6 TARGET DETECTION FOR NEUTRAL WORDS IN POST-TRAUMATIC STRESS DISORDER

6.1 SUMMARY

Background: This study investigated scalp topographic activity related to visual target word detection and associated response activation in post-traumatic stress disorder (PTSD). The study was expected to demonstrate visual, linguistic abnormalities, similar to previous findings of abnormal N2 and P3 ERPs for target tones in PTSD.

Task Manipulation: The task involved target detection during presentation of a pseudo-random series of red and blue words, for which attention was directed to one or another color. Attended words were monitored for any occurrence of a specific target word. Analysis of ERP activity involved comparison of attended target activity with attended non-targets for both the scalp potential and SCD.

Results: There was greater amplitude for targets during early stages of visual word processing, including a posterior temporal P80 ERP, superior prefrontal P150 ERP, superior parietal P150 SCD, posterior temporal N200 SCD, occipital and prefrontal P250 ERP, and a prefrontal P250 SCD. There was less target activity in PTSD for the posterior temporal N200 SCD and the prefrontal P250 SCD. Later, there was greater amplitude for targets in a posterior temporal N300 ERP, superior frontal N300 SCD, occipital and prefrontal P350 SCD, occipital P450 SCD, parietal P450 ERP, frontal N600 SCD, and a parietal P700 ERP. These latter effects were clear in the difference waves, which also demonstrate smaller amplitude in PTSD for a posterior temporal ND300 and a superior frontal PD700. There were indications of motor activity in left superior central peaks, including N300 and N600 SCD. There were delayed peaks in PTSD for the P450 and N600 SCD, which were associated with reaction times.

Conclusions: These diverse spatio-temporal effects in ERP and SCD components indicate the complexity of distributed processing systems engaged in target detection. The main finding here is deficits in target processing over the posterior temporal region at 200-300 ms in PTSD, which suggest abnormal discrimination of visual word forms. This activity was associated with reaction time and PTSD patients demonstrated slower reaction time, which was also indicated by delayed peaks over motor cortex during the response phase of target processing. This study provides specificity in measurement of target detection processes and it indicates, for the first time, spatio-temporal abnormalities in PTSD for linguistic target detection.

6.2 Target Detection in Post-traumatic Stress Disorder

This chapter investigates target detection. The previous chapter examined the role of working memory in the updating and maintenance of target representations. Given a valid and reliable target representation in working memory, any new stimulus is simply compared with this representation. The chapter on selective attention examined stimulus evaluation for a non-target stimulus. In that case, there were no overt responses required. In contrast, this chapter further examines the activity related to target recognition and associated response activation. In this study, the target representation was a fixed target, so there were no concerns about the integrity and validity of working memory for the target representation. Any presentation of a target word was assumed to simply consolidate the current target representation.

6.2.1 Target Detection and the P3 ERP

The conventional ERP indication of target detection is the N2/P3 complex (see reviews by Donchin, 1981; Rösler et al., 1986; Johnson, 1988; Donchin & Coles, 1988; Näätänen, 1990, 1992). Early studies employed "oddball" tasks that involved attention to a series of stimuli in order to detect and respond to infrequent targets, which demonstrated a positive parietal ERP at approximately 300 ms, with an amplitude that increased with lower probability of target occurrence (e.g., Sutton, Braren, Zubin & John, 1965; Squires, Wickens, Squires & Donchin, 1976; Duncan-Johnson & Donchin, 1977). The observed variation with stimulus probability promoted a proposal that P3 indicates stimulus expectancy and an important finding was the generation of P3 potentials for the absence of an expected stimulus in a regular stimulus sequence (e.g., Ruchin, Sutton & Tueting, 1975; Ruchin & Sutton, 1978). It was proposed that the stimulus absence conveyed information about the nature of the stimulus sequence and the P3 amplitude is related to the amount of information processing engaged by

unexpected or novel events (see Duncan-Johnson & Donchin, 1977; Johnson, 1988). Further studies of the relationship between P3 latency and reaction time demonstrated dissociations that support the interpretation of P3 potentials as an indication of stimulus evaluation rather than response selection and execution activity (Kutas et al., 1977; Squires, Donchin, Squires & Grossberg, 1977; Duncan-Johnson, 1981; McCarthy & Donchin, 1981; Magliero et al., 1984). For example, McCarthy and Donchin (1981) demonstrated that both P3 latency and reaction time decrease with easier stimulus discrimination, but only reaction time increases when response coordination becomes more difficult in a choice reaction task. In addition, several studies reported relationships between memory and P3 amplitude, whereby greater P3 amplitude during initial study of words was positively correlated with the effective retrieval or recognition of words after a long delay (Karis, Fabiani & Donchin, 1984; Fabiani, Karis & Donchin, 1986). Thus, evidence indicated a role of P3 in the updating of stimulus expectancy and evaluation of novel events, or "context updating" (see Donchin, 1981; Donchin & Coles, 1988). Several pharmacology studies indicate that catecholamines enhance P3 amplitude and decrease P3 latency under conditions of demanding information processing, providing evidence of a link between neurophysiology and cognitive capacity (Coons et al., 1981; Klorman et al., 1984; see also Clark et al., 1987).

It has also been shown that P3 potentials arise in the absence of attention, challenging the conception of P3 as a controlled process. Investigation of oddball tasks in the absence of attention indicated a frontal positive component, called P3a (Squires, Squires & Hillyard, 1975). The P3a is earlier and more frontally distributed than the parietal P3b generated while attending to the oddball task. The amplitude of the preceding N2 and the frontal P3a increases with greater differences in the physical attributes of rare stimuli from the common stimulus sequence, so these potentials indicate an automatic detection of physical stimulus deviance or novelty. The N2/P3a

potentials may be differentiated from the modality specific mismatch negativity (MMN), whereby the MMN indicates automatic, unconscious sensory detection of physical stimulus deviance and the N2/P3a reflect initial engagement of awareness and orientation to a novel stimulus (Simson, Vaughan & Ritter, 1977; Näätänen & Picton, 1987; Gaird, Perrin, Pernier, & Bouchet, 1990; Näätänen, 1992). During attention to the oddball task, the N2/P3a responses remain, but they are often obscured by superposition of larger positive P3b and slow wave potentials (Squires et al., 1975), although they are easier to differentiate with increasing inter-stimulus intervals and longer latency responses (e.g., Friedman, Vaughan & Erlenmeyer-Kimling, 1978). In addition, a frontal P3 potential has been identified for highly novel distracters during the attended oddball task (Courchesne, Hillyard & Galambos, 1975).

Many studies of P3 ERP sources have indicated contributions from various locations, including hippocampus, parietal and frontal cortex, which vary with different task parameters, suggesting that a distributed network of activity adapts to different aspects of stimulus processing (Desmedt & Debecker, 1979a,b; McCarthy & Wood, 1987; Halgren, 1988; Johnson, 1989, 1993; Ford et al., 1994; Halgren et al., 1995a,b; Baudena, Halgren, Heit & Clarke, 1995; Ebmeier et al., 1995; Halgren, Marinkovic & Chauvel, 1998; Moores et al., 2003). Some evidence suggests differential cortical involvement in novelty orienting (P3a) and conscious target processing (P3b). For example, frontal lobe injury incurs deficits in novelty P3 responses, but has limited impact on P3 amplitude and responses to target stimuli (Nielson-Bohlman & Knight, 1999; Daffner et al., 2000). Similarly, P3 amplitude during orienting is associated with frontal gray matter volume, while P3 amplitude during controlled processing is related to parietal gray matter volume (Ford et al., 1994). The source estimation studies are largely consistent with functional interpretations of P3a as the initial orientation of

frontal systems to a novel event, whereas the P3b indicates posterior systems engaged in further evaluation and sustained attention for the new information.

6.2.2 The Present Study

Important aspects of executive functions are the discrimination of relevant events from irrelevant distractions and the further evaluation of their significance in the context of adaptive action plans or goals. The task of this study combined both determination of relevance and the requirement of a simple response to significant target events. The selective attention chapter demonstrated ERPs related to the determination of relevance, while this chapter focuses on the latter determination of significance and appropriate action.

The task of this study consisted of a series of red and blue words, with attention required for one or the other color. A *fixed target* word in the attended color was defined prior to task commencement. This task only required a static working memory representation of a single target, which is compared against each new attended stimulus. Previous analysis has identified the activity engaged in stimulus discrimination and evaluation for attended compared with unattended common words; the comparison between the attended common and target words was expected to indicate the additional activity of target word recognition and motor response activity. The task is similar to a conventional oddball task, designed to maintain a constant target representation and rare target recognition and response activation. In large part, the target words are expected to elicit stimulus evaluation activity that commonly elicits a P3 ERP, but some modifications of this activity are expected for word processing. Also, the regular presentation of words doesn't replicate the physical novelty of rare target tones or simple visual features that are normally employed in P3 studies.

6.2.2.1 Stimulus Evaluation

As discussed in the selective attention chapter above, all attended words are candidates for target detection, which requires evaluation of their attributes against those of the target representation (see Magliero et al., 1984; Wijers et al., 1989a, 1989b, 1989c; Pritchard et al., 1999). That chapter illustrated that evaluation processes generate large positive scalp components at 250-600 ms over occipito-temporal, parietal and frontal regions. As noted previously, the amplitude and the duration of this evaluation processing can vary according to the similarity of the current stimulus with the target (e.g., Näätänen, 1992). Given the identity of target stimuli with the internal representation, target events can be expected to generate larger or more extended potentials than the non-targets.

6.2.2.2 Target Word Recognition

This study required detection or 'recognition' of a target word. ERP studies have clearly demonstrated a P3 ERP for target events, but many of these studies employ simple auditory or visual features, with clear physical differences between common and novel target stimuli (see Johnson, 1988). In this study, there are no immediate visual features of the targets that are easily discriminated from common words. Therefore, the task is unlikely to elicit the novelty P3a. Rather, the target ERP components are likely to indicate analysis of word form and further evaluation of linguistic information.

The initial processing of visual word features involves elementary visual form encoding within 90-150 ms, leading to an orthographic encoding at 150-250 ms, which has been associated with activity in the posterior fusiform and lingual gyri of the ventral occipital lobe (Petersen et al., 1993; Allison et al., 1994; Nobre et al., 1994; Halgren et al., 1994; Kuriki et al., 1998; Schendan et al., 1998; Cohen et al., 2000; Rao et al., 2003). Given that initial word form or orthographic encoding is required for target detection, the target evaluation and recognition activity was expected no earlier than

150-250 ms. The nature of target evaluation and recognition depends on the depth of encoding for the linguistic information, ranging from orthographic and phonological to semantic encoding (e.g., Gevins et al., 1995). Phonological representations generate the auditory phonetic features usually associated with visual linguistic stimuli, which has been related to left temporal scalp potentials at 450-500 ms (e.g., Gevins et al., 1995). Semantic encoding of visual words activates a distributed cortical network, including word form areas in lateral temporo-parietal cortex at 200-250 ms (Cohen et al., 2000), anterior fusiform gyrus at 400 ms (Nobre et al., 1994) and lateral prefrontal cortex (Petersen et al., 1990; Buckner & Koutstaal, 1998). Given these various processes of linguistic encoding, the recognition of target words might vary in timing from anywhere between 250-500 ms.

Studies of word recognition often present a list of words followed by a retention period and then a recognition list that contains both words studied and new words. Several studies have demonstrated that greater P3 amplitude is positively correlated with effective memory retrieval, under conditions of incidental learning (Karis et al., 1984; Fabiani et al., 1986). Also, under conditions of intentional learning, studies indicate greater positive ERPs over frontal sites at 300-500 ms and parietal activity at 500-800 ms for correct recognition of studied words compared to new words (e.g., Rugg et al., 1998). Note that the frontal activity appears to be similar to differences in congruous and incongruous semantic context effects on an N400 peak, which is larger for semantically incongruous words (Kutas & Hillyard, 1980a,b, 1983). Note that a distinction has been made between implicit and explicit memory (e.g., Schacter, 1992, 1995; Rugg et al., 1998). Implicit memory is an unconscious, automatic sensory register of familiarity, which can be measured in sensory activity for repeated events, called repetition priming (Schacter, 1992; see also Desimone, 1996). Rugg et al. (1998) found that ERP activity at parietal areas from 300-500 ms was more positive for

previously studied words than any new words, regardless of the depth of processing or whether any words are recognized, suggesting a posterior process for implicit memory. Note that at the same latency, frontal ERP activity was more positive for recognized words, providing evidence of simultaneous explicit recognition processing (Rugg et al., 1998). The parietal activity at 300-500 ms may be related to similar activity found in the incidental memory task of Fabiani et al. (1986). Also note that the parietal indication of implicit memory at 300-500 ms was followed by further parietal positivity at 500-800 ms that was sensitive to depth of processing (Rugg et al., 1998). Fabiani et al. (1986) found similar results for parietal activation during recollection and elaborate word encoding. Thus, perceptual implicit memory for words evokes parietal ERP activity at 300-500 ms, which may be accompanied by frontal explicit recognition activity in the same latency, while further stimulus evaluation or depth of processing is indicated by later positive activity at parietal areas.

These reports of word recognition involve retrieval of words from memory and evaluation of their recent familiarity, whereas the present study does not involve discrete stages of study, retention and recognition. In this study, the task requires a continuous maintenance of a target representation in working memory, to facilitate evaluation of the similarity of the current word and the target representation. This evaluation might involve some degree of familiarity or recognition processing, but as the target representation is maintained in working memory it should not involve any long-term memory retrieval (assuming that associative memory processing is limited in scope). So, it is important to differentiate this target detection process from the usual 'recognition' construct, which involves retrieval processes, although it is not unreasonable to consider it as that component of 'recognition' that involves a working memory evaluation and matching process. To some extent, the activity generated may resemble the word recognition potentials discussed. That is, it is important to note that

these potentials may differ, to some extent, from simple auditory and visual P3 potentials, due to the linguistic information available.

6.2.2.3 Response Execution

It is expected that target words elicit not only stimulus evaluation and recognition processes, with enhanced amplitude and duration, but also response execution activity. Once a target word is recognized, the prepared response can be executed. It is most likely that response processes are prepared and primed during the course of the task and that target processing involves a release of the response activity from controlled inhibition (see Rösler et al., 1986; Massaro & Cowan, 1993; Ilan & Miller, 1999). In this respect, it is difficult to attribute target-processing effects on ERP activity to either word processing and recognition or response preparation and execution. It may be assumed that most of the difference between common and target processing might be attributed to word processing and recognition, given that some degree of response preparation is common to both non-target and target events. However, at the moment of response activation and execution, the combined activity from target recognition and response execution cannot be easily decomposed in this study. To the extent that scalp topography reflects shallow cortical activity, the response activation is expected to manifest in scalp potentials over the left superior central region. Note that response selection is not required, as the task involved only a constant right index finger response.

6.2.2.4 Target Detection in PTSD

Impaired processing in PTSD has been indicated by delayed N2 and diminished P3 ERPs for rare target or distracter stimuli in neutral auditory tasks (see McFarlane et al., 1993; Charles et al., 1995; Metzger et al., 1997; Galletly et al., 2001). The diminished target P3 indicates deficits in contextual evaluation of stimulus significance

and associated maintenance of working memory representations for relevant stimulus attributes. The present study investigates whether there are any additional target recognition deficits in PTSD. As noted previously, this study estimates the component processes engaged during linguistic cognition, for which several neuropsychology reports document abnormalities in PTSD (Everly & Horton, 1989; Gil et al., 1990; Uddo et al., 1993; Yehuda et al., 1995; Anagnostaras et al., 1999; Vasterling et al., 2002). Given these findings of abnormal linguistic functions, it can be expected that measures in this study will identify abnormal activity in PTSD patients.

6.2.3 Hypotheses

An internal representation of target attributes is compared with every new attended stimulus. The degree to which they match is often reflected in the amplitude of the P3 ERP (for simple auditory and visual features). This study employs visual words, which involve linguistic recognition processes. These processes engage frontal executive systems, in addition to sensory and perceptual processes. Perceptual implicit memory for words evokes parietal ERP activity at 300-500 ms, which may be accompanied by frontal explicit recognition activity in the same latency, while further stimulus evaluation or depth of processing is indicated by later positive activity at parietal areas. Prior PTSD research has identified abnormal linguistic processes and abnormal auditory N2 and P3 ERPs (e.g., McFarlane et al., 1993). In this study, the PTSD patients may demonstrate deficits in the target activations, especially activity similar to the N2/P3 ERP complex. Response activation is expected in scalp potentials over the left superior central region. Previous studies indicate delayed reaction time in PTSD (e.g., McFarlane et al., 1993), so the response time and associated scalp components may demonstrate increased latency for PTSD.

6.3 METHOD

This chapter focuses on target detection for words in the fixed target task. This involved a pseudo-random series of red and blue words and responses to a specific word in the attended color (counterbalanced across red and blue). ERPs for attended non-target and target words are compared for both the scalp potential and scalp current density (SCD). See the general method chapter for further details.

6.4 RESULTS

6.4.1 Task Performance

For the fixed target task, PTSD patients were both slower to detect targets (PTSD: 589.56 ± 79.88 ms; CONT: 477.70 ± 51.25 ms) and detected fewer targets (PTSD: 94.44 ± 5.00%; CONT: 98.83 ± 1.77%). Patients and controls had similar low rates of false positives. See task performance chapter for further details.

6.4.2 ERP Signal-to-Noise Ratio

The number of EEG trials contributing to averaged ERPs for each condition and each group are summarized in Table 6-1. There was a significant group by condition interaction for the number of EEG trials in the attended common and target ERPs for the fixed target task (GROUP, $F[1,18] = 5.67$, $p<.05$; CONDITION, $F[1,18] = 234.84$, $p<.001$; GROUP x CONDITION, $F[1,18] = 5.31$, $p<.05$). After Bonferroni corrections, there were significant group differences for both attended common words ($M = 56.0$ trials, $SE = 23.77$ trials, $p<.05$) and target words ($M = 12.2$ trials, $SE = 5.09$ trials, $p<.05$). All subjects were presented with many more attended commons than targets, so the condition differences were expected. The group differences are solely due to artifact reduction procedures. Nevertheless, the mean number of trials for all averages was

large enough to obtain reasonable signal-to-noise ratios for the components of interest in this study.

Table 6-1. EEG trials for Attended Common and Target Words

Fixed Target Task	Control [a]				PTSD [a]			
	M	SD	Min	Max	M	SD	Min	Max
Attended Commons	209.80	(41.97)	166	271	153.80	(62.35)	64	264
Attended Targets	42.20	(9.03)	34	57	30.00	(13.33)	13	59

[a] n = 10.

6.4.3 Event-Related Potential Components

6.4.3.1 Group Means

The topographic layout of group mean ERP waveforms is given in Figure 6-1 and Figure 6-2, the superimposed waveforms and the difference waveforms are given in Figure 6-3 (see also Figure 6-4 to Figure 6-12). The waveforms demonstrate a consistent ERP component structure in response to both common and target stimuli, for both groups (see Figure 6-3). The following components were identified for further analyses, where the initial letter indicates polarity and the following number indicates the peak latency:

- superior frontal N80
- occipital and posterior temporal P80 and N150
- superior fronto-central P150
- P250 over the prefrontal, occipito-temporal and superior parietal regions
- a left posterior temporal N300
- a large, extended P450 over prefrontal and parietal regions
- prefrontal N550
- parietal P700

Summary statistics for these components are given in Table 6-2 and the inferential analyses are described below (see Table 6-3), with the mean differences for significant effects.

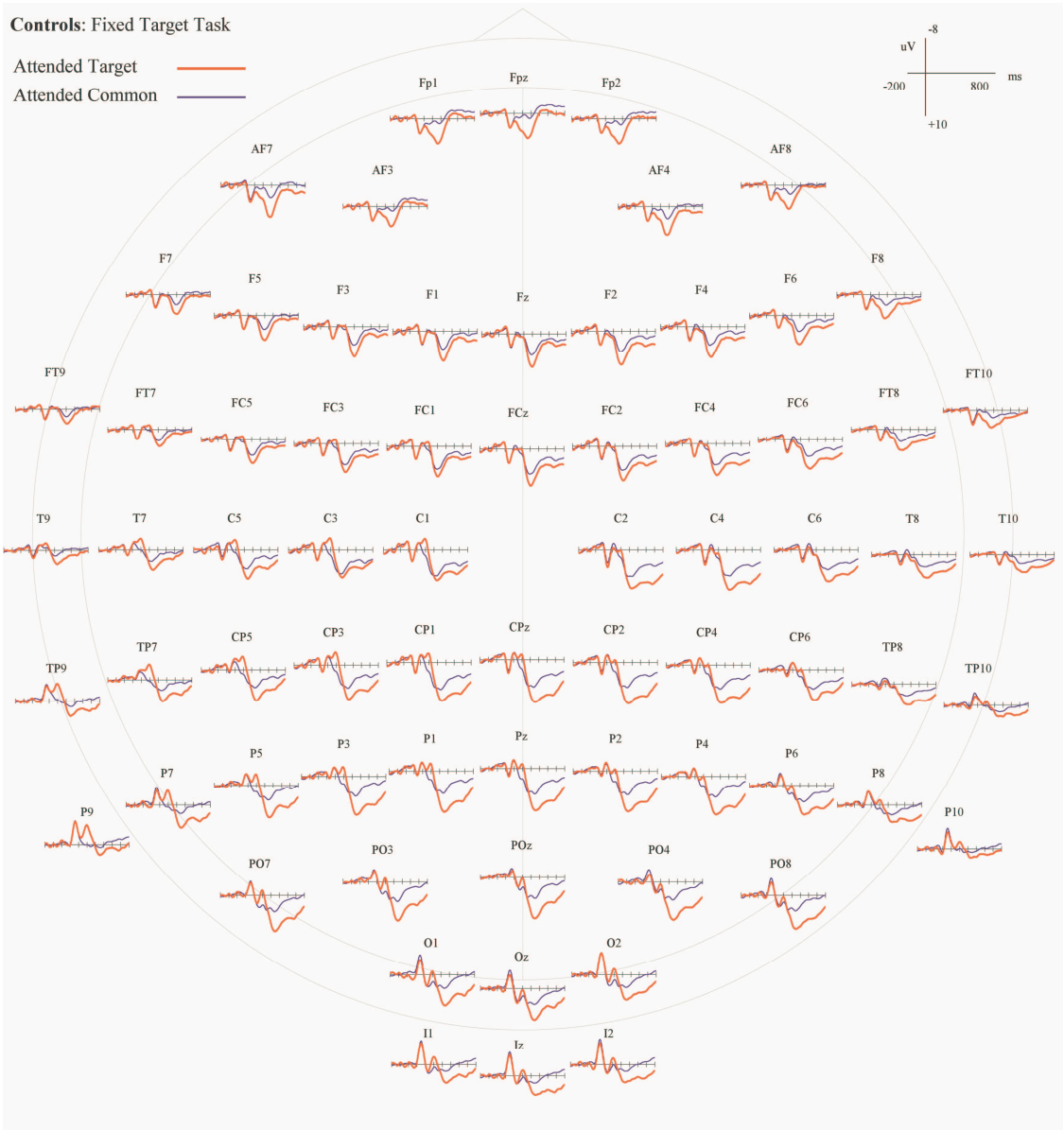

Figure 6-1. Controls (n = 10) ERP waveforms at 70 representative scalp sites (-200 to 800 ms, 100 ms intervals) for attended target and common words in the fixed target task. There is greater negative amplitude at 200-300 ms over left posterior temporal and central areas for the target words. The target words clearly elicit greater positive potentials at 400-800 ms over occipital, parietal and frontal areas. These components may correspond to conventional N2/P3 ERPs.

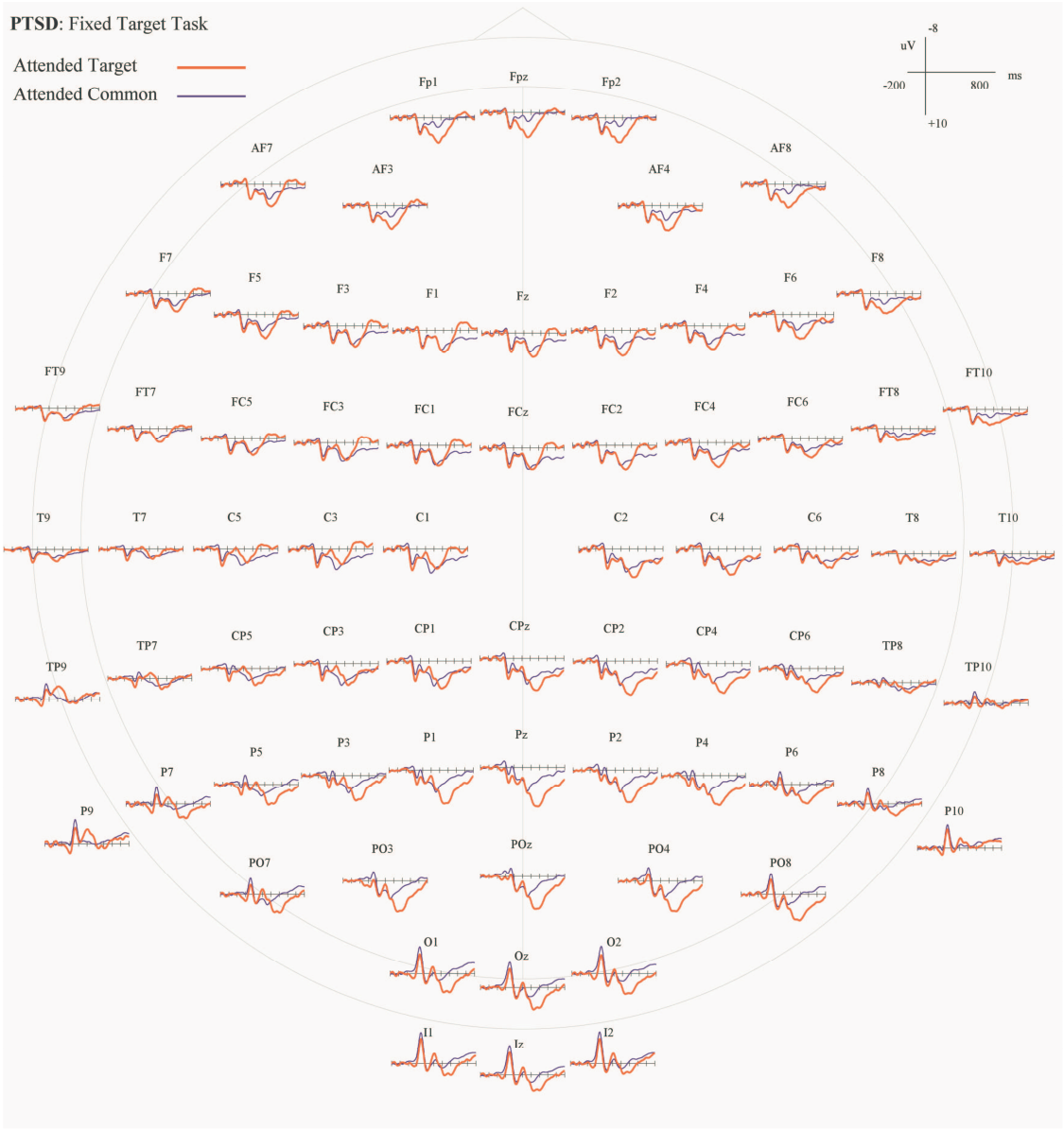

Figure 6-2. PTSD (n = 10) ERP waveforms at 70 representative scalp sites (-200 to 800 ms, 100 ms intervals) for attended target and common words in the fixed target task. There are small negative potentials for targets over left posterior temporal areas at 200-400 ms, followed by large positive potentials for targets at occipital, parietal and frontal areas between 400-800 ms. PTSD patients also demonstrate large negative potentials for targets over left fronto-central areas from 500-800 ms.

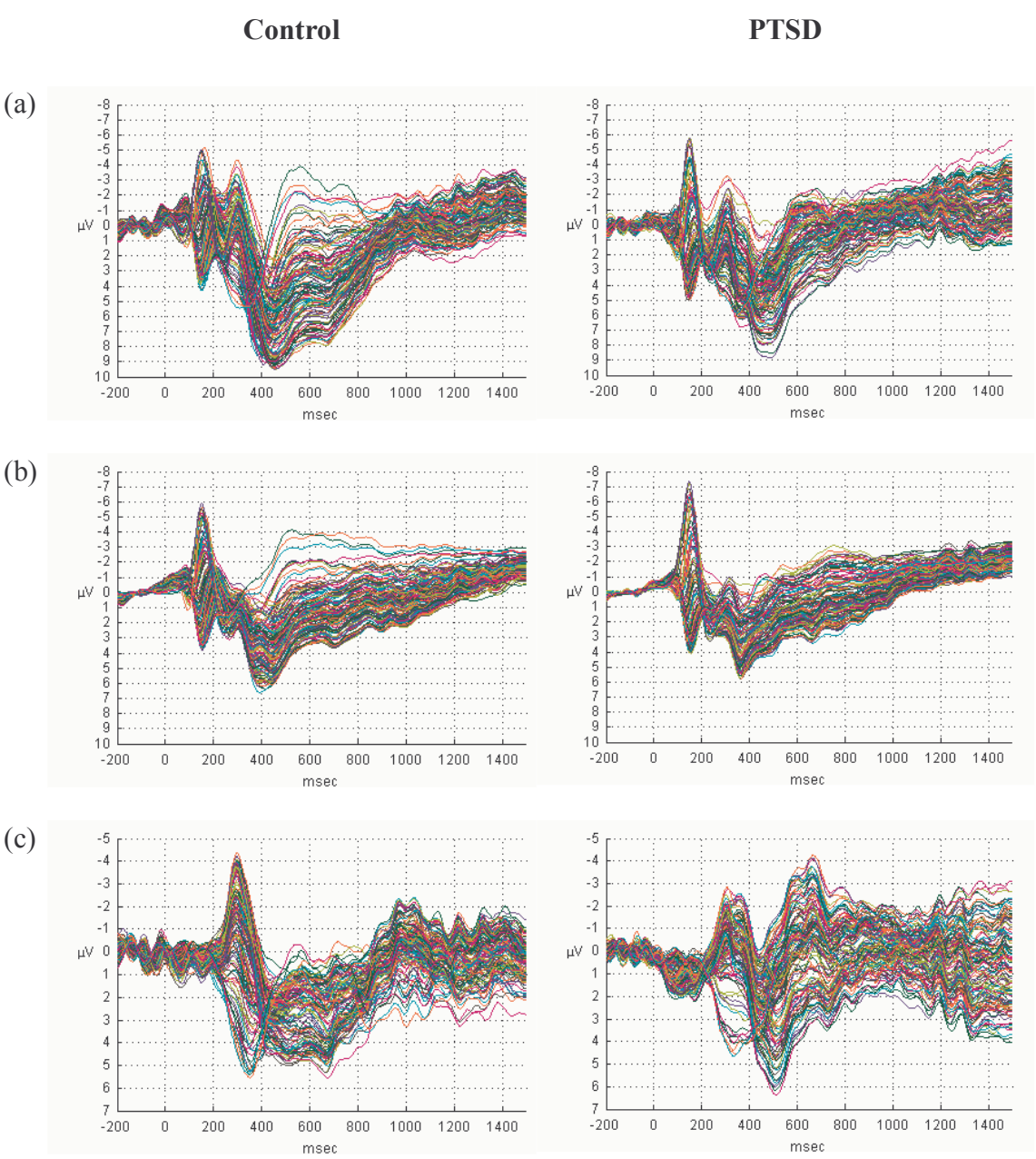

Figure 6-3. ERP waveforms at 124 scalp sites for controls (n=10) and PTSD patients (n=10): (a) target, (b) common, (c) target – common. The waveforms demonstrate similar component structures for both target and common words, across both groups. There appears to be larger or more extended target activity for controls from 300-800 ms. The difference waves illustrate discrimination of targets from commons at 200-400 ms, followed by further evaluation or response activation at 400-800 ms.

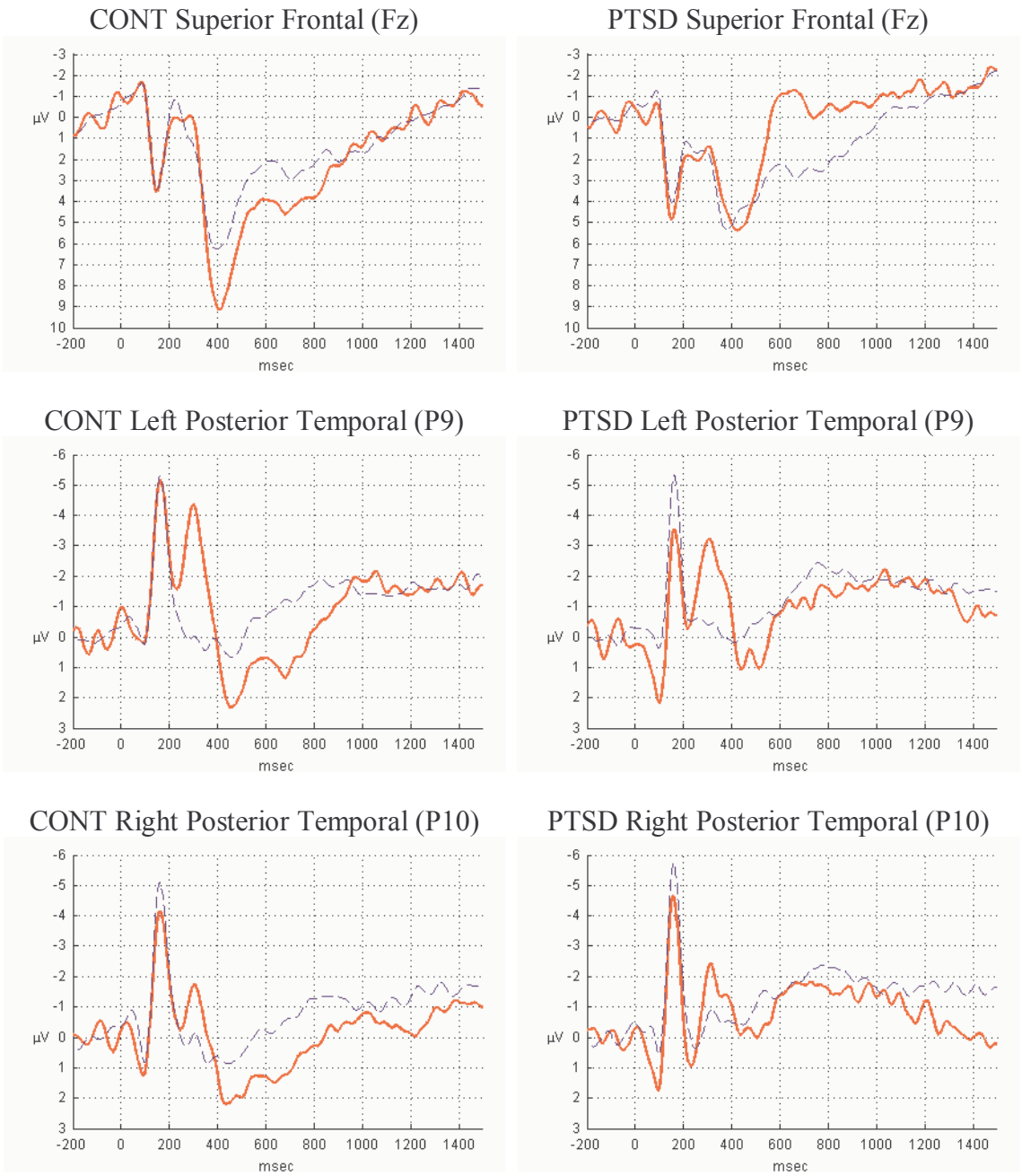

Figure 6-4. ERP waveforms for controls (n = 10) and PTSD patients (n = 10) at superior frontal and bilateral posterior temporal sites for both attended target (red, solid) and attended common (blue, dash) words. Note the small N80 at frontal sites and the small P80 at posterior temporal sites. These are followed by a larger N150 at posterior temporal sites, which appears larger for common than target words. The later posterior temporal N300 and the P450 are significantly larger for targets. From 500-900 ms, there are slow wave differences between controls and PTSD at frontal sites. See the component topography below.

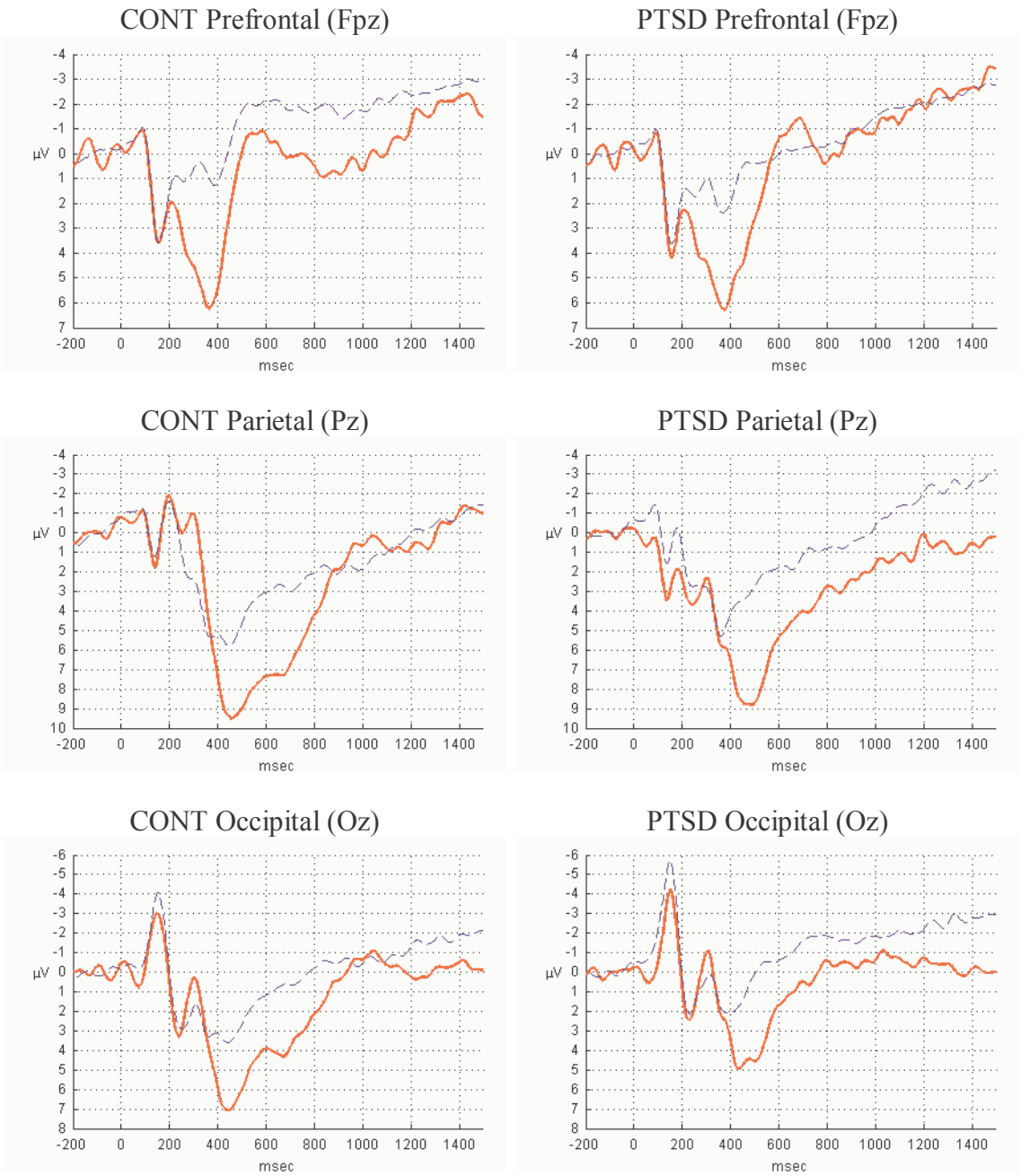

Figure 6-5. ERP waveforms for controls (n = 10) and PTSD patients (n = 10) at prefrontal and occipital sites for both attended targets (red, solid) and common words (blue, dash). There are eight components to note: (i) the prefrontal P150, (ii) the occipital N150, (iii) the occipital P250 (with a small inflection at prefrontal sites), (iv) the N300, which was larger at posterior temporal sites (see Figure 6-4), (v) the prefrontal P400, (vi) the parietal P450 and (vii) P700 (inflection), which are larger for attended targets, especially for controls, and (viii) the prefrontal N550, which coincides with reaction times (at 450-600 ms) and appears later in PTSD. Also note that controls demonstrate a frontal positivity for targets between 600-1200 ms. See the component topography below.

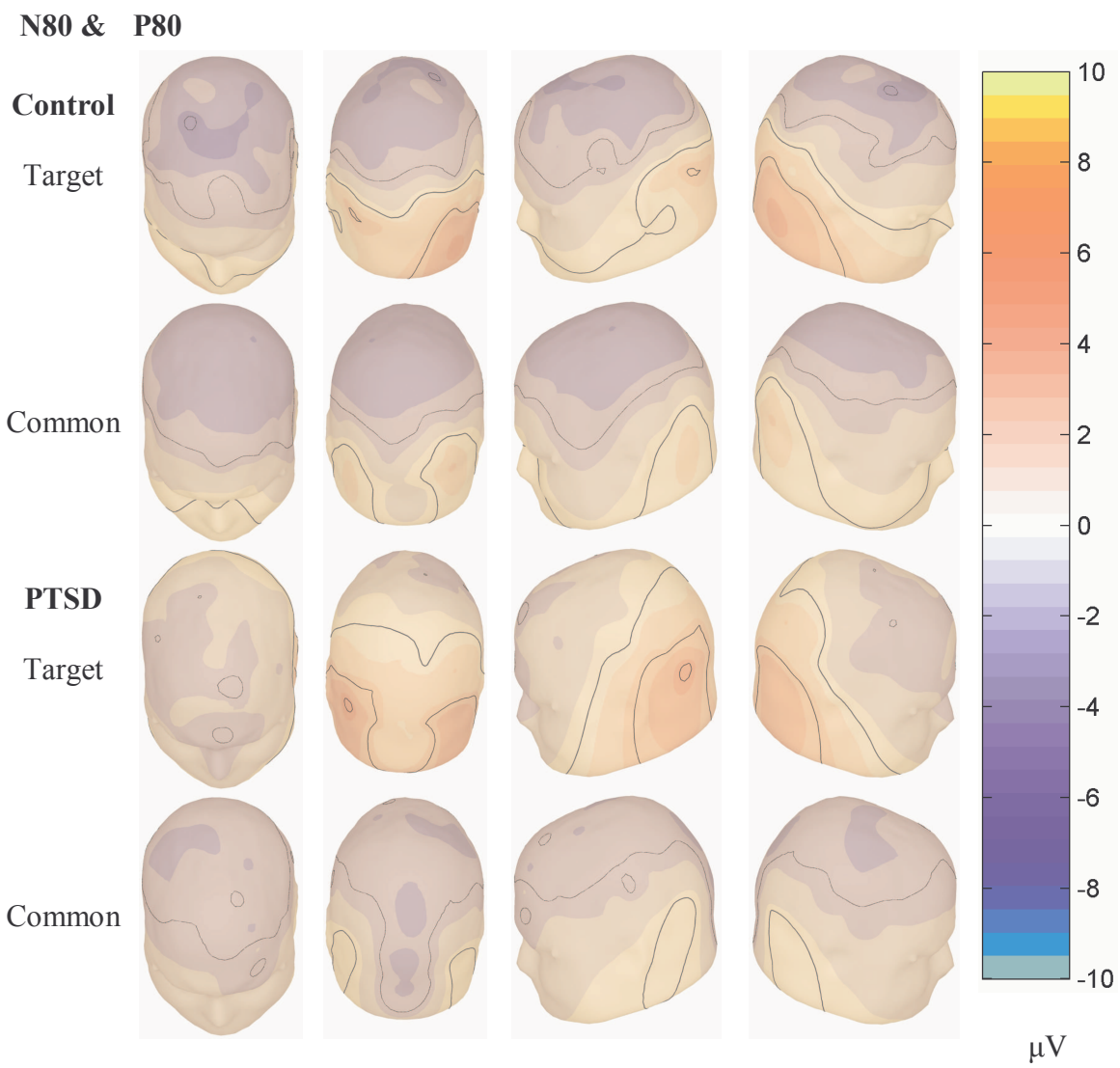

Figure 6-6. N80 and P80 ERP topography for controls (n = 10) and PTSD patients (n = 10) at 85 ms for both attended target and common words (contours at 1 μV intervals).

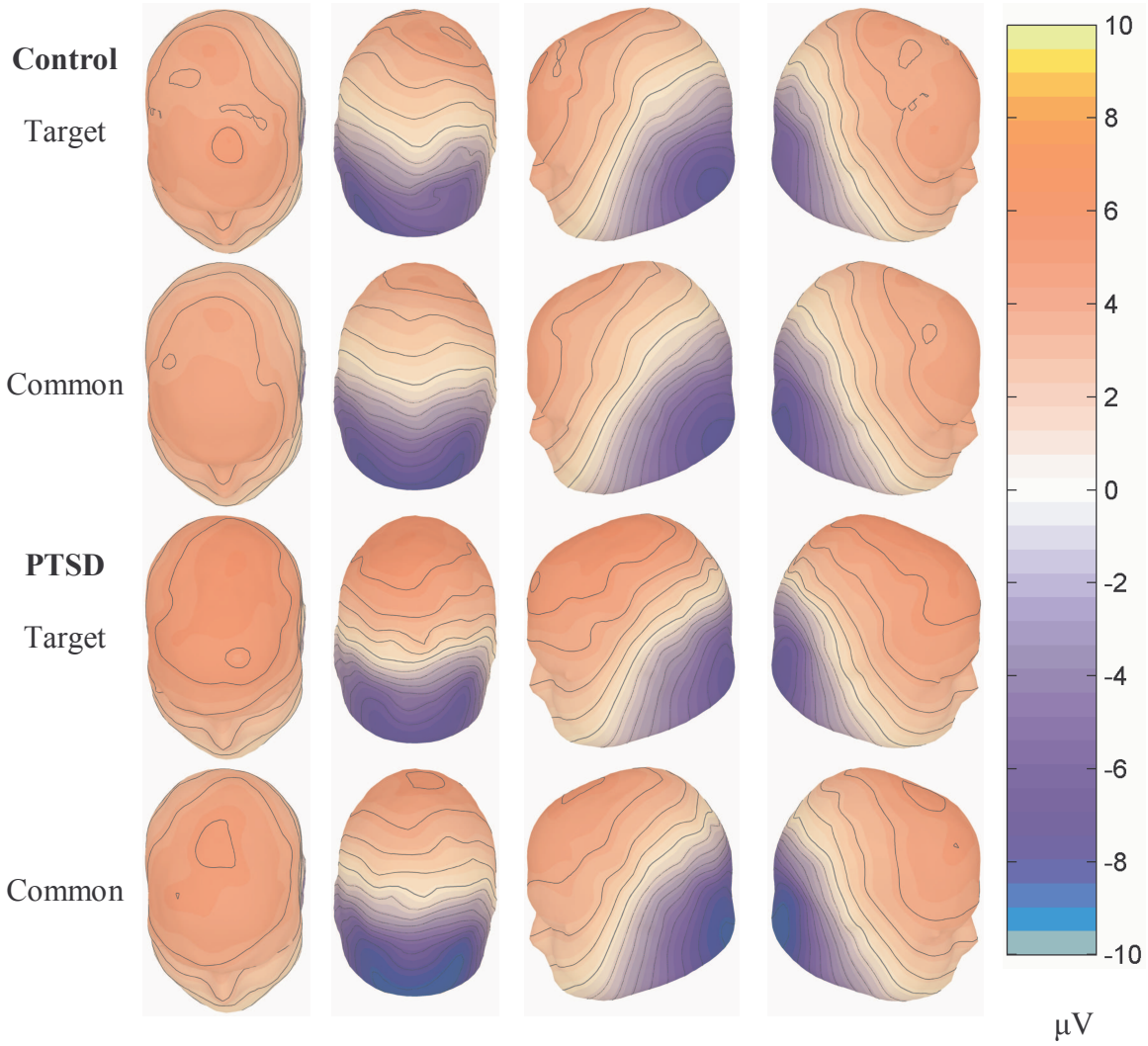

Figure 6-7. ERP topography for N150 (over occipital regions) and P150 (over frontal regions) for controls (n = 10) and PTSD patients (n = 10) at 155 ms for both attended target and common words (contours at 1 μV intervals).

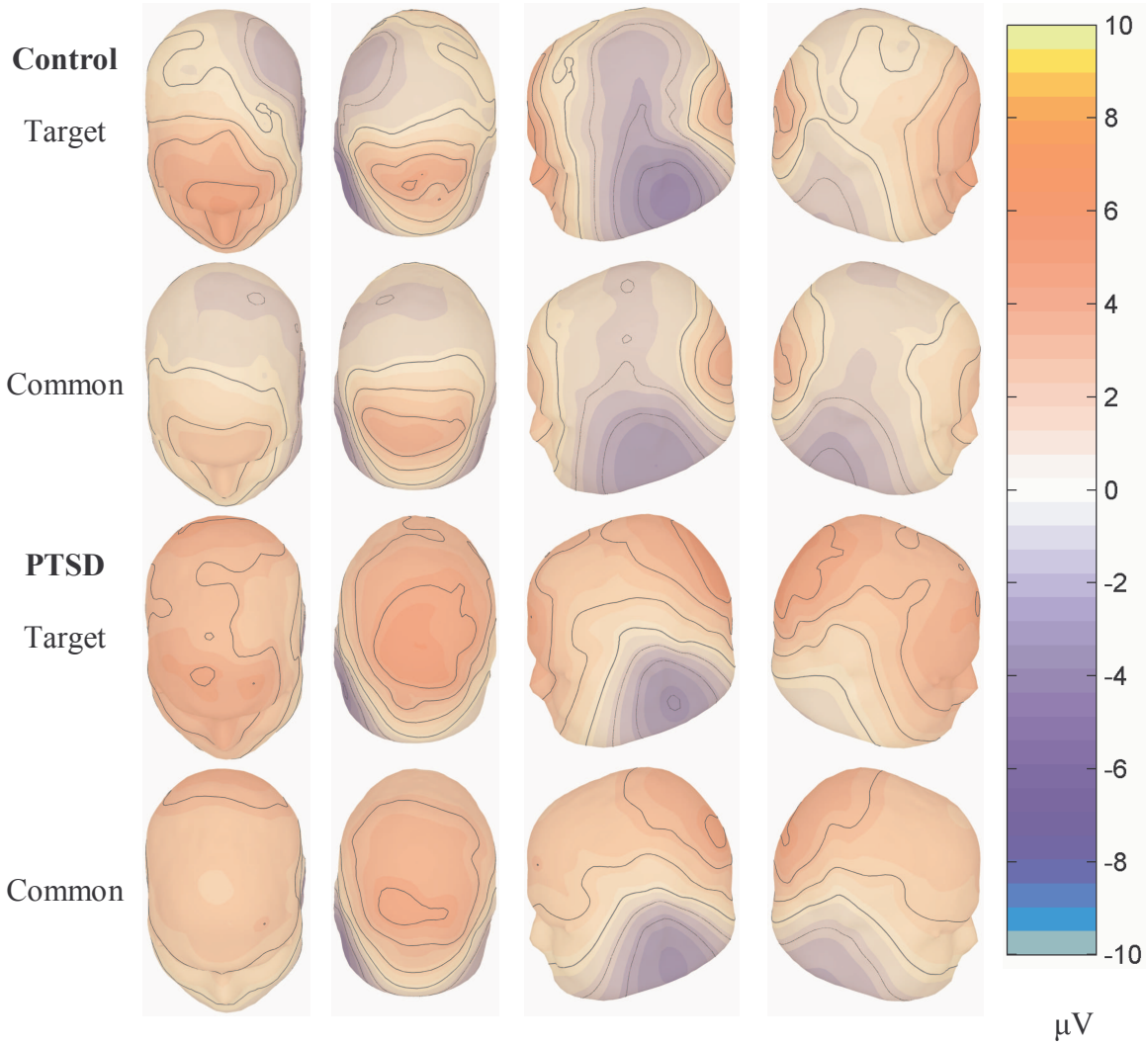

Figure 6-8. P250 ERP topography for controls (n = 10) and PTSD patients (n = 10) at 240 ms for both attended target and common words (contours at 1 μV intervals).

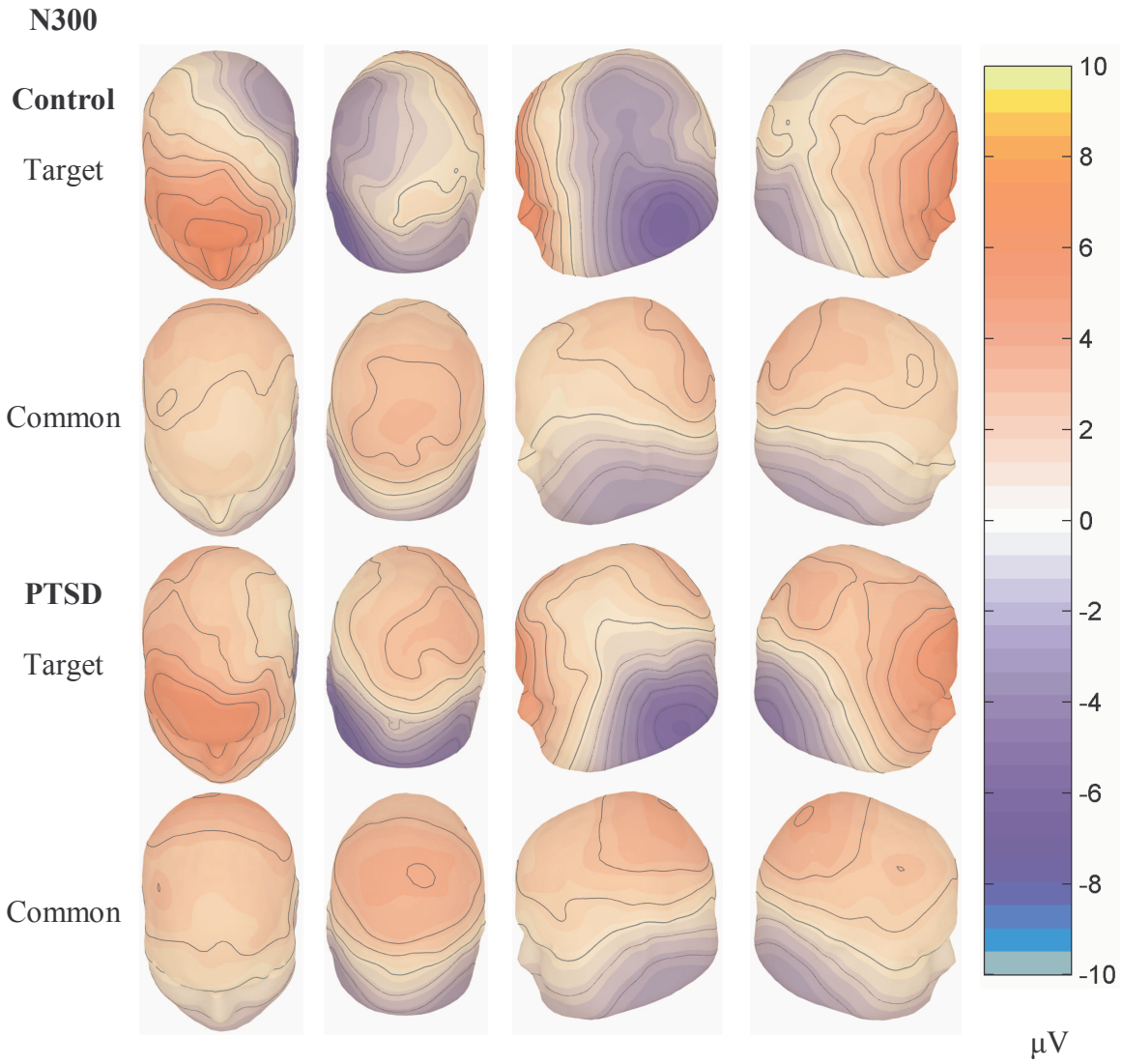

Figure 6-9. N300 ERP topography for controls (n = 10) and PTSD patients (n = 10) at 300 ms for both attended target and common words (contours at 1 μV intervals). The N300 is dominant at left posterior temporal sites for target words. This activity precedes the mean reaction times (CONT: 480 ms; PTSD: 590 ms).

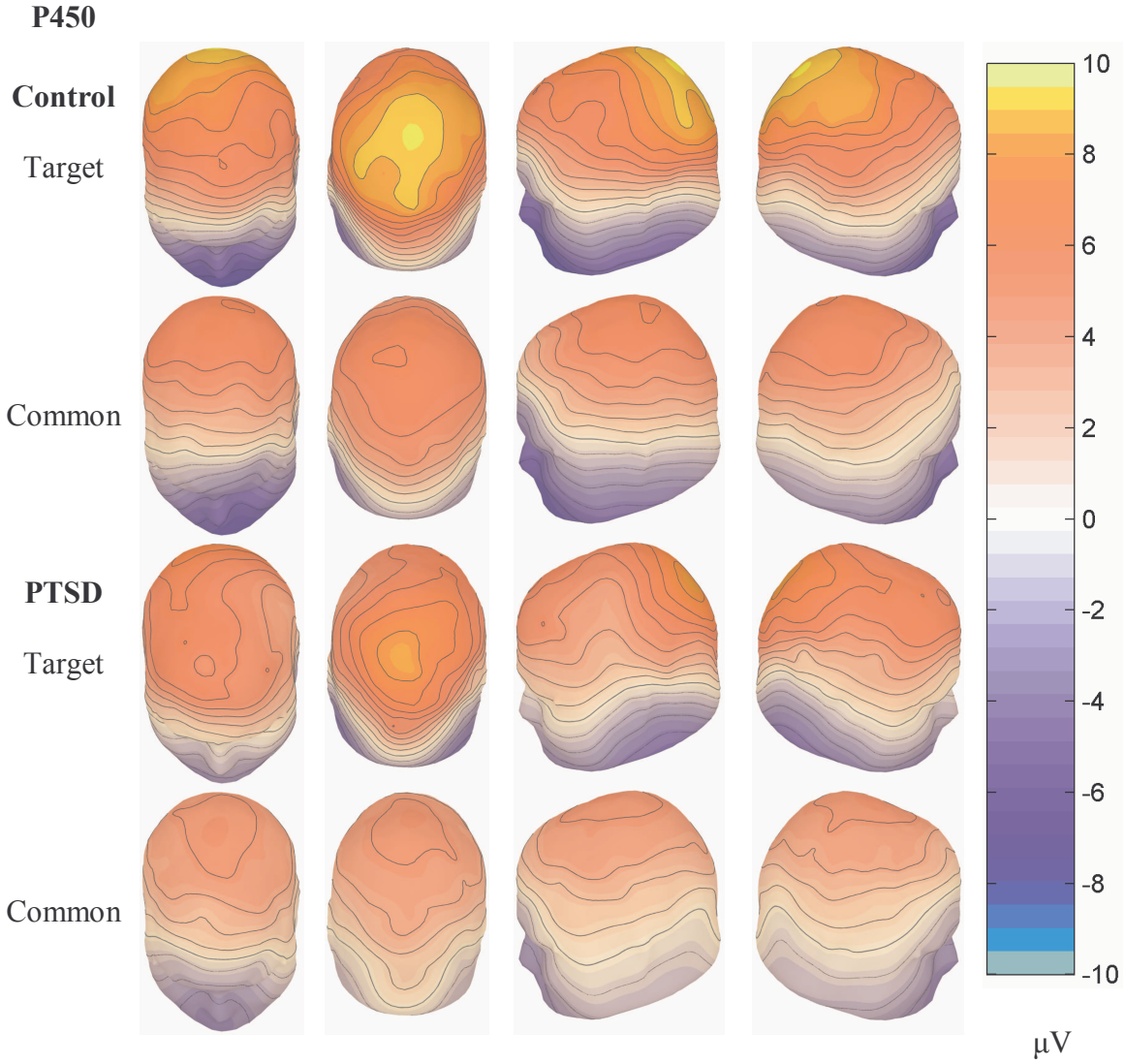

Figure 6-10. P450 ERP topography for controls (n = 10) and PTSD patients (n = 10) at 450 ms for both attended target and common words (contours at 1 μV intervals). The P450 is dominant at parietal sites for target words; it most closely resembles a conventional P3 ERP. The peak coincides with reaction times for controls (480 ms).

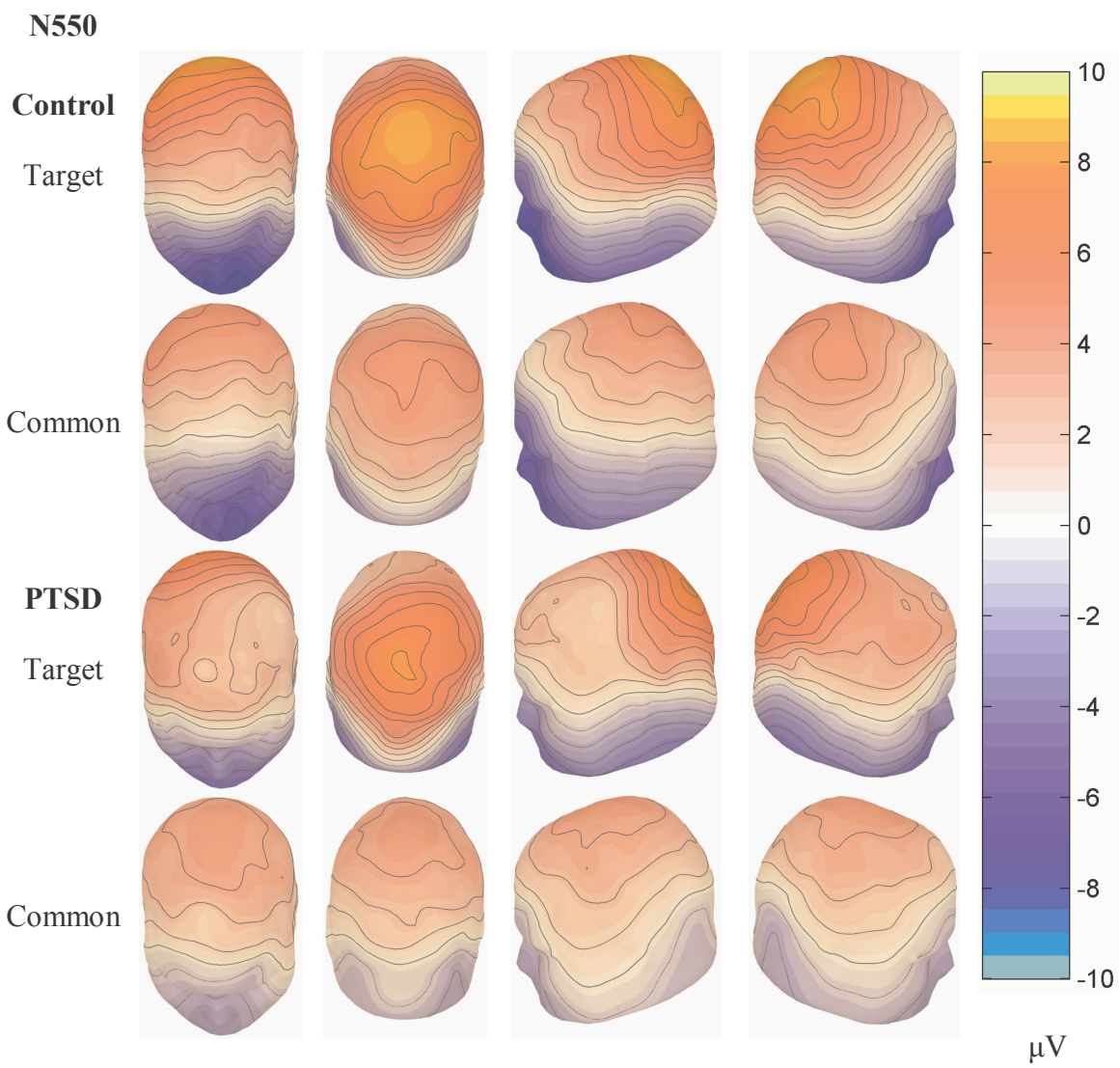

Figure 6-11. N550 ERP topography for controls (n = 10) and PTSD patients (n = 10) at 520 ms for both attended target and common words (contours at 1 μV intervals). The parietal positivity dominates the scalp topography, but a prefrontal negativity is apparent. This negativity becomes greater in PTSD and coincides with their mean reaction time (590 ms).

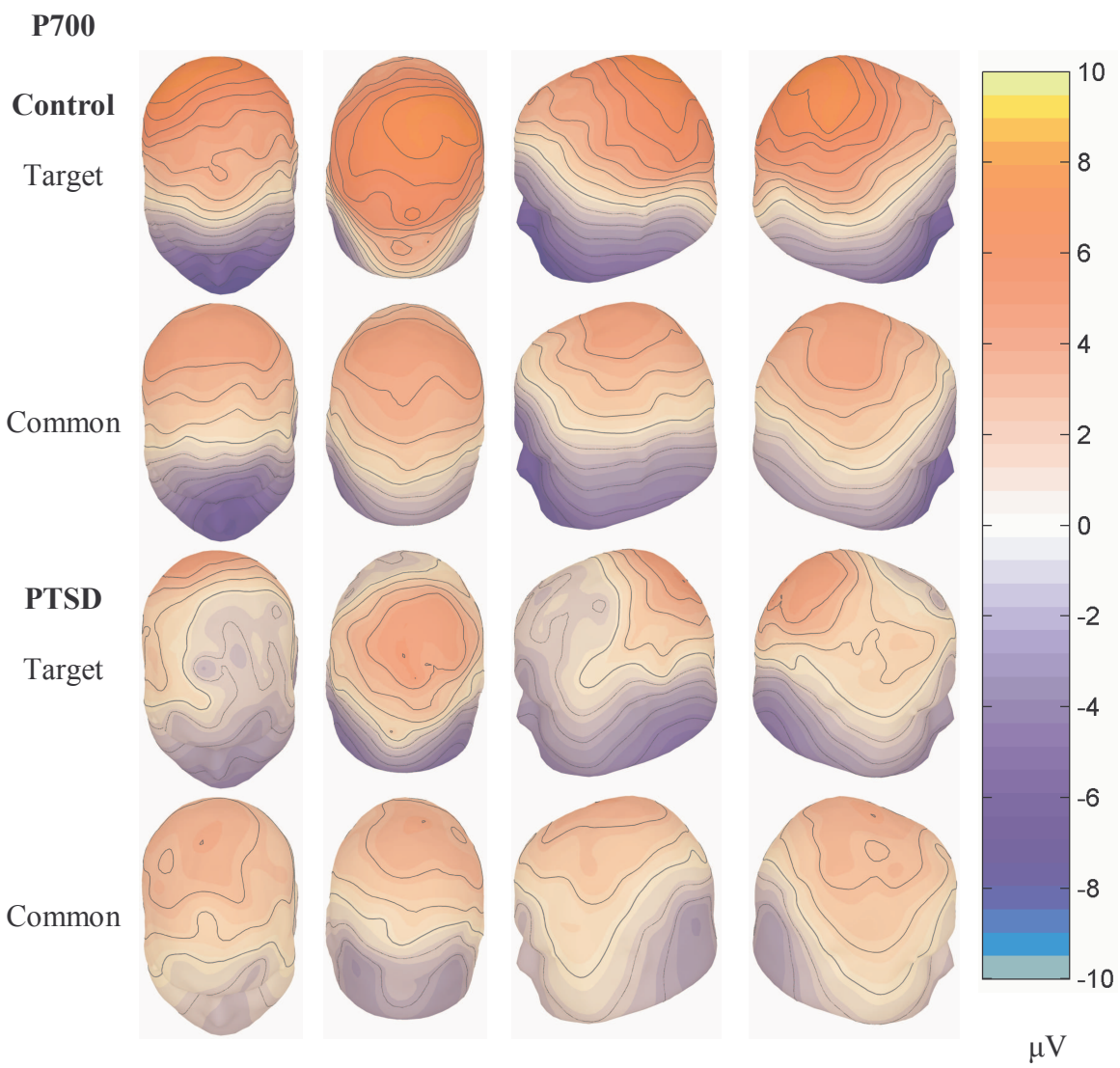

Figure 6-12. P700 ERP topography for controls (n = 10) and PTSD patients (n = 10) at 700 ms for both attended target and common words (contours at 1 μV intervals). The parietal positivity is dominant for targets in controls. PTSD patients have smaller parietal positivity, but larger frontal negativity.

Table 6-2. ERP Summary Statistics for Target and Common Words [a] (continued below)

			Amplitude (μV)		Latency (ms)	
			CONT	PTSD	CONT	PTSD
N80	Left	T	-2.62 (1.63)	-1.53 (1.43)	89.25 (14.14)	88.25 (15.86)
SF		C	-1.54 (0.71)	-1.81 (1.06)	87.75 (4.92)	88.75 (19.08)
	Right	T	-2.78 (2.12)	-2.06 (1.05)	86.25 (11.20)	86.50 (16.12)
		C	-1.72 (0.83)	-1.70 (1.08)	82.00 (13.53)	87.50 (17.48)
P80	Left	T	2.25 (2.11)	2.90 (1.11)	89.25 (14.14)	88.25 (15.86)
PT		C	0.16 (1.10)	0.63 (1.78)	87.75 (4.92)	88.75 (19.08)
	Right	T	2.28 (1.28)	2.53 (1.70)	86.25 (11.20)	86.50 (16.12)
		C	0.90 (1.36)	0.83 (2.04)	82.00 (13.53)	87.50 (17.48)
N150	Left	T	-7.09 (3.88)	-5.96 (4.81)	161.75 (16.75)	162.00 (15.22)
PT		C	-6.99 (3.99)	-6.83 (4.46)	162.50 (17.80)	164.50 (14.47)
	Right	T	-6.84 (4.35)	-6.73 (4.56)	161.50 (15.42)	160.75 (15.19)
		C	-6.58 (4.08)	-7.30 (3.59)	160.75 (14.24)	160.50 (14.47)
P150	Left	T	5.65 (2.69)	5.80 (3.38)	158.75 (22.24)	152.25 (16.01)
SPF		C	3.95 (2.33)	4.62 (2.62)	150.50 (14.13)	155.25 (13.72)
	Right	T	5.30 (2.31)	6.02 (3.31)	152.25 (16.73)	152.75 (15.34)
		C	4.22 (2.22)	4.91 (2.73)	151.25 (14.73)	158.25 (13.39)
P250	Left	T	4.21 (3.76)	5.12 (3.86)	238.25 (18.49)	233.25 (21.31)
OC		C	3.46 (2.47)	3.55 (4.57)	244.50 (12.12)	233.50 (12.20)
	Right	T	4.65 (3.20)	3.99 (4.07)	245.75 (14.82)	232.00 (20.20)
		C	3.47 (2.26)	3.03 (4.90)	248.00 (13.37)	233.50 (13.08)
P250	Left	T	4.34 (3.04)	4.33 (3.12)	246.50 (18.83)	246.75 (12.97)
IPF		C	1.76 (3.11)	2.27 (2.77)	241.75 (10.54)	246.25 (10.69)
	Right	T	4.25 (3.15)	4.00 (3.65)	246.75 (18.45)	245.50 (12.18)
		C	1.76 (3.31)	2.08 (2.60)	241.25 (10.88)	247.25 (10.24)

[a] T = Target; C = Common; CONT, n = 10; PTSD, n = 10; values are mean (SD).

Table 6-2 (continued). ERP Summary Statistics for Target and Common Words [a]

			Amplitude (μV)		Latency (ms)	
			CONT	PTSD	CONT	PTSD
N300	Left	T	-5.25 (3.80)	-3.60 (2.88)	302.25 (21.46)	299.00 (26.83)
PT		C	-0.19 (2.44)	-0.27 (1.70)	298.50 (19.90)	301.50 (20.39)
	Right	T	-3.07 (3.10)	-3.62 (3.40)	306.75 (21.76)	310.75 (16.96)
		C	-0.43 (1.85)	-1.03 (3.09)	306.50 (12.03)	312.00 (14.33)
P450	Left	T	12.47 (5.20)	10.91 (5.36)	469.50 (47.58)	459.75 (83.20)
SP		C	6.66 (3.40)	6.11 (3.22)	440.75 (70.94)	374.25 (56.20)
	Right	T	12.63 (5.57)	11.09 (5.48)	464.25 (49.05)	457.50 (79.98)
		C	6.50 (3.68)	6.60 (3.69)	440.75 (73.52)	396.00 (95.36)
N550	Left	T	-5.18 (4.67)	-3.99 (7.06)	524.50 (93.56)	479.00 (138.97)
IPF		C	-4.51 (5.55)	-2.19 (3.25)	453.00 (105.28)	489.75 (121.99)
	Right	T	-3.87 (1.83)	-2.03 (7.08)	521.75 (91.80)	462.25 (138.92)
		C	-3.14 (3.54)	-2.15 (2.89)	436.25 (111.53)	486.00 (97.13)
P700	Left	T	8.34 (5.77)	6.03 (4.44)	673.75 (55.29)	698.75 (56.51)
SP		C	3.47 (2.37)	2.79 (1.60)	720.75 (50.13)	673.00 (55.04)
	Right	T	8.90 (5.50)	6.08 (3.98)	688.75 (43.19)	685.00 (60.15)
		C	3.79 (3.02)	2.21 (1.55)	714.00 (59.54)	667.25 (49.71)

[a] T = Target; C = Common; CONT, n = 10; PTSD, n = 10; values are mean (SD).

Table 6-3. Inferential Statistics for Target Detection ERP Components [a]

ERP		GP	TD	GPxTD	HS	GPxHS	TDxHS	GPxTDxHS
N80	Amp	1.23	1.72	1.48	1.24	0.01	1.89	1.96
SF	Lat	0.07	0.10	0.30	3.56	0.85	0.19	0.41
P80	Amp	0.44	21.99***	0.10	0.21	0.50	10.07**	0.10
PT	Lat	0.07	0.10	0.30	3.56	0.85	0.19	0.41
N150	Amp	0.01	0.55	1.51	0.05	0.51	0.36	0.03
PT	Lat	0.00	0.23	0.23	0.67	0.13	1.45	0.13
P150	Amp	0.25	7.20*	0.07	0.56	0.96	0.69	0.46
SPF	Lat	0.05	0.01	6.81*	0.11	1.93	3.02	0.72
P250	Amp	0.00	4.69*	0.09	0.64	1.94	0.14	5.13*
OC	Lat	3.74	0.44	0.19	2.19	3.46	0.33	1.21
P250	Amp	0.01	24.63***	0.36	0.55	0.29	0.08	0.00
IPF	Lat	0.27	0.41	0.68	0.01	0.00	0.09	0.34
N300	Amp	0.01	43.46***	0.73	0.29	1.58	8.41**	2.39
PT	Lat	0.10	0.00	0.28	12.76**	1.00	0.04	0.20
P450	Amp	0.32	17.78***	0.28	0.86	0.88	0.00	0.46
SP	Lat	1.44	10.18**	2.30	0.35	1.05	1.90	0.78
N550	Amp	0.74	0.92	0.01	2.99	0.07	1.21	1.36
IPF	Lat	0.01	1.53	3.73	0.50	0.00	0.00	0.13
P700	Amp	1.82	17.83***	0.50	0.21	3.34	1.31	0.26
SP	Lat	1.10	0.25	4.00	0.17	1.01	0.57	2.65

[a] Values are $F[1,18]$, GP = group, TD = target detection, HS = hemisphere.

* $p<.05$, ** $p<.01$, *** $p<.001$, *2-tailed*; † $p<.05$, †† $p<.01$, ††† $p<.001$, *1-tailed*.

6.4.3.2 N80 ERP

The N80 peak was measured between 50-110 ms, it was largest over the superior frontal region at 80-90 ms (see Table 6-2, Figure 6-4 & Figure 6-6). ANOVA indicated no significant differences (see Table 6-3).

6.4.3.3 P80 ERP

The P80 peak was measured between 50-110 ms, it was largest over the posterior temporal region between 80-90 ms (see Table 6-2, Figure 6-4 & Figure 6-6). ANOVA

indicated a significant interaction of target detection and hemisphere in P80 amplitude (see Table 6-3). In the left posterior temporal region, there was larger mean amplitude for target than common words (M = 2.18 μV, SE = 0.44 μV, p<.001). Similarly, over the right posterior temporal region, there was larger mean amplitude for target than common words (M = 1.54 μV, SE = 0.37 μV, p<.01). There were no significant hemisphere differences for targets (M = 0.17 μV, SE = 0.37 μV, *ns*) or commons (M = -0.47 μV, SE = 0.31 μV, *ns*). Although the mean comparisons, after Bonferroni correction, indicate no significant interaction effect, the trend was toward greater condition differences in the left hemisphere.

6.4.3.4 N150 ERP

The N150 peak was measured between 100-200 ms, it was largest over the posterior temporal region between 160-165 ms (see Table 6-2, Figure 6-5 & Figure 6-7). ANOVA indicated no significant differences (see Table 6-3).

6.4.3.5 P150 ERP

The P150 peak was measured between 100-200 ms, it was largest over the superior prefrontal region between 150-160 ms (see Table 6-2, Figure 6-5 & Figure 6-7). ANOVA indicated a significant effect of target detection on P150 amplitude (see Table 6-3). There was larger mean amplitude for target than common words (M = 1.23 μV, SE = 0.47 μV, p<.05). Also, ANOVA indicated a significant interaction of target detection and group in P150 latency (see Table 6-3). However, mean comparisons after Bonferroni correction indicated no significant differences in target detection effects on P150 latency (CONT: M = 4.63 ms, SE = 2.41 ms, *ns*; PTSD: M = -4.25 ms, SE = 2.41 ms, *ns*).

6.4.3.6 P250 ERP

The P250 peak was measured between 210-270 ms, it was largest over the prefrontal and occipital regions between 230-250 ms (see Table 6-2, Figure 6-5 & Figure 6-8).

Occipital: ANOVA indicated a significant interaction of group by target detection by hemisphere in P250 amplitude (see Table 6-3). However, mean comparisons after Bonferroni correction indicated no significant target detection differences (CONT: left, $M = 0.75$ μV, $SE = 0.75$ μV, *ns*; right, $M = 1.18$ μV, $SE = 0.74$ μV, *ns*; PTSD: left, $M = 1.57$ μV, $SE = 0.75$ μV, *ns*; right, $M = 0.97$ μV, $SE = 0.74$ μV, *ns*). Similarly, there were no significant group differences (TARGET: left, $M = -0.91$ μV, $SE = 1.70$ μV, *ns*; right, $M = 0.66$ μV, $SE = 1.64$ μV, *ns*; COMMON: left, $M = 0.00$ μV, $SE = 1.64$ μV, *ns*; right, $M = 0.44$ μV, $SE = 1.71$ μV, *ns*). Thus, there was simply a significant main effect of target detection, where mean occipital P250 peak amplitude was larger for target than common words ($M = 1.12$ μV, $SE = 0.52$ μV, $p<.05$).

Inferior prefrontal: ANOVA indicated a significant target detection effect on P250 amplitude (see Table 6-3). The mean inferior prefrontal P250 amplitude was larger for target than common words ($M = 2.26$ μV, $SE = 0.46$ μV, $p<.001$).

6.4.3.7 N300 ERP

The N300 peak was measured between 250-350 ms, it was largest over the left posterior temporal region between 295-315 ms (see Table 6-2, Figure 6-4 & Figure 6-9). ANOVA indicated a significant interaction of target detection by hemisphere (see Table 6-3). Mean comparisons after Bonferroni correction indicated that there was larger mean N300 amplitude for target than common words in both left and right posterior temporal regions (LEFT: $M = -4.19$ μV, $SE = 0.64$ μV, $p<.001$; RIGHT: $M = -2.62$ μV, $SE = 0.53$ μV, $p<.001$). After Bonferroni correction, there was a near significant hemisphere difference for target words, with larger amplitude over the left

posterior temporal region (TARGET: M = -1.08 μV, SE = 0.62 μV, p<.1; COMMON: M = 0.49 μV, SE = 0.59 μV, *ns*). Also, ANOVA of N300 peak latency indicated a significant difference across hemispheres (see Table 6-3). The N300 peak arose earlier over the left than the right posterior temporal region (M = -8.69 ms, SE = 2.43 ms, p<.01).

There was a positive linear trend between N300 amplitude and target reaction time [RT; $r(17) = 0.43$; $\beta = 12.91 \pm 7.16$, $F(17) = 1.80$, p<.1; $R^2 = 0.18$]. The linear regression equation is: mean(RT) = 578.28 + 12.91(N300 amplitude). That is, for each unit *decrease* in the mean N300 amplitude, there is a mean reaction time increase of 12.91 ms (numeric increase of a negative potential is a *decrease* in *absolute* N300 amplitude, so this relationship indicates quicker reaction time with larger N300). No causality is implied in this association.

6.4.3.8 P450 ERP

The P450 peak was measured between 250-625 ms, it was largest over the superior parietal region between 370-470 ms (see Table 6-2, Figure 6-5 & Figure 6-10). ANOVA indicated a significant target detection effect on P450 amplitude and latency (see Table 6-3). There was larger mean amplitude for target than common words (M = 5.31 μV, SE = 1.26 μV, p<.01). The mean P450 peak latency was later for target than common words (M = 49.81 ms, SE = 15.61 ms, p<.01).

6.4.3.9 N550 ERP

The N550 peak was measured between 400-625 ms, it was largest over the inferior prefrontal region between 430-525 ms (see Table 6-2, Figure 6-5 & Figure 6-11). ANOVA indicated no significant differences (see Table 6-3).

6.4.3.10 P700 ERP

The P700 peak was measured between 600-800 ms, it was largest over the superior parietal region between 665-725 ms (see Table 6-2, Figure 6-5 & Figure 6-12). ANOVA indicated a significant target detection effect on P700 amplitude (see Table 6-3). There was larger mean P700 peak amplitude for target than common words (M = 4.27 μV, SE = 1.01 μV, p<.01).

There was a significant positive linear relationship between P700 latency and target reaction time [RT; r[17] = 0.59; β = 0.96 ± 0.28, F(17) = 3.44, p<.01]. The linear regression equation is: mean(RT) = -157.18 + 0.96(P700 latency). That is, for each ms increase in the mean P700 latency, there is a mean reaction time increase of 0.96 ms (no causality is implied in this association).

6.4.3.11 Summary of ERP Component Findings

There were no significant group differences in the ERP components. The target words elicit:

- larger posterior temporal P80 amplitude,
- larger superior prefrontal P150 amplitude,
- larger occipital and inferior prefrontal P250 amplitude,
- larger posterior temporal N300 amplitude (with decreases in absolute N300 amplitude associated with increased target reaction time),
- greater superior parietal P450 amplitude and latency, and
- larger superior parietal P700 amplitude (with P700 latency positively associated with target reaction time).

6.4.4 ERP Difference Waves

The difference waves demonstrate two clear periods of divergence (see Figure 6-13). The following difference wave components were identified for further analyses

(see also Figure 6-14 to Figure 6-16), where the initial letter indicates polarity and the following number indicates the peak latency:

- left posterior temporal ND300
- prefrontal PD350
- parietal PD500
- frontal ND650
- parietal PD700

Summary statistics for these components are given in Table 6-4 and the inferential analyses are described below (see Table 6-5), with the mean differences for significant effects.

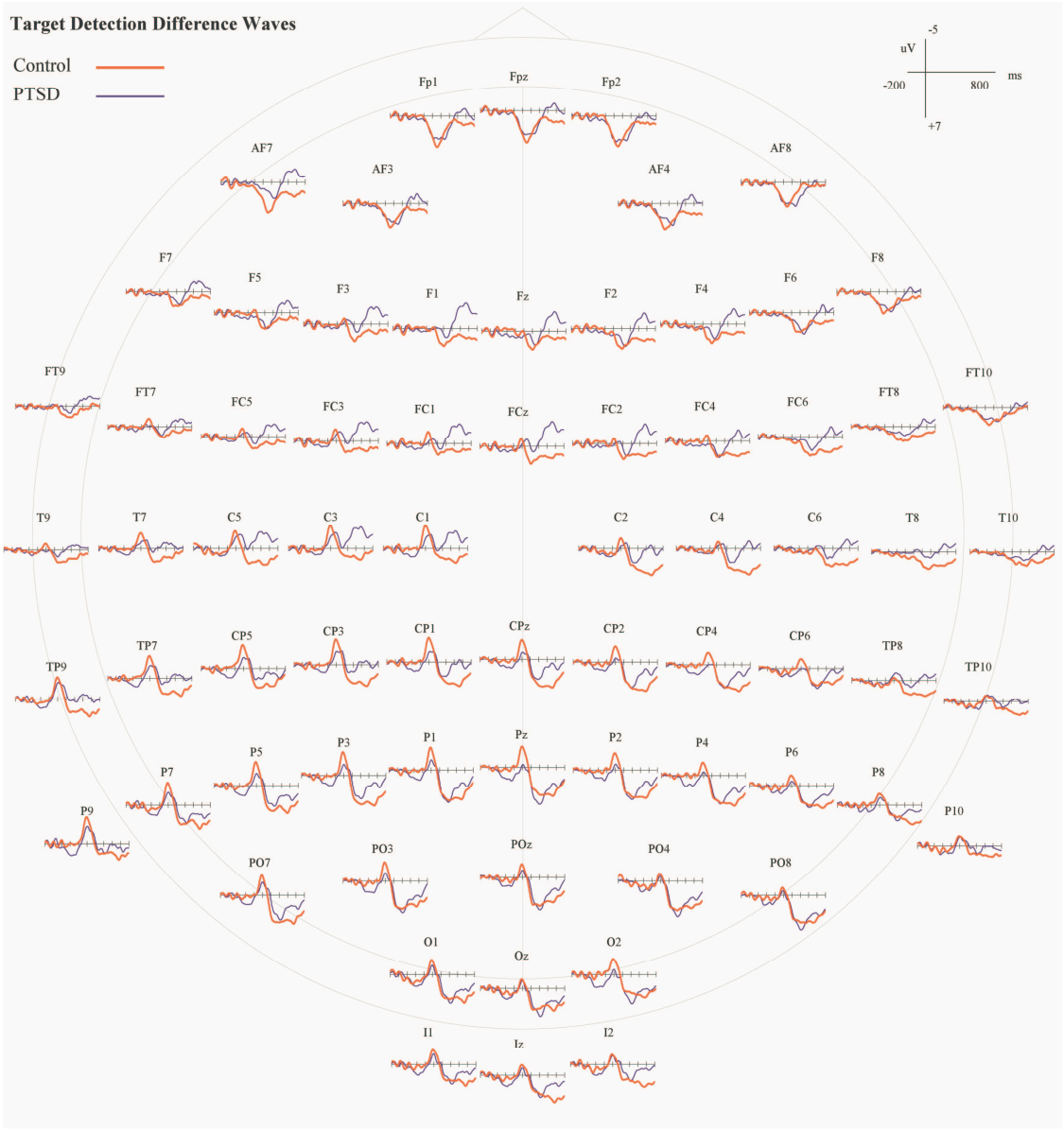

Figure 6-13. Target detection ERP difference waves for controls (n = 10) and PTSD patients (n = 10). Waveforms are given at 70 representative scalp sites (-200 to 800 ms, 100 ms intervals). Controls demonstrate larger positive difference wave components over parietal areas at 400-800 ms, whereas PTSD patients have larger negative difference wave potentials over left fronto-central areas at 400-800 ms.

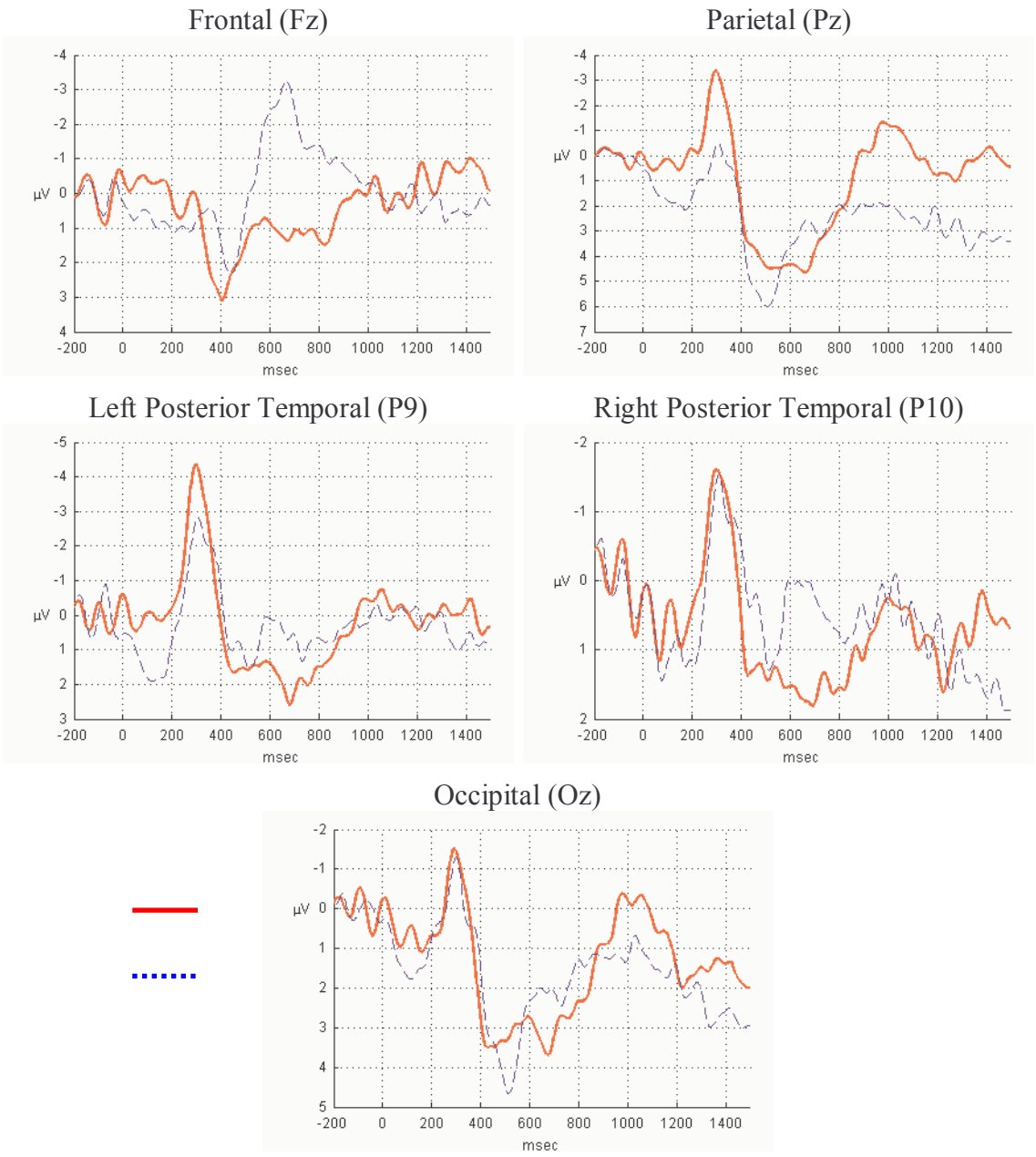

Figure 6-14. Target detection ERP difference waveforms for controls (n = 10; red, solid) and PTSD patients (n = 10; blue, dash). There is larger ND300 in controls than PTSD patients at the left posterior temporal site. The following PD500 is larger for PTSD patients at parietal and occipital sites, while the following PD700 is larger for controls at all sites illustrated, but especially the frontal and posterior temporal sites. Note how the frontal PD400 precedes the posterior PD500/PD700 peaks. Also, patients demonstrate a large frontal ND650 peak, at the time when controls demonstrate continued positive activity. See the topography of these components below.

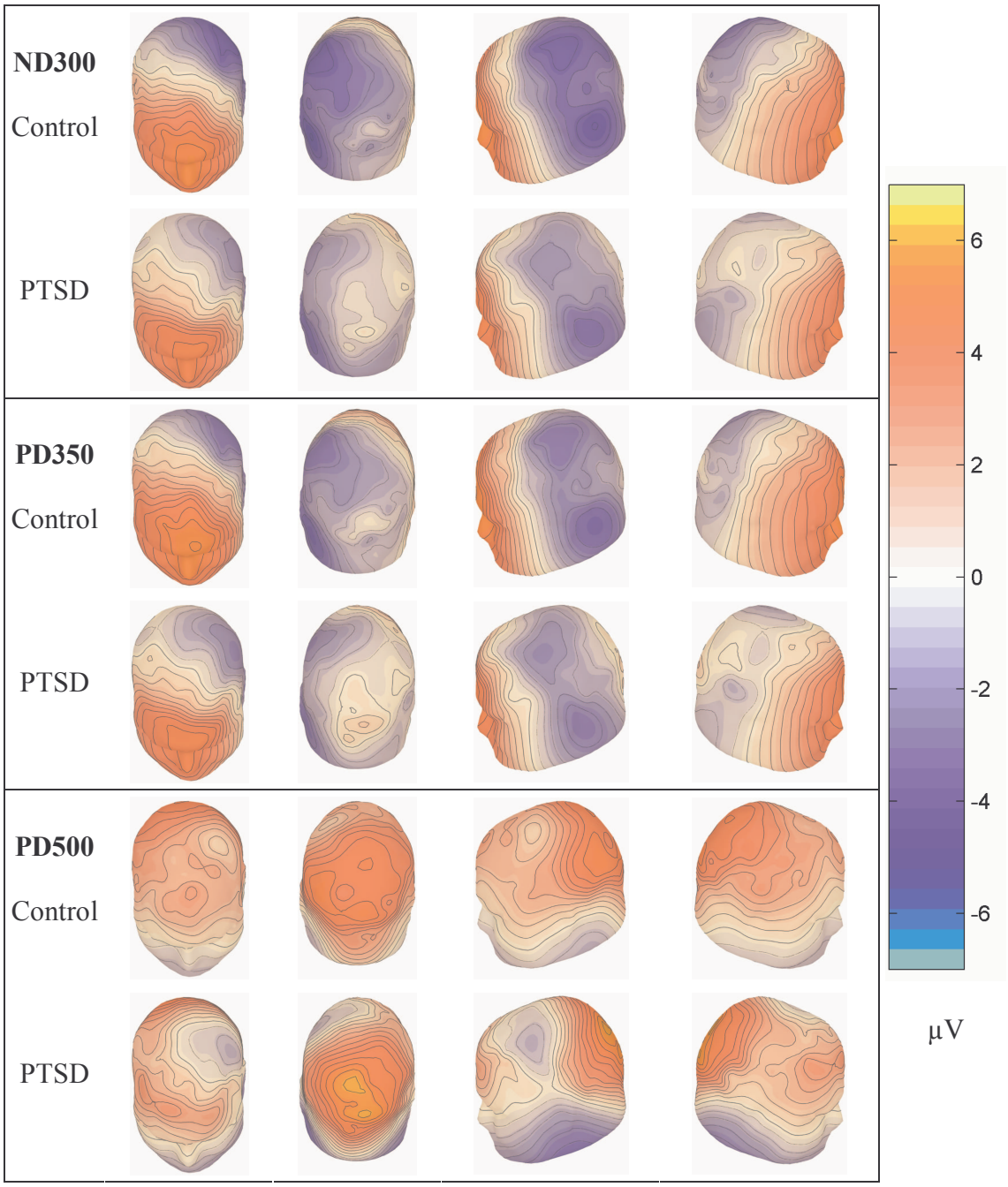

Figure 6-15. ERP difference wave topography for controls (n = 10) and PTSD patients (n = 10; 0.5 μV contours). The ND300 is given at 320 ms, the PD350 is given at 340 ms and the PD500 is given at 495 ms. All maps are given on the same scale.

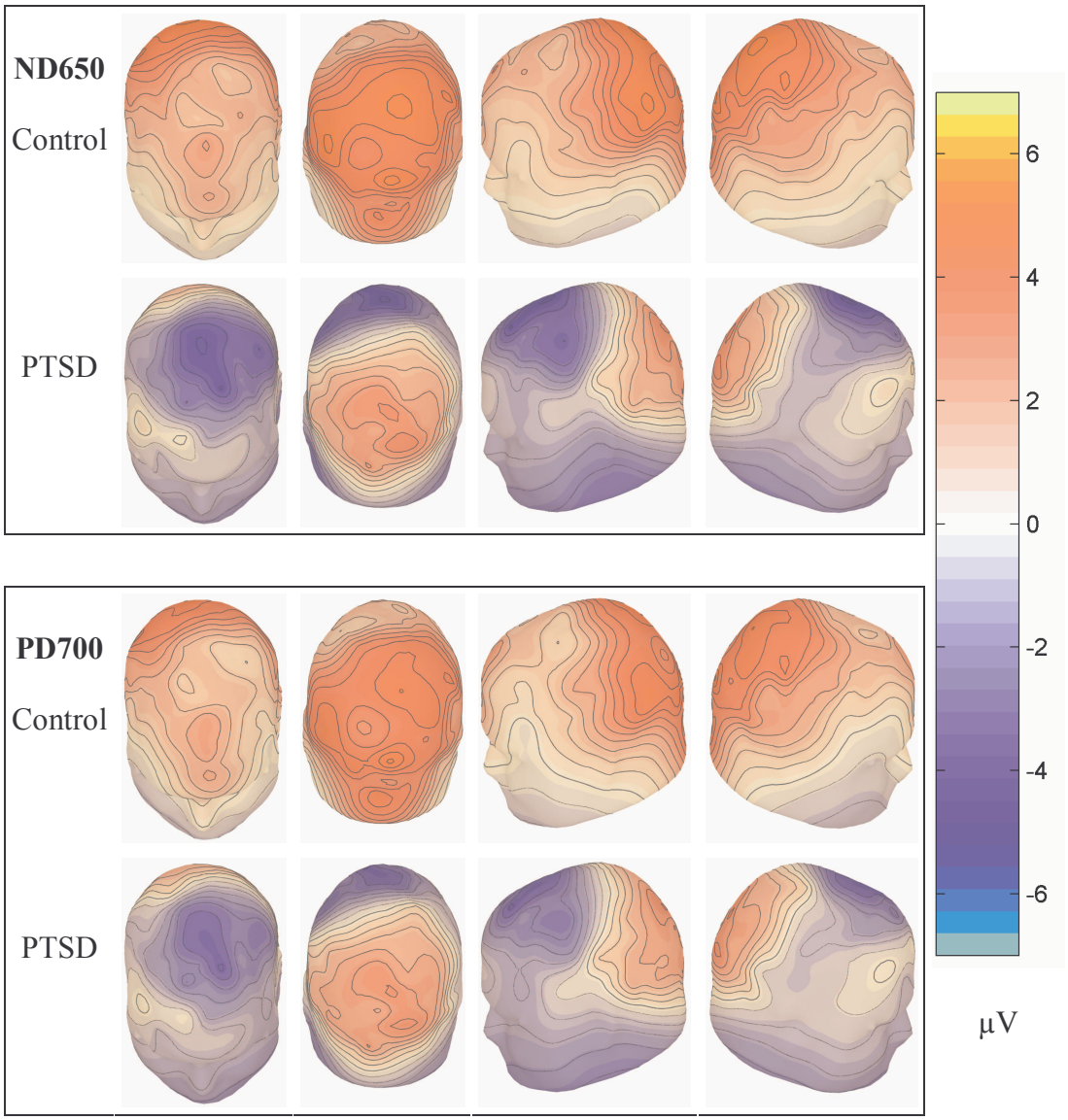

Figure 6-16. ND650 and PD700 ERP topography for controls (n = 10) and PTSD patients (n = 10) for the target detection difference wave (contours at 0.5 µV intervals). The ND650 is given at 650 ms and the PD700 is given at 700 ms. All maps are given on the same scale.

Table 6-4. Summary Statistics for Target Detection ERP Difference Components [a]

		Amplitude (μV)		Latency (ms)	
		CONT	PTSD	CONT	PTSD
ND300	Left	-6.39 (2.81)	-4.12 (2.18)	314.75 (33.53)	330.25 (38.68)
PT	Right	-3.89 (1.72)	-3.66 (2.13)	315.25 (51.85)	341.00 (37.18)
PD350	Left	7.68 (3.18)	6.03 (2.25)	342.75 (34.06)	329.75 (36.71)
IPF	Right	6.99 (2.58)	6.23 (2.45)	330.00 (31.99)	340.50 (35.68)
PD500	Left	7.19 (5.32)	7.19 (4.14)	488.25 (49.03)	486.00 (41.95)
OC	Right	7.30 (5.47)	8.00 (4.88)	493.75 (51.82)	492.75 (48.44)
PD500	Left	6.55 (6.58)	8.00 (5.66)	500.00 (38.69)	485.75 (45.87)
SP	Right	6.88 (6.89)	7.30 (5.77)	505.50 (47.40)	494.50 (39.82)
ND650	Left	-3.67 (6.24)	-6.97 (3.96)	612.00 (109.17)	605.00 (99.90)
SF	Right	-2.64 (5.98)	-5.70 (2.78)	647.25 (110.04)	648.75 (117.23)
PD700	Left	7.12 (4.54)	4.15 (4.73)	678.50 (46.64)	718.00 (34.88)
IP	Right	5.75 (4.06)	4.05 (2.77)	700.00 (43.04)	720.50 (38.11)
PD700	Left	4.46 (3.92)	0.58 (4.43)	691.75 (51.98)	743.75 (31.58)
SF	Right	5.60 (3.81)	1.56 (3.72)	687.75 (56.64)	727.25 (47.21)

[a] Target - common words; CONT, n = 10; PTSD, n = 10; values are mean (SD).

Table 6-5. Inferential Statistics for Target Detection ERP Difference Components [a]

ERP		GP	HS	GPxHS
ND300	Amp	2.00	9.82**	4.63*
PT	Lat	1.71	0.37	0.31
PD350	Amp	1.09	0.87	3.06
IPF	Lat	0.01	0.03	3.79
PD500	Amp	0.03	0.97	0.56
OC	Lat	0.01	3.12	0.03
PD500	Amp	0.12	0.19	1.50
SP	Lat	0.46	1.78	0.09
ND650	Amp	2.22	3.63	0.04
SF	Lat	0.00	7.99*	0.09
PD700	Amp	1.76	2.16	1.59
IP	Lat	2.89	6.20*	3.88
PD700	Amp	5.40*	4.20	0.02
SF	Lat	5.46*	1.43	0.53

[a] Values are $F[1,18]$, GP = group, HS = hemisphere.

* $p<.05$, ** $p<.01$, *** $p<.001$, *2-tailed*; † $p<.05$, †† $p<.01$, ††† $p<.001$, *1-tailed*.

6.4.4.1 ND300 ERP

The ND300 peak was measured between 220-420 ms, it was largest over the posterior temporal region between 300-350 ms (see Table 6-4, Figure 6-14 & Figure 6-15). ANOVA indicated a significant group by hemisphere interaction in ND300 amplitude (see Table 6-5). Over the left posterior temporal region, there was an indication of larger mean ND300 amplitude in controls than PTSD patients (M = -2.27 μV, SE = 1.12 μV, $p<.05$, *1-tailed*). There was no significant group difference over the right posterior temporal region (M = -0.23 μV, SE = 0.87 μV, *ns*). Also, for controls, there was larger mean ND300 peak amplitude over the left than the right posterior temporal region (M = -2.50 μV, SE = 0.67 μV, $p<.01$). For PTSD patients, there was no significant difference in mean ND300 peak amplitude between the left and right posterior temporal regions (M = -0.47 μV, SE = 0.67 μV, *ns*).

There was a positive linear trend between ND300 amplitude and target reaction time [RT; $r[17] = 0.43$; $\beta = 17.51 \pm 9.40$, $F(17) = 1.86$, $p<.1$]. The linear regression equation is: mean(RT) = 606.68 + 17.51(ND300 amplitude). That is, for each unit *decrease* in the mean ND300 amplitude, the reaction time increases by 17.51 ms (because numeric increases of a negative potential actually mean decreased absolute amplitude of the negative ND300 peak, this relationship indicates faster reactions for larger ND300 peaks). No causality is implied in this association.

6.4.4.2 PD350 ERP

The PD350 peak was measured between 250-400 ms, it was largest over the inferior prefrontal region between 320-350 ms (see Table 6-4, Figure 6-14 & Figure 6-15). ANOVA indicated no significant differences (see Table 6-5).

6.4.4.3 PD500 ERP

The PD500 peak was measured between 420-580 ms, it was largest over the occipital and superior parietal regions between 480-510 ms (see Table 6-4, Figure 6-14 & Figure 6-15). ANOVA indicated no significant differences over the occipital or the superior parietal regions (see Table 6-5).

6.4.4.4 ND650 ERP

The ND650 peak was measured between 450-800 ms, it was largest over the left superior frontal region between 600-650 ms (see Table 6-4, Figure 6-14 & Figure 6-16). ANOVA indicated a significant hemisphere difference in mean ND650 latency (see Table 6-5). The mean ND650 peak latency was earlier over the left than the right superior frontal region (M = -39.50 ms, SE = 13.98 ms, $p<.05$).

6.4.4.5 PD700 ERP

Inferior parietal: The PD700 peak was measured between 620-800 ms, it was largest over the inferior parietal region between 675-725 ms (see Table 6-4, Figure 6-14 & Figure 6-16). ANOVA indicated a significant hemisphere difference in mean PD700 latency (see Table 6-5). The mean ND700 peak latency was shorter over the left than the right inferior parietal region (M = -12.00 ms, SE = 4.82 ms, p<.05). There was a positive linear relationship between PD700 latency and target reaction time [RT; $r(17)$ = 0.64; $\beta = 1.28 \pm 0.40$, $F(17) = 3.23$, p<.01; $R^2 = 0.41$]. The linear regression equation is: mean(RT) = -370.19 + 1.28(PD700 latency). That is, for each ms increase in the mean PD700 latency, the reaction time increases by 1.28 ms (no causality is implied in this relationship).

Superior frontal: There was also a large PD700 peak over the superior frontal region between 685-745 ms (see Table 6-4, Figure 6-14 & Figure 6-16). ANOVA indicated a significant group difference in mean PD700 amplitude and latency (see Table 6-5). There was larger mean PD700 peak amplitude in controls than PTSD patients (M = 3.96 μV, SE = 1.70 μV, p<.05). The mean ND700 peak latency was shorter in controls than PTSD patients (M = -45.75 ms, SE = 19.58 ms, p<.05). There was a positive linear relationship between PD700 latency and target reaction time [RT; $r(17)$ = 0.63; $\beta = 1.08 \pm 0.35$, $F(17) = 3.12$, p<.01; $R^2 = 0.40$]. The linear regression equation is: mean(RT) = -240.27 + 1.08(PD700 latency). That is, for each ms increase in the PD700 latency, the mean reaction time increases by 1.08 ms (no causality is implied in this relationship).

6.4.4.6 Summary of ERP Difference Component Findings

The left posterior temporal ND300 was larger in controls than PTSD and the superior frontal PD700 amplitude and latency were larger and earlier in controls than

PTSD. There was a linear association of both ND300 amplitude and PD700 latency with target reaction time.

6.4.5 Scalp Current Density Components

6.4.5.1 Group Means

The topographic layout of SCD waveforms is illustrated in Figure 6-17 and Figure 6-18, while the superimposed waveforms are given in Figure 6-19. The following components were identified for further analyses, where the initial letter indicates polarity and the following number indicates the peak latency:

- P150 over the parietal and anterior temporal regions
- posterior temporal N200
- occipital and prefrontal P250
- fronto-central N300
- P350 and P450 over the occipital, parietal and frontal regions
- frontal N600

Summary statistics for these components are given in Table 6-6 and the inferential analyses are described below (see Table 6-7), with the mean differences for significant effects.

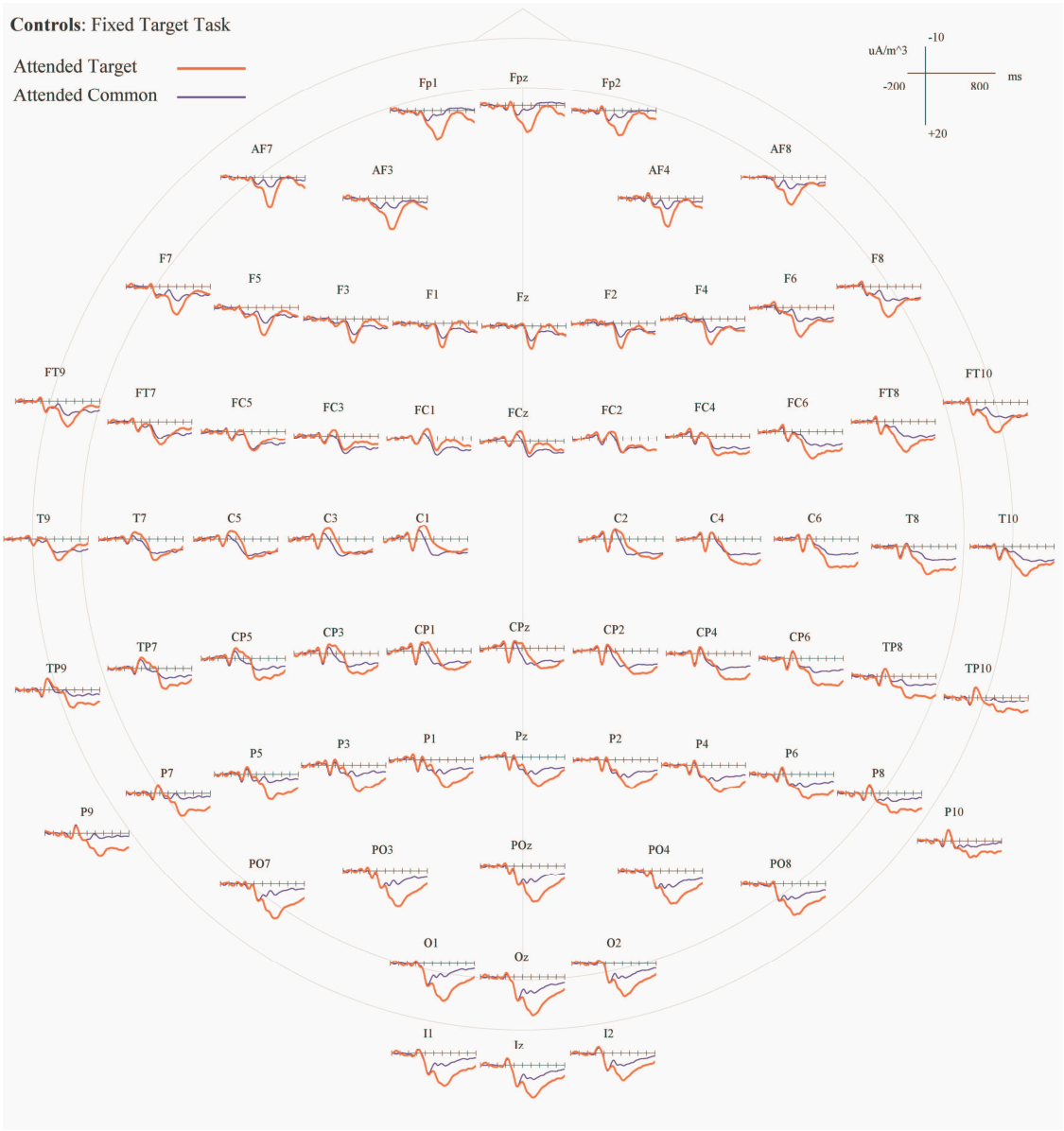

Figure 6-17. Event-related SCD in controls (n=10) at 70 scalp sites (-200 to 800 ms, 100 ms intervals). Attended target words in the fixed target task elicit larger positive SCD over the occipital, temporal, parietal and prefrontal regions at 400-800 ms.

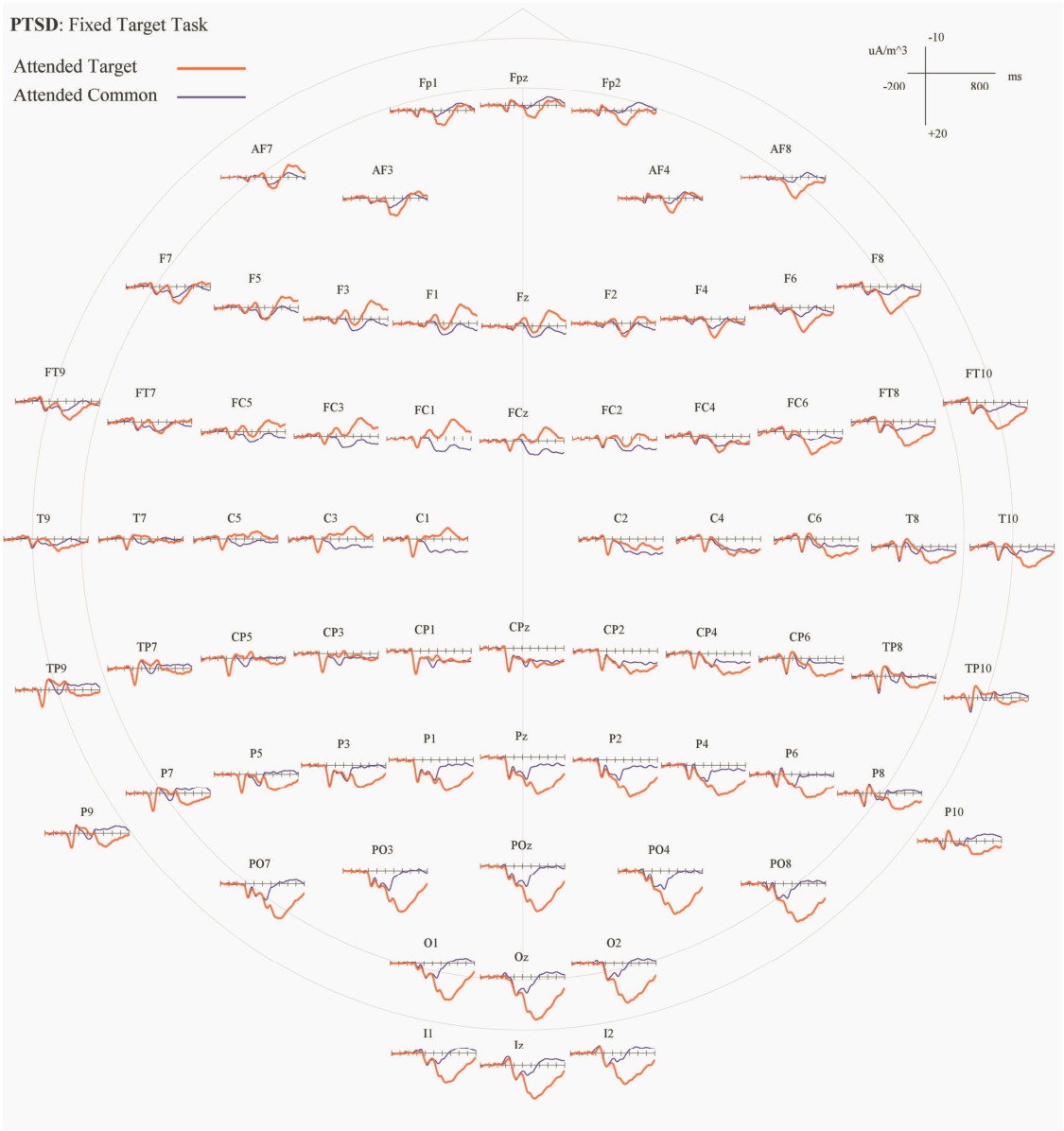

Figure 6-18. Event-related SCD in PTSD (n=10) at 70 scalp sites (-200 to 800 ms, 100 ms intervals). Attended target words in the fixed target task elicit larger positive SCD over the occipital, temporal, parietal and prefrontal regions at 400-800 ms. At the same time, there is greater negative SCD over medial frontal areas.

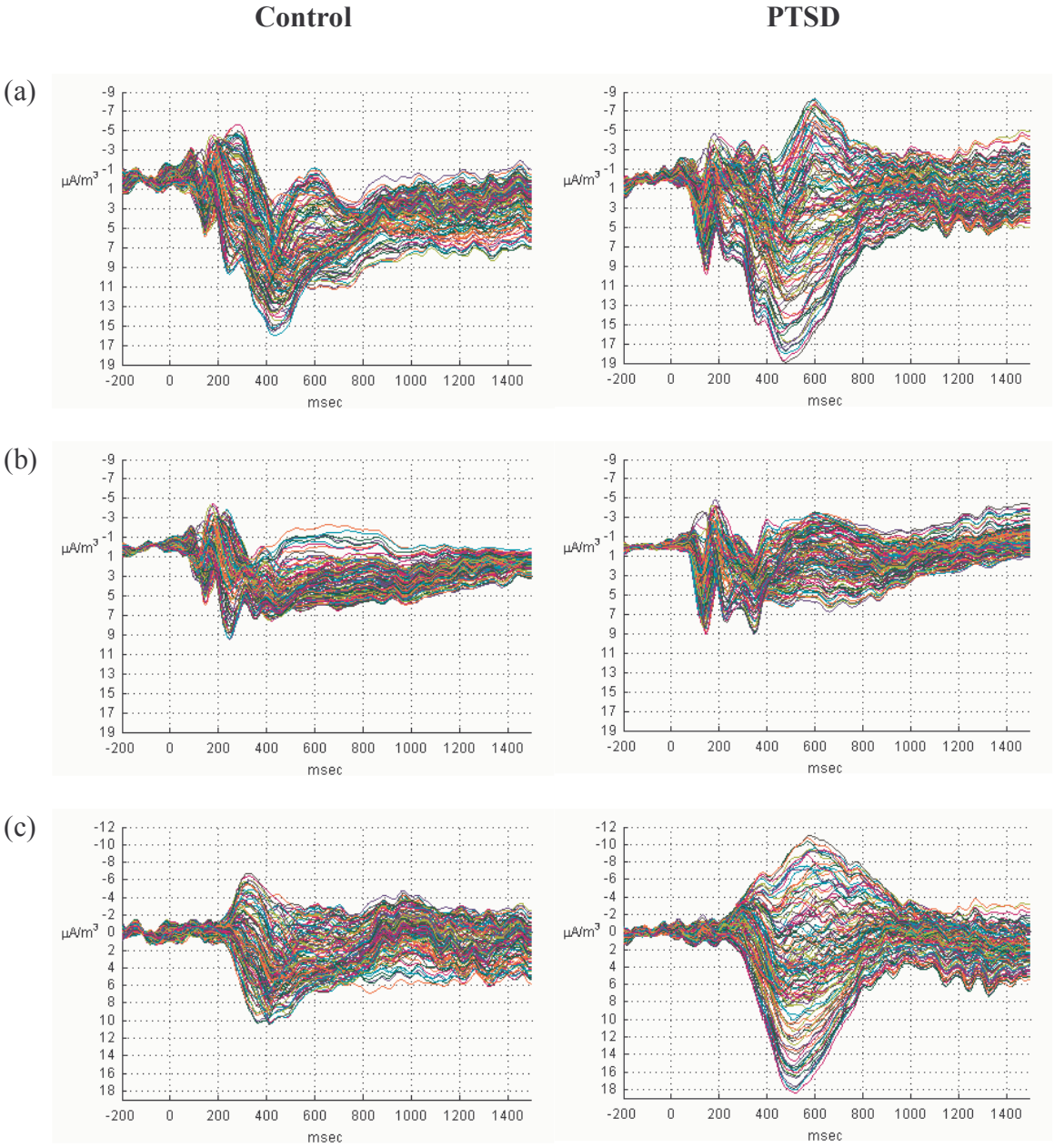

Figure 6-19. SCD waveforms for attended conditions of the fixed target task at 124 scalp sites for controls (n=10) and PTSD patients (n=10): (a) target, (b) common, (c) target – common.

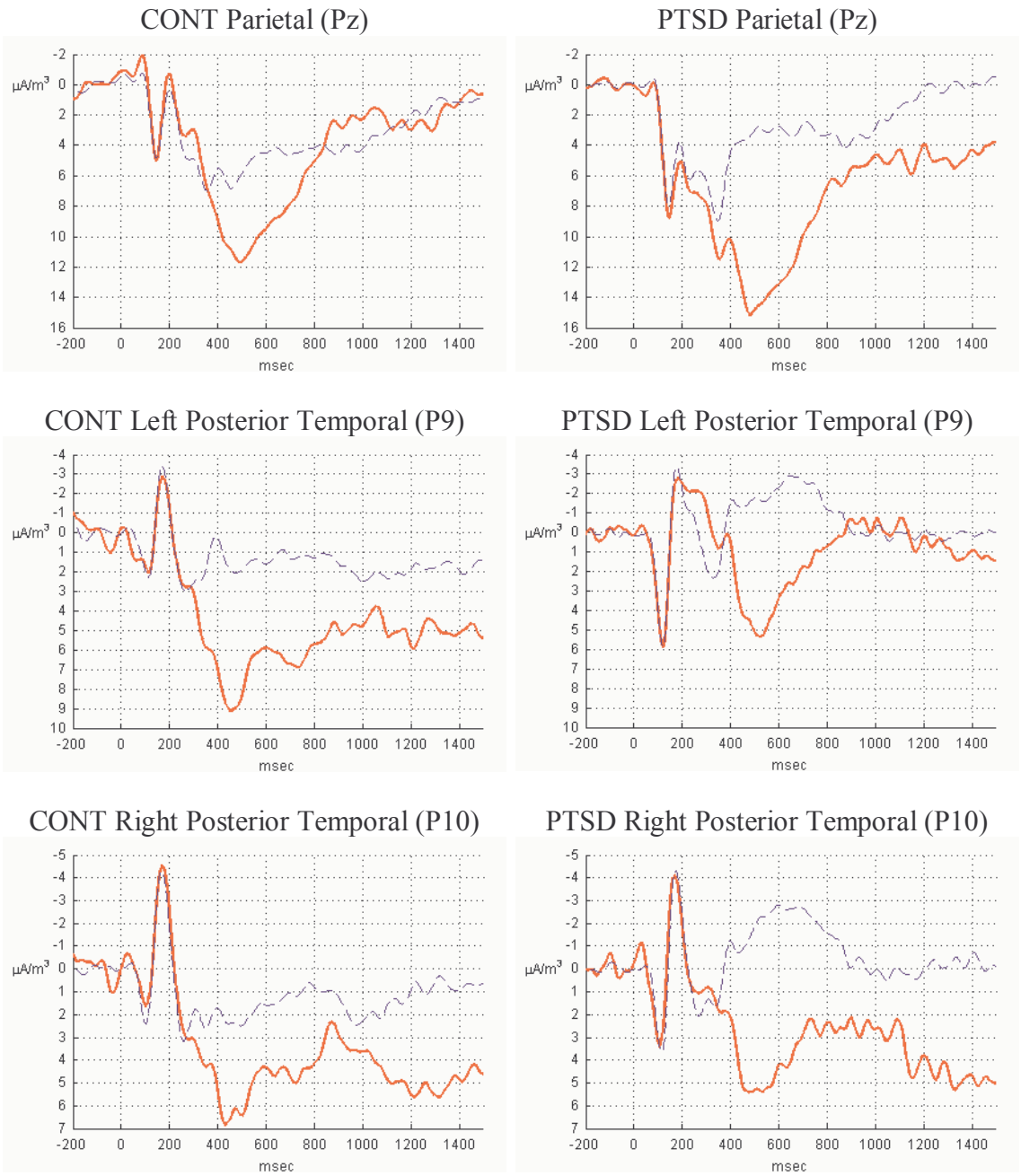

Figure 6-20. SCD waveforms for controls (n = 10) and PTSD patients (n = 10) at parietal and posterior temporal sites for both attended target (red, solid) and attended common words (blue, dash). Note the P150 at parietal and posterior temporal sites, which is larger for PTSD patients than controls. This was followed by the N200 at posterior temporal sites, with similar amplitude for both groups. Also apparent here are the P450 peaks; patients had larger P450 over the right than the left, while controls had the opposite. See the topography of the components below.

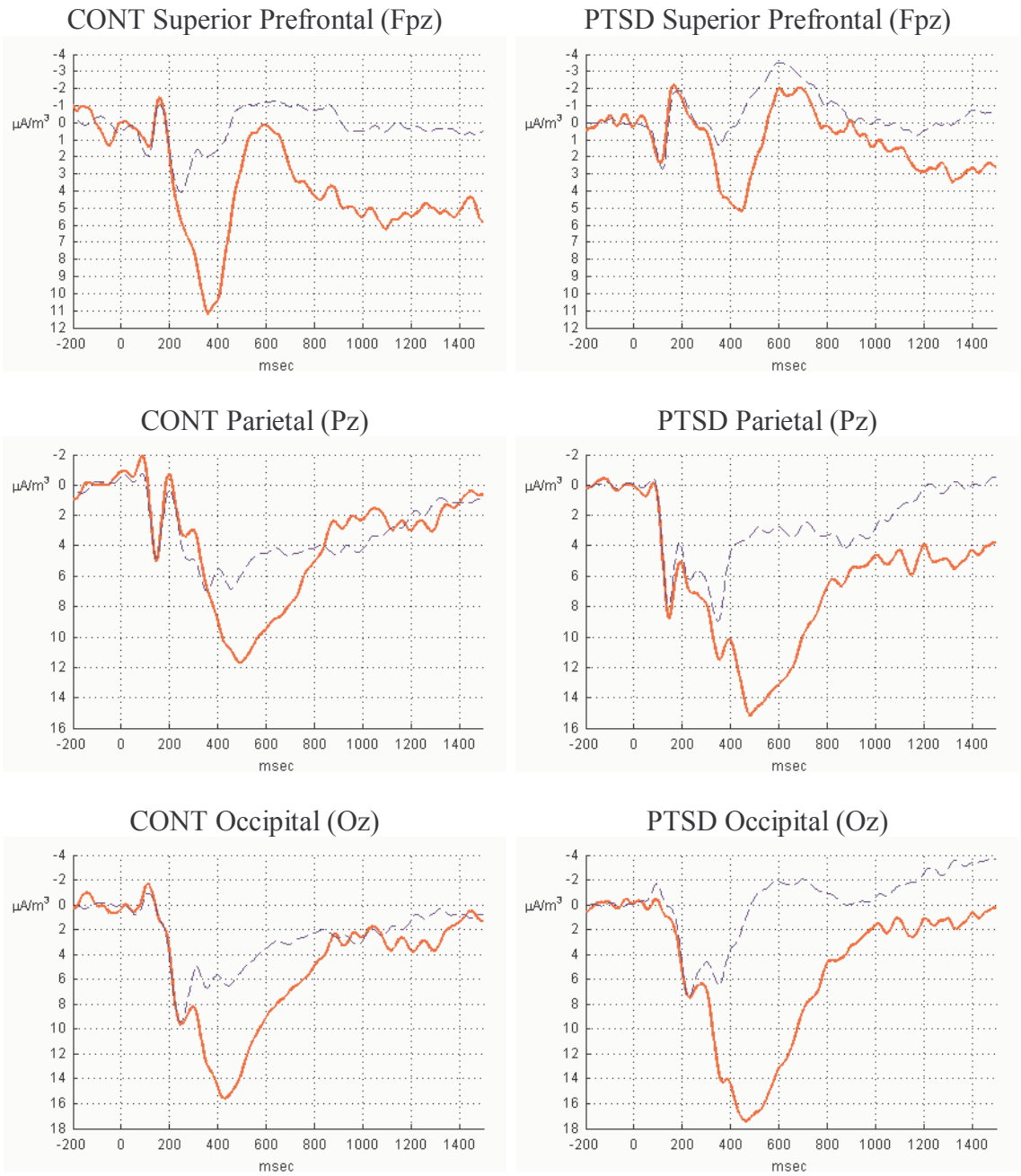

Figure 6-21. SCD waveforms for controls (n = 10) and PTSD patients (n = 10) at prefrontal, parietal and occipital sites for both attended targets (red, solid) and common words (blue, dash). There are several features to note: (a) the occipital P250, followed by a small N300 and the larger P450, (b) the prefrontal P350 and the parietal P450, which are larger for targets, and (c) the prefrontal N600. Generally, controls demonstrate similar amplitude of frontal and parietal P450, with earlier onset of frontal than parietal activity, while the PTSD patients demonstrate much greater parietal than frontal P450. There is also an extended prefrontal target positivity at 600–1500 ms in controls that is smaller in PTSD patients. See the topography of the components below.

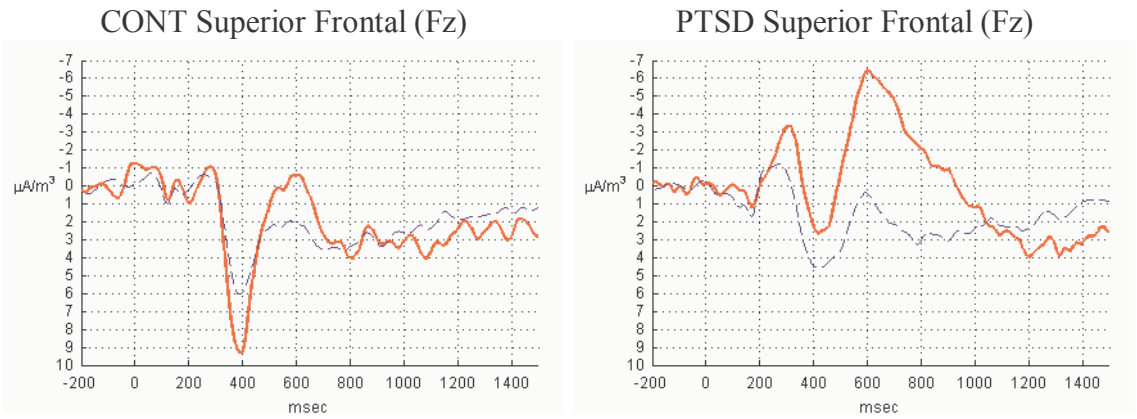

Figure 6-22. SCD waveforms for controls (n = 10) and PTSD patients (n = 10) at superior frontal sites for both attended targets (red, solid) and common words (blue, dash). Note the N300, followed by the larger P400 and the N600. Generally, controls demonstrate greater P400 than PTSD patients, while the PTSD patients demonstrate greater frontal N300 and N600 than controls. See the component topography below.

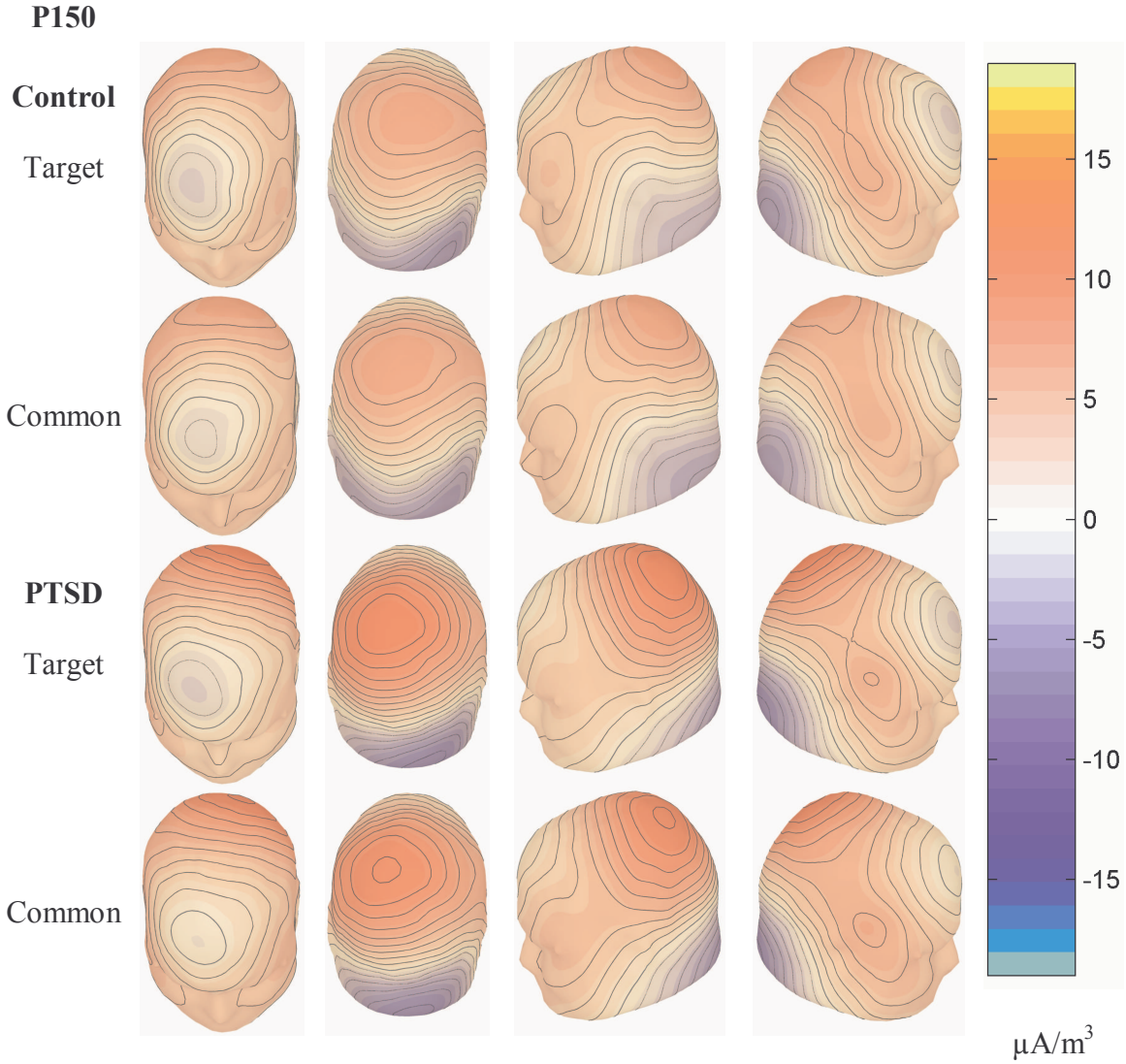

Figure 6-23. P150 SCD topography at 145 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The P150 peaks over the superior parietal region. Note also the negative peaks at occipital regions, which develop into the posterior temporal N200 SCD.

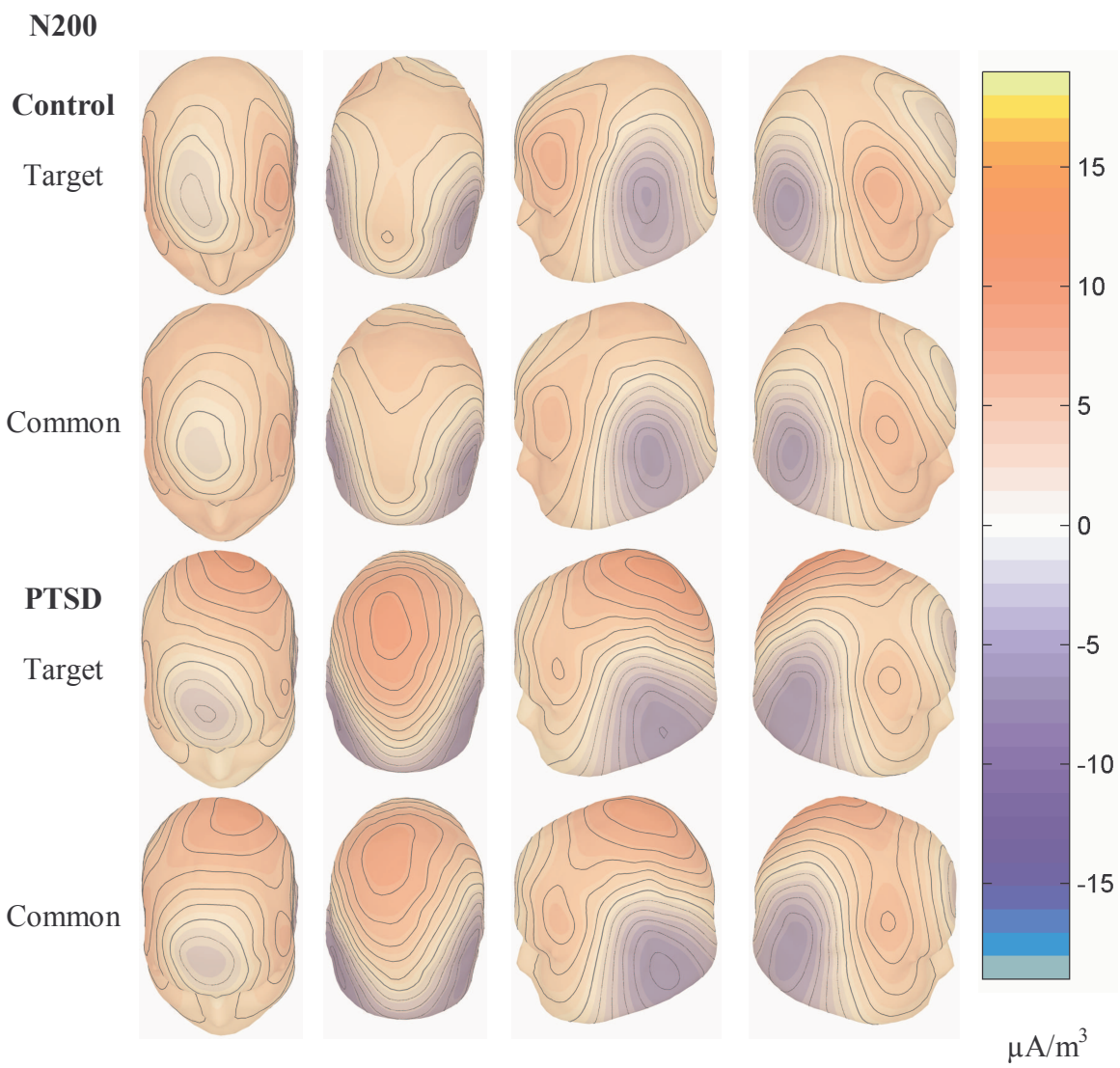

Figure 6-24. N200 SCD topography at 175 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The N200 peaks over the posterior temporal region.

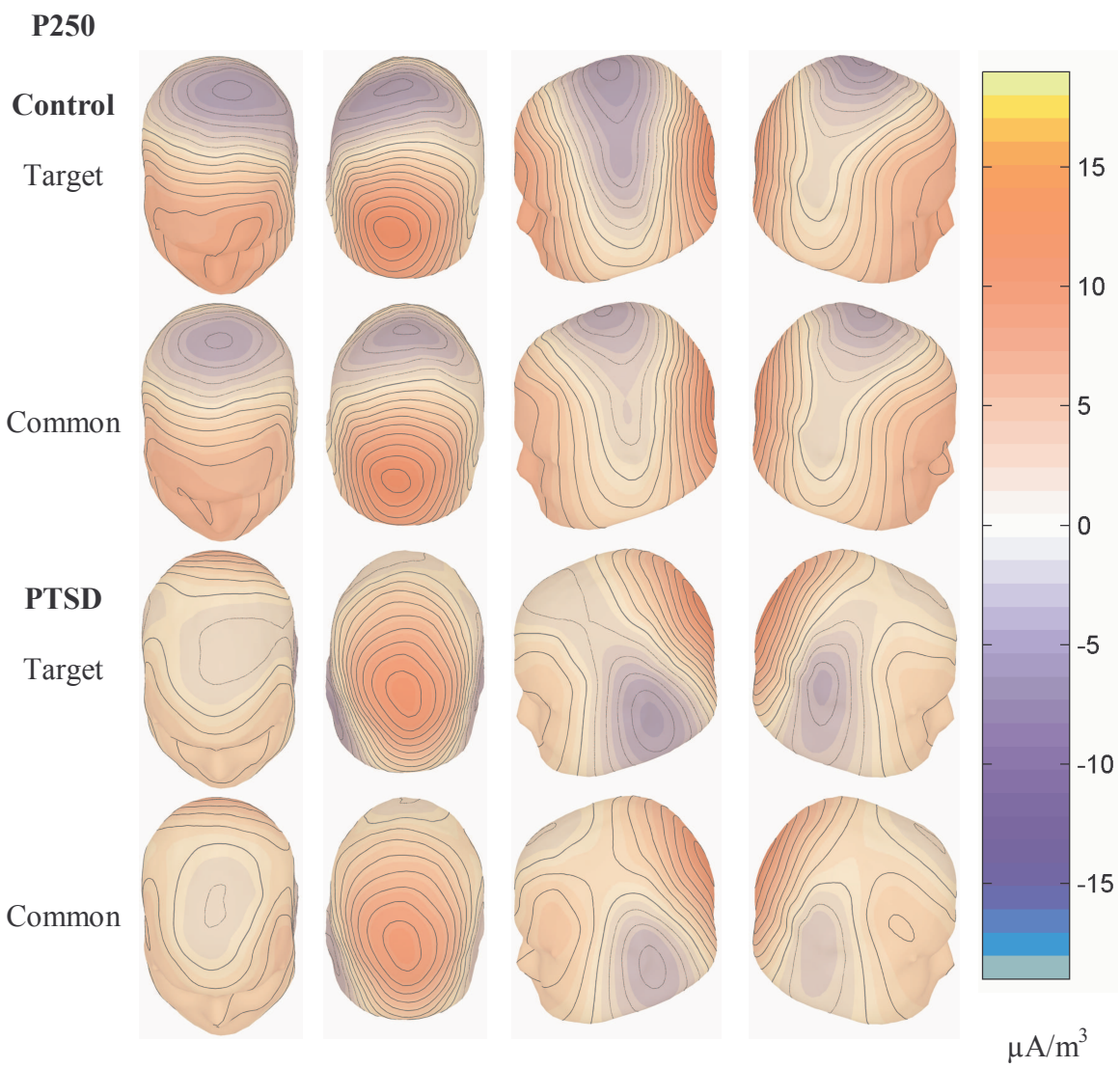

Figure 6-25. P250 SCD topography at 240 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The P250 peaks over the occipital region, with another smaller peak in the inferior prefrontal region. The negative peaks here are maximal in the N300 (see below).

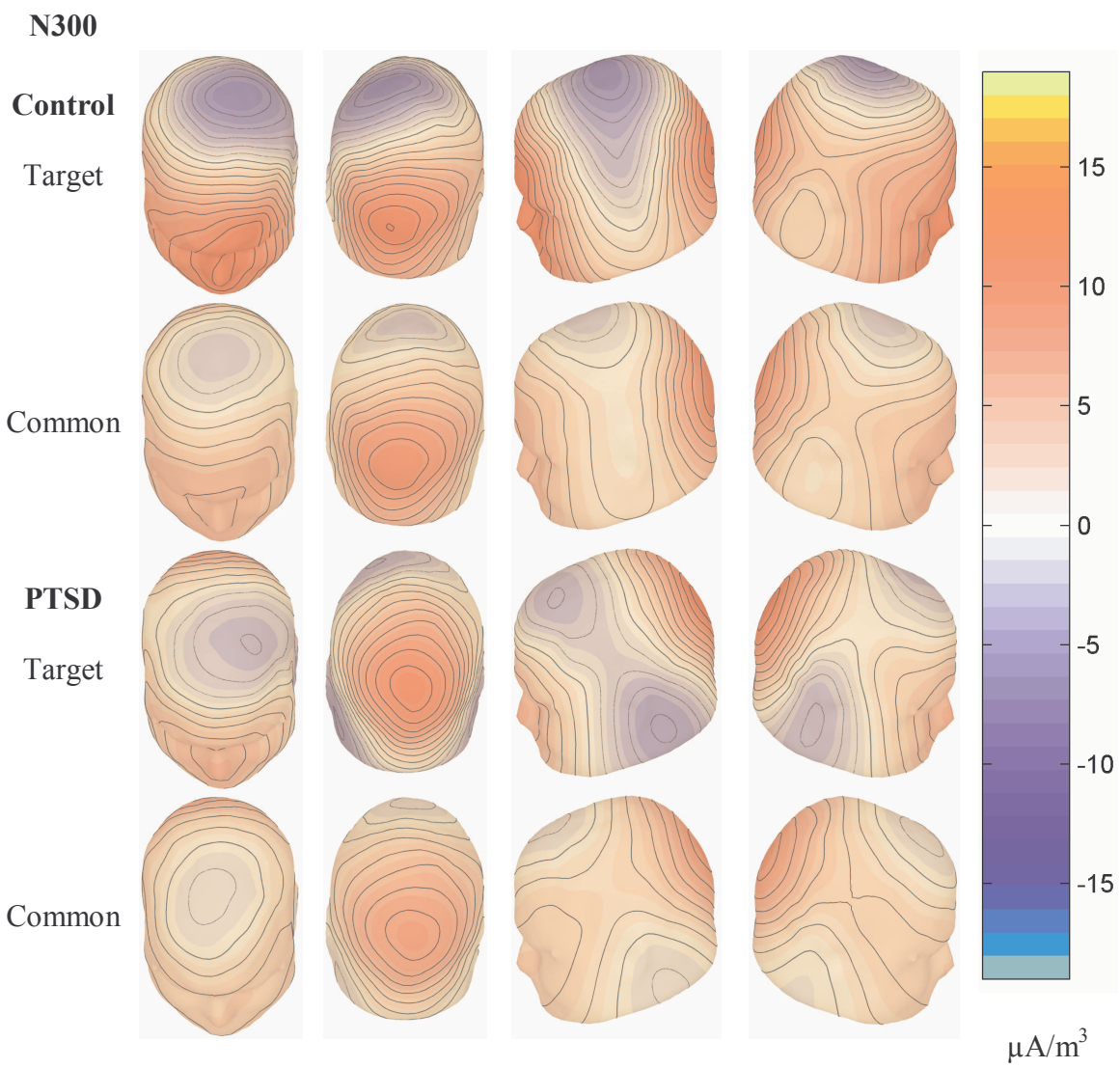

Figure 6-26. N300 SCD topography at 275 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The N300 peaks over the superior fronto-central region.

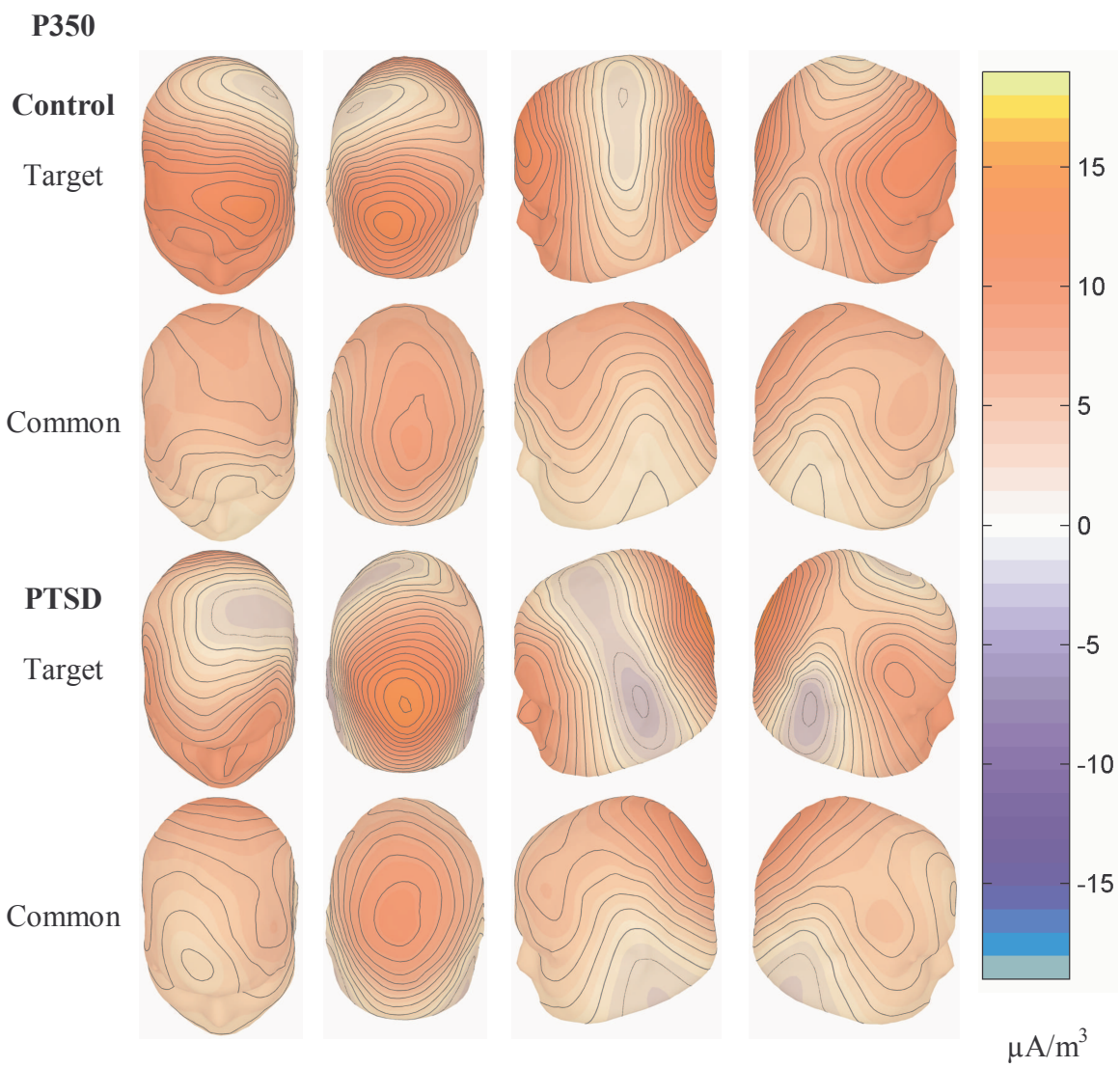

Figure 6-27. P350 SCD topography at 360 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The P350 peaks at the occipital and prefrontal regions (similar to P250).

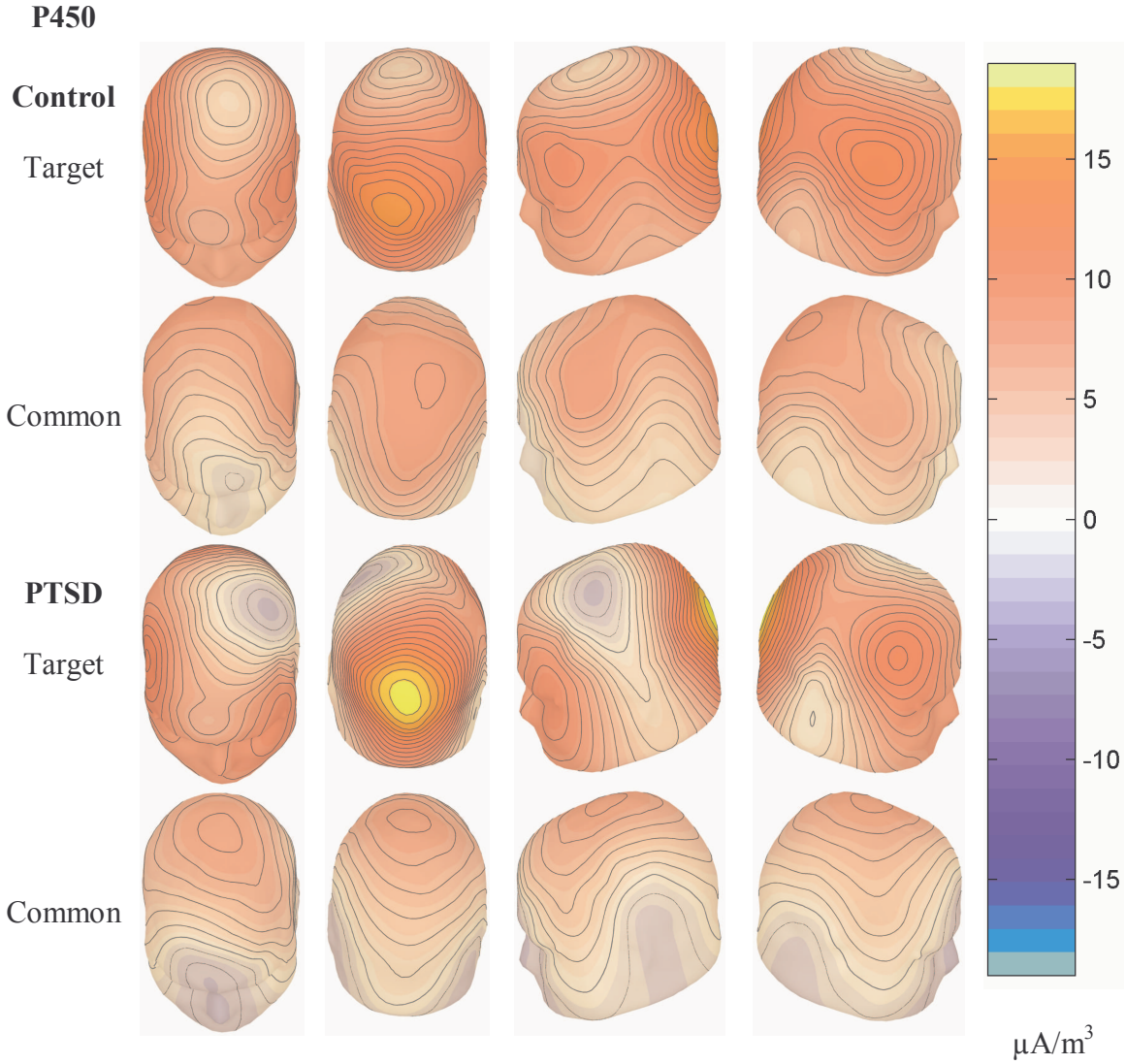

Figure 6-28. P450 SCD topography at 460 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The P450 peaks at the occipital region, with smaller peaks at lateral frontal regions (similar to P250 and P350).

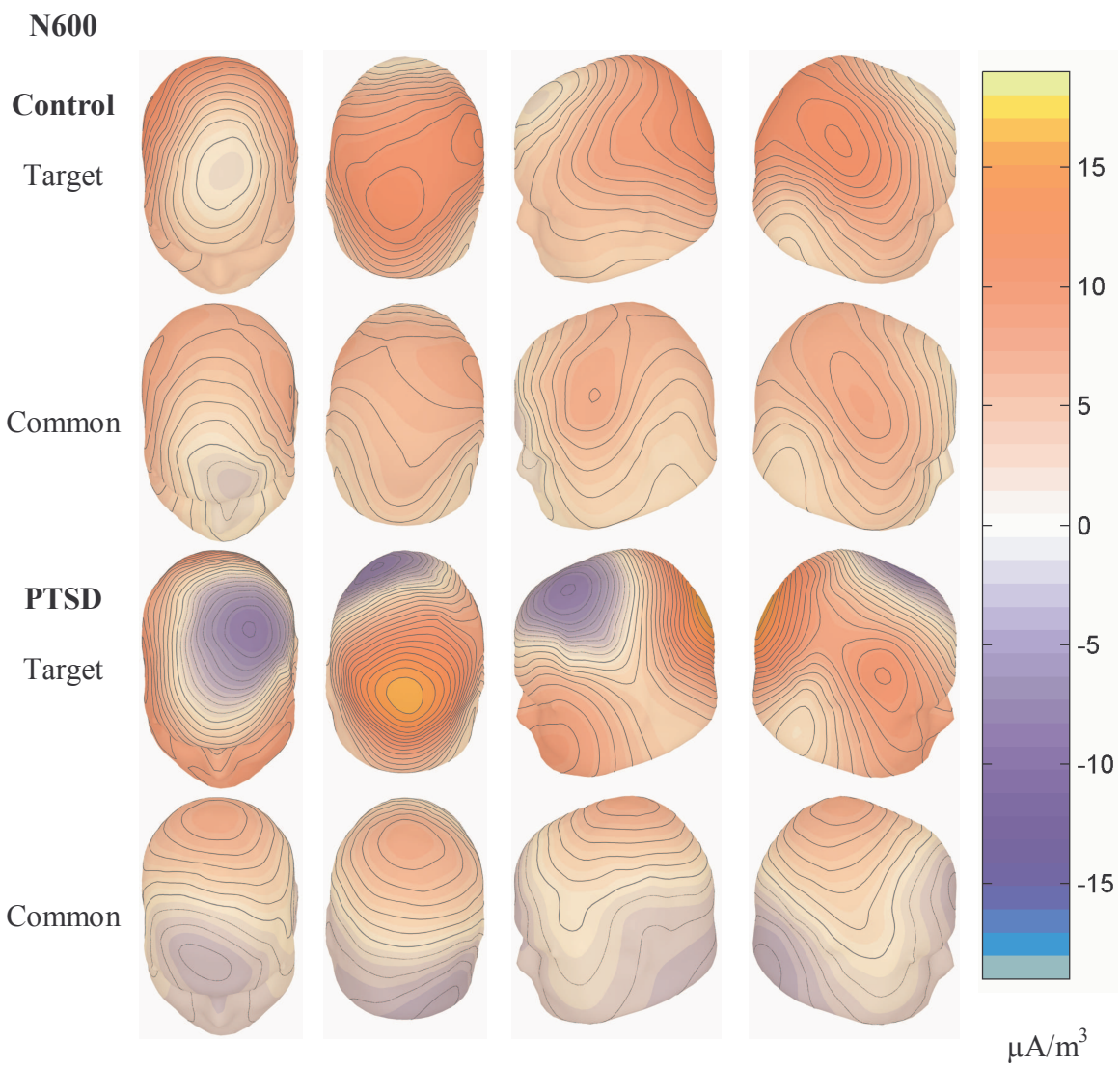

Figure 6-29. N600 SCD topography at 575 ms for controls (n = 10) and PTSD patients (n = 10) for both attended target and common words (contours at 1 μV intervals). The N600 peaks at the superior prefrontal region.

Table 6-6. SCD Summary Statistics for Target and Common Words [a] (continued below)

			Amplitude (μA/m^3)		Latency (ms)	
			CONT	PTSD	CONT	PTSD
P150	Left	T	8.27 (7.40)	14.20 (10.19)	148.75 (15.06)	141.50 (25.72)
SP		C	7.93 (6.92)	12.97 (9.57)	145.50 (9.56)	141.25 (23.22)
	Right	T	8.39 (5.74)	13.35 (9.36)	142.75 (22.65)	139.00 (25.34)
		C	6.69 (5.86)	11.63 (9.51)	138.75 (18.27)	138.50 (24.19)
N200	Left	T	-8.42 (5.47)	-8.53 (4.67)	178.75 (30.94)	178.50 (26.41)
PT		C	-6.10 (6.35)	-8.26 (4.81)	175.00 (19.86)	178.50 (24.73)
	Right	T	-7.27 (6.34)	-8.94 (5.89)	175.50 (26.35)	174.00 (25.12)
		C	-6.66 (4.93)	-7.62 (5.03)	172.75 (22.37)	172.00 (20.30)
P250	Left	T	11.44 (6.72)	8.41 (8.94)	243.25 (16.42)	227.50 (20.62)
OC		C	10.09 (5.92)	8.34 (8.49)	242.25 (17.97)	230.50 (11.77)
	Right	T	10.89 (5.44)	11.41 (8.82)	242.25 (16.69)	235.75 (24.95)
		C	10.29 (4.55)	9.82 (7.84)	240.75 (16.12)	234.50 (20.27)
P250	Left	T	7.73 (4.65)	2.29 (6.82)	245.25 (23.88)	228.50 (24.70)
IPF		C	5.56 (3.89)	1.91 (3.89)	240.25 (22.25)	239.00 (24.04)
	Right	T	7.94 (3.94)	2.56 (4.97)	244.75 (30.22)	231.75 (27.49)
		C	5.49 (3.16)	0.85 (3.31)	240.00 (23.86)	242.75 (20.02)
N300	Left	T	-8.42 (3.60)	-7.13 (6.99)	282.50 (31.42)	302.25 (34.43)
SF		C	-4.33 (2.03)	-2.12 (5.05)	259.00 (33.67)	269.25 (34.01)
	Right	T	-7.23 (4.65)	-4.47 (8.95)	274.75 (38.01)	261.50 (34.96)
		C	-4.58 (3.25)	-1.67 (5.41)	247.25 (23.05)	280.00 (47.52)

[a] T = Target; C = Common; CONT, n = 10; PTSD, n = 10; values are mean (SD).

Table 6-6 (continued). SCD Summary Statistics for Target and Common Words [a]

			Amplitude (μA/m^3)		Latency (ms)	
			CONT	PTSD	CONT	PTSD
P350	Left	T	15.06 (7.87)	16.17 (9.64)	356.00 (20.82)	357.75 (8.03)
OC		C	6.18 (8.11)	9.64 (5.69)	357.50 (8.08)	354.00 (13.90)
	Right	T	14.38 (6.66)	17.73 (9.92)	354.50 (21.04)	359.75 (15.48)
		C	8.50 (8.38)	9.10 (6.52)	361.50 (12.20)	356.25 (12.20)
P350	Left	T	12.70 (10.01)	4.91 (7.96)	373.00 (20.17)	374.00 (14.54)
SPF		C	5.28 (6.10)	5.50 (4.66)	363.00 (14.57)	374.25 (19.26)
	Right	T	12.37 (9.24)	4.44 (8.56)	375.50 (21.56)	361.00 (15.86)
		C	5.69 (5.20)	4.25 (3.81)	368.25 (17.80)	369.50 (19.21)
P450	Left	T	20.13 (9.27)	21.72 (10.42)	445.50 (37.21)	477.75 (32.26)
OC		C	7.39 (7.41)	3.76 (4.31)	468.50 (25.42)	449.50 (14.66)
	Right	T	19.27 (9.00)	22.12 (10.13)	447.50 (32.72)	485.00 (27.39)
		C	8.24 (6.64)	4.02 (5.36)	463.50 (36.00)	453.00 (20.30)
N600	Left	T	-6.53 (7.64)	-10.40 (5.69)	568.50 (52.38)	629.50 (41.28)
SPF		C	-1.21 (4.74)	0.08 (3.55)	581.50 (76.16)	608.50 (55.58)
	Right	T	-5.76 (7.86)	-7.55 (7.59)	568.00 (56.81)	625.75 (43.92)
		C	-0.93 (4.00)	0.27 (3.46)	581.00 (75.63)	603.00 (43.84)

[a] T = Target; C = Common; CONT, n = 10; PTSD, n = 10; values are mean (SD).

Table 6-7. Inferential Statistics for Target Detection SCD Components [a]

SCD		GP	TD	GPxTD	HS	GPxHS	TDxHS	GPxTDxHS
P150	Amp	2.07	5.74*	0.18	4.65*	0.48	7.45*	1.70
SP	Lat	0.19	0.81	0.54	4.39	0.76	0.29	0.07
N200	Amp	0.32	2.93	0.26	0.06	0.01	0.33	5.63*
PT	Lat	0.00	0.80	0.22	0.71	0.08	0.01	0.13
P250	Amp	0.14	2.01	0.01	3.32	4.53*	0.71	6.04*
OC	Lat	1.92	0.01	0.28	0.61	1.39	0.85	0.53
P250	Amp	7.79*	4.83*	0.68	0.07	0.14	1.59	0.70
IPF	Lat	0.83	0.51	3.60	0.11	0.16	0.00	0.00
N300	Amp	1.21	15.47**	0.08	2.07	0.59	7.59*	0.33
SF	Lat	2.07	3.11	0.97	2.48	0.11	5.41*	7.39*
P350	Amp	0.45	26.10***	0.00	1.26	0.07	0.25	8.21*
OC	Lat	0.01	0.01	1.12	0.45	0.03	0.64	0.53
P350	Amp	2.47	4.48*	5.02*	0.34	0.41	0.00	0.82
SPF	Lat	0.00	0.24	2.24	0.37	2.37	1.14	0.29
P450	Amp	0.07	74.51***	3.14	0.11	0.11	1.39	1.92
OC	Lat	1.77	0.39	8.58**	0.12	0.37	0.76	0.07
N600	Amp	0.13	31.66***	2.60	6.15*	1.45	3.29	1.55
SPF	Lat	8.07*	0.06	0.85	0.19	0.12	0.00	0.00

[a] Values are F[1,18], GP = group, TD = target detection, HS = hemisphere.

* $p<.05$, ** $p<.01$, *** $p<.001$, *2-tailed*; † $p<.05$, †† $p<.01$, ††† $p<.001$, *1-tailed*.

6.4.5.2 P150 SCD

The P150 peak was measured between 80-180 ms, it was largest over the superior parietal region between 135-150 ms (see Table 6-6, Figure 6-20 & Figure 6-23). ANOVA indicated a significant interaction of target detection by hemisphere in P150 amplitude (see Table 6-7). Mean comparisons after Bonferroni correction indicated larger mean P150 peak amplitude for targets than common words over the right superior parietal region (M = 1.71 μA/m^3, SE = 0.57 μA/m^3, $p<.01$), but no significant target

detection effect over the left superior parietal region (M = 0.79 μA/m^3, SE = 0.53 μA/m^3, *ns*).

6.4.5.3 N200 SCD

The N200 peak was measured between 140-220 ms, it was largest over bilateral posterior temporal regions between 170-180 ms (see Table 6-6, Figure 6-20 & Figure 6-24). ANOVA indicated a significant interaction of group by target detection by hemisphere in N200 amplitude (see Table 6-7). The interaction effect comprised a larger N200 for target than common words over the left posterior temporal region for controls (M = -2.32 μA/m^3, SE = 0.96 μA/m^3, p<.05).

6.4.5.4 P250 SCD

The P250 peak was measured between 225-250 ms, it was largest over bilateral occipital and inferior prefrontal regions between 190-280 ms (see Table 6-6, Figure 6-21 & Figure 6-25).

Occipital: ANOVA indicated a significant three-way interaction of group by target detection by hemisphere in P250 amplitude (see Table 6-7). Mean comparisons after Bonferroni correction indicated no significant group or target detection differences; the P250 was larger over the right than the left occipital region for target words in PTSD (M = 3.00 μA/m^3, SE = 0.98 μA/m^3, p<.01).

Inferior prefrontal: ANOVA indicated significant main effects of group and target detection in P250 amplitude (see Table 6-7). The mean P250 peak amplitude was larger in controls than PTSD patients (M = 4.78 μA/m^3, SE = 1.71 μA/m^3, p<.05). The mean P250 peak amplitude was larger for targets than common words (M = 1.68 μA/m^3, SE = 0.76 μA/m^3, p<.05).

6.4.5.5 N300 SCD

The N300 peak was measured between 215-340 ms, it was largest over the superior frontal region between 240-310 ms (see Table 6-6, Figure 6-22 & Figure 6-26). ANOVA indicated a significant two-way interaction of target detection by hemisphere in N300 amplitude (see Table 6-7). The mean N300 peak amplitude was larger for targets than common words in both left (M = -4.56 μA/m^3, SE = 0.73 μA/m^3, p<.001) and right superior frontal regions (M = -2.72 μA/m^3, SE = 1.18 μA/m^3, p<.05). The interaction consists of a larger mean N300 peak amplitude for targets in the left than the right superior frontal region (M = -1.93 μA/m^3, SE = 0.79 μA/m^3, p<.05), but no hemisphere effect for common words (M = -0.09 μA/m^3, SE = 0.76 μA/m^3, *ns*). Also, ANOVA indicated a significant three-way interaction of group by target detection by hemisphere in N300 latency (see Table 6-7). The interaction consists of an earlier mean N300 peak latency for targets than commons in the left superior frontal region in PTSD (M = 33.00 ms, SE = 14.90 ms, p<.05).

6.4.5.6 P350 SCD

The P350 peak was measured between 320-400 ms, it was largest over the occipital and superior prefrontal regions between 350-380 ms (see Table 6-6, Figure 6-21 & Figure 6-27).

Occipital: ANOVA indicated a significant three-way interaction of group by target detection by hemisphere in P350 amplitude (see Table 6-7). The mean P350 peak amplitude was larger for targets than common words (M = 7.48 μA/m^3, SE = 1.46 μA/m^3, p<.001). The interaction consisted of only a hemisphere difference for common words in controls, where mean P350 peak amplitude was larger over the right than the left occipital region (M = 2.31 μA/m^3, SE = 0.98 μA/m^3, p<.05).

Superior prefrontal: ANOVA indicated a significant two-way interaction of group by target detection in P350 amplitude (see Table 6-7). There was larger mean

P350 peak amplitude for targets than common words for controls (M = 7.05 μA/m^3, SE = 2.29 μA/m^3, p<.01), but not for PTSD patients (M = -0.20 μA/m^3, SE = 2.29 μA/m^3, *ns*).

6.4.5.7 P450 SCD

The P450 peak was measured between 380-550 ms, it was largest over bilateral occipital regions between 440-485 ms (see Table 6-6, Figure 6-20, Figure 6-21 & Figure 6-28). ANOVA indicated a significant target detection effect on P450 amplitude (see Table 6-7). The mean P450 peak amplitude was larger for targets than common words (M = 14.96 μA/m^3, SE = 1.73 μA/m^3, p<.001). Also, ANOVA indicated a significant two-way interaction of group by target detection in P450 latency (see Table 6-7). For target words, the mean P450 peak latency was shorter for controls than PTSD patients (M = -34.88 ms, SE = 13.57 ms, p<.05), there was no significant group difference for common words (M = 14.75 ms, SE = 8.59 ms, *ns*). Furthermore, for PTSD patients, the mean P450 peak latency was longer for targets than common words (M = 30.13 ms, SE = 11.98 ms, p<.05), while there was no significant latency difference between targets and common words for controls (M = -19.50 ms, SE = 11.98 ms, *ns*).

There was a positive linear trend between P450 latency and target reaction time [$r(17)$ = .36; β = 0.97 ± 0.53, $F(17)$ = 1.83, p<.1; R^2 = 0.13]. The linear regression equation is: mean(RT) = 71.16 + 0.97(P450 latency). That is, for each ms increase in the mean P450 latency, there is a target reaction time increase of 0.97 ms (no causality is implied in this relationship).

6.4.5.8 N600 SCD

The N600 peak was measured between 450-700 ms, it was largest over the superior prefrontal region between 565-630 ms (see Table 6-6, Figure 6-22 & Figure 6-29). ANOVA indicated significant main effects for target detection and hemisphere

in N600 amplitude (see Table 6-7). The mean N600 peak amplitude was larger for targets than common words (M = -7.11 μA/m^3, SE = 1.26 μA/m^3, p<.001) and it was larger over the left than the right superior prefrontal region (M = -1.02 μA/m^3, SE = 0.41 μA/m^3, p<.05). Also, ANOVA indicated a significant group difference in N600 latency (see Table 6-7). The mean N600 peak latency was shorter in controls than PTSD patients (M = -41.94 ms, SE = 14.76 ms, p<.05).

There was a significant positive linear relationship between N600 latency and target reaction time [$r(17)$ = .63; β = 0.93 ± 0.33, $F(17)$ = 2.85, p<.05; R^2 = 0.39]. The linear regression equation is: mean(RT) = -29.19 + 0.93(N600 latency). That is, for each ms increase in the mean N600 latency, there is a target reaction time increase of 0.93 ms (no causality is implied in this relationship).

6.4.5.9 Summary of SCD Component Findings

There was enhanced amplitude for target words in the right superior parietal P150, although the focus of this activity was in the left superior parietal area, where no target effect was found. The left posterior temporal N200 demonstrated enhanced target activity in controls, but not PTSD. There was similar topography, with concurrent occipital and frontal peaks, in the P250, P350 and P450. The occipital P350 and P450 were enhanced for targets. The prefrontal P250 and P350 had larger amplitude for targets, for which controls demonstrated greater effects than PTSD. There were left frontal peaks for the N300 and N600, which were greater for targets. The P450 and N600 latency were positively associated with reaction time, which all indicate delays in PTSD.

6.4.6 SCD Difference Waves

The difference waves (see Figure 6-19 & Figure 6-30 to Figure 6-32) demonstrate three peak components that were further analyzed:

- superior central ND350
- prefrontal and occipital PD450
- superior frontal ND600

Summary statistics for these components are given in Table 6-8 and the inferential analyses are described below (see Table 6-9), with the mean differences for significant effects.

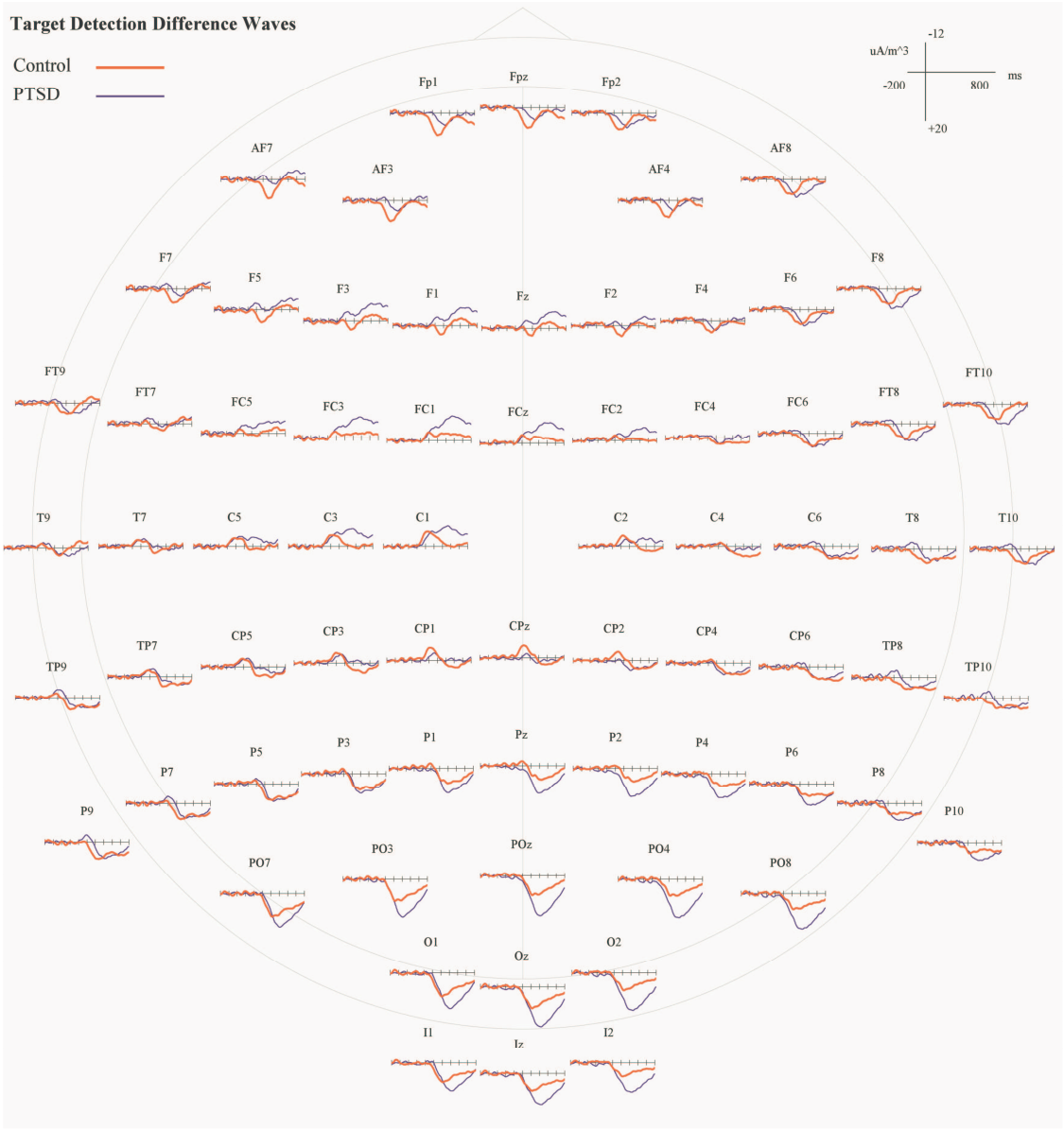

Figure 6-30. Target detection SCD difference waves for controls (n = 10) and PTSD patients (n = 10). Waveforms are given at 70 representative scalp sites (-200 to 800 ms, 100 ms intervals). Between 400-800 ms, PTSD patients demonstrate larger positive SCD difference wave activity at occipital and right inferior frontal areas, whereas controls demonstrate larger and earlier activity in left prefrontal areas. PTSD patients also demonstrate larger negative SCD difference wave activity in medial frontal areas.

Figure 6-31. Target detection SCD difference waveforms for controls (n = 10; red, solid) and PTSD patients (n = 10; blue, dash). The ND300 is illustrated at the fronto-central site, where it was followed by the larger ND600, which was clearly larger for PTSD patients. The prefrontal PD450 was earlier and larger for controls, whereas the parietal and occipital PD450 was larger for PTSD patients.

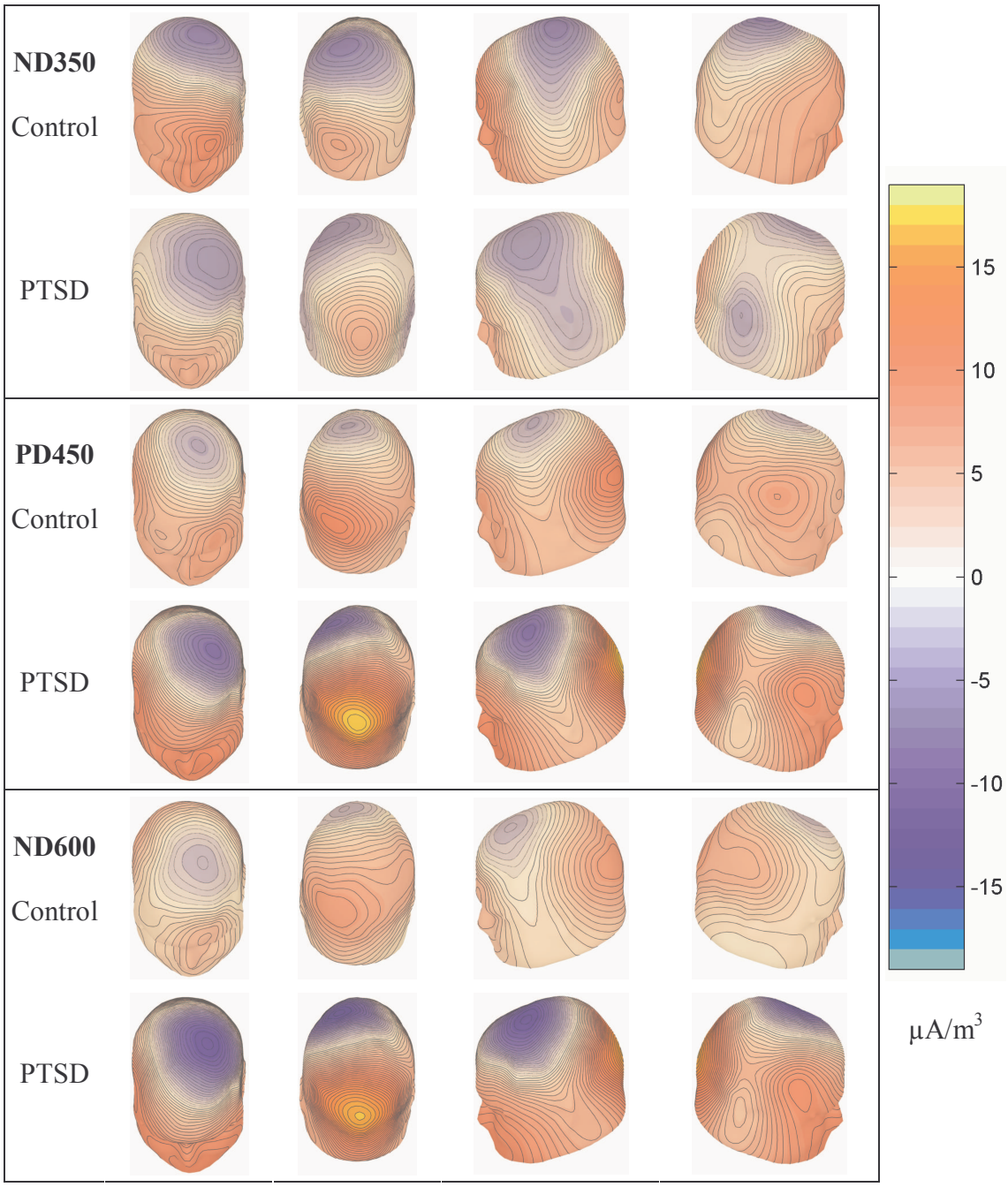

Figure 6-32. ND350, PD450 & ND600 SCD topography for controls (n = 10) and PTSD patients (n = 10) for the target detection difference wave (contours at 0.5 μV intervals). The ND350 is given at 330 ms, the PD450 is given at 480 ms and the ND600 is given at 575 ms. All maps are given on the same scale.

Table 6-8. Summary Statistics for Target Detection SCD Difference Components [a]

		Amplitude (μA/m^3)		Latency (ms)	
		CONT	PTSD	CONT	PTSD
ND350	Left	-9.93 (5.55)	-7.97 (5.28)	326.50 (36.02)	335.00 (49.96)
SC	Right	-8.30 (5.10)	-6.05 (7.55)	329.25 (37.58)	340.25 (52.07)
PD450	Left	14.87 (4.80)	20.14 (10.07)	460.00 (81.76)	524.25 (54.43)
OC	Right	12.78 (5.86)	21.35 (9.55)	482.00 (76.58)	524.75 (61.38)
PD450	Left	13.36 (5.91)	11.72 (7.97)	385.25 (58.36)	476.00 (81.65)
IPF	Right	11.73 (4.98)	11.75 (10.07)	380.75 (46.25)	466.00 (62.01)
ND600	Left	-9.67 (5.14)	-15.70 (7.67)	537.00 (94.32)	582.25 (75.62)
SF	Right	-7.40 (5.32)	-12.07 (6.07)	577.50 (97.31)	590.25 (90.70)

[a] Target - common words; CONT, n = 10; PTSD, n = 10; values are mean (SD).

Table 6-9. Inferential Statistics for Target Detection SCD Difference Components [a]

SCD		GP	HS	GPxHS
ND350	Amp	0.69	5.03*	0.03
SC	Lat	0.29	0.23	0.02
PD450	Amp	3.98	0.44	6.07*
OC	Lat	3.40	1.05	0.95
PD450	Amp	0.06	0.70	0.75
IPF	Lat	12.98**	0.25	0.04
ND600	Amp	4.51*	7.39*	0.39
SF	Lat	0.82	0.99	0.44

[a] Values are F[1,18], GP = group, HS = hemisphere.

* p<.05, ** p<.01, *** p<.001, *2-tailed*; † p<.05, †† p<.01, ††† p<.001, *1-tailed*.

6.4.6.1 ND350 SCD

The ND350 peak was measured between 220-420 ms, it was largest over the superior central region between 325-345 ms (see Table 6-8, Figure 6-31 & Figure 6-32). ANOVA indicated a significant hemisphere difference in ND350 amplitude (see Table 6-9). The mean ND350 peak amplitude was larger over the left than the right superior central region (M = -1.77 μA/m^3, SE = 0.79 μA/m^3, p<.05).

6.4.6.2 PD450 SCD

The PD450 peak was measured between 250-650 ms, it was largest over the prefrontal (380-480 ms) and occipital (460-525 ms) regions (see Table 6-8, Figure 6-31 & Figure 6-32).

Inferior prefrontal: ANOVA indicated a significant group difference in PD450 latency (see Table 6-9). The mean PD450 peak latency was shorter for controls than PTSD patients over the inferior prefrontal region (M = -88.00 ms, SE = 24.43 ms, $p<.01$). Also, there was a significant positive linear relationship between PD450 latency and target reaction time [$r(17) = .56$; $\beta = 0.73 \pm 0.25$, $F(17) = 2.95$, $p<.01$; $R^2 = 0.31$]. The linear regression equation is: mean(RT) = 219.94 + 0.73(PD450 latency). That is, for each ms increase in the mean PD450 latency, the target reaction time increases by 0.73 ms (no causality is implied in this relationship).

Occipital: ANOVA indicated a significant two-way interaction of group by hemisphere in PD450 amplitude (see Table 6-9). The mean PD450 peak amplitude was larger over the left than the right occipital region in controls (M = 2.09 μA/m^3, SE = 0.94 μA/m^3, $p<.05$), but not PTSD (M = -1.20 μA/m^3, SE = 0.94 μA/m^3, *ns*). There was no group difference in mean PD450 peak amplitude over the left occipital region (M = -5.28 μA/m^3, SE = 3.53 μA/m^3, *ns*), but there was smaller mean PD450 peak amplitude for controls than PTSD patients over the right occipital region (M = -8.57 μA/m^3, SE = 3.54 μA/m^3, $p<.05$).

6.4.6.3 ND600 SCD

The ND600 peak was measured between 420-720 ms, it was largest over the superior frontal region between 535-595 ms (see Table 6-8, Figure 6-31 & Figure 6-32). ANOVA indicated significant main effects of group and hemisphere in ND600 amplitude (see Table 6-9). The mean ND600 peak amplitude was larger over the left

than the right superior frontal region (M = -2.95 μA/m^3, SE = 1.08 μA/m^3, p<.05). The mean ND600 peak amplitude was larger for PTSD patients than controls (M = -5.35 μA/m^3, SE = 2.52 μA/m^3, p<.05).

Also, there was a significant positive linear relationship between ND600 latency and target reaction time [$r(17)$ = .53; β = 0.67 ± 0.26, $F(17)$ = 2.59, p<.05; R^2 = 0.28]. The linear regression equation is: mean(RT) = 149.98 + 0.67(ND600 latency). That is, for each ms increase in the mean ND600 latency, the target reaction time increases by 0.67 ms (no causality is implied in this relationship).

6.4.6.4 Summary of SCD Difference Component Findings

The ND350 and ND600 SCD had larger activity over left frontal sites, which could indicate left motor cortex activity. The ND600 was larger for PTSD than controls. It appeared that controls had earlier activity in this region for the ND350, while PTSD patients had later activity in the ND600. If these components indicate motor activity, this pattern is consistent with slower reaction time in PTSD. The ND600 latency was positively associated with reaction time.

The PD450 SCD topography is consistent with occipital visual activity. The controls demonstrate left hemisphere dominance in this activity, consistent with left hemisphere linguistic processing, whereas PTSD patients had similar amplitude over the left and right hemisphere. A prefrontal PD450 component peaked earlier for controls than PTSD patients. The prefrontal PD450 was positively associated with reaction time.

6.5 DISCUSSION

6.5.1 Overview

This study investigated target detection for neutral words in PTSD. The task required attention to a series of red and blue words, with selective attention to one or

another color, and detection of a specific target word in the attended color. It was expected that target word recognition would elicit greater activity in frontal areas at 300-500 ms and parietal areas at 500-800 ms. Scalp potentials associated with motor responses were expected in left fronto-central areas. Decreased amplitude of target recognition components and delayed motor responses were expected for PTSD patients (especially activity that resembles the N2/P3 ERP complex).

During visual processing of attended word features (within 250 ms), there was larger target activity in posterior temporal, prefrontal and parietal areas. Again, after 250 ms, there was greater amplitude for targets in posterior temporal, frontal and parietal components. These components suggest enhanced target activity during visual linguistic processing in posterior temporal areas, followed by frontal and parietal executive systems engaged in evaluation and word recognition processing. Motor responses were indicated by greater fronto-central negativity at 300-600 ms.

For PTSD patients, there were abnormal components at 200-400 ms, including smaller activity in posterior temporal visual processing and associated executive activity in frontal regions. PTSD patients demonstrated slower response activity, which was also apparent in the increased latency of fronto-central components. There was some evidence of greater reliance on occipital and parietal areas for target processing, which may be an adaptation to deficits in frontal activity.

6.5.2 Target Detection Processes

The posterior temporal P80 ERP was enhanced for target words. It might be possible to attribute this effect to greater residual alpha frequency in the averages for the target words. The baseline period indicates larger alpha oscillations for targets than commons, which might arise from fewer trials in the target averages and therefore less averaging of asynchronous alpha oscillations. Nevertheless, the effect at this early latency has topography consistent with visual activity. The positive shift in the ERP

components at this early latency remains present during the occipital and posterior temporal N150 ERP (see Figure 6-4), giving the appearance of smaller target N150 ERP amplitude (although no significant condition differences were found for this component). At the same time, there was a larger prefrontal P150 ERP for targets, suggesting the early involvement of frontal executive systems (see also Foxe & Simpson, 2002; Giesbrecht, Woldorff, Song, & Mangun, 2003). The timing of this positive shift at posterior sites coincides with the arrival of word stimulus information in striate and extrastriate cortex, where the word forms would be analyzed within 150-250 ms (Petersen et al., 1993; Allison et al., 1994; Nobre et al., 1994; Halgren et al., 1994; Kuriki et al., 1998; Schendan et al., 1998; Cohen et al., 2000; Rao et al., 2003). At 200-250 ms there was enhanced target activity in a posterior temporal N200 SCD, an occipital and prefrontal P250 ERP, and a prefrontal P250 SCD. In general, these findings suggest occipito-temporal activity related to visual word processing and associative processing in frontal regions during the analysis and elementary linguistic encoding of word forms. It is likely that at the point when stimuli are no longer visible, the frontal working memory system is engaged to facilitate the encoding of visual information into working memory. The posterior temporal N200 SCD and the occipital P250 ERP could indicate the modulation of visual activity during integration of new visual information into working memory.

Further enhanced activity for target words was clear in a posterior temporal N300 ERP, which was clearly illustrated in the ND300 ERP. These components demonstrated dominant peaks over the left posterior temporal area, consistent with engagement of visual linguistic processes. The N300 ERP is the main point of processing where target words are most clearly distinguished from common words. Moreover, the amplitude of this component was linearly related to reaction time, which suggests that this processing stage is critical for target stimulus evaluation and associated response processing. The

timing of this component is consistent with the estimated time to complete visual word form processing, which requires approximately 150-250 ms (e.g., Nobre et al., 1994; Halgren et al., 1994; Cohen et al., 2000). The timing of the ND300 precedes or coincides with reports of an enhanced N400 ERP in semantic incongruity studies (e.g., Kutas & Hillyard, 1980a,b, 1983). The brain sources of this scalp component could arise from processing visual word forms in the inferior occipito-temporal cortex, as activity in the anterior fusiform gyrus has been identified during semantic processing (Nobre et al., 1994) and the ventral visual stream is implicated in the discrimination of visual forms during working memory tasks (see, Desimone & Duncan, 1995; Desimone, 1996; Miller & Cohen, 2001). It is possible that it indicates the visual word form processing that contributes to further linguistic activity in the angular gyrus (Wernicke's area), where auditory and visual linguistic stimuli may be transformed into semantic information. Also, the topography of this component is consistent with similar work that suggests phonological encoding for visual linguistic stimuli, although that study showed phonological activity at 450-500 ms (e.g., Gevins et al., 1995). If these scalp components are engaged in the initial discrimination of target features, they may be a visual linguistic homologue of the auditory N2 for target events. The pattern of activity for the N300 ERP indicates discrimination of the significant target event and it precedes larger positive components that may indicate further stimulus evaluation.

The parietal P450 ERP is similar to the conventional P3 ERP. The P450 ERP has a parietal peak that is enhanced for target words, with a latency that indicates more extensive processing of the targets. Similar activity is observed in the posterior P450 SCD component, with enhanced activity for target words. The SCD difference waves indicate an additional prefrontal PD450 component that precedes the posterior scalp activity; this prefrontal component could be an indication of frontal executive systems engaged in attention, working memory and linguistic processing (e.g., Petersen et al.,

1990; Buckner & Koutstaal, 1998; see also Halgren et al., 1998). There are parallels in the frontal and parietal activity of this study with results from word recognition studies, which show activity in parietal areas at 300-500 ms, which coincides with frontal activity, followed by later parietal activity at 500-800 ms (Fabiani et al., 1986; Rugg et al., 1998). These studies may support the interpretation of the frontal and parietal activity in the present study as the engagement of linguistic recognition and associative processing. These large positive components generally indicate the additional activity involved in encoding, evaluation and recognition of target words. The linear relationships between these components and reaction times demonstrate that this activity comprises some degree of stimulus-response association.

The response activity was indicated by N300 (ND350) and N600 (ND600) SCD components that demonstrate topography consistent with left motor cortex activity. These components were focused over the left fronto-central area, with enhanced target activity. The SCD is more sensitive than scalp ERPs to shallow cortical sources, so the SCD components are more likely to indicate the response activity from motor cortex. The linear relationship between the N600 and ND600 SCD components and reaction time further supports the inference that these scalp components are related to response processing (this may be response initiation or post-response evaluation or monitoring).

The later parietal P700 ERP was enhanced for target words, which was also clear in the PD700 ERP. This scalp activity follows the response activity, so it may be an indication of further associative linguistic encoding or the consolidation of the target representation. The P700 ERP (& PD700) arises after the response is made, so it may not indicate stimulus evaluation in the same manner as a P300 ERP, but it could indicate the engagement of linguistic associative processing, target consolidation and/or response evaluation processes. It is possible that semantic associative processing is engaged within 400 ms, as indicated by the N400 ERP in semantic incongruity studies

(e.g., Kutas & Hillyard, 1980a,b, 1983; see also Nobre et al., 1994), so this P700 activity could proceed from semantic encoding and associative activity. Näätänen (1990, 1992) proposes that the occurrence of a target event reinforces the target representation; the linguistic information available in this study may encourage more extended consolidation processing, apparent in the P700 ERP, which indicates the greater activity invested in maintaining the target word information. Also, after 500 ms the controls demonstrate extended positive slow wave activity in frontal regions for target words in both the ERP and SCD waveforms. Similar, although larger, sustained frontal and parietal components are observed in working memory tasks during delay periods that require the maintenance of working memory content (e.g., Gevins et al., 1996; Ruchin et al., 1990, 1992, 1995). This task requires constant maintenance of a target representation in working memory, so the occurrence of any target event could both refresh that representation and engage processes to maintain and consolidate the representation.

6.5.3 Target Detection in PTSD

There were no indications of abnormal early visual processing in PTSD. Several early effects of target processing were identified in posterior temporal, parietal and frontal regions, within 200 ms, without any clear abnormality in PTSD. These findings are consistent with prior findings of normal N1/P2 activity in auditory target detection tasks (e.g., McFarlane et al., 1993).

The earliest indication of abnormal activity in PTSD was the absence of enhanced target activity in the posterior temporal N200 SCD – the mean peak amplitudes indicated discrimination of targets from commons for controls, but not for PTSD. A clearer indication of abnormal activity in this posterior temporal region was diminished amplitude of the ND300 ERP in PTSD. This component appeared similar to the N2 ERP, in that it may represent perceptual discrimination, leading to further evaluation of

target stimuli. As discussed above, these posterior temporal scalp components may index the processing and encoding of visual word forms, including transformation of visual linguistic codes into more meaningful phonological and semantic information, thereby facilitating evaluation and working memory processes. The posterior temporal processing is important for effective responses to target words, as there was a linear relationship between the ND300 ERP and reaction time, which indicated that larger ND300 activity is associated with quicker responses. The slower responses in PTSD might be attributed, in part, to abnormal activity at this stage of target word processing. Deficits in this activity may parallel findings of abnormal auditory discrimination, indicated by the N2 ERP (e.g., McFarlane et al., 1993).

There were several indications of abnormal prefrontal activity in PTSD during target processing: (a) the P250 SCD was smaller in PTSD, (b) the P350 SCD demonstrated differential target processing for controls, but not PTSD, and (c) the PD450 latency was shorter in controls than PTSD. The abnormal prefrontal P250 SCD might indicate the failure of frontal executive systems to become involved in the early visual processing of targets. This deficit is then further reflected in the latter components at 350-450 ms and again in response times. PTSD patients demonstrated slower response activity, which was also indicated by increased latency of frontal N600 SCD.

There were indications of larger PD450 SCD over the right occipital area for PTSD. The controls demonstrate left hemisphere dominance in this activity, consistent with left hemisphere linguistic processing, whereas PTSD patients had larger amplitude over the right hemisphere. Controls also demonstrate a prefrontal PD450 component, which peaked earlier than for PTSD patients, and there was a significant positive linear relationship between PD450 latency and target reaction time. This pattern of activity may be an indication of greater reliance on posterior visual systems for effective target

detection in PTSD. If there is an earlier deficit in visual, linguistic processing and associated frontal executive engagement, PTSD patients may rely on more visual spatial processing in occipital and parietal systems. In this light, it is interesting to note that PTSD patients had larger amplitude over the right hemisphere for the earlier occipital P250 SCD, which might indicate right hemisphere dominance for early visual-spatial analysis of target events. In contrast, controls demonstrate clear left hemisphere posterior temporal activity and larger, earlier PD450 SCD at frontal areas, leading to earlier response activation. The grand mean waveforms indicate that controls demonstrate similar amplitude of frontal and parietal P450 SCD, with earlier onset of frontal than parietal activity, while the PTSD patients demonstrate much greater parietal than frontal P450 SCD. Controls also have an extended prefrontal target SCD positivity at 600–1500 ms, which is absent in PTSD. These patterns of scalp activity may indicate greater controlled, linguistic processing in controls, while patients employed visuo-spatial strategies to accommodate for frontal deficits (see associated PET results; Shaw et al., 2002; Clark et al., 2003; see also van der Kolk, 1997).

PTSD patients demonstrated deficits in a superior frontal PD700 ERP. This result is a combination of greater frontal positive activity in controls and greater negative activity in PTSD. The negative activity for PTSD could be a delay in frontal negative components related to response processing. The ND350 – ND600 SCD are located over left motor cortex (for right handed responses) and they appear to be larger for controls early (300-400 ms), while larger for PTSD patients later (400-600 ms). This delay in the scalp components is consistent with the delays identified in response times.

6.5.4 Conclusions

The target word processing engages enhanced activity in a distributed network of visual, linguistic systems. There are indications of top-down modulation of visual word feature processing, with enhanced activity for attended target words in posterior

temporal areas from 100-300 ms. During this visual feature extraction and discrimination, there is also enhanced activity in frontal and parietal areas. At the point when stimuli are no longer visible, frontal activity suggests the initial integration of information into working memory. Associated activity in occipital and posterior temporal areas signifies more intensive visual activity during integration of attended target information into working memory. A posterior temporal negativity at 300 ms clearly distinguishes target from common words and its relation to reaction time indicates this processing stage is involved in stimulus-response associations. At 300 ms, this scalp activity most likely reflects the completion of visual word form processing, providing a basis for target discrimination, and the initiation of semantic encoding. The further stimulus evaluation and target recognition was indicated by enhanced frontal and parietal activity at 300-600 ms. This activity coincided with left fronto-central activity that suggests motor cortex activity. There was also extended target activity over posterior areas, which could be an indication of sustained attention or consolidation of target information.

There are no clear abnormalities of scalp activity within 200 ms for PTSD. However, during the discrimination of word forms, there are deficits in the posterior temporal area at 200-300 ms. This suggests some difficulty with target word discrimination and further encoding of visual word forms into phonological and semantic information, which facilitates working memory processes. There was also diminished prefrontal activity at 250-450 ms, which signifies the failure to engage frontal executive systems for working memory. The deficits at 200-400 ms are further reflected in latter processing stages and response times. However, there is larger occipital and parietal activity at 400-500 ms, which could be an indication of greater reliance on posterior visual and executive systems for effective target detection. Given earlier deficits in visual, linguistic processing and associated frontal executive

engagement, PTSD patients may rely more on perceptual processing. There were delays in the large negative activity in left fronto-central areas related to motor responses, consistent with longer reaction times in PTSD.