Error-related brain activity in obsessive–compulsive undergraduates[☆]

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Abstract

Error-related negativity (ERN/Ne) is a component of the event-related brain potential (ERP) associated with monitoring action and detecting errors. It is a sharp negative deflection that generally occurs from 50 to 150 ms following response execution and has been associated with activity involving the anterior cingulate cortex (ACC). An enhanced ERN has recently been observed in patients with obsessive–compulsive disorder (OCD). We extended these findings by measuring the ERN in college undergraduates with OC characteristics as measured by the Obsessive–Compulsive Inventory (OCI). Eighteen high-OC subjects and 17 low-OC subjects performed a modified Stroop task with equal emphasis placed on speed and accuracy. Response-locked ERPs revealed a frontally maximal negativity associated with erroneous responses that was significantly larger in the high-OCI group. There were no performance differences between the two groups. Our results support the view that the characteristics associated with OCD are related to hyper-functioning error and action-monitoring processes. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

Obsessive-compulsive disorder (OCD) is characterized by 'recurrent obsessions or compulsions…that are severe enough to be time consuming…or cause marked distress or significant impairment' (American Psychiatric Association, 1994). The repeated doubt and subsequent checking that surround actions is particularly salient features of OCD. Within a cognitive framework, these intrusive obsessions and subsequent compulsions associated with OCD can be viewed in terms of dysfunctional response monitoring.

Veale et al. (1996) reported that patients with OCD engage in excessive response monitoring, particularly after the commission of errors. Using a variation of the Tower of Hanoi problem, Veale et al. noted that OCD patients performed slower than control subjects after an error, but interesting-

ly, OCD patients performed just as well as the control subjects in terms of overall success.

Response monitoring is one of many executive processes or functions that have been localized, in numerous studies, to the frontal areas of the brain (Damasio, 1996; Rolls, 1996; Cohen et al., 1996; Kolb and Whishaw, 1996). Purcell et al. (1998) provide evidence that patients with OCD have specific cognitive deficits that indicate frontal–striatal dysfunction.

Hypothesized frontal dysfunction in OCD is also supported by recent event-related potential (ERP) studies. Di Russo et al. (2000) examined stimulus-locked ERPs while OCD and control subjects performed a go/no-go task. The go/no-go task was chosen as an example of a discrimination response task that requires both response activation and inhibition—functions that rely on frontal regions of the brain. Interestingly, the Di Russo et al. (2000) study found that OCD patients showed comparable activation on both target (go) and non-target (no-go) trials, whereas controls showed greater activation on non-target (no-go) trials. Di Russo et al. (2000) argue that the go/no-go P3 component reflects a frontal response-inhibition system—and their results indicate that this frontal system is hyperactive in the OCD group.

PET and fMRI imaging studies have supplied additional evidence that the symptoms associated with OCD are related to the hyperactivity of particular components of the frontostriatal system: orbitofrontal cortex (OFC); anterior cingulate cortex (ACC); and dorsolateral prefrontal cortex (DLPFC; Breiter et al., 1996; Baer et al., 1995). The neurobiological finding that OCD is associated with a hyperactive ACC may actually underlie the cognitive-behavioral component of enhanced response monitoring in OCD. Posner and Rothbart (1998) suggested that the human ACC may be involved in the subjective monitoring of behavior. In a similar vein, ERP experiments have suggested that the ACC is active in a system responsible for action monitoring, especially in terms of the commission of errors (Dehaene et al., 1994).

Studies that measure ERPs during speeded reaction time tasks consistently find a sharp negative deflection in the response-locked ERP that begins around the time of an incorrect response and peaks approximately 50–150 ms after the commission of an error (Gehring et al., 1993; Dikman and Allen, 2000; Luu et al., 2000; Falkenstein et al., 2000; Scheffers and Coles, 2000; Nieuwenhuis et al., 2001). This component is found at frontal recording sites along the midline, and is virtually non-existent in the waveform for correct trials. The sharp negative deflection associated with errors is referred to as error negativity (Ne: Hohnsbein et al., 1989) or error-related negativity (ERN; Gehring et al., 1990).

The ERN is thought to reflect the activity of a general error-processing system, active across stimulus and response modalities. In terms of response modality, it has been shown that the ERN is generated when subjects respond with foot or hand movements, as well as with finger or eye movements (Holroyd et al., 1998; Van't Ent and Apkarian, 1999). As far as stimulus modality is concerned, the ERN is generated when stimuli are presented in either the visual or auditory modality (Bernstein et al., 1995). The magnitude of the ERN is the same for fast vs. slow errors (Falkenstein et al., 2000); it is larger when accuracy is emphasized over speed and larger when subjects are more certain that they have made a mistake (Falkenstein et al., 2000; Gehring et al., 1993).

The size of the ERN has also been shown to be sensitive to the 'magnitude' of the error. Falkenstein et al. (2000) had subjects respond in four ways to a speeded reaction time task with two fingers on their right hand and two fingers on their left hand. Interestingly, Falkenstein et al. (2000) found that the ERN for hand-errors was larger than the ERN for finger-errors. Additionally, 'double' errors (wrong hand and wrong finger) produced a larger ERN than 'single' errors (wrong finger or wrong hand). In a similar study, Bernstein et al. (1995) quantified the number of movement parameters used in responding, and found that the ERN magnitude was enhanced for errors that were wrong in more response dimensions.

The generality of ERN involvement in error processing is further demonstrated in a study by Miltner et al. (1997), who found an ERN following negative feedback. In this study, subjects had to estimate an amount of time, and were provided positive or negative feedback depending on wheth-

er their estimation was acceptable or not acceptable, respectively. Because the subjects had no other information by which they might judge the accuracy of their estimations, the feedback was crucial to the response-monitoring process. Even though the feedback was provided 600 ms after the response, Miltner et al. (1997) found an ERN time-locked to the negative feedback signal.

ERN studies that have been carried out with whole-head recording systems and analyzed with Brain Electromagnetic Source Analysis (BESA; Scherg, 1990), a computer algorithm used to estimate the number and location of the neural generators producing the scalp activity, have indicated that the ERN is generated by a single source in the medial frontal cortex (Dehaene et al., 1994; Holroyd et al., 1998). This is consistent with the suggestion of Gehring et al. (1993) that the source is the ACC. As indicated above, the ACC has also been associated with symptoms of OCD.

To establish an association between the ERN and OCD symptoms, Gehring et al. (2000) measured the ERN in a group of patients with OCD; they found an enhanced ERN in the OCD group, relative to matched controls. Additionally, the magnitude of the ERN was correlated with OCD symptom severity. In a similar study using fMRI, Ursu et al. (2001) found increased error-related activity in the ACC of patients with OCD; error-related activity was positively correlated with OCD symptom severity.

The present study was conducted to determine whether the enhanced error-related activity found in patients with OCD could also be found in a non-clinical population with OC characteristics as assessed by the Obsessive–Compulsive Inventory (Foa et al., 1998). To evaluate this hypothesis, we recorded ERP activity while subjects performed a speeded reaction time task that has been related to cingulate activity in fMRI studies (Peterson et al., 1999). In particular, we used the Gehring et al. (2000) modified Stroop in which subjects were shown color words such as 'red', presented in either a congruent color (red) or an incongruent color (blue). The subjects' task was to respond to the color of the stimuli with a button press as quickly and accurately as possible.

2. Methods

2.1. Subjects

Undergraduate students in an introductory psychology class completed the Obsessive–Compulsive Inventory (OCI; Foa et al., 1998), a scale composed of 42 items that are scored for both frequency and distress. The OCI was designed to be administered to both clinical and non-clinical populations and can be used as a screening test, as well as a way of determining symptom severity. The OCI has excellent reliability (both coefficient alpha and test–retest above $r = 0.84$) and convergent and discriminant validity (for a more complete description of the OCI, see Foa et al., 1998).

Thirty subjects (15 male, 15 female) from the top 12% of the OCI distribution were randomly selected and assigned to a high-OC group and thirty subjects (15 male, 15 female) from the bottom 12% were randomly selected and assigned to a low-OC group (OCI frequency: high OCI = 75.2; low OCI = 15.3; OCI distress: high OCI = 66.1; low OCI = 1.7). From the original list of 60 potential subjects, 12 low- and 7 high-OCI subjects either cancelled or failed to keep their laboratory appointments and the data from six additional subjects were lost due to equipment malfunction or poor quality recordings. No subjects discontinued their participation in the experiment once the procedures had begun. The final sample consisted of 18 high-OCI (9 male, 9 female) and 17 low-OCI (6 male, 11 female) subjects. All subjects received course credit for their participation and the experimenter was blind to group membership until data reduction was complete.

2.2. Task

The Stroop task was administered on a Pentium I class computer, using Presentation software (Neurobehavioral Systems, Inc.) to control the presentation and timing of all stimuli, the determination of response accuracy, and the measurement of reaction times.

Throughout the task, subjects were shown three color words ('red', 'green' and 'blue') presented either in red or green font on a 17-inch computer

monitor using a black background. Each word occupied approximately 3° of visual angle. A fixation mark (+) was presented below the stimuli, prior to each word. The subjects were instructed to press the right or left mouse button in response to the color of the words. In this way, there were congruent conditions ('red' in red, 'green' in green), incongruent conditions ('red' in green, 'green' in red), and neutral conditions ('red' in blue, 'green' in blue).

2.3. Procedure

After a brief description of the experiment, EEG/EOG sensor electrodes were attached and the subject was given detailed task instructions. Each subject was seated 0.5 m directly in front of the computer monitor and given two blocks of 24 practice trials. The subject was told to press one mouse key if the color word was presented in red, and press the other if the color word was presented in green. In one condition, the subjects were told to press the left button on the mouse when the color word was written in red, and the right mouse button when the word was written in green. In the other condition, the correspondence between mouse button and word color was reversed. These conditions were counter-balanced across subjects. The subjects were told to place equal emphasis on speed and accuracy in their responses. Following two practice blocks, the subjects received 24 blocks of 48 trials (1152 total trials) with each block initiated by the subject. Word stimuli were presented for 200 ms at random intervals between 2000 and 2400 ms.

2.4. Psychophysiological recording, data reduction and data analysis

The electroencephalogram (EEG) was recorded using an ECI electrocap. Recordings were taken from three locations along the midline: frontal (Fz), central (Cz) and parietal (Pz). In addition, Med-Associates miniature Ag–AgCl electrodes were placed on the left and right mastoids (A1 and A2, respectively). During the recording, all activity was referenced to Cz. The electro-oculogram (EOG) generated from blinks and vertical

eye movements was also recorded using Med-Associates miniature electrodes placed approximately 1 cm above and below the subject's right eye. The right earlobe served as a ground site. All electrode impedances were below 10 KΩ.

Fz, Pz, A1, A2 and EOG were recorded by a Grass Model 7D polygraph with Grass Model 7P1F preamplifiers (bandpass = 0.05–35 Hz). The EEG was digitized on a laboratory microcomputer at 200 samples/s, using VPM software (Cook, 1998). Data collection began at stimulus presentation and continued for 1500 ms.

Off-line, the EEG for each trial was corrected for vertical EOG artifacts (see Gratton et al., 1983; Miller et al., 1988) and then re-referenced to the average activity of the mastoid electrodes. Trials were rejected and not counted in subsequent analysis if there was excessive physiological artifact, or if the reaction time fell outside of a 200–800-ms window. Finally, the EEG for each trial was time-locked to its respective reaction time and averaged across trials to yield error- and correct-trial ERPs for each electrode site.

To quantify the ERN, each data point after response onset was subtracted from a baseline equal to the average activity in a 100-ms window prior to the response. The ERN was then defined as the most negative peak occurring in a window from 0 to 150 ms post-response. Because there has been some suggestion that uniformly fast reaction times can give rise to stimulus-related activity in the response-locked ERN, the ERN was evaluated for two sets of ERPs. The first set of ERPs involved the comparison of errors to *all* correct trials; the second set of ERPs involved the comparison of errors to a sub-set of *reaction-time matched* correct trials. The ERN and performance measures were statistically evaluated using SPSS (Version 10.0) General Linear Model software.

3. Results

3.1. ERN—all trials

The response-locked average waveforms are presented in Fig. 1. Consistent with previous studies, the ERN was observed as a negative deflection that began shortly after the response was made
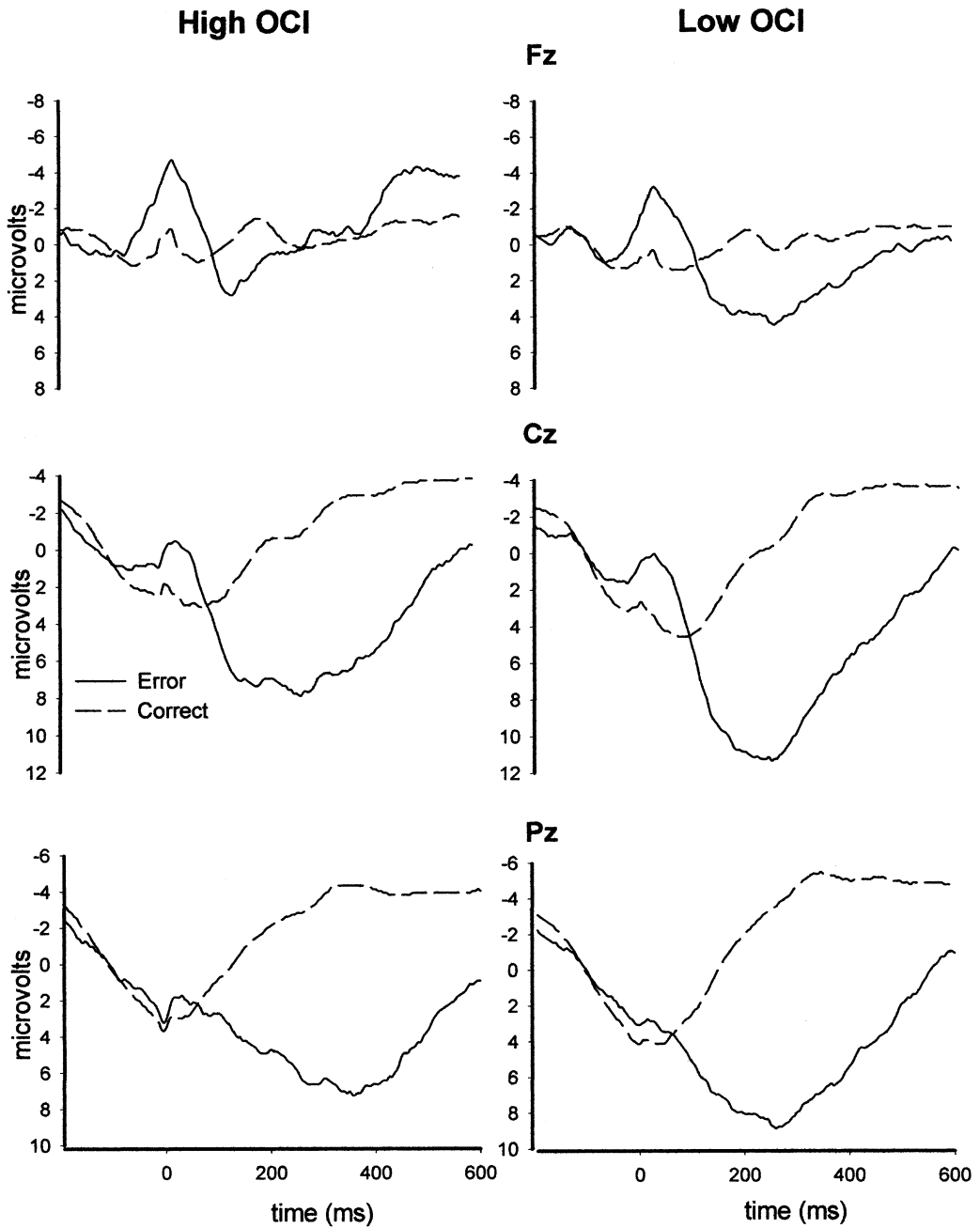
Fig. 1. ERP waveforms from high-OCI (left) and low-OCI (right) subjects for both error and correct trials at the Fz, Cz and Pz recording sites.
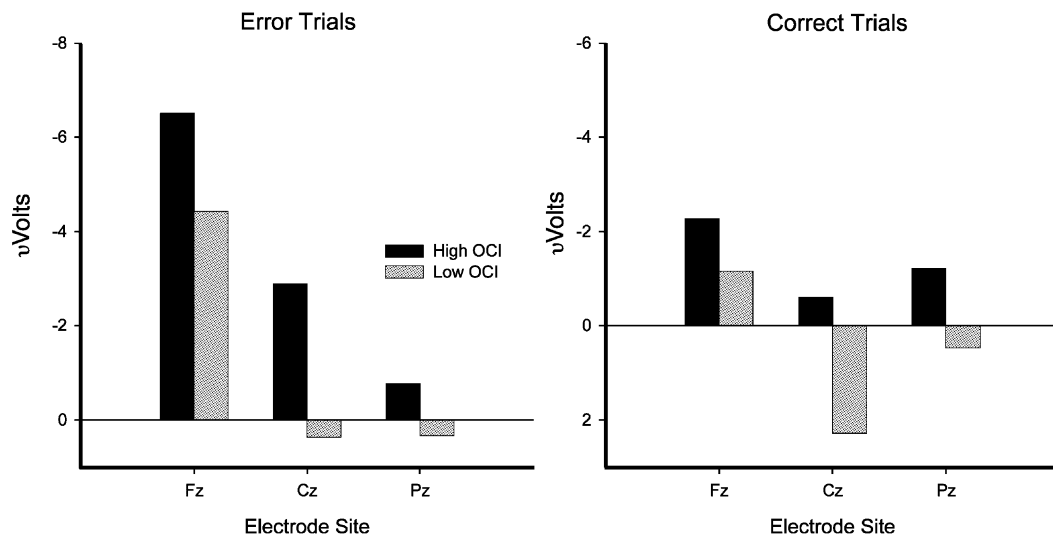
Fig. 2. ERN magnitude for Error and Correct trials as a function of subject group and electrode site.

and generally peaked 40–100 ms later. The quantified ERN magnitudes are presented in Fig. 2.

Both Figs. 1 and 2 clearly show that when all subjects made errors, there was a sharp negative deflection that peaked at approximately 55 ms post-response, primarily at the frontal recording site. A 2 (Group)×2 (Trial Type)×3 (Electrode Site) analysis of variance (ANOVA) with Greenhouse–Geisser corrected *P*-values supported the impression that the ERN was predominantly frontal ($F_{2,66} = 46.57$, $P < 0.001$), that negative activity was significantly greater when subjects made errors ($F_{1,33} = 17.75$, $P < 0.001$) and that trial and site interacted such that the difference in the ERN magnitude between correct and incorrect trials was largest at the Fz recording site ($F_{2,66} = 55.08$, $P < 0.001$). Thus, our results are consistent with previously reported ERN morphology and topography.

The hypothesis that ERN peak magnitude would systematically vary between high- and low-OCI groups was confirmed. The ANOVA yielded a significant group effect ($F_{1,33} = 6.75$, $P < 0.05$); the ERN was larger among high-OCI subjects than it was among the low-OCI subjects. Interestingly, there was no three-way group by trial by location interaction and no interaction between group and trial type, suggesting that ERN activity was

enhanced in the high-OCI group following both correct and error responses.

3.2. ERN—RT matched trials

Although, on average, each ERP is response-locked between 400 and 500 ms after the presentation of the stimulus, it is possible that some of the ERP activity could nonetheless be stimulus driven (Gehring et al., 1993; Scheffers et al., 1996). If RTs on error trials are faster and more uniform than on correct trials, these trials might contribute stimulus-related artifact to the ERN. To avoid this potential confound, we further examined a subset of the correct trials for each subject. To do this, each error trial was matched to a correct trial on the basis of reaction time. In this way, any stimulus-locked activity resulting from fast and uniform reaction times would equally affect correct and error trials and eliminate the potential confound.

After the subset of matched trials was selected on the basis of reaction time, ERP averages for matched-correct and error trials were once again created and the peak negativity in the 0–150 ms window was identified and scored. As before, the ANOVA revealed a highly significant effect for

Table 1
Reaction time and accuracy means

	Reaction time (ms)		Accuracy	
	Error	Correct	No. of errors	% Correct
High OCI	375	419	73.33	93.33
Low OCI	367	415	84.12	92.22

electrode site ($F_{2,66} = 64.27$, $P < 0.001$) and trial type ($F_{1,33} = 27.04$, $P < 0.001$), as well as a significant interaction between electrode site and trial type ($F_{2,66} = 16.93$, $P < 0.001$). Most importantly, the between-subject effect of group was still significant ($F_{1,33} = 5.46$, $P < 0.05$), and there was no interaction between group and trial type or three-way interaction between group, location, and trial type. That is, the ERN was enhanced in high-OCI subjects, and this enhancement was present on both correct and incorrect trials.

3.3. Performance measures

Accuracy and RT data are presented in Table 1. Because the number of rejected trials varied between subjects, the number of errors and percentage correct are not redundant statistics, and both are reported. Although subjects tended to make relatively few mistakes, there were no accuracy differences between groups in terms of percentage correct ($F_{1,33} < 1$) or number of errors ($F_{1,33} < 1$). In terms of reaction time, subjects had faster reaction times for errors, relative to correct responses ($F_{1,33} = 111.82$, $P < 0.001$); there was, however, no interaction between trial and group ($F_{1,33} < 1$) and there was no difference in RT between the two groups ($F_{1,33} < 1$).

Several recent ERN studies on speeded reaction-time tasks have noted a slow-down following commission errors (Falkenstein et al., 2000; Luu et al., 2000; Nieuwenhuis et al., 2001). To examine this effect and to rule out the possibility that the slow-down effect was non-specific—i.e. the result of fast responses and simply a matter of regression toward the mean—we calculated the average reaction time for trials that followed an error and compared this RT to the average RT for trials that followed correct trials matched to the error trials on the basis of reaction time. These data are presented in Table 2.

The analysis of these data confirmed that trials subsequent to error trials and RT-matched correct trials were associated with slower RTs ($F_{1,33} = 105.08$, $P < 0.001$), that this slow-down effect was significantly greater after errors than after equally rapid correct responses ($F_{1,33} = 18.89$, $P < 0.001$). Thus, although significant regression was noted, there remains compensatory slowing that is specific to error trials. As was the case for the other performance measures, however, this compensatory slowing was independent of OCI status ($F_{1,33} < 1$).

4. Discussion

The results of our analyses indicate that the high-OCI and low-OCI groups differ in electrophysiological measures related to error monitoring. Thus, our initial hypothesis that ERN magnitude would be enhanced in a group of undergraduates with obsessive–compulsive characteristics, as assessed by the OCI (Foa et al., 1998), was confirmed. In particular, we found significantly

Table 2
Accuracy and reaction-time (RT) data for error trials, RT-matched correct trials and trials subsequent to each

	High OCI	Low OCI
RT for error trials	375	367
RT for RT-matched correct	375	367
RT following error	434	420
RT following RT-matched correct	405	396
Accuracy following error	92%	92%
Accuracy following RT-matched correct	94%	91%

larger error-related activity at the anterior (Fz) recording site in the high-OCI group. These results can be viewed as a systematic replication of Gehring et al. (2000), who found a heightened ERN in a population with clinical OCD. Specifically, we extended the Gehring et al. (2000) finding to a non-clinical sample, thereby demonstrating that an enhanced ERN is related to obsessive–compulsive characteristics, independent of formal clinical diagnosis.

Interestingly, we did not find an interaction between group (high-OCI vs. low-OCI) and trial type (correct vs. error) suggesting that the heightened error-related activity in the high-OCI group was not specific to error trials. Rather, the enhanced ERN was associated with both error *and* correct trials. This finding is somewhat at odds with the finding reported by Gehring et al. (2000) who interpreted their results in terms of specificity. That is, they argued that OCD patients show an enhanced ERN only following errors. Interestingly, however, the ERP waveforms presented in their Fig. 1 show a distinct correct-trial ERN in the OCD group that is absent in the waveforms obtained from control subjects.

The meaning and significance of ERN-like activity on correct trials is currently unclear. It has led some researchers to reject the view that the ERN is associated exclusively with error-processing (Falkenstein et al., 2000; Vidal et al., 2000), though others (e.g. Coles et al., 2001) argue that error-processing, more broadly construed, is still the most parsimonious conceptualization. Regardless of the degree of specificity in the relationship between ERN and errors per se, it is not unreasonable that obsessive–compulsive characteristics would be associated with excessive response monitoring even following 'correct' behaviors. After all, it is precisely this excess, continually checking and doubting actions, that characterizes OCD symptomatology in the clinical literature, and this excessive response monitoring occurs regardless of whether or not errors have actually been made.

Although the electrode montage employed in the present study was limited to three recording sites, the distinct frontal maximum of the ERN is consistent with other more focused studies using both ERP and functional MRI techniques that consistently localize the neural source of the error-related activity to medial frontal cortex, specifically the ACC (Dehaene et al., 1994; Gehring et al., 1993, 2000; Luu et al., 2000). As indicated above, the localization of the ERN to medial frontal brain areas, the localization of executive functions to frontal brain areas, and the enhancement of the ERN in patients with OCD are all consistent with the hypothesized hyperactivity of at least some components of the frontal-striatal system. The present data suggest that this hyperactivity can be observed in young-adult college students who report a large number of obsessive–compulsive characteristics.

While high- and low-OC subjects differed significantly on the electrophysiological measure, no performance differences between the two groups were evident. As in the Gehring et al. (2000) study with OCD patients, high- and low-OCI subjects were similar in their reaction times and their accuracy during the Stroop procedure. Like previous studies on the ERN (Gehring et al., 1993; Nieuwenhuis et al., 2001), we found that the RT on trials that followed an error were significantly slower than they were on the error trials, but again, this slow-down effect was equivalent in the two groups. In short, although the high-OCI group had a significantly enhanced ERN, there was no corresponding group difference on overall RT or accuracy, and no evidence that the ERN enhancement was associated with compensatory post-error slowing.

Though we refer to our high-OCI subjects as 'non-clinical', a formal clinical assessment was not conducted. In point of fact, both the OCI-frequency and OCI-distress scores for subjects in the high-OCI group were comparable to scores reported in the clinical range for patients with OCD (Foa et al., 1998). It is likely that if these high-OCI subjects had presented for a diagnostic workup, at least some would have received an OCD diagnosis. Thus, our belief that the ERN is enhanced in subsyndromal, or non-clinical, OCD must be tempered until a more thorough clinical assessment of these subjects is possible.

Nonetheless, the finding that college students with obsessive–compulsive characteristics show electrocortical evidence of excessive response

monitoring is consistent with the Gehring et al. (2000) finding with OCD patients and fits well into the general body of research on abnormal frontal activity associated with OCD. For example, a number of recent PET studies have found that distinct patterns of pretreatment frontal activity differentially predict OCD treatment outcome for pharmacotherapy and behavior therapy. Brody et al. (1998) found that lower pretreatment left orbito-frontal cortex (OFC) metabolism predicted better response to treatment with fluoxetine, whereas higher pretreatment left OFC metabolism predicted better response to behavior therapy. In a similar study, Brody et al. (1996) report that higher OFC metabolism was associated with poorer response to pharmacotherapy. Finally, Baxter et al. (1996) found that the pre-treatment ratio of activity in the left OFC to the left anterior cingulate was one of the best predictors of behavior modification treatment outcome for OCD.

Pre-treatment ERP components have also been studied in relation to treatment outcome. Using a verbal auditory oddball paradigm, Morault et al. (1998) found that differences in pre-treatment components related to attentional processes predicted pharmacotherapy treatment outcome for OCD. In particular, OCD patients who responded to pharmacotherapy had reduced N2 amplitude and shorter N2 and P3 latencies, relative to those patients who did not respond to treatment. Morault et al. (1998) discuss these results in terms of neural indices of attentional and frontal executive mechanisms that may differentiate treatment responders from non-responders. This raises the possibility that pre-treatment ERP components such as the ERN could also predict treatment success. Considering the relationship between the ERN and the dysfunctional response monitoring associated with OCD symptomology, it stands to reason that ERN amplitude changes may actually be a better electrophysiological measure and predictor of treatment outcome.

At present, the specificity of the relationship between ERN enhancement and the OC 'trait' is unknown. Further research is needed that includes participants with other disorders within the anxiety spectrum, for example. Because we compared the high-OCI group to a low-OCI group, future studies might include a 'middle' OCI group that would be more characteristic of the population at large. Finally, given the absence of obvious performance consequences of ERN enhancement, the functional significance of the ERN in both clinical and normal control subjects merits additional attention.

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