the endolymphatic layer cell where it is ingested. Picture Width (PW) = 24 μM

Figure 2 Underside of tectorial membrane shows "W" imprints from attachment of outer hair cell stereocilia. Hensen's stripe is an imposing structure at top of micrograph. PW = 45 μM

Figure 3 Stereocilia of third row outer hair cells have pulled out of poorly fixed cochlea. They are seen in their place of normal attachment to outermost part of marginal net of tectorial membrane. PW = 75 μM

Figure 4 Stereocilia of inner hair cells have lost their rigidity after acoustic stimulation. PW = 7 μM
Figure 5  Arrows indicate the fracture in the root of an erect stereocilia in rabbit cochlea following acoustic overstimulation. (From Engstrom et al., reference 1) PW = 0.6 μM

Figure 6  Adjacent outer hair cells from a sound damaged cochlea show two stages of deterioration. Right hair cell shows fused stereocilia. Left hair cell shows autolysis process which follows. PW = 10 μM

Figure 7  Area from middle turn of chinchilla treated with dihydrostreptomycin. Inner hair cells appear anatomically intact. All outer hair cells are destroyed. PW = 100 μM

Figure 8  TEM micrograph of thin section cut through area shown in Fig. 7. Fluid spaces appear relatively normal. PW = 220 μM