

Original article

Visual evoked potential and Brainstem auditory evoked potential changes in primary hypertension

*Jyoti Nigam¹, Ashish Shrivastava², Leena Jain³

1- Resident, Department of Physiology, Gajra Raja medical College, Gwalior, Madhya Pradesh, India,

2- Assistant Professor, Department of Surgery, Gajra Raja medical College, Gwalior, Madhya Pradesh, India

3- Associate Professor, Department of ENT, Peoples college of medical sciences and research center, Bhopal, Madhya Pradesh, India,

Corresponding author *

Abstract

Introduction: Systemic hypertension or Essential hypertension is a major public health problem, though usually asymptomatic, hypertension may lead to a number of complications involving almost all organ systems but its effects on brain contribute to the major part of morbidity and mortality. VEP and BERA have also been used to study changes in hypertensive patients.

Method: In the present study, 30 patients of primary hypertension were examined to enroll in the study along with a group of 30 normotensive individuals as controls. The study was aimed at assessing the VEP and BAEP of these subjects and to compare the results between the hypertensive and normotensives and to find correlation, if any, between the blood pressure and various parameters of VEP and BAEP.

Observation and Result: Mean values of VEP in the two groups of subjects compared, showed statistically non-significant increase of absolute latencies of VEP waves. Prolongation of the absolute latencies of BAEP waves I and IV in hypertensive group compared to normotensive group showed statistically significant difference. The latencies of other absolute waves were also found to be prolonged but the statistical significance could not be established.

Conclusion : These changes in our group of subjects support our hypothesis and need further confirmation in patients with longer duration of disease as well as study of effect of various factors such as age, target organ involvement and anti-hypertensive drugs.

Keywords: Brainstem auditory evoked potential, Visual evoked potential, primary hypertension

Introduction

Systemic hypertension or Essential hypertension is a major public health problem in fact is one of the most common chronic diseases in our society. Though usually asymptomatic, hypertension may lead to a number of complications involving almost all organ systems but its effects on cardiovascular system, the kidneys and the brain contribute to the major part of morbidity and mortality. Central nervous system dysfunction occurs frequently in patients with primary hypertension⁽¹⁾. This might be due to arterial and arteriolar spasm in the blood vessels of the brain⁽²⁾,

which in combination with fibrin degeneration of small arteries leads to microinfarction and brain edema in a severe case of hypertension. In addition, hypertension predisposes small penetrating cerebral arteries to vascular endothelial changes including hyalinization leading onto demyelination and infarction, in the gray nuclei and white matter.⁽³⁾ Such demyelination might lead to dementia through disconnection of subcortical-cortical association pathways⁽⁴⁾. Subclinical changes in CNS function may not be easily detected with traditional methods of medical assessment⁽⁵⁾. Today the availability of more sensitive and non-invasive methods like the Brainstem auditory evoked

potentials (BAEPs) and Visual evoked potentials (VEPs), to evaluate subtle alterations in CNS functions allow us to better correlate blood pressure with possible early brain alterations. Visual evoked potential (VEP) and Brainstem auditory evoked potential (BAEP) have proven to be very useful markers in detecting subclinical abnormalities involving the visual and auditory pathways.

Aims & Objectives:

1. To compare the sensitivity of BAEP and VEP changes in primary hypertensive individuals
2. To correlate the changes in VEP and BAEP, if any, with the severity of hypertension

Material & Methods:

The study of Brainstem Auditory Evoked Potentials and Visual evoked potential in individuals in the age group of 30 to 70 yrs and its relationship to essential hypertension was conducted in a government hospital.

Selection of subjects

The study comprised of 30 subjects of essential hypertension selected from hypertensive clinic and 30 controls selected from amongst the staff of government hospital. An informed written consent was taken and a proforma for a detailed medical history was filled for all the subject.

Inclusion criteria

1. Subjects of either sex in age group of 30 to 70 yrs.
2. Subjects having normal hearing.
3. Subjects having normal vision.

Exclusion criteria

1. Patients with <30 and >.70 yrs in age.
2. Patients with any positive history or physical examination findings suggestive of CNS disease.
3. Patients with well controlled blood pressure.
4. Subjects with metabolic disorders, known to affect hearing and or vision like diabetes , hypot-hyroidism etc.

5. Subjects with pathology in the eye, which precludes examination of fundus or in which VEP is known to be affected or uncorrected refractive errors.
6. Subject with any external ear, middle ear or cochlear disease.
7. Subjects taking ototoxic drugs .
8. Subjects with history of chronic smoking and /or alcohol abuse.

Study design :Control group

Age and sex matched controls were normotensives and free from the diseases were selected. Subjects with SBP \leq 120 mmHg or DBP \leq 80mmHg were enlisted after obtaining an informed written consent. Detailed history was taken and physical examination performed to rule out hearing and vision abnormality. The BP was measured using sphygmomanometer under ideal conditions. Blood samples were collected for hematological and bio-chemical parameters like Hemogram, lipid profile, blood sugar, serum creatinine. Apart from these routine urine examination was also done.

The patients were excluded from the study if they had shown any probable cause of secondary hypertension, based on physical examination, history and above mentioned lab test. The patients were then graded as per the JNC VIII classification of hypertension and were investigated further. Apart from these the patients were also tested for an ECG. Patients with Hb more than 10gm% with normal RBS, Blood urea and Sr. creatinine were then enrolled for the study. The patients were excluded from the study if they displayed any probable cause of secondary hypertension based on history, physical examination and above mentioned laboratory tests.

On the suitable patients BERA and VEP were performed using RMS Portable Aleron and RMS Mark II machines.

Observations & results:

Visual evoked potentials

Mean values of VEP in the two groups of subjects compared, showed statistically non-significant increase of absolute latencies of VEP waves. In the present study the group of hypertensive patients showed a statistically non-significant but prolonged latency of waves N₁₄₅ & P₁₀₀ in Hypertensive group (Table 1)Brainstem auditory evoked potentials

Prolongation of the absolute latencies of BAEP waves I and IV in hypertensive group compared to normotensive group showed statistically

significant difference. The latencies of other absolute waves were also found to be prolonged but the statistical significance could not be established (Table 2). The present study also showed non-significant but, prolonged IPLs in hypertension group when compared to the control group (Table 3). The current study also showed correlation between systolic blood pressure and absolute wave latencies of waves I. Our study also showed correlation between diastolic blood pressure and absolute latencies of wave I (Table 4)

Table 1 : Mean values of wave latencies in VEP in the two groups .

	Normotensives Mean±SD (n=30)	Hypertensives Mean±SD (n=30)	P value
N ₇₅	64.3±4.60	64.58±6.551	> 0.05 (NS)
P ₁₀₀	92.42±3.91	96.43±6.480	> 0.05 (NS)
N ₁₄₅	130.5±11.59	137.1±10.56	> 0.05 (NS)

S: Significant (P<0.05); NS : Non Significant (P>0.05)

Table 2 : Mean values of absolute latencies of BAEP of both ears in the two groups

Absolute Latencies	Normotensives Mean±SD (n=30)	Hypertensives Mean±SD (n=30)	P value
Wave I (msec)	1.709 ±0.16	1.737 ±0.21	<0.05 (S)
Wave II (msec)	2.76±0.12	2.799±0.18	> 0.05 (NS)
Wave III (msec)	3.583±0.20	3.685±0.19	> 0.05 (NS)
Wave IV (msec)	4.813±0.16	4.820±0.21	< 0.05 (S)
Wave V (msec)	5.48±0.19	5.59±0.28	> 0.05 (NS)

S: Significant (P<0.05); NS : Non Significant (P>0.05)

Table 3 : Mean values of BAEP in both ears – interpeak latencies (IPL) and amplitude of wave I and wave V in two groups

	Normotensives Mean±SD (n=30)	Hypertensives Mean±SD (n=30)	P value
IPL I-III (msec)	1.878±0.18	1.98±0.21	> 0.05 (NS)
IPL III-V (msec)	1.90±0.28	1.89±0.34	> 0.05 (NS)
IPL I-V (msec)	3.78±0.20	3.87±0.32	> 0.05 (NS)
Amplitude I (µv)	0.31±0.18	0.36±0.25	> 0.05 (NS)
Amplitude V (µv)	0.55±0.21	0.57±0.30	> 0.05 (NS)

S: Significant (P<0.05); NS : Non Significant (P>0.05)

Table 4 : Correlation between Blood Pressure and BAEP latencies (Karl Pearson’s coefficient of correlation)

BAEP Waves	Systolic BP Correlation coefficient (r)	Diastolic BP Correlation coefficient (r)
Wave I	0.56 (S)	0.51 (S)
Wave IV	0.08 (NS)	0.16 (NS)

* Significant correlation (r>0.5)

Discussion:

In the present study the group of hypertensive patients showed a statistically non-significant but prolonged latency of waves N_{145} & P_{100} in Hypertensive group (Table 1). The findings are similar to the results obtained in a similar study conducted by Tandon et al⁽⁶⁾. They showed that the N_{75} latency is prolonged in the hypertensive patients, as compared to the controls. There was no significant difference in the P_{100} latency in present study group of hypertensive individuals compared to the controls, though there was increase in the mean value in the hypertension group (Table 1). This finding is similar to the observation made earlier by Marsh et al⁽⁷⁾ in their study of VEP in pregnant women with preeclampsia. There were no significant changes in the latency of P_{100} between normotensive and the pre-eclamptic pregnant women in their study. Tandon et al⁽⁶⁾ also found a prolonged P_{100} latency in their study group of hypertensive patients. Complex neurohormonal mechanism, environmental and genetic factors are hypothesized to interplay in the development and maintenance of hypertension. Histologically, cerebral parenchymal arterioles show a thickened media and a narrowed lumen. Such changes may reduce the vasodilatory capability of cerebral blood vessels and thereby limit maximum delivery of oxygen and nutrients to active brain tissue. Hypertension may change the permeability of the vascular wall to vasoregulatory compounds and thus alter the responsiveness of the vasculature of metabolic needs of the tissue⁽⁸⁾. Richard et al⁽⁸⁾ have given the hypothesis that hypertension alters the responsiveness of the cerebrovasculature to neural activation. Responsiveness may decline because of chronic arteriolar vasoconstriction and reduced distensibility of nutritive vessels resulting from the homeodynamic adjustment of the brain to systemic hypertension⁽⁹⁾. The current study

showed an increase in the absolute latencies of all the waves of BAER. The changes were significant in the absolute latencies of Wave I and Wave IV (Table 2). The results of the present study are in accordance with those reported by Tandon et al⁽⁶⁾ where a significant increase in the absolute peak latencies of I, II, V and IPL III-V in one patient with stage 3 hypertension was noted. The results of the present study also showed a similar trend of raised latencies but significant increase was found in wave I and wave IV. The difference was however not more significant in comparison to control in both the studies. This could be due to the small number of patients. A large study is more likely to show some significant findings. The present study also showed non-significant but, prolonged IPLs in hypertension group when compared to the control group (Table 2). These findings are consistent with those observed by Tandon et al⁽⁶⁾. Study by Tandon et al⁽⁶⁾ suggests that the changes were more severe in the higher degrees of hypertension. Since in our study most subjects are in pre hypertensive or stage 1 hypertensive group the relation of increased latencies with severity of hypertension cannot be justified.

The current study also showed correlation between systolic blood pressure and absolute wave latencies of waves I. Our study also showed correlation between diastolic blood pressure and absolute latencies of wave I (Table 4). These findings were similar to those found by Tandon et al⁽⁶⁾.

The findings of the current study, thus suggest that hypertension does affect the neuronal excitation/conduction in the visual and auditory pathways. The absence of significant differences between all the waves of hypertensives and normotensives could be due to the small number of subjects in all the groups and this needs larger studies for further interpretation. As to what is the

exact cause of this derangement is still a matter of debate.

The changes in hypertensive patients, as observed, can be due to multiple factors that include associated dyslipidemia, oxidative stress or arteriosclerosis leading to poor blood supply to different organs. Due to paucity of studies conducted in this aspect, a definite cause cannot be attributed to these changes. However, in view of the consistent changes seen in many studies, it does emphasize the need for more research in this field.

Conclusion:

There are changes in the VEP in patients with hypertension but these changes were not statistically significant. No correlation could be established between these changes and the severity of hypertension, at least in the early stages of hypertension. However changes in higher grades of hypertension needs further exploration. Further

studies are needed to establish that the changes are also related to the duration of illness. There are changes in the BAEP in patients with hypertension but the result was statistically significant from normotensive group only in absolute wave latencies of wave I & IV. However statistical significance could not be established for rest of the parameters.

BAEP appeared to be more sensitive and early indicator of these changes than in VEP.

It is concluded that further studies are needed to substantiate these changes, especially in large number of patients in various grades of severity of hypertension. These changes in our group of subjects supports our hypothesis and needs further confirmation in patients with longer duration of disease as well as study of effect of various factors such as age, target organ involvement and anti-hypertensive drugs.

References:

1. Williams GH. Hypertensive vascular disease. In: Isselbacher KJ, Braunwald D, Wilson JD, Martin JB, Fauci AS, Kasper DL, editors. Harrison principles of internal medicine. 13th ed. New York: McGraw-Hill Inc; 1994. p.1116-31.
2. Fredric MW. Cerebrovascular disease. In: Conn HL, Horwitz O, editors. Cardiac and vascular diseases. Philadelphia: Lea & Febiger; 1971. p.1473-99.
3. Tandon OP, Ram D, Awasthi R. Brainstem auditory evoked responses in primary hypertension. Indian J Med Res. 1996; 104(NOV): 311-5.
4. Smoog I, Lernfelt B, Landahl S, Palmertz B, Andreasson LA, Nilsson L, et al. 15 year longitudinal study of blood pressure and dementia. The Lancet. 1996;347:1141-45.
5. P. Cicconetti, M Cacciafesta. Event related potentials in the elderly with new mild hypertension. Clin and Exper Hypertension. 2000;22(6):583-93.
6. Tandon OP, Ram D. Visual evoked potentials in primary hypertension Indian J Physiol Pharmacol 1997 Apr;41(2):154-8.
7. Richard J, Mathew FM. Cerebral blood flow in hypertensive patients. New York, NY: Raven Press; 1993.
8. Koska J, Syorva D. Malondialdehyde, lipofuscin and activity of antioxidant enzymes during physical exercise in patients of essential hypertension. J Hypertens. 1999;17:529-35.
9. Walker BB, Sandman AC. Visual evoked potentials change as heart rate and carotid pressure change. Psychophysiology. 1982;19:520-7.