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Modulation of activity in medial frontal and motor cortices during error observation

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We used measures of the human event-related brain potential (ERP) to investigate the neural mechanisms underlying error processing during action observation. Participants took part in two conditions, a task execution condition and a task observation condition. We found that activity in both the medial frontal cortex and the motor cortices, as measured via the error-related negativity and the lateralized readiness potential, respectively, was modulated by the correctness of observed behavior. These data suggest that similar neural mechanisms are involved in monitoring one's own actions and the actions of others.

Although committing an error is generally considered to be a negative event, errors are crucial for learning and adjusting future behavior. Research on the neural processes underlying errors and their use in modifying behavior has been facilitated by the discovery of the error-related negativity (ERN), a component in the human ERP elicited by errors[1,2] and negative feedback[3]. Dipole analysis has indicated that the ERN is associated with activity in the anterior cingulate cortex (ACC) in the medial frontal cortex[3,4]. Functional imaging studies confirm this finding, showing more ACC activation on error trials and trials with negative feedback than on correct trials[5–8].

Traditionally, the ACC is considered part of a neural network involved in executive control[9], with more recent research indicating a role for the ACC in reward-based selection for action[10–13]. For instance, in both monkeys[14] and humans[15], the ACC is active when a decrease in reward signals a need for a change of action. A recent model of reinforcement learning[16] suggests that the ACC functions as a motor control filter, selecting appropriate responses from the options available. An error or negative feedback serves as a negative reward, allowing the network to learn appropriate responses suitable to the task at hand[13,16].

The role of the ACC in learning has been studied mostly under conditions of individual task performance, where participants are presented with task cues, execute responses, and then receive feedback. However, learning often occurs in a social setting. For instance, children learn from observation and imitation through social interaction, processes that are considered to be crucial for the development of their cognitive motor skills[17,18].

The results of studies on observational learning suggest that the mechanisms by which observation contributes to learning are very similar to those involved in learning 'by doing'[19–21]. Indeed, neuroimaging studies of brain mechanisms involved in action observation support the idea that actions are not just recognized at a visual level but directly activate the motor system[22–24]. These results raise the question of whether the neural processes associated with self-generated errors are also invoked by the observation of errors committed by others. If so, this would indicate that reinforcement learning and observational learning rely on similar neural mechanisms. A recent study provides the first evidence that systems underlying generation of the ERN are also active when participants observe an error committed by another person[25]. In that previous study, however, participants observed simulated rather than real task performance, and the extent of parallel activation of the motor system was not determined.

In the present study, we investigated the hypothesis that the same mechanisms for error processing are active in response to both self-generated errors and errors committed by others. We determined whether both the ACC and the motor cortex are involved in the observation of errors. The ERN was used as a measure of ACC activity, and the lateralized readiness potential[26] (LRP) was used as a measure of relative activation of the participant's motor cortices[27]. These measures were taken both when participants performed a modified Eriksen flanker task[28] and when they observed another person performing the same task (**Fig. 1**). We found that activity in both the medial frontal cortex and the motor cortices is modulated by the correctness of observed behavior in others.

RESULTS

Overall task performance

In both execution and observation conditions, the standard effects of the Eriksen flanker task were observed[28]. Incorrect responses were executed faster than correct responses (250 ms vs. 314 ms; $F_{1,25} = 275.6$, $P < 0.001$). Reaction times (RTs) to compatible stimuli were significantly faster (300 ms) than RTs to incompatible stimuli (328 ms; $F_{1,30} = 64.6$, $P < 0.001$), and fewer errors were made on compatible (4.6%) than on incompatible trials (12.4%; $F_{1,30} = 53.0$, $P < 0.001$).

Pure errors, with only a single response of the wrong hand, were found on 8.5% of all trials. Trials with responses from both hands were recorded on 8.8% of all trials, and on 1.5% of all trials no response was registered in the 150–550 ms response interval. Reaction

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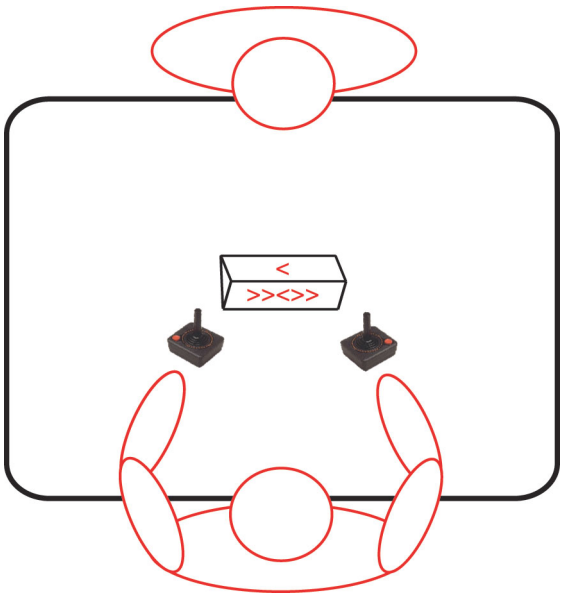

Figure 1 Experimental setup. Schematic overview of the experimental setup showing the actor (bottom) and the observer (top).

times were longer in the execution condition (297 ms) than in the observation condition (267 ms), but the percentage of pure errors did not differ significantly between execution and observation conditions (7.9% and 9.1%, respectively; $F_{1,30} = 0.62$, $P = 0.437$).

Error-related negativity

In the execution condition, there was a large negative deflection on (pure) incorrect trials, as compared to correct trials (**Fig. 2**, upper left). The onset latency of the negativity was 6 ms (s.d. = 10 ms) before the response. The peak latency of the negative difference (between incorrect and correct trials) was 80 ms after the response, and maximal at medial frontal electrode sites (**Fig. 2**, lower left). These features are characteristic of the ERN observed in previous studies[1,2,4].

In the observation condition (**Fig. 2**, upper right), we also found a negative deflection on incorrect trials. This deflection started 90 ms (s.d. = 30 ms) after the observed response, and peaked at 252 ms. Scalp distribution of this negative deflection was similar to that of the ERN in the execution condition (**Fig. 2**, lower right).

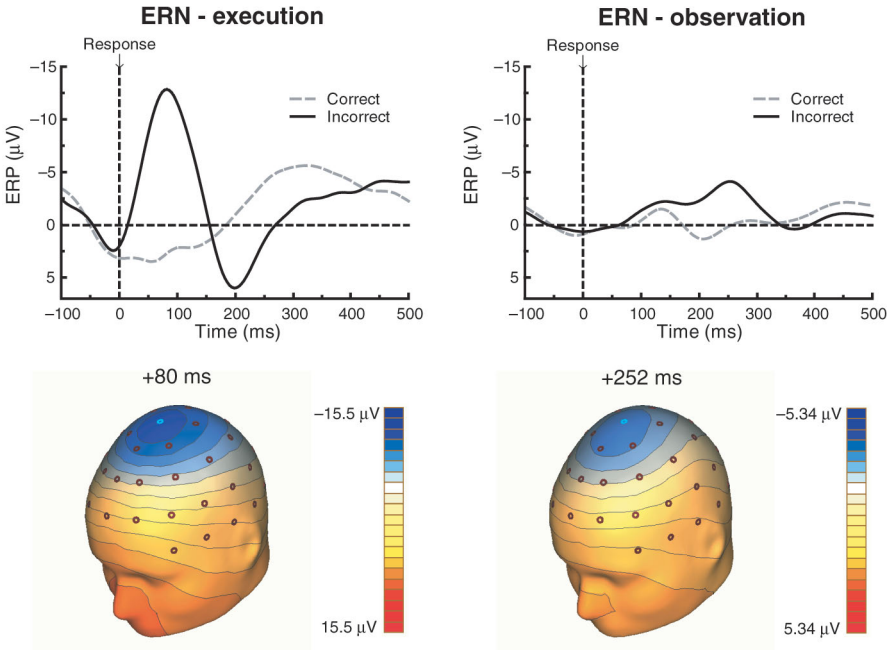

Figure 2 Error-related negativities. Top, response-locked averages at electrode Cz for correct and incorrect responses in the execution condition (left) and the observation condition (right). Dashed gray lines indicate correct, and solid black lines indicate incorrect response trials. Bottom, spline maps showing the topography of the ERN difference wave in the execution condition and the observation condition, taken at the peak where correct and incorrect ERPs differed maximally—80 ms and 252 ms after the response, respectively. The Cz electrode at the vertex is marked in light blue for reference.

Source localization

Grand average difference waveforms between ERPs to correct and incorrect responses were used for source localization to determine the possible neural generators of the negativities in the execution and observation conditions. As in previous studies, we modeled the source of the ERN using a single source[3,4]. In the execution condition, a single regional source (**Fig. 3**, blue), located in the medial frontal cortex (Talairach coordinates (x, y, z): –0.5, 0.6, 28.4), explained 97.3% of the variance in the scalp distribution for the interval where correct and incorrect waveforms differed significantly.

To test the hypothesis that the negativities in the execution and observation conditions are generated in the same neural structures, we determined how well the source for the execution condition would fit as a model for the observation condition. The same source explained 92.4% of variance for the negativity in the observation condition. In a separate analysis, we modeled the data for the observation condition using an unconstrained source. This analysis yielded a source (**Fig. 3**, red) that was slightly more frontal (x, y, z: 3.8, 4.0, 23.8), explaining 92.5% of variance. These data support the hypothesis that medial frontal structures involved in the processing of self-generated errors are also engaged by observing erroneous behavior in others.

Lateralized readiness potential

LRPs in the execution condition (**Fig. 4**, top left) showed a pattern similar to that observed previously[26]. For correct trials, the motor potential was more negative over the hemisphere contralateral to the correct response (indicated by negative values for the LRP), whereas for incorrect trials the motor potential was more negative over the hemisphere contralateral to the incorrect response (reflected by positive LRP values). LRPs for correct and incorrect responses in the execution condition shared a distribution over the lateral motor cortex as revealed by current source density maps (CSDs; **Fig. 4**, bottom left; see Methods for further details).

In the observation condition, an LRP differed significantly from zero from 212 ms ($t_{15} = –1.836$, $P = 0.043$) until 514 ms after the stimulus. The analysis of the LRP time-locked to the response of the actor (**Fig. 4**,

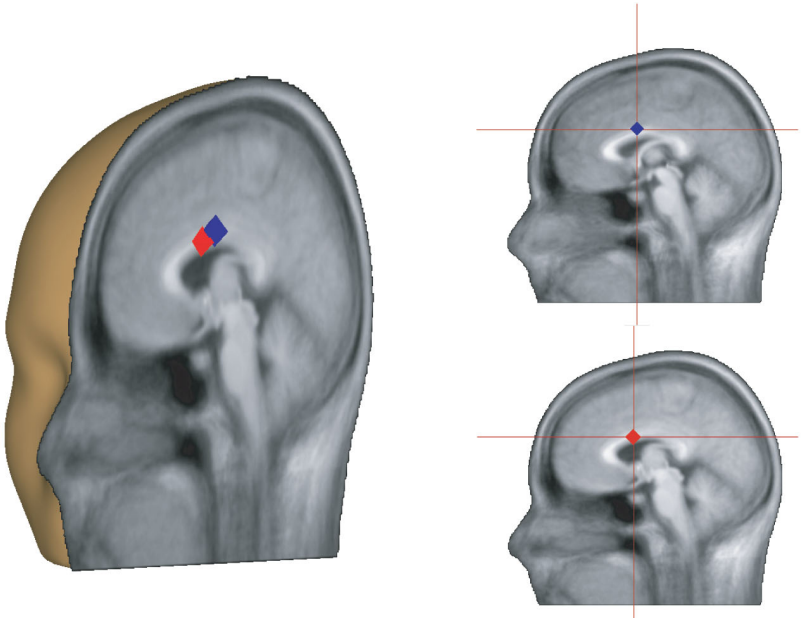

Figure 3 ERN source localization. Sagittal view of the brain showing the source for the ERN difference wave in the execution condition (blue) and in the observation condition (red). Left, displayed together within the same head model; right, projected onto a standard MRI template.

top right) suggests that the observer's motor cortex started to be activated before the actor's response. This activation was associated with greater negativity over the motor cortex contralateral to the correct response side, as viewed from the perspective of the observer. CSD topography also showed lateralized activation over posterior areas, probably related to processing of the preceding stimulus. This posterior activation was more prominent for incorrect trials, whereas motor activation was less prominent (**Fig. 4**, bottom right, +64 ms maps). This is due to a difference in response times between correct and incorrect trials (incorrect responses are faster), resulting in less time for the posterior activation to dissolve on incorrect trials.

Following the observation of a correct response, the LRP continued to develop, reaching a maximum 160 ms after the actor's response was observed. However, when the actor responded incorrectly, the correct lateralization in the observer (which had begun before the actor's response) rapidly decreased, and a widespread lateralized activity developed over parietal areas (**Fig. 4**, bottom right). Statistically, LRPs following observed correct and incorrect responses started to differentiate 146 ms after the actor's response ($t_{15} = -1.7645$, $P = 0.049$).

To summarize, LRPs in the observation condition indicate that, for both correct and incorrect trials, the correct response is initially activated by the observer's motor system. Following the actor's response, the observer's motor system is differentially activated as a function of the accuracy of the observed response.

DISCUSSION

The results of the present study provide insight into the neural mechanisms underlying action observation and error processing. Importantly, we found evidence that neural activity in both the medial frontal and the motor cortex is modulated by the correctness of both self-generated and observed responses. This suggests that similar neural mechanisms are involved in monitoring one's own actions and the actions of others.

Evidence for similar involvement of medial frontal cortex activity in both the execution and observation conditions is provided by source analysis of the negativities associated with errors. As in previous research, a medial frontal source reliably accounted for the ERN following self-generated errors and, importantly, the same source accounted for the negativity follow-

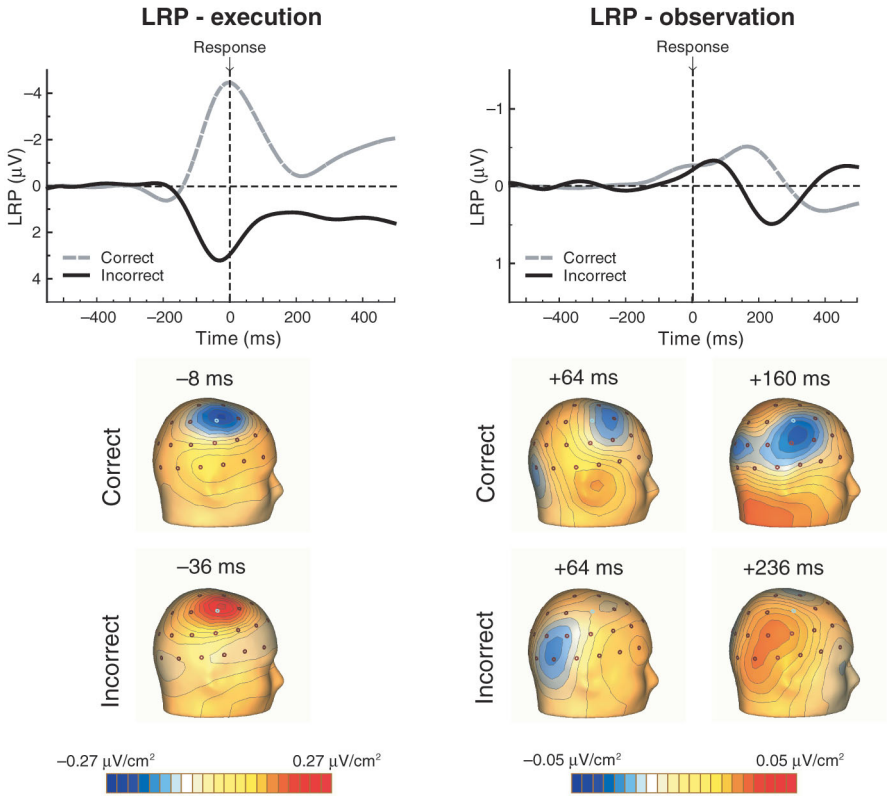

Figure 4 Lateralized readiness potentials. Top, response-locked lateralized readiness potentials in the execution condition (left) and the observation condition (right). LRPs recorded to correct response trials are indicated by dashed lines in gray, and LRPs to incorrect trials by solid lines in black. Bottom, CSD maps of LRP effects in the execution condition (left) and the observation condition (right), for correct and incorrect responses separately. The C3/C4 electrode over the lateral motor cortex is marked in light blue for reference. The relevant time-point (relative to the response) is indicated above each map.

ing observed errors. For this reason, we infer that an ERN is elicited by observing errors.

The relationship between the ERN and other ERP components, involving medial frontal cortex (*e.g.*, the N2), is currently a subject of debate[29]. The present data do not inform this debate. They do, however, enable the critical inference that medial frontal cortex is involved in processing both self-generated and observed errors. This suggests that the brain systems associated with action selection and error processing in the medial frontal cortex are also activated under conditions of action observation.

The data from the observation condition are compatible with those obtained by an earlier study[25]. However, the extent of the ERN in the present data was longer (230 ms versus 130 ms in the earlier study) and had a shorter onset latency. These differences are most likely due to a difference in the precision with which the observer could determine when the actor responded. In the present study, the observers watched a real actor, whereas in the earlier study, the behavior of a virtual actor was displayed in symbolic form on a computer monitor. In the latter case, the onset of the actor's movement could be precisely defined, whereas in the present case there could be ambiguity in the time at which the actor was judged to have responded. As a result, variability in the timing of the detection of the erroneous response would have occurred.

In contrast to the earlier study, data from the present study allow us to evaluate the activation of the observer's motor system when the actor executes correct and incorrect responses. LRPs in the observation condition showed that the observer's motor system was activated in two ways. First, motor cortex was active prior to the actor's response, suggesting that the observer generated a representation of the appropriate response following stimulus presentation. Then, following the actor's response, correct response activation continued to develop when the response was correct. However, following an incorrect response, differential motor activation decreased and activity lateralized over posterior areas. This lateralized activity may be associated with perceptual or attentional processing of the actor's incorrect hand movement.

The LRP results of the present study are consistent with and extend previous studies that report activation of the motor system in response to the observation of behavior[22–24,30–32]. Our data indicate that under conditions of action observation, subjects' motor activation may reflect subthreshold preparation of (correct) responses, and modulation by observed responses. Noteworthy is that the initial motor lateralization following the stimulus, and the subsequent lateralization following the observation of the correct response, were both found over the observer's motor cortex contralateral to the side of the correct response (from the observer's own perspective). Thus, the observer's LRP shows what the observer would have done if he/she had actively done the task him/herself[32], instead of maintaining a representation of the task from the perspective of the actor. This pattern of results is consistent with studies of imitation[33], which show that when imitating, subjects mostly try to replicate the goal of the action, but tend to ignore how the actor did it (*e.g.*, subjects may use their left hand although the actor used his/her right hand).

Given that activation in motor areas in the observation condition is similar to motor activation found with task execution, it is important to investigate the level at which there was covert response activation in the observer. To this end, we ran five additional participants from whom we recorded electromyogram (EMG) activity in both execution and observation conditions. Response-locked averages of high-pass filtered (20–100 Hz) and rectified EMG recordings from both hands showed a strong response with task execution, but no effect in the observation condition. This indicates that the level of covert response activation in the observer does not extend to the periphery[34].

Data from the present study suggest that neural mechanisms used to monitor individual task performance are also activated under conditions of task observation. These mechanisms may play a central role in our ability to predict and classify the behavior of others, and thus provide a possible pathway for observational learning.

METHODS

Participants. Eighteen volunteers with no known neurological impairments and normal or corrected-to-normal vision participated in the experiment. Data from one participant were discarded because of recording artifacts, and data from another participant were discarded because of excessive noise in the EEG. Thus we analyzed data from 16 participants (11 female, age range 19–34, mean 23.4). All participants received 6 euros per hour and provided written consent according to institutional guidelines of the local ethics committee (CMO region Arnhem-Nijmegen, Netherlands).

Apparatus and procedure. Two experimenters were continuously present during the experiment. One controlled the experimental measurements, while the second participated in the experiment. Participants were seated in front of a table facing an experimenter (**Fig. 1**). On the table were two custom-made joystick devices, positioned to the left and right of an LED stimulus device.

The LED device contained two display sides, one facing the participant, the other facing the experimenter. Both display sides contained five horizontally aligned dot matrices (13 mm wide, 18 mm high), each consisting of a 7×5 LED array. The size of the display was 77×18 mm, subtending a visual angle of $5.9 \times 1.4°$ at an average viewing distance of 75 cm. Stimuli consisted of left- and right-pointing arrowheads ($0.8 \times 1.4°$ each) that were generated by turning on a selection of LED dots.

Joysticks consisted of a 4-cm lever fitted in an electronic control box (10 cm wide and long, 3 cm high). The lever was constrained to move only in lateral directions and a pair of springs ensured that it would return to its original position after a response. Deviations of more than 5° from the relaxed position (maximum angle 30°) were measured as responses. Joysticks were positioned bilaterally to either side of the stimulus device, slightly in front of it (4 cm), at a viewing angle of 15.5°. Joystick movement generated no auditory cues.

Participants took part in two conditions: an execution condition in which they performed a choice reaction task, and an observation condition in which they observed an experimenter performing the same task. In this second condition, the participant is referred to as the 'observer' and the experimenter as the 'actor'.

The task consisted of a modified Eriksen flanker task[28], in which center arrowheads were presented in conjunction with four flanker arrowheads, two on each side, which either pointed 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 equal, as well as the probability of congruent and incongruent flankers. A trial sequence began with a centrally presented, diamond shaped, fixation point ($0.6 \times 0.6°$) that was displayed for 200 ms. Following a 200 ms stimulus-free interval, a target display was presented for 300 ms, showing the four flankers and the center arrowhead. A 600 ms stimulus-free interval completed the trial.

In the execution condition, participants were instructed to respond both quickly and accurately in the direction of the center arrowhead. Joysticks were moved with the thumb in an outward direction. Participants were instructed to give only one response per trial, and to try to avoid correcting initial errors. Also, participants were instructed to refrain from making eye movements and to reduce blinking during task performance.

The experimental session began with 40 practice trials to allow participants to familiarize themselves with the task. After this, they performed 8 runs of 100 trials of the task, each run taking approximately 2.2 min. Between runs, the participants were given feedback about their average response times and number of errors. One experimenter sat facing the participant and reported the number of observed errors after each run.

In the observation condition, which always followed the execution condition, participant and experimenter changed roles. Participants were now instructed to observe the behavior of the experimenter performing the Eriksen

flanker task and to count the number of errors made by the experimenter. In this way, we could confirm that the observer was engaged in the task. The observer's display only included the center arrowhead, ensuring that error detection was not compromised by the presence of flankers. The distance between the observer and the display was held constant, but joysticks were moved to the experimenter's side of the display (4 cm behind the LED device), resulting in a 12.4° viewing angle for each joystick relative to the center of the display. Participants were instructed to maintain fixation on the fixation point and to identify responses without making eye movements. All participants could view the stimulus and the actor's responses without moving their eyes. A total of 8 runs of 100 trials each were completed in this condition.

Behavioral recording and analysis. The onset of the target display and behavioral responses were sampled continuously at a frequency of 1,000 Hz. RTs, errors and misses were analyzed offline for individual stimulus types and conditions. Only trials with RTs in the 150–550 ms range were included for analysis. Responses with only the incorrect hand were labeled as 'pure' errors. Trials with responses from two hands were not included in the analysis.

Electrophysiological recording. Brain electrical activity was recorded from 47 Ag/AgCl electrodes, referenced to linked earlobes. Electrodes were mounted in an elastic cap (Easycap, Montage 10) configured for equal arrangement of the electrodes over the scalp. Vertical and horizontal electro-oculograms were recorded from sites above and below the left eye, and 1 cm outwards to the outer canthus of each eye, respectively. The electrode common was placed on the sternum. All electrode impedances were kept below 5 kΩ. EEG recordings were amplified and digitized at 250 Hz. Data were filtered offline, using a 1–14 Hz bandpass for the ERN analyses[35] and a 4 Hz lowpass for the LRPs[36], using Butterworth zero phase filters. Ocular artifact was corrected[37], whereas trials containing amplifier artifacts were discarded.

Error-related negativity. For both correct and incorrect trials in both conditions, a 600 ms epoch (baseline 100–0 ms before response) was extracted. To mitigate the effects of differential contribution from stimulus-related activity to the ERP, we adopted a matching procedure[38]. For each condition and for each participant, the data for each incorrect trial were randomly matched by RT (±4 ms) with the data for a corresponding correct trial. On average, about 90% of all error trials and 10% of all correct trials were matched for further analysis.

Lateralized readiness potential. LRPs were calculated using signals recorded from C3 and C4 electrodes. The average asymmetry, defined as the difference between C3 and C4, was derived by averaging the asymmetries associated with trials where the left movements were correct and those where right movements were correct according to the following equation: LRP = [left hand(C4 – C3) + right hand(C3 – C4)]/2. Negative values of the LRP indicate relative activation of the correct response, and positive values indicate relative activation of the incorrect response[26]. For both conditions, stimulus-locked LRPs (700 ms epoch, baseline –100 to 0 ms) and response-locked LRPs (–550 to 500 ms epoch, baseline –550 to –450 ms) were calculated. In the execution condition, LRPs were response-locked to the participant's own response, whereas in the observation condition the participant's (observer's) LRPs were response-locked to the actor's response.

To derive a topographical visualization of motor activation, the LRP equation was applied to all lateral electrode pairs. Lateralized effects were (arbitrarily) projected over the right hemisphere in the form of current source density (CSD) maps. These maps emphasize the difference in voltage across the scalp and provide an indication of the loci of the underlying neural sources.

ERP statistical analyses. For both ERN and LRPs, onset latencies and onset of the difference between correct and incorrect trial waveforms were assessed by a stepwise series of one-tailed serial *t*-tests (step size of 4 ms). For each test, data from a time window of 40 ms (*i.e.*, point of measure, ±20 ms) were averaged. The onset latency was defined as the first point at which five consecutive *t*-tests showed a significant difference ($P < 0.05$).

In the matching procedure, the pool of potential correct trials was larger than the pool of potential incorrect trials, resulting in an arbitrary selection of matched correct trials, which could result in variability associated with the particular set of matching correct trials chosen. In turn, this could lead to variability in the computation of the onset of the difference between correct and incorrect trial waveforms. For this reason, we used a bootstrapping procedure[39] to generate a distribution of onsets over different sets of matching trials. The matching procedure was run 500 times, and the mean onset time of the distribution was taken as an indication of the time at which correct and incorrect trials started to differ.

Source localization. ERN source localization was performed on the difference between grand-averaged incorrect and matched correct trial waveforms, using Brain Electric Source Analysis[40] (MEGIS software GmbH). For both the execution and observation condition, source analysis was performed for the interval in which the difference between correct and incorrect trials was statistically significant (between –6 and 146 ms and between 90 and 318 ms, respectively). A four-shell ellipsoidal head model was used. For details of source localization, see **Supplementary Methods** online.

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COMPETING INTEREST STATEMENT

The authors declare that they have no competing financial interests.

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