Aversive Imagery in Posttraumatic Stress Disorder: Trauma Recurrence, Comorbidity, and Physiological Reactivity

Lisa M. McTeague, Peter J. Lang, Marie-Claude Laplante, Bruce N. Cuthbert, Joshua R. Shumen, and Margaret M. Bradley

Background: Posttraumatic stress disorder (PTSD) is characterized as a disorder of exaggerated defensive physiological arousal. The novel aim of the present research was to investigate within PTSD a potential dose-response relationship between past trauma recurrence and current comorbidity and intensity of physiological reactions to imagery of trauma and other aversive scenarios.

Methods: A community sample of principal PTSD (n = 49; 22 single-trauma exposed, 27 multiple-trauma exposed) and control (n = 76; 46 never-trauma exposed, 30 trauma exposed) participants imagined threatening and neutral events while acoustic startle probes were presented and the eye-blink response (orbicularis occuli) was recorded. Changes in heart rate, skin conductance level, and facial expressivity were also indexed.

Results: Overall, PTSD patients exceeded control participants in startle reflex, autonomic responding, and facial expressivity during idiographic trauma imagery and, though less pronounced, showed heightened reactivity to standard anger, panic, and physical danger imagery. Concerning subgroups, control participants with and without trauma exposure showed isomorphic patterns. Within PTSD, only the single-trauma patients evinced robust startle and autonomic responses, exceeding both control participants and multiple-trauma PTSD. Despite greater reported arousal, the multiple-trauma relative to single-trauma PTSD group showed blunted defensive reactivity associated with more chronic and severe PTSD, greater mood and anxiety disorder comorbidity, and more pervasive dimensional dysphoria (e.g., depression, trait anxiety).

Conclusions: Whereas PTSD patients generally show marked physiological arousal during aversive imagery, concordant with self-reported distress, the most symptomatic patients with histories of severe, cumulative traumatization show discordant physiological hyporeactivity, perhaps attributable to sustained high stress and an egregious, persistent negative affectivity that ultimately compromises defensive responding.

T his research explores a potential dose-response relationship between trauma recurrence and intensity of physiological reactions to trauma memories in principal posttraumatic stress disorder (PTSD). Epidemiological work has revealed that exposure to multiple compared with single traumatic events more strongly predisposes the development of PTSD (1). Cumulative exposure is associated with more severe (2–4) and chronic posttraumatic stress (5,6), more generalized symptoms (e.g., nonspecific anxiety, anger) (7–9), increased morbidity rates (e.g., depression, panic, substance abuse) (9,10), and poorer socio-occupational functioning (2,6,11). In effect, multiple compared with single traumatic exposure more perniciously sensitizes individuals to subsequent stress (1), prolonging pathological emotional processing across numerous symptom domains.

Script-driven emotional imagery is a valuable tool in studies of

From the Center for the Study of Emotion & Attention (LMM, PJL, M-CL, JRS, MMB), University of Florida, Gainesville, Florida; and Department of Psychology (BNC), University of Minnesota, Minneapolis, Minnesota.

Address correspondence to Peter J. Lang, Ph.D., Center for the Study of Emotion & Attention, University of Florida, PO Box 112766, Gainesville, FL 32611; E-mail: plang@phhp.ufl.edu.

PTSD, permitting presentation of idiographic trauma challenges. Findings that physiological arousal to fear imagery parallels anticipatory reactions to threatening events corroborates its ecological validity (12), similarly mobilizing the autonomic nervous system (e.g., heart rate, skin conductance), communicating threat through facial musculature (e.g., corrugator "frown" muscle), and prompting somatic reflexive action (e.g., startle potentiation) (13,14). Animals confronting survival threat show similar reactions, mediated by the brain's defense circuit (centered on the amygdala) (15,16) and neuroimaging studies suggest that a comparable circuit (17–19) underlies human fear.

Autonomic and somatic hyperarousal during trauma-related imagery are hallmark symptoms of PTSD (20) demonstrated in many trauma populations (combat [21-23], childhood sexual abuse [24], breast cancer [25], war zone nursing [26], heterogeneous civilian events [27]). Exaggerated reactivity is not consistent, however, across all physiological measures. Although heart rate and skin conductance responses are often both recorded, frequently only a single autonomic measure shows increases during trauma-related imagery (21,24,27–29). Equivalent reactivity between control and PTSD groups has been observed in autonomic or facial muscle measures (30) and diminished rather than heightened fear potentiation and heart rate responses in PTSD (31–34). Surveying a cumulative sample of 96 patients across multiple studies, Pitman et al. (25) estimated that 30% to 40% of PTSD participants are physiologically nonresponsive during trauma-related processing.

In a series of imagery investigations, Cuthbert et al. (34), Cook et al. (35), McNeil et al. (36), Weerts and Lang (37), and Lang et

al. (12,38,39) have explored evoked defensive arousal differences across the spectrum of anxiety diagnoses: specific and social phobia patients demonstrated the greatest autonomic and startle responses. Paradoxically, patients with more pervasive and diffuse anxiety symptomatology—panic disorder with agoraphobia, generalized anxiety disorder (GAD)—showed less robust fear potentiation (despite reports of intense fear). This reflex blunting was consistently more pronounced across and within respective diagnoses, coincident with increased clinician-rated severity, poorer prognosis, greater comorbidity (depression and anxiety), elevated questionnaire-based indexes of negative affectivity, and lengthier disorder chronicity (40,41), suggesting that defensive engagement during imagery might be compromised by long-term stress and accompanying dysphoria.

In the current study, it was expected that similar to many studies (21,23), PTSD patients as a whole would demonstrate heightened defense circuit activation relative to control participants when confronting trauma-related imagery (i.e., potentiating startle and enhancing skin conductance, heart rate, and facial muscle action [corrugator]). Furthermore, patients and control participants were expected to react similarly during neutral scenarios and threatening contexts for which defensive mobilization is normal and adaptive (e.g., facing an attacking animal). Standard anger and panic attack scenarios were also assessed in expectation that these symptom-relevant, but nontrauma-related, scenarios would prompt more reactivity in patients than control participants (30,42), as PTSD patients often report anger during aversive imagery (21,23,28) and anger (43) and panic attacks (44,45) are prominent posttraumatic symptoms.

Regarding trauma extent in PTSD, single-trauma PTSD patients were expected to show robust physiological responses during aversive imagery similar to phobic disorders (36–41). However, multiple-trauma PTSD patients—likely more severe with higher depression and anxiety comorbidity—would demonstrate blunted physiology as found in other anxiety spectrum

Table 1. Proportion of Control and PTSD Groups by Index Trauma

Trauma type	Trauma-Exposed Control (*n* = 30)	Single-Trauma PTSD (*n* = 22)	Multiple-Trauma PTSD (*n* = 27)
Childhood			
Accident/Injury	.10	0	0
Natural Disaster	.03	0	0
Physical &/Sexual Abuse	.03	0	.33
Sexual Assault[a]	.03	.09	.11
Witnessed Injury/Death	.17	0	0
Adulthood			
Accident/Injury	.13	.45	.11
Combat Exposure	.03	0	.07
Domestic Violence/Stalking	0	0	.07
Natural Disaster	.07	.05	0
Sexual &/ Physical Assault	.14	.23	.26
Witnessed Injury/Death	.27	.18	.04

PTSD, posttraumatic stress disorder.
[a]Includes childhood stranger and extra/intrafamilial single incident sexual assaults.

Table 2. Proportion of Multiple-Trauma PTSD Group Endorsing the Following Traumatic Events in Addition to Index Trauma

Trauma Type	Multiple-Trauma PTSD (*n* = 27)
Childhood	
Accident/Injury	.04
Natural Disaster	0
Physical &/ Sexual Abuse	.26
Sexual Assault[a]	.33
Witnessed Injury/Death	.18
Adulthood	
Accident/Injury	.11
Combat Exposure	0
Domestic Violence/Stalking	.22
Natural Disaster	.04
Sexual &/ Physical Assault	.15
Witnessed Injury/Death	.18

PTSD, posttraumatic stress disorder.
[a]Includes childhood stranger and extra/intrafamilial single incident sexual assaults.

disorders characterized by pervasive anxiety and prominent depression. Finally, control participants with a trauma history were not expected to differ in responsiveness from nonexposed control participants (46).

Methods and Materials

Participants

Participants (81% Caucasian) were assessed at the University of Florida Fear and Anxiety Disorders Clinic: 49 treatment-seeking adults with principal diagnoses of PTSD (66% female) and 76 healthy community control participants (71% female).

Diagnostic Classification

Diagnostic groups were established using the Anxiety Disorder Interview Schedule for DSM-IV (ADIS-IV) (47), a semistructured interview for assessing current anxiety, mood, substance use, and somatoform disorders and for screening psychosis and major medical disease.

For multiple Axis I disorders, diagnostic primacy was determined by clinician-rated severity (ranging from 0, No features present, to 5, Diagnosis present; severe) reflecting both distress and interference. control participants denied current or lifetime diagnoses of psychiatric illness.

For trauma subtype assignment, patients with a lifetime history of one criterion A event (reported during ADIS administration and meeting both A1 and A2 criteria) were classified single-trauma (*n* = 22), whereas those with two or more were classified multiple-trauma (*n* = 27; Table 1). Multiple-trauma patients reported a minimum of three different types of high magnitude traumas (e.g., interpersonal/assaultive violence). Types of exposure in addition to the index trauma are listed in Table 2. control participants were simply classified as exposed (*n* = 30) or nonexposed (*n* = 46) to at least one trauma. All trauma-positive participants endorsed direct exposure.

Procedure

The University of Florida Institutional Review Board (IRB-01) approved the study and participants provided informed consent before assessment. Participants completed questionnaires and interview in the morning; psychophysiological assessment and clinical debriefing followed in the afternoon.

Experimental Stimuli. Twenty-four narrative imagery texts were used (48). Analyses focused on two idiographic, "personal" threat narratives representing each patient's primary clinical fear[1] or for control participants their "worst fear" experiences (Table S1 in Supplement 1). Standard scenes included two anger (witnessing a dog intentionally harmed, having parking spot taken), two panic attack (in busy checkout line, while driving), four survival threat (physical attack by animal/human), and two neutral (watching documentary, reading magazine) events. Filler scripts were low arousal or engaging pleasant scenes to impede development of an overall unpleasant arousal context. Scripts were ~20 words designed to quickly reveal affect and reflect active participation. A woman recorded the scenes using minimal prosody for presentation over earphones (Telephonics TDH-49, Telephonics Corporation, Huntington, New York).

Imagery Assessment. Seated in a quiet, dimly lit room with electrodes placed, participants were instructed to listen to the auditory scripts with eyes closed, vividly imagining the events described, as if actively involved. Throughout the recording session, soft tones cued participants to relax, breathe slowly, and silently repeat the word "one" to stabilize between-trial physiological activity (49). Imagery scripts were interspersed every 36 seconds in the tone series with content pseudorandomized so that no more than two stimuli of the same hedonic valence (pleasant, neutral, unpleasant) or content category (e.g., survival threat) were presented consecutively. The script series was repeated in a counterbalanced order.

Trials consisted of a 1-second baseline, the 6-second auditory script, and 12 seconds of imagery. Startle probes (50 msec 95 dB[A] white noise, instantaneous rise-time) were presented at 4 to 5.5 seconds or 10 to 11.5 seconds postscript onset, or both, and on 25% of intertrial intervals, at 22 to 23.5 seconds postimagery offset.

Following imagery assessment, participants rated each scene for experienced pleasure and emotional arousal (50).

Experimental Control and Data Collection

A PC-compatible computer running VPM software (51) controlled stimulus presentation and data acquisition. Bioamplifiers recorded electromyograph (EMG) potentials at left orbicularis occuli and corrugator supercilii, skin conductance level (SCL), and electrocardiogram (ECG) as reported (41).

Data Reduction and Analysis

Univariate analyses of variance (ANOVAs) and Tukey honestly significant difference tests for planned comparisons determined group differences in demographic and questionnaire data.

Using VPM software, EMG, SCL (normalized [log(SCL+1)]), and ECG R-R intervals (converted to beats-per-minute) were reduced into half-second bins. Responses were determined by subtracting amplitude during the 1 second before script presentation from averages during the 12-second imagery period.

Startle blinks from orbicularis oculi EMG represented the

magnitude difference between onset and peak muscle potential (52), standardized within subject in relation to the mean and standard deviation of intertrial probe responses (34).

Using SPSS (SPSS Inc., Chicago, Illinois), omnibus repeated measures ANOVAs were performed separately for each physiological measure, with diagnostic status (control subject, patient) as the between-subjects factor and imagery content as the within-subjects factor. Startle and autonomic reactivity during imagery have been shown to strongly covary with rated emotional arousal (34–36); thus, contents were entered according to the linear increase in arousal reported by the patients (i.e., neutral, anger, panic, survival threat, idiographic/personal threat). Significant overall group effects were followed up with between-group tests by contents to specify which imagery scenarios evoked different sensitivities in patients and control participants, facilitating comparisons with preceding imagery studies of PTSD that utilized different contents (21–28). Within-group comparisons explicated interactions. Analyses were repeated for exposure subtypes (i.e., no exposure/trauma-exposed control subject; single-trauma/multiple-trauma PTSD). Guided by prior investigations focused on idiographic threat-related imagery (20,22,27), group comparisons on that content were tested irrespective of omnibus results. Wilks' lambda addressed sphericity issues (53).

Results

PTSD Versus Control Groups

Affective Judgments. Both groups rated personal threat images most and neutral scenes least unpleasant, $F(4,116) = 145.26$, $p < .001$. Patients rated panic and personal threat scenes more unpleasant than control participants, $p\text{s} < .05$. Furthermore, control participants rated personal threat, anger, and survival threat scenes equivalently, all ns; patients rated personal threat as more aversive than all other contents, all $p\text{s} < .001$; content × diagnosis interaction, $F(4,116) = 3.77$, $p < .01$.

Emotional arousal also varied with content (Table 3), content $F(4,116) = 119.98$, $p < .001$; content × diagnosis $F(4,116) = 3.55$, $p < .01$. control participants rated personal threat scenes most arousing followed by survival threat, anger, panic, and neutral scenes. Patients showed the same extremes, but anger, panic, and survival threat did not differ. Additionally, patients endorsed higher arousal than control participants for panic, anger,[2] and neutral scenes.

Baseline Physiology. No group differences emerged for blink magnitude to intertrial startle probes or for SCL, corrugator, or orbicularis activity in the 1-second baseline before script onset (Table S2 in Supplement 1). Consistent with preceding studies (54), patients exceeded control participants in heart rate, $F(1,121) = 25.24$, $p < .001$.[3]

Startle Reflex Potentiation. Blink magnitude (Figure 1, Table 3) was larger during unpleasant compared with neutral imagery, content $F(4,104) = 7.85$, $p < .001$, all unpleasant-neutral comparisons, $p < .01$; patients were generally more reactive than control participants, diagnosis $F(1,107) = 5.14$, $p < .05$, content × diagnosis $F(4,104) = 1.00$, ns, exceeding control participants in reflexes elicited during all unpleasant contents except survival threat.

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[1]Personal scenes were based upon descriptions of prior experiences. For patients with PTSD, both personal scenes described fearful and threatening aspects of their index trauma. Among the control participants, 60.5% of participants described a traumatic or potentially traumatic event (e.g., physical assault, motor vehicle accident, witnessing violence, home invasion) for at least one of their two personal scenes, whereas others described intense nontraumatic, fearful events (e.g., giving a speech, receiving injections, undergoing surgery, panic attacks, exposure to snakes/insects).

[2]One-tailed test based on directional hypothesis that patients would exceed control participants.
[3]Analyses for heart rate change were calculated on residuals secondary to removing the trial-specific baseline (1-second average before script onset) effects via linear regression.

Table 3. Mean Responses and Standard Deviations to Imagery Scenes by Control and PTSD Groups

Response Modality/Imagery Scene	Control	PTSD	Group Effect
Pleasure (1-9)			
Neutral	6.93 (1.53)	6.41 (1.74)	$F(1,121) = 3.04$, ns
Anger	2.79 (1.22)[a]	2.59 (1.14)[a]	$F(1,121) = .90$, ns
Panic attack	4.00 (1.08)[a]	3.49 (1.49)[a]	$F(1,121) = 4.82, p < .05$
Survival threat	2.68 (.99)[a]	2.59 (1.36)[a]	$F(1,121) = .20$, ns
Personal/idiographic threat	2.65 (1.41)[a]	1.62 (1.11)[a]	$F(1,119) = 17.99, p < .001$
Arousal (1-9)			
Neutral	2.31 (1.54)	3.10 (1.78)	$F(1,121) = 6.81, p < .05$
Anger	6.15 (1.78)[a]	6.73 (1.71)[a]	$F(1,121) = 3.22, p < .05$[b]
Panic attack	5.37 (1.90)[a]	6.88 (1.44)[a]	$F(1,121) = 21.80, p < .001$
Survival threat	6.69 (1.53)[a]	6.99 (1.70)[a]	$F(1,121) = 1.00$, ns
Personal/idiographic threat	7.69 (1.67)[a]	8.12 (1.66)[a]	$F(1,119) = 1.93$, ns
Startle Reflex (*t* score)			
Neutral	49.76 (5.01)	52.16 (10.78)	$F(1,107) = 2.50$, ns
Anger	51.16 (6.08)	56.34 (15.56)[a]	$F(1,107) = 5.54, p < .05$
Panic attack	51.51 (7.20)[a]	55.30 (9.53)[a]	$F(1,107) = 5.53, p < .05$
Survival threat	54.57 (8.22)[a]	57.42 (13.80)[a]	$F(1,107) = 1.83$, ns
Personal/idiographic threat	53.58 (7.33)[a]	59.37 (24.86)[a]	$F(1,107) = 3.24, p < .05$[b]
SCL Δ (log [μS + 1])			
Neutral	−.006 (.036)	−.013 (.061)	$F(1,118) = 4.87, p < .05$
Anger	.0005 (.035)	.012 (.048)	$F(1,119) = 2.52$, ns
Panic attack	.0009 (.042)	.011 (.067)	$F(1,117) = .98$, ns
Survival threat	.004 (.022)[a]	.031 (.089)[a]	$F(1,118) = 6.30, p < .05$
Personal/idiographic threat	.045 (.086)[a]	.063 (.145)[a]	$F(1,118) = .79$, ns
Heart Rate Δ (bpm)			
Neutral	−.46 (2.03)	−.95 (2.04)	$F(1,120) = 1.68$, ns
Anger	−.27 (1.43)	−.68 (1.37)	$F(1,120) = 2.49$, ns
Panic attack	−.22 (1.50)	.20 (1.62)[a]	$F(1,121) = 2.40$, ns
Survival threat	.04 (1.00)[a]	−.29 (1.12)[a]	$F(1,121) = 3.02$, ns
Personal/idiographic threat	1.00 (1.00)[a]	2.02 (3.07)[a]	$F(1,120) = 4.56, p < .05$
Corrugator EMG Δ (μV)			
Neutral	−.07 (.79)	.18 (1.71)	$F(1,122) = 1.19$, ns
Anger	1.17 (2.77)[a]	.80 (1.67)[a]	$F(1,122) = .72$, ns
Panic attack	.49 (1.40)[a]	.60 (1.24)[a]	$F(1,122) = .19$, ns
Survival threat	.96 (1.98)[a]	1.34 (2.10)[a]	$F(1,122) = 1.05$, ns
Personal/idiographic threat	1.17 (2.60)[a]	2.66 (5.15)[a]	$F(1,122) = 4.52, p < .05$

Note: Pleasure rated on SAM (50): 1 = completely unhappy, 9 = completely happy; Arousal rated on SAM: 1 = completely relaxed, 9 = completely aroused.

Δ, change; bpm, residual beats per minute after removal of baseline effects; EMG, electromyographic; μS, microsiemen; μV, microvolt; PTSD, posttraumatic stress disorder; SAM, Self-Assessment Manikin; SCL, skin conductance level.

[a]Within-group comparison to neutral significant at $p < .05$.
[b]One-tailed test.

Autonomic and Facial Responses. control participants and patients showed similar patterns of sympathetic reactivity across contents, content $F(4,114) = 6.03, p < .001$, content × diagnosis $F(4,114) = .96$, ns, with increased skin conductance during survival and personal threat relative to neutral imagining, ps < .01. An overall difference was suggested, diagnosis $F(1,117) = 3.90, p = .05$, attributable to larger increases for patients during survival threat and unexpectedly, although consistent with arousal ratings, for neutral as well (Figure 1, Table 3).

Paralleling startle and SCL findings, heart rate change was modulated by imagery scene, content $F(4,116) = 10.46, p < .001$, with significant increases above neutral for panic, survival, and personal threat imagery, ps < .05. Contents varied similarly between groups, content × diagnosis $F(4,116) = 2.03$, ns, except that control participants showed a significant linear increase from neutral, with personal threat most ex-

treme, followed by survival threat; conversely, patients showed their second largest response to imagery of panic attacks—a response discordant from their arousal ratings, content × diagnosis (linear contrast), $F(1,116) = 4.23, p < .05$. As predicted, acceleration in patients surpassed control participants during personal threat imagery (Table 3).

Both patients and control participants demonstrated increased corrugator tension during unpleasant relative to neutral imagining (Figure 2), content $F(4,119) = 8.87, p < .001$. However, reactivity to specific contents differed within diagnosis, content × diagnosis $F(4,119) = 2.51, p < .05$: control participants augmented similarly to anger, survival, and personal threat imagery and more modestly, but reliably, for panic scenarios, comparisons with neutral ps < .01. In contrast, patients showed by far the most robust contraction to personal threat imagery, exceeding all other contents, ps < .05. Furthermore, the survival threat increase was secondary, surpassing reactivity to panic and
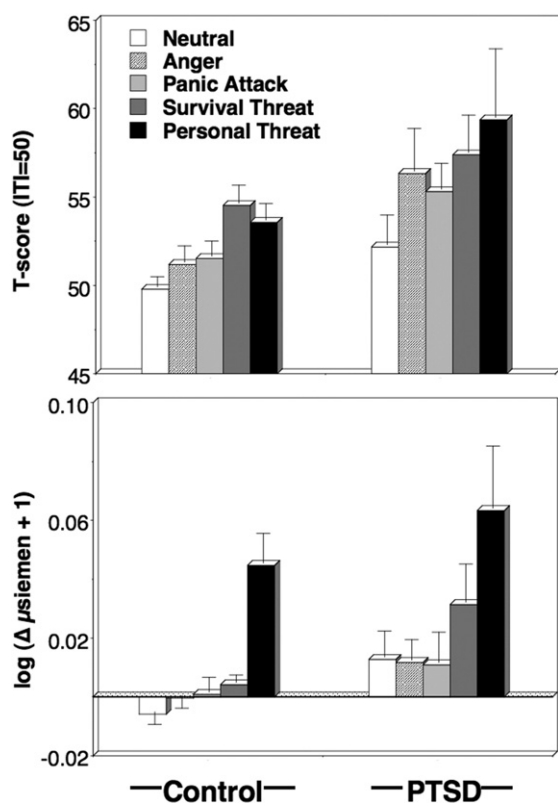
Figure 1. Mean startle reflex responses (standardized to the distribution of responses during intertrial intervals; top panel) and skin conductance level change (bottom panel) during neutral, anger, panic, survival threat, and personal threat imagery in control and PTSD groups. Error bars refer to standard error of the mean. PTSD, posttraumatic stress disorder.

anger and the minimal response to neutral scenarios. Notably, patients' corrugator response to personal threat reliably surmounted that for control participants (Table 3).[4]

Trauma Subtypes

Evaluative Ratings. Multiple-trauma patients rated imagery contents overall more aversive than both control groups, diagnosis $F(3,117) = 5.88$, $p < .01$, control subgroups versus multiple-trauma comparisons, $ps < .01$. Single-trauma patients were intermediate (subgroup comparisons ns). Specifically for personal threat, both PTSD subgroups exceeded control participants, content × diagnosis $F(12,301.91) = 1.79$, $p = .05$, control versus patient comparisons, $ps < .05$. Multiple-trauma patients also surpassed control participants in overall arousal, diagnosis $F(3,117) = 4.75$, $p < .01$, multiple-trauma versus trauma-exposed control participants, $p < .01$, versus nontrauma, $p < .05$, with single-trauma patients again intermediate.

Defensive Physiology. The exaggerated startle potentiation characteristic of the overall PTSD group was clearly driven by the strong responding of single-trauma patients,[5] group $F(3,105) =$

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[4]In follow-up analyses of baseline and imagery physiology, gender exerted neither a main effect nor interaction in any measure.
[5]No reliable differences emerged between patient and control subtypes in blink magnitude to intertrial startle probes or for SCL, corrugator, or orbicularis EMG activity in the 1-second baseline before script onset. Single-trauma (M = 74.94, SD = 13.03) and multiple-trauma (M = 75.90, SD = 12.65) patients showed similarly rapid baseline heart rate, both demonstrating higher rates than control participants (nonex-
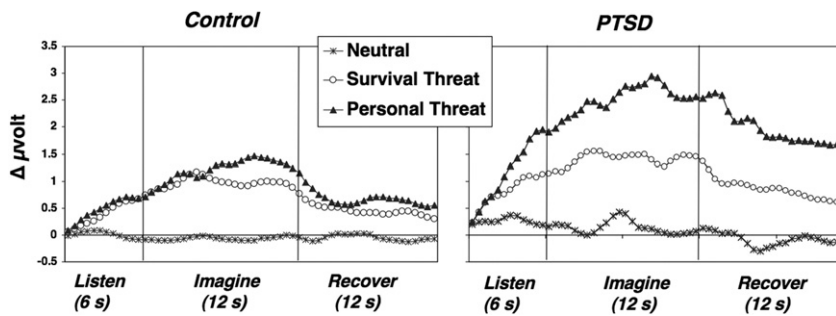
5.33, $p < .01$, exceeding both the multiple-trauma and control groups, $ps < .01$ (Figure 3). Between-group content tests were significant except neutral and survival threat. Single-trauma patients showed augmented responding relative to control and multiple-trauma groups during anger and personal threat, $ps < .01$, and compared with control participants during panic imagery, $p < .05$. For the multiple-trauma group, responses to personal threat did not differ from neutral imagery and were also reliably less than for both panic and survival threat imagery, post hoc: $ps < .05$. Startle response differences between neutral and unpleasant imagery were also analyzed for the single-trauma patients. These results further underscored the defensive hyper-responsivity of the single-trauma group: fear potentiation was greater for the single-trauma than control and multiple-trauma groups for both anger, diagnosis $F(3,105) = 3.15$, $p < .05$, and personal threat imagery, diagnosis $F(3,105) = 4.31$, $p < .01$, all between-groups, $ps < .05$.

A strikingly similar pattern emerged in skin conductance (Figure 3): single-trauma patients showed heightened sympathetic activation relative to the multiple-trauma and both control groups, $ps < .05$: content $F(4,112) = 6.33$, $p < .001$, diagnosis $F(3,115) = 4.42$, $p < .01$, content × diagnosis $F(12,296.62) = .25$, ns. For survival threat, the single-trauma group evinced larger conductance increases than the multiple-trauma and both control groups, $ps < .01$, diagnosis $F(3,116) = 7.13$, $p < .001$, and during personal threat imagery reliably exceeded the multiple-trauma group, $p < .05$, with the same tendency relative to control participants, $ps = .05$,[2] diagnosis $F(3,116) = 2.90$, $p < .05$. This same pattern of increased responding in the single-trauma group was weakly evident during neutral imagery, $p = .06$, no such trend was found for multiple-trauma patients.

Heart rate changes during imagery were generally similar over contents in the four subgroups. However, planned comparisons for personal threat imagery revealed greater acceleration in the single-trauma group (M = 2.66, SD = 3.75) than for both nonexposed (M = 1.05, SD = 2.28) and trauma-exposed (M = .93, SD = 2.04) control participants, $ps < .05$.[2]

In contrast to startle and autonomic indexes, marked corrugator reactions during survival and personal threat imagery were equivalent for both single- and multiple-trauma groups, significantly greater than for control participants.[6]

Trauma Characteristics. Relative to the single-trauma group, the multiple-trauma group indicated significantly more prevalent intentional trauma (single: 40.9%, multiple index: 92.6%, multiple

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posed, M = 65.14, SD = 10.81; trauma-exposed, M = 65.05, SD = 9.06), group, $F(3,119) = 8.31$, $p < .001$.
[6]Twenty-seven of the 49 patients indicated current use of psychotropic medication. Most frequently, these were selective serotonin reuptake inhibitors (34.7%) and/or benzodiazepines (25.5%). The effects of these and less frequently endorsed compounds (e.g., norepinephrine and dopamine reuptake inhibitors, 8.2%; serotonin norepinephrine reuptake inhibitors, 6.1%) were assessed by comparing resting and imagery reactivity among the medicated and nonmedicated patients both for patients as a whole and within subtypes. Considering either general psychotropic usage or more specific classes of drugs, no reliable effects emerged, perhaps due to the relatively small proportion of the sample on any single medication. These null medication findings are consistent with prior physiological investigations in PTSD (23,33,55,56). Reported usage of prescription and over-the-counter physical health medications for promoting physical health, as well as recreational substance use were also collected but low frequencies of endorsement precluded statistical analysis.

Figure 2. Corrugator electromyography change in half-second averages during neutral, survival, and personal threat script perception; imagery; and recovery for control (left panel) and PTSD (right panel) groups. Throughout all epochs, PTSD patients were reliably more reactive than control subjects to personal threat. PTSD, posttraumatic stress disorder.

additional: 74.1%), childhood trauma (single: 9.1%, multiple index: 44.4%, multiple additional: 51.9%), and specifically, childhood sexual and/or physical abuse (single: 0%, multiple index: 33.3%, multiple additional: 26%). Rate of index trauma occurring to self (vs. witnessing) was similarly high in both patient groups (single: 81.8%, multiple: 96.3%).[7] Overall, these characteristics suggest not only cumulative but also more severe trauma exposure in the multiple-trauma group. In post hoc analyses to explore the attenuated reactivity of the multiple-trauma group, these variables exerted neither a main effect nor interaction on any measure of defensive physiology.

Trauma Duration and PTSD Chronicity. Traumatic events persisted over a lengthier period for multiple-trauma patients (M = 18.14 years, SD = 11.87), with the initial event occurring at approximately 11 years (SD = 9.24) and the most recent event at 29 years (SD = 10.0); traumatic exposure in single-trauma PTSD occurred at approximately 32 years of age (SD = 13.35). Posttraumatic stress disorder onset for single-trauma patients was approximately 9 years later than for multiple-trauma patients (Table 4). Because age at evaluation did not differ, PTSD chronicity was significantly longer in the multiple-trauma than single-trauma patients (Figure 4).

Symptomatology. A highly consistent pattern of elevated distress and functional impairment was observed in the multiple-trauma compared with single-trauma patients but with the single-trauma group still far exceeding both control groups (Table 4). Specifically, questionnaire measures of anxiety sensitivity, non-specific trait anxiety, and depression were lowest for the two control groups, increased in severity for the single-trauma group, and were highest for the multiple-trauma patients (Figure 4).[8] The multiple-trauma patients consistently surpassed the single-trauma patients in frequency of comorbid anxiety and depression, total number of Axis I disorders, and clinician-conferred ratings of PTSD severity and poorer treatment prognosis. In analysis of PTSD symptom clusters as delineated by Suvak *et al.* (57), single-trauma and multiple-trauma groups indicated commensurate severity of re-experiencing, while the multiple-trauma exceeded the single-trauma group in extent of emotional numbing and hyperarousal and showed the same trend for strategic avoidance. Ability to generate vivid mental imagery was equivalent across subgroups.

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[7] The prevalence rates for trauma-exposed control participants were intentional trauma (46.7%), childhood trauma (36.7%), childhood sexual and/or physical abuse (.03%), and trauma occurring to self (versus witnessing) (43.3%).

[8] Age did not differ among trauma-exposed control participants and PTSD patients, although the nonexposed control participants were somewhat younger than the multiple-trauma PTSD group (Table 3). Both PTSD subgroups were similar in age (21,22,24) or younger (23) than prior PTSD samples that showed robust defensive engagement during aversive imagery.

Discussion

Defensive Physiology and PTSD

As in many prior studies of idiographic trauma imagery, the total cohort of PTSD patients evinced more pronounced heart rate acceleration than control participants (21) and, concordant with more extreme aversiveness ratings, greater facial expressions of displeasure (34,58). Patients also surpassed control participants in startle reflex potentiation during idiographic threat-related imagery, consistent with enhanced limbic (particularly amygdala) and paralimbic activation shown in parallel neuroimaging research (59–61). Although mean skin conductance change loomed larger in PTSD patients than control participants, the groups did not differ significantly (as in a subset
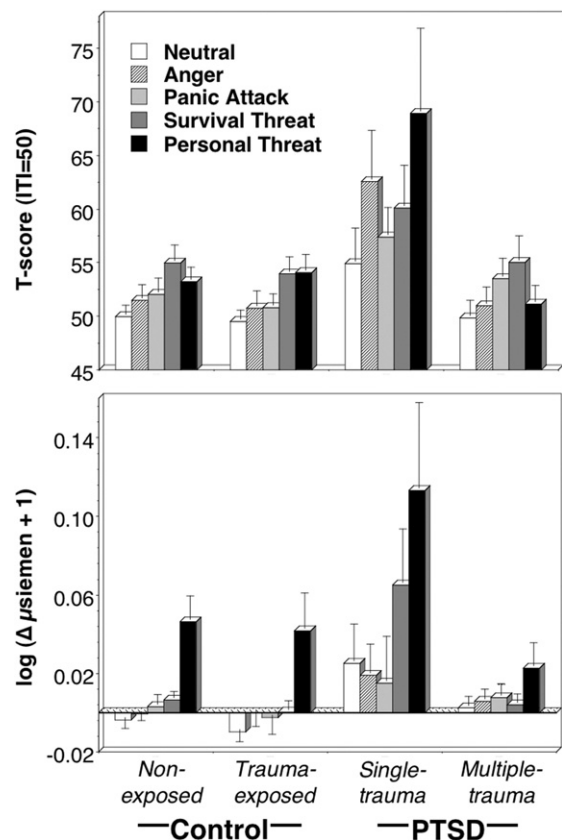

Figure 3. Mean startle reflex responses (top panel) standardized to the distribution of responses during intertrial intervals and skin conductance level change (bottom panel) during neutral, anger, panic, survival threat, and personal threat imagery for nonexposed and trauma-exposed control groups and single- and multiple-trauma PTSD groups. Error bars refer to standard error of the mean. PTSD, posttraumatic stress disorder.

Table 4. Demographic, Interview, and Questionnaire Responses (Means and Standard Deviations) for Control and PTSD Exposure Subtypes

Measure	Nonexposed Control	Trauma-Exposed Control	Single-Trauma PTSD	Multiple-Trauma PTSD	Group Effect
Questionnaire Measures					
ASI total	8.41 (6.40)[c,d]	10.05 (7.45)[c,d]	28.0 (15.74)[a,b,d]	43.17 (9.88)[a,b,c]	$F(3,113) = 89.46, p < .001$
STAXI-Trait	13.89 (3.47)[c,d]	15.23 (4.52)[c,d]	20.63 (7.41)[a,b]	19.46 (6.22)[a,b]	$F(3,117) = 11.53, p < .001$
STAXI-State	10.26 (.74)[c,d]	10.53 (1.74)[c,d]	15.55 (7.28)[a,b]	16.38 (7.64)[a,b]	$F(3,119) = 79.89, p < .001$
STAI-Trait	30.87 (8.51)[c,d]	30.69 (8.82)[c,d]	53.67 (15.92)[a,b,d]	61.87 (8.76)[a,b,c]	$F(3,116) = 73.91, p < .001$
STAI-State	28.59 (7.17)[c,d]	27.87 (7.86)[c,d]	54.55 (16.48)[a,b]	59.37 (10.77)[a,b]	$F(3,119) = 79.89, p < .001$
BDI total	3.00 (3.85)[c,d]	4.30 (5.42)[c,d]	23.29 (11.45)[a,b,d]	28.82 (8.38)[a,b,c]	$F(3,120) = 10.89, p < .001$
QMI total	87.83 (31.91)	75.50 (24.62)	97.95 (38.74)	87.96 (33.07)	$F(3,117) = 2.07,$ ns
Interview Measures					
Re-experiencing symptoms (0–40)			25.86 (8.03)	27.69 (8.15)	$F(1,47) = .62,$ ns
Strategic avoidance symptoms (0–16)			10.20 (4.57)[d]	12.11 (3.77)[c]	$F(1,47) = 2.60, p = .05^{e}$
Emotional numbing symptoms (0–24)			12.50 (6.44)[d]	17.76 (5.37)[c]	$F(1,47) = 9.72, p < .01$
Hyperarousal symptoms (0–40)			24.32 (5.90)[d]	27.67 (6.14)[c]	$F(1,47) = 3.12, p < .05^{e}$
Age of disorder onset (years)			32.09 (12.81)[d]	23.74 (13.15)[c]	$F(1,47) = 5.00, p < .05$
PTSD chronicity (years)			5.05 (10.31)[d]	17.22 (14.25)[c]	$F(1,47) = 11.24, p < .01$
PTSD severity (0-5)			3.77 (.81)[d]	4.44 (.58)[c]	$F(1,47) = 11.41, p < .01$
Prognosis (1–4)			1.86 (.77)[d]	3.04 (.90)[c]	$F(1,47) = 23.38, p < .001$
Comorbid Axis I disorders (count)			1.23 (1.48)[d]	2.52 (1.42)[c]	$F(1,47) = 9.83, p < .01$
Comorbid anxiety disorder (%)			31.8[d]	74.1[c]	$X^{2}(1) = 8.75, p < .01$
Comorbid depressive disorder (%)			51.5[d]	85.2[c]	$X^{2}(1) = 5.58, p < .05$
Demographics					
Age (years)	31.00 (11.08)[d]	33.00 (12.48)	37.14 (13.60)	40.96 (11.14)[a]	$F(3,121) = 4.48, p < .01$
Gender (% female)	67.39	63.33	68.18	74.07	$X^{2}(3) = .77,$ ns
Race (% Caucasian)	80.44	90.00	72.73	77.78	$X^{2}(3) = 2.72,$ ns
College graduate (%)	63.04[d]	56.67[d]	40.91[a]	22.22[a,b]	$X^{2}(3) = 12.36, p < .001$

Note. PTSD symptoms clusters = sum of severity ratings (9-point scale ranging from 0, None, to 8, Very severe) endorsed for each DSM-IV PTSD criterion on ADIS-IV (47) according to clusters (57). Age of onset = patient-reported onset of PTSD diagnosis. Chronicity of PTSD = years from patient-reported onset of diagnosis to assessment. PTSD Severity = clinician-rated severity (6-point scale ranging from 0, No features present, to 5, Diagnosis present; severe) reflecting both distress and interference. Prognosis = clinician-rated estimate of treatment prognosis (4-point scale ranging from 1, Excellent, to 4, Poor).

ADIS-IV, Anxiety Disorder Interview Schedule for DSM-IV; ASI, Anxiety Sensitivity Index (82); BDI, Beck Depression Inventory (85); HSD, honestly significant difference; PTSD, posttraumatic stress disorder; QMI Total, Questionnaire on Mental Imagery (86); STAXI-State, State scale of State Trait Anger Expression Inventory (83); STAXI-Trait, Trait scale STAXI (83); STAI-Trait, Trait scale of State Trait Anxiety Inventory (84); STAI-State, State scale of STAI (84).

[a]Post hoc between-group comparison to nonexposed control is significant at $p < .05$ (results of Tukey HSD pairwise comparisons).
[b]Post hoc between-group comparison to trauma-exposed control group is significant at $p < .05$ (results of Tukey HSD pairwise comparisons).
[c]Post hoc between-group comparison to single-trauma PTSD group is significant at $p < .05$ (results of Tukey HSD pairwise comparisons).
[d]Post hoc between-group comparison to multiple-trauma PTSD group is significant at $p < .05$ (results of Tukey HSD pairwise comparisons).
[e]One-tailed test based on directional hypothesis that multiple-trauma PTSD group would exceed single-trauma PTSD group.

of earlier studies [23,27,30,34]). Both patient and control groups rated their "worst" threatening scenes most arousing and correspondingly evinced their most palpable electrodermal increases when imagining these scenarios.

Posttraumatic stress disorder patients also surpassed control participants in responses to standard unpleasant imagery, rating anger and panic scenes more aversive and arousing and showing greater startle potentiation. Imagery of animal and human survival threats prompted elevated skin conductance in PTSD, similar to reactions reported during imagery of standard exposure to combat (21,22), missile attack (62), and nursing war zone casualties (26). Overall, PTSD patients showed defensive hyperreactivity foremost to trauma-related imagery, as well as a broader sensitivity to aversive cues as found subsequent to shock-threat conditioning (63).

Defensive Physiology and Trauma Recurrence. control participants, whether or not they had experienced prior traumatic events, showed similar, reliable physiological increases and subjective distress during aversive imagery. However, the impression prompted by the overall comparison of patients and control participants—that PTSD patients reliably exceed control

participants in defensive reactivity—was qualified by the subgroup analyses. The hyperreactivity of the PTSD group was carried by less than half the total cohort of patients, specifically those with posttraumatic stress consequent to a single, discrete trauma. Single-trauma PTSD patients showed more robust startle and skin conductance responses than both control participants and multiple-trauma PTSD patients. The latter group, in fact, showed no startle potentiation or conductance increases during personal threat that differed from those amid neutral imagery. Furthermore, single-trauma patients clearly exceeded both control groups in heart rate increases during personal threat; the lesser reaction of the multiple-trauma group did not surpass control participants.

Evaluative Reports and Facial Expressivity. Both single- and multiple-trauma PTSD groups reported that unpleasant imagery prompted greater experienced negative emotion than did control participants, with the highest ratings for multiple-trauma patients, emphasizing a dramatic discordance from this group's impaired defensive activation in autonomic and reflex potentiation. Curiously, corrugator frown muscle action was not strongly discordant with ratings, as it increased significantly during unpleasant
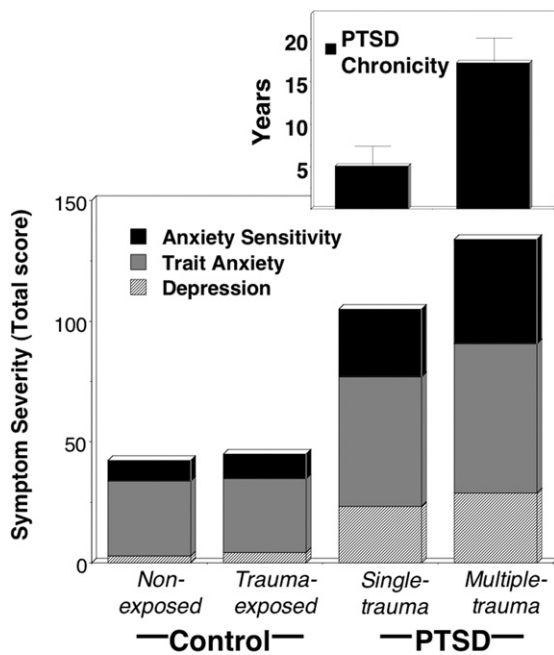
Figure 4. To illustrate the differences in total negative affectivity between groups, mean stacked symptom severity scores on the STAI (trait), BDI, and ASI for nonexposed and trauma-exposed control groups and single-trauma and multiple-trauma PTSD groups are illustrated in the bottom panel. In the top panel, mean duration of PTSD (i.e., disorder chronicity) in years for single-trauma and multiple-trauma PTSD groups are plotted, showing that broad negative affectivity and chronicity demonstrate concurrent increases. ASI, Anxiety Sensitivity Index; BDI, Beck Depression Inventory; PTSD, posttraumatic stress disorder; STAI, State Trait Anxiety Inventory.

imagery in both patient subgroups, suggesting that facial expression and evaluative reports are coordinate communication channels under instrumental control.

Physiological Blunting in PTSD. Current findings for multiple-trauma PTSD of obtunded startle during aversive imagery seem paradoxical. However, Cuthbert *et al.* (34) also observed this phenomenon in PTSD during imagery in their smaller sample. Similarly, a reduced electrodermal response has been reported (64) during threat of shock in PTSD relative to control participants. The reason for this attenuation in multiple-trauma PTSD is unclear. Although dissociation is a viable candidate process, multiple investigators have shown no impact of high dissociative tendencies on the defensive physiology of PTSD during imagery (33,65,66) and the profile of multiple-trauma PTSD here revealed heightened negative facial expressivity during aversive imagery rather than complete defensive suppression (67). Additionally, lack of heart rate increase during aversive imagery has been associated with verbal reports of dissociation in PTSD (68,69); however, here, multiple-trauma patients showed significant heart rate increases relative to neutral processing that, although less pronounced than for single-trauma patients, were equivalent to control participants responding to their worst fear. Considering that multiple-trauma patients endorsed more extreme aversiveness and arousal ratings and equivalent ability to generate vivid imagery to the other groups, the data do not strongly support a dissociation/disengagement interpretation.

Concerning neural mediation of reduced reactivity, several neuroimaging studies found no heightened amygdala activation during trauma imagery in PTSD (70–73) and one study even observed amygdala deactivation (74) relative to control participants. Startle potentiation and skin conductance increases to emotionally salient cues have been described as downstream effects of amygdala activation (15,16,75,76). The present findings of their coincident attenuation in chronic PTSD patients with cumulative trauma histories suggest deficient amygdalar recruitment during internally generated trauma recall that may extend to nontrauma-related contents nevertheless pertinent to the long-term posttraumatic presentation (i.e., anger, panic, physical danger).

Trauma Recurrence, Chronicity, and Comorbidity. The multiple-trauma patients sustained more, higher magnitude traumatic events that began at an earlier age and posttraumatic stress persisted, on average, over three times longer than the single-trauma patients (i.e., 17 years and 5 years, respectively). Recurrent compared with single traumatization was also associated with more severe PTSD that, importantly, was concomitant with more extreme and broader anxious and depressive comorbidity, as quantified by questionnaires (i.e., trait anxiety and anger, cognitive and somatic symptoms of depression), and prevalence of additional anxiety and mood diagnoses.

When compared with prior studies of trauma imagery (23,25,27,28,62,77), the multiple-trauma PTSD sample was particularly extreme in comorbidity of anxiety and depression. For example, the veterans in the Orr *et al.* sample (23) who showed increased skin conductance and heart rate acceleration to idiographic trauma imagery were most similar in trait anxiety (State-Trait Anxiety Inventory; trait M = 48.4) and depression (Beck Depression Inventory = 19.4) to the present highly reactive, single-trauma group. Furthermore, 74% of the multiple-trauma group met criteria for a comorbid anxiety disorder and 85% surpassed the threshold for comorbid depression, far exceeding the prevalence in the single group (anxiety disorder: 32%; depressive disorder 52%). Importantly, previous PTSD samples that demonstrated exaggerated defensive reactivity were generally characterized by depression comorbidity at (21,22) or below (27) the level of the single-trauma group—far below the multiple-trauma group.

The Anxiety Spectrum. The current findings suggest that for PTSD patients attenuated defensive reactivity is associated with broad distress, severe and recurrent trauma exposure, and lengthier disorder chronicity. This blunting phenomenon has been observed not only in other broadly symptomatic anxiety disorders (i.e., panic disorder with agoraphobia and GAD [34,38-40]) but also within fear diagnoses (41). For example, in social phobia, the most severe patients (generalized social phobia with comorbid depression) endorsed the most pronounced negative affectivity and enduring dysfunction but showed the least physiological reactivity during aversive imagery.

High Stress and Defensive Responses. Animal data suggest that variations in stressor intensity, duration, and recurrence can result in dampened defensive responses. For example, using a conditioning paradigm, Davis and Astrachan (78) observed a nonmonotonic relationship between fear-potentiated startle and shock intensity: rats exposed to light cues paired with intermediate levels of shock evinced the greatest conditioned potentiation; rats exposed to low shock intensities demonstrated modest augmentation; and those exposed to the highest shock intensity demonstrated no discernible increase in startle magnitude. Chalmers *et al.* (79) similarly found an absence of conditioned fear potentiation among rats exposed to highly intense, prolonged, and inescapable shock.

More specific to stressor chronicity, animals exposed to brief (i.e., 10 days) and/or less severe "resident/intruder" stress demonstrated hypervigilance and hyperarousal, whereas those exposed to longer duration stress (20 to 30 days) developed more generalized anxiety and depressive-like symptoms—including passivity, limited movement, and reduced communication and consumption behaviors—that persisted even in the absence of the aggressor (80,81).

Conclusion

Single- and multiple-trauma exposures yield identifiably different psychophysiological profiles, obscured when PTSD is considered, irrespective of trauma recurrence. Posttraumatic stress disorder secondary to a discrete trauma is characterized by heightened defensive reactivity during aversive imagery, whereas PTSD after higher magnitude, multiple traumas is marked by higher anxious and depressive comorbidity and a blunted reflex reaction. These findings suggest that trauma accumulation and the associated context may prompt sustained traumatic stress, ultimately impairing defensive physiological reflexes and broadening symptom severity. In summary, patients' verbal reports were consistent with the diagnostic criteria implicating exaggerated hyperarousal in PTSD. However, objective physiological measures revealed that defensive responding did not uniformly increase with PTSD severity. In fact, the most extreme constellation of psychopathology was characterized by a compromised defense response to aversive imagery.

Supplementary material cited in this article is available online.

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