The Effects of Short Noise Exposures in the Guinea Pig*

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Summary. Guinea pigs have been exposed to 20 kHz at 120 dB SPL for exposure durations of 7.5, 6.5, 5.0, and 3.25-min and killed either 3 or 12 weeks post-exposure. One series of guinea pigs exposed for 30-min had cochlear potentials recorded 3 weeks post-exposure. The damage was assessed by surface preparations and quantified as percentage hair cell loss per segment in every row, total number of outer, and inner hair cells missing and the area of total outer hair cell damage. A significantly smaller number of ears were found to be damaged after the shortest exposures, but no significant differences could be detected in the amounts of damage when all the series were compared. Myelinated nerve fibre degeneration had increased after the longer post-exposure interval, but no such differences were observed in the sensory hair cell degeneration.

Key words: Guinea pig – 20 kHz – Short exposures – Cochlear damage

Experimental studies using high-frequency pure-tone noise exposures at moderate intensities in the guinea pig have still been rather neglected. In 1968 Peterson [7] recorded cochlear microphonics at various time intervals after over-stimulation with high-frequencies and stated that individual susceptibility to damage varied considerably even when all the parameters were kept constant. Stockwell et al. [14] looked at permanent damage, but only up to 4 kHz, while Spoendlin [13] used various noise exposures, with narrow-band noise up to 8 kHz. Cody and Robertson [2] have looked at physiological/morphological correlates immediately after carefully controlled exposures using 10 kHz at intensities of 112–118 dB SPL. Goulis and Robertson [6] used the same frequency at various intensities and durations to look at the equal energy principle. More recently, Cody [1] used 5 kHz and 16 kHz separately and together. Simultaneous exposures seemed to indicate an acceleration in the recovery process, even in the chronic studies.

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Previously, guinea pigs have been exposed to 20 kHz at 120 dB SPL for varying periods and the cochleae examined after 3 or 12 weeks [8-11]. The initial exposure duration was 30-min and doubling this to 1 or 2 h did not increase the area of outer hair cell damage. Halving the exposure durations to 15 and 7.5-min resulted at times in a slightly smaller area of damage when examined after 3 weeks, but this difference was no longer apparent after 12 weeks. Myelinated nerve fibre degeneration, however, had increased considerably when viewed after the longer post-exposure interval.

The aim of the present series was to see whether by decreasing the exposure durations still further, the severity or the extent of the area of damage would be altered in any way. The sensory hair cell damage has also been quantified more accurately by counting destroyed hair cells onto cochleograms, in order to make better comparisons. In an additional series of 30-min exposures, recordings of cochlear microphonic and compound action potentials were carried out by Dr. Brown, prior to sacrifice of the animal. This series will be compared with the present results.

Methods

Six randomly selected Dunkin-Hartley albino guinea pigs, 5–6 weeks old, were used for each exposure series. The left ear only was exposed to 20 kHz at 120 dB SPL for exposure durations of 7.5 (repeat), 6.5, 5.0, and 3.25-min. The post-exposure interval remained at 3 or 12 weeks, except for 6.5 min series, where it was 16 weeks. The right ears were used as controls, together with non-experimental controls. Six comparable guinea pigs were exposed for 30-min and electrophysiological recordings carried out after 3 weeks.

The cochleae were fixed in 1.3% Veranol-buffered osmium tetroxide and the whole cochlea was micro-dissected. Surface preparations were viewed under phase and NDI contrast microscopy. The cellular membrane, cytoplasm, stereocilia, and nucleus were examined when assessing the state of a sensory hair cell. Cochleograms were prepared according to Coleman [3], who calculated the correct ratio of inner hair cells per 100 outer hair cells along the whole length of the cochlea. Since it proved difficult to obtain consistent and reliable counts of damaged hair cells in the round window region, this area has been excluded from statistical calculations. Thus the quantification onto cochleograms was started at point zero (0), which was the anterior tip of the round window membrane 2 mm from the very base. Cochleograms were divided into segments of 100 outer hair cell (OHC) lengths from point zero and percentage OHC and inner hair cell (IHC) loss calculated for each segment. Kruskall Wallis analysis of variance by ranks was used to compare damage in every cell row at every segment ($p = 0.05$). Total numbers of OHC and IHC lost and the area of total OHC loss were calculated by the Mann-Whitney U-test. Myelinated nerve fibre degeneration was quantified more grossly. Cochlear microphonic (CM) and compound action potentials (CAP) were recorded in anaesthetised animals, with the recording electrode being placed near the round window membrane [5]. The stimuli were tone pulses of 5 ms duration with 0.5 ms rise and fall times. The intensity was adjusted for a just detectable N1 response (32 averages) and for a 20 µV CM response on the frequency range 0.3–30 kHz. The maximum intensity available from the loudspeaker at 30 kHz was 70 dB SPL.

Results

OHC Degeneration

As reported previously [10] the sensory hair cell damage obtained from exposures to this frequency and intensity occurs in one main area. Generally, the