



## Evaluation of auditory neuropathy in type-2 diabetes mellitus using brainstem auditory evoked potentials

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### Abstract:

Diabetes is a major cause of morbidity and among its complications neuropathies are the most common. Central diabetic neuropathy may lead to delayed evoked potentials in the central pathways. To evaluate the auditory neuropathy in diabetes mellitus, this study was undertaken in the Department of Physiology and Department of Medicine at Govt. Medical College and Hospital, Amritsar, Punjab during 2008-2009. For this, Brainstem auditory evoked potentials were recorded in patients of type 2 diabetes mellitus divided into three groups based upon the duration of disease. The parameters recorded were absolute latencies of waves I, III and V & Inter-peak latencies (IPL) I-III and I-V. These were compared with age and sex matched controls. The results showed no statistically significant changes in any of the above parameters. This could be attributed to fairly good glycemic control of the patients included in the study. Thus, we conclude that patients with good glycemic control develop no auditory diabetic neuropathy irrespective of the duration of disease. However more studies are required to substantiate these results.

**Keywords:** Absolute Latencies, BAEP, IPL, Type-2 Diabetes Mellitus

### Introduction

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. The chronic hyper-glycemia of diabetes is associated with long term damage, dysfunction and failure of various organs especially the eyes, kidneys, nerves, heart and blood vessels [1].

Diabetes may alter both the peripheral and central nerve functions but the peripheral manifestations of diabetic neuropathy are more frequently discussed in the literature than the impairment of Central Nervous System. Delay of evoked potentials in the central pathways has been reported in diabetic patients but the exact pathophysiology of these alterations is not clear [2].

Central diabetic neuropathy is a newer concept and it can be detected by simple and non invasive methods. One of these methods is Brainstem Auditory Evoked Potentials (BAEPs). By this method, functional and autonomic pathologies from the acoustic nerve to

the upper part of brain stem can be demonstrated at an early stage. Lesions in these levels result in changes in BAEP amplitudes and latencies [3].

The ABR (Auditory Brainstem Response) test is a measure of neural synchrony of the auditory nerve through the auditory brainstem structures [4]. Evoked potentials are electric signals from the central nervous system triggered in response to the stimulation of a receptor. These tests are characterized by very high sensitivity, although not specificity, they are non-invasive and have no side effects. A nerve tract damage increases the latency and reduces the amplitude of the response. Five waves are distinguished in the auditory brainstem response. Waves are corresponded to the conduction of potential in the particular structure in the central nervous system: the auditory nerve (wave I), cochlear nucleus (wave II), Superior olivary nucleus (wave III), Lateral lemniscus (wave IV) and the inferior colliculus (wave V). Conduction times between these structures are also evaluated (inter peak latencies): I-III inter peak latency (conduction time from the 8th cranial

nerve to the brainstem nuclei) and III-V inter peak latency (conduction time from the inferior part of the brainstem to the mesencephalon). Evoked potentials offer the possibility to perform a functional evaluation of neural pathways in the central nervous system. They are extremely useful in clinical practice, as it is possible to ascribe changes in the wave latencies to specific anatomic structures in the central nervous system[5].

## Material And Methods

Brainstem Auditory Evoked Potentials (BAEPs) were recorded in four groups of 30 subjects each after obtaining ethical approval from institutional ethical committee. The groups were divided as:

Group I: 30 controls-Age and sex matched non diabetics from general population

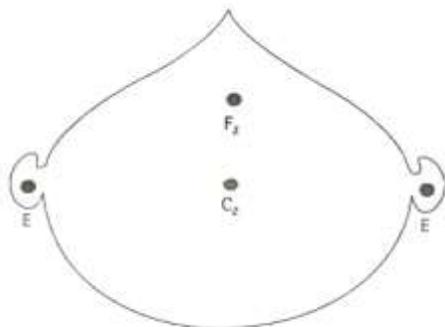
Group II: 30 diabetic patients with duration of the disease < 5 years

Group III: 30 diabetic patients with duration of the disease 5-10 years

Group IV: 30 diabetic patients with duration of the disease > 10 years

Fasting blood sugar levels of all the subjects in all the four groups were recorded and mean fasting blood sugar level of each group was calculated. All the cases of diabetes were taken from the OPD of Medicine department of Guru Nanak Dev Hospital, Amritsar .Patients who had no subjective symptoms of hearing loss and had normal hearing test(Weber's and Rinne's) were selected for the study. BAEPs were conducted after getting a written and informed consent from the patients.

BAEPs were performed on an outpatient basis. BAEP were tested in the department of Physiology, Government Medical College, Amritsar using RMS EMG EP Mark II version: 7.5.7, 2 channel (PC based) machine.



Electrode placement for BAEP. E—ear lobe recording, C<sub>1</sub>—reference, and F<sub>1</sub>—ground electrodes.

The hearing threshold of the patients was done and the stimulus was delivered at 60 dB higher than the hearing threshold. The stimulus used was a brief click which was a square wave pulse of 0.1 milli second duration. The clicks were delivered through earphones. A click rate of 11.1 Hz was used. 2000 clicks were delivered to both the ears and the average of them was accepted as BAEP response. The stimuli were picked by the surface electrodes and a recording in the form of waves I-V was obtained on the screen on being averaged, filtered and amplified by the machine. The machine has an in built muscle artifact rejecting facility.

The following parameters were measured for the analysis of BAEP:

- i) Absolute latency of waves I to V
- ii) Interpeak Latency of I-III and I-V.

The results obtained were analyzed by one way Anova test to study the mean, standard deviation and standard error. Also, advanced Post Hoc Tukey HSD test was applied to study the difference of means of the values obtained and to study the correlation of the data obtained for different study groups.

## Observations and Discussion

The peripheral and autonomic neuropathy occurring in diabetic patients is well known. Whether there is also a specific central nervous system involvement has not been well documented. It is reasonable to ask whether such a ubiquitous metabolic derangement and diffuse angiopathy might involve any part of the nervous system [6].

A team of researchers concluded from pathological material that diabetic neuropathy is a disease of peripheral nerves and that degeneration in the CNS is unimportant. [7]Major textbooks on diabetes either disregard cerebral involvement or minimize its existence[6]. Similar results have been found in the present study. Our findings showed no statistical significant variations in the absolute latencies of waves I,III and V and inter peak latencies I-III and I-V in any of the study groups irrespective of the duration of disease.

Our findings are consistent with a study which also showed no change in any of the above parameters in diabetic patients of variable duration of illness ranging from 6 months to 25 years[6] but do not tally with other researchers[8,9,10,11,12,13,14,15,16].

Table 1 shows a comparative study of recorded parameters (Absolute latencies of waves I, III and V; IPL I-III, I-V and Mean FBS) in all the study groups. Table 2 shows the P value (statistical significance) of the

**Table 1: Comparison of Recorded Parameters in the Study Groups**

Parameters	Mean +/- SD Group I (Controls)	Mean +/- SD Group II (DM < 5 Years)	Mean +/- SD Group III (DM 5-10 Years)	Mean +/- SD Group IV (DM >10 Years)
<b>Wave I (Latency)</b>	1.7970 +/- 0.2065	1.7413 +/- 0.2080	1.7430 +/- 0.2438	1.7267 +/- 0.2460
<b>Wave III (Latency)</b>	3.6610 +/- 0.2347	3.6977 +/- 0.2061	3.6690 +/- 0.2121	3.6523 +/- 0.2429
<b>Wave V (Latency)</b>	5.7557 +/- 0.3628	5.6107 +/- 0.3006	5.7133 +/- 0.2804	5.7667 +/- 0.3560
<b>Inter Peak Latency I-III</b>	1.8640 +/- 0.2885	1.9563 +/- 0.2394	1.9260 +/- 0.3119	1.9257 +/- 0.2811
<b>Inter Peak Latency I-V</b>	3.0530 +/- 0.2721	3.0607 +/- 0.3220	3.0620 +/- 0.3423	3.1437 +/- 0.2891
<b>Fasting Blood Sugar(mg%)</b>	100.37	129.03	138.06	132.8

**Table 2: Comparison of P value of Parameters of BAEP in normal subjects (Group I) and Diabetic Patients (Groups II, III and IV)**

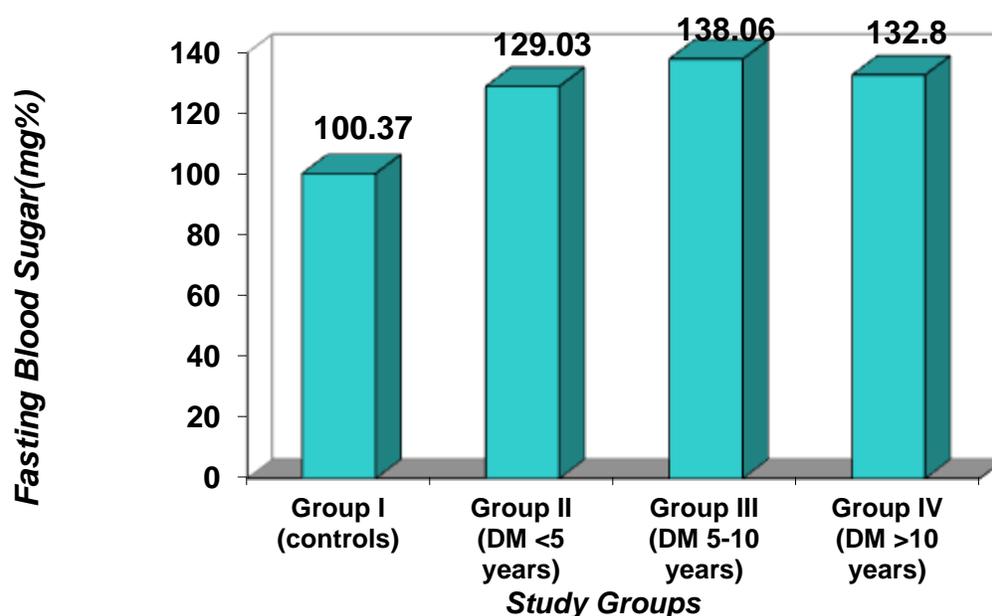
Parameters	P value			Significance
	II Vs I	III Vs I	IV Vs I	
<b>Wave I (Latency)</b>	0.778	0.793	0.628	NS
<b>Wave III (Latency)</b>	0.921	0.999	0.999	NS
<b>Wave V (Latency)</b>	0.319	0.959	0.999	NS
<b>Inter Peak Latency I-III</b>	0.583	0.829	0.831	NS
<b>Inter Peak Latency I-V</b>	1.00	0.999	0.665	NS

recorded parameters of BAEP in controls as compared to diabetic groups. It is seen that there is no statistically significant difference in any of the parameters in the diabetic groups as compared to controls ( $p > 0.05$ ). Table 3 shows inter group comparison of the recorded

parameters amongst the diabetic groups (II, III and IV). It is observed that there are no statistically significant changes in any of the parameters irrespective of duration of disease.

**Table 3: Comparison of P value of Parameters of BAEP in Diabetic Patients (Groups II, III and IV)**

Parameters	P value			Significance
	III Vs II	IV Vs III	IV Vs II	
Wave I (Latency)	1.000	0.992	0.994	NS
Wave III (Latency)	0.960	0.992	0.862	NS
Wave V (Latency)	0.618	0.922	0.256	NS
Inter Peak Latency I-III	0.975	1.000	0.975	NS
Inter Peak Latency I-V	1.000	0.733	0.723	NS

**Figure 1: Bar chart showing mean of fasting blood sugar of the study subjects**

Thus, it is seen that the diabetic patients with disease duration of even more than 10 years do not show a statistically significant change in any of the above parameters.

These statistically insignificant changes can be attributed to:

(i) Better controlled diabetic patients in our study groups (group II mean FBS 129.03 mg %; group III mean FBS 138.06 mg% & group IV mean FBS 132.8mg%)(fig.1).

(ii) The diabetic patients were taken from the OPD of GNDH, Amritsar who were on anti diabetic therapy and hence had better glycaemic control.

(iii) No serious patients with probably poorly controlled blood sugar and/or clinical complications from medical wards were taken in our study.

(iv) The study group comprised of most of the diabetic patients with disease duration of 1-10 years (60 patients) and only one third of the diabetic patients were with disease duration of more than 10 years.

Thus, there is no damage done to the peripheral as well as central neuronal pathways of auditory nervous system even with disease duration of up to 10 years when the fasting blood sugar is within well controlled limits. Thus, it can be concluded that no auditory peripheral or central diabetic neuropathy occurred in

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diabetic patients irrespective of duration of the disease, if the patients had a fairly good glycaemic control. And probably it might take a longer duration of the disease and/or poorly controlled diabetes mellitus to develop peripheral as well as central auditory diabetic neuropathy.

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