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Indications of central regulation of middle ear pressure from pressure evoked brain potentials

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Purpose: In clinical practice, the presence of negative MEP is very common and most often related to secretory otitis media. Although pathological changes in the tympanic membrane may to be associated with impaired baroreceptor function little is known about the middle ears active regulation of pressure. New information on these aspects would be of major significance in otological research, and have been emphasised as goals of research. On this background we have designed a new experimental method for conducting pressure stimuli to the ear canal and investigated the pressure related early brain potentials in humans. Furthermore, we employed spatial analysis to localise the brain areas involved in the processing.

Materials and Methods: The experiments were conducted by stimulating the tympanic membrane with a novel ear computer controlled pressure triggering system for rapid synchronized pressure loads (≈3 kPa). In six adult subjects the resulting brain evoked responses were recorded from 64 surface electrodes using a standard EEG cap. A full band EEG acquisition method was adopted, signals were sampled at 20,000 Hz, and band-pass filtered between 0.05 and 3000 Hz. Results: The study showed the first ever recorded early pressure evoked potentials. The characteristics of the pressure evoked brain potentials and their individual topographic localization were reproducible. Source localization was adopted on a realistic head model to show the location of these early neural generators. The dipole model showed a residual variance lower than 5%, and it could be reliably applied to the individual data. The earliest dipolar activities were observed in the medulla followed by the activity, which was generated by the cerebellum.

Conclusions: Earlier experiments in primates have demonstrated anatomical and physiological associations between the tympanic plexus of the middle ear cavity, respiratory centres in the brainstem (nucleus of the solitary tract) and the muscles of the ET. Thus, a neural feedback mechanism controlling the MEP has been suggested. In agreement, our current findings showed an early activation of the brainstem in response to pressure stimulation of the TM and the middle ear. The additional activation of the cerebellum was assumed to play a role in controlling the activity of the Eustachian tube. Further studies in this line are likely to provide basic knowledge on the possible role of an overall neural control of the MEP.