Error-Related Brain Activity in Anxiety Disorders:

The Hyperactivity of the ERN

by

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Abstract

Abnormalities in the anterior cingulate cortex (ACC) have been reported in anxiety disorders.

The error-related negativity (ERN), a component of the event-related brain potential (ERP), has

been found to be increased in both adults and children with anxiety disorders, such as in people

with Obsessive-Compulsive Disorder (OCD). The purpose of this study was to assess the ERN

and behavioral correlates during an Eriksen flanker task in anxiety probands compared to other

groups. The dataset consisted of participants aged 8-17; 25 probands with a lifetime diagnosis of

OCD, 8 probands diagnosed with social, separation, or generalized anxiety disorder (SSGAD)

and 43 healthy controls. ERN during correct- and error-trials was larger in the SSGAD group

than the OCD group or controls.

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The study of mental disorders is one that has been of great interest in psychology but has

not yet been conquered. Anxiety disorders have come to be known as the most prevalent class of

mental disorders. Thayer and Lane (2000) asserted that anxiety disorders represented a failure to

either elect an adaptive response or to inhibit a maladaptive response given a situation. Anxiety

disorders, while being a diverse set of phenotypes, are alike in that they all involve excessive

negative effect typically in the form of fear and anxiety. There are many varieties and types

within the disorder; the most commonly known ones include social anxiety, separation anxiety,

generalized anxiety, obsessive-compulsive disorder (OCD), and a wide array of phobias.

One of the most recognized anxiety disorders is OCD, a disorder that is characterized by

recurrent intrusive thoughts, or obsessions, which result in repetitive and ritualistic behaviors,

also known as compulsions (Rosenberg & Keshavan, 1998). Obsessive-compulsive disorder is a

debilitating disorder for those who suffer from it. Due to their obsessions, people feel driven to

carry out their compulsive acts, which may take several hours a day to complete. Some

examples are washing their hands repeatedly or checking to see if their doors are locked

numerous times. These acts, which must be performed precisely and according to rules, are done

to reduce anxiety stemming from their obsessions.

This disorder very commonly emerges in childhood; as many as 80% of all cases of

anxiety have their onset in childhood and adolescence (Pauls et al., 1995). This is a serious

illness because in severe cases of anxiety, it is usually associated with other psychiatric

disorders. In the study by Hanna (1995), over 80% of the subjects were diagnosed with at least

one additional psychiatric disorder. These psychopathologies included lifetime depressive

disorder, other anxiety disorders, disruptive behavior disorders (such as attentional-deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder), and lifetime tic disorder. OCD, which is comorbid with anxiety, is produced in individuals with OCD due to their obsessions. In children with anxiety disorder, error signals in the brain are generated during the course of doing a flanker task. In control children, there is no appearance of these error signals (Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006). This is noteworthy since the error signals are typically observed in mid- to late- adolescence but they are shown in anxious children.

Despite its high prevalence and the attendant morbidity, the pathophysiology of anxiety disorders remains unclear with a plethora of theories and a lack of well-replicated findings. In the study by Rosenberg and Keshavan (1998), they utilize a series of neurobiological studies aimed at testing the hypothesis that neurodevelopmental abnormalities of ventral prefrontal-striatal circuits may be involved in and contribute to the etiology and presentation of the illness. Ventral prefrontal cortical (VPFC) abnormalities early in illness and the lack of association between illness duration and anterior cingulate volume suggest that VPFC abnormalities may have a neurodevelopmental basis in anxiety. The prefrontal cortex matures throughout childhood and adolescence up until the mid-twenties. The positive correlation between age and anterior cingulate volumes in controls, but not in anxious patients, further supports the neurodevelopmental model for anxiety.

Further research has been done using genetic and imaging strategies in anxiety for potential implications for treatment development. Disturbances in VPFC-striatal interactions are believed to be involved in causing anxiety. Damage to the VPFC circuits results in impairment in the ability to inhibit context-inappropriate emotional responses, leading to inappropriate

behaviors (Rosenberg & Keshavan, 1998). Volumetric abnormalities in VPFC-striatal-thalamic

circuitry may be associated with the clinical presentation of anxiety and represent a central

developmental neurobiological deficit in the illness. Hyperactivity in the VPFC is directly linked

to structures of the basal ganglia and thalamus in a parallel circuit that mediates the expression

and manifestation of anxiety symptoms (Rosenberg & Hanna, 2000).

There is also substantial evidence for a genetic contribution to the etiology of anxiety

disorder. Twins have a greater chance of having OCD if the other has it, and the likelihood is

even greater for monozygotic twins. Analyses have provided a heritability estimate of 47% for

obsessional symptoms. Due to the evidence, there have been studies which tried to identify

chromosomal regions which were likely to contain susceptibility alleles for early-onset OCD

(Hanna et al., 2002).

Regardless of whether there is a genetic susceptibility to this disorder, a consensus in

literature shows that there is a functional and neural basis behind it. There is a great deal of

research that illustrates hyperactivity in certain regions of the brain, which is associated with

symptoms of anxiety. Neuroimaging and electrophysiological studies have revealed an

overactivity of fronto-striatal brain regions including the orbitofrontal cortex, the caudate

nucleus, the thalamus, and most notably, the anterior cingulate cortex (ACC). These structures

are thought to make up a circuit in which connections form a positive feedback loop, thus

causing excessive activity to produce the symptoms of anxiety.

In individuals with anxiety, the error signals are larger and persist for a longer period of

time than in people without the disorder. As can be seen from the error signals, an individual

with anxiety feels that something is wrong and that action is needed to correct the problem;

therefore, the error signals contribute to nervousness, doubt, feeling of incompleteness, and

compulsive behavior. The system forms a positive feedback loop; the individual is unable to reduce the error signals so they repeat the actions (such as in OCD), which generate further error signals.

Gehring et al. (2000) explored the hypothesis that the excessive neural activity involved in OCD symptomatology represents "hyperactive error signals". According to this notion, the brain maintains internal standards that represent desired internal and environmental states. It compares these standards with environmental stimuli, internal stimuli (such as thoughts or feelings), and actions. When conflicts are detected, this comparator system generates an error signal which alerts cognitive, motoric, and affective systems of the need to correct this problem. These systems then determine whether and how to act, depending on the level of error signal and the motivational significance of the alerting information. This study was the first to examine electrophysiological indicators of performance monitoring (PM) in OCD patients. Enhanced error-related negativity (ERN) amplitudes were found in OCD patients compared with controls in a Stroop task. The correlation between ERN amplitude and symptom severity supports the view that OCD is related to excessive performance monitoring.

Performance monitoring has been examined using event-related potentials (ERPs). The ERN, a component of the ERP, has been identified as specifically related to incorrect response execution. It is characterized by a fronto-central negative deflection, arising shortly (< 100 ms) after the execution of an incorrect response and is generated in the ACC (Gehring, Goss, Coles, Meyer, & Donchin, 1993). Several studies reported a smaller negative ERP component following a correct response, the correct-related negativity (CRN, Ford, 1999); however, the presence of a negativity after correct responses is controversial since the CRN is not as consistently observed as the ERN (Gehring et al., 1993).

Endrass et al. (2008) examined whether performance monitoring is altered in anxious patients. Earlier studies illustrated that errors were associated with increased ERN amplitudes in patients with OCD. Endrass and colleagues were also interested in whether the CRN amplitudes are also altered in patients. OCD patients are concerned with their errors but also with the correctness of their actions so it was hypothesized that CRN amplitudes should also be enhanced in OCD patients. The analysis of response-locked correct trials revealed substantial CRN amplitudes in both groups. Stimulus-locked data of correct trials varying in response time showed a second negative deflection which appears synchronously with the onset of the correct response. This clearly indicates that the CRN is not a mere stimulus artifact since the patients showed enhanced CRN amplitudes compared with controls. The significance of OCD patients showing an amplitude enhancement for response-related negativities that is not specific to error processing is that it illustrates that response monitoring might be overactive in anxiety.

The ERN is thought to reflect part of an action-monitoring system; some have argued that it reflects error detection (Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996) while others have argued that it detects response conflict (Carter et al., 1998). It has been suggested that errors may represent a form of response conflict such that error processing and conflict detection may be one and the same process (Carter et al., 1998). Fitzgerald et al. (2005) tried to better localize the source of error-detection and conflict processing differences between OCD and controls. Based on the ERN data and functional neuroimaging evidence for hyperactivity of the ACC in OCD, they hypothesized that ACC activation during error commission would be greater in OCD patients compared to controls. An interference task was used, which elicited errors and conflict between competing response tendencies, to test whether conflict processing alone

elicited hyperactivity of the ACC. As predicted, greater ACC activation was found in OCD

patients, but this difference was localized to the rostral ACC (rACC).

Hajak and Simons (2002) determined whether the enhanced error-related activity found

in OCD patients could also be found in a non-clinical population with obsessive compulsive

characteristics as assessed by the Obsessive-Compulsive Inventory. Results demonstrated that

high obsessive-compulsive students show larger ERN and CRN amplitudes than subjects with

low symptom scores. This result indicates that performance monitoring is overactive in

obsessional subjects even though they respond correctly.

These findings are consistent with the hypothesis that ACC involvement in OCD may be

related to functional abnormalities in the processing of errors or perceived errors. Increased

conflict-related activity during correct task performance, as well as during errors, is associated

with a critical self-evaluation of performance in patients with OCD, leading to an inappropriate

need for correction and repetitive action. Pitman (1987) suggested that false errors generated by

an action-monitoring system explain why OCD patients experience the constant need to correct

their actions. Pitman's cybernetic model indicates that a disturbance in behavioral output does

not necessarily have to originate in the output system; a healthy output system may generate

repetitive stereotypical behavior as long as it is driven by persistent high error signal.

Clinically, patients with OCD are impaired in the natural inhibition of repetitive thoughts

and behaviors, and a selective disturbance in the ability to suppress responses to irrelevant

stimuli has been demonstrated. It has been hypothesized that a disturbance in the orbital

prefrontal cortex and its ventral striatal target fields may have a disinhibitory effect that could

underlie the inhibitory deficit suggested by the symptomology of OCD. Rosenberg, Dick,

O'Hearn, and Sweeney (1997) evaluated response-inhibition and delayed-response functions to

determine whether there is a selective impairment in inhibitory controls of neurobehavioral

processes in OCD. By studying medication-free adults, they were able to examine the cognitive

skills subserved by the prefrontal cortex without the potential confounds of medications. Their

results provide important new data about disturbances in prefrontral neurocognitve functions in

OCD by demonstrating that performance in response-inhibition tasks appears to be selectively

impaired relative to that observed during spatial delayed-response tasks.

Neuropsychological examinations have suggested that deficiencies in executive control

adjustment might underlie the disorganized and inflexible behavior that is seen in patients with

OCD. Patients afflicted with OCD typically exhibit an 'obsessional slowness' in many cognitive

tasks, and although some studies have reported normal conflict costs in these patients, others

have found greater interference effects in their reaction time. For instance, during Stroop tasks,

the behavioral effects of conflict are not just limited to the current trial, but they also affect

performance in the subsequent trials. This 'obsessional slowness', sometimes noted as post-error

slowing, occurs since OCD patients focus on the fact that they have made an error. Patients with

OCD have been found to have heightened ACC-associated ERN in ERP studies and ACC

hyperactivity in neuroimaging studies. A failure of OCD patients to accomplish task switching,

however, has been associated with hypoactivation of the ACC, the prefrontal cortex (PFC), the

parietal cortex, and the striatum. The cognitive inflexibility that is exhibited by OCD patients

could be due to a general impairment in inhibitory functioning as well as to an abnormal

monitoring system (Mansouri, Tanaka, & Buckley, 2009).

In this study, we examined the ERN, response time, and accuracy during a flanker task in

76 child participants. The study observed whether the ERN was more significant in children

with anxiety disorders. We hypothesized that the ERN amplitude would be significantly higher in OCD and SSGAD patients in comparison to controls.

Method

Participants

Through the help of the University of Michigan Psychiatry department, OCD and non-OCD anxious probands were recruited. Healthy controls were also recruited, although many of our healthy controls found out about our project and inquired to be a part of it. Our sample included 25 probands with a lifetime diagnosis of OCD, 8 probands diagnosed with social, separation, or generalized anxiety disorder (SSGAD) and 43 healthy controls, all between the ages of 8 to 17. For both the OCD and SSGAD group, a portion of the participants were medicated, while the others were not yet treated for their disorder. The participants were given a monetary compensation for partaking in the experiment.

Task

The flanker task was administered using Presentation software to control the presentation and timing of all stimuli, the determination of response accuracy, and the measurement of reaction times. Throughout the task, the participants were shown 4 different congruent (e.g., →→→→→, ←←←←←) or incongruent (→→←→→, ←←→←←) stimuli on a 15-inch computer monitor using a black background. A fixation mark (+) was presented prior to each stimuli. The participants were instructed to press the right or left keyboard button in response to the direction of the central arrow.

Procedure

After a brief description of the experiment, EEG/EOG sensor electrodes were attached, and the participant was given detailed task instructions. Each participant was seated directly in

front of the computer monitor and given a practice block. The participant was told to press the

left key if the central arrow pointed left and press the right key if they central arrow pointed

right. These 4 different conditions were counter-balanced across participants. The participants

were told to place equal importance on speed and accuracy in their responses. Following the

practice block (which contained 40 trials), the participants received 8 blocks of 64 trials (512

total trials). After each block, the participant was checked upon and told whether or not to speed

up or slow down since the experiment necessitated the participants to make 6 to 12 errors per

block. The fixation mark, that was presented prior to the stimuli, lasted for 1500 ms. The

stimuli, which appeared immediately afterwards, remained on the screen for 250 ms.

Psychophysiological Recording, Data Reduction and Analysis

The electroencephalogram (EEG) was recorded using an electrocap and the BioSemi

ActiveTwo system. Recordings were taken from 64 locations positioned all over the head

according to the 10/20 system (see American Society of Electrophysiology reference in Gehring

& Fencsik, 2001). During the recording, all activity was referenced to Cz. The electro-

oculogram (EOG) generated from blinks and vertical eye movements was recorded using face

electrodes placed approximately 1 cm above, below, and on the outer corners of the participant's

eyes in addition to the electrodes placed on the mastoids. The EEG for each trial was corrected

for EOG and muscle artifacts 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 many of the channels were very noisy. Finally, the EEG for each trial

was time-locked to its respective reaction time and averaged across trials to yield correct- and

error-trial ERPs for each electrode site. To quantify the ERN, each data point after response

onset was subtracted from a baseline to the average activity. The ERN was then defined as the

most negative peak occurring in a window from -150 to 50 ms post-response. The ERN and performance measures were assessed using Matlab software and statistically evaluated using the Statistica program.

Results

The response-locked average waveforms for error and correct trials are presented in Figures 1 and 2. Participants tended to have faster reaction times for errors than for correct responses; however, there was no between-group effect and no interaction between trial and group. Although OCD and SSGAD probands tended to make more mistakes, this accuracy difference was not significant for either the percent correct or number of errors performance measure. It is important to note that the number of rejected trials varied between participants. Figure 1 illustrates that when participants made errors, there was a sharp negative deflection that peaked around 50 ms immediately after the response, primarily at the fronto-central recording site. The ERN was largest for the anxiety group, while the ERNs of the control and OCD group were much smaller in size.

Figure 2 illustrates that during correct trials there was also an occurrence of a negative deflection, although the CRN was smaller for all three groups. The CRN of the anxiety group, however, was still much larger than that of the other two groups. A repeated measures analysis of variance (ANOVA) yielded a statistically significant group effect, $F(2,56) = 4.45$, $p<0.05$; the ERN and CRN were larger among the SSGAD participants than the OCD participants and controls (Table 1). Post-hoc analysis (Table 2) shows the difference between the SSGAD group and controls in the ERN and CRN and also a difference between the SSGAD group and OCD group. The correct- and error-trials for the SSGAD group were significantly different from the

correct- and error-trials of the OCD group and controls while the correct- and error-trials of the

OCD group and controls were not significantly different from each other.

Discussion

The results of our analyses indicate that the SSGAD group differs from the OCD group

and controls in electrophysiological measures associated with error monitoring. As predicted,

we found that the SSGAD patients had a significantly larger error-related activity than both other

groups. Interestingly, however, we did not find that the OCD group had an exaggerated ERN

like the SSGAD group. It was even more surprising that their ERN looked very similar to the

controls. These results may indicate that within the anxiety spectrum disorders, an enhanced

ERN may not be specific to OCD.

In addition, not only did the SSGAD group have an amplified ERN, they also showed a

large negative peak during correct trials. This CRN, similar to the ERN, was considerably bigger

in the SSGAD group than in the OCD group and controls. Although the CRN in the OCD group

was slightly larger than in controls, it was more comparable to those of the controls than to those

of the SSGAD group. Like the previous study done by Hajcak et al. (2003), we did not find an

interaction between group (SSGAD, OCD, controls) and trial type (correct vs. error), suggesting

that the enhanced error-related brain activity in the SSGAD group is not specific to errors.

Rather, the data indicate that enhanced error-related brain activity is associated with both correct-

and error-trials.

Coles, Scheffers, and Holroyd (2001) have proposed that this correct response negativity

could be the result of stimulus artifact, partial error-processing on correct trials with sub-

threshold incorrect activity, or violations of implicit temporal parameters that define a correct

response. While the explanation of information-processing mechanisms that underlie both the

ERN and CRN is still incomplete, it is nevertheless true that both the ERN and CRN is enhanced in SSGAD, relative to OCD and controls. These data provide some support for the similarity of the ERN and CRN.

There are many possible reasons that the ERN and CRN are amplified in the SSGAD group but not in the OCD group. Some possible explanations are that the groups were not age matched, gender matched, or matched for symptom severity. The SSGAD group may have had more severe anxiety symptoms, which may have led to the larger negativities. Also, some of the patients have already been treated with medication, which may have altered their ERN and CRN. One study, however, demonstrated that that did not have an effect at all. Hajcak et al. (2008) conducted a treatment study showing that anxious children who were treated showed the same size ERN after the treatment as they did before. Since medicated and non-medicated children showed the same size ERN, we expected that the kids with OCD would show larger ERNs than controls. Our negative results suggest that further research is needed to find out why the ERN was larger in their study but not in the present study.

Past ERN studies on OCD illustrated an enhanced ERN in OCD probands, but the OCD probands may have had other anxiety disorders on top of OCD. There is added variability since there are many types of OCD and non-OCD anxiety disorders, which make these studies even more difficult to replicate. Since there are so many sub-types of anxiety, as well as OCD, future ERP studies should incorporate different combinations of these subtypes. For future studies, these factors should definitely be taken into consideration and groups should be matched better for age, gender, and symptom severity. In addition, since these studies imply that the ERN and CRN are different depending on the type of anxiety disorder, future studies can look into the ERN and CRN in other anxiety spectrum disorders, such as tic disorder or Trichotillomania. If

significant differences are found, there may be important clinical implications. For instance, if there are differences in negativities in the different types of anxiety disorders, a spectrum could be mapped out, which would imply that certain types may have more severe symptoms than others.

As a result, this present study fits well into a growing body of research showing that anxiety disorders are associated with neural hyperactivity of the ACC (Hajcak, McDonald & Simons, 2003). The present study adds to the literature by establishing that anxiety effects are clearly present in children, which is noteworthy since the majority of the research done in this field has focused on adults. The present study also demonstrates that within the anxiety spectrum disorders, enhanced error-related brain activity may not be specific to just one type of anxiety disorder. Rather, the enhanced ERN and CRN found previously in OCD, and now in SSGAD, suggest that there are many factors, such as symptom severity and personality dimensions, that may best explain neural differences related to response monitoring.

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Table 1

Repeated Measures Analysis of Variance (ANOVA)

Effect	SS	df	MS	F	p
Intercept	201.752	1	201.7523	6.80586	0.011630
Group	263.953	2	131.9767	4.45207	0.016056
Error	1660.059	56	29.6439		
Trial (correct vs. error)	555.421	1	555.4214	33.04884	0.000000
Trial*group	s16.288	2	8.1438	0.48458	0.618517
Error	941.140	56	16.8061		

Table 2

Newman-Keuls Test/Post-Hoc Analysis

Group	Trial	{1} 2.5035	{2} -3.294	{3} 2.2822	{4} -1.906	{5} -1.781	{6} -6.920
OCD	Correct		*0.003145*	0.901681	0.070877	*0.047516*	*0.000129*
OCD	Error	*0.003145*		*0.012337*	0.438857	0.674543	*0.044933*
CON	Correct	0.901681	*0.012337*		*0.021233*	*0.025030*	*0.000125*
CON	Error	0.070877	0.438857	*0.021233*		0.944339	*0.016388*
ANX	Correct	*0.047516*	0.674543	*0.025030*	0.944339		*0.007090*
ANX	Error	*0.000129*	*0.044933*	*0.000125*	*0.016388*	*0.007090*	

Note. The numbers represent p-values for each pairwise comparison. The red numbers are statistically significant.
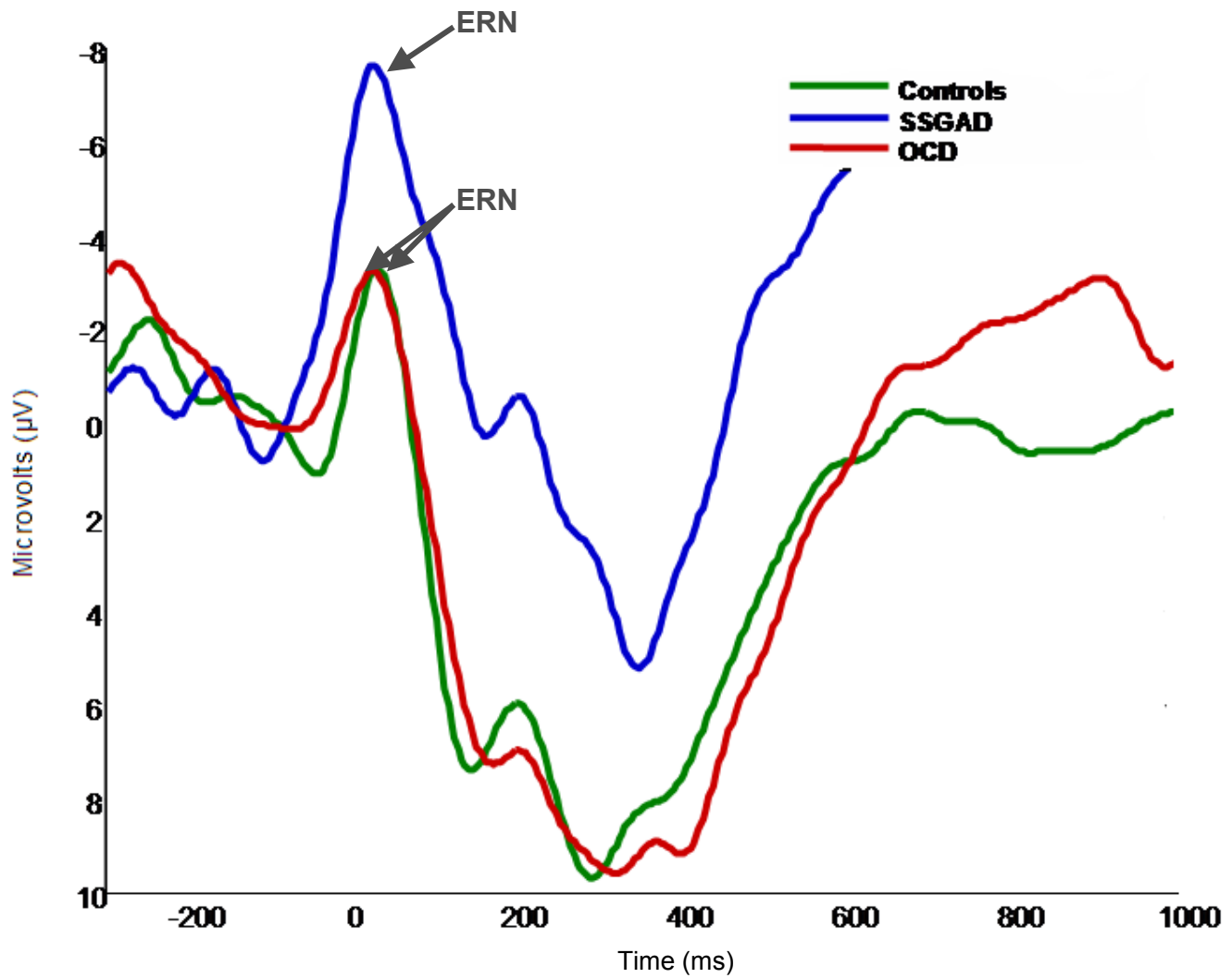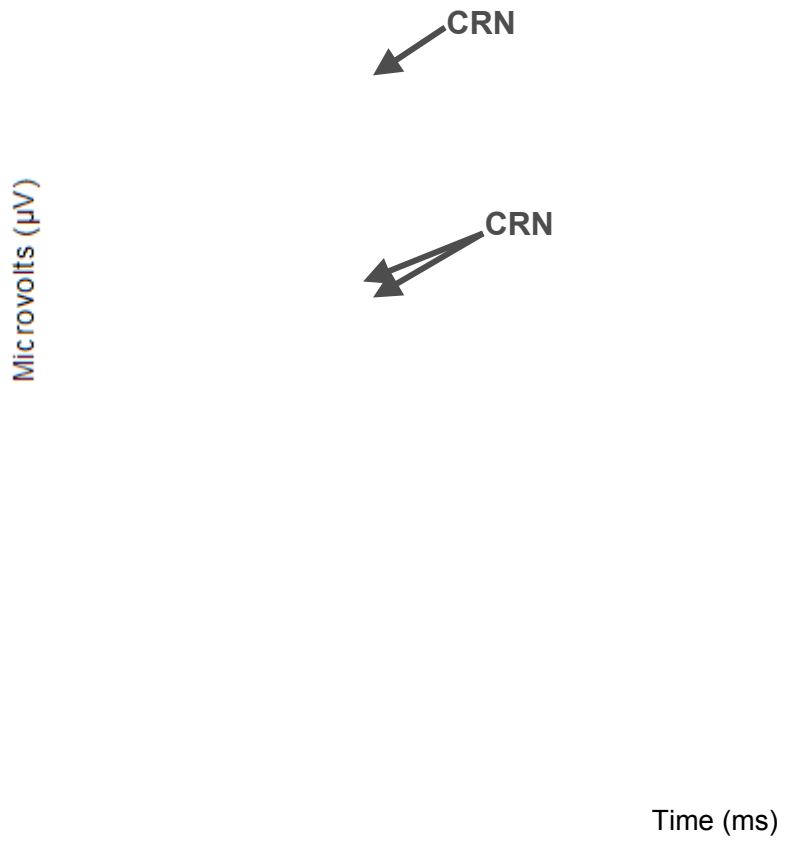
Figure 1. These are the ERP waveforms during error trials for all three groups. SSGAD is the social, separation, or generalized anxiety group and OCD is the obsessive-compulsive disorder group. Time 0 represents when the ERN peak is about to occur.
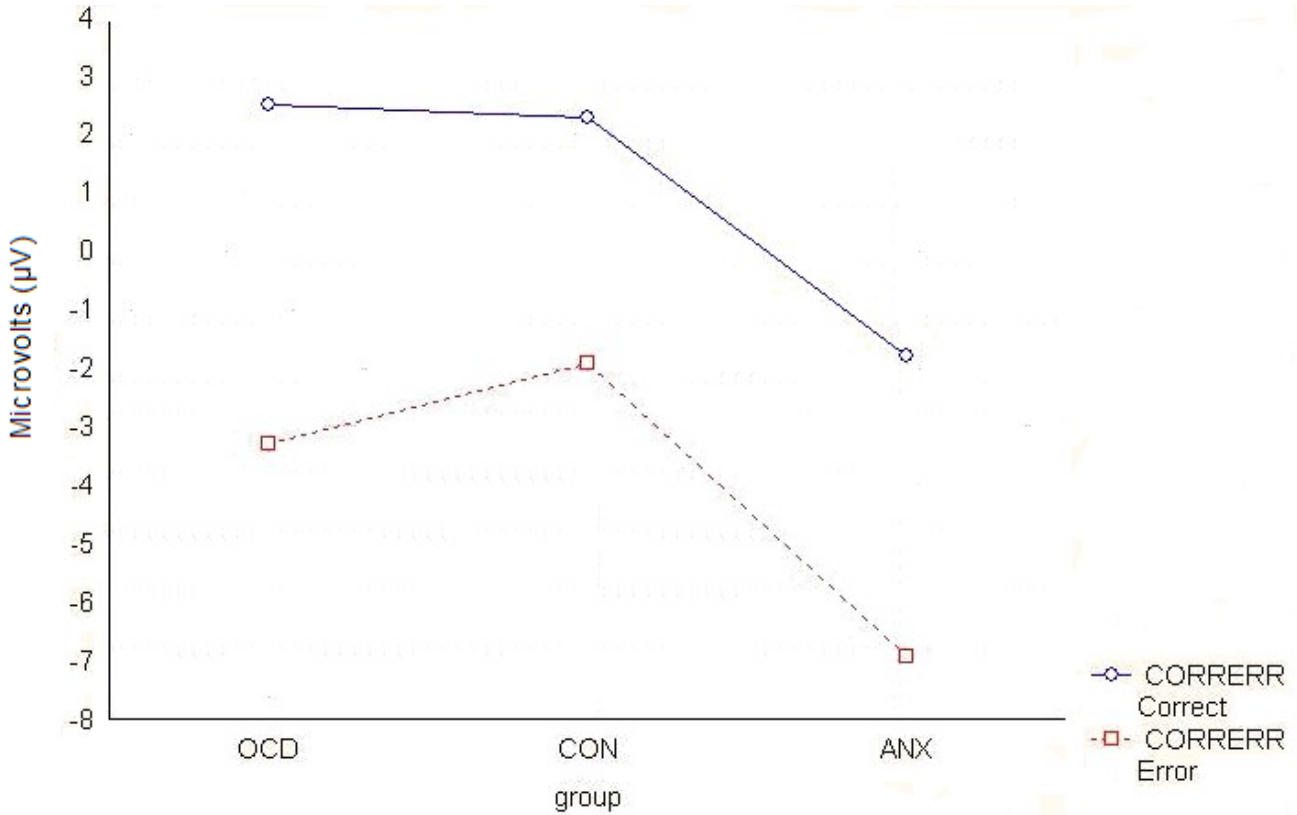
Microvolts (µV)

CRN

CRN

Time (ms)

Figure 2. These are the ERP waveforms during correct trials for all three groups. Time 0 represents when the CRN peak is about to occur.

Figure 3. The non-OCD (SSGAD) group has a significant ERN and CRN compared to the OCD group and control group. The non-OCD (SSGAD) group has the largest negative deflection for correct- and error-trials. The OCD group has a slightly larger negative deflection for error trials compared to controls, but the negative deflection for correct trials is almost the same as the controls. Larger ERN amplitudes are associated with larger negative (downward) values.