Indian J Physiol Pharmacol 1997; 41(1): 79–82

EVENT RELATED EVOKED POTENTIALS IN HYPERTENSIVE SUBJECTS

O. P. TANDON* AND M. K. JOON

Department of Physiology,
University College of Medical Sciences & GTB Hospital,
Dilshad Garden, Delhi - 110 095

(Received on September 5, 1996)

Abstract: Cognitive functions were assessed in hypertensive and sex and age matched healthy controls using auditory event related evoked potential 'odd ball' paradigm. The N2 and P3 latencies in the hypertensive subjects were significantly delayed as compared to controls. There was also correlation of blood pressure (BP) with N2 latency in hypertensive group. These findings suggest that cortical neurophysiological events, depicting information processing and memory are modulated by rise in BP and cognition is delayed in hypertensive cases.

Key words : event related evoked potential hypertension
P_3 potential cognition

INTRODUCTION

Diffuse cerebrovascular abnormalities on postmortem have been found in hypertensive patients despite their having no neurological complications (1). Inspite of such frequent cerebral damage, neurological symptoms and signs seem to be rare and mild in hypertensive patients (2). Higher brain functions including attributes of behaviour have not been well documented in hypertensive cases, though behavioral factors have been implicated in the pathophysiology of hypertension. These psychosomatic or behavioural factors may induce personality profiles making individual vulnerable to stressful influences and cause permanent enhancement of the CNS autonomic drive (3). Hence the hypertensive individuals often report slightly decreased power of concentration, light headedness and easy fatigue causing impairment in intellectual performance (4). These symptoms might correlate with diffuse and mild cerebrovascular abnormalities, but might also Be explained by neurophysiological cortical depression, clearly shown in experimental models (5) or by emotional reactions to illness. As such, effect of raised blood pressure on cognitive function is a controversial issue. Some reports advocate adverse effect on memory, logical reasoning and attention (6). While others deny this and also the association of BP with cognitive performance (7). In the present study we wish to report an electrophysiological evidence of delay in cognitive function in patients suffering from essential hypertension.

METHODS

Twenty four middle aged (twelve normal healthy controls, 12 hypertensive patients) were the subjects of the study. Newly diagnosed patients from OPD of GTB Hospital having a diastolic BP above 90 and systolic > 140 mm Hg on three consecutive visits, belonging to mild to moderate grades of hypertension were selected for this study. They were thoroughly investigated and labelled as cases of essential hypertension. Before putting them on any therapy, they were tested for cognitive evoked potential responses. Age and sex matched normal controls were picked

*Corresponding Author

up from the paramedical staff of the UCMS & GTB Hospital. Auditory event related evoked potential responses were studied in these subjects using standard 'odd ball' paradigm. The details of the methods used are given in our earlier studies (8–9). The process basically involves recording of P_3 potential from the CZ–PZ scalp regions during a task in which subject concentrates on rare high pitched click sound and presses button when he hears them in a train of low pitched frequent and high pitched infrequent click sounds. Thirty two such responses were averaged during each trial. Unpaired student 't' test was done to find out significant changes in latency and amplitude of P_3 in hypertensive subjects and compared to controls. Analysis of variance (ANOVA) was also done to compare various parameters (wt, ht, age) and BP (systolic and diastolic) with N_2, P_3 latencies and P_3 amplitude of the event related evoked potentials and correlation coefficients worked out.

RESULTS

The subjects were middle aged and in each group there were 8 males and 4 females. The latencies of N_2, P_3 and amp. of P_3 along with BP and physical parameters are shown in Table I. N_2 and P_3 latencies are significantly increased in hypertensive subjects. The other positive finding is the correlation between N_2 latency and diastolic BP in hypertensives (Table II).

TABLE I : Composite data giving Mean ± SD values of various physical and B.P. parameters and P_3 event related evoked responses in controls and hypertensives.

	Control	*Hypertensive*	*P-value*	*Significance*
Age (yrs)	49.83 ± 11.0	48.75 ± 7.0	0.777	NS
Height (cms)	158.5 ± 7.7	162.33 ± 4.3	0.152	NS
Weight (kg)	64.08 ± 5.3	58.55 ± 7.0	0.165	NS
Systolic BP (mmHg)	129.17 ± 4.1	155.50 ± 20.5	0.001	S
Diastolic BP (mmHg)	77.7 ± 8.0	102.83 ± 8.1	0.000	S
N_2 (msec)	207.45 ± 45.5	256.83 ± 23.9	0.004	S
P_3 (msec)	325.42 ± 17.4	360.0 ± 34.1	0.005	S
P_3 amp (μV)	17.62 ± 2.2	12.47 ± 3.5	0.356	NS

TABLE II : Showing values of correlation coefficients between different parameters in controls and hypertensives.

	Control			*Hypertensive*		
	N_2	P_3	P_3 amp	N_2	P_3	P_3 amp
Wt.	0.154	0.3181	0.914	−0.2415	0.2064	0.914
Ht.	−0.3602	−0.515	−0.2057	−0.1512	0.1478	−0.2057
BPS	0.2754	0.1656	−0.4464	0.1178	−0.4959	−0.4464
BPD	0.514	0.3569	0.1358	−0.6398*	−0.2108	0.1358
N_2	1.0000	−0.0235	−0.3395	1.0000	0.0688	−0.3395
P_3	−0.0235	1.0000	0.1261	0.0688	1.0000	0.2370
P_3(amp)	−0.3395	−0.1261	1.0000	−0.3745	0.2877	1.0000

*Significant 0.05

DISCUSSION

The values of N_2, P_3 latencies in our control group were 207.4 ± 45.5 and 325.4 ± 17.4 (m sec), which are higher than the ones reported in our earlier study in young healthy adults (8). This may be due to the fact that these subjects were middle aged (49.8 ± 11.0 yrs) and age related increase in latency of P_3 has been well documented (10). There was no significant difference in N_2 and P_3 latencies in male and females. Similar observation indicating that P_3 remains stable within individuals and is unaffected by sex has been reported (11).

The significant increase in N_2 and P_3 latency in the hypertensive subjects is an important finding (Table I). This would suggest that raised BP, somehow interacts with the generators of these potentials in the brain. The location of these generators is debated. Most of the reports say that they lie in hippocampus with cholinergic mediation and are concerned with memory (13-14), The P_3 latency and to a lesser extent preceding N_2 component is systematically related to the cognitive status of the patient as revealed by psychometry (14). Both N_2 and P_3 latencies covary and N_2 latency was found to correlate with the P3 latency (15). Hence increased latency of both N_2 and P_3 components in the hypertensive group, would reflect impaired cognition. The initial N_2 component of the event related potential might represent sensory information processing and perceptive function and the P_3, the actual memory updating process.

Reports suggesting changes in vascular responses of blood vessels in the brain to the raised blood pressure, delaying sensory conduction are available (17). Hence something similar might be happening to blood vessels of

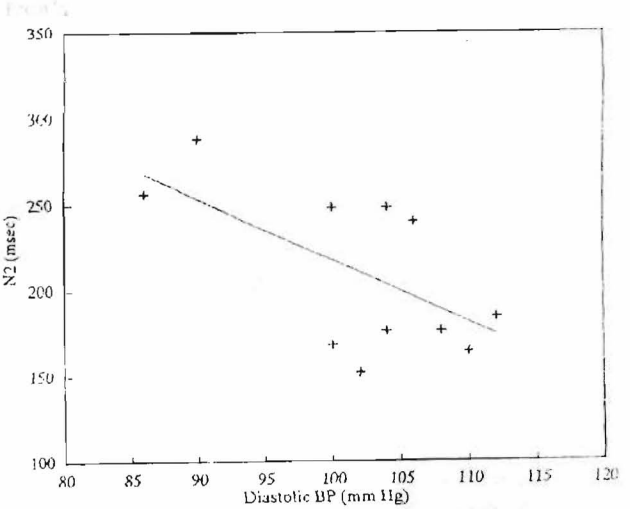

Fig. 1 Scatter diagram and regression line showing correlation between N_2 latency and diastolic B.P. in hypertensive cases.

(Corr coeff. –0.6398, Reg Eq N_2 = 576.1769 – 3.59, P value 0.0251)

the cortical brain areas where generators of event related evoked potentials are located, causing delay in sensory information processing (N_2 latency) and memory updating (P_3 latency). Exact mechanism of this effect on cognition in hypertensive patients needs further systematic studies. The negative correlation between diastolic BP and N_2 latency (Table II and Fig. 1) is yet another important finding, at present, it is difficult to explain this correlation. Since N_2 represents sensory information processing and it has correlation with BP in hypertensive subjects, it implies that brain generators for N_2 component of event related evoked potentials, modulate its own sensory inputs/transmission for cognitive functions. Already numerous studies have established this fact including studies from our laboratory (18-20). The exact interactions between vasomotor mechanisms controlling BP and the higher cortical areas concerned with cognitive functions needs to be explored.

REFERENCES

1. Fisher CM. Cerebral miliary aneurisms in hypertension. *Am J Pathol* 1972; 66: 313.
2. Ziegler DK. Hypertensive vascular disease in the brain. In Hand book of clinical neurology Vinken PJ, Bruyn GW (Ed) Elsevier Publishing Company New York 1972; 12 : 552.
3. Julius S, Petrin J. Autonomic nervous and behavioral factors in hypertension. In Hypertension: Pathophysiology, Diagnosis and management. Laragh OH and Brenner BM (Eds) Raven Press Ltd. New York 1990: PP 2083–2090.
4. Sokolow M. Hypertensive cerebrovascular disease, In Current medical Diagnosis and treatment Krupp M.A., Chatton MJ (Eds) Lange Medical Publication Altos California 1978; P 185.
5. Dworkin BR, Filewich RJ, Miller NE, Craigmyle N, Pickering TG. Baroreceptor activation reduces reactivity to noxious stimulations : Implication for hypertension. *Science* 1979; 205 : 1299.
6. Franceschi M, Tancredi O, Smirne S, Mercinelli A, Canal N. Cognitive process in hypertension. *Hypertension* 1982; 4: 226–229.
7. Former ME, White LR, Abbott RD, Kittner SJ, Kaplan E, Wolz MM, Brody JA, Wolf PA. Blood pressure and cognitive performance. The Framingham study. *Am J Epid* 1981; 28 : 1103–1114.
8. Tandon OP. P_3 event related evoked potential in young adults. *Indian J Physiol Pharmacol* 1990; 34 :191–194.
9. Tandon OP, Kumar S. Event related cerebral evoked potential in chronic pain patients. *Indian J Physiol Pharmacol* 1993; 37: 51-55.
10. Goodin DS. Event related (Endogenous) potentials, In Electrodiagnosis in Clinical Medicine, Aminoff AJ (Ed) Third Edition. Churchil Livingstone - Edinburgh 1992. pp 627–648.
11. Sklare DA, Lynn GE. Latency of P_3 event–related potential : Normative aspects and within subject variability. *Electroencephalogr Clin Neurophysiol* 1984; 59: 420–440.
12. Okada YC, Kaufman L, Williamson SJ. The hippocampal formation as a source of slow endogenous potential. *Electroencephalogr Clin Neurophysiol* 1983; 55: 417–426.
13. Meador KJ, Hammond EJ, Loring DW, Allen M, Bowers D, Heilman KM. Cognitive evoked potentials and disorders of recent memory. *Neurology* 1987; 37: 526–528.
14. Homberg V, Vegter H, Granseyer G, Strauss S, Lange W, Hennerici M. Event related potentials in patients with Huntington's disease and relatives at risk in relation to detailed psychometry. *Electroencephalogr Clin Neurophysiol* 1986; 63: 552–569.
15. Michalewski HJ, Rosenberg C, Starr A. Event related potentials in dementia, In Evoked potential, Cracco RQ Bodis Wollner I (Eds). Lin New York 1986;3:521-528.
16. Solliway BM, Schaffer A, Pratt H, Yannai S. A multidisciplinary study of lead exposed subjects I. Delayed target delection P 300 latency: an electrophysiological parameter, correlates with urinary δ-ALA. *Environ Res* 1994;67:168-182.
17. Marsh MS, Smith S. The visual evoked potentials in the assessment of central nervous effects of pre-eclampsia - a pilot study. *Brit J Obstet Gynae* 1994; 101 : 343-346.
18. Walker BB, Sandman CA. Visual evoked potentials change as heart rate and carotid sinus pressure change. *Psychophysiology* 1982; 19 : 520-572.
19. Sethi A, Vaney N, Tandon OP. Sensory nerve conduction during cold pressor response in human. *Indian J Med Res* 1994; 99 : 279-282.
20. Vaney N, Tandon OP. Brainstem auditory evoked responses during cold pressor test. *Indian J Physiol Pharmacol* 1994; 38 : 145-147.