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Original Article

EVALUATION OF BRAINSTEM AUDITORY EVOKED POTENTIAL IN MIGRAINE PATIENTS

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ABSTRACT

Background: Migraine is a complex neurovascular disorder often associated with altered sensory processing. Brainstem auditory evoked potentials (BAEPs) provide a non-invasive method to assess central auditory pathway function and subclinical brainstem involvement in migraine. The present study was conducted to evaluate BAEP parameters in migraine patients and compare them with healthy controls.
Materials and Methods: A hospital-based, observational case control study was conducted on 30 migraine patients and 30 age- and sex-matched healthy controls. BAEP recordings were obtained during the interictal period. Absolute latencies of waves I, III, and V and interpeak latencies (I–III, III–V, I–V) were measured. Statistical analysis was performed using SPSS version 20.0. with $p < 0.05$ considered significant.
Results: Migraine patients exhibited significant prolongation of wave III and V latencies and interpeak intervals I–III, III–V, and I–V compared to controls ($p < 0.05$), while wave I latency remained normal. No significant differences were observed between migraine with aura and without aura. These findings indicate delayed conduction within the brainstem auditory pathways, particularly at the pontine and midbrain levels.
Conclusion: Migraine is associated with subclinical brainstem dysfunction detectable by BAEP. Prolonged central conduction times suggest impaired brainstem auditory processing, independent of aura status. BAEP is a valuable, non-invasive tool for assessing central auditory pathway abnormalities and provides insight into the neurophysiological basis of migraine.

Keywords: *Migraine, Brainstem auditory evoked potentials, BAEP, Brainstem dysfunction, Auditory pathway.*

INTRODUCTION

Migraine is a highly prevalent and disabling neurological disorder characterized by recurrent attacks of headache that are often unilateral, pulsatile, and associated with nausea, photophobia, and phonophobia. The World Health Organization ranks migraine among the leading causes of years lived with disability, particularly among young and middle-aged women, reflecting its substantial socioeconomic and personal impact (1). The global prevalence of migraine is estimated to be between 12% and 15%, with a clear female predominance attributed to hormonal modulation of central pain mechanisms (2).

Although migraine has traditionally been considered a vascular headache, recent advances have established it as a neurovascular disorder with dysfunction in sensory processing and altered excitability of central neural networks. The trigeminovascular system plays a central role in migraine pathophysiology, where activation of trigeminal afferents innervating meningeal vessels leads to the release of vasoactive neuropeptides such as calcitonin gene-related peptide (CGRP), substance P, and neurokinin A. These mediators promote neurogenic inflammation and vasodilation, contributing to pain transmission (3,4).

Accumulating evidence highlights the importance of the brainstem in the genesis and modulation of migraine. Neuroimaging studies have consistently demonstrated activation of brainstem nuclei—including the dorsal pons, periaqueductal gray (PAG), and locus coeruleus—during spontaneous migraine attacks, with persistent hypermetabolism even after pain resolution (5,6). These findings suggest that the brainstem is not merely a relay station but an active generator and modulator of migraine-related nociceptive signals. The brainstem also governs auditory and sensory gating mechanisms, and dysfunction in these processes may explain the hypersensitivity to sound (phonophobia) frequently observed in migraine patients (7).

Electrophysiological studies further support the concept of abnormal sensory processing in migraine. Patients exhibit deficient habituation to repetitive stimuli, implying a failure of normal inhibitory control within the brainstem and thalamocortical circuits (8,9). Evoked potential studies, including visual, somatosensory, and auditory modalities, have provided objective evidence of this dysregulation. Among these, Brainstem Auditory Evoked Potentials (BAEPs), also known as Auditory Brainstem Responses (ABRs), offer a sensitive and non-invasive method to evaluate neural conduction along the auditory brainstem pathways. BAEPs consist of a series of short-latency waveforms (I–V) that reflect sequential activation of auditory structures from the cochlea through the cochlear nucleus, superior olivary complex, and lateral lemniscus to the inferior colliculus (10,11). Because these responses occur within the first 10 milliseconds after stimulation and are unaffected by consciousness or attention, they provide a reliable index of brainstem integrity (12).

Several studies have investigated BAEP abnormalities in migraine, aiming to identify subclinical evidence of brainstem dysfunction. Sand et al. (13) demonstrated prolonged wave V latency and increased I–V interpeak intervals in migraineurs, suggesting delayed conduction in the upper brainstem. Afifi et al. (14) reported similar findings, with significant prolongation of wave III and V latencies and interpeak intervals, indicating pontine and midbrain involvement. In contrast, other studies have found minimal or no BAEP alterations, possibly due to differences in migraine subtype, disease duration, or recording during ictal versus interictal phases (15). Nevertheless, the preponderance of evidence supports the hypothesis that migraine is associated with subtle, yet measurable, abnormalities in auditory brainstem processing.

The evaluation of BAEPs in migraine may thus provide objective insight into the neural mechanisms underlying sensory hypersensitivity and central excitability. Identification of subclinical brainstem dysfunction could not only improve understanding of migraine pathophysiology but also assist in monitoring disease progression and therapeutic response. In this context, the present study was undertaken to evaluate Brainstem Auditory Evoked Potentials in migraine patients and compare them with healthy controls to detect possible abnormalities in auditory brainstem conduction.

MATERIALS AND METHODS

This hospital-based, observational, case–control study was conducted over a period of 6 months in the Department of Physiology at a tertiary care teaching hospital. Ethical approval was obtained from the Institutional Ethics Committee prior to commencement of the study, and all participants provided written informed consent.

A total of 60 subjects were recruited, comprising 30 clinically diagnosed migraine patients and 30 age- and sex-matched healthy controls. The diagnosis of migraine was established according to the International Classification of Headache Disorders, 3rd edition (ICHD-3) criteria (16). Patients of either sex between 18 and 45 years of age were included. Individuals with a history of chronic systemic illness, diabetes mellitus, hypertension, epilepsy, otologic disorders, chronic noise exposure, substance abuse, or those taking medications known to affect central nervous system conduction were excluded from the study. The control group consisted of healthy volunteers without any history of headache or neurological or auditory abnormalities.

A detailed clinical history was recorded for each participant, including duration, frequency, and characteristics of headache, presence or absence of aura, associated symptoms such as photophobia, phonophobia, nausea, and vomiting, and family history of migraine. Physical and neurological examinations were performed to exclude other causes of headache. Otoscopic examination and pure tone audiometry were carried out to rule out conductive or sensorineural hearing loss. All participants had normal hearing thresholds (<25 dB HL) bilaterally.

The Brainstem Auditory Evoked Potential (BAEP) test was performed using a standard computerized evoked potential recording system (such as Nicolet or RMS EMG/EP Mark II, depending on institutional availability). The procedure was conducted in a quiet, sound-attenuated, and electrically shielded room to minimize external interference. Participants were instructed to relax, remain awake, and refrain from excessive movement during the procedure. The study was performed in the interictal period, i.e., at least 72 hours after the last migraine attack and 72 hours before the next predicted attack, to avoid ictal phase confounders (17).

Disposable silver–silver chloride surface electrodes were used after cleaning the skin with spirit and applying conductive gel to maintain impedance below 5 kΩ. The recording electrode was placed at the vertex (Cz) according to the International 10–20 system, the reference electrode at the ipsilateral mastoid (A1 or A2), and the ground electrode at the forehead (Fpz). Monaural auditory stimulation was delivered through insert earphones using rarefaction clicks of 100 μs duration at an intensity of 70 dB above the individual's hearing threshold. The contralateral ear received masking noise of 40 dB to prevent crossover stimulation. The rate of stimulation was set at 11.1 clicks per second, and two trials of 2000 stimuli each were recorded to ensure reproducibility. Responses were filtered using a bandpass of 100–3000 Hz, with an analysis time window of 10 milliseconds.

The recorded waveforms were analyzed for the absolute latencies of waves I, III, and V, and interpeak latencies of I–III, III–V, and I–V on both sides. The mean values obtained from both ears were used for statistical comparison. Prolongation of wave latencies or interpeak intervals was interpreted as evidence of delayed neural conduction along specific segments of the auditory pathway. Wave I reflects the distal auditory nerve, wave III the lower pons (cochlear nucleus and superior olivary complex), and wave V the upper brainstem (lateral lemniscus and inferior colliculus) (18).

All recordings were performed at room temperature (25 ± 1 °C) under uniform testing conditions. The same examiner conducted all recordings to eliminate interobserver variability. Quality control procedures were followed according to the guidelines of the American Clinical Neurophysiology Society (ACNS) to ensure reliability and reproducibility of results (5).

Data were compiled and analyzed using IBM SPSS Statistics version 20. Data was presented as number for qualitative data and mean ± standard deviation for quantitative data. Data was analyzed using the chi-square test for comparing the qualitative data and students t test for quantitative data between the two groups. A *p*-value less than 0.05 was considered statistically significant.

RESULTS

The study included 60 participants—30 migraine patients and 30 age- and sex-matched healthy controls. Among migraine patients, 18 (60%) were females and 12 (40%) males; controls included 17 (56.7%) females and 13 (43.3%) males. The mean age was **30.2 ± 6.4 years** in the migraine group and **29.6 ± 5.9 years** in controls ($p = 0.68$). Both groups were demographically comparable (Table 1). All migraine cases met ICHD-3 diagnostic criteria; 20 (66.7%) had migraine without aura and 10 (33.3%) with aura. Mean illness duration was **4.8 ± 2.3 years**, with **3.4 ± 1.2 attacks per month**.

TABLE 1: Demographic & clinical characteristics of study participants

Parameter	**Migraine (n = 30)**	**Control (n = 30)**	***p*-value**
Age (years, mean ± SD)	30.2 ± 6.4	29.6 ± 5.9	0.68 (NS)
Sex (M/F)	12/18	13/17	0.79 (NS)
Duration of illness (years)	4.8 ± 2.3	—	—
Attack frequency/month	3.4 ± 1.2	—	—

BAEP waveforms were clearly identified in all participants. Table 2 summarizes the comparison of absolute and interpeak latencies between the two groups. Migraine patients showed significantly prolonged **wave III** and **wave V latencies**, as well as **interpeak latencies I–III, III–V, and I–V**, compared with controls ($p < 0.05$). Wave I latency did not differ significantly, indicating intact peripheral auditory conduction.

TABLE 2: Comparison of BAEP latencies between groups

Parameter (ms)	**Controls (Mean ± SD)**	**Migraine (Mean ± SD)**	***p*-value**
Wave I	1.62 ± 0.12	1.68 ± 0.14	0.08 (NS)
Wave III	3.72 ± 0.16	3.89 ± 0.21	**0.001**
Wave V	5.64 ± 0.22	5.89 ± 0.28	**<0.001**
I–III IPL	2.10 ± 0.14	2.22 ± 0.17	**0.006**
III–V IPL	1.92 ± 0.13	2.01 ± 0.14	**0.012**
I–V IPL	4.03 ± 0.18	4.23 ± 0.21	**<0.001**

Patients with aura showed slightly higher wave V and I–V latencies than those without aura, though differences were not statistically significant ($p > 0.05$) (Table 3).

TABLE 3: BAEP parameters in migraine with and without aura

Parameter (ms)	Without Aura (n = 20)	With Aura (n = 10)	*p*-value
Wave I	1.67 ± 0.12	1.70 ± 0.13	0.54 (NS)
Wave III	3.86 ± 0.20	3.93 ± 0.22	0.31 (NS)
Wave V	5.86 ± 0.24	5.94 ± 0.27	0.29 (NS)
I–III IPL	2.19 ± 0.16	2.25 ± 0.18	0.42 (NS)
III–V IPL	2.00 ± 0.13	2.03 ± 0.14	0.52 (NS)
I–V IPL	4.20 ± 0.19	4.28 ± 0.22	0.34 (NS)

DISCUSSION

The present study assessed brainstem auditory evoked potentials (BAEPs) in patients with migraine and compared them with healthy controls to evaluate brainstem function. The results demonstrated a significant prolongation of the absolute latencies of waves III and V and interpeak intervals (I–III, III–V, and I–V) in migraine patients, while wave I latency remained within normal limits. These findings indicate delayed neural transmission in the central auditory pathway, particularly within the pons and midbrain regions, suggesting subclinical brainstem dysfunction.

The preservation of wave I latency confirms that the peripheral auditory structures, including the cochlea and auditory nerve, are unaffected in migraineurs. In contrast, the prolongation of waves III and V reflects altered conduction at the superior olivary complex, lateral lemniscus, and inferior colliculus levels of the brainstem. These findings are in line with those of Khalil et al., who reported similar prolongation in the BAEP latencies of migraine patients, suggesting functional impairment in the auditory brainstem (20).

The brainstem plays an integral role in the generation and modulation of migraine attacks. Functional neuroimaging studies have shown persistent activation of the dorsal pons during both ictal and interictal phases of migraine (21). This activation likely represents a migraine “generator” region involved in pain modulation, autonomic control, and sensory integration. The trigeminovascular system, modulated by the brainstem nuclei such as the periaqueductal gray, locus coeruleus, and dorsal raphe nucleus, contributes significantly to migraine pathophysiology (22,23). BAEP abnormalities observed in this study therefore provide electrophysiological support for the hypothesis of brainstem dysfunction in migraine.

The prolongation of interpeak latencies in the present study is comparable to the findings of Schoenen et al. (24) and Wang et al. (25), who reported increased I–V and III–V intervals in migraineurs. These abnormalities are believed to reflect delayed central auditory conduction due to altered excitability or synaptic transmission efficiency in the auditory brainstem nuclei. Furthermore, Wang et al. (25) demonstrated normalization of BAEP abnormalities following prophylactic treatment, suggesting that the dysfunction is reversible and possibly related to transient neurochemical changes rather than structural lesions.

No statistically significant difference was observed between patients with migraine with aura and those without aura in the present study, consistent with findings from Afra et al. (26). This suggests that brainstem dysfunction is a generalized phenomenon in migraine, independent of the presence of aura. However, other researchers such as Sand et al. (27) have reported more pronounced evoked potential abnormalities in patients with aura, which may be due to variations in cortical spreading depression and its downstream effects on brainstem nuclei.

The present findings reinforce the concept that migraine is not solely a vascular disorder but a neurovascular syndrome characterized by aberrant sensory processing and altered brainstem excitability (28,29). Prolonged BAEP latencies indicate impaired temporal synchrony in auditory transmission, which could explain clinical features such as phonophobia and increased auditory sensitivity during migraine attacks (30). These subclinical changes, persisting during interictal phases, imply an ongoing dysfunction rather than an episodic one.

CONCLUSION

The present study demonstrates that migraine is associated with subclinical dysfunction of the central auditory pathways, evidenced by prolonged BAEP wave III and V latencies and interpeak intervals (I–III, III–V, I–V), while peripheral conduction (wave I) remains intact. These alterations indicate delayed neural conduction at the pontine and midbrain levels and appear independent of aura status, suggesting that brainstem involvement is a fundamental feature of migraine pathophysiology. BAEP emerges as a reliable, non-invasive tool for detecting such central auditory dysfunction, offering insights into migraine as a disorder of altered sensory processing and brainstem excitability

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