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Why do we make mistakes? Neurocognitive processes during the preparation–perception–action cycle and error-detection

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ABSTRACT

The event-related potential (ERP) literature described two error-related brain activities: the error-related negativity (Ne/ERN) and the error positivity (Pe), peaking immediately after the erroneous response. ERP studies on error processing adopted a response-locked approach, thus, the question about the activities preceding the error is still open. In the present study, we tested the hypothesis that the activities preceding the false alarms (FA) are different from those occurring in the correct (responded or inhibited) trials. To this aim, we studied a sample of 36 Go/No-go performers, adopting a stimulus-locked segmentation also including the pre-motor brain activities. Present results showed that neither pre-stimulus nor perceptual activities explain why we commit FA. In contrast, we observed condition-related differences in two pre-response components: the fronto-central N2 and the prefrontal positivity (pP), respectively peaking at 250 ms and 310 ms after the stimulus onset. The N2 amplitude of FA was identical to that recorded in No-go trials, and larger than Hits. Because the new findings challenge the previous interpretations on the N2, a new perspective is discussed. On the other hand, the pP in the FA trials was larger than No-go and smaller than Go, suggesting an erroneous processing at the stimulus-response mapping level: because this stage triggers the response execution, we concluded that the neural processes underlying the pP were mainly responsible for the subsequent error commission. Finally, sLORETA source analyses of the post-error potentials extended previous findings indicating, for the first time in the ERP literature, the right anterior insula as Pe generator.

Introduction

The ancient philosopher Seneca wrote "*errare humanum est, perseverare autem diabolicum*" (i.e., to err is human, but to persist in error is diabolical). Indeed, being aware of own errors is a crucial skill of the human brain. In the last decades neuroscientists investigated the neural substrates of error detection through electrophysiological techniques using cognitive tasks requiring decision making and motor responses, such as Odd-ball, Go/No-go, Flanker and Stop-Signal tasks (Braver et al., 2001; Debener et al., 2005; Matthews et al., 2005; Dhar et al., 2011). The error negativity (Ne; Falkenstein et al., 1991) or error-related negativity (ERN; Gehring et al., 1993), a frontal wave peaking at 50–100 ms after the erroneous response, is the most investigated error-related brain activity. After the Ne, at 200–400 ms after the erroneous response, a second activity, called error positivity (Pe; Falkenstein et al., 1994, 1996), is commonly observed in posterior areas. These two components are associated to different aspects of the error processing. The Ne is thought to reflect both the response conflict processing (Yeung et al., 2004) and the mechanism of early mismatch between the intended and actual response (Falkenstein et al., 1991; Coles et al., 2001). The main generator of the Ne was localized within the fronto-medial wall (FMW), specifically in the anterior cingulate cortex (ACC) and pre-supplementary motor area (pre-SMA) (Dehaene et al., 1994; Holroyd et al., 1998; Miltner et al., 1998; Luu et al., 2000; van Veen and Carter, 2002). However, recent evidences suggest also the contribution of a more distributed network in its generation, including the dorsolateral and ventrolateral prefrontal cortex (dlPFC and vlPFC), the cingulate motor area (CMA), and the lateral parietal cortex (typically Brodmann area 40) (Menon et al., 2001; Ullsperger and von Cramon, 2001; Brázdil et al., 2002; Garavan et al., 2003; Ramautar et al., 2006; Taylor et al., 2007). The Pe component is usually linked to the awareness of the error commission because its amplitude was larger in case of consciously perceived error than in the undetected error condition (Davies et al., 2001; Nieuwenhuis et al., 2001; Mathalon et al., 2003; Dhar et al., 2011). The main neural sources of the Pe component were localized in the ACC and parietal cortices (van Veen and Carter, 2002), while an intracerebral recording study suggested also the participation of the orbitofrontal cortex and the mesio-temporal regions (Brázdil et al., 2002). Further, recent evidences revealed an insular source of the Pe component, suggesting the

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involvement of this brain area in a more general process of error detection that includes both the conscious perception of response errors (Klein et al., 2007; Ullsperger et al., 2010; Dhar et al., 2011) and the detection of a failure in inhibition (Ramautar et al., 2006).

The main literature in this field typically investigated the processes related to the error detection stage, that is, the brain activities immediately following the fallacious response; consequently, the brain activity *prior* to the execution of the erroneous action has never been described. Thus, the fundamental question about what went wrong before we commit error is lacking. The main goal of the present study is to answer this question. To this aim, we used a Go/No-Go task, testing the hypothesis that the *false alarms* (FA; i.e. responses to No-go stimuli) could be associated to processing deficits taking place at one or multiple stages before response emission. We considered both the cognitive and premotor anticipatory processing in the pre–stimulus activities, and the post−stimulus stages such as perceptual processing, inhibitory−or conflict-related activities and stimulus-response mapping. Differently from previous studies, which used response-locked ERPs, we used a stimulus-locked ERPs with a large time window including both pre- and post-stimulus response activities. In previous works (Berchicci et al., 2014; Di Russo et al., 2013b; Perri et al., 2014, in press) we demonstrated that this method allows to investigate the typical post-stimulus ERPs (related to sensory, motor and cognitive processing) without masking the motor preparation and cognitive anticipation processes.

At pre-stimulus level, we might expect error-related modulations at one or multiple anticipatory activities. Specifically, three pre-stimulus processing should be considered. First, an increased negative activity on the right frontal electrodes could reflect an increased baseline of the accuracy decision system (Perri et al., 2014); this should restrict the possibility to accumulate enough sensorial evidences to reach the decision (Reddi and Carpenter, 2000; Usher and McClelland, 2001; Simen et al., 2006; Bogacz et al., 2010). Second, the FA could be caused by lapses of attention: this would be associated to a reduced PFC top-down control (e.g. Weissman et al., 2006), reflected by a reduction of the *prefrontal negativity* (pN) component over the frontopolar derivations (Perri et al., in press). Third, the FA could be the outcome of a defective processing in the motor unit during the motor preparation phase; this latter is mainly processed by the SMA activity, as electrophysiologically reflected by the Bereitschaftspotential (BP; Deecke and Kornhuber, 1978; Shibasaki and Hallett, 2006). An amplitude increase of this component could reflect a greater baseline level in the speed system (Perri et al., 2014), accounting for fast but inaccurate performance (Bogacz et al., 2010).

As regard the earliest stage of the post-stimulus phase, i.e. the sensory processing, we considered the visual P1 and N1 components. The P1 reflects task accuracy, since it makes possible to discriminate accurate performers (Perri et al., 2014), while the N1 component mainly reflects the orienting of attention to task-relevant stimuli (Luck et al., 1990; Vogel and Luck, 2000); thus, we may expect error-related modulations at these levels.

Finally, we evaluated two other post-stimulus components preceding the response emission: the well-know N2 component, usually peaking at 250–300 ms after the stimulus, and the recently described prefrontal positivity (pP) peaking about 300 ms after stimulus onset (Berchicci et al., 2014; Di Russo et al., 2013a, 2013b; Lucci et al., 2013; Perri et al., 2014). In the context of a Go/No-go task, the fronto-central N2 was mainly related to the inhibitory processing because it is usually enhanced when motor responses are correctly inhibited, such as in the case of No-go trials (e.g., Van Boxtel et al., 2001). However, the functional role of this component is still an open question. According to the inhibitory control theory (Bokura et al., 2001; Van Boxtel et al., 2001; Schmajuk et al., 2006), the N2 reflects the inhibitory control to No-go trials; thus, since FA represent instances of failed inhibition, the amplitude of N2 in case of FA is expected to be smaller than that recorded in correctly inhibited No-go trials, and comparable to the Go condition. On the other hand, according to the conflict monitoring theory (Nieuwenhuis et al., 2003; Donkers and van Boxtel, 2004), the enhancement of the N2 component should be linked to the high conflict level: this latter would mainly increase as effect of the low frequency stimuli, independently of their category (Botvinick et al., 2001). According to this view, the present task should induce a low conflict level, because Go and No-go stimuli had equal probability of occurrence. As consequence, we should not expect differences between the N2 amplitude of Go and No-go trials. In case of FA, literature on the N2 is currently lacking, because the typically adopted motor-locked segmentations mask the pre-response activities such as the N2.

The pP component is a positive wave peaking on the frontopolar derivations, and previously localized in the anterior insula (aIns; Di Russo et al., 2013b). We showed that the pP amplitude is larger in the Go than No-go trials, suggesting that it reflects the stimulus–response (S–R) mapping process finalized to response execution (Di Russo et al., 2013b; Berchicci et al., 2014; Perri et al., 2014). As also suggested by Boettiger and D'Esposito (2005), the function of aIns would be to trigger the motor response when enough action-related information are accumulated. Based on these latter suggestions we hypothesized that error-related activity could be detected also at this stage of processing, as probably reflected by a different pP amplitude of FA when compared to the other conditions. It is noteworthy that a frontal positive wave in the pP interval was also described by Makeig et al. (1999) that labeled it as *P3f*. Shortly after, Bruin et al. (2001) challenged the traditional view of the N2 suspecting that response activation (instead of response inhibition) processes might take place immediately before the response emission. Finally, in the same year when our group firstly described the pP (Di Russo et al., 2013a,b; Lucci et al., 2013), Gajewski and Falkenstein (2013) reached similar conclusions reporting the so-called *Go-P2* that can be assimilated to the present pP.

An additional goal of this study was to contribute to the knowledge about the neural sources of the post-error potentials, i.e., the Ne and Pe components. To this aim we used the Standardized Low Resolution Electromagnetic Tomography (sLORETA) method, which does not assume a priori generators.

Furthermore, a common issue in studying the error-related brain activity is represented by the low number of erroneous trials available, leading to a small signal to noise ratio. In the present study we tried to overcome this limit by selecting subjects from a large database and focusing only on participants who made a relevant number of errors.

Material and methods

Subjects

From a large database of 136 subjects who participated in the Go/No-go task (described below), we firstly excluded subjects who did not report FA, i.e. responses to No-go stimuli. Then, we processed the electroencephalographic (EEG) data of the remaining subjects ($n = 127$), selecting only the subjects with at least 20 artifact-free trials of FA. Following this procedure, 36 subjects were selected for further analyses (6 females; age mean = 38.9, SD = 11.3).

The participants had normal or corrected-to-normal vision and no history of neurological or psychiatric disorders; all of the subjects were right-handed (Edinburgh handedness inventory; Oldfield, 1971). After procedures were explained, all of the participants provided written informed consent, approved by the local Ethical Committee.

Procedure and task

Subjects were tested in a sound attenuated, dimly lit room; they were comfortably seated in front of a computer screen at a distance of 114 cm. A board was fixed on the armchair in order to allow participants to push with the right index finger the button panel positioned on it. Four visual stimuli (i.e. four squared configurations made by vertical

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