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RESEARCH ARTICLE | *Control of Movement*

Movement speed effects on beta-band oscillations in sensorimotor cortex during voluntary activity

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Zhang X, Li H, Xie T, Liu Y, Chen J, Long J. Movement speed effects on beta-band oscillations in sensorimotor cortex during voluntary activity. *J Neurophysiol* 124: 352–359, 2020. First published June 24, 2020; doi:10.1152/jn.00238.2020.—Beta-band oscillations are a dominant feature in the sensorimotor system, which includes movement-related beta desynchronization (MRBD) during the preparation and execution phases of movement and postmovement beta synchronization (PMBS) on movement cessation. Many studies have linked this rhythm to motor functions. However, its associations to the movement speed are still unclear. We make a hypothesis that PMBS will be modulated with increasing of movement speeds. We assessed the MRBD and PMBS during isotonic slower self-paced and ballistic movements with 15 healthy subjects. Furthermore, we conduct an additional control experiment with the isometric contraction with two levels of forces to match those in the isotonic slower self-paced and ballistic movements separately. We found that the amplitude of PMBS but not MRBD in motor cortex is modulated by the speed during voluntary movement. PMBS was positively correlated with movement speed and acceleration through the partial correlation analysis. However, there were no changes in the PMBS and MRBD during the isometric contraction with two levels of forces. These results demonstrate a different function of PMBS and MRBD to the movement speed during voluntary activity and suggest that the movement speed would affect the amplitude of PMBS.

NEW & NOTEWORTHY Beta-band oscillations are a dominant feature in the sensorimotor system that associate to the motor function. We found that the movement-related postmovement beta synchronization (PMBS) over the contralateral sensorimotor cortex was positively correlated with the speed of a voluntary movement, but the movement-related beta desynchronization (MRBD) was not. Our results show a differential response of the PMBS and MRBD to the movement speed during voluntary movement.

EEG; movement-related beta desynchronization; postmovement beta synchronization; speed; voluntary movement

INTRODUCTION

Speed is one of the important movement-related parameters that would affect the accuracy of a motor task. Most of our daily tasks involve continuous changes in movement speed that is maintained by a continuous drive from the cortex to spinal motoneurons and by fine motor adjustments according to proprioceptive feedback (Omrani et al. 2013). The neurophysiological basis of this communication is the neural oscillations in the sensorimotor cortex. Previous studies have suggested a central role of the sensorimotor oscillations at beta band (14–30 Hz) in both the encoding of the motor command and the processing of proprioceptive feedback (Aumann and Prut 2015; Baker 2007; Riddle and Baker 2005; Witham et al. 2011).

Sensorimotor activity in beta band is observed in the forms of movement-related beta desynchronization (MRBD) and postmovement beta synchronization (PMBS). MRBD is a power decrease during the preparation and execution phases of movement, while PMBS is a power increase following movement cessation. Studies showed that MRBD likely reflects an increase in processing during movement planning and execution and even movement imagination (Kühn et al. 2004; Pfurtscheller and Lopes da Silva 1999; Pfurtscheller and Neuper 1997; Pfurtscheller et al. 1997). In contrast, the PMBS is thought to reflect the reinforcement of existing motor states and steady motor output (Engel and Fries 2010; Gilbertson et al. 2005; Jenkinson and Brown 2011; Swann et al. 2009). Furthermore, PMBS has been associated with the processing of movement-related sensory afference. Evidence supporting the latter includes observations that a similar phenomenon also follows passive movements (Alegre et al. 2002; Cassim et al. 2001) and that the PMBS is modulated by how a movement is terminated (Alegre et al. 2008) and by errors related to the completed movement (Tan et al. 2014). Despite a vast number of studies about the functional contributions of sensorimotor activity in beta band to motor control, however, the modulation of movement speed on the sensorimotor beta activities is still unclear.

Regarding the MRBD, many observations had linked this to a cortical gate that would switch off beta oscillations necessary to facilitate local processing (Fry et al. 2016; Stančák et al. 1997; Stevenson et al. 2011). For example, the reduction in beta amplitude during volitional contractions of the fingers/arm has been shown to be unrelated to movement speed (Pfurtscheller et al. 1998; Stancák and Pfurtscheller 1996) and the rate of force development (Fry et al. 2016). In addition, the amplitude of MRBD has been shown to be unrelated to the weight of a manipulated load (Pistohl et al. 2012; Stančák

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et al. 1997). In contrast, the PMBS is more variable in its relationship with movement parameters (Fry et al. 2016; Stancák and Pfurtscheller 1995). The increases in movement speed also result in increments in electromyographic (EMG) activity during movement execution. A greater PMBS has been observed following finger extension movements performed against a heavy resistive load compared with unloaded extensions (Stančák et al. 1997). In an isometric wrist flexion task to remove the influence of movement, the increase in the rate of force development results in increments in the amplitude of PMBS (Fry et al. 2016). Furthermore, accumulating evidence suggests that the quantity of afferent input is correlated to the amplitude of PMBS. For example, the magnitude of PMBS is modulated by intensity of electrical stimuli (Stancák et al. 2003). Furthermore, voluntary movement and mixed-nerve stimulation elicit a stronger beta rebound than pure tactile stimulation (Houdayer et al. 2006). Indeed, the afferent signals generated during voluntary activation are proportional to movement speed (Cordo et al. 1994; Gritsenko et al. 2007; Sittig et al. 1987). Therefore, we hypothesized that although MRBD is unchanged with increasing of movement speeds, PMBS will be modulated with increasing of movement speeds. To test our hypothesis, we used EEG over the contralateral sensorimotor cortex to measure the PMBS and MRBD during isotonic slower self-paced and ballistic index finger movements.

METHODS

Subjects

Fifteen volunteers (5 women, 22.2 ± 0.89 yr old) participated in this study. All participants were right handed according to the Edinburgh Inventory of Handedness (Oldfield 1971) and had good physical and mental health, with no dyskinesia or mental illness. Participants provided written informed consent before the recordings. The experimental procedures were approved by the local ethics committee at Jinan University and were in accordance with the guidelines established in the Declaration of Helsinki.

Experimental Paradigm

The experiment was carried out in a quiet and comfortable environment. During the experiment, the subjects sat in a chair 1 m away from the screen, with their hands on the table in front of the chair flexed at their elbows at a 90-degree angle and the wrist restrained by straps. The position of the tested right hand was shown as in Fig. 1, *A* and *B*. The pegs were used to limit the distance for the index finger movement. In Fig. 1*A*, the distance of the two pegs was modulated based on comfort for each subject, while the two pegs in Fig. 1*B* were used to constraint the movement of the index finger and avoid the interference of the other fingers.

The paradigms for experimental condition and control condition were based on the experimental procedure in Fig. 1*C*. At the start of each experiment, subjects performed three brief maximal voluntary isometric contractions (MVICs) for 3–5 s into index finger abduction, separated by 30 s. The MVIC was calculated as the maximal average rectified EMG amplitude within a window of 500 ms screening across the trials (Tazoe and Perez 2013).

At the beginning of each trial, a fixation cross appeared in the center of the screen for a variable delay (1.25–1.75 s). Then, an imperative stimulus consisting of a green or a red circle appeared for 0.25 s (Fig. 1*C*). These two colors appeared randomly. During the experiment, subjects were instructed to complete a single isotonic abduction movement with right index finger at two different speeds after the stimulus (Fig. 1*A*): as fast as possible (ballistic movement; red circle) and at a slower comfortable speed (self-paced movement; green circle). In the control experiment, subjects were instructed to perform isometric abduction with right index finger at two different force levels, which were computed in ballistic movement and self-paced movement, separately (Fig. 1*B*). To ensure similar force levels

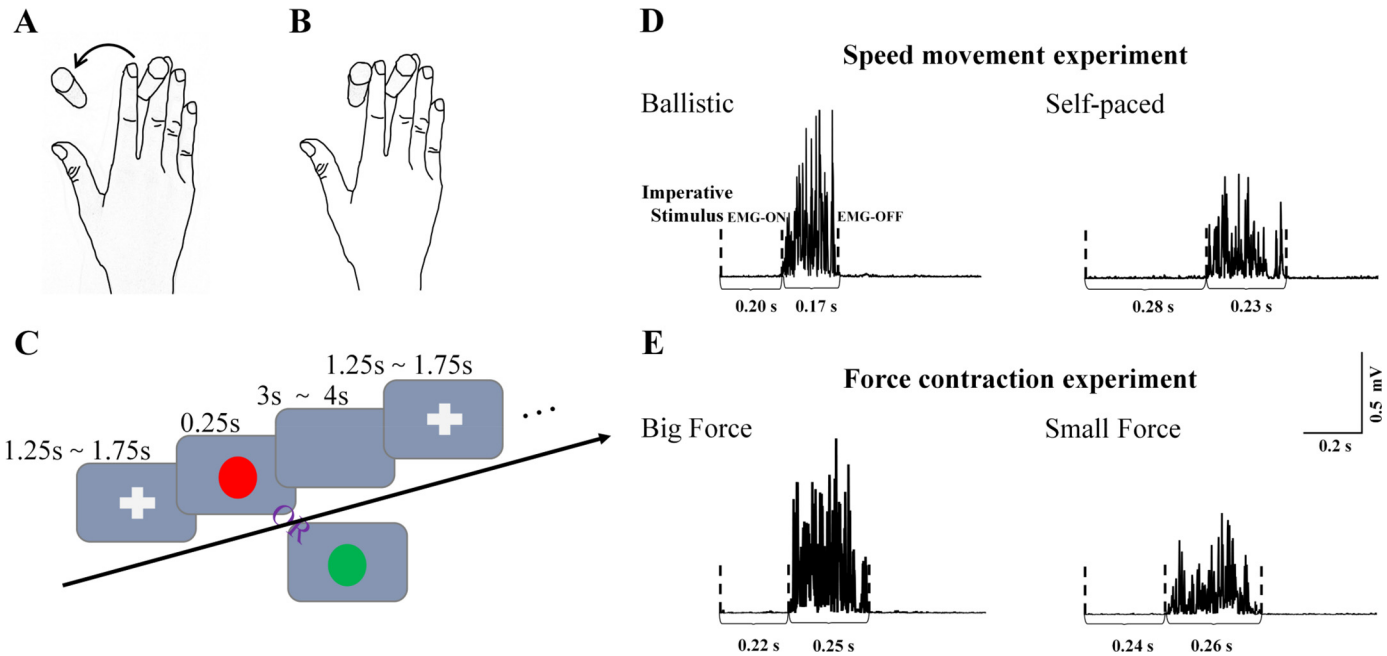

Fig. 1. Experimental setup. *A* and *B*: schematic of the experimental setup showing the posture of the hand during index finger abduction movement at speed movement experiment (*A*) and at force contraction experiment (*B*). *C*: diagram showing the visual display presented to subjects during testing. A fixation cross appeared in the center of the computer screen at the beginning of all trials, followed by an imperative visual signal of red or green circle. Raw traces show rectified electromyographic (EMG) activity in the right first dorsal interosseous muscle in a representative subject during speed movement experiment (*D*) and during force contraction experiment (*E*).

between the speed movement experiment and the force contraction experiment (i.e., the control experiment), the EMG activity from the first dorsal interosseous (FDI) muscle in the speed movement experiment was recorded. Then, in the control experiment, the average EMG activities recorded in the speed movement experiment were shown on the screen of a oscilloscope to visually show the force level that subjects need to reach (i.e., the big force to match the force in the ballistic movement, and the small force to match the force in the self-paced movement). Verbal feedback was given to subjects to remind them that they should reach similar levels of EMG activity by adjusting the force during the big force or small force trials (Federico and Perez 2017). In other words, the subjects were told to put more or less force to make sure the EMG activities were at similar level during the force contraction tasks with the level during the speed movement tasks. In addition, the participants were instructed to exert their force in the control experiment in the same way as in the main experiment. In other words, they should keep accelerating the speed during the big force condition just like how they did in the ballistic movement and should keep relatively constant speed during the small force condition just like they did in the self-paced movement. The recorded EMG activities show that there was no significant difference in EMG activity between ballistic movement and big force contraction ($P = 0.886$), and between self-paced movement and small force contraction ($P = 0.939$), which suggests that the force level was well matched between the speed movement experiment and the force contraction (i.e., control) experiment.

A blank screen was presented to indicate the intertrial interval of 3–4 s during which subjects should relax. A familiarization trial was completed at the beginning of each experimental condition to ensure that subjects were able to complete the task at the adequate speed. There were six blocks for each of the speed movement experiment and the force contraction experiment. In each block, 15 red and 15 green trials were tested in a randomized order. After each trial, the subjects were required to report whether they ever missed the task or performed the wrong movement. In addition, we would also check the raw data during offline analysis to figure out which trials were missed or not. For further analysis, we discarded the trials that the participants ever missed or performed the wrong movement. If the discarded trials exceeded 20%, this session needed to be recollected. This led to the exclusion of zero to three trials per session per subject, and no session was recollected.

EEG and EMG Recordings

EEG data were acquired from 64 scalp sites (extended 10-20 system) using a cap with active Ag/AgCl electrodes (quickcap64). Wet electrodes were used in the cap, and the electrode impedance was modulated to be less than 5 KΩ. The reference electrode was on the bilateral mastoid. A Neuroscan Synamps2 amplifier amplified the EEG signal. The sampling rate of the EEG signal was 1 kHz. The bandpass filtering range was 0.5–30 Hz with a 50-Hz notch filter. In the preprocessing stage of the offline data, we performed the infomax independent component analysis (ICA) algorithm using the EEGlab toolbox (Delorme and Makeig 2004). After visual inspection of the scalp map and of the time course of the activation in each ICA component, we eliminated the components clearly related to eye blinks. The EEG data were back-projected with the remaining subset components for further analysis.

The FDI is a functional muscle responsible for index finger abduction (Pereira Botelho et al. 2019). Therefore, the EMG activity was recorded from the FDI muscle using Ag-AgCl surface electrodes (10-mm diameter). EMG signals were amplified and filtered (5 Hz–2,000 Hz) with a bioamplifier (Neurolog System, Digitimer, UK), and then digitized at 5 kHz with an analog-to-digital (A/D) converter (CED Micro 1401, Cambridge Electronic Design, UK). The signals were stored with sampling rate of 5 kHz with an A/D converter (CED Micro 1401, Cambridge Electronic Design) on a computer for off-line analysis. The mark from the presentation software (E-Prime 3.0) was sent to the EEG acquisition software (Curry 8) and EMG acquisition software (Spike) simultaneously for their synchronization in the experiment.

Data Processing

EMG, reaction time, and movement speed. The EMG was rectified and averaged across the trials. The EMG signal was then normalized as a percentage of MVIC. We measured reaction time (RT) as the latency (in milliseconds) of the first burst of EMG activity following stimulus presentation. In each trial, the time point of imperative stimulus was defined as the onset and the RT was defined as the time point in which EMG activity exceeded the >4 SD of the average contracting mean rectified EMG, measured 100 ms beforethe stimulus artifact, while the end time of movement in that trial, defined as the time point in which the mean EMG activity returned to <4 SD of the average contracting mean rectified EMG, measured 100 ms before the stimulus artifact. Therefore, the movement duration was set to the interval between movement initiation and movement termination. Since the distance for index finger movement was constant during the experiment for each subject, the average movement speed for each trial can be defined as the reciprocal of movement duration by set the distance to be 1. For the force contraction experiment, we recorded the time taken from 0 to the maximum value of EMG (with a sliding window of 10 ms), and then the mean rate of change of force was calculated for each trial (the maximum EMG/time taken). Mean averages of these values were calculated within each individual.

EEG analysis. The response-locked EEG time series (−0.25 s before and 2 s after the stimulus) were extracted for artifacts screening. Artifacts were defined as EEG signals with amplitude larger than mean + 5 SD, with the mean and SD calculated for each time point based on the response-locked EEG time series for all trials. The response-locked EEG time series from each artifact-free single trial were decomposed into their time-frequency representations using the Fieldtrip software package (http://www.ru.nl/fcdonders/fieldtrip/). Finger movement presents a contralateral activation advantage in the cerebral hemisphere (Pfurtscheller et al. 1998; Stančák et al. 1997; Stancák and Pfurtscheller 1996). Hence, we used the EEG data of C3 channel in the sensorimotor area for the following analysis.

Spectral power in time-frequency domain was calculated for each trial using a fast Fourier transform (multi)taper approach to the short sliding time windows (Osipova et al. 2006; Percival and Walden 1993). For frequency band of 5–30 Hz (in steps 0.5 Hz), we applied an adaptive time window of three cycles for each frequency $\Delta t = 3/f$. The Hanning taper was used to reduce spectral leakage and control the frequency smoothing. The time windows were advanced in steps of 50 ms. The time-frequency representation of power was calculated by averaging the squared absolute values of the convolutions over trials. Event-related EEG power change was subsequently calculated as the percentage change relative to the baseline by dividing the power at each frequency and each time point by the average power of that frequency across baseline and then subtracting 100 from the normalized value. The baseline was defined from −250 ms to 0 ms of the starting of movement task (Fry et al. 2016; Stančák et al. 1997; Stevenson et al. 2011). If the value of event-related EEG power change was larger than 0, then it indicated that the power was higher than the baseline average power of that frequency and vice versa.

Event-related power changes in the beta band (14–30 Hz) were investigated. In the ballistic and slow motion of the index finger, the duration of MRBD and PMBS is generally longer than 0.5 s (Stancák and Pfurtscheller 1996). Since this study focuses on observing the differences in the peak value and its vicinity between different conditions, the PMBS was defined as the average normalized power over a 200-ms window centered on the peak of the power change after movement termination (Tan et al. 2014), while the MRBD was

defined as the average normalized power over a 200-ms window centered on the trough of the power change during movement.

Statistical analysis. Overall, this study used a 2 (speed movement versus force contraction) by 2 (big force versus small force) design. In the speed movement experiment/condition, ballistic movement had a big force, whereas the self-paced movement had a small force. In the force contraction experiment/condition, subjects were asked to perform isometric abduction with right index finger at two different force levels (big force versus small force). The temporal structure of the big force contraction was similar with that of the ballistic movement, whereas the temporal structure of the small force contraction was similar with that of the self-paced movement. The force contraction experiment (big force versus small force) serves as a control experiment in this study aiming to rule out the possibility that the main finding in the speed movement experiment was due to the difference in force, rather than in speed.

First, repeated-measures two-way ANOVAs were performed to determine the effect of movement task (speed movement versus force contraction) and force condition (big force versus small force) on EMG, RT, rate of change of force, movement duration, and power change at beta band. One-way repeated-measures ANOVA was performed on the movement speed and movement acceleration during speed movement. Bonferroni post hoc correction was used to check for significant comparisons (significance level α of 0.05). A priori comparisons were made as specified. Normal distribution was tested by the Shapiro-Wilk test (all $P > 0.05$). The significance level was set at $P < 0.05$ and group data were presented as mean SD in the text and as SE in the figures. To explore the relationship between MRBD/PMBS and behavior variables (i.e., EMG, RT, rate of change of force, movement speed, movement duration, and movement acceleration), we chose the simplest model, a linear model, to describe the effects of these correlations. Pearson correlation analysis was conducted to evaluate the relationships in different experiment conditions using IBM SPSS (1,000 Bootstrap samples, confidence interval 95%). To avoid the influence of mutual interference between behavioral variables, we corrected the respective correlation between MRBD/PMBS and behavior variables by means of partial correlation (Pearson).

RESULTS

Behavioral Measurements

Table 1 presents the behavioral measurements during the speed movement experiment and force contraction experiment. Note that EMG activity, RT, rate of change of force, movement duration, movement speed and movement acceleration increased during the ballistic movement as compared with the self-paced movement.

Repeated-measures ANOVA showed a significant effect of movement task ($F_{1,43} = 16.76$, $P = 0.002$), but not force condition ($F_{1,43} = 2.35$, $P = 0.1\ 6$) nor in their interaction ($F_{1,43} = 2.24$, $P = 0.17$) on the magnitude of EMG. Post hoc analysis showed that EMG in the speed movement task was larger during ballistic compared with the self-paced movement condition (ballistic,15.5 ± 3.2%; self-paced, 8.7 ± 2.1%; $P < 0.001$), and the same significance was found within force contraction task (big force, 18.1 ± 4.9%; small force,10.4 ± 3.3%; $P = 0.01$). We found that EMG was not changed between ballistic movement and big force ($P = 0.886$) and between self-paced movement and small force ($P = 0.939$).

For RT, similar analysis showed no significant effects of movement task ($F_{1,43} = 0.0004$, $P = 0.983$), and their interaction ($F_{1,43} = 3.87$, $P = 0.075$; Table 1) but showed significant effects of force condition ($F_{1,43} = 43.96$, $P < 0.001$). Post hoc analysis showed that the RT was decreased during ballistic compared with self-paced movement condition (ballistic, 0.23 ± 0.04 s; self-paced, 0.28 ± 0.05; $P < 0.001$); RT showed same significance in force contraction task (big force, 0.23 ± 0.05 s; small force, 0.27 ± 0.06 s; $P < 0.001$). In addition, we found that RT showed no difference between ballistic movement and big force ($P = 0.469$) and between self-paced movement and small force ($P = 0.493$).

For rate of change of force, repeated-measures ANOVA showed no significant effects of movement task ($F_{1,43} = 0.65$, $P = 0.444$) or their interaction ($F_{1,43} = 1.21$, $P = 0.303$; Table 1) but showed significant effects of force condition ($F_{1,43} = 9.07$, $P = 0.017$). Post hoc analysis showed that the rate of change of force was increased during ballistic compared with self-paced movement condition (ballistic, 2.70 ± 0.63 mV/s; self-paced, 1.59 ± 0.27 mV/s; $P = 0.027$) and had the similar significance in force contraction task (big force, 3.11 ± 0.58 mV/s; small force, 1.79 ± 0.27 mV/s; $P = 0.011$). We found that rate of change of force was no change between ballistic movement and big force ($P = 0.317$) and between self-paced movement and small force ($P = 0.630$).

For movement duration, repeated-measures ANOVA showed no significant effects of movement task ($F_{1,43} = 0.20$, $P = 0.667$) and their interaction ($F_{1,43} = 1.07$, $P = 0.323$; Table 1) but showed significant effects of force condition ($F_{1,43} = 32.42$, $P < 0.001$). Post hoc analysis showed that movement duration was decreased during ballistic compared with self-paced movement condition (ballistic, 0.13 ± 0.07 s; self-paced, 0.18 ± 0.06 s; $P = 0.002$) and had similar significance in the force contraction task (big force, 0.13 ± 0.05 s; small force, 0.20 ± 0.05 s; $P < 0.001$). We found that movement duration was no change between ballistic movement and big force ($P = 0.985$) and between self-paced movement and small force ($P = 0.421$). For speed, one-way ANOVA showed a significant effect ($F_{1,23} = 7.19$; $P = 0.012$) that the subjects performed faster during ballistic compared with the self-paced movement condition. Similar analysis showed

Table 1. *Behavioral measurements*

	Speed Movement Experiment			Force Contraction Experiment		
	Ballistic	Self-paced	*P* value	Big force	Small force	*P* value
EMG, %MVIC	15.50 ± 3.20	8.7 ± 2.10	<0.001	18.10 ± 4.90	10.40 ± 3.30	0.010
Reaction time, s	0.23 ± 0.04	0.28 ± 0.05	<0.001	0.23 ± 0.05	0.27 ± 0.06	<0.001
Rate of change of force	2.70 ± 0.63	1.59 ± 0.27	0.027	3.11 ± 0.58	1.79 ± 0.27	0.011
Movement duration, s	0.13 ± 0.07	0.18 ± 0.06	0.002	0.13 ± 0.05	0.20 ± 0.05	<0.001
Movement speed, cm/s	9.03 ± 3.66	6.21 ± 1.79	0.012			
Movement acceleration, cm/s^2	94.16 ± 46.45	41.51 ± 23.05	0.016			

Values are means ± SD. EMG, electromyogram; MVIC, maximal voluntary isometric contractions.

a significant effect ($F_{1,23}$ = 6.52; P = 0.016) on movement acceleration.

MRBD and PMBS

Figure 2 illustrates the time-frequency results during speed movement and force contraction tasks. We can see that a relative decrease in EEG power across the beta frequency band (14–30 Hz) was consistently observed over contralateral sensorimotor cortex during speed movement (Fig. 2, *A* and *C*) and force contraction tasks (Fig. 2, *B* and *D*). The decrease in power was followed by a rebound synchronization after movement. Note that this PMBS was increased during ballistic compared with self-paced movement.

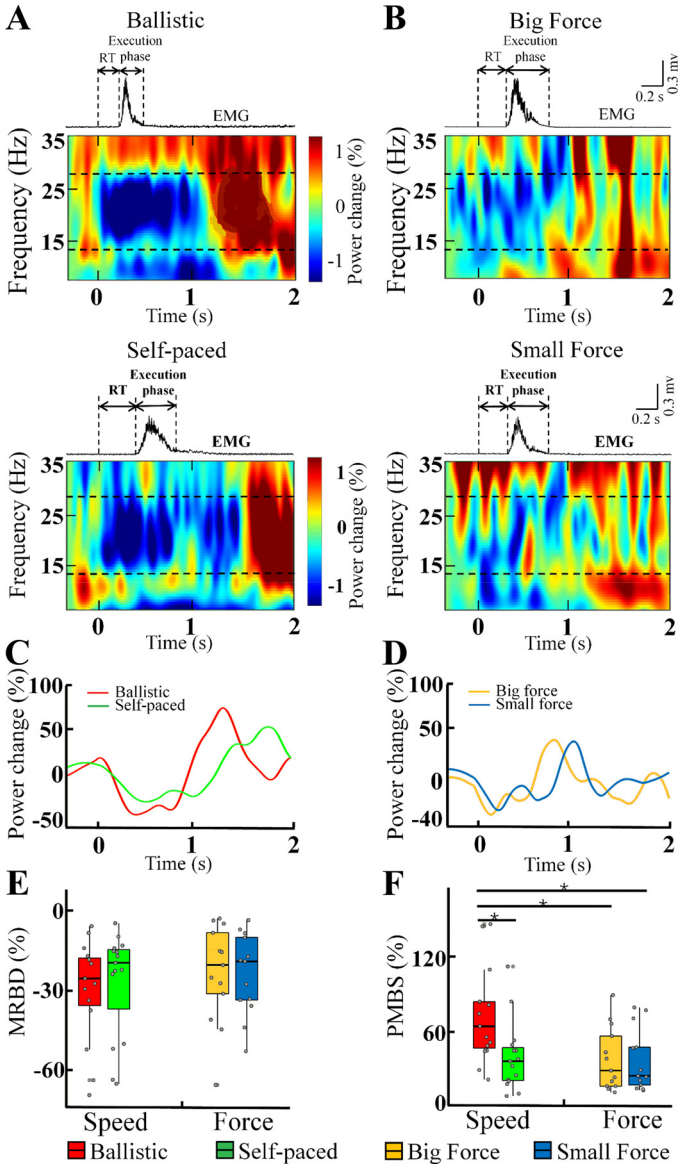

Fig. 2. Power spectra of electroencephalogram (EEG) over sensorimotor cortex (C3 channel). *A* and *B*: average power change at different frequencies in a representative subject (averaged across trials) during speed movement task (*A*) and force contraction task (*B*). The dashed lines represent the beta band between 14 and 30 Hz. *C* and *D*: average power change time courses at beta band (14–30 Hz) during speed movement task (ballistic, red; self-paced, green; *C*) and force contraction task (big force, yellow; small force, blue; *D*). *E* and *F*: group data (n = 15). The abscissa shows the measurements of MRBD (*E*) and PMBS (*F*). The ordinate shows the average power change across subjects, while the gray circle represents the individual value for each subject. Error bars indicate SEs. *P < 0.05. EMG, electromyogram; MRBD, movement-related beta desynchronization; PMBS, postmovement beta synchronization; RT, reaction time.

Repeated two-way ANOVA with task and condition as main factors showed no effects of movement task ($F_{1,43}$ = 0.19, P = 0.682), force condition ($F_{1,43}$ = 3.054, P = 0.111), and their interaction ($F_{1,43}$ = 4.71, P = 0.06) on MRBD (Fig. 2*E*). In contrast, the main effect of movement task on PMBS was not significant ($F_{1,43}$ = 0.230, P = 0.642), but the main effect of force condition ($F_{1,43}$ = 5.373, P = 0.030) and their interaction ($F_{1,43}$ = 9.18, P = 0.013; Fig. 2*F*) were significant. Post hoc analysis showed that the PMBS was increased during ballistic compared with self-paced movements (ballistic, 57.13 ± 30.84%; self- paced, 36.01 ± 19.98%; P = 0.001; Fig. 2*F*), but there was no difference in PMBS between the big force and small force conditions for the force contraction task (big force, 32.57 ± 27.53%; small force, 31.18 ± 28.25%; P = 0.783; Fig. 2*F*).

Correlations Analysis

Table 2 and Fig. 3 show the partial correlation results. We found that a positive correlation was found between PMBS measured during the speed movement experiment and the movement speed (r = 0.482, P = 0.017; Fig. 3*A*) and movement acceleration (r = 0.641, P = 0.001; Fig. 3*B*). Here, note that individuals with larger increases in PMBS were those who performed movements at faster speeds. However, the PMBS did not significantly correlate with EMG (r = 0.102, P = 0.64), RT (r = −0.192, P = 0.37), rate of change of force (r = 0.106, P = 0.63), and movement duration (r = −0.156, P = 0.48). The MRBD during speed movement experiment did not significantly correlate with any behavioral measurements of the EMG (r = −0.176, P = 0.42), RT (r = −0.028, P = 0.90), rate of change of force (r = 0.145, P = 0.50), movement duration (r = 0.175, P = 0.41), movement speed (r = 0.072, P = 0.74), and movement acceleration (r = −0.065, P = 0.77). In addition, in the force contraction experiment, we did not find any significant correlation between EMG and the magnitude of PMBS (r = 0.081, P = 0.74) and MRBD (r = −0.039, P = 0.87), between RT and the magnitude of PMBS (r = 0.351, P = 0.14) and MRBD (r = −0.139, P = 0.57), between the rate of change of force and the magnitude of PMBS (r = −0.359, P = 0.13) and MRBD (r = 0.181, P = 0.46), and between movement duration and the magnitude of PMBS (r = −0.354, P = 0.10) and MRBD (r = 0.233, P = 0.34).

DISCUSSION

We demonstrate that PMBS but not MRBD over the contralateral sensorimotor cortex increases in the ballistic compared with self-paced movements. And as revealed in the control experiment, this increase was not due to the changes in force level. The PMBS was positively correlated with the movement speed and movement acceleration during speed movement. Our results show a differential modulation of amplitude in the PMBS and MRBD with increasing movement speed.

Table 2. *Results for the correlations between PMBS/MRBD with different behavioral measurements during speed movement experiment or force contraction experiment*

	Speed Movement				Force Contraction			
	PMBS		MRBD		PMBS		MRBD	
	r	*P* value	*r*	*P* value	*r*	*P* value	*r*	*P* value
EMG, % MVIC	0.102	0.636	−0.176	0.423	0.081	0.741	−0.039	0.874
Reaction time, s	−0.192	0.369	−0.028	0.901	0.351	0.140	−0.139	0.571
Rate of change of force	0.106	0.631	0.145	0.498	−0.359	0.132	0.181	0.458
Movement duration, s	−0.156	0.478	0.175	0.412	−0.354	0.100	0.233	0.337
Movement speed, cm/s	0.482	**0.017**	0.072	0.738				
Movement acceleration, cm/s^2	0.641	**0.001**	−0.065	0.765				

EMG, electromyogram; MRBD, movement-related beta desynchronization; MVIC, maximal voluntary isometric contractions; PMBS, postmovement beta synchronization. Boldface indicates that behavioral parameters are significantly correlated with PMBS ($P<0.05$).

Modulations of Amplitude in the MRBD with Different Movement Speeds

Previous studies hypothesized that the MRBD may act as a cortical gate which would switch off beta oscillations necessary to facilitate local processing (Fry et al. 2016; Stančák et al. 1997; Stevenson et al. 2011). Indeed, it is likely that the magnitude of MRBD will not change with the stimulus parameters. Our data indicate that the magnitude of MRBD is not related to the movement speed and contraction force levels. This is in agreement with previous evidence showing that the reduction in beta amplitude during volitional contractions of the fingers/arm has been shown to be not influenced by the movement speed (Pfurtscheller et al. 1998; Stancák and Pfurtscheller 1996), contraction force (Cremoux et al. 2013; Stančák et al. 1997), and the rate of force development (Fry et al. 2016). Moreover, the previous studies demonstrated that the MRBD can be observed in motor planning (Liddle et al. 2016; Tzagarakis et al. 2010; van Wijk et al. 2009) and motor imagery (Pfurtscheller and Neuper 1997; Schnitzler et al. 1997). Thus, our results demonstrate that the magnitude of MRBD is not speed-related modulations.

Modulations of Amplitude in the PMBS with Different Movement Speeds

In contrast, the PMBS is more variable in its relationship with movement parameters. For example, previous studies have shown that the PMBS is related to the rate of force development in an isometric wrist flexion task by removing the influence of movement (Fry et al. 2016) and related to the passive movement speed (Iwane et al. 2019). Our data indicate that during voluntary activation the PMBS is increased in the ballistic compared with self-paced movements. Furthermore, in the partial correlation analysis by controlling the force factor, the PMBS is significantly correlated with the movement speed. This partially agrees with those of Stancák and colleagues, who demonstrated a larger PMBS following ballistic finger extensions compared with slow movement (Stancák and Pfurtscheller 1995). Someone would argue that the increase in PMBS during ballistic movement was related to the force levels due to the increases in movement speed resulting in increments in EMG activity during movement execution. In our control experiment, we have demonstrated that the PMBS is not related to the contraction force level. Contrary our results, a recent study has demonstrated that the amplitude of PMBS was positively correlated with force during isometric wrist flexion (Fry et al. 2016). The reason why the results found in this paper are different from such results may be related to the experimental design that the difference of force levels between ballistic and self-paced movements may be too small to elicit the significant different PMBS. For example, a previous study has been shown that the finger extension movements with a heavy resistive load elicited greater PMBS compared with unloaded extensions but not without two closer loads (e.g., 30 g and 80 g) (Stančák et al. 1997). In addition, the PMBS could be modulated also by the forced termination of a movement (Alegre et al. 2008). However, we don't think that this was the factor that drove the changes in PMBS in our study because we have asked the subjects to exert their force in a similar way in the speed movement experiment and the force contraction experiment. The temporal structures of the forces were well matched as shown in Figs. 1, *D* and *E*, and 2*A*, which suggests that the subjects did exert forces as instructed. Although our study revealed that PMBS is modulated by movement speed, it is worth noting that this does not mean that the speed is the only parameter for modulating PMBS. Similar to MRBD, PMBS can be observed in the absence of actual movement. Liddle et al. (2016) point out that it is possible to motivate PMBS when there is no actual movement but only a movement plan. Therefore, PMBS is a complex signal feature modulated by cognitive processes, sensory inputs and movement parameters (Fry et al. 2016).

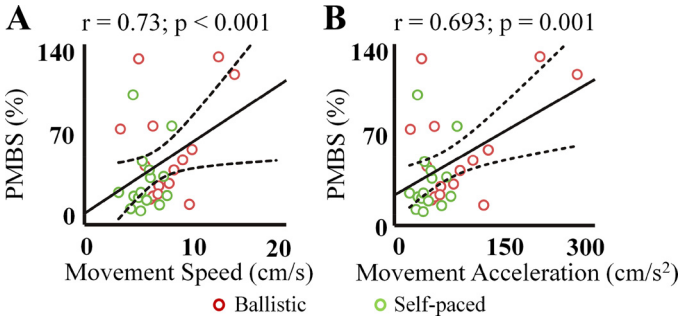

Fig. 3. The scatter plots with significant associations. The relationships between postmovement beta synchronization (PMBS) and movement variables of movement speed (*A*) and movement acceleration (*B*) during speed movement experiment. The solid and dotted lines show the regression lines and the 95% confidence limits.

Conclusions

This paper conducted two experimental schemes to assess the effects of speed on beta rhythmical activity. The findings of this study were as follows: *1*) the amplitude of the MRBD was not modulated by speed; and *2*) there was a significant positive correlation between PMBS and movement speed. These indicate that speed may be a modulator of PMBS during isotonic index finger abduction, while force is not the main factor affecting PMBS amplitude. These results show that changing the amplitude of PMBS can be regulated by precisely setting motion parameters, which provides a new way for further clinical and basic neuroscientific understanding of PMBS phenomenon.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

X.Z. and J.L. conceived and designed research; X.Z. and J.L. performed experiments; X.Z. and J.L. analyzed data; X.Z., H.L., T.X., Y.L., J.C., and J.L. interpreted results of experiments; X.Z. and J.L. prepared figures; X.Z. and J.L. drafted manuscript; X.Z., H.L., T.X., Y.L., J.C., and J.L. edited and revised manuscript; X.Z., H.L., T.X., Y.L., J.C., and J.L. approved final version of manuscript.

REFERENCES

Alegre M, Alvarez-Gerriko I, Valencia M, Iriarte J, Artieda J. Oscillatory changes related to the forced termination of a movement. *Clin Neurophysiol* 119: 290–300, 2008. doi:10.1016/j.clinph.2007.10.017.

Alegre M, Labarga A, Gurtubay IG, Iriarte J, Malanda A, Artieda J. Beta electroencephalograph changes during passive movements: sensory afferences contribute to beta event-related desynchronization in humans. *Neurosci Lett* 331: 29–32, 2002. doi:10.1016/S0304-3940(02)00825-X.

Aumann TD, Prut Y. Do sensorimotor β-oscillations maintain muscle synergy representations in primary motor cortex? *Trends Neurosci* 38: 77–85, 2015. doi:10.1016/j.tins.2014.12.002.

Baker SN. Oscillatory interactions between sensorimotor cortex and the periphery. *Curr Opin Neurobiol* 17: 649–655, 2007. doi:10.1016/j.conb.2008.01.007.

Cassim F, Monaca C, Szurhaj W, Bourriez JL, Defebvre L, Derambure P, Guieu JD. Does post-movement beta synchronization reflect an idling motor cortex? *Neuroreport* 12: 3859–3863, 2001. doi:10.1097/00001756-200112040-00051.

Cordo P, Carlton L, Bevan L, Carlton M, Kerr GK. Proprioceptive coordination of movement sequences: role of velocity and position information. *J Neurophysiol* 71: 1848–1861, 1994. doi:10.1152/jn.1994.71.5.1848.

Cremoux S, Tallet J, Berton E, Dal Maso F, Amarantini D. Motor-related cortical activity after cervical spinal cord injury: multifaceted EEG analysis of isometric elbow flexion contractions. *Brain Res* 1533: 44–51, 2013. doi:10.1016/j.brainres.2013.08.008.

Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods* 134: 9–21, 2004. doi:10.1016/j.jneumeth.2003.10.009.

Engel AK, Fries P. Beta-band oscillations—signalling the status quo? *Curr Opin Neurobiol* 20: 156–165, 2010. doi:10.1016/j.conb.2010.02.015.

Federico P, Perez MA. Altered corticospinal function during movement preparation in humans with spinal cord injury. *J Physiol* 595: 233–245, 2017. doi:10.1113/JP272266.

Fry A, Mullinger KJ, O'Neill GC, Barratt EL, Morris PG, Bauer M, Folland JP, Brookes MJ. Modulation of post-movement beta rebound by contraction force and rate of force development. *Hum Brain Mapp* 37: 2493–2511, 2016. doi:10.1002/hbm.23189.

Gilbertson T, Lalo E, Doyle L, Di Lazzaro V, Cioni B, Brown P. Existing motor state is favored at the expense of new movement during 13-35 Hz oscillatory synchrony in the human corticospinal system. *J Neurosci* 25: 7771–7779, 2005. doi:10.1523/JNEUROSCI.1762-05.2005.

Gritsenko V, Krouchev NI, Kalaska JF. Afferent input, efference copy, signal noise, and biases in perception of joint angle during active versus passive elbow movements. *J Neurophysiol* 98: 1140–1154, 2007. doi:10.1152/jn.00162.2007.

Houdayer E, Labyt E, Cassim F, Bourriez JL, Derambure P. Relationship between event-related beta synchronization and afferent inputs: analysis of finger movement and peripheral nerve stimulations. *Clin Neurophysiol* 117: 628–636, 2006. doi:10.1016/j.clinph.2005.12.001.

Iwane F, Lisi G, Morimoto J. EEG sensorimotor correlates of speed during forearm passive movements. *IEEE Trans Neural Syst Rehabil Eng* 27: 1667–1675, 2019. doi:10.1109/TNSRE.2019.2934231.

Jenkinson N, Brown P. New insights into the relationship between dopamine, beta oscillations and motor function. *Trends Neurosci* 34: 611–618, 2011. doi:10.1016/j.tins.2011.09.003.

Kühn AA, Williams D, Kupsch A, Limousin P, Hariz M, Schneider GH, Yarrow K, Brown P. Event-related beta desynchronization in human subthalamic nucleus correlates with motor performance. *Brain* 127: 735–746, 2004. doi:10.1093/brain/awh106.

Liddle EB, Price D, Palaniyappan L, Brookes MJ, Robson SE, Hall EL, Morris PG, Liddle PF. Abnormal salience signaling in schizophrenia: the role of integrative beta oscillations. *Hum Brain Mapp* 37: 1361–1374, 2016. doi:10.1002/hbm.23107.

Oldfield RC. The assessment and analysis of handedness: the Edinburgh inventory. *Neuropsychologia* 9: 97–113, 1971. doi:10.1016/0028-3932(71)90067-4.

Omrani M, Diedrichsen J, Scott SH. Rapid feedback corrections during a bimanual postural task. *J Neurophysiol* 109: 147–161, 2013. doi:10.1152/jn.00669.2011.

Osipova D, Takashima A, Oostenveld R, Fernández G, Maris E, Jensen O. Theta and gamma oscillations predict encoding and retrieval of declarative memory. *J Neurosci* 26: 7523–7531, 2006. doi:10.1523/JNEUROSCI.1948-06.2006.

Percival DB, Walden AT (Editors). *Spectral Analysis for Physical Applications*. Cambridge, UK: Cambridge University Press, 1993.

Pereira Botelho D, Curran K, Lowery MM. Anatomically accurate model of EMG during index finger flexion and abduction derived from diffusion tensor imaging. *PLOS Comput Biol* 15: e1007267, 2019. doi:10.1371/journal.pcbi.1007267.

Pfurtscheller G, Lopes da Silva FH. Event-related EEG/MEG synchronization and desynchronization: basic principles. *Clin Neurophysiol* 110: 1842–1857, 1999. doi:10.1016/S1388-2457(99)00141-8.

Pfurtscheller G, Neuper C. Motor imagery activates primary sensorimotor area in humans. *Neurosci Lett* 239: 65–68, 1997. doi:10.1016/S0304-3940(97)00889-6.

Pfurtscheller G, Neuper C, Andrew C, Edlinger G. Foot and hand area mu rhythms. *Int J Psychophysiol* 26: 121–135, 1997. doi:10.1016/S0167-8760(97)00760-5.

Pfurtscheller G, Zalaudek K, Neuper C. Event-related beta synchronization after wrist, finger and thumb movement. *Electroencephalogr Clin Neurophysiol* 109: 154–160, 1998. doi:10.1016/S0924-980X(97)00070-2.

Pistohl T, Schulze-Bonhage A, Aertsen A, Mehring C, Ball T. Decoding natural grasp types from human ECoG. *Neuroimage* 59: 248–260, 2012. doi:10.1016/j.neuroimage.2011.06.084.

Riddle CN, Baker SN. Manipulation of peripheral neural feedback loops alters human corticomuscular coherence. *J Physiol* 566: 625–639, 2005. doi:10.1113/jphysiol.2005.089607.

Schnitzler A, Salenius S, Salmelin R, Jousmäki V, Hari R. Involvement of primary motor cortex in motor imagery: a neuromagnetic study. *Neuroimage* 6: 201–208, 1997. doi:10.1006/nimg.1997.0286.

Sittig AC, van der Gon Denier JJ, Gielen CC. The contribution of afferent information on position and velocity to the control of slow and fast human forearm movements. *Exp Brain Res* 67: 33–40, 1987. doi:10.1007/BF00269450.

Stancák A Jr, Pfurtscheller G. Desynchronization and recovery of beta rhythms during brisk and slow self-paced finger movements in man. *Neurosci Lett* 196: 21–24, 1995. doi:10.1016/0304-3940(95)11827-J.

Stancák A Jr, Pfurtscheller G. Event-related desynchronisation of central beta-rhythms during brisk and slow self-paced finger movements of dominant and nondominant hand. *Brain Res Cogn Brain Res* 4: 171–183, 1996. doi:10.1016/S0926-6410(96)00031-6.

Stančák A Jr, Riml A, Pfurtscheller G. The effects of external load on movement-related changes of the sensorimotor EEG rhythms. *Electroencephalogr Clin Neurophysiol* 102: 495–504, 1997. doi:10.1016/S0013-4694(96)96623-0.

Stancák A, Svoboda J, Rachmanová R, Vrána J, Králík J, Tintera J. Desynchronization of cortical rhythms following cutaneous stimulation: effects of stimulus repetition and intensity, and of the size of corpus callosum. *Clin Neurophysiol* 114: 1936–1947, 2003. doi:10.1016/S1388-2457(03)00201-3.

Stevenson CM, Brookes MJ, Morris PG. β-Band correlates of the fMRI BOLD response. *Hum Brain Mapp* 32: 182–197, 2011. doi:10.1002/hbm.21016.

Swann N, Tandon N, Canolty R, Ellmore TM, McEvoy LK, Dreyer S, DiSano M, Aron AR. Intracranial EEG reveals a time- and frequency-specific role for the right inferior frontal gyrus and primary motor cortex in stopping initiated responses. *J Neurosci* 29: 12675–12685, 2009. doi:10.1523/JNEUROSCI.3359-09.2009.

Tan H, Zavala B, Pogosyan A, Ashkan K, Zrinzo L, Foltynie T, Limousin P, Brown P. Human subthalamic nucleus in movement error detection and its evaluation during visuomotor adaptation. *J Neurosci* 34: 16744–16754, 2014. doi:10.1523/JNEUROSCI.3414-14.2014.

Tazoe T, Perez MA. Speed-dependent contribution of callosal pathways to ipsilateral movements. *J Neurosci* 33: 16178–16188, 2013. doi:10.1523/JNEUROSCI.2638-13.2013.

Tzagarakis C, Ince NF, Leuthold AC, Pellizzer G. Beta-band activity during motor planning reflects response uncertainty. *J Neurosci* 30: 11270–11277, 2010. doi:10.1523/JNEUROSCI.6026-09.2010.

van Wijk BC, Daffertshofer A, Roach N, Praamstra P. A role of beta oscillatory synchrony in biasing response competition? *Cereb Cortex* 19: 1294–1302, 2009. doi:10.1093/cercor/bhn174.

Witham CL, Riddle CN, Baker MR, Baker SN. Contributions of descending and ascending pathways to corticomuscular coherence in humans. *J Physiol* 589: 3789–3800, 2011. doi:10.1113/jphysiol.2011.211045.