International Journal Dental and Medical Sciences Research (IJDMSR)
ISSN: 2393-073X Volume 2, Issue 1 (Jan- 2018), PP 38-44
www.ijdmsr.com

"A Study on Visual, Brainstem Auditory and Cognitive Evoked Potential In Prehypertension And Hypertension" In Saraswathi Institute of Medical Sciences Hapur Uttar Pradesh.

Dr. Manisha Shrivastwa*, Dr. Shreetesh Mishra**
Department of Physiology, Saraswathi institute of Medical Sciences, Hapur Uttar Pradesh India
**Corresponding author: Dr. Manisha Shrivastwa*

ABSTRACT:
Introduction: Hypertension is the new era pandemic which is the leading cause of mortality in the world and is ranked third as a cause of disability-adjusted life years. Prehypertension is an American medical classification for cases where a person's blood pressure is elevated above normal, but not to the level considered hypertension (high blood pressure). Prehypertension is blood pressure readings with a systolic pressure from 120 to 139 mm Hg or a diastolic pressure from 80 to 89 mm Hg.The seventh report of the Joint National Committee (JNC 7) proposed the new labeling for elevated blood pressure values below 140/90 to more accurately communicate the tendency of blood pressure to rise with age. Hypertension is present if the resting blood pressure is persistently at or above 140/90 mmHg. Hypertension is also a risk factor for cognitive impairment and dementia. Other complications include hypertensive retinopathy and hypertensive nephropathy. . In addition, hypertension predisposes small penetrating cerebral arteries to vascular endothelial changes including hyalinization leading into demyelination and infarction, in the gray nuclei and white matter. Such demyelination might lead to dementia through disconnection of sub-cortical-cortical association pathways. Visual Evoked Potential are evoked potentials, which are generated by the Nervous system in response to photo stimulation. Brainstem Auditory Evoked Potentials (BAEPs) are a type of evoked potentials.
Aims & Objective: The preesent study was undertaken to estimate the effect of **prehypertension and hypertension on visual,brainstem auditory and cognitive evoked potential in saraswathi institute of medical sciences hapur uttar pradesh.** VEP, BAEP AND ERP test were performed and different wave pattern recorded. the test was performed on RMS EMG EP MARK II (RMS(P) LTD. CHANDIGARH).
MATERIALS AND METHODS: The present study was carried out in Department of Physiology, Saraswathi Institute of Medical Sciences, Anwarpur, Hapur. The study was an open randomized comparative study. study population is grouped into,group 1 - 30 controls (normotensives), group 2 – 30 prehypertensives, group 3 – 30 hypertensives for duration less than 5 years, group 4 – 30 Hypertensives for duration more than 5 years
RESULT: The findings of the current study, thus suggests that hypertension does affect the neuronal excitation/conduction in the visual and auditory pathways and it is related to the duration of hypertension. The central neuropathy involving auditory pathway may be impaired earlier than visual pathway as seen in prehypertension. BAEP is appeared to be more sensitive and early indicator of these changes than VEP and ERP.
CONCLUSION: The present study identified that hypertension affects visual pathway and these changes in visual pathway becomes severe as duration of illness increases. Statistically significant delay in P100 latency suggests that the development of Hypertensive neuropathy subclinically, occurs in very early stages of Hypertension, which can be detected by VEP even before the onset of overt neuropathy.Our study also showed that hypertension also affects auditory pathways and there was bilateral abnormality in peripheral portion of the auditory nerve and progresses to cochlear nucleus, however there was unilateral abnormality in lateral lemniscus (pons) in left ear and in inferior colliculus (midbrain) in right ear, it is further concluded that the first change in auditory pathway may be attributed at level of lateral leminiscus seen in prehypertensives in left earwhich may be interpreted as beginning of central neuropathy.

Keywords: prehypertension, hypertension, VEP, ERP, BAEP

I. INTRODUCTION

Hypertension is also a risk factor for cognitive impairment and dementia. Other complications include hypertensive retinopathy and hypertensive nephropathy. Central nervous system dysfunction occurs frequently in patients with hypertension. This might be due to arterial and arteriolar spasm in the blood vessels of the brain, which in combination with fibrin degeneration of small arteries leads to microinfarction and brain edema in a

severe case of hypertension. Hypertension further appears to modify and accelerate atherosclerotic cerebral vascular disease, with an increasing incidence of thrombotic infarction. In addition, hypertension predisposes small penetrating cerebral arteries to vascular endothelial changes including hyalinization leading into demyelination and infarction, in the gray nuclei and white matter. Such demyelination might lead to dementia through disconnection of sub-cortical-cortical association pathways. Similar demyelinating process might occur in vulnerable areas of the brain and cause conductive problems in sensory tracts. This produces a variety of clinical features of motor or sensory deficits, along with the usual symptoms of occipital headache, dizziness, vertigo, tinnitus, dimmed vision or syncope. Moreover the dysfunction of brainstem regulatory mechanism of blood pressure in essential hypertension interacting with sensory neuronal tracts might cause sensory deficits.

The sensory derangement could be associated either with the effect or the cause i.e., the primary disorder of sympathetic system responsible for development of essential hypertension.Diffuse cerebrovascular abnormalities have been found in hypertensive patients despite their having no neurological complications. Any behavioral effects of hypertension may be mediated by structural changes in the brain e.g. disseminated microaneurysms or lacunar infarcts. But is also concievable that such effects could be due to non-structural changes in brain function such as decreased cerebral blood flow. White matter lesions in brain have been associated with hypertension. Few studies have shown correlation between metabolic disorders and abnormalities in brain stem auditory evoked potentials due to defects in process of myelination in CNS.Subclinical changes in CNS function may not be easily detected with traditional methods of medical assessment.Today the availability of more sensitive and non-invasive method like the Visual evoked potential(VEP), Brainstem auditory evoked potentials(BAEP) and Cognitive evoked potential(P3/ERP) to evaluate subtle alterations in CNS functions allow us to better correlate blood pressure with possible early brain alterations.

VEP, BAEP have proven to be very useful markers in detecting subclinical abnormalities involving the visual and auditory pathways.Similarly P3/ERP have also been very useful in detecting brain atrophy (grey and white matter changes). These methods have been used to study changes in hypertension and whether they have any correlation with clinical parameters is yet to be thoroughly assessed. The BAEP comprises several well defined early (less than 10 msec) and late (upto 200 msec) components evoked from the brainstem and central auditory pathways respectively. Cognitive Evoked Potentials,the P300 (P3) wave is an event related potential (ERP) component elicited in the process of decision making. It is considered to be an endogenous potential, as its occurrence links not to the physical attributes of a stimulus, but to a person's reaction to it.

II. METHODOLOGY

STUDY AREA: The study was conducted in department of physiology, saraswathi institute of medical sciences, Hapur **Study design:** The study was an open randomized comparative study.
Study period: The study was conducted from March 2017 to NOVEMBER 2017.A thorough clinical examination was done to exclude any other pathological disorder beside hypertension.

STUDY POPULATION: The present study was carried out in Department of Physiology, Saraswathi Institute of Medical Sciences, Anwarpur, Hapur. A total number of 120 patients are selected and grouped into, group 1 - 30 controls (normotensives), group 2 – 30 prehypertensives, group 3 – 30 hypertensives for duration less than 5 years, group 4 – 30 Hypertensives for duration more than 5 years. The following parameters was taken for the study:Examination – Measurement of blood pressure,Visual evoked potential,Brainstem auditory evoked potentials,Cognitive evoked potential

STATICAL ANALYSIS: The one way ANOVA was used to statistically analyze and compare the various proportions which were derived in the different groups. Pearson's correlation was used for correlations. A P-value which was >0.05 was considered as non-significant and <0.05 as significant and <0.001 was considered to be highly significant. SPSS version - 20 was used for all statistical analysis.

In right eye we found a significant difference in Hypertensives less than 5 years(97.57±5.41) P=0.014 as compared to Normotensives (90.99±8.67) and P<0.001 as compared to Hypertensives more than 5 years(114.45±8.68), Also we found significant increase in P100 latency in Hypertensives for more than 5 years P<0.001 as compared to Normotensives, Prehypertensives and Hypertensives for less than 5 years in both eyes as in

TABLE 1 Comparison of VEP among Normotensives, Prehypertensives, Hypertensives less than 5 years and Hypertensives more than 5 years

Mean ± S.D						
VEP	N	Pre HT	HT<5	HT>5	ANOVA (F value)	Post HOC (Bonferroni)
Left eye	90.92±6.69	94.12±9.55	98.93±4.18	117.24±9.96	F = 65.626 P <0.001	N vs PreHT P = ns N vs HT<5 P <0.001 N vs HT>5 P <0.001 PreHT vs HT<5 P= ns PreHT vs HT>5 P<0.001 HT<5 vs HT>5 P<0.001
right eye	90.99±8.67	93.99±9.34	97.57±5.41	114.45±8.68	F = 49.360 P <0.001	N vs PreHT P = ns N vs HT<5 P = 0.014 N vs HT>5 P<0.001 PreHT vs HT<5 P=ns PreHT vs HT>5 P<0.001 HT<5 vs HT>5 P<0.001

*N – Normotensives, PreHT – Prehypertensives, HT<5 – Hypertensives for less than 5 years.
HT>5 – Hypertensives for more than 5 years.
ns – not significant, (P<0.05 – significant)

TABLE 2 Comparison of BAEP In left ear

Left ear	N	PreHT	HT<5	HT>5	ANOVA (F value)	Post Hoc (Bonferroni)
Wave I	1.50±0.12	1.48±0.20	1.67±0.23	1.73±0.22	F = 11.51 P <0.001	N vs PreHT P=ns N vs HT<5 P = 0.008 N vs HT>5 P<0.001 PreHT vs HT<5 P = 0.002 PreHT vs HT>5 P<0.001 HT<5 vs HT>5 P = ns,
Wave II	2.47±0.10	2.66±0.20	2.68±0.37	2.72±0.21	F = 4.108 P = 0.008	N vs PreHT P = ns N vs HT<5 P = ns N vs HT>5 P = 0.009 PreHT vs HT<5 P = 0.002 PreHT vs HT>5 P<0.001 HT<5 vs HT>5 P = ns,
Wave III	3.56±0.23	3.48±0.40	3.53±0.35	3.65±0.21	F = 1.464 P = ns	N vs PreHT P = ns N vs HT<5 P = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
Wave IV	4.45±0.24	4.47±0.36	4.70±0.32	4.78±0.19	F = 7.454 P <0.001	N vs PreHT P = 0.003 N vs HT<5 P = 0.008 N vs HT>5 P<0.001 PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
Wave V	5.36±0.30	5.35±0.50	5.44±0.46	5.57±0.23	F = 2.041 P = ns	N vs Pre HT P = ns N vs HT<5 P = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns

						HT<5 vs HT>5 P = ns
I-III	2.06±0.23	2.01±0.49	1.85±0.40	1.92±0.32	F = 1.772 P = ns	N vs Pre HT P = ns N vs HT<5 P = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
I-V	1.78±0.22	1.85±0.61	1.91±0.47	1.91±0.32	F = 0.637 P = ns	N vs PreHT P = ns N vs HT<5 P = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
III-V	3.82±0.34	3.86±0.41	3.77±0.3	3.78±0.45	F = 0.323 P = ns	N vs PreHT P=ns N vs HT<5 P = ns N vs HT>5 P = ns

TABLE 3 Comparison of BAEP in Right Ear

Right ear	N	PreHT	HT<5	HT>5	ANOVA (F value)	Post HOC (Bonferroni)
Wave I	1.43±0.10	1.52±0.16	1.65±0.22	1.70±0.24	F = 12.182 P <0.001	N vs PreHT P = ns N vs HT<5 P<0.001 N vs HT>5 P<0.001 PreHT vs HT<5 P = ns PreHT vs HT>5 P = 0.02 HT<5 vs HT>5 P = ns
Wave II	2.5±0.13	2.55±0.30	2.62±0.27	2.71±0.22	F = 4.107 P = 0.009	N vs PreHT P = ns N vs HT<5 P = ns N vs HT>5 P = 0.008 PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
Wave III	3.55±0.26	3.53±0.40	3.56±0.35	3.60±0.21	F = 0.292 P = ns	N vs PreHT P = ns N vs HT<5 P = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
Wave IV	4.63±0.19	4.65±0.28	4.66±0.20	4.78±0.20	F = 2.573 P = ns	N vs PreHT P = ns N vs HT<5 p = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
Wave V	5.32±0.17	5.52±0.41	5.45±0.38	5.59±0.36	F = 3.134 P = 0.02	N vs PreHT P = ns N vs HT<5 p = ns N vs HT>5 P = 0.02 PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
I-III	2.10±0.28	2.14±0.24	2.0±0.37	1.87±0.32	F = 4.226 P = 0.007	N vs PreHT P = ns N vs HT<5 p = ns N vs HT>5 P = 0.02 PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns

TABLE 4 Comparison of ERP(P300) in Normotensives, Prehypertensives, Hypertensives less than 5 years and Hypertensives more than 5 years

ERP (P300)	Mean ± S.D				ANOVA (F Value)	Post HOC (Bonferroni)
Left	N	PreHT	HT<5	HT>5	F = 0.860 P = ns	N vs PreHT P = ns N vs HT<5 P =ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns
	295.03 ± 23.75	303.07 ± 23.04	302.87 ± 33.82	295.12 ± 25.62		
Right	321.53 ± 22.23	315.78 ± 25.70	304.94 ± 29.69	307.98 ± 28.80	F = 2.372 P = ns	N vs PreHT P = ns N vs HT<5 P = ns N vs HT>5 P = ns PreHT vs HT<5 P = ns PreHT vs HT>5 P = ns HT<5 vs HT>5 P = ns

*N – normotensives, PreHT – prehypertensives, HT<5 – hypertensives less than 5 years
HT>5 – hypertensives more than 5 years.
ns – not significant, P<0.05 – significant

III. DISCUSSION

This study was undertaken to evaluate the VEP, BAEP and ERP as potential markers of early nervous system damage and also to correlate the changes if any, between blood pressure and VEP, BAEP and ERP in patients with prehypertension and hypertension.

In VEP, we found that both groups of hypertension (hypertensives for duration less than and more than 5 years) showed a statistically significant prolonged P100 latency. they found prolonged P100 latency in 26% cases out of twenty three subjects. However, there was no significant difference found in amplitude of P100 wave in present study . There was no significant difference in P100 latency in present study group of prehypertensives as compared to normotensives and hypertensives for less than 5 years duration, though there was significant difference (P< 0.001) as compared to hypertensives for more than 5 years duration. Hence, It is highly probable that the changes are due to duration of the hypertension and this need a further longitudinal study. Increase in the production of free radicals such as superoxide anions and subsequent degradation of nitric oxide and lipid peroxidation in various tissues has been the focus of major research to explain some of these unanswered questions. Brain, due to its very high rate of oxygen consumption and the non-regenerative nature of neurons, is particularly susceptible to efforts of oxidative stress. Retinal tissue due to large quantity of polyunsaturated fatty acids and rich supply of oxygen are also likely to be highly sensitive to ill effects of oxidative stress.

The findings of the current study, thus suggests that hypertension does affect the neuronal excitation/conduction in the visual and auditory pathways, and it may or may not affect the cognitive function. The absence of significant changes between all the waves in all the groups of patients of the study could be due to small number of subjects in all groups and this needs larger studies for further interpretation. As to what is the exact cause of this derangements still a matter of debate.

Due to paucity of studies conducted in this aspect, a definite causecannot be attributed to these changes. However, in view of the consistent changes seen in our and many more studies, we can conclude that hypertension does causes damage to the optic and auditory pathways, which seems to be related with the duration of disease. It can be further concluded that perhaps the first changes in the auditory pathway may be attributed at level of lateral lemniscus in even in prehypertension as we found increased latency of wave IV in BAEP in prehypertensives.

IV. CONCLUSION

Statistically significant delay in p100 suggests that the development of hypertensive neuroopathy sub clinically, occurs in very early stages of Hypertension, which is not detectable on routine clinical examination. VEP can be suggested for screening in high-risk individuals to evaluate the functional integrity of visual pathway in hypertension and as a key to unravel the mystery of hidden Hypertensive Morbidity and Mortality. There was no statistical significance found in amplitude of VEP, this discrepancy in result may be due to the

fewer number of subjects in our study. There are changes in the BAEP in patients with prehypertension and significant increase in latency can be seen in wave IV in left ear concluding that the impairment in the auditory pathway may start from the level of lateral leminiscus. However in right ear there was no statistical significance found in BAEP in patients with prehypertension, but significant changes found in patients with hypertension explains that the impairment in the auditory pathway may start in either ear and progresses to the other ear with the progression of hypertension. In hypertension the results are statistically significant in absolute wave latencies of waves I, II, &IV in left ear and waves I, II, & V and in IPLs I-III & I-V in right ear suggesting bilateral abnormality in peripheral portion of the auditory nerve and progresses to cochlear nucleus, however there was unilateral abnormality in lateral lemniscus (pons) in left ear and in inferior colliculus (midbrain) in right ear.In ERP (P300) we did not found any significant change in between any of the groupin our study this may be due to the fewer number of subjects in our study and cognition function may be impaired in longer duration of hypertension.The findings of the current study, thus suggests that hypertension does affect the neuronal excitation/conduction in the visual and auditory pathways and it is related to the duration of hypertension. The central neuropathy involving auditory pathway may be impaired earlier than visual pathway as seen in prehypertension. BAEP is appeared to be more sensitive and early indicator of these changes than VEP and ERP.Therefore, it is concluded that early diagnosis and treatment of High Blood Pressure through lifestyle modifications in prehypertension can prevent neurological complications as Hypertension causes central neuropathy even in earlier duration

REFERENCES

[1]. Chobanian AV, Bakris GL, Black HR, et al. (May 2003). "The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report". JAMA. **289** (19): 2560–72. doi:10.1001/jama.289.19.2560. PMID 12748199

[2]. American Heart Association website

[3]. Smetana, GW (2011). "Rethinking "Abnormal" Blood Pressure: What is the Value?". Journal of General Internal Medicine. **26** (7): 678–680. doi:10.1007/s11606-011-1737-2. PMC 3138596. PMID 21557032

[4]. Taylor, BC; Wilt, TJ; Welch, HG (2011). "Impact of diastolic and systolic blood pressure on mortality: Implications for the definition of "normal"". Journal of general internal medicine. **26** (7): 685–90. doi:10.1007/s11606-011-1660-6. PMC 3138604. PMID 21404131.

[5]. Port, S; Demer, L; Jennrich, R; Walter, D; Garfinkel, A (2000). "Systolic blood pressure and mortality". Lancet. **355** (9199): 175–80. doi:10.1016/S0140-6736(99)07051-8. PMID 10675116.

[6]. http://content.onlinejacc.org/article.aspx?articleid=1140179

[7]. National Heart, Lung and Blood Institute<http://www.nhlbi.nih.gov/hbp/hbp/whathbp.htm>

[8]. Qureshi AI, Suri MF, Kirmani JF, Divani AA, Mohammad Y (September 2005). "Is prehypertension a risk factor for cardiovascular diseases?". Stroke. **36** (9): 1859–63. doi:10.1161/01.STR.0000177495.45580.f1. PMID 16081866

[9]. 101

[10]. Vasan RS, Larson MG, Leip EP, et al. (November 2001). "Impact of high-normal blood pressure on the risk of cardiovascular disease". N. Engl. J. Med. **345** (18): 1291–7. doi:10.1056/NEJMoa003417. PMID 11794147.

[11]. American Heart Association (2005, August 6). Prehypertension Triples Heart Attack Risk

[12]. James, PA.; Oparil, S.; Carter, BL.; Cushman, WC.; Dennison-Himmelfarb, C.; Handler, J.; Lackland, DT.; Lefevre, ML.; et al. (Dec 2013). "2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8)". JAMA 311 (5): 507–20. doi:10.1001/jama.2013.284427. PMID 24352797.

[13]. Lewington, S; Clarke, R; Qizilbash, N; Peto, R; Collins, R; Prospective Studies, Collaboration (Dec 14, 2002). "Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies.". Lancet 360 (9349): 1903–13. doi:10.1016/s0140-6736(02)11911-8. PMID 12493255.

[14]. Marik PE, Varon J; Varon (June 2007). "Hypertensive crises: challenges and management". Chest 131 (6): 1949–62. doi:10.1378/chest.06-2490. PMID 17565029.

[15]. 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; 1: 1116-31.

[16]. Fredric MW. Cerebrovascular disease. In: Conn HL, Horwitz O, editors. Cardiac and vascular diseases. Philadelphia: Lea &Febiger, 1971; 2: 1473-99.

[17]. Tandon OP, Ram D, Awasthi R. Brainstem auditory evoked responses in primary hypertension. Indian J Med Res. 1996 Nov; 104: 311-5.

[18]. Smoog I, Lernfelt B, Landahl S, PalmertzB, Andreasson LA, Nilsson L, Persson G, Oden A, Svanborg A. 15 year longitudinal study of Blood pressure and Dementia. The Lancet 1996:347:1141-1145
[19]. McGhee TB, Cerebrovascular disease and neurological manifestations of heart disease. In: Hurst JW editor-in-chief. Heart arteries and veins 5th ed. New York: McGraw Hill Inc. 1982; 1986-98
[20]. Pavfilov VV. Reid JL. Brian and autonomic mechanisms in hypertension. J Hypertens 1994; 12: 337-343
[21]. Boller et al. Neurological correlates of hypertension. Arch Neurol: 1977:34:701-705

Source of Support: Nil, **Conflict of Interest:** None declared.

Corresponding Author:** **Dr. Manisha Shrivastwa
Department of Physiology, Saraswathi institute of Medical Sciences, Hapur Uttar Pradesh India