Verbal learning and memory impairments in posttraumatic stress disorder: The role of encoding strategies

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Abstract

The present study examined mechanisms underlying verbal memory impairments in patients with posttraumatic stress disorder (PTSD). Earlier studies have reported that the verbal learning and memory alterations in PTSD are related to impaired encoding, but the use of encoding and organizational strategies in patients with PTSD has not been fully explored. This study examined organizational strategies in 21 refugees/immigrants exposed to war and political violence who fulfilled DSM-IV criteria for chronic PTSD compared with a control sample of 21 refugees/immigrants with similar exposure, but without PTSD. The California Verbal Learning Test was administered to examine differences in organizational strategies and memory. The semantic clustering score was slightly reduced in both groups, but the serial cluster score was significantly impaired in the PTSD group and they also reported more items from the recency region of the list. In addition, intrusive errors were significantly increased in the PTSD group. The data support an assumption of changed memory strategies in patients with PTSD associated with a specific impairment in executive control. However, memory impairment and the use of ineffective learning strategies may not be related to PTSD symptomatology only, but also to self-reported symptoms of depression and general distress.

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1. Introduction

This study focuses on mechanisms underlying verbal memory alterations in refugees/immigrants with posttraumatic stress disorder (PTSD) after war exposure. PTSD is classified as an anxiety disorder and develops in some individuals as a response to traumatic stress and is characterized by three main clusters of symptoms: intrusive reexperiencing, avoidance behaviors, and hyperarousal (American Psychiatric Association, 1994). There is evidence that PTSD is associated with distinct brain dysfunction patterns and cognitive impairments (Elzinga and Bremner, 2002), and verbal learning and memory alteration have consistently been documented (e.g., Bremner et al., 2004; Gilbertson et al., 2001). In addition aspects of attention and memory dependent on executive control appear to be impaired in individuals with PTSD (Kanagaratnam and Asbjørsen, 2007; Vasterling et al., 1998).

Previous studies on the relationship between verbal memory impairments and PTSD have reported both acquisition impairment (Yehuda et al., 2005b), and
recall impairments related to an reduced ability to consolidate memory material (e.g., Brewner et al., 1995; Uddo et al., 1993). Lately, studies have been designed to examine more explicitly the relation between stage of processing and memory impairment. Two recent studies have provided evidence that impairment in encoding accounted for the verbal memory impairments in PTSD patients when controlling for attention and the effect of total learning (Yehuda et al., 2005a; Yehuda et al., 2004). In addition, we previously found evidence that learning and memory impairments in refugees with PTSD were related to the ability to elaborate memory material to facilitate a more efficient encoding (Johnsen et al., 2008). In that study, we found no differences between a diagnosed PTSD group and a control group on memory span, but less effective learning emerged over trials on a standardized verbal learning test. Controlling for measures of attention ruled out the possibility that the difference in verbal learning and memory was secondary to attentional dysfunction. No impairments on tests of recognition were found. This suggested a pattern of memory impairment secondary to difficulties using effective learning or organizational strategies (Sternberg and Tulving, 1977) during encoding. The above-mentioned studies did not analyze organizational strategies. Jenkins et al. (1998) reported that although their PTSD sample did not seem to be impaired on acquisition over learning trials, they were impaired at short and long delay recalls. Follow-up tests revealed no differences in semantic and serial organizational strategies. Two previous studies have analyzed recall errors, which may indicate executive failure. Both increased intrusive errors and perseverative responses were found on free recall of list-learning tests (Uddo et al., 1993; Vasterling et al., 1998).

PTSD has been seen as a failure to process and integrate trauma, and is described as an information processing disorder (Brewin et al., 1996). Decreased executive and inhibitory control of trauma memories, emotions, and impulses characterizes the disorder. The verbal memory impairments among trauma samples have been linked both to increased arousal state and intrusive reexperiencing phenomena (Kolb, 1987). If intrusive symptoms in PTSD reflect a general failure in executive control, with a deficit in the ability to suppress involuntary thoughts, it is assumed that this impairment also is seen on specific memory tests involving organizational strategies and executive control.

However, traumatized refugees exhibit a spectrum of clinical signs, and in addition to PTSD, major depressive disorder is common (Mollica et al., 1987). In a study of Bosnian refugees, comorbidity of PTSD and depression was related to high rates of psychosocial disability (Mollica et al., 1999). Most of the studies examining cognitive alteration in PTSD have been conducted on groups with coexisting psychiatric conditions (Vasterling et al., 2002; Yehuda et al., 2005b). Lately, there has been a growing awareness that complex and prolonged trauma may result in many posttraumatic outcomes (Bremner, 2002; van der Kolk et al., 2005). Some studies that have applied statistical control for comorbidity or concurrent conditions have suggested that PTSD makes an independent contribution to the group differences (Bremner et al., 2004; Gilbertson et al., 2001; Jelinek et al., 2006; Jenkins et al., 1998; Jenkins et al., 2000); other studies have found depressive symptoms in traumatized individuals to be related to the cognitive impairments (Brandes et al., 2002; Johnsen et al., 2008). In a prospective study, early depressive symptoms were found to predict PTSD better than early PTSD symptoms (Freedman et al., 1999). This makes it important to assess concurrent conditions such as depression, in order to understand if the underlying memory impairment and executive failure are specifically related to PTSD symptoms or to the depressive symptoms connected to it. Depression has been linked to impairment on tasks requiring effortful processing (Hasher and Zacks, 1979). In addition, general intellectual functioning has been found to be a risk factor for PTSD (Macklin et al., 1998) and controlling for IQ measures is necessary.

To date, neuropsychological studies of PTSD suggest that verbal memory impairments in patients with PTSD are related to problems with encoding and executive memory. However, the previous studies have focused mostly on the number of items recalled and to a lesser extent on qualitative aspects underlying the memory impairments such as intervening cognitive processes, e.g., organizational strategies and error types. The association between memory impairments and difficulties in implementing organizational strategies has been documented in other psychiatric disorders and brain disease (Gershberg and Shimamura, 1995; Lundervold et al., 1994; Savage et al., 2000). Thus, the question of whether the encoding impairments found in patients with PTSD are related to ineffective organizational strategies still remains to be answered. Studies documenting memory impairment in war-related PTSD have mostly been conducted on US war veteran samples, with only a few presenting data from civilian samples and different cultural groups. War veterans have been specially trained for challenging situations, which may affect their coping and later symptom development.
The aim of the present study is twofold. The first aim is to examine the use of memory-enhancing strategies such as semantic and serial clustering and to assess the implications of such strategies on differences in serial position effects and recall errors in refugees with PTSD compared with exposed refugees without PTSD. The second aim is to study how strategy use and recall errors are related to the symptomatology. The current study is based on the same sample as reported by Johnsen et al. (2008). Based on the previous findings of encoding and executive impairment, we hypothesized that learning and memory impairments in patients with PTSD would be mediated by difficulties in using effective semantic clustering strategies during encoding. Second, we hypothesized that the executive impairment would impact serial clustering, recall errors, and serial position effects. Third, we assumed that ineffective use of organizational strategies in the PTSD group could be explained by self-reported symptoms of PTSD, depression, general distress, and/or estimated IQ. If one of these conditions were an important contributor to the insufficient organization of the memory material, then there would be reduced group effects when the variables were introduced as covariates, and there is evidence that depressive symptoms, in addition to PTSD symptoms, would be expected to be important contributors.

2. Method

2.1. Participants

The sample consisted of 42 refugees/immigrants, 32 men and 10 women. They were mainly from the Middle-East, the former Yugoslavia and Chile, and were voluntary participants in the study. Twenty-one were diagnosed with chronic PTSD according to DSM-IV criteria following experiences with actions of war and political violence (the PTSD group). They were compared with a healthy control group of 21 refugees with the same exposure, but who had never developed PTSD (the non-PTSD group). Both groups were matched as closely as possible with regard to age, trauma exposure, and ethnicity profile. Gender distribution was 14:7 in the PTSD group and 18:3 in the non-PTSD group. The controls had no previous history of psychiatric illness.

Clinical assessment included The Mini-International Neuropsychiatric Interview (M.I.N.I.), version 5.0.0 (Sheehan et al., 1998). The M.I.N.I. was used to investigate the presence of PTSD and other psychiatric disorders. The PTSD diagnosis was given based on the Clinician Administered PTSD Scale for DSM-IV (CAPS; Blake et al., 1998). The CAPS examines all the diagnostic criteria for PTSD, including criterion A, both frequency and intensity of criteria B–D, criteria E and F. The Life Event Check List in the CAPS, which identifies the main trauma of the participants, confirmed that their PTSD syndromes were related to political violence and war events.

Participants were excluded from the study if they had organic brain damage or other neurological diseases. Those who had been involved in events that gave reason to expect brain injuries such as blows to the head or loss of consciousness exceeding 30 min were excluded from the study (Vasterling et al., 1998), as were subjects with a history of alcohol/substance abuse and psychotic disorders. For the PTSD participants, the diagnosis of major depressive disorder was not an exclusion criterion, and in the PTSD group all participants were diagnosed with a major depressive episode. Subjects with other diagnoses such as bipolar disorder, obsessive–compulsive disorder, generalized anxiety disorder, and panic disorder were excluded. Participants on tricyclic antidepressant, benzodiazepine, and neuroleptic medication were excluded. Participants on selective serotonin reuptake inhibitors (SSRIs) were included.

2.2. Procedure

The study was approved by the Regional Committee for Medical Research Ethics. Information about the study was given to health personnel in outpatient clinics and at health services for refugees/immigrants. Written information was posted at local public health services. Participants who had been exposed to war or political violence and who had a tentative PTSD diagnosis were either referred by counselors or recruited themselves. Exposed participants for the non-PTSD group were recruited by the same formal or informal contacts. The participants gave written informed consent prior to participation and were informed that participation was voluntary. All patients went through a diagnostic assessment administered by a licensed clinical psychologist experienced in traumatology. Participants received no economic compensation for taking part in the study. All the participants in the study were interviewed about their exposure to war and political violence, and special care was taken in the interviews and assessment procedures in order to avoid retraumatization.

The clinical self-report scales and the neuropsychological tests were administered in the same order for all the participants. Other tests were included in the overall neuropsychological battery, but these are not reported in the current study. All the test material and self-report
scales were presented in the participants’ native language, and the interviews and testing were done in collaboration between the clinician, a specially trained test technician, and an authorized interpreter. Efforts were made to use the same interpreters within the same language groups.

2.3. Clinical assessments

The Impact of Event Scale — Revised (IES-R), a 22-item self-report measure, was used to examine the severity of PTSD symptoms the week before testing (Weiss and Marmar, 1997). Current depressive symptoms were assessed with the Montgomery and Åsberg Depression Rating Scale (MADRS), a 10-item questionnaire measuring depressive symptoms in the last 3 days (Montgomery and Åsberg, 1979). The MADRS was used as a structured interview to examine depressive symptoms. General distress was measured with the Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 1983).

The subtests Similarities and Picture Completion from the Wechsler Adult Intelligence Scale-Revised were used to estimate current IQ level (Wechsler, 1981). Scores on the Similarities subtest have a strong correlation with general intelligence. Picture Completion is seen as a “hold” test, and is described as relatively stable and not so easily influenced by external factors. This test is thus expected to be an indicator of premorbid IQ, and both subscales have high loadings on the g-factor (Lezak, 1995).

The War Exposure Questionnaire (WEQ) was used to assess exposure to war and political violence. The WEQ is a 27-item self-report measure assessing potentially traumatic events such as threat, loss, injury, deprivation, grotesque impressions and other aspects of war and political violence. The WEQ is a modified version of the Childhood War Exposure Questionnaire, caretaker version (Netland and Kanaaneh, 1989).

2.4. Neuropsychological testing

The California Verbal Learning Test (CVLT; Delis et al., 1987) was administered for testing organizational strategies, serial position effects, and recall errors. The CVLT is a multi-trial serial learning test, and provides an assessment of multiple strategies and processes involved in learning and remembering verbal material. It also provides a measure of organization and learning strategy. The CVLT identifies two measures of organizational strategies: semantic and serial clustering. The test consists of two lists (list A and list B), each list containing 16 items in four different semantic categories. The two lists have two common semantic categories, but the category items are specific in each list. During the learning trials, the items are spoken aloud by the examiner in a fixed order, and no two consecutive items are derived from the same semantic category. In this way an evaluation of the degree to which items are grouped into semantic categories in the first five test trials (semantic clustering; “fruits” and “clothes”) or to which degree recall follows the order in which the items were presented (serial clustering) is possible. In order to assess semantic clustering, participants receive scores based on the number of words they recall from the same semantic category in succession. This score is corrected for the total number of words recalled.

List A is presented five times, and the participants are instructed to recall as many words as possible after each presentation of the list. After the five presentations of list A, a new list of words (list B) is presented to the participants, and they are instructed to recall as many words as they can from list B. Participants are then asked to recall list A again in short delay free recall (SDFR), and after a 20-minute interval they are asked to recall list A in long delay free recall (LDFR). The standard procedure includes the use of semantic cues to enhance recall (cued recall). The CVLT measures and indexes used in this study are presented in Table 2.

2.5. Statistical analyses

Group differences in demographic characteristics, clinical assessments, and neuropsychological test performance were analyzed using independent t tests. Differences in CVLT clustering scores were analyzed by analysis of variance (ANOVA). Correlation analyses were performed to examine the relationship between CVLT organizational strategy scores and clinical measures. In addition, an analysis of covariance (ANCOVA) was used to assess the influence of PTSD symptoms, depressive symptoms, general distress, and estimated IQ on organizational strategies and recall errors. To correct for multiple comparisons, Bonferroni tests were conducted.

3. Results

3.1. Characteristics of participants

Characteristics of the participants in this study are presented in Table 1. The two groups were similar in terms of age, years of education, estimated IQ, duration of exposure to war and political violence, and time
passed since their exposure. As would be expected, the PTSD group had higher scores on the measures of IES-R, MADRS and SCL-90-R.

3.2. Organizational strategies, serial position effects, and recall errors

Pearson product-moment correlations revealed no significant correlation between age and education and the cognitive measures. The performance of the two groups on the CVLT, organizational strategies, serial position effects, learning slope, recall consistency and recall errors are presented in Table 2.

An ANOVA with group as the between-groups factor, and coding strategy as a within-subjects factor revealed a significant main effect of groups, with the highest score for serial coding ($F_{(1,36)}=7.70, P=0.008$, see Fig. 1), but no interaction effect. The effect was rejected when using Bonferroni tests. The Least Significant Difference test showed significant differences between serial and semantic organization strategies within the non-PTSD group ($P=0.02$). When testing the effect of gender, a significant group by gender, interaction was found ($F_{(1,34)}=4.30, P=0.05$, see Fig. 2), and a nearly a three-way interaction by strategy, grouping, and gender ($F_{(1,34)}=3.99, P=0.053$). The next set of analyses showed that on the serial position effect of the CVLT list the PTSD group recalled significantly more recency words. The PTSD group was characterized by an increased number of intrusive errors. The effect sizes, using Cohen's $d$ for serial clustering, recency, and intrusions, were medium to large (Cohen, 1969). These findings are detailed in Table 2.

Table 1
Demographic and clinical characteristics and estimated intelligence for the PTSD group and the non-PTSD group

<table>
<thead>
<tr>
<th></th>
<th>PTSD group ($n=21$)</th>
<th>Non-PTSD group ($n=21$)</th>
<th>t-value</th>
<th>Effect size ($d$)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>36.90 (8.77)</td>
<td>39.24 (10.06)</td>
<td>−0.80</td>
<td>−0.25</td>
<td>n.s.</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12.12 (2.86)</td>
<td>14.05 (4.12)</td>
<td>−1.76</td>
<td>−0.56</td>
<td>n.s.</td>
</tr>
<tr>
<td>Exposure range (months)</td>
<td>83.14 (66.87)</td>
<td>100.81 (71.62)</td>
<td>−0.83</td>
<td>−0.26</td>
<td>n.s.</td>
</tr>
<tr>
<td>Time since exposure (months)</td>
<td>107.43 (96.00)</td>
<td>166.27 (109.66)</td>
<td>−1.71</td>
<td>−0.59</td>
<td>n.s.</td>
</tr>
<tr>
<td>MADRS$^a$</td>
<td>26.20 (6.93)</td>
<td>5.85 (4.21)</td>
<td>11.23</td>
<td>3.64</td>
<td>0.000</td>
</tr>
<tr>
<td>CAPS</td>
<td>73.76 (18.55)</td>
<td>n.a.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IES-R$^b$ Avoidance</td>
<td>18.29 (5.83)</td>
<td>8.10 (8.36)</td>
<td>4.54</td>
<td>1.45</td>
<td>0.000</td>
</tr>
<tr>
<td>IES-R Intrusion</td>
<td>20.81 (4.34)</td>
<td>8.15 (7.41)</td>
<td>6.71</td>
<td>2.15</td>
<td>0.000</td>
</tr>
<tr>
<td>IES-R Hyperarousal</td>
<td>20.29 (3.42)</td>
<td>6.60 (6.54)</td>
<td>8.45</td>
<td>2.71</td>
<td>0.000</td>
</tr>
<tr>
<td>IES-R Total score</td>
<td>59.38 (9.89)</td>
<td>22.85 (21.22)</td>
<td>7.12</td>
<td>2.28</td>
<td>0.000</td>
</tr>
<tr>
<td>IES-R GSI$^d$</td>
<td>2.70 (0.45)</td>
<td>1.09 (0.94)</td>
<td>7.03</td>
<td>2.25</td>
<td>0.000</td>
</tr>
<tr>
<td>SCL-90-R$^e$</td>
<td>190.95 (56.55)</td>
<td>76.75 (52.35)</td>
<td>6.28</td>
<td>2.12</td>
<td>0.000</td>
</tr>
<tr>
<td>SCL-90-R GSI$^d$</td>
<td>2.12 (0.63)</td>
<td>0.85 (0.58)</td>
<td>6.27</td>
<td>2.12</td>
<td>0.000</td>
</tr>
<tr>
<td>Estimated IQ$^e$</td>
<td>82.19 (13.00)</td>
<td>91.90 (17.90)</td>
<td>−2.01</td>
<td>−0.64</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

$^a$ Montgomery and Åsberg Depression Rating Scale.
$^b$ The Impact of Event Scale — Revised.
$^c$ Symptom Checklist — Revised.
$^d$ GSI = General Symptom Index.
$^e$ Estimated IQ from the WAIS-R subtests Picture Completion and Similarities.

Table 2
Cognitive measures for the PTSD group and the non-PTSD group

<table>
<thead>
<tr>
<th></th>
<th>PTSD group ($n=21$)</th>
<th>Non-PTSD group ($n=21$)</th>
<th>t-value</th>
<th>Effect size ($d$)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVLT 1–5</td>
<td>41.75 (13.61)</td>
<td>52.83 (7.26)</td>
<td>−3.08</td>
<td>−1.03</td>
<td>0.004</td>
</tr>
<tr>
<td>Semantic clustering</td>
<td>1.44 (0.66)</td>
<td>1.65 (0.68)</td>
<td>−0.99</td>
<td>−0.33</td>
<td>n.s.</td>
</tr>
<tr>
<td>Serial clustering</td>
<td>1.68 (1.03)</td>
<td>2.49 (1.28)</td>
<td>−2.17</td>
<td>−0.72</td>
<td>0.03</td>
</tr>
<tr>
<td>Learning slope</td>
<td>1.02 (0.56)</td>
<td>1.31 (0.60)</td>
<td>−1.48</td>
<td>−0.49</td>
<td>n.s.</td>
</tr>
<tr>
<td>Consistency</td>
<td>73.66 (19.22)</td>
<td>81.63 (8.87)</td>
<td>−1.61</td>
<td>−0.54</td>
<td>n.s.</td>
</tr>
<tr>
<td>Perseverations</td>
<td>3.10 (2.59)</td>
<td>3.61 (3.79)</td>
<td>−0.49</td>
<td>−0.16</td>
<td>n.s.</td>
</tr>
<tr>
<td>Intrusions</td>
<td>2.45 (3.50)</td>
<td>0.61 (1.46)</td>
<td>2.07</td>
<td>0.69</td>
<td>0.05</td>
</tr>
<tr>
<td>Primary %</td>
<td>27.25 (6.73)</td>
<td>23.67 (5.03)</td>
<td>1.84</td>
<td>0.61</td>
<td>n.s.</td>
</tr>
<tr>
<td>Middle %</td>
<td>41.96 (10.52)</td>
<td>50.26 (3.90)</td>
<td>−3.15</td>
<td>−1.05</td>
<td>0.003</td>
</tr>
<tr>
<td>Recency %</td>
<td>30.79 (7.23)</td>
<td>26.06 (4.40)</td>
<td>2.40</td>
<td>0.80</td>
<td>0.02</td>
</tr>
</tbody>
</table>
3.3. Relationship between encoding problems and symptomatology

Pearson product-moment correlations revealed no significant correlations between semantic clustering, clinical measures (IES-R, MADRS, SCL-90-R), and estimated IQ. Serial clustering correlated with IES-R ($r=0.39$, $P<0.05$), MADRS ($r=0.40$, $P<0.05$), SCL-90-R ($r=0.34$, $P<0.05$), and estimated IQ ($r=0.42$, $P<0.05$).

To analyze the relative importance of self-reported symptoms of PTSD (IES-R), depression symptoms (MADRS), general distress (SCL-90-R) and estimated IQ on semantic and serial clustering, a covariate analysis (ANCOVA) was performed with these variables as covariates. When controlling for IES-R, the group differences for semantic and serial clustering disappeared ($F_{(1,35)}=1.46$, $P=0.23$). This analysis revealed that self-reported PTSD symptoms had the greatest effect on serial clustering in the PTSD group. When MADRS scores were controlled for, the differences between the groups on semantic and serial clustering disappeared ($F_{(1,34)}=0.05$, $P=0.83$), and the self-reported depressive symptoms had a stronger effect than PTSD symptoms on the serial coding in the PTSD group. When SCL-90-R scores were controlled for, the difference between the groups was reduced ($F_{(1,32)}=2.60$, $P=0.12$), and the general distress symptoms had the strongest effect on semantic clustering in the non-PTSD group. When estimated IQ was controlled for, the differences between the groups persisted ($F_{(1,35)}=4.91$, $P<0.03$).

In order to test the relative importance of the three PTSD symptom clusters on organizational clustering, we used covariance analyses. When the effects of the Intrusion and the Arousal scales were removed, the between-groups differences of organizational clustering disappeared ($F_{(1,35)}=0.61$, $P=0.44$ and $F_{(1,35)}=1.78$, $P=0.19$).
Since depressive symptoms had a strong effect on the use of organizational strategies, we also performed a covariate analysis with MADRS as the control variable on intrusive errors. The analysis yielded an interaction effect \((F_{(1,34)}=5.13, P=0.03\), with reduced intrusions in the PTSD group, and increased intrusions in the non-PTSD group when the effect of the MADRS score was removed.

### 4. Discussion

The main findings in this study were less serial organization, more intrusive errors, and more pronounced recency effect in patients with PTSD compared with exposed controls during learning and memorizing verbal material. This contributes to our understanding of impaired verbal learning and memory associated with PTSD. Age, education, and estimated intelligence did not explain the findings, since the participants were matched on these variables. These results disputes Jenkins et al’s (1998) study showing no differences on organizational strategies using the CVLT.

The hypothesis of less effective semantic coding in the PTSD group was not supported. Compared with norms for the CVLT, both groups were below average for semantic clustering (Delis et al., 1987). Since both groups are slightly impaired, we cannot say if they have an intact semantic organization. The reduced scores seen may partly be related to the different use of the semantic categories within the different languages used by the participants in this study. Both a Norwegian validation of the norms (Egeland et al., 2005) and the American norms (Delis et al., 1987) indicate more serial coding in men and more semantic coding in women. This may also have influenced our results as our samples include more men.

Both the males and the females in the control group had scores on encoding strategies as expected, with males using more serial clustering and females more semantic clustering. Interestingly, the females in the PTSD group used more serial clustering, which indicates that women under stress use less sophisticated encoding strategies (serial clustering) than the controls. The men in the PTSD group had difficulties with both strategies. To our knowledge there are no published studies on gender differences in verbal memory or encoding strategies in PTSD samples, but women tend to perform better on verbal learning and memory in normal samples (Kramer et al., 1988). Our finding related to gender is significant despite low power, and it should be tested further.

The increased recency score in the PTSD group compared with the non-PTSD group could indicate a tendency to use a passive recall strategy of echoing back the last items presented. In addition intrusive responses dominated the PTSD patients, so the results confirmed previous studies in war veterans (Vasterling et al., 1998), suggesting that intrusion in PTSD is not limited to trauma-related cognitions, but reflects a more general pattern of impaired inhibition and cognitive control. In addition, the depression score after war exposure explains an extensive part of the variation in intrusive errors in the PTSD group. Monitoring describes the ability to control the verbal output, and this function was affected differently in the two groups, and was also differently related to comorbid depression. When controlling for the MADRS score was controlled for, intrusive errors were reduced in the PTSD group, but increased in the control group. This could suggest that the control group maintained executive control in the test situation, whereas those who developed PTSD as a clinical syndrome did not. Maintained executive control may act as a protective mechanism after trauma exposure. Deficits in verbal memory shortly after the traumatic event have been related to a greater risk for developing PTSD (Bustamante et al., 2001). Furthermore, the covariance analysis suggested that IES-R and MADRS scores had a relatively large impact on serial clustering in the PTSD group, especially the MADRS score. In addition, the SCL-90-R score had an important effect on semantic clustering in the control group. This suggests that encoding deficits are mainly related to depressive symptoms, and not directly to symptoms unique to PTSD. This finding is consistent with the findings of Brandes et al. (2002), who reported that cognitive impairments in PTSD were related to depressive symptoms, although other studies have reported that PTSD makes an independent contribution to the memory impairments (e.g., Bremner et al., 2004; Gilbertson et al., 2001; Jenkins et al., 1998). These seemingly contradictory findings may be related to methodological differences, as tasks, samples, and diagnostic criteria vary. The PTSD group in the present study had relatively high scores on current depressive symptoms (MADRS). In their country of origin they were all exposed to complex and prolonged trauma, involving threat to life as well as loss. Complicated adaptations to severe and prolonged trauma have been described as both complex trauma (Herman, 1992) and Disorders of Extreme Stress Not Otherwise Specified (DESNOS; Roth et al., 1997) where posttraumatic problems other than PTSD are included. Van der Kolk et al. (2005) have been critical of the PTSD literature, which discusses psychiatric symptoms that do not fall within the...
framework of PTSD as comorbid conditions, as if they occur independently from the PTSD symptoms. It is also possible that overlapping symptoms in our PTSD and depression measures contribute to our findings, but viewing the depressive symptoms as independent of the PTSD symptoms may not be valid, since PTSD captures only a limited aspect of posttraumatic psychopathology.

When analyzing the impact of PTSD on cognitive functioning, one has to discuss what a relevant comparison group might be. Our control group was exposed to the same traumatic events as the PTSD group, but had not developed a PTSD diagnosis. In addition, they were sharing the same strain as being a refugee and therefore could not be assumed to be asymptomatic (Hondius et al., 2000). According to Søndergaard et al. (2001), refugees are influenced to a great extent by political events and the situation of significant others in the home country. However, this also strengthens the possibility to analyze the unique cognitive patterns of PTSD, since confounding factors to a large extent are controlled for.

The finding of impaired organizational strategies in PTSD is intriguing. However, functional neuroimaging studies are showing a significant relationship between the use of semantic organization during learning and recall and frontal lobe activation in healthy controls (Fletcher et al., 1998). Our results suggest that intrusive and arousal symptoms may account for difficulties in using serial organizational strategies. Organization of information during encoding challenges the executive processes, and these functions are associated with the left prefrontal cortex (Fletcher et al., 1998). Structural and functional neuroimaging studies have shown dysfunction in both prefrontal and temporal cortex in patients with PTSD (Kitayama et al., 2005; Shin et al., 2001), although support for hippocampus alterations is not found in all studies (Pederson et al., 2004; Schuff et al., 2001). Further studies should assess whether patients with PTSD also show impaired use of organizational strategies on other cognitive tasks in order to determine the generalizability of the organizational dysfunction. Given the possible source of errors connected to the different use of semantic categories within the different language and ethnic groups participating in this study, organizational strategies underlying memory impairments should be examined further in a more homogeneous group with more balanced distribution between the sexes.

There are some important limitations in this study that should be mentioned. First, the sample was small, which may limit the generalizability of our findings. The findings need to be confirmed in larger samples. Second, it is problematic to use standardized tests with a translator. Even when the stimulus words are translated by authorized translators, this procedure is not fully equivalent to using a validated translation of the test. However, this also strengthens the relevance of the study in a multi-ethnic clinical setting. Efforts were made to use the same interpreters within the same language groups, and the study used a between-group design with control for ethnicity, which should reduce the probability of a systematic bias in our conclusion. Both groups are in the lower range on measures of IQ, and one can only speculate on the possible explanation for this. The most commonly proposed factors contributing to variance on IQ measures within different cultural groups are education, characteristics of the test, familiarity with stimuli and test situations in general (Berry et al., 2002). PTSD has in addition been associated with low premorbid intelligence (Macklin et al., 1998), but the lower intelligence scores in the PTSD group may also be connected to the development of PTSD and to mild related information-processing deficits (Gil et al., 1990).

Our findings suggest that future research with PTSD in this area should focus on specific components of the memory system, especially executive control of memory. The implication of the present findings, however, is that memory impairments in refugee groups might be associated with one or more strain factors common to various forms of posttraumatic psychopathology.

To conclude, we have found that impaired memory in PTSD can be explained by ineffective use of memory-enhancing strategies, and is not limited to memory capacity. This finding is important to both theory development and clinical work with patients with PTSD. An inefficient memory system is likely to be a key factor affecting the individuals with PTSD’s ability to function educationally and occupationally. Learning and memory alteration in refugees may affect their lives in the host country with regard to learning the new language and their ability to participate in education and employment. Memory impairment has not been sufficiently focused on in the rehabilitation of patients with PTSD, and deficient organization and strategy use should be targeted within the clinical educational work with these patients.

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References


