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Running title:
Consciousness and online error monitoring

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Abstract

In experimental settings, most overt behavioral errors are consciously perceived. They are, however, only the tip of the iceberg, and electromyographic recording of the muscles involved in the response reveals subthreshold incorrect response activations. Although they are all efficiently corrected, such “partial errors” are poorly consciously detected. Electroencephalographic recordings (CSD estimate), revealed the sequence of cortical activities that lead, or not, to conscious detection. Besides medio-prefrontal activities related to action monitoring and error detection, the motor command sent by the primary motor cortices also differed between detected and undetected partial errors: while it develops identically, it is stopped earlier for the latter than for the former, suggesting a critical role in partial error detection. Second, the analysis of the “Error positivity” – Pe, classically linked to error awareness, confirmed its absence just after partial errors, be they detected or not. However, a Pe occurs after the corrective response of partial errors that were detected, suggesting that we become aware of our partial errors only after their correction. The implication of these results for the link between consciousness and cognitive control are discussed.

Keywords

Action monitoring, error awareness, medial-preFrontal cortex, partial errors, primary motor cortices

Human errors play an essential role in regulating behavior, and can have dramatic consequences in industrial contexts (Reason, 1991). The pioneering work of Rabbitt (1966; 1978) showed that we consciously perceive the large majority of our behavioral errors. Moreover, the classically reported post-error behavioral changes (Laming, 1979) are often considered to reflect strategic adaptation to avoid new errors (Dutilh et al., 2012, Danielmeier and Ullsperger, 2011, Wessel, 2018 for reviews, see however, Notebaert et al. 2009). Our understanding of error-related mechanisms has dramatically grown after the discovery of brain activities modulated by error processing: behavioral errors elicit a large medio-frontal EEG negative potential that starts around 30 ms after the onset of the (incorrect) electromyographic (EMG) activity, termed the "Error Negativity" (Ne, Falkenstein et al., 1990; 1991) or "Error Related Negativity" (ERN, Gehring et al., 1993), followed by the "Error Positivity" – Pe – (Falkenstein et al., 1990; Gehring et al., 1993), a neurophysiological component peaking 200-400ms later. While it was originally reported that the amplitude of the Ne does not depend on the conscious detection of errors (Nieuwenhuis et al., 2001, Endrass et al., 2007, O'Connell et al., 2007; although see Sheffers et al., 2000), recent data mitigated this conclusion (Shalgi & Deouell, 2012; Hewig et al., 2011; Wessel et al., 2011; Wessel, 2012 for a review). In contrast, the Pe is commonly associated with conscious awareness of errors (Overbeek et al., 2005, Murphy et al., 2012).

Besides overt errors, recording the EMG activity of the muscles involved in the responses revealed that, in about 15-20% of behaviorally correct trials, a subliminal activation of the incorrect response occurs before the correct response is given (Eriksen et al., 1985, Smid et al., 1990, see Figure 1A). Previous studies reported that partial errors also elicit an Ne, although of smaller amplitude (Vidal et al., 2000; Roger et al., 2010; Bonini et al., 2014), but no Pe (Burle et al., 2008; Vidal et al., 2000).

However, despites being corrected, partial errors are rarely consciously detected (about one third, Rochet et al., 2014), which contrasts with the high detection rate of overt errors. This poor detection, however, appears coherent with the absence of Pe. More surprisingly, a small post partial-error slowing

has been reported (Allain et al., 2004). The relative contribution of aware and unaware partial errors to such slowing remains to be deciphered.

Such “partial errors” are of particular interest to study online control since, in few tens of milliseconds, a potential error has been stopped and corrected (Hasbroucq et al., 1999; Burle, et al., 2002, Spieser et al., 2015). In the present study, we aimed at comparing aware and unaware partial error to better evaluate their respective contribution to post-partial error slowing and to investigate the link between Ne, Pe, error awareness and corrective processes. Anticipating a larger Ne for aware than unaware partial errors, we further sought the origin of such difference.

Among other possibilities, the efference copy (Angel, 1976; Wolpert, 1997) has been suggested to play a pivotal role in action evaluation (Rochet et al., 2014, Haggard & Magno, 1999; Haggard & Whitford, 2004). To account for their results, Rochet et al. (2014) predicted a stronger motor command for aware partial errors, hence leading to a stronger efference copy. Such a difference in the strength of the incorrect motor command could secondarily determine (at least partially) the size of the error potentials. To test this hypothesis, we investigated the EEG markers of the primary motor cortices activity related to the cortico-spinal command (Vidal et al., 2003, Burle et al., 2004, Servant et al., 2016). Based on these different brain activities, we aim to unveil the spatio-temporal sequence of neural events that allow participants to detect their own (potential) failures and to recover them.

Materials and Methods

Participants

Eighteen right-handed volunteers (9 women/men, mean age 23.8 years old, ranging from 19 to 38 years, mainly students from Aix-Marseille University) with normal or corrected-to-normal vision (a

priori inclusion criteria) participated in the study after giving written informed consent. This sample size was determined based on the first study by Rochet et al. (2014), All participants reported being free of psychological and neurological disorders (a priori inclusion criteria). Participants performed two 2-hours-long sessions on separate days and they were reimbursed 15€ per hour. The study was approved by the “Comité de Protection des Personnes Sud Méditerranée 1” (agreement n° 10-41). Participants were informed about EMG recordings and partial errors, through images depicting examples and by showing them their own EMG traces on a monitor. None of the participants that took part in the experiment were excluded from the analysis.

Stimuli and Procedure

Participants seated in a comfortable chair placed inside of a Faraday cage throughout the duration of the experiment. Stimuli were delivered by three light-emitting diodes (LEDs) placed 1 meter away from participants’ eyes. Responses were collected through two buttons (force necessary for closure: 7 N) placed on a table in front of the participants. The cylindrically-shaped hand-grips were vertically fixed on the table and participants held them throughout the experiment, resting their thumb on the top (where the force sensor is placed). Participants were asked to press the button with their thumb to reach the response threshold as quickly as possible, to deliver a response. All of the stimuli and responses were controlled by a PC running a custom-made computer program based on Tscope (Stevens et al., 2006). On each session, participants performed 12 blocks of 64 trials (1536 trials for the two sessions) of a variant of the Simon Task (Simon, 1990): participants were requested to press the left or right response button depending on the color of the LED. Each trial started with a central blue fixation LED presented for 150ms, followed, 500ms later, by the target stimulus. The target LED (red or green) was randomly presented for 1000 ms (the time limit to respond) to the left or to the right of the fixation light. Although the location of the stimulus was irrelevant for the task at hand, the LED could light-up

ipsi- (congruent trial) or contra-laterally (incongruent trials) to the required response. On one of the two sessions, participants were asked to press the right button (with the right thumb) as fast as possible at the presentation of a green LED, or the left button (with the left thumb) for a red one. On the other session, the color-to-response side mapping were reversed to reduce learning effects across sessions. The between session mapping order was counterbalanced across participants.

At the end of each trial, a visual scale from 1 to 6 was displayed on a monitor placed under the LEDs. Participants were asked to orally report how sure they were that, on that trial, they committed a partial error, from 1: “I am sure I did not produce a partial error”, to 6: “I am sure I did produce a partial error”. They had to report “7” if they instead produced an error (pressed the wrong button). The subsequent trial started after 1 second.

EMG and EEG recordings

The EMG activity was bipolarly recorded from the *flexor pollicis brevis* of the two hands, using surface flat type active (Biosemi, Amsterdam, The Netherlands) Ag/AgCl electrodes, glued approximately 2 cm apart on the *thenar* eminences. Data were sampled at 1024Hz (band-pass: DC at 208Hz, 3dB per octave). The quality of EMG recordings was online visually monitored by the experimenter, which reminded participants to relax the hand muscles whenever the EMG signal showed tonic muscular activity.

The EEG activity was recorded using a Biosemi cap including 128 Ag/AgCl electrodes (pin-type active electrodes, Biosemi, Amsterdam). Additional surface electrodes, identical to the ones used for EMG recordings, were applied above and under the left eye to record vertical eye movements (VEOG) and on each canthus for horizontal ones (HEOG). The reference electrode was placed on the right mastoid.

EMG pre-processing

The signal was offline high-pass-filtered at 10 Hz. The onset of any EMG activity following stimulus presentation was detected by a homemade program based on Hodges and Bui (1996) variance ratio algorithm to detect partial errors and EMG activities leading to responses. All onsets were verified by visual inspection. The researcher who checked the quality of EMG onset was not informed of trial type (congruent vs. incongruent) nor of participant rating of partial error occurrence, preventing any bias in the detection. Based on these EMG activities, behaviorally correct trials were then sorted into “pure correct” (PC – only one EMG on the correct side) and “partial errors” (in which a subthreshold incorrect EMG activation was followed by the corrective response, see Figure 1A). Behavioral errors were treated separately. All other trials types (7.8%) were excluded from the analyses. Based on the confidence levels, trials were divided into “undetected” (confidence levels 1 and 2) and “detected” (confidence levels 5 and 6), whereas (aware) error trials had confidence level 7. The mid-values 3 and 4 of the confidence scale were not included in the analyses firstly because they were relatively rare (5% on average) and secondly to avoid confounding (partial) error monitoring with confidence effects (see Shalgi and Deouell, 2012 and the discussion section). Combining the objective presence of a partial error or not and the subjective rating, we could form four trials categories: correct rejection (pure correct rated as 1-2), false alarms (pure correct rated as 5-6), omissions (partial error rated as 1-2) and correct detection (partial errors rated as 5-6).

EEG pre-processing

EEG data were offline re-referenced to the right mastoid. To reduced slow drifts due to hot summer temperatures, EEG data had to be offline high-pass filtered (Butterworth Zero Phase Filter, Low Cutoff: 1 Hz, Time constant 0.16s, 12 dB/oct). Ocular artifacts were corrected using the method of Gratton et al. (1983). These cleaned data were then visually parsed for other artifacts with the help of a semi-automatic artifact method to detect short-lasting artifacts (maximal allowed voltage step: 30 µV/ms

over 400ms). A great care was taken to reject and/or correct (see below) even small, local artifacts since the computation of Current Source Density (CSD) is very sensitive to them. When these artefacts were short (few tens of milliseconds) and localised on only one electrode, the noisy part of this electrode signal was replaced by interpolating the clean neighboring channels using the spherical interpolation method (Perrin et al. 1989) implemented in EEGLAB (Delorme and Makeig, 2004). This allows to not reject a whole epoch, because of limited artifacts on only one channel, while not building averages based on different numbers of trials per channel. If the interpolation was not judged satisfactory, the whole epoch was manually rejected. All epochs containing artefacts that could not be corrected were rejected.

EMG analyses

On partial error trials, nine parameters where extracted from the EMG (see Rochet et al., 2014 for details), the most relevant ones being displayed in Figure 1A: the latency of the incorrect EMG burst (IncLat); the correction time (CT), defined as the time between the incorrect and correct EMG bursts onsets; the motor time (MT) that separated the correct EMG onset from the mechanical response; the surface area under the incorrect (IncSurf) and correct (CorSurf) EMG bursts; the duration of the incorrect (IncDur) and correct (CorDur) EMG bursts; the leading edges of the two EMG bursts (IncSlope and CorSlope for the incorrect and correct EMGs, respectively, see Rochet et al., 2014 for more details). To investigate which of these 9 EMG parameters (for descriptive data of these parameters see table S1) are influenced by partial errors conscious detection and trial congruency, we conducted a multifactorial ANOVA, including factors awareness (detected vs. undetected) and congruency (congruent vs. incongruent trials). Note, however, that based on Rochet et al. (2014) results, we expect only few of these analyses to be significant.

EEG analyses

After averaging the cleaned epochs of interest, CSD were estimated by computing the Surface Laplacian of the scalp potentials (Perrin et al., 1987, 1989). The order of spline was set to 4 and the smoothing parameter to 10^{-5}. Compared to scalp potentials, CSD dramatically improves spatial (Nunez et al., 1994, Babiloni et al., 2001) but also temporal resolution (Burle et al., 2015). With high electrodes density settings, the electrodes best capturing the activity of interest after CSD may vary slightly across participants (due either to small shift in the cap, or to the underlying cortical folding). Since we had strong a priori expectations on the location of the neurophysiological activities of interest, we focused on the ones related to action execution (on fronto-lateral electrodes, above M1, in close vicinity of C3/C4 standard locations) and monitoring (on fronto-central electrodes, over medial-prefrontal cortex in close vicinity of FCz electrode), on both pure-correct and (partial) errors. For each activity (fronto-lateral and fronto-central ones), we hence first identified the electrode that best captures the targeted activity (assessed in terms of amplitude and of signal/noise ratio). For the fronto-central activity, depending on participants, two neighbor electrodes best captured the monitoring activities, namely Cz and FCz (see figure 1B, black box). For the fronto-lateral activities, besides C3/C4 electrodes, for some participants, the best capturing electrode were more medial, for some slightly more caudal (see figure 1B, grey boxes). Three comments are in order. First, this identification was done without assessing any potential experimental effects of interest. Second, no other cluster of electrode showed the activities of interest. Third, the across participant rostro-caudal shift was perfectly coherent between fronto-lateral and fronto-central electrodes: participants with more caudal/rostral “M1” electrodes (grayed squares/circles), were also the ones with the more caudal/rostral “MPFC” (grayed squares/circles).

Individual average EEG activity recorded from the selected electrodes, time-locked to the relevant events (see below), were extracted, and statistical analyses were based on these selected electrodes

(topographic maps, for illustration only, are built on standard grand averaging of the between participants' data). Peak values were defined as the most negative or the most positive value within the predefined temporal window, whereas surface was calculated as the sum of the points within the selected time interval. The slope was calculated by fitting a linear regression to the signal in the selected time windows.

Experimental Design and Statistical Analysis

Data were collected on eighteen volunteers in a within subjects design. They performed two separate sessions that differed on the stimulus-response mapping only (see *Stimuli and Procedure* section). Each participant was pseudorandomly assigned to the first mapping version so to equally divide subjects in two groups. Data from the two sessions were merged. To increase trial numbers (and hence the signal-to-noise ratio), response sides were combined by mirroring electrodes: for left hand responses, right (left) electrodes were recoded as left (right) ones (e.g. C4 was recoded as C3). Hence, in all topographic plots, the left hemisphere electrodes in fact refer to the electrodes contralateral to the correct response, while right hemisphere ones correspond to the ones contralateral to the incorrect one.

Full design ANOVAs were performed, with the error term being the interaction between the random factor "participant" and the factor under analysis. When necessary, plain effect and interaction were broken into planned orthogonal (all sum to zero) contrasts; the vector values used for the corresponding contrasts will be provided. Measures of effect size (as estimated by η_p^2) are provided for standard tests. Normality of the inter-participant data was assessed with the Kolmogorov-Smirnov test.

Based on strong hypotheses for EEG data, statistical analyses were performed only on the selected electrodes as defined above, to avoid multiple comparisons.

Several analyses are based on a limited number of trials (partial-errors and errors). For some activities (e.g. the Ne), their signal/noise ratio is large enough for the activity to be identified on an individual basis. For some other activities (Pe, M1 activities), the signal/noise ratio on individual averages might be low, hence impeding a reliable estimate of the individual component of interest. In such cases, it is recommended to adopt the Jackknife procedure to perform the statistical tests (Tukey, 1958, see Ulrich and Miller, 2001, Kiesel et al., 2008). This procedure relies on grand-averages performed on all participants but one, the missing one being changed on each estimation. Hence, if one has *N* participants, one can build *N* pseudo-grand-averages that will slightly differ, providing an estimation of the contribution of the missing participant to the grand average. These pseudo-grand-averages are then submitted to a standard ANOVA, but the obtained raw F values are corrected as: $Fcor = F/(N - 1)^2$ (Ulrich and Miller, 2001, Kiesel et al., 2008) before assessing significance.

No part of the study procedures or analyses was pre-registered in a time-stamped, institutional registry prior to the research being conducted.

Results

Behavioral and EMG results

We first investigated the effect of trial type (congruent vs. incongruent) on reaction times (RTs) accuracy (percentages of errors) and percentages of partial errors. Replicating published data, participants were significantly slower on incongruent (439ms) that on congruent (403ms) trials ($t(17)=5.974$, $p < .0001$, two-tailed paired t-test) and they produced more overt (9,1%) and partial (21%) errors on incongruent than congruent trials (4,4% and 9%, $t(17)=-4.737$, $p=.0001$ and $t(17)= -6.961$, $p < .0001$, two-tailed paired t-tests, for overt and partial errors, respectively). In agreement with Rochet et al. (2014), 35% of partial errors' trials were classified as "detected" (scores 5 or 6), 15% were unsure (scores 3 and 4), and 50% were undetected (scores 1 and 2). Participants committed very few

false alarms, incorrectly reporting partial errors on very few trials (2.27%). Rochet et al. (2014) reported that parameters related to the partial error "size" (amplitude, duration, leading edge, etc...) are sensitive to awareness, along with the "Correction time", that is the time between the incorrect and correct EMG activities. A multifactorial ANOVA including the nine EMG parameters and factors awareness (detected vs. undetected) and congruency (congruent vs. incongruent trials) essentially confirmed these previous results (see Table 1). Averages from the Variance Inflation Factor (VIF; see Kutner, Nachtsheim, & Neter, 2004) was computed to test the potential multi-collinearity of the EMG parameters. The VIF was 3.6±1.28, which indicates some colinearity (as can be expected) but in a reasonnable range.

To investigate the impact of conscious detection on strategic adaptation, we investigated the presence of post-(partial) error slowing (PES). With this aim, we compared average RTs of pure correct trials following four trial types: pure correct (PCPC, mean RTs 391ms), unaware partial errors (PUnawPC, mean RTs 393ms), aware partial errors (PAwPC, mean RTs 411ms) and errors (ErrPC, mean RTs 440ms, see Figure 2). RTs on PC trials significantly differed depending on the preceding trial type ($F(3,51)=16.134$, $p<.001$, $\eta_p^2 = 0.48$, ANOVA). To specify the nature of the effect, we planned three orthogonal contrasts taking PCPC as reference[1]. Compared to PCPC trials, both PAwPC ($t(17)=-2.472$, $p=.024$, two-tailed paired t-test) and ErrPC ($t(17)=-5.412$, $p=.00002$, two-tailed paired t-test) trials were significantly slower. In contrast, PeUnawPC trials did not significantly differ from PCPC trials ($t(17)=-0.300$, $p=.77$, two-tailed paired t-test). Hence while participants slow down after a conscious incorrect response activation, they do not when this incorrect activation remains undetected.

[1]Formally, the contrasts used were: c1= 1,-1,0,0; c2=1,0,-1,0; c3 = 1,0,0,-1, with the order being: PCPC, PawPC, ErrPC and PeUnawPC. These three vectors are orthogonal.

EEG results

While Pure-Correct and error trials elicit only one EMG burst, two EMG bursts are present on partial error trials: the (small) incorrect one, and the contralateral larger corrective one. We will hence analyse the presence and/or the size Ne, of the Pe and M1 activity, time locked to both EMG activities (see dotted lines on panels A to D on figures 3 and 4).

In a first set of analysis (see Figure 3), we evaluated the impact of awareness on error processing-related brain components evoked by the incorrect EMG bursts of partial errors trials. To do so, averages were computed time-locked to the onset of the partial errors (see dotted lines on panels B and C of figure 3). These activities were compared to the ones elicited on Error and Pure-Correct trials, averaged time-locked to the onset of the incorrect and correct EMG bursts, respectively (see dotted lines on panels A and D of figure 3). Secondly, we compared these activities to EEG components time-locked to the onset of partial errors corrective response (see dotted lines on panels B and C of Figure 4).

Besides plain ANOVA, as for the behavioral data, orthogonal contrasts were designed to qualify the observed effects. They were constructed to address the most relevant questions for a given analysis and, when possible, by taking into account previous studies allowing to expect specific effects.

Awareness and brain activities induced by incorrect EMG of partial errors.

In this section, we report the analyses performed to compare the brain activities related to partial errors (incorrect) EMG, with the same activities measured on error and pure correct trials. Figure 3 shows the brain components of interest (Ne and Pe in 3E; M1 activity in 3F) time-locked to the EMG onsets of interest. Specifically (as indicated by the vertical dotted lines in Figure 3A-D), time 0 represents the onsets of the incorrect EMG on partial error (Aware and Unaware in orange and yellow traces, respectively) and error (red) trials, and the (correct) EMG on pure-correct trials (PC in green), for comparison. Data used for the analyses are summarized in the supplementary table S2.

Error Negativity, Ne

The peak amplitude of the Ne component (Figure 3E) was calculated on each subject as the difference between the most positive and the most negative values in the temporal window from 0 to 170ms post-EMG onset (first gray region on Figure 3E). Single subjects-averages were visually inspected and the most positive value search was restricted to the temporal window from 0 to the latency of the (negative) peak, to insure that the Ne component was correctly selected in all subjects. The amplitudes significantly differ between the four trial types ($F(3,51)=20.318$, $p<.0001$, $\eta_p^2 = .54$, repeated measures ANOVA). Planned comparisons using orthogonal contrasts (same as above) were conducted to compare Ne peak amplitude in the PC condition vs. the other three. Ne amplitude was significantly larger on both aware partial errors - PAw ($F(1,17)=31.122$, $p=.00003$, $\eta_p^2 = .65$, repeated measures ANOVA) and errors - Err ($F(1,17)=16.432$, $p=.0008$, $\eta_p^2 = .49$, repeated measures ANOVA) trials, compared to PC trials, while the latter did not differ from the unaware partial errors - PUnaw condition ($F(1,17)=0.026$, $p=.87$, $\eta_p^2 = 0$, repeated measures ANOVA, see table S2 for data).

The latency of the Ne (measured in the same time-windows, from 0 to 170ms) significantly differs across the four trial types ($F(3,51)=19.231$, $p < .0001$, $\eta_p^2 = .53$, repeated measures ANOVA). Planned orthogonal contrasts revealed that, while the Ne peaked significantly later on Err trials compared to PC trials ($F(1,17)=13.03$, $p=.002$, $\eta_p^2 = .43$), partial error trials peaked earlier than PC trials (PAw: $F(1,17)=4.35$, $p=.052$, $\eta_p^2 = .20$; PUnaw $F(1,17)=20.105$, $p=.0003$, $\eta_p^2 = .54$, see table S2 for data).

Error Positivity (Pe)

We calculated the surface of the Pe component within the temporal window from 250 to 450ms after EMG onset (second gray region on Figure 3E). The Pe being more difficult to extract on individual participants, we resorted to the jackknife procedure to assess its presence. The comparison of the surface area across the four trial types yielded significant results ($F(3,51)=23.59$, $p< .0001$, Jackknife

ANOVA). In agreement with the literature (Burle et al., 2008; Vidal et al., 2000), Figure 3E suggests the presence of a Pe for Err trials that differ from the three other categories. The same planned comparisons as above reveal that PC trials do differ from errors ($F(1,17) = 28.0$, $p < .0001$, jackknife ANOVA), but do not differ from Aware partial errors ($F(1,17) = .99$, $p = .33$, jackknife ANOVA). A significant difference was observed with Unaware partial errors ($F(1,17) = 9.99$, $p = .005$, jackknife ANOVA). However, this reveals a larger **negativity** for Unaware than for PC trials. Hence, while a Pe clearly follows the Ne on Err trials, a second negative going component follows the Ne on partial error trials. This effect could be due to the corrective response that follows the subthreshold incorrect activation on partial error trials (see table S2 for data).

While a Pe is clearly visible on CSD obtained over medio-frontal electrodes, this component is more often analysed on surface potentials (monopolar montage) over more parietal electrodes (e.g. Murphy et al., 2012, see van den Borght et al., 2016 for a discussion). We hence performed the same analysis on surface potentials data over CPz (same time windows from 250 to 450 ms, see figure 5, panels A to E). The main effect of trial type just felt short of significance ($F(1,17) = 2.57$, $p = .06$, jackknife ANOVA). We nonetheless performed the same contrasts as the ones used above. They confirmed that PC do differ from errors ($F(1,17) = 5.81$, $p = .03$, jackknife ANOVA), and that PC did not differ from Unaware ($F(1,17) = .98$, $p = .76$, jackknife ANOVA) nor from aware ($F(1,17) = .33$, $p = .57$, jackknife ANOVA).

M1 activities

We analysed the “M1 activities” (as recorded from the electrodes best capturing the motor components, see Method section and Figure 1B) occurring around the onset of the incorrect EMG of partial error trials (Burle et al., 2008, Servant et al., 2016), reflecting the activation of the incorrect motor command. These activities were compared to the same activity for Err trials (Figure 3F). A comparison of peak latency (within the temporal window from -50 to 50ms) and surface measure (calculated from 0 to 70ms) showed significant differences across the three conditions ($F(2,34)=19.57$, $p<.0001$ and

F(2,34)=11.04, p=.0002, Jackknife ANOVAs, for peak latency and surface, respectively). In the two planned contrasts, we first compared PAw and PUnaw trials, finding that M1 peaked significantly earlier (F(1,17)=6.55, p=.02, Jackknife ANOVA) and tended to be less active (F(1,17)=3.67, p=.07, Jackknife ANOVA) on PUnaw trials. In a second contrast, we compared M1 activity for Err and PAw trials. Results showed that Err elicited a bigger M1 activity (F(1,17)=7.87, p=.012, Jackknife ANOVA) that peaked later (F(1,17)=9.32, p=.007, Jackknife ANOVA) than PAw trials. Although M1 activity lasted longer and was bigger for errors than Paw and for PAw than PUnaw, this activity initially started in the same way, as there was no difference in the initial slope (computed between -50 ms and the EMG onset, F(2,34) = 1.16, p=.33).

Awareness and brain activities induced by the corrective EMG of partial errors.

While the previous analyses focused on incorrect EMG-locked activities, we next analysed brain activities related to partial errors corrective responses (see dotted lines on panels A-D of Figure 4). They were compared to Err (Figure 4E for the MPFC components) and to PC trials (Figures 4E and F for MPFC and M1 activities, respectively). Data used for the analyses are summarized in the supplementary table S3.

Error Negativity (Ne)

We compared the Ne peak-to-peak amplitude and peak latency in the same temporal window as before (0-170ms from EMG onset, first gray zone on Figure 4E) across the conditions that elicit a correct response (partial errors and pure correct trials). Although the traces look very similar on Figure 4E, a general effect of trial type was found for the peak-to-peak amplitude (F(2,34)=3.525, p=.04, η_p^2 = .17, repeated measures ANOVA) but not for the peak latency (F(2,34)=0.150, p=.86, η_p^2 = .01 repeated

measures ANOVA). Planned orthogonal contrasts (c1 = PC: 2, PAw:-1, PUnaw:-1; c2 = PC: 0, PAw:1, PUnaw:-1) for the Ne amplitude indicates that Ne amplitude was slightly higher for partial errors trials than for PC trials ($F(1,17)=7.694$, $p=.013$, $\eta_p^2 = .31$, repeated measures ANOVA), but PUnaw and PAw trials do not differ ($F(1,17)=0.68$, $p=.42$, $\eta_p^2 = .04$, repeated measures ANOVA).

Error Positivity (Pe)

The Pe surface calculated over FCz (CSD) in the temporal window from 250 to 450ms from overt EMG onset across the four trial types differed significantly ($F(3,51)=20.80$, $p < .0001$, Jackknife ANOVA). Pre-planned comparisons show significant differences in Pe surface measure between the PC condition and both PAw ($F(1,17)=11.10$, $p=.004$, Jackknife ANOVA) and Err trials ($F(1,17)=28.0$, $p<.0001$, Jackknife ANOVA), while no differences were found between PC and PUnaw trials ($F(1,17)= 2.53$, $p=.13$). In other words, only consciously detected partial errors elicited a Pe over frontal electrodes.

We also conducted the same analysis on the surface potential (monopolar montage) data recorded over CPz (Figure 5, panels F to J). A clear effect of trial types was observed ($F(3,51) = 7.9$, $p = .002$, jackknife ANOVA). The same contrasts as reported above reveal that PC did not differ from PUnaw ($F(1,17) = .66$, $p = .42$, jackknife ANOVA), but did differ from both PAw ($F(1,17) = 16.95$, $p = .0007$, jackknife ANOVA) and Errors ($F(1,17) = 8.87$, $p = .008$, jackknife ANOVA). Hence, both Errors and PAw elicited a Pe.

M1 activities

To investigate the motor activity associated with the corrective response on partial error trials we analysed M1 activities time-locked to the corrective response, and compared them to the motor execution of the correct (and unique) response on PC trials (Figure 4F). The peak latency and

amplitude were calculated in the temporal window from -50 to 50ms with respect to EMG onset. They did not differ across conditions ($F(2,34)= 0.034$, $p=.96$ for peak latency and $F(2,34)=0.72$, $p=.49$ for peak amplitude, Jackknife ANOVAs).

To investigate the build-up of the motor command, we measured the slope of M1 activation in the temporal window from -50 to 10ms (approximately the latency of the peak). We found significant differences in the steepness of the slope across conditions ($F(2,34)=3.31$, $p=0.048$, Jackknife ANOVA), that we further explored through planned orthogonal contrasts. We first compared the PC condition to the two partial error conditions together (PC:1, PUnaw: -.5, Paw: -.5), finding that partial error trials elicit a steeper slope ($F(1,17)=5.91$, $p=.026$, Jackknife ANOVA). Secondly, the comparison between PAw and PUnaw trials (PC:0, PUnaw: 1, Paw: -1) revealed no difference ($F(1,17)=1.97$, $p=.18$, Jackknife ANOVA).

Discussion

In everyday life, as in the laboratory, we do make mistakes, often due to improper processing of the relevant available information. Detecting failures in ongoing processes is essential to flexibly adapt our behavior to the environment. Studies on error processing have largely concentrated on behavioral failures, that is overt errors. Although detecting potential failures before they end-up in overt errors appears essential, it has been much less studied. Investigating how (and when) partial errors are detected aims to bridge this gap. Partial errors are of particular interest in this respect: first, being smaller response activations than overt ones, they leave more space for modulation; second, they all have been, by definition, corrected; finally, they represented a prototype of an efficient online control: although an error was close to be committed, control mechanisms were efficient enough to overcome it. The present study aims at clarifying the spatio-temporal dynamics of error detection and correction.

Besides replicating Rochet et al. (2014) behavioral results, EEG recordings in the present study allowed to better characterize the time course, and the origin, of partial error conscious detection. Furthermore, the design used in the present study eliminates previous confounds. First, both PAw and PUnaw are based on high confidence judgment ("I am sure I did not produce a partial error" vs. "I am sure I did produce a partial error", Yeung and Summerfield, 2012). Second, correction (Rabbit et al., 1978) is also controlled for, since partial errors are, by definition, always corrected. Finally, a scale, which seems to be the optimal method to investigate error-awareness Ne modulations (Wessel, 2012), was used.

Ne amplitude determines awareness, its timing relates to correction

We observed a clear awareness-related modulation of the Ne when time-locked to incorrect EMG activity. While this result fits with some previous reports on overt errors (Shalgi & Deouell, 2012; Wessel et al., 2011), it contrasts with older ones. More detailed analyses revealed interesting features. In terms of amplitude, PAw did not differ from errors, while PUnaw did not differ from correct responses. These results indicate that the Ne amplitude is strongly linked to error detection, but not to correction. In contrast, the latency analyses cluster trials in a different way: Ne elicited by partial errors (both aware and unaware) peaked earlier than correct and erroneous trials. The timing of the Ne is, therefore, independent from error conscious detection, but covaries with correction (Bonini et al., 2014, Roger et al., 2014, see also Fiehler et al., 2005, for comparable results on overt errors). This timing pattern is compatible with the proposition that the Ne may work as an "alarm signal" developing until appropriate remediating action is issued (Burle et al., 2008, Bonini et al., 2014): if correction starts too late, the response cannot be stopped anymore and an error occurs. Conversely, if the remediation starts early enough, the incorrect response can be stopped and the correct response issued, resulting in a partial error. This view is corroborated by latency and peak differences of M1 activations (Figure 3F), that we will now discuss.

M1 activity and incorrect activations awareness

Given the earliness of the Ne, its modulation is more likely (one of) the possible cause(s), rather than the consequence, of conscious detection (Steinhauser & Yeung, 2010, see below for further arguments). If so, the observation of a larger Ne on PAw, compared to PUnaw trials, simply pushes the question of the origin of conscious detection one step back, without solving it. Indeed, it remains to be deciphered why is the Ne larger in the first place. Although we cannot fully answer this fundamental question, some results help clarifying it.

EMG activity is larger for PAw than PUnaw (Table 1, see also Rochet et al. 2014). Although one may think that a larger EMG burst may lead to a stronger reafferent signal, leading to an easier detection, Rochet et al. argued against this possibility since: i) partial error hardly induce any movement, leading to almost no spindles re-afferent activity and, ii) reafferences are known to be gated during motor command (Abbruzzese et al., 1981). Instead, Rochet at al. speculated about a pivotal role of the efference copy, sent by the M1s to the SMA, in (partial) errors detection (see also Roger et al., 2014). Indeed, these authors proposed that the strength of the efference copy should covary with the strength of the motor command. They hence predicted that the motor command should be of smaller amplitude for undetected partial errors than for detected ones, which is supported by the current data: M1 activity leading to the incorrect response was larger and lasted longer for PAw than for PUnaw, and even larger for overt errors. Importantly, the initial portion of the motor command did not differ across conditions, suggesting that the factor(s) leading (or not) to incorrect activations' correction and detection occurs later in time. After similar initial dynamics, the incorrect motor command is interrupted on partial error trials. If this interruption occurs early enough, this will lead to a weak efference copy, a small Ne and thus to an unaware incorrect response activation (PUnaw). If it is interrupted slightly later, the efference copy will be stronger, and the Ne will be become large enough for partial error to become

aware (PAw). If interrupted even later, this inhibition will be too late to prevent the behavioral error; the only sign of this inhibition will be truncated EMG bursts (see Allain et al., 2004, Rochet et al., 2014, Roger et al., 2014). Partial errors detection hence depends on the time needed to interrupt it. In line with this hypothesis, PAw are associated to longer CTs than PUnaw. A question remains open, however, as to the origin of this interruption: an active inhibitory control (see e.g. Ridderinkhof, 2002) or a passive decay due to a progressive lack of evidence for the incorrect response (see e.g. Servant et al., 2015). Answering this question is arguably beyond the scope of the present work, which nonetheless paves the way by establishing markers on which this interruption can be studied.

When do we become aware of partial errors?

Contrary to the Ne, the Pe has classically been linked to the conscious detection of errors (see e.g., Overbeek et al., 2005, Murphy et al., 2012). In agreement with the literature, we found that overt errors elicited a Pe (Figure 3E) after the Ne. No such Pe is observed right after partial errors (Figure 3E and 5E, see also Vidal et al., 2000, Burle et al., 2008), be they consciously detected or not. While the absence of Pe on PUnaw fits with this view, its absence for PAw may seem more problematic. Time-locking the EEG activity to the corrective response shed new light on this issue. Indeed, on PAw a Pe is elicited *after* the corrective response (Figure 4E and 5J). The presence of such a late Pe has several interesting functional consequences. First, it fits with the idea that Pe is indicative of conscious detection. Second, if one accepts that the timing of the Pe is an indicator of the timing of conscious access, this observation suggests that we become aware of our partial errors only after this incorrect response activation has been corrected. Importantly, however, the corrective response, by itself, does not seem to play a critical role in the awareness. Indeed, neither the Ne nor M1 activity induced by the corrective response differed between PAw and PUnaw (Figures 3E and 3F, although they might differ from pure-correct trials). Third, if one becomes aware of partial errors only after they have been

corrected, this indicates that the processes leading to the incorrect response interruption and correction are entirely non-conscious. In turn, this means that cognitive control can occur without awareness (van Gaal et al., 2008; 2009) and challenges the common view that cognitive control requires consciousness to operate (Dehaene & Naccache, 2001).

What are the consequence of partial error detection?

While the nature of post-(partial) error adjustment is still a matter of debate (see Wessel, 2018 for an overview), our results help shed light on the origin of the such adjustments. The consequences of the present data for three main accounts of sequential adjustments will now be discussed.

The conflict monitoring account of post-error slowing states that, on errors trials, the correct response is nonetheless activated, but too late, leading to post-error conflict. Conflict is assumed to be captured by the Ne (Botvinick et al., 2001, Yeung et al., 2004, see however, Burle et al., 2008). When conflict is detected, control adjustments take place (re-focus of attention, increase of response threshold, etc...). We found the Ne to be larger for overt errors and PAw which may fit with this view. However, while the size of the Ne is comparable between errors and PAw, post-(partial)error slowing was different, weakening the link between Ne amplitude and slowing. Furthermore, conflict is assumed to be computed at the end of the trial, as integrated conflict activity. In such case, if the Ne represents conflict, the total amount of conflict at the end of the trial should be the sum of the Ne triggered by the incorrect EMG and the one induced by the corrective one; the amount of conflict should, therefore, be larger for partial errors, even undetected, than for PC trials. In this case, a post-error slowing should also be present after PUnaw that instead we found not to be different from PCPC trials. Therefore, the present data do not support a (direct) link between Ne/conflict and post-error adjustments.

Another account of post-error slowing considers errors as a special case of “surprise” effect, that is the automatic reaction to unexpected events (Notebaert et al., 2009, Wessel & Aron 2017). At first sight,

the observed slowing may seem to follow the probabilities of events: errors being rarer (about 6%) than partial-errors (about 15%), they should induce a larger slowing. However, only aware partial errors induce a slowing effect on the subsequent trial. Since they represent approximatively only 5% of all trials, their probability is lower (or equal) to overt errors. If one assumes that an event must be perceived for its probability to be estimated, detected partial-errors and errors have approximately the same likelihood and should, hence, induce comparable post-trial adjustments. If, in contrast, one assumes that even non consciously perceived events trigger a surprise effect (and hence that PAw and PUnaw are processed similarly), one should observe the same post-partial error slowing after PAw and PUnaw. The present data do not support any of these possibilities and invalidate a **direct** relationship between (non) expectancy and slowing and some additional hypothesis are needed.

Finally, it has been proposed that the post-error slowing is due to interference with post-response action monitoring (Jentzsch and Dudschig, 2008). The negativity induced by the corrective response on partial error trials, although slightly larger, is of comparable amplitude to the one observed on PC trials (Figure 4F). Hence, this early response evaluation cannot explain the slowing effect observed after PAw. In contrast, the effects observed on the Pe covary with the observed slowing: no Pe on PC and PUnaw (which have similar RT) and a Pe on PAw and errors, both inducing slowing adjustments. Although speculative, the present data may well fit with bottleneck interpretation (Jentzsch and Dudschig, 2008), which states that the size of the interference depends on the duration of the response monitoring process. The Pe appears to last longer for errors than for PAw, suggesting that response evaluation also takes more time in the case of errors, hence inducing a larger delay (i.e. slowing) of the next response. Note, however, that the inter-trial interval used on the present study was pretty long (due to the partial-error evaluation scale), which should have eliminated this bottleneck effect, hence weakening, and/or requiring more precise specification of, this account.

Conclusions

To wrap-up, the incorrect response activation at M1 levels seems to trigger the Ne, whose amplitude is (at least partly) determined by the duration of the incorrect M1 activity. Once this incorrect activation subsumes, the Ne is interrupted. If this occurs early enough, the incorrect response is stopped and a correct response issued. An error is committed if the interruption occurs too late. The information conveyed by the Ne start being accumulated (Steinhauser & Yeung, 2010, Murphy et al., 2012) and one becomes aware of the incorrect activation if a threshold is reached. This accumulation process might be degraded by the production of the correct response: if correction occurs too early (short CT), it may interrupt the accumulation process that will never reach the threshold. If it occurs later (longer CT) a higher level of accumulation will already be gained, and the correct response will not erase the accumulation, but simply delay it. It will reach the consciousness threshold after the corrective response, triggering a Pe and awareness of the partial error. A critical question that still needs to be addressed in future work is how is the incorrect response activation detected and is interrupted.

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Figures caption

Figure 1*. Example of partial error (A) and electrodes of interest (B). A. Example of a trial containing a partial error. This panel plots the EMG activities as a function of time post-stimulus (solid line on the left, time 0) in the muscles involved in the incorrect response (top trace) and in the correct one (bottom trace). The long rightmost vertical dotted line indicated the moment of the (correct) mechanical response. A large EMG burst starts just before this mechanical response (bottom trace). This correct EMG activity is preceded by a small EMG activity on the incorrect muscle (top trace). Inset: the same EMG traces are plotted after rectification (i.e. taking their absolute values). The main variables of interest are also depicted: IncLat (latency of partial error, from stimulus to partial error onset), CT (correction time, from partial error onset to correct EMG onsey), MT (motor time, from correct EMG onset to mechanical response), IncSurf (surface of partial error, measured as the surface under the rectified incorrect EMG trace), CorSurf (surface of the corrective response, measured as the surface under the rectified correct EMG trace). For more precise definition of the other variables, see Rochet et al. (2014). B. Electrodes position and electrodes of interest. The 128 electrodes are depicted as empty circles along with the built-in reference system CSM-DRL. Based on single participants grand-average inspection, the "best" electrodes (with higher amplitude/best signal-to-noise ratio) were chosen. For the medial-prefrontal cortex – MPFC – activity (black box), electrode Cz was picked for 12 participants (gray squares), while FCz was chosen for the remaining 6 (gray circles). The same approach was used to select M1 activity (gray boxes). Interestingly, the "best electrodes" for both MPFC and M1 followed the same rostro-caudal gradient: more anterior M1 electrodes (gray code indicates the number of participants for each electrode) were selected for the group in which FCz (most rostral, gray circle) was chosen for MPFC activity, while the more rostral M1 electrodes were chosen for participants in which Cz (more ventral, gray square) was chosen for MPFC. Note that the two choices were made independently, making the spatial consistency even more striking.*

Figure 2: *Sequential adjustments. The figure plots the RT for pure-correct trials depending on the nature of the previous trial. Error bars represented the confidence interval (95%), following the proposition of Cousineau, 2005, and the correction proposed by Morey, 2008, adapted to within participant design. PCPC: Pure-correct preceded by pure-correct, PUnawPC: pure-correct preceded by an unaware partial error, PAwPC: pure-correct preceded by aware partial error, ErrPC: pure-correct preceded by an error. PUnawPC do no differ from PCPC, while both PAwPC and ErrPC present a slowing down. ns: not significant, *: p<.05, **: p<.0001.*

Figure 3*. Brain activities (Current Source Densities) evoked by the incorrect EMG of partial errors. A to D: representative traces of the different types of trials, along with the indication of the event used to form the averages (dotted lines). E: fronto-central (Ne and Pe) activities (current source density as computed by surface Laplacian, baselined between -200 and 0 ms) obtained for errors (in red, see panel A), partial errors (aware in orange, unaware in yellow, see panels B and C) and pure-correct trials (in green, see panel D). Time 0 correspond to the event indicated on panels A to D. Inset: topography of the Ne at 150 ms (peak of the Ne) post EMG onset. F: M1 activity (current source density as computed by surface Laplacian) for errors, and partial errors (same color code). Time 0 is the same as in panel E (see panels A to D). Inset: topography of M1 activity at 25 ms post EMG onset.*

Figure 4*. Brain activities (Current Source Densities) evoked by the corrective EMG of partial errors. A to D: representative traces of the different types of trials, along with the indication of the event used to form the averages (dotted lines). E: fronto-central (Ne and Pe) activities (current source density as*

computed by surface Laplacian, baselined between -200 and -100 ms) obtained for errors (in red, see panel A), partial errors (aware in orange, unaware in yellow, see panels B and C) and pure-correct trials (in green, see panel D). Time 0 correspond to the event indicated on panels A to D. Inset left: topography of the Pe at 255 ms post EMG onset for errors. Inset right: topography of the Pe at 255 ms post EMG onset for aware partial errors. The two topographies are at the same scale. F: M1 activity (current source density as computed by surface Laplacian, baselined between -200 and -100 ms) for partial errors (same color code) and pure-correct (in green). Time 0 is the same as in panel E (see panels A to D). Inset: topography of M1 activity at 29 ms post EMG onset.

***Figure 5**. Parietal Pe (scalp potential, monopolar data) recorded over CPz evoked by the incorrect (panel E) or the corrective EMG of partial errors (panel J). A to D: representative traces of the different types of trials, along with the indication of the event used to form the averages (dotted lines) presented in panel E. E: centro-parietal (CPz) activities (surface potential, monopolar recordings, baselined between -200 and -100 ms) obtained for errors (in red, see panel A), partial errors (aware in orange, unaware in yellow, see panels B and C) and pure-correct trials (in green, see panel D) to assess the presence of a Pe after the incorrect EMG of partial errors. Time 0 correspond to the event indicated on panels A to D. F to I: representative traces of the different types of trials, along with the indication of the event used to form the averages (dotted lines) presented in panel I. I: centro-parietal (CPz) activities (surface potential, monopolar recordings, baselined between 0 and 100 ms) obtained for errors (in red, see panel A), partial errors (aware in orange, unaware in yellow, see panels B and C) and pure-correct trials (in green, see panel D) to assess the presence of a Pe after the corrective EMG of partial errors trials . Time 0 correspond to the event indicated on panels F to I.*

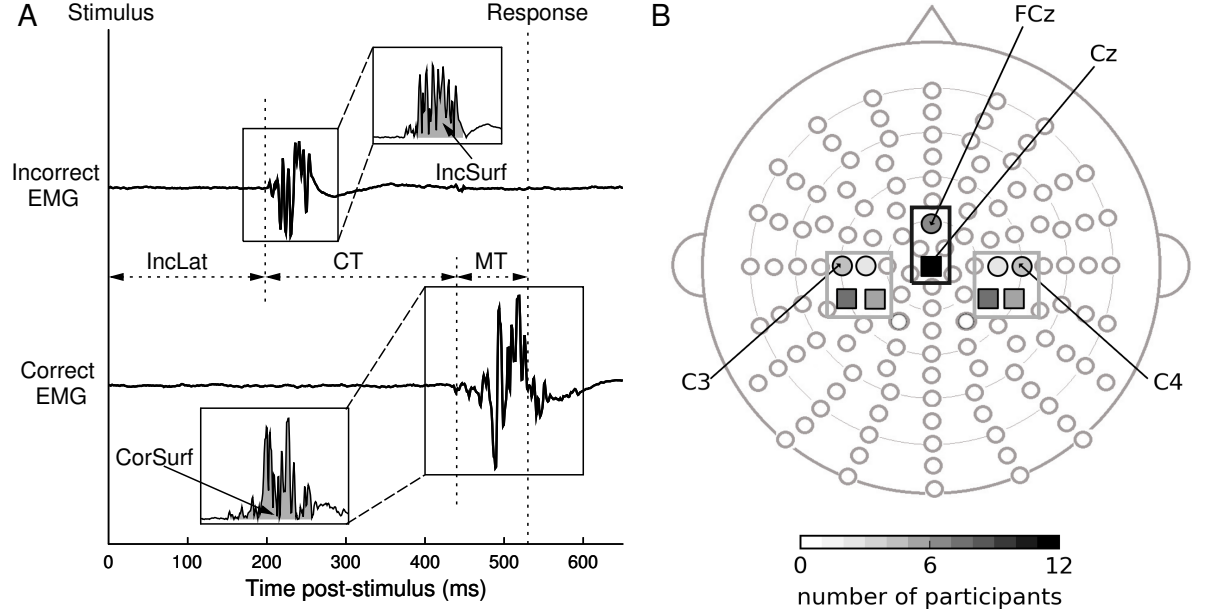
Tables

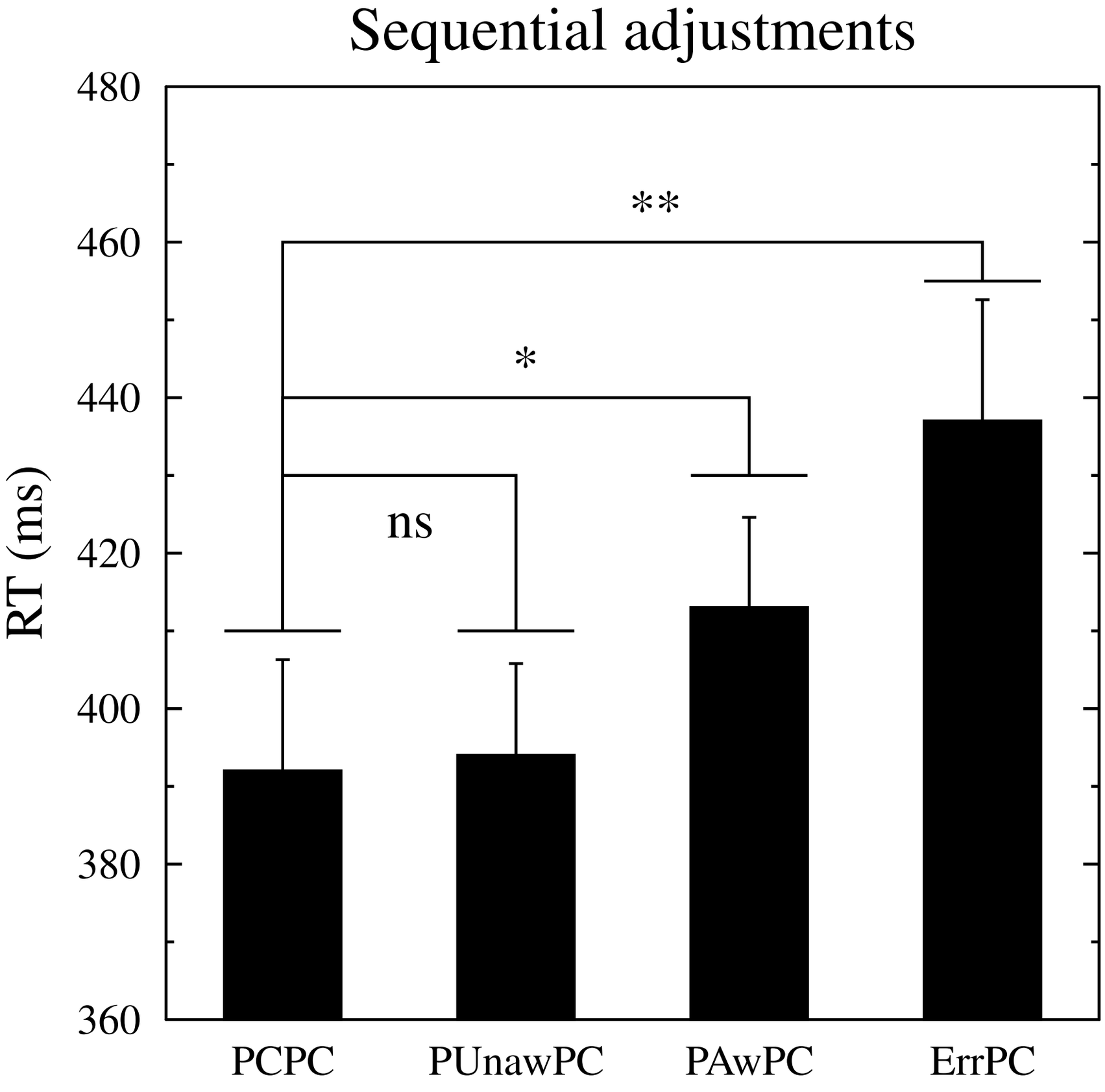
Table 1: *Results of the multifactorial ANOVA to check for effects of factors awareness and congruency on the nine EMG parameters. Sig. = significance level (p-value). Sig. FDR = significance level (p-value) after controlling for multiple comparisons. Note, however, that for many analysis, non significant effects were expected, while the significant ones were predicted to be. This correction does not take into account such theoretical expectations. For all analyses, the degrees of freedom were F(1,17). Description of the dependent variables' names is included in the method section.*

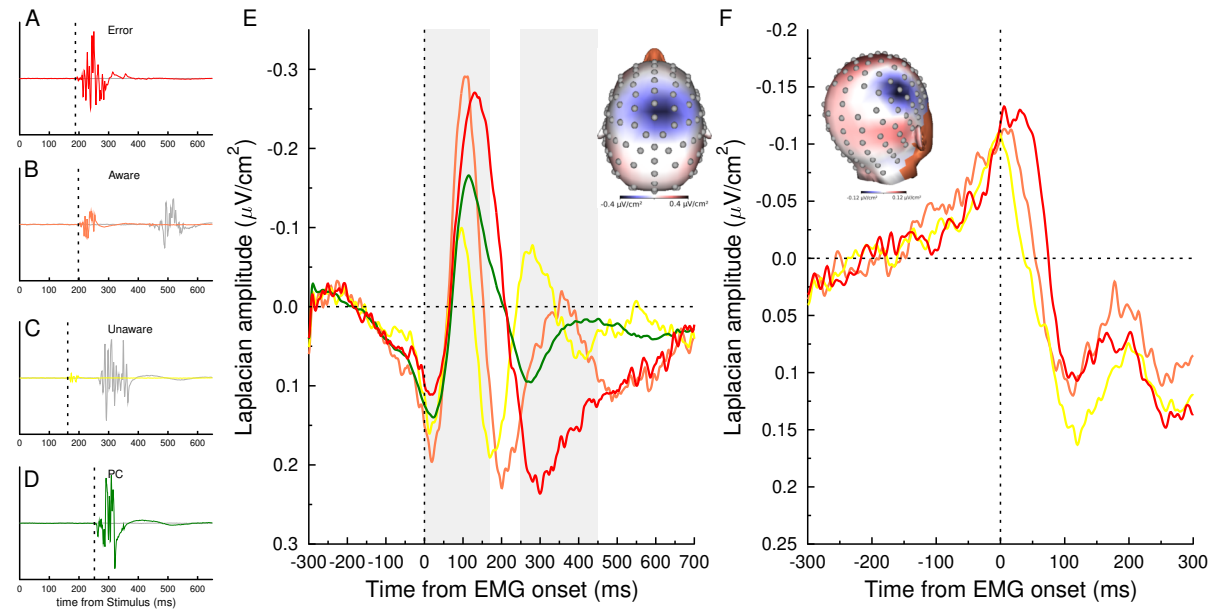
Source	Dependent Variable	Mean Square	F	Sig.	Sig. FDR	Partial Eta Squared	Observed Power[b]
awareness	IncLat	29.325	.019	.892	.989	.000	.052
	CT	9267.050	4.243	**.043**	*.096*	.059	.528
	MT	544.665	.661	.419	.754	.010	.126
	IncDur	313.710	6.178	**.015**	*.067*	.083	.688
	IncSurf	52.839	17.302	**.000**	**.000**	.203	.984
	IncSlope	1558.494	4.522	**.037**	*.096*	.062	.554
	CorDur	340.083	.343	.560	.840	.005	.089
	CorSurf	.036	.000	.989	.989	.000	.050
	CorSlope	392.560	.181	.672	.864	.003	.070
congruency	IncLat	10.359	.007	.935	.948	.000	.051
	CT	48.249	.022	.882	.948	.000	.052
	MT	10.773	.013	.909	.948	.000	.051
	IncDur	10.208	.201	.655	.948	.003	.073
	IncSurf	.048	.016	.901	.948	.000	.052
	IncSlope	15.755	.046	.831	.948	.001	.055
	CorDur	25.134	.025	.874	.948	.000	.053
	CorSurf	.775	.004	.948	.948	.000	.050
	CorSlope	89.825	.041	.839	.948	.001	.055
awareness * congruency	IncLat	249.873	.160	.691	.880	.002	.068
	CT	5377.191	2.462	.121	.465	.035	.340
	MT	141.204	.171	.680	.880	.003	.069
	IncDur	105.053	2.069	.155	.465	.030	.294
	IncSurf	17.268	5.654	**.020**	.180	.077	.650
	IncSlope	389.577	1.130	.291	.655	.016	.182
	CorDur	63.958	.064	.800	.880	.001	.057
	CorSurf	4.191	.023	.880	.880	.000	.053
	CorSlope	124.241	.057	.811	.880	.001	.056

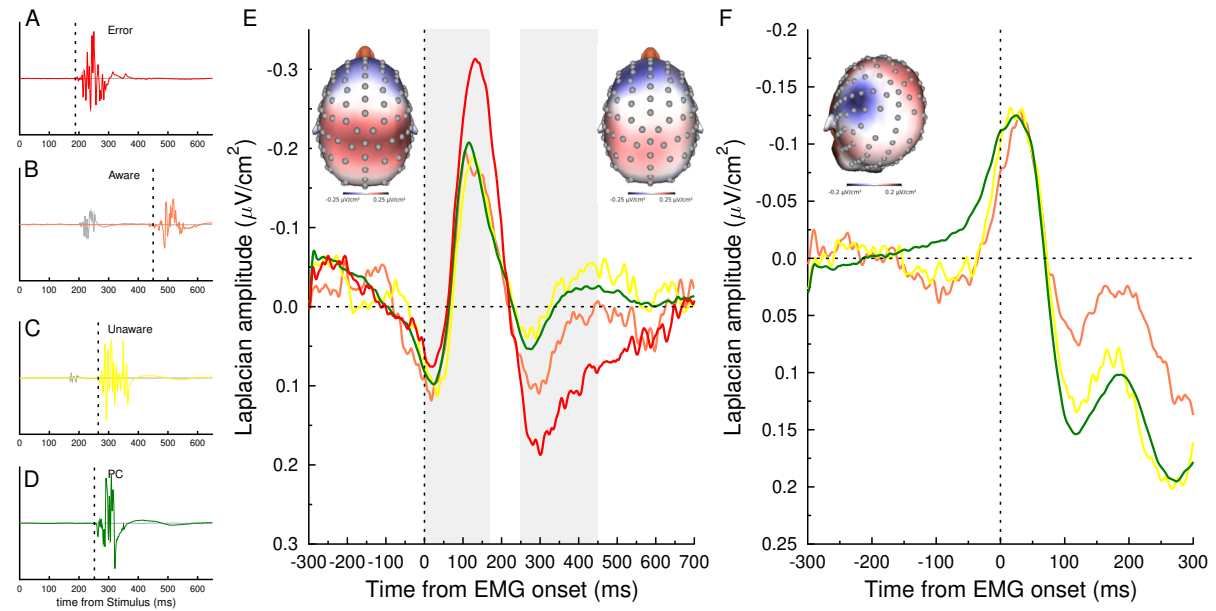
Table 2. Average and standard deviation for each of the nine EMG parameters, separately for Aware/Unaware and Congruent/Incongruent trials are reported.

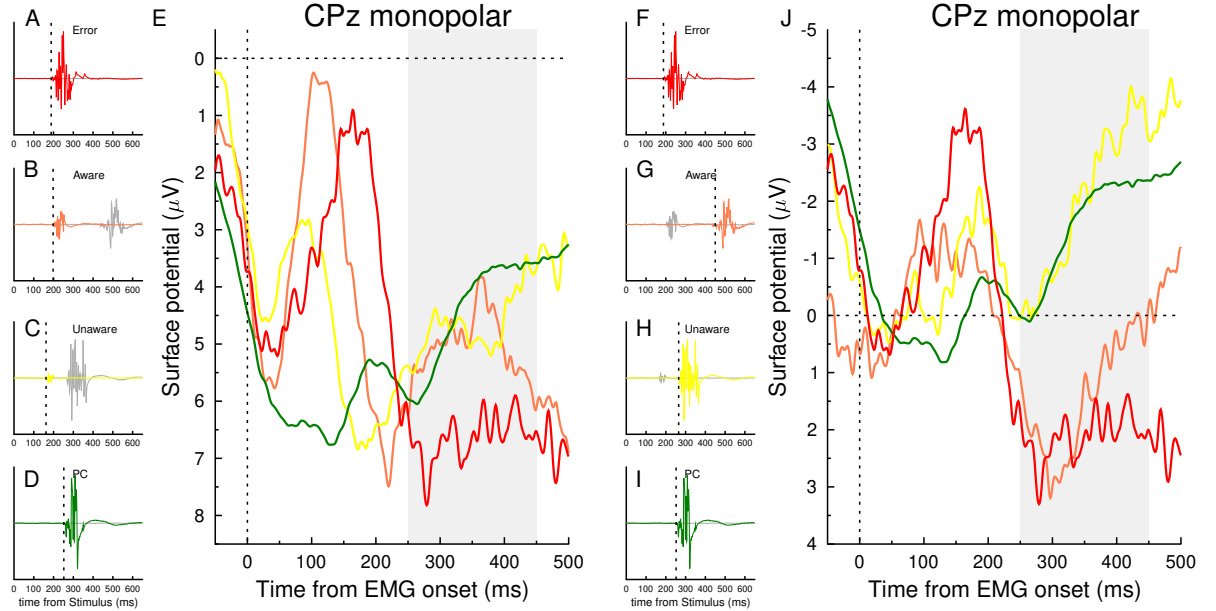
	Aware-Congruent	Aware-Incongruent	Unaware-Congruent	Unaware-Incongruent
IncLat(ms)	212,77± 43	217,26 ± 39	215,22 ± 39	212,25 ± 36
CT(ms)	177,96 ± 50	162,31 ± 49	137,98 ± 41	156,91 ± 46
MT(ms)	122,98 ± 30	120,95 ± 29	114,68 ± 27	118,25 ± 28
IncDur(ms)	29,46 ± 8	27,80 ±7	22,87 ± 6	26,04 ± 7
IncSurf(mV)	4,43 ± 3	3,50 ± 2	1,74 ± 1	2,77 ± 1
IncSlope	39,44 ± 22	35,72 ± 20	25,48 ± 14	31,07 ±17
CorDur(ms)	82,40 ± 34	79,33 ± 31	76,17 ± 30	76,87 ±30
CorSurf(mV)	27,52 ± 14	27,79 ± 13	28,04 ± 14	27,35 ± 13
CorSlope	79,81 ± 49	80,20 ± 43	87,11 ± 47	82,24 ±46


A
Stimulus
Response
Incorrect EMG
IncSurf
IncLat
CT
MT
Correct EMG
CorSurf
0
100
200
300
400
500
600
Time post-stimulus (ms)
B
FCz
Cz
C3
C4
0
6
12
number of participants


Sequential adjustments
480
460
440
420
400
380
360
RT (ms)
**
*
ns
PCPC
PUnawPC
PAwPC
ErrPC


A
Error
B
Aware
C
Unaware
D
PC
0 100 200 300 400 500 600
time from Stimulus (ms)
E
Laplacian amplitude (μV/cm^2)
-0.3 -0.2 -0.1 0.0 0.1 0.2 0.3
-300 -200 -100 0 100 200 300 400 500 600 700
Time from EMG onset (ms)
-0.4 μV/cm²
0.4 μV/cm²
F
Laplacian amplitude (μV/cm^2)
-0.2 -0.15 -0.1 -0.05 0.0 0.05 0.1 0.15 0.2 0.25
-300 -200 -100 0 100 200 300
Time from EMG onset (ms)


A
Error
B
Aware
C
Unaware
D
PC
0 100 200 300 400 500 600
time from Stimulus (ms)
E
Laplacian amplitude (μV/cm^2)
-0.3 -0.2 -0.1 0.0 0.1 0.2 0.3
-300 -200 -100 0 100 200 300 400 500 600 700
Time from EMG onset (ms)
-0.25 μV/cm²
0.25 μV/cm²
F
Laplacian amplitude (μV/cm^2)
-0.2 -0.15 -0.1 -0.05 0.0 0.05 0.1 0.15 0.2 0.25
-300 -200 -100 0 100 200 300
Time from EMG onset (ms)
-0.2 μV/cm²
0.2 μV/cm²


A
Error
B
Aware
C
Unaware
D
PC
0 100 200 300 400 500 600
time from Stimulus (ms)
E
CPz monopolar
Surface potential (μV)
Time from EMG onset (ms)
F
G
H
I
J