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TREATMENT FOR TRAUMATIC MEMORIES: REVIEW AND RECOMMENDATIONS

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ABSTRACT. *The aim of this article is to provide recommendations concerning the use of exposure-based therapy for reduction of emotional responding to traumatic memories. Background for these recommendations consists of a summary of the literature on traumatic stress and symptoms of posttraumatic stress disorder (PTSD); an overview of biological, cognitive, and behavioral models for traumatic memory; and a selective review of evidence for the effectiveness of therapeutic exposure as a treatment for trauma memories and PTSD. The recommendations themselves demonstrate how clinical decision making during the course of treatment might be informed by empirical evidence and theoretical models concerning human memory, as well as ethical and legal considerations that mark this topic. © 1998 Elsevier Science Ltd*

THERE ARE NUMEROUS points of intersection between the topics of memory and psychological trauma, extending from assessment and diagnosis (e.g., recall of and reporting on traumatic experiences), through the study of basic processes that underlie posttraumatic disturbance (e.g., cuing of emotional reactions), to the challenge of developing interventions that can reduce the distress and debilitation associated with posttraumatic stress disorder (PTSD). Our aim is to present recommendations regarding the application of an empirically demonstrated treatment technique in light of recent advances in the theoretical and empirical field of human memory. We begin by providing a general overview of trauma and PTSD, with emphasis on issues

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related to memory. Next we describe three of the primary theoretical approaches to memory phenomena within the context of human traumatic stress. The biological, cognitive, and behavioral approaches are distinct in their respective emphases and offer unique perspectives on the traumatic memory issue. Together they provide a foundation for the treatment recommendations offered later. The third section reviews empirical evidence for the use of therapeutic exposure as a treatment for traumatic memories and thereby provides a rationale for our focus on this type of intervention. Finally, we present a set of practically oriented recommendations concerning the application of exposure procedures. The emphasis is on clinical decisions that can be informed by recent advances related to human memory, particularly traumatic memory.

OVERVIEW OF TRAUMATIC EVENTS AND POSTTRAUMATIC STRESS DISORDER

One consideration that distinguishes PTSD from other formal mental disorders is the required identification of a (putatively) causal traumatic experience. Specifically, diagnostic Criterion A (*DSM-IV;* American Psychiatric Association, 1994) requires that "the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others," and that "the person's response involved intense fear, helplessness or horror." Traumatic events with empirically demonstrated links to PTSD include childhood physical and sexual abuse (Duncan, Saunders, Kilpatrick, Hanson, & Resnick, 1996; Lindberg & Distad, 1985), adult rape (Kilpatrick & Resnick, 1993; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992), military combat (Keane, Zimering, & Caddell, 1985; D. W. King, King, Gudanowski, & Vreven, 1995; Kulka et al., 1990), and natural and technological disasters (Baum, Gatchel, & Schaeffer, 1983; Green et al., 1994; Pynoos et al., 1993). Such events vary in their potential to be traumatogenic, but evidence suggests that those which involve personal injury, forced sexual penetration, and threat of death are associated with more severe PTSD symptoms (Kilpatrick et al., 1989). In addition, many events that are potent sources of stress are largely unpredictable and uncontrollable, factors that are thought to undermine an individual's sense of safety and increased personal vulnerability (Foa, Rothbaum, & Zinbarg, 1992; Janoff-Bulman, 1992). The combination of high stress potency and individual vulnerability may be especially detrimental to mental health and functioning.

Several epidemiological studies have examined the prevalence of exposure to extremely stressful events (Breslau, Davis, Andreski, & Peterson, 1991; Norris, 1992; Resnick et al., 1993) and rates of PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Kulka et al., 1990). The National Comorbidity Study (NCS; Kessler et al., 1995) was the first to conduct face to face interviews with individuals drawn from the general population to determine the prevalence of exposure to potentially traumatic events and *DSM-III-R* disorders. The results of the NCS show that trauma exposure is a common feature of modern life. Lifetime prevalence rates for the occurrence of at least one traumatic event were 51.2% for women and 60.7% for men.

In the NCS, Kessler et al. examined the frequencies of some specific types of traumatic events. Among the most striking, 9.2% of women reported a history of rape, 12.3% reported childhood molestation, and 15.2% reported being in a fire. For men,

35.6% reported witnessing violence, 25% reported being in a life-threatening accident, and 2.8% reported history of molestation. These rates are consistent with other large scale, community samples that have found high rates of exposure to sexual and physical violence. These population estimates indicate that exposure to a wide variety of life-threatening and violent events occurs with relative frequency across a broad spectrum of the population.

Although the prevalence of PTSD is much lower than prevalence of exposure to potentially traumatic experiences, the disorder is diagnosed at relatively high rates in comparison with other mental conditions. For example, 7.8% of respondents in the NCS sample were diagnosed with a lifetime history of PTSD. This rate is comparable to the PTSD rates of 6% among men and 11% among women found in another community sample (Breslau et al., 1991). In the National Women's Study, Resnick et al. (1993) examined a representative random sample of 4,008 adult women in the United States and found similar lifetime PTSD rates (12%) for women following exposure to sexual or physical assault.

At-risk populations (e.g., combat veterans) have increased potential for traumatic exposure and demonstrate even higher PTSD rates. The National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990) is the most extensive study to date investigating the prevalence of PTSD in a representative, community sample of military veterans. The NVVRS found a PTSD lifetime rate of 30.9% for male Vietnam theater veterans and 26.9% among female Vietnam veterans. The risk of developing PTSD may be even higher among special populations with multiple or repeated exposures to potentially traumatic events. For instance, the rate of lifetime PTSD was in the range of 60% for male veterans from the NVVRS sample who had the highest levels of reported combat exposure. Similarly, the lifetime rate was 58% in a group of hospitalized veterans with histories of both childhood abuse and combat (Triffleman, Marmar, Delucchi, & Ronfeldt, 1995). As the preceding figures indicate, a substantial proportion of individuals exposed to traumatic events develop acute PTSD symptoms and a significant subgroup remains chronically symptomatic. Although recent longitudinal findings suggest that many individuals with acute PTSD show decreased symptom intensity 1 to 4 months following a traumatic event (Foa, Hearst-Ikeda, & Perry, 1995; Rothbaum et al., 1992), symptoms can and do remain high for 25 years or more (Frueh, Mirabella, Chobot, & Fossey, 1994).

Symptoms of PTSD

A core set of both acute and chronic symptoms involves re-experiencing of memories of the traumatic event (Criterion B; *DSM- IV*). Flashbacks are a striking example because of their highly arousing quality. This appears to be due to their multisensory nature and sudden, typically unexpected occurrence. Most individuals with PTSD also report intrusive memories that may be characterized as long-lasting preoccupations with their traumatic experiences. These memories are often emotionally volatile and subjectively disruptive to cognitive functions such as attention and concentration. Another re-experiencing symptom, nightmares, appears to be an emotionally charged replay of the traumatic experience in literal or thematically similar terms. Nightmares also promote sleep disturbance that can have debilitating effects on both physical and cognitive functioning beyond the immediate negative emotional impact of the nightmares themselves. Finally, two frequently reported symptoms can be characterized as cued re-experiencing. These involve heightened distress or psychophysi-

ological reactivity to reminders of the traumatic events that presumably reflect the emotional conditioning established by a traumatically stressful experience.

Individuals with PTSD often try to impede recollection or limit processing of memories in an apparent effort to control re-experiencing symptoms. Attempts are made to avoid physical reminders of the trauma, such as people and places. In addition to these concrete avoidance strategies, the symptom-related states of emotional numbing and cognitive dissociation can be conceptualized as covert means to avoid distress that accompanies traumatic memories. Amnesia for some or all aspects of the traumatic event may be considered motivated forgetting that also qualifies as a form of covert avoidance. These strategies can be viewed as self-protective actions that provide short-term emotional quiescence or relief and promote a sense of control for the traumatized individual. Reciprocal oscillation between re-experiencing and avoidance symptoms may even be a hallmark feature of PTSD adjustment (Foa, Riggs, & Gershuny, 1995; King & King, 1994; Litz, 1992). On the other hand, predominant use of self-protective actions may impede beneficial emotional processing (e.g., Foa & Kozak, 1986) and have the effect of preserving emotional memories and their associated symptoms.

The third major category of PTSD symptoms (Criterion D) is labeled hyperarousal and includes affect dysregulation, exaggerated startle response, insomnia, rageful outbursts, and hypervigilance. One prominent theory posits conditioned fear as the primary agent responsible for hyperarousal symptoms and associated anxious reactions (Keane et al., 1985). An implication of this view is that hyperarousal is a consequence of and exists secondary to re-experiencing phenomena. From this perspective, a rather diverse set of symptoms can be ultimately linked back to processes that involve memory as a key element. One reason the hyperarousal category may appear distinct from re-experiencing is that different self-protective actions may be employed. Behavioral avoidance and cognitive dissociation appear to be means for managing the re-experiencing aspects of posttraumatic symptoms, while emotional numbing may serve to dampen hyperarousal symptoms (Litz, 1992).

The Course of PTSD

Most often PTSD develops relatively soon (i.e., weeks) after a traumatically stressful experience. However, the literature is replete with descriptive cases documenting delayed-onset PTSD in concentration camp survivors (Chodoff, 1963), Hiroshima bomb survivors (Lifton, 1968), American World War II veterans (Archibald & Tuddenham, 1965), Korean War veterans (Pomerantz, 1991), Vietnam war veterans (Van Putten & Emory, 1973), Israeli veterans of the Lebanon War (Z. Solomon, Blumenfeld, & Singer, 1995; Z. Solomon, Kotler, Shalev, & Lin, 1989), rape survivors (Burgess & Holmstrom, 1985), and victims of sexual abuse (Sharpe, Tarrier, & Rotundo, 1994). It appears that stressful or traumatic incidents in an individual's life also can precipitate the activation of symptoms linked to a previous traumatic stressor. Precipitating events include trauma anniversaries, interpersonal losses, changes in health status, witnessing other's trauma and stressful life changes (Christenson, Walker, Ross, & Maltbie, 1981). In this vein, we've heard informal clinical descriptions of individuals with combat experience in their 20s who were relatively symptom-free until they experienced a life-threatening disorder (e.g., heart attack) in their 50s or 60s and then became acutely symptomatic for PTSD. However, rather than having intrusive thoughts and nightmares about the recent stressor, they reported re-experiencing

symptoms with content tied to events that occurred in combat. This contrasts with the more typical situation in which PTSD symptoms that abate after one traumatic event are reactivated by a subsequent, distinctly different stressor or traumatic experience. These patterns of symptom activation may offer clues about the underlying memory processes involved in PTSD, just as demonstrations of treatment generalization (or lack thereof) across exposure session content (Fairbank & Keane, 1982; McCaffrey & Fairbank, 1985) have begun to illuminate the related issue of fear network organization in the disorder.

Overview Summary

Epidemiological studies demonstrate that potentially traumatic events affect the lives of many people and that PTSD is a common consequence of such experiences. The PTSD symptom cluster most related to memory processes is re-experiencing. This includes the occurrence of cued emotional distress and physiological reactions, recurrent thoughts about the experience, nightmares, and flashbacks. High levels of anxiety may accompany these symptoms and the affected individual may use behavioral and/or cognitive means to circumvent the emotional distress and physiological arousal that is evoked by them. These efforts can afford short-term control, but they also have potential to preserve the emotional learning tied to memories of traumatic experiences. In the absence of suitable processing, the memories and associated emotion can remain capable of being elicited by reminder cues for years—potentially for a lifetime.

MODELS OF TRAUMATIC MEMORY

As noted earlier, a flashback is a distinctive PTSD symptom in which there is reliving of a traumatic incident in the form of intense images and emotion. For instance, a military veteran may hear a helicopter flying overhead and be bombarded by visualizations from a traumatic war experience and overcome with terror. How does a seemingly harmless occurrence, like hearing a helicopter, trigger a powerful multisensory response? Extraordinarily stressful events can produce enduring, emotional memories that are recalled very clearly and accurately (Cahill & McGaugh, 1995; LeDoux, 1992). Once cued, the event's meaning, fearful imagery and associated defensive responses emerge, unexpectedly and unpredictably, as PTSD symptoms. Thus, theoretically, the sound of a helicopter activates memory processes that precipitate a fearful re-enactment of the original traumatic experience. The remainder of this section provides an overview of biological, cognitive, and behavioral models of traumatic memories using flashbacks and other re-experiencing symptoms to illustrate how trauma affects memory and how traumatic memories lead to PTSD symptoms from each perspective. We begin by exploring the process of memory encoding and storage.

Biological Models

Several investigations have examined how stress-related hormones may regulate or mediate the development of traumatic memories, alter the synaptic circuitry of brain regions, and influence the expression of flashbacks and other intrusive memories

(for reviews, see Bremner, Krystal, Southwick, & Charney, 1995; McCarty & Gold, 1996). Stressful events activate the hypothalamic-pituitary-adrenal (HPA) axis, a set of structures which supports adaptation to environmental demands by mobilizing energy and redirecting nonnecessary bodily processes. The stress-response process begins when the pituitary gland releases a peptide, adreno-corticotropic hormone (ACTH), which signals the adrenal cortex to release the stress hormone cortisol throughout the body. Stress also leads to the release of catecholamines, such as norepinephrine, from the adrenal medulla and thereby affects the release of glucose into the central nervous system. Recent investigations suggest that both cortisol and norepinephrine influence memory encoding (LeDoux, 1995), consolidation (McEwen, 1995), and retrieval (Gold & McCarty, 1995). These hormones may enhance memory formation process at the synaptic level, but excessively elevated hormonal levels may cause neuronal atrophy or death (Post, Weiss, & Smith, 1995).

Cortisol and norepinephrine appear to affect memory formation through two brain regions, the amygdala and hippocampus. One of the amygdala's primary functions seems to be the development and expression of conditioned fear (Davis, 1992). The hippocampus, on the other hand, has been shown to have an important role in contextual memory; for example, encoding and storing temporal, linguistic and spatial features of the traumatic experiences (Kesner, Hopkins, & Chiba, 1992). The relevance of conditioned fear and contextual memories will be discussed in greater detail later.

Cortical (related to cognitive processes) and limbic (related to emotional responses) projections reach the thalamus which then relays the neuronal signals on to the amygdala. Once activated, the amygdala mediates the impact of adrenal hormones, cortisol and norepinephrine on other brain regions and thereby regulates the strength of fear memories (Cahill et al., 1996). Indeed, the amgydala has been called the "emotional computer" (LeDoux, 1992) because of its role in coordinating the process that begins with evaluation of sensory information for significance (e.g., threat) and then controls the resulting behavioral and autonomic responses.

The amygdala and hippocampus are vulnerable to neurotoxic effects because they contain a large number of cortisol receptors. Importantly, excessive cortisol release following an aversive or stressful experience can cause hippocampal neurons to atrophy (McEwen, 1995; Sapolsky, 1996; Yehuda & Sapolsky, 1997) and may impair subsequent hippocampal functioning related to learning and memory (Jacobs & Nadel, 1985). Bremner et al. (1995) investigated hippocampal atrophy in humans using Positron Emission Tomography (PET) images. Combat veterans with PTSD were found to have 8% less volume in the right hippocampus when compared to non-PTSD controls. It is unclear whether these volume differences in the hippocampus are related to elevated cortisol and stress, however, it is theoretically plausible that they are trauma related and have a role in the phenomenon of memory re-experiencing in PTSD.

Another line of evidence suggests that norepinephrine levels mediate flashbacks, as well as other recurring traumatic memories and accompanying arousal that is found with PTSD (Krystal, Bennett, Bremner, Southwick, & Charney, 1995; Pitman, 1989). The theoretical connection between norepinephrine and flashbacks has been investigated by administering yohimbine, an alpha-adrenergic receptor antagonist, to PTSD patients (Bremner et al., 1997; Southwick et al., 1993). Yohimbine appears to cause increased levels of catecholamine release and activation of the locus ceruleus neurons in the brain. Stimulation of the locus ceruleus through norepinephrine re-

lease has been found to produce high states of arousal and fear (Redmond & Huang,1979). Results from the yohimbine administration studies with PTSD patients indicate that 60–70% of those tested report panic attacks and 30–40% report intense visual flashbacks. These findings offer insight into the possible neurobiological consequences of severe stress and link them to phenomenological consequences involving traumatic memories expressed as flashbacks.

Cognitive Models

Cognitive approaches to PTSD have focused on social beliefs, information processing systems and coping strategies related to trauma. One popular set of cognitive theories posits that a traumatic experience violates an individual's basic beliefs about personal safety, control, and survival and thereby fosters an increased sense of vulnerability, decreased confidence in one's capacity to negotiate life's difficulties, and greater willingness to entertain ideas about the malevolence of human nature. The magnitude of changes in these beliefs is tied to the severity of the violation (not necessarily the severity of the trauma), with increasing likelihood of the beliefs being overgeneralized to inappropriate situations as the severity of violation increases. From this perspective, self-protective (e.g., avoidant) actions can be understood as strategies for surviving in a seemingly dangerous world, with the maladaptive aspects of such behavior a function of the degree to which the beliefs are generalized to inappropriate situations (for additional reviews of social-cognitive theories, see Horowitz, 1976; Janoff-Bulman, 1992).

Freyd (1996) has formulated a cognitively based account for self-protective avoidance and inaccessibility of memories found with a particular class of traumatic experiences, those involving maltreatment of children at the hands of their caregivers. This theory, termed *betrayal trauma theory*, emphasizes the evolutionary mandate for dependency of children on adults, and the dual relationship between abusive caregiver and child characterized by the provision of protection and love, on one hand, and abusive violence, on the other. The fact of dependency limits possibilities for physical escape, so children are hypothesized to resort to cognitive processes that make the situation more tolerable. These processes include appraisal of the situation in terms which are less incompatible with caregiving and thereby less threatening to the relationship (e.g., I'm bad and deserve this), as well as suppression of or disengagement from current reality (e.g., dissociation) as a means for dampening emotional pain. The long-term impact of the cognitive maneuvers is to reduce the likelihood of subsequent cuing of abuse-related memories outside the situation because they are encoded in a manner specific to the cognitive set and developmental level of the child. This negative effect on recall is sustained by the conspiracy of silence that surrounds incest and abuse.

Information processing theories focus on the structure of memory and the processes of attention, behavioral action, and arousal that relate to that structure (see Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Creamer, Burgess, & Pattison, 1992; Foa & Kozak, 1986; Litz & Keane, 1989). A fruitful example of this approach is the theorizing of Foa and Kozak (1986), who apply Lang's (1977, 1979) bioinformational theory to explain the information contained in fear structures in memory. They propose that a fear network contains representations of three elements: (a) the stimulus aspects of the feared situation; (b) the response aspects of fear, including subjective, physiological and behavioral components; and (c) an interpretation of the connection between stimulus and response elements (i.e., the meaning of the information). According to Chemtob et al. (1988), a network of fear underlying PTSD

can demonstrate sustained activation in the service of keeping the individual alert, ready and on guard against danger. This may explain the hypervigilance aspect of the disorder.

A recent attempt to integrate the social-cognitive and information processing perspectives on PTSD comes from Brewin, Dalgleish, and Joseph (1996). They proposed a dual representation theory that conceptualizes traumatic memories at two levels: elements that are verbally accessible and elements that are situationally accessible. Verbally accessible memories are considered part of an individual's conscious processing of a traumatic experience. They are easily retrieved, long-term memories that include personal beliefs and everyday working knowledge about the world. Similar to the social-cognitive approach, they are used to explain preoccupation with a trauma in terms of the individual's attempts to create meaning from the experience, for example, by determining cause and ascribing blame for it.

By contrast, situationally accessible memories are nonconscious, not subject to control by the individual, and not directly available for recall or introspection. They are applicable to the process whereby fear and other emotions are linked to cues that are present at the time of a traumatic event, as well as the subsequent elicitation of such emotions by contact with reminder cues in nontraumatic settings. Dual representation theory does not specify the form in which situationally accessible memories are represented, but it does point to models for schematic emotional memories (Leventhal, 1984) and parallel distributed processing (Rumelhart, McClelland, & the PDP Research Group, 1986) as possible options. Like schematic memories, situationally accessible memories function to store, organize and process information. There also is growing empirical evidence from information processing paradigms that, despite their nonconscious nature, these memory structures can actively direct attention toward threat-related environmental stimuli leading to biases in the recall and perception of danger (see Litz & Keane, 1989).

It is important to note that re-exposure to stimuli resembling those present during a trauma can cause the information contained in the situationally accessible memory to erupt into a conscious experience. Stimulus triggers for these memories include internal states, like emotions or thoughts, as well as external reminders. Once activated, the person can become fully aware of the fear structure, including the arousal, somatic reactions, imagery, and dissociative experience. It is the concept of situationally accessible memory that seems to fit the phenomenology of the re-experiencing symptoms such as flashback and cued distress or physiological responding. Intrusive thoughts, by contrast, seem more consistent with the verbally accessible format.

A Behavioral Model

The behavioral perspective on traumatic memories emphasizes the role of conditioned emotional responses (Foa, Steketee, & Rothbaum, 1989; Keane et al., 1985; Kilpatrick, Veronen, & Best, 1985). Mowrer's two-factor theory provides a framework that applies especially well to many of the re-experiencing and avoidance features of PTSD. A reaction of fear or horror can be classically conditioned to the context in which it occurs, including discrete stimuli (e.g. odors, noises, scenery) present during the traumatic experience. Re-exposure to similar contexts or stimuli can evoke the memory of the trauma and a powerful conditioned fear response. This conceptualization readily applies to flashbacks.

Passive avoidance (e.g., not leaving home) may prevent conditioned distress altogether, active avoidance may reduce anxiety that is elicited in anticipation of trauma-reminder cues, and escape behavior can terminate distress evoked by contact with the conditioned cues themselves. However, the trade-off for this emotional quiescence or relief is the preservation of the conditioned reaction and continuing susceptibility to distressing encounters with trauma reminders.

The two-factor model has been extended beyond this simple paradigm in a way that can explain the paradox of memory inaccessibility for some traumatic experiences despite evidence of enhanced memory associated with emotionally arousing events. The extended model, as formulated and empirically demonstrated by Levis and his colleagues (e.g., Levis & Boyd, 1979; Malloy & Levis, 1988; Williams & Levis, 1991), postulates that conditioned fear cues can be made especially resistant to extinction if they are ordered in serial fashion. A simple example comes from a study (see Levis & Boyd, 1979) with rats that received either one long (18 second) auditory signal followed by electric shock or a series of three distinct auditory signals presented one after another (for 6 seconds each), where only the final signal is followed by shock. After a conditioned emotional reaction was established by trials pairing the signal(s) with shock, the animals were allowed to begin avoiding the shock by jumping to a safe compartment when an auditory signal (conditioned stimulus) was present. This study, and others like it, have produced two findings of particular interest for present discussion. First, the animals which received the serial cues showed much greater resistance to extinction of the avoidance response (and the underlying fear state), taking more than three times the number of trials to reach the extinction criterion. Second, nearly four times as many avoidance responses occurred during the first 6-second stimulus in the three cue series as compared to the comparable 6-second period during the single stimulus. Extrapolating this latter finding to humans who experience trauma, we posit that avoidance which occurs very early in encounters with a series of conditioned emotional cues substantially reduces the likelihood of contact with later cues which were part of the traumatic experience itself. This limiting of cue exposure can serve as a barrier to retrieval of the trauma memories and may resemble forgetting in its effect. However, the fact that emotional conditioning to these cues is preserved by the lack of exposure leaves the memory susceptible to later reactivation. If reactivation occurs, it has potential to be rather dramatic because of strong emotional reactions and vivid recollections that can be triggered. Although the serial conditioning model may not apply to all traumatic experiences, it is worth considering in the conceptualization of cases in which affective memories seem to suddenly reappear after years of apparent forgetting.

Summary of Models

Biological, cognitive, and behavioral models appear to offer complementary rather than competing accounts for traumatic memory, with some degree of convergence in their implications for treatment. The biological literature demonstrates that specific stress-related hormones can selectively influence areas of the brain involved in memory encoding, storage, and retrieval. The result may be trauma-related memories that are especially potent and durable. Cognitive models are probably strongest in accounting for heightened readiness for threat perception and in representing interconnections among elements of traumatic memory that we term *meaning*. The behavioral models seems best able to address conditioned emotional responses and the

associated strategies for escape and avoidance that preserve fear learning. The serial-conditioning model offers an account for instances of apparent forgetting and eventual recovery of memories related to trauma. These models also provide the foundation for treatment based on the principle of extinction.

TREATMENT OF PTSD

Behavioral and cognitive-behavioral methods that fall in the category of exposure therapy are now arguably the treatment of choice for PTSD (S. D. Solomon, Gerrity, & Muff, 1992; Keane, 1995, 1998). The theoretical basis for this approach is the process of extinction, and the method for achieving extinction is the sustained presentation of stimuli to which a conditioned emotional reaction has been learned. Exposure therapy has a history of demonstrated success with conditions that include phobia (Chambless, Foa, Groves, & Goldstein, 1982; Heimberg & Barlow, 1988; Mathews, 1978; Steketee, Bransfield, Miller, & Foa, 1989), obsessive-compulsive disorder (Foa & Kozak, 1996; Kozak, Foa, & Steketee, 1988; Rabavilas, Boulougouris, & Stefanis, 1976), and panic disorder (Agras, 1985; Barlow & Cerny,1988). Exposure treatment procedures vary in terms of the method for presenting feared stimuli (in-vivo or via imagery) and in term of the rate or intensity of presentation (e.g., gradual and hierarchical or nongraded). The procedures are variously referred to as desensitization, flooding, or implosive therapy, but they have in common the aim of presenting material for sufficient time to producing within-session decreases in the intensity of emotional responding. They also share the aim of sustaining these decreases across treatment sessions as a demonstration of durable response extinction.

Exposure therapy provides a direct method for reducing conditioned anxiety and modifying trauma-related memories (Hawton, Salkovskis, Kirk, & Clark, 1994; Keane, 1995), and prolonged exposure is now cited as the most effective nonpharmacological treatment for reducing PTSD related intrusive memories (Otto, Penava, Pollack, & Smoller, 1996). Even multicomponent treatments for PTSD that include cognitive interventions have exposure as one of their key elements (Brewin et al., 1996; Keane, 1998; Paunovic, 1997). As the following review of treatment studies indicates, exposure therapy has been used successfully to treat PTSD due to combat, sexual assault, incest, motor vehicle accidents, and a range of other traumatic events. It has been used extensively with adults, as well as with adolescents and with children as young as six years old (Saigh, 1986, 1987).

Case Studies and Single Case Designs

Soon after the establishment of PTSD in the psychiatric nomenclature (*DSM-III;* American Psychiatric Association, 1980), Keane and others began applying exposure therapy to Vietnam veterans with PTSD (see Black & Keane, 1981; Fairbank & Keane, 1982; Keane & Kaloupek, 1982; McCaffrey & Fairbank, 1985). Black and Keane (1981) described the treatment of a World War II veteran with implosive therapy in one of the first demonstrations of exposure therapy as a means for reducing symptoms of combat-related PTSD. Keane and Kaloupek (1982) reported on the effects of imaginal flooding for PTSD with a Vietnam combat veteran and demonstrated sustained decreases in anxiety and physiological arousal to combat stimuli, as well as increased hours of sleep per night at 12-month follow-up.

As exposure therapy began to be considered as a viable treatment option for veterans with PTSD, investigators began to consider important theoretical issues related to the selection of a target for extinction. Fairbank and Keane (1982) employed a multiple-baseline design to demonstrate that exposure treatment can generalize across memories that contain similar stimulus and response elements or themes. In clinical terms, this suggests that trauma scenes with different thematic content require separately targeted interventions. In theoretical terms, it suggests that there are boundaries in the organization of traumatic memories.

One of the first single-case studies to examine the treatment efficacy of exposure on sexual trauma was conducted by Rychtarik, Silverman, Van Landingham, and Prue, (1984). They reported on the use of implosive therapy with a young adult female, incest victim. The treatment was able to reduce trauma-related intrusive memories of forced sexual relations by repeatedly presenting imagery with this content. Rychtarik et al. reported that the patient experienced a sustained reduction in intrusive thoughts and showed improved overall psychosocial adjustment.

These early case studies were important demonstrations of exposure treatment as a method for reducing PTSD symptoms. They provided encouragement for initiation of more elaborate and scientifically rigorous investigations.

Randomized Clinical Trials

A series of randomized clinical trials has tested whether exposure, especially imaginal flooding, is more effective for reducing PTSD symptoms than other therapy approaches (see Boudewyns & Hyer, 1990; Boudewyns, Hyer, Woods, Harrison, & McCranie, 1990; Brom, Kleber, & Defares, 1989). Highlights of this work include the study by Cooper and Clum (1989) that examined the impact of combining flooding with a standard psychotherapy procedure. Results indicated that flooding increased the effectiveness of standard psychotherapy, especially in the areas of re-experiencing, sleep disturbances, anxiety and depression.

Keane, Fairbank, Caddell, and Zimering (1989) examined flooding effectiveness in a design that compared two active treatments, exposure therapy and stress management, to a wait-list group. Individuals in the group receiving stress management dropped out of treatment at inordinately high rates, so Keane et al. compared the 11 Vietnam combat veterans who were randomly assigned to imaginal flooding with the thirteen veterans assigned to the wait-list control group. The flooding group demonstrated reductions in depression, anxiety, fear and re-experiencing intrusive memories.

Another pioneering study that also used rigorous clinical outcome methods was conducted by Foa, Rothbaum, Riggs, and Murdock (1991). They examined the effectiveness of four treatment conditions (exposure therapy, stress inoculation therapy, supportive counseling, and a wait list control condition) on PTSD in women who had been sexually assaulted. The key finding for present purposes was that the exposure group showed the greatest overall PTSD symptom reduction of all groups at the 3-month follow-up evaluation.

Summary of Treatment Research

The results from early case studies and the randomized clinical trials clearly indicate that exposure therapy is effective for reducing PTSD symptoms across trauma types. In particular, nightmares, flashbacks, and other intrusive re-experiencing symptoms

can be reduced by repeatedly reliving emotional aspects of the traumatic experience under controlled conditions in therapy. The next section addresses some of the ways in which theory and evidence regarding emotional memory can influence decisions related to the application of exposure therapy for PTSD.

RECOMMENDATIONS FOR EXPOSURE TREATMENT

Evaluation for Potentially Traumatic Experiences

Formal, multimodal assessment (e.g., Keane, Wolfe, & Taylor, 1987) is an important prelude to exposure treatment because it can provide information about both the historical aspects of traumatic experience and the cues and reminders that are present in the individual's contemporary environment. It is essential to identify material of this type that can be used to extinguish trauma-related emotional responding. This can be accomplished most readily when the individual seeking treatment is able to report spontaneously on an experience that both qualifies as traumatic according to PTSD Criterion A (*DSM-IV;* American Psychiatric Association, 1994) and appears to have a causal connection with their identified problems. There is minimal risk that the process of interviewing has biased an individual's recall or reporting if they are able to describe a suitable emotionally evocative memory with little or no prompting by the interviewer. Concern about bias enters the picture because of research evidence indicating the potential susceptibility of memory to the influence of suggestion. In addition, although it is important to minimize potential sources of bias in all clinical evaluations, it is particularly so during the assessment of traumatic experiences because of both ethical and forensic issues that arise in this context. These include liability incurred in relation to so-called false memories of abuse that were legally determined to be therapist initiated (e.g., Schooler, 1994).

The task of gathering material for exposure imagery scenes becomes more challenging and more at risk for iatrogenic influences on memory when an individual's report about a traumatic experience is incomplete or fragmentary. As discussed earlier, there are various practical and theoretical reasons why the usual enhancement of memory strength associated with traumatic experience may not be evident in some instances. Practically, as with memories in general, distance from the context of an experience in terms of location and circumstances (e.g., jungle combat vs. urban civilian activities), time (i.e., years since the event), and developmental level (e.g., early childhood vs. adulthood) may reduce the likelihood of memory cuing and reactivation. Theoretically, an explanation can be formulated on the basis of biological evidence suggesting adverse effects of stress hormones on brain structures that have important functions in memory (e.g., hippocampus), by considering the cognitive effects of emotional conflict that occurs for children who are abused by a caregiver, or in terms of the serial ordering of conditioned cues leading to early avoidance behavior that prevents reactivation of memories concerning a traumatic experience.

Irrespective of the reason, instances of poor recall may necessitate assistance by the clinician. This assistance should be designed to avoid hypothetical or leading questions, and to instead aid reconstruction of the temporal or sequential features of events (e.g., "what happened next?") and provide clarification or feedback (e.g., "earlier you said that . . .") similar to the approach advocated in client-centered therapy (Rogers, 1951, 1961). The strategy is to provide the individual with a structured opportunity to discuss their recollections of the experience and to help them be system-

atic in their efforts to recall and synthesize important details. The technique may be described as having patients cue themselves in order to achieve the goal of improved memory retrieval.

Fortunately, it is not necessary to have a complete picture of a traumatic experience before treatment can begin. One advantage of the exposure method is that even incomplete descriptions or depictions of an experience will often facilitate recall. Thus, as long as the assessment generates some material related to the traumatic experience, it should be possible to begin exposure treatment. Subsequently, new information collected in the course of exposure sessions can be incorporated into the process.

Two suggestions offered by Scheflin (1997) with regard to professional liability are pertinent to this incremental approach to the construction of exposure scenes. First, it may be wise to use a written consent form (see examples in Pope & Brown, 1996) to make the patient aware of the uncertain legal standing of any information retrieved from memory under these conditions. It is clear that information gathered under the influence of hypnosis is routinely excluded from testimony in court because of its perceived inherent unreliability due to the influence of suggestion. Although the present approach is intended to avoid suggestion, it remains to be seen whether this distinction will be evident in the legal arena. Second, it is essential to draw a distinction between the clinical setting of treatment and the everyday activities of the patient with respect to the question of memory accuracy or veracity. Treatment can proceed even if the patient's recollection is not fully correct or complete. It may not even matter if patient-generated recall is quite imprecise or incorrect in its details so long as certain key emotion evoking cues are identified and used in exposure treatment to reduce distress and arousal. By keeping the stated focus of treatment on symptom reduction and improved functioning rather than on reconstruction of literal history, it may be possible to prevent unwarranted expectations or unnecessary complications related to the quest for a truth that may not be possible to establish or verify.

Evaluation for PTSD and Other Trauma-related Symptoms

From a diagnostic perspective, the emotion elicited by presentation of trauma-relevant cues falls under the category of re-experiencing. Given the importance of emotional response and habituation to the process of exposure therapy, evidence of re-experiencing is a virtual prerequisite for such treatment. But what about instances where little or no re-experiencing is reported or where the content of re-experiencing is indistinct? For example, a military veteran might report nightmares with combat themes that don't have specific details they can relate to their own experiences. Or someone with no previously identified trauma history might report distress or physiological arousal triggered by cues that are not uniquely related to any particular type of traumatic experience (e.g., the scent of a particular cologne; the sight of someone wearing all white clothes). Assuming a reasonable degree of clarity in the recall, either the context without details (e.g., the combat example) or details without context (e.g., the cologne example) may offer a starting point for exposure treatment.

A more directive strategy advocated by Levis (e.g., Levis, 1980) makes use of hypothesized stimulus or response cues as probes of memory, especially emotional memory. The clinician decides on the cues to present based on inference, theory, or base rates (e.g., it is common for people to report awareness of their heart pounding when they are afraid), and then delivers them in relatively brief vignettes with the aim of generating emotional response and prompting additional recall. From a memory

perspective, the risk associated with the use of cues that are hypothesized rather than drawn directly from a patient's report is that they (the patient) may confuse the origin of the material and come to consider it part of their memory—even if it is incorrect or inaccurate. Efforts to use hypothesized cues to trigger recall must be careful to stay close to the information provided by the patient in order to avoid pitfalls similar to the biasing that may occur if leading questions are used to reconstruct memory for an event. For example, a hypothesized probe presentation in which a rapidly pounding heart is incorporated into the depiction of a recalled event may confuse the patient about which details they recalled on their own, perhaps even encouraging the addition of the hypothesized detail to subsequent recollections. It seems likely that the risk of unintended impact on memory is less when probes are used once, and that risk increases as the hypothesized material is repeated because of the subjective sense of familiarity that repetition engenders.

Dealing With the Recall of New Material During Treatment

As noted earlier, one of the expected consequences of exposure treatment sessions is reactivation of additional details for reported memories, as well as triggering previously unrecalled memories. This effect is understandable as a form of cued memory retrieval (e.g., Spear, 1973). Furthermore, to the degree that exposure scenes are constructed to reflect Lang's concept of stimulus, response, and meaning elements of imagery, it is understandable why they might be particularly good triggers for recall. The presence of all three imagery elements should increase the likelihood that scenes have features that correspond to elements of the memory and thereby increase the probability and completeness of retrieval.

Once additional details or events are reported, there is a question about how to proceed with the administration of exposure trials. We believe that it is best to continue with the ongoing presentation until a point of demonstrated extinction has been reached before incorporating the new material. The rationale for this recommendation rests in part on the fact that an inadvertent negative reinforcement paradigm may be created by allowing the report of new material to terminate ongoing scene presentation. In other words, the new material might allow the patient to avoid the therapeutic exposure by introducing less emotionally evocative material. If this happens, the act of reporting new information may be rewarded by relief from unpleasant emotion, and this relief may, in turn, encourage reporting of material that is not relevant to treatment in order to interrupt subsequent exposure trials.

Reactivation by exposure treatment often involves elaboration of the identified target memory, but there also are instances of additional, relatively distinct memories being recalled. For example, a military veteran being therapeutically exposed to scenes depicting hand-to-hand combat may suddenly recall a childhood episode of parental physical abuse. Cuing of the childhood memory might come from any number of shared elements contained within the combat memory. Possibilities include similarities between the enemy combatant and the parental perpetrator in appearance or expression, an emotional reaction provoked by both experiences, or comparable meaning (e.g., fending off aggression) attached to them.

If the newly evoked memory is relatively clear, it may become a target of treatment after exposure for the initial target event has reached a reasonable point of affective extinction. If the new memory is partial or fragmentary, a more cautious approach is warranted. For example, a woman being exposed to depictions of a motor vehicle ac-

cident in which she was trapped inside her badly damaged car might report sensations of suffocation that didn't occur during the accident. Or she might report intrusive images of a dark room or the outline of a human face that are not linked to the accident. It may be reasonable to hypothesize that these new memory fragments relate to another important emotional or traumatic event in her life, but, as discussed earlier, the most justifiable course is to assess this possibility with considerable reliance on material supplied by the patient herself. If this cautious approach yields more complete recall of the memory or any other trauma-related material, treatment of the new material can be undertaken. However, if the new memory remains fragmentary and incomplete, exposure treatment may be best left for another time when, if ever, it is recalled in more complete detail.

CONCLUSION

There is an intimate connection between emotion and memory that is evident in the developing empirical and theoretical literature on PTSD. The cross-fertilization of these topics is leading to exciting advances in our understanding of mechanisms underlying adjustment to psychological trauma, and in the development of interventions to decrease distress and improve functioning when adjustment is impaired. At the same time, there are ethical and forensic issues that highlight the limits of our knowledge and encourage caution with regard to our actions. Responsible translation of scientific knowledge into therapeutic action requires respect for the autonomy of individuals who seek treatment and appreciation for the medical dictum, "first, do no harm."

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