Central and Peripheral Conduction Abnormalities in Diabetes Mellitus

Fawi GH[1], Khalifa GA[1], Kasim MA[2]
Departments of Neurology[1], General Medicine[2], South Valley University

ABSTRACT

Background: *Diabetes mellitus is one of the most serious challenges to health care world wide and its effect on peripheral and central nervous system are known.* ***Aim of the work:*** *To detect and analysis possible correlation between central , peripheral and autonomic neuropathies in one hand with duration of illness, type of treatment, glyceamic control on the other hand.* ***Subject and methods:*** *20 patients type 1 diabetes, 20 patients type 2 and 20 age matched controls, all were subjected to nerve conduction studies, electromyography, brainstem auditory evoked potential and autonomic function test, with exclusion of other possible cases which can cause peripheral or central neuropathies.* ***Results:*** *Peripheral neuropathy detected in 75% of type 1 patients and 80% in type 2 and autonomic neuropathy 20% in type 1 and 10% in type 2.Severities of neuropathy were related to duration of illness and blood glucose level. Abnormalities of brainstem auditory evoked potential were reported in all patients of the study and all were correlated positively with blood glucose level, presence of neuropathy but not with duration of illness.* ***Conclusion:*** *Peripheral neuropathy is more in type 2 than type 1, while central neuropathy detected by abnormalities in brainstem auditory evoked potential were detected in all diabetic and was not related to duration of illness. Central and peripheral neuropathies were positively related to type of treatment, glyceamic control and duration of illness except central neuropathy which was not related to duration of illnesses. Central neuropathy was positively correlated to presence of peripheral neuropathy.* ***(Egypt J. Neurol. Psychiat. Neurosurg., 2005, 42(1): 209-221).***

INTRODUCTION

Diabetes mellitus is one of the most serious challenges to healthcare worldwide and is projected to affect 239 million people by the year 2010, a doubling in the prevalence since 1994.[1] There are a great numbers of researches were done to detect the effect of diabetes on the peripheral and central nervous system, and the central neuropathy was documented in several recent studies in diabetic patients by the using of the evoked potentials studies.[2]

Diabetes affects conductive function in central as well as peripheral conduction. By using of somatosensory evoked potentials studies there are prolongation of central conduction time and also lowering of the peripheral sensory conduction in diabetic patients.[3]

Diabetes cause central neuropathy which was detected by using of auditory brainstem response (ABR), Visual evoked potential (VEP), somatosensory evoked potentials (VEP) where all of these tests showed abnormalities as prolonged latencies and also abnormalities of wave form morphology in (ABR). These abnormalities are present at different levels and may appear before appearance of overt complications.[4] These abnormalities in evoked potential studies are present in central afferent and efferent pathways, in afferent pathway the primary sensory neuron is more affected than the subsequent stages, probably as an expression of a central - peripheral distal axonopathy. Also those central nervous system abnormalities are more frequent in-patients with peripheral neuropathy but evoked potentials

can be abnormal even in-patients without neuropathy. The pathophysiology of the central nervous system abnormalities is uncertain, many causes are probably active including neuronal damage; chronic hyperglycemia, hypoglycemic episodes, angiopathy, blood-brain barrier dysfunction and others still unknown.[5]

These central conduction abnormalities which occur with diabetes are more or less correlated with glycemic control, but in somatosensory evoked potentials the abnormalities is still present but strict glycomic control may influence and retard the progression of central conduction involvement.[2, 6]

The brain stem neuropathy in diabetes mellitus, which is proved by using of auditory brain stem response in diabetic patients with and without known complications as diabetic retinopathy, and or nephropathy, is in the form of, prolonged of I-V interpeak latency, with also abnormal waveform morphology in 55.2% of diabetic patient and these abnormalities increase with presence of diabetic complication.[7]

Aim of the work:

1- To characterize the afferent brainstem functions by using of neurophysiological studies (BAEP)
2- To detect peripheral neuropathy clinically and by using of neurophysiological studies (NCS and EMG).
3- To analyze possible correlations between central neural dysfunctions and peripheral neuropathy.
4- To analyze possible correlations between duration of illness, type of treatment, blood glucose level and central and peripheral conduction abnormalities in diabetic patients.

SUBJECTS AND METHODS

A case control study in which we were evaluating 20 patients with type 1diabetes mellitus (9 males and 11 females) and 20 patients with type 2 diabetes mellitus (7 males and 13 females) and 20 age-matched controls were obtained from the healthy relatives of the patients (6 males and 14 females)with ages ranged from 21 to 61 years, with mean of 36 years. Non-had a history of diabetes, all reported normal hearing, and non-were talking regular medication, which could be expected to affect the cortical functioning

Inclusion Criteria: Patients were chosen randomly from the medical out patient clinic of Sohag University Hospital. Their ages ranged from 20 to 60 years, with a mean of 40 years. Duration of illness ranged from 6 month to 45 years, with the mean duration of 16 years.

Exclusion Criteria: Patients were excluded if they suffered from any concurrent diseases that affect the brain or the nervous system, such as uremia, cerebrovascular stroke, hepatic encephalopathy, ect…..

No patient in the sample was being treated with any medication, which might be expected to interfere with the functioning of the central nervous system such as methyldopa, reserpine, ect….

The patients and the control groups were informed about the study and oral concepts were taken from them .

The patients and the controls were subjected to the following:

I. Detailed medical and neurological history and examination.
II. Autonomic neuropathy was diagnosed by presence of history suggesting autonomic neuropathy, and abnormal cardiovascular adrenergic (sympathetic) function tests which includes systolic blood pressure response to standing, diastolic blood pressure response to sustained handgrip, and systolic blood pressure response to tilting.
III. The hearing of the diabetic patients and control subjects were assessed in the Department of Audiology of Sohag University Hospital. Patients and control subjects were examined otoscopically, followed by audiometery to rule out peripheral hearing loss.
IV. Blood glucose level (fasting and postprandial).

V. Glycoselated hemoglobin (to detect metabolic control).

VI. Neurophysiological studies were done by using of Neuropack IV- mini System as the following:
   * Nerve conduction studies (motor and sensory) of median nerve and common peroneal nerve.
   * Electromyographic studies of abductor pollicis brevis and extensor digitorum brevis.
   * Brainstem Auditory Evoked Potential (BAEP).

VII. Peripheral neuropathy was evaluated by using simple two-stage scheme in which patients with possible neuropathy are identified and then referred to a standardized clinical and electrophysiological assessment to confirm the presence of neuropathy and to gauge its severity again concentrating on lower limb function[8] as the following:

Table 1. Stage 2 of Diabetic Neuropathy Score Evaluation[8].

Clinical component 1: Muscle strength (right and left).			
Assessment	Muscle strength (maximum score - 12)		
	Normal	Mild/Moderate weakness	Severe weakness
Finger spread	0	1	2
Ankle dorsiflexion	0	1	2
Extension of hallux	0	1	2
Clinical component 2: Tendon reflexes (right and left).			
Assessment	Tendon reflex (maximum score =16)		
	Present	Only on reinforcement	Absent
Biceps brachii	0	1	2
Triceps brachii	0	1	2
Quadriceps	0	1	2
Ankle	0	1	2
Clinical component 3: Sensory impairment (right and left).			
Assessment	Sensory impairment (maximum score =12)		
	Normal	Reduced	Absent
Vibration on hallux	0	1	2
10 g nylon filament	0	1	2
Pinprick on dorsum of hallux	0	1	2
Electrophysiological component (dominant side).			
Assessment	Electrophysiology (maximum score =5)		
	Normal	Abnormal	
Motor nerve conduction velocity			
Peroneal	0	1	
Median	0	1	
Sensory conduction			
Sural	0	1	
Median	0	1	
Ulnar	0	1	
Diabetic Neuropathy Score			
Stage	**Clinical score definition**	**Electrophysiological score definition**	
Class 0	6 ≤	0 –1	
Class 1	7 – 12	2	
Class 2	13 – 29	3 – 4	
Class 3	30 ≥	5	

RESULTS AND DISCUSSION

Table 2. Neurological signs and symptoms.

	Type 1	Type 2	P value
Weakness			
Distal weakness upper limb	10%	5%	0.179
Proximal weakness upper limb	5%	-	0.023
Distal weakness lower limb	35%	35%	1
Proximal weakness lower limb	10%	-	0.000
Autonomic manifestations	30%	10%	0.000
Muscle wasting			
Distal upper limb	-	-	-
Proximal upper limb	-	-	-
Distal lower limb	30%	15%	0.011
Proximal lower limb	5%	-	0.023
Lost reflexes and hyporeflexia			
Biceps	40%	25%	0.023
Brachioradials	40%	30%	0.138
Triceps	40%	30%	0.138
Knee	50%	40%	0.155
Ankle	70%	55%	0.028
Sensory manifestations in both upper and lower limbs			
1. Parasthesia			
Tingling and numbness	25%	30%	0.428
Radicular pain	5%	-	0.023
2. Superficial sensation			
Glove hyposthesia	-	5%	0.023
Stoke hyposthesia	30%	15%	0.011
Radicular sensory loss	5%	-	0.023
3. Deep sensory loss	45%	35%	0.148
4. Loss of cortical sensation	40%	25%	0.023
Percent of neuropathy	75%	80%	0.869

According to Stage 2 of Diabetic Neuropathy Score Evaluation:

1. **Type 1 diabetes:**
   a) According to clinical evaluation:
   50% have neuropathy and severity of neuropathy was:
   - 5% class 1"mild neuropathy".
   - 25% class 2 "moderate neuropathy".
   - 20% class 3 "sever neuropathy".

   b) According to electrophysiological evaluation:
   75% have neuropathy and severity of neuropathy was:
   - 10% class 1 "moderate neuropathy".
   - 30% class 2 "moderate neuropathy".
   - 35% class 3 "sever neuropathy"

2. Type 2 diabetes:

a) According to clinical evaluation:

60% have neuropathy and severity of neuropathy was:

- 20% class 1“ mild neuropathy”.
- 20% class 2 “moderate neuropathy”.
- 20% class 3 “sever neuropathy”

b) According to electrophysiological evaluation:

80% have neuropathy and severity of neuropathy was:

- 5% class 1 “moderate neuropathy”.
- 50% class 2 “moderate neuropathy”.
- 25% class 3 “sever neuropathy”

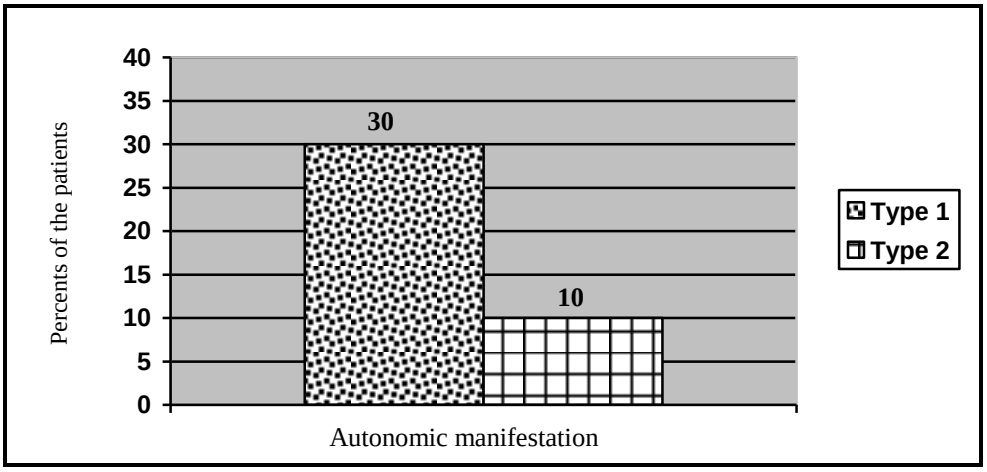

Chart (1): Autonomic manifestation.

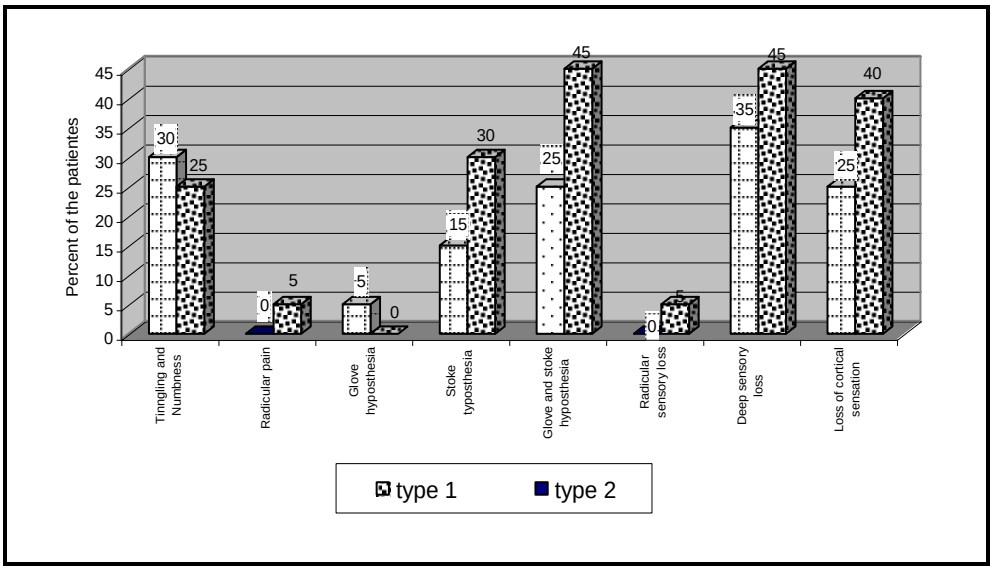

Chart (2): Sensory manifestation.

1. Incidence of neuropathy:

Our study showed that the incidence of diabetic peripheral neuropathy was 80% in type 2 and 75% in type 1, and that is in agreement with Nathan[9], who reported that approximately two-thirds of type 1 and type 2 patients had subclinical or clinical evidence of peripheral neuropathy, and that is higher than the results reported by Thomas and Tomlinson[10], which reported that the incidence of diabetic peripheral neuropathy ranged from 50-60% of diabetic patients and also is lower than that reported by Dyck et al.[11], who reported that all of diabetic patients have neuropathy, but the condition is subclinical in many. In our study there is no significant difference between both groups of diabetes, and that is in agreement with Dyck[12]. In our study the incidence of autonomic neuropathy was 30% in type 1 patients and 10% in type 2 patients with significant difference between both groups ($P<0.05$) and that is agreement with Low et al.[13], who reported that autonomic neuropathy, being detectable in approximately 30% of diabetic patients. Lower than our study, Stevens et al.[14] found that the incidence of autonomic neuropathy was 16.7%, in another study[15], reported that 25.3% of patients with type 1 diabetes and 34.3% of patients with type 2 diabetes had abnormal findings in more than two of six autonomic function tests, also with O'Brien et al.[16], who reported that (16.6%) of individuals with insulin-dependent diabetes had autonomic neuropathy, and with Purewal and Watkins[17], which found that 42% of diabetic children and adolescents showed one or more abnormal tests for cardiovascular autonomic dysfunction. These results, however, reported that incidence rates will vary depending on:

1) Different patient cohorts studied.
2) Varied testing modalities utilized.
3) Different criteria used to define autonomic dysfunction.[18]

2. Relation between Duration and Peripheral Neuropathy:

Table 3. Correlation coefficient between EMG and NCS and duration in type 1 and type 2 diabetic patients.

	Type 1		Type 2	
	Correlation	**P-value**	**Correlation**	**P-value**
Amplitude in upper limb	-0.286	0.235	0.025	0.922
Amplitude in lower limb	0.480	0.058	-0.130	.0544
Distal latency of left common peronial nerve	0.514	0.029	-0.690	0.049
Motor conduction velocity of left common peronial nerve	-0.467	0.050	-0.548	0.041
Distal latency of left sural nerve	0.513	0.037	0.644	0.014
Motor conduction velocity of left sural nerve	0.513	0.037	0.644	0.014
Sensory conduction velocity of right ulnar nerve	-0.691	0.045	-0.675	0.047
Distal latency of sensory conduction of right ulnar nerve	-0.792	0.032	-0.686	0.045
Distal latency of motor conduction right median nerve	0.595	0.037	0.754	0.014
Motor conduction velocity of right median nerve	-0.091	0.034	-0.275	0.047
Sensory conduction velocity of right median nerve	-0.742	0.002	-0.447	0.053
Distal latency of sensory conduction of right median nerve	0.318	0.048	0.487	0.055

In our study, there was a positive relation between duration and presence of peripheral neuropathy (P<0.05). Our results also are in agreement with the results of previous studies[19,20] which examined 1441 patients divided into two groups, one of which included patients with no retinopathy and diabetes for 1 to 5 years (primary prevention cohort), and another group with minimal to moderate nonprolifrative retinopathy and diabetes for 1 to 15 years (secondary intervention cohort). Sural, and median sensory and peroneal and median motor nerve conduction velocity results were reported at baseline and at 1, 2, 9 years. Some abnormalities in nerve conduction in at least two nerves was measured in about 20% of patients in the primary prevention cohort and about 45% of the secondary intervention cohort at baseline.

3. Relation between Metabolic Control and Peripheral Neuropathy:

In our study, there was a positive relation between metabolic control and presence of peripheral neuropathy. Our results are in agreement with the results of the previous studies.[19,20]

4. Relation between Metabolic Control and Autonomic Neuropathy:

In our study, there was a positive relation between metabolic control and presence of autonomic neuropathy (P<0.05) and that is agreement with the results of the previous study[21], which reported that incidence and severity of autonomic neuropathy are related with metabolic control.

Auditory Brainstem Response

1- Type of Diabetes and BAEPs abnormalities:

In our study auditory brainstem response in patients showed that all of patients have BAEPs abnormalities, in the form of prolongation of absolute latencies of waves II, III, IV and V in right and left ear of both groups of diabetic patients and wave I only in type 1 DM, with very highly significant difference of waves III, IV, V of right and left ear between both groups of diabetic patients and the control group (P<0.01), also there is significant difference in wave II in type 1 patients in comparison with control group (P<0.05). As regard interpeak latencies, there are prolongation of I-III, I-V and III-V interpeak latencies in both groups of diabetic patients in comparing with the control group.

In type 1 diabetic patients there are very highly significant difference of I-III, III-V and I-V interpeak latencies of right ear and I-V interpeak latencies of left ear (P<0.01) with insignificant difference regarding I-III, III-V interpeak latencies of left ear in comparison with control group.

In type 2 diabetic patients there are very highly significant difference of I-III, III-V and I-V interpeak latencies of left ear and I-III interpeak latencies of right ear (P<0.01) and significant difference of III-V in left ear (P<0.05) with insignificant differences of III-V interpeak latencies of right ear in comparison with control group.

Our results agreed with Donald et al.[22], who examined 20 patients with type 1 diabetes mellitus, and reported normal latencies of waves I and II and prolonged latencies of waves III and V and prolonged interpeak latencies I-III and I-V but in disagreement regarding prolongation of waves I and II in our study.

The delay in interpeak latencies I-III and I-V, are evidence of a central conduction delay at brainstem-to-midbrain level. The fact that normal wave latency of wave I and wave II suggest that eighth nerve transmission time is still normal and so the eighth's nerve transmission time is delayed in type 1 and normal in type 2 diabetic patients.[22]

Our results also are in agreement with the results of Pozzessere et al.[4], who examined 25 diabetic patients and reported BAEP abnormalities in all of them. Also we agreed with Toth et al.[23], which examined 15 patients with long standing type 1 diabetes mellitus, all of them had abnormal BAEP with prolonged latencies of waves I, III and V and prolonged interpeak latencies I-III and I-V and that was statistically significant with the control group. Also in

agreement with our study, Bayazit et al. reported prolonged absolute latency of I, III, V and prolonged interpeak latency I-III and I-V but disagreed with him only significant to control group was I-III interpeak latency (P<0.01). Also agreed with Lisowska et al.[24], which reported prolonged absolute latency of I, III, V and interpeak latency I-III and I-V but disagreed with him in the significant difference to control group which in his study the only significant to control group was I-V interpeak latency and absolute latency of wave I. Also agreed with Chaudhari et al.[25], which reported significantly prolongation of absolute latencies of waves I to V, Inter peak latencies I-III and I-V of BAEP of pregnant women with gestational diabetes compared with 20 age matched normal pregnant women. But disagreed with them in prolongation of latency of wave I who in our study was normal was normal in type 2 and insignificant prolongation of type 1.

On the other hand, much lower results were reported by Kharodi et al.[26], who examined 34 patients with long standing type 1 diabetes mellitus and reported only 32% of diabetic patients had abnormal brainstem auditory evoked potentials, also Goldsher et al.[27] reported only 48% of patients with type 1 DM with average durations was 5 years had prolonged latencies I, III and V and interpeak latency I-III and III-V. Also Das et al.[28], who reported prolonged latencies of BAEP in 50% of type 2 and 14.8% of type 1 diabetic patients. That discrepancy may be explained by high percent of neuropathy in our patients, which reported that BAEP abnormality are correlated positively with presence of peripheral neuropathy which was reported by the previous studies[23,5]. Also the possibility of silent stroke in our patients is still present, as the computer tomography of the brain were not done, also may be due to the difference in duration of illness where in our study the duration of illness was reported to be more prolonged than pervious studies, which (mean±SD) in type 1 was 15.7±8.9 years and in type 2 was 11.4±21 years.

In our study there is no statistically significance difference of BAEP abnormality in both types of diabetic patients except for wave V latency in left ear which is more prolonged in type 1 and that statistically significant, which conclude that BAEP abnormalities not affected by the type of diabetes and that agreement with Pozzessere et al.[4].

The delay of the central transmission time in diabetics may be related to diffuse neuropathological changes that have been found in the optic nerves, periventricular regions, brainstem and spinal cord in postmortem pathological studies. Similar changes have been found in animals with experimental diabetes. The pathophysiology of central nervous system (CNS) abnormalities is uncertain, many causes are probably active in including neural damage: chronic hyperglycemia, hypoglycemic episodes, angiopathy, blood-brain barrier dysfunction and others, still unknown.[5]

Table 4. Auditory Brainstem Response in Type 1 and control.

Absolute Latency "mean ±SD"	**Type 1**		**Control**		**P value**	
	Rt ear	**Lt ear**	**Rt ear**	**Lt ear**	**Rt ear**	**Lt ear**
Wave I	1.62±0.28	1.85±0.28	1.52±0.18	1.55±0.13	0.602	0.071
Wave II	2.75±0.27	2.82±0.25	2.62±0.16	2.59±0.15	0.116	0.004
Wave III	3.87±0.35	3.91±0.32	3.55±0.18	3.49±0.21	0.000	0.000
Wave IV	5.32±0.38	5.25±0.44	4.77±0.19	4.84±0.19	0.000	0.014
Wave V	5.96±0.48	6.08±0.6	5.39±0.23	5.41±0.21	0.000	0.000
Interpeak Latency						
I-III	2.3±0.4	2.11±0.55	1.96±0.16	1.98±0.11	0.001	0.331
III-V	2.11±0.45	2.19±0.43	1.84±0.2	1.98±0.33	0.018	0.101
I-V	4.23±0.18	4.38±0.68	3.86±0.3	3.86±0.17	0.000	0.003

Table 5. Auditory Brainstem Response in Type 2 and control.

Absolute Latency "mean ±SD"	**Type 2**		**Control**		**P value**	
	Rt ear	**Lt ear**	**Rt ear**	**Lt ear**	**Rt ear**	**Lt ear**
Wave I	1.5±0.38	1.57±0.24	1.52±0.18	1.55±0.13	0.523	0.742
Wave II	2.74±0.32	2.69±0.31	2.62±0.16	2.59±0.15	0.257	0.228
Wave III	3.9±0.36	3.84±0.3	3.55±0.18	3.49±0.21	0.003	0.000
Wave IV	5.06±0.28	5.04±0.22	4.77±0.19	4.84±0.19	0.003	0.017
Wave V	5.77±0.4	5.74±0.46	5.39±0.23	5.41±0.21	0.002	0.006
Interpeak Latency						
I-III	2.37±0.43	2.32±0.39	1.96±0.16	1.98±0.11	0.001	0.001
III-V	1.94±0.35	2012±0.23	1.84±0.2	1.98±0.33	0.206	0.000
I-V	4.24±0.56	4.08±0.58	3.86±0.3	3.86±0.17	0.041	0.000

Table 6. Auditory Brainstem Response in Type 1 and Type 2.

Absolute Latency "mean ±SD"	**Type 1**		**Type 2**		**P value**	
	Rt ear	**Lt ear**	**Rt ear**	**Lt ear**	**Rt ear**	**Lt ear**
Wave I	1.62±0.28	1.85±0.28	1.5±0.38	1.57±0.24	0.348	0.416
Wave II	2075±0.27	2.82±0.25	2.74±0.32	2.69±0.31	0.927	0.160
Wave III	3.87±0.35	3.91±0.32	3.9±0.36	3.84±0.3	0.775	0.329
Wave IV	5.32±0.38	5.25±0.44	5.06±0.28	5.04±0.22	0.014	0.094
Wave V	5.96±0.48	6.08±0.6	5.77±0.4	5.74±0.46	0.183	0.025
Interpeak Latency						
I-III	2.3±0.4	2.11±0.55	2.37±0.43	2.32±0.39	0.952	0.186
III-V	2.11±0.45	2.19±0.43	1.94±0.35	2.1±0.23	0.228	0.006
I-V	4.23±0.18	4.38±0.68	4.24±0.56	4.08±0.58	0.952	0.115

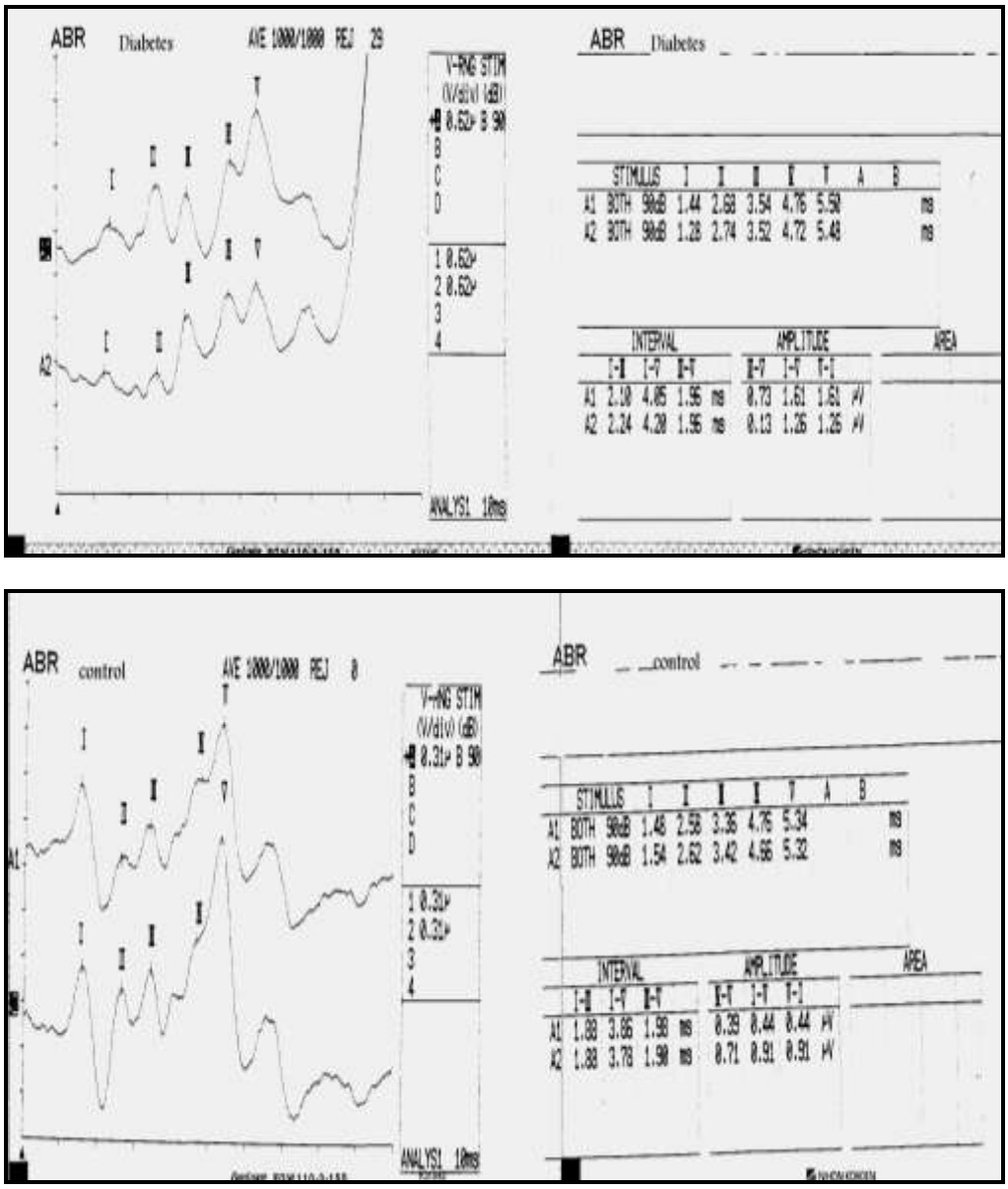

Chart (3): Traces of ABR in Diabetic and control.

2- Duration and BAEPs abnormalities:

There is no relation between duration of illness and BAEP in type 2 diabetic patients and that is in agreement with the previous studies[22,26,27], but in disagreement with these studies is the presence of positive correlation between latencies of wave III and wave V in left ear and interpeak latencies of I-III and I-V in the right ear in type 1 diabetic patients with no exact explanation for difference.

3- Relation between metabolic control and BAEPs abnormalities:

In our study, there was a positive relation between metabolic control (which detected by serial random blood glucose level in both groups of diabetes and was in normal range) and BAEPs abnormalities in both groups of diabetic patients ($P<0.05$). Our results are in agreement with Pozzessere et al.[4], who reported that evoked potential abnormalities are correlated with metabolic control also the previous studies[6,2], reported that evoked potential abnormalities are reversible in diabetic patients after improvement of metabolic control status. But in contrast with Donald et al.[22] and Kharodi et al.[26], who reported that BAEPs abnormalities are not correlated with duration of illness or blood glucose level (fasting and postprandial).

4- Relation between BAEPs abnormalities and Neuropathy:

In our study, there was a positive relation between BAEPs abnormalities and presence of neuropathy either peripheral or autonomic ($P<0.05$) and that is in accordance with the previous studies[27,23] who reported that BAEPs abnormalities reported more increase with presence of autonomic and peripheral neuropathy

Summery and Conclusion

Our study showed that the incidence of peripheral neuropathy was 75% in type 1 diabetic patients and 80% in type 2 diabetic patients and the incidence of autonomic neuropath was 30% in type 1 diabetic patients and 10% in type 2 diabetic patients, also we reported that the severity of the neuropathy are more in type 1 diabetic patients which was reported clinically and by neurophysiological methods. Our results showed that the nerve conduction studies are affected by the duration of illness and blood glucose level, but there is no difference between both types of diabetes regarding abnormalities reported by NCS and EMG. Also our study showed that all diabetic patients in the study had abnormal BAEP with no difference between both type of diabetes, these BAEP abnormalities were correlated positively with blood glucose level but not correlated with duration of illness. Also the BAEP abnormalities were correlated positively with presence of peripheral neuropathy.

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الملخص العربي

يعتبر مرض السكري من اكثر واخطر الأمراض التي تؤثر على الصحة العامة حيث انه متوقع أن يبلغ عدد المصابين بالمرض الى 239 مليون نسمة بحلول عام 2010. وعلما بان اعتلال الأعصاب الطرفية هو من أهم المضاعفات التي تنتج عن مرض السكري .فأنة أيضا يوجد اعتلال في الوظائف المركزية للجهاز العصبي على مستوى وظائف العقل العليا مثل التركيز والذاكرة ومعدل الذكاء والتي تم إثباتها سواء لمرض السكري عن طريق اختبارات فسيولوجيا الأعصاب أو عن طريق التصوير بالأشعة للمخ أو عن طريق الاختبارات والقياسات النفسية.

الهدف من الرسالة :

هو تقييم مرض السكري إكلينيكيا وعن طريق الوسائل الفسيولوجية الكهربية لتحديد التأثر في الوظائف التوصيلية للجهاز العصبي الطرفي والمركزي وذلك بعمل اختبارات سرعة توصيل الأعصاب ورسم العضلات والاستجابة السمعية لجزع المخ .وأيضا محاولة إثبات وجود علاقة بين تأخر التوصيل بالجهاز العصبي المركزي ووجود اعتلال بالأعصاب الطرفية الناتج عن مرض السكري. وأيضا محاولة إثبات وجود علاقة بين هذه التغيرات في سرعة التوصيل (الطرفية والمركزية) وبين مستوى السكر بالدم ونوع العلاج ومدة الإصابة بالمرض. وقد تمت هذه الدراسة في قسم الأمراض العصبية بكلية طب سوهاج وفيها تم تقييم (20 مريضا من النوع الأول لمرض السكري) المعتمد علي الأنسولين و (20 مريضا من النوع الثاني لمرض السكري) الغير معتمد علي الأنسولين و 20 شخص سليم من أقارب المرضى كمجموعة سليمة للقياس. وقد تم اختيار المرضى عشوائيا من عيادة مرضى السكري التابعة لقسم الأمراض الباطنة وكان عمر المرضى يتراوح ما بين 20 إلى 60 سنة وكان متوسط عمر المرضى 45 سنه وكان متوسط مدة الإصابة بالمرض 16 سنة. وقد تم استبعاد المرضى الذين يعانون من أية أمراض أو يتناولون عقاقير قد تؤثر على الجهاز العصبي أو يعانون من أية مشاكل بالسمع. ولقد خضع كل من المرضى ومجموعة القياس للآتي:

1. فحص طبي مفصل.
2. عمل نسبه السكر بالدم (صائم وبعد ساعتين).
3. رسم عضلات وسرعة توصيل الأعصاب.
4. الاستجابة السمعية لجزع المخ.

وقد بينت الدراسة أن الإصابة باعتلال الأعصاب في النوع الأول لمرض السكري تصل إلى 75% وفي النوع الثاني تصل إلى 80% وان الإصابة باعتلال الأعصاب الذاتية تصل الي 30% في النوع الأول وفي النوع الثاني تصل إلى 10% وان قوة الإصابة باعتلال الأعصاب في النوع الأول لمرض السكري اكثر من النوع الثاني لمرض السكري. كما بينت أيضا أن سرعة توصيل الأعصاب تتأثر سلبيا بنسبه السكر بالدم وانه لا يوجد اختلاف في رسم العضلات وسرعة توصيل الأعصاب بين النوع الأول و الثاني لمرض السكري. كما بينت أيضا أن الاضطرابات في الوظيفة التوصيلية لجزع المخ تتأثر إيجابيا بنسبه السكر بالدم ووجود اعتلال بالأعصاب الطرفية ولكنها لا تتأثر بمدة الإصابة بالمرض. كما أظهرت الدراسة أن كل المرضى قي العينة المأخوذة يعانون من اضطرابات في الوظيفة التوصيلية لجزع المخ وانه لا يوجد اختلاف في الوظيفة التوصيلية لجزع المخ بين النوع الأول والثاني.

التوصيات :

نستنتج من هذه الدراسة أن الاستجابة السمعية لجزع المخ اختبار سهل وبسيط ويمكن أن يحدد أية اضطرابات بالجهاز العصبي المركزي قبل ظهور أية مضاعفات لمرض السكري. وأن تنظيم نسبة السكر بالدم و توصيلها إلى المعدل الطبيعي لها يمنع ظهور المضاعفات العصبية للمرض.