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The value of somatosensory and motor evoked potentials in pre-clinical spondylotic cervical cord compression

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Abstract Previous studies have yielded conflicting data concerning the value of evoked potential parameters in the assessment of clinical relevance of cervical cord compression in clinically “silent” cases. The aim of this study was to assess the value of somatosensory (SEP) and motor evoked potentials (MEP) in the evaluation and prediction of the clinical course, by means of a 2-year follow-up prospective electrophysiological and clinical study performed in patients with clinically “silent” spondylotic cervical cord compression. Thirty patients with MR signs of spondylotic cervical cord compression but without clinical signs of myelopathy were evaluated clinically and using SEPs and MEPs during a 2-year period. The results of the

study showed that SEPs and MEPs documented subclinical involvement of cervical cord in 50% of patients with clinically “silent” spondylotic cervical cord compression. During the 2-year period clinical signs of cervical myelopathy were observed in one-third of patients with entry EP abnormality in comparison with no patients with normal EP tests. Combined SEPs and MEPs proved to be a valuable tool in the assessment of the functional relevance of subclinical spondylotic cervical cord compression. Normal EP findings predict a favourable 2-year clinical outcome.

Key words Spondylotic cervical cord compression · Somatosensory evoked potentials · Motor evoked potentials

Introduction

The treatment of cervical myelopathy is contentious [10, 22]. There is no common agreement on the timing of surgery and on the selection of patients for surgical therapy. On one side is the argument that patients with spondylotic cervical cord compression and clinical signs of mild myelopathy or even without any clinical deficit could profit from early decompression. The rationale for this attitude is based on assumption that mild, ephemeral or even subclinical lesions would be prone to reversal, while severe and long-lasting clinical deficit could not be expected to subside after operation. On the other side, there is a lack of clear-cut evidence to show that surgical de-

compression in patients with mild and stable clinical deficits is preferable to conservative treatment.

Clinical scales used are insensitive and subject to observer bias, while MR defines anatomical lesions, but cannot give information about the functional cervical cord damage. In clinically “silent” spondylotic cervical cord compression, the questions arise as to whether this compression causes any functional cervical cord involvement in the individual patient, and whether this involvement predicts unfavourable clinical course and thus justifies early surgical decompression before clinical manifestation of any functional deficit.

Both somatosensory evoked potentials (SEPs) and motor evoked potentials (MEPs) have been reported to be sensitive methods not only in the detection of the presence

of spinal cord involvement in spondylotic cervical myelopathy (SCM) patients, but also in the evaluation of the level of compression and the degree of spinal involvement [2, 5–7, 14, 15, 19–21, 23]. There are, however, few and discrepant data documenting the value of SEPs and MEPs in the evaluation of patients with clinically “silent” cervical stenosis or with MR-documented spondylotic cervical cord compression, as well as in the prediction of further clinical course in these patients.

The goals of the present study were:

1. To assess the sensitivity of SEPs and MEPs in clinically “silent” spondylotic cervical cord patients
2. To correlate the EP findings with the 2-year clinical outcome

Materials and methods

Experimental design

A 2-year follow-up prospective electrophysiological and clinical study was performed. All EP records were evaluated by one investigator (J. B.) who was “blind” to the clinical status of the patients. Ethical approval for the study was granted by the Ethical Committee of the Faculty Hospital, Brno.

Population

Control group

To establish the normal values and limits of SEPs and MEPs we examined 40 normal subjects (21 men and 19 women, mean age 49.3 ± 7.3 years, range 29–69 years). They were free of clinical symptoms and signs of either central or peripheral nervous system involvement including symptoms and signs of radiculopathy or myelopathy. The group was not systematically screened for the signs of spondylosis.

Study sample

The study sample consisted of 30 subjects recruited consecutively from a total of 285 patients admitted to the department of neurology between 1993 and 1996 with various clinical signs and symptoms of suspected vertebral origin (signs and/or symptoms of cervical myelopathy, radiculopathy and/or pain in the neck or shoulder region). The subjects were free of clinical signs and symptoms of spondylogenic cervical myelopathy at time of admission, but were at high risk of developing symptomatic cervical myelopathy. They met the following inclusion criteria:

1. Signs of cervical spondylosis in plain radiograms at the level of at least two discs and adjacent vertebral bodies
2. Pavlov’s ratio [18] below 0.8 as a sign of congenital stenosis and/or anteroposterior cervical canal diameter below 12 mm as a sign of acquired degenerative stenosis
3. MR criteria for cervical cord compression and/or myelopathy (see below)
4. Absence of any current unequivocal clinical signs that could be attributed to cervical cord involvement

The spectrum of clinical symptoms and/or signs at admission were as follows:

1. Pain in the cervical, shoulder and/or scapular region (10 patients)

2. Cervical pain plus Lhermitte’s sign (2 patients)
3. Sensory and/or motor symptoms and/or signs in the upper extremity (6 patients)
4. Cervical pain with sensory and/or motor symptoms and/or signs in the upper extremity (12 patients)

In all included patients the objective sensory and/or motor signs were classified as radicular; they fulfilled the following criteria:

1. Typical sensory radicular symptoms (pain or paraesthesias) were always present
2. Motor (weakness and atrophy) and electromyographic signs of acute axonal neuropathy within 2 months after the beginning of symptoms were confined to one myotome

Three patients had a history of transient signs that could be attributed to myelopathy, but were not present at time of admission: painless transient weakness of the upper extremity (2 patients) and transient paraesthesias and hypaesthesia on homonymous extremities and trunk (1 patient). Patients with other involvement of the motor and/or sensory system that could lead to abnormalities in either SEPs or MEPs (*especially patients with polyneuropathy*) were not included in the study.

There were 17 men and 13 women, mean age 50.0 ± 6.5 years, who fulfilled the inclusion criteria and were enrolled into the 2-year follow-up study.

Clinical evaluation

The clinical deficit of the patients was evaluated by a detailed neurologic examination and expressed in terms of the 18-point cervical spondylotic myelopathy functional assessment scale (mJOA) [1], modified from the assessment scale proposed by Japanese Orthopaedic Association (JOA) [26]. Evaluations were carried out at the beginning of the study and after 6, 12, and 24 months. At entry, 17 patients had maximum score on the mJOA scale (18 points), 13 of them had decreased mJOA score (between 16 and 17) due to signs of cervical radiculopathy (both clinical and electromyographic). The duration of symptoms before the beginning of the study that could be attributed to cervical spondylosis were correlated with clinical course over the 2-year follow-up period.

Radiologic investigation

In all patients in the study group, plain anteroposterior, oblique and lateral radiograms were performed. The presence of spondylosis at the level of each cervical disc was registered. The absolute anteroposterior canal diameter at the level of the narrowest cervical canal and the Pavlov’s ratio [18] at the C5 level were measured. MRI of the cervical spine and the spinal cord was performed using T1- and T2-weighted and proton-density sagittal and axial images of the cervical cord. The MR signs of cervical cord compression and/or myelopathy were defined as: impingement of the cervical cord (i.e. a concave defect in the spinal cord adjacent to a site of disc bulging) and/or compression of the cervical cord (compression ratio of less than 40% [11, 17]) and/or the intramedullary T2 signal hyperintensity at the level of cord compression. The presence of MR medullary hyperintensity was correlated with the clinical course. The group was not systematically screened for the signs of lumbar spondylosis and/or discopathy.

Evoked potentials evaluation

Somatosensory evoked potentials

Short-latency SEPs from the median (SEP MED) and the tibial nerves (SEP TIB) were elicited with electrical stimulation of mixed nerves at the wrist and the ankle and recorded using a Nicolet four-

channel Viking IIe unit, at the beginning of the study and after 6, 12, and 24 months. During SEP MED the brachial plexus N9 response (from the ipsilateral Erb's point to reference electrode at F_z), the segmental dorsal horn medullar N13 response (from the spinous process C6 to an anterior cervical – AC – electrode above the thyroid cartilage), the medial lemniscus P14 response (from ipsilateral parietal C3/4' – Par_i – 2 cm posterior to the vertex and 7 cm lateral to the non-cephalic reference at contralateral Erb's point) and the cortical parietal response N20 (from contralateral parietal C3/4' – Par_c to ipsilateral parietal C3/4' – Par_i) were recorded. During SEP TIB the lumbar medullar N22 response (from the spinous process L1 to the reference over the contralateral iliac crest) and the cortical P40 response (from C_z 2 cm behind the vertex to the cephalic reference at F_{pz}). The absolute peak latencies and peak-to-peak amplitudes of all responses were measured and interpeak latencies between N13 and N20 and between N22 and P40, as "central sensory conduction times" were calculated for each side independently. Absolute right-left (R-L) differences in all latency parameters and R-L ratios of the N20 and P40 amplitudes were also calculated. The amplitude of the N13 response was measured to the preceding positive wave (P9) and expressed as an absolute value and as the N13/P9 ratio [20].

Central conduction abnormality attributed to possible cervical spinal cord lesion was defined as follows:

SEP MED abnormality: absent N13, P14 and/or N20 waves and/or abnormal N13-N20 interpeak latency and/or abnormal P9/N13 amplitude ratio and/or abnormal R-L amplitude ratio of N20 wave, all with normal N9 wave.

SEP TIB abnormality: absent P40 wave and/or abnormal N22-P40 interpeak latency and/or abnormal R-L amplitude ratio of P40 wave, all with normal N22.

Motor evoked potentials

MEPs were elicited using a MAGSTIM 200 magnetic stimulator and circular 90-mm (type 9784) stimulating coil with a peak magnetic field strength of 2.0 T, at the beginning of the study and after 6, 12, and 24 months. On-line data acquisition was performed using a Dantec Keypoint electromyograph. MEPs were elicited by means of transcranial and root magnetic stimulation and recorded from abductor digiti minimi (UMEP) and abductor hallucis muscles (LMEP) on both sides, with surface electrodes placed on the belly and tendon of the muscles. The stimulation intensity used was approximately 15–20% above the threshold intensity until at least two reproducible responses were obtained. Brain stimuli were delivered with the stimulation coil placed directly over the vertex (during the activation of abductor digiti quinti muscle); for activation of the abductor hallucis muscle the centre of the coil was moved 5 cm frontally and 2–3 cm contralaterally to the side of the recording. For the stimulation of cervical or lumbar nerve root exits, the coil was positioned on the midline at the neck (with the centre of the coil over the C7 spine) and lumbosacral region (with the centre of the coil at the S1 vertebral spine). During the cortical stimulation the patient held a steady isometric contraction of the target muscle (about 10–20% of maximum strength), while during root stimulation the target muscle was at rest. The shortest latencies of motor responses with brain stimulation (central latency – CL) and root stimulation (root latency – RL) and peak-to-peak amplitudes of the largest MEP after cortical stimulation were measured. The difference between CL and RL was defined as central motor conduction time (CMCT). To establish the contribution of the lower motor neuron to the absolute amplitude of MEP, the MEP/compound muscle action potential (CMAP) ratio was also calculated. Central conduction abnormality attributed to possible cervical spinal cord lesion was defined as abnormal CMCT and/or abnormal MEP/CMAP ratio. In one patient with clinical and electromyographic signs of C8/T1 root lesion CMCT was calculated

using the shortest F-wave latency (CMCT-F) [8, 12], to exclude false signs of central motor lesion due to lesion of the ventral motor root between the ventral horns and exit of motor roots from the intervertebral foramina. For the assessment of SEP and MEP recordings in the study group we used normal data obtained by the examination of the control group (Table 1). In order to minimize the false-positive rate of EP tests we set the normal limit at the level of the mean $\pm$ 3 SD; the parameters with other than normal Gaussian distribution (amplitude ratios, R-L differences of latency parameters) were considered abnormal when reduced below the lowest value obtained in the control group.

Criteria for intra-individual deterioration in SEP and MEP

A deterioration in a patient's MEP or SEP was recorded when any one of the following criteria had been met:

1. New absence of any main component, i.e. N13 or N20 (SEP MED), P40 (SEP TIB), motor response with transcranial stimulation
2. Change from normal to abnormal test (in case of abnormality based on latency change, prolongation of the latency under consideration exceeding 1 SD of normal values; in case of abnormality based on amplitude changes, decrease in the amplitude ratio under consideration exceeding 25% of the initial value)
3. Prolongation of the latency within abnormal results exceeding 2 SD of normal values or decrease in the amplitude ratio exceeding 50% of the initial value.

Data analysis

For statistical analysis of the data we used the χ^2 test and Fisher's exact test.

Results

Clinical course

New clinical evidence of cervical myelopathy (CM) and the decrease in the mJOA scale of at least one point within the 2-year follow-up period was found in five patients (16.7%). Other possible causes of new clinical signs and symptoms were excluded. New clinical signs of CM were as follows:

Patient 3: Paraesthesias and hypaesthesia of the left extremities and the left half of the trunk (excluding the face) – present at month 6, but not at month 24

Patient 9: Paraesthesias and weakness of both hands; mild spastic monoparesis of the right lower extremity; urinary symptoms (precipitant voiding) – present at month 6 and 24

Patient 26: mild spastic paraparesis and pallanaesthesia of the lower extremities – present at month 24

Patient 27: Paraesthesias and tactile hypaesthesia of the whole left upper extremity with weakness of the left hand and no pain – present at month 6 and 24

Patient 29: Paraesthesias of both hands; hypaesthesia in the left C8 dermatome and weakness of the left hand; mild

Table 1 Normal limits of selected evoked potential (EP) parameters (CMCT central motor conduction time, ADQ abductor digiti quinti, AH abductor hallucis, R-L right-left, MEP motor evoked potential, CMAP compound muscle action potential)

Parameter	Normal limits
CMCT (ADQ)	10.6 ms
CMCT (ADQ): R-L difference	3.7 ms
CMCT (AH)	21.0 ms
CMCT (AH): R-L difference	3.8 ms
MEP/CMAP amplitude ratio (ADQ)	11%
MEP/CMAP amplitude ratio (AH)	6%
N13 latency	16.1 ms
N13-N20 interpeak latency	7.0 ms
N13/P9 amplitude ratio	0.96
R-L N20 amplitude ratio	20%
N22-P40 interpeak latency	21.2 ms
P40 latency	46.3 ms

spastic monoparesis of the left lower extremity, impotence (erectile dysfunction) and urinary symptoms (precipitant micturition) – present at month 24

Duration of clinical symptoms and signs (≥ 5 years in 16 patients, < 5 years in 14 patients) or the presence of signs and/or symptoms of radiculopathy before the follow-up did not correlate with the appearance of new clinical signs of CM in our patients.

Radiologic findings

The anteroposterior diameter of the cervical spinal canal varied between 6 and 13 mm (mean 9.9 ± 1.6 mm). The mean value in five patients with new clinical signs of CM was 9.8 ± 2.3 mm. Pavlov's ratio varied between 0.5 and 1.1 (mean 0.803 ± 0.14). The mean value in five patients with new clinical signs of SCM was 0.80 ± 0.2

Table 2A, B The exact values of the selected EP parameters at entry examination of the study group. Patients displaying new clinical signs of cervical myelopathy during the 2-year period are signed with an asterisk; abnormal values are in **bold**

A Sensory evoked potentials (MED median nerve, ampl. r. amplitude ratio)

Patient	N13-N20 R (ms)	N13-N20 L (ms)	Other abnormal SEP MED parameters	N22-P40 R (ms)	N22-P40 L (ms)
1	6.3	5.9		16.6	17.4
2	5.3	5.7		26.7	21.6
3*	6.1	6.1	N13/P9 ampl. r. R: 0.67; L: 0.77	16.1	15.7
4	6.7	6.7		16.5	17.6
5	5.2	6		14.8	16
6	5	5.4		19.4	P 40 L: 48.8 ms^a
7	6.5	6.3		22	21.2
8	5.9	5.9		17.7	17.2
9*	6.9	6.5	N13/P9 ampl. r. L: 0.6	23.9	24.7
10	5.9	6.3		18	19.2
11	5.8	5.6		17.6	17.4
12	6.1	5.7		16.9	16.9
13	5.8	5.6		16.4	16.2
14	5.5	4.9		14.2	14.9
15	6.1	5.7		14.9	14.9
16	5.7	5.3		16.2	15
17	6.8	6.8		19.6	19.6
18	5.3	5.5		18	17.4
19	4.9	4.7	N13/P9 ampl. r. R: 0.5	26.2	20.4
20	5.5	5.9		20.4	22.7
21	5.9	6.1		22.4	22
22	5.7	5.5		Absent P40^b	24.3
23	6.3	5.7		20.4	27.1
24	6.3	6.3		18	17.4
25	5.9	6.9		16.2	17
26*	6.3	5.9		19.5	18.2
27*	Absent N13	7.1	N13/P9 ampl. r. L: 0.55	21.6	20.8
28	6.4	5.2		14.4	13.8
29*	6.4	7.1		Absent P40^b	Absent P40^b
30	6.2	5.3		17.4	16.6

^a N22–P40 latency could not be calculated due to unreproducible N22 wave

^b In all patients with absent cortical P40 wave the medullar N22 wave was recognizable

MR signs of medullar hyperintensity on *T2-weighted images* were found in nine patients (30%); new clinical signs of CM were found in two of these patients.

Study group

The exact values of selected EP parameters at entry examination are given in Table 2.

Evoked potentials

Control group

The normal limits based on the results obtained in the control group are given in Table 1. In the control group there were two tests with at least one parameter outside the established normal limits: in one control subject we found absent N22 wave and P40 latency of 46.8 ms on one side and N22-P40 interpeak latency of 21.1 ms on the other side; another control subject showed CMCT of 21.3 ms on one side.

Sensitivity

The frequency of EP abnormalities found at the beginning of the study using SEP MED and SEP TIB (40%) was similar to that found using UMEP and LMEP (36.7%) (Table 3). At least one abnormal EP test was found in 50% of all patients.

Correlation of EP and clinical changes

Changes in EP test results correlated well with the development of new clinical signs of cervical myelopathy (Table 4). At least one EP test showed deterioration at the time of the appearance of new clinical signs of cervical

Table 2B Motor evoked potentials. In patients with absent MEP to scalp stimulation the motor response to root stimulation was always present (*UE* upper extremities, *LE* lower extremities, *LMEP* MEP recorded from the abductor hallucis muscles)

Patient no.	CMCT UE R	CMCT UE L	CMCT LE R	CMCT LE L	Other abnormal LMEP parameters
1	6.7	6.7	14.7	13	
2	6.7	6	16.3	16.7	
3*	6.3	6.3	18.3	21.1	
4	5.8	4.4	13	12.2	
5	5.9	6	14.4	14.6	
6	7.7	8	18.4	19.6	
7	6.6	7.7	20.3	19	
8	6	6.4	16	15.4	
9*	6.1	6.4	17.7	16.9	
10	6.3	5.3	18	19.2	
11	5.4	6.1	Absent	19.2	
12	6	6	15.7	16.3	
13	7.2	7.3	18.2	17.7	
14	7.3	5.2	15.7	16.1	
15	5.3	6.2	15.6	15	
16	5	6.4	20^a	14	MEP/CMAP R: 4%
17	6.8	6.9	16.9	16.6	
18	7.4	7	15	15.3	
19	5.3	5	14.3	15.3	
20	5.7	5.3	26.4	26.5	
21	6.7	6	14.3	14.7	
22	8.2	8	18	17	
23	6.2	7	Absent	15.1	
24	5.5	6.1	16.7	16.9	
25	6	6.3	13.6	15	
26*	10.9	5.9	Absent	23.1	MEP/CMAP L: 3%
27*	6.3	5.7	21.1	17.1	
28	7.6	7.7	14.6	14.7	
29*	8.9	8.7	17	17.9	
30	5	6	17	16	

^a Abnormal CMCT R/L difference

Table 3 Sensitivity of entry SEPs and MEPs in 30 patients with pre-clinical spondylotic cervical cord compression (*TIP* tibial nerve, *UMEP* MEP recorded from the abductor digiti minimi)

	<i>EP modality</i>						
	SEP MED	SEP TIB	SEP	UMEP	LMEP	MEP	SEP + MEP
Sensitivity	16.7%	36.7%	40%	6.7%	30%	36.7%	50%

Table 4a Changes in EP between months 0 and 6 in patients with new clinical signs of cervical myelopathy within the 2-year follow-up period (↑ improvement, 0 unchanged, ↓ deterioration)

Patient no.	Clinical signs of cervical myelopathy at month 6	<i>EP modality</i>			
		SEP MED	SEP TIP	UMEP	LMEP
3	Yes	0	0	0	↓
9	Yes	0	↑	↓	↓
26	No	0	0	0	0
27	Yes	0	↓	0	↓
29	No	0	↓	0	0

Table 4b Changes in EP between months 0 and 24 in patients with new clinical signs of cervical myelopathy within the 2-year follow-up period

Patient no.	Clinical signs of cervical myelopathy at month 24	<i>EP modality</i>			
		SEP MED	SEP TIP	UMEP	LMEP
3	No	0	0	0	0
9	Yes	0	0	↓	↓
26	Yes	0	↓	0	0
27	Yes	0	↓	0	↓
29	Yes	0	↓	↓	↓

myelopathy. At month 6 clinical signs of CM in three patients were accompanied by deterioration in five EP tests (LMEP three times, UMEP once, SEP TIB once) and improvement in one EP test (SEP TIB); at month 24 clinical signs of CM in four patients were accompanied by deterioration in eight EP tests (LMEP three times, SEP TIB three times, UMEP twice).

New abnormality of the median nerve SEP segmental N13 response was found in four patients without a corresponding clinical change that could be attributed to cervical cord involvement.

Predictive value of the abnormality of EP tests

Fifteen patients from the study group had at least one abnormal EP test at the entry examination; in five of them we observed new clinical signs and symptoms of cervical myelopathy within 2 years. In no patient from the remainder of the study group, with all normal EP tests (15 patients), did we find new clinical signs and symptoms of cervical myelopathy; the false-negative rate of the normal

EP test in predicting the new clinical signs of myelopathy in our group was equal to 0. The association between EP abnormality and clinical manifestation of SCM during the 2-year period was statistically significant (*Fisher's exact test*, $P = 0.02$).

Discussion

Although the effect of surgery in SCM seems to be beneficial, a significant number of patients undergoing surgery do not improve [4, 9, 10]. Greater benefit would probably result from judicious intervention at an earlier stage of the disease, before considerable neurological deficit is present. However, the effect of surgical intervention in unselected cervical myelopathy patients with mild degree of involvement, or even in patients with pre-clinical spondylotic cervical cord compression, needs to be proved in large multicentre controlled studies [22]. Evoked potentials could potentially serve as an additional objective tool in the assessment of the result of therapy and in the timing of surgery in SCM patients.

In a substantial subgroup of cervical spondylosis patients, EPs could disclose subclinical involvement of both somatosensory and upper motor neuron pathway. This evidence would be especially useful in pre-clinical spondylotic cervical cord compression documented by MR, where it could help in the evaluation of the relevance of such a compression. The incidence of morphologic abnormalities is high in asymptomatic individuals, where the effect of surgical treatment could not be predicted [4]. Teresi et al. [24], in 100 asymptomatic patients, reported spinal cord impingement seen on MR in 16% of patients under 64 years of age and 26% of those over 64 years.

Previous reports have given conflicting data on the frequency of EP abnormalities in patients with asymptomatic cervical stenosis. Kaneyama et al. [13] reported normal MEPs in clinically silent cervical stenosis, while other authors have found electrophysiological signs of subclinical cervical cord involvement in a substantial part of these patients – Maertens de Noordhout et al. [16] in 11% and Tavy et al. [23] in 25% using MEP, Khan et al. [14] in 37.5% using SEP and EMG. Travlos et al. [25], in patients with radiculopathies at the level C7 or above without overt clinical myelopathy, documented MEP signs (using the recording from *abductor digiti quinti muscle* of the C8/T1 myotome) of functional cervical cord involvement in as many as 65% of them. The sensitivity of EPs in our group of patients with clinically “silent” cervical stenosis with

MR-documented cervical compression was substantial (MEP and SEP sensitivity of 40 and 36.7%, respectively, and that of combined MEP and SEP recording of 50%). An unknown proportion of patients with spondylotic compression of the cervical cord may remain free of myelopathic symptoms for an uncertain period of time. EPs could help to evaluate functional cervical cord involvement in these patients.

In cervical spondylosis patients with clinical symptoms and signs in the upper extremities, it is sometimes very difficult to distinguish clinically between signs and symptoms of radiculopathy and myelopathy; moreover, both conditions may coincide in the same patient. Electromyographic examination could exclude distal entrapment neuropathies and delineate the distribution of motor axonal neuropathy; radiculopathy is more likely to be distributed within one myotome, but the exact EMG differentiation between radiculopathy and myelopathy with anterior horn cell lesions is impossible. Both SEPs and MEPs may be helpful in differentiating the two conditions [8, 12]. They also correlated well with the clinical manifestation of overt myelopathy, accompanied by change in at least one EP test in five patients, mostly in MEP and SEP from the lower extremity. Upper extremity SEP segmental N13 medullar response was shown to be a sensitive indicator of medullar involvement in SCM [19–21] and is believed to be a hallmark of potentially reversible segmental dorsal horn cervical cord dysfunction due to ischaemia [21], with a great potential for clinical improvement. New abnormality of N13 potential in our group, recorded in several of our patients during the 2-year follow-up, was not accompanied with change in clinical status that could be interpreted as manifestation of cervical myelopathy. We may speculate on the possibility that transient cord ischaemia could play a more important role in advanced cervical cord compression, while initial clinical signs of cervical cord involvement in patients with mostly focal cervical cord spondylotic impingement may partly be caused by demyelinating lesion of long motor and sensory tracts.

The high sensitivity of the EP battery in our study group may raise suspicion of false-positive results. In general, abnormal results in “healthy” subjects may be due to statistical error (type I error). In order to minimize the false-positive rate of each EP test, we set the normal limit at the level of the mean $\pm$ 3 SD (the probability that a “normal person” will have an abnormal EP parameter in the event of the normal limit being set at mean $\pm$ 3 SD will be 0.3%). The false-positive rate of the whole test increases with the number of evaluated parameters. We therefore used only a limited number of EP parameters for the evaluation of both our groups. We found two “abnormal” tests out of 160 tests performed in the 40 subjects of the control group, i.e. 1.25% of tests and 6% of subjects (considering the whole EP battery as one test), which is acceptable. Our control subjects were not systematically screened for the presence of cervical spondylosis. Despite the fact that

they were free of local cervical and radicular pains and other signs of radicular or medullar cervical lesion, the possibility of subclinical compression of cervical cord causing abnormality of EP test in either of the two abnormal control cases cannot be excluded.

Another potential problem in the interpretation of EP abnormalities in our cervical spondylotic study group is the well-known association of this condition with lumbar spondylosis. The changes in EP parameters were frequently observed in SEP TIB and LMEP, and hypothetically they could partly be caused by “subclinical” lumbar spondylotic stenosis. The SEP TIB abnormalities in all but one test from one side with absent N22 wave and prolonged P40 latency (patient 6) were of the “central type”, thus excluding the possibility of root S1 or cauda equina lesions. Prolonged CMCT to the lower limbs could, on the other hand, be caused by centrally localised lumbar stenosis [3]. In seven patients with “central” type LMEP abnormality (i.e. prolonged CMCT and/or abnormal MEP/CMAP ratio) we registered historical and slight residual clinical signs of S1 root lesion in one side, i.e. S1 hyporeflexia in patient no. 20 with bilateral symmetrical prolongation of CMCT. The symptoms of neurogenic claudication were absent in all patients with abnormal UMEP. As we did not systematically screen our patients for the signs of lumbar spondylotic stenosis, the possibility of subclinical “proximal” root or cauda compression should be taken into consideration in the interpretation of the “central” UMEP abnormality in our patients. The establishment of “subclinical” cauda equina or root compression is, however, less reliable and more prone to subjective observer bias than the establishment of MR signs of “subclinical” cervical cord compression or myelopathy.

Several factors have proved to be prognostic indicators of poorer outcome after surgery in spondylotic cervical myelopathy: the duration of the disease plus the presence of a hyperintense T2-weighted signal on the preoperative MR scan and low transverse cord area [17]. The former two factors, however, were not associated with the clinical manifestation of overt cervical myelopathy during the 2-year follow-up period in our patients. EP abnormality found in half of these subjects, on the other hand, predicted clinical manifestation of overt myelopathy in one-third of these patients over the 2-year period, while negative EP tests signalled favourable prognosis. The advantages of surgical therapy in mild and pre-clinical SCM and, above all, the timing of the operation, remain open problems, and need investigation in a large, multicentre, randomised and controlled study [10, 22]. Selected patients may potentially profit from early decompression, and EP testing may help in selecting such a subgroup for the purposes of further research.

Conclusions

1. SEPs and MEPs documented subclinical involvement of cervical cord in 50% of patients with pre-clinical spondylotic cervical cord compression.
2. Over a 2-year period, clinical signs of cervical myelopathy were observed in one-third of patients with entry EP abnormality in comparison with no patients with normal EP tests.

3. Combined SEPs and MEPs proved to be a valuable tool in the assessment of the functional relevance of subclinical spondylotic cervical cord compression and in predicting a 2-year clinical outcome.

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