Effects of cognitive demands on postmovement motor cortical deactivation

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Postmovement β-rebounds induced by different intermovement intervals were investigated using magnetoencephalography in 14 healthy participants to test the hypothesis that postmovement motor cortical deactivation over the primary motor cortex depends on movement-related cognitive demands. Shorter latency and lower amplitude in postmovement β-rebounds over the contralateral primary motor cortex were noted in the short-movement interval movement (repetitive finger lifting). Greater latency span of postmovement β-rebounds jittering using single-trial analysis in the long-movement interval movement (discrete finger lifting) was observed. The study elucidates that the temporal interval between two adjacent movements reflecting different degrees of cognitive demands can affect postmovement motor cortical deactivation in terms of postmovement β-rebounds latency and amplitude, and latency span of postmovement β-rebounds jittering. Postmovement motor cortical deactivation can reflect cognitive demands in addition to motor and somatosensory processing. *NeuroReport* 17:371–375 © 2006 Lippincott Williams & Wilkins.

Keywords: cognitive demands, intermovement interval, latency jittering, magnetoencephalography, postmovement β-rebound, single-trial analysis

Introduction

Cognitive participation in motor control has been documented using various methods including functional magnetic resonance imaging (fMRI) [1,2], positron emission tomography (PET) [3], electroencephalography (EEG) [4], and magnetoencephalography (MEG) [5]. These studies reported that cognitive-motor function is processed in several cortical motor areas involving the primary motor cortex (M1). Recent fMRI and PET studies have demonstrated that cognitive demands during voluntary finger movement are related to the length of the intermovement interval (IMI) [6]. Short-IMI (e.g. 0.5 and 1 s) movement, deemed as automatic timing, recruits motor circuitry comprising M1, premotor cortex, supplementary motor cortex, cingulate cortex, basal ganglia, and thalamus that make feasible neural timing without overt attentional modulation, whereas long-IMI movement (e.g. greater than 8 s), involving cognitively controlled timing mechanisms, draws on multipurpose cognitive circuits, for example, attention and working memory, within additional prefrontal and parietal areas [6]. The subtle differences in cognitive demands between short-IMI and long-IMI movements provide an opportunity to study the effects of cognitive demands on motor control mechanisms underlying self-paced finger movement.

Postmovement motor cortical deactivation (PMMCD) plays an important role in motor performance, primarily affecting movement preparation and execution [7]. EEG studies in Parkinson's disease have revealed that an impaired PMMCD of ongoing movement can result in a poor motor performance because of a protracted preparation of the subsequent movement [7,8]. An MEG study has also indicated that the lack of PMMCD is supported to reflect prolonged excitation of the motor cortex after somatosensory stimuli in patients with progressive myoclonus epilepsy [9]. Nevertheless, the underlying mechanisms of PMMCD are not fully understood. PMMCD is conventionally regarded as either a transient cortical idling [10] or movement-related somatosensory processing [11]. PMMCD can also be proposed to associate with movement-related cognitive load, because the cognitive demands are inevitably engaged in time measurement as indexed by finger movements with different IMIs [6].

PMMCD is usually indexed using the aspects of postmovement β-rebound (PMBR) [7,8,12]. PMBR denotes the maximum energy activity of brain signaling after movement

in the frequency range of 15–30 Hz. PMBR amplitude may reflect the amount of synchronized neuronal ensembles during the cortical deactivation stage and can be suppressed by sustained cortical activation [11,13]. PMBR latency (time interval from movement onset to PMBR) indicates the temporal profile of cortical deactivation [8,12], which could be modulated by the amount of serial information processing [14]. PMBR changes are usually accessed in the M1 as it is the final corticospinal outflow and participates in both motor execution and temporal-related cognitive processes [2,15]. Short-IMI movement, compared with long-IMI, makes few cognitive demands, diminishing the movement-related information processing loads [1,6]; we therefore reason that M1 exhibits shorter latency and/or lower amplitude in PMBR as the IMI becomes shorter.

In this study, whole-head MEG, merited with millisecond-by-millisecond temporal resolution for subtle brain dynamics and fairly good spatial resolution as compared with fMRI and PET, was used to explore the effects of cognitive demands on PMMCD underlying different lengths of IMI. MEG signals were analyzed using Morlet wavelets [16] to study the dynamics of PMBR parameters and the single-trial analytic approach [17] to elucidate subtle differences in individual PMMCD. Here, PMBR was computed using a baseline-free measure to prevent a potential fallacy [18,19] as the baseline activity for short-IMI movements is not available.

Materials and methods
Participants
Fourteen right-handed (according to the Edinburgh Handedness Inventory), healthy volunteers (nine women and five men; mean age 23.7 years; range 19–36 years) without any neurological and orthopedic diseases participated in this study. Written informed consent was obtained from each participant with a protocol approved by the Institutional Review Board and Research Committee.

Motor and control tasks
Motor tasks of discrete movements (DM) and repetitive movements (RM) were conducted using the index fingers of dominant and nondominant hands in different sessions. For the DM, participants performed self-paced, brisk finger lifting with IMIs longer than 8 s. For the two RMs, similar finger movements with rhythmic IMIs of 1 (RM1) and 0.5 (RM2) s were conducted. Finger lifting was followed immediately by brief muscle relaxation. Participants were trained with the monitoring of electromyogram to ensure correct task performance before the MEG experiment. During the MEG experiment, breaks of 2–3 min were given intermittently within the session to prevent muscle fatigue. For a control task, participants were instructed to sit silently with no intended finger movement, and eyes were kept open during the 8-min recording time in two separate sessions.

Recording
MEG signals were recorded in a magnetically shielded room with a whole-head 306-channel neuromagnetometer (Vectoview; Elekta Neuromag; Helsinki, Finland) at a sampling rate of 1 kHz using a 0.03–330 Hz band pass filter. Participants sat comfortably with the forearm pronated on the table, and visually fixated on a stationary cross

displayed 1 m in front of them. Movement onset time was registered using an optical pad (4-D Neuroimaging; Helsinki, Finland). Electrooculograms were recorded to reject online the epochs contaminated by eye movements and/or blinks during the time series ranging from 4 s before to 3 s after movement onset time. Approximately 100 7-s epochs of MEG measurements surviving artifact rejection, out of each session of motor tasks, were obtained for later analysis. Head movement was less than 5 mm between sessions as determined by the head position indicators.

Time–frequency energy analysis
The time–frequency energies were calculated for every single trial at 0.5 Hz intervals in the 10–40 Hz range using Morlet wavelets [16]. Morlet wavelet $W(t, f)$ is a complex function in time (S_t) and frequency domain (S_f) with the shape of a Gaussian-filtered sinusoid around its central frequency (f)

$$W(t,f) = (S_t \sqrt{\pi})^{-1/2} \exp\left(\frac{-t^2}{2S_t^2}\right) \exp(2i\pi ft),$$

where

$$S_t = (2\pi S_f)^{-1}$$

A wavelet family is characterized by a constant ratio (f/S_f), which is defined as 8. The time-varying energy [$E(t, f)$] of an MEG signal for a specific frequency band is the square norm of the convolution of the complex wavelet and the MEG signal (for an extended description of Morlet wavelets, see [16]).

The time–frequency energy, $E_{norm}(t, f)$, of each sensor was then calculated by taking the vector product of energy $E(t, f)$ from two orthogonal planar gradiometers in the time–frequency coefficient matrix. The $E_{norm}(t, f)$s of 102 sensors were averaged across all trials time-locked to movement onset. The averaged $E_{norm}(t, f)$, in the frequency range of 15–30 Hz over the most reactive sensor, was summed within the indicated frequency range for time-varying β energy, summed $E_{norm}(t)$. PMBR latency and amplitude (baseline-free) were calculated from the averaged $E_{norm}(t, f)$. Beta energies in both the baseline stage of DM and the resting stage of control task (-4 to -2.5 s) were calculated from the summed $E_{norm}(t)$. Differences between baseline and resting β energies were obtained to inspect the variation of different quiescent states.

Single-trial analysis
The E_{norm}s over the sensor of maximal PMBR amplitude were sorted on the basis of the latency measured between movement onset and PMBR to inspect the variation of individual trial. Correlation strength between the trial's PMBR latency and its corresponding IMI indexed to the preceding trial was calculated to explore any possible monotypic coupling between these two factors. The differences between maximum and minimum PMBR latencies were obtained to represent the span of latency jittering relative to movement onset.

Statistics
The nonparametric Friedman test was used to compare the differences in amplitudes, latencies and the span of latency jittering of PMBR among DM, RM1 and RM2. Post-hoc testing for these three motor tasks was conducted using the

Holm *t*-test procedure to adjust the significance level of individual comparisons between motor tasks. The Wilcoxon signed rank test was used to analyze paired differences of baseline β energy between DM and the control task. The Spearman rank correlation coefficient (r_S) was used to evaluate the relationship between individual PMBR latency and the preceding trial's IMI. Differences were considered significant at $P < 0.05$.

Results

Beta energy representation

Baseline β energy [median (interquartile range; IQR)] of left hand DM was significantly lower than the resting β energy of the control task [6557 (3093–8606) vs. 8497 (5310–11 330) (fT/cm)^2, $P < 0.05$] (Fig. 1a). Such a phenomenon was not observed for right hand DM ($P = 0.33$).

Beta energy rebound over contralateral M1 was noted after movement for each hand. PMBR amplitudes of right hand movements were 654 (359–913) (fT/cm)^2 for DM, 433 (293–775) (fT/cm)^2 for RM1, and 237 (150–296) (fT/cm)^2 for RM2, while those of left hand movements were 896 (567–1258), 721 (464–893), and 237 (168–383) (fT/cm)^2, respectively (Fig. 1b). PMBR amplitude was significantly lower in RM2 than in DM and RM1 for each hand ($P < 0.05$). The difference in PMBR amplitude between DM and RM1 failed to reach a significant level ($P = 0.052$ and 0.124 for right and left hand movements, respectively). PMBR latencies of right hand movements were 912 (732–1148) ms for DM, 713 (612–804) ms for RM1, and 320 (234–432) ms for RM2,

while those of left hand movements were 1096 (888–1160), 662 (568–748), and 376 (228–454) ms, respectively (Fig. 1c). PMBR latency was significantly shorter in RM2 and longer in DM than in RM1 for each hand ($P < 0.05$).

Single-trial analyses

PMBR latencies over contralateral M1 were jittered in all motor tasks with largest variation for DM (Fig. 2a). Correlation analysis revealed no linear relationship between PMBR latency and the preceding trial's IMI in the three motor tasks for each hand (not shown). The latency spans of PMBR jittering [median (IQR)] of right hand movements were 2522 (2312–2820) ms for DM, 722 (688–766) ms for RM1, and 300 ms for RM2, while those of left hand movements were 2880 (2272–2946), 744 (642–782), and 300 ms, respectively (Fig. 2b). The latency span of PMBR jittering was significantly larger in DM and smaller in RM2 than in RM1 regardless of the hand used ($P < 0.05$).

Discussion

The results demonstrated that timing and extent of the contralateral PMMCD, harboring the amount of serial information processing [14] and the number of synchronized neuronal ensembles [11,13], respectively, can be modulated by different degrees of cognitive demands (e.g. attention and/or working memory for timing). Evidence of fMRI studies suggests that M1 is involved in the working memory for temporal discrimination [15] and attention to action [2]. PMBR, an index of PMMCD, has been proposed
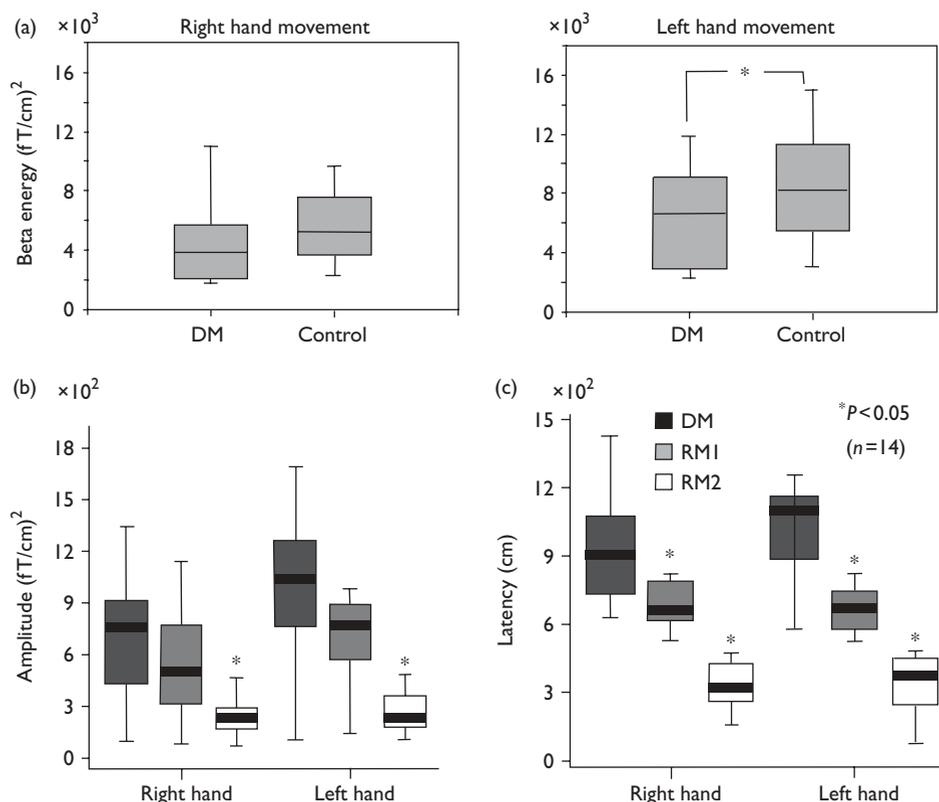
Fig. 1 Comparisons of β energy, PMBR amplitudes and latencies of contralateral MI. (a) Baseline β energy preceding left hand DM (but not right hand one) significantly decreased as compared with resting β energy of control task ($P < 0.05$). (b) Median PMBR amplitude across participants was significantly lower in RM2 for each hand ($P < 0.05$). (c) Median PMBR latency across participants was significantly shorter in RM2 and lengthened in DM as indexed to RMI for each hand ($P < 0.05$). DM, discrete movements; MI, primary motor cortex; PMBR, postmovement β-rebound; RM, repetitive movement.
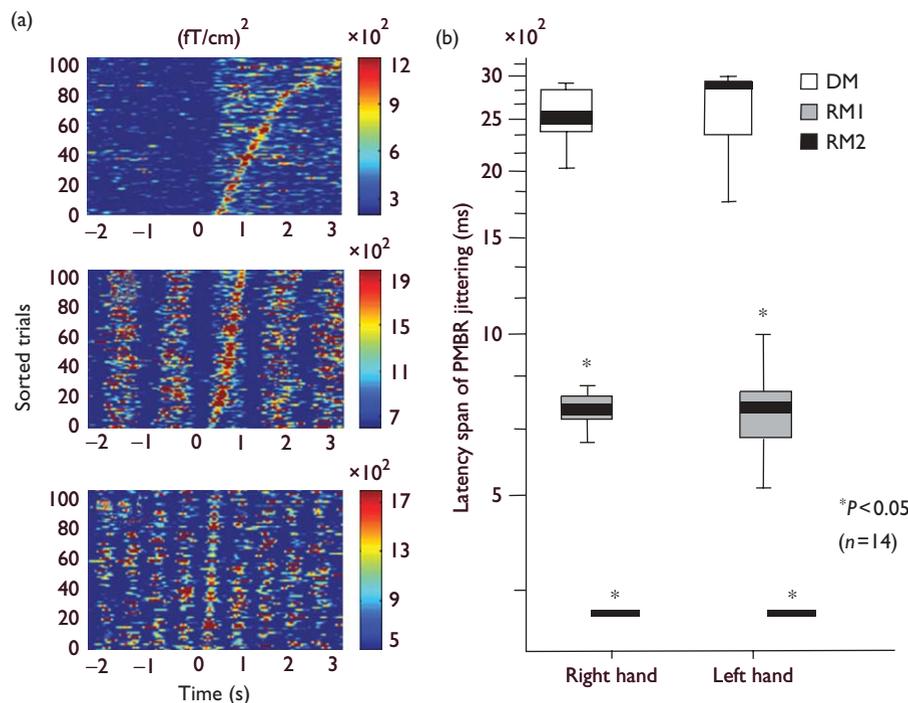
Fig. 2 PMBR jittering on the contralateral MI. (a) Raster plots of individual PMBR in one representative participant. For brevity, only data from right hand movement are shown. β energies were sorted by the latency between PMBR and movement onset. Latency jittering was more pronounced in DM and less in RM2. (b) Latency span of PMBR jittering. This latency span was significantly larger in DM and smaller in RM2 than in RMI regardless of the hand used ($P < 0.05$). DM, discrete movement; MI, primary motor cortex; PMBR, postmovement β-rebound; RM, repetitive movement.

to reflect an idling state over the M1 [10] or the somatosensory processing arising from cutaneous or proprioceptive afferents [11]. Therefore, PMMCD is not only related to the termination of motor command but also to somatosensory processing. Assuming that PMMCD is related to information processing, the observations of longer PMBR latency for longer-IMI movement should indicate the increased cognitive processing demands (e.g. attention and working memory) after voluntary finger movement. This suggestion is congruent to previous fMRI and PET studies that long-IMI movement requires more movement-related cognitive demands [1,6], which in turn may lead to a prolonged PMMCD.

In the same vein, the observation of the robust PMBR latency jittering among different IMI lengths can be ascribed to the participant's cognitive state under internal pacing that varied from time to time [17]. One plausible explanation of the fact that short-IMI movements resulted in a smaller latency span of PMBR jittering than long-IMI DM is that short-IMI movements belonging to an automatic rhythm make few cognitive demands and require more rhythmical operation of RMs, which in turn may reflect certain degrees of automatism [20]. Alternatively, central pattern generators, yielding rhythmic activity of ∼50 ms to ∼2 s [21], may provide another possible neural mechanism for automatic timing control [1,6] that leads to little PMBR latency jittering in short-IMI movement.

Short-IMI movement (i.e. RM2) expressing an attenuated PMBR amplitude compared with long-IMI DM implies relatively fewer synchronized neuronal ensembles during the PMMCD stage. Short-IMI movement makes few cognitive demands [6], recruits few synchronized neuronal ensembles over the contralateral M1, and eventually results

in an attenuated PMBR amplitude. An alternative explanation is an 'aborted' PMMCD. The attenuation of PMBR amplitude in RM2 probably indicates the interference from the coinciding motor cortical activation of the subsequent movement [8,20]. This interpretation is congruent with a previous EEG study on Parkinson's disease according to which M1 overactivity after movement may interfere with PMMCD, leading to a reduced PMBR amplitude [8], and is corroborated by EEG studies in normal participants that a partial or complete suppression of PMBR can result from a sustained cortical activation [11,13]. Nevertheless, our results are incongruent with a previous fMRI study reporting a positive correlation between M1 activity and movement frequency (IMI) [22]. This can be ascribed to the differences in underlying substrates of neurophysiology as measured by fMRI and MEG techniques [23].

In this study, the baseline β energy was modulated by both motor task and hemispheric dominance. This is in agreement with previous fMRI and PET studies that the baseline default mode of brain function can be altered during specific goal-directed behaviors, which is manifested in a change of 'baseline' activity for the active task compared to the 'real' resting brain activity [18,19]. In addition, the observation of reduced movement-related baseline β energy, especially for the nondominant hand of right-handed participants, may mirror the theory of hemispheric asymmetry of motor function, that is, a more demanding performance is mandated by the nondominant hand [24]. Hence, mental efforts for left hand movement may increase the work load of neurons in the right hemisphere, resulting in the increase of cortical activation and the reduction of baseline β energy.

Conclusions

The neural mechanisms of PMMCD between long-IMI and short-IMI, even for a simple motor task, are basically dissimilar. PMMCD can reflect cognitive demands in addition to motor and somatosensory processing. Studies of similar protocol will be furthered to investigate the possibility of separating effects of cognitive impairment from those of motor dysfunction in neurological disease, for example, Parkinson's disease.

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