

VISUAL EVOKED POTENTIAL IN NON-INSULIN DEPENDENT DIABETES MELLITUS PATIENTS WITHOUT DIABETIC RETINOPATHY

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Purpose:

Diabetes mellitus has multiple complications involving many organs like eyes, brain, heart and kidneys etc. In visual complications retinopathies and neuropathies are most important. Visual evoked potentials (VEPs) is used to find subclinical involvement of visual pathways in type 2 diabetics without retinopathy and comparison of visual evoked potentials (VEP) with non diabetic control subjects.

Material and Methods:

This study was conducted over a period of 6 months from 1st January 2016 to 30 June 2016 in ophthalmology department of Islam medical college teaching hospital Sialkot. Both male and female diabetic patients with good glycemic control and control subjects of 40-60 years of age were included. Neurostar modelec 92B equipment was used to record visual evoked potentials (VEPs) by pattern reversal stimulations and their glucose level was estimated by glucose oxidase method.

Results:

VEP recordings of diabetic patients showed that P_{100} latency was highly significantly prolonged ($P < 0.01$) as compared to control subjects. The prolonged P_{100} latency is indicative of sub clinical involvement of visual pathway as compared to control subjects. In this study the correlation between P_{100} latency of visual evoked potentials (VEPs) and duration of disease was highly significant and subclinical involvement of visual pathways was present in 30%.

Conclusion:

It is an objective sensitive and non-invasive technique to detect subclinical involvement of visual pathways which is important in early detection of diabetic complications concerned with vision. Prolongation of visual evoked potentials (VEPs) particular P_{100} latency showed subclinical involvement of visual pathway in diabetic patients.

Key Words: Diabetic Retinopathy, Visual Evoked Potential, Non-Insulin Dependent, Diabetes Mellitus

INTRODUCTION:

Diabetes mellitus is a set of metabolic disorder peculiarized by hyperglycemia eventualizing from faults in functioning of insulin, its secretion and actions on tissues. The lingering hyperglycemia of diabetes is related with malfunctioning, long-term impairment, and collapse of variant organs, notably the eyes, nerves, heart, blood vessels and kidneys¹. Visual deficit in diabetics occur due to vascular and metabolic aberrations². it is well known that in diabetics peripheral and autonomic neuropathies develop as well as it

affects central nervous system with degenerations³. VEPs (visual evoked potentials) are visually evoked electrophysiological signals elicited from the electroencephalographic animation in the visual cortex recorded from the overlying scalp. Visual cortex is stimulated predominantly by the central visual field, VEPs

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bank on functional rectitude of central vision at any level of the visual pathway including the retina, the optic nerve, optic radiations, and visual occipital cortex⁴. In diabetic patients a decline in nerve conduction velocity might be noticed with normal ocular examination⁵. In diabetics Visual evoked potential (VEP) is efficacious in descrying retinal dysfunction with normal visual acuity². VEP exhibits a resultant response of cortical as well as subcortical areas to photostimulation and it gamble on the functional virtue of the central vision at any level of the visual pathway¹. The present study was conducted with an aim of establishing the role of VEP in the assessment of retinal ganglion cell damage, which is a sign of diabetic neuropathy and to detect sub clinical involvement of visual pathways in patients of non insulin depenent and to compare visual evoked potentials (VEPs) of diabetic (type II) patients with non diabetic control subjects.

MATERIALS AND METHODS:

This study was conducted in ophthalmology department of Islam medical college teaching hospital Sialkot. Forty subjects both males and females between 40-60 years of age were included by simple random sampling technique. In group 1 twenty non diabetics(control subjects) 15 males and 5 females of mean age 45.95 ± 5.82 and in group 2 twenty non insulin dependent with duration of diabetes more than six months without retinopathy 13 males and 7 females mean age of 55.10 ± 6.03 were included. persons presented with visual acuity less than 6/9 and other ocular abnormalities like tumors, glaucoma retinopathies, ocular discharge, cataract etc and other systemic diseases were excluded. Written consent with history Performa's was taken from selected subjects. General physical and clinical examination of each subject was done. Ocular examination included visual acuity by snellen chart, colour vision, visual fields, intraocular pressure, fundoscopy, visual evoked potentials were recorded. Neurostar modelec 92-B equipment was used for the recordings of visual evoked potentials. Silver silver chloride (Ag-AgCl) electrodes were applied at following sites

Active electrode OZ was applied 5cm above inion

Reference electrode FZ was placed in frontal portion of midline

Ground electrode FPZ was placed on forehead in the middle

The subject was instructed to focus constantly on the centre of the screen at the distance of 100cm. Pattern reversal stimulations were recorded monocularly. Approximately 128 responses were averaged, latencies $N_{15,p_{100}}$ and N_{145} and their amplitudes were recorded on print paper.

RESULTS:

Arithmetic mean and standard deviation (SD) of each variable in the control and diabetic groups were calculated. For latencies and amplitudes of visual evoked potentials (VEPs) the average of values of two eyes was used for statistical analysis⁶. Student (t) test was applied and difference of results was considered significant when $P < 0.05$, highly significant when $P > 0.01$, 0.001 and non-significant when $P > 0.05$ ⁷.

Mean \pm SD ages of non-diabetic controls and non-insulin dependent diabetics were 45.95 ± 5.82 years and 55.10 ± 6.03 years respectively. Age range in both groups was 40-58 years and 42-60 years respectively. Mean \pm SD random values of blood glucose in non diabetic controls and type II diabetic were 112.3 ± 15.45 mg/dl and 186 ± 57.06 mg/dl respectively. Statistically the difference between these values was highly significant $P < 0.001$. Mean \pm SD duration of disease in type II diabetes was 5.55 ± 4.08 years (range 1-15 years).

Table 1 shows latencies and amplitudes of VEPs in type II diabetics and non-diabetic controls.

- i. Mean \pm SD N_{75} latency (79.08 ± 6.95 msec) of type II diabetics was non-significant ($P > 0.05$) as compare to values (76.26 ± 2.44 msec) in non-diabetic controls.
- ii. The P_{100} amplitude in type II diabetics were also non-significantly ($P > 0.05$) different from non-diabetic control values ($5.91 \pm 4.55 \mu v$, $6.2 \pm 3.82 \mu v$) respectively.

- iii. Mean \pm SD latency of P₁₀₀ (105.04 ± 8.54 m sec) in type II diabetics was highly significantly ($p < 0.001$) prolonged than this latency (99.27 ± 3.46 msec) in non-diabetic controls.
- iv. Mean \pm SD latency of N₁₄₅ (136 ± 13.63 msec) in diabetics was significantly ($P < 0.05$) prolonged as compared to this latency (130.12 ± 6.8 msec) in non-diabetic controls.

Table 1: Comparison of Latencies and amplitudes of visual evoked potentials (VEPs) between type II diabetics and non-diabetic controls.

VEPs parameters	Latencies and amplitudes of VEPs (Mean \pm SD)		
	Non-diabetic controls n=20	Type II diabetics n=20	P value
P ₁₀₀ Latency (m sec)	99.27 ± 3.46	105.04 ± 8.54	$P < 0.01$ (HS)
N ₇₅ Latency (m sec)	76.26 ± 2.44	70.08 ± 6.95	$P > 0.05$ (NS)
P ₁₀₀ Amplitude (μ v)	6.2 ± 3.82	5.91 ± 4.55	$P > 0.05$ (NS)
N ₁₄₅ Latency (m sec)	130.12 ± 6.8	136.0 ± 13.63	$P < 0.05$ (S)

HS = Highly Significant

S = Significant

NS = Non significant

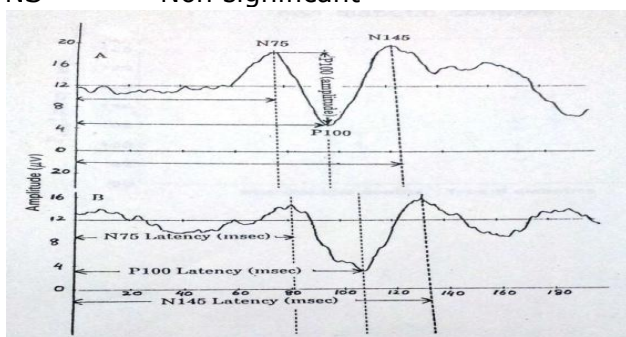


Fig.1: Latencies of N₇₅, P₁₀₀ and N₁₄₅ were prolonged in diabetic subjects as compare to the non-diabetic control. Amplitude of the P100 was reduced in diabetic than non-diabetic control.

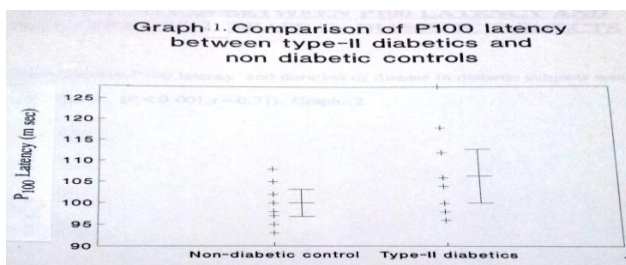


Fig. 2: Correlation between p 100 latency and duration of disease in diabetic subjects was highly significant. ($P < 0.001$, $r = 0.71$).

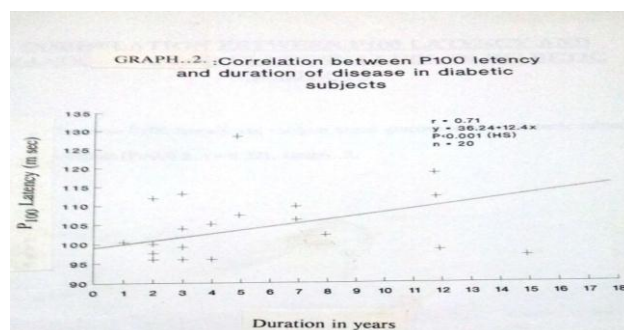
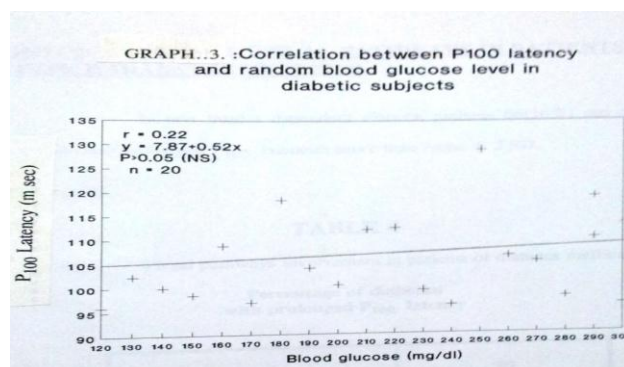
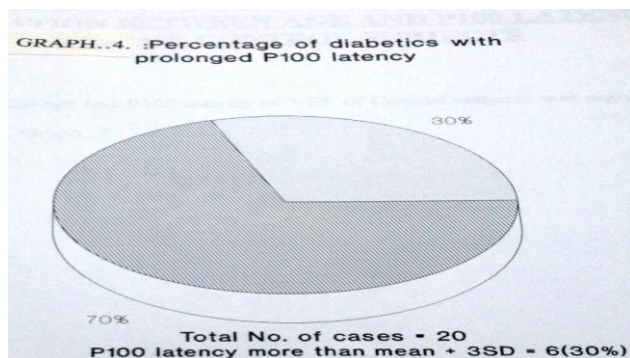


Fig. 3: Correlation between P₁₀₀ latency and random blood glucose level in diabetic subjects was non-significant ($P > 0.05$, $r = 0.22$).



In non-insulin dependent diabetic patients 06(30%) out of 20 patients had prolonged P₁₀₀ latencies more than mean + 3 SD.



P₁₀₀ latency more than mean + 3 SD was considered prolonged.

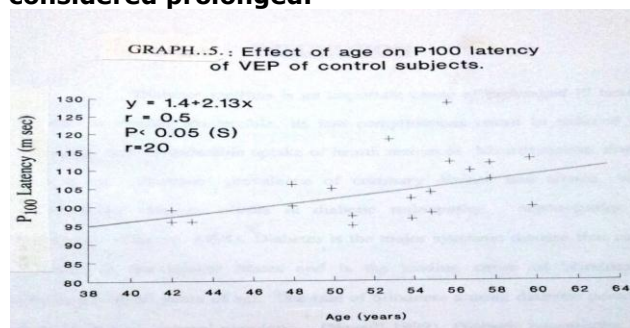


Fig. 6: Coorelation between age and P100 latency of control subjects was significant ($p < 0.05$, $r = 0.5$).

DISCUSSION:

Diabetes mellitus is an important cause of prolonged ill health. Diabetes is usually irreversible, its late complication result in reduced life expectancy and considerable uptake of health resources. Macro vascular disease leads to an increased prevalence of coronary disease and stroke while micro vascular damage results in diabetic retinopathy, nephropathy and neuropathy⁸. the rate of blindness among diabetic persons is twenty times that of general population⁹. diabetic neuropathy is the most common complication which may be either clinically evident or suclinical¹⁰. In 1983 puvanendran et al reported optic nerve involvement by prolonged latencies of visual evoked potentials (VEPs) in patients of diabetes mellitus with no retinopathy or ocular disease and having an almost normal visual acuity. Later on, the work of Martenelli

and his co workers and pozzessers and his coworkers is the main landmark in this field.

In our study the p₁₀₀ latency of VEPs was highly significantly ($P < 0.01$) prolonged in type II diabetics as compare to non diabetic controls the prolonged P₁₀₀ latency is indicative of subclinical involvement of visual pathway in diabetic patients while other visual functions were normal.

Similar results of prolonged P₁₀₀ latency have been reported^{11,12,13,14,6,15,16,17,18}.

A non significant increase of P₁₀₀ latency in patients of diabetes mellitus was reported^{19,20}. This might be due to varied group of patients studied by different methods. Latencies of N₁₄₅ were significantly ($P > 0.05$) different than in non diabetic controls.

These parameters of VEPs, particularly P₁₀₀ amplitude and N₇₅ latency are highly variable and less reproducible. Our results correlate with the results of^{21,22,16,20,18}.

In our study the correlation between the P 100 latency of VEPs and duration of the disease was highly significant. in our study the correlation between the P₁₀₀ latency and random blood glucose levels was non-significant ($P > 0.05$) in diabetic subjects.

In our subclinical involvement of visual pathways was 30% in diabetic patients without retinopathy. our results correlate with results reported by various workers 30%¹¹, 48%²³, 22.2%²⁴ and 42.8%²².

The effect of age on VEPs latencies is due to reduction in retinal luminance due to small pupil on neuronal cell loss in visual pathways as a result of aging process. In our study coorelation between age and P 100 latency of VEPs of control subjects was significant ($P < 0.05$). similar effect of age on VEPs latency have been reported^{25,26,27}. Our study has confirmed the previous reports by various workers regarding the clinical utility of visual evoked potentials changes in detection of visual pathway involvement at subclinical changes in patients of diabetic mellitus. This sensitive objective and non-invasive test

enable physicians to commence proper treatment to prevent further progression of this subclinical involvement of visual pathways by achieving stricted glycemic control.

CONCLUSION:

This study of visual evoked potentials (VEPs) in type II diabetic patients without retinopathy reveals that:

1. The subclinical involvements of visual pathways occur in patients of type II diabetes mellitus without retinopathy.
2. Prolongation of VEPs latencies occur with advancing age, particularly P₁₀₀ latency.
3. Recording of VEPs is an objective, sensitive and non-invasive technique to detect subclinical involvement of visual pathways.

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2	Saba Anwar	Data collection analysis, writing of introduction & results
3	Erum Ehsan	Tables, Graphs, Statistical result, Bibliography