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An Event-Related Potential Study of Attention Deficits in Posttraumatic Stress Disorder During Auditory and Visual Go/NoGo Continuous Performance Tasks

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Abstract

Post-traumatic Stress Disorder (PTSD) is characterized by disturbances in attention, such as increased arousal and hypervigilance. This study examined the event-related potential (ERP) P3 component to target detection (Go), response inhibition (NoGo) and irrelevant nontarget stimuli during auditory and visual A–X continuous performance tasks. NoGo N2 amplitude effects were also analyzed. Participants were 23 Vietnam veterans with PTSD and 13 civilian controls. No group differences were present for N2 or P3 amplitude to Go and NoGo stimuli. The PTSD group, however, had shorter P3 latency to Go and longer P3 latency to NoGo stimuli than controls, regardless of modality. The PTSD group also had greater frontal P3 amplitude to irrelevant nontarget stimuli than controls. Significant P3 amplitude and latency findings were associated with higher hyperarousal and reexperiencing scores from the Clinician-Administered PTSD Scale. The findings suggest that attentional problems in PTSD are related to slowed central processing when response inhibition is required, and to an impaired ability to screen irrelevant information. This study provides further evidence that the attentional impairments in PTSD are not confined to trauma-related stimuli. Heightened arousal appears to enhance the attentional dysregulation seen in PTSD.

Introduction

The characteristic features of posttraumatic stress disorder (PTSD) include re-experiencing of the traumatic event, avoidance of stimuli associated with the trauma, and persistent symptoms of increased arousal, including difficulty concentrating, hypervigilance, exaggerated startle response, and heightened physiological reactivity to cues that symbolize or resemble the event (American Psychiatric Association, 1994). In addition to the above sequelae, there is evidence that PTSD is associated with a dysfunction in certain biological systems related to information processing and attention. Numerous neuropsychological (Buckley et al., 2000; Murburg et al., 1994; Vasterling et al., 1998; Vasterling et al., 2002), electrophysiological (see for review Karl

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et al., 2006) and imaging studies (Bryant et al., 2005) have provided support for the involvement of neurobiological systems in the attentional dysregulation seen in PTSD.

The now well established neurobiological model of PTSD proposes that a dysregulation occurs in the temporal amygdaloid complex and its interrelation with the prefrontal cortex and the locus coeruleus (Bremner, 2005; Charney et al., 1994; Kimble & Kaufman, 2004; Kolb, 1987; McFall et al., 1990; Vermetten & Bremner, 2002). Both visual and auditory fear-producing stimuli follow pathways to the thalamus, which, in turn, sends signals to the amygdala and locus coeruleus. Frontal projections from the amygdala are sent to the anterior cingulate (ACC) and dorsolateral prefrontal cortex, which send feedback to both the amygdala and hypothalamus (see for review Gunnar & Quevedo, 2007). A dysregulation of this system produces a state of hypersensitivity in which internal and external stimuli lead to heightened arousal (Kolb, 1987). Behavioral and cognitive manifestations can persist for years after the trauma and include attentional disturbances, hyperarousal, concentration difficulties, exaggerated startle response, hypervigilance, autonomic hyperactivity, and executive dysfunction (McFarlane et al., 1993; Sutker et al., 1995; van der Kolk, 1987; Wolfe & Charney, 1991).

Attentional problems in PTSD may be activated either by heightened arousal to threat stimuli or by the requirement of sustained attention (Kimble et al., 2000). Imaging studies have shown that affectively laden trauma stimuli activate brain areas associated with the stress response in individuals with PTSD and not in controls (see for review Liberzon & Martis, 2006). It has also been demonstrated that attention tasks using neutral cognitive stimuli show differences between PTSD and control participants (Bryant et al., 2005; see for review Karl et al., 2006).

Event-related potentials (ERPs) have been employed in a number of studies to examine attention and information processing of both affectively laden and neutral stimuli in PTSD. In particular, the P300 (P3) complex of the ERP, consisting of the P3a and P3b components, has been the most widely examined in studies of PTSD because it provides temporal information about attentional processes such as the ability to detect a target, the salience of the target to the participant, and the response to novel or distractor stimuli (Johnson, 1986; Polich, 2003; Polich & Kok, 1995; Näätänen, 1990). Generally, these studies employed an oddball paradigm with either two stimuli (infrequent target tones vs. frequent tones) or three stimuli (infrequent target tones, frequent tones, and infrequent distractor tones). Target, frequent, and distractor tones are each presented at a designated frequency (e.g., 2000, 1000, and 500 Hz, respectively). The most common finding has been that participants with PTSD show a reduction in the expected centro-parietal amplitude enhancement to target stimuli (identified as P3b), such that the P3b is present, but attenuated in amplitude in PTSD participants relative to controls (Charles et al., 1995; McFarlane, et al., 1993; Paige, et al., 1990; Metzger, Orr, Lasko, & Pitman, 1997a; Metzger et al., 1997b). The P3b amplitude was found to be less attenuated in PTSD participants when medications and panic disorder were considered (Metzger et al., 1997a), and was not reduced in a female sample of veterans with PTSD (Metzger et al., 2002).

A variation of the three-stimulus oddball task includes novel non-repeating sounds (whistles, chirps, etc.) or trauma-related stimuli (non-repeating words, pictures) as the third stimulus. Non-repeating novel stimuli tend to elicit a P3a, which is distinguished from P3b by its fronto-central maximum amplitude distribution. Näätänen (1990) proposed that the P3a is a preattentive shift in attention upon detection of sensory changes in the environment, while the P3b reflects attention to infrequent, relevant target stimuli. P3a has not been studied as extensively as P3b in PTSD, although it may be more directly linked to frontal lobe structures than the P3b (Polich, 2003). Kimble et al. (2000) found that combat veterans with PTSD had a greater frontal P3a than combat veterans without PTSD to non-repeating novel stimuli in a three stimulus oddball task, but no frontal P3a enhancement to repeating distractors in a second

oddball task. Another study used a visual oddball task with words rather than tones, and found that the PTSD group produced an enhanced frontal P3a to combat-related words as well as an attenuated parietal P3b to neutral target words (Stanford et al., 2001). Contrary to most P3 findings, Neylan et al. (2003) reported that combat veterans with PTSD, compared to combat veterans without PTSD, did not show an enhancement of P3a to novel non-trauma related distractors or a reduction of P3b to target stimuli in a three-stimulus oddball paradigm. Conflicting findings may be related to other factors that could account for the enhancement of P3a amplitude observed in PTSD, such as the level of hyperarousal or panic disorder symptoms. Although there is some evidence that panic disorder affects P3a (e.g., Metzger et al., 1997b; Clark et al., 1996), hyperarousal effects have not been studied fully in PTSD.

The majority of P3 studies of PTSD presented stimuli in the auditory modality. It is yet unclear the extent to which modality of stimulus presentation is significant with respect to the activation of brain systems involved in PTSD, particularly to nonthreatening neutral stimuli. Several P3 studies used visually presented trauma-related distractors (Attias et al., 1996a; Attias et al., 1996b; Bleich et al., 1996; Stanford et al., 2001), but there are no studies that used only neutral visual stimuli. Only one study (Neylan et al., 2003) presented a three stimulus novelty oddball paradigm in both the auditory and visual modalities for the purpose of investigating the reliability of P3 recordings in PTSD across two different time points.

In addition to the oddball paradigms, continuous performance task (CPT) paradigms are also used to study attentional processes. The CPT paradigms can be more complex than the oddball paradigms and can require multiple cognitive operations. Various behavioral studies used CPT paradigms to investigate sustained attention in PTSD and reported deficits on measures of sustained attention such as difficulty inhibiting inaccurate responses, filtering irrelevant information, and greater errors of omission and/or commission than controls (Vasterling et al., 1998; Vasterling et al., 2002; Zimering et al., 1993). No studies, to date, have measured ERPs during CPT task performance in PTSD.

In a previous study of normal adults in our laboratory (Tekok-Kilic et al., 2001), ERPs were recorded during an A-X version of a CPT (Halpern et al., 1988) to examine target detection (Go) and response inhibition (NoGo) in both the visual and auditory modalities. In the A-X version of the CPT, the task requires participants to respond to the X only when it follows an A, and inhibit their response when any other letter follows an A. Tekok-Kilic et al. (2001) found that detection of the target X produced the expected P3b amplitude distribution, with the greatest P3 amplitude at the parietal scalp site. The NoGo inhibitory stimuli, on the other hand, produced a shift in amplitude to a more fronto-central response similar to the P3a distribution. These findings were consistent across both the visual and auditory modalities, suggesting that the different neural systems required for the cognitive operations of target detection and response inhibition are not dependent on the modality of stimulus presentation.

A number of studies have used various Go/NoGo paradigms, in which participants respond to a designated stimulus (Go) and withhold responding to another stimulus (NoGo), in order to examine the effect of these response types on the N2 and/or P3 components of the ERP. The consistent findings have been that NoGo stimuli produce a greater fronto-central amplitude P3 and a greater negativity of N2 relative to Go stimuli. These findings are thought to reflect inhibitory processes related to the frontal lobes (Bekker et al., 2005; Bokura et al., 2001; Bruin & Wijers, 2002; Falkenstein et al., 1999; Freitas et al., 2007; Kopp et al., 1996; Pfefferbaum & Ford, 1988). Some studies have examined the degree to which the NoGo N2 and P3 components reflect inhibitory processes, as well as whether or not the two components reflect different aspects of the response control process (Bekker et al., 2004; Bruin et al., 2001; Verleger et al., 2006). Verleger et al. (2006) showed that the Go/NoGo effect was enhanced contralaterally to the left or right motor response from the preceding Go stimulus in a stop-

signal task, and proposed that the NoGo effect may be related to a reduction in motor activation from the Go response, and not necessarily inhibition. Other studies that used a CPT paradigm, in which the NoGo stimulus is not preceded by a motor response, have demonstrated the anterior distribution of the NoGo P3 (Bekker et al., 2004; Bokura et al., 2001; Tekok-Kilic et al., 2001), with localization of the NoGo N2 and P3 to frontal areas involved in inhibitory control (Bokura et al., 2001).

Although the effects of the motor response in Go/NoGo paradigms cannot be ruled out entirely, both ERP and fMRI studies support a general consensus that there is involvement of frontocortical systems, including the anterior cingulate cortex (ACC), in the anteriorization of the NoGo response (Bokura et al., 2001; Carter et al., 1998; van Veen and Carter, 2002). Further, the N2 and P3 components appear to reflect different aspects of response control, with the NoGo N2 being more related to conflict monitoring and the NoGo P3 to response inhibition (Bekker et al., 2004; Bruin et al., 2001; Donkers & van Boxtel, 2004). ACC activation has been shown to occur more as a result of response conflict (e.g. to respond or not respond) than merely as a result of task-related attentional control (see for review van Veen and Carter, 2002).

The ACC has also been implicated in functional imaging studies of PTSD. Decreased activation of the ACC has been shown to occur to threat stimuli in PTSD compared to controls (Bremner et al., 1999; Britton, et al., 2005; Lanius et al., 2001; Shin et al., 2001; Yang et al., 2004), and increased activation of the ACC to salient non-threatening information in PTSD in a three-stimulus oddball paradigm (Bryant et al., 2005). Thus, it is likely that, in addition to target stimuli, NoGo stimuli would produce differences in PTSD compared to non-PTSD controls.

In light of previous studies, it is now known that attentional deficits are present in PTSD to non-threatening neutral stimuli that are presented primarily in the auditory modality. A majority of the ERP studies had control groups that were trauma-exposed without PTSD, suggesting that it is the diagnosis of PTSD, and not trauma exposure alone, that produces these deficits. The goal of the current study was to use ERPs with parallel auditory and visual A-X CPT paradigms to: (1) examine ERP and behavioral performance differences between PTSD and controls under sustained attention conditions that incorporated target detection (Go), response inhibition (NoGo), and screening of irrelevant stimuli; (2) examine the possible effects of modality on these cognitive processes; and (3) relate ERP measures of attention in PTSD to behavioral measures of hyperarousal.

Methods and Materials

Participants

Twenty three adult male Vietnam combat veterans meeting the DSM-IV diagnostic criteria for PTSD and thirteen non trauma-exposed civilian controls were tested in all domains of the study. The groups were matched for sex, years of education, and estimated IQ (see Table 1). The age range for participation was 33 to 52 years. Vietnam veterans with PTSD were recruited from the Buffalo and Batavia VA Medical Centers. Controls were recruited from the community through advertisements at the Buffalo VA and in local newspapers. Participants were treated in accordance with the "Ethical Principles of Psychologists," signed an informed consent, and were paid for their participation.

Volunteers were initially screened for selection criteria through a phone interview. Those individuals who passed the phone screen were scheduled for a clinical assessment. Individuals were excluded from the study, based on the phone screen and/or clinical assessment, if they had a history or current presence of traumatic head injury, neurological disorder, cerebrovascular disease, major illness affecting cognition, learning disability, dementia, or hearing loss. PTSD participants could not have alcohol or substance abuse or dependence

within six months of testing. Axis I disorders that were allowed, other than PTSD, were panic disorder (with or without agoraphobia), specific phobia, dysthymia, and major depression in the mild to moderate range. Controls were excluded if they had previous or current alcohol or substance abuse or dependence, were taking psychotropic medications, or had an axis I diagnosis. All participants who met selection criteria following the clinical assessment continued in the study and underwent neuropsychological and electrophysiological testing.

Procedures

Clinical Assessment—The diagnosis of PTSD for the Vietnam combat veterans was determined by a psychiatrist or clinical psychologist using the Clinician-Administered PTSD Scale (CAPS-DX; Blake et al., 1995). The CAPS-DX provides frequency and intensity ratings of current and lifetime symptoms of PTSD and parallels diagnostic criteria specified in the DSM-IV. Vietnam combat veterans who met the criteria for PTSD were tested in the remaining phases of the study. The Davidson Trauma Scale (Davidson, 1996) and Life Events Checklist (Blake et al., 1995; Gray et al., 2004) were administered to control participants to rule out PTSD. The control participants were also given the Hyperarousal Symptoms section of the CAPS-DX to obtain frequency and intensity scores to compare with the PTSD participants. The Structured Clinical Interview for the DSM-IV (SCID – IV research version 2.0; First, Spitzer, Gibbon, & Williams, 1996), excluding the PTSD section, was administered to all participants to assess for Axis I disorders. The SCID-IV also was used to obtain detailed and quantifiable measures of current and past alcohol and drug use. In addition, all participants completed the Beck Depression Inventory (BDI; Beck, 1987). Medications were carefully documented for all PTSD participants. Table 2 presents a summary of medications and psychiatric diagnoses for the PTSD group.

Neuropsychological Testing: A neuropsychological test battery similar to that used by Sutker et al. (1995) was administered to assess three cognitive domains: (1) attention, (2) learning and memory, and (3) executive functions. In addition, premorbid verbal IQ was estimated using the North American Adult Reading Test (NAART; Blair & Spreen, 1989).

Electrophysiological Procedures: To verify that the participants could hear within the normal range, a brief pure tone audiometric hearing screening was conducted before participants completed the ERP testing. Electrophysiological data were obtained under three experimental paradigms: Sustained auditory attention (auditory A–X CPT), sustained visual attention (visual A–X CPT), and auditory focused attention. Only data for the first two paradigms are presented in this paper. The auditory and visual paradigms were counterbalanced for order of presentation. Level of arousal was monitored throughout the recording sessions via EEG recording and video monitoring of the participant's face.

Auditory and Visual A–X CPT Paradigms: The auditory CPT consisted of 11 letters (A,B,C,D,E,F,G,H,I,L,X) presented in a quasi-random order with a total of 400 letters for a duration of 12.6 minutes. The auditory letter stimuli were recorded using a female voice and presented using a NeuroScan computerized stimulus delivery/data acquisition system. Each letter was 400 msec in duration, with a 1500 msec interstimulus interval. The auditory CPT was presented to the participant over earphones at a sound level intensity of 70 dB. Participants were instructed to press two buttons with the thumbs of their left and right hands simultaneously, and as quickly and accurately as possible, to the letter "X" only when it followed an "A" (A–X). Participants were told to withhold responding to any letter other than "X" that followed an "A" (e.g., A–G). The letter "X" also appeared intermittently throughout the CPT without being preceded by an "A" (X-only). There were 40 target X (Go stimuli), 40 NoGo stimuli, and 40 "X-only" stimuli located randomly throughout the series of 400 letters. All of the task-irrelevant letters were considered "Nontarget" stimuli.

The letter stimuli in the visual A–X CPT were presented in the same order, and with the same duration and interstimulus interval as the auditory paradigm. Stimuli were 2.8 by 2.8 cm white capital letters on a black background presented at eye level in the center of a computer screen. The screen was located 60 cm from the bridge of the participant's nose. The same task instructions as the auditory CPT were given for the visual CPT.

Behavioral Measures: Mean reaction times (RT) for correct target detections were obtained for both the auditory and visual tasks. Task accuracy scores consisted of correct responses, errors of omission, and errors of commission.

Electrophysiological Measures: Electrophysiological recordings were obtained during the visual and auditory A–X CPTs while participants sat in a sound attenuated room with normal illumination. Gold plated electrodes were placed at 12 scalp sites (Fz, Cz, Pz, Oz, F3, C3, T3, P3, F4, C4, T4, P4) according to the International 10–20 system of electrode placement. Electrodes were referenced to linked ears, and a ground was located on the forehead. Electrooculographic artifact was monitored from bipolar electrodes at supra- and infraorbital sites of the left eye for vertical eye movements, and at the outer canthi of both eyes for horizontal eye movements. To reduce eye blink artifact, participants were instructed to try not to blink while the letters were presented. Electrode impedance was kept below 5 kilo-ohms.

A NeuroScan electrophysiological system was used for data collection and processing. The EEG data were filtered during acquisition with a bandpass of 0.1 to 100 Hz using Grass 7P511 amplifiers, and digitized at 250 Hz and stored on the NeuroScan system. The data were segmented into 1200 msec epochs (trials) and digitally filtered with a 0.1 to 25 Hz bandpass at 24 dB/oct. The data were then subjected to artifact rejection procedures. Trials contaminated by excessive artifact in any of the EEG channels were rejected if the artifact exceeded $\pm 200 \mu\text{V}$. Trials with artifact that exceeded $\pm 100 \mu\text{V}$ in the horizontal eye movement channels were rejected. Vertical eye movement artifact was removed using the correction procedure of Semlitsch et al., (1986). ERP peaks were identified with a procedure routinely used in our laboratory in which template time windows are established based on the grand averages for each stimulus type. The greatest positive or negative going deflections from baseline to peak are identified within those windows. The N2 window was set between 180 and 300 msec and the P3 window was set between 250 and 600 msec.

Event-related potentials (ERPs) were averaged separately for the Go, NoGo, “X-only,” “A” and Nontarget CPT letters. Only correct responses to the stimuli were included in the averages (e.g. correct button press to Go, or correct inhibition of button press to NoGo and X-only). None of the participants had less than 20 out of 40 stimuli included in the averages after correction for artifact and task performance. For the auditory stimuli, the mean number of trials per average was 31.7, SD 5.9 (PTSD) and 36.4, SD 3.5 (controls); and for the visual stimuli 36.2, SD 3.9 (PTSD) and 38.1, SD 1.6 (controls).

Statistical Analyses—Differences between the groups for the demographic variables were compared using independent samples *t*-tests. Unequal sample size was accounted for by using Levene's Test for Equality of Variances. Significance was determined using a two-tailed test with a 95% confidence interval. Analyses of variance (ANOVA) and analyses of co-variance (ANCOVA), with depression score and age as the covariates, were performed to examine amplitude and latency differences among the task conditions and between the groups. Greenhouse-Geisser corrections to the degrees of freedom were used and the corrected probability values are reported. ANOVAs were also repeated using a reduced sample of 10 PTSD and 10 control participants who were matched for age, education, and IQ to ensure that age effects did not account for observed group differences in the ERP measures. Pearson product moment correlational analyses were conducted to determine the relationship between

P3 measures (amplitude and latency) and behavioral measures of PTSD symptomatology. To account for the possible confounding effects of medications and panic disorder on the ERP and behavioral findings in the PTSD group, the PTSD participants were divided into two groups and additional t-test comparisons were made between these groups. The “low” group was composed of ten participants who were either on no medications or a maximum of one medication and had no panic disorder diagnosis (history or current), and the “high” group was composed of thirteen participants who were on three or more medications, with eleven having a history or current diagnosis of panic disorder.

Results

Participant demographic information, psychological interview, and behavioral data can be found in Table 1. The control and PTSD groups did not differ on years of education [$t(34) = -1.37$, ns] or estimated IQ [$t(34) = -1.87$, ns]. The mean age for the controls was about 6 years younger than the mean for the PTSD group [$t(34) = 3.65$, $p = .003$], although the age ranges for both groups were similar. Scores for the BDI and the CAPS-DX showed, as anticipated, that depression and hyperarousal scores were significantly higher for the PTSD group [$t(34) = 6.72$, $p < .001$; and $t(34) = 15.12$, $p < .001$, respectively]. Behavioral data from the auditory and visual CPTs indicated that RTs to the Go stimuli did not differ between the groups in either modality [$t(34) = .45$, ns; and $t(34) = 0.82$, ns, respectively]. The percentage of correct responses to auditory Go and NoGo and visual Go and NoGo stimuli for the PTSD group was 90.5%, 98.7%, 94.3%, and 98.4%, respectively; and for controls 95.8%, 100%, 93.7%, and 99.8%. The total number of correct responses to Go stimuli differed between the groups for the auditory CPT [$t(34) = -2.65$, $p = .013$], with the PTSD participants having fewer correct responses than controls, but did not differ between the groups for the visual CPT [$t(34) = -.30$, ns]. The SCID-IV provided a detailed history of the amount and chronicity of alcohol and/or drug use for each participant. Out of the twenty-three participants, 6 had a history of alcohol dependence, 2 had a history of drug dependence, and 4 had a history of both drug and alcohol dependence. None of the PTSD participants had alcohol or substance abuse or dependence within a minimum of six months of testing.

P3 Amplitude Effects

Figure 1 illustrates the grand averages from the PTSD and Control groups for the Go, NoGo and nontarget stimuli during the auditory and visual CPTs.

Go and NoGo—First, a Modality (auditory, visual) X Condition (Go, NoGo) X Site (Fz, Cz, Pz) X Group (PTSD, Control) ANOVA was conducted to determine if previous P3 amplitude topography distributions produced by the Go and NoGo conditions (Tekok-Kilic et al. 2001) were replicated here, and if group topographical differences were present. ANCOVA was also conducted, with depression (BDI scores) and age as separate covariates. None of the interactions were affected by the covariates of depression or age. The ANOVA revealed that a significant Modality X Condition X Site interaction was present [$F(2,68) = 7.72$, $p = .002$], indicating that the Go and NoGo conditions produced the anticipated differences in topographical distributions regardless of group, and that these differences were affected by modality. As seen in figure 2, the Go stimuli produced a posterior maximum distribution, while the NoGo stimuli produced a relatively greater fronto-central distribution.

The Modality X Condition X Site interaction was probed further to determine the effects of modality on the topography distributions. First, Condition X Site ANOVAs for the auditory and visual modalities showed that Condition X Site interactions were present for both modalities [$F(2,70) = 26.19$, $p < .001$; $F(2,70) = 58.37$, $p < .001$, respectively]. As seen in figure 2, both modalities produced the parietal maximum scalp distribution for the Go condition, and

the more fronto-central maximum distribution for the NoGo condition (response inhibition). Second, Modality X Site ANOVAs for the Go and NoGo stimuli separately showed significant Modality X Site interactions for both stimulus types [$F(2,70) = 21.83$, $p < .001$; $F(2,70) = 10.49$, $p < .001$, respectively]. Examination of each of the three midline sites separately showed the visual modality produced greater P3 amplitude than the auditory modality at each site during both the Go and NoGo conditions. Lastly, Modality X Condition ANOVAs did not produce significant interactions at any of the midline sites. These findings suggest that although the parietal maximum distribution to Go, and fronto-central maximum distribution to NoGo, were present for both modalities, the three-way interaction was produced by an enhancement of these topographical differences between the Go and NoGo stimuli for the visual compared to the auditory modality.

The next question of interest regarding P3 amplitude was whether or not target detection (Go) produced a reduction in P3 amplitude at the parietal site in the PTSD group compared to controls, and if this effect was dependent on modality. To test this question, a Modality X Site X Group ANOVA was conducted only for the Go condition. No main effect or interactions for Group were present. Thus, in the CPT task, P3 amplitude to target detection was not reduced for the PTSD group relative to controls, as has been observed in previous studies using oddball tasks.

Nontarget, X-only, and A—Modality X Site X Group ANOVAs (and ANCOVAs with depression and age as covariates) were performed to examine the group differences in P3 amplitude to the nontarget, “A,” and “X-only” stimuli separately. No significant main effect for group or group interaction was present for the “X-only” stimuli. For the “A” stimuli, a significant Site X Group interaction was present [$F(2,68) = 4.84$, $p = .022$]. This interaction, however, was no longer significant when ANCOVAs using depression and age were conducted. Main effects for modality were present for both the “X-only” and “A” stimuli due to overall greater amplitude for the visual compared to the auditory modality.

For the nontarget stimuli, again, a main effect was present for modality [$F(1,34) = 14.16$, $p = .003$] due to greater P3 amplitude during the visual compared to the auditory CPT. A significant Site X Group interaction was present [$F(2,68) = 9.88$, $p = .001$]. This interaction remained significant when depression and age were used as covariates. Figure 3 illustrates this interaction. Group differences were tested at each electrode site separately using one-way ANOVA. A significant P3 amplitude difference was present between the PTSD and control groups at the Fz site [$F(1,34) = 12.31$, $p = .001$], but not at the Cz or Pz sites. Comparisons were also made between Fz and Pz amplitudes within each group separately. Amplitude at Fz and Pz did not differ for the PTSD group, but for the control group, Pz was significantly greater than Fz [$F(1,12) = 40.77$, $p < .001$]. Thus, as seen in Figure 3, regardless of modality, the PTSD group had greater P3 amplitude to nontarget stimuli than controls only at the frontal site. Further, within the PTSD group, no difference in amplitude was present between the frontal and parietal sites, but a significant increase in amplitude from Fz to Pz was present for controls.

As an additional precaution to ensure that the significantly greater frontal P3 amplitude to the nontarget stimuli in PTSD was not confounded by the age difference between the groups, a secondary analysis was conducted. The three youngest control participants were eliminated, leaving 10 controls. Ten PTSD participants were selected to match the controls for age, education, and IQ, with the result that no differences were present between the groups for these measures. In this reduced sample, the Site X Group interaction remained significant for the nontarget stimuli [$F(2,36) = 5.92$, $p = .006$], with the PTSD group showing greater P3 amplitude than controls only at the frontal site.

P3 Latency Effects

Table 3 presents the mean latencies to the Go, NoGo, and nontarget stimuli for the PTSD and control groups. Latency effects for the Go and NoGo stimuli were examined with a Modality X Condition (Go, NoGo) X Site X Group ANOVA. When depression and age were used as covariates, they had no effect on the findings. A main effect was present for modality [$F(1,27) = 7.76, p = .01$] due to longer latency for the visual than the auditory modality. There was also a significant Condition X Group interaction [$F(1,34) = 14.60, p = .001$]. Figure 4 illustrates this interaction. For the Go stimuli, latency did not differ between the groups [$F(1,34) = 2.87, ns$]. For NoGo, however, the PTSD group had longer latency than controls irrespective of electrode site [$F(1,34) = 18.96, p < .001$]. Further comparisons revealed that P3 latency for the control group did not differ between the Go and NoGo [$F(1,12) = 2.14, ns$], while the PTSD group had significantly longer latency to NoGo than Go [$F(1,22) = 68.71, p < .001$]. For nontarget P3 latency, a Modality X Site X Group ANOVA showed no significant main effects or interactions.

Effects of Medication and Panic Disorder

Additional analyses were conducted to determine whether or not the significant ERP and behavioral findings described above were due to the effects of medication and/or panic disorder in the PTSD group rather than to the effects of PTSD itself. The PTSD participants were divided as described in the methods into “low” and “high” medication/panic disorder groups and t-test comparisons were performed to determine if the measures of interest differed between the groups. The means did not differ between the two groups for frontal P3a amplitude to nontarget stimuli [“low” group mean = 2.20, SD = 1.47; “high” group mean = 2.75, SD = 1.18; $t(21) = -.99, ns$], or for latency to the NoGo stimuli [“low” group mean = 400.8, SD = 11.2; “high” group mean = 405.7, SD = 23.3; $t(21) = -.61, ns$]. Neither were there differences between the groups for the total number of correct responses to Go stimuli during the visual CPT [$t(21) = .61, ns$]. The “high” medication/panic group did not differ from the “low” group for depression and hyperarousal scores [$t(21) = -1.99, ns$; $t(21) = -1.56, ns$, respectively]. Thus, the major significant findings of this study do not appear to be due to the effects of medications or panic disorder, although these effects cannot be ruled out.

ERP and PTSD Symptom comparisons (CAPS-DX)

The P3 amplitude and latency findings for Go, NoGo, and nontarget stimuli in the PTSD and control groups were compared with the avoidance, re-experiencing, and hyperarousal scores of the CAPS-DX. These comparisons were conducted to determine if heightened arousal was related to the shorter Go/longer NoGo P3 latency for the PTSD compared to Control group, and to the greater frontal P3 amplitude to nontarget stimuli seen in the PTSD group. Table 4 presents the relevant correlations. No significant correlations were present between CAPS-DX hyperarousal scores and Go, or NoGo P3 amplitudes when all participants were included, or for the PTSD group separately. P3 latency to Go stimuli (collapsed across modality and site) was significantly related to hyperarousal scores only in the PTSD group. P3 latency to NoGo stimuli was related to hyperarousal scores for the full sample and for PTSD separately, indicating that longer P3 latency to NoGo stimuli was associated with heightened arousal (see Figure 5, top). Nontarget P3 latency at Fz, Cz, and Pz correlated significantly with reexperiencing scores for the PTSD group, with longer latency related to greater reexperiencing. Nontarget P3 amplitude at the Fz scalp site was significantly correlated with hyperarousal scores for all participants, indicating that greater frontal amplitude was related to heightened arousal. This relationship was also present when controls were examined alone. It was not present for the PTSD group alone, although the PTSD group had greater frontal amplitude, in general, to nontargets than controls (Figure 5, bottom).

N2 Amplitude Effects

The amplitude of N2 was examined to determine whether or not group differences were present in the amount of negativity of N2 to NoGo relative to Go stimuli. The N2 component during the auditory modality was difficult to identify for many of the participants, and, thus, only data for the visual modality were analyzed. Here, the N2 component was identified for 17 PTSD participants and 12 controls. A measure of N2 negativity relative to P2 and P3 was obtained by calculating the mean amplitude of P2 plus P3 and subtracting the N2 amplitude from this mean. Therefore, the greater the negativity of N2, the greater the value obtained. A Condition (Go, NoGo) X Site (Fz,Cz,Pz) X Group (PTSD, Control) ANOVA revealed a significant Condition X Site interaction [$F(2,54) = 15.04, p = .001$], but no main effect or interaction for group was present. The Condition X Site interaction was produced by greater N2 negativity at the frontal and central sites relative to the parietal site for the NoGo compared to the Go condition.

Discussion

The present study revealed behavioral and ERP differences between Vietnam combat veterans with PTSD and healthy non trauma-exposed controls that suggest deficits in the processing of non affect-laden attentional stimuli in the PTSD group. The behavioral data in this study replicated the finding of Vasterling et al. (2002) that Vietnam veterans with PTSD had more errors of omission but not commission than controls on an A–X CPT task. In our study, however, errors of omission were greater only for the auditory and not the visual A–X CPT, while Vasterling et al. only used a visual CPT. We did not find reaction time differences between the groups to the Go stimuli for the auditory or visual CPT. Medications, comorbid panic disorder, and intellectual function did not appear to influence our finding. Vasterling et al. (2002) also reported that intellectual function was not a factor in their behavioral A–X CPT finding.

Amplitude effects

The general findings for P3 amplitude topography in this study were consistent with a previous investigation from our laboratory (Tekok-Kilic et al., 2001), in which, irrespective of modality, the Go condition produced a parietal maximum amplitude distribution consistent with target detection, and the NoGo condition produced a fronto-central maximum distribution similar to a P3a distribution. In the present study, both the PTSD group and controls showed these same Go and NoGo scalp topographical distributions. The visual modality, however, produced an overall greater P3 amplitude than the auditory modality, and a significant enhancement of the parietal maximum Go distribution than the auditory modality, but these findings were not dependent on group (see Figure 2). Both groups also showed the expected “NoGo N2” effect (see for example Freitas et al., 2007), with more N2 negativity present at fronto-central sites during the NoGo compared to the Go condition.

P3 amplitude was also examined to determine whether the PTSD group had reduced amplitude to the target X (Go) compared to controls in a CPT paradigm. This reduction in P3b amplitude to targets in PTSD participants relative to controls has been the predominant finding in previous PTSD studies that employed an oddball paradigm. In our study, we did not find that the PTSD group had a reduction in P3b amplitude at Pz compared to controls in response to target detection (Go). The context within which target detection occurs in the CPT task differs from a standard oddball paradigm, and thus, the absence of the lower amplitude may be explained by the differences between the two tasks. Unlike the oddball paradigm, the target X in the CPT is preceded by a stimulus that warns the participant to prepare to respond (the letter “A”). The decision-making balance between responding and not responding in Go/NoGo tasks, as described by Verleger et al. (2006), is a brief period of time in which participants are on a

decision-making “edge.” The finding of no difference in P3b amplitude between the PTSD participants and controls to the Go stimuli suggests that the warning stimulus (“A”) may be a strong enough stimulus to draw an equivalent amount of attentional resources to the task in both groups during this anticipatory period. This same interpretation may also apply to N2 and P3a amplitude to NoGo, in that no group effects were present for either the NoGo N2 or the P3a amplitude. Thus, the overall lack of group differences for Go and NoGo amplitude may indicate that the neuronal output, or attentional resources, required for response inhibition and response control processes does not differ between PTSD participants and controls. However, as discussed below, the decision-making process not to respond may be delayed in the PTSD group, producing a longer P3a latency to NoGo.

An important finding was present between PTSD participants and controls for P3 amplitude to the Nontarget stimuli. The PTSD group had significantly greater amplitude to Nontarget stimuli at the frontal site than controls. This finding was present regardless of modality (Figure 3). Further, the finding was robust. The significantly greater frontal P3 amplitude in the PTSD group was not affected when depression or age was covaried from the data, or when a smaller number of age-matched PTSD and control participants were analyzed separately.

The task-irrelevant (Nontarget) stimuli in the A–X CPT paradigm differ from the repeated nontarget (standard) stimuli in an oddball paradigm because in the CPT, each letter differs from the previously presented letter. As described above, healthy participants in the present study and in a previous study that employed the A–X CPT (Tekok-Kilic et al, 2001) showed a parietal maximum (P3b) amplitude distribution to nontargets (see Figure 3). This parietal maximum distribution to nontargets occurred in both the auditory and visual modalities and was similar to the target X distribution but lower in amplitude. The PTSD participants, however, showed a different scalp topography to the nontargets, with a higher fronto-central (P3a) amplitude distribution relative to controls. The P3a has been shown to occur in healthy young adults to auditory distractor stimuli of varying pitch, intensity, and location, even when the distractors are presented at a fixed 50% probability sequence (Jankowiak & Berti, 2007). In this same study, the degree to which the frontal amplitude was enhanced was demonstrated to be a function of the level of distraction created by the task-irrelevant distractor stimuli. These authors interpreted their findings to represent an attentional orientation to new information that is not dependent on the frequency or predictability of the occurrence of the distractor.

Kimble et al. (2000) found that combat veterans with PTSD showed greater P3a amplitudes at frontal electrode sites in response to novel non-repeating sounds than combat veterans without PTSD, but smaller P3s in response to rare, repeated distracting tones. Presumably, repeated distractors, as opposed to nonrepeating novel or distracting stimuli, tend to produce a more parietal maximum P3b because they do not provide enough new information over time to draw attention away from task-relevant stimuli. In our study, the irrelevant “nontarget” stimuli were nine randomly presented letters, which were neither novel, nor repeating. The relatively greater frontal P3 amplitude observed in the PTSD group to these irrelevant stimuli suggests that PTSD participants were responding to these stimuli as if they were novel or distracting stimuli rather than irrelevant nontargets. A disruption in attentional processes, such as in PTSD, could result in heightened attention to minimal changes in environmental stimuli (such as the nontarget stimuli of the A–X CPT) and thus, draw attention away from more relevant stimuli.

P3 latency effects

Latency findings in the present study revealed that the PTSD group had significantly longer latency than controls to the NoGo stimuli (see Figure 4). Interestingly, latency did not differ between Go and NoGo for controls, while the PTSD participants had significantly longer latency to NoGo than Go. Latency is thought to measure central processing speed and, thus, in the A–X CPT task, would reflect the timing of the decision making process to respond or

withhold responding to the stimulus. The control group did not show any difference in the timing of the response to either stimulus type. The PTSD group, however, was significantly slower when the decision to withhold responding was required.

Support for both the NoGo P3 latency and nontarget P3 amplitude findings in our study can be found in functional imaging studies. Carter et al. (1998) used an A–X CPT paradigm and demonstrated anterior cingulate activation to the response inhibition (NoGo) condition. The anterior cingulate also has shown increased activation in PTSD compared to controls to salient nonthreatening information in an oddball paradigm (Bryant et al., 2005). These authors proposed that because the ACC is associated with attention, vigilance, and processing of salient stimuli, the increased ACC activation seen in PTSD may be the result of heightened attention to the stimuli (i.e., hyperarousal).

Taken together, the findings related to ACC activation may help to explain the findings of our study. Generalized hypervigilance, or heightened attention to non-threatening stimuli, may be related not only to the P3a amplitude enhancements to neutral nontarget stimuli in our study, but also to the slowed latency to inhibitory NoGo stimuli. Although the ACC shows activation to attentional tasks in general, the NoGo stimuli may produce competition for additional resources from the ACC due to involvement of response control and inhibition systems (van Veen & Carter, 2002). This competition for ACC resources may be reflected by the longer P3a latency to the NoGo, but not Go, stimuli in the PTSD group compared to controls. As such, the delayed latency of the response in the PTSD group could be explained by abnormalities in the decision-making process not to respond.

P3 and PTSD symptomatology comparisons

To examine the relationship between electrophysiological measures of cognitive function in PTSD and independent measures of PTSD symptomatology, P3 latency and amplitude measures were compared with CAPS-DX avoidance, reexperiencing, and hyperarousal scores (see Table 4). For all participants, as hyperarousal scores increased, P3 latency became more prolonged to NoGo stimuli (see Figure 5, top). This same relationship was present for the PTSD group separately, and might have become significant for the control group if the sample size was larger. Higher hyperarousal scores were also related to longer P3 latency to Go stimuli, but only for the PTSD participants. Thus, as hyperarousal scores increased, P3 latency increased, suggesting longer time to process Go stimuli in PTSD participants with the greater levels of hyperarousal.

Additionally, nontarget P3 latency at all electrode sites was related to reexperiencing scores for the PTSD participants, with longer latency being associated with higher reexperiencing scores. A previous study (Attias et al., 1996b) lends support to these latency findings. They found significant correlations between intrusiveness scores on the Impact of Events Scale and P3 latency at Cz and Pz to combat-related picture stimuli in a three stimulus oddball paradigm. Longer latency was associated with higher intrusiveness scores. Intrusiveness scores are indicative of the degree to which distressing images, thoughts, or perceptions related to the trauma are reexperienced over time. The authors proposed that delayed processing of the stimuli, or longer P3 latency, occurred as a consequence of a preoccupation with traumatic recollections. In the present study, we showed that longer latency occurred to attentional stimuli that were noncombat-related, suggesting that delayed processing in PTSD is present, in general, for cognitive stimuli, particularly in those individuals with higher levels of arousal and reexperiencing. Continual reexperiencing of the trauma would logically produce a feedback loop to activate neural networks involved in the stress response, and, in turn, perpetuate heightened arousal.

The relationship between hyperarousal scores and nontarget P3 amplitude was also significant, and only for amplitude at the frontal site (Fz). For all participants, as hyperarousal scores increased, nontarget P3 amplitude became greater (Figure 5, bottom). Interestingly, this relationship was also present for the control group separately, but not for the PTSD group. However, the PTSD group had significantly greater mean frontal P3 nontarget amplitude than controls, as well as significantly higher hyperarousal scores than controls. It is important to consider that hyperarousal may be a common feature of anxiety disorders in general, including PTSD. Panic disorder and generalized anxiety disorder each have a comorbid occurrence rate with PTSD of approximately 30 – 45% (see for review Fairbank, et al., 1995). In the present investigation, panic disorder was allowed in the PTSD group, and 13 of the 23 participants had a history of, or current diagnosis of panic disorder. Twelve out of thirteen of these participants fell in the “high medication” group. When comparisons were made between the “low” and “high” medication groups, no differences were found between the groups for the ERP or behavioral measures. Hyperarousal symptoms, however, were present in both the “low” and “high” groups and appear to play the more significant role in producing enhanced P3a amplitude in PTSD than the effects of panic disorder. This finding provides convincing evidence that hyperarousal alone, without a diagnosis of panic disorder, plays the primary role in producing an enhanced frontal P3a to nontarget stimuli.

Auditory/visual modality findings

Comparisons of attentional functioning between the visual and auditory modalities revealed no differences between the PTSD and control groups for the topography of P3 amplitude to Go and NoGo stimuli. That is, both groups showed greater posterior P3 amplitude for target detection (Go) and a fronto-central shift for NoGo stimuli. The major findings were independent of the modality of stimulus presentation. The findings of Neylan et al. (2003) lend support to our findings. They found no differences in P3 amplitude or latency between PTSD and a combat-exposed nonPTSD group regardless of stimulus type (target, novel) or modality (auditory, visual). Although they did not analyze the topographical differences between target P3b and novel P3a, their published grand averages show that for both modalities, both groups produced a distinct fronto-central shift in amplitude to novel stimuli compared to targets. What cannot be determined from their study, is whether topographical differences were present between the groups or whether a noncombat control group would have produced a less prominent P3a compared to both combat-exposed groups.

Although the findings of the present study provide new information about disruptions in attention in PTSD compared to non trauma-exposed controls, there are several limitations that warrant discussion. First, the use of male Vietnam veterans with chronic PTSD presents a number of confounds that prevent generalization to other PTSD groups. In our sample, there were fairly high rates of comorbid depression and panic disorder, psychotropic medication use, and history of alcohol and substance abuse. We attempted to control for most of these confounds, but their possible effects on the results cannot be dismissed. Second, the lack of a trauma-exposed control group prohibits drawing a firm conclusion that the attentional differences were specific to PTSD, and not to the effects of trauma exposure itself. Other studies, however, have demonstrated P3 differences that were specific to PTSD and not to trauma exposure. We also found, as mentioned above, that hyperarousal, even at the lower levels seen in controls, has some relationship with P3 measures. Third, an age difference was present between the PTSD and control groups. The range of ages of both groups, however, was very similar, with no young or elderly adults in either group. Because frontal P3 amplitude has been shown to be sensitive to the effects of aging, we conducted an additional analysis on an age-matched sample of 10 PTSD participants and 10 controls. This analysis provided fairly convincing evidence that the higher frontal P3a to nontargets in the PTSD group is robust and not due to age. Finally, the scope of this study did not allow for the evaluation of the Contingent

Negative Variation (CNV) on the anticipatory response to the cue and the expectancy to respond following the cue, as described by Bekker et al. (2004). The CNV could provide additional important information about inhibition and attention, respectively, in PTSD.

In summary, the findings of the present study support a growing body of evidence that cognitive impairments in PTSD are related to arousal mechanisms associated with fronto-cortical circuits. Further, these cognitive difficulties are not confined to trauma-related stimuli, but are also seen with neutral stimuli that place demands on attentional systems. This study was the first to demonstrate that response inhibition and screening of irrelevant stimuli each produced significant differences between PTSD and control participants in both the auditory and visual modalities. We also established a link between behavioral measures of hyperarousal and electrophysiological measures related to information processing and attention. Further studies are needed to determine whether these findings can be generalized to females with PTSD, to other trauma groups in addition to combat veterans, or to earlier diagnosed PTSD victims, and whether the findings are specific to PTSD or may include other disorders that involve heightened arousal, such as panic disorder.

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Figure 1.

ERP grand averages to Go, NoGo and nontarget stimuli at Fz, Cz, and Pz during the auditory and visual CPTs for the PTSD and Control groups separately. The arrow indicates stimulus onset (0.0 msec). Tick marks are every 300 msec.

Figure 2. Topographical effects of P3 amplitudes to Go and NoGo stimuli during the auditory and visual CPTs. The PTSD and control group data are presented separately to illustrate the group similarities in topographies for Go and NoGo. Error bars represent the standard error of the mean.

Figure 3.

P3 amplitude comparisons between the PTSD and control groups for nontarget stimuli collapsed across modality. The asterisk indicates a significant amplitude difference between the PTSD and control groups. Error bars represent the standard error of the mean.

Figure 4. Group differences in P3 latencies to Go and NoGo, collapsed across electrode site and modality. Error bars represent the standard error of the mean.

Figure 5.

Scatterplot (top) of the significant correlation between NoGo P3 latency and hyperarousal score for all participants, and (bottom) nontarget Fz amplitude collapsed across modalities and hyperarousal score. The dashed line on the X axis indicates a separation of data points for PTSD and control participants, with controls falling to the left of the line, and PTSD to the right.

Table 1
Demographic and task performance variables for Age Matched Groups: Means, ranges, and standard deviations.

Demographics	M	PTSD (N=23) (range)	SD	M	Control (N=13) (range)	SD
Age [*]	48.96	(44 – 52)	2.4	42.77	(38 – 53)	5.8
Education Level	13.7	(10 – 18)	2.0	14.6	(12 – 19)	1.8
Est. IQ ^a	107.0	(100 – 114)	4.2	110.1	(101 – 119)	5.6
CAPS-DX						
Avoidance	30.2	(6 – 49)	11.4	-	-	-
Re-experiencing	22.5	(3 – 36)	9.2	-	-	-
Hyperarousal ^{**}	26.6	(15–35)	4.8	4.5	(1 – 11)	3.0
BDI total score^{**}	26.6	(4 – 46)	10.8	4.4	(0 – 23)	6.6
CPT measures						
Auditory CPT # correct (Go) [*]	36.0	(27–40)	3.5	38.3	(37–40)	1.1
Visual CPT # correct (Go)	37.2	(34–40)	1.9	37.5	(32–40)	2.6
Auditory CPT # correct (NoGo)	39.5	(35–40)	1.2	40.0	(40–40)	0.0
Visual CPT # correct (NoGo)	39.4	(31–40)	1.9	39.9	(39–40)	0.3
Auditory CPT # commissions	1.7	(0–13)	2.7	0.9	(0–3)	1.2
Visual CPT # commissions	1.8	(0–15)	4.3	0.6	(0–3)	1.1
Auditory RT (Go)	.526	(.30–1.21)	.20	.498	(.24–.72)	.13
Visual RT (Go)	.469	(.32–1.07)	.18	.424	(.26–.60)	.11

CAPS-DX = Clinician Administered PTSD Scale for DSM-IV. BDI = Beck Depression Inventory. CPT = Continuous Performance Task.

^a IQ was estimated based on the North American Adult Reading Test (NAART)

* significant difference between PTSD group and controls, $p < .05$

** significant difference between PTSD group and controls, $p < .01$

Table 2**Medication and Comorbid Psychiatric Conditions**

	PTSD (N=23)
Medications	
Antidepressants: SSRIs	9
Antidepressants: Atypical	13
Antidepressants: Tricyclic	1
Mood Stabilizers/Anticonvulsants	3
Anxiolytics	7
Narcotics	1
Hypnotics/Sleep Aids	4
Psychiatric Diagnoses	
Bipolar Disorder I or II	0
Major Depression: Lifetime History	18
Major Depression Current	7
Dysthymia	7
Panic Disorder Lifetime History	13
Panic Disorder Current *	8
Social Phobia	1
Specific Phobia	2

* all 8 had lifetime hx (8 of 13), 7 of the 8 had panic disorder with agoraphobia

Table 3

Mean P3 latencies and standard deviations.

	Auditory			Visual		
	PTSD	Controls	PTSD	PTSD	Controls	Controls
Go	Fz	299.4 (52.6)	341.4 (86.9)	349.4 (57.3)		355.5 (53.7)
	Cz	300.7 (49.2)	342.3 (78.6)	358.1 (53.8)		370.7 (45.8)
	Pz	308.6 (57.7)	354.8 (72.0)	369.0 (56.7)		376.9 (60.1)
NoGo	Fz	371.9 (26.1)	345.2 (24.8)	426.2 (26.1)		393.5 (22.4)
	Cz	376.5 (24.6)	352.2 (31.8)	424.3 (25.8)		390.8 (24.0)
	Pz	392.2 (33.2)	363.8 (43.4)	430.3 (21.3)		398.1 (28.6)
NT	Fz	350.8 (35.6)	354.0 (34.3)	362.6 (49.7)		372.5 (48.0)
	Cz	351.9 (34.7)	354.0 (28.4)	366.8 (53.9)		370.8 (48.4)
	Pz	355.5 (40.8)	370.6 (36.0)	360.7 (52.0)		378.2 (51.2)

Table 4

Correlations (Pearson r values) between CAPS-DX scores and latency to Go and NoGo stimuli collapsed across modality and site; and Nontarget amplitude collapsed across modalities for each site separately.

		All Participants (n = 36)	Controls Only (n = 13)	PTSD only (n = 23)		
		Hyperarousal	Hyperarousal	Avoidance	Reexperience	Hyperarousal
Latency	Go	-.121	.330	.011	.271	.439 *
	NoGo	.688 **	.484	.157	.111	.460 *
Latency	NT Fz	.010	.150	.114	.684 **	.309
	NT Cz	.082	.342	-.003	.632 **	.340
	NT Pz	-.077	.209	-.053	.567 **	.360
Amplitude	NT Fz	.527 **	.557 **	-.047	.166	.052
	NT Cz	.251	.467	.100	.202	-.115
	NT Pz	-.114	-.079	-.063	.150	-.222

NT = nontarget stimuli.

*
p < .05 two tailed

**
p < .01 two tailed