The Effects of Dexamethasone Following an Acute Acoustic Trauma

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Abstract: An acute acoustic trauma (AAT) results when the unprotected ear is exposed to very high sound pressure levels, usually as the result of an industrial or military accident. A study was performed to evaluate the effects of a corticosteroid (dexamethasone) on the hearing loss from a series of blast waves that would simulate an AAT. Groups of chinchillas were exposed individually to ten 160-dB peak SPL reverberant blast waves from a conventional shock tube at a rate of one blast per minute. Immediately following the exposure, the animals were injected with dexamethasone alone (1 - 2 mg/kg IV) or in combination with Dimethyl Sulfoxide (DMSO, 1 ml/kg). Groups of animals showed the well-known extreme variability in hearing losses as noted in earlier studies (1). The median permanent threshold shifts (PTS) in the various groups of animals showed a dose-dependent effect up to 16 dB, with increasing dosages of dexamethasone associated with lower levels of PTS in the frequency region most affected by the noise exposure.

INTRODUCTION

Acute acoustic trauma (AAT) occurs when the unprotected ear is exposed to a high-level noise as the result of an industrial or non-industrial accident. These noise transients (impulsive or noise bursts) can be in excess of 160 dB peak SPL, and occur most frequently as a result of equipment or material failure, explosion, incorrect operation of equipment, or failure to observe safety precautions. AATs also may be the result of proper deployment of vehicle air bags or direct blows to the ear such as might occur in sports accidents and assaults. As a result of the AAT, the cochlea is subjected to tremendous mechanical forces that cause initial (primary) structural and subsequent (secondary) metabolic injury to the delicate tissues of the inner ear.

This paper reports the effects of a potent anti-inflammatory agent (dexamethasone, a synthetic corticosteroid) with or without a free-radical scavenger [Dimethyl Sulfoxide (DMSO)] on the effects of an impulse noise exposure designed to mimic an AAT.

METHOD

The chinchilla was used as the animal model. Three groups of six animals for each experimental (drug-treatment) group were used. Each animal was prepared for auditory evoked potential (AEP) testing by surgically destroying the left cochlea and implanting EEG electrodes into the inferior colliculus. Animals were allowed to recover for at least two weeks before additional procedures.

Following recovery, each animal’s pure-tone hearing thresholds were measured using the AEP at the octave frequencies from 0.5 to 16.0 kHz. The test stimulus was a 20 ms pure tone burst with 5 ms rise fall times, presented at a rate of 10 bursts per second. Raw EEGs collected for 25 ms at 48,000 samples per second following the onset of the tone burst were averaged over 250 burst presentations. Thresholds were determined by recording average AEPs over a 30-dB range in 5-dB steps. The control of the stimulus presentation and response collection was performed using an Apple Quadra 950 computer with National Instruments NB-2100 audio board, NB-DMA2800 direct memory access controller, and NB-DSP-2300 digital signal processing board. After the average AEPs were collected and stored for the six intensity series, the seven average AEP waveforms in each intensity series were displayed and a visual determination of threshold was made. Threshold was determined to be 2.5 dB (one-half step) below the first averaged AEP waveform that showed a “response” consistent (e.g., amplitude growth and latency shifts) with the responses at higher intensities.

The average of three threshold determinations at each test frequency collected over at least two days determined the animal’s preexposure thresholds (referred to as the preexposure AEP audiogram). A threshold determination was accepted if the standard deviation of the three measures was not more than 5 dB.

Following determination of thresholds, each animal was exposed individually to ten reverberant impulses from a conventional shock tube [Ahroon et al. (2)] at a rate of one impulse per minute. The impulses were calibrated in a reverberant chamber to a level of 160 dB peak SPL. The frequency spectrum and pressure-time waveform of one of these impulses is displayed in Figure 1. Additional details of the experimental procedure and exposure paradigm may be found in Ahroon et al. (2).
Immediately following the exposure to the 10 impulses, each animal was injected (I.V. pinna) with a 1.0 or 2.0 mg/kg solution of dexamethasone. A third group of animals was given the 2.0 mg/kg injection of dexamethasone in conjunction with 1.0 ml/kg Dimethyl Sulfoxide (DMSO). A fourth group of 15 animals was not given any drug and served as a no-drug control group.

Thirty days following impulse noise exposure, AEP thresholds were determined again and the difference between the 30d post-exposure and the pre-exposure measures defined the permanent threshold shift (PTS).

RESULTS AND DISCUSSION

Because of the often-observed large variability in hearing loss following impulse noise exposures of this type (1,2), median threshold shifts were evaluated. The effect of the drug was determined using the Student t distribution. Figure 2(a) displays the median PTS for the no-drug control group and the two groups of animals administered 1.0 or 2.0 mg/kg dexamethasone and Figure 2(b) displays the median PTS for the no-drug control group and the group administered 2.0 mg/kg dexamethasone along with a 1.0 ml/kg treatment of DMSO.

It is apparent from this figure that dexamethasone exerts a dose-dependent effect on the PTS resulting from an exposure to an acute acoustic trauma. The addition of DMSO to the drug therapy improves the therapy slightly and shows an over 16 dB reduction in PTS as a result of the AAT.

CONCLUSION

If drug regimes are capable of reducing hearing loss as a result of a simulated AAT in chinchillas, then there are clear implications for improved outcome in the hearing of human victims of an AAT. A clinical protocol for treating victims of AATs may significantly improve quality of life and disability status of such patients.

REFERENCES