BRIEF REPORT

Subjective Premorbid Memory in Posttraumatic Stress Disorder

Florentine Larbig¹, Lena Jelinek¹, Michael Kellner¹, Karl-Heinz Biesold², Klaus Barre², and Steffen Moritz¹

¹University Hospital Hamburg-Eppendorf, Department of Psychiatry and Psychotherapy (Clinical Neuropsychology Unit), Hamburg, Germany
²Department of Psychiatry and Neurology, Military Hospital of the Federal Armed Forces Hamburg, Germany

Corresponding author: Florentine Larbig, Dipl.-Psych., University Hospital Hamburg-Eppendorf, Department of Psychiatry and Psychotherapy (Clinical Neuropsychology Unit), Martinistrasse 52, D-20246 Hamburg, Germany, E-mail: florentine2000@yahoo.de

Abstract

Background: It is unclear whether memory deficits found in patients with PTSD (posttraumatic stress disorder) precede the traumatic event and may thus represent a vulnerability factor or a consequence of the trauma.

Objective: The aim of this study was to shed light on this question.

Method: A newly developed neuropsychological interview to estimate premorbid memory performance was administered along with an objective memory test on 21 PTSD patients and 21 healthy controls. Possible confounds, such as comorbid depressive symptoms and social desirability, were considered.

Results: PTSD patients and controls did not exhibit significant differences on estimated premorbid memory. The majority of the PTSD patients (73%) reported a decline of memory performance following the traumatic event that was associated with objective memory impairment.

Conclusion: The findings suggest that memory deficits in PTSD emerge after and not before the trauma. Further research with larger sample sizes and prospective studies are warranted to substantiate our results (German J Psychiatry 2008; 11: 149-152).

Keywords: PTSD, premorbid memory, trauma, neuropsychology, cognition

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Introduction

Studies on neuropsychological functioning have rather consistently reported memory deficits in posttraumatic stress disorder (PTSD), with only few studies failing to confirm impairment (Danekwerts & Leathem, 2003; Horner & Hamner, 2002). It is still subject of an ongoing debate, however, whether memory deficits precede the traumatic event and may thus represent a vulnerability factor or arise as a consequence of the trauma.

A twin study conducted by Gilbertson and colleagues (Gilbertson et al., 2002) concludes that a smaller hippocampus, a brain region known for its role in declarative memory, may predispose to PTSD. This data contrasts with animal studies, which suggest that stress-induced secretion of neurotoxic glucocorticoids damages the hippocampus, indicating that memory deficits follow the trauma (Sapolsky, 2000). These findings were not supported by a longitudinal study with non chronic PTSD patients, where no reduction in hippocampal volume could be found after the traumatic event (Bonne et al., 2001).
The present study pursued an alternative strategy to elucidate this unresolved issue. Patients with PTSD were asked to estimate their level of memory performance prior and subsequent to the trauma. Retrospective estimates of different memory domains in PTSD patients were contrasted with those of healthy (non-traumatized) controls.

As little is known about the relationship between subjective and objective measurement of memory deficits, an objective memory task was administered to establish the association between current memory performance and subjective estimates of current functioning.

Depression, a common comorbid disorder of PTSD (Kessler et al., 1995) and known to aggravate memory deficits (Burt et al., 1995), was considered as a potential confounding variable as well as social desirability.

Material and Methods

Sample

A sample of 21 PTSD inpatients or outpatients with heterogeneous causes of trauma was recruited (16 male/5 female; age: mean = 32.57, SD = 8.39; years of education: mean = 10.86, SD = 1.49). Traumatic events included serious accidents (n=12), combat or war zone experiences (n=5), sexual assaults (n=3), and other (n=1). Comparison subjects were 21 healthy participants (11 male/10 female; age: mean = 32.76, SD = 9.48; years of education: mean = 11.62, SD = 1.47).

Psychopathological and neurocognitive assessment

Diagnosis relied on standardized diagnostic interviews according to DSM-IV criteria (Mini Psychiatric Interview/SCID). The Posttraumatic Stress Diagnostic Scale (PDS, Foa et al., 1996), the Beck-Depression Inventory (BDI), and the Hamilton Depression Rating Scale (HDRS, 17-item version) were administered to quantify psychopathology. Estimated verbal intelligence was assessed with a vocabulary test (MWT-B, Lehrl, 1995). A “lie scale” was administered to the degree to which responses were made in terms of social desirability; subscale 10 (openness) of the Freiburger Personality Inventory-Revised Version (FPI-R, Fahrenberg et al., 2001). Exclusion criteria were psychotic symptoms, current/lifetime alcohol or substance dependence, significant neurological disorders, or traumatisation before the age of 18.

Premorbid and current memory assessment

Premorbid memory function was assessed with a newly developed structured Interview for the Assessment of Premorbid Memory (IAPM). Along six memory dimensions, participants estimate their memory performance in retrospect: memory of names, prospective, narrative, spatial memory, dyslexia and attention. For both samples, the reference point of performance was the time of (late) adolescence (before the age of 18). Participants were asked to rate their performance on a five-point Likert scale in comparison to their peers of that time (1=severe problems, 2=moderate/light problems, 3=comparable with peers, 4=slightly better, 5=outstanding). Additionally, 15 patients (subsample) were asked if they had noticed a decline of memory performance after the traumatic event.

To assess objective performance on short-term and long-term verbal free recall, the Picture Word Memory Test (PWMT), a task similar to the Auditory Verbal Learning Test (AVLT), was administered (Jelinek et al., 2006).

After complete description of the study to the subjects, written informed consent was obtained.

Results

No significant group differences emerged for age, gender, years of education and “openness”/social desirability (at least p > .1). However, both groups differed significantly on estimated verbal intelligence (t = -2.38, df = 40, p ≤ .05) with the PTSD patients presenting a lower estimated verbal intelligence (PTSD patients: M = 103.52, SD = 17.18; healthy controls: M =113.62, SD = 9.18).

PTSD patients differed significantly on self-assessed and clinician-assessed depression from the control group and showed a moderate degree of depression (HDRS: t = 8.92, df = 40, p ≤ .001; M = 15.10, SD = 6.70; BDI: t = 7.31, df = 39, p ≤ .001; M = 23.10, SD = 11.79). Control subjects revealed no clinical relevant depressive symptoms (HDRS: M = 0.95, SD = 2.8; BDI: M = 2.80, SD = 3.96).
There were no significant group differences on five of the six domains of the IAPM, except for “spatial memory” (see Table 1). Here, patients tended to rate their memory performance higher than the controls.

To determine the impact of potential moderator variables on subjective memory performance, the relationship between the six IAPM subscale scores, depression, openness, and estimated verbal intelligence was investigated. None of the parameters of the IAPM in both groups correlated significantly with self-assessed, clinician-assessed depressive symptoms, openness/social desirability or estimated verbal intelligence.

Seven patients with PTSD reported current involvement in legal disputes and for this reason might have downplayed premorbid memory dysfunction. The exclusion of this group from the analysis did not alter results.

From the above-mentioned subsample of 15 PTSD patients, 11 (73.3%) reported that they had noticed a decline of memory performance after the traumatic event (20% fairly, 20% severe, 33.3% very severe).

PTSD patients performed significantly worse than healthy controls on the (objective) short- and long-term verbal memory test PWMT (short-term memory: $t = -3.26$, df = 40, $p = .002$; long-term memory: $t = 2.59$, df = 40, $p = .02$). Current subjective memory performance and the two objective memory parameters correlated significantly ($r > .57$; $p \leq .05$).

A number of limitations should be brought to the readers’ attention. Most notably, the present findings rely on retrospective reports, which are vulnerable to confounding effects. However, the impact of major confounds, such as comorbid depression, openness, and estimated verbal intelligence, was controlled for and their influence was negligible.

The data revealed a significant correlation between subjective estimates of current memory performance and objective memory performance. This result indicates that patients who had reported a decline of memory performance after the trauma also performed significantly worse on the objective memory task adding to the validity of the IAPM. Admittedly, this relation has to be considered with some caution: the PWMT was applied to assess objective memory performance without relation to earlier levels of performance or changes of mnestic competence, whereas the self-report provides information about the subjective extent of aggravated memory performance after traumatisation.

The present results indicate that patients with PTSD do not differ from controls on several dimensions of estimated premorbid memory. This finding lends support to claims made in several animal studies that memory impairment follows the PTSD. Further research in this area that includes larger sample sizes of PTSD patients and traumatised persons without PTSD as well as prospective designs are needed to further validate the premorbid memory interview and provide insight into the “chicken and egg” problem of memory dysfunction in PTSD.

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References


<table>
<thead>
<tr>
<th>Domain (IAPM)</th>
<th>Analysis</th>
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<tbody>
<tr>
<td>Score</td>
<td>PTSD</td>
</tr>
<tr>
<td>Memory of names</td>
<td>3.48 (1.08)</td>
</tr>
<tr>
<td>Prospective memory</td>
<td>3.57 (0.93)</td>
</tr>
<tr>
<td>Narrative memory</td>
<td>3.10 (1.14)</td>
</tr>
<tr>
<td>Spatial memory</td>
<td>3.62 (1.24)</td>
</tr>
<tr>
<td>Dyslexia</td>
<td>3.29 (0.90)</td>
</tr>
<tr>
<td>Attention</td>
<td>3.10 (1.14)</td>
</tr>
</tbody>
</table>

*When preconditions for t-tests (normal distribution/homogeneity of variance) were not met, results were recalculated with the Mann-Whitney U-Test. In each case status of significance remained unchanged. Higher scores designate better performance.


Sapolsky RM. Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. Arch Gen Psychiatry 2000; 57: 925-935.