

# Retrieval and emotional processing of traumatic memories in posttraumatic stress disorder: Peripheral and central correlates

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## Abstract

Posttraumatic stress disorder (PTSD) is thought to be characterized by dysfunctional memory processes, i.e., the automatic re-experiencing of the traumatic event and the inability to consciously recall facts about the traumatic event, as well as altered emotional processing of trauma-relevant cues.

The present study examined the cerebral mechanisms underlying the cued recall of trauma-specific memories and the emotional processing of the presented cues in 16 PTSD patients, 15 trauma-exposed subjects without PTSD and 16 healthy controls. Subjects received questions about their specific trauma as well as other disastrous and neutral events while the electroencephalogram and heart rate were measured. The PTSD patients showed no impairment in trauma-specific declarative memory compared to non-PTSD subjects but had some deficits in general declarative memory as assessed by the Wechsler Memory Scale-Revised. Compared to healthy control subjects, PTSD patients displayed increased P300 and late positive complex amplitudes to trauma-specific questions, indicating enhanced emotional processing of these cues. In line with their behavioral performance, both trauma-exposed groups showed decreased terminal contingent negative variation amplitudes to trauma-specific questions over frontal electrodes reflecting altered memory retrieval. Within-group comparisons revealed that only the PTSD group but not the other groups showed a differentiation between trauma-specific and neutral questions with respect to the LPC, tCNV and P300. Concordantly with previous studies, PTSD patients showed elevated resting heart rate compared to the healthy controls. These findings are discussed in the context of current models of the role of declarative memory in the development and maintenance of PTSD.

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## 1. Introduction

Dysfunctional memory processes are thought to play a crucial role in the development and maintenance of posttraumatic stress disorder (PTSD). Two different types of memory disturbances are part of the diagnostic criteria of PTSD (DSM-IV-TR, American Psychiatric Association, 2000): the automatic re-experiencing of the traumatic event and the inability to consciously recall facts about the traumatic event. This phenomenon has been referred to as dissociation between declarative and non-declarative trauma memories (e.g., Elzinga & Bremner, 2002). It has been suggested that traumatic memories of PTSD patients are disorganized and fragmented due to the failure to integrate

these memories into the autobiographical context (e.g., van der Kolk, 1997). Based on his dual-representation theory of memory, Brewin (2001) postulated an overactivation of situationally dependent and a deficit in the activation of verbally accessible memories in PTSD patients. Although Lanius et al. (2004) did not study memory processes but the neuronal reactivity of PTSD patients to trauma-related and neutral scripts, their findings support Brewin's theory. PTSD patients and trauma-exposed subjects without PTSD revealed different brain connectivity patterns, whereas traumatized subjects without PTSD showed a left dominated activation pattern while hearing trauma-relevant scripts, PTSD patients exhibited a mainly right distributed network of brain activation. This difference might account for the non-verbal nature of traumatic memory retrieval in PTSD patients compared to a more verbal nature of trauma memory recall in traumatized subjects without PTSD. As other neuroimaging studies (Hendler et al., 2003; Protopopescu et al.,

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2005) observed an increased amygdala activation in response to trauma-related stimulus material in PTSD patients, but not traumatized subjects without PTSD, it was hypothesized that this hyperactivation may suppress activation of the hippocampus and thus result in an inhibition of declarative memory retrieval and limited access to trauma-specific memories.

An overly active amygdala together with a hypoactivation of the anterior or medial prefrontal cortex has been shown not only to trauma-related, but also to trauma-irrelevant emotional stimuli (Shin et al., 2001, 2004). In addition, on the behavioral level, an enhanced processing of and an increased reaction to general threat and trauma-related stimuli (Buckley, Blanchard, & Neill, 2000) has been reported. Several studies that used interference paradigms, for example, the emotional Stroop paradigm (Bryant & Harvey, 1995; Foa, Feske, Murdock, Kozak, & McCarthy, 1991; Kaspi, McNally, & Amir, 1995; McNally, Kaspi, Riemann, & Zeitlin, 1990; Vrana, Roodman, & Beckham, 1995) have shown altered processing of trauma-related information, indicated by increased response latencies to threat- or trauma-related stimuli. The deviant processing of trauma- and threat-relevant information in PTSD patients has also been observed on an electrophysiological and neuronal level. A meta-analytic review of event-related potential studies in PTSD by Karl, Malta, and Maercker (2005) revealed that PTSD patients reliably showed enhanced P300 amplitudes to trauma-related cues (e.g., distractors in an oddball paradigm) compared to non-PTSD trauma controls and compared to neutral or non-trauma-related cues. Blomhoff, Reinvang, and Malt (1998) observed a significant positive correlation between symptoms of hyperarousal and amplitudes of positive event-related potentials occurring 200 and 350 ms after the presentation of negative, non-trauma-related words and a significant positive correlation between avoidance symptoms and negative as well as positive words.

The review by Buckley et al. (2000) and the meta-analytic study by Karl and co-workers (2005) indicate that the psychophysiological reactivity to threat- and trauma-related information in PTSD has been studied intensively, using various experimental paradigms and response levels (reaction times, event-related potentials). However, studies on declarative trauma memories have not been examined to the same extent. The majority of available studies on traumatic memory retrieval in PTSD patients used interview data (Merckelbach, Dekkers, Wessel, & Roefs, 2003), self-report questionnaires or classical cued recall paradigms (e.g., Harvey, Bryant, & Dang, 1998) and the findings of these studies are inconsistent. Merckelbach et al. (2003) reported an absence of trauma-specific amnesic symptoms in a sample of Danish survivors of Japanese–Indonesian concentration camps. Harvey et al. (1998) studied the cued recall of general and trauma-specific memories in trauma-exposed subjects with and without acute stress disorder (ASD). In this study, patients with ASD recalled fewer traumatic as well as more overgeneralized, non-specific autobiographical memories. The recall deficit of trauma-specific memories accounted for 25% of the variance of PTSD symptom severity 6 months later.

Experimental studies examining neurobiological processes underlying traumatic memories are rare. Specifically, no study

examined the electrophysiological basis of traumatic memory retrieval. In contrast to functional neuroimaging studies (fMRI, positron-emission-tomography), event-related potentials (ERP) can indicate various stages of memory processes due to their high time resolution (Rugg, 1996). In addition, ERPs have already been shown as a useful tool in studying cognitive processes, such as memory retrieval. One slow wave potential, which has been shown to be indicative of memory retrieval is the negative contingent variation (CNV). The CNV amplitude is *negatively* correlated with the effort involved in retrieving a specific memory (e.g., Rösler, Heil, & Hennighausen, 1995) and is usually observed during an S<sub>1</sub>–S<sub>2</sub>-paradigm in which subjects are required to hold information about a warning stimulus (S<sub>1</sub>) in working memory and make a decision at S<sub>2</sub>, the imperative stimulus. Within the CNV two components can be distinguished: the initial CNV (iCNV), reflecting the processing and evaluation of the warning stimulus (S<sub>1</sub>; Fenk, 1978) and the terminal CNV (tCNV), being associated with motor and preparatory processes and the anticipation of relevant information (S<sub>2</sub>; Leynes, Allen, & Marsh, 1998).

Two positive potentials, the P300, occurring 200 and 350 ms after stimulus onset and the late positive complex (LPC) starting at about 600 ms after stimulus onset have been shown to covary *positively* with allocation of psychological resources to process an event (e.g., Johnson, 1993; Rugg & Doyle, 1994) and even more with the value or salience of a stimulus in the context of a given task (Johnson, 1986). Several studies have reported increased P300 and LPC amplitudes to emotionally meaningful compared to neutral stimuli (e.g., Keil et al., 2002).

In the present study event-related potentials (P300, LPC and CNV) were used to examine the cerebral mechanisms underlying the cued recall of trauma-specific memories and the emotional processing of the presented cues in PTSD patients, trauma-exposed subjects without PTSD and matched controls. The memory performance and reaction times as well as self-report measures (e.g., confidence ratings) were assessed to characterize traumatic memory retrieval on a behavioral level. To be able to draw conclusions not only about group differences in traumatic memories but also the specificity of traumatic memory impairment in PTSD patients, memories about neutral events as well as other disastrous events were included in the experimental procedure. Apart from trauma-exposed subjects without PTSD, healthy controls without any trauma experience in the past served as a second control group. To differentiate a possible impairment in trauma-specific declarative memory from a general declarative memory deficit, we additionally included a behavioral memory test (Wechsler Memory Scale; WMS-R) in the present study.

Based on the theoretical assumptions and empirical findings for declarative trauma memories described above, we assumed that PTSD subjects recall significantly less trauma-specific memories than non-PTSD subjects, accompanied by an increased reaction time and lower confidence ratings. The healthy controls had never experienced the trauma and were therefore hypothesized to report significantly less correct trauma-specific items than the two trauma-exposed groups. This impaired access to trauma-specific memories in PTSD patients

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