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Research Article

Comparative study of visual evoked potentials in diabetic vs non-diabetic individuals

Nikhat Yasmeen[*1], Mohammed Muslaiuddin Khalid[2], Abdul Raoof Omer Siddique[1], Sanghamitra Panda[1] and Madhuri Taranikanti[1]

[1]*Department of Physiology, Shadan Institute of Medical Sciences, Hyderabad, Andhrapradesh, India*
[2]*Department of Emergency Medicine, Care Hospital, Hyderabad, Andhra Pradesh, India*

***Correspondence Info:**
Dr. Nikhat Yasmeen,
Assistant Professor,
Department of Physiology,
Shadan Institute of Medical Sciences, Hyderabad -500 008
E-mail: yasmeen78692@yahoo.com

Abstract

Background: Diabetes is a metabolic syndrome which affects most of the organs of the body including the central nervous system. Diabetic retinopathy is one of the complications resulting from derangement of neurovascular coupling & altered cell signaling pathway. Visually evoked potentials have evolved as a sensitive tool for detecting the neuropathic changes even before the clinically evident disease.
Objectives: To compare the latencies & amplitudes of visually evoked potentials i.e. N75, P100 & N145 in diabetic & normal individuals.
Materials & methods: 20 Diabetic individuals with type II diabetes mellitus in the age group of 45-60 yrs were included in the study group .Control group consisted of 10 age & sex matched non-diabetic individuals. Diabetic individuals with documented fundoscopy changes were included in the study and diabetic individuals with type I diabetes mellitus were excluded from the study. Visually evoked potentials were recorded using Viking select neurodiagnostic system.
Conclusions: P100 Latency is significantly prolonged in diabetic individuals when compared to control group suggesting that the processing of information by the visual cortex is slower in diabetics due to central neuropathy changes.
Keywords: Diabetic retinopathy, metabolic syndrome

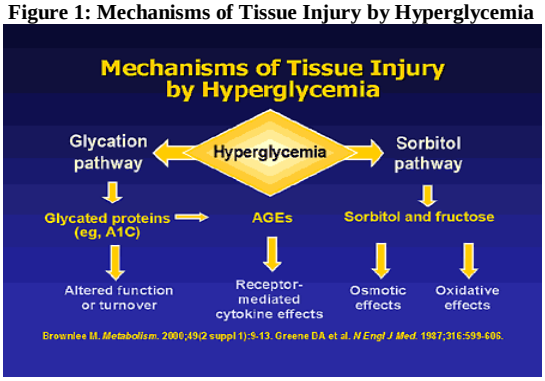
1. Introduction

Diabetes type II is typically multifactorial disease characterized by hyperglycemia due to genetic factors causing insufficient production of insulin from the beta cells along with insulin resistance; environmental factors like obesity, overeating, sedentary lifestyle & stress.

Diabetic retinopathy is considered to be neurovascular disease resulting from chronic hyperglycemia, oxidative stress & protein glycation. Neural & vascular tissues are closely dependent on each other such that hemodynamic abnormalities, hypoperfusion & ischemia resulting from capillary basement membrane thickening & endothelial hyperplasia results in structural & functional derangement in neuronal function.[1,2,3,4]

Glucose & its metabolites can form advanced glycation end product's by directly combining with proteins which can contribute to oxidative stress along with increased production of free radicals, protein kinase C, glycoxidation & lipoxidation products.[5]

Figure 1: Mechanisms of Tissue Injury by Hyperglycemia



In a study conducted in 30 patients with newly diagnosed insulin dependent diabetes mellitus it was found that the p100 latency was significantly reduced in patients with elevated HbA1c levels when compared to the control group.[6]

Evoked potentials are a convenient and non-invasive tool for the evaluation of central nervous system. VEPs are also helpful in determining subclinical lesions in the optic nerve, spinal cord and the brain stem; therefore, it is a convenient tool in the diagnosis & follow–up of neurologic disorder.[7,8]

According to Hikmet *et al* observed prolonged latencies suggestive of central neuropathy in DM type II and most of the electrophysiological parameters in patients with DM type II correlated with the duration of the disease, some of them with the age of the patient.[9]

In another study visual evoked potentials were recorded in 30 young insulin –dependent diabetic with HbA1c of 9.4%.latencies & amplitudes of visually evoked potentials were compared with 30 age & sex matched control group. Results of the study found that P100 latency was significantly prolonged ($p<0.01$) & there was normalization of all the observed parameters at the second evaluation after 6 months[10]

In another comparative study done on 40 diabetic patients including 20 subjects with non-proliferative diabetic retinopathy (NPDR) and 20 without any retinopathy P100 latency found to be longer duration (P<0.001) when compared with 40 age & sex matched control group. In diabetic individuals there was significant reduction in amplitudes of N75 & P100 potentials .The study has emphasized that the increase in PVEP latency signifies retinol ganglion cell damage & visually evoked potential can be used as a diagnostic tool for detecting retinopathy changes even before clinically evident disease.[11]

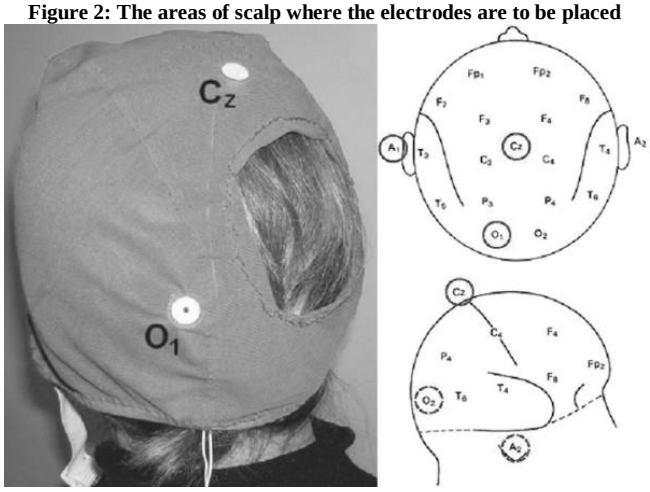
2. Material & Methods

20 Diabetic individuals with type II diabetes mellitus in the age group of 45-60 yrs were included in the study group. Control group consisted of 10 (age & sex matched) non-diabetic individuals. Diabetic individuals with documented fundoscopy changes were included in the study and diabetic individuals with type I diabetes mellitus were excluded from the study. Visually evoked potentials were recorded using Viking select neurodiagnostic system. Consent was taken from all the participants, ethical guidelines were followed & the subjects were informed about the aims & objectives of the study.

2.1 Procedure

In this study, 1 channel recording having 2 electrodes is used. Electrodes are placed according to 10-20 system (EEG).Surface EEG electrodes are used for recording the potentials. Electrode placement & recording were done according to Chiappa.[12] The active electrode is placed at Oz position and reference electrode is placed at Cz position of 10-20 system using 2 anatomical landmarks; nasion & inion. Ground electrode is placed at Fpz position.

Figure 2: The areas of scalp where the electrodes are to be placed



Preparation for the test-

1. The patient is instructed to shampoo the hair before coming for the test.
2. Pattern stimuli for VEPs should be presented when the pupils of the eyes are unaltered by mydriatic or miotic drugs.
3. The patient must get eyeglasses used for reading. For pattern stimulation, the visual acuity of the patient should be recorded and the patient must be optimally refracted for the viewing distance of the screen.
4. The areas of scalp where the electrodes are to be placed are prepared with NUPREP skin preparation gel.

2.2 Recording technique

The patient is made to sit comfortably at a distance of 100 cm from a TV monitor which displays the checkerboard pattern.The preferred stimulus for clinical investigation of the visual pathways is a shift (reversal) of a checkerboard pattern (usually black and white.) He is asked to fix his vision at a point in the center of the pattern field and view it with a single eye. (Monocular testing)

Data-sheet of latencies & amplitudes in diabetic & non-diabetic individuals was made using Microsoft word & excel sheets. Statistical analysis was done using software PASW 18.0 (SPSS Inc. Chicago, USA)

3. Results

This Comparative study consisted of 20 diabetic individuals (Group A) and 20 non-diabetic normal individuals (Group B).

Table I: Analysis of Mean Pattern of Latencies of visually evoked potentials between the groups. (Group A vs Group B)

Mean Pattern of latency	Eye	Group A (Mean ± S.D)	Group B (Mean ± S.D)	P-Value
N75	Right	68.15 ± 5.07	65.83± 7.56	0.325
	Left	70.27±8.85	67.15±5.32	0.316
P100	Right	100.34± 4.32	96.32±3.73	0.018
	Left	101.45± 3.46	97.65±2.73	0.0053*
N145	Right	140.59± 7.93	136.78 ±11.64	0.298
	Left	138.7± 10.99	132.98±6.78	0.144

Table II: Analysis of Mean Pattern of Amplitudes of visually evoked potentials between the groups. (Group A vs Group B)

Mean Pattern of Amplitude	Eye stimulation	Group A (Mean ± S.D)	Group B (Mean ± S.D)	P-Value
N75-P100	Right	5.14 ± 0.86	6.20± 1.97	0.117
	Left	4.35±2.31	6.59±4.86	0.09
P100 –N145	Right	8.58±1.89	7.85±4.37	0.52
	Left	7.89± 3.23	7.25±4.63	0.64

4. Discussion

In the comparative study, VEP Parameters – N75, P100 & N145 latencies and amplitude in diabetics were compared with that of age & sex - matched normal control group.

P100 latency was found to be significantly prolonged in diabetics indicating that there is a decrease in efficiency of processing information by the visual cortex which is due to the pathological changes occurring in conduction pathway in diabetic individuals.

Moreover the results were highly significant for the left side when compared to right side emphasizing on the dominance of right visual cortex in processing of information on repetitive visual stimulation.

This is in accordance to previous studies conducted by Hikmet *et al* & Heravian *et al* signifying the increase in P100 latency which relates to the central neuropathy changes even before the clinically evident disease.

References

1. Cameron NE, Eaton SE, Tesfaye **S.** Vascular factors and metabolic interactions in the pathogenesis of diabetic neuropathy. *Diabetologia.* 2001; 44:1973-88.
2. Gianni C, Dyck PJ. Basement membrane thickening and pericyte degeneration precede development of diabetic polyneuropathy and are associated with its severity. *Ann Neurol.* 1995; 37:498-504.
3. Timperley WR, Ward JD, Preston FE *et al*. Clinical and histological studies in diabetic neuropathy. *Diabetologia.* 1977; 12: 237-43.
4. Sheetz MJ, King GL. Molecular under-standing of hyperglycemia's adverse effects for diabetic complications. *JAMA.* 2002; 288:2579-88.
5. Baynes JW, Thorpe SR**.** Role of oxidative stress in diabetic complications: a new perspective on an old paradigm. *Diabetes.* 1999; 48:1-9.
6. Parapid B, Petronic I, Cirovic D, Nikolic D. Matanovic D, Popovic S. Influence of the metabolic control on latency values of visual evoked potentials (VEP) in patients with diabetes mellitus type 1, *Arch Ital Biol*. 2012 Dec;150(4):251-8. doi: 10.4449/aib.v150i4.1400.
7. Algan M, Ziegler O, Gehin P, Got I, Raspiller A, Weber M, Genton P, Saudax E, Drouin P. Visual evoked potentials in diabetic patients. *Diabetes Care* 1989; 12: 227 9.
8. Puvanendran K, Devathasan G, Wong PK. Visual evoked responses in diabetes. *J. Neurol Neurosurg Psychiatry* 1983; 46: 643-7.
9. Hikmet Dolu, Umit Hidir Ulas, Erol Bolu, Abdullah Ozkardes, Zeki Odabasi, Metin Ozata and Okay Vural**.** Evaluation of central neuropathy in type II diabetes mellitus by multimodal evoked potentials. *Acta Neurologica belgica* 2003; 103/4 (4).
10. Alberto Verrotti, Lucio Lobefalo, Daniela Trotta, Giuseppe Della Loggia, Francesco Chiarelli, Colangelo Luigi, Guido Morgese and Pierenrico Gallenga. Visual evoked potentials in young persons with newly diagnosed diabetes: a long-term follow up**.** *Developmental Medicine & Child Neurology* 2000; 42 (04): 240 – 244.
11. Heravian J, Ehyaei A, Shoeibi N, Azimi A, Ostadi-Moghaddam H, Yekta AA, Khoshsima MJ, Esmaily H. Pattern Visual Evoked Potentials in Patients with Type II Diabetes Mellitus *J Ophthalmic Vis Res*. 2012; 7(3): 225–230.
12. Chiappa K.H. Evoked Potentials in Clinical Medicine. 2nd ed. Raven Press, New York 1990,