

PTSD symptoms and cognitive performance in recent trauma survivors

Dalia Brandes^{a,b}, Gershon Ben-Schachar^b, Assaf Gilboa^a, Omer Bonne^a, Sara Freedman^a,
Arieh Y. Shalev^{a,*}

^aCenter for Traumatic Stress, Department of Psychiatry, Hadassah University Hospital, P.O. Box 12000, Jerusalem, Israel

^bDepartment of Psychology, Hebrew University, Jerusalem, Israel

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Abstract

Chronic post-traumatic stress disorder (PTSD) has been associated with cognitive impairments involving memory and attention. The association between cognitive impairment and early PTSD symptoms is unknown, yet such association may lead to poorer processing of traumatic memories and thereby contribute to subsequent PTSD. This study evaluated the relationship between PTSD symptoms and cognitive functioning within 10 days of traumatic events. Forty-eight survivors were assessed for symptoms of PTSD, anxiety, depression and dissociation and for immediate and delayed verbal and figural memory, attention, learning and IQ. Survivors with high levels of PTSD symptoms showed impaired attention and immediate recall for figural information and lower IQ. They did not show, however, an impairment of verbal recall and learning. The observed difference was not explained by anxiety or dissociation. It disappeared, however, when the effect of depressive symptoms was controlled for. Lower IQ and impaired attention are associated with early PTSD and depressive symptoms. Poorer attention may have a role in shaping traumatic memories. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

Intrusive recall and partial forgetfulness of the traumatic event are cardinal features of post-traumatic stress disorder (PTSD; American Psychiatric Association, 1994). Additionally, the disorder has been associated with subjective complaints of poor concentration and memory (Klonoff et al., 1976;

Goldfield et al., 1988; Weiner, 1992; Uddo et al., 1993), and with biased allocation of attention towards threatening stimuli (McNally et al., 1990; Vrana et al., 1995; Semple et al., 1996).

Beyond trauma-related memory, PTSD has been associated with measurable impairment of memory and attention, and with lower IQ (for review, see Wolfe and Schlesinger, 1997; Buckley et al., 2000). Studies to date point to two hypothetical domains of cognitive impairment in PTSD, namely, impaired *declarative memory* (e.g. Bremner et al.,

*Corresponding author. Tel.: +972-2-6777184; fax: +972-2-6413642.

E-mail address: ashalev@cc.huji.ac.il (A.Y. Shalev).

1993, 1995) and impaired *attention* and *working memory* (e.g. Uddo et al., 1993; Vasterling et al., 1998).

Specifically, Bremner et al. (1993) found poorer immediate and delayed verbal memory in 26 Vietnam veterans with PTSD compared with 15 healthy controls. Bremner et al. (1995) found poorer immediate and delayed verbal memory among 21 survivors of childhood abuse with PTSD, compared to 20 matched controls. Significant correlation between verbal memory and trauma severity was found in both studies. Yehuda et al. (1995) found higher proactive interference with memory acquisition in PTSD. Uddo et al. (1993), who examined 16 PTSD Vietnam veterans and 15 regular soldiers with no history of trauma, found impaired learning, perseverative errors, poorer word fluency and impaired immediate visual memory among PTSD patients. Vasterling et al. (1998) showed deficiencies in sustained attention, mental manipulation, acquisition of new information, retroactive interference, and errors of commission and intrusion in 19 Gulf War veterans with PTSD compared with 24 mentally healthy veterans.

Findings of cognitive deficits in PTSD may reveal the involvement of specific brain areas in this disorder. Accordingly, impaired declarative memory has been attributed to hippocampal dysfunction, which in PTSD may reflect a damaging effect of chronic stress on hippocampal cells (e.g. Bremner et al., 1995; McEwen, 2000). Deficits in attention and working memory have been interpreted as suggesting an involvement of the prefrontal lobes (e.g. Vasterling et al., 1998). Specific anatomical allocations, however, may not reflect the complexity and interconnectedness of the neural systems that sub-serve memory and attention.

Previous cognitive studies concerned chronic PTSD. As such, they could not reveal the origin of their findings. Cognitive impairments may precede the onset of PTSD, or develop along with the disorder. The former hypothesis received indirect support from studies showing lower pre-military IQ in Vietnam veterans who developed PTSD (e.g. Macklin et al., 1998). A hypothetical deficiency of declarative memory, at the early aftermath of a traumatic event, may result from

stress-induced hormonal inhibition of hippocampal functions (e.g. McEwen, 2000).

Exploring the association between early PTSD symptoms and cognitive impairment is compelling. Subjects who express high levels of PTSD symptoms in the early aftermath of traumatic events are at higher risk for developing PTSD (e.g. Shalev et al., 1997). A cognitive impairment may contribute to this link: cognitive deficits may interfere with recovery from mental trauma, a process that requires re-learning and adaptation (Shalev, 2000). Early cognitive deficits may also lead to impaired acquisition of traumatic memories. In contrast, the absence of early cognitive dysfunction, among symptomatic survivors, will suggest that cognitive impairments develop along with PTSD.

This study evaluated the link between PTSD symptoms and cognitive functioning, 10 days after a traumatic event. At such time, a formal diagnosis of PTSD cannot be made, yet some survivors already express high levels of PTSD symptoms whereas others do not. The study evaluated attention, learning, memory and IQ, as well as symptoms of depression, dissociation and anxiety. The latter were used to probe the specificity of the hypothesised association between early PTSD symptoms and cognitive dysfunction.

2. Methods

2.1. Subjects

Forty-eight survivors (28 women and 20 men) were recruited, by telephone, within 10 days of their release from an emergency room of a large public hospital in Jerusalem following a traumatic event. The traumatic events consisted of 37 motor vehicle accidents, five terror attacks, three physical assaults, two home or work accidents and one rape. Survivors whose age was between 20 and 55 and whose traumatic events met DSM-IV PTSD criterion 'A' (exposure and intense response) were invited to participate in the study. Subjects were not included in the study if they suffered from head injury or loss of consciousness during the traumatic event, or if they had a history of neurological disorder, substance abuse, mental retardation or psychosis. Consenting subjects signed an

informed consent and were assessed by an experienced clinical psychologist (DB) within 10 days of the traumatic event. The assessment included psychometrics and neuropsychological tests (detailed below). Subjects were paid \$50 for their participation.

2.2. Psychometric instruments

These included Hebrew versions of *The Impact of Event Scale-Revised* (IES; Horowitz et al., 1979; Weiss and Marmar, 1997), *the State-Trait Anxiety Inventory* (STAI-State; STAI-trait; Spielberger et al., 1970), *Beck Depression Inventory* (BDI; Beck et al., 1961) and the *Peritraumatic Dissociation Experiences Questionnaire* (PDEQ; Marmar et al., 1995). The latter is an eight-item self-report questionnaire, assessing subjects' recall of dissociation experiences during the traumatic event. All the instruments have been widely used in studies of PTSD, including in recent survivors (e.g. Shalev et al., 1997; Freedman et al., 1999).

2.3. Neuropsychological tests

These included validated Hebrew versions of the following: (a) information, picture completion, block design, digit span, similarities and digit symbol sub-scales of the *Wechsler Adult Intelligence Scale Revised* (WAIS-R; Wechsler, 1981); (b) mental control, logical memory, memory span, visual reproduction and associate learning, sub-scales of the *Wechsler Memory Scale* (WMS; Wechsler and Stone, 1974); and (c) *Rey's Auditory Verbal Learning Test* (AVLT, Vakil and Blachstein, 1993).

According to previously recommended guidelines (e.g., Spreen and Strauss, 1998; Lezak, 1995), the results of the above were grouped into the following seven cognitive clusters:

Verbal memory—immediate: WMS logical memory—immediate recall and AVLT-1 (immediate recall of a word list). *Verbal memory—delayed*: WMS logical memory—delayed recall, WMS logical memory percent retention and the eighth trial of the AVLT (delayed recall of a word list). *Figural memory—immediate*: WMS reproduction—immediate. *Figural memory—delayed*: WMS

reproduction—delayed and WMS reproduction percent retention. *Attention*: WMS digit span—forward and backward and WAIS-R-digit symbol. *Learning*: sum of the first five trials of the AVLT. *IQ*: an extrapolation of IQ from six sub-tests of the WAIS-R (information, digit span, block design, digit symbol, picture completion, similarities).

2.4. Statistical analysis

The median IES score for the whole group was 65.5 (range 20–96). This median score was used to separate the cohort into those expressing *high levels of PTSD symptoms* ($n=14$, mean IES score=77.7) and those expressing *low levels of PTSD symptoms* ($n=14$, mean IES score=47.3.)

Multivariate analysis of variance (MANOVA) was used to compare the groups with high and low PTSD symptoms on all cognitive clusters. Analysis of covariance (ANCOVA) was used to control for the effects of anxiety, dissociation and depressive symptoms on the relationship between PTSD symptoms and cognitive performance. To examine further the relations between PTSD symptoms and cognitive functioning, Pearson correlation coefficients between the IES score and the cognitive sub-tests were computed.

3. Results

The study groups had similar age, gender, type of traumatic event and STAI-trait scores (Table 1). Subjects with high levels of PTSD symptoms had lower level of education and higher STAI-state, BDI and PDEQ scores.

Subjects with high levels of PTSD symptoms had lower scores on figural memory (immediate and delayed recall) and lower IQ (Table 2). The groups, however, had similar percent retention of figural memory.

Subjects with high levels of PTSD symptoms also showed poorer attention (digit span—backward and forward, and digit symbol). The groups did not differ in verbal memory (immediate recall, delayed recall and percent of retention) or in learning.

Very similar results were obtained when Pearson's correlation statistics were used: PTSD symp-

Table 1
Mean (and standard deviation) of background and psychometric variables

Variable	High PTSD symptoms (<i>n</i> = 24)	Low PTSD symptoms (<i>n</i> = 24)	<i>t</i> (d.f. = 1,46) χ^2	<i>P</i>
Age	34.8 (9.4)	30.6 (8.3)	<i>T</i> = 1.65	n.s.
Gender (M/F)	9/15	11/13	χ^2 = 0.3	n.s.
Type of trauma (MVA ^a /other)	17/7	20/4	χ^2 = 1.06	n.s.
Education level ^b	11.4 (3.1)	13.1 (2.7)	<i>T</i> = 2.07	0.04
IES ^c -total	77.67 (7.9)	47.25 (13.6)	<i>T</i> = 9.49	0.000
BDI ^d	22.9 (6.7)	12.5 (6.10)	<i>T</i> = 5.57	0.000
PDEQ ^e	29.6 (6.1)	23.4 (8.4)	<i>T</i> = 2.89	0.006
STAI ^f -state	57.9 (12.5)	47.6 (10.7)	<i>T</i> = 3.07	0.004
STAI ^f -trait	41.2 (8.4)	36.8 (9.7)	<i>T</i> = 1.68	0.10

^a Motor vehicle accidents.

^b No. of years studied.

^c Impact of Event Scale.

^d Beck Depression Inventory.

^e Peri Traumatic Dissociation Questionnaire.

^f State-Trait Anxiety Inventory.

toms significantly correlated with measures of attention, immediate figural memory, delayed figural memory and IQ (Table 3).

The observed group differences remained statistically significant after correction for state anxiety by means of ANCOVA (for figural memory-immediate recall: $F = 7.20$, d.f. = 1,44, $P < 0.01$; for figural memory—delayed recall: $F = 3.61$, d.f. = 2,43, $P < 0.04$; for attention: $F = 4.78$, d.f. = 3,43, $P < 0.004$). The observed group differences remained statistically significant when the effect of peri-traumatic dissociation (PDEQ) was controlled by means of ANCOVA (for figural memory-immediate recall: $F = 8.51$, d.f. = 1,44, $P < 0.01$; for figural memory-delayed recall: $F = 3.53$, d.f. = 2, 43, $P < 0.05$; for attention: $F = 2.75$, d.f. = 3, 43, $P < 0.06$).

When the effect of depressive symptoms (BDI scores) was controlled by means of ANCOVA, however, the previously observed group differences were not statistically significant (for figural memory-immediate recall $F = 0.96$; for figural memory-delayed recall: $F = 1.01$, d.f. = 2, 43, $P < 0.37$; for attention: $F = 1.59$, d.f. = 3, 43, $P < 0.21$).

4. Discussion

The results of this study show that individuals who express high levels of early PTSD symptoms

have poorer attention and lower IQ. The results of this study do not show an association between early PTSD symptoms and either learning or verbal memory. Additionally, this study shows that poorly acquired information is normally retained by subjects with high levels of PTSD symptoms (i.e. the groups showed similar percents of retention). This finding suggests that attentional difficulties may affect the long-term recollections of traumatic events.

Our results are in line with previous observations of attention deficit in PTSD (Gilbertson et al., 1997; Vasterling et al., 1998) and with those showing lower pre-military IQ in Vietnam veterans with PTSD (Macklin et al., 1998). However, because subjects' performance 10 days following a traumatic event may differ from that obtained under normal circumstances, the IQ scores obtained here may not represent a pre-traumatic trait measure. Deficits in learning and verbal memory (e.g. Bremner et al., 1995; Yehuda et al., 1995) may develop during the course of chronic PTSD.

The association between early PTSD symptoms and cognitive performance was not accounted for by differences in anxiety and dissociation symptoms. Impaired cognitive performance, therefore,

Table 2
Cognitive measures of subjects with high and low PTSD symptom levels

Variables	High PTSD symptoms (<i>n</i> = 24)	Low PTSD symptoms (<i>n</i> = 24)	<i>F</i> (d.f.)	<i>P</i> <
Attention			3.39 (3.43) ^a	0.03
WMS digit span—forward	5.75 (2.15)	7.21 (2.15)	5.53 (1.46) ^b	0.03
WMS digit span—backward	4.58 (1.72)	6.29 (2.12)	9.43 (1.46) ^b	0.005
WAIS-R—digit symbol	7.75 (2.09)	9.35 (2.89)	4.76 (1.45) ^b	0.05
Verbal memory—immediate			1.02 (2.45) ^a	0.37
WMS—logical—immediate recall	11.10 (3.41)	12.33 (3.31)	1.61 (1.46) ^b	n.s.
AVLT 1—immediate recall of list	6.25 (1.65)	6.96 (2.18)	1.62 (1.46) ^b	n.s.
Verbal memory—delayed			0.61 (3.44) ^a	n.s.
WMS—logical: delayed recall	9.44 (3.77)	10.56 (3.89)	1.04 (1.46) ^b	n.s.
WMS—logical: percent retention	0.84 (0.12)	0.83 (0.15)	0.09 (1.46) ^b	n.s.
AVLT 8: delayed recall	12.13(2.23)	11.38 (2.83)	1.04 (1.46) ^b	n.s.
Figural memory—immediate				
WMS—reproduction: immediate recall	8.22 (4.14)	11.25 (2.96)	8.37 (1.45) ^a	0.006
Figural memory—delayed			3.63 (2.44) ^a	0.03
WMS—reproduction: delayed recall	7.13 (4.57)	10.13 (3.47)	6.45 (1.45) ^b	0.02
WMS—reproduction: percent retention	0.83 (0.31)	0.89 (0.22)	0.62 (1.45) ^b	n.s.
Learning				
AVLT sum 1-5	59.19 (10.44)	61.13 (6.98)	0.53 (1.42) ^a	n.s.
IQ				
WAIS-R sum	87.74 (9.63)	97.00 (12.57)	7.87 (1.44) ^a	0.007

^a MANOVA.

^b Post-hoc ANOVA.

One subject did not complete the WAIS-R. AVLT=Rey's Auditory Verbal Learning Test; AVLT 1=First (immediate) recall; AVLT 8=Eighth (delayed) recall; WAIS=Wechsler Adult Intelligence Scale; WMS=Wechsler Memory Scale.

is not a simple correlate of anxiety, nor does it result from a trend to develop dissociation symptoms during stress.

The relationship between PTSD symptoms and poorer cognitive performance was accounted for by concurrent depressive symptoms. PTSD and depression are very often co-morbid, including in the early aftermath of traumatic events (e.g. Shalev et al., 1998). In that sense, both clusters of symptoms may represent a unique and common factor. Yet, prospective studies have shown that early depressive symptoms contribute to the occurrence of PTSD beyond the contribution of early PTSD symptoms (e.g. Freedman et al., 1999). Additionally, depressive symptoms independently affect cognitive performance (McAllister-Williams et al., 1998). The specific contribution of depression to

cognitive dysfunction among traumatised survivors should be further explored.

This study is limited by sample size and by the fact that the tests were performed at one point in time. Another apparent limitation of this study is the use of continuous symptoms measures instead of the existing diagnostic category of Acute Stress Disorder (DSM-IV). The latter, however, has been criticised for being both restrictive and heuristically unnecessary (e.g. Marshall et al., 1999). Furthermore, the extent to which symptoms expressed at the early aftermath of traumatic events should be grouped into syndromes is debatable, given the enormous fluidity of these symptoms (e.g. Shalev, 2002). The inclusion, in this study, of measures of PTSD and dissociation does capture the essential elements of ASD.

Table 3
Correlation between cognitive clusters and PTSD symptoms

Cognitive clusters:	Attention		Verbal memory immediate		Verbal memory delayed		Figural memory immediate		Figural memory delayed		Learning IQ	
	WMS Digit span forward	WMS Digit span backward	WMS Logical immediate recall	AVLT 1 Word list immediate recall	WMS Logical delayed recall	AVLT 8 delayed recall	WMS Logical percent retention	WMS Reproduction immediate recall	WMS Reproduction delayed recall	WMS Reproduction percent recall	AVLT Sum 1–5	WAIS-R Sum
IES—intrusion	-0.27	-0.31 ^a	-0.14	0.04	-0.05	0.17	0.11	-0.28	-0.23	-0.01	0.01	-0.19
IES—avoidance	-0.26	-0.21	-0.17	-0.29 ^a	-0.06	-0.12	0.10	-0.29 ^a	-0.25	-0.09	-0.27	-16
IES—arousal	-0.43 ^c	-0.44 ^c	-0.20	-0.03	-0.14	0.10	0.01	-0.38 ^b	-0.26	0.07	-0.05	-0.33 ^a
IES—total	-0.42 ^c	-0.42 ^c	-0.23	-0.12	-0.11	0.07	0.09	-0.42 ^c	-0.32 ^a	-0.01	-0.13	-0.29 ^a
BDI	-0.43 ^c	-0.37 ^b	-0.14	-0.20	-0.17	0.02	-0.05	-0.51 ^c	-0.37 ^b	0.07	0.08	-0.34 ^a
STAI—state	-0.06	-0.02	0.16	0.13	0.16	0.27	0.12	-0.14	-0.02	0.22	0.29 ^a	-0.01
PDEQ	-0.26	-0.17	-0.06	-0.006	-0.03	0.37 ^a	0.02	-0.07	-0.04	0.09	0.06	-0.15

^a $P < 0.05$.

^b $P < 0.01$.

^c $P < 0.005$.

AVLT = Rey's auditory verbal learning test; AVLT 1 = First (immediate) recall; AVLT 8 = Eighth (delayed) recall; WMS = Wechsler Memory Scale; IES = Horowitz's Impact of Event Scale (revised); BDI = Beck Depression Inventory; STAI = State-Trait Anxiety-State; PDEQ = Peritraumatic Dissociation Experience Questionnaire.

Most subjects who express early PTSD symptoms recover (e.g. Kessler et al., 1995). Prospective studies are therefore needed to evaluate the extent to which early cognitive difficulties interfere with the recovery from early PTSD symptoms. From a practical point of view, cognitive difficulties in the early aftermath of traumatic events should be acknowledged in planning early treatment interventions.

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