Original Article

The impact of severity of hypertension on auditory brainstem responses

Abstract

Background: Auditory brainstem response is an objective electrophysiological method for assessing the auditory pathways from the auditory nerve to the brainstem. The aim of this study was to correlate and to assess the degree of involvement of peripheral and central regions of brainstem auditory pathways with increasing severity of hypertension, among the patients of essential hypertension. **Method:** This study was conducted on 50 healthy age and sex matched controls (Group I) and 50 hypertensive patients (Group II). Later group was further sub-divided into - Group IIa (Grade 1 hypertension), Group IIb (Grade 2 hypertension), and Group IIc (Grade 3 hypertension), as per WHO guidelines. These responses/potentials were recorded by using electroencephalogram electrodes on a root-mean-square electromyography, EP MARC II (PC-based) machine and data were statistically compared between the various groups by way of one-way ANOVA. The parameters used for analysis were the absolute latencies of Waves I through V, interpeak latencies (IPLs) and amplitude ratio of Wave V/I. **Result:** The absolute latency of Wave I was observed to be significantly increased in Group IIa and IIb hypertensives, while Wave V absolute latency was highly significantly prolonged among Group IIb and IIc, as compared to that of normal control group. All the hypertensives, that is, Group IIa, IIb, and IIc patients were found to have highly significant prolonged III-V IPL as compared to that of normal healthy controls. Further, intergroup comparison among hypertensive patients revealed a significant prolongation of Wave V absolute latency and III-V IPL in Group IIb and IIc patients as compared to Group IIa patients. These findings suggest a sensory deficit along with synaptic delays, across the auditory pathways in all the hypertensives, the deficit being more markedly affecting the auditory processing time at pons to midbrain (IPL III-V) region of auditory pathways among Grade 2 and 3 hypertensives. **Conclusion:** Hence, we conclude there has been greater involvement of pontomesenchymal region with the increasing severity of the disease.

Key words: Auditory brainstem responses, auditory pathways, hypertension, interpeak latency

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INTRODUCTION

Essential hypertension is associated with increased risk for cerebral, cardiac, and renal events. Subtle target-organ damage such as left-ventricular hypertrophy, microalbuminuria, and cognitive dysfunction takes place early in the course of hypertensive cardiovascular disease.[1]

Central nervous system dysfunctions are common in patients of essential hypertension due to micro-infarctions resulting from arterial and arteriolar spasm in cerebral blood vessels.[2,3] These changes are believed to contribute to hypoperfusion, loss of auto regulation, compromise of the blood-brain barrier and ultimately to subcortical white matter demyelination, and cognitive decline. The derangement of blood pressure (BP) regulatory mechanisms at brainstem level interacting with sensory neuronal substrate might be responsible for sensory deficits.[4] Such central neuronal damage and/or dysfunction may alter electrical activity in the central nervous system and may affect evoked potentials - somatosensory, auditory, and visual. Thus, current study aimed to further explore this phenomenon by investigating changes in brainstem auditory evoked potentials with increasing severity of disease in patients of essential hypertension.

MATERIALS AND METHODS

Following approval by the ethical committee of the institute, the present study of auditory brainstem responses (ABRs) was conducted on 50 patients of essential hypertension (age group 40-60 years), attending Medical Outpatients Department (OPD) of Guru Nanak Dev Hospital, Amritsar and compared with 50 normotensive (age and sex matched) controls.

Selection criteria

The criterion of considering a patient hypertensive was a BP >140/90 mmHg based on the average of two or more readings taken during each of his/her visits to the OPD.

These subjects were not on any antihypertensive medication. The patients were divided into various groups depending upon the grade of hypertension, as per WHO guidelines. The WHO BP classification includes three grades of hypertension.

All the patients were interviewed on a pretested proforma that included clinical history was completed for each subject. These subjects were investigated and diagnosed as cases of essential hypertension.

Exclusion criteria

The patients with any associated diseases such as diabetes mellitus, ischemic heart disease (IHD), cerebrovascular disease, hyperlipidemia, renal disease and having any clinical auditory abnormality, or subjective symptoms of hearing loss were excluded from the study.

The patients were explained the procedure and informed written consent was taken before carrying out the procedure.

The controls were having a diastolic pressure <90 and systolic pressure below 140 mm of Hg.

Auditory brainstem responses were performed on an outpatient basis in the Department of Physiology, Government Medical College, Amritsar using root-mean-square electromyography EP MARC II 2CH (PC based) machine. The subjects were asked to wear earphones and electrodes placed on the top of head (vertex) and mastoid process. Active electrode was placed at ipsilateral mastoid process (Ai), reference electrode was placed at Cz and Grounding electrode was placed at the forehead (Fz). Electrical impedance was kept below 5 kΩ. Auditory stimuli were delivered through the earphones to the ear being tested while masking the other one with white noise of 40 dB.

Auditory brainstem responses were recorded using standardized technique after giving 2000 sound click stimuli of intensity 70 dB above normal hearing threshold, at 11/s frequency and 0.1 ms duration. The signals picked up by the electrodes were recorded using filter bandpass of 300-3000 Hz with artifact rejection level up to 25 μV. 2-3 repetitions of the recording were done to ensure reproducibility that is - latency measured on separate recordings agreed with each other within 0.1 ms or less.

The following parameters were measured for analysis of ABRs in the patients and controls:

1. Absolute latency of Waves I to V
2. Interpeak latencies (IPLs)
3. Amplitude ratio of Wave V/I.

Data were analyzed by means of unpaired Student's *t*-test for the comparison of control and study groups. Further the multigroup comparison was done using one-way ANOVA. Thus, where there was a significant difference, Turkey's honestly significant difference (Turkey's honestly significant difference) *post-hoc* test was used to identify the source of the significance. The whole data had been assessed by Levene's test for equality of variance.

RESULTS

Hypertensive group

There were 50 subjects in this group with their age between 40 and 60 years, average being 50.2 ± 5.9 years. They had an average weight: 67.3 ± 5.8 kg, height: 166.4 ± 7.0 cm, body surface area: 1.75 ± 0.11 kg/m^2, Systolic BP (SBP): 148.6 ± 15.5 mmHg and diastolic BP (DBP): 97.2 ± 7.5 mmHg. On the basis of the extent of raised BP, these subjects belonged to Grade 1, 2, and 3 hypertension as per WHO/ISH guidelines.

Control group

There were 50 subjects in this group between 40 and 60 years of age with the average being 51.1 ± 6.9 years. They had a mean weight: 64.0 ± 5.6 kg, height: 166.3 ± 5.6 cm, body surface area: 1.73 ± 0.09 kg/m^2, SBP: 121 ± 5.5 mmHg, and DBP: 78.4 ± 3.5 mmHg.

Since values of BEAPs of left and right ear did not vary significantly, an average of the two ears was calculated and composite data are given.

Data are shown in Table 1 revealed statistically significant increase in the mean values of absolute peak latencies of Waves I, and III-V, in Group IIa (Wave I 1.76 ± 0.22 and III-V 2.00 ± 0.16) when

Table 1: Comparison of BAEP parameters of Group I with Group IIa

Parameters	Group I Mean ± SD	Group IIa Mean ± SD	*P* value	Significance
I	1.6260±0.20247	1.7670±0.22504	0.04	S
II	2.7296±0.19344	2.7320±0.21564	1	NS
III	3.751±0.1573	3.667±0.2032	0.36	NS
IV	4.8292±0.20956	4.7855±0.20119	0.89	NS
V	5.5420±0.21333	5.6750±0.16211	0.10	NS
I-III	2.1196±0.28662	1.9040±0.32150	0.14	NS
I-V	3.9104±0.31410	3.9115±0.34368	1	NS
III-V	1.7924±0.21693	2.0090±0.15791	0.00	HS
Amplitude R	1.6870±1.55262	2.0710±2.18150	0.79	NS

BAEP = Brainstem auditory evoked potentials, SD = Standard deviation

compared with Group I healthy controls (Wave I 1.62 ± 0.20; IPL I-III 1.90 ± 0.32 and III-V 1.79 ± 0.21).

Table 2 shows an increase in the mean values of absolute peak latencies of Waves I, V, and IPL III-V, in Group III when compared with Group I. The increase in the absolute peak latency of Wave I was statistically significant, while that in Wave V and IPL III-V was statistically highly significant ($P < 0.001$).

Table 3 shows, a statistically highly significant ($P < 0.001$) prolongation in absolute peak latency of Wave V and IPL III-V in Group IIc (Wave V 5.99 ± 0.30 and III-V 2.31 ± 0.27) as compared with Group I (Wave V 5.54 ± 0.21 and III-V 1.79 ± 0.21).

Table 4 shows an increase in Wave V absolute peak latency and III-V IPL in Group IIb (Wave V 5.85 ± 0.24 and III-V 2.19 ± 0.16) in comparison with Group IIa (Wave V 5.67 ± 0.16 and III-V 2.00 ± 0.16), the increase being statistically significant ($P < 0.05$).

Similarly, as depicted in Table 5, a statistically significant ($P < 0.05$) prolongation was found in Wave V absolute peak latency and III-V IPL in Group IIc (Wave V 5.99 ± 0.30 and III-V 2.31 ± 0.27) as compared with Group IIa (Wave V 5.67 ± 0.16 and III-V 2.00 ± 0.16).

However, we could not find any statistically significant change in ABRs parameters in Group IIc as compared to Group IIb ($P > 0.05$) as is evident from Table 6.

Table 2: Comparison of BAEP parameters of Group I with Group IIb

Parameters	Group I Mean ± SD	Group IIb Mean ± SD	*P* value	Significance
I	1.6260±0.20247	1.7870±0.17859	0.01	S
II	2.7296±0.19344	2.7252±0.19718	1	NS
III	3.751±0.1573	3.659±0.2324	0.24	NS
IV	4.8292±0.20956	4.7109±0.31742	0.20	NS
V	5.5420±0.21333	5.8517±0.24325	0.00	HS
I-III	2.1196±0.28662	1.8726±0.32625	0.11	NS
I-V	3.9104±0.31410	4.0665±0.35907	0.26	NS
III-V	1.7924±0.21693	2.1943±0.15957	0.00	HS
Amplitude R	1.6870±1.55262	1.5696±1.10187	0.99	NS

BAEP = Brainstem auditory evoked potentials, SD = Standard deviation

Table 3: Comparison of BAEP parameters of Group I with Group IIc

Parameters	Group I Mean ± SD	Group IIc Mean ± SD	*P* value	Significance
I	1.6260±0.20247	1.8171±0.14784	0.08	NS
II	2.7296±0.19344	2.8029±0.24743	0.80	NS
III	3.751±0.1573	3.676±0.2759	0.77	NS
IV	4.8292±0.20956	4.7586±0.20860	0.88	NS
V	5.5420±0.21333	5.9971±0.30462	0.00	HS
I-III	2.1196±0.28662	1.8643±0.36859	0.17	NS
I-V	3.9104±0.31410	4.1829±0.41011	0.19	NS
III-V	1.7924±0.21693	2.3171±0.27675	0.000	HS
Amplitude R	1.6870±1.55262	1.4514±0.40945	0.98	NS

BAEP = Brainstem auditory evoked potentials, SD = Standard deviation

Table 4: Comparison of BAEP parameters of Group IIa with Group IIb

Parameters	Group IIa Mean ± SD	Group IIb Mean ± SD	*P* value	Significance
I	1.7670±0.22504	1.7870±0.17859	0.98	NS
II	2.7320±0.21564	2.7252±0.19718	1	NS
III	3.667±0.2032	3.659±0.2324	0.99	NS
IV	4.7855±0.20119	4.7109±0.31742	0.73	NS
V	5.6750±0.16211	5.8517±0.24325	0.04	S
I-III	1.9040±0.32150	1.8726±0.32625	0.98	NS
I-V	3.9115±0.34368	4.0665±0.35907	0.44	NS
III-V	2.0090±0.15791	2.1943±0.15957	0.01	S
Amplitude R	2.0710±2.18150	1.5696±1.10187	0.72	NS

BAEP = Brainstem auditory evoked potentials, SD = Standard deviation

Table 5: Comparison of BAEP parameters of Group IIa with Group IIc

Parameters	Group IIa Mean ± SD	Group IIc Mean ± SD	*P* value	Significance
I	1.7670±0.22504	1.8171±0.14784	0.94	NS
II	2.7320±0.21564	2.8029±0.24743	0.85	NS
III	3.667±0.2032	3.676±0.2759	1	NS
IV	4.7855±0.20119	4.7586±0.20860	0.99	NS
V	5.6750±0.16211	5.9971±0.30462	0.006	S
I-III	1.9040±0.32150	1.8643±0.36859	0.99	NS
I-V	3.9115±0.34368	4.1829±0.41011	0.26	NS
III-V	2.0090±0.15791	2.3171±0.27675	0.004	S
Amplitude R	2.0710±2.18150	1.4514±0.40945	0.80	NS

BAEP = Brainstem auditory evoked potentials, SD = Standard deviation

Table 6: Comparison of BAEP parameters of Group IIb with Group IIc

Parameters	Group IIb Mean ± SD	Group IIc Mean ± SD	*P* value	Significance
I	1.7870±0.17859	1.8171±0.14784	0.98	NS
II	2.7252±0.19718	2.8029±0.24743	0.81	NS
III	3.659±0.2324	3.676±0.2759	0.99	NS
IV	4.7109±0.31742	4.7586±0.20860	0.96	NS
V	5.8517±0.24325	5.9971±0.30462	0.41	NS
I-III	1.8726±0.32625	1.8643±0.36859	1.0	NS
I-V	4.0665±0.35907	4.1829±0.41011	0.85	NS
III-V	2.1943±0.15957	2.3171±0.27675	0.48	NS
Amplitude R	1.5696±1.10187	1.4514±0.40945	0.99	NS

BAEP = Brainstem auditory evoked potentials, SD = Standard deviation

DISCUSSION

The effect of hypertension on the sensory conduction in the auditory pathway has already been studied. The findings of a pioneer study in North India showed the prolongation of latencies of all waves of ABRs along with IPL III-V.[5] In another study, 55 essential hypertensive patients and 55 normal elderly subjects between the ages of 55 and 99 years were selected. ABRs were measured along with serum cholesterol and triglyceride levels. The results of the ABR demonstrated that the latencies of Wave V, IPL I-V, and IPL III-V were prolonged compared

with normal elderly subjects. The patient's duration of illness and the complications of hypertension had an influence on hearing disorders in relation to the ageing process. Hence, they concluded that the hearing disorders in the elderly people are a result of long duration of illness and the complications of hypertension.[6]

Karamitsos *et al.* studied ABRs in 30 patients of ischemic heart disease and in an equal number of healthy age-matched control subjects. The parameters measured were absolute latency of Waves I through V, the IPLs I-III, III-V, and I-V, and the peak amplitudes of Wave I, III, and V. The measured absolute latencies and IPLs were found to be significantly increased, and the peak amplitudes were found to be diminished in the study group. Hence, ABRs may become part of the noninvasive assessment IHD and essential hypertension patients.[7] Auditory brainstem-evoked responses were conducted on 28 patients with otologic symptoms (pulsatile tinnitus, hearing loss, and aural fullness) secondary to benign intracranial hypertension syndrome. Abnormalities consisting mainly of prolonged IPLs were detected in one-third of these patients. It is speculated that the pathophysiologic mechanisms responsible for these auditory brainstem-evoked abnormalities are stretching-compression of the cochlear nerve in the brainstem, caused by the intra-cranial hypertension and/or primary edema. Normalization or improvement was noticed in the majority of the patients after management of intra-cranial hypertension.[8] Since, the number of patients in this study is small, the diagnostic and prognostic value of this test needs further evaluation. Significant prolongation of Waves IV, V, and VII of ABRs were also seen in rabbits with intra-cranial hypertension compared with controls.[9] Another study[10] showed that raised BP in preeclamptic women may affect the vascular responses of the blood vessels in the brain and cause ischemic delay in P1 latency of visual evoked potentials. This may also explain the delay in ABR latencies in hypertensive cases. Narrowing and sclerosis of small penetrating arteries in the subcortical regions of the brain are common findings on autopsy in chronic hypertension. Magnetic resonance imaging studies in persons with chronic hypertension have revealed greater numbers of subcortical white matter lesions and micro-infarcts, astrogliosis, ventricular enlargement, and extracellular fluid accumulation than in age-matched controls.[11-14]

However, the changes in the electrophysiological correlates were not studied in terms of increasing severity of the disease among the hypertensives. Our study, though preliminary, is first of its kind in evaluating the changes in ABRs in terms of increasing severity of hypertension. Our findings suggest the involvement of pontomesenchymal region especially pons to midbrain (IPL III-V) region along the auditory pathways with the increasing severity of hypertension. The delay in auditory processing time may be considered a subclinical expression of central as well as peripheral neuropathy and an index of the gravity of visceral damage during hypertensive disease.

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