Sensitivity of Distortion Product Otoacoustic Emissions in Noise-Exposed Chinchillas

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Abstract

The present study investigates the effect of small amounts of outer hair cell (OHC) loss on distortion product otoacoustic emission (DPOAE) levels and evoked potential permanent threshold shifts (PTS) in a population of 12 noise-exposed chinchillas. The group mean DPOAE level, which decreased by up to ~15 dB in the presence of less than 8 dB PTS and 15% OHC loss, indicates that DPOAEs can detect an underlying cochlear pathology (i.e., OHC damage/loss) despite the presence of normal to near normal thresholds. The sensitivity of DPOAEs in detecting OHC loss makes this test measure suited for diagnosing sensorineural hearing impairment, particularly when abnormal auditory symptoms (i.e., speech discrimination problems) are associated with a normal audiogram in the clinical setting and as part of a hearing conservation program.

Key Words: Auditory evoked potential, distortion product otoacoustic emissions, permanent threshold shift, sensory cell loss

Abbreviations: AEP = auditory evoked potential; DPOAE = distortion product otoacoustic emissions; IHC = inner hair cell; OHC = outer hair cell; PTS = permanent threshold shift

Sumario

El presente estudio investiga el efecto sobre los niveles de las emisiones otoacústicas por productos de distorsión (DPOAE) y sobre los cambios permanentes de umbral (PTS) de los potenciales evocados, de la pérdida de pequeñas cantidades de células ciliadas externas (OHC) en una población de 12 chichillas expuestas a ruido. El nivel promedio grupal de las DPOAE, que disminuye hasta en ~15 dB en presencia de 8 dB de PTS y de un 15% de pérdida de OHC, indica que las DPOAE pueden detectar una patología coclear subyacente (p.e., daño/pérdida de OHC) a pesar de la presencia de umbrales auditivos normales o cercanos a la normalidad. La sensibilidad de las DPOAE para detectar la pérdida de OHC convierte a esta medición en algo apropiado para diagnosticar pérdidas auditivas sensorineurales, particularmente cuando los síntomas auditivos anormales (p.e., problemas de discriminación del lenguaje) están asociados con un audiograma normal en el contexto clínico y como parte de un programa de conservación auditiva.

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During the past several years, distortion product otoacoustic emissions (DPOAEs) have received attention as a clinical test of cochlear status for the differential diagnosis of hearing disorders, monitoring of potential progressive hearing disorders, and newborn hearing. Much less attention has been given to an assessment of the ability of DPOAEs to detect reduced cochlear function (i.e., outer hair cell [OHC] loss) in the presence of normal auditory sensitivity (i.e., as an indicator of the early onset of cochlear damage).

Audiologists and otolaryngologists who encounter cases which present with auditory difficulties of speech discrimination in noise despite normal audiograms can be dealing with potential problems attributed to a variety of etiological factors (e.g., middle ear, cochlear, central auditory nervous system dysfunction; linguistic and cognitive factors). To some extent, the various etiologies responsible for this condition have contributed to the difficulty involved in making an accurate diagnosis. The few studies that attempted to provide evidence of an underlying physiological basis for the abnormal auditory symptoms in such obscure cases are not consistent (Lutman and Saunders, 1992; Zhao and Stephens, 1998, 2000). Zhao and Stephens (2000), for instance, suggest that most of these patients have a mild cochlear pathology as determined by abnormal otoacoustic emission measurements. This supports their earlier results (Zhao and Stephens, 1998) that showed that notch center frequencies obtained in Audioscan tests correlated well with notches found on DPOAE audiograms. However, earlier research by Lutman and Saunders (1992) using evoked otoacoustic emissions failed to show abnormal OHC activity in a group of 50 patients diagnosed with obscure auditory dysfunction who were matched against a group of 50 normal controls.

A number of experiments have shown that the DPOAE may be predictive of the early onset of noise-induced hearing loss in humans (Sutton et al, 1994; Attias et al, 1998; Xu et al, 1998; Lucertini et al, 2002; Satar et al, 2003) and animal models (Mensh et al, 1993; Hamernik et al, 1996; Fraenkel et al, 2003; Davis et al, 2004). Fraenkel et al (2003), for instance, reported that the group mean DPOAE amplitude reduction in four groups of rats exposed to various durations of 113 dB SPL broadband noise indicated a "clinical advantage" for DPOAEs in detecting slight temporary changes. In 76 military personnel, Attias et al (1998) also reported that DPOAE amplitudes were significantly reduced and often not detectable in individuals with a history of noise exposure but with normal audiograms in comparison to those with a negative history and normal hearing. A higher percentage of abnormal DPOAEs (SNR) was also observed in 20 normal hearing humans with tinnitus who had a history of exposure to noise (Satar et al, 2003). In a related study (Xu et al, 1998), transient evoked otoacoustic emissions (amplitude and SNR measures) were reported to be more sensitive than threshold measures in the detection of early noise induced cochlear damage in 38 humans. In addition, Lucertini et al (2002) demonstrated that clinically normal hearing subjects with mild cochlear damage could be identified using transiently evoked otoacoustic emissions.

Over the past few years, we had the opportunity to collect DPOAEs and auditory evoked potential (AEP) thresholds before and after noise exposure in 187 chinchillas for which sensory cell loss data were available. One of the objectives of this exercise was to attempt to build a normative DPOAE and AEP threshold database from which one could estimate the extent of sensory cell loss. Of the 187 animals examined, we selected for inclusion in this study only those animals with PTS $\leq$15 dB at each AEP test frequency.
Sensitivity of DPOAEs in the Chinchilla

Davis et al

(i.e., 0.5, 1.0, 2.0, 4.0, and 8.0 kHz). This strict criteria was adopted as a means to incorporate only ears from our database with normal to near-normal sensitivity (i.e., a condition analogous to humans with normal to near-normal audiograms) across the test frequency range to determine the relation among DPOAE, OHC loss, and PTS. This group of 12 animals constitutes our “experimental group” for the purposes of this report. An analysis of this subset of the database illustrates the potential value of DPOAE measures in documenting mild OHC loss, despite the presence of normal to near-normal threshold test results. The objective of this exercise was to determine if small amounts of OHC loss could be reflected in changes in DPOAE levels, and whether or not this loss has a different effect on the relative magnitude of change in DPOAE levels and AEP thresholds.

Given the lack of information in the literature that documents an etiological basis for the hearing complaints and/or abnormal DPOAEs of individuals with normal to near-normal audiograms, the extent of OHC loss in ears with very little to no permanent threshold shift (PTS) was determined and compared to the pattern of DPOAE level measurements obtained before and after noise exposure in the chinchilla. Thus, in cases involving sensory cell pathology, the nature of the relation among OHC loss, hearing thresholds, and DPOAEs should begin to provide insights into the diagnostic value of DPOAEs in both a clinical setting and as part of a hearing conservation program, where the DPOAE may compliment the audiogram as an indicator of sensory cell pathology.

MATERIALS AND METHODS

DPOAEs, AEPs, and frequency-specific sensory cell data were collected on a population of 187 chinchillas exposed to a variety of continuous noise for a period of five uninterrupted days. Data were acquired over a five-year period as part of a protocol to assess the effects of complex (non-Gaussian) noise environments on hearing. The twelve subjects that are the focus of this paper were selected, based on the criteria described above, from the population of 187 subjects.

Animal Model

The chinchilla is generally agreed to be a good model for studying the effects of noise on hearing. The AEP has been shown to be an acceptable alternative to the extremely time-consuming behavioral conditioning approach (Salvi et al, 1982). The correlation between AEP and behavioral measures has been strengthened by direct comparisons of AEP and behavioral thresholds made on the same animal before, during, and after a noise exposure that induced a TTS and PTS. During all three periods in time there was close agreement between threshold estimates obtained with 20 msec test tones (Davis and Ferraro, 1984). The audiogram of the chinchilla is also similar to the human’s between 0.25 and 8 kHz (Miller, 1970).

Normal and abnormal temporal processing of acoustic signals in terms of temporal integration (Henderson, 1969), temporal resolution (Salvi et al, 1979), and forward masking are also similar for human and chinchilla. In many cases the differences between the two species amounts to a frequency dependent scaling factor. This association has been strengthened using otoacoustic emission data (Davis et al, 2004). The cumulative distributions from normal-hearing humans (N = 107) obtained by Gorga et al (1996) and functions obtained from a population (N =187) of normal pre-exposure chinchillas (Davis et al, 2004) illustrates the parallel behavior of the population DPOAE data between the two species.

Surgical Preparation

All animals were made monaural by the surgical destruction of the left cochlea, and an AEP-recording electrode was implanted into the left inferior colliculus. Details of the AEP procedures and surgery can be found in Ahron et al (1993). Briefly, each animal was anesthetized [IM injection of ketamine (35 mg/kg body weight) and xylazine (1 mg/kg body weight)] and made monaural by the surgical destruction of the left cochlea. A bipolar, platinum EEG electrode, with electrode lengths of 7.5 mm (probe) and 2.5 mm (ground) was implanted into the region of the inferior colliculus under stereotaxic control for single-ended recordings of the AEP (Henderson et al, 1973; Salvi et al, 1982). A xylazine reversing agent [yohimbine
(2 mg/kg body weight) IM] was administered after the surgical procedure. The animals were allowed to recover for at least two weeks before AEP and DPOAE testing began.

**Threshold Testing**

The animals were awake during testing and restrained in a yokelike apparatus to maintain the animal’s head in a fixed position within the calibrated sound field (Blakeslee et al, 1978). AEPs were collected to 20 msec pure-tone bursts with 5 msec rise/fall times, presented at a rate of 10/sec. A general purpose computer was used to acquire the AEP data and control the frequency, intensity, and timing of the stimulus. The electrical signal from the implanted electrode was amplified (50,000x), filtered (30 to 3000 Hz), and sampled using an analog-to-digital (12-bit resolution) converter at 20,000 samples/sec (50 µsec period) over 500 points to obtain a 25 msec sampling window. Each digitized waveform was analyzed for large amplitude artifacts, and if present, the sample was rejected from the average and another sample taken. Averaged AEPs were obtained from 250 presentations of the 20 msec signal. Each waveform was stored to be used in threshold determination following the completion of the test stimulus intensity series.

Thresholds were estimated by one investigator from each tone-burst intensity series using 5 dB steps at octave intervals from 0.5 to 16 kHz. Threshold was determined to be one half step size (2.5 dB) below the lowest intensity that showed a response consistent with the responses seen at higher intensities. The average of at least three separate threshold determinations at each frequency obtained on different days was used to define the pre-exposure audiogram.

Following a 30-day postexposure recovery period, thresholds were measured again on three different days and averaged to establish the animal’s PTS. PTS was defined as the difference between the 30-day post- and pre-exposure audiograms.

**Cubic DPOAEs**

Cubic distortion products (2f1-f2) were measured in the ear canal of the awake but restrained animal with the Etymotic ER-10C instrument using CUBeDIS (v2.40) software. DPOAEs were measured at 32 points per octave across the test frequency range to produce a DP-gram. The following parameters were used in collecting the DPOAEs: 1.0 kHz ≤ f2 ≤ 10 kHz, where f2 was the higher frequency primary tone; f2/f1 = 1.22. The level (L1) of the f1 primary tone was 65 dB with L1 = L2 +10 dB and the averaging time was constant at 2 sec. All DPOAE data were plotted as a function of f2. The same number of DP-grams were collected and at approximately the same times during the experimental sequence as the AEP audiograms. The average of the three pretreatment and three 30-day posttreatment DPOAE measurements was used to establish permanent treatment effects.

**Noise Exposures**

Twenty-six groups of animals with 2 to 12 subjects per group were exposed to one of several different noises. Noise exposures lasted 24 h/day for five days and were interrupted once daily for approximately 20 to 30 minutes for AEP testing. Each exposure had in common approximately the same flat spectrum between 0.125 and 10.0 kHz. Exposure levels were presented at 90, 100, or 110 dB(A) SPL with most of the data coming from the 100 dB exposure. The 26 exposures differed only in their temporal structure, which was designed to produce 4 Gaussian and 22 non-Gaussian exposure conditions (Hamernik et al, 2003). During exposure, individual chinchillas were confined to cages (10” x 11” x 16”) with free access to food and water. A maximum of six animals was exposed at a time. Peak SPLs in the exposure field were uniform to within less than 2 dB.

**Histology**

Following the last AEP or DPOAE test protocol, each animal was euthanized under anesthesia and the right auditory bulla removed and opened to gain access to the cochlea for perfusion. Fixation solution consisting of 2.5% glutaraldehyde in veronal acetate buffer (final pH = 7.3; 605 mOs) was perfused through the cochlea. After 12 h of fixation, the cochlea was postfixed in 1% OsO4 in veronal acetate buffer. Surface preparation mounts of the entire organ of Corti were prepared, and IHC and OHC populations, computed over 0.24 mm lengths of the basilar membrane of individual
animals, were plotted as a function of frequency and location using the frequency-place map of Eldredge et al (1981). Group mean sensory cell population data is presented as averages taken over octave band lengths of the cochlea centered on the primary AEP test frequencies.

Animal Care

The care and use of the animals reported on in this study were approved by the SUNY-Plattsburgh Institutional Animal Care and Use Committee. In conducting the research described in this report, the investigators adhered to the Guide for Care and Use of Laboratory Animals (1996), as promulgated by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources Commission on Life Sciences, National Academy of Sciences, National Research Council.

RESULTS

An example of the results from one group of animals (N = 11) exposed to a continuous non-Gaussian noise at 100 dB(A) for five consecutive days is shown in Fig. 1. This group is one of the noise-exposed groups that constitutes our normative pre-exposure and noise-exposed population of 187 subjects. This figure is presented in order to illustrate the nature of the database that is available on each subject and each group. The mean pre- and postexposure DPOAE levels, AEP thresholds, and the profile of IHC and OHC loss produced in this group by the exposure were consistent in showing changes that reflect the effects of the noise-induced sensory cell loss on indices of auditory functioning. In this example, there are large OHC losses across the length of the cochlea that are reflected in depressed DPOAEs and elevated thresholds across the test frequency range. Note that each DPOAE datum point was obtained by averaging the 32 DPOAEs recorded in each octave over 1/3-octave bands centered on the indicated frequencies. Also, larger amounts of OHC loss in the 2–4 kHz region of the basilar membrane are associated with slightly more PTS at 2 and 4 kHz and slightly greater reductions in DPOAE level in the 4 to 8 kHz range. The agreement among DPOAEs, AEP, and OHC losses in this group of noise-exposed chinchillas should serve to increase the confidence that can be placed in the diagnostic value of the DPOAE metric for clinicians who use emissions for diagnostic purposes.

The mean pre- and postexposure DPOAE level for $L = 65$ dB SPL measured in the experimental group (N = 12) defined above is shown in Fig. 2a, where it is compared to the preexposure normative database (N = 187) for

Figure 1. (a) The group mean (N = 11) DPOAE level measured at the indicated times for the animals exposed to a non-Gaussian noise presented at 100 dB (A) for 5 days. Each DPOAE datum point represents the mean DPOAE level measured over adjacent 1/3-octave bands plotted as a function of $f_2$. (b) The corresponding group mean AEP thresholds. (c) The group mean cochleogram. Each datum point represents the mean percent IHC or OHC loss measured over octave band lengths of the basilar membrane.

Bar = standard error (se). Where no error bar is shown the se was smaller than the size of the datum symbol.
The mean preexposure DPOAE level in the experimental group varied between 17 dB SPL at the lower frequencies to about 32 dB SPL at the higher frequencies with the highest DPOAE levels occurring between 4 and 8 kHz. Standard errors (se) of the mean were less than 2 dB at each frequency. The mean preexposure DPOAEs obtained in the experimental group were very similar to those of the normative database. These results are consistent with those reported by Hamernik and Qiu (2000) and Clock Eddins et al (1999) in the chinchilla. The mean postexposure DPOAE for this group showed a decrease in level of approximately 3 to 17 dB for f2 ranging between 1.0–10.0 kHz. Standard errors for the postexposure group mean DPOAE levels were less than 3 dB. The postexposure DPOAEs were reduced by up to 6 dB below one standard deviation of the normative preexposure population.

Fig. 2b presents the mean pre- and postexposure AEP thresholds for the same two populations of animals, that is, the normative group and the experimental group. The preexposure thresholds in the experimental group were very similar (se ≤ 2 dB) to that of the normative population across the frequencies tested. Following noise exposure the thresholds decreased by up to 7 dB across the 0.5–16 kHz test frequency range (postexposure se ≤ 2 dB). The corresponding group mean PTS and the profile of sensory cell loss averaged over 1/3-octave band lengths of the cochlea is shown in Fig 2c. Table 1 summarizes the group mean PTS, OHC loss in the 1/3-octave bands centered on the selected AEP test frequency for the experimental group, and the corresponding reduction in DPOAE level (pre- minus postexposure DPOAE; ∆ DPOAE) averaged over the 1/3-octave band centered at each frequency. The experimental group showed only a small amount of OHC loss in any 1/3-octave band (2 to 8% at 1.0, 2.0, and 4.0 kHz and <15% at 0.5 and 8.0 kHz) and PTS ≤ 8 dB at corresponding frequencies. For a small loss of OHCs (~12%) there were significant differences (t-test, P > 0.01 or 0.05) between the amount of change in DPOAEs and PTS at 2, 4, and 8 kHz as shown in Table 1.

Individual DPOAE levels decreased an average of 12 dB, which was 6 dB more than the corresponding mean change in PTS at the test frequencies where significant differences occurred. In addition, correlation coefficients between OHC loss and post DPOAE levels and between OHC loss and PTS values were both similar and equally poor (r = 0.42-0.02) across the test frequencies of 1.0, 2.0, 4.0, and 8.0 kHz. That is, there was a large range of emission responses and PTS for a given OHC loss. This result is consistent with the results of Hamernik and Qiu (2000) and Davis et al.
(2004), which showed that in the chinchilla there was a poor correlation between OHC loss and DPOAE and PTS, and between PTS and DPOAE, for PTS up to 25 dB.

Despite poor correlations, the DPOAE showed postexposure decrements that signaled the onset of OHC loss in cases where there were relatively small amounts of PTS. Two individual cases from the experimental group illustrate this. Data from one animal (#2958) that serves as an example of 9 out of the 12 animals in the experimental group is shown in Fig. 3.

Fig. 3a shows the pre- and postexposure DPOAE and noise floor while Fig. 3b shows the PTS and sensory cell loss. In this example, the animal exhibits little change in thresholds (PTS \( \leq 7 \) dB), small amounts of sensory cell loss (<10%) between 1 and 12 kHz, and reductions in DPOAE output that ranged from about 5 to 20 dB above 1 kHz. Fig. 4 shows the results from another animal (#2782) that is typical of the remaining three animals in the experimental group. This animal also showed little PTS (\( \leq 12 \) dB) across the test frequencies in the presence of OHC losses of up to 30% in the 0.5 kHz region and 50% in the 8 kHz region of the cochlea. Although thresholds were not greatly affected by the considerable OHC loss, DPOAEs were reduced by 17 to 40 dB across the entire test frequency range.

**DISCUSSION**

The preceding analysis examined the relation between DPOAEs and OHC loss in a group of experimental animals having little PTS to determine if changes in the DPOAE level can be reliably used to detect the presence of small amounts of OHC loss for diagnostic purposes (i.e., cochlea versus eighth nerve, early onset of cochlear damage). The mean results from the experimental group, which showed a reduction in DPOAE level of \( \sim 15 \) dB in the presence of less than 15% OHC loss, indicates that this test can be

**Table 1. Group Mean (N = 12) PTS at the Indicated Frequencies, Change in DPOAE Level (\( \Delta \) DPOAE), and OHC Loss Over the 1/3-Octave Band Length of the Basilar Membrane at the Indicated Frequencies in the Experimental Group**

<table>
<thead>
<tr>
<th>kHz</th>
<th>PTS (dB)</th>
<th>( \Delta ) DPOAE (dB)</th>
<th>OHC loss (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>4</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>1.0</td>
<td>4</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>2.0**</td>
<td>6</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>4.0*</td>
<td>8</td>
<td>15</td>
<td>4</td>
</tr>
<tr>
<td>8.0*</td>
<td>15</td>
<td>15</td>
<td>12</td>
</tr>
</tbody>
</table>

* \( p < .05 \) ** \( p < .01 \)

Figure 3. (a) The mean pre- and postexposure DPOAE for animal #2958. (b) The sensory cell loss and PTS audiogram for this animal.
used as a reliable and sensitive measure to detect small amounts of sensory cell loss resulting from noise exposure in the chinchilla when thresholds are within normal limits (<10 dB PTS). The relatively large reduction (12–15 dB) in the DPOAE in the presence of smaller OHC losses (3–12%) at 2, 4, and 8 kHz may be accounted for not only by the OHC loss but also by morphological changes (e.g., cilia defects or intracellular changes) that can affect the function of cells that are present and for which the cochleogram provides no information. In addition, the statistically significant larger reduction in DPOAE levels than PTS resulting from small amounts of OHC loss less than ~15% in the 1/3-octave band centered at 3 of the 4 test frequencies as indicated in Table 1, suggests that the DPOAE is more sensitive to OHC damage/loss than threshold measures. This finding is consistent with earlier results that indicated that the DPOAE is sensitive to the onset of both hearing and sensory cell loss in noise exposed animals (Hamernik et al, 1996; Le Calvez et al, 1998; Hamernik and Qiu, 2000; Davis et al, 2004). OHC losses, which may not be reflected in elevated thresholds, may be reflected in reduced DPOAEs since the OHC system is the source of the DPOAE. Thus, DPOAEs can be used to detect the early stages of cochlear pathology that target the OHCs (e.g., noise-induced hearing loss, ototoxicity, presbycusis). DPOAE measures, therefore, may help to validate auditory complaints (e.g., speech discrimination difficulties, tinnitus) that are often seen in the presence of normal to near-normal thresholds. An example of such a case was reported by Lonsbury-Martin et al (1999) in which a 41-year-old patient with a history of noise exposure complained of hearing difficulties (e.g., sensitivity, tinnitus, speech discrimination difficulties in noise) in the presence of normal hearing thresholds (<20 dB HL) concomitant with reduced DPOAE responses (greater than 1 sd beyond the mean DPOAE levels of 94 normal-hearing adults). Animal #2782 shown in Fig. 4 shows some similarities to the above subject. In this animal, reduced DPOAEs (17 to 40 dB) were seen in the presence of relatively normal thresholds (≤12 dB PTS) despite OHC losses of up to 50%. OHC losses of this magnitude have been shown to alter frequency selectivity (Dallos et al, 1977; Harrison et al, 1977; Liberman and Dodds, 1984; Davis et al, 1989; Leeuw and Dreschler, 1998), which can result in discrimination difficulties. Good frequency selectivity, which is dependent on a normal OHC system (Liberman and Dodds, 1984; Davis et al, 1993), is required for good speech discrimination. Since DPOAEs are also dependent on a normal OHC system (Brownell, 1990), reduced DPOAEs should also signal reduced speech discrimination.

The results presented indicate that on the basis of threshold information alone, one might underestimate the sensory cell loss without the DPOAE data. This conclusion is supported by the results of others, which show up to 30% OHC loss in subjects with less than 10 dB of PTS (Hamernik et al, 1989; Hamernik and Qiu, 2000; Davis et al, 2004). Bohne et al (1987) also showed that 20–30% OHC loss in the low frequencies was often not accompanied by corresponding behaviorally measured threshold shifts in the chinchilla. Thus, clinically defined “normal” hearing thresholds can exist in the presence of sensory cell pathology that can contribute to auditory

**Figure 4.** (a) The mean pre- and postexposure DPOAE for animal #2782. (b) The sensory cell loss and PTS audiogram for this animal.
symptoms. DPOAE data can provide evidence of OHC loss and thus confirm a physical basis for reduced auditory function (e.g., tinnitus, speech discrimination difficulty, distorted hearing) despite the presence of normal hearing thresholds.

Our results support the findings of Zhao and Stephens (2000), who reported a decrease in DPOAE levels over the frequency range of 1400–5600 Hz in 77% of 110 patients with audiometrically “normal” hearing despite speech discrimination difficulties. The reduced DPOAEs suggest that OHC loss may have been primarily responsible for the auditory processing problems reported in these cases. Thus, in cases involving sensory cell pathology, the nature of the relation among OHC loss, hearing thresholds, and DPOAEs should begin to provide insights into the diagnostic value of DPOAEs in a clinical setting where the DPOAE may complement the audiogram as an indicator of sensory cell pathology. The chinchilla data presented in this report combined with the results in humans (Sutton et al, 1994; Attias et al, 1998; Xu et al, 1998; Zhao and Stephens 2000; Lucertini et al, 2002; Satar et al, 2003) and in animal models (Mensh et al, 1993; Hamernik et al, 1996; Fraenkel et al, 2003; Davis et al, 2004) suggest that OAEs are better diagnostic indicators for the auditory symptoms reported than are clinical tests of hearing sensitivity. However, in patients presenting with speech discrimination difficulty despite normal audiograms and emissions, it is clear that involvement of more central auditory pathways must be considered.

The findings, which indicate that small amounts of OHC loss have a more significant effect (reduction) on DPOAE amplitude levels than on measures of threshold sensitivity in the chinchilla, suggest that DPOAEs can indicate an early onset of cochlear damage in humans. Although the relation among thresholds, DPOAEs, and sensory cell loss in humans cannot be easily examined, the sensitivity of DPOAEs to small amounts of OHC loss in the chinchilla suggests that this metric should be adopted as an index of auditory damage in the clinical setting. These results also suggest that the DPOAE be considered, within the context of hearing conservation practices, as a complement to existing threshold test results to detect OHC loss resulting from noise exposure.

CONCLUSION

The results above that indicate that small amounts of OHC loss can be reliably detected with DPOAEs suggest that this test should be included in an audiological test battery to diagnose mild cochlear pathology (i.e., OHC loss) in individuals where cochlear injury may be suspected (e.g., the initial signs of noise exposure, ototoxicity). Because DPOAEs are a common clinical audiological measure, their reduction in the presence of a normal audiogram should not be interpreted as a subclinical auditory impairment but, rather, an objective measure of OHC damage or loss. The DPOAE test can provide important clinical evidence of an underlying physiological abnormality (OHC damage/loss), which can help resolve clinical ambiguities resulting from the appearance of auditory symptoms due to OHC pathology despite the presence of normal to near-normal threshold test results.

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