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Effect of smoking on transient evoked otoacoustic emissions and contralateral suppression

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Abstract

Objective: The present study compared the amplitudes of transient evoked otoacoustic emissions (TEOAEs) and efferent suppression in smokers and non-smokers taking age into consideration.
Methods: Fifty smokers and fifty non-smokers who had normal hearing sensitivity with age range of 20–69 years were considered for the present study. TEOAEs were measured in both the groups of subjects across different age groups. The functioning of the efferent auditory system was evaluated in both the groups by recording the TEOAEs in the presence of a contralateral white noise (CWN) of 70 dB SPL.
Results: Age did not have a significant effect on the TEOAEs amplitude in both the groups of subjects. However, the TEOAEs amplitude was significantly reduced in smokers compared to non-smokers. The results found a significant effect of age on the amplitude of efferent suppression in smokers, however, no significant effect was found in the non-smokers group.
Conclusions: It was found that the difference in the amplitude of efferent suppression in smokers was significantly greater for each age group between 20 and 49 years compared to each age group from 50 to 69 years. Results have important implication on the damage to the cochlear structures from smoking.

Keywords: Smoking; Otoacoustic emissions; Contralateral suppression; Outer hair cells

1. Introduction

Cigarette smoking contributes significantly to cardiovascular morbidity resulting in damage to the hearing structures. Smoking reduces blood supply by vasospasm induced by nicotine and may increase the risk of diseases such as stroke, heart disease, obstructive pulmonary disease and peripheral vascular disease. Since the middle ear connects with the upper respiratory tract through the Eustachian tube, it is reasonable to consider potential harm as a result of smoking. Studies have shown that exposure to cigarette smoke was associated with a 4.9 times increase in the prevalence of hearing deficits and 75% of the cases of hearing loss were statistically attributable to this cause. Also, exposure to the cigarette smoke is a cause of middle ear effusion and hearing loss in children [1].

It may be observed that the damage that occurs to the outer hair cells (OHCs) due to smoking resulting in reduced hearing sensitivity has been extensively studied [2,3]. However, in these studies smokers had hearing loss that may be as a result of damage to the OHCs. Thus, it may be difficult to interpret the early damage that may occur to the OHCs without actually resulting in decreased hearing sensitivity. Thus the present study aims to investigate the functioning of OHCs when the hearing sensitivity is within normal limits in smokers to detect if there is an early damage to the OHCs.

Studies have shown that otoacoustic emissions (OAEs) are used as a reliable tool to detect the functioning of the OHCs in the cochlea [4,5]. The OAEs that are elicited using transient stimuli is called as transient evoked otoacoustic emissions (TEOAEs). TEOAEs are frequency-dispersive responses that seem to originate from the OHCs [4,6]. The

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OHCs receive rich efferent innervations from the central nervous system and the changes in OAEs with the external stimulation reflect the influence of the CNS on the outer hair cell activity. OAEs have provided new possibilities in investigation of the efferent system suppression effect of the medial olivo-cochlear bundle (MOCB) [7]. Studies have also shown that age has an effect on the TEOAE amplitude [8,9] and on the efferent suppression [10,11] in subjects with normal hearing.

However there is a dearth in literature comparing TEOAEs amplitude and the efferent suppression in smokers and non-smokers having normal hearing sensitivity. The present study investigated the effect of smoking and age on otoacoustic emission amplitude and contralateral efferent suppression in subjects with normal hearing. This may help in identifying the possible factors responsible for damage to the cochlea and may assist us in understanding the functioning of the efferent system in smokers and non-smokers.

2. Patients and method

2.1. Subjects

A total of 50 smokers and 50 non-smokers were evaluated in this study. Subjects having normal hearing were randomly selected in each age group based on the history of smoking/no-smoking. Subjects were divided into five groups ($n = 10$ in each group), representing the following age ranges in years: 20–29 (mean = 24.7), 30–39 (mean = 36.2), 40–49 (mean = 47.1), 50–59 (mean = 54.3) and 60–69 (mean = 66.7). All subjects were males. None had any history of ear disease, significant noise exposure, ototoxicity, familial hearing loss or cardiovascular disease. All subjects had normal blood pressure levels and there was no history of diabetes. The amount of smoking was calculated in terms of the number of pack-years. Pack-years of smoking were defined as the number of packs (one pack equal to 20 cigarettes) smoked per day, multiplied by the duration of smoking in years. The smokers group in the present had a history of a minimum of 1 pack-year to a maximum of 5 pack-years. No subject in the control group had ever been a smoker. All subjects had audiometric thresholds at or better than 15 dB HL. This criterion was kept in order to ensure that hearing loss did not have an effect on the otoacoustic emissions amplitude. All subjects had normal middle ear functioning. The purpose of the study was explained to the subjects, and their consent was obtained for participation in the study.

2.2. Method

2.2.1. Procedures

(1) History taking and otological examination were carried out on all subjects before audiological testing.
(2) *Basic Audiological evaluation*. Audiometric thresholds were measured using a calibrated Madsen OB922 clinical audiometer [12]. Air conduction thresholds were measured for frequencies from 250 to 8000 Hz using Telephonics TDH39 earphones. Bone conduction thresholds were obtained for frequencies from 250 to 4000 Hz using a Radio Ear B71 bone vibrator. The audiometric thresholds were measured using the modified Hughson-Westlake method [13].
(3) *Immittance evaluation*. Tympanometry was carried out using a Grason-Stadler TympStar middle ear analyzer to rule out middle ear pathology.
(4) *Transient evoked otoacoustic emissions*. Transient evoked otoacoustic emissions (TEOAEs) were recorded in all subjects using the ILO292 Otodynamic Analyzer. The eliciting stimuli consisted of a non-linear click delivered at about 80 dB peak SPL in the ear canal. The spectrum analyzer was triggered at 4 ms after presentation of the stimuli to avoid acoustic ringing of the input stimuli, and the temporal window was set at 20 ms. A total of 260 averages were recorded. TEOAEs were considered present when the reproducibility and stability was >80%. TEOAEs were recorded with and without continuous contralateral white noise (CWN). To potentially minimize the contribution of the middle ear reflex, the CWN was presented at 70 dB SPL using a Maico 52 dual-channel clinical audiometer via an insert ear phone to the subject's ear. To ensure that a constant CWN intensity was maintained, the sound pressure level of the continuous CWN was monitored during TEOAEs testing by using a probe microphone (ER-10B) placed deep in the external auditory canal.

All testing was carried out in a double-walled, sound treated booth within permissible noise limits [14].

3. Results

3.1. Audiometric thresholds

In order to control the effect of hearing sensitivity on the TEOAE amplitude, audiometric evaluation was carried out on both the groups of subjects at octave and mid-octave frequencies of 250, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000 and 8000 Hz. Analysis of variance (ANOVA) was used to effect of age, frequency and smoking on the audiometric thresholds. Results revealed no significant effect of age and frequency on the audiometric thresholds ($p > 0.05$). The independent sample 't' test was used to know if there was a significant difference in the audiometric thresholds between smokers and non-smokers. Results revealed no significant difference between the two groups ($p > 0.05$).

3.2. TEOAEs test results

TEOAEs were present in 100% of subjects in each age group. The mean amplitude of TEOAE at different age groups in smokers and non-smokers are depicted in Fig. 1.