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The Function of Image Control in the Psychophysiology of Posttraumatic Stress Disorder

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The physiological response to trauma-related stimuli of up to one third of participants with posttraumatic stress disorder (PTSD) cannot be discriminated from that of controls. Psychophysiological measures (heart rate and blood pressure) of 22 PTSD and 23 control civilian participants, all exposed to missile attacks during the Gulf War, were recorded while listening to five scripts. The physiological response of PTSD subjects with high image control (IC) was lower than that of PTSD participants with low IC and similar to that of non-PTSD subjects. The physiological response poorly discriminated high IC PTSD participants from controls, but was successful in discriminating low IC PTSD subjects from controls with 91% specificity and 92% sensitivity. Image control is proposed as a function influencing physiological response in PTSD.

KEY WORDS: PTSD; psychophysiology; emotion; image control.

Posttraumatic stress disorder (PTSD) constitutes a complex set of reactions to the overwhelming experience of circumstances perceived as endangering, horrifying, and severely compromising one's sense of security. Symptoms include persistent reexperiencing of the traumatic event, numbing of general responsiveness, avoidance of stimuli associ-

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ated with the trauma, and increased arousal (American Psychiatric Association, 1994).

During the last decade, researchers have measured the physiological reaction of subjects with PTSD to trauma-related stimuli (Blanchard, Kolb, & Prins, 1991; Orr, Pitman, Lasko, & Herz, 1993; Pallmeyer, Blanchard, & Kolb, 1986; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987; Pitman, Orr, Forgue, Altman, et al., 1990; Pitman, Orr, & Steketee, 1989; Shalev, Orr, & Pitman, 1993). Testing the capacity of psychophysiological measures to differentiate pathological from normal reactivated memory of the event may also serve to overcome the biases inherent in diagnosing PTSD based exclusively on the self-report of the individual. It was found that upon exposure to trauma-related stimuli, PTSD combat veterans manifested a significant increase in physiological responses as measured by systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), and frontalis electromyogram (EMG). It was further found that those responses allowed correct discrimination between PTSD and non-PTSD combat participants with a specificity of 75% to 100% and a sensitivity of 60% to 90% (for a review see Orr, 1990).

Lang's theory of emotion is probably the most common theoretical explanation that has been offered for the increased psychophysiological reactivity in PTSD (Orr et al., 1993; Pitman et al., 1987, 1989, 1990; Shalev et al., 1993). Lang defined emotion in cognitive terms, i.e., as a specific information-memory structure organized into a multidimensional network of propositions (Gatchel, Baum, & Lang, 1982; Lang, 1979; Lang, Levin, Miller, & Kozak, 1983). This network, which also includes memory representations of psychophysiological events, is assumed to be accessed as a unit by specific stimuli.

Lang's theory has been applied to the study of physiological responses to fear stimuli (Cook, Melamed, Cuthbert, McNeil, & Lang, 1988) as well as to trauma-related stimuli (Orr et al., 1993, Pitman et al., 1987, 1989; Shalev et al., 1993). Research data, however, showed that about one-third of PTSD subjects were "physiological nonresponders" (Pitman et al., 1989, p. 428), even when presented with individually tailored trauma-related scripts. Research methodology or the overdiagnosis of PTSD via structured clinical interview may be responsible for these data, or the psychophysiological reaction of PTSD nonresponders is less sensitive than that of responders to trauma-related stimuli. Endorsing the view of PTSD as reflective of disrupted control of imagery (Horowitz, 1976, 1983), we propose a third possibility.

Horowitz offers a theory that pertains to the image system in the normal and pathological states. It proposes that PTSD can be formulated as a disorder of image control, which is at the root of the severe

symptomatology. The present study introduces the capacity to control and manipulate mental images as an independent cognitive parameter mediating the physiological sensitivity of PTSD subjects to trauma-related stimuli.

The test of visual imagery control was devised half a century ago by Gordon (1949), who attempted to discriminate subjects who had autonomous imagery (independent of volitional control) from those who had controlled imagery (under conscious volitional control). Since then, research has shown that the capacity to control images normally carries with it cognitive, affective, and physiological components. Subjects with a higher capacity for image control score higher on the Necker Cube reversal state test (Gordon, 1950), are more able to form less stereotypic images, think and perceive creatively, and produce original verbal images (Gordon, 1949; Khatena, 1975, 1976; Shaw & DeMers, 1986). They have a better capacity to recall dreams (Hiscock & Cohen, 1973) and to mentally rehearse motor skills (Rawlings & Rawlings, 1974). Low control of imagery has been found to correlate with neuroticism (Stricklin & Penk, 1980), as well as with state and trait anxiety (Euse & Haney, 1975). Martin and Williams (1990), however, while studying anxiety disorders, found that the capacity to control mental images may be preserved in the face of psychopathology. In the present study, we examine the relation of image control to the physiological reaction of PTSD and non-PTSD participants under two tasks. These tasks involve the manipulation of mental imagery: (1) forming images in response to trauma-related stimuli, and (2) faking the response to the stimuli.

The capacity of non-PTSD individuals to simulate posttraumatic symptomatology has serious social and legal implications. Orr and Pitman (1993) found that 4 out of 16 non-PTSD participants could simulate the psychophysiological reaction of individuals with PTSD to trauma-related stimuli. Gerardi, Blanchard, and Kolb (1989) found a greater proportion of non-PTSD successful simulators. The present study explores the role of the capacity to control and manipulate mental images in simulating PTSD psychophysiology.

The following hypotheses were tested: (a) among individuals with PTSD exposed to the same stressful event, the psychophysiological reaction to trauma-related stimuli is greater in those with low capacity for image control than in those with high capacity for image control; (b) among participants without PTSD, only those with high capacity for image control will successfully fake their physiological response to the trauma-related stimuli; (c) the capacity of psychophysiological data to discriminate PTSD from non-PTSD subjects will be greater for subjects with low image control than for those with high image control.

Methods

The Traumatic Event

The Gulf War broke out in the Middle East on January 15th, 1991, and lasted for 40 days, during which 39 missiles were launched in 17 different attacks on Israeli civilian targets. Each missile alert represented a genuine threat (of conventional and chemical attacks) transmitted throughout the whole country by the sound of the siren. With each alert, families were required to gather fully masked in sealed security rooms until instructed otherwise. The population participating in this study was hit by the first two missile attacks occurring 48 hr apart. After the end of the war, community services resumed their normal functioning within 6 months.

Participants

The sample of the present study was defined to overcome some methodological limitations that had already been identified by prior researchers (Shalev et al., 1993): Both males and females were included. All participants were civilians, and all were exposed to the same traumatic event.

Two groups were defined: (1) the *PTSD* group consisted of 22 participants diagnosed with current PTSD as a result of their exposure to the missile attack and treated at the PTSD Clinic;. (2) following a screening process of 60 consecutive clients applying to the general medical clinic, the *non-PTSD* group was defined, individually matched for socioeconomic status (same low-income neighborhood), sex, and age. This group included 23 individuals with no PTSD symptoms following the war. PTSD and non-PTSD groups also did not differ in marital status or years of education.

The participants, all residing in the same low income neighborhood in the city of Tel Aviv (Israeli bottom quartile: Har-Paz, Alterson, Ganani, Hadad, & Fedida, 1986) directly hit by SCUD missiles, were approached approximately 15 months after the end of the war. All shared the same level of exposure, i.e., they witnessed the attack from a similar distance (100–500 meters) without suffering injury to self or family or marked damage to their homes. They were interviewed by an experienced psychiatrist and diagnosed according to the Structured Clinical Interview for DSM-III-R (SCID) (Spitzer, Williams, Gibbon, & First, 1989). The following disorders served as exclusion criteria: current medical illness, PTSD prior to the Gulf War, depressive episodes during the year prior to the Gulf War, current or prior diagnosis of substance related disorders, organic mental disorders, and psychotic disorders, including schizophrenia as well as bipolar

Table 1. Demographic Characteristics of PTSD and non-PTSD Subjects

	PTSD	non-PTSD
Sex		
Males	9 (41%)	9 (39%)
Females	13 (59%)	14 (61%)
Marital status		
Single	3 (13%)	1 (5%)
Married	18 (78%)	18 (82%)
Divorced	1 (4%)	3 (14%)
Widowed	1 (4%)	0 (0.0%)
Age (*M* and *SD*)	46.1 (15.8)	45.9 (12.7)
Education (Years, *M* and *SD*)	7.1 (3.8)	7.6 (3.8)
Image control (*M* and *SD*)	35.7 (16.6)	32.9 (20.7)
Symptoms scales (*M* and *SD*)		
IES-Intrusion	25.2 (3.6)	8.0 (1.5)
IES-Avoidance	20.9 (2.9)	10.9 (3.6)
IES-Total	46.1 (4.7)	18.9 (4.6)
PTSD Inventory	56.0 (7.2)	18.7 (3.1)
Civilian Mississippi	131.0 (20.6)	63.3 (12.5)

Note. For statistical significance see text.

mood disorder. Table 1 summarizes the demographic characteristics of the sample.

Twelve (55%) individuals with PTSD also met DSM-III-R criteria for major depression; one (5%) had panic disorder with agoraphobia; one (5%) had panic disorder without agoraphobia, and one (5%) had somatization disorder. The control group revealed no psychiatric disorders. The subjects with PTSD had received only short-acting benzodiazepines prior to their entry into the study. The medication was discontinued two weeks before the laboratory session. After a complete description of the study to the subjects, written informed consent was obtained. A structured interview concerning prewar functioning (work, family, and social relationships) revealed that all subjects had functioned well prior to the war.

Instruments

Psychological measures. Each participant completed the following psychometric scales: (a) The Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979); (b) the Civilian Mississippi Scale (Vreven, Gudanowsky, King, & King, 1995); (c) the PTSD Inventory (Solomon et al., 1993); (d) Betts' Questionnaire upon Mental Imagery (QMI; Sheehan, 1967); and (e) Richardson's adaptation of the Gordon Test of Visual Image Control (Richardson, 1969), a 12-item scale which assesses the ability to manipulate visual mental images.

The QMI which measures the capacity to form vivid mental images is a 35-item scale with high test-retest (.76; Sheehan, 1967) and split-half (.95; Juhasz, 1972) reliability, and its validity has repeatedly been demonstrated by high correlations obtained between scores on the test and the direct evocation of imagery in a wide variety of experimental settings (Sheehan, 1967).

In Richardson's adaptation of the Gordon Test [(e) above], the participants were asked about their capacity to change in their minds the properties of a mental image (a car parked in front of their house), i.e., changing the car's color, position, or movement. White, Sheehan, & Ashton (1977), in their review of imagery measures, reported that "Gordon's test of visual imagery control is internally consistent . . ., has adequate test-retest reliability . . ., [and] correlates with other pencil and paper measures of imagery . . ." (p. 153). White and Ashton (1977) reported test-retest reliabilities ranging from .81 to .95 and the internal consistency for the scale (Cronbach Alpha) in our sample was .90. Concerning construct validity, test scores on the Gordon scale were significantly correlated with scores on the Necker Cube reversal states test, which requires a capacity to manipulate (rotate) a mental image (Gordon, 1950).

Physiological examination. The following four physiological variables were measured: Forehead electromyogram (EMG) was measured using two small surface Ag/AgCl pre-gelled electrodes (Beckman Instruments, Inc.) attached to the forehead and centered over each eye. A ground electrode was placed midway between the two active electrodes. Recordings were made with a biofeedback system (Autogenic 8000 Inc.), in which the EMG was rectified and averaged. The recorded EMG instrument activity had a bandpass filter (1 to 300 Hz) to minimize artifacts. A 50 Hz notch filter was used to eliminate 50 Hz interference. The recording duration was 90 sec. Blood pressure (BP) and heart rate (HR) were measured on the left arm with an automatic blood pressure device (Autogenic System, Inc.). After the cuff was inflated for one minute, systolic BP (SBP) and diastolic BP (DBP) were assessed, and mean HR per minute was displayed.

Procedure

Following the collection of the psychological measures, subjects were instructed about the type of physiological examination they were about to undergo (instructions adapted from Blanchard, Kolb, Gerardi, Ryan, & Pallmeyer, 1986). The instructions were structured and read aloud by the experimenter to ensure standardization. Participants were told that the psychophysiological assessment would be initiated with the recording of base-

line measures, and that the assessment would continue while sounds played through the earphones. Participants were invited to sit quietly for 5 to 8 min to adapt to the experimental condition and were instructed to keep their eyes closed and refrain from gross motor movement. They continued sitting quietly for the duration of the baseline EMG measurements and the subsequent BP and HR measurements. The duration of each trial was 5.5 min consisting of an audio period and an image period. During the *audio period*, subjects were told that they would be listening to several audiotapes ("scripts") and were asked to allow themselves to experience anything that came to mind in response to the sounds. No information was given regarding length, number, or content of the scripts. Each audioscript was played for 3 min: 30 sec for mental "setting", 90 sec for EMG measurement, and 60 sec for BP and HR measurements. The remaining 2.5 min constituted the *image period*: The subjects were asked to maintain in their minds the image that had been evoked by the audioscript, but this time without the auditory stimulus. During the first 90 sec of the image period, EMG measurements were taken, followed by 60 sec for BP and HR measurements. At the end of each trial, participants spent 2 to 4 min in a recovery period. They were asked to clear their minds of images, to relax, and to open their eyes.

The audioscripts were played through earphones at the average intensity of 55 decibels. A pilot study showed that stimuli at this intensity, presented to PTSD subjects, are comfortable as well as perceptually and semantically clear. Since the inflation of the cuff might introduce a sensation that influences the EMG measurement, BP and HR were recorded after EMG to prevent interference between the measured variables.

Each subject was presented with five different scripts in six trials. The order of presentation of the first four was counterbalanced. The following scripts were the first four: (a) "Positive experience": Ocean waves customarily used to evoke relaxation; (b) "Action": A car chase, with an outlaw trying to flee law-enforcement agents; (c) "Neutral": Background noise of party chatter; and (d) "Fear": A child screaming, with dogs barking fiercely in the background. Following the first four scripts, all participants were presented with final script, (e) "Event": The missile alert siren followed by the specific distress signals and emergency code-words, followed by the sound of a missile explosion. Following the presentation of the five scripts, the last script (Event) was presented again (sixth trial). This time, both groups of subjects were asked to "fake" their physiological responses ("faking trial"). Instructions were similar to those described by Gerardi, Blanchard, and Kolb (1989) and tailored to our specific traumatic event (missile attack). Individuals with PTSD were asked to emulate the reaction of non-PTSD participants (i.e., trying to keep the heart rate, blood pres-

sure, and muscle tension from increasing when listening to the missile attack sounds), whereas non-PTSD participants were instructed to try and emulate reactions of posttraumatic participants (i.e., control body responses when listening to the missile attack sounds in order to increase the heart rate, blood pressure, and muscle tension).

Data analysis. Since electrode placement during the EMG measurement differs from the standard procedure (see Fridlund & Cacioppo, 1986), analyses concerning EMG were not included in the present report. The physiological variables (HR, SBP, and DBP) were evaluated for both the audio and the image periods of each script. These variables were used in order to define the various parameters reflecting the physiological changes following the psychological stimuli: 1) "Provocability scores" were computed by subtracting the corresponding baseline measures from the response to the audio and the image scores, and 2) "Emulability scores" were calculated by computing the difference between the physiological responses to the Event script and those generated during the faking trial.

To examine the interaction between Group and Image Control on PTSD symptoms and/or the physiological responses to the scripts, subjects were classified based on the sample's median value on the Gordon scale as low (11 PTSD and 13 non-PTSD subjects) or high (11 PTSD and 10 non-PTSD subjects) in image control. The mean and standard deviation of the image control scale of PTSD subjects in the present study were similar to those we found in PTSD Israeli combat veterans in an ongoing study: 35.7 ± 16.6 (this study) and 39.8 ± 19.0 (Laor, Abramowitz, Wolmer, Nakash, & Ron, 1995).

The baseline assessment, as well as the scores obtained for each physiological measure, were subjected to multivariate analyses of variance (MANOVA). Finally, stepwise discriminant analyses were performed on the physiological response to the trauma-related script; first for all subjects, and then separately for subjects scoring low or high on the image control scale.

Results

Psychometric Data

PTSD and non-PTSD subjects did not differ in their capacity for image control, $t(43) < 1$ or in their vividness scores, both ts < 1.

As expected, the PTSD group scored significantly higher than the non-PTSD group on all the specific PTSD symptom scales, $F(4, 35) = 177.6$, $p < .001$: the Intrusion, $F(1, 38) = 612.4$, $p < .001$, and Avoidance, $F(1, 38) = 86.1$, $p < .001$, subscales of the IES, the Civilian Mississippi scale,

Table 2. PTSD and Non-PTSD Physiological Responses to the Event Script and During the Faking Trial

	Event				Fake[a]			
	PTSD		Non-PTSD		PTSD		Non-PTSD	
	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
HR (bpm)	91.5	21.0	74.5	9.3	81.7	13.5	76.2	9.5
SBP (mm Hg)	142.6	22.0	124.0	15.6	134.7	18.6	125.7	13.3
DBP (mm Hg)	88.2	15.7	72.9	11.3	79.0	13.8	74.7	11.5

Note. For statistical significance see text.
[a]During the faking trial PTSD subjects were asked to decrease their physiological response and non-PTSD participants were asked to increase the response.

$F(1, 38) = 172.4, p < .001$, and the PTSD Inventory, $F(1, 38) = 650.4, p < .001$. Men and women did not differ in image control, $t < 1$, nor in any symptom scale score (MANOVA, $F < 1$). Table 1 presents the psychometric profiles of the PTSD and the non-PTSD subjects.

No significant correlations were found between the capacity for image control of PTSD subjects and the IES Intrusion, $r(22) = -.33$; IES Avoidance, $r(22) = -.11$; or Mississippi scale, $r(22) = -.31$ (all $p > .05$). However, a significant correlation appeared with the PTSD Inventory, $r(22) = -.39, p < .05$.

Psychophysiological Data

1. Baseline assessment. Baseline data (HR, SBP, and DBP) were assessed prior to the presentation of the first script. A MANOVA analysis revealed that no main effects were found for Group, $F < 1$, or Image Control, $F(3, 39) = 1.69, p > .05$. Further, the Group × Image Control interaction was not statistically significant, $F < 1$.

2. Physiological response to audio scripts. Mean physiological scores of the PTSD and non-PTSD groups under the event and fake conditions are presented in Table 2. Since physiological responses to the scripts of men and women were not significantly different, the latter were collapsed and sex was not considered in further analyses, HR, $F(5, 39) = .65$; SBP, $F(5, 39) = 1.04$; DBP, $F(5, 39) = .63$; all $p > .05$.

A 5 (Script) × 2 (Group) × 2 (Image Control) MANOVA with repeated measures examined the physiological responses in PTSD and non-PTSD subjects under the different conditions (five scripts) and for the two levels of image control (high vs. low). No Group or Image Control main effect appeared for the three physiological variables (all $p > .05$). A Script main effect revealed that overall, the subjects' physiological responses to the various scripts were different for HR, $F(4, 164) = 3.36, p < .05$; SBP, $F(4, 164) = 5.66, p < .001$; and DBP, $F(4, 164) = 10.65, p < .001$. This difference

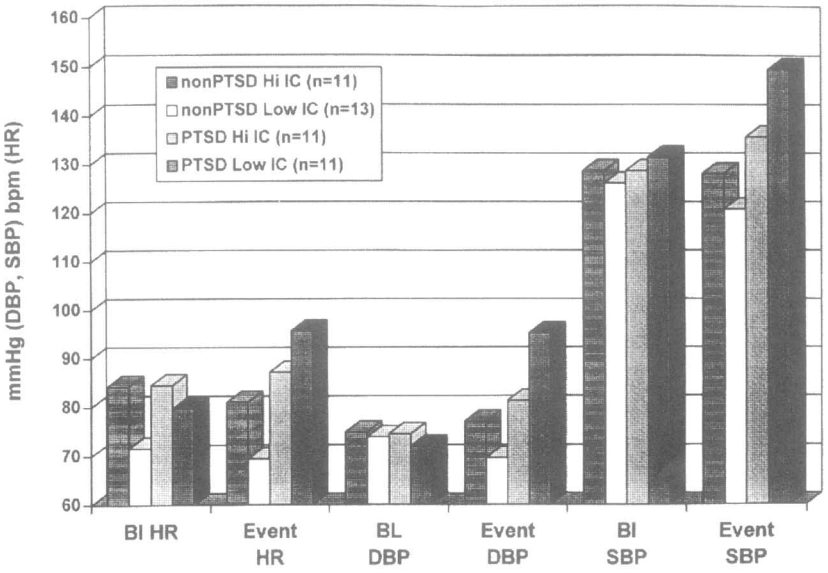

Fig. 1. HR, DBP, and SBP baseline (BI) measures and responses to the Event audio script by Group and Image Control (IC).

is accounted for by higher physiological reaction to the Event script as compared to the reaction to the other scripts. Significant Group × Script interactions for HR, $F(4, 164) = 7.74, p < .001$; SBP, $F(4, 164) = 5.78, p < .001$, and DBP, $F(4, 164) = 11.50, p < .001$; showed that the higher physiological reaction to the Event script appears only in subjects with PTSD. The Image Control X Script interaction was not significant for the four physiological measures (all $p > .05$).

To test our first hypothesis, we compared the subjects' physiological responses to the Event script with their baseline measures (within-subject) through MANOVA with repeated measures analyses. Group and Image Control capacity were considered the between-subject factors. Significant main effects for Group, HR, $F(1, 41) = 10.6, p < .005$; SBP, $F(1, 41) = 20.9, p < .001$; and DBP, $F(1, 41) = 20.3, p < .001$; showed that in subjects with PTSD, the physiological response to the Event script reached values significantly higher than baseline. In non-PTSD subjects, the response to the Event script did not render physiological values that significantly differ from baseline. The Image Control main effects did not reach significance (all $p > .05$). Furthermore, Group × Image Control interactions for SBP (marginal), $F(1, 41) = 5.37, p < .05$, and DBP, $F(1, 41) = 10.4, p < .005$, indicated that the reaction to the Event script was conspicuously higher

among PTSD subjects with low capacity for image control, SBP: $F(1, 10) = 17.9, p < .002$; DBP: $F(1, 10) = 21.9, p < .001$. Figure 1 shows HR, SBP and DBP measures of baseline and response to the Event audio script according to group and capacity for image control (IC).

Pearson correlations were computed between the provocability scores (response to Event minus Baseline) and the capacity for image control in PTSD and non-PTSD subjects. These correlations indicate that among subjects with PTSD, the higher the capacity for image control, the smaller the difference between the reaction to the Event script and baseline, especially when DBP was considered: SBP: $r(22) = -.37, p < .05$; DBP: $r(22) = -.58, p < .005$; and HR: $r(22) = -.32, p > .05$). When PTSD subjects were subdivided by the median score of the IES (46), the negative correlations between image control and the physiological reaction did not appear within highly symptomatic participants (all $p > .30$), but were found to be very high within less symptomatic participants, SBP, $r(10) = -.67, p < .02$; DBP, $r(10) = -.82, p < .005$; HR, $r(10) = -.73, p < .01$.

No significant correlations appeared between measures of PTSD symptomatology and the provocability scores (all $p > .05$). For non-PTSD individuals, we found no significant correlations between image control and the provocability scores. It should be noted that when the correlations were calculated between image control and the physiological responses to the different scripts, without controlling for the baseline measure (as in the provocability scores), the PTSD group demonstrated a single significant correlation, DBP of the Event script, $r(22) = -.45, p < .05$. However, within the non-PTSD group, the correlations with the HR measure of every script were positive and significant: Positive: $r(23) = .54$; Fear: $r(23) = .54$; Action: $r(23) = .58$; Event: $r(23) = .56$ (all $p < .005$).

3. Physiological response during the faking trial. A MANOVA analysis compared the physiological simulation of non-PTSD individuals to the response of PTSD subjects to the Event script. Significant group differences were found for SBP, $F(1, 37) = 17.90, p < .001$, and DBP, $F(1, 37) = 10.95, p < .005$, and marginal differences for HR, $F(1, 37) = 6.08, p < .05$. The means presented in Table 2 show that the physiological response to the Event script was higher in the PTSD group than in the non-PTSD group ("simulation") during the faking trial. No significant Group X Image Control interactions were found.

To test our second hypothesis, we compared the physiological responses to the Event script and during the faking period through MANOVA with repeated measures. These analyses revealed that non-PTSD participants were unable to significantly increase their physiological responses during the faking trial as compared to their response to the Event script (all $p > .05$), whereas individuals with PTSD could decrease their

Table 3. Discriminant Function Analyses of Physiological Data: PTSD vs. Non-PTSD Groups

	Correctly Classified[a]			
	PTSD	Non-PTSD	Wilks λ	χ^2
Total sample	68% (16/22)	87% (18/23)	.617	20.29*
Low image control	91% (10/11)	92% (12/13)	.370	20.85*
High image control	55% (6/11)	70% (7/10)	.821	3.54

[a]Numbers in parentheses refer to the number correctly classified relative to the total number in the given cell.
*$p < .001$.

responses: SBP (marginal): $F(1, 20) = 5.74, p < .05$; DBP: $F(1, 20) = 23.0$, $p < .001$; HR: $F(1, 20) = 11.62$, $p < .005$. Moreover, the physiological values obtained for the PTSD group during the faking trial were not significantly different from those obtained for the non-PTSD group in response to the Event audio script (all $p > .05$). Image Control did not interact with Group on these analyses.

4. PTSD vs. non-PTSD discriminant analysis. We performed stepwise discriminant analyses (p in/out = .05) to test the extent to which physiological data correctly classified PTSD and non-PTSD participants. The predictor variables employed were the physiological responses (provocability scores for HR, DBP, and SBP) to the trauma-related audio script. As can be seen in Table 3, 68% of PTSD and 87% of non-PTSD participants were correctly classified by SBP and DBP.

To test our third hypothesis, similar stepwise discriminant analyses were performed separately for low and high image control subjects (Table 3). The results for low image control subjects showed that 10 out of 11 (91%) PTSD and 12 out of 13 (92%) non-PTSD individuals were correctly classified by SBP and DBP. Within the high image control subgroup, the levels of both specificity and sensitivity were poorer (55% and 70%, respectively). HR and SBP were the significant predictors for the latter analysis.

Discussion

This study tested the relation between the capacity for image control and the physiological response of subjects with and without PTSD to trauma-related stimuli. Before the main results are discussed, two observations contributing to the psychophysiological PTSD literature should be noted. First, the results obtained in this study with civilian males and females are consistent with those obtained in previous studies of male combat veterans. Second, this study found no significant differences in the physiological responses of males and females to the different audio stimuli.

PTSD, Image Control, and Physiological Response

Consistent with previous studies, among the five stimuli presented to the subjects, only the trauma-related stimuli elicited different physiological responses in PTSD and non-PTSD individuals. Thus, it seems that the physiological response in PTSD participants is specifically related to the trauma stimuli and is not engendered by fear-related stimuli (Pitman et al., 1990). However, the increased physiological reaction was not observed in all PTSD subjects and it was dependent on the subjects' capacity for image control.

The findings confirm our first hypothesis: the physiological response to trauma-related stimuli of PTSD participants with low capacity for image control is greater than that of individuals with high image control. The latter is similar to the response of non-PTSD participants (which was independent of image control). Horowitz's (1976, 1983) theory pertains to the image system in the normal and pathological states and assumes an image system integrating perceptual, cognitive, and emotional information. We assume image control to be a cognitive function within this system. Horowitz further conceptualizes trauma as due to stress from the processing of perceptual and emotional information, preventing integration of new with existing data. Despite the prevailing clinical impression that PTSD subjects suffer from disrupted control of imagery that leads, for example, to intrusive symptomatology (Brett & Ostroff, 1985; Horowitz, 1983), our findings show that PTSD measures somewhat correlate with image control and do not correlate with the physiological reaction to the trauma-related stimuli. Since most of the physiological parameters correlate significantly with image control, one may conclude that the loss of control that characterizes PTSD symptomatology does not always result in physiological activation, which, in turn, relates to the capacity for image control. In other words, image control could be viewed as a mediating factor in the system of physiological activation due to posttraumatic stimuli.

Further, as was found in other anxiety disorders (Martin & Williams, 1990), the capacity to control mental images may be preserved in the face of psychopathology; in this study PTSD. Indeed, our findings show that PTSD and control subjects do not differ in their capacity for image control, but in the correlation between the latter and the subjects' physiological response to audio scripts. In PTSD subjects, image control did not correlate with the physiological response to trauma-unrelated scripts, while it correlated negatively with the response to the trauma-related script. This latter finding, however, is demonstrated only in the PTSD participants who display low to moderate symptomatology. For severe PTSD, the correlations between image control and the physiologic response to the trauma-related

script do not occur. The correlations in the non-PTSD group are directly opposite to those found in the PTSD group.

The opposing findings found in the correlations between image control and physiological response in PTSD and non-PTSD individuals suggest that image control may function differently under the different states, i.e., normal and posttraumatic. In non-PTSD individuals, where the physiological response appears within normal bounds, image control may serve a facilitatory function which is reflected in the positive correlations between the two parameters. In individuals with PTSD, a disorder characterized by a dysregulation of the image system, image control appears to serve a protective compensatory function. This function seems neutralized in severe PTSD pathology, but is apparent in subjects with low to moderate PTSD, yet only in the context of the trauma-related script.

PTSD and Non-PTSD Subjects, Ability to Fake Physiological Responses

Our results do not confirm the second hypothesis concerning the capacity of non-PTSD participants with high image control to fake their physiological responses to the trauma-related stimuli. The emulability scores for the non-PTSD group were independent of image control. These results are consistent with those of Orr and Pitman (1993), who reported on non-PTSD individuals' failure at faking physiological responses. In the present study, only the PTSD group was successful at this task. However, when the PTSD group was divided into high and low image control, only the latter showed a significant decrease in the physiological response.

It may be that the subgroup of PTSD participants with high image control displayed a "floor effect" (i.e., the physiological response of this subgroup to the Event script was originally as low as that of normal subjects, and therefore could not be lowered any further by faking). Despite their higher vulnerability to trauma-related stimuli, low image control PTSD participants were successful at faking. These data may indicate that control achieved by faking is exerted not only on the image modulation function but also through some form of distancing, or preventing stimuli from accessing the emotion structure itself. Personal reports of PTSD subjects regarding to their methods of distancing from adverse stimuli support the notion of distancing as an expression of adaptive avoidance. Hence, avoidance need not always be pathological in nature and may also serve a protective function. In the case of PTSD, the control function may appear at times as rigid and brittle, resulting in dissociative symptoms. However, we cannot exclude the possibility that the decreased physiological response

of individuals with PTSD with low image control during the faking trial is a result of some form of habituation.

The response of the non-PTSD low image control group may explain the seeming conflict of our results with Gerardi et al's (1989), that show PTSD failure and non-PTSD success at faking. We view Gerardi et al.'s procedure of six consecutive trials of event audio scripts presented at 40 to 80 decibels as flooding the control functions of both groups of these postcombat subjects. It may be, however, that a procedure using auditory stimuli of intermediate strength could allow us to explore the possible differential physiological responses of non-PTSD participants with low and high image control.

It is important to note that the paradigm underlying the faking trial requires different tasks of PTSD and non-PTSD individuals. This difference is a clear limitation on our capacity to use this paradigm to test hypotheses concerning the psychological mechanisms involved. It does allow us, however, to approach cautiously the difficult problem of client malingering that so often plagues the diagnostician in the area of PTSD.

Discriminant Analyses: Identifying the PTSD Nonresponders

The discriminant analyses performed confirmed our third hypothesis, namely, that physiological responses of PTSD and non-PTSD groups permit a clearer discrimination of low image control subjects, compared to the discrimination of high image control. PTSD and non-PTSD participants with low image control were classified with a sensitivity of 91% and specificity of 92%. Classification rates of high image control PTSD and non-PTSD individuals were significantly lower (55% and 70%, respectively). When participants were not divided according to image control, our results were similar to those previously reported in the literature (Blanchard et al., 1991; Orr et al., 1993; Pallmayer et al., 1986; Pitman et al., 1987, 1990; Pitman et al., 1989; Shalev et al., 1993). Thus, individuals who were considered as "PTSD nonresponders" (Pitman et al., 1989) may be characterized by high capacity for image control.

Conclusions

The present study provided an experimental account for the failure of some individuals with PTSD to exhibit increased physiological response to trauma-related stimuli. This failure is explained partially by individual variation in the functional characteristics of the image system, particularly in

the independent parameter of image control. Introducing this parameter increases the capacity of psychophysiological data to discriminate PTSD from non-PTSD groups.

Image control, however, does not allow the complete discrimination between PTSD and non-PTSD participants. Hence, although a function in the physiological expression of PTSD, it cannot exclusively account for the loss of control specific to the disorder. Future research may shed more light on other aspects of mental imagery that play a role in the pathological dysregulation characteristic of PTSD and its physiological expression. Questions remain about whether or not the loss of control of individuals with PTSD that is expressed in physiological terms pertains solely to the image system, and/or is sufficiently captured by the visual image control measure used in the present study, and whether or not reduced image control in itself constitutes a vulnerability factor for PTSD.

To conclude, our study establishes in PTSD the link between psychophysiological response to trauma-related scripts and image control. Our data may argue for theory development. However, even at this preliminary stage of research, our findings may invite clinical experimentation with cognitive psychotherapeutic techniques aimed at improving image control in individuals with PTSD, particularly those suffering from symptomatology of increased arousal.

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