Original Article

Brain Stem Evoked Auditory Response in Hypertension

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Abstract

Aim: To Assess the involvement of Peripheral and Central Brainstem Auditory Pathways in Hypertension Patients

Materials & Methods: Eligibility Criteria for Hypertension Patients in the Study: BP > 140/90 mmHg, as per JNC 7 Guidelines, and Under Control with Antihypertensive Medications. Subjects with Acute Illness were excluded. Informed Written Consent Acquisition. Informed Written Consent Acquisition in the Study was made to Ensure Ethical Participation.

Exclusion Criteria and Instrumentation in the Study: Patients with associated diseases (e.g., Diabetes Mellitus, Ischemic Heart Disease, Cerebrovascular Disease) or auditory abnormalities were excluded. Medicaid Neuroperfect plus Instrument was utilized, and electrodes were placed following the 10-20 international system of EEG electrode placement.

The parameters include:

1. Absolute latency of all the waves from I to V

2. Interpeak latency I-III, I-V and III-V

Result: The results of the study indicate that the Absolute Latency of Wave I in the hypertensive group showed a statistically significant prolongation, with a P value of 0.0001. This suggests that there is a notable delay in the auditory nerve response in these individuals compared to the control group.

The statistically significant prolongation observed in the latency values of Waves I, V, Inter-peak latency I-V, and Inter-peak latency III-V highlights the impact of hypertension on auditory function and warrants further investigation for a better understanding of the underlying mechanisms.

Conclusion: Individuals with hypertension show measurable delays in auditory nerve responses and the conduction of auditory signals along brainstem pathways. Further research in this area may enhance understanding of underlying mechanisms and explore innovative approaches to manage auditory abnormalities in hypertensive patients.

Key Words: Hypertension, BrainStem Evoked Auditory Response, Auditory pathways.

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Introduction

Essential hypertension is a major cause of premature vascular disease leading to cerebrovascular events, ischaemic heart disease and peripheral vascular disease. The risk of mortality or morbidity rises progressively with increasing systolic and diastolic pressures.[1]

Hypertension continues to be the predominant, easily identifiable, and reversible risk factor for various serious health conditions, including myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection, and peripheral arterial disease. With the growing prevalence of obesity and an aging population, the worldwide burden of hypertension is increasing, and it is projected to affect approximately 1.5 billion individuals globally by the year 2025. This highlights the urgent need for effective prevention, management, and public health initiatives to address the escalating impact of hypertension on global health.[2]

As of now, high blood pressure (BP) is responsible for approximately 54% of stroke cases and 47% of ischemic heart disease cases worldwide. Consequently, high BP remains the leading cause of death globally and represents a significant public health challenge. Addressing this critical issue is paramount to reducing the burden of stroke and heart disease and improving overall public health on a global scale. [3]

Hypertension can arise from various mechanisms, including malfunctions in ion pumps in smooth muscle cells of systemic arterioles, leading to chronic increases in vascular tone and elevated blood pressure. Essential hypertension often involves central nervous system dysfunction, potentially triggered by spasms in brain blood vessels. Severe hypertension may lead to brain microinfarctions and edema due to arteriolar spasms and fibrinoid degeneration. Additionally, small cerebral arteries become more susceptible to atheromatous changes, increasing the risk of lacunar infarctions. These mechanisms offer insights into how hypertension impacts the vascular system and the brain, providing a better understanding of its potential health consequences.[4]

Essential hypertension's dysfunction in brainstem regulatory mechanisms, interacting with sensory neuronal substrate, can lead to various clinical features of motor or sensory deficits. [5]

The primary objective of this study was to investigate alterations in neural generator activity and sensory conduction within the central nervous system (CNS) by monitoring auditory evoked potentials in patients diagnosed with essential hypertension.

Methods and Materials

In this study, Brainstem Auditory Evoked Responses (BAER) were conducted on a total of 20 hypertensive patients and 20 normotensive controls, all of whom were of the same age and sex. The auditory potentials were recorded using a Four Channel Digital Polygraph along with a Digital Intexcolor Monitor, 17" Model no: IT – 173 SB.

The research took place at the Department of Physiology, Thanjavur Medical College, Thanjavur. The study participants were selected from the general community residing in and around Thanjavur.

The goal of this investigation was to analyze and compare the auditory evoked responses between the hypertensive and normotensive groups, aiming to identify potential differences in neural generator activity and sensory conduction within the central nervous system associated with essential hypertension.

In this study, hypertensive patients were recruited based on specific criteria. Participants were required to have a confirmed diagnosis of hypertension with blood pressure readings consistently exceeding 140/90 mmHg, as per the guidelines set forth in the seventh report of the Joint National Committee (JNC 7) for hypertension. Additionally, all the included hypertensive patients were on antihypertensive

medications, and their blood pressure was under control at the time of enrollment.

Subjects with acute illnesses were excluded from the study to ensure that the data collected remained focused on the effects of essential hypertension, avoiding potential confounding factors introduced by acute health conditions.

Before participating in the study, all subjects provided informed written consent, demonstrating their willingness to be involved in the research while being aware of the study's objectives, procedures, and potential risks or benefits. This ensured that the study adhered to ethical standards and respected the autonomy and rights of the participants.

Patients with coexisting conditions, including Diabetes mellitus, Ischemic heart disease (IHD), and Cerebrovascular disease, as well as those presenting with any clinical auditory abnormalities or subjective symptoms of hearing loss, were not included in the study.

In this study, the Medicaid Neuroperfect Plus instrument was utilized. Electrodes were positioned following the 10-20 international system of EEG electrode placement. This standardized system ensures consistent and precise electrode positioning, facilitating accurate measurement and recording of brainstem auditory evoked potentials.[6]

In this study, following steps were followed during the examination and recording of Brainstem Auditory Evoked Potentials (BAEPs):

The subject was directed to avoid using hair oil after the last hair wash. A detailed examination of the external ear was conducted, removing earwax as necessary. The scalp was prepared for electrode contact by abrasion and degreasing. Electrodes were strategically placed for auditory stimuli delivery, and BAEPs were recorded using sound click stimuli. The waveform analysis identified Waves I to V, with specific characteristics for each wave. Interpeak latencies provided information on neural conduction times between these peaks, enhancing understanding of auditory processing.By following these steps and analyzing the obtained BAEP waveform, the study aimed to gather relevant data on the auditory evoked potentials and the interpeak latencies in the study participants.

The parameters include:

1. Absolute latency of all the waves from I to V
2. Interpeak latency I-III, I-V and III-V

Statistical method:

The electrophysiological parameters were analyzed using SPSS version 18, and the statistical analysis was conducted using the student's "t" test.

Result

In this study, a total of 40 subjects were included. The study group comprised 20 hypertensive patients, while the control group consisted of the remaining 20 normal subjects.

Hypertensive group

This group consisted of 20 subjects with ages ranging between 30 and 50 years.

Control group

This group consisted of 20 subjects with ages ranging between 30 and 50 years.

The Brainstem Auditory Evoked Potentials (BAEPs) values for the left and right ear did not show significant variation. An average of the BAEPs from both ears was calculated, and the combined data are presented in the analysis.

The P value was calculated using SPSS version in the data analysis, and the statistical test used was the student's "t" test. A P value of less than 0.05 was considered statistically significant for determining the significance of the results.

Table 1: Mean Values

	n	Mean	S.D	T	df	Statistical inference
I						
Control	20	1.4955	.26354	-3.854	38	.000<0.01
Hypertension	20	1.7815	.20165			Significant
II						
Control	20	2.4155	.40511	-.341	38	.735>0.05
Hypertension	20	2.4545	.31329			Not Significant
III						
Control	20	3.7387	.34486	-.311	38	.758>0.05
Hypertension	20	3.7880	.61932			Not Significant
IV						
Control	20	4.6023	.50905	.011	38	.992>0.05
Hypertension	20	4.6000	.78968			Not Significant
V						
Control	20	5.9280	.51699	-4.296	38	.000<0.01
Hypertension	20	6.7715	.70970			Significant
I - III						
Control	20	2.0065	.59002	-1.096	38	.280>0.05
Hypertension	20	2.1867	.43879			Not Significant
I - V						
Control	20	4.4325	.52187	-2.645	38	.012<0.05
Hypertension	20	4.9900	.78484			Significant
III - V						
Control	20	2.1893	.30533	-3.545	38	.001<0.01
Hypertension	20	2.9835	.95423			Significant

Table 1 shows the Mean values of Absolute and Inter-peak Latencies for both Hypertensive and Control groups. The study indicates that Wave I and Wave V have statistically significant prolongations in the Hypertensive group (P value of 0.0001 for both). Inter-peak latency I-V and III-V also demonstrate statistically significant prolongations in the Hypertensive group, with P values of 0.012 and 0.001, respectively.

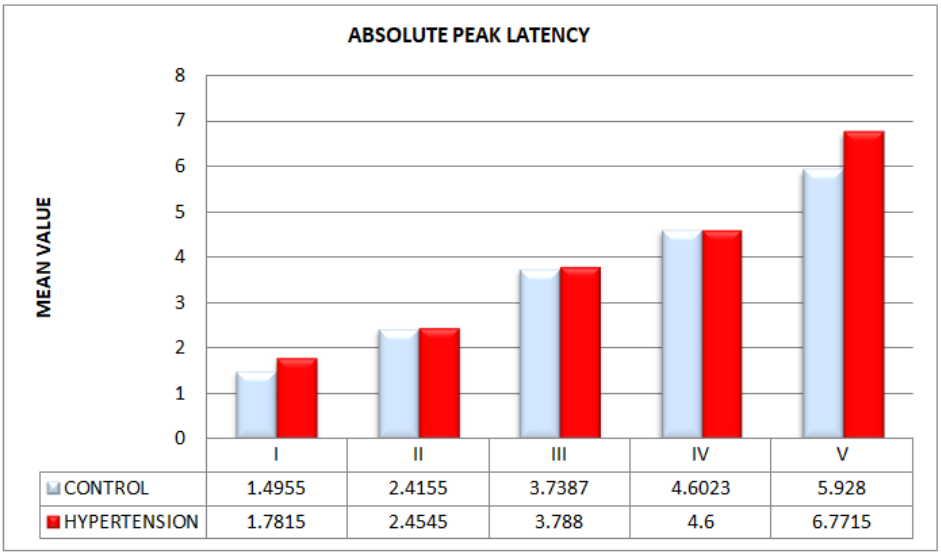

Figure 1

Figure 1 displays the Mean values of Absolute Latency for both the Hypertensive and Control groups. The results indicate that Wave I exhibits a statistically significant prolongation in the Hypertensive group, with a P value of 0.0001. Similarly, Wave V also shows a statistically significant prolongation in the Hypertensive group, with a P value of 0.0001.

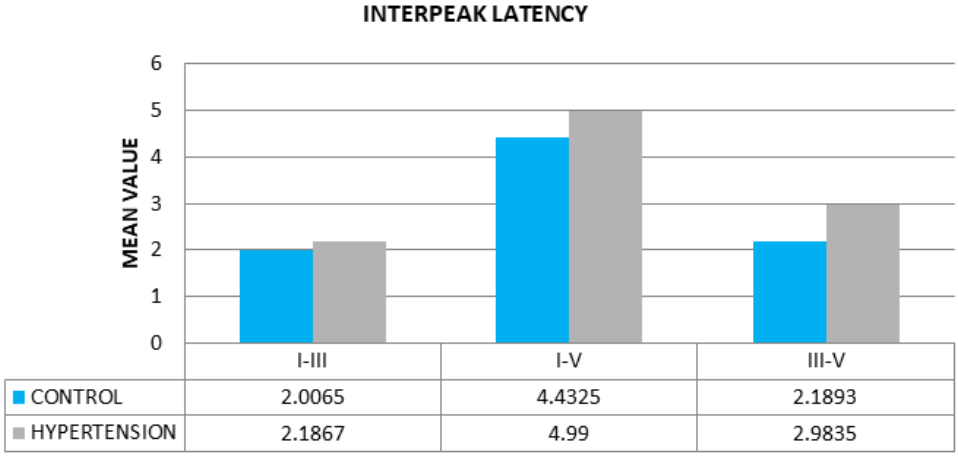

Figure 2

Figure 2 presents the Mean values of Inter-peak Latency for both the Hypertensive and Control groups. The analysis reveals that Inter-peak latency I-V demonstrates a statistically significant prolongation in the Hypertensive group, with a P value of 0.012. Additionally, Inter-peak latency III-V shows a statistically significant prolongation in the Hypertensive group, with a P value of 0.001.

Discussion

The findings of this study indicate a significant increase in the absolute peak latencies of wave I and V of Brainstem Auditory Evoked Potentials (BAEPs) in primary hypertensive patients. Moreover, both Interpeak latencies I-V and III-V were also observed to be significantly prolonged in the hypertensive group. These results suggest alterations in auditory processing and neural conduction in individuals with primary hypertension, potentially providing valuable insights into the effects of hypertension on auditory pathways.

This study reveals delayed auditory conduction in patients, especially at the inner ear and auditory pathways up to the medulla. Wave I reflects conduction through the inner cochlea, originating from the peripheral portion of the VIIIth nerve, while Waves V are thought to come from the Inferior colliculus nuclei in the brainstem. Interpeak latency I-V assesses conduction from the proximal VIIIth nerve through the pons to the medulla, and Interpeak latency III-V measures conduction from the lower pons to the medulla. These findings pinpoint specific areas of auditory processing delays in hypertensive patients, offering insights into the neurological effects of hypertension on the auditory system.

The regulatory mechanisms controlling arterial blood pressure in the brainstem may impact the functional integrity of auditory pathways. Electrophysiological evidence suggests an interaction between the brainstem's blood pressure control and sensory conduction in individuals with essential hypertension. These findings enhance our understanding of the intricate relationship between blood pressure regulation and auditory function in hypertensive individuals.

In a study by Rosen et al. in the United States, a correlation was found between high blood pressure and age-related hearing loss in high frequencies among hypertensive patients. However, a separate study by the same author, focusing on the Sudanese population, revealed that neither blood pressure nor hearing thresholds increased significantly with age.[7]

The findings of this study align with the pioneer research conducted by Tandon et al., which also reported the prolongation of latency in waves I, II,

and V of Auditory Brainstem Response (ABR), along with Interpeak Latency (IPL) III-V.[8]

In a separate study, 55 essential hypertensive patients and 55 normal elderly subjects aged between 55 and 89 years were included. Auditory Brainstem Responses (ABRs) were measured, along with serum cholesterol and triglyceride levels. The ABR results revealed that the latencies of wave V, IPL I-V, and IPL III-V were prolonged in comparison to the normal elderly subjects. These results are consistent with the findings of the present study.[9]

This study aligns with the findings of Khullar et al., which demonstrated Auditory Brainstem Responses in Essential Hypertension. The study group exhibited a significant increase in auditory threshold. Furthermore, the absolute peak latency of waves I, II, and V was significantly higher in hypertensive individuals compared to controls ($P< 0.05$). Additionally, Interpeak Latency (IPL) III-V was notably prolonged in the study group when compared with controls. These consistent results provide further evidence of the impact of hypertension on auditory processing and the neural conduction in auditory pathways. [10]

The results of the present study are in agreement with the findings of Goyal GL et al., which investigated the impact of Elevated Mean Arterial Pressure (MAP) and Pulse Pressure (PP) on Auditory Brainstem Responses. Both studies showed that hypertensive individuals had significantly higher absolute peak latencies of waves I and V, as well as prolonged IPL III-V, when compared to controls ($p< 0.05$). These consistent findings further support the association between hypertension and alterations in auditory processing and neural conduction in auditory pathways.[11]

Halwadi et al. found that primary hypertensive patients exhibited significantly increased peak latencies (waves I, IV, and V) in Brainstem Auditory Evoked Potential Responses compared to controls. The study revealed a notable correlation between mean arterial pressure and absolute peak latencies of BAEP waves I, IV, V, and interpeak latencies I-V and III-V in hypertensive individuals. These findings suggest a potential link between blood pressure regulatory mechanisms in the brainstem and the generators of different waves in BAEPs, consistent with prior research by Halwadi et al.[12]

Karamitsos et al. observed elevated absolute and interpeak latencies, as well as reduced peak amplitudes in Auditory Brainstem Responses (ABRs) of patients with ischemic heart disease compared to age-matched controls. The study suggests that BAEPs could serve as a non-invasive tool for assessing ischemic heart disease with impaired microcirculation, and it highlights the association between cardiovascular dysfunction, prolonged BAEPs, and the risk of reduced cerebral blood flow, leading to potential consequences like loss of consciousness and patient mortality.[13]

Auditory brainstem-evoked responses in 28 patients with benign intracranial hypertension syndrome revealed abnormalities, mainly prolonged interpeak latencies, possibly due to cochlear nerve compression. After managing intracranial hypertension, most patients demonstrated normalization or improvement in auditory brainstem-evoked responses. Further assessment is necessary to determine the diagnostic and prognostic value of this test due to the study's limited sample size.[14]

High pressure in the vascular system can potentially cause damage to the cochlear artery and anterior vestibular artery in the inner ear, resulting in a gradual loss of hearing in hypertensive patients. [15,16]

In hypertension, blood viscosity increases, which hinders capillary blood flow, consequently reducing the transport of oxygen to the cells of the inner ear. Additionally, sodium retention and increased extracellular volume occur in hypertensive individuals. These physiological changes may contribute to the development of hypoxia and hearing loss in these patients. [17]

Sensory derangement, including changes in pain threshold in experimental animals and impairments in sensory conduction in humans, has been reported in hypertension. [5,18,19]

Autopsy studies in chronic hypertension often show narrowed and sclerosed small arteries in the brain's subcortical regions. Magnetic resonance imaging (MRI) in chronic hypertension reveals

increased subcortical white matter lesions, micro-infarcts, astrogliosis, ventricular enlargement, and extracellular fluid accumulation compared to age-matched controls, indicating structural changes in the brain.[20-23]

The latencies of Auditory Brainstem Responses (ABR) reflect the speed of neural conduction in the corresponding segment of auditory pathways. In the hypertensive study group, the delay in the absolute latency (AL) of waves I and V suggests sensory deficits at both the level of the auditory nerve and the auditory pathways in the brainstem.

Furthermore, the interpeak latencies provide valuable insights into the time required for auditory information processing from one site to the next in the auditory pathway. Our observations indicate that auditory information processing time is notably affected, particularly from the pons to midbrain (IPL III-V) in the auditory pathway.

The delay in auditory processing time may be considered as a subclinical expression of both central and peripheral neuropathy and may serve as an index of the severity of visceral damage during hypertensive disease. These findings shed light on the potential impact of hypertension on the auditory system and its pathways, suggesting possible subclinical neurological effects.

Limitations of the Study:

The duration of hypertension may be a confounding factor. The study's small sample size and lack of imaging to exclude potential impacts on BAEPs, such as infarcts, lacunae, or leukoaraiosis, are limitations. Additionally, the effect of antihypertensive treatment on prolonged latencies of BAEPs was not investigated. Further studies are needed to address these limitations and improve upon the findings of this study.

Conclusion

The findings of this study reveal a significant delay in Absolute latencies and Inter-peak latencies in hypertensive cases, providing electrophysiological evidence of an interaction between the blood pressure control mechanism in the brainstem and sensory conduction in auditory pathways.

Ethical Clearance: Taken from Institutional Ethical Committee, Thanjavur Medical College, Thanjavur.

Source of Funding: Self

Conflicts of Interest: None declared.

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