The use of evoked potentials for clinical correlation and surgical outcome in cervical spondylotic myelopathy with intramedullary high signal intensity on MRI

R K Lyu, L M Tang, C J Chen, C M Chen, H S Chang, Y R Wu

Objective: To investigate the use of motor evoked potentials (MEPs) and somatosensory evoked potentials (SEPs) for clinical significance and surgical outcome in patients with cervical spondylotic myelopathy (CSM) with intramedullary high signal intensity on T2 weighted MRI.

Methods: Forty nine patients were scored according to the modified Japanese Orthopaedic Association (JOA) score for cervical myelopathy. MEP and SEP studies were performed and the results were categorised as normal or abnormal. Thirty nine patients who had received surgical decompression were re-evaluated after 6 months. Surgical outcome was represented by the recovery ratio of the JOA score.

Results: Abnormal MEPs were observed in 44 patients (arm: 43; leg: 30). Abnormal SEPs were found in 32 patients: (median: 24; tibial: 23). Patients with abnormal SEPs had a worse JOA score than those with normal SEPs. Thirty nine patients received surgical treatment. Patients younger than 55 had better recovery ratios than those who were 55 or older (p = 0.005, two sample Student’s t test). Patients with normal median SEPs also had better recovery ratios than those with abnormal median SEPs (p = 0.007, two sample Student’s t test). Among median SEP variables, only N9-20 was significantly associated with recovery ratio (p = 0.016, stepwise linear regression), with age factor controlled (p = 0.025, stepwise linear regression).

Conclusion: Arm MEP was the most sensitive EP test for detecting myelopathy in patients with chronic CSM. Median and tibial SEPs correlated well with the severity of myelopathy while normal median SEPs correlated with good surgical outcome. Among median SEP variables, only N9-20 correlated with surgical outcome.

CSM, a slowly progressive devastating disease. Its clinical manifestations include gait disturbance, clumsiness and paresthesia of the hands, and signs of pyramidal and posterior column dysfunction. Its diagnosis is based on typical clinical manifestations and appropriate radiological studies. In the past, the radiological diagnosis of CSM was dependent on myelography, computed tomography (CT), and CT myelography. At present, magnetic resonance imaging (MRI) is the preferred radiological diagnostic tool that can not only depict anatomically how the spinal cord is compressed but also reflect the pathologic changes in the spinal cord. Many authors have reported intramedullary high signal intensity on T2 weighted MRI in patients with compressive lesions of the cervical spinal cord. Such intramedullary high signal intensity abnormality is presumed to be myelomalacia or cord gliosis secondary to a long standing compressive effect of the spinal cord. Therefore, the presence of intramedullary high signal intensity in patients with CSM indicates an existence of a chronic spinal cord compressive lesion.

Motor evoked potentials (MEPs) and somatosensory evoked potentials (SEPs) are two useful neurophysiological studies for detecting functional abnormality of the spinal cord. When clinical presentations of cervical myelopathy are equivocal, neurophysiological investigations may be of clinical value. Many reports have shown that the incidence of abnormal SEPs or MEPs is high in patients with CSM. Although it has been reported that there is a significant correlation between the presence of a spinal cord compression and abnormal MEPs, SEPs, or both in patients with CSM, the predictive value of MEPs and SEPs for surgical outcome has not been studied systematically. Also, most previous studies describing MEP or SEP findings in CSM usually included patients with CSM diagnosed radiologically by myelography or CT myelography. There has been no study addressed on MEPs and SEPs findings in CSM with intramedullary T2 high signal intensity lesions disclosed by MRI.

The purpose of this study was to investigate the clinical and neurophysiological significance of MEPs and SEPs in patients with CSM with intramedullary T2 high signal intensity lesion, as well as the usefulness of evoked potential studies in predicting surgical outcome.

METHODS

Patients

We included 49 patients (mean age 56.7 years, range 27–77 years, 32 men and 17 women) who had had a clinical diagnosis of cervical spondylotic myelopathy, an intramedullary T2 high signal intensity lesion disclosed by MRI and studies of MEPs and SEPs. The diagnosis of cervical spondylotic myelopathy was made on the basis of clinical and radiological findings. All patients showed neurological manifestations and appropriate radiological studies. All patients showed neurological findings. All patients showed neurological findings.

Abbreviations: APB, abductor pollicis brevis muscles; CMCT, central motor conduction time; CSM, cervical spondylotic myelopathy; CT, computed tomography; IPL, interpeak latency; JOA, Japanese Orthopaedic Association; MEP, motor evoked potential; MRI, magnetic resonance imaging; SEP, somatosensory evoked potential; TA, tibialis anterior muscles.
findings compatible with cervical myelopathy, including signs of pyramidal tract or posterior column involvement in legs or arms. The patients’ clinical symptoms and signs are summarised in table 1. While all patients had motor dysfunction in the arms or legs, only 38 (78%) had sensory dysfunction. None of the patients had other neurological diseases such as multiple sclerosis, motor neurone disease or vitamin B-12 deficiency. Based on the clinical symptoms and signs, the severity of neurological deficits of all patients was scored according to the modified Japanese Orthopaedic Association (JOA) score for cervical myelopathy (table 2). 17 The range of this score is 0 to 21; 0 = maximal neurological deficits, 21 = no neurological deficits.

All patients showed unequivocal findings of cervical cord compression due to cervical spondylotic changes, as well as intramedullary high signal intensity lesion on T2 weighted MRI. The number of segments with intramedullary high signal intensity lesion ranged from one to three segments: one segment in 38 patients (78%), two in 10 patients (20%), and three in one patient (2%). Location of intramedullary high signal intensity lesion was at C2-3 level in one patient (2%), C3-4 in 23 patients (47%), C4-5 in 15 patients (30%), C5-6 in 17 patients (35%), and C6-7 in 5 patients (10%).

Thirty nine patients received a surgical decompression of the cervical cord; all were clinically re-evaluated 6 months postoperatively. Surgical outcome of the patients was represented by a recovery ratio calculated by the formula of Hirabayashi et al: 14

\[ \text{recovery ratio percentage} = \left( \frac{\text{postoperative JOA score}}{21 - \text{preoperative JOA score}} \right) \times 100 \]

**Controls**

To provide an age comparable control group for the results of the evoked potential tests, 50 healthy volunteers, 26 men and 24 women, aged 56.3 (11.8) years (range, 26–79 years) without any signs and symptoms of neurological diseases were studied. Results of MEPs and SEPs in these 50 normal control subjects are given in table 3. This study was approved by the ethics committee of Chang Gung Memorial Hospital, and informed consent was obtained from all subjects.

**Motor evoked potentials (MEPs)**

MEPs were recorded with surface electrodes from the abductor pollicis brevis (APB) and the tibialis anterior (TA) muscles, evoked by magnetic stimuli generated by a magneto-electrical stimulator (Magstim 200; circular coil with an outer diameter of 140 mm). For cortical stimulation, the coil was held with its centre above the Cz position of the 10-20 system for the APB muscle, and 2–4 cm more frontally for the TA muscles. Stimulation was performed with the stimulus output 20–25% above threshold of each muscle. During stimulation the target muscle was contracted slightly. At least five consecutive trials were recorded and superimposed. The shortest onset latency of the evoked compound muscle action potentials after cortical stimulation (cortical latency) was recorded for each muscle tested. After the procedure of magnetic stimulation, electrical stimulation was delivered to the median and peroneal nerves at the wrist and at the head of fibula, respectively, to elicit F and M waves. The peripheral conduction time was determined using the following formula: \((F+M)/2\), where F and M were onset latencies of the F wave and the M response, respectively. 16

The central motor conduction time (CMCT) was calculated by subtracting the peripheral conduction time from the shortest cortical latency. Both the MEPs and the F wave study were recorded on a Nicolet Viking EMG-EP system (Madison, Wisconsin).

**Somatosensory evoked potentials (SEPs)**

SEPs of the arm were recorded using surface electrodes on the Erb’s point, C2 spinous process, and the scalp overlying the primary sensory area in the parietal lobe contralateral to the stimulated limb (2 cm behind the 10-20 System, C3 and C4 locations). A reference electrode was placed on Fz. The median nerve was stimulated at each wrist using 0.2 ms square wave electrical pulses. The stimulus intensity was adjusted to produce a visible twitch of the APB muscle without causing discomfort. SEPs of the leg were recorded on the first lumbar spinous process and on the midline of the scalp 2 cm posterior to the vertex. The tibial nerve was stimulated at the ankle. At least 1024 responses were averaged for each test. To confirm SEP reproducibility, each measurement was carried out at least twice. SEPs were recorded on a Nicolet Spirit EP system (Madison, Wisconsin).

The following measurements of SEPs were recorded for the median SEPs: peak latency of responses recorded at Erb’s point (N9), the C2 spinous process (N13) and the scalp (N20). Interpeak latencies (IPLs) between N9 and N13, and N9 and N20 were calculated. If there was no C2 spinal response, then both N13 and N9-N13 IPLs were considered absent. If there was no cortical response, then both N20 and N9-N20 IPLs were considered absent. In the tibial SEPs, peak latency of responses recorded at the L1 spinous process (N22) and the scalp (P40), as well as IPL between N22 and P40 were recorded. If there was no cortical response, then both P40 and N22-P40 IPL were considered absent.

**Data analysis**

Because MEP and SEP responses could be obtained from right or left limbs, only data from the worse side were included for analysis. The limits of normal values were set at mean±3 SD. The arm or leg MEPs were considered abnormal if either cortical latency or CMCT exceeded normal limits or was absent. An MEP study was considered abnormal if either of the arm or leg MEPs was abnormal. The median SEP was considered abnormal if any of the following measures was
prolonged or absent: peak latencies of N13 and N20, as well as N9-N13 and N9-N20 IPLs. However, the median SEP was still considered to be normal if N9-N13 and N9-N20 IPLs were within normal limits, in case the absolute latencies of N13 and N20 were prolonged. The tibial SEP was considered to be abnormal when P40 peak latency was prolonged or absent or N22-P40 IPL was prolonged. An SEP study was considered abnormal if either the median or tibial SEP was abnormal.

Because the height of our subjects was not recorded and age was not taken into account for evoked potential measures, broader limits of normal values were used in our study (mean +3 SD). This simplified routine use of these tests. The data were analysed using the $x^2$ test, two sample Student’s $t$ test, or Pearson's correlation coefficient. Stepwise linear regression was used to compare the results of MEPs and SEPs with the severity of myelopathy (initial JOA scores). Subsets of the MEP and SEP variables were analysed separately. The same model was used to compare the results of MEPs and SEPs with surgical outcome (—that is, recovery ratio). The significance level was set at 5%. Statistical analysis was performed on an IBM compatible personal computer, using SPSS 10.0 (SPSS, Chicago, Illinois).

RESULTS
Clinical data
Symptoms suggestive of cervical myelopathy had existed for 1–60 months (average 8.7 months) when the 49 patients were examined. Gait disturbance and numbness of the arms were the most frequent symptoms. Hyperreflexia in the legs

| Table 2: Modified Japanese Orthopaedic Association score for cervical myelopathy*

| I. Motor dysfunction score for the upper extremities |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| 0 | Unable to move hands  | 1 | Unable to eat with a spoon but able to move hands  | 2 | Unable to button shirt but able to eat with a spoon  | 3 | Able to button shirt with great difficulty  | 4 | Able to button shirt with slight difficulty  | 5 | No dysfunction  |
| 1 | Severe sensory loss or pain  | 2 | Mild sensory loss  | 3 | No sensory loss  |

| II. Motor dysfunction score for the lower extremities |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| 0 | Complete loss of motor and sensory function  | 1 | Sensory preservation without ability to move legs  | 2 | Able to move legs but unable to walk  | 3 | Able to walk on flat floor with a walking aid (cane or crutch)  | 4 | Able to walk up or down stairs with handrail  | 5 | Moderate to significant lack of stability but able to walk up or down stairs without handrail  | 6 | Mild lack of stability but able to walk with smooth reciprocation unaided  | 7 | No dysfunction  |

| III. Sensory dysfunction score for the upper extremities |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| 0 | Complete loss of hand sensation  | 1 | Severe sensory loss or pain  | 2 | Mild sensory loss  | 3 | No sensory loss  |

| IV. Sensory dysfunction score for the lower extremities |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| 0 | Complete loss of foot sensation  | 1 | Severe sensory loss or pain  | 2 | Mild sensory loss  | 3 | No sensory loss  |

| V. Sphincter dysfunction score |
|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|
| 0 | Unable to micturate voluntarily  | 1 | Marked difficulty with micturition  | 2 | Mild to moderate difficulty with micturition  | 3 | Normal micturition  |

*Adapted from Yonenobu et al.

| Table 3: Results of MEPs and SEPs in 49 patients with cervical spondylotic myelopathy and 50 normal controls

<table>
<thead>
<tr>
<th>Motor evoked potentials</th>
<th>Somatosensory evoked potentials</th>
</tr>
</thead>
</table>
| APB       | CMCT   | Tibialis anterior  | Median       | N13       | N20       | N9-13      | N9-20      | P40       | N22-P40*
| Col    | CMCT   | Col    | CMCT   | N13       | N20       | N9-13      | N9-20      | P40       | N22-P40*
| Patients |
| Normal | 8 (16%) | 10 (20%) | 24 (49%) | 20 (41%) | 28 (57%) | 29 (59%) | 27 (55%) | 27 (55%) | 26 (53%) | 26 (65%) |
| Prolonged | 27 (55%) | 25 (51%) | 3 (6%) | 7 (14%) | 7 (14%) | 10 (20%) | 6 (16%) | 12 (25%) | 9 (18%) | 6 (12%) |
| Absent | 14 (29%) | 14 (29%) | 22 (45%) | 22 (45%) | 14 (29%) | 14 (29%) | 10 (20%) | 14 (29%) | 8 (20%) |
| Mean (SD)† | 29.5 (4.8) | 14.3 (4.4) | 40.2 (6.2) | 17.8 (7.2) | 14.3 (1.5) | 20.9 (2.2) | 4.7 (1.0) | 11.3 (1.7) | 44.1 (6.6) | 21.1 (5.9) |
| Normal controls |
| Mean (SD) | 19.9 (1.9) | 6.3 (1.5) | 38.9 (2.6) | 13.0 (2.2) | 12.8 (1.0) | 18.3 (1.3) | 3.6 (0.6) | 9.1 (1.0) | 37.8 (2.9) | 16.9 (2.0) |
| Mean+3 SD | 25.6 | 10.7 | 46.8 | 19.9 | 15.8 | 22.3 | 5.5 | 12.2 | 46.5 | 23.0 |

APB, abductor pollicis brevis; CMCT, cortical latency; CMCT, central motor conduction time
†N22 could not be recorded and thus immeasurable N22-P40 was not included for calculation in nine patients. †From those patients with recordable responses (ms). p<0.001, compared with normal control, unpaired Student’s t test.
Median and tibial SEPs were abnormal. Tibial SEP: N22: 22.7 ms, P40: 47.4 ms, N22-P40: 24.7 ms. Both N13: 15.3 ms, N20: 22.4 ms, N9-13: 5.7 ms, N9-20: 12.8 ms. (C) was abnormal and leg MEP was normal. (B) Median SEP. N9: 9.6 ms, time was 10.9 and 13.6 ms in the arm and leg, respectively. Arm MEP and 32.7 ms in the arm and leg, respectively; central motor conduction were 14 and 0.28, respectively. (A) MEPs. Cortical latency was 25.9 year old woman with CSM. Her initial JOA score and recovery ratio Figure 1

Motor and sensory evoked potentials recordings from a 65 year old woman with CSM. Her initial JOA score and recovery ratio were 1.4 and 0.28, respectively. (A) MEPs. Cortical latency was 25.9 and 32.7 ms in the arm and leg, respectively; central motor conduction time was 10.9 and 13.6 ms in the arm and leg, respectively. Arm MEP was abnormal and leg MEP was normal. (B) Median SEP. N9: 9.6 ms, N13: 15.3 ms, N20: 22.4 ms, N9-13: 5.7 ms, N9-20: 12.8 ms. (C) Tibial SEP. N22: 22.7 ms, P40: 47.4 ms, N22-P40: 24.7 ms. Both median and tibial SEPs were abnormal.

Results of MEPs and SEPs
These results are summarised in table 3. Abnormal MEPs were observed in 44 patients (90%): arm MEP abnormality was more frequently encountered than leg MEP abnormality (88% versus 61%, p = 0.005, two side Fisher’s exact test). The mean cortical latency and CMCT to the APB muscle in those patients with recognisable responses were significantly longer than in normal controls. The prolonged cortical latency or CMCT was more frequently encountered than a lack of response in the arm MEP (see the figure). In many patients the cortical latency and CMCT were markedly prolonged, up to 38.5 ms for cortical latency (upper normal limit: 25.6 ms) and up to 21.6 ms for CMCT (upper normal limit: 10.7 ms). In the leg MEP study, no response after cortical stimulation was more frequently noted than prolonged cortical latency or CMCT. The mean CMCT to the TA muscle in those patients with recognisable responses was significantly longer than that in normal controls but the mean cortical latency was not different from that in normal controls. Only one patient had abnormal leg MEP but normal arm MEP.

Abnormal SEPs were found in 32 patients (65%): abnormal median SEP in 24 (49%) and abnormal tibial SEP in 23 (47%). Results of various measurements of the median and tibial SEPs from patients with recognisable responses were all significantly prolonged as compared with those from normal controls (table 3). There were nine patients whose N22 could not be recorded. The P40 of these patients was either prolonged (in three patients) or absent (in six patients). Because clinically all these patients had increased reflexes in the legs and spastic gait, their tibial SEPs were regarded to be abnormal. The abnormal rates of various measurements of the median and tibial SEPs, ranged from 33% to 47%, did not differ from one another. A lack of response was more frequently observed than a prolonged latency in most SEP measurements. Of the 11 patients without sensory deficit clinically, three had abnormal SEPs. None of the 49 patients had abnormal SEPs but normal MEPs.

Correlation between evoked potentials, clinical features, and surgical outcome
Table 4 summarises the correlations between clinical data and results of evoked potential studies. Initial JOA scores of patients with abnormal median or tibial SEP results were worse than those with normal median or tibial SEP results. The two sample Student’s t test or Pearson’s correlation coefficient test did not show a significant difference or a correlation between initial JOA score and age (Pearson’s correlation coefficient, r = −0.239, p = 0.098), sex, and

<p>| Table 4 Correlations of initial JOA score, clinical features, and evoked potential data |
|---------------------------------|------------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Initial JOA score</th>
<th>No. of patients</th>
<th>Mean (SD)</th>
<th>p Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>49</td>
<td>13.9 (4.4)</td>
<td>0.231</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>32</td>
<td>13.3 (4.2)</td>
<td>0.231</td>
</tr>
<tr>
<td>Women</td>
<td>17</td>
<td>14.9 (4.7)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>=55 years</td>
<td>26</td>
<td>13.7 (4.5)</td>
<td>0.783</td>
</tr>
<tr>
<td>&lt;55 years</td>
<td>23</td>
<td>14.0 (4.4)</td>
<td></td>
</tr>
<tr>
<td>Arm MEP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>6</td>
<td>17.7 (4.8)</td>
<td>0.079</td>
</tr>
<tr>
<td>Abnormal</td>
<td>43</td>
<td>13.3 (4.1)</td>
<td></td>
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<tr>
<td>Leg MEP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>23</td>
<td>14.8 (4.4)</td>
<td>0.150</td>
</tr>
<tr>
<td>Abnormal</td>
<td>26</td>
<td>13.0 (4.3)</td>
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<tr>
<td>Median SEP</td>
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<tr>
<td>Normal</td>
<td>25</td>
<td>16.2 (3.6)</td>
<td>&lt;0.001</td>
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<tr>
<td>Abnormal</td>
<td>24</td>
<td>11.5 (3.9)</td>
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<tr>
<td>Tibial SEP</td>
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<tr>
<td>Normal</td>
<td>26</td>
<td>15.4 (3.8)</td>
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</tr>
<tr>
<td>Abnormal</td>
<td>23</td>
<td>12.1 (4.4)</td>
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</table>

*Two sample Student’s t test.

<p>| Table 5 Correlations of clinical features, evoked potential data, and recovery ratio |
|---------------------------------|------------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Recovery ratio</th>
<th>No. of patients</th>
<th>Mean (SD)*</th>
<th>p Value†</th>
</tr>
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<tbody>
<tr>
<td>Total</td>
<td>39</td>
<td>51.0 (29.4)</td>
<td>0.602</td>
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<td>Sex</td>
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<tr>
<td>Men</td>
<td>27</td>
<td>49.4 (31.9)</td>
<td>0.602</td>
</tr>
<tr>
<td>Women</td>
<td>12</td>
<td>54.8 (23.8)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>=55 years</td>
<td>19</td>
<td>37.8 (24.9)</td>
<td>0.005</td>
</tr>
<tr>
<td>&lt;55 years</td>
<td>20</td>
<td>63.6 (28.4)</td>
<td></td>
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<tr>
<td>Arm MEP</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Normal</td>
<td>3</td>
<td>77.7 (25.4)</td>
<td>0.179</td>
</tr>
<tr>
<td>Abnormal</td>
<td>36</td>
<td>48.8 (28.9)</td>
<td></td>
</tr>
<tr>
<td>Leg MEP</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>18</td>
<td>49.2 (31.8)</td>
<td>0.729</td>
</tr>
<tr>
<td>Abnormal</td>
<td>21</td>
<td>52.6 (28.0)</td>
<td></td>
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<tr>
<td>Median SEP</td>
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<td></td>
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</tr>
<tr>
<td>Normal</td>
<td>16</td>
<td>65.8 (26.9)</td>
<td>0.007</td>
</tr>
<tr>
<td>Abnormal</td>
<td>23</td>
<td>40.7 (27.0)</td>
<td></td>
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<tr>
<td>Tibial SEP</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>19</td>
<td>53.9 (28.6)</td>
<td>0.561</td>
</tr>
<tr>
<td>Abnormal</td>
<td>20</td>
<td>48.3 (30.7)</td>
<td></td>
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</table>

*Data are presented as percentages. †Two sample Student’s t test.
The mean JOA scores before surgery and at 6 months after surgery were 13.1 (4.0) (range 2–21) and 16.8 (3.2) (range 9–21), respectively. The improvement of the JOA score was significant (\(t\) test, \(p = 0.001\)). Age was an important prognostic factor. The recovery ratio was 51.0% (29.4%) (range 0–100%) and significantly correlated with age (Pearson's correlation coefficient, \(r = -0.443\), \(p = 0.005\)). Patients younger than 55 years and those with normal median SEP results had a better recovery ratio (table 5). The recovery ratio did not correlate with the initial JOA score (Pearson's correlation coefficient, \(r = 0.298\), \(p = 0.065\)), sex, arm or leg MEP findings, or tibial SEP results. Of the median SEP variables, N9-20, N9-13 and N20 abnormalities were associated with a worse recovery ratio (two sample Student's \(t\) test, table 6). Using stepwise linear regression to test the correlation of the median SEP variables and the recovery ratio, only N9-20 (\(t\) of coefficient = \(-2.517\), \(p = 0.016\)) was significantly associated with the recovery ratio. With age factor controlled, the recovery ratio was still worse in those with abnormal N9-20 than in those with normal N9-20 (\(t\) of coefficient = \(-2.346\), \(p = 0.025\)).

**DISCUSSION**

Previous studies showed that MEP abnormalities were frequently found in patients with CSM. Jaskolski et al. found abnormal CMCT of hand muscles in 10 of their 16 (63%) patients with CSM. Maertens de Noordhout et al. reported prolonged CMCT of hand muscles in 32 of 44 (73%) patients, and abnormal configuration or amplitude of the MEP responses in another five patients. Di Lazzaro et al. reported abnormal CMCT in all 24 patients studied. Tavy et al. reported arm MEP abnormality in 25 of 28 (89%) patients and leg MEP abnormality in 23 of 25 (92%) patients. Maertens de Noordhout et al. reported that 48 (87%) and 47 (85%) of their 55 patients with CSM had an abnormal CMCT of the arm and the leg, respectively. Therefore, the incidence of MEP abnormality has been high in patients with CSM and prolonged CMCT is the major MEP abnormality noted.

In our study, the incidence of arm MEP abnormality was 88%; prolonged CMCT or cortical latency was found in 55% of patients and a lack of cortical response in 30% of patients. The mean values of CMCT and cortical response in the hands following magnetic stimulation were markedly prolonged in the patient group as compared to those of normal controls (14.3 ms against 10.7 ms for CMCT, 29.5 ms against 25.6 ms for cortical latency). Hence, the high incidence of arm MEP abnormality in our patients, as well as in the previous studies, indicates that arm MEP study is a sensitive test to detect spinal cord dysfunction in patients with CSM.

The incidence of leg MEP abnormality in our study was 61%; prolonged CMCT or cortical latency was found in 14% of patients and a lack of cortical response in 45%. Hence, leg MEP abnormality was less frequently encountered in our patients than those of previous studies (87–92%).

The basis for the lower incidence of leg MEP abnormality in our patients is unclear. It is unlikely that our patients' myelopathy was mild; most of our patients had gait disturbance and hyperreflexia in the legs. One possible explanation for the lower sensitivity of MEPs in the leg than in the arm might be the greater variability of latency values in normal subjects. Thus, the delay of leg MEPs at the abnormal cervical segments may not be sufficient to take the leg CMCT or cortical latency outside the normal limit. These could explain why a lack of cortical response is the major abnormal finding rather than prolonged CMCT or cortical latency following magnetic stimulation in leg MEPs observed in our patients.

Several studies have shown that more than half of patients with cervical myelopathy had SEP abnormality in at least one nerve; median and tibial SEP abnormalities were presented in 18–69% and 43–100%, respectively. Several authors reported that tibial SEP was the most sensitive SEP test in CSM. A few authors found that ulnar SEP was more sensitive than tibial SEP, or found no difference between the arm and leg SEPs. In our study, abnormal SEP was detected in 32 (65%) patients, including three (6%) who had no sensory deficits clinically. Thus, our results confirmed the value of SEPs in managing CSM, including detecting clinically silent lesions. Also, there was no significant difference between the incidences of median and tibial SEP abnormalities (49% against 47%).

Several factors have been reported to affect surgical outcome in CSM. These are preoperative neurological deficits, age, and several neuro-image findings including cervical curvature, compression ratio of the spinal cord, and presence of intramedullary high signal intensity on T2 weighted MRI. One recent study showed that the different patterns of intramedullary hypertensive T2 signals might be associated with different surgical outcome. Although many studies have shown that evoked potentials are useful in detecting myelopathy in CSM, the prognostic value of these tests has rarely been examined in detail. Tavy et al. reported that CMCT in the hands correlated significantly with the clinical disability. Restuccia et al. found that clinical recovery was accompanied by SEP improvement and that this improvement was more pronounced in patients with isolated loss of N13. In our study, age was an important prognostic factor for surgical outcome. Besides, both median and tibial SEPs correlated well with the severity of myelopathy whereas normal median SEPs correlated with good surgical outcome. It was noted that arm SEP, rather than leg SEP, provides more information about the segmental level of dorsal column compression in CSM. However, it is unclear why median SEPs correlated with the recovery ratio whereas tibial SEPs did not.

In CSM, the clinical signs of motor dysfunction are usually more obvious and more frequently encountered than those of sensory dysfunction. Previous studies, as well as our own, have shown that pure motor dysfunction is not uncommon whereas isolated posterior column involvement is only rarely seen in spondylotic myelopathy. This clinical characteristic is reflected in our evoked potential study: MEPs were more sensitive than SEPs in detecting myelopathy in patients with chronic compressive myelopathy (90% against 65%). Our finding is consistent with the results of Maertens de Noordhout et al. The different sensitivity of MEPs and SEPs was probably caused by the different mechanisms of compression and the different locations of the lesions. Therefore, in our opinion, evaluations of motor dysfunction should include studies of both MEPs and SEPs.
Evoked potentials in cervical spondylotic myelopathy

SEP to extrudal compression is caused by the anatomical position of ventrolateral and dorsal columns. In CSM, the degenerative and herniated cervical discs bulged posteriorly into the spinal canal, resulting in a reduction of the anteroposterior diameter of the spinal canal and a decreased vascular supply due to the compression of the anterior spinal artery. Hence, the ventrolaterial column is more vulnerable than the dorsal column in CSM. In animal studies, differential changes in axonal calibre on different spinal cord columns. As a consequence, abnormal SEPs would be less favourable.

Competing interests: none

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