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The role of cingulate cortex in the detection of errors with and without awareness: a high-density electrical mapping study

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Abstract

Error-processing research has demonstrated that the brain uses a specialized neural network to detect errors during task performance but the brain regions necessary for conscious awareness of an error are poorly understood. In the present study we show that two well-known error-related event-related potential (ERP) components, the error-related negativity (ERN) and error positivity (Pe) have a differential relationship with awareness during performance of a manual response inhibition task optimized to examine error awareness. While the ERN was unaffected by the participants' conscious experience of errors, the Pe was only seen when participants were aware of committing an error. Source localization of these components indicated that the ERN was generated by a caudal region of the anterior cingulate cortex (ACC) while the Pe was associated with contributions from a more anterior ACC region and the posterior cingulate–precuneus. Tonic EEG measures of cortical arousal were correlated with individual rates of error awareness and showed a specific relationship with the amplitude of the Pe. The latter finding is consistent with evidence that the Pe represents a P3-like facilitation of information processing modulated by subcortical arousal systems. Our data suggest that the ACC might participate in both preconscious and conscious error detection and that cortical arousal provides a necessary setting condition for error awareness. These findings may be particularly important in the context of clinical studies in which a proper understanding of self-monitoring deficits requires an explicit measurement of error awareness.

Introduction

Since the initial behavioural work of Rabbitt (1966), understanding how the brain detects and processes performance failures has been a major challenge for cognitive neuroscience. A clear message emerging from this literature is that error processing represents a discrete component of a broader action-monitoring system and is reliant on a specialized brain network. Functional imaging and event-related potential (ERP) studies have consistently pointed to the anterior cingulate cortex (ACC) as a critical brain region responsible for the initial detection of unfavourable performance outcomes (Ridderinkhof *et al*., 2004; Kennerley *et al*., 2006). Importantly, however, investigators have not typically made the distinction between error detection and conscious error awareness. As a result, it is often not clear how the error-related brain activations that have been demonstrated actually relate to the conscious experience of making an error. The present study aims to identify some of the key electrophysiological markers for error awareness.

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The first of only two ERP studies to explicitly measure error awareness was conducted by Nieuwenhuis and colleagues (Nieuwenhuis *et al*., 2001) who focused on two components commonly seen in response-locked ERP waves that have been time-locked to erroneous responses; the error-related negativity (ERN) and the error positivity (Pe). The early onset of the ERN, before the erroneous response has been completed, is suggestive of a rapid internal detection mechanism that is not dependent on conscious processing of the error (Falkenstein *et al*., 2000). Rather than directly detecting errors, the ERN may reflect monitoring processes that are sensitive to response conflict (Van Veen & Carter, 2002) or changes in reward probability (Holroyd *et al*., 2004). Several studies have shown that ERN amplitude predicts short-term posterror compensatory adjustments (Gehring *et al*., 1993; Rodriguez-Fornells *et al*., 2002; Debener *et al*., 2006). In contrast, the functional significance of the Pe, maximal over parietocentral scalp sites, is poorly understood but this component peaks sufficiently late (300–500 ms posterror) for sensory or proprioceptive information to be available and is therefore more likely to index conscious aspects of error processing. Indeed, Nieuwenhuis *et al*. (2001), who asked participants to perform an antisaccade task, found that consciously perceived errors elicited far larger Pe

amplitudes than unperceived errors while the ERN remained unaffected. This finding, which was replicated more recently by Endrass *et al.* (2005), provided the first evidence that the Pe, and not the ERN, specifically reflects conscious aspects of error processing. However, the generalisability of this relationship beyond the occulomotor modality has yet to be verified.

Source localization studies of the ERN and Pe have confirmed the critical role of the ACC in error detection by identifying generators in this region for both the ERN and the Pe. The ERN has been consistently localized to caudal regions of the ACC thought to govern performance monitoring processes such as conflict detection and interference control (Dehaene *et al.*, 1994; Van Veen & Carter, 2002; Hermann *et al.*, 2004; Van Boxtel *et al.*, 2005). Attempts to identify the precise ACC generator for the Pe have been less consistent. While Van Veen & Carter (2002) and Van Boxtel *et al.* (2005) located the source of the Pe in a rostral portion of the ACC associated with motivation and affective processing (Bush *et al.*, 2000; Taylor *et al.*, 2006), the source identified by Hermann *et al.* (2004) was located in a far more caudal region. Interestingly, in the only fMRI study to explicitly distinguish between consciously perceived and unperceived errors (Hester *et al.*, 2005) there was no additional ACC activity when participants were aware of their errors. This finding appears to be at odds with data suggesting an ACC source for the Pe. Thus the neural processes necessary for conscious error awareness have not yet been clearly identified.

The present study was designed to shed further light on the neural substrates of error awareness by addressing three key questions. First, given that error-related brain activity has primarily been investigated while participants performed manual response tasks the present study aimed to verify the generalisability the findings of Nieuwenhuis *et al.* (2001) beyond the oculomotor modality using the same manual response task developed by Hester *et al.* (2005). A recent study has indicated that consciously detected errors may also be distinguished by the presence of a strong autonomic reaction (O'Keeffe *et al.*, 2004). Here, measures of autonomic system activity are acquired to further validate the sensitivity of this task to error awareness. Second, in order to further explore the role of the ACC in error awareness the present study sought to localize the neural generators of the ERN and Pe while distinguishing between errors made with and without conscious awareness. Finally, a limitation of purely event-related approaches to error processing is that we can learn little about potentially critical brain states that could provide the setting conditions for conscious awareness. Research has previously demonstrated a clear association between alertness deficits and failures in conscious error detection in brain-injured populations (McAvinue *et al.*, 2005). In addition, several authors have suggested that the Pe may be part of the same evaluative process as the stimulus-locked P3 (Nieuwenhuis *et al.*, 2001; Overbeek *et al.*, 2005), a component which is reliably modulated by arousal (Nieuwenhuis *et al.*, 2005). A hypothesis that emerges from this work is that one's basal level of cortical arousal, as measured by the ratio of slow (alpha and theta) to fast (beta) wave oscillations across the task duration, will be associated with the extent of conscious error processing. Therefore the third aim of this study was to investigate the relationship between tonic electroencephalogram (EEG) measures of cortical arousal and error awareness.

Materials and methods

Subjects

Nineteen (one female, one left-handed) neurologically normal volunteers participated. Participants received a gratuity of €32 to cover expenses incurred on the day of testing. Subjects were aged between 18 and 30 years (mean ± SD age 22.07 ± 2.85 years). All participants gave written informed consent and all procedures were approved by the ethical review boards of St Vincents Hospital, Fairview, and Trinity College Dublin. All subjects reported normal or corrected-to-normal vision. Ethical guidelines were in accordance with the Declaration of Helsinki.

Error awareness task (EAT) and procedure

We used an error awareness paradigm developed by Hester *et al.* (2005). The EAT (see Fig. 1) is a motor Go/No-go response inhibition task in which participants are presented with a serial stream of single colour words with congruency between the word and its font colour manipulated. Subjects were trained to respond to each of the words with a single 'Go trial' button press and to withhold this response when either of two different circumstances arose. The first circumstance was if the same word was presented on two consecutive trials (Repeat No-go), and the second was if the word and its font colour did not match (Incongruent No-go). In the event of a commission error

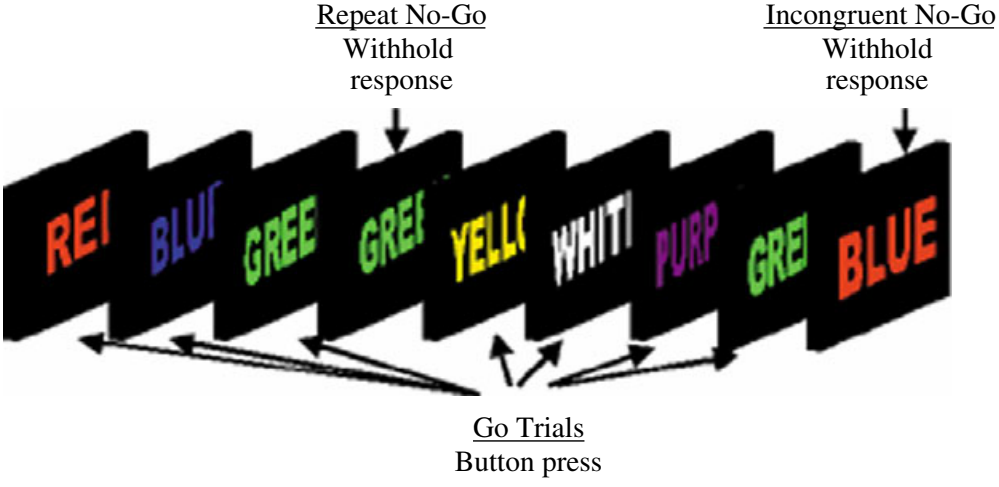

FIG. 1. The error awareness task required subjects to respond with a button press to a stream of colour words and withhold their response when either a word was repeated on consecutive trials or the font and word were incongruous. Subjects were trained to press a different button following any commission errors.

(failure to withhold to either of these No-go scenarios) subjects were trained to press a second 'awareness button' on the next Go trial after the error and were not required to make the standard Go response. Participants were instructed to time their button presses to the offset (i.e. end) of each stimulus. This kind of 'response-locking' has been shown to reduce interindividual variability and eliminate speed–accuracy tradeoffs (Stuss *et al*., 2003). In the present experiment response-locking allowed us to rule out the possibility that certain undetected errors could be attributed to an overemphasis on speed over accuracy.

To maximize the number of errors for ERP averaging participants completed an average of 11.2 blocks of the EAT (range 8–14). Each EAT block consisted of 225 stimuli of which 200 were Go stimuli and 25 were No-go stimuli (of which 13 were Repeat No-gos and 12 were Incongruent No-gos or vice versa). All stimuli were presented for 600 ms followed by an interstimulus interval of 900 ms and appeared 0.25° above a white fixation cross on a grey background at a distance of ∼150 cm. Participants were instructed to focus on the fixation cross during the task in order to minimize eye movements.

Electrodermal activity (EDA) acquisition and analysis

EDA is recorded as changes in electrical conductance due to sweat gland activity and is controlled by sympathetic innervation of the autonomic nervous system. EDA has been commonly used as an index of the psychological processing of stimulus properties such as significance, novelty, emotional relevance and effortful processing. Previous work has demonstrated that EDA responses are absent when participants are not aware that they have made an error (O'Keeffe *et al*., 2004). In the present study we acquire EDA as a further means of verifying that errors after which there was no 'awareness' response were not associated with significant amounts of conscious processing.

EDA measurements were taken from all participants during EAT testing with a five-channel BIOPAC MP30B unit, calibrated to skin conductance responses (SCRs) in microsiemens (μS). Two Ag/AgCl BIOPAC electrodes, with contact areas of ∼6 mm, were filled with SIGNA electrode gel and secured with a velcro strap to the volar surface of the distal phalanges of the index and middle fingers of the participant's nondominant hand. After a 5-min rest period to ensure skin hydration by the gel, the BIOPAC software was calibrated to the participants' own electrodermal parameters before EAT testing began.

EDA data were analysed using Matlab 6.1 according to previously established criteria (Dawson *et al*., 2000). A rise in skin conductance level (SCL) was considered to be a response (SCR) if its onset was between 0.5 and 4.5 s after a particular event (presentation of No-go stimulus or alert). SCRs were measured by subtracting the SCL at stimulus onset from the peak SCL within the latency period. The criterion for the smallest acceptable SCR was set at 0.02 μS. Any trials on which the SCL exceeded a response threshold of 0.02 μS in the first 0.5 s after the event were thus rejected. The SCR for a given accepted trial was measured by taking the maximum within the interval 0.5–4.5 s and subtracting the nearest preceding local minimum within that interval. For the plotting of EDA waveforms, the SCL time courses were simply averaged across all trials for each condition (see Fig. 2).

EEG data acquisition and statistical analysis

Continuous EEG was acquired through the ActiveTwo Biosemi™ electrode system from 72 scalp electrodes, digitized at 512 Hz. Vertical eye movements were recorded with two vertical (V) electrooculogram (EOG) electrodes placed below the left and right eye, while HEOG electrodes at the outer canthus of each eye recorded horizontal movements.

Data were analysed using BESA Version 5.1 (Brain Electric Source Analysis) software (http://www.besa.de). For analysis and display purposes, data were average-referenced and filtered with a low-pass 0-phase shift 48 dB 30 Hz filter after acquisition. Response-locked data were segmented into epochs of 400 ms before to 500 ms after button press and were baseline-corrected relative to the interval −400 to −200 ms. All electrode channels were subjected to an artifact criterion of ±100 μV from −400 to +500 ms to reject trials with excessive electromyogram or other noise transients. The single-trial EEG signals were also corrected for horizontal and vertical EOG artefacts by means of an eye movement correction procedure developed by Berg & Scherg (1994). Accepted trials were then averaged separately for correct Go presses and commission errors (Repeat and Incongruent No-gos) after which the participants indicated awareness (Aware Error) and commission errors after which the participants did not indicate awareness (Unaware Error). Unaware Errors were rejected if the participant failed to make any response on the next Go trial or if they pressed the 'awareness' button within three trials of the No-go error.

Inspection of the grand-average waveforms revealed a clearly defined ERN following both commission error types peaking at ∼80 ms after the button press. A smaller ERN-like component was also evident at the same latency after correct Go presses. For all three conditions the maximal peak amplitude of the ERN was seen at FCz with little topographical variation across participants. The ERN was therefore defined as the most negative peak at FCz occurring in a window from 50 to 120 ms postresponse. In all three response conditions (correct Go, aware error and unaware error) the ERN was immediately followed by a positive deflection with maximal amplitude also at FCz and a peak at ∼190 ms postresponse. On aware errors this early positive peak was followed by the classic Pe component in the form of a large positive wave over posterior scalp regions and maximal amplitude at CPz. The early positive component was calculated for all three response conditions as the most positive peak at FCz between 140 and 240 ms postresponse. Because the Pe is a more sustained low-frequency component the mean amplitude at CPz between 300 and 500 ms postresponse was used.

Finally, the average power spectrum over the first eight blocks of EAT testing was calculated for each participant using the discrete Fourier transform (eight blocks was the minimum number of EAT blocks that were completed by all participants). Each participant's tonic theta, alpha and beta powers (μV^2) were calculated as the power in the 4–7, 8–12 and 13–29 Hz ranges, respectively. Theta/beta and alpha/beta ratios were subsequently calculated.

The behavioural and EEG measures reported were calculated over the first eight blocks for all participants. Due to the low number of unaware error trials, ERP averages were calculated for the full number of blocks completed by each participant (between 8 and 14) to maximize the number of single trials included in each average. Only those participants who made at least 20 aware and 20 unaware errors were included in the ERP analysis. This led to a reduced sample of 12 participants who made an average of 76 aware errors and 35 unaware errors. Because of the relatively low data yield for unaware errors it was important to equate the number of single trials that contributed to the averages for these comparisons. Consequently half the aware errors were removed by selecting only even matches to ensure that they were not

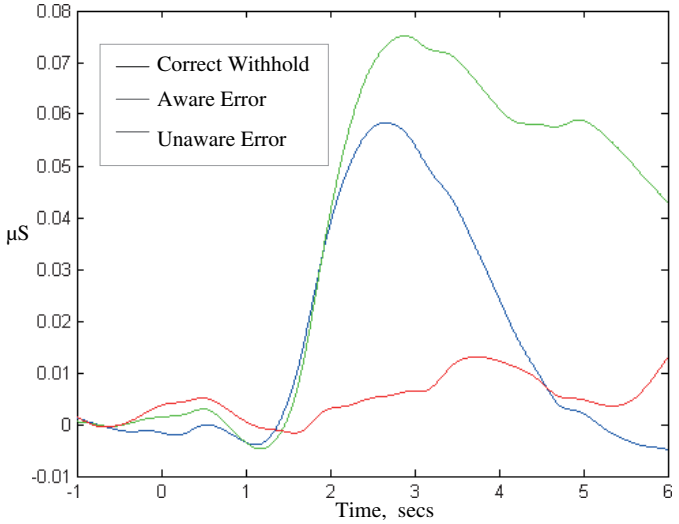

FIG. 2. Skin conductance responses (SCR) averaged separately for aware errors, unaware errors and correct withholds on No-go trials and time-locked to stimulus end (time-point 0). Here we see that, as expected, unaware errors did not elicit the autonomic response typically seen following significant events such as errors, confirming that conscious processes were not active.

overrepresented either quantitatively or temporally in these comparisons.

Mean values are quoted ± SD.

Results

Behavioural performance

Participants successfully withheld their response on 69.3% of No-go targets. A significantly greater proportion of total commission errors occurred on Incongruent No-gos (61%) than on Repeat No-gos (39%; $t_{18} = 5.69$, $P < 0.001$). Participants were aware of 75.8% of all commission errors (aware errors/total errors). Although more errors were made on Incongruent No-go targets, participants were significantly less likely to be consciously aware of errors on Repeat No-gos (Repeat No-gos, 67.7%; Incongruent No-Gos, 82%; $t_{18} = 3.301$, $P < 0.01$). Mean reaction time (RT) to Go stimuli was 625 ± 96.87 ms, indicating that participants were successfully timing their responses to stimulus-end. Participants had significantly longer RTs for unaware errors (mean RT unaware, 676.3 ± 112.8 ms) than for aware errors (mean RT aware, 593.4 ± 119.3 ms; $t_{18} = -4.0$, $P < 0.01$).

Modulation of EDA by awareness

As Fig. 2 illustrates, strong SCRs were elicited by aware errors (0.243 ± 0.19 μS) and correct withholds (0.203 ± 0.16 μS) on No-go Trials. In contrast, this response was absent following unaware errors (0.080 ± 0.083 μS). An ANOVA on the magnitude of SCRs for the three No-go conditions (aware error, unaware error and correct withhold) revealed a significant main effect of No-go condition ($F_{1,11} = 7.96$, $P < 0.01$). *Post hoc* Bonferonni tests indicated that

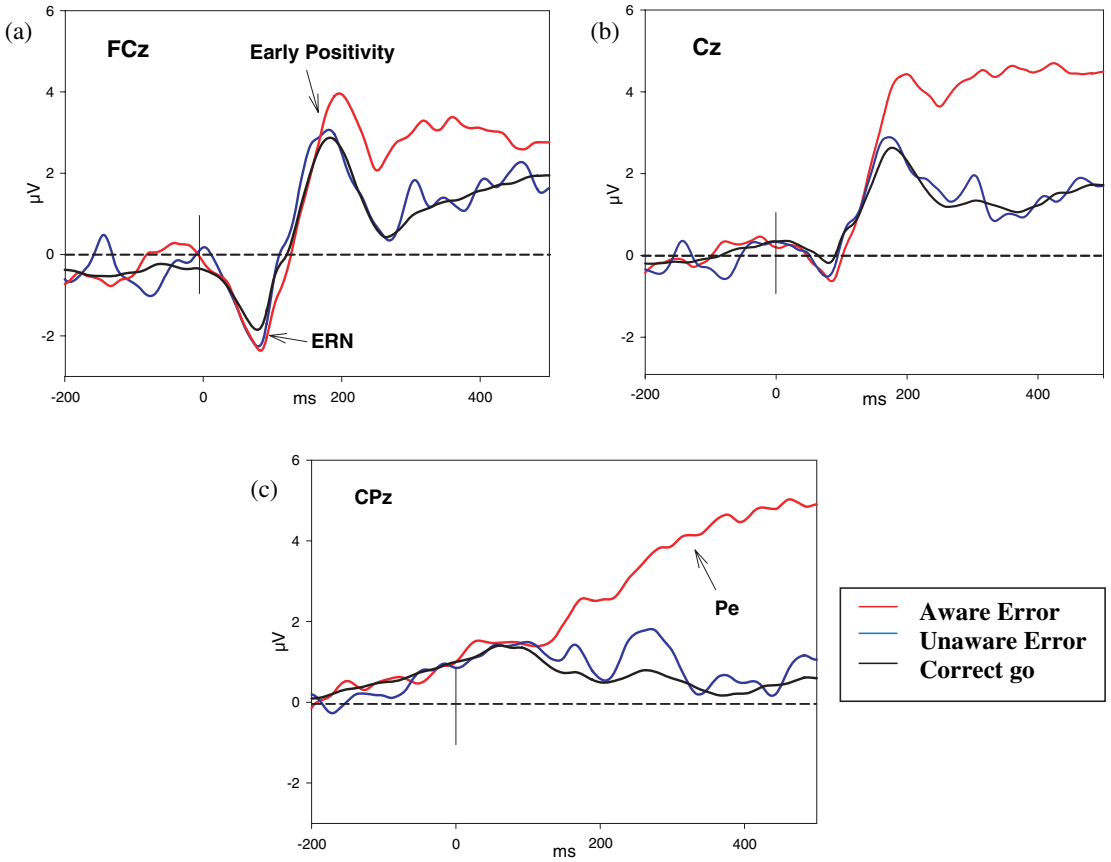

FIG. 3. Grand-average ERP waveforms at (a) FCz, (b) Cz and (c) CPz time-locked to button press (time-point 0). Panel (a) shows that the ERN and early positivity were not modulated by participants' awareness of errors. In panel (c) we note the large Pe wave following aware errors that is absent on unaware errors and correct-Go presses.

participants had similar SCRs following aware errors and correct withholds ($P = 0.17$) but SCRs following unaware errors were significantly smaller than those elicited by aware errors ($P < 0.01$) or correct withholds ($P < 0.05$). These data indicate that the cognitive–emotional processes indexed by EDA were absent on unaware errors.

Modulation of error-related ERPs by awareness

An ANOVA on the ERN amplitudes for the three Response conditions (see Fig. 3a) indicated a significant main effect of Response (mean amplitudes: aware error, -2.7 ± 1.5 μV; unaware error, -2.9 ± 1.4 μV; correct go, -1.9 ± 1.2 μV; $F_{2,22} = 4.6$, $P < 0.05$). Tests of within-subjects contrasts revealed that the ERN was significantly larger following both error types than following correct Go presses (Go vs. Aware $F_{1,11} = 6.37$, $P < 0.05$; Go vs. Unaware $F_{1,11} = 6.407$, $P < 0.05$) but importantly there was no difference between ERN amplitude for aware and unaware errors ($F_{1,11} = 0.015$, $P = 0.872$).

An ANOVA on the early positive peak immediately following the ERN revealed no reliable differences across conditions (mean amplitudes: aware error, 4.4 ± 2 μV; unaware error, 3.78 ± 2 μV; correct go, 3.0 ± 1.2 μV; $F_{2,22} = 2.88$, $P = 0.083$; see Fig. 3a).

Finally, an ANOVA on the Pe amplitudes revealed a significant main effect of Response (mean amplitudes: aware error, 4.9 ± 1.2 μV; unaware error, 0.5 ± 0.13 μV; correct Go, 0.14 ± 0.7 μV; $F_{2,22} = 68.73$, $P < 0.001$). Tests of within-subjects contrasts confirmed that this late positive wave was significantly smaller on correct Go presses and unaware errors than on aware errors (correct Go vs. aware $F_{1,11} = 112.87$, $P < 0.001$; unaware vs. aware $F_{1,11} = 72.94$, $P < 0.001$). Unaware error and correct Go ERPs did not differ in terms of this late positivity ($F_{1,11} = 0.025$, $P = 0.877$; see Fig. 3c).

Source localization of error-related ERPs

Strong ERN and early positive waves were evident in all three response conditions including correct Go presses, and their scalp topographies did not vary (see Fig. 4a). Due to these similarities, source localization of the ERN and early positivity was conducted on aware error ERPs instead of a difference waveform. Source localization was implemented by BESA 5.1 using a four-shell spherical head model approximation. First the ERN was selected by highlighting a 20-ms interval around its negative peak. A single dipole model located in the ACC accounted for most of the variance in the ERN ($x = -14.7$, $y = 0.1$, $z = 45$ mm; Residual Variance (R.V.) = 6.9%, Best (minimum variance within the fit interval) = 5.6%). When selecting a 20-ms interval around the peak of the early positivity the same ACC dipole accounted for most of the variance in this component (R.V. = 9.2%, Best = 7.9%; see Fig. 4b). Keeping the location of the ERN source constant, free-fit source localization was performed on the 20-ms interval around the peak of the error positivity. The resulting additional dipole did not explain a significant amount of variance in the early positivity (change in R.V. < 1%). Finally, the ERN source was removed and a broader interval extending from 20 ms prior to the peak of the ERN to 20 ms after the peak of the early positivity was selected. Again, the free-fit algorithm indicated a generator in a very similar caudal region of the ACC ($x = -16.3$, $y = -13.6$, $z = 50.6$ mm). Although the ERN and early positivity share similar frontocentral scalp distributions, the early positivity does appear to be distributed more centrally (see Fig. 4a). It is possible therefore that these two components are generated by areas of the ACC that are very close together and that the spatial resolution of source localization was insufficient to prise them apart.

Keeping the location and orientation of the ERN and early positivity dipole constant, source localization was then performed on the Pe between 300 and 500 ms after response. A two-source model was indicated for the Pe (R.V. 3.1%, Best 2.1%) with one dipole located in the ACC but more anterior to the ERN and early positivity dipole ($x = 2.9$, $y = 20.5$, $z = 42.5$) and the other located in the vicinity of the posterior cingulate cortex and precuneus ($x = -4.5$, $y = -37.6$, $z = 39.9$). Given the proximity of the two ACC dipoles a further test was conducted to verify the accuracy of this source model. The grand average three-dipole model and a simpler, two-dipole, model which did not include the anterior ACC dipole, were applied to individual subject ERPs. Removal of the anterior ACC dipole caused an increase of 8.9% in the average R.V. (three dipoles: mean R.V., 16.2%; range, 8.6–30.5; two dipoles: mean R.V., 25.1%, range, 10.4–57.5). An additional source analysis of the Pe was also conducted on a difference waveform subtracting the unaware-error waveform from the aware-error waveform, thus isolating activity specifically associated with conscious error perception. This analysis revealed a very similar two-source Pe model with one dipole located in the same ACC region ($x = 6.9$, $y = 13.1$, $z = 46.5$) and another located around the posterior cingulate cortex and precuneus, though in a slightly more inferior location than that indicated by the first solution ($x = -5$, $y = -59.1$, $z = 20.7$). The dipole models and source waveforms for the aware error and difference ERPs are compared in Fig. 4b and c.

Tonic cortical arousal and error awareness

Finally, we investigated whether there was a relationship between tonic slow/fast wave ratios in the EEG power spectrum and errors of commission, error awareness and the amplitudes of the three error-related components (ERN, early positivity and Pe) on aware errors. Fifteen of our 19 participants made sufficient aware errors (at least 20) to be included in the correlation between the ERPs and slow/fast wave ratios but the remaining correlations included all 19 participants. A significant relationship was observed between tonic theta/beta ratios and the percentage of aware errors (aware errors/total errors: $r = -0.478$, $P < 0.05$) and the amplitude of the Pe ($r = -0.661$, $P < 0.01$). Alpha/beta ratios were also correlated with Pe amplitude ($r = -0.546$, $P < 0.05$) and there was a close-to-significant relationship with percentage of aware errors ($r = -0.44$, $P = 0.059$). Thus a low ratio of slow/fast wave activity in the EEG spectrum (indicating increased cortical arousal) was associated with better awareness of one's errors and a larger Pe amplitude. No significant correlations were observed between theta/beta or alpha/beta ratios and errors of commission or the amplitudes of the ERN or early positivity.

Discussion

In the present study, participants performed a Go/No-go response task and were asked to indicate any commission errors by pressing a second 'awareness' button. Although this requirement imposes a dual-task element which could conceivably contaminate 'unaware' errors with dual-task failures we argue that error awareness cannot be explicitly verified without requiring a secondary response. Moreover, the EDA data (Fig. 2) indicates a marked absence of the cognitive–emotional response that is seen following conscious recognition of

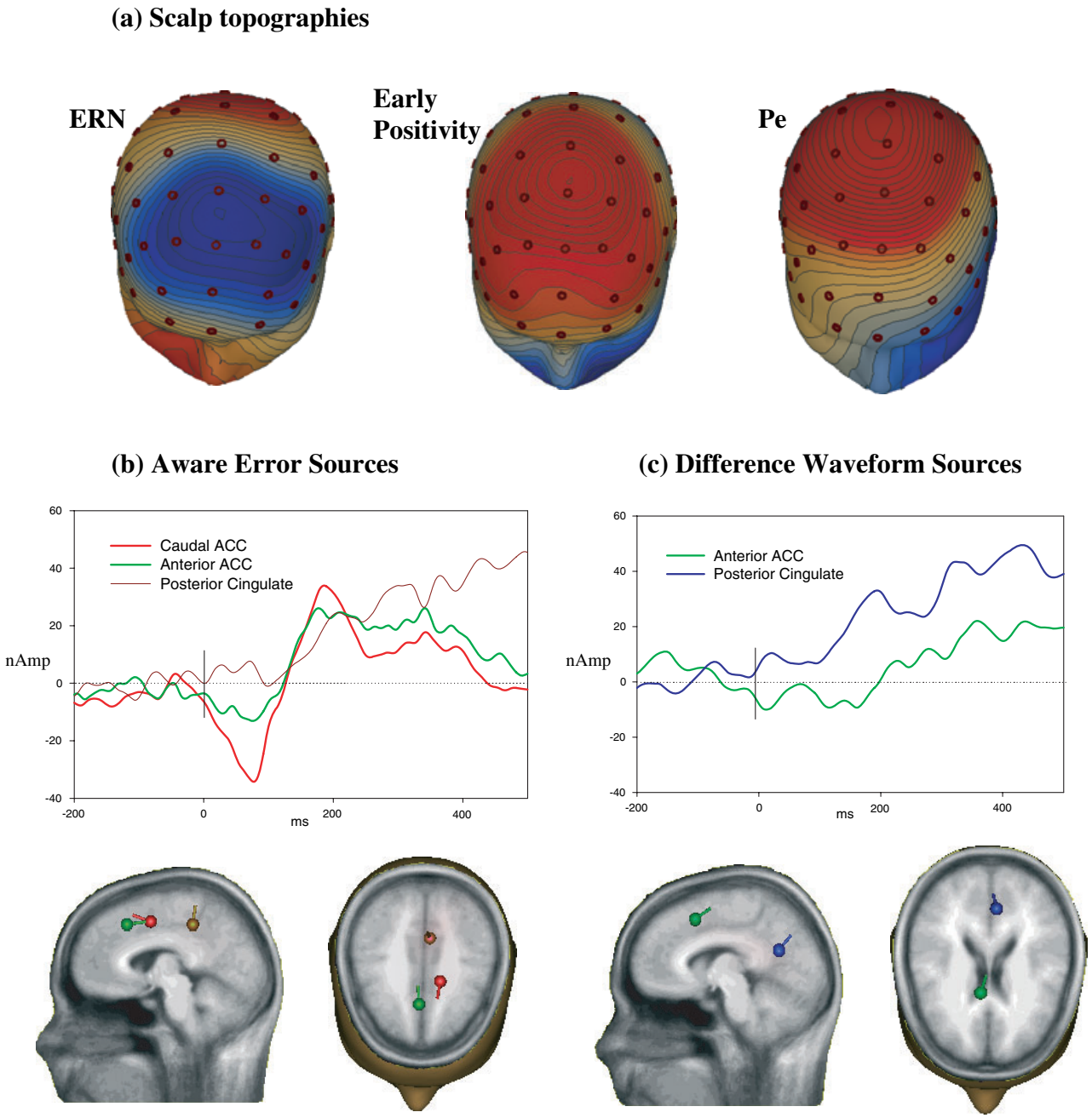

FIG. 4. ERP scalp topographies (a), source time courses and dipole locations for the aware error (b) and difference waveform models (c). The caudal ACC dipole (red) explains the ERN and the early positivity; the anterior ACC dipole (green) and the posterior cingulate–precuneus dipole [brown in (b), blue in (c)] accounts for the Pe.

significant events such as action errors (O'Keeffe *et al.*, 2004), suggesting that the EAT does provide a good measure of error awareness. It has been suggested that autonomic changes provide 'somatic markers' that guide our behaviour and may also contribute to the emergence of conscious awareness (Damassio *et al.*, 1991). An interesting question for future work would be to investigate the temporal relationship between the onset of autonomic responses and behavioural indices of error awareness.

The behavioural data indicated that, while participants made more commission errors on Incongruent trials, a significantly greater proportion of unaware errors were made on Repeat trials. This difference may be explained by the fact that when participants make an error on Incongruent trials, the current stimulus can be immediately identified as a No-go target. In contrast, when an error is made on a Repeat trial the participant must have a memory of the preceding trial in order to identify the current stimulus as a No-go target. Given that participants appear to have found it easier to withhold on Repeat No-go trials the increased rate of unaware errors suggests that failed inhibitions on Repeat trials may have been accompanied by a more dramatic lapse of attention than errors made on Incongruent trials. In addition, it was found that response times were significantly slower on unaware errors than on aware errors. This finding is similar to that reported by Endrass *et al.* (2005) and also appears consistent with the view that unaware errors are precipitated by lapses of attention.

When participants made an error of commission a response-locked waveform was generated which exhibited three clear components. The ERN was the earliest component and its amplitude was not modified by awareness of errors. Although the ERN was most pronounced for errors, an attenuated ERN-like component was also evident following correct Go responses. This phenomenon, known as the 'correct response negativity', is thought to reflect levels of continuous performance monitoring during the task (Vidal *et al*., 2000; Ridderinkhof *et al*., 2003). Thus, while the processes reflected in the ERN are enhanced when one makes an error, they are also engaged in an ongoing manner throughout task performance. The latter probably facilitates the detection of subthreshold levels of conflict or uncertainty that do not necessarily result in errors but signal the need for fine-grained performance adjustments. The second component noted in the present study was a strong early positive deflection immediately following the ERN and maximal over frontocentral scalp sites. A similar positive deflection has been noted in previous studies and previous analyses using prestimulus baselines and analyses within the frequency domain have failed to distinguish it from the ERN (Luu *et al*., 2004; Debener *et al*., 2006). In addition, a previous study has reported that the ERN and early positivity share the same cortical generator (caudal ACC), leading to the suggestion that they are part of a single performance-monitoring component (Van Veen & Carter, 2002). Interestingly, in the present study the amplitude of this early positive wave did not distinguish between any of the response types (correct Go, aware error, unaware error). The strong ERN and early positive waveforms on correct trials may be explained by the introduction of two competing No-go conditions in the EAT task (Repeat and Incongruent No-gos), resulting in a higher degree of uncertainty on Go trials and therefore increased engagement of monitoring processes.

In contrast, the classic Pe, which followed the early positive wave and had a more posterior distribution, was only present when participants were aware of their errors. The marked absence of a Pe when participants were not aware of their errors provides clear confirmation of the findings of Nieuwenhuis *et al.* (2001) and therefore we have shown that modulation of the Pe by error awareness is not limited to the oculomotor modality but generalizes to manual responses. The presence of the ERN and early positivity whether or not participants were aware that they had made an error indicates that the brain possesses preconscious detection mechanisms while later processing stages indexed by the Pe only occur when an error has been consciously detected.

Although there is now a wealth of evidence implicating the ACC in error processing, and in the generation of the ERN and Pe, there has been little investigation of the role that this region plays in error awareness. In the present study we conducted the first source analysis of electrophysiological activity that was specific to errors made with and without awareness. Our results appear to confirm a role for the caudal ACC in early aspects of error processing reflected in both the ERN and early positivity and in this respect confirm previous reports (Dehaene *et al*., 1994; Van Veen & Carter, 2002; Hermann *et al*., 2004; Van Boxtel *et al*., 2005; Debener *et al*., 2006). In common with two previous studies, we found that the ERN and early positivity were generated by the same ACC region (Van Veen & Carter, 2002; Debener *et al*., 2006), thus supporting the contention that they are part of the same monitoring component. However, the early positivity did have a more central scalp distribution than the ERN, suggesting that it may be generated by a distinct region of the ACC. Further work will be required to specify the functional significance of this component. Source localization of the Pe also indicated an ACC generator but in a distinctly more anterior region. Our Pe model also indicated a second source around the posterior cingulate–precuneus, a region which has been previously attributed a role in posterror processing (Badgaiyan & Posner, 1998; Menon *et al*., 2001) and more broadly in self-awareness and consciousness (Cavanna & Trimble, 2006). The same regions were indicated when the unaware error waveform was subtracted from the aware error waveform leaving only activity relating to error awareness.

There have been four previous attempts to localize the generator(s) of the ERN and Pe simultaneously. Source analyses performed by Van Veen & Carter (2002) and by Van Boxtel *et al*. (2005) also identified an ACC generator for the Pe although in a more rostral region than in the present study. Hermann *et al*. (2004) also found separate ACC sources for the ERN and Pe but the source of the Pe was found in a more caudal ACC region. Imaging and lesion studies indicate that the ACC can be broadly divided into two functionally distinct subregions, a caudal region associated with basic cognitive processes such as conflict and uncertainty detection and a more rostral region thought to process the subjective or emotional significance of events and stimuli (Bush *et al*., 2000). A recent study by Taylor *et al*. (2006) has noted clear individual differences in the ACC subregions activated during error processing. Individual variations in affective and motivational responsiveness to making an error may explain in part why there has been a certain degree of inconsistency in localizing the source of the Pe. It is also important to acknowledge that variation across studies may also arise from the limited spatial resolution of source localization associated with the inverse problem. Nevertheless the findings of the present study are consistent with those of Van Veen & Carter (2002) and of Van Boxtel *et al*., 2005) such that the ERN and Pe do appear to be generated by distinct regions within the ACC. Understanding the neural basis of conscious error detection may provide an important avenue to a better understanding of the failures of self-awareness seen in clinical groups in which reduced awareness of one's deficits can represent a significant barrier to rehabilitative interventions and independent living (Hart *et al*., 1998).

The source model obtained in the present study does appear to be at odds with the fMRI findings of Hester *et al*. (2005), who found no additional ACC or posterior cingulate–precuneus activations when participants were aware of their errors. Such differences are not necessarily surprising given the ability of ERPs to separate minute portions of trial activity. The comparatively limited temporal resolution of fMRI may cause subtle effects, particular to finite portions of processing within a trial, to go undetected. For example, the subtle increases in regional ACC activity that are evident in source analysis might not be detected by fMRI if averaging across a longer epoch causes rostral and caudal activations to become convolved. Nevertheless, caution should be exercised when interpreting the present source findings. These data highlight the potential pitfalls in making direct comparisons between different measures across studies. More direct evidence that the ERN is generated by the ACC has been provided through trial-by-trial couplings of EEG and fMRI signals (Debener *et al*., 2006) and through intracerebral recording studies (Brazdil *et al*., 2005). Further work of this kind will be required in order to elucidate the discrepancy between fMRI correlates of conscious error processing and repeated attempts to localize the generators of the Pe through source analysis.

It is important to note that the observed modulatory role of awareness does not necessarily imply causality in the generation of the Pe. Our results are also consistent with alternative accounts of the Pe as a reflection of compensatory adjustments (Hajcak *et al*., 2003) or subjective and/or emotional appraisal (Van Veen & Carter, 2002) but suggest that these processes may be reliant on conscious awareness. The finding that the Pe was correlated with tonic EEG measures of

arousal may be particularly illuminating in the context of recent models linking the Pe to the same underlying process as the P3, a component which can be elicited by any motivationally significant stimulus and which is thought to reflect the evaluation of that stimulus (Nieuwenhuis *et al*., 2005; Overbeek *et al*., 2005). The P3 and Pe share several obvious characteristics including centroparietal scalp topography, positive polarity and relative peak latency (300–500 ms relative to stimulus and response onset, respectively). These similarities have led to speculation that the two components may be part of the same process (Falkenstein *et al*., 2000; Nieuwenhuis *et al*., 2005) whereby the Pe would reflect an additional P3-like evaluation of the incorrect response. This possibility raises a number of interesting and testable questions. In the present paper, we examined the influence of arousal on the Pe.

Recently, Nieuwenhuis *et al*. (2005) reviewed the accumulating evidence that the P3 indexes a phasic arousal response, originating in the locus coeruleus system, which is designed to increase the speed of information processing by enhancing neural responsivity in task-relevant cortical regions. Thus when a motivationally significant stimulus has been detected, an arousal response is triggered which facilitates further processing of that stimulus. Locus coeruleus (LC) neuronal activity has been shown to precede changes in behavioural states and appears to play a modulatory role in maintaining the alert state, most probably via the action of the neurotransmitter noradrenaline. Experimental alterations in LC activity indicate a causal relationship between LC activity and cortical arousal reflected in EEG power spectra (Swick *et al*., 1994). In addition, manipulations of tonic LC activity have been shown to affect the amplitude of the P3. Suppression of LC activity, associated with drowsiness and hypo-arousal, precedes increases in slow wave (theta, alpha) EEG activity, decreases in fast wave (beta) activity and attenuation of the P3 (Swick *et al*., 1994). When LC activity is enhanced these effects are reversed. Our results show a significant relationship between tonic levels of cortical arousal (as measured by the ratio of slow to fast wave activity during task performance) and the amplitude of the Pe. Hence the present study offers new evidence that the Pe and P3 may share a key functional characteristic: modulation by cortical arousal. The absence of any such relationship with the ERN in the current study further distinguishes these two error-related ERP components. Recent work has demonstrated that the ERN, and not the Pe, is sensitive to changes in dopaminergic neurotransmission relating to reinforcement learning (reviewed in Overbeek *et al*., 2005). Therefore, evidence now exists that the ERN and Pe represent functionally distinct elements of error processing that are dependent on different brain regions and different neurotransmitter systems. An obvious question which remains is how Pe-related processes actually influence task performance.

With some exceptions, the majority of studies have failed to show any relationship between the Pe and posterror performance adjustments (Falkenstein *et al*., 2000; Fiehler *et al*., 2005; Ullsperger & von Cramon, 2006). Most of these studies, however, have relied on a short-term measure of corrective behaviour, such as immediate RT slowing, that may not necessarily reflect a definite change in performance strategy. For example, Gehring & Knight (2000) showed that patients with prefrontal lesions exhibited normal posterror slowing but were less likely to correct their errors on the next target. In addition there is evidence that posterror slowing can occur even when errors have not been consciously perceived (Rabbitt, 2002; Hester *et al*., 2005). These findings tell us that there may be dissociable forms of posterror control. One hypothesis is that while the ERN reflects short-term increases in cognitive control that are not reliant on awareness and result in remedial action on the current trial, the conscious error processes indexed by the Pe may engender broader adaptations of performance strategy that are likely to result in longer term changes in behaviour. Experiments that use a wider variety of posterror correction measures (e.g. short-term measures such as response force on error trials and posterror RT vs. longer term measures such as performance on the next target or changes in RT variability) will be necessary to answer this question.

Our data also show a relationship between EEG measures of cortical arousal and individual rates of error awareness. Again, this finding is consistent with evidence of LC functioning which indicates that low levels of tonic activity can lead to the absence of the normal phasic response to motivationally significant events (Aston-Jones & Cohen, 2005; Nieuwenhuis *et al*., 2005). Reduced tonic activity in the LC, reflected in our tonic EEG measures, would lead to a dampening of the phasic response to error feedback which, as a result, may be too weak for the error to reach conscious awareness. Thus we speculate that when tonic levels of arousal fall below a certain threshold, the erroneous response will not trigger the phasic facilitation of cortical error processors, reflected in the Pe, that are necessary for awareness. Further investigation using trial-by-trial couplings of error awareness, Pe amplitude and both tonic and phasic LC activity would be desirable to confirm this relationship.

The present study clearly demonstrates that a proper understanding of the error processing system requires differentiating error-related brain activations in terms of their relationship with conscious awareness. Verifying levels of error awareness may be particularly important in the context of studies that have used these ERP components to investigate self-monitoring deficits in clinical populations such as attention-deficit hyperactivity disorder and schizophrenia (e.g. Mathalon *et al*., 2002; Wiersema *et al*., 2005).

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Abbreviations

ACC, anterior cingulate cortex; Best, minimum variance within the fit interval; EAT, error awareness task; EDA, electrodermal activity; EEG, electroencephalogram; EOG, electrooculogram; ERN, error-related negativity; ERP, event-related potential; LC, locus coeruleus; Pe, error positivity; R.V., residual variance; RT, reaction time; SCR, skin conductance response.

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