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Research paper

Auditory steady-state responses as neural correlates of loudness growth

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ABSTRACT

The aim of this study was to find an objective estimate of individual, complete loudness growth functions based on auditory steady-state responses. Both normal-hearing and hearing-impaired listeners were involved in two behavioral loudness growth tasks and one EEG recording session. Behavioral loudness growth was measured with Absolute Magnitude Estimation and a Graphic Rating Scale with loudness categories. Stimuli were sinusoidally amplitude-modulated sinusoids with carrier frequencies of either 500 Hz or 2000 Hz, a modulation frequency of 40 Hz, a duration of 1 s, and presented at intensities encompassing the participants' dynamic ranges. Auditory steady-state responses were evoked by the same stimuli using durations of at least 5 min. Results showed that there was a good correspondence between the relative growth of the auditory steady-state response amplitudes and the behavioral loudness growth responses for each participant of both groups of listeners. This demonstrates the potential for a more individual, objective, and automatic fitting of hearing aids in future clinical practice.

1. Introduction

Loudness growth functions characterize the relation between sound intensity and loudness (Marks and Florentine, 2011). They are highly listener dependent, and thus offer unique information about the hearing of an individual. To date, most prescription rules for non-linear amplification include some aspects of loudness normalization, i.e. they try to make the loudness of the amplified sounds similar to the loudness for normal-hearing listeners listening to the same sound (Dillon, 2012). However, complete loudness growth functions are usually not measured in clinical practice because the procedures for measuring them are time-consuming, complicated, demand active cooperation of the client, are often perceived as difficult by the client, and large variability across people and measurement techniques have been described (Al-Salim et al., 2010; Elberling, 1999).

Examples of loudness growth measures that were used in the past for fitting hearing aids are LGOB or Loudness Growth in half-Octave Bands (Allen et al., 1990), the IHAFF or Independent Hearing Aid Fitting Forum protocol, also known as the Contour Test (Cox, 1995; Valente and Van Vliet, 1997), and ScalAdapt (Kiessling et al., 1996). In these procedures the client needs to estimate the loudness of different sounds, based on loudness categories ranging from not audible or very soft to uncomfortably loud or too loud. The gain of the hearing aid is adjusted to try to achieve normalized loudness. In the ScalAdapt procedure, loudness growth is measured while the client is wearing the hearing aid and its parameters are adaptively adjusted until the client gives a desired loudness rating.

Loudness categories are perceived as simple and easy to understand for inexperienced participants due to their meaningful labels, and previous experience in loudness scaling has no influence on the loudness judgments (Launer, 1995). Because of these factors categorical loudness scaling is more frequently preferred for clinical practice compared to other loudness growth procedures (Marks and Florentine, 2011; Launer, 1995), even though their reliability and validity have been questioned (Al-Salim et al., 2010; Elberling, 1999). Many loudness scales in the literature have discrete loudness categories, and the number of categories has been the subject of discussion, with too few categories leading to response biases. In the procedure described by Allen et al. (1990), many participants

Abbreviations: ABR, Auditory Brain Stem Response; AME, Absolute Magnitude Estimation; ASSR, Auditory Steady-State Response; DPOAE, Distortion-Product Otoacoustic Emissions; EEG, Electroencephalogram; GRS, Graphic Rating Scale; HI, Hearing-Impaired; MSE, Mean Square Error; NH, Normal-Hearing; OAE, Otoacoustic Emissions

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reported that the number of categories, i.e. 6, was insufficient. As a solution, one can add intermediate response categories without labels (Brand and Hohmann, 2001), or use a continuous visual-analogue scale or a graphic rating scale, which is a visual-analogue scale with categories added as guidelines (Marks and Florentine, 2011; Svensson, 2000).

Other procedures have been described to measure loudness growth such as Absolute Magnitude Estimation (AME). The AME task is a classical method for measuring loudness, often proposed as the most direct and effective method (Hellman and Meiselman, 1990; Marks and Florentine, 2011).

Attempts have been made to find an objective, more automatic, and physiological correlate of loudness growth functions using different kinds of measures. While these measures have sources at different stages of the auditory pathway, at present, it is not fully understood at which stage of the auditory pathway the loudness coding is complete for different stimuli.

Otoacoustic emissions (OAEs) have been assessed as one correlate of loudness growth. OAEs might be practical to use because they are fast to acquire. OAEs are generated by the outer hair cells in the cochlea in response to acoustic stimuli, and can be measured in the ear canal. Thus, this approach is based on the assumption that the perception of loudness is mainly determined at the level of the outer hair cells, while it is likely that loudness is also affected by other auditory processes for which OAEs are insensitive, such as processes at the level of the inner hair cells, synaptic and neural functions, and central auditory processes. Loudness growth has been linked to both distortion-product otoacoustic emissions (DPOAE) (Neely et al., 2003; Müller and Janssen, 2004; Rasetshwane et al., 2013; Thorson et al., 2012) and tone-burst otoacoustic emissions (Epstein and Florentine, 2005; Epstein and Silva, 2009; Silva and Epstein, 2010, 2012) for normal-hearing and hearing-impaired participants. However, correlations between loudness and DPOAEs were only found if multiple linear regression analyses utilizing the entire DPOAE input-output function were used instead of individual DPOAE input/output function parameters. Furthermore, the DPOAE data showed large inter-subject variability, with good agreement only with group medians. Other disadvantages have been described for the use of OAEs. First, the use of OAEs is limited to individuals with mild-to-moderate hearing loss, since OAEs are absent for individuals with greater degrees of hearing loss. Second, the reliability of OAE measurements is affected by several factors, such as calibration errors, probe-tip placement, recording instruments, and environmental noise (Keppler et al., 2010). Third, at frequencies near the ear-canal resonance, such as 4 kHz, loudness estimates using OAEs are unreliable (Silva and Epstein, 2010, 2012).

Another possible correlate of loudness that has been extensively investigated is the auditory brain stem response (ABR), an auditory evoked potential. Correlations between loudness and ABR amplitude or latency growth functions were either low and not significant for all participants (Wilson and Stelmack, 1982), or were only significant when averaged results were used across participants or test trials. For normal-hearing participants, most of the studies do not show a direct link between the ABR and loudness growth (Babkoff et al., 1984; Darling and Price, 1990; Davidson et al., 1990; Pratt and Sohmer, 1977; Serpanos et al., 1997). Davidson et al. (1990) analyzed the ABR wave V amplitude, and Serpanos et al. (1997) the ABR wave V latency, while the other studies investigated the amplitudes and latencies of multiple waves (I-VI). Serpanos et al. (1997) found a relation between the ABR wave V latency and loudness growth for participants with a flat hearing loss. However, there was no such relation for participants with a sloping hearing loss. This study also used averaged group results. Furthermore, the use of ABR has two major disadvantages. First, the waveform of the ABR is often subjectively labeled. Second, there is a lack of frequency specificity, since ABRs are often evoked by click stimuli. To address these problems, Silva and Epstein (2010, 2012) developed an automatic analysis and segmentation method to use with ABRs evoked by 1- and 4- kHz tone-burst stimuli and reported reliable loudness growth estimates if residual noise levels, i.e. the amount of noise left in the final averaged waveform that affects the ABR amplitude estimation, are controlled. Residual ABR noise levels were estimated through the weighted nonstationary fixed-multiple-point (WNS FMP) statistic, and used as weights in a subsequential non-linear fit with a polynomial or with shifted versions of the INEX function. In summary, mixed results were reported for the relation between ABRs and loudness growth, with many studies finding a lack of correspondence.

Since a lack of correspondence with loudness growth was often found with OAEs and ABRs, loudness may not be fully determined by neural activity at the level of the outer hair cells or the brain stem. Evidence regarding a cortical basis of loudness was suggested by Heinz et al. (2005) and described by Thwaites et al. (2016). Heinz et al. (2005) found that the auditory nerve rate functions of cats with noise-induced hearing loss were inconsistent with the hypothesized neural correlates of loudness recruitment. Thwaites et al. (2016) investigated the location of cortical entrainment to two realistic models of sound magnitude, i.e. the instantaneous and short-term loudness models. Instantaneous loudness is assumed to be the loudness after transformation at peripheral levels, and is already represented in the brain but not yet available to conscious perception, while short-term loudness is formed by running temporal integration of the instantaneous loudness. The location of cortical entrainment to instantaneous loudness was found in Heschl's gyrus. It was suggested that it is moved or copied to the dorsal lateral sulcus and from there back to Heschl's gyrus. Cortical entrainment to the short-term loudness was found in both the dorsal lateral sulcus and superior temporal sulcus.

Correlations between loudness growth and objective measures based on sources further in the auditory pathway than the outer hair cell, auditory nerve, or brain stem have also been investigated. Madell and Goldstein (1972) found high correlations across participants between the peak-to-peak middle-latency response amplitudes and loudness estimates, with correlation coefficients of 0.94, 0.85, and 0.75 for P0-Na, Na-Pa, and Pa-Nb, respectively. However, no significant correlations were found for individual participants. Pratt and Sohmer (1977) found no correlations between loudness estimates and the cortical responses P1-3 evoked by a series of click stimuli with peak energy in the 3–5 kHz range. They proposed that the loudness estimate is likely determined by neural activity that is not registered by the recording technique and that another set of neural parameters might be required to estimate loudness.

Several fMRI studies support the hypothesis that the loudness percept is not complete before the level of the auditory cortex. For normal-hearing participants, significant correlations between loudness and the extent and the magnitude of cortical activation or the fMRI blood oxygen level dependent signal (BOLD-signal) were found at the auditory cortices, but not at any lower sources of the auditory pathway such as the inferior colliculus or the medial geniculate bodies (Hall et al., 2001; Röhl et al., 2011; Röhl and Uppenkamp, 2012; Uppenkamp and Röhl, 2014). For participants with a high-frequency hearing loss, steeper growth in the magnitude of the cortical responses with sound intensity was found for high-frequency FM-tones (4–8 kHz) than for low-frequency FM-tones (0.5–1 kHz), which was interpreted as a correlate of the psychoacoustic effect of loudness recruitment (Langers et al., 2007).

The auditory steady-state response (ASSR) might be a good objective correlate of loudness growth. The ASSR is a stationary