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Corticospinal tract conduction block results in the prolongation of central motor conduction time in compressive cervical myelopathy

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Abstract

Objective: The objective of this study was to analyze corticospinal function in patients with compressive cervical myelopathy and to elucidate the mechanism underlying its prolonged central motor conduction time (CMCT).

Methods: Motor evoked potentials following transcranial magnetic stimulation (TMS) and peripheral conduction time in the ulnar and tibial nerves following electrical stimulation were measured from the abductor digiti minimi (ADM) and abductor hallucis (AH) muscles in 24 patients with compressive cervical myelopathy and used to calculate CMCT. Spinal cord evoked potentials (SCEPs) following transcranial electric stimulation were recorded intraoperatively from the C2-3 to C6-7 intervertebral levels. Correlations between prolonged CMCT and SCEP values were then estimated.

Results: The shorter/longer CMCT between the patients' right and left ADM and AH were $8.5 \pm 2.9/11.5 \pm 3.3$ and $16.2 \pm 3.1/18.4 \pm 3.3$ ms, respectively (mean $\pm$ SD). The SCEPs amplitude at C6-7, compared to C2-3, was $25.7 \pm 21.0\%$. The attenuation of SCEP amplitude, but not latency, at C6-7 correlated significantly with CMCT prolongation.

Conclusions: Our data support the view that CMCT prolongation is primarily due to corticospinal conduction block, rather than conduction delay.

Significance: Insight was provided into the mechanism of corticospinal dysfunction in compressive cervical myelopathy.

Keywords: Motor evoked potentials; Central motor conduction time; Spinal cord evoked potentials; Cervical myelopathy; Transcranial magnetic stimulation

1. Introduction

Measurement of motor evoked potentials (MEPs) following transcranial magnetic stimulation (TMS) is a noninvasive, useful means to evaluate the electrophysiologic function of the corticospinal tract (Jaskolski et al., 1989; Maertens de Noordhout et al., 1991). In particular, measurement of central motor conduction time (CMCT) can be used to electrophysiologically evaluate corticospinal function in compressive cervical myelopathy (Di Lazzaro et al., 1992; Kaneko et al., 2001; Ofuji et al., 1998; Tavy et al., 1994). In fact, an excellent correlation was reported between magnetic resonance imaging (MRI) findings and CMCT in patients with cervical myelopathy (Lo et al., 2004). Specifically, CMCT was found to be more prolonged in patients who had more severe cervical spinal cord compression as determined by MRI analysis.

CMCT is calculated by subtracting peripheral conduction time (PCT), determined by peripheral nerve stimulation, from MEP latency. Thus, the physiology of prolonged CMCT is complex and there have been only a few reports regarding the mechanism by which it occurs in cervical myelopathy. Kaneko and his colleagues (2001) examined CMCT following TMS, as well as spinal cord evoked potentials (SCEPs) following transcranial electric stimulation (TES) in patients with compressive cervical myelopathy and normal subjects. Their results showed that

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CMCT was prolonged in such patients and that they exhibited a significant attenuation in their SCEP amplitudes following TES, but no significant delay in their SCEP latencies. Thus, they concluded that impaired temporal summation of multiple descending potentials following TMS produced delays in motor neuron firing that contributed to the prolongation of CMCT.

In light of the above data, we hypothesized that there may be a correlation between the degree of conduction block and the prolongation of CMCT in these patients. To examine this, we analyzed a series of 24 compressive cervical myelopathy patients who underwent both MEP recordings for CMCT measurement preoperatively, and SCEP recordings after TES during surgery.

2. Materials and methods

2.1. Patients with compressive cervical myelopathy

Twenty-four patients (9 women and 15 men) with compressive cervical myelopathy that were treated in our department between September 2003 and August 2004 were included in this study. Patients with other brain, thoracic spinal cord, cauda equina, or peripheral nerve disorders were excluded. Their mean age was 65 years (range 36–81 years) and their mean height was 159 cm (range 136–173 cm). All patients provided informed consent prior to the initiation of the study.

All of the above subjects exhibited a sensory disturbance in their upper and/or lower limbs, and had a spastic gait disturbance. The presence of compressive cervical myelopathy was confirmed by neurological testing and MRI, and was found to have been due to either cervical spondylosis (16 patients), ossification of the posterior longitudinal ligament (OPLL; 4 patients), or cervical disc herniation (4 patients). All patients underwent laminoplasty after which some exhibited neurological improvement. None of these subjects exhibited any abnormalities in their sensory or motor peripheral nerve conduction velocities.

T2 weighted (repetition pulse 3000 ms, echo time 120 ms) MRI (SIGNA 1.5 T device; GE Yokogawa Medical Systems, Tokyo, Japan) was used to determine, in a blinded fashion by a roentgenologist and two orthopedic surgeons, the levels at which the spinal cord was most severely compressed. Inter-observer agreement (kappa) vis-à-vis the patients' MRI findings was 0.899; since the agreement ratio was high, one of the three assessments was randomly selected and used for data analysis.

2.2. Measurement of CMCT

Surface recording electrodes were bilaterally placed on the abductor digiti minimi (ADM) and abductor hallucis (AH) muscles using the standard belly-tendon method. TMS was delivered using a round 14 cm outer diameter coil (Model 200; Magstim, Whitland, UK), the center of which was held over the vertex of the cranium when MEP recordings were made from the ADM. A clockwise current in the coil, as viewed from above, was delivered to stimulate the left hemisphere and a counterclockwise current was used to stimulate the right hemisphere. The magnetic stimulus intensity was set at 20% above the threshold for the MEPs. The coil was then shifted anteriorly when the MEP recordings were made from the AH muscles. The MEPs were recorded at least four times; all responses were superimposed and their latencies measured (Fig. 1(a) and (b)).

Compound muscle action potentials (CMAPs) and F-waves were recorded following continuous current stimulation at supramaximal intensity (0.2 ms square wave pulses) of the ulnar and tibial nerves at the wrist and ankle, respectively. Thirty two serial responses were obtained and the shortest F-wave latency was measured.

All muscle responses were recorded using a commercially available system (Viking IV; Nicolet Biomedical, WI, USA) after they traversed a bandpass filter of 0.5–2000 Hz. An epoch of 100 ms after stimulation was digitized at a 5 kHz sampling rate. The peripheral conduction time (PCT), excluding the turnaround time at the spinal motor neuron (1 ms), was calculated from the latencies of the CMAPs and F-waves as follows: (latency of CMAPs + latency of F-waves − 1)/2 (Kimura, 1984). The conduction time from the motor cortex to the spinal motor neurons (i.e. the CMCT) was calculated by subtracting the PCT from the onset latency of the MEPs.

2.3. Recording of the SCEPs

SCEPs were recorded intraoperatively before decompression. Needle electrodes (Needle Electrode; Unique Medical, Tokyo, Japan) were inserted into the ligamentum flavum in the interlaminar space between C2-3 and C6-7, and a reference electrode was placed in the paravertebral muscles. The transcranial stimulating electrodes (Cranial Coil Electrode; Unique Medical, Tokyo, Japan) were placed subcutaneously and were used to stimulate the motor cortex electrically 5 cm lateral and 2 cm anterior to the vertex (Fig. 1c). The anode was placed into the scalp contralateral to the side of the ADM that showed the longer CMCT, i.e. the side which was more severely affected; an anodal TES current was used because it depolarizes axons and cell bodies in the motor cortex more effectively than does a cathodal current. The motor cortex was stimulated transcranially with 0.2 ms square wave pulses using a constant current of 100 mA. The SCEPs were detected from the needle electrodes following TES, and were recorded after they traversed a bandpass filter of 0.5–2000 Hz. An epoch of 50 ms after stimulation was digitized at a 5 kHz sampling rate, and 50 responses were averaged. Latencies and amplitudes of the first negative peaks were measured at each level, according to the method of Kaneko et al. (2001).