Neuroscience Letters 559 (2014) 132–135

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Efferent-mediated reduction in cochlear gain does not alter tuning estimates from stimulus-frequency otoacoustic emission group delays

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HIGHLIGHTS

- Contralateral noise variably suppresses stimulus-frequency otoacoustic emissions.
- Tuning estimates based on otoacoustic delays are unchanged by contralateral noise.
- The amount of contralateral suppression does not affect tuning estimates.

ARTICLE INFO

Article history:
Received 11 July 2013
Received in revised form 24 October 2013
Accepted 29 November 2013

Keywords:
Cochlea
Contralateral suppression
Cochlear filters
Humans
Medial olivocochlear efferent neurons
Otoacoustic emissions

ABSTRACT

The existence of efferent feedback from cortical and subcortical brain centers to the hair cells of the cochlea has been recognized for many years, but the role that efferent neurons play in hearing is not completely known. Stimulation of medial olivocochlear (MOC) efferent neurons suppresses sound-evoked basilar membrane responses and changes the tuning of single auditory nerve fibers in animal models. Both of these effects are linked to a MOC-induced reduction in the gain of the cochlear amplification provided by outer hair cells. To non-invasively examine the link between cochlear suppression and tuning in humans, stimulus-frequency otoacoustic emissions (SFOAEs) were recorded in conditions with and without contralateral acoustic stimulation (CAS) from 28 normal-hearing participants. SFOAEs were measured using clusters of closely-spaced probe-tone frequencies centered near 1.4 and 2.0 kHz. An index of cochlear tuning, Q_{ERB}, was calculated based on measures of SFOAE group delay at both 1.4 and 2.0 kHz. A statistically significant ($p < 0.01$) decrease in SFOAE levels acquired during CAS was detected only for the SFOAE cluster centered at 2 kHz. No statistically significant differences in Q_{ERB} were found between conditions with and without CAS at 1.4 and 2.0 kHz. These findings suggest that in humans, tuning based on SFOAE group delay estimates is not appreciably altered at cochlear locations with MOC efferent-induced reductions in cochlear gain.

1. Introduction

The role that feedback originating from higher brain centers and terminating on the hair cells of the mammalian cochlea plays in hearing has been debated for well over half a century. Medial olivocochlear (MOC) efferent neurons, with their cell bodies located near the superior olivary complex of the brainstem, target the outer hair cells (OHCs) for synaptic innervation. They can be activated by electrical shocks and ipsi- or contralateral acoustic stimulation (CAS), and produce several well-known effects. In animal subjects, firing of MOC neurons both suppresses basilar membrane (BM) displacement [7] and elevates auditory nerve (AN) fiber thresholds [13,20], evoked by characteristic frequency (CF) tones. Tuning curves of AN fibers are also modified, with a decrease in tuning in most fibers and an increase in tuning in some low-frequency (<2.0 kHz) fibers [13]. The parallel effects of BM suppression and loss of AN tuning produced by MOC efferent neurons are replicated in computational cochlear models that decrease the gain of OHCs [5,6]. Evidence indicates that OHC oscillations amplify BM displacements in a level-dependent manner, with more amplification provided for CF tones presented at low levels near to the threshold of neighboring AN fibers and less amplification provided for CF tones presented at higher levels [8]. Synaptic transmissions

Abbreviations: AN, auditory nerve; BM, basilar membrane; CF, characteristic frequency; CAS, contralateral acoustic stimulation; MOC, medial olivocochlear; OAE, otoacoustic emission; PTC, psychophysical tuning curve; OHC, outer hair cell; SFOAE, stimulus-frequency otoacoustic emission.

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http://dx.doi.org/10.1016/j.neulet.2013.11.059

from MOC efferent neurons hyperpolarize OHCs by increasing the conductance of calcium-activated potassium channels, ostensibly leading to a decrease in the gain of the OHC amplifiers [9].

Because the effects of MOC efferent stimulation cannot be directly assessed in human cochleae for ethical reasons, several non-invasive methods have been developed to indirectly examine them. A common paradigm entails measurement of psychophysical or physiological responses from an ear during conditions with and without CAS. There is some evidence that CAS broadens psychophysical tuning curves (PTCs) at 4.0 kHz and sharpens PTCs at 1.0 kHz, consistent with the findings from AN fiber tuning curves in animal subjects [15,19]. In contrast, other evidence indicates that CAS has no effect on PTCs at 4.0 kHz and broadens PTCs at 0.5 kHz [14]. Notably, direct comparisons from the results of PTC studies to findings from AN tuning curves are limited by: (1) the PTC procedure involves presenting the test tone and masker to the same ear, unlike the AN tuning curve method where only the test tone is presented, and (2) the PTC procedure requires an aggregate response reflecting the combined output from the entire auditory nervous system of the individual being tested whereas AN tuning curves are obtained from single fibers at an early stage of peripheral processing. It is feasible that greater insight concerning how MOC efferent neurons modulate tuning characteristics could be garnered by monitoring responses more proximal to the AN fibers from human ears.

Otoacoustic emissions (OAE), which are sounds emitted from the cochlea that can be recorded in the ear canal using a sensitive microphone, are often selected as a proxy measure of BM mechanics in lieu of the inability to make direct BM observations in humans. Proposed to partly derive from OHC oscillations and linear reflections in the cochlea, they are linked to the BM amplification provided by these cells [4,16]. A number of effects induced by MOC efferent neurons seen at the level of the BM in animal subjects can be observed in OAEs from human ears measured in conditions designed to trigger activity of the MOC efferents, including the suppression of OAE amplitude [11] and decompression of OAE growth functions [2] produced by CAS. Stimulus-frequency OAEs (SFOAEs) are preferred over other OAE types to study the effects of MOC efferent activation as they are evoked by frequency-specific continuous tones. At low levels, these stimuli do not elicit their own MOC efferent activity, which avoids the complication of determining if these responses are suppressed by the stimulus or by CAS [12]. In addition, SFOAE-derived estimates of BM tuning have been employed in two previous studies examining the MOC efferents [3,10]. SFOAE delay an index predominantly of the cochlear travel time of the emission, was compared in each of these studies in conditions with and without CAS. The rationale for using SFOAE delays to examine potential changes in cochlear filter bandwidth caused by MOC efferent neurons comes from filter theory (i.e., MOC efferent-induced broadening of cochlear filters results in a decrease in the SFOAE delay) [17]. Although trends in both studies indicated that SFOAE delays were reduced in the condition with CAS, the difference in SFOAE delays between the conditions when SFOAEs were obtained with and without CAS were not statistically significant in either study. While these studies were informative, several questions remain concerning how MOC efferent activation in the cochlea may (or may not) be manifested through SFOAE delays. For example, past research did not directly examine if the reduction in SFOAE delays varied with the amount of suppression in SFOAE level produced by CAS. CAS-induced suppression of SFOAEs is known to be highly variable when SFOAEs are measured with high resolution of the stimulus tones [1].

An approach to indirectly examine cochlear tuning developed by Shera and colleagues [18] involves the calculation of the Q_{ERB} sharpness of tuning factor, which takes into account both the center frequency of the filter and the SFOAE group delay. The cochlear analysis of sounds results in a logarithmic frequency-to-place mapping for most of the audible frequency range, complicating the comparison of tuning based on filter bandwidth across different cochlear locations. An advantage of the Q_{ERB} technique is that allows for more direct comparisons of sharpness of tuning at different center frequencies. The aim of this study was to examine if a variable amount of CAS-induced suppression of SFOAEs in different cochlear locations influenced indirect estimates of cochlear tuning (Q_{ERB}) based on SFOAE delays.

2. Materials and methods

Twenty-eight young adults (24 females, four males) aged 22–33 y participated in this study. The experimental procedures were approved by the Institutional Review Board of the University of Memphis and written informed consent was obtained from all of the participants. Each participant enrolled in the study had hearing thresholds at or better than 20 dB HL for the standard audiometric frequencies from 0.25 kHz to 8.0 kHz and normal middle-ear function assessed via tympanometry. A Mimosa Acoustics Hear ID™ system interfaced with an Etymotic Research ER 10 C probe assembly was used to measure SFOAEs from the right ear of each participant. The measurements took place in a double-walled sound-treated enclosure. The probe assembly containing two transducers and a microphone was placed in participant ear canals using a soft foam tip. SFOAEs were acquired with probe and suppressor tones following the suppression method of Shera and Guinan [16]. Each probe tone was presented at 40 dB SPL and the suppressor tone was higher in frequency by approximately 47 Hz and at a level of 55 dB SPL. The "SF clusters" protocol was selected from the Hear ID™ v. 3.5 software, which utilizes five closely-spaced probe tones with each probe tone separated from the next by approximately 12 Hz in each cluster. The probe frequencies in the 1.4 kHz cluster were 1.371, 1.382, 1.394, 1.406, and 1.417 kHz. The probe frequencies in the 2.0 kHz cluster were 1.980, 1.992, 2.003, 2.015, and 2.027 kHz. SFOAE measurements were completed first without presentation of CAS. Next, an insert earphone was coupled to the left ear of each participant and broadband noise bursts (duration = 0.8 s, level = 60 dB SPL) were presented while SFOAEs were measured in the right ear. This noise level is effective at evoking MOC efferent activity and is below levels of noise usually required to evoke the middle-ear muscle reflex in humans [12].

The frequency resolution offered by the SF clusters protocol enabled SFOAE phase to be unwrapped without aliasing. The unwrapped phase as a function of probe frequency was plotted and fitted by a linear function. Calculation of the SFOAE group delay (in ms) utilized the negative slope of the fitted line, in accordance with previous research [3,10,17]. Group delays were calculated for SFOAEs measured in conditions with and without CAS. In order to be considered for further analysis, SFOAE data from individual participants had to meet the following criteria: (1) the SFOAE signal-to-noise ratio was ≥9 dB for at least three out of five test frequencies within a cluster, (2) the group delay estimate error was <2.0 ms, and (3) the sign of the group delay estimate was positive. These criteria were adopted from recommendations from a previous report [21]. Data from 24 participants at the 1.4 kHz cluster and 25 participants at the 2.0 kHz cluster met these criteria and were used in the statistical analyses. The formulae developed by Shera et al. [18] were used to derive the Q_{ERB} estimates from the SFOAE group delays and are shown below as Eqs. (1) and (2).

$$N_{SFOAE} = f \times \tau_{SFOAE} \quad (1)$$

$$Q_{ERB} = k\frac{N_{SFOAE}}{2} \quad (2)$$