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RESEARCH ARTICLE

Human Supplementary Motor Area Contribution to Predictive Motor Planning

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ABSTRACT. The supplementary motor area (SMA) is involved in planning limb movements. An important component of such planning is the prediction of the sensory consequences of action. The authors used transcranial magnetic stimulation (TMS) to probe the contribution of SMA to motor planning during a predictive load-bearing task. Single TMS pulses were delivered over the SMA after a cue instructing the participant to release a platform supporting his or her right hand, which in turn held a 2 kg mass. Participants were less able to bear the load successfully when TMS was delivered 400–500 ms prior to the response. This result suggests that the SMA contributes to the prediction of the sensory consequences of movement well before movement onset.

Keywords: efference copy, motor planning, supplementary motor area, transcranial magnetic stimulation

The supplementary motor area (SMA) has long been thought to be involved in motor planning (Cunnington, Bradshaw, & Insek, 1996; Deeke, Scheid, & Kornhuber, 1969). The SMA is active before the execution of motor tasks, and is thought to underlie the preparation and readiness for action (Deeke, Grozinger, & Kornhuber, 1976; Lang et al., 1991), even if the motor task is simple (Ikeda et al., 1995), and generates premotor potentials for contralateral, ipsilateral, and bilateral movements (Roland, Meyer, Shibasaki, Yamamoto, & Thomson, 1982). It has been observed, moreover, that the SMA is active even when movement is imagined but not executed (Cunnington, Iansek, Bradshaw, & Phillips, 1996). This has led to suggestions that the SMA plays an important role in preparing and encoding actions prior to initiation, whether those actions are subsequently executed (Cunnington, Windischberger, & Moser, 2005). Other researchers have suggested that the SMA plays a role in representing or encoding action prior to intended and executed movements, during motor imagery, and from the observation of others' actions. SMA has also been suggested to generate and encode motor representations in sustained activity prior to movement, maintaining these representations in readiness for action (Passingham, 1996).

A vital aspect of motor planning is the prediction of the sensory consequences of movement. Recent computational theories of limb motor control have included a forward model of the motor system dynamics that uses an efference copy of the motor command to produce a predictive sensory signal (Shadmehr, Smith, & Krakauer, 2010). This signal can then be used as the basis of comparison with the actual sensory signals to rapidly update the limb movement and achieve endpoint accuracy quickly and efficiently.

The neural mechanisms underlying this predictive process during ongoing movements are poorly understood. Most researchers that have examined this issue have inferred the contribution of the predictive mechanism during the trial-to-trial changes that occur during sensorimotor adaptation (e.g. Bastian, 2008). However, Miall, Christensen, Cain, and Stanley (2007) showed using transcranial magnetic stimulation (TMS) that the cerebellum was involved in predictively compensating for changes in limb position during ongoing responses. The goal of the present study was to examine the contribution of the SMA to predictive processes that take place well before a required motor response. In particular, we used TMS to disrupt the SMA during a load-bearing task (Dufosse, Hugon, & Massion, 1985; Hugon, Massion, & Wiesendanger, 1982) to probe the time window of preemptive control involved in keeping a heavy object in place. The proper timing of this skill is not only of research interest but is also very important in real-life situations requiring precisely timed force adjustments when manipulating objects with the hands. We predicted that SMA TMS would disrupt this predictive planning in a manner consistent with that observed in patients with SMA lesions (Viallet, Massion, Massarino, & Khalil, 1992), and provide new insight into the temporal contribution of the SMA to the processing underlying this skill. Therefore, our primary goal was to gain further insight into the time period around movement onset during which SMA was involved in this task.

Method

Participants

Ten naive healthy volunteers (20–28 years old; 6 women, 4 men) participated in the experiment. All participants were right-handed according to the Edinburgh Handedness Inventory (Oldfield, 1971). The participants signed an informed consent form that explained the nature of the procedure and the small but potential risks of the application of TMS. The research was approved by the Committee for the Protection of Human Subjects at the University of Oregon. Participants were paid $10 for each of the two sessions of the experiment.

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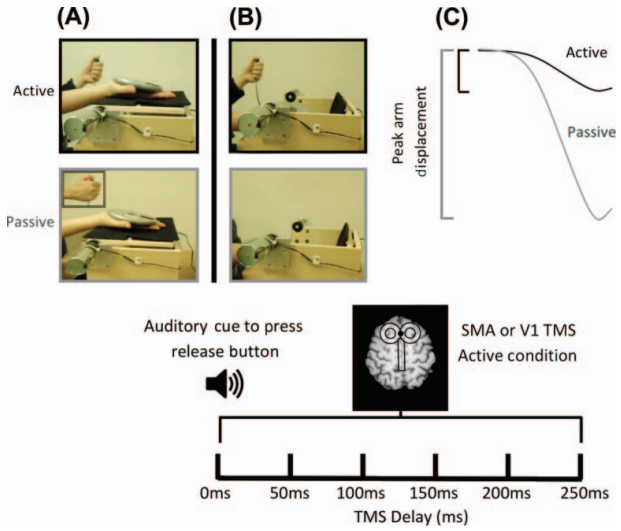

FIGURE 1. Experimental setup and task—participants held a 2 kg mass in their right hand, which in turn was supported by (**A**) a platform that could drop when (**B**) a release button was pressed. The release button press was made either by the participant (active condition, top) or by the experimenter (illustrated by inset in passive condition, bottom) in response to an auditory cue. The participant could neither see the experimenter's hand holding the trigger button nor hear the release cue in the passive condition. (**C**) Peak arm displacement was measured to quantify the effects of the different conditions. (Color figure available online).

Experimental Apparatus

Participants were seated with a 2 kg mass in their right hand, which in turn rested on a horizontal platform (Figure 1A) that could be made to drop from underneath the hand when a release button was pressed (Figure 1B). Two infrared emitting diodes (IREDs) attached to the mass and the platform were monitored by an Optotrak system (100 Hz) and allowed their respective motions to be recorded.

Transcranial Magnetic Stimulation

A 2T Magstim 200 was used to deliver single TMS pulses via a 70 mm figure-eight coil. Stimulation was delivered at 110% of the resting motor threshold for reliably eliciting an observable twitch of the first dorsal interosseus (FDI) of the contralateral hand at the left motor hot point. During two experimental sessions separated by at least 5 days, the stimulating coil was positioned over either the SMA (experimental site), defined as 3 cm posterior to a location one third the distance along the midsagittal line between the nasion and inion (Kwan et al., 2007; Verwey, Lammens, & van Honk, 2002), or the left V1 (control site), defined as 1 cm lateral to the inion. For the SMA site, the coil was oriented parallel to the midsagittal line with the handle pointed in the posterior direction; whereas for the V1 site the handle was oriented perpendicular to the midsagittal line. The order of the SMA and V1 sessions was counterbalanced across participants. Participants wore a swim cap on which markings were made to facilitate stimulator localization and the head was stabilized with respect to the stimulating coil with a clamping system and chin rest. Earplugs were also provided to protect the participant's hearing. None of the participants reported any undesirable side effects resulting from the stimulation.

Experimental Procedures

On each trial, the participant was required to bear the load of the mass when the platform supporting the hand and mass was released. The participant performed this task either passively, during which the experimenter pressed the release button in response to an auditory cue presented through headphones to the experimenter, or actively, during which the participant pressed the release button with his or her left thumb in response to the auditory cue presented through speakers. During each trial, the participant was told to maintain a relaxed arm posture until the platform was released. After each trial was completed, the experimenter returned the platform to the horizontal position and rearmed the trigger mechanism.

After a series of practice trials, the active and passive conditions were completed in a counterbalanced order across participants. In the passive condition, TMS was not delivered, whereas in the active condition TMS was delivered 0, 50, 100, 150, 200, or 250 ms after the auditory cue. We chose to not use TMS on the passive trials because we wanted to use the data from this condition as an uncontaminated baseline with which to compare the effects of TMS in the active condition. An additional set of trials was interleaved in the active condition in which no TMS was delivered. Twenty trials were completed in the passive condition and 120 in the active condition (20 for each of the 5 stimulation times plus 20 no TMS control trials). Muscle activity was not recorded so that we could limit the amount of time the participant was in the experimental setup. The duration of the experiment was ~45 min.

Data Analysis

The main dependent variables of interest were the reaction time in responding to the auditory cue in the active condition and the peak vertical displacement of the arm during the load-bearing response in the active and passive conditions (Figure 1C). These variables were measured to probe whether TMS disrupted the planning and execution of the load-bearing response, respectively. Reaction time was defined as the time from the auditory cue to the button press causing the release of the supporting platform. Peak vertical displacement was the maximum amplitude of the movement of the IRED attached to the mass held in the hand of the participant. To better visualize the effects of TMS, for each participant we calculated the difference in reaction time or peak vertical displacement for each TMS delay with respect to that observed in the active condition without TMS. For the peak displacement variable, we also calculated this

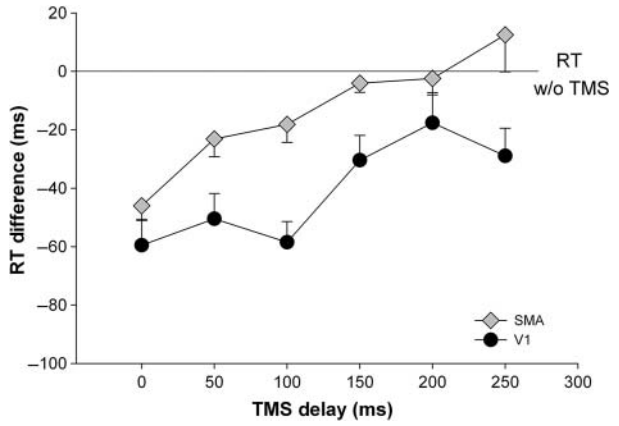

FIGURE 2. Group averages for normalized reaction time calculated by the difference between the active condition with and without transcranial magnetic stimulation (TMS). Positive values indicate longer reaction times in trials with TMS and negative values indicate shorter reaction times in trials with TMS relative to trials without TMS. The horizontal line set to zero represents the normalized reaction time in the active condition without TMS. The reaction time difference is plotted as a function of TMS delay for the supplementary motor area (SMA; gray diamonds) and primary visual cortex (V1; black circles) stimulation sites. Error bars = 1 interparticipant standard error.

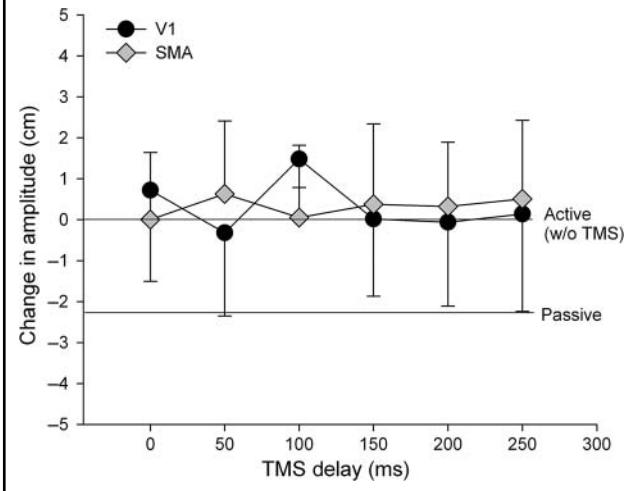

FIGURE 3. Group averages for normalized peak arm displacement calculated by the difference between the active condition with and without transcranial magnetic stimulation (TMS). Positive values indicate smaller peak arm displacements in trials with TMS and negative values indicate larger peak arm displacements in trials with TMS relative to trials without TMS. The horizontal line set to zero represents the normalized peak arm displacement in the active condition without TMS, whereas the lower horizontal line represents the normalized peak arm displacement in the passive condition. The normalized peak arm displacement is plotted as a function of TMS delay for the supplementary motor area (SMA; gray diamonds) and primary visual cortex (V1; black circles) stimulation sites. Error bars = 1 interparticipant standard error.

difference for the passive condition relative to the active condition without TMS as a reference.

The reaction time and displacement data were analyzed for the SMA and V1 TMS conditions using a 2 (stimulation site: SMA vs. V1) × 6 (TMS delay: 0, 50, 100, 150, 200, or 250 ms) repeated measures analysis of variance (ANOVA). Pairwise post hoc multiple comparisons were completed with a Bonferroni corrected alpha level set to .05.

Results

Reaction Time

The group average for reaction time in the active condition during trials without TMS was 377 ± 35 ms. As is clear in Figure 2, when TMS was delivered over either the SMA or V1, reaction time was significantly reduced for the three shortest TMS delays (0, 50, and 100 ms) but not for the three longer delays (150, 200, and 250 ms), TMS Delay main effect: $F(6, 126) = 7.860$, $p < .001$; 0 ms delay versus no TMS: $t(18) = 5.398$, $p < .001$ (effect size = 0.86); 50 ms delay versus no TMS: $t(18) = 3.770$, $p < .001$ (effect size = 0.37); 100 ms delay versus no TMS: $t(18) = 3.924$, $p < .001$ (effect size: 0.39). The main effect of TMS site approached significance, $F(1, 126) = 2.570$, $p = .08$ (effect size = 0.16), indicating that there was a trend for reaction times to be more reduced overall with V1 stimulation. By contrast, the interaction between TMS delay and site was not significant, $F(6, 126) = 1.174$, $p = .324$. The reduced reaction times at the earliest TMS delays are likely due to intersensory facilitation (Nickerson, 1974) or increased arousal from the auditory stimulation associated with the TMS (Bertelson & Tisseyre, 1969; Hackley, 2009). Such reaction time changes have been observed in previous TMS studies (e.g., Sawaki, Okita, Fujiwara, & Mizuno, 1999). It is difficult to explain the trend for a greater overall reaction time reduction with V1 stimulation. The implication is that the nonspecific effects of the TMS on reaction time were more substantial with stimulation delivered near the back of the head.

Peak Arm Displacement

As expected, peak arm displacement was significantly smaller in the active compared to the passive condition when no TMS was delivered, $t(18) = 2.631$, $p < .05$. This is consistent with previous studies (Dufosse et al., 1985; Hugon et al., 1982) and reflects the fact that predictive motor planning processes contributed to the responses in the former but not the latter condition. TMS did not appear to cause any systematic change in this dependent variable when examined as a function of TMS delay (Figure 3), stimulation site main effect: $F(1, 126) = 0.394$, $p = .530$; TMS

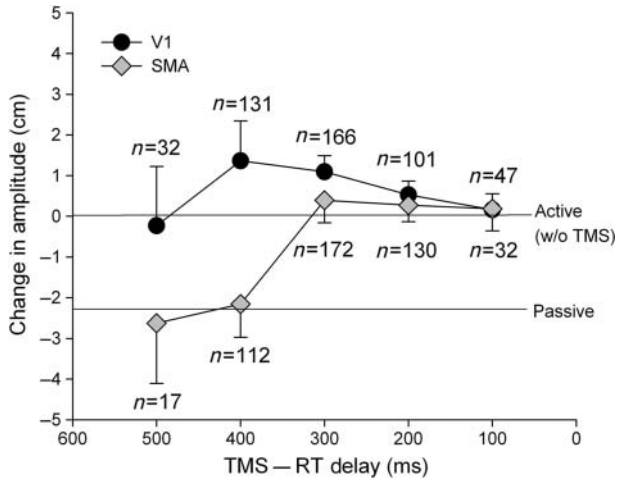

FIGURE 4. Group averages for normalized peak arm displacement as in Figure 3 plotted as a function of the delay between the transcranial magnetic stimulation (TMS) delivery and reaction time. The data have been sorted into five 100 ms bins from 0 to 500 ms for the supplementary motor area (SMA; gray diamonds) and primary visual cortex (V1; black circles) stimulation sites. Error bars = 1 interparticipant standard error; n = number of trials comprising each time bin for each stimulation site.

delay main effect: $F(6, 126) = 0.128$, $p = .996$; interaction: $F(6, 126) = 0.450$, $p = .435$.

It is possible that the lack of a TMS effect was due to the use of the TMS delays as an independent variable in the statistical analysis. Because these delays were triggered by the auditory cue, and the reaction times to those cues varied across trials and participants, the TMS ended up being delivered at variable times relative to the onset of the response. Given that the predictive motor planning processes were presumably more tightly linked to the onset of the response rather than to the auditory cue used to initiate the response, it follows that the potential effects of the TMS would be washed out using this approach.

To examine this possibility we reanalyzed the arm displacement data after sorting it into 100 ms bins based on the difference between the TMS delivery time and the onset of the response. Using this approach, we had sufficient data for five 100 ms time bins from 0 to 500 ms. For example, trials with longer TMS delays and relatively quick reaction times fell mainly into the 0–100 ms time bin, whereas trials with shorter TMS delays and longer reaction times fell into the 400–500 ms time bin.

Figure 4 displays the resulting effects across the different combinations of stimulation site and TMS-RT time bins. In contrast to the data based purely on TMS delay, there was a marked modulation in the peak arm displacement values when plotted as a function of the TMS-RT delay that was specific to the SMA stimulation site. In particular, an ANOVA revealed a significant interaction between stimulation site and TMS-RT delay, $F(4, 783) = 2.780$, $p = .047$. Post hoc Bonferroni corrected pairwise comparisons revealed that this interaction effect was due to differences in peak arm displacement between the SMA and V1 stimulation sites for the 400 ms TMS-RT time bin (effect size = 0.54; the difference also approached significance for the 500 ms time bin). Moreover, within the SMA condition, the TMS-induced change in peak arm displacement at the 400 ms time bin was significantly larger than at the 300, 200, and 100 ms time bins (effect sizes = 0.38, 0.41, 0.36, respectively). Thus, when the effects of stimulation were examined relative to the onset of the response rather than trigger cue, significant modulation was apparent with SMA stimulation, implying that it was playing a role in the predictive processes inherent in the load-bearing task.

Discussion

In the present study, we examined the timing of the contribution of the supplementary motor area (SMA) to predictive motor planning during a load-bearing task. Transcranial magnetic stimulation (TMS) delivered over SMA disrupted the ability of participants to predictively bear the load of a mass when the platform supporting the mass and hand was actively released. This disruption was specific to a critical time period ~400 ms prior to the onset of the response. By contrast, control stimulation over V1 had no impact on the load-bearing responses. Based on this evidence, we suggest that SMA contributes to the predictive processes inherent in this task very early in the planning process, well before the response actually occurs.

The SMA has been shown in many previous studies to be involved in various aspects of movement planning. Perhaps the most direct evidence that this area is involved in the predictive motor planning processes examined in the current study comes from the work of Viallet et al. (1992), who demonstrated that patients with SMA lesions had impaired unloading responses. In particular, the arm displacement magnitudes of SMA patients were similar when the load was removed either actively (by the participant) or passively (by the experimenter)—suggesting that the predictive planning associated with the reduced muscle activation normally occurring before the active unloading condition was disrupted following SMA damage.

The present results confirmed that SMA is vital to these predictive planning processes by showing that load-bearing responses were similar in active and passive conditions when SMA activity was temporarily disrupted with TMS. In addition, insight into the temporal dynamics of these predictive processes was made possible by delivering the TMS pulses at specific times relative to the onset of the response. In particular, the disruption to the predictive processing was limited to a ~400 ms time period prior to the onset of the load-bearing response. This insight into the temporal contribution of specific brain regions to predictive planning processes was examined in two previous TMS studies, which addressed

questions related to those examined in the present study. In the first study, Haggard and Whitford (2004) examined the role of the SMA in the sensory suppression observed during the preparation and execution of voluntary actions. They showed that voluntary finger flexion reduced the perceived magnitude of motor-evoked potentials (MEP) induced by TMS over the motor cortex (M1), but that this suppression was abolished when the SMA was stimulated with TMS 10 ms before the M1 stimulation. Based on this evidence, they suggest that the SMA provides the efference copy signal that contributes to the suppression of sensory signals that takes place during movement generation. In the second study, Miall et al. (2007) used TMS to disrupt cerebellar processing during a task that required participants to make a rapid reaching movement to a target located in front of them during the course of an ongoing medialateral arm movement. Analysis of errors in initial direction of the pointing movement and the final finger position were consistent with the response being planned and executed based on a hand position estimate that was ~120–140 ms out of date (see also Miall & King, 2008).

In both of these studies, the time course over which the TMS effectively disrupted the predictive processes was substantially shorter (10–140 ms) than that observed in the present study (~400 ms). Why might this be the case? One possibility is that the predictive processes were contributing to planning mechanisms that occurred over differing time frames in the tasks used in each study. In the Haggard study, the predictive processes were contributing to a perceptual decision occurring a brief time after an external stimulus (i.e., the M1 TMS). Given the short time frame over which MEPs occur relative to the TMS (e.g. ~20 ms; Day et al., 1989), it is perhaps not surprising that prior stimulation of the SMA can only have an effect at relatively short intervals under these circumstances. With respect to the study by Miall et al. (2007), the task used required an adjustment to the ongoing response based on an external cue rather than the initiation of a response from rest as was the case in the present study. Such online adjustments must occur over a much quicker time frame to be effective relative to the planning processes associated with response initiation. Indeed, it has been suggested that the visual and proprioceptive signals that contribute to online corrections are able to have their influence within approximately 100 ms (Jeannerod, 1988)—a time period in line with the results from the study by Miall et al. and consistent with the disruption of online adjustments following delivery of TMS over the posterior parietal cortex (PPC) just after the onset of reaching movements (Desmurget et al., 1999).

Given that these previous studies appeared to be probing slightly different aspects of the planning process, the fact remains that we observed TMS-induced disruptions to the load-bearing response ~400 ms prior to the onset of the movement. Why was the SMA making a contribution so early in the movement planning period? Electroencephalographic studies have demonstrated that the Bereitschaftspotential can begin as early as 1 s prior to movement onset and likely originates in cortical tissue including the SMA (Deecke, 1987). In addition, monkey single-unit studies have demonstrated that SMA neurons are activated during cue–go planning periods (Tanji & Mushiake, 1996) and are heavily engaged in memory-guided sequences of actions (Mushiake, Inase, & Tanji, 1991; Tanji & Shima, 1994) over similarly long lead times as those observed in the present study. Thus, it is not uncommon to observe SMA contributions to motor planning occurring well before movement onset. The nature of the task we used may have also contributed to these early planning effects: because the response was the same on every trial, participants could start planning well in advance of the actual cue to begin the response. By contrast, the lack of TMS-induced changes later in the planning period (i.e., 100–300 ms before movement onset) suggest that for this task context, the SMA had completed its contribution to response preparation at these times.

It is possible that the disruptions we observed in the load-bearing responses with SMA stimulation were not due to altered predictive motor planning, but rather to disruptions in the ability of the participants to effectively shift attention across specific points in time (Coull, 2004; Coull & Nobre, 2008) or effectively plan actions across time (Bengtsson, Ehrsson, Forssberg, & Ullén, 2005; Bortoletto, Cook, & Cunnington, 2011; Lewis, Wing, Pope, Praamstra, & Miall, 2004)—two sets of functions to which the SMA has previously been shown to contribute and which are broadly consistent with the present results. From this perspective, the SMA is thought to contribute to the temporal expectations of action consequences rather than the sensory predictions associated with those actions, per se. The present results do not necessarily differentiate between these two alternatives and the distinctions between them appear to be quite subtle. For further studies in which these distinctions are systematically manipulated, researchers should fully understand the exact nature of the contribution of the SMA to predictive motor planning.

It is also possible that the changes induced in the load-bearing responses were due to more direct reductions in muscle activation patterns in the arm contralateral to the site of stimulation. In other words, the TMS pulse may have reduced the steady-state muscle activation, thus leading to a greater peak arm displacement when the support platform was released. Because we did not record muscle activity, we were unable to directly assess whether this potential muscle activation effect could account for the changes in arm displacement that we observed. However, previous research examining the interaction between SMA and M1 on the characteristics of MEPs provides some insight. In particular, Matsunaga et al. (2005) demonstrated that repetitive TMS (5 Hz for 2.5 min) over the SMA increased the magnitude of MEPs induced by TMS over M1 suggesting that activation of the SMA can increase the excitability of M1. This implies that the reduction in the ability to bear the load following SMA TMS in the present study was most likely not due to a reduction in steady-state muscle activation.

In summary, we have shown that disruption of the SMA with TMS ~400–500 ms before the onset of a self-induced load-bearing task may lead to a significant increase in the amplitude of the resulting arm displacement to a level that is analogous to that observed when the response is triggered externally. We conclude that this implies that SMA normally contributes to the predictive motor planning processes during this task and does so well before the response is initiated.

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