

## Post-movement beta synchronization after kinesthetic illusion, active and passive movements

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### Abstract

After the completion of a voluntary movement or in response to somatosensory stimulation, a short-lasting burst of beta oscillations (post movement beta ERS, beta rebound) can be observed. In the present study, we investigated if this is also true for the illusion of movements, induced by a vibration at 80 Hz on the biceps tendon. We compared the post-movement synchronization of EEG beta rhythms induced by active and passive movements and illusion in eight right-handed healthy subjects. As a result, a short-lasting post-movement beta ERS was present over motor areas after both active and passive and also after illusion of movement in all subjects. These results suggested a possible role of MI and the somatosensory cortex in the somatic perception of limb movement in humans.

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### 1. Introduction

Different cerebral rhythms, which display modality specific modulation, characterize the brain activity of sensory and motor areas during body movements and somatosensory processing. For example, several EEG studies reported that the amplitude of ongoing rhythmic activity in the beta (15–30 Hz) frequency bands can be influenced by active or passive motor activity of distinct body parts. Using the event-related desynchronization/synchronization (ERD/ERS) technique, it has been shown that such movements are preceded by a desynchronization of beta EEG rhythms (Pfurtscheller and Berghold, 1989), beginning over the contralateral central region about 1.5 s before movement onset. After the termination of the movement, beta ERD is followed by a short lasting burst of beta oscillations. Since these bursts of beta oscillations, described as post-movement beta synchronization

(PMBS) or beta rebound (Pfurtscheller et al., 1996, 2005), are most prominent immediately after termination of movement, they have been related to a state of cortical “deactivation” of locally restricted motor networks (Neuper and Pfurtscheller, 2001). Indeed, the beta rebound appears to be maximal over sensorimotor cortical areas involved in movement execution (Salmelin et al., 1995), it has also been suggested to reflect “active inhibition” of motor cortical neurons (Pfurtscheller and Neuper, 1997).

Summarizing the literature the beta rebound was found to have some important features. It shows a somatotopical organization (Salmelin et al., 1995), a frequency-specificity for the hand and foot representation areas (Neuper and Pfurtscheller, 2001) and a maximum which coincides with a reduced excitability of corticospinal neurons (Chen et al., 1998).

For example, Chen et al. (1998) found in several transcranial magnetic stimulation (TMS) studies that cortical activation either by median nerve stimulation or self-paced finger movement is accompanied by a significant decrease of the corticospinal excitability level after termination of the stimulation and the movement, respectively. This phasic post-movement (stimulation) excitability decrease might be associated with the “beta rebound”

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reflecting an active inhibition of motor cortical neurons reported in the EEG (Pfurtscheller et al., 2005; Neuper and Pfurtscheller, 2001) and MEG (Hari, 1995; Hari et al., 2000) as well.

Furthermore, EEG and MEG (magneto-encephalographic) studies have demonstrated that median nerve stimulation or mechanical stimulation of the finger can induce a beta burst (Salmelin and Hari, 1994; Salenius et al., 1997; Pfurtscheller et al., 2002; Stancák et al., 2003). In these studies, a beta rebound could have been observed which showed the same characteristics as the beta rebound induced by voluntary finger movement. This suggests that beta ERS could also reflect cortical processing of sensory inputs from the muscles, that is refferent input from the limbs may at least partly account for the beta ERS (Cassim et al., 2001). On the other hand, there is evidence that mental imagination of movement (Neuper et al., 1999; Pfurtscheller et al., 2005) or even passive movements (Alegre et al., 2002; Müller et al., 2003; Cassim et al., 2001) can also produce a reliable beta ERS.

For example, Pfurtscheller et al. (2005) investigated the beta rebound after four different types of motor imagery (left/right hand, both feet, tongue) at the vertex. They found that only both feet motor imagery elicited a constant beta rebound, whereas hand motor imagery was less consistent and in tongue imagery it was totally absent. Thus, the reported findings do not support the hypothesis that the SMA acts as the main source of the midcentral beta rebound (Ohara et al., 2000). Indeed, if this would be the case, one should expect that also tongue or hand motor imagery would induce beta oscillations in the central area. They concluded that the termination of motor cortex activity, independent of whether it follows the actual execution

or just imagination of a movement, may involve at least two networks, one corresponding to the primary motor area and another one in the SMA.

The similarity of observed beta ERS after voluntary movements, median nerve stimulation and imagination provides arguments for the notion that this type of beta oscillation is more likely related to the previous motor cortex activation than to the processing of somatosensory refferences, since the latter are probably not present during motor imagery.

Another kind of motor imagery is the kinesthetic illusion, which is compared to motor imagery less studied in the context of post movement beta synchronization. Kinesthetic illusions, e.g. illusory sensation of limb movement, can be elicited by artificially manipulating proprioceptive pathways through tendon vibration at optimal frequencies at around 80 Hz (Roll and Vedel, 1982; Naito et al., 1999; Goodwin et al., 1972). Subjects experience that the vibrated muscles are being stretched. This kinesthetic illusion is caused because the vibration of the tendon excites the muscle spindles in a manner similar to when the muscle actually stretches (Roll and Vedel, 1982; Roll et al., 1989). The direction of such kinesthetic illusion depends on the anatomical site of the vibrated muscles and corresponds to their lengthening. Naito et al. (1999) and Naito and Ehrsson (2001) found in several positron emission tomography (PET) studies that kinesthetic illusions experienced by the subjects during tendon vibration lead to activation of the contralateral somatosensory cortex as well as the primary motor cortex (MI). Furthermore, they found stronger activation of MI compared to somatosensory cortex. This result together with the findings that MI is also active during passive limb movements

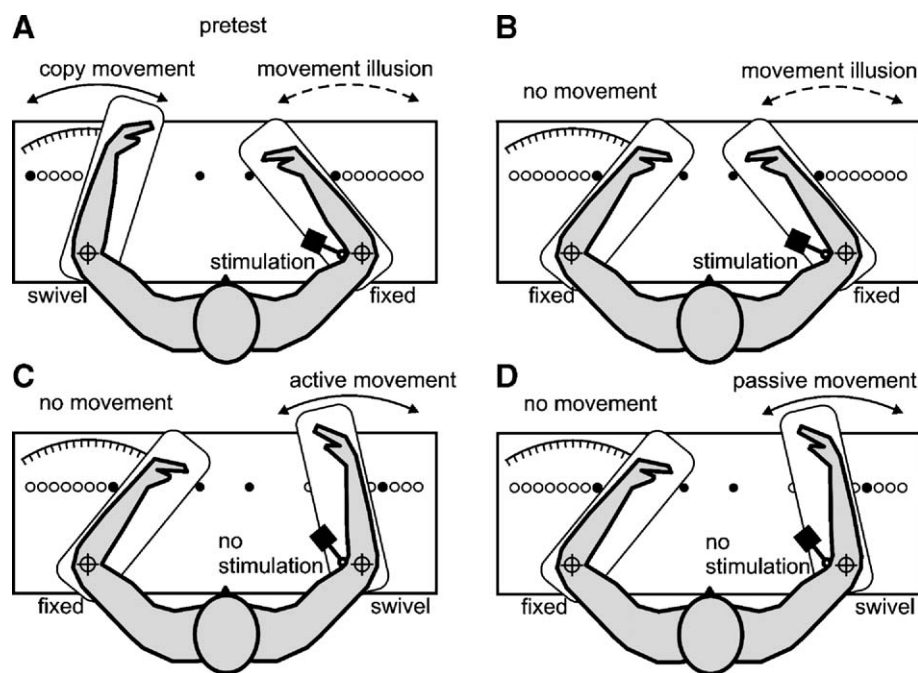


Fig. 1. Experimental setup with three different types of movements. (A) Setup for the pretest. (B) Illusion of movement assembly. Both arms are fixed. (C) Active movement. The right board is movable, whereas the left board is fixed. (D) Passive movement. Passive movement is induced by the investigator by pulling a cord. There is no mechanical stimulation within the active and passive movement condition. Beginning and ending of the movements are indicated by the presentation of noise via a loudspeaker.

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