Temporal suppression of long-latency click-evoked otoacoustic emissions

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This paper investigates suppression of click-evoked otoacoustic emissions (CEOAEs) using the same paradigm as Hine and Thornton (2002) in which a *suppressor*-click was presented close in time to a *test*-click. The experiment was repeated and the analysis extended to long-latency CEOAEs (duration > 20 ms), whereas the previous study only focussed on the 'short-latency' CEOAE (duration < 20 ms). The hypothesis was that suppression would continue on the long-latency CEOAE since this region is probably dominated by spontaneous OAEs (SOAEs) synchronising with the click stimulus. The results for five exemplary subjects showed that suppression remained on the long-latency CEOAE, indicating that both SOAEs and CEOAEs originate from the same cochlear nonlinearities, as suggested by Kemp and Chum (1980a). Further proof of suppression of long-latency SOAE components was sought in the spectral domain. A comparison of the magnitude of certain SOAE components in the suppressed and unsuppressed condition for a particular subject showed that suppression affected SOAE components in a similar way as the purely click-evoked OAE components.

INTRODUCTION

The active mechanisms in the cochlea which are responsible for the sharp auditory filters and compression of the dynamic range of the cochlear response are also believed to give rise to otoacoustic emissions (OAEs), which was first demonstrated by Kemp (1978). The majority of normally hearing adults (98 %) have measurable click-evoked otoacoustic emissions (CEOAEs) with emission spectra containing several discrete frequencies, known as dominant frequencies (Probst *et al.*, 1991). These dominant frequencies may be generated by oscillations at specific locations along the basilar membrane giving rise to frequency components in the CEOAE that are only present when evoked (Probst *et al.*, 1991). These oscillations are categorised by a relatively weak damping term, such that the dominant frequencies disappear about 20 ms after being evoked, creating 'short-latency' CEOAEs (Probst *et al.*, 1991). Alternatively, these dominant frequencies might be spontaneous otoacoustic emissions (SOAEs), synchronised by the click stimulus so that they become 'long-latency' CEOAEs with a duration longer than 20 ms (Probst *et al.*, 1991). SOAEs are often thought of as a consequence of particular locations along the cochlea having active mechanisms in a region of instability that can create self-sustaining oscillations (Eguiluz *et al.*, 2000).

The nonlinearity in otoacoustic emissions can be investigated by the suppression of an emission when presenting a *suppressor*-stimulus before the *test*-stimulus (Hine

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and Thornton, 2002, Kapadia and Lutman 2000b, Kemp and Chum, 1980a, Tavartkiladze *et al.*, 1994). The degree of suppression varies systematically with the timing and the level of the suppressor-click, being greatest for suppressor-clicks occurring 2-4 ms before the test-click (Hine and Thornton, 2002, Kapadia and Lutman, 2000b). Almost no suppression occurs when the suppressor-click leads the test-click by more than 6 ms, which was set by Hine and Thornton (2002) as the temporal nonlinearity limit of the cochlea. Both the studies of Kapadia and Lutman (2000b) and Hine and Thornton (2002) focussed on suppression of small time frames (3-5 ms) of the short-latency CEOAE (duration < 20 ms). The aim of the present study was to investigate suppression of the long-latency CEOAE response, as this region might be dominated by SOAEs. The hypothesis was that suppression would continue on the long-latency CEOAE if SOAEs and CEOAEs originate from identical cochlear nonlinearities inside the cochlea. The double-click suppression experiment set out by Hine and Thornton (2002) was repeated and extended to comprise a longer recording window. Furthermore, the behaviour of certain dominant SOAEs of one particular subject was investigated in the spectral domain to test the hypothesis.

MATERIALS AND METHODS

Recording CEOAEs

The stimuli used in the experiment were generated using Matlab and sent to an ADI-8Pro, a Hi-Precision 24 bit A/D-D/A converter. The signal level was controlled via a DT PA5 programmable attenuator. The stimulus was presented to the test subject via an ER-2 probe. Click response recordings were made with an ER-10dB low noise microphone and the preamplified signal was bandpass filtered between 600 Hz and 5000 Hz. The recorded signal was A/D converted and stored digitally. Test subjects were screened to ensure all had a hearing loss of less than 15 dB across the audiogram.

Experimental paradigm

Click stimuli were used in order to activate a broad range of frequency regions on the basilar membrane and to measure a spectrally rich CEOAE recording. The stimuli were presented at 65 dB peak equivalent sound pressure level (peSPL) to obtain a response in the nonlinear compressive region of the growth curve for CEOAEs (Probst *et al.*, 1991). The inter-click intervals (ICIs) used here were: ICI = [0.2 0.33 0.5 1 2 3.33 5 6 7 8] ms. The ICIs were identical to the values used by Hine and Thornton (2002), however the ICIs of 7 and 8 ms were added in order to test whether the suppression effect disappeared after the 6 ms ICI. The analysis window for the CEOAE recording was 39 ms long to investigate the longer latency click response associated with synchronised SOAEs. Hine and Thornton's experiment focussed only on subjects without SOAEs and on the short-latency click response and used a shorter analysis window of 17 ms.

For each ICI, two responses were measured: the double-click response and the single-click response with the click at the position of the second click of the double-click stimulus. The single-click response was measured again for every ICI value to reduce

the probe fitting error induced by movement of the test subject. For every measurement point, a minimum of 2000 click recordings were made in order to get to an averaged click response with a reasonable signal-to-noise ratio.

Five subjects with long-latency CEOAEs were tested in this experiment and their results are discussed here.

Post processing

An artefact rejection template was applied before the overall click response was separated in an early-latency linear response, associated with the ear-canal and middle-ear transfer function, and the CEOAE response, associated with the cochlea. This separation point was found to be $\tau s = 9$ ms after the last click onset and further analysis was only carried out on this CEOAE component (Verhulst, 2006).

The response to a single-click stimulus was referred to as the unsuppressed response and was aligned with the first click of the double-click stimulus case. The single-click response was then subtracted from the double-click response to remove the component due to the first click from the suppressed response and to maintain the component related to the ICI. Suppression in CEOAEs was measured as the difference between the derived-suppressed response and the unsuppressed response, as shown in Fig 1.

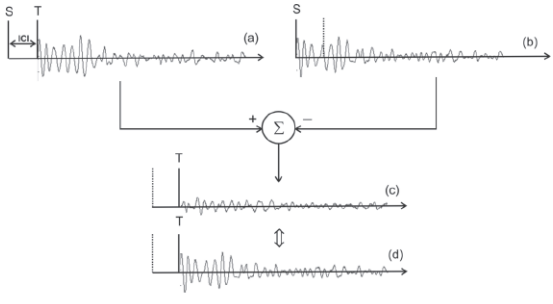

Fig. 1: Trace (a) and (b) show a pair of CEOAE responses. The stimulus in (a) contains a suppressor- and test-click separated by a certain ICI and the stimulus in (b) only contains a suppressor-click. The derived-suppressed response in (c) is obtained by aligning the S-click in (b) with the S-click in (a) and subtracting (b) from (a). The S-click component of the suppressed CEOAE in (c) has been removed, leaving the derived T-click response. Suppression is found as the difference between the unsuppressed CEOAE (d) and the derived-suppressed CEOAE (c) and is by definition positive when (c) is smaller than (d).

Suppression occurs because CEOAEs are inherently nonlinear; for linear signals, linear superposition would be applicable and the suppressed response would be identical to the unsuppressed result. Suppression levels were calculated as the difference between the rms level in dB SPL of the unsuppressed CEOAE and the rms level in dB SPL of the derived-suppressed CEOAE. The rms leels were calculated for 3-ms time frames starting from $\tau s = 9$ ms after the last click onset (for the unsuppressed and the derived-suppressed CEOAE) via:

$$L_{rms}\left(t_p\right) = 20 \cdot \log_{10}\left[\frac{1}{N-1}\sum_{i=1}^{N}\left(x_i - \mu\right)^2\right]^{1/2} \qquad \text{(Eq. 1)}$$

In equation (1), $N = 3\ ms \cdot f_s$, with N being the number of samples in the 3-ms analysis window. A value for L_{rms} is found for the time frame t_p of the CEOAE. x_i are the samples of the CEOAE and μ stands for the mean of the CEOAE within the considered 3-ms window.

RESULTS

Suppression levels for 3-ms time frames of the CEOAEs were averaged across five subjects and shown in Fig 2 as a function of ICI. The average data in the left panel of Fig 2 show that, for all short-latency time frames, suppression is maximal for inter-click intervals between 0.5 and 2 ms with values between 3 and 6 dB. Suppression decreases for ICIs longer than 2 ms and nearly disappears after an ICI of 6 ms is reached. The overall suppression levels per considered time frame follow the same tendency across ICI. The short-latency suppression data on the left panel of Fig 2 resembles the literature data quite well. For a stimulus level of 65 dB peSPL, Hine and Thornton (2002) found that maximal suppression was observed for a 1-2 ms inter-click interval. Kapadia and Lutman (2000b) also reported maximal suppression with a value around 5 dB when the suppressor click led the test click by 2 ms, for a stimulus level of 60 dB peSPL. Whereas Hine and Thornton limited their analysis to a maximal inter-click interval of 6 ms, the presented results on the left panel of Fig 2 reveal augmentation of the CEOAE when a suppressor-click is presented around 6 to 7 ms before the test-click. The augmentation at these ICIs is quite small but consistent with results from Tavartkiladze (1994) who also found a small 'overshoot' around an ICI of 7 ms, after which the influence of the inter-click interval between suppressor- and test-click on the CEOAE response disappeared.

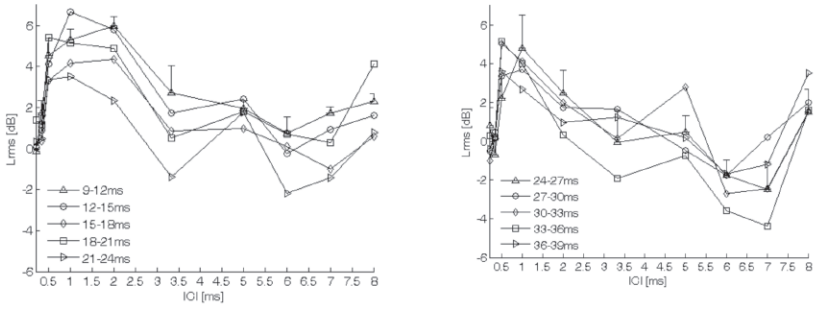

Fig. 2: Rms levels (L_{rms}) of suppression as a function of ICI, plotted for different short-latency time frames after the last click onset (left panel) and long-latency time frames (right panel), as an average over five test subjects. The standard error across subjects was plotted on the rms levels of the 9-12 ms time frame (left panel) and of the of the 24-27 ms time frame (right panel) and representative for all the time frames considered.

The right panel of Fig 2 shows suppression levels for the long-latency CEOAE region. It is apparent that the nonlinear suppression effect also works on the long-latency CEOAE, associated with the presence of SOAEs. Two main differences between the short- and long-latency suppression results are observed. Firstly, maximum suppression occurs at an ICI of 0.5 or 1 ms, which represents a small shift to narrower ICIs for maximal suppression of the long-latency CEOAE. Secondly, the augmentation observed for ICIs of 6 and 7 ms is more apparent and larger for the long-latency CEOAEs than for the short-latency CEOAEs. The augmentation results are in accordance with Tavartkiladze's data (1994), which showed a significant amount of augmentation of the CEOAE near an ICI of 6-7 ms for people with dominant SOAEs and thus long-latency CEOAEs.

The next step was to investigate the influence of temporal suppression on certain dominant SOAEs. The analysis needed to be carried out on single subjects, as the SOAE frequencies are unique for the person under test. The top panel of Fig 3 shows the suppression levels for a single subject and the bottom panel shows the augmentation levels in response to the double-click suppression experiment.

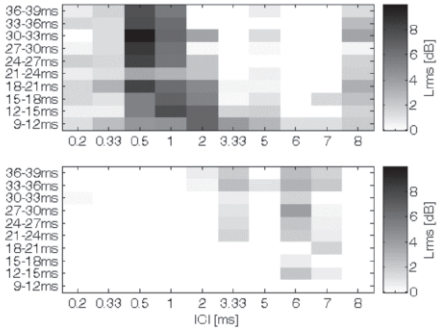

Fig. 3: Rms levels (L_{rms}) of suppression (top panel) and augmentation (bottom panel) per ICI, analysed in 3-ms long time frames for a single subject. The white regions in both panels represent the zero suppression/augmentation limit because the results are separated into a suppression and augmentation plot and only show positive levels.

The suppression and augmentation results of this test subject are representative for the average suppression data of Fig 2. Maximal suppression is found for inter-click intervals of 0.5, 1 and 2 ms and maximal augmentation is observed for ICIs of 6 and 7 ms. To investigate the behaviour of the dominant SOAEs related to the reported suppression and augmentation levels in Fig 3, the unsuppressed and suppressed CEOAE responses were analysed in the spectral domain for ICIs of 1 ms (representing maximal suppression) and 7 ms (representing maximal augmentation). First, the SOAE spectrum was determined with a recording of 5 minutes with no stimulus presented. After applying an FFT-averaging technique, the SOAE spectrum on the top panel of Fig 4 was obtained. This spectrum shows that the person under test has about eight dominant SOAE peaks in the region of 1500 to 2000 Hz and one distinct SOAE peak at 4580 Hz. The bottom panels of Fig 4 show the spectra of the whole CEOAE time series

(9-39 ms) for two inter-click intervals: 1 ms (left) and 7 ms (right). Suppression/Augmentation is the difference between the suppressed and unsuppressed CEOAE spectra. The obtained spectra in the bottom panel of Fig 4 are a combination of the dominant SOAE peaks of this person together with some distinct CEOAE frequencies that are evoked only when presenting a click.

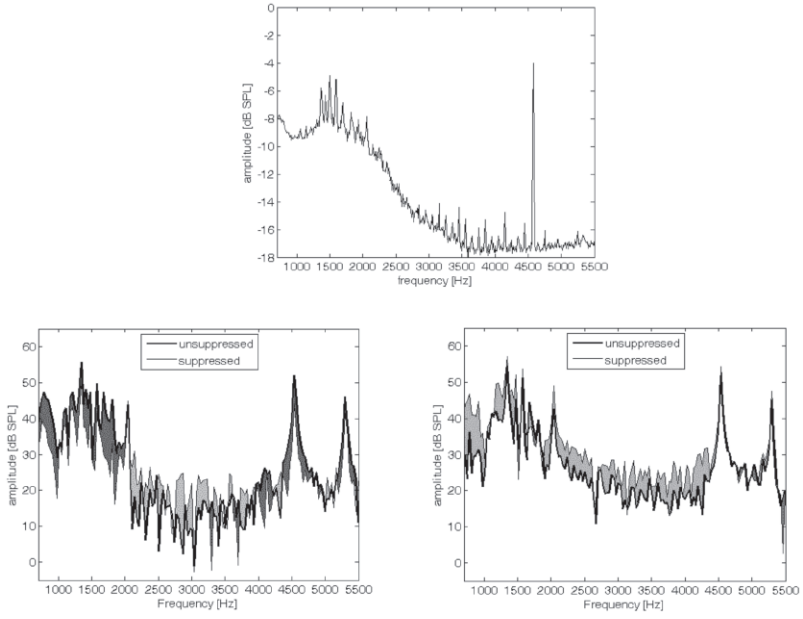

Fig. 4: Top panel: SOAE spectrum obtained by FFT-averaging 100-ms long blocks from the 5 minutes long SOAE recording, resulting in a spectral resolution of 10 Hz. The bottom panels show the spectra of the unsuppressed and suppressed CEOAEs for different ICIs. In this case the FFT was taken of the 30-ms long CEOAE window (9-39 ms after the last click onset), leading to a spectral resolution of 33 Hz. The bottom left panel shows the CEOAE spectra for when the ICI was 1 ms and the bottom right panel shows the CEOAE spectra for when the ICI was 7 ms. The dark gray areas on the bottom panels represent suppression of the spectrum (unsuppressed CEOAE > suppressed CEOAE) and the light gray areas represent augmentation of the spectrum by introducing a suppressor-click before the test-click.

The bottom left panel of Fig 4 shows the suppression in the spectra of the suppressed and the unsuppressed response for the ICI of 1 ms. Most suppression is found in the region between 700 and 2000 Hz (dark gray area, when unsuppressed CEOAE > suppressed CEOAE). Many dominant SOAE frequencies are present in this frequency region, as can be verified from the top panel of Fig 4, and most of them are suppressed by a certain amount for this ICI. Also the distinct SOAE peak at 4580 Hz is suppressed by about 5 dB. Most of the purely click-evoked peaks within the 700-2000 Hz region are also suppressed, but some augmentation (light gray area, when unsuppressed CEOAE < suppressed CEOAE) is reported in the 2000-4500 Hz region.

The suppression levels for 3-ms long windows for an ICI of 1 ms in Fig 3 can be explained by the spectra on the bottom left panel of Fig 4. Most energy within a CEOAE spectrum is usually found in the region of 1000-2000 Hz and around the dominant SOAE frequencies (Probst *et al.*, 1991). It is clear from the spectra on the bottom left panel of Fig 4 that components in these frequency regions get suppressed by presenting a suppressor-click 1 ms before the test-click. Suppression of frequency components within these dominant regions is the main reason for the overall suppression levels calculated over the 3-ms windows in Fig 3 because the energy in these regions dominates in the Lrms calculation. The longer-latency suppression levels of Fig 3 for an ICI of 1 ms can be explained by certain dominant 'long-latency' SOAE peaks that are suppressed. The bottom right panel of Fig 4 shows the spectra of the suppressed and the unsuppressed CEOAE response when presenting a suppressor-click 7 ms before the test-click. In this case, overall augmentation is observed over the whole spectrum, both for the dominant SOAEs as for the purely CEOAE frequencies. The augmentation levels for the 7 ms ICI of Fig 3 could be explained by the observed augmentation in the spectra. Again, it is believed that augmentation of some of the dominant SOAEs is responsible for the observed positive levels of augmentation for the long-latency regions of Fig 3 for an ICI of 7 ms.

SUMMARY AND DISCUSSION

The obtained short-latency suppression data the data found by Hine and Thornton (2002) and Kapadia and Lutman (2000). The experimental results showed that suppression was still present in the long-latency region of the response, i.e. 20 ms after the last click onset. Since this region is probably dominated by SOAE components synchronising with the CEOAE, it was indicated that both SOAEs and CEOAEs originate from the same nonlinearities inside the cochlea, as suggested by Kemp and Chum (1980a). The apparent similar origin of both types of emissions would imply that the same temporal effects influence their responses. This was further examined in the spectral domain where a relation was sought between the long-latency suppression and the behaviour of certain dominant SOAE components. It was shown that dominant SOAE components can be suppressed or augmented when presenting a suppressor-click before a test-click. This can explain why people with- and without SOAE components show the same overall short-latency suppression behaviour. The dominant SOAE components seem to behave identically to the purely evoked components in response to temporal suppression. However, the presented spectral analysis does not reveal anything about the temporal behaviour of the suppression of these dominant SOAE peaks since the whole recording window was needed to achieve sufficient spectral resolution. Do they stay suppressed/augmented over the whole 39 ms of the recording or do they only get suppressed/augmented for a certain time period? The hypothesis that long-latency suppression is being caused by dominant SOAE components would support the first approach. A time-frequency analysis of the dominant peaks in the CEOAE spectra should give a clearer view on the suppression/augmentation behaviour of these frequency components over time.

The amount of suppression, regardless of the considered time frame, increased to a maximum as the ICI increased to a value of 1-2 ms, where after suppression decreased and disappeared when the ICI reached 6 ms. Hine and Thornton (2002) set this 6 ms as the temporal nonlinearity limit of the cochlea. This limit was shown to hold in the experiments of the present study, but only for the reported suppression. Both the experimental suppression data and the literature data from Tavartkiladze *et al.* (1994) indicate a significant increase of the CEOAE response when the suppressor-click is presented 6-7 ms before the test-click. This augmentation disappears when the ICI increases further to 8-9 ms, where after neither suppression nor augmentation are found. The temporal nonlinearity limit should thus be extended to 9 ms to account for the 'overshoot' in the general suppression behaviour leading to a time-limit that comprises the complete nonlinear temporal suppression effect.

The results of the double-click suppression experiment did not give a clear view on the underlying generator mechanisms of the temporal suppression effect. Still, one idea is presented here. Since both SOAE and CEOAE components can be suppressed or augmented in response to the double-click suppression experiment, it is believed by the authors that suppression is associated with the underlying cochlear compression mechanism, rather than with the specific OAE generation mechanism. This would imply that both the SOAE and the purely CEOAE components have the same suppression across all frequencies. This is not observed in the data but it seems however, that there is a certain phase relationship for SOAE suppression with respect to the ICI and the natural period of a SOAE component. It is not clear whether this phase relationship exists for the purely CEOAE components as their behaviour could be masked by the presence of strong SOAE components. More experimental data are needed to test this phase relationship for both the SOAE and CEOAE components, and a separation in subject groups for people with and without SOAE components is necessary.

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