



ORIGINAL ARTICLE

Postural control and central motor pathway involvement in type 2 diabetes mellitus: Dynamic posturographic and electrophysiologic studies

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Abstract *Background:* Postural instability causes limitations in daily activities of diabetic patients. There is paucity of data regarding central motor pathway involvement in these patients and its relation to postural control.

Aim: To evaluate postural control and central motor pathway involvement in type 2 diabetic patients.

Subjects and methods: The study included 30 type 2 diabetic patients and 15 healthy, age and sex-matched control subjects. Both groups were subjected to physical and full neurological examination, in addition to electrophysiological study including peripheral conduction study and MEPs recorded from the feet muscles. Total neuropathy score was calculated. In addition, dynamic posturographic tests were performed including sensory organization test and MCT.

Abbreviations: CMCT, central motor conduction time; MEP, motor evoked potential; MCT, motor control test.

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Results: Most of the dynamic posturographic parameters were significantly impaired in diabetic patient group. There were significant abnormalities in most of the parameters of the peripheral conduction study of the patients compared to the controls. According to the Total neuropathy score, 20 patients had peripheral neuropathy. In addition, there was significant prolongation of the left CMCT, decreased left MEP amplitude and increased MEP resting motor threshold on both sides in the patients compared to the control group. Dynamic posturographic parameters showed correlation with most of the parameters of the peripheral conduction study and few of the MEP parameters. Logistic regression analysis showed peripheral neuropathy as the main factor implicated in postural instability in these patients. However, significant correlation was found between MEP amplitude and MCT composite score in patients without peripheral neuropathy.

Conclusion: Although type 2 diabetic patients had prolonged CMCT, decreased amplitude and increased resting motor threshold of the MEP response, peripheral neuropathy was the main factor implicated in postural instability. However, the central motor pathway changes documented could be implicated as a possible cause.

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1. Introduction

Postural instability is one of the complications associated with diabetes mellitus (DM).¹ Type 2 diabetic patients often exhibit impaired balance and gait dynamics, and are at a greater risk of falling. Many individuals who fall develop a fear of falling, resulting in a further limitation of activity, reduced mobility and physical fitness.²

Postural sway is greater in diabetics with peripheral neuropathy in both eyes open and eyes closed conditions.³ Peripheral neuropathy (PN) seems to be a primary factor leading to sensory and motor deficits, which often result in balance impairments.⁴ However, other factors as autonomic neuropathies, foot disorders, visual impairments and changes in postural coordination cannot be ruled out as additional causes of postural impairment in these patients.^{1,5} In addition, several parts of the central nervous system (CNS), which consists of the spinal cord and the brain, take part in controlling posture.⁶ The automatic postural responses are the earliest functionally effective responses that mediate a person's active postural movements' control in response to external balance perturbations.⁷ They are mediated by peripheral and central long latency pathways. Posture is also guided by a mixture of programs and sensory feedback. Calculations of these postural programs and this feedback are made ahead of time in the CNS and are always corrected after comparison to central and peripheral reports about reality.⁸ Consequently, damage to the CNS can interfere with the coordination of posture and movement control, placing an individual in fear of his or her own movements.

Central nervous system complications resulting from DM is a problem that is gaining high attention. Diabetic neuropathy involves not only the peripheral nervous system, but also the central nervous system.⁹ Few data about the incidence of central diabetic neuropathies are available but CNS degeneration is a well known pathology in diabetic patients in the long term.¹⁰ It was assumed that the damage of the CNS is mostly caused by changes in cerebral circulation as well as metabolic disturbances.^{11,12} Neuropathological and neuro-imaging techniques revealed structural damage in the brain and spinal cord tissues, demyelination, signs of micro and macro-angiopathies, and cerebral atrophy.¹³⁻¹⁵ These changes may contribute to postural impairment in diabetic patients. However, the effect

of central motor pathway changes on posture control in these patients has not been confirmed.

Abnormalities of central efferent pathways can be measured by evoked potential studies.¹⁶ Motor evoked potentials (MEPs) are useful as a non invasive and nearly painless investigation of propagation along corticospinal tracts controlling limb and axial musculature.¹⁷ On the other hand, dynamic posturography is a quantitative method for assessing upright balance function under a variety of tasks that effectively simulate the conditions encountered in daily life.¹⁸

The aim of this work was to evaluate postural control and central motor pathway involvement in type 2 diabetic patients.

2. Subjects and methods

This study included 30 type 2 diabetic patients and 15 healthy, age and sex-matched control subjects. All the studied subjects were chosen fulfilling the following criteria: age between 40 and 65 years, duration of diabetes mellitus discovery more than 5 years and a normal or fully corrected visual acuity.¹⁹ Patients having any of the following were excluded from the study: musculoskeletal problems that could interfere with their postural control mechanisms,²⁰ neurological disease other than those attributed to diabetes mellitus (e.g. stroke, parkinsonism, myopathy, non diabetic neuropathy, etc...),²⁰ previous trauma or surgical operation involving the back or lower limbs,²⁰ concurrent intake of medications that would affect balance,²¹ as well as the presence of contraindications to the transcranial magnetic stimulation.²²

Both groups were subjected to general clinical and full neurological examinations. Glycosylated hemoglobin (HbA1c) was measured in the patient group. In addition, the following tests were applied to all studied subjects:

2.1. Computerized dynamic posturography (CDP)

Dynamic posturography¹⁸ was performed using the "Equitest System"; NeuroCom International, Inc. Portland, OR, USA. The following tests were performed:

2.1.1. Sensory organization test (SOT)

The test consists of six experimental conditions. Each condition provided a special set of sensory inputs, while the subject

under test maintained his balance with minimal sway.¹⁸ Based on the center of gravity data recorded by the equipment, the following parameters were measured through software version 4 of the equitest program.

- 1) The equilibrium score for each condition (C): It quantifies mean score of each of the six test conditions.
- 2) SOT composite score: reflects the patient's overall performance in SOT.
- 3) Sensory ratios: the relative differences in scores are expressed as ratios. The four sensory ratios and their physiologic meaning are summarized in Table 1

2.1.2. Motor control test (MCT)

It is designed to measure the automatic postural responses elicited by translating the support surface in the horizontal direction (forward and backward translations). During these two experimental sets, the tested subject stood quietly with eyes opened, facing the visual surrounds, and tried to maintain balance.

The measured parameters recorded were the response latency (milliseconds), strength symmetry and response strength (degrees/second) following mechanical translation (forward/backward).The composite MCT latency was recorded as well.

2.2. Electrophysiological tests

Neuropack 2 electromyograph apparatus from Nihon Kohden (Japan) was used to perform the electrophysiological studies. The following procedures were carried out according to standardized techniques.

2.2.1. Peripheral nerve conduction study

The following peripheral nerves^{23,24} were studied to determine the existence of peripheral neuropathy: **A)** Motor conduction study for posterior tibial nerves with recording from abductor hallucis muscle bilaterally, and deep peroneal nerves with recording from extensor digitorum brevis muscle in both lower limbs. In addition, posterior tibial minimal F wave latency was recorded bilaterally. **B)** Sensory conduction study of sural nerve. Recording electrodes used were surface electrodes, 7 mm in diameter. Stimulation was done using bipolar stimulator.

Table 1 The four sensory ratios and their physiologic meaning.

Sensory ratio	Ratio pair	Significance
SOM	Condition 2	Patient makes poor use of
Somatosensory ratio	Condition 1	Somatosensory reference
VIS	Condition 4	Patient makes poor use of visual
Visual ratio	Condition 1	Reference
VEST	Condition 5	Patient makes poor use of vestibular
Vestibular ratio	Condition 1	Cues, or vestibular cues unavailable
PREF	Condition 3 + 6	Patient relies on visual cues even
Visual preference	Condition 2 + 5	When they are inaccurate

2.2.2. Motor evoked potentials (MEPs)

Transcranial magnetic stimulations (TMS)^{17,25,26} of the motor cortex were delivered through a single pulse stimulator, Magstim 200 (Magstim company, Whitland, Wales, UK), equipped with a high power 90 mm circular coil, capable of generating 2 T maximum field intensity.

For the left hemisphere stimulation, the coil was held with face A of the coil visible from above (current anticlockwise) and for stimulation of the right hemisphere, the coil was held with face B visible from above. The coil was positioned tangentially over the skull, with the center of the coil placed over the cortex and the handle parallel to the sagittal plane. The abductor hallucis muscle was activated if the center of the coil was moved 4–6 cm frontally from the vertex and 2–3 cm laterally, contralateral to the side from which the MEPs were recorded according to the international 10–20 system of international electroencephalographic electrode placement.^{25,26}

The abductor hallucis muscle responses were recorded with 7 mm surface disk electrodes taped in a belly/tendon montage.¹⁷ The following parameters were determined:

1. **Resting motor threshold (RMT):** It was determined by applying the stimulus strength (given in percentage of the maximum output of stimulator) increasingly in 5% increments with the target muscle (AH in lower limbs) in complete relaxation until a compound muscle action potential (CMAP) was seen. The RMT was defined as the lowest intensity that gives three reproducible responses about 50–100 μV.²⁶
2. **Motor evoked potential recording:** The patient was asked to do mild voluntary contraction of the target muscle. Stimulus intensity was set at 20% above threshold for evoking reproducible muscle responses.¹⁷ Three consecutive responses were superimposed. The following were recorded:
 - i. The shortest MEP cortical latency (CL) was determined from the first visible deflection from the baseline.¹⁷
 - ii. Then the central motor conduction time (CMCT) was calculated.^{25,26}

The formula for calculation of CMCT by using F-wave recording is:

$$CMCT = CL(ms) - \text{Peripheral latency}(PL)(ms).$$

$$PL = (\text{Minimal F - wave latency} + \text{M - wave latency} - 1)/2$$

where 1 ms is the estimate delay for turnaround time of the antidromic volley at the anterior horn cell.

- i. Peak to peak maximal amplitude of MEPs was determined.¹⁷

2.3. Total neuropathy score (TNS)

It was calculated for each patient to determine peripheral nerve function.²⁷ Data from symptoms (sensory, motor, autonomic), signs (superficial and deep sensations, muscle strength using Medical Research Council grading,²⁸ and reflexes), and nerve conduction studies (sural and peroneal nerves amplitudes) were used for calculation of the score.

3. Statistical analysis

Statistical analyses were performed using SPSS® Statistics version 20.

The data of the studied sample were not normally distributed. Quantitative data were described using measures of central tendency (mean and median) and dispersion (standard deviation, minimum and maximum). Mann–Whitney U test was used to compare quantitative variables including peripheral conduction study, MEP, and dynamic posturographic variables. Spearman's rho correlation coefficient was used to test correlation between these variables. Readings that lie above the third quartile (Q75) were considered to be abnormally high, while those that lie below the first quartile (Q25) were considered to be abnormally low. **Factor analysis** was used to construct a quantitative measure for the central changes from the MEP parameters namely, MEP amplitude, RMT and CMCT. Central factor was extracted to represent these MEP variables. **Logistic regression model** was developed to determine the independent predictors (central factor, total neuropathy score) of impaired posture among diabetic patients.

4. Results

None of the patients had autonomic manifestations, motor weakness, or signs of upper motor neuron involvement.

The studied patients showed significantly multiple SOT test abnormalities. Low scores were observed in condition C1, C2, C4 and C6 ($U = 109, p = .005$), ($U = 75, p = .000$), ($U = 56.5, p = .001$) and ($U = 94, p = .042$) respectively). The SOT composite score was significantly low ($U = 69, p = .004$). Patients also had significantly low somatosensory ($U = 155.5, p = .003$) and visual ratios ($U = 33, p = .000$) compared to the control group. Regarding the MCT, there was a statistically significant higher MCT composite score ($U = 62.5, p = .006$) of patient group compared to control group. The results also showed significant prolongation of the response latency for large backward perturbation of both right ($U = 95, p = .013$) and left lower limbs ($U = 95.7, p = .013$), right side medium backward perturbation

($U = 84.500, p = .012$), right side small forward perturbation ($U = 77, p = .01$) Table 2.

No correlations were found between age, disease duration and any of the dynamic posturographic parameters used.

Patient group showed impairment in motor and sensory nerve conduction study of lower limbs as compared to control group. Posterior tibial nerves of both sides showed statistically significant prolongation of distal latency (DL) (right ($U = 90.5, p = .028$), left ($U = 55, p = .001$)), decrease of compound muscle action potential (CMAP) amplitude (right ($U = 64.5, p = .003$), left ($U = 68, p = .004$)), slowing of conduction velocity (CV) of the leg segment (right ($U = 69.5, p = .005$), left ($U = 58.5, p = .002$)) and prolongation of F wave latency (right ($U = 74, p = .007$), left ($U = 53, p = .001$)) of the patient group compared to the control group. Similarly, deep peroneal nerve on both sides showed statistically significant prolongation of DL (right ($U = 48, p = .027$), left ($U = 48, p = .027$)), decrease of CMAP amplitude (right ($U = 49, p = .003$), left ($U = 48, p = .003$)) and slowing of CV of the leg segment (right ($U = 33.5, p = .006$), left ($U = 39, p = .01$)) of the patient group compared to the control group. As regards sural nerve, it showed statistically significant slowing of conduction velocity of patients compared to controls ($U = 46.5, p = .002$)).

According to the TNS, it was found that 20 (66.67%) patients had peripheral neuropathy, whereas 10 (33.34%) patients had no peripheral neuropathy.

On the other hand, TMS of the cerebral cortex demonstrated statistically significant prolongation of the right ($U = 59.5, p = .001$) and left ($U = 43.5, p = .001$) MEP cortical latency, decrease in the left MEP amplitude ($U = 71, p = .002$), increase in right ($U = 30.5, p = .000$) and left ($U = 30.5, p = .000$) MEP motor threshold as well as a significant left CMCT ($U = 110, p = .05$) delay in patient group compared to control group. Table 3

Delayed CMCT was demonstrated in 12 (40%) and 19 (63.33%) patients on right and left sides, respectively.

Neither MEP parameters nor TNS was correlated with age. Cortical latency was positively correlated with disease duration ($r = 0.44, p = .015$) and with the TNS ($r = .376, p = .04$). There was positive correlation between glycosylated

Table 2 Comparison between patients and control groups regarding the sensory ratios, SOT composite score and MCT composite score.

Variables	Patients group $n = 30$	Control group $n = 15$	Mann whitney U (p -value)
<i>Somatosensory ratio</i>			
Median (Min–max)	0.96(0.86–1)	0.98(0.96–1)	155.5*
Mean \pm SD	0.95 \pm 0.04	0.98 \pm 0.02	(.003)
<i>Visual ratio</i>			
Median (Min–max)	0.8(0.69–0.92)	0.9(0.82–0.96)	33*
Mean \pm SD	0.81 \pm 0.07	0.9 \pm 0.04	(.000)
<i>SOT composite equilibrium score</i>			
Median (Min–max)	76(69–87)	81(77–87)	69*
Mean \pm SD	76.76 \pm 4.59	81.03 \pm 2.93	(.004)
<i>MCT composite score</i>			
Median (Min–max)	143.5(125–168)	135(124–143)	62.5*
Mean \pm SD	143.13 \pm 9.55	134.20 \pm 6.8	(.006)

SOT = sensory organization test, MCT = motor control test, SD = standard deviation, Min = minimum, Max = maximum.

* Significant at $p \leq .05$.

Table 3 Comparison between patients and controls regarding motor evoked potential parameters.

Variables	Patients group <i>n</i> = 30	Control group <i>n</i> = 15	Mann Whitney <i>U</i> (<i>p</i> -value)
<i>Rt resting motor threshold (%)</i>			
Median (Min–max)	80(60–100)	60(40–80)	30.5*
Mean ± SD	77 ± 6	55 ± 14	(.000)
<i>Rt motor evoked potential</i>			
• Cortical Latency (ms)			
Median (Min–max)	41.8(36.6–52.2)	37.7(36.6–40.4)	59.5*
Mean ± SD	42.24 ± 4.8	37.97 ± 1.14	(.001)
• Amplitude (mV)			
Median (Min–max)	0.93(0.09–1.7)	1.99(0.9–5.37)	122.5
Mean ± SD	0.87 ± 0.55	2.32 ± 1.99	(.109)
• CMCT (ms)			
Median (Min–max)	14.13(8.1–19.85)	12.85(10.75–15.35)	137.5
Mean ± SD	14.42 ± 2.79	13.27 ± 1.44	(.237)
<i>Lt Resting motor threshold (%)</i>			
Median (Min–max)	80(60–100)	50(40–80)	20*
Mean ± SD	78 ± 6	48 ± 15	(.000)
<i>Lt Motor evoked potential</i>			
• Cortical latency (ms)			
Median (Min–max)	43.6(36.2–61.8)	38.5(34.8–41.6)	43.5*
Mean ± SD	44.11 ± 5.45	37.85 ± 2.26	(.000)
• Amplitude (mV)			
Median (Min–max)	0.55(0.13–3.5)	1.71(0.14–7.17)	71*
Mean ± SD	0.74 ± 0.67	2.58 ± 2.4	(.002)
• CMCT (ms)			
Median (Min–max)	16.1(6.1–29.05)	12.8(10.35–17.95)	110*
Mean ± SD	15.59 ± 4.42	13.16 ± 2.09	(.05)

n = number, *rt* = right, *lt* = left, *ms* = millisecond, *mV* = millivolt, *CMCT* = central motor conduction time, *Max* = maximum, *Min* = minimum, % = percentage, *SD* = standard deviation.

* Significant at $p \leq 0.05$.

hemoglobin and both MEP cortical latency ($r = .373$, $p = .042$) and CMCT ($r = .552$, $p = .002$).

Table 4 shows the statistical correlation of dynamic posturographic variables with the electrophysiologic variables in patient group. There was statistically significant correlation between the MCT composite score and posterior tibial nerve DL ($r = .410$, $p = .009$), CV ($r = -.450$, $p = .004$), and CMAP amplitude ($r = -.359$, $p = .029$), as well as with the deep peroneal CV ($r = -.483$, $p = .003$), and CMAP amplitude ($r = -.396$, $p = .019$). In addition, the MCT composite score was positively correlated with the TNS ($r = .498$, $p = .001$) as well as negatively correlated with the MEP amplitude ($r = -.363$, $p = .021$).

Regarding the SOT composite score, it was significantly correlated with the posterior tibial CMAP amplitude ($r = -.346$, $p = .048$) and *F* wave minimal latency ($r = -.451$, $p = .008$). In addition, it was positively correlated with the MEP cortical latency ($r = .361$, $p = .0329$).

There was statistically significant correlation between the somatosensory ratio and posterior tibial nerve DL ($r = -.539$, $p = .000$), CV ($r = .354$, $p = .003$), CMAP amplitude ($r = .454$, $p = .021$), deep peroneal nerve CMAP amplitude ($r = .340$, $p = .04$), TNS ($r = -.372$, $p = .043$), as well as amplitude of the MEP response ($r = .328$, $p = .034$) and cortical latency ($r = -.330$, $p = .033$). Regarding the visual ratio, it was correlated with posterior tibial nerve DL ($r = -.382$, $p = .028$), NCV ($r = .346$, $p = .048$), CMAP

amplitude ($r = .354$, $p = .043$), *F* wave minimal latency ($r = -.449$, $p = .009$), as well as cortical latency of the MEP response ($r = -.371$, $p = .034$). No statistically significant correlations were found between the vestibular or visual preference ratio and any of the electrophysiologic variables.

Every subject with at least one altered posturographic parameter was considered to have impaired postural control. After recording every subject with impaired posture, logistic regression analysis was applied.

The logistic regression model explains only 45.9% of the occurrence of postural impairment. Only the peripheral neuropathy is significantly contributing to the model ($p = .016$) (Adjusted OR = 1.167, 95% CI = 1.030, 1.323). Accordingly, only peripheral neuropathy appeared to significantly contribute to the postural impairment in the patient group. Whereas peripheral neuropathy explains only 45.9% of the occurrence of postural impairment, it was found that the central factor was not an independent predictor of postural impairment ($p > .05$, i.e. $p = 0.78$).

As regards correlation of TNS and central factor with posturographic parameters TNS was positively correlated with somatosensory ratio ($r = .322$, $p = .031$) and MCT composite score ($r = .498$, 0.001). On the other hand, Central factor was positively correlated only with MCT composite score ($r = .314$, 0.048). Table 5

Regarding the 10 patients having no PN, correlation of the MEP parameters with dynamic posturographic variables

Table 4 Correlation of dynamic posturographic variables with selected electrophysiologic variables and TNS in patient group.

Variables	MCT composite score		SOT composite score		Somatosensory ratio		Visual ratio	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
<i>Posterior tibial nerve</i>								
• Latency (msec)	.410**	.009	-.361	.089	-.539**	.000	-.458**	.007
• CV (m/sec)	-.450**	.004	.270	.128	.354**	.003	.432*	.012
• CMAP amplitude (mV)	-.359*	.029	.346*	.048	.454*	.021	.382*	.028
• F wave latency	.498	.001	-.451**	.008	-.274	.079	-.449**	.009
<i>Deep peroneal nerve</i>								
• Latency (msec)	.268	.119	-.301	.675	-.282	.091	-.116	.557
• CV (m/sec)	-.483**	.003	.190	.334	.295	.077	.390*	.040
• CMAP amplitude (mV)	-.396*	.019	.115	.560	.340*	.04	.098	.621
Resting motor threshold(%)	.292	.067	.157	.383	-.158	.317	.343	.051
<i>Motor evoked potential</i>								
• Cortical latency (ms)	.308	.053	-.399*	.022	-.330*	.033	-.371*	.034
• Amplitude (mV)	-.363*	.021	.328	.063	.328*	.034	.097	.593
• CMCT	-.086	.598	.034	.849	.254	.105	.124	.492
Total neuropathy score	.498**	.001	-.103	.655	-.372*	.043	-.115	.620

msec = millisecond, SOT = Sensory organization test, m/sec = meter per second, MCT = motor control test, mV = milliVolt, CV = Conduction velocity, CMAP = Compound muscle action potential, CMCT = central motor conduction time, % = percentage, *r* = Spearman's rho correlation coefficient.

* Significant at $p \leq .05$ level.

** Significant at $p \leq .01$ level.

Table 5 Correlation matrix between central factor, total neuropathy score and posturographic parameters.

Variable	Central factor		Total neuropathy score	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
SOT composite score	-.076	.673	-.278	.100
Somatosensory ratio	-.067	.675	-.322*	.031
Visual ratio	-.253	.156	-.319	.058
Vestibular ratio	-.060	.739	-.114	.507
Visual preference ratio	-.101	.575	.070	.686
MCT composite score	.314*	.048	.498**	.001

MCT = motor control test, SOT = Sensory organization test, *r* = Spearman's rho correlation coefficient,

* Significant at $p \leq .05$ level (2-tailed).

** Significant at $p \leq .01$ level.

showed significant negative correlation between amplitude of the MEP response and MCT composite score ($r = -.658$, $p = .038$) Table 6.

5. Discussion

It is well known that gait characteristics and balance are altered in diabetic patients.⁴ Regarding the postural control of the diabetic patients in the present study, SOT analysis indicates an overall decrease in patients' performance and sensory function with specific affection of somatosensory and visual system.²⁹ This contributes significantly and negatively to their balance. Low condition 2 score and somatosensory ratio of patient group relative to the controls points to somatosensory impairment.²⁹ On the other hand, low condition 4 score and visual ratio of the patient group points to visual impairment that could be at the subclinical level. Many visual problems can appear soon in diabetics, even sooner than neuropathy.³⁰ A significant deterioration in color vision and/or contrast

sensitivity in diabetic patients without retinopathy compared to non diabetic controls has been documented.³¹ Lack of significant difference between patient and control groups regarding condition 3 and visual preference ratio signifies that low condition 6 scores are probably due to the difficulty in using the visual information by the patient group.¹⁸

MCT automatic response latencies showed significant prolongation in the patient group compared to the control group. The presence of prolonged MCT response latency translations as well as MCT composite score with normal MCT response strength and absence of asymmetry in weight bearing indicate a pathological deficit within the long loop pathway mediating automatic postural responses. The latter includes sensory and motor peripheral nerves, the ascending and descending motor pathways, and the motor regions of the brain and cerebral cortex. Besides, prolonged MCT response latency results for medium and large translations limited to one movement direction (i.e. backward translation) suggest that the lesion is more likely to be in the efferent branch of the long loop pathway.¹⁸

Table 6 Correlation of the motor evoked potential parameters of the 10 patients without PN with dynamic posturographic variables.

Variable	Cortical latency (msec)		Amplitude (mV)		Resting motor threshold (%)		CMCT (msec)	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
SOT composite score	-.600	.116	.123	.772	-.089	.853	-.494	.214
Somatosensory ratio	-.268	.453	.357	.312	.529	.116	-.463	.178
Visual ratio	-.543	.164	.145	.731	.103	.808	-.506	.201
Vestibular ratio	-.148	.726	.355	.388	.048	.909	-.427	.292
Visual preference ratio	-.147	.728	.172	.684	.596	.596	-.346	.402
MCT composite score	.019	.959	-.658*	.038	.470	.171	.297	.404

msec = millisecond, mV = millivolt, SOT = sensory organization test, MCT = motor control test, CMCT = central motor conduction time.

* Significant at $p \leq .05$, r = Spearman's rho correlation coefficient.

Current study showed that Hb A1c was not correlated with any of the dynamic posturographic variables. This was in agreement with several authors^{32,33} who reported a lack of this association. HbA1c can be interpreted as an average of the blood glucose present over the past 3–4 months.³⁴ It might not reflect cumulative hazardous effects of diabetes mellitus on postural control. This might explain the lack of this association. However, larger sample size might demonstrate such relation.

On the other hand, the consequences of chronic diabetes mellitus in the CNS are less known than diabetic PN and autonomic nervous system neuropathy.¹⁰ In diabetic patient group, prolonged CMCT could be due to central motor neuropathy. In addition, MEP amplitude was significantly lower than that of the control group. Taken together, the combined low amplitude and prolonged CMCT of the MEP response suggests the presence of central motor pathway affection, whose underlying pathology may be combined demyelination and loss of axons or neurons of the corticospinal tract.^{26,35,36} This indicates the presence of subclinical central motor changes in those patients as documented in many studies.^{37–40} In this context, the present results also revealed increased MEP resting motor threshold (RMS) on both sides in the studied patients reflecting decreased excitability of the cortical and/or spinal motor neurons.^{35,37,38}

In the present work, age and disease duration were not correlated with most of the studied variables of the MEPs response and the dynamic posturography indicating that abnormalities of central motor pathway and postural control might be related to other disease variables rather than these. In support of this view, cortical latency of the MEP response and CMCT were positively correlated with glycosylated hemoglobin. This indicates that central affection might be related to the degree of glycemic control which agrees with many authors^{37–40} who reported that CMCT delay in diabetic patients was related to the degree of metabolic control. Our results were in agreement with those of Sabry et al.¹⁰ who found no correlation between CMCT delay and patients' age or disease duration. However, contrary to the present findings, the studies conducted by Imam et al.³⁸ demonstrated a statistically significant positive correlation between CMCT delay and both the age of type 2 diabetic patients and the duration of diabetes. The alteration of the central motor pathways supports the hypothesis that the central nervous system involvement in DM represents a process that is possibly partially dependent on degree of metabolic control as well as other factors. These factors were suggested to include enhanced flux through the polyol pathway, accelerated formation of

advanced glycation end products, oxidative damage and microvascular changes and alterations of the cerebral vascular system.^{41,42}

Most of the posturographic parameters correlated well with the majority of the peripheral conduction study parameters (supported by logistic regression analysis) whereas the MEPs study showed few correlations with these posturographic parameters. Therefore, it seems that PN is the main cause of the postural instability in the studied patients. In agreement with this result, Di Nardo et al.²¹ suggested that the main cause of postural instability in diabetes was PN rather than lesions of the spinal cord. Similarly, Emam et al.⁴³ suggested that PN is the main cause of postural impairment in diabetic patients. The mechanisms by which PN leads to postural instability are complex including the lack of accurate proprioceptive feedback from the lower limbs,⁴⁴ impairment of ankle strength, balance recovery and walking stability in diabetics.⁴³

Despite numerous studies on different aspects of CNS affection in diabetic patients, yet little work was reported on its relationship to postural impairment in such patients. In the current study, it was found that PN, assessed by total neuropathy score, has the primary influence on the postural control. However, the presence of significant negative correlation between the amplitude of the MEP response and the MCT composite score in the diabetic patients without peripheral neuropathy (decreased MEP amplitude is correlated with increased MCT composite score and vice versa) indicates that central motor changes could be a possible cause of postural instability in these patients together with visual and somatosensory dysfunction (as proved by SOT results).

It can be concluded that alteration in the peripheral nerve function was the main factor implicated in postural instability in the studied patients. However, the central motor pathway changes documented could be implicated as a possible cause.

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