## Letter to the Editor

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## Brainstem Auditory-Evoked Potential and Migraine: Is There an Association?

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Dear Editor,

Migraine, an episodic headache disorder characterized by acute disabling attacks, presents with recurrent headache attacks and various combinations of symptoms related to the gastrointestinal and autonomic nervous systems [1]. In addition, up to one third of migraineurs experience neurological symptoms most often involving the visual system before or during the migraine attack, which are known as migraine aura [1]. Migraine affects about 18% of the female and 6% of the male population during the most productive periods of their working lives [2]. In the United States, migraine causes the majority of the 5 million headache visits to the Emergency Departments (ED) annually, and it has been estimated that the health cost for migraine-related ED visits is at least USD 700 million [3]. This creates a major public health burden.

Over the past several decades, based on a strong body of evidence [4], it has been postulated that migraine, particularly with aura, is associated with cardiovascular outcomes (such as stroke, myocardial infarction, coronary heart disease and cardiovascular disease mortality) [4]. Hence, the screening and prevention of migraine should be an important public health priority.

To our knowledge, the pathophysiology of migraine has not been completely elucidated, though several brain circuits have been implicated [5]. Since the early 1990s, the nature of the electroencephalogram changes indicates a brainstem and midbrain dysfunction in migraine [6]. Brainstem nuclei seem to be activated during migraine attacks, and brainstem auditory-evoked potential (BAEP) was a sensitive measure of central nervous system dysfunction [7].

Recently, an experimental study aimed at determining whether BAEP in the central sensitization model had similar changes to those in migraine, and whether it added objective and functional information to understanding the early sensory changes in the migraine model. Arakaki et al. [8] reported that BAEP alternations could reflect changes in neurotransmitters and/or hypoperfusion in the midbrain. The similarity of those findings with previous human studies had been suggested.

Dash et al. [9] prospectively evaluated the audiovestibular functions in 50 cases of migraine with or without vertigo. They reported that the auditory brainstem-evoked responses of all these pa-

tients showed some abnormalities in the form of prolonged absolute latency or prolonged interwave peak latencies or both, thereby demonstrating that BAEP abnormalities might be the earliest indicator of impending auditory involvement in migraine. Interestingly, in a longitudinal study, Sand et al. [10] reported that the intensity dependence of BAEP in migraine was probably not a passive reflection of brainstem dysfunction. Waves I, V and interpeak III–V latency increased after the attack, but no evidence for preattack brainstem auditory sensitization was observed in migraine. The BAEP changes seemed to reflect a slight impact of migraine on serotonergic brainstem pathways.

In view of these findings, is migraine associated with BAEP changes? Which efficacy measures are clinically relevant and could be used to study acute migraine? The following recommendations should be considered. (1) Migraine patients have a substantially increased risk of autonomic symptoms which may be suggestive of brainstem or midbrain dysfunction. Meanwhile, BAEP should be used to determine whether or not the auditory system has potential abnormalities, especially after an attack. (2) Patients suffering from migraine should undergo BAEP investigation using 10/S clicks and an increased stimulus rate of 55/S. (3) Where intensity dependence of BAEP is not sensitive enough, future studies could explore whether or not the colliculothalamic and thalamocortial auditory transmission should be investigated accordingly.

## References

- 1 Silberstein SD: Migraine. Lancet 2004;363:381-391.
- 2 Lipton RB, Bigal ME: The epidemiology of migraine. Am J Med 2005; 118:3S–10S.
- 3 Insinga RP, Ng-Mak DS, Hanson ME: Costs associated with outpatient, emergency room and inpatient care for migraine in the USA. Cephalalgia 2011;31:1570–1575.
- 4 Schurks M, Rist PM, Bigal ME, et al: Migraine and cardiovascular disease: systematic review and meta-analysis. BMJ 2009;339:b3914.
- 5 Noseda R, Burstein R: Migraine pathophysiology: anatomy of the trigeminovascular pathway and associated neurological symptoms, CSD, sensitization and modulation of pain. Pain DOI: 10.1016/j.pain. 2013.07.021.
- 6 Sand T: EEG in migraine: a review of the literature. Funct Neurol 1991; 6:7–22
- 7 Bahra A, Matharu MS, Buchel C, et al: Brainstem activation specific to migraine headache. Lancet 2001;357:1016–1017.
- 8 Arakaki X, Galbraith G, Pikov V, et al: Altered brainstem auditory evoked potentials in a rat central sensitization model are similar to those in migraine. Brain Res 2014;1563:110–121.
- 9 Dash AK, Panda N, Khandelwal G, et al: Migraine and audiovestibular dysfunction: is there a correlation? Am J Otolaryngol 2008;29:295–299.
- 10 Sand T, Zhitniy N, White LR, et al: Brainstem auditory-evoked potential habituation and intensity-dependence related to serotonin metabolism in migraine: a longitudinal study. Clin Neurophysiol 2008;119:1190– 1200.



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