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# Motor inhibition during motor imagery: A MEG study with a quadriplegic patient

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## Abstract

The neurophysiological substrates underlying motor imagery are now well established. However, the neural processes of motor inhibition while mentally rehearsing an action are poorly understood. This concern has received limited experimental investigations leading to divergent conclusions. Whether motor command suppression is mediated by specific brain structures or by intracortical facilitation/inhibition is a matter of debate. Interestingly, although motor commands are inhibited during motor imagery (MI) in healthy participants, spinal cord injury may result in weakened motor inhibition. Using magnetoencephalography, we observed that mental and actual execution of a goal-directed pointing task elicited similar primary motor cortex activation in a C6-C7 quadriplegic patient, thus confirming the hypothesis of weakened motor inhibition during MI. In an age-matched healthy control participant, however, primary motor area activation during MI was significantly reduced compared to physical practice. Brain activation during actual movement resulted in enhanced recruitment of premotor areas in the patient. In the healthy participant, we found functional relationships between the primary motor area and peri-rolandic sites including the primary sensory area and the supplementary motor area during MI. This neural network was not activated when the quadriplegic patient performed MI. We assume that the primary sensory area and the supplementary motor area may be part of a functional network underlying motor inhibition during MI. These data provide insights into brain function changes due to neuroplasticity after spinal cord injury and evidence cortical substrates underlying weakened motor inhibition during MI after deafferentation and deafferentation.

**Keywords:** Motor imagery, Neuroplasticity, Spinal cord injury, Motor inhibition, Magnetoencephalography

## Background

Motor imagery (MI) is the mental rehearsal of a motor act without overt body movement. Previous neuroimaging studies provided evidence for functional equivalence between MI and physical practice (PP) of the same movement (Jeannerod, 1994). Accordingly, cortical and subcortical brain areas involved in motor planning, programming, and control of action execution are also recruited during MI, although the corresponding neural networks are hierarchically organized and not totally overlapping (Macuga and Frey, 2012). During MI, activations originating from pre-motor regions (Gerardin et al., 2000; Guillot et al., 2008) and primary sensorimotor areas (Porro et al., 1996) are commonly reported. Specifically, the involvement of the contralateral primary motor cortex (cM1) is observed (Gerardin et al., 2000; Porro et al., 2000; Sabbah et al., 1995), even in the absence of any electromyographic activity (Lang et al., 1996; Schnitzler et al., 1997). Although some studies failed to detect cM1 activity (Decety et al., 1994; Stephan et al., 1995), whereas others reported a fleeting involvement (Dechent et al., 2004), inconsistencies may account for differences in experimental designs (Lotze and Halsband, 2006).

Although often observed, the contribution of cM1 to MI remains unclear. Sharma et al. (2008) pointed out that cM1 could play a non-executive function, thus assisting in motor planning. This would explain cM1 activation without overt movement production. Other experiments showed that MI elicited subliminal muscle activity depending on the MI content (Guillot et al., 2007), hence suggesting that the motor command is effectively built during MI but partially inhibited. Indeed, the neural commands for muscle contractions might be blocked at some level of the motor system by active inhibitory mechanisms, thus preventing phasic muscle activity (Jeannerod, 1994).

The neural processes involved in the suppression of motor output during MI are poorly understood (Guillot et al., 2012). Many cortical regions including the supplementary motor area (SMA), the parietal regions, and the cerebellum have been supposed to play a critical role (for a review, see Guillot et al., 2010). Based on fMRI data, Kasess et al. (2008) revealed a strong suppressive influence of the SMA over cM1 during MI. Solodkin et al. (2004) recorded a suppressive influence of both the contralateral primary sensory area (cS1) and SMA on cM1, thus supporting the fact that the inhibition of cM1 during imagined movements might result from a complex functional network. Interestingly, Schwoebel et al. (2002) observed that a patient with hemiparesis overtly performed the movement concurrently to its mental representation, hence revealing that the inhibitory processes of the motor command that should be active during MI were probably suppressed. The authors suggested that such motor control impairment originated from a disturbance of the functional loop between cS1 and cM1 due to the brain lesion. To which extent Central Nervous System (CNS) disorders enable better understanding of brain processes underlying motor inhibition during MI is therefore a matter of interest.

There is now ample evidence that central reorganizations occur within cortical motor regions after Spinal Cord Injury (SCI—Green et al., 1998; Jurkiewicz et al., 2007; Kokotilo et al., 2009; Lotze and Halsband, 2006; Lotze et al., 1999a). Neuroplasticity following SCI involves consistent reshaping of sensorimotor maps (Bruehlmeier et al., 1998; Kokotilo et al., 2009). Neural reorganizations involve axonal sprouting and reduced intracortical inhibition, thus promoting increased functional efficiency within the spared neural pathways (Dunlop, 2008; Topka et al., 1991). Those plastic changes are reflected during MI. Brain activation patterns mediating MI among SCI participants differ from those observed in healthy controls. Specifically, Cramer et al. (2005) reported impaired

modulation of cerebral activity between MI and attempted movement with the paralyzed limb, as well as abnormal involvement of sensory areas during MI in SCI patients. Furthermore, brain activation patterns observed during MI in SCI participants closely match those recorded during PP in healthy controls (?). Plastic changes in primary and secondary motor areas during MI following deafferentation might therefore reflect weakened inhibitory processes of the motor commands, probably because these are no longer required (Lacourse et al., 1999). However, this proposal remains an inference requiring further experimental investigation, specifically with the currently growing interest in the possibilities of physical rehabilitation including MI interventions.

The present study was designed to investigate the effect of cerebral neuroplasticity following deafferentation and deafferentation elicited by SCI on the central processes underlying PP and MI. We recorded the central activity in a C6-C7 quadriplegic patient and a healthy age-matched control participant (CP) while executing and imagining a goal-directed pointing task. Using magnetoencephalography (MEG), we gained access to the time-course of cortical activity in cM1, and examined whether cM1 activation might differ between the two participants. We hypothesized that the SCI participant might exhibit reduced inhibition of this area after deafferentation. Altogether, we aimed to provide new highlights regarding the neural mechanisms of motor inhibition during MI, and to further delineate changes in brain motor functions after SCI.

## Methods

### Participants

Our 23-year-old patient sustained a traumatic complete SCI due to a C6 burst fracture resulting in quadriplegia. The neurological check-up showed general infra-lesional sensorimotor deficit, as clinically assessed by the American Spinal Injury Association score on the Impairment Scale (ASIA=A, Maynard et al. (1997)). Muscular testing showed normal force of shoulder muscles and elbow flexors. Elbow extensors exhibited residual force reaching the second level on a 5-level scale. There was a general paralysis of the flexor carpi radialis and hand muscles, although the patient had moderate spasticity without joint movement restriction. He was able to grasp objects within the haptic space, using tenodesis, i.e., a modified type of grasp Mateo et al. (2012). He was not comatose and did not have any brain injury. The present experiment took place 1 year post injury. The SCI participant was matched according to gender, age, and handedness with a healthy control participant who did not have any neurological or psychological disorder. Both participants were right-handed.

Participants' consent to participate in the present experiment was obtained according to the Declaration of Helsinki. The local ethical committee of the CERMEP ("in vivo imaging" center Lyon, France) approved the present experiment.

### MI assessment

We assessed the individual MI ability of both participants before MEG recordings with a validated battery of behavioral measures. Participants first completed the Kinesthetic and Visual Imagery Questionnaire (KVIQ), specifically designed to evaluate MI capacities in patients suffering from motor deficits Malouin et al. (2007). They had to rate the level of visual and kinesthetic perception associated with MI of actions after actual execution

or after observation of someone else whenever actually performing this movement was impossible.

Second, the ability to achieve temporal congruence between MI and PP was assessed as a reliable marker of MI accuracy Collet et al. (2011); Guillot et al. (2011); Malouin et al. (2008). Both participants therefore underwent mental chronometry by actually performing and mentally rehearsing a pointing task with their dominant arm, from a standardized position to a target located 30 cm ahead (10 trials). They were instructed to concurrently combine first-person and kinesthetic MI. Trials were separated from 20s-rest periods to avoid possible deleterious effects of central fatigue due to mental work Guillot et al. (2004).

Finally, as autonomic nervous system responses represent non-conscious physiological mechanisms of mental processes Collet and Guillot (2010), we recorded electrodermal activity during the mental chronometry task to assess MI quality Collet et al. (2011). As electrodermal response duration should match that of the movement mentally rehearsed, we compared the Ohmic Perturbation Duration (OPD) during MI with that recorded during PP. OPD was measured between the sudden drop elicited by MI and response end, i.e., when the slope showed no fluctuation just before recovering the basal level, and resembled that observed before stimulation Vernet-Maury et al. (1995).

## MEG acquisition

During MEG recordings, participants were asked to physically perform the same goal-directed pointing task used for MI ability assessment (PP, 10 trials per block). Owing to forearm muscles impairment of the quadriplegic patient, participants were requested to linearly slide their forearm toward the target rather than point to it with a parabolic trajectory per se. This experimental design prevented a different muscular control of the movement between participants, who were also requested to mentally imagine the same movement (MI, 10 trials per block). They were finally instructed to remain motionless during a control condition (CONTROL, 10 epochs per block). An auditory soundtrack was broadcasted during MEG recordings to provide instructions regarding each experimental condition. An auditory stimulus triggered the onset of each trial every 10s. Movement duration was recorded using an optical fiber switch system, spotting movement start and end.

Experimental conditions (PP, MI, and CONTROL) were randomly performed within a block-design three times each, for a total of 30 trials in each condition (i.e., 3 blocks of 10 trials per condition). This limited number of trials was expected to preserve the quadriplegic patient from the effects of muscle and mental fatigue. After completion of the MEG design, we used oral debriefs and self-report ratings to control adherence to experimental instructions and assess MI vividness. Participants were requested to rate the vividness of MI as perceived during the MEG recording. We used the same 5-level Likert Scale that was used during KVIQ assessments. More specifically, both participants provided a general rating based on vividness, clarity, and intensity of MI.

MEG recordings were performed using a whole-head CTF<sup>®</sup>-MEG system (CERMEP), with 275 radial gradiometers over the scalp and 33 reference channels for ambient field correction. Signals were digitalized at a sampling rate of 600 Hz and recorded continuously using a low-pass filter (0–150 Hz). Participant’s head position was continually recorded using head coils placed on the nasion and pre-auricular points prior to scanning. Eye movements were recorded for artifact control. Structural MRI pictures were also recorded

for co-registration with MEG data in each participant. Brain MRI was performed using a 1.5 T Siemens Magnetom scanner (CERMEP—Siemens AG, Erlangen, Germany) for the acquisition of 3D anatomical T1-weighted (TR: 9.7 ms, TE: 4 ms) pictures covering the whole brain volume with  $1\text{ mm}^3$  cubic voxels.

## MEG data processing

We used Single Aperture Magnetometry (SAM) as the spatial filter to map brain Event-Related Desynchronization/Synchronization (respectively, ERD and ERS, Robinson (2004)). SAM is a minimum variance beamformer emphasizing the activity that originates from a defined region while blocking outside signals. SAM beamforming was performed on MEG beta (13–35 Hz) signals, as the beta band is a frequency range reflecting specific oscillatory neural activity associated with human voluntary movements Pfurtscheller and Lopes da Silva (1999). The beta band was further evidenced to reflect brain activity elicited during MI in previous MEG studies, based on single-trial SAM analysis Battapady et al. (2009a,b).

In a recent study, Burianova et al. (2013) observed that the strongest ERD occurred within the beta (13–30 Hz) and low gamma (30–40 Hz) frequency bands during MI and PP, and originated from primary sensorimotor and premotor cortices (using beamforming for source localization). The 13–35 Hz beta frequency domain was thus of specific interest in the present experiment. All calculations were also performed in the mu frequency band (8–12 Hz), since this frequency domain was shown to be relevant to reflect sensorimotor and premotor activity during PP and MI Babiloni et al. (1999); McFarland et al. (2000).

SAM displays the spatial distribution of event-related changes in cortical rhythm within a frequency range using t-paired Jackknife statistics between active and control states as defined by time-windows analysis. In the present study, control and active states were established immediately before and after stimulus onset to highlight the regions displaying task-related ERD/ERS patterns of activity. Time-window duration corresponded to the average duration of the goal-directed movement recorded during PP. Depending on the ability of each participant to achieve temporal congruence between overt and covert actions, time-windows for SAM analysis would remain unchanged between MI and PP, otherwise increased or shortened. This methodological design aimed to compensate for self-triggering MI, due to its concealed nature.

SAM virtual sensors were computed in specific sites of interest (SOI). SAM virtual sensors provide an estimate of the MEG time-course signals originating from a defined region of the 3D brain grid used for source localization, by applying their beamformer coefficients to the MEG gradiometers signals. Power gain/loss within a frequency range in a defined brain area is referred to a task-specific neural activity modulation, hence reflecting inhibition (ERS) or activation (ERD, Neuper and Pfurtscheller (2010)). Time-course beta power in pre-determined SOI was obtained under each experimental condition by applying a Root Mean Square (RMS) power filter (Hanning window= .2s) to SAM sensor signals. We averaged the power elicited during active and control states, and obtained the difference ( $\Delta P$ ).  $\Delta P$  was investigated in all subsequent analyses as a MEG-dependent variable quantifying ERD/ERS within SOI.

We first explored cM1 task-related activity in each participant, using the precentral gyrus as an individual anatomical mask for accurate localization Lotze et al. (1999b); Lotze and Zentgraf (2010). Then, we selected additional SOI in brain regions playing a potential role in the inhibition of cM1 during MI, i.e., cS1 and SMA Alkadhi et al. (2005); Kasess

et al. (2008); Schwoebel et al. (2002); Solodkin et al. (2004). Anatomically, contralateral and ipsilateral SMAs are reciprocally connected, and both project to the contralateral and ipsilateral M1 Muakkassa and Strick (1979); Pandya and Vignolo (1971). In the context of the present experiment, we thus considered a priori the two SMAs as potential SOI in the study of cM1 inhibition during MI.

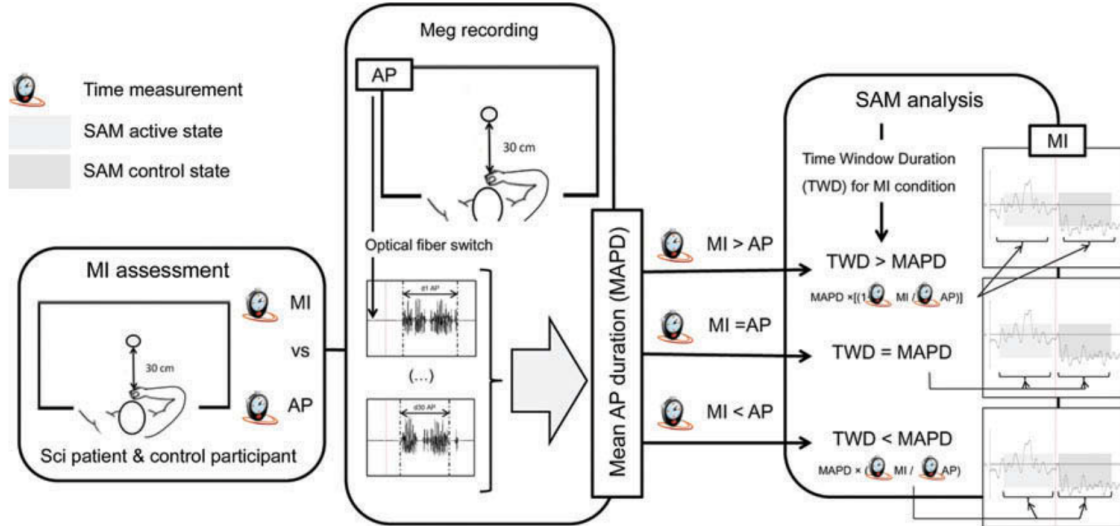


Figure 1: Mental chronometry and MEG data processing. SAM time-windows were determined immediately before and after stimulus onset (auditory soundtrack). Time-window amplitudes for MI condition were obtained from PP duration during the MEG task.

## Statistical procedure

### General considerations

Type 1 error rate was set at 5% for statistical significance. We used the R freeware and CTF(r)-Software for all data processing and statistical computing. Owing to auto-correlation bias in a two-participant design, we did not perform inter-subject statistical comparisons Crawford et al. (2010). Accordingly, we based our approach on intra-subject analysis and used overall corrected significance thresholds, thus enabling descriptive comparisons. We used a sequential method for Bonferroni corrections Holm (1979) to control the false discovery rate.

### MI ability

We used paired Wilcoxon tests with Bonferroni corrections to assess differences in KVIQ scores (discrete variables) between participants. t-tests with Bonferroni corrections were used for intra-subject MI ability assessments with respect to the ability to achieve temporal equivalence between MI and PP durations and electrodermal response duration (OPD).

## MEG data

For visualization of the spatial distribution of SAM ERD or ERS patterns during MI and PP, the uncorrected maximal SAM t-value (since all subsequent analyses were performed on the time course of SAM sensors beta power) elicited during the CONTROL condition within our SOI was used as threshold for PP and MI SAM activation maps.

$\Delta P$  was obtained in each SOI, for each participant under each experimental condition. To assess ERD/ERS patterns, we used paired t-tests with Bonferroni corrections to compare mean beta power values during active (pre-trigger) and control (post-trigger) states. In both participants, we tested the effect of condition (i.e., CONTROL, MI, and PP) on  $\Delta P$  by implementing a MANOVA model [ $\Delta P(\text{cS1}; \text{cM1}; \text{SMA}) \sim \text{condition}$ ]. We also used t-tests with Bonferroni corrections for post-hoc investigations.

We computed linear models (i.e.,  $\Delta P_{\text{cM1}} \sim \Delta P_{\text{cS1}} \times \Delta P_{\text{SMA}}$ ) in each participant to assess whether cS1 and SMA  $\Delta P$  significantly explained (i.e., predicted) cM1 desynchronization during MI and PP.

## Results

### Motor imagery assessment

Mean KVIQ scores were 3.5 and 4.0 in the CP and SCI participants, respectively. KVIQ visual and kinesthetic subscores indicated that participants perceived sensory information close to “intense” during kinesthetic MI, and formed “moderately clear” to “very clear” images during visual MI (Table 1). There was no significant difference in KVIQ general or subscores between participants, albeit the SCI participant tended to score higher on the visual MI subscore ( $W = 5.0, p = .07$ ; Table 1).

Mean KVIQ score	SCI	CP	Paired Wilcoxon’s test
General	4.0 $\pm$ .3	3.5 $\pm$ .2	W = 33.5, p = .23
Visual	3.9 $\pm$ .3	2.8 $\pm$ .2	W = 5.0, p = .07
Kinaesthetic	4.0 $\pm$ .3	4.2 $\pm$ .2	W = 13.5, p = .59
SCI	PP	MI	Paired t-value
Mean times	2.34 $\pm$ .11	2.43 $\pm$ .31	t = -0.89, p = .39
OPD	3.64 $\pm$ .62	3.74 $\pm$ .54	t = -0.77, p = .45
CP	PP	MI	Paired t-value
Mean times	2.19 $\pm$ .25	2.26 $\pm$ .22	t = 0.80, p = .43
OPD	3.21 $\pm$ .76	3.25 $\pm$ .13	t = 0.16, p = .88

Table 1: Mean KVIQ scores and paired comparisons between SCI and CP participants.

There was no difference regarding durations and OPDs between executed and imagined actions in both participants (Table 1).

### MEG patterns

Both participants reported no difficulty complying with experimental instructions, and both reported a high score (4.0 out of 5.0) for MI vividness after completion of the MEG

design. Both participants were similarly able to respect the temporal congruence between MI and PP (Table 1). Based on mean PP duration as recorded by the optical fiber system (Figure 1), time-windows from 2.2 to 2.6s were defined to set up SAM analysis.

The CONTROL condition did not elicit significant ERD or ERS in both participants' sensorimotor regions. SAM maximal t-values within SOI during the CONTROL condition were 2.0 and 2.1 in the SCI and CP participants, respectively. SAM maps in the beta band revealed specific ERD patterns in both participants. PP resulted in contralateral brain activations including primary sensorimotor and premotor regions. Ipsilateral M1 and S1 ERD were also observed during PP, but with lower SAM t-ratios than those recorded in the contralateral hemisphere. During MI, ERD occurred in the left contralateral hemisphere only (Figure 2). Likewise, SAM activation maps yielded contralateral involvement of the SMA (cSMA). Importantly, the pattern of hemispheric recruitment was similar between the two participants. ERD patterns elicited during PP and MI largely overlapped within the left contralateral hemisphere (Figure 2). We thus focused on contralateral activations in subsequent analyses.

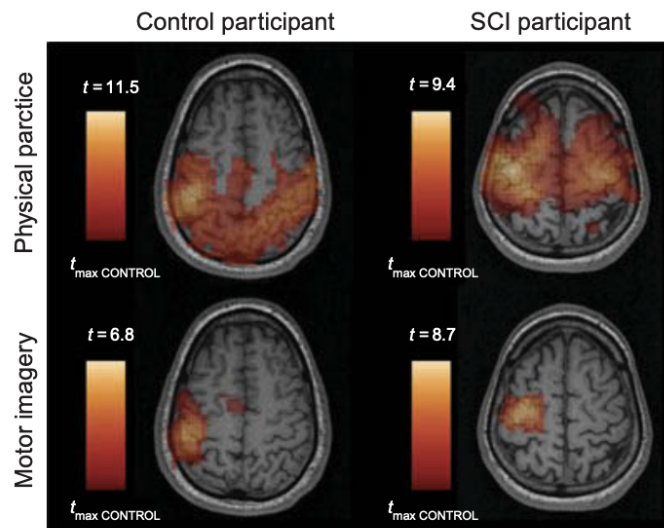


Figure 2: Task-dependent beta ERD in the SCI and control participants. We observed primary contralateral involvement of the SMA in both participants. We recorded bilateral sensorimotor activations during PP, with stronger activations in the contralateral hemisphere. Conversely, only contralateral ERD was observed during MI.

SAM maps within the mu frequency band provided congruent results with those obtained in the beta band regarding spatial distribution of ERD and ERS, although we observed lower SAM t-values during both PP and MI (SAM t-values reduced by 30–58% and 41–48%, respectively). Conversely, SAM t-values in the mu band during the CONTROL condition were similar to those observed in the beta band (i.e., reduced by only 5%) for both participants.

<b>ROI</b>		<b>x</b>	<b>y</b>	<b>z</b>
	cM1	0.80	4.40	8.15
CP	cS1	0.00	4.40	9.20
	cSMA	1.40	0.50	9.20
	cM1	1.20	3.60	9.60
SCI participant	cS1	0.10	4.75	8.70
	cSMA	1.25	0.65	7.40

Table 2: fMRI coordinates of SAM virtual sensors.

### **Power analysis in cM1, cS1, and cSMA**

Beta  $\Delta P$  was obtained in cM1, cS1, and cSMA (for coordinates, see Table 2). Significant cM1 and cS1 ERD were observed during PP and MI in both participants (Table 3, Figures 3 and 4). However, no significant ERD emerged from cSMA during MI in the SCI participant ( $t = -0.04$ ,  $p = .96$ ; Table 3 and Figure 4).

MANOVA yielded the main effect of condition on beta  $\Delta P$  in both the CP ( $F(6, 172) = 5.59$ ,  $p < .001$ ) and SCI participants ( $F(6, 172) = 10.01$ ,  $p < .001$ ). MANOVA also revealed the main effect of condition on mu  $\Delta P$ , in both the CP ( $F(6, 172) = 2.72$ ,  $p = .01$ ) and SCI participants ( $F(6, 172) = 2.22$ ,  $p = .04$ ).

Control participant			
Task	SAM t-value	$\Delta P$ (A.m. $10^{-10}$ /T)	t-test P ( $\mu = 0$ )
Physical practice	8.0	$1.21 \pm .18$	$t = 6.45, p < .001$
Motor imagery	3.0	$.39 \pm .18$	$t = 2.10, p = .04$
Control	-1.0	$-.30 \pm .16$	$t = -1.79, NS$
Physical practice	9.9	$1.97 \pm .18$	$t = 9.55, p < .001$
Motor imagery	6.2	$.97 \pm .26$	$t = 3.61, p = .001$
Control	1.0	$-.04 \pm .63$	$t = -.06, NS$
Physical practice	4.1	$.63 \pm .18$	$t = 3.54, p = .001$
Motor imagery	2.5	$.81 \pm .27$	$t = 2.92, p = .006$
Control	-0.8	$-.17 \pm .39$	$t = .45, NS$
SCI participant			
Task	SAM t-value	$\Delta P$ (A.m. $10^{-10}$ /T)	t-test P ( $\mu = 0$ )
Physical practice	9.5	$1.35 \pm .14$	$t = 9.73, p < .001$
Motor imagery	8.4	$1.07 \pm .12$	$t = 8.45, p < .001$
Control	-0.5	$-.31 \pm .15$	$t = -2.51, NS$
Physical practice	7.9	$1.08 \pm .11$	$t = 9.38, p < .001$
Motor imagery	6.0	$.84 \pm .13$	$t = 6.35, p < .001$
Control	0.4	$.11 \pm .35$	$t = .32, NS$
Physical practice	4.6	$1.12 \pm .22$	$t = 5.16, p < .001$
Motor imagery	0.5	$.01 \pm .30$	$t = .04, NS$
Control	0.5	$.17 \pm .36$	$t = .45, NS$

Table 3: SAM t-values, SAM virtual sensor beta power analysis in cM1, cS1, and cSMA. More specifically, mean desynchronization amplitude between active and control states is provided, and compared with the reference value 0 using univariate t-tests. No ERD was observed in cM1 during MI in the CP when corrections were applied. During the control condition, we recorded a marginally significant cM1 ERS in both participants. During MI, cSMA ERD reached significance in the CP only.

Investigation of  $\Delta P$  values in cM1 revealed a significant effect of the experimental conditions (PP, MI, and CONTROL), in both CP ( $F(2, 87) = 16.03, MSE = 15.35, p < .001$ ) and SCI participants ( $F(2, 87) = 34.37, MSE = 19.76, p < .001$ ; Table 3, Figure 3). Critically, we observed different patterns regarding the time course of cM1 power beta rhythm across participants (Figure 3). The difference of cM1 desynchronization amplitude between MI and PP did not reach significance in the SCI participant ( $t = 1.49, p = .14$ , mean values being  $1.08 \text{ A.m.}10^{-10}/\text{T}$  and  $1.35 \text{ A.m.}10^{-10}/\text{T}$ , respectively), whereas a greater neural desynchronization was recorded in cM1 during PP (mean =  $1.21 \text{ A.m.}10^{-10}/\text{T}$ ) compared to MI (mean =  $0.39 \text{ A.m.}10^{-10}/\text{T}$ ) in the CP ( $t = 3.13, p < .01$ ).

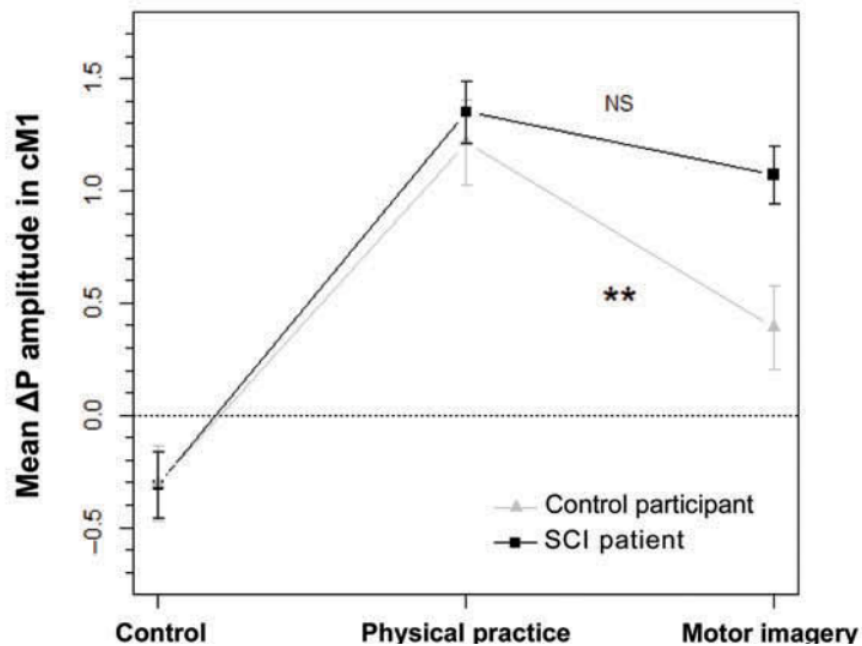
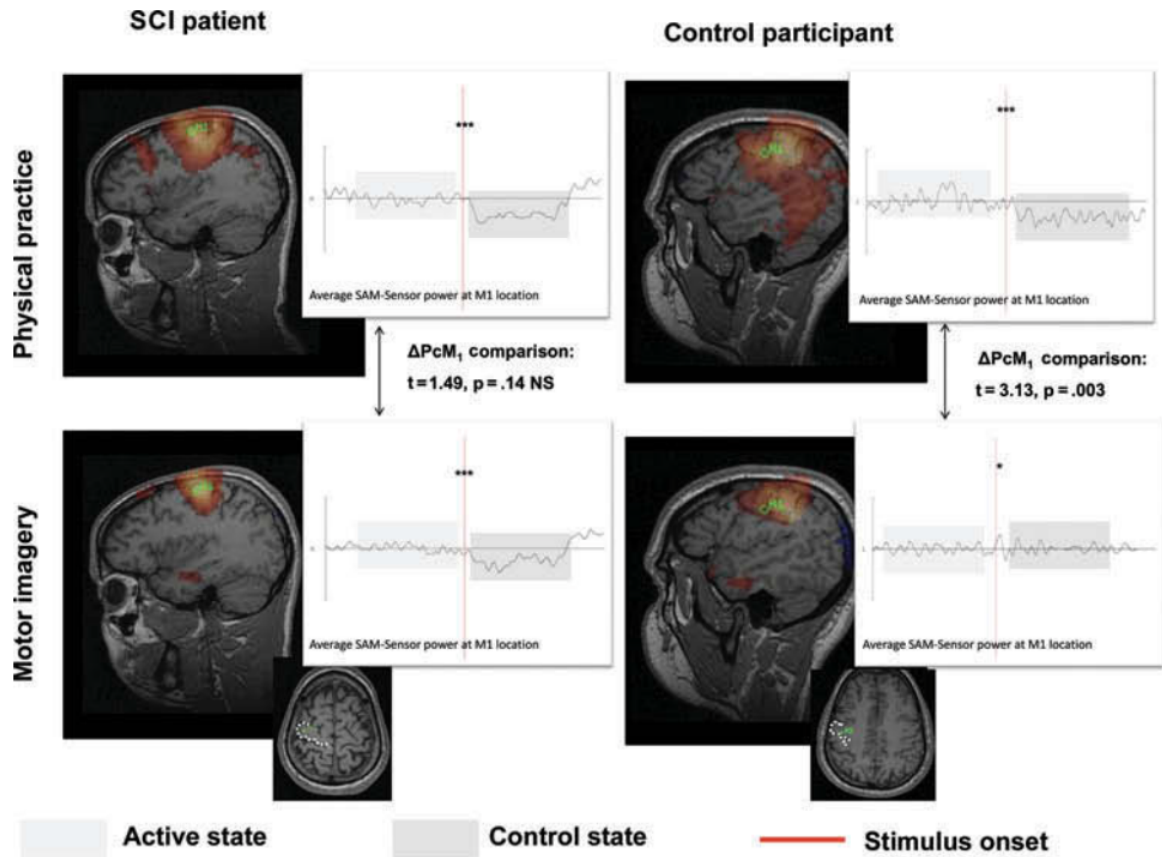


Figure 3: Mean beta  $\Delta P$  values in cM1 with standard errors. Compared to PP, MI resulted in reduced ERD amplitude in the control participant, whereas the SCI participant preserved a similar cM1 ERD during both PP and MI.

A similar pattern of results was observed in the mu band. The whole effect of experimental conditions on cM1 reached significance in both CP ( $F(2, 87) = 3.02, MSE = 6.34,$

$p = .05$ ) and SCI participants ( $F(2, 87) = 3.01$ ,  $MSE = 1.73$ ,  $p = .05$ ). Mu desynchronization tended to be stronger during PP than during MI in the CP ( $t = 2.00$ ,  $p = .05$ ); however, the difference was non-significant when corrections were applied. Conversely, a similar cM1  $\Delta P$  was observed in the SCI participant ( $t = 0.70$ ,  $p = .48$ , NS).

In cS1, MANOVA carried on beta  $\Delta P$  yielded a significant effect of experimental conditions in both CP ( $F(2, 87) = 8.19$ ,  $MSE = 35.83$ ,  $p < .001$ ) and SCI participants ( $F(2, 87) = 5.58$ ,  $MSE = 7.30$ ,  $p < .01$ ; Figure 4). In cSMA, MANOVA yielded a significant effect of experimental condition in both the SCI ( $F(2, 87) = 4.10$ ,  $MSE = 11.15$ ,  $p = .02$ ) and CP ( $F(2, 87) = 3.31$ ,  $MSE = 7.51$ ,  $p = .04$ ) participants. Post-hoc tests revealed specific ERD patterns in cS1 and cSMA (Figure 4). In CP, cS1 elicited stronger ERD during PP than during MI ( $t = 2.91$ ,  $p < .01$ ), whereas we observed similar ERD in the SCI participant during both tasks ( $t = 1.31$ ,  $p = .20$ , NS). In the cSMA, MI and PP elicited similar ERD in CP ( $t = -0.52$ ,  $p = .60$ , NS), whereas a stronger ERD was recorded during PP in the SCI participant ( $t = 2.92$ ,  $p < .01$ ).

In the mu band, MANOVA yielded the effect of condition on cS1  $\Delta P$  in the CP ( $F(2, 87) = 3.22$ ,  $p = .04$ ) but not in the SCI participant ( $F(2, 87) = 1.12$ ,  $MSE = 5.82$ ). The effect of experimental conditions on cSMA  $\Delta P$  reached significance in the SCI ( $F(2, 87) = 4.02$ ,  $MSE = 3.07$ ,  $p = .02$ ) and CP ( $F(2, 87) = 3.88$ ,  $MSE = .17$ ,  $p = .02$ ) participants. Mu cS1 desynchronization tended to be stronger during PP than during MI in the CP ( $t = 2.16$ ,  $p = .04$ ), albeit the difference was not statistically significant when corrections were applied. cSMA desynchronization also tended to be stronger during MI than during PP in the SCI participant ( $t = 2.31$ ,  $p = .03$ ), yet the difference failed to reach significance when corrections were applied.

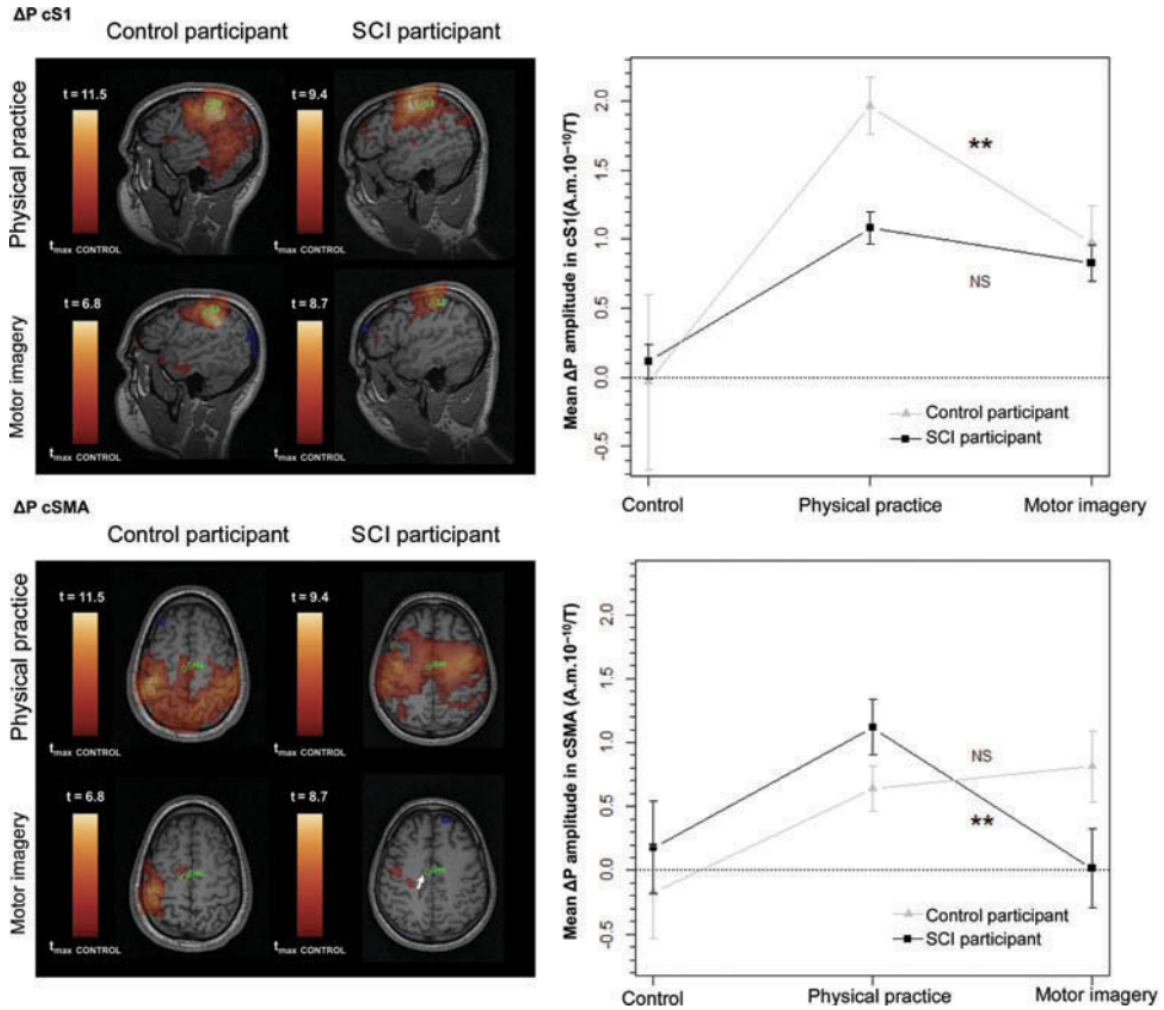


Figure 4: Mean beta  $\Delta P$  values with standard errors in cS1 and SMA for both participants during MI and PP.

### Analysis of SMA and cS1 interactions on cM1 neural activity

The beta " $\Delta P_{cM1} \sim \Delta P_{cS1} \times \Delta P_{cSMA}$ " model in PP presented high effect sizes in both participants ( $\eta^2 = .55$  and  $\eta^2 = .62$  in the CP and SCI participants, respectively). During MI, the " $\Delta P_{cM1} \sim \Delta P_{cS1} \times \Delta P_{cSMA}$ " model sustained an effect size of  $\eta^2 = .56$  in the CP, along with a significant  $\Delta P_{cS1} \times \Delta P_{cSMA}$  interaction ( $F(1, 28) = 7.73$ ,  $MSE = 3.52$ ,  $p = .01$ ). Conversely, we observed a weak effect size ( $\eta^2 = .08$ ) in the SCI participant, associated with a non-significant  $\Delta P_{cS1} \times \Delta P_{cSMA}$  interaction ( $F(1, 28) = 0.03$ ,  $MSE = 0.01$ ). The effect of each variable and their interaction was extracted from ANOVA for each model (Figure 5).

As in the beta band, the mu " $\Delta P_{cM1} \sim \Delta P_{cS1} \times \Delta P_{cSMA}$ " model presented high effect sizes during PP in both CP and SCI participants ( $\eta^2 = .25$  and  $\eta^2 = .56$ , respectively). During MI,  $\Delta P_{cS1}$  and  $\Delta P_{cSMA}$  significantly explained mu  $\Delta P_{cM1}$  in the CP ( $\eta^2 = .94$ ,  $p < .001$ ), but not in the SCI participant ( $\eta^2 = .09$ ).

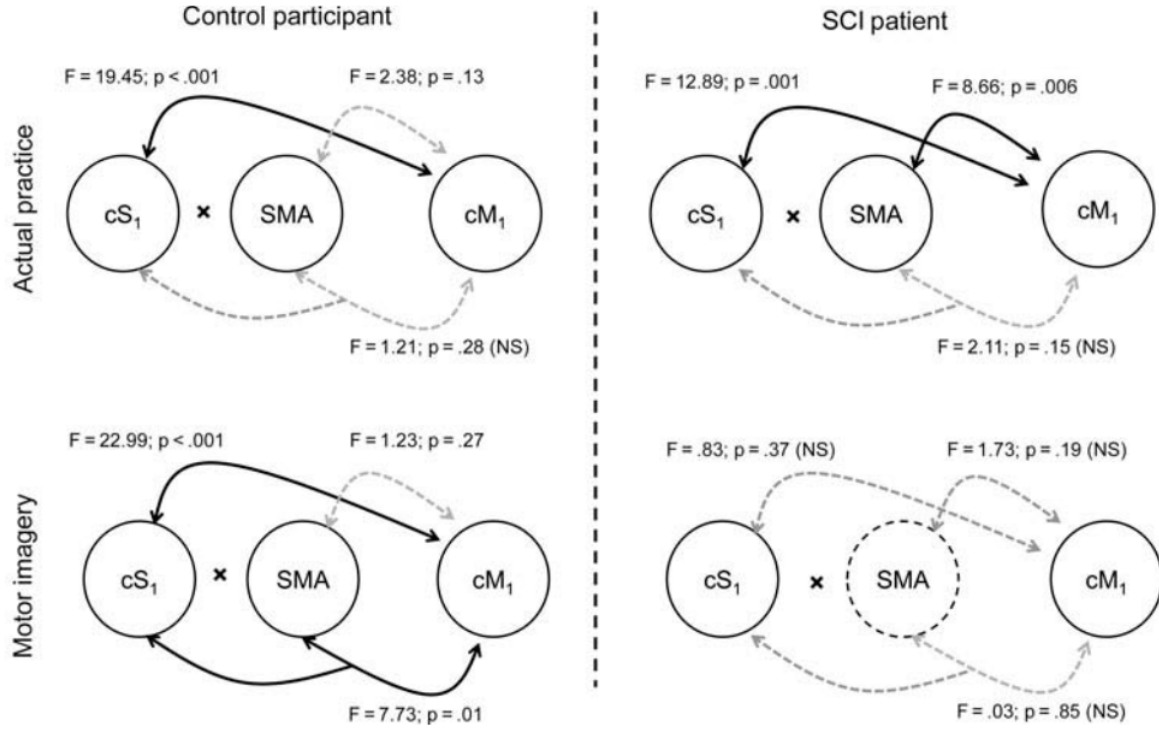


Figure 5: Representation of beta  $\Delta P_{cM1} \sim \Delta P_{cS1} \times \Delta P_{cSMA}$  models in both participants during MI and PP based on ANOVAs. During MI, the cSMA is circled with a dotted line in the SCI patient, since no significant ERD was recorded in this area under this condition.

## DISCUSSION

### Summary of main findings

The purpose of this study was to explore cM1 activity during PP and MI in a patient with SCI and a healthy age-matched control participant exhibiting comparable MI abilities. MI and PP were expected to elicit a different contribution of cM1 to task processing between participants. The main findings may be summarized as follows: (i) cM1 activation was reduced during MI compared to PP in the CP but not in the SCI participant, (ii) cM1 activation during MI was related to cS1 and cSMA activation in the CP, but not in the SCI participant.

### MI ability in the two participants

Testing MI accuracy requires the simultaneous contribution of neurophysiological and psychological methods (Guillot and Collet, 2005). Here, we used the KVIQ as a psychological measure (Malouin et al., 2007), mental chronometry as a valuable behavioral outcome, and electrodermal response as a neurophysiological-dependent variable reflecting ANS activity during MI (Collet et al., 2011). Both participants exhibited similar KVIQ general scores, hence suggesting comparable ability to imagine motor acts with good MI vividness. Since participants were requested to concurrently use internal visual imagery and

kinesthetic imagery during the MI task, general KVIQ scores (i.e., including all visual and kinesthetic items) were reliable to determine participants' compatibility in the perspective of MEG assessments. Noteworthy, the SCI participant showed a non-significant trend to higher KVIQ visual scores than the CP. The SCI participant could not actually perform seven out of the ten KVIQ items due to paralysis below the level of injury. One can thus hypothesize that increased dependence on visual information in the SCI participant to maintain/update motor representations after deafferentation accounts for this result.

Regarding the goal-directed pointing task, MI ability assessments revealed highly similar MI capacity between the two participants, who performed MI with a close temporal congruence with PP. Both had also similar OPDs during MI, which closely resembled those recorded during PP, hence supporting a high level of MI ability. Finally, they both reported high vividness scores after MEG task completion, requiring to concurrently perform visual and kinesthetic MI, which resembled KVIQ global scores. Taken together, these data support high and comparable MI ability among participants, hence allowing comparison of cerebral activity.

## **MEG activation patterns during MI and PP**

SAM analysis revealed ERD in the brain motor network during MI and PP. Indeed, desynchronization in beta oscillations occurs during motor planning, execution, and mental representation of movements within cortical motor areas (Neuper and Pfurtscheller, 2010). Comparable activations during PP and MI confirm that mental representation and execution of actions share common neural substrates (Jeannerod, 1994). These also support that the spatial distribution of the neural networks mediating MI and PP largely overlap in SCI participants and healthy controls (Alkadhi et al., 2005; Cramer et al., 2005). In both participants, bilateral primary sensorimotor recruitment was observed during PP. Conversely, primary sensorimotor activations during MI were lateralized in the contralateral hemisphere, and involved cM1, cS1, and cSMA. These data are consistent with previous studies related to lateralization of activations during MI (Ehrsson et al., 2003; Michelon et al., 2006). Contralateral involvement of the SMA is in keeping with findings supporting that, despite its critical role during control and coordination of bimanual movements, more lateralized recruitment of the SMA may occur during processing of unilateral movements (Beaulé et al., 2012) and goal-directed pointing actions (Winstein et al., 1997).

## **cM1 activation during PP and MI**

According to Lotze and Halsband (2006), cM1 activation during MI is inversely related to the duration and complexity of the task mentally performed. Alkadhi et al. (2005) added that cM1 activity was proportionally related to imagery vividness after SCI. In the present experiment, participants imagined a simple and brief pointing sequence for which they reported high MI vividness. As shown by previous studies, we observed individual differences regarding the intensity of cM1 activation during MI. Accordingly, time-course analysis of ERD revealed stronger cM1 activity during PP than during MI in the healthy participant, whereas executed and imagined movement yielded similar activity during both conditions in the SCI participant.

Transcranial magnetic stimulation and neuroimaging studies provided evidence that cortical reorganizations can occur after deafferentation and deafferentation (Jurkiewicz

et al., 2007; Levy et al., 1990). Increased cortical excitability and reduced intra-cortical inhibition were reported because of deafferentation (Dunlop, 2008; Roy et al., 2011; Saturno et al., 2008). In healthy participants, reduced primary sensorimotor activation during MI, as compared to PP, has been referred to as accounting for inhibitory processes required during MI to prevent any peripheral activity of muscles (Porro et al., 1996). The present results concur with findings by Lacourse et al. (1999), suggesting weakened motor command inhibition mechanisms in SCI participants during MI. Jurkiewicz et al. (2007) observed that brain plasticity is likely to affect secondary motor areas during the subacute stages following SCI, while changes in cM1 occur during the chronic stages of motor recovery. In the present study, the SCI participant was 1 year post-injury and medically stable. The occurrence of changes in primary motor regions was therefore compatible with the delay usually observed for cerebral plasticity in primary motor sites.

Weakened cM1 inhibition during MI might reflect adaptive brain changes. Indeed, MI abilities after SCI should be preserved since cerebral structures are not damaged. However, inhibitory mechanisms during MI would become unnecessary due to SCI. This working hypothesis is relevant for MI of infra-lesional movements. Although residual voluntary motor functions remained available, the SCI participant exhibited important upper limb motor impairment. Paralysis of forearm flexor muscles and central deafferentation of the forearm and palm haptic sensors are also likely to favor brain plasticity reflected during MI by weakened inhibitory processes. This hypothesis is in agreement with the findings by Curt et al. (2002) who observed that brain changes following SCI also affected non-paralyzed limb motor cortical representation.

## **Cortical substrates for weakened inhibition during MI after SCI**

Measurement of forearm kinematics by the optical fiber system did not reveal any overt movement during MI in both participants, hence attesting the inhibition of the corresponding motor command during MI. This question thus addresses whether the difference observed in cM1 activation was attributable to weakened inhibitory processes of motor commands during MI. Increased cM1 activity during MI could also reflect a general increase in upper limb cM1 excitability. This could also occur during MI given the close functional similarity between the two tasks. Investigation of cM1 activity therefore required further analysis with regard to the hypothesis of weakened inhibitory processes during MI. Accordingly, we recorded task-related power activity in cS1 and SMA, which are believed to play a key role in cM1 inhibition during MI (Kasess et al., 2008; Schwoebel et al., 2002; Solodkin et al., 2004). We also investigated to which extent cM1 neural signals might be explained by cS1 and cSMA activities during PP and MI in both participants.

## **cS1 and cSMA activity during PP and relationships with cM1**

During PP, cS1 and cSMA were significantly activated in both participants. The cSMA and cS1 are important cortical areas of the functional loop underlying motor control. Likewise, we observed a significant relationship between cS1 and cM1 activity. This functional link is involved in the central processing of reaching movements (Koch and Rothwell, 2009; Salinas and Romo, 1998). We recorded stronger neural desynchronization in cS1 in the healthy CP. The primary sensory cortex is involved in recalling the expected sensorial consequences of actions, and is also believed to control the central integration of feedback associated with movement execution (Schwoebel et al., 2002).

The ability to generate accurate forward models of movement seemed unaffected in the patient as revealed by his capacity to achieve temporal congruence between MI and PP. Lower cS1 activity during PP measured in the SCI participant may reflect impaired sensory integration due to partial upper limb deafferentation. Schwoebel et al. (2002) reported that impaired sensory feedback integration was a potential cause regarding the failure to inhibit movements during MI. A consistent part of cortico-spinal fibers (about 20%) originate from the postcentral gyrus (Hofer and Frahm, 2009) and project down to the dorsal horn of the spinal cord. Impaired recruitment of cS1 during PP compared to MI in the SCI participant may thus potentially reflect altered motor output processing due to disruptive events in the sensorimotor feedback loop controlling movements with an impaired limb.

During PP, we also observed a significant correlation between cM1 and SMA ERDs, but only in the SCI participant. The contribution of the cSMA to motor processes during PP might therefore differ among healthy and SCI participants. In the healthy participant, pointing toward a target was easy to perform and likely to involve cortical and subcortical loops controlling automated actions, e.g., basal ganglia and cerebellum (Doyon et al., 2003). Lafleur et al. (2002) provided evidence of a fronto-striatal network controlling retrieval of cognitive strategies associated with learned routines. We thus hypothesize that activation of the SMA during PP in CP might reflect communication between pre-motor sites and subcortical regions.

Additional relationship between SMA and cM1 in the SCI participant during PP supports the recruitment of additional cognitive resources to assist motor programming. Similar observations were reported in SCI participants who attempted to perform movement from infra-lesional body segments, hence suggesting impaired central input and output after deafferentation/deafferentation (Hotz-Boendermaker et al., 2008). After SCI, quadriplegic patients need to elaborate and work on new motor programs to achieve efficient muscular control of their partially deafferented and deafferented upper limbs. This learning process is mediated by reorganizations within sensorimotor cortical maps. In the present study, increased premotor activity during PP in the SCI participant would probably reflect implementation of a new motor command.

## **cS1 and cSMA activity during PP and relationships with cM1**

Interestingly, distinct cortical patterns were observed in the two participants during MI. The cSMA was significantly activated in CP but remained silent in the SCI participant. A lack of activation in the cSMA is congruent with the hypothesis of reduced cM1 inhibition during MI in the SCI participant. This finding supports the potential inhibitory role of the cSMA toward cM1 during MI, as suggested by Kasess et al. (2008).

Furthermore, the neural activity of cS1 and cM1 was significantly correlated in CP, but not in the SCI participant. Distinguishing between forward models of movement supporting motor representation and their actual execution, Schwoebel et al. (2002) postulated that sensory feedback integration was required to separate MI from PP. Therefore, this outcome also supports the working hypothesis of weakened inhibitory mechanisms after SCI.

Lastly, cM1 neural activity was explained by cS1 and cSMA interaction in CP, whereas these areas, presumably involved in the inhibition of cM1 during MI, did not influence cM1 activity in the SCI participant. Taken together, these differences revealed by SOI analysis during MI may, at least partially, underlie the different patterns of cM1 activation

observed during MI and PP in the two participants.

## Neuroplasticity and weakened inhibition after SCI

Roy et al. (2011) reported reduced intracortical inhibition after SCI. This neural mechanism is potentially reflecting the unmasking of latent cortical synaptic connections (Saturno et al., 2008). Thus, weakened inhibition of the motor cortex might enable adaptive plastic changes after deafferentation and deafferentation. In the present study, reduced cM1 inhibition during imagined movements in the SCI participant could highlight the specific role of cM1 in the shaping of new motor commands after partial deafferentation and deafferentation (i.e., cortical efferences until the C6-C7 spinal level was preserved in the patient, since the CNS damage was at this level), which was not observed in the CP. This proposal is congruent with the fact that MI and motor preparation are functionally equivalent neural processes (Jeannerod, 1994), and that cM1 is believed to serve non-executive functions during MI (i.e., assist in motor planning—(Sharma et al., 2008)).

## CONCLUSION

The present results support the hypothesis of both cS1 and cSMA participation in the inhibition of cM1 during MI. However, motor command inhibition during MI might represent a multimodal process that should not be reduced to its cortical component (Guillot et al., 2012). Although cortical inputs toward cM1 seem to contribute to motor command suppression, the cerebellum is also believed to exert inhibitory influences on motor commands during MI (Lotze et al., 1999b). Furthermore, Bonnet et al. (1997) hypothesized that such processes could intervene at the spinal level, with cerebral structures potentially interacting with the spinal cord neurons, to maintain corticospinal facilitation below the motor threshold during MI (Stinear, 2010).

As with all experimental research, there are some limitations in our study that should be underlined before drawing final conclusions. It remains a single-case study, even if we included a control participant. This design obviously requires generalization to larger samples of patients with similar SCI injury. From a methodological viewpoint, we investigated neural activity and statistical relationships in predetermined SOI, but we did not identify the origin or pathways of the neural inputs inside these SOI, neither did we clearly establish whether the neural links observed were inhibitory or excitatory. Because of our necessarily limited number of MEG trials ( $n = 30$ ), we could not investigate the contribution of subcortical structures known to play a critical role during MI (Gerardin et al., 2000; Lotze et al., 1999b; Munzert et al., 2009), which is a perspective for future studies.

The present work must therefore be considered a preliminary MEG study focusing on MI use after complete quadriplegia. In keeping with findings by Lacourse et al. (1999), the present work might contribute to bridging the gap between brain plasticity after SCI and inhibitory processes involved in covert motor stages. Understanding the neural underpinnings of MI after SCI might further contribute to determining the MI content for fruitful implementations in physical rehabilitation.

## References

- Alkadhi, H., Brugger, P., Boendermaker, S., Crelier, G., Curt, A., Hepp-Reymond, M., and Kollias, S. (2005). What disconnection tells about motor imagery: Evidence from paraplegic patients. *Cerebral Cortex*, 15(2):131–140.
- Babiloni, C., Carducci, F., Cincotti, F., Rossini, P., Neuper, C., Pfurtscheller, G., and Babiloni, F. (1999). Human movement-related potentials vs desynchronization of eeg alpha rhythm: A high-resolution eeg study. *NeuroImage*, 10(6):658–665.
- Battapady, H., Lin, P., Fei, D., Huang, D., and Bai, O. (2009a). Single trial detection of human movement intentions from sam-filtered meg signals for a high performance two-dimensional bci. In *Annual International Conference of the IEEE Engineering in Medicine and Biology Society. IEEE Engineering in Medicine and Biology Society Conference*, pages 524–527.
- Battapady, H., Lin, P., Holroyd, T., Hallett, M., Chen, X., Fei, D., and Bai, O. (2009b). Spatial detection of multiple movement intentions from sam-filtered single-trial meg signals. *Clinical Neurophysiology*, 120(11):1978–1987.
- Beaulé, V., Tremblay, S., and Théoret, H. (2012). Interhemispheric control of unilateral movement. *Neural Plasticity*, 2012.
- Bonnet, M., Decety, J., Jeannerod, M., and Requin, J. (1997). Mental simulation of an action modulates the excitability of spinal reflex pathways in man. *Cognitive Brain Research*, 5(3):221–228.
- Bruehlmeier, M., Dietz, V., Leenders, K., Roelcke, U., Missimer, J., and Curt, A. (1998). How does the human brain deal with a spinal cord injury? *European Journal of Neuroscience*, 10(12):3918–3922.
- Burianova, H., Marstaller, L., Sowman, P., Tesan, G., Rich, A., Williams, M., and Johnson, B. (2013). Multimodal functional imaging of motor imagery using a novel paradigm. *NeuroImage*.
- Collet, C. and Guillot, A. (2010). Autonomic nervous system activities during imagined movements. In *The neurophysiological foundations of mental and motor imagery*, pages 95–108. Oxford University Press.
- Collet, C., Guillot, A., Lebon, F., MacIntyre, T., and Moran, A. (2011). Measuring motor imagery using psychometric, behavioral, and psychophysiological tools. *Exercise and Sport Science Reviews*, 39(2):85–92.
- Cramer, S., Lastra, L., Lacourse, M., and Cohen, M. (2005). Brain motor system function after chronic, complete spinal cord injury. *Brain*, 128(Pt 12):2941–2950.
- Crawford, J., Garthwaite, P., and Wood, L. (2010). Inferential methods for comparing two single cases. *Cognitive Neuropsychology*, 27(5):377–400.
- Curt, A., Alkadhi, H., Crelier, G., Boendermaker, S., Hepp-Reymond, M., and Kollias, S. (2002). Changes of non-affected upper limb cortical representation in paraplegic patients as assessed by fmri. *Brain*, 125(Pt 11):2567–2578.

- Decety, J., Perani, D., Jeannerod, M., Bettinardi, V., Tadary, B., Woods, R., Mazziotta, J., and Fazio, F. (1994). Mapping motor representations with positron emission tomography. *Nature*, 371(6498):600–602.
- Dechent, P., Merboldt, K., and Frahm, J. (2004). Is the human primary motor cortex involved in motor imagery? *Cognitive Brain Research*, 19(2):138–144.
- Doyon, J., Penhune, V., and Ungerleider, L. (2003). Distinct contribution of the cortico-striatal and cortico-cerebellar systems to motor skill learning. *Neuropsychologia*, 41(3):252–262.
- Dunlop, S. (2008). Activity-dependent plasticity: Implications for recovery after spinal cord injury. *Trends in Neurosciences*, 31(8):410–418.
- Ehrsson, H., Geyer, S., and Naito, E. (2003). Imagery of voluntary movement of fingers, toes, and tongue activates corresponding body-part-specific motor representations. *Journal of Neurophysiology*, 90(5):3304–3316.
- Gerardin, E., Sirigu, A., Lehericy, S., Poline, J., Gaymard, B., Marsault, C., Agid, Y., and Le Bihan, D. (2000). Partially overlapping neural networks for real and imagined hand movements. *Cerebral Cortex*, 10(11):1093–1104.
- Green, J., Sora, E., Bialy, Y., Ricamato, A., and Thatcher, R. (1998). Cortical sensorimotor reorganization after spinal cord injury: An electroencephalographic study. *Neurology*, 50(4):1115–1121.
- Guillot, A. and Collet, C. (2005). Contribution from neurophysiological and psychological methods to the study of motor imagery. *Brain Research Reviews*, 50(2):387–397.
- Guillot, A., Collet, C., and Dittmar, A. (2004). Relationship between visual and kinesthetic imagery, field dependence-independence, and complex motor skills. *Journal of Psychophysiology*, 18(4):190–198.
- Guillot, A., Collet, C., Nguyen, V., Malouin, F., Richards, C., and Doyon, J. (2008). Functional neuroanatomical networks associated with expertise in motor imagery. *NeuroImage*, 41(4):1471–1483.
- Guillot, A., Di Rienzo, F., Macintyre, T., Moran, A., and Collet, C. (2012). Imagining is not doing but involves specific motor commands: A review of experimental data related to motor inhibition. *Frontiers in Human Neuroscience*, 6:247.
- Guillot, A., Hoyek, N., Louis, M., and Collet, C. (2011). Understanding the timing of motor imagery: Recent findings and future directions. *International Review of Sport and Exercise Psychology*, 5(1):3–22.
- Guillot, A., Lebon, F., and Collet, C. (2010). Electromyographic activity during motor imagery. In *The neurophysiological foundations of mental and motor imagery*, pages 83–93. Oxford University Press.
- Guillot, A., Lebon, F., Rouffet, D., Champely, S., Doyon, J., and Collet, C. (2007). Muscular responses during motor imagery as a function of muscle contraction types. *International Journal of Psychophysiology*, 66(1):18–27.

- Hofer, S. and Frahm, J. (2009). Topographical organization of the pyramidal fiber system—diffusion tensor mri of the human and rhesus monkey brain. *Open Medical Imaging Journal*, 3:6–14.
- Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics*, 6:65–70.
- Hotz-Boendermaker, S., Funk, M., Summers, P., Brugger, P., Hepp-Reymond, M., Curt, A., and Kollias, S. (2008). Preservation of motor programs in paraplegics as demonstrated by attempted and imagined foot movements. *NeuroImage*, 39(1):383–394.
- Jeannerod, M. (1994). The representing brain: Neural correlates of motor intention and imagery. *Behavioral and Brain Sciences*, 17(2):187–202.
- Jurkiewicz, M., Mikulis, D., McIlroy, W., Fehlings, M., and Verrier, M. (2007). Sensorimotor cortical plasticity during recovery following spinal cord injury: A longitudinal fmri study. *Neurorehabilitation and Neural Repair*, 21(6):527–538.
- Kasess, C., Windischberger, C., Cunnington, R., Lanzenberger, R., Pezawas, L., and Moser, E. (2008). The suppressive influence of sma on m1 in motor imagery revealed by fmri and dynamic causal modeling. *NeuroImage*, 40(2):828–837.
- Koch, G. and Rothwell, J. (2009). Tms investigations into the task-dependent functional interplay between human posterior parietal and motor cortex. *Behavioral Brain Research*, 202(2):147–152.
- Kokotilo, K., Eng, J., and Curt, A. (2009). Reorganization and preservation of motor control of the brain in spinal cord injury: A systematic review. *Journal of Neurotrauma*, 26(11):2113–2126.
- Lacourse, M., Cohen, M., Lawrence, K., and Romero, D. (1999). Cortical potentials during imagined movements in individuals with chronic spinal cord injuries. *Behavioral Brain Research*, 104(1–2):73–88.
- Lafleur, M., Jackson, P., Malouin, F., Richards, C., Evans, A., and Doyon, J. (2002). Motor learning produces parallel dynamic functional changes during the execution and imagination of sequential foot movements. *NeuroImage*, 16(1):142–157.
- Lang, W., Cheyne, D., Hollinger, P., Gerschlagner, W., and Lindinger, G. (1996). Electric and magnetic fields of the brain accompanying internal simulation of movement. *Cognitive Brain Research*, 3(2):125–129.
- Levy, W. J., Amassian, V., Traad, M., and Cadwell, J. (1990). Focal magnetic coil stimulation reveals motor cortical system reorganized in humans after traumatic quadriplegia. *Brain Research*, 510(1):130–134.
- Lotze, M. and Halsband, U. (2006). Motor imagery. *Journal of Physiology, Paris*, 99(4–6):386–395.
- Lotze, M., Laubis-Herrmann, U., Topka, H., Erb, M., and Grodd, W. (1999a). Reorganization in the primary motor cortex after spinal cord injury—a functional magnetic resonance (fmri) study. *Restorative Neurology and Neuroscience*, 14(2–3):183–187.

- Lotze, M., Montoya, P., Erb, M., Hulsmann, E., Flor, H., Klose, U., and Grodd, W. (1999b). Activation of cortical and cerebellar motor areas during executed and imagined hand movements: An fmri study. *Journal of Cognitive Neuroscience*, 11(5):491–501.
- Lotze, M. and Zentgraf, K. (2010). Contribution of the primary motor cortex to motor imagery. In *The neurophysiological foundations of mental and motor imagery*, pages 31–45. Oxford University Press.
- Macuga, K. and Frey, S. (2012). Neural representations involved in observed, imagined, and imitated actions are dissociable and hierarchically organized. *NeuroImage*, 59(3):2798–2807.
- Malouin, F., Richards, C., Durand, A., and Doyon, J. (2008). Reliability of mental chronometry for assessing motor imagery ability after stroke. *Archives of Physical Medicine and Rehabilitation*, 89(2):311–319.
- Malouin, F., Richards, C., Jackson, P., Lafleur, M., Durand, A., and Doyon, J. (2007). The kinesthetic and visual imagery questionnaire (kviq) for assessing motor imagery in persons with physical disabilities: A reliability and construct validity study. *Journal of Neurologic Physical Therapy*, 31(1):20–29.
- Mateo, S., Revol, P., Fournassi, M., Rossetti, Y., Collet, C., and Rode, G. (2012). Kinematic characteristics of tenodesis grasp in c6 quadriplegia. *Spinal Cord*.
- Maynard, F. J., Bracken, M., Creasey, G., Ditunno, J. J., Donovan, W., Ducker, T., Garber, S., Marino, R., Stover, S., Tator, C., Waters, R., Wilberger, J., and Young, W. (1997). International standards for neurological and functional classification of spinal cord injury. *Spinal Cord*, 35(5):266–274.
- McFarland, D., Miner, L., Vaughan, T., and Wolpaw, J. (2000). Mu and beta rhythm topographies during motor imagery and actual movements. *Brain Topography*, 12(3):177–186.
- Michelon, P., Vettel, J., and Zacks, J. (2006). Lateral somatotopic organization during imagined and prepared movements. *Journal of Neurophysiology*, 95(2):811–822.
- Muakkassa, K. and Strick, P. (1979). Frontal lobe inputs to primate motor cortex: Evidence for four somatotopically organized ‘premotor’ areas. *Brain Research*, 177(1):176–182.
- Munzert, J., Lorey, B., and Zentgraf, K. (2009). Cognitive motor processes: The role of motor imagery in the study of motor representations. *Brain Research Reviews*, 60(2):306–326.
- Neuper, C. and Pfurtscheller, G. (2010). Electroencephalographic characteristics during motor imagery. In *The neurophysiological foundations of mental and motor imagery*, pages 65–81. Oxford University Press.
- Pandya, D. and Vignolo, L. (1971). Intra- and interhemispheric projections of the precentral, premotor and arcuate areas in the rhesus monkey. *Brain Research*, 26(2):217–233.
- Pfurtscheller, G. and Lopes da Silva, F. (1999). Event-related eeg/meg synchronization and desynchronization: Basic principles. *Clinical Neurophysiology*, 110(11):1842–1857.

- Porro, C., Cettolo, V., Francescato, M., and Baraldi, P. (2000). Ipsilateral involvement of primary motor cortex during motor imagery. *European Journal of Neuroscience*, 12(8):3059–3063.
- Porro, C., Francescato, M., Cettolo, V., Diamond, M., Baraldi, P., Zuiani, C., Bazzocchi, M., and Di Prampero, P. (1996). Primary motor and sensory cortex activation during motor performance and motor imagery: A functional magnetic resonance imaging study. *Journal of Neuroscience*, 16(23):7688–7698.
- Robinson, S. (2004). Localization of event-related activity by sam(erf). *Neurology and Clinical Neurophysiology*, 2004:109.
- Roy, F., Zewdie, E., and Gorassini, M. (2011). Short-interval intracortical inhibition with incomplete spinal cord injury. *Clinical Neurophysiology*, 122(7):1387–1395.
- Sabbah, P., Simond, G., Levrier, O., Habib, M., Trabaud, V., Murayama, N., Ceccaldi, M., and Salamon, G. (1995). Functional magnetic resonance imaging at 1.5 t during sensorimotor and cognitive task. *European Neurology*, 35(3):131–136.
- Salinas, E. and Romo, R. (1998). Conversion of sensory signals into motor commands in primary motor cortex. *Journal of Neuroscience*, 18(1):499–511.
- Saturno, E., Bonato, C., Miniussi, C., Lazzaro, V., and Callea, L. (2008). Motor cortex changes in spinal cord injury: A tms study. *Neurological Research*, 30(10):1084–1085.
- Schnitzler, A., Salenius, S., Salmelin, R., Jousmaki, V., and Hari, R. (1997). Involvement of primary motor cortex in motor imagery: A neuromagnetic study. *NeuroImage*, 6(3):201–208.
- Schwoebel, J., Boronat, C., and Branch Coslett, H. (2002). The man who executed “imagined” movements: Evidence for dissociable components of the body schema. *Brain and Cognition*, 50(1):1–16.
- Sharma, N., Jones, P., Carpenter, T., and Baron, J. (2008). Mapping the involvement of ba 4a and 4p during motor imagery. *NeuroImage*, 41(1):92–99.
- Solodkin, A., Hlustik, P., Chen, E., and Small, S. (2004). Fine modulation in network activation during motor execution and motor imagery. *Cerebral Cortex*, 14(11):1246–1255.
- Stephan, K., Fink, G., Passingham, R., Silbersweig, D., Ceballos-Baumann, A., Frith, C., and Frackowiak, R. (1995). Functional anatomy of the mental representation of upper extremity movements in healthy subjects. *Journal of Neurophysiology*, 73(1):373–386.
- Stinear, C. (2010). Corticospinal facilitation during motor imagery. In *The neurophysiological foundations of mental and motor imagery*, pages 47–61. Oxford University Press.
- Topka, H., Cohen, L., Cole, R., and Hallett, M. (1991). Reorganization of corticospinal pathways following spinal cord injury. *Neurology*, 41(8):1276–1283.
- Vernet-Maury, E., Robin, O., and Dittmar, A. (1995). The ohmic perturbation duration, an original temporal index to quantify electrodermal responses. *Behavioral Brain Research*, 67(1):103–107.

Winstein, C., Grafton, S., and Pohl, P. (1997). Motor task difficulty and brain activity: Investigation of goal-directed reciprocal aiming using positron emission tomography. *Journal of Neurophysiology*, 77(3):1581–1594.