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Original Research Article

A Comparative Study of Brainstem Auditory Evoked Potential Responses among Hypertensive Patients and Normotensive Controls

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Conflict of interest: Nil

Abstract:

Background: Hypertension produces progressive vascular changes that may lead to subclinical involvement of the central nervous system. The brainstem auditory pathway is particularly vulnerable due to its high metabolic demand. Brainstem Auditory Evoked Potentials (BAEPs) provide an objective and sensitive method for detecting early auditory brainstem dysfunction.

Objective: To evaluate and compare BAEP parameters in hypertensive patients and age- and sex-matched normotensive controls.

Materials and Methods: This cross-sectional comparative study included 70 participants aged 40–60 years, comprising 35 essential hypertensive patients and 35 normotensive controls. BAEP recordings were obtained using monaural click stimuli at 40, 50, and 60 dB sensation levels. Absolute latencies of waves I, III, and V, interpeak latencies (I–III, III–V, I–V), and wave amplitudes were analyzed. Statistical comparison was performed using Student's unpaired t-test, with $p < 0.05$ considered significant.

Results: Hypertensive patients showed significant prolongation of wave III and V latencies and increased interpeak latencies III–V and I–V at all stimulus intensities ($p < 0.05$), while wave I latency remained unaffected. Wave III and V amplitudes were significantly altered at higher intensities, indicating impaired central auditory conduction with preserved peripheral auditory nerve function.

Conclusion: Essential hypertension is associated with early subclinical dysfunction of central auditory brainstem pathways. BAEP is a useful, non-invasive tool for early detection and monitoring of central nervous system involvement in hypertensive patients.

Keywords: Hypertension, BAEP, Auditory Brainstem, Evoked Potentials.

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Introduction

Hypertension is one of the most prevalent chronic non-communicable diseases worldwide and a major contributor to cardiovascular, cerebrovascular, and neurological morbidity [1]. Chronic elevation of arterial blood pressure produces widespread vascular changes, including endothelial dysfunction, arteriolar narrowing, and microangiopathy, which compromise blood supply to vital organs such as the brain [2]. The brainstem, owing to its high metabolic demand and end-arterial blood supply, is particularly vulnerable to hypertensive vascular changes [3].

Hypertension affects about 31 % of adults worldwide, corresponding to approximately 1.4 billion people (using the standard definition of systolic BP ≥140 mm Hg or diastolic ≥90 mm Hg). Prevalence is slightly higher in low- and middle-income countries (LMICs) compared with high-income countries. The absolute number of people living with elevated blood pressure and related disease burden has increased substantially over recent decades, driven by population growth and ageing [4][5].

Auditory dysfunction has been increasingly recognized as an early manifestation of central nervous system involvement in hypertension. Brainstem Auditory Evoked Potentials (BAEPs) are short-latency evoked responses generated within the auditory pathway from the auditory nerve to the midbrain. They provide an objective, non-invasive, and reproducible method for assessing the functional integrity of the auditory

brainstem pathway and are sensitive to subtle conduction delays that may not be evident clinically [6][7][8]. Several studies have reported prolonged BAEP latencies and inter-peak latencies in hypertensive individuals, suggesting subclinical auditory brainstem involvement [9]. However, data on amplitude changes and detailed comparison across stimulus intensities remain limited. Therefore, the present study was undertaken to evaluate and compare BAEP responses among hypertensive patients and normotensive controls to detect early central auditory pathway dysfunction.

Objective:

The objective of the present study was to assess and compare Auditory Brainstem Evoked Response (ABER) parameters between hypertensive patients and age - sex matched normotensive controls in terms of amplitudes, latencies and interpeak latencies at different intensities for both ears.

Materials and Methods

The present study was an observational, cross-sectional, comparative study conducted in the Department of Physiology in collaboration with the Department of Medicine at S.M.S. Medical College and Attached Hospitals, Jaipur, Rajasthan, from March 2024 to April 2025 after obtaining desired approval from the Institutional Ethics Committee.

A total of 70 study participants aged 40–60 years of either sex were recruited and divided into two groups: 35 diagnosed essential hypertensive patients (case group) and 35 age- and sex-matched normotensive healthy individuals (control group).

Hypertensive patients were selected from the Medicine outpatient department and included those with stage 1 and stage 2 hypertension. Normotensive controls were recruited from healthy volunteers. Written informed consent was obtained from all participants prior to inclusion in the study.

Inclusion Criteria: Diagnosed patients with hypertensive disorder, aged 40-60 years of either sex as cases, age matched healthy volunteers of either sex as controls and participants giving written informed consent were included in the present study.

Exclusion Criteria: Subjects who have any acute or chronic illness other than hypertension, history of ear diseases, head injury, psychiatric illness, structural brain lesion or neurological disorders, individuals on medications which are known to affect auditory function, occupational exposure to loud noise. and subjects who have history of smoking and alcoholism were excluded from the present study.

All participants underwent clinical evaluation, including blood pressure measurement using standard sphygmomanometry. BAEP recordings were performed in a quiet, electrically shielded room using RMS EMG SALUS-2C equipment. Surface electrodes were placed at the vertex (Cz), mastoid processes (A1 and A2), and forehead (Fz) as ground, with electrode impedance maintained below 5 kΩ.

Monaural click stimuli of 0.1 ms duration were delivered through supra-aural headphones at intensities of 40, 50, and 60 dB sensation level. The stimulus rate was 10 clicks per second, with rarefaction polarity. Responses were averaged over 1000 stimuli, with band-pass filters set between 200 and 2000 Hz. Absolute latencies of waves I, III, and V, inter-peak latencies (I–III, III–V, I–V), and amplitudes waves I, III, and V, were recorded for both ears. Data were analyzed using SPSS software version 25. Results were expressed as mean ± standard deviation. Statistical comparison between groups was done using Student's unpaired t-test, and a p value < 0.05 was considered statistically significant.

Results

ABER parameters at 40 dB SL stimulus intensity are shown in Table 1 and graphically represented in Figure 1.

At 40 dB SL, wave amplitudes (I, III, V) in both left and right ears showed no statistically significant difference between cases and controls ($p > 0.05$).

However, latency analysis revealed significant prolongation in cases.

In the left ear, wave III and wave V latencies were significantly increased in cases compared to controls ($p < 0.05$), while wave I latency showed no significant difference. Similarly, interpeak latencies I–V and III–V were significantly prolonged in cases, whereas I–III interpeak latency was not statistically significant. In the right ear, wave V latency was significantly prolonged in cases ($p < 0.05$), while wave I and III latencies were comparable between groups. Interpeak latencies I–V and III–V were significantly increased, whereas I–III interpeak latency showed no significant difference. ABER parameters at 50 dB SL stimulus intensity are shown in Table 2 and graphically represented in Figure 2.

Table 1: ABER Parameters at 40 dB SL stimulus intensity

BERA Parameter	BERA wave	Cases (Mean ± SD)	Control (Mean ± SD)	P- value
Left ear				
Amplitudes(µV)	I	0.12 ± 0.06	0.12 ± 0.05	0.68
	III	0.22 ± 0.03	0.21 ± 0.03	0.43
	V	0.37 ± 0.17	0.34 ± 0.09	0.30
Latencies (ms)	I	1.64 ± 0.18	1.59 ± 0.22	0.26
	III	3.67 ± 0.25	3.52 ± 0.34	**0.04***
	V	5.56 ± 0.27	5.37 ± 0.33	**0.01***
Interpeak latencies (ms)	I – III	1.94 ± 0.25	1.92 ± 0.32	0.1
	I – V	3.91 ± 0.24	3.78 ± 0.28	**0.04***
	III - V	1.97 ± 0.23	1.84 ± 0.35	**0.02***
Right ear				
Amplitudes(µV)	I	0.13 ± 0.05	0.13 ± 0.06	0.63
	III	0.19 ± 0.03	0.18 ± 0.04	0.42
	V	0.34 ± 0.12	0.33 ± 0.06	0.5
Latencies (ms)	I	1.64 ± 0.28	1.64 ± 0.3	0.94
	III	3.53 ± 0.22	3.39 ± 0.44	0.1
	V	5.53 ± 0.44	5.29 ± 0.48	**0.03***
Interpeak latencies (ms)	I – III	1.76 ± 0.17	1.76 ± 0.32	0.8
	I – V	3.88 ± 0.3	3.71 ± 0.29	**0.02***
	III - V	2.12 ± 0.32	1.95 ± 0.41	**0.04***

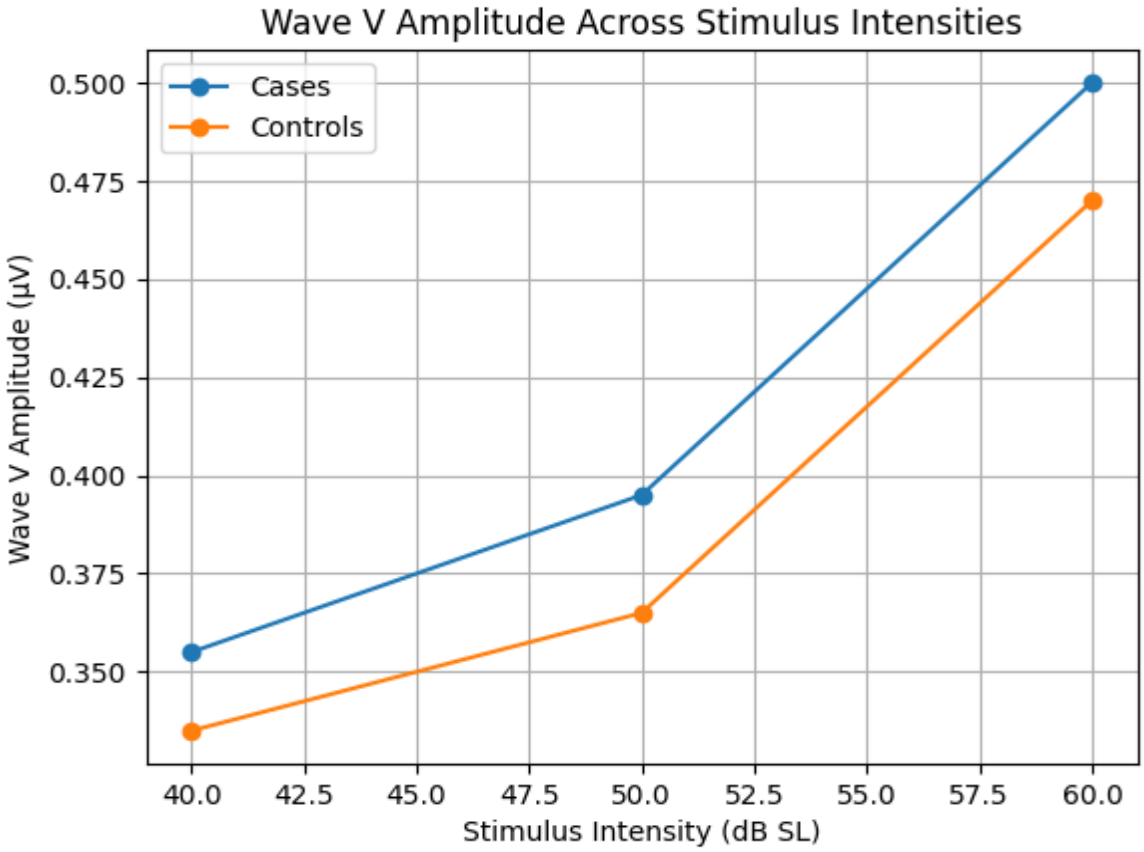

Figure 1: Comparison of Amplitude wave across increasing stimulus intensities

Table 2: ABER Parameters at 50 dB SL stimulus intensity

BERA Parameter	BERA wave	Cases (Mean ± SD)	Control (Mean ± SD)	P- value
Left ear				
Amplitudes(μV)	I	0.23 ± 0.03	0.21 ± 0.04	0.13
	III	0.28 ± 0.02	0.26 ± 0.05	**0.01***
	V	0.38 ± 0.06	0.35 ± 0.04	**0.03***
Latencies (ms)	I	1.62 ± 0.26	1.61 ± 0.3	0.93
	III	3.62 ± 0.19	3.5 ± 0.27	**0.03***
	V	5.7 ± 0.23	5.53 ± 0.24	**0.004***
Interpeak latencies (ms)	I – III	1.98 ± 0.24	1.95 ± 0.29	0.66
	I – V	4.07 ± 0.25	3.95 ± 0.28	**0.04***
	III - V	2.11 ± 0.23	1.99 ± 0.23	**0.03***
Right ear				
Amplitudes(μV)	I	0.24 ± 0.03	0.23 ± 0.05	0.13
	III	0.32 ± 0.02	0.29 ± 0.04	**0.01***
	V	0.41 ± 0.06	0.38 ± 0.04	**0.03***
Latencies (ms)	I	1.65 ± 0.29	1.65 ± 0.29	0.9
	III	3.65 ± 0.19	3.55 ± 0.35	**0.04***
	V	5.72 ± 0.28	5.48 ± 0.34	**0.003***
Interpeak latencies (ms)	I – III	1.92 ± 0.19	1.92 ± 0.22	0.92
	I – V	3.95 ± 0.16	3.84 ± 0.26	**0.03***
	III - V	2.03 ± 0.15	1.91 ± 0.26	**0.02***

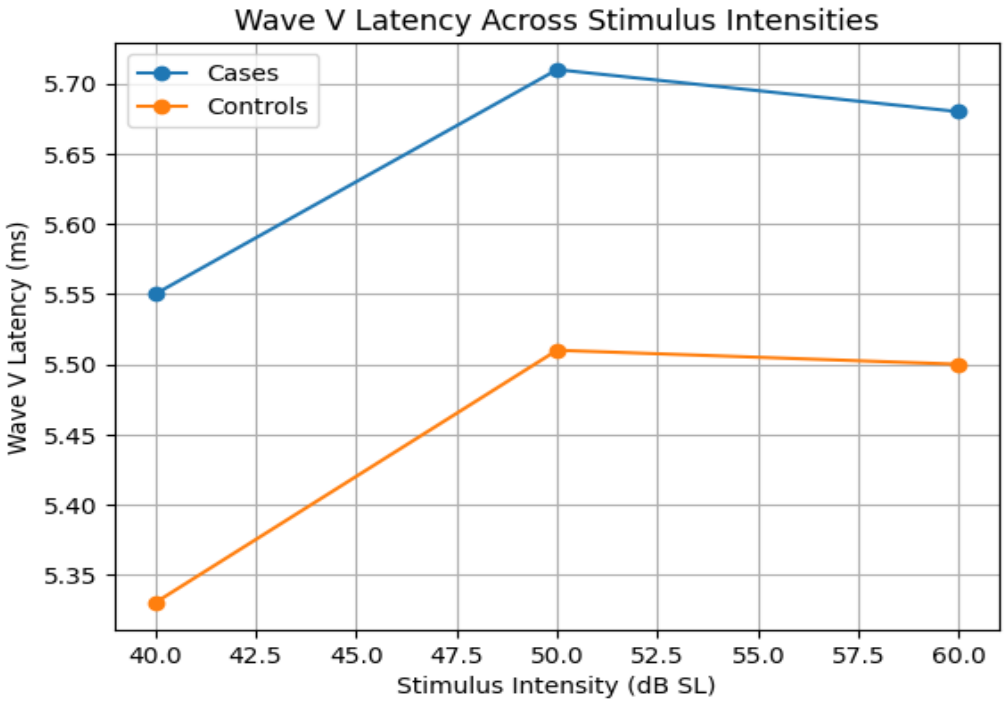

Figure 2: Comparison of Latency wave across increasing stimulus intensities

At 50 dB SL, wave amplitudes of III and V were significantly higher in cases compared to controls in both ears ($p < 0.05$), while wave I amplitude showed no significant difference.

Regarding latencies, wave III and wave V latencies were significantly prolonged in cases in both left and right ears ($p < 0.05$). Wave I latency did not differ significantly between cases and controls.

Analysis of interpeak latencies demonstrated significant prolongation of I–V and III–V intervals in cases in both ears ($p < 0.05$), while I–III interpeak latency remained statistically comparable between the two groups.

ABER parameters at 60 dB SL stimulus intensity are shown in Table 3 and graphically presented in Figure 3.

Table 3: ABER Parameters at 60 dB SL stimulus intensity

BERA Parameter	BERA wave	Cases (Mean ± SD)	Control (Mean ± SD)	P- value
Left ear				
Amplitudes(μV)	I	0.27± 0.03	0.26± 0.03	0.26
	III	0.32 ±0.03	0.3 ±0.03	**0.01***
	V	0.48 ± 0.05	0.45 ± 0.05	**0.005***
Latencies (ms)	I	1.61 ± 0.22	1.61 ± 0.28	0.91
	III	3.57 ± 0.18	3.44 ±0.34	**0.04***
	V	5.66 ±0.25	5.46 ±0.33	**0.01***
Interpeak latencies (ms)	I – III	1.93 ± 0.12	1.91 ± 0.21	0.68
	I – V	3.99 ± 0.23	3.88 ±0.17	**0.022***
	III - V	2.06 ± 0.15	1.97 ± 0.22	**0.031***
Right ear				
Amplitudes(μV)	I	0.27± 0.03	0.26 ± 0.02	0.11
	III	0.32 ±0.03	0.31 ± 0.02	**0.012***
	V	0.52 ±0.04	0.49 ±0.05	**0.013***
Latencies (ms)	I	1.69 ± 0.33	1.64 ± 0.28	0.48
	III	3.72 ± 0.29	3.66 ± 0.25	**0.043***
	V	5.69 ±0.25	5.53 ±0.29	**0.011***
Interpeak latencies (ms)	I – III	2.04 ±0.3	2.03 ±0.26	0.92
	I – V	4 ±0.29	3.86 ±0.29	**0.039***
	III - V	1.97 ± 0.31	1.82 ± 0.28	**0.045***

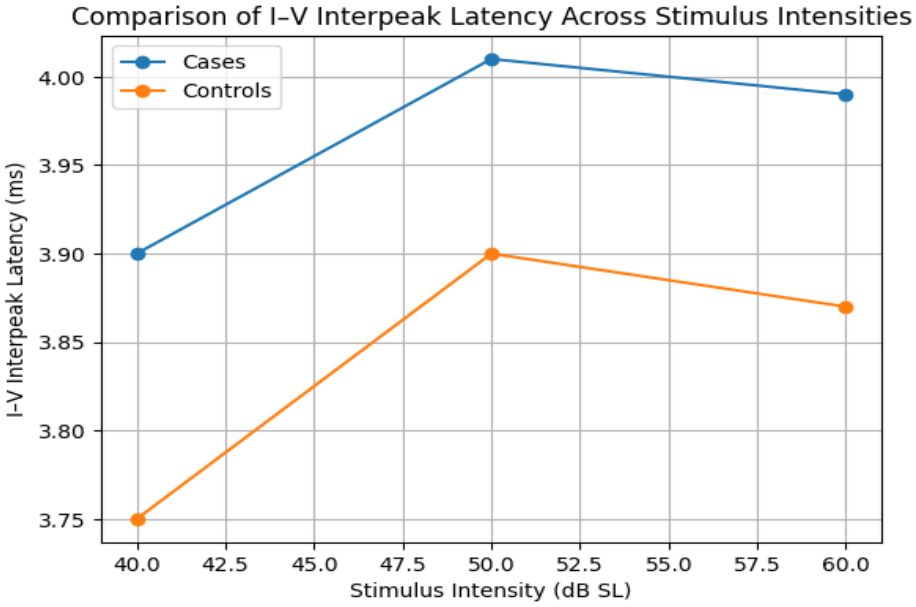

Figure3: Comparison of Interpeak Latency wave across increasing stimulus intensities

At 60 dB SL, wave III and wave V amplitudes were significantly higher in cases compared to controls in both ears ($p < 0.05$), whereas wave I amplitude did not show a statistically significant difference.

Latency analysis showed that wave III and wave V latencies were significantly prolonged in cases in both ears ($p < 0.05$). Wave I latency remained comparable between cases and controls.

Similarly, interpeak latencies I–V and III–V were significantly increased in cases in both ears ($p < 0.05$), while I–III interpeak latency showed no statistically significant difference.

Finally, across increasing stimulus intensities (40–60 dB SL), cases demonstrated consistent prolongation of brainstem conduction time, predominantly affecting later waves (III and V) and long interpeak intervals (I–V, III–V), with preserved wave I latency, suggesting involvement of central auditory pathways.

Discussion

The present study demonstrates significant alterations in Brainstem Auditory Evoked Potential (BAEP) parameters among hypertensive patients compared with normotensive controls, indicating early and subclinical involvement of the central

auditory pathways. The principal findings include prolongation of wave III and V latencies, significant increase in inter-peak latencies III–V and I–V, with relative preservation of wave I latency, suggesting predominant central rather than peripheral auditory pathway dysfunction. Prolongation of wave III and V latencies reflects delayed neural conduction at the pontine and midbrain levels of the auditory pathway. Similar observations were reported by Tandon et al. [10], who attributed BAEP latency prolongation in essential hypertension to microvascular ischemic changes affecting the brainstem. Khullar et al. [11] also demonstrated significant prolongation of wave V latency and III–V inter-peak latency in hypertensive subjects, supporting the involvement of higher auditory relays.

The significant increase in inter-peak latencies III–V and I–V observed in the present study indicates impaired conduction between the cochlear nucleus, superior olivary complex, lateral lemniscus, and inferior colliculus. These findings are consistent with Roseline et al. [12], who suggested that chronic hypertension leads to progressive deterioration of central auditory conduction due to sustained vascular insufficiency and endothelial dysfunction.

The absence of significant changes in wave I latency and I–III inter-peak latency suggests relative preservation of peripheral auditory nerve function. This selective central involvement aligns with the findings of Gawali et al. [13], who reported predominant central auditory pathway dysfunction in hypertensive patients, with sparing of the auditory nerve.

Alterations in BAEP amplitudes observed in the present study likely reflect reduced neuronal synchrony and impaired recruitment of auditory neurons, secondary to chronic hypoperfusion of brainstem auditory nuclei. Similar amplitude reductions were reported by Sismanis et al. [14], who attributed these changes to vascular compromise affecting the auditory brainstem structures.

Further support is provided by Bhatia et al.[15], who demonstrated that BAEP abnormalities were more pronounced in patients with longer duration of hypertension, particularly prolongation of the III–V inter-peak latency, suggesting cumulative vascular damage. Although disease duration was not analyzed separately in the present study, consistent conduction delays across stimulus intensities support a chronic hypertensive effect on brainstem transmission.

The subclinical nature of these abnormalities is highlighted by Patil et al. [16], who reported delayed BAEP latencies in hypertensive patients despite normal audiometric findings, closely paralleling the present study. Verma et al. [17] further noted persistent BAEP abnormalities even in treated hypertensive patients, suggesting that structural brainstem changes may not be fully reversible with blood pressure control.

Population-based evidence from Rai et al.[18] demonstrated a positive association between systolic blood pressure levels and BAEP latency prolongation, indicating a dose–response relationship. Additionally, Kumar et al.[19] reported that BAEP abnormalities often precede changes in other evoked potentials, supporting the role of BAEP as an early and sensitive marker of hypertensive central nervous system involvement.

Taken together, the findings of the present study and previous literature strongly suggest that essential hypertension leads to early subclinical dysfunction of the auditory brainstem, even in the absence of overt neurological symptoms. Chronic elevation of blood pressure results in endothelial dysfunction, arteriolar narrowing, and microangiopathy, which collectively impair neural transmission within the brainstem auditory pathway.

Conclusion

The present study concludes that essential hypertension is associated with significant abnormalities in Brainstem Auditory Evoked Potentials, particularly prolonged wave III and V latencies, increased inter-peak latencies III–V and I–V, and reduced wave amplitudes. These changes indicate subclinical involvement of the central auditory pathways.

BAEP is a sensitive, objective, and non-invasive tool for early detection of central nervous system dysfunction in hypertensive patients and may be useful in routine evaluation and long-term monitoring.

Limitations and Future Directions

The present study was limited by a relatively small sample size and its cross-sectional design, which restricts causal inference and generalizability of the findings. Duration and severity of hypertension, as well as the effect of antihypertensive therapy, were not analyzed separately. Future longitudinal studies with larger cohorts, stratification by disease duration and treatment status, and correlation with imaging or other neurophysiological modalities are recommended to further elucidate hypertensive brainstem involvement.

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