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Do Motor Evoked Potentials Allow Quantitative Assessment of Motor Function in Patients with Spinal Cord Lesions?

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Summary. Motor evoked potentials (MEP) were recorded in a total of 110 patients with tumorous ($n = 39$) and non-tumorous ($n = 71$) lesions of the cervical ($n = 59$), thoracic ($n = 37$) and thoracolumbar ($n = 14$) spinal cord. In all cases MEP were elicited by electrical stimulations, and in 50 of them also by magnetoelectric stimulation, of the motor cortex. The peripheral conduction time was determined by electrical stimulation of the lumbar nerve roots.

It was the aim of this study to determine whether 1. MEP are sensitive for detection of lesions along the spinal cord and 2. whether they allow quantitative assessment of motor function. To achieve this goal, we compared potential and clinical findings of our patients, each divided into seven categories. Our results clearly showed the high sensitivity of MEP for semi-quantitative evaluation of motor function, as there were no false negative results in our series. Moreover, unilaterally accentuated motor deficits correlated significantly with changes in MEP, which were affected more strongly on the corresponding side (Student's t-test, $\alpha = 0.05$). However, clinical and electrophysiological findings did not correlate in the quantitative evaluation of the motor status as established by variance analysis ($F = 0.52$). There was no difference in results with respect to the electrical and magnetoelectric stimulation technique. Our results lead to the following conclusions. MEP are sensitive for semi-quantitative evaluation of spinal motor function; however, MEP do not allow quantification of the clinical motor status. We hypothesize that MEP changes are related more closely to the type of spinal cord damage (demyelination vs axonotmesis) and the duration of disease than to the actual clinical motor function.

Key words: Motor evoked potential – Spinal cord lesion

Introduction

Transcranial electrical and magnetoelectric stimulation of the motor cortex and subsequent recording of the electromyographic responses (motor evoked potientials, MEP) allow noninvasive electrophysiological assessment of the descending pathways [1, 6]. As has been shown previously both in animal experiments with spinal cord trauma and ischaemia as well as in humans, MEP have proven to be a valuable diagnostic tool for detection of lesions along the spinal cord [2, 3, 5, 7–12]. However, reports addressing the significance of MEP for quantitative evaluation of spinal function are still lacking. Therefore, this study was designed to assess whether MEP elicited by both electrical and magnetoelectric stimulation are not only suitable for detection of spinal cord lesions, but also whether they allow quantitative assessment of the actual motor function. To achieve this goal, we correlated the clinical with the electrophysiological findings in 110 patients who had various lesions along the spinal cord.

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Patients and Methods

A total of 110 patients (68 males, 42 females) with ages ranging from 28 to 83 years (mean age 59 years) were included in this study. The lesion was tumorous in 39 and non-tumorous in 71 patients. Localisation of disease was cervical in 59, thoracic in 37 and thoracolumbar in 14 cases. In all 110 patients, MEP were elicited by electrical, in 50 of them additionally by magnetoelectric stimulation of the motor cortex, while the peripheral conduction time was determined by electrical stimulation of the lumbar nerve roots.

Electrical stimulation was performed with voltage constant condenser discharges using a Digitimer D 180 which delivers a maximum output of 750 V with a time constant of 50 or 100 µs. For stimulation of the motor leg area the anode was placed over the bregma and the cathode 6 cm behind it in the midline. When the lumbar nerve roots were stimulated, the anode was positioned at the intervertebral space of D11–12 and the cathode one segment below, each in the midline. Magnetoelectric excitation of the motor leg area was achieved by positioning the coil over Fz with the current running clockwise for activation of the right side and counterclockwise for the left side (Magstim 200, 1.5 T).

Electromyographic responses were recorded from the anterior tibial muscle using EMG-electrodes in a belly/tendon-fashion. While electrically evoked responses were recorded with the target muscle at rest, magnetoelectrically elicited potentials were facilitated by voluntary contraction of the target muscle with about 10% of maximum muscle strength. This was controlled by audio-EMG. Stimulus strength was gradually increased until a clear EMG re-

CMCT / Category	Stimulation Electrical msec.	Magnetoelectric msec.
0	< 14.0	< 16.3
1	14.1 - 15.0	16.4 - 17.0
2	15.1 - 20.0	17.1 - 22.0
3	20.1 - 25.0	22.1 - 27.0
4	25.1 - 30.0	27.1 - 32.0
5	> 30	> 32
6	absent responses	

Fig. 1. Classification of MEP findings

Category	Clinical Examination
0	normal motor status
1	unremarkable gait, slightly elevated muscle tone
2	spastic gait, able to walk without support, sinking of legs
3	able to walk with support, legs can be lifted from surface
4	able to stand with support, legs cannot be lifted against gravity
5	minimal movements, frustrated contractions
6	completely paralyzed

Fig. 2. Classification of clinical findings

sponse was obtained or the absence of any response was documented despite a stimulus strength as great as could be tolerated by the individual patient. The time base was 100 µs with a gain ranging from 100 µV to 1 mV per division. Filter settings ranged from 20 Hz to 3 kHz. Only single stimuli were applied. The time interval between the stimuli was 3–5 s. At least 3 potentials were obtained from recording site. The central motor conduction time (CMCT) was determined by substraction of the peripheral latency from the total latency (electrophysiological system Compact 4, Nicolet).

We decided upon the following criteria for pathological MEP changes: Absence of any response on one or both sides, CMCT cortex-D12 > 14 ms (electrical stimulation) or > 16.3 ms (magnetoelectric stimlulation). These limits for acceptable CMCT result from our normal data plus 2.5 SD as obtained from 50 healthy volunteers.

To determine the overall sensitivity of MEP for detection of lesions along the decending pathways regardless of their extent, we considered normal potentials in patients with an unremarkable motor status and pathological potentials in patients with a clinical motor deficit as a correct correlation. False positive correlation means pathological MEP findings despite an unremarkable motor status. Correlation was considered to be false negative in patients with a clinical motor deficit and normal MEP.

In order to determine whether unilateral clinical accentuation correlates with MEP, we compared side differences in CMCT with the clinical findings. Statistical analysis was performed by calculating the mean side differences in CMCT and standard deviations for patients with and without unilateral accentuation of motor deficits using the Student's *t*-test.

For quantitative correlation of electrophysiological and clinical findings, MEP and the results of neurological examination were divided into 7 categories, respectively. Category 0 of MEP means normal latencies and category 6 absence of any responses. MEP category 1 represents a borderline increase in CMCT of up to 1 ms, while categories 2–4 comprise patients with increase of CMCT in steps of 5 ms. Category 5 of MEP represents patients with a CMCT of more than 30 ms (electrical) and 32 ms (magnetoelectric) (Fig. 1). Correspondingly, the clinical motor status was divided into 7 categories as defined by the extent of the motor deficit. Categories ranged from normal motor status to complete paralysis (Fig. 2). Based on these categories, MEP were correlated with the clinical motor status. Statistical evaluation was performed by means of a one-way analysis of variance.

Results

In all patients, distinct responses were obtained following stimulation of the lumbar nerve roots. Comparing MEP findings with the clinical motor status in terms of semi-quantitative evaluation, there were no false negative results either for electrical or magnetoelectric stimulation. Following electrical stimulation, 99 patients (90%) showed a correct and 11 (10%) a false positive correlation. Magnetoelectric stimulation provided correct results in 43 (86%) and false positive ones in 7 (14%) cases (Fig. 3). All patients with a false positive correlation showed radiological evidence of myelomalacia.

Clinically, both sides were equally affected in 67 patients while motor deficits were unilaterally pronounced in 43 cases. In patients with symmetrical motor deficits, differences in CMCT right/left in response to electrical stimulation ranged from 0 to 5.6 ms (mean 2.7 ms, SD 2.1 ms). Corresponding values for magnetoelectric stimulation were 0.4 and 3.2 ms, respectively (mean 1.4 ms, SD 1.1 ms). In all of the 43 patients with unilaterally pronounced motor deficits, MEP changes were more severe on the clinically more affected side. With electrical stimulation, differences in CMCT ranged from 2.6 to 13.0 ms (mean 4.3 ms, SD 2.8 ms), and from 1.2 ms to 14.2 ms (mean 4.2 ms, SD 2.1 ms) with magnetoelectric stimulation. These results were highly significant (Student's *t*-test; $\alpha = 0.05$; Fig. 4). An illustrative case is shown in Fig. 5. Statistical evaluation of our clinical and electrophysiological results, each divided into 7 catego-

Correlation / Stimulation	Correct N	Correct %	False positive N	False positive %	False negative N	False negative %	Total N
Electrical	99	90.0	11	10.0	-	-	110
Magnetoelectric	43	86.0	7	14.0	-	-	50

Fig. 3. Correlation of MEP with the clinical motor status (semi-quantitative evaluation). Results of 110 patients are shown

	MOTOR DEFICIT					
CMCT (msec) / Stimulation	Unilaterally Accentuated N = 43			Symmetrical N = 67		
	Mean	Range	S. D.	Mean	Range	S. D.
Electrical	4.3	2.6 - 13.0	2.8	2.7	0 - 5.6	2.1
Magnetoelectric	4.2	1.2 - 14.2	2.1	1.4	0.4 - 3.2	1.1

Fig. 4. Central motor conduction time (CMCT) in patients with unilaterally ($n = 43$) and symmetrical ($n = 67$) motor deficits

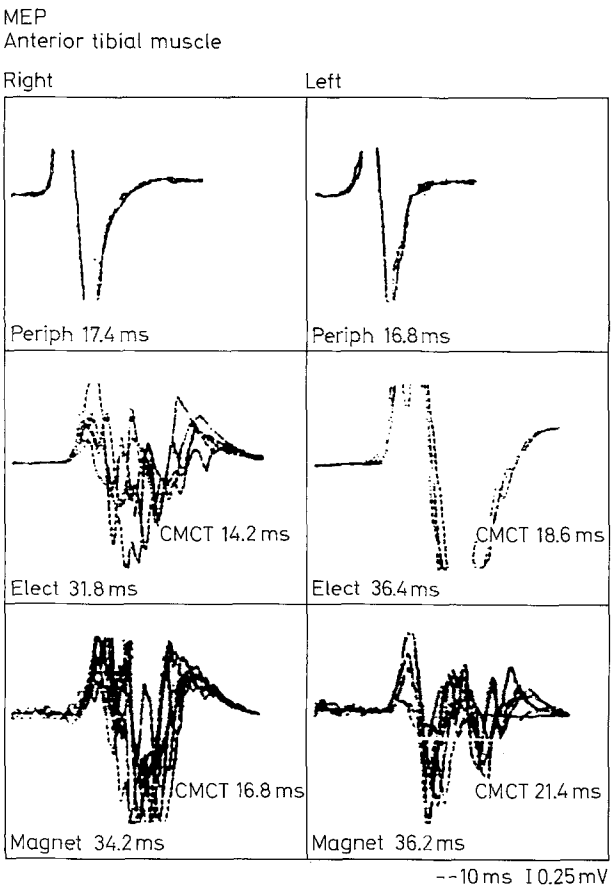

Fig. 5. A 62-year-old male patient with a hard disc at C5–6. Clinically, he presented with a spastic paresis of the left leg. The right side was normal. MEP of the right anterior tibial muscle showed a borderline increase in CMCT, while latencies were noticeably prolonged on the left side

	M0	M1	M2	M3	M4	M5	M6	Σ
C0	[14]	2	5	3	1			25
C1		[3]	3	4	2	1		13
C2		1	[3]	5	3	1		13
C3		1	3	[4]	6			14
C4		2	3	3	[2]	2		12
C5		1	4	2	2	[2]	4	15
C6							[18]	18
Σ	14	10	21	21	16	6	22	

Fig. 6. Correlation of clinical (C0–C6) and MEP (M0–M6) categories in 110 patients (quantitative evaluation). MEP were elicited by electrical stimulation of motor cortex

ries (Figs. 6 and 7), by means of a one-way analysis of variance shows that there was no correlation between the extent of the clinical motor deficit and the prolongation of CMCT ($F = 0.42$). Only the absence of electromyographic responses usually correlated with complete paralysis. These results were valid both for electrical and magnetoelectric stimulation.

	M0	M1	M2	M3	M4	M5	M6	Σ
C0	[6]	2	2	3				13
C1		[ ]	3		2			5
C2		1	[1]	2	1			5
C3				[3]	2	1		6
C4			2	1	[1]	2	1	7
C5			1			[ ]	2	3
C6							[11]	11
Σ	6	3	9	9	6	3	14	

Fig. 7. Correlation of clinical (C0–C6) and MEP (M0–M6) categories in 50 patients (quantitative evaluation). MEP were elicited by magnetoelectric stimulation of the motor cortex

Discussion

Our results demonstrate the high sensitivity of MEP for detection of lesions along the descending motor pathways. Pathological MEP findings were observed in all patients with motor deficits as established by clinical examination. We encountered no false negative findings in our series. The high rate of false positive results (10% with electrical and 14% with magnetoelectric stimulation) is remarkable. In our opinion, these results are rather "true" than "false positive," indicating that MEP are able to detect subclinical lesions. This is strongly supported by the fact that all of our patients showing "false positive" results had radiological evidence of myelomalacia. Therefore, MEP seem to be a valuable diagnostic tool for detection of lesions along the descending pathways, especially when clinical examination shows ambiguous results.

Comparing prolongation of the CMCT right/left we found differences of up to 5.6 ms (electrical stimulation) and 3.2 ms (magnetoelectric stimulation) in patients without clinical evidence of unilaterally accentuated deficits. However, statistically analysis of side differences of CMCT in patients with and without unilaterally accentuated motor deficits was highly significant ($\alpha = 0.05$; Student's t-test). Thus, changes in MEP are usually more severe on the clinically more affected side.

The main question of this study was whether MEP allow quantification of the clinical motor deficit. According to our results, this is not the case. We did not observe significant correlation between the extent of the clinical deficit and the prolongation of CMCT as documented by statistical analysis. Only complete paralysis usually correlated with the absence of electromyelographic responses. On the other hand, absence of MEP does not necessarily indicate complete paralysis in every case, since this was also observed inpatients showing residual motor function. Here we found the only difference between results obtained with the electrical and magnetoelectric stimulation techniques. With electrical stimulation, MEP were overwhelmingly pathological due to prolonged CMCT, while responses were absent in only a few patients. With magnetoelectric stimulation, the rate of absent responses was much higher.

One may speculate whether evaluation of differences in amplitudes right/left could contribute to a more precise assessment of motor deficits. However, it is well known that MEP amplitudes are very variable and depend on the degree of preinnervation [4]. Thus, evaluation of amplitudes is difficult even in the same individual and may not be possible at all comparing different patients. For these reasons, amplitudes of potentials were not evaluated in our patients.

In conclusion, examination of the descending pathways by means of MEP is a useful test for electrophysiological assessment of lesions along the spinal cord. Pathological MEP indicate damage even in cases when clinical examination produces ambiguous results. Moreover, MEP may indicate the clinically more affected side. However, MEP fail to provide quantification of the clinical motor deficit, since there is no linear correlation between the clinical motor status and the electrophysiological changes. In our opinion, changes in MEP may be related more closely to the type of spinal cord damage, e.g. demyelination vs. axonotmesis, and the duration of the disease than to the actual clinical motor function. However, further studies are necessary to more clearly define the essential factors influencing latencies of motor responses and to provide evidence whether additional evaluation of other variables, e.g. configuration and/or amplitudes of the potentials, could attribute to a more precise correlation between clinical and electrophysiological findings.

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