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SHORT REPORT

Exploring the role of primary and supplementary motor areas in simple motor tasks with fNIRS

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Abstract Studies employing functional magnetic resonance imaging (fMRI) have highlighted a covariation between the amplitude of hemodynamic responses recorded in primary and supplementary motor areas (M1 and SMA) and the duration of a motor task. A subset of these studies have hinted to a possible functional dissociation between processing carried out in these areas, with SMA primarily involved in action preparation, while M1 involved in action execution. This proposed functional dissociation was explored in the present study using a different technique—functional near-infrared spectroscopy—which enabled a finer-grained monitoring of the temporal characteristics of the hemodynamic response compared to fMRI. Here, hemodynamic responses in M1 and SMA were recorded in 7 participants during a right-finger-tapping task of short (1 s) or long (3 s) duration. Hemodynamic responses of larger amplitude were recorded from both contralateral M1 and SMA during long-duration than short-duration tapping. Furthermore, the analysis of the temporal profiles of these responses revealed a more sustained and prolonged activity for long-duration versus short-duration tapping in M1, but not in SMA. Rather than functionally dissociable areas, the present results are more compatible with the hypothesis that M1 and SMA subserve different, though strongly interacting, functional subroutines subtended in motor task preparation and execution.

Keywords Near-infrared spectroscopy · fNIRS · Motor task · M1 · SMA

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Introduction

One well-established finding encompassing neuroimaging studies on simple movement execution is the positive correlation between movement duration and hemodynamic response amplitude recorded from cortical areas involved in motor processing. When movement duration exceeds 1 s (Glover 1999), long-duration movements are generally associated with hemodynamic responses of larger amplitude relative to movements of short duration. Movement duration appears to modulate differently the hemodynamic response amplitude of two distinct areas underpinning motor action, that is, the primary motor (M1) and supplementary motor (SMA) areas (Miller et al. 2001). Birn et al. (2001), for instance, monitored the hemodynamic responses of 3 participants performing a bilateral finger-tapping task for 4 different durations (i.e., 500, 1,000, 2,000, and 4,000 ms) and found a positive correlation between task duration and hemodynamic response amplitude in M1, while such correlation was nil in SMA. Based on this finding, these authors put forth a serial two-stage functional architecture of the interplay between M1 and SMA ascribing to SMA a first stage of motor planning and to M1 a subsequent stage of motor execution required by the finger-tapping task. Accordingly, neural activity in SMA would be affected by factors underlying task preparation *prior* to movement initiation, whereas M1 would be

affected by factors underlying task execution, such as task duration.

A different account of the interplay between M1 and SMA has been proposed by Kasess et al. (2008), who had participants performing (or imaging to perform) a brief right-hand finger movement at the end of a countdown sequence of numbers, while fMRI data were recorded at higher-than-standard rate to monitor subtle changes in cortical hemodynamics. In contrast to Birn's et al. (2001) proposal, Kasess' et al. (2008) results suggested that M1 and SMA subserve motor task execution interactively, via a closed-loop control circuit composed of basal ganglia-thalamo neurons connecting M1 and SMA. As a result, the activity of SMA and M1 would be temporally intertwined (and not discretely separable) throughout the execution of a movement. Thus, although SMA would be primarily involved in motor planning, its activity would also reflect an active supervising role *during* motor processing occurring in M1.

Aim of the present study was to pit these two accounts one against the other using functional near-infrared spectroscopy (fNIRS; see Leff et al. 2011, for a review of fNIRS explorations of the motor cortex), a technique providing a reasonable compromise between spatial and temporal resolution when monitoring hemodynamic activity reflected in oxygenation changes at the cortical level. Compared to fMRI, fNIRS provides finer-grained temporal information and a richer picture of cortical hemodynamic activity, in terms of concentration changes of both oxygenated (HbO) and deoxygenated (HbR) hemoglobin (see Cutini et al. 2012, for a review of optical imaging in cognitive neuroscience). Furthermore, fNIRS is less sensitive to motion artifacts and imposes negligible physical constraints on participants. Here, we measured hemodynamic responses in M1 and SMA during an event-related right-finger-tapping task of short or long duration. Crucial for the present investigation was the possibility to augment the range of parameters of hemodynamic responses in M1 and SMA, enabling a stringent test including a multi-level parametric comparison of the temporal profiles between the two task duration conditions. While the discrete two-stage account of Birn et al. (2001) would predict a modulation by task duration confined to M1, the interactive account of Kasess et al. (2008) would predict that both M1 and SMA should be concomitantly affected by task duration.

Materials and methods

Participants

Seven male students (6 right-handed)[1] at the University of Padova (mean age 29.3, range 24–35) participated in the experiment after providing informed consent. All participants had normal or corrected-to-normal vision. None of them reported a history of neurological or psychiatric disorders or was under medication at the time of testing.

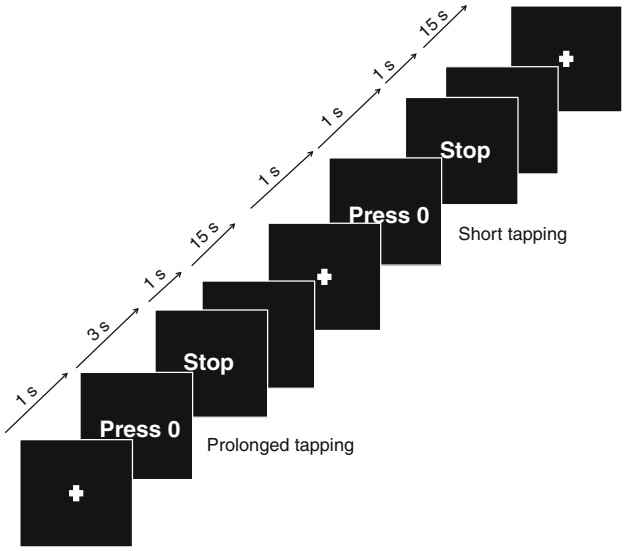

Fig. 1 A schematic representation of the paradigm used

Stimuli and procedure

Each participant was seated on a comfortable chair in a dimly lit room in front of a LCD monitor placed at a viewing distance of 60 cm. The index and middle fingers of the right hand were placed on the '0' key in the numeric keypad. Each trial (Fig. 1) began with a central fixation point, which was followed 1 s later by the instruction 'Press 0' at the center of the screen. At that point, participants had to press the '0' key using both fingers as quickly as possible and to release the key upon the presentation of a 'Stop' signal, which was displayed unpredictably and with equal probability after 1 s on short-duration trials, or after 3 s on long-duration trials. Given the somatotopic organization of neurons in the motor areas under examination in the present study, the expedient of asking participants to respond using two fingers was adopted in order to maximize the probability to detect a reliable hemodynamic response from a larger portion of the motor cortex than it would be possible by employing just one finger. An inter-stimulus interval of 15 s followed the key release. Participants performed a single block of 80 experimental trials.

[1] The left-handed participant was not excluded from analysis in order to prevent lack of power. When examined in isolation, the pattern of cortical activity exhibited by this left-handed participant was not dissimilar from that observed in right-handed participants.

Probe placement and fNIRS data acquisition

Hemodynamic activity was recorded with a multi-channel frequency-domain NIR spectrometer (ISS Imagent^{TM}, Champaign, Illinois), equipped with 20 laser diodes (690 and 830 nm) modulated at 110.0 MHz and two photo-multiplier tubes (PMT) modulated at 110.005 MHz. A recent probe placement method (Cutini et al. 2011) based on the correspondence between 10–20 points and the underlying cerebral regions was used. Sources (1–5) and detectors (L–R: left and right) are shown in Fig. 2. In left hemisphere, source 1 was placed about 1 cm behind C1 (right: C2), while sources 2 and 5 were placed as close as possible to the notional line connecting Cz and C3 (right: C4). This created a triple spatial bind allowing us to place the optodes in a reproducible and reliable way across participants. In particular, channels L1 and R1 were placed on M1, while channels L3 and R3 were placed on SMA (see Sharp et al. 2010). The optical signal detected by the PMTs was converted into concentration changes of HbO and HbR through the modified Beer–Lambert Law (Boas et al. 2002).

Signal processing and data analysis

Individual time series were zero-mean-corrected and band-pass-filtered (0.01–3 Hz) in order to remove very slow drifts and high frequencies. Starting from the 'Press 0' onset, the time series were divided into segments of 12 s each. The algorithm proposed by Scarpa et al. (2011) was applied to minimize the impact of global physiological noise on the optical signal. To further remove measurement noise and the remaining global physiological noise, a non-parametric Bayesian approach (Scarpa et al. 2010) was applied. Peak amplitude and peak latency were computed for each individual mean hemodynamic response in a 2- to 10-s interval following the onset of 'Press 0'. Peak amplitude values were submitted to analysis of variance (ANOVA) considering task duration (short vs. long), hemisphere (left vs. right), and channel (1–5) as within-subject factors. Peak latency values recorded from the left hemisphere (i.e., contralateral to the fingers used for tapping) were submitted to ANOVA considering task duration (short vs. long) and channel (1–5) as within-subject factors. Full-width half-maximum (FWHM) values were computed for each mean hemodynamic response in the left hemisphere to estimate the difference in the temporal distribution of the hemodynamic activity between short- versus long-duration trials. FWHM values represent a measure of how prolonged the hemodynamic activity is in each duration condition. Figure 3 illustrates graphically how these three parameters (i.e., peak amplitude, peak latency, and FWHM) have been computed. FWHM values were submitted to ANOVA considering task duration (short vs. long) and channel (1–5) as within-subject factors. A series of one-tailed t tests was then conducted on the left hemisphere data to compare the two tapping durations based on peak amplitude, latency, and FWHM values recorded from each channel. Since HbR is notoriously characterized by a lower signal-to-noise ratio compared to HbO, the analyses concentrated on HbO data only.

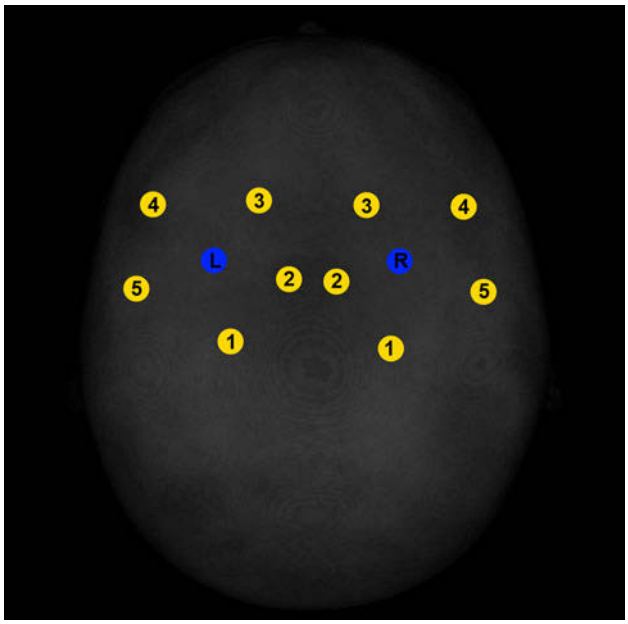

Fig. 2 Sources (*yellow*) and detectors (*blue*) overlaid onto the scalp of a standard MRI template. Channels are located at the cerebral projection of the middle point between each source–detector pair

Results

The analysis on the peak amplitude revealed a significant effect of task duration ($F(1,6) = 12.716$, $p < .05$) and a

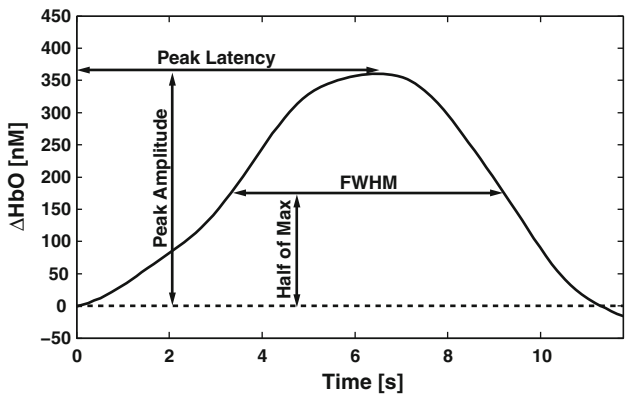

Fig. 3 Sample curve to explain how peak amplitude, peak latency, and FWHM were computed

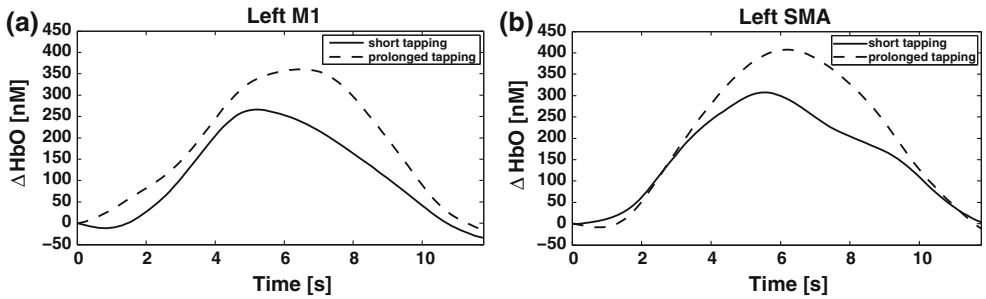

Fig. 4 Mean hemodynamic responses in **a** left M1 (*channel L1*) and **b** left SMA (*channel L3*) during short and prolonged tapping

significant interaction between task duration and hemisphere ($F(1,6) = 14.337$, $p < .01$). Significant differences between the peak amplitude in the two tapping durations were found for all channels (min $t = 2.095$, max $p < .05$) located in the left hemisphere. The ANOVA on peak latency indicated a significant effect of task duration ($F(1,6) = 7.627$, $p < .05$), which was, however, confined to M1 (channel L1: $t = 2.183$, $p < .05$). The analysis on FWHM values indicated a significant effect of task duration ($F(1,6) = 7.355$, $p < .05$), which was confined to M1 and close areas (channel L1: $t = 1.981$, $p < .05$; channel L2: $t = 3.996$, $p < .01$; channel L5: $t = 2.265$, $p < .05$). Fig. 4 shows the mean hemodynamic responses in left M1 (channel L1; MNI coordinates: −28, −19, 70, Brodmann Area 4) and left SMA (channel L3; MNI coordinates: −22, 15, 64, Brodmann Area 6). As visual inspection of Fig. 4 suggests, and as the analysis on FWHM values supports statistically, M1 activity was temporally more sustained throughout the time window of analysis on long- than short-duration trials, whereas the temporal profile of SMA activity was unaffected by task duration ($t = 0.525$, n.s.).

Discussion

The primary aim of the present study was to investigate the presence of a functional dissociation between M1 and SMA, related to the duration of a simple motor task. Our approach was based on fNIRS, a neuroimaging technique that allows to detect subtle temporal differences in cortical hemodynamic activity. Two different models derived from previous fMRI studies (Birn et al. 2001; Kasess et al. 2008) provided different accounts of the interaction between M1 and SMA in a motor task. Birn et al. (2001) proposed a serial, two-stage functional architecture of the interplay between M1 and SMA, with SMA involved in the planning of the movement and M1 in its execution. In this view, only M1 should be modulated by task duration. Kasess et al. (2008), in contrast, hypothesized that M1 and SMA are nodes of an interactive closed-loop control circuit subserving motor task execution, whereby effects of task duration should pervade the entire circuit. Here, we found a relation between peak amplitude and task duration in both cortical areas, a result that provides direct support for Kasess' et al. (2008) proposal. Interestingly, a different temporal pattern emerged in the two examined cortical regions. While the entire temporal profile of M1 response was influenced by task duration (i.e., M1 exhibited a more sustained and prolonged cortical activity during long-duration than short-duration tapping), the temporal profile of hemodynamic responses in SMA was in fact unaffected by task duration, as temporally overlapping responses were observed in SMA during the two tapping conditions (i.e., the initial and final parts of the hemodynamic responses are close to coincident). In conclusion, a further co-registration study exploiting the spatial resolution of fMRI and the temporal resolution of fNIRS might be the optimal approach to better understand the controversial relation between such cerebral areas.

Conflict of interest This supplement was not sponsored by outside commercial interests. It was funded entirely by ECONA, Via dei Marsi, 78, 00185 Roma, Italy.

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