The bounce Event in Humans: Parameter Dependence

Irina Burdzgla1, Markus Pietsch1, Zurab Gamagebeli1, Michael Tushishvili1, Gert Hofmann2, Zurab Kevanishvili1
1Centre of Audiology and Hearing Rehabilitation, Tbilisi, Georgia
2Otorinolaryngological Clinic, Technical University, Dresden, Germany
3Ludwig-Maximilian-University, Munich, Germany

Introduction
The bounce phenomenon implies an alteration of hearing acuity after presentation of low-frequency loud sounds. Via the objective approach the bounce has been investigated predominantly in animals, while the most of the former human studies has utilized psychoacoustic methods. To compensate the deficiency, in present experiments the influence of the low-frequency tone applications on transiently evoked otoacoustic emissions (TEOAE) has been estimated in humans and the bounce phenomenon has been correspondingly been evaluated by the objective approach.

Material and Methods
Investigation were carried out on normally hearing adults. TEOAEs were recorded in response to wideband clicks. 250-Hz frequency tone served as an exposure sound. The exposure lasted 3 minutes. ILO 88 (Otoscape 942) device was utilized for TEOAE averaging. In each bounce session, three TEOAEs were recorded before tone exposure and the mean level of all three traces was judged as a control. Alterations of EOAE magnitudes, happened after the exposure, were referred just to this control level. EOAE registrations lasted 7-10 minutes following exposures. In 1rd experiment, individual and mean postexposure TEOAE alterations were calculated. In 2nd, the significance of exposure tone intensity and in 3rd of exposure tone frequency in bounce manifestations was estimated. In 4rd experiment, the exposure and test stimuli were consecutively applied to the same and opposite ears of tested subjects and bounce manifestations were searched under ipsilateral and contralateral presentation of exposure tone. In 1st, 3rd, and 4th experiments the intensity of exposure tone amounted to 95 dB SPL. In 2nd, it was changed from 55 to 100 dB SPL. The intensity of clicks in all experiments amounted to 70 dB SPL, respectively.

Results
After 250-Hz frequency, 95-dB SPL intensity tone exposure for 3 minutes, TEOAE alterations were documented in all ears tested in 1st experiment (Fig. 1). The changes appeared as augmentation in overall TEOAE levels followed by reduction. Maximal individual TEOAE augmentations approximated 3.0 dB and mini-mail 0.5 dB. The increments on the mean amounted to 1.3 dB. In separate ears the TEOAE decrements exceeded 1.5 dB, being on the mean 1.0 dB. The peak of the decrements was followed by the gradual recovery that lasted some minutes afterwards. TEOAE alterations were statistically significant at each of 7 minutes after the exposure. Later, TEOAE rest-rotations appeared complete. Actually no TEOAE alteration was observed at the lowest exposure tone intensities applied in 2rd experiment, 55 dB SPL (Fig. 2). At consecutive intensities 75 dB SPL, the process was manifested in an apparent TEOAE augmentation that was not followed by any noticeable reduction. At higher intensities, 85 SPL, the bipolar post-exposure alterations of TEOAEs were evident: obvious augmentations were replaced by similarly clear reductions. At the highest intensity, 100 dB SPL, the EOAE reduction was prominent while it has hardly been preceded by the augmentation. At all and opposite ears.

Discussion
Proceeding from the results of the above experiments, the bounce phenomenon is considered as a compound of two opposite events, augmentation and reduction, the share of each in individual post-exposure TEOAE pattern being dependent upon the exposure-tone intensity. The augmentation in the inner ear function seems to occur at wide exposure-tone intensities, starting from the lower ones. The depression, in contrast, occurs preferentially at higher intensities. Similar to the augmentation, the depression most probably follows the exposure-tone presentation. As a result, at lower exposure intensities it partially and at higher intensities totally blocks the augmentation while continues further without any contamination with that. Generally, thus, the depression vs. the augmentation possesses higher threshold, longer duration and steeper input/output function. Taking into account the differences, the augmentation and the depression could hardly be considered as the events, having a sole origin. The augmentation is believed to be determined by shift in operating points of outer hair cells that takes place due to the displacement of the basilar membrane from scala vestibule to scala tympani. Conversely, the depression can be attributed to the direct action of the high-intensity tones upon the receptor cells. Generally, it can have a link to the psychoacoustic process that is manifested in a temporary threshold shift, TTS. The mechanism of bounce phenomenon can further judged when matching the consequences of ipsilateral and contralateral tone exposures. The bounce manifestation in the respective investigations were observed under ipsilateral exposure tones only while no TEOAE alterations were observed under contralateral exposures. Considering these data, any involvement of brainstem-cochlear efferents and, generally of central neural processes in bounce manifestations could be kept out. As a matter of fact, olivocochlear efferents are known to be triggered when applying acoustic stimuli while even monoaural stimulation activates the efferents bilaterally and influences thus both ears. Conversely, selective ipsilateral but not contralateral manifestations of tone exposures imply that the bounce phenomenon involves exclusively peripheral but no central mechanisms.

This work is supported by the Volkswagen grant.