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# Motor cortex excitability changes following a lesion in the posterior columns of the cervical spinal cord

Raffaele Nardone [a,*], Stefan Golaszewski [c], Jürgen Bergmann [c], Alessandro Venturi [b], Igor Prünster [d], Arianna Bratti [a], Günther Ladurner [c], Frediano Tezzon [a]

[a] *Department of Neurology, Franz Tappeiner Hospital, Via Rossini 5, 39012 Meran/o (BZ), Italy*
[b] *Department of Radiology, Franz Tappeiner Hospital, Meran/o, Italy*
[c] *Institute of Neurology, Christian Doppler Clinic, Paracelsus Private Medical University, Salzburg, Austria*
[d] *Department of Statistics and Applied Mathematics, Collegio Carlo Alberto and ICER, University of Turin, Torino, Italy*

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**Abstract**

We used transcranial magnetic stimulation (TMS) to explore if an impairment of central sensory function produced by an isolated lesion in the cervical posterior white columns would change motor cortex excitability. Cortical silent period duration was prolonged when compared with the control subjects, while central motor conduction and motor thresholds were in the normal limits. We first demonstrate that the involvement of the ascending proprioceptive sensory pathways in spinal cord diseases may have direct consequences on the activity of intracortical inhibitory interneuronal circuits. These findings further elucidate the role of afferent inputs in motor cortex reorganisation.


*Keywords:* Transcranial magnetic stimulation; Cortical silent period; Posterior columns; Somatosensory afferents

The motor system continuously receives peripheral sensory inputs and uses this information to modulate motor tasks. The interactions between the sensory and the motor system are essential for normal motor performance. Lesion studies have demonstrated that the loss of sensory discrimination abilities affected motor functions in monkeys [21]; clinical experience shows that sensory deficits could impair motor recovery after stroke [24].

Several experimental studies demonstrated that afferent proprioceptive and/or cutaneous inputs can modulate the motor cortex excitability in humans. A conditioning electrical stimulation of a mixed or sensory nerve is able to modulate cortical excitability following the arrival of the afferent volley to the primary sensory cortex [5,12,13,16–18,30,31]. Furthermore, a peripheral sensory nerve block decreases motor excitability of muscle beneath the anesthetic skin [27,29], while an increased motor excitability can be induced by ischaemic nerve block in adjacent non-affected areas [2].

However, much less is known about the consequences of lesions in the central somatosensory pathways on motor cortex excitability.

To determine whether the loss of sensory function due to a selective impairment of ascending pathways in the dorsal columns can also induce changes in the excitability of motor cortex, we used transcranial magnetic stimulation (TMS) in a patient who acutely developed clinical and radiological signs and symptoms of cervical posterior column myelopathy.

The 40-year-old right handed man was referred to our department because of sudden onset of numbness and impaired sensation over both hands and legs.

Clinical examination showed a discrete hypoesthesia, a reduced vibration perception and an impaired position sense on upper and lower limbs. When extending the arms with the finger out stretched and the eyes closed, there was no pronator drift and no downward movement. The patient was significant slower to complete the Nine-Hole-Peg Test, employed to study

* Corresponding author. Tel.: +39 0473 264616; fax: +39 0473 264449.
*E-mail address:* raffaele.nardone@asbmeran-o.it (R. Nardone).

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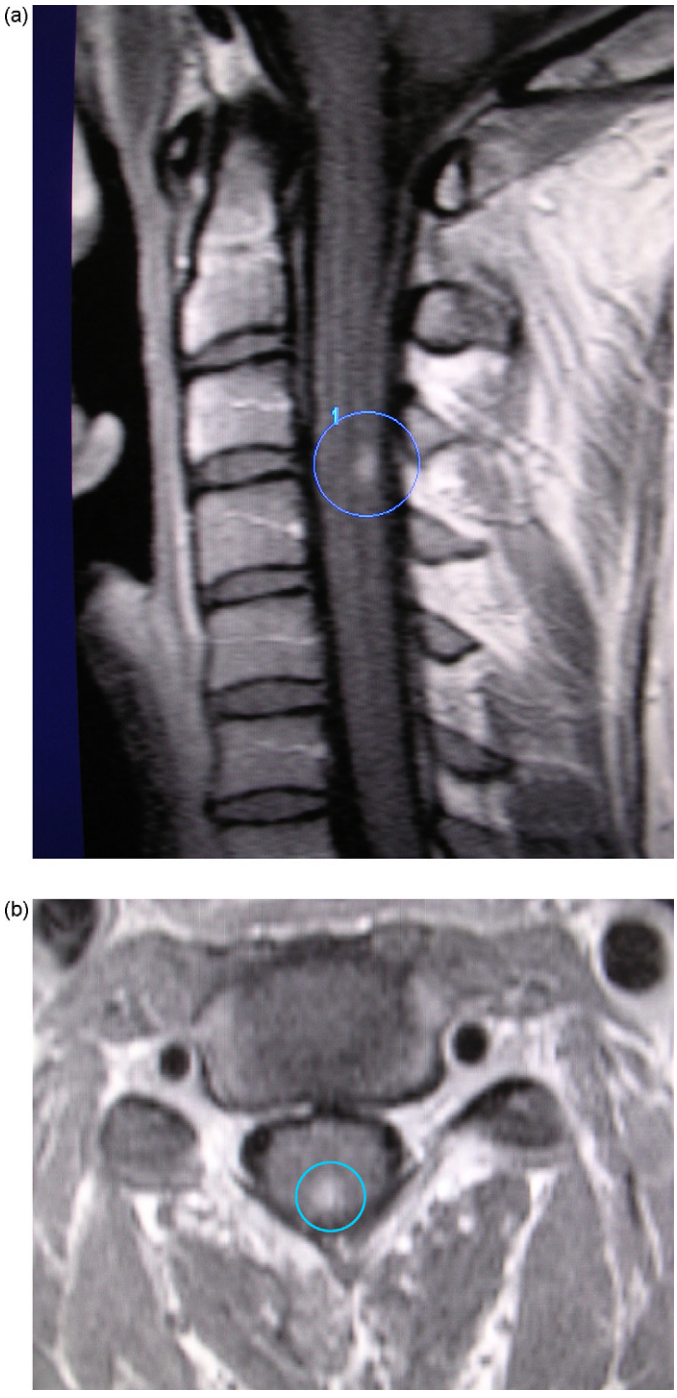


Fig. 1. Sagittal (a) and axial (b) T2-weighted MR images show a bilateral hyperintensity in the posterior columns at level C3–C4.

dexterity [32] with both hands compared to the controls; hand grip strength, as investigated using a mechanical dynamometer, was not significant different when compared to the control group.

Magnetic resonance imaging (MRI) of the cervical spine revealed a hyperintensity in the left white posterior column at level C3–C4. Brain MRI was unremarkable (Fig. 1).

Cerebrospinal fluid (CSF) contained 44 cells per microliter (80% lymphocytes or mononuclear cells). The CSF protein and glucose concentrations were normal. The diagnosis of acute viral myelitis was made, although virus isolation from CSF was unsuccessful.

To assess the impairment of the central sensory pathways, somatosensory evoked potentials (SEPs) were recorded from the upper and lower limbs. Stimulation were delivered at the wrist for median nerve SEPs and at the ankle for tibial nerve SEPs. Stimuli (0.2 ms square pulses) were delivered at a rate of 3 Hz at motor threshold intensity. The electrical impedance was kept below 5 kΩ; the filter bandpass was 30–3000 Hz; the analysis time was 50 ms for median nerve SEPs and 100 ms for tibial nerve SEPs. Two average of 2048 trials were obtained to ensure wave reproducibility. The median nerve SEP were recorded using a four-channel montage (EPi-EPc; Cv7-AC; Pc-Fz; Pc-EPc) according to the IFCN Guidelines [19]. N9, N13 and N20 peak latencies and peak-to-peak amplitude of N20–P25, using Fz reference, were measured. Plexus-cord conduction time, by subtracting the N9 and N13 latencies, and central conduction time (CCT), defined as interpeak latency N13–N20, were also considered. The tibial nerve SEPs were recorded by two-channel montage (L1-Um; Cz′-Fz) [19]. Latency at maximum peak for the N22 and P39 waves and P39–N50 peak-to-trough amplitude were analysed; furthermore, the CCT was measured as the difference between the N22 and P39 latencies.

TMS was performed using a High-power Magstim 200 (Magstim Co., Whitland, Dyfed, UK). A figure-of-eight coil (external loop diameter 90 mm) was held over the motor cortex at the optimum scalp position to elicit motor responses in the contralateral first dorsal interosseous (FDI) muscle. Both hemispheres were subsequently examined. The induced current flow in a posterior–anterior direction. Surface muscle responses were obtained via two 9 mm diameter Ag–AgCl electrodes with the active electrode applied over the motor point of the muscle and the reference on the metacarpophalangeal joint of the index finger. Muscle responses were amplified and filtered (bandwidth 3–3000 Hz) by D150 amplifiers (Digitimer, Welwyn Garden City, Herfordshire, UK). We evaluated the following TMS parameters: threshold and amplitude of MEPs, the central motor conduction time (CMCT), the cortical silent period (CSP). Resting motor threshold (RMT) was defined as the minimum stimulus intensity that produced a liminal motor evoked response (about 50 μV in 50% of 10 trials) at rest. Active motor threshold (AMT) was defined as the minimum stimulus intensity that produced a liminal motor evoked response (about 200 μV in 50% of 10 trials) during isometric contraction of the tested muscle at about 10% maximum. We evaluated the excitability of the motor cortex at increasing stimulus intensity by measuring the average response to five stimuli at 100% AMT, 150% AMT and 200% AMT during at 50% maximum contraction. Central motor conduction was calculated by subtracting the peripheral conduction time from spinal cord to muscles from the latency of responses evoked by cortical stimulation with the formula: MEP latency − (F latency + M latency − 1)/2 in ms [28]. The CSP was elicited whilst subjects held a tonic voluntary contraction of approximately 20% of maximum voluntary contraction. Five stimuli at 150% AMT were given. CSP duration was measured from the end of the EMG response to the return of sustained post-stimulus EMG activity. To assess spinal and peripheral motor