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The Impact of Fear Activation and Anger on the Efficacy of Exposure Treatment for Posttraumatic Stress Disorder

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This paper explores the hypothesis that fear activation during exposure treatment promotes improvement. Twelve female assault victims diagnosed with posttraumatic stress disorder (PTSD) received treatment that included prolonged repeated reliving of the assault in imagination. Two measures of fear activation were used: facial fear expression coded from videotapes of the first reliving session and the client's highest reported distress score during the same session. The results indicated that clients who evidenced more severe PTSD prior to treatment displayed more intense facial fear expressions during the first reliving of the assault and benefitted more from treatment than did clients who had less severe PTSD and displayed less fear. In contrast, clients who reported more anger prior to treatment tended to display less fear expression during reliving of the trauma and benefitted less from treatment than less angry clients. The relationship of pretreatment PTSD and anger severity to improvement seems to be mediated by fear facial expression and were not simply a product of regression toward the mean of extreme pretreatment scores. The results are discussed within an emotional processing theory of fear.

Exposure-based treatments have proven quite effective in reducing fear and anxiety in various anxiety disorders, including obsessive compulsive disorder,

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agoraphobia, specific phobia, and social phobia (e.g., Foa, Rothbaum, & Kozak, 1989). Posttraumatic stress disorder (PTSD) has been viewed as an anxiety disorder, and therefore variants of exposure treatments have been employed with it as well. Because the anxiety experienced by PTSD sufferers typically is evoked by memories of their traumatic experience, exposure treatment for them has consisted of repeated reliving of the trauma in imagination. This treatment approach has been found effective in reducing PTSD symptoms in Vietnam veterans (Boudewyns, Hyer, Woods, Harrison, & McCranie, 1990; Keane, Fairbank, Caddell, & Zimering, 1989) and in criminal assault victims (Foa, Rothbaum, Riggs, & Murdock, 1991). The present paper explores processes underlying the efficacy of exposure treatment for posttrauma pathology.

To understand why treatment is effective, one must be able to conceptualize the pathology that the treatment aims to correct. Lang's (1977, 1979) bioinformational theory is one framework that has been employed to explain pathological anxiety. It proposes that fear is represented in memory as a structure that includes representations of feared stimuli, fear responses, and the meaning of these stimuli and responses. The fear structure is distinguished from other cognitive structures by the association of stimulus and response elements to threat meaning. Using this framework, Foa, Steketee, and Rothbaum (1989) suggested that a traumatic memory can be conceptualized as a fear structure that includes representations of trauma-related stimuli, responses, and their meaning. A pathological trauma structure, they proposed, is distinguished by the presence of a large number of stimulus representations associated with danger and by particularly strong response elements.

Foa and Kozak (1986) suggested that if treatment for anxiety is to be successful, it must correct pathological elements of the fear structure. They further proposed that two conditions are necessary for such correction to occur. The treatment must 1) activate the fear memory, and 2) introduce new information that is incompatible with the pathological elements of the structure. Much emphasis has been placed on the role of fear activation in the treatment of pathological anxiety. Lang (1977), for example, suggested that the activation of affect during systematic desensitization is the key to the success of the treatment. Similarly, Wolpe (1978) noted that individuals who did not benefit from systematic desensitization appeared unable to experience fear while imagining their feared situation. Empirical studies support the hypothesis that fear activation is positively related to the efficacy of treatment for anxiety. Phobic individuals who were best able to experience anxiety (measured by increased heart-rate) during imagery of their feared situations benefited most from treatment (Borkovec & Sides, 1979; Lang, Melamed, & Hart, 1970; Watson & Marks, 1971). Similarly, physiological responsiveness during exposure to feared situations or images was positively related to treatment outcome in agoraphobic patients (Watson & Marks) and in obsessive compulsive patients (Kozak, Foa, & Steketee, 1988).

The present study explores further the hypothesized association between fear activation during exposure and treatment outcome in a group of assault

victims with chronic PTSD. Facial expressions of fear, rather than the more commonly used autonomic activity, were used as a measure of fear activation, because they constitute a more specific measure of emotional activation. For example, the pattern of facial movement that indicates fear in Ekman and Friesen's (1978) coding system is different from the pattern that indicates anger or elation. In contrast, heart-rate elevation occurs during situations that provoke fear, anger, or elation. Self-reported distress during prolonged reliving also was used as a measure of fear activation. It was predicted that more intense fear expressions during reliving of the traumatic experience in therapy (and perhaps self-reported distress) would be related to greater improvement in posttrauma psychopathology.

Furthermore, it was hypothesized that these measures of fear activation during treatment would mediate any observed association between initial symptoms and outcome. The intensity of fear responses during the first exposure session, rather than during later sessions, was used as an indicator of fear activation because it was thought to reflect better the client's ability (or willingness) to engage emotionally with the traumatic memories. First, during exposure treatment, fear responses usually decrease across sessions (Foa & Kozak, 1986) and, thus, fear responses during later sessions may not be as sensitive as those during the first session for detecting individual differences in fear activation. Second, because the degree of fear during later sessions can be conceptualized as an indicator of outcome itself, to use it as a predictor of improvement could well be perceived as tautological. One factor that may impede fear activation is anger (Butler, 1975; Goldstein, Serber, & Piaget, 1970; Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992). Anxiety and anger have been conceptualized by many authors as alternative or incompatible emotional reactions to threat (e.g., Danesh, 1977; Zillman, 1979; Zwemer & Deffenbacher, 1984). If anger and fear are incompatible reactions to threat, then the presence of anger should inhibit the experience of fear during exposure and, consequently, reduce treatment efficacy. This hypothesis was also examined in the present study.

Method

Subjects

Twelve female assault victims, 7 rape and 5 nonsexual assault, participating in a project to evaluate the efficacy of treatment for PTSD, were included in the present study. Eight victims were Caucasian, and 4 were African American. Mean age was 39.4 years (range 29–55) and mean time since assault was 3.2 years (range 3 months to 24 years). All had sought treatment for psychological symptoms related to their assaults and met DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria for PTSD based on the Structured Clinical Interview for DSM-III-R (SCID-R; Spitzer, Williams, & Gibbon, 1987). Diagnostic interviews were conducted by masters and doctoral level psychologists trained in the use of the SCID-R. Because this training involved

reaching a criteria of at least .90 interrater reliability, we did not obtain diagnostic reliability for the patients in this study. Victims were excluded from the treatment outcome project if they met DSM-III-R criteria for schizophrenia, organic mental disorder, substance abuse, or bipolar disorder or if they did not speak English. In addition, victims who were assaulted by a spouse, family member, or intimate partner were also excluded.

Procedure

Participants in this study received cognitive behavioral treatment for assault-related PTSD. Treatment consisted of nine twice-weekly individual sessions lasting 90 minutes each. The first two sessions were devoted to gathering information about the trauma and the client's symptoms; the remaining sessions were devoted to active intervention. Active treatment sessions included the repeated reliving of the traumatic experience. The client was instructed to close her eyes and recount the events surrounding her assault, including the event, her actions and the perpetrator's actions, and her emotional responses (including physiological responses such as heart beat) and thoughts. The therapist stressed the importance of reexperiencing the emotions that the client felt at the time of the assault. The instructions were as follows:

> I'm going to ask you to recall the memories of the assault. It is best for you to close your eyes so you won't be distracted. I will ask you to recall these painful memories as vividly as possible. We call this reliving. I don't want you to tell a story about the assault in the past tense. Rather, I would like you to describe the assault in the present tense, as if it were happening now, right here. I'd like you to close your eyes and tell me what happened during the assault in as much detail as you remember. This includes details about the surroundings, your activities, the perpetrator's activities, how you felt including your physiological responses like your heart beating fast, and what your thoughts were during the assault. If you start to feel too uncomfortable and want to run away or avoid it by leaving the image, I will help you to stay with it. We will audiotape the narrative so you can take the tape home and listen to it. From time to time, while you are reliving the assault, I will ask you for your anxiety level on the 0 to 100 SUDS scale. Please answer quickly and don't leave the image.

The reliving continued for 45 to 60 minutes. The therapist probed for more details when the client did not provide enough details about the trauma. When the recounting of the trauma lasted less than 45 minutes, the client was asked to repeat it again and again for 45 minutes. Thus, reliving often constituted recounting the trauma several times. The reliving portion of the session was terminated when the client indicated some reduction of subjective anxiety on the measure of subjective distress described below. Each session of recounting

was audiotaped and the tape was given to clients with the instructions to listen to it at home daily and to try to relive the trauma while doing so. Thus, during the course of treatment, clients had the opportunity to relive their assault anywhere between 35 to hundreds of times, depending on the length of their narrative and number of homework assignments they completed.

The therapist and the client then discussed the reliving experience, focusing on changes in the recounting between and within sessions. Also, the reduction in distress that had taken place during the session was pointed out to the client with the emphasis that direct confrontation with the memory did not result in loss of control or "going crazy."

Clients were assessed within 2 weeks before treatment and immediately after the last treatment session.

Measures of Psychopathology

The *State-Trait Anxiety Inventory* (STAI; Spielberger, Gorsuch, & Lushene, 1970) contains 40 items, 20 for state anxiety and 20 for trait anxiety. The trait-anxiety scale was not included in the study because it is designed to measure a stable characteristic that was not hypothesized to change weekly.

The *Structured Clinical Interview for DSM-III-R I and II* (SCID-R; Spitzer et al., 1987) is a diagnostic interview to acquire information about DSM-III-R Axis I and II criteria.

The *PTSD Symptom Scale* (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993) is a standardized interview consisting of 17 items that correspond to the DSM-III-R symptoms of PTSD. Each item is rated on a severity scale of 0 (no symptoms) to 3 (most severe symptoms) so that the total score ranges from 0–51. Internal consistency for the scale was .85 and test-retest reliability across 1 month was $r = .80$. Interrater reliability for the interview was kappa = .91 for diagnosis and $r = .97$ for symptom severity. It is strongly correlated with Intrusion ($r = .73$) and Avoidance ($r = .63$) subscales of the Impact of Events Scale (Horowitz, Wilner, & Alvarez, 1979), and with Kilpatrick's (1988) Rape Aftermath Symptom Test [RAST] ($r = .79$). The PSS has been found to be sensitive to treatment effects (Foa & Riggs, 1993).

The *State-Trait Anger Inventory* (ANGER; Spielberger, 1988) is a 20-item questionnaire that evaluates feelings of anger. It comprises a 10-item state anger scale that evaluates the intensity of anger at the time the instrument is completed, and a 10-item trait anger scale that evaluates general feelings of anger. Only the state anger scale was used in the present study. The internal consistency (Cronbach's alpha) for the state anger scale is .93.

The *RAST* (Kilpatrick, 1988) is a 70-item self-report inventory of psychological symptoms and potentially fear-producing stimuli rated on 5-point Likert type scales. The RAST includes those items from the Derogatis SCL-90-R (1977) and the Veronen-Kilpatrick Modified Fear Survey that most differentiates rape victims from nonvictims. Only the items from the Modified Fear Survey were used in the present study.

Measures of fear activation. Subjective Units of Distress (SUDS), used as ratings of distress ranging, from 0 to 100, were obtained from the client every 10 minutes during the reliving of the assault. This measure has been used pre-

viously to assess habituation of fear during exposure sessions (Foa & Chambless, 1978; Foa, Grayson, & Steketee, 1982). In the present study, the highest rating made during the first reliving session was used.

The *Facial Action Coding System* (FACS; Ekman & Friesen, 1978) is a comprehensive anatomically based system that distinguishes all visually distinct facial movements. In discussing the FACS, Fridlund (1994) concluded that "One major shortcoming of past research has been solved. Anecdotal or inductive reportage of human facial displays has been supplanted by anatomically based facial scoring . . ." (p. 315). The FACS emphasizes patterns of movement and the changing nature of facial appearance. It includes codes for 46 single action units (AUs) that are coded for presence/absence and, in some cases, intensity. Particular emotions are defined by specific combinations of action units. The magnitude of the emotion was calculated as the product of the intensity (1, 2, 3) and the duration of a given pattern of action units (maximum 15 seconds) producing a possible range of 0 to 45. The third author performed all the coding for the present study after receiving 60 hours of training on the FACS by the fourth author, who had passed the test of reliability administered by Ekman and Friesen. The fourth author supervised the coding in the study.

In the present study, AUs, defined by Ekman and Friesen (1978) or by Wiggers (1982) as fear and anger, were coded. The AUs recorded as fear were: AUs 1 (inner brow raise) + 2 (outer brow raise) + 5 (upper lid raise) + 20 (lip stretch) and 25 (lip part) or 26 (jaw drop) or 27 (mouth stretch). The AUs recorded as anger were: AUs 4 (brow lower) + 5 (upper lid raise) or AUs 4 (brow lower) + 23 (lips tight). None of the clients exhibited facial movements that indicated the presence of anger, so this measure could not be analyzed. Facial movements were coded from 15 second segments of videotaped treatment sessions during which female assault victims with PTSD relived their trauma experience.

During these reliving sessions, clients are routinely asked to report how distressed they feel on a 0 to 100 distress scale. The segments selected for coding were the 15 seconds immediately preceding the victim's highest reported distress level during the first reliving session. These segments were selected and marked on the video by a research assistant who was uninformed as to the hypotheses of the study. Thus, the coder, who was not uninformed of the hypotheses, had only access to the 15-second sections and was uninformed of the subjective distress level, the degree of habituation on SUDS between and within sessions, and the client's pre- and post-scores on the outcome variables. We selected the 15-inch segments prior to highest SUDS for facial expressions coding because these were thought to represent the highest fear experienced by the client during the session. Because of the time consuming nature of facial action codings, it is customary to use short segments (e.g., Ekman, Friesen, & Ancoli, 1980; Levenson, Ekman, & Friesen, 1990). Indeed, each 15 second segment required 1 hour of coding time because the segment was reviewed repeatedly using slow motion until 10 codings yielded the same score.

TABLE 1
MEANS AND STANDARD DEVIATIONS OF PSYCHOPATHOLOGY MEASURES AND FACS*
PRE- AND POSTTREATMENT

	Pretreatment	Posttreatment	*t*-tests	*df*	*p*
PSS*	30.92 (11.55)	12.67 (6.16)	4.93	11	<.001
RAST*	94.75 (39.41)	52.18 (31.32)	4.58	10	<.01
STAI*	49.58 (12.30)	34.42 (7.19)	4.18	11	<.01
ANGER*	13.73 (5.64)	10.75 (2.60)	1.67	10	NS
FACS*	14.58 (13.32)	–			
SUDS*	92.83 (9.58)	–			

* PSS = PTSD Symptom Scale total score; RAST = Rape Aftermath Symptom Test fear cue score; STAI = State-Trait Anxiety Inventory state score; ANGER = State-Trait Anger Inventory state anger score; FACS = Facial Action Coding System fear score; SUDS = Highest Subjective Units of Distress.

Results

Treatment Efficacy

Means and standard deviations were first calculated separately for pre- and posttreatment scores for all measures of psychopathology. These are presented in Table 1.

Inspection of the means revealed that, after treatment, PSS, RAST, and STAI decreased. An examination of individual change scores indicated that all clients showed reduction in psychopathology after treatment, although the degree of change varied across clients and across measures. Because all clients had chronic PTSD (more than 3 months post assault; mean of 3.2 years post assault) and because clients in the wait-list control group of the study did not show significant reduction in psychopathology, the decrease in symptoms observed in the clients presented here was interpreted as reflecting improvement due to treatment.

The total fear expression score for each client, as measured by the FACS, was calculated and the mean and standard deviations of FACS and SUDS scores were obtained for the sample (see Table 1).

Relationship Among Psychopathology Measures and Percent Improvement

In order to examine the relationship between measures of psychopathology and fear activation, we calculated Pearson correlation coefficients. Prior to treatment, intercorrelations among measures of PTSD and trauma-related fears (RAST) were significant. Fear expression on the FACS was positively correlated with pretreatment PSS, $r(10) = .65, p < .05$; there was a trend for a negative correlation with pretreatment anger, $r(9) = -.44, p < .10$. Self-rating of subjective distress during reliving was significantly correlated with pretreatment PSS, $r(10) = .78, p < .01$ and RAST, $r(10) = .54, p < .05$. Unfortunately,

after Bonferroni correction, only the relationship between pretreatment PTSD and self-rating of subjective distress was significant.

The relationship of initial psychopathology, fear activation, and the degree of improvement during treatment was examined using Pearson correlations. Improvement was defined as the percentage of reduction in symptoms relative to pretreatment levels. In order to obtain a reliable and broad estimate of improvement in trauma-related anxiety percentage improvement scores for measures of psychopathology (PSS, RAST, STAI) were summed and divided by 3. This composite score comprised the three DSM symptom clusters: reexperiencing, avoidance, and increased arousal. The RAST is conceptually related to the reexperiencing and avoidance clusters, and the STAI is conceptually related to the increased arousal cluster.

Facial expression of fear was correlated with pretreatment composite psychopathology measure $r(10) = .65, p < .05$, reduction of PSS, $r(10) = .53$, $p < .05$, RAST, $r(9) = .53, p < .05$, STAI, $r(10) = .52, p < .05$, and the composite psychopathology measure, $r(9) = .78, p < .01$. Only the correlation between facial expression of fear and the composite psychopathology measure was significant after the Bonferroni. Subjective distress was related to pretreatment composite psychopathology measure, $r(10) = .51, p < .05$, reduction in PSS, $r(10) = .71, p < .01$, RAST, $r(9) = .58, p < .05$, and the composite overall anxiety measure, $r(9) = .61, p < .05$. Unfortunately, only the correlation between subjective distress and PSS was significant after the Bonferroni.

Because of the small number of subjects in the study, we examined whether the observed correlations were influenced by a few outliers by plotting the individual scores of: (a) initial PSS and FACS; (b) initial PSS and percent improvement on the composite overall anxiety measure, and (c) FACS and percent reduction of the composite psychopathology measure. All three scatterplots suggested that the obtained correlations were not due to a few outliers

In order to investigate the hypothesis that fear activation mediates the relationship between pretreatment symptoms and improvement, we first examined the relationship among SUDS, FACS, and percent reduction on the composite psychopathology scores (i.e., improvement scores). SUDS scores were not significantly correlated with improvement scores when FACS scores were partialled out. In contrast, FACS scores remained significantly correlated with improvement when SUDS scores were partialled out. Therefore, subsequent analyses to test the mediational hypothesis were conducted on FACS scores only. We then computed three partial correlations between improvement scores and each of the three pretreatment measures partialling out FACS scores. Next, we computed three partial correlations between FACS scores and improvement scores, partialling out each of the three pretreatment measures.

In support of the mediational hypothesis, when the FACS scores were partialled out none of the correlations between pretreatment measures and improvement remained significant: PSS, $r(8) = .33$; Anger, $r(7) = -.35$; and RAST, $r(8) = -.18$. In contrast, all correlations between FACS scores and improvement remained significant after scores of pretreatment measures were partialled out. When PSS was partialled out, the correlation was $r(8) = .61$

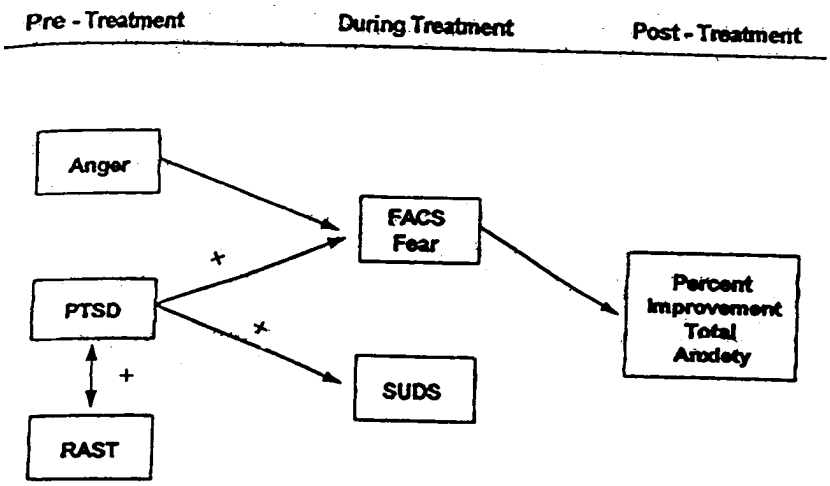

FIG. 1. Model of the relationships among anger, facial fear expression, subjective distress, pretreatment symptom severity, and percentage of symptom improvement post treatment.

$p < 05$; when Anger was partialled out, the correlation was $r(7) = .71$. $p < .05$; when RAST was partialled out, the correlation was $r(8) = .78$, $p < .05$. The model suggested by these partial correlations is depicted in Figure 1.

In summary, higher pretreatment PTSD symptoms as well as greater facial expression of fear and greater distress ratings during the first reliving session were related to greater reduction in outcome measures. In contrast, high pretreatment anger was related to inferior outcome. The partial correlations suggest that the effects of the pretreatment measures on outcome were mediated by facial expression of fear.

Discussion

The present study demonstrates that prolonged, repeated reliving of the trauma ameliorates posttrauma psychopathology. All clients improved with treatment, although the degree of improvement varied across clients and measures. These results converge with those found in other studies (e.g., Foa et al., 1991; Keane et al., 1989). The results also indicate that clients who evidenced more severe trauma-related psychopathology prior to treatment displayed more intense facial fear expressions during the first reliving of the assault and benefitted more from treatment than those who had less severe psychopathology and displayed less intense fear. In addition, clients who reported more anger prior to treatment tended to display less fear during reliving of the trauma and benefitted less from treatment than clients who were less angry.

Partial correlations supported the hypothesis that facial fear expressions during reliving of the trauma mediated the effects of pretreatment psycho-

pathology on outcome and were not simply a product of regression toward the mean of extreme pretreatment scores. The finding that more intense fear expressions during reliving facilitated improvement in trauma-related pathology is consistent with Foa and Kozak's (1986) proposition that fear activation during exposure treatment is a necessary condition for successful outcome. These results converge with those of other studies that measured fear activation via increase in heart rate. With simple phobic individuals, Lang et al. (1970) found that clients who evidenced higher heart rate response during the first imaginal exposure to feared stimuli manifested greater improvement in their phobias. Similar results were reported with obsessive compulsive patients; a strong positive correlation was found between heart-rate increase during the first in vivo exposure to the patients' most feared situations and change in measures of obsessional fear (Kozak et al., 1988).

The results of the present study suggest that a victim's inability to successfully process a trauma may arise from a victim's difficulties in engaging emotionally with the traumatic memories. Further support for the importance of emotional engagement comes from studies demonstrating a positive relationship between dissociation and chronic PTSD (for a review, see Foa & Hearst-Ikeda, in press).

In the present study, we have investigated the role of fear activation in exposure treatment using a specific measure of emotional expression of fear rather than the customary measures of autonomic nervous system activation (e.g., heart-rate, skin conductance). The fact that facial expression of fear, like autonomic arousal, relates positively to improvement following treatment strengthens the argument that fear activation is a prerequisite for emotional processing. However, Foa and Kozak (1986) noted that fear activation is a necessary, although not sufficient, indicator of successful emotional processing during treatment; habituation within and across sessions is also necessary to conclude that the cognitive structure underlying the disorder has been corrected. The present study has addressed only the first assertion (i.e., that fear activation is a necessary condition for successful outcome); it did not examine the second hypotheses that habituation and correction of the pathological elements of the trauma structure are required for improvement.

As noted earlier, angry clients tended to display less intense facial fear expressions and benefitted less from treatment than did less angry clients. These results converge with the findings that intense anger shortly after an assault predicted PTSD severity one month later (Riggs et al., 1992). If PTSD symptoms indicate a failure to emotionally process the traumatic event, then anger may impede the mechanisms underlying both "normal" emotional processing and processing during treatment. The tendency for a negative association between anger and facial fear expression may imply that anger impedes processing of the trauma by inhibiting the activation of fear. That anger inhibits fear was also suggested by Butler (1975) and Goldstein et al. (1970), who used anger to modulate fear during desensitization.

The mechanism by which anger blocks fear activation is unclear. The finding that no client showed facial expressions of anger during the most distressing period of reliving suggests that anger did not simply replace fear in clients who expressed little fear. Perhaps anger reflects an avoidant style of coping with emotional pain, and by mobilizing diffuse anger, one is able to describe the traumatic experience without reliving it emotionally. Foa and Riggs (1993) suggested that the numbing symptoms of PTSD reflect an attempt to evade emotional pain when attempts at effortful avoidance fail. It is possible that anger represents another means by which individuals with PTSD can regulate distress when the emotional pain cannot be avoided. Results from a factor analytic study of PTSD symptoms support the suggestion that numbing and anger are related; numbing symptoms (i.e., detachment from others, restricted affect, loss of interest in activities) and irritability loaded on the same factor (Foa, Riggs, & Gershuny, in press).

An alternative explanation to the negative relationship between anger and fear activation in the first session is that angry clients may be initially reluctant to comply with the therapist's instructions to engage in the distress-evoking task of reliving the trauma. This explanation would lead to the prediction that if anger and fear activation were measured at a later phase of treatment, the negative association between the two variables would dissipate. The covariation between these two variables across different phases in therapy should be examined in future studies.

It is important to note that neither the intensity of fear during the first exposure session nor the initial anger level were thought to be the only predictors of improvement. Other variables such as compliance with homework assignment, the number of trauma relivings, the number of previous traumas are likely to affect treatment efficacy, and the relationships of these variables to anger and fear activation need to be examined.

A word of caution regarding the present findings is warranted. First, the sample of this study is rather small for a correlational investigation. Indeed, the size of the sample prevented the testing of the full mediational model proposed here via regression analyses as suggested by Baron and Kenny (1986); instead, the small sample dictated the use of partial correlations which tested separately the different parts of the model. Second, the data on facial fear expression were based on short time samples from one treatment session. Third, the negative correlation between pretreatment anger and facial fear expression was only moderate (−.44) and did not reach significance with the small sample of the present study.

Future investigations should not only attempt to replicate and extend the present study but also examine the utility of facial expression measures for identifying other indicators of emotional processing. Facial expression coding may be a valuable methodology for studying the processing of traumatic events because it provides a sensitive measure that can distinguish emotions such as fear, guilt, anger, and disgust that are often evoked by traumatic experiences.

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