Increased DPOAE levels following high level noise exposure: a case study using DPOAE level/phase mapping

D. Meinke[1], O. Clavier[2], J. Norris[2], R. Kline-Schoder[2], J. Buckey[3]

[1] University of Northern Colorado – Audiology and Speech-Language Hearing Sciences, Campus Box 140, Greeley, CO, 80639, Deanna.Meinke@unco.edu

[2] Creare Inc., 16 Great Hollow Road, Hanover, NH 03755-3116 ohc@creare.com

[3] Dartmouth Medical School, One Medical Center Drive, Lebanon, NH 03756, jay.buckey@dartmouth.edu

ABSTRACT

Distortion product otoacoustic emission (DPOAE) testing shows promise for detecting noise-induced hearing loss early and monitoring cochlear status. A reduction in DPOAE levels is commonly associated with cochlear damage from hazardous noise; however, some studies have reported sporadic instances of increased DPOAE levels in noise exposed subjects. We studied one such normal hearing individual before and after exposure to hazardous occupational noise (400 % noise dose) using a series of DPOAE level/phase (LP) maps based upon techniques first reported by Knight & Kemp (2001). The unique characteristics of this subject's LP maps will be described, specifically highlighting increased $2f_1$-f_2 and $2f_2$-f_1 DPOAE levels. These findings exemplify that DPOAE changes are complex and more research is needed before DPOAE measurements can be simply implemented into hearing loss prevention programs. Additionally, these data suggest that DPOAE L/P mapping may prove useful for expanding our diagnostic and monitoring abilities, as well as providing a better understanding of the mechanisms of cochlear damage from noise. Work supported by ONR Grant N00014-09-1-0859.

INTRODUCTION

Otoacoustic emissions (OAEs) are low-level sounds produced by the cochlea's active amplifier in outer hair cells (OHCs) and reverse transmitted through the middle ear into the external auditory canal, where they can be detected by a sensitive microphone (Kemp 1978). These sounds occur spontaneously in the majority of normal ears, and they can be deliberately evoked by presenting an acoustic stimulus to the ear. OAEs are diminished, and can disappear entirely, in instances of sensory hearing loss (Harris 1990; Gorga et al. 1999).

One particular type of evoked OAE is called the distortion-product otoacoustic emission (DPOAE). This class of evoked emission is elicited by the simultaneous presentation of two pure-tone stimuli. DPOAEs are particularly appealing as a clinical measure because the two tones, termed f_1 (lower frequency) and f_2 (higher frequency), can be swept in frequency and amplitude to form a DP-gram, in a manner very similar to procedures routinely used to obtain a clinical hearing test (audiogram). The DP-grams can then be related to the subjective hearing capability of the same individual (Gorga et al. 1999).

Due to the non-linear amplification mechanism of the cochlea, the presentation of two pure-tones to the ear actually results in a family of distortion-product emissions of which the $2f_1$-f_2 and the $2f_2$-f_1 DPOAEs (Figure 1) are the most prominent members (Brown & Kemp 1985; Lonsbury-Martin et al. 1987). In humans, the $2f_1$-f_2 emission has the highest absolute magnitude and the $2f_2$-f_1 the next highest. Both of these

Therefore, changes in outer hair cell electromotility have been explored with OAEs. It is possible that OAEs may be more sensitive to early signs of noise-induced hearing loss (NIHL) and may be useful in determining susceptibility to NIHL (Lapsley Miller & Marshall 2007). Similar to pure-tone testing, DPOAEs can be used to detect a temporary vs. a permanent change (shift) in cochlear status as a consequence of hazardous noise exposure. Both a temporary emission shift (TES) and a permanent emissions shift (PES) can be measured relative to a baseline measurement when the individual serves as their own control and when DPOAEs are measured before and after noise exposure (Attias & Bresloff 1996). In a laboratory environment, the TES time-course for recovery qualitatively resembles the temporary threshold shift (TTS) recovery functions (Rossi et al. 1991; Sutton et al. 1994).

A reduction in measured DPOAE levels is commonly associated with cochlear damage from hazardous noise; however, some researchers have reported sporadic instances of increased DPOAE levels in noise exposed subjects (Lonsbury-Martin & Martin 1990; Avan et al. 1996). Martin et al. (2005) performed DPOAE LP mapping on rabbits and noted that noise-damaged rabbit ears show more vertical phase banding at narrow ratios when compared to the non-exposed control ear. More recently, we have studied one such normal hearing individual before and after exposure to hazardous occupational noise (400 % noise dose) using a series of DPOAE level/phase (LP) maps.

OBJECTIVE

This single case study was collected as part of a larger study to determine the effects of hazardous noise exposure on various distortion-product measures of cochlear function. Specifically, we are measuring DPOAE LP maps in three experimental groups: normal-hearing ears without noise exposure; normal-hearing ears with a positive history of noise exposure; and high-frequency hearing-impaired ears using both a cross-sectional and longitudinal study design. This case study subject comes from the normal-hearing, noise-exposed experimental group.

METHODS

Pure-tone hearing tests were obtained using a GSI 16 audiometer calibrated to American National Standards Institute (ANSI) S3.6 – 2004 and conducted in a single-walled Interacoustics sound booth meeting ANSI S3.1-1999 (R2008; 2003) permissible ambient noise level requirements. Tympanometry screening at 226 Hz was conducted with an EROScan (Etymotic Research) system. Both audiometry and tympanometry screenings were conducted prior to DPOAE mapping.

DPOAE LP maps were obtained with the prototype Creare Hearing Assessment system (Creare, Inc., Hanover, NH). The system includes an Etymotic 10B+ probe with ER-2 speakers (Elk Grove, IL), a USB data acquisition card (Data Translation, DT9841E), and custom software to execute the DPOAE tests via a laptop PC. The probe's microphone amplifier was set to provide a sensitivity of 0.5 V/Pa. For DPOAE measurements, the USB data acquisition card drives the speakers and synchronizes recording with the microphone signal at 44.1 kHz with 24-bit resolution. Data from the tests were stored in a Microsoft Access database and additional post-processing analysis was performed in MatLab.

DPOAEs were measured in DPOAE frequency steps of approximately 44 Hz (0.5-6.0 kHz) in response to primary-tone sweeps at two levels; 65,55- and 75,75 dB SPL us-

DPOAE Level Maps

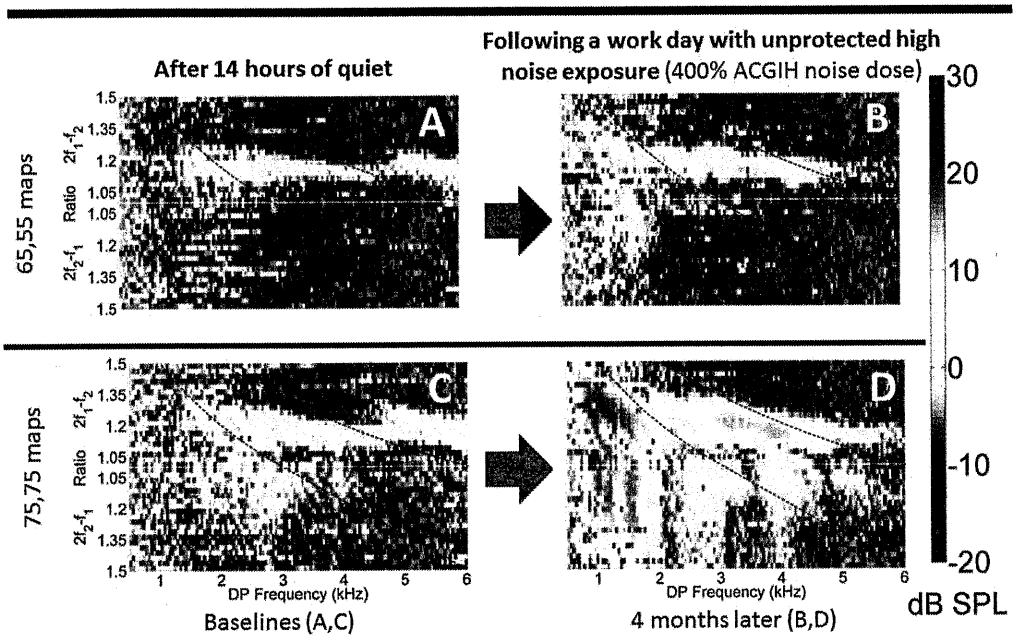

Figure 2: DPOAE level maps obtained 4 months apart in one male subject. Baseline (A,C) and re-test after noise exposure (B,D) obtained at two stimulus levels 65,55 dB SPL (A,B) and 75,75 (C,D). Dotted lines represent f_2 at 3 kHz and 6 kHz

DPOAE Phase Maps

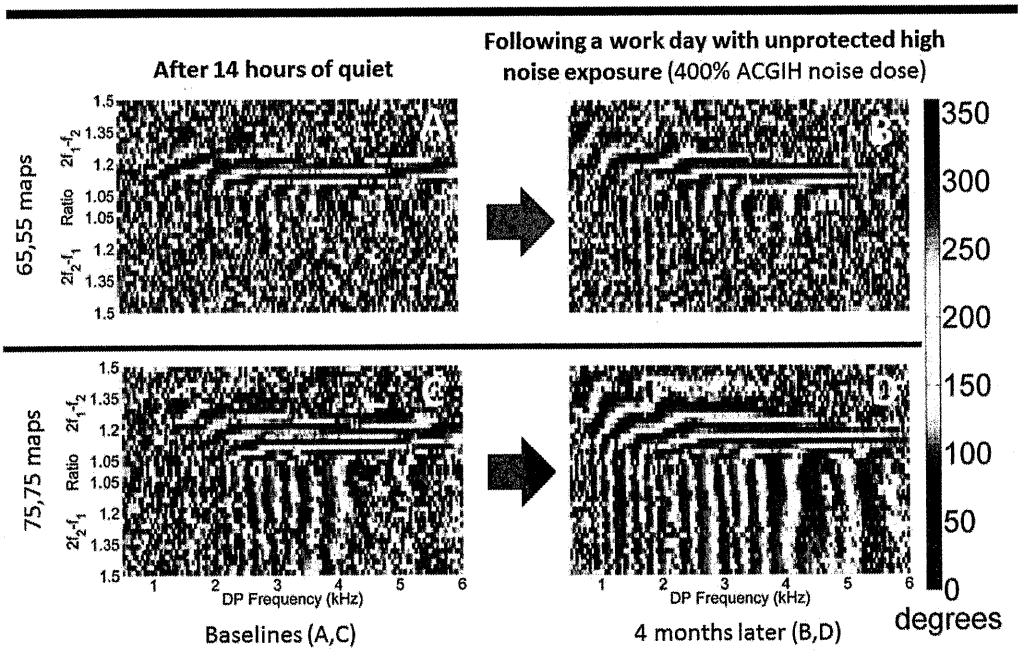

Figure 2: DPOAE phase maps obtained 4 months apart in one male subject. Baseline (A,C) and re-test after noise exposure (B,D) obtained at two stimulus levels 65,55 dB SPL (A,B) and 75,75 (C,D)

Gorga MP, Nelson K, Davis T et al. (2000). Distortion product otoacoustic emission test performance when both 2f1-f2 and 2f2-f1 are used to predict auditory status. J Acoust Soc Am 107: 2128-2135.

Hamernik RP, Patterson JH, Turrentine GA et al. (1989). The quantitative relation between sensory cell loss and hearing thresholds. Hear Res 38: 199–211.

Hamernik RP, Ahroon WA, Lei SF (1996). The cubic distortion product otoacoustic emissions from the normal and noise-damaged chinchilla cochlea. J Acoust Soc Am 100: 1003–1012.

Harris FP (1990). Distortion-product otoacoustic emissions in humans with high frequency sensorineural hearing loss. J Speech Hear Res 33: 594–600.

Harris FP, Lonsbury-Martin BL, Stagner BB et al. (1989). Acoustic distortion products in humans: systematic changes in amplitude as a function of f2/f1 ratio. J Acoust Soc Am 85: 220-229.

Kemp D (1978). Stimulated acoustic emissions from within the human auditory system. J Acoust Soc Am 64: 1386-1391.

Knight RD, Kemp DT (2001). Wave and place fixed DPOAE maps of the human ear. J Acoust Soc Am 109: 1513-1525.

Lapsley Miller JA, Marshall L (2007). Otoacoustic emissions as a preclinical measure of NIHL and susceptibility to NIHL. In: Robinette MS, Glattke T (eds): Otoacoustic emissions, clinical application (3rd ed), (pp 321–341). New York: Thieme.

Lonsbury-Martin BL, Martin GK (1990). The clinical utility of distortion product otoacoustic emissions. Ear Hear 11: 144-154.

Lonsbury-Martin BL, Martin GK, Probst R et al. (1987). Acoustic distortion products in rabbit ear canal. I. Basic features and physiological vulnerability. Hear Res 28: 173–189.

Martin GK, Jassir D, Stagner BB et al. (1998) Locus of generation for the 2 f 1– f 2 vs. 2 f 2– f 1 distortion product otoacoustic emissions in normal-hearing humans revealed by suppression tuning, onset latencies, and amplitude correlations. J Acoust Soc Am 103:1957–1971.

Martin G, de la Garza A, Stagner B et al. (2005). Detailed DPOAE level/phases maps in normal and noise-damaged rabbit ears: Insights into generation processes. Abstracts of the twenty-eight annual midwinter research meeting Association for Research in Otolaryngology (ARO), Mt. Royal, NJ. P 152.

Rossi G, Solero P, Rolando M et al. (1991). Recovery time of the temporary threshold shift for delayed evoked otoacoustic emissions and tone bursts. Otorhinolaryngology 53: 15-18.

Sutton LA, Lonsbury-Martin BL, Martin GK et al. (1994). Sensitivity of distortion-product otoacoustic emissions in humans to tonal over-exposure: Effects of L1-L2 differences. Hear Res 75: 161-174.