Letter to the Editor

Concerning the article by Lotze et al., 2006: Combination of TMS and fMRI reveals a specific pattern of reorganization in M1 in patients after complete spinal cord injury

Phillip Krause, Johann Szecsi and Andreas Straube Department of Neurology, University of Munich, Munich, Germany

Received 8 September 2006 Revised 14 November 2006 / 21 March 2007 Accepted 29 March 2007

We read with interest the article by Lotze, Laubis-Herrmann and Topka (2006) in which they demonstrated an increased inhibition within the corticospinal tract of patients with spinal cord injury (SCI). Inhibition was measured by detecting the duration of the cortical silent period (cSP) of muscles above the level of the SCI (M. biceps brachii and M. abductor pollicis brevis). The cSP was significantly longer in the patients than in healthy subjects.

Recently we also performed a study in a group of SCI patients and recorded the intracortical inhibition (ICI) and facilitation (ICF) using the classical paired-pulse stimulation paradigm released by transcranial magnetic stimulation.

A group of nine SCI patients (mean age of 36 years; range 29 to 46 years) who were all paraplegic at a lower thoracic level, participated in the experiments. In contrast to the experiments of Lotze, we only recorded from small hand muscles, namely the first dorsal interosseus muscle (FDI). The ICI/ICF was recorded by stimulating with two single pulses in a paired interval. The first was a conditioning pulse of subthreshold (80% of resting motor threshold) and the second (test pulse) of suprathreshold (120%) intensities (Weber & Eisen, 2002). Using short (3 ms) interstimulus intervals the conditioning pulse reduces the MEP-response of the test pulse (ICI) and longer intervals (10 ms) facilitate (ICF) it. For measuring the cSP subjects were instructed to precontract the FDI moderately while single stimuli (120% of resting motor threshold) were released on the corresponding motor cortex (Krause, Foerderreuther, & Straube, 2005).

The patient's data were compared with data from a group of healthy subjects (n = 10), who were age and gender matched. This comparison revealed two interesting aspects. First, we can confirm the significantly (p < 0.05) longer cSP in SCI patients, when compared to healthy subjects. The duration of the cSP was around 153 ms at the right hand and 168 ms at the left in patients; durations in healthy subjects were 72 ms and 83 ms respectively.

Second, the measurement of ICI/ICF did not show any statistically significant differences between both groups, SCI patients or healthy subjects. In the ICI paradigm MEP-amplitudes were reduced by around 36% on the right and 23% on the left motor cortex in patients. The inhibition was more pronounced in healthy subjects (52 and 60% for right and left motor cortex). The ICF-paradigm led to an increase of amplitudes of around 75% on the right and 88% on the left cortex in patients. Healthy subjects showed a similar increase of around 71% (right) and 67% (left).

In conclusion, the increased inhibition is existent in proximal (Lotze et al., 2006) but also distal upper limb muscles, which are unaffected, and beyond the SCI level. In the literature it is discussed that the cSP is not only a phenomenon of cortical but also, mainly the first third, is of motor spinal fiber systems (Fuhr, Agostino, & Hallett, 1991). Our results of not significantly different ICI and ICF amplitudes when comparing SCI patients and healthy subjects let us assume that subcortical or spinal fiber systems may play a major role in the development of inhibitory activity within the corticospinal tract in SCI patients. On the one hand, this would be in accordance with the results demonstrated by Lotze et al. (2006). But, we should also consider the small number of patients, so that a larger group might result in statistically significant differences in ICI. This would be in accordance with our data, showing a distinct reduced intracortical inhibition in the group of patients.

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