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MOTOR ACTION EXECUTION IN REACTION-TIME MOVEMENTS: MAGNETOENCEPHALOGRAPHIC STUDY

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Running title: Cortical control of reaction time movement

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ABSTRACT

Objective: Reaction-time (RT) movements are internally planned in the brain. Presumably, proactive control in RT movements appears as an inhibitory phase preceding movement execution. We identified the brain activity of RT movements in close proximity to movement onset and compared it to similar self-paced (SP) voluntary movements without external command.

Design: We recorded 18 healthy participants performing RT and SP fast index finger abductions with 306-sensor magnetoencephalography and EMG. RT movements were performed as responses to cutaneous electrical stimulation delivered on the hand radial nerve area. Motor field (MF) and movement-evoked field 1 (MEF1) corresponding to the sensorimotor cortex activity during motor execution and afferent feedback after the movement were analysed with Brainstorm's scouts using regions of interest analysis.

Results: Primary motor (M1) and sensory (S1) cortices were active before and after movement onset. During RT movements, M1-S1 cortices showed higher activation compared to SP movements. In M1, stronger preparatory activity was seen in SP than in RT.

Conclusions: Both M1 and S1 cortices participated in the movement execution and in the prediction of sensory consequences of movement. Cutaneous stimulation facilitated cortical activation during MF after RT movements, implying the applicability of cutaneous stimulation in motor rehabilitation.

Key Words: Voluntary movement, Movement-related cortical field, Motor cortex, Sensory cortex

INTRODUCTION

Voluntary movements can be roughly defined as intentional actions that are consciously activated or suppressed.[1] Reaction-time (RT) movements are an explicit type of voluntary movement necessary in many behaviours, such as an accurate and fast reaction to an unexpected event, e.g., to avoid an accident. These reactive movements differ from predictive movements where a person is able to plan features of the movement well in advance, such as its timing and strength. A distributed, associative system in the brain is involved in the initiation of any voluntary motor action.[2] Voluntary movement execution can be registered non-invasively with electroencephalography (EEG). EEG-based movement-related cortical potentials (MRCP) are comprised of components well preceding and following voluntary movement onset, and they are used to delineate the cortical regions involved in planning, executing and processing sensory feedback of voluntary movements.[3-6]

Movement-related cortical fields (MRCF) are equivalent to MRCPs recorded with magnetoencephalography (MEG) and consist of the readiness field (RF) prior to movement onset, the motor field (MF) at the time of movement execution, and the movement-evoked field 1 (MEF1) first post-movement component.[7] The difference between electrically and magnetically recorded signals is mainly observed in the premovement component, where RF is registered much later than the readiness potential mainly because of its source orientation in the hand premotor cortices and electrical current direction in the supplementary motor area (SMA). A radial orientation of sources in the premotor cortex and bilateral activation in the posterior SMA (where concurrent activities likely cancel each other out) challenge RF detection in MEG.[8]

Previous MRCF studies of self-paced (SP) movements have indicated minor ipsilateral activation of the motor cortex in addition to major contralateral activation and peripheral afferent contribution to MEF1.[7,9] It is agreed that the MF component is generated in the contralateral area 4.[8,10] The exact generators of post-movement deflections are not unanimously agreed upon.[11] Additionally, preparation for self-initiated voluntary movements differs from that of the RT movements, as the preparation of the RT movements is strictly engaged in the temporal evaluation and expectation of the go-signal. Psychophysiological studies have amassed further research concentrating on controlling spatial attention as opposed to temporal. It is noteworthy that among patients suffering from severe disorders of consciousness, there are those who are able to produce event-related potential signatures of conscious access to temporal stimuli, but if only spatial stimuli are used to elicit attention, they do not arouse their consciousness.[12,13] Universally physical medicine and motor rehabilitation techniques utilise voluntary movements and various electrical stimulation strategies, but little is known of their diverse processing in the brain. Behavioural movement times and reaction times have been carefully analysed,[14,15] but knowledge of their differences in cortical processing at the millisecond-level is sparse. The exact timing and activation patterns of cortical sensorimotor sources contributing to RT movement are important in distinguishing between RT and SP movements, not only to understand the details of motor execution but also because understanding these processes would allow for better use of MRCF components in neurological diagnosis and development of different motor rehabilitation methodologies. For instance, patients with stroke who belong to a high-fatigue group show slower movement times than otherwise corresponding patients with stroke who belong to a low-fatigue group.[14] Furthermore, it was shown recently that cutaneous electrical stimulation delivered at the time of command for RT movement yielded faster reaction times and facilitated movement execution in patients with chronic stroke than a similar task without

electrical stimulation.[15] It may be that with a better understanding of the various factors affecting voluntary motor control, we will be able to enhance rehabilitation methodologies.

We investigated the brain areas involved in the immediate planning and execution of RT and SP movements and whether their activation patterns differ between these movement types. The execution of these movements was recorded with whole-head MEG and actual movement onset with electromyography (EMG). The RT task was compared to a corresponding voluntary SP movement. Active cortical sources were analysed in the same time window for both movement types before and after the movement execution.

EXPERIMENTAL PROCEDURES

Eighteen healthy adults participated in the study (10 men, mean age 30.4 ± 6.1 years). The research plan was approved by the Ethics Committee of the University of Jyväskylä, and the study was conducted in accordance with the Declaration of Helsinki. All participants gave a written informed consent prior to participation. None of the participants had a history of neurological or psychiatric diseases, alcoholic or narcotic addictions, and they had no metal objects in the head or upper body that would contaminate the MEG recording. All participants were right-handed.

Experimental conditions

The required movement was a fast abduction of the right index finger while the hand and forearm were resting on a table, fingertips lightly touching the table surface (see Fig. 1 for the

recording device and electrode placements on the hand). Condition I: SP, 80 self-paced finger abductions were performed randomly at 4–6-second intervals. Condition II: RT, reaction time movements to weak electric stimulus (square-wave pulse, 0.2 ms duration) (Digitimer Ltd., model DS7A, Welwyn Garden City, UK). Eighty stimuli were delivered to the dorsal surface of the right hand randomly at 4–6-second intervals as go-stimuli. The stimulating electrodes (1 cm in diameter) were placed at the proximal end of the first metacarpal (anode) and at the distal head of the ulna (cathode). The stimulus intensity was set to twice the individual sensory threshold (mean 7.7 ± 2.1 mA). The stimulus did not induce any reported pain.

Recording

In both conditions, the surface EMG was recorded bipolarly from the first dorsal interosseus muscle (FDI) with a bandpass of 10–330 Hz (6^{th}-order Butterworth IIR filter) and the gain set to 2000. The FDI muscle location was determined while the participant was asked to abduct the index finger against resistance. The EMG electrodes were placed over the FDI muscle belly oriented according to the muscle origin and insertion. Eye movements were recorded with an electrooculogram with a bandpass of 0.1–330 Hz. Five head position indicator (HPI) coil locations in relation to nasion and bilateral preauricular points with additional points from the scalp and nose crest were measured with a 3-D digitiser (Fastrak, Polhemus, Vermont, USA). MEG was recorded in a magnetically shielded room (Vacuumschmelze, GmbH, Hanau, Germany) with the helmet-shaped 306-channel device (Elekta Neuromag®, Triux™, Stockholm, Sweden). MEG signals were recorded with a bandpass of 0.1–330 Hz. Both MEG and EMG signals were stored for offline processing.

To further evaluate the similarity of the voluntary movements, acceleration recordings of movements were also performed with five participants outside the MEG chamber. SP and RT movements were recorded simultaneously with the EMG of the FDI muscle and fingertip angular acceleration. The 3-axial accelerometer (Bittium Biomonitor ME6000, Bittium, Oulu, Finland) was attached to the distal phalanx of the index finger.

Data analysis

First, MEG data were filtered with MaxFilter software (Elekta Neuromag®, Stockholm, Sweden) using signal space separation.[16] Data preprocessing and analysis were conducted with Brainstorm software (version 2/15/2017).[17] Since no individual MRI images were available, an anatomy template (ICBM152) was used across participants in Brainstorm. According to MEG guidelines,[18] individual digitised head shapes can be used instead of the individual MRI to approximately align the participant's head to a template head,[19] allowing for an average among the participants. Anatomy templates were aligned and warped for each participant with HPI data registered before the MEG measurements.[20] EMG onset, designating movement onset, was determined visually by the researcher as the beginning of a clear increase in EMG amplitude deviating from the EMG baseline (see Figs. 3A and B). Event markers were applied at each EMG onset time point for each movement. EMG was baseline-corrected and rectified in order to be able to build grand averages across individual participants and calculate integrals on the same scales. Event markers for the electrical stimulation in condition II were recorded with MEG registration, and reaction times were calculated by subtracting the stimulus onset time point from the EMG onset time point. Artefacts from eye movements were cleaned using the signal-space projection method.[21] Data

were segmented to epochs from -1000 to +200 ms in relation to EMG onsets, and the first 100 ms of the epoch was used as a baseline.

The forward model was computed with overlapping spheres, with one local sphere assigned to each sensor. Source models were generated from each participant's averaged epochs using minimum norm estimate current density maps. Orientations of source dipoles were constrained normally to the cortical surface, and all MEG sensors were included. Current density maps were normalised with Z-transformation with respect to the baseline (-1000 to -900 ms). Regions of interest (ROI) were identified from current source density maps and were analysed using Brainstorm's scout function. Scouts were applied for each participant's averaged source map using MF and MEF1 waveform components as temporal cues. The locations of the scouts were determined in the source map by the maximum amplitude during two time periods: from -10 to +30 ms (MF) and from +110 to +140 ms (MEF1). The RF activity was identified from the MF scout as a slow rising waveform prior to the movement onset. Scouts, representing mean activity in each ROI, were set to cover 20 vertices each, corresponding to approximately 4 cm^2 on the cortical surface. Within MF and MEF1 scouts, the maximum amplitudes and their corresponding MNI coordinates, as well as a mean time period of 10 ms for RF prior to stimulus in the RT task (from -270 to -261 ms), were used to compare brain activity between conditions.

Statistical analysis

Statistical analysis was performed with IBM SPSS 24 (IBM, Armonk, USA). All group analysis in the MEG data was done in source space. Variables were tested for normality with

the Shapiro–Wilk test. Normally distributed variables were tested with a paired samples t-test and not normally distributed variables with the Wilcoxon signed-rank test.

RESULTS

The grand average MEG waveforms of RT and SP tasks are depicted for data visualisation (Fig. 2). The gradiometer waveforms are shown flattened from the helmet-shaped MEG from -1000 ms to +200 ms. As the voluntary movements were performed with the right hand, the contralateral (left) sensorimotor cortex shows major components. The left side of Fig. 3 shows typical examples of one individual's rectified EMG of the FDI muscle in RT (A) and in SP (B) movements.

Two scouts were analysed from each participant's data, assessing both the scout's maximum amplitude and its latency in relation to 0 (Figs. 3C and D). During the motor preparation, before the go-stimulus, a significant difference was found in the averaged amplitude in the RF time period from -270 ms to -261 ms ($p = 0.028$, $Z = -2.20$), where the SP task displayed stronger amplitudes (see also Table 1). In the MF activity, peak amplitude in RT was stronger than in SP ($p = 0.001$, $Z = -3.29$), but their peak latencies at 17 ms did not differ ($p = 0.971$). The statistical analysis of the MF peak amplitude coordinates revealed no location difference in MF between RT and SP movements. MEF1 scout peak amplitude in the RT task was significantly stronger than in the SP task ($p = 0.048$, $Z = -1.98$). A latency difference was also detected. The RT condition (113 ms) showed an earlier peak amplitude than the SP condition

(122 ms) ($p = 0.023$, $t = 2.51$, $df = 17$); however, the source locations did not differ between conditions. When the peak amplitude location coordinates (x, y, z) were compared between MF (RT: -32, -16, 53; SP: -32, -12, 56) and MEF1 (RT: -38, -25, 50; SP: -39, -27, 50) within conditions, MF peaked anterior to MEF1 (SP, y-coordinate $p = 0.001$, $t = 6.68$, $df = 17$; RT y-coordinate $p = 0.002$, $t = 3.64$, $df = 17$). In the SP condition, the MEF1 mean peak location was deeper and more lateralised than that of MF for about 7 mm in both depth and laterality. The source amplitude differences are further visualised in Fig. 4, with grand average current density maps showing MF and MEF1 at mean peak amplitude time points (17 ms for MF and 113 ms/122 ms for MEF1).

The analysed mean number of repetitions in the RT task was 76 ± 3.2, and in the SP task it was 72 ± 17.3. Looking at all 18 participants, the mean EMG integrals for the window 0–200 ms differed significantly ($p = 0.003$, $t = 3.42$, $df = 17$). Integrals were higher in RT than in SP, with a mean of 14364 ± 7473 μV vs. 11679 ± 6677 μV. As we aimed for similar movements, movement acceleration was also measured outside the MEG chamber. These recordings of five participants did not show differences between RT and SP movements' accelerations ($p = 0.339$, $t = 1.09$, $df = 4$) or EMG integrals ($p = 0.329$, $t = 1.11$, $df = 4$). The mean reaction time for all 18 participants was 221 ± 50 ms.

DISCUSSION

We identified the sensorimotor components of MRCF in both RT and SP movements in healthy participants. Sources of the MF and MEF1 components were localised in M1 and S1 cortices based on MNI coordinates. Supplementary motor area (SMA) activation preceding motor execution has usually been best localised with EEG.[3,8] RF, most likely originating

232 from the SMA confirmed in intracortical recordings, has not been easily identifiable in MEG
233 studies.[22,23] A likely reason for the somewhat problematic identification of RF is that MEG
234 detection is inherently biased towards tangential cortical currents and may fail to localise
235 more radially oriented sources. Another factor, which may explain relatively weak RF in our
236 data and others, is the possible cancellation effect of bilateral deep tangential SMA
237 activation. In the present study, we did not focus on the early preparatory period well before
238 movement, which is common in EEG studies, but rather on the time very close to the EMG
239 onset.
240
241 The main activities in M1-S1 were localised in the contralateral hemisphere of the active
242 hand. Minor bilateral hemispheric activation in the sensorimotor and premotor cortices has
243 been reported in voluntary movement.[24] Also, in our data, a trace of ipsilateral activity in the
244 RT condition can be observed in Fig. 4.
245
246 Brain activation fields were different between RT and SP movements in the present MF and
247 MEF1 scouts and in the current density maps (Figs. 3 and 4). A statistically significant
248 difference between conditions in the movement preparation phase was observed. SP
249 movement displayed stronger RF activity in M1 observed in the MF scout activation before
250 movement execution. This activity around 260 ms prior to EMG onset was observed in M1,
251 which agrees well with previous reports utilising MEG.[7,10] Executing RT or SP movements
252 has previously been suggested to activate the same brain areas in both types of movement,
253 similarly in M1-S1 cortices but differently in the SMA and the anterior cingulate cortex
254 (ACC).[1] Those findings are based on cerebral blood flow experiments, which provide
255 location information but cannot provide equally accurate information regarding timing as
256 MEG. The activity in the SMA and the ACC shown by Jenkins et al.,[1] as well as the fronto-

cortical-striatal system implicated by Wijeakumar et al.,[25] likely represent a corresponding planning and engagement phase, as we detected in RF.

Stronger activation in RF suggests that facilitatory processes in M1 contribute to movement preparation in SP, while inhibition in the RT condition may occur. A recent fMRI experiment supports our view that important parts of the larger network in the fronto-cortical-striatal system are engaged in planning relevant motor events.[25] This system does not play a selective role in response inhibition, which may occur while waiting for the go-signal in the RT condition. It is possible that our RF period in RT before the go-stimulus is more actively inhibited than the same period in SP. This idea is supported by a transcranial magnetic stimulation study showing that a significant inhibition related to task anticipation influences a cortical representation of task-relevant muscles.[26] In the present study, only after the go-stimulus had occurred in RT, M1 activity increased and reached activation that was significantly stronger than in SP (Fig. 3C), even though peak activities occurred at the same time in both conditions.

Our MF activation coincided with previous reports of M1 activation in voluntary finger movements.[5,10,11] We detected higher amplitudes in MF and MEF1 components in RT compared to SP. We have to consider that the electrical go-signal might also be a factor for higher amplitudes in RT. The contralateral sensory cortex would be activated about 20 ms after the stimulus, and subsequent facilitatory components may occur; however, these activations are well over by the time of MF. Still, there may be an overall facilitatory effect following cutaneous stimulation, as shown in some behavioural studies.[15] Thus, the difference in the MF and MEF1 amplitudes in our data may reflect divergent progressive facilitation of the cortical-subcortical network, contributing to various parameters of the

movement initiation sequence. The imperative cue may release higher subthreshold activation in RT compared to SP, producing a higher peak amplitude. Another thing to consider is that part of the difference in cortical activity may be explained by the higher force generated in RT movement. During the MEG recording, the produced force could not be measured, but our separate angular acceleration and EMG recordings revealed a similarity between RT and SP movements. Rapid voluntary muscle activation during the first 50–70 ms of force development, the relevant time period in this study, is achieved by a reduction in the motor unit recruitment threshold and an increase in the motor unit discharge rate, but it is still unclear how much of this control is achieved by supraspinal or spinal control and/or is related to agonist-antagonist control.

The first deflection *after* the movement execution, MEF1, reached its peak amplitude earlier in RT than in the SP task (Fig. 3D). MEF1 has been thought to represent proprioceptive feedback from muscle spindles and possibly other sources, such as cutaneous afferents.[9,24,27] Slightly diverging origins of MEF1 have been suggested, such as from the post-central region, Brodmann's area 3a and the precentral motor area.[9-11,23,28] A rather precise origin of MEF1 can be suggested based on our current density maps and their peak coordinates. There is a significant location difference in the anterior-posterior direction between MF and MEF1 (in RT, 9 mm, and in SP, 15 mm), implying a more posterior generator for MEF1 compared to MF. Moreover, the MEF1 source in SP was deeper than in MF, allowing speculation of the generator location in area 3a for MEF1. Our MEF1 scout waveform overlapped with the MF scout waveform, already showing activity before EMG onset, which may indicate a contribution from both generators in the M1-S1 cortex to both components. As emphasised by Wolpert and Flanagan,[29] in motor control, a forward model can be exploited to predict the sensory consequences of planned motor actions. Presumably, both pre- and postcentral

regions are involved in the proactive control of voluntary movements, and this is also suggested by intracortical recordings by Sun et al.[30] We believe that the present data at large support the internal forward model functioning throughout human voluntary movement.

Previous studies have shown that electric stimulation used in the reaction-time movement paradigm facilitates movement execution in healthy participants and patients with stroke.[15] The current results provide evidence for dissociated cortical facilitation after reaction time and self-paced movements. This may be helpful information in designing individualised therapies in various types of sensory and motor disorders. In practice, this data support the application of cutaneous stimulation to assist motor rehabilitation.

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323 References

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Figure legends

Figure 1. Participant seated in the 306-channel MEG device (A) with right hand resting on top of the table. The start position for fast second-finger abduction in B. The end position of the abduction followed by immediate return to start position in C. Surface EMG electrodes are placed over the first dorsal interosseous muscle (blue cords). Electrical stimulation electrodes are placed at the wrist (white cords and ground electrode placed proximally).

Figure 2. Gradiometer grand average waveforms of 18 participants in each condition in relation to EMG onset shown from -1000 to 200 ms. Larger amplitudes were recorded over the left hemisphere, contralateral to the right-hand movements in the tasks.

Figure 3. The rectified and averaged EMGs of one individual in reaction time (A) and self-paced (B) tasks. Point 0 depicts EMG onset. Electrical artefact originating from electrical stimulus is visible in RT task spread around -200 ms as reaction times differ. Scout amplitude waveforms of motor field, MF (including RF activity) (C) and movement-evoked field 1, MEF1 (D) components shown from grand average current density maps (red waveform = reaction time, blue = self-paced). EMG onset at 0 ms. Vertical black line at 221 ms indicates stimulus onset before RT movement.

Figure 4. Grand average current density maps of motor field (MF) and movement-evoked field 1 (MEF1). Left = reaction time (RT) task, right = self-paced (SP) task. MF peak activity at 17 ms and MEF1 maps are shown for RT task at 113 ms and for SP task at 122 ms.

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Table 1

TABLE 1. Comparison of reaction time (RT) and self-paced (SP) tasks as analysed in scout parameters and EMG, means (SD).

	RT	SP	Sig.
MF scout amplitude	9.17[z] (2.94)	6.94[z] (2.30)	0.001**[w]
MEF1 scout amplitude	12.37[z] (5.28)	10.02[z] (3.08)	0.048*[w]
MF scout peak latency (ms)	17.22 (12.14)	17.00 (24.21)	0.971[p]
MEF1 scout peak latency (ms)	113.70 (13.52)	122.83 (18.05)	0.023*[p]
MF scout mean amplitude from -270 to -261 ms	0.93[z] (0.51)	1.66[z] (1.18)	0.028*[w]
EMG integral (μV)	14364 (7473)	11679 (6676)	0.003**[p]

[p] Paired samples t-test. * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$
[w] Wilcoxon Signed Rank -test
[z] Z-score

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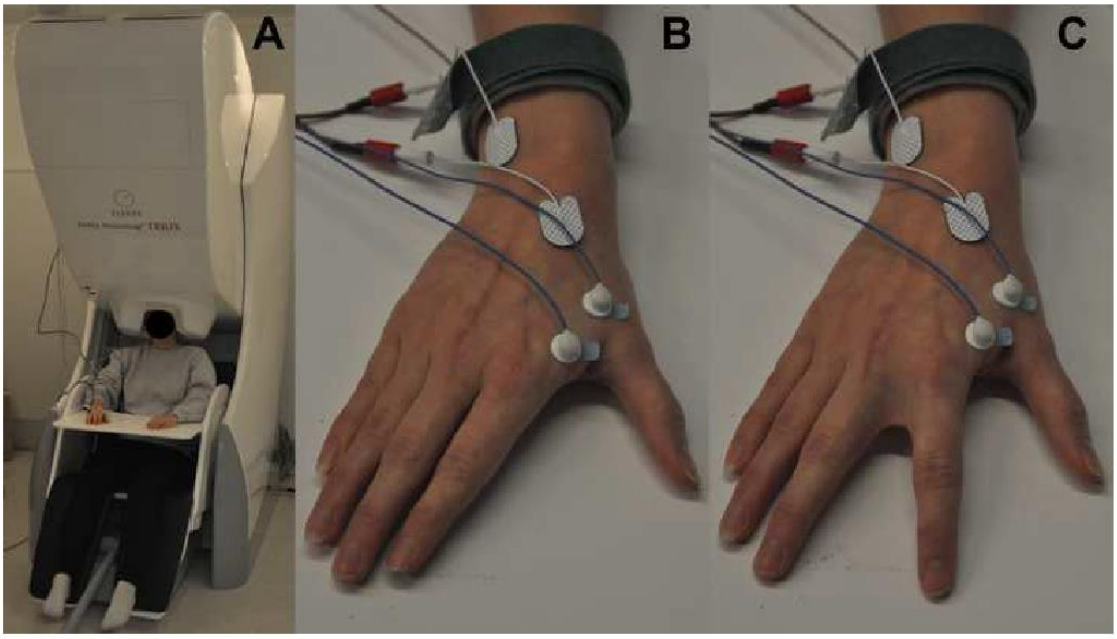
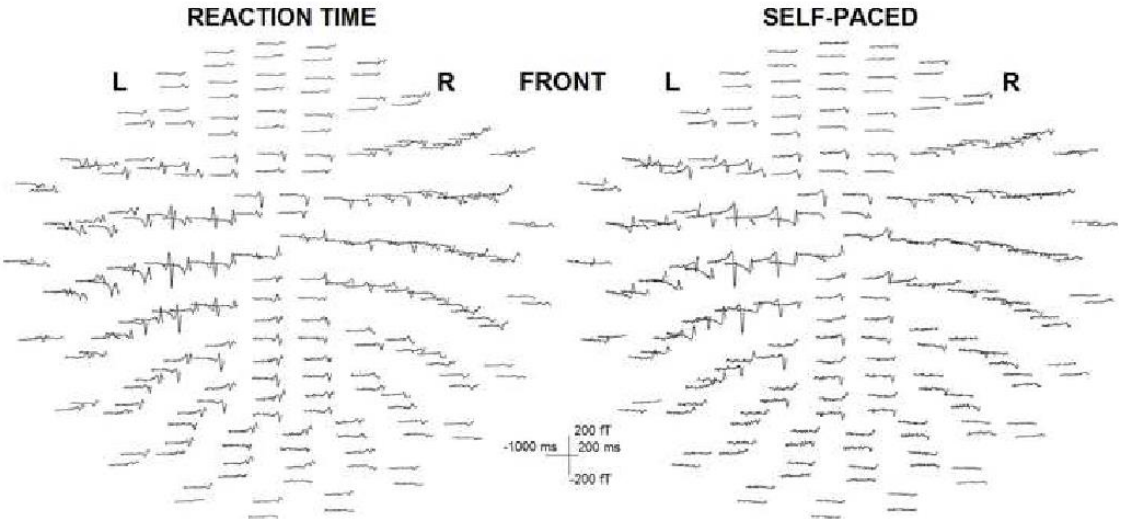
Figure 1



438

Figure 2

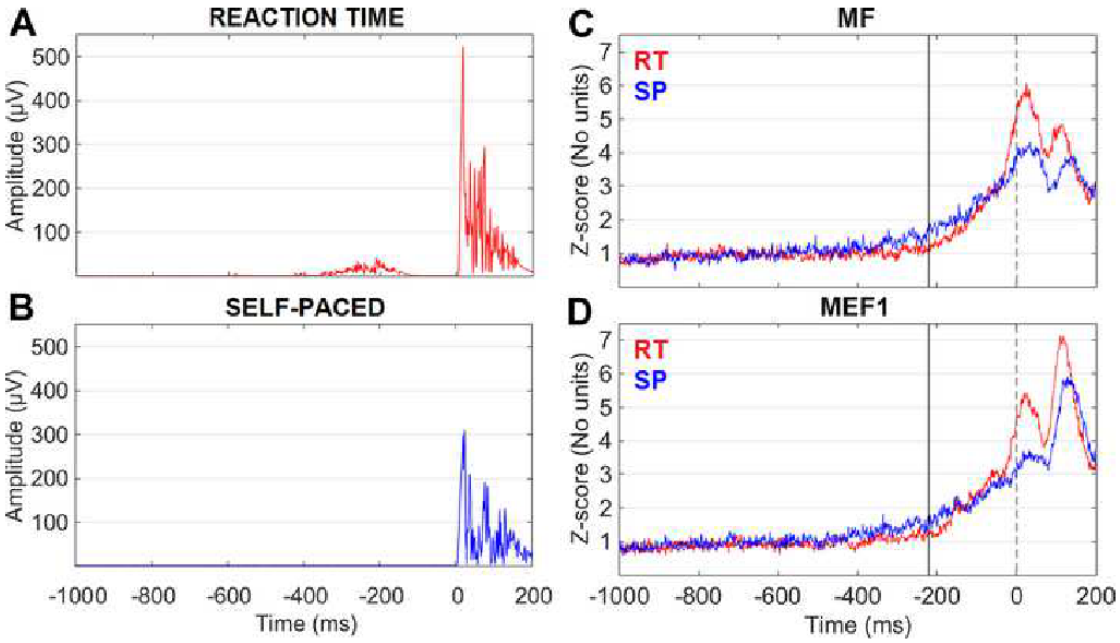


439

440

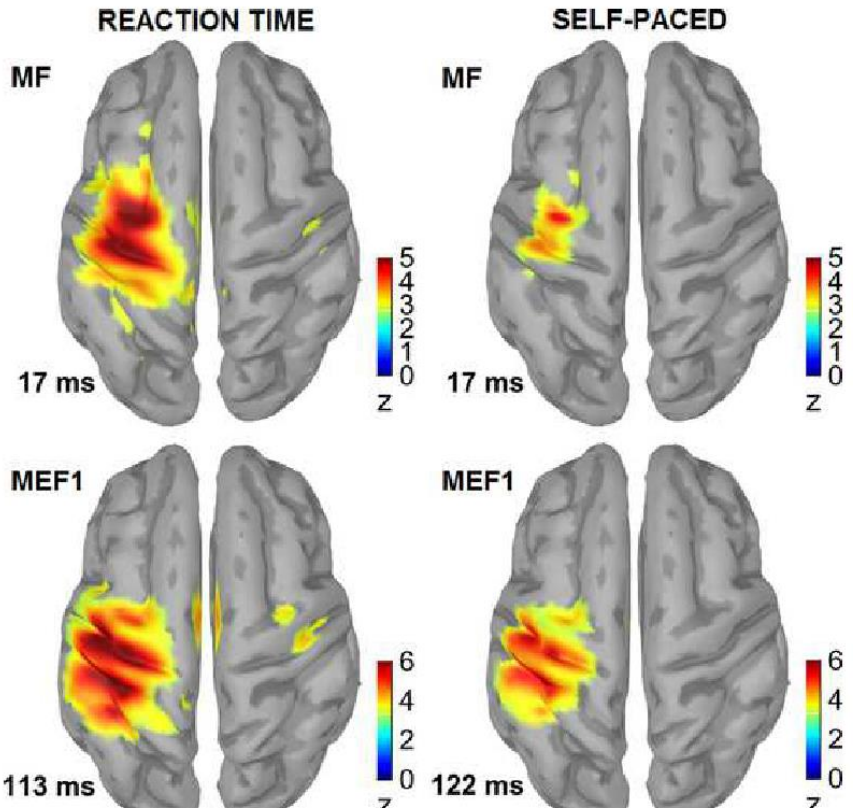
441

Figure 3



442

Figure 4



443