The Longitudinal Course of Posttraumatic Stress Disorder Symptom Clusters Among War Veterans

Zahava Solomon, Ph.D.; Danny Horesh, M.A.; and Tsachi Ein-Dor, M.A.

Objective: The aim of this study was to examine the long-term trajectories and interrelationships of posttraumatic stress disorder (PTSD) symptom clusters (intrusion, avoidance, and hyperarousal) in clinical and nonclinical groups of war veterans.

Method: Six hundred seventy-five Israeli veterans from the 1982 Lebanon War were assessed. The clinical group consisted of 369 who had combat stress reaction (CSR) during the war, and the nonclinical group consisted of 306 veterans with no antecedent CSR. The 2 groups were matched in age, education, military rank, and assignment. They were prospectively evaluated 1, 2, and 20 years after the war.

Results: The clinical group endorsed a higher number of symptoms than the nonclinical group, both cross-sectionally and across time. In both the clinical and nonclinical groups, the clusters of intrusion, avoidance, and hyperarousal were interrelated at any given point in time and across 20 years. In both groups, avoidance was found to be a particularly stable symptom cluster over time. Finally, hyperarousal levels 1 year after the war were found to play an important role in both groups, as they predicted future avoidance and intrusion symptoms.

Conclusions: The findings of this study suggest that PTSD is not a monolithic disorder, as symptom clusters differ in several important aspects. Also, the course and severity of symptoms differ between clinical and nonclinical groups. Finally, practitioners are encouraged to focus on the identification and treatment of early hyperarousal due to its prominent role in the development of other PTSD symptoms.

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Corresponding author and reprints: Zahava Solomon, Ph.D., Bob Shappell School of Social Work, Tel Aviv University, Tel Aviv, Israel 69978 (e-mail: solomon@post.tau.ac.il).

Despite substantial research on the phenomenology of posttraumatic stress disorder (PTSD) symptoms, significant gaps remain in current understanding of the psychological sequelae of traumatic events. The natural course of symptom formation and the dynamic interplay of symptoms over time are not yet fully understood. The aim of this study is to prospectively examine the trajectories and interrelationships of PTSD symptoms in clinical and nonclinical populations of war veterans over a 20-year period.

Various formulations and conceptual models aim to shed light on the longitudinal course of PTSD and the relationship between its symptom clusters. One of the first and most influential conceptualizations was that of Horowitz.[1] He argued that, in the immediate aftermath of the trauma, casualties feel flooded by powerful emotions. To cope, they employ various psychological defense mechanisms, until a certain degree of emotional balance is achieved. To restore balance, avoidance and intrusion appear in a cyclic fashion. During the "working through" stage, intrusive symptoms initially dominate, but, as time passes, avoidant symptoms become more significant, thereby allowing better processing of the experience. The Creamer and colleagues[2] model postulates that intrusive reexperiencing emerges as a direct response to the traumatic threat, and avoidance is the coping mechanism that is mobilized in response. Keane and colleagues[3] offer a similar explanation using terms from learning theory. In their 2-factor learning theory model of PTSD, trauma survivors initially learn to fear trauma-related stimuli by various conditioning processes, and subsequently learn to avoid both exposure and thoughts of previously conditioned stimuli.

Despite these elaborate theoretical formulations, there is only limited empirical evidence documenting the dynamic relationships among PTSD symptom clusters. The psychiatric establishment mostly conceives of this disorder as a monolithic entity consisting of several symptom clusters. However, it is unclear whether changes in the different clusters occur in the same manner and direction over time or if different clusters follow differential routes.

In an impressive study, Schell and colleagues[4] prospectively assessed adult survivors of community violence at 24 hours, 3 months, and 12 months posttrauma. They found that, whereas absolute levels of all PTSD clusters declined over time, reexperiencing symptoms

FOR CLINICAL USE

- In the early stages following trauma, clinicians are encouraged to use interventions targeting survivors' hyperarousal symptoms. Treating initial hyperarousal symptoms may help alleviate future symptoms of intrusion and avoidance as well.
- Clinicians are encouraged to assess acute stress reactions among their patients. An acute reaction to trauma may be a powerful marker for heightened levels of intrusion, avoidance, and hyperarousal symptoms, both in the present and in the future. Also, the long-term course of posttraumatic stress disorder symptom clusters may be different for survivors with and without an acute stress reaction.

remitted more rapidly than other symptom clusters. More importantly, hyperarousal emerged as the best single predictor of subsequent symptom severity. Other studies have investigated the specific relationships between avoidance and intrusion symptoms. For example, a study of burn victims revealed that avoidant behavior played an important role in the maintenance, but not in the exacerbation, of intrusive thoughts.[5] Some of the evidence on the interrelations between symptom clusters comes from psychotherapy studies. For example, a study of earthquake casualties[6] showed that an initial improvement in avoidance symptoms, as a result of therapy, was followed by an improvement in other symptom clusters as well. Finally, a review by McFarlane[7] suggested that different courses of PTSD occur depending on the initial severity of the traumatic event. McFarlane argued that, in severe trauma, all 3 symptom clusters remain stable over time, whereas, in less severe trauma, a reduction in both intrusion and avoidance, but stability in hyperarousal, is expected.

The interrelations among PTSD symptom clusters were also assessed prospectively among military populations. For example, in a study of Operation Desert Storm veterans,[8] posttraumatic symptom severity increased from 1 month to 6 months after homecoming and hyperarousal symptoms were initially more severe than reexperiencing and avoidance symptoms. In another prospective study assessing Gulf War veterans 1 and 2 years after the war,[9] an increase was observed in hyperarousal and in psychic numbing, whereas intrusion and avoidance remained stable. Finally, in a retrospective study of Vietnam War veterans, hyperarousal symptoms were reported as being the first to develop after exposure to combat stress, followed by avoidance and, finally, intrusion.[10]

As can be seen, these studies present mixed findings. This may be partially related to methodological differences. First, many studies[4,10] did not differentiate between trauma casualties who have already been identified or diagnosed with some kind of posttraumatic distress (i.e., clinical populations) and those who were exposed to traumatic events but were not necessarily identified as such (nonclinical populations). In addition, some studies relied on retrospective designs, which are subjected to various risks of reporting bias.[10] Other studies were based on a prospective design, but relied on relatively short follow-up periods that only allowed for a limited evaluation of symptom interrelations over time.[8]

The present study attempts to overcome some of these methodological shortcomings. It includes a long-term prospective follow-up of 20 years, which allows for a more comprehensive evaluation of the trajectories of PTSD symptom clusters over time. Finally, the study also compares a clinical and nonclinical population of combat veterans.

The study aims to examine (1) the stability/changes of intrusion, avoidance, and hyperarousal symptom clusters over time and (2) the interrelationship among intrusion, avoidance, and hyperarousal over time and to assess (3) these patterns in both clinical and nonclinical populations.

METHOD

Participants and Procedure

Two groups of male subjects participated in this study. The clinical group comprised 369 Israeli veterans who fought in the 1982 Lebanon War and had been identified by military mental health personnel as having acute combat stress reaction (CSR). Combat stress reaction consists of various polymorphic and labile psychiatric and somatic symptoms and is diagnosed based on impaired functioning by trained clinicians. Among the symptoms that may characterize this condition are paralyzing fear of death, emotional and physical numbness, withdrawal, severe depression, and impaired combat functioning. Criteria for inclusion in this group were (1) participation in frontline battles during the war, (2) a referral for psychiatric intervention made by the soldier's battalion surgeon during the war, (3) a diagnosis of combat stress reaction made on the battlefield by clinicians trained and experienced in the diagnosis of combat-related reactions, and (4) no indication in the clinician's report of serious physical injury and other psychiatric disorders. The nonclinical group comprised 306 soldiers who had participated in the same combat units as the CSR group, but were not identified as

Table 1. Correlations and Mean (SD) Number of Symptoms for Intrusion, Avoidance, and Hyperarousal Measures in Combat Stress Reaction and Non–Combat Stress Reaction Groups[a,b]

	Combat Stress Reaction Group									Non–Combat Stress Reaction Group								
Symptom Cluster	IN83	AVO83	AR83	IN84	AVO84	AR84	IN02	AVO02	AR02	IN83	AVO83	AR83	IN84	AVO84	AR84	IN02	AVO02	AR02
IN83	1.00									1.00								
AVO83	0.62	1.00								0.56	1.00							
AR83	0.64	0.63	1.00							0.53	0.65	1.00						
IN84	0.60	0.39	0.50	1.00						0.50	0.33	0.42	1.00					
AVO84	0.53	0.60	0.54	0.56	1.00					0.37	0.51	0.46	0.57	1.00				
AR84	0.48	0.44	0.63	0.64	0.65	1.00				0.49	0.44	0.59	0.56	0.67	1.00			
IN02	0.31	0.28	0.31	0.33	0.33	0.38	1.00			0.17	0.24	0.24	0.42	0.37	0.30	1.00		
AVO02	0.30	0.35	0.35	0.30	0.38	0.39	0.65	1.00		0.15	0.31	0.30	0.22	0.32	0.26	0.47	1.00	
AR02	0.34	0.36	0.40	0.31	0.38	0.45	0.73	0.77	1.00	0.18	0.28	0.35	0.30	0.33	0.34	0.72	0.73	1.00
Mean	1.83	1.61	2.76	1.71	1.61	2.71	1.50	1.34	2.87	0.69	0.55	1.16	0.58	0.62	1.17	0.84	0.59	1.41
SD	1.13	1.18	1.56	1.18	1.14	1.57	1.24	1.23	1.86	0.90	0.86	1.36	0.88	0.89	1.32	1.05	0.91	1.73

[a]All bivariate correlations are significant at $p < .05$.
[b]The numbers 83, 84, and 02 indicate the years in which the study groups were examined.
Abbreviations: AR = hyperarousal, AVO = avoidance, IN = intrusion.

having CSR. The 2 groups were matched in age, education, military rank, and assignment. Participants were assessed at 3 points in time after the war had ended: 1 year postwar (time 1), 2 years postwar (time 2), and 20 years postwar (time 3). The data presented here are based on the responses of veterans who participated in all 3 assessments. Written informed consent was obtained from participants. In addition, the study received approval from both the Israel Defense Forces and Tel Aviv University review boards.

Measure

The Posttraumatic Stress Disorder Inventory[11] is a self-report measure using DSM criteria for PTSD. Items were divided into 3 categories, according to DSM symptom clusters: (1) reexperiencing of the trauma (intrusion), (2) numbing of responsiveness to or reduced involvement with the external world (avoidance), and (3) additional symptoms, including hyperalertness, sleep disturbance, and concentration difficulties (hyperarousal). Participants were asked to indicate the frequency with which they experienced the described symptom within the past month on a 4-point scale ("never" = 1, "rarely" = 2, "often" = 3, "very often" = 4).

The scale was found to have good psychometric properties. In a recent study based on the same sample,[12] Cronbach αs for each of the 3 symptom clusters ranged between 0.90 and 0.93. Concurrent validity of the scale was also high[13] when it was compared both with widely accepted self-report measures, such as the Impact of Event Scale,[14] and with clinical diagnoses of PTSD.

Data Analyses

We aimed to assess the interrelations among intrusion, avoidance, and hyperarousal clusters across 3 waves of measurements via autoregressive cross-lagged (ARCL) modeling strategy. This strategy incorporates 2 main components.[15] First, later measurements of a construct are predicted by earlier measurements of the same construct, thus giving rise to the "autoregressive" term. Higher positive values of the regression parameter are usually interpreted as indicating greater stability of the construct over time.

In addition, later measures of one construct are regressed on earlier measures of other constructs. For example, in the current study we tested whether avoidance and/or intrusion in 1983 predicted hyperarousal in 1984. This is sometimes referred to as a residual change model, given that earlier measure of avoidance and intrusion predict later measures of hyperarousal above and beyond the effects of earlier hyperarousal. This model can be extended to examine bidirectional relations (referred to as a cross-lagged influence) such that earlier measures of hyperarousal predict later measures of avoidance as well.

Moreover, in order to examine whether the clinical (CSRs) and nonclinical groups differ in their PTSD cluster symptom trajectories over time, we used latent growth modeling (LGM; see Bollen and Curran[16] for an extensive review). The LGM is based on the premise that a set of repeated measures are functionally related to the passage of time. First, we estimated separately for the clinical and nonclinical groups, whether or not the trajectory of change in PTSD cluster symptoms was constant over time (i.e., linear). Then, we assessed whether the clinical and nonclinical groups differed in (1) their severity of intrusion, avoidance, and hyperarousal symptoms over time and (2) their trajectory of change in intrusion, avoidance, and hyperarousal symptoms over time (i.e., the trajectories' slopes).

We used EQS 6.1 Structural Equation Modeling (SEM) software.[17] Model fit was estimated with the comparative fit index (CFI) and the root-mean-square error of approximation (RMSEA). A model is judged as reasonably fitting the data when CFI and 1-RMSEA are larger

Figure 1. Autoregressive Cross-Lagged (ARCL) Model Assessing Longitudinal Stability and Cross-Lagged Effects of Combat Stress Reaction's Intrusion, Avoidance, or Hyperarousal Clusters

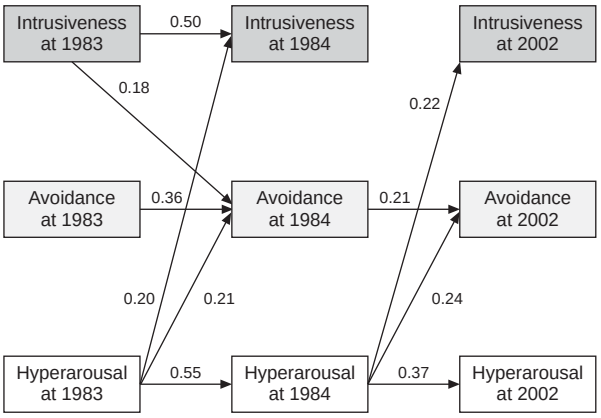
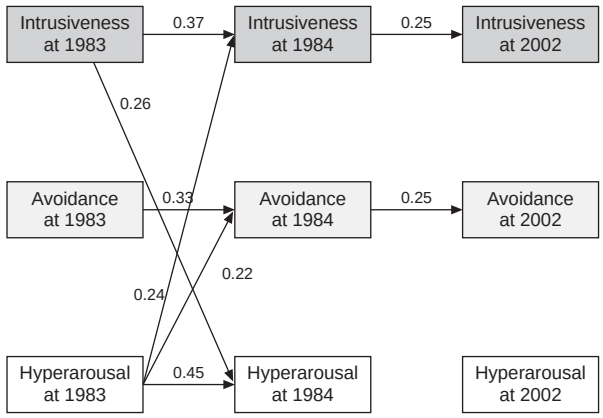


Figure 2. Autoregressive Cross-Lagged (ACRL) Model Assessing Longitudinal Stability and Cross-Lagged Effects of Non–Combat Stress Reaction's Intrusion, Avoidance, or Hyperarousal Clusters



than 0.90.[16] Missing data was handled with the case-wise maximum likelihood estimation for possible nonnormality when running EQS models.

RESULTS

Descriptive Statistics

First, we calculated descriptive statistics separately for the CSR and non-CSR groups. Mean and standard deviation values for intrusion, avoidance, and hyperarousal clusters are presented in Table 1. In addition, the relations between intrusion, avoidance, and hyperarousal clusters were examined with Pearson correlations between the scales at each point of time (see Table 1).

As can be seen in Table 1, there were reasonably high associations between intrusion, avoidance, and hyperarousal clusters, both cross-sectional and over time. Using Cohen and Cohen's[18] procedure, we found that associations between the intrusion, avoidance, and hyperarousal clusters did not differ significantly between the CSR and non-CSR groups (Fisher $Z < 1.6$, $p > .11$).

Autoregressive Cross-Lagged Modeling

In this section, we examined the stability and cross-lagged influence of intrusion, avoidance, and hyperarousal clusters separately for the CSR and non-CSR groups.

Figure 1 presents the stability and cross-lagged effect of CSRs' intrusion, avoidance, and hyperarousal clusters. The model showed excellent fit to the data ($\chi^2 = 6.84$, $df = 9$, $p = .65$, CFI = 1, 1-RMSEA = 1). The analyses revealed that the stability of avoidance and hyperarousal clusters was noticeably high: participants with above-average avoidance and hyperarousal in 1983 tended to report above-average avoidance and hyperarousal in both 1984 and 2002. In contrast, the stability of intrusion was low: there were no significant autoregressive effects.

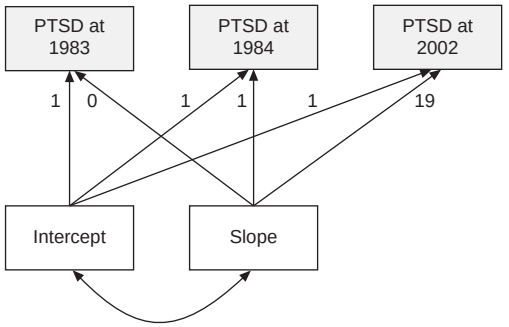
More importantly, the analyses revealed that hyperarousal predicted avoidance and intrusion above and beyond the autoregressive effects, but not vice versa. In other words, the CSR group's hyperarousal scores were related to higher levels of avoidance and intrusion in subsequent waves of measurement. However, higher avoidance and intrusion did not influence the level of hyperarousal in subsequent waves of measurement.

Lastly, the analysis revealed that the CSR group's intrusion in 1983 was related to higher levels of avoidance in 1984, but not vice versa. However, intrusion did not influence the level of avoidance in the subsequent wave of measurement at 2002.

Regarding the non-CSR group, the model showed excellent fit to the data ($\chi^2 = 9$, $df = 9$, $p = .43$, CFI = 1, 1-RMSEA = 1). Figure 2 presents the stability and cross-lagged influence of the non-CSR group's intrusion, avoidance, and hyperarousal clusters. The analyses revealed that the stability of intrusion and avoidance clusters was noticeably high: participants with above-average intrusion and avoidance in 1983 tended to report above-average intrusion and avoidance in both 1984 and 2002. In contrast, the stability of hyperarousal was low: there were no significant autoregressive effects.

More importantly, the analyses revealed that hyperarousal in 1983 predicted avoidance and intrusion in 1984 above and beyond the autoregressive effects. In other words, the non-CSR group's initial hyperarousal scores were related to higher levels of avoidance and intrusion in the subsequent wave of measurement. In addition, intrusion in 1983 predicted hyperarousal in 1984 above and beyond the autoregressive effects. In other words, the non-CSR group's intrusion scores were related to higher

Figure 3. Latent Growth Modeling for Estimating Posttraumatic Stress Disorder (PTSD) Developmental Trajectories



levels of hyperarousal in the subsequent wave of measurement. In contrast, higher avoidance did not influence the level of hyperarousal or intrusion in subsequent waves of measurement. However, unlike those with CSR, this effect is short lived: the non-CSR group's intrusion, avoidance, or hyperarousal clusters in 1984 did not show any cross-lagged influences in the subsequent wave of measurement in 2002.

Trajectories of PTSD Clusters in CSR and Control Groups

In this section, we examined (1) whether or not the trajectory of change in PTSD cluster symptoms was constant over time (i.e., linear) and (2) whether CSR and control groups' PTSD clusters behave differently over time.

To examine the change in PTSD intrusion, avoidance, and hyperarousal clusters, we estimated an LGM for the repeated measures of PTSD clusters reported in 1983, 1984, and 2002. Two latent factors were estimated: one to define the intercept of the developmental trajectory of PTSD clusters (with all factor loadings fixed to 1.0) and one to define the linear shape of the trajectory (with factor loading of 0, 1, and 19 in order to capture the passage of time). This model is presented in Figure 3. A mean was estimated for the intercept and linear shape factors, and these values represented the mean model-implied developmental trajectory pooled over all individuals.

The LGM for the CSR group presented in Figure 3 was estimated, separately for PTSD intrusion, avoidance, and hyperarousal clusters. The LGMs were found to fit the observed data well for intrusion ($\chi^2 = 4.33$, df = 4, p = .36, CFI = 1, 1-RMSEA = 1), avoidance ($\chi^2 = 0.07$, df = 4, p = .99, CFI = 1, 1-RMSEA = 1), and hyperarousal ($\chi^2 = 0.93$, df = 4, p = .92, CFI = 1, 1-RMSEA = 1). The models revealed significant intercepts of 1.84, 1.61, and 2.76 for PTSD intrusion, avoidance, and hyperarousal clusters, respectively. These intercepts indicate the mean level of PTSD cluster symptoms at the first time period ($t > 29.32$, $p < .001$). In addition, the analyses revealed that, whereas there was a significant linear decrease in PTSD intrusion and avoidance symptoms over time ($M = –0.02$, $p < .001$ for intrusion and $M = –0.01$, $p < .001$ for avoidance), there was no significant change in PTSD hyperarousal symptoms over time ($M = 0.008$, $p = .1$).

Regarding the control group, the separate LGMs for PTSD intrusion, avoidance, and hyperarousal clusters were estimated and were found to fit the observed data well for intrusion ($\chi^2 = 6.78$, df = 4, p = .15, CFI = 1, 1-RMSEA = 1), avoidance ($\chi^2 = 0.93$, df = 4, p = .92, CFI = 1, 1-RMSEA = 1), and hyperarousal ($\chi^2 = 0.52$, df = 4, p = .97, CFI = 1, 1-RMSEA = 1). The models revealed significant intercepts of 0.69, 0.55, and 1.16, for PTSD intrusion, avoidance, and hyperarousal clusters, respectively. These intercepts indicate the mean level of PTSD cluster symptoms at the first time period ($t > 13.02$, $p < .001$). In addition, the analyses revealed that, whereas there was a significant linear increase in PTSD intrusion and hyperarousal symptoms over time ($M = 0.01$, $p < .01$ for intrusion and $M = 0.01$, $p < .05$ for hyperarousal), there was no significant change in PTSD avoidance symptoms over time ($M = 0.001$, $p = .85$).

Following the separate LGMs for the CSR and control groups, we conducted multiple-group LGMs in order to examine whether the developmental trajectories were the same for CSR and control groups. To this end, we compared the default model that allowed different effects within the groups, with a series of constrained models that presupposed equality of the latent factors (i.e., intercept and shape).

The results indicated significant differences between the default models and the intercept factor constrained models for intrusion ($\Delta\chi^2 = 153.29$, df = 1, $p < .001$), avoidance ($\Delta\chi^2 = 213.13$, df = 1, $p < .001$), and hyperarousal ($\Delta\chi^2 = 183.33$, df = 1, $p < .001$). In other words, the findings indicated that the mean level of the CSR group's PTSD intrusion, avoidance, and hyperarousal symptoms at the first time period was significantly higher than the control group's mean level of PTSD cluster symptoms at the first time period.

In addition, whereas the results indicated significant differences between the CSR and control groups in the developmental trajectories of PTSD intrusion ($\Delta\chi^2 = 12.25$, df = 1, $p < .001$) and avoidance ($\Delta\chi^2 = 6.96$, df = 1, $p < .01$) clusters over time, no significant difference was found in the developmental trajectory of PTSD hyperarousal ($\Delta\chi^2 = 0.34$, df = 1, $p < .56$). In other words, the findings indicated that, whereas the CSR group's PTSD intrusion symptoms faintly decreased over time, the control group's PTSD intrusion symptoms tended to slightly increase over time. Moreover, whereas the CSR group's PTSD avoidance symptoms faintly decreased over time, the control group's PTSD avoidance symptoms

tended to remain at the same level over time. In contrast, the analyses indicated that although the control group's PTSD hyperarousal symptoms tended to somewhat increase over time, this trajectory of change was not significantly different from the CSR group's trajectory of change in PTSD hyperarousal symptoms.

DISCUSSION

Our findings indicated that, in both the clinical and nonclinical groups, the clusters of intrusion, avoidance, and hyperarousal were interrelated at any given point in time and across 20 years. This finding is in line with previous studies[5] and provides further validation for the well-established PTSD diagnosis. It seems that the clusters of intrusion, avoidance, and hyperarousal are in fact facets of the same clinical entity, and, therefore, share a similar, but by no means identical, longitudinal course.

While the clinical and nonclinical groups examined here did not differ in the intercluster associations, the former endorsed more severe symptoms, both cross-sectionally and across time. This finding is in line with previous studies showing that both civilian trauma casualties[19] and traumatized veterans who had an acute stress reaction[20] are at increased risk for subsequent psychopathology, particularly PTSD. Finally, this finding reveals the importance of differentiating between clinical and nonclinical populations following trauma, as they differ in length and intensity of all 3 PTSD symptom clusters.

The clinical and nonclinical groups differed in the long-term trajectories of intrusion and avoidance but not hyperarousal. More specifically, a small linear decline in intrusion was found in the clinical group, as opposed to a small linear increase in the nonclinical group. A small linear decline in intrusion was also found in the clinical group. However, no change in intrusion was found in the nonclinical group. Thus, it seems that while the initial level of symptoms was higher in the clinical group, these levels gradually decreased over time. This finding may be attributed to a ceiling effect, wherein initial high symptom levels are expected to decline in the future. Also, the decline in symptom levels may be the result of both natural recovery with time and therapeutic interventions administered to those who were identified by the military as psychiatric casualties. Finally, it may be interesting to view these results in light of findings from previous studies based on the same sample. Solomon and Mikulincer[21] have found that PTSD rates in the clinical group gradually declined between the 3 points of assessment. Thus, the general trajectory of PTSD was similar to the specific trajectory of both the intrusion and avoidance clusters in the clinical group. In the nonclinical group, Solomon and Mikulincer[21] found a more fluctuating course, with an initial increase in PTSD rates followed by a decrease 20 years after the war. This pattern is only partially similar to the course of the specific symptom clusters in this group.

As part of this study, we have also assessed the relative stability of each symptom cluster over time. Avoidance was found to be a particularly stable symptom cluster over time. This was true for both the clinical and nonclinical groups. This finding is in line with studies showing that avoidance plays a particularly important role in the chronicity of PTSD over time among war veterans.[22] We also found that hyperarousal was relatively stable in the clinical group but not in the nonclinical group. It may be interesting to review these findings in light of McFarlane's[7] suggestion that different courses of PTSD may occur depending on the initial severity of the traumatic event. While we did in fact find differences in the PTSD symptom course between the 2 groups, these differences were not those described by McFarlane. We may conclude that while trauma severity does contribute to the course of symptoms, the exact nature of this contribution may vary by population and type of trauma.

One of the main findings of this study has to do with the prominent role of hyperarousal symptoms. In both study groups, initial hyperarousal symptoms predicted both avoidance and intrusion symptoms in later assessments. This indicates that hyperarousal symptoms may function as the psychological "engine" of PTSD by providing the platform on which other symptom clusters subsequently appear. Our findings are in line with previous studies, in both laboratory[23] and nonlaboratory[4] settings, which have shown these symptoms to predict both intrusion and avoidance. They may also be related to findings showing that hyperarousal was the first symptom cluster to emerge after the occurrence of the trauma.[10] These findings hint at the unique nature of this cluster, which has often been neglected by trauma researchers. One possible explanation for the central role of hyperarousal may be the biologic nature of this symptom cluster. Hyperarousal symptoms are related to various neurologic processes,[24] which may have subsequent effects on other symptom clusters as well.

The present study has several methodological shortcomings. First, it is correlative and therefore does not allow clear-cut inferences regarding causal relations. Second, since the study is based on only 1 measure (i.e., the PTSD Inventory), some variance in the results may be associated with measurement alone. Finally, our prediction of PTSD symptom course is based on time intervals that vary in length. In particular, it is impossible to know what changes in symptom courses occurred in the 18-year time interval between 1984 and 2002.

Despite these limitations, however, this study may have important theoretical and clinical implications. First, the core question of this study was whether or not PTSD is a monolithic disorder or, rather, one comprising various

symptom clusters that vary in their course and stability. Our findings seem to suggest the latter, as PTSD clusters were found to differ in several important aspects. This finding shows the need for trauma researchers to put more emphasis on the study of specific symptom clusters, instead of treating PTSD as a unified, undifferentiated disorder. Our study has also shown the need to differentiate more clearly between clinical and nonclinical populations of trauma casualties, as they differ in various aspects of the PTSD symptom course. Finally, our finding regarding the central role of arousal symptoms also has important implications. First and foremost, it may help practitioners identify trauma survivors who face an increased risk of developing future posttraumatic symptoms. An important clinical implication of our findings may be the development of therapeutic interventions aimed at calming the casualty's initial arousal, in hope that other symptom clusters will also be attenuated as a result.[4] These findings are highly logical in light of the fact that PTSD is, in its core, an anxiety disorder and, as such, is based on hyperarousal mechanisms of both the body and the psyche. Unfortunately, most theoretical formulations of PTSD, including those reviewed here,[1,3] have focused on symptoms of intrusion and avoidance, while neglecting the potential role of hyperarousal. Future studies are encouraged to attempt to explore the structure of the arousal cluster further in order to gain a broader understanding of its constituents.

Disclosure of off-label usage: The authors have determined that, to the best of their knowledge, no investigational information about pharmaceutical agents that is outside US Food and Drug Administration–approved labeling has been presented in this article.

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