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EMOTIONAL NUMBING IN COMBAT-RELATED POST-TRAUMATIC STRESS DISORDER: A CRITICAL REVIEW AND REFORMULATION

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ABSTRACT. *Emotional numbing symptoms are considered in the clinical literature as cardinal signs of Post-Traumatic Stress Disorder (PTSD) and have been formally codified in DSM-III-R. However, the term has not been consistently defined nor adequately researched. The present paper critically reviews the extant empirical and theoretical literature in combat-related PTSD that has explored emotional numbing symptoms. A theoretical framework, based on Levanthal's (1984) perceptual-motor theory of emotion, is posited to account for the parameters of emotional processing in PTSD, and specific hypotheses concerning selective or differential emotional processing deficits in PTSD are described in order to clarify empirical issues about the development and maintenance of emotional processing deficits in PTSD and to stimulate future research in this underexplored, yet clinically important area.*

Emotional numbing (EN), or restricted range of affect, has become an accepted diagnostic feature of Post-Traumatic Stress Disorder (PTSD) both in the clinical literature and in the official psychiatric nomenclature, the DSM-III-R (APA, 1987). However, there is little empirical evidence that EN is a cardinal symptom of PTSD, or that it is useful for distinguishing between PTSD and other diagnostic categories. To date, there have been few systematic investigations into the phenomenology, response parameters, or prevalence of EN in PTSD. The research data that are available chiefly consist of survey and clinical observations (e.g., Horowitz, Wilner, Kaltreider, & Alvarez, 1980; van der Kolk & Ducey, 1989).

A major obstacle to research thus far is that EN has never been adequately defined either conceptually or operationally. For example, DSM-III-R suggests two contrasting definitions. Broadly defined, EN can be seen as a generalized deficit in emotional responding. Narrowly defined, EN can be seen as a deficit in emotional responding to positive events only (i.e., restricted range of affect). It is not clear in either case what

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precipitates or maintains EN, nor is the relationship of EN to other symptoms of PTSD well understood. In fact, it is not even clear whether EN results directly from traumatization experiences or from other post-trauma, co-morbid problems such as depression or substance abuse. Also, it is difficult to reconcile a broad definition of EN with other well-defined and researched symptoms of PTSD: for example, states of fear, anger, disgust, or sadness that occur in response to exposure to trauma-related stimuli (Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). These issues suggest the need for a clearer operational definition of the emotional functioning deficits in PTSD.

There are many possible explanations for the "numbness" or lack of emotional responsiveness that some PTSD patients report: (a) Numbing symptoms may result from chronic avoidance of trauma-related stimuli that, in effect, have limited patients' contact with interpersonal stimuli that would otherwise elicit a wide range of emotional reactions; (b) emotional stimuli may be available but not attended to; (c) emotional stimuli may be perceived but the emotional response is inhibited or thwarted; or (d) PTSD patients may expend so much effort coping with re-experiencing symptoms and hyperarousal that they exhaust their affective resources.

Each of the above hypotheses suggests specific empirical questions that need to be explored so as to more clearly delineate the parameters of emotional processes in PTSD. This task is best accomplished by application of a theoretical model that can yield testable differential predictions. At present emotional-processing variables have not been specifically included in theoretical models of PTSD. The purpose of this paper is to first review the empirical studies and theoretical models that have explored the construct of EN in combat-related PTSD and then to propose an alternative theoretical framework that may have greater explanatory and predictive utility in accounting for the development and maintenance of emotional processing deficits in PTSD. Vietnam combat-related PTSD forms the basis for the present review because the greatest empirical literature exists for this type of PTSD.

EMOTION-RELATED SYMPTOMS OF PTSD

Traumatic life experiences elicit overwhelming negative emotions. Clinicians and researchers from all theoretical perspectives affirm that a defining feature of trauma is the elicitation of these intense emotions, typically horror and fear (see Barlow, 1988; Fairbank & Brown, 1987; Jones & Barlow, 1990). For example, Vietnam veterans' accounts of their experiences in combat often reflect a sense of terror about the threat of annihilation, as well as sorrow and anguish about lost buddies, all of which typically occurred in states of generalized physiological arousal (Card, 1983; Egendorf, Kadushin, Laufer, Rothbart, & Sloan, 1981; Figley, 1978). These veterans also report experiences immediately following trauma that reflect unresponsiveness to stimulation, a lack of emotional involvement in their surroundings, shock, and exhaustion (e.g., Laufer, Yager, Frey-Wouters, & Donnellan, 1981). Veterans' reports of their postcombat adjustment, long after their discharge from the military, sometimes parallel the divergent emotion-related experiences of acute trauma (e.g., a shifting pattern of intense emotionality triggered by reminders of combat trauma as well as emotional response deficits or numbing).

These contrasting experiences of emotional distress and numbness have been formally codified in the diagnostic criteria for PTSD. On the one hand, symptoms that could be readily regarded as manifestations of *hyper*emotionality are specified as likely sequelae of traumatization. These include fear and anxiety responses to trauma-related stimuli (e.g., intrusive painful thoughts of traumatic events or external events that trigger traumatic memories), emotional lability, irritability, and excessive or inappropriate anger (see

Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar, & Weiss, 1988). On the other hand, symptoms of *hypo*emotionality include numbing of general responsiveness or EN, and restricted range of affect. The latter symptoms have also been described as "psychic numbing," or "emotional anesthesia," and are further described as a loss of "the ability to become interested in previously enjoyed activities," or "the ability to feel emotions of any type, especially those associated with intimacy, tenderness, and sexuality" (APA, p. 248).

Symptoms of hyper-emotionality have been systematically investigated in Vietnam combat veterans (e.g., Blanchard, Kolb, & Prins, 1991; Malloy, Fairbank, & Keane, 1983). Empirical work has delineated individual difference variables (e.g., extent of traumatization, depression, etc.) and environmental or internally generated precipitants (e.g., imagery) of fear responses in combat-related PTSD (e.g., Litz, Weathers, Kaloupek, Monaco, Gerardi, & Keane, 1990; Pitman et al., 1987). Clinician and researcher alike can operationally define, reliably measure, and systematically assess the parameters of fear responses to trauma-related stimuli in PTSD. However, the same cannot be claimed for the symptoms of EN. The next section reviews the few empirical studies that have explored EN in combat-related PTSD.

EMPIRICAL EVIDENCE

Clearly from all clinical accounts, dysregulated, deficient, or inhibited capacity to experience emotion and to engage in the interpersonal world utilizing the response flexibility afforded by emotions is part of the core effects of traumatization (e.g., Horowitz, 1986; Keane, Fairbank, Caddell, Zimering, & Bender, 1985). To date, however, no study has *specifically* explored EN or avoidance symptoms in combat-related PTSD. Rather, several case-controlled or epidemiological studies have explored the kinds of symptoms reported, overall, by Vietnam veterans, typically to test the validity of the diagnostic criteria proposed for PTSD (see Keane, Wolfe, & Taylor, 1987). Only within the context of these global research efforts have the EN symptoms of combat-related PTSD been explored (e.g., Card, 1983; Laufer, Frey-Wouters, & Gallops, 1985; Wilson, Smith, & Johnson, 1985).

Early studies exploring emotional processes in Vietnam combat veterans had many methodological problems that have diminished their usefulness in shedding light on numbing phenomena. The methodological limitations include:

(a) *Terminological confusion and definitional specificity problems.* EN has not been operationally defined and terminology has typically led to circular explanations of symptoms. In addition, unspecified terms have denoted emotional dysfunctions. For example, in some studies veterans are asked whether they feel "emotionally numb," while in other investigations combat veterans are asked how often they have felt "emotionally distant from others," or "have difficulty with emotions" (e.g., Frye & Stockton, 1982; Silver & Iacono, 1984).

(b) *Inconsistent data-gathering methods.* Methods of acquiring data on the phenomenology of EN in PTSD have been chiefly limited to static analysis of one or several items on psychological tests (e.g., the Impact of Events Scale: Horowitz, Wilner, & Alverez, 1979; MMPI: Penk, Robinowitz, Roberts, Patterson, Dolan, & Atkins, 1981), or in a few instances, survey questionnaires/symptom checklists (e.g., the Vietnam Veteran Survey: Figley, 1978). Studies are needed that independently assess the parameters of emotional reactivity with multiple methods of inquiry in a controlled laboratory environment. In this context, meaningful data can be gathered about the cognitive, physiological, and behavioral dimensions of emotional processing in PTSD.

(c) *Diagnostic imprecision and inattention to co-morbidity.* Early studies have at times failed

to use valid methods of assessment to reliably diagnose veterans (ideally, assessment would be done through the use of a structured clinical interview, corroborated by psychological testing data, see Litz, Penk, Gerardi, & Keane, 1991). Combat-related PTSD diagnoses have in some instances been derived from chart reviews or by checklists of unknown reliability and validity (e.g., Atkinson, Sparr, Sheff, White, & Fitzsimmons, 1984). In addition, researchers have not attended to the diagnosis of co-morbid Axis-I or Axis-II psychiatric disorders. Co-morbidity is particularly important to consider due to the high rates of depression, as well as drug and alcohol problems in the Vietnam veteran PTSD population (Kulka et al., 1988). The latter two problems would need to be assessed in any study that explored emotional problems in PTSD because of their powerful or destructive effects on interpersonal functioning and emotional processing in PTSD patients (alcohol and drug problems: Helzer, Robins, & Davis, 1975; Keane, Gerardi, Lyons, & Wolfe, 1988; Schnitt & Nocks, 1984; depression: Fairbank, Keane, & Malloy, 1983; Helzer, Robins, Wish, & Hesselbrock, 1979).

(d) *Insufficient or imprecisely defined control groups.* Most studies include non-PTSD Vietnam combat veteran control groups to compare symptom-reporting patterns. Ideally, however, Vietnam combat veteran psychiatric control groups are needed to determine if EN or restricted range of affect is a problem that is specific to the syndrome of PTSD. This is important because preliminary evidence suggests that EN symptoms are highly correlated with the Hamilton Anxiety and Depression Scales in combat veterans diagnosed with PTSD (Davidson, Smith, & Kudler, 1989).

Nonetheless, several studies have attempted to validate the symptoms of PTSD by determining the differential reports of symptoms in Vietnam combat veterans with and without combat-related PTSD, and in so doing have shed some light on the prevalence of EN symptoms of PTSD. Atkinson et al. (1984) compared the PTSD symptom reporting of 20 Vietnam veterans with PTSD and 20 without PTSD in this manner and found that detachment distinguished the groups, but numbing of affect and loss of interest in activities did not. In a similar study, van Kampen, Watson, Tilleskjor, Kucala, and Vassar (1986) attempted to validate the DSM-III criteria for PTSD by gathering self-report data on Vietnam combat veterans in an inpatient substance abuse program. They found that, in general, the diagnostic criteria distinguished the groups well. In terms of EN symptoms, the PTSD patients, relative to substance abusers, reported greater "diminished pleasure"; reports of "problems with emotional expressiveness and intimacy" did not differentiate the groups.

Two factor analytic studies explored the stability of the DSM-III criteria of PTSD and, in so doing, have allowed for an examination of the report of emotional deficits reported by combat veterans. Silver and Iacono (1984) factor analyzed the symptom reporting of Vietnam combat veterans based on a checklist of all possible DSM-III PTSD criteria. They found four factors: depression, guilt/grief, re-experiencing, and detachment/anger. This latter factor included the items: feeling emotionally distant from family and others, and difficulty feeling emotions. These data suggest that emotional unresponsiveness holds up in the symptom reporting of Vietnam combat veterans; however, no data were gathered on actual criterion PTSD diagnoses. Davidson et al. (1989) similarly factor analyzed the diagnostic criteria and found that "constricted" affect loaded with decreased sleep, impaired concentration, and psychogenic amnesia, and this factor appeared to be distinct from an "arousal" and "re-experiencing" factor. However, the factor that included the item "constricted affect" apparently was distinct from a third factor that included items conceptually related to numbing: detachment-estrangement, reduced interest, and avoidance of reminders.

Other symptom validation studies have lumped all possible avoidance or numbing

symptoms as specified in DSM-III and DSM-III-R to a single factor (Category C) and explored the psychometric parameters of this set of interrelated symptoms. The most rigorous and representative of these studies was the National Vietnam Veterans Readjustment Study (NVVRS: Kulka et al., 1988). These researchers found that a large representative sample of extremely well-diagnosed non-treatment-seeking Vietnam combat veterans with PTSD reported an average of 2.8 "numbing-avoidance" symptoms, while combat veterans without PTSD, on the average, reported 0.6 of these symptoms (nearly 70% of these subjects reported no Category C symptoms). It also appears from these data that the avoidance-related symptoms of PTSD are the most commonly reported trauma-related problems of PTSD patients.

Another indirect method of determining the impact of combat trauma on emotional processing is to explore the association of PTSD to social functioning. One would predict that if PTSD is associated with a restricted range of affective functioning that this problem would affect social relationships. Roberts, Penk, Gearing, Robinowitz, Dolan, and Patterson (1982) determined that substance-abusing Vietnam combat veterans with PTSD reported more problems with interpersonal intimacy and sociability as measured by psychological tests. In a similar study, Carroll, Rueger, Foy, and Donahoe (1985) found that treatment-seeking non-substance-abusing Vietnam combat veterans with PTSD reported more problems with self-disclosure and expressiveness to their partners than a matched group of combat veterans without PTSD. However, the two groups did not differ on a measure of intimate and affectionate behavior with their partners. Neither of these studies determined the specificity of EN symptoms to interpersonal problems in Vietnam veterans.

Only two studies have gone beyond questionnaire data to determine the nature of emotional deficits in combat-related PTSD. In one study, Zimering, Caddell, Fairbank, and Keane (1984) compared a group of Vietnam combat veterans with PTSD with a matched group of combat veterans without PTSD on various tasks designed to directly and independently measure various DSM-III PTSD criteria. One measure, the affect recognition task, was designed to assess subjects' ability to correctly appraise the emotion presented from dialogue presented on audio tape. Zimering et al. found that the PTSD subjects had more difficulty appraising and labeling the presented emotional situations than the non-PTSD subjects. They labeled this as a deficit in "interpersonal perceptiveness." Finally, Orr (1991) found that when asked to imagine a pleasant beach scene, Vietnam combat veterans with PTSD did not differ from non-PTSD subjects in terms of their report or expression of emotional experience. The latter was rigorously measured by self-report assessments of pleasantness and intensity of emotional reactions, heart rate and skin conductance, and facial electromyography. This study suggests that *the capacity* to experience non-trauma-related emotions is intact in combat-related PTSD.

Summary

The empirical evidence for a numbing of emotional responsiveness specific to PTSD is inconsistent and methodological problems preclude definitive conclusions from these data. There has been a lack of definitional specificity about EN, inconsistent data-gathering methods, diagnostic imprecision, and inattention to co-morbidity. With the exception of the Zimering et al. (1984) and the Orr (1991) studies, the processes and parameters of non-trauma-related emotional functioning in PTSD have not been examined outside the realm of global and retrospective self-reports. Nevertheless, it appears that some kind of interpersonally oriented emotional deficit does indeed co-vary with trauma history and PTSD status.

Clearly, a great deal remains to be empirically studied about the parameters of emotional processing in PTSD. In part, the scarcity of research focused on emotional processes is due to the tacit acceptance and reification of the construct of EN in the clinical literature. In addition, the available theoretical models of PTSD (reviewed next) have failed to hypothesize *specific* (and at times testable) affective mechanisms or processes operative in PTSD that would be responsible for the emergence of EN problems. The latter has inadvertently impeded the construct validation of EN in PTSD.

THEORETICAL BACKGROUND

In this section, the three most relevant theoretical models that have proposed psychological mechanisms for the development and maintenance of EN symptoms of PTSD will be reviewed subsequent to a brief presentation of the biological model of PTSD that has relevance to EN.

Biological Model of PTSD

The biological model that has received the most attention as a broad-based theory intended to account for the development of PTSD is the infrahuman model of inescapable shock (for a review see van der Kolk, Boyd, & Krystal, 1984; see also Kolb, 1988; van der Kolk, Greenberg, Boyd, & Krystal, 1985). Briefly stated, this model proposes that EN phenomena are caused by: (a) motivational deficits created by the traumatically conditioned effects of learned helplessness (see Flannery, 1987); (b) a depletion of catecholamines produced through conditioned responses to trauma-related stimuli (see van der Kolk & Greenberg, 1987); and (c) a conditioned analgesic effect caused by the release of endogenous opioids during conditioned fear responses that serve to suppress pain and tranquilize the organism (e.g., Pitman, van der Kolk, Orr, & Greenberg, 1990).

The extension of the infrahuman model of inescapable shock to the study of PTSD has appeal because of the vast infrahuman literature that has indeed empirically demonstrated chronic, post-shock conditioned sympathetic hyperreactivity and catecholamine depletion, behavioral constriction, immunosuppression, and conditioned, naloxone-reversible endogenous opioid release (see van der Kolk, 1984). These symptoms, however, have thus far been shown to have the most relevance to conditioned fear responses in PTSD (Pitman et al., 1990). The biological models' attempt to explain the development of EN is incomplete for the following reasons. First, there have been few specific predictions posed about the response parameters of emotional processing in PTSD that emerge from catecholamine hyperreactivity and subsequent depletion or conditioned analgesic responses. These biological phenomena are clearly operative in conditioned responses to fear-related stimuli in PTSD (and other anxiety disorders); however, their relevance for substantiating the extension of the inescapable shock/learned helplessness model and in specifically accounting for EN phenomena (rather than simply fear-arousal) remains to be shown theoretically and empirically. Second, the model has failed to take into account the human extension of the infrahuman models of inescapable shock (learned helplessness) and has similarly failed to address the psychological mechanisms that might mediate or interact with biological events (e.g., appraisals, attributions). The theories that have incorporated psychological constructs in an attempt to account for emotional processes in PTSD are explored next.

Psychological Models of PTSD

Horowitz (1986) has proposed a comprehensive model for the emotional deficits in PTSD, involving both psychodynamic and information-processing components. For Horowitz, trauma creates two opposing sets of internal processes, called intrusion and denial, that are used by the cognitive system to cope with and resolve extreme stressors. The intrusion "phase" of adjustment following trauma entails the hallmark cognitive and emotional symptoms of PTSD: painful re-experiencing and hyperreactivity. Intrusive memory reactivations occur because the organism is motivated by a tendency to completion or closure so that trauma memories can be integrated or accommodated into internal cognitive structures or schemas. Horowitz argues that until a trauma is integrated into a person's schemas it is, in part, present in short-term memory. This drives the periodic, painful, intrusive re-experiencing of traumatic memories: a hallmark symptom of PTSD. Such intrusions, however, trigger an opponent process of ideational and affective denial that represents the defensive phase wards off painful trauma-related affects and memories. This process may be considered numbing in the sense that PTSD patients avoid certain stimuli to minimize memories of the trauma. A traumatized person is said to shift from this numbing phase to an intrusion phase until resolution of the trauma. Resolution, then, entails accommodation of the trauma with the person's internally organized view of the world and the self. Therefore, PTSD reflects an inability to integrate the trauma due to excessive denial that thwarts the necessary emotional processing.

Horowitz's schema model accounts well for patterns of hyper- and hypoemotionality in acute forms of PTSD (Horowitz, Krupnick, Kaltreider, Wilner, Leong, & Marmar, 1981; Horowitz et al., 1980). Although the model has had widespread heuristic value, there have been few satisfactory accounts of the applicability of Horowitz's schema model to the study of chronic forms of combat-related PTSD in the literature. Moreover, the model has several limitations: (a) The model is strictly intrapsychic without recognition of the situational cues that may trigger denial (Foa, Steketee, & Olasov-Rothbaum, 1989); (b) it is difficult to reconcile several of the proposed cognitive mechanisms with well-established information-processing theory (e.g., Litz & Keane, 1989; Wegner, Shortt, Blake, & Page, 1990); and (c) finally, and most importantly, it is not clear from Horowitz's model which emotions are accessible to experience during the intrusive phase versus the numbing phase, nor is it clear whether PTSD patients have the capacity or capability to experience pleasurable, positive emotions.

Keane et al. (1985) developed a conditioning model to explain the development and maintenance of combat-related PTSD. In this model, EN symptoms are conceptualized as avoidance behavior. Escaping from emotional reactions by reducing contact with evocative stimuli (both external and internal in the form of memories, images, etc.) or by suppressing their expression promotes anxiety reduction. This negatively reinforces the escape behavior that, over time, can lead to overlearned and quite rapid emotional avoidance responses. It is these avoidance responses that are reported as "numbness" by PTSD patients.

Chemtob, Roitblat, Hamada, Carlson, and Twentyman (1988), Foa et al. (1989), and Litz and Keane (1989) have all expanded on the Keane et al. (1985) model by incorporating constructs from information-processing theory to explain the cognitive mechanisms that activate PTSD symptomatology. Within these models, trauma-related information is seen as activating a rich network of memories that trigger re-experiencing symptoms and selective attention to threat cues in the environment.

The information-processing models of PTSD have incorporated constructs from the

bio-informational theory of emotion that are relevant to the analysis of EN in PTSD (Lang, 1985). In Lang's model, emotional experiences are encoded in memory in highly organized, semantic networks that contain three related types of information: (a) information about stimulus cues that elicit emotion; (b) information about cognitive, motor, and psychophysiological responses; and (c) information about the meaning of the stimulus cues and responses for the individual (e.g., "I will go crazy unless I escape this situation"). These three elements of emotion are thought to be stored in memory in a way that facilitates integrated cognitive, motor, and physiological responding (Foa & Kozak, 1986; Lang, Bradley, & Cuthbert, 1990; McNally, Kaspi, Riemann, & Zeitlan, 1990).

PTSD patients are characterized according to Lang's model as having an unusually coherent and stable trauma network that requires few matching elements in the environment before the network is activated (e.g., Foa et al., 1989). Weak or degraded trauma-relevant stimuli (e.g., ambiguous threat situations) can lead to trauma network activation in PTSD. In addition, a trauma network can be distinguished from fear networks in other anxiety disorders in that it contains *multiple* emotions. In addition to fear, the trauma network contains memory elements associated with emotions such as sadness, disgust, and rage, each with their own stimulus, response, and meaning elements (e.g., Pitman et al., 1987). Two additional tenets of information-processing theory are relevant to the study of emotion in PTSD. First, there is greater *accessibility of trauma networks*. The network of trauma memories are so broadly generalized across the domain of thoughts, images, feelings, and physiological arousal that they are more readily triggered and accessible to experience than other emotion networks. When the trauma network is activated, other more adaptive responses to interpersonal stimuli are less accessible and thus less likely to influence behavior (e.g., McNally & Litz, 1991). Second, *attention moderates the production of emotional responses*. When the trauma network is activated, a PTSD patient is said to have less attentional resources available to process non-trauma-related stimuli which would trigger other kinds of emotional experiences (Barlow, 1988).

However, a great deal remains to be explained about the long-term emotional deficits in PTSD. Neither Horowitz (1986), Keane et al. (1985), or information-processing variants (e.g., Litz & Keane, 1989) have proposed specific limiting conditions for emotional deficits in PTSD, nor a paradigm that would allow for specific differential predictions. The next section contains a brief description of how human emotion is currently conceptualized, measured, and studied. This is followed by a depiction of a theoretical model that can facilitate the empirical analysis and understanding of emotional processing in PTSD.

THE STUDY OF HUMAN EMOTION

In general, emotion is an integrated response with some degree of physiological, motor, and cognitive activation (Staats & Eifert, 1990). The measurement of emotion should, however, be multimodal, because the response channels are imperfectly correlated and, at times, a lack of correspondence between modes of action of a particular emotional response can be informative regarding potential dysfunction (e.g., Clore, Ortony, & Foss 1987; Evans, 1986). Nevertheless, a fairly specific pattern of psychophysiological responding appears to be associated with specific types of emotions which are automatically activated by specific types of stimuli, with cognitive appraisals of events serving a moderating role (e.g., Cacioppo, Petty, Losch, & Kim, 1986; Ekman, Friesen, & Ancoli, 1980; Smith & Ellsworth, 1985). Some of these automatic emotional responses are "primary," unconditioned, and innate to the extent that they are seen cross-culturally and are seen developmentally very early (e.g., happiness: Ekman & Oster, 1979; Levanthal, 1984).

Also, most investigators agree that emotions are functional or adaptational responses that provide flexibility (and thus survivability) to human behavior (see Plutchik, 1980). As Levanthal and Scherer (1987) suggest, "emotional processes decouple automatic, reflex responses from their eliciting stimuli and provide the opportunity for more adaptive reactions . . . " (p. 7). Depending on the context in which they occur, the specific quality with which events are cognitively appraised, or the specific emotions that are produced (e.g., fear versus joy), emotional responses can serve physiologically mobilizing (e.g., Smith & Ellsworth, 1985), motivational and affiliative (see Lazarus, 1991), and communicative (e.g., facial-expressive-motor responses; Ekman, Levenson, & Friesen, 1983) functions. Also, some researchers have found that there is an inverse relationship between emotional expressiveness and general physiological arousal, which suggests that emotional reactions can have a stress-reducing function in normals (e.g., Notarius & Levenson, 1979).

After many years of neglect, emotions have emerged as integral variables in human information-processing models of cognition and behavior (Lang, 1985; Levanthal, 1984). One model that has received considerable attention in the experimental literature on human emotion, and has applicability to the study of PTSD, is Levanthal's perceptual-motor theory of emotion. Levanthal's theory has been applied empirically to a variety of different clinical areas in behavioral medicine (e.g., Cioffi, in press; Levanthal, Levanthal, Shacham, & Easterling, 1989; Ward, Levanthal, & Love, 1988) and psychopathology (see Greenberg & Safran, 1989; Safran & Segal, 1990). This model is reviewed in detail because it provides the conceptual basis for hypotheses posed in the subsequent section of the paper about emotional processing factors specific to combat-related PTSD.

Perceptual-Motor Theory

Levanthal (1984) has developed an integrative and comprehensive model that defines emotions as the experiential product of a number of different information-processing components that can be reciprocally interactive. In Levanthal's perceptual-motor theory, an emotional response is a synthesis of the activation of three distinct systems or modules: expressive-motor, schematic, and conceptual. This aspect of Levanthal's theory corresponds to current theorizing about the modular nature of human memory (e.g., Johnson, 1983; Schacter, 1987).

The *expressive-motor* level of processing is proposed to consist of a set of innate, unconditioned expressive-motor programs that are activated unconsciously, without intention, by a variety of external stimuli and internal states. Regardless of the state of the organism, external and internal stimuli are evaluated for their novelty, survival value, and pleasantness. The responses elicited by the expressive-motor system are the organism's primary emotional response capabilities and provide the building blocks for further emotional learning. Thus, there always exists a "hard-wired" expressive-motor component to emotional experience. Those emotional responses are always available to experience; however, higher order cognitive processes (see below) can distort the subjective experience of emotion, thwart its manifestation, or distort its meaning (cf. Beck & Emery, 1985).

The *schematic subsystem* is posited to integrate experiences with expressive-motor reactions and consists of conditioned emotional responses and associated emotional memories. The schematic system closely corresponds to the emotion networks proposed by Lang (1985) described above. The activation of a schema[1] links a current stimulus situation

[1]The term "network" and "schema" can be used interchangeably, both are defined as organized associative structures in memory.

with prior knowledge and experiences related to emotional episodes and serves to guide and organize the experience of emotion and emotional behavior. Stimuli that match a schema serve to potentiate or increase the probability of emotional responding. As in Lang's conceptualization, emotion schema can be automatically cued by generalized environmental stimuli, by activation of central neural events (e.g., images), or by expressive behaviors and autonomic responses (e.g., Ekman et al., 1980).

The *conceptual subsystem* represents the highest level of the hierarchically arranged emotional-processing system. It represents the human's capacity to reflect upon experience and draw conclusions about the environment and responses to it (cf. Johnson, 1983). The conceptual system provides default assumptions, expectations, and attributions that are used by the information-processing system in uncertain or unfamiliar situations (cf. Tversky & Kahneman, 1974). Conceptual processing is volitional and effortful, which requires the allocation of attentional resources (see Hasher & Zacks, 1979). It allows for the strategic and voluntary evocation of emotions by calling up schematic memories. It can also inhibit affective reactions to events or augment emotional behavior (Levanthal & Scherer, 1987). Higher order cognitive appraisals or evaluations of stimuli (e.g., in regard to their personal significance and coping potential) are generated by the conceptual system as well (cf. Lazarus & Smith, 1988).

Parameters of Emotional Responding

An emotional response results from an integration of expressive-motor information, schematic activation, and conceptual system activity. The emotional response itself can also provide information that guides and motivates behavior. Indeed, emotional experience is a powerful source of information about what a situation represents for a person. Similarly, the emerging responses provide efferent stimulation that further prime emotional networks or activate inhibitory processes. These, in turn, may enhance or attenuate behavioral output (cf. Zajonc, Murphy, & Inglehart, 1989). Particularly relevant to the study of PTSD is the principle that emotional responses vary in terms of: the involvement of a particular emotion subsystem, intensity, channel(s) of activation (e.g., facial-expressive-motor), how sustained the reaction is over time, and the degree of conscious awareness a person has about the reaction or the eliciting stimuli that prompted the emotional response. These issues suggest that simply asking PTSD patients, for example, to judge overall whether they can feel emotions will prove grossly insufficient as a test of EN deficits. Reliable multiple-response channel, real-time measurements that capture responses to a variety of evocative stimuli with particular attention to the role of response automaticity are required in order for advances to be made in the construct validation of EN in PTSD.

Levanthal's model has recently been used by clinical researchers to operationally define abnormal aspects of emotional functioning (e.g., Cioffi, 1991; Greenberg & Safran, 1989). Broadly defined, an emotional dysfunction is said to exist when: (a) there are few emotional response options available; (b) when certain emotion networks are probabilistically less likely to be accessed because of the concurrent activation of other incompatible networks; or (c) when an emergent emotional response is thwarted, inhibited, or blocked by environmental events or through top-down conceptual injunction. Each of these types of dysfunctions suggest specific deficits in emotional processing that can serve as targets in treatment (e.g., Greenberg & Safran, 1989). The specific aspects of emotional-processing deficits in PTSD are outlined next. These are offered as models in order to stimulate further research in this area. As outlined above, since very little empirical data exists in this area, the proposals below are hypothetical in nature and will require extensive empirical validation.

EMOTIONAL DEFICITS IN PTSD

Expressive–Motor Programs

These innate emotion programs are unaffected by stress, trauma, or PTSD. Thus, the capacity or capability to experience and express a variety of emotions is *intact* in PTSD. However, higher order, executive functions produced by the conceptual system can inhibit the production of full-blown responses. An example may be instructive: A married Vietnam veteran with chronic combat-related PTSD has a baby who smiles broadly at him. The expressive–motor (facial) program to respond with a smile, in kind, is *as available* to the veteran as before his trauma. In a nonsymptomatic state (i.e., no cued re-experiencing or reactivity) he will spontaneously and automatically respond with a smile if he sufficiently attends to the baby's face. The likelihood of a *sustained* automatic expressive–motor response is *acutely* decreased, however, if the patient is symptomatic because of the interference created by trauma network (schema) activation or the concurrent activation of conceptual processes (outlined below) that contravene such emotional reactions (cf. Chemtob et al., 1988).

Schematic Processes

As previously outlined, conditioned emotional responses to trauma-related stimuli are preeminent in the information-processing system in PTSD. When the trauma network is activated it lessens the likelihood of the activation of more adaptive, alternative emotion networks with their attendant conditioned emotional reactions. Non–trauma-related emotion networks are intact in PTSD, for example, the schema about "children who smile at me," yet they are made less accessible in symptomatic states. Using the example above, the veteran may have difficulty engaging positively with his child because it is difficult to access the autonomic, expressive–motor, and verbal–semantic programs for such engagement, particularly during symptomatic states. He may require more of an intense, sustained, and unambiguous affiliative response from his child in order to access his own schemas of affiliative emotional behaviors. Thus, a wide range of emotion networks are intact (and available to experience) but inhibited or made less accessible by trauma-related cognitive processes.

In addition to potentiating fear and other intense emotions that interfere in emotional responses to positive events, the trauma network can itself prompt reactions that may create the experience of "numbness." Although speculative, this may be due to original traumatic conditioning experiences that entailed a sense of being stunned, fatigued, or numbed to further stimulation.

Conceptually Driven Deficits

It is hypothesized that due to changes in fundamental assumptions about safety and predictability which are induced through trauma, as well as subsequent repeated painful interpersonal experiences (e.g., Carroll et al., 1985; Janoff-Bulman, 1989), PTSD patients develop, over time, a conceptual system that is inflexible and maladaptive. The conceptual emotion system of combat veterans with PTSD drives the top-down processes that often attenuate or inhibit the sustaining of expressive–motor and schematic reactions to positive events and augment or embellish responses to negatively valenced stimuli, particularly in symptomatic states. Furthermore, there may be an expectation that negative feelings are derived from interpersonal events, or that one's positive responding is not worth the effort because of an expectation of noncontingency, or noncontrol (Chemtob et

al., 1988; Foa et al., 1989). PTSD patients are also likely to develop an organized conceptual system that diminishes emotional responding by injunctions that reflect beliefs that *any* form of arousal is to be avoided because it will lead to trauma network activation.

The rigid conceptual rules that govern emotional behavior in combat-related PTSD can also directly arise from strategies originally learned in combat. This style of action has been characterized as the "combat-mode," which entails a hypervigilance that had obvious survival value and a series of strategies that served to reduce intimacy with fellow soldiers in order to lessen the impact of potential losses (see Chemtob et al., 1988). These formerly adaptive strategies are difficult to extinguish and have profound effects on emotional processing post-trauma. The combat-mode entails strategies to avoid intimacy and affect. These avoidance strategies might entail self-statements that thwart sustained emotional reactions, particularly to interpersonal events (e.g., "don't let your guard down," "don't get too close," "don't show your feelings").

Differential Emotional Processing Deficits in PTSD

It is hypothesized that positively valenced emotional responses, in symptomatic or activated states, are reduced in strength in patients with PTSD. A positive emotional response may only be discreetly manifested (e.g., a father with PTSD may smile only briefly in response to his child). When positively valenced emotional reactions are inaccessible to experience (or muted), they fail to provide sufficient efferent feedback that would otherwise enhance emotional experience, and thus exacerbate emotional deficits in PTSD. Conversely, when the network of trauma memories is activated in PTSD, there is an enhancement of reactions to negative events. This selective or differential emotional response deficit is proposed as a theory-based operational definition of EN in PTSD: one that can explain the clinical observation of restricted range of affect as well as the intense emotionality endorsed by combat veterans with PTSD.

FUTURE RESEARCH DIRECTIONS

A great deal of research remains to be done to more clearly delineate the parameters of emotional processes in PTSD. The variant of Levanthal's model that is proposed can allow for a clearer delineation of the specific environmental, cognitive, and response dimensions of emotion that may be deficient in PTSD and poses specific empirical questions that should guide research in this area. A few guidelines for future research are proposed next, as well as some potential areas of inquiry.

Since emotions are a multiply determined, multiply expressed facet of human functioning, future research will need to systematically assess a variety of stimulus and response aspects of affective functioning in traumatized populations. Future research will need to use a variety of methods of stimulus presentation (e.g., slides depicting a variety of emotionally valenced events, conversations with confederates, etc.) and modes of stimulation (e.g., auditory, visual, etc.) in a variety of social contexts (both in vitro and in vivo) to validly capture the parameters of emotional deficits in PTSD. In regard to the response side of the equation, it will prove fruitful for future research to measure various aspects of emotional experience: self-report (e.g., likert-type ratings on a variety of emotional dimensions; see Lang, 1985), autonomic nervous system activity (e.g., skin conductance, heart rate; see Notarius & Levenson, 1979), and facial-expressive-motor activity (e.g., using facial electromyography or the coding and rating of facial activity; see Ekman et al., 1983). For example, since the facial-expressive-motor component of emotion is hypothesized to be relatively intact in PTSD (especially in nonsymptomatic states, see below), the measurement of facial electromyography in key areas that reflect positively

and negatively valenced responding (e.g., the Zygomatic Major and Corrugator muscles) could be used as an index that when used in conjunction with self-report could assess emotional responsivity to a variety of stimuli in the laboratory.

The model proposed in the present paper suggests that specific emotional response deficits are probabilistically more likely to occur when PTSD patients are re-experiencing trauma memories and/or during hyperaroused states (i.e., when their trauma networks are activated). Thus, future research should manipulate extent of network activation in PTSD subjects (and controls) by using a psychological priming procedure (e.g., showing combat veterans slides of combat) prior to directly measuring emotional responsivity as outlined above.

CONCLUSIONS

It has been proposed in the present paper that emotional numbing in PTSD is a complex, multiply determined problem that is best characterized as a selective emotional-processing deficit. This emotional deficit is chiefly manifested during symptomatic states (and is thus episodic in nature) and entails a muting of positively valenced responses and a heightened reactivity to negative events. The capability to respond emotionally to a broad range of stimuli is hypothesized to be intact but relatively inaccessible because of: (a) the activation of trauma-related cognitive processes that are antagonistic to the expression of non-trauma-related emotions; or (b) the activation of top-down rules that contravene sustained, positive emotional reactions.

Emotional deficits in PTSD have been found to be particularly resistant to treatment (e.g., Keane, Fairbank, Caddell, & Zimering, 1989). If the proposed model is correct, however, specific treatments can be used to train patients to access and sustain expressive-motor emotional behaviors and to change rigid conceptual rules about feelings. Interventions of this type have been extensively outlined in the clinical literature (e.g., Beck & Emery, 1985; Greenberg & Safran, 1989; Safran & Segal, 1990).

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