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Evaluation of central neuropathy in type II diabetes mellitus by multimodal evoked potentials

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Abstract

The electrophysiological results in 51 patients with diabetes mellitus type II were compared with those in 30 age and sex matched healthy control subjects.

Peripheral and cortical latencies of median and tibial somatosensory evoked potentials (SEP), bilateral I-III and I-V interpeak latencies (IPL) of brainstem auditory evoked potentials (BAEP), bilateral P100 latency of visual evoked potentials (VEP) and bilateral cortical latency and central motor conduction time of motor evoked potentials (MEP) were evaluated. We observed prolonged latencies suggestive of central neuropathy in DM type II.

It has been shown that most of the electrophysiological parameters in patients with DM type II correlate with the duration of the disease, some of them with the age of the patient, and few of them with the onset of the disease. To our knowledge, there is no correlation between the electrophysiological parameters and the level of glycemia or the degree of metabolic control. We conclude that central and peripheral neuropathies in DM are related to the duration of the disease and not to the degree of hyperglycemia and metabolic control.

Key words : Somatosensory Evoked Potentials (SEP) ; Motor Evoked Potentials (MEP) ; Visual Evoked Potentials (VEP) ; Brainstem Auditory Evoked Potentials (BAEP) ; Diabetes Mellitus (DM).

Introduction

Abnormalities of central afferent and efferent pathways can be measured by evoked potential studies. The peripheral nervous system in DM has been investigated a lot in the literature. The central nervous system could also be abnormal in patients with peripheral neuropathy. Brainstem auditory evoked potential (BAEP), somatosensory evoked potentials (SEP) and visual evoked potentials (VEP) can be affected together, but isolated abnormalities are more frequently observed. The pathophysiology of central nervous system (CNS) abnormalities in DM is not well understood, probably many causes are responsible for the neural damage, including, chronic hyperglycemia, hypoglycemic episodes, blood-brain barrier dysfunction, angiopathy, and others (Chokroverty, 1897 ; Dejong, 1976 ; Carsten *et al.* 1989 ; Comi *et al.* 1997).

In contrast to pathological studies, electrophysiological investigation is a very sensitive method in determining peripheral and central neuropathy in diabetic patients. In many patients with normal clinical examination, a decrease in nerve conduction velocity can be observed (Felsenthal and McLuar, 1984 ; Abraham and Abraham, 1986 ; Lopez-Alburquerque *et al.* 1987). Central neural conduction can be evaluated byclinical use of the evoked potentials. Latency delay of the evoked potential is a specific finding that can be seen in demyelinating diseases. Evoked potential studies are non-invasive and easily applicable methods in determining focal brain damage and subclinical central demyelination (Carsten *et al.* 1989 ; Cerizza *et al.* 1990).

The aim of this study was to investigate central neuropathy in DM Type II by using multimodal evoked potentials.

Subjects and methods

Patients were selected after informed consent was obtained. The inclusion criteria included confirmed diagnosis of DM ; normal liver and renal function ; no concomitant systemic disease, malignancy, or cerebrovascular disease ; able to cooperate ; normal hearing (the auditory deficit was determined by testing the auditory threshold during the clinical examination and BAEP testing).

Patient Group

We evaluated 51 subjects, 29 females and 22 males, with DM Type II (mean age was 48.46 ± 15.43 years). Confirmation of DM was based on several fasting plasma glucose values exceeding 126 mg/dl, according to the current criteria of the American Diabetes Association. The duration of disease varied from 2 to 21 years (average 5.3 ± 6.2 years). The patients had no other obvious risk factor for neuropathy (such as alcoholism, exposure to neurotoxic drugs, or renal failure).

Table 1

Clinical and laboratory features of the patients and the control group

	Diabetes mellitus	Control group	P
N	51	30	
Age	48.76 ± 15.43.	48.46 ± 15.43	0.23
Sex (F/M)	29/22	19/11	
Onset age of the DM (month)	42.76 ± 14.24		
Duration of diabetes (year)	5.30 ± 6.2		
BMI	25.44 ± 10.21	22.23 ± 3.02	0.32
Glycemia (mg/dl)	204.20 ± 96.07	96.55 ± 9.11	0.001*
HbA1c (%)	9.25 ± 1.04	5.47 ± 0.44	0.001*

Data are means ± SEM.*$p < 0.05$.

Control Group

Age- and sex- matched control subjects 19 females and 11 males were selected as a control group. Their mean age was 48.4 years (mean 48.46 ± 15.43 years). Each control subject had a normal physical and neurological examination.

Electrophysiological measurements

Median and tibial SEP, BAEP, VEP, MEP were bilaterally obtained in all subjects. In all subjects age, sex, onset age of the diabetes, glycemia level during the examination and the degree of metabolic control and BMI were recorded.

Evoked potentials were recorded by a 4 channel EMG device, DISA (Denmark). Standard methodswere used as described elsewhere (Kimura, 1989 ; Nakamura *et al.* 1986 ; Yaltkaya *et al.* 1988).

Body mass index (BMI) was determined as the ratio of body weight (kg) to the height square (m^2).

All statistical analysis was performed by SPSS 10.0 (SPSS Inc. Chicago, Illi., USA). For the descriptive statistics we used mean ± SEM notation. In comparising patients and control groups, we used “Independent sample t test”. The correlation (coefficients) between electrophysiological results, demographic and laboratory data were given as Pearson correlation coefficients. In order to investigate the statistical significance between groups, we used “Paired sample t test” or “ Mann-Whitney U test” ($p < 0.05$; $p < 0.01$ or $p < 0.001$ were considered statistically significant)

Results

Clinical and Laboratory Findings

Clinical features of the patients and the control group are given in Table 1.

Median Somatosensory Evoked Potentials

Prolongation in both Erb, cervical and cortical potential latencies was measured determined and compared to the control group. The results are given in Table 2.

Tibial Somatosensory Evoked Potentials

Both thoracic and cortical potential latencies and thoraco-cortical conduction time were acquired separately after stimulation of left and right tibial nerves. Prolongation in both thoracic and cortical potential latencies and thoraco-cortical conduction time was determined compared to the control group. The results are given in Table 2.

Auditory Brain Stem Evoked Potentials

The increase in I-V IPL was due to I-III IPL, as the III-V IPL was in normal range. The results are given in Table 2.

Visual Evoked Potentials

The prolongation of P100 latency in diabetics was compared to the control group. The results are given in Table 2.

Motor Evoked Potentials

The prolongation of motor central conduction time (MCCT) was compared to the control group. The results are given in Table 2.

The Correlation with metabolic and demographic values

The duration of diabetes mellitus is positively correlated with Erb, cervical, cortical median SEP, cortical tibial SEP, VEP, cervical MEP, cortical MEP latencies and MCCT ($r = 0.35$; $p < 0.05$, $r = 0.37$; $p < 0.05$; $r = 0.35$, $p < 0.05$; $r = 0.48$, $p < 0.001$; $r = 0.57$, $p < 0.001$; $r = 0.68$, $p < 0.001$; $r = 0.75$, $p < 0.001$, $r = 0.49$, $p < 0.05$, respectively).

There is a positive correlation between the age and cortical tibial SEP, BAEP III-V IPL, VEP P100, cervical MEP, cortical MEP latencies, and

Table 2

Results of median and tibial SEP, BAEP, VEP and MEP in patients and control group

	Diabetic Group	Control group	P
Median SEP			
Erb	10.22 ± 1.65	9.18 ± .49	0.001*
Cervical	13.95 ± 1.05	12.61 ± 0.69	0.001*
Cortical	19.93 ± 1.54	18.44 ± 1.19	0.001*
CCT	6.18 ± 0.93	5.97 ± 1.02	0.24
Tibial SEP			
Thoracal	25.11 ± 2.64	21.87 ± 2.08	0.03*
Cortical	41.87 ± 4.87	38.27 ± 1.59	0.001*
CCT	19.26 ± 4.05	16.78 ± 1.86	0.002*
BAEP			
Wave I	1.68 ± 0.09	1.65 ± 0.12	0.59
I-III IPL	2.25 ± 0.19	2.10 ± 0.21	0.01*
III-V IIPL	1.96 ± 0.19	1.94 ± 0.23	0.65
I-V IPL	4.20 ± 0.27	4.02 ± 0.27	0.03*
VEP			
P100	115.22 ± 7.82	104.40 ± 7.33	0.001*
MEP			
Cortical	23.22 ± 1.79	21.33 ± 1.21	0.001*
Cervical	14.64 ± 1.51	14.21 ± 0.93	0.08
MCCT	8.67 ± 1.18	6.97 ± 1.11	0.004*

Results were given as msn, * $p < 0,05$.

MCCT ($r = 0.50$, $p < 0.001$; $r = 0.28$, $p < 0.05$; $r = 0.36$, $p < 0.05$; $r = 0.31$, $p < 0.05$; $r = 0.44$, $p < 0.05$; $r = 0.46$, $p < 0.05$, respectively).

There is a positive correlation between the age of onset of the disease and cortical tibial SEP latencies, and BAEP III-V IPL ($r = 0.38$, $p < 0.05$; $r = 0.31$, $p < 0.05$, respectively).

There is no significant correlation between the degree of metabolic control, the level of glycemia, the demographic values and the electrophysiological parameters (Table 3).

Discussion

Although the peripheral nervous system in DM has been investigated a lot, the term “central neuropathy” has been unknown until recently. Electrophysiological investigations are sensitive in determining peripheral and central neuropathy in diabetic patients. Decrease of nerve conduction velocity was found in many patients with normal clinical examination (Felsenthal and Mc Luar, 1984 ; Abraham *et al.*, 1986 ; Lopez-Alburquerque *et al.* 1987). A latency delay in evoked potentials is found in central demyelinating diseases. Evoked potentials are usefull as an investigational method in establishing neuropathy developing in the central nervous system.

In our study, an increase in Erb, cervical, and cortical potential latency in median SEP was shown, but central conduction time (CCT) was normal. In the study of Cerizza *et al.* (1990) in 20 old patients with DM Type II, median SEP were evaluated, and no increase in central conduction time was found. Harkins *et al.* (1985) found prolongation in peripheral components of median SEP in 10 patients with DM.. Gupta and Dorfman (1981) showed a specific anomaly in supra-spinal conduction (spinal cord-cortex) and argued that the delay in SEP latency was related to the decrease of conduction in peripheral nerves.

Collier *et al.* (1988) did not find any change in the cervical- cortical and Erb-cervical conduction in median SEP of 18 patients with DM. They concluded that the prolongation in cervical and cortical potential latency was due to the prolongation in Erb potential latency. Kandha *et al.* (1990) reported that SEP abnormalities were correlated with the decrease of conduction in peripheral nerves. The results of our study show that there is a prolongation in peripheral response latency in median SEP. The latency prolongation of cortical evoked potential is of peripheral origin. In other studies, it has been also reported that there is a central conduction slowing in median SEP. But the fact that the duration of the disease in the patients included in this study is long so that it can be explained by theprolongation in central conduction. In median SEP study that Nakamura *et al.* (1992) performed in the 54 patients with DM, while Erb potential latency found to be measured, the prolongation in CCT has not been observed. Suziki *et al.* (2000)

Table 3

Correlation between the demographical and electrophysiological results, and metabolic parameters

	Age	Onset of the disease	Duration of the disease	BMI	$HbA1_C$	Glycemia
Median SEP						
Erb	0.23	0.11	0.35*	-0.04	-0.09	0.11
Cervical	0.23	0.11	0.37*	-0.07	-0.10	-0.03
Cortical	0.34*	0.25	0.35*	-0.11	-0.10	0.07
CCT	0.23	0.24	0.08	-0.21	-0.06	0.15
Tibial SEP						
Thoracal	0.12	0.07	0.16	-0.15	0.02	-0.03
Cortical	0.50***	0.38*	0.48***	-0.15	0.01	-0.01
BAEP						
Wave I	0.11	-0.21	-0.11	0.16	0.13	0.14
I – III IPL	-0.01	-0.01	-0.01	0.21	-0.25	0.02
III– V IPL	0.28*	0.31*	0.06	-0.18	-0.11	0.05
I – V IPL	0.19	0.21	0.03	0.00	-0.24	0.05
VEP						
P100	0.36*	0.18	0.57***	0.23*	0.16	0.22
MEP						
Cervical	0.31*	0.07	0.68***	-0.15	0.02	0.11
Cortical	0.44*	0.20	0.75***	-0.12	0.01	0.14
MCCT	0.46*	0.34	0.49*	-0.02	0.02	0.13

*p < 0.05, **p < 0.01, *** p < 0.001.

reported that both peripheral and central median SEP components were prolonged in patients with diabetes.

In our study, thoraco- cortical conduction time as well as latency of thoracic and cortical responses in SEP was also determined. In diabetic patients, studies performed by tibial SEP are sparse. Stimulating the tibial nerve generally made somatosensory investigations. Nakamura *et al.* (1989) found an increase in cortical potential latency in tibial SEP in 53% of 34 diabetic patients. In this study thoracic responses were evaluated. The author suggested that the prolongation of the cortical response latency resulted from both peripheral and spinal cord damage in another study (Nakamura *et al.* 1992). Maetzu *et al.* (1995) reported that thoracic CCT was prolonged in diabetes. Varsik *et al.* (2001) reported that somatosensory evoked potentials and conduction time were the best way of investigating and confirming an unapparent lesion of the spinal cord in diabetics.

We found a prolongation in both thoracic and cortical latencies in tibial SEP ; we suggest that dysfunction of both peripheral and central projections of the dorsal root ganglion cells that is first neuron in the sensory tracts, causes the prolongation in CCT.

In our study with median SEP, we did not find a central effect, which can be explained by the shortness of the central projections of the cervical dorsal root ganglions. It is possible to explain the differences affects between median and tibial SEP by the length of axons of the involved nerves (dying back neuropathy).

Khardori *et al.* (1986) studied BAEP in 34 patients with DM type I, and found a delay in I-V and III-V IPL's. Donald *et al.* (1984) showed increase in wave V latency and I-III IPL in 50 patients with DM. Verma *et al.*(1984) found no BAEP changes in 22 patients with DM, while Fedele *et al.*(1984) showeda delay in wave I latency as well as in IPL's in 30 patients with DM. The results of these studies are not in agreement. In our study, as Donald *et al.* anticipated, a prolongation of I-III IPL was found. The fact that III-V IPL is normal indicates that the increase in I-V IPL results from an increase in I-III IPL. This also indicates that the dysfunction is localized in the caudal part of the pons. We conclude that the auditory brain stem responses are disturbed in central segments in diabetic patients. But when we consider all published studies, it is impossible to determineif there is an influence in the lower part, upper part or the whole brainstem.

In our study, bilateral increase of VEP latency was found. None of our diabetic patients had retinopathy. In 19 subjects with DM Type II, Algan *et al.* (1989) showed an increase in P100 potential latencies. Mariani *et al.* (1990) also found an increase in P100 latency in 35 diabetic subjects without retinopathy. Lanting *et al.* (1991) investigated pupil light reflex latency and P100 latency in 42 diabetic subjects and found that pupil light reflex latency was prolonged in 55% of subjects

and P100 latency was increased in 19 % of subjects. There was no correlation between diabetic retinopathy, pupil light reflex latency and P100 latency. Ponte *et al.* (1986) reported an increase in P100 latency in 50 subjects with asymptomatic DM type I who had no retinopathy. Puvamendran *et al.* (1983), Crillo *et al.* (1984) and Anastasi *et al.* (1987) reported abnormal VEP in subjects with DM. Trick *et al.* (1988) reported that the increase of VEP latency was related to retinopathy. Yaltkaya *et al.* (1988) showed an increase in N 140 latency and N90-N140 IPL, as well as a prolongation in P100 latency in diabetic subjects and suggested that this was due to retrochiasmal involvement. Comi *et al.* (1986) also reported identical findings. Millinger *et al.* (1989) reported that abnormal VEPs could reflect maculopapular fiber or optic nerve involvement. Bortek *et al.* (1989) found abnormal VEPs in 77% of27 diabetic subjects, not correlating with retinopathy. Moreo *et al.* (1995) reported that the P100 wave latency was delayed in NIDDM patients.

In our study, we used bilateral P100 potential latency analyses ; VEP latency was prolonged as in the other studies ($p = 0.001$). It is impossible to say that this prolongation results from retinal or central pathways dysfunction.

In our study, an increase in MCCT and in potential latency after bilateral cortical stimulation in MEP was found ($p = 0.004$). When estimating the central motor conduction by magnetic stimulation, it is well established that cervical stimulation excites the cervical spinal roots at their exit through the neuroforamina ; so, the central motor conduction time includes a small peripheral segment. Some studies reported that central motor pathways were affected much more than peripheral motor pathways (Maetzu *et al.*1995 ; Abbruzzese *et al.* 1993 ; Kimura, 1989 ; Tchen *et al.* 1992), these findings are not in accordance with the clean slowing in peripheral nerve conduction velocity. MEP measurements were not always reliable and we have to find more sensitive methods to investigate the central motor pathways.

In summary, evoked potentials (SEP, BAEP, VEP and MEP) are complementary studies rather than their superiority to each other in evaluating the central neuropathy associated with DM.

We conclude that central and peripheral neuropathies in DM are related to the duration of the disease and not to the level of glycemia and metabolic control.

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