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A Study of Auditory Brainstem Evoked Responses in Type 1 and Type 2 Diabetes Mellitus Patients with Normal Hearing

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Abstract

Background: Diabetes mellitus (DM) is a common metabolic disorders with millions of cases word-wide. Its effect on the functioning of central nervous system (CNS) and peripheral nerves is a matter of current neurological research. Our study aimed to find out changes in auditory brainstem responses if any, in patients with Type 1 & Type 2 DM patients with apparently normal hearing. **Subjects and Methods:** 50 cases each of Type 1 and Type 2 diabetic patients with apparently normal hearing were chosen along with 50 healthy controls. Patients were classified according to duration of disease. Pure tone audiometry and brainstem evoked response audiometry was performed in all cases. The BERA results were interpreted for latencies of waves I-V and inter-peak latencies I-III, I-V and III-V. **Results:** Significant delay in absolute latency of wave I, III, IV, V and inter-peak latencies I-V and III-V was seen in Type 1 diabetic patients. In Type 2 diabetic patients, latencies of waves I, II, III, IV and V and inter-peak latencies I-III, I-V and III-V were significantly delayed. Prolonged latencies were not related to type and duration of diabetes. Latencies of waves III and IV were significantly prolonged in type 2 DM patients with fasting blood sugar level >130 mg/dl. **Conclusion:** BERA is non-invasive and easy to perform test that can detect minor CNS changes at early stage of diabetes and can be used to detect peripheral (auditory nerve) and central neuropathy in diabetics even in absence of clinical signs and symptoms of deafness.

Keywords: Diabetes Mellitus, Auditory Brainstem Responses, Central Neuropathy.

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Introduction

Diabetes mellitus (DM) is a condition characterised by poor glycemic control leading to a state of hyperglycaemia. It is one of the most common metabolic disorders affecting the human population with millions of cases world-wide. Depending upon the aetiology of DM, factors contributing to hyperglycaemia include reduced insulin secretion, decreased glucose utilisation and increased glucose production.[1] It is estimated that 20 percent of the current global diabetic population resides in South-East Asian Region. The number of diabetic persons in the countries of this region is likely to triple by the year 2025.[2]

In the early period of DM, diabetic neuropathy can be clinically detected as a result of autonomic and peripheral nerve function impairment. The involvement of the central nervous system (CNS) in diabetic neuropathy is also common.[3] It has been well documented in the literature that long standing cases of Diabetes Mellitus is associated with progressive bilateral high sensorineural hearing loss starting at an earlier age than the normal population.[4]

Brainstem evoked response audiometry (BERA) is a simple and non-invasive procedure to detect the integrity and functioning of the eighth cranial nerve and the central auditory pathway. It can therefore be used to detect early impairment of functioning of the acoustic nerve and central auditory pathways even in the absence of specific signs and symptoms of clinical deafness.[5] Brainstem auditory evoked response is the electrical potentials recorded from the ear and vertex in response to a brief auditory stimulation to assess the conduction through the auditory pathway up to the midbrain.[6] A normal BERA wave has seven components which originate from specific structures of the auditory pathway.[7] The 7 waves are as follows -

Wave 1 – It is the representation from the compound action potential in the distal portion of the vestibulocochlear cranial nerve. The response is believed to originate from afferent activity of the vestibulocochlear cranial nerve fibres as they leave cochlea and enter the internal auditory canal.

Wave 2 – It is generated by the proximal vestibulocochlear nerve as it enters the brainstem.

Wave 3 – It is generated mainly in the cochlear nucleus (second order neuron).

Wave 4 – It arises from pontine third order neuron. Mostly located in superior olivary nucleus, but additional contributions may come from cochlear nucleus and nucleus

of lateral lemniscus.

Wave 5 – Generation of wave 5 reflect activity of multiple anatomic auditory structures. Sharp positive peak of wave 5 arises mainly from the lateral lemniscus, following slow negative wave represents dendritic potential in the inferior colliculus.

Wave 6 & 7 – These waves appear to be generated in the inferior colliculus, perhaps in the medial geniculate body.

Our study was performed to detect if there were any changes in auditory brainstem responses in patients with Type 1 and Type 2 DM patients with apparently normal hearing. In other words we have tried to find out if any there are any specific and predictable changes in the BERA responses in this group of patients before the clinical onset of sensorineural hearing loss.

Aims and Objectives

This study was conducted with the following aims & objectives:

- To find the effect of Type 1 DM and Type 2 DM on the various wave forms of BERA in patients with apparently normal hearing.
- To find whether any correlation exists between the observed abnormalities (if any) with the duration & type of diabetes.
- To compare the results with normal individuals.
- To assess the utility of BERA as screening tool in early detection of diabetic neuropathy in patients with apparently normal hearing.

Subjects and Methods

This prospective study was carried out in the Department of Otorhinolaryngology and Head Neck Surgery in our hospital and included a total of 150 subjects with normal hearing that were divided into three groups:

- Group 1 - 50 Type 1 DM subjects
- Group 2 – 50 Type 2 DM subjects and
- Group 3 - 50 Non-diabetic healthy subjects (controls)

Only proven cases of Type 1 and Type 2 diabetes by history, clinical examination and blood investigations in the age group of 25-50 years were included in the study. Patients with history of any ear disease like COM, past ear surgery, exposure to prolonged loud noise, intake of ototoxic drugs, head trauma, stroke, meningitis or family history of hearing impairment were excluded. Patients taking any medication which might be expected to interfere with the functioning of CNS (e.g. Methyldopa, Reserpine, Phenytoin, antipsychotics, anti-depressants) were also excluded from this study. Any patient with an abnormal pure tone audiometry test was also automatically excluded from the study.

Detailed history was taken and clinical examination was done. Biochemical studies for fasting blood sugar (FBS) and random blood sugar (RBS) was done.

Subjects were first tested by pure tone audiometry (PTA) and then BERA was performed under standard conditions. For PTA we have used ALPS advanced digital audiometer (Model - AD2100) and for BERA we have used the Nicolet EDX advanced electrodiagnostic system.

The BERA results were interpreted for the latencies of waves I, II, III, IV and V and interpeak latencies (IPL) I-III, I-V and III-V. The BERA results of patients with DM were also classified according to the duration of disease (those with DM less than 10 years duration and those with DM for more than or equal to 10 years). Evaluation of the data was carried out by independent student's t-test for unpaired data. 'p' value less than 0.05 and 0.005 were considered significant and highly significant respectively.

Results

In this study, total 150 subjects were included and divided into three groups. Mean age of Type 1 and Type 2 diabetic subjects was 41.7±11.75 years and 48.24±6.23 years respectively in this study. The mean age of controls was 45.54□7.49 years. There was no statistical significant difference between mean age of both diabetic and control groups. However, the mean FBS and RBS levels were much higher in diabetics. There was no significant difference between pure tone average of diabetic subjects and controls in both ears and lies within normal hearing thresholds.

Table 1: Mean and standard deviation (S.D.) of various parameters in group 1 (Type 1 DM), group 2 (type 2 DM) and group 3 (control) subjects

Parameters	Group 3(Controls) (Mean±S.D.)	Group 1 (type1 DM) (Mean±S.D.)	Group 2 (type 2 DM) (Mean±S.D.)
Age (Years)	45.54±7.49	41.7±11.75	48.24±6.23
Weight (kg)	67.38±11.74	67.58±9.08	69.26±11.42
Height (cm)	159.8±9.84	159.84±7.39	160.06±7.8
Haemoglobin (gm%)	11.40±1.45	11.07±2.13	11.25±1.60
FBS (mg/dl)	79.68±6.13	123.32±21.07	131.76±24.33
RBS (mg/dl)	101.88±10.97	153.58±23.60	165.56±23.66
PTA (dB) left ear	16.87±3.41	17.40±3.50	15.90±3.83
PTA (dB) right ear	15.84±3.76	16.84±3.90	16.07±3.76

Table 2: BERA results of patients in group 1 (Type 1 DM), group 2 (Type 2 DM) and group 3 (control) subjects in left ear and their comparison

Waves and IPL	Control (group 3) (Mean±SD)	Type1 DM (group1) (Mean±SD)	Type2 DM (group 2) (Mean±SD)	p value group 1-3	p value group 2-3	p value group 1-2
Wave I	1.60±0.11	1.68±0.20	1.71±0.14	0.014*	0.000*	0.344
Wave II	2.70±0.18	2.76±0.23	2.79±0.20	0.172	0.028*	0.493
Wave III	3.65±0.19	3.83±0.23	3.88±0.21	0.001*	0.000*	0.241
Wave IV	4.77±0.20	4.86±0.25	4.92±0.25	0.079	0.002*	0.196
Wave V	5.51±0.17	5.75±0.25	5.83±0.28	0.000*	0.000*	0.172
IPL I-III	2.09±0.22	2.15±0.12	2.17±0.18	0.076	0.043*	0.542
IPL III-V	3.91±0.17	4.07±0.20	4.11±0.26	0.000*	0.000*	0.384
IPL I-V	1.83±0.24	1.92±0.18	1.94±0.27	0.034*	0.027*	0.635

[*-Significant (p<0.05)]
Values in columns 2-4 in milliseconds

Table 3: BERA results of patients in group 1 (Type 1 DM), group 2 (Type 2 DM) and group 3 (control) subjects in right ear and their comparison

Waves and IPL	Control (group 3) (Mean±SD)	Type1 DM (group1) (Mean±SD)	Type2 DM (group 2) (Mean±SD)	p value group 1-3	p value group 2-3	p value group 1-2
Wave I	1.59±0.14	1.70±0.22	1.76±0.17	0.005*	0.000*	0.125
Wave II	2.73±0.21	2.77±0.31	2.80±0.25	0.478	0.111	0.508
Wave III	3.69±0.19	3.83±0.25	3.93±0.22	0.002*	0.000*	0.051
Wave IV	4.69±0.24	4.90±0.29	4.87±0.28	0.000*	0.001*	0.654
Wave V	5.56±0.28	5.80±0.29	5.89±0.23	0.000*	0.000*	0.084
IPL I-III	2.10±0.21	2.13±0.13	2.15±0.21	0.381	0.308	0.729
IPL III-V	3.97±0.25	4.10±0.25	4.13±0.23	0.009*	0.001*	0.528
IPL I-V	1.86±0.31	1.97±0.22	1.96±0.22	0.063	0.067	0.967

*-Significant (p<0.05)
Values in columns 2-4 in milliseconds

Table 4: Comparison of BERA parameters in relation to duration of type 1 DM

Parameters			Duration < 10years(n=27) Mean±S.D.	Duration ≥ 10years (n=23) Mean±S.D.	't' value	'p' value
LEFT EAR	Waves	I (ms)	1.65±0.17	1.72±0.23	1.33	0.190
		II (ms)	2.72±0.21	2.80±0.24	1.31	0.197
		III (ms)	3.80±0.19	3.87±0.26	1.18	0.246
		IV (ms)	4.81±0.22	4.91±0.28	1.30	0.201
		V (ms)	5.71±0.20	5.79±0.31	1.08	0.286
	Interpeak Latencies	I-III (ms)	2.15±0.12	2.15±0.12	0.01	0.987
		I-V (ms)	4.07±0.19	4.07±0.22	0.05	0.962
		III-V (ms)	1.92±0.14	1.92±0.23	0.04	0.967
	Amplitude	I-Ia (µV)	0.93±0.84	1.37±2.26	0.89	0.383
		V-Va (µV)	0.95±0.62	1.20±1.50	0.76	0.452
		V/I Amp. Ratio	1.52±1.13	1.57±1.10	0.14	0.892
RIGHT EAR	Waves	I (ms)	1.69±0.19	1.71±0.26	0.30	0.766
		II (ms)	2.76±0.30	2.78±0.32	0.20	0.840
		III (ms)	3.83±0.23	3.83±0.28	0.01	0.990
		IV (ms)	4.86±0.22	4.93±0.36	0.82	0.419
		V (ms)	5.78±0.22	5.82±0.36	0.46	0.648
	Interpeak Latencies	I-III (ms)	2.14±0.15	2.12±0.11	0.51	0.612
		I-V (ms)	4.09±0.16	4.11±0.32	0.27	0.788
		III-V (ms)	1.95±0.11	1.99±0.30	0.59	0.560
	Amplitude	I-Ia (µV)	1.00±0.80	0.91±0.75	0.384	0.703
		V-Va (µV)	1.35±0.95	1.09±0.88	0.98	0.333
		V/I Amp. Ratio	1.89±1.50	1.51±1.07	1.06	0.295

Table 5: Comparison of BERA parameters in relation to duration of type 2 DM

Parameters			Duration < 10years (n=26) Mean±S.D.	Duration ≥ 10years (n=24) Mean±S.D.	't' value	'p' value
LEFT EAR	Waves	I (ms)	1.72±0.16	1.71±0.12	0.39	0.699
		II (ms)	2.79±0.23	2.78±0.17	0.14	0.891
		III (ms)	3.88±0.26	3.89±0.16	0.23	0.823
		IV (ms)	4.96±0.29	4.88±0.19	1.22	0.267
		V (ms)	5.84±0.35	5.81±0.19	0.50	0.623
	Interpeak	I-III (ms)	2.16±0.19	2.18±0.17	0.57	0.571

	Latencies	I-V (ms)	4.12±0.31	4.00±0.20	0.32	0.749
		III-V (ms)	1.97±0.34	1.91±0.17	0.70	0.485
	Amplitude	I-Ia (µV)	0.97±0.80	1.26±1.65	0.79	0.435
		V-Va (µV)	1.07±0.90	1.62±2.37	1.08	0.291
		V/I Amp. Ratio	1.17±0.82	1.89±1.59	1.97	0.057
RIGHT EAR	Waves	I (ms)	1.76±0.19	1.77±0.16	0.20	0.843
		II (ms)	2.78±0.26	2.83±0.24	0.77	0.445
		III (ms)	3.91±0.25	3.94±0.20	0.41	0.686
		IV (ms)	4.84±0.33	4.91±0.23	0.87	0.389
		V (ms)	5.85±0.28	5.93±0.16	1.33	0.192
	Interpeak Latencies	I-III (ms)	2.14±0.23	2.15±0.19	0.27	0.789
		I-V (ms)	4.09±0.28	4.17±0.16	1.18	0.246
		III-V (ms)	1.93 ±0.25	1.99±0.17	0.99	0.326
	Amplitude	I-Ia (µV)	1.32±1.33	1.04±0.84	0.90	0.374
		V-Va (µV)	2.05±2.43	1.59±1.72	0.77	0.445
		V/I Amp. Ratio	1.40±0.86	1.45±0.89	0.19	0.852

Auditory Brainstem Response (ABR) morphology was normal in all groups. Wave latencies were prolonged in diabetic groups as compared to control group in both right and left ear. In Type 1 DM patients, mean absolute latencies of waves I, III, V and IPL III-V of both ears and latency of wave IV in right ear and IPL I-V in left ear were significantly prolonged ($p<0.05$) as compared to control group [Table 2]. In Type 2 DM patients, absolute latencies of all waves and interpeak latencies show significant difference ($p<0.05$) in comparison with controls in both ears except wave II latency and IPL I-III on right side which show no significant difference [Table 3]. When the two diabetic groups were compared with each other, latency prolongation was more pronounced in Type 2 DM patients, but the difference was not statistically significant.

Diabetic patients were divided according to the duration of disease into two categories: a) duration less than 10 years and b) duration more than or equal to 10 years. For Type 1 diabetic group, number of patients under each category were 27 and 23 respectively. Similarly for type 2 DM group, the division was 26 and 24 patients respectively. The difference between absolute latencies of waves and interpeak latencies was not statistically significant between two categories in both groups of DM patients.

There was no significant difference ($p>0.05$) between latency of waves, interpeak latencies and amplitude in Type 1 DM patients with duration of disease < 10 years and those with ≥ 10 years in both left and right ear.

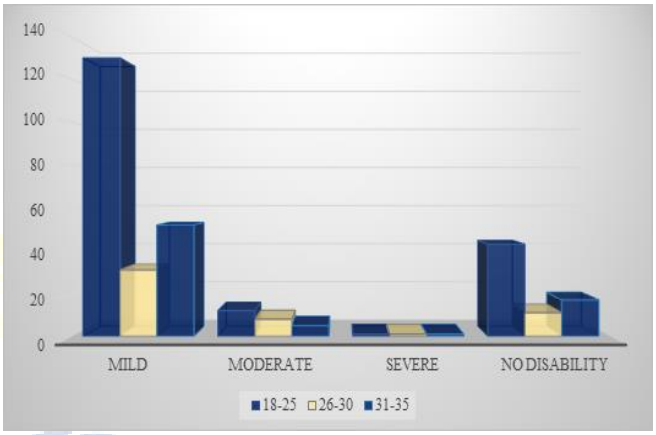

Figure 1: Shows the Association Between Neck Disability with Age Group

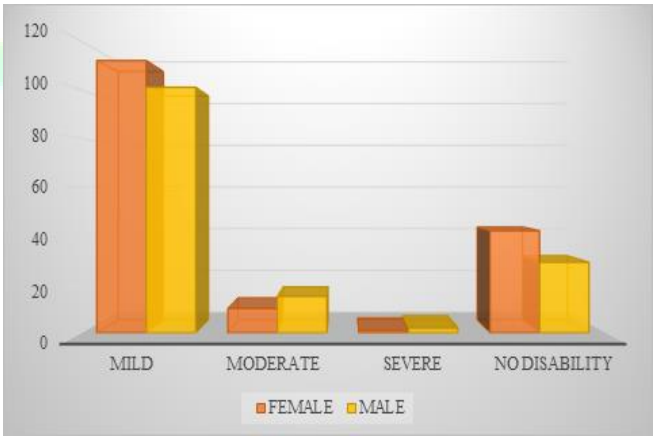

Figure 2: Shows the Association Between Neck Disability with Sex

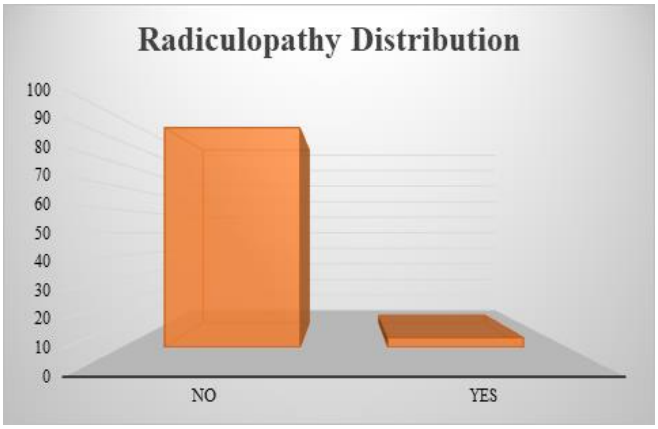

Figure 3: Shows the Association Between Neck Disability with Radiculopathy

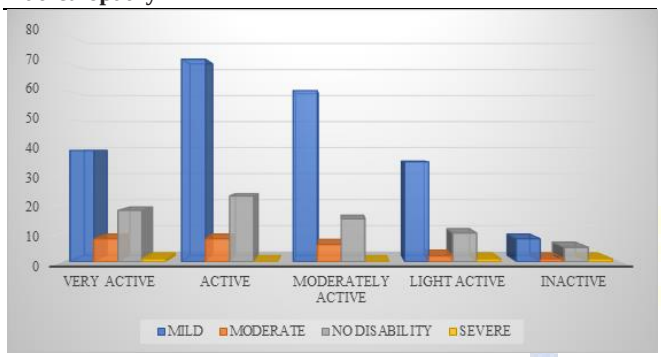

Figure 4: Shows the Association Between Physical Activity Group with Neck Disability

Discussion

For this study, 312 individuals with chronic neck pain between the ages of 18 and 35 years old were considered. The findings of the study revealed that the largest proportion of neck disability was recorded in the 18-25 age group, followed by the 31-35 age group i.e. 59.6% and 24%. However, our study discovered no statistical relationship between neck disability and age group in the population studied. Hildebrandt et al.[13] did a study to find association of physical activity and pain in neck, 2030 cases were taken and revealed that the mean age was 33.7 years. Côté et al,[14] on the other hand, did a study on 2184 cases and found that the highest frequency percentage was seen in the 40-49 year age range i.e 53%.

In terms of gender distribution, our study found out that females (52.9%) were affected more than male 47.1%. However, findings of the analysis revealed that there wasn't any statistically significant relationship between Neck disability and sex of the cases. Hill et al.[15] revealed in his research that in cases with pain in neck for more than three months, females were more affected than males i.e 52% and 48% respectively as the females are less physically active than males. But according to Hey et al.[16] who studied 626 cases complaining of chronic neck pain in which the prevalence of male 54% were shown to be greater than females 46%

In this study total 4.2 percent of all the cases of neck pain had presented to us with radiculopathy and majority 95.8 percent did not have radiculopathy. According to Rodine et al,[17] radiculopathy is a common source of neck pain and functional impairment in the elderly. Salemi et al,[18] reported that the annual incidence of radiculopathy was 83.2 per 100,000 cases, while Murphy et al,[19] assessed that the frequency of radiculopathy with chronic neck pain was 3.5 per 1000 people in their study. The findings of this study show a statistically significant relationship between neck disability and radiculopathy as p value is 0.048.

The largest percentage of participants with chronic neck pain was recorded in active group 32.7%, followed by the moderately active 26%, very active 21.2%, light active 15.4% and inactive 4.8% categories in terms of physical activity distribution. Physical activity has also been linked to a lower occurrence of cervical pain. According to Feldman and Diepenmaat,[20] physical activity is not related to neck and shoulder pain, whereas Niemi et al., Siivola et al,[21] said that physical activity can reduce the risk of neck and shoulder pain. Geene et al,[22] study showed that the maximum cases were recorded in active group 44% followed by moderate active i.e 22% and stated that there was no relation between physical activity and neck disability in the cases. In our study, we however, found that the association between the physical activity group and neck disability was statistically non-significant as p value was 0.30.

Strength of the study

This study has focused on finding out any possible association of neck disability with chronic neck pain in young age population as there is higher incidence of neck disability in young aged working population. This study also rules out any relation between the co-morbidities and neck disability.

Limitations of the study

Study was done in a limited age group (18-35 age) and there was a time constraint too.

Conclusion

This study adds to the evidence that neck pain is frequent among people between the ages of 18 and 35 years. According to the findings of this study, there are significant connections between comorbidities for some cases. In this study we also found that there is no significant association between neck disability with physical activity. Individuals who are seriously hampered by neck pain and who have comorbidities should get appropriate medical guidance, which is highly crucial.

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