Comparison of peripheral compression estimates using auditory steady-state responses (ASSR) and distortion product otoacoustic emissions (DPOAE)

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ABSTRACT

The healthy auditory system shows a compressive input/output (I/O) function as a result of healthy outer hair cell function. Hearing impairment often leads to a decrease in sensitivity and a reduction of compression, mainly caused by loss of inner and/or outer hair cells. Compression is commonly estimated based on behavioral procedures (Plack et al., 2004), which are time consuming and rely on assumptions regarding the ability to selectively investigate cochlear processing, or on objective recordings such as otoacoustic emissions (OAEs) (Neely et al., 2003), which allow to selectively study cochlear processing but the interpretation of results for individual data is challenging.

Auditory steady-state responses (ASSR) are another objective method which allows fast, reliable and frequency-specific measurements of hearing function. It is hypothesized that compressive behavior is observed in normal-hearing (NH) listeners while in hearing-impaired (HI) listeners, sensitivity and compression are reduced. ASSR data are later compared to data from distortion-product otoacoustic emissions (DPOAEs) recordings.

RESULTS

Peripheral compression can be estimated through ASSR I/O functions in NH subjects. HI subjects show a change in sensitivity and compression estimate.

How do compression estimate correlate when measured using ASSRs versus DPOAEs?

HYPOTHESIS

- NH subjects consistently show compressive functions with slopes between 0.1 and 0.5 dB/dB.
- ASSR saturates or even decreases at higher stimulus levels.
- Repeated points (●) recorded in different sessions show small variability in the response.
- HI subjects show higher variability in the results.
- Significant responses at input levels of 30 dB SL and above have been obtained for HI subjects.
- ASSR I/O functions in HI subjects reflect the loss of sensitivity at lower stimulus levels.

METHODS

ASSR (20 subjects, 13 NH and 7 HI)
- 64-channel EEG system with active electrodes (Biosens).
- ASSR magnitude obtained from the recorded ASSR spectrum, computed from the weighted averaged waveform.
- Detection of significant results using F-test (p-value ≤ 1%)

DPOAE (12 NH subjects)
- Fitting curves
- Least-squares-fit (LSF) method used to obtain the magnitude and phase of the 2f2 − f1 DPOAE component.
- DPOAEs recorded using a time windowing technique (Long et al., 2008).

DPOAE in NH:
- The panels show ASSR I/O functions for four different carrier frequencies recorded in a NH subject. Panel A: f2 = 0.5 kHz @ f1 = 81 Hz, Panel B: f2 = 1 kHz @ f1 = 87 Hz.
- Panel C: f2 = 2 kHz @ f1 = 93 Hz, and Panel D: f2 = 4 kHz @ f1 = 98 Hz. The subject has normal-hearing (pure tone audiogram ≤ 20 dB HL), as shown in the inset audiogram (Panel A).

CONCLUSIONS

- ASSR compression estimates for levels above 30 dB HL are consistent with psychoacoustical data.
- ASSR I/O functions recorded in HI subjects reflect the loss of sensitivity at lower input levels.
- Correlation analysis between ASSR and DPOAE recordings showed more compressive functions in ASSR than in DPOAE.
- Reduced compression at levels close to threshold (≤ 20 dB HL) could not be estimated using ASSR. Longer recording times are required to estimate compression with ASSR near threshold.

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REFERENCES