



Corticomuscular coherence in acute and chronic stroke



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HIGHLIGHTS

- We studied corticomuscular coherence (CMC) in the acute and chronic stroke following up the course of recovery.
- In acute stroke CMC frequency was decreased on the affected side and CMC amplitude was increased on the unaffected side.
- In the chronic period there was no inter-hemispheric difference in CMC parameters.

ABSTRACT

Objective: Motor recovery after stroke is attributed to neuronal plasticity, however not all post-stroke neuronal changes relate to regaining fine motor control. Corticomuscular coherence (CMC) is a measure allowing to trace neuronal reorganizations which are functionally relevant for motor recovery. Contrary to previous studies which were performed only in chronic stage, we measured CMC in patients with stroke at both acute and chronic stroke stages.

Methods: For the detection of CMC we used multichannel EEG and EMG recordings along with an optimization algorithm for the detection of corticomuscular interactions.

Results: In acute stroke, the CMC amplitude was larger on the unaffected side compared to the affected side and also larger compared to the unaffected side in the chronic period. Additionally, CMC peak frequencies on both sides decreased in the acute compared to the chronic period and to control subjects. In chronic stage, there were no inter-hemispheric or group differences in CMC amplitude or frequency.

Conclusions: The changes in CMC parameters in acute stroke could result from a temporary decrease in inhibition, which normalizes in the course of recovery. As all patients showed very good motor recovery, the modulation of CMC amplitude and frequency over time might thus reflect the process of motor recovery.

Significance: We demonstrate for the first time the dynamical changes of corticomuscular interaction both at acute and chronic stage of stroke.

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1. Introduction

Motor dysfunction is the most frequent consequence of stroke (Rathore et al., 2002) that dramatically affects the everyday life

of patients. Appropriate treatment and rehabilitation procedures of motor paresis strongly depend on our understanding of the neuronal processes related to recovery of normal motor functioning. It is generally believed that motor recovery after stroke is due to massive neuronal reorganization occurring both locally and remotely to the lesion site (Talelli et al., 2006; Jang, 2007; Nudo, 2007; Grefkes and Fink, 2011). Thus, new areas that were previously not engaged are recruited in a motor activity. Neuroimaging studies reported the involvement of ipsi- and contralesional brain regions in the recovery process (Ward, 2005; Dancause, 2006; Nudo, 2007).

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However, not every change in brain activation and wiring after stroke can be considered as functionally relevant for re-establishing motor performance. Some alterations in the neuronal dynamics can be unspecific/maladaptive – as a consequence of compensatory movements, ipsilateral involvement and inter-hemispheric competitive interaction (Takeuchi and Izumi, 2012). Maladaptive plasticity interferes with motor recovery and should be differentiated from neuronal processes which are specifically associated with regaining normal motor control. Studies utilizing neuroimaging techniques such as fMRI and PET provide rather an indirect reference with respect to the involvement of activated cortical areas into the recovery processes (Calautti and Baron, 2003; Assaf and Pasternak, 2008).

In the present study we use corticomuscular coherence (CMC) as a tool to identify functionally relevant contributions of reorganized cortical areas to motor recovery. CMC is a well-established neurophysiological measure, which indicates the amount of synchronization between cortical and spinal cord activities during the execution of a movement (Brown et al., 1998; Mima and Hallert, 1999; Salenius and Hari, 2003). CMC appears predominantly during periods of isometric contraction (Kilner et al., 2000; Riddle and Baker, 2006) and reaches its maximum in the beta frequency range (16–32 Hz) over the primary sensorimotor cortices contralateral to the innervated limb (Salenius et al., 1997; Tsujimoto et al., 2009; Witham et al., 2010).

There are only a few stroke-related CMC studies (Mima et al., 2001; Braun et al., 2007; Fang et al., 2009; Meng et al., 2009; Graziadio et al., 2012) and all of them were performed at the chronic stage, mostly at least 1 year after the stroke when many compensatory processes already took place (Rijntjes, 2006). Currently, longitudinal CMC studies following stroke patients from acute to chronic period are missing. Such studies could provide new insight into the temporal evolution of corticomuscular interaction after stroke and add to the understanding of mechanisms underlying motor recovery. The present study demonstrates for the first time the changes in the dynamics of corticomuscular interaction both at acute and early chronic stage of stroke.

2. Materials and methods

The experimental protocol was approved by the Institutional Review Board of the Charité, Berlin, and the subjects gave their written informed consent prior to the experiments. All subjects were right-handed according to the Edinburgh Handedness Inventory (Oldfield, 1971) and had normal or corrected to normal vision.

2.1. Patients

Eleven ischemic stroke patients (5 female, mean age \pm SD: 71.7 ± 11.3 years) with hemiparesis were recruited in 12 months of the study. Only patients with first ever acute ischemic lesions localized in cortical or subcortical regions were included in the study. The lesion location was confirmed with MRI; four patients had left, seven right hemispheric strokes. Exclusion criteria were multiple scattered lesions, previous stroke or lesion, muscle disorders, peripheral neuropathy, hemorrhagic stroke, cognitive impairment and complete plegia. The mean time for the recordings was 3.5 (range: 2–5) days after the stroke. Motor strength of the target muscles (abductor pollicis brevis, APB) was graded according to the Medical Research Council (MRC) scale. To be selected for the study, the patients had to have moderate to severe hemiparesis at the onset of the stroke. On the day of experiment, patients had regained their muscle strength such that it was greater than 3 on the MRC scale except for one patient. The details of the patients are summarized in Table 1.

The same group of patients was contacted 6 months post-stroke and asked to participate in the follow-up study. Seven patients agreed and were recorded 194.6 (range: 174–250) days after the first experiment. In the follow-up period the force level was assessed again and found to be 5 (on the MRC scale) for all patients.

2.2. Controls

Fourteen healthy subjects (4 female, mean age \pm SD: 51.7 ± 10.35 years) without any history of neurological or psychiatric disease served as control.

2.3. Paradigm

We used a digit displacement paradigm, which includes the manipulation of a compliant object because CMC was found to be larger when the task involved a compliant object compared to a simple isometric condition (Kilner et al., 2000; Riddle and Baker, 2006).

During the experiment the patients stayed in the bed, reclined at 60°. Control subjects were seated in a comfortable chair with their arms resting on the chair handles. The subjects were instructed to press a spring-loaded lever with the left or right thumb with 0.5 N force, requiring a lever displacement of ~ 3.5 cm. The force was measured with a load sensor (FSG15N1A, Honeywell, USA). Visual feedback of the force level was provided on a computer screen as a horizontal bar of varying lateral extent proportional to the exerted force with fixed vertical lines indicating the target value. A cross in the center of the screen served as an eye-fixation point.

The task was performed with each hand separately and the hand order was counter-balanced between the subjects. The subjects were instructed to reach the required force level as fast as possible after a single tone and hold it constant until the presentation of a double tone. Subjects performed 4 blocks of 25 trials per hand with 60 s of rest between the blocks. Each trial lasted 9 s consisting of 5 s pressing and 4 s rest.

2.4. Data acquisition

EEG and EMG data were acquired with BrainAmp MR-plus (Brain Products, Germany) amplifiers, filtered in the frequency range of 0.015–250 Hz and sampled at 1000 Hz. The voltage resolution for the EEG and EMG channels was 0.1 μ V and 0.5 μ V, respectively.

2.4.1. EEG

During the acquisition, the EEG was referenced to physically linked earlobes and recorded using an EEG cap (61 Ag/AgCl sintered ring electrodes, EasyCap, Germany) with a denser electrode configuration complying with the 10–5 system (Oostenveld and Praamstra, 2001) above the sensorimotor cortices. Ocular artifacts were recorded with two electrodes placed on the right zygomatic and supraorbital processes.

2.4.2. EMG

EMG was recorded from the abductor pollicis brevis (APB) muscle with three EMG electrodes (Ag/AgCl sintered electrodes 4 mm in diameter) over the thenar side of each hand. The skin surface was abraded with NuPrep (Weaver & Co., USA) before the electrode application. The electrode–skin conductive contact was established with Ten20 electrode paste (Weaver & Co., USA) and the electrodes were secured to the skin with adhesive medical tape.

An EMG reference electrode was placed on the styloid process of the ulnar bone and a ground electrode on the inner surface of

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