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2 **MOTOR ACTION EXECUTION IN REACTION-TIME MOVEMENTS:**
3 **MAGNETOENCEPHALOGRAPHIC STUDY**

4

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8

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11

12 Running title: Cortical control of reaction time movement

13

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ABSTRACT

23

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

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

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

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

42

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

INTRODUCTION

45

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

57

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

68

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

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

96

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

103

104

EXPERIMENTAL PROCEDURES

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

112

Experimental conditions

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

116 recording device and electrode placements on the hand). Condition I: SP, 80 self-paced finger
117 abductions were performed randomly at 4–6-second intervals. Condition II: RT, reaction time
118 movements to weak electric stimulus (square-wave pulse, 0.2 ms duration) (Digitimer Ltd.,
119 model DS7A, Welwyn Garden City, UK). Eighty stimuli were delivered to the dorsal surface
120 of the right hand randomly at 4–6-second intervals as go-stimuli. The stimulating electrodes
121 (1 cm in diameter) were placed at the proximal end of the first metacarpal (anode) and at the
122 distal head of the ulna (cathode). The stimulus intensity was set to twice the individual
123 sensory threshold (mean 7.7 ± 2.1 mA). The stimulus did not induce any reported pain.

124

125 **Recording**

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

138

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

144

145 **Data analysis**

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

162 were segmented to epochs from -1000 to +200 ms in relation to EMG onsets, and the first
163 100 ms of the epoch was used as a baseline.

164

165 The forward model was computed with overlapping spheres, with one local sphere assigned
166 to each sensor. Source models were generated from each participant's averaged epochs using
167 minimum norm estimate current density maps. Orientations of source dipoles were
168 constrained normally to the cortical surface, and all MEG sensors were included. Current
169 density maps were normalised with Z-transformation with respect to the baseline (-1000 to -
170 900 ms). Regions of interest (ROI) were identified from current source density maps and
171 were analysed using Brainstorm's scout function. Scouts were applied for each participant's
172 averaged source map using MF and MEF1 waveform components as temporal cues. The
173 locations of the scouts were determined in the source map by the maximum amplitude during
174 two time periods: from -10 to +30 ms (MF) and from +110 to +140 ms (MEF1). The RF
175 activity was identified from the MF scout as a slow rising waveform prior to the movement
176 onset. Scouts, representing mean activity in each ROI, were set to cover 20 vertices each,
177 corresponding to approximately 4 cm² on the cortical surface. Within MF and MEF1 scouts,
178 the maximum amplitudes and their corresponding MNI coordinates, as well as a mean time
179 period of 10 ms for RF prior to stimulus in the RT task (from -270 to -261 ms), were used to
180 compare brain activity between conditions.

181

182 **Statistical analysis**

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

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

187

188

189

190

RESULTS

191 The grand average MEG waveforms of RT and SP tasks are depicted for data visualisation
192 (Fig. 2). The gradiometer waveforms are shown flattened from the helmet-shaped MEG from
193 -1000 ms to +200 ms. As the voluntary movements were performed with the right hand, the
194 contralateral (left) sensorimotor cortex shows major components. The left side of Fig. 3
195 shows typical examples of one individual's rectified EMG of the FDI muscle in RT (A) and
196 in SP (B) movements.

197

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

208 (122 ms) ($p = 0.023$, $t = 2.51$, $df = 17$); however, the source locations did not differ between
209 conditions. When the peak amplitude location coordinates (x , y , z) were compared between
210 MF (RT: -32, -16, 53; SP: -32, -12, 56) and MEF1 (RT: -38, -25, 50; SP: -39, -27, 50) within
211 conditions, MF peaked anterior to MEF1 (SP, y -coordinate $p = 0.001$, $t = 6.68$, $df = 17$; RT y -
212 coordinate $p = 0.002$, $t = 3.64$, $df = 17$). In the SP condition, the MEF1 mean peak location
213 was deeper and more lateralised than that of MF for about 7 mm in both depth and laterality.
214 The source amplitude differences are further visualised in Fig. 4, with grand average current
215 density maps showing MF and MEF1 at mean peak amplitude time points (17 ms for MF and
216 113 ms/122 ms for MEF1).

217

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

226

227

DISCUSSION

228 We identified the sensorimotor components of MRCF in both RT and SP movements in
229 healthy participants. Sources of the MF and MEF1 components were localised in M1 and S1
230 cortices based on MNI coordinates. Supplementary motor area (SMA) activation preceding
231 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-

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

259

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

272

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

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

292

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

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

310

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

317

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322

323 References

324

325 1. Jenkins IH, Jahanshahi M, Jueptner M, Passingham RE, Brooks DJ. Self-initiated versus
326 externally triggered movements. II. the effect of movement predictability on regional cerebral
327 blood flow. *Brain*. 2000;123 (Pt 6)(Pt 6):1216-1228.

328 2. Deecke L. There are conscious and unconscious agendas in the brain and both are
329 important-our will can be conscious as well as unconscious. *Brain Sci*. 2012;2(3):405-420.
330 doi: 10.3390/brainsci2030405 [doi].

331 3. Deecke L, Eisinger H, Kornhuber HH. Comparison of Bereitschaftspotential, pre-motion
332 positivity and motor potential preceding voluntary flexion and extension movements in man.
333 *Prog Brain Res*. 1980;54:171-176. doi: S0079-6123(08)61621-0 [pii].

334 4. Colebatch JG. Bereitschaftspotential and movement-related potentials: Origin,
335 significance, and application in disorders of human movement. *Mov Disord*. 2007;22(5):601-
336 610. doi: 10.1002/mds.21323 [doi].

337 5. Milliken GW, Stokic DS, Tarkka IM. Sources of movement-related cortical potentials
338 derived from foot, finger, and mouth movements. *J Clin Neurophysiol*. 1999;16(4):361-372.

339 6. Tarkka IM, Hallett M. Topography of scalp-recorded motor potentials in human finger
340 movements. *J Clin Neurophysiol*. 1991;8(3):331-341.

341 7. Kristeva R, Cheyne D, Deecke L. Neuromagnetic fields accompanying unilateral and
342 bilateral voluntary movements: Topography and analysis of cortical sources.
343 *Electroencephalogr Clin Neurophysiol*. 1991;81(4):284-298.

344 8. Shibasaki H, Hallett M. What is the Bereitschaftspotential? *Clin Neurophysiol*.
345 2006;117(11):2341-2356. doi: S1388-2457(06)00229-X [pii].

- 346 9. Cheyne D, Endo H, Takeda T, Weinberg H. Sensory feedback contributes to early
347 movement-evoked fields during voluntary finger movements in humans. *Brain Res.*
348 1997;771(2):196-202. doi: S0006-8993(97)00765-8 [pii].
- 349 10. Suzuki M, Wasaka T, Inui K, Kakigi R. Reappraisal of field dynamics of motor cortex
350 during self-paced finger movements. *Brain Behav.* 2013;3(6):747-762. doi: 10.1002/brb3.186
351 [doi].
- 352 11. Onishi H, Sugawara K, Yamashiro K, et al. Neuromagnetic activation following active
353 and passive finger movements. *Brain Behav.* 2013;3(2):178-192. doi: 10.1002/brb3.126 [doi].
- 354 12. Faugeras F, Rohaut B, Weiss N, et al. Event related potentials elicited by violations of
355 auditory regularities in patients with impaired consciousness. *Neuropsychologia.*
356 2012;50(3):403-418. doi: 10.1016/j.neuropsychologia.2011.12.015 [doi].
- 357 13. Faugeras F, Naccache L. Dissociating temporal attention from spatial attention and motor
358 response preparation: A high-density EEG study. *Neuroimage.* 2016;124(Pt A):947-957. doi:
359 S1053-8119(15)00873-3 [pii].
- 360 14. Kuppuswamy A, Clark EV, Sandhu KS, Rothwell JC, Ward NS. Post-stroke fatigue: A
361 problem of altered corticomotor control? *J Neurol Neurosurg Psychiatry.* 2015;86(8):902-
362 904. doi: 10.1136/jnnp-2015-310431 [doi].
- 363 15. Marinovic W, Brauer SG, Hayward KS, Carroll TJ, Riek S. Electric and acoustic
364 stimulation during movement preparation can facilitate movement execution in healthy
365 participants and stroke survivors. *Neurosci Lett.* 2016;618:134-138. doi: S0304-
366 3940(16)30140-9 [pii].
- 367 16. Taulu S, Kajola M, Simola J. Suppression of interference and artifacts by the signal space
368 separation method. *Brain Topogr.* 2004;16(4):269-275.

- 369 17. Tadel F, Baillet S, Mosher JC, Pantazis D, Leahy RM. Brainstorm: A user-friendly
370 application for MEG/EEG analysis. *Comput Intell Neurosci*. 2011;2011:879716.
- 371 18. Gross J, Baillet S, Barnes GR, et al. Good practice for conducting and reporting MEG
372 research. *Neuroimage*. 2013;65:349-363. doi: 10.1016/j.neuroimage.2012.10.001 [doi].
- 373 19. Holliday IE, Barnes GR, Hillebrand A, Singh KD. Accuracy and applications of group
374 MEG studies using cortical source locations estimated from participants' scalp surfaces. *Hum*
375 *Brain Mapp*. 2003;20(3):142-147. doi: 10.1002/hbm.10133 [doi].
- 376 20. Darvas F, Ermer JJ, Mosher JC, Leahy RM. Generic head models for atlas-based EEG
377 source analysis. *Hum Brain Mapp*. 2006;27(2):129-143. doi: 10.1002/hbm.20171 [doi].
- 378 21. Uusitalo MA, Ilmoniemi RJ. Signal-space projection method for separating MEG or EEG
379 into components. *Med Biol Eng Comput*. 1997;35(2):135-140.
- 380 22. Erdler M, Beisteiner R, Mayer D, et al. Supplementary motor area activation preceding
381 voluntary movement is detectable with a whole-scalp magnetoencephalography system.
382 *Neuroimage*. 2000;11(6 Pt 1):697-707. doi: 10.1006/nimg.2000.0579 [doi].
- 383 23. Nagamine T, Kajola M, Salmelin R, Shibasaki H, Hari R. Movement-related slow cortical
384 magnetic fields and changes of spontaneous MEG- and EEG-brain rhythms.
385 *Electroencephalogr Clin Neurophysiol*. 1996;99(3):274-286. doi: S0921884X96951541 [pii].
- 386 24. Huang MX, Harrington DL, Paulson KM, Weisend MP, Lee RR. Temporal dynamics of
387 ipsilateral and contralateral motor activity during voluntary finger movement. *Hum Brain*
388 *Mapp*. 2004;23(1):26-39. doi: 10.1002/hbm.20038 [doi].
- 389 25. Wijekumar S, Magnotta VA, Buss AT, et al. Response control networks are selectively
390 modulated by attention to rare events and memory load regardless of the need for inhibition.
391 *Neuroimage*. 2015;120:331-344. doi: 10.1016/j.neuroimage.2015.07.026 [doi].

- 392 26. Duque J, Labruna L, Cazares C, Ivry RB. Dissociating the influence of response selection
393 and task anticipation on corticospinal suppression during response preparation.
394 *Neuropsychologia*. 2014;65:287-296. doi: 10.1016/j.neuropsychologia.2014.08.006 [doi].
- 395 27. Woldag H, Waldmann G, Schubert M, et al. Cortical neuromagnetic fields evoked by
396 voluntary and passive hand movements in healthy adults. *J Clin Neurophysiol*.
397 2003;20(2):94-101.
- 398 28. Onishi H, Oyama M, Soma T, et al. Neuromagnetic activation of primary and secondary
399 somatosensory cortex following tactile-on and tactile-off stimulation. *Clin Neurophysiol*.
400 2010;121(4):588-593. doi: 10.1016/j.clinph.2009.12.022 [doi].
- 401 29. Wolpert DM, Flanagan JR. Motor prediction. *Curr Biol*. 2001;11(18):729. doi: S0960-
402 9822(01)00432-8 [pii].
- 403 30. Sun H, Blakely TM, Darvas F, et al. Sequential activation of premotor, primary
404 somatosensory and primary motor areas in humans during cued finger movements. *Clin*
405 *Neurophysiol*. 2015;126(11):2150-2161. doi: 10.1016/j.clinph.2015.01.005 [doi].
406

407 **Figure legends**

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

413

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

417

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

425

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

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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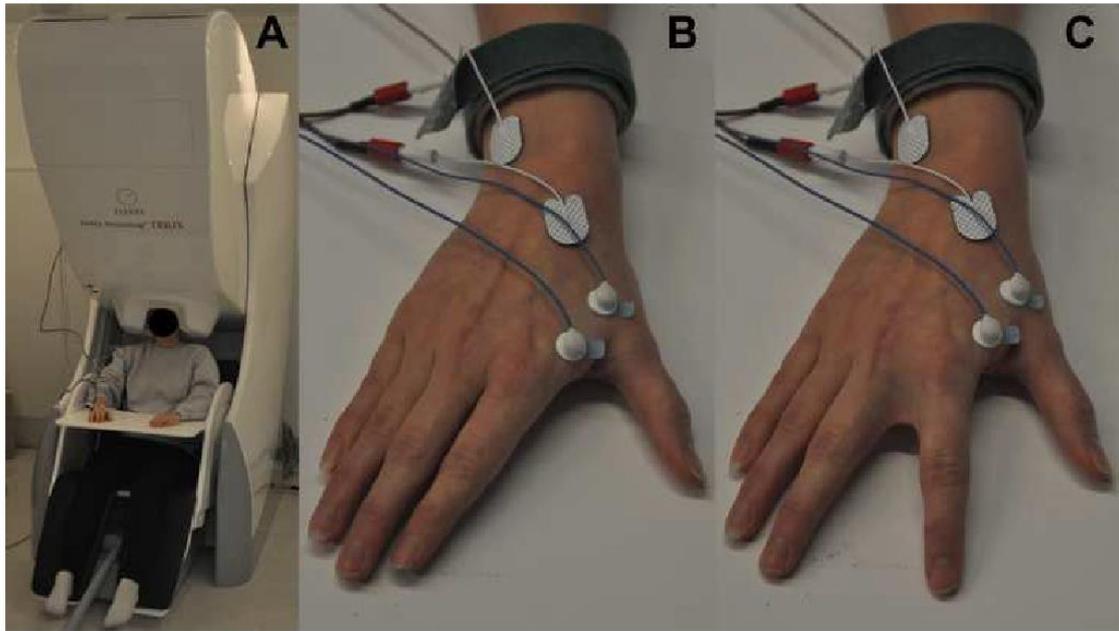
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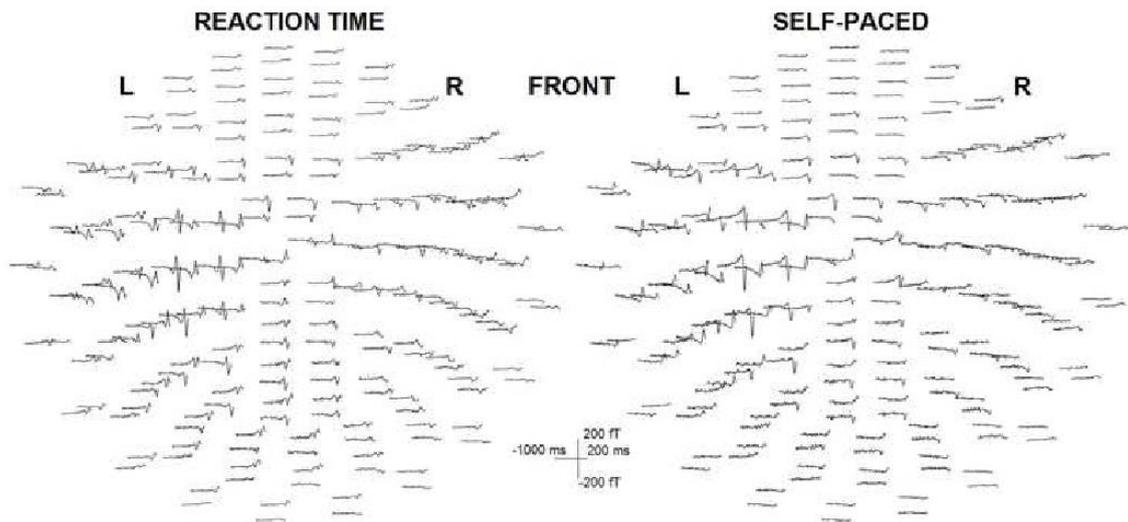
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Figure 1



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Figure 2

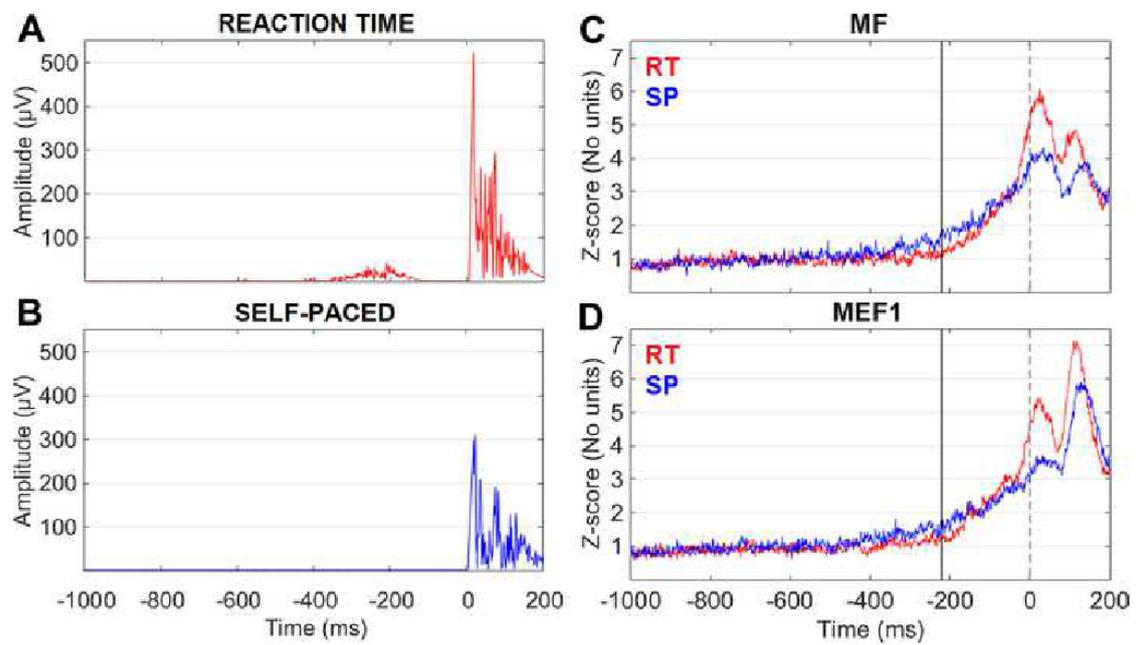


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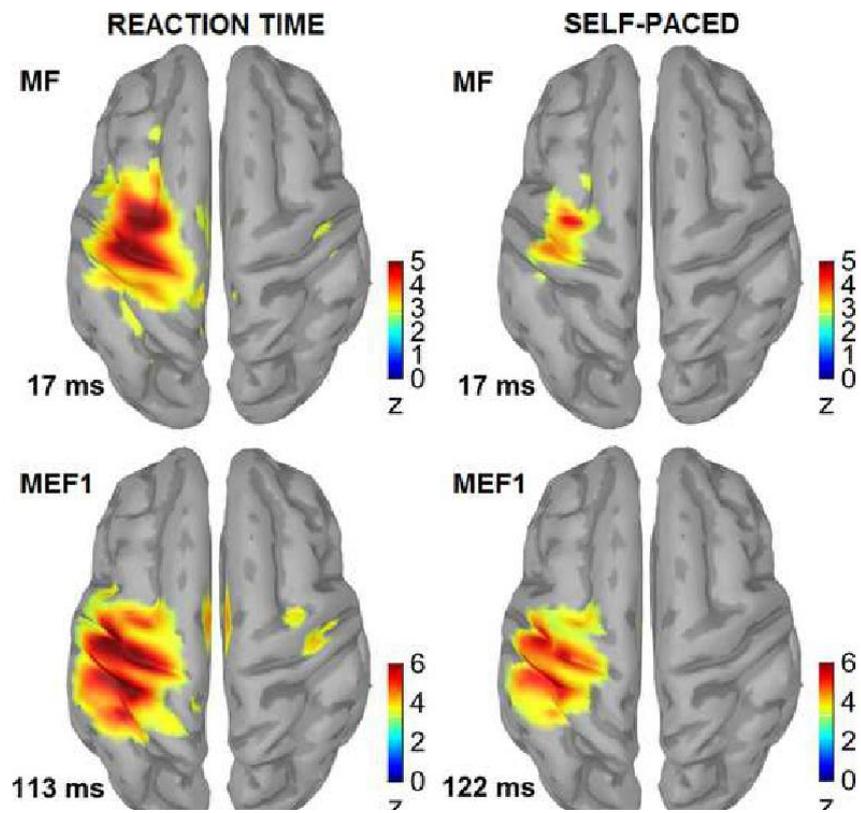
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Figure 3



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Figure 4



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