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Is an absolute level of cortical beta suppression required for proper movement? Magnetoencephalographic evidence from healthy aging

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ABSTRACT

Previous research has connected a specific pattern of beta oscillatory activity to proper motor execution, but no study to date has directly examined how resting beta levels affect motor-related beta oscillatory activity in the motor cortex. Understanding this relationship is imperative to determining the basic mechanisms of motor control, as well as the impact of pathological beta oscillations on movement execution. In the current study, we used magnetoencephalography (MEG) and a complex movement paradigm to quantify resting beta activity and movement-related beta oscillations in the context of healthy aging. We chose healthy aging as a model because preliminary evidence suggests that beta activity is elevated in older adults, and thus by examining older and younger adults we were able to naturally vary resting beta levels. To this end, healthy younger and older participants were recorded during motor performance and at rest. Using beamforming, we imaged the peri-movement beta event-related desynchronization (ERD) and extracted virtual sensors from the peak voxels, which enabled absolute and relative beta power to be assessed. Interestingly, absolute beta power during the pre-movement baseline was much stronger in older relative to younger adults, and older adults also exhibited proportionally large beta desynchronization (ERD) responses during motor planning and execution compared to younger adults. Crucially, we found a significant relationship between spontaneous (resting) beta power and beta ERD magnitude in both primary motor cortices, above and beyond the effects of age. A similar link was found between beta ERD magnitude and movement duration. These findings suggest a direct linkage between beta reduction during movement and spontaneous activity in the motor cortex, such that as spontaneous beta power increases, a greater reduction in beta activity is required to execute movement. We propose that, on an individual level, the primary motor cortices have an absolute threshold of beta power that must be reached in order to move, and that an inability to suppress beta power to this threshold results in an increase in movement duration.

1. Introduction

Proper movement execution is served by a specific pattern of oscillatory activity throughout the sensorimotor network. Basically, prior to movement, there is a strong event-related desynchronization (ERD) in the beta (14–30 Hz) band, commonly termed the peri-movement beta ERD, which is sustained throughout movement and dissipates shortly after movement termination (Cheyne et al., 2006; Gaetz et al., 2010; Heinrichs-Graham and Wilson, 2015; Heinrichs-Graham et al., 2014b, in press; Jurkiewicz et al., 2006; Pfurtscheller and Lopes da Silva, 1999; Wilson et al., 2014, 2010, 2011b). After this beta ERD, there is a robust resynchronization in the beta band, called the post-movement beta rebound (PMBR), that overshoots baseline levels and is sustained for about 2.0 s after movement termination (Cheyne et al., 2006; Gaetz et al., 2010; Heinrichs-Graham et al., 2014b; Jurkiewicz et al., 2006; Ohara et al., 2000; Parkes et al., 2006; Szurhaj et al., 2003; Wilson et al., 2010, 2011b). The peri-movement beta ERD has been associated with movement planning and execution, while the PMBR is thought to be related to movement termination and inhibition processes (for a review, see Cheyne, 2013). Finally, there is a strong, transient gamma (60–90 Hz) synchronization that occurs shortly after movement onset and lasts about 50 to 250 ms (Cheyne et al., 2008; Dalal et al., 2008; Hall et al., 2011; Muthukumaraswamy, 2010; Muthukumaraswamy et al., 2013; Wilson et al., 2010). These responses are at least somewhat spatially distinct, and are most often localized to the precentral and postcentral gyri bilaterally (stronger contralateral to movement), premotor cortices, parietal cortices, and supplementary motor area (Cheyne et al., 2006, 2008; Gaetz et al., 2010; Heinrichs-Graham and Wilson, 2015; Heinrichs-Graham et al., 2014b; Jurkiewicz et al., 2006; Muthukumaraswamy, 2010; Parkes et al., 2006; Wilson et al., 2010, 2011b).

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Understanding the unique pattern of beta activity prior to, during, and after movement is a fundamental step in determining the basic mechanisms of motor control in humans. Further, a multitude of studies examining the neurophysiology of movement disorders, such as Parkinson's disease (Brown, 2007; Cassidy et al., 2002; Heinrichs-Graham et al., 2014a; Heinrichs-Graham et al., 2014b; Little and Brown, 2014; Pollok et al., 2012; Weinberger et al., 2006), cerebral palsy (Kurz et al., 2014), Tourette syndrome (Franzkowiak et al., 2010; Niccolai et al., 2015; Tinaz et al., 2014), dystonia (Hinkley et al., 2012), and stroke (Rossiter et al., 2014a; Shiner et al., 2015; Wilson et al., 2011a) have shown aberrant sensorimotor beta power at rest and/or during movement. These beta aberrations are often correlated with symptom severity, which suggests that the degree of motor impairment is closely tied to beta activity in the motor cortices. Importantly, movement-related beta oscillatory activity is almost always expressed as a percent power change relative to the baseline, which is generally defined as a 0.5 to 1.0 s period of time that occurs 1.5 to 3.0 s prior to movement onset. Given this practice, abnormal resting beta activity would directly affect motor-related beta activity, as the baseline or "starting place" would be altered. This biasing could potentially mask beta ERD effects related to the baseline, depending on whether the amplitude of the beta ERD or the absolute beta level is the most critical parameter for motor performance. In short, the source of beta aberrations is not entirely clear and no study to date has directly investigated the relationship between resting and movement-related beta activity, in health or disease. Such data is imperative to understanding the basic mechanisms that serve motor control, as well as the impact of pathological beta oscillations on proper movement execution.

Recent work from our lab provides intriguing evidence as to the relationship between resting and task-related motor beta oscillations (Wilson et al., 2014). Briefly, we examined how circadian rhythms (e.g., time of day effects) affected movement-related beta oscillatory activity. Four participants were recorded at rest and during performance of a simple right-hand finger tapping task in the morning, midday, and afternoon for three consecutive days. We found that the amplitude of the peri-movement beta ERD significantly increased as a function of time of day in a number of motor regions, including the primary motor cortices bilaterally, left premotor cortex, and left supplementary area (Wilson et al., 2014). Resting beta levels also increased in these same brain regions as a function of time of day. Interestingly, the PMBR only increased in the SMA as a function of time of day; there were no time of day differences in PMBR amplitude in any other brain region. While this study had a relatively small sample size, it suggests, at least tentatively, that the beta ERD response is closely related to the local level of resting beta activity; essentially, as resting beta levels go up, beta ERD amplitude also goes up. An indirectly related MEG study from Rossiter and colleagues (Rossiter et al., 2014b) examined the effects of age on sensorimotor cortical beta rhythms during rest and a controlled-force grip task. They found a significant positive relationship between age and resting beta amplitude in the left primary motor cortex, such that the older the participant, the more elevated the resting beta amplitude. Further, they found a significant correlation between age and peri-movement beta ERD amplitude during movement execution in the ipsilateral primary motor cortex, with increased age being associated with greater beta suppression (i.e., stronger decrease relative to baseline). While the relationship between resting and movement-related beta activity was not directly probed in this study, the pattern of results again suggests that the beta ERD during movement is closely tied to the spontaneous beta level in the sensorimotor cortices, and also provides new evidence that resting and movement-related beta levels may be modulated by healthy aging. As such, it appears that healthy aging may be a useful, naturalistic model by which to study the association between resting beta activity and movement-related beta oscillatory activity.

In the current study, we used a complex motor sequence paradigm to study the relationship between spontaneous beta activity and movement-related beta oscillations in the context of healthy aging. The goal of this study was two-fold. First, using healthy aging as a model, we aimed to identify whether spontaneous (i.e., resting) beta activity in the motor cortex modulates movement-related beta oscillatory activity. Secondly, we sought to evaluate the effects of healthy aging on motor performance and peri-movement beta oscillatory activity. To this end, we utilized high-density MEG to record healthy younger and older participants at rest and while they performed sequences of finger movements. The peri-movement beta ERD response was imaged using beamforming, and the effects of aging on absolute and relative beta power were assessed. We hypothesized that beta ERD dynamics would be tightly yoked to spontaneous beta power. Further, we hypothesized that healthy older adults would have elevated beta power at rest, and as such would have an elevated beta ERD response during movement.

2. Methods

2.1. Subject selection

We studied 16 healthy younger males (mean age: 28.31 (SD: 5.44) years) and 17 healthy older males (mean age: 65.41 (SD: 7.09) years), all of whom were recruited from the local community. We focused on males in this study due to several recent reports of sex differences in the aging brain (Scheinost et al., 2015; Shaw et al., 2016). Exclusionary criteria included any medical illness affecting CNS function, neurological or psychiatric disorder, history of head trauma, current substance abuse, and the MEG Laboratory's standard exclusion criteria (e.g., dental braces, metal implants, battery operated implants, and/or any type of ferromagnetic implanted material). After complete description of the study was given to participants, written informed consent was obtained following the guidelines of the University of Nebraska Medical Center's Institutional Review Board, which approved the study protocol.

2.2. Experimental paradigm and stimuli

During MEG recording, participants were seated in a nonmagnetic chair within the magnetically-shielded room. Each participant rested their right hand on a custom-made five-finger button pad (see Fig. 1b) while fixating on a crosshair presented centrally. This response pad was connected such that each button sent a unique signal (i.e., TTL pulse/trigger code) to the MEG system acquisition computer, and thus behavioral responses were temporally synced with the MEG data. This allowed accuracy, reaction times, and movement durations (in ms) to be computed offline. In order to create a sufficient baseline, participants initially fixated on a crosshair for 3.75 s before the beginning of each trial (Fig. 1a). After this baseline period, a series of three numbers, each corresponding to a finger on the hand (Fig. 1b), was presented on the screen in black for 0.5 s. After 0.5 s, the numbers changed color, signaling the participant to tap the fingers corresponding to the motor plan sequentially. The participant was given 2.25 s to complete the motor plan and return to rest. Then, the numbers disappeared and only the fixation crosshair remained. This series of slides constituted one trial; Fig. 1a depicts the total time course of a single trial. A total of 160 trials were completed for this task. Participants also completed a six minute block of eyes-closed rest during each MEG session. Total MEG recording time was ~22 min per session (including both tasks).

2.3. MEG data acquisition & coregistration with structural MRI

All recordings were conducted in a one-layer magnetically-shielded room with active shielding engaged. Neuromagnetic responses were sampled continuously at 1 kHz with an acquisition bandwidth of 0.1–330 Hz using an Elekta MEG system with 306 magnetic sensors (Elekta, Helsinki, Finland). Using MaxFilter (v2.2; Elekta), MEG data from each subject were individually corrected for head motion and