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Auditory spatial deficits in brainstem disorders

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ABSTRACT

Purpose: Brainstem disorders seem to negatively influence the central auditory system, causing spatial hearing deficits.

Material and methods: We tested 11 patients with brainstem lesions due to ischemic stroke (IS), multiple sclerosis (MS), or cerebellopontine angle tumor (CPAT) together with 50 age- and sex-matched healthy volunteers. We used pure tone audiometry (PTAud), brainstem auditory evoked potentials (BAEPs) and the horizontal minimum audible angle test (HMAAT) for 8 azimuths with binaural stimulation.

Results: The chosen patients and the controls had normal or near normal hearing in PTAud. BAEPs interaural wave I–V latency difference was over 7 times longer in the patients group compared to the controls. Additionally, 9 of the 11 patients (81.1%) had abnormal HMAAT results. The biggest quantitative disturbances in HMAAT were present in the CPAT and the MS patients. The sound localization ability in HMAAT was significantly worse in the patients in 0° azimuth in comparison with the controls, and in 45° and 90° azimuth in patients with auditory pathway involvement compared with the ones without the involvement.

Conclusions: Our study confirms the strong relationship between various brainstem pathologies and sound localization disability and sheds some light on the complexity of the relationship.

1. Introduction

Sound localization ability, or in other words directional hearing, was important for avoiding predators and thus is regarded as a phylogenetically older function than the reception of pure tones and understanding speech [1]. It is one of higher auditory functions and includes identification of distance, azimuth and directions of a moving sound in space

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Abbreviations: SPL, sound pressure level; HL, hearing level; HMAAT, the horizontal minimum audible angle test; BAEPs, brainstem auditory evoked potentials; PTA, pure tone average; PTAud, pure tone audiometry; kHz, kiloHertz; dB, decibel; ITD, interaural time delay; IID, interaural intensity delay; HRTFs, head-related transfer functions; IS, ischemic stroke; CPAT, cerebellopontine angle tumor; MS, multiple sclerosis; FDA, Food and Drug Administration; DCN, dorsal cochlear nucleus; VAS, ventral acoustic stria; TB, trapezoid body; SOC, superior olivary complex; LL, lateral lemniscus; IC, inferior colliculus.

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[2–4]. Giovanni Battista Venturi (1746–1822) is regarded as a forerunner of research on sound localization. He suspected that sound localization depends on asymmetry of the ears [5]. Brainstem anatomical structures responsible for proper sound localization seem to be active from birth [6]. The nuclei of the trapezoid corpus, nuclei of the lateral lemniscus, superior nucleus of the oliva and the nucleus of the inferior colliculus of the tectal lamina, constitute the basis of sound localization in the mechanism of interaural time delay (ITD), interaural intensity delay (IID) and head-related transfer functions (HRTFs) [7–9]. Pathological processes such as ischemic stroke (IS), demyelination in multiple sclerosis (MS) and cerebellopontine angle tumors (CPAT) can impair sound localization substantially because they restrict the broad stream of information going to the upper levels of the central nervous system (CNS) [2,10–15]. However, brainstem pathology is not the only reason for disturbed sound localization. The cortical sound localization centers are localized in the temporal, the parietal and the frontal lobes and pathological processes of theses regions can also substantially disturb the sound localization ability [2,4,16]. Since Venturi's time scientists have been trying to explain complicated neuronal mechanisms of sound localization. Research on sound localization in patients with brainstem pathologies showed a correlation between lesion localization and disturbed binaural auditory tasks. The correlation was particularly strong if the pathological process took place in the inferior colliculus and the lateral lemniscus [13,17,18]. However, results of the studies are inconsistent as far as the influence of side and volume of a lesion on sound localization ability is concerned. Additionally, the high redundancy of brainstem structures of the auditory pathways and the diversity of neuronal networks are the reason for small pathological lesions of these regions to be often clinically silent. The primary aim of this study was to find spatial hearing deficits characteristic for different brainstem pathologies. Secondly, we aimed to investigate a putative correlation between the side and the level of auditory pathway involvement in the MRI and the type of sound localization disturbance.

2. Methods

2.1. Patients

34 patients with brainstem pathology were included in the study. They were patients of the Neurology Outpatient Clinic, the Department of Neurology, the Audiology Outpatient Clinic or the Department of Otolaryngology, in years 2006–2011. Hearing tests were performed in the Audiology Outpatient Department. All patients underwent the pure tone audiometry (PTAud), the brainstem auditory evoked potentials (BAEPs) and the horizontal minimum audible angle (HMAAT) testing. The most important exclusion criteria for the study was the interaural difference of hearing threshold for medium frequencies-pure tone average (PTA-0.5–1–2 kHz) $>$ 20 dB HL and contraindications for the head MRI. Additional exclusion criteria were: age older than 80 years, patients with previous history of stroke (but not transient ischemic attack), serious general state, dementia, neurodegenerative disorders, other previously identified neurological diseases, patients without logical verbal contact due to aphasia, psychotic symptoms, visual spatial neglect syndrome tested with the line bisection test and the nonverbal shape cancelation task [19,20], conductive or mixed type hearing loss, history of ear surgery. Due to the abovementioned criteria we included 11 patients in the analysis. There were 4 ischemic stroke (IS) patients (2 women and 2 men). All subjects were right-handed. All neurootologic evaluations were performed during the early stage after the incidence of stroke (up to 30 days, average 10 $\pm$ 7 days). The diagnosis of stroke was based on the WHO criteria in patients with neurologic symptoms lasting for at least 24 h. We excluded patients with ischemic lesions localized in the hemispheres. There were 5 patients with multiple sclerosis (3 women and 2 men) in the group. The diagnosis of MS was based on the McDonald et al. [21] criteria. We excluded patients with demyelinating lesions affecting the hemispheric part of the auditory pathway. All neurootologic evaluations were performed 2 months to 12 years from first MS symptoms. Only two patients from the study group suffered from cerebellopontine angle tumor (CPAT) (1 woman and 1 man). The diagnosis was based on the criteria of Kanzaki et al. [22]. The study was approved by the regional independent ethics committee (NKEB/32/2006). All the patients and the control subjects provided written, informed consent for involvement in the study.

2.2. Controls

The control group consisted of 50 age-matched subjects, 19 men and 31 women (on average 4.6 controls per one patient). The average age of the group was 50.1 (SD $\pm$ 17.4) years (range 21–80 years) and it consisted of healthy volunteers. The exclusion criteria for the control group were: previously identified neurological diseases, diabetes, circulatory insufficiency, alcoholism, smoking, use of medications affecting the CNS, history of noise exposure at work, hearing disorders including the conductive and the mixed type hearing loss, and history of ear surgery. All subjects underwent otological and neurological examination. We also excluded patients with abnormal BAEPs results. According to the Food and Drug Administration (FDA) classification [23] 3 subjects had sensorineural mild hearing loss. All of them were over 60 years of age and their hearing loss was due to cochlear presbyacusis.

2.3. Study design

A detailed protocol was developed prior to conducting this study. Randomly selected neurological patients and matched controls were examined for peripheral and central hearing deficits by an independent audiologist in a standard case–control study fashion.

2.4. Localization of brainstem lesions in the MRI

All patients underwent magnetic resonance imaging (MRI) of the brain according to standard imaging protocols in 1.5T scanners. Determination of areas of focal brain damage was performed manually based on supplied imaging data sets. Localization of the lesions and involvement of the structures