

## **771** Distortion Product Otoacoustic Emissions from the Basilar Papilla of the Tree Frog, *Hyla cinerea*.

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For the generation of distortion product otoacoustic emissions (DPOAE) in the cochlea, currently a disparate-place two-source model is accepted (Talmadge et al. '99). One source originates from the place of generation in the overlap region, and the other source originates from the distortion product tonotopic site. In these models, the interaction between the two cochlear sources gives rise to the DPOAE fine structure, and can explain the distinct notch in input-output functions of DPOAE (Mills '97). The frog inner ear lacks a cochlea but contains two hearing papillae, the amphibian papilla (AP) and the basilar papilla (BP), respectively. The BP is a simple auditory receptor mainly tuned to one specific frequency in each individual. Furthermore, neither papilla in the frog inner ear is over a basilar membrane. Here we report on DPOAE emitted from the BP of the tree frog. The primary tone frequencies ( $f_1$  and  $f_2$ , respectively) for which the BP elicited maximum cubic distortion tone (CDT) were determined. Next, for this optimum set of stimulus frequencies, the level of the CDT was measured as function of tone levels  $L_1$  and  $L_2$ . The levels were varied independently between 35 and 85 dB SPL with a 5 dB step interval. Preliminary results are in qualitative agreement with the model and experimental results in mammals presented by Mills (Mills '97). The results presented here are in support of the idea that DPOAE from the BP are generated in the same way as in the cochlea. Because of the anatomical properties of the BP it is hard to see how the two-source model is applicable in the frog inner ear.

Mills, DM (1997). Interpretation of distortion product otoacoustic emission measurements. I. Two stimulus tones: JASA 102(1), 413-429.

Talmadge, CL, Long, GR, Tubis, A, and Dhar, S (1999). Experimental confirmation of the two-source interference model for the fine structure of distortion-product otoacoustic emissions: JASA 105(1), 275-292.

## **772** Changes in Evoked Otoacoustic Emissions and Hearing Thresholds after a Six-month Deployment on an Aircraft Carrier

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Evoked otoacoustic emissions and hearing thresholds were measured in 339 sailors from the USS Dwight D. Eisenhower aircraft carrier before and after a six-month deployment to the Mediterranean. Sailors from the Air, Reactor, and Engineering departments were targeted because they were considered most at risk for noise-induced hearing loss. At pre-deployment and post-deployment testing, hearing thresholds (0.5 to 6 kHz) were measured using a modified Hughson-Westlake procedure and normal middle-ear pressure was established. Transient-evoked otoacoustic emissions (non-linear click stimulus at 74 dB pSPL) and distortion-product otoacoustic emissions ( $f_2/f_1=1.22$ , at four stimulus levels) were then measured using the Otodynamics ILO292 Echoport. There was no consistent change in average hearing thresholds for the group; however, some individuals showed significant threshold shifts. Temporary threshold shifts were confirmed for two sailors (two ears) and permanent threshold shifts were confirmed for fifteen sailors (eighteen ears), based on their noise history and a confirmatory audiogram. Some additional significant threshold shifts were unable to be confirmed. Preliminary group results indicated that after deployment there was a decrease in average distortion-product and transient-evoked otoacoustic-emission amplitudes. Changes in otoacoustic-emission amplitudes might be a more sensitive indicator of noise-induced damage to the inner ear than changes in hearing thresholds.

## **773** Correlation of DPOAE level shifts and ABR threshold shifts with noise-induced histopathological changes in chinchillas: Some surprises

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DPOAEs are thought to be produced by OHC. However, comparison with noise-induced pathology suggests that their origin is much more complex. DPOAE levels ( $f_1$  &  $f_2$  at 55-75dB,  $f_2=1.22 f_1$ , 6 points/octave, 0.5-20kHz) & ABR thresholds were determined pre-exposure. Noise was a 4kHz OBN at 108dB (1.75h, n=6) or 85dB (24h, n=5). DPOAE level shifts (LS) & ABR threshold shifts (TS) were determined at 0 & up to 30 days post-exposure. The cochleae were fixed with OsO<sub>4</sub>, embedded in plastic & dissected into flat preparations. The length of the organ of Corti (OC) was measured; missing IHC & OHC counted; stereocilia damage graded; & regions of OC, nerve fiber & stria loss determined. Cytocochleograms were made showing loss/damage by % distance from the apex (& frequency) with the LS & TS overlaid. The best correlation of LS with pathology required plotting at  $f_1$ . The best correlation of TS was with IHC & nerve fiber loss. Wide regions of up to 40% scattered apical OHC loss showed little LS. In 2 cases, LS occurred with OHC abnormalities but not loss. In 3 cases, there were R/L LS asymmetries with symmetric pathology; in 2 cases, LSs were symmetric with asymmetric pathology. At all recovery times, the largest LS occurred at  $3f_1-2f_2$  for mid-frequencies (MF, 4-12kHz) & at  $2f_1-f_2$  below & above that. With 108 dB, there was up to 40% DPOAE recovery at MF in 3/6 cases where there was 80-100% OHC loss in the basal half of the OC. Partial recovery at MF also occurred in regions where the OC was missing. With 85 dB, there was no LS at small focal lesions (100% loss of OHC over 0.4 mm) when  $f_1$  or  $f_2$  was within the lesion but not both. There was no correlation of LS with stereocilia damage. In 2/5 cases with a stria lesion, the only pathological correlate with LS was this damage. These results suggest that either noise-induced DPOAE LSs at MF include a component from the basilar membrane or they are augmented from someplace other than  $f_1$  or  $f_2$ , possibly the basal 20%.

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## **774** Audiometric Threshold Estimation in Cochlear Hearing Loss Ears by Means of Weighted Extrapolated DPOAE I/O-Functions

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DPOAE threshold and audiometric threshold have been shown to be closely related when estimating DPOAE threshold by simply extrapolating DPOAE I/O-functions using linear regression analysis (Boege and Janssen 2001). The purpose of this study was to improve and to evaluate this new method for clinical application.

DPOAE I/O-functions were recorded in 826 sensorineural hearing loss ears at up to 50 frequencies between 500 Hz and 8 kHz in a wide level range from  $L_2=20$  to  $L_2=65$  dB SPL at up to 10 levels ( $L_1=0.4 L_2+39$ ). DPOAEs were accepted as valid for signal-to-noise ratios (SNR) exceeding 6 dB. For estimating DPOAE threshold an extrapolation of the DPOAE pressure I/O-function was performed by determining the point of intersection of the extrapolated regression line with the  $L_2$  coordinate. In the linear fit  $p_{DP}(L_2)=a+bL_2$ ,  $a$  and  $b$  give the threshold and the slope of the DPOAE growth, respectively, representing estimates of sensitivity and compression of the cochlear amplifier.

According to Boege and Janssen, in 54% of the DPOAE I/O-functions a linear dependency of the DPOAE sound pressure  $p_{DP}$  on the primary tone level  $L_2$  was found. However, when adapting the proposed