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THEORETICAL AND REVIEW ARTICLES

A Cognitive Action Theory of Post-Traumatic Stress Disorder

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Abstract—Post-Traumatic Stress Disorder (PTSD) is an anxiety disorder characterized in part by such phenomena as intrusive memories, flashbacks, numbing of affect, memory and attentional impairment, hyperalertness, and adjustment difficulties. In this paper we describe a new theoretical approach to understanding PTSD in combat veterans that has implications for understanding PTSD resulting from other life-threatening situations, and for understanding other anxiety disorders and ordinary cognitive processes. Previous approaches to understanding PTSD have derived from associative learning theory, from a reinterpretation of psychodynamic theory, or from general notions of information processing. The perspective presented in this paper integrates these previous approaches and elaborates them into a hierarchical network view of cognition and action. According to this view, emotion, action, and memory all flow from the processing of information by specific mental network structures. The symptoms of PTSD are derived from structures that were acquired during combat because they were then adaptive in promoting the soldier's survival, but now lead to actions that are inappropriate for the civilian environment. The presence of these structures continues to produce difficulties for some individuals. Such occurs because they are particularly susceptible to a vicious-cycle positive feedback loop in which mild evidence of threat activates threat-response structures that bias the individual to interpret ambiguous evidence as threatening. This, then, further raises the threat arousal, further activates the threat-response structures, and so forth. The implications of this view for the treatment of PTSD and other anxiety disorders are discussed.

Conservative estimates are that 16.6% of the approximately 2.5 million Vietnam veterans and 29.6% of Vietnam combat veterans have significant problems adjusting to civilian life (Egendorf, Kadushin, Laufer, Roth-

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bart, & Sloan, 1981; see also Wilson, 1980). Hence, treatment of these veterans with psychological problems presents a major health-delivery challenge of national proportions. Although there has been a serious attempt to meet the needs of these veterans (e.g., Blank, 1982), there remains great need for conceptual models, assessment instruments, and treatment approaches developed for and validated on Vietnam veterans (Keane, Fairbank, Caddell, Zimering, & Bender, 1985).

There is considerable agreement that Vietnam era veterans seeking psychological help at Veterans Administration Medical Centers are more disturbed than are the combatants of other wars seeking help at the same centers (Egendorf, Kadushin, Laufer, Rothbart, & Sloan, 1981; Keane & Fairbank, 1983; Keane, Fairbank, Caddell, Zimering, & Bender, 1985). Although the problems for which they seek help span the entire range of diagnoses in the American Psychiatric Association's (1980) Diagnostic and Statistical Manual (DSM-III), the most prevalent diagnosis appears to be post-traumatic stress disorder (PTSD: Keane et al., 1985), often accompanied by other problems including substance abuse (Boscarino, 1979, 1981; Keane, Caddell, Martin, Zimering, & Fairbank, 1983; O'Brien, Nace, & Mintz, 1980), aggression or fear of aggression (Silver & Iacono, 1984), and marital problems (Egendorf et al., 1981; Carroll, Rueger, Foy, & Donahue, 1985).

There is a clear clinical need to understand PTSD, both to devise effective treatments and to gain some insight into its prevention. Current conceptually-based research investigating PTSD and its treatments has concentrated on three lines of development. One line derives from associative learning theory, another from modifications of psychodynamic theory, and the third from a general information-processing perspective. We review this research and propose a fourth approach that synthesizes lines of development and extends the earlier conceptualizations of PTSD. The proposed cognitive action theory of PTSD also aids in the description of the cognitive organization of people who have been faced with severely traumatic life-threatening events and delineates the components of an emergency response system and the regulation of that system. Finally, the theory has applications in the description of other anxiety disorders as well.

Beyond their clinical contributions, investigations of PTSD also offer an opportunity to study processes at the basis of cognition and emotion. The power of the phenomena comprising the PTSD experience provide one clear "model" system in which to study how people acquire cognitive organizations and use those organizations in the control of their daily lives.

Although PTSD can result from many different kinds of serious life events, including rape (Burgess & Holmstrum, 1974; Katz & Mazur, 1979), natural disasters such as floods (Erikson, 1976), personal loss (Lindemann, 1944), and accidents (Leopold & Dillon, 1966), in this paper we are primarily concerned with the sequelae to combat experience. We believe that many of the principles developed to describe PTSD in combat veterans are applicable to other etiologies as well.

CHARACTERISTICS OF PTSD AND ITS ATTENDANT PROBLEMS

DSM-III

Formal descriptions of PTSD are provided in DSM-III. The DSM-III diagnostic criteria for PTSD include (a) reexperiencing of the traumatic event through intrusive memories, nightmares, and flashbacks, (b) inhibitory cognitive processes such as "numbing" of affect, interpersonal isolation, and withdrawal, (c) memory impairment or difficulty concentrating, disturbances of memory and concentration, and (d) hyperalertness or an exaggerated startle response. Additionally, the DSM-III criteria indicate that patients with PTSD are psychophysiologically hyperarousable, and that they show an intensification of symptoms following exposure to events associated with the trauma.

Combat provides a clear example of one of the DSM-III criteria for diagnosing PTSD—specifically, combat provides an example of "a recognizable stressor that would evoke significant symptoms of distress in almost everyone." In fact, intensity of combat experience has been found to be the most reliable predictor of PTSD in combat veterans (Egendorf et al., 1981; Foy, Sipprelle, Rueger, & Carroll, 1984; Frye & Stockton, 1982; Penk, Robinowitz, Roberts, Patterson, Dolan, & Atkins, 1981; Worthington, 1977).

Immediate stress reactions are common among combat soldiers. These often take the form of heightened startle responses and nightmares or dissociative symptoms, such as emotional numbing (West & Coburn, 1984). These symptoms frequently disappear without any treatment (Ewalt & Crawford, 1981). Of greater clinical and practical importance are the delayed and chronic forms of PTSD, which may appear in Vietnam veterans following symptom-free periods lasting weeks, months, or even years. This paper is primarily concerned with these longer-lasting forms of PTSD.[1]

Phenomenological characteristics of PTSD

Perhaps more informative for understanding PTSD are the phenomenological reports of Vietnam combat veterans with chronic PTSD. Chief among distressing phenomena they report are recurring episodes of intrusive morbid and anxiety-filled thoughts and memories (e.g., Horowitz, 1974, 1976), nightmares and combat dreams that are so powerful they lead to avoidance sleep disorders (i.e. individuals have difficulty sleeping

[1] We also recognize that there may be other components to PTSD, including affective disorders (e.g., depression, survival guilt) and certain secondary disorders (e.g., personality disorders) that developed as subsequent adaptations to the disorder. The presence of these additional components is consistent with the kind of theory we describe. For ease of exposition we have concentrated on the components related more directly to threat response. The depressive aspects of PTSD will be addressed in a subsequent paper.

or attempt to avoid sleep in order to avoid the distressing dreams), and flashbacks that are so vivid that they preempt ordinary experience in favor of reexperiencing previous traumatic events. Hyperalertness, excessive startle responses, irritability, and explosively aggressive impulses are also frequent complaints. PTSD sufferers also report a number of less dramatic cognitive deficits. Many veterans report a feeling of "discontinuity" between their experience during these disruptive episodes (for lack of a better term we will call these PTSD episodes) and their ordinary experience. They characterize their experience during a PTSD episode as involving a separate mode of thinking: They behave more impulsively, are less attentive to detail (e.g., to facial expressions), and are less controlled by external stimuli and events. They report an inability to remember or think about information not relevant to their intrusive memory and an enhanced tendency to recall Vietnam-related material from memory. They show evidence of abnormal muscle tension and increased startle responses. They seem to have lower tolerance for stress. They tend to overreact to mild stressors, are very sensitive to putative threats, and are easily provoked to fight (Friedman, 1981; Horowitz, 1976; Zimering, 1984).

The cognitive features characteristic of chronic PTSD sufferers appear to be derivative of a special "survival mode" of functioning. Many of these characteristics were probably adaptive in combat situations. They aided the combat soldier to react more quickly to threats of danger, and hence to survive it. Narrow focusing of attention and concentration on potential signs of danger probably increased the soldier's sensitivity to threat signals, making them easier to detect. Increased physiological arousal probably speeded the soldier's response to danger once it was detected. Increased psychological arousal also suggests a state of sensitization (see Groves & Thompson, 1970), which itself tends to increase responsiveness. Presumably, this survival mode and its attendant behavioral repertoire were activated whenever the soldier perceived himself to be in a dangerous situation. This combination of obvious life-threatening danger, increased arousal, and focused concentration and attention promoted the soldier's survival and, in some instances, produced "permanent" changes in the individual's information processing system that continue to affect behavior in civilian situations.

Whereas this survival mode was adaptive in combat, its persistence in civilian situations interferes with the veteran's functioning because the relatively innocuous threats that activate it in civilian life are not handled appropriately by the same behavior patterns that were effective in combat and because once activated, it preempts the "normal" task-related cognitive processes that are more suited to civilian life. The cognitive action theory that we describe below attributes this survival mode to high levels of self-potentiating threat-arousal, confirmation bias, inhibition of alternative modes of information processing, and narrow attentional focusing. These are each described in detail later in the paper.

THEORETICAL APPROACHES TO PTSD

Learning theory approach to PTSD

A number of approaches have evolved that seek to understand the nature of chronic PTSD. One of the earliest and most successful of these sees PTSD and other anxiety disorders as the result of classical conditioning. This approach has led to a number of successful treatment regimens, and, as a result, bears much surface validity as an explanation of anxiety disorders. This approach has its origins in Mowrer's (1947, 1960; see also Levis & Boyd, 1979; Mineka, 1979) two-factor learning theory (Keane et al., 1985). Mowrer argued that cues present during a traumatic episode serve as conditional stimuli (CSs) and the unpleasant traumatic events function as unconditional stimuli (USs). These cues then come to elicit conditional responses (CRs) similar to the unconditional responses (URs), such as fear and anxiety, elicited by the traumatic episode. He further argued that the conditioned fear then serves as a drive for the performance of instrumental responses that reduce it. Removal of the fear-eliciting CS reduces fear, and can, therefore, function as a negative reinforcer strengthening instrumental avoidance behaviors. Fearful stimuli elicit anxiety and anxiety responses. Individuals then perform instrumental responses that are reinforced by the reduction in anxiety. Such conditioning can occur without the individual's awareness (Verplanck, 1955).

Soldiers in a combat situation may have reduced their anxiety, and thereby promoted their survival (Frye & Stockton, 1982) by engaging in such actions as returning fire and assault and such cognitive responses as denial, psychic numbing (West & Coburn, 1984), and repression. As a result, these responses became conditioned. Through generalization, higher-order conditioning, and as the result of ordinary life stresses, stimuli in the civilian environment also come to elicit anxiety in the veterans. This anxiety is not easily differentiated from the anxiety that occurred under more stressful conditions. The same behaviors that were effective in ameliorating the combat anxiety are, therefore, activated in an attempt to reduce the civilian anxiety. Because the civilian environment is so different from the combat environment, however, actions that formerly were effective in reducing anxiety are frequently inappropriate to deal with these sources of tension. Although these inappropriate behaviors frequently lead to difficulties for the veteran in the civilian environment, they persist because they continue to affect, though perhaps weakly, the individual's anxiety level (Annau & Kamin, 1961; Solomon, Kamin, & Wynne, 1953; Solomon & Wynne, 1954). As long as the response is effective in reducing the anxiety, it will continue to be reinforced and will not extinguish.

Whereas the parallel between the acquisition of fears in humans and classical conditioning in animals is apt enough, learning theory has

changed substantially since Mowrer conceived his theory (see Bolles, 1967; Dickinson, 1980; Mackintosh, 1983; Roitblat, 1987; Wagner, 1981). Mowrer's interpretation of fear conditioning appears to be overly simplistic even when dealing with animal avoidance learning (see, e.g., Schwartz, 1984). Furthermore, many of the predictions of two factor theory are not borne out in the conditioning laboratory. For example, two-factor theory claims that avoidance behavior depends on the reduction of fear or anxiety, but direct evidence for the presence of fear has not always been observed (see Rescorla & Solomon, 1967). Sometimes when fear responses do occur they happen at the wrong time for their reduction to act as instrumental reinforcers. Furthermore, dogs who have been successfully avoiding a shock for some time show no behavioral evidence of fear at all. Instead, they appear very relaxed and nonchalant in performance of their avoidance response (Solomon & Wynne, 1954). As a result, it is difficult to argue that reduction of fear or anxiety is the mechanism that maintains avoidance behavior. The data do not deny that classical conditioning plays a role in avoidance conditioning, only that its role is more complex than that proposed by Mowrer.

More recent theories of avoidance learning (e.g., Irwin, 1971; Seligman & Johnston, 1973) seek to explain avoidance conditioning through the operation of expectancies. The animal expects that performance of the avoidance response will result in the omission of shock. Confirmation of that expectancy, rather than reduction of fear responses, reinforces the behavior. The cognitive action view, described below, continues the development of these cognitive models of avoidance learning by including many of the recent advances in the analysis of learning in humans (see, e.g., Anderson, 1983; Glass & Holyoak, 1986) and animals (e.g., Roitblat, 1987).

Psychodynamic theories

Another approach to PTSD has its roots in a reinterpretation of psychodynamic concepts and their integration with current conceptualizations in cognitive psychology. For example, Horowitz (1976) attributes the dysfunction characteristic of PTSD (e.g., the emotional numbing, hyperarousability, and narrow focusing of attention) to conflicting demands on the individual. One demand is to assimilate threat-related information into existing cognitive schemata. The other demand is to reduce arousal. Cognitive schemata are representational frames or organizational structures that contain information about recurring (or generic) features of events and concepts. The demand to assimilate threat information and the demand to escape it are incompatible. Attempts to assimilate threat-related information require exposure to the stressful information, thereby causing an increase in arousal, and an increase in the drive to escape from that arousal. The presence of unassimilated stress information, however, also activates a "drive for completion or mastery" (Horowitz

& Becker, 1971) similar to that found by Zeigarnik (1927/1938)—people who are interrupted prior to finishing a task have a tendency to resume its performance later in preference to other tasks and also show an enhanced memory for the incompleted task. Although the Zeigarnik effect refers to the resumption of voluntary activities and memory, Horowitz argues that the same process drives individuals to resume interrupted assimilation of stress information, for example, through involuntary repetitions in thought.

Specifically, Horowitz's model is based on negative feedback. High levels of stress arousal activate compensatory inhibitory processes that reduce the arousal, but this reduction comes at the expense of also inhibiting the assimilation of the stress information. These compensatory mechanisms thus reduce stress, but increase the drive for completion and the tendency to produce aversive intrusive thoughts. The individual trades a short-term decrease in arousal for a long-term perseveration of the factors eliciting the arousal. Clinically, the PTSD sufferer is observed to oscillate between stages of hyperarousal and activity and states of emotional numbing and ideational avoidance.

Although the Horowitz model is interesting and largely consistent with available data, it places heavy emphasis on a "drive for completion," for which there is little independent evidence. Some of its other difficulties will be described below after we have described our own approach. Horowitz's model does, however, serve to highlight the importance of understanding stress events in terms of the individual's preexistent cognitive schemata and representational structures. As Horowitz (1980) notes, "Phenomenological description of stress response syndromes has been difficult because psychological reactions always combine response keyed to recent serious life event with previous inner models of the self and the world" (p. 85).

Cognitive theories

Building on the theme that stress-response must be interpreted in light of interactions between preexistent cognitive structures and life experiences, Beck and Emery (1985) describe a model of threat reaction. Although their model is intended to deal primarily with responses to actual and current threats, features of the model are relevant for understanding chronic PTSD. They propose that a person's response to a threat-filled situation depends on an appraisal of the potential for harm in the situation and the person's resources for avoiding or escaping from that harm. This appraisal involves the activation of a cognitive schema. Activation of a schema leads the individual to attend to evidence consistent with the schema and to ignore evidence that is inconsistent. In short, people tend to see what they expect to see in a situation. Unless subsequent evidence is strongly inconsistent with the expectancy, individuals tend to maintain the expectations derived from their initial assessment of the situation and

their schema for it. The final appraisal of the situation results from an interaction among the evidence provided by the situation, the person's preexistent cognitive set—the composite of expectations, interests, and concerns—and the resources available to deal with the threat.

Beck and Emery (1985) view this process of threat reaction as an outgrowth of ordinary cognitive processes. Ordinarily people select from among all incoming sources of information those most appropriate to current needs and goals. For example, a hungry person will differentially process food-related information at the expense of other information. Similarly, if a person perceives a threat, a threat schema may be activated that would selectively process information relevant to the threat at the expense of other kinds of information. As a result, detection of even ambiguous information about the presence of a threat serves to focus the individual's attention on obtaining further evidence regarding threat. Perception of threat also activates in a reflex-like manner cognitive and motor responses, such as freezing. These responses are part of the person's normal biological response to threat (see, Bolles, 1970, 1972; Bolles & Fanselow, 1980).

We can summarize the aspects of Beck and Emery's approach that are most relevant to the present discussion: Individuals come to situations with preexistent mental schemata. These schemata contain important information about the individual's past experience in similar situations, rules, beliefs, assumptions, and expectancies regarding likely future events. Activation of a schema causes the individual to seek information consistent with the schema, to interpret ambiguous data as consistent with the schema, and to ignore other information.

Lang (1979) proposed a related cognitive anxiety theory. On the basis of psychophysiological evidence, he noted that specific patterns of muscle activity are associated with the type and content of an imagined object, scene, or activity (e.g., Deckert, 1964) and that the content of images can powerfully affect subjects' physiological responses in ways that are typically associated with emotional responding (Lang, Melamed, & Hart, 1970; Marks & Hudson, 1973). Lang interpreted these data as evidence that perceptual response information is encoded along with information about the stimuli being imaged in an abstract propositional network (e.g., Anderson, 1980). This network includes stimulus labels, related semantic information, perceptual response elements, and the motor program of affective expression. Individuals with anxiety disorders suffer from maladaptive networks that contain inappropriate semantic information (e.g., overestimates of danger or frequency of a feared object) and inappropriate response information (e.g., inappropriate physiological responses and avoidance behaviors).

To summarize Lang's theory, emotional stimuli are represented by the individual in propositional networks (called fear structures by Foa & Kozak, 1986). These networks contain information about the semantic content of the stimuli, about their imaginal properties, about their valences (e.g., that they are unpleasant or dangerous), and about the be-

haviors that the person will perform in response to the stimuli. These responses include verbal behavior—expressive vocalizations or reports of feelings, behavioral acts such as avoidance, coping responses, and performance deficits, and somato-visceral responses. Foa and Kozak (1986) view these networks as programs for escape or avoidance.

A HIERARCHICAL COGNITIVE ACTION THEORY

Basic theoretical assumptions

The present model continues the development of cognitive schema theories. In this section we present the theoretical groundwork for the theory. The basis for our present proposal includes an assumption that emotion, action, cognition, and memory all flow from the processing of information by specific mental network structures (Roitblat, 1987). We further assume that at least some psychological disorders result either from failures of these structures to operate appropriately or from the correct action of inappropriate structures (e.g., those containing inappropriate beliefs or intentions). This view is related to those proposed by Horowitz (e.g., 1976, 1980), Beck and Emery (1985), Lang (1979) and Foa and Kozak (1986), and extends these earlier approaches. The structures we propose are also in the spirit of the hierarchical action structures described by Gallistel (1980; see also Norman, 1981) and the recent developments in learning theory (e.g., Dickinson, 1980; Mackintosh, 1983; Roitblat, 1987; additional details on cognitive action theory can be found in Roitblat, 1987, in press).

Recent theoretical developments in cognitive psychology view the mind as a real-time, self-organizing network[2] (e.g., Eich, 1985; Grossberg & Stone, 1986; Hinton & Anderson, 1981; McClelland & Rumelhart, 1986; Oden, 1987). According to this model, information processing occurs in the brain by transmitting simple signals through complex assemblages of relatively simple processing units. These structures are parallel distributed information processing networks. It is important to note that these structures, themselves, process the information; they are not read or interpreted by some other mechanism. They are parallel in that information can be processed by more than one part of the network at a time. They are distributed in that an action, idea, cognition, etc., is not represented at any one point in the network, but is represented by the state or structure of the network at many points. Further, these networks are physically realizable, given what we currently know about the brain and the properties of neurons. To be sure, our descriptions of these networks are greatly simplified (we do not, for example, attempt to describe

[2] The networks are real-time in that they process information as it comes in. They are self-organizing in that their structure is derived from experience, no designer (other than evolution and experience) controls their structure.

all of the parallelism and distributedness necessarily inherent in the actual brain mechanisms implementing these networks), but the principles we (and other authors) do describe are generalizable to more realistically complicated networks (see McClelland & Rumelhart, 1986, for a discussion of these networks at a more molecular level).

The schematic information processing networks underlying our model consist of hierarchically arranged lattices of interconnected nodal elements (Gallistel, 1980) or "nodes." An example of this kind of network is shown in Figure 1. Levels in the hierarchy correspond roughly to levels of abstraction. Nodes at the lowest level directly control patterns of muscular and neuroendocrine activity. Nodes at somewhat higher levels represent abstractions of these actions. Nodes at the highest levels represent intentions, thoughts, expectancies, goals, and motivations. For example, a node at one level may represent the action of raising a fork from a plate. A node at this level does not specify which of the various muscles will move or the specific pattern in which they will move,[3] these variables are specified by lower level nodes that are influenced by this node. Nodes at a still higher level may represent the action of getting food from a plate.

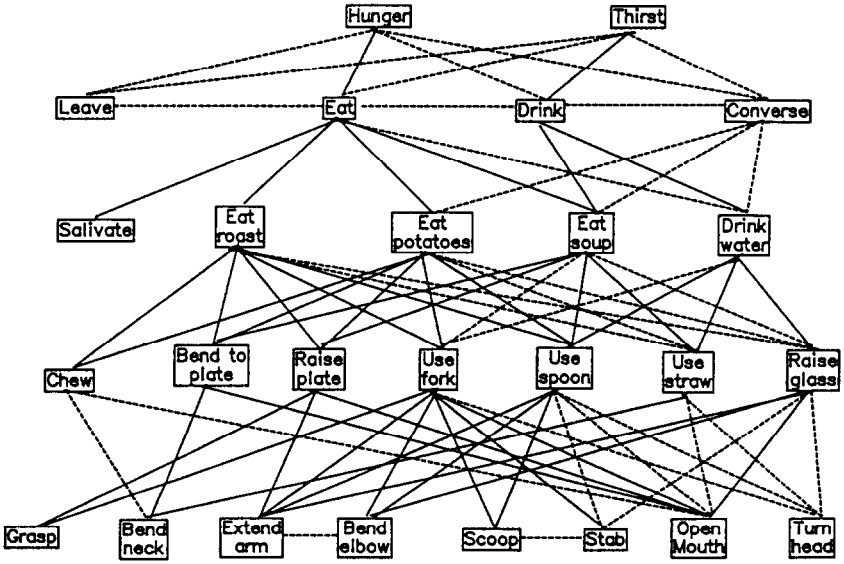

FIG. 1. A schematic sketch of a portion of a hierarchical cognitive action network for eating. We do not intend this network to represent a serious analysis of eating dinner. Rather, this figure is intended to illustrate the *kinds* of relationships that can exist among hierarchically organized units. Boxes represent nodes in the network. Solid links represent potentiating connections and broken lines represent inhibitory connections.

[3] These variables are controlled by such factors as the weight, size, and viscosity of the food. See McClelland, Rumelhart, and Hinton (1986).

There are many ways that food can be moved from plate to lips, including using a fork, using a spoon, lifting the plate, and so forth. Again, the particulars are left to the lower levels. A node at a still higher level might represent a motivation for food. When this node is active it may influence a whole host of lower level nodes including, perhaps, one that represents going to a restaurant, one that represents preparing dinner, or one that represents moving toward the kitchen.

Nodes interact with one another by transmitting potentiation and inhibition. When a node is activated it potentiates one set of lower level nodes and inhibits others. Typically, each of the potentiated lower-level nodes represents an alternative way of accomplishing the action specified by the higher level node. Potentiation makes a unit easier to activate and inhibition makes a unit more difficult to activate. In addition to potentiation and inhibition, nodes at practically every level are also influenced by the stimulus inputs they receive. Nodal activation, thus, is controlled by the (nonlinear) combination of the potentiation or inhibition the node receives from other units and the stimuli it receives from the environment. Each unit has its own "rules of evidence," or specification of the stimulus properties that will activate it. Potentiation lowers the unit's threshold for activation, so that weaker evidence is capable of activating the node and inhibition raises its threshold, so that stronger evidence is necessary to activate the unit. Environmental events (including those in the internal milieu) serve to select among the potentiated nodes. To return to the example of eating food from a plate described above, activation of the node representing eating potentiates a number of different actions, including using a fork, using a spoon, cutting the food, and so forth. Each of these actions are alternative components of eating. The particular action that will be activated depends on a number of factors, including the viscosity of the food (whether a spoon or fork is necessary), its size (whether it must be cut), its location, etc.

In addition to potentiating or inhibiting lower level nodes, activation of a node also inhibits alternative nodes that represent incompatible actions. For example, when a low-level node representing arm flexion is activated, it inhibits nodes representing arm extension. Similarly, when a node representing food-seeking is activated, it inhibits other activities that are inconsistent with food seeking. This mutual inhibition pattern helps to prevent the organism from dithering—e.g., trying simultaneously to eat two stacks of food, one on either side, and as a result, being caught in the middle out of reach of both. The same kind of lateral inhibition occurs at all levels in the hierarchy.

Another major characteristic of these network systems is that thoughts, images, actions, emotions, and behaviors are all represented in the same kind of network. There is no principled way to distinguish cognitive from noncognitive parts of the organism's information-processing system (Roitblat, 1982, 1986, 1987). All are the results of complex networks of neurons and their activity. Nodes that represent what we conventionally think of as thoughts and intentions connect via the same

kinds of links with nodes that we typically think of as representing specific muscular movements.

Learning in this model occurs at all levels in the network through two mechanisms (Roitblat, 1987, in press). Learning results in the formation of new nodes or in the formation of new connections among existing nodes (see also Hayes-Roth, 1977, for further discussion regarding the formation of new nodes). This learning can occur at any level in the hierarchy, and some evidence exists that there is a bias for learning to be represented at the highest appropriate level (Bever, 1984).

We can speculate that extinction weakens the connections between nodes. Recovery following extinction, whether spontaneous or the result of retraining, can occur relatively quickly because the nodes and their connections remain in memory, and need only to be strengthened in order to control behavior. We can also speculate that counter-conditioning forms new nodes and connections, which then serve to inhibit the previously existent nodes, through the principle of lateral inhibition of incompatible nodes.

One other feature of the network view that must be highlighted is that expectancies result not only in the potentiation of appropriate learned behaviors, but also in the potentiation of other "unlearned" behaviors or physiological responses. For example, food expectancies result not only in the potentiation of food-getting behaviors, but also in such physiological responses as salivation (Gallistel, 1980; Shettleworth, 1975). At this level too, activation of one response inhibits alternative incompatible responses.

PTSD conceptual network

The kind of network shown in Figure 1 can be applied to PTSD as follows: At the highest level of the network shown in Figure 2 is one node representing threat arousal and a mutually inhibitory node representing work-related motivations and arousals. This is obviously a highly schematic representation of the network. Many other kinds of motivations, intentions, and arousals, too numerous to include in the figure, also obviously would be present. Also not shown in Figure 2, are the complex semantic networks that would presumably be employed in the interpretation of incoming stimuli.

For individuals with PTSD, the threat arousal is presumably always at least weakly potentiated. Such arousal is ordinarily kept in check, however, by the lateral inhibition from alternative incompatible active nodes. The detection of some perhaps ambiguous evidence for a possible threat can cause a catastrophic (in the sense of catastrophe theory, see Saunders, 1980; Woodcock & Davis, 1978)[4] activation of the threat-

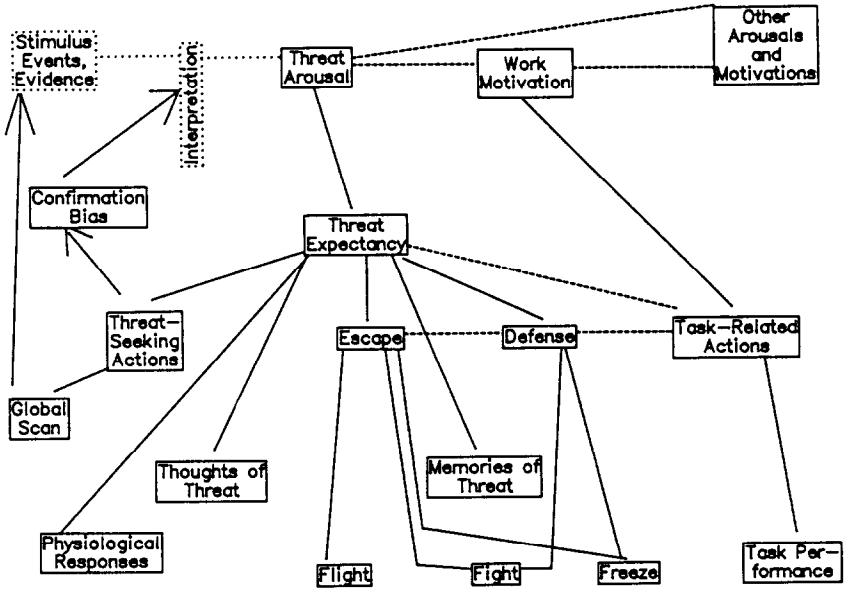

FIG. 2. A portion of a hierarchical cognitive action network for PTSD. This figure presents a very schematic representation of the kind of PTSD network that may be present in combat veterans.

arousal node and a consequent inhibition of alternative nodes representing other incompatible arousals, motivations, etc.

Activation of the threat-arousal node also potentiates a threat expectancy—the belief that some threatening event will occur—as well as other thoughts, images, and memories related to the expectation of a threat. This spreading activation (see e.g., Anderson, 1980) from the threat-arousal node to related nodes is ordinarily helpful to an individual because it allows activation of related concepts and actions, which in

[4] This activation corresponds to a cusp catastrophe in which the level of evidence and potentiation are the control variables and level of activation is the "behavior" variable. A catastrophe is a topological construct representing variables that change continuously over some part of their range and discontinuously over other parts. In this case, certain combinations of threat-evidence and threat arousal can cause either a gradual increase in the unit's level of activation or a sudden jump in activation. A catastrophic activation rule also predicts the presence of hysteresis in activation. For example, the level of threat evidence at which a node changes from inactivity to activity (its activation threshold) may be considerably higher than the level of evidence at which it changes from activity to inactivity (its deactivation threshold). This property implies that activation of a PTSD episode may require very little evidence of danger, but deactivation of the episode may require a great deal of evidence for safety.

normal situations would potentiate their availability for dealing adaptively with the present situation. In the individual with PTSD, however, this spreading activation may be the source of intrusive thoughts and nightmares as nodes corresponding to these events become inadvertently activated. The threat expectancy, in turn, potentiates a number of actions and physiological responses such as epinephrine release, tachycardia, and escape and defense motivations. Perception of these physiological responses may provide the individual with further evidence of danger. Activation of a threat expectancy also potentiates actions that seek evidence for threat. These actions focus attention on limited, threat-salient features both in the environment and within the individual (cf. Wachtel, 1967; Beck & Emery, 1985); they increase the likelihood that ambiguous stimuli will be interpreted as threatening (e.g., by lowering the strength of evidence necessary to activate a threat-related node), and they increase estimates of the degree to which this evidence is threatening (see also, Butler & Mathews, 1983). The detection of threat-evidence, although possibly spurious, increases the level of threat arousal, which then further potentiates the threat expectancy, further focuses attention, and further potentiates the individual's bias to discover and confirm evidence for threat. In short, weak evidence for threat leads to a positive feedback loop that provides more and stronger (subjective) evidence of threat, which increases threat arousal and promotes the interpretation of evidence as threatening, and so forth, until the individual is in a state or "mode" of specific hyperarousal (cf. Beck & Emery, 1985). This hyperarousal serves to activate cognitive and behavioral actions that are specifically relevant to the context and to seeking additional evidence for threat. As a result, hyperarousal results in increasing focus on potentially threatening information and in decreasing focus on alternative sources and types of information. This positive feedback mechanism is shown in Figure 3.

It is also a common finding that the general capacity to process information first increases and then decreases with increasing levels of arousal (Easterbrook, 1959; Kahneman, 1973). Therefore, increasing focus on the detection and interpretation of putatively threat-related stimuli uses up increasingly large proportions of a decreasing information-processing capacity. Alternatively (or, more likely, additionally), the mutual inhibition associated with activation of threat arousal inhibits the operation of other information-processing modes or schemata, thereby preventing their operation and further narrowing the attentional focus on threat-related stimuli.

A catastrophic activation function, with its inherent hysteresis, also implies that this threat-arousal node will ordinarily be strongly inhibited by the other competing nodes in the network. Inhibition of this node also inhibits the lower-level nodes to which it is connected. Just as arousal of this node makes the information represented by lower level nodes more available, inhibition of this node makes that information less available. As the individual passes from inhibition to activation, he or she will expe-

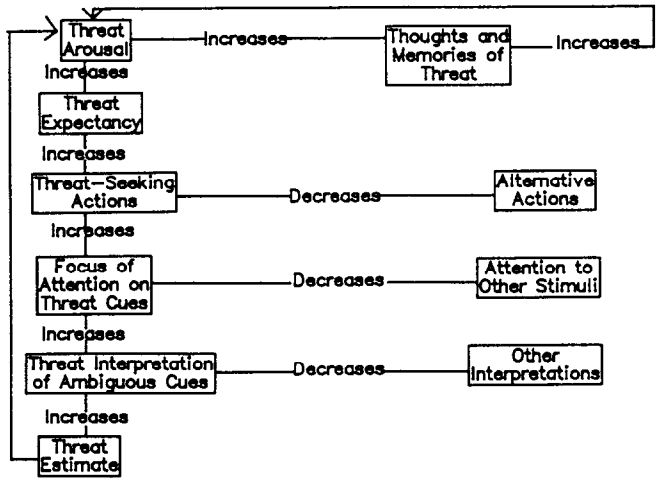

FIG. 3. A description of the vicious-cycle positive feedback loop underlying the induction of a PTSD episode.

rience first difficulty retrieving information (e.g., memories) about the traumatic episode and then experience difficulty suppressing that information. The individual may oscillate between emotional numbing and hyperarousal (e.g., Horowitz, 1976, 1980).

PTSD networks versus normal networks

Presumably, the same kinds of conceptual networks are present in both normal individuals and those suffering with PTSD. For example, even among normal individuals, some stimuli seem to have powerful attention-attracting characteristics. "Shadowing" is a task in which the subject is instructed to repeat a message as it is heard in one ear and to ignore whatever message may be presented to the other ear. Moray (1959) found that subjects shadowing a message presented to one ear reported hearing their own name repeated in the other ear, despite the fact that they could not report the content of the message presented to the neglected ear. In a related experiment, Treisman (1960) found that listeners would shift from shadowing a message in one ear to shadowing a message in the other, which they had been instructed to ignore, if the message in the unattended ear provided a better continuation of the message than did the message in the attended ear.

Attended ear:
"*John withdrew all of his* price worried the possible."
Unattended ear:
"For several newcomers the *money from the bank to pay his bill.*"

Subjects repeat the words, shown in italic, that provide good continua-

tion of the message. One explanation that has been offered of findings like these is that detection of certain kinds of stimuli is potentiated, either permanently as a result of the personal relevance of the stimuli to the individual (e.g., the person's name) or as a result of expectancies derived from the context (Treisman & Geffen, 1967). Presumably, detection of threat-related stimuli is similarly potentiated in individuals suffering from PTSD.

Confirmation bias, that is, the tendency to seek information that is consistent with an individual's expectations at the expense of other kinds of information, is very commonly observed among normal individuals and even among psychologists and other scientists (Wason, 1960, 1968; Tweney, Doherty, & Mynatt, 1981). Confirmation bias provides another example of the presence in normal individuals of a mechanism central to the description of PTSD.

Consistent with the hypothesis that individuals with PTSD have potentiated mechanisms for detection of threat-related stimuli, data derived from studies of anxious subjects indicate that these subjects' attention is strongly attracted by stress-related or fear-related stimuli. MacLeod, Mathews, and Tata (1986) found that clinically anxious subjects showed reduced response times to detect visual stimuli that appeared in the vicinity of threat-related words as compared to when these stimuli appeared near neutral words. A number of experiments found that naming the color of an anxiety-related word in a Stroop-like task was slower than naming the color of a neutral word (e.g., Ray, 1979; Mathews & MacLeod, 1985; Watts, McKenna, Sharrock, & Treasize, in press). Similarly, anxious subjects are better able to detect threat-related than neutral auditory stimuli. For example, mothers who were anxious because their children were about to undergo surgery identified more stress-related words among distracting auditory information than did control subjects (Parkinson & Rachman, 1981; see also, Burgess, Jones, Robertson, Radcliffe, & Emerson, 1981).

Although the mechanisms we propose are present in both those suffering from PTSD and those not so afflicted, we argue that individuals with PTSD may be distinguished from other individuals by at least three factors in the network. *First,* they show higher standard (or "resting") levels of potentiation of the threat arousal node. Even in the absence of any threat-evidence, PTSD subjects have some precurrent level of potentiation of the threat-arousal node that prepares them to seek and interpret evidence of threat.

Second, individuals with PTSD can be characterized by the "gain" of their arousal positive feedback loop. Gain is a notion derived from control theory. It refers to the speed with which the vicious-cycle positive feedback loop leads to extraordinary increases in arousal level. The model proposes that increases in the level of threat arousal increase the individual's readiness to interpret evidence as threatening. The perception of threat information, in turn, increases the threat arousal. Gain is the amount by which perception of threat increases arousal and increases

the readiness to interpret information as threatening. By hypothesis, individuals who have or are at risk for PTSD are very susceptible to this positive escalation in arousal and interpretation.

Third, PTSD sufferers have higher limits on the magnitude to which the threat arousal node can be activated. There must be limits on the extent to which a node can become activated. The nervous system, for example, has limits on the speed with which it can respond and on the magnitude of its response. Limits may also come from self-inhibiting feedback links. Above a certain threshold level of activation, for example, a node may begin to inhibit itself. In any event, arousal is normally limited to be within certain bounds and typically for limited durations. Relative to normal individuals, those with PTSD may have higher limits to their arousal or higher thresholds for the activation of arousal-damping mechanisms. Further, nodes representing other arousals and activities may be less effective inhibitors of threat arousal in individuals with PTSD than in other individuals. PTSD sufferers thus reach abnormally higher levels of arousal, which then serve to inhibit more and more strongly alternative information-processing and behavioral strategies, modes, and schema. The abnormally high level of arousal may correspond to the individual's subjective reports that experiencing an episode of stressful arousal often results in a switch in the individual's "mode" of experience. Other affective states are inhibited by the abnormally high levels of threat arousal, which may correspond to the subjective numbing characteristic of PTSD sufferers. Similarly, alternative nodes that ordinarily keep threat arousal low may activate actions, intentions, and so forth that are incompatible with threat-arousal and that seek to avoid cues for threat. When threat-related cues are encountered, however, they lead catastrophically to an increase in threat arousal and inhibition of alternative nodes. This pattern of mutual inhibition may explain why PTSD sufferers seem to oscillate between states of threat-avoidance and threat confrontation (e.g., Horowitz, 1976).

We can only speculate as to the possible causes of the differences between individuals with and those without PTSD. One obvious possibility is that individuals who are susceptible to PTSD are susceptible precisely because their information-processing system characteristically shows high gain. On this hypothesis, individuals who are at risk for developing PTSD would be expected to show stronger confirmation bias than other individuals. Increased gain and confirmation bias, thus, would provide a marker for susceptible individuals before they have the traumatic experience that would lead to PTSD. Consistent with this hypothesis, individual differences in arousability have been found to correlate with ease of conditioning and resistance to extinction to phobic stimuli (Hugdahl, Frederickson, & Ohman, 1977).

A second possibility is that the hyperarousal associated with life-threatening events produces permanent changes in the degree to which the threat-arousal is potentiated and, therefore, in the magnitude of confirmation bias. For example, neural hormones released under conditions

of extreme arousal may modulate the degree and type of learning that may take place, perhaps permanently modifying the individual's conceptual network (see Jacobs & Nadel, 1985). If this second hypothesis is correct, then modification of the conceptual structure back to a normal state may depend on a reinstatement of the abnormal neural-hormone levels that were necessary to produce it. Variations in the degree to which individuals produce these neural hormones in response to stress could provide another potential marker for PTSD susceptibility. Actual induction of PTSD, however, would still depend on the degree to which the individual were aroused by the traumatic event. Jacobs and Nadel (1985) suggest that reinstatement of these neural hormone levels may be necessary to successfully modify responses learned under these conditions of hyperarousal.

SUMMARY AND CONCLUSION

The cardinal features of PTSD include intrusive memories, avoidance, memory and concentration difficulties, and hyperalertness. The hierarchical cognitive action theory we have described accounts for each of these features. According to this view, intrusive memories (including nightmares and flashbacks) are due to spreading activation from the threat-arousal node to related nodes representing information about related situations, episodes, etc. The greater the activation of the threat-arousal node, the greater the activation of these related memories, and presumably the greater their tendency to intrude. Avoidance of threat-related stimuli is normally controlled by the activation of ordinary arousals and intentions. Because these arousals are inconsistent with the threat-response mechanisms, and because the experience of threat-related material is unpleasant, these nodes serve to actively inhibit threat arousal. Despite this active inhibition, however, even small amounts of evidence for the presence of a threat can serve to activate the threat-arousal node and inhibit the alternative nodes. Inhibition of the alternative nodes removes one stumbling block to activation of the threat arousal (their inhibition of this node) and accounts for the difficulty individuals report with (nonthreat) task-related memory and concentration. Finally, the vicious cycle positive feedback controlling activation of the threat arousal also accounts for the reported hyperarousal and hyperalertness as the individual seeks additional evidence for threat and prepares to respond to it.

The hierarchical network view of PTSD that we describe here has important treatment implications. First, it is consistent with the two effective treatment methods derived from viewing PTSD as a conditioning phenomenon (Keane et al., 1985). Implosive therapy repeatedly exposes the patient to presentations of the threatening CS without the occurrence of the dreaded consequences—the US. Presumably these presentations allow extinction of the conditioned response (Fairbank, Gross, & Keane, 1982; Fairbank & Keane, 1982; Keane & Kaloupek, 1982). Stress man-

agement serves as the basis of the second major treatment method. This method repeatedly presents the putative CS while simultaneously counter-conditioning incompatible relaxation responses. Therefore, this treatment allows opportunities for both extinction and counter-conditioning. The hierarchical network view is consistent with both of these treatments because they both can be seen to result in modifications of the fear structure.

In fact, the present view argues that any treatment that results in modification of the fear structure at sufficiently high levels in the hierarchy will be effective. For example, one could modify either the feedback loop, the precurrent fear-arousal level, or the strength of the connections between the fear arousal and lower levels in the hierarchy. The current conceptual network approach also argues for other treatment methods such as specific instruction regarding the validity of ambiguous evidence as a threat predictor and specific training to potentiate alternative nodes that are incompatible with threat arousal. In short, any treatment that reduces the gain of the feedback loop, reduces threat potentiation, or results in the production of strong incompatible competing responses will be predicted to be effective. These treatment implications also suggest a number of research opportunities—investigating the suggested markers for PTSD and investigating other treatment regimens that may be successful at modifying the network and the gain of the positive feedback loop.

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