A SCANNING ELECTRON MICROSCOPIC AND ELECTROPHYSIOLOGICAL INVESTIGATION OF EXPERIMENTAL ACOUSTIC TRAUMA

VOLUME 1

by

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ABSTRACT

Investigations were undertaken to describe and quantitate the topographical abnormalities which develop in the organ of Corti as a result of acoustic trauma, and to determine their relationship to the associated losses of cochlear function assessed by the compound action potential (N_1) and cochlear microphonics (CM).

Thirty guinea pigs were exposed, while anaesthetised, to a tone of 3 KHz at 125 dB SPL for 30 minutes. Both organs of Corti were examined by scanning electron microscopy either immediately or 1,3,7 or 14 days after exposure. In the second study 26 anaesthetised guinea pigs were exposed to 5 KHz at 125 dB SPL for 30 minutes. Cochlear potentials were recorded from the round window of the right cochlea and N_1 audiograms (sound pressure level required to elicit N_1 plotted against frequency) and 10 μv isopotential curves (sound pressure level required to produce 10 μv CM plotted against frequency) were produced for each animal either within one hour or 1,7,14 or 28 days later. The same organ of Corti was examined by scanning electron microscopy. Cochlear potentials were compared to mean values from 16 normal guinea pigs.

Most animals developed topographical changes in one or both organs of Corti after exposure to 3 KHz (90%) and in the right organ of Corti after 5 KHz (92%). There

was a wide variation in both the length (3 KHz: 0.1-4.15 mm; 5 KHz: 0.5-16.0 mm) of lesions and the number of hair cells affected. Both these indices of damage increased significantly ($p < 0.01$) in the 24 hours following exposure to 5 KHz. Early damage to hair cells included either collapse, fusion or loss of stereocilia and there was an increase in the proportion of affected cells towards the centre of the lesions where supporting cells were damaged also. Subsequent to exposure, collapsed stereocilia appeared to become fused and some hair cells, particularly OHC, with stereocilia abnormalities were lost. However, others, particularly IHC, remained for up to 28 days despite abnormal stereocilia. Early changes occurred around the position of maximum displacement of the basilar membrane and subsequent extension of the lesions occurred equally in both apical and basal directions. Fusion was the only stereocilia change to develop after exposure. Regions of the organ of Corti showing supporting cell damage were replaced within 3-7 days by the proliferation of inner sulcus and Claudius' cells.

The substantial initial loss of both N_1 thresholds and CM sensitivity partially recovered during the first 24 hours after exposure, but paradoxically, this was associated with the significant increase in the topographical changes in the organ of Corti. N_1 threshold loss occurred over all frequencies and was maximum ½ - 1

octave higher than the exposure frequency (5 KHz). All lesions occurred within regions corresponding to changes in N_1 thresholds. In the first 24 hours topographical changes occurred over a much smaller area of the organ of Corti than indicated by changes in N_1 thresholds. Seven or more days later the longer lesions (30%) reflected the extent of changes in N_1 but the remainder were smaller than indicated by this functional loss. This suggests that functionally important damage to the cochlea is more extensive than indicated by hair cell loss, stereocilia abnormality or supporting cell changes in the organ of Corti. Therefore, investigations of the effects of noise should not be based simply on topographical changes in the organ of Corti as these often underestimate the extent of injury to the cochlea.

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