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Original Article

Motor evoked potentials elicited from erector spinae muscles in patients with thoracic myelopathy

S Taniguchi*,[1], T Tani[1], T Ushida[1] and H Yamamoto[1]

[1]*Department of Orthopaedic Surgery, Kochi Medical School, Nankoku City, Kochi, Japan*

Study design: A prospective study was conducted to test the utility of the motor evoked potentials (MEPs) from the erector spinae muscles as a measure to estimate the motor level of thoracic compression myelopathies in 13 consecutive patients.
Objective: To confirm whether this test is a useful addition to the neurological examination in non-invasively localizing the level responsible for the main functional change in mild to moderate thoracic myelopathy.
Setting: Department of Orthopaedic Surgery, Kochi Medical School, Kochi, Japan.
Methods: This electrophysiological study consisted of transcranial magnetic stimulation (TMS) of the brain and surface recording of MEPs from voluntarily contracted erector spinae muscles with the patient in the prone position. The recordings were obtained unilaterally from the same side as the lower-limb affected at 12 serial interspinous levels from T5-6 to L4-5. The results were compared to the MEP data from normal subjects and to neurological and MRI findings.
Results: Multisegmental MEP studies demonstrated a focal conduction block in one patient, a single site of conduction delay in seven, and normal conduction in five. The conduction block was characterized by an abrupt reduction in amplitude of the MEPs. Examination of the sites conduction delay showed that the latency difference between the two adjacent levels was longer than the corresponding normal upper limit by 1.00 ± 0.40 ms (range, 0.62–1.61 ms). The site of conduction abnormalities approximated to the compressive lesion site shown by MRI. All five patients with false-negative MEP findings had the lesion site at or caudal to the T10-11 vertebral level.
Conclusion: This method has the advantage of instantaneously testing multisegments of the thoracic spinal cord. The technique is of particular value in estimating the motor level of the lesions rostral to T10-11 vertebral level, which can not be achieved by clinical examinations or MEP recordings from the lower limb.
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Keywords: motor evoked potential; transcranial magnetic stimulation; thoracic myelopathy; erector spinae muscle

Introduction

Surgical intervention for multilevel compression of the spinal cord requires localization of the site responsible for the main functional change. An electrophysiological test consisting of transcranial magnetic stimulation (TMS) of the brain and surface recording of the motor evoked potential (MEP) from the upper limb muscles helps localize the motor lesion to the upper or lower cervical cord in cervical spondylotic myelopathy.[1–4] Similarly, MEP recordings from the erector spinae muscles after TMS can be a useful diagnostic tool in non-invasively localizing the thoracic cord lesion. The technique is of particular value in non-traumatic spinal cord disorders of mild to moderate degree, where the neurological examination often fails to estimate the level of thoracic cord involvement. An MEP study for the lower limb muscles is also insufficient for this purpose, because it would serve neither to localize the thoracic lesion site nor to exclude the MEP abnormalities caused by a coexisting lumbar lesion.

As shown previously,[5] voluntary back muscle contraction of approximately 20% of maximal force is necessary to consistently record the MEPs from the erector spinae muscles at multiple spinal levels. With such facilitation, we have tested patients with

*Correspondence: S Taniguchi, Department of Orthopaedic Surgery, Kochi Medical School, Kohasu Oko-cho, Nankoku city, Kochi 783-8505, Japan

compression myelopathy (established radiologically) of the thoracic cord, by recording the MEPs from the erector spinae muscles at 12 serial vertebral levels, the results are compared with the data from normal subjects which we have previously published.[5]

Materials and method

Patients

From 1996 to 2000, 13 patients (eight men), 26–72 years of age (mean, 55 years), with thoracic myelopathy underwent MEP studies of the erector spinae muscles. Myelopathy resulted from ossified ligamentum flavum (six patients), disc herniation (five patients) and syringomyelia (two patients). We excluded five patients with severe paraparesis who were unable to raise the upper back and to lift the knees off the table in the prone position for the MEP recordings (see the section of MEP study of erector spinae muscles).

Clinical picture

Based on the impairment scale developed by the American Spinal Injury Association (ASIA),[6] more than half of the lower-limb key muscles were of a grade greater than '3' in eight patients (D), while less than half of those muscles showed such grades in five patients (C). ASIA motor score (maximum 100 points) in 13 patients averaged 91 points (range, 66–100 points). ASIA sensory score (maximum 112 points) averaged 97 points (range, 80–112 points) for light touch and 96 points (range, 68–112) for pin prick. Weakness of the abdominal muscles was not severe enough to demonstrate Beevor's sign in every patient (Table 1). According to the functional scale developed by the Japanese Orthopaedic Association, eight patients had bladder symptoms; three complaining of hesitancy and urgency and five of retention and incontinence.

Stretch reflexes were generally hyperactive in the lower limbs with normal upper limb reflexes, except for diminished response for the knee reflex and the ankle reflex in three patients each. Extensor plantar responses were elicited in six patients.

MEP study of erector spinae muscles

TMS of the brain was achieved with a magnetic stimulator (Mag Lite, Dantec Medical, Skovlunde, Denmark). The 14 cm-diameter round coil, tangentially positioned and centered over the vertex, delivered a stimulus with an intensity of 90% to 100% of the maximal output.

The recording procedure with a standardized facilitation technique was the same as described earlier.[5,7] Briefly, the patient, lying prone on a table with an overhead frame to which a strain-gauge transducer was attached, raised his or her upper back against the transducer head. The knees were lifted off the table to activate the lumbar erector spinae muscles.

Table 1 Data for 13 patients with thoracic myelopathy

						ASIA scoring system										
									Motor score			*Light touch score*			*Pin prick score*	
Patient no.	*Age (years)*	*Gender*	*Compression lesion*	*Intervertebral disc levels of cord compression on MRI*	*Interspinous level of abnormal MEP*	*Motor level*	*Sensory level*	*Impairment scale*	*R (50)*	*L (50)*	*Total (100)*	*R (56)*	*L (56)*	*Total (112)*	*R (56)*	*L (56)*	*Total (112)*
1	57	F	OLF	T11–12	T11–12	L2	L2	D	50	46	96	52	50	102	52	50	102
2	62	M	TDH	T10–11	T11–12	L2	T12	C	33	33	66	46	46	92	46	46	92
3	37	M	TDH	T8–9	T9–10	L2	T10	D	40	46	86	56	47	103	56	47	103
4	72	F	TDH	T10–11	none	L2	L2	C	46	37	83	56	51	107	56	51	107
5	62	M	Syringomyelia	T3–12	T7–8	L2	T12	C	48	41	89	56	46	102	56	46	102
6	56	F	OLF	T10–11	none	L2	T11	D	48	49	97	45	45	90	45	45	90
7	26	F	Syringomyelia	C7-L2	T12–L1	L2	T3	C	45	45	90	34	46	80	24	44	68
8	72	M	OLF	T10–11	T10–11	normal	normal	D	50	50	100	56	56	112	56	56	112
9	49	M	OLF	T12–L2	none	S1	L5	D	48	48	96	50	51	101	49	51	100
10	51	M	OLF	T8–10	T8–9	L5	T11	D	49	49	98	45	45	90	45	45	90
11	52	M	OLF	T6–7,8–9,10–11	T11–12	normal	T11	D	50	50	100	47	45	92	47	45	92
12	59	F	TDH	T11–12	none	L2	L1	D	48	48	96	44	46	90	44	46	90
13	57	M	TDH	T11–12	none	L2	L1	C	37	48	85	51	52	103	52	53	105

The patients are listed in order of date of MEP study. OLF, ossified ligamentum flavum; TDH, thoracic disc herniation

A digital readout of the back extensor muscle force allowed the patient to adjust voluntary muscle contraction to 20% of the maximum effort at the moment of magnetic stimulation. Multiple recording covered 12 interspinous levels from T5-6 through L4-5 were identified by palpation and marked. An anteroposterior radiograph was then taken for confirmation. In some cases, repositioning of the marks was needed under fluoroscopic guidance. We tested unilateral erector spinae muscles from the same side as the most severely affected lower-limb, minimizing the burden on the part of the patient who has to maintain steady back muscle contraction during the procedure. Active disk electrodes were placed over the erector spinae muscles, 3.5 cm lateral from each of the marks, with the reference electrodes placed 9 cm further laterally at the same levels. An 8-channel evoked response analyzer (Evomatic 8000, Dantec Medical) allowed simultaneous recording of eight out of 12 pairs of electrodes for each stimulation. The trial was repeated to obtain several traces from each pair of electrodes for clear definition of the latency to the onset of the major negative potential despite the presence of randomly discharging motor unit activities.

We measured the latency to the onset of the major negative MEP response, and then calculated the latency difference between two adjacent levels from T5-T6/T6-T7 through L3-4/L4-5. Each value was compared to the upper limit of normal latency difference, defined as a mean plus three standard deviations (+3 SD) in 15 healthy adults.[5] The upper normal limits were as follows: 0.84 ms for T5-6/T6-7, 1.53 ms for T6-7/T7-8, 2.23 ms for T7-8/T8-9, 2.97 ms for T8-9/T9-10, 2.63 ms for T9-10/T10-11, 2.39 ms for T10-11/T11-12, 3.48 ms for T11-12/T12-L1, 3.46 ms for T12-L1/L1-2, 3.38 ms for L1-2/L2-3, 2.79 ms for L2-3/L3-4, and 2.89 ms for L3-4/L4-5.

Radiological evaluation

All patients underwent surface coil MRI examination of the thoracic cord with a superconducting system (1.5T MRT-200; Toshiba Corp, Tokyo, Japan, or 1.5T Signa; GE Corp, Milwaukee, USA). The MRI protocol included sagittal and axial T1-weighted images, and sagittal T2-weighted images, with a slice thickness of 5 mm.

All but one had CT scans, with a CT-9800 machine (GE Corp, Milwaukee, USA). The axial sections perpendicular to the long axis of the thoracic canal were obtained at midvertebral and intervertebral levels.

Results

TMS evoked reproducible MEPs at all spinal levels from T5-6 through L4-5 in all 13 patients but one. In

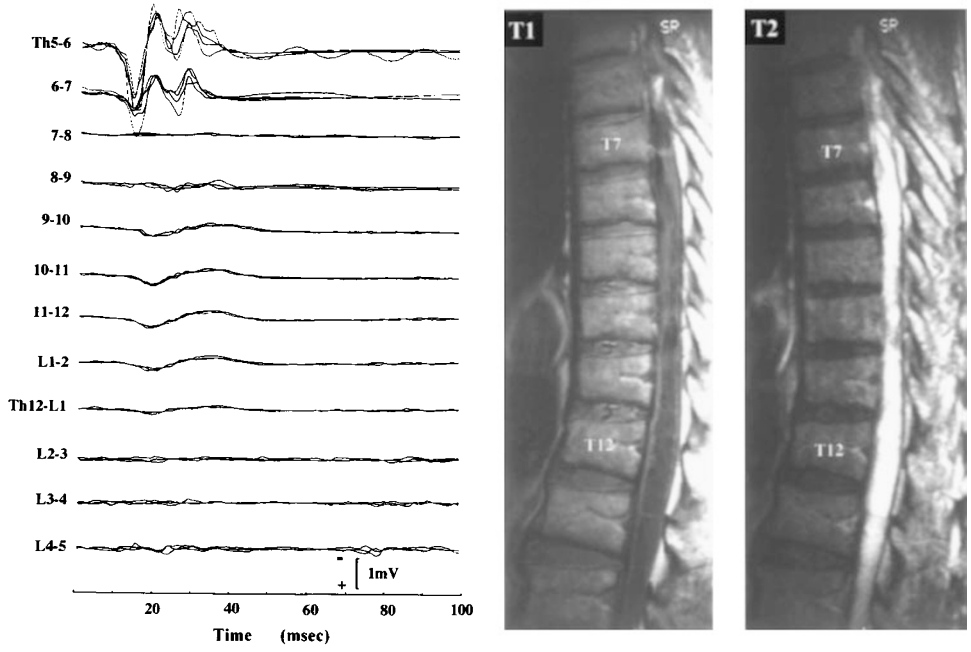

Figure 1 Case 5. MEPs recorded from left erector spinae muscles after TMS of the brain (left), and sagittal T1 and T2 weighted MRIs of the thoracic spinal cord (right) in a 62 year old man with paraparesis. The ASIA impairment scale was C with the motor level of L2 and the sensory level of T12. The incremental MEP study revealed normal responses at T5-6 and T6-7, followed by absent or very small responses at more caudal recording sites, indicating a conduction block in the thoracic cord near the T7-8 level. The MRI demonstrated a septated syrinx of the thoracic cord extending from the T12 up to the T3 vertebral level

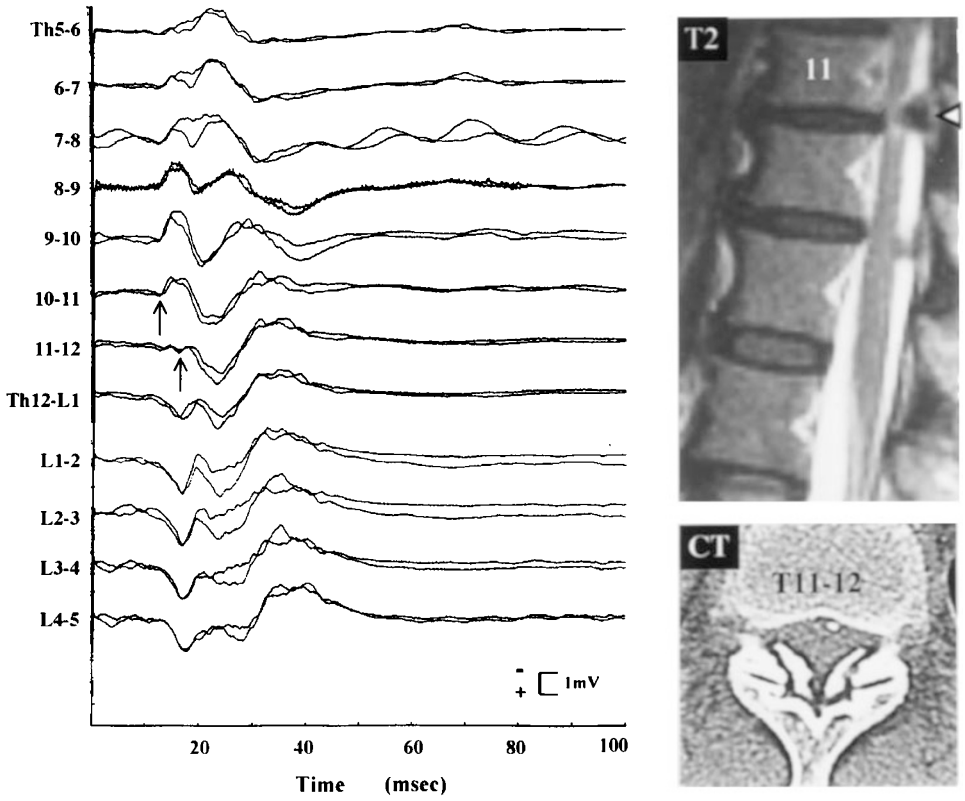

Figure 2 Case 1. MEPs recorded from left erector spinae muscles after TMS of the brain (left), and sagittal T2 weighted MRI of the thoracic spinal cord and CT scan at T11-12 level (right) in a 57 year old woman with paraparesis. The ASIA impairment scale was D with the motor level of L2 and the sensory level of L2. The incremental MEP study uncovered a localized conduction delay as evidenced by a disproportionate latency increase of 3.90 ms from T10-11 to T11-12 compared with the normal upper limit of 2.39 ms (mean $\pm$ 3 SD in a healthy population) across the segment (arrows). The MRI and CT scanning demonstrated intense cord compression caused by OLF at T11-T12 intervertebral disc level (arrow head), which is located immediately rostral to the T11-12 interspinous level of delayed MEP

the one exceptional patient (case 5), incremental recording revealed normal responses at T5-6 and T6-7, followed by absent or very small responses at more caudal recording sites, indicating a conduction block in the thoracic spinal cord near the T7-8 level (Figure 1).

Of the 12 patients with relatively well preserved MEPs at all spinal levels tested, seven showed a single segment of longer latency difference than the normal upper limit, three between T10-11 and T11-12 (Figure 2 and 4), and 1 each between T7-8 and T8-9, T8-9 and T9-10 (Figure 3), T9-10 and T10-11, and T11-12 and T12-L1. The focal latency change across these affected segments was longer than the corresponding normal upper limit by 1.00 ± 0.40 (range, 0.62 – 1.61) ms. In contrast, the latency difference between more rostral or caudal two adjacent levels was well within the normal range of variability.

In the remaining five patients, despite the clinical signs and symptoms of myelopathy, there was a predictable latency change from T6-7 through L4-5 (Table 1).

Neurological correlation

The motor examination determined the motor level in all patients but two using the lower-limb key muscles, because Beevor's sign was negative in every patient (Table 1). Therefore, the most rostral motor level was L2. In two patients (cases 8 and 11), all key muscles were normal despite signs of spinal cord compromise, but the MEP study revealed conduction delay at T10-11 in case 8, and at T11-12 in case 11. The sensory examination determined the sensory level in all but one using the dermatomal key points (Table 1). The sensory level approximated to the level of the MEP abnormality in five patients (cases 1, 2, 3, 10 and 11), whereas there was a discrepancy between the two levels in two patients with syringomyelia (cases 5 and 7).

Radiological correlation

MRIs and CT scans revealed cord compression from various causes which were potentially responsible for the symptoms under consideration. Of 13 patients, six had ossified ligamentum flavum (OLF), five thoracic

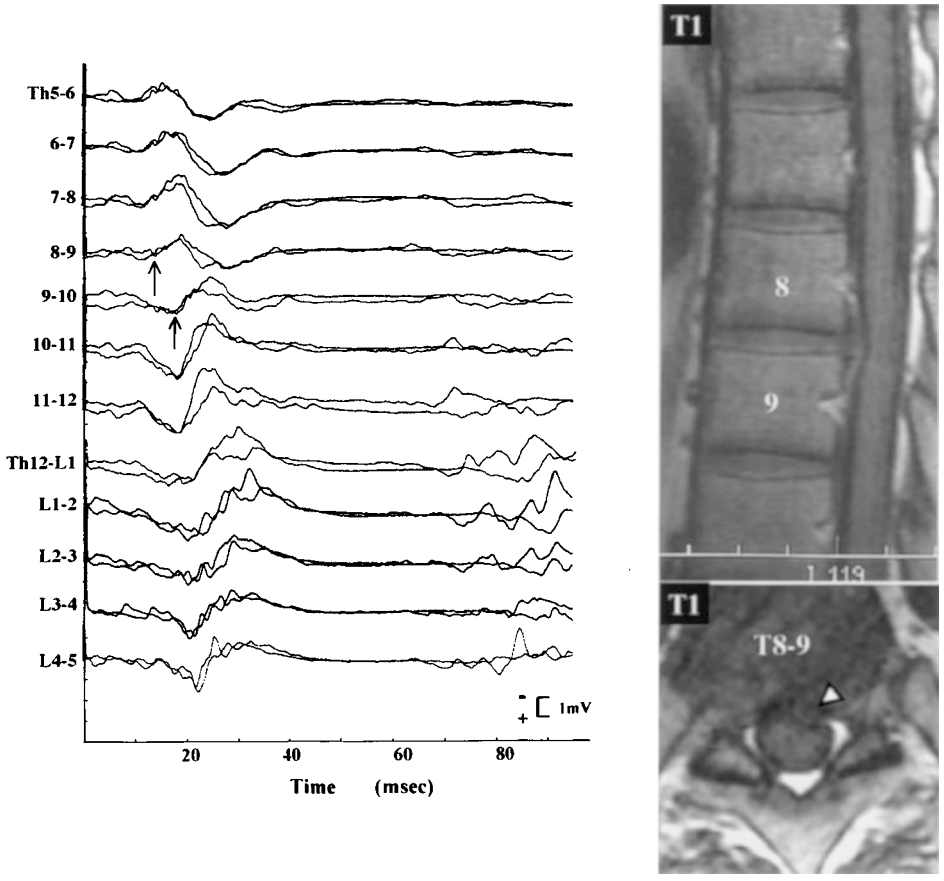

Figure 3 Case 3. MEPs recorded from left erector spinae muscles after TMS (left), and sagittal T1 weighted MRI of the thoracic spinal cord and axial T1 weighted MRI at T8-9 level (right) in a 37 year old man with paraparesis. The ASIA impairment scale was D with the motor level of L2 and the sensory level of T10. The incremental MEP study uncovered a localized conduction delay as evidenced by a disproportionate latency increase of 3.61 ms from T8-9 to T9-10 compared with the normal upper limit of 2.97 ms (mean $\pm$ 3 SD in a healthy population) across the segment (arrows). The MRI showed distortion of the spinal cord caused by a herniated disc at the T8-9 intervertebral disc level (arrow head), which is located one level rostral to the T9-10 interspinous level of the delayed MEP

disc herniation (TDH), and two syringomyelia. The OLF involved a single level in three patients, two levels in two, and three levels in one, whereas the TDH involved a single level in all five patients. Syringomyelia of the thoracic cord extended from T12 up to T3 in one patient and from L2 up to C7 vertebral level in the other (Table 1).

The sites of cord compression shown by MRI approximated to those predicted by abnormal MEPs (Table 1).

The MRI of the patient who showed absent or very small MEPs at and caudal to the T7-8 interspinous level (case 5) demonstrated a septated syrinx of the thoracic cord extending from the T12 up to the T3 vertebral level (Figure 1). In the seven patients with a delayed MEP at single levels, MRIs disclosed cord compression near the level of the delayed MEP; the cord compression involved the same disc level as the corresponding interspinous level of the delayed MEP in four patients (Figure 2) and one level rostral to that level in three (Figures 3 and 4).

Interestingly, all five patients with normal MEP had a compressive lesion at or caudal to the T10-11 vertebral level; two each at T10-11 and T11-12, and one at T12 through L2 (Table 1).

Discussion

The MEP assessment as a means of localizing the site of functional involvement remains largely unexplored in thoracic compression myelopathy in contrast to cervical[1,4,8,9] and lumbar lesions.[10–13] The MEPs recorded from the erector spinae muscles with surface electrodes, as in the present study, originate primarily, if not exclusively, from the long spinal muscles, located more superficially than the short muscles. The long spinal muscles, the longissimus and the iliocostalis, although innervated serially, receive a nerve supply overlapping at least one to two segments caudally and rostrally.[14,15] The polysegmental innervation of the muscles has been shown to cause a disparity between the lowest level of voluntary EMG activities and the corresponding

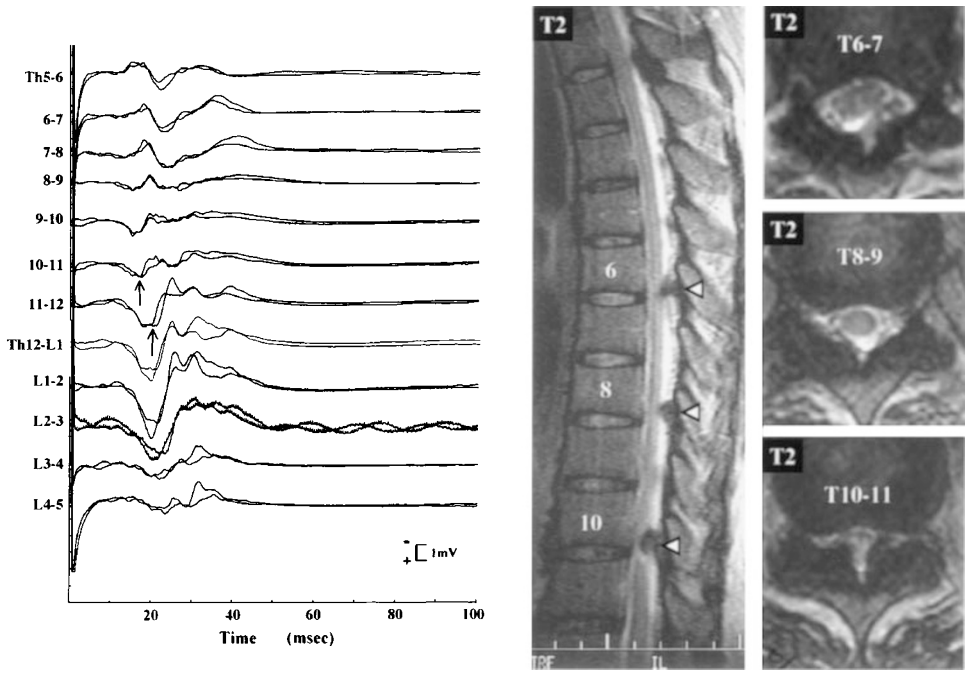

Figure 4 Case 11. MEPs recorded from the left erector spinae muscles after TMS of the brain (left), and sagittal T2 weighted MRI of the thoracic spinal cord and axial T2 weighted MRI at T6-7, T8-9, and T10-11 levels (right) in a 52 year old man with paraparesis. The ASIA impairment scale was D with normal motor function and the sensory level of T11. The incremental MEP study uncovered a localized conduction delay as evidenced by disproportionate latency increase of 4.00 ms from T10-11 to T11-12 compared with the normal upper limit of 2.39 ms (mean $\pm$ 3 SD in a healthy population) across the segment (arrows). The MRI scanning showed cord compression caused by OLF at the T6-7, T8-9 and T10-11 intervertebral disc levels (arrow heads). The cord compression was most prominent at the T10-11 disc level, which is located one level rostral to the T11-12 interspinous level of delayed MEP

vertebral lesion level in complete spinal cord injuries.[14,16] This tendency was more marked for low thoracic and high lumbar vertebral injuries, reflecting the long branches of the thoracic dorsal rami, running caudally and dorsolaterally in an oblique direction.[17,18]

Despite such a complicated innervation of the long spinal muscles, the multisegmental MEP exploration of the muscles showed evidence of focal conduction abnormalities in eight of 13 patients with thoracic compression myelopathy. The site of a sharply localized latency increase or, less frequently, failure of conduction across a cord segment was consistent with the compressive lesion site shown by MRI. In the electrophysiological evaluation of a focal lesion, inclusion of the unaffected segment dilutes the effect of slowing at the lesion site and lowers the sensitivity of the test. Therefore, the multisegmental recording with the latency analysis of successive responses helps isolate conduction abnormalities that may otherwise escape detection. The conduction abnormalities of the motor pathway revealed in this study can be a result of the pathologic changes in the spinal cord, which may include compression-induced local ischemia and segmental demyelination in the corticospinal tract, with the gray matter involvement causing loss of large anterior horn cell with the fast-conducting lower motor neurons.

All five patients with normal MEPs had the lesion site at or caudal to the T10-11 vertebral level as shown by MRI, indicating that the false-negative MEP findings may have resulted from the considerable overlap in segmental representation of the long spinal muscles for lower thoracic spinal nerves. In fact, according to the systematic dissection of the erector spinae, the fleshy thoracic fibers of longissimus and iliocostalis extend down to L3 vertebral level, covering the intrinsic lumbar fibers.[19] This anatomical arrangement implies that, even in the lumbar region, the strongest surface MEP would arise from the thoracic components of longissimus and iliocostalis.[19] Nevertheless, this method has the advantage of instantaneously testing the multisegments of the thoracic spinal cord with painless excitation of the motor system. The technique is of particular value in estimating the motor level of the lesions rostral to T10-11 vertebral level, which can not be achieved by clinical examinations or MEP recordings from the lower limb.

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