CHAPTER ONE

Back to the Future of Understanding Trauma

Implications for Cognitive-Behavioral Therapies for Trauma

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Cognitive-behavioral therapy (CBT) for trauma represents a broad class of therapies unified by a shared emphasis on observable outcomes, symptom amelioration, time-limited and goal-oriented intervention, and an expectation that patients will assume an active role in getting better. An additional strength of CBT applied to trauma is its adherence to evidence-based conceptualization of patients' posttraumatic psychopathology. We assert that increased understanding of the nature of posttraumatic reactions can translate into enhanced effectiveness and innovations in CBT for trauma. Here we trace the evolving history of understanding posttraumatic pathology, and with an appreciation of this past, offer a vision of upcoming achievements and challenges in the application of CBT for trauma.

POSTTRAUMATIC REACTIONS: LONG RECOGNIZED BUT VARIABLY LABELED

Documented human history is replete with descriptions of individual reactions to traumatic events. For example, a survivor of the Great Fire of London in the 1600s wrote in his diary 6 months after his exposure, "it is strange to think how to this very day I cannot sleep a night without great terrors of the fire; and this very night could not sleep to almost two in the morning through great terrors of the fire" (quoted in Saigh & Bremner, 1999, p. 1). There has been remarkable consistency in the description of such posttrau-

matic reactions throughout the centuries, whether written by poets and novelists or clinicians and scientists. Despite this general agreement on observable phenomenology, many different causal mechanisms and diagnostic labels have been proposed. Indeed, the theoretical etiology of these reactions as organic versus psychological as well as the diagnostic classification of traumatic reactions have evolved over time.

Historical Conceptualizations

When the scientific approach to psychopathology emerged in the 19th century, the zeitgeist was to determine organic pathogeneses, such as lesions of the nervous system, as the major cause of nervous disorders. Posttraumatic reactions were no exception to this theoretical organic orientation. Some of the most detailed writings and elaborated conceptualizations of traumatic reactions are found in the literature on combatants.

Starting with the Civil War, American conceptualizations of posttraumatic reactions were understood mostly as somatic/physiological reactions, usually affecting the cardiovascular system. According to Hyams, Wignell, and Roswell (1996), proposed somatic/physiological diagnoses were Da Costa syndrome/irritable heart (Civil War), soldier's heart, neurocirculatory asthenia and shell shock (World War I), and effort syndrome (World War II). Attributing these reactions to organic causes had a number of sociopolitical implications: Soldiers could avoid the stigma and sense of personal failure associated with mental disorders, and the military could ignore the need for psychological interventions.

Although there is only a smattering of accounts of the psychological sequelae of natural and technological disasters during the late 19th century, it is known that civilian traumas were also attributed to organic causes. For example, "Railway spine" was considered to be the result of railroad accidents that produced theoretical, but usually unobservable, physical lesions or insults to the brain, spinal cord, or peripheral nervous system. This condition is representative of the tendency to attribute otherwise unexplainable physical disabilities to abnormal central nervous system mechanisms. Indeed, an English surgeon, John Erichsen (1882), cautioned against confusing (what he assumed to be) the organically caused symptoms of railway spine with hysteria, the prevailing diagnosis of the times (van der Kolk, Weisaeth, & van der Hart, 1996). When physical injuries could not be found in these patients, their symptoms were attributed to subtle forms of neurological damage and a general functional disturbance of the nervous balance or tone. The German neurologist Herman Oppenheim (1915) is credited with coining the term "traumatic neurosis." He proposed that functional problems were a result of subtle molecular changes in the central nervous system following exposure to trauma.

Posttraumatic reactions were not left out of Kraepelin's (1896) efforts in the 1800s to classify and organize mental disorders. He developed a com-

mon label for these multiple nervous and psychic phenomena: "schreckneuroses," or fright neuroses. Schreckneuroses were believed to result from severe emotional upheaval or sudden fright, and to have neurological underpinnings. The symptoms of schreckneuroses were observed after serious accidents and injuries, particularly fires, railway derailments or collisions (Saigh & Bremner, 1999).

Sigmund Freud rebelled against the primary focus on organic explanations for psychopathology in vogue during that period. Because of his influence, psychological etiologies began to be proposed for understanding and treating psychopathology, in general, and posttraumatic reactions, in particular. Freud theorized that, because traumatic events overwhelm the psyche, traumatized individuals must engage extremely primitive defense mechanisms such as dissociation, repression, and denial. Catharsis and abreaction, involving high levels of emotional expression, were considered the necessary treatment for countering these primitive defenses (Freud, 1950). Other contemporaneous psychological conceptualizations of combat trauma included nostalgia (Civil War), battle fatigue/combat exhaustion/operational fatigue (World War I), and war/traumatic neurosis (World War II) (Hyams et al., 1996).

Although Freud stood strong against the winds of the medical and scientific culture pertaining to organic versus psychological explanations of psychopathology, he unfortunately wavered in the winds of Victorian culture regarding childhood sexual abuse. His emphasis on the internal workings of individuals—psychosexual drives and early developmental processes—to the exclusion of external stressors such as childhood sexual abuse was a serious oversight from our modern perspective (see Pendergrast, 1999, for more thorough review of this debate). Freud's legacy is also found in the recovered memory versus false memory debate that erupted in the early 1990s. His notion of the primitive defenses involved in traumatization, and especially repression, as the foundation of claims regarding recovered memories of sexual abuse. Although the potential for psychogenic amnesia of traumatic events cannot be completely ruled out, the past 15 years of scientific evidence questions the veracity of such memories and the possible iatrogenic effects of psychotherapy in creating them (Brewin, 2003).

Freud's contemporary, Pierre Janet, was also instrumental in bringing a psychological approach to posttraumatic reactions, and his writings include some precursor elements of CBT. Indeed, cognitive-behavioral theories of traumatic reactions find their roots in Janet's writings about the categorization and integration of memories. He contended that people develop meaning schemes based on past experiences that prepare them to cope with subsequent challenges. When people experience "vehement emotions" in response to frightening experiences, their minds are not capable of integrating the events with existing cognitive schemes. When the memories cannot be integrated into personal awareness, something akin to dissociation occurs. Janet also introduced the notion of patients experiencing a "phobia

of memory" that prevents the integration of traumatic events. The memory traces linger as long as they are not translated into a personal narrative. In his conception of trauma, synthesis and integration are the goals of treatment, which was in contrast to the psychoanalytic goals of catharsis and abreaction prevalent at the time (Janet, 1907).

Abram Kardiner, a psychoanalyst who treated World War I veterans, was an early proponent of uniting these organic and psychological conceptual streams. He proposed that veterans who experienced an enduring clinical syndrome resulting from war-zone exposure suffered from a "physioneurosis." This label denotes both physiological and psychological components of trauma reactions and the complex biobehavioral clinical picture exhibited by these veterans. In that regard, Kardiner anticipated, by almost 40 years, many of the symptoms included in the first formal diagnosis of posttraumatic stress disorder (PTSD). Because of this insight, which contradicted prevailing psychoanalytic doctrine, Kardiner might be considered the father of psychobiological theory, research, and practice concerning trauma. As a therapist he acknowledged the changes in self-concept that can occur after trauma exposure, and he was a proponent of psychotherapy to ameliorate both psychological and physiological trauma sequelae (Kardiner, 1941).

Kardiner's work was rediscovered by Lawrence Kolb (1987), who theorized that fear conditioning in the limbic system, especially the amygdala, was responsible for the stable psychological and physiological abnormalities found in posttraumatic reactions. Since Kolb's work, there has been an explosion of basic and translational research documenting psychobiological alterations in trauma patients and thereby providing a rationale for pharmacological interventions (Charney, 2004; Friedman, 2003; Friedman, Charney, & Deutch, 1995; Yehuda & McFarlane, 1997).

Diagnostic Evolution

Our evolving conception of posttraumatic reactions is exemplified by sequential revisions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) with regard to both diagnostic categories and PTSD diagnostic criteria across the DSM revisions. To account for the war-related psychopathology discussed above, the first edition of the DSM (DSM-I; American Psychiatric Association [APA], 1952) included the diagnosis "gross stress reaction." This diagnosis was seen as appropriate for cases involving exposure to "severe, physical demands or extreme stress, such as in combat or civilian catastrophe" (p. 40). Like other disorders in the DSM-I, diagnostic criteria delineating the disorder were not specified. Bucking the prevailing notion of the times that those who developed this reaction were characterologically weak, the DSM-I noted that the diagnosis often applied to "previously more or less 'normal' persons who experience intolerable stress" (p. 40). Unfortunately, gross stress reaction was diluted in the second edition of the DSM

(DSM-II; APA, 1968) to "transient situational disturbance." Although there was a continued emphasis on the "overwhelming" nature of an environmental stressor(s) over individual diatheses in causing the reaction, the focus was exclusively on "transient fear associated with military combat and manifested by trembling, running and hiding" (p. 48). There was no diagnostic acknowledgment that such symptoms might characterize a chronic, rather than an acute and naturally resolving, condition.

Influential writings in the 1970s and 1980s about the clinical presentations of sexual assault and domestic violence victims led to the "rape trauma syndrome" and "battered women syndrome" designations (Burgess & Holmstrom, 1974; Walker, 1984). These newly recognized conditions, in tandem with research on the mental health of World War II prisoners of war, survivors of the Nazi Holocaust, and returning Vietnam veterans, led to greater realization of the generalizability of reactions to life-threatening stressors. During this time, the PTSD diagnosis was unveiled as an anxiety disorder in the third edition of the DSM (DSM-III; APA, 1980). Criteria for the traumatic stressor and specific symptoms were organized into three clusters. Accounting for the range of potentially traumatic events, the stressor criterion was described as something "generally beyond the realm of normal human experience that would evoke significant symptoms of distress in most people" (p. 236). The DSM-III revision (DSM-III-R; APA, 1987) resulted in few changes in the stressor definition and symptom inclusion and organization, but did delineate age-specific features.

The fourth revision of the DSM (DSM-IV; APA, 1994) and its text revision (DSM-IV-TR; APA, 2000) excluded the provision that the traumatic stressor be generally outside the range of normal human experience. This change reflects the empirical evidence that the experience of a stressor capable of producing PTSD is actually quite common. In fact, 75% or more of people will experience such a stressor in their lifetime (Breslau, 2002). More importantly, in the DSM-IV the nature of the individual's reaction to a traumatic stressor was taken into account. The nomothetic standard that the experience would evoke significant symptoms of distress in most people was replaced with an idiographic, subjective criterion. According to the DSM-IV, individuals who have been "traumatized" must have had an overwhelming emotional reaction, defined as "intense fear, helplessness or horror" (p. 428) when confronted by an extremely stressful experience. The operational definition of stressful experiences was also expanded to include observing or receiving information about the traumatic events suffered by others. Although some of the symptom clusters were rearranged and diagnostic thresholds were adjusted, the greatest changes in the symptom criteria were the requirements of additional functional impairment and 1–month of symptom duration.

As described by Brewin (2003) in his more complete discussion of the controversy surrounding diagnosis of posttraumatic reactions, "skeptics" of the PTSD diagnosis assert that the diagnosis is a sociopolitical invention that

has been created in a litigious Western society that seeks to place blame and identify victims and perpetrators. Skeptics argue that PTSD is not found in non-Westernized cultures and contend that normal human reactions to a stressful event only become pathological when diagnoses are applied to them. At their worst, these opponents propose that diagnosing posttraumatic reactions has iatrogenic effects on those who are diagnosed.

These criticisms have been countered by empirical data showing that individuals manifest ongoing trauma-related reactions when there are no identifiable secondary gain issues, and after any of these potential gains has been resolved (e.g., disability compensation, civil or criminal lawsuits; Bryant & Harvey, 2003). Furthermore, evidence has accumulated that PTSD is readily identifiable in traditional, nonindustrialized cultures, although it remains controversial whether more culture-specific idioms of posttraumatic distress might provide a better diagnostic characterization of such syndromes (de Jong, 2002; Green et al., 2003; Marsella, Friedman, Gerrity, & Monsour, 1996).

Prospective studies reveal that a large majority (i.e., 94%) of traumatized individuals will manifest symptoms consistent with a PTSD diagnosis or other mental health problems (e.g., depression, panic, anxiety) in the immediate aftermath of trauma. However, by 3 to 6 months, most individuals' symptoms have resolved (Foa & Riggs, 1995; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Marsella et al., 1996; Norris, Murphy, Baker, & Perilla, 2003; Schlenger et al., 2002). Thus it is important to emphasize that there is a significant amount of "normal" distress that follows exposure to traumatic events that should not be construed as pathological. These data have led several researchers to offer the conceptualization of PTSD as a disorder of "nonrecovery" from trauma exposure (e.g., Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992; Shalev, 1997). It is the persistence and severity of symptoms and the functional impairments that merit diagnosis. Epidemiological studies also argue against the notion of a naturally remitting course for those who do not recover from traumatic events and develop PTSD, given that approximately one-third of affected individuals continue to suffer from the disorder 10 years after their trauma exposure (Kessler et al., 1995). Biological investigations, including psychophysiological, neurohormonal, and neuroimaging studies, contradict the notion that all traumatic reactions are part of a normal stress adaptation process (Yehuda & McFarlane, 1997).

It is important to acknowledge the criticisms leveled against the diagnosis of posttraumatic reactions because they have important implications for deciding whether or not, and when, to provide intervention following traumatic events. From our perspective, there are definitely pathological posttraumatic reactions that call for intervention. We contend that the challenges of treating trauma with CBT are not related to uncertainty regarding the pathological conditions that can develop in response to traumatic exposure, but rather concern the nature and clinical phenomenology of such reactions for treatment.

ANTICIPATED CHALLENGES AND ACHIEVEMENTS

As we previously noted, a scientifically grounded conceptualization of patients' problems is the first step to effective CBT for trauma. Historical review of the understanding of posttraumatic reactions illuminates several important opportunities for the future of CBT for trauma. Translational research and continued interface between science and practice will further the conceptualization of traumatic reactions in order to improve CBT of them. In general, developers and practitioners of CBT for trauma, looking toward the future should capitalize on the evidence that the sequelae of trauma are wide-ranging, multidimensional, and multidetermined.

Several factor-analytic studies since DSM-IV was published have raised questions about the nature and processes underlying PTSD (Foa, Riggs, & Gershuny, 1995; King, Leskin, King, & Weathers, 1998). These studies reveal that, contrary to the DSM-IV, there appear to be four, not three, clusters of PTSD symptoms. Symptoms of effortful avoidance and emotional numbing, included together in the DSM-IV, appear to have different properties, functions, and possible etiologies, according to these studies. Moreover, memory loss, a symptom included in the DSM-IV's avoidance/numbing cluster, does not appear to be associated with the overall construct of PTSD or the symptom clusters. Interestingly, the most conclusive of these studies (King et al., 1998) does not support the notion that PTSD is an overarching, unitary disorder comprised of four symptom clusters. Rather, PTSD appears to be best conceptualized as a heterogeneous disorder with correlated, but separate, symptom manifestations. Recent typology efforts also support this heterogeneity in PTSD presentation (Miller, Greif, & Smith, 2003).

Another important classification consideration on the horizon is whether or not acute stress disorder (ASD) and PTSD should be classified as anxiety disorders. Evidence supporting abandonment of the anxiety disorder placement indicates that a myriad of emotions, including guilt, shame, disgust, anger, and sadness, have been implicated in preventing recovery from posttraumatic symptoms (Resick, 2001). Moreover, Pitman (1993) has argued that the pathophysiology of arousal in posttraumatic reaction is not simply anxiety. The *International Statistical Classification of Diseases, Injury, and Causes of Death–10th Edition* (ICD-10; World Health Organization [WHO], 1992) does not classify PTSD as an anxiety disorder; rather, it is categorized within the spectrum of "reactions to severe stress, and adjustment disorders," with the common denominator of stress-related precipitation. A recent taxometric study also buttresses the dimensional versus categorical system of trauma-related diagnoses (Ruscio, Ruscio, & Keane, 2002).

A spectrum of stress disorders, with specifiers beyond "acute," "chronic," and "delayed onset" currently used for PTSD, could more fully describe the phenomenology of trauma survivors and have important treatment ramifications. Like other major DSM-IV disorder classes (e.g., mood, psychotic), there could be a range of disorders with various symptom con-

stellations and specifiers. SD as well as the dissociative disorders, could be placed in this class. PTSD specifiers such as "prominent dissociation," "prominent emotional numbing," and "prominent anger" could have important theoretical and treatment implications. Additionally, age-related features and presentations of these stress reactions are important. There may even be room for chronic stress reactions to nontraumatic stressors.

It is important to remember that previous statistical approaches to organizing the core features of posttraumatic reactions are limited by the items that comprise the statistical analyses. The DSM-IV PTSD Work Group restricted criteria to "essential features" for making the PTSD diagnosis. However, this approach risks the danger of missing characteristics that have important clinical and treatment relevance. We suggest that, in addition to moving beyond anxiety-based symptom presentations and to enhance recovery among survivors of traumatic stress, CBT for trauma consider and address other frequently observed serious psychological, emotional, and interpersonal problems. Regardless of the diagnostic scheme used, the epidemiological and taxometric findings argue for distinct assessment of, and multicomponent treatment for, the multidimensional nature of posttraumatic pathology (Flack, Litz, Weathers, & Beaudreau, 2002; Keane & Kaloupek, 2002).

In spite of having several very efficacious CBTs for trauma-related pathology (described in this book), it is important to realize that about 50% of the patients in efficacy studies maintain their trauma-related diagnoses at the end of treatment and at follow-up periods (Zayfert, Becker, & Gillock, 2002). This symptom maintenance may be related, in part, to our current conceptualization of trauma sequelae and to the fact that the current evidence-based treatments, in isolation, address some specific aspects of trauma better than others. For example, some treatment studies reveal that avoidance and numbing symptoms, and especially emotional numbing, may be less responsive to our current CBT treatments (e.g., Glynn et al., 1999; Keane & Kaloupek, 1982). There is also some early evidence that different CBTs may be better at addressing the different emotional disturbances resulting from traumatization (e.g., Resick, Nishith, Weaver, Astin, & Feuer, 2002).

In this vein, efforts to determine predictors of treatment response to CBT for trauma may help address diagnostic dilemmas and ultimately improve treatment planning and outcomes. We recommend that future studies consider predictors beyond those that have been traditionally investigated (e.g., PTSD severity, anger, substance abuse), and develop theoretically driven models that can be tested. Following from our recommendations about broadening the range of trauma symptoms to consider, interpersonal functioning, social support, affective regulation, and self-efficacy might be considered. Biological markers may even be useful to consider in the future, as the psychobiological findings become more robust and are shown to correspond with CBT treatment response.

In the last decade the field of CBT for trauma has seen a series of head-to-head trials designed to determine the treatment "winner." These trials have resulted in many more "ties" than declared winners. We anticipate that the next generation of dismantling, combination therapy, and effectiveness studies will reveal very intriguing findings about the key ingredients of efficacious treatment as well as the limits and challenges to using these treatments in clinical settings. Given that many patients simultaneously receive two or more treatments in clinical practice (e.g., Rosen et al., 2004), studies that determine how best to time or integrate treatments for greater efficacy will be valuable. The possibility for psychopharmacological treatments to potentiate or possibly interfere with CBT for trauma should also be investigated. Like others (Foa, Rothbaum, & Furr, 2003), we call for more combination studies aimed at addressing nonresponse or partial response to treatment, in lieu of the rates of non- and partial response found in previous studies.

An additional factor to investigate with regard to treatment timing and sequencing relates to the co-occurring diagnoses often given to traumatized individuals. Determining the best sequence or combination of treatments to treat these disorders is very important for the future of CBT for trauma. As an example, many prior PTSD treatment studies have excluded patients with comorbid substance dependence, suggesting that these issues should be addressed prior to a course of CBT for PTSD. There have been a few developing efforts to provide serial or integrative trauma and substance abuse treatment (Coffey, Dansky, & Brady, 2003; Najavits, 2002). Depression, personality disorders, anger problems, self-harming behavior, and relationship dysfunction are other frequently co-occurring diagnoses or clinical issues to address. Researchers have designed several treatments to specifically address these problems in tandem with PTSD treatment (Chemtob, Novaco, Hamada, & Gross, 1997; Cloitre, Koenen, Cohen, & Han, 2002; Monson, Schnurr, Stevens, & Guthrie, 2004). However, other researchers have argued that the existing CBTs for PTSD should be undertaken first, because effective treatment for PTSD can remedy many of these co-occurring issues (e.g., Cahill, Rauch, Hembree, & Foa, 2003). These are questions in need of further empirical investigation.

The cognitive-behavioral framework has an important role in informing prevention and early-intervention efforts. Because this area has been wrought with controversy, leading with a strong theoretical grounding for these interventions will be crucial. In addition, the caricature of CBT is that it is a mechanical and technical venture devoid of any humanity. A solid therapeutic relationship is essential to all forms of psychotherapy. Treatment process studies that pinpoint specific dimensions of the therapeutic relationship that are detrimental or facilitative of trauma recovery are essential (Cloitre, Stovall-McClough, Miranda, & Chemtob, 2004).

There are a number of intriguing questions to be answered with regard to the effectiveness, versus efficacy, of CBT for trauma. Most of the outcome

studies to date have been undertaken in outpatient research clinics. Ongoing efforts to transport these best practices into clinical settings, and likewise, to use the clinical experiences to inform research, will be invaluable.

Although several CBTs for trauma, with solid evidence bases, are available there remains a need for innovative treatments that can help the significant number of patients who do not respond to our current treatments. Understanding of the nature and treatment of trauma is a continuously evolving process. We have come a long way in conceptualizing the aftereffects of trauma and in developing elegant, theoretically driven CBTs that work. We look forward to the advancements that will be made in the next generation of CBT for trauma.

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