Changing your mind before it is too late: the electrophysiological correlates of online error correction during response selection

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Running head: Mechanisms of online executive control

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Abstract

Inhibiting actions when they are no longer appropriate is essential for adaptive goal-directed behavior. In this study, we used high-density EEG and a standard flanker task to explore the spatio-temporal dynamics of cognitive control and inhibitory mechanisms aimed to prevent the commission of errors. By recording hand-related EMG activity, we could disentangle successful from unsuccessful inhibition attempts. Our results confirm that (i) the latency of the ERN (or Ne) component is too late to be associated with these online inhibitory mechanisms; (ii) instead, a frontal slow negative component with an earlier time-course was associated with the implementation of online inhibition. These findings are consistent with single-cell recordings in monkeys showing that the supplementary motor area (SMA) provides cognitive control signals to the primary motor cortex to exert online inhibition and in turn rectify the course of erroneous actions.

Keywords: Executive control, Inhibition, Motor cortex, Electroencephalography, Electromyography

Introduction

Daily life requires constantly adjusting behavior to rapidly changing contingencies in the external environment: conflicts between competing responses need to be solved in favor of the correct response and error commission has to be prevented. In this respect, it is known that cognitive control mechanisms monitor behavior and instigate behavioral adjustments upon encountering events such as errors or conflicts, with the purpose of minimizing the probability of errors on subsequent trials. Such mechanisms may explain why humans usually slow down and perform better after errors (Rabbitt, 1966; Laming, 1979) or after conflict-situations (Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; Stürmer, Leuthold, Soetens, Schroter, & Sommer, 2002).

These cognitive control mechanisms have mainly been studied when operating from the current trial to the next one. Yet, efficient and successful behavior also requires adaptive control mechanisms that operate during the course of a single action allowing to correct behavior online, as it occurs for instance, when driving a bicycle and realizing that the current steering action is not compatible with the current goal (e.g. turning left, but rapidly correcting the motor behavior to produce the expected action, i.e. turning right). This requires rapid monitoring of an action while it unfolds and, if necessary, immediate inhibition of the ongoing action and a rapid switch to the execution of a corrective behavior. Despite its daily-life importance and unlike trial-to-trial adjustments, brain mechanisms underlying online behavioral control are still poorly understood and explored.

Because corrective behavior results from active online inhibition of the initial (incorrect) action and the rapid switch to the initiation of the correct action, the study of partial errors as measured with electromyography (EMG), offers a useful means to explore online behavioral control. Partial errors correspond to subthreshold (yet EMG detectable) motor responses that are successfully inhibited before they turn into an overt error (Hasbroucq, Possamaï, Bonnet, & Vidal 1999). As shown in figure 1C, partial errors can be derived from muscular activity occurring in the incorrect hand before the correct response is given with the other (competing) hand.

The involvement of an online stopping mechanism in partial errors has to our knowledge never been explicitly demonstrated but it is indirectly supported by the observation of traces of inhibitory activity in errors, in which, contrary to partial errors, the remedial process was visibly unsuccessful. For instance, Rabbitt (1978) noticed that the trace of the ink left on paper by professional typists pressing a wrong key during typewriting was actually less pronounced than for a correct key, indicating that less force was exerted during the former case (for consistent results, see Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996; Gehring & Knight, 2000). More recently Allain, Carbonnell, Burle, Hasbroucq and Vidal (2004) reported a reduced EMG activation for errors compared with correct responses, providing physiological evidence for the involvement of an online inhibition mechanism in (overt) error commission. This result has been confirmed by Cohen & van Gaal (2014).

As the online control mechanisms that are involved in these corrective actions have, to our knowledge, not been described extensively yet, the goal of our study was to characterize the electrophysiological correlates of online control mechanisms supporting the internally driven corrective behavioral adjustments. We address this question using EMG combined with concurrent electroencephalographic (EEG) recordings. More specifically, while participants performed a standard flanker task, we compared EEG activities over the motor and premotor areas as a function of three different EMG outcomes (see figure 1): (i) correct responses (figure 1A) without online adjustment; (ii) full errors (figure 1B) where online adjustment is detected but an incorrect response is produced nevertheless and (iii) partial errors with initial covert activation of the erroneous response followed by the production of the correct response (Incorrect-Correct trials - figure 1C). The systematic comparison of the EEG cortical activities related to these three separate outcomes enables us to characterize the time-course and actual electrophysiological manifestations of the online inhibition mechanism that prevents error commission during the course of the action. An asset of this approach is the possibility offered to investigate changes in the ERP components time-locked to the onset of the response as a function of (either enhanced or weaker) cognitive control, as opposed to previous attempts that explored a similar

mechanism by focusing on stimulus-locked ERP effects (using GO-NOGO or stop-signal tasks; e.g. Kropotov, Ponomarev, Hollup & Mueller, 2011).

Because primary motor cortices (M1s) execute motor commands by transmitting information to the muscles via the spinal cord, we first verified whether M1s are involved in the reduction of the EMG activity in erroneous actions. This hypothesis derives from the fact that inhibition over the M1s has already been observed using various methods like EEG, transcranial magnetic stimulation (TMS) and monosynaptic reflex technique investigations (H reflex). More precisely in the context of a task requiring a selection between the two hands, the unimanual motor command is characterized by the activation of the contralateral M1 revealed by a negative wave observed with EEG, an increase of the cortico-spinal and the spinal excitabilities investigated respectively with TMS and H reflex (Hasbroucq, Akamatsu, Burle, Bonnet, & Possamaï, 2000; Burle, Bonnet, Vidal, Possamaï, & Hasbroucq, 2002; Vidal, Grapperon, Bonnet, & Hasbroucq, 2003; Burle, Vidal, Tandonnet, & Hasbroucq, 2004; Duque, Lew, Mazzocchio, Olivier, & Ivry, 2010). Interestingly, these studies have also shown that this contralateral M1 activation is associated with an inhibition of the ipsilateral M1 (revealed by a concurrent positive EEG activity and a decrease of the cortico-spinal and the spinal excitabilities). This has been interpreted as a neural coupling that prevents the execution of the erroneous response. Furthermore, it has been shown with TMS that an external signal of stopping triggers an increase in excitability of $GABA_B$ergic intracortical inhibitory circuits in the contralateral M1 that is about to execute the motor response (van den Wildenberg et al., 2010). We aimed at investigating to what extent a similar mechanism is at play in internally driven online inhibition. We expected to observe a trace of online inhibition on M1 activity during the response execution in both partial errors and overt errors, but not in correct responses. Since the stopping is successful in partial errors, the trace of online inhibition should be stronger and/or appear earlier in partial errors than in full error responses.

The next step then was to identify the mechanisms that precede and enable the online inhibition at the level of M1. Earlier research suggests two plausible neural candidates, namely the ACC because of its

well-known involvement in error monitoring and trial-to-trial behavioral adaptations (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Falkenstein, Hohnsbein, & Hoormann, 1994) and SMA/preSMA because of its implication in stopping behavior (Li, Huang, Constable, & Sinha, 2006; Duann, Ide, Luo, & Li, 2009; Aron, 2011).

To evaluate the possible involvement of the ACC, we investigated the Ne/ERN, a large negative deflection observed after error commission over fronto-central electrodes along the midline, likely generated within the ACC (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring et al., 1993; Dehaene, Posner, & Tucker, 1994; Debener et al., 2005). A tight link between the characteristics of this event-related potential (ERP) component and the nature of error correction has been investigated repeatedly (Gehring et al., 1993; Falkenstein et al., 1994; Rodríguez-Fornells, Kurzbuch, & Münte, 2002; Fiehler, Ullsperger, & von Cramon, 2005; Ullsperger & von Cramon, 2006). However, these studies only investigated correction of overt errors in which online inhibition mechanisms failed. By relating the temporal characteristics of the Ne/ERN to the temporal onset of inhibition of the erroneous response (as revealed by EMG information), we evaluated the possibility that the Ne/ERN might send a command sufficiently rapidly in order to enable stopping the erroneous action before it is fully executed. Despite the fact that the Ne/ERN usually peaks around 100 to 150 ms, it builds up before EMG onset, allowing us to test the hypothesis that this ERP drives the online inhibition (observable from 30 ms post-EMG onset on EMG traces, Allain et al. (2004)).

The second possibility is that the online inhibition is driven by the SMA/preSMA. The preSMA has been associated with the suppression of unwanted actions (Isoda & Hikosaka, 2007; Chen, Scangos, & Stuphorn, 2010), the facilitation of action reprogramming (Yang, Heinen, & Missal 2008; Mars et al., 2009; Neubert, Mars, Buch, Olivier, & Rushworth, 2010) and the preparation for the execution of a second action (Nakajima, Hosaka, Mushiake, & Tanji, 2009). Furthermore, patients with SMA/preSMA lesions are impaired at inhibiting an initiated response when they are instructed to do so (Floden & Stuss, 2006; Nachev, Wydell, O'neill, Husain, & Kennard 2007). To test the involvement of

SMA/preSMA in online inhibition, we explored activities over fronto-central regions that emerge before the onset of the muscular activity (as established using EMG). Such activity might be related to a small fronto-central negativity, called the N-40, that has been shown to develop 100 ms prior to EMG onset in correct trials particularly when the risk of error was high (Vidal, Burle, Grapperon, & Hasbroucq, 2011), although it has not been explored in the context of online corrective behavior. Finally, a third candidate region for this online inhibition could be the right inferior frontal cortex (rIFC), given its predominant role in inhibition during stop signal tasks (e.g. Aron, Robbins, & Poldrack, 2004). However, we could not put to the test this hypothesis because there is no clear response-locked ERP component that has previously been linked to activity originating from the rIFC. The contribution of this lateral frontal region to inhibition has mostly been evidenced using fMRI and patient studies (e.g. Forstmann, Jahfari, Scholte, Wolfensteller, van den Wildenberg, & Ridderinkhof, 2008).

Material and Method

Participants

Nineteen right-handed volunteers (9 males and 10 females; age 18–23 years; mean age 20.1) with normal or corrected to normal vision and no history of neurological or psychiatric disease took part in the experiment. The experiment took place in the department of experimental psychology of Ghent University. Before the start of the experiment, all the participants gave their informed written consent according to the Declaration of Helsinki. The study was approved by the local ethics committee.

Stimuli, material and task

A social version of the classical flanker task (see Eriksen and Eriksen (1974) for the original task) was used. In this task, either action execution or observation (of a confederate) was required, alternating from trial to trial. In this article we report the results of the execution trials only.

A specific experimental set up with a table was used with two subjects sitting on each side of this table (Núñez Castellar, Notebaert, Van den Bossche, & Fias, 2011). In the center of the table a touch-screen

monitor was placed with the screen facing up (30 cm wide x 22.5 cm high). The screen contained two display sides (15 cm x 11 cm). Each side displayed a stimulus zone and two response buttons (white, 3 cm x 2.2 cm, positioned bilaterally around the stimulus but slightly towards the participant). The task was programmed using Tscope (Stevens, Lammertyn, Verbruggen, & Vandierendonk, 2006).

During the execution trials, the task required to identify the center stimulus of a horizontal stimulus array. Stimuli consisted of a central arrowhead pointing either to the left or to the right, flanked by arrowhead distractors (left and right of the central arrowhead) that pointed either in the same direction as the center arrow (congruent trials), or in opposite direction (incongruent trials). The probability of left and right pointing center arrows was balanced, as well as the proportion of congruent and incongruent flankers. Two buttons were positioned bilaterally on the touch screen to either side of the stimulus. Participants were asked to press the button corresponding with the direction of the central arrow (with the index finger of either hand).

Procedure

At the beginning of each session, participants were instructed to perform the task as accurately and as fast as possible.

For each trial, flankers were presented 80 ms prior to target onset to maximize the expected flanker compatibility effect (Kopp, Rist, & Mattler, 1996). Then the target was presented together with the flankers during 30 ms. Flankers and target disappeared simultaneously. Starting from target onset, participants had a maximum duration of 1500 ms to press one of the two response buttons. Once a response was given or the response deadline was passed, an inter-trial interval of 600 ms was included before the next trial started.

One practice block of 20 trials and 12 experimental blocks containing 60 trials were presented with a short break between them. The experiment lasted about 50 min.

Data Acquisition and Pre-processing

EEG activity was recorded with 64 Ag/AgCl scalp electrodes (positioned according to the extended 10-20 International EEG system; Jasper, 1958) distributed evenly over the scalp using an elastic cap. Electromyographic activities (EMG) from the first dorsal interosseus of each hand were recorded simultaneously by paired surface Ag/AgCl electrodes (BIOSEMI Active-two electrodes, Amsterdam). Electro-oculographic (EOG) activities were recorded bipolarly using electrodes placed near both canthi (horizontal EOG) and above or below the left eye (vertical EOG). The sampling rate was set to 1024 Hz (filters: DC to 268 Hz, 3 dB/octave). The data were referenced off line to the left mastoid.

A 0.16 high-pass and a 100 Hz low-pass filter were applied to the raw EEG data. Eye movement artifacts were corrected by the statistical method of Gratton, Coles and Donchin (1983). All other artifacts were rejected manually by visual inspection on the raw traces, with the experimenter not being aware of the nature of the trials.

A 10 Hz high pass filter was applied to the raw EMG data. The onset of each EMG burst was determined visually by inspecting the changes in EMG, and were marked manually thereby following the recommendation of van Boxtel, Geraats, Van den Berg-Lessen and Brunia (1993) and Staude, Flachenecker, Daumer and Wolf (2001) who showed that manual detections of the EMG onsets are more accurate than using automated algorithms. Importantly, trials were classified as a function of both the accuracy and the information given by the EMGs. Among the erroneous trials, only the ones showing a single EMG burst and without any visible correction after the recorded response were kept and labeled as “full error trials”. Likewise, the “pure-correct trials” retained in the statistical analyses corresponded to a single EMG burst alike. The trials belonging to last category were special correct trials in the sense that an incorrect EMG burst (i.e. a “partial error”) was observed before the correct EMG burst leading to the response. These trials were labeled as “incorrect-correct trials” because the error was about to be performed but has been refrained on time (see figure 1 for examples of the three types of trials). Since our main goal was to study cognitive control during the course of an action, we treated congruent and incongruent trials together. This choice was also motivated by the need to collect

as many trials as possible in each cell of the design (pure-correct trials, full errors and incorrect-correct trials).

Data Processing

The chronometric variables analyzed in the present study were defined as a function of the latency of the EMG activations (see Figure 1). The reaction time (RT), as measured from target onset to the recording of the response by the device, was fractionated into pre-motor (PMT) and motor time (MT) for the trials showing a single EMG burst (i.e. pure-correct and full error trials). PMT corresponds to the time separating target presentation and onset of the EMG, and the MT to the time separating onset of the EMG activity and the mechanical response. For incorrect-correct trials, we were also interested in the partial error's onset or incorrect activation time (IAT, i.e. the time separating target onset and start of the partial error) and the correction time (CT, i.e. the time separating the start of the partial error and the onset of the EMG of the remedial action).

EEG and EMG time-courses were time-locked to the EMG onset of the burst leading to the response in pure-correct and full error trials, and to the EMG onset of the partial error for incorrect-correct trials. To analyze the motor activities associated with correct and incorrect responses, we specified the EMGs as a function of the required response (i.e. "Correct EMG" and "Incorrect EMG", see Figure 1), instead of hand performing the movement (i.e. left or right). Then the EMG traces from the hand associated with the correct response (i.e. left EMGs in trials requiring a left response and right EMGs in trials requiring a right response) as well as the traces from the hand associated to the incorrect response (i.e. left EMGs in trials requiring a right response and right EMGs in trials requiring a left response), were averaged together. A baseline correction was applied before the EMG onset (between -150 and 0ms) and the signals were rectified before being averaged as a function of the trial type (Hasbroucq et al., 1999). The area under the curve was calculated in time windows on the averaged traces between 0 and 150 ms for each participant.

Usually, two complementary methods can be used to extract cerebral motor activities using EEG. The classical one consists in measuring the lateralized readiness potential (LRP) which is assessed by using the ERP waveforms on monopolar data recorded at C3 and C4 using the double subtraction-averaging method (de Jong, Wierda, Mulder, & Mulder, 1988). Although this method provides a good approximation of motor activities, it also has limitations. The LRP is blind to the respective contributions of each motor cortex in each hemisphere (Gratton, 1998; Vidal et al., 2003). Moreover, the LRP is measured on monopolar data which are characterized by a limited spatial resolution, partly due to volume conduction effects (Nunez & Srinivasan, 2005).

To overcome these limitations, we computed the Laplacian transform on the monopolar data in order to improve substantially the spatial resolution of motor-related EEG activities (a technique also called "current source density" - CSD). This method is basically acting as a high-pass spatial filter by removing the blurring effect due to volume conduction (Nunez & Srinivasan, 2005; Babiloni, Cincotti, Carducci, Rossini, & Babiloni 2001) and is considered as a correct approximation of the electrocorticogram (Gevins, 1989). In this work, we used a standard surface Laplacian transformation, as implemented in BrainAnalyser software (Munich, Germany). Firstly a spherical spline interpolation was applied using the following parameters: 3 as the degree of spline and 15 as a maximum of degrees for the Legendre polynomial (Perrin, Bertrand, & Pernier, 1987; Perrin, Pernier, Bertrand, & Echallier, 1989). Secondly, the second derivative was computed in two dimensions of the space. This method is particularly valuable to study the electrophysiological time-course of motor activities since it allows disentangling the electric activities originating from the contralateral vs. ipsilateral motor cortices relative to the responding hand with an improved spatial resolution compared with monopolar recordings (3-4 cm and 10 cm, respectively). Because of these specific properties, nowadays, a growing number of studies are using Laplacian or CSD methods (e.g. Burle et al., 2004; Praamstra & Seiss, 2005; Roger, Bénar, Vidal, Hasbroucq, & Burle, 2010; Oldenburg, Roger, Assecondi, Verbruggen, & Fias, 2012; van de Laar, van den Wildenberg, van Boxtel, Huizenga, & van der Molen, 2012). A

limitation of this method is its low sensitivity to deep sources. This might potentially be a problem for the ERN component, given its putative deep sources in the ACC. However several earlier ERP studies already used this method successfully in the past to analyze the spatio-temporal dynamics of the ERN component, and they did not report spurious results when compared to other methods (e.g. Roger et al. (2010) for a direct comparison between Laplacian transform and ICA decomposition of the ERN component).

Following the same logic as with EMG data, we distinguished the activities contralateral and ipsilateral to the responding hand at the M1 level. More precisely we merged the activities at C3 in right responses with the activities at C4 in left responses to obtain the "Contralateral M1" virtual electrode, and we merged the activities at C4 in right responses and at C3 in left responses to obtain the "Ipsilateral M1" virtual electrode. Arbitrarily we decided to present on the topography maps the contralateral activities on the left hemisphere and the ipsilateral activities on the right one.

The baseline time windows were chosen such that they fall before the activities of interest and overlap neatly across conditions (i.e. -300/-150 ms). Time courses were averaged as a function of the trial type. Statistics were performed on surfaces in the time window between -150 ms and 150 ms which encompassed the peak of the activity of interest (i.e. around 0 ms).

Plots of sorted single-trials were calculated using ERP-images, as implemented in the EEGLAB toolbox (Delorme & Makeig, 2004). This method is particularly valuable in the present case, as it enables the visualization of the raw amplitudes, at the single trial level, of any systematic relationship between either the amplitude or latency of evoked brain responses, and a given behavioral or chronometric variable (Jung et al., 2001). First, for incorrect-correct trials, response-locked ERP data were time-locked to the onset of the partial error (incorrect activation). Using ERP-images, the Laplacian-transformed EEG data traces and the EMG traces were plotted concurrently, and sorted as function of the CT. As a result, in all figures, the vertical line corresponds to the actual onset of partial error and the additional S-shaped curve to the EMG onset of the remedial action. Following standard practice, the

color code corresponds to the amplitude of the signal (blue for negative and red for positive values). The individual single trial decompositions performed for each subject separately were used to provide a "grand-averaged" ERP-image. However a simple averaging of the individual data matrices containing the single trial data is not possible since the number of trials differs across participants, resulting in different matrix sizes. To overcome this problem and build a "grand-averaged" ERP-image, individual-subject matrices were re-sized according to the method used previously by Burle et al. (2008, see appendix A on p. 1651). Furthermore, this correction ensures that each subject contributes equally to the grand-averaged ERP-image. As recommended by Jung et al. (2001), the ERP images were finally smoothed with a moving window (with a width set at 10% of the available trials) to increase the signal-to-noise ratio.

To enable statistical comparisons, a median split of CTs was performed for each subject to distinguish the incorrect-correct trials in which the correction was fast (Short CTs) and the ones in which the correction was slow (Long CTs).

Statistics

The chronometric variables and the psychophysiological measurements were submitted to univariate repeated measures analyses of variance (ANOVAs) involving a single within-subject factor: response type (pure-correct, pure-error and incorrect-correct trials) in the analyses including all trials types, or the CT (short and long) in the analyses of the incorrect-correct trials. Greenhouse-Geisser correction was used for univariate repeated measures ANOVA tests involving more than one degree of freedom, in which case the uncorrected degrees of freedom, the corrected p value, and the ε value were reported. Post-hoc paired Student's t tests were performed.

Results

Behavioral data:

Using the EMG signal, 73.3 % of the trials were classified as pure-correct, 12.3 % as incorrect-correct and 14.3 % as full error. RTs for the three types of trial were significantly different ($F(2,36) = 48$; $p<.001$; $\varepsilon=0.6$), with the shortest RTs observed for full error trials (453 ms), followed by pure-correct trials (504 ms) and by incorrect-correct trials (584 ms).

The fractionation of the RTs, shown in table 1, revealed longer PMTs but faster MTs for pure-correct compared with full error trials (PMT: 314 ms and 251 ms respectively; $t(1,18) = 62$; $p<.001$; MT: 189 ms vs. 201 ms; $t(1,18) = 17$; $p<.001$), replicating previous results (Allain et al., 2004; Cohen & van Gaal, 2014). Longer RTs for incorrect-correct trials were mainly due to the presence of partial errors, i.e. an erroneous action that had been initiated, stopped and eventually replaced by a remedial action. The mean latency of the partial error (IAT) was 242 ms and the CT was 158 ms. Interestingly, there was no significant difference between the IAT of the partial error and the PMT in full error trials ($t(1,18) < 1$) suggesting that the capability to stop the wrong action could not be attributed to a difference in the speed of initiation of the erroneous action.

Electrophysiological data

First, we analyzed the EMG activities in order to track the mechanisms of online inhibition at the effector level. Next, we verified that these observations correlated with the activities of the primary motor areas. Finally, we evaluated the possible involvement of premotor areas in online inhibition mechanisms observed at the central and peripheral motor levels.

Online inhibition at the effector level: evidence from EMG

Figure 2 presents averaged rectified EMG curves for full error, pure-correct and partial error responses, each time-locked to the onset of the EMG burst. Importantly, in case of incorrect-correct trials the data were time-locked to the onset of the partial error's EMG burst and not to the one of the remedial action. To test whether EMG activity for full errors was different compared with pure-correct and partial error responses, we ran an ANOVA on the area under the curve measured in the time interval from 0 to 150 ms. The same criteria as Allain et al. (2004) was used to choose the time window for the analysis (i.e. a

time window encompassing the peak of the bursts for individual subjects). As expected, we observed a strong effect of the response type on the surface under the curve ($F(2,36) = 67.1$; $p < .0001$; $\varepsilon=0.6$) with the largest EMG surface for correct responses and the lowest for partial error responses. Moreover the EMG was significantly reduced in full error compared with pure-correct responses ($t(1,18) = 10.1$; $p < .01$). Evidently, the EMG associated with partial errors was always smaller in size than full error and pure-correct responses in both time windows ($ps<.0001$).

Online inhibition at the central nervous system level: EEG results for M1

To verify that a global effect on the motor activation is present across the different types of responses, we first computed the LRP. A global decrease in the amount of motor activity for the two types of errors is observed (with a smaller activity for partial errors compared to errors), which could explain the reduction of EMG activity in these conditions, as described here above (see supplementary material for the figure and further details). To gain further insight into the actual nature and direction of these modulatory effects in the motor cortices, the Laplacian transform was computed on the monopolar data. Figure 3 (bottom) shows the temporal dynamics of the Laplacian transformed EEG activities recorded over the contralateral and ipsilateral M1s separately for the three types of responses. The Laplacian horizontal topographies (Figure 3 top) are displayed for three different and successive time points, namely -150 ms, 0 ms and 150 ms from EMG onset (contralateral activities are represented on the left hemisphere, see method).

For all response types, the unimanual command is expressed bilaterally with a negative wave developing over the contralateral M1 prior to the EMG onset and a positive wave developing during the same latency range over the ipsilateral M1. Interestingly, an influence of the response type was observed on the contralateral negative wave (the largest wave for pure-correct responses, intermediate for full errors and the lowest for partial errors) but not on the ipsilateral positive wave. A second interesting feature was the reversal of the polarity between the activities over the M1s starting about 100 ms in partial errors only, which reflects the implementation of a remedial action following the online

detection of a response error (i.e. partial error). This phenomenon was clearly visible when computing the Laplacian map at 150 ms following EMG onset, which happened to occur during the same latency range as the mean CT (158 ms).

The area under the curve was measured at the time window encompassing the peak of the M1 activities (i.e. -150/150ms relative to the onset of the EMG burst), as determined based on the grand-averages (see Figure 3). The baseline was taken between -300 and -150 ms. The ANOVA disclosed a significant effect of response type (pure-correct, full error, partial error responses) on the contralateral M1 ($F(2,36) = 15.9$; $p < .0001$; $\varepsilon=0.8$). Post-hoc paired-samples t tests confirmed that the negativity was larger for pure-correct compared with full error responses ($t(18) = 9.3$; $p<.01$), the latter being larger than partial error responses ($t(18) = 9.2$; $p<.01$). Concerning the ipsilateral M1 activities between -150 and 150 ms, the response type did influence the concurrent positivity ($F(2,36) = 6.5$; $p < .005$; $\varepsilon=0.9$). Post-hoc paired-samples t tests indicated that there was no amplitude difference (positive component) between pure-correct and full error responses ($t(18) = 0.02$; $p = .9$). However this positive activity was smaller for partial errors than both pure-correct and full error responses ($t(18) = 8.9$; $p<.01$ and $t(18) = 7.9$; $p<.05$, respectively).

According to these results, the actual difference between a successful vs. failed interruption of the erroneous action might be ascribed to differences in the negative wave over the contralateral M1 around the time when the incorrect action was initiated. However, to be able to attribute the reduction of the negative wave to online inhibition mechanisms, it was necessary to evidence a significant brain-behavior relationship between the decrease of the negative wave and the speed of the online inhibition. Partial errors were appropriate to test this hypothesis since an index of the speed of the online inhibition (i.e. the correction time, see CT Figure 1C) was available at the single trial level. We reasoned that the efficiency of the inhibitory mechanisms might actually correlate with the speed of the partial error's correction. If this conjecture holds true, we should observe that the faster a partial error was corrected,

the stronger was the corresponding reduction of the negativity over the contralateral M1 at the cortical level (as well as of the afferent EMG activity at the peripheral level).

To test this prediction, we sorted single trial EMG and EEG data depending on CT and used ERP-images for visualization purposes (see method). Figure 4 shows the grand average ERP-images of Laplacian transformed EEGs over the M1s (first row) for incorrect and correct activations (panels A and B corresponding respectively to the cortices contralateral and ipsilateral to the partial error's hand). The second row of Figure 4 presents the ERP images for the incorrect and correct EMGs (panels C and D respectively).

First, the results confirmed that the emergence of the EMG signal was preceded by a negative wave observed over the M1 contralateral relative to the responding hand. This phenomenon was observed for the emergence of the partial error movement (blue activations on figures 4A and 4C surrounding the beginning of the partial error represented by the vertical black line), and for the onset of the corrective action (blue activations on figures 4B and 4D surrounding the beginning of the corrective action shown by the S-shaped black line).

Second, we focused on the activities governing the partial error's emergence and interruption. We observed that the partial error's EMG burst lasted longer with increasing CT (Figure 4C). A similar observation was made at the M1 level: the negative contralateral wave lasted longer with increasing CT (Figure 4A). Moreover, the positive inhibition of the incorrect hand at the time of the partial error also seemed to last longer with increasing CT (Figure 4B).

To corroborate these observations statistically, we performed a median split analysis of the partial error trials depending on the CT and obtained EMG and grand average Laplacian activities for the fast corrected partial errors (Short CTs) vs. the slow corrected ones (Long CTs; see Figure 5).

At the EMG level (Figure 5B), a direct statistical comparison disclosed that the area under the curve for the incorrect EMG (measured between 0 to 150 ms) was smaller for Short CTs than for Long CTs ($t(1,18) = 21.7$; $p < .001$). At the M1 level, areas under the curve differed significantly between Short

and Long CTs both at contralateral and ipsilateral sides in the interval -150/150 ms ($t(1,18) = 4.8$; $p < .05$ and $t(1,18) = 5.5$; $p < .05$, respectively). The contralateral activity was more negative and the ipsilateral activity is more positive in Long CTs than in Short CTs. These effects were driven by the difference in activity in the second part of the interval (i.e. 0/150 ms after the partial error onset). Indeed the interaction between the CT (short versus long) and the interval (-150/0 ms versus 0/150 ms) were significant for both the contralateral and the ipsilateral activities (respectively $F(1,18) = 17.3$; $p<.001$ and $F(1,18) = 8.9$; $p<.01$). Contrasts did not show and difference between Short and Long CTs in the interval -150/0 ms (contralateral: $t(1,18) = 1.3$; $p = .27$; ipsilateral: $t(1,18) < 1$). By comparison, in the interval 0/150 ms the contralateral negativity and the ipsilateral positivity were larger for Long CTs compared to Short CTs ($t(1,18) = 13.1$; $p < .002$; $t(1,18) = 8.9$; $p < .01$, respectively).

To obtain an index of the timing of the error's interruption, we investigated the latency differences between the peaks at Short and Long CTs at both EMG and M1 levels. The latency of the maximum of activity in the incorrect EMG burst appeared earlier in Short CTs compared with Long CTs (38 ms versus 49 ms, respectively, see Figure 5B; $t(1,18) = 12.7$; $p < .005$). The maximum peak of activity over the contralateral M1 appeared significantly earlier for Short than for Long CTs (7 ms versus 19 ms see Figure 5A; $t(1,18) = 9.7$; $p < .01$). Note that the mean difference in the peak latencies between Short and Long CTs was in the same range at the M1 level and at the EMG level (12 ms and 11 ms respectively). Additionally, the maximal activity of the contralateral M1 took place 30.5 ms before the maximal activity of the EMG, which perfectly accorded to the cortico-spinal conduction time (22.4 to 32.4 ms from M1 to dorsal interosseus, as reported in Hess et al., 1987). Hence, these results show that the faster the decrease in the contralateral M1 activation, the faster the decrease of the (corresponding) muscular activity of the partial errors and the earlier the remedial action was engaged. Since the difference in the timing between the decrease of the activities at the M1 and at the EMG level corresponded nicely to the time necessary to conduct the information along the corticospinal tract, we

can conclude with high confidence that the reduction of the EMG activity was primarily driven by the contralateral M1 cortex.

Altogether, these results indicate that the contralateral M1 was crucially involved in online inhibition mechanisms. Presumably, the online inhibition observed at the M1 level was reflecting the output or consequences of an upstream motor or premotor area that directly gated the amount of inhibition eventually instantiated in M1s.

Online inhibition driven by premotor sources acting on the M1s

To gain insight into the possible involvement of upstream premotor regions during the implementation of online inhibition mechanism, we analyzed EEG activities likely generated in ACC, SMA and preSMA, all of which have been associated with cognitive control processes (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Nachev, Kennard, & Husain, 2008). We tested the prediction that a partial error might occur and be corrected if an upstream premotor effect (presumably unlocking and guiding the online inhibition in M1) arrives on time, but that a response error is inadvertently committed if the premotor signal occurs too late in order to stop the unfolding of the incorrect action. To test this prediction, we looked selectively at premotor activity, whose electrophysiological properties (latency and amplitude) are compatible with such fast premotor-motor loops. Specifically, the following requirements need to be fulfilled: (i) be present in full error and partial error responses but not in pure-correct responses, (ii) show a latency or amplitude difference between partial error and full error responses, consistent with the success or the failure of the inhibition; and (iii) show a pattern of activity for partial error responses that is consistent with the difference between a fast vs. a slow correction.

Ne/ERN at FCz

Because the Ne/ERN, presumably originating from the ACC, is an early response-related ERP component reflecting error detection (Falkenstein et al., 1991; Gehring et al., 1993), we tested whether this deflection might reflect the precursor of online inhibition mechanism or not. Specifically, we assessed whether the amplitude and the latency of the Ne/ERN were related to the negativity over the

contralateral M1. Such an outcome would be consistent with an interruption signal that prevented the unfolding of the incorrect action. The amplitude and latency of the Ne/ERN were analyzed at FCz (see Figure 6A), following standard practice (Debener et al., 2005; Frank, Woroch, & Curran 2005; Gentsch, Ullsperger, & Ullsperger, 2009). Although the amplitude of the Ne/ERN appeared to be larger during partial errors than during full errors, this difference did not reach significance (0.79 $\mu V/cm^2$ and 0.73 $\mu V/cm^2$ respectively, $t(1,18) < 1$). Nonetheless and as expected, both reliably differed from pure-correct responses (0.42 $\mu V/cm^2$, $t(1,18) = 31$; $p < .0001$ and $t(1,18) = 33$; $p < .0001$ respectively), with a larger negative component for errors relative to correct responses. In terms of peak latency, the mean latencies of the Ne/ERN peak were 128 ms, 142 ms and 156 ms following EMG onset for partial error, pure-correct and full error responses, respectively. Peak latencies differed significantly between full errors and partial-errors ($t(1,18) = 33$; $p < .0001$), whereas the difference approached significance when comparing partial errors to pure-correct responses, or full errors to pure-correct responses ($p = 0.09$ and $p = 0.07$, respectively). Importantly, since the unfolding of the online inhibition mechanism was already observed at the EMG and M1 levels during the first tens of milliseconds following the EMG onset for partial errors (see Figure 5), the Ne/ERN peaked clearly too late in such a way to play a significant proactive role in this online inhibition of the erroneous response.

Early onset negativity at Fz

An EEG component likely generated by SMA or preSMA and developing prior to the response, i.e. the N-40 ("*N minus 40*") which peaks 40 ms before EMG onset, has previously been associated with response selection (Burle et al., 2004; Vidal et al., 2011). Accordingly, we were wondering whether such an early activity, prior to response initiation, is also observed in situations where the participant has to stop the erroneous action. This assumption appears plausible in light of the fact that preSMA has been associated with inhibition of the unwanted action and/or the reprogramming of actions in

situations where a sudden change of correct response was required (see Isoda & Hikosaka, 2007, for primate neurophysiology evidence; Mars et al., 2009, for human TMS evidence).

Visual inspection of the waveforms at Fz (Figure 6B), anterior to the FCz electrode studied above, suggested that the slowly developing negative activity was sensitive to response type. The ANOVA performed on the amplitude (calculated between baseline and the most negative peak between 0 and 300 ms) showed a significant effect of response type ($F(2,36) = 18.1$, $p < .0001$; $\varepsilon=1$) with amplitudes observed at -4.7, -3.9 and -1.8 $\mu V/cm^2$ for partial errors, full errors and pure-correct responses respectively. Contrasts showed a lower amplitude for pure-correct response compared with partial errors and errors ($t(1, 18) = 31.9$, $p < .0001$ and $t(1, 18) = 22.9$, $p < .0001$, respectively) and no difference between partial errors and full errors ($t(1, 18) = 2.0$, $p = .18$).

Importantly, the waveforms developed earlier for pure-correct and partial error responses compared with full errors. Statistically this was confirmed by slope analyses that tested whether the slopes were significantly different from zero using steps of 100 ms starting 300 ms prior to the EMG onset and ending 300 ms after this EMG onset. Slope values were estimated by computing a linear regression for each time window separately and were compared to zero. Table 2 shows the *t* (and p) values for each time window. The slopes became significantly negative starting -200 ms before the EMG onset for pure-correct responses and partial errors, whereas it was the case only starting from the EMG onset for full errors. Moreover, the slope remained negative for a longer period for partial errors (from -200 to +100 ms) compared with pure-correct responses (from -200 to 0 ms) and full errors (from 0 to +200ms).

To sum up, these results showed that the negativity developed earlier for partial-errors and pure-correct responses compared with full errors. Hence, these findings were compatible with our hypothesis that this premotor effect might actually correspond to an upstream proactive cognitive control effect signaling the urge to inhibit an incorrect action. In this framework, the slightly delayed premotor activation for full errors could account for the failure to inhibit the incorrect action.

To be meaningfully associated to online inhibition mechanisms, it was crucial to show that the slow negative wave developed earlier in partial errors trials with a fast correction, compared to partial error trials with a slow correction. Following the same logic as with EMG and M1 activities described above, we analyzed the Laplacian-transformed EEG data as a function of CT. Figure 7 shows the waveforms separately for the rapidly corrected partial errors and the slowly corrected partial errors, computed on the basis of a median split. First, we did not find any difference in the amplitude between short and long CTs ($t(1, 18) = .06$, $p = .81$). Second, slope analyses on this negative wave (see table 3) revealed a delay in the development of the negative wave as a function of the CT. For partial error trials with short CTs, the negative wave started to develop earlier (from -200 to -100 ms) than for trials with long CTs (from -100 to 0 ms).

In sum, these results show that the premotor negative activity recorded from electrode Fz during partial errors paralleled both the unfolding of the EMG activity at the effector level and the online-inhibition expressed at the contralateral M1 level. As such, this premotor effect qualifies for a plausible source of the online inhibition mechanism expressed in M1 (and detected equally well at the EMG level) during partial errors.

Discussion

In this study, we characterized the electrophysiological correlates of the capacity to block the execution of an incorrect action online and to swiftly switch to the correct one. To address this question, we recorded scalp EEG concurrently with peripheral EMG to track the precise electrophysiological time-course of this online inhibition mechanism, capitalizing on partial errors committed during a Flanker task. Although a previous experiment already examined the temporal dynamics of partial errors using EMG and EEG methods (Burle et al. 2008), our study, using a spatially enhanced Laplacian technique, is the first to provide a detailed electrophysiological characterization of the temporal unfolding of the online inhibition process during partial errors within M1, looking separately at possibly different

contralateral vs. ipsilateral contributions of the primary motor cortex. Results of our study show clear-cut inhibition-related effects in contralateral M1. Moreover, we were able to gain insight in the role of upstream premotor regions on the generation of this inhibitory control signal. Our findings demonstrate that the Ne/ERN, likely generated within the ACC following error detection, does not causally trigger the inhibitory mechanisms as it is taking place too late in order to do so. Rather, our results suggest that premotor areas (either the SMA or preSMA) likely implement the inhibitory signal in M1 in a proactive manner, allowing the prevention of error commission through enhanced cognitive control during action monitoring. Our results are thus consistent with a premotor (source) - motor (consequence) sequence during the online implementation of remedial action while monitoring the content of self-generated actions. Below, we discuss the implications of these results in greater detail.

Online inhibition takes place within the contralateral M1

Our results show that the negative wave recorded over the contralateral M1 was substantially reduced in amplitude immediately after the initiation of the response for both full and partial errors, compared to correct responses. As a matter of fact, the strongest decrease was evidenced for partial errors, paralleling the amplitude differences found in the EMG. Furthermore, by sorting partial errors trials as a function of the CT, a close relationship between the temporal dynamics of this blunted negative component within contralateral M1 and the substantial reduction of the corresponding EMG activation was clearly evidenced. Moreover, the short delay between the two peak latencies at the M1 vs. EMG level was perfectly in line with the average cortico-spinal conduction time (e.g. Hess, Mills, & Murray, 1987), reinforcing the neurophysiological validity of our new finding. Thus, these results unambiguously indicate that this specific amplitude reduction found at the contralateral M1 level during partial errors was associated with a lower activation of the muscles responsible for the execution of the incorrect action at the peripheral level. In light of this, we can conclude that contralateral M1 is the cortical site where the online inhibition of the non-desired action presumably takes place. A recent study investigating cortico-spinal excitability with TMS pulses to contralateral M1 during a stop task

(van den Wildenberg et al., 2010) is informative in this respect. These authors found that intracortical $GABA_B$ergic circuits (reflected by the duration of the silent period) were involved within M1 in stop-inhibit trials. Drawing a parallel with these recent TMS findings, we surmise that similar intracortical inhibitory circuits are likely responsible for the decrease of the negativity over the contralateral M1 observed in our study. Nonetheless, given that M1 lies at the top of the cortico-spinal tract, we cannot exclude the possibility of additional loci, located downstream relative to M1 and not generating measurable scalp ERP activities, where this online inhibition actually takes place.

As outlined in the introduction, the unimanual motor command in a bimanual choice RT task is not only expressed over the contralateral motor cortex but also ipsilaterally in the form of a clearly distinctive inhibitory activity (Burle et al., 2004). This activation/inhibition pattern can be seen as the electrophysiological correlate of the set of neurons belonging to the response layer described in decision making models. This response layer contains sets of neurons (units) that accumulate evidence in favor of one of the (two) competing responses. In these models, the architecture is designed in such a way that the inhibition of the undesired alternative response is implemented. However, two types of models differing in the way they actually implement inhibition have been proposed in the literature. Models favoring lateral inhibition assume that the inhibition is driven by the response units themselves (Usher & McClelland, 2001). By contrast, feed-forward inhibition models bear that the inhibition is driven by units located upstream from response level (Heuer, 1987). Our new results are coherent with this second view. As a matter of fact, lateral inhibition models would predict a proportional decrease of the ipsilateral positivity (inhibition) with the decrease of the contralateral negativity in the different trial types (pure-corrects, full errors and partial errors), which is not observed in the present case however. Moreover, converging physiological evidence showed that such an ipsilateral inhibition has a functional role in preventing errors to occur and is not simply reflecting motor processes related to the activation of the response. First, this ipsilateral inhibition cannot be accounted for by a “hardwired” connection via transcallosal inhibitory connections, because it does not occur when a response has to be selected from

two responses with the same hand (Meynier, Burle, Possamaï, Vidal, & Hasbroucq, 2009). Second, the positivity on the ipsilateral M1 is largely reduced or even absent when there is no need to inhibit the ipsilateral cortex: i.e. in simple RT tasks (Burle et al., 2004; Vidal et al., 2011) or in spontaneous internally-driven movements (Ikeda et al., 1995).

According to the argumentation outlined here above, we do not consider the activation/inhibition pattern as the neurophysiological marker of the accumulation of evidence (in favor of one out of the two competing responses). Rather, the contralateral activation is seen as the motor command that is sent to the structures downstream relative to M1, while the ipsilateral inhibition is seen as a guard against the execution of the undesired response. This inhibition might stem from motor control mechanisms preventing error commission. On the other hand, the intraparietal sulcus appears to be a plausible candidate area for the locus where the accumulation of evidence takes place (Huk & Shadlen, 2005). Interestingly, the presence of the ipsilateral inhibition of the non-responding hand is informative for the type of motor command likely engaged in the action. An absent ipsilateral inhibition would mean that the type of the motor command is similar to the ones engaged in simple RT task and spontaneous movement. By contrast, the presence of the ipsilateral inhibition in errors and correct trials would mean that the participant did somehow select the incorrect response. Our new results support this last interpretation: the motor command is expressed bilaterally in both erroneous actions (partial and full errors) and in correct responses alike. Hence, this result implies that response errors are not simply due to uncontrolled response activation but are rather the result of a decision process.

Another interesting issue is whether online inhibition is selective for the effector involved in the erroneous response or whether all the effectors are equally affected. Following the first scenario, we should observe a decrease of activity in full errors compared to correct responses only over the contralateral M1, but not over the ipsilateral M1. On the other hand, following the other alternative, both over the ipsilateral and contralateral M1s the activities should become more positive for full errors. Interestingly, we did not find any change in the positivity over the ipsilateral M1 between full errors and

correct trials, a result which suggests that the inhibition is selective to the effector involved in the error. Consequently, beyond the capacity to detect that the action is wrong in the course of its planning and execution, brain systems can also accurately identify the response that has to be inhibited. On top of that, such a mechanism appears to be necessary to efficiently prepare the correction of incorrect actions, like it is the case for incorrect-correct trials.

In sum, our results demonstrate that M1 is the cortical structure via which online inhibition is exerted. Neural activity recorded from M1 also provides critical temporal information regarding the onset of the online inhibition, both at the central and peripheral levels. The use of these remarkable temporal characteristics enabled us to search for non-overlapping brain regions that might proactively guide and tailor this online inhibition mechanism during partial errors, presumably at an earlier latency than in M1.

The Ne/ERN and online inhibition of the incorrect action

A large number of fMRI and electrophysiology studies have associated the ACC to cognitive control functions (Falkenstein et al., 1991; Gehring et al., 1993; Botvinick, Braver, Carter, Barch, & Cohen, 2001; Ridderinkhof et al., 2004; Rushworth, Buckley, Behrens, Walton, & Bannerman, 2007). In a majority of them, this structure is considered as a key component in a chain of functional areas that enable behavioral adjustments to take place, from one trial to the next with the purpose of optimizing performance (Carter et al., 1998; MacDonald, Cohen, Stenger, & Carter, 2000; Ullsperger & von Cramon, 2001; Kerns et al., 2004). For instance, the conflict monitoring theory postulates that the role of the ACC reveals the need to increase cognitive control resources by detecting a conflict between responses (Botvinick et al., 2001). The dorsolateral prefrontal cortex (DLPFC) receives this information and orchestrates a reorganization of attentional resources to prevent errors to occur again during the next trials. Interestingly, other studies attempted to evaluate whether the ACC is also related to the online adjustments in control. To do so, the Ne/ERN, considered as the electrophysiological correlate of the ACC activity recorded with fMRI during errors (Van Veen & Carter, 2002; Debener et al., 2005),

was thought to be responsible for the initiation of the correction of overt errors that have just been made. Results were not unequivocal however. In some studies, the latency and/or the amplitude of the Ne/ERN were found to be predictive of the speed of the remedial action after error commission (Gehring et al., 1993; Falkenstein et al., 1994), but in some studies this link was not found (Rodríguez-Fornells et al., 2002). However, these studies did not investigate successful online adjustment mechanisms since they analyzed overt error trials for which a correction was made just after the wrong key press. Obviously, correcting an overt error involves the programming of the remedial action but does not necessarily require the inhibition of the erroneous action. The partial errors in our study guarantee that both inhibitory activity and the remedial action had taken place. In line with previous findings (Rodríguez-Fornells et al., 2002, Burle et al., 2008), our results show that the latency range of the Ne/ERN occurs later than the time window during which the online inhibition takes place in contralateral M1. Hence, it turns out that if the Ne/ERN may very well exert an adaptive influence on the next trial (e.g., post-error slowing; see Gehring et al., 1993; Debener et al., 2005; Marco-Pallarés, Camara, Münte, & Rodríguez-Fornells, 2008; or adjustments of selective attention; see Maier, Yeung, & Steinhauser 2011) it is however relatively too slow in order to regulate more immediate and rapid online behavioral adjustments during the course of the action itself. Accordingly, the rapid detection of the need to inhibit the action in the course is driven by another mechanism which does not seem to include activity from the ACC. Rather, the Ne/ERN seems to reflect a reactive mechanism. The fact that the Ne/ERN peaked earlier during partial error than during full error trials might be the consequence of a more efficient on-line cognitive control mechanism driven by an upstream region in the network, leading to a faster evaluation of the outcome by the ACC during partial errors than during full errors. This view suggests the presence of an additional region distinct from the ACC but working in concert in order to guarantee the most efficient behavior (for the current and the following trials).

The slowly developing negative wave over Fz and online inhibition of the incorrect action

If the ACC, and the Ne/ERN as its electrophysiological correlate, does not incorporate the neural mechanism through which the online inhibition is imposed within the contralateral M1, the question arises as to which alternative mechanism might entail this adaptive control process. Interestingly, our results reveal a premotor frontal mechanism as a plausible candidate for the modulatory effect in M1 during action monitoring. Over the medial prefrontal region covered by the Fz electrode, we found a slow-developing negative wave with a similar morphology for partial and full errors, though with important differences in their respective time courses. In particular, this premotor activity started earlier for partial errors than for full errors. Moreover, a similar negativity was also observed for correct trials, but with a smaller amplitude compared to the incorrect responses. Additional median split analyses as a function of CT showed that this activity arose earlier in the case of fast corrections during partial errors relative to slower corrections. Remarkably, we found that the speed at which this negative wave developed predicted the capacity to exert inhibitory control over the incorrect action, reinforcing the notion that this activity was somehow related to an upstream and proactive cognitive control signal guiding the online inhibition within the (contralateral) M1. In principle, one could argue that the development of the slow negative wave reflects conflict (see Botvinick et al., 2001) and/or the preparation of the remedial action, rather than the inhibition itself. This would be in line with the fact that this component developed earlier in trials where the correction was faster (Figure 7). First according to the definition of conflict provided in Botvinick et al. (2001), conflict emerges when the responses in competition are activated at the same time. Hence, the conflict theory cannot explain the presence of the negativity developing before the partial error in incorrect-correct trials since the remediation has not started yet. Second, the conflict theory as well as the alternative interpretation in terms of preparation of remedial action can be ruled out given that this component was also present during errors trials where there was no sign of remedial action, leaving an interpretation in terms of online inhibition as the most likely explanation.

Based on the literature and the electrode position, i.e Fz, where this proactive control effect was observed, one may suggest the preSMA as the main generator of this slow negativity. Results of a recent study using single cell recordings in rhesus monkeys showed that the preSMA is directly engaged in the active suppression of unwanted actions (Isoda & Hikosaka, 2007). Using an oculomotor switching task, monkeys occasionally had to switch their motor behavior from an automatic to a more controlled response mode. PreSMA neurons were found to discharge during these switch trials, but not during non-switch trials. Interestingly, the authors showed that the switch turned out to be successful if the firing of these neurons preceded the initiation of the incorrect and unwanted action, while the switch actually failed when these preSMA neurons started firing after the beginning of the incorrect action. These electrophysiological properties are perfectly in line with the slow negative wave over Fz reported in our study, as well as with our interpretation that the timing of the wave recorded at Fz is a crucial determinant for either a successful remedial action (i.e. partial errors) or a failure to prevent the incorrect response and switch to the correct one (i.e. full errors). This negative slow wave at Fz clearly started before the EMG onset during successful inhibition of errors (i.e. partial errors) but only after the EMG onset during full errors (i.e. failure to impose inhibition over the incorrectly initiated motor action).

An important difference between the study of Isoda & Hikosaka (2007) and our study has to be noted, nonetheless. In their study, inhibitory mechanisms were "externally" guided by a cue. By contrast, in our study, the inhibition was "internally" generated. Despite these differences, it is interesting to note the obvious similarities between our new results and these earlier monkey neurophysiology data, showing each time a cascade effect from the early involvement of the preSMA in inhibition to the actual change and consequences of this proactive cortical effect at the muscular/output level. The same remark can be done with protocols investigating inhibition in stop signal tasks with humans and where an increased activity in the preSMA and the right inferior frontal gyrus were found (Aron et al., 2004; Rubia et al., 2001). According to these studies, it is conceivable that the negative slow wave that we described in our study is

reflecting the command to inhibit the motor efference at the M1 level. Future research is needed however in order to clarify which factors may facilitate or instead hamper the implementation of this ultra-fast and labile inhibition command generated within the premotor cortex during the course of the action. In this context, the direct comparison between internally versus externally driven inhibition appears as a promising line of research in future investigations, especially with patient populations showing inhibition deficits or breakdowns of impulse control. Finally, as a caveat, it appears important in future studies to disentangle inhibitory control for congruent from incongruent trials when interference/Flanker tasks are used (as in the present case). Presumably, dissociable inhibitory control processes might be at play in these two cases (i.e., fast guesses or anticipations for congruent trials, as opposed to a genuine conflict monitoring process in the case of incongruent stimuli).

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Author Note

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Tables

Table 1: Mean values of the reaction Times (RTs) in milliseconds for Pure-Correct Trials, Full error Trials, and Incorrect-Correct trials, fractioned into the premotor time (PMT) and the motor time (MT) separately for each trial type; and additionally into the incorrect activation time of the partial error (IAT) and the correction time (CT) for incorrect-correct trials.

Table 2: Values of the measured slopes at Fz electrode, separately for each time period included in the analysis. Laplacian estimated from spherical spline interpolation ($\mu V/cm^2/s$). t values for the comparison to zero of these slopes are provided in brackets and the corresponding level of significance is shown in parentheses.

Table 3: Values of the measured slopes at Fz electrode in each tested time period. Laplacian estimated from spherical spline interpolation ($\mu V/cm^2/s$). t values for the comparison to zero of these slopes are in brackets and the corresponding level of significance is in parentheses.

Figure Captions:

Figure 1: Representative examples of the three types of trials: (A) a pure-correct trial, (B) a full error trial and (C) an incorrect-correct trial. EMG activities of the muscles involved in the correct and the incorrect responses are represented in black and in gray respectively. Time zero corresponds to the presentation of the target. The solid lines indicate the moment when the response was registered by the touch screen. The dotted lines indicate the onsets of the EMGs for the three types of activation (i.e. correct, incorrect and partial error). PMT = pre-motor time (i.e. time interval between the target presentation and EMG onset of the activation leading to the response); MT (i.e. time interval between EMG onset of the activation leading to the response and the response). Only in incorrect-correct trials: IAT = Incorrect Activation Time (i.e. time interval between the target presentation and EMG onset of the partial error); CT = correction time (i.e. time interval between the EMG onset of the partial error and the EMG onset of the correct activation).

Figure 2: Averaged rectified EMG activities, time-locked to EMG onset for pure-correct, full error and for partial error responses in thick, thin and dashed lines respectively.

Figure 3: Grand-averaged Laplacian activities over M1s (bottom) and the corresponding Laplacian horizontal topographies (top), separately for the three types of responses (pure-correct, full error and partial error responses). The graph (bottom) shows the time course of the activities locked to the EMG onset recorded above the contralateral vs. ipsilateral M1 to the responding hand (respectively shown in blue and red). Pure-correct, full error and partial error responses were displayed respectively in thick, thin and dashed lines. The baseline interval spanned from -300 ms to -150 ms. the topographical maps showed a top/horizontal view of the scalp (nose up) with the actual distribution of the Laplacian-transformed EEG data, separately for each response type (pure-correct/top; full error/middle; and partial

error/bottom) and at three successive time points relative to the EMG onset (-150ms, 0ms and 150ms). Contralateral activities were represented on the left hemisphere (see method).

Figure 4: ERP-images of the Laplacian EEG and EMG activities (grand averages) for the partial errors as a function of the correction time (CT, see Figure 1). Time zero corresponds to the partial error onset. The trials were sorted as a function of the CT, represented by the time interval between the vertical black line and the S-shaped line (EMG onset of the partial errors and EMG onset of the remedial actions). The incorrect and correct response activations are shown for the cortical level by Laplacian EEG activities over the M1s (top row) and for the peripheral/EMG level (bottom row). Panel A shows the incorrect activation registered over the contralateral M1 relative to the partial error hand (ipsilateral M1 relative to the remedial action hand), its muscular output is shown at the incorrect EMG level (panel C). Panel B shows the correct activation over the ipsilateral M1 relative to the partial error hand, which is also the contralateral M1 relative to the remedial action hand. Its muscular output is shown at the correct EMG level (panel D).

Figure 5: Laplacian EEG data over the M1s and EMGs during partial errors. A median split analysis was performed based on the CT (see Figure 1). Rapidly corrected partial errors ("Short CT" in continuous line) vs. slowly corrected partial errors ("Long CT" in dotted lines) were grouped. Time-zero corresponds to the partial error onset. (A) Laplacian-transformed EEG activities over the contralateral (gray lines) vs. ipsilateral (black lines) M1s to the partial error's hand. (B) Incorrect vs. correct EMGs (gray and black lines, respectively). The digital values on the graphs indicate the mean latency of the peaks corresponding to the condition towards which the arrow points.

Figure 6: Laplacian-transformed EEG data at electrodes FCz (A) and Fz (B) for pure-correct, full error and partial error responses. The time-zero corresponds to the EMG onset. (A) The negativity,

considered as the Ne/ERN, peaked between 100 and 150 ms as a function of the response type. Its amplitude was larger for partial-errors and full errors compared with pure-correct responses. (B) A slow negative activity starting before the EMG onset and increasing until 200 ms after the EMG onset was visible. The moment of start and the amplitude differed as a function of the response type. The negative wave started about 200 ms before the EMG onset for pure-correct and partial error responses and started later for full error trials. Its amplitude was larger for the two types of errors and smaller for the correct trials.

Figure 7: Laplacian-transformed waveforms at Fz separately (median split analysis) for the trials with short CTs (solid line) and long CTs (dotted line). Time zero corresponds to the partial error onset.

Figure 1

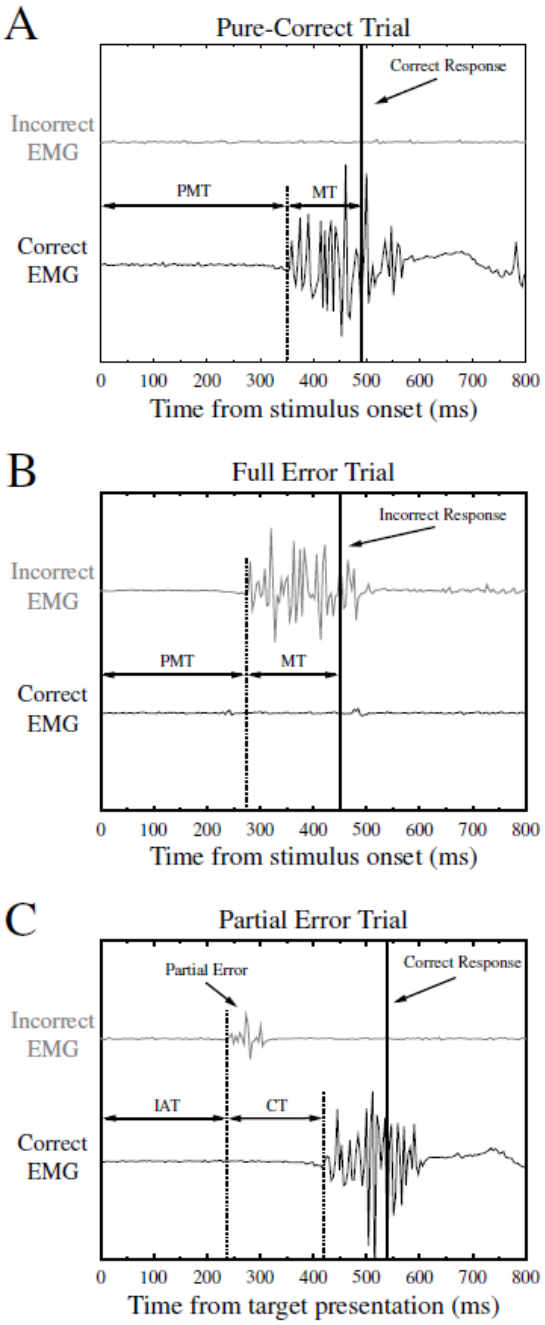

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Figure 1

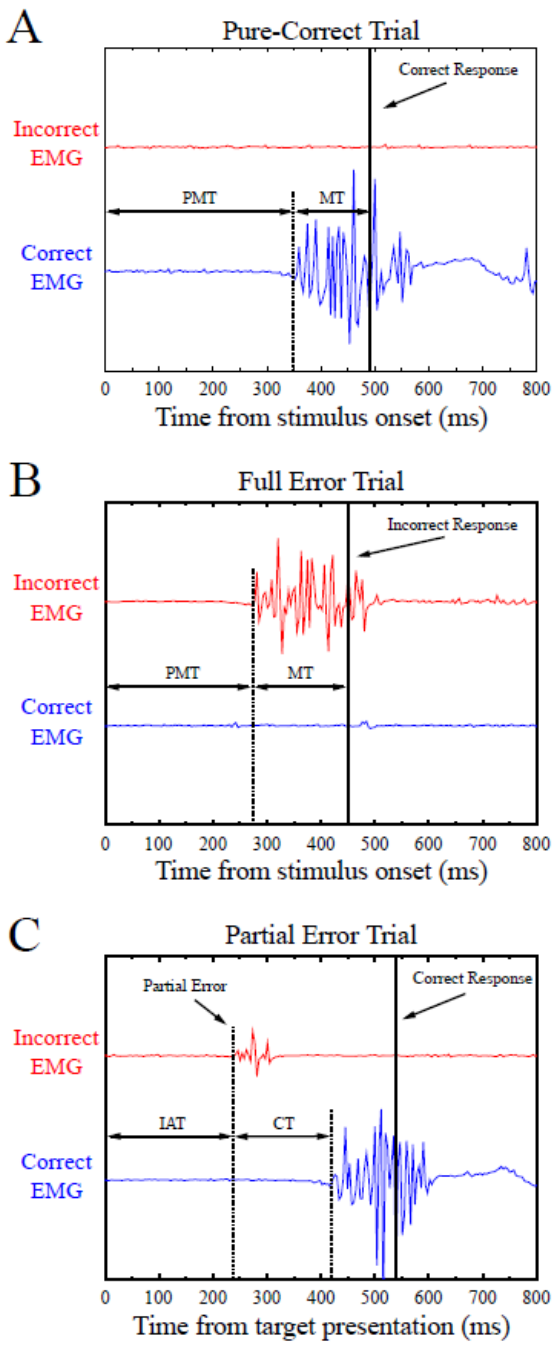
A
Pure-Correct Trial
Correct Response
Incorrect EMG
PMT
MT
Correct EMG
0 100 200 300 400 500 600 700 800
Time from stimulus onset (ms)
B
Full Error Trial
Incorrect Response
Incorrect EMG
PMT
MT
Correct EMG
0 100 200 300 400 500 600 700 800
Time from stimulus onset (ms)
C
Partial Error Trial
Partial Error
Correct Response
Incorrect EMG
IAT
CT
Correct EMG
0 100 200 300 400 500 600 700 800
Time from target presentation (ms)

Figure 2

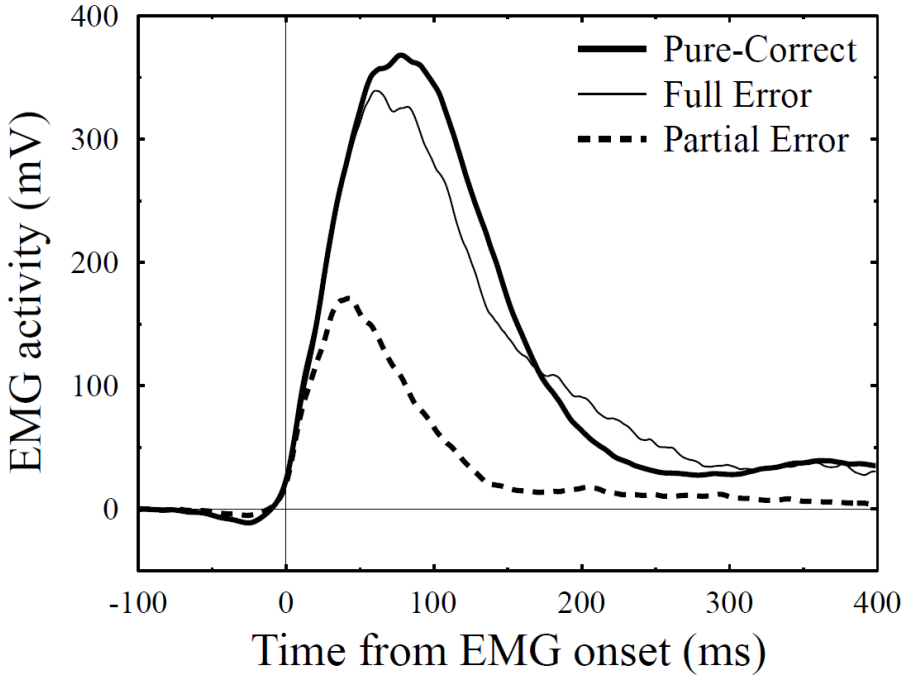

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Figure 3

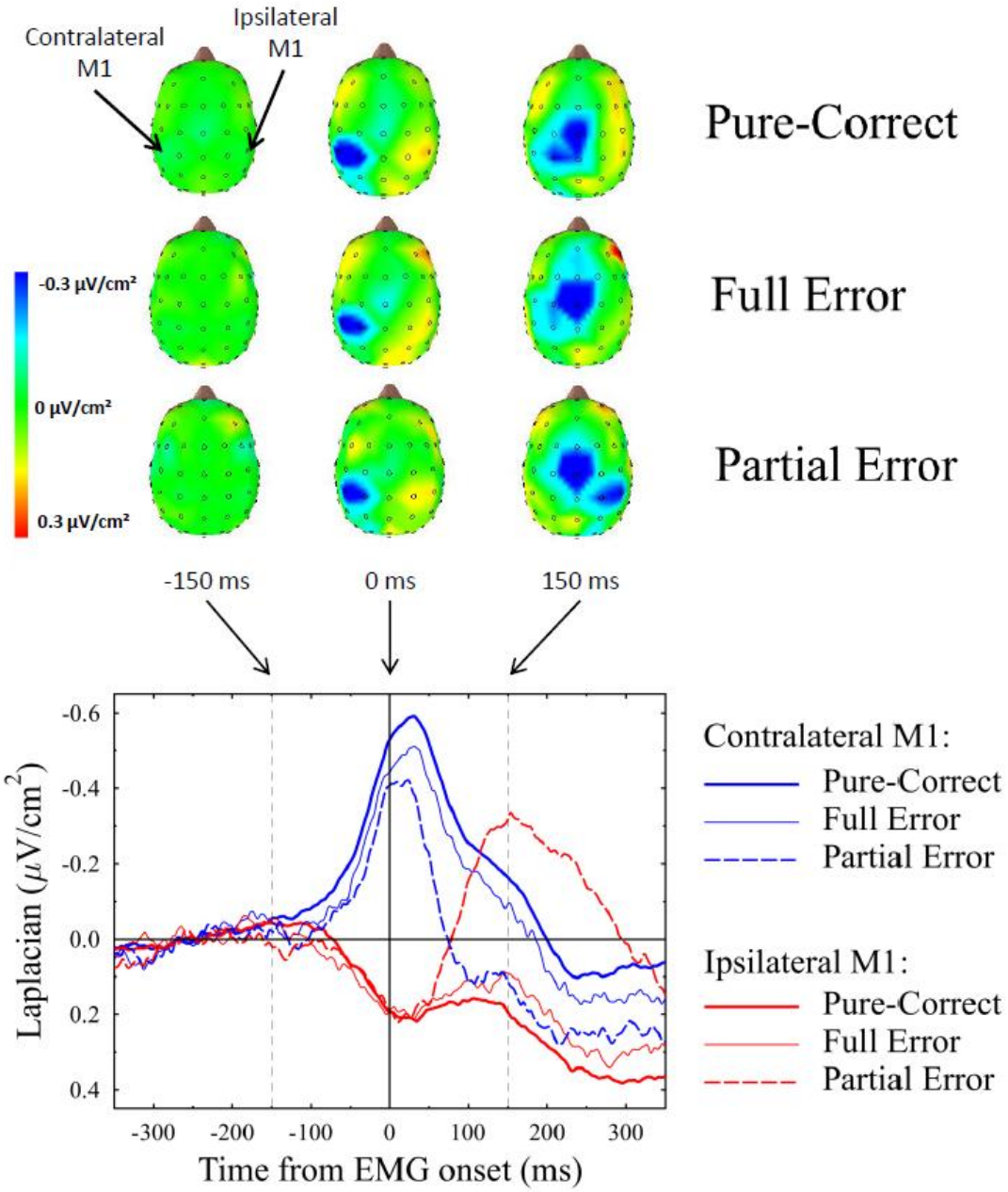

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Figure 4

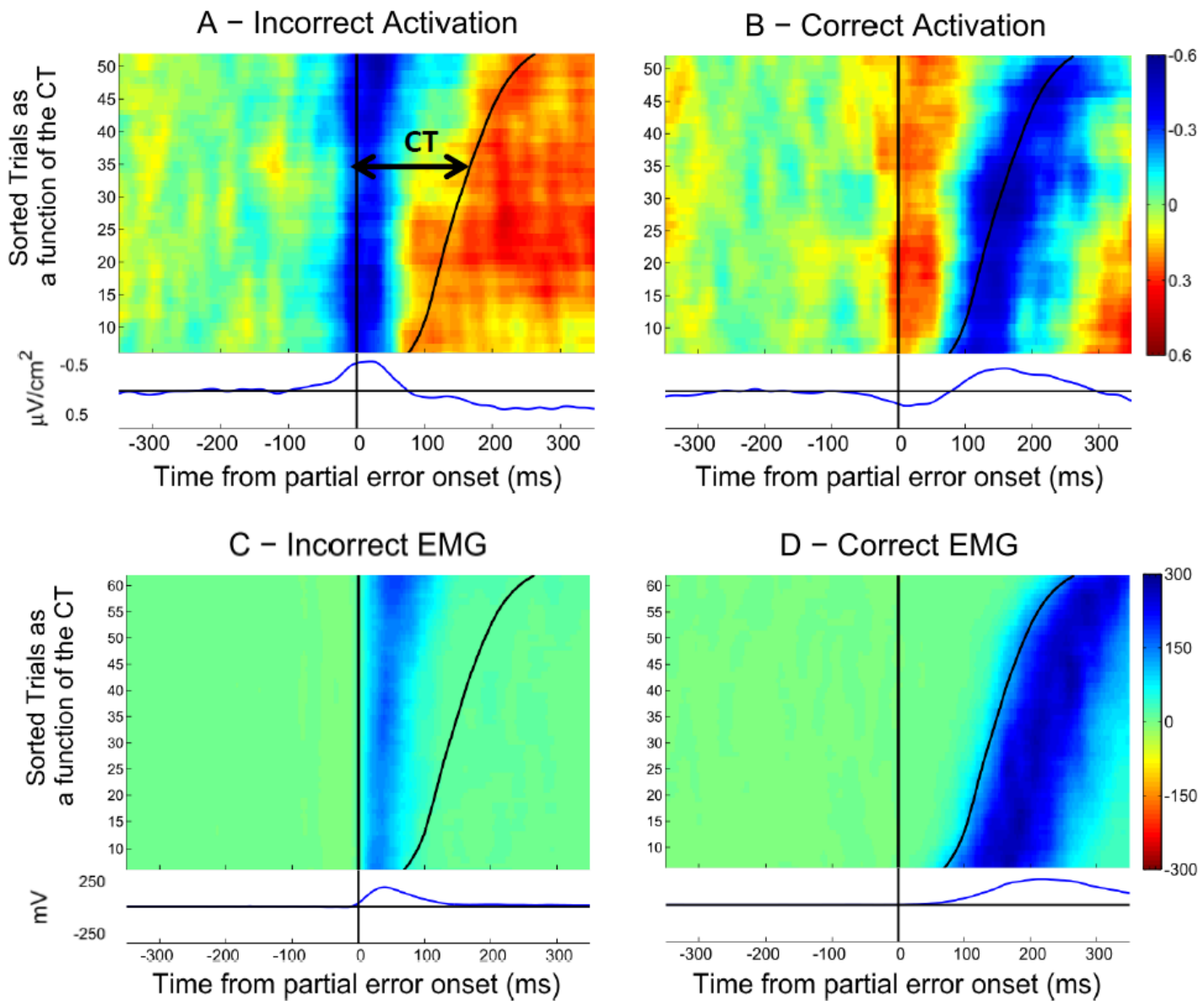
A − Incorrect Activation
B − Correct Activation
C − Incorrect EMG
D − Correct EMG
Sorted Trials as a function of the CT
CT
μV/cm²
mV
Time from partial error onset (ms)
-300 -200 -100 0 100 200 300

Figure 4: ERP-images of the Laplacian EEG and EMG activities (grand averages) for the partial errors as a function of the correction time (CT, see Figure 1). Time zero corresponds to the partial error onset. The trials were sorted as a function of the CT, represented by the time interval between the vertical black line and the S-shaped line (EMG onset of the partial errors and EMG onset of the remedial actions). The incorrect and correct response activations are shown for the cortical level by Laplacian EEG activities over the M1s (top row) and for the peripheral/EMG level (bottom row). Panel A shows the incorrect activation registered over the contralateral M1 relative to the partial error hand (ipsilateral M1 relative to the remedial action hand), its muscular output is shown at the incorrect EMG level (panel C). Panel B shows the correct activation over the ipsilateral M1 relative to the partial error hand, which is also the contralateral M1 relative to the remedial action hand. Its muscular output is shown at the correct EMG level (panel D).

Figure 5

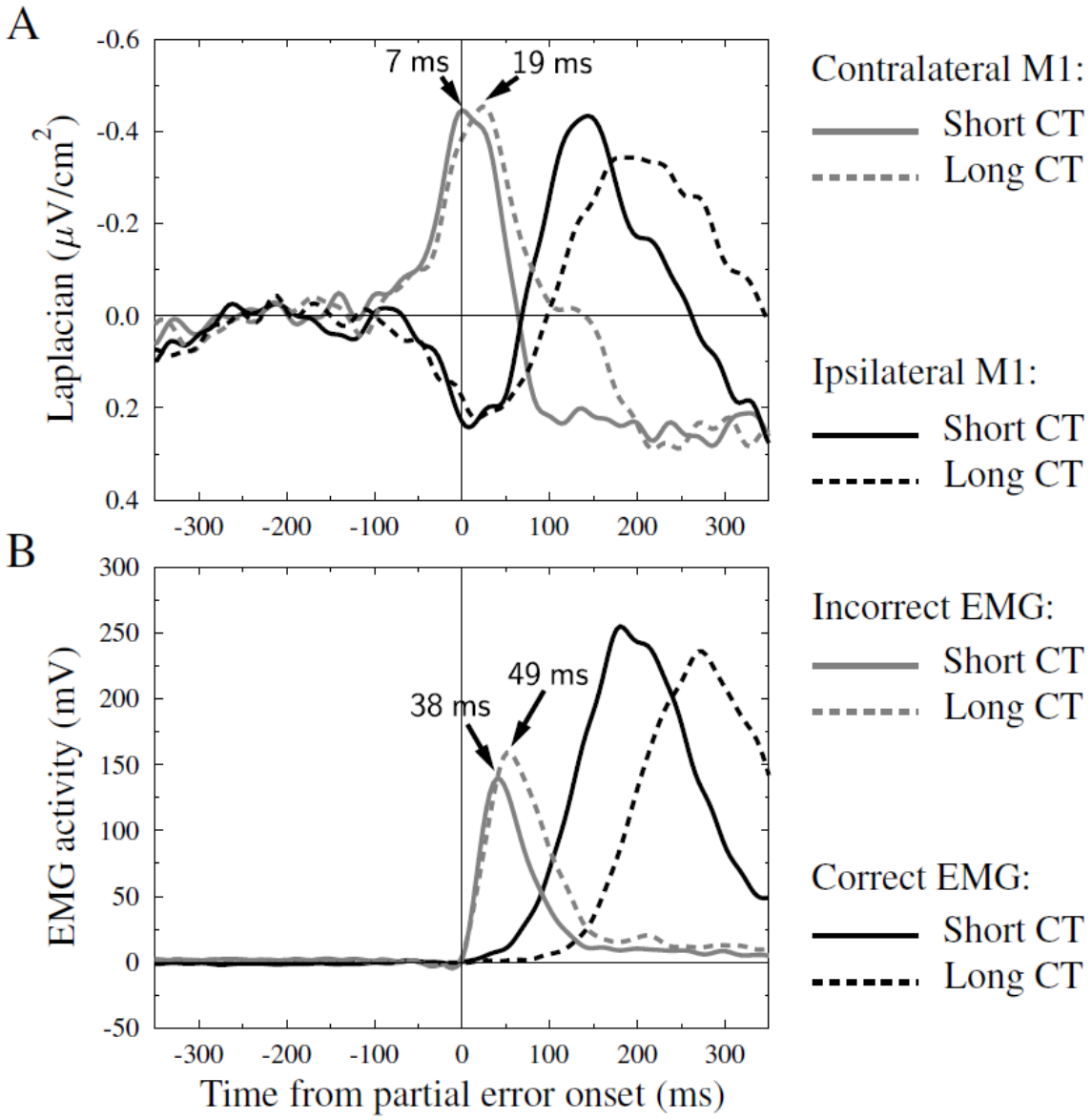

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Figure 5

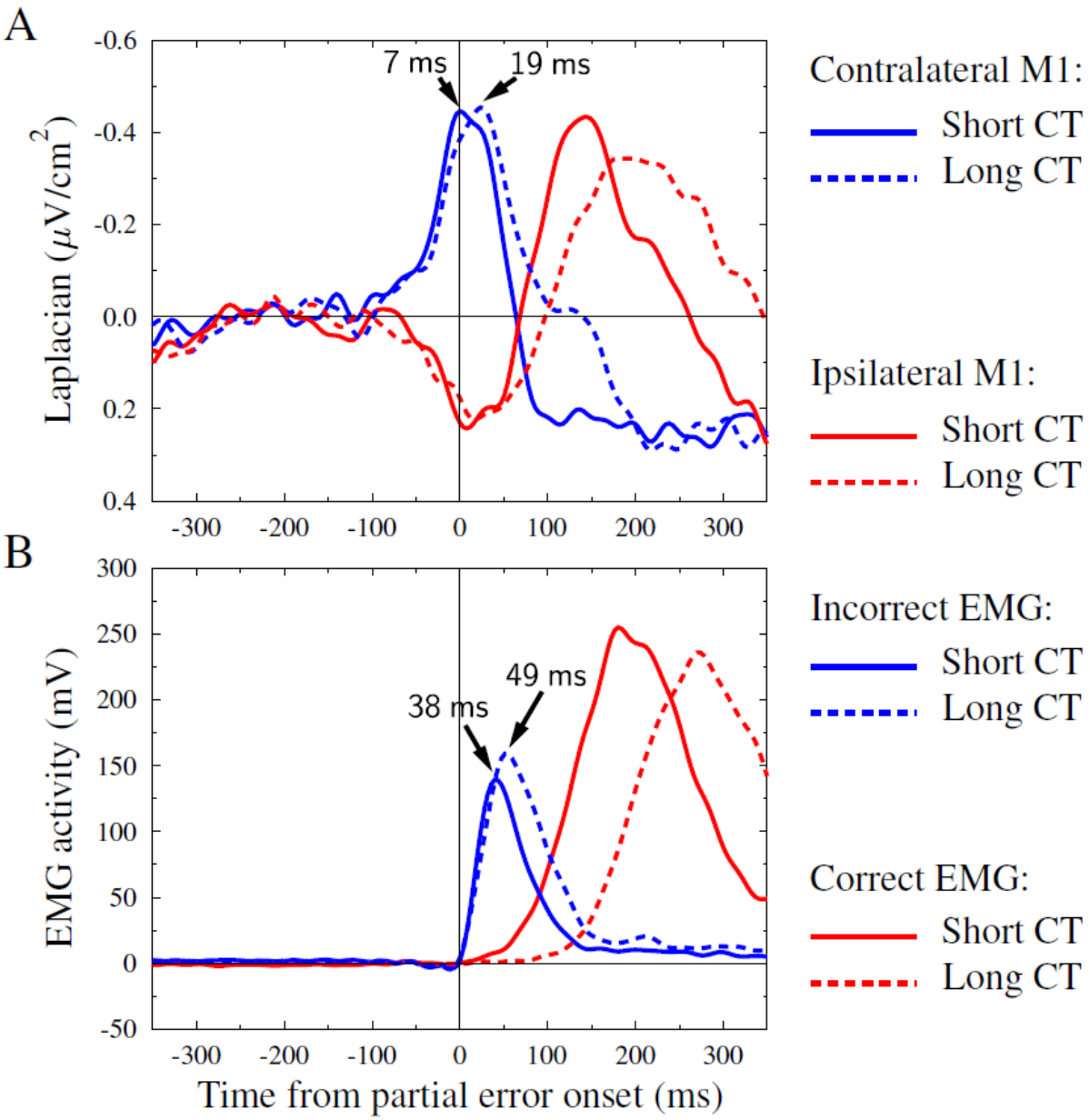

A
B
7 ms
19 ms
38 ms
49 ms
Laplacian (μV/cm²)
EMG activity (mV)
Time from partial error onset (ms)
-0.6
-0.4
-0.2
0.0
0.2
0.4
300
250
200
150
100
50
0
-50
-300
-200
-100
0
100
200
300
Contralateral M1:
Short CT
Long CT
Ipsilateral M1:
Short CT
Long CT
Incorrect EMG:
Short CT
Long CT
Correct EMG:
Short CT
Long CT

Figure 6

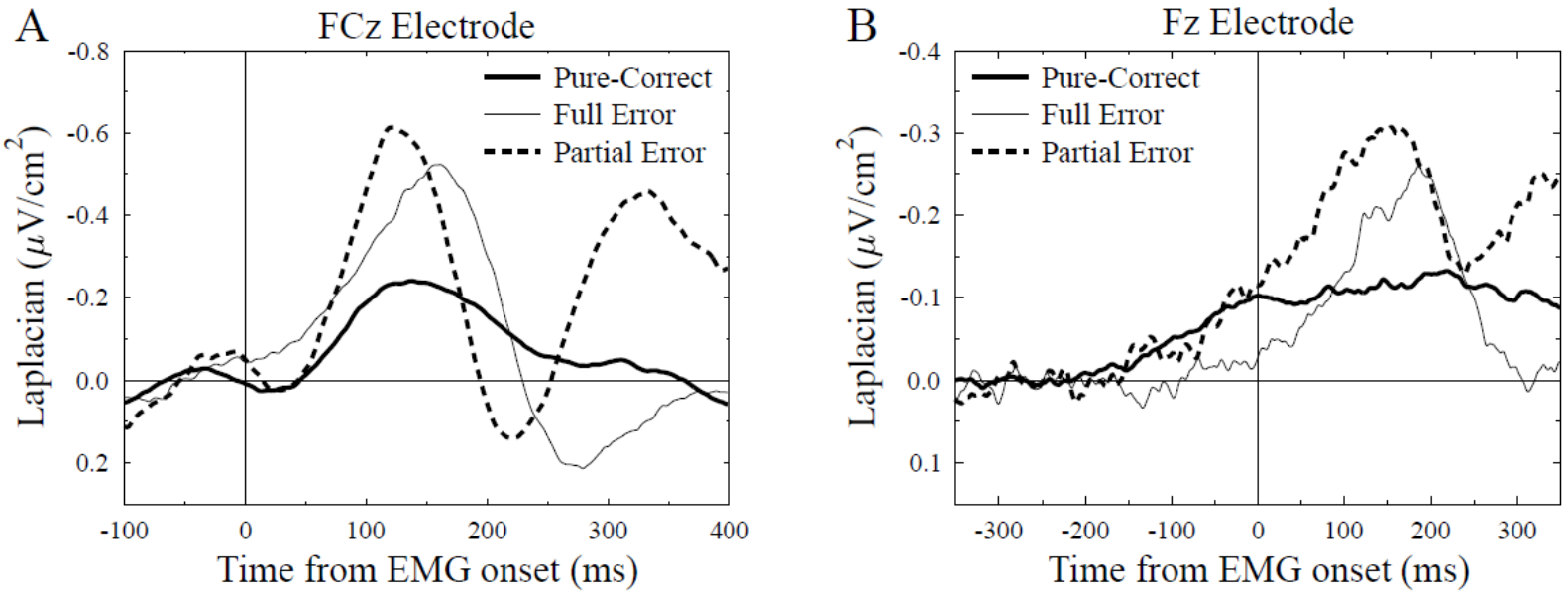
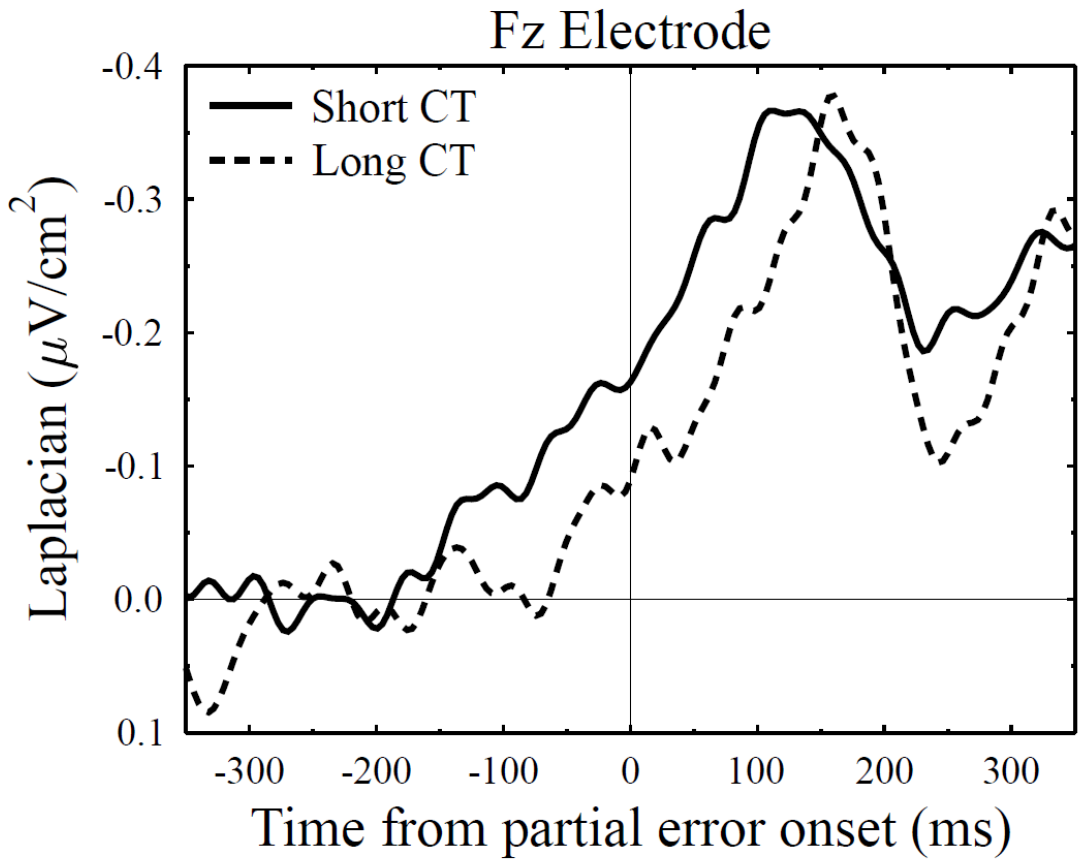


Figure 6: Laplacian-transformed EEG data at electrodes FCz (A) and Fz (B) for pure-correct, full error and partial error responses. The time-zero corresponds to the EMG onset. (A) The negativity, considered as the Ne/ERN, peaked between 100 and 150 ms as a function of the response type. Its amplitude was larger for partial-errors and full errors compared with pure-correct responses. (B) A slow negative activity starting before the EMG onset and increasing until 200 ms after the EMG onset was visible. The moment of start and the amplitude differed as a function of the response type. The negative wave started about 200 ms before the EMG onset for pure-correct and partial error responses and started later for full error trials. Its amplitude was larger for the two types of errors and smaller for the correct trials.

Figure 7



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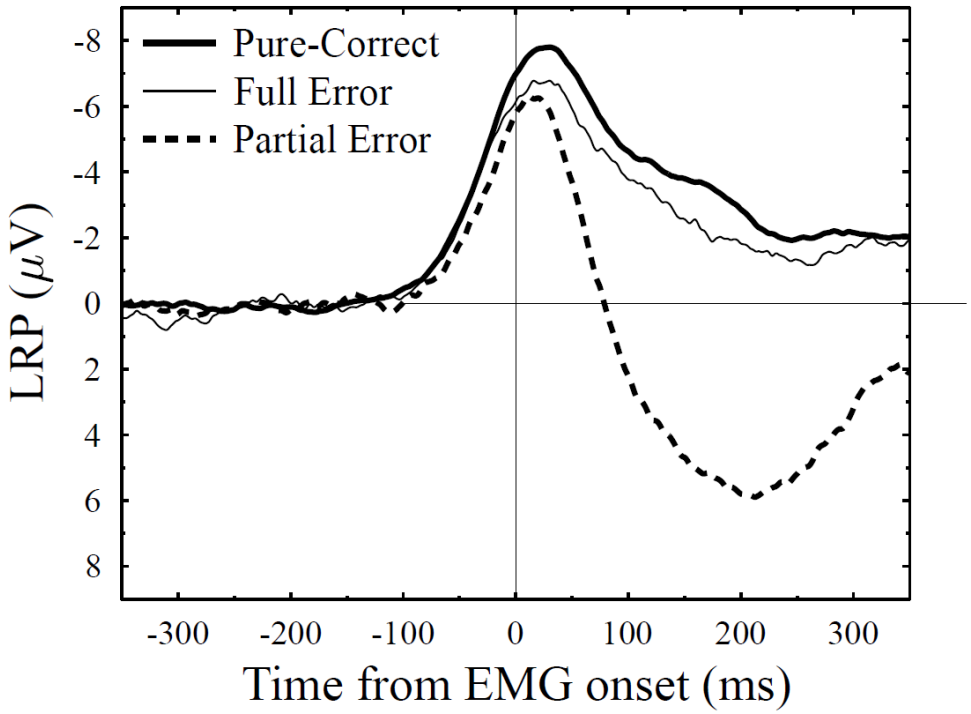
Sup Mat



This figure shows the averaged EMG-locked lateralized readiness potential (LRP) computed from monopolar recordings over the M1s (at C3 and C4 electrodes). For each condition, the difference between contralateral and ipsilateral activity to the responding hand was computed. The baseline was taken between -300 and -150 ms. LRP decreased for full errors and partial errors compared with correct responses. The area under the curve was measured between -150 ms to 150 ms relative to the EMG onset. The ANOVA confirmed a significant effect of response type (pure-correct, full error, partial error responses) on the LRP activity ($F(2,36) = 50.4$; $p < .0001$; $\varepsilon=0.7$). Post-hoc paired-samples t tests confirmed a larger negativity for pure-correct compared with full error responses ($t(18) = 6.1$; $p<.05$), the latter being larger than partial error responses ($t(18) = 56.1$; $p<.0001$).