Eur Arch Psychiatr Neurol Sci (1990) 239:285–289

European Archives of Psychiatry and Neurological Sciences

Diagnostic Significance of Motor Evoked Potentials in Space-occupying Lesions of the Brain Stem and Spinal Cord

J. Zentner and G. Rieder

Department of Neurosurgery, Medical School, University of Tübingen, D-7400 Tübingen, Federal Republic of Germany

Received February 13, 1989

Summary. Motor evoked potentials (MEP) were examined in 50 patients with space-occupying lesions of the brain stem and spinal cord. MEP findings were correlated with the motor status as established by clinical examination. The results clearly show the high sensitivity of MEP for detection of motor deficits: 17 recordings (77%) from the thenar muscle and 42 (84%) from the anterior tibial muscle correlated correctly with the clinical motor status. False-positive results were found in 5 (23%) thenar recordings and 8 (16%) and anterior tibial recordings. False-negative correlation was not observed. The high rate of false-positive results appears to indicate that MEP detect subclinical motor deficits. This electrophysiological test is therefore recommended, especially when involvement of the descending pathways is suspected and clinical examination reveals no abnormality.

Key words: Motor evoked potential– Brain stem lesion – Spinal cord lesion

Introduction

Non-invasive assessment of the descending pathways by means of transcranial excitation of the motor cortex and recording the electromyographic response has been possible since 1980 using electrical stimulation [7] and since 1985 using magnetic stimulation [1]. So far there have been reports of motor evoked potentials (MEP) in electrophysiological evaluation of demyelinating diseases and other neurological disorders [3, 8, 11, 13] as well as in intraoperative monitoring during orthopaedic [2] and neurosurgical [6] operations on the spinal cord. In this study MEP were examined in patients with space-occupying lesions of the brain stem and spinal cord. The aim has to determine if involvement of the descending pathways can be reliably ascertained with this electrophysiological test.

Offprint requests to: J. Zentner, Abteilung für Neurochirurgie, Calwerstrasse 7, D-7400 Tübingen, Federal Republic of Germany

Patients and Methods

A total of 50 patients (34 males, 16 females) aged between 19 and 81 years (mean 56) were examined. Tables 1–3 show the diagnoses, localization of disease, and the clinical presentation of the patients. Twenty-one patients (42%) had an extradural, 15 (30%) an intradural/extramedullary and 14 (28%) an intramedullary space-occupying lesion. The lesion was neoplastic in 38 and non-neoplastic in 12 cases.

The motor cortex was stimulated transcranially with voltage-constant condenser discharges using a Digitimer D 180. The time constant of the discharges was 100 µs. Only single stimuli of 250–750 V were applied. The time interval between the stimuli was at least 3 s. For stimulation of the motor hand area the anode was placed at the foot of the precentral gyrus (8 cm from the midline, just behind the coronary suture) and the cathode was positioned at the bregma. For motor foot area stimulation, the anode was placed at the bregma and the cathode 6 cm behind that at the midline. Electromyographic (EMG) responses were recorded from the contralateral thenar and anterior tibial muscle using EMG electrodes in a belly/tendon fashion. 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 (electrophysiological system Compact 4, Nicolet).

Table 1. Diagnoses

Diagnosis	Patients	
	n	%
Metastasis	11	22.0
Neurinoma	8	16.0
Meningioma	6	12.0
Cavernoma	5	10.0
Ependymoma	4	8.0
Astrocytoma	3	6.0
Chordoma	1	2.0
Syringomyelia	2	4.0
Arteriovenous fistula	1	2.0
"Hard" disc	7	14.0
"Soft" disc	2	4.0
Total	50	100.0

Table 2. Localization of disease

Localization	Patients	
	n	%
Brain stem	7	14.0
Cervical spine	24	48.0
Thoracic spine	19	38.0
Total	50	100.0

Table 3. Clinical presentation of investigated patients

Clinical presentation	Patients	
	n	%
No motor deficit	9	18.0
Hemiparesis	5	10.0
Paraparesis	17	34.0
Tetraparesis	19	38.0
Total	50	100.0

In every patient in whom thenar muscle responses were recorded, the lower cervical spine was additionally stimulated with the cathode placed at the level of C6-7 in the midline and the anode 6 cm homolateral to the recording site at the same level. Similarly, in every patient in whom responses were recorded from the anterior tibial muscle, the conus medullaris was also stimulated with the anode placed at the level of D11-12 and the cathode at D12-L1, each in the midline. This was done in order to assess the integrity of peripheral motor pathways. Furthermore, by subtracting the latency of the potential following lower cervical and conus medullaris stimulation from the corresponding values obtained in response to transcranial stimulation, the central motor conduction time (CCT) cortex–C6-7 and cortex–conus medullaris, respectively, was evaluated.

Only patients with an intact peripheral motor pathway showing distinct potentials following lower cervical and conus medullaris stimulation, respectively, were considered. The stimulus strength applied transcranially was gradually increased until a response was obtained or the absence of any response was documented, even though a maximum stimulus strength of up to 750 V, as accepted by the patient, was applied.

We decided upon the following criteria for pathological MEP changes: absence of any response on one or both sides, CCT cortex–C6-7 greater than 5.9 ms and CCT cortex–conus medullaris greater than 13.9 ms. These limits for acceptable latencies of CCT result from our normative data including 2.5 SD.

On the basis of these criteria for pathological MEP changes we correlated MEP findings with the motor status as established by clinical examination. Correct correlation means either normal MEP and normal motor status or pathological MEP and motor deficit. The correlation was considered false positive when MEP showed pathological changes despite a normal motor status, and false negative in patients with normal MEP findings despite a motor deficit.

Results

Nineteen of the 22 (86%) recordings from the thenar muscle and 41 of the 50 (82%) form the anterior tibial muscle showed pathological results. In 2 (9%) thenar and 5 (10%) anterior tibial muscle cases the EMG response was absent on at least one side. MEP were considered pathological because of a prolonged CCT in 17 recordings (77%) from the thenar muscle, and in 36 (72%) from the anterior tibial muscle. On the whole, 60 of 72 recordings (83%) were pathological, in 7 cases (10) owing to absent responses and in 53 cases (73%) owing to a prolonged CCT (Table 4).

Regarding correlation of MEP findings with the motor status as established by clinical examination, we found a correct correlation in 17 of the 22 recordings (77%) from the thenar muscle and in 42 of the 50 (84%) from the anterior tibial muscle. False-positive results

Table 4. Pathological motor evoked potential (MEP) findings

Criterium	Recording site				Total ($n = 72$)	
	Thenar muscle ($n = 22$)		Anterior tibial muscle ($n = 50$)			
	n	%	*n*	%	*n*	%
Absent response	2	9.1	5	10.0	7	9.7
CCT cortex–C6–7 > 5.9 ms; CCT cortex–conus medullaris > 13.9 ms	17	77.3	36	72.0	53	73.6
Total	19	86.4	41	82.0	60	83.3

Table 5. Correlation of MEP findings with the clinical motor status

Recording site	Correct		False positive		False negative		Total	
	n	%	*n*	%	*n*	%	*n*	%
Thenar muscle	17	77.3	5	22.7	–	–	22	100.0
Anterior tibial muscle	42	84.0	8	16.0	–	–	50	100.0
Total	59	81.9	13	18.1	–	–	72	100.0

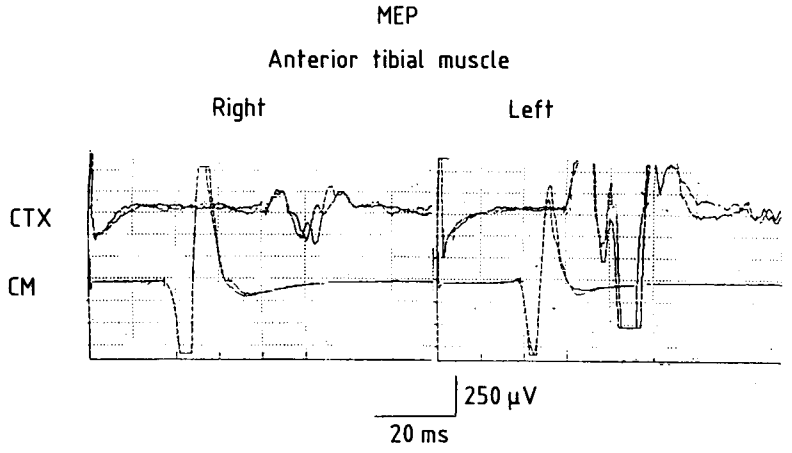

Fig. 1. A 61-year-old male patient with a soft disc at C5–6 on the right side. Clinical examination revealed mild paresis of the right leg. Central conduction time (CCT) was normal on the left side (10.2 ms) but noticeably prolonged on the right (23.6 ms). Note also differences in amplitudes right/left using the same stimulus strength of 450 V. Correlation of motor evoked potentials (MEP) with the motor status was considered to be correct. *CTX,* Stimulation of the cortex (transcranially); *CM,* stimulation of the conus medullaris

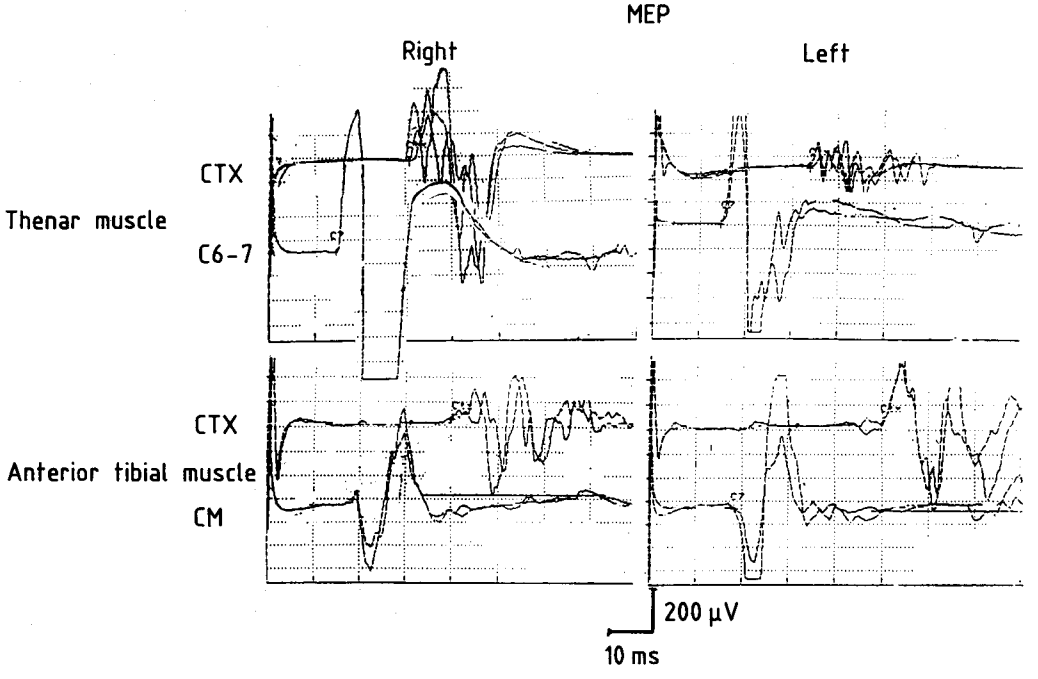

Fig. 2. A 78-year-old male patient with a metastasis at C3–4. He had a marked tetraparesis pronounced on the left side but was able to walk some steps with intensive support. CCT of the thenar muscle MEP was prolonged to 16.8 ms *(right)* and 19.4 ms *(left)*. CCT of anterior tibial muscle MEP was 21.3 ms on the right and 31.4 ms on the left side. Correlation of MEP with the motor status was considered to be correct

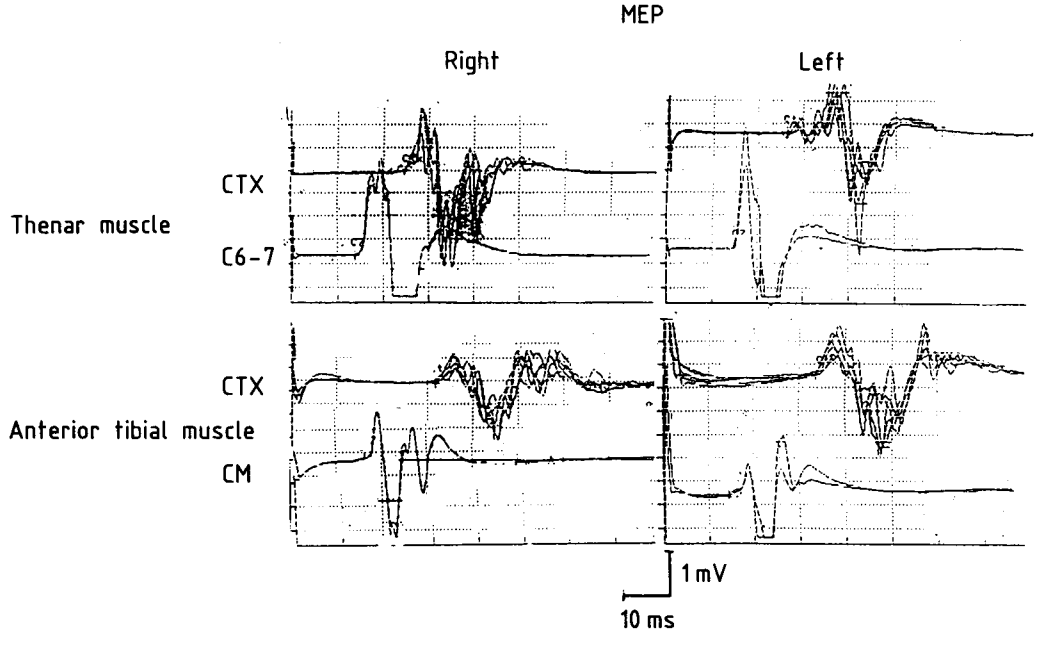

Fig. 3. A 50-year-old male patient with a hard disc at C4–5. Clinical examination revealed a mild paraparesis of the lower extremities pronounced on the left side. He was able to walk without support. CCT of thenar muscle MEP was prolonged to 10.8 ms on the right and to 12.0 ms on the left side. CCT of the anterior tibial muscle MEP was 15.4 ms *(right)* and 19.2 ms *(left)*. Correlation of MEP findings with the motor status was considered to be correct for recording form the anterior tibial muscle but "false" positive for thenar muscle potentials

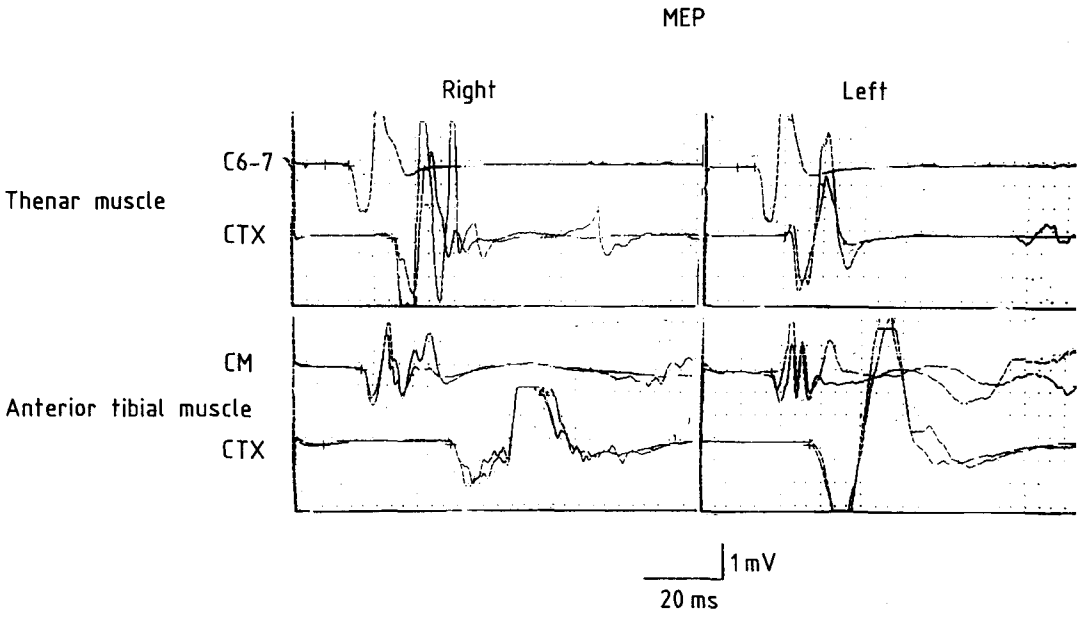

Fig. 4. A 25-year-old female patient with an astrocytoma of the right medulla oblongata (histologically verified). Clinical examination revealed an incomplete paresis of the 6th cranial nerve on the right side. There were no motor deficits of the extremities. CCT cortex–C6-7 was prolonged to 11 ms on the right and to 8 ms on the left side. CCT cortex–conus medullaris was normal on the left (9.8 ms) but noticeably prolonged on the right (22 ms). Correlation of MEP findings with the motor status was considered to be "false" positive. Note also differences in amplitudes right/left of anterior tibial muscle MEP using the same stimulus strength of 350 V

were observed in 5 (23%) thenar and in 8 (16%) anterior tibial muscle cases. We did not find any false-negative correlation. On the whole, 59 (82%) recordings showed a correct and 13 (18%) a false-positive correlation (Table 5; Figs. 1–4).

Discussion

Our results demonstrate the high sensitivity of MEP for electrophysiological measurement of disorders of the central motor pathways. Pathological MEP findings were observed in all patients with motor deficits as established by clinical examination. We encountered no false-negative correlation in our series. It also seems remarkable that pathological MEP changes were overwhelmingly due to prolonged central latencies and only minimally due to absent responses. On the whole, our results correspond with those of others who have found a close correlation between MEP and the motor status in animal experiments on traumatic and ischaemic brain and spinal cord lesions [4, 9, 10, 12].

The high rate of false-positive results (23% when recording from the thenar muscles and 16% when recording from the anterior tibial muscle) seems remarkable. This may result in part from inadequate evaluation criteria for pathological MEP changes. However, as some of our false-positive cases had distinct lesions unavoidably affecting descending pathways, as shown by neuroradiological investigation, we believe that MEP are "true" rather than "false" positives in these cases, indicating that MEP not only provide objective evidence of existing motor deficits but also assessment of subclinical deficits of the descending pathways. The latter would be sufficient motivation to use MEP for diagnostic reasons: obviously it is unnecessary to study MEP when affection of the descending pathways is already established by clinical examination. However, it might be very useful for therapeutic decision making, e.g. surgical procedure, if by means of MEP involvement of the motor system is either proven or ruled out in patients with radiologically confirmed lesions affecting the descending pathways but with no apparent clinical motor loss.

We did not observe any correlation between the degree of pathological MEP changes, e.g. prolonged CCT, and the degree of motor deficit. Some patients with minor motor deficits showed considerably prolonged CCT and vice versa. However, in patients with differences in motor deficits comparing right/left, we usually observed noticeable differences in amplitudes between the sides using the same stimulus strength. The more motor deficits differed right/left, the more amplitudes right/left also differed. One may speculate whether quantification of motor deficits would be possible if amplitudes were included as evaluation criteria for pathological MEP changes in addition to latencies. However, it is well known that MEP amplitudes are very variable and depend on the degree of pre-innervation [5]. Thus, evaluation of amplitudes is difficult even in the same individual and may not be possible at all by comparing different individuals.

To conclude, examination of the descending pathways with MEP is a useful test for electrophysiological assessment of space-occupying lesions of the brain stem and spinal cord. We recommend MEP investigation especially when involvement of the motor system is suspected and clinical examination reveals no abnormality, in order to demonstrate subclinical deficits. Owing to the

relatively unpleasant character of electrical stimulation, the practicability of the magnetic stimulation procedure [1] should be considered for such cases.

References

1. Barker AT, Jalinous R, Freeston IL (1985) Non-invasive magnetic stimulation of the human motor cortex. Lancet I: 1106–1107
2. Boyd SG, Rothwell JC, Cowan JMA, Webb PJ, Morley T (1986) A method of monitoring function in corticospinal pathways during scoliosis surgery with a note on motor conduction velocities. J Neurol Neurosurg Psychiatry 49:251–257
3. Cowan JMA, Dick JPR, Day BL, Rothwell JC, Thompson PD, Marsden CD (1984) Abnormalities in central motor pathway conduction in multiple sclerosis. Lancet II:304–307
4. Fehlings MG, Tator CH, Linden RD, Piper IR (1987) Motor evoked potentials recorded from normal and spinal cord-injured rats. Neurosurgery 20:125–130
5. Hess CW, Mills KR, Murray NMF (1986) Magnetic stimulation of the human brain: facilitation of motor responses by voluntary contraction of ipsilateral and contralateral muscles with additional observations on an amputee. Neurosci Lett 71:235–240
6. Levy WJ (1987) Clinical experience with motor and cerebellar evoked potential monitoring. Neurosurgery 20:169–182
7. Merton PA, Morton HB (1980) Stimulation of the cerebral cortex in the intact human subject. Nature 285:227
8. Mills KR, Murray NMF (1985) Corticospinal tract conduction time in multiple sclerosis. Ann Neurol 18:601–605
9. Oro J, Levy WJ (1987) Motor evoked potential as a monitor of middle cerebral artery ischemia and stroke. Neurosurgery 20: 192–193
10. Patil AA, Nagaray MP, Mehta R (1985) Cortically evoked motor action potentials in spinal cord injury research. Neurosurgery 16:473–476
11. Rossini PM, Caramia MD, Zarola F (1987) Mechanisms of nervous propagation along central motor pathways: noninvasive evaluation in healthy subjects and in patients with neurological disease. Neurosurgery 20:183–191
12. Simpson RK, Baskin DS (1987) Corticomotor evoked potentials in acute and chronic blunt spinal cord injury in the rat: correlation with neurological outcome and histological damage. Neurosurgery 20:131–137
13. Snooks SJ, Swash M (1985) Motor conduction velocity in the human spinal cord: slowed conduction in multiple sclerosis and radiation myelopathy. J Neurol Neurosurg Psychiatry 48: 1135–1139