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Anxiety not only increases, but also alters early error-monitoring functions

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Anxiety has profound influences on a wide range of cognitive processes, including action monitoring. Event-related brain potential (ERP) studies have shown that anxiety can boost early error detection mechanisms, as reflected by an enhanced error-related negativity (ERN) following errors in high-anxious, as compared with low-anxious, participants. This observation is consistent with the assumption of a gain control mechanism exerted by anxiety onto error-related brain responses within the dorsal anterior cingulate cortex (ACC). However, whether anxiety simply enhances or, rather, alters early error detection mechanisms remains unsolved. In this study, we compared the performance of low- versus high-trait-anxious participants during a go/no-go task while high-density EEG was recorded. The two groups showed comparable behavioral performance, although levels of state anxiety increased following the task for high-anxious participants only. ERP results confirmed that the ERN/Ne to errors was enhanced for high-anxious, relative to low-anxious, participants. However, complementary topographic analyses revealed that the scalp map of the ERN/Ne was not identical between the two groups, suggesting that anxiety did not merely increase early error detection mechanisms, but also led to a qualitative change in the early appraisal of errors. Inverse solution results confirmed a shift within the ACC for the localization of neural generators underlying the ERN/Ne scalp map in high-anxious participants, corroborating the assumption of an early effect of anxiety on early error-monitoring functions. These results shed new light on the dynamic interplay between anxiety and error-monitoring functions in the human brain.

The early and efficient detection of a mismatch between the actual and expected or desired motor action provides human organisms with adaptive and flexible behaviors, since error detection typically leads to learning and the implementation of remedial action (Holroyd & Coles, 2002; Rabbitt, 1966). Converging neuroscientific evidence has revealed that the medial frontal cortex and the dorsal anterior cingulate cortex (ACC) are primarily involved in the early detection of errors or, more generally, conflicts, whereas lateral frontal or prefrontal regions are implicated in behavioral adjustments following errors (Carter et al., 1998; MacDonald, Cohen, Stenger, & Carter, 2000; Ridderinkhof, Nieuwenhuis, & Braver, 2007; van Veen & Carter, 2006). In this view, the medial frontal cortex (as well as the dorsal ACC) provides important cognitive control mechanisms, including early error detection. However, errors are also typically rare, deviant, and negative (arousing) events. Hence, errors also call for affective control processes (Hajcak & Foti, 2008; Ochsner & Gross, 2005), beyond their ubiquitous effects on cognitive control processes. Nonetheless, much less is known about the nature and extent of affective influences on early error detection processes, in comparison with a wealth of studies that have focused primarily on cognitive control effects (Ridderinkhof et al., 2007).

Event-related brain potential (ERP) studies have largely contributed to gaining new insight into the time course and neural bases of cognitive control mechanisms, including error detection (Taylor, Stern, & Gehring, 2007). The commission of errors is typically associated with the generation of a reliable negative ERP component early on following the onset of incorrect motor responses, the error-related negativity (ERN/Ne; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring, Coles, Meyer, & Donchin, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN/Ne component peaks at frontocentral electrodes along the midline (FCz or Fz electrode position), roughly 0–100 msec after (incorrect) response onset, and is thought to be primarily generated within the dorsal ACC (Debener, Ullsperger, Fiehler, von Cramon, & Engel, 2005; Dehaene, Posner, & Tucker, 1994; Herrmann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004; O'Connell et al., 2007; Vocat, Pourtois, & Vuilleumier, 2008). Hence, the ERN/Ne occurs too early to reflect sensorimotor or proprioceptive feedback; instead, it is assumed to reflect the automatic and rapid detection of a mismatch between the actual and expected or desired motor action (Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blow, Band, & Kok, 2001). A similar but smaller negativity

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is also usually observed after correct responses during speeded tasks and has been labeled the *correct-related negativity* (CRN; Falkenstein et al., 2000; Luu, Flaisch, & Tucker, 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). Following the ERN/Ne, the error positivity (Pe) is usually elicited in response to incorrect responses (Falkenstein et al., 2000; Nieuwenhuis et al., 2001). The Pe component is a broad positive deflection resembling the P3 component, peaking over the vertex (or more posterior parietal scalp positions along the midline, such as PZ) roughly 150–300 msec after (incorrect) response onset, with neural generators involving more posterior cingulate regions (Herrmann et al., 2004; O'Connell et al., 2007; Ridderinkhof, Ramautar, & Wijnen, 2009). Unlike the ERN/Ne, the Pe is thought to index a more elaborate stage of error detection, possibly reflecting the activation of remedial processes consecutive to the conscious detection/recognition of errors (Falkenstein et al., 2000; Nieuwenhuis et al., 2001; Ridderinkhof et al., 2009).

Although the ERN/Ne is usually described as reflecting cognitive or learning processes (Bernstein, Scheffers, & Coles, 1995; Coles, Scheffers, & Holroyd, 2001; Falkenstein et al., 1991), several ERP studies have shown that the ERN also captures variations in affect or motivation. This observation is consistent with the assumption that errors not only provide important learning or cognitive signals, but also convey an important emotional significance (Bush, Luu, & Posner, 2000; Gehring & Willoughby, 2002; Pailing & Segalowitz, 2004; Pourtois et al., 2010). For example, Hajcak, Moser, Yeung, and Simons (2005) suggested that an error is primarily a motivationally salient event, since the ERN was significantly larger for errors related to high monetary value. More evidence on the relationship between affect and the ERN has come from studies looking at variations in trait affect. Several researchers have found that individuals scoring high on trait anxiety and negative affect are characterized by increased ERNs (Boksem, Tops, Wester, Meijman, & Lorist, 2006; Hajcak, McDonald, & Simons, 2003, 2004; Olvet & Hajcak, 2008). This increased sensitivity for errors in individuals with anxiety characteristics suggests that the ERN also somehow reflects an affective evaluation during error detection (Bush et al., 2000; Olvet & Hajcak, 2008). By contrast, later stages of error monitoring (reflected in the Pe) seem to be resistant to individual differences in affect (Hajcak et al., 2004; Holmes & Pizzagalli, 2008; Vocat et al., 2008), emphasizing the distinction between these two early error-related ERP components.

Interestingly, research on anxiety-related differences in ERN has not been linked to the broader literature on cognitive control in anxiety. The cognitive literature on anxiety allows one to derive relatively specific predictions in this context. According to the processing efficiency theory (Eysenck & Calvo, 1992), trait anxiety influences the efficiency (rather than the amount or effectiveness) of cognitive performance. Eysenck and Calvo claim that anxious individuals will not show performance decrements on most tasks as they recruit extra processing resources, which eventually hampers the amount of resources available for concurrent task processing. In this model, attentional control is the key mediating factor between anxiety and cognitive performance (Eysenck, Derakshan, Santos, & Calvo, 2007). This theory predicts that attention is more readily allocated to internal threatening stimuli (i.e., worrying thoughts) in high-anxious participants, reducing therefore the attentional focus on the current task demands. However, to maintain a standard level of performance, anxious participants compensate for this reduced efficiency by increasing cognitive efforts. This mechanism could potentially account for the fact that whereas an altered ERN is generally observed in high-, as compared with low-, anxious participants, no direct effect of anxiety on behavior (e.g., the number of errors) is usually evidenced (Hajcak et al., 2003; Vocat et al., 2008). Since our discussion of error detection suggests that errors are associated with cognitive as well as affective correlates, the attentional control theory would predict that errors in high-anxious individuals not only are associated with an increased ERN related to ACC activity, but also will be related to a different pattern of neural activity in areas involved in emotion processing and cognitive control.

Brain-imaging studies have confirmed that increased effort translates as enhanced activation in brain regions associated with cognitive control, including the dorsolateral or ventrolateral prefrontal cortex (DLPFC or VLPFC) or the dorsal ACC (Cazalis et al., 2003; Donohue, Wendelken, & Bunge, 2008; Wagner, Maril, Bjork, & Schacter, 2001). Interestingly, anxiety was found to reduce activation in these cognitive control areas (Bishop, 2007; Bishop, Duncan, Brett, & Lawrence, 2004). Moreover, a reduced efficiency might actually result from a change in the temporal recruitment of these cognitive control areas, as has recently been shown (Braver, Gray, & Burgess, 2007; Fales et al., 2008). These findings therefore suggest that anxiety may alter the recruitment of cognitive control areas during task performance and, as a result, lead to a reduced processing efficiency. However, with respect to error detection mechanisms (which are a crucial component of cognitive control), to our knowledge, no study has examined whether low- and high-anxious participants differ only in their reaction to errors (as primarily reflected by the size of the ERN component) or, alternatively, also make use of different cognitive control, and more specifically error detection brain networks during the early processing of these negative events.

The goal of this study was to address this question, using a modern ERP topographic-mapping technique (Murray, Brunet, & Michel, 2008; Pourtois, Delplanque, Michel, & Vuilleumier, 2008). More specifically, our aim was to test whether trait anxiety merely enhances early error-related brain activities or, alternatively, may alter the expression (and not only the strength) of these brain responses (as revealed by a topographic change of the ERN/Ne scalp map with anxiety), in keeping with the main prediction of the processing efficiency theory (Eysenck et al., 2007). We therefore compared, using high-density EEG, the electrophysiological responses to commission errors in two groups of healthy participants, differing only with respect to their subclinical levels of trait anxiety. We used a speeded go/no-go task, previously validated in a group

of adult participants (Vocat et al., 2008). The added value of this task is that it enables one to collect a high number of commission errors (consisting of false alarms [FAs] on no-go trials) for each participant, despite interindividual differences in reaction times (RTs), within a short period of time (~30 min) and without inducing excessive frustration. This was an important prerequisite for computing reliable ERP waveforms on the basis of a substantial number of trials, including for errors. Furthermore, neutral stimuli (i.e., colored arrows) were used during this task in such a way that electrophysiological responses to errors committed with neutral stimuli could be compared between the two groups and a relatively pure modulation of trait anxiety on these brain responses could eventually be assessed. On the basis of the evidence reviewed above, we predicted that behavior would not differ between low- and high-anxious participants and that high-anxious participants would show larger ERN/Ne for errors than would low-anxious participants (Olvet & Hajcak, 2008). We also surmised a substantial change in the configuration of the electric field of the ERN/Ne for high-anxious, relative to low-anxious, individuals. This result would suggest a change in the configuration of the neural generators of the ERN, possibly reflecting the activation of additional brain structures in high-anxious participants, in agreement with the processing efficiency theory (see Eysenck et al., 2007; Fales et al., 2008). More specifically, in this study, we examined whether the effect of anxiety on processing efficiency during the early detection of errors is associated with (1) an increased activation of the same brain structures in high-anxious, as compared with low-anxious, individuals; (2) the activation of partly overlapping neural generators, but with the recruitment of additional structures in high-anxious individuals; or (3) the activation of completely different neural networks in these two groups. Whereas the first hypothesis would indicate a stronger reaction to response errors in anxious participants, the second and third hypotheses suggest, instead, not only that high-anxious individuals respond more strongly to their own response errors, but also that they likely recruit a different network of cognitive control brain regions during this process, relative to low-anxious individuals.

METHOD

Participants

Four hundred seventy-nine first-year university psychology students were asked to fill out several questionnaires, including measures of anxiety (primarily screened using a validated Dutch version of the Spielberger State Trait Inventory–Trait Version [STAI–T]; Spielberger, 1983, translated by Defares, van der Ploeg, & Spielberger, 1979), in exchange for course credits. Within this large sample of psychology students, individuals scoring within the lowest quartile (range, 20–32; $n = 120$) and the highest quartile (range, 51–75; $n = 120$) of the distribution of trait anxiety scores were invited to participate in the ERP experiment in such a way as to obtain two homogeneous groups, being comparable with respect to age, gender, and handedness and differing only in their average levels of subclinical trait anxiety. A total of 32 undergraduate psychology students eventually agreed to freely take part in this experiment in exchange for €20 payment. Sixteen of them (belonging to the lowest quartile) were assigned to the low-anxious group (STAI–T, $M = 29.69$, $SEM = 0.80$, range = 25–36; 2 male; 2 left-handed; age, $M = 18.56$ years, $SEM = 0.26$), whereas the other 16 individuals (belonging to the highest quartile) were assigned to the high-anxious group (STAI–T, $M = 51.50$, $SEM = 0.99$, range = 45–58; 2 male; 3 left-handed; age, $M = 19.06$ years, $SEM = 0.39$). None of the participants had a history of psychological or neurological disease. They were free of any psychoactive medication and had normal or corrected-to-normal vision. The study was approved by the local university ethical committee, and all the participants signed an informed consent before the beginning of the experiment.

Speeded Go/No-Go Task

We used a modified version of a speeded go/no-go task previously validated with a group of healthy participants (see Figure 1; Vocat et al., 2008). Visual stimuli were shown on a 17-in. LCD screen. They consisted of an arrow (11.4º × 0.05º of visual angle at a 60-cm viewing distance) that was presented in the center of the screen on a white background. Each trial started with a blank screen that lasted for 1,000 msec. Then a black arrow (i.e., cue), oriented either up or down, was presented. After a variable interval ranging from 1,000 to 2,000 msec, the black arrow became either green (i.e., target) or turquoise, while its orientation could either remain identical or shift in the opposite direction. The participants were asked to perform a speeded color plus orientation discrimination task. When the black arrow turned green and the orientation remained unchanged, the participants were instructed to press the space bar as quickly as possible with a predefined finger of their dominant hand (go trials). However, the participants had to withhold responding either when the arrow became green but changed orientation or when the arrow became turquoise and kept its initial orientation, enabling two types of no-go trials (based on either the orientation or the color). For no-go trials, this color arrow remained on the screen for a maximum duration of 1,000 msec. Instructions emphasized both speed and accuracy.

We used an online adaptive algorithm to set up a limit for "correct"/fast RT (i.e., deadline procedure). The rationale for this procedure was to facilitate the occurrence of fast decisions and, hence, the occasional making of errors on no-go trials. The participants had to respond quickly on go trials, but their performance actually depended on this strict time limit, updated on a trial-by-trial basis. At the beginning of the experiment, the RT limit was set to 300 msec (this cutoff was determined on the basis of previous pilot testing). This limit was adjusted online as a function of the immediately preceding trial history—more specifically, as the sum of current and previous RTs divided by two. This procedure was found to be particularly efficient for producing a high number of commission errors within a short period of time. For any given go trial, the actual RT was always compared with the RT on the previous go trial. If the current RT was longer than the previous RT, the participant received a negative feedback (red dot). If the RT was shorter than the previous one, a positive feedback (green dot) was presented. Hence, feedback was used to stress both speed and accuracy. When the response was incorrect—that is, either an FA (response on a no-go trial) or an omission (absence of response on a go trial), the red dot was presented. A similar negative feedback was displayed after correct go responses, but ones performed too slowly as compared with the arbitrary RT cutoff. By contrast, the participants received a positive feedback (green dot) when they correctly withheld responding on no-go trials or when they responded quickly enough (see above for the exact procedure) on go trials. This procedure ensured obtaining many FAs on no-go trials, despite fluctuations in speed on a trial-by-trial basis, because this arbitrary cutoff for correct responses was updated and adjusted online after each trial, separately for each participant.

The experiment consisted of 20 practice trials and 360 test trials. The test trials were divided into six blocks of 60 trials each (40 go trials and 10 no-go trials of each type). Trial presentation was randomized within blocks. After every block, the experimenter emphasized the importance of speed, as well as accuracy, in this task. Stimulus presentation and response recording were controlled using E-Prime software (V2.0, www.pstnet.com/products/e-prime/).

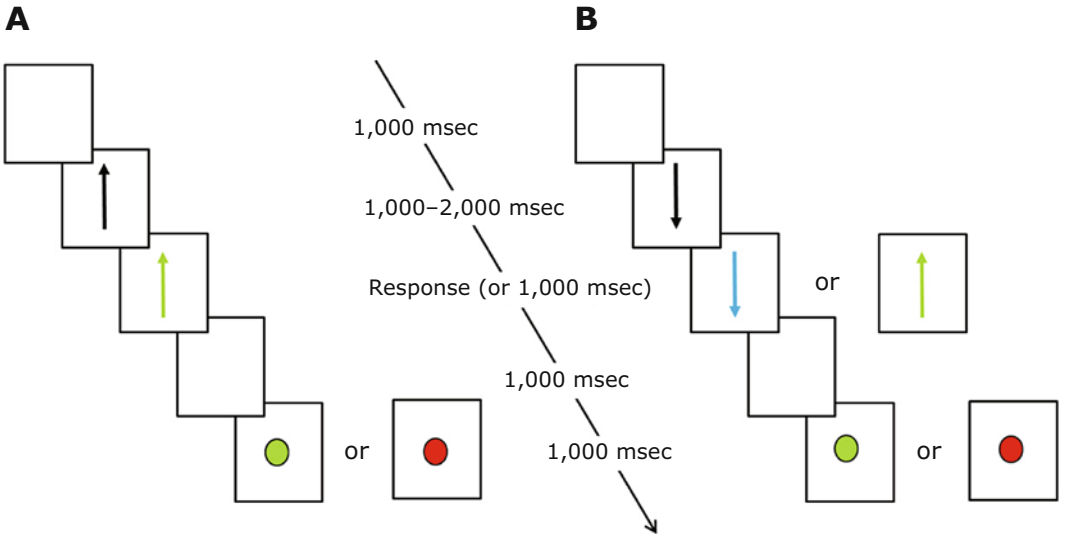

Figure 1. Stimuli and task. On each trial, a black arrow was presented (either upright or inverted). After a variable interval of 1,000–2,000 msec, the black arrow turned either green or turquoise. Participants had to respond by pressing the space bar as quickly as possible only when the arrow became green and kept its initial orientation (A), but not otherwise (B). Either a positive feedback (a green dot, when the participant was fast enough on go trials or inhibited correctly on no-go trials) or a negative feedback (a red dot, when the participant was too slow on go trials or wrongly pressed the space bar on no-go trials) reflecting actual task performance was presented for 1,000 msec. Speed for correct responses (on go trials) was calculated and adjusted online on a trial-by-trial basis (see the Method section).

Questionnaires

The 32 participants filled out the Dutch version of the STAI–T (Defares et al., 1979; Spielberger, 1983), and their scores served as a basis to form the low- and high-anxious groups. Because anxiety is typically related to punishment sensitivity (Bijttebier, Beck, Claes, & Vandereycken, 2009; Gray, 1982), the participants also completed the Dutch version of the Behavioral Inhibition Sensitivity (BIS)/Behavioral Activation Sensitivity (BAS) scales (Carver & White, 1994; Franken, Muris, & Rassin, 2005). Importantly, we also measured levels of state anxiety for these 32 participants, both before and after the go/no-go task, using the STAI–S.

EEG Acquisition and Analysis

Continuous EEG was acquired at 512 Hz using a 128-channel (pin-type) Biosemi Active Two system (www.biosemi.com) referenced to the CMS-DRL ground. ERPs of interest were computed offline following a standard sequence of data transformations (Picton et al., 2000): (1) −500/+1,000 segmentation around the motor response, (2) preresponse interval baseline correction (from −500 msec to motor response), (3) vertical ocular correction for blinks (Gratton, Coles, & Donchin, 1983) using the difference amplitude of two electrodes attached above and below the left eye, (4) artifact rejection [$M = -75/+75\ \mu$V, $SEM = 2$, amplitude scale across participants; no significant difference between low-anxious ($M = 76.56$, $SEM = 2.84$) and high-anxious ($M = 72.5$, $SEM = 2.81$) participants was observed, $t(30) = 1.02$, $p > .10$], (5) averaging of trials for each of the four experimental conditions (fast hits, slow hits, color FAs, and orientation FAs), and (6) low-pass digital filtering of the individual average data (30 Hz).

We focused primarily on two well-documented error-related ERP components following motor execution (Falkenstein et al., 2000)—that is, the ERN/Ne, with a maximum negative amplitude over frontocentral electrodes along the midline (electrode FCz) early on following motor execution (0–100 msec post-response-onset), immediately followed by the Pe component (150–300 msec post-response-onset), with a maximum positive amplitude over more posterior and central locations along the midline (electrode Cz). Hence, we performed a conventional peak analysis for each of these two error-related ERP deflections (Picton et al., 2000). For each ERP component and each condition separately, we calculated the area under the curve, during the 30- to 60-msec interval post-response-onset at electrode FCz for the ERN/Ne amplitude and during the 180- to 270-msec interval post-response-onset at electrode Cz for the Pe component. The selection of these two specific scalp locations (and time window) was based on the topographic properties of the present data set, as well as on converging results obtained in previous ERP studies for these two electrode positions (Dehaene et al., 1994; Gehring et al., 1990; Hajcak et al., 2003). A mixed model ANOVA including the between-subjects factor of anxiety (low anxiety vs. high anxiety) and the within-subjects factor of accuracy (error vs. hit) was performed on the mean amplitude of each area, with a significance alpha cutoff set to $p < .05$.

Topography

In order to capture more global ERP differences between low- and high-anxious individuals during the detection of errors, a detailed topographic-mapping analysis of the ERP data was next performed, following a conventional data analysis scheme (Michel, Seeck, & Landis, 1999; Michel et al., 2001; Murray et al., 2008; Pourtois, Dan, Grandjean, Sander, & Vuilleumier, 2005; Pourtois et al., 2008; Pourtois, Thut, Grave de Peralta, Michel, & Vuilleumier, 2005).

To precisely characterize topographic modulations over time and conditions, we applied a pattern or spatial cluster analysis procedure. The pattern analysis efficiently summarizes ERP data by a limited number of field configurations, previously referred to as *functional microstates* (Lehmann & Skrandies, 1980; Michel et al., 1999). Here, we performed a topographic pattern analysis on group-averaged data from −150 to 450 msec after response onset (300 consecutive time frames at a 512-Hz sampling rate), using a standard cluster (or spatiotemporal segmentation) method (K-means; Pascual-Marqui, Michel, & Lehmann, 1995) and then fitted the segmentation results back to individual data for subsequent statistical testing. The rationale and basic principles of this temporal segmentation method have been extensively described elsewhere (Michel et al., 1999; Murray

et al., 2008). The spatiotemporal segmentation algorithm is derived from spatial cluster analysis (Pascual-Marqui et al., 1995) and allows the identification of the most dominant scalp topographies appearing in the group-averaged ERPs of each condition and over time, while minimizing the biases for the selection of time frames or electrodes of interest. Importantly, this procedure allows identifying dominant scalp topographies, irrespective of (local or global) changes in amplitude (Michel et al., 1999; Murray et al., 2008). The optimal number of topographic maps explaining the whole data set is determined objectively using both cross-validation (Pascual-Marqui et al., 1995) and Krzanowski–Lai (Tibshirani, Walther, & Hastie, 2001) criteria. The dominant scalp topographies (identified in the group-averaged data) are then fitted to the ERPs of each individual participant using spatial-fitting procedures to quantitatively determine their representation across participants and conditions. This procedure thus provides fine-grained quantitative values, such as the duration of a specific topographic map or its global explained variance (GEV, or goodness of fit), which are critical indices of the significance of a given topography, not available otherwise in a classical component analysis (Picton et al., 2000). GEV represents the sum of the explained variance weighted by the global field power at each moment in time. GEV was entered in a mixed model ANOVA with two within-subjects factors—accuracy (error vs. hit) and map configuration (i.e., the electric field distributions previously identified by the spatial cluster analysis)—and with anxiety (low anxiety vs. high anxiety) as a between-subjects factor. These topographic analyses were carried out using CARTOOL software (Version 3.34; developed by D. Brunet, Functional Brain Mapping Laboratory, Geneva, Switzerland).

Source Localization

Finally, to estimate the likely neural sources underlying the electrical field configurations identified by the previous analyses, we used a specific distributed linear inverse solution—namely, standardized low-resolution brain electromagnetic tomography (sLORETA; Pascual-Marqui, 2002). Source localization was therefore used as a secondary/confirmatory analysis, relative to the primary topographic analyses (see Michel et al., 1999; Murray et al., 2008; Pourtois et al., 2008). sLORETA is based on the neurophysiological assumption of coherent coactivation of neighboring cortical areas (known to have highly synchronized activity; see Silva, Amitai, & Connors, 1991), and accordingly, it computes the "smoothest" of all possible activity distributions (i.e., no a priori assumption is made on the number and locations of the sources). Mathematical validation of this distributed source localization technique has recently been demonstrated (Sekihara, Sahani, & Nagarajan, 2005). sLORETA solutions are computed within a three-shell spherical head model co-registered to the MNI152 template (Mazziotta et al., 2001). The source locations were therefore given as (x, y, z) coordinates (x from left to right; y from posterior to anterior; z from inferior to superior). sLORETA estimates the 3-D intracerebral current density distribution in 6,239 voxels (5-mm resolution), each voxel containing an equivalent current dipole. This 3-D solution space in which the inverse problem is solved is restricted to the cortical gray matter (and hippocampus). The head model for the inverse solution uses the electric potential lead field computed with a boundary element method applied to the MNI152 template (Fuchs, Kastner, Wagner, Hawes, & Ebersole, 2002). Scalp electrode coordinates on the MNI brain are derived from the international 5% system (Jurcak, Tsuzuki, & Dan, 2007). The calculation of all reconstruction parameters was based on the computed common average reference. sLORETA units were scaled to amperes per square meter (A/m^2).

RESULTS

Behavioral Results

RTs shorter than 150 msec ($M = 0.83\%$) and longer than 500 msec ($M = 1.18\%$) were removed from the subsequent analyses. The numbers of excluded trials did not differ between groups [$t(30) = 0.52$, $p > .10$]. Color and orientation FAs were collapsed together (error condition), since there was no significant difference between the two error types. Likewise, fast hits and slow hits were collapsed and treated as a single condition (hit condition). Accuracy and RT data are presented in Table 1. The selected task turned out to be efficient in inducing a high number of unavoidable errors, consisting of FAs on no-go trials. By contrast, the number of omissions (i.e., absence of overt response on go trials) remained very low (<1%), such that only response errors corresponding to FAs were used in the analyses of ERP data (see below). Error rates (FAs) did not differ between the low-anxious group (number, $M = 38.81$, $SEM = 3.84$; percentage, $M = 32.43$, $SEM = 3.20$) and the high-anxious group (number, $M = 46.56$, $SEM = 3.98$; percentage, $M = 38.80$, $SEM = 3.32$) [$t(30) = 1.40$, $p > .10$]. Likewise, comparable hit rates were observed for the low-anxious group (number, $M = 237.81$, $SEM = 0.74$; percentage, $M = 99.09$, $SEM = 0.31$) and the high-anxious group (number, $M = 238.50$, $SEM = 0.62$; percentage, $M = 99.38$, $SEM = 0.26$) [$t(30) = 0.71$, $p > .10$]. As was expected, the participants were quicker for FAs than for hits [$F(1,30) = 296.58$, $p < .001$]. However, the speed did not differ between low- and high-anxious individuals ($F < 1$), and the interaction between accuracy and anxiety did not reach statistical significance ($F < 1$). Moreover, no group difference in efficiency (computed as the ratio between accuracy and speed; see Stoeber & Eysenck, 2008, for an exact formula) was observed [$t(30) = 0.84$, $p > .10$]. These results suggest comparable behavioral performance in these two groups.

During the go/no-go task, a classical posterror slowing effect was observed (Laming, 1979; Rabbitt, 1966). Consistent with a systematic adaptation following errors, RTs were reliably longer for hits immediately following errors ($M = 301.31$, $SEM = 4.18$) than for hits following another hit ($M = 292.52$, $SEM = 3.17$) [$F(1,30) = 7.46$, $p < .05$]. The magnitude of the posterror slowing effect did not differ between groups ($F < 1$).

Table 1
Behavioral Results During the Go/No-Go Task, Separately for Low- and High-Anxious Participants

	Reaction Time (msec)				Accuracy (Number)			
	Errors		Hits		Errors		Hits	
Anxiety	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
Low	264.99	4.61	298.67	4.04	38.81	3.84	237.81	0.74
High	261.08	4.93	292.44	4.68	46.56	3.98	238.50	0.62

Questionnaires

As was expected, the level of state anxiety before the task differed significantly between the two groups [low trait anxiety, $M = 31.62$, $SEM = 1.11$, range = 25–42; high trait anxiety, $M = 38.12$, $SEM = 1.56$, range = 31–51; $t(30) = -3.39$, $p < .01$]. After the go/no-go task, this level of state anxiety reliably increased [$F(1,30) = 5.20$, $p < .05$], although low-trait-anxious individuals still had a lower level of state anxiety ($M = 33.50$, $SEM = 2.17$) than did high-trait-anxious individuals ($M = 41.69$, $SEM = 1.56$) [$t(30) = 3.06$, $p < .01$]. Planned comparisons revealed that this increase of state anxiety after, relative to before, the go/no-go task was significant only in high-anxious participants [$t(15) = 2.11$, $p = .05$], but not in low-anxious participants [$t(15) = 1.11$, $p > .10$; see Table 2]. There was also a significant positive correlation between trait anxiety and state anxiety at baseline ($r = .52$, $p < .01$), but not between trait anxiety and the difference state anxiety score ($r = .17$, $p > .10$). These results suggest a differential influence of the speeded go/no-go task on subjective levels of state anxiety in low- versus high-anxious participants.

Moreover, the scores on the BIS/BAS further confirmed that the two groups differed significantly with respect to the trait-related anxiety characteristic, punishment sensitivity. BIS scores were significantly higher for high-trait-anxious participants ($M = 22.00$, $SEM = 0.67$, range = 18–26) than for low-trait-anxious participants ($M = 19.13$, $SEM = 0.49$, range = 16–22) [$t(30) = -3.48$, $p < .01$]. No significant difference between the low- and high-trait-anxious groups was evidenced for the BAS scores [BAS–drive: low trait anxiety, $M = 11.69$, $SEM = 0.27$; high trait anxiety, $M = 11.63$, $SEM = 0.43$; $t(30) = 0.12$, $p > .10$; BAS–fun: low trait anxiety, $M = 11.63$, $SEM = 0.24$; high trait anxiety, $M = 11.81$, $SEM = 0.46$; $t(30) = -0.36$, $p > .10$; BAS–reward: low trait anxiety, $M = 15.19$, $SEM = 0.34$; high trait anxiety, $M = 15.56$, $SEM = 0.44$; $t(30) = -0.67$, $p > .10$].

ERP Results

Consistent with many previous ERP studies (Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1993; Hajcak et al., 2004; Nieuwenhuis et al., 2001), we recorded two distinct and conspicuous error-related ERP components following motor execution, which have previously been associated with error detection brain mechanisms—that is, the error-related negativity (ERN/Ne) and error positivity (Pe). During the speeded go/no-go task, the commission of errors was unambiguously associated with the generation of these two well-characterized error-related ERP components (Figure 2).

Table 2
State Anxiety Scores (STAI–S) Before and After Performing the Speeded Go/No-Go Reaction Time Task

Trait Anxiety	State Anxiety			
	Before		After	
	M	*SEM*	*M*	*SEM*
Low	31.62	1.11	33.50	2.17
High	36.56	1.00	41.44	1.48

ERN/Ne. When participants made errors, there was a clear sharp negative deflection that peaked roughly 40–50 msec post-response-onset, with a maximum amplitude at frontocentral electrodes along the midline, including FCz (Figure 2). These electrophysiological properties are consistent with the ERN/Ne. Consistent with previous ERP studies (Falkenstein et al., 1991; Gehring et al., 1993), the amplitude of the ERN/Ne was reliably larger for errors ($M = -3.11$, $SEM = 0.59$) than for hits ($M = -1.62$, $SEM = 0.52$) [$F(1,30) = 22.02$, $p < .001$]. An ANOVA performed on the amplitude values of the ERN/Ne, as measured at the standard electrode FCz, disclosed a nearly significant interaction between anxiety and accuracy [$F(1,30) = 3.53$, $p = .07$]. As compared with hits (CRN), errors elicited a larger ERN/Ne component in high-anxious participants [$t(15) = 4.61$, $p < .001$; see Figure 2E] than in low-anxious participants [$t(15) = 2.00$, $p = .06$; see Figure 2B]. However, a direct comparison of the ERN/Ne between high-anxious ($M = -3.95$, $SEM = 0.65$) and low-anxious ($M = -2.27$, $SEM = 0.97$) participants did not reach statistical significance [$t(30) = 1.46$, $p > .10$]. Likewise, for hits, the early negativity—that is, the CRN (Burle, Roger, Allain, Vidal, & Hasbroucq, 2008; Coles et al., 2001; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003)—was comparable across the two anxiety groups [low anxiety, $M = -1.37$, $SEM = 0.83$; high anxiety, $M = -1.86$, $SEM = 0.63$; $t(30) = -0.54$, $p > .10$]. Note that because our speeded go/no-go task was quite demanding and uncertainty about accuracy (at the time of motor execution) was presumably equally high for errors and hits, it was not surprising to find a large CRN component for correct hits in this study (see also Pailing & Segalowitz, 2004). Importantly, the CRN component was still reliably smaller in amplitude than was the ERN/Ne in both low-anxious ($p = .06$) and high-anxious ($p < .001$) participants. Several authors have already pointed out the electrophysiological similarities between the ERN/Ne and the CRN (Allain, Carbonnell, Falkenstein, Burle, & Vidal, 2004; Vidal et al., 2003; Vidal et al., 2000). These authors argued that the ERN/Ne (and CRN) might reflect either a more general comparison process (active after both errors and correct responses) or an emotional/arousal reaction (instead of an error detection process per se).

Pe. For errors, the ERN/Ne was immediately followed by a large positive potential, with maximum amplitude over more posterior scalp positions, including Cz. This positive component was strongly attenuated for correct hits (Figure 2). These electrophysiological properties are compatible with the error-related Pe component (Falkenstein et al., 2000; Ridderinkhof et al., 2009).

FAs on no-go trials elicited a large Pe, relative to correct hits (Figure 2). However, this accuracy effect at the level of the Pe component was similar for low- and high-anxious participants, unlike what was found for the ERN/Ne. Statistical analyses confirmed these observations. An ANOVA performed on the mean amplitude of the Pe recorded at electrode Cz revealed a main effect of accuracy

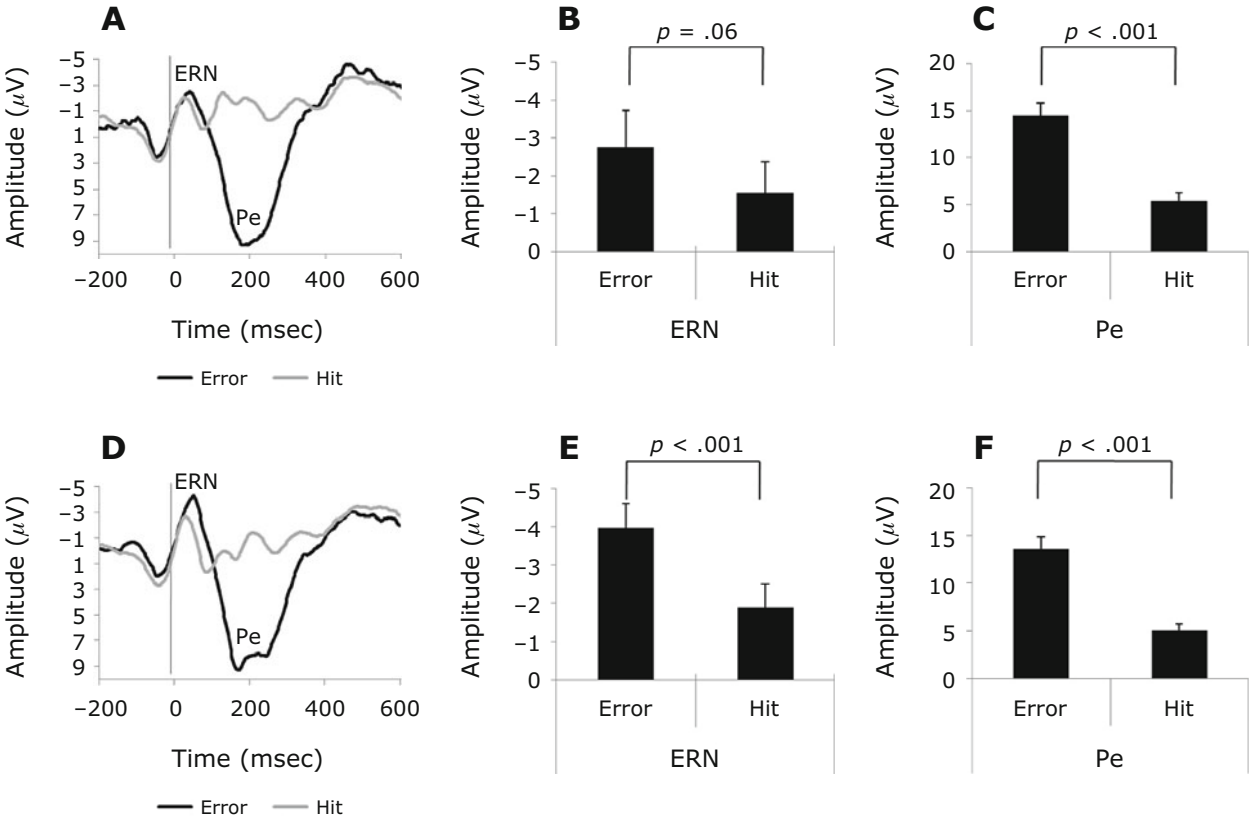

Figure 2. ERP results. (A) Grand average ERP waveforms (electrode FCz) for low-anxious participants. (B) Mean amplitude (μV; ±1 standard error of the mean) of the ERN/Ne for errors and hits in low-anxious participants. (C) Mean amplitude (μV; ±1 standard error of the mean) of the Pe for errors and hits in low-anxious participants. (D) Grand average ERP waveforms (electrode FCz) for high-anxious participants. (E) Mean amplitude (μV; ±1 standard error of the mean) of the ERN/Ne component for hits and errors in high-anxious participants. (F) Mean amplitude (μV; ±1 standard error of the mean) of the Pe component for hits and errors in high-anxious participants. The difference in ERN/Ne amplitude between errors and hits was larger in high-anxious than in low-anxious participants. No such interaction effect was observed for the amplitude of the Pe component.

[$F(1,30) = 146.29$, $p < .001$], indicating a much larger Pe component for errors ($M = 13.95$, $SEM = 1.31$) than for correct hits ($M = 4.90$, $SEM = 1.24$). This significant accuracy effect was not influenced by trait anxiety (Figures 2C and 2F) ($F < 1$).

Results of Topographic Analyses

Following standard practice (Michel et al., 1999; Murray et al., 2008; Pourtois et al., 2008), the topographic segmentation analysis was first performed using a broad temporal window, starting 150 msec before response onset and ending 450 msec after response onset (i.e., 300 consecutive time frames, corresponding to 600 msec), encompassing the two main error-related ERP components (ERN/Ne and Pe). A solution with 10 maps explained 97% of the variance. Remarkably, during the time interval corresponding to the ERN/Ne and CRN components (~20–50 msec post-response-onset), we found that the scalp distribution for errors had a different configuration for high-anxious than for low-anxious participants, whereas the scalp distributions for correct hits were similar between these two groups (Figure 3). Hence, the scalp maps corresponding to the CRN were similar between the two groups (Figure 3A; Map 1), whereas differential distributions of the negative activity over frontocentral electrodes were evidenced between low- and high-anxious participants for errors (ERN/Ne). Clearly, the frontocentral negative activity associated with errors (ERN/Ne) showed a broader and more extended (pre)frontal distribution for high-anxious participants (Figure 3C; Map 3), relative to low-anxious participants (Figure 3B; Map 2), where this negative activity was clearly circumscribed to a few electrode positions, including FCz. This result showed, therefore, a change in the configuration of the electric field (topography), regardless of (local) variations in amplitude (ERPs; see the Method section).

These observations were further verified by statistical analyses performed on the topographic data—that is, the GEV obtained from the fitting procedure (Figures 3D, 3E, and 3F). These three dominant scalp topographies (identified in the group-averaged data) were fitted to the ERPs of each individual participant during the time interval corresponding to the ERN/Ne and CRN to quantitatively determine their representation across participants and conditions. Finally, we submitted these GEV values to a 3 (map) × 2 (anxiety) × 2 (accuracy) mixed model

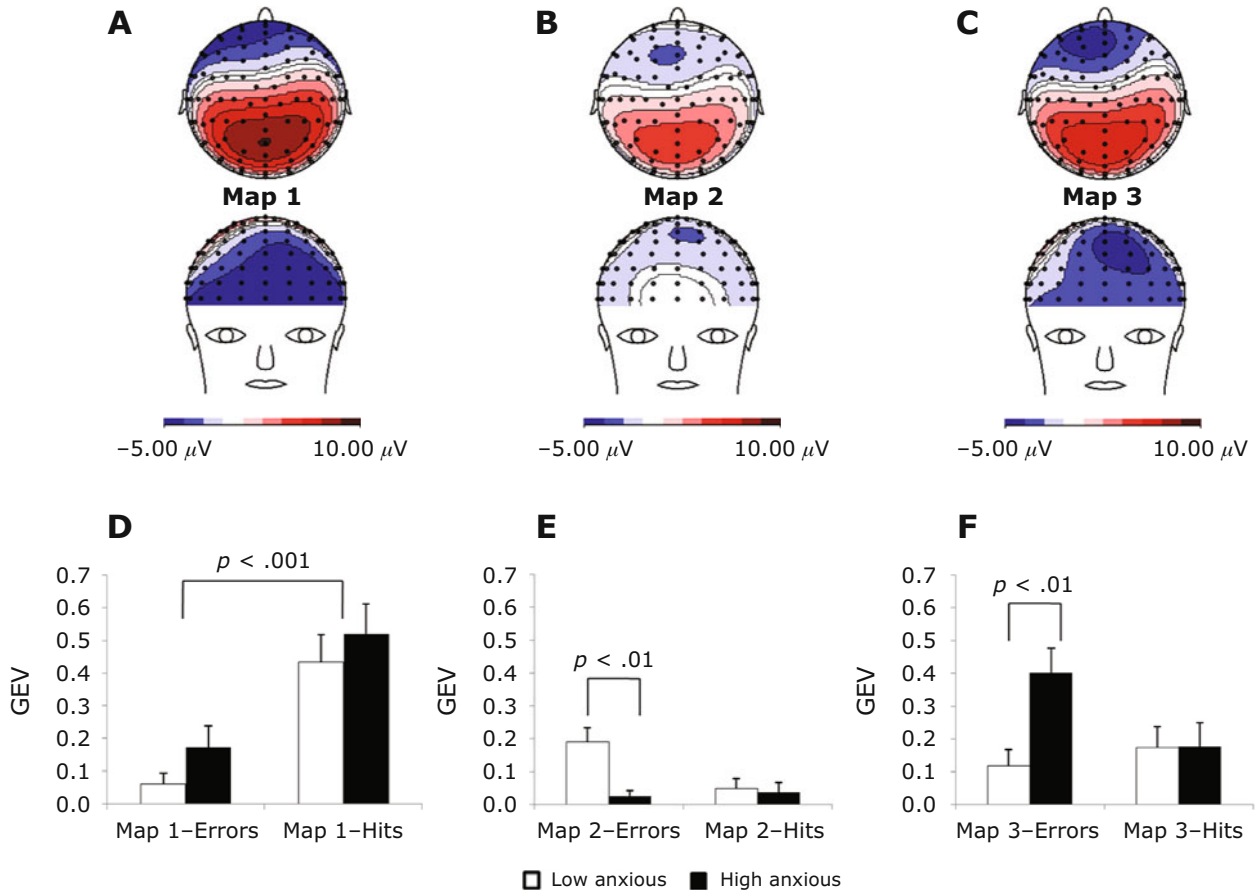

Figure 3. Dominant topographic maps (horizontal and frontal views) during the time interval corresponding to either the ERN/Ne or the CRN (20–50 msec post-response-onset). (A) The scalp map of the CRN (Map 1) was shared between the two groups. (B) The scalp map of the ERN/Ne for low-anxious participants (Map 2), showing a circumscribed negative activity around the FCz electrode position. (C) The scalp map of the ERN/Ne for high-anxious participants (Map 3), showing a broader negative activity over frontal and prefrontal electrodes, as compared with low-anxious participants. This ERN scalp map had a reliable different spatial configuration (regardless of changes in amplitude; see the Method section), relative to low-anxious participants. (D) The CRN scalp map (Map 1) showed a significant main effect of condition (explaining more variance for hits than for errors, regardless of the experimental group). (E) The ERN/Ne scalp map for low-anxious participants (Map 2) was found to be specific for errors in this group [significant interaction between group and condition, $F(1,30) = 6.70, p < .05$]. (F) Likewise, the ERN/Ne scalp map for high-anxious participants (Map 3) was found to be specific for errors in this group [significant interaction between group and condition, $F(1,30) = 5.18, p < .05$]. Error bars represent ±1 standard error of the mean. GEV, global explained variance.

ANOVA. This analysis revealed a significant three-way interaction [$F(2,60) = 3.05, p = .05$]. An additional 2 (anxiety) × 2 (accuracy) ANOVA run for each map separately confirmed that Map 1 (Figure 3D) was specific to correct hits but was shared across the two groups, as revealed by a significant main effect of accuracy [$F(1,30) = 40.85$, $p < .001$] but no interaction with anxiety ($F < 1$), whereas Maps 2 and 3 were specific to errors (Figures 3E and 3F), although with a clear-cut dissociation between the two groups for these two error-related scalp topographies. This first result is in line with a previous topographic-mapping study showing that the ERN/Ne and CRN led to different scalp distributions (and not only to a change in the electric field strength; see Vocat et al., 2008). More important, for both Map 2 and Map 3, the ANOVA disclosed a significant interaction between accuracy and anxiety [$F(1,30) = 6.70$, $p < .05$, and $F(1,30) = 5.18$, $p < .05$, for Map 2 and Map 3, respectively]. For low-anxious participants (Figure 3E), post hoc paired t tests showed that Map 2 had a larger GEV for errors, relative to hits [$t(15) = 2.88, p = .01$], whereas such an effect was not observed with Map 3 in this group [$t(15) = -0.59, p > .10$]. Symmetrically, for high-anxious participants (Figure 3F), Map 3 had a larger GEV for errors, relative to hits [$t(15) = 2.89, p = .01$], whereas such an effect was absent with Map 2 in this group [$t(15) = -0.35, p > .10$].

These topographic-mapping results therefore suggested a clear dissociation in the configuration of the electric field associated with errors (ERN/Ne scalp map) between low- and high-anxious participants. Note that this difference concerned the topography (electric field distribution), but not the amplitude or strength of the ERP signal (see the Method section). Because changes in the distribution of the electric field over the scalp surface (topography) necessarily denote alterations in the underlying configuration of intracranial generators (Lehmann & Skrandies, 1980;

Michel et al., 2001), these results indicated that high-anxious individuals may recruit a different network of brain regions early on following the occurrence of errors, as compared with low-anxious participants. This assumption was next verified, using a distributed source localization technique (sLORETA).

Source Localization Results

To gain insight into the putative configuration of the intracranial generators of these different topographic maps during the time interval corresponding to the ERN/Ne and CRN, we performed a source localization analysis, using sLORETA (Pascual-Marqui, 2002). For Map 1 (corresponding to the CRN scalp map, which was clearly shared across the two groups and specific to correct hits), sLORETA disclosed a main generator/cluster within the posterior parietal cortex, extending ventrally toward the posterior cingulate gyrus (Figure 4). A maximum activation was found in the precuneus (Brodmann Area 7, with an extended activation toward Brodmann Area 31) for this CRN scalp map (MNI coordinates: $-10x$, $-80y$, $+50z$). More important, sLORETA confirmed that the configurations of the intracranial generators underlying the ERN/Ne scalp map (errors) were roughly similar between low- and high-anxious participants and primarily involved medial frontal/dorsal ACC regions (Dehaene et al., 1994; Herrmann et al., 2004; O'Connell et al., 2007; Vocat et al., 2008), although with some substantial differences in the exact localization of these intracerebral generators within the dorsal ACC, as suggested by the topographic-mapping analyses. Whereas Vidal et al. (2000) found that the CRN and the ERN/Ne had the same neural generators (i.e., the dorsal ACC), here we found, in contrast, that the neural generators of the CRN were different from those of the ERN/Ne and that they primarily involved more posterior cingulate regions, whereas the ERN/Ne was associated with neural activity originating from the dorsal ACC (Dehaene et al., 1994; Herrmann et al., 2004; O'Connell

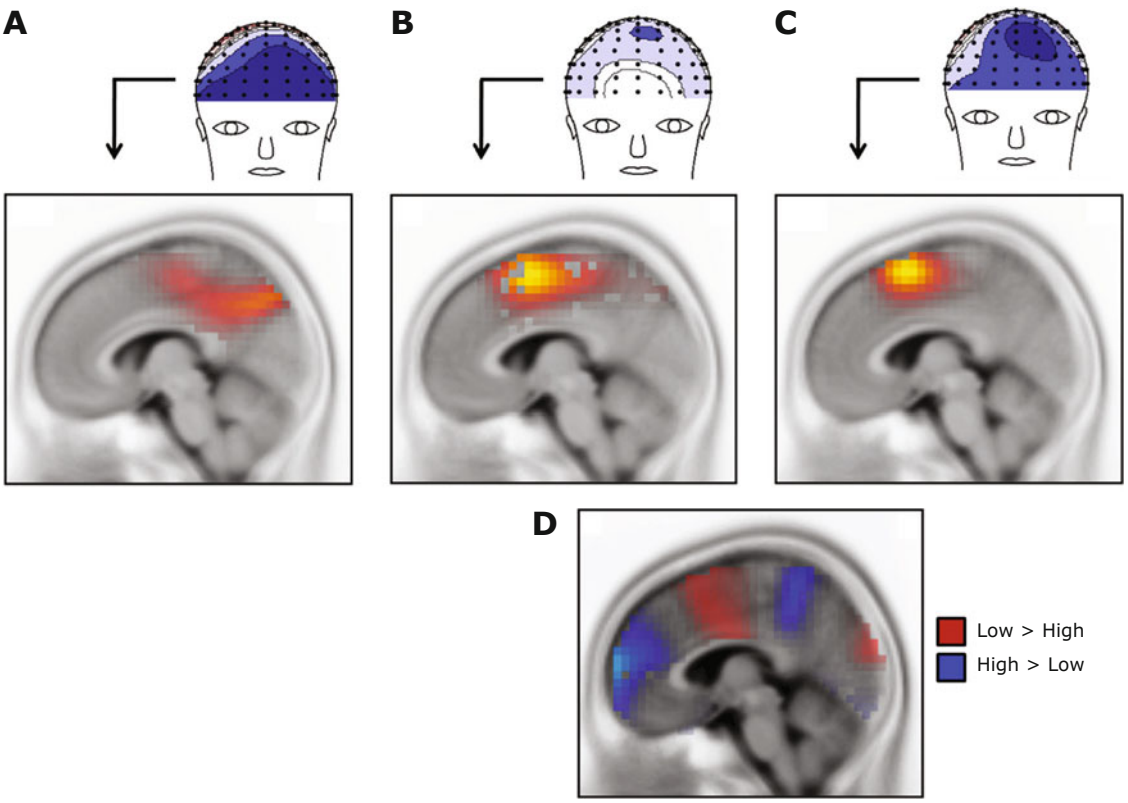

Figure 4. Source localization results, based on sLORETA. (A) Inverse solution for the CRN scalp map (Map 1), revealing a main cluster within the posterior parietal cortex, extending ventrally toward the posterior cingulate cortex (Brodmann Area 31). (B) Inverse solution for the ERN/Ne scalp map for low-anxious participants (Map 2), revealing a main cluster in the dorsal ACC (Brodmann Area 32) and medial frontal cortex (superior frontal gyrus, Brodmann Area 6). (C) Inverse solution for the ERN/Ne scalp map for high-anxious participants (Map 3), revealing a main cluster in a more anterior part of the dorsal ACC (Brodmann Area 24) and medial frontal cortex (superior frontal gyrus, Brodmann Area 6), as compared with low-anxious participants. A slight shift along the anterior–posterior axis in the location of the activation was evidenced between the two groups, with a more anterior dorsal ACC activation for high-anxious (C), relative to low-anxious (B) participants. (D) A direct comparison between the two groups confirmed different configurations of the intracranial generators for the ERN. The main generators of the ERN primarily involved the dorsal ACC for low-anxious participants (Brodmann Area 24), whereas they were localized in a more anterior region for high-anxious participants, corresponding to the most anterior part of the ACC (rostral ACC) and medial frontal gyrus (Brodmann Areas 32 and 10).

et al., 2007; Vocat et al., 2008). Therefore, our results support the hypothesis that errors do not simply amplify the activity of a generic action-monitoring system that would be equally engaged by correct and incorrect actions (Vidal et al., 2000) but, rather, rely on a specialized brain system localized within the dorsal ACC, with a significant modulation in the latter brain network as a function of levels of trait anxiety.

For low-anxious participants (Figure 4B), the neural generators of the ERN/Ne were localized mainly within the superior frontal gyrus/dorsal ACC (maximum: 5*x*, 10*y*, 60*z*; Brodmann Areas 6 and 32), whereas for high-anxious participants (Figure 4C), they also involved the superior frontal gyrus/dorsal ACC (maximum: −5*x*, 5*y*, 60*z*; Brodmann Areas 6 and 24), but with a shift toward the front, as compared with low-anxious participants (Figure 4B). Importantly, a direct comparison between the two groups confirmed a different configuration of intracranial generators for the ERN (Figure 4D). Whereas the main generators of the ERN primarily involved the dorsal ACC for low-anxious participants (Brodmann Area 24), they were localized in a more anterior region for high-anxious participants, corresponding to the most anterior part of the ACC and medial frontal gyrus (Brodmann Areas 32 and 10). In addition, another generator was found within the posterior cingulate gyrus/paracentral lobule for high-anxious participants (Brodmann Areas 31 and 5).

DISCUSSION

The goal of this study was to investigate the links between error-monitoring functions and individual differences in trait anxiety, primarily using ERP measurements. More specifically, we tested the prediction not only that the ERN/Ne, an early electrophysiological marker of error detection (Falkenstein et al., 2000) would be relatively enhanced in high- as compared with low-anxious individuals (Olvet & Hajcak, 2008) (quantitative account), but also that trait anxiety could alter the expression of this ERP component (qualitative account), consistent with the assumption of abnormal or altered early error-related brain reactions in anxious individuals. A number of important new results emerge from this study.

First, we found comparable behavioral performance (and efficiency) for the two groups during the speeded and demanding go/no-go task. High-anxious participants did not commit more errors (nor were they slower or faster with errors) than did low-anxious participants. This rules out the possibility that ERP differences observed between these two groups actually resulted from different behavioral effects during this go/no-go task. In addition, the two groups showed comparable classical posterror slowing effects (Laming, 1979; Rabbitt, 1966), suggesting preserved error monitoring and adaptation effects in these two groups. These results corroborate previous findings showing that behavioral measures of cognitive control abilities do not differ between low- and high-anxious participants (Gehring, Himle, & Nisenson, 2000; Hajcak et al., 2003; Hajcak & Simons, 2002; Luu, Collins, & Tucker, 2000). However, we found that the speeded go/no-go task had a differential influence on subjective levels of state anxiety in low- and high-anxious participants. Only high-anxious participants showed increased levels of state anxiety following the task (relative to a baseline state anxiety measure obtained before the task), as compared with low-anxious participants. This result suggests that the speeded go/no-go task had a differential influence on the experience of negative affect in high- versus low-anxious participants.

Importantly, ERP results confirmed a dissociation between the two groups. High-anxious participants showed a larger difference between the ERN/Ne and the CRN, as compared with low-anxious participants, suggesting an increased sensitivity to errors in the former group. It is noteworthy that complementary topographic analyses actually indicated that the ERN/Ne scalp map underwent a reliable configuration change for high-anxious, relative to low-anxious, participants, although the CRN scalp map was shared across these two groups, suggesting that errors, but not correct hits, were differentially processed in these two groups. Clearly, the ERN/Ne scalp map had a different configuration for high-anxious, relative to low-anxious, individuals and concerned more anterior and (pre)frontal electrodes. This result suggests that these two groups used partly nonoverlapping brain networks early on following the onset of an incorrect response during performance monitoring. This conjecture was formally verified by the subsequent source localization analysis, which disclosed a shift of neural generators within the ACC for high-anxious, relative to low-anxious, participants during the time interval corresponding to the ERN/Ne. We discuss the implications of these new results in greater detail below.

Augmented ERN/Ne to Errors in High-Anxious Participants

The results of the conventional peak analysis were in line with previous ERP findings, which showed links between trait anxiety and the magnitude of the ERN/Ne. Earlier ERP studies already reported that the ERN/Ne to errors was increased during speeded RT tasks in participants with anxiety characteristics (Boksem et al., 2006; Endrass, Klawohn, Schuster, & Kathmann, 2008; Gehring et al., 2000; Hajcak et al., 2003, 2004; Hajcak & Simons, 2002; Luu, Collins, & Tucker, 2000). Although some previous studies also showed an effect of trait anxiety on ERN/Ne amplitudes for both errors and hits (Hajcak et al., 2003), here we found an interaction effect between accuracy (error vs. hit) and anxiety (low vs. high), precluding the possibility that trait anxiety affected equally the early processing of errors and hits during the speeded go/no-go task. The results for the ERN/Ne (peak analysis) showed that the amplitude difference between errors and hits was larger in high- than in low-anxious participants, suggesting a higher sensitivity to errors in high-anxious participants, despite similar behavioral performances in these two groups. These new results are therefore consistent with the motivational significance theory of the ERN/Ne (Hajcak & Foti, 2008; Hajcak et al., 2003; Luu, Collins, & Tucker, 2000), which predicts that this specific

error-related ERP component indexes mainly the motivational significance of errors. Hence, participants, such as high-anxious individuals, who are more sensitive to negative events and punishment should also react more strongly to errors and, as a corollary, present a (relatively) larger ERN/Ne to errors. Our new results for the ERN/Ne component support this assumption. By contrast, the amplitude of the Pe component was comparable between the two groups, suggesting that individual differences in anxiety did not modulate more elaborate stages of error monitoring, presumably reflected by this later ERP component (Ridderinkhof et al., 2009). This dissociation confirms that, unlike the ERN, the Pe is not directly associated with the affective processing of errors, corroborating previous ERP findings showing that these two early ERP components may reflect different processes during performance monitoring (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005).

Alteration of Early Error Detection Brain Mechanisms in High-Anxious Participants

Although our new ERP results are, overall, compatible with the motivational significance theory of the ERN/Ne (Hajcak & Foti, 2008; Hajcak et al., 2003; Luu, Collins, & Tucker, 2000), they also provide important new information, since they show a dissociation in the expression of the ERN/Ne between the two groups. This global difference concerning the distribution of the electric field (rather than its strength) could not be captured using a conventional peak analysis (Picton et al., 2000). Hence, not only was the ERN/Ne − CRN amplitude difference larger in high-anxious participants than in low-anxious participants, but also the scalp distribution of the ERN was altered in the former, as compared with the latter, group. Whereas the CRN scalp map was shared across the two groups (and mainly involved posterior parietal regions—Brodmann Area 7, with an extended activation toward Brodmann Area 31), the ERN/Ne scalp map had different configurations in high- versus low-anxious participants. The frontocentral negative activity associated with errors (ERN/Ne) showed a broader and more extended (pre)frontal distribution for high-anxious participants, relative to low-anxious participants, where this early negative activity was clearly circumscribed to a few electrode positions, including FCz.

For each group, we found that the ERN/Ne scalp map could be reliably modeled by a solution with distributed generators within the dorsal ACC, consistent with many previous ERP studies that primarily ascribed the ERN/Ne to the activity of either the premotor/supplemental motor area or the dorsal ACC, or sometimes both (Dehaene et al., 1994; Herrmann et al., 2004; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner, Braun, & Coles, 1997; O'Connell et al., 2007). However, we found that the differential scalp maps for the ERN/Ne between the two groups could be explained by a slight shift within the dorsal ACC for the exact location of the intracranial generators. For low-anxious participants, the ERN/Ne was generated primarily in the premotor/supplemental motor area and in the dorsal ACC (Brodmann Areas 6 and 32), whereas for high-anxious participants, the maximum within the dorsal ACC shifted toward the front and involved more frontal and dorsal parts of the medial frontal cortex (Brodmann Areas 6 and 24). Furthermore, a direct comparison between groups (Figure 4D) revealed that high-anxious participants recruited more anterior medial frontal regions, as well as posterior cingulate regions, during the time interval of the ERN, relative to low-anxious participants. It is noteworthy that this contrast disclosed that the rostral ACC, as well as more posterior regions (including the posterior cingulate cortex), was more activated in high-anxious participants, as opposed to more dorsal ACC regions in low-anxious participants (Figure 4D). This finding suggests that anxiety alters the configuration of the neural network activated during early error monitoring. The involvement of the rostral ACC may indicate that not only cognitive but also emotional monitoring effects were temporarily active in high-anxious participants during the early detection of response errors (see Bush et al., 2000; Devinsky, Morrell, & Vogt, 1995). Moreover, the additional involvement of posterior cingulate regions may suggest that high-anxious individuals not only monitor, but also likely appraise (or alternatively, readily orient to) their motor plans at a very early stage following the onset of the response (Badgaiyan & Posner, 1998; Menon, Adleman, White, Glover, & Reiss, 2001; Vogt, Finch, & Olson, 1992).

Interestingly, this substantial alteration of the electric field configuration underlying the ERN/Ne as a function of trait anxiety could be explained by the attentional control theory (Eysenck & Calvo, 1992; Eysenck et al., 2007). This model predicts that with similar task demands, high-anxious participants recruit more cognitive resources (i.e., they are less efficient) than do low-anxious participants to reach the same level of performance. Our behavioral results are consistent with this theory, since trait anxiety did not influence performance (see also Compton et al., 2007; Hajcak et al., 2003). To compensate for this reduced efficiency, the use of more cognitive (or emotional) resources in the high-anxious group could translate as a different recruitment of cognitive control areas in anxiety (for converging evidence, see Braver et al., 2007; Fales et al., 2008). Our observation of a qualitative difference in the neurophysiological expression and intracranial generators of the ERN with trait anxiety therefore corroborates this view. This effect might reflect the activation of distinct cognitive control processes in high-anxious participants, a self-generated compensatory strategy used by these participants to deal with the immediate need of behavioral adjustments imposed by the early detection of unforced errors during this go/no-go task (Eysenck & Calvo, 1992).

More specifically, we suggest that trait anxiety alters early error detection mechanisms (an important component of cognitive control) within the dorsal division of the ACC (Brodmann Areas 24 and 32). Previous studies already demonstrated that different areas in the rostral division of the ACC contribute differentially to action monitoring and cognitive control. For example, whereas the anterior part of the rostral ACC was assumed to exhibit conflict-specific effects, the posterior part of the rostral ACC was found to be less sensitive to conflict and showed

more general action-monitoring effects (Milham & Banich, 2005). Moreover, different subdivisions of the ACC may serve different functions, with a shift between emotional and cognitive operations during behavioral control along an anterior–posterior axis (Bush et al., 2000). Hence, our results suggest that high-anxious participants may call extra emotional control regions within the rostral ACC during the early detection of errors, relative to low-anxious participants, who showed a more typical dorsal ACC contribution during this process (see Bush et al., 2000; Dehaene et al., 1994). The observed shift of the neural generators for the ERN/Ne within the dorsal ACC as a function of trait anxiety suggests that different cognitive control areas may also exist within the dorsal ACC. Moreover, low- and high-anxious participants seem to differentially recruit these areas, indicating that errors may acquire a different cognitive or motivational significance in high-anxious, as opposed to low-anxious, participants. Thus, high-anxious participants not only respond (relatively) more strongly to self-generated errors, but also react in a different way, relative to nonanxious participants. Because we tested mainly female anxious participants, some caution is, however, needed before we can generalize these results to a population of male adult participants.

To conclude, the results of this study show that trait anxiety can lead to qualitative (and not only quantitative) changes during the earliest stage of error monitoring. As such, these findings are consistent with the attentional control theory (Eysenck et al., 2007), and they may help us to better understand the effects of trait anxiety on cognitive control brain mechanisms. Future ERP studies should further investigate what may be the influence of these qualitative changes during early error monitoring on the regulatory component of this process, which presumably takes place later after error commission and involves other brain structures, including the dorsolateral prefrontal cortex. Because response errors typically lead to subsequent adaptation effects (i.e., posterror slowing), it might be useful in future work to look at the electrophysiological correlates of these adaptation effects (e.g., CRN generated for the correct response that immediately follows an error) and verify how they might relate to previous early error detection brain mechanisms.

AUTHOR NOTE

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