National Journal of Physiology, Pharmacy and Pharmacology

RESEARCH ARTICLE

A comparative study on visual evoked potential in normotensive and hypertensive individuals

Arunkumar Balakrishnan[1], Nirmala Natarajan[2]

[1]Department of Physiology, Saveetha Medical College & Hospital, Chennai, Tamil Nadu, India, [2]Department of Physiology, Sri Venkateshwara Medical College and Research Institute, Pondicherry, India

Correspondence to: Arunkumar Balakrishnan, E-mail: ak30051987.ak@gmail.com

Received: June 11, 2018, **Accepted:** July 23, 2018

ABSTRACT

Background: Hypertensive retinopathy is one of the most common complications of hypertension. Hypertension can cause changes in vascular endothelium including hyalinization, which can lead to demyelination of the optic nerve and results in conduction abnormalities in the visual pathway. Visual evoked potential (VEP), a non-invasive neurophysiological technique, is useful in detecting the changes in functional integrity in the visual pathway. The present study was done to compare the changes in the VEP between normotensive and hypertensive individuals. **Aims and Objectives:** This study aims to compare the VEP changes among normotensive and hypertensive individuals. **Materials and Methods:** The study was conducted in 30 normotensive and 30 hypertensive subjects (BP ≥140/90 mmHg, according to JNC-7 classification) with normal visual acuity. VEP was recorded using the pattern reversal stimulation technique. The peak latencies of the waves N75, P100, and N145 were measured. **Results:** There was a statistically significant prolonged N75 ($P < 0.05$), P100 ($P < 0.05$), and N145 ($P < 0.05$) latencies in hypertensive individuals when compared to normotensives. **Conclusion:** VEP changes occur in hypertensive patients before the development of hypertensive retinopathy. Thus, VEP can be used as a routine screening test for hypertensive individuals, and it can also be used as a better prognostic tool during the treatment of hypertensive retinopathy.

KEY WORDS: Hypertension; Hypertensive Retinopathy; Visual Evoked Potential

INTRODUCTION

Hypertension is said to be a "silent killer" as it has no initial symptoms, but it can lead to long-term life-threatening disease and complications. The cause for hypertension is multifactorial, and the common risk factors include obesity, lack of exercise, and excess salt intake. Hypertension is estimated to cause 7.5 million deaths worldwide, which is about 12.8% of the total deaths. Hypertension is a major risk factor for coronary heart disease as well as for stroke. In addition to these, complications of hypertension also include heart failure, peripheral vascular disease, renal impairment, retinal hemorrhage, and visual impairment.[1] Hypertensive retinopathy is one of the most common ocular manifestations of hypertension. It is suggested that hypertension causes vascular endothelial changes including hyalinization, which can lead to demyelination of the sensory tracts which are involved in carrying the visual sensation to the higher centers which might result in conduction abnormalities.[2] Hypertension leads to ischemia which can cause demyelination of the optic nerve or damage of the retinal ganglion cell before the development of hypertensive retinopathy. Hypertensive retinopathy progresses from a mild non-proliferative stage to a severe proliferative stage

Access this article online	
Website: www.njppp.com	Quick Response code
DOI: 10.5455/njppp.2018.8.0620723072018	

and eventually can lead to retinal detachment and blindness. Thus, it is better to diagnose this condition at an earlier stage and treat it appropriately. Visual evoked potential (VEP), a simple, sensitive, and non-invasive neurophysiological technique, represents the resultant response of cortical as well as subcortical areas to photostimulation. VEP is electrical potential differences recorded from the vertex in response to visual stimuli. VEP is helpful in detecting any change in functional integrity in the visual pathway.[3,4] Altered responses in VEP may indicate that there is some defect in the visual pathway and also in assisting a clinical diagnosis of demyelinating diseases, optic neuropathy, and cortical blindness.[5] There are very few studies that have been done on evaluating the visual pathway abnormalities in hypertension. Thus, the present study was done to assess VEP changes in hypertension.

Aims and Objectives

This study aims to compare the VEP changes among normotensive and hypertensive individuals.

MATERIALS AND METHODS

The study was conducted in 30 normotensive and 30 hypertensive individuals. After obtaining the Institutional Ethical Committee clearance, subjects were recruited from Medicine OPD of Sri Venkateshwara Medical College and Research Centre. The normotensive subjects were recruited from college and hospital after measuring the blood pressure. The subjects were informed about the purpose of the study and written informed consent was obtained. Blood pressure was recorded with standard mercury sphygmomanometer in sitting posture after 5 min of rest. The criterion for diagnosing hypertension was BP ≥140/90 mmHg based on the average of three consecutive readings at an interval of 3 weeks. Subjects in the age group of 25–55 years of both the sexes with BP values of 100–119/60–79 mmHg were recruited as normotensives (Group I) and BP values of ≥140/90 mmHg were recruited as hypertensives (Group II), according to JNC-7 classification.[6] All the subjects were screened for complete eye checkup including visual acuity and ophthalmoscopy to rule out any eye pathology. Those individuals with any eye pathology including retinopathy, diabetics, smokers, and alcoholics were excluded from the study. Height of the subject was measured with stadiometer, to the nearest of 0.1 cm. Weight was measured with weighing scale, to the nearest of 0.5 kg. VEP was recorded in Physiology Research Laboratory, in the morning hours at a pleasant temperature of around 20–25°C using the PHYSIOPAC-PP4, MEDICAID SYSTEM, CHANDIGARH. Each subject was seated at a distance of 1 m from the pattern generator screen in dark air-conditioned room and was asked to look at the central spot on screen with one eye, other being patched. The scalp electrodes were placed according to the 10–20 international system of electrode placement. The active electrode was placed at O_z, which is the highest point on the occiput. The reference electrode was placed at F_{pz}, which is 12 cm above the inion. The VEPs were picked up as the difference between active electrode (O_z) and the reference electrode (F_{pz}). The ground electrode was fixed at wrist. The shift pattern test stimulus on the TV monitor was white and black checks (15 mm × 15 mm size). Electrode impedance was kept below 5 KΩ, with automatic artifact rejection[5]. The recording was done in each eye separately, till the end of 2000 waves.

Table 1: Comparison of wave N75 latency between normotensives and hypertensives

Wave N75 latency	Normotensive	Hypertensive	*P* value
Right (ms)	69.83±4.89	89.55±8.68	0.0001*
Left (ms)	72.41±7.05	91.19±8.22	0.0001*
Mean (ms)	71.12±5.17	90.37±6.41	0.0001*

*P<0.05 - significant. Data expressed as mean±SD

The commonly seen waveforms in VEP are N75, P100, and N145. The waves N75, P100, and N145 are a result of electrical stimulation of the area 17, 19, and 18 of the occipital cortex, respectively.[5,7] The latencies of all these waves were analyzed. The data were analyzed using SPSS version 17. Data were expressed as mean ± SD. Student's unpaired *t*-test was used to compare the values between hypertensive and normotensive groups. The correlation between BP and mean P100 latency was assessed using Pearson correlation coefficient.

RESULTS

- Normotensive group: There were 30 normotensives with an average age of 34 ± 9.14. They had average weight 60.9 ± 11.63 kg, height 154.5 ± 9.98 cm, systolic BP 110.4 ± 4.76 mmHg, and diastolic BP 71.26 ± 4.56 mmHg.
- Hypertensive group: There were 30 hypertensive patients with an average age of 42 ± 10.29. They had average weight 68.79 ± 12.03 kg, height 164.25 ± 7.91 cm, systolic BP 147.2 ± 5.79 mmHg, and diastolic BP 94.8 ± 3.22 mmHg.

The values of all the wave latencies of both the eyes in hypertensive group were compared with the normotensive group. The correlation was assessed between mean P100 latency and BP in normotensives and hypertensives separately. There were a statistically significant prolonged latencies of waves N75, P100, and N145 ($P < 0.05$) in hypertensive patients when compared to normotensives[Tables 1-3].

A significant positive correlation (R^2=0.76; $P < 0.05$) between mean P100 latency and systolic BP was seen in hypertensive group [Figure 1].

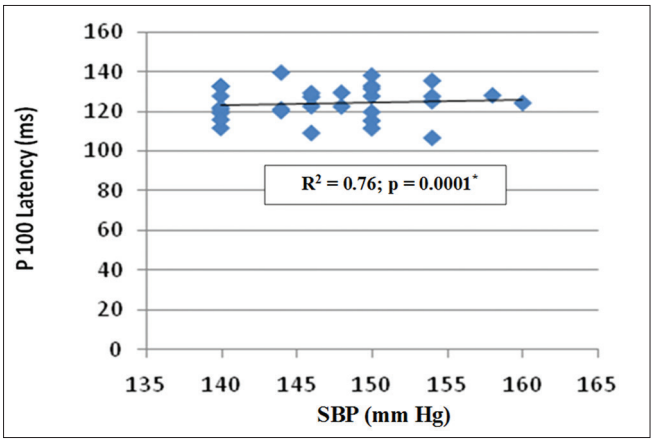

Figure 1: Correlation between mean P100 latency and SBP in hypertensives

Table 2: Comparison of wave P100 latency between normotensives and hypertensives

Wave N75 latency	Normotensive	Hypertensive	*P* value
Right (ms)	102.01±3.66	123.30±8.74	0.0001*
Left (ms)	103.69±3.42	124.98±10.01	0.0001*
Mean (ms)	102.85±3.06	124.15±8.41	0.0001*

*P<0.05 - significant. Data expressed as mean±SD

Table 3: Comparison of wave N145 latency between normotensives and hypertensives

Wave N75 latency	Normotensive	Hypertensive	*P* value
Right (ms)	148.95±14.94	162.23±12.96	0.0005*
Left (ms)	153.42±14.70	164.49±8.29	0.0007*
Mean (ms)	151.19±14.07	163.36±14.53	0.0017*

*P<0.05 - significant. Data expressed as mean±SD

DISCUSSION

In the present study, the latencies of all the waves were prolonged in hypertensives, which indicate that there was conduction delay in the visual pathway due to hypertension. A significant positive correlation of mean P100 latency with systolic BP was seen in hypertensive group. No such correlation was seen in normotensive group. This indicates that the severity of conduction delay increases with increase in SBP.

In the study conducted by Tandon *et al*., P100 latency was prolonged in hypertensives and suggested that hypertensive milieu affects neuronal excitation/conduction in the visual pathway.[2] In the study conducted by Marsh *et al*., P100 latency of visual evoked response was delayed in pre-eclamptic women.[8] There was a delayed conduction in brainstem auditory evoked response in Grade III hypertension.[9] There was impairment in sensory conduction in human beings in hypertension.[10] P100 latency was prolonged in patients with demyelinating disease like multiple sclerosis.[11] There is a significant change in VEP with normal fluctuation in carotid pressure and heart rate.[12] These findings were similar to the results of the present study. Thus, our results suggest that there is some demyelination occurring in the sensory pathways during hypertension. The pathophysiology of central nervous system dysfunction in hypertension might be due to the arterial and arteriolar spasms in the blood vessels of the brain, which in combination with fibrinoid degeneration of the small arteries can lead to microinfarction in the grey nuclei and white matter.[9] Moreover, there is baroreceptor resetting mechanism occurring in hypertension that might interact with the sensory neurons and causes sensory deficits.[2]

Limitations

1. Sample size was less
2. Degree of demyelination could not be assessed.

CONCLUSION

VEP changes occur in hypertensive patients before the development of hypertensive retinopathy. Hence, VEP measurements can be used as a routine screening for hypertensive individuals to diagnose hypertensive retinopathy at an earlier stage and also for a better prognosis during treatment.

REFERENCES

1. WHO/Raised Blood Pressure. Avilable from: http://www.who.int/gho/ncd/risk_factors/blood_pressure_prevalence. [Last accessed on 2018 May 20].
2. Tandon OP, Ram D. Visual evoked potential in primary hypertension. Indian J Physiol Pharmacol 1997;41:154-8.
3. Bhanu R, Shankar V, Kutty K, Annamalai N. Visual evoked potentials in non-insulin dependent diabetes mellitus without retinopathy. Curr Neurobiol 2012;3:55-9.
4. Chopra D, Gupta M, Manchanda KC, Sharma RS, Sidhu RS. A study of visual evoked potentials in patients of Type-2 diabetes mellitus. J Clin Diag Res 2011;5:519-22.
5. Pal GK, Pal P. Textbook of Practical Physiology. 3rd ed., Vol. 43. Hyderabad, India: Universities Press Private Limited; 2010. p. 303-6.
6. Chobanian AV, Bakrish GL, Black HR, Cushman WC, Green LA, Izzo JL, *et al*. Seventh report of the joint national committee on prevention, detection, evaluation and treatment of high blood pressure. Hypertension 2003;42:1206-125.
7. Vinodha R, Priya CS. Visual transmission in iron deficiency anemia. Int J Med Sci Public Health 2016;5:2256-8.
8. Marsh MS, Smith S. The visual evoked potential in the assessment of central nervous effects of preeclampsia - A pilot study. Br J Obstet Gynaecol 1994;101:343-6.
9. Tandon OP, Ram D, Awasthi R. Brainstem auditory evoked responses in primary hypertension. Indian J Med Res 1996;104:310-4.
10. Sethi A, Vaney N, Tandon OP. Sensory nerve conduction

during cold pressor response in humans. Indian J Med Res 1994;99:279-82.
11. Movasat M, Piri N, Nili AM. Visual evoked potential study in Multiple Sclerosis disease. Iran J Ophthalmol 2009;21:37-44.
12. Walker BB, Sandman AC. Visual evoked potentials change as heart rate and carotid pressure change. Psychophysiology 1982;19:520-7.

How to cite this article: Balakrishnan A, Natarajan N. A comparative study on visual evoked potential in normotensive and hypertensive individuals. Natl J Physiol Pharm Pharmacol 2018;8(10):1437-1440.

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