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Research paper

Further tests of the local nonlinear interaction-based mechanism for simultaneous suppression of tone burst-evoked otoacoustic emissions

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ABSTRACT

Tone burst-evoked otoacoustic emission (TBOAE) components measured in response to a 1 kHz tone burst (TB_1) are suppressed by the simultaneous presence of an additional tone burst (TB_2). This "simultaneous suppression of TBOAEs" has been explained in terms of a mechanism based on local nonlinear interactions between the basilar membrane (BM) travelling waves caused by TB_1 and TB_2. A test of this local nonlinear interaction (LNI)-based mechanism, as a function of the frequency separation (Δf, expressed in kHz) between TB_1 and TB_2, has previously been reported by Killan et al. (2012) using a simple mathematical model [Killan et al., Hear. Res. 285, 58–64 (2012)]. The two experiments described in this paper add additional data on the extent to which the LNI-based mechanism can account for simultaneous suppression, by testing two further hypotheses derived from the model predictions. Experiment I tested the hypothesis that TBOAE suppression is directly linked to TBOAE amplitude nonlinearity where ears that exhibit a higher degree of amplitude nonlinearity yield greater suppression than more linear ears, and this relationship varies systematically as a function of Δf. In order to test this hypothesis simultaneous suppression at a range of values of Δf at 60 dB peak-equivalent sound pressure level (p.e. SPL) and TBOAE amplitude nonlinearity from normal human ears was measured. In Experiment II the hypothesis that suppression will also increase progressively as a function of increasing tone burst level was tested by measuring suppression for a range of Δf and tone burst levels at 40, 50, 60 and 70 dB p.e. SPL. The majority of the findings from both experiments provide support for the LNI-based mechanism being primarily responsible for simultaneous suppression. However, some data were inconsistent with this view. Specifically, a breakdown in the relationship between suppression and TBOAE amplitude nonlinearity at $\Delta f = 1$ (i.e. when TB_2 was reasonably well separated from, and had a higher frequency than TB_1) and unexpected level-dependence, most notably at $\Delta f = 1$, but also where $\Delta f = -0.5$, was observed. Either the LNI model is too simple or an alternative explanation, involving response components generated at basal regions of the basilar membrane, is required to account for these findings.

1. Introduction

Transient-evoked otoacoustic emissions (TEOAEs) are physiological signals recorded in the ear canal in response to short duration acoustic stimuli (e.g. Probst et al., 1991; Robinette and Glattke, 2007). Most commonly, TEOAEs are recorded in response to clicks (i.e. click-evoked otoacoustic emissions, CEOAEs), or less commonly tone bursts (i.e. tone-burst-evoked otoacoustic emissions, TBOAEs). In both cases, the presence of a response is reliant on normal functioning of the physiological processes that enhance hearing at low sound levels, known as the cochlear amplifier (e.g. Ashmore et al., 2010). TEOAEs (CEOAEs more so than TBOAEs) are therefore used widely in clinical settings as an assessment of cochlear function.

Based primarily on CEOAE data, two components are thought to be present in the TEOAE response. The first component is characterised by its short latency and near-linear amplitude growth with stimulus level (e.g. Withnell and McKinley, 2005; Withnell et al., 2008; Goodman et al., 2011; Moleti et al., 2012). Because of its

Abbreviations: BM, Basilar membrane; CEOAE, Click-evoked otoacoustic emission; FFT, Fast Fourier transform; LNI, Local nonlinear interaction; p.e., peak-equivalent; SPL, sound pressure level; SOAE, Spontaneous otoacoustic emission; SSOAE, Synchronised spontaneous otoacoustic emission; TBOAE, Tone burst-evoked otoacoustic emission; TEOAE, Transient-evoked otoacoustic emission

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short latency, this component is assumed to be generated at basal regions of the basilar membrane (BM) via two possible mechanisms; nonlinear intermodulation distortion (e.g. Yates and Withnell, 1999; Carvalho et al., 2003; Withnell and McKinley, 2005; Notaro et al., 2007; Withnell et al., 2008) and linear reflection (Goodman et al., 2011; Moleti et al., 2012; Sisto et al., 2013). Recent modelling efforts suggest that the second of these mechanisms, the basal-reflection mechanism, is most likely to account for the short-latency, basal-source component (Moleti et al., 2013). The second, long-latency component exhibits compressive growth with stimulus level and frequency-dependent latency that is consistent with its generation via linear reflection at the peak region of the travelling wave (Shera, 2004; Sisto and Moleti, 2007, 2008; Withnell et al., 2008). The presence of this second component is compatible with the existence of compressive "generator channels" tonotopically distributed along the BM. TEOAE components are assumed to be generated locally within these channels (i.e. at their characteristic place) in response to stimulus components at the same frequency (e.g. Kemp and Chum, 1980; Tavartkiladze et al., 1994; Zettner and Folsom, 2003; Kalluri and Shera, 2007). This local, long-latency component dominates the overall TEOAE response at lower stimulus levels, whilst at higher levels the basal-source component dominates (Withnell et al., 2008; Goodman et al., 2011; Moleti et al., 2012; Sisto et al., 2013).

TEOAEs exhibit a number of suppression behaviours. Previous investigators (Yoshikawa et al., 2000; Killan et al., 2012) have shown that the amplitude of a TBOAE recorded from normal human ears in response to a 1 kHz tone burst (TB_1) can be suppressed by the simultaneous presence of an additional (equal level and phase) tone burst (TB_2). Specifically, components at 1 kHz in the response obtained to simultaneous presentation of TB_1 and TB_2 were reduced in amplitude compared to the corresponding components in the response obtained by (offline) summation of the individual responses to TB_1 and TB_2. Findings presented by Yoshikawa et al. (2000) show that where TB_2 had a higher centre frequency than TB_1, the amount of suppression increased as a function of decreasing frequency separation between the centre frequencies of TB_1 and TB_2 (referred to here as Δf and expressed in kHz). Killan et al. (2012) showed a similar dependence of suppression on Δf for higher frequency TB_2 (i.e. when $\Delta f = 0.5$, 1 and 2). In addition they demonstrated that greatest suppression tended to occur when TB_2 had the same frequency as TB_1 (i.e. $\Delta f = 0$), with a reduction in suppression observed when TB_2 had a lower frequency than TB_1 (i.e. $\Delta f = -0.5$).

Different mechanisms have been proposed to account for this "simultaneous suppression of TBOAEs". One view states that when TB_2 has a higher centre frequency than TB_1, its simultaneous presence somehow interferes with the generation of basal-source components in the response evoked by TB_1 (Xu et al., 1994; Yates and Withnell, 1999), although the detail of this interference is unclear. Further, the finding that suppression progressively increases as Δf decreases so that maximum suppression was measured when TB_1 and TB_2 had the same frequency, is at odds with the involvement of basal-source components. If basal-source components were responsible for suppression then it could be argued that maximum suppression would occur when TB_2 had a higher frequency than TB_1. Similarly, it is not clear how TB_2 is able to cause suppression of TB_1 response components when TB_2 had a lower frequency than TB_1. An alternative mechanism states that simultaneous suppression of TBOAEs results from local nonlinear interactions between the BM travelling waves caused by TB_1 and TB_2 (Killan and Kapadia, 2006; Killan et al., 2012). This local nonlinear interaction (LNI)-based mechanism assumes the dominant component of the TBOAE response is the long-latency component that originates from compressive generator channels located at the tonotopic place. Specifically, Killan et al. (2012) argued if TB_1 and TB_2 are closely spaced in frequency (i.e. $\Delta f = -0.5$ or 0.5) then their travelling waves would overlap following simultaneous presentation. As a result, both TB_1 and TB_2 will cause excitation of generator channels at BM sites tuned to those between the centre frequencies of TB_1 and TB_2. These generator channels will therefore experience increased excitation with simultaneous presentation compared to individual presentation of TB_1 and TB_2. However, because generator channels are compressive, TBOAE components output from these channels will have smaller amplitude than the corresponding components in the offline sum of the individual responses, and suppression of the simultaneous response will be observed. Greatest suppression would be expected when TB_1 and TB_2 had identical centre frequencies, with TB_1 and TB_2 well-separated in frequency causing least suppression. This LNI-based mechanism is able to account for the Δf-dependence of suppression, including the finding that a lower frequency TB_2 was able to cause suppression of the TB_1 response. The LNI-based mechanism is also similar to mechanisms proposed to explain other TEOAE suppression phenomena (Kemp and Chum, 1980; Kapadia and Lutman, 2001; Harte et al., 2005; Lineton et al., 2006; Thornton et al., 2006).

The extent to which the LNI-based mechanism can account for simultaneous suppression of TBOAEs has previously been tested using a simple mathematical model (Killan et al., 2012). This model incorporated a single generator channel represented by a static gammachirp filter with peak frequency at 1.2 kHz, in series with a static compressive input–output function. This input–output function allowed the nonlinearity of the generator channel to be varied in accordance with reports of TBOAE amplitude nonlinearity reported in the literature. Pairs of TB_1 and TB_2 at a range of values of Δf were applied to the model to obtain prediction of simultaneous suppression. The aim of the model was to provide a simple indication of the LNI-based mechanism for a single generator channel located in the region of 1 kHz, rather than accurately represent the physiological processes that occur in the cochlea following simultaneous stimulation TB_1 and TB_2. The predictions of the model were compared with TBOAE suppression data recorded from normal human ears for the same values of Δf. A close agreement between the model predictions and mean TBOAE suppression was taken to indicate that the LNI-based mechanism was responsible for simultaneous suppression of TBOAEs.

Though not tested by Killan et al. (2012), their model also predicted that suppression governed by the LNI-based mechanism would be dependent on generator channel nonlinearity so that larger amounts of suppression would be expected when the generator channel was more nonlinear, compared to when the channel was relatively linear. Further, the model predicted that this channel nonlinearity-dependence would vary as a function of Δf so that for the same increase in nonlinearity, greater levels of suppression would be evident at smaller values of Δf compared to higher values of Δf. This is understood in terms of suppression being dependent on generator channel nonlinearity *and* the amount of overlap between the excitation patterns caused by TB_1 and TB_2. A manifestation of generator channel nonlinearity is the nonlinear growth of TBOAE amplitude with increasing tone burst level, typically observed via TBOAE level functions (e.g. Rutten, 1980; Johnsen and Elberling, 1982; Elberling et al., 1985; Norton and Neely, 1987; Epstein and Florentine, 2005). It therefore follows that ears exhibiting a high degree of TBOAE amplitude nonlinearity should yield greater suppression than ears exhibiting less nonlinearity, and that this relationship will vary systematically as a function of Δf.

A second, related prediction can also be derived from the relationship between suppression and TBOAE amplitude nonlinearity. Because TBOAE amplitude nonlinearity is compressive (i.e. it